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THE OXFORD MEDICAL PUBLICATIONS

# A SYSTEM OF MEDICINE

BY EMINENT AUTHORITIES IN GREAT BRITAIN, THE UNITED STATES AND THE CONTINENT

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VOLUME VII.

DISEASES OF THE NERVOUS SYSTEM


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# DISEASES OF THE NERVOUS SYSTEM.

## CHAPTER I.

### INTRODUCTION TO DISEASES OF THE NERVOUS SYSTEM.

By LEWELLYS F. BARKER, M.D.

#### THE SENSES AND THEIR SYMPTOMATOLOGY.

It is convenient to divide the senses into two groups, *interior* and *exterior*. The interior senses are those of which we project the sensations into some part of our own bodies. They include pain, movement sense, position sense, resistance sense, the vestibular sense, hunger, thirst, the sexual sense, and the sense of fatigue. The exterior senses are those of which we project the sensations into the world outside ourselves. They include sight, hearing, taste, smell, touch and pressure, and the two temperature senses, heat and cold.

**The Interior Senses.—Pain.**—Physiologists now regard this as a special sense, and not simply as a negative feeling tone associated with overstimulation of other sense organs (von Frey). This pain sense has a punctiform distribution in the skin. As tested by very fine needles, the points are more numerous even than the touch points. The strength of the stimulus required to call forth a sense of pain is called the threshold stimulus, and this varies for different pain points. The localization of pain sensations is due less to the pain nerves than to the simultaneous stimulation of the sense of touch.

Visceral pain is often not localized in the viscera themselves, but is referred to certain areas in the skin (Head). The cutaneous hyperæsthesias in cardiac, gastric, intestinal, and prostatic diseases are examples of such misreference. The pain sense of the skin and viscera has been designated by Head and Rivers as *protopathic* sensibility, in contrast with touch and temperature senses, which they call *epicritic* sensibility.

Sometimes the peripheral nerves are oversensitive to pressure, particularly at places where they emerge from bony openings (*Valleix's points*).

**Deep or Mesoblastic Sensibility (Sensations from Muscles, Bones, Joints, and Fasciæ). Sensations of Movement of Parts of the Body; of Their Position, and of Resistance. Bathyæsthesia.**—Under the term "muscle sense," a whole series of different sensations have been roughly lumped together. This is due largely to the lack of sharpness of these sensations in consciousness. We

must distinguish at least the following: (1) The sense of position of the individual parts of the body; (2) the sense of movements of these parts; (3) sensations of resistance and weight; (4) the stereognostic sense; (5) the so-called vibration sense (pallæsthesia).

1. *Sense of Position of Individual Parts of the Body.*—The normal person is able actively to imitate with one-half of his body positions in which portions of the other half are passively placed.

2. *Sense of Movement of Individual Parts of the Body.*—When the eyes are closed and active or passive movements of a part of the body are made, a normal person is able to state (*a*) that a movement has occurred, (*b*) the degree of this movement, and (*c*) its direction. This sense of movement differs from the sense of position. In normal individuals any movement that the examiner himself can see should be recognized. When a joint has to be flexed or extended through several degrees before a sensation is detected, the movement sense is subacute. It should be remembered that smaller movements can be recognized in the shoulder and wrist than in the knee, hip, or elbow. The ankle-joint and the joints of the fingers and toes are still less sensitive. In right-handed individuals the movement sense is more delicate on the right side than on the left.

3. *Sensations of Resistance and Weight.*—The sense of weight is not identical with that of resistance and is more delicate in the upper than in the lower extremities.

4. *Stereognostic Sense, or Perception.*—The capacity to recognize the form and consistence of objects by touching and feeling them is known as stereognosis. One places in the hand of the blindfolded patient blocks of various shapes or familiar objects, such as knives, keys, and coins, and asks him to describe their form and consistence and to name them. This capacity involves the combined activities of the movement sense, resistance sense, position sense, sense of touch, and sense of temperature. Even more, certain higher cortical faculties are involved, including the spatial and temporal arrangement of the sense impressions and the memory pictures of previous sensations. Obviously, we have to do here not simply with sensation, but with a combination of sensory, associative, and reproductive processes.

5. *Vibration Sense, or Pallæsthesia.*—Rapidly vibrating objects when placed upon the skin over a bone yield a peculiar vibrating sensation known as pallæsthesia. Clinically, one examines the sensation best by placing the handle of a vibrating tuning-fork of 64 or 128 oscillations per second firmly upon the surface of the skin over one of the bones.

**Vestibular and Allied Senses. Sense of Static Equilibrium. Sensations of Position of the Body as a Whole. Sensations of Movement of the Body as a Whole (Rotary and Non-rotary or Progressive). Sensations of Orientation in Terrestrial Space. Sensibility to Accelerations in a Straight Line and to Angular Accelerations.**—Here we have to deal with a great group of centripetal impulses which are of high importance for the functions of equilibrium, locomotion, and orientation. It seems likely that most of the centripetal impulses concerned are infraconscious, or that their psychic correlate is relatively insignificant.

The directions up and down are recognized by everybody, though how the force of gravity is made known to us is not clear. Even under water the capacity for orientation as to the vertical direction is unimpaired. The mechanism, whatever it is, is sensitive to *accelerations in a straight line*, and

gives rise not only to our knowledge of the vertical direction, but also to sensations of progressive or non-rotary movements. In addition to this mechanism, there is another apparatus in the head which is sensitive to *angular accelerations* and gives us sensations of change of velocity of rotary movements. These sensations, which follow accelerations in a straight line and angular accelerations, are associated with characteristic eye-muscle reflexes. An alteration of the position of the head relative to the vertical causes a compensatory change in the setting of the eyeballs, and if the body be rotated around its vertical axis the eyes show movements of nystagmus as long as the sensations of rotation are felt.

These sensations are connected with the functions of the membranous labyrinth of the internal ear. It seems probable that the information which the brain receives regarding the position of the head when it is at rest and regarding the acceleration or retardation of progressive (non-rotary) movements depends upon the nerve endings in the utricle and saccule, upon the maculæ of which the little ear-stones or otoconia rest (Breuer), while the sense organ underlying the sensations dependent upon angular accelerations appears to be situated in the ampullary crests of the membranous ampullæ of the semicircular ducts. While both the utricle and saccule, on the one hand, and the semicircular ducts, on the other, appear to have to do with static as well as dynamic sensations, it seems probable that the static are mediated rather more by the former, the dynamic rather more by the latter. The loss of orientation as to the vertical when under water and the absence of vertigo on rotation so often seen in deaf mutes are thought to be due either to imperfection of the otoconic apparatus or of the semicircular ducts.

**Other Interior Senses.—Hunger.**—In its mild form this sensation is familiar to everyone as the appetite for food localized in the gastric region. The hunger sense is to some extent dependent upon metabolism. The well-known increase of appetite after muscular exertion, after exposure to cold, and in diabetes mellitus makes this clear. On fasting, the sense of hunger soon disappears, in marked contrast with the sense of thirst.

**Thirst.**—The sense of thirst is referred to the back of the throat, and if no water be taken the sensations become painful. The sufferings of fasting patients are due to thirst rather than to hunger.

**Sense of Fatigue.**—If the muscles are made to contract beyond a certain point, a feeling of fatigue is encountered. Whether this is due to centripetal impulses arising in the muscles themselves or to a change in the nerve centres in the cerebral cortex is not certain.

**Sexual Sense.**—The impulse to sexual gratification is technically known as *libido*. The sexual impulse of adults appears to be a unification of various childhood impulses; these culminate normally in an effort toward a single specific goal. Originally auto-erotic, the impulses become later hetero-erotic.

**The Exterior Senses.—Touch or Pressure.**—The skin contains sensory mechanisms that permit us to recognize very slight contact and very light degrees of pressure. The sense organs are closely related to the hairs in parts of the body which possess these and to Meissner's corpuscles in other parts. When delicate stimuli are employed it is found that sensations are set up only when definite points are pressed upon. These are the so-called *touch points* of von Frey. They are much more numerous than cold points and warm points, but are less numerous than pain points. They are very numerous on

the lips, tongue, and finger tips, and are relatively much scantier on the leg and upper arm. The stimuli do not act upon the sense organs directly, but rather indirectly through the skin surface or its hairs, producing deformations of the skin and thus causing alterations of pressure tensions or traction tensions in the tissue. The stimulus in reality is a function of the tension difference from place to place. To use a physical expression, the excitation depends upon the "pressure fall" and not on the pressure itself.

Touch sensations following brief stimuli last only a short time, and if stimuli be repeated rapidly one receives a corresponding number of sensations without fusion in consciousness. The threshold stimulus for individual touch points varies. One can determine the threshold most easily by the use of von Frey's test hairs.

No marked feeling tone accompanies touch sensations, a fact which distinguishes them sharply from sensations of pain. The time elapsing from the moment of application of the stimulus to a touch organ to the moment of appearance of sensation in consciousness is shorter for touch than for temperature or pain (reaction time). Touch sensations can be very accurately localized upon the surface of the body; indeed, the sense of touch, together with sight and movement sense, has done most to give to man his conceptions of spatial relations. Each touch point on the surface of the body seems to have its own "local sign;" that is, the sensation from it can be distinguished from that from every other. If two touch points are close together, however, they must be stimulated successively and not simultaneously if the two separate sensations are to be recognized (*successive threshold* of von Frey). When touch points are a certain distance apart, stimulation of them simultaneously gives rise to two separate sensations (*simultaneous duplicity threshold* of von Frey; *space threshold* of Weber).

**Tickling and Itching.**—These are as yet not well understood. Tickling is probably to be separated physiologically from itching. However, both are closely related to touch sense and pain sense. It is possible that they should be regarded as secondary sensations rather than primary (H. Quincke), and that they are associated with vasomotor reflexes. It is interesting that the sensations are aroused by slight stimuli and can often be inhibited by more powerful stimulation in the same areas.

**The Temperature Sense.**—In the skin and mucous membranes there are at least three varieties of sensory nerve beginnings which respond to thermal stimuli; they are connected with the so-called cold points, warm points, and pain points. The adequate stimulus for a cold point is cold, that for a warm point is warmth, but both sets of points react to various disparate stimuli also; thus, one gets a sensation of cold when he stimulates a cold point by a sharpened stick, by the application of menthol, or by an electrical current, and a warm point can be stimulated not only by heat, but by mechanical irritation with fur, by exposure to carbon dioxide, or by electricity. Pain points are stimulated by cold below a certain temperature or by heat above a certain temperature. Touch points appear to be uninfluenced by thermal stimuli. The cold points are far more numerous than the warm points, but they are less evenly distributed than are the touch points and the pain points.

Natural stimuli, as a rule, affect areas of the skin which contain a number of sensory points, so that our ordinary cutaneous sensations are complexes made up of a number and variety of elementary sensations. When the ther-

mal stimulus corresponds in temperature to that of the skin no sensation at all is felt; if it correspond to a temperature slightly lower than that of the skin, the cold points alone respond and a sensation of coolness results; if it correspond to a temperature a little above that of the skin, a sensation of warmth is felt. Very low temperatures affect both the pain points and the cold points; very high temperatures may affect the warm points, the cold points, and the pain points simultaneously, and yield a mixed sensation compounded of warmth, cold, and pain, usually designated "burning hot."

A word must be said about the power of *adaptation* of the skin to various external temperatures. The skin soon adjusts itself to objects of constant temperature applied to it, and on their removal a new thermal stimulus must have a temperature a quarter of a degree above or below that of the object previously applied if sensations of warmth or cold are to be experienced, owing to the so-called *indifference zone* (physiological zero), through which no sensations are excited. The position of this indifference zone varies with the external temperature and with the blood supply, and this explains why it is that there is often a lack of correspondence between the physiological temperature descriptions and the readings which we meet on the scale of a physical thermometer.

The clinical methods for testing the temperature sense are very crude compared with our physiological knowledge of these senses. More accurate studies have been made in certain instances, and in some cases the threshold stimuli have been determined.[1] A thermæsthesiometer has been introduced by Eulenburg, but has been but little used.

**Taste.**—There are four simple elementary taste qualities: sour, sweet, salty, and bitter. All other so called taste sensations are either combinations of these, or are mixtures of taste sensations with sensations of smell, touch, temperature, or pain. Some taste sensations are associated with pleasurable feeling tone, others with negative feeling tone. Substances which can be tasted are known as "sapid substances," and are soluble in water or in saliva. They are crystalloid in nature, colloids being devoid of taste.

Apparently there are entirely different sense organs for sour, sweet, salty, and bitter sensations. The adequate stimulus for arousing sour sensations appears to be the hydrogen ion of acids. No common chemical character has as yet been ascribed to the sapid substances which arouse sweet sensations; salty sensations, are best aroused by a 2 per cent. solution of sodium chloride, though various other salts can stimulate the sense organ concerned. Bitter sensations are aroused by very different chemical substances. Many of the alkaloids (strychnine) are bitter, but numerous sapid substances are bitter which are chemically unrelated to these (*e. g.*, magnesium sulphate). The taste organs thus acted upon by chemical stimuli do not respond to mechanical or thermal stimuli. They do, however, sometimes respond to electric currents (so-called *electric taste*).

Single taste modalities are variably represented in various parts of the tongue; thus, sweet taste is best represented at the tip, salty at the tip and margins, sour at the margins, and bitter at the posterior part of the dorsum linguæ. The centripetal nerves of taste leave the tongue by way of the lingual branch of the trigeminal nerve and by way of the glossopharyngeal nerve. The few taste buds of the larynx belong to the pneumogastric nerve.

[1] Barker, *Deutsches Arch. f. Nervenheilk.*, 1898, viii, 348.

Threshold determinations in taste sensation have as yet been very little used, although the *boite gustatométrique* of Toulouse and Vaschide and the *sapid wedges* of Zwaardemaker may be found useful.

**Smell.**—This is closely related to taste sense, and naïve observers often confound sensations of smell with those of taste. The nose possesses touch sense, temperature sense, and pain sense; when we inhale formalin, it is the pain sense which is chiefly aroused, and when we inhale menthol the temperature sense is also affected. Aside from distinguishing sensations of smell proper from other modalities of sensation, we have the still greater difficulty of analyzing olfactory sensations among themselves and resolving them into their simpler psychological constituents.

The olfactory sensations are mediated by the neuro-epithelial cells in the olfactory region high up in the nose. The only approach to this olfactory area is through the narrow olfactory sulcus, so that the application of localized stimuli is fraught with extreme difficulty. The chemical substances brought to the olfactory area by air currents probably dissolve in the film of fluid which bathes the neuro-epithelium.

Smell is to be regarded as a chemical sense, and the adequate stimuli are chemical substances which act directly upon the peripheral olfactory neurones. Other forms of stimuli seldom call forth an olfactory response, although certain smell sensations have been produced by electrical stimulation. Very minute amounts of certain chemical substances are sufficient to stimulate the sense organ, and they must be gaseous or volatile in order that they may mix with the air that enters the cavity of the nose. For human beings the smell sensations aroused by the entrance of chemical substances through the nasopharynx are more important than those dependent upon substances entering through the front of the nose.

While there are only four fundamental varieties of taste sensations, it seems likely that there are, at least, twenty or thirty elementary olfactory sensations; the difficulties surrounding experimental investigation prevent any dogmatic statement concerning this number.

Attempts at quantitative methods of examination of the sense of smell have been made, especially by Zwaardemaker. The term *olfactometry* has been applied to the measurement of the sensibility of the olfactory organ for adequate stimuli, and the term *odorimetry* is used to indicate the making of comparative measurements of the stimulating action of different substances.

We know nothing of exact localization of olfactory sensations. The olfactory sense is not easily fatigued, although it varies in this regard in its different modalities. The close relation of the olfactory sense to various reflex, instinctive, and affective phenomena need only be mentioned to be appreciated. The odor of food excites the impulse to eat and starts the secretion of gastric juice. The relation of the sense of smell to the excitation of the sexual impulses is well known, and the relation of odors to various affective states is close. The mood may be changed in a moment by a scent or a stink.

The clinical examination of the sense of smell is usually carried out by holding odoriferous substances before one nostril at a time; the patient inhales and tries to name the substance. Well-known substances, chiefly, are used. As the majority of people are surprisingly inaccurate in the discrimination of smell sensations, the following series can be recommended:

Rubber, gutta-percha, ether, yellow beeswax, camphor, oil of anise, vanilla, musk, asafœtida, cacodyl or ammonium sulphide, roasted coffee, pyridin, tar or carbolic acid, cheese or mutton tallow, tincture of valerian, skatol.

Where smell is defective a careful examination of the nose should be made to make sure that the defect is not due to obstruction of the air currents in the nose rather than to any disturbance of the nervous mechanism itself. In addition to trying the olfactory sense, one may, if he likes, test the functions of the trigeminus in the nasal mucosa with the fumes of acetic acid, ammonia, formalin, and menthol.

**Hearing.**—The organ of hearing mediates the sensations of sound. Sounds are divided into two great groups known as *musical sounds* and *noises*. The former are smooth, steady, and the latter rough, unsteady, or restless. Both are made up of elements known as *tones*, and all sounds in nature represent combinations of several or many different tones. In compound sounds one simple tone is often dominant. This is the so-called *fundamental tone*. The associated higher tones are known as *overtones*. The compounds of tones which are musical are known as *clangs*, and the musical ear can distinguish among the partial tones of a clang not only the fundamental tone, but also the various overtones.

On looking for the *adequate stimulus* for the organ of hearing, it is found to consist of vibrations of varying periodicity, and the sensory nerve beginnings seem to be attuned to definite frequencies of vibration. The vibrations reach the organ of Corti either by *air conduction* through the external and middle ear or by *osteotympanal conduction*, the transmission occurring then through the bones of the skull. The organ of hearing is attuned to certain periodic movements only; thus, oscillations less frequent than 16 per second or more frequent than 50,000 per second fail to stimulate the ear.

Sounds are ordinarily heard with both ears (*diotic hearing*), though sometimes with one alone (*monotic hearing*). If a tuning-fork (*a'*) be placed on the mastoid, the sound is heard by osteotympanal conduction. If at the moment when the sound ceases the fork be removed from the mastoid and placed before the ear, the tone can again be heard through air conduction (*Rinne's experiment*).

Auditory sensations are projected into the external world, but man's power of localizing them is not very great. Although he can tell, when his ears are normal, whether a sound is more to his left than to his right, he is often in error when attempting to say whether a sound comes from below or above or from in front or behind. If a vibrating fork be placed in the median line of the head, exciting the two ears equally, and then one ear be loosely closed with the palm of the hand, the sound is markedly intensified in the closed ear—so-called lateralization of the sound (*Weber's experiment*).

Quantitative measurements of hearing capacity are best carried out by using simple tones as stimuli. In order that two forks with vibration numbers close to one another may give rise to tones which are distinguishable by the ear, there must be a certain difference in the number of vibrations (*difference threshold for pitch*). For a good musical ear the difference amounts to about half a vibration per second in the most favorable part of the scale. For poorer ears the vibration numbers must stand in relation to one another as 5 to 6, or even as 2 to 3. In otiatric diagnosis it is often important to determine the *upper and lower limits of audible tones*. The best apparatus for this is the "continuous tone series" of Bezold-Edelmann,

which permits of stimulating with vibrations numbering from 16 to 48,000 per second.

The *intensity threshold* of a tone must also sometimes be studied; that is, the intensity which a given tone must have in order to be just perceptible by the ear. Furthermore, a tone must continue for a certain length of time if tone sensation is to be aroused (*duration threshold*).

*Clinical Examination of the Sense of Hearing.*—Ordinarily clinicians are satisfied with (1) determining the so-called auditory acuity; (2) testing the upper and lower tone limits; (3) Rinne's test; (4) Schwabach's test; (5) Weber's test; (6) Gelle's test.

To determine the *auditory acuity*, the only exact way is to use the threshold methods referred to above; most of these methods are as yet too difficult or too circumstantial to permit of extensive application. In otiatry, therefore, various rough-and-ready tests, serving for general orientation as to hearing power, have been introduced. One tests the ear first for sounds of different kinds. Thus the power to hear *spoken* and especially *whispered* words is examined, preferably in a still, closed room. The average distance at which whispered sounds can be heard (the whisper being produced with the aid of the residual air left in the thorax after deep expiration) is about 25 meters in a very quiet room, about 20 meters in ordinary rooms. A patient whose hearing permits these words to be heard as far away as 6 meters suffers no practical inconvenience. Each ear is tested separately, the patient standing at the end of the room, the other ear being tightly closed with the finger and the face and eyes looking straight forward so as to render lip reading impossible. Beginning at a distance of 6 meters a word is whispered, and the distance gradually decreased until the word is recognized and correctly repeated.

If whispered words are not heard at all, one uses "conversational tones" in the same way. Spoken words can, however, be heard, when one ear is deaf, at a distance of 6 meters, even when the healthy ear is closed. To be sure about this, if the patient hears conversational tones at a distance of 3 meters when the good ear is closed and the bad ear open, one tries again when he closes also his bad ear (*Lucae-Dennert test*); if he can no longer repeat the test words, this proves that he heard with the bad ear, but if he still repeats them one can be sure that the bad ear did not hear.

More accurate are measurements with Politzer's *acumeter*, an instrument which yields sounds of equal intensity; the drawback to it is that the noise it produces consists exclusively of a few high tones. For the same purpose the tick of a watch may be used. But the distance at which a sound can be heard is not a good measure of auditory acuity.

The poorer the hearing in one ear, the more quickly a waning tone ceases to be perceived, and the measurement of the time a tone is heard is, therefore, an important aid in otiatric diagnosis. The numerical expression of defective auditory acuity is not simple. If a patient, for example, hears the fork *c* for 92 seconds, while a normal ear hears it 183 seconds, the auditory acuity under test (*H P*) is not $\frac{1}{2}$ the normal acuity (*H N*) but a much smaller fraction, about $\frac{1}{3\,5\,2}$ (Ostmann). This depends on the fact that during the waning of vibrations of a fork the intensity does not diminish by equal amounts in successive seconds; at first the intensity decreases rapidly, then ever more slowly.

But for any measurement of auditory acuity which pretends to accuracy

one must be able easily (1) to use tones of any desired pitch, (2) to use pure tones, (3) to measure their intensity exactly, and (4) to offer them to the ear in even intensity for any desired period. The instrumentarium which best meets these requirements is that of Wien, in which a telephone is used as a source for the tones, the sinusoidal alternating currents being yielded by a sinus inductor (for low tones) and by an alternating current siren (for higher tones).

Having determined the *degree* of auditory defect, one has next to try to ascertain its *cause;* one must decide whether the defect is due to the *sound-conducting apparatus* (auditory meatus, tympanic membrance, middle ear) or to the *sound-perceiving apparatus* in the internal ear. The data to be gathered consist in the following:

1. *Determination of upper and lower tone limits* by means of the Bezold-Edelmann continuous tone series or more simply by tuning forks $C_{11}$ $c_1$ $c^4$; the upper limit may be normal and the lower limit changed in middle-ear disease, while the opposite holds for labyrinthine deafness.

2. *Rinne's Test* (*vide supra*).—The difference between the time of air conduction and of bone conduction should be noted in seconds for a fork having a pitch somewhere between $c$ and $c^2$.

3. *Schwabach's Test.*—Comparison of the time a fork is perceived when held on the vertex with the time in a person of normal hearing; the time is shortened in labyrinthine disease.

4. *Weber's Test* (*vide supra*).—The sound may be "lateralized" in one ear without artificial closure—toward the deaf ear in middle-ear disease, toward the healthy ear in labyrinthine deafness.

5. *Gelle's Test.*—If one compresses the air in one meatus by means of a rubber bulb, the tone of a fork held on the same half of the skull will be less loud at the moment of compression if the base of the stirrup be movable, while it will remain unchanged if it be fixed (ankylosis of the stirrup).

**Sight.**—When the eyes are open the visual sensations are spatially extended and exteriorized as the so-called *binocular visual field.* If one eye is closed we project a *monocular field.* When both eyes are closed, or if they be open in the dark, there is a sense of blackness, something quite different from the absence of sensation corresponding to the region peripheral to the visual fields.

There are two series of visual sensations: (1) An achromatic series—black, gray, white—which can be represented geometrically in their transitions by a straight line; and (2) a chromatic series—red, orange, yellow, green, blue, violet, purple (in all at least 160 tones at full saturation)—which can be represented geometrically in their transitions by a line which returns to the starting point, *e. g.*, by a circle. This series of sensation is related to the achromatic series in such a way that the greatest saturation of each color occurs normally at a certain luminosity, *e. g.*, yellow is brighter than orange when each is fully saturated. This relation of the chromatic to the achromatic series gives rise to the so-called variables in sensations of color. There are three conditions of variability—the first dependent on quality, the second on luminosity, the third on saturation.

The organ of visual sense includes the eye and a series of neurone chains in the central nervous system. The *dioptric* apparatus of the eye determines the mode of application of the *adequate stimuli* (light rays) to the sense organ proper, the *retina.* The impulses arise in the rods and cones and pass

through the bipolar cells and the ganglion cells of the retina into the optic nerve and thence to the primary and secondary optic centres of the brain. The whole path from the retina to the cortex, composed of several sets of superimposed centripetal neurones, is known as the *visual conduction path.*

It is interesting that the different portions of the retina behave very differently in regard to color sensations; thus, at the fovea (*central vision*) and the area immediately adjacent (*paracentral vision*) the color sense is quite different from that of the more peripheral portions of the retina (*excentric vision*). Objects which seem colored in central vision may have different colors in paracentral vision, and may be colorless when excentrically viewed. In other words, the periphery of the retina is totally color blind and its vision is monochromatic; the central portion of the retina has polychromatic vision, while there is an intermediate zone which is partially color blind, having dichromatic vision. This explains why it is that the visual fields for colors are of different sizes even in the normal eye.

The ether vibrations of the light rays lead to photochemical, histological, and electrical changes in the retina and give rise to the nervous impulses. The luminosity of a visual sensation is a function of the actual energy of the ether vibrations and of the parts of the retina stimulated. The eye cannot analyze white into its constituent components, and thus differs from the ear, which is able to recognize the partial tones in a clang.

*Color blindness* is a deviation from the normal relations in a system of color sensations. It may be most properly defined simply as abnormal color sense. In its least abnormal form it manifests itself in a general weakening of the color sense, while in its most abnormal form no color can be seen at all but only differences in luminosity are distinguished. Between these extremes various deviations from the normal occur.

The following may serve as a general classification of such abnormal cases.[1]

1. *Abnormal polychromates*, who see many colors but who differ from normal individuals (*a*) in that one or more color qualities are absent or (*b*) in outstanding color relations (*e. g.*, in the similarity of certain colors to one another). These are the most common cases, and in the majority it is the colors red and green which are not distinguished.

2. *Dichromates*, who see only one pair of colors, these being complementary, though sometimes different from the normal complementary colors.

3. *Achromates*, who see no colors at all, all lights giving sensations of gray in varying intensity. These are extremely rare.

The theories of color vision deal altogether with the processes supposed to occur in the retina, neural paths, and centres. The theories are widely divergent, but may be roughly divided into three groups: (1) Which holds that there are three fundamental chemical substances in the retina reacting to the three so-called fundamental colors: red, green, and blue-violet; (2) which holds that there are three substances reaching respectively to red-green, yellow-blue, black-white, somewhat after the analogy of anabolic and catabolic processes; and (3) which holds that there are no fundamental color processes, but that highly complex substances react variously for different colors and for colorless light. As a matter of fact these theories are

[1] A. Kirschmann, *Normale und anormale Farbensysteme, Arch. f. d. gesamte Psychol.*, 1907.

pure constructions of the intellect; as yet no one of the postulated chemical substances is really known.

Visual sensations depend not only upon the stimuli applied to the eye, but also upon the state of the eye at the time of application of the stimulus. The change in the eye in the different phases of activity is known as its power of adaptation. Among the phenomena which pertain to this function of adaptation are: (1) Positive and negative images; (2) light induction and color contrasts; and (3) the adaptations undergone by the eye in the light and in the dark. Studies of the *dark-adapted eye* make it probable that the rods of the retina are especially concerned with vision in dim light (night), while the cones of the retina have to do rather with vision in strong light (day).

*Localization of Visual Stimuli.*—Two retinal stimuli which have the same value as regards quality and luminosity may differ in that they are projected into different parts of the visual field. All the retinal excitations taken together give rise in consciousness to the continuous extended field. We concentrate our attention normally upon the point upon which our eyes focus, while the more peripheral portions of our visual fields are less focal in consciousness. The arrangement in space of visual perceptions, therefore, depends upon the local sign which adheres to the sensation aroused by stimulation of each retinal point.

The localization of visual sensations depends, however, not only upon this local sign, but especially in so far as measurement is concerned, also upon eye-muscle sensations.

In examining a patient clinically regarding his capacity for visual sensations we are provided with very exact methods and delicate apparatus. Here we can call attention only to some of the methods most essential for neurological diagnosis, especially to (1) the determination of visual acuity, (2) the determination of the type of central vision, (3) the determination of the capacity for vision in dim lights, (4) the determination of the extent of the visual fields, and (5) the ophthalmoscopic examination.

In *determining the visual acuity*, one uses letters or similar signs, made so as to be as long as they are broad; the strokes of the letters should be of even thickness and one-fifth of the thickness of the whole letter (*e. g.*, Snellen's "Optotypi"). The visual acuity V (*visus*) is expressed by a fraction whose numerator gives the distance $d$ in which the latter is recognized, and whose denominator gives the distance $D$ which corresponds to a visual angle of five minutes for the letter: $V=\frac{d}{D}$. The dioptric apparatus must be borne in mind (anomalies of refraction, accommodation) and, if faulty, the visual acuity determined before and after correction.

In *determining the types of central vision* we decide whether the patient has polychromatic (normal), dichromatic, or achromatic vision. For this Holmgren's colored wools are very satisfactory. From a large collection of wools of different color one chooses one of rose-color (pale bluish red) and asks the patient to put beside it from the collection all the wools of the same color, telling him that he may make mistakes either by choosing wools of a wrong color or leaving behind wools of the same color. If his vision be dichromatic, the patient will pick out bluish green and gray wools and put them with the rose-colored wools. If his vision be achromatic, he will be wholly unable to discriminate among the wools except by differences in brightness. One may use Adler's colored crayons or Stilling's pseudo-

isochromatic charts, Nagel's lantern with colored lights, or color equations with rotating discs.

In *determining the capacity for vision in dim lights*, one darkens the room and finds out whether the patient sees as long as the examiner and whether his eye becomes dark-adapted as quickly. For quantitative tests, Foerster's photometry or Masson's disc may be employed.

The *determination of the extent of the visual field* for each eye (*perimetry*) is of very great importance in neurological diagnosis. A *rough test* may be made by seating the patient with his back near a window before the examiner; a bandage is placed over one eye and he is told to look with the other into the eye of the examiner opposite (distance, 40 cm.). Closing one eye, one holds the white or colored object (1 cm.) midway between the patient and the examiner and controls by the extent of his own visual field the examination of that of the patient. With a little practice one can detect existing hemianopsias, scotomata, or concentric contractions easily; the method is important for quick preliminary orientation.

For exact registration of the extent of the visual fields, Foerster's *perimeter* or some similar apparatus (*e. g.*, Sydow's) should be used; self-registering perimeters are obtainable, but are not necessary. Care must be taken to avoid fatigue; one should not work too long at one visual field, but determine limits quickly (though accurately), encouraging the patient frequently to give the matter his closest attention. The patient is told that while he may wink as often as he likes, he is to look straight forward at a fixed point and on no account to follow the object with his eye. The objects are brought in from the periphery and the patient announces the time of first appearance and the color; white objects are brought in vibrating, colored objects quietly, from the periphery. No one color field should be determined all at once, but one tries different colors, so that the patient never knows which color is entering the visual field. If a defect (scotoma) be found, its limits may be very accurately localized by using small test objects (2 mm. broad). For testing *color fields*, one usually uses green, red, and blue, the field for the former being smallest, that for the latter largest, under normal conditions.

There are great individual differences, however, in normal color fields, and much depends upon the exact colors used. If a green which is complementary for a red be employed, the fields for red and green are approximately co-extensive.

The technique of *ophthalmoscopic examination* should be learned and regularly practised by every active physician. Through it we are able to look directly at the papilla n. optici, a part of the central nervous system, and, from it, to draw conclusions regarding a whole series of nervous diseases; it is one of the chief aids to neurological diagnosis. The examination in the *inverted image* is more desirable than in the *upright image*, since the technique is easier and a wider view of the retina is afforded; the upright image is, however, very valuable for the study of finer details. As to the papilla n. optici itself, one notes any changes (*a*) in the normally distinct boundaries, and (*b*) in its normally delicate rose color. It is to be borne in mind that the temporal half of the papilla is normally of a somewhat lighter rose color than the nasal, but it is never really white. The centre of the papilla often looks white normally, owing to the "physiological" excavatio papillæ; the periphery is, however, always rose color. Acquaintance with

the normal appearances of the arterioles and venules of the retina, the color of the eye-ground, and the macula lutea should be cultivated, in order that abnormalities may be recognized.

## DISTURBANCES OF SENSATION.

**General Remarks.**—Having reviewed (1) the sensations mediated by the various sense organs and centripetal conduction paths, and (2) the methods of investigating them clinically, we may turn to a general consideration of the disturbances in disease. In certain diseases like tabes and multiple neuritis a whole series of sensory conduction paths may be simultaneously disturbed; in other pathological conditions (*e. g.*, tumor, vascular lesion), one sense organ or one sensory conduction path may alone be affected. In intoxications there may be elective injury with loss or perversion of particular sensory function (*e. g.*, tobacco amblyopia, cinchonism).

It is customary to divide sensory disturbances into subjective and objective. By *subjective* disturbance one means those which are announced spontaneously, in the consciousness of the patient; by *objective* disturbances, those abnormalities of sensation recognized by the patient when he himself or his physician applies adequate stimuli to his organs of sense.

Subjective disturbances are nearly always pathological sensations which arise, owing to (1) abnormal internal stimuli (acting on the sense organ, conduction path, or cortex cerebri), or (2) abnormal increase in excitability of these parts. The pathological sensations may not differ in quality from the sensations producible by adequate stimuli; when they do differ (*e. g.*, formication), they are spoken of as *dysæsthesias* or as *paræsthesias*.[1]

Objective disturbances are usually classed under the headings: (1) *Anæsthesia*, (2) *hyperæsthesia*, and (3) *paræsthesia*, corresponding to (1) decrease or loss, (2) increase, and (3) perversion of sensibility. This is a very crude classification, and must sooner or later be modified and extended. A more rational classification is needed, and we shall doubtless from now on begin to classify anomalies of sensation according to changes in (1) *quality*, (2) *intensity*, (3) *reaction time*, (4) *duration*, (5) *feeling tone*, (6) *localization* or *exteriorization*, (7) *fusion*, (8) *adaptation*, (9) *fatigue*, and (10) *relation to reflex, affective, and associative processes.*

By *anæsthesia* is meant a condition in which an adequate stimulus no longer arouses a sensation. Anæsthesia may be temporary (*e. g.*, hysteria, cocainization) or permanent (*e. g.*, optic atrophy). It may be peripheral in its origin (sense organ, centripetal nerve) or central (conduction path, cortical sense area). Aside from the complete loss of sensation which anæsthesia denotes, conditions of *hypæsthesia* are met with in which the sensations aroused by adequate stimuli are less intense than normal, or the physical stimulus necessary to elicit a just perceptible sensation must be stronger than normal (elevation of intensity threshold).

In *hyperæsthesia* stimuli when applied call forth sensations of greater intensity than normal or a stimulus normally subliminal possesses a

[1] The term *paræsthesia* is used very loosely; some mean by it abnormal sensations like formication; others use it for anomalies in reaction time, fusion, localization, etc.

threshold value (*depression of intensity threshold*). Much of what is designated hyperæsthesia clinically is really an affective state due to *abnormal negative feeling tone* (*e. g.*, the "acoustic hyperæsthesia" of psychoneurotics). The "positive or pleasurable feeling tone" of other sensations may be lost (*anhedonia*), diminished (*hyphedonia*), or increased (*hyperhedonia*); changes here are met with especially in mental diseases.

The *area of sensory surface* in which the anomaly of sensation exists, or so-called *distribution* or *extent* of the anæsthesia or hyperæsthesia, should be accurately determined and its *limits* exactly recorded, since these are of much importance in differential diagnosis, permitting one frequently to locate the lesion in periphery, spinal cord, or brain.

When an organ of sense is diseased it is important also to make sure whether the anomaly concerns all the sense modalities or sense qualities mediated by it (*total anomaly*) or only certain of them (*elective anomaly*). Thus, as an example of an elective anomaly of cutaneous sense, the condition often met with in syringomyelia may be mentioned in which touch sense may be intact when the sense of temperature and pain is abolished.

The *apperception time* for sensation may be pathologically delayed (*e. g.*, delayed pain and touch sensations in hypæsthetic, or more often hyperæsthetic, areas in neuritis and tabes).

The *localization* and *exteriorization* of sensations are sometimes abnormal. Irritation of a nerve trunk is, according to the "law of excentric localization," always referred to the region whence the nerve receives normally its centripetal impulses; thus, when one "hits his funny-bone," the little finger tingles, and in amputation neuromata the disagreeable sensations are referred to the lost member. Again, a stimulus applied to one part of a sensory surface may be localized in another; in tabes, hysteria, and multiple sclerosis a skin stimulus to one leg may be localized in the other (*allocheiria* or *allæsthesia*). When a stimulus applied to one point is properly localized there but gives rise also to a subjective sensation (perhaps of wholly different modality) localized elsewhere we speak of *synæsthesia*.[1]

There may be anomalies of *fusion* of sensations; stimuli which should be felt as continuous and simultaneous may be experienced as successive, and one touch to the skin felt as two or more, or two compass-points as three or four (*polyæsthesia*). This phenomenon, in the writer's opinion, is sometimes due to stimulation simultaneously of a number of touch points; owing to the pathologically delayed apperception time for some of them, the sensation of several separate, successive stimuli is experienced rather than normal unitary sensation, which depends upon fusion of simultaneous stimuli. Here an apparent faulty fusion would in reality be prolonged apperception time of some stimuli, due probably to delayed conduction. On the other hand, fusion of sensations may sometimes occur when normally they would be felt separately (change in "successive threshold"); this is seen in the pain sense of the skin occasionally when a number of pricks applied successively, though not felt at all at first, give rise a little later to a continuous sensation. This is closely allied to the so-called *summation of excitations*.

**Disturbances in Individual Sense Domains.—Anomalies of Pain Sense.**—Subjective anomalies are very common. Spontaneous pains occur in

[1] Smith, H. L., *Johns Hopkins Hospital Bulletin* 1905, xvi, 256. Here belong the so-called sound feeling, color hearing, or sound seeing, and number form.

various organic diseases (*e. g.*, neuritis, tabes, visceral diseases), as well as in so-called functional diseases (psychoneuroses). Neuralgias, cephalalgias, hemicranias, visceralgias, and topoalgias or psychalgias are symptoms met with daily. In studying abnormal pain sensations, the elements of the pain should be carefully analyzed, its relations to space and to time, the exact quality of the pain, its topography, and any accompanying phenomena should be attended to. The modifying influence of position, of motion, of pressure, of food, of drugs and chemicals, and of the organic functions of the body should be looked into. The excellent treatise of Schmidt[1] can be heartily recommended as a valuable guide to the interpretation of pains complained of by patients.

Anæsthesia in the domain of the sense of pain is called *analgesia*, while hyperæsthesia here is called *hyperalgesia*. When spontaneous pains appear in an anæsthetic area, we speak of *analgesia dolorosa*.

**Anomalies of the Deep or Mesoblastic Sensibility.**—Anæsthesia in this domain has been designated *bathyanæsthesia* (Oppenheim). (1) *Anomalies of the sense of position.* Hypæsthesia and anæsthesia are common in neuritis and tabes. If a limb be placed in a certain position, this can be only imperfectly or not at all reproduced by the opposite limb. (2) *Anomalies of the sense of movement.* If anæsthesia exists, passive movements are not felt; if hypæsthesia, greater excursions than normal have to be made to arouse the sensation of movement (elevation of threshold). Not only the extent, but the direction of the movement may be mistaken in these *kinanæsthesias.* As a result, there is disturbance of the coördination of movements, or ataxia. Localized loss is often cortical in origin and helps in topical diagnosis (cerebral tumor, etc.). (3) *Anomalies of the sense of resistance and weight.* This sense is prone to suffer also when the sense of movement is disturbed. Weights are improperly judged and the difference sensibility is altered. (4) *Anomalies of stereognostic perception.* In disease the stereognostic perception may be more or less impaired, even to complete loss (*astereognosis* of C. K. Mills). It may be lost even when the sense of touch, temperature, and pain are normal. Astereognosis is common in hemianæsthesia of cerebral origin, but it is difficult to be sure about it unless the accompanying hemiplegia is so slight as to permit the patient to make the movements necessary in palpation. It is more likely to be present if the hemianæsthesia be cortical (Regio centroparietalis) in origin rather than capsular, pontine, or bulbar. In the cerebral palsies of children the power of stereognostic perception may never develop. Astereognosis will appear in tabes and peripheral neuritis if the sensory conduction paths from the upper extremities be involved. Astereogenosis is sometimes a symptom in hysteria. (5) *Anomalies of the vibration sense* (*osseous sensibility*). Anæsthesia of the bones to the vibrating tuning-fork (*pallanæsthesia*) is common in neuritis, tabes, syringomyelia, hæmatomyelia, Brown-Séquard paralysis, myelitis, cerebral hemianæsthesias, and hysteria (Dejerine).

**Anomalies of the Vestibular and Allied Senses.—Vertiginous Disorientation.**—Disturbances of the vestibular or labyrinthine sense in its two components: (*a*) Sensibility to acceleration in a straight line (sense of the vertical direction and of progressive, non-rotary movements), and (*b*) sensibility to angular

[1] *Pain: Its Causation and Diagnostic Significance in Internal Disease.* English translation by Vogel and Zinsser, 1908.

accelerations (sense of change of velocity of rotary movements)—have only recently had attention more carefully directed to them.

A remarkable phenomenon associated with disturbance (even temporary) of the vestibular sense is an *illusion regarding the state of movement of the body*, a deception as to its momentary stability, in which one has the mistaken sensation either (1) that objects in the environment are revolving around one's body, or (2) that the body is rotating around one of its axes. This sensation, often accompanied by a decided feeling of discomfort, occasionally, even with nausea or vomiting, constitutes the form of dizziness or *vertigo* known as *sham-movement vertigo* (to distinguish it from height vertigo, felt on looking downward from a great height or upward on a steep wall). This sham-movement vertigo appears to be the result of a "*contradiction*" *of one set of sensations by another.* If one of our sense organs signals "the body is at rest," and another signals "the body is in motion," or if the signals conflict regarding movement in the environment, we feel confused, disoriented, dizzy. This *vertiginous disorientation* is most often due to a *discrepancy between the reports* given us by our vestibular sense and those emanating from other sense organs; it is usually associated with a tendency to move the body in a direction opposite to a movement really occurring or to a sham movement, and, indeed, the body is often so moved.

This feeling of vertigo can be aroused by stimulating the N. vestibuli in various ways: (1) By rotating the body around any one of its axes; (2) by moving the body progressively in a curved path (swinging; pitching of a ship); (3) by passing a galvanic current through the head from one ear to the other (galvanic vertigo). Here the abnormal vestibular sensations contradict visual, muscle-joint, and tactile sensibility. But the same sensation can be aroused when vestibular sense is normal through contradictory abnormal sensations of sight or of eye-muscle movement, *e. g.*, whenever binocular vision is suddenly disturbed (eye-muscle paralysis), or whenever the image in one eye is sharply focussed and the other is blurred. Further, in cerebral lesions (local irritation of abscess), cerebral anæmia or cerebral intoxications (acute alcoholism), in all probability, the central stimuli affect sensory neurones of certain modalities more than others, so as to produce contradictory impressions with resulting disorientation.

Obviously, then, any factors which disturb the functions of the vestibular nerve or its central connections or the eye-muscle apparatus (peripherally or centrally) may cause vertigo, especially if the disturbance take place quickly. Many deaf mutes experience no vertigo on rotation, probably owing to loss of function of the labyrinth.

**Anomalies of Other Interior Sensations.**—*Hunger.*—Hypæsthesia and anæsthesia in this domain are met with in the various grades of *anorexia;* hyperæsthesia, expressed as exceedingly strong feeling of hunger, is called *bulimia*, or "wolf hunger;" when this is accompanied by powerfully unpleasant feeling tone, the patient describing it as painful, it is called *gastralgokenosis* (Boas).

The *sense of satiety* may be diminished or abolished (*akoria, aplesia*); patients (tumor cerebri, dementia) may then ingest extraordinary amounts of food and yet remain entirely unsatisfied; they exhibit *polyphagia.* The feeling of *nausea* is probably an affective state rather than a sensory disturbance in the strict sense. *Gastralgia* (including cardialgia and pyrosis) is a disturbance of visceral pain sense.

*Thirst.*—Abnormal thirst occurs in diabetes and often also in the psychoneuroses; it may lead to excessive drinking (*polydipsia*), and is probably central in origin.

*Fatigue.*—Under abnormal feelings of fatigue are often grouped three distinct varieties of sensations: (1) The *painful weariness* or *feeling of being completely "knocked out,"* which is associated even with slight exercise in many nervous patients; (2) the *weakness* which is experienced by patients who have paralysis or paresis of muscles; and (3) the *general lassitude* felt on slight exercise in states of exhaustion, in which slight movements cause palpitation, dyspnœa, perspiration, tremor, and faintness.

*Sexual Sense.*—Anæsthesia of the sexual sense (abnormal *frigidity*) may be present owing either to *loss of libido* or to *dyspareunia;* the libido may be pathologically increased, as in *nymphomania* and *satyriasis;* for various *perversions of the libido*s, pecial texts may be consulted. The hyperæsthesia of the vagina (with painful contraction of the constrictor cunni), known as *vaginismus*, is an anomaly of visceral pain sense and nearly always associated with abnormal affective states.

**Anomalies of the Sense of Touch or Pressure.**—It is only recently that clinicians have begun to separate sharply anomalies of this sense from anomalies of pain sense, owing to the mistaken idea that pain is due to overstimulation of the same nerves which on milder stimulation respond by contact or pressure sensations. Anæsthesia and hypæsthesia of the touch sense very frequently occur; it is doubtful whether true hyperæsthesia of this sense has been observed. The distribution of the anæsthesia or hypæsthesia is important for topical diagnosis, as is also association with (or dissociation from) anomalies of pain and temperature sense.

Spontaneous itching (*pruritus*) is regarded as a dysæsthesia (or paræsthesia) of the touch sense by some, as are also the "creepy sensations" known as *formication*. Pruritus appears to be due to pathological stimuli affecting the nerve endings in the skin (intoxications), formication rather to pathological stimulation of nerve trunks in their course, the sensations being referred to the periphery (law of excentric localization). Special forms of formication are met with in *acroparæsthesia* (tingling and pain at ends of extremities), in *meralgia paræsthetica* (dysæsthesia in domain of N. femoris lateralis), and in *erythromelalgia* (painful redness of legs). In these various conditions the touch sense is rarely involved alone, pain sense and the temperature senses may also be involved, and the writer considers that the peculiar qualities of sensations known as dysæsthesias (or paræsthesias) are due to the unusual combinations of sense modalities which result from pathological stimuli.

**Anomalies of the Temperature Senses.**—Subjective feelings of chilliness or cold or heat are common in infections (*e. g.*, initial chill), and in the psychoneuroses ("hot flushes"). The feeling of heat (especially in the back) which occurs in paralysis agitans is well known, and patients suffering from exophthalmic goitre frequently complain of abnormal sensations of heat. In *cutis anserina* the "chilliness" contains besides its cold component also a factor due to the stimulation of the touch points by contraction of the musculi pilorum. In objective disturbances the *thermanæsthesias* usually involve cold sense and warmth sense simultaneously, but this is not necessarily the case; there may be loss of cold sense (kryanæsthesia or alganæsthesia) without change in warmth sense, or there may be loss of warmth sense (*ardanæsthesia*) without involvement of cold sense,

the latter dissociation being more frequent than the former. *Thermhypæsthesias* are common; *thermhyperæsthesia* is rare, much that is described as such being in reality hyperalgesia.

**Topographical Distribution of Disturbances of Sensation.**—Disturbances of cutaneous and deep sensibility, especially studied accurately as to their topographical distribution on the surface of the body, throw much light upon the site of the lesions which give rise to them. In the peripheral nerve trunks and in the roots of the spinal and cerebral nerves the fibers which carry centripetal impulses are more intimately mixed with one another than are their intracentral communications, and this explains why it is that dissociations, although they occur in peripheral and radicular lesions, are more common when the pathological process is located in the spinal cord or cerebrum.

**Anomalies of the Sense of Taste.**—Anæsthesia in this domain is known as *ageusia*, hypæsthesia as *hypogeusia*, and hyperæsthesia as *hypergeusia*. If one or more of the four fundamental qualities of taste sensations remain intact when others are abolished, we speak of an *elective ageusia*. Aside from lesions of the peripheral neurones and certain gustatory hallucinations, the pathology of the sense of taste is as yet a *terra incognita*. Various perversions of the sense of taste have been described under the designation *parageusia*.

**Anomalies of the Sense of Smell.**—Here, similarly, we have to deal with *anosmia*, *hyposmia*, and *hyperosmia*, which may be total or elective. When an anosmia is due to interference with the passage of odoriferous air currents toward the regio olfactoria it is called an *anosmia respiratoria*, a subdivision of which is the *anosmia gustatoria* when the odoriferous currents from food and drink by way of the choanæ are obstructed. Anosmia may be due also to disease of the regio olfactoria itself (tobacco poisoning; syphilis) or to intracranial lesion of the olfactory nerves, bulbs, tracts, or centres. The left-sided anosmia which sometimes accompanies right-sided hemiplegia and aphasia is said to be due to lesion of the lateral root of the olfactory tract. In hysteria a unilateral "functional" anosmia frequently accompanies, or rather forms a part of, the hysterical hemianæsthesia. Anosmia may be a congenital anomaly, and hyposmia is common in imbeciles, although not constant, some imbeciles appearing to be hyperosmic.

**Anomalies of the Sense of Hearing.**—Total deafness is known as *anacusis*, partial as *hypacusis*, while abnormally sharp auditory acuity, rarely met with, is *hyperacusis* or *true hyperæsthesia acustica*.[1] The painful sensations in the ear felt when noises or tones are heard not only in diseases of the middle and internal ear, in otitic paralysis of the N. facialis, in trigeminal neuralgia, in migraine, and in many psychoneurotic states has been called *dysacusis* (*false hyperæsthesia acustica*); in facial paralysis it takes especially the form of increased sensitiveness to noises, and has been called *oxyakoia*. The so-called *paracusis Willisii*, met with especially in sclerosis of the middle ear, is of but little importance neurologically; the name is used when a deaf person subjected to an acoustic stimulus ordinarily infraliminal for him hears the sound when other sound stimuli (noises) are acting simultaneously; thus, many deaf people hear better when travelling in a train than when at home. The term paracusis has also been used to mean the hearing of a tone

[1] The latter form has unfortunately been applied to the pain, or negative feeling-tone experienced by psychoneurotics when noises or even sounds of any kind are heard.

as although higher or lower in pitch than it really is; when one ear hears the tone of normal pitch and the other higher or lower, we speak of *diplacusis.*

But little is known as yet regarding auditory disturbances due to *lesions of the central auditory paths;* the latter, owing to the semidecussations at the corpus trapezoideum, are laid down bilaterally, the lemniscus lateralis, etc., on each side containing conduction paths from both ears. Focal lesions in the pons and midbrain (involving the lemniscus lateralis or the colliculus inferior) or higher up (involving the medial geniculate bodies or the auditory radiations) cause disturbances of hearing on both sides (not complete deafness on either side). Total unilateral deafness is sometimes caused by *neurofibroma of the N. acusticus,* but usually only after the tumor has attained to a considerable size; the deafness is here due chiefly to the lesion of the peripheral (not the central) auditory neurones, although it may be due in part to involvement of the nuclei terminales N. cochlearis (dorsal and ventral). Cortical lesions involving the auditory sense areas in both temporal lobes must be widespread to cause total "*cortical deafness,*" a clinical fact in accord with the experiments of Munk.

**Anomalies of the Sense of Sight.**—Anæsthesia in this domain is known as *anopsia,* hypæsthesia as *hypopsia,* and hyperæsthesia as *hyperopsia.*

Anopsia (*amaurosis* or *blindness*) or *hypopsia* (*amblyopia*) may involve the whole of one eye or of both eyes. Or one-half of each retina or its conduction paths may be blind, leading to loss of half of the visual fields (*hemianopsia*); if it be the two right halves or the two left halves of the visual field which are lost, we speak of *hemianopsia homonyma* (due to lesions of visual sense area in the cortex, of occipitothalamic radiation, or of one tractus opticus), while if it be the two medial (nasal) halves or the two lateral (temporal) halves (the latter due to chiasm lesion) of the field, we call it *hemianopsia heteronyma.* If a hemianopsia involve the color sense only or chiefly, it is called a *hemichromatopsia;* if vision be present in half the field but dimmed, the condition is designated *hemiamblyopia.* A hemianopsia is said to be *total* when the line separating the blind area from the area in which the field is still visible passes perpendicularly through the fixation point; if the visual field is not so limited, but encroaches upon the blind area, the hemianopsia is *not total* and the area of encroachment is called the *surplus field* (Wilbrand). Sometimes this surplus field is so large as to occupy a whole quadrant of the visual field and then a *quadrantic hemianopsia* is spoken of. There are even references in the bibliography to sextantic and octantic hemianopsias; when the loss in the visual field is as small as this, however, it is probably better to call it a *scotoma,* as this name is applied to small areas of loss or defect in the visual field, such as the *blind spot,* which exists normally. It is customary, however, to reserve the term scotoma for a defect in the visual field which does not reach the periphery. A *central scotoma* is one situated at or near the fixation point; if it involve only sensations of red and green, it is called a *relative scotoma* (*e. g.,* in chronic form of retrobulbar neuritis, limited to the papillomacular bundle, most often seen in alcoholism or tobacco poisoning), while if no light be sensed in the defect it is said to be an *absolute scotoma* (met with in diabetes, multiple sclerosis, and occasionally "idiopathically").

When the visual field is restricted regularly at the periphery we speak of *concentric restriction or contraction of the visual fields;* when this is due to progressive diseases of the Nn. optici (primary and secondary optic atrophy) the contraction proceeds from the periphery centralward, the central acuity

for white and colors (especially green and red) also suffering early; the vision is "fogged;" then marked disturbances of orientation supervene. The beginning and progress may be very insidious in simple (so-called primary) progressive atrophy (tabes; dementia paralytica), while the fogginess and darkness may come suddenly in neuritis (meningitis; lues). In choked disk (tumor, lues, brain abscess, hydrocephalus) vision may remain good for some time; if the cause persists, there will be gradual loss, due to atrophy; the color sense may go first, and, finally, there may be complete loss of the visual field. Bordley has made a careful study of the color sense in cases of increased intracranial tension, especially when the latter was due to tumor.

It is interesting that the contraction of a visual field due to glaucoma is especially marked in the medial field, the last of the field to go being an excentric temporal area (*glaucomatous visual-field slit*); in contrast to what happens in simple optic nerve atrophy the color sense remains long intact in the retained field and the central visual acuity is longer maintained (Axenfeld).

In detached retina there is contraction of the field corresponding to the degree of detachment; this may in daylight affect, at first, colors only, especially blue, although in dim light the contraction is absolute.

Recent retinitis due to nephritis or diabetes frequently injures central vision and may give rise to central scotomata of different kinds corresponding to the various patches and hemorrhages; the periphery is less often injured.

In retinitis pigmentosa and in the retinal atrophy following syphilitic chorioretinitis there may be either marked concentric contraction of the visual field, or, in cases in which the degeneration attacks an intermediary zone of the retina, a so-called *ring scotoma* which later extends both peripheralward and centralward. Central vision may, despite this, be long retained as an "island" of good visual acuity and good color vision, the patient seeing as though through a tube of small caliber. Similar limitations of the visual field are met with in hysteria and in traumatic neurosis; here, however, by testing first at a distance of 10 cm. (and marking limits with chalk) and then at a distance of a meter, the visual field does not correspondingly enlarge, the psychoneurotic state here behaving like that of a simulant.

In contrast with the *immobile positive scotomata* (due to diseases of the retina or visual path) above described, the *mobile positive scotomata* (movable shadows) due to turbidity of the corpus vitreus should be borne in mind. In neurasthenics and myopics these *muscæ volitantes* may be present when the vitreus is normal (*myodesopsia*); the threads, flocculi, veils, chains, etc., seen in certain cases may, however, be due to pathologically formed elements in the vitreus visible with the ophthalmoscope. Important for neurologists, too, is the so-called *fortification scotoma* of migraine; a positive and sharply localized sensation of glimmering followed by darkening due to irritation of the retina or visual sense area.

Sudden *blindness of one eye* may be due to embolism or thrombosis of the arteria centralis retinæ. Other *sudden disturbances of vision* may occur in (1) cerebral hemorrhage involving the central visual paths (hemianopsia associated with other symptoms of cerebral apoplexy); (2) retinal detachment (disturbance beginning at periphery of visual field, preceded by characteristic irritative phenomena); (3) choked disk (obscurations temporary). Less sudden but rapid blindness may occur in (1) very acute inflammatory glaucoma (excruciating unilateral headache); (2) large hemorrhages into the

corpus vitreus (red or green stripes before the eye precede the blindness); and (3) acute retrobulbar neuritis.

The terms nyctalopia and hemeralopia need explanation. When a patient can see better in dim light (or when the eye is in shadow) than in bright light he has *nyctalopia* (*e. g.*, in beginning cataract, the widening of the pupil in dim light leaving more transparent lens uncovered). When, on the other hand, a patient does not see at all in dim light, or only after long adaptation, *hemeralopia* or *night blindness* exists (retinitis pigmentosa; chorioditis diffusa; glaucoma; retinal detachment; general disturbances of nutrition).

By *photopsia* is meant an abnormal sensation of color or light due to retinal lesions, or to central irritation, by *metamorphopsia*, the distorted vision, occurring in lesions of the retina and chorioid, in which straight lines look curved or jagged, or letters seem out of place. The term *macropsia* is used to designate the symptoms "of seeing things too large" (*e. g.*, strained accommodation), while *micropsia* is the term for "seeing things too small" (*e. g.*, paresis of accommodation); both macropsia and micropsia may be met with in the psychoneuroses.

In many functional nervous cases a study of anomalies of refraction has an important bearing upon etiology and treatment. We distinguish the normally refracting eye (*emmetropia*) from the short-sighted eye (*myopia*) and the far-sighted eye (*hyperopia, hypermetropia*). When in order to secure normal visual acuity it is necessary to use not simple spherical lenses, but lenses which correct certain meridians only, the condition of *astigmatism* exists; if the curvature in each meridian is equal throughout its course, but the curvatures of different meridians vary so that the meridians of greatest and least curvature are approximately at right angles to each other the astigmatism is *regular;* when the vertical meridian has the greatest curvature it is astigmatism *with the rule*, but when the horizontal meridian has the greater it is astigmatism *against the rule*. The astigmatism is *irregular* either when the meridians of greatest or least curvature are not approximately at right angles or when the curvature in a single meridian is irregular.

Anomalies of vision due to *disturbances of accommodation and convergence* may also be mentioned here, although they belong more properly to disturbances of motility. If near vision is faulty, or cannot be maintained long, *asthenopia* exists. If this is due to faulty power of accommodation, it is known as *accommodative asthenopia*. The most frequent form of this is due to the progressive sclerosis of the crystalline lens, which normally diminishes the power of accommodation as life advances, and soon after the fortieth year the near-point of a normal (emmetropic) eye comes to lie more than 25 cm. distant (*presbyopia*). Accommodative asthenopia due to paralysis of the M. ciliaris often follows diphtheria.

Another form of defective near vision is *muscular asthenopia*, especially that due to faulty convergence (most often seen in incorrected myopia) with consequent faulty binocular near vision (*convergence asthenopia*).

The lesions of the eye, most important for the neurologist, discoverable by the *ophthalmoscope*, may be briefly referred to here. In optic neuritis the papilla looks turbid, grayish red in color, its margins are indistinct, and there are often minute hemorrhages in or near it and the veins may be tortuous and dilated. It is most often due to cerebral disease, especially when bilateral. If the papilla be swollen (2 diopters or more) and enlarged, we speak of *choked disk;* it is most often due to increase of intracranial pressure (brain

abscess, hydrocephalus, syphilis, tumor).[1] *Unilateral optic neuritis* occurs in diseases of the orbit, in various infections and intoxications, and in beginning tumor, etc. Optic neuritis may be followed by optic atrophy; the papilla has indistinct margins, but becomes whiter as the swelling goes down; the arteries gradually narrow and have white sheaths (*neuritic optic atrophy*).

*Retrobulbar neuritis* occurs in two forms—acute and chronic. The acute form is seen in rheumatism and in multiple sclerosis, the chronic in diabetes, in various intoxications (tobacco, alcohol), and sometimes when its cause is not known. Here the ophthalmoscopic picture is very different from that described above for "optic neuritis;" no distinct alteration can be seen in the papilla N. optici for some time, although after a while a retrograde atrophy leads to pallor, especially on the temporal side of the disk.

When the papilla N. optici looks white, grayish white, or greenish white instead of its normal rose color there is atrophy of the optic nerve (*atrophia N. optici*); ophthalmologists distinguish several types: (*a*) Simple atrophy, (*b*) retinal atrophy, (*c*) glaucomatous atrophy, and (*d*) neuritic atrophy.

Of *simple optic atrophy* in which there is simply pallor of the disk with retention of its well-outlined margins and but little change in the vessels, three main types are described: (1) *That occurring in tabes and dementia paralytica*, the changes in the disk being visible from the beginning of the visual disturbance; (2) *retrograde atrophy*, following upon diseases at base of brain, *e. g.*, tumors, chiasm lesions, sphenoidal-sinus disease, etc., the changes in the papilla becoming visible through the ophthalmoscope only after the visual disturbance has lasted some time; and (3) *atrophy after occlusion of the A. centralis retinæ*, in which the simple atrophy differs from (1) and (2) by the existence of a high grade of vascular constriction.

The so-called *retinal atrophy of the optic nerve* is characterized by a yellowish-white turbid papilla, constricted bloodvessels, and degeneration of the peripheral retina; it occurs in diffuse degenerations of the retina (chorioido-retinitis; retinitis pigmentosa).

The form of *atrophy accompanying chronic glaucoma* is very characteristic. The increased intra-ocular pressure leads to "glaucomatous excavation," and this after a time is followed by atrophy with abrupt kinking of the bloodvessels at the margin of the papilla.

Of the *diseases of the retina* revealed by the ophthalmoscope, the more important for the neurologist are: (1) Acute retinitis; (2) atrophy of the retina; (3) detachment of the retina; and (4) subretinal cysticercus.

Of the *diseases of the chorioidea* discoverable by the ophthalmoscope, those which interest the neurologist especially are: (1) *Chorioiditis disseminata*, and (2) *miliary tuberculosis;* in both one sees gray or yellowish-gray rounded spots with ill-defined margins; some of them may be crossed by retinal bloodvessels.

**Anomalies of Sense Perception Known as Illusions, Hallucinations, and Pseudo-hallucinations.**—Normal *sense perception* is more complex than mere sense impression; it involves *association* and *assimilation*. An "object" is composed only in small part of sense impressions directly received by way of the peripheral sense organ, but very largely of revivals of impressions earlier experienced (*reproduced images; representations*). A *percept* arises largely through the arousal of a host of old experiences by a few newly

[1] See Cushing and Bordley, *Johns Hopkins Hospital Bulletin*, 1909, xx, 95.

entering sensations; perception is not, as it may seem, the mere entrance of a group of new sensations. The percept as thus constituted depends, especially in its meaning or the value which it has in consciousness, upon the general direction of thought or upon the most outstanding idea in the immediately preceding moment; this is often, though inadequately, expressed by the term *attention*.

We can profitably pass on to the consideration of the anomalies of sense perception known as *sense deceptions*, including illusions, hallucinations, and pseudo-hallucinations. In all these sense deceptions there is an "abnormal relation between the objective and the subjective setting, and the factors that control the course of the associations are not suited to the circumstances of the physical world." Events occur in accord with the laws governing normal perception, but the conditions are unusual and individual, not those which obtain with the mass of healthy people.

By an *illusion* we mean a sense perception in which the subjective factor takes too large a part in the process of assimilation, so that the mental supplementation of the sense impressions entering into it leads to a result contradictory to the reality as determined in more accurate examination with the same sense organ or on correction by control through other senses.

By an *hallucination* we mean a sense perception in which the abnormal relation between subjective and objective conditions is very pronounced; objective sense impressions may be entirely absent or present in unusually small amount and dissimilar to the content of the hallucination, and yet the sense perception is vivid, possessing for the patient the ear-marks of objective reality, being definitely localized in perceived space; the annihilation of the perception normally localized in this space is unexplained.

By a *pseudo-hallucination* we mean a psychic experience which resembles a sense perception in intensity and in its involuntary appearance (thus, different from a mere image, idea, or representation called up by association), but differing from a normal sense perception, from an illusion, and from an hallucination in that the patient notices the absence of the character of objectivity.

We have then a series of transitionally related psychic phenomena beginning with sense impressions at one end and pure reproductions at the other, the series consisting of (1) sense impressions, (2) normal sense perceptions, (3) illusions, (4) hallucinations, (5) pseudo-hallucinations, and (6) pure reproductions (ideas, images, representations). In normal people in ordinary circumstances, the sense deceptions (illusions, hallucinations, and pseudo-hallucinations) do not occur; they are experienced occasionally by normal people in extraordinary circumstances and frequently in mentally abnormal people in ordinary circumstances.[1]

**Illusions.**—The psychic experiences known as *illusions* are best studied in connection with the sense of sight and hearing, since with these senses one can be sure as to the character of the external stimuli acting. Sounds heard and sights seen are misinterpreted owing to the incorrect supplementation (by

[1] Some of the more important contributions to the general bibliography of sense deceptions are the following: Kahlbaum, *Die Sinnesdelirien Allg. Ztschr. f. Psychiat.*, xxiii, 1–86; Parish, E., *Hallucinations and Illusions*, 1897; Sully, J., *Illusions*, 1883; *Text-books on Psychiatry* by Krafft-Ebing, Kraepelin, and Paton have good chapters on the subject. The discussion in Störring's volume is excellent.

reproductions—images; ideas) of the sense impressions received. A melancholic patient, hearing the whistle of a locomotive engine, says that she hears the cry of her child being murdered, or, seeing a relative, fails to identify him as such, but takes him to be a stranger and impostor. Even normal people are often the subjects of illusion when the sense impressions are feeble and the mind is in a state of expectation; for example, an anxious mother may take the mewing of a cat for the cry of her sick child in a distant room. But the stronger and more distinct the sense impressions when illusions occur, other things being equal, the greater the degree of abnormality indicated. Deficient attention to sense impressions (as in alcoholic delirium) and short duration of the sense impressions favor the origin of illusions. Tense expectation, emotional states or moods, a lively imagination, or pathologically increased facility of reproducing ideas will also favor illusion, for in these circumstances certain ideas are prone to he held focal in consciousness and they will obviously have the best opportunity for coöperating with any sense impression which may enter. Where some idea is prominently in mind and the sense impression, simultaneous with it, gives rise to a sense deception, we speak of the idea or image as primary and the sense impression as secondary, the illusion resulting from the combination being an *active illusion* (Sully) or an *apperception illusion* (Kraepelin); where the entering sense impression finds no special idea focal in consciousness (the expectation or emotion existing having no definite reference), but by association arouses some image which, owing to the general state of consciousness at the moment, is particularly easy to reproduce, the sense deception resulting is said to be a *passive illusion* (Sully) or a *perception illusion* (Kraepelin).

In insanity, illusions are probably just as important as hallucinations, the sense deceptions being due to the delusional ideas which are in consciousness at the time of entrance of the sense impressions.

**Hallucinations.**—These may be elementary or complex. An *elementary hallucination* is one in which the subjective experience approaches that of a simple sense impression (*e. g.*, a flash of light, a simple sound), while a *complex hallucination* is one in which the subjective experience approaches that of a compound sense perception (*e. g.*, vision, voice). When the hallucinations in the domain of a single sense are uniform in character, recurring monotonously in the patient's experience, they are called *stabile hallucinations* (Kraepelin's "perception phantasms"), experienced by many neuropathic individuals just before going to sleep, in which they see a certain flower, an animal, or a face, or hear a word pronounced always in the same manner; when the hallucinations vary greatly in their content they are spoken of as *labile*. If one sense only is involved at a time it is a *unisensual* hallucination; if two or more senses are involved simultaneously, as when the hallucinant sees the figures of the people whose voices he hears, the hallucination is *disensual* or *multisensual*. Hallucinations and illusions conform to the law of excentric projection or exteriorization of sense perception; the patient "projects" what he sees, hears, or feels into the external visual, auditory, or tactile fields, or localizes the sensation either in space outside or in various parts of his body. When a hallucination occurs in one sense domain as a result of actual peripheral stimulation of another sense domain it has been called a *reflex hallucination* (Kahlbaum).

Hallucinations seem even more real to those who experience them than do normal sense perceptions; their power is irresistible; the patient is

compelled to believe in them, the judgment being quickly overcome by them. This peculiar feature is due in part to the sensory distinctness, the seeming objective reality of the experience, but much more to the fact that hallucinations most often correspond to the pathological ideas of the patient, to his abnormal thoughts, fears, or wishes. Thus, although sense deceptions often play a part in the origin of delusions, some persistent direction of thought (*Aufgabe*) is still more often provocative of sense deceptions. Further, the strong feelings and emotions co-existent with the hallucinations go far toward giving the latter their power in the mental life.

Among *visual hallucinations* the elementary types are represented by flashes of light or color, stars, flames, and the like, the complex types by *visions* in which, especially at night, illuminated animal and human forms appear, or God, angels, the Devil, or various horrible shapes may be seen; if the hallucination occur in the daytime, which is less common, the patients complain of seeing a black dog, the face of a corpse looking in at the window, and the like. Sometimes in hemianopsia the visions are projected to the blind halves of the visual fields; occasionally, as in convalescence from alcohol delirium, visions which have begun to disappear may be recalled by pressure on the closed eyes (Liepmann).

Even more influential in mental life than visual are *auditory hallucinations*, the elementary forms of which are called *akoasmata*, the complex forms being known as *phonisms* or *voices*. The akoasmata consist of indefinite noises of different sorts—rustling, murmuring, thundering, bell-ringing, and the like; the phonisms or voices are often whispered words, more rarely words or brief sentences spoken aloud. The voices make unpleasant references to the patient, they insult him, abuse him, or threaten him ("rascal," "thief"). They may emanate from the patient's children, who are being murdered by an enemy, from a revengeful lover, from an annoying neighbor, or it may be the Deity or Satan who addresses him. Occasionally a voice may bring happiness and inspiration, but this is unusual; nearly always the voices irritate and pain, arouse suspicion and defence, cause anxiety or confusion; they may command, and the patient may be led to the most unnatural acts in the belief that he is obeying the order of a supernatural power. The patients can tell the distance and direction from which the voices come—they localize them in space—a fact closely related to the objectivity (perception value) which attaches to the voices. If the voices are heard in one ear only, there is usually abnormal stimulation of the auditory path on that side, and the hallucination (or illusion) can often be called forth by electric stimulation.

A word must be said about *audible thinking* (*Gedankenlautwerden* of the Germans), not of the delusional form of it in which the patient is convinced that his thoughts are audible to other people, although he does not hear them himself, but of the hallucinatory form in which a man hears his own thoughts, perhaps in his feet or in the ticking of a clock or the ringing of the doorbell. The patient hears the voices only when he pays attention to them. Sometimes a patient's thoughts seem doubly existent to him, first, in the normal way and then also as audible voices (*double thinking*); sometimes the voice dictates what the patient writes or it may repeat what the patient reads; occasionally the voice precedes the reading. Many of these "inner voices" are examples of pseudo-hallucination rather than of true hallucination.

It is extremely difficult to distinguish *olfactory* and *gustatory hallucinations* from illusions of these senses, and the same is true of *hallucinations of visceral sense*. They are, however, of great practical importance, for they often account for the refusal of food. If the food smells of fæces or foul sweat, one can scarcely blame a patient for not eating it. In the domain of the cutaneous senses, *tactile* or *haptic hallucinations* and *thermal hallucinations* are not infrequently met with, the former especially in toxic psychoses; some of the subjective dysæsthesias may be hallucinatory in nature, as may also certain so-called *psychalgias* or *topoalgias* (*hallucinations of pain sense*). Much study has been devoted also to *hallucinations of the movement* and *position senses*, sometimes referred to as *kinæsthetic hallucinations*. They lead to the maintenance of peculiar attitudes and also affect the voluntary movements, being sometimes the cause of imperative positions or monotonous defensive movements. The hallucinations may concern the eye-muscle movements, the locomotory movements, or the speech-muscle movements. Sometimes there are *hallucinations of vestibular sense*, the patient having the sensation of progressive or rotary movement. Doubtless many impulsive acts and various psychomotor inhibitions are traceable to hallucinations as a cause; in epilepsy and alcoholism there may be an irregular *hallucinatory agitation*, sometimes combined with paroxysms of rage, in which acts of atrocity may be committed.

While the hallucinations above described are subjectively equivalent to perceptions, the patients believing that they are dealing with something objective, this is not true of what Hagen has called *pseudo-hallucinations* (psychic hallucinations of Baillarger; apperception hallucinations of Kahlbaum; imagination deceptions of Kraepelin). Faces, printed pages, yellow roses, groups of people appear for a moment and vanish, to be followed by others not logically connected with them. They seem to stand before the eyes, but are not at all related to the black field of vision of the closed eyes. Despite their sharp contours and lively colors, the images do not possess the character of objectivity; the patient says he sees them with "inner eyes," not with his "outer eyes." The images come up involuntarily and remain, even when the patient desires to be rid of them; the details of the vision are clear to him without any feeling of internal activity on his own part, nor can he alter the detail by voluntary effort of imagination. Although the objects seen "stand before the eyes," they are not related to any real objects about him. In pseudo-hallucinations of hearing the patients complain that they are "forced to hear internally" (not with their ears) things which they least wish to hear. Obviously, although these psychic states resemble perceptions on the one hand, and lively reproductions of mere ideas on the other, they differ from both. They differ from percepts in that the patients recognize the lack of objective reality (physical) and their independence of the movements of the peripheral sense organ concerned; they differ from ordinary reproductions (images, ideas) in that they have greater intensity and are independent of the will.

## DISTURBANCES OF MORE COMPLEX PSYCHIC PROCESSES.

**Anomalies of Consciousness as a Whole.**—Consciousness is the fact that we have experiences—sensations, ideas, feelings, etc. To understand

consciousness, therefore, we must understand experience fully. In spite of ages of speculation we are certainly, as yet, only making a beginning in the understanding of experience, in that we are gradually determining some of the conditions under which it occurs. These conditions are (1) partly of the order of physical facts (*e. g.*, light-waves, sound-waves); (2) partly of the order of physiological facts (*e. g.*, processes in sense organs, nerves, and central nervous structures); and (3) partly of the mind itself (*e. g.*, imagination, feeling, volition).

As ideas come and go in consciousness we are aware that some of them are more prominent or outstanding than others. We commonly speak of *paying attention* to these more prominent ideas. Subjectively this prominence of certain ideas is, at times, definitely accompanied by a peculiar feeling of "activity," a sense of strain or effort, which is in contrast to the feeling of "submission" (passivity) which accompanies a distracting sensation or memory. When ideas have the prominence just referred to we feel that they are *ours* in a peculiar sense—that we are *doing* something with them. In analogy with the visual field, we often refer to these prominent ideas as being in the focal part of consciousness (*Apperception* of Wundt), while the other ideas are marginal (*Perception* of Wundt). Thus according to Wundt, "Perception" (not *Wahrnehmung*) denotes the mere appearance of a content in consciousness, while "Apperception" denotes its reception as a content which is being related definitely to other facts—in a word, which is being *thought about*. While no sharp line can be drawn between "Apperception" and "Perception" in Wundt's sense, still there can be no doubt that in the extremes the difference indicated exists.

By the *mind* we usually mean not simply the abstract *state of consciousness* or the *total content* or *field of consciousness* at any given moment; we include in it also that unity with past experience through which the present moment has a meaning and value, dependent on the whole education of the individual which makes it truly personal. When we distinguish between the *activity of the mind* (Bewusstseinstätigkeit) and the *content of the mind* (Bewusstseinsinhalt) we should recognize that an abstraction is being made between two aspects of every idea which have no significance apart from one another.

In the central neurones changes take place which permit the calling up of memory pictures in simple and complex groups. The totality of memory pictures possibly revivable is what is designated as the *contents of the mind.* This totality of possible memories is divisible into three parts: (1) That which refers to the *external world;* (2) that which refers to the *body* or *soma;* and (3) that which refers to the *personality* of the individual. Wernicke has given to these three parts of the mind's contents the names *allopsyche*, *somatopsyche*, and *autopsyche.*

The *allopsyche*, or portion of the mind's contents which concerns the external world, is made up of memories of concrete things, the result of sense impressions, their combination into perceptions, and the shorthand epitomes of these which we call concepts and notions. The allopsyche of an individual will depend then largely upon his sense organs; that of the congenitally blind will differ greatly from that of the congenitally deaf, and that of the anosmic will be wholly other than that of the ageusic. Given brains of equal birth, the allopsychic contents will vary quantitatively according as the bearer is a resident of a city or a lonely shepherd or backwoodsman, or may differ qualitatively with the environment (*e. g.*, tropical or arctic).

The *somatopsyche* or portion of the mind's contents which concerns the body consists of all the memories which represent residues of previous visceral, muscular, fascial, and cutaneous sensations. In all probability the so-called "local sign" which accompanies the exterior sensations is to be counted as a part of the body consciousness, the local sign of a visual sensation representing, for example, an "organ sensation" of the retina (Wernicke). Here the allopsyche and the somatopsyche overlap.

The somatopsyche depends largely upon the condition of the viscera, and the tendency to-day is to relate the hypochondriacal symptoms of mental disease to anomalies of visceral sensations.

It is probable that in every allopsychic sense perception the somatopsyche plays a part; indeed, this somatopsychic participation may be the main difference between a perception and a revived image of memory, for in the perception an organ sensation (local sign, feeling tone) is present while it is absent in the memory pictures (Wernicke).

The various projection fields for the somatopsyche must be intimately connected with one another by associative neurone systems. The connections of the memory pictures concerning the body are much more intimate than those uniting the memories of concrete things of the external world on account of the unalterable relations of the different parts of the body as contrasted with the continuous changes in the world outside the body. In sleep the impressions from the external world cease to affect the brain, although bodily impressions are still entering it. The external world is at times cut off; the body is always there. When the body is undergoing marked changes, as at puberty, the climacteric period, there must be a marked alteration in the character of the afferent impulses from the viscera. Is it any wonder, then, that disturbances of somatopsychic consciousness should tend especially to prevail at such periods?

The relative constancy of bodily sensations, the continuity of the somatopsyche, as compared with the variety of sensations from the external world and its discontinuity, give rise in each individual to a conception of unity, of indivisibility, of the body consciousness (*primary ego* of Meynert).

The *autopsyche* or portion of the mind's contents which concerns the personality or individuality is something built upon the allopsyche and the somatopsyche which serve as its foundations. The autopsyche or *consciousness of personality* includes all the mental possessions and acquisitions resulting from instruction, culture, and education, the influence of the family and of the social milieu. Speech and written language play an important part in its development. School knowledge is among the early acquisitions of the autopsyche; later on come all the experiences peculiar to the individual. The interest in work, in family, in friends, and in other people belongs here. The development of the autopsyche depends largely upon the kinds of allopsyche and somatopsyche which form its foundations; as Wernicke puts it, "consciousness of personality is a function of the consciousness of the external world and of that of the body. . . . The man with sharp senses and strong body must develop a mental personality different from that of a man with dull senses and a weak body." An individual's own estimate of his place in human society depends upon the state of his autopsyche. Among the striking anomalies in this domain may be mentioned ideas of grandeur or of self-depreciation, ideas of reference and delusions of persecution.

There seems to be no doubt that the somatopsyche, the allopsyche, and the autopsyche are, to a certain degree at any rate, localizable; or, more exactly, their physiological correlates appear to be to some degree localizable in the cerebrum. Clinical and pathological studies of dementia paralytica, of cerebral arteriosclerosis, etc., show us how the autopsyche, the allopsyche, and the somatopsyche may separately or successively become involved.

**Circumspection; Confusion; Unconsciousness.**—In the normal awake state there exists a certain equilibrium in perceptions, ideas, and feelings, the external manifestation of which is known as *circumspection* or *presence of mind* (*Besonnenheit* of German writers); here the mind is calm and collected with its faculties ready at command. It is the opposite of *mental confusion* (*Verwirrtheit* of German writers) or *incoherence*, in which ideas are jumbled and chaotic, the mind perplexed or bewildered, the speech aimless or unintelligible, the acts disconcerted. By *unconsciousness* is meant a state in which the psychical powers are wholly in abeyance (dreamless sleep, profound narcosis). Attempts have been made to distinguish various grades of pronounced lowering of consciousness, and references will be found to a series of states designated in order as *somnolence*, *sopor*, *coma*, and *carus*.

**States of Psychical Cloudiness or of Clouded Consciousness; Mental Fog; Twilight States; Automatism.**—By *mental fog*, *psychical cloudiness*, or a *twilight state* (*Dämmerzustände* of German writers) is meant a condition in which there is diminution of clearness of consciousness or a loss of memory for a part of past experiences or both together. Such mental fog is often met with in epilepsy and in hysteria. In *epileptic mental fog* there are often fluctuations of consciousness intensity, partial loss of power of recalling the past, alterations of mood and false identification. In *hysterical mental fog* we meet with the so-called *monoideic* and *polyideic somnambulisms* and *fugues*. In somnambulism an individual thinks and acts in an odd way, different from that of other people, while he is in some way like a person asleep. During a *monoideic somnambulism* the patient acts as though in a dream, but the phenomena of the dream are most perfectly and intensely developed.

In *fugues* (*flights or ambulatory automatisms*) we have to deal with the hysterical mania for running away. During the abnormal state a certain idea or train of ideas develop to an exaggerated degree (attempt to elude capture; desire for travel, etc.), and lead to the flight. At the same time other thoughts which would normally counteract this idea or train of ideas appear to be suppressed; the patient may forget his name, his family, his social position, or, in other words, his ordinary personality. After the fugue is over and circumspection returns the patient has forgotten many or even all of the events of the flight, and also the ideas which dominated him during it. Through hypnosis it is sometimes possible to reproduce the mental state of the fugue artificially, and so to study it at will.

Intermediate between fugues and monoideic somnambulisms are the so-called *polyideic somnambulisms*. These are characterized by the multiplicity of the ideas that fill them. Instead of the one idea or event of the monoideic type, we see evidence of the existence of a series of ideas succeeding one another in the somnambulic state, ideas which pertain to some strong feeling which develops independently of the rest of consciousness. Where still larger systems of ideas and feelings develop independently we get the manifestation of what are known as *double personalities* or *multiple personalities*.

Twilight states are usually temporary, but they may last for varying lengths of time; they may last for weeks or months. The cessation of the state is, as a rule, sudden, the patient waking as though from sleep. Indeed, sleep seems to be the physiological paradigm of a twilight state, dreams being the analogue of the psychical activity, but differing from the consciousness of the twilight state in that usually they are unaccompanied by motor reactions.

**Disorientation.**—Many patients are not well oriented as to their own persons and their spatial and temporal relations. For differential diagnostic purposes it is very important to determine the presence or absence of certain other symptoms at the same time orientation is tested, especially anomalies of mood, ideas of persecution and hallucinations. A few simple questions along with those concerning orientation may quickly give the clue to the existence of a hallucinatory confusion, of a paranoid state, or of some other well characterized type of mental disorder. Especially important for the general practitioner is the valuation of the syndrome in which disorientation as regards time and space is combined with so-called pseudo-reminiscences, in which the patient narrates long tales of recent events as though perfectly true, which on inquiry are found to have no basis in fact (*Korsakow's syndrome, polyneuritic psychosis*).

**Anomalies of Attention.**—The importance of disorders of attention in psychiatry is coming to be very generally recognized. The power of directing thought toward a definite task (*Aufgabe*) (*vigility*), and of maintaining this task despite intercurrent stimuli (*tenacity*), are essential in all intellectual operations. We meet with most variable ability to direct and maintain the task, some being able to do this easily, while others are easily distracted. The greater the distractibility the more dirigible from without, the less thorough is the intellectual work. The term *hyperprosexia* is by some reserved for the pathological persistence of a task in which the mind is concentrated upon certain abnormal ideas or sensations (hypertenacity), while *aprosexia* is defined as a state in which there is inability to set or to maintain a task (*e. g.*, in imbecility). There is both hypovigility and hypotenacity. Some writers use the word *hyperprosexia* to indicate a state in which there is increased tendency to change the task set (*hypervigility*) with diminished tenacity. It would be well if psychiatrists would agree upon the definition of these terms, as the different sense in which they are employed leads to much confusion.

**Examination of the Recording Faculty.**—For testing the *recording faculty*, which depends in large part upon attention, much simpler methods must be used in medical practice than in experimental psychological examinations, and we have to thank Ranschburg[1] and Boldt[2] for supplying us with such simple methods. It is necessary to test the recording faculty in several different domains, and most experiments consist of acoustic and optic tests. Thus, in one group of experiments the acoustic word-memory, using the principle of idea associations, may be tested. The patient memorizes fifteen pairs of words, of which five represent customary word compounds, like dog-kennel, chair-leg; five according to the laws of causality (spatial and temporal coincidence), like fish, pond; day, night, etc.; and five according to the principle of similarity of sound, like wound, wonder; die, dynasty. In another

[1] *Monatsschr. f. Psychiat. u. Neurol.*, Berlin, 1905, ix, 241 to 259.
[2] *Ibid.*, 1905, xvii, 97 to 115.

group of tests the memory of persons is tried by using twenty-five portraits, from which the patient is to recognize five. In a third group the memory for colors may be tested. Many colored skeins of red, blue, green, and yellow wool are shown and a definite example of each is to be recognized. In a fourth group the orientation memory is tested. One shows the patient a chart containing six simple geometrical figures, exposes it for twenty seconds to permit of certain perception, and after a time the patient is required to pick out these six figures from a chart containing twenty or twenty-two figures. In a fifth group the memory for nonsense words is tested, five pairs of words being chosen, *e. g.*, lum, rar; grat, smor. In a sixth group of tests the memory for names is tried. The patient is shown five photographs and the name and surname given. Later on the patient picks out these five photographs from a group of twenty-five and tries to give correct name and surname. Lastly, in a seventh group the memory for numbers is tested. Some number is given to each one of the five fingers, and later on, naming each finger, he is asked to give the corresponding number. Suitable methods for testing the recording faculty for touch sensations, smell, and taste will probably later be worked out. The reproduction is best tried shortly after the perception (five or fifteen minutes) and again after twenty-four hours.

The recording faculty consists of two factors, namely, perception and reproduction. Of course, where perception is deficient there can be no reproduction later. One makes sure, first, that the task is correctly understood. The patient is warned not to try to reproduce repeatedly in his mind the things perceived, for even in normal persons the test turns out less favorably later when such efforts are made. The first reproduction is permitted after five minutes have elapsed, the second after fifteen minutes, and twenty-four hours later the third test is made. The reproduction at the end of twenty-four hours may in normal persons be much better than that at the end of five minutes, but in patients with injured recording faculty this is not the case. The recording faculty appears to be the one of the mental powers first and most seriously to suffer in dementing processes. Injury to the recording faculty as manifested in loss of memory for most recent events never exists long alone; there are added to it very quickly other characteristic symptoms of mental deterioration. The recording faculty is most efficient in childhood especially between the twelfth and the fourteenth years of life.

When the recording faculty begins to be impaired it seems to be the memory for numbers which goes first. As Kraepelin has emphasized, studies of this recording faculty have drawn the attention of clinicians to a group of anomalies previously entirely unknown to us, viz., (1) the quick fading of perceptions, and (2) the slow development of perceptions, both of which appear especially to favor the origin of memory falsifications.

**Anomalies of Ideation.**—Great importance attaches to the examination of the processes of ideation; that is, to the formation and association of ideas. Ideation may be disturbed on the formal side, or there may be falsification of the content of ideas. Of the formal disturbances of association may be mentioned: (1) Anomalies in velocity; (2) anomalies in the order of association or mode of reaction; (3) anomalies in the intensity and duration of ideas; (4) anomalies in comprehension or apperception; (5) anomalies in so-called identical reproduction (memory); (6) anomalies in so-called original production (phantasy). The falsification of content of ideas is manifest in the production of the so-called insane ideas or delusions.

**Anomalies in the Velocity of Association; Flight of Ideas; Thought Inhibitions; Stupors.**—The association of ideas may be pathologically accelerated or pathologically retarded. Even in health there seems to be an *acceleration* of the velocity of association during excitement, especially during joyful excitement. A feebler associative impulse suffices to awaken the memory, to raise it, so to speak, above the psychic threshold.

In maniacal states we meet with a peculiar form of association known as *flight of ideas* (*Ideenflucht* of the Germans), which is often associated with the form of motor agitation known as *pressure of activity* and accompanied by marked gesticulation and rapid speech (*logorrhœa*). In a careful study of the flight of ideas, however, it is found that we have to deal less with acceleration of association than with an alteration in the mode of reaction. The quicker thinking of the maniacal patient seems rather due to the fact that in the same time unit more different objects pass through consciousness than in normal states (Liepmann). In the healthy person ideation is dominated by some principal idea, while in mania the process of association is not controlled by such a dominant idea. One of the most striking characters of the associations in the so-called flight of ideas is the frequent *change of direction of ideation*.

In normal man the kind of reproduction of ideas and the duration depend not only upon the reproduction tendency present in the individual, but also upon the effect of *concentration upon the task set*. Consciousness is, in a way, set, determined, or prepared in the sense of a specific task. In mania the task set has but little effect upon the process of association as compared with healthy states. The healthy man goes only very gradually from one ideational direction to another and tends to remain for a longer time within the confines of the idea made dominant by the task set. The healthy man, it is true, may for a short time experience rapid associations frequently changing in direction, the quality of the association then being superficial, like those seen in flight of ideas; but he is incapable of so continuing to associate for any length of time, whereas the characteristic of flight of ideas is the continuance of such associations until the physical strength is exhausted.

At bottom the disturbance known as flight of ideas must be largely a disturbance of attention. The main change in the attention seems to be an increase in the capacity for directing the thought quickly and energetically to single ideas (hypervigility), and also away from them again (hypotenacity). To explain the abundance of ideas reproduced, however, it is necessary to assume also an alteration of the power of reproduction, permitting the appearance in consciousness of a wealth of lively ideas capable of being easily replaced one by another (Isserlin).

Psychiatrists distinguish a *primary flight of ideas* (*e. g.*, reminiscence flight in neurasthenic and hypochondriacal states and in the exaltation of mania) from a *secondary flight of ideas* due to quickly changing hallucinations and delusions; in the latter case the idea flight is not a new disease symptom, but is secondary to the affective excitation called forth by the hallucinations or delusions. When the associations run unbridled by a dominant idea, as they do in the marked forms of idea flight, there is an ever-increasing inability to follow anything like a normal train of thought (*secondary incoherence*).

Pathological *retardation* of association (*thought inhibition*) may also appear, either as a primary phenomenon or as a secondary symptom. When pronounced it leads to the condition known as *stupor*.

Such slowed association is nearly always associated also with *motor*

*inhibition* in which the so-called voluntary movements are slowed or absent altogether. The patient is unable to complete the series of associations between the initial idea and the terminal motor idea. Speech becomes more and more slowed until there may be complete mutism. The various voluntary muscles of the body may be held rigidly (as in catatonic rigidity).

As examples of *primary stupor* may be mentioned (1) the so-called excitation stupor which appears in the slowly developing conditions of cerebral excitation, and (2) the stupors which appear in the course of various intoxications. Unless the history is known in such cases it is sometimes difficult to tell whether one is dealing merely with an inhibitory process or with actual defect (dementia), as *secondary stupor* may be designated the form which accompanies or follows various states of psychic depression. An emotional shock or a paroxysmal anxiety condition may call forth a severe secondary thought inhibition. A special form of secondary stupor due to delusions and hallucinations accompanied by strong affective states is met with in certain conditions of catatonic rigidity (pseudo-stupor of Westphal).

The so-called waxy flexibility (*flexibilitas cerea*) may be regarded as a part of the general inhibition of association.

For actually *measuring the velocity of associations* (determination of reaction times) psychologists have provided us with a number of very exact methods. Many of these are too complex for use with nervous and mental patients, and experience teaches that the ordinary "answer method" with the aid of a stop-watch which marks one-fifth of a second is sufficient for most purposes. The examiner explains the method to the patient, calls the test word, and at the same moment starts the stop-watch; the moment the answer is heard he notes this and the time required.

In collecting a series of reaction times it is well to establish a *middle position* and a *middle zone* of times, according to Kraepelin's suggestion.

**Anomalies in the Order of Association or Mode of Reaction.**—Perhaps most progress in recent years has been made in the study of the *mode of reaction* in association tests. Psychologists have taught us that even in health, associations of different types occur. A study of diseased conditions reveals marked deviations from the normal modes of reaction. Anyone working in this field should early inform himself of the researches of Kraepelin, Aschaffenburg, Jung, Sommer, and Isserlin. It is customary to classify the kinds of association as follows:

A. Sense of test words correctly understood.
   - (*a*) Internal associations. (1) Associations of coördination and subordination. (2) Predicative associations. (3) Causality associations.
   - (*b*) External associations. (1) Associations of spatial and temporal co-existence. (2) Identities. (3) Speech reminiscences.

B. Sense of test words not understood.
   - (*c*) Test words acting only through their sound. (1) Word supplements. (2) Sound associations and rhyme associations. (*A*) Intelligible. (*B*) Nonsensical.
   - (*d*) Test words acting only by setting free reactions. (1) Repetition of test word. (2) Repetition of earlier reactions without sense. (3) Associations for words used earlier. (4) Reactions without recognizable connection.

It is also important to note how many of the reactions are more *objective* in nature and how many of them are more *subjective* (*egocentric*).

A word of explanation as to the meaning of some of the terms used above seems desirable. By an *internal association* is meant one directed toward the sense of the test word, while an *external association* is one dependent upon custom, habit of speech, and sound relation. In the *predicative associations* there is some affirmation or assertion regarding the object of the test word. In the *causality associations* there is a relation of cause and effect. By *word supplementation* one means only such reaction words as, taken with the test word, make together an indivisible word. Such reactions belong, of course, to *clang reactions* in the wider sense.

A distinction is made between *mediate association* and *immediate* (Aschaffenburg). In the former the only way one can understand a connection between the test word and the reaction is by assuming some intermediate member of an association series. As a rule, such intermediate member turns out to be a clang association for the test word, while the relations of the reaction word to the intermediate member may be any one of the forms of association mentioned.

It is customary to designate instances in which more than sixty seconds elapse before an answer is given to the test word as "*faults.*" Reactions in which there is simply a repetition of the test word are in a class by themselves. It is well also to count up the number of perseverations and repetitions which occur. By *perseveration* is meant a reaction in which there has been evidently a connection on an earlier test or reaction word instead of to the actual test word given; by *repetition* is meant a reaction in which the test or reaction word immediately preceding recurs.

*Discursive associations* should also be especially noted (*Weiterschweifen* of Isserlin). By this is meant a spontaneous continuation of the association. Such further association may be intelligible or nonsensical.

Special attention should be paid to the instances in which the test word is repeated by the patient. When this is a marked feature in a case it should be determined whether it is due to anomalies of comprehension from marked distractibility, in which the attempt to concentrate leads to repetitions, or whether it is dependent upon inability to draw upon the reproductive elements sufficiently, that is to say, is due to a lack of associated ideas (as in the thought inhibition of depressive states). A patient will often give the clue himself, since he may mention, on the one hand, that nothing occurs to him, or, on the other, that so many things pass through his mind that he repeated the test word in order to answer correctly. The two types are further distinguishable by the fact that, in the one, reproductions often in large number appear immediately after the repeated test word, while in the instances due to thought inhibition associations occur only after quite a pause, and often only after several slow repetitions of the test word.

In the selection of a list of test words certain principles should be borne in mind, including (1) the principle of the uniform stimulus, (2) limitation of total number of tests so as to avoid fatigue, (3) selection of words which will bear upon a whole series of different groups of ideas.

Sommer has prepared three lists, one containing adjectives pertaining to the different sense domains (including light and colors, extent and form movement, feeling, temperature, sound, smell, taste, etc.), the second a list of words referring to ideas of objects, the third list including designations

for things which are associated with affective states or actual terms for such affective states, and also words referring to conditions of the intellect, consciousness, and social affairs.

TEST WORDS FOR ASSOCIATION EXPERIMENT I.

I.

1. Light.
2. Dark.
3. White.
4. Black.
5. Red.
6. Yellow.
7. Green.
8. Blue.

II.

1. Bright.
2. High.
3. Deep.
4. Thick.
5. Thin.
6. Round.
7. Angular.
8. Sharp.

III.

1. Quiet.
2. Slow.
3. Quick.

IV.

1. Rough.
2. Smooth.
3. Solid.
4. Hard.
5. Soft.

V.

1. Cold.
2. Tepid.
3. Warm.
4. Hot.

VI.

1. Low.
2. Loud.
3. Shrieking.
4. Yelling.

VII.

1. Fragrant.
2. Stinking.
3. Mouldy.

VIII.

1. Sweet.
2. Sour.
3. Bitter.
4. Salt.

IX.

1. Painful.
2. Ticklish.
3. Hungry.
4. Thirsty.
5. Nauseating.

X.

1. Beautiful.
2. Ugly.

TEST WORDS FOR ASSOCIATION EXPERIMENT II.

XI.

1. Head.
2. Hand.
3. Foot.
4. Brain.
5. Lungs.
6. Stomach.

XII.

1. Table.
2. Chair.
3. Mirror.
4. Lamp.
5. Sofa.
6. Bed.

XIII.

1. Stairs.
2. Room.
3. House.
4. Palace.
5. Town.
6. Street.

XIV.

1. Mountain.
2. River.
3. Valley.
4. Sea.
5. Star.
6. Sun.

XV.

1. Root.
2. Leaf.
3. Stem.
4. Flower.
5. Bud.
6. Blossom.

XVI.

1. Spider.
2. Butterfly.
3. Eagle.
4. Sheep.
5. Lion.
6. Mankind.

XVII.

1. Man.
2. Woman.
3. Girl.
4. Boy.
5. Children.
6. Grandchild.

XVIII.

1. Peasant.
2. Citizen.
3. Soldier.
4. Pastor.
5. Doctor.
6. King.

TEST WORDS FOR ASSOCIATION EXPERIMENT III.

XIX.

1. Disease.
2. Unhappiness.
3. Crime.
4. Need.
5. Persecution.
6. Misery.

XX.

1. Happiness.
2. Reward.
3. Kindness.
4. Health.
5. Peace.
6. Joy.

XXI.

1. Alas!
2. Oh!
3. Fie!
4. Ha!
5. Hello!
6. Ouch!

XXII.

1. Anger.
2. Love.
3. Hate.
4. Light.
5. Fear.
6. Fright.

XXIII.

1. Impulse.
2. Will.
3. Command.
4. Wish.
5. Activity.
6. Decision.

XXIV.

1. Reason.
2. Insight.
3. Wisdom.
4. Intention.
5. Knowledge.
6. Stupidity.

XXV.

1. Consciousness.
2. Sleep.
3. Dream.
4. Recollection.
5. Memory.
6. Thoughts.

XXVI.

1. Law.
2. Order.
3. Custom.
4. Right.
5. Justice.
6. State.

Practice has shown that it is better not to use the test words in groups, as Sommer first recommended, but to make the series so that single words of different groups follow one another. Jung has especially emphasized the importance of this on account of the emotional relations of certain of the test words.

Experience with these association tests soon permits one by their aid to differentiate quickly among the great groups of psychotic symptoms. Another advantage of systematic monthly association tests consists in the ability to follow the exacerbation or retrogression of psychopathic symptoms.

In valuing the results of association tests one should familiarize himself, first, with a large material obtained from healthy people. Educated people show, on the average, a more "superficial" type of reaction than uneducated people, the latter reacting more to the sense of the test word than the former. Differences in individuality determine also certain important variations in association. Jung and Riklin distinguish two main types: (1) *Objective types*, and (2) *subjective types with egocentric adjustment*. To the objective types belong those persons by whom the test word is understood objectively, either according to its sense or as a speech stimulus, while to the subjective types belong (*a*) the so-called "*constellation*" *types*, in which personal experience (idea complexes) with strong feeling tone appear in the associations, and (*b*) the "*predicate*" *types*, which present lively, subjectively valued ideas.

The reaction time of normal persons varies greatly according to the age, education, and individuality. The more phlegmatic and the intellectually feebler individuals show, as a rule, longer reaction times. The quality of the association is an important factor in its duration; thus the more complicated an association is psychologically the longer the reaction time. The associations which are most used in daily life are those with the shorter reaction times, a fact which explains the velocity of "external" associations as contrasted with "internal" associations. One of the most important results of association tests is the bringing of the proof of the delay in reaction time in associations to which *idea complexes with strong feeling tone* are attached.

Association tests in the *melancholic states of manic-depressive insanity* reveal a marked prolongation of the reaction time and apparently a characteristic change in the content of the associations. There appears to be a limitation to idea change. The reaction type is usually predominantly objective. Even in the very delayed associations when the reaction is recorded, it is as a rule found to correspond in sense to the test word.

In the *maniacal states of manic-depressive insanity* the striking features are a "flattening" of the associations and the replacement of associations due to the sense of the test words by associations which depend upon custom, speech, and mere sound relations (clang associations). The frequency of discursive associations (Weiterschweifen) in mania has been pointed out by Isserlin. As in flight of ideas in general the reaction times are not shorter than normal; indeed, they are sometimes really longer, although in maniacal states one does meet perhaps with a greater number of short reaction times than in health. The most characteristic phenomenon in mania is the greater number of single ideas produced during a given time—many more than one ever meets with in health. It is the extraordinarily frequent *change of direction* of the ideas which is so striking. Healthy persons are incapable of associating so quickly with such frequent change of direction and in such a "superficial" manner as is characteristic of the idea flight of maniacal states.

Very recently progress has been made in the study of associations in the so-called *mixed states of exaltation and depression*, in which certain of the signs of mania are associated with certain of the signs of depression. One tries to find out whether the reaction time is prompt or delayed, whether the type of reaction is according to the sense of the test words or superficial, whether the direction of the ideas is changed frequently or rarely, whether there are many or few perseverations and repetitions, whether the form of the answers indicates a precise reaction or a vague "talking around the subject," whether the feeling tone is positive or negative, and whether or not egocentric relations, faults, and test-word repetitions are prominent (Isserlin). In the "mixed states," egocentric relations may be extremely frequent. Test-word repetitions tend also to be especially common in these mixed states.

In view of the help which comes from a study of the results of association tests, some physicians seem inclined to overestimate their value. It should be borne in mind that they reflect only one side of mental life, and that one must not neglect other psychological methods of examination. The general practitioner has not yet formed the habit, however, of resorting to association tests, and to him the method can be heartily recommended as a means of extending the anamnesis. Especially in the differential diagnosis of manic-depressive insanity from dementia præcox the method will be found helpful. If one bears in mind the characteristic signs of inhibition and excitation revealed in the patient's *entrance upon the task* of association in manic-depressive insanity, and compares therewith the refractory, perverse behavior with regard to the task characteristic of the patient suffering from dementia præcox, and observes, further, the stereotyped replies and repetitions or perseverations which may seem to disregard the test words, the paralogia, and mannerisms which come out during the association tests, one will find the method one of the best available in distinguishing these two types of psychosis from one another. Even in cases where strong inhibition simulates apathy and dementia the association experiment helps to differentiate. If, in spite of long reaction times, we see an effort to understand the sense of the test word, we can, as Isserlin emphasizes, make the diagnosis of manic-depressive insanity even when the change of direction of the ideas is only moderate and there seems to be relatively little affectivity. In dementia præcox the patient reveals himself by the reaction time (ordinarily not so evenly prolonged as in the depression of manic-depressive and, when not normal, more lawlessly desultory) and by the apathetic or perverse behavior toward the task set.

The association tests also help in differentiating the depressive states of manic-depressive insanity from some forms of *hysteria*. In the latter disease it is especially the emotional phenomena, the signs of the idea complexes emphasized by feeling (*gefühlsbetonte Komplexe*) which are characteristic. In manic-depressive insanity the inhibition is demonstrable aside from the slowing effect upon the reaction time of such emotive complexes, and, further, the associations in general in this psychosis present a much less capricious and variable appearance. It must not be forgotten, however, that hysteria and manic-depressive insanity may be combined in the same patient.

**Anomalies in the Associative Connection of Ideas (Incoherence or Dissociation).**—Normally, the course followed in associations from the initial idea to the terminal idea is a well-regulated one, depending upon some dominant or superior idea. In many psychoses this normal connection of ideas in the associative processes is disturbed. The initial idea may be followed by a

second idea which stands in no recognizable relation to it, an anomaly designated by psychiatrists as *dissociation* or *incoherence* of association. If a patient be asked what time it is, and she answer "fox," a severe disturbance of this sort is manifest. In milder cases the disturbance is manifested by the fact that the patient continuously loses the thread of thought. When a patient is disoriented as to time, place, and personal relations, along with general incoherence of association, the movements and speech of the patient being correspondingly disturbed (*motor incoherence*), the condition is technically known as *confusion* (*Verwirrtheit* of the Germans). Very often incoherence and confusion are used as synonymous terms.

It is customary to distinguish a *primary* from a *secondary incoherence*, the former appearing autochthonously, independent of any other psychopathic symptom (*e. g.*, in the so-called acute hallucinatory confusion or amentia), the latter as a sequel to other psychopathic phenomena (*e. g.*, flight of ideas, hallucinations, strong emotions, dementias, etc.). The secondary dissociation or incoherence is, therefore, common to many severe mental disturbances. The process of loosening or dissociation has been designated *sejunction* by Wernicke. When a dissociation occurs suddenly and lasts only a short time, there result lacunæ in consciousness (the so-called *psychic eclipses* or *deliquia* of L. Meyer), which may form the starting point of manifold disturbances of the content of the ideas. The differential diagnosis of the different forms of incoherence is often very difficult.

In mild instances of general incoherence a peculiar symptom is sometimes met with, known as *pathological notions* (*Einfälle* of the Germans). In the midst of a well-ordered and often extensive thought series, there comes suddenly and apparently without reason an idea or complex of ideas which stands in no connection whatever, by external or internal association as far as can be seen, with the former. The patient betrays the presence of such pathological notions by a sudden word or a peculiar gesture.

**Anomalies in the Intensity and Duration of Ideas.**—Instead of the ordinary course of associations, single ideas or idea complexes may attain to abnormal strength and be very frequently repeated in consciousness. Here belong the *exaggerated* or *hyperdynamic ideas* (*überwerthige Ideen* of Wernicke) and the *imperative ideas* (*Zwangvorstellungen* of Krafft-Ebing), sometimes known as *obsessions*. Such ideas arise especially in individuals who are constitutionally psychopathic at times when they are mentally or physically exhausted. They are especially characteristic in the so-called *psychasthenic states* (Janet).

Wernicke distinguishes between hyperdynamic ideas, imperative ideas, and autochthonous ideas. The *exaggerated* or *hyperdynamic ideas* are characterized by the fact that the patients do not recognize them as intruders in consciousness; on the contrary, the patient regards them as the expression of his innermost nature, and in battling for them he is struggling for the maintenance of his own personality. They are, nevertheless, often felt as troublesome, and the patients frequently complain that they can think of nothing else. *Imperative ideas* or *obsessions*, however, are recognized as unjustifiable ideas, and are often spoken of as absurd. The *autochthonous ideas* have the attention forcibly directed toward them and are felt as troublesome intruders. Wernicke separates them from imperative ideas, since the latter are not regarded as foreign to the personality of the patient, and hence are not serious for the mental life of the patient as are autochthonous

thoughts. The closest relations between hallucinations and autochthonous ideas have been pointed out by various psychiatric writers; indeed, transitions between the two sometimes may occur, the hearing of voices being sometimes preceded, for example, by a stage in which autochthonous thoughts are present.

Many of the imperative ideas or obsessions are combined with marked affective disturbances, giving rise to the so-called *phobias;* thus, a patient may fear to cross an open place (*agoraphobia*), may fear shut-in places (*claustrophobia*), may fear contamination (*mysophobia*), may fear disease (*nosophobia*), or may be afraid of everything (*panphobia*).

When the same idea comes up over and over again the patients may be led to stereotyped methods of expression. The patient may answer a first question correctly, but may tend to give the same answer to various other questions subsequently put (perseveration).

In contrast with hyperdynamic ideas are the *hypodynamic ideas.* Certain experiences which in normal life may be well remembered and attended to are lost sight of or do not seem to have their normal value in the patient's mental life.

**Anomalies of Reproduction or Memory (Hypermnesia; Hypomnesia; Amnesia; Paramnesia).**—The power of reproduction may be pathologically facilitated (*hypermnesia*), diminished or retarded (*hypomnesia*), abolished (*amnesia*), or perverted (*paramnesia*).

Hypermnesia is often met with in exalted or maniacal states and is usually associated with abnormal affective phenomena. Past acts, feelings, or ideas appear vividly in the mind, which in its natural state may have wholly lost the remembrance of them. Ordinary individuals sometimes experience hypermnesia in sleep or in certain unnatural conditions, *e. g.*, under the influence of drugs (alcohol, morphine).

Weakened memory or hypomnesia may betray itself in the increased length of time it takes to recall an image or in the vagueness of the reproduction.

In outspoken pathological diminution or destruction of the power of memory (amnesia) there is complete inability for reproduction of a part or the whole of past experiences. The term amnesia is by some reserved for complete loss of memory for a certain period; for example, that of dreamless sleep, of epileptic coma, of hysterical somnambulism, and the like. When the amnesia concerns not only the period of the pathological condition itself, but extends to a longer or shorter time preceding the attack, it is called *retrospective* or *retrograde amnesia.* The term *anterograde amnesia* has been used to designate the defect of memory which extends to occurrences of the period subsequent to the termination of the clouding of consciousness, a condition doubtless due to the depression of the recording faculty and the injury to the associative processes following the period of mental fog. When in the period subsequent to the termination of the mental fog the patient still remembers certain experiences, but soon after forgets them entirely, the condition is spoken of as *retarded amnesia.* Among the most interesting amnesias are those of the *alternating personalities* of hysterical patients, in which two, three, or more alternating states of consciousness develop, of which the first and third and second and fourth may stand in close associative connection with one another, while in the second and fourth states complete amnesia for the experiences of the first and third exists.

The amnesia may be partial, involving only the events of the recent past and not those of the distant past. Especially in progressive failure of memory, such as that met with in dementia senilis and in dementia paralytica, there is a gradual loss of memories, beginning with the most recent ones and involving gradually those of the past (*law of regression* of Ribot).

*Circumscribed amnesias* in special domains are met with very frequently in focal diseases of the brain. Thus, when optic memory images can no longer be called up, the condition is known as *mind blindness* (*Seelenblindheit* of the Germans). The mind-blind person may still have intact visual sensations, but he is unable to recognize what he sees because his visual memories are lost or, at any rate, cannot be awakened in the ordinary way by retinal stimuli, since the association between the primary optic centres in the cortex and the areas concerned with visual memory are interfered with. Two great groups of cases can be distinguished from one another. In the first group we have to deal rather with a disturbance in optic perception than in optic memory. In this group belongs the so-called *apperceptive form of mind blindness* of Lissauer, which corresponds to the so-called *cortical blindness* (*Rindenblindheit* of Munk). In this group the principal disturbances are hemianopsia, diminution of visual acuity, and disturbance of color sense. Stereoscopic vision is also sometimes involved. On the psychic side there is usually an incapacity for orientation in space. There need not, however, be any marked disturbance of the visual memory or of the capacity for recognizing ordinary objects. Reading and writing may be nearly intact.

The second group of cases includes *mind blindness proper*, which is less a disturbance of perception than a loss of capacity for the intellectual valuation of retinal images. The condition is spoken of by Lissauer as the *associative form of mind blindness*. The patient is unable to recognize objects shown him because the visual images do not call up by association the memories which permit their identification, and so they appear to him as strange confusing figures (*optic asymbolia*). A patient looking at his wife, may know that she is a woman and see the details of her form and clothing exactly, but yet not recognize that she is his wife.

If very circumscribed areas in which optic memories are localized or the pathways to them be destroyed, the phenomena of word blindness or of optic aphasia may be met with. In *word blindness* or *alexia* there is an incapacity to read, although speech and the understanding of speech may be retained. The power to write may or may not be present. In the former case one speaks of *subcortical* or *pure alexia* (probably due to lesion of the associative paths between the visual sense area in the cortex and the sensory speech areas). In the *optic aphasia* of Freund, a rare condition, objects held before the patient are seen and recognized but cannot be designated, although the patients can otherwise speak well and can find the particular word concerned when the stimulus is awakened through some other sense organ. It appears to follow most frequently upon lesions at the junction of the left occipital and left temporal lobes interrupting the association paths which extend from the two occipital lobes to the centre for word-clang memories.

A similar isolated loss of acoustic memorial images is known as *mind deafness*. The patient may hear everything, but cannot recognize the sounds. The most frequent form met with is that known as *word deafness* in the *sensory aphasia of Wernicke*. The patient hears the word, but no

longer recognizes it and cannot understand it (lesion of the first temporal gyrus on the left side). Here, too, we probably have to distinguish a group of cases in which the disturbance is *perceptive* and a second group in which it is *associative*, the latter being sometimes referred to as *amnestic aphasia*.

When tactile memorial images can no longer be awakened we have *mind anæsthesia for touch*. The patients feel contact, but cannot recognize objects through it. Similarly, there may be a loss of the memorial images for kinesthetic sensations.

Under the term *paramnesia* come the falsifications of memory, including what Sully has described as memory phantasms and memory illusions.

In the *memory phantasms* the patient seems to remember things which he has never experienced. The combinations of pure phantasy are regarded as actual experiences. They seem to be due to a loss of critical power, and depend upon enfeebled judgment. Here must be placed the tendency to confabulation and pseudo-reminiscences, so common, for example, in Korsakow's psychosis.

By *memory illusions* the memorial images themselves are falsified, the disturbance being due to faulty reproduction and lively fancy or the falsifying influence of a temporary affective state. As Sully puts it, the past now appears in the colors of the present. Such patients may appear to be liars, since they unconsciously distort their experiences in reproduction. A very good example of such illusions of memory is often seen in connection with the uncinate gyrus fits of H. Jackson.

A special form of memory deception is seen in the identification of a present situation with one presumably previously experienced. This *sentiment du déja vu* is one of the most interesting paramnesias met with among psychasthenics. It seems to be as much a disturbance of perception as of memory. The patient, feeling that impressions escape him, comes to the belief that he is experiencing memories.

**Methods of Testing Memory.**—The tendency among neurologists at present is to treat all memory loss not as a disturbance of a unitary capacity of the brain, but rather as disturbances of a large group of single capacities. It is true that the memory as a whole, that is, the conglomeration of all these partial faculties, may be impaired particularly in some of the psychoses, but in neurological conditions in which circumscribed lesions of the cerebrum are met with we see more often injuries of the partial memory faculties. On testing the memory clinically, therefore, one must use methods which permit us to judge not only of the memory power in general but of the capacity for and fidelity of reproduction in each individual domain.

It is best to begin by testing the memory for fresh impressions or for recent events. One determines a defective power for the immediate acquisition and retention of ideas (recording faculty), and also defective capacity for the acquisition of general and relative ideas (power of abstraction). In addition, it is necessary to test the memory for older impressions or for events which have occurred earlier in the life of the individual, and here one does best to examine the patient's memory for what he has learned at school and to try his power of calculation. In testing school memories, one should have some knowledge of the extent to which the patient has been educated, as obviously the testing of illiterate persons for school memories would be useless. It is advantageous to use a uniform set of questions in

order that comparisons of value may be made among the replies received from different patients. It is customary, therefore, to ask the patient to repeat a number of series which in school are learned by heart: for example, the alphabet, the numerals, the names of the months, and the names of the days of the week. Certain national and religious facts which everyone may reasonably be expected to know may also be inquired into. To these questions may be added others regarding well-known facts of geography and history. Normal answers do not necessarily indicate mental normality, since in non-paralytic dementias, for example, school memories are often very well retained. In dementia paralytica, however, there are usually marked disturbances of memory for simple series of letters and numbers. In catatonic dementia there often seems to be a loss of school memories, although this is more often dependent less upon lack of knowledge than upon the grotesque irrelevancy exhibited by such account of his strenuous assertion of incapacity, while in maniacal states ignorance may be simulated by the associative discursiveness which the patients manifest. An imbecile may give fluent and correct answers to a number of questions which involve the automatic reproduction of a series of patients in replying to questions, the so-called paralogia. In melancholia the patient may appear to have lost his memory for school knowledge on numbers or names.

The solution of the problems themselves is not the only important point. The time taken to solve them and concomitant phenomena of physiognomy and speech should be taken into account. The use of these methods has demonstrated that certain phenomena recur typically in different groups of clinical cases, and Sommer especially recommends the methods for the differential diagnosis of different forms of dementia. Through their application one obtains not only a more exact insight into the partial functions of the power of calculation, but also can give mathematical expression (where tests are repeated at intervals in the same cases) to the symptoms of periodic variation, of advancing intellectual disintegration, of stereotypism, etc.

**Falsification of the Content of Ideas (Delusions).**—The power of forming judgments and conclusions by means of the association of ideas depends in health upon the existence of certain regular relations between the various memories derived from preceding sensations regarding the body and the external world. Judgments depend, however, not entirely upon one's own personal experience, but upon tradition and upon the ideas dominant in the circle in which one lives at the time. One's own personal knowledge is supplemented by certain prevalent beliefs (religious, scientific, political, etc.). On account of the stronger feeling tone which accompanies beliefs their influence upon the power of judgment can scarcely be overestimated. While it is true that men of rich individual experience with strong powers of observation and analysis may attain to a high power of independent judgment; still no one, even the intellectually greatest, remains uninfluenced by belief, for individual experience, no matter how extensive, must be supplemented by beliefs which bridge over the gaps of experience. Everyone is, therefore, liable to make mistakes due to prejudice and superstition, and there is every transition from the mistaken judgments of human beings whom we call normal to the *delusions* or *false judgments* of the insane. In both instances the false judgment arises as a result of combinations of ideas which are out of accord with the facts of the so-called external world. The mistakes of the normal man can, however, be corrected by subsequent judgments depending

upon new perceptions and more correct inferences, but the insane delusion is not amenable to such correction. Even in people who are not insane there may be certain false judgments which cannot be corrected. People brought up in an atmosphere of strong prejudice and superstition may be incorrigible in their beliefs and yet not be insane in the strict sense. Believers in Christian Science, in spiritualism, and the like may form false judgments, but we would hesitate to call them insane. The false judgments are to be regarded as insane delusions when associated with other undoubted phenomena of alienation.

Insane delusions are divided into *primary delusions* and *secondary delusions*. The former usually arise from abnormal states of feeling in connection with a pathological exaggeration of the consciousness of personality. The latter are, as a rule, explanation delusions or attempts on the part of the individual who has passed through a psychosis with injury to his mental processes to explain the changes in his consciousness.

In the primary delusions egocentric ideas dominate the consciousness and lead to the formation of false ideas of reference—that is to say, to false ideas regarding the individual and the processes of the external world. Such false ideas may, if there be a decided alteration in the associative mechanism, form the starting point for a permanently developing chronic delusional system (paranoid states). Clinically such individuals may appear to have normal recording faculty, recognition, and memory, but despite thereof manifest fundamental disturbance in the formation of judgments. On superficial examination such patients may seem very intelligent, and one would not suspect them of being insane until one learned from prolonged conversation the falsification of their judgments regarding their own personality and their entire *lack of disease insight* as regards such falsification of the content of their ideas. Temporary and curable delusions may occur in acute psychoses as a symptom of inhibition of normal associations, but the chronic, incurable delusional insanities depend rather upon permanent loss of certain possibilities of association, the patient being unable critically to sift and arrange his judgments as a normal individual can do.

Many of the chronic systematized and progressive delusional states begin with a period of *psychic eclipse*. The patients complain that for some days or weeks their minds have been empty and that they have felt restless. Some go so far as to assert that they must have been anæsthetized or poisoned at the time. In the acute psychoses, especially those accompanying infections and intoxications, delusions are prone to arise as a result of the primary incoherence. The mixed-up ideas of the patient lead to peculiar and nonsensical judgment associations. In some cases delusions arise in which a primary associative disturbance cannot be demonstrated, the falsification of content of the ideas here depending upon pathological affective processes.

There can be no doubt that illusions and hallucinations are of great importance also for the origin of delusions. This is well illustrated in the so-called acute hallucinatory confusion or amentia; in curable cases the delusions disappear, but where a dementing process succeeds, the hallucinatory delusions may continue unaltered or may develop farther (Binswanger).

In chronic paranoid states the delusions may precede the hallucination and illusions, the latter being the outcome of the former. In the so-called acute hallucinatory paranoia the delusions arise primarily (in contrast with amentia, in which they are more secondary); in this disease the primary

suspiciousness of the patient may be followed by hallucinations which convert the suspicions into certainty.

Explanation delusions (secondary delusions in the strict sense) occur where pathological states of feeling are protracted (prolonged exaltation or prolonged depression) and form the basis of the false judgments.

The hyperdynamic ideas corresponding to an exalted state may easily lead to *delusions of grandeur* (dementia paralytica, dementia præcox). Similarly, the hyperdynamic ideas corresponding to the depressed mood of the melancholic and the hypochondriac may become fixed as *depressive delusions*, which persist even after the pathological mood has disappeared.

The *expansive* (*megalomanic*) *delusions* and *depressive* (*micromanic*) *delusions* together make up the *primordial deliria* of Griesinger. Depressive delusions include the delusions of sin, the hypochondriacal delusions, the poverty delusions, and the delusions of persecution.

*Delusions of persecution* differ from the other forms of micromanic delusions in that other people (enemies, persecutors) are held responsible therefor. Sometimes they arise primarily and are connected with abnormal sensations. The feeling of fatigue which sometimes follows the drinking of a glass of beer may excite the persecutory idea that the beer has been poisoned or that someone intended to drug the patient, perhaps in order to do some crime. Still more frequently persecutory ideas arise as a result of hallucinations or illusions. Complementary or contrasting persecutory delusions are often associated with delusions of grandeur, the patient assuming that he is being attacked on account of the greatness of his position.

*Ideas of reference* (*Beziehungswahn*) may be the forerunners of ideas of persecution. The patient refers looks, acts, remarks of other people to himself without sufficient reason. He thinks he is being influenced or watched or injured by someone else. He may become abnormally jealous (*jealousy delusion*) or he may get the idea that he is suspected of a crime and that he is to be brought into court (*imputation delusion*). The patient may attribute the persecution sometimes to definite persons about him, sometimes to invisible and unknown enemies. Sooner or later such a patient is likely to become convinced that a great conspiracy has been formed against him.

A word must be said about the so-called *general delusion of denial* (*délire de négation généralisé* of the French). It usually follows delusions of sin, the patient becoming so bad in his own estimation that he identifies himself with Satan. He can atone for this only by eternal punishment, and so comes to regard himself as immortal. Such a patient may think that his body is infinite in size as well as in duration (*délire d'enormité*). As this leaves no room for the rest of the world, such patients assert that the world no longer exists and that all men are shadows; even God ceases to exist.

When delusions first appear they are usually accompanied by affective states which correspond to the content of the delusion, but after long standing this feeling tone of the delusions may disappear.

The mode of onset of a delusion may be sudden or it may develop gradually. Primary delusions tend to have the latter form of development, and are more apt to become fixed; hence the unfavorable prognosis in such cases, as a rule.

Fixed delusions are prone to be followed later by a so-called *systematization*, the patient adding complementary delusions to the fixed delusion, the new delusion being connected logically with the original false judgment. Such systematized delusional states may last throughout the life of the patient.

To be distinguished from the delusions above described are the *defects in judgment* met with in imbeciles and patients, who for some reason or another have had some arrest of development. In such cases the memory pictures are defective and the associative relations among them are fewer than normal; as a result, incorrect judgments are very likely to be formed. As Ziehen puts it, poverty in ideas and in associative connections among them, together with feebleness of judgment, are the essential features of the intellectual defect, both in *acquired* and *congenital feeble-mindedness.*

All grades of such feeble judgment are met with, from the mildest lack of critical power to the most complete lack of judgment. The former is due to absence of a few complicated ideas and associations, while the latter depends upon absence of the simplest everyday ideas and associations.

Imperative ideas and delusions often disappear, but the feeble judgment once established nearly always remains.

**Anomalies of Affective and Emotional States.**—We have referred repeatedly in the preceding pages to the feeling tone accompanying sensations and ideas. Whereas sensations and their memory pictures (the so-called ideas or images) represent the elementary phenomena of consciousness which refer to objects external to a given perceiving subject or personality, the feelings, on the other hand, and the affective or emotional states in which they play the essential role represent the more intimately subjective side of experience in that this portion of the content of consciousness expresses the attitude of the individual to objects, etc., in its most fundamental form; that is, in particular, as it bears upon his conduct. In the affective states generally, then, we approach one of the most complex and difficult aspects of the psychologist's problem.

The elements of our experience of external objects about which no difference of opinion exists are sensations. In the same sense *the elements of the affective side of consciousness* include *agreeableness* and *disagreeableness* (pleasure and pain of the older psychologists, and *positive* and *negative feeling tones* used by physiologists generally); these two, at least, are elementary feelings. Whether Wundt's assumption that there are six directions in which elementary feelings may vary—the *agreeable* and *disagreeable*, the *exciting* and *depressing*, and the *straining* and *relaxing*—be correct or not need not here detain us.

*Emotions* are more complex than simple feelings, in that they contain not merely *affective*, but, quite as essentially, *sensational* (perceptional) elements also. The analysis of an emotion, for example, fear, reveals not only a feeling of disagreeableness, but also the sensational factors which represent the object which is feared, together with complexes of kinæsthetic and visceral sensations, the exact nature of which has not yet been discovered. The close relation between emotions and bodily movements is evident.

*Moods* are of the nature of emotions having the same general constitution, but, as a rule, they lack definiteness in that the sensational or objective side of a mood is not so frequently one particular object. Moods are, in general, of longer duration than emotions, and do not so readily express themselves in some definite act which tends to bring the state, as in emotion, to an end. On the contrary, they persist for longer periods as a kind of general affective tone of experience (*e. g.*, depression, exaltation). Manifestly, in both emotions and moods, memory and imagination are of, at least, quite as much importance as the sensations of the present moment,

and in the majority of cases of much greater significance, since they permit of the constant development of the objective content of both emotions and moods entirely freed from objective control.

Feelings and emotions become associated with objects other than those originally experienced with them, and this association occurs at times in most fantastic ways. This fact of the *transfer of feelings* helps us to understand the origin and permanence of moods in both those of normal and abnormal mentality. On this basis also the affective states are designated as *primary* or *secondary* according as they are connected with the original objects or with others to which they have been transferred.

It has long been held that *the feelings have an important relation to the state of the body;* as far back as the seventeenth century one meets the view (in Hobbes) that pleasurable feelings are associated with bodily well-being, while disagreeable feelings are both expressive of and conducive to the ill-being of the body. This relation has been subjected to much experimental investigation, and while the results of these researches have not been unequivocal in connection with the simpler and less intensive feelings—the heart and respiration in any case being but slightly affected—in stronger and more complex affective states the heart, bloodvessels, respiration, secretions, and general musculature of the body may all be involved.

The effects of strong feeling and emotion upon the intellect are often marked, depending, of course, upon the intensity as well as upon the quality of the affective state. If not too strong, the association of ideas is favored and accelerated, but when violent there may be inhibition or even complete arrest. The positive affective states tend to increase the rapidity of associations, while those negative in direction tend to retard associations.

Psychiatrists have been much influenced lately by Bleuler's monograph entitled *Affectivität.*

The most important anomalies of the affective life, following Ziehen's classification, include:

1. Pathological exaltation or hyperthymia.
2. Pathological sadness (depression) or dysthymia.
3. Pathological irritability.
4. Pathological apathy, general and circumscribed (hypothymia; athymia).
5. Pathological instability of feelings (moods).
6. Pathological general increase of affective excitability.

1. **Pathological Exaltation or Hyperthymia.**—This is best seen as a primary phenomenon in maniacal states. All the intellectual processes are accompanied by positive feeling tone. Even some of those which normally are associated with slight negative feeling tone appear in many to be accompanied by joyful feelings. It is not known whether the hyperthymia is really primary or is secondary to the psychic overexcitation. The experimental production of pleasurable feelings by the use of alcohol, opium, etc., supports the view that the affective state may be primary (Binswanger).

Hyperthymic states may appear in the course of many psychoses, both acute and chronic. The hyperthymia is usually an episodic phenomenon. A form of secondary exaltation has been described in acute and chronic paranoid states. Here hallucinations and delusions of joyful content determine the affective anomaly (Ziehen).

2. **Pathological Sadness (Depression) or Dysthymia.**—This pathological predominance of negative feeling tones may be primary or secondary. In

the *primary depression* the sadness may be out of all proportion to that which would normally accompany any sensations and ideas present. The depression may, indeed, be present without any apparent reason whatever.

In addition to his general sadness the patient may present symptoms of *anxiety*. Usually this anxiety is accompanied by abnormal somatic sensations (præcordial anxiety, epigastric pulsation, general unrest). Many have looked upon these somatic sensations as primary (originating peripherally), but it seems more likely that they are cortical in origin.

The motor phenomena accompanying anxious states are characteristic. The rubbing of the hands, the restless movements of the legs, the rapid respiration, the palpitation of the heart, the constriction of the peripheral vessels are some of the motor and vasomotor phenomena accompanying anxious states. Such primary depressions with anxiety are usually followed by symptoms of mental retardation (thought inhibition) and the patient answers questions and performs the simplest calculations only very slowly. The general musculature of the body may be abnormally lax or abnormally rigid, as in the catatonic states. Where the anxiety movements of the body are pronounced, one speaks of *anxious agitation*. Sometimes movement inhibition alternates with agitation of movements.

The patients are inclined to seek an explanation for their anxiety, and then arise the so-called explanation delusions. These may take the form of delusion of sin, of poverty, or of incurable disease. Primary depressions are met with as episodic states in various psychoses; in a mild form they are not infrequent in neurasthenia. The severest forms are met with in true melancholia. In the so-called *secondary depressions* the affective state seems to be directly dependent upon sensations and ideas with strong feeling tone.

3. **Pathological Irritability.**—The pathologically irritable person shows an abnormal tendency to anger and vexation. These affects appear too easily; exciting causes which in a normal person would not cause vexation call forth an affective reaction of abnormal intensity and long duration.

*Anger* is an affect which accompanies the tendency to attack persons or objects in the surroundings. Although it is a negative affect, it differs from sadness or depression in that the feeling of self is usually elevated. The association of ideas may at first be slowed, although as the anger grows there may be a sudden and explosive acceleration of the course of ideas. At the beginning there may be a tendency to motor inhibition, but after a time, as though through summation, actions are accelerated and are often explosively violent (raving mad).

During anger there is but little time for motives to come into play. The patient seems to fail to consider things at all; inhibitory ideas seem to have lost their power, or do not often come into his consciousness. His violence pays no regard to the welfare of his own person or of his surroundings. The energy of the movements may be greatly increased. The movements of anger are, however, so irregular as to be almost incoherent. This reveals itself in the speech of the angry man. He stammers and his sentences lack grammatical sequence or coherence (*anacoluthia*). It is not surprising, therefore, that after an attack of anger has passed individuals frequently show a partial amnesia of the motives leading to the passion, and even of their acts during the outbreak.

4. **Pathological Apathy, General and Circumscribed (Hypothymia; Athymia).**—In certain mental states feeling tones appear to be depressed or abolished. The condition is most pronounced in cases of melancholia. In many instances of neurasthenia the patients complain of loss of interest in things which normally should arouse them. Their feelings for their ideals have left them. They no longer enjoy the beauties of nature, and they complain that to those nearest and dearest to them they have become indifferent. Some patients do seem much exercised over this coldness of feeling; others show no depression on this account. An appearance of general apathy may be simulated when it does not exist. The non-participation of the patient in her usual interests may be due to a conscious suppression of emotional expressions, depending upon delusions or hallucinations, or it may be the result of a motor inhibition.

In contrast with this general apathy or depression of emotional excitability, as a whole, are the circumscribed defects in intellectual and reflected feeling tone met with especially in imbecility and dementia. In imbecility there is a faulty development of the emotional nature which may keep pace with the defect in intellectual development or may exceed it, as in moral insanity. In imbecility of milder grade, anger, envy, hate, love, gratitude, hope, and fear may be well developed, but the individual does not attain to any intellectual interests. In spite of careful education it is impossible to arouse interest in art or science. In still milder cases the ethical or altruistic feelings alone may be deficient, the egoistic feelings being abnormally strong.

In acquired dementia, such as one sees in dementia paralytica, the loss of the higher feelings usually goes parallel with the loss of intellectual power. The changes in this disease show us very clearly the relation of conduct and behavior to our emotional life (altruistic, social, religious feelings). In alcoholic dementia, epileptic dementia, and in senility one meets with symptoms similar to those presented by the paralytic dement. In the later stages of these acquired dementias the whole emotional life may be undermined and a general apathy or bluntness of feeling result.

5. **Pathological Instability of Feelings (Moods).**—In normal individuals, there is a certain persistence or inertia to emotional states. In mental disease the moods are often much more variable. Two forms of pathological lability of moods may be distinguished, a primary and a secondary. The *primary* form occurs most often in imbecility and dementia, where it is associated with intellectual defects. The loosening of the associative connection characteristic of the intellectual defect also accounts for the instability of the irradiated feeling tones. A new sensation or idea will then sometimes in a moment lead to new irradiations of feeling tones and cause a *bouleversement* of mood. This is best illustrated perhaps in dementia paralytica, where a patient in tears or anger is by a suitably chosen word converted into a state of joyful exaltation.

In the *secondary* form of instability of moods the changeability of feeling is not a primary affective disturbance, but is secondary to a pathological inconstancy and incoherence of the contents of the ideas or sensations of the patient. Where the ideas are constantly changing the moods also change. This is well illustrated in hysteria and in some forms of paranoid states. A pathological capriciousness is very characteristic of many hysterical patients.

6. **Pathological General Increase of Affective Excitability.**—In contrast with general apathy, a general increase of affective excitability seems sometimes to be met with. The individual is abnormally susceptible for all emotional impressions. This is the case in some instances of neurasthenia, but it is also occasionally met with at the beginning of severe organic psychoses. When the increase in excitability is limited to the so-called higher feelings—ethical, æsthetic, and religious—it is designated as *pathological transport* or *ecstasy* (*krankhafte Ergriffenheit*). Such patients become over-enthusiastic for political, religious, or humane movements. When delusions develop upon the basis of this pathological ecstasy an *eknoic state* is said to exist.

The so-called *imperative affects* are those pathological feelings which arise without cause, and which the patients themselves designate as strange or compulsory and independent of imperative ideas, delusions, or hallucinations. A patient in good humor may suddenly and without reason have a strong sympathy or a strong antipathy for some person near him, combined with a feeling that some force has artificially brought about such a tendency in him.

**Anomalies of Conduct (So-called Will or Volition).**—Psychologists no longer recognize will as a special "faculty" of the mind. Conduct is, for them, the direct result of the total content of consciousness (sensations, percepts, memories, imaginations, feelings, emotions) of the moment. In states in which there is a general increase of motor action in mental disease we speak of *motor agitation;* where motor action is diminished or abolished, as a whole, we speak of *aboulia* or *motor stupor.*

In observing anomalies of volition one should notice not only the ordinary voluntary acts, but also alterations in speech, in expression, and in gesticulation. The so-called voluntary movements depend directly upon an associative process leading to some goal idea, while the expressive movements (mimic and pantomimic) appear to be determined chiefly by the intensity and quality of the feelings. Disturbances of sensation, of memorial reproduction, affective disturbances, as well as of the association of ideas proper, can all influence conduct. Of the sensory disturbances which influence volition, hallucinations and illusions are most important. A sudden hallucination may lead to a wholly unexpected act of violence.

Wernicke has classified movements (outside of the reflex movements) into expressive, reactive, and initiative movements.

By *expressive movements* are meant all those by which the effects and emotional states of a person are manifested. The *reactive movements* are those which follow direct external stimulation (answers to questions by speech or other movement; behavior on physical examination). The *initiative movements* are those which arise apparently spontaneously and not as a result of an immediate external stimulus, the whole conduct, behavior, action of a person in a certain situation. All movements not reflex, expressive, or reactive belong to the initiative group. Wernicke divides all psychomotor disturbances into (1) those in which there is lowered excitability or conductivity of nerve paths (*akinesis*); (2) those in which there is increased excitability or power for conduction (*hyperkinesis*); and (3) those in which there is a perverse excitability (*parakinesis*).

It will be seen that the above classification of Wernicke is somewhat more elaborate than that of Meynert, who subdivided movements into (1) those of defence (*Abwehrbewegungen*) and (2) those of offence (*Angriffsbewegungen*).

Action may be pathological on account of the absence of ideas which are present in normal states; this absence of ideas may be due to defective development (imbecility) or to loss of memory images, following upon disease (acquired dementias). Pathological actions of this sort are designated *defective acts*. They often resemble the normal acts of lower animals. They may be sly and skilful, but are pathological in human beings on account of the fact that they are uninfluenced by higher and more abstract ideas.

In the motor agitation of *mania* the pleasurable feelings lead to remarkable mimic and pantomimic motor discharges and to a logorrhœa characterized by rhymes and alliterations.

In the milder forms of maniacal exaltation in which the associative connection of the ideas is still fairly well retained, initiative movements occur more rapidly and in greater numbers than normally, and lead to conduct which, although apparently consistent, is absurd in its motives and aims. In the severer outbreaks, stormy, contradictory, impulsive acts result from the hodge-podge of goal ideas which swim through the consciousness.

The inhibiting and slowing of motor action which accompanies negative feeling tones is characteristic of *melancholic states*. In simple depression pantomimic movements (gesticulation) are reduced to a minimum. The voluntary muscles are generally relaxed, the arms hanging loose by the sides or the hands folded on the lap. The head sinks upon the breast, the eyes are lowered, and there is a tendency to convergence. The mandible falls and the angles of the mouth are drawn downward. The lid slits are narrowed and the eyebrows lowered except at their medial extremities. Usually the patients do not weep; indeed, they sometimes complain of an inability to weep. Often the secretion of the lacrimal glands is actually diminished, accounting for the peculiar lack of lustre of the eyes. Depressed patients have little to say, and when they speak it is in low tones.

The influence of depression upon motor activity becomes changed as soon as a state of *anxiety* is added to the depression. At first anxiety increases the inhibition of cortical associations and tends also to slow action, but as the anxiety increases there arises the idea of flight, and the patient seeks in every way deliverance from his anxiety. He cannot rest and is impelled to walk about day and night, lamenting his state (*motor agitation of anxiety*). It is here that the patient so often seeks relief in *suicide*. Perfectly quiet for a time as a result of motor inhibition, the anxious patient may suddenly make a suicidal attempt, or he may become violent and try to injure his surroundings (*incendiarism, homicide*). Other patients try to still their anxiety by alcoholic excess or onanism. These attacks due to anxiety occur in paroxysms. Between the paroxysms the motor inhibition of depression is resumed.

The behavior of apathetic patients varies according to the extent of the apathy (general or partial). In *general apathy* motor activity is reduced to a minimum. Since, normally, acts occur only when movement ideas with positive feeling tones arise in consciousness, acts cease in general apathy because most of the movement ideas which arise in consciousness are devoid of feeling tone (*apathetic motor stupor*). In *partial apathy*, where certain only of the feeling tones are lacking, it is the conduct which depends upon the presence of more complex and especially the ethical ideas which is most prone to be defective. The patient acts wholly according to his lower egoistic interests, not distinguishing between right and wrong.

In general apathy all the muscles of the body are lax; even the cheeks hang down. The head and limbs assume positions which depend upon gravity. The upper lids fall so as to simulate ptosis. The patients incline to lie down much of the time; in severe forms the apathy may resemble sleep. The condition of the pupils distinguishes the pseudo-sleep of apathy from true sleep; in the latter, when the eyelid is opened there is at first dilatation and subsequent contraction, while in apathy the pupils are usually midway between dilatation and contraction and there is a distinct contraction at the moment light enters the eye on opening the lid.

**Acceleration of Motor Actions.**—When motor discharges occur more rapidly then normal we speak of *motor agitation*, or sometimes of pathological *pressure of activity*. One meets with every degree of it from abnormal talkativeness with overactive facial expression to the enormous acceleration of speech known as logorrhœa. The mimic and pantomimic movements may be so much exaggerated that we have excessive grimacing and violent gesticulation. The patients are restless, they assert that they cannot sit still, and they spend their time pacing up and down the room or wandering about. Such patients may be pathologically busy, beginning a dozen and one tasks hastily and enthusiastically but quickly turning from each.

In the severest forms of motor agitation when the patient becomes "raving mad," his cries may become inarticulate and his movements violent and purposeless. Such a patient may tear up his clothing or his bedding, break the furniture of his room, and attack people who come near him.

Psychiatrists distinguish a primary from a secondary pressure of activity, the former corresponding to primary flight of ideas and occurring with it, the latter depending upon hallucinations or violent affects (*hallucinatory agitation* and *affective agitation*). In trying to determine whether one is dealing with a primary or secondary agitation one ascertains the presence or absence in the first place of hallucinations and delusions, or of abnormal affects, and tries to decide whether the motor agitation can be explained by these if they exist. In case they are insufficient to explain the agitation, it is probably primary.

**Retardation of Motor Actions.**—Slowing of motor acts accompanies depressed states, which are accompanied by thought inhibition. When there is motor inhibition, together with thought inhibition and aprosexia, the condition of stupor is said to exist. One meets with mild and severe grades of *motor stupor*. In the milder forms movements seem difficult and are carried out more slowly than normal. The patient understands questions or commands less promptly than when he is in health. He pronounces the words of the answer more slowly, and any movements he makes are more deliberate than they should be. Mimic and pantomimic movements may be suppressed entirely. In his every-day life the patient manifests feeble will (aboulia), and is the victim of indecision.

When the motor stupor reaches a high grade it may assume any one of three different forms:

1. There may be complete relaxation (*resolution*) of the body musculature, the patient lying for weeks or months without stirring a limb, with complete absence of resistance to passive movements, the eyes kept closed.

2. There may be a general rigidity or tension of the muscles of the body (*catatonic rigidity* or *attonnity*), the body being held stiffly in a general position of flexion (flexion type) or of extension (extension type), or in various bizarre

attitudes, the eyes held tight shut, the teeth clinched. Passive movements are strongly resisted, and, as a rule, attempts at passive movement increase the general rigidity. The patients are mute and in general *negativistic*. Sometimes, however, there is a peculiar susceptibility to motor suggestion known as *command automatism*. On passive movement the limbs move like wax, retaining the position in which they are put often for three or four hours (*flexibilitas cerea*); sometimes they repeat movements made before them (*echopraxia*), or repeat words pronounced in their hearing (*echolalia*).

3. There may be a limitation of initiative movements to a few acts repeated continuously for hours, days, or months at a time (*stereotyped movements*), the patient rocking to and fro or from side to side, or continuously rotating the head or the trunk.

One tries to make out in each case whether the motor inhibition is primary (*true stupor*) or secondary (*pseudo-stupor*). *Primary motor stupor* corresponds to primary depression of thought activity, being part of the general slowing of associative processes (*e. g.*, in melancholia). Usually it takes the type of relaxation of the muscles or of simple catatonic rigidity. In *secondary motor inhibition* the pseudo-stupor may depend upon definite hallucinations, delusions, or affects.[1]

**Incoherence of Motor Actions.**—When there is incoherence of the association of ideas the motor acts also become incoherent (*motor incoherence*); the patient is in a state of confusion. In its mild form it betrays itself in a peculiar lack of plan in the more complex acts of the patient. On going for a walk he may wander hither and thither without aim and out of accord with the purpose which he at first had in mind. Such a patient will busy himself first with one thing and then with another in a most unsystematic way.

In the worst forms of confusion the acts become nonsensical and stand in no correct relation to the ideas preceding them (*parapraxia* and *apraxia*).[2] The patient does not know how to use simple objects. He cannot light a match, cannot open a knife, may try to eat milk with a fork, and may bite his own finger instead of a morsel of food held in his hand. The confusion may extend to speech and writing, the patient designating objects falsely (*paraphasia, paragraphia*). The movements of the body may become incoördinated, resembling ataxia or chorea. Even the mimic movements may no longer correspond to the underlying affect. The patient's grimaces are out of accord with his feelings; his laughing and crying stand in no ascertainable relation to the content and feeling tone of his ideas (*paramimia*).

Motor incoherence is sometimes combined with motor agitation, the mixed state being designated *incoherent agitation* and the excessive incoherent movements described as *jactitations* (chorea magna of the older writers). When this is accompanied by fever the condition has been called *delirium acutum*.

The motor incoherence is called primary when it cannot be attributed to other psychopathic states (*e. g.*, in the incoherent form of paranoia). It is called secondary when it is due to hallucinations, delusions, flight of ideas, affective disturbances, or imbecility.

**Anomalies in the Intensity and Duration of Acts (Change of Acts).**—In health initiative movements undergo changes corresponding to the changes which

[1] For the differential diagnosis of the different forms of stupor the reader is referred to Ziehen's *Psychiatrie*, second edition, Leipzig, 1902, p. 156.

[2] Wilson (S. A. K.), *Studies in Apraxia*, *Brain*, 1908.

take place in the sensations and ideas in consciousness. When special motives are present certain movements may become dominant. In mental disease, however, dominant movements or acts may appear without adequate motivation (*hyperdynamic acts*). These hyperdynamic acts may be *primary* (not due to other psychopathic symptoms). A good example is seen in the so-called *tics*. Most hyperdynamic acts are, however, *secondary* to delusions, hallucinations, or anxious states. The monotonous recurrence of a certain act or attitude (*motor stereotypy*) is probably due to a more or less permanent psychomotor hyperdynamy.

Among the interesting stereotyped movements frequently met with are the snout-like movements of the lips (*snout cramp*) and constant blepharospasm. Sometimes the stereotypy manifests itself in certain bizarre movements, the so-called *mannerisms*. Many patients have a stereotyped scanning speech or vary the pitch of their voice in monotonous repetition. Sometimes single syllables or sounds are intercalated in the speech, a habit usually regarded as affectation, but spoken of by psychiatrists as *stereotyped embolophrasia*.

When a motor innervation has been completed and there is a tendency on the part of a patient to repeat it afterward, even when other movements are required of him, the condition is designated *motor perseveration*. A common example is the repeated showing of the tongue, after one has asked to see it, even when the patient is subsequently requested to show his teeth, to beckon with his finger, or to close his eyes. This motor perseveration, being due to a request or stimulus from the outside, is to be distinguished from the stereotypy which has its origin within.

Temporary motor hyperdynamy reveals itself in the so-called *impulsive acts* due to a sudden affect, delusion, imperative idea, or hallucination.

**Anomalies of Conduct Due to Delusions and Imperative Ideas or to Imbecility and Dementia.**—A patient who has delusions or imperative ideas will often reveal the character of the delusions or the ideas in his conduct. The whole facies and the attitude of the patient with delusions of grandeur are characteristic and are in marked contrast with the appearance and behavior of the patient suffering from delusions of self-depreciation. When delusions of persecution exist, the patient is ever on the defence (bolting of doors, sudden flights to escape enemies). Sometimes the *persecuté* becomes the *persecuteur;* then, instead of defensive movements, he may assume the attitude of attack. Such patients are usually dangerous in the community (paranoia).

Impulsive acts are sometimes due to the "pathological notions" described in an earlier paragraph.

The so-called *imperative acts* are the result of the imperative ideas also previously described. Here the patient recognizes the absurdity of his act, but performs it all the same. One should distinguish carefully the imperative idea which is accompanied by the impulse to motor action and that not associated with such motor impulse. In the former case the imperative act frequently results, in the latter it does not. In mysophobia the continuous washing of the hands for hours at a time is an illustration of an imperative act. A child's imitation of its parents may be regarded at a certain stage of development as a normal echokinesis, but later on direct imitation of movements observed ceases to be desirable, and the tendency is normally suppressed. The abnormal patient may, however, imitate movements which he sees or sounds which he hears, as in the echolalia above mentioned.

The influence of defective judgment upon the conduct becomes obvious

in the study of imbecility and the various forms of dementia. The acts of defectives reveal the absence of those complex ideas which normally determine the play of our motives. There is an absence of due consideration before action. The so-called criminal acts doubtless belong here.

**Disturbances of Motility Proper.**—In the preceding sections motor disturbances of psychic origin have been referred to. We have now to consider the disturbances of motility less directly psychic or entirely infrapsychic in their origin, disturbances due to alterations in function of the motor pathways from the cerebral cortex to the muscles, or to alterations in the cortex itself or in the muscles themselves.

**Atrophy and Hypertrophy of Muscles.**—The *state of nutrition of the muscles* yields important clues to the nature of many nervous diseases. *Muscular atrophy* may or may not be associated with paralysis of muscles. *Simple atrophy* of the muscles should be distinguished from so-called *degenerative atrophy*. In the former there is only a quantitative diminution in size; in the latter there is in addition an actual degeneration of the muscle substance. Degenerative atrophy occurs in lesions of the lower motor neurones (anterior horn cells, motor nuclei, or cerebral nerves, peripheral motor nerves). Simple atrophy may be due to disease of the muscles themselves or to lesions of the motor conduction paths situated above the lower motor neurones (*e. g.*, pyramidal tract lesions). It may be due simply to disuse (*atrophy of inactivity*). In true degenerative atrophy the so-called reaction of degeneration is found on electrical examination. Where the degenerative atrophy is due to lesion of the anterior horns or of the motor nuclei of the cerebral nerves, *fibrillary twitching* is commonly present. It is rare to find it in the lesions of the peripheral motor nerves.

The muscles may be the seat of a true *hypertrophy* in athletes. There is localized hypertrophy of muscles which are subject to cramp-like processes which continue a long time. A condition resembling hypertrophy of the muscles, but which in reality is due to increase of connective tissue and fat, the muscle substance itself being in reality diminished in amount, is seen in certain types of muscular dystrophy (so-called *pseudo-hypertrophy*).

**Muscular Tone, Atony, Hypotony, Hypertony, Contractures.**—The condition of *tone* (*tonus*) presented by the voluntary muscles is of importance for diagnosis. When the resistance to passive movement is abnormally small, we speak of *hypotony* or *atony* or *resolution* of the voluntary muscles.

When there is an abnormally strong resistance to passive movement, we speak of rigidity or *hypertony* of the muscles. It is conspicuously present in the various spastic paralyses, in many motor spasms, in catatonia, etc. When along with hypertony a muscle remains permanently contracted, we speak of *spastic contracture*. When certain muscles are paralyzed and on subsequent voluntary innervation only the antagonists contract, there comes a time when relaxation of these antagonists no longer results in a normal position of the limbs. Such a permanent shortening of the antagonists is sometimes spoken of as *paralytic contracture*. In contrast with these *neuropathic contractures* (spastic and paralytic) there occur also *myopathic contractures* due to inflammation of the muscles and tendons, *contractures from shortening of fascia* as a result of scars, *contractures of reflex origin*, and *hysterical contractures*.

**Paralyses.**—Inability to bring the muscles to contraction is known as paralysis. Paralyses may be *classified* in different ways: (*a*) According to

the state of nutrition of the muscles; (*b*) according to the state of tonus of the paralyzed muscles; (*c*) according to the distribution of the paralysis.

As regards the classification according to the *state of nutrition of the muscles*, paralyses may be divided into (1) atrophic, and (2) non-atrophic paralyses. By *atrophic paralysis* is meant a special form not due simply to disuse and accompanied by rapid reaction of degeneration. It indicates a lesion of the lower motor neurones, either in their nuclei of origin or in the peripheral nerves. By *non-atrophic paralysis* is meant the form in which the paralyzed muscles undergo but little shrinking in volume; on electrical examination no reaction of degeneration can be made out.

According to the *state of tonus* of the paralyzed muscles, paralysis may be divided into (1) flaccid paralyses, and (2) spastic paralyses. Flaccid paralyses are usually associated with loss of the deep reflexes, while spastic paralyses are usually associated with exaggeration of the deep reflexes. The flaccid paralyses are due, as a rule (although not always), to lesions of the lower motor neurones. The spastic paralyses are due nearly always to lesions of the upper motor neurones. As regards the *distribution of the muscles paralyzed*, paralyses are divisible into: (1) Neural paralyses; (2) plexus paralyses; (3) radicular paralyses; (4) paralyses of one extremity or of the face (monoplegias); (5) paralyses of half the body (hemiplegias); (6) paralysis of both lower or both upper extremities (paraplegias).

In the *neural paralyses* a single muscle or the muscle innervated by a single nerve may be involved (*e. g.*, Bell's palsy). In the *plexus paralyses* the whole or a part of the muscles supplied by the brachial or lumbosacral plexuses may be involved. The paralyzed muscles in *radicular paralyses* correspond to the motor innervation from single nerve roots. The *monoplegias* may be due to lesions of peripheral nerves or of the central nervous system. The face may be alone affected (*monoplegia facialis*); if it be the arm alone we speak of *monoplegia brachialis;* if the leg alone, of *monoplegia cruralis.* When the musculature of one-half of the body is paralyzed the condition is known as *hemiplegia.* If the muscles are only weakened, the term *hemiparesis* is employed. A hemiplegia may be organic or functional; it may be flaccid at first, but is usually spastic later; it may or may not be accompanied by sensory disturbances (hemianæsthesia; hemianopsia). It may be cortical, capsular, peduncular, pontile, or medullary in origin. A hemiplegia may be associated with paralysis of the muscles supplied by the opposite oculomotor nerves (*hemiplegia alternans superior*, or *Weber-Gubler type*); or the face may be paralyzed on one side and the arm and leg on the other (*hemiplegia alternans inferior*, or *Millard-Gubler type*).

The term *paraplegia* is usually restricted to paralysis of both lower limbs, with more or less involvement of the trunk muscles. The paraplegia may or may not be associated with sensory changes; it may be organic or functional, flaccid or spastic. The flaccid paraplegias may be due to disease of the spinal cord or the peripheral nerves, while the spastic paraplegia is due to lesions of the upper motor neurones, either in the spinal cord or at a higher level. The Brown-Séquard syndrome is sometimes spoken of as a *hemiparaplegia.* When all four extremities are paralyzed, owing to a lesion in the cervical cord, the paralysis is spoken of as a *cervical paraplegia.*

**Motor Irritation.**—The various forms met with include: (1) Tremor. (2) Fibrillary twitchings. (3) Athetoid and choreiform movements. (4) Clonic and tonic spasms.

By *tremor* is meant rhythmical, involuntary, frequent oscillatory movements of slight extent. The tremor may be fine or coarse; thus, that of Graves' disease is very fine and the rate varies from 8 to 10 oscillations per second; in paralysis agitans, on the other hand, the tremor is coarser and the oscillations less frequent (2 to 4 per second). A tremor which appears only when voluntary movements are undertaken is known as an *intention tremor*. It is met with especially in multiple sclerosis. Closely related to it is *nystagmus*.

By *fibrillary twitchings* are meant the contractions of single fiber bundles of any given muscle, the contractions being insufficient to lead to any locomotor result. Fibrillary twitching is a totally different thing from tremor. It is met with especially in disease processes in which the muscle is degenerating, owing to lesion of the cell bodies of the lower motor neurones.

By *athetoid movements* are meant certain slow, involuntary movements of extension, flexion, adduction, and abduction, each individual part moving by itself independent of the others, so that the various parts may at any given moment occupy very different relative positions in space. The movements may be unilateral (*hemiathetosis*) or bilateral.

*Choreiform movements* include the involuntary, quickly changing movements, made without plan or purpose, met with in various diseased conditions. They are not confined to the hands and feet, but may involve any of the voluntary muscles of the body. This fact, together with the greater quickness of the movements, distinguishes them from athetoid movements. The movements are best studied in *chorea minor* (St. Vitus' dance; Sydenham's chorea). A unilateral chorea is not infrequent after hemiplegia (*post-hemiplegic hemichorea*). In hysteria a very coarse form of chorea known as *chorea major* is sometimes met with. It is frequently associated with *clownism;* in the same disease a form of chorea occurs in which there are quick, lightning-like contractions in single muscles similar to those which follow upon electrical stimulation (*chorea electrica*).

Involuntary movements of certain muscles which accompany voluntary movements of other muscles are known as *associated movements* (*muscle synergies*). One sees such movements frequently in the paralyzed limb of hemiplegics on strong voluntary innervation of the healthy limb. The *tibial phenomenon of Strümpell* may be regarded as an instance of a spinal associated movement due to injury of the pyramidal tracts. When it is present flexion of the lower extremity at the hip-joint and knee-joint calls forth a dorsal flexion of the foot and elevation of the medial margin of the foot, despite the patient's effort to suppress these movements.

*Spasms and convulsive movements* are among the commonest instances of motor irritation. By *clonic spasms* are meant involuntary quick jerkings of the muscle which follow one another rapidly without interruption. If the distribution includes a large number of muscles at any one time, we speak of *clonic convulsions*. In *tonic spasm* the individual muscular contractions last a long time. When a single muscle is involved with severe pain the condition is known as *cramp*. When muscle groups are affected together, or the whole body is involved, we speak of *tetanus* and *tetanic contractions*.

*Convulsive seizures* or *convulsions* occur in epilepsy, hysteria, tetanus, and eclampsia. In the ordinary *epileptic attack* the convulsion consists at first of a tonic spasm of the whole body musculature, followed after a short time by clonic spasms. In cortical irritation a peculiar type of epileptiform convul-

sion known as the *Jacksonian attack* occurs. The muscular contractions begin in single muscle groups and radiate into other motor domains (so-called "march" of the convulsion). In hysteria the convulsive movements may resemble those of epilepsy, although they are usually much more violent and the movements are more varied, even leading to remarkable attitudes (arching of the back, etc.).

In *tetanus* there is tonic spasm of the muscles affected. This often leads to lockjaw (*trismus*), to spasm of the face muscles, giving rise to a laughing expression (*risus sardonicus*), to arching of the spine with retraction of the neck (*opisthotonos*), and sometimes to tonic spasm of the abdominal and respiratory muscles.

In *tetany* we meet with intermittent tonic spasms of bilaterally symmetrical groups of muscles, associated with painful sensations and paræsthesias. The attitude of the hands and forearms is especially characteristic (*"obstetrical hand"*), but muscles in various parts of the body may be attacked. Even when the spasm has passed off in the arms it may be reproduced by applying pressure to the arm above the elbow with a blood pressure apparatus (*Trousseau's phenomenon*). The mechanical excitability of the muscles of the face is greatly increased. Mere stroking of the cheek or tapping upon the branches of the facial nerves (pes anserina) with a percussion hammer calls forth quick muscular contractions in the face (*Chvostek's phenomenon*). The sensory nerves may also be hypersensitive, and tapping at Valleix's points then calls forth abnormally intense sensations (*Hoffmann's symptom*). On electrical stimulation the motor nerves are found to be hyperexcitable in tetany (*Erb's phenomenon*).

In Thomsen's disease (*myotonia congenita*) there exists a so-called *intention rigidity* of the muscles. On voluntary contraction of any group of muscles there results an abnormally strong contraction of long duration which leads to motor inhibition. Gradually the muscles relax again.

In psychasthenia and in certain other psychoneurotic states sudden contractions of the muscles known as tics are met with. In the so-called *maladie des tics* of Gilles de la Tourette the musculature of the whole body may be involved, and the condition is associated with mental deterioration.

**Disturbances of Coördination (Ataxia).**—The mechanism by which muscles act together for purposeful effects is known as *coördination*. Almost every voluntary movement requires the simultaneous and successive activity of several muscles (*synergists*); some of the muscles are contracting (*agonists*); others are relaxing (*antagonists*). The grouping of contractions, their succession, and the force of each have to be carefully regulated in order that the movements shall be harmonious and purposeful. Disturbance of this mechanism leads to the anomaly of movement known as *ataxia*.

The presence of ataxia is not necessarily combined with weakness of the muscles. It is, however, frequently associated with loss of deep sensibility and with hypotony of the muscles. It seems certain that ataxias are due more to interference with the centripetal or sensory paths than to interference with motor conduction paths.

In the so-called *cerebellar ataxia* there is a disturbance of equilibrium, manifested especially on standing and walking. The patient stands with his feet wide apart and sways on walking from side to side like a drunken man. His tendency on walking is to let his legs run ahead of his body (*asynergie*

*cerebelleuse* of Babinski). In trying to rise from the recumbent position, the patient, instead of lifting his trunk, is likely to lift his legs in the air.

In cerebellar disease, the patient lying on his back with the lower extremities in the air, the thighs flexed and the legs abducted, can maintain sometimes a *fixation of position* beyond that possible for a normal man; the lower extremities behave as though cataleptic (Babinski). On the other hand, the capacity (*diadokokinesis*) to carry on quickly a series of antagonistic movements, such as rapidly alternating pronation and supination, may be lessened (so-called *adiadokokinesis*).

**Electrical Condition of the Muscles and Nerves.**—Much information of value concerning the state of the muscles and motor nerves can be arrived at by careful electrical examination. One determines the *excitability and conductibility of the motor nerves* and the *direct excitability of the muscles.*

*Increased excitability* to electrical stimulation is not very common. It is occasionally met with in a beginning neuritis, in tetany, in beginning dementia paralytica, etc. A *lessened excitability* is met with in all old paralyses due to lesions of the upper motor neurones and also in the myopathic form of muscular atrophy.

The most important electrical reaction for diagnostic purposes is the so-called *reaction of degeneration* (*De R*). When it is complete, changes in the electrical excitability, both in the nerves and muscles, are demonstrable. The excitability of the nerves for both faradic and galvanic current grows less and less until finally it disappears. The excitability of the muscle decreases for the faradic current *pari passu* with that of the nerves until it also is finally abolished, but the excitability to the galvanic current undergoes a remarkable change. Instead of a quick, lightning-like contraction the reaction to the galvanic current becomes slow, worm-like, and long drawn out, and the current, when applied directly to the muscle, shows an increased excitability of the latter, very feeble currents calling forth contractions. In addition there is a *reversal of the normal law of contraction;* normally, the cathodal closure contraction can be produced with the feebler current, whereas in reaction of degeneration anodal closure contraction is more easily produced than cathodal closure contraction, and the cathodal opening contraction approaches in case of producibility that of the anodal opening contraction. In curable cases the movements return before the electrical excitability becomes normal.

When the reaction of degeneration is present it is proof positive of a lesion of the lower motor neurones, although it may affect either the cell bodies of the neurones, the peripheral motor nerves, or the nerve endings in the muscles; thus, a reaction of degeneration is common in anterior poliomyelitis, in progressive muscular atrophy, in syringomyelia involving the anterior horns, in the various forms of neuritis, and in diseases affecting the bulbar motor nuclei. There is no reaction of degeneration in lesions of the brain and cord which do not involve the lower motor neurones, nor is it present in the so-called pure primary muscular atrophies (dystrophies).

In myotonia congenita the so-called *myotonic reaction* of Erb is obtained, which is peculiar in this, that the muscular contraction lasts for a long time on direct faradic and galvanic stimulation after the current is again opened, and, besides, peculiar wave-like contractions can be produced if one stimulates the muscle near its insertion, placing the other electrode near the origin of the muscle.

In myasthenia gravis the so-called *myasthenic reaction* of Jolly can usually be obtained. It is a sign of exhaustion quite in accord with the other phenomena of the disease. A tetanizing faradic current when first applied shows the presence of normal excitability, but gradually the response diminishes and in a short time no response can be elicited.

**Anomalies of Gait.**—The limits of normal variation in gait are wide and should be borne in mind in studying pathological cases. There are several tolerably characteristic gaits to be distinguished. Among the more important are the wobbly gait, the paretic gait, the spastic-paretic gait, the spinal ataxic gait, the cerebellar ataxic gait, the gait of tremor, the hemiplegic gait, and the gait of intermittent claudication.

The *wobbly gait* is often due to paralysis or atrophy of the M. gluteus medius et minimus. It is also seen in congenital dislocation of the hip-joint.

The *paretic gait* assumes two types, the simple paretic gait and the partial paretic gait. In the *simple paretic gait*, due to muscular weakness only, the movements of walking are slowed and the steps are shortened. Frequently there is an exaggerated flexion at the knee-joints. In severer cases the patient has to walk with crutches. In the *partial paretic gait* certain only of the muscles are weak. The most common form is that due to paralysis of the peroneal muscles on both sides. There is toe-drop, leading to lengthening of the leg; in order to compensate for this the lower extremity must be overflexed at the hip and knee. This gait resembles to a certain extent the movements of a horse; it was described by Charcot under the name of *steppage*.

In the *spastic-paretic* gait the weak muscles are hypertonic and the stiffness slows the movements and diminishes the excursions. The lower extremity moves more or less, as a whole. The toes cling to the ground; the difficulty in flexing the knee and hip is partly overcome by elevation of the pelvis on the side of the swinging leg. Often there is adductor spasm in the thighs, so that the knees rub against one another, and there is a tendency of the legs to cross on walking. In hemiplegia with spasticity the rigid limb swings lateralward, making a movement of circumduction, distinguishing it from the gait of hysterical hemiplegia, where the paralyzed leg is "dragged" forward. In cerebral softening and especially in pseudo-bulbar paralysis the steps are often very short, the foot being lifted from the ground only with difficulty. This gait is spoken of as *demarche à petits pas*.

The *spinal ataxic gait* is very characteristic and is easily recognized. The excursions of the movements are all exaggerated, the hip is overflexed and rotated lateralward, the toes are lifted and the whole leg suddenly thrown forward, the foot being brought to the ground with a stamping sound. The feet are kept wide apart and the patient watches his movements closely, being almost sure to fall if he looks away.

Two kinds of *cerebellar ataxic gait* are described, one due to disturbance of equilibrium and often associated with vertigo, the other depending upon a movement ataxia. In the former, the gait resembles that of a drunken man, the patient swaying from side to side in a very irregular manner. In the latter, the patient walks with his feet far apart, stamping on the ground, but without the wide excursions of the spinal ataxic gait.

The character of the *gait in tremor* depends upon the cause of the tremor. Gaits more or less characteristic are met with in multiple sclerosis, in hysteria, and in paralysis agitans. In the latter, in addition to the stooped attitude

of the patient, we meet with the phenomena of *propulsion* and *retropulsion;* the patient walking forward or backward has difficulty in stopping himself. In hysteria inability to stand and walk is sometimes a prominent symptom (*astasia-abasia*).

In *intermittent claudication* the patient after walking a short time has pain, fatigue, and numbness in the leg; these symptoms increase rapidly in severity until further movement becomes impossible. On resting for a short time the patient can again walk for a little while, but only to meet with a recurrence of the symptoms (arteriosclerosis of the arteries of the lower extremities).

**Anomalies of Speech and Writing.**[1]—Disturbance in the articulation of the speech sounds is known as *dysarthria*. In its highest grade speech sounds can no longer be emitted (*anarthria*). Dysarthria may depend upon weakness of the muscles of the lips, of the tongue, of the velum palatinum, or of the larynx. The speech has a *nasal twang* in cleft palate and in paralysis of the velum palatinum. It is often heard in patients with adenoids. In bulbar paralysis it is the *lingual* letters (s, l, d, t, n) which are difficult at first; later the *labials* (p, b, m, f, w, o, u) are affected, and finally the *gutterals* (g, k, ch, r) become indistinct.

When the speech is especially slowed (convalescence from acute disease, psycho-motor retardation) the condition is known as *bradylalia*. When the individual syllables are separated by abnormally long pauses, the condition known as *scanning speech* is said to exist. It is especially characteristic of multiple sclerosis, and can be demonstrated by asking the patient to pronounce such words as Constantinople, circumstantiality, etc.

In *stuttering* there are spasmodic contractions of some of the speech muscles, interfering with production of the speech sounds. It is to be distinguished from *stammering* (*dysarthria literalis*) by the fact that in the latter spastic muscle contractions are absent. In stuttering the trouble with speech often disappears when the words are sung. Under observation stuttering is exaggerated, while stammering is usually somewhat lessened. There is a form of *syllable stumbling* which is met with especially in dementia paralytica, and is of great importance for diagnosis.

In studying *aphasic disturbances* it must be kept in mind that speech consists of two great groups of functions, the *perceptive* (understanding of speech) and the *expressive* (act of speech). The anomalies on the perceptive side are included under the general term of *amnesic* or *sensory aphasia* (Wernicke). The memory for words can be disturbed in various ways; thus, the memory of names may be wholly or partially lost. The disturbance in which a patient hears spoken words but does not recognize their meaning is known as *word deafness*.

When the patient whose speech muscles are not paralyzed has certain words and syllables in his consciousness but is unable to give expression to them in speech, he is said to suffer from *motor aphasia*. Such patients also are unable to speak words pronounced before them. They are never entirely dumb, being always able to give expression to a few words or parts of words; indeed, there is every gradation from syllable stumbling to almost complete dumbness.

[1] For the literature of the subject and for excellent accounts the reader is referred to von Monakow (C.). *Gehirnpathologie*, second edition; Dejerine (J.), *Sémiologie du système nerveux* in Bouchard's *Traité de pathologie générale*, Paris, 1901, vol. v; and Moutier (F.), *L'Aphasie de Broca*, Paris, 1908.

Similar to aphasia are the disturbances known as agraphia and apraxia. In *agraphia*, although there is no actual paralysis of the muscles of the upper extremity, the patient is unable to write down words which he hears or sees or remembers. Usually agraphia is combined with aphasia, but in pure agraphia it exists by itself without aphasia. In *apraxia* (or dyspraxia) the patient is unable to carry out from memory certain complicated movements of his limb muscles. Apraxia seems to be due most often to lesions of the left hemisphere or of the corpus callosum.

The incapacity to understand written or printed matter is known as *alexia*.

When all the components of speech (perceptive and expressive) are interfered with, the condition is known as *total aphasia*. When the speech anomaly is chiefly on the expressive side, we designate it a *motor aphasia*. In the most common form of motor aphasia, that known as *Broca's type*, speech is almost abolished and the patient is unable to pronounce words spoken before him. He cannot read aloud, he has difficulty in spontaneous writing, in writing to dictation, and in copying. He may, however, understand words which he hears and sees. In cases of this type, in which the agraphia is much more pronounced than the speech disturbance, the special term of *cheirokinesthetic agraphia* has been applied.

In so-called *pure motor aphasia* (*subcortical motor aphasia* or *aphemia; pure word dumbness*) there is almost complete inability for spontaneous speech, for pronouncing words spoken before the patient, and for reading aloud, but there is no disturbance of writing and the understanding of speech and of writing is normal. In sensory aphasia, as has been said above, it is the perceptive side of speech which is interfered with. Here, again, we distinguish ordinary sensory aphasia (Wernicke's type) from pure word deafness.

In *ordinary sensory aphasia of Wernicke's type* (*cortical sensory aphasia*) the patient is unable to understand what he hears and reads. His storehouse of spoken words is somewhat diminished and his speech is *paraphasic* in that instead of using the words which he intends to employ he gives expression to others which have a similar sound. He is unable to pronounce words spoken before him, or if able to do so does not understand their meaning. The emphasis he puts upon spoken words is faulty. On spontaneous writing and on writing to dictation he manifests a verbal agraphia or mixes up the letters in words (*paragraphia*). He copies only with difficulty.

In so-called *pure word deafness* (*subcortical sensory aphasia*) internal speech may be normal, there is some deafness, and the patient is totally unable to understand words spoken to him, nor can he speak words pronounced before him. He may, however, be able to express himself quite well in writing and to understand what he reads.

At the beginning of the sensory aphasias, especially in tumors or after brain injuries, patients sometimes pass through a transitory state in which motor speech is undisturbed, but there is difficulty in finding the names for objects and persons (concrete substantives). Such patients understand spoken and written words perfectly well, but on spontaneous writing they have the same difficulty as on speaking, although writing to dictation and copying may be quite normal. This particular condition is sometimes spoken of as *word amnesia* or *anomia*.

Two kinds of *pure word blindness* (*subcortical alexia* of Wernicke) have been described, one without agraphia, the other with agraphia. In *pure*

*word blindness without agraphia* spontaneous speech and the understanding for spoken words are normal, but single letters and syllables cannot be read. The patients may write, but fail to understand the words that they have written themselves. They can write to dictation, but cannot copy. The condition is usually associated with hemianopsia dextra.

In *pure word blindness with agraphia* spontaneous speech and the understanding of spoken speech are normal, but there is complete alexia combined with agraphia and paragraphia, these symptoms being optic in their origin. The patient cannot write to dictation nor can he copy the writing of others.

**Anomalies of the Reflexes.**—A study of the disturbances of reflex action is of great importance in neurological and psychiatric diagnosis. Three main groups of reflexes should be examined: (1) The pupillary reflexes. (2) The deep reflexes (tendon and periosteal reflexes). (3) The superficial reflexes (cutaneous and mucosal reflexes).

**Pupillary Reflexes.**—These include (1) the light reflex, and (2) the accommodation and convergence reaction.

In testing the *light reflex* one must avoid calling forth movements due to accommodation or convergence. Each eye should be tested for itself, and one should also see whether or not the *consensual* reflex is present, remembering that on unilateral illumination there should be a pupillary contraction in both eyes.

In testing the *reaction on convergence* one asks the patient to look first at the ceiling and then quickly at the end of the nose. When the eyes converge for near vision there is also a contraction of the pupil due to accommodation. But even when the medial recti are paralyzed and convergence is no longer possible, there may be an accommodative pupillary contraction.

If the light reflex is abolished or diminished the condition is known as *reflex pupillary rigidity*. This may be due to partial or complete blindness (amaurosis or amblyopia) or to paralysis of the M. sphincter iridis; in other words, it may depend upon the centripetal fibers of the reflex arc (nervus opticus) or upon the centrifugal fibers of that arc (nervus oculomotorius).

When the pupils do not react to light but do react well on accommodation and convergence, we speak of the *Argyll-Robertson pupil*. Its presence is of very great significance in the diagnosis, especially of tabes and of dementia paralytica. The condition is usually bilateral, but may in early stages be unilateral. When the pupils react neither to light nor accommodation (*absolute pupillary rigidity*), there is complete ophthalmoplegia interna. This condition is not uncommon in cerebral syphilis, and may be met with also in brain tumor, tabes, or dementia paralytica.

The size of the pupils varies a good deal in health with moderate illumination. Abnormal contraction of the pupils (*myosis*) occurs in opium poisoning and in various conditions which irritate the oculomotor nerve or paralyze the sympathetic nerve. It should be borne in mind that myosis occurs physiologically in old people and in sleep. Abnormally large pupils (*mydriasis*) are seen in atropine and cocaine poisoning. The condition may also be due to oculomotor paralysis, to atrophy of the optic nerve, or to irritation of the cervical sympathetic.

Inequality in the size of the pupils (*anisocoria*) may be due to unequal illumination or to differences in the refractive media of the two eyes, but most often it indicates unilateral nervous disease of the optic, oculomotor, or

sympathetic nerves. It is not infrequently present in dementia paralytica and in tabes.

**Deep Reflexes.**—Of these, by far the most important are the knee-jerk, the heel-jerk, and the periosteal reflex.

The *knee-jerk* (*patellar tendon reflex, knee kick*) is the contraction of the M. quadriceps femoris which follows tapping upon the ligamentum patellæ. Sometimes the so-called *reinforcement* of Jendrassik is necessary; the patient clasps his hands and is told to pull at the moment one taps the patellar tendon. As a rule, it is more satisfactory to make the examination in the recumbent position.

*Achilles-jerk or Foot-jerk.*—The patient is placed on his knees in a chair, the feet hanging loosely over the end of the chair. One then taps upon the Achilles tendon with a percussion hammer. The reflex is not always present, even in healthy people, although generally so. If the Achilles jerk be exaggerated the percussion stroke may give rise to clonic contractions or an actual foot clonus instead of to a single contraction.

*Periosteal Radial Reflex*—On tapping the lower end of the radius one can see a contraction of the M. brachioradialis at the bend of the elbow, leading to flexion and slight pronation of the forearm and hand.

The other deep reflexes which may be tested are: (1) *Tibial reflex* (striking anterior surface of tibia to get contraction of M. quadriceps femoris); (2) the *biceps reflex* (tapping the biceps tendon at the bend of the elbow); (3) the *triceps reflex* (holding arm at a right angle and tapping on the triceps tendon); (4) the *jaw-jerk* (laying finger above chin with the mouth closed and giving slight tap with percussion hammer to get contraction of the masseters).

The deep reflexes may be increased or diminished. An increase may be due to irritation of the sensory limb of the arc (neuritis, meningitis), to stimulation of the anterior horn cells (strychnine poisoning), or to diminution of inhibitory influences acting from above upon the reflex arc (neurasthenic states, lesions of the pyramidal tract). The deep reflexes may be diminished or abolished through injury to the sensory or motor nerves or injury to the portion of the arc within the central nervous system.

**Cutaneous Reflexes.**—The three principal ones to be considered are: (1) The plantar reflex; (2) the cremaster reflex; (3) the abdominal reflex.

The *plantar reflex* is elicited by applying a stimulus to the sole of the foot. Under normal conditions it leads to an involuntary contraction of certain muscles of the lower extremity, the so-called "tickle response" being a kind of movement away from the irritating object. Most important, however, is the behavior of the toes, and especially of the great toe. Under normal conditions plantar stimulation is followed by plantar flexion of the toes.[1] On the contrary, when the pyramidal tract is injured, instead of plantar flexion there is dorsal flexion, especially of the great toe, and the movement of the toe occurs less rapidly than under normal conditions (*Babinski's phenomenon*). In children during the first few months of life, plantar stimulation causes dorsal flexion of the great toe, but after the first few months of life the normal reflex is one of plantar flexion. When the pyramidal tract is diseased the dorsal flexion of the great toe is often accompanied by

[1] Babinski, *Sur le réflexe cutané plantaire, Comptes rendus de la Soc. de Biol.*, 1897.

spreading of the other toes, and especially by abduction of the little toe. This has been called by Babinski the *fan sign* (*signe de l'eventail*).

Similar in its meaning to the Babinski phenomenon is the response obtained by rubbing the medial surface of the tibia downward with the pulp of the thumb or with the handle of a percussion hammer. Normally, as the malleolus is approached, this causes plantar flexion of the toes, but in spastic states there results, as a rule, dorsal flexion of the foot and especially of the great toe (*Oppenheim's sign*).

Still another sign of similar import may be mentioned. If one taps the lateral part of the proximal half of the back of the foot, corresponding to the base or middle of the third and fourth metacarpal bones, the cuboid bone, and the second cuneiform bone, dorsal flexion of the toes occurs under normal conditions, while in spastic states we meet, instead, with a plantar flexion of the toes, sometimes with spreading of the toes (*Mendel's sign*).

**Cremaster Reflex.**—This is elicited by stroking the medial surface of the thigh in the adductor region or by pinching the skin in this location. The normal response consists in a contraction of the cremaster muscle, with elevation of the testicle. This reflex is not to be confused with the scrotal reflex, which consists of a contraction of the tunica dartos with wrinkling of the skin of the scrotum on stimulation of the skin in this neighborhood.

**Abdominal Reflexes.**—One strokes the skin of the abdomen with the end of the finger or with the blunt point of some instrument, and notices the contraction of the abdominal muscles which follows. It may be elicited either in the supra-umbilical or in the infra-umbilical region on each side (*epigastric and hypogastric reflexes*). A unilateral absence of the reflex is of most importance.

**Other Cutaneous and Mucosal Reflexes.**—Among the other reflexes may be mentioned the *palmar reflex*, *scapular reflex*, and the *uvular reflex*. Especial mention perhaps should be made of the *lid reflex* (closure of the lids when an object is brought suddenly near one eye) and the *conjunctival* and *corneal reflexes* (closure of lid on stimulation of the conjunctiva or cornea).

The reflexes set free by stimulation of the skin and mucous membranes have an entirely different significance from the deep reflexes. The cutaneous reflexes may be absent when the peripheral nerves are diseased; indeed, whenever the reflex arc is interrupted.

One of the most interesting facts connected with the cutaneous reflexes is that they are especially disturbed in unilateral cerebral disease. Aside from the Babinski phenomenon, a hemiplegia usually leads to loss of the abdominal and cremaster reflexes on the paralyzed side, perhaps owing to an increase of the influences which inhibit the activity of the reflex arc, although this explanation is not universally accepted.

**Anomalies of the Vasomotor, Secretory, and Trophic Functions.**—The vasomotor functions of the nervous system are very complex, and disturbances in different parts can lead to vasoconstriction on the one hand, or to vasodilatation on the other. For a full discussion of these vasomotor phenomena the reader is referred to the recent lecture by Porter,[1] the article by Eulenburg-Landois, and to the excellent treatise of Cassirer.[2] Here belong the angioneuroses, acroparæsthesias, angioneurotic œdemas, symmetrical gangrenes, erythromelalgias, acrocyanoses, etc.

[1] *Vasomotor Relations, The Harvey Lectures*, 1906–1907.
[2] *Die vasomotorisch-trophischen Neurosen*, Berlin, 1901

Trophic disturbances may concern the muscles, the bones and joints, the skin, and its appendages. The trophic changes in the bones and joints have been best studied in tabes, in syringomyelia, and in acromegaly. In tabes spontaneous fracture of the bones frequently occurs, and still more frequently certain arthropathies, which occur suddenly, usually without pain, and lead quickly to disintegration of the joints.[1] In acromegaly, owing apparently to disease of the hypophysis, there is hypertrophy of many of the bones of the body, especially of the bones of the hands and feet, of the nose, and of the lower jaw.

Trophic changes which take place in the skin include bedsores (decubitus), falling of the hair, nails, and teeth in tabes, perforating ulcer of the foot in tabes and syringomyelia, and the panaritium of the finger tips in syringomyelia. Scleroderma and progressive facial hemiatrophy also belong here. Disturbances of the secretion of the sweat glands and of the sebaceous glands of the skin are sometimes met with in nervous disorders. The anomalies of the secretion of saliva, of the stomach juice, of the urinary secretion, etc., should also be mentioned. As a matter of fact, but little is known as yet concerning the exact mechanisms underlying the vasomotor, trophic, and secretory neuroses.

[1] Barker (L. F.). *Joint Affections in Nervous Diseases, Jour. Amer. Med. Assoc.*, 1907, vol. i.

# CHAPTER II.

## DISEASES OF THE MOTOR TRACTS.

By WILLIAM G. SPILLER, M.D.

### PROGRESSIVE SPINAL MUSCULAR ATROPHY.

**Synonyms.**—Muscular atrophy, type Duchenne-Aran; chronic anterior poliomyelitis.

**Definition.**—By the Duchenne-Aran type of muscular atrophy is meant a progressive muscular wasting related to degeneration only of the cells of the anterior horns of the spinal cord and their peripheral processes. While it usually begins in the small muscles of the hand, it may begin in the muscles of the shoulder, and is even then designated as the Duchenne-Aran type. While usually asymmetrical, it may be unilateral for a time. The scapulo-humeral type was first described by Vulpian.

**History.**—Many authors had contributed to the subject of muscular atrophy before the time of Duchenne of Boulogne, and Aran, but the impetus to the study of this condition was given by the writings of these two men. Among the early investigators were Dubois, van Swieten, Abercrombie, Sir Charles Bell, Graves, Darwall, and Cruveilhier. Beevor says the first case recorded was by Sir Charles Bell in 1836. The name of Aran is sometimes placed before that of Duchenne and sometimes follows it. A paper was published by Duchenne in 1849, and it was not until a year later that Aran's work appeared. A further study by Duchenne was made in 1853. Both Duchenne and Aran looked upon the atrophy as muscular in origin. The anatomical investigations of Cruveilhier led to the disease being regarded as atrophy of the anterior roots. Luys demonstrated degenerative changes in the cells of the anterior horns. These and the investigations of Charcot, Joffroy, and others led to the conclusion that all progressive muscular atrophy was of spinal origin from disease of the cells of the anterior horns, a view that later was shared by Duchenne, although it was contrary to that he had previously held. The confusion at that period was very great, nor can it be said at the present day that complete order has been brought out of the chaos. Duchenne included all progressive muscular atrophy, whether occurring in families, in young or old persons, or as an acquired process, under one head. Gradually different types of muscular wasting were separated from the Duchenne-Aran form. Progressive muscular dystrophy in the pseudo-hypertrophic form had been observed by Duchenne in 1853, and was clearly established by the writings of Eulenburg, Charcot, Leyden, Möbius, Lichtheim, and Landouzy and Dejerine. Duchenne at first erred in believing the pseudo-hypertrophic muscular paralysis was of cerebral origin, inasmuch as his earlier cases manifested feeble intelligence, but later he recognized his mistake. He called the disease *paralysie*

*musculaire pseudo-hypertrophique*, and the name he gave it has clung to it to this day. Erb established the unity of the various types of myopathy, but at first only the pseudo-hypertrophic and the Leyden-Möbius types were recognized. Charcot separated the amyotrophic lateral sclerosis from the general groups of atrophy. Charcot and Joffroy distinguished the cervical hypertrophic pachymeningitis in 1871 to 1873, although it cannot be said that the symptoms of this disease are so sharply defined as are those of amyotrophic lateral sclerosis. Charcot recognized as protopathic the atrophy due primarily to lesions of the nerve cells of the anterior horns, and as deuteropathic that resulting from changes within the spinal cord but affecting the anterior horns secondarily. Schultze and Kahler, in 1888, still further weakened the Duchenne-Aran type of muscular atrophy by separating the great group of syringomyelia. It began to look as though nothing would be left in the Duchenne-Aran type, and indeed it is stated that Charcot himself felt that it stood on an insecure foundation.

In 1897 Marie came forth boldly in defiance of the accepted teaching. He pointed out that Duchenne at first believed that progressive muscular atrophy was of peripheral origin; later, he believed it to be of spinal origin. The disease at first, as understood by Duchenne, included all the forms of muscular atrophy not resulting from fracture. Marie then described how amyotrophic lateral sclerosis, progressive muscular dystrophy, multiple neuritis, and syringomyelia were separated from the great group of atrophies. Fully one-third of Duchenne's cases, from his own description, Marie thinks, belonged to syringomyelia, although this disease at that time was not recognized. Thus, Pierre Marie says that the progressive muscular atrophy of Duchenne-Aran, which earlier authors regarded as the most solid foundation of neuropathology, has ceased to exist. Even the hand which is commonly regarded as of the Duchenne-Aran type is, according to Marie, not the deformity Duchenne described, and is what Duchenne believed to be characteristic of leprosy. The hand that he described as typical of his progressive muscular atrophy Marie believes belongs to syringomyelia. The atrophy is not so intense, and the fingers are not so much flexed; and yet in comparing the two hands, as pictured by Marie, the differences do not appear very great, and seem to be more in the degree of deformity. Marie, however, believes that Duchenne merely described a symptom complex, and that nothing now remains of his progressive muscular atrophy.

**Etiology.**—Pregnancy may have some influence on the development of spinal muscular atrophy, though it would seem questionable whether it be able to originate it when no tendency to the disease exists. In Oppenheim's second case of chronic poliomyelitis, a complicated case, weakness of the muscles of the right shoulder and of the right upper limb developed during the fourth pregnancy; the following year the left upper limb became affected during pregnancy. Nonne attributed the poliomyelitis in his second case to diabetes. A case reported by Erb seems to show some relation to trauma. Other authors speak of trauma, notably Ziehen.

Heredity does not play a role, and yet Bruining has observed chronic anterior poliomyelitis in father and son, and in one of the cases obtained a necropsy; this family occurrence was probably merely a coincidence.

Age seems to be the most important factor, the symptoms usually appearing in middle life. The infantile form of progressive spinal muscular atrophy is considered separately. Myelopathy is probably an abiotrophy, to employ

a term originated by Gowers, and is the result of an imperfectly developed motor system unable to withstand the stress of advancing years.

It is exceedingly questionable whether infections or intoxications cause the symptoms, although the development of progressive muscular atrophy many years after an arrested acute poliomyelitis is regarded by Léri as evidence of this origin. Undoubtedly infection is a cause of the acute form of poliomyelitis, and the nerve cells left weakened by this process may degenerate later in life, but this is not a proof that the chronic form is so caused. Some toxic substance acting on the cells weakened by acute poliomyelitis may cause progressive muscular atrophy. It seems probable that lead may be one of these agents. The acute and chronic forms probably have a very different pathology. The male sex is regarded by certain authors as much more frequently affected than the female.

Some of the cases supposed to represent progressive spinal muscular atrophy are caused by myelitis, especially the syphilitic form. The inflammation may be almost confined to the gray matter, and such being the case, sensory disturbance may be absent. The symptoms then would be those of progressive spinal muscular atrophy, although the lesion would be a diffuse process of inflammatory character.

**Pathology.**—The characteristic changes are atrophy and degeneration of the nerve cells of the anterior horns of the spinal cord, and also in some cases of the motor nuclei of the medulla oblongata. Sometimes many of the nerve cells entirely disappear. Numerous hemorrhages have been observed in the gray matter in some instances (Bielschowsky, Spiller), extending even throughout the cord. The motor roots are atrophied, and contain fewer fibers than normal. The white matter of the spinal cord remains unaltered in a typical case, or at least the sclerosis is not in the area occupied by the crossed pyramidal tracts. It is questionable whether those cases in which a very slight degeneration of these tracts occurs, detectable only by the Marchi stain, should be classed as progressive spinal muscular atrophy or amyotrophic lateral sclerosis. The symptoms and the findings in the anterior horns may indicate that the lesions of the anterior horns have been of long duration, while the degeneration of the lateral columns evidently is very recent, and no clinical evidence of the slight degeneration of the pyramidal tracts may have been detected during life. It seems best to regard such cases as belonging to progressive spinal muscular atrophy or chronic poliomyelitis, but as marking a transitional stage to amyotrophic lateral sclerosis.

Beevor in disputing the correctness of Gowers' view that amyotrophic lateral sclerosis and progressive spinal muscular atrophy are the same disease, mentions that he (Beevor) has had a case in which the symptoms began with atrophy of the small hand muscles, and later the shoulder muscles became affected, the lower limbs were only slightly implicated. Rigidity or increase of the deep reflexes was not present at any period. Atrophy of the cells of the anterior horns was found, but the lateral columns were intact.

Degenerative changes in the anterolateral columns may be recognized as belonging to anterior poliomyelitis and progressive spinal muscular atrophy, provided the pyramidal tracts are not affected. It must be accepted that certain of the column cells may degenerate as well as the cells of the anterior roots. When the pyramidal tracts become implicated the cases are on the border line of amyotrophic lateral sclerosis. Credit has been given to

Oppenheim as reporting, in 1888, the first well-described case of chronic poliomyelitis with necropsy. The nerve cells of the anterior horns of the cord, and to some extent those of the medulla oblongata, were degenerated; a slight atrophy was found in the anterolateral columns. The anterior roots were affected, as would be expected, but not to the degree that the cellular degeneration seemed to demand. Perivascular round-cell infiltration has been seen in some cases (Bielschowsky).

It is uncertain whether the cellular changes are primary or secondary. In some instances they may be primary, in others they may be the result of inflammation in the surrounding tissues. Where the process has advanced so far that most of the cells have disappeared, the neuroglia of the anterior horns appears denser and the few nerve cells that remain show much pigmentation. The cellular destruction is usually greater in the cervical swelling; and the anterior roots usually are much atrophied. The peripheral nerves and muscles show more or less alteration.

Syphilitic myelitis probably is the lesion underlying a certain number of the cases of progressive muscular atrophy. Progressive ophthalmoplegia may become associated with degeneration of the motor cells of the medulla oblongata and spinal cord, as in two cases observed by Dana.

Cases of progressive spinal muscular atrophy sometimes occur in museums as examples of "living skeletons." Such a case with necropsy was reported by E. W. Taylor. The nerve cells of the anterior horns had almost entirely disappeared, but the pyramidal tracts were not degenerated.

It seems to Dana an unnecessary confusion of symptomatology to transfer the diagnosis of progressive muscular atrophy of the Duchenne-Aran, or other type, to that of amyotrophic lateral sclerosis so soon as a little spasticity begins, especially as there may be nothing peculiar in age, course, or duration. The atrophy may start in the hands and ascend to the shoulders before there is any evidence of lateral sclerosis. The disease may then become arrested, the spastic symptoms disappear, and the patient may again present the type of Duchenne-Aran atrophy without any spastic symptoms. All that we can do, in the writer's opinion, is to acknowledge that the lateral columns may be slightly affected without producing clinical manifestations, but because we cannot always recognize this slight degeneration clinically is not sufficient reason to decline to make distinctions in the pathological forms of spinal muscular atrophy. It is a question whether we are solving the problem by following Dana in limiting the use of the term amyotrophic lateral sclerosis to those cases which show only from the beginning and dominantly the spastic and contracturing type of progressive muscular atrophy. Why must degeneration of the lateral columns develop previously to or simultaneously with disease of the cells of the anterior horns, and not later?

**Symptoms.**—The symptoms of chronic anterior poliomyelitis and progressive spinal muscular atrophy vary in typical cases. In the former the paralysis is supposed to develop within a few days or a few weeks, the atrophy appears later, and entire muscles or groups of muscles are paralyzed, the course of the disease is more rapid, and the paralysis develops first in the muscles of the lower limbs or shoulders. In progressive spinal muscular atrophy, the paralysis is proportional to the atrophy, one muscle fiber after another is affected, and the course is longer than in poliomyelitis. These distinctions are not always regarded, and some of the reported cases of

so-called poliomyelitis might with greater right be regarded as examples of progressive spinal muscular atrophy, or even as transitional forms to amyotrophic lateral sclerosis.

Dejerine and Thomas, who describe the disorder under the name of chronic anterior poliomyelitis, say that atrophy is the primary and essential symptom, and paralysis is secondary and proportional to the atrophy. Thus, according to these authors, one of the most important diagnostic points between chronic poliomyelitis and progressive spinal muscular atrophy disappears. Beevor, likewise, makes no sharp distinction between the two processes, and remarks that wasting may begin in the lower limbs.

The first indication of disturbance may be awkwardness in the use of one or both hands. Usually one is affected before the other. Abduction and apposition of the thumb with the other fingers become affected, then the interossei and lumbricals waste, and the whole hand becomes much atrophied, including the thenar and hypothenar eminences. The tendons of the palm become very prominent. When the thenar eminence is wasted the hand is of the simian type. Later, when the interossei and lumbricals are attacked the flexion of the phalanges upon the metacarpal bones and the extension of the phalangeal articulations become impaired, so that the first phalanges are extended and the second and third flexed, the palmar tendons are prominent (*main en griffe*). The atrophy gradually extends to the flexors on the forearm, then to the extensors. According to Beevor, the flexors of the fingers and thumb are affected before the flexors of the wrist. The atrophy reaches the deltoid and muscles of the upper part of the limb and shoulder girdle. Beevor states that the triceps, latissimus dorsi, and the lower half of the pectoralis major usually escape. The trunk and neck muscles waste and the ribs become prominent; the lower limbs likewise become greatly atrophied. Dejerine and Thomas dispute the extension of the process to muscles innervated by bulbar nerves, as no necropsy has demonstrated such an extension, and they assert that the face is always intact, and Dejerine believes that bulbar palsy belongs to amyotrophic lateral sclerosis. Beevor, however, speaks of bulbar paralysis as a part of chronic anterior poliomyelitis, and Léri also mentions atrophy of the muscles of the face, although he adds that the condition is very different from that of bulbar palsy.

Quite a number of cases are recorded in which the atrophy began in the lower limbs, especially in the peroneal distribution, and in some instances (Moleen and Spiller) the weakness and atrophy developed rapidly. The upper limbs became implicated later. In other cases the muscles of the shoulder girdle are first affected (Vulpian, Dejerine) or the extensors of the fingers and wrists, as in a case reported by C. S. Potts. The muscles of the trunk may be first affected.

The weakness and atrophy probably begin more commonly in the hands, because the movements of these parts are highly differentiated; it is not probable that the weakness is first noticed in muscles employed frequently in specialized movements simply because the interference with these movements is more striking to the patient, and that in reality other muscles are affected at the same time. Edinger's exhaustion hypothesis, that those muscles most used are most likely to be the first to show alteration, is a reasonable explanation for the commencement of the symptoms in the hands in most cases. The paralysis is flaccid and the joints are relaxed,

producing the condition known as flail joint, especially noticeable at the wrists. Grunow and Loevegren go so far as to make the atrophy of the lower limbs the first sign diagnostic for poliomyelitis in distinction to progressive spinal muscular atrophy.

Fibrillary tremors are very common in the atrophying muscles; sometimes they implicate small bundles of muscle fibers, and then they may more properly be described as fasciculary. These quick, wave-like movements are not present in the atrophying muscles at all times, but in most cases may be detected at some period or other. They may be so pronounced as to correspond to the condition known as "muscular madness" (*folie musculaire*), but they do not cause movement of a segment of the limb, unless possibly of a finger. They are an early sign, and disappear as the atrophy of the muscles becomes intense. They may be present at one period in spinal atrophy, but later disappear, so that the absence of fibrillary tremors by no means excludes a spinal origin.

The tendon reflexes of the affected limbs become diminished or lost, and may be lost before atrophy appears, the cutaneous reflexes become less active where the muscles are wasted. Exaggerated reflexes should make the diagnosis of progressive spinal muscular atrophy or chronic anterior poliomyelitis doubtful, but, according to certain authors, do not exclude this diagnosis. Thus Oppenheim does not regard exaggeration of the tendon reflexes as sufficient to make a case of muscular atrophy one of amyotrophic lateral sclerosis.

The electrical reactions are important in the diagnosis between the myelopathic and the myopathic forms of muscular atrophy. In the latter the irritability is diminished, but the formula is not altered; in the former the nerves may respond to the faradic and galvanic currents, although not in the same degree; later the response to either current may be lost. The muscles at first require a stronger current, then their faradic irritability may be lost; when the galvanic irritability is increased, the formula may be reversed, so that the anodal closing contraction is equal to or greater than the cathodal closing contraction (reaction of degeneration); still later all electrical irritability of the affected muscles may be lost. Especially valuable as a sign of myelopathy is the modal change, so that the muscles contract very slowly to the galvanic current. The entire muscle does not always show electrical alteration, and only a few fibers may present this change, while adjoining fibers in the same muscle are normal; this is because fibers much atrophied may be in juxtaposition to normal fibers.

Sensory changes are not a part of myelopathy. Objective disturbance of sensation does not occur in an uncomplicated case, and while pain may occasionally be complained of, it is of a peculiar kind; it is the pain from overuse of wasting muscles, and not that from irritation of sensory fibers. It is more a dull aching like that in fatigued muscles, and not sharp, such as is felt in toothache. Some authors speak of pain occurring in the beginning of myelopathy, but skepticism as regards the correctness of the diagnosis is justifiable when pain is intense.

The sphincters of the bladder and rectum escape, but theoretically one may understand how they could become affected if the nerve cells of the conus were degenerated. Atrophy of bone has been described by some authors. Cutaneous lesions are probably epiphenomena. Vasomotor symptoms may be present, and the atrophied limbs may be cold.

Progressive spinal muscular atrophy may develop on a foundation of old arrested acute poliomyelitis. C. S. Potts has paid particular attention to muscular atrophy occurring in this way, and has collected the records of 36 cases. In 28 the condition was probably progressive muscular atrophy; in 2, amyotrophic lateral sclerosis; in 2, myelitis; and in 4, another attack of acute poliomyelitis. The interval elapsing between the primary and secondary attacks ranged from seven years to about fifty-five years, the average being about twenty-three years. In 18 cases out of 33, in which the part secondarily affected could be determined, the late atrophy began in a limb which had previously been affected by the primary disease; in 7 it began in the other limb on the same side that had been primarily affected; in 5 in the corresponding limb of the side opposite to the part first affected; in 2 in a limb of the opposite side not corresponding to the limb first affected; and in one the involvement was general, this being a case in which acute poliomyelitis occurred a second time. The most plausible explanation for this late extension is that nerve cells are left in a weakened condition by the acute anterior poliomyelitis, and readily succumb to any damaging process.

*Chronic external ophthalmoplegia* is probably a form of progressive muscular atrophy of the central type. It is a progressive paralysis of the external ocular muscles. Numerous cases are recorded in the literature. It is usually a disease of infancy, but may develop later in life.

**Diagnosis.**—Remembering the pathology we understand why the symptoms are muscular atrophy, weakness, loss of reflexes, and changes in electrical reactions. The process is slow (progressive muscular atrophy) or in periods of exacerbations (subacute or chronic poliomyelitis).

*Progressive muscular dystrophy* is to be distinguished by the age at the onset, the involvement first of the proximal parts of the limbs, the hereditary or family tendency, the absence of fibrillary tremors and of reaction of degeneration, the presence of pseudo-hypertrophy, etc. *Amyotrophic lateral sclerosis* is clinically progressive spinal muscular atrophy or chronic poliomyelitis with exaggeration of tendon reflexes, and pathologically with degeneration of the cells of the anterior horns and peripheral motor fibers and the pyramidal tracts; the latter causes exaggeration of the tendon reflexes. *Multiple neuritis* is usually associated with spontaneous pain, objective sensory disturbances, and tenderness of nerve trunks. The onset is usually more rapid, and the symptoms may be confined to the distribution of certain nerves. *Acute poliomyelitis* is to be recognized by signs of infection and rapid development of symptoms. The paralysis at first may be extensive, but gradually certain of the affected muscles recover, leaving others permanently paralyzed. *Syringomyelia* has the dissociation of sensation, impairment or loss of temperature, and pain sensations, with much better preservation of tactile sensation; also trophic disturbances, and it may be more nearly unilateral. The *neurotic muscular atrophy* of Charcot, Marie, and Tooth may closely resemble chronic poliomyelitis, but where sensory disturbances are present the distinction is clear. It is likely to be hereditary, and is more common in males. The atrophy is confined to the peripheral parts of the limbs, even after many years. *Leprosy* may have some resemblance in that it may begin in the hands and extend toward the trunk in the upper limbs, implicate the lower limbs in a similar manner, and be associated with reaction of degeneration. The lepra bacillus in the tissues, the dissociation of sensation like that of syringomyelia, the local

swellings in the nerves, and the history of contagion make the diagnosis possible. *Cervical hypertrophic pachymeningitis* may cause an atrophy of the hands like that of progressive spinal muscular atrophy, but there is a period of pain in the upper limbs with objective sensory disturbances.

**Prognosis.**—Arrest is possible, but improbable. The weakness and atrophy are likely to extend until a large portion of the body is affected, or muscles of vital function, as those of respiration or of the heart, are attacked, or death occurs from some intercurrent disease, but usually the process is very slow, and in one of Dejerine's cases the symptoms lasted about sixteen years.

**Treatment.**—This is of little value. Electricity, especially in the form of the constant current, moderate exercise, and passive movements are recommended and may be useful. Tonics and exercise in moderation help to keep up the general strength, but nothing will restore the muscles atrophied from degeneration of their nerve cells, or probably have much effect in arresting the course.

## PROGRESSIVE SPINAL MUSCULAR ATROPHY OF CHILDHOOD, OF FAMILIAL OR HEREDITARY CHARACTER (TYPE WERDNIG-HOFFMANN).

**History.**—The first cases were described by Werdnig, who reported 2; Hoffmann followed, with a report of 20 cases; Thomson and Bruce, with 1 case; Bruns gave, in 1901, a description founded on the cases in the literature to that date. Hoffmann's cases occurred in three families; he examined 6 clinically, 2 anatomically. Both of Werdnig's cases were with necropsy, and occurred in the same family, the patients had the same mother but different fathers. In Hoffmann's third family the disease was evidently transmitted through the mother. Bruns' 3 cases came from three different families. The family character was evident in the first case, as two of the other offspring of the parents had died from the same disease. Nothing concerning family tendency could be determined in the second case. The third patient was the only one affected of four children. Although 2 of his 3 cases manifested no family tendency, Bruns classifies them under this type, as Bruce's case also was the only one in the family. The cases of Bruns, added to those of Werdnig, Hoffmann, and Bruce, make 28 cases in the literature, of which only 12 were observed by physicians. To these should be added 2 cases without necropsy observed by Senator. The cases of Werdnig and Hoffmann began between the sixth and ninth months; only a few of the children could stand before the disease began, and none could walk. Death occurred in the most rapid case in the eleventh month; in the slowest in the fifth year. Bruns' first patient could walk when about a year and three-quarters old, and died in the fifteenth year. His third patient also could walk when about one year and one-quarter old, and was still alive in the twelfth year. Senator's two cases were in brother and sister. The symptoms began in both at about the age of two years.

Hoffmann has attempted to classify the cases of hereditary progressive spinal and bulbar muscular atrophy, and makes the following types:

1. Occurrence in early childhood. Implication first of the pelvic girdle

and atrophy extending from there toward the ends of the limbs (Werdnig, Hoffmann).

2. The bulbar paralytic facial type of childhood (Fazio, Londe).

3. A Duchenne-Aran type (Strümpell, Gowers).

4. An intermediate form (Bernhardt), which Bernhardt regards as of spinal and bulbar origin.

It seems to the writer that the atrophy of Werdnig-Hoffmann may be regarded clinically as progressive spinal muscular atrophy, differing from the usual type seen in adults in the commencement within the first years of life, in the implication first of the pelvic girdle and portions of the limbs near the trunk, and in the strong hereditary tendency, a tendency very feebly manifested in the progressive spinal muscular atrophy of adults; but pathologically the disease is amyotrophic lateral sclerosis of childhood, differing from the type in adults by the absence of exaggeration of tendon reflexes, by which we may infer that the peripheral motor segments are always involved first. The case of Thomson and Bruce shows that they alone may be affected.

**Pathology.**—The findings described by Hoffmann are: Symmetrical intense degeneration of the peripheral neurones of all the motor fibers below the hypoglossus, including the accessorius, *i. e.*, degeneration or disappearance of the multipolar cells of the anterior horns, so that few are left in a transverse section; intense degeneration of the anterior roots of the spinal cord, less intense changes in the peripheral nerves and the intramuscular nerve endings; degeneration of the crossed and direct pyramidal tracts, and of a portion of the lateral fundamental columns of the cord, more intense in the upper thoracic and cervical regions, but not traceable above the pyramidal decussation. The muscles show simple atrophy in all stages without complete disappearance of fibers, without multiplication of the sarcolemma nuclei, and with very little lipomatosis of the muscles. There seems to be rather a decrease in the number of muscle nuclei. The transverse striation of the muscles is preserved, but some of the muscle fibers may completely disappear. The alteration of the spinal cord is more intense than that of the muscles, so that the disease in Hoffmann's opinion is spinal in origin. In one case the anterolateral column was not degenerated (Werdnig). In only two of the four cases was lipomatosis found, and in one (Werdnig) homogeneous and granular degeneration of the muscle fibers was observed.

**Symptoms.**—The description given by Bruns is as follows: The disease begins in early childhood, progresses slowly, without fever or convulsions. The children never learn to walk, or gradually lose the ability to walk if they learned before the symptoms began. They cease to move the lower limbs in bed and to hold the trunk erect. Standing becomes difficult, and is possible only in the beginning of the disease, and then only with support. Paresis and atrophy of muscles occur nearly simultaneously in the muscles of the pelvic girdle and trunk, implicating symmetrically the iliopsoas and quadriceps femoris, and later the upper limbs and neck muscles. The proximal portions of the limbs, shoulder girdle, and pelvic girdle are affected, and the atrophy and weakness diminish toward the hands and feet, although these parts do not entirely escape in the late stages, but their movements are better than would be expected from the atrophy. The erect position of the head and trunk is difficult to maintain; the child cannot lie down slowly,

and raises itself with difficulty. The vertebral column is distorted because of muscular weakness. The feet are in the position of talipes equinus. The atrophy is *en masse*, and may be partly concealed by fatty tissue. Diminished electrical response or reaction of degeneration is observed in the paralyzed muscles. Fibrillary tremors are present in some cases, absent in others. The bulbar muscles are occasionally affected. Hypertrophy or pseudo-hypertrophy does not develop. The paralysis is flaccid, the tendon reflexes are absent, and contractures occur. There are no sensory or mental disturbances, or impairment of the sphincters of bladder and rectum. The course is rather rapid, and death results from paralysis of the muscles of respiration. The disease is progressive, the progress of the atrophy is from the parts near the trunk to the peripheral portions of the limbs, and the atrophy is entirely symmetrical. Hoffmann believes that fibrillary tremors do not occur, mental development is not arrested, and the muscles of face, tongue, and throat are not paralyzed.

**Diagnosis.**—The greatest difficulty is in regard to muscular dystrophy, and so true is this that Senator says of his two cases that when the children were seen dressed and their mode of sitting, lying down, and standing up (climbing upon the lower limbs as in muscular dystrophy) was observed, the diagnosis of pseudo-hypertrophy seemed correct, but when the clothes were removed no hypertrophy was visible. Most important in the diagnosis were the presence of fibrillary tremors and diminution in the electrical response, and possibly a commencing reaction of degeneration in one case. The cases were without necropsy, and it seems questionable to which type they belonged. The type of Werdnig-Hoffmann shows a more rapid extension of the atrophy than occurs in progressive muscular dystrophy.

Congenital myatonia (congenital hypotonicity of the muscles) differs in being a congenital disorder, in the absence of heredity and family tendency, and in the much greater involvement of the trunk and portions of the limbs near the trunk at a very early age.

## AMYOTROPHIC LATERAL SCLEROSIS.

**History.**—If it be ever justifiable to employ the name of a man in the designation of a disease, it is especially so in regard to amyotrophic lateral sclerosis. Charcot described the affection so fully that Marie has likened his description to the origin of Minerva fully equipped from the head of Jupiter. The French are fond of calling the disorder Charcot's disease. The great French neurologist first directed attention to it in 1865, and in connection with Joffroy wrote on it in 1869, and with Gombault in 1871. In the years 1872 to 1874 he described the disease still more fully. Gombault made a further publication in 1877, and later with Debove, in 1879. Dejerine pointed out the relation of amyotrophic lateral sclerosis to the labio-glosso-laryngeal paralysis of Duchenne.

In this chapter amyotrophic lateral sclerosis is considered as distinct from progressive spinal muscular atrophy and from chronic or subacute poliomyelitis.

**Etiology.**—Amyotrophic lateral sclerosis is probably an abiotrophy. The individual is born with a certain potentiality of his motor system, but at middle age the strain becomes too great and degeneration begins.

There is very little to warrant the opinion that the disease is of toxic origin. It is possible that lead may be a cause, or at least it may cause a symptom complex very closely resembling that of this disease, as shown by S. A. K. Wilson. This author has studied four cases in which chronic lead poisoning began with double wrist drop, and in three of these the small muscles of the hand were not affected until late in the process, and in the other they escaped so long as the patient was under observation. In all four cases the atrophy was progressive, and was associated with some exaggeration of the tendon reflexes. Babinski's reflex may be found in this lead palsy associated with a certain degree of spasticity and with ankle clonus. The electrical reactions may be altered quantitatively but not qualitatively, and sensation may be intact, although in one of Wilson's cases lancinating pains were present, and this occurrence in association with the Argyll-Robertson pupil led to a diagnosis at first of tabes. No necropsies have been obtained, and, therefore, it is not known whether lead may produce exactly the changes of amyotrophic lateral sclerosis.

It is not unreasonable to assume that lead poisoning may appear as a systemic disease of the nervous system, and there is even a possibility that it may assume the type of tabes dorsalis, as shown by a case reported by Redlich. The commencement of the paralysis in the extensors of the hands and fingers is unusual in amyotrophic lateral sclerosis, but does occur. Muscular pains are not uncommon in lead poisoning, and cramps sometimes are felt in amyotrophic lateral sclerosis. An Argyll-Robertson pupil is rare in the latter disease, but Wilson seems to have observed it three times.

The attempt to give causes for which there is no evidence is hardly a justifiable procedure. We have no reason to believe that traumatism, exposure to cold and wet, or sexual excesses play a role. The occurrence of some trauma shortly before the first symptoms are noticed, as in Naka's case, is no proof that these are caused by the trauma.

It is not probable that syphilis causes amyotrophic lateral sclerosis. Syphilitic disease of the cord may produce a clinical picture resembling it, in that there may be spasticity, weakness of the limbs, atrophy of the small muscles of the hands, exaggerated tendon reflexes and fibrillary tremors. Careful examination, however, will usually reveal other symptoms; thus there may be severe pain in different parts, or disease of the optic nerves; or the history may mention the sudden onset of a hemiplegia, perhaps transitory. The writer has studied such a case, in which many of the symptoms suggested amyotrophic lateral sclerosis.

**Frequency of Disease and Sex Involved.**—Raymond and Cestan reported, in 1905, 18 cases with necropsy in the service of the former at the Salpêtrière; 13 were in men and 5 in women. In Collins' cases, 55 were males and 49 females. Probst, in 1898, found that of 53 cases the number was evenly distributed between males and females. Heredity and occupation do not seem to exert any influence.

The writer has reported 8 cases of primary degeneration of the central motor tracts with necropsy; 2 of these belong to the type of primary lateral sclerosis, as the nerve cells of the anterior horns of the spinal cord escaped, and in neither of these cases was muscular atrophy present. Amyotrophic lateral sclerosis seems to be a rare disease, about as rare or possibly more so than syringomyelia, but not so infrequent as some observers believe.

**Pathology.**—Amyotrophic lateral sclerosis is a degeneration of the pyramidal tracts, of the nerve cells of the anterior horns of the spinal cord, of the anterior roots, intramedullary and extramedullary portions; of the peripheral nerves, of the muscles, and sometimes of the nerve cells and fibers in the motor cortex. The corpus callosum and the posterior longitudinal bundles may be affected. It is essentially a death of the motor system and muscles.

The fibers that stained by hematoxylin in the pyramidal tracts of the spinal cord in this disease were noticed by Charcot and Marie to be very fine. Anton also observed an unusual number of fine fibers in the pyramidal tracts, and a similar observation was made by the writer, although fibers of larger size were mingled with these small fibers. The latter in the sclerotic tracts suggest that atrophy as well as degeneration of nerve fibers plays a role, but the Marchi method indicates very clearly that degeneration of the myelin sheaths is very important. The central motor tracts develop from above downward, and the terminal portions being the last formed are the least resistant, and, therefore, degenerate first.

The degeneration of the pyramidal tracts is not so intense in amyotrophic lateral sclerosis as in the sclerosis resulting from a cerebral lesion. This is probably because one fiber after another is attacked in the process, and all do not begin to degenerate at the same time. There is some doubt whether the process in most cases is ascending in each nerve fiber attacked, and the mere cessation of the degeneration at a certain level is no proof of an ascending process. It is reasonable to believe that there is a gradual death of the portions of the central motor neurones most remote from the cells of origin, and that the extent of the degeneration varies in different cases, but that the whole portion that is affected in each fiber is diseased usually almost simultaneously, although the individual nerve fibers are attacked at different periods.

Hoche's findings would seem to indicate that the degeneration of the pyramidal tracts may be ascending, but this observation has not been matched, and we cannot conclude that the ascending degeneration has been demonstrated as the general rule.

Kojewnikoff was the first (1883) to trace degeneration of the pyramidal fibers from the lower end of the cord to the cerebral hemisphere. In the two hemispheres the "corps granuleux" (fatty granular cells) were in perfectly symmetrical areas, and in almost equal quantity on the two sides.

Charcot and Marie (1885) traced the degeneration to the motor cortex in two cases. Kojewnikoff (Koschewnikoff) in a second case found degenerated fibers throughout the central motor tracts, and did not depend merely on the presence of fatty granular cells. In Lennmalm's case the pyramidal tracts were said to be degenerated from the cerebral cortex throughout their extent. Lumbroso traced the degeneration to the cortex. Strümpell could not follow the degeneration of the pyramidal tract above the superior part of the inner capsule, and he was unable to detect pathological changes in the nerve cells of the motor cortex. Mott traced the degeneration by Marchi's method through the inner capsule and into the motor cortex. Hoche, by the same method, traced degenerated fibers into the central gyri. Rossi and Roussy, in 1907, found 18 cases on record in which the degeneration had been traced to the cortex.

As regards the degeneration of the pyramidal tracts the writer has been

able to trace degeneration of the tracts as high as the motor cortex in one case, as high as the internal capsule in 2 cases, as high as the cerebral peduncle in 2 cases, as high as the pons in 3 cases. The degeneration does not usually extend above the pons, and gradually disappears. The higher regions of the pyramidal tracts show a more normal manner of staining. In one case degeneration of the pyramidal tract was traced on one side as high as the pons, and on the other as high as the lower part of the internal capsule. The degeneration therefore is not always symmetrical on the two sides, although it usually is. The writer has observed great spasticity of the limbs when the degeneration of the pyramidal tracts was barely detectable. The weakness may be due entirely to implication of the central motor tract alone, but the writer has observed paralysis on one side greater when the degeneration of the pyramidal tract on the same side was less intense than that of the other side. A study of the nerve cells showed that the motor cells of the spinal cord on the side of the less affected pyramidal tract were the more diseased.

Marie has stated that the degeneration of the lateral columns involves a greater area than that occupied by the pyramidal tracts, as shown by secondary degeneration from cerebral lesions or by myelination of the cord. It is unquestionable that the degeneration does extend beyond the pyramidal tracts, and this alteration of the supplementary zone has been described in progressive spinal muscular atrophy by J. B. Charcot. As in this disease, the pyramidal tracts are not degenerated, it is evident that this supplementary zone is not a part of the pyramidal tract. Degeneration of this zone does not occur in every case of progressive spinal muscular atrophy.

Pilcz found degeneration by the Marchi method in the anterolateral columns much greater than in the pyramidal tracts. This would indicate that the fibers in the former were more recently affected, and the condition in this respect resembles the degenerative changes occurring in the spinal cord in cases of grave anæmia. There is, however, another explanation. The fibers of the pyramidal tracts extend a long distance, and when they are degenerated the change is seen through a great part of these tracts, and has the appearance of long-standing alteration; whereas in the anterolateral columns the fibers are much shorter, and many more fibers must degenerate to cause a long-standing alteration as intense as that of the pyramidal tracts, but there may be more fibers in a stage of acute degeneration.

The anterior horns may contain fewer fibers than normal; the cells of the anterior horns may be much affected, in some the nucleus may be displaced to the periphery, and the chromophilic elements entirely destroyed, except where they surround the nucleus, and the remainder of the cell may have a pale green appearance by the theonin stain. The pigment is sometimes much increased. In some cells the nucleus may disappear, in many the chromophilic elements are changed to fine granules. Many cells have no dendritic processes, or only imperfect ones; some of the cells are tumefied and rounded, some have altered cell bodies and normal dendritic processes, in some cells a faint, bluish tint along the periphery is all that is left of the chromophilic elements.

The bulbar changes are often pronounced. The nuclei of the hypoglossal nerves may be much degenerated or even disappear, so that the cells may be few in number, with chromatolysis and much pigmentation, and loss

of dendritic processes; or they may entirely disappear. The intramedullary fibers of the hypoglossus may be intensely degenerated. The tongue muscles may be greatly wasted, in association with the alteration of the hypoglossal nerve, and show excess of interstitial fatty tissue. The nucleus ambiguus, as well as the posterior nucleus of the vagus nerve, may be much degenerated, but as the cells of the former do not form a very sharply defined group, it is often difficult to determine a partial destruction of this nucleus. The fibers arising in this nucleus may be few in number. The soft palate may be wasted.

The motor nucleus of the trigeminal nerve has been found affected (Rossi and Roussy, and others), but may escape when other bulbar nuclei are diseased. The facial, the glossopharyngeal, and spinal accessory nerves, and their nuclei, may share in the alteration. The nuclei of the ocular muscles, according to Marie, are never affected in this disease, but Hoche has found degeneration of the oculomotor, trochlear, and abducent nerves, and this indicates that ophthalmoplegia might occur.

Bulbar symptoms, however, may exist without distinct alteration of the bulbar nuclei. The microscopic examination of a case reported by Dercum and the writer showed that the bulbar nuclei were for the most part intact, but the degeneration of the pyramidal tracts extended above these nuclei. It is possible that nerve fibers controlling these bulbar nuclei were affected, and this would seem to support Charcot's view of the process as primarily a degeneration of the pyramidal tracts. Oppenheim observed symptoms of disease of the ninth, tenth, and eleventh nerves in amyotrophic lateral sclerosis without changes in the nuclei of these nerves.

The bulbar nuclei, on the other hand, may be affected without implication of the pyramidal tracts. Duval and Raymond have reported a case in which the bulbar nuclei were affected and no trace of sclerosis of the motor tracts was seen. Raymond refers to a number of other cases in which the nuclei were affected but the motor tracts escaped. Miura asserts, however, that no case of progressive bulbar palsy without degeneration of the pyramidal tracts is to be found in the literature.

It occurred to the writer in 1899 that inasmuch as amyotrophic lateral sclerosis is a disease of the motor system, a case in which the cortex was degenerated could be employed in delineating the extent of the motor area in man. The process seemed almost like an experiment. This use of amyotrophic lateral sclerosis was original with the writer, and later was adopted by others. Intense degeneration of the precentral convolution was found and considerably less degeneration of the postcentral convolution, and therefore it was concluded that the latter is also a part of the motor cortical area. The postcentral convolution may escape. It is proper to state, however, that the tendency is to exclude the postcentral convolution from the motor region.

Degeneration of the corpus callosum has been found by Probst, Spiller, Rossi and Roussy, and Naka in the middle portion of this structure. In the writer's case it extended in less intensity to the knee.

The large cells of the motor cortex have been found diseased. Mott found that many of them had disappeared. Marinesco has found the giant cells of the motor cortex greatly diseased. Most of these cells had disappeared, and the few remaining were atrophied, and had undergone chromatolysis and displacement of the nucleus. Kojewnikoff noticed atrophy and pigmentation of these cells.

Some degeneration of the columns of Goll in the cervical and upper thoracic regions has been observed in a case of primary lateral sclerosis reported by Dejerine and Sottas. Sensation had been normal. Moeli observed degeneration of these columns in these regions in a case of amyotrophic lateral sclerosis.

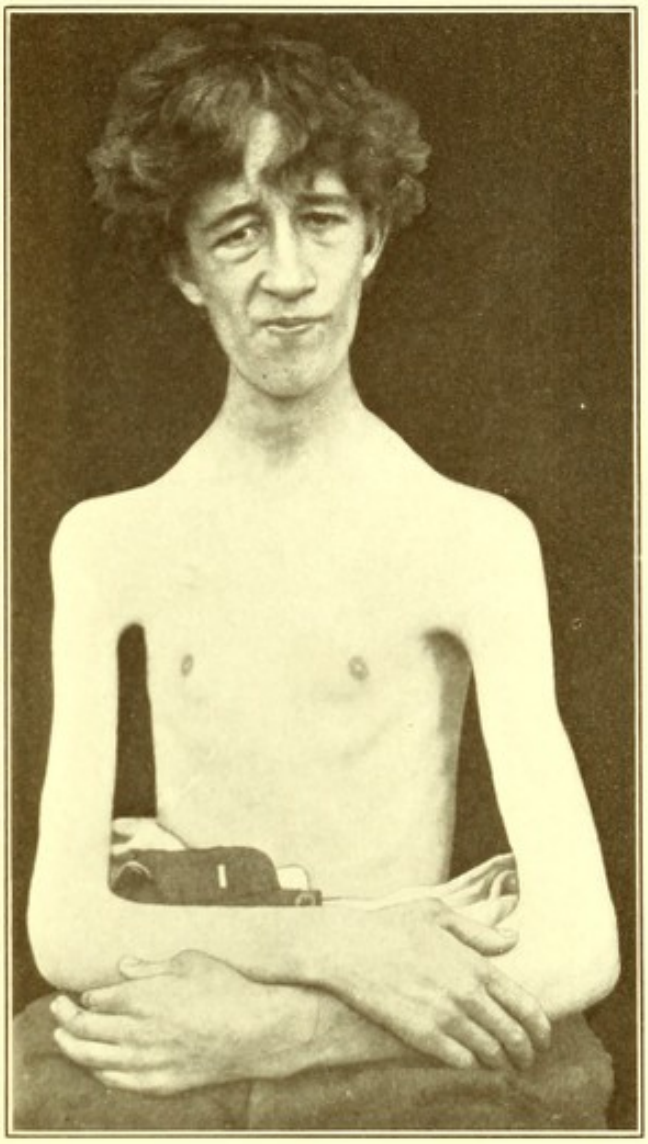
The columns of Goll are very liable to undergo a slight sclerosis in the lower cervical and upper thoracic regions in cases where no symptoms of this sclerosis have been present, and in some way it seems to be related to disease of the central motor tracts. Oppenheim, and Charcot and Marie have noticed sclerosis of these columns in amyotrophic lateral sclerosis. These cases must be distinguished from those in which the symptoms of posterior sclerosis were added to those of sclerosis of the lateral columns, a posterolateral sclerosis. Nonne has observed a distinct though not excessive rarefaction of fibers in the median portion of the posterior columns in chronic anterior poliomyelitis; it was most evident in the cervical region. The degeneration of the posterior columns in these cases of primary degeneration of the motor system is not tabetic in character, it is not systemic. The posterior roots are not degenerated, and the region affected, lower cervical and upper thoracic, is not that usually diseased in tabes. It was present in the case described by Dercum and the writer. In only two of Raymond and Cestan's cases was a slight sclerosis of the columns of Goll in the cervical region observed, and as these patients were sixty-one and sixty-eight years old respectively, the authors are inclined to attribute it to old age.

The muscles may be intensely atrophied, but even in many of these atrophied fibers the transverse striation may be distinct; in others the longitudinal striation may be more visible. The muscle fibers may or may not show fatty degeneration. In some of the fibers striation may not be clearly seen. The vessels may be much altered. Much fatty tissue may be found in some parts of the field. The nerve fibers between the muscular fibers may appear normal. The ulnar nerve has been found degenerated in one case (Spiller).

**Symptoms.**—Amyotrophic lateral sclerosis is a disease of middle life. Marie regards those cases (Erb, Seeligmuller) in which it is said to have begun in childhood as very doubtful, and he believes that some of them belong to the family spastic paralysis. Marie gives the commencement between the ages of thirty-five and fifty years; Dejerine, the second half of adult life; Oppenheim, middle life; Eichorst, between thirty-five and fifty years; Gowers, between twenty-five and forty-five years; Strümpell, between twenty-five and forty years. In Probst's statistics of 47 cases the disease began most frequently between thirty and fifty years, in 13 cases between fifty and sixty years, in 5 cases between sixty and seventy years, and in one case after seventy. Rossi and Roussy conclude that the beginning is usually between thirty and fifty, although frequently between fifty and sixty years; it is rare between sixty and seventy, and exceptional after seventy years. They have not been able to find any case recorded with a commencement after seventy years, except one case of Probst, but they report two cases from the service of Pierre Marie in which the commencement was at the seventy-first and seventy-third year.

Marie fixes the duration at eighteen months to two years, but death may occur in three to six months. Dejerine has observed a duration of nine years, and Florand of ten years. Marie questions whether these were truly

# PLATE I



Amyotrophic Lateral Sclerosis.

With bulbar symptoms and greater implication of the proximal part of the upper limbs, in a boy, aged nineteen years. (Spiller and Gittings.)

cases of amyotrophic lateral sclerosis. Collins selected the records of 94 typical cases, and added to these 10 of his own cases, but not all these were with necropsy. He found that in 100 selected cases, 30 patients were between thirty and forty, 29 between forty-five and fifty, 28 between fifty and sixty, and 2 under thirty. In 26 cases the average duration was a little more than two years. The briefest duration was five months, and the patient died from influenza, so that the case may be rejected so far as its duration was concerned. In 2 cases the disease seems to have terminated in six months. Records of 4 cases were found in which the disease lasted only nine months. A few cases lasted only about five years; the longest on record was ten years.

Amyotrophic lateral sclerosis may begin occasionally at an early age. The writer has seen, in association with Dr. Gittings, a boy, aged nineteen years, who had marked bulbar symptoms with signs of degeneration of the nerve cells of the anterior horns in the cervical and thoracic regions, and possibly slight degeneration of the pyramidal tracts. The course was rapidly fatal, the symptoms being present only about one year.

The symptoms were first manifested in two of the writer's cases in one lower extremity, although in one of these the upper limb of the same side may have been implicated at nearly the same time. The paralysis in these two cases became a hemiplegia and later a triplegia. In another case the paralysis seemed to begin as a hemiplegia. The statements of the patient or of the relatives must be taken in determining the part first affected, and there is, therefore, an element of doubt. The weakness and atrophy usually begin in the muscles of the hands, but sufficient cases are on record to show that they may commence in the feet. In a case reported by Dercum and the writer the weakness of the lower limbs preceded the bulbar symptoms about three years. In 81 cases collected by Collins, the upper extremities were first involved in 39 cases, the lower in 14 cases, the limbs of one side in the hemiplegic type, or all four limbs in 11 cases.

The writer collected reports of 11 cases (Dejerine, Pick, Vierordt, Leyden, Lennmalm, Mott, Senator, Probst (4)) in which the symptoms were confined to one side of the body, although both pyramidal tracts were affected in some of these cases. A case reported by C. S. Potts should be included in the list. Where the disease begins as a unilateral affection it does not remain as such, but sooner or later, and usually soon, the other side of the body becomes implicated. The shoulder muscles in rare instances may be affected first, and Dejerine and Thomas report two such cases, or the extensor muscles of the hand may be first affected.

Raymond and Cestan[1] from a study of their cases make four types of amyotrophic lateral sclerosis: (1) Ordinary spinal type; (2) labio-glossolaryngeal paralysis; (3) amyotrophic type; (4) spastic type.

1. **Ordinary Type.**—This corresponds to the classical description of Charcot with spastic paraplegia or tetraplegia, ankle clonus, later amyotrophy, fibrillary tremors, and disturbances in the electrical reactions. Babinski's reflex was not observed, which seems to be remarkable. Only three of their cases corresponded to this type, and in two of them the amyotrophy began in the lower limbs and later implicated the upper limbs and the muscles innervated from the medulla oblongata. In one case the amyotrophy began

[1] Raymond and Cestan, *Revue Neurologique*, 1905, p. 504.

in the hands. These cases lasted twenty-four, twenty-six, and twenty-six months respectively.

2. **Labio-glosso-laryngeal Type.**—This was observed five times, four times in women, and Raymond and Cestan are inclined to think it is more common in this sex. The affection began in the muscles of the lips, tongue, and larynx, with exaggeration of the masseter reflex. Soon the limbs became implicated with exaggeration of the reflexes and progressive amyotrophy of the upper limbs. One patient had in the beginning attacks of choking, probably from spasm of the glottis; another had spastic laughing and weeping, without impairment of intellect. The cord showed degeneration in the anterolateral columns of the cervical and thoracic regions by the Marchi method. The duration of these five cases was from fourteen to twenty-four months.

3. **Amyotrophic Type.**—The disease appears as progressive muscular atrophy. The spasticity is slight, and ankle clonus and Babinski's sign are not obtained, but the bony and tendon reflexes are exaggerated, sometimes only slightly. The slight spasticity makes the diagnosis somewhat doubtful. The duration ranged from eight months to five years. The diagnosis must be made from the Duchenne-Aran type of muscular atrophy and subacute anterior poliomyelitis, but the necropsies showed degeneration of the anterolateral columns. Nine of their cases were of this type. The disease of the peripheral motor segments is prominent in this type, but the implication of the central motor segments is indicated by some exaggeration of the tendon reflexes, although spasticity is slight or even absent. It resembles subacute anterior poliomyelitis very closely, and, indeed, several of the cases of this latter disease with necropsy that have been reported belong to the non-spastic type of amyotrophic lateral sclerosis, as pictured by Raymond and Cestan.

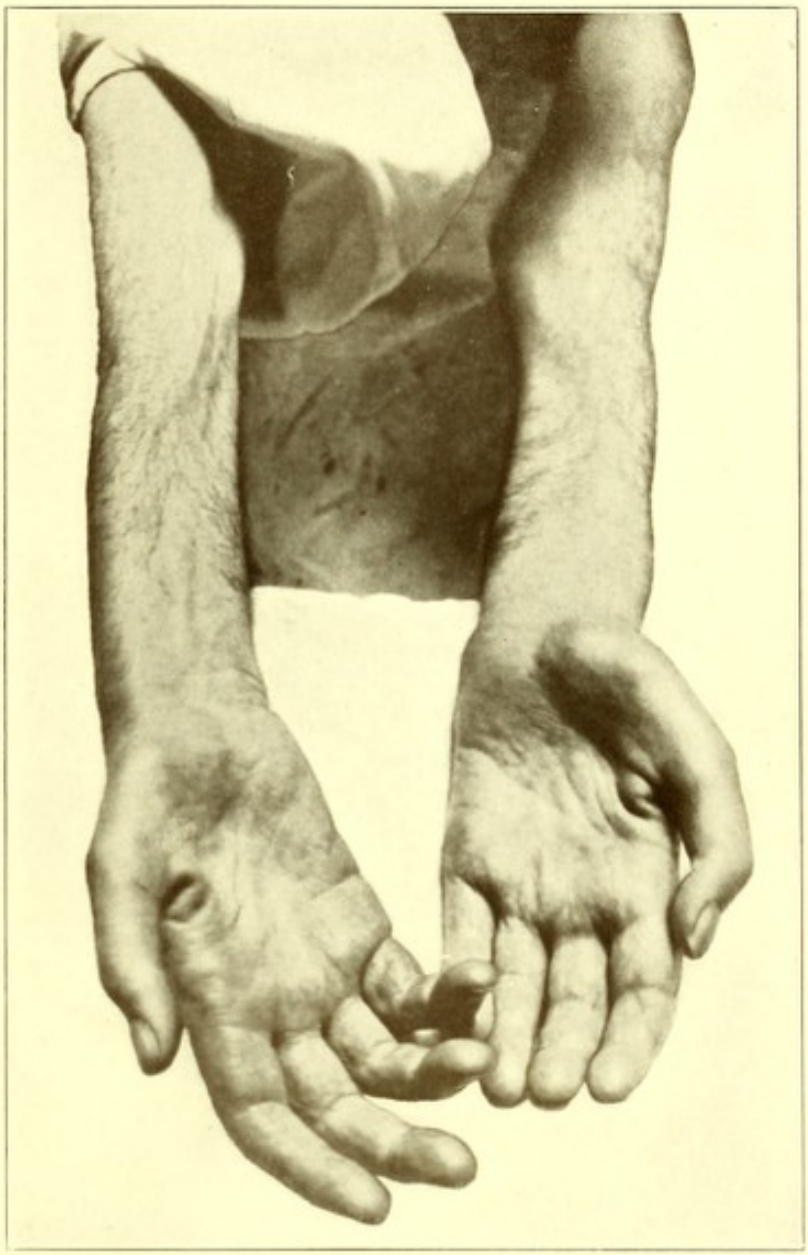
4. **Spastic Type.**—Only one of Raymond and Cestan's[1] cases belonged to this type. Spastic paraplegia was intense during one year, and was associated with ankle clonus and Babinski's sign, presenting the clinical picture of what is described by French writers as spastic dorsal tabes. Disseminated sclerosis, Erb's form of spinal syphilitic myelitis, and combined sclerosis in an early stage may present the same clinical picture. After a year progressive amyotrophy of the hands was observed, then the legs became affected, and still later the bulbar muscles.

Mills excludes Gowers' atonic variety in which both upper and lower limbs are the seat of wasting without spasm, except in such cases in which the lower motor segments degenerate first, especially if the degeneration be complete, as any degeneration of the upper motor segment may then fail to produce the usual symptoms. It may be said, however, that all such cases clinically belong to progressive spinal muscular atrophy or chronic poliomyelitis, and their true relation cannot be recognized without necropsy. Not only may the peripheral motor segments be alone affected, but sufficient cases are now on record to show that degeneration may be confined to the central motor segments.

The most important symptoms are weakness, spasticity, fibrillary tremors, exaggerated tendon reflexes, contractures, muscular atrophy, and bulbar disturbances. The weakness is an early manifestation, and is caused

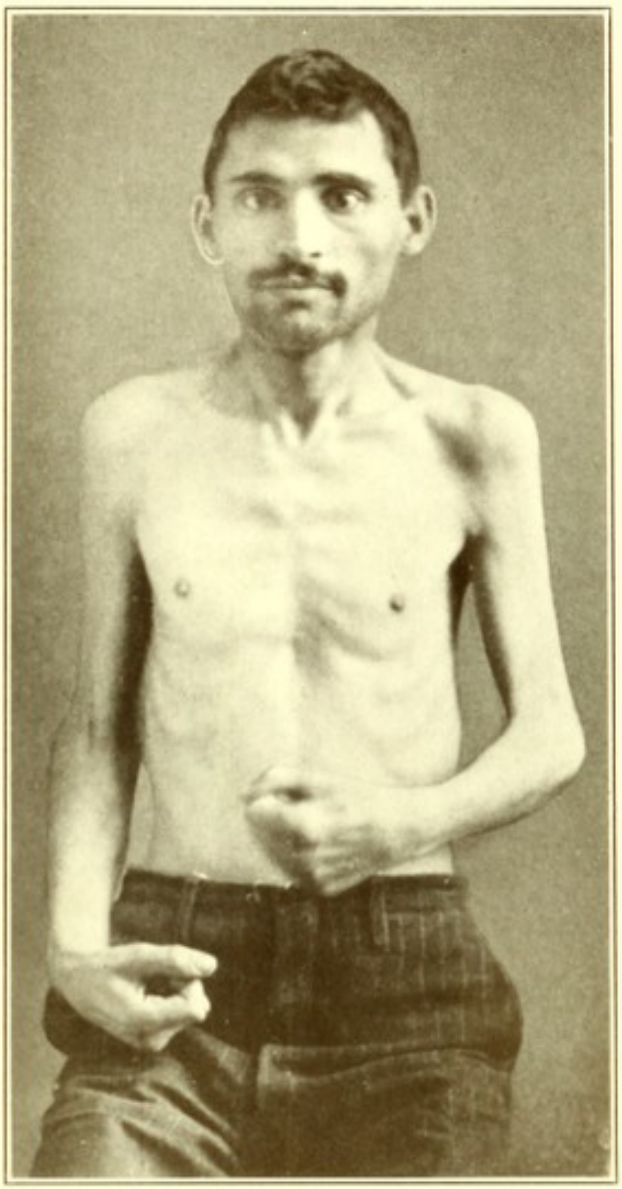
[1] Raymond and Cestan, *Revue Neurologique*, 1905, p. 504.

# PLATE II



Amyotrophic Lateral Sclerosis. Intense atrophy of the hands.

# PLATE III



Amyotrophic Lateral Sclerosis beginning in the Shoulder Girdle and Proximal Parts of Upper Limbs.

The utmost effort was made to elevate and flex the upper limbs, with little result. Flexion of the fingers was feeble, but far greater than movement in the rest of the upper limbs.

by the degeneration occurring in the central and peripheral motor tracts, as well as by the atrophy of the muscles. It is usually first noticed in the hands; the patient loses the finer movements of the fingers and can no longer approximate the thumb to the little finger with the same force, because of weakness in the muscles of the distribution of the median nerve. Sewing and writing become difficult. If he has learned any form of skilled movements, like playing on a musical instrument or typewriting, he has difficulty in performing these. Soon the wasting in the thenar and hypothenar eminences and in the interosseous spaces becomes pronounced, the weakness of the hand muscles progresses, and causes the simian hand, so called because of the loss of the thenar eminence. The extension to the forearm is gradual, and yet likely to occur early, while the arm and shoulder muscles usually are not implicated until considerably later. The weakness and atrophy increase *pari passu*, but the former is not dependent alone on the latter, as degeneration of the pyramidal tracts must cause weakness independent of any atrophy of the muscles.

The lower limbs are more or less spastic from the beginning, and weakness of them gradually develops. They do not, as a rule, present much atrophy until the disease has attained a considerable development; this is because the pyramidal tracts are early affected, while the cells of the anterior horns in the lumbar region may escape until late. Cases in which the symptoms begin in the lower extremities, and the upper extremities are not implicated until later, occur much more frequently than those in which the shoulder muscles are first affected. The muscles of the neck and trunk, as well as those of the face, may become greatly wasted, and the patient may be unable to hold the head erect.

The tendon reflexes, the biceps and triceps, von Bechterew's scapula reflex, the patellar and Achilles tendon reflexes are early exaggerated, and those of the lower limbs usually as much as those of the upper limbs. Clonus is common, both ankle and patellar clonus. It may even be seen in the upper limbs, as wrist, biceps or triceps clonus; but it is much less likely to occur in the upper than in the lower limbs. In ten cases reported by Raymond and Cestan, in which the Babinski reflex was searched for, it was found only once, and that in an extremely spastic case. It is hard to understand why this sign should have been absent in the other nine cases, as extreme spasticity is not necessary for its production when the central motor tract is affected. The masseter reflex often remains normal when the tendon reflexes of the limbs are exaggerated, but if the disease be intense, or extend far upward in the pyramidal tracts, the chin-jerk may be intensified as well as the other reflexes.

Beevor was the first to observe, in 1881, spontaneous clonus of the lower jaw. The sign, according to Beevor, shows that the sclerosis affects the pyramidal tract as high as the level of the fibers going to the motor nucleus of the fifth cranial nerve. He never observed clonus of the lower jaw in any other disease, except once in a case of hemiplegia. There may be some rigidity of the lower jaw with jaw clonus preceding the wasting of the bulbar muscles in the bulbar form of amyotrophic lateral sclerosis, and this may be absent in the true bulbar palsy.

In the case reported by the writer in which degeneration was traced to the cortex, there was some loss of power in the orbicularis palpebrarum of the left side, preventing the patient from closing the left eye tightly. The

case is like one reported by Marinesco in the implication of the upper part of the face; and this is remarkable, as in bulbar palsy the upper part of the face usually escapes. The patient had a low-grade neuroretinitis and pronounced perivasculitis. The presence of this ocular condition does not exclude amyotrophic lateral sclerosis.

Fibrillary tremors occur usually in the atrophying muscles and may be intense, but may not be always present when the patient is under observation, and may disappear when the atrophy has attained an intense degree. They usually persist, however, a long time, and may precede the atrophy. They are indicative of degenerative changes going on in the cells of the anterior horns of the spinal cord, and have been described by the not very appropriate name of "fibrillary chorea."

The upper and lower limbs are usually spastic, but if the atrophy attains a considerable development the spasticity gradually diminishes and may disappear. It is not unusual to observe the spasticity greater in the lower than in the upper limbs, as atrophy is less likely to develop in the former. Contractures of the intensity seen in hemiplegia are rare, but there may be more or less fixity of the limbs from the increased tonicity of the muscles, and if this continue sufficiently long, contraction will occur and cause permanent fixity even after the spasticity has disappeared. Contractures are not very pronounced, but when present are usually of the flexor muscles. They may be so intense that the feet may be in the position of talipes equino-varus. In the upper limbs the arm may be held closely to the chest, the forearm flexed on the arm almost at a right angle, the hand flexed at a right angle to the forearm, the thumb turned inward and the fingers flexed. Involuntary jerking of the fingers is common, and may be related to fibrillary contractions. The wasting of the muscles and the gradual diminution in their tonicity diminish the contractures as the case progresses.

Marie has observed intention tremor of less intensity than in disseminated sclerosis. If such occur it probably is caused by muscular weakness and the inability therefrom to coördinate properly the movements.

The reaction of degeneration is not usually very pronounced, as normal muscle fibers are mingled intimately with degenerated fibers, and the former mask the reaction presented by the latter. A change in the formula as well as a modal change may occur, but much more common is diminished response.

Retention of urine and loss of control over the bowels have been observed, and are probably the result of lesion of the pyramidal tracts.

A certain degree of mental failure may be observed occasionally, and the patients may take little notice of what is going on about them. Some writers have recognized mental deterioration as a part of the symptom-complex. Marie says that mentality is always more or less affected, intelligence is enfeebled, emotionality is increased, and tendency to laugh or weep is pronounced. Sometimes a demented condition exists. The patient is moody, and intellectually and morally approaches the attitude of a child; at times the condition is like that of neurasthenia. These conditions are in association with bulbar symptoms.

Suicidal impulses, such as occurred in Collins' case, are not necessarily a sign of mental failure. One cannot wonder that the hopelessness of the disease should so impress the patient as to make death preferable to a miserable and prolonged existence.

*Pain* is sometimes observed in one of the writer's cases; the man had complained of pain in both lower limbs below the knees five or six months previously. These were probably the pains of fatigue, as the patient was weak in the lower limbs and became very tired by night. Muscles undergoing atrophy and partially paralyzed may readily cause pain if exercised beyond the limited power remaining in them. Naka observed pain in the beginning of the disease in his case, and no cause for it was found. He could not attribute it to the spasms, as Oppenheim has done, as his patient did not have spasms. Beevor also speaks of the occurrence of pain. Sensory disturbances of the objective form (anæsthesia, hypoæsthesia, analgesia, hypoalgesia) do not belong to the ordinary clinical picture, but pain on pressure has been observed.

It seems reasonable to attribute these painful phenomena to an associate neuritis, especially as in the case recorded by Claude and Lejonne the patient was somewhat alcoholic. Much caution is needed in making a diagnosis of amyotrophic lateral sclerosis when sensory symptoms are pronounced, as in most such cases some complication is probable, or the diagnosis is incorrect.

The disease may begin as bulbar palsy, and when this type occurs death is usually too rapid to permit of much implication of the limbs. Or the bulbar implication may develop within a few months or a year or two after the limbs are affected, as in one of the writer's cases in which dysphagia and dribbling of saliva developed almost at the same time as the loss of power in the upper and lower limbs. The symptoms in this case lasted about one year when death occurred. The man became almost completely paralyzed and greatly wasted.

The muscles about the mouth become affected, the patient can no longer properly pucker up his lips as in whistling or in blowing out a candle, the lip sounds—l, b, t, r, s, k, g, ch, i, d, n—become imperfect, and fibrillary tremors become very distinct in the face and tongue. The upper part of the face, as in progressive bulbar palsy, usually is not affected. The tongue becomes deeply furrowed, and the atrophy and weakness may be so great that the patient may be unable to protrude it beyond the lips. The soft palate may be paralyzed and atrophied, so that fluids regurgitate through the nose when the attempt is made to swallow; the voice is nasal and very indistinct, and the soft palate no longer rises, or rises imperfectly when the attempt is made to say "*a*." The pharyngeal muscles also may be affected and swallowing becomes very difficult, and the pharyngeal reflex may disappear. The face may be much atrophied, death not occurring rapidly enough to prevent the atrophy, even when the bulbar nuclei are involved. Even when the lower part of the face is intensely implicated the eyelids usually can be held firmly closed. The vocal cords may be paralyzed.

Bulbar symptoms do not necessarily indicate a speedy fatal termination. In Raymond and Cestan's cases, in which the bulbar disturbances were the first symptoms, the duration of the disease was not shorter than in those in which the first symptoms were spinal, and bulbar disturbances developed later. Claude and Lejonne emphasize that the existence of symptoms in the domain of the pneumogastric nerve does not necessarily mean speedy death, as such symptoms had been present in their case one year, and were manifested by rapid action of the heart after slight exertion, irregular

pulse while the patient was at rest, lowered arterial tension, pallor, and syncope.

The muscles of the face, outside of those of the lips, usually escape. The muscles of mastication are not usually among the first affected in bulbar palsy, but they may be to such an extent that the patient is unable to chew or to move the jaw from side to side. Food may enter the larynx and cause attacks of coughing or aspiration pneumonia. Bulbar symptoms occurred first in 21 of the cases collected by Collins. In upward of 50 per cent. of all the cases, bulbar symptoms appeared within the first year. Marie is inclined to believe that labio-glosso-laryngeal paralysis is always a part of amyotrophic lateral sclerosis. The investigations of Charcot and Marie, and Dejerine seem to show that the pyramidal tract is always affected in bulbar palsy.

**Diagnosis.**—What has been said in the differential diagnosis of progressive spinal muscular atrophy applies equally well to amyotrophic lateral sclerosis, when it is remembered that in the latter disease we have the symptoms of progressive spinal muscular atrophy with the additional symptoms of spasticity, exaggerated reflexes, and Babinski's sign.

**Prognosis.**—This is exceedingly grave. The disease is necessarily fatal, and may terminate within one year, and usually does so within three or four years. We have no means to arrest its course, and the end usually comes through the implication of vital centres in the bulbar palsy. The preservation of consciousness and the full recognition by the patient of the seriousness of his affection make the disorder greatly to be dreaded, for there is no merciful blunting of the intelligence.

**Treatment.**—The section on treatment in the primary degenerations of the motor system need not be very extensive, as the results are not brilliant. The administration of strychnine may be of service. Overexertion is to be avoided, as in this way too rapid exhaustion of the nervous system may be prevented. Massage, passive movements, and electricity are of doubtful value when the atrophy and weakness are progressive, although when the course is very slow or has been arrested they may be of great service.

## LATERAL SCLEROSIS (SPASTIC TABES, SPASTIC SPINAL PARALYSIS, SPASTIC DORSAL TABES).

In a paper written in 1903, Erb remarks that he did not suppose when he first described spastic spinal paralysis in 1875 he would be compelled twenty-eight years later to fight for its recognition. His views expressed in 1875 received the support of Charcot and others, but met with the skepticism of prominent neurologists, especially of the Berlin school. The condition was regarded merely as a symptom complex caused by various disorders of the spinal cord and brain; as a condition without a definite pathology. Necropsies did not confirm the views of Erb and Charcot that primary degeneration of the pyramidal tracts was the underlying lesion, and while degeneration of these tracts was found, it was usually secondary to hydrocephalus, multiple sclerosis, tumor, syringomyelia, or symmetrical cerebral lesions; or was part of postero-lateral sclerosis or of amyotrophic lateral sclerosis.

Erb, in his paper of 1903, reviewed the opinions of different writers as

regards the existence of primary lateral sclerosis. Leyden and Goldscheider (1897) do not accept it as more than a symptom complex produced by many causes; Schultze (1898) accepts it as an entity; Redlich (1900) is very conservative and regards the matter as undecided; Schmaus (1901) acknowledges it as an entity, but of rare occurrence; Oppenheim (1902) does likewise; among French neurologists, Brissaud (1895) accepts it as a well-defined affection, while Gerest (1898) and Raymond do not; Grasset and Guilbert recognize a primary lateral sclerosis, but as an interstitial vascular disorder; Dejerine (1901) is cautious and does not accept it as a morbid entity, although he has had a typical case with necropsy, but with Thomas (1902) describes primary lateral sclerosis briefly under primary sclerosis.

Leyden and Goldscheider acknowledge the possibility of primary degeneration confined to the pyramidal tracts, but the clinical diagnosis of this condition they believe is impossible, and therein is the important factor. It is impossible, they think, to speak of an etiology, pathogenesis, and therapy of spastic spinal paralysis. Strümpell does not accept this view, but regards as the important factors the clinical and anatomical findings; when these are established the diagnosis will be easy. As justifying the recognition of spastic spinal paralysis, Erb accepts 11 cases; they are those of Morgan and Dreschfeld, Minkowski, Strümpell (2 cases), Dejerine and Sottas, Donaggio, Friedmann, Bischoff (2 cases), Demock, and Kühn.

**Pathology.**—This, if it is to be accepted as an entity, consists of primary degeneration of the pyramidal tracts and nothing else, and this degeneration may stop at any level or extend to the cortex. If we accept all the 11 cases that Erb includes, we must be a little more liberal in our interpretation. Thus, in Morgan and Dreschfeld's case, atrophy of some cells of the anterior horns in the thoracic and lumbar regions was found, but Erb accepts it because this degeneration seemed unimportant and amyotrophy was not present during life. Grainger Stewart stated that he examined the sections of this case and found the changes in the gray matter distinct. At the time this case was reported Nissl's method was not in vogue, and could it have been employed it is not improbable that more evidence might have been obtained justifying the classification of the case as one of amyotrophic lateral sclerosis, as has indeed been done by some authors.

Implication of the direct cerebellar tracts, as in Minkowski's case, may be accepted as possibly causing no clinical signs, and therefore need not exclude a case from the designation of spastic spinal paralysis. Whether the same may be said of a slight degeneration of Goll's columns in the upper part of the cervical region, as in one of Strümpell's cases and in others, is open to question; but it occurs in certain cases of amyotrophic lateral sclerosis, in which no clinical evidence of its presence is observed. Strümpell, however, made the diagnosis in his case as combined system disease. Where syphilitic meningomyelitis exists the degeneration of the pyramidal tracts cannot be regarded as primary, and if a nuclear stain be not employed the cellular infiltration of the pia and cord may be overlooked.

**Symptoms.**—These, as originally described by Erb, are motor weakness, spasticity, and exaggerated reflexes; to these should be added the Babinski reflex. No other symptoms should be present, there should not be sensory disturbance, atrophy, sphincteric disorder, tumor, ataxia, or implication of the cranial nerves. The symptoms may persist many years, and Erb

says he knows cases in which they have lasted nineteen to twenty-seven years. They may develop very slowly, during twenty-three years in the case of Dejerine and Sottas.

The weakness usually affects one lower limb some time before the other lower limb becomes implicated, but it is often much less intense than the spasticity. It is at first a sense of fatigue after slight exertion, and spasticity is associated with the paresis. The patient stubs his toe in walking because of the spastic condition of the muscles of the lower limbs, and the manner in which the soles of the shoes are worn is often very diagnostic, the sole at the point of the shoe may be completely worn away. The Achilles tendon and patellar tendon reflexes are much exaggerated, so that ankle clonus and patellar clonus may be persistent, or the whole lower limb may be thrown into clonic movement, a condition inappropriately called spinal epilepsy by French writers. The lower limbs are rigid, the thighs adducted, so that walking is interfered with, and the scissors gait, so called because of the tendency for one limb to be drawn in front of the other, is produced. The spasticity may be so pronounced that marked spasm is caused by passive movement. If the leg be extended upon the thigh, passively, a sudden spasm may jerk the leg to full extension, the "clasp-knife" reaction.

The upper limbs may escape entirely, but if they be affected the symptoms may be less marked, though of the same type as those in the lower limbs.

In later stages of the disease the patient becomes bedridden and unable to stand. Death results from some intercurrent disease.

Slight flexion at the knees Strümpell believes will be found in all cases of spastic spinal paralysis beginning in early youth. This is usually attributed to spastic contracture in the affected muscles, but Strümpell believes it is caused by differences in growth of certain muscle groups. The flexors are more affected than the extensors, therefore their growth is arrested when the lesion develops early in life. Strümpell thinks it is very doubtful whether hypertonia of the muscles can occur without exaggerated tendon reflexes; where the latter are not seen the hypertonia may obscure the exaggeration. Exaggeration of the tendon reflexes, the writer believes, may occur without hypertonia.

Strümpell distinguishes the following forms of spastic spinal paralysis:

1. A form certainly endogenous, characterized by family and hereditary appearance, males especially affected, commencing between the twentieth and thirtieth years, progressing very slowly, lasting a long time (twenty to thirty years or longer). The symptoms are gradually increasing rigidity and hypertonia of the muscles of the lower limbs, exaggeration of tendon reflexes, etc. Gait is spastic and for a long time without marked paresis, but paresis occurs later, especially in the flexors and muscles of the lower part of the lower limbs. The upper limbs remain intact until the end, likewise the bulbar and cerebral functions.

2. Infantile spastic spinal paralysis. This commences in childhood, at times as early as the third to sixth year, although it may begin later. This type is related to the cases of congenital abnormal development. A careful history must be obtained in order to decide whether the condition be congenital agenesia, or progressive degeneration beginning later. Inquiry must be made regarding injuries at birth, healed encephalitis, etc. The symptoms are those of spastic paralysis of adults, but modified by certain conditions

of the developing organism. The upper limbs are often unimplicated, or at least only slightly so, but they may be distinctly affected. Arrest in cerebral development is often present, shown by mental deficiency. Such cases are not very rare.

3. A third type is also endogenous in origin. It begins in late life, progresses comparatively rapidly, implicates the entire pyramidal tracts, so that the upper limbs and bulbar muscles are affected. The symptoms are general spastic rigidity, peculiar spasticity in bulbo-cerebral territories (glottis spasm, forced phonation, forced laughter, forced crying, involuntary movements of the facial muscles, etc.). The tendency to implication of the peripheral motor neurones is strong, shown by fibrillary twitchings, muscular atrophy, etc. These cases are closely related to amyotrophic lateral sclerosis. They usually are isolated, not occurring in families.

4. An exogenous type. The syphilitic spinal paralysis, with the exception of lathyrismus, is the only important form. The lesions are seldom confined to the pyramidal tracts, and the columns of Goll are usually partially affected. The clinical picture of spastic spinal paralysis is seldom uncomplicated.

5. A fifth form may occur in women after confinement and the puerperium. Strümpell has observed a number of cases in which spastic spinal paralysis developed following these conditions. No necropsies of cases of this form have been obtained.

**Diagnosis.**—This is often difficult so far as the exclusion of other processes is concerned, and among these may be mentioned spinal tumor without pain, multiple sclerosis, hysteria, latent caries of the spine, myelitis, secondary degeneration from cerebral lesions, and combined sclerosis in which the symptoms of posterior column disease are indistinct. Indeed, it is questionable whether the diagnosis of primary lateral sclerosis can be made with absolute certainty during the life of the patient.

*Tumor of the spinal cord* is occasionally difficult to distinguish. In it the symptoms are usually unilateral at first, bilateral later, and associated with severe shooting pains, but cases of spinal tumor occur in which the symptoms are bilateral from the beginning and pain is absent. In a number of cases the diagnosis between spinal tumor and primary lateral sclerosis seems impossible.

*Multiple sclerosis* usually presents symptoms that are very characteristic, but there is no denying the fact that occasionally it may cause exactly the symptom complex of lateral sclerosis, when the lesions are almost confined to the spinal cord. In such cases the differential diagnosis may be impossible.

*Hysteria* may cause a spastic paraplegia, but careful study will usually reveal some evidence of this neurosis. There may be some disturbance of sensation in segmental forms, some ocular signs; or the symptom complex may not be typically that of lateral sclerosis. An ankle clonus, if it occur, is seldom exactly the same as that of organic disease, although it may be found in grave hysteria. This statement is disputed by certain writers. If hysterical the gait will usually afford some clue to the neurosis, and the symptoms may disappear under suggestive treatment.

*Caries of the spine* may occur without kyphosis and without other symptoms than those of implication of the lateral columns by compression. The diagnosis, while not usually difficult, may be impossible.

*Myelitis* may be very hard to distinguish and the clinical picture may be exactly that of lateral sclerosis. It is often syphilitic in character, and cyto-diagnosis and sero-diagnosis promise much in such conditions.

*Secondary degeneration* from cerebral lesions is usually distinguishable by the presence of cerebral symptoms and the much greater implication of one side of the body. The onset is likely to be acute, with first one side affected in one attack and later the other side in a second attack. The paralysis is more intense, as in lateral sclerosis the spasticity is usually more pronounced than the weakness.

*Combined sclerosis* usually will cause at least slight indication of involvement of the posterior columns; there may be some indication of ataxia.

*Spastic spinal paralysis* must also be distinguished from congenital arrest in the development of the pyramidal tracts or degeneration of these tracts from injury of the brain, with epilepsy, mental defect, etc. Similar cases may be caused by premature birth, intra-uterine cerebral disease, hydrocephalus, etc. The upper limbs may be more affected than they are in the typical spastic spinal paralysis, or cranial nerve implication may be detected.

**Treatment.**—Division of a certain number of the posterior lumbar roots may prove to be of benefit in lessening the spasticity, as may also injection of the nerves supplying the more spastic muscles with alcohol (Schwab and Allison). These forms of treatment are too recent to permit a judgment as to their value. It is possible that massage and electricity directed only to the less spastic muscles may bring about a more even degree of tonicity in all the muscles. Special forms of movements may be of service. All such attempts will prove useless if the disease be progressive.

## HEREDITARY SPASTIC SPINAL PARALYSIS.

**History.**—Recognition of this has been accomplished by the writings of Strümpell, Bernhardt, Newmark, Krafft-Ebing, and others. Strümpell, in the early eighties, recognized the peculiar clinical features, and reported the first necropsies with microscopic examination. Newmark has enriched our knowledge in showing that while the process is a combined sclerosis, the degeneration of the pyramidal tracts in a mild case may be insignificant, whereas that of the columns of Goll may be pronounced.

**Etiology.**—Syphilis, trauma, etc., seem to have no strictly causal relation, although trauma may act as an exciting cause in persons predisposed. The second of Newmark's cases with necropsy seems to show that agenesia of the pyramidal tracts may exist, so that these tracts from the beginning are imperfectly developed, and if slight degeneration occur it would be likely to produce pronounced symptoms. This hypothesis would explain those mild cases where the only sign of the disease is exaggeration of the tendon reflexes, as in two members of Newmark's O'Connor family.

There is much to support the view that the spastic spinal paralysis of family type is an abiotrophy, especially the commencement in several members of the same family at nearly the same age. The motor fibers have limited potentiality, and sooner or later degenerate under the stress of life. Jendrássik believed blood relationship of the parents to be important, but in a number of cases the relationship did not exist.

Strümpell thinks the male sex is more predisposed, and that when the males are affected in a family the females always escape, but this latter statement may be challenged. The difference between the sexes as regards frequency is not very striking. The disease is both familial and hereditary. Several observations show, especially Bernhardt's, that in the same family other nervous diseases occur in addition to spastic paralysis. The heredity is not always in exactly the same type, some neurosis or some organic nervous disease of different character may have been present in an ancestor, and yet the cases in which exactly the same form of paralysis has been observed in parent and child, or even grandparent, are sufficiently numerous to show that the heredity may be direct.

**Pathology.**—The findings are degeneration of the pyramidal tracts extending throughout the cord, most intense in the lumbar and lower thoracic regions, and becoming indistinct in the upper thoracic region (Newmark), or extending in lessened intensity to the pyramidal decussation (Strümpell). The degeneration of the crossed pyramidal tracts in a mild case may be found only in the lower lumbar region, and may be very slight even there, with a little greater involvement of one side of the cord than of the other (Newmark). The direct pyramidal tracts were probably degenerated in one of Strümpell's cases (Gaum).

The direct cerebellar tracts may be degenerated, but not intensely (Strümpell), or may be intact (Newmark). This degeneration may be followed above the pyramidal decussation into the medulla oblongata. The columns of Goll are degenerated, and more in the upper regions of the spinal cord than in the lower. Whereas, Strümpell has found the degeneration of the columns of Goll less intense than that of the crossed pyramidal tracts, Newmark has observed the reverse. The degeneration has been traced to the nuclei of these columns in the medulla oblongata. Newmark found in one of his two cases the net-like condition in the posterior columns, due to degeneration and dropping out of fibers, such as is seen in cases of severe anæmia, and he suggests the possibility of an exogenous factor, such as an acute febrile disease, in addition to the endogenous factor.

Strümpell has maintained that the degeneration of Goll's columns is secondary. If these columns degenerate first and the pyramidal tracts much later, real spasticity could not occur, as shown in Friedreich's ataxia, but Newmark believes, and apparently with reason, that his own second case with necropsy proves that a previous degeneration of Goll's columns does not prevent the spasticity resulting from the degeneration of the pyramidal tracts, as the degeneration of these tracts was only in the lower lumbar region and very slight, while that of Goll's columns was pronounced. Although the degeneration of the last-mentioned columns seems to have occurred in every case with necropsy, in none has it produced recognizable symptoms. The lesions are those of a combined sclerosis. The anterior and posterior roots, and the nerve cells of the anterior horns have not been found affected.

**Symptoms.**—Spastic paraplegia occurs in children or adults of the same family and may be observed in several generations of the same family, sometimes commencing at an earlier age in the later generations. The symptoms, at first with little or no weakness, are spasticity, exaggerated reflexes, usually only in the lower limbs, Babinski's reflex, and paralysis as a late manifestation. Contractures of the feet may be very pronounced, but sensory symptoms are not present; although in Strümpell's case Polster

tactile sensation was a trifle less acute in the legs than in the thighs, and temperature sensation was not entirely normal in the legs, although it was so in the thighs. This disturbance could not be attributed to vasomotor changes, as these as well as trophic symptoms were absent. Newmark would not exclude cases in which optic atrophy, feeble mentality, muscular atrophy, bulbar symptoms, bone lesions, and cataract occur. Such cases may not be pure examples of spastic paraplegia, but they are manifestations of abiotrophy. The symptoms usually develop nearly at the same period in the same family, but not always, and the periods vary in different families. In some families the disease first shows itself in the first years of life; in others, in early adult age; in others, still later. In Achard and Fresson's cases the symptoms in one member of the family began at the age of one year; in the other member at the age of sixteen months. Three members of one family in Hochhaus' cases became affected in the second year. Erb has observed the disease in two sisters at the fourth year; Souques, in a girl at three years, in her brother at five years. In one family Newmark found the ages at which the symptoms began fourteen, seven, nine, sixteen, eight, and eight years respectively. Krafft-Ebing observed the commencement at about twenty-seven, twenty-four, and twenty-two years respectively in members of the same family. Bernhardt's cases began in the commencement of the thirtieth decade; Strümpell's cases in two brothers in the third and sixth decades.

The symptoms may reach their height within a short time, especially after fever or injury, and remain at a certain stage a long time or permanently. In mild cases some improvement is possible. In some cases slow progression begins again after a period of arrest; in others the symptoms progress slowly from the time they first become manifest.

Cases with mental symptoms, implication of cranial nerves, unless possibly strabismus, may be regarded as unimportant, history of traumatism at birth, symptoms existing from birth, epileptic attacks, disturbances of sensation or muscular atrophy, can hardly be regarded as typical of the spinal form of spastic family paralysis. The family form of spastic paraplegia, probably being the result of degeneration of the distal portions of the pyramidal tracts from some imperfection of development, differs from the cases of Little's disease resulting from premature birth, in that in the latter the pyramidal tracts are arrested in their development, but are capable of further development even many years after birth, and probably do not degenerate. In the form occurring in childhood, the pyramidal tracts probably degenerate at their distal ends early, while in Strümpell's type the resistance of these tracts seems to be greater, and disturbance of function does not occur until adult life.

In some families the cerebral type seems to occur in certain members, and the spinal type in others (Bernhardt, Pribram, Melotti, and Cantalemessa). This would seem to indicate that there may be a close relation between these two types.

In a family studied by the writer, a father and his second son showed clearly the typical symptoms of the disease, and a younger son showed indications of it. Only the lower limbs were affected. The disease seems in the latest generation to be commencing at an earlier period. It has been almost as common in this family in the female sex as in the male. In late years the disease is said to have commenced before or about the fifth year of life, and

it has always presented the same symptoms. A young member of the family manifested the first symptoms about the age of eighteen months. The disease in this family was traced back five generations. This family is doubtless the same as that reported at an earlier date by Bayley, although the writer was not aware of this fact at the time of his report, and gladly gives Bayley credit for his earlier publication.

Dejerine and Thomas say the affection extends later to the upper limbs and eyes and to the muscles of speech, and yet in most cases these portions of the body, except for exaggeration of the tendon reflexes, escape.

In late stages the lower limbs may be almost completely paralyzed; Strümpell says it is characteristic of the hereditary spastic paralysis to have great exaggeration of tendon reflexes precede many years' true paralysis. It is uncertain whether reflex exaggeration and weakness result from disease of different fibers or from different degrees of degeneration of the same fibers. Strümpell has not seen involvement of the upper limbs beyond exaggerated reflexes. The condition must be distinguished from the spastic cerebral paralysis of childhood, not always with defect of mentality. This is an agenesia and may be familial in type and has an etiological relation to the spinal form. He gives no means of differentiation.

**Diagnosis.**—This must be made from cerebral paralysis, Little's disease, myelitis, Pott's disease, lateral sclerosis, family form of disseminated sclerosis, Friedreich's ataxia, and cerebellar hereditary ataxia. The difficulties are those mentioned for primary lateral sclerosis. Strümpell acknowledges that he, like Charcot, diagnosed primary sclerosis in a case in which the necropsy showed multiple sclerosis.

In some cases bulbar and cerebral disturbances existed in addition to the spastic paraplegia (Bernhardt), suggesting multiple sclerosis; in others premature birth, strabismus, slow acquirement of speech, feeble intelligence suggested cerebral origin. Family spastic paraplegia may be cerebral when associated with such symptoms, and Dejerine and Thomas would regard it merely as a symptom complex common to family disease of different character and situation. It seems that cases in which the upper limbs are contractured, intention tremor, scanning speech, nystagmus, atrophy of the optic disks, strabismus, etc., are present, should be accepted with reservation. They do not belong to the typical form as described by Strümpell and others, and may be examples of multiple sclerosis. Disseminated sclerosis, it is to be remembered, has been known to occur in two members of the same family.

## UNILATERAL ASCENDING AND UNILATERAL DESCENDING PARALYSIS.

Attention was first called to these forms of paralysis by Charles K. Mills. In many cases it is a symptom complex instead of a well-defined disease, just as bilateral spastic paraplegia is often a symptom complex, but may be the clinical expression of primary degeneration of the pyramidal tracts. The question that now interests us is, Does unilateral primary degeneration of the pyramidal tract occur? Some years ago the writer searched for all the cases of the unilateral form of amyotrophic lateral sclerosis in the literature, and found ten; to these should be added a case reported by Potts, one reported by the

writer, and one by Mills. The unilateral type of amyotrophic lateral sclerosis is only of short duration, and bilateral implication of the spinal cord occurs within a short time. The thirteen cases are those of Dejerine, Pick, Vierordt, Leyden, Lennmalm, Mott, Senator, Probst (3, possibly 4), Potts, Spiller, and Mills. Of these, only 5 were of the ascending type, the weakness commencing in one lower limb and later extending to the upper limb of the same side. These were the cases of Vierordt, Mott, Probst, Potts, and Spiller. The symptoms may begin with bulbar palsy (Dejerine, Leyden, Lennmalm, Probst (2 cases) ). The upper limb of one side may be affected before the lower (Pick, Leyden, Probst). In some of the cases the limbs of one side seem to have been affected simultaneously or nearly so (Dejerine, Lennmalm, Senator, Probst (2 cases), Mills). In those cases in which a necropsy was obtained the pyramidal tract of each side was degenerated. It is yet to be shown whether amyotrophic lateral sclerosis may ever remain confined to one side; at present it seems improbable from the reported cases.

A case of primary lateral sclerosis, reported by Mills and Spiller, would seem to indicate that the unilateral form of the disease without implication of the nerve cells of the anterior horns is possible. The patient, a male, aged sixty years, developed gradually hemiplegia on the right side, the lower extremity being more markedly, and for this reason probably earlier, affected than the upper; the case, therefore, at first belonged to the clinical type of unilateral progressive ascending paralysis. After several years the left lower extremity also became paralyzed, but not to the same extent as the right. The reflexes were all markedly exaggerated, the Babinski response being present. Sensory symptoms were absent. The lesions were primary degeneration of the motor tracts, much greater and of longer duration in the right crossed and left direct pyramidal tracts, with integrity of the nerve cells of the anterior horns.

It seems positive that unilateral ascending or descending paralysis may be caused by different conditions. It may, in the opinion of Mills, be the expression of a gradually developing cerebral lesion such as softening or tumor, of disseminated sclerosis, of a developing posterolateral sclerosis, of spinal syphilis, of tumor of the cord or of other form of compression of the cord, of paralysis agitans, of hysteria. The type has varied in different cases even when the degeneration has been believed to be confined to the pyramidal tract. In some spasticity has been present from an early period, in others it has been absent or slightly marked. The tendon reflexes have usually been exaggerated, but in a few instances have been diminished or slight. The wasting of the muscles has varied in degree. Optic atrophy and vesical disturbance have been observed. The explanation of these differences is to be sought chiefly in the greater or less implication of the cells of the anterior horns of the spinal cord.

Pathological evidence as yet does not warrant the assumption that degeneration of the crossed pyramidal tract may begin in the lower part of this tract, or in the upper part, gradually extend, so as to implicate the motor fibers for the other limb of the same side, and remain confined to one pyramidal tract.

## PROGRESSIVE NEURAL (NEUROTIC) MUSCULAR ATROPHY (PROGRESSIVE MUSCULAR ATROPHY OF THE CHARCOT-MARIE-TOOTH TYPE).

Charcot and Marie and Tooth described this form of atrophy independently in the same year (1886). The subject soon received attention from Joffroy, Hoffmann, B. Sachs in this country, and from many others, but evidently much has been included that does not properly belong here.

**Etiology.**—No satisfactory cause has been found, but the fact that it is likely to occur both as an hereditary and as a family disease shows that some defect in development is probably responsible for it. It is more likely to occur in males (five times in males to once in females, according to Sainton), but no explanation for this can be found. The disease may show a similar heredity, *i. e.*, father and son may both be afflicted in the same way, and this is true also of the spastic family paralysis. It has in by far the greater number of cases begun before the age of twenty-two years.

**Pathology.**—The lesions are sclerosis of the posterior columns of the cord, slight degeneration of both pyramidal tracts (Sainton), with integrity of the anterolateral columns in some instances (Marinesco), alteration of the columns of Clarke, atrophy of the cells of the anterior horns of the cord, alteration of the peripheral nerves, which may be slight, and of the intramuscular branches, atrophy of muscle fibers, and chronic meningitis (Dejerine and Armand-Delille). The nerve trunks, the cutaneous sensory nerves, and the anterior and posterior nerve roots, with slight exception, may be normal.

**Symptoms.**—The characteristic features of this form of atrophy as given in the first description by Charcot and Marie are: Progressive muscular atrophy implicating first the feet and legs, and not appearing in the upper limbs (hands and, later, forearms) until several years later, the progression of the atrophy being slow. Relative integrity of the muscles of the limbs near the trunk, or at least much longer preservation of these than of the muscles of the distal ends of the limbs. Integrity of the muscles of the trunk, shoulders, and face. Fibrillary contractions in the atrophying muscles. Vasomotor disturbances in the portions of the limbs atrophied. No pronounced contractions of tendons in the atrophied limbs. Sensation usually intact, but sometimes affected. Cramps frequent. Reaction of degeneration in the atrophying muscles. Commencement of the affection usually in childhood, the disease often found in several brothers and sisters, and sometimes in the previous generations.

Charcot and Marie, in their first publication, acknowledged the possibility of implication of the muscles of the limbs near the trunk, as they speak of relative integrity of these muscles. They also stated that the muscles of the thigh seem to preserve their power and volume during a certain period, but that this integrity often is not real. The vastus internus is the first of the thigh muscles involved. Undoubtedly they emphasized the earlier and greater implication of the muscles at the distal ends of the limbs as the most characteristic feature of the disease. Cases absolutely typical in other respects have existed with grave contractures of the tendons of the feet.

The implication of the distal portions of the limbs, with complete or nearly complete integrity of the proximal parts, is the essential feature of the disease.

So soon as we permit cases with intense atrophy of the proximal parts of the limbs, and especially with atrophy of the muscles of the trunk, to be classified under this designation, the diagnosis is probably incorrect. The only typical cases with necropsy reported in the literature are those of Marinesco, Sainton, and Dejerine and Armand-Delille. The cases of Dubreuilh and Siemerling are questionable. In Siemerling's, atrophy was intense in the upper parts as well as in the lower parts of the limbs, and also in the trunk. Such a condition may be caused by progressive muscular dystrophy.

The tendon reflexes are usually lost, but at least one case, although without necropsy, is reported in which they were exaggerated (Dercum). This is so extraordinary, and so in contradiction to what we know of the pathology of the affection, that when it occurs we must think of a complicating lesion, such as pronounced degeneration of the lateral columns.

Although the atrophy begins so frequently in the distal parts of the lower limbs that the type has been designated by Tooth as the peroneal, it may begin in the upper limbs, but its commencement is always in the peripheral parts of the limbs, and the progress of the disease is slow. A few cases have been observed in which the atrophy began in the distal parts of the upper and lower limbs nearly at the same time.

Sensation may be disturbed in the peripheral parts of the limbs, or pain may be felt, but, as a rule, the sensory symptoms are not nearly so pronounced as the motor. They may be various forms of paræsthesia, tingling, numbness, etc.

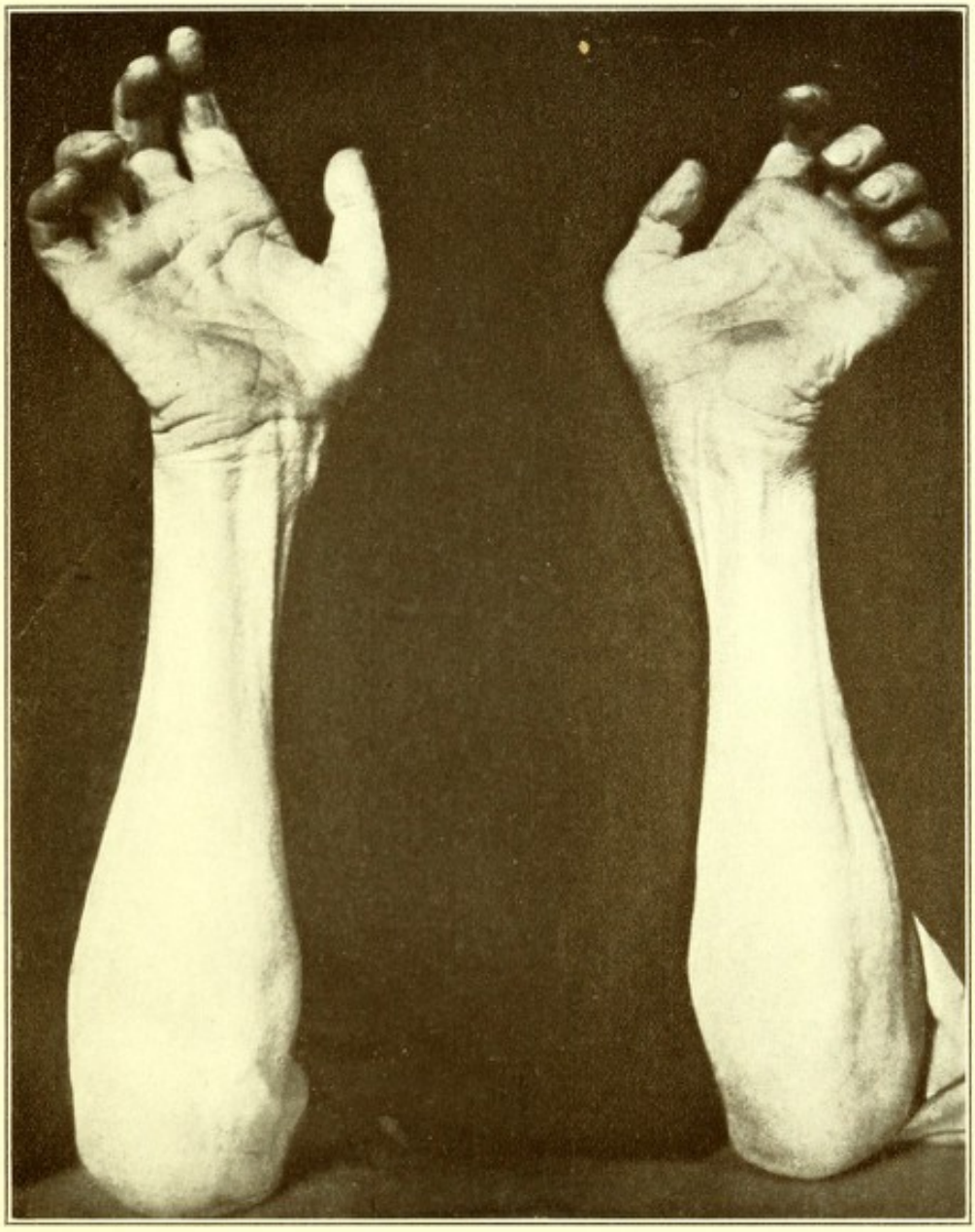
Contractures (talipes equino-varus) are not uncommon in the muscles controlling the movements of the feet, and often they impede the gait very greatly. The atrophy of the legs below the knees may be excessive, and when this stage has been reached the lower parts of the thighs will usually be found comparatively less well developed than the upper parts. All movements of the toes or of the foot at the ankle may be impossible, both because of the atrophy and because of the contractures. The hands may present the condition seen in progressive spinal muscular atrophy, with wasting of the interosseous spaces and contracture, the so-called griffon's claw. The lower part of the forearms may be intensely atrophied, but the upper part is usually in a more nearly normal condition. Fibrillary contractions occur in the wasting muscles, just as they occur in all muscular atrophy when the cells of the anterior horns are diseased. Vasomotor disturbances are common. The wasted limbs feel colder to the touch and appear more or less cyanotic.

The electrical reactions are often altered, and this alteration may vary from diminution in the intensity of the electrical response to reaction of degeneration or even complete loss of the electrical response. Diminution in the faradic response of nerves in regions where muscular atrophy has not been as yet detected has been emphasized especially by Hoffmann.

Mental symptoms, implication of the sphincters, pupillary phenomena, are not part of the symptom complex.

The disease is of slow development, and after reaching its height the patient may live many years without additional symptoms. Thus, in a typical case reported by the writer, the symptoms are said to have been present about forty-five years, and the patient's condition has not changed during the many years he has been under observation. There is not likely to be any tendency toward improvement.

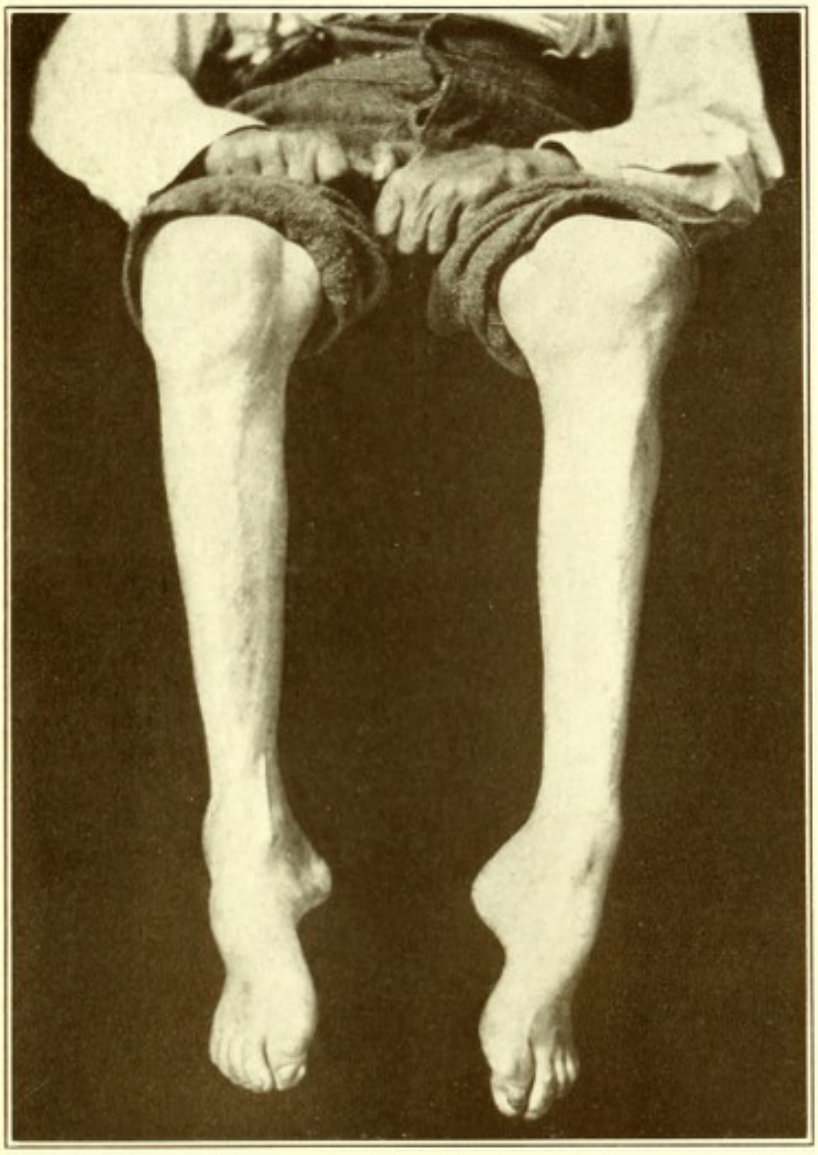
# PLATE IV



Progressive Neural Muscular Atrophy.

Atrophy of the hands and forearms, with escape of the upper arms in the writer's case, lasting about forty-five years.

# PLATE V



Atrophy and Contracture of the Feet and Legs.

With escape of the thighs, in the case represented in Plate IV.

The ocular manifestations have been studied by W. Krauss. They are not frequent, or at least little has been described concerning them. Krauss has found pupillary changes mentioned four times, which, however, are inaccurate, and in 3 or 5 cases atrophy of the optic nerve. In numerous cases the ocular muscles, especially the orbicularis palpebrarum, are described as intact. Krauss concludes that the only reliable ocular manifestation in the neurotic muscular atrophy is optic atrophy, and he reports a case of this kind.

**Diagnosis.**—When the symptom complex is typical the diagnosis is easy. The hereditary or family tendency, the youthful age at which the symptoms commence, the slow progress, the limitation of the atrophy to the peripheral parts of the limbs, the loss of tendon reflexes, the motor disturbances much greater than the sensory—all these make a definite clinical picture. The diagnosis will not be difficult if we refrain from including cases with atypical features. The literature is full of cases that probably do not belong here; indeed, almost any form of atrophy has been accepted as belonging to this type provided it began in the distal portions of the limbs.

Progressive muscular dystrophy is most liable to cause mistakes, and a case with necropsy reported by the writer shows the difficulty in diagnosing between these two diseases. The atrophy began in the lower part of the lower limbs, and was associated with slight reaction of degeneration in these parts and talipes equino-varus on each side. These and the symmetry of the atrophy suggested the neurotic form, but the extension of the atrophy to the proximal parts of the limbs and the muscles of the trunk was unlike the typical form of the neurotic muscular atrophy. The microscopic examination showed that the nervous system was normal, even the intramuscular nerve fibers, but the muscles were intensely degenerated.

*Anterior poliomyelitis* with paralysis confined to the distal parts of the lower limbs may occur and cause some confusion, but when a history of the onset is obtainable the diagnosis is easy.

*Progressive spinal muscular atrophy* or chronic poliomyelitis may resemble the Charcot-Marie-Tooth form, but these diseases are neither hereditary nor familial, do not occur at so early an age, nor does the atrophy remain confined to the distal ends of the limbs.

*Multiple neuritis* usually does not implicate only the peripheral portions of the limbs, and the sensory disturbances are pronounced. Motor neuritis, with little or no sensory involvement, does occur, however, and the Charcot-Marie type is regarded by some as a motor neuritis in which the peripheral ends of the longest nerves are affected, and there is much to be said in favor of this view, although the spinal cord is also implicated.

## INTERSTITIAL HYPERTROPHIC PROGRESSIVE NEURITIS OF CHILDHOOD.

This disease was first observed by Dejerine and Sottas, and was reported by them in 1893. They had two cases occurring in brother and sister, and necropsy was obtained in both. They referred to a case of Gombault and Mallet as belonging to this type, and this case was the first published, although it was considered by the authors as a form of tabes.

Syphilis may have some causal relation; at least, in the second case of Dejerine's a history of syphilitic infection at the age of twenty-four years

was obtained. Alcoholism and poor mental development were present in this second case.

Marinesco has regarded the disease as a variety of the neurotic muscular atrophy, and this view Brasch takes in the report of two clinical cases, although it has been most vigorously combated by Dejerine, who mentions that in the Charcot-Marie-Tooth form incoördination of movements, marked disturbance of sensation, Argyll-Robertson pupils, kyphoscoliosis, and hypertrophy of nerves do not occur, no matter how long the affection may have lasted. The duration of the affection has nothing to do with the hypertrophy of the nerves, according to Dejerine.

Marie presented at the Neurological Society of Paris, in 1906, two cases occurring in a family of seven children, all similarly affected. He regarded the disease as a special form of the interstitial neuritis, although his cases differed in certain respects from the cases of Dejerine and Sottas. Dejerine accepts Marie's cases. They presented the same deformity of the feet, kyphoscoliosis, loss of reflexes, diminution of sensation, disturbance of gait and station, muscular atrophy, and considerable hypertrophy of the nerve trunks. Marie believed the muscular atrophy was different in his cases, inasmuch as it affected only the muscles of the feet and legs, while in the cases of Dejerine the atrophy was general. Marie did not find the Argyll-Robertson sign, but the light reflex was diminished; he also noted absence of disturbance of the sphincters and of the sexual functions.

**Pathology.**—The lesions of the spinal cord are only such as result from degeneration of the posterior roots, and are like those of tabes. The lesions of the nerves are very characteristic, and are parenchymatous and interstitial. The nerve fibers may be altered to such a degree as to leave only empty neurilemma sheaths, but only a certain number are so intensely degenerated. The connective tissue is greatly thickened, and the alteration begins in the periphery and extends to the nerve trunks and nerve roots. The lesions are more pronounced in the nerves and roots of the lower limbs than in those of the trunk and upper limbs, and still more so than in the bulbar nerves; but only the optic and olfactory nerves escape. Such hypertrophy occurs in no other form of neuritis.

**Symptoms.**—The symptoms may be said to be ataxia of the four limbs with muscular atrophy, marked disturbance of sensation with retardation of sensation, fulgurant pains, nystagmus, myosis, inequality of the pupils, Argyll-Robertson's sign, kyphoscoliosis, marked hypertrophy and hardness of all nerve trunks accessible to palpation, *i. e.*, the symptoms of tabes associated with general muscular atrophy, kyphoscoliosis, and hypertrophy of nerves.

Muscular atrophy may vary in different cases; in some it may be very intense, and predominate in the distal parts of the limbs, diminishing toward the trunk. The muscles of the face may be paralyzed and atrophied, and the facies may resemble that of myopathy, with the prominent lips and protrusion of the upper lip, the lips of the ant-eater (tapir), with the transverse laugh (*rire en travers*), so that the corners of the mouth are not drawn upward, and with difficulty to pout the lips or whistle. The territory of the upper branch of the facial so far has escaped. The feet are always in the varus position, the nails are deformed, the first phalanx of the toes is dorsally flexed, and the second and third are plantarly flexed upon the first, forming a right angle with it.

The disturbance of function in the lower limbs is the result of atrophy and ataxia. The gait is steppage, but not like that resulting from paralysis of the extensors of the toes and of the anterior tibial muscle. The steppage is abrupt, the legs are raised and thrown outward, the feet falling quickly upon the ground. A cane is necessary in walking, and the head is held forward, and the steps are carefully watched. Turning around is done slowly, and with caution. Ataxia is pronounced when the feet are together and is pronounced in the upper limbs. The muscles of the larynx may be paralyzed, as in tabes, but, unlike tabes, the sphincters and genital powers are always intact.

The nerves may be double the normal size, and the hypertrophy is uniform without nodes of swelling. Pressure on these enlarged nerve trunks does not cause pain; on the contrary, the nerves are analgesic to pressure and electrical irritation.

There can be no doubt that the interstitial hypertrophic neuritis of childhood resembles the neurotic muscular atrophy in certain of its symptoms, but in others it resembles tabes dorsalis; and yet it is impossible to regard it as an association of the two diseases in the same person. There is evidently a strong family tendency to the affection. It is not likely to be mistaken for any other disorder than those mentioned.

The *treatment* is unsatisfactory. The pains may be relieved by analgesics, and the ataxia lessened by coördinated movements. The treatment is much like that of tabes.

## PROGRESSIVE BULBAR PALSY (GLOSSO-LABIO-LARYNGEAL PARALYSIS).

**History.**—This disease seems to have been first observed by Duménil in 1859, but Duchenne, of Boulogne, first attracted attention by his description, and the credit for making the disorder known has been given to him. Trousseau gave it the name of paralysie-labio-glosso-laryngée. An excellent résumé of the history of this disorder is given by E. W. Taylor.

**Etiology.**—No cause is known, but it is probably an abiotrophy. The motor cells of the medulla oblongata and pons have a vitality sufficient to enable the functions of life to be properly performed until about middle age, or later, and then they begin to degenerate and finally disappear. No reason can be given for the escape of the sensory nuclei in the same regions. Syphilis does not seem to play a role, although it is possible that lead may do so. Infectious diseases may cause acute bulbar palsy by producing a bulbar encephalitis, but it is questionable whether they ever give origin to chronic bulbar palsy. It is exceedingly rare in early life, but the occasional occurrence at that period and in several members of the same family can be explained only by a congenital tendency. Guillain regards intoxication as the real cause, but it is certainly exceptional to be able to determine any intoxication.

Most authors state that males are more frequently affected. In the writer's experience females have been as often subjects of the disease as males. The patient is usually in or past middle life before any of the symptoms develop. It is necessarily a fatal process, and the termination is usually only a question of a few years, two or three.

**Pathology.**—The nuclei of the hypoglossal nerves are usually intensely altered, so much so that nerve cells in these nuclei may have disappeared; in other cases they are few in numbers and much atrophied, with loss of dendritic processes. The intramedullary fibers of the hypoglossal nerves become atrophied and may also disappear. The alteration of the nucleus ambiguus may be very pronounced, but great care is requisite in forming an opinion concerning its condition. The posterior nuclei of the pneumogastric nerves in a case examined by the writer were so intensely pigmented that they appeared degenerated. The nuclei of the facial nerves and more rarely the motor nuclei of the trigeminal nerves are degenerated. The nerves whose function is interfered with are the hypoglossal, facial, pneumogastric, glossopharyngeal, vago-accessorial, and motor trigeminal. Hemorrhages or round-cell infiltration are not found in true progressive bulbar palsy. The process is gradual, and atrophy and degeneration of the motor nuclei and nerves of the medulla oblongata and pons occur. The possibility of degeneration of the motor cells of the medulla oblongata and pons occurring without implication of the pyramidal tracts has been accepted by some and rejected by others. It can hardly be disputed that usually chronic bulbar palsy is a part of amyotrophic lateral sclerosis, but there is not lacking authority for the statement that it may exist alone.

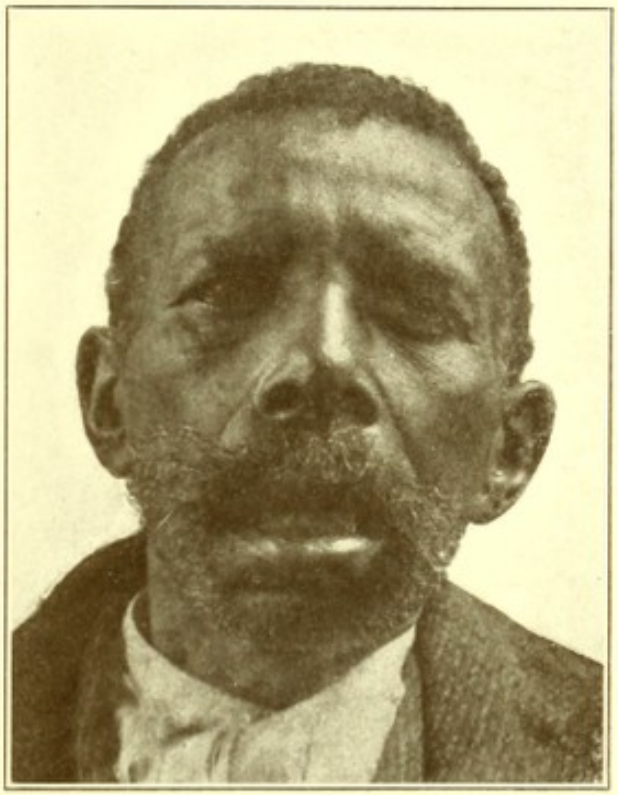
Raymond believes that progressive bulbar palsy is almost always a part of amyotrophic lateral sclerosis, although it may occasionally exist alone. Leyden does not admit the latter as a possibility, and Dejerine, in a treatise with Thomas on diseases of the spinal cord, mentions that in 1883 he demonstrated that the lesion is not confined to the bulbar nuclei, and that the pyramids are distinctly affected. The bulbar paralysis of Duchenne is a pyramidal amyotrophic sclerosis descending in type. With the exception of Reinhold's case (1890), which is not entirely satisfactory, no necropsy of a case of labio-glosso-laryngeal paralysis exists with integrity of the pyramidal tract. Remak's case (1892), quoted by some writers as one with integrity of these tracts, is not one of glosso-labio-laryngeal paralysis of Duchenne, as the superior facial branch was as much implicated as the lower, and the elevators of the eyelids were paralyzed. These symptoms belong to polioencephalitis, but not to Duchenne's bulbar palsy. The latter may occur in two forms, alone or in association with the symptoms of amyotrophic lateral sclerosis. The lesions of the bulbar nuclei are the same in either case, and the pyramidal tract is involved, as demonstrated by exaggeration of the tendon reflexes when the picture is typically that of Duchenne's bulbar palsy.

Chronic poliomyelitis never terminates by bulbar palsy.

Notwithstanding this very positive statement, reasoning by analogy would lead us to believe that uncomplicated bulbar palsy might occur. Dejerine himself has reported two cases in which the nerve cells of the anterior horns of the spinal cord were degenerated without implication of the motor tracts, and if this may occur in the spinal cord it is hard to understand why it may not occur in the medulla oblongata and pons. In several instances the writer has found bulbar palsy in association with amyotrophic lateral sclerosis, but never with integrity of the motor tracts.

The muscles of the tongue are intensely atrophied, and if examined by the method of Marchi may show the presence of fat drops within the muscle fibers. The muscles of the lips and larynx also may be atrophied.

# PLATE VI



Bulbar Palsy.

**Symptoms.**—The clinical picture is most distressing. The patient has difficulty in speaking, progressing to loss of speech; dribbling of saliva, disturbance in swallowing, so that fluids return through the nose, and with much coughing, from the entrance of some of the fluid into the larynx. The ability to pucker the lips, to whistle, or blow out a lighted match is lost, and with all this, intelligence is preserved, and the unfortunate individual has no dulling of the mentality to the frightful state into which he is passing. The disease is remarkably symmetrical in its development.

*Speech.*—Difficulty in pronouncing certain letters—d, g, k, l, r, s, t—is at first noticed; the words are slurred, and gradually speech becomes so indistinct that those who have known the patient many years and are familiar with his voice fail to understand what he says. Beevor observed one case in which the disturbance was observed in singing before it was perceptible in conversation. The defect at first may be noticed only after the patient is a little fatigued in speaking. The voice becomes nasal because of paralysis of the soft palate, and when the larynx is paralyzed the defect of speech has reached its height. The tongue is atrophic, and may be so intensely affected that it cannot be protruded beyond the lips, is full of wrinkles, and shows an intense degree of fibrillary tremor. The reaction of degeneration may be obtained. The weakness of the tongue interferes with mastication of food, as the food cannot be controlled by the action of this organ, and pushed into the pharynx. The atrophy of the lips may be masked by the deposit of fat in the tissue.

The muscles of mastication are less often affected, but may be weak, so that mastication of food becomes difficult. The atrophy and weakness in the distribution of the facial nerve is limited almost to the muscles of the mouth and chin, so that the upper part of the face is not involved, except in the infantile form of bulbar palsy.

Paralysis of the pharynx makes deglutition impossible when the paralysis is complete, but the weakness develops gradually. Liquids then not only may ascend into the nasal passages, but solids may pass into the larynx and thus cause bronchitis or aspiration pneumonia. The reflexes of the soft palate and pharynx are much impaired. The dribbling of saliva because of the weakness of the muscles of the mouth and the difficulty of swallowing may be so great that patients may be obliged to wear bibs to catch the saliva.

The upper part of the facial distribution escapes in progressive bulbar palsy, and this peculiar limitation of the paralysis has led to the conclusion by some neurologists that the centre for the upper distribution probably has a situation remote from that of the facial nucleus, and possibly is in the oculomotor nucleus.

The tendon reflexes of the upper and lower limbs are usually exaggerated, and there may be a pronounced jaw-jerk. Beevor describes a lower jaw clonus which he was the first to report (1886).

The disease is essentially motor in its manifestations, and sensory disorder or enfeeblement of the intellect does not occur. Intelligence is preserved until near death, but the patient is often emotional, laughs and cries on slight provocation, and for a long time maintains a hopeful attitude.

The heart may become affected, the pulse rapid, weak, and irregular, and the muscles of respiration impaired. When these signs develop, death cannot be delayed long. The end may be from inanition.

The gait is often peculiar. The steps may be short and slow, but this form of progression is more common in pseudo-bulbar palsy.

In some instances ocular palsies have been associated with bulbar palsy, but they are to be regarded more as epiphenomena, and not as an essential part of the disease. They may occur in other organic diseases of the nervous system, as in tabes or paretic dementia.

**Diagnosis.**—The greatest difficulty is in regard to *pseudo-bulbar palsy*, and yet in typical cases the difficulty is not great. The writer has seen pseudo-bulbar palsy, however, in which the only distinguishing features were the absence of atrophy, of fibrillary tremors, and of reaction of degeneration in the tongue. Pseudo-bulbar palsy is caused by multiple lesions in the cerebrum, implicating the nerve fibers innervating the motor nuclei of the medulla oblongata and pons. As the lesions are supranuclear, atrophy, fibrillary tremors and reaction of degeneration do not occur.

*Multiple neuritis* confined to the cranial nerves may occur, but it is seldom that the typical picture of bulbar palsy will be presented; often nerves other than the cranial are affected, and sometimes sensory as well as motor nerves are implicated, and the development is usually more rapid in multiple neuritis. Following diphtheria, the soft palate may be paralyzed as well as the sphincters of the eyes and mouth, the frontalis may be weak, and the muscles of expression about the mouth may escape, although the tongue may be affected, and there may be difficulty of deglutition. The symptom complex differs in several details from that of the typical progressive bulbar paralysis.

Bulbar palsy may develop as a result of electricity, as in a case reported by C. K. Mills in association with T. H. Weisenburg.

*Myasthenia Gravis Pseudo-paralytica.*—The resemblance of this disease to progressive bulbar palsy may be very striking, but the rapid exhaustion after slight exertion, the myasthenic reaction to electricity, the implication of ocular nerves and of the limbs, the absence of atrophy, at least in most cases, make the diagnosis possible.

*Tumor, hemorrhage, softening,* or *encephalitis* of the pons and medulla oblongata does not cause symmetrical lesions, does not develop in the same way, and implicates sensory fibers as well as motor.

The *bulbar type of muscular dystrophy*, as described by Hoffmann, is so rare that it may almost be ignored. The presence of other signs of muscular dystrophy should make the diagnosis easy.

*Bulbar palsy* may be a part of tabes dorsalis, but in all the cases except those of Charcot and Pfeifer the tabetic symptoms occurred first. The symptom complex differs from that of progressive bulbar palsy in the implication of sensory cranial nerves, so that the diagnosis of tabes associated with progressive bulbar palsy could hardly be maintained. Optic atrophy, disturbance of deglutition, reflex rigidity of the iris, ocular muscle palsies, and sensory disturbances in the distribution of the trigeminal nerve were present in all the cases; and weakness of the muscles of mastication in all the cases except one. Increased flow of saliva and facial paresis were observed in most of the cases, and only the distribution of the lowest branch of the facial was affected. Disturbances of coördination in the muscles of the face, associated movements, and involuntary movements were present in some of the cases. Atrophy of the tongue was seen in only about half the cases, and was not complete in any case, this being

unlike the condition in progressive bulbar palsy. The case of Oppenheim and Grabower, and that of Cohen and Spiller are the only instances of tabes with bulbar symptoms and necropsy in the literature.

The *bulbar palsy of childhood* has been observed only a few times. In the two young brothers studied by Londe the symptoms began by bilateral paralysis of the upper branch of the facial nerve, with greater intensity of one side. Winking was infrequent, and the eyes could not be opened quickly. In one case slight ptosis was present. There was at first no paralysis of the muscles of the eyeballs. In the beginning of the disorder the facies resembled that of myopathy of the Landouzy-Dejerine type. It differed from the facies of labio-glosso-laryngeal paralysis, as in the latter the lower part of the face is affected, and in Londe's cases the upper part of the face at first alone was affected. Gradually in Londe's cases the double superior facial palsy extended, and the entire facial distribution became implicated as well as the oculomotor, the hypoglossal, the spinal accessory, the trigeminal, and the pneumogastric nerves. The lips became inactive and flaccid, without being protruded. The face became an immobile mask, and the smile was impossible. Fibrillary tremors occurred in the chin. The tongue was paretic and atrophied. The muscles of mastication were affected in one case. Paralysis of the abductors of the larynx occurred in one of the brothers.

**Treatment.**—Little can be done to arrest the development of progressive bulbar palsy, and nothing to stop it. The feeding with liquid or semi-liquid food is important, and tonics, especially strychnine, may be used.

## PROGRESSIVE MUSCULAR DYSTROPHY (PRIMARY MYOPATHY).

**History.**—The various types of myopathy which at one time were regarded as distinct entities have been classed by Erb under one designation, and recognized as being merely varieties of one disease. Credit has been given to Duchenne, as reporting the first case (1861), although at that early period he regarded its origin as central. Later he recognized its independence of the central nervous system. The muscular origin of the disease was demonstrated by Eulenburg, Cohnheim, and Charcot. Duchenne described the facial form, but believed it to be of central origin. Landouzy and Dejerine clearly showed that the facio-scapulo-humeral type is a primary myopathy.

**Etiology.**—Like in the other affections considered in this chapter, abiotrophy is the chief cause of this disease. Here, however, the muscular system instead of the central nervous system is affected, and its vitality may be so slight that wasting begins in some cases soon after birth. It is not improbable that some acute infectious disease may be the exciting cause, but the predisposition already exists. The resistance of the muscles varies in different cases, and, therefore, the time of commencement of the disease varies. Heredity and familial occurrence demonstrate clearly the congenital weakness of the tissues, and the mother seems to be more liable to transmit the disease than the father. There are many cases in which no heredity or familial tendency can be traced. A few cases have been observed in which the disease seemed to develop after typhoid fever (Friedländer, Josserand, Guillain).

The disease first makes its appearance in early childhood, but cases are on record in which it began in middle life or even later. Erb believes that functional alteration of the nerve cells, undetectable by any means at our command, may cause the alteration of the muscle fibers, so that in this way muscular dystrophy may be of nervous origin. Landouzy and Dejerine, and still earlier Vulpian, entertained similar views.

Convulsions, as well as diabetes insipidus, nystagmus, hemicrania, hysteria, chorea, and psychoses have been observed with muscular dystrophy. Such combinations, in Erb's opinion, are too frequent to be without influence on our judgment regarding the central origin of the dystrophy. Hypertrophied fibers also have been seen in poliomyelitis (Müller, Dejerine), and Hitzig and Kawka have found exactly the same changes in the muscles in this disease as in muscular dystrophy, and in the former disease the cause is spinal.

Direct heredity in muscular atrophy can no longer, according to Hoffmann, possess the diagnostic value which it was once supposed to have, although it must be conceded that it is more important in the myopathic and neurotic forms (Charcot-Marie-Tooth type) than in the myelopathic variety. At first the Leyden-Möbius type was regarded as the only form of muscular dystrophy hereditary in origin. Onuf has found six cases of epilepsy in which symptoms of muscular dystrophy were present.

The negro seems to enjoy largely an immunity to muscular dystrophy, as shown by Eshner.

Some cases indicate that trauma may be a cause, but when the case is merely a clinical one, as Kramer's was, and the picture is not typical, the relation of trauma to the disease must be accepted with hesitation.

The congenital tendency to the disease was especially well shown in a family observed by Bunting, in which three male members were affected at the same age (five years). The boys of this family were susceptible, the girls escaped. It seems, indeed, extraordinary that sex should influence the onset of this disease, especially at so early an age.

**Pathology.**—The lesions are in most cases purely muscular, the atrophy may be so intense that no muscular fibers remain, in other cases the fibers are smaller in size, and yet the muscle spindles are intact, even in the most extreme atrophy of the surrounding muscle. It is probable that these spindles are organs of sensation and possibly are concerned with the sense of position.

Landouzy and Dejerine believed muscular dystrophy to be a progressive atrophic myopathy resulting from a primary parenchymatous myositis with hypertrophy of certain muscular fibers, terminating in "simple" atrophy. The interstitial myositis is slight, and fatty infiltration may be found in some muscles. The vessels and intramuscular nerves, they stated, had never been found diseased. Some investigators have believed that the disease begins in the interstitial tissue of the muscles, others in the vessels, and still others in the muscle fibers. By "simple" atrophy a condition is meant in which the muscle fibers become smaller, but otherwise normal (Löwenthal); when the structure of the muscle fibers is changed the atrophy is called "degenerative," but these distinctions cannot be sharply maintained.

The findings are hypertrophy and atrophy of muscle fibers, proliferation of their nuclei, vacuolation, splitting of muscle fibers, hyperplasia of connective tissue and proliferation of its nuclei, thickening of the walls of the vessels and proliferation of their nuclei, accumulation of fat cells. The

degree of alteration varies in different cases, but the differences are merely quantitative.

Erb mentions that more or less important changes have been found in the anterior horns of the spinal cord, but that negative findings are the rule, the positive the exception. These cellular changes are important in the understanding of the pathology, as they demonstrate that the most typical clinical manifestations of progressive muscular dystrophy may be with important alteration of the nervous system, although we do not know why this should be; whether it depends on the type, duration, intensity, external causes or complications. The changes in the muscles are primary, those in the connective tissue secondary.

The question as to whether the atrophy of muscular dystrophy differs from that of other processes has interested many, and the question arises whether the histological findings are sufficient to permit a diagnosis of the disease. On one occasion the writer examined a piece of tissue removed from the sterno-cleido-mastoid muscle at operation. The findings were so similar to those described by Erb that it was suspected the case might be one of muscular dystrophy, and such it proved to be, but still those findings were not pathognomonic. Erb acknowledged that muscular findings similar to those of dystrophy might occur in other diseases, as they had been seen in spinal disorders, in myositis, near tumors situated in muscles, and in muscles in regeneration after injury.

**Symptoms.**—The characteristic features are: The early age of commencement. The hereditary or familial appearance. The portions of the body affected, the atrophy beginning in the proximal portions of the limbs and trunk. Pseudo-hypertrophy of certain parts of the body, especially of the calves. Quantitative diminution of electrical responses, depending on the degree of atrophy, but reaction of degeneration absent in most cases. Diminution or loss of tendon reflexes. Absence of fibrillary tremors.

The atrophy almost invariably begins in the proximal parts of the body and the muscles of the trunk; in some instances the muscles of the shoulder girdle are affected first, in others those of the lower part of the trunk. The atrophy, however, is not always the first sign, for the muscles of one calf may enlarge in association with a little weakness of the leg, before any atrophy can be detected. In rare instances the atrophy first begins in the distal portions of the limbs (Oppenheim and Cassirer, Dejerine and Thomas, Gowers, Spiller).

Weakness usually precedes atrophy, and at first is confined to certain portions of the body; later the muscles of all the limbs as well as those of the trunk may be greatly wasted. Where two or more members of a family are affected the atrophy may show different types, not commencing in corresponding parts in members of the same family, but occasionally the same type is preserved in a family. Dystrophy of the muscles of the shoulder girdle in father and son was observed by Ogilvie and Easton. The shoulder girdle is frequently implicated, and the muscles affected are the pectorals, supraspinatus, infraspinatus, trapezius, rhomboid, subscapularis, biceps, triceps, brachialis anticus, and supinator longus; and it is not until late that the muscles of the forearms and hands are likely to show wasting. In the lower limbs the muscles commonly affected are the gluteal, and the muscles of the anterior and posterior parts of the thighs. The muscles of the face and neck are only occasionally wasted, and when this occurs a distinct

type is produced. Usually, in such cases, only the muscles of expression are involved, but the masseters, tongue, and ocular muscles have been found implicated a few times.

In a patient observed by Marie closure of the eyelids was impaired, as in the infantile type, and marked double ptosis existed. The muscles of mastication were affected in the same case. Marie has found a few cases reported in which either ptosis or implication of the muscles of mastication occurred alone in muscular dystrophy, but no case in which both occurred together.

The pseudo-hypertrophy attacks most frequently the muscles of the calves. This hypertrophy is caused by overgrowth of fibrous and fatty connective tissue, although here and there true hypertrophy of certain muscle fibers may be found in the midst of the atrophied muscles, but they do not cause any increase in the bulk of the muscle. In some cases the pseudo-hypertrophy may occur in the upper limbs in the triceps or deltoid muscles. The atrophy is usually seen in groups of muscles functionally associated.

Reactions of degeneration should arouse suspicion that the atrophy may be both myopathic and myelopathic. Certain cases have been reported of the myopathic type in which it has occurred.

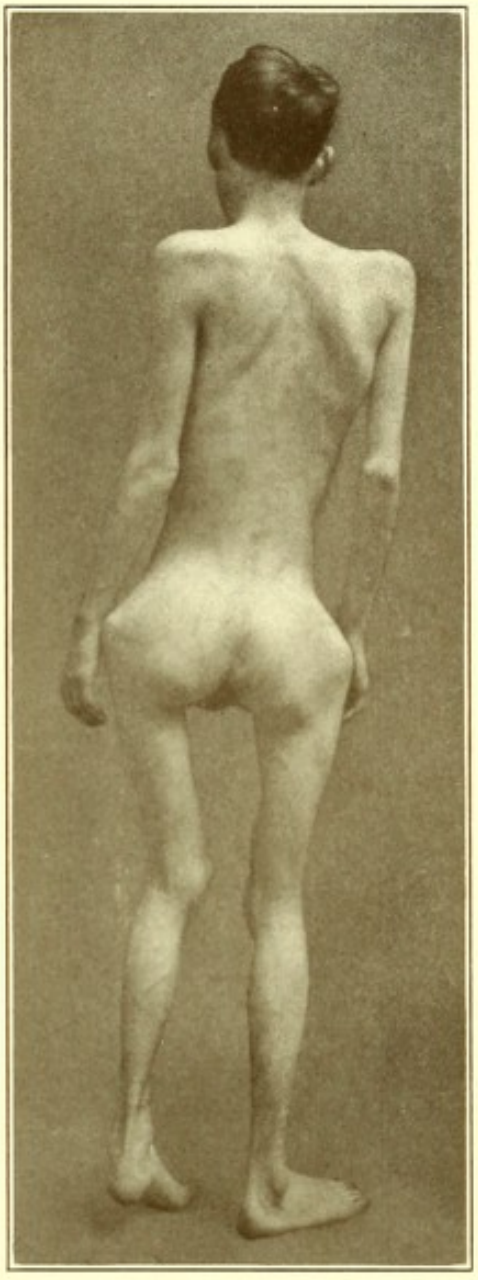
Fibrillary tremors do not belong to progressive muscular dystrophy, but they have been observed in some cases. They are of much value in diagnosis when the nerve cells of the spinal cord and bulb are believed to be affected. When they occur in muscular dystrophy they are usually confined to one or a few muscles, and are usually of transitory duration.

The condition of the tendon reflexes depends, in large measure, upon the degree of atrophy. Where the quadriceps femoris is much wasted the patellar reflex must be diminished or lost, but in some cases this reflex may disappear before any diminution in the size of the muscles can be detected. It is presumable, however, that some change has already begun in the muscle fibers, making them less capable of responding to stimulation.

Peculiar attitudes may be caused by the wasting of certain groups of muscles. Where the shoulder girdle is attacked the condition of "winged scapula" and "loose shoulders" may be produced. If the patient be lifted by the axillæ the shoulders are carried so far upward as the tissues permit. When the muscles of the back are wasted lordosis may be very pronounced, in rare cases it may be so extreme that the abdomen rests upon the thighs. More rarely lordosis is caused by weakness of the abdominal muscles. A condition occurring in muscular dystrophy, in which the muscles of the neck are affected, has been called by Ballet and Delherm "facies of the sphinx." It consists of an enlargement transversely of the base of the neck and flattening in the anteroposterior diameter. It is associated with atrophy of other muscles, and is said to be common in muscular dystrophy. Marie regards the deformity sufficient for a diagnosis of muscular dystrophy. The commencement of the atrophy in the muscles of the neck has been observed by Long.

Contraction of the biceps tendon so that the forearm cannot be extended has been noted especially by Landouzy and Dejerine, but it is not of common occurrence, and probably depends on pronounced muscular atrophy. The flexor muscles on the back of the thighs are sometimes much contractured, and the legs are greatly flexed upon the thighs. Retraction of muscles occurs early or late in the disease, especially in the biceps of the arm, flexors of the

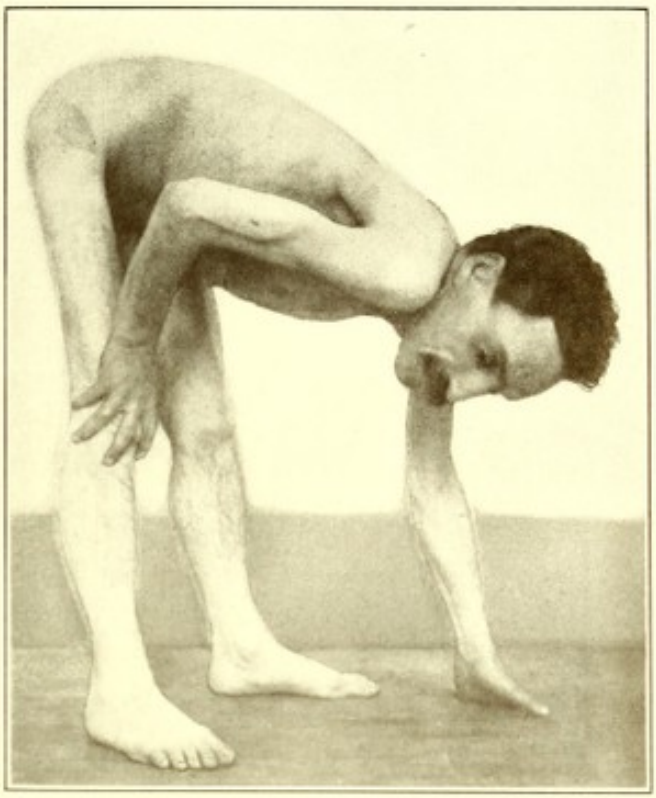
# PLATE VII



Progressive Muscular Dystrophy. Intense and general muscular wasting.

# PLATE VIII

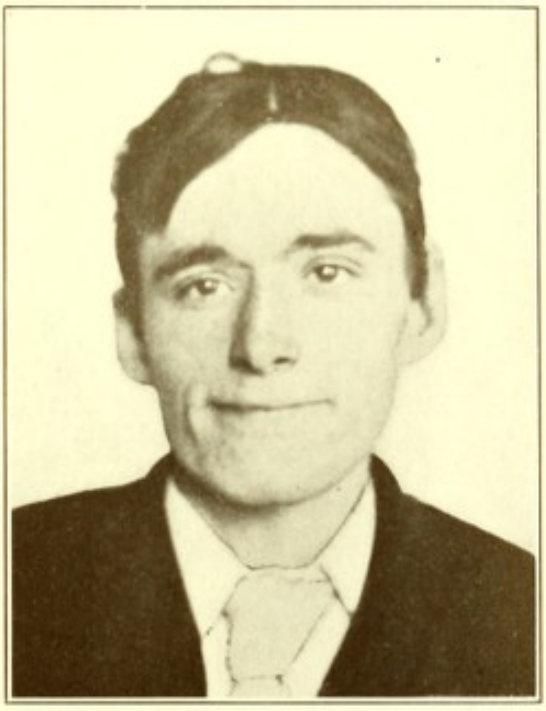
FIG. 1



The Method of Rising from the Floor as shown in Progressive Muscular Dystrophy.

This is seldom seen so late in life as in the patient represented, inasmuch as the disease begins usually in childhood, and by middle life the disability has become too great.

FIG. 2



The Transverse Laugh (rire en travers) seen in the Facial Type of Progressive Muscular Dystrophy.

knee, and muscles of the calf. The cause is not very evident. It is produced partly in the same way as the paralytic contractures, partly by proliferation and retraction of the connective tissue. Where contracture is the first sign of dystrophy it is probably not produced by retraction of fibrous tissue.

The wasting of the gluteal muscles causes a peculiar elevation of first one hip then of the other in walking, the duck walk (*marche de canard*), as it has been called by the French writers. It is extremely characteristic of progressive muscular dystrophy.

The weakness of the muscles of the back causes a peculiar disturbance in rising from a sitting position on the floor. The patient turns so as to support himself upon his feet and hands; he then places one hand after the other upon his lower limbs, gradually by raising his hands pushing his trunk upward, and in this way climbs up on his lower limbs to the erect position. While not pathognomonic of progressive muscular dystrophy, it is seldom seen in any other affection.

Landouzy and Dejerine believed that the muscles of respiration escape, and in their first case with necropsy the diaphragm was normal, but in a case studied by the writer it was intensely atrophied. Duchenne observed clinically atrophy of the muscles of respiration, and it seems probable, from the study of Bunting, that the heart muscle may be affected.

The belief in the escape of the muscles innervated from the bulb, except those affected in the infantile form, has been shattered by Hoffmann. This investigator has shown that the disease may begin as myopathic bulbar paralysis, and remain as such for some time, and that external ophthalmoplegia may be a part of the symptom complex. Marie observed one case of progressive muscular dystrophy in which the orbicularis palpebrarum muscles were feeble, but the lids could be closed. Double ptosis was pronounced and the muscles of mastication were very weak. It is questionable if any group of muscles possesses more than a relative immunity.

Mental symptoms are not pronounced, but the tendency to degenerative change in the muscles seems to be associated with a form of abiotrophy of the brain; the child, while not imbecile, usually lacks the capacity for high mental development.

Atrophy of bone has been observed in a few cases (Lloyd, Marie, Cruzon, Spiller, Schlippe). In a case reported by the writer the bones of the face as well as those of the limbs were atrophied. An *x*-ray examination showed that the humerus and scapula on the left side were smaller than the corresponding bones on the right side. The left acromion process and glenoid cavity were unusually small, and the head of the humerus appeared as though it were dislocated forward. The humerus seemed to be deficient in lime salts. The ribs on the left side were larger than those on the right side.

Scleroderma occurs with muscular dystrophy, and is regarded by Meige as not infrequently associated with it.

Various types of progressive muscular dystrophy have been recognized: (1) The pseudo-hypertrophic; (2) the infantile (Duchenne); (3) the scapular, or juvenile (Erb); (4) the facio-scapulo-humeral (Landouzy-Dejerine); (5) the hereditary (Leyden-Möbius).

The pseudo-hypertrophic is the form best known, as it was the earliest recognized (Duchenne), although the juvenile is of frequent occurrence. The implication of the face is extremely uncommon in America, and, indeed

is seldom seen in other countries than France. The pseudo-hypertrophic form affects especially the muscles of the calves, and is more common in males. One or both calves may be affected, and where only one is involved the implication of the other is usually only a question of time. Later, the enlargement may disappear when the atrophy has become intense.

The facio-scapulo-humeral type includes the infantile type of Duchenne and the juvenile type of Erb. The muscles of the shoulder girdle are wasted, also those of the upper part of the upper limbs (deltoid, biceps, triceps, brachialis anticus), as well as the muscles of the neck and face. The eyelids cannot be closed, so that the appearance of bilateral facial paralysis is presented on superficial examination, the lips cannot be puckered as in whistling, and they are usually prominent because of pseudo-hypertrophy, the corners of the mouth are drawn outward in laughing instead of upward, and the muscles of the cheeks and those of mastication may be much wasted. The peculiar appearance of the face has received the name of facies myopathique.

Infantile form is an unfortunate designation, as the atrophy may commence in adult life, or even at an advanced age. It is often associated with atrophy of the rest of the trunk and of the lower limbs, and sometimes may follow the latter. This form has interested the French especially. Duchenne was the first to clearly separate this type clinically; he called it progressive muscular atrophy of childhood, and believed that the commencement in the facial muscles never occurred in the adult. He regarded it as the same disease as the progressive spinal muscular atrophy of adults, and called it "atrophie musculaire progressive de l'enfance."

The hereditary type (Leyden-Möbius) hardly deserves recognition as distinct from other forms. It begins in the lower limbs, as does the pseudo-hypertrophic, ascends from the leg and thigh muscles to those of the pelvic girdle and trunk, but is not associated with pseudo-hypertrophy. It shows very pronouncedly the hereditary tendency. It is dystrophy on an hereditary basis, as are all the other forms. The localization of the first signs and the unimportance of the true or false hypertrophy in many cases gave the first stimulus to a separation of this type (Erb). Only a few distinct cases of this type are on record, and Leyden himself reported only one; this, according to Erb, was merely the juvenile form, with predominance of the involvement of the lower limbs. Leyden believed the absence of pseudo-hypertrophy was sufficient to distinguish it from the pseudo-hypertrophic form, but Möbius showed that this idea was erroneous. The pseudo-hypertrophic form may, by disappearance of the fatty tissue, change into the Leyden-Möbius type. The latter has, therefore, merely historical interest, and can no longer be recognized.

The type of Zimmerlin consists of atrophy of the proximal parts of the limbs and upper part of the trunk, but may begin in the lower limbs and be associated with atrophy of the face. It begins usually about puberty or a little later, but the type is not sharply defined and is closely allied to Erb's juvenile form; indeed, the latter includes the pseudo-hypertrophic form and the Leyden-Möbius form.

**Diagnosis.**—The clinical appearance usually is so well defined that the diagnosis is not difficult. Babinski has observed hypertrophy of a limb developing after typhoid fever, and this condition may resemble muscular dystrophy. Hypertrophy may occur also in syringomyelia. Lipomatosis

may be found in the limbs paralyzed in acute anterior poliomyelitis, and may then present the appearance of pseudo-hypertrophic muscular paralysis. Multiple neuritis may be confined to the thighs, as observed by Patrick. The subjective and objective sensory disturbances usually make the diagnosis easy, but in a later stage where the symptoms are chiefly or entirely motor, difficulty in diagnosis might arise.

The resemblance to the Charcot-Marie-Tooth type has already been mentioned, and so soon as this type is enlarged to include cases in which the atrophy is not confined to the distal portions of the limbs, or atypical signs such as implication of facial muscles occur, the diagnosis becomes questionable.

*Anterior poliomyelitis* may resemble in its late stages progressive muscular dystrophy, especially if the abdominal muscles be paralyzed, but when a history of the onset can be obtained the diagnosis is easy.

In rare instances myasthenia gravis may have some resemblance to progressive muscular dystrophy (Grund), but the diagnosis between these two disorders should be easy.

The greatest difficulty may occur in distinguishing between the myelopathic and myopathic forms, and in certain cases the decision must be left in doubt, at least for a time.

Disease of the spinal column or of the lumbar muscles may cause the patient to rise from the sitting posture on the floor much as does one afflicted with progressive muscular dystrophy. Congenital defect of muscle on superficial examination may cause difficulty in diagnosis. The defect is not progressive.

**Prognosis.**—Muscular dystrophy is very chronic in its development, and not likely to cause death unless vital muscles become implicated. The afflicted person may, however, have diminished resistance to other diseases.

**Treatment.**—Overexertion is to be avoided. A moderate degree of massage and electricity may be useful in the early stage when only a few muscles are wasted. Exercise should be recommended, as it is probable that a muscle will not waste so rapidly if it be employed in such a manner that use does not become abuse. It seems doubtful whether we have any means of influencing the progress of the disease to a notable degree, but in certain cases massage, passive movements, and electricity seem to be of decided benefit.

# CHAPTER III.

## THE COMBINED SYSTEM DISEASES OF THE SPINAL CORD.

By COLIN K. RUSSEL, M.D.

Combined disease of both afferent and efferent systems of fibers in the spinal cord is found in many conditions, although not all of these can be regarded as true system diseases. In meningomyelitis, myelitis, and multiple sclerosis, the involvement of several of the spinal tracts may, with the subsequent ascending and descending degeneration, give the clinical and anatomical picture of a combined sclerosis. Such a condition has been described also in cases of arteriosclerosis and following injury with contusion of the spinal cord. In these cases we have rather a pseudo-systemic disease of the cord. Clinically, the fundamental character of this form is the spastic weakness of the lower extremities, associated in many cases with ataxia, some loss of sense of position in the extremities or other form of sensory loss. These conditions will be taken up more fully under their respective chapters. It will suffice to state here that ataxic paraplegia of Gowers and spastic paraplegia of Strümpell should be regarded as symptoms only, and not as definite clinical entities. In the majority of cases showing these symptoms in young or middle-aged individuals, with the exception of the subacute conditions to be taken up later, they are merely the early evidence of disseminated sclerosis.

In tabes dorsalis, while the disease is usually confined to the posterior columns, occasionally the direct cerebellar tract also shows degenerative changes following on the destruction of the cells in Clark's column (Kattwinkel, Oppenheim) without causing recognizable symptoms. Not infrequently also, as was first observed by Erb in advanced cases of tabes, degeneration of the upper motor neurone may occur, giving rise to associated sclerosis of the crossed and direct pyramidal tracts—a true systemic combined sclerosis in some cases—while in others, and according to Crouzon[1] more frequently, it is a pseudo-systemic sclerosis, due to involvement of the lateral columns by disease spreading in from the periphery of the cord from lesions of the lymphatic and circulatory systems. Clinically we have then the symptoms of a spastic paraplegia superimposed on the ordinary tabetic features. If, on the contrary, these myelitic lesions involve the lumbar enlargement and destroy the root zone of the posterior columns, we will have a flaccid paraplegia, and the only direct evidence of involvement of the pyramidal tract may be the presence of Babinski's extensor plantar response.

In general paresis of the insane combined degeneration of the posterior lateral columns was first observed by Westphal, and has since been con-

[1] *Paris Thesis*, 1904.

firmed by many other observers. In the combined statistics of Tuczek, Furstner, Sibelius, and Crouzon, comprising 200 cases, combined sclerosis was present in 96 cases, that is, in 48 per cent.

## SUBACUTE COMBINED SCLEROSIS OF THE SPINAL CORD.

Leichtenstern,[1] and later Lichtheim,[2] first called attention to the association of symptoms pointing to involvement of the spinal cord in cases of pernicious anæmia, and since that time, thanks to the observations of Putnam,[3] Dana,[4] and more recently Grinker[5] in America, of Bowman,[6] James Taylor,[7] and especially Risien Russell,[8] in England, and of Minnich, Van Noorden, Eisenlohr, and Nonné, on the Continent, our knowledge of the disease has been considerably increased, and it may now be considered as a definite clinical entity.

**Etiology.**—Subacute combined sclerosis occurs usually in individuals in the fourth or fifth decade, although some cases have been reported as occurring in the third and a few in the sixth. Males are just as frequently affected as females, and there is some neuropathic hereditary tendency present in many cases. More important and more constant than this is the almost invariable evidence of some toxic condition, some wasting disease, prolonged suppurative process, chronic digestive disturbances with diarrhœa, or acute or chronic infections. Syphilis apparently plays no important part as an etiological factor. The blood changes so commonly found in these cases are more often of the nature of a secondary anæmia than of the pernicious variety, and are probably associated conditions rather than etiological factors, certainly in the majority of cases.

**Pathology.**—The cerebrum, cerebellum, and pons in the great majority of cases show no lesions. Externally the spinal cord and membranes are normal except for an anæmic appearance in some cases. Macroscopically, on section the grayish discoloration of sclerosis may be evident in the posterior columns, and sometimes in the region of the crossed pyramidal tract. In many cases, however, this becomes evident only after hardening in Müller's fluid.

Microscopically, with the Pal-Weigert method, the most extensive lesions are found in the mid-dorsal region of the cord. At this level almost the whole of the white matter, especially the peripheral parts, may be involved, leaving only the gray matter and a small area of white matter immediately surrounding it perfectly normal. Endogenous and exogenous fibers are alike affected.

The posterior columns, which are usually most affected, may be completely sclerosed except for a small area immediately on the inner border of the gray matter of the posterior horns. At higher levels of the cord the lesions diminish progressively and tend to be limited to the posterior columns,

[1] *Deutsch. med. Woch.*, 1884. [2] *Neuro. Centralblatt*, 1887, p. 236.
[3] *Journal of Nervous and Mental Disease*, 1891, xvi, 69.
[4] *Ibid.*, xvi, p. 205; 1899, xxvi, 1.
[5] *Journal of the American Medical Association*, 1908, l, p. 1109.
[6] *Brain*, 1894, xvii, 198.
[7] *Trans. Royal Med. and Chir. Soc.*, 1895, lxxviii, 151.
[8] *Lancet*, 1898, ii, 4.

the direct cerebellar tracts, and the pyramidal tracts, both the direct and crossed. Especially in the upper dorsal and lower cervical segments is the degeneration in the region of the direct pyramidal tracts marked. The degeneration in the pyramidal tracts gradually diminishes, and usually has disappeared at the medullary level, although occasionally it can be followed up into the pons by the Marchi method. The degeneration of the direct cerebellar tract is followed by the same method into the inferior cerebellar peduncle, and that of the upper portion of the ventral cerebellar tract (Gowers) has been traced into the velum medullare anterius.

Below the mid-dorsal region the diffuse destructive lesion diminishes progressively until in the lumbar region the degeneration is confined to the crossed pyramidal and posterior columns. Marchi's method reveals lesions similar to those already described, but somewhat more extensive. Lissauer's tract is not affected, even in the mid-dorsal region, where the periphery of the cord shows marked destructive changes. The cells of the gray matter may show degenerative changes (Putnam, Dana), but, as a rule, these are not at all marked. The nerve roots, especially the anterior ones, were more or less affected in Putnam's cases, but in only one of the cases, reported by Russell, Batten, and Collier,[1] could degenerative changes be demonstrated in the peripheral nerves.

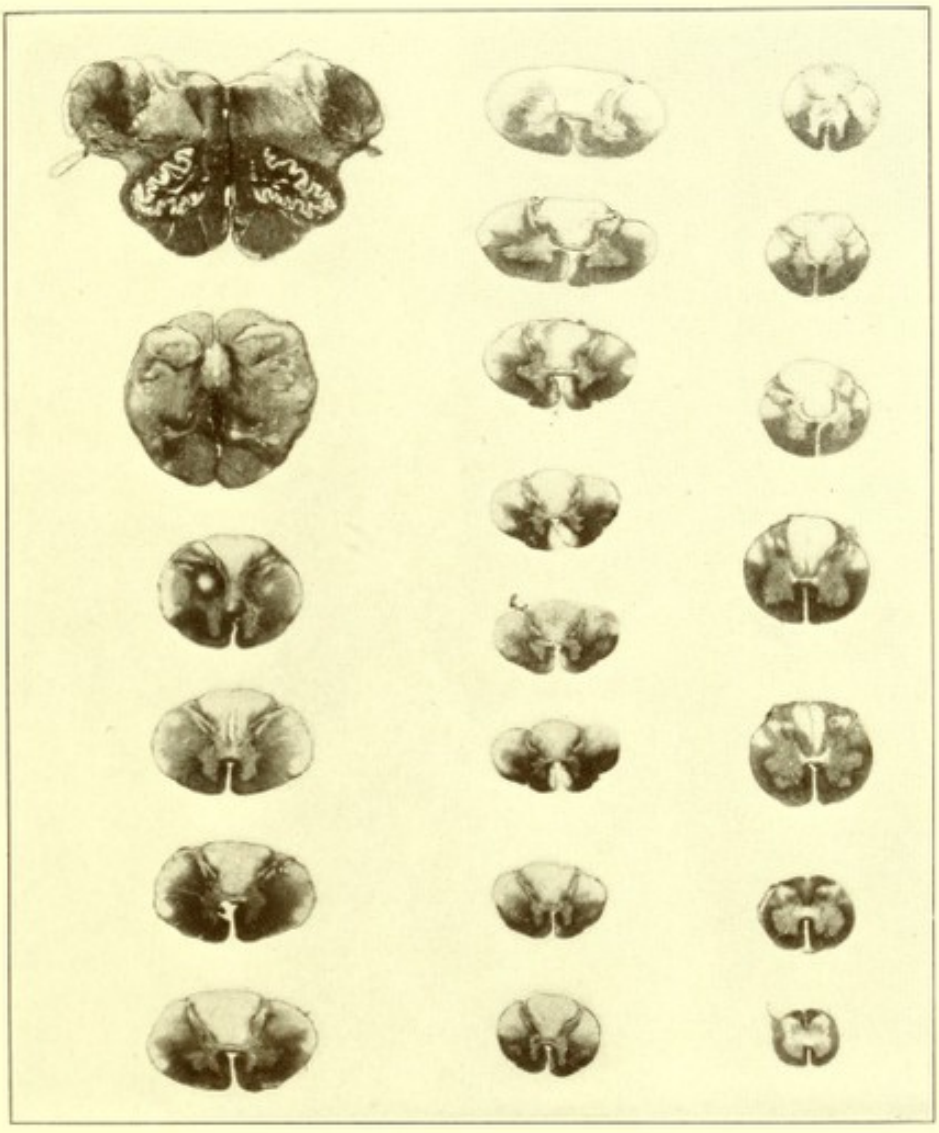
The vessels in the cord, in the affected areas at least, may be engorged and their walls in some cases thickened, but no evidence of thrombosis has been observed. In other parts of the cord they may be quite normal. Minute hemorrhages may be present, associated with the anæmia, but having no etiological relationship to the degenerative changes. Russell and his fellow observers are of the opinion that there are two distinct processes at work, first, a system degeneration, and secondly, a focal destructive lesion. With regard to the latter, the first step is a swelling of the medullated sheaths, going on to fatty degeneration, with disappearance of the axis cylinders, absorption of the degenerative products, and later, overgrowth of fibrous tissue. There was in Dana's cases no evidence of any true inflammatory process. As to the former—the system degeneration—the long tracts of the cord exhibit well-marked degeneration and sclerosis. Generally there is clinical evidence to show that the combined sclerosis exists for some time before the onset of the diffuse lesion. This latter always affects the lower cervical or dorsal part, and is usually a warning of an early exitus.

**Symptoms.**—While Risien Russell has divided the course of the disease into three stages, further observations show that these are not always so evident. For the purpose of description, however, they will be retained.

1. The period of mild spastic or ataxic paraplegia. The onset of the disease is in most cases slow and insidious, although in some instances quite acute, being ushered in by headache, vomiting, pyrexia, and malaise. In others it is the symptoms of anæmia, palpitation, fainting attacks, and so on, that bring the patient first under observation. The first symptoms complained of, attributable to the involvement of the nervous system, are subjective feelings of numbness and tingling in the lower extremities with perhaps some stiffness. These are never severe at first, but soon signs of slight spasticity and ataxia develop. The lower extremities are clumsy, feel stiff and cold, and the toes stick to the floor a little in walking. At this period

[1] *Brain*, 1900, xxiii, p. 39.

# PLATE IX



Subacute Combined Sclerosis.

there is no loss of control of the sphincters and no evidence of involvement of any other part of the nervous system. One finds on examination some impairment of the sense of position in the lower extremities when they are moved passively, with increased reflexes, and the extensor plantar response (Babinski). As a rule, later, though sometimes coincidently, the same symptoms appear in the upper extremities. This period of ataxic paraplegia occupies half or three-quarters of the whole duration of the disease.

2. This period is one of complete spastic paraplegia, with considerable loss of sensibility in all its forms in the lower extremities and lower part of the trunk. The transition from the first to the second stage is, as a rule, abrupt; often in the course of twenty-four hours the patient loses the power of standing or walking, owing to the absolute loss of sense of position in the limbs, and not from any loss of motor power; which is usually not much impaired. Throughout this second stage the motor power diminishes gradually, as a rule, but is not entirely lost until the third stage is reached. The upper extremities are affected also with the loss of sense of position, and often show tabetic athetosis. The loss of cutaneous sensibility is at first peripheral in type, but as it extends upward becomes segmental in distribution, and its upper limit on the trunk is usually well defined, it may extend as high as the area of distribution of the sixth cervical segment. Girdle sensation and lightning pains are not uncommon, and frequently the patient complains of a severe constant dragging pain beneath the lower costal margin. Herpes may occur, and Russell reports one case with cutaneous hemorrhages similar to those observed by Strauss in tabes. The mental condition remains unimpaired; and only occasionally is there any implication of the cranial nerves. There may be some loss of sphincter control; trophic disturbances are not present. The deep reflexes are all exaggerated, and there is extensor plantar response (Babinski).

In some, though by no means in all cases, the symptoms of involvement of the nervous system are associated with the evidences of anæmia. There is constantly some irregular pyrexia. The average duration of this stage of the disease is about five weeks, then in the course of a few days the third period follows.

3. The third stage, that of *flaccid paraplegia*, is characterized by an absolute flaccid motor paralysis of the lower extremities. The trunk muscles also, with the onset of the third stage, show complete paralysis below a segmental level, and Beevor's sign (*i. e.*, the riding up of the umbilicus on the attempt to raise the head from the bed, due to the paralysis of the lower segments of the recti abdominis) can, as a rule, be observed in this stage. Subsequently the upper abdominal and spinal muscles and the lower intercostals are progressively attacked. There is absolute loss of sensibility, loss of the tendon-jerks, rapid muscular atrophy, and rapid loss of excitability of the muscles to faradic stimulation; œdema of the lower extremities and the lower part of the trunk with loss of sphincter control follow.

With the onset of these signs there is an elevation of temperature, with general malaise, drowsiness, anorexia, and general asthenia. If anæmia be already present, it is much increased. In the lower limbs and trunk, then, the wasting is general, but in the upper extremities it is of a segmental character, the intrinsic muscles of the hand being first affected, then the ulnar flexors, radial flexors, extensors of the wrist, extensors of the elbow, supinators, and fifth cervical group in that order. In some cases the diaphragm may

become paralyzed. Involvement of the cranial nerves is very exceptional. Mental disturbance is usually seen in this stage, and there may be convulsions. Trophic changes and bedsores are apt to occur in spite of the most careful treatment. The deep reflexes are lost with the onset of flaccidity, while the extensor plantar response (Babinski) remains present. The duration of this period is about six weeks, though exceptionally it may last several months, death being due either to sudden syncope or respiratory failure.

One of the characteristic features of this disease is its progressiveness; neither remissions nor amelioration of the nervous symptoms ever occur, even though the general condition may improve somewhat. There is always some irregular fever not to be accounted for by associated conditions, such as cystitis, bedsores, etc. The duration of the disease varies from a few months to two or three years. The relation of the anæmia to the cord lesion is not invariable; in some cases it precedes the appearances of the nervous symptoms, in others it may appear coincidently with the nervous symptoms and increase progressively with them; or it may be present only at the end of a prolonged third stage when emaciation becomes extreme, or again there may be no anæmia during the whole course of the disease.

The essential nature of this process is evidently a primary nerve degeneration. The same toxin that brings this about may cause more or less severe disturbance of the blood-forming elements. Its action on the nervous system affects first, as one would expect, those long tracts whose mere length must necessitate a more elaborate nutrition for their maintenance. The circulatory conditions probably affect the preterminal diffuse lesion in the dorsal region in the same manner that acute transverse myelitis usually affects the dorsal region of the cord.

**Diagnosis.**—In the first stage this disease must be differentiated from the early condition of disseminated sclerosis, that is, the ataxic paralytic stage. Of course, when the classical advanced picture is present with nystagmus, intention tremor, scanning speech, etc., no special art is required. In the early stages, however, the similarity may be striking. Disseminated sclerosis usually comes on at an earlier age than does subacute combined sclerosis. If anæmia be present, it should be suggestive; and the subjective and objective sensory disturbances are much more marked and persistent in subacute combined than in disseminated sclerosis. The subacute progressive character of the one disease with its preterminal emaciation is of course quite different from the slow and prolonged course of the other.

In the later stages, with the flaccid paraplegia, the condition may simulate peripheral neuritis, myelitis, tumor of the cord, or tabes. The history of spastic gait, the involvement of the sphincters, the paralysis and loss of sensibility extending to the trunk and its nerve-root distribution, but especially the extensor plantar response, should be sufficient to make a diagnosis from multiple neuritis. Acute myelitis is of course excluded by the prolonged history. The absence of severe root pains and the distribution of the physical signs in unequal degree in the lower and upper limbs should exclude a tumor in the spinal canal.

The condition of flaccidity, anæsthesia, loss of deep reflexes, and complete incontinence often may suggest tabes. The history of lightning pains in the second stage may make it more suggestive. The previous history of spasticity, changing rapidly to flaccidity, the absence of the Argyll-Robertson

pupil, the great loss of motor power, the rapid muscular wasting and the loss of faradic excitability and the extensor plantar response, should prevent an error in diagnosis.

Enough has been said in describing the symptoms and course of the disease to make it evident that the *prognosis* is extremely bad. Any *treatment*, to be at all effectual, must be prophylactic. Attention to the general nutrition in all such debilitated and cachectic conditions is, of course, necessary. The patient should be at rest in bed in the early stages, to prevent as far as possible any exhaustion of neurones whose vitality and recuperative power are impaired by the presence of the hypothetical toxic matter in the circulation. Iron and arsenic are indicated to combat the anæmia, and the usual dietary and hygienic measures.

## FRIEDREICH'S ATAXIA. MARIE'S HEREDITARY CEREBELLAR ATAXIA.

Friedreich's ataxia is a disease, probably of abiotrophic nature, characterized by a slow and progressive incoördination of the four limbs. The disturbance begins in the lower limbs, extends to the trunk and upper limbs. and finally involves the tongue, larynx, and eyes. The disease generally affects several members of the same family. It is named after the famous Heidelberg professor, who first described the disease.[1] He looked upon it as an hereditary form of tabes dorsalis. One must remember that Duchenne, of Boulogne, had recently described his progressive locomotor ataxia, so that sclerosis of the posterior columns of the cord and tabes were synonymous in the minds of most neuropathologists of that time. Schultz, of Bonn, first drew attention to the associated sclerosis of the lateral columns and posterior horns. In Rutimeyer's publication,[2] in 1883, the view was advanced that we have in this disease a primary systemic combined sclerosis developing on an hereditary foundation.

In 1893 Pierre Marie[3] gave the name of *hereditary cerebellar ataxia* to a syndrome characterized by ataxic gait and cerebellar incoördination. He took this stand on the study of sixteen cases of familial disease, published by Fraser, Nonné, Sanger Brown, Klippel, and Durante. The main differences between this type and the type of Friedreich are, in the first place, a more definite hereditary influence in the former cases, although this is accounted for by the fact that the onset being much later, in fact, about the third decade, allows an opportunity for procreation, which is not possible in the patient suffering from the usual Friedreich type, in which the onset is before or about puberty; and secondly, the occurrence of ocular disturbances, the retention and even exaggeration of the knee- and ankle-jerks, and the absence of those trophic disturbances, the pes cavus and curvature of the spine, so characteristic of the Friedreich type. Marie considered that the syndrome bearing his name and Friedreich's disease were possibly different types of one and the same affection, due to an initial heredodegeneracy of the nervous system. He was of the opinion that the lesion in the cases of his type was localized more definitely in the cerebellum itself.

[1] *Virchow's Archiv*, 1863, xxvi, 391 and 433; 1864, xxvii, 1; 1876, lxviii, 145; 1878, lxx.

[2] *Virchow's Archiv*, 1883, xci, 106.

[3] *Semaine Médicale*, 1893, xiii, 444.

The appearance of cases which clinically are intermediate between the ordinary Friedreich type and Marie's hereditary cerebellar ataxia, and, moreover, the microscopic examinations in Sanger Brown's and similar cases, which reveal no particular affection of the cerebellum, but a condition very similar to that found in the ordinary spinal type of the disease, justify us in including Marie's type as a form of the same disease. In fact, no dividing line can be drawn between the two types either clinically or pathologically.

**Etiology.**—The disease is evidently what Sir William Gowers describes as an abiotrophic condition, a congenital inherited and inherent lack of vitality in certain parts of the nervous system. Other stigmata of degeneration are, as a rule, not lacking, and cases have been reported associated with conditions of infantilism, feminism, maldevelopment of the testicles, and so on. The congenital tendency is shown by the usual occurrence of the disease in several members of the same family, its onset at approximately the same age, and the similarity in the type of the developed disease in the different members affected. A neuropathic diathesis is present in most cases, as is frequently shown by the occurrence of hysteria, migraine, epilepsy, or insanity in collaterals or ancestors. Alcoholism in the parents was first insisted upon as an etiological factor by Friedreich, his first six cases being members of two families the fathers of which were drunkards and the children conceived in drunkenness. Quincke, Rutimeyer, Althaus, Everett Smith, Destree, and others have related analogous examples. Hereditary lues and tuberculosis may be predisposing causes. Consanguinity in the parents has been reported in many of the cases, and doubtless had some influence on the causation of the disease. Any fall, injury, or infectious disease may appear to determine the onset, and any cause acting as a depressant to the vital forces may produce a rapid advancement of the symptoms. The influence of sex is not noticeable in the occurrence of the disease, females being as frequently affected as males.

**Pathological Anatomy.**—In many cases of Friedreich's ataxia the cord has been found to be markedly smaller than normal. Thus, Newton Pitt[1] gives the following comparative measurements:

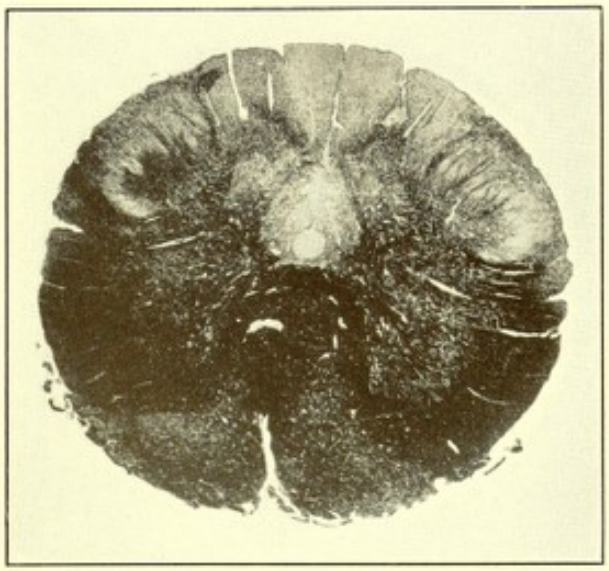
| Normal diameters of the cord. Millimeters. | Diameters of cord in a case of Friedreich's ataxia. Millimeters. | Distance of section level from upper limit of cervical region. Millimeters. |
|---|---|---|
| 13 x 10 | 13.0 x 8 | 0 |
| 15 x 9 | 12.0 x 8 | 33 |
| 16 x 9 | 11.0 x 7 | 39 |
| 12 x 8 | 10.0 x 7 | 51 |
| 10 x 8 | 9.5 x 7 | 67 |
| 10 x 8 | 8.0 x 7 | 81 |
| 13 x 8 | 8.0 x 8 | 107 |
| 12 x 8 | 8.0 x 7 | 127 |

This atrophy affects chiefly the posterior columns. Anomalies of structure may be present; thus Friedreich found a supplementary central canal in three cases, and Everett Smith[2] reports a similar condition in one of his cases. The cerebellum, too, has been reported as smaller than normal in some cases. The cerebrum is not affected. There is occasionally some thickening of the

[1] *Guy's Hosp. Reports*, London, 1887, xliv, p. 369.
[2] *Boston Medical and Surgical Journal*, 1885, cxiii, 361.

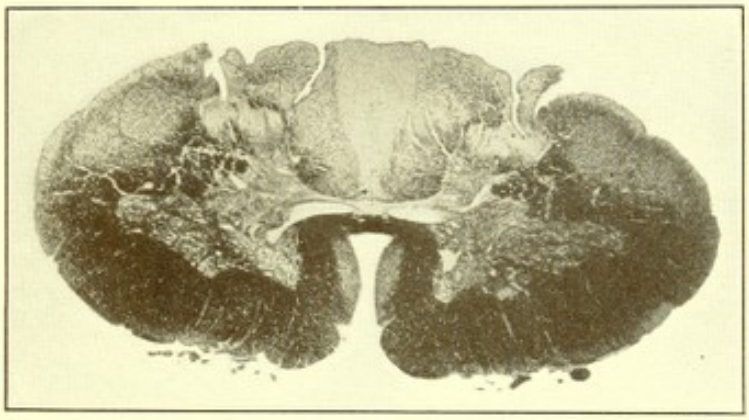
# PLATE X

FIG. 1



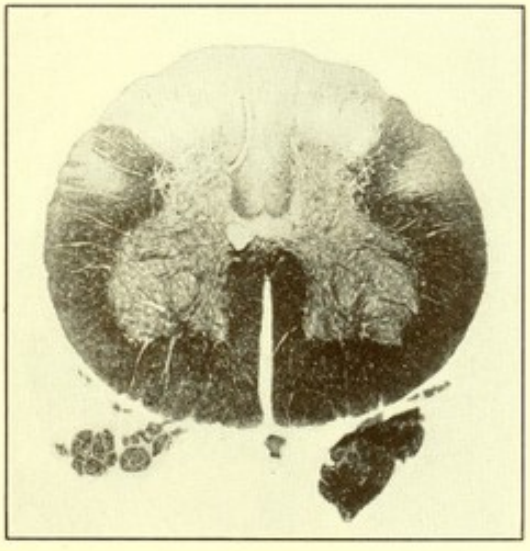
Friedreich's Ataxia. Lower medulla.

FIG. 2



Friedreich's Ataxia. Cervical region.

FIG. 3



Friedreich's Ataxia. Lumbar region.

membranes, particularly over the posterior aspect of the cord, sometimes, as in Mingazzini's case, more accentuated in the lumbar region.

On section of the cord, microscopically there is found a posterolateral sclerosis affecting the columns of Goll and Burdach, the posterolateral or direct cerebellar tract, and in many cases the anterolateral or Gowers' tract. The crossed pyramidal tract is constantly affected in some degree at least, and in a few cases the direct pyramidal tract has also shown degenerative changes. In the gray matter, cellular lesions in the column of Clarke have been invariably found in association with the degeneration of the direct cerebellar tract. Associated with atrophy of the muscles of the extremities, atrophy of the anterior horn cells and thinning of the reticulum has been described by some observers.

Lissauer's tract shows degeneration, which may be chiefly in the lumbar region. The posterior roots in most of the cases examined recently show a variable amount of degeneration. The spinal ganglia are, as a rule, practically normal, although atrophic degeneration of the nervous elements with connective-tissue hyperplasia has been found by some observers. The peripheral nerves show no constant change. The sclerosis of Goll's column, is, as a rule, complete, and can be followed up to its nucleus in the medulla, while that in Burdach's column is not so complete and varies in extent at different levels. In Mingazzini's case, and in those reported by Dumon, Philippe, and Oberthür, the lesions of the posterior roots seem to correspond more or less closely with the lesions in the posterior columns.

There are always some intact fibers left along the inner border of the posterior horns and immediately posterior to the gray commissure. Dejerine first called attention to the formation of what he termed "*tourbillons*," *i. e.*, small twirls of neuroglial tissue, in sclerosed areas of the posterior columns. These probably have no particular significance, and may be found in other long-standing sclerotic conditions in the cord. The sclerosis of the direct cerebellar tract follows its usual course, beginning in the lower dorsal region and gradually becoming attenuated toward the medulla. Gowers' tract shows an almost constant sclerosis, which according to Marie is invariably present if the disease be sufficiently advanced. The sclerosis of the crossed pyramidal tract is not as dense as in the posterior columns, and may be present only in the lumbar and lower dorsal regions, though in more advanced cases it may be visible even at the upper cervical level.

Marie, in his type of the disease, or what he termed hereditary cerebellar ataxia, basing his views on the findings in Fraser's and Nonné's cases, was of the opinion that the underlying pathological lesion here was an atrophic condition of the cerebellum. In Fraser's[1] cases the cerebellum was reduced to less than half its normal weight, its cortex was little more than half the normal thickness, very few Purkinje cells remained in it, and these were atrophied and shrunken. There was no macroscopic degeneration in the spinal cord. In Nonné's[2] case the cerebellum was abnormally small, but no further lesion was demonstrable. Later autopsies, however, performed by Meyer and by Barker on three cases of Sanger Brown's remarkable series, have not altogether confirmed these observations. Adolph Meyer[3] found no

[1] *Glasgow Medical Journal*, 1880, i.
[2] *Arch. f. Psych.*, 1891, xxii, 283.
[3] *Brain*, 1897, xx, 276.

circumscribed cerebellar lesion; Barker[1] found no marked diminution in the size of the cerebellum, although it, with the cord, medulla, and pons, looked small in proportion to the cerebrum, which itself was small. The microscopic study in both cases revealed a marked degeneration in the gray and white matter of the spinal cord, medulla oblongata, and cerebellum. The degeneration involved chiefly the nerve cells and fibers of the centripetal paths, including the posterior columns, the dorsal nucleus of Clarke, the direct cerebellar tract with its continuation in the restiform body. There was practically no change in the cerebellum beyond a possible diminution in the number of cells in the nucleus dentatus and nucleus fastigii.

**Symptoms.**—Following the order taken up in the preceding section, that type of the disease described by Friedreich will be first discussed, then the so-called hereditary cerebellar ataxia of Marie, and finally, attention will be directed to the numerous intermediate forms bridging over the interval between these clinical types.

In the type of Friedreich, the disease commences at an early age, usually between four and ten, or at puberty; in exceptional cases the onset may be later. It is probable that in many cases the first symptoms pass unobserved until after some infectious fever or injury of some kind, when a slowly developing ataxia of the lower extremities is noticed. The child walks somewhat hesitatingly, with legs rather far apart, and attempts to balance himself with the arms; the feet are brought down in rather a stamping fashion, the paces are irregular, with some deviation in the line of march, the picture resembling somewhat a partially intoxicated person. The child is not steady on his feet, but falls more readily than other children. In standing alone there is usually some unsteadiness, and this, as a rule, is not increased by closing of the eyes; so that, although Romberg's sign has been reported as present in some cases, it is certainly not typical of the disease.

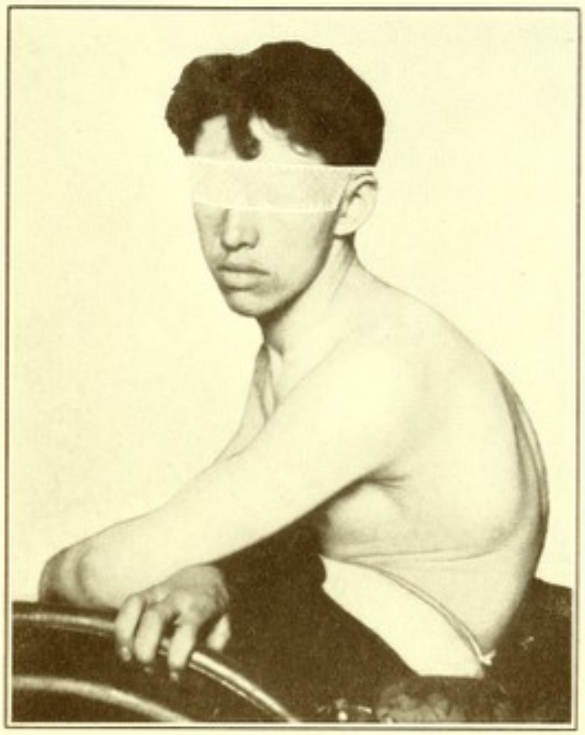
On further examination in this early stage the cranial nerves are not found affected; speech may in exceptional cases be somewhat slow and drawling quite early in the course of the disease, but, as a rule, the defects in speech and nystagmus are observed later. There is no atrophy or loss of power to be made out in the muscles. Their tone is usually about normal; there is no impairment of the sense of position of the muscles, nor can any loss of any other form of sensibility be demonstrated. The knee-jerks in this early stage are most commonly lost, but are often retained and may even be active. The extensor plantar response (Babinski) may or may not be present. There is very often, even at this early stage, some scoliosis of the spinal column, and there may be evidence of the beginning of the club foot so typical of the fully developed disease. The bladder functions are not disturbed, nor do they suffer, as a rule, during the course of the disease.

Gradually the disease progresses, and in the course of a few years the typical picture is developed. The ataxia affects the muscles of the upper extremities, trunk, and neck. At first it may be observed associated with voluntary movements only. If the child has already learned to write, it is noticed that his penmanship, instead of improving, becomes awkward and clumsy. Articles are knocked over in the attempt to grasp them. The movements are associated with a swaying, sometimes almost choreiform,

[1] *University of Chicago, Decennial Publication*, 1903, x.

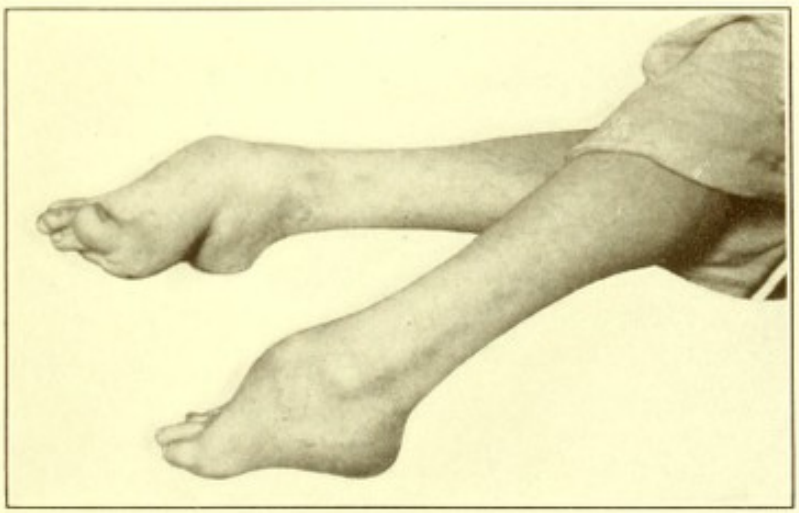
# PLATE XI

FIG. 1



Friedreich's Ataxia, showing the Spinal Curvature.

FIG. 2



Friedreich's Ataxia, showing the Typical Deformity of the Feet.

jerkiness, until finally the patient may not be able to convey a glass of water to his mouth. The ataxia is of the nature of a motor incoördination, due to the imperfect fusion of simple contractions, which compose voluntary movements. The disturbance of speech and the nystagmus are of a similar nature. A condition of static ataxia also develops, caused by this same defect in the permanent, regular, harmonious innervation of those muscles and their antagonists, which are necessary for the maintenance of equilibrium and the upright position of the body. This may be first noticed in the muscles of the neck, causing a jerky, irregular, almost tremor-like movement of the head. Later, as the disease progresses and the trunk muscles become affected, the patient has difficulty in maintaining the sitting position without support, on account of the irregular movements of the trunk on the pelvis. Associated with the ataxia there may be a certain amount of muscular weakness, but this is not usual except in advanced cases, when the patient becomes confined to a chair or bedridden. In this stage and sometimes even earlier, atrophy of the muscles of the extremities may be present, associated with contractures and malposition. Faradic excitability of the muscles may be lost and the reaction to galvanism diminished. The writer has observed this also in the muscles of the lower extremities where there was no apparent atrophy.

The cerebral functions are commonly not impaired, although the general facial expression may suggest extreme stupidity; this is more often due to simple apathy and indolence. Impulsive laughter has been described in some cases, and the writer has a patient under observation at present who shows this to a marked degree. Excessive salivation has been known to occur. Speech becomes slower, hesitating, and jerky; articulation is imperfect, some syllables are prolonged, others forced out quickly in an explosive manner, as if the ataxia had affected the muscles of articulation. In a typical case the optic nerves are not affected, nor is the Argyll-Robertson pupil a symptom. Nystagmus, associated with lateral movements of the eyes, is not infrequent. Disturbances of respiration may be caused by the involvement of the abdominal muscles in the ataxia, and if the medullary centres be affected a definite dyspnœa results.

The sensory system is, as a rule, unaffected. Lancinating pains were present in some few cases reported, but are not common. Cramp-like pains in the limbs are perhaps rather more frequently complained of. Only in exceptional cases is there any impairment of sensibility, and then usually late on in the disease and of an indefinite incomplete type, being confined to the more distal parts of the extremities. There is rarely any loss of the sense of position of the muscles, although it has been observed in exceptional cases (Oppenheim). Visceral crises never occur. The tendon-jerks gradually diminish, disappearing first in the lower extremities. The presence of the extensor plantar response (Babinski), with the absence of the knee and ankle jerks, is typical.

Vasomotor paresis occurs, so that in many cases the feet and ankles are congested, reddish blue in color, and clammy. Lateral curvature of the spine is almost always present, commencing early in the disease and often reaching an extreme grade. A peculiar deformity of the feet is commonly seen, namely, a condition of pes equinovarus, with hyperextension of the proximal phalanx and flexion of the distal phalanx of the great toe. In families affected with the disease this sign has in some cases been what they

themselves looked for, knowing from hard experience what the ultimate outcome would be when it once made its appearance.

In *hereditary cerebellar ataxia* the same familial character is present, but, as has been remarked above, the hereditary tendency is more evident on account of the disease commencing only in the third or fourth decade, and thus permitting the victims to marry and produce offspring. In the celebrated series reported by Sanger Brown,[1] twenty-three members of one family, spread over four generations, were affected. Females are perhaps slightly more frequently affected than males; and Sanger Brown remarks that the disease was inherited more frequently through the girls than through the sons of the family. Occasionally the disease may skip one or even two generations, appearing perhaps in several members of the third.

The disease commences with a slowly progressive ataxia, more or less marked in walking and standing; lancinating pains in the legs may be present. After some time the ataxia affects the upper extremities, and soon disturbances of speech are noticed, and the patient complains of difficulty of vision. Tendon-jerks are increased, instead of being diminished or lost, as we find in the Friedreich type, and occasionally other evidences of spasticity in the lower extremities are seen. The extensor plantar response is present. Only in a few cases has there been any disturbance of cutaneous sensibility. Frequently mental weakness develops in the course of the disease. Disturbance of deglutition and impairment of the control of the sphincters occur only very rarely. The disease is essentially progressive, but may present remissions. Some intercurrent disease, probably years after the onset, is the usual cause of death.

The disturbance of function of the lower extremities is absolutely analogous to what is observed in Friedreich's disease. The gait and station are similar to that already described, starting gradually, progressing steadily, increasing with fatigue. Frequently in the early stages the unsteady staggering gait leads to the accusation of inebriety. Closing of the eyes does not increase the unsteadiness in standing to any extent.

The upper extremities are affected to a much less degree and later in the course of the disease. As in Friedreich's type, it is the more delicate movements that are most interfered with in the early stages; later even the gross movements may become equally affected. Static ataxia also develops; there is no muscular weakness.

The signs of spasticity of the lower extremities, on which the differentiation of this type from Friedreich's is based, to a great extent consist in increased knee-jerks and in some cases ankle clonus, contrasting with the absence of knee-jerks in the typical picture of Friedreich. The extensor plantar response (Babinski) is present. The tone of the muscles is increased, in some cases to such a degree that it can be overcome only with difficulty.

Apart from the lancinating pains occasionally present in the earlier stages, there is seldom any disturbance of sensibility. The sense of position in the muscles was disturbed in Klippel and Durante's cases, but, as a general rule, it is unaffected.

In the domain of the cranial nerves ptosis is sometimes present, nystagmus is a common symptom, and frequently one finds paralysis of one or other of

[1] *Brain*, 1892, xv, 250.

the external ocular muscles. The pupils often show a diminished or even an abolished reaction to light and occasionally disturbance of the reaction to accommodation. The visual field often shows some diminution in size, and color vision may be impaired. Primary optic atrophy is not uncommon. The disturbance of speech is similar to that already described in Friedreich's type. A peculiar overaction of the muscles of the face, especially marked on the attempt to speak, is frequently observed. Spinal curuature and club foot form no part of the symptoms.

Numerous forms intermediate between these two types have been observed. Thus, for instance, Bonnus reported a case, otherwise conforming to the classical description of Friedreich's disease, in which the onset of symptoms began after the twenty-fifth year. In a brother and sister, reported by Baumlin, typical cases of the cerebellar ataxia type, with the ataxia, pains, convergent strabismus, optic atrophy, and increase of the patellar and other tendon-jerks, the onset in the boy was during childhood, in a girl at the age of fifteen years. Similar cases to these have been reported by Rossolimo and Lennmalin.

While scoliosis and club foot are not, as a rule, included among the symptoms of cerebellar ataxia, they do sometimes occur (Londe, Muira, Rossolimo).

In typical cases of Friedreich's disease the knee-jerks are abolished. Many cases have been observed, however, in which in the early stages they have been preserved, and disappeared only later in the course of the disease, presumably when the degeneration in the posterior fibers affected the part of the cord interested.

The two types cannot be differentiated clinically by the presence or absence of signs of spasticity in the lower extremities. Erb has reported two sisters whom he regards as examples of Friedreich's disease in spite of normal knee-jerks. Hodge has described three cases of the same type, all presenting marked increase in the knee-jerks. Allen Starr found in one family two of the affected members with loss of knee-jerks, while in the youngest, with quite similar symptoms, the knee-jerks were rather increased. Brock and Tressider report similar instances in which the knee-jerks were present in some and absent in others of the affected members of a family, and it is by no means infrequent. Paralysis of the external muscles of the eye in otherwise typical cases of Friedreich's disease has been observed by Gowers, Mendel, Omerod, Jaffroy, Anderson, Starr, and others. Optic atrophy was present in the case of the two brothers reported by Cohn.

It is evident that no fast dividing line can be drawn between the various forms of this disease. We have here an abiotrophic condition in which the localization of the degenerative process determines the clinical signs in the individual cases.

**Diagnosis.**—In the early stages it is possible that this disease may be mistaken for chorea, especially if there happens to be only one member of the family affected. The family history, curvature of the spine, absent knee-jerks, with the extensor plantar response, should leave no doubt, and of course the progress of the disease renders such an error impossible for any length of time.

*Tabes dorsalis* may be suggested by the ataxia and the loss of knee-jerks, but as a matter of fact there is little danger of such a mistake. The ataxia is quite different. In tabes it is due to a loss of the sense of position of the

muscles; in Friedreich's it is entirely a motor incoördination, not increased to any extent in the dark or when the eyes are closed. The gait is quite distinctive; there is not the same overaction of all the movements in Friedreich's ataxia. The difficulty is more in maintaining the equilibrium than in governing the action of the limbs, and the patient does not watch his feet so intently. The age of onset of the disease, the history of antecedent lues, the disturbance of the bladder functions, the lightning pains, girdle sensations, and paræsthesias in tabes, with objective sensory disturbances and the Argyll-Robertson pupil, leave no doubt about the diagnosis. Scoliosis and the characteristic club foot form no part of the picture of tabes. Visceral crises, although reported in one case,[1] cannot be included among the symptoms of Friedreich's disease. The mental condition and absence of Argyll-Robertson pupil exclude juvenile general paresis.

The differential diagnosis between *disseminated sclerosis* and those sporadic cases of Friedreich's ataxia, which occasionally occur, is sometimes difficult, especially when, as often happens, the knee-jerks are not lost in the very early stages. Of course, the incidence of the disease in several members of a family will give the clue immediately, and spinal curvature, club foot, and cerebellar gait will settle the question. Especially in sporadic cases of the cerebellar ataxic type will the differential diagnosis be difficult. The disturbance of volitional movement, the nystagmus, the speech defect, the increased knee-jerk, and extensor plantar response will suggest disseminated sclerosis most strongly. The difficulty will be much increased if the patient be already in a bedridden condition when he first comes under observation. The muscular incoördination of the upper extremities with no sensory disturbance is similar in the two diseases; increased reflexes and extensor plantar response are common to both. In each there is disturbance of speech, nystagmus, ocular paralyses, and not infrequently optic atrophy. In the cerebellar ataxia, scoliosis and club foot are only exceptionally seen, and never in disseminated sclerosis. The gait, however, is distinctive. In sclerosis it is spastic, progression is direct enough, the paces regular but short, the legs stiff, and the toes, clinging as it were to the floor, are apt to catch on any little obstacle or unevenness. The history, then, of staggering like a drunken man should be suggestive.

The steady progression of the disease in hereditary ataxia is quite different from the intervals of remissions even with some improvement followed by exacerbations, which one sees in the disseminated form. In the latter, disturbances of bladder functions are more common. Absence or slowness of the reaction of the pupil to light is more commonly seen in hereditary ataxia.

The appearance of the movements, and the family character of the disease in Huntington's *hereditary chorea*, may also give rise to some uncertainty. The presence of nystagmus, ocular palsies, optic atrophy, and especially the state of the reflexes, should remove all doubt. In hereditary chorea mental aberration is the rule sooner or later.

**Prognosis.**—In either type of the disease the prognosis is bad. Its steadily progressive character has been sufficiently emphasized. Sooner or later the patient becomes bedridden, but he may live for twenty or thirty years, eventually being carried off by some intercurrent disease.

[1] Bramwell, *Brit. Med. Jour.*, 1897, ii, 896.

**Treatment.**—Drugs have no effect on the disease process. Reëducative exercises, as in tabes, may be of some benefit. More might be looked for from the prophylactic treatment of the younger and unaffected members of the family; proper hygienic measures and every care should be taken to improve and maintain their general health.

## COMBINED SCLEROSIS OF THE CORD CAUSED BY VEGETABLE POISONS.

**Lathyrism.**—Lathyrism, according to Sheube, is a disease with a spastic spinal paralytic course, of the nature of an intoxication which is attributable to poisoning by various kinds of grass pea or Papilionaceæ lathyrus. The disease has been recognized for many centuries, Hippocrates and Galen having noted the "crurum impotentia" of those who lived on "ervum," a vegetable analogous to lathyrus. So widespread was the disease in 1671 that the use of bread made from this grain was forbidden by an edict of George, Duke of Würtemburg. In 1873 Cantani reported several cases, and gave the name of *lathyrism* to the condition.

**Etiology.**—The disease is endemic in certain parts of France, British India, and Algiers. Only the poorer farm laborers are affected, as a rule, and the incidence is much increased in famine years, the poorer classes being forced to live on the cheapest sort of food, such as Lathyrus sativus, L. cicera, or L. clymenum. Men are more frequently affected than women.

**Symptoms.**—The disease commences with general weakness and trembling of the legs, and, according to Brunelli,[1] if the food be not changed a condition of spasmodic tabes develops in the course of a month or two; the onset may in some cases be even more abrupt. It is characterized by spasticity of the lower extremities, bladder disturbance, paræsthesia, and blunting of cutaneous sensibility. There is no muscular atrophy, and no involvement of the cranial nerves. The patient walks with the aid of a long two-handed staff in a typically spastic manner.

Pigs, dogs, and rabbits are also occasionally affected, the disease showing itself by paralysis of the posterior extremities. Birds appear to be immune.

The underlying pathological lesion is a posterolateral sclerosis, but the pathology is still obscure.

**Pellagra.**—The name was first applied by Frapolli in the beginning of the eighteenth century (Italian "pelle agra," rough skin). It is endemic in Italy and Egypt among the poorest classes. It exists also in most of the other countries of southern Europe; and in the western hemisphere it has been observed in Mexico, Brazil, the Argentine Republic, and within the last two years has appeared in the southern part of the United States to an alarming extent. It has been reported from North Carolina, South Carolina, Maryland, Georgia, Alabama, and Texas. E. J. Wood was able to collect 200 cases reported from these States alone. The disease was first described by Cassale in the beginning of the eighteenth century. In 1906 there were said to be 72,000 individuals in Italy infected with pellagra. Bouchard, in 1864, first called attention to the changes in the spinal cord in these patients.

[1] *Trans. Seventh International Congress*, London, vol. ii.

**Etiology.**—In spite of long study and observation this is still obscure, although it is generally conceded that it is in some way associated with the consumption of maize. The first appearance of pellagra and the later spread of the disease is said to have followed the introduction and culture of maize in Spain, Italy, and the other countries of southern Europe. The disease is found to be endemic only in those countries where maize is grown and extensively used as an article of food, and is not found where maize is not used as a food.

There are many theories. Lombroso holds that under the influence of parasitic growths (bacteria and moulds) the corn undergoes certain changes with the formation of one or more toxic substances of a chemical nature and the use of such maize as food produced pellagra. Various toxic substances have been abstracted from spoiled maize, but so far none can be said to be the specific cause of the disease. Then there is a theory that pellagra is a specific infection derived from the maize, the infectious agent being either a mould or a bacterium. Ceni, in 1902, believed he had found the source of the infection in two moulds, the Aspergillus fumigatus and flavescens, and more recently in working with Besta he discovered toxic properties in the Penicillium glaucum. Tizzoni has also described a streptobacillus which he isolated from the blood and organs, and which he found pathogenic for rabbits and guinea pigs. These observations still require confirmation.

**Pathology.**—There is a congestion and thickening of the leptomeninges, with sometimes the formation of osseous plaques. The cord itself, at least in the very advanced cases, shows a posterolateral sclerosis involving the columns of Goll and Burdach and the crossed pyramidal tracts, the direct cerebellar tract escaping. In Sandwith's[1] cases Batten found degeneration in the posterior columns only, and this had evidently commenced in the posterior roots. The anterior horn cells show degenerative changes in some cases. It seems probable that when the symptoms have been present for a relatively short period the posterior columns only are affected, but when the toxin has been at work over a prolonged period the pyramidal tract also shows evidence of exhaustion.

**Symptoms.**—The onset of the disease is usually with digestive disturbances, diarrhœa, tenesmus, and abdominal pain, and very soon an erythematous roughness of the skin is noticed, appearing first on the hands, feet, and neck. The action of the chemical rays of the sun has some influence on the appearance of this eruption, which has been compared to erythema exfoliativum, but probably this is only an indirect effect attributable to the atrophic condition of the skin. This skin eruption is always most evident in early spring, subsiding in the winter months; this recrudescence, according to Ceni, corresponds to the period of greatest natural development of the aspergillus, which he regards as the essential cause.

The mouth is sore and red, the tongue denuded and fissured; the lower extremities become extremely spastic, with increased tendon-jerks and the extensor plantar response. There is some loss of control of the bladder and bowels. Pain and tenderness over the vertebral column are not uncommon. Burning pains in the hands and feet are often very severe. A maniacal or,

[1] *Jour. of Path. and Bact.*, 1901, vii, 460.

more commonly, a melancholic mental condition frequently necessitates confinement in an asylum.

The course of the disease is, as a rule, chronic, lasting fifteen or twenty years, but occasionally an intensely acute variety known as *typhus pellagrosus* is seen. Searcy,[1] in 1907, published a report upon an epidemic of the acute type of the disease, occurring among the inmates of the Mount Vernon Hospital for Colored Insane, in Alabama, United States of America, with the high mortality of 64 per cent. The outbreak, in which eighty-eight of the patients were affected, was traced to the use of diseased meal.

**Treatment.**—The prophylactic treatment among such utterly illiterate people seems impossible; nevertheless the Italian government has numerous asylums scattered through the infected regions, where the victims are treated and the younger members are educated by enthusiastic Sisters of Mercy. Ceni believes in the use of serum of immunized animals, and hopes for good results. Babes claims to have obtained excellent and rapid results, not only in the skin condition, but in the mental state as well, by the use of atoxyl.

[1] *Jour. Amer. Med. Assoc.*, 1907, xliv, 37.

# CHAPTER IV.

## SCLEROSES OF THE BRAIN.

By EDWIN BRAMWELL, M.B., F.R.C.P. (Lond. and Edin.).

**Introduction.**—An increase of the neuroglia and mesodermic connective tissue of the central nervous system occurs in a variety of pathological conditions. Proliferation of the neuroglia is met with, for example, in the systemic disorders, whether these be of abiotrophic, ischæmic, or toxic origin. A similar neuroglial increase accompanies the systemic degenerations consequent upon interruption of the nerve fibers in some part of their course. A local proliferation of the interstitial tissue may be observed in the neighborhood of inflammatory lesions and old foci of hemorrhage, while a cortical sclerosis is found post mortem in some cases of infantile hemiplegia as a sequel, in the opinion of Strümpell, to a primary acute encephalitis. The neuroglial proliferation in the instances mentioned is either of compensatory origin, the tissue multiplying in order to occupy the place left vacant by the disappearance of the nerve elements, or secondary, in the sense that the change constitutes a later stage in the course of an inflammatory process.

A group of affections remains for consideration, the scleroses proper of the nervous system, in which the neuroglial proliferation, if not a definite primary process, is at least the dominant feature. Disseminated sclerosis, one of the commonest diseases of the nervous system, is by far the most important of these. Diffuse sclerosis, a very rare affection, belongs naturally to this group. Pseudo-sclerosis, another disease of great rarity, may also be included, since it closely resembles clinically the two last-mentioned affections, while pathologically it is regarded by some authorities as an early stage of diffuse sclerosis. Miliary and tuberose scleroses are pathological entities of little clinical interest which call for brief notice.

### DISSEMINATED SCLEROSIS.

**Synonyms.**—Multiple sclerosis; insular sclerosis; sclérose en plaques.

**Historical.**—Cruveilhier (1835) and Carswell (1838) were the earliest writers to figure the gross morbid appearances, while Frerichs (1849) appears to have been the first observer to make an *intra vitam* diagnosis. Valentiner, in 1856, collected 15 cases, and indicated the long duration of the disease, the early involvement of the lower extremities, and the tendency to remissions and exacerbations. Charcot and Vulpian established the clinical picture of the so-called "classical type" by the recognition of the symptomatic triad —scanning speech, nystagmus, and intention tremor. The observation of Charcot, that the axis cylinders commonly remain intact, was of great pathological importance. Moxon's record of 8 cases in 1875 was the first British contribution to the subject. Great advances were made in early diagnosis during the last two decades of the nineteenth century, with a

result that *formes frustes*, which had previously escaped recognition, were found to be more common than the classical type. Uhtoff was the first to indicate the frequency of the characteristic changes in the optic disks. To Thomas Buzzard is due the credit of insisting on the differentiation from certain types of hysteria which closely resemble this disease. Babinski, by the discovery of the "toe phenomenon," has given us an important aid in differential diagnosis.

Of recent years the disease has received much attention. Sachs (1896) and Borst (1904) have published valuable critical digests; Hoffman (1903) has recorded 100 cases from the Heidelberg clinic, with three autopsies; Byrom Bramwell (1903) has analyzed 110 cases which have come under his personal observation; while in 1904 Eduard Müller's exhaustive monograph appeared.

**Frequency.**—Disseminated sclerosis is one of the commonest organic affections of the nervous system. Its frequency appears to vary in different countries. In Scotland it seems to be much commoner than in the United States (Byrom Bramwell, Jelliffe). E. W. Taylor (1906), who has collected 13 cases with autopsies from the American literature, is of opinion that even in the States it is a common disease, though far fess frequent than tabes, and is probably often overlooked. Among 1000 consecutive cases of nervous disease recently seen by Byrom Bramwell in his private practice, there were 32 cases of disseminated sclerosis and 34 of tabes. Strümpell observes that in a mixed population, to judge from his Erlangen and Breslau material, disseminated sclerosis is distinctly commoner than tabes. E. Müller, who has analyzed 80 cases of disseminated sclerosis from Strümpell's Erlangen clinic, found that during the same period syringomyelia had been diagnosed on 29 occasions. Schlesinger, of Vienna, actually finds that in his experience syringomyelia is commoner than disseminated sclerosis; it may be, however, that this author's widely recognized reputation in connection with the former disease accounts for his unusual experience. In this country, unless many cases of syringomyelia are overlooked, which seems scarcely probable, syringomyelia is relatively to disseminated sclerosis very much rarer than these statements suggest.

**Etiology.**—The causes are most obscure, and no factor common to a large proportion of cases has hitherto been demonstrated, although in individual instances indications of an etiological relationship either predisposing, exciting, or augmentative are discernible.

**Sex.**—There is a surprising difference of opinion as to the incidence of sex. No doubt this is, in part, at least, to be explained by the hospital accommodation at the disposal of various observers. Thus, among 34 cases observed by Charcot there were only 9 males. In Byrom Bramwell's series of 110 cases, 67 were females. Bruns and Berlin also record a preponderance in the female sex. On the other hand, Uhtoff, among 100 cases, met with 67 males, while in Frankl-Hochwart's series of 206 cases there were 140 males. Probst, Pierre Marie, and Krafft-Ebing also found in their experience that males outnumbered females. Among E. Müller's 75 Erlangen cases there were 35 males, while of 81 cases collected from the literature by this author, in all of which the diagnosis was confirmed post mortem, there were 40 males and 41 females. Tredgold, in 1904, collected 373 recent cases from the literature; 200 of these were males as against 173 females. Thus it would appear that the sexes are almost equally liable.

**Age.**—The first symptoms usually develop during early adult life. E. Müller found that in 44 per cent. of his cases the earliest signs appeared during the third decade. Byrom Bramwell met with 55 per cent. commencing during this period, while in 85 per cent. of his cases the disease first manifested itself between the fifteenth and thirty-fifth years. It is very unusual for the disease to begin after forty years of age.

Instances have been reported in which some of the symptoms appeared to date back to infancy. Schupfer,[1] who in 1902 collected 59 cases reported as commencing in childhood, considers that the diagnosis was often unwarranted. Müller has discussed the data exhaustively, and comes to the conclusion that a true multiple sclerosis in childhood identical with that occurring in the adult has yet to be demonstrated.

**Infections.**—Kahler and Pick indicated in 1879 that insular sclerosis often develops after an acute infective disease. Pierre Marie in his *Diseases of the Spinal Cord* (1892) strongly advocates this view. Of 25 cases which appeared to follow upon an infective malady, typhoid fever was in 11 instances the preceding disease. The apparent onset of the first symptoms after influenza, measles, scarlet fever, diphtheria, smallpox, cholera, etc., has been observed. To account for this, Marie supposes that its cause is "the result of one of those combined infections which are so frequent during the course of the different infective diseases." It cannot be denied that an infective malady sometimes appears to act as an augmentatory cause, yet it is extremely doubtful whether it alone is ever the essential factor. The writer's experience has convinced him that in a considerable proportion of these cases, in which the patient attributes his illness to one or other of these diseases, careful inquiry demonstrates that undoubted symptoms of the affection have been previously in existence. The prevalence of influenza during the last two decades has been regarded as possibly accounting for the increase in the number of cases of disseminated sclerosis met with during the same period. Advance in diagnosis offers an alternative explanation. Finally, although it is improbable that the infective diseases actually determine the incidence of disseminated sclerosis, it must be admitted that a very pronounced increase in the severity of the symptoms is a common sequel to these disorders.

Syphilis plays no causative role in relation to disseminated sclerosis. Proof of this assertion is found in the rarity with which a history of syphilis or evidence of that disease is forthcoming (there was a history of syphilis in only 3 of the 206 cases analyzed by Frankl-Hochwart), the fact that the sexes suffer almost equally, and the circumstance that among a population in which syphilis and the parasyphilitic affections are common, there is no corresponding increase in the cases of disseminated sclerosis. The writer's experience in this connection is of some interest. In the Out-patient Department of Leith Hospital, which draws its material from a seaport town of 80,000 inhabitants, during a period of four years, he met with 62 cases of tabes as compared with 2 cases of disseminated sclerosis.

**Trauma.**—In 1901 Gumbrecht[2] collected 54 cases in which it was claimed that trauma was of etiological importance. Grossmann[3] has recently

[1] *Monatsschr. f. Psych. u. Neurol.*, Band xii, Heft 1 und 2.
[2] *Inaug. Diss.*, Leipzig.
[3] *Deutsche med. Wochenschr.*, 1905, No. 41.

reviewed the literature. Von Leyden, Hoffmann, and a number of other observers have recorded cases in which they regarded injury as the cause. Strümpell, Marie, and others, on the contrary, doubt whether trauma ever acts as the actual cause. E. Mendel held, and with this opinion the writer is in agreement, that injury may act as an exciting cause in an individual who is predisposed.

**Metallic Poisons.**—Oppenheim has published observations which suggest that poisoning by lead and other metals may play a role. Among 28 cases in which the diagnosis was certain, and in which special inquiry was made into this point, in 11 a history of exposure to possible metallic poisoning by lead, copper, zinc, and other metals was obtained. The observations of subsequent writers have failed to demonstrate this relationship although Embden has described very similar symptoms in manganese workers.

**Hereditary Predisposition.**—Eichorst and Lenot have recorded cases of disseminated sclerosis occurring at an early age, in each instance the mother of the patient having suffered from that disease. In Eichorst's case, which was examined post mortem, the pathological findings are not, however, altogether conclusive. There is no undoubted instance on record, so far as the writer knows, in which a diagnosis of family disseminated sclerosis has been verified by autopsy. Some of the cases referred to have undoubtedly belonged to the category of the familial spastic paraplegias. A special family tendency to nervous disease is not usually met with in this disease.

**Social Conditions.**.—The fairly well-to-do, lower and middle classes are, in the writer's experience, at least as liable to the disease as are the lowest grades of society, if not more so. The subjects of disseminated sclerosis are indeed often healthy, well-marked girls brought up in the country.

**Emotion.**—Mental shock, fright, worry, or grief are sometimes blamed by the patient as the cause of the symptoms, which certainly appear to be aggravated by psychical disturbances.

The first symptoms sometimes appear after undue fatigue or exposure to cold.

From what has been said it will be seen that the onset has been attributed to a variety of exogenous influences. The very multiplicity of these apparent causative factors, the circumstance that each only occurs in a small proportion of cases, and their frequency apart from the disease in question would suggest that they are most probably all of secondary importance.

**Pathology.**—Disseminated sclerosis is characterized anatomically by placques or islets of disease scattered haphazard throughout the central nervous system. The cranial nerves are often invaded, while similar lesions have also been found in the spinal nerve roots. A classification into cerebral, spinal, and cerebrospinal types, although of clinical value, cannot in the pathological sense be strictly adhered to, since it seldom if ever happens that the patches are confined either to the brain or spinal cord.

The pia mater has been sometimes described as unduly adherent. A slight degree of atrophy of the cerebral convolutions has been occasionally observed. In one or two exceptional instances, as in a case recently examined by the writer, the unusually small size of the spinal cord attracted attention. Glistening patches of variable size and shape and of a grayish color are in some cases to be seen shining through the pia mater which covers the surface of the cord, and on section these are seen to correspond with diseased placques situated in its substance.

Upon section, islets of a gray or grayish-pink color are, as a rule, readily recognizable. It may be, however, that the diseased patches are not to be detected until the tissue has been placed in Müller's fluid or chrome alum. A slight degree of shrinking of the cord is sometimes observed in the neighborhood of patches of old standing, yet the contour of the transverse section is seldom modified in a striking manner thereby. The islets of disease are often firmer than the surrounding tissue; indeed, they may have almost the consistence of india-rubber. This fact may be demonstrated by attempting to pass a horse hair through the affected region. Sometimes the placques are distinctly softer than the normal cord. Both firm and soft patches may co-exist in the same case.

The patches vary greatly in size and shape. In the brain they may be as large as a walnut; the majority, however, are very much smaller, and many are only recognizable on microscopic examination. The greater part of the transverse section of the spinal cord may be involved at one level, while a section a centimeter above or below may present an almost healthy appearance. A patch involving a large area may sometimes be traced through several segments. The patches may be wedge-shaped, rounded, oval, or quite irregular in contour; indeed, there is no characteristic type. It is most unusual to find a patch of any size which does not extend to the periphery of the cord. The one situation in which the writer has found a notable exception to this rule is in the posterior columns. In two cases a large oval patch extended through several segments in this region. Personal observations from an examination of seven cases lend no support to Obersteiner's statement that the lumbar region of the spinal cord is less commonly involved than the parts above this level.

It has been affirmed that certain parts of the transverse section are more liable to suffer than others. Thus most observers hold, and with this conclusion the writer is inclined to agree, that the white matter is especially implicated. E. W. Taylor,[1] from a study of eight cases, concludes that both gray and white matter are irrespectively involved. Ribbert asserts that the gray matter often offers a barrier to the spread of the diseased area. The writer's personal observations are in accord with those of Dejerine and E. W. Taylor, who failed to find evidence in support of this conclusion.

Bourneville and Guerard regarded the long tracts, Ziegler and E. Müller the posterior columns, as the regions of predilection. Obersteiner and others have noted a striking tendency to symmetrical affection of the spinal cord, medulla, and pons. This is, however, far from a constant feature. Williamson and Rossolimo have reported cases in which the distribution of the patches corresponded to that of the bloodvessels. In not one of the seven cases examined by the writer was it possible to assert that this was so.

In the brain the basal ganglia and the neighborhood of the ventricles, the corpus callosum, the pons (Strümpell), centrum ovale (Gowers), and the neighborhood of the olives are favored sites. For long it was supposed that the cerebral cortex was always spared. Oppenheim, E. W. Taylor, and Sander have shown that patches in the cortex are by no means uncommon. Islets of disease are comparatively rarely met with in the cerebellum. Weigert, however, has drawn attention to diffuse changes consisting in an increase of the tangential and radial fibers in this situation.

[1] *Journal of Nervous and Mental Disease*, June, 1906.

For long it has been known that patches of sclerosis may occur in all the cranial nerves in some part of their course. The optic nerve and tract appear to be specially favorite situations. Changes in the anterior and posterior spinal nerve roots have been noted by several observers. On the other hand, it is doubtful whether the peripheral spinal nerves are ever implicated, although it must be admitted they appear to have been seldom systematically examined.

**Histology.**—The two most striking features of the diseased areas are the disappearance of the myelin sheaths and the increase in the neuroglial tissue. The consistence of the patches varies with the degree of neuroglial proliferation. Tredgold[1] divides the islets of disease into three varieties, hard, soft, and intermediate. Hard islets occur chiefly in the cord, and consist of a dense interlacement of thickened neuroglial fibers with very few glia cells. Nerve fibers are practically absent, although occasionally a naked axis cylinder is met with. There are no products of degeneration and but few bloodvessels. Soft islets, which were confined to the brain in Tredgold's three cases, usually had a well-defined margin, often surrounded by a zone of leukocytes, and consisted of a loose reticulum containing a semifluid material. They are generally devoid of nerve cells or fibers, there is no neuroglial proliferation, and usually no degeneration products. The vessels are often much congested. Intermediate islets contain nerve fibers in every stage of degeneration. Products of degeneration are abundant and the neuroglia is somewhat thickened and has a finely granular structure. Glia cells are often plentiful, the vessels are distended, and their lymphatics filled with leukocytes and fat containing cells. Although the borders of the patches usually appear well defined to the naked eye, under the microscope a gradual transition from diseased to healthy tissue is generally recognizable.

The changes in the myelin sheaths are well seen in the intermediate islets, particularly toward their periphery. The first alteration appears to consist in swelling of the myelin substance. This often presents a finely granular structure, and is stained faintly by coloring reagents. Later the myelin sheaths begin to disintegrate, undergoing a process of lecitholysis or fatty degeneration. At this stage the myelin sheath presents in longitudinal sections a varicose or scalloped appearance. Finally, it completely disappears.

The axis cylinders persist after their medullary sheaths have disappeared. This observation, first made by Charcot, is of great pathological and clinical interest. The unclothed axis cylinders do not, however, always present a healthy appearance. Some of them are swollen, many, on the other hand, are considerably reduced in diameter, and on longitudinal section are found to be fusiform in shape. Popoff, Erben, and Strähuber assert that these fine axis cylinders are newly formed, while Weigert and others believe that they are atrophic fibers. Bielschowsky, using his silver impregnation method, has demonstrated that the axis cylinder sometimes presents a knobby appearance like a string of pearls, and that it is sometimes split up into a bundle of fine fibrils. The same observer has traced the axis cylinder through a diseased patch to its continuation once more as a myelinated fiber. Kaplan, using his elective axis cylinder method, has demonstrated what he regards as a cement substance (axostrome) in the axis cylinder, with staining properties

[1] *Rev. of Neurol. and Psychiat.*, 1904.

identical with the medullary sheath. Some naked axis cylinders retain their axostrome, others lose it.

Secondary degenerations, although described (Babinski, Jolly, Rossolimo), are undoubtedly uncommon, as the comparative integrity of the axis cylinders within the placques might lead one to expect. Even when present they rarely extend more than a short distance from the placques which produce them. The nerve cells of the spinal cord and posterior ganglia often show a degree of chromatolysis and an increase of the intracellular pigment, but these alterations are usually surprisingly slight, even in cells situated within the more chronic patches.

The neuroglia in the firmer patches consists of a dense felt-work of fibers with few nuclei. Recent patches show a more open network. According to Weigert, the neuroglial increase met with in disseminated sclerosis is denser than that in any other condition.

Changes in the bloodvessels were first described by Rindfleisch. Ribbert found a large vessel situated more or less in the centre of most of the patches in those cases he examined, and in two instances he found appearances suggesting with great probability an arterial thrombosis. Dejerine and Thomas point out that the vessels may be more numerous than normal in the sclerosed areas. Thickening of the vessel walls has been described by several observers, and narrowing of the lumen with, indeed, actual occlusion has been recorded. Hyaline degeneration of the wall has also been noted.

Notwithstanding the above-mentioned records, it is to be borne in mind that in a number of cases which have been examined no pathological changes in the bloodvessels have been met with (Taylor, Schüler, Buchwald, Jolly, the writer), while the circumstance that these changes when present are limited to the patches, and may well be secondary to the neuroglial increase, must be kept in view.

Rossolimo indicates that slight inflammatory changes are to be found in some cases in the neighborhood of the vessels. Thus the perivascular spaces are dilated and contain both mononuclear and polynuclear leukocytes in various stages of fatty degeneration. Borst, who also emphasizes these changes in the perivascular lymph, holds that a lymph stasis may be a factor of importance. Goldscheider described an acute case after typhoid fever with marked changes around the inflamed vessels.

**Pathogenesis.**—This is most obscure. The problem involves the nature and sequence of the histological changes and their cause. In the first place, the author sides with E. Müller, who, following Ziegler and Schmaus, speaks of a primary as opposed to a secondary disseminated sclerosis, thereby excluding such cases as those described by Williamson, Marburg, and others, in which prominent changes in the bloodvessels and the distribution of the diseased areas suggest their dependence upon interference with the vascular supply. It is obvious that the diseased process may begin either in the neuroglia or in the parenchyma, or that it may affect both tissues simultaneously to a greater or less degree. E. Müller is a strong supporter of the view that the initial change consists in a neuroglial proliferation, as a result of which the nerve elements are strangled. In favor of his opinion he states that the patches are especially prone to occur in regions where the neuroglia is normally most dense, and, further, that the density of the neuroglia in disseminated sclerosis is much in excess of that met with in any other

affection of the nervous system. Again, he points out that toward the edge of a placque increase in the neuroglia may sometimes be seen surrounding perfectly healthy nerve fibers. Even if we grant that the neuroglial increase is greatest in the regions in which the neuroglia is normally most dense, this is not a weighty argument in support of the primary affection of the neuroglia. Further, in cases of sharply defined secondary degeneration occurring in the spinal cord, a neuroglial proliferation which must be secondary to the atrophy of the nerve fibers is sometimes observed beyond the limits of the degenerated tract (Mott and others).

Very strong evidence against the primary neuroglial origin of the disease is afforded by the facts that in some patches very little neuroglial increase is to be observed, and that such islets may co-exist in the same case with others in which the neuroglial proliferation is excessive. Further, the rapidity with which symptoms develop and the manner in which they vary in intensity are difficult to explain on this hypothesis.

Strümpell,[1] who holds very similar views to Müller, regards the condition as a multiple gliosis predisposed to by a congenital factor, although determined, it may be, by a variety of exogenous causes. His conclusions are largely based upon two cases examined post mortem, in which a multiple gliosis was associated with hydromyelia.

The view, which is supported among others by Risien Russell[2] and Tredgold, that the primary change is in the parenchyma, the neuroglial increase being a compensatory process, has much more to commend it in the opinion of the writer. The strongest argument in support of this view is the circumstance that very profound changes are sometimes found in the myelin sheaths, with little or no increase of the neuroglia in the same region. Toward the periphery of a patch, for instance, degenerative changes are sometimes found in nerve fibers which are lying in the midst of perfectly healthy neuroglia.

A third body of observers take up the position that both the myelin sheaths and the neuroglia suffer primarily as a result of a common cause. Erb, Gowers, Rossolimo, and others regard the process as a form of chronic myelitis. Leyden and Goldscheider and Oppenheim state that disseminated sclerosis may develop as a sequel to an acute myelitis, or an encephalitis circumscripta. E. Müller and E. W. Taylor, on the other hand, stoutly deny any evidence of inflammatory changes in the very considerable material at their disposal.

Ribbert, E. W. Taylor, Strähuber, T. Buzzard, and others believe that some toxin of undetermined nature circulating in the blood is the agent responsible for the histological changes.

**Symptoms.**—As the multiplicity of the lesions and their widespread distribution would lead one to expect, the symptoms met with in this disease are both numerous and varied. The clinical picture is none the less distinctive. A classification into three clinical types—spinal, cerebral, and cerebrospinal—as suggested by Charcot, can only be accepted in a relative sense, for cases are seldom met with in which the symptoms are exclusively spinal, while still more rarely is a diagnosis made in the presence of purely cerebral manifestations. The circumstances, first, that the patches are scattered

[1] *Neurol. Centralbl.*, 1896, p, 961.
[2] *Allbutt's System of Medicine*, 1899, vii, 83.

indiscriminately throughout the nervous system, and secondly, that in the spinal cord the motor fibers are gathered into small compass, readily explain the frequency with which paresis or paralysis of the lower limbs is observed in the early stages. Moreover, it is to be remembered that small patches scattered through the brain substance, unless they implicate special situations, may fail to afford indications of their presence.

The characteristic features of the morbid process, the preservation of the axis cylinders, and the destruction of their medullary sheaths, may account for the special characters of certain symptoms, such as the optic atrophy, absence of muscular wasting, and perhaps the intention tremor.

**Motor Symptoms.**—Weakness in the lower extremities is almost always complained of, even in the earliest stages. The paresis may be so slight that its presence is only perceptible after prolonged exercise. Thus, undue fatigue in one or both lower extremities, after a long walk, is one of the commonest early symptoms. The loss of power in the limbs, which is often asymmetrical, may be so pronounced that it is only with difficulty that the patient can walk up stairs or across the room. In advanced cases the legs are completely paralyzed. Temporary paralysis of one arm is by no means uncommon as an early symptom. A slight degree of weakness in the hands and arms is often present, yet persistent paralysis in the extremities is exceptional unless in the terminal stages. Pronounced muscular atrophy is very rare. When this symptom is present the case may simulate amyotrophic lateral sclerosis, as in a few recorded instances.

Muscular hypertonicity is usually associated with the paraplegic state. The spasticity is slight in some cases; occasionally it is so pronounced that this rather than the motor weakness is the chief cause of difficulty in locomotion. Sudden flexor spasms often constitute a troublesome symptom, since they interfere with sleep. In the later stages of the disease flexor contractures tend to develop.

A slight degree of ataxia is a common early symptom in the upper limbs. The uncertainty of the required movement is clearly to be distinguished from the intention tremor presently to be referred to. Ataxia in the lower extremities may usually be demonstrated; indeed, in some cases this symptom constitutes the most striking feature of the picture, even in the earliest stages. The ataxia is, as a rule, of the cerebellar type, and hence is little increased when the eyes are closed.

The gait is usually altered at a very early date, yet it is sometimes surprising to find no appreciable alteration even in the presence of certain evidence of pyramidal disease. A common type of progression is the spastic paretic gait, in which there is a combination of spasticity and weakness, one leg being often more affected than its fellow. The patient shuffles along, dragging his feet as if the toes were stuck to the ground. In addition to the spasticity there is often some unsteadiness in walking, a condition, in short, of ataxic paraplegia. Sometimes, indeed, incoördination is the conspicuous feature; the ataxia, however differs notably from that of tabes, for it is rarely increased when the eyes are closed. The stiff way in which the patient holds his head and the rhythmic tremor in head and trunk met with in the classical type is almost pathognomonic. The gait is sometimes staggering and reeling, as in cerebellar disease.

Another symptom, and one which is so characteristic that Charcot included it with nystagmus and speech defect as one of the cardinal signs

of the disease, is the so-called intention tremor. So long as the patient is in a posture of complete muscular relaxation no tremor is to be observed; if he raises his hand with the object of carrying out some definite movement, the tremor commences. It is well brought out on attempts to write. Even when the patient is able to touch the point of the nose with the finger tip without any tremor, if the finger is kept in contact with the nose for a few seconds a rhythmical tremor of small amplitude may manifest itself. The rapidity of the tremor varies from four to six per second. The tremor may spread to other groups of muscles not directly called into action by the required movement. The intention tremor is usually most marked in the hands, but it is also seen in the lower extremities. It may be seen also in the head and neck during the act of walking. This constitutes no exception to the statement that the tremor only occurs during the performance of a purposive act, for while the patient is sitting or walking, the muscles of the neck require to be constantly in a state of contraction to maintain the head in this position. Tremor of the face and jaw has occasionally been observed. Intention tremor, when present, is one of the most characteristic symptoms, yet it is often absent.

Much discussion has taken place as to the origin of the tremor. Charcot was of opinion that it was due to an irregularity of conduction of the motor impulses through the sclerosed areas, a result of defective insulation of the axis cylinders or changes in them consequent upon disease of the medullary sheaths. Others hold that it is due to disease located in a particular situation, and in support of their view, point out that intention tremor is absent in cases in which the disease is practically limited to the spinal cord.

**Sensory Symptoms.**—The older writers emphasized the absence of sensory disturbances. This experience is quite contrary to that of more recent observers, by whom subjective sensory symptoms, often of a fleeting character, it is true, are regarded as among the most characteristic of the early signs. Feelings of numbness, formication, deadness, tingling, and pins and needles, particularly in one or both hands or feet, are very common. A girdle sensation is of comparatively frequent occurrence, while a sensation of constriction around a limb is occasionally experienced. Shooting pains, especially in the lower limbs, are sometimes complained of. Giddiness, a common symptom, may occur early, and often it occurs in paroxysms. Headache, although seldom severe, is often present. Vomiting is an occasional symptom, and may occur in association with paroxysms of headache or dizziness, or independently. A fixed pain in the back situated over the lower part of the sacrum is of frequent occurrence. Objective sensory defects of slight degree may be detected in most cases at one time or another if carefully looked for. Thus, Freund, who made a special study of 33 cases, found some definite alteration in no less than 29. All forms of sensation may be implicated. The hands and feet are most often involved, but hemianæsthesia is not uncommon. The relative degree of the sensory disturbances and their fleeting nature are very characteristic.

**Tendon-jerks and Skin Reflexes.**—The knee-jerks are almost always exaggerated and the increase is often more marked on one side than the other. In some cases a patella clonus can be elicited. Absence of the knee-jerks has been recorded, but is undoubtedly very rare. The writer cannot recall ever having seen such a case. Thomas Buzzard regards absence of the knee-jerks in association with ankle clonus as a symptom complex which is

suggestive of the disease. The activity of the tendo-Achilles jerk is almost always increased. Ankle clonus, usually bilateral, but sometimes confined to one side, is demonstrable in a considerable proportion of cases. The tendon reflexes in the upper extremities are, as a rule, abnormally active.

The great value of the Babinski sign or extensor plantar reflex as an indication of disease of the corticospinal descending tract has been amply confirmed. The collected experience of recent observers shows that in disseminated sclerosis, even at the time when the earliest symptoms are complained of, the plantar reflex is almost always of the extensor type. Moreover in the writer's experience the Babinski sign is usually present in its most typical form, the slow dorsiflexion of the great toe being easily elicited from the whole plantar surface. A crossed plantar reflex may sometimes be demonstrated (Byrom Bramwell).

The abdominal reflexes have recently attracted considerable attention in this malady. They are sometimes difficult to demonstrate in healthy individuals. Recent observations indicate (1) their absence as specially characteristic of disseminated sclerosis, and (2) that they disappear early in the disease. Thus, Strümpell, who examined 185 persons free from nervous disease, noted their absence in 13.5 per cent. of these; while among 24 cases of disseminated sclerosis they were absent in 67 per cent. E. Müller found that the reflex was only absent in 5 per cent. of individuals free from nervous disease, as compared with 62.5 per cent. in 47 cases of disseminated sclerosis. The observations of Probst and Gang corroborate these conclusions. The latter writer remarks that although the abdominal reflexes fail in many organic affections of the nervous system, they never fail with so much regularity as in disseminated sclerosis. There is nothing of special importance to note with regard to the cremasteric and other superficial reflexes in this disease.

**Micturition and Defecation.**—Disorders of micturition are ultimately present in all cases. Notwithstanding contrary statements, evidence of disturbance of the function of the bladder, often so slight as to cause little or no inconvenience to the patient, is usually forthcoming if carefully inquired for even before the more permanent symptoms of the disease are fully established. Thus in 80 per cent. of Oppenheim's cases there was some disorder of micturition. The writer obtained a history of bladder disturbance in 30 out of 34 consecutive cases in which a special inquiry was made. "Precipitate micturition," or a feeling of inability to retain the urine immediately the desire to empty the bladder is felt, is the abnormality usually first experienced. Another indication of defective function which usually appears at a later date is the delay which precedes the commencement of the act. The patient may require to strain for several minutes before the urine begins to flow. When the urine commences to flow it comes in a stream of full volume, although it may be defective in force. Retention of urine, a not uncommon symptom later, is a complication which must always be regarded with anxiety on account of the liability to cystitis. Incontinence is common in advanced cases, and is usually of the remittent type, but rarely depending upon complete paralysis of the bladder wall. Like so many of the symptoms of this disease, the bladder symptoms often vary from time to time.

Constipation is the rule. Some loss of control is also often seen when any tendency to looseness of the bowels exists. True incontinence of fæces is most exceptional unless in the later stages.

The sexual functions are at times interfered with. Impairment both of the sexual desire and power have been described, and an exaggerated sexual desire has been noted in a few cases.

**Defects of Vision and Optic Atrophy.**—Visual defects, which are frequent and characteristic, often appear at an early date. A temporary unilateral amblyopia of sudden onset and often regarded as hysterical in origin, since it is unaccompanied by changes in the fundus oculi, may indeed be the first indication of disease. Diplopia with no visible impairment of the ocular movements, another early symptom for which the patient seeks advice, was present in 13 of 31 cases recently examined by the writer. Although the visual defects are often fleeting, a permanent diminution in central vision, associated with atrophic changes in the optic discs, is often met with. Unlike the amblyopia of tabes, this defect of sight very rarely progresses to complete blindness. A central scotoma, usually bilateral, it may be for color alone, is often to be found if carefully looked for, and when present is a valuable aid in diagnosis. Concentric contraction of the field of vision is found in some cases. Uhtoff,[1] who has studied the alterations of the visual fields in 150 cases, describes six groups of cases: (1) In one-half of all cases with anomalies of the visual fields, central scotomata, usually bilateral, often relative, with no alteration of the periphery of the field, are present. (2) In rare cases peripheral contraction of the field occurs together with scotomata. (3) In a considerable percentage of cases there is an irregular peripheral contraction of the field. (4) Isolated cases present regular concentric contraction. This condition occurs in pure multiple sclerosis as well as in those cases which are complicated by hysteria. (5) In one case there was a ring scotoma, and in another (6) a central scotoma was present, but cleared up, and then a persistent peripheral contraction of the field appeared.

Atrophic changes in the optic disk are of very great importance in diagnosis. There may be some pallor of the temporal side of the disk, an incomplete pallor of the whole papilla, so that the inner part still retains a pinkish tint, or, when the process is more advanced, the whole papilla may be uniformly pale. As T. Buzzard[2] has pointed out, slight differences in the color of the papillæ on opposite sides is often of the greatest value in determining whether the appearance is to be regarded as pathological. The frequency of recognizable pallor of the disk has been variously estimated by different authors. Uhtoff found that the papillæ presented a pathological degree of pallor in 37 per cent. of all cases, and other observers have found an even higher percentage. The dependence of the changes in the optic disks upon an islet of disease situated in the optic disks has been repeatedly demonstrated.

A small number of cases have been observed in which there was a slight degree of optic neuritis, never amounting to a true papillitis. The appearances, which are those of a retrobulbar neuritis, have been proved to be due to the presence of a placque in the immediate vicinity of the papilla.

**Ocular Muscles, Nystagmus, Pupils, etc.**—The ocular muscles are particularly liable to be affected (Uhtoff, 20 per cent.). Diplopia is often complained of. Usually when this symptom is present the paresis is so slight in degree as to escape detection unless specially examined for by the appropriate visual tests. The sixth nerve is most commonly affected, and paresis of the

[1] *The Ophthalmoscope*, 1905, p. 429.
[2] *British Medical Journal*, 1893, ii, 779.

third is usually partial. These muscular defects are almost always of slight degree. Complete ophthalmoplegia has, however, been recorded. Ptosis either confined to one side or bilateral is of more frequent occurrence than any other form of ocular palsy. Wildbrand and Sanger lay stress on this symptom, believing that in certain cases it may be of even greater diagnostic importance than nystagmoid movements. Müller, who met with ptosis in 8 per cent. of his cases, also observed paresis of lateral conjugate movement in three cases, of upward movement in two.

Nystagmus belongs to the symptomatic triad formulated by Charcot. Three degrees may be conveniently recognized: (1) Nystagmus which occurs while the eyes are in a position of rest; (2) nystagmus which is only present in association with a fixed purposive movement such as lateral or vertical deviation or upon fixing a near object; and (3) movements which occur during fixation, but which rapidly cease and are at the same time frequently irregular both in time and amplitude. To this last group we would reserve the term nystagmoid movements. In few cases of disseminated sclerosis do we find the type which we have classed in the first group. On the other hand, the second and third types are very common. The movements are usually fairly rapid and regular, but vary considerably in amplitude in different cases. Coarse nystagmus, for example, may be elicited when the patient looks to one side, while when he looks in the opposite direction the movements may be much finer in quality. The nystagmus is usually most obvious when the patient looks to the extreme left or right and at the same time directs the eyes slightly upward; sometimes, however, when he looks directly upward or downward the movements are also visible. Occasionally the movements are only to be seen when the eyes are fixed at the extreme of the vertical plane. The direction of the oscillation is commonly from side to side, the two eyes acting in unison. A certain amount of rotation of the eyeball is, however, by no means uncommon, and in rare cases the oscillations take place in a vertical direction. The writer has seen one case in which the movements were confined to one eye.

Kunn has noted a "Zitterbewegung" in the ciliary muscle. The writer has also observed evidence of rhythmic contractions in this muscle on ophthalmoscopic examination. Alterations in the size and activity of the pupil are not uncommon. There was a difference in the size of the pupils in 24 per cent. of Uhtoff's cases. Parinaud remarks that inequality of the pupils occurs early in the disease, and that miosis when present is spastic as opposed to the paralytic miosis of tabes.

The pupils react well on convergence provided that the movement of convergence is not itself defective. The light reflex, although sometimes sluggish, is very rarely lost. Müller notes that among 364 cases observed by Fränkl-Hochwart, Probst and Uhtoff, the Argyll-Robertson pupil was only present in 4. Fränkl-Hochwart met with hippus in 12 cases.

**Speech and Articulation.**—In the older description of this disease speech affections occupy a prominent position. They are, however, by no means of constant occurrence. Byrom Bramwell found some defect of speech in 62 per cent. of his series, while some alteration was present in two-fifths of Müller's cases. Among 33 consecutive cases recently examined by the writer, in 15 (45 per cent.) speech was altered.

It may be that the patient notices some slight difficulty in speech, especially toward the end of a sentence, even before any change can be detected by

the observer. The alteration in speech in the early stages of the disease may be so slight that it is difficult to describe, and yet it may be sufficiently characteristic to the experienced observer to help him greatly in making his diagnosis. The slow and measured way in which the patient enunciates and a singular monotony of voice are usually the salient features to attract attention. When he reads aloud it is interesting to note the way in which he tries to vary the pitch of his voice, of the monotony of which he is obviously conscious. At a later date he chops his words, each syllable is pronounced slowly, hesitatingly, distinctly, and precisely. This is the syllabic, scanning, or staccato speech which has been compared by Charcot to that of incipient intoxication, by Oppenheim to that of a child learning to spell, and by Marie to that of an individual who is steadily but surely ascending a hill. Each syllable is produced with apparent effort, an impression which the associated tremor often present in the face and head tends to accentuate. Occasionally a nasal twang is noticeable. Müller found a true staccato speech in only 15 per cent. of his cases. Certain letters, notably l, p, g, b, and d, are especially apt to give rise to difficulty in pronunciation. Later in the course of the disease, defective articulation may render speech quite unintelligible. As Oppenheim observes, when an attempt is made to intone the letter e, a distinct tremor in the voice is often noticeable. This observer has drawn attention to the scanning character of the respiration, to which he is inclined to attribute the corresponding peculiarity in speech. Goldscheider believes that the monotonous character of the speech may be due to defective control over the act of expiration, for he has shown that the augmentations and interruptions in the expiratory blast, which he has demonstrated take place during the act of speaking, are in this disease abnormally slow and defective. Aphonia is occasionally present. Réthi has recently (1907) given a detailed description of the appearances in 44 collected cases in which the larynx was examined. In 63 per cent. some form of laryngeal paralysis was present, while in half the cases tremor of the vocal cords was observed.

**Mental Symptoms.**—It is somewhat surprising to find that profound psychic alterations are exceptional. Even in fully developed cases there may be no obvious mental disturbance. As a rule, however, after the disease has been in progress for any length of time, a certain degree of mental impairment is noticeable. Thus in Byrom Bramwell's series of cases defective memory was present in 50 per cent., some emotional alteration in 40 per cent., and further evidence of intellectual impairment in 27 per cent., while E. Müller found a slight enfeeblement of the psychical faculties in about 25 per cent. The psychic alteration shows itself especially in enfeeblement of memory, in a certain slowness of thought, in the apathy and indifference with which the patient regards his surroundings, and in a degree of stupidity which the facial expression would almost lead one to expect. A sense of well-being, or euphoria akin to the *spes phthisica*, is often observed, less commonly a depression of spirits which has been known to pass into actual melancholia. Emotional control is often impaired. The occurrence of outbreaks of emotion does not necessarily imply a corresponding defect in the intellectual capacity.

Seiffer[1] has recently studied elaborately the mental state in 10 consecutive

[1] *Arch. f. Psych.*, Band xl, Heft 1, p. 252.

cases. He found that 9 of these showed considerable disorder of intellect. Concrete ideas derived from the remote and recent past and the association of ideas he found to be especially defective. Attention and the grasp of abstract ideas were frequently but not so constantly impaired. He concludes that this condition of dementia is indistinguishable from that which is associated with epilepsy and alcoholism. Delusions of persecution, maniacal states, ideas of grandeur and exaltation have been observed in occasional instances. In this connection it is interesting to note that several cases have been reported in which a pathological examination established the co-existence of general paralysis of the insane.[1]

**Trophic and Vasomotor Changes.**—These are extremely rare and of corresponding minor significance. James Collier has reported five cases in which he noted the presence of erythromelalgia. There are a number of cases on record in which muscular atrophy was present, although this is altogether exceptional. Bedsores may develop in the terminal phases.

**Congestive and Epileptic Attacks.**—Attacks similar in character to the congestive attacks of general paralysis of the insane have been recorded. Their rarity may be gathered from the fact that the writer, who has examined over 150 cases of this disease, can only recall one instance. Fränkl-Hochwart met with apoplectic attacks in 10 of his series. The attacks have been sometimes preceded by giddiness, the patient falls to the ground, is sometimes convulsed, and may remain comatose for several hours. The temperature is raised. Hemiplegia is an occasional sequel. Sudden attacks of giddiness and faintness without loss of consciousness are less uncommon. Müller has met with two cases in which Jacksonian attacks occurred. Epileptic attacks were noted in 3 of Frankl-Hochwart's 206 cases.

**Earliest Symptoms.**—Motor weakness in the limbs, and especially in both lower extremities, paræsthesias in the limbs, giddiness, tremor, or ataxia and amblyopia are, in the order stated, the most common early symptoms. The development of the initial symptoms as complained of by the patient has been studied by Byrom Bramwell (110 cases), who states: "In 41 cases the initial symptoms occurred in the extremities, and were purely motor or ataxic; in 3 cases the symptoms were purely sensory; in 22 cases the symptoms were both sensory and motor; in 24 cases the symptoms were purely cerebral or ocular; in 17 cases there were different combinations of motor and sensory symptoms with cerebral or ocular symptoms." Ashley W. Mackintosh, who has tabulated 80 cases, for the most part drawn from Ferrier's material, remarks on the extraordinary variability of the modes of onset, the comparatively large number of cases with acute or sudden onset (10 cases), the frequent occurrence of purely unilateral symptoms at the onset (15 cases), and the comparative frequency of the occurrence of sensory symptoms at the onset, either alone (10 cases) or combined with other symptoms (19 cases).

**Types.**—A number of clinical types are recognizable according to the preponderance of certain symptoms.

1. The classical type, characterized by intention tremor, nystagmus, speech affection, and it may be other cerebral manifestations in association with a spastic or ataxic paraplegia.

2. A cerebrospinal type, in which even in the absence of the symptomatic

[1] Hunt, *American Journal of the Medical Sciences*, 1903, cxxvi, 126.

triad of Charcot, symptoms alike cerebral and spinal point to widespread disease of the central nervous system.

3. The hysterical or intermittent type, in which fleeting symptoms suggestive of hysteria constitute the most striking feature of the case.

4. The spinal type, characterized by prominent spinal symptoms resembling a variety of spinal diseases. Of these, a progressive paraplegia unaccompanied by sensory disturbance and indistinguishable at first, it may be, from primary lateral sclerosis is very common. Again, a spastic paraplegia may develop with the rapidity of an acute myelitis. The co-existence of ataxia and paraplegia may constitute a symptom complex which closely resembles the combined systemic diseases.

5. The cerebral type is much less common. Occasionally vomiting, giddiness, and headache are the most striking symptoms. In rare cases, a cerebral hemiplegia is met with as the leading feature of the case. Very exceptionally, bulbar manifestations or mental symptoms are the most conspicuous alterations.

6. A sacral type, recently described by Oppenheim and Kurt Mendel, in which pains in the legs, disturbance of the sphincters and sexual functions, point to a lesion in the lower part of the spinal cord.

**Diagnosis.**—This is not so difficult as the irregular distribution of the pathological lesions and the multiplicity of types described might lead one to expect. The diagnosis is based upon (1) the detection of certain characteristic symptoms or association of symptoms, upon which alone a positive opinion may sometimes be advanced; (2) a history of variation in the severity of the symptoms, which, when present, is in itself so suggestive as to be almost pathognomonic; and (3) the exclusion of a variety of diseases, which may closely simulate disseminated sclerosis. The classical type presents features which are distinctive, for intention tremor, scanning speech, and nystagmus in association with a spastic or ataxic paraplegia and an extensor response place the problem beyond all doubt. These cases, however, form only a small percentage.

It is especially in the early stages that errors are made, notably when temporary manifestations disappear, leaving the patient in perfect health, or when the symptoms are such that the supposition of a single focus of disease appears to satisfactorily explain them.

Weakness in the lower extremities, it matters not how slight, in a young adult, if accompanied by exaggerated tendon-jerks and the Babinski sign, should always arouse in the mind of the physician the possibility of this disease, when on further examination no evidence of vertebral disease, no muscular wasting, and no objective sensory disturbances are detected. If in addition a degree of ataxia is present and the abdominal reflexes are absent, the suspicion is considerably strengthened. Further, if there is a history of previous temporary paresis or paræsthesia in a limb or of a fleeting amblyopia or diplopia, the suspicion amounts almost to a certainty. A slight degree of ataxia in the upper extremities, the occurrence of vertigo, and, above all, pathological pallor of one or both optic disks in conjunction with a spastic or ataxic paraplegia render the diagnosis almost certain, even in the absence of a history of variations in the clinical picture of nystagmus, intention tremor, or characteristic alterations in speech.

*Hysteria.*—Of the many diseases for which disseminated sclerosis may be mistaken, this takes the first place. Disseminated sclerosis often occurs

in young women, its early symptoms are often transient and completely recovered from, while hysterical stigmata are often met with in association with the organic affection. To Thomas Buzzard especially is due the credit of showing the frequency with which disseminated sclerosis is mistaken for hysteria, an error which is fortunately becoming every day less common. Transitory paresis or paræsthesia in a limb, even though completely recovered from, symptoms which some years ago would have been diagnosed as certainly functional, are to be regarded with the gravest suspicion, since they are so often the precursors of the affection which we are considering. Even although a hemianæsthesia with involvement of the special senses is detected, the observer must remember that the recognition of hysteria does not exclude associated organic disease. Nystagmus, pallor of the optic disks, and the Babinski sign justify a positive diagnosis of organic disease, while incontinence of urine, a symptom which it is true does not usually occur until the later stages of the disease, carries with it the same significance.

Acute disseminated myelitis or encephalomyelitis presents a very similar picture; some believe that it is a more intense degree of the same process. The symptoms develop acutely, it may be with fever, and reach their height in a few days. There is muscular wasting. Variations in the intensity of the symptoms do not occur. Progress is in the direction of improvement, although this is very often limited.

*Subacute combined degeneration* of the spinal cord may simulate disseminated sclerosis very closely. The diagnosis is very important, for these cases often run a very rapid course. In both diseases ataxic paraplegia is often the most striking feature of the clinical picture. Subacute combined degeneration usually develops rather later in life, and its symptoms once present show no tendency to disappear, as in disseminated sclerosis. Pallor of the optic discs is not met with, while pronounced anæsthesia, beginning in the feet and progressing steadily upwards, is the rule. The symptom complex of the later stages, viz., the flaccid palsy, often suddenly developed, with abolition of the tendon jerks and complete loss of control over the sphincters, places the diagnosis beyond all doubt.

*Cerebrospinal syphilis* occasionally closely resembles disseminated sclerosis. The peculiar pallor of the optic disks, the presence of a color scotoma, and absence of the abdominal reflexes speak strongly for disseminated sclerosis. Scanning speech and intention tremor do not occur in cerebrospinal syphilis. The Argyll-Robertson pupil is almost conclusive evidence of syphilis of the nervous system, corroborative evidence of which is afforded by the detection of a cerebrospinal lymphocytosis or a positive Wassermann reaction. Improvement under antisyphilitic remedies may confirm the diagnosis.

*General paralysis of the insane* has been known to cause trouble in diagnosis. The writer has met with one instance in which this was so. The more profound mental alterations, a history of syphilis, and the existence of the Argyll-Robertson pupil, a cerebrospinal lymphocytosis or positive Wassermann reaction are data of importance. Cases have been recorded in which the lesions of disseminated sclerosis and general paralysis of the insane co-existed post mortem.

*Intracranial tumor* is a diagnosis sometimes made in the early stages of disseminated sclerosis. The writer has seen this error made on three occasions by eminent authorities, the subsequent progress of the case demonstrating its true nature. Giddiness with vomiting, unsteady gait, nystagmus,

and tremor may all result from a patch of sclerosis situated in the cerebellar peduncles. Headache may be present, though rarely severe, while in very exceptional cases there may be a slight degree of optic neuritis.

*All forms of spastic paraplegia* in which the paralysis of the lower limbs is the dominant feature may cause difficulty. Many of the cases formerly classed as primary lateral sclerosis, an affection which we now know to be one of the rarest of organic nervous diseases, were without doubt examples of disseminated sclerosis. It may be, indeed, that there is at the present time a tendency for the pendulum to swing too far in the opposite direction. The possibility of amyotrophic lateral sclerosis must always be kept in view.

The differential diagnosis from *pseudo-sclerosis* and *diffuse sclerosis* will be considered under these diseases.

**Prognosis.**—It is very doubtful whether permanent recovery ever takes place. Cases have been observed in which the patient has remained free from symptoms for several years. Thus, Oppenheim mentions three examples in which the patients had recovered and been in perfect health for from five to ten years, and similar instances have been met with by others. No less uncommon, however, are cases such as those recently reported by T. Buzzard in which after intervals of several years the symptoms of the disease have reappeared. Time may show that very exceptionally permanent recovery may take place. For the present this question cannot be answered in the affirmative.

The disease tends in the majority of cases to run a chronic course, often interrupted, it is true, by remissions and relapses. There are instances in which death has occurred within a year, but these are exceptional. Byrom Bramwell has ascertained the duration of life from the appearance of the first symptom in the 35 fatal cases of his series. He found that in 10 cases (28.5 per cent.) death occurred in less than five years, in 23 (65.7 per cent.) in less than ten years, and in 31 (88.5 per cent.) in less than fifteen years; one patient died within a year. In no instance was the duration more than twenty-one years. On the other hand, among 61 non-fatal cases, 5 had lived at the time of writing for various periods from twenty-two to thirty-three years. The average duration of life in 96 fatal and non-fatal cases was found to be ten and a half years.

There are practically no available data which permit of an opinion as to the probable course or duration of a particular type of case when seen in its early stages, although if bulbar symptoms or pronounced bladder trouble appear early the expectation of life will probably be less than the average.

**Treatment.**—Conclusions as to the beneficial effect of this or that line of treatment are naturally extremely difficult to arrive at in the case of an affection such as disseminated sclerosis, in which there is a tendency for intermissions and remissions to occur quite independently of all therapeutic intervention.

No known remedy has been proved beyond doubt to exert a favorable effect, hence in our present ignorance as to its pathology, treatment is limited (1) to certain general directions indicated by common-sense; (2) to the avoidance of circumstances which may be expected or have actually been proved to have a deleterious influence; and (3) to attempts to alleviate individual symptoms as they arise.

The spirit of optimism which these patients not uncommonly present should, so far as possible, be left undisturbed by the physician, for even

although it is perhaps unreasonable to suppose that such a mental state can influence the course favorably, yet it is certain that prospects of improvement do much to make the patient's outlook on life more bearable.

Strict injunctions are to be laid down as to the avoidance of muscular exhaustion and in particular the patient should be cautioned when walking always to stop short of fatigue. Confinement to bed is, on the other hand, a mistake; indeed, the patient should rather be encouraged to regularly exercise his muscles. A warm climate may be recommended to those who can afford it. Plenty of fresh air and a nutritious diet are advisable. Worries of all kinds are to be, as far as possible, avoided. Hot baths are contraindicated, experience having shown that sometimes harm is done thereby. Marriage should be forbidden. The prejudicial effect of pregnancy is undoubted and should be explained to female patients. The induction of abortion may be even justifiable, especially when it is ascertained that during a previous pregnancy the symptoms were intensified.

Ataxia when pronounced is at times benefited by a course of coördinated exercises, although the brilliant results sometimes obtained by this method in the treatment of tabes are not to be expected. No drug is known which influences the intention tremor. Flexor spasms, often a troublesome symptom, since they interfere with sleep, are occasionally alleviated by five to ten grains of veronal given at night. When rigidity of the limbs is extreme the cautery applied to the back on either side of the spine may prove of service. Incontinence of urine, if due to overflow from a distended bladder, is to be treated by the passage of the catheter at regular intervals, while if it is of the intermittent variety, benefit may result from tincture of belladonna in 10 minim doses. Obstinate constipation is best relieved by a daily soap and water enema.

In bedridden cases a water bed is indicated. Bedsores are to be avoided by thorough attention to cleanliness; the skin is to be washed with alcohol twice a day and the parts dusted with some bland non-irritating powder.

Associated hysterical symptoms should be treated by a confident prediction that they will disappear and by the methods of suggestion and persuasion commonly employed in the treatment of such manifestations.

Although no drug has been definitely proved to have any effect upon the course of the disease, the writer is inclined to believe that arsenic in moderate doses, 2 to 6 minims of the liquor three times a day, is sometimes of service. Strychnine is to be avoided, since it tends to increase the spastic state.

**Pseudo-sclerosis.**—**Historical.**—Westphal described under this name two cases, diagnosed during life as disseminated sclerosis, in one of which the autopsy was entirely negative, while in the other the only change found was a general increase in the consistency of the brain. Strümpell gave the first distinctive description of the symptom complex. Fränkl-Hochwart[1] has studied the cases reported up to 1903, 13 in number. Two important contributions have since appeared, one by Fickler[2] who has reported two cases and has reviewed the literature up to date. The other is by Rebizzi,[3] who regards pseudo-sclerosis and diffuse sclerosis as the early and late stage of the same affection, for which he proposes the inclusive title of Westphal-Strümpell's disease.

[1] *Arb. aus dem Neurol. Institut an der Wiener Universität*, 1903, Heft 10, p. 1.
[2] *Deutsche med. Wochenschr.*, 1904, No. 51.
[3] *Riv. di patol. nerv. a ment.*, vol. x.

**Etiology.**—Bäumlin's four patients were sisters, and Rebizzi has described cases occurring in father and daughter. A strong neuropathic heredity has been a striking feature in several instances. In no case has there been any definite relationship to trauma. Males and females are equally affected. The symptoms developed in 5 cases during the first decade, in 3 during the second, in 1 during the third, and in 4 during the fourth.

**Pathology.**—No changes were found post mortem in the majority of the cases examined. It should be mentioned, however, that in 6 cases a microscopic examination of the brain was not made, and that the majority of the observations predated the introduction of elective staining methods. The consistence of two of the brains examined is described as abnormally dense. A spinal leptomeningitis, slight in degree, was found by Bäumlin in one case. Strümpell met with a very slight degeneration in the cervical portion of the pyramidal tracts in both his cases, in one of which similar changes were also observed in the cervical portion of Gowers' tracts.

The disease has been regarded by Marie and other French writers as a neurosis; the changes above described are, however, apart from any clinical considerations, quite sufficient to negative this view. Rebizzi and Fränkl-Hochwart remark that the morbid anatomy of pseudo-sclerosis cannot be strictly separated from that of diffuse sclerosis, for cases are on record which might be included in either group, the two conditions merging into one another.

**Symptoms.**—Mental changes are the rule. A degree of apathy, peculiarities of disposition, irritability, and attacks of temper are among the alterations noted, while maniacal outbursts with hallucinations have been reported. Epileptic attacks were present in 6 of the 13 cases collected by Fränkl-Hochwart. Some disorder of speech was observed in 9 cases, in 5 of which speech was described as scanning, while in 2 it was unintelligible. Facial paralysis has not been noted, although in 3 cases a defect in facial mimicry was remarked on. The optic disks were normal in every case, with the exception of one instance reported by Fickler. The special senses have always been normal, as were the pupils. Nystagmus was present in one case only, and once paralysis of the oculomotor nerve was observed. Dysphagia was present in one case. Giddiness was complained of in three cases, while headache, faints, apoplectiform attacks, and uncontrollable outbursts of laughter were each noted on two occasions.

Weakness of the extremities is described in two-thirds of the reported cases. Three times the paresis was hemiplegic in distribution; in one case all four extremities were involved. The gait is referred to as "clumsy, ataxic, or unsteady" in 5 cases, twice it is noted as spastic, and in two instances the patient was unable to walk. Tremor in the upper extremities was a constant symptom. In four cases it occurred during repose. It was of larger amplitude and less regular than in disseminated sclerosis. Three times the lower limbs were also affected, the head was implicated in 3 cases, while twice the face and tongue were also involved. Contractures, sometimes temporary, sometimes permanent, were present in 9 cases. In 3 cases the neck muscles were affected, in 6 the arm, and in 7 the legs.

Pains, sometimes rather intense, were occasionally complained of. Three patients suffered from paræsthesias of various kinds. In one of Westphal's cases hyperæsthesia in different situations was noted at times in association with some loss of sense of position.

The tendon reflexes, examined in 10 cases, were once found to be normal, while on nine occasions they were exaggerated, clonus being present in five. In Fickler's case the superficial reflexes were absent, otherwise no anomalies of the skin reflexes have been met with. In 2 cases there was incontinence of urine, while in 1 there was incontinence of fæces. Transitory vasomotor disturbances were occasionally noted, and in 3 cases bedsores developed toward the termination.

In 3 cases death occurred in from nine to ten years, in 3 from four to six years, and in 4 from one to two years. Fränkl-Hochwart's patient lived for fifty-seven years after the onset of symptoms. Pronounced intermissions similar to those met with in disseminated sclerosis were present in three cases, while in the records of several others there are indications of the same.

**Diagnosis.**—Pseudo-sclerosis bears a very close resemblance clinically to disseminated sclerosis, from which it has rarely been distinguished during life. The chief points of importance which distinguish pseudo-sclerosis are the frequency of mental changes, notably attacks of excitement and pronounced dementia; the wild character of the tremor, which sometimes occurs during rest, a very rare occurrence in disseminated sclerosis; the occurrence of epileptic attacks, which were present in half the recorded cases of pseudo-sclerosis, but is very rare in disseminated sclerosis; the absence of changes in the optic disks; the comparative insignificance of the paretic symptoms in the lower limbs until the later stages, and the relative rarity of bladder symptoms. The development of the earliest symptoms during the first decade would be a strong point in favor of pseudo-sclerosis, as would a history of a similar malady affecting a relative. The diagnosis from diffuse sclerosis we shall consider under that affection.

**Diffuse Sclerosis.**—This rare affection, like pseudo-sclerosis, bears a close resemblance clinically to disseminated sclerosis.

**Etiology.**—It commences not uncommonly in childhood, sometimes not until the third or fourth decade. Unlike pseudo-sclerosis, heredity does not appear to be of etiological importance. On the other hand, there is a history of associated trauma in about a third of the reported cases. A relationship with alcohol, meningitis, and syphilis has been claimed in individual cases.

**Pathology.**—The most striking feature is the dense consistence of the brain, which has been described by individual writers as leathery, like india-rubber, elastic, and cutting like cartilage. The cut section looks almost like ivory. Weiss points out that where there is much shrinking, hydrocephalus internus and externus with ventricular dilatation may result. The alteration in consistence is associated with a great neuroglial increase throughout the nervous system, the neuroglia fibers forming a dense feltwork with numerous nuclei. The vessels are not greatly increased in number, although their walls are thickened. As already mentioned, the changes in some of the reported cases of pseudo-sclerosis are so similar to those met with in diffuse sclerosis that Rebizzi regards the two affections as merely the early and late stages of the same disease.

**Symptoms.**—These are so similar to those of pseudo-sclerosis that it will be unnecessary to do more than indicate the main resemblances and differences. In both the mental alterations occupy a prominent place, though in diffuse sclerosis dementia is more common and more profound. Speech

disturbances occur in a large proportion of cases of diffuse sclerosis, and are also more marked than in pseudo-sclerosis. Tremor occurs in about two-thirds of the cases while paresis in the lower limbs with an unsteady gait are about as common as in pseudo-sclerosis, although, as a rule, more pronounced in degree. Aphasia, present in a third of the reported cases, is unknown in pseudo-sclerosis. Facial paresis has been described in a number of instances, and dysphagia, a symptom only once observed in pseudo-sclerosis, was present in half the reported cases. Giddiness and epileptic attacks occur about as frequently in the two conditions. Muscular atrophy has been described in a few cases of diffuse sclerosis, never in pseudo-sclerosis. Objective disturbances of sensation are much more frequent in diffuse sclerosis, occurring in almost half the cases; incontinence of urine and of fæces are also of frequent occurrence. The average duration of life is from one to three or four years, and the course is steadily progressive.

**Diagnosis.**—The distinctive features from disseminated sclerosis are almost identical with those laid down in considering the differential diagnosis of pseudo-sclerosis from that disease. Pseudo-sclerosis may, in certain instances, possibly be distinguished clinically from diffuse sclerosis, but it must be remembered that cases are met with in which one disease runs into the other.

**Tuberose or Hypertrophic Sclerosis.**—Bourneville[1] described in 1880, under the title "Sclerose tubereuse des circonvolutions cérébrales," a condition characterized by overgrowth of the neuroglia in circumscribed islets which project from the surface of the cortex cerebri and from the walls of the lateral ventricles. Peculiar subcapsular tumors of the kidney are also met with. The disease is probably dependent upon a developmental anomaly. Sailer[2] has collected 28 cases. A. W. Campbell[3] has analyzed 20 cases, and found that in no less than seventeen there was idiocy or imbecility, and that congenital mental weakness could not be excluded even in the remaining three. This author refers to the constant occurrence of adenoma sebaceum, a congenital affection of the skin, affecting especially the region of the nose and the naso-labial folds. The occurrence of adenoma sebaceum in a young patient suffering from amentia and epileptic attacks would probably justify a diagnosis.

**Miliary Sclerosis.**—Under this name Gowers[4] described a case in which "throughout both hemispheres of the brain, and in all parts of them, the cortex contained minute reddish-gray spots at the junction of the gray and white substance." There was wasting of the nerve elements and an increase in the connective tissue in the affected areas. The patient was a man, aged fifty years; the chief symptom was general weakness of the limbs, with some rigidity and a few unilateral convulsive attacks. The speech became mumbling and unintelligible, and the patient became comatose and died. There was a history of syphilis. The total duration of the symptoms was about ten weeks. Greiff has described similar appearances in general paralysis of the insane.

[1] *Archiv. de Neurol.*, 1880, i, 91.
[2] *Journal of Nervous and Mental Disease*, 1898, xxv, p. 402.
[3] *Brain*, 1905, xxviii, p. 382.
[4] *Lancet*, 1886, i, 145.

# CHAPTER V.

## THE DISEASES OF THE MENINGES.

By EDWIN BRAMWELL, M.B., F.R.C.P. (Lond. and Edin.).

**Anatomical and Physiological Considerations.**—The central nervous system is invested by three membranes: the dura mater, the arachnoid mater, and the pia mater.

The dura mater, a dense fibrous tissue membrane, divisible into two layers and lined on its inner surface by endothelium, lies in close contact with the inner table of the skull and is continued as a fibrous sheath on to the cranial nerves. Three prolongations of the dura mater, the falx cerebri, tentorium cerebelli, and falx cerebelli, extend into the cranial cavity and play an important role as supporting structures. The dura mater is for the most part irrigated by the middle meningeal artery, while the trigeminus is its source of nerve supply.

The arachnoid mater and pia mater, the leptomeninges, are commonly described as the "pia arachnoid," since diseased processes which affect the one always involve the other. The pia mater is a thin vascular layer of fibrous tissue, which lies in the closest contact with the outer surface of the central nervous system. A prolongation of the pia mater, the velum interpositum, passes into the transverse fissure of the brain, carrying with it the choroid plexuses. The arachnoid, on the other hand, is a non-vascular fibrous tissue membrane. It lies external to the pia mater, with which it is connected by a delicate meshwork of fibrous tissue strands, and it is covered on its outer surface by a layer of endothelium. The arachnoid does not dip into the sulci as does the pia, but bridges them over. The inner surface of the ventricles is lined by a layer of endothelium known as the ependyma.

The subarachnoid space which lies between the pia and arachnoid contains the cerebrospinal fluid. Its meshes communicate freely with one another. The two membranes are widely separated where they lie between the cerebellum and medulla, and the space so formed is sometimes known as the posterior subarachnoid space or reservoir. The foramen of Magendie and the two adjacent foramina of Luschka offer the only means of communication between the subarachnoid space and the intraventricular system.

The cerebrospinal fluid is a perfectly clear fluid with a slight alkaline reaction and a specific gravity of 1006. Its principal constituent is sodium chloride. Traces of carbonates and phosphates are also present. It contains traces of globulin and of urea, also a Fehling reducing substance which is probably glucose. With the exception of a few lymphocytes it is devoid of cellular elements. Although it is held by some that the cerebrospinal fluid is a transudation, the researches of Halliburton as to its chemical composition afford strong evidence for believing that it is not an exudation, but a true secretion. The fluid is in all probability secreted by the epen-

dymal cells, which cover the choroid plexuses, and the secretion is probably a continuous process. The amount produced in the twenty-four hours has been variously estimated, some observers placing the quantity as high as one or two liters. Observations upon this point are possible when after certain cerebral operations the cerebrospinal fluid which escapes from the wound can be collected. It is, however, somewhat doubtful whether these results can be regarded as a true estimate of the normal secretion, since the operative procedure and the pathological condition for which it has been undertaken may alter its amount (Cushing). The fluid fills the whole intraventricular system, and through the foramina of Magendie and Luschka has free access to the cerebrospinal subarachnoid space. Since the secretion of fluid is in all probability a continuous process, it follows that in order to regulate the intracranial pressure there must be a constant return of the fluid into the blood stream. This may take place by the lymphatics of the outgoing cranial and spinal nerves, the fluid passing by way of the thoracic duct into the venous circulation. Key and Retzius believed that Pacchionan bodies act as filters through which the fiuid finds a path of egress. Cushing doubts this, since these structures do not exist in the lower animals or in young people. The last named observer believes with Leonard Hill that the fluid passes directly through the walls of the superior longitudinal sinus. Ford Robertson has demonstrated a network of large capillaries immediately subjacent to the surface endothelium of the dura, which capillaries are surrounded by spaces regarded by him as lymph channels. He believes that the cerebrospinal fluid may escape into these spaces, which he terms the perivascular canals of the dura, and thence into the veins of the skull.

## DISEASES OF THE DURA MATER.

### PACHYMENINGITIS.

The diseases of the dura mater are naturally divisible into two groups, according to the situation of the disease, whether on the outer or inner surface of this membrane.

**Pachymeningitis Externa.**—Inflammation of the outer surface of the dura mater is uncommon. It occurs almost always as a secondary consequence of neighboring disease. A cerebral and a spinal form are described.

**Cerebral.**—The outer surface of the cerebral dura mater lies in close contact with the inner table of the skull, hence external pachymeningitis in this situation is always localized. It may be a sequel to fracture of the skull with extravasation of blood on the outer surface of the dura, erysipelas of the scalp, syphilitic caries, or tumors of the cranium. The extension of an inflammatory process arising in the labyrinth or mastoid cells may result in a localized external pachymeningitis or an extradural abscess. The clinical features are usually overshadowed by those of the primary affection, to which it owes its origin.

**Spinal.**—The dura mater which surrounds the spinal cord does not lie in contact with the bony canal, but is separated from it by a space, which is occupied by loose areolar tissue. Hence pachymeningitis externa spinalis

may be either widespread or localized. In the great majority of cases the process is secondary to disease of adjacent structures. Gowers recognizes a primary form; he says: "It is certain that acute general external meningitis occurs as a primary disease and may run an intensely rapid course with profuse suppuration between the membrane and the bone. The subjects have generally been young, ill-nourished adults, and when any exciting cause has been traced this has usually been exposure to cold." A bedsore perforating the sacrum may set up an acute and extensive perimeningitis; the infection may even penetrate the dura and lead to a leptomeningitis. Very rarely does a leptomeningitis extend through the dura and cause inflammation on its outer surface. Primary acute osteomyelitis or periostitis, actinomycosis, syphilitic disease of the vertebræ, or an aneurism which has eroded the vertebral bodies, may determine a localized external pachymeningitis. Tuberculous disease of the spine is a much more common cause. In the great majority of instances the disease originates in the surrounding bone, and extends, it may be, for a considerable distance up and down the cord. The dura which is often thickened is surrounded by a mass of tuberculous granulation tissue, in which caseous foci and abscesses of variable size may be contained. The tuberculous tissue is firmly adherent to the dura, the inner surface of which is usually smooth. The dura, however, may be infiltrated, and the diseased process may even extend through to its inner surface. In very rare cases tuberculous pachymeningitis is a primary affection (Schlessinger).

**Symptoms.**—The *symptoms* vary according to the nature and extent of the lesion upon which they depend. Thus when the process is acute and diffuse, pain in the back, root pains, muscular spasms, and hyperæsthesia, together with an elevation of temperature, are met with. Again, when the pachymeningitis is localized and chronic, symptoms indicative of increasing pressure on the spinal cord, similar to those which occur in the case of a tumor, are produced. Indeed, in cases of tuberculous pachymeningitis in which there is no spinal curvature or evidence of disease of the vertebræ or of abscess formation the diagnosis from tumor may be difficult if not impossible.

**Treatment.**—The treatment is that of the primary disease.

**Pachymeningitis Interna.**—A purulent internal pachymeningitis occurs as a very rare result of an extension from inflammation of the leptomeninges. Osler has met with a pseudo-membranous inflammation of the lining membrane of the dura as a secondary process in pneumonia.

**Pachymeningitis Interna Hæmorrhagica.**—This rare condition is sometimes known as hæmatoma of the dura mater. It is customary to describe it with inflammatory diseases of the meninges, although it is not unlikely that the initial change is a hemorrhage from the inner surface of the dura.

**Pathology.**—The pathology of the condition has evoked considerable discussion. A clinical diagnosis is rarely made, and even when recognized during life it is doubtful whether therapeutic measures have any distinct influence in arresting its progress. Huguenin's article in von Ziemssen's *Cyclopædia* still remains the classical monograph upon the subject. Indeed, during the past thirty years little of material importance has been added to our knowledge of the disease.

The condition is rarely seen in the postmortem room of a general hospital, though not uncommon in asylum practice; thus in 1185 postmortems at the Government Hospital for the Insane, Washington, there were 197 cases

with "a true neomembrane of internal pachymeningitis" (Blackburn). The disease is an affection of later life. Of the cases collected by Huguenin, 41 per cent. were between sixty and eighty, and 31 per cent. between forty and sixty years of age. During the first year of life 2.7 per cent. occurred; injury to the cranium during delivery no doubt accounts for this. Males suffer more frequently than females, in the proportion of three to one (Durand-Fardel).

A great variety of associated morbid conditions have been met with. Among Blackburn's 197 cases in 45 there was chronic dementia, in 37 general paralysis, in 30 senile dementia, in 28 chronic mania, in 28 chronic melancholia, in 22 chronic epileptic insanity, in 6 acute mania, and in 1 case imbecility. Krönig, who analyzed 135 cases examined post mortem at the Berlin Pathological Institute, found phthisis (23 per cent.), general paralysis of the insane (19 per cent.), cardiac valvular disease (18 per cent.), syphilis (11 per cent.), puerperal sepsis (9 per cent.), and chronic alcoholism, carcinoma, and infectious diseases (about 6 per cent. each). Hæmophilia and scurvy are also recognized causes, as are diseases of the blood, especially pernicious anæmia. Traumatism is sometimes a determining factor (Schneider, 17 out of 74 cases). According to Huguenin, the great majority of cases occur with changes which cause a reduction in the volume of the brain, especially general paralysis of the insane. It is reasonable to suppose that the loss of support to the dural vessels which must accompany wasting of the brain predisposes to hemorrhage from the dural vessels. Further, disease of the vessels is usually demonstrable in association with the pathological states which give rise to shrinkage of the cerebrum—dementia paralytica, senile atrophy of the brain, chronic alcoholism. The congestion of the brain which is often associated with cardiac valvular disease and abnormal conditions of the blood, such as occur in pernicious anæmia, must also render the liability to hemorrhage more likely, while it is probable that the increased intracranial pressure associated with the act of coughing may, as Huguenin suggests, account for the association with phthisis.

Pachymeningitis interna hæmorrhagica was regarded by the older writers as primarily due to hemorrhage on the inner surface of the dura. This view was actively combated by Heschl and especially by Virchow. The latter held that the first change was a hyperæmia of the inner surface of the dura and that the membrane was a highly vascular, inflammatory new formation, between the successive layers of which hemorrhage occurred. This was the accepted teaching for twenty years, when Huguenin (1876), who examined the condition in its earliest stages in cases of general paralysis, asserted that the first discoverable appearances consisted in a thin layer of coagulated blood covering the inner surface of the dura, that at this stage there were no connecting vessels between the blood clot and the dura, and that the internal surface of the dura presented a perfectly healthy appearance.

On postmortem examination there may be only a thin red or grayish-red film on the inner surface of the dura mater. The convexity of the cerebrum is its most common situation, although it may extend over a considerable extent of one or both hemispheres. Among 54 cases personally examined by Wigglesworth, 20 were strictly unilateral and 34 were bilateral, while 56 per cent. of the cases observed by Huguenin had a bilateral distribution. The membrane may extend to the base of the brain and is not uncommonly

met with on the upper surface of the tentorium, although very rarely beneath it (Wigglesworth). In more advanced cases it may be of considerable thickness, consisting of a series of laminæ which have been successively deposited, the most recent layers being next the cerebrum. There may be as many as twenty of these laminæ, of which those of later origin are of a reddish color, the older layers being paler. Between the individual layers collections of fluid blood may be found. In some cases the false membrane may actually measure 2 or 3 cm. in thickness.

**Symptoms.**—These are various, and do not form a precise clinical picture. In many cases no symptoms are produced, and it may be that those which are present are obscured by those dependent on the original disease. A cerebral complication may be diagnosed, but its exact nature often remains a matter of uncertainty. The symptoms depend on the situation and extent of the morbid process. A degree of excitability with general restlessness, elevation of temperature, headache, giddiness, and vomiting may signalize the onset or extension of the lesion. The headache is sometimes very severe, and the patient may become delirious. Localized and recurring convulsive attacks, monoplegic or hemiplegic in distribution with, it may be, conjugate deviation of the eyes, paralysis or paresis of the hemiplegic or monoplegic type, slowing and irregularity of the pulse, and the gradual development of stupor and coma is a common history. The cranial nerves are rarely involved. Aphasia is occasionally present. There may be optic neuritis. Recovery may take place, to be followed by another attack.

**Diagnosis.**—The diagnosis presents the greatest difficulty. Schultze suggests that the condition may be suspected when one of the predisposing causes is present, particularly chronic alcoholism, in an individual advanced in years, in whom in addition to the stage-like development of stupefaction and coma there are cerebral and especially cortical irritative phenomena associated with paralyses, which vary in intensity within considerable limits. Hemorrhagic fluid may be obtained on lumbar puncture in traumatic cases.

**Treatment.**—The head may be elevated and ice applied, but it is very doubtful whether any known treatment tends to arrest the bleeding.

**Pachymeningitis Cervicalis Hypertrophica (Hypertrophic Internal Pachymeningitis).**—This condition, originally described by Charcot, is characterized anatomically by great thickening of the spinal membranes, more especially in the cervical region, with consequent pressure upon the spinal cord and its nerve roots. In a typical case the lower cervical portion of the cord is found to be surrounded by a dense fibrous covering, which may be five or ten times (Oppenheim) as thick as the normal dura. The cord and its nerve roots are in fact embedded in an annular fusiform tumor of dense fibrous tissue of, it may be, the consistence of cartilage, usually thickest posteriorly and consisting of concentric layers unseparable from one another. The anatomical features are similar to those in chronic syphilitic meningomyelitis. According to Wieter, the process actually originates in the leptomeninges, and should be named meningomyelitis cervicalis chronica rather than pachymeningitis, since the dura is only secondarily involved. Pressure on the nerve roots induces necrotic changes in them with increase of interstitial tissue. The cord is flattened anteroposteriorly and ultimately ascending and descending degenerations are produced. Cavity formation, a consequence of intramedullary softening, is sometimes observed. It must not be supposed, however, that the process is limited to the cervical region.

True, it is usually most pronounced here, but it often extends through a considerable extent and occasionally implicates the medulla and pons.

There is little known with regard to *etiology*. Cold, injury, and overexertion are all cited as possible factors. Syphilis appears to have a very definite relation, and, as before stated, syphilitic meningomyelitis is indistinguishable pathologically.

Neuralgic pains, often very severe, and referred to the distribution of the posterior roots entering the cord at the level of the lesion, are usually the earliest *symptoms*. This is the neuralgic stage of Charcot. At a later date, local and increasing muscular weakness, with wasting and impairment or loss of sensation, a consequence of interference with conduction in the spinal nerve roots, result. The small muscles of the hand and the flexors of the fingers and wrist are usually, because of the situation of the lesion, first affected, hence he deformity known as the "preacher's hand," which is not uncommon. Later still a third stage is reached, which is characterized by interference with conduction in the spinal cord, spastic paraplegia with increased reflexes, contractures, anæsthesia of the lower extremities, loss of power over the bladder and rectum, and bedsores. When the process involves the medulla and pons, various cranial nerves may be implicated.

The very gradual development of severe pains in the neck and arms, succeeded by weakness, muscular wasting, and, it may be, objective sensory disturbance having a root distribution, and at a later date by symptoms of progressive pressure on the spinal cord, is a very suggestive history when no evidence of vertebral disease can be detected.

Syphilitic pachymeningitis may be indistinguishable. A history of syphilis or the detection of other symptoms due to that disease or of a cerebrospinal lymphocytosis are points of value in differential diagnosis. Tuberculous pachymeningitis may present an identical clinical picture when there is no evidence of disease of the bone. The detection of tuberculous lesions in other parts of the body, an *x*-ray photograph of the spine, and the tuberculin test may be of value under these circumstances. When pressure symptoms are due to a simple tumor of the meninges, they are often unilateral.

Syringomyelia and amyotrophic lateral sclerosis may closely simulate hypertrophic pachymeningitis, but these conditions are unaccompanied by the severe pains in the neck and arms, which are so characteristic of the affection under consideration. The co-existence of syringomyelia and pachymeningitis has been noted.

A thorough antisyphilitic course of *treatment* is called for in any case in which there is a possibility of syphilis. Counterirritation with the cautery over the lower cervical vertebræ is sometimes useful. The various analgesics may be used to relieve the pains, which will often, however, only yield to morphine.

## DISEASES OF THE LEPTOMENINGES.

### ACUTE CEREBROSPINAL MENINGITIS.

**Etiology.**—Cerebrospinal fever, tuberculous meningitis, the acute affection of the meninges met with as an occasional sequel of syphilis, and the amicrobic serous meningitis of Quincke are described elsewhere. A

large group of cases, sometimes included under the comprehensive title of Acute Purulent Meningitis, remain for consideration. These may be subdivided according to their mode of production and with special reference to the causative agents upon which they depend. The causes are very varied, and yet the distribution of the inflammatory process, its naked-eye and microscopic characters, and the accompanying symptoms present no constant features distinctive of the individual infections.

Leptomeningitis may be produced by many organisms, yet if we except the types due to the meningococcus, pneumococcus, streptococcus, and the tubercle bacillus, the general statement holds good that acute cerebrospinal meningitis is of very rare occurrence. Councilman in 60 consecutive cases of the disease found the following organisms: Pneumococcus (18), streptococcus (18), meningococcus (21), staphylococcus (2). In two instances the nature of the bacterial infection was undetermined.

Acute leptomeningitis may be of primary or secondary origin. The latter group of cases may be subdivided according to the conditions with which they are associated.

Primary leptomeningitis, apart from cerebrospinal fever, is of very rare occurrence, and is almost always due to the pneumococcus. Thus, Marchal, quoted by Weichselbaum, found that the meningococcus was responsible for 50.5 per cent. and the pneumococcus for 42 per cent. of the primary cases of sporadic meningitis. The mode of entrance of the organisms in primary meningitis is a debatable question. Infection by way of the nose is very probable in some cases. It is to be remembered that two factors are necessary in order that a primary inflammation of the meninges may be produced: (1) the infective organism must be present, while (2) predisposing causes resulting in lowered resistance such as traumatism, insolation, exposure to cold and alcoholism, must also exist (Weichselbaum).

Secondary meningitis may be associated with a variety of conditions. Trauma is a not uncommon factor; thus a leptomeningitis may be directly produced by a penetrating wound or a compound fracture of the skull. The manner in which the organisms obtain access to the meninges under these circumstances is obvious. Infection through some of the accessory sinuses may account for cases in which a meningitis follows a fracture of the skull in which the skin surface has not been broken.

Extension from adjacent disease is responsible for many cases of secondary meningitis. An acute or chronic otitis is much the most common factor in this connection. The pus may pass by direct extension through the bone, or the infection may spread by way of the sheaths of the facial or auditory nerves, or be carried by a suppurative phlebitis. When the infective process stops short of the dura a serous meningitis which shows no tendency to become purulent may result. If it perforates the dura an encapsuled meningeal or brain abscess or a general purulent meningitis follows. Much more rarely disease in the nasal pharynx, the orbit or frontal sinuses is the focus from which the inflammation arises. Erysipelas of the scalp or a carbuncle in this situation are occasional causes. A generalized meningitis may be set up by an abscess of the brain, even though rupture has not taken place. Cases are recorded in which a sacral bedsore has by direct extension involved the dura and given rise to an acute spinal leptomeningitis.

In the course of the infective fevers meningitis is met with as an occasional complication. It is to be remembered that a meningitis so occurring is not

necessarily due to the same cause as the original disease, for mixed infections are not uncommon (Fürbringer). Pneumonia is the acute fever in which meningitis is most often met with. Osler found meningitis in 8 of 100 autopsies, while among 253 cases of pneumonia examined after death, Aufrecht met with 7 in which meningitis was present. Musser and Norris found a postmortem record of meningitis in 180 of 4833 cases of pneumonia. An acute meningitis is sometimes met with in septicæmia. Its occurrence in erysipelas is much rarer than was at one time supposed. Thus Anders in his analysis of 1674 cases of this disease does not refer further to it than to remark that it is an occasional complication. Typhoid fever is very rarely complicated by true meningitis. Cole has collected 21 cases of meningism, serous and purulent meningitis, from the literature, and 6 instances were present in the Johns Hopkins Hospital series of 1500 cases of typhoid fever analyzed by McCrae. Of eight cases in which typhoid bacilli were cultivated from the cerebrospinal fluid, in only one were they found on microscopic examination of the fluid. Dubois, in 1903, collected 11 cases in which a purulent meningitis was due to Pfeiffer's bacillus. The rarity of influenzal meningitis may be gathered from the fact that of 55,263 cases of this disease occurring in the German army, meningitis was only noted in four. There are eight cases of primary suppurative meningitis in scarlet fever recorded in the literature (Teissier, Boudon and Duvoir, 1908). G. Henderson and W. T. Ritchie (1908) have reviewed the reported cases of gonorrhœal meningitis. Vennet (1908) has collected the cases of meningitis occurring in association with mumps.

The occurrence of meningitis in measles, smallpox, ulcerative endocarditis, acute rheumatism, actinomycosis, and anthrax has also been recorded. Lastly, acute meningitis may be associated as a terminal infection with such diseases as chronic nephritis, arteriosclerosis, heart disease, gout, and the wasting diseases of children (Osler).

**Pathology.**—As its name implies, acute cerebrospinal leptomeningitis is an inflammatory process which implicates the spinal as well as the cerebral meninges. The degree and distribution vary in different cases; in some the base of the brain, in others the vertex is chiefly affected. The simple non-tuberculous meningitis of children is basal. Otitic meningitis when it becomes generalized is almost invariably basal. The meningitis which occasionally occurs in gout is usually basilar, as is that which is seen in chronic nephritis and in cachectic conditions (Osler). The statement sometimes made that a meningococcal meningitis may be distinguished from one due to the pneumococcus by the fact that the former is chiefly basal, the latter vertical in distribution, can only be accepted very generally, for in the meningococcus cases the exudation is sometimes chiefly confined to the convexity of the cerebrum, while in the pneumococcal variety the base is often and may be alone involved. Thus among Nauwerck's 27 pneumococcal cases the base alone was involved in 4, the convexity and base in 16, while in 7 the convexity, base, and cord were all implicated. When a leptomeningitis is secondary to adjacent disease, as, for example, otitis media, the process may remain for a time confined to the corresponding hemisphere, but in the great majority of cases of acute leptomeningitis, whatever the cause, the infection soon becomes general. There are no absolutely distinctive anatomical features which justify a positive opinion as to the bacteriological nature.

The earliest visible change is a hyperæmia of the pia arachnoid, which is

soon followed by some cloudiness of these membranes. Accompanying this is an excessive secretion of cerebrospinal fluid at first clear, later turbid, and ultimately, it may be, purulent. Inflammatory exudation occurs along the course of the engorged vessels. The yellow lymph may gradually spread from the sulci over the convolutions. Finally, the surface of the brain may be covered, as is not uncommon in pneumococcal cases, with a thick layer of greenish pus. It must not be supposed that the underlying brain escapes. An inflammatory œdema of the cortex is almost always present, and may go on to a true encephalitis with hemorrhages, hence the term meningo-encephalitis so often applied. Sooner or later a serous or seropurulent exudation occurs into the ventricles, the ependyma of which may be covered with lymph. When communication between the ventricles and subarachnoid space is interfered with, a degree of ventricular distention develops, which, however, rarely reaches the marked hydrocephalus met with in the tuberculous and serous variety. In some very acute cases death occurs before the exudation takes on a purulent character, while in other cases in which recovery takes place the cerebrospinal fluid from first to last may never present more than a slightly turbid appearance.

The microscopic changes in acute cerebrospinal meningitis are practically identical with the appearances described in detail under cerebrospinal fever. The reader is referred to the writings of Faure and Laignel-Lavastine and Councilman, and to the elaborate monograph by Thomas for an exhaustive account of the subject. Engorgement of the meningeal and cortical vessels, the walls of which are seen to be infiltrated with leukocytes, is the most striking feature. The inflammatory process extends along the course of the bloodvessels into the brain substance. The neuroglia commonly shows some increase, while very pronounced changes in the pyramidal cells of the cortex are commonly present (Voisin). An acute proliferative inflammation of the veins and arteries is often seen in the forms of meningitis due to the pneumococcus and streptococcus (Councilman).

**Symptoms.**—The symptoms in acute purulent meningitis are very numerous and vary to some extent with the distribution, nature, and intensity of the process. Sometimes there is a premonitory stage in which such symptoms as irritability, restlessness, somnolence, malaise, headache, and vomiting are complained of. When the meningitis occurs in the course of an acute disease, such as pneumonia, the earlier symptoms, if slight, may be obscured by those of the primary affection. Sometimes the first indication is intense headache, cerebral vomiting, or a general convulsion, followed, it may be, by delirium and rapidly deepening coma. A rigor is not uncommon at the outset, especially when the ventrical meninges are attacked. Two stages are at times recognizable, in the first of which irritative phenomena predominate, while in the latter the manifestations are of the paralytic order.

Headache, frequently the earliest as well as the most distressing symptom, is rarely absent. It is, as a rule, widespread, although often referred especially to the frontal or occipital regions. When the meningitis is due to extension from adjacent disease, the headache may remain localized for a time. The pain is often very severe, the patient, even when in a semicomatose state, putting his hand to his head and crying out. The "meningeal cry" which is described in this disease is probably to be associated with intense paroxysms of pain. "Cerebral vomiting," a common symptom, often develops at the outset and may persist. Occasionally it is absent. In a case recently seen

by the writer, vomiting and associated abdominal pain, together with constipation, had suggested the possibility of acute intestinal obstruction. Delirium is often present and may appear at an early date, especially in vertical cases; it is sometimes violent. The patient is in some cases, even from the first, in a somnolent state, which gradually passes into coma.

Photophobia and hypersensitiveness to sounds are common early symptoms, while cutaneous and muscular hyperæsthesia is almost invariably present at some stage.

Rigidity of the neck, with pain on movement and perhaps head retraction, the latter symptom being rarely, however, so extreme as the retraction met with in the meningococcal form, is the rule in those cases in which the meningitis is basal. When the meninges of the cord are prominently involved, rigidity of the back with opisthotonos may be observed.

The temperature is usually raised, although it is exceptional to find it over 103° unless immediately prior to death, when hyperpyrexia is occasionally present. Sometimes, on the other hand, the temperature falls below the normal at this time, while very exceptionally there is no rise from first to last.

The pulse, although sometimes increased in frequency, is usually slow at the commencement of the illness and not uncommonly irregular both in time and force. Later it is often small and rapid. Considerable variations in the pulse rate within a short period are often to be noted. A pulse rate which is unduly slow in comparison with the height of the temperature is a striking feature in some cases. The frequency of the respirations may be increased, and they are often irregular. Cheyne-Stokes breathing may be observed, especially in the event of an approaching fatal termination.

Convulsions of the Jacksonian type are not uncommon, while fits, unilateral or generalized, are prone to occur in children, and especially, according to most authors, in cases in which the meningitis is vertical in distribution. It is interesting to note in this connection that convulsions were not present in a single instance of the cases of pneumonia, ulcerative endocarditis, or septicæmia complicated with meningitis observed by Osler. Choreic movements are very occasionally seen. Paralysis is far from infrequent in the later stages, and would doubtless be more often recognized were it not for the coma which often obscures it. Reinhold in 53 cases found hemiparesis fourteen times and a monoplegia on three occasions. The motor weakness may be associated with rigidity. Aphasia is an occasional symptom.

Kernig's sign is, as a rule, present; thus, Netter found it in 41 of 46 cases. This sign is, it must be remembered, by no means pathognomonic of a meningeal affection.

Optic neuritis is fairly frequent in basal cases, although pronounced papillitis, such as is seen in cases of intracranial newgrowth, is very rare. The pupils are often unequal, and are apt to vary much in size from time to time. In the early stages they are usually contracted, while at a later date dilatation is the rule. Reflex iridoplegia is sometimes observed. Strabismus and ptosis are frequent, nystagmus not uncommon. Conjugate deviation of the eyes is occasionally met with. Paresis of the face, often fugitive, and accompanied at times by tremor in the affected muscles, is sometimes noted. Trismus and trophic ulceration of the cornea indicating involvement of the fifth nerve are more unusual symptoms. Dysphagia is by no means rare.

The tendon jerks, generally increased in the early stages, are usually lost

later in the course. The plantar reflex may be of the extensor type. Evidence of vasomotor irritability is forthcoming in flushing of the skin and in the *tache cérébrale*, which is usually pronounced. Constipation is almost invariable. Sometimes there is retention of urine, while later there may be incontinence. Glycosuria has been recorded. Rapid emaciation is very often observed. The leukocyte count is commonly high although notable exceptions occur.

Upon lumbar puncture a turbid fluid, which emerges under pressure, is usually obtained, even in the early stages; occasionally, however, the fluid is quite clear, very rarely it is so thick it will not flow through the needle. The fluid is found to contain large numbers of cells, the great majority of which are polymorphonuclears, with, in addition, perhaps, the specific organisms which are responsible for the meningitis.

The clinical picture of acute leptomeningitis varies greatly. The onset in some cases is very sudden and is associated with convulsions and delirium. Again, the early development of coma is sometimes the striking feature. In other cases the symptoms develop much more gradually.

**Diagnosis.**—Acute leptomeningitis may be suspected when cerebral symptoms, such as headache, "cerebral vomiting," irritability, general hyperæsthesia, delirium, and convulsions, develop in association with the phenomena attendant on a febrile process. All these symptoms may, however, occur in the course of some of the specific fevers, notably pneumonia, typhoid, and influenza, as a result of congestion of the meninges. Rigidity of the neck and Kernig's sign increase the probability of meningitis, while the additional presence of local palsies of the limbs or cranial nerves affords strong corroborative evidence of a meningeal inflammation.

Schultze holds that headache, rigidity of the neck, and hyperæsthesia constitute a symptomatic triad, in the absence of which the diagnosis of meningitis is uncertain. Corroborative evidence as to the existence of a meningitis is forthcoming if on lumbar puncture a turbid cerebrospinal fluid containing organisms is obtained. According to Körner, a thick purulent fluid containing organisms means a diffuse purulent leptomeningitis; a slightly muddy fluid in which organisms are present may occur either with a localized or diffuse meningitis, while a clear fluid with no pus cells or bacteria signifies either that there is no meningitis present or that the meningeal inflammation is localized. Apart from cerebrospinal fever, the various forms of acute generalized meningitis cannot be said to present symptomatic features distinctive of their cause. The nature of a meningitis may, however, be suspected when it occurs as a complication in the course of co-existing disease, or determined if specific organisms are detected in the cerebrospinal fluid. In doubtful cases the serum should be examined for agglutinins and inoculation experiments carried out.

*Meningism* or *pseudo-meningitis* is a term applied to a symptom complex met with in the course of some of the infective fevers, which is characterized by symptoms indistinguishable from the general symptoms of meningitis, and yet when opportunity is afforded of examining these cases post mortem no inflammatory changes are detected. In some of these cases there may be a slight degree of meningitis actually present which is recovered from; in many there is probably merely a meningeal hyperæmia; while in others the symptoms would appear to be due to the direct effects of a toxin upon the brain itself. Meningism may be distinguished from true meningitis by the fact

that although the cerebrospinal fluid may emerge under pressure, it presents normal appearances.

*Hysteria* may simulate meningitis closely, and the diagnosis may be very difficult when hysterical symptoms occur in the course of an infective disorder. Tuberculous meningitis is, according to Gowers, especially apt to be mistaken for hysteria, while the reverse mistake is seldom made. Such signs of organic disease as optic neuritis, ankle clonus, Babinski's sign, and nystagmus are of great importance in diagnosis, while a turbid fluid obtained on lumbar puncture will always determine the point at issue.

*Acute suppurative otitis* sometimes presents a picture identical with that of acute meningitis. This is especially so in children, for delirium and convulsions, symptoms so apt to occur in early life in any acute febrile disturbance, may be accompanied by headache, vomiting, and giddiness due to the otitis. Here, again, lumbar puncture will be found to be of value.

An intracranial *abscess* may give rise to symptoms very similar to those of meningitis. Further, abscess and meningitis may co-exist. An acute onset, with high temperature, rapid pulse, hyperæsthesia, irritative phenomena, and a high leukocytosis are in favor of the latter affection, as are head retraction, rigidity of the spine, and Kernig's sign. When there is no co-existing ear disease the likelihood of abscess is small. In an uncomplicated case of abscess the cerebrospinal fluid is clear and contains few cells. A localized meningitis due to ear disease may be indistinguishable from abscess.

*Tuberculous meningitis* sometimes gives rise to difficulty, and a discharge from the ear does not exclude tuberculous meningitis, for the ear disease is sometimes due to the tubercle bacillus. Tuberculous lesions are usually present elsewhere in the body, hyperæsthesia is commonly absent, and these cases, as a rule, run a more chronic course. The absence of a leukocytosis favors tubercle (Cabot found a leukocytosis in 32 of 43 cases of tuberculous meningitis). The tuberculin reaction may prove of value, although it must be remembered that in acute tuberculous cases this test may give a negative result. Choroidal tubercles when present make the diagnosis certain (Koplik found choroidal tubercles in 9 of 46 cases in the first two days of the illness). The cerebrospinal fluid is usually clear in tuberculous meningitis, and in contrast to the purulent form the increase of cells which is often present is found to consist almost entirely of mononuclear elements. Bernstein found the tubercle bacillus in the fluid obtained on lumbar puncture in 100 cases of 102 in which he looked for it.

The differential diagnosis of meningococcal meningitis from other forms of acute purulent meningitis has been considered in a previous volume.

The delirium of alcohol and the uræmic state may resemble acute meningitis, although it very rarely happens that the diagnostic problem is attended with much difficulty.

**Prognosis.**—The prospect of recovery from an acute purulent leptomeningitis is small. Death is almost invariable in cases with a sudden onset. In any form if the stage of coma has been reached, death is all but certain (Gowers). If the meningitis is localized the outlook is more favorable than when a general meningitis exists. The prognosis varies with the nature of the bacterial infection. The probability of recovery is greater in cerebrospinal fever than in purulent meningitis due to other causes. Recovery from pneumococcal meningitis, whether primary or secondary

in origin, is most unusual. Thus of the 33 cases collected by Musser and Norris, 31 (93.3 per cent.) died, while only 1 of the 11 cases described by Cantley recovered. Death occurred in all of Nauwerck's cases. The chances of recovery are greater in a staphylococcal than in a streptococcal infection.

Macewan fifteen years ago reported 12 cases of local otitic meningitis upon which he had operated, in 6 of which recovery took place. Cases of otitic meningitis, with pus and staphylococci in the cerebrospinal fluid, may recover after operative treatment of the purulent focus in the petrous bone (Körner, Alexander). The duration of acute meningitis varies from a few hours to two or three weeks. Of the 65 cases collected by Netter, 54 died within four days.

**Treatment.**—This is very similar to that of cerebrospinal fever. It may be that in the future vaccine and serumtherapy will have a recognized place. At the present time, the management consists in the adoption of general measures and the alleviation of symptoms. The patient must be kept absolutely at rest in a quiet, darkened room; a dose of calomel may be given with possible advantage if the case is seen early. The head should be shaved and an ice-bag applied to it. Counterirritation is generally considered to be of service, and with this object a blister may be applied to the back of the neck. Potassium iodide in doses of five to ten grains is employed by some authorities, while mercury and perchloride of iron are, in the opinion of others, of possible value. There appears to be no evidence to show that injections of collargol and other substances into the subdural space are followed by any beneficial effect.

Headache and convulsions may be relieved by bromide and chloral. Morphine may be required when the headache is very severe. Vomiting is sometimes alleviated by a mustard leaf to the epigastrium or by giving the patient ice to suck. For general irritability, hyperæsthesia, and delirium, warm baths are undoubtedly of value. When the temperature reaches an abnormally high point, tepid sponging should be employed.

When the meningitis is secondary to trauma or ear disease, surgical measures should be promptly resorted to.

Lumbar puncture is certainly of value in relieving the headache, and the patient may even be roused from a semicomatose state. The procedure is unattended with risk if care is taken not to lower the pressure of the cerebrospinal fluid below the normal. A marked amelioration of the symptoms sometimes follows the withdrawal of 10 cc. Cases have been recorded by Körner and others in which repeated tappings appeared to have exerted a beneficial influence. Leonard Hill has suggested irrigation of the membranes through openings in the cranium and lower part of the spinal cord. The establishment of a through and through current has been attempted, but as a matter of practice it has been found to be impossible (Cushing).

**Chronic Leptomeningitis.**—The chronic inflammations of the meninges call for passing notice. Syphilis is the most common cause. A chronic inflammation of the meninges is also met with in confirmed alcoholics and in general paralysis of the insane. Tuberculous meningitis sometimes runs a chronic course. The duration in purulent meningitis is, as we have seen, sometimes protracted, especially when the inflammation is localized, and necessarily in cases which ultimately recover. Posterior basic meningitis is a chronic disease, and in cerebrospinal fever the symptoms may persist for many months before death or recovery takes place. A localized chronic

spinal leptomeningitis occurs in connection with caries of the spine and other forms of pressure paraplegia.

**Symptoms.**—The *symptoms* of chronic leptomeningitis vary greatly. In the syphilitic and tuberculous forms they often simulate closely those of tumor. The meningitis of chronic alcoholism may be suspected by its association rather than by any characteristic symptoms which it produces. The treatment varies with the cause.

**Localized Serous Spinal Meningitis.**—This is an affection which recently has attracted some attention. Cases have been recorded by Krause, Oppenheim, Bruns, Kurt Mendel, Spiller, Adler, and others. Horsley, who has recently reviewed the literature of this affection under the name of chronic spinal meningitis, states that he has operated on 21 cases. A circumscribed collection of subarachnoid fluid is found pressing on the cord. In some cases it would appear that the condition is related to an inflammatory process, trauma, or syphilis; Bruns, however, is strongly of opinion that it may result as a primary idiopathic affection apart from disease of the spine, membranes, or cord. The *symptoms* are indistinguishable from those of tumor. Krause has collected 8 cases in which the Brown-Séquard syndrome and other symptoms led to laminectomy for the removal of a tumor, instead of which a localized serous meningitis was found. The same author, in 22 laminectomies performed for the removal of a supposed tumor, found in no less than six that a localized serous meningitis was the essential if not the only cause of the paralytic phenomena. In any case in which this condition is suspected an operation should be undertaken, since a subsidence of the symptoms usually follows.

**Lumbar Puncture and the Examination of the Cerebrospinal Fluid.**—The operation is a very simple one, it causes little pain, and with due precautions is unattended with risk. The patient sits astride a chair, over the back of which he leans, or, better still, he lies on his side with his knees fully flexed upon his abdomen and his spine well curved. The fourth lumbar space is that usually selected for the puncture. The cord cannot be injured, since it terminates opposite the second lumbar vertebra. Further, there is more room between the vertebræ here than at a higher level, while another reason for choosing this situation is that the dura is more firmly adherent to the bone here than elsewhere, hence there is little chance of pushing the membrane in front of the needle. A line joining the highest point of the iliac crests passes between the fourth and fifth lumbar spines. The needle should be three and a half inches in length and made of steel or of platinum with an iridium point, the advantage of the latter being that it can be boiled. The skin should be very carefully sterilized. A general anæsthetic is unnecessary except in the case of excitable patients, but ethyl chloride may be used locally.

The needle is introduced midway between the fourth and fifth lumbar spines a quarter of an inch to one side of the middle line, and is passed almost directly forward with a slight inclination inward and upward. When the needle reaches the intervertebral ligament it receives a momentary check, but gentle, firm pressure sends it through the ligament and dura, and its point then lies in the subarachnoid reservoir. The distance traversed by the needle before it reaches the dura varies from 2 to 7 cm., according to the age and development of the patient. Should the needle come in contact with bone, the point should be withdrawn for a short distance and passed in at

a rather different angle. If after several attempts it is found impossible to avoid the bone, it is best to withdraw the needle altogether and reintroduce it in another situation. It not uncommonly happens that no fluid comes, even though the needle has been felt to pass through the ligament. Under these circumstances suction is not to be used, but a stilette should be passed along the needle, the lumen of which has probably been blocked on its way through the skin and muscles. Occasionally after this manipulation no fluid is obtained. This may be accounted for by the fact that the dura has been pushed in front of the needle, which has failed to penetrate it, or it may be that, as in some cases of hydrocephalus, the ventricular system has been shut off and there is no fluid in the subarachnoid space. Again, in some cases of purulent meningitis the fluid is so thick that it will not flow. Should the needle come in contact with a nerve root as it passes through the ligament, the patient complains of intense pain often radiating down the leg. The needle should then be partially withdrawn and reinserted.

Unpleasant effects very seldom follow the operation, provided that only a small quantity of fluid is withdrawn. Headache, sickness, or faintness is sometimes complained of, hence it is advisable that the patient should rest for an hour after the procedure. A rest of twenty-four hours after the operation, advocated by Nissl, appears in the writer's experience to be unnecessary. A number of fatal results have been recorded after lumbar puncture in cases of intracranial tumor in which there was a great increase of intracranial pressure. Almost all these fatalities took place before the importance of withdrawing only a small quantity of fluid was realized. Nevertheless, lumbar puncture should only be undertaken after very careful consideration in these cases.

Under normal circumstances the fluid emerges drop by drop and is collected in a sterilized test-tube. The first few drops are sometimes turbid, owing to the fact that a little blood has been collected in the lumen of the needle in its passage through the muscles and skin, it is therefore advisable to allow this to escape.

The pressure under which the fluid emerges is of some importance. When the pressure is raised, the fluid, instead of emerging in drops, may spurt out in a steady stream. Various instruments have been devised with which to measure the pressure of the cerebrospinal fluid (Quincke, Krönig, Eve), but the observations so obtained cannot be said to be of great practical value. Quincke states that the normal pressure equals on the average a column of water of 40 to 60 mm., but that it can only be regarded as pathological when it is above 150 mm. Krönig, on the other hand, finds the average normal pressure in the horizontal posture to be from 100 to 150 mm., while in the sitting position he puts the average at 410 mm. According to the latter writer the pressure may rise to 700 mm. in pathological states. The pressure is often very greatly raised in cases of intracranial tumor and in the various forms of meningitis. It may also be increased in uræmia and in certain of the acute infective fevers. According to Quincke moderate increase of pressure with severe pressure symptoms points to an acute, while a great rise of pressure with slight pressure symptoms indicates a chronic process.

Valuable information is often derived from the naked-eye appearance of the fluid. Thus in meningeal hemorrhage, and in hemorrhage into the ventricles, the fluid may be blood-stained. If such fluid is obtained on lumbar puncture, the question arises, Is this an accidental contamination,

or, in other words, is the color of the fluid to be explained by puncture of a bloodvessel during the passage of the needle? If the fluid is received in three different tubes, and it is found that the tint of the three is the same, probably the blood is not due to the puncture (Campbell). Again, when hemorrhage has occurred into the cerebrospinal fluid, after the fluid is centrifuged it often retains a yellowish color, whereas when the hemorrhage is accidental it presents its normal appearance (Mathieu). Hæmatoidin crystals may be found in cases in which the hemorrhage is of some standing (Krönig). The fluid may be purulent; indeed, in some cases thick pus is withdrawn. A purulent cerebrospinal fluid means a purulent meningitis or a ruptured abscess. Even when there is a purulent meningitis the fluid obtained by lumbar puncture may be free from leukocytes and organisms, no doubt because communication between the cerebral and spinal subarachnoid space has been interfered with. Hence a positive observation is alone of conclusive value. In cases of abscess the fluid is clear unless a meningitis either localized or generalized co-exists. The presence of a turbid fluid does not insure a certain diagnosis between general meningitis and a localized inflammation of the meninges associated with brain abscess, sinus phlebitis, and purulent disease of the labyrinth, for in all of these cases leukocytes and bacteria may pass into the cerebrospinal fluid. The cerebrospinal fluid in tuberculous meningitis is, as a rule, quite transparent, although it may show very slight turbidity. A yellow coloration of the fluid has been observed in jaundice.

The chemical examination yields some information of diagnostic importance. The normal fluid contains a trace of serum globulin, and when boiled becomes slightly cloudy. An abnormal excess of the albuminous content is met with in meningitis and the parasyphilitic affections of the nervous system. The amount of albumin present may be estimated by special methods described by Reiss and Nissl. Fibrin, although absent from the normal fluid, is present in acute meningitis. The Fehling reducing substance, which is constantly present in the normal fluid and in the examination for which at least 10 cc. should be used, is, according to Bernstein, always absent in purulent meningitis, while in the tuberculous variety, although present in small quantities at the commencement, it disappears in the later stages. Considerable quantities of sugar have been found in the cerebrospinal fluid in diabetes. Cholin, a product of myelin degeneration, has been detected in the blood and cerebrospinal fluid by Mott and Halliburton in cases of acute degenerative diseases of the nervous system. This observation has been confirmed by Donath, Wilson, and others. The detection of cholin may serve to distinguish organic from functional disease, but the test is too elaborate for general application.

The cytological examination yields valuable data. The same technique is to be constantly employed for comparative purposes. That originally recommended by Widal, Sicard, and Ravault is as follows: Three cubic centimeters of the fluid are withdrawn; this is centrifuged for ten minutes in an instrument that revolves 3000 times per minute; the tube is inverted for two minutes, the bottom of the tube is then scraped with a fine capillary pipette (a vaccine tube does excellently for this purpose), the fluid is blown on to a slide, the deposit is allowed to dry in the air, it is fixed in equal parts of alcohol and ether for twenty minutes, stained with methylene blue, eosin, Jenner's or Ehrlich's stain, and mounted in Canada balsam.

The observations of importance in the examination of the film are (1) the number of leukocytes, (2) the types of cells present and their relative proportions, and (3) the presence of other elements. Both in the epidemic form and in the group of cases included in the present article there is commonly a great excess of the cellular elements in the cerebrospinal fluid, and, further, the increase is found to be largely due to polymorphonuclear cells which are not present in the normal fluid. An exception is found in the circumstance that in long-standing cases in which recovery is taking place the mononuclear cells are found to preponderate. Very exceptionally a fluid containing no excess of cells is met with in acute meningitis.

Tuberculous meningitis differs from the other forms of cerebrospinal inflammation in that although the cells in the fluid are increased the increase is almost entirely due to an increase in the mononuclear leukocytes, and may be described as a lymphocytosis. Cases of acute tuberculous meningitis are occasionally met with in which the polymorphonuclear cells are in excess, but these are of rare occurrence. The cerebrospinal fluid in serous meningitis contains, as a rule, no increase in the cellular elements.

In general paralysis of the insane, in tabes dorsalis, and in cerebrospinal syphilis a pronounced cerebrospinal lymphocytosis is almost invariable, and is usually most marked in the earlier stages of the disease. Thus, Purves Stewart found in eleven cases of general paralysis of the insane an average count of 127.5 lymphocytes per field, while in 22 tabetics the number in each field averaged 131.4. Very occasionally a case of tabes is met with in which there is no lymphocytosis. The presence of a lymphocytosis in an individual who has had syphilis points to implication of the central nervous system, and is an indication for mercurial treatment. In cases of syphilis in which the nervous system is not affected there is no cerebrospinal lymphocytosis. A lymphocytosis has also been found to occur in cases of herpes zoster, very occasionally in cases of intracranial tumor, in chronic alcoholism, and in mumps. When present it is to be regarded as an expression of chronic meningeal irritation. Tumor cells have been detected in occasional cases of malignant tumor of the meninges, and trypanosomes have been found constantly present in the cerebrospinal fluid in sleeping sickness.

The bacteriological examination of the cerebrospinal fluid is sometimes of great use, and will doubtless become even more important as the field of serum and vaccine therapy widens. The centrifuged deposit may be examined, cultures may be made, or injection experiments on animals carried out. The detection of microörganisms enables us to exclude conditions in which bacteria are not present, and also to distinguish between the various forms of meningitis. In tuberculous meningitis the bacillus may be found in the large proportion of cases if only sufficient care is used. The pneumococcus, streptococcus, and meningococcus are the organisms most often found in meningitis. Among other organisms which have been detected in the fluid are those of typhoid fever, influenza, and diphtheria, as well as the colon bacillus, the *Bacillus proteus*, the *Bacillus lactis aërogenes*, the *Bacillus pyogenes fœtidus*, the *Bacillus pyocyaneus*, the *Bacillus anthracis*, various staphylococci, the *Bacillus tetragonus*, and a leptothrix. The tubercle bacillus was found together with the meningococcus by Lenhartz and Heubner in three cases of cerebrospinal meningitis. A number of other instances of mixed infection have been reported. Syphilitic antibodies have been found by Morgenroth and Stertz in cerebrospinal syphilis.

# CHAPTER VI.

## DIFFUSE AND FOCAL DISEASES OF THE SPINAL CORD.

By E. FARQUHAR BUZZARD, M.D.

### 1. THE LOCALIZATION OF NON-SYSTEMIC DISEASE AFFECTING THE SPINAL CORD AND CAUDA EQUINA.

**Segmental Localization.**—In discussing the various morbid conditions which affect the spinal cord, it will be necessary to state on more than one occasion that the symptoms produced by a particular disease will depend upon the segmental level of the lesion. In order to avoid repetition, it is expedient to review the data upon which the diagnosis of the segmental level of disease is based. A *focal lesion* may be regarded as one which is limited longitudinally to one, two, or three segments of the cord, but which may involve either a part or the whole of the transverse area of those segments. A *diffuse lesion* may be defined as one which affects in a confluent or discrete fashion a considerable length of the cord, amounting to many segments. In neither case have we to deal in this section with morbid processes confined to one or more neuronic systems. From a clinical standpoint, when a diffuse lesion involves a considerable length of the spinal cord it is often difficult and sometimes impossible to do more than define the upper limit of the affected area. For this reason a focal lesion limited to a single segment is the most convenient proposition upon which to base a discussion of segmental localization.

Each segment of the spinal cord has two chief functions; in the first place, it forms a link in the chain of nervous paths which traverse it, the conduction of impulses along these paths being dependent upon the normal condition of the fibers within the limits of the segment; in the second place it is a centre for various activities of motor, sensory, vasomotor, trophic, or reflex character, all of which rely upon the integrity of the segment, and especially of the gray matter, together with the anterior and posterior roots, for their healthy expression.

**The Segment as a Link in the Chain of Nerve Tracts.**—The result of a lesion affecting the conducting paths depends upon the extent of interference with the function of these paths.

**Total Flaccid Paralysis.**—If the injury to a spinal segment is so severe that there is complete severance of all impulses, a condition of total flaccid paralysis supervenes in all parts of the body innervated from the spinal cord below the level of the lesion. The motor palsy is associated with absolute loss of appreciation of all sensory impressions arising from the skin and other tissues supplied by the same region of the cord, and with abolition of all tendon reflexes in the same parts, at any rate for a considerable period of time. The loss of tendon reflexes is combined with absence of superficial reflexes, with the exception that an extensor response may in some cases be

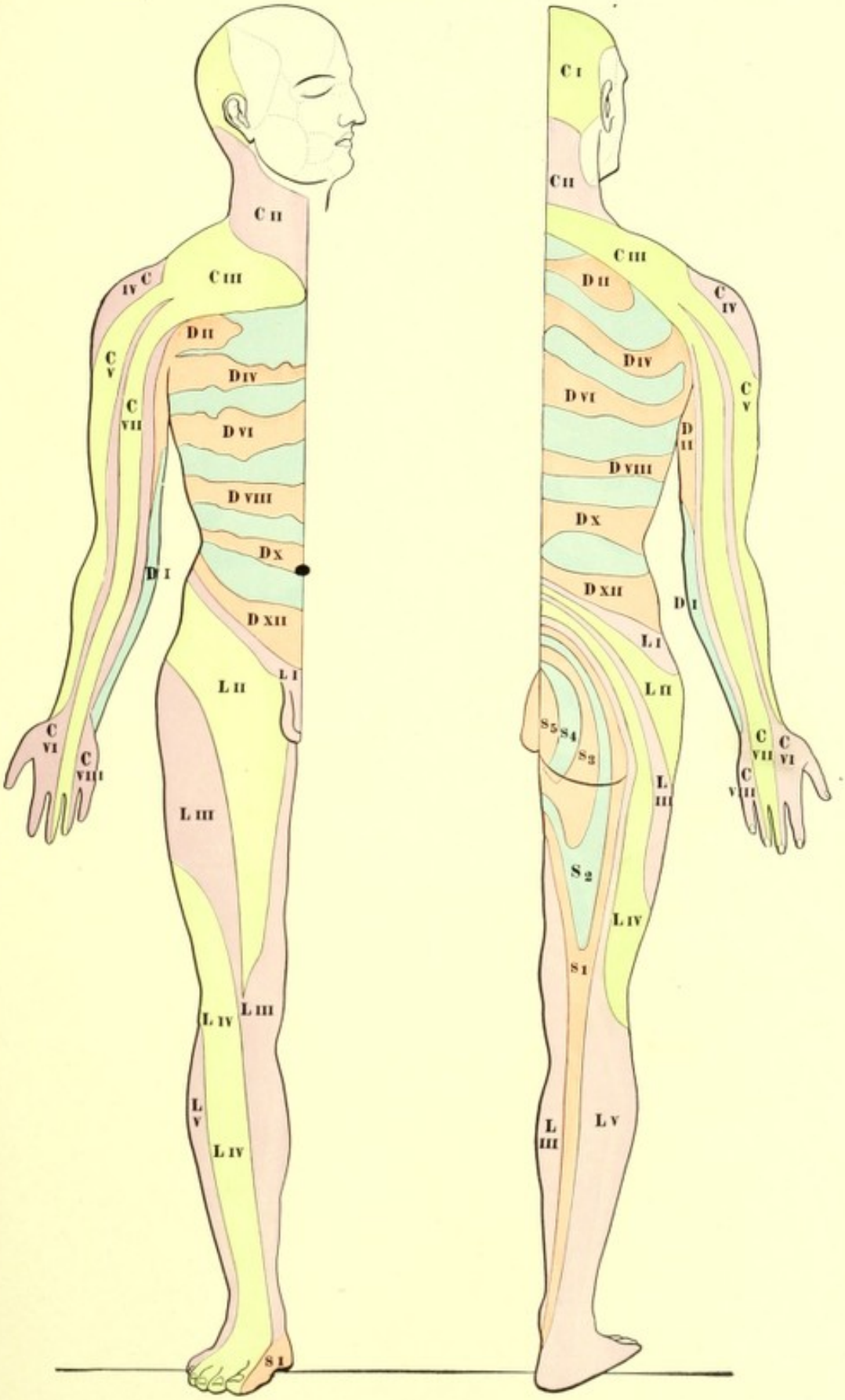
obtained on stimulating the soles of the feet. It is not quite certain whether such survival of the plantar reflex denotes or does not denote an incomplete physiological discontinuity of all impulses at the site of the lesion. Visceral reflexes are also interfered with, and complete incontinence of urine and fæces rapidly supervenes, although this may be preceded, especially in young subjects, by a short period during which reflex evacuation takes place. There is cessation of all sexual power or desire, with a tendency to incomplete priapism in certain male patients. The skin of the paralyzed parts is liable to trophic changes, and if the lesion is acute there is often a period of vasodilatation with œdema, which is, however, soon replaced by vasoconstriction and a dry exfoliative cutis. In the early stages the muscles supplied from the cord below the lesion, although completely paralyzed and flaccid, preserve their nutrition and their normal reaction to electrical and mechanical stimuli. When the complete transverse lesion is of long duration an atrophic process gradually attacks the paralyzed muscles, which ultimately lose their faradic excitability.

Under these conditions it may be difficult to decide whether the lesion is limited to a particular level above the centres for the paralyzed muscles, or whether it has extended downward so as to involve these centres. Collier states that in the former condition prolonged faradization of the paralyzed muscles may lead to a temporary return of their tendon reflexes, a phenomenon which is not obtained when the spinal centres are directly involved.

**Spastic Paralysis.**—If the interference is less profound, and yet sufficient materially to affect the conductivity of the chief motor and sensory tracts, the flaccid paralysis is replaced by a spastic paralysis of all muscles supplied by the cord below the level of the lesion. This spastic paralysis, which is sometimes known as "upper motor neuron paralysis," and which we attribute to changes in the pyramidal tracts, is characterized by the presence of some degree of tonic muscular spasm or rigidity, often associated with a tendency to involuntary reflex spasms of a painful type. The muscles retain their nutrition, size, and normal electrical excitability, but their increased tone (hypertonicity) offers some resistance to passive movements. The amount of voluntary power varies, of course, with the severity of the lesion, but the relationship of degree of spasticity to degree of paresis is one the laws of which are not yet perfectly understood. The tendon reflexes of all the spastic muscles are increased and there is a tendency to the production of clonus. On the other hand, the superficial skin reflexes are diminished or abolished below the level of the lesion, with the exception that a plantar response of extensor type is usually present on stimulating the sole of the foot, and a cremasteric reflex is occasionally obtainable. With spastic paralysis the sensory loss may vary from complete insensibility to one in which only very fine tests are capable of eliciting some evidence of anæsthesia in the skin supplied by segments below the level of the lesion. The intensity of anæsthesia is often not equal all over this area, but further reference will be made to this later on. The condition of the sphincters is also variable, the slight cases generally showing, after an initial retention, some hesitancy or precipitancy of micturition, with constipation of the bowels; the severe cases, retention or incontinence of urine, associated with constipation and rectal incontinence after aperients. At the same time the sexual powers of the male are diminished or in abeyance.

When spastic paralysis of the lower limbs has been of long duration it

# PLATE XII




Areas of Anæsthesia upon the Body after Lesions in the Various Segments of the Spinal Cord.

The segments of the cord are numbered: C I to VIII, D I to XII, L I to V, S 1 to 5, and these numbers are placed on the region of the skin supplied by the sensory nerves of the corresponding segment.

tends to the production of certain deformities, of which a pes cavus is the most common. Permanent contracture of muscles in the position of flexor spasm is another troublesome complication.

**The Segment as a Centre of Activity.**—The destruction of the gray matter by a focal lesion gives rise to certain phenomena distinct from those which result from the destruction of the conducting paths in the white matter. Each segment contains anterior horn cells, from which originate the anterior root fibers. As a result of the degeneration of these ganglion cells the efferent nerve roots degenerate, and the muscles innervated by them are paralyzed and rapidly waste. Fibrillary tremors or twitchings and a tendency to the formation of contractures are additional characteristics of what is known as "lower motor neuron paralysis." If the muscles are examined electrically during this process of atrophy they are found to undergo the reaction of degeneration, *i. e.*, diminution and abolition of response to faradism, together with an altered slow response to galvanism. In this respect they differ from the paralyzed but spastic muscles supplied by nerves arising from segments below the focal lesion, which retain their size and nutrition and present no alteration in electrical excitability. The tendon reflexes of the muscles affected by the atrophic palsy disappear, as do all the reflexes subserved by the segment which is the seat of the disease. In the skin of the area innervated by the same segment, vasomotor and trophic changes are also to be noted, a moist, sodden surface, with atrophy of nails or other structures, being the usual result. Sensibility in the skin area, from which the afferent fibers of the corresponding posterior root arise, is altered, but the anæsthesia is less marked than the analgesia and is not so complete in the upper part of the root area as it is in the lower and in the areas subserved by the segments of the cord below the lesion. A zone of hyperæsthesia may under certain conditions be present just above the anæsthetic level. Should the segment involved by the focal lesion innervate the anal or vesical sphincter, the latter becomes toneless.

Speaking generally, the results of a severe transverse or focal lesion of the spinal cord are atrophic paralysis of the muscles supplied by the segment in which the lesion is situated, together with a spastic paralysis of the muscles innervated by segments below the level of the disease. Alteration of sensibility is present as high as the area supplied by the affected segment. In order to apply the above data to the localization of a spinal lesion, it is obviously necessary to know which muscles are supplied by each segment of the cord, the skin area from which sensory impulses reach each segment of the cord, and the situation of certain centres subserving visceral and other reflexes. The large majority of muscles receive fibers from two or more spinal segments, and a knowledge of their peripheral nerve supply is therefore but a poor indication of their spinal supply. There is the same discrepancy between the nervous and spinal supply in the segmental sensory areas, which are entirely different from the areas of distribution of peripheral nerves. In addition there are factors concerned in segmental localization which deserve some discussion.

**Spinal Root Symptoms.**—The recognition that certain symptoms may be dependent on a lesion of a spinal root rather than of a spinal segment may be of immense clinical service, especially when the root concerned is a large one. It must be remembered that the result of an injury to the root may be much the same whether its site is close to the spinal origin of the root or at

the point where it leaves the neural canal, a difference, it may be, of several inches. Root symptoms are sensory, motor, reflex, trophic, and vasomotor in character, but it is only by means of the sensory and trophic phenomena that any marked distinction can be drawn between a lesion of a spinal segment and one which involves its anterior and posterior roots.

In the first place, severe pain of a sharp, stabbing kind, referred to a segmental skin area, is suggestive of a root lesion, and this may be associated with some degree of hyperæsthesia in the same region. In the second place, a destructive lesion of a single posterior spinal root may give rise to slight disturbances of sensibility of a certain character. For instance, the section of a first dorsal posterior root will produce some loss of painful and gross thermal sensibility over a region on the ulnar side of the forearm, which is smaller than the whole area supplied by the first dorsal root. The slighter degree of tactile anæsthesia in the same area is due to the greater overlapping in contiguous roots of fibers subserving touch compared to those which are concerned with painful and gross thermal sensibility. From a localizing standpoint the presence of herpes zoster within a segmental skin area denotes disease involving the corresponding spinal root ganglion, and affords a valuable guide to the site of the lesion in certain cases in which the spinal column or meninges are affected. Sometimes a subcutaneous hemorrhage or other skin affection of similar distribution may appear as an indication of a root lesion. Motor and reflex activities are affected in a similar manner whether the anterior gray matter of the segment or its efferent roots are the seat of disease.

**Oculopupillary Symptoms.**—A number of fibers pass down from a centre in the brain-stem through the cervical cord in order to join the cervical sympathetic system, leaving the cord by the eighth cervical, the first and second dorsal nerve roots. In some lesions of the cervical cord, but more often in focal lesions of the first dorsal segment, injury to these fibers causes diminution in size of the palpebral fissure and of the pupil of the same side. In such instances of sympathetic miosis and ptosis the pupil may not actively dilate to shade or to the influence of cocaine. Irritation of the same system of fibers produces a reverse condition of mydriasis and exophthalmos.

**Optic Neuritis.**—In lesions of the cervical and upper dorsal cord the occurrence of a mild and occasionally of a severe degree of papillitis has been often noted, but the mechanism of its origin has not been adequately explained.

**Vasomotor and Secretory Phenomena.**—An acute transverse lesion often produces temporary cutaneous vasodilatation and hyperidrosis in the paralyzed parts, and this may sometimes be associated with œdema of the skin, the deeper tissues, and occasionally of the synovial cavities. The fact that this is rapidly followed by complete secretory paralysis has been utilized by Horsley for the purpose of demarcating the upper limit of the affected skin by means of a dose of pilocarpine. A successful result is sometimes obtained in cases of not very recent origin. Paralysis of the cervical sympathetic, with consequent hyperæmia and hyperidrosis of the face and neck, is met with in lesions of the cervical and upper dorsal segments.

**Cardiac and Respiratory Symptoms.**—Sympathetic fibers for the cardiac and pulmonary plexuses leave the upper four segments of the dorsal cord; lesions above or involving that region give rise to modification of cardiac and probably also of respiratory action, although the latter is difficult to estimate on account of the associated interference with the innervation of the respira-

tory musculature. It is important to remember that the diaphragm is innervated from the fourth cervical segment, and that lesions of that or higher segments affect the thoracic and abdominal respiratory muscles.

**Splanchnic Symptoms.**—The superior splanchnic nerve arises from fibers leaving the dorsal cord from the fifth to ninth segments. Injuries to these fibers in their intramedullary course is attended as a rule with marked but often only temporary tympanitic distention of the hollow abdominal viscera.

**Vesical, Rectal, and Sexual Reflexes.**—The generally accepted view that there are definite centres in the lumbosacral enlargement of the cord connected with the vesical, rectal, and sexual reflexes has been strongly controverted recently on experimental and other grounds by Müller, who maintains that the central mechanism subserving these functions is situated in the sympathetic system. It would be out of place to discuss here the relative merits of these views, since the question is by no means settled; at the same time, the confession must be made that some of the clinical phenomena associated with the sphincters in spinal and cerebral disease have never received satisfactory explanation. Müller, experimenting on dogs, found that after extirpation of the lumbosacral cord, periodic evacuations of the bladder and rectum were soon established after a short period of retention, and that in male animals erection of the penis, associated even with ejaculation, was still possible. The voluntary muscles of the external anal sphincter and of the higher parts of the urethra become toneless and flaccid when the lumbosacral cord is destroyed, but if the lesion only isolates this part of the cord from the higher centres the striated muscles retain their tone and also their reflex action, although it may be in a modified manner. According to Müller the lumbosacral cord is only necessary for the conduction of sensory impulses from the organs to the brain and for the conduction of voluntary impulses to the striated muscles just mentioned. Periodic contractions of the non-striated muscles of these parts are only dependent on the sympathetic system, just as normal emptying of the pregnant uterus will take place in complete motor and sensory paralysis of the lower part of the body. It is highly probable that the simplicity of the mechanism is complicated in human conditions by the unnatural supine position of the patient and the paralysis of other voluntary muscles which are normally concerned with the evacuation of the bladder and rectum, as well as by the common secondary changes in the vesical walls resulting from sepsis.

**Pain as a Localizing Symptom.**—This is a subject of much importance and difficulty in actual practice. It is probably true that focal or diffuse disease of the cord never gives rise to pain directly, but frequently does so indirectly by involvement of the osseous or meningeal coverings or posterior nerve roots. For this reason it is obvious that pain is more often associated with morbid processes which attack the cord from without than with those which originate in its own substance. It is necessary, however, to distinguish carefully between several forms of pain. A dull, aching localized pain in the back is often present in cases in which the spinal cord and theca are undergoing steady, slow compression, or, more rarely, when an intramedullary growth is causing a focal expansion of the cord of considerable size. This form of pain may be increased by movements of the vertebral column, and it may spontaneously undergo paroxysmal exacerbations of a burning character. It is generally referred to a point only slightly below the level of the lesion.

Sharp, shooting pains referred to particular regions, it may be at some

distance from the spine, are characteristic of disease implicating the posterior spinal roots, and their accurate study may be of considerable value in determining the seat of injury. These pains may also be excited or intensified by movements, especially when the disease actually involves the vertebræ. In considering these root pains two important facts must be borne in mind; in the first place, the region to which the pain is referred will correspond to the cutaneous area supplied by that root; in the second place, the area of referred pain will be the same whether the root is affected near its medullary origin or in its course through the intervertebral foramen, this being of great significance when the root is one of considerable length. In the latter case, associated symptoms will aid in localizing the injury to the root more exactly. Care must be exercised in investigating the character of so-called girdle pain, and also of the pain which is associated with flexor spasms. Girdle pain is in some cases a root pain distributed through one of the belt-like dorsal segmental areas; in others it is a sense of constriction, which is often marked at the upper level of spasticity and anæsthesia. Pain and cramps, associated with flexor spasms of the hips and knees, are generally referred to the front of the hip and back of the knee, and must not be confounded with the pain originating from injury to the lumbosacral roots.

**Disturbances of Sensibility.**—Hitherto the disturbances of sensibility in transverse lesions of the spinal cord have been merely referred to as complete or incomplete anæsthesia to various stimuli, extending over all parts of the body innervated from the cord below the site of disease. This may be sufficient when the lesion is really transverse, and influences equally all afferent paths as they traverse the affected segment, but it is totally inadequate to describe the various forms of sensory loss which may arise in connection with less complete and more patchy focal lesions, and especially those which are associated either with disease of the central gray matter or with unilateral morbid processes. For the proper understanding of such conditions reference must be made to the work of Head and Thompson on the grouping of afferent impulses within the spinal cord.

Head, in conjunction with Rivers, had previously shown that the afferent mechanism in the peripheral nervous system consists of three systems:

1. *Deep Sensibility.*—This conveys impulses excited by pressure and by all movements of joints, tendons, and muscles. Painful impulses derived from excessive pressure are also carried by this system. The patient in whom this system is intact is not only able to recognize movements of joints, but also the locality of the stimulus and the direction of the movement. The fibers which conduct these sensory impulses run mainly with the muscular nerves, and are not destroyed by division of all the sensory nerves to the skin.

2. *Protopathic Sensibility.*—This system is capable of responding to painful cutaneous stimuli and to the more extreme degrees of heat and cold. The appreciation of these stimuli is diffuse and inexact as to the locality of the spot stimulated.

3. *Epicritic Sensibility.*—To this system is due the power of perceiving and locating light touches, of discriminating between two points applied simultaneously to the surface, and of recognizing the finer grades of temperature, called cool and warm.

Head and Thompson have shown that the impulses of these three different systems combine in new groups very soon after they enter the spinal cord.

Some impulses cross to the opposite side immediately, others cross after running a short course on the same side, and others ascend to the upper extremity of the cord entirely on the side of their entry. This re-arrangement of afferent impulses and their rough position in their spinal paths may be briefly summarized in the following manner:

1. Impulses of pain, whether excited by cutaneous stimuli or by excessive pressure, run together in the spinal cord and cross, probably early, to the opposite side.

FIG. 1

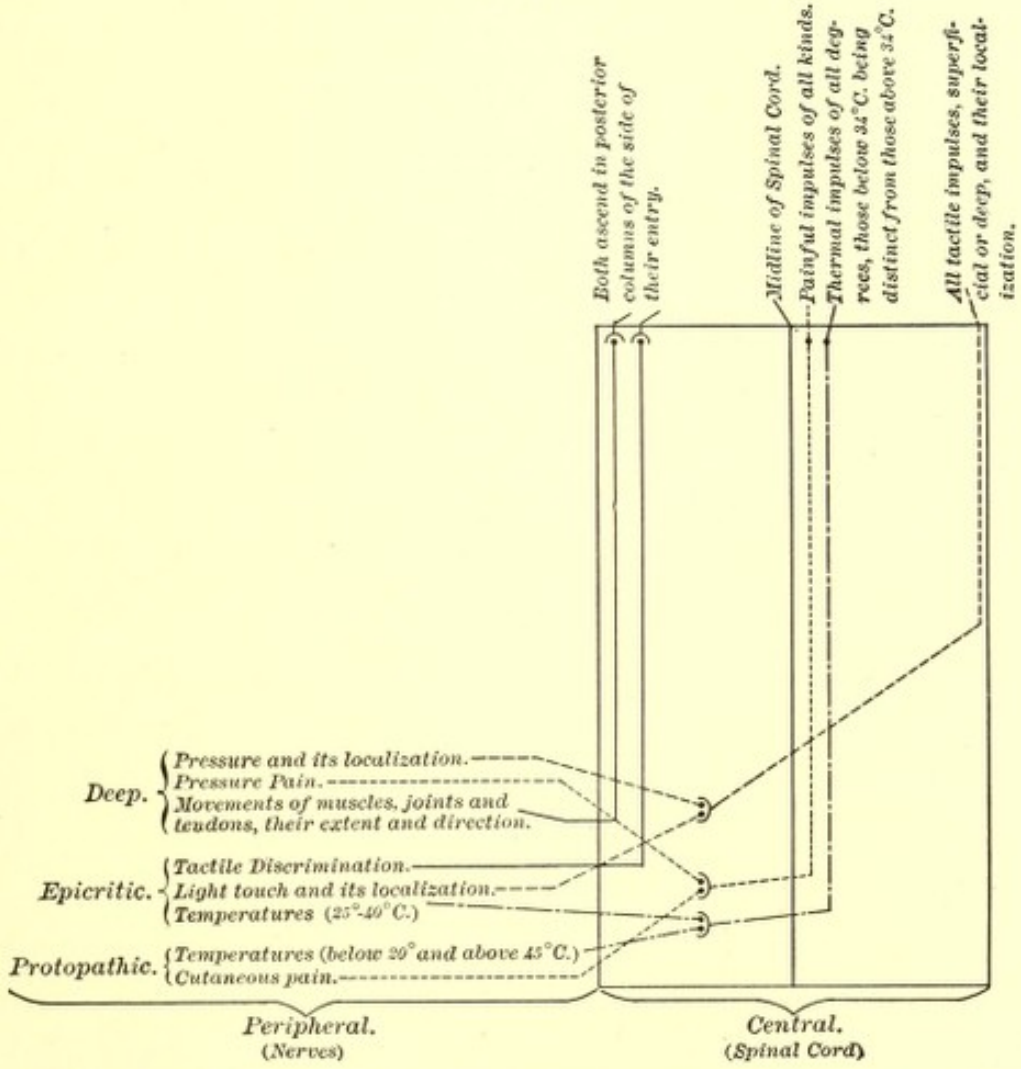



Diagram illustrating the grouping of afferent impulses within the spinal cord according to Head and Thompson.

2. Impulses of temperature *of all degrees* are combined, cross to the opposite side, and are closely associated, but not intermingled, with those of pain. Impulses of heat are also separated from those of cold.

3. Impulses excited by light touch and by pressure and those which subserve their localization accompany each other, cross to the opposite side, probably less rapidly than those of pain and temperature, and ascend in a path which is distinct from that of the latter.

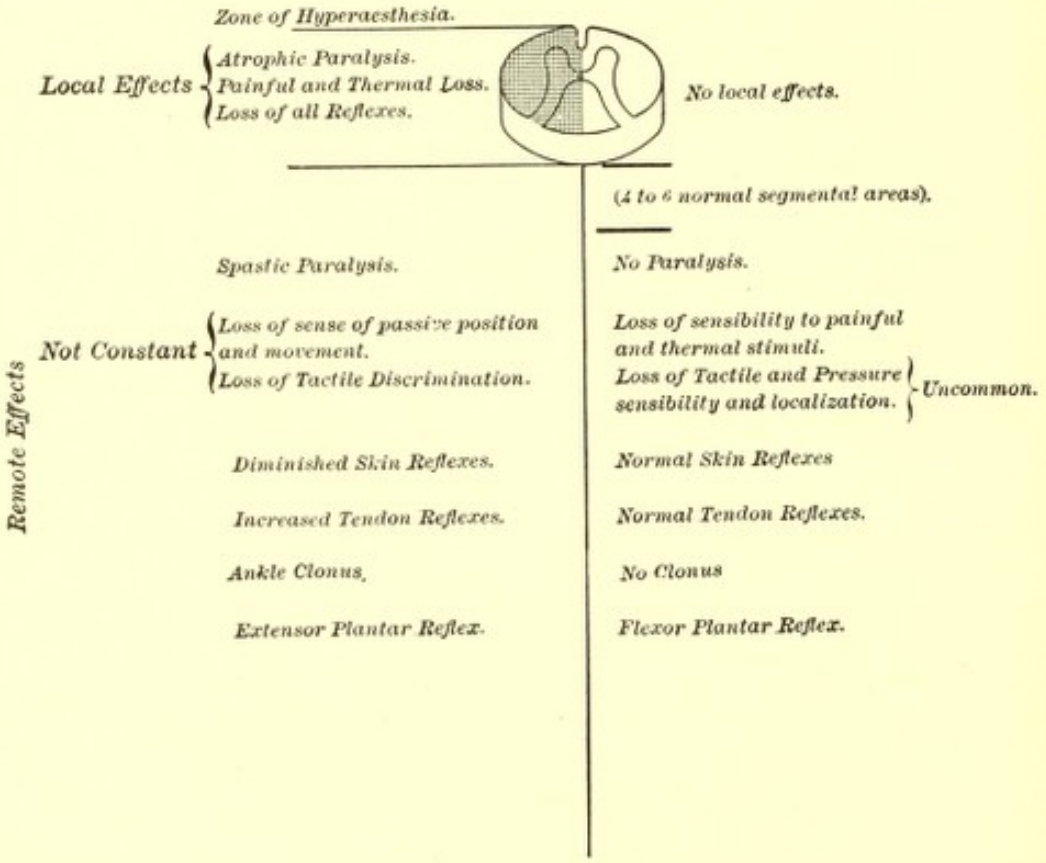
4. Impulses subserving the sense of passive position and movement are associated with those of tactile discrimination (compass points) in their

ascent of the cord on the same side as their entry. These impulses probably pass up the posterior columns.

5. Of the non-sensory afferent impulses, many ascend in the secondary system of the direct cerebellar tract to reach the cerebellum.

Head and Thompson then state their belief that the recombination of afferent impulses into new groups takes place on the side by which they enter the spinal cord, and that all those impulses which cross to the opposite side are carried by systems of fibers belonging to a secondary or intramedullary level. On the other hand, the impulses subserving the sense of passive position and movement and of tactile discrimination remain in a system belonging to the primary or peripheral level in their ascent on the same side of the cord.

FIG. 2




In the light of their observations, the symptom complex which is known by the name of Brown-Séquard's paralysis may now be considered. He observed that a hemisection of the spinal cord was followed by motor paralysis and loss of muscular sense on the side of the lesion, and by loss of other forms of sensibility at the level of the lesion on the same side, and below the level of the lesion on the opposite side of the body. Well-defined local lesions of one-half of the spinal cord are of rare occurrence, but modified forms of Brown-Séquard's paralysis are not infrequent, and those which have been examined bear out in the main the conclusions at which Brown-Séquard arrived more than fifty years ago. With improved methods of examination in relation not only to sensory, but to motor and reflex disturbances, a perfect hemisection of the cord should produce a condition which may be graphically represented in the following manner.

If an exception be made for those phenomena marked "uncommon" or "not constant" in the diagram, the remaining symptoms present a picture which is by no means rare in focal diseases of the spinal cord.

Apart from the sensory disturbances connected with unilateral lesions of the cord, there are one or two other points of interest. It is by no means rare to find that the loss of cutaneous sensibility below an incomplete transverse lesion of the cord is not equal in all the skin areas which are affected. In the first place it may happen that when pressure is slowly applied to the spinal cord at the level, for instance, of the second dorsal segment, the sensory loss in the early stages may only be traced as high as the mid-dorsal segmental areas, or may only be complete up to that level and relative in those above that level. This is readily understood when it is remembered that the long secondary afferent paths lying in the periphery of the cord are first affected by pressure, and that the decussation of sensory impulses is a gradual process not completed in less than four, five, or six segments.

In the second place, when the upper limit of sensory loss is clear and well defined, and corresponds to the level of the lesion, it may be found that some of the lower sacral areas have escaped the anæsthesia of surrounding regions. Such a phenomenon can only be explained, as Head points out, by a lamellar arrangement of the segmental impulses as they pass up the spinal cord.

**Diagnosis of Disease of the Cauda Equina.**—The cauda equina is contained within the vertebral canal from the second lumbar vertebra to the coccyx, losing a pair of roots at every intervertebral space on either side. In certain cases there arises a difficulty in distinguishing between lesions of the lumbosacral segments and those of the cauda equina. Certain principles must be remembered in this connection. In both segmental and root lesions the symptoms may be very similar, the distribution of motor and sensory loss being of the segmental type. A lesion of a lumbar segment, however, produces symptoms relating not only to that segment, but to each segment below it, while a lesion of a lumbar root may be distinct from any involvement of the roots or segments above or below.

It is clear that all the sensory and motor symptoms are of the spinal root type in lesions of the cauda equina, and that the presence of any spasticity, any increase in the deep reflexes, clonus, or an extensor plantar response, negatives the diagnosis of disease limited to that region. All muscles which are paralyzed are also wasted, have undergone the reaction of degeneration, and have lost their tendon reflexes. When the sphincters are involved, as they frequently are, they are relaxed and incontinent. Pain is nearly always present, is of a stabbing character, and is referred to a root area where, as a rule, it is ultimately replaced by anæsthesia. It must be remembered, however, that there is sometimes a stage in which pain and anæsthesia are co-existent in the same area. Modifications in the sensibility of the affected parts follow the rules already indicated in lesions of the peripheral nervous system, and do not conform to those of the spinal cord as laid down by Head and Thompson. Additional assistance in locating the lesion in the cauda equina may sometimes be derived from physical signs pointing to disease of the second lumbar or lower vertebræ.

## II. THE DIFFERENTIATION BETWEEN THE INTRA- AND EXTRA-MEDULLARY ORIGIN OF FOCAL DISEASE.

It not infrequently occurs that, the level of a particular spinal lesion having been diagnosed, it is necessary to come to a conclusion as to whether the lesion is produced by a morbid process originating within or without the cord. The decision of this question may, for instance, have an important bearing upon treatment and may, in some instances, actually indicate or contra-indicate the performance of a laminectomy.

The early incidence of *pain*, and particularly root pain, to the characters of which reference has been made, is strong evidence of the extramedullary origin of the disease. This kind of pain may be increased by movement, and then suggests some involvement of the vertebral column, or it may be associated with local *tenderness* on pressure and even with *deformity*, which point in the same direction. *Hyperæsthesia* in the cutaneous root area, followed by *anæsthesia* of a dissociative type, is also found in extramedullary lesions in their early stages as the result of injury to a posterior spinal root.

On the other hand, an *atrophic paralysis* of the muscles innervated from one or two segments preceding spastic paralysis at lower levels, raises suspicions of a primary intramedullary disease, especially if pain has been insignificant or absent during its development. In addition to these differences in local effects, an intramedullary may be distinguished from an extramedullary lesion in some instances by its remote effects. When symptoms below the level of the lesion point for a long time to the *unilaterality* of the latter, an intramedullary cause must be regarded as the more likely. Pressure from without may and does cause unilateral symptoms, but usually for a short time only, as the effect of compression can, naturally, not long be limited to one side of the spinal cord. Early *sensory* loss, especially of a dissociative type, in regions innervated from below the level of the lesion, indicates the intrinsic origin of the disease, and syringomyelia affords the most striking example of this condition.

With regard to the action of the *sphincters* no definite rules can be laid down. Intramedullary disease, even of the cervical region, may sometimes produce bladder troubles before any disturbance of motion or sensation, but this is not a common phenomenon. With extramedullary disease the vesical and rectal symptoms are usually first noticed when the paralysis of one side is being followed by that of the other, or, in cases where the lesion is bilateral from the beginning, as soon as the legs show signs of weakness. In difficult cases skiagraphy may throw some light on the question at issue.

In conclusion, it must be admitted that none of these rules are without exceptions, and that in some instances there is insufficient evidence to give a definite opinion without the aid of laminectomy.

## COMPRESSION OF THE SPINAL CORD.

The possible causes of compression paraplegia are numerous. Fracture dislocations and other injuries of the vertebral column belong to surgery rather than to medicine, and will not be dealt with here. Inasmuch, however,

as the lesion produced by such injuries is of the focal transverse type, its symptomatology at any level can be readily deduced.

The following are the chief causes of compression paraplegia: (1) Fracture dislocation and injuries of the vertebral column. (2) Spinal caries. (3) Newgrowths of (*a*) the vertebral column and surrounding tissues; (*b*) of the meninges; and (*c*) of the cord or its roots. (4) Aneurism of the aorta. (5) Parasites, particularly hydatids in the extrathecal space. (6) Arthritis deformans of the spinal column.

**Spinal Caries or Pott's Disease.—Historical.**—To Percival Pott, in 1779, belongs the credit of having first described the form of paralysis which is the result of spinal caries. Since his day the opinion as to the manner in which the spinal symptoms depend upon the vertebral disease has undergone more than one change. The first conception was based on the belief that the spinal cord was directly injured by displaced bone, of which the deformity was the outward and visible sign. The occurrence of extreme degrees of kyphosis without paralysis led to further investigation, and to the belief in a secondary spinal inflammation or compression myelitis. In recent years this view has been largely modified by increased knowledge, much of which is due to Schmaus, who showed that more than one pathological factor was involved in the production of the focal lesions. Horsley has also assisted in exposing the true pathological anatomy in a large number of instances. With the advance in our knowledge of the pathology, variations in the accepted treatment have kept pace. The day of counter-irritation has passed, but the last word upon the relative merits of rest and extension, on the one hand, and of operative interference, on the other, has not been said.

**Etiology.**—The tubercle bacillus is necessarily the most important factor, and spinal caries is often associated with tuberculosis of other organs, of which pulmonary tuberculosis is one of the most common. Tuberculosis of the vertebræ occurs most frequently in children and young adults, increasing age diminishing progressively the liability to the disease. It may exist at birth, and has been seen in the fetus *in utero*. After middle age it is the most common of primary tuberculous affections. In the young, both sexes are equally affected; after thirty years of age, men are probably rather more liable. Injury is often said to precede the onset, and it is possible that a traumatic lesion may determine the site of infection or excite a latent focus. Although tuberculosis is by far the most common process, a syphilitic caries may occasionally present identical features.

**Pathology.**—The favorite site of origin for tuberculous disease of the vertebræ is in the spongy substance of the bodies, especially in their anterior halves. Occasionally the first focus is in the cancellous tissue of the laminæ or, still more rarely, in the smaller articulations of the spinal column. From its origin in the body of the vertebra there is a tendency to invade the neighboring intervertebral cartilages and vertebræ, and to extend posteriorly through the bone and periosteum, so as to invade the extradural space. Having thus reached a region of very limited resistance and considerable vascularity, fungoid granulations spread upward and downward as well as laterally, and finally may attack, and even, in rare cases, penetrate, the dura mater. If an abscess has formed within the bone, it tends to force its way backward or forward. In the latter case the pus will find a path along the anterior surface of the column beneath the anterior common ligament, and may give

rise eventually to a psoas, a retropharyngeal, or other form of "gravity" abscess. In the former case it encounters the posterior common ligament, on one or both sides of which it projects into the lumen of the neural canal. In other instances the pus may find its way into the perispinal tissues, and present itself near the surface in the form of a lumbar abscess.

It is the rule to find some degree of bony deformity; the collapse of one or more diseased bodies produces generally a kyphosis, a kyphoscoliosis, or, occasionally, only a lateral curve. It is obvious that a narrowing of the lumen of the canal may be occasioned (1) by the ingrowth of fungoid granulation tissue, (2) by the intrusion of an abscess, or (3) by displacement of bone. As a matter of fact, more than one of these causes may be at work at the same time. In those rare cases in which an acute compression of the cord is induced, either spontaneously or as the result of some slight injury, it is usually dependent upon a sudden bony displacement still further narrowing a canal which has already been largely encroached upon by granulation tissue. More gradual compression is exercised when the growth of granulations or the formation of an abscess follows the vertebral luxation.

The injury to the cord is not, however, always a result of compression or of compression alone. According to Schmaus, the most important factor bringing about a loss of conductivity is an œdema of the medullary tissue. This is a consequence of the strangulation of bloodvessels, especially veins, in the surrounding granulation tissue. In the first place, the chief effect is upon the myelin sheaths, but later, if the condition persists, the axis cylinders and nerve cells degenerate, and ultimately become destroyed and replaced by proliferated neuroglia. This is the process which earned at one time the name of compression myelitis, but which is more appropriately referred to passive congestion and œdema. Less commonly interference with the arterial supply to a segment or segments of the cord invokes a true evascularization and ischæmia, and still more rarely the tuberculous process penetrates the dura and sets up a tuberculous meningomyelitis. The latter may remain localized, but occasionally a general tuberculous meningitis follows. Very uncommonly a tuberculous intramedullary neoplasm may be discovered post mortem in the neighborhood of the caries.

Dry caries, or *caries sicca*, is the name used to denote that form of the disease in which a rarefying osteitis, rather than abscess or granulations, is the most conspicuous feature. It is usually found in old people, and may lead to bony deformity. In these cases the condition usually proves to be an infective myelitis or meningomyelitis rather than true compression.

It is obvious that the macroscopic changes in spinal caries are very variable, and that the cord, in particular, may be found in widely differing states. In one case it will be severely compressed, reduced to a thin ribbon consisting mainly of thickened membranes and sclerosed tissue. In another the caliber may be less diminished, but the consistence, that of cream, indicates the necrotic results of circulatory interference. In another there may be no evidence of compression, but an area, embracing a segment or two, in which swelling and œdema are the most conspicuous features. Lastly, there are the instances of acute compression, displaying the bruising, softening, and hemorrhage usually associated with fracture dislocations of the vertebral column. Before alluding to the secondary changes it may be mentioned that central cavities of the cord, extending some distance beyond the site of the focal lesion, have been recorded.

The spinal roots may be, and very frequently are, directly involved in the granulation tissue. On the other hand, the anterior roots may be atrophied as a result of the changes taking place in the central gray matter. Ascending and descending tracts of degeneration can be traced above and below the focus of disease in the cord, if it is of sufficiently long standing, and resemble those found in other transverse lesions of the same organ. Not infrequently a diffuse spinal caries may lead to compression at more than one level, in which case the resulting degenerations are all the more complicated.

**Symptoms.**—Caries of the spine is most common in the dorsal, less common in the lumbar, and comparatively rare in the cervical vertebræ and sacrum. On the other hand, it is probable that paralytic cord symptoms are more often associated with cervical than with lumbar caries. In the majority of cases the first symptoms are those which are referable to the disease in the bones, although the results of pressure upon a spinal root, or upon the cord itself, do occasionally manifest themselves before the process in the vertebral column has been suspected. *Localized pain* referred to one part of the spine is usually the first symptom, and has certain characteristics. It is of a dull, boring kind, and is accentuated by movement, especially by pressure applied deeply and directly to spinous processes at its seat. It may also be increased by lateral movements exerted on the part involved, or by jarring the column as a whole. A hot sponge passed over the spine may elicit an expression of hypersensitiveness at the site of the lesion, but in the experience of the writer this popular test has no advantages over a careful manual examination.

*Localized rigidity* of the spine next deserves attention. In early cases this is due to inhibition of painful movements, and is induced by a subconscious muscular fixation of the part. In later cases mechanical conditions due to displacement of bone or involvement of ligaments may take a share in the production of immobility. The rigidity may be apparent in every movement of the patient, especially when the cervical spine is the seat of disease, or only when he attempts to stoop. In other cases it is only observed in the course of a careful examination. It is a symptom of more moment in the cervical and dorsal than in the less mobile lumbosacral region.

Next in importance is the presence of a *deformity*. An acute angular curvature denotes a less extensive involvement of vertebræ than a more rounded kyphosis. The most prominent spinous process belongs usually to the vertebra immediately below that which has collapsed. The deformity varies from a slight prominence of one spinous process to a malformation in which the greater part of the vertebral column is directly or indirectly involved. A certain amount of scoliosis is a common accompaniment of, or sequel to, the kyphotic curve, and is often only of secondary origin. On the other hand, scoliosis may be the only form of curvature, in which case it will be distinguished from those of muscular or rachitic origin by the presence of some rigidity and by the failure to reduce it on stooping. Congenital malformations are only to be excluded by the use of skiagraphy. Deformities may be completely lacking, even in those cases of spinal caries which are associated with marked symptoms of paraplegia, and severe root pains should suggest the possibility of vertebral disease in spite of the absence of all curvatures.

When a spinal root is involved certain symptoms are wont to appear. Of these, the most important are *root pains*, which must be carefully distinguished from the localized pains of bony origin. A root pain is generally sharp or stabbing in character, or may only amount to a constant sense of

pressure or tightness. Its distribution is more important than its quality from the diagnostic standpoint, and will always be found to correspond to one of the root areas. In the case of the limbs the pain is often described as shooting into the periphery; in the case of the trunk it may give rise to one form of girdle sensation. If any *anæsthesia* is present as the result of destruction of a single posterior root, it is of narrow limits, and affects the painful and thermal sensibility rather than the tactile. It is very unusual for this to be detected in the trunk areas. On the other hand, an eruption of herpes zoster may suggest that a spinal root ganglion has not escaped injury. When the anterior root fibers are involved, some *atrophic paresis* of certain muscles may be noted, but this again is slight when only one root is affected, and may be overlooked altogether on the trunk. It must be remembered that nearly all the striated muscles are innervated by more than one, usually by three, different spinal nerve roots. According to the text-books, a partial irritative affection of the anterior root fibers gives rise to a spastic muscular condition. It is a pity to use the term spastic in this connection at all, and it is at least doubtful whether a condition of sustained muscular contraction is ever produced by spinal caries involving the spinal roots. Among the root symptoms may be mentioned the oculopupillary phenomenon which follows injury to the first dorsal segment or its anterior root fibers.

In the usual order of clinical sequence, the root symptoms are followed by those which are referable to the spinal compression or to those lesions of the spinal substance which are at any rate induced by the surrounding disease. These are identical with those of any focal lesion of the cord, and are fully described elsewhere, for instance, in the discussion of transverse myelitis. The signs vary with the level of disease, and are referable in the first place to the destruction of certain spinal centres at the site of the lesion, and in the second place to the interference with the conductivity of efferent and afferent tracts. It is impossible to distinguish the signs of destruction of spinal centres from those of the destruction of corresponding roots, and these have already been described. In the case of cervical and upper or mid-dorsal caries a varying degree of spastic paralysis is found below the level of the spinal compression, and is associated with a certain amount of sensory loss and of sphincter trouble.

Since the compression is usually of gradual onset, it is well to point out that the earliest evidence is usually to be detected in the examination of the lower extremities. The patient may complain of being easily tired in walking or of a tendency to drag one or both feet, and the observer will find slight weakness of ankle dorsiflexion, brisk knee- and ankle-jerks, and extensor plantar responses. These early signs may precede any anæsthesia, and may or may not be associated with precipitate or hesitating micturition. When compression destroys all conductivity in the cord, either after a long time, or as the result of a sudden collapse in rare cases, a flaccid paraplegia will result, and then the deep and superficial reflexes also disappear. Toneless relaxation of the sphincters will also eventuate, as well as complete loss of all sensibility as high as the segmental area corresponding to the spinal lesion.

When the compression involves the lumbosacral region of the cord, a mixture of atrophic palsy of certain muscles, with spastic paresis of others, forms the clinical picture on the motor side. The sensory disturbance follows the distribution of the lumbosacral roots, and the vesical and rectal difficulties are those which are associated with paralysis of their respective

sphincters. The preservation of the patellar and Achilles tendon jerks depends on the integrity of the third and fourth lumbar segments on the one hand and of the fifth lumbar and first sacral segments on the other. The plantar reflex is only obtainable if the lesion extends no lower than the first sacral segment, but is of the extensor type.

Trophic or vasomotor disturbances are common when the spinal lesion is severe, at whatever level it is situated. Sexual impotence results from all but the slightest degrees of compression, and priapism is occasionally observed in the dorsal and cervical cases, with more or less complete paraplegia. Compression of, or above, the fourth cervical segment interferes not only with the intercostal, but also with the diaphragmatic respiration, and lesions of the upper dorsal cord are frequently responsible for distention of the hollow abdominal viscera as the result of splanchnic palsy.

Some reference should be made to the mode of onset and the course of the spinal symptoms. Except in rare cases in which a sudden luxation causes severe bruising or destruction of the cord, the onset of paralysis is slow, taking many days, more often weeks or months, to reach its height. The advent of paralysis may, on the one hand, rapidly succeed the symptoms of vertebral disease, or, on the other, may take place after the latter have been in existence for years. It may be said that no case of spinal caries, of whatever duration, is free from the risk of compression paraplegia.

*Pyrexia* of slight degree, with daily variations, is often present, but the disease sometimes runs an afebrile course, or rapidly becomes afebrile under rest in bed and other appropriate treatment.

The course must necessarily vary with the treatment, the nature of the morbid process underlying the spinal lesion, and the severity of the damage, but the condition must always be regarded as chronic, one which is slow to develop and slow to disappear, even when the issue is favorable. Speaking generally, the more subacute cases are found in young, and the more chronic in old subjects. The paralytic symptoms are nearly always bilateral and symmetrical. Some degree of asymmetry, however, is not uncommon, and rarely the picture of Brown-Séquard's paralysis is displayed.

Remissions and exacerbations are well known to occur, and may have no obvious relation to the progress of the bone disease. In a case observed by the writer the symptoms of compression were steadily progressive over many months, during which everything else pointed to a cure or latency of the caries. A trauma may induce a rapid accentuation of paralysis, or the spontaneous evacuation of an abscess may be associated with marked improvement. When a favorable turn is taken, the sensory disturbances usually disappear before the motor. Some spasticity remains in a majority of cases.

In the unfavorable cases death may result from decubitus, cystitis, pyonephritis, or from tuberculosis of other organs. An intermediate course generally means a chronic spastic paraplegia, the length of life depending largely upon the circumstances and surroundings of the patient. The occurrence of gravity abscesses is of surgical rather than medical interest, but it is well to recollect that a psoas or lumbar abscess may be the first indication of vertebral disease, and may indicate the origin of an otherwise unexplained paraplegia.

**Diagnosis.**—From the point of view of diagnosis cases of spinal caries may be divided into two classes: (1) Those in which spinal deformity is

present, and (2) those in which the spinal column displays no obvious signs of disease.

1. When the curvature is extensive and prominent the diagnosis is usually easy and straightforward, but slight kyphotic curves are not uncommon in vertebral carcinoma and aortic aneurism. Prominence of a spinous process is, moreover, occasionally met with in intravertebral tumor originating in the meninges or extrathecal space.

Vertebral carcinoma is naturally suspected when malignant disease is, or has been, present in other organs, especially in the breast. In such cases pain, although severe, is less often limited to one particular spot, and examination of the spinal column will probably reveal a loss of the normal curves and a general flatness, which may be combined with some diminution in the patient's height. The age must be taken into account when either carcinoma or aneurism is being considered, and in the latter disease assistance may be forthcoming in the shape of the signs of intrathoracic or abdominal disease. A skiagram may be of the greatest value in the diagnosis.

The presence of a scoliosis without kyphosis does not exclude caries, but other causes must be eliminated. The reduction of the curve on extending the spine in the act of stooping, and the absence of rigidity in carrying this out, point to the common forms of scoliosis seen in children and young adults. In such cases there is no paralysis, but a well-marked scoliosis may be seen in syringomyelia, and be associated with spastic paraplegia. The characteristic sensory and trophic disturbances of syringomyelia will serve, as a rule, to differentiate it. Congenital abnormalities of the vertebral column may simulate spinal caries, but a skiagram indicates the true condition.

2. When no spinal deformity is present the inquiry is much more difficult. In the first place, it must be definitely ascertained that the lesion is a focal one and that there are no signs of any disease above a particular level. Having decided that the entire motor and sensory disturbance can be referred to a single focal lesion, and having thus excluded all the diffuse or disseminate scleroses of the cord, the diagnosis will rest between spinal caries, the various forms of intra- and extramedullary tumors, and other, more rare, causes of spinal compression. The differentiation between these conditions must depend, not upon the character and extent of the paralytic phenomena, which may be seen in any one of them, but upon considerations of a wider and accessory character. Tenderness and rigidity of the spinal column at a level corresponding to the signs of compression will, in young children, forcibly suggest vertebral caries. In other persons the same symptoms may be found in connection with vertebral or intravertebral tumors as well as with caries, whether it be of the moist or dry variety. A gumma or local syphilitic meningomyelitis must also be excluded. Here, again, the use of skiagraphy may clinch the diagnosis. It is usually stated that the root pains are more severe and precede by a longer period signs of compression in cases of tumor than in cases of spinal caries, but this is not the universal rule. On the other hand paralysis more often commences unilaterally when a tumor is the cause of compression than when spinal caries is at work. The detection of tuberculosis in other organs must favor the presumption of spinal caries, but in a considerable number of cases the certain diagnosis must be arrived at by an *exploratory laminectomy*. This should not be delayed too long in doubtful cases, and it is still more important to remember that the diagnosis of a chronic myelitis to explain slow progres-

sive signs of paraplegia is never admissible except in certain rare syphilitic cases, which can generally be excluded by the results of appropriate treatment or by examining the cerebrospinal fluid.

The pain complained of by neurasthenic patients is often referred to the spine, which may be exquisitely tender. It is rare, however, for the pain and tenderness to be constantly localized at a particular level, and examination generally shows that the tenderness is more superficial than deep. Local rigidity is not observed in these cases, although the patient may hold the spinal column stiffly as a whole.

The differentiation between a true compression paraplegia and a *functional paraplegia* may sometimes require attention. In the functional cases the legs are usually stiff and extended, without tendency to flexor spasms. The weakness is general throughout all the groups of muscles instead of being more marked in the dorsiflexors of the ankles and flexors of the knees, as it is in all cases of spastic paraplegia of organic origin. The characteristic interference with the sphincters is absent in the functional cases, and, although the tendon jerks are generally exaggerated and a form of clonus of irregular rhythm may be elicited, the plantar responses are either absent or flexor in type. That these points are of importance is evident when the possibility of a functional paraplegia supervening in a spinal caries is remembered.

A great deal of space might be devoted to the differentiation of spinal compression due to caries from other forms of paraplegia, but the diagnosis is of most practical importance in those cases when a decision between a tumor and caries has to be made. If a skiagram fails to give sufficient information, and if tests for tuberculosis are negative, the question will often have to be settled by an exploratory operation.

**Prognosis.**—There are few general rules which can guide one in giving a prognosis in any particular case of spinal caries which is complicated by paraplegia. Youth favors the outlook and age adds to the gravity. Oppenheim gives statistics from Billroth's clinic which show that out of 97 patients suffering from caries, 48 died, 22 were cured, and 11 were dismissed as incurable. Speaking generally, the more complete the paralysis the more serious is the case as regards life, on account of the complications which arise in connection with decubitus, cystitis, etc. The chief difficulty arises from the impossibility in many cases of saying in what way the cord symptoms have arisen, but this difficulty has been lessened to a large extent by the use of *x*-rays. By means of a skiagram it is sometimes possible to gauge the amount of bony deformity and to detect the presence or absence of an abscess or of masses of caseating granulations. This allows of appropriate treatment, and renders the prospect of recovery more favorable than if remedial measures are undertaken in the dark. When there is a history of sudden paraplegia, and the skiagram shows evidence of great compression by bone, the outlook is always grave. On the other hand, the absence of signs of true compression engenders hope, and among such instances the dorsal and cervical cases are more favorable than the lumbosacral. It may be admitted that the cervical cases are more dangerous, on account of the interference with the respiratory musculature, than many of those in which the mid- or lower dorsal region is involved, but the writer has been favorably impressed by the power of recovery exhibited by some cervical cases, especially in young subjects. Tuberculosis in other parts naturally adds to the danger, but treatment may

have as good results upon one focus as upon another, and the presence of pulmonary tuberculosis, for instance, is not an insuperable obstacle to recovery. Gowers truly says that there is no disease of the cord in which symptoms of equal gravity so often pass away.

In about 1 per cent. of cases of spinal caries the atlo-axoid region is involved. The danger of sudden death is due to acute dislocation of the odontoid process. Extension of the disease and its inflammatory products in this region may also menace life by interfering with the function of the lower cranial nerves.

**Treatment.**—This is a question which may provoke considerable diversity of opinion, especially in regard to the advantages and disadvantages of operative interference.

*Preparalytic Prophylaxis.*—The earlier the disease of the vertebræ is recognized and treated, the less likely are cord symptoms to supervene. This is an axiom of importance, but it is not equivalent to a statement that paraplegia will not develop in cases which have had the advantage of early and good treatment. As soon as caries of the spine has been diagnosed the patient should be placed on his back, kept at complete rest for many months, and the general treatment for tuberculosis should be carried out.

*Rest and Extension.*—In certain cases of spinal caries in which paraplegia has supervened, especially in young children, the adoption of simple rest will suffice to restore power to the paralyzed limbs in a comparatively short length of time. In others recovery will ensue after many months, even when no other remedial measures are superadded. The application of extension to the vertebral column may, and often does, make all the difference in those cases in which rest alone has proved of no avail. Extension should certainly be tried in all cases where displacement of bone is obvious and recent, but it is often useless and adds to the discomfort of the patient when the bony deformity is of long standing. It is probably more serviceable in cervical than in dorsal caries, and is of little value when the lumbar spine is the seat of disease.

In all cases in which rest alone or rest and extension are being used every precaution must be taken against bedsores, especially by attention to the smoothness of sheets, by the use of water pillows and rings, and by scrupulous cleanliness.

The employment of plaster, poroplastic, and leathern jackets cannot satisfactorily replace recumbency in bed, but is necessary as a supplementary measure in order to maintain immobility for a time after recovery has taken place. In very young children the use of bandages and splints may be necessary to insure the requisite amount of rest. Massage, passive movements, and perhaps electricity may be requisitioned while the patient is in bed in order to improve and maintain the nutrition of the paralyzed limbs, so long as no undue movement is imparted to the trunk.

*Operative Treatment.*—This should be regarded as a supplementary measure, one not to be undertaken lightly and not to be regarded as replacing rest and open-air treatment. It may be resorted to before rest has been tried, when there are indications that no relief of pressure can be procured by the latter, or it may be employed when a course of rest has failed to produce any amelioration in the paraplegia. The evacuation of any superficial abscess must be carried out at once, and a deep abscess, if within reach, should be subjected to the same treatment, particularly when it is suspected of causing

compression symptoms. On the other hand, a deep-seated abscess may sometimes dry up without surgical interference under favorable conditions. Laminectomy should not be postponed until secondary changes in the spinal cord render the relief of pressure ineffectual in restoring power to the paralyzed parts, and, speaking generally, it should be undertaken earlier in the case of adults than in that of children.

It is probable that surgical interference would enjoy a greater popularity if the cases were chosen with greater care and submitted to the surgeons at an earlier period. The indications and contra-indications for laminectomy as usually cited are not to be relied upon. For instance, it is often stated that the involvement of several vertebræ excludes the use of the knife. The writer has had an experience which directly contravenes such a view. A child, aged three years, with cervical caries, after five months of rest, during which period symptoms of paraplegia continued to progress, was operated on by D. Armour. A large abscess was evacuated, and what appeared to be the necrosed remains of several vertebral bodies was extracted. The abscess cavity was washed out and the wound closed. Within four months the child, with a shortened neck, was running about without a sign of paralysis and without any need of artificial support for her head.

In spite of the success which attends operation in some suitable cases, it must be remembered that spinal caries is not a condition with regard to which it may be said than an operation can do no harm. There are instances in which this course has appeared to excite the disease into renewed activity, and others in which a fatal issue from tuberculous meningitis has rapidly followed.

It may justifiably be hoped that the use of skiagraphy will encourage and promote that careful selection of cases which is necessary to bring this treatment into the position it deserves. Equally important is it that emphasis should be laid on the after treatment, so that each may enjoy for a prolonged period the rest, fresh air, and good nourishment which are essential for satisfactory results. Finally, no hesitation should be felt in advising laminectomy when the patient's life is seriously threatened by increasing respiratory embarrassment, as is not infrequently the case in caries of the higher spinal regions.

## TUMORS OF THE SPINAL CORD.

For the sake of convenience, although at the expense of terminological accuracy, the title "Tumors of the Spinal Cord" is here, as often elsewhere, intended to include all tumors which habitually modify the functions of the spinal cord by pressure upon or invasion of that organ, as well as those which originate in its substance. In fact, the latter as a class are less common and, from a therapeutic standpoint, less important than the former. This last statement has, however, only become wholly truthful during the last twenty years, the classical and successful removal of an extramedullary tumor by Horsley in a case diagnosed by Gowers in 1887 having opened a new field in which medicine, surgery, physiology, and anatomy have all been interested. Previous to this period the study of spinal tumors was of pathological rather than clinical interest.

The classification of spinal tumors followed by Bruns may be adopted

here, but it is necessary to explain that the word tumor embraces, in this connection, granulomata and parasitic cysts as well as neoplasms proper. On the other hand, spinal gliomatosis is reserved for the article on syringomyelia.

I. Tumors which, originating in its envelopes, secondarily affect the spinal cord.

(*a*) Vertebral tumors arising from the spinal column or the soft tissues immediately surrounding it.

(*b*) Intravertebral tumors, which may be divided into two classes in accordance with their relation to the dura mater.

1. Extradural tumors originating in the periosteum of the vertebræ, the outer layers of the dura mater, or the fatty areolar tissue of the epidural space.
2. Intradural tumors originating from the inner layers of the dura mater, the arachnoid, the ligamentum denticulatum, the spinal roots, or the pia mater.

II. Intramedullary tumors of intrinsic spinal origin.

**Vertebral Tumors.**—The bones of the spinal column, especially their bodies and less commonly their arches and processes, are frequently the site of malignant growths, which may be of a carcinomatous, sarcomatous, or myelomatous nature.

*Carcinoma* in this region is always secondary and generally metastatic, although occasionally the vertebræ are directly involved by extension of disease from other organs. The primary focus may be in the uterus, stomach, intestine, prostate, thyroid, lung, kidney, or gall-bladder, but most commonly, by far, is in the mammary gland. This special connection with mammary carcinoma and perhaps uterine cancer is sufficient to account for the greater incidence of the disease in females, although males are by no means exempt. Probably 70 or 80 per cent. of cases occur in women. The age which predisposes to vertebral carcinoma is that of carcinoma of other organs. Considering the rarity of bronchial carcinoma, secondary vertebral disease from this source is relatively frequent.

The lumbar and dorsal parts of the column are more often involved than the cervical, and the disease may be limited to one or more foci or spread diffusely over many vertebræ. It is not uncommon to find the spine much more extensively affected after death than was suspected during life, and occasionally the replacement of bony by cancerous tissue is so complete that the spinal cord can be removed from the vertebral canal without the use of any instrument except a knife. The neoplasm is found to attack first the spongy portions of the bone, to destroy the medulla, and to fill the medullary spaces. The more compact bony cortex may survive as a thin shell for a time, but is ultimately also replaced by the expanding growth. Along with osseous resorption, a certain amount of calcification in portions of the neoplasm is not infrequently observed. When this osteoplastic process is excessive a compact and firm ankylosis of a large part of the vertebral column may ensue, and there may be a complete absence of any osseous deformity. On the other hand, it is more common for the softening produced by the infiltration of a spongy growth to allow the body weight to effect a general shortening of the column, a condition which is sometimes described as "entassement." When the softening is local it is often described as carcinomatous caries, and, like the tuberculous variety, may lead to

collapse and the formation of a curve like that of Pott's disease. In this manner the spinal cord may be compressed, but is only affected in comparatively few cases, the spinal roots being much more prone to suffer close to or within the intervertebral foramina. Nodules of carcinoma projecting from the inner surface of the vertebral bodies or arches may exert pressure upon the cord, and very occasionally the tissue of the epidural space becomes invaded by growth, with the result that the cord is surrounded by a kind of neoplastic cylinder. Very rarely does the disease penetrate the theca.

*Sarcoma* of the vertebral column or of the soft tissues immediately surrounding it may be primary or secondary, may form a single well-defined tumor, or be diffused as multiple growths. The lumbar and sacral regions appear to be the favorite seat of the disease. The results of the infiltration of the vertebral column resemble those of the carcinomata, and may comprise collapse and luxation, the formation of curvatures, and the compression of nerve roots or of the spinal cord. The spinal cord compression is usually brought about by the presence of growths in the epidural space, but occasionally by the penetration of the dura along the course of a spinal root, and the formation of masses upon the surface of the pia.

The secondary sarcomata of the spinal column either extend directly from the neighboring tissues, the organs of the thorax or abdomen, or are metastatic deposits from more distant parts. The writer has seen a renal sarcoma invade the vertebral column along many of the spinal nerves, and evoke symptoms both of root involvement and of compression paraplegia. Metastatic sarcoma is a rare growth in the spinal bones; according to Schlesinger secondary invasion of the vertebral column is most common when the primary sarcoma is of osseous origin. The same author expresses the view that relatively sarcoma produces metastases in the spine more often than carcinoma, but the latter disease is absolutely the more common in that situation.

*Myeloma* occupies an intermediate position between the malignant and benign tumors of the vertebræ, both as regards its course and histological characters. It is less frequent than carcinoma and sarcoma, and perhaps less common than benign osseous and cartilaginous growths. Multiple myelomatosis is usually characterized by a wide infiltration of the bony tissue of many vertebræ, rendering large extents of the column soft and pliable, with the result that deformities are easily produced by the influence of weight or position. Less often one or more masses of growth are found in the form of definite tumors. Other bones, especially the ribs, sternum, clavicle, etc., are frequently involved at the same time. The spinal cord may suffer from compression, in consequence of deformities leading to narrowing of the neural canal or from a nodule of growth projecting from the internal surface of a vertebra. Myelomata are not invariably soft throughout; occasionally they indulge in ossification and productions of ivory-like character. Small nodes of myelomatous growth are at times observed also on the outer surface of the dura mater. The disease is confined to the osseous system, and affects males more often than females.

The *benign tumors* of the vertebral column are very rare, and comprise osteomata and exostoses, chondromata and osteochondromata, myxomata, the so-called luxuriant callus formations, and the bony excrescences associated with arthritis deformans. The bony tumors only affect the nervous system when they grow in such a way as to encroach upon the neural canal

or the intervertebral foramina. The cartilaginous growths may penetrate the intervertebral foramina and produce root palsies and sensory symptoms. The occasional erosion of the spine by aneurism of the aorta or by hydatid cysts may be mentioned, but it is still an open question whether the latter ever originate primarily from the bony envelopes of the spinal cord.

**Intravertebral Tumors.**—1. **Extradural.**—Tumors of the epidural space are more often secondary than primary. Sarcomata and lipomata share the reputation of being the most common varieties, but fibromata, myxomata, chondromata, are also very occasionally seen in this situation. It is only rarely that the malignant growths penetrate the dura mater. Hydatid cysts are found most commonly in this region when they give rise to spinal symptoms.

2. **Intradural.**—These originate from the inner layers of the dura mater, the arachnoid, the ligamentum denticulatum, the spinal roots, or the pia mater. Sarcomata may be diffuse or localized. The former variety, which may be termed pial sarcomatosis, is usually of the round-celled type, and characterized by its tendency to envelop more or less extensive portions of the spinal cord in a kind of cylindrical covering. It is a growth of the leptomeninges, and is usually less luxuriant on the ventral than on the dorsal and lateral aspects of the cord. Not infrequently it may cover the whole length of the cord and cauda equina and penetrate even into the cranial cavity. In some instances it never actually invades the substance of the cord, in others it infiltrates that organ as well as the spinal roots. In spite of the absence of direct invasion by the neoplasm the cord is often softened owing to the interference with its vascular supply and perhaps to some degree of pressure. More rarely secondary growths from sarcomata of the vertebræ, the pleura, or peritoneum gain access to the subdural space and extend in a longitudinal direction upon the surface of the cord. Localized circumscribed sarcomata are also found in this situation and tend to exert mechanical pressure upon the spinal marrow as well as upon spinal roots. Endotheliomata arising from the theca, cylindromata (Billroth), psammomata, fibromata, lipomata (Gowers), lymphangiomata (Schlesinger), are less common. On the other hand, fibromyxomata or fibrosarcomata are frequently found in connection with the nerve roots. These are often single, but neurofibromata are generally multiple, and may be associated with more or less neurofibromatosis of the peripheral nervous system.

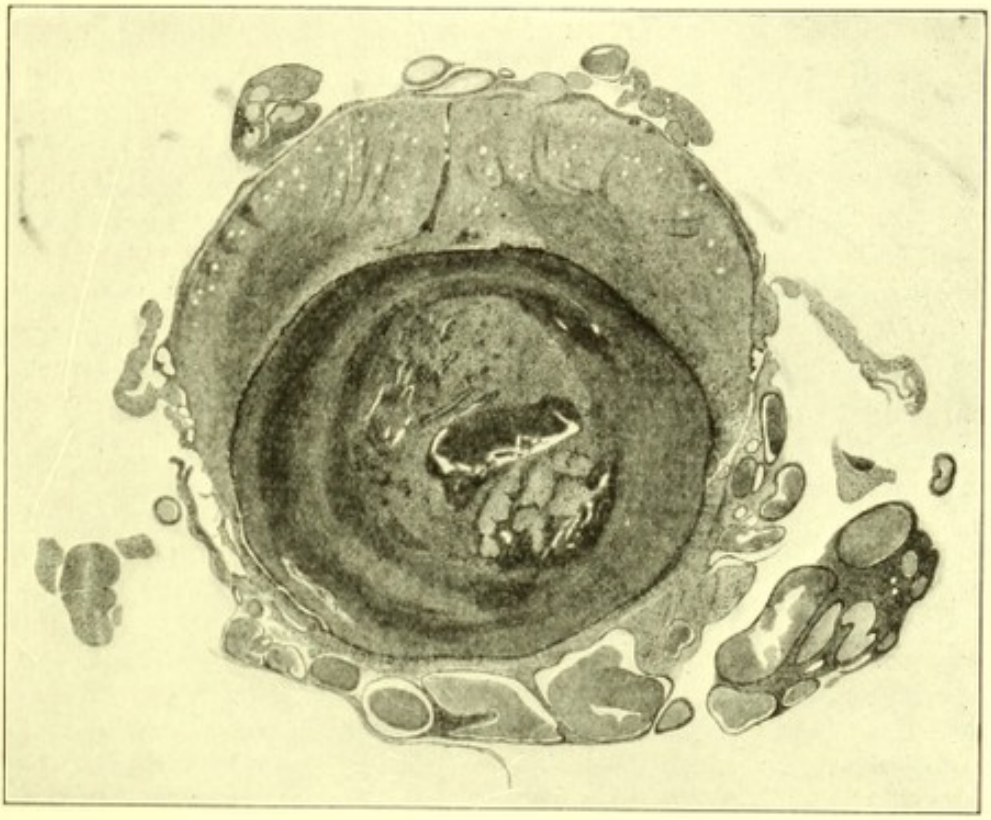
Such vascular growths as angiosarcoma and true angioma are observed now and then within the dura, and cysticerci as well as echinococci are also recorded as present in the same cavity. Intradural tumors are usually situated laterally or posterolaterally, rarely on the anterior surface of the cord. The latter is often displaced to one side and compressed or indented. In the region of the cauda equina there is room for greater development in lateral dimensions than in the higher regions, and neoplasms may here reach a considerable size with relatively few symptoms.

3. **Intramedullary Tumors.**—These comprise gliomata, sarcomata, angiosarcomata, as well as tubercles and gummata. Cysticerci are rarely found in the substance of the cord. Gliomata are usually diffuse and conform to the type which is discussed under syringomyelia, but they are occasionally circumscribed, producing the ordinary changes and symptoms of an intramedullary growth. The sarcomata, solitary tubercles, and gummata arise in the first instance from the pial septa, and are more often single than multiple.

Cases in which gummata have been found in the brain as well as in the cord, and on several spinal roots, are described in the literature.

**Pathological Processes in the Nervous Tissues Resulting from the Pressure of Spinal Tumors.**—Meningeal tumors rarely occasion more than slight erosion of the bony parts, but their effects upon the nervous structures within the vertebral column are important. The spinal roots, although more resistant than the spinal cord, are usually the first to suffer. They may be directly involved in the growth, in which it is sometimes difficult to trace their course, or they may be compressed as they pass through the dura or even the intervertebral foramina. Degenerative changes follow in either case, and may be traced in the efferent fibers in their peripheral, and in the afferent fibers in their intramedullary course. The central pro-

FIG. 3



Gumma of third lumbar segment.

longations of a single (third lumbar) posterior root have been thus determined by the writer in a case of vertebral carcinoma in which the spinal cord itself remained intact, and similar observations have been made by other writers.

The spinal cord may suffer in different ways. In most cases a certain amount of displacement is the first result, and this is followed by compression. The early effects of the latter may be only mechanical, but sooner or later they lead to interference with the circulation and the production of œdema and ischæmia. Venous stasis has usually been regarded as an important element in producing the œdema, and the arterial and capillary blood supply is modified in consequence of the latter. Superficial arteries may also be compressed, thus contributing to the local anæmia. The more

specialized nervous structures, particularly the ganglion cells, are the first to show signs of nutritional change, and the myelin sheaths of the conducting tracts readily undergo degeneration. More pronounced degrees of ischæmia lead to areas of softening, which may be associated with sclerotic changes in the neuroglia if the pressure is slowly exerted. Inflammatory phenomena do not play so important a part as was formerly supposed, but the toxic effects of the neoplasmic metabolism must be allowed some share in the process of disintegration. The presence of secondary degeneration above and below the site of compression denotes definite changes in the axis cylinders at that level. Similarly degeneration of the anterior root fibers follows the atrophy of the cells of the ventral gray matter. The cord may be softened without direct compression, owing to the interference with the arteries, veins, and lymphatics on the surface. Direct infiltration of the cord by extramedullary tumors is less frequent than might be supposed, the pia-arachnoid affording a certain amount of protection. Occasionally sarcomatous cells can be traced along the perivascular lymphatic channels which accompany the vessels of the pial septa, thus gaining access to the central parts of the medullary tissue. Intramedullary growths may be sharply defined or may present little in the way of differentiation from the nervous matter. Those which are circumscribed are often surrounded by an area of softening, and occasionally by evidences of inflammation or of toxic œdema.

**Incidence.**—Schlesinger has shown that tumors affecting the cord are by no means common, and says that in 35,000 necropsies only 147 instances were recorded. Their relation to tumors of the body as a whole is a little over 2 per cent. and to tumors affecting the brain nearly 8 per cent. The malignant nature of these growths is illustrated by Schlesinger's figures, giving the proportion as 10 malign to 1 benign.

**Age.**—Starr collected 100 cases of intravertebral tumor, and found that 70 occurred above and 30 under the age of fifteen years. The following table represents the age incidence in 82 cases collected by Armour from the records of the National Hospital (London).

| Years. | Vertebral carcinoma. | Vertebral sarcoma. | Spinal tumors. | Medullary. | Intradural. | Extradural. | Both. |
|---|---|---|---|---|---|---|---|
| Under 10 | 0 | 0 | 1 | 1 | 0 | 0 | 0 |
| 11 to 20 | 0 | 4 | 10 | 2 | 3 | 4 | 1 |
| 21 to 30 | 0 | 1 | 11 | 3 | 5 | 3 | 0 |
| 31 to 40 | 1 | 3 | 12 | 4 | 5 | 3 | 0 |
| 41 to 50 | 6 | 1 | 17 | 2 | 10 | 4 | 1 |
| 51 to 60 | 2 | 2 | 5 | 1 | 4 | 0 | 0 |
| 61 to 70 | 3 | 1 | 1 | 0 | 1 | 0 | 0 |
| 71 to 80 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total | 13 | 12 | 57 | 13 | 28 | 14 | 2 |

**Nature and Site.**—Of 400 cases collected by Schlesinger, 126 were intramedullary and 239 extramedullary. The extramedullary included 151 intradural and 88 extradural growths.

The level at which intravertebral growths are most commonly met with is illustrated by Armour's series in which the most notable feature is the preponderance of extramedullary tumors in the dorsal region and of the intramedullary tumors in the enlargements.

| | Medullary. | Intradural. | Extradural. | Both. |
|---|---|---|---|---|
| Cervical | 5 | 5 | 0 | 1 |
| Cervicodorsal | 0 | 1 | 1 | 0 |
| Dorsal | 4 | 13 | 10 | 1 |
| Dorsolumbar | 0 | 1 | 2 | 0 |
| Lumbar | 3 | 2 | 0 | 0 |
| Cauda equina | 0 | 6 | 1 | 0 |
| Sacrum | 0 | 0 | 0 | 0 |
| Diffuse | 1 | 0 | 0 | 0 |
| Total | 13 | 28 | 14 | 2 |

**Symptoms.**—These can be conveniently divided into three groups. The first comprises those associated with disease of the bone, the second those which indicate involvement of the spinal roots, and the third those referable to derangement of the functions of the spinal cord.

**Vertebral Symptoms.**—As has already been stated, the vertebral column is only, with few exceptions, seriously involved in those cases in which the disease originates in its substance or in the surrounding tissues, and this condition finds its most striking and most frequent examples in carcinoma or sarcoma. The earliest symptom is pain referred to some part of the back, dull, boring, or burning in character, and liable to exacerbations, either spontaneous or the result of pressure, movement, or concussion. Conscious of the increased suffering, which is excited by movements of the trunk or head, and particularly by those which involve rotation of the vertebral axis, the patient holds himself stiffly and avoids exposing himself, as far as he is able, to the possibility of a sudden jar. On examination the observer will detect limitation of the normal mobility of one or more parts of the spine, and will easily elicit evidences of tenderness in the same regions. Inspection and palpation of the back and even of the anterior surface of the sacrum by rectum may fail to reveal any mass, especially in carcinoma. On the other hand, obliteration of the normal spinal curves, shortening of the vertebral column, or difficulty in distinguishing the spinous processes by palpation, may afford indications. The most striking examples of loss of height are seen in vertebral carcinoma or myeloma, and may be inferred from the statement of the patient or from the obvious collapse and condensation of certain parts of the column. A narrowing of the normal space intervening between the lower ribs and the iliac crests, and the resulting prominence given to the abdomen, may afford some indication of this "*entassement.*" While obliteration of natural curves is marked in some cases, the formation of abnormal kyphoses is equally characteristic of others. A sharp-angled curvature is generally acquired, more or less rapidly, when the body of a vertebra becomes so softened as no longer to be able to bear the weight of those above. More rounded kyphosis is the result of a general and diffuse infiltration such as is seen in cases of multiple myelomata.

The degree of prominence to which bone symptoms attain in any particular case varies considerably. On the one hand, pain and tenderness of the back, deformities, and curvatures may monopolize attention throughout the course. On the other hand, pain and paræsthesia referred to the extremities and disturbance of general health may fail to attract particular notice to the spinal column, and very extensive disease of that part of the skeleton runs the risk of escaping detection.

A word is necessary upon the occurrence of vertebral symptoms in

instances of *intravertebral* tumor in which the bones escaped involvement. It is not rare to observe limitation of movement, some local tenderness, and even the prominence of a spinous process beyond its neighbors; the occurrence of these physical signs must not be allowed to weigh too heavily against the diagnosis of a meningeal growth.

**Spinal Root Symptoms.**—It is desirable to utter a word of warning against placing the symptoms and signs of the spinal tumors derived from this source in too important a position. From a positive point of view their occurrence is valuable, but it is unwise to hold that the absence of any phenomena associated with disease of the spinal roots in any way negatives the existence of a spinal tumor. Pain is by far the most common of root symptoms, and yet a painless or almost painless development of paraplegia is met with sufficiently often in intravertebral neoplasm to prove that this symptom has sometimes been held in too high esteem. It is only right to add that the combination of acute pain in a root distribution and progressive paraplegia, in the absence of any evidence of spinal caries, is characteristic of cases of spinal tumor (*paraplegia dolorosa*).

The pain associated with disease of the posterior roots, whether in their intrathecal or intravertebral course, is usually severe, neuralgic, stabbing, and referred to the peripheral distribution of the afferent fibers. It may be unilateral or bilateral, and in the latter case may give rise to a painful form of girdle sensation. Exacerbations may be caused by movement, especially when the root is involved by growth in the intervertebral foramen, which is most frequent in vertebral carcinoma or sarcoma. The skin of the areas in which pain is felt is often hyperæsthetic, and later may become anæsthetic and analgesic, although pain persists (*anæsthesia dolorosa*). Appreciable anæsthesia and analgesia are rarely evident unless two or more consecutive roots are interfered with, although some loss of thermal and painful sensibility may be detected in the case of single root lesions in connection with the upper or lower limbs. When the spinal root ganglion is involved an eruption of herpes zoster may appear in the corresponding root area.

Spasms and twitchings in the muscles supplied by their efferent fibers are usually stated to result from irritation of the anterior roots. In a theoretical sense this is possibly true, but practically it is rare to get any signs of irritative motor phenomena if one excludes the local increase in tone of certain muscles supporting the vertebral column, which may well be regarded as of reflex rather than of direct origin. The search for muscular spasm, of radicular origin, for the purpose of diagnosing the level of disease, will rarely lead to any results of importance. On the other hand, the destruction of the anterior root fibers gives rise to physical signs which are difficult to separate from those of compression of the cord itself, and which often make their appearance at a time when the latter organ has begun to suffer. Thus, abolition of function in a single anterior root does not, as a rule, give rise to complete palsy or complete atrophy of any muscle, but a few muscles, partially innervated by the root involved, present some paresis, some wasting, partial reaction of degeneration, and possibly fibrillary twitchings such as are seen in cases of progressive muscular atrophy. These signs are much more readily detected in the limbs than in the trunk, where the overlapping of muscular root innervation is much more pronounced. When two, three, or more adjacent anterior roots are affected, the paralysis and wasting is more or less complete in certain muscles and partial in others, the contractures

usually associated with lower motor neuron palsy following in the ordinary course of events. It is hardly necessary to add that the reflexes subserved by the affected spinal roots are abolished.

Cases of tumor of the cauda equina afford the most striking examples of multiple root lesions uncomplicated by symptoms of spinal cord origin. In other regions it is sometimes difficult to draw a sharp line of distinction between radicular and central evidences of disease. Root symptoms play perhaps a more prominent part in the clinical course of vertebral than of intravertebral tumors, and they are certainly more often bilateral in the former than in the latter.

**Spinal Cord Symptoms.**—Sooner or later the neoplasm gives rise to the symptoms of a focal lesion of the spinal cord. In vertebral carcinoma death may occur before any compression has taken place, but in the majority of instances of intravertebral growth the spinal symptoms engross most attention before the disease has been very long in progress. It is characteristic of spinal tumors that their effects upon the cord are progressive, more characteristic, indeed, than the occurrence of pain. The symptoms attendant upon transverse lesions and the diagnosis of their level have already been fully discussed, but a few points, bearing particularly upon the onset and course of spinal tumor, must be referred to here.

With vertebral tumors signs of compression are mostly bilateral from the beginning, with intravertebral and particularly intradural growths, although they are frequently situated to one side of the cord, the symptoms of paralysis are rarely unilateral for long. On the other hand, inequality in degree of disablement of the two sides is common enough, and it is not always the side first affected which is later the more profoundly paralyzed. True unilateral lesions of considerable duration are most commonly met with in cases of intramedullary tumors, and their symptomatology is that of Brown-Séquard's paralysis.

In extramedullary growths motion is usually affected before sensation, and the earliest symptoms of compression paraplegia in cases of tumor above the lumbosacral enlargement are easily induced fatigue in walking and a tendency to drag the toes. Examination at this time will probably reveal some paresis in the dorsiflexors of the ankle and flexors of the knees, as well as increased tendon jerks and the extensor type of plantar reflex. These signs may, however, only be found on one side at first. S long as the symptoms are unilateral there is rarely any interference with the sphincters, but the extension of the effects of compression to the other side is attended by precipitancy or hesitancy in micturition. Increasing spasticity and paresis in the muscles innervated from the segments below the site of disease mark the advance of pressure until a time comes when the lesion amounts to a complete physiological section; a flaccid paralysis then replaces the spasticity, tendon and superficial reflexes disappear, and the sphincters become quite incontinent.

Similarly, on the sensory side paræsthesia in the periphery of the lower extremities—a vague numbness or coldness of the feet—precedes the development of definite diminution of sensibility. When the latter is established it is found to involve all forms of sensibility, perhaps in unequal degree, and to spread rapidly upward toward the level of the disease. For some time the upper limit of diminished sensibility may correspond to a segmental area some distance below that of the site of compression, the decussation

of tactile, painful, and thermal impulses being gradual, and their supra-decussational paths lying more exposed to the effects of pressure than the more central areas in which the crossing takes place. It is common, however, to find that, while diminution in cutaneous sensibility only reaches a certain level, between that level and the area innervated from the segment which is being compressed, stimuli, tactile or painful, may be felt by the patient as altered in character, although they are perfectly appreciated. The more nearly the lesion approaches completeness, the more closely does the loss of sensibility approximate upward to the area subserved by the segment which is on the verge of physiological disintegration. Emphasis is laid upon this clinical fact, because it is often of supreme importance to estimate accurately the upper limit of sensory change in order to localize the site of compression. In other cases, in which the afferent root is compressed, or the posterior gray matter suffers early at the level of the tumor, the local anæsthesia in the corresponding segmental area is sufficient to guide the observer to a correct estimation of the height of the lesion.

Sensory loss in cases of *intramedullary* growth tends to exhibit special features. Its early appearance, its frequent unilateral distribution, and dissociative characters are among the most important of these. The sensory phenomena associated with Brown-Séquard's paralysis have been described, but a duplication of these may sometimes be caused by a central tumor, with the result that the local and remote areas of dissociative anæsthesia are met with on each side of the body at the same time. Although this is characteristic of syringomyelia, it is not limited to that disease, and may occasionally be found with medullary tumors other than gliomata.

**Upper Cervical Region.**—Tumors in this region tend to produce a picture of spinal hemiplegia or double hemiplegia which resembles in many respects that of pontine or cerebral origin. The absence of symptoms pointing to involvement of the cranial nerves and the local signs in the region of the neck usually separate the two conditions. In these cases pain is referred to the neck and to the region of the occiput, extending as high as the vertex, and is followed by the development of hemiplegia involving the arm, leg, and trunk on one side, less commonly by bilateral motor paralysis. In a case seen by the writer, pressure, exerted by a tumor at the level of the foramen magnum, caused hemiplegia on one side, shortly followed by a similar condition on the other, the side last affected being the more disabled at the time the patient came under observation. Although usually regarded as a dangerous site, in relation to life, it is worthy of note that the patient in this instance had suffered for over three years from symptoms referable to the presence of the growth. More profound paralysis of the respiratory musculature would, of course, have brought about an early fatal termination.

**Cervical Enlargement.**—Owing to our fairly precise knowledge of the spinal motor and sensory innervation of the upper extremities, tumors of the cervical enlargement can generally be readily recognized. An atrophic paralysis of the arm, with spastic paralysis of the parts below, associated with sensory disturbances and sphincter troubles, all steadily progressive, are characteristic. Oculopupillary symptoms and grave respiratory paresis may often be noted in the same connection.

**The Dorsal Region.**—The chief difference between tumors of the dorsal cord and those of other levels lies in the absence of well-marked atrophic palsies as local symptoms at the level of the lesion. This is partly due to

the overlapping of the muscular spinal innervation, but still more to the difficulty in distinguishing clinically how far separate sections of a large muscle supplied by many spinal segments are in a state of functional activity, or how large a share of a movement, such as inspiration, is carried out by each separate muscle, such as an intercostal, concerned in it. These difficulties are not so great if a careful examination is made, and there are certain points which are helpful. Paralysis of the lower half of the rectus abdominis is readily demonstrated by the upward movement of the umbilicus in the attempt to rise from a supine to a sitting posture. Palsy of the lower intercostals may be recognized if the hands of the observer are placed over the lower ribs during respiration, especially if the patient is not too well covered with fat. Perhaps the accurate testing of the superficial trunk reflexes from segment to segment affords more dependable evidence of the level of the lesion than any of the motor phenomena, but the accomplishment of this requires considerable experience and is often difficult in obese subjects. If, for instance, the skin reflexes are obtained on either side above the umbilicus and are absent below that level, the indication is in favor of a lesion reaching as high as the tenth dorsal segment, and this may be confirmed by the movement of the umbilicus already referred to. On the whole the disturbances of sensibility offer, in most cases of tumors of the dorsal cord, the most reliable indication of the height to which the disease has attained. Apart from the questions involved in their segmental localization, tumors of the dorsal cord present no special clinical features if exception is made for the splanchnic palsy, occasionally associated with pressure on the upper half of this region. The distention of the abdomen in these cases is often only temporary, but, while it lasts, may be productive of great discomfort and respiratory embarrassment, as well as of troublesome constipation. The sphincter troubles and the tendency to decubitus depend directly on the severity of the lesion and the amount of interference with the sensory tracts.

**The Lumbosacral Enlargement.**—The short length of this part of the spinal cord and the fact that it can be easily exposed by the removal of two or three laminæ minimizes the necessity for the accurate segmental localization which is so necessary in cases of tumor of other regions. On the other hand, it is most important to distinguish between lesions of this enlargement and those of the cauda equina, which present some features in common.

Pains and atrophic muscular paralysis of the regions innervated by the lumbar plexus, sensory disturbances over the whole of the lower extremities, absence of patellar jerks and presence of ankle clonus and extensor responses, together with retention of urine and fæces, from a symptom complex characteristic of compression of the lumbar segments and roots. When the lesion also involves the sacral segments, the atrophic paralysis and anæsthesia are more extensive, and absence of all reflexes in the legs is combined with incontinence of the sphincters. Under these circumstances the ultimate picture may closely resemble that of a tumor of the cauda equina, and the diagnosis may depend upon a consideration of the development of the symptoms. A Brown-Séquard paralysis is occasionally met with in cases of lumbar tumor, more often with intra- than extramedullary growths.

**The Cauda Equina.**—It must be admitted that tumors, apart from injuries, form the large majority of morbid conditions of the cauda equina. The first symptom of some neoplasm attacking the lumbosacral roots is nearly

always pain in one lower limb. The supervention of anæsthesia and of atrophic muscular palsy and the gradual spread of these three signs in that order, from one part to another, together with the early involvement of the opposite side in a more or less symmetrical manner, point with certainty to a gross organic basis for the disease. The loss of sphincter control when the third and fourth sacral roots begin to suffer, and the abolition of the various reflexes, with the spread of the morbid process to the radicular paths, upon the integrity of which they depend, follow in the natural course of events. The question as to whether the growth is of vertebral or intravertebral origin is sometimes difficult to answer, but the solution may possibly be arrived at by a careful examination of the lower lumbar vertebræ and sacrum, supplemented by investigation of the pelvis.

Contra-indications to the diagnosis of tumor of the cauda equina are afforded by the presence of a Brown-Séquard's paralysis, of any spasticity, exaggeration of tendon reflexes or extensor type of plantar reflex. It may be mentioned here that neither the presence nor absence of fibrillary tremors, nor the character of the electrical reactions of the muscles, can be relied upon for the purpose of distinguishing between an affection of the anterior gray matter and of the anterior roots.

**Course and Result.**—With few exceptions the course of spinal tumors is gradually progressive and their result fatal. The rapidity with which symptoms reach their greatest intensity differs considerably with the nature and seat of the growth, and the relative duration of the pre-paralytic and paralytic stages may present wide variations. Rare instances of apoplectiform paraplegia are afforded by the collapse of diseased portions of the spinal column and by the development of acute "myelitic" changes in the cord at the seat of slight pressure, but in the first cases the morbid condition of the bones has usually made itself obvious by other symptoms, and in the others premonitory signs of compression have afforded some clue to the presence of a tumor, even if local or root pains have been absent.

With regard to duration a sharp distinction may be drawn between cases of vertebral and intravertebral growths. In the former, whether of carcinomatous or sarcomatous origin, the inevitable fatal issue is generally reached within nine months; in the latter, life may be prolonged for two or two and one-half years, and in some instances for much longer periods. Isolated nodules of sarcoma, fibroma, or psammoma have been known to produce symptoms over eight, fourteen, and even twenty-five years. Remissions and exacerbations cannot be said to be marked or even usual, but, as with neoplasms in other parts, they are not by any means of great rarity.

The primary intravertebral tumors are hardly ever responsible for metastases in other parts; it is less uncommon for intracranial growths to be associated with secondary deposits in the spinal cord or on the spinal roots.

The mortality depends on the almost inevitable supervention of decubitus, cystitis, and renal disease, except in those cases in which respiratory paralysis leads to a rapid death.

**Diagnosis.**—Three important questions have to be answered in this connection. In the *first* place, the differentiation between a spinal tumor and some other spinal or nervous diseases has to be established. In the *second* place, the precise situation of the neoplasm must be ascertained. *Thirdly*, the nature of the growth is a matter of some moment when the line of treatment has to be decided upon.

1. **The Diagnosis of Spinal Tumor.**—This is not so difficult a task in the case of vertebral tumors, because, on the one hand, these are frequently secondary, and the presence or history of another growth may indicate the nature of the disease, and, on the other, spinal caries is almost the only morbid process with which they may be confounded, if an exception is made for vertebral aneurism and hydatids. No absolute rules can be laid down, but in a general way caries of the spine is associated with more marked curvatures and gibbosities, with less prolonged, less severe, and perhaps more symmetrical root pains, and often with definite skiagraphic appearances. Vertebral tumors are characterized by less sharp curvatures and more condensation of the spine, by prolonged and severe pains, often asymmetrical, and by a more rapidly progressive course and cachexia.

Tuberculosis in other organs must always be looked for, but it is too early to say how far other tests can be relied upon for the purpose of deciding a question of this kind. In their early stages vertebral tumors are often overlooked because pain may be the only symptom and physical examination may yield no further help. To neurasthenia or "rheumatism" must not be too hastily assigned persistent pains in patients who are obviously suffering, but it is not often that a definite decision can be arrived at until some more objective evidence is available.

With intravertebral growths there are certain diagnostic points of primary importance. The fact that all symptoms can be ascribed to a single lesion at a particular level in the cerebrospinal system serves to exclude such diffuse diseases as disseminate sclerosis, in which paraplegia, sensory and sphincter disturbances may have a more or less gradual and painless onset. When everything points to a single localized lesion, the fact that the onset is slow and the course of symptoms progressive practically excludes all forms of spinal disease except a tumor, a local meningeal cyst, and some rare forms of spinal syphilis. With the exception of the latter there is no form of chronic myelitis which can simulate the insidious onset or the progressive course of a compression paraplegia. The rare cases of vertebral caries without deformities or evidence of osseous disease must of course be remembered, although it is often impossible to diagnose these without exploration. Mention has just been made of local meningeal cysts with the object of emphasizing the difficulty of their differentiation from spinal tumors, this being the greater on account of our ignorance of their pathogenesis. That they are a real and not infrequent source of error is proved, although the mistake is of academical rather than practical or therapeutic importance.

*Hypertrophic cervical pachymeningitis* may suggest the early stages of an intravertebral neoplasm, but the limitation of the symptoms to those of root or meningeal origin points in the direction of the true diagnosis, which may often be confirmed by the favorable course.

As a matter of fact, there are few conditions with which a spinal tumor can be confounded if we exclude caries and meningeal cysts, and if we remember that a tumor produces a local lesion and that the onset of symptoms is slow and their course steadily progressive.

When multiple tumors of the meninges or roots exist, there is usually much difficulty in forming a true estimate of the amount and situation of the spinal damage. Multiple neurofibromata and multiple sarcomata, although located on the roots, often evoke symptoms of spinal cord compression before giving rise to anything very definite in the way of radicular palsies

or disturbances of sensation. The symptom complex may be so mixed in degree and height that a true conception of the anatomical condition is impossible.

2. The exact situation of the tumor must be gauged from the data which have been given, but attention may be directed to certain general principles. In estimating the highest level of damaged spinal marrow the opinion must be arrived at by a careful comparison of the results of examination on the motor, sensory, and reflex sides, helped by any information which can be gained from investigating the spine of the patient manually, visually, or by the *x*-rays. The most minute changes in sensibility should be noted, especially in case of tumor of the dorsal region, and when a segmental area shows well-marked anæsthesia it should be concluded that at least one spinal segment above that by which the area is innervated is, in all probability, severely damaged. Thus, if definite anæsthesia can be traced as high as the seventh dorsal segmental area, the sixth dorsal segment is compressed, and the removal of the fourth and fifth dorsal laminæ will expose the seat of disease. The highest indication of any disturbance of function is nearly always the safest guide to follow. The downward extent of the lesion is generally impossible to estimate.

3. It is hardly ever possible even to hazard a guess as to the histological characters of a primary intravertebral tumor until it has been exposed. It is therefore unwise to allow the question of its nature to influence in any way the consideration of treatment. On the other hand, a vertebral tumor, primary or secondary, or a secondary intravertebral growth, usually presents evidences or associations pointing to its nature. In the great majority of cases, unfortunately, the knowledge thus acquired contra-indicates the hope of a radical cure.

The diagnosis between extra- and intramedullary lesions of the cord has been discussed, and the principles there laid down can be applied to cases of spinal tumor. It is necessary to repeat the warning that there are not infrequent instances in which the question can only be positively answered by an appeal to exploration, and that the gravity of the outlook justifies that measure in any doubtful case.

**Prognosis.**—In the large majority of cases of vertebral tumors their malignant characters render the prognosis hopeless and the fatal termination a matter of months. With intravertebral growths the outlook depends entirely on the possibility of successful removal, the liability to recurrence and the condition of the spinal cord at the time the attempt to remove them is made. The prognosis must always be grave until these questions can be answered. The most favorable cases are those of localized benign or comparatively benign tumors in the extradural or intradural spaces, and the least favorable those of diffuse or multiple extramedullary neoplasms and all those of intramedullary origin. It is fortunate that compression of the cord may be prolonged and severe without destroying all hope that its functional activity can be restored if the source of pressure is removed. Indications as to the expectation of life in inoperable cases of spinal tumor have already been given in discussing their course and result.

**Treatment.**—From the therapeutic point of view cases of spinal tumor may be divided into operable and inoperable classes. With those which are inoperable remedial measures must be directed toward the alleviation of pain and the prevention of complications, such as sepsis, bronchitis,

pneumonia, cystitis, and pyonephritis. The alleviation of pain may be difficult, but less compunction need be felt about the administration of narcotics than in diseases with a more favorable outlook. Unfortunately the pain produced by pressure upon the afferent roots and the great discomfort evoked by flexor spasms of the lower limbs in the stage of advanced spastic paraplegia do not always yield even to opium. In this contingency the advisability of dividing the posterior roots for the relief of the more urgent sufferings may be well considered.

The possibility of a syphilitic basis for the symptoms of spinal tumor, in the shape of an intramedullary gumma or a local gummatous meningitis, must not be forgotten, and antisyphilitic remedies should always be given a trial in doubtful cases. The examination of the spinal fluid should be resorted to for the purpose of setting these doubts at rest.

A large number of cases of intravertebral tumor are suitable for surgical exploration, even if only a small proportion attain a really successful result. With our present knowledge it is usually possible to give an accurate diagnosis of the site of the lesion, but rarely possible to state the nature, size, origin or possibility of removal of the growth. Nothing short of exposure of the tumor can clear up these points, and there is less harm done by operating on cases of what prove to be ineradicable spinal neoplasms than by choosing unsuitable cases of intracranial growth for surgical intervention. An opportunity is afforded for relieving pain even when the disease cannot be arrested, and the operation in skilled hands is attended by no undue risk considering the gravity of the case. The surgeon who can stay his hand when a laminectomy exposes a hopeless condition will do no harm even if he effects no good.

This is not the place to discuss surgical details, but one or two points may be noted. It sometimes happens that on the removal of two or three laminæ no tumor is found in the expected situation. The absence of pulsation in the theca at this level should encourage the surgeon to go higher, and he will probably find the source of pressure by removing another arch or two. It is less common for the laminectomy to be performed at too high a level, but in such a case the healthy pulsation of the theca may suggest the advisability of extending the wound downward. The removal of several arches is not attended by the disastrous results in the way of lack of support which might be expected.

The after treatment of successful operations is that of convalescing myelitis. The restoration of function in paralyzed limbs may be hastened by the employment of massage and electricity, but attention to the general nutrition and health of the patient will do the rest.

## CAVITIES AND FISSURES OF THE SPINAL CORD.

Under this title are included a number of conditions of little practical importance with the exception of syringomyelia. For some reason we are accustomed to term "cavities" in the spinal cord what would, perhaps more properly, be termed "cysts" in other organs. At any rate we mean by a cavity a space, with or without an organized wall, containing fluid. Cavities, continuing the abuse of this term, are the not infrequent terminal results of an acute inflammation, in the course of which portions of the spinal tissue have become necrosed and ultimately absorbed. An example may be

found in the cord of a long-standing case of poliomyelitis, just as a cyst or porencephaly may represent an old encephalitis. Of very similar origin are the cysts or cavities resulting from an old focal hemorrhage or thrombosis. These cavities have no definite lining membranes, their walls consisting of neuroglia which may or may not be compressed so as to give it a condensed laminated appearance.

The term "fissure" is usually applied to a cavity which is narrow, and which may appear narrower after it has lost its fluid contents than it did before it was opened. Care must be taken not to regard as fissures those spaces which are seen in microscopic specimens as the result of hardening processes, and which are particularly liable to be artificially formed along the tracts of bloodvessels when the walls of the latter have been thickened by disease or when the embedding has not been skilfully carried out.

**Hydromyelia.**—This name has been variously used, and it would cause less confusion if it were confined to cases of simple dilatation of the central canal without co-existing alterations in surrounding structures, apart from the results of simple stretching, and without clinical symptoms. The condition is most frequently found in infants with congenital hydrocephalus, spina bifida, or other developmental abnormalities, but may also be present unsuspected in older persons in minor degrees. A satisfactory explanation of its production is difficult, but increase of fluid pressure due to mechanical causes, or physiological increase of fluid output by an overactive ependyma, may be of sufficient account in certain cases. The hydromyelic cavity is lined by ependymal cells, and its shape is usually round, but sometimes T-shaped, owing to a diverticulum in the direction of the posterior median septum. The addition of some peri-ependymal gliosis or of ependymal proliferation with the formation of accessory canals is the first step toward the production of what is termed "syringomyelia," and is evidence of the very imperfect line of demarcation separating the two conditions.

**Syringomyelia** (*συριγξ*, marrow; *μυελος*, tube).—**Synonyms.**—French, syringomyélie; German, Syringomyelie; Italian, siringomielia.

**Definition.**—A chronic disease of the spinal cord characterized anatomically by the existence of one or more pathological cavities, and clinically, in its typical form, by the presence of dissociative anæsthesia and trophic changes in muscles, skin, and bone, together with numerous other, but less constant, physical signs.

**History.**—The presence of a central canal in the normal spinal cord was known to Etienne in 1545, and numerous references to cavities occur before the name of syringomyelia was first used by Ollivier (D'Angers), in 1837. Curiously enough, this observer denied the existence of a normal canal and applied the name he invented to any canal or cavity in the spinal cord. Calmeil (1828) was one of the first to attempt an explanation of a central canal on developmental grounds, but its universal presence in the normal human cord was not definitely established until the middle of the nineteenth century by the work of Stilling and Waldeyer. For a time, "hydromyelia" replaced the term "syringomyelia" to denote spinal cavities, all of which were then supposed to be of developmental origin; but investigations showed that additional factors had to be reckoned with, and the name "syringomyelia" reappeared, with the object of separating the acquired from the congenital cases. As will be seen later, no such clear line of distinction can be drawn between the two with our present knowledge.

Hallopeau introduced the term "diffuse peri-ependymal myelitis" to describe what he considered to be the inflammatory origin of certain cases of syringomyelia. Charcot and Joffroy interpreted the central glial changes leading to cavity formation as a form of granular disintegration, and then Grimm, Simon, Westphal, and Leyden adopted the view that the hyperplasia of the neuroglia was neoplastic in character and that cavitation was the result of degenerative processes in the newgrowth. Up to this time no one had regarded syringomyelia as anything more than a pathological curiosity, which might, or might not, be attended by clinical symptoms, and although Charcot in 1874 referred to the condition as one of those in which progressive muscular atrophy might possibly occur, the correlation of a symptom complex with the morbid anatomy was due to Schultze, of Dorpatt, and Kahler, of Prague, who, between 1882 and 1888, published conclusive evidence on this point. A clinical picture which exhibited muscular atrophy, sensory disturbances, and trophic changes was now presented, and the diagnosis of syringomyelia was more and more frequently made and cases of amyotrophic lateral sclerosis became correspondingly less numerous. Anæsthesia having been often mentioned as occurring in amyotrophic lateral sclerosis, it is safe to assume that up to this period many cases of syringomyelia had been wrongly classified as instances of what we now know to be a disease confined to motor tracts. During the last quarter of a century the pathogenesis has engrossed a large amount of attention and has been fruitful in widely different theories. The part played by developmental anomalies has been reëmphasized by Leyden, Kahler, and others, with the result that reasons for separating hydromyelia from syringomyelia have appeared less and less justified. The inflammatory theory, originated by Hallopeau, has also found new adherents in Jaffroy and Achard and in Critzman, who regard the disease as "une myélite cavitaire." The view that a morbid vascular condition may exert an important influence has been seriously urged, especially in France, where "une syringomyélie vasculaire" is recognized by some as a separate entity.

It should be stated that Morvan's disease, first described in 1883 and for a time regarded as a separate disease, is included in this article only as a variety of syringomyelia. This is justified by the prevailing opinion that there are no grounds for putting it in a distinct category.

**Etiology.**—*Distribution.*—Syringomyelia can nowhere be regarded as a common disease, although it is now well recognized in all civilized nations. There is no reason to suppose that it is not universal in its distribution, and yet its literature appears to indicate that it is of more frequent occurrence in France and Germany than in America or the British Isles. Statistics are wanting, and it may be that the more careful investigation of the infirmary class of patients in the first-named countries accounts for the apparent difference in numbers.

*Sex.*—It is usually stated that men are more frequently affected, and this is borne out by an analysis of 40 cases which have been in the National Hospital (London) during recent years. Of these, 25 were men and 15 women.

*Age.*—The second and third decades represent the most frequent time of onset. In the 40 cases just mentioned the first indication of any abnormality was observed, on an average, at the age of twenty-four and one-half years. Among the males the average age was twenty-eight and among the females

nineteen, a difference which would probably be less marked in a larger series. The latest date of onset (fifty-six) was in a man, and the earliest in a girl of seven.

*Heredity.*—There is no evidence that heredity plays any important part, and, although the occurrence of the condition in more than one member of a family has been recorded, it can certainly not be termed a familial disease.

*Congenital Anomalies.*—The not uncommon presence of congenital anomalies in the victims of syringomyelia is important. A careful study will often reveal some skeletal peculiarities. The patient may be unduly small, much smaller than his brothers and sisters; he may be infantile in proportion, presenting a large head with small trunk and limbs. Another patient may have unusual deformities of the skull; for instance, bosses in the occipital or temporal regions; he may have, without other signs of acromegaly, very large hands and feet. Pronounced degrees of genu valgum or of pes cavus have been present from early infancy in cases of the disease, and examination of the spinal column may elicit signs of a spina bifida occulta.

*Trauma.*—A history of preceding injury must be regarded with judicious suspicion in all diseases, and syringomyelia is no exception. It is safe only to say that more or less severe trauma to the head and spine is by no means an infrequent antecedent, and that considerable importance has been attached to this fact by some observers. The relationship of spinal hemorrhage to syringomyelia is intimately connected with this question.

*Other Diseases.*—Syphilis may be excluded as a direct cause, but its influence in producing meningitis and vascular disease must be taken into account. The common infective diseases do not appear to have any causal relationship, although the occurrence of one or other of them may coincide with the development of, or may intensify, its early symptoms. The co-existence of conditions such as tabes, spinal caries, etc., is rare and probably unimportant; on the other hand, cases of combined acromegaly and syringomyelia have been recorded sufficiently often to make the association interesting and worthy of further investigation.

**Pathology.**—The exposure of the spinal cord is generally sufficient to reveal characteristic changes. The organ is altered, often very irregularly in shape; the cervical region, as a rule, is enlarged and flattened, the lateral dimension being proportionately more increased than the anteroposterior. The dorsal segments may share in the change, or may be narrow with irregular thickenings. Usually the lumbosacral enlargement has a more normal appearance. The extension upward of the cervical deformity may cease high up in the cord, may occasionally involve the medulla, or very rarely the pons. Folds and furrows are not infrequently observed on the surface of the cord, especially if the fluid contents have drained away. The meninges, in the majority of cases, present no abnormality. Exceptions are found in those cases of spinal cavitation definitely associated with pachy- or lepto-meningitis (often syphilitic in origin), a considerable number of which have been recorded. The anterior spinal roots, particularly those of the cervical enlargement, may be atrophied and of a gray translucent color. On palpation the swollen parts are soft and usually fluctuate; the narrower regions may be normal in consistence or suggest the presence of a hard core lying within an outer ring of the medullary substance. A series of transverse slices will demonstrate striking changes in the interior of the cord, probably varying considerably at different levels. In the region of the greatest swelling,

usually the cervicodorsal region, the section may at first sight appear to have traversed an area of necrotic softening, or even an abscess, but closer scrutiny will show that near the centre of the cord is a cavity, from which clear fluid may be oozing, and that this cavity is surrounded by an enveloping mass of gelatinous material either pale and translucent or yellowish brown, according to the amount of altered blood pigment it contains. The immediate wall of the cavity sometimes stands out as a more opaque pale yellow membrane which has been thrown into folds by the escape of the contents and collapse of surrounding parts. Further sections at different levels will discover the length of the cavity. In some cases it extends through a few segments only, in others through the whole length of the cord. Rarely the tube is prolonged into the fourth ventricle, although in the unique case described by Spiller it reached the right internal capsule.

Frequently the cavity disappears at one level, the surrounding opaque tissue continuing for another segment or two, sometimes to develop another cavity beyond. Two cavities may be present side by side, but if these are traced farther it is usual to find that one is an offshoot of the other. The lumen of the tube varies both in size and shape at different levels.

*Position of Cavity.*—To describe this accurately it is necessary first of all to know its shape and size at any particular level, but speaking generally, these tubes lie behind the anterior commissure, their centre of origin, if one may use the expression, being situated in the gray matter behind the central canal, either on the median line or laterally in the base of one or other posterior cornu. From this centre diverticula may extend in any direction, with resulting varieties in the shape of the cavity in transverse section, but more often than not some part of the posterior white columns are invaded at one level or another. Without doubt the posterior horns and posterior columns are more often involved than the anterior horns, while the anterolateral white matter is rarely encroached upon directly by the cavity, although it may suffer indirectly from the effects of pressure. As a necessary corollary of the above description the shape of the cavity may be round, oval, crescentic, stellate, or irregular, and it is certainly unusual to find any great degree of symmetry in its relation to the two halves of the cord.

In the bulb, as in the cord, the morbid process has a favorite site. Occasionally the spinal cavity may reach the fourth ventricle, but more often it ends below, the only evidence of the disease in the bulb being a fissure or fissures originating in the floor of the ventricle, a little to one side of the midline, and extending forward and outward in such a way as to cut off the restiform body from the central parts of the medulla. Such a fissure may destroy the descending root of the fifth nerve, the solitary fasciculus and some of the nuclei belonging to the vagoglossopharyngeal nerves, but it rarely extends as high as the facial nucleus. It is sometimes unilateral and sometimes bilateral.

What has been said with regard to the position of the spinal cavity applies equally to the disposition of its surrounding tissue, which roughly follows the various diverticula when it does not form prolongations of its own, and which, it must be remembered, extends in most cases to levels beyond the ends of the tube.

Deformities of the surface of the cord and of the outline of the gray and white matter are dependent upon, and present as many varieties as, the distortions of the cavity and its walls.

*Gliomatosis.*—The glial tissue surrounding the cavity varies enormously in thickness not only in different cases, but at different levels in the same case. It may only present the characters of a thin lining membrane to a large cavity, or it may in other instances be so voluminous as to overshadow completely the narrow slit-like cavity it contains. In fact, it may exist in the form of a hard core without cavitation through considerable lengths of the cord. Whatever may be the prevailing theory as to its mode of production, there can be no doubt that it is essentially of neuroglial origin, the term gliomatosis being useful in that it suggests an overgrowth of that tissue and yet separates it from the true glioma which belongs to the group of malignant neoplasms. This gelatinous-looking material is mainly composed of fibers and cells whose relative proportion varies, but it may be stated roughly that there are more cells than in normal neuroglia, and less cells than in a true glioma. The glial tissue may form the wall of the cavity or may be separated from the lumen, either entirely or in parts, by a narrow border of ependymal cells. The peripheral zones of the mass are less sharply defined and merge into the surrounding normal neuroglia and true nervous elements. At levels where no true cavity exists, foci of softening, to which the name granular disintegration has been applied and which are composed of very finely granular or homogeneous material, are occasionally present in the centre of the gliomatous formation.

*Central Canal.*—The fact that the cavity may be lined or partly lined by ependymal cells has been noted, and this condition usually obtains when the cavity represents an enlarged central canal or when a cavity formed in the peri-ependymal tissue has fused with the central canal. Rarely, a cavity situated at some little distance from an intact central canal may be lined with ependymal cells, in which case the explanation is offered that the cavity has originated in a nest of embryonal tissue persisting from the time of the closing up of the posterior columns. In most cases the cavity is either obviously a dilatation of the central canal or appears to be quite independent of the latter. According to Schlesinger the independence is only apparent. At any rate it is quite a common thing to see the central canal, either open or obliterated, lying quite separate from the pathological cavity in a long series of sections. In other instances the cavity is fused with the central canal over several segments and separated from it in regions beyond.

*Vascular Lesions.*—In a certain number of cases the vessels of the cord and its membranes are perfectly healthy, but in others the vessels have undergone definite alterations. For this reason French observers in particular have attached considerable importance to "les espaces vasculo-conjunctifs." Vessels radiating from the surface of the cord into the central glioma present the following changes: The vessel walls and the accompanying glial tissue are swollen, hyaline, and often thrown into folds. As a rule, the lumen is diminished or obliterated, especially when it reaches the glial mass, and there is no evidence of proliferation of the nuclear elements of the walls or sheath. In the neighborhood of the cavity the only remnant of a vessel may be an undulating ribbon of hyaline connective tissue which has been unfortunately called a papillary membrane, and this, owing to degeneration of neighboring tissues, may at times line the cavity or a portion of its wall. Schlesinger has observed these vascular changes in a case of syringomyelia without gliomatosis.

In sections the central parts of the glial mass are usually poor in vessels, but

the more peripheral zones contain a number, mostly running parallel with the long axis of the cord. It is stated that newly formed vessels are found in these parts, but the evidence of this does not appear to be very clear. Hemorrhages old and recent are by no means infrequent, and the amount of pigment may give a decidedly brownish tinge to the tissue.

*Lesions of the Nervous Elements.*—The gliomatosis and cavitation, after attaining a certain bulk, produce changes in the surrounding tissues partly by compression and partly by direct invasion. The latter method is the one by which the posterior columns are principally affected, the newly formed neuroglia eating its way, as it were, between the bundles and producing slow destruction. Closely associated with compression is the production of an œdema of the tissues surrounding the glial mass which is responsible for the rarefaction of the gray matter often seen in the anterior horns. The nerve cells become isolated and their processes disappear, with the result that a species of cavity may be formed to which the name perigliomatous, as opposed to endogliomatous, has been given. A still further loss of glial tissue may eventually lead to fusion of the cavities originating in these two different ways, and thus to the appearance of a large central canal surrounded only by a thin layer of white matter.

The secondary degenerations which result from the lesions of the white matter and from the destruction of ganglion cells in the gray matter conform to the general laws governing secondary degeneration in the cord and peripheral nerves.

Sclerosis of the pyramidal tracts, of the posterior columns, and of the ascending anterolateral tracts is of common occurrence, as well as atrophy of the anterior roots.

Mention must be made of certain neuromata which have been described in the gliomatous tissue, white matter, intramedullary roots, and subglial tissue by Raymond, Schlesinger, Bischofswerder, Hauser, and others. These small tumors are composed of numerous fine medullated fibers running in various directions and accompanied only in some instances by bloodvessels. Their origin is uncertain, Raymond suggesting that they afford evidence of attempted regeneration, and Schlesinger that they are due to local irritation. Patoir and Raveirt have found neurofibromata in the spinal roots in a case of syringomyelia, and have regarded them as of similar origin to the neuromata of the cord and perhaps allied in their etiology to the interstitial changes occurring in the peripheral nerves. The pathological changes which occur in other tissues, in the nerves, muscles, skin, joints, and bones, must be all considered as secondary to the lesions of the central nervous system. They do not present features which are peculiar to this disease.

The occurrence of syringomyelic cavities in association with marked pachymeningitis or leptomeningitis or with vertebral caries is not extremely rare, although the exact relationship to the spinal condition has not been fully established. Still more common is the concurrence of some true neoplasm with the syringomyelia. At any level of the cord, commonly in the cervical region, may be found a true tumor, involving more or less the entire transverse area, which has evidently originated from a part of the gliomatous mass. Such are commonly very cellular gliomata or very vascular angiogliomata, but may sometimes be of a more sarcomatous nature. The writer has seen neoplasms of this kind in the medulla, the cervical and sacral regions in different cases, all associated with syringomyelia.

**Pathogenesis.**—Numerous theories have been put forward to explain the origin and progression of the morbid process. Their number suggests that many cases of spinal cavitation and gliomatosis, although resembling one another roughly in their clinical and anatomical characters, may yet have different modes of origin, and this view is finding more and more favor with those whose knowledge of the disease is most profound. While it is impossible to elaborate dogmatically a genetic theory applicable to every case, a consideration of certain developmental, anatomical, and pathological facts cannot fail to throw some light on particular examples, and may serve as a basis for more scientific grouping and classification.

*Developmental.*—The medullary canal of early fetal life is only represented clearly in the child by the central canal of the cord, a cylindrical tube running throughout the length of that organ lined by ependymal cells of epiblastic origin. The central canal, however, represents only the anterior limb of the medullary canal, the posterior and lateral limbs disappearing in the coalescence of the posterior columns of either side along the posterior median fissures and in the process of forming the gray commissure and the bases of the posterior horns. Normally no cells resembling the ependymal cells of the central canal can be seen in the gray commissure, in the bases of the posterior horns, or in the walls of the posterior median fissure. Presumably, however, there are in these three contiguous parts embryonic remnants, which may take on renewed activity and once again produce neuroglia, the most primitive tissue of epiblastic origin in the central nervous system. On such an assumption the occurrence of a neuroglial hyperplasia appears to have a reasonable, if not complete, explanation and gliomatosis practically always originates either in the gray commissure, the bases of the posterior horns, or in the anterior third of the posterior columns. The occasional presence of groups of ependymal cells away from the central canal may be regarded as a developmental accident in otherwise normal cords, but it is possible that such groups may be the starting point of a peri-ependymal hyperplasia, just as the latter certainly originates in some instances from the walls of the central canal itself.

*Anatomical.*—A consideration of the spinal vascular supply will bring out one important point. That part of the spinal cord which includes the gray commissure, the anterior third of the posterior columns, and the bases of the posterior cornua is the centre of the cord from the vascular point of view. It receives blood from various radiating vessels, of which the most important are those entering along the posterior roots and along the posterior median septum, as well as from some of the terminal branches of the anterior spinal artery. It is the central terminus of those various arteries, and contains, therefore, vessels of small caliber, some of which have a transverse and some a longitudinal course.

This is of importance in two ways. In the first place, any general disease of the spinal arteries which reduces the muscularity and elasticity of their walls will produce its greatest effect on the vascularization of this central zone. In the second place, relying as it does very largely on the posterior spinal arterial system for its blood supply, any morbid process leading to pressure upon or strangulation of the pial vessels on the posterior surface of the cord will have a profound effect upon the same area.

The lymphatic supply of this central spinal area must also be considered, the more so because theories suggesting an infective origin for the disease

have been propounded. The central canal is possibly an important lymph channel, and the presence in it of an irritant agent capable of inciting ependymal proliferation and peri-ependymal gliosis has been suspected. More important is the fact that lymphatic infection of the cord from other organs generally takes place along the lymph channels of the posterior roots. On facts such as these, and experimental work in connection with lymphatic infections of the spinal cord through the peripheral nerves, some have attempted to isolate a group of syringomyelias due to an ascending neuritis.

The closure of the central canal by pressure at one level of the cord has been quoted as a sufficient reason to explain dilatation of that canal at other levels. The justice of this is by no means proved, and, in fact, may be severely questioned when it is remembered that in normal adult cords the canal may be obliterated in some segments and yet patent without dilatation above and below. The whole question of the normal patency and obliteration of the central spinal canal has not been finally answered.

There are certain *pathological* facts to be taken into account, of which the most important is the partiality of intramedullary hemorrhages and abscesses for particular paths in their progress through the cord. There is a great tendency for blood and pus to track from segment to segment along the tissues at the bases of the posterior horns or in the central gray matter, so much so that this area has been termed the zone of least resistance. It is evident that gliomatosis spreads along similar lines, and the close relationship existing between the two processes has led to the belief that many cases of syringomyelia have their remote origin in spinal hemorrhage occurring at birth. Cases have been recorded among adults in which the clinical history has suggested a traumatic intramedullary hemorrhage followed, perhaps years later, by the symptoms of a progressive syringomyelic lesion. It is possible, therefore, that an old pathological process may after a lapse of time be the starting point for gliomatosis and cavitation.

There are other forms of syringomyelic cavities apparently originating from a level of the cord, which has been compressed by meningitis, by vertebral caries, or by some other external cause. It is difficult to see how this comes about unless as the result of evascularization of the cord with the production of necrotic cavities in the central zone and the subsequent spread of disintegration along the favorite lines just mentioned. The presence of gliomatous tissue surrounding the cavity so formed is not easy to explain, but the theory has been advanced that the products of disintegration are sufficiently irritating to incite glial proliferation. Such an explanation appears in a few cases more likely than one which assumes the necessary existence of a congenital abnormality as a predisposing factor.

These developmental, anatomical, and pathological data, together with clinical observations, suggest that various influences are at work in the production of spinal cavities. Many examples of syringomyelia fall naturally into groups in which one or other of these influences plays a predominant part, while others are not so easily classified. Thus there are cases in which a congenital defect appears to carry great weight; others in which a morbid condition of the vascular supply seems too important to overlook, and others in which some source for a chronic lymphatic infection or a history of some traumatic hæmatomyelia require careful consideration. The fact that gliomatosis is always associated in greater or less degree with the formation of these cavities can only be explained on the supposition that the neuroglia

of the central zone of the cord has an inherent tendency toward this form of overgrowth, the initiation of which may be due to a variety of agents, physiological, chemical, mechanical, or pathological. The tendency exhibited by the gliomatous tissue after reaching a certain bulk to undergo central degeneration and then to help in the formation of cavities must be referred either to deficient vascular supply or again to some inherent property of its own.

With regard to the ordinary cases of chronic progressive syringomyelia little hesitation can be felt in placing foremost some congenital anomaly of the central embryonal tissue, the latent activity of which is prone to display itself during the early years of adult life in the form of a slow-growing hyperplasia, having some of the characters of a benign neoplasm and strong tendencies toward the formation of cavities. As far as the other morbid influences we have referred to are concerned, they can only be looked upon as secondary exciting agents or, in very occasional instances, as of primary causal importance.

**Symptoms.**—A disease so prolific in physical signs and symptoms as syringomyelia can never be portrayed by a serial enumeration of its manifestations. The detailed consideration of these must be postponed, therefore, until a picture has been drawn of an ordinary typical case which may serve as a basis for further remarks. A man, aged twenty-seven years, seeks advice on account of some paralysis of his hands. He says that the first symptom occurred about four years before, when a sensation of numbness in his left hand was followed before very long by some weakness and awkwardness of the same part. After some months he noticed that the hand was wasting, the leaders and bones standing out more prominently than on the other side. Little notice was taken of this until he became aware that similar alterations were taking place in his right hand and that he was beginning to find himself awkward in writing, buttoning his clothes, and other fine movements. The legs are perhaps more easily tired than formerly, and on one or two occasions the left toes have caught a step in going up stairs. He says that he has had little or no pain, an occasional "burning sensation" in the region of the neck, but nothing to worry about.

The patient appears healthy and strong, and his face is natural, except that the left pupil is smaller and the left palpebral fissure narrower than the right, giving an appearance of slight ptosis on the left side. This, however, disappears on his looking up, both upper eyelids moving equally well. The pupils react to light, but do not dilate very readily to shade, nor do they react when the skin of the neck is pinched. The ocular movements are natural except that lateral deviation is associated with marked nystagmus. The observer is struck by the good muscular development of the shoulder and upper arm compared to the wasted condition of the forearm and hand. This is more marked on the left side, where the contour of the inner border of the forearm is completely altered and flattened. The left hand is noticeable for the "claw" position of its fingers, the flatness of its palm, and for the fact that the palmar surface of the thumb is parallel instead of nearly at right angles to that of the other fingers. There are an unusual number of scars on the hand and forearm, and the patient, on being questioned, admits to many cuts and burns. One long scar was the result of leaning his forearm on an oven door, the only knowledge of its having been burnt being conveyed to him by the discovery of a blister. The terminal phalanx of the middle finger is missing, and on inquiry it appears that three or four years ago, subsequent to a slight

injury, a whitlow formed, which in spite of incision and scraping on two or three occasions ended after many months in the loss of the top of his finger. There was no pain with it. The skin of the hand is rough and hard, and the remaining nails are thick and striated. All his finger movements are feeble, and flexion of the wrist is much less powerful than extension. Except for the triceps, which may be a little impaired, the muscles about the upper arm and shoulder are quite as strong as their appearance would suggest. Turning the man around, the only thing to catch the eye is a slight yet definite lateral curvature in the dorsal part of the vertebral column, of which the patient is quite unaware. Inspection of the legs reveals nothing visibly wrong, although, if each group of muscles is tested against resistance, dorsiflexion of the feet is not so powerfully performed as it should be. A glance at his boots shows that the toes of each are unduly worn down, more noticeably on the left side. Tapping the patellar or Achilles tendon produces brisk jerks, and stimulation of the soles of the feet elicits an extensor response on either side. An attempt to obtain the abdominal reflexes is unsuccessful. The patient's sensibility to tactile stimuli is found to be perfect everywhere, but of painful and thermal stimuli his appreciation is practically absent over a large area, which includes both arms, the neck, and the thorax as far down as the xiphisternum on the left side and the third rib on the right. On the other hand, his sense of passive movement and position and his stereognosis are intact even in the upper limbs.

The subsequent history illustrates the usual course of events. After a temporary improvement in the strength of the muscles of the upper limbs due to local treatment, the inevitable slow progress of the disease displays itself in the fact that several more muscles in each arm are affected and the area of analgesia and thermo-anæsthesia extends lower down the trunk and over a great part of the left side of the face. Five years later the patient is found to be unable to work on account of the condition of his hands, and his walking has become seriously interfered with by the increasing spasticity of his lower limbs. The right shoulder has recently become the site of a large fluctuating swelling, a skiagram showing that the joint has become completely disorganized. The left side of the tongue is slightly wasted and the left palatal arch lags behind the right during phonation. Inquiry elicits information that the patient's control over his vesical sphincter is not so good as it was, and hesitating or precipitate micturition is the result. A few more years and the bedridden stage is reached, the length of which will depend very largely on the circumstances surrounding the sufferer and the care with which he is nursed. Some pulmonary complication or septic infection will end an illness which may have lasted anywhere from fifteen to thirty-five or even forty-five years.

This is the usual progress, but there are exceptional cases, in which no increase in symptoms may take place for many years, and some in which the disease appears to become permanently arrested. Slow, insidious progress is the general rule, although every now and then a case is observed in which a sudden increase in symptoms has been followed by some improvement, leaving the patient, however, worse off than he was before the sudden change. Such an event has generally been attributed to hemorrhage taking place into a syringal cavity.

Such a history is typical of the majority of syringomyelic patients, and these may be included in a class to which the name "amyotrophic" is applicable on

account of the early appearance of progressive muscular atrophy and the prominent part taken by that symptom throughout. The remaining cases may be divided into three other classes, making four altogether, viz.: (1) Amyotrophic, (2) spastic, (3) Morvan's type, and (4) spinal tumor type.

This classification is useful, although quite artificial, and depends upon the predominance of certain clinical features. Thus, the *spastic* case ("forme spasmodique") is characterized by marked rigidity affecting all four limbs and producing attitudes and fixed positions which are worthy of note. The back is kyphotic, the head low between the shoulders and forced forward in a manner suggestive of paralysis agitans. The hands, too, adopt special attitudes, the three inner fingers flexed into the palm, and the index firmly pressed against the thumb. Such a case may and usually does present some muscular atrophy, although the latter is not so striking a feature as in the amyotrophic class, in which spasticity is less marked and usually confined to the trunk and lower extremities.

The *Morvan's type* of case, at one time regarded as a separate disease, is remarkable for trophic changes in the extremities, especially for a slow necrotic dactylitis, in the course of which most of the small bones of the hands or feet may be lost and the parts deformed almost beyond recognition. Amyotrophy and sensory disturbances are associated with the trophic changes in skin and bones.

The fourth class includes a number of cases in which a diagnosis of spinal tumor is more often made than that of syringomyelia until operation or postmortem examination reveals the presence of a cavity associated with a local gliomatous or sarcomatous tumor, to which the symptoms and physical signs have been mainly due. In such cases the absence of the characteristic dissociation of sensation and of trophic disturbances may have rendered an accurate diagnosis difficult or impossible.

*Onset.*—In 17 of 40 cases this was marked by some disturbance of motion or sensation of one of the upper extremities. In at least half of them the patient first noticed some sensation of cold, heat, numbness, tingling, or pain in the hand or forearm, but in all the weakness and subsequent wasting either followed quickly or were the earliest to appear. At the time they came under observation all had some objective loss of sensation in the affected limb, and it may well be that this preceded the other signs, without its existence having come to the patient's notice. Some difficulty in gait was stated by 8 patients to be their first trouble, but it is remarkable how rarely any incoördination in walking is seen later in the disease. Subjective sensations in the lower extremities were noted before any other symptoms in three cases, one of whom described his right leg as cold and dead, and another his left leg as always hot when the rest of the body and the other leg were cold.

Scoliosis preceded all other signs of disease in four cases, and painless whitlow in a like number. Pain in the occiput and neck (in two instances), difficulty in articulation, and difficulty in micturition were the other initiatory symptoms.

**Disturbances of Motion.**— (*a*) *Atrophic Paralysis.*—This is more common in the upper extremities than elsewhere, usually beginning in the intrinsic hand muscles on one side and spreading up the arm in accordance with the segmental innervation of the muscles and not following the peripheral nerve distribution. Less often it commences in the muscles of the shoulder girdle and rarely in the muscles of the legs. Awkwardness or feebleness of finger move-

ments usually precedes the appearance of obvious atrophy, but later on the amount of paralysis corresponds roughly to the destruction of muscle fibers. In consequence of the interosseal palsy and the preservation of the long extensors of the fingers the hand may present the "main en griffe" appearance. When vasomotor and trophic changes are associated, the "main succulente" is the result. Any muscle which is undergoing atrophy may display fibrillary tremors, and its response to faradic and galvanic currents is proportionately diminished; in some instances true reaction of degeneration is observed. All atrophic muscles sooner or later lose their tendon reflexes.

(*b*) *Spastic Paralysis.*—The degree of spastic paralysis depends upon the amount of degeneration in the pyramidal tracts and its extent upon the level at which the pressure upon, or the invasion of, the lateral columns occurs. Mention has already been made of the spastic form in which the affection of the pyramids extends at least as high as the upper cervical segments, with the result that all four limbs as well as the trunk are more or less rigid. In the more common amyotrophic or, speaking anatomically, cervicodorsal form the spasticity usually appears later and affects the trunk and legs only. In addition to their passive and active rigidity, severely spastic parts are subject to flexor spasms, sometimes of a painful character, which may occur spontaneously or as a more or less easily elicited reflex phenomenon. Associated with the spasticity is the form of flexed hand already referred to and a tendency to clubbing of the feet. All spastic muscles have increased tendon reflexes sometimes amounting to clonus. Occasionally a moderate degree of atrophy is associated with spasticity in the same muscle, which may preserve a tendon jerk of diminished excursion and power. More often than not the atrophic paralysis of one hand is accompanied by spastic paresis of the leg on the same side before the opposite limbs have become affected. In all spastic cases it is the rule to find diminution or absence of the abdominal reflexes and an extensor response to plantar stimulation. These constitute the earliest evidences of this form of paralysis, and are present before any definite paresis or rigidity can be detected.

(*c*) *Incoördination.*—A marked degree is not a common feature, and the majority do not suffer from any great loss of sense of position and active movement. Some interference with this sense may be present in those patients who complain of reeling gait in the early stages. Tabes and syringomyelia occurring in the same individual is not unknown, but the association is only accidental.

(*d*) *Spinal Curvature.*—The common dorsal scoliosis must be regarded in most instances as of muscular origin. The asymmetrical character of the paresis affecting the spinal muscles and the legs is quite sufficient to account for its presence in the ordinary way. When the deformity is extreme or when combined with some kyphosis it is possible that osseous or arthritic changes may be additional factors.

(*e*) *Tremors.*—These are not common features, although fine tremors, resembling those of Graves' disease, and coarse "intention" tremors have been described.

*Bulbar Symptoms.*—When the disease affects the bulb either primarily or as an extension from the cervical region of the spinal cord the clinical picture is often described as "syringobulbia." Atrophy of the tongue, unilateral or bilateral, paralysis of one or both vocal cords or palatal arches are among the more constant symptoms.

Facial paralysis is more rare, and ocular palsies very exceptional. On the other hand, nystagmus is a common feature, and this may be lateral, vertical, or rotatory. The latter type was present in 2 out of 40 cases, and the former was of frequent occurrence. Sensory disturbances in the area supplied by the trigeminal nerve are often present, even without other bulbar symptoms. Defects in articulation, deglutition, and phonation are the results of the various palsies mentioned.

The only special senses to be interfered with in any degree of frequency are those of taste and hearing, and even these are usually intact except in severe examples of the bulbar form. Vision remains unaffected almost without exception, but H. S. Hutchinson has once observed degeneration of the optic nerves.

**Disturbances of Sensation.**—(*a*) *Thermo-anæsthesia.*—This is one of the most constant symptoms, and is almost invariably present to some extent even in the earliest stages. The upper extremities or thorax are usually first affected, extension of the area taking place with the progress of the disease. One patch of thermo-anæsthesia is sometimes separated for a time from another by a healthy area, coalescence occurring later on. The defect in sensibility exists generally for both cold and heat and for all degrees of temperature, but occasionally cold is recognized and heat is not, or vice versa. It may happen that an area of complete thermo-anæsthesia has a border separating it from the normal skin, in which cold only is appreciated. When the defect of sensibility is only partial, it would appear, according to Rosenfeld, that the power of appreciating heat or cold depends on the extent of the area stimulated rather than on the degrees of temperature.

The association of thermo-anæsthesia and analgesia together with complete preservation of tactile sensibility in the same cutaneous region is very characteristic, but tactile anæsthesia may coincide with the other forms, especially in advanced stages. Although thermal sensibility may be absent from the skin and from the mucous membrane of the mouth and gullet, yet appreciation of the temperature of a liquid may be derived from the stomach. A syringomyelic patient may burn his mouth and be unaware of the heat of the food until after it is swallowed.

(*b*) *Analgesia.*—More often than not analgesia is present in thermo-anæsthetic areas. Frequently the distribution of the one is co-extensive with that of the other, but this is not always the case, and wide variations may be observed. Cutaneous analgesia is generally combined with a certain amount of deep analgesia (loss of sensibility to painful pressure), although their degrees may be unequal. Head and Thompson record a case in which an area of complete cutaneous analgesia was co-existent with deep hyperalgesia in the neighborhood of a painful arthropathy of the shoulder. "The lightest pressure stimuli caused pain, and the extent of the hyperalgesia could be marked out on the skin. Yet the superficial layers of the skin were analgesic to the prick of a pin when raised from the subcutaneous structures."

(*c*) *Distribution of Dissociative Anæsthesia.*—Thermo-anæsthesia and analgesia are usually found first in the upper extremities and thorax, and spread thence all over the body. In rare instances they commence in the lower extremities or on the face. Their distribution is nearly always asymmetrical. The borders of the cutaneous loss are not sharp, but shaded off and correspond to the limits of spinal root areas. At the same time charts sometimes show regions of dissociative anæsthesia which correspond

laterally to one or more root areas, but do not cover their longitudinal extent. For instance, the sensory loss in one hand may be limited above by a line encircling the forearm, so as to give it the appearance of a glove distribution. Similarly on the face a central area, including the nose, mouth, and eyes, may preserve its sensibility intact, while the surrounding regions are completely insensitive to painful and thermal stimuli. The explanation of these limitations is somewhat difficult; the presence of a cavity running through several segments and interfering, in the gray matter of each, with corresponding systems of afferent fibers connected with one posterior root after another, may throw some light on the matter.

(*d*) *Tactile Anæsthesia.*—Sensibility to tactile stimuli and with it the power of localizing touches and distinguishing between the head and point of a pin is usually preserved until the later stages, when it is lost over areas of varying distribution. It is lost earlier, however, in that class of cases which has already been referred to as the spinal tumor class, in which no dissociation of anæsthesia may be present.

(*e*) *Loss of Other Afferent Impulses.*—The sense of passive position and movement is often preserved until late, but may disappear progressively from one joint after another in either the upper or lower extremities. According to the observations of Head and Thompson, the power of discriminating between the two points of a compass tends to disappear pari passu with the sense of passive movement. Other afferent but non-sensory impulses, for instance those destined for the cerebellum, may be interfered with and defective equilibrium be the result, but this loss is not so easy of investigation.

(*f*) *Subjective Sensation.*—Subjective sensations occasionally form the initial evidence of disease, and may be of a thermal, painful, or tactile character. Lancinating pains have been described, and cramps of considerable severity are not unknown. More curious is the subjective sensation of drenching sweat in a part which is dry. A girdle sensation occurs in this disease, as it does in many other affections of the spinal cord.

*Vasomotor and Secretory Disturbances.*—These form a very large group of symptoms, many of which, although common in syringomyelia, are not characteristic of the disease. The appearance of an affected limb may vary from time to time, now dry and pale, then moist and hyperæmic. Vasomotor paralysis has the effect of making affected parts react in an exaggerated way to the temperature of the environment, with the result that the skin is overwarm and moist in the summer, and overcold, perhaps cyanotic, and dry in the winter. A cutaneous tâche is of common occurrence in analgesic areas, a pin prick producing a red spot with a central white area which may last for hours, or even days, and may resemble a parasitic bite. Urticarial rashes are very prone to make their appearance with little provocation. A vasomotor disturbance of the subcutaneous tissues is largely responsible for the "main succulente" first described by Marinesco. In this the whole of the dorsum of the hand and all the fingers may swell to an extent which renders their detailed anatomy unrecognizable, while the overlying skin is tense, glossy, and smooth. Pressure produces no marked pitting. Hyperidrosis is another common feature, and often affects the analgesic areas. It may be spontaneous or easily elicited by the warmth of a hot bath. In this way half the head and neck, together with the corresponding arm and side of thorax, may be bathed in sweat while all the rest of the body is dry. Exces-

sive lacrymation or salivation is sometimes observed in bulbar cases, and Guthrie has reported a unique instance of unilateral seborrhœa.

*Ocular Sympathetic Paralysis.*—Owing to the frequent involvement of the first dorsal and eighth cervical segments by the gliomatous process, oculo-pupillary symptoms occur in about 85 per cent. of cases. As a rule, the paralysis of the sympathetic fibers is unilateral, or is more advanced on one side than on the other. On the affected side the pupil is smaller and the palpebral fissure narrower than on the other side. Owing to paralysis of Müller's unstriated muscle, the eyeball is often slightly retracted. The visible effect is one of false ptosis, which disappears at once when the patient looks upward. It is stated that paralysis of unstriated muscle fibers in both the upper and lower eyelids allows of their approximation. Further examination would show that the pupil does not dilate or dilates sluggishly when cocaine is applied or when the eye is shaded, and that no enlargement takes place when the skin of the neck on the same side is stimulated.

**Trophic Disturbances.**—(*a*) *Skin.*—The glossy skin, or "peau lisse," represents the common atrophic variety of the cutaneous affections. Hypertrophy or thickening of the skin occasionally takes place, and this is particularly liable to lead to fissures, which deepen and ultimately ulcerate. All forms of ulceration tend to spread and perforate deeper subcutaneous structures, necrosis of bones eventually being produced. Healing is slow and sometimes impossible. In extreme degrees the panaris of Morvan's type of the disease is the result. Keloid has been known to develop in the cicatrices of these trophic ulcers.

Among many forms of dermatitis which have been described, the eruption of vesicles or bullæ is one of the most common. These may appear suddenly in the night, accompanied sometimes by fever, and are distributed generally over the arms or thorax. The superficial layers of the rete mucosum are only involved at first, but when the fluid has escaped deeper ulceration generally ensues. True pemphigus is a rare but occasional complication.

Trivial injuries may lead to serious septic processes, owing to the analgesia preventing their early discovery and attention. The nails are generally thickened, striated, and brittle; sometimes they tend to drop off. Cases are on record in which nails grew in unwonted positions, while the natural nail-beds remained inactive. The hair may be altered in quality, becoming brittle and coarse, or it may disappear from large tracts of skin. A complete alopecia has been seen by the writer. The growth of hairs in unaccustomed sites has been also noted.

(*b*) *Bones.*—In addition to suffering in the general tissue necrosis, the bones, particularly of the extremities, are liable to undergo trophic changes in the form of rarefaction or increased density. Osseous excrescences or exostoses, symmetrical in distribution, have been observed, and in at least one case suppuration has taken place in the newly formed tissue. Fragility of the long bones has given rise to spontaneous fracture, the occurrence of which is more common in the radius and ulna than in all the other bones of the body put together.

General enlargement of parts of the skeleton occasionally produces a semblance of acromegaly, although it is often asymmetrical and may not be associated with increase of the softer tissues. Chiromegaly is the most common example of this condition. The deformity described under the name of "thorax en bateau" by Marie and Astié is the result of osseous

changes in the upper part of the sternum and upper ribs. In this there is a depression of these parts of the thoracic skeleton, which is associated with a forward and upward projection of the shoulders. It is not always combined with vertebral deformities, and therefore not dependent upon the latter.

(*c*) *Joints.*—Arthropathies occur in 20 to 25 per cent., the incidence on particular joints being very different to that which characterizes tabes dorsalis. Thus, 80 per cent. of the arthropathies of syringomyelia are found in the upper extremities, and the most frequent joint to suffer is the shoulder. As a general rule, the joint troubles are unilateral, but occasionally bilateral and symmetrical arthropathies are observed. According to Schlesinger, males present this symptom more commonly than females in the proportion of two to one. While trauma has an undoubted influence in certain instances, it is equally certain that they may appear spontaneously, and probably do so in the majority of cases.

The development of a Charcot's joint is often sudden, although it may be preceded by pains or by unpleasant sensations in the part about to be affected. The first evidence in some cases is an extensive fluid swelling involving the joint and surrounding tissues, in others a creaking or crepitus evoked by movement. The fluid is apt to disappear in the course of a few days or weeks, leaving a relaxed and abnormally mobile joint. Fresh effusions may occur spontaneously or as a result of some slight injury, every such occurrence producing increased disorganization. The changes are of an atrophic or hypertrophic nature. Sometimes the two varieties are combined. The hypertrophic changes include thickening of the synovial membrane, ligaments, and capsule, with the appearance of osteophytes and cartilaginous masses often embedded in the softer tissues. Bony excrescences originating in the muscular insertions add to the deformities. Ankylosis has occasionally occurred. In the atrophic form the articular cartilage gradually disappears and the subjacent bone may be rarefied and destroyed so as to cause considerable shortening.

In the majority of instances these arthropathies run a painless course, but this is not invariably so in their early stages when deep analgesia may not have developed. Suppuration occurs in a certain number, and this leads to increased disintegration and perhaps to a general septicæmia. Operative measures may often be carried out without an anæsthetic, although sinuses very difficult to close generally persist.

*Disturbances of Control over Excretory and Sexual Organs.*—In the common type of cervicodorsal syringomyelia the interference with the control over the bladder and rectum varies with the degree of spasticity in the lower extremities. In the early stages there may be no abnormality. With moderate degrees of spasticity it is usual to find precipitate or hesitating micturition and rather obstinate constipation associated with precipitate defecation after aperient medicine. In later stages retention and incontinence are experienced. In the more rare lumbosacral form of the disease sphincter trouble may be prominent from the beginning, and in this condition the sexual power is not preserved so long as it is in the more common type. In the spinal tumor type the symptoms of a complete transverse lesion may include priapism.

**Diagnosis.**—Syringomyelia is one of the easiest and one of the most difficult diseases to diagnose. The majority of cases cannot escape recognition if a systematic examination of the nervous system is carried out. The

minority may successfully simulate a number of other conditions for a time, and may even elude discovery until post mortem.

The ordinary atrophic case will seldom be mistaken for progressive muscular atrophy if the patient's sensibility is carefully tested. Vague sensory disturbances with spastic palsies, nystagmus, and sphincter troubles may sometimes suggest disseminated sclerosis, but the absence of the remitting history of the latter and the presence of trophic disorders and perhaps of congenital anomalies point to syringomyelia. Spinal hemorrhage or hæmatomyelia may reproduce the symptoms and physical signs of syringomyelia exactly, but the history of the acute onset will make the diagnosis. At the same time there are cases in which the evidence points to hemorrhage occurring in a syringal cavity, and thus exciting a clinical condition which was merely latent, or others in which the track of an old hemorrhage may possibly be the starting point of a gliomatosis.

By far the most difficult problems are presented by intramedullary tumors, on the one hand, and syringomyelia with localized neoplasms on the other. The signs of a cavity extending above the level of the transverse lesion are often vague and sometimes absent, while the extension downward is generally entirely obscured by the paraplegia. Fortunately the distinction between these two maladies, equally incurable, is not a matter of vital importance. Extramedullary tumors less often cause confusion, but in any case of doubt it is better to explore than to allow any possibility of a removable growth being left.

Finally, it must be remembered that muscular atrophy, dissociative anæsthesia, and trophic changes of skin, bones, or joints are the cardinal symptoms, and that two at least of these must be present to justify a certain diagnosis of syringomyelia.

**Prognosis.**—There is no cure, and therefore the prognosis is never favorable. The chances of life are variable, and can only be estimated by an extensive observation of the rate at which the disease is progressing. The lower the disease the shorter the life is an axiom of some veracity. Fortunately the statement can be truthfully made to patients and their friends that it is not unreasonable to expect an arrest in the morbid process, and to hope that such an arrest may occur before the paralysis has reached the stage of complete disablement.

**Treatment.**—Drugs have no power to arrest the course, and surgery cannot help us. The alleviation of pain, if there is any, and careful protection against all the septic processes to which these patients are so exposed comprise the chief treatment. In the earlier stages massage may temporarily increase the power of enfeebled muscles, but neither this nor electricity will do more. Antisyphilitic remedies should be tried when a history of lues is obtained. Belladonna and ergot will be found useful in relieving the early sphincter trouble and in diminishing painful spasms. Suppuration of bones and other tissues must be treated in the usual manner, amputation being resorted to with more hesitation than in otherwise normal persons.

Speaking generally the patient should be advised to lead a quiet healthy life, paying particular attention to the avoidance of all injuries, however trivial they may appear to be.

## MYELITIS.[1]

**Definition.**—A non-systemic, diffuse, or focal affection of the spinal cord, of heterogeneous yet usually infective and extrinsic origin, disconcerting in the variety of its pathological processes, but characterized by a clinical picture, which depends for its details in individual cases more upon the segmental level than upon the exact nature of the underlying lesion.

**History.**—This is essentially the record of a process of elimination which has been going on for many years and which may not yet be complete. The name "myelitis" has been applied to almost every disease of the spinal cord; it has in its time played many parts and has covered a multitude of diagnostic sins. With the advance of knowledge one entity after another has been separated and given a separate title, with the result that some sort of order has been evolved. Abercrombie (1836) did not favor the word, but used *ramollissement* of the cord to denote any condition of the spinal marrow with modification of its normal consistence. Ollivier, writing about the same time, used the word myelitis, which he had borrowed from Harless, for diseases of the spinal cord.

Only a few years later the field covered by the term myelitis was so wide that Landry wrote an essay in 1853 entitled "Existe-t-il des paralysies indépendantes de la myélite? En cas d'affirmative, tracer leur histoire." Gull, in his careful study of "cases of paraplegia," in 1856–58, appears to avoid the term, but in 1861, while combating Brown-Séquard's advocacy of reflex paralysis, compares cases of myelitis with cases of paraplegia in which no anatomical lesion could be found. About this time myelitis was almost equivalent to organic disease of the cord, and the confusion in regard to the classification of chronic spinal diseases was very great.

Further elucidation of the problem was retarded by the dragging of the red herring of reflex paralysis across the path, but the work of Lockhart Clarke, Gull, Leyden, Charcot, and a host of other observers served to rescue several forms of systemic and other scleroses from the remaining myelopathies. Acute central and transverse myelitis became recognized as definite entities, but in spite of the fact that Bastian, in 1866, had shown that cerebral *ramollissement* was more often due to vascular disease than to inflammation, it was not until about fifteen years later that the possible thrombotic origin of spinal softening was seriously considered. In looking at the records of cases one cannot help being struck by the frequent occurrence of so-called urinary paraplegia, and, making due allowance for the fact that many instances of bladder trouble were probably secondary to spinal disease, it cannot be doubted that myelitis following long-standing cystitis or other pelvic inflammation was much more common than it is now. It is justifiable to suppose that infective myelitis of secondary origin had a clinical frequency and importance in advance of its present position.

The vascular element in the production of spinal softening has gained well-deserved recognition in recent years, and as a result of this and the

[1] Myelitis from a true etymological standpoint only signifies disease of the spinal marrow. The suffix -itis has, however, for a number of years been regarded as denoting inflammation. Consequently, by analogy, myelitis is generally held to mean inflammation of the spinal cord, although it has been used with a great deal of license.

prevailing view that myelitis should only be applied to an inflammatory condition, the desire to introduce such names as spinal thrombosis and myelomalacia has found expression. Syphilis now takes such an important place in the etiology of so-called myelitis that considerable controversy has arisen as to how far the preservation of the name in its wide sense is justifiable.

The difficulties in the way of a general agreement on this and other points may be discussed here because they are of fundamental importance in arriving at a true conception of the etiology and pathology of acute myelitis. In the first place, the result of an acute or subacute focal lesion of the spinal cord is a clinical picture, which varies with the site rather than with the nature of the morbid process. It will be much the same whether the latter is of inflammatory or vascular origin, and in many cases a discrimination between the two is only possible after death, and not always then if the disease is of long standing. For this reason the term "myelitis" is convenient from a clinical point of view, and has by long usage become the natural designation for cases of the kind.

In the second place, thrombosis can never be regarded as the primary and sole factor in any of these cases. As a general rule, it occurs either as the result of disease of the bloodvessels or as one of the phases of an inflammatory process. In the latter case the primary factor is of such a character as to justify the term myelitis. In the former the disease of the bloodvessels may be either degenerative, as in certain forms of arteriosclerosis, or, much more commonly, inflammatory, as in syphilitic arteritis.

Syphilitic thrombosis has some claim to be called inflammatory, and the clinical analysis of the conditions influencing the production of an acute paraplegia must be uncommonly close in many instances if the observer is to give a truly scientific diagnosis. Further difficulty arises from the fact that in syphilitic cases it is often impossible to differentiate clinically a spinal thrombosis from an intramedullary gumma.

The issue must also be considered from its histological side. Is it possible to distinguish, even in recent cases, a purely thrombotic from an "inflammato-thrombotic" lesion? Singer, who has strongly upheld the importance of vascular disease in the genesis of myelitis, makes the presence or absence of a cellular infiltration the chief criterion upon which this question must be decided. He maintains that in cases of syphilitic thrombosis, although the larger vessels may show the usual perivascular small-celled infiltration, yet the necrosed areas are free from cellular infiltration and neuroglial proliferation. In conditions of simple inflammation, on the contrary, the excess of cells both in the perivascular lymph spaces and in the surrounding tissues is an essential and striking feature. The former condition was seen in the cases Singer described, and is undoubtedly present in many others. On the other hand, there are instances of acute spinal syphilis in which thrombosis and consequent necrosis play a very insignificant part, and in which gummatous infiltration of the tissues is so marked that many aspects of a true inflammation present themselves, with the result that the luetic origin of the lesion can only be gauged from the specific changes in the walls of the bloodvessels. Under these circumstances inflammation is at least as prominent as thrombosis in the microscopic field. If, moreover, the opportunity of examining the cord does not take place until a long time after the onset, it is more than probable that an accurate discrimination between its vascular or

inflammatory origin will be impossible. Necrotic, sclerotic, or rarefied areas might be the result of either process, and a thickened, perhaps hyaline condition of the bloodvessels is as likely in the one case as in the other. Nor do the points of similarity end here. In some cases of inflammation of bacterial origin areas of necrosis without cellular infiltration, and not dependent upon vascular occlusion, are met with, and are generally supposed to be due to toxic influences.

In spite of the difficulties, there can be no doubt that at the present day thrombosis due to syphilitic arteritis, and occasionally phlebitis, is responsible for the symptoms of a large number of examples of acute myelitis. It is equally certain that the same criticism can be applied to cerebral syphilitic thrombosis, and it is to be regretted that the close analogy of the two conditions, differing chiefly in their site, is not preserved in their terminology.

Another point of interest now claims attention. It might be supposed that no difficulty would arise in comprising under the term myelitis all cases of spinal disease which are presumably due to the action of bacteria or their toxins, but this is not universally admitted. There are on record instances of paraplegia in which the clinical picture has suggested an acute or subacute inflammatory myelitis, but in which postmortem examination has revealed no interstitial changes, no cellular infiltration of the vessel walls, perivascular spaces, or surrounding tissues, but only degenerative changes in the more highly specialized ganglion cells and nerve fibers. The question as to how far toxic retrograde changes of the parenchymatous elements can be properly regarded as constituting evidence of myelitis is one which has raised considerable controversy and has been thoroughly discussed by Schmaus. The two characteristics of this type of case are (1) œdema of the tissues and (2) degenerative changes in the nervous elements. Both may be present in the more truly inflammatory and infiltrative forms of myelitis, but they may be found in certain cases as the only changes. Storch and v. Kahlden consider that inflammatory œdema is sufficient alone to justify the use of the term "myelitis." Mayer, on the other hand, demands an acute infiltration of the vascular adventitia and perivascular lymph spaces with small round cells.

It has long been the custom to describe as "compression myelitis" the altered condition of the spinal marrow which has resulted from the pressure exerted by tumors, thickened meninges, or displaced bone. In the absence of secondary infection by bacteria it is probably more correct to look upon these changes as due to evascularization and dependent upon the ischæmia produced by slow vascular occlusion. The introduction of some term such as compression evascularization of the spinal cord is not, however, likely to be welcomed.

Reference has now been made to many of the difficulties which beset the use of the term myelitis. They have arisen from time to time as the result of advancing clinical knowledge, improved histological technique, and the growth of bacteriology. No perfect solution can be presented, which would not render confusion worse confounded, and the only course which remains is to retain the word myelitis in its broader and, etymologically, more accurate sense, preserving distinctive characters by the use of qualifying adjectives.

Acute or subacute myelitis will be considered under the following heads: (*a*) Acute infective (or infiltrative) myelitis; (*b*) acute toxic (or degenerative)

myelitis; and (*c*) acute syphilitic myelitis. Acute poliomyelitis will be discussed as an entirely separate disease.

(*a*) **Infective (or Infiltrative) Myelitis.—Etiology and Pathogenesis.**—Infective myelitis is now, at any rate, a rare affection which has no definite relation to age or sex, although young adults are probably its most common victims. It does not occur in epidemic form, nor has it seasonal or climatic proclivities. No real importance can be given to the influence of chill, trauma, strain, or sexual excess, although it is possible that these may be of some predisposing or determining value in certain cases. On the other hand, the onset has not infrequently occurred in the course of one or other of the specific infections. Barlow and Primangeli have recorded fatal cases during or following an attack of measles. Auché and Hobbs and Marinesco and Oetlinger have described its association with variola. Lépine and Schiff have each seen myelitis occurring as a fatal complication of typhoid fever. Gonorrhœa has also been reported in a similar connection by Leyden, Gowers, and others. In the majority of these cases the myelitis must be regarded as secondary, although Lépine leans to the view that a specific typhoid myelitis may be recognized, and Curschmann actually found typhoid bacilli in the cord.

The incidence of myelitis upon pregnancy or the puerperium has been of sufficient frequency to attract attention, but in many such cases the description is not detailed enough to enable one to judge whether they shall be included under the inflammatory or the syphilitic groups. There is probably a special type of myelitis, which occurs in connection with childbearing, and which will be referred to among the "toxic" varieties.

Passing from the more remote to the more immediate causes, it may be presumed that the disease can arise in one of several ways:

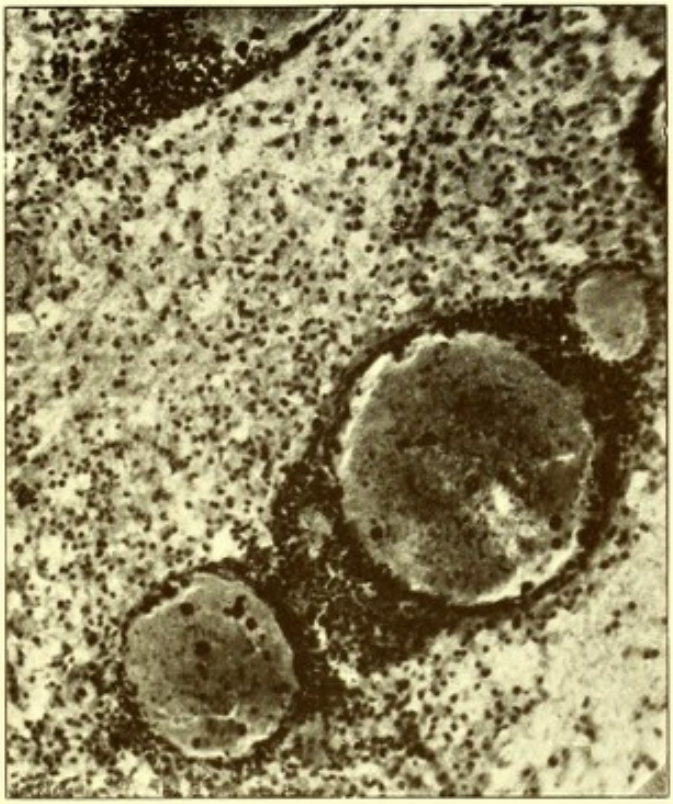
1. As an extension from neighboring inflammatory processes, particularly when the latter involve the meninges or vertebral column. A tuberculous form of myelitis occurs in connection with spinal caries and less frequently with tuberculous meningitis. Some degree of myelitis is often present in meningitis due to pneumococci, staphylococci, streptococci, and other organisms, but in these the symptoms of myelitis are largely overshadowed by those originating from the meningeal affection. On the other hand, infective myelitis practically never exists without some degree of meningitis caused by the same agent. It must then be regarded as an essential part of the disease.

2. As the result of an infection of the spinal tissues through the blood stream. This may take place either as an incident in a general pyæmia or as a purely local phenomenon.

3. As the result of an infection through the lymphatics. The possibility of this is based more on the analogy of experimental pathology than on verified cases of human myelitis. The investigations of Homen and others prove that infection of the spinal cord may take place along the course of the lymphatics accompanying the spinal nerves, and that this may give rise to foci of intramedullary inflammation similar in all respects to those seen in the human cord. The paucity of clinical evidence on this point would be remarkable were it not for the difficulty attending their elucidation, but Marinesco has succeeded in tracing an infection of the spinal marrow by way of the neural lymphatics in a patient suffering from crural gangrene. Further investigation of such sources is urgently needed, and might throw

# PLATE XIII

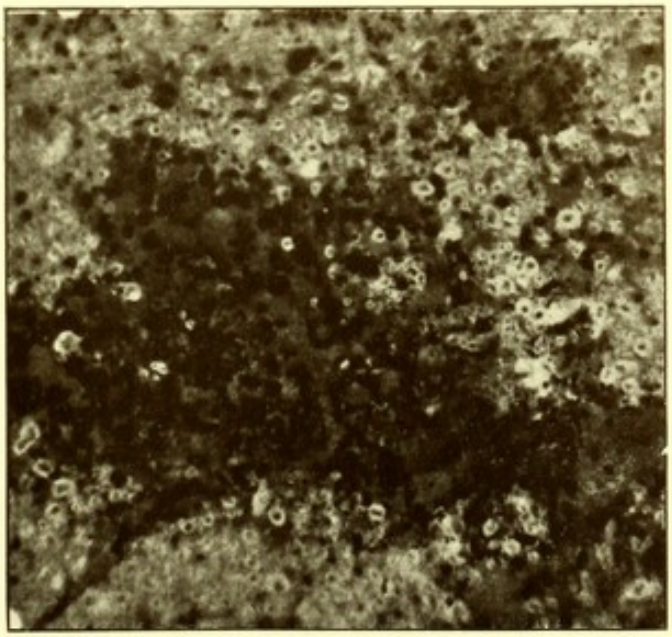
FIG. 1



Acute Infective Myelitis.

Vessels engorged with blood and surrounded by cells in the white matter of the cord.

FIG. 2



Acute Infective Myelitis. Hemorrhage into the white matter.

light on the pathology of spinal disease following cystitis and other septic conditions of the pelvic or abdominal organs.

The bacteriology of myelitis is only in its infant stage, and is hampered by the fact that many organisms appear to be very short-lived in the tissues of the spinal cord. Streptococci and staphylococci have been found in some cases, and a diplococcus was certainly the causal agent in a case reported by T. Buzzard and Risien Russell. Marinesco has shown experimentally that myelitis can be produced by several of the ordinary pyogenic organisms.

**Pathology.**—The gross changes are generally striking and essentially the same in character, whether the extent of the lesion justifies the use of the terms, transverse, diffuse, or disseminated, in order to further describe it. A transverse myelitis is one which is limited in the longitudinal direction to one, two, three, or, at most, four segments, and which affects the transverse area of the cord within these limits more or less completely. A diffuse myelitis involves a considerable length of the spinal marrow without definite interruption, and is often the anatomical equivalent of a clinical "ascending myelitis." The term "disseminated" implies the presence of two or more foci of the disease separated by comparatively healthy intervening tissue.

The morbid appearances vary somewhat with the length of time which has elapsed since the onset. In early cases the affected area is softer than normal, sometimes diffluent, swollen, and œdematous and generally hyperæmic throughout. Hemorrhages may give a dark red or brown color to the cut surface. The definition between the white and the gray substance is frequently lost. The soft meninges on the surface of the altered parts present dilated bloodvessels, and in some cases contain serous or even purulent exudation. The exudation, if present, is usually more profuse on the posterior than on the anterior aspects of the cord. In the later cases the softening is at least as marked, but is associated with shrinkage rather than swelling of the diseased portions. Hyperæmia is less evident, both superficially and on section, and the intramedullary tissue may be mottled with brown or yellowish patches. At levels distant from the seat of inflammation degenerative changes may possibly be detected in the ascending and descending tracts of the white column. In very severe lesions a segment or two of the cord may be reduced to a purulent fluid contained within the pia arachnoid membranes, from which it may ooze unless great care is practised in removing the organ.

At a still later period the consistence of the diseased area is more firm, even sclerotic, and a transverse section may reveal the presence of one or more cysts. The tissues are devascularized, in part opaque white, and in part gray or translucent in appearance. The secondary degenerations in the white matter are now very evident to the naked eye throughout the cord.

**Histology.**—The features belong partly to the vascular tissues, partly to the neuroglia, and partly to the true nervous elements. In individual cases the relative importance may vary, but in all examples of this particular form of myelitis the vascular phenomena play some part. Looking at a section of an acutely inflamed area under a low power, the two striking things are the dilatation of the bloodvessels and the excess of nuclear elements in their walls and the surrounding tissues.

*Vascular Changes.*—There is general engorgement of all bloodvessels both of large and small caliber. The adventitial sheaths and the perivascular lymph spaces show a large excess of nucleated cells. Examination of

the latter under a high power proves them to be chiefly of the lymphoid type. The majority resemble the lymphocytes of the blood, but plasma cells, mast cells, and polymorphonuclear leukocytes are also present. The proportion of the latter to the total number of cells varies very considerably, and depends upon factors which are still obscure. If the lesion is of some days' duration, large cells filled with the granular products of disintegration may also be seen in the perivascular lymph spaces.

The *neuroglia* usually presents marked alterations. It is the seat of an œdema, in the early stages, at any rate, and microscopic evidence of this is either negative or positive. In the former case empty spaces or lacunæ represent the fluid which has been absorbed in the process of hardening; in the latter these spaces are filled with an amorphous granular material deposited from the albuminous fluid by which the tissues have been bathed. The neuroglial cells undergo great changes. Many swell to a large size and display two or more nuclei which may be vesicular. The cell plasma is liable to vacuolation, and the processes may be thickened or lost. In addition to variety in size and shape, the neuroglial cells are greatly increased in number, especially in the neighborhood of the dilated vessels and their lymphatics. Among them are also many migratory cells from the blood, which are not always easily distinguished from those of neuroglial origin. Hemorrhages into the tissues and masses of granular debris are also common in these situations.

*Changes in the Nervous Elements.*—The nerve cells of the gray matter undergo changes which may be partly due to the œdema, but which are mainly the result of toxic influences. They become swollen, rounded, and homogeneous in appearance. The chromatin granules disappear and the nuclei lie excentrically or protrude from the cell periphery. The axis cylinder processes are swollen and often broken. The earliest changes in the white matter are those which affect the myelin of the nerve fibers. This swells and rapidly loses its power of staining deeply by the Weigert-Pal method. The Marchi method reveals the presence of fatty changes. The axis cylinders are also enlarged in transverse and irregular in longitudinal outline. In sections the myelin is apt to disappear in places, and the axis cylinders remain naked and unsupported, with the result that a rarefied and vacuolated appearance is given to the affected areas. Evidence of a varying degree of meningitis in the form of round-cell infiltration of the pia arachnoid completes the picture in the acute stage.

Three weeks after the onset a Marchi preparation shows that a great deal of fatty degeneration has taken place and that much of the fat has been taken up by large granular cells, which are probably of neuroglial origin. Sections from other parts of the cord display in a striking manner the ascending and descending degenerations in the white columns.

At a later period these degenerations are visible only when the Weigert-Pal method is used, and by this time the acute changes in diseased areas have been replaced by those which represent attempted repair. The vessels have lost much of the perivascular cell infiltration, but their walls are thickened and perhaps hyaline in appearance. Proliferated neuroglial tissue has replaced the nervous elements which have been destroyed, and possibly cysts may indicate areas of necrosis. In the sclerotic zones the number and distribution of vessels may suggest that there has been some new formation of the latter. Secondary degeneration appears in the anterior spinal

roots, the efferent fibers of the peripheral nerves, and in the muscles innervated by those segments of the cord in which the gray matter has been seriously involved.

**Symptoms.**—These are dependent on the site and extent of the spinal inflammation, and vary accordingly. In the majority the onset is associated with some constitutional disturbance, pyrexia, anorexia, and perhaps vomiting. The more severe paralytic troubles are usually preceded by pain in the back of a burning character and by paræsthesia in the limbs or in those parts about to be the seat of serious disorders of motion and sensation. Sometimes the attack is really sudden; sometimes one symptom may anticipate by many hours the full development of the disease.

*Transverse.*—A focal lesion affecting more or less completely the whole transverse area of the cord produces a clinical picture which varies according to whether the cervical, dorsal, or lumbosacral regions are involved.

(*a*) *Cervical.*—A focal myelitis high up in the cervical region is rare, and usually rapidly fatal, owing to paralysis of the entire respiratory musculature. More common are cases of myelitis affecting the cervical enlargement. In such a case there may be atrophic flaccid paralysis of the hand muscles and the flexors of the wrist and fingers, while the extensors of the wrist and the muscles moving the elbows and shoulders remain intact; at the same time there is partial or complete spastic paralysis of all muscles below the arms, including those of the trunk and legs with the exception of the diaphragm. Such a distribution of paralysis indicates a lesion of the eighth cervical and first dorsal segments, and is associated with diminution or loss of cutaneous sensibility in the area supplied by those segments and by all the segments below that level.

Respiration is embarrassed on account of interference with the intercostal muscles, and control over the sphincters is impaired. Absence of the abdominal reflexes, exaggeration of the tendon jerks in the lower limbs, clonus, and extensor responses are also observed. The incidence of the lesion on the eighth cervical and first dorsal segments would probably cause irritation or paralysis of the oculopupillary fibers and lead to widening or narrowing of the pupils and palpebral fissures.

The above picture indicates a partial lesion. If the morbid process produces a complete discontinuity of function, the paralysis below the lesion is of the flaccid type, and accompanied by loss of all tendon jerks, at any rate for a considerable time. Moreover, the anæsthesia is complete for all forms of sensation, and trophic changes in the skin, especially bedsores, are readily excited. Total paralytic incontinence of urine and fæces adds to the difficulty in preserving an intact skin. Splanchnic paralysis giving rise to distention of the hollow abdominal viscera is another result of any transverse lesion situated above the dorsal region.

(*b*) *Dorsal.*—In this case the arms escape, and it is generally easier to locate the upper limit of the lesion from the sensory than from the motor disturbances. Paralysis of the lower half of the rectus abdominis may, however, give rise to drawing up of the umbilicus when the patient attempts to sit up. Examination would then reveal the presence of the epigastric and the absence of the hypogastric superficial reflexes, which, with the other observation, points to the ninth dorsal segment as the highest one involved. The atrophic paralysis due to destruction of the lower motor neuron elements is not so easy to detect in the trunk muscles as it is in those of the limbs, owing to their

great vertical length and their innervation from many segments. The paraplegia resembles that of the cervical cases; it is spastic with partial and flaccid with complete lesions.

If the inflammation is limited to either the left or the right half of the transverse area of the cord, the signs are more or less those which have already been described under the name of Brown-Séquard's paralysis. It is, however, rare for an inflammatory lesion to be so limited as to reproduce exactly the symptom complex of an experimental hemisection of the cord.

(*c*) *Lumbosacral.*—Myelitis of the lower parts of the cord may give rise either to a mixed spastic and atrophic or to a purely atrophic type of paraplegia. Rapid paresis, atrophy, and reaction of degeneration in groups of muscles associated with areas of impaired sensibility, loss of tendon reflexes, and paralytic incontinence of the sphincters are the characteristic features. If the disease is limited to the upper part of the enlargement, atrophic palsy of certain muscles of the hip and thigh may be combined with spastic paresis of those moving the ankles and toes. In such a case the knee-jerks may be absent, and yet ankle clonus and extensor plantar responses be easily elicited. A clinical picture of this kind can be readily understood if the segmental innervation of the musculature is remembered.

The conditions so far described represent the fully established disease in its early days. A certain number of cases run a rapid course to a fatal termination, others last a few weeks, but succumb finally to some complication. In those which survive, recovery of function modifies the clinical picture in varying degree. When the recovery is insufficient to allow of walking or standing, the resulting condition, in the cervical and dorsal cases, is one of spastic paraplegia in a bedridden patient who often suffers from painful flexor spasms of the lower extremities, from a sense of constriction at a level varying with the site of the lesion, and from retention or incontinence of urine and fæces. Improvement in the cutaneous sensibility usually precedes and remains in advance of any improvement in voluntary power. Such a patient may live for many years and enjoy good general health if surrounding circumstances are favorable. In less severe instances a remarkable return of function may be witnessed and the patient may recover with a normal gait or more often with some degree of spastic paraparesis, necessitating, perhaps, the aid of a stick. Control over the sphincters may be entirely regained, or any deficiency in this respect may only be noticeable in a tendency to precipitate micturition or defecation after aperients, or after drinking much fluid. Sexual impotence is the rule in most cases of myelitis. Complete restoration of sensibility is general in such cases, and the last evidence of the disease to disappear is the extensor type of plantar response. In the lumbosacral type the prospect of complete regeneration of the atrophied muscles is not bright and the more serious interference with the sphincters adds to the gravity. Considerable degrees of recovery, resulting in a condition of partial disablement not unlike that which follows lumbar poliomyelitis, may, however, be expected in patients who survive the early stages

*Disseminated Myelitis.*—This must be regarded more as a multiple of the transverse type than as a separate disease. It is very rare, and can only be recognized clinically when, for instance, a focal lesion in the dorsal region of the cord causing a spastic paraplegia is associated with an atrophic paralysis of one arm or with a patch of anæsthesia above the level of that which is the result of the dorsal myelitis. Further evidence may be afforded by the

ppearance of some affection of one or other of the cranial nerves, demonrating the extension of the disease to the brain-stem.

More often dissemination of the patches of inflammation is only revealed ost mortem, the clinical picture having suggested a transverse myelitis orresponding to the highest focus in the spinal cord. In other cases the inical course was that of an acute ascending myelitis, although examination f the spinal cord showed considerable dissemination of the lesions. Possibly here is no room for the two separate terms, disseminated and ascending, the ne referring rather to the anatomical and the other to the clinical aspect of he same kind of case.

*Acute Ascending Myelitis.*—This forms a morbid entity which can be eadily recognized during life. It is characterized by a progressive paralysis f motion and sensation beginning in the lower extremities and more or less apidly climbing up the body, segment by segment, until involvement of the espiratory musculature threatens life, and not infrequently produces a atal termination. The extension of the disease to the brain stem is occaonally indicated by the implication of cranial nerves before death, and the ccurrence of optic neuritis has been recorded in non-fatal cases when all ther symptoms have been referable to the affection of the spinal cord. With the more usual employment of lumbar puncture the bacteriology is kely to become better known.

**Toxic (or Degenerative) Myelitis.**—It has already been stated that nfective or infiltrative myelitis is by no means a common disease. Toxic nyelitis must be very rare, and the name can only be applied to a small proortion of all cases. Very little is known about its origin, although it appears rom experiments that bacteria and their toxins are able to produce similar onditions in the lower animals. Moreover, it seems possible that the same rganisms may at one time or under one set of circumstances produce an nfiltrative and at another time or in other conditions determine a toxic form f myelitis. It is equally certain that the effect of some organisms upon the pinal cord is in part to excite inflammation in the interstitial tissues, in part o produce retrograde changes in the nervous elements.

Speaking generally, toxic myelitis differs from infective myelitis in the ss acute, more subacute onset and course, the small amount of constitutonal disturbance, and perhaps the power of more complete recovery. This ast point must necessarily be one rather of speculation than of actual fact, but is natural to suppose that toxic changes, which are known to be capable of ecovery, leave less permanent effects than inflammatory lesions, resulting as hey usually do in necrosis and sclerosis. Of the etiology, little can be said, ecause, as a rule, the toxic nature of the process is not discovered until after eath. As an exception must be mentioned the cases of toxic myelitis assoiated with pregnancy, which have been specially studied and described by . Hösslin and by Rosenberger and Schminche. These authors regard the nyelitis as the result of an auto-intoxication, which may and often does ause at the same time serious trouble in connection with the cardiac and enal functions. The close association between the childbearing and the nyelitis is shown by the improvement in the latter at the birth of the child nd by its recurrence in subsequent pregnancies.

Probably some inorganic substances, lead, alcohol, and certain poisonous ases, are capable of producing a toxic myelitis, but if so they are not comnonly concerned in this connection, and the part they play is referred to

elsewhere. Diphtheritic toxins are among the possible causal agents, but it is doubtful whether they ever excite a general toxic myelitis.

Among the cases of unknown bacterial origin must be grouped those which for many years have gone by the name of Landry's paralysis, but these require separate treatment.

**Pathology.**—Gross changes are not very obvious, but some œdema of the spinal tissues with consequent softening may be observed. On cross-section some degeneration may be seen in the white columns. Microscopically, the chief changes consist in swelling of the myelin sheaths and of the axis cylinder processes, especially in the white matter. The change is generally patchy and shows no tendency to follow a systemic distribution. In addition there are toxic changes in the nerve cells as well as in those of the neuroglia. The ganglion cells swell, become rounded and chromatolytic with excentric nuclei. The neuroglial cells also become round and enlarged, but do not show the great increase in number characteristic of the infective form. In the early stages the Marchi stain demonstrates extensive fatty changes in the myelin sheath; later on the disappearance of groups of these sheaths and the dilated spaces they leave give a somewhat "worm-eaten" appearance to the affected areas.

**Symptoms.**—These depend upon the site of the disease and resemble those of the infective type in all respects. The onset and course are generally less acute, and the power of recovery is probably greater, but in the majority of cases it is quite impossible to differentiate the two conditions at the bedside. Motion, sensation, and sphincter control are all impaired and the relative amount of muscular spasticity and atrophy varies with the degree in which the upper and lower neurons are involved in the morbid process. In a case described by Rhein the clinical picture resembled that of a subacute ascending myelitis of the inflammatory kind. Post mortem, only cell changes and myelin degeneration were found to account for the symptoms. The cases in pregnant women are also characterized by an ascending progress of their symptoms. In fatal instances bulbar symptoms followed those of spinal origin.

**Syphilitic Myelitis.—Etiology.**—Syphilis is by far the most important factor in the production of myelitis, and for this reason cases of syphilitic myelitis easily outnumber those of other origin. Singer found that out of 19 cases of myelitis admitted in the course of two years into the National Hospital (London), 15 had a definite history of syphilis. In 12 of them the onset of myelitis occurred within three years of the primary infection. In the opinion of the writer these figures, roughly 80 per cent., do not exaggerate the preponderating causal influence of syphilis. Males are much more frequently affected than females, and the greatest susceptibility prevails during the third, fourth, and fifth decades. It is probable that age has also some influence upon the rapidity with which myelitis follows the infection, the interval being shorter in the younger patients. This interval varies from a few months to many years, but the liability to the disease diminishes after three years have elapsed. Gummatous meningomyelitis may also be the result, although rarely, of congenital syphilis. In at least 75 per cent. of cases of syphilitic myelitis the incidence of the disease is upon the dorsal region of the cord and more particularly upon the middle third of that region. This may be explained by the fact that this area is more poorly supplied with large contributing root vessels than any other, and is in distinct contrast to

the incidence of the infective type which favors the lumbar and cervical enlargements at least as frequently as the dorsal region.

It may be doubted whether any other factor than that of lues is of material importance in the etiology of this form, but it seems possible that great fatigue, exposure, and perhaps alcoholism may have an exciting influence. It is certainly common among soldiers.

**Pathology.**—The influence of the syphilitic virus upon the spinal tissues is not a simple one, but is exerted in a number of different ways. Syphilitic myelitis is really the manifestation of more than one morbid process, and each variety may be represented to a greater or less degree in one case of the disease. It may be said that the spirochæte produces its results in part by its power of causing changes in the walls of bloodvessels with consequent effects upon the local circulation, in part by the diffusion of toxic substances capable of exciting profound influence upon certain of the tissues, and in part by exciting a cellular infiltration of an inflammatory or granulomatous character.

Any or all of these results may be concerned in any particular case, but, as a rule, one or other has a more or less preponderating importance. It must always be remembered that morbid changes may be silently at work for a considerable time before some sudden catastrophe, such as the occlusion of an important bloodvessel, provides a suggestion of acuteness to the illness to which it is in reality hardly entitled. For much the same reason it is the rule to find evidence of syphilitic changes in the bloodvessels and meninges over a much wider area of the cord than the segment or two forming the site of the lesion which has determined a disturbance of function. Although concerned for the moment with the intramedullary changes, the fact that these are often secondary to, or intimately associated with, syphilitic processes in the surrounding tissues, especially with those in the dura mater, must not be lost sight of.

The incidence of syphilitic myelitis is much more frequent in the dorsal region than elsewhere, but wherever placed the focus of disease is generally well defined. The lesion is usually single and generally limited to a very few segments, so that it may be included under the term transverse myelitis. The diseased area is recognized by its soft consistence and possibly by some slight alteration in color. The pia arachnoid overlying it may be milky and contain distended veins, but the milkiness may not be more noticeable at the level of the softening than elsewhere. In long-standing cases there is marked narrowing in the caliber of the cord, as well as a wrinkled condition of the membranes covering it. Atrophy of anterior roots arising from the softened segments may be detected. Adhesions connecting the dura mater with the leptomeninges are of frequent occurrence and may be too dense to permit the former to be separated from the cord.

**Histology.**—Having regard to the variety of the processes involved, the changes in each kind of tissue are best considered separately.

*The Bloodvessels.*—The vascular changes may be divided into at least three chief types: (1) Moderate perivascular and adventitial cell proliferation with thickening of the intima of sufficient degree to produce diminution or complete occlusion of the lumen. This may be very general throughout the cord or confined to the area of softening. The proliferation of small round cells may only be slight, and yet the walls of the vessel may be very thickened and perhaps hyaline in appearance. This would suggest that

the morbid process has been subacute or chronic, and that the acute onset of symptoms has been due essentially to a rapid ischæmia originating in a local obstruction or following on a general lowering of blood pressure. (2) Excessive perivascular cellular infiltration with little or no endarteritis. In this case there may be no evidence of actual obstructive thrombosis, although slowing of the circulation and blood stasis is suggested by the excess of white corpuscles in the vessels. On the other hand the changes in the neighboring tissues are probably due to a toxic necrobiosis or to an extension of the cellular proliferation from the immediate precincts of the vessels. (3) Paralytic vasodilatation with very moderate perivascular infiltration and with no obvious thickening of the vessels. Retardation of the blood stream with capillary thrombosis and profound alterations in the nervous elements are the characteristics of this change. The general appearance produced is one of hyperæmia of the white as well as of the gray matter associated with retrograde changes in myelin sheaths and ganglion cells. At the same time, there is no true necrosis of the tissues as a whole, although an amorphous, perhaps colloid, exudate permeates their interstices.

*The Neuroglia.*—This offers the greatest amount of resistance to the vascular changes. In areas of complete necrosis it succumbs with everything else, the cell nuclei rapidly lose their staining reaction, and the cells as well as fibers taking part in the general liquefaction. In other regions the neuroglial cells survive when the more specialized structures are destroyed. Under these circumstances they are enlarged, rounded, and often multinuclear. In later stages they proliferate and take a leading part in the process of repair, sending out long interlacing fibers in all directions. It is not improbable that they give origin to at any rate some of the large granular cells, which act as scavengers and which, at first distributed in the tissues, are later collected in large numbers in the perivascular lymphatics. With the neuroglial changes must also be mentioned the gummatous infiltration which occasionally goes hand in hand with the vascular phenomena. In some instances this forms the predominant feature, and a true neoplasmic gumma, with the usual central degeneration and caseation, occupies a considerable part of the transverse area of the cord. In others the aspect is that of a gummatous meningomyelitis, either involving the cord in the form of a ring or penetrating its substance at one or two points.

*The Nervous Elements.*—Demyelinization of the nerve fibers and retrograde changes in the ganglion cells are the results either of the circulatory disturbances or of the diffusion of toxins. In the course of the morbid change the myelin sheaths swell, disintegrate, and finally disappear leaving axis cylinders in a naked, unsupported, but often swollen and perhaps varicose condition. The ganglion cells undergo the usual changes in shape, in granulation, and in nuclear displacement, which have been described. These alterations are strictly local and do not extend into parts of the cord which are distant from the myelitic focus. On the other hand, they lead to secondary degenerations, which can be traced through the central and peripheral nervous system in accordance with the Wallerian laws.

*The Leptomeninges.*—Round-cell infiltration of the pia arachnoid is almost universal in the syphilitic form, and is usually associated with varying degrees of meningeal thickening. The arteries are often hypertrophied, and the veins may display an obliterative phlebitis. The walls of the latter not infrequently present a laminated appearance, layers of round cells being separated

from each other by strands of connective tissue, with the result that their bulk is at least as great as that of the arteries.

The ultimate results resemble those of other forms of myelitis. The processes of repair, of sclerosis, of new vessel formation, and perhaps of cyst or cavity production are essentially the same, and an examination of a focal lesion a year or more after the acute stage might reveal nothing which would enable the observer to speak with certainty as to its exact origin.

The type of syphilitic myelitis which has been referred to as transverse is the common one, and it is rare to find a diffuse myelitis of acute or subacute syphilitic origin. Occasionally a few patches of disease scattered through a few segments may invite the term "disseminated," but the clinical picture even then is rather that of a transverse myelitis. On the other hand, it is not rare for a spinal syphilitic lesion to be associated with a cerebral one of the same nature, and occasionally their occurrence is contemporary.

**Symptoms.**—Perhaps no disease has a more characteristic onset than syphilitic myelitis. A man in good health, for no reason that he can see, finds himself unable to empty his bladder, or finds one or both legs heavy and weak or numb and "prickly." He is reassured in a few hours by the disappearance of this trouble, and it may be two or three days or two or three weeks before anything further occurs. Then, perhaps after an unusually long day's work, or perhaps with no departure from the ordinary routine, he rapidly, sometimes suddenly, loses power in his legs, loses his ability to micturate, and is conscious of a numbness from his waist downward. Throughout this period he has experienced no feeling of illness, and unless he is anæmic and sallow, as so many victims of recent syphilis are wont to be, has been regarded by his relations as strong and healthy. Even as he lies in his paraplegic condition his appetite and temperature are little disturbed. Such a history is common enough, but it may be varied by the absence of any premonitory symptoms or by the more gradual development of the paralysis. For instance, one leg may be paralyzed some hours or even a day or two before the other, and very occasionally the disablement is limited from the first more or less completely to one lower extremity, with the production of a modified form of Brown-Séquard's paralysis. Not uncommonly the patient has gone to bed in his usual health, and on getting up in the morning falls to the ground. Pain is not a prominent feature; it may be absent altogether, or the patient may complain of a burning sensation at a particular spot on the spinal column. A girdle sensation corresponding roughly to the upper border of paralysis may be present early or may supervene in the later stages, but paræsthesia referred to the paralyzed limbs is frequently experienced. Such is the onset of a dorsal syphilitic myelitis, and the results of an examination will correspond more or less exactly to those alluded to under infective myelitis of the same region. According to whether the physiological continuity of the spinal cord is completely or incompletely severed at the site of the lesion a flaccid or a spastic paraplegia will present itself. In the former case paralysis will be complete from some level on the trunk downward, the legs will be extended, toneless, and powerless; all deep and superficial reflexes will be abolished. The bladder may at first empty itself at intervals as a reflex involuntary act without the consciousness of the patient, but later will dribble more or less continuously or whenever any change of posture is attempted or the abdomen is pressed upon. The relaxation of the anal sphincter may be realized by the introduced finger; solid fæces may be

retained, but diarrhœa will lead to constant soiling. Incomplete priapism is sometimes observed. Loss of sensibility to all forms of stimuli exists up to the segmental area corresponding to the spinal disease, and a band of hyperæsthesia may be present just above that level. The skin of the lower extremities is dry and sometimes œdematous. Of extreme and urgent importance is the liability to bedsores and cystitis.

In the spastic cases the lower extremities are usually flexed at the hip and knee, and strong adductor spasm brings the limbs in apposition. The tendon-jerks are lively; ankle clonus and extensor responses are easily elicited. The mere handling of the limb or the movement of bedclothes suffices to excite painful clonic spasms, which may last for some little time. The relation of the sensory disorders to the paralysis is very variable, but sensation is nearly always less affected than motion. Constipation and retention of urine with overflow incontinence are the characteristic sphincter troubles in this type.

In the more rare cervical and lumbosacral cases the symptoms are those already described under infective myelitis.

A few words only are necessary for the purpose of indicating the course. A flaccid paraplegia may become spastic in the course of a few days or a week or two. Rarely does a case of the flaccid type survive long in that condition, a fatal termination being usual. The spastic cases, under appropriate treatment, display a general tendency toward improvement and the less complete the initial paralysis the more favorable is the ultimate outlook. Only a very few cases recover completely, but a considerable number attain a condition in which they are able to get about with more or less difficulty and with greater or less degrees of spastic gait. Not infrequently the sphincter troubles prove very obstinate, and the patient is worried by urgent and precipitate micturition and defecation.

**Diagnosis.**—This can be conveniently divided into two parts: (1) The diagnosis of acute myelitis from other forms of paralysis, and (2) the discrimination between the various forms of acute myelitis.

1. The diagnosis of acute myelitis from other forms of paralysis is not attended, as a rule, with much difficulty in the simple transverse cases. The rapid onset of a paraplegia with a well-defined upper limit to the motor and sensory disturbances could only result otherwise from some sudden pressure exerted by displaced bone or by a tumor. In the very exceptional cases in which symptoms of compression are really acute there is usually some indication of the gross nature of the lesion. Moreover, pain is generally a more prominent feature in these cases. Compression paraplegia developing in a patient who is suffering from cancer may suggest acute myelitis if the possibility of compression is forgotten. More deceptive are the attacks of acute, although often incomplete, paraplegia which form part of the course of disseminated sclerosis, and which sometimes constitute the earliest symptom of that disease. The absence of a well-defined upper limit to the paralysis and the presence of some unsuspected symptom or physical sign above the level of the lesion will generally clear up any doubt. The observer should pay particular attention to the cranial nerves and keep a sharp lookout for nystagmus, diplopia, or pallor of the optic disks, the presence of any one of which would point strongly toward the diagnosis of disseminated sclerosis. An acute form of multiple sclerosis has occasionally been described, and may require careful differentiation from acute dissemi-

nated myelitis. A lumbosacral myelitis with its atrophic changes in the lower extremities may simulate an acute poliomyelitis until the presence of anæsthesia and of severe sphincter troubles are recognized.

Only a few years ago the distinction between myelitis and peripheral neuritis was a prerogative of the expert. Nowadays it is hardly necessary to draw attention to the differences between the two diseases. In the dorsal and cervical cases no mistake can arise. In the lumbosacral form of myelitis it must be remembered that the distribution of sensory and motor phenomena follow a segmental arrangement, and have not a special incidence upon the peripheral parts of the limbs as they have in neuritis. Furthermore, the sphincter disturbances of the myelitic, and the muscular tenderness of the neuritic cases help to distinguish the two conditions. Finally, a case of peripheral neuritis is not very often sufficiently acute to give rise to the suspicion of an acute myelitis.

In all cases of myelitis lumbar puncture should be performed, and the examination of the cerebrospinal fluid will sometimes settle any doubts as to the nature of the disease.

Many cases of dorsal myelitis have been regarded in their first stage as functional or hysterical, especially when young women have been the victims. Loss of power and sensibility, very brisk tendon-jerks, pseudoclonus, and even retention of urine may be found in functional paraplegia, but incontinence of urine or fæces and, above all, an extensor type of plantar reflex serve as infallible signs of an organic basis for the paralysis.

2. A diagnosis of transverse myelitis having been arrived at, the next question to be answered really amounts to this: Is it syphilitic or the result of some other infective process? The history, or absence of history, of syphilis must of course be allowed due weight, and when an undoubted luetic record exists it will necessarily indicate the line of treatment. In doubtful cases the mode of onset affords some guide. With those of syphilitic origin premonitory symptoms are often observed over a considerable period before the actual development of the disease, and the initial stage is generally associated with no constitutional disturbance. In the infective type, on the other hand, warnings are less common, and, when they do occur, precede by a few hours only the onset of paralysis. Moreover, some degree of general malaise, general pain, pyrexia, and perhaps vomiting are the rule rather than the exception in these patients. The site of the lesion is not of very great diagnostic significance, but, *ceteris paribus*, the dorsal region favors the syphilitic, and the cervical enlargement the infective form. The examination of the cerebrospinal fluid may or may not be of assistance. The presence of organisms or the discovery of the *Spirochæta pallida* would of course be more or less decisive if secondary infections could be excluded. A preponderance of polymorphonuclear leukocytes would point to a non-syphilitic virus, but a lymphocytosis does not afford definite proof of the luetic nature of the lesion.

If syphilis is suspected careful search should be made for corroborative evidence in other parts, and the pupillary light reaction in particular should be the object of investigation. A myelitis not infrequently represents one feature or phase of cerebrospinal syphilis, and it may be associated with one or other of the parasyphilitic diseases. The writer has seen tabes, myelitis, and cerebral thrombosis all present in one subject. Equally important in the infective type is the discovery of some source for the secondary invasion

of the cord, and the possibility of gonorrhœa as well as of septic processes must be diligently inquired into.

The acute ascending types of myelitis, whether of infective or toxic origin, present a clinical picture, which may for a moment suggest a Landry's paralysis or an acute poliomyelitis, but the presence of sensory disturbances and of sphincter trouble suffice to exclude the latter diseases.

**Prognosis.**—The general outlook has already been indicated in describing the progress of the different forms, but certain principles may be deduced which should be of use in an individual case. The severity of the lesion is of importance both as regards the prospect of life and the hope of useful degrees of recovery. A complete loss of conducting power in the cord at the site of disease, although it may only be temporary, is a menace to life whenever it presents itself, and the higher the level of the lesion the greater the immediate danger. Interference with the respiratory musculature may lead to a rapid fatality, and even when the dorsal region is the part affected the tendency to severe decubitus and to cystitis with its renal complications constitutes a grave element. Should the patient survive these dangers, the prospect of good recovery in such severe cases is a very poor one. Instances of lumbar myelitis are particularly hopeless in regard to the possibility of regaining the power of walking, and their irremediable sphincter troubles usually determine an early death. Less complete paraplegias may be confidently expected to show marked improvement, but only after the effects of treatment, especially in the syphilitic cases, have been gauged, can any precise estimate of the ultimate result be formed. It must be stated that cases of spastic paraplegia resulting from myelitis are disappointing to themselves and to their physicians. Improvement sets in and goes on up to a certain point, and then when hopes of further success appear justified, progress is arrested and a stationary condition, obstinate to all forms of treatment, is permanently maintained.

**Treatment.**—*Infective and Toxic Cases.*—In the acute stage the patient must be given complete rest preferably, and often imperatively, on a water bed. Changes of posture are necessary for the prevention of sores, but cannot be seriously recommended for any influence they can exert upon the morbid process. The supposition that the volume of blood in the spinal cord is lessened by placing the patient on his face, or that such a depletion, if it occurs, is beneficial, do not appear to have any reasonable basis. On the contrary, the determination of blood to the site of infection must be regarded as an essential factor in Nature's effort of resistance and the effect of gravity in altering the hyperæmia as insignificant. Immediate attention should be paid to the bladder, and the urine drawn off if there is retention. Free action of the bowels should be secured at least every alternate day after an initial purge, and the greatest care must be taken to keep the skin clean. If pyrexia and pain are present, a diaphoretic mixture containing salicylates or quinine and a light fluid diet are indicated. There is no reason to believe that the application of heat or cold to the spine can be of use, and disturbing the patient for the purpose of cupping is probably more harmful than beneficial. A lumbar puncture should be performed at the earliest opportunity and an examination of the fluid carried out. As soon as acute symptoms have passed off all paralyzed parts should be rubbed and moved every day, and each joint prevented from becoming fixed. In the dorsal cases the spastic muscles need no treatment, but in the

lumbosacral cases atrophied muscles of the lower extremities should receive electrical treatment, provided that this can produce contractions. The kind of current must be chosen accordingly. A careful watch must be kept upon the urine and any sign of cystitis combated by means of urotropin by the mouth and by irrigation of the bladder with some antiseptic solution. The sphincter trouble may in the less severe cases be favorably influenced by giving belladonna internally, and the same drug combined with extract of ergot may relieve the painful flexor spasms which so often cause much suffering and are sometimes very difficult to treat successfully. These spasms are particularly liable to be troublesome at night, and when sleep is much disturbed by them the use of such drugs as veronal, sulphonal, hydrobromide of hyoscine, and even morphine is not only justifiable, but urgent.

As the patient gains in strength more energetic massage and passive movements may be carried out, and he should make every endeavor to perform movements on his own account. It is his duty to force impulses, as it were, through the block on the conducting lines or to find some other way round for the resumption of traffic. This side of the treatment is apt to be forgotten in the modern craze for massage and electricity. If the patient can be induced to attach less importance to the energy displayed by the rubber and to impart more energy into his own efforts at initiating voluntary movements, the medical attendant will have gained valuable coöperation in his task. When the limit of improvement by these means appears to have been reached a change to some suitable health resort should be advised.

*Syphilitic Cases.*—Antisyphilitic treatment can in most cases be commenced as soon as paralysis sets in, or, better still, during the premonitory stage. Both mercury and iodide of potassium should be given, the method of administration being of minor importance. Beginning with 5 grains (gm. 0.3) three times a day, the iodide of potassium should be increased rapidly until 20 to 30 grains (gm. 1.3 to 2) are taken in each dose. The mercury may be given by the mouth in the form of perchloride or the red iodide, by inunction, or by hypodermic injection, but it is not advisable to apply the inunction or the injection to anæsthetic parts. This should be pushed to salivation and renewed after an interval of two or three weeks. Bearing in mind the fact that the victim of syphilitic myelitis is prone to other manifestations of cerebral or spinal syphilis, and that there is no criterion as to when that liability is successfully eradicated, the patient should undergo a course of mercurial treatment two or three times a year for an indefinite period. Freedom from further syphilitic lesions may be easily secured in this way by the simple administration of gray powder for a few weeks at a time at the stated intervals. It must be admitted that there are instances of syphilitic myelitis in which these remedies are of no avail, but they form the exceptions to the rule.

It is doubtful whether any other drugs can take the place of mercury and the iodides in the treatment of the syphilitic lesions of the cord, although atoxyl deserves a trial, and may, at any rate, be used in the intervals between the courses of mercury.

## CHRONIC MYELITIS.

There are perhaps few facts more deserving of recognition in neurological medicine than the extreme rarity of a primary chronic myelitis. One meets with patients who exhibit the results—generally a stationary spastic paraplegia—of a former acute or subacute myelitis, but rarely, if ever, does one encounter a case in which slow progressive symptoms of a spinal affection justify the diagnosis of a chronic myelitis. If such a diagnosis suggests itself every effort must be made to exclude (1) the various forms of systemic scleroses, such as subacute combined sclerosis, amyotrophic lateral sclerosis, etc.; (2) disseminated sclerosis; (3) syringomyelia; and (4) compression paraplegia due to tumors or vertebral disease. It is to the exclusion of spinal compression that attention must especially be paid, because the life of the patient may depend on the accuracy of the opinion.

It may well be asked whether, if the chronic stages of acute myelitis and the list of diseases quoted above are put on one side, there is any condition which deserves the name of chronic myelitis. The answer to this is in the negative if an exception is made in favor of certain ill-defined and rare cases of syphilitic spinal disease to which reference will shortly be made. In other words, slowly progressive symptoms pointing to a lesion of the spinal cord indicate in the vast majority of cases one or other of the diseases above mentioned, and not a chronic myelitis. It is true that a chronic myelitis secondary to some infective process, such as tuberculous disease of the vertebræ and spinal membranes, may be induced, but the primary cause must be firmly established before the chronic myelitis can be legitimately inferred.

In former days the term chronic myelitis was used as a cloak to ignorance, and many cases of multiple sclerosis, of subacute combined sclerosis, and particularly of compression paraplegia went under that designation. The importance of a general recognition of this is especially obvious in cases in which there is evidence of a focal transverse lesion of the spinal cord with symptoms increasing in intensity. These must not on any account be labelled chronic myelitis, but, speaking generally, must be submitted to surgical exploration in the hope that the removal of a tumor pressing upon the cord may change the prognosis of the case from that of a progressive incurable paraplegia to one which holds out good prospect of a complete or partial recovery. Emphasis having been laid upon the rarity of any spinal disease justifying the diagnosis of chronic myelitis, a condition which is known under the title of Erb's syphilitic paraplegia may be briefly described, although the writer has some doubts as to how far a separate morbid entity answering to that name may fairly be recognized.

**Erb's Syphilitic Paraplegia.**—In 1892 Erb sought to establish a type of disease to which he gave the name "syphilitic spinal paralysis." According to his view the disease was a form of spastic paraplegia characterized (1) by its syphilitic origin; (2) by the marked exaggeration of the tendon reflexes as compared to the moderate degree of muscular rigidity; (3) by bladder trouble, which might precede by a long time the other symptoms; (4) by distinct paræsthesias of subjective rather than objective character; and (5) by the gradual development of the disease and its tendency to improve under appropriate treatment. Rumpf had already shown that all these symptoms could be the result of syphilitic disease of the bloodvessels of the

cord, though he had not laid so much emphasis on the activity of the reflexes in relation to muscular rigidity. At the present time it must be confessed that this point is not characteristic of Erb's type, because it may be observed in the late stages of acute myelitis and under other conditions.

The occurrence of this form of spastic paraplegia with a gradual onset and progressive course must be very rare, but it is interesting, as it provides at any rate one possible form of chronic syphilitic myelitis. It would be suggested in any particular case by a history of syphilis, and its diagnosis would be readily confirmed by a lumbar puncture, with examination of the fluid, and also by the results of antisyphilitic treatment. In a case of the kind which came under the writer's observation the bladder trouble anticipated the other spinal symptoms by many months, thus raising the suspicion that some urethral disease was responsible for the difficulties associated with micturition.

**Pathology.**—Little is known about the anatomy of these cases, because the majority, which have been called examples of Erb's syphilitic paraplegia during life, have been shown post mortem to be really the subjects of a transverse syphilitic myelitis in the dorsal region of the cord, with the usual ascending and descending tract degenerations. Other cases are the result of diffuse syphilitic disease of the spinal vessels, sclerosis taking place in areas which are poorly nourished. Finally, it is stated, without much positive evidence, that the syphilitic toxins may induce a degeneration of the pyramidal tracts and of the long ascending tracts in the posterior and lateral columns without the intervention of any specific changes in the blood-vessels or meninges of the cord.

**Diagnosis.**—This must depend on the progressive character of the symptoms and on the results of lumbar puncture and antisyphilitic treatment. The absence of pain serves to distinguish it from most, but not all, cases of compression due to tumors or vertebral disease. The definite limitation of motor and sensory disturbances to parts of the body innervated from below a certain level in the spinal cord must always suggest a focal lesion, and not a diffuse affection of the long tracts in the cord, such as is found in Erb's syphilitic paraplegia.

**Prognosis.**—Appropriate treatment tends to ameliorate the spinal symptoms, and some improvement in the paraplegia may generally be anticipated, especially if an early diagnosis is made and acted upon. On the other hand, it is unusual to obtain a perfect recovery, particularly in regard to the vesical troubles. A patient may reach a stage in which he is able to perform all his ordinary duties, and even to walk many miles without great fatigue, but his control over micturition often remains very incomplete.

**Treatment.**—What has already been said with regard to the treatment of acute syphilitic myelitis applies to this more chronic type of lesion. The danger of bedsores and of cystitis is not nearly so urgent, and complete rest is rarely indicated. Mercury and the iodides should be administered, persevered with, and repeated at intervals over a long period of time. If the bladder trouble proves intractable, regular daily catheterization, with due precautions against sepsis, should be instituted, and the patient taught to carry this out himself. A portable urinal is sometimes a convenience and occasionally a necessity.

## LANDRY'S PARALYSIS.

**History and Introduction.**—In 1859 Landry described a case of acute paralysis which ran to a fatal termination in the course of a few weeks. Certain clinical features and the absence of any demonstrable changes in the spinal cord made the case remarkable, and subsequent records of similar cases led to the recognition of a morbid entity bearing the name of its original observer. During the last half century this disease, or, at any rate, its title, has enjoyed a chequered character. Every form of acute ascending paralysis which has borne the faintest resemblance to the case originally described by Landry has been called Landry's paralysis, whether the changes in the cord have been gross or insignificant, with the result that the literature has become hopelessly confused. E. W. Taylor says that "the tendency to consider cases of rapidly advancing paralysis, whether fatal or not, regardless of pathological findings, as Landry's paralysis, is very noticeable in much of the best recent work in America;" but this tendency is not confined to America, and may be found in any country of importance.

This unfortunate confusion can be traced to three principal sources. In the first place, many cases of acute poliomyelitis of the adult have been mistaken for Landry's paralysis during life, and the diagnosis has not been revised when examination of the spinal cord has revealed the inflammatory changes characteristic of the former disease. In the second place the occurrence of acute cases of polyneuritis of unknown toxic origin has led observers to believe that Landry's original case, in which the peripheral nerves were not examined, was only an example of this condition. In the third place, acute ascending myelitis presents features which have provoked the diagnosis of Landry's paralysis in certain instances.

The writer desires to avoid any attempt, which can never be successful, to reconcile the various views which have been, and are, held upon this subject, or to classify the different morbid processes which have at one time or another carried the name of Landry's paralysis. It does not matter, and it will certainly never be known, what was the actual disease from which the cases recorded by Landry suffered. It is sufficient for our purpose that he called attention to a class of case in which the symptoms were those of a rapidly spreading motor paralysis, without atrophy or electrical changes in the muscles, with slight sensory phenomena and no sphincter disturbances, and in which he was unable by the methods at his disposal to detect anatomical changes. Does this class of case still exist? The answer to this question is in the affirmative if allowance is made for improved histological technique, and if it is not forgotten that Landry would certainly have been able to detect the changes of acute poliomyelitis or any other acute inflammatory process in the spinal cord. It is clear, however, that "acute ascending paralysis" and "Landry's paralysis" cannot be regarded as synonymous terms; the latter is only one variety of the former.

It will be the object of this article to describe a morbid condition, which corresponds clinically to Landry's cases, in which the anatomical changes in the spinal cord would certainly have escaped notice with the methods of fifty years ago, and which presents no grounds for its inclusion in the group of the neuritides. There is nothing to be gained from imitating the course

adopted by some writers, who describe a central and a peripheral form of Landry's paralysis.

**Etiology and Bacteriology.**—There are but few points of etiological importance, if we exclude those cases which should properly be classed under the term myelitis. A recent history of some infectious disorder, such as gonorrhœa, influenza, or typhoid fever, is sometimes forthcoming, but not with sufficient constancy to merit attention. In the majority of instances the disease attacks healthy adults, and the period of life between twenty and forty appears to be the most susceptible. Men are more often affected than women, but it is difficult to say whether the preponderance is great or insignificant. Exposure to extremes of heat or cold may be a predisposing factor. Seasonal or climatic influences are not known to play any part, nor is there any evidence of the occurrence of epidemics. During epidemics of acute poliomyelitis the earlier severe cases have often been mistaken for examples of Landry's paralysis until the opportunity for examining the tissues has revealed the true condition.

The bacteriology is in an immature condition, and it is not possible to say whether the disease is specific or whether it may occur as a result of various forms of intoxication, bacterial or otherwise. The large majority of cases in which spinal lesions have been absent or insignificant have given a negative response to bacteriological investigation. On the other hand, a few have provided findings of some interest. Roger and Josué recorded a case of acute ascending paralysis in which they found toxic changes in the cells of the lumbar enlargement. Sections stained for bacteria did not demonstrate the presence of any organism, but cultivation from the heart blood produced a diplococcus resembling the pneumococcus. Injections of the coccus into a mouse did not prove fatal, but a rabbit submitted to the same experiment died at the end of twenty-one days with symptoms of paraplegia, and the organism was recovered from its blood.

The writer investigated a case of the disease which was in the National Hospital under the care of Sir William Gowers with the following results: (1) A micrococcus was isolated in pure culture from the blood of the patient after death. (2) An organism indistinguishable from that which was cultivated was found in large numbers in the loose vascular tissue forming the external layer of the spinal theca. (3) A subdural injection of the cultivated coccus into a rabbit produced after some days a rapidly spreading palsy. (4) The same organism was discovered in the theca of the rabbit, and isolated in pure culture from its blood. (5) In neither the patient nor the rabbit was the organism demonstrated in the spinal cord or the pia arachnoid, and in neither case were there inflammatory reactions in these tissues.

Macnamara and Bernstein isolated from the blood and cerebrospinal fluid of a non-fatal case a coccus which bore some resemblance to that just described, but they were unable to obtain any positive results from experimental inoculations.

**Pathology.**—To the naked eye the central and peripheral nervous systems present little that is remarkable, with the exception of some general hyperæmia of the cord. It is particularly to be noted that this organ is always firm and natural in consistence, provided that postmortem decomposition has not taken place. On cross-section the vascularity of the gray matter may be noticeable, and sometimes it is possible to detect small hemor-

rhages in its substance. The soft meninges present no evidence of exudation, serous or purulent.

The microscopic examination may, especially in cases which have run a rapidly fatal course, reveal practically no signs of disease beyond a few capillary hemorrhages, but if the Nissl and Marchi methods are employed a careful inspection will usually result in the discovery of the following changes:

1. **Cells.**—A smaller or larger number of the spinal cord cells, especially those of the anterior horns and of Clarke's column, present either early pericentral chromatolysis, or more or less complete loss of chromatin granules and excentration of nuclei. The most intense cell change is found in those parts which, judging from the clinical symptoms, were earliest affected; in the majority of cases the legs are the first limbs to fail, and the most marked cell changes are detected in the lumbosacral enlargement.

2. **Myelin.**—The myelin sheaths of the spinal cord nerve tracts, and to a less extent those of the peripheral nerves, often present a form of diffuse fatty change. In longitudinal sections the small droplets of fat are seen lying singly, or two or three together, along and between the nerve fibers, and do not fill the transverse area of the myelin sheaths. This appearance may be found in toxic states unassociated with paralysis, and does not therefore indicate necessarily any alteration of function on the part of the nerve fibers.

3. **Neuroglia and Vessels.**—There is no evidence of neuroglial proliferation, although some of the cells may appear to contain more protoplasm than normal. The vessels are engorged, but are free from changes in their walls or perivascular sheaths. Very rarely a slight excess of small round cells may be seen in the immediate neighborhood of one or two vessels.

The above are the only morbid changes found in most cases at the time of death. Occasionally, when the fatal termination has been postponed, the Marchi method will reveal the presence of true Wallerian degeneration in the spinal cord and peripheral nerves, probably secondary to the cell changes. Early fatty changes in skeletal muscles are often detected, but definite atrophy is not present until some weeks after the onset.

Outside the neuromuscular tissues, the most constant findings have been an enlarged spleen, enlarged mesenteric glands, and some evidence of pulmonary or pleural complications, generally of a secondary character.

**Symptoms.**—There is frequently a distinct prodromal stage lasting some hours, days, or even weeks, during which the patient may complain of various subjective sensations. Pain in the back is not common, although it does occur, but pricking, tingling, numbness, pins and needles, are usually described and referred to the peripheral parts of the limbs. A general sense of languor or fatigue and a feeling of heaviness in the legs and arms may also precede any definite loss of power.

The development of paralysis takes place somewhat rapidly and smoothly or by definite stages. In the latter case there may appear to be stationary periods followed by rapid involvement of fresh areas. Usually the legs are affected first, but occasionally the arms and rarely the cranial nerves present the earliest signs. The paralysis spreads from one part to another, following roughly the lines of spinal innervation. The lower intercostals, for instance, are weakened before the diaphragm is involved if the disease is following an ascending course, and the peripheral parts of the arms are affected before the muscles of the shoulder girdle. When a whole limb has become paretic, however, the proximal muscles are just as powerless, or sometimes even

more powerless, than those moving the hands or feet. The writer has on more than one occasion seen a patient lying perfectly helpless as regards any gross movement of the trunk or legs, yet able slowly to flex and extend his toes. In this respect the condition affords a most striking contrast to many cases of multiple neuritis, in which it is the general rule to find the peripheral muscles more paralyzed than the proximal.

From the beginning and throughout its course the paralysis is of the flaccid type, the muscles lacking their normal tone and presenting no opposition to passive movements of the limbs. No atrophy or diminution in electrical excitability can be detected in any muscle until two or three weeks after the onset, and even then the wasting is slight and the electrical alterations rarely exceed a lessened response to the faradic current.

If the motor paralysis continues to ascend, the movements of the head, and later those of the tongue, larynx, pharynx, and palate, may be interfered with, but it is unusual for a patient to survive sufficiently long after the respiratory musculature has been seriously embarrassed to develop marked cranial nerve symptoms. The loss of the accessory muscles of respiration, difficulty in swallowing and in articulation, are the principal phenomena brought about by the upward extension of the disease, and, of course, add greatly to the gravity of the sufferer's condition. The complete paralysis of all trunk as well as all limb muscles renders the patient particularly helpless.

In contrast to the severity of the motor symptoms everything else appears almost insignificant. The constitutional disturbance may be slight or even absent, and the temperature rarely exceeds 100° or 101°, unless it rises rapidly within a few hours of death. The intellect and memory remain clear so long as respiration is effectually carried on, and, in spite of dyspnœa, the patient is usually cheerful and sanguine concerning his condition. The paræsthesia associated with or preceding the onset may continue for some time, and, in addition, complaint is often made of cramp-like pains in the immobile limbs, which may be relieved by change of posture.

Tests for detecting loss of sensibility in the skin or deep tissues usually discover no change from the normal, unless it be a slight dulling in the cutaneous areas below the knees. On the other hand, a suggestion of muscle tenderness may be elicited by firm pressure. The sense of passive movement and of position remains unaffected. Every deep or superficial reflex disappears in the paralyzed parts, and is usually unobtainable as soon as any decided paresis has developed. The plantar response remains flexor in type so long as it can be elicited at all.

The rectal and vesical sphincters are unaffected, although, in consequence of the paralysis of the abdominal muscles, some difficulty may be experienced in emptying both the rectum and bladder. Incontinence does not occur, except as the result of a distended bladder, and the patient is aware of the natural calls to defecation and micturition. With ordinary care decubitus can be avoided, as there is no special tendency to the formation of trophic sores nor any deficiency in healing power on the part of the patient. Vasomotor disturbances are slight, if present at all; the skin over the paralyzed parts is moderately moist, at any rate in the early days. A tâche may sometimes be elicited.

The pulse may continue of fair strength and regular rhythm long after respiration has become difficult. With a fatal termination respiration gives

out for a considerable time before the circulation fails, and artificial respiration may often preserve life for many hours if persevered with. If life is prolonged the most serious complications are pulmonary and bronchial catarrhs, with which the patient, owing to his weakened respiratory muscles, is hardly fit to cope.

When neither the disease itself nor any complication proves fatal, the patient then enters upon the stage of recovery, and this presents some features of interest. The muscles often show a moderate degree of general wasting within three or four weeks of the onset, but the atrophy is diffuse, and not limited to particular groups. The response to faradism may be diminished, but is very rarely lost, and the galvanic current produces a normal or slightly sluggish contraction.

Recovery in power and in nutrition takes place slowly and equally over the whole of the musculature, and it is rare for any particular set of muscles to hang behind the others. Occasionally those of the limbs do not respond to treatment as quickly as those of the trunk. Contraction and deformities are rarely seen, except as the result of unskilled attention. Remissions and recurrences are not known to occur.

**Prognosis.**—Owing to the confusion which has existed, it is impossible to give statistics throwing any valuable light upon the mortality. In the opinion of the writer Landry's paralysis is a very fatal disease, and in this respect contrasts unfavorably with acute toxic polyneuritis and also with acute poliomyelitis, although the severe cases of the latter disease, which most resemble Landry's paralysis during life often terminate in death.

At the same time it is undoubtedly true that there are instances of true Landry's paralysis which do end favorably, and in such there is no reason to expect anything but an exceedingly good or even perfect recovery of function. As will be readily understood, the gravity of any particular case depends almost entirely on the condition of the respiratory musculature. So long as the diaphragm and the accessory respiratory muscles are intact, or at any rate capable of considerable work, there is, in the absence of complications, no serious cause for anxiety. When the diaphragm and intercostals are both very feeble, and the respiration is being carried on largely by the sternomastoids and the other neck muscles, the appearance of cyanosis, the look of distress, and the fogging of the patient's intellect are danger signals of the gravest import. The most favorable cases are those in which, although muscular weakness is very general, the loss of power is nowhere complete. Pulmonary and bronchial troubles, even after the acute stage has passed, must always be regarded as serious, but few cases survive the primary disease to die from complications.

**Diagnosis.**—The diagnosis is of importance not so much from the point of view of treatment (although it may not be many years before a correct diagnosis may be essential for this also) as for the purpose of giving an accurate prognosis while the disease is still in its acute stage.

When a patient is suddenly seized with an acute form of paralysis involving a large part of his musculature, two questions may well be asked by his friends: Is he going to live? If he lives, will he be permanently disabled? The medical attendant will ask himself, "Is this a case of acute ascending myelitis, of acute poliomyelitis, of acute toxic polyneuritis, or of Landry's paralysis?" Upon the answer to this question must the prognosis largely depend. For instance, it may be stated without hesitation that the diagnosis

of acute toxic polyneuritis renders the prognosis bright as regards both life and fair recovery of power, that of acute poliomyelitis, on the other hand, hopeful as regards life, but very grave as regards return of activity.

There should be no difficulty in distinguishing acute ascending myelitis from Landry's paralysis on account of the severe sensory loss and sphincter disturbance of the former condition, although the spread of motor palsy is often similar in the two diseases. The mistake of calling a case of acute and widespread poliomyelitis one of Landry's paralysis has been often made, but attention to the following general principles will assist toward a proper diagnosis.

1. *The constitutional symptoms* in the severe cases of acute poliomyelitis are more marked than those in Landry's paralysis, the range of temperature is higher, often reaching 102° to 104°, the malaise and anorexia more profound, vomiting and disturbance of the alimentary canal more frequent. In children convulsions are often associated with the former ailment. The older the patient the less likely is acute poliomyelitis to be present. Cases of acute toxic polyneuritis may, in many instances, run their course with no corresponding features.

2. *Sensory Phenomena.*—In Landry's paralysis there may be, and often is, complaint of pain in the back, of sensations of numbness and tingling in the extremities before and during the onset of paralysis. The only sensation complained of in the paralyzed limbs is that of discomfort or of cramps, arising, partly at any rate, from their immobility; they may be handled without giving rise to anything more than slight tenderness on deep muscular pressure. In acute poliomyelitis the pains complained of are more urgent, and affect not only the back, head, and neck, but frequently the limbs as well. Paræsthesia may be present, but the more prominent feature is the pain, sometimes very severe, elicited by passive movements of the affected extremities. In both diseases cutaneous sensibility is usually unimpaired. In acute toxic neuritis, numbness, pins and needles, and sharp pains are common in the extremities, and very marked tenderness of muscles, sometimes of nerves, is the rule. There may or may not be relative anæsthesia in the glove and stocking areas.

3. *Motor Phenomena.*—In both Landry's paralysis and acute poliomyelitis the paralysis is flaccid and may be general. The escape of a single muscle or of a group of muscles in a region where all the others are affected, or a marked asymmetry in the condition of corresponding muscles on the two sides of the body, are suggestive of acute poliomyelitis rather than of Landry's paralysis. Within a few days or a week of the onset in cases of acute poliomyelitis, if death has not occurred, it is usual to find rapid recovery in some parts and early atrophy and electrical changes in others. In Landry's paralysis recovery, if it takes place, is slow and evenly distributed, without marked atrophy.

In acute toxic polyneuritis the limbs are more affected than the trunk, and the peripheral parts of the limbs more than the proximal. Atrophy and electrical changes, perhaps only slight, quickly make their appearance, especially in the dorsiflexors of the ankles and extensors of the wrists. In the same disease, the most marked incidence is generally upon the diaphragm, in which case respiration becomes entirely costal. Of the cranial nerves, those supplying the facial muscles are often picked out and may be the only ones to suffer. Occasionally the palatal or ocular muscles are affected.

In Landry's paralysis the musculature is affected generally and evenly, the trunk and limbs presenting a degree of paresis which is more or less symmetrical and equal. In cases in which the paralysis first affects the lower extremities it is not uncommon to find these parts most paretic at the time of observation, but the spread of the disease will then be a progressive one in an upward direction, and the thoracic muscles may succumb before the diaphragm, or both may be equally weak. When the muscles innervated by the cranial nerves are involved, deglutition, phonation, articulation, and more rarely the movements of the face and jaws, may be impaired, but the fatal termination generally takes place before any of these actions are abolished. The condition may be progressive over two, three, or more weeks, and yet be unassociated with any definite local atrophy of muscles,

4. The *deep reflexes* are abolished in the affected parts in Landry's paralysis and toxic polyneuritis. The abdominal reflexes are often retained in acute toxic polyneuritis and nearly always absent in Landry's paralysis. The plantar reflexes are absent or flexor in type in both instances, and the sphincters are only temporarily, if at all, disturbed.

In addition, it is interesting to note that relapses and recurrences are not infrequent in toxic polyneuritis, although they are extremely rare or unknown in poliomyelitis and Landry's paralysis.

**Treatment.**—No measures are known to have any definite influence on the course of Landry's paralysis, and none are likely to be efficient until more is discovered about the etiology. The patient must be placed at complete rest, the head slightly raised, and changes of posture allowed for the sake of comfort. There is little liability to the formation of sores provided ordinary care and cleanliness is exercised. The bladder may require catheterization in the first day or two. A purge should be given, and if there is any fever a diaphoretic mixture is indicated. Every effort must be made to prevent the excitement of bronchial catarrh by exposure to cold, and the administration of atropine or belladonna, together with strychnine, may relieve the respiratory distress by diminishing bronchial secretion when the intercostal muscles and diaphragm become involved. Oxygen and artificial respiration may prolong life, although the necessity for their use is generally a token that a fatal termination is impending. Ergot has been tried, but has generally proved useless.

No active treatment of the paralyzed parts is indicated until the dangerous stage has passed and the disease has definitely taken a favorable course. The employment of massage and electricity may then tend to hasten the recovery of power. Passive movements should be carried out daily in order to prevent arthritic adhesions.

## ABSCESS OF THE SPINAL CORD.

Intramedullary collections of pus are very uncommon in the spinal cord as compared to the brain, and are not of great clinical importance. It is doubtful whether the fluid found in some of the early recorded cases was really pus or the result of necrotic disintegration of nerve tissue following spinal thrombosis.

**Etiology.**—Spinal abscess occurs most frequently as a secondary complication of distant septic processes, especially those involving the urogenital

system, the lungs, and the heart. Thus Nothnagel, Eisenlohr, Homen, and Chiari report cases which were secondary to a putrid bronchitis or bronchiectasis. Gonorrhœa or cystitis was probably responsible for those recorded by Ullmann, Schlesinger, and Sternberg. Two interesting cases observed by Cassirer arose in connection with a suppurating dermatitis and an infective endocarditis respectively. The latter, which presented multiple abscesses in the lumbar cord, supervened on a long-standing syringomyelia.

In some of the above cases, especially those of pulmonary origin, suppurative meningitis has been found in association with the central abscess, but it must not be too hastily assumed that the abscess was secondary to the meningitis. The frequency of suppurative meningitis and the rarity of spinal abscess are strongly opposed to this view. It is possible that the meningitis is sometimes a consequence of the abscess, and it is probable that in other instances the two conditions arise independently from a common cause. On the other hand small abscesses are very occasionally seen within the spinal cord in cases of suppurative meningitis under circumstances which point to the dependence of the former upon the latter.

In another class may be grouped those instances of spinal abscess which have been grafted upon a cord already damaged by fracture dislocation or caries or carcinoma of the vertebral column. Most of these must be regarded as secondary to a skin infection or to a purulent cystitis, the result of the initial lesion, the incidence of the metastatic process being determined by the evascularized and non-resistant condition of the spinal tissues at the seat of compression. The cases reported by Turner and Collier are good examples of this sequence of events. Finally, a small focal abscess is sometimes found in the midst of a non-purulent infective myelitis. De Gueldre and Sano have drawn attention to this in an acute myelitis of gonorrhœal origin.

The bacteriology of these abscesses has not yet matured, and it can only be stated that staphylococci, streptococci, and pneumococci have been found. In Chiari's case a streptothrix was associated with the pneumococci in the pus of the spinal abscess.

**Pathology.**—An abscess may be suspected when the cord presents a fluctuating swelling, especially if a purulent meningitis is apparent on the surface, but the organ must be opened in order to exclude a syringomyelic or hæmatomyelic cavity. The purulent collection is always more or less central, occupying a position at the base of the posterior horns or in the posterior commissure so much favored by extravasations of blood and by gliomatous cavities. Occasionally the white matter, usually that of the posterior columns, is encroached upon. The pus is of a greenish-white color, generally thick, and contains the remains of nervous tissue in the form of drops of myelin and fat. The abscess cavity may be focal or may extend through many segments of the cord. The walls in acute cases are friable; in more chronic cases a more definite lining may be formed by proliferated neuroglial tissue. Degenerative changes are found in relation to those nerve elements which have been involved in the process.

**Symptoms.**—Little is known of the symptomatology for several reasons. The evidence of abscess formation is often obscured by the signs of meningitis or myelitis, or the suppuration is frequently a terminal event, and occurs at a time when the patient's general condition does not allow of careful investigation. The process may run an afebrile course, or may be associated with rigors and an irregular pyrexia. The spinal symptoms are those of a

focal or diffuse myelitis, and will depend upon the site of the lesion. Flaccid motor paralysis, disturbance of sensibility and of the sphincters, loss or exaggeration of the tendon reflexes, form part of the clinical picture, but the patient rarely survives long enough for muscular atrophy to take place. Occasionally symptoms pointing to multiple purulent foci present themselves.

**Diagnosis.**—This is most likely to be arrived at in cases of pulmonary sepsis, although the symptoms may be ascribed to myelitis or meningitis, and the discovery of an intramedullary abscess on the postmortem table may come as a surprise. Metastatic abscesses in other organs are the chief indications of the spinal condition. In cases of compression paraplegia followed by cystitis, the upward spread of spinal symptoms may suggest the spread of a purulent focus, especially if it be accompanied by severe constitutional disturbance.

**Prognosis.**—The prognosis is invariably bad and a fatal termination not often long delayed.

**Treatment.**—Treatment is only prophylactic and must be directed against the primary cause of the disease, which is almost invariably grave enough to defy remedial measures.

## ACUTE POLIOMYELITIS.

**Synonyms.**—Acute anterior poliomyelitis; acute atrophic paralysis; infantile spinal paralysis; anterior cornual myelitis; tephromyelitis (τεφρος, ash-colored); regressive paralysis; essential paralysis of infants; *French,* poliomyélite antérieure aigue; *German,* Kinderlähmung spinale, essentielle Kinderlähmung; *Italian,* poliomielitide anteriore; *Latin,* poliomyelitis anterior acuta.

The term "acute poliomyelitis" is chosen on account of its simplicity and because the additional word "anterior" is neither necessary nor strictly in accord with modern knowledge of the pathology. The use of the word "infantile" should be discouraged, because it suggests a children's disease distinct from that which affects adults—a distinction which has no scientific basis. The use of the term "essential" dates from the time when the condition was supposed to have no characteristic anatomical lesions.

**Definition.**—An acute, probably specific, febrile illness affecting children and young adults, characterized anatomically by a confluent or discrete inflammation of the gray matter of the spinal cord and brain stem, with resulting destruction of the nerve cells and consequent degeneration of their axis-cylinder processes and atrophy of muscles, and clinically by a rapid atrophic paralysis of various skeletal muscles, usually those of the limbs, but occasionally also those of the head and trunk, a paralysis which reaches its maximum in a few hours and tends toward recovery in some parts and to the production of permanent disabilities and deformities in others.

**History.**—Heine, in 1840, was one of the first physicians to observe the disease and recognize that the spinal cord was the seat of the lesion. Three years later Colmer referred to an epidemic of "teething paralysis" identical with acute poliomyelitis in West Feliciana. Vogt and Duchenne, of Boulogne, described the disease in adults in 1858 and 1864 respectively. The first pathological researches were made by Cornil in 1863, and in 1865 Prevost and Vulpian examined a long-standing case in a woman aged seventy-eight

years, and found degeneration of the anterior horn cells, with chronic interstitial changes in the surrounding tissue. Their view that the disease was due to a primary cell change was upheld by Lockhart Clarke, Charcot and Joffroy, and other observers, but an entirely new light was thrown upon the pathology by Roger and Damaschino, who published three cases of young children dying at intervals of two months, six months, and thirteen months after the onset of paralysis. The morbid changes found by these authors, and confirmed by Charlewood Turner, Frederick Taylor, Drummond, Rissler, Putnam, and others, gave rise to the opinion that a primary inflammatory process was the basis of the disease. As in many other diseases, the accumulated work of the last half century has produced the opinion that an infective agent is responsible.

**Etiology.—Distribution.**—Acute poliomyelitis is a disease of wide if not universal distribution, as it is known to occur on every continent. Judging from medical literature, it would seem that North America and Scandinavia suffer far more severely than many southern countries.

**Sex.**—Among children the two sexes are about equally affected, but several observers, including Gowers, state that after adult age is reached, males are more frequently victims of the disease than females.

**Age.**—The large majority of cases occur in the first five years, after which the liability diminishes progressively up to the third and fourth decade. The occurrence of acute poliomyelitis is almost unknown after forty years of age. The question of an intra-uterine attack is not settled, but the writer has seen (with Dr. F. E. Batten) an instance in which that diagnosis appeared to be justified. Duchenne described a case in an infant twelve days old, but the frequency of the disease, even if the possibility of fatal cases occurring and being overlooked is taken into account, does not reach its maximum until the second or third year of life.

Taking the figures of Seeligmüller, Galbraith, Sinkler, Gowers, and Starr together, they show 121 in the first, 228 in the second, 158 in the third, 66 in the fourth, 46 in the fifth, and 16 in the sixth year of life. A similar age incidence is a prominent feature of the epidemics which have been recorded; 85 of Caverley's 119 cases were under six years of age, and in Nannestad's epidemic of 41 cases, 24 had not completed their fifth year.

**Season.**—Barlow and Sinkler were the first to point out the undoubted influence of season, and it is now generally agreed that in the hot summer months cases are much more common than at other times of the year. Out of 452 cases collected by Barlow, Sinkler, Gowers, and Starr, 369 had their onset in June, July, August, September, and October, and all the chief epidemics have been observed during the hot season. Both in America and England the months of July, August, and September claim the greatest prevalence, and in large cities some years seem to be more favorable to the development of the disease than others.

**Trauma.**—Trauma has so long been regarded as a possible cause for many diseases of the nervous system that it attracts, if it does not deserve, some attention. There is no evidence that trauma can play any important role in this disease, although parents are very frequently able to remember some fall or blow preceding, more or less remotely, the onset. It is possible that a central hemorrhage may be produced by a severe trauma, and that the resulting symptoms may be those of an atrophic local paralysis, but if such cases exist they are not examples of acute poliomyelitis. Changes of tem-

perature, prolonged bathing or paddling, may be predisposing causes, but the fact that the disease is more prevalent in hot weather should displace "chill" from the high pedestal it occupies in the lay mind as an exciting agent.

The relationship of the specific fevers of childhood to acute poliomyelitis has been the subject of some attention, but there is obviously a danger of its gaining undue importance. It is generally agreed that the disease sometimes follows, at varying intervals, measles, scarlet fever, diphtheria, and other fevers. There are, however, plenty of instances of one acute specific fever following quickly on another without any suggestion forthcoming that the later condition is a result of the earlier one. Moreover, Gowers is unquestionably right when he says, "The initial general disturbance is constantly mistaken for an independent general disease, and the opinion is often maintained after the discovery of the paralysis, which is then supposed to be of secondary origin."

Most observers will readily admit that the victims of acute poliomyelitis are, generally speaking, healthy if not robust children, on whom the paralysis falls with particularly cruel and unexpected suddenness. There is no evidence to support the view that teething even favors the development of the disease beyond the fact that the period of the first dentition is also the period of life when acute poliomyelitis is most rife.

**Pathology.**—The morbid changes which are present in the early days of the disease will be dealt with first. In this connection certain broad statements may be made: (1) The morbid anatomy of the epidemic cases is similar to that of the sporadic cases. (2) The changes in the adult are the same as those in the child. (3) In all cases the extent of the disease as shown by examination of the tissues is greater than could be inferred from the clinical symptoms. If, for instance, a patient dies from the effects of paralysis of the limbs and trunk muscles, it is safe to prophesy that anatomical evidence of the disease will be found also in the brain stem, and perhaps even in the cerebral and cerebellar hemispheres.

To the naked eye the meninges covering the central nervous system are hyperæmic. The substance of the cord and brain, and particularly the gray matter, appears vascular, and the latter may, in the more severe cases, be hemorrhagic and softened. A section of the spinal cord stained with logwood presents a striking appearance. The gray matter stands out unusually well on account of the numerous cells it contains, and all vessels, in both white and gray matter, are rendered prominent by their engorgement and by the dark staining of their perivascular sheaths.

The *histological anatomy* may be discussed under the following heads: (1) Meninges, (2) White Matter, (3) Gray Matter, (4) Central Canal.

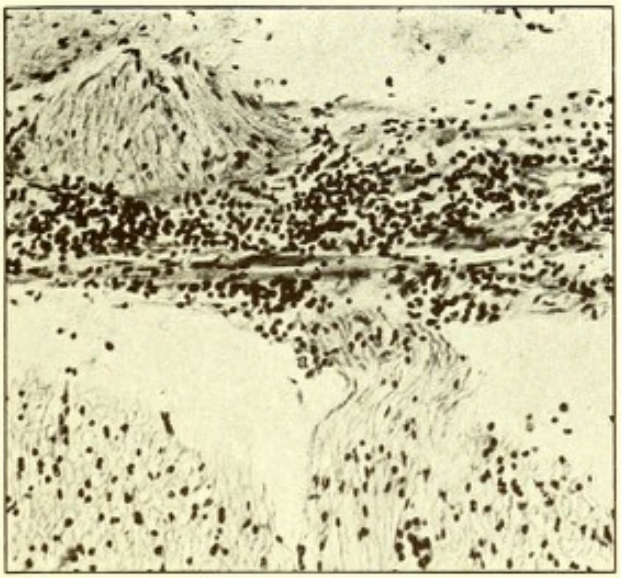
1. *Meninges.*—It is usual to find the vessels of the soft meninges full of blood, and in the more severe instances of the disease the membranes contain an excess of cells, chiefly lymphocytes, lying in the tissue meshes between the vessels. In many cases this cellular infiltration is only evident in the lumbosacral region, but in a few it extends to the higher parts of the cord and even to the basal meninges of the brain. It never gives rise to an appearance of pus to the naked eye, nor do the cells themselves resemble the leukocytes of that fluid. The amount of cellular infiltration is usually greater on the anterolateral than on the posterior surface of the cord. In the cases examined by the writer the majority of the cells in the pia arachnoid were of

# PLATE XIV

Section of the Spinal Cord in Acute Poliomyelitis.

# PLATE XV

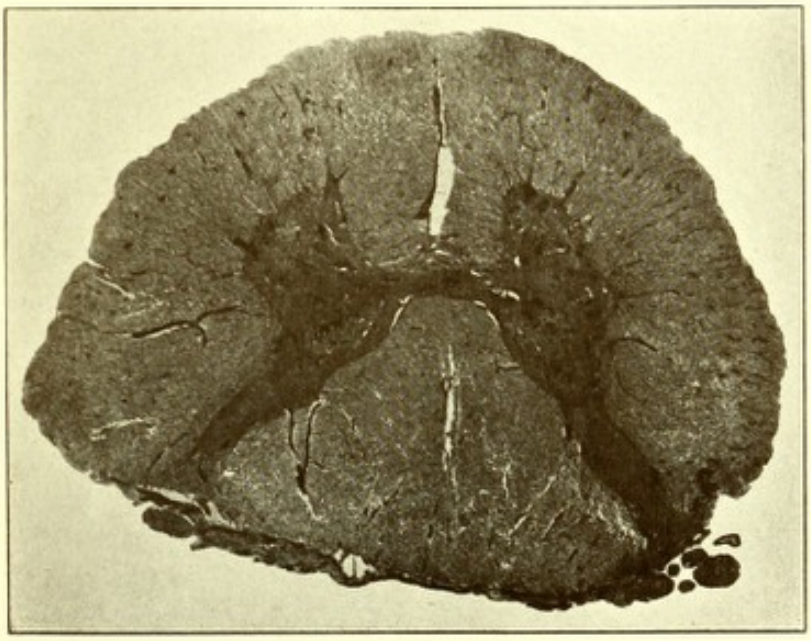
FIG. 1



Acute Poliomyelitis.

Anterior edge of cord in lumbar region. An anterior root bundle is seen passing through the soft meninges, which are infiltrated with round cells.

FIG. 2

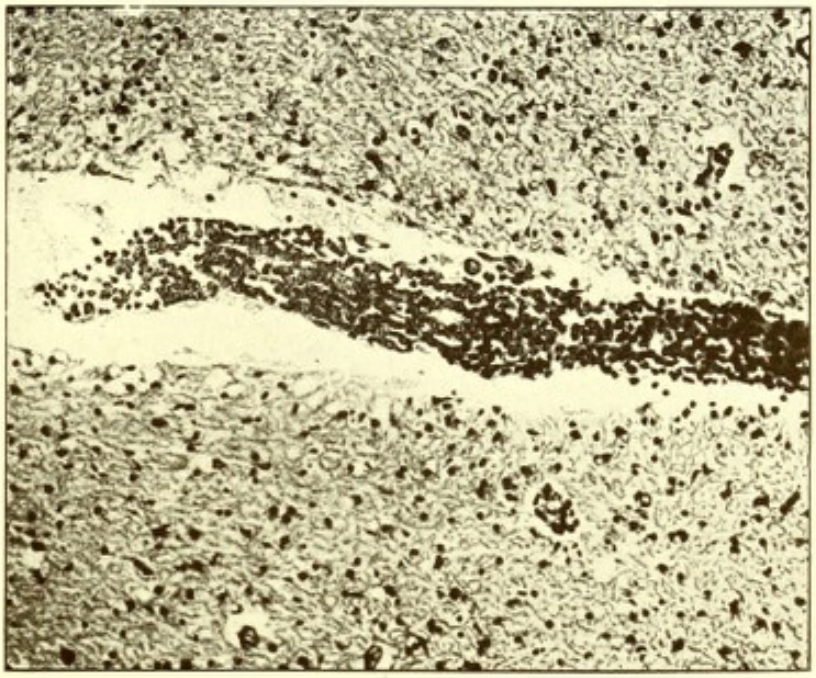


Acute Poliomyelitis.

From a case which died on the fourth day of illness. Twelfth dorsal segment. Note the swollen and prominent aspect of the gray matter and the dark outlines of the radial vessels in the white columns, due to intense perivascular round-cell infiltration.

# PLATE XVI

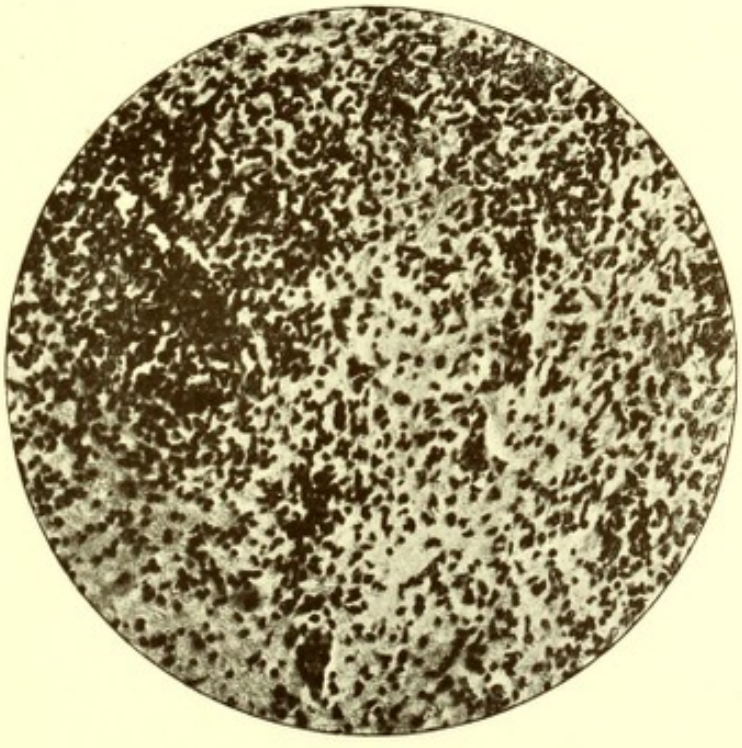
FIG. 1



Acute Poliomyelitis.

This photograph illustrates the fact that the vessels of the posterior arterial system are involved, as well as the branches of the anterior spinal artery. This vessel lies in the posterior median fissure.

FIG. 2



Acute Poliomyelitis.

Dense cellular exudation and proliferation in the anterior gray matter of the lumbar cord. All the ganglion cells have disappeared.

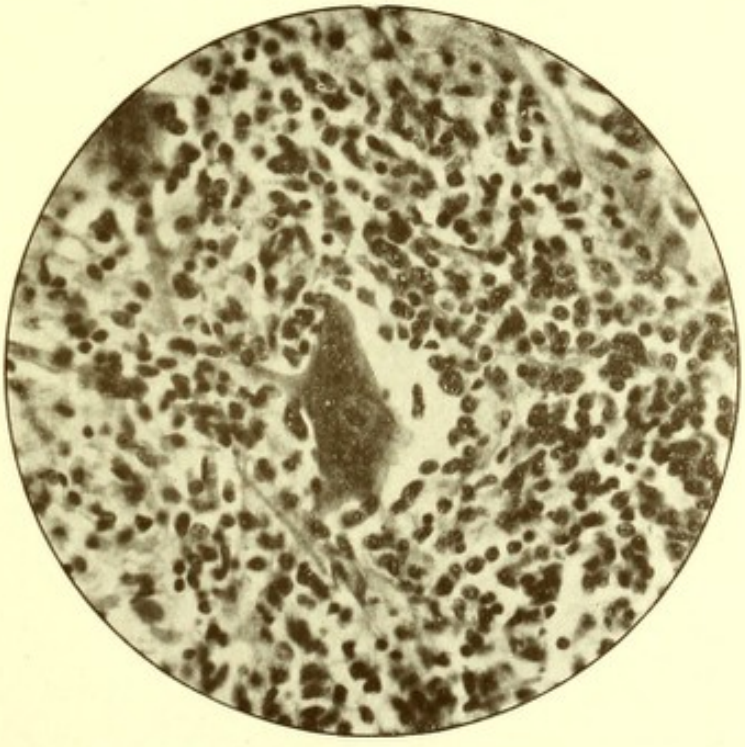
# PLATE XVII

FIG. 1

Acute Poliomyelitis.

Photograph to show the swollen ghost-like cells of Clarke's column and the surrounding cellular infiltration of the tissues.

FIG. 2



Acute Poliomyelitis.

An anterior cornual cell in fair preservation, although surrounded on all sides by inflammatory cells.

the mononuclear type; a smaller number showed the various intermediate stages between the lymphocytic and the plasma-cell characters, and only a small percentage of other cells, among which were a few polymorphonuclear leukocytes, was observed. That this meningeal infiltration is not a feature of fatal cases only is proved by the fact that a lymphocytosis of the cerebrospinal fluid has been demonstrated in non-fatal cases. On the other hand, it is probably an indication of considerable severity in the morbid process.

2. *White Matter.*—The columns of nerve fibers do not suffer much, although some scattered degeneration takes place, chiefly in the anterolateral columns in the neighborhood of the gray matter. The neuroglial cells undergo some change, the smaller ones swelling and the Deiter's cells becoming round and large, with two or more nuclei, and losing their processes. The most striking feature is the intense cellular infiltration of the walls of the vessels, which radiate from the surface inward. The cells lie really in the lymphatic space existing between the media and adventitia and in the meshes of the latter coat. Only very occasionally are foci of inflammatory cells seen in the white matter away from the bloodvessels and these are always of small size.

3. *Gray Matter.*—The vessels of the gray matter resemble those of the white matter in the fact that their outer walls are packed with cells chiefly of the mononuclear type, and this applies in the spinal cord, not only to those which are derived from the anterior spinal arterial system, but also to those which originate from the posterior root vessels, and applies equally to arteries, veins, and capillaries. In virulent cases small hemorrhages from the capillaries are found; in other cases, which have survived some days or weeks, evidence of blood stasis and thrombosis is forthcoming in the form of tissue necrosis and softening. In addition, the gray matter is the seat, particularly where it is most generously supplied with blood, as in the anterior horns of the cord, of extensive cellular infiltration of varying density.

The nerve cells of the anterior horns, of Clarke's column, and of the posterior horns all suffer, in varying degree. Owing to the greater vascularity of the anterior horns, the cells of that region are generally most affected, but marked changes are frequently found in the other parts. Speaking generally, those ganglion cells undergo the greatest change which are most closely embedded in the masses of inflammatory cells, but this rule has its exceptions, and occasionally healthy looking nerve cells are met with in areas of intense inflammation. On the other hand, it is very unusual to encounter definite degeneration of ganglion cells which are far removed from the foci of disease. Evidence of neuronophagia appears in the presence of neuroglial cells within the pericellular spaces or actually invading the ganglion cell substance. The myelinated fibers of the gray matter, as well as the cell processes, are partially or completely lost in the inflammatory areas.

4. *The Central Canal.*—In many cases it is impossible to detect changes in the contents or walls of the central canal, but the inflammatory areas of the gray matter may extend to the ependymal cells.

The above facts may be summarized thus: (1) Congestion of all bloodvessels, both in white and gray matter, and packing of their adventitial sheaths and lymph channels with cells. (2) An intense proliferation or exudation of cells, concerning the origin of many of which there is a difference of opinion, limited almost entirely to those parts of the central nervous system most

freely and generously supplied with blood, namely, the neighborhood of the rich capillary network of the gray matter of the cord and to the well vascularized regions of the brain stem and brain. (3) Round-cell infiltration of the soft meninges, more marked in the lower parts of the cord and on its anterior aspect, but extending in some cases to the intracranial membranes. (4) Retrograde changes in those nerve cells and nerve fibers which are intimately involved in the areas of inflammation.

The examination of cases which have died many months or years after the disease has run its course shows that the results of inflammation here are similar to those in other tissues. In some parts resolution may have taken place with the loss of a few or no ganglion cells; in other parts fibrosis has led to deformities of the gray matter and to complete loss of its cellular elements; in other cases the necrosis may have been sufficient to produce actual cavities in the cord. The Weigert-Pal method of staining may reveal some evidence of scattered sclerosis in the white columns, particularly in the neighborhood of the gray matter.

*Nerves.*—The efferent fibers of the anterior roots and of the spinal and cranial nerves of which the ganglion cells have been destroyed undergo the usual secondary degeneration, but there is no evidence to show that the morbid process ever involves directly the peripheral nerves.

*Muscles.*—The changes which take place in muscles paralyzed by an attack of acute poliomyelitis resemble in almost every respect, as might be expected, those consecutive to an experimental neurectomy. They constitute a simple atrophy. The connective tissue seems to be proportionately increased with the shrinkage of the muscle fibers. At a later date examination may reveal changes which are not seen after simple nerve section, but which are not always present or are only present in varying degrees in individual muscles. Lipomatosis is the most striking of these, the fat in some cases replacing whole bundles of fibers, and even whole muscles. The presence of hypertrophic poorly striated fibers is not uncommon, and these may perhaps denote incomplete degeneration of central nerve cells. In other instances a whole muscle may only be represented by a fibrous band.

*Other Organs and Tissues.*—The general examination of the body has revealed no constant lesion outside the central nervous system. An enlarged soft spleen and catarrh of the respiratory or alimentary tracts have occasionally, but not frequently, been found, and appear to have no causal or etiological significance.

**Bacteriology.**—The steadily increasing belief that the disease is due to a microbial infection has led numerous observers to examine the spinal fluid during life and the tissues after death for the presence of bacteria. So far the results have been unsatisfactory and, taking into consideration the possibility of contamination and accidental infections, chiefly negative. Landsteiner and Popper, in 1909, successfully inoculated two monkeys with the spinal cord from fatal cases of poliomyelitis. Flexner and Lewis (Rockefeller Institute, New York) have infected monkeys and produced a disease similar to that in the human. They were able to transmit the disease through a series of monkeys by way of the brain, peritoneal cavity and circulation. They were unable to discover bacteria by any method.

**Pathogenesis.**—The anatomical evidence of an acute inflammation affecting the most vascular parts of the spinal cord, taken in conjunction with the clinical evidence of an acute febrile disorder occurring sporadically

and in epidemics, and resembling in its onset and course the acute specific fevers, justifies the view that acute poliomyelitis is an infective disorder. The causative agent is still unknown, and we are ignorant as to whether it produces its results by its direct presence in the nervous tissues or indirectly by means of a toxin. It is clear, however, from the behavior of the ganglion cells that these are not specifically susceptible to the influence of the virus, but suffer rather from the effects of inflammatory changes in surrounding tissues. If the poison has any selective action, it is probable that this is vented upon the neuroglia, seeing that the gray matter is particularly rich in this tissue and that the peripheral nerves, devoid of neuroglia, remain directly unaffected during the disease.

The writer feels that acute poliomyelitis has at least as strong claims as some of the acute specific fevers to be regarded as one of them, and anticipates its early inclusion in their ranks. It has the constitutional disturbances, the age incidence, the seasonal exacerbations, the sporadic and epidemic types, the immunity from second attacks, associated with the obscurity of bacterial origin, which are shared by measles, scarlatina, and varicella; on the other hand it presents the severe inflammatory lesion of a particular tissue which is met with in typhoid fever, diphtheria, and variola. It resembles the latter particularly in its permanent cicatrices and their deforming results.

*The relation of acute poliomyelitis to certain forms of acute encephalitis* has attracted considerable notice since Strümpell and later Marie drew attention to the probable identity of the pathological process in the two diseases. That their morbid anatomy is strikingly similar has been shown by several observers, but still more interesting are the recorded occurrences of the two diseases in the same individual or in two or more members of the same family at the same time. Such cases could easily be multiplied, but we will only refer to Pasteur's family epidemic. Within ten days seven children were attacked by an acute febrile disorder, and three developed paralytic symptoms. Two of these were examples of acute poliomyelitis, and another became hemiplegic on the right side as the result of encephalitis. Transitory nervous phenomena were exhibited by two of the remaining members of the family. Finally, in many of the larger epidemics, cases of encephalitis have been found among the cases of poliomyelitis, and paralysis of the cranial nerves has occurred either with or without spinal symptoms in the same individuals.

There is abundant evidence, therefore, to show that encephalitis may be, and often is, excited by the causal agent of poliomyelitis, although it is not yet clear what determines the incidence of the disease upon the brain or spinal cord.

**Symptoms.—Initial Stage.**—In the autumn a child previously healthy is noticed to be out of sorts, is restless, refuses food, and is found to have a temperature of 100° to 103°. He may vomit or may have one or more convulsions. In the course of one or two days it is noticed that he has lost power in both legs and can no longer stand or run about. The arms are also paralyzed during the next few hours. With the onset of the paralysis the child resents handling, crying when any attempt is made to move him, and, even when at rest, appearing in some distress. Another day or two and all constitutional disturbance has passed off, the arms are already beginning to recover power, and the movements of one leg are certainly less com-

pletely lost than those of the other. Evidence of pain is only elicited on passive movement of the lower limbs. At the end of ten days or a fortnight there is much less pain even on handling, the arms have recovered power, although they are flabbier than formerly, the better leg has regained movements at the hip and knee, dorsiflexion of the ankle being still absent, but the other leg hangs limp and lifeless. Examination at this stage reveals the presence of some wasting and greater flaccidity in the paralyzed muscles, and the helpless leg is colder than its fellow. A stronger faradic current than usual is required to produce any contraction in the affected parts, and the reaction to galvanism is less brisk than in the arms. The patellar and Achilles jerks are absent on the one side and can only just be elicited on the other. If cutaneous sensibility is tested, it will be found perfect everywhere. Some retention of urine, which was present for the first day or two, has now passed off, but the patient, if in the first or second year of life, does not learn to hold his urine for some time to come. Lumbar puncture finds the spinal fluid at high tension and possibly containing an excess of lymphocytes.

This is the common history of the first two weeks in an attack of medium severity. Variations, however, are frequent and depend largely on the extent and severity of the disease. In milder cases the constitutional disturbance may be absent or so slight as to escape notice, and is often forgotten in after days, the mother declaring that the child was perfectly well all through. Only one lower limb may be paralyzed at the outset, and in the course of a few days the disability may be limited to one or two muscles below the knee. In more virulent forms all four limbs and the trunk muscles are rapidly and completely paralyzed, with the result that the child is dependent on its diaphragm and accessory muscles for respiration. If this condition does not hastily mend, bronchopneumonia or bronchitis is more than likely to bring about an early fatal termination.

A still more fulminant type sometimes occurs—and probably occurs at times without the true diagnosis being made—in which the paralysis quickly spreads upward, and, by involvement of all respiratory muscles and perhaps those of deglutition, ends the patient's life in the course of thirty-six to forty-eight hours. Head retraction is not uncommon in such cases, and leads to the suspicion of meningitis. The age has no material influence on the clinical picture of this stage, with the exception that convulsions are less common in older subjects. On the other hand, an adult is better able to describe the sensory side and on questioning will complain of pain, often dull or gnawing in character, in the back and of sharp neuralgic pains, increased by movement and often of several days' duration, in the affected limbs. However variable the different cases may be in the rapidity of onset, in the progress of the paralysis, and in associated disorders, the disease is characterized by the fact that the initial paralysis is invariably in excess of what remains at the end of a few days or weeks.

With regard to the reflexes it is the rule that all muscles which show any degree of paralysis lose their tendon jerks at first. If atrophy does not supervene, the jerks return, but if wasting and reaction of degeneration follow the paralysis, the jerks are lost for many months if not permanently. The superficial reflexes of the trunk are preserved in most cases unless the abdominal muscles are paralyzed. The plantar reflex is lost when the muscles moving the toes are paralyzed, but if these are intact, stimulation of the sole will produce generally a flexor, but in some instances an extensor

response. When the latter occurs it signifies some extension of the disease into the pyramidal tracts, an occurrence which has already been alluded to in the description of the morbid anatomy. An extensor response may be associated with an increased patellar jerk, or even with ankle clonus in rare cases, the extensors of the knee and the flexors and extensors of the ankle having escaped paresis. In a case of poliomyelitis in a young man the writer obtained a well-marked extensor response in both feet, and a brisk knee-jerk on the left side, the right being absent, on examination between two and three weeks after the onset, and he has observed a similar condition on several occasions in children.

**Stage of Repair.**—This cannot be said to have very well-defined limits, its length varying in different cases, but it begins roughly at the end of the second or third week and lasts until the end of a year or eighteen months after the onset. It is during this time that the full extent of the permanent damage will be ascertained, that some muscles gradually shrink and become useless, that others after a period of paralysis and atrophy slowly become restored to a condition approaching, if not equal to, their former competence, and that the effect of the disease on the growth and nutrition of the limbs becomes more and more apparent.

Examination now reveals unmistakable wasting of the affected muscles, resulting in almost complete loss of soft tissue in certain regions, or in other cases, chiefly young children, in the replacement of the muscles by fatty deposits. The skin over these parts is generally colder, more purple in hue, and either more dry or more clammily moist than elsewhere. Lax muscles lead to alterations in joints, and with the occurrence of permanent shortening in other muscles, combine to produce serious deformities. The chief factor in the production of the shortening is the natural unopposed action or tone of a healthy muscle, its antagonist being paralyzed. This physiological contraction may be assisted by an increase in the connective tissue of the muscle due to the degeneration of some of its fibers when it has not completely escaped. Bones to which many paralyzed muscles are attached cease to grow in children, although in rare cases actual elongation of a bone, due, probably, to traction on the epiphyses, may take place. As a general rule, shortening is present and trophic changes in the bone may be demonstrated by means of a skiagram.

The electrical reactions during this stage are of importance, especially with regard to prognosis. Those muscles which, although paralyzed, never completely lose their faradic excitability may be confidently expected to recover their function. Muscles which lose their faradic excitability, and in which the galvanic response is slow and more easily produced by the anodic than the kathodic closure current, have undergone the reaction of degeneration. Their fate hangs in the balance until either (1) the galvanic response becomes quicker and more natural, which is of good omen and precedes the return of voluntary contraction and faradic excitability, or (2) the galvanic response becomes gradually more difficult to obtain and finally disappears, denoting a complete and permanent destruction. Some reference may be made to the incidence of permanent paralysis. The statistics of Duchenne and Seeligmüller show that in about 55 per cent. of cases one leg, in about 17 per cent. one arm or both legs, in about 5 per cent. all four limbs, and in 2 per cent. both arms, are affected. The trunk muscles, innervated as they are by the less vascular dorsal segments of the cord, are

not so frequently paralyzed. On the other hand, the muscles of respiration are commonly involved in the most virulent and fatal cases.

Taking the limbs separately, it is noticeable that the dorsiflexors of the ankle, the extensors of the knee, and the extensors of the hip are more often affected than their respective antagonists, and that the muscles moving the shoulder and elbow are more prone to disablement than the intrinsic muscles of the forearm and hand. Generally speaking, there is no tendency to symmetry in the results of the disease on the limbs of the two sides; on the contrary, asymmetry may almost be considered a characteristic feature.

**Permanent Results.**—After eighteen months have passed nature may be considered to have brought about any repair of which she is capable, and the remaining effects must be regarded as permanent, unless artificial means of modifying them can be employed. The clinical picture is now one of deformities, the most common of which are talipes equino varus and valgus, pes calcaneus, contracture of the knee in the flexed position, scoliosis, and lordosis. The growth of healthy parts emphasizes the small size and shortening of the paralyzed member or members.

**Diagnosis.**—Any difficulty is only rarely a serious one in the acute stage and in young infants. A febrile disturbance associated with a painful condition of the limbs, especially evoked by handling, and with a pseudo-paralysis, is produced by rickets, scurvy rickets, syphilitic epiphysitis, osteomyelitis, and rheumatic fever, and must be differentiated from acute poliomyelitis by general examination, by inspection of the affected parts, by attention to the reflexes, and, if necessary, which is not often, by electrical stimulation of the muscles. In very acute cases poliomyelitis may simulate meningitis, owing to the rigidity of the back and neck muscles, the vomiting, and the convulsions, but the simulation is only a temporary one, and the early appearance of flaccid palsy with the disappearance of the other symptoms puts an end to the difficulty. Probably some acute cases of this kind die in the first few hours, and then the diagnosis can only be made post mortem, although the examination of the spinal fluid may afford a clue.

From *multiple peripheral neuritis* the diagnosis is chiefly based on the gradual onset, the symmetrical incidence of the paralysis, and the presence of more or less sensory disturbance in this disease. A cerebral monoplegia, especially of the leg, may simulate an old lumbar poliomyelitis at first sight, owing to the shortening and diminutive size of the limb with some circulatory disturbance and perhaps a tendency to talipes equinovarus. However, in spite of the general wasting, the limb preserves its natural contours, the reflexes are those associated with spastic conditions, and the muscles respond to mechanical and electrical stimulation. Reference has been made to certain acute spinal atrophies, which come on very suddenly, without constitutional disturbance, limited from the first to a single region. These are probably examples of a vascular thrombosis or hemorrhage, although our pathological knowledge concerning them is very small, and, except for the history of their onset, cannot always be differentiated from acute poliomyelitis when once established. A rare disease, amyotonia congenita, may be mistaken for the results of poliomyelitis, but close observation will show that true paralysis is not present, although the tendon reflexes are not obtained and the muscles may be unresponsive to faradism.

Finally, the comparative rarity of poliomyelitis in the adult and the occasional ascending character of its symptoms has not infrequently led observers

to make a diagnosis of Landry's paralysis. Unfortunately this has not always been corrected when the examination of the tissues has revealed the presence of gross inflammatory changes in the spinal cord.

**Prognosis.**—In the acute stage the prognosis as regards life depends upon the extent of the disease with particular relation to the respiratory musculature. In a case published by Green, Wilson, and Rothrock, artificial respiration was carried on for forty-one days before sudden death brought a "merciful solution to a hopeless problem." If the patient survives the first few days without grave respiratory difficulty, the prognosis may be regarded as favorable in the absence of complications such as bronchopneumonia or intestinal troubles. In the second stage the probable recovery of power must be estimated from the electrical reactions.

**Treatment.**—We are unacquainted with any method of combating the acute attack, and until further investigation discovers the causative agent must be content to treat the initial stage similarly to that of any other acute infective disease. Confinement to bed, a diaphoretic, and a purge are indicated, and pain may be relieved by the exhibition of salicylates, phenacetin, or, in severe cases when rest is interfered with, of an opiate. While the fever lasts the diet should be light and easily digestible. Attention should be paid to the bladder and a catheter used if there is any evidence of retention. Great care must be employed to avoid any risk of bronchial or pulmonary complications. In the event of the respiratory musculature becoming embarrassed, inhalations of oxygen are indicated, and artificial respiration may be carried out in the hope that the involvement of these muscles may be only temporary.

The belief that any benefit can be derived from the application of heat or of ice to the spine, or from wet cupping, has too little substantial basis to justify its recommendation. If headache is severe, it is possible that the withdrawal of cerebrospinal fluid, which is often at high tension, may relieve it.

After the first two or three weeks there are several important points to be remembered:

1. Passive movements of paralyzed parts should be performed two or three times daily, and changes of position should be enforced, for the purpose of preventing contractures.

2. The patient should be encouraged to attempt movements of paretic parts every day. The adult will do this according to instruction, but ingenuity must be exercised to induce an infant to make the effort. The attachment of a bell or rattle to the paralyzed limb is sometimes effectual.

3. All paralyzed limbs should be warmly clad with woollen garments and massage should be practised daily. In cold weather hot bottles may be employed to keep up the temperature of the lower extremities.

4. Electrical treatment may be employed, although it is not as important as the measures already detailed. The object is to make the paralyzed muscles contract and to retain their power of contraction until their innervation is restored. The galvanic current must therefore be employed, the kathodic being used as the mobile electrode. In young children it is well to begin with an inappreciable current, which can be gradually increased in strength from day to day until a definite contraction is elicited. When all the benefit possible has been obtained from these measures, and it is justifiable to expect continued even if very slight improvement up to at least

eighteen months after the onset, the question of artificial aid in the form of instruments, tenotomies, or other surgical procedures must be raised. Instrumental aid is often effectual in allowing a child to walk by fixing one or more joints, but care should be taken that the apparatus is as light as possible, and that any shortening in the limb is compensated by additional thickness in the sole of the boot. Frequent alterations are necessary with the growth of the child. Tenotomies are useful for the purpose of putting a limb or part of a limb into an advantageous position.

The grafting of portions of a healthy muscle into the tendon of a paralyzed muscle has been advocated, but the writer has seen no case in which the object aimed at had been really attained. Nerve grafting deserves a more conscientious trial, and may give good results in suitable cases. Unfortunately the results claimed for this method do not in some cases bear critical analysis.

In certain instances the patient will be better served by an amputation stump than by a badly deformed foot, which is liable to become bruised and sore.

The question of drugs can be shortly dealt with. No drug has any specific effect either on the disease itself, or on the regeneration of neuromuscular tissue. The general health of the patient may in some instances require the exhibition of iron, arsenic, or other tonics.

## THE BLOOD SUPPLY OF THE SPINAL CORD AND ITS PATHOLOGY.

Before considering the question of circulatory disturbances a short description of the blood supply is an important necessity. This is derived from the vertebral, intercostal, lumbar, and sacral arteries, and the arrangement of the contributing vessels can be best discussed if an arbitrary division is made between an anterior and a posterior system.

The *anterior system* consists chiefly of the anterior spinal artery, which originates at the upper end of the spinal cord from the union of branches of each vertebral artery, and which lies on the anterior surface of the cord at the mouth of the anterior median fissure throughout its whole length. This vessel is reinforced at each segment by branches from the vessels, which reach the cord with every anterior spinal root, but, with two or three exceptions, the latter are of very small caliber, and contribute poorly to the spinal supply. The exceptions named are found at slightly varying levels in different individuals, but the most important are those which accompany the sixth or seventh cervical and the eleventh or twelfth dorsal anterior roots. Another vessel of fair size is frequently found with one of the roots of the cauda equina. As a result of this arrangement the anterior spinal artery varies in its size at different levels, having a smaller caliber in the mid-dorsal region than in the neighborhood of the cervical and lumbosacral enlargements. From the anterior spinal artery pass inward at intervals, along the anterior median fissure, a number of vessels which penetrate the anterior commissure and divide into several branches within the gray matter of the cord. Most of these branches run horizontally, supplying the anterior horns, and, at some levels, the region of Clarke's column, but a few in the immediate vicinity of the central canal take a longitudinal course, anastomosing with corresponding arteries from other levels. The horizontal

branches break up into terminal arterioles, between which there is little or no capillary anastomosis.

On the surface the anterior spinal artery sends branches right and left, which, together with some offshoots from the root arteries, form a network of vessels in the pia mater on the lateral aspects of the cord. This network is connected by anastomosis with similar vessels from the posterior system, and the two combined go to complete the "vasocorona" encircling the spinal cord.

The *posterior system* contains no vessel equal in size to the anterior spinal artery, but it is made up in a similar way from branches of the vertebral arteries, and reinforced segmentally by posterior root vessels of varying importance. The chief longitudinal components of this system are the two posterior spinal arteries, directly continuous with the vertebral branches, which lie on either side of the cord, just lateral to the entering posterior roots. These vessels are not so well defined as the anterior artery; they form, rather, a part of the vasocorona, sending numerous branches medialward and laterally to combine with vessels from the opposite side and from the anterior system.

The *vasocorona*, thus formed, provides radiating vessels, which penetrate the surface of the cord at different spots. The more important are those which enter along the posterior median septum, along the posterior horns, between the columns of Goll and Burdach, and certain others supplying the white matter of the lateral columns. The area vascularized by these centripetal radiating vessels comprises the whole of the white matter, the posterior horns, and, in common with the anterior system, some of the outlying parts of the anterior and lateral gray matter. The capillary network in the gray matter, particularly that of the anterior horns, of the central region and of the posterior commissure, is of a far richer character than that which obtains in the white columns. On the other hand, it must be remembered that capillary anastomosis between the various branches is insignificant.

Some of these data have considerable pathological importance. The small size of the anterior spinal artery in the middle third of the dorsal region and the absence of any contributory root vessels of good caliber at the same level renders that part of the spinal cord the poorest in vascular supply. For this reason it is not surprising that thrombotic lesions are more frequent in this situation than in any other. Moreover, its comparatively poor vascularity helps to explain the chief incidence of these thrombotic lesions upon the white matter. On the other hand, the regions in which the gray matter is most abundant, in the cervical and lumbar enlargements, possess a correspondingly rich vascular supply, and these are the common sites of toxic and infective lesions of hæmatogenous origin.

Experimental pathology has demonstrated the relative insufficiency of the spinal blood supply, and also the relative susceptibility of certain nervous elements to the effects of partial or temporary ischæmia.

If the abdominal aorta is compressed below the origin of the renal arteries, and the contribution of the lumbar arteries thus diverted from the cord, paralysis of the hinder limbs rapidly develops, and examination of the spinal tissues, after varying periods of compression, shows that the ganglion cells of the anterior horns are the first to exhibit signs of degeneration. This occurs in spite of the fact that blood can reach the parts through the vertebral and intercostal branches, and demonstrates the relative insufficiency or

instability of the vascular supply, as well as the delicacy of the highest nerve elements in the face of vascular disturbances.

**Anæmia.**—There is no proof that a *general* anæmia is capable of producing organic lesions of the human spinal cord, nor does our knowledge permit us to attribute any spinal symptoms to that source. On the other hand experimental results justify the supposition that the most specialized nervous elements react quickly to alterations in the blood state, and conduce to the belief that disorders of nervous function may result from anæmia of the spinal cord, without the presence of signs of organic change. It may readily be granted that nerve cells, when exposed to poverty in the quantity or quality of their blood supply, may exhibit evidences of fatigue more rapidly than they would do under better conditions.

In certain varieties of general anæmia, particularly in pernicious anæmia, it is not uncommon to find changes in the spinal cord, but it is highly probable that these are not the direct result of the altered blood state, and that they should properly be referred to the effect of some toxic agent, to which the anæmia is also secondary.

The paraplegia which results from excessive loss of blood has no known anatomical basis, and it is difficult, therefore, to say which part of the nervous system is chiefly at fault. Speaking generally, it may be quoted as an example of the vulnerability of the nervous tissues to anæmia, and the question of its cortical, spinal, or peripheral origin left for further investigation.

When the subject of *local* anæmia is brought under consideration, there are more facts to guide us in forming conclusions. Local anæmia may be partial or complete, and may have its origin in (1) thrombosis due to a blood state or to disease of vessels, (2) embolism, (3) strangulation of vessels by pressure of tumors, thickened meninges, or displaced bone, and (4) vasomotor constriction or spasm or dilatation.

1. Little is known about thrombosis as the result of an abnormal blood state in the spinal cord, unless some of the changes in toxæmic conditions can be accounted for in this way. Arteriosclerosis of the spinal vessels is by no means uncommon, but for some reason which is not obvious spinal thrombosis is far less common than cerebral thrombosis. Probably some of the cases which are usually termed senile paraplegia or senile myelitis have a pathological basis founded on softening of arteriosclerotic origin. If this is so, their symptomatology is more or less identical with that of cases of syphilitic myelitis. Arteritis due to syphilis, and more rarely to tubercle, is by far the most potent and frequent cause of spinal thrombosis.

2. Embolism in the spinal cord is a rare event, but cases have been described by von Leyden, Weiss, and Gowers which bear this interpretation. In all these instances the patients were suffering from mitral disease, and in two of them the postmortem examination showed the presence of spinal softenings. Experimental spinal embolism has been successfully produced by various investigators by means of foreign particles introduced into the lumbar arteries. Hoche showed that foci of simple softening were generally found in the central gray matter, owing to the larger caliber of the anterior spinal artery. On the other hand, the use of irritating vegetable seeds resulted not only in simple necrosis, but in an inflammatory reaction. The animals operated on became rapidly paraplegic.

3. Spinal vessels are frequently narrowed or occluded by the pressure of tumors, thickened meninges, or displaced bone, with the result that areas of

tissue are evascularized and sooner or later become necrotic. The term "compression myelitis" was formerly used to denote the condition.

4. Vasomotor constriction or spasm of the spinal arteries is one of those phenomena the occurrence of which may be regarded as possible, but the proof of which is not yet forthcoming. Paroxysmal paraplegia or monoplegia in a person who presents no symptoms of spinal disease between the paroxysms may be interpreted on the hypothesis of vasomotor spasm, and a similar construction placed upon cases of intermittent paræsthesia of the extremities, but these are matters of speculation rather than of demonstrated fact. Vasodilatation can certainly be instrumental in slowing the blood stream, and so producing a relative anæmia and possibly a thrombosis. Such an event may be the result of the vasomotor paralysis produced by toxic agents, and a special form of paralytic vasodilatation has been described in connection with syphilitic myelitis.

The *effects of local anæmia* are dependent upon the degree to which the cutting off of the blood supply attains. A diminution in the blood stream may exert deteriorating influences upon the nerve cells of the anterior horns, while the rest of the tissues escape. A further diminution may destroy the myelinated nerve fibers, including their axis cylinders, the neuroglia still maintaining sufficient integrity to undergo proliferation, and thus to inaugurate a process of repair and sclerosis. Finally, when the evascularization is complete all tissues succumb in the affected area, and, according to Schmaus, the attempts at repair and the removal of debris are carried out by mesoblastic elements, with the result that a connective tissue scar, with or without the formation of a cyst, replaces the parts which have been destroyed. In this respect the effects of focal thrombosis in the spinal cord resemble those which obtain in the brain, although the common etiological factors are not the same in the two cases.

**Hyperæmia.**—Hyperæmia of the spinal cord at one time enjoyed a position of considerable importance in relation to the pathology of spinal diseases. Nowadays it has fallen from its pedestal, and, whether active or passive, is regarded rather as a concomitant phenomenon of physiological or morbid activities than as a primary factor capable of evoking clinical symptoms or anatomical lesions. It is sufficient to say that the spinal cord shares with other organs the power of attracting an excess of blood during functional activity and the inability to avoid plethora when it is the seat of inflammation or of passive congestion.

## HEMORRHAGE INTO THE SPINAL MEMBRANES.

**Extradural Hemorrhage.**—Extravasation of blood may occur between the dura mater and the periosteum lining the inner surface of the vertebræ. This space contains, especially posteriorly, a considerable quantity of loose areolar and fatty tissue, in which lies a rich plexus of veins. Bleeding into this epidural region occurs in the large majority of cases as the result of trauma, fractures of the vertebræ, with or without laceration of the dural sheath, being the most common cause. Less frequently extravasation may be due to violent involuntary muscular contractions, such as those which are associated with tetanus, eclampsia, and infantile convulsions. The venous congestion of cardiac and pulmonary disease combined with the excessive

action of the respiratory musculature in terminal dyspnœa may also be responsible for hemorrhage from the venous plexus in this situation.

Finally, the bursting of an aortic aneurism into the extradural space will in rare instances produce a hemorrhage of a gross character. With the exception of the latter cases, the amount of blood poured into the extradural space is rarely sufficient to produce symptoms, and its almost invariable occurrence within a few hours of death from other causes renders these symptoms likely to be overlooked. Only in exceptional instances, such as with aneurism, is pressure upon the spinal cord likely to be brought about. These hemorrhages are therefore usually recognized only post mortem. The blood may be limited to a small area or diffused throughout the length of the vertebral column, sometimes penetrating, with the spinal nerves, the intervertebral foramina. Owing to the susceptibility of the cervical vertebræ to serious injury, hemorrhage is more common in that region than elsewhere.

**Intradural Hemorrhage; Hæmatorrachis.**—In this the blood is effused into the arachnoid sac and is more or less mingled with the cerebrospinal fluid, to which it gives a hue proportionate to the size of the extravasation. As a general rule, the source of bleeding is in the vessels with which the pia mater is richly supplied. In other cases blood has made its way into the spinal theca from the cranial cavity, where it has escaped as the result of a hemorrhage of traumatic or non-traumatic origin. Laceration of the dura mater may also allow of extravasation into the arachnoid sac from surrounding tissues. Aneurism of a vertebral artery is a very rare cause of intradural hemorrhages. The most common origin of small hemorrhages or ecchymoses on the surface of the pia is to be found in the fulminating forms of septic meningitis. In such cases numerous extravasations, sufficient to color the cerebrospinal fluid, are not infrequent. Hemorrhages are also met with in connection with tetanus, eclampsia, and epilepsy, as well as with purpura, scurvy, and hæmophilia. Urgent dyspnœa as a result of pulmonary or cardiac disease or of respiratory paralysis is an occasional causal factor.

In most cases there are no symptoms referable to the effusions of blood which can be distinguished from those of the primary lesion, whether it is of cerebral or spinal origin. In other instances, usually of traumatic, but very occasionally of spontaneous origin, to which the term spinal apoplexy (or *apoplexia canalis spinalis*) has been given, the amount of blood effused is sufficient to produce a recognizable symptom complex.

**Symptoms.**—The symptoms are produced by pressure upon the leptomeninges and upon the spinal roots, and are characterized by the rapidity of their development. Sudden, violent pain referred to the back, associated with tenderness over a considerable length of the vertebral column, is the first indication. This may give rise to spinal rigidity or opisthotonos, and is quickly followed by root pains in the distribution of the spinal nerves, by various paræsthesias in the extremities, and by involuntary spasmodic contractions in the muscles of the trunk, legs, and arms. Pain may be evoked by attempts at micturition and defecation or there may be temporary retention of urine and fæces.

Such are the symptoms of meningeal and root irritation. They may be short-lived or they may merge into more serious disturbances. Corresponding to the site of the hemorrhage, paralysis of the arms or legs may develop, and hyperæsthesia of certain cutaneous areas may be followed by anæsthesia and analgesia, both superficial and deep. This is particularly liable to

affect the lumbar and sacral root areas, owing, probably, to the collection of blood in the lowest part of the thecal sac and consequent pressure upon the cauda equina. Abolition of the tendon reflexes and retention or even incontinence of urine and fæces may be observed at this stage.

The evolution of this clinical picture is usually associated with a certain degree of shock and in severe cases with loss of consciousness. The pupils are contracted at first and may be dilated at a later period. The pulse is small and rapid, and there may be an initial fall of temperature.

The disease reaches its height in the course of a few hours or a day or two, and it is during this time that life is most dangerously threatened either from shock, respiratory paralysis, or in very rare instances from extension of the hemorrhage into the cranial cavity. An early retrogression of symptoms may be expected in those who survive, but the period of blood resorption is often attended by an exacerbation of the phenomena and by pyrexia and constitutional disturbances. This is not often fatal, although some days may elapse before definite evidence of progressive recovery from paralysis is forthcoming. From this time onward complications arising from decubitus or cystitis are the chief source of anxiety, and these may generally be successfully dealt with provided the spinal cord itself has not sustained irreparable damage. A considerable degree of recovery is generally attained in the course of six or eight weeks, but residual troubles in the form of atrophic palsies, bladder disorders, and paræsthesias may require many months to clear up, or may even be permanent in the worst cases.

**Prognosis.**—Prognosis has already been indicated by reference to the chief sources of danger. The nature of the primary lesion in traumatic cases, and particularly the level of the cord, to which the symptoms point, must be taken into account. The confinement of symptoms to those of meningeal irritation is a favorable sign and generally denotes a rapid recovery. Signs of an intramedullary lesion render the outlook very grave from the point of view of recovery from paralysis and sphincter disorders.

**Diagnosis.**—The diagnosis of spinal apoplexy is rendered easy, as a rule, by the history of trauma immediately preceding the development of symptoms of meningeal irritation. A slow evolution of the latter without pain is a very exceptional occurrence, although it has been recorded. When there is no history of injury, meningitis may be suspected, and the differentiation will then depend on the slow onset of the latter condition, the more pronounced early pyrexia, and especially on the examination of the cerebrospinal fluid. This may be blood-stained in either case, but an excess of polymorphonuclear leukocytes or of lymphocytes and the presence of bacteria will determine the diagnosis of meningitis.

The diagnosis of hæmatorrachis from hæmatomyelia depends chiefly upon the early preponderance of paralytic over irritative phenomena and the more localized character of the pain in the latter disease. It must be remembered that intradural and intramedullary hemorrhage may co-exist in traumatic cases.

Gowers refers to the possibility of mistaking spinal apoplexy for strychnine poisoning, and notes the case reported by Dixon, in which an extensive hemorrhage into the dural sac gave rise to violent paroxysms of muscular spasm and general pain. In such instances the question as to whether an effusion of blood into the arachnoid space is the result or the cause of muscular spasms may often be difficult to answer.

**Treatment.**—The first essential in treatment is physical and mental rest, which is best promoted by placing the patient in bed and administering a hypodermic injection of morphine. It is generally assumed that the prone position exerts a favorable influence on hemorrhage or inflammation in the spinal tissues, but on what grounds this opinion is based it is difficult to see. The importance of securing the patient's repose and comfort probably outweighs any advantage that can be gained from invoking the help of the laws of gravity. The application of ice to the spinal column may be of service in arresting hemorrhage. Scarification of the surface over the spine at the seat of pain is suggested by Gowers as probably the wisest treatment at the onset. Calcium salts may be given by the mouth in order to avert further oozing, although they can hardly exert any influence on the initial hemorrhage.

The after-treatment is that of atrophic palsies in general, and the persistence of symptoms indicating pressure on the spinal cord or cauda equina may eventually justify an exploratory laminectomy.

## HÆMATOMYELIA (HEMORRHAGE INTO THE SPINAL CORD).

Hemorrhages within the substance of the spinal cord are not infrequently found post mortem in cases of spinal inflammation or spinal thrombosis, but the symptoms of these cases belong to the primary disease, and are not those of a true hæmatomyelia. Capillary hemorrhages are also common in patients who have died with urgent dyspnœa, particularly in cases of pulmonary disease, myasthenia gravis, and in all instances of paralysis of the respiratory musculature. Here, again, there are no clinical symptoms referable to the hemorrhages. The term hæmatomyelia is reserved for cases in which a hemorrhage is mainly responsible for the clinical picture.

**History.**—The first recorded case of spontaneous hæmatomyelia appears to be that observed by Gaultier de Cloubry in 1808. This is referred to by Ollivier d'Angers in 1827, and the latter, in the third edition of his *Traité de Maladies de la Moelle Epinière*, gave the name hæmatomyelia to the condition. In 1828 other cases were recorded, and Cruveilhier drew attention to the tendency on the part of the hemorrhage to confine itself to the central gray matter of the cord. Lœvier, in 1864, reported a pure case of hæmatomyelia in a young girl, and suggested the name of *hæmatomyelia tubularis* for extensive longitudinal hemorrhage. In 1869 Koster expressed the opinion that the majority of cases of hæmatomyelia were secondary to myelitis, and this view was shared by Hayem and Charcot. Hayem appears to have neglected the primary traumatic hemorrhages, and wrote at a period when all forms of softening in the spinal cord were regarded as inflammatory or myelitic. A careful investigation of a case, carried out by Eichhorst in 1874, showed that the hemorrhage was primary in a previously normal cord. Erb (1875), Weber (1876), Fox and Ross (1883) were among the first to make the clinical diagnosis, and in 1888 von Leyden described a case in connection with pregnancy. In 1890 Minor brought forward his view that many instances of syringomyelia originate in the track of spinal hemorrhages. Further contributions have been made in recent years by many writers. A good bibliography may be found in a paper on the subject by Carl Doerr.[1]

[1] Deuts. Zeit. f. Nervenheilk, 1907.

**Etiology.**—Since sclerotic vascular change is one of the most important factors in the production of cerebral hemorrhage, it might be expected that similar changes in the spinal cord would determine the occurrence of spinal hemorrhage, but this is not the case. Sclerotic degeneration of spinal arteries is not uncommon, but it rarely gives rise to hæmatomyelia, and the comparative rarity of the latter condition as compared to cerebral hemorrhage is sufficient evidence of the essential difference in their etiology. Possibly the circuitous course of the spinal vascular supply modifies the effects of high arterial tension.

By far the most common factor is trauma or strain, although there are a few cases in which hemorrhage appears to have occurred without any obvious cause or predisposition. Included under the term trauma are cases of injury to the spinal column, with or without fracture or dislocation, cases of concussion due to falls on the head, on the feet, or upon the sacral region. Obstetrical injuries have been also shown by Schultze and Spencer to cause hemorrhage into the child's spinal cord. The strain of childbirth and that of great muscular exertion may give rise to the condition, and excessive coitus has been cited by Gowers as a possible causal factor. Hæmophilia, congenital or acquired fragility of vessels, purpura, and other rare conditions have been assumed to exert an influence in exciting spinal hemorrhage.

Hæmatomyelia may occur at all *ages*, but is most common between twenty and forty years, the period of greatest physical exertion and exposure to injury. The laborious nature of their occupations renders men much more liable than women to the disease. The incidence of hemorrhage upon the gray matter is generally explained by the rich vascular supply of that tissue and the comparative looseness of its texture.

**Pathology.**—In recent cases the appearance of the cord may be normal or the soft meninges may present some ecchymoses. Palpation will often detect a soft fluctuating swelling, and, if the hemorrhage is extensive in its transverse direction, the dark bluish-red color of the blood clot may be visible through the surrounding white matter. In more severe cases a large part or the whole length of the cord may form a tubular thin-walled, blood-containing sac. The most common site is the cervicodorsal enlargement; its occurrence in the dorsal or lumbosacral region is rare.

A series of transverse sections shows that the extravasation may be limited to a segment or two, when it will have a round or oval shape, or it may extend through many segments in the form of tapering prolongations upward or downward, in which case it will have from the vertical aspect a more or less spindle-shaped contour. Closer scrutiny reveals the limitation of the blood to the gray matter at most levels, although at the seat of the original leakage the white matter may also be seriously encroached upon and torn up. The track pursued by the hemorrhage usually involves the bases of the posterior horns, but extends also into the anterior and lateral gray substance.

Multiple foci are relatively often seen (in 10 out of 32 cases according to Doerr). The color depends on the age of the hemorrhage, red in the early cases, it becomes brown or ochre in those of longer standing. In very old cases the site may be marked by a serous cyst, or more commonly by longitudinal cracks or fissures with well-defined walls in the posterior gray matter. It is stated that the blood rarely courses along the central canal.

**Histology.**—The nerve substance surrounding the blood in recent cases is seen to be partially disintegrated by mechanical means and by the second-

ary œdema. Proliferation of the neighboring neuroglia soon takes place, and granular cells are seen in considerable numbers. These appearances have been regarded as inflammatory, but this is not correct if the word is used in its ordinary sense, and it seems a pity to assume a secondary myelitis in order to describe the process of repair.

Secondary changes in the nervous elements comprise disappearance or atrophy of ganglion cells, degenerations in the ascending and descending spinal tracts, and atrophy of the anterior root fibers.

**Symptoms.—Onset.**—Only in rare cases is this preceded by warning in the form of tingling or numbness corresponding to the site of the future lesion. As a rule, the development of symptoms is absolutely sudden, exceptionally occupying a few minutes to half an hour. A slower onset is suggestive of a secondary hemorrhage, except in those cases when a slight leakage is followed shortly by a more extensive extravasation.

Abrupt paralysis involving all parts below a certain level is characteristic of the disease, and may or may not be accompanied by pain. The latter, when it is present, is often of an intense radiating character, or may be described as a burning sensation constricting a part of the trunk, usually the upper thoracic region. Consciousness is usually retained, except in the most severe hemorrhages, and no disturbance of general health is immediately noticeable. If the lesion occupies the cervical *enlargement*, examination at this period reveals a flaccid palsy of all four extremities, together with paralysis of the thoracic and abdominal muscles. Unless the fourth cervical segment has been involved, the diaphragm will continue to act. In addition to the motor paralysis complete loss of sensibility to all stimuli is found up to a level corresponding with the abolition of voluntary power, no deep or superficial reflexes can be elicited in the affected parts, and complete retention of urine and fæces supervenes. Interruption of the oculopupillary fibers leaving the cord at the level of the eighth cervical or first dorsal segments produces contraction of the pupils and narrowing of the palpebral fissures. Splanchnic palsy entails distention of the abdomen, which may add to the respiratory embarrassment occasioned by the absence of intercostal action. The pulse may be slowed or unaltered. The temperature usually remains normal at first, and rises after a day or two.

In the course of a few days, unless a fatal termination has ensued, a marked retrogression of symptoms usually takes place. The arms remain flaccid and present evidence of muscular atrophy, associated with changes in the electrical reactions. The lower extremities, on the other hand, become spastic, with increased tendon reflexes and extensor plantar responses. A certain amount of recovery in power is often exhibited at this stage both in the trunk and legs, but the abdominal reflexes remain absent. Incontinence of urine sets in and obstinate constipation, alternating with incontinence of fæces, may be expected. Priapism has been noted, but is not always present.

Cutaneous sensibility may remain generally impaired, but more frequently tactile stimuli become recognizable, while painful and thermal sensibility are still lost. Unless great care has been exercised bedsores may have rapidly developed in the first few days, and may prove difficult to heal.

Occasionally the symptoms quickly assume, or have from the beginning, a unilateral character corresponding to what is known as Brown-Séquard's paralysis. In that case one arm may present flaccid, and the corresponding

leg spastic, paralysis, painful and thermal sensibility being lost in the affected arm and in the trunk and leg of the opposite side.

Hemorrhage into the *dorsal cord* produces a similar picture, with the exception that the arms are unaffected and the oculopupillary fibers escape. When the *lumbosacral enlargement* is the site of disease the lower extremities are rendered powerless, all their deep and superficial reflexes are abolished, and the sphincters are relaxed and incontinent. In these rarer cases the retrogression of symptoms is usually less marked, although a few muscles may regain power and complete sensory loss may be replaced by that of the dissociative type in certain areas.

With hemorrhage into the *conus medullaris* there is paralysis of the bladder and rectum, impotence, and anæsthesia of the perineal region, the anus and the genital organs, occasionally of the posterior surface of the thighs. Vasomotor and sensory phenomena may be observed in paralyzed regions, early vasodilatation and hyperidrosis being succeeded by a pale dry skin in the later stages.

The future course of the severe cases described is generally marked by continued improvement up to a certain point, but some muscular atrophy usually remains when the cervical or lumbar enlargements have been the site of hemorrhage, and some degree of spastic paraplegia when the cervical or dorsal regions have been the parts affected. In less extensive hemorrhages the injury may be confined to one side of the gray matter, when sensory and motor phenomena are limited to a single limb. More rarely an area of motor paralysis is observed without any sensory loss, or even a partial hemianæsthesia without any degree of paresis. These mild cases may recover completely, although the occurrence of some muscular atrophy usually precludes such a favorable outlook.

**Diagnosis.**—Hæmatomyelia is, as a rule, easily distinguished from other forms of spinal disease by reason of its abrupt onset and the rapid partial amelioration which supervenes on the resorption of extravasated blood. *Hæmatorrachis* or meningeal hemorrhage is characterized by more diffuse pain along the whole length of the spine, by the preponderance of signs of irritation over those of paralysis, and by the still more rapid retrogression of symptoms. Lumbar puncture may aid in the diagnosis, but it is contra-indicated in cases of hæmatomyelia. *Acute infective myelitis* is associated with marked constitutional disturbance and pyrexia, and a less rapid onset of symptoms. *Syphilitic myelitis* may have a very abrupt development, but the history of lues and the premonitory symptoms are usually sufficient for a correct diagnosis.

In children hæmatomyelia may simulate an *acute poliomyelitis*, and in certain cases the differentiation may conceivably be impossible. The almost invariable occurrence of sensory and sphincter disturbances in the former and the pyrexia and malaise associated with the latter disease are safe guides in the majority of instances.

The clinical picture of a case of hæmatomyelia may be identical with that of *syringomyelia*, in which circumstances the diagnosis can only be arrived at from a consideration of the history of onset and the absence or presence of evidence of progressive symptoms.

**Prognosis.**—Life is threatened at the onset when the extravasation is at a high level or when it is severe and continuous. A few cases die rapidly, either from respiratory failure or from shock. The majority survive the early

dangers, and the future outlook then depends upon the avoidance of pulmonary complications and of sepsis in connection with bedsores or cystitis. Complete recovery from paralysis can hardly ever be expected, and the prospects in this connection must be guaged from the atrophy and the electrical reactions of affected muscles. So long as a muscle responds to the faradic current the hope of its regeneration may be maintained. The disappearance of all response to electrical stimuli is a sure sign that the paralysis is permanent. The parts affected by spastic paresis may reasonably be expected to show continued improvement over a long period of time because the encroachment of the disease upon the white columns is usually only temporary and incomplete. The return of sphincter control depends upon much the same anatomical data, except in cases of lumbosacral hemorrhage, in which the anal and vesical sphincters are more likely to be permanently paralyzed.

**Treatment.**—It is imperative to secure absolute rest. The patient should be placed in bed, on a water mattress if possible, and given a dose of morphine hypodermically. The prone position is generally advised, but is probably quite unnecessary and certainly not conducive to the patient's repose or comfort. Ice may be applied to the back. The bladder and skin should from the very beginning receive assiduous care and attention. The bowels must be relieved by aperients or enemata, or both. The patient should be warned that coughing, sneezing, and all movements are injurious, and his diet should be nutritious and non-stimulating. Insomnia may be treated by bromides, veronal, sulphonal, or paraldehyde, but the administration of sedatives must be regulated by the condition of the respiratory muscles and organs.

Active treatment of the paralyzed parts should not be undertaken for six or eight weeks after the last sign of hemorrhage has been observed, and the first form allowable is electricity, which entails no active movements. This may be followed by massage and passive movements, and still later by gymnastics, baths, and other remedies devised for those who have faith in them. Iron, quinine, and arsenic are preferable to strychnine and digitalis in the way of tonics, and a change of air and scene is probably of most value to a patient who has had a prolonged period of dull inactivity and confinement.

The treatment of the cystitis and bedsores, which are sometimes inevitable in spite of all precautions, is the same as of those of other origin, if it is remembered that every care must be taken not to move the patient more than is absolutely necessary in the early days of the disease.

# CHAPTER VII.

## TOPICAL DIAGNOSIS OF DISEASES OF THE BRAIN.

By JOSEPH COLLINS, M.D.

**Introduction.**—There are certain areas of the cerebral cortex which have definite, highly specialized function, to which the name *centres* has been given. Thus, we speak of the motor centre, the centres for the different varieties of sensation, and the speech centres. Some of these areas are more closely delimited than others, such, for instance, as the motor area. Others, such as the sensory area, are spread over considerable territory, and their limitations have not been strictly and satisfactorily defined. The determination of these areas of the cortex, to which highly specialized function is confined, was one of the brilliant contributions to medicine of the nineteenth century. That the brain was the originator of motor, and the recipient and interpreter of sensory impulses, was taught from the time of Herophilus, although it was always in question until the seventeenth century. The idea that the brain was not functionally homogeneous was, from the point of view of the physician, much more important, for it has been the fundament of the present-day wondrous knowledge of the plurality of function of the brain and the allotment of office to separate centres. The impulse to this conception was given by Thomas Willis, who was one of the first to consider the brain as an assemblage of various apparatuses and to assign special capacity to some of its divisions, and by Gall, whose name is unalterably linked with fantasy. The next impetus came from the publication of Charles Bell's idea of a new anatomy of the brain, in 1807, in which he announced the discovery of the different functions of the nerves corresponding with their relations to different parts of the body. The claims of Dax, in 1836, that the faculty of speech was located in the left frontal lobe, and of Broca, in 1861, that the foot of the left third frontal convolution presided over this faculty, were pioneer steps in the study of brain localization. Many investigators have had an important share in establishing the accepted status of cerebral localization, but the work done about the year 1870 by Jackson, Fritsch and Hitzig, Ferrier, Horsley and Munk initiated the revolution which established the facts upon which are based the present-day doctrines of the functions of the brain cortex. Since then neurologists the world over have, by close observation and exact report of persons suffering from limited disease of the brain, made important contributions to it. These, in conjunction with the conclusions of the physiologists based upon experimentation, represented particularly by Sherrington and Grünbaum and their pupils, of the anatomists based largely upon embryological research, particularly of Flechsig and v. Gudden, and of the histologists represented by Cajal, who described a specific motor type of cortex, have served to raise cerebral localization to a position of much exactitude.

Topical diagnosis of brain disease is possible only when focal symptoms exist. A discussion of topical diagnosis resolves itself, therefore, into a consideration of the location of function to definite parts of the brain; in other words, to a discussion of brain localization. The so-called general symptoms of disorder of the brain are of great importance in leading to a diagnosis of disease of the brain, but they are of no value in determining the part that is diseased.

The focal symptoms of disease of the brain are, first, those that manifest themselves through the emissive channels, twitching, spasm, convulsion, paralysis, dysarthria, anarthria, and second, those that manifest themselves through the percipient, apperceptive, and interpretative mechanism, such as disorder of tactile, thermal, pain, postural and spatial sensibility, apraxia, agnosia, astereognosis, aphasia, agnesia, and anopsia.

The localization of diseases within the substance of the brain is dependent upon the existence of symptoms similar in a measure to those resulting from disease of the cortex, because they are due to lesions of pathways leading to and from the cortex. In addition, there occur symptoms due to involvement of parts having more or less specific function, such for instance as the nuclei of some of the cranial nerves.

**The Motor Area of the Brain-cortex.**—The centres for voluntary movement are situated in the anterior central, precentral, or ascending frontal gyrus. This gyrus extends on the mesial surface of the brain from the paracentral lobule to the operculum. It was to this area of the cortex that Fritsch and Hitzig first attributed representation for voluntary movements. A vast amount of clinical and experimental evidence was offered to show that motor representation was not narrowly confined to this area, and until recently, practically all writers upon the subject have stated that the motor area of the brain is in the anterior and posterior central convolutions, *i. e.*, in the cortex on either side of the fissure of Rolando. But the preponderance of the evidence to-day shows that representation is as stated above, and the most convincing part of this evidence has been furnished by the work of Sherrington and Grünbaum on the higher anthropoid apes, which corroborated the results of Fritsch and Hitzig, and of Ferrier.

For a long time Munk, whose work upon this subject was very important, contended that in addition to the true motor zone there are a number of areas scattered over the entire cortex which will produce movement if an electric current is applied to them, but Sherrington and Grünbaum have shown that true motor responses are never obtained save from stimulation anterior to the Rolandic fissure. To different parts of this anterior central convolution are allotted centres for the different parts of the body. It may be said that a manikin, with its head resting on the operculum and its feet over the paracentral lobule, will adequately convey the representation, *i. e.*, the centre for the face and its components occupies the lower third, that for the arm and the trunk the middle third, and that for the legs and feet the upper third. Formerly it was thought that there was considerable localization of function to the mesal surface of this area, *i. e.*, the paracentral lobule, but this view has gradually lost ground. It is not only the visible surface of the anterior central convolution which has motor representation, but the entire cortex. Stimulation of the hidden surface of the cortex in the depths of the Rolandic fissure produces definite movements. Notwithstanding the statements just made that motor representation of volun-

tary movement is confined narrowly to the anterior central convolution, it must be admitted that there is an area anterior to the precentral convolution, and particularly in the posterior end of the second frontal convolution, irritation of which produces movement of the eyes. Moreover, irritation of the posterior end of the first frontal convolution produces movement of the head to the opposite side.

**The Focal Symptoms of Disease of the Motor Cortex.**—The most striking symptoms which disease of the cortex of the brain produces are motor symptoms, which manifest themselves in two forms: In expression of irritation—twitchings, spasms, and convulsions; and of obliteration—paralysis more or less complete. As a rule, it is lesions that develop slowly, such as new-growths of various kinds, and lesions which are not of sufficient severity completely to overwhelm, such as poisonous matters circulating in the blood, that produce irritative effects, while rapidly occurring lesions, such as hemorrhage, acute inflammation, and traumatic destruction, produce paralysis. In many instances, disease of the motor cortex is manifest first by irritative phenomena: twitchings and convulsions, which, after a variable time—in one instance a few minutes and in another many months—are followed by paralysis. The kind of lesion that produces such a condition does not call for lengthy consideration. Occasionally paralysis follows immediately upon convulsions and is transitory in its duration. In such cases the paralysis is interpreted as an acute exhaustion of functional capacity of the motor cells following an intense so-called motor discharge.

The distribution of spasm due to irritation of the motor cortex depends largely upon the area that is involved, although it does not absolutely correspond to this. An irritative lesion of the motor area of the brain may manifest itself in twitchings and spasms, limited to the corresponding contrololateral peripheral parts, such as the phenomena known as Jacksonian epilepsy. It may, on the other hand, show itself first in these parts, but before the convulsive seizure has passed it may extend to the entire body. Any attempt to explain this would necessitate discussion of a theory to explain the action of the motor cortex.

Spasm and convulsion caused by irritation of the motor cortex are usually followed by loss of consciousness, but in many instances, and especially in diseases that are of small extent and slight intensity, and those which develop very slowly, the twitching manifestations frequently occur many times (*i. e.*, over a protracted period) without accompanying loss of consciousness. Sooner or later unconsciousness becomes an accompaniment of the attack.

The antithesis of spasm is paralysis. When the motor cortex no longer responds to irritation, lesion of it is evidenced by paralysis. Paralysis, it matters not how brief its duration, indicates abolition of function of the motor cortex. The intensity and distribution of the paralysis depend upon the nature and extent of the lesion that produces it. A slowly growing tumor in the middle of the motor area may produce a slight, slowly increasing paralysis of one upper extremity, showing itself in a finger or thumb, and gradually, as the tumor increases in size and encroaches upon other parts of the motor area, the paralysis involves the face and the lower extremity. On the other hand, a cortical hemorrhage following trauma may produce a complete hemiplegia within a few minutes. Circumscribed encephalitis may be so narrowly confined in the cortex that only the face or the face and

shoulder may be paralyzed for any considerable length of time, although the œdema, which is secondary to the inflammatory process, may cause a disturbance of motor function over a much more extensive area. As a rule, paralysis of cortical origin is characterized by incompleteness and by definite clinical phenomena: slowness of development, spasticity, diminution or abolition of the plantar-jerk, exaggeration of the tendon-jerks, these phenomena having been preceded or accompanied by spasm or convulsion, and particularly by the fact that it is confined in the beginning at least to one member of the body. Naturally, a lesion that obliterates suddenly the functions of the motor cortex causes paralysis of abrupt onset. In such cases spasm and convulsions are likely to be added to the clinical picture later when the results of the reparative process (scar tissue, adhesions) act as an irritant. The permanence of a paralysis depending upon a cortical lesion is intimately associated with the severity of the disease process. It is a common experience to have hemiplegia follow surgical interference of, or in the vicinity of, the motor cortex, which disappears after a few days or weeks. In other words, the motor cortex tolerates a considerable injury without consequent permanent paralysis.

**The Focal Symptoms of Disease of the Sensory Cortex.**—Knowledge of sensory localization in the cortex is much less definite than that of motor, the reason for which is not far to seek. The manifestations of lesion of the motor sphere are very striking, mainly objective, and not readily susceptible of misinterpretation. The manifestations of sensory irritation, on the other hand, are frequently indefinite, subject to wide variation, often confined to one variety of sensibility and not affecting another, and in every instance subject to personal interpretation. One form of sensation may be disordered, while others such as muscular may be preserved.

For a long time it was believed that sensory representation in the cortex coincided very closely with motor, and this view, promulgated by Munk, is still held by many students of physiology, such as Horsley. It was thought that the Rolandic cortex was in reality sensorimotor. A few, such as Bastian, carried this idea to the length of admitting that there was no real motor zone, and that the zone thus designated was in reality kinæsthetic, *i. e.*, an area in which were stored memories of motion and which conditioned motion in response to sensory impulses. Gradually, however, the advocates of the separate localization of the sensory and motor areas have gained ground, and the soundest teaching at present seems to be that the sensory area of the brain is confined largely to the posterior central convolution on the external surface of the brain, and to the falciform lobe on the mesal surface, particularly the central portion of the latter. On the external surface of the brain, sensory representation extends backward into the superior parietal convolution, to the anterior portion of which is allotted form-perception representation, while to the anterior part of the inferior parietal convolution is allotted muscular sensibility.

Much effort has been made to give the various forms of common sensibility definite localization. The various sensibilities making up the "muscular sense" are thought to be located in the posterior half of the postcentral gyrus, the tactile, pain, and thermal senses in the anterior half of the same convolution.

Mills, whose contributions to the subject of cerebral localization are of the first order, maintains that the cortical representation of cutaneous and

muscular sensibility is subdivided into a mosaic of centres, each of which is correlated anatomically and functionally to one or more motor centres, and that the areas of sensibility are subdivided like the motor area. Although this view is not yet accepted generally, there is much incontrovertible evidence in favor of it, quite enough to justify the position here taken.

**Allochiria.**—Allochiria (other hand) is the name given to a condition in which, though sensibility is retained more or less completely, the patient cannot tell which side of the body is being touched. The patient can feel the touch and can often describe the qualities of the object exciting it, but refers it to a spot on the opposite limb corresponding to that touched. Although allochiria in connection with the special senses, motor and reflex allochiria, have been described, here only sensory allochiria will be considered.

E. Jones has recently maintained that under the name of allochiria two fundamentally different conditions have been confused: (1) Part of a general defect in localization (alloæsthesia), and (2) a specific defect independent of any error in localization (dyschiria). In other words, dyschiria of this writer, in reality, is allochiria of the majority of writers. He divides dyschiria into three stages—achiria, in which the patient has no knowledge as to which side of the body the stimulus was applied; allochiria, in which he refers the stimulus to the corresponding part of the opposite side; and synchiria, in which he refers it to both sides. In alloæsthesia only about half the sensory stimuli are referred to the contralateral side, and there are usually disturbance of common sensibility and often corresponding motor manifestations, whereas in allochiria all of the stimuli are referred to the contralateral side. It bears no relationship to cutaneous sensibility and there is no motor or special sense disturbance, but it is a mental manifestation in which there is an inadequacy of appreciating the sense of laterality. Neither allochiria nor alloæsthesia has any topical diagnostic value. All the evidence so far is in favor of the view that allochiria is a manifestation of hysteria, and does not occur with organic diseases.

**Astereognosis.**—Recently much clinical importance has been given to astereognosis and to the localization of the so-called "stereognostic sense." Great confusion exists because of the loose way in which the term has been used by different writers. Stereognosis means form perception, but most writers use it to mean the capacity to recognize objects by handling them. which incites a great number of sense impressions, tactile, muscular, thermal, and articulatory.

A number of years ago Burr contended that the "stereognostic sense" is not a sense at all but a judgment, and Prince has recently emphasized this point. Unfortunately the term has come to have this application. In this sense stereognosis being an intellectual process, it cannot be localized any more than any other feature of the intellect. However, if it is limited to perception of form alone, tactual astereognosis may well have definite localization. The term agnosia, meaning inability to recognize objects because of lack of information about them, is a term that has a more legitimate usage. Tactual agnosia is the inability to recognize objects by touch. Such capacity may also have definite localization, insomuch as the various forms of tactual sensibility may have definite localization, but no more. Disturbance of stereognostic perception may follow injury, accidental or surgical, of the motor area, the ascending frontal convolution, and in a few instances it has accompanied disease of the area; but at the present time

the occurrence of astereognosis indicates lesion of the superior parietal convolution, and especially its anterior portion in the vast majority of instances.

**"Soul Paralysis."**—We sometimes encounter cases of motor paralysis, that is, loss of voluntary and spontaneous motor action, in which there is no disease of the cortical motor areas or of their projections. Physiologists do not all admit that unilateral motor paralysis may result from destruction of the avenues that convey common sensation alone, but the preponderance of opinion is in the affirmative. It is certain, however, that when the cortical sensory centres or the tracts connecting them with the motor areas are destroyed, ability to perform voluntary motor acts is lost and spontaneous motor power is impaired, occasionally wholly abolished. This condition, called "soul paralysis," "Seelenlähmung" of the Germans, first described by Munk to indicate a bizarre state noticed in dogs after extirpation of the sensory cortical area, has been studied in man by Bruns, Anton, Hoppe, Pick, and many others. Like other conditions or symptoms thought to be new, we find on examination of the older writers that it had not escaped their notice. There is no doubt that Duchenne described it under the name of "perd de la conscience musculaire." It is usually associated with profound sensory disturbances, and its most striking feature is due to a break in the sensory or afferent section of the reflex arc. Voluntary acts are really reflex acts and are possible only when the reflex arc is intact. An act performed in response to an effort of the will calls for a revival of the sensory memories that accompanied similar performances in the past and the stimuli that pass from them to the motor centre produce the movement that the will desires to produce. These afferent stimuli pass from the areas in which such memories are "stored," to use a convenient colloquial expression. In practically all cases of soul paralysis that have been investigated anatomically there has been found destruction of the cortex or of the subcortical region of the parietal lobes or of the sensory tracts.

Soul paralysis is not always used in the sense here employed. Nothnagel used it to designate loss of the memory pictures of movement for one extremity or for one-half of the body. This condition Meynert called motor asymbolia. This word, and agnosia, apraxia, and other designations, which have been used with much latitude, are now coming to have very definite meaning attached to them. Their value in topical diagnosis of brain disease has not been definitely assigned, but they are sufficiently important to merit brief discussion.

**Asymbolia.**—The word asymbolia was first used by Finkelnburg to denote inability to understand certain conventional signs aside from speech signs. Wernicke used it in this sense, but enlarged it to include failure to recognize the images of objects, *i. e.*, the loss of the "memory pictures" of the object. Thus, in Wernicke's sense, patients with asymbolia can see, hear, feel, smell, and taste, but they cannot re-recognize objects by these means. That is, they do not call up the previously existing memory pictures. They cannot turn sense impressions to account. The term asymbolia is therefore used by some in a sense not unlike that of agnosia.

**Agnosia.**—This is a term used to indicate inability to recognize things for what they are. An object is perceived by the peripheral sense apparatus, but it is not seen by the "mind's eye." Thus, there may be as many kinds of agnosia as there are special senses, for it is only through the mediation of one of the special senses that we recognize objects. It is legitimate to speak

of visual agnosia (mind blindness), auditory agnosia (mind deafness), tactile agnosia, gustatory agnosia, and olfactory agnosia. Tactile agnosia is not the same as astereognosis, although it is frequently confounded with it. Before an object can be used properly, it must be recognized; therefore, agnosia entails dyspraxia, often apraxia, but apraxia does not necessarily indicate the existence of agnosia; in fact, there are many cases on record in which it existed without agnosia.

**Apraxia.**—Our knowledge of apraxia, the most important symptom of lesion of the cortex of the brain next to disturbance of motion, sensation, and hemianopsia, is due very largely to the recent work of Liepmann, although Hughlings Jackson called attention to it more than forty years ago. Since that time the subject has been extensively studied by neurologists. An excellent review of the subject and the literature has recently been published by Wilson.[1]

Apraxia is the inability to perform purposeful familiar acts by a person who is not paralyzed, anæsthetic, or ataxic. It is a much commoner symptom of organic disease of the brain than is generally admitted. The fact that the localization of the lesion which causes apraxia is not so definite as that which causes other symptoms, paralysis and spasm, for instance, explains why so little attention is given to it. The status of apraxia as a definite reliable localizing symptom is not yet satisfactorily established. Two varieties of it are distinguished—motor apraxia and ideational apraxia. In the first there is inability to translate the idea of a movement into a movement and in the second there is some defect in putting together the components of the idea of the movement. It generally involves both sides of the body, but it may be unilateral. Indeed, it may occur in one group of muscles only. Apraxia has been noted with cortical and subcortical lesions of various parts of the brain, but there is constantly increasing evidence to show that lesion of the anterior pole of the left hemisphere, especially the upper two frontal convolutions, will cause it, other portions of the brain cortex being normal. Lesions of the corpus callosum also cause it. v. Monakow is of the opinion that lesion of the gyrus supramarginalis causes apraxia. Bechterew has recently reported a case of apraxia of the right hand, in which in addition to the diffuse changes of dementia paralytica a sharply defined lesion of the left middle portion of the posterior central gyrus, the posterior segment of the gyrus supramarginalis, was found.

Apraxia is associated with lesions of the left hemisphere more often than with lesions of the right. It has not yet been found with lesions below the capsule. The apraxia associated with disease of the left hemisphere may be homolateral or heterolateral. Although the lesions with which apraxia are commonly associated are those of arteriosclerosis and brain tumor, it is not infrequent in the confusional states following attacks of epilepsy, in the dementia of alcoholism, and other toxic states. Like all symptoms, it may be caused by hysteria. Its most frequent symptomatic association is with aphasia.

**Hemianopsia.**—The centre of sight (the primary cortical visual area, so-called) is more definitely located than any other. It is easily accessible in animals, and its blood supply is often disordered in man. Despite this,

[1] *Brain*, 1908, xxxi, 164.

there is no unanimity of opinion on the part of the physiologist and the clinician regarding its limitations. Henschen maintains that it is confined to the calcarine fissure, while Vialet, on the other hand, claims that it includes the entire mesotentorial surface of the occipital lobe. The best opinion at present seems to be that it is situated in the mesial surface of the brain, around the calcarine fissure, in the cuneus of the occipital lobe. Its exact limitations have not been made out. It occupies the cortex and the entire lingual lobe behind the junction of the calcarine and the parieto-occipital fissures, and the entire cuneus extending for half an inch or more on the external aspect of the occipital lobe. Destruction of this area in one hemisphere causes homonymous hemianopsia. It is now generally admitted that permanent visual disturbance occurs only when the entire occipital lobe is destroyed. The primary cortical area for sight has two divisions, a peripheral and a central. The central is for the representation of the macula, and the peripheral for the remainder of the retina. The view of Henschen, that the centre for the macula is situated toward the apex of the cuneus, in the inferior extremity of the calcarine fissure, is the one that has received most substantiation. The lower quadrant of the field is represented in the upper half of the cuneus; the upper quadrant in the lower half of the cuneus.

The secondary, or higher visual area (visuopsychic of Campbell) is an association area variously allocated in the posterior end of the inferior parietal convolution, particularly the angular gyrus, or a considerable part of the parietotemporo-occipital cortex. Its function may be that of a storehouse of visual memories and a part of the mechanism for the appreciation of things seen.

The symptoms which disorder of the primary and secondary visual areas cause, which are available in making a topical diagnosis, are anopsia and hemianopsia, achromatopsia and hemiachromatopsia, central amblyopia, word blindness, and mind blindness. Cases of purely cortical anopsia and hemianopsia are extremely rare, for in the majority of such cases the optic radiations are found to be involved. Still, there are a few cases recorded, such as those of Hun, Beevor, Collier, Holden, and others. Hemianopsia may be partial or it may be complete, depending upon the extent of the primary visual area involved. It may also be double, as the result of successive attacks of bilateral hemianopsia.

In attempting to distinguish hemianopsia due to lesion of the cortex and to lesions of the optic radiations from hemianopsia dependent upon disease of the optic tracts and of the basal ganglia, the peculiar pupillary reaction first described by Wernicke is of importance, although perhaps not of as great utility as was at one time supposed. Wernicke pointed out that when hemianopsia was dependent upon involvement of the cortex and optic radiation, the pupillary response was intact for the entire retina, while when the optic nerves and optic tracts were the seat of the disease, only one half of the retina responded. All that may be said with positiveness concerning the attribution of hemianopsia to the cortex is that when it is unassociated with other symptoms, such as sensory disturbances, pupillary disturbances, and aphasia, it is probably not of cortical origin. Further, quadrant anopsia is suggestive of cortical lesion.

Cortical color blindness, or achromatopsia, is not a symptom of importance in the diagnosis of focal disease of the brain. In other words, the

distinction cannot be made clinically between color blindness due to disorder of different parts of the central ocular mechanism.

*Mind blindness* is the name given to the condition in which an affected individual is unable to recognize familiar objects by the sense of sight. In other words, it is a condition in which visual memories of things previously seen and comprehended cannot be reinvoked. Mind blindness is dependent upon disease of the cerebral cortex, or possibly of tracts which assist certain parts of the cortex. An enormous amount of confusion has arisen in connection with the usage of the term, first, because of the indefinite way in which it is used by some writers, and secondly, because of the synonyms that are used. In the literature one finds mind blindness used synonymously with apraxia, soul blindness, and even soul paralysis. In truth, many of the cases reported under the head of mind blindness are cases of apraxia. The term is here used to indicate object blindness, word blindness, letter blindness, and symbol blindness, with intactness of the peripheral ocular apparatus and the primary visual centre, and due to lesion or disturbance in function of the higher or secondary visual area, and possibly also to lesion of the tracts or association fibers which connect the higher visual field with the lower visual centre.

**The Centres of Hearing and Smell.**—The centre for hearing is situated in the middle of the first temporal convolution. The right temporal lobe serves the left peripheral auditory apparatus, and vice versa. Destruction of this centre is followed by degeneration of the internal geniculate body, which is intercalated into the auditory tract, and of the pathway between the auditory nerve and the auditory centre. The termination of the cochlear division of the eighth nerve, after the ascent of its fibers in the lemniscus, in the internal geniculate body and around the large cells of the quadrigeminal bodies, has been fairly well established. Each of these nuclei of termination is connected with the motor mechanism and with the centre for hearing in the temporal lobe.

*Auditory disorder*, aside from word deafness, is not an important symptom suggestive of topical diagnosis. Subjective auditory symptoms, auditory hyperæsthesia, and variously described paræsthesia may occur with tumor or other slowly developing lesion of the pons, or at the base of the brain encroaching upon the auditory nerve. Similar symptoms preceding an epileptic attack should suggest lesion of the temporosphenoidal lobe of the opposite side. Deafness of some degree is the result of disease of the central auditory apparatus. When it is of the cortex, it is called word deafness (see aphasia); when of other part of the central auditory mechanism, nerve deafness. Instances of bilateral deafness from tumors of the quadrigemina encroaching upon the tegmentum have been recorded.

The centre for *smell* is situated in the uncinate gyrus of the same side, and probably to a certain extent on the opposite side. Jackson and other clinicians have shown that this gyrus is diseased in cases of epilepsy attended with olfactory auræ, such as unaccountable smells. Disorder of the sense of smell, especially when associated with symptoms indicative of brain lesion, should suggest at least an involvement of the uncinate gyrus, but it has also been noted with tumor of the cerebellum.

The higher mental faculties, such as abstraction, introspection, judgment, comparison, reasoning, etc., are subserved mainly by the brain anterior to the motor areas, particularly in conjunction with association areas in other

parts of the brain. So far nothing definite has been established concerning the subdivision of the prefrontal lobe into centres to which are allocated definite components of the higher intellectual functions, and, considering how complex the higher psychical faculties are, and that modern psychology views all mental faculties as arising from a complicated coördination or adjustment of movement to the environment, it is not legitimate to seek to localize them.

There is much convincing evidence to show that Flechsig's anterior centre of association is more concerned with attention and other co-ordinations of psychic process than any other part of the brain. It is an area undeveloped in cases of idiocy and diseased in cases of secondary dementia.

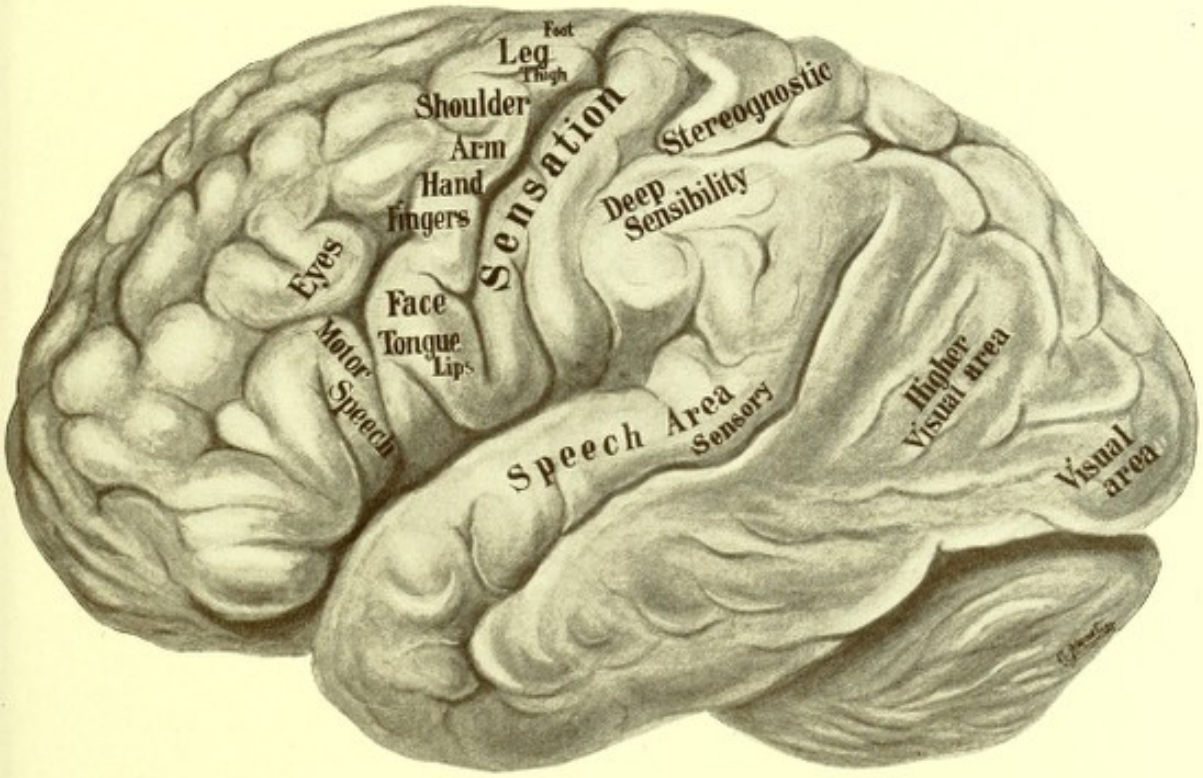
Although there is no centre of attention or thought or apperception or morality in the frontal lobes or elsewhere in the brain, the frontal lobes contain neural elements whose integrity must be maintained if the individual is to develop and persist in habits of attention, concentration of thought, balance of feeling, sound judgment, and moral conduct.

Lesion of the frontal lobes, aside from the so-called ascending frontal (which in this article is referred to as the anterior central), and the foot of the third frontal convolution (Broca's convolution), produces no constant, definite, localizing symptoms. In the majority of cases, however, such lesion is attended with distinct mental symptoms, such as inattentiveness, lack of concentration, impaired judgment, emotionality, and impairment of the higher mental faculties, reaching even to imbecility. If the lesion is of the orbital surface of the frontal lobe, olfactory and optic tract symptoms often occur even in the early stages of the disease. Such symptoms as those enumerated, followed by motor symptoms, disturbance of speech, monoplegia, and hemiplegia, are highly suggestive of lesion in this part of the brain.

Grainger Stewart suggests that absence, diminution, or easy exhaustion of the superficial abdominal reflexes on the contralateral side and a fine, rapid, vibratory tremor in the limbs of the same side are suggestive of tumor of the frontal region of the brain.

**Summary.**—Briefly to summarize the present-day teachings of localization of function in the cortex: The motor area and the sensory area are situated in the centroparietal region, the motor anterior to the fissure of Rolando, the sensory posterior. These areas are subdivided so that different portions of them serve different muscles and different varieties of sensation. An inverted manikin, parallel to the fissure of Rolando, will serve to remind one of the divisions of the motor area. The centres for cutaneous and muscular sensibility are in the posterior central convolution; that for so-called stereognostic sensibility in the anterior end of the superior parietal and probably the adjacent posterior central. The centre for vision is in the occipital lobe, particularly in the cortex around the calcarine fissure, that for hearing in the middle third of the first temporal lobe, and that for smell in the hippocampal region. The left hemisphere contains the speech centres in right-handed individuals, and vice versa; the part of the brain that subserves this function includes the foot of the third frontal, the superior temporal, and the posterior end of the inferior parietal convolutions. In addition to these centres, the cerebral cortex has special constituents serving for association processes, which are located by some

# PLATE XVIII

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in definite centres, but which are by the majority assumed to be scattered over the entire cortex. Speech is related both to the sensory areas and the associative areas. On the one hand, the word-component of ideation is dependent upon the presence of association fibers; on the other hand, it is based upon various sensations which have their seat in the sensory centres. Finally, the frontal lobes are necessary for the maintenance of those processes which underlie the higher psychical faculties.

From this brief presentation of cerebral localization it should follow that if these areas have the functions that have been allotted them, disease of these areas should cause definite symptoms, and it is a fact that it does. It would seem, therefore, that topical diagnosis of brain disease should not be difficult, and it would not be were it not that (1) the disease process is not often confined narrowly to one of these areas; (2) the immediate and subsequent result of disease of these areas is disturbance of function of other areas both proximate and remote; (3) disease of one area may be but part of a widespread disease of the vascular system, the centre that apparently manifests the disease being either the one which is bearing the brunt of the lesion or is most tolerant of encroachment; and (4) in every case of topical disease of the brain there are general symptoms which vary with the nature, seat, extent, and duration of the lesion. Such symptoms occur practically in every case, and are called the general symptoms. They are headache, dizziness, vomiting, convulsions, apathy, stupor, disturbance of gait and station, disorder of intellect usually manifest by sluggishness of the so-called psychical reflex, and disorder of the only part of the brain susceptible to ocular examination, viz., the optic nerves. This, in conjunction with the fact that a very considerable extent of the brain cortex and almost all of the brain substance beneath the cortex has no definite functional allotment, makes accurate topical diagnosis difficult. Occasionally when a lesion is confined narrowly to one of the areas whose function has been definitely determined, especially when the disease process is small and of slow onset, so as not to cause obscuring symptoms, the diagnosis may be made with something approximating mathematical accuracy.

In every case the most essential thing for such diagnosis is careful, repeated physical examination, the results of which are noted and, after a sufficient number of them have been made, compared. If, then, there are persistent constant disturbances of function of one or more of the centres, which have special function, and likewise one or all of the general symptoms that have been enumerated, a definite topical diagnosis is possible.

**Lesion of the Central Convolutions.**—Lesion of the anterior central convolution causes spasm of a muscle or of functionally associated groups of muscles, such as those of the leg, the hand, and the face, of the opposite side, or paralysis, partial or complete, in similar parts. Such paralysis is nearly always spastic unless the lesion is traumatic and dependent upon extensive laceration or hemorrhage, when it is flaccid. When it is of the spastic variety, there will be exaggerated tendon-jerks and the Babinski phenomenon. If the lesion extends backward into the posterior central convolution and the adjacent parietal convolutions, disturbance of muscular, cutaneous, postural sensibility and impaired recognition of the forms and uses of objects, so-called astereognosis will be present. Lesions which permit of topical diagnosis in these regions are tumor, abscess, inflammatory and degenerative processes such as encephalitis and arteriosclerosis, which sometimes confine

themselves narrowly to the anterior central convolution, but in the majority of instances eventually, through pressure or encroachment upon the adjoining area posteriorly, are attended with sensory symptoms. For instance, the occurrence of a series of tonic or clonic spasms in the hand, followed or not by loss of consciousness and other manifestations of an epileptic attack, would immediately suggest lesion of the middle third of the motor area. In case such symptoms were followed by slowly developing monoplegia, and later by such sensory disturbances as impairment of the deep sensibility and astereognosis, the justifiable assumption would be that the lesion starting in this area had extended posteriorly.

A lesion confined narrowly to the posterior central convolution does not produce motor phenomena, but does cause impairment of tactile sensibility and of muscular and articular sensibility, while lesion of the anterior end of the superior parietal convolution, possibly also of the posterior central convolution, produces tactile astereognosis, tactile asymbolia, hemiataxia, and abolition of the superficial reflexes, especially the plantar reflex. Extension of the lesion from this area into the motor area is accompanied by motor manifestations and exaggeration of the tendon-jerks, while extension backward to involve the cuneus or in depth to implicate the fibers of Gratiolet is followed by hemianopsia. Lesion of this area is usually not attended with psychical symptoms or disturbance of speech until late in the disease.

Lesion of the occipital area, and particularly of the cuneus, produces homonymous hemianopsia, which is often associated with word blindness in conjunction with the general symptoms of intercranial disease. This is often a most important localizing symptom. Visual hallucinations, transitory but recurring, are occasionally early symptoms of disease of the occipital lobe. Such hallucinations are referred to one-half of the visual fields.

**Lesion of the Centrum Semiovale.**—The white substance of the brain is made up largely of fibers that convey impulses to and from the cortex and fibers that connect different parts of the same hemisphere or one hemisphere with the other. Disease of it should therefore be evidenced by symptoms varying with the variety and number of the fibers involved, *i. e.*, varying with the situation and size of the lesion. If the fibers coming down from the motor centres to pass through the internal capsule to the pyramidal tract are involved, and these only, the prominent symptom will be paralysis of the opposite side of the body. Lesion of this sort, unless close to the cortex, is not so frequently attended with convulsions, particularly of the localized variety, as is lesion of the motor cortex itself. On the other hand, general epileptoid attacks often occur. If, again, the lesion interferes with or interrupts the sensory fibers radiating to the posterior central and superior parietal convolutions the symptoms will be predominantly disturbance of sensation with hemiataxia. If the lesion is in the posterior part of the white substance, then symptoms referable to vision or to the speech faculty are likely to predominate. A lesion of the white substance of the brain in the anterior pole cannot ordinarily be diagnosed or even suspected. As a rule, the general symptoms are those of intracranial disease with no localizing symptoms. In many instances of disease of this region, tumor, pronounced hebetude, apathy, indifference, and disorientation are early phenomena, but further than this no localizing symptoms have been described. As a matter of fact, lesion of the centrum semiovale, such as tumor, often gives rise to

symptoms thought to be characteristic of remote parts of the brain, as the cerebellum.

**Lesion of the Corpus Callosum.**—Disease of the corpus callosum, particularly tumor, causes symptoms which, although not pathognomonic, are suggestive of invasion of this portion of the nervous system. Mental symptoms occurring early are most characteristic. These are peculiar delusional states, eccentricity of manner and action, impairment of memory, dulness, excitability, want of sequence in ideas coupled with an apparent conservation of the intelligence, followed by the general symptoms of intracranial disease, which may, however, develop very late. Marked weakness of one side of the body may appear and amount to hemiplegia, often associated with hemiparesis of the other side, both attended with more or less contracture, epileptiform attacks with the motor display more often on one side than on the other, ataxia of the paretic extremities. The motor paralysis, which is usually primarily hemiplegic, becomes eventually more or less diplegic, especially the paralysis of the lower extremities, due to pressure on the conducting tracts lying outside the corpus callosum. Cranial nerve palsies are usually absent. An attempt has been made at a subdivision of localization in the corpus callosum, but this is a refinement of diagnosis that is unnecessary.

**Lesion of the Internal Capsule.**—The neuraxones or axis-cylinder processes of the cells of the cortical motor area pass downward in the white matter of the brain, forming a part of the corona radiata, which, constantly narrowing as it descends, passes into the internal capsule, a narrow pathway between the so-called basal ganglia. It is the avenue which impulses originating within the brain must traverse in order to be externalized, and likewise the avenue through which impressions coming from without must pass in order to be interpreted by the brain. In other words, it contains practically all the projection fibers of the cerebral cortex. Therefore, lesion of the internal capsule may produce symptoms that parallel those of lesion of the cortex. A small lesion in this area of the brain will be accompanied by symptoms which could otherwise be caused only by most extensive disease of the brain cortex. Complete destruction of the internal capsule produces flaccid hemiplegia, hemianæsthesia, vasomotor disturbances of the paralyzed side of the body, hemianopsia, and disturbance of hearing on one side.

The internal capsule, which is in the form of an obtuse angle, is divided by the apex of the angle, or the knee, into two portions, an anterior and a posterior. The anterior part, or the lenticulo-caudate segment, contains the fibers from the frontal lobes, particularly those going to the thalamus. The posterior division, the lenticulo-optic and the retrolenticular segments, contains, in its anterior part, the pathway of the lower third of the precentral convolution. These fibers are located in the anterior part of the posterior division. They pass from there into the ventral half of the pons, to end mainly around the nuclei of the facial, the motor nucleus of the trigeminal and hypoglossal nerves. A lesion of this portion of the internal capsule, it is maintained by many, especially by the school of Charcot, causes symptoms referable to these nerves, particularly paralysis of the muscles of the face which are under volitional control. The pathway made up of projection fibers coming from the other regions of the motor cortex, and known as the pyramidal tract, especially after it reaches the pons, is situated just posterior to the tract mentioned above. These two pathways or fascicles constitute the anterior two-

thirds of the posterior limb. Adjacent to this, posteriorly, is the pathway of the general sensibility from the entire body, which, coming in from the tegmentum of the crus, passes up through the internal capsule to go to the parietal convolutions. Charcot and his pupils, particularly Ballet, claimed that lesion of a certain area in the posterior part of the posterior limb of the internal capsule, the "carrefour sensitif," causes hemianæsthesia similar to that of hysteria. But such a view is not now accepted, and it has not been substantiated clinically or anatomically. Capsular hemianæsthesia of organic origin may be accompanied by hemianopsia, but not sensorial hemianæsthesia. When alteration of the most posterior segment of the internal capsule is attended with hemiplegia accompanied by hemianæsthesia and hemianopsia, the optic tract participates in the capsular lesion. Between the pyramidal tract and this fascicle, practically a portion of the former, Charcot, Raymond, and others place the "fascicle of hemichorea," *i. e.*, the portion of the posterior limb of the internal capsule, irritation of which causes hemiathetosis, unilateral paralysis agitans, unilateral intention tremor, and hemiataxia. Finally, the visual tract passing from the pulvinar of the thalamus to the occipital lobe, and the auditory tract going from the auditory nucleus to the temporal lobe, complete the internal capsule.

This being the relative composition, functionally, of the internal capsule, it follows that lesion of different parts of it produces different symptoms. Clinically there is very little satisfactory evidence that such localization exists. Horsley and Beevor have demonstrated, apparently to the satisfaction of physiologists, that such localization exists in the bonnet monkey, and in most of the recent standard works on pathological anatomy and neurology it is stated that it is true for man. But Marie maintains he is not able to corroborate such teaching and the writer's experience leads him to agree with this. The teaching that there is definite capsular localization has been largely the outcome of the acceptance of statements of Charcot and his school, and of homologizing in man the results of experiments on the lower animals. It is impossible to say that there is a special territory of the internal capsule of man that is exclusively sensory, and even with intense capsular and cortical lesion, hemianæsthesia may be absent.

Marie maintains that the motor pathway occupies the entire posterior segment of the internal capsule as far as the level of the posterior angle of the lenticular nucleus. In this lenticulo-optic territory, in which descend motor fibers, it is impossible from clinical data to specify distinct territories for the fascicles that go to the arm, leg, foot, etc. A lesion of this part of the capsule produces the syndrome of hemiplegia, and that is all, and, conversely, a lesion limited to the internal capsule causes degeneration of the entire pyramidal tract at the level of the oblongata or the cord. Clinically one cannot establish any persistent segmentary localization in the internal capsule. Very small lesions of the posterior limb of the internal capsule may produce irritative symptoms manifest as contracture and irregular movements on purposeful effort. After a considerable vascular lesion has been partly absorbed, the destruction of the pathway will be manifest by hemiplegia and the irritative effect on the partially destroyed fibers may be evidenced by contracture. When the lenticulo-optic section of the internal capsule is diseased, there may be a flaccid, complete hemiplegia with, later, hemicontracture. Conjugate deviation of the eyes associated with forced movements of the head is sometimes an initial symptom. If the part of the

internal capsule just anterior to the knee is affected, there will be hemiplegia, anarthria, and dysarthria, or, if it is a small area in front of the knee, facial paralysis alone may be the symptom. A noteworthy feature in these cases is that the upper branch of the facial is not involved, the reason being that the cortical centres for the face are bilateral.

Occasionally a focus in the lenticular nucleus or in the optic thalamus encroaches upon the internal capsule in such a way as to produce a characteristic clinical picture, paralysis of the face and of the leg, while the arm is spared. In such cases the lenticulostriate and the retrolenticular section of the internal capsule are affected, while the lenticulo-optic section remains comparatively free.

Double-sided lesion in the internal capsule, like certain bilateral cortical lesions, produces the condition known as pseudo-bulbar paralysis, manifest by impairment of speech, dysarthria, impaired phonation, peculiar disorder of gait, disturbances of swallowing and respiration, and, varying with the encroachment upon the pyramidal tract, hemiplegia of one or both sides.

**Lesion of the Corpora Striata (Ganglia of the Cerebral Hemispheres); the Lenticular Nucleus and the Lenticular Zone.**—The corpora striata, the greater part of which are embedded in the white substance of the hemisphere, are constituted of two parts, an intraventricular part, called the caudate nucleus, and an extraventricular, called the lenticular nucleus. The functions of this body have not been satisfactorily established. The symptoms that accompany lesion of it are not very definite, as the lesion is rarely confined to it, nor are the results of experimental lesion constant. Embryologists and anatomists are unable to agree as to the constitution of the striate bodies, particularly the lenticular nucleus which constitutes the most important part. Dejerine denies that it has any connection with the motor and other portions of the cortex or that it plays a part in the control of movement or in the function of speech. Mingazzini, on the other hand, has proved conclusively that certain parts of the lenticula transmit motor and sensory fibers and have motor and sensory functions. A focus of disease, even of small size, involving only the lenticular nucleus, never fails to cause motor disorder usually partial paralysis, to which, sometimes, are added irritative symptoms. Motor paresis of the entire half of the body, often of slight intensity and therefore overlooked, is the most frequent symptom. This often disappears, but a residue can always be found by careful examination. The upper branch of the facial is rarely involved, and the upper extremity assumes peculiar attitudes. Disturbance of speech is also often associated with lesions of the left lenticula. The lesion in the majority of cases with the syndrome of hemiparesis has been softening of the putamen. The motor functions of the lenticula are supplementary to the pyramidal tract. These conclusions of Mingazzini probably approximate the truth concerning the functions of the lenticula. There is no justification for regarding it as a vestigial organ, and Mills and Spiller have furnished ample proof that destructive lesions of certain portions of the lenticula cause paresis of the limbs or face, which differs from that produced by capsular lesions in so much as the impairment of power is not so severe, and from cortical lesions in that it is less likely to be dissociated.

Marie maintains that the lenticula and the area around it, which he calls the lenticular zone, play an important role in the causation of motor aphasia. There is no doubt that lesion of this zone causes disturbance of speech, the

result of impairment of coördination of the complex muscular movements concerned in the exteriorizing of speech, and apparent motor aphasia must be considered a symptom of disease of the lenticular nucleus.

Certain symptoms, mental and physical, accompany with much constancy disease of the globus pallidus (the inner part of the lenticular nucleus), due to illuminating gas, to which the lenticula is peculiarly vulnerable. These are vasomotor and trophic disturbances of any of the soft parts (discoloration of the skin, ulceration simulating bedsores, etc.), disturbed control of the bladder, very rarely of the bowels, amnesia or loss of memory, especially the variety known as retrograde, and a peculiar mental state, which shows itself most strikingly in the appearance of the patient, reminding one of catatonia. Such patients are fully oriented, apparently appreciative of all that goes on around them, but wholly indifferent, apparently uninterested, reflecting in their features and attitudes absence of initiative.

**Lesion of the Optic Thalamus.**—It has been taught with much confidence that the optic thalamus is a region which manifests disease or encroachment upon it by motor, sensory, secretory, and emotional symptoms. These are hemiplegia, posthemiplegic choreic movements, intention tremor, hemiataxia, disorder of involuntary mimetic movements, hemianæsthesia, hemianopsia, vasomotor and trophic manifestations in the controlateral extremities.

The "thalamic syndrome" has been rather widely accepted. Gradually, however, it has become obvious that there are two reasons why it should be subject to critical investigation: First, because destruction of parts of the optic thalamus by disease may occur without symptoms; and second, because many of the cases upon which the claim has been made that lesions of the optic thalamus caused such symptoms have been investigated only macroscopically, and therefore it is impossible to say that the subthalamic region, the red nucleus, and even the retrolenticular constituent of the internal capsule were not affected as well. Finally, many cases of thalamus lesion which did not produce the above-mentioned symptoms have been carefully studied.

Lesions confined to the thalamus do not cause hemiplegia. Although hemiplegia occasionally occurs as an initial and transitory accompaniment of thalamus lesions, its existence is to be interpreted as a coincidence, or as the result of a lesion at a distance. In other instances in which hemiplegia has been present with lesion of the thalamus careful examination of the crura (cerebral peduncles) or the capsule has not been made. Certain other disturbances of motility occur in thalamic lesions, especially lesions of vascular origin. These are so-called posthemiplegic choreic movements, intention tremor, ataxia of purposeful movements, athetoid movements, and disturbance of the involuntary mimetic musculature. Lesion of the pulvinar and of the lateral geniculate body causes spasticity of the contralateral extremities and ataxia or tremor especially of the upper extremity. In some instances the movement which accompanies such lesion is more or less deliberate and rhythmical, known as athetoid. The term athetosis, meaning without fixed position, was introduced by William A. Hammond to describe slow, apparently determinate systematic movement with a tendency to distortion of an extremity or part of an extremity, such as the hand or fingers. Athetosis is the result of thalamic lesion and not of lesion of the corpus striatum, as was at one time believed. Choreic movements, ataxia, and intention tremor may result from lesion in the cortical motor

pathways, but athetosis does not. In several cases of athetosis recently studied microscopically, lesions of the red nucleus, the tegmentum, and the vicinity have been found.

Disturbed innervation of the muscles that cause involuntary emotional display is perhaps the most constant symptom of lesion of the thalamus. It has long been established by clinical observation that there are patients with slight hemiplegia or hemispasticity and other symptoms indicative of thalamus lesion, such as paræsthesia of one-half of the body, disordered muscle sensibility, choreic or athetoid movements, who have the peculiarity that they cannot innervate the emotional muscles of the face voluntarily, but they are able to smile and weep emotionally. Many of these cases have been carefully observed and studied microscopically, and lesion of the thalamus is invariably found. This symptom constitutes an important feature of the thalamic syndrome. The occurrence of forced smiling and weeping is due to lesion of the corticothalamic fibers, the mimetic centre reacting convulsively to every stimulus whether from the periphery or from the cortex. It should be said that Rousay claims the mimetic capacity was not disordered in the animals in which he produced lesion of these fibers.

The occurrence of sensory symptoms with lesion of the thalamus is by no means constant, although it would seem to be conclusively proved that sensory disturbances may be caused by experimental lesion of the thalamus in the lower animals. Moreover, there are many cases of thalamic disease with sensory disturbances. The chief reason why our knowledge is so unsatisfactory on this point is that in these cases there has not been adequate investigation of the state of the surrounding parts, only possible by microscopic examination. For instance, Dejerine and Long are inclined to interpret the sensory disturbances attending thalamic lesion as evidences of coexisting lesion of the retrolenticular division of the internal capsule. Lesions of the basal portion and of the subthalamic region are undoubtedly attended with sensory disturbances more constantly than lesion of the thalamus elsewhere. The disorder of sensation is usually limited to certain qualities, such as the tactile. Pain and temperature sensibility are sometimes undisturbed. Another feature of the resulting tactile and muscle anæsthesia is that they are not permanent. This feature explains in a measure some of the cases recorded in which lesion of the thalamus was not accompanied by sensory disturbances, and especially cases of tumor which are of slow growth and long duration. In such cases there may have been sensory disturbances in the course of the disease.

Vasomotor and trophic disturbances which occur with thalamic lesion are manifest particularly in the controlateral extremities. They are subjective and objective sensations of warmth or of cold, redness, swelling, and hyperidrosis. Such symptoms have been observed so often with thalamic lesions that many writers maintain the existence of vasomotor centres in the thalamus. Numerous cases have been reported of hemiplegia with sensibility disturbances on the paralyzed side, in which there has been well-marked muscular atrophy on the opposite side, a condition that cannot result from lesion of the pyramidal tract alone. Some interpret this atrophy as the manifestation of a trophic influence, others as a vasomotor disturbance.

The thalamic syndrome may then be considered to consist of slight hemispasticity (which, of course, is a manifestation of co-involvement of some constituent of the pyramidal tract), sensory disturbances of the contralateral

extremities, particularly of tactile and muscle sensibility, often associated with paræsthesia, occasionally with hyperæsthesia and pain, disorder of volitional movement, forced smiling or weeping, hemichorea, tremor, ataxia, athetosis, disorder of the involuntary mimetic capacity, and trophic disorders, such as change of temperature, color, and nutrition. Very rarely are all the components of this syndrome present in any given case. In brief, it may be said that the thalamic syndrome consists of (1) a persistent partial hemianæsthesia, (2) slight hemiplegia, (3) ataxia and astereognosis of the contralateral half of the body, (4) pains, and (5) choreic and athetotic movements on the hemiplegic side.

Our knowledge of the symptomatology of thalamus lesion is indefinite because lesions limited closely to the thalamus are of rare occurrence, save tumors, and of such slow development and protracted course that compensation occurring meanwhile makes it difficult to say that the symptoms are, indeed, due to thalamic disease. Vascular lesions are rarely confined to the thalamus, and it is not at all improbable that many symptoms produced by them are due to simultaneous disease of the parts of the brain adjacent to the cortex. von Monakow says that the thalamus constitutes an important station within the central nervous apparatus for sensory and cutaneous perception. The representation of individual sensory organs is present in it in a sharply localized way. The final relation of the anatomical connections must be deemed bilateral. Further than this, it must be accepted that other parts of the brain may functionate vicariously for the thalamus. In this way is to be explained the fact that symptoms of focal disease of the thalamus are transitory and that many instances of lesion of this region run their course without symptoms.

**Lesion of the Crura Cerebri, Crusta, and Tegmentum.**—After the motor fibers pass through the internal capsule they leave the cerebral hemisphere by the crus, of which they occupy the middle three-fifths. The axis-cylinder processes of the peripheral motor neurones begin to tap it at the level of the origin of the third nerve. Until the motor tract reaches the oblongata, it continues to give fibers at different levels, to end in the nuclei of the motor cranial nerves supplying the opposite side of the body. The motor tract is represented in the pons and oblongata by the pyramidal tract. In the oblongata the residue of the internal capsule, after the fibers going to the cerebral nerves have been given off, crosses to the opposite side and constitutes the decussation; from this point it gradually diminishes and ends in the lower part of the spinal cord.

Lesion of a crus may be limited to the crusta or to the tegmentum. When confined to the crusta, it causes a characteristic syndrome, paralysis of one-half of the body on the side opposite the lesion, and of one or more of the cranial nerves, usually the third, more rarely the fourth, on the same side.

*Tegmental Syndrome.*—The tegmentum of the crus cerebri, which is made up of a continuation of the formatio reticularis of the oblongata and pons, the superior peduncle of the cerebellum, and other white and gray matter, is sometimes the seat of disease which confines itself rather narrowly to it. Such disease produces fairly constant symptoms, which are said to constitute the "syndrome of the tegmentum." These are alternating sensorimotor hemiplegia, paralysis of the associated movements of elevation and depression of the eyes, of convergence, and disorder of the internal musculature of the eyes with preservation of the associated movements of bilaterality. Such symptoms

may be associated with involuntary movements (in rest and in action), hemiataxia, diadococinesis, hemihypoæsthesia, and abolition of deep sensibility of the opposite side of the body. Raymond has reported a case in which there was in addition to such symptoms atrophy in the domain of the motor root of the fifth nerve on the same side as the ocular palsies. There was no disturbance of sensibility in the distribution of the fifth nerve.

Gruner and Bertolotti have described two typical cases of this affection. The sensory hemiplegia was more marked than the motor, but disorder of voluntary movement, incoördination and asynergia, which superimposed themselves upon the hemiplegia, were well marked. To the hemiplegia was added bilateral and symmetrical paralysis of all the muscles innervated by the third and fourth pairs of cranial nerves. The only conjugate movements preserved were those of laterality. The lesion was a tubercle situated in the middle of the tegmentum at the level of the nucleus of the third nerve. It was bordered on its left side by a zone of softening which reached through the median lemniscus and totally destroyed the anterior quadrigeminal body. It had caused complete destruction of the gray column of the motor nuclei of the aqueduct extending from the level of the third ventricle into the pons as far as the level of the nucleus of the superior oblique.

*Superior Pontile Syndrome.*—Raymond and Cestan describe a superior pontile syndrome characterized by paralysis of the lateral associated movements of the eyes, by paralysis of the arm and leg with the characteristics of a central paralysis, the inferior facial being affected on the same side. The motor symptoms were manifest particularly by trembling, athetosis, incoordination, and asynergia. All forms of sensibility, stereognostic, muscular, articular, and postural, were affected. The eyes were fixed straight ahead, and the pupils reacted, *i. e.*, there was paralysis of the voluntary movement of the sixth pair and paralysis of the associated lateral movements of the eyes. This was associated with preservation of the power to converge and with integrity of the movements of elevating and lowering the eyeballs, which is known in France as the "Syndrome of Parinaud," a quasi-pathognomonic sign of lesion of the superior part of the pons. The lesion was a tubercle of the right half of the pons affecting the median fillet, destroying the fibers going from the cortex to the sixth pair of the fibers which are supposed to unite the nuclei of the third and the sixth pairs.

**Lesion of the Quadrigeminal Bodies; Mid-brain.**—Disease confined closely to the quadrigeminal bodies causes symptoms from which a localizing diagnosis may be made with some confidence, but such disease is comparatively rare, especially hemorrhage and softening, which are of common occurrence in other parts of the mid-brain and hind-brain, but rarely confined to these bodies alone. Tumor, however, is sometimes so confined; it is from consideration of the symptoms accompanying such lesion, and from experimental work, that the symptomatology of disease of this part of the mid-brain has been made out. Experiments on the lower animals seem to show that destruction of the entire roof of the corpora quadrigemina produces complete blindness, and that lesion of one-half of it produces hemianopsia, but in man and the higher animals this does not occur, although in man an important optic nerve bundle is in connection with the anterior corpora quadrigemina. Disturbances of vision, even complete blindness, occurring in affections of the corpora quadrigemina have been described by many authors, but the fact that there are a great number of negative cases and

cases in which there is no disturbance of vision during the entire course makes it certain that other parts of the mid-brain than the quadrigeminal bodies were implicated in those instances in which vision was affected. Profound visual disturbance accompanying such lesions is indicative of associated lesion in the external geniculate bodies or in the optic tract. Ferrier and Turner concluded from their experiments on monkeys that the quadrigeminal bodies have only a subsidiary relation to the special senses of sight and hearing, although it is generally believed that lesion of the posterior quadrigeminal bodies causes defect or abolition of the sense of hearing, and the intimate association which exists anatomically between the posterior quadrigeminal bodies and the trapezoid body by means of the lateral fillet would appear to give foundation to the important relation between these structures and the sense of hearing. They are also of the opinion that the sensory auditory tract lies outside the quadrigeminal bodies, the posterior quadrigeminal body being merely an accessory structure and end-station of the fibers of the lateral fillet; clinically, disturbance of hearing is not noted with sufficient constancy to justify us in placing much reliance upon it as a localizing symptom of disease of the posterior quodrigeminal bodies.

Weinland concludes, from a study of tumors of the corpora quadrigemina, that disease of the posterior corpora quadrigemina causes disturbance of hearing on the contralateral side, and that the diagnosis of quadrigeminal disease is to be made quite positively if, in addition to the symptoms referable to the cerebellum, the eyes, and eventually the eye muscles, there is disturbance of hearing on the contralateral side.

Foci of disease of the quadrigeminal bodies adjacent to the tegmentum or the crura cannot be differentiated from lesions of these structures, but foci in the roof of the quadrigeminal body produce symptoms which are more or less characteristic. These are disturbance of vision, pupillary disturbances, limitation of movements of the eyeball, *i. e.*, some degree of ophthalmoplegia, especially of the oculomotor and trochlear nerves, sensory disturbances, and disorder of voluntary motion and of gait. These symptoms vary with the extent of the lesion and with the implication of the surrounding bodies. This fact cannot be too strongly emphasized.

According to Bach, double-sided complete destruction of these bodies, but more particularly of the roof, causes lesion of the pupillary light reflex on both sides, while unilateral destruction causes it only on one side. But from experiments on the higher animals it seems to have been conclusively proved that there is retention of iridomotor action after complete ablation of the quadrigeminal bodies. This seems to indicate that the pupillary fibers of the optic nerve reach the third cranial nucleus by some other route than through the anterior quadrigeminal bodies.

The truth of the matter is that until a lesion of the corpora quadrigemina extends basalward, involving the tegmentum or the central gray matter around the aqueduct of Sylvius, the location of the lesion can only be suspected, but when it does so extend, fairly characteristic symptoms occur, the most important of which is ophthalmoplegia. A lesion confined narrowly to the corpora quadrigemina, and causing no pressure around the surrounding parts, is not accompanied by paralysis of associated ocular movements. The many cases in which the corpora quadrigemina have been destroyed without causing disturbance in the movements of the eyeballs attest this

statement. Complete ophthalmoplegia is rare, but partial limitation of the associated movements of the eyes, especially the upward and downward movements, is fairly common, as is also ptosis. Ophthalmoplegia in connection with ataxia resembling cerebellar ataxia constitutes a most important association of symptoms. When the ataxia develops first and is very severe, the localization of the lesion is very likely to be in the cerebellum; when ocular palsies develop first, the ataxia much later, the lesion is likely to be in the corpora quadrigemina.

Limitation of the ophthalmoplegia to the oculomotor and the trochlear, speaks in favor of affection of the corpora quadrigemina. Involvement of the abducens may occur both in quadrigeminal and cerebellar lesions, but involvement of other cranial nerves from the abducens downward indicate an affection of the cerebellum.

Paralysis of the trochlear nerve on one or both sides and disturbance of the chewing capacity have been described as symptoms of mid-brain disease, and especially of the posterior quadrigeminal bodies, the reticular formation and the locus cœruleus. Such paralysis is manifest by difficulty of moving the eyes outward and downward and circularly. In the majority of these instances this paralysis is associated with hemiataxia, cerebellar ataxia, choreic movements, and oculomotor palsy. Its occurrence bespeaks a fairly extensive involvement of the central gray matter and of the thalamic region. Difficulty in chewing, pointing to involvement of the descending root of the fifth nerve, in reality a pons symptom, has been mentioned in a number of cases of quadrigeminal disease. Some writers speak of an association of symptoms consisting of atrophy in the region of the neck and shoulder, and later in the muscles of mastication, as the "Syndrome of Kojevnikoff," and maintain that it is dependent upon lesion of the posterior quadrigeminal bodies.

Disease of the mid-brain involving the tegmentum, particularly a vascular lesion, disorder of the functions of the red nucleus, the fillet, the reticular formation, and other pontile connections, is always accompanied by ataxia of a cerebellar nature often confined to one side of the body. In fact, this symptom, in conjunction with irregular choreic movements and intention tremor occurring on the opposite side of the body, is perhaps the most characteristic combination of symptoms of mid-brain disease. The ordinary ataxia of movement is unquestionably dependent upon involvement of the fillet and of the reticular formation. The "cerebellar" ataxia is not so easily explained. In fact, up to this time no satisfactory explanation has been given.

A lesion, such as glioma, affecting the gray matter around the aqueduct of Sylvius at a point midway between the two corpora quadrigemina and the third ventricle, may produce forced movement with a constant tendency to fall backward or to bear backward against a support, loss of the upward movements of the eyeballs and upper eyelids, exaggeration of the knee-jerks, ankle clonus, patellar clonus, and general symptoms of brain tumor. Such a case has been reported by Bruce, and other authors have reported cases very similar in which the lesions were in the inferior vermiform process of the cerebellum, in the optic thalamus, and corpus striatum.

**Lesion of the Cerebellum.**—The symptoms of disease of the cerebellum as a whole are often pathognomonic. The symptoms of disease of definite portions of it are equivocal. Although disease of the cerebellum may be

diagnosed with much certainty, and although it is justifiable to attempt the diagnosis of the nature of the lesion, the diagnosis of disease of a definite portion of the cerebellum cannot be made with certainty. Symptoms of disease of the cerebellum vary profoundly with the suddenness or slowness of the lesion, and without full recognition of this fact the diagnostician is bound to be disappointed frequently. As Hughlings Jackson has very properly said, "the symptoms are so different in different cases that it is nearly impossible to make general statements."

Some of the chief functions of the cerebellum have long been established, but it is only very recently that we know with any certainty that there is definite localization of function in it. The cortex is a massive recipient organ (Flourens, Jackson, Edinger, Horsley), the head ganglion of the proprioceptive system (Sherrington), to which different impulses from all parts of the body converge for rearrangement and coördination, so as to render them suitable for transmission and interpretation to the deep nuclei, which may be looked upon as stations interposed between the different cortical fibers and the rest of the nervous system. The relation of these nuclei to the cortex has been fairly well established by experimentation, but the relation of the nuclei to the extracerebellar nervous system is only now being determined. The chief role of the cerebellum is to collect and arrange the various afferent impulses outside consciousness for other parts of the nervous system. This collection and rearrangement of afferent impulses is manifested in motor coördination, equilibration, and in the maintenance of muscular tonus. The cerebellum is also intercalated into sensory pathways which receive excitation from the periphery and from the higher centres, but its functional relationship to the sensory sphere becomes more doubtful the more experimentation forces the secrets of its purpose. The middle lobe, the worm or vermiform process, is functionally more important than the lateral hemispheres, and removal of half of this lobe in lower animals causes disturbances similar to that caused by removal of a lateral lobe. The effects of unilateral ablation are manifest in disturbance of equilibrium, peculiar attitude, disordered sensibility, and ocular symptoms. In human beings injury and disease of the cerebellum cause similar symptoms and also disorder of speech.

The results of experimental lesion and destruction of portions of the cerebellum are very soon compensated by the sensorimotor cortex, and after a few months animals that have been thus operated upon present no abnormalities unless very careful examination is made. If the Rolandic area of the brain, however, is destroyed, simultaneous compensation does not result, and the cerebellar symptoms persist. In the human being injury to the cerebellum early in life, and arrested development of the cerebellum, may not give rise to any symptoms during adult life, the functions of the cerebellum having been assumed by other portions of the brain.

The disturbance of equilibration caused by experimental destruction of the vermis of a hemisphere is manifest by a tendency of the animal to rotate on its own axis. When the right half of the cerebellum is removed the rotation is toward the right, and vice versa. This phenomenon is not always present, but unsteadiness of gait and profound incoördination of all voluntary movement, invariably result. No true paralysis occurs in cerebellar lesions, but despite this, in animals experimented upon, there seems to be a motor paresis of the limbs on the side on which the operation has been performed. Bruce has suggested that these motor disturbances may possi-

bly find explanation in the affection of the opposite cerebral cortex, which results from the unilateral ablation of the cerebellum. It is possible, however, that impulses from the cerebellum to the muscles of the limbs go by way of a pathway from the cerebellum through Deiter's nucleus to the spinal cord. Cutting off such influences causes the paresis which has been so often noted.

The position of the head in cerebellar disease, to which attention was first called by Hughlings Jackson, is of importance, particularly in conjunction with other cerebellar symptoms. The head is bent toward one side, the face looks upward away from the lesion. The ear opposite the side of the lesion approximates the shoulder. This position of the head, so far as the approximation of the ear to the shoulder is concerned, is the reverse, while the position of the face is the same as that seen after experimental ablation of one lobe of the cerebellum.

In animals there is a spasm in the muscles of the limb on the side of the lesion, and the knee-jerk is increased on the same side, diminished on the other, but this diminution soon disappears, and both knee-jerks often remain exaggerated for a considerable time. Bruce believes that he has been able to make out a diminution of cutaneous sensibility in animals whose cerebellar hemispheres have been injured, which is most marked in the limbs on the side of the lesion. This has not yet been noted in man with cerebellar disease. Displacement of the eyes always occurs from unilateral ablation, both eyes being turned away from the seat of injury, the eye on the opposite side of the lesion usually being more displaced than the one on the same side, and nystagmus is invariably present.

The *symptoms* of cerebellar disease may be divided into those due to injury or irritation of the cerebellum itself, those due to increased intracranial pressure, particularly pons and oblongata symptoms, and those due to sudden changes in the blood supply, which, perhaps, cause seizures.

The constant symptoms are motor asthenia, hypotonus, astasia, titubation, asynergia, and ataxia, the latter having definite features which entitle it to the designation *cerebellar ataxia*, nystagmus, and disordered phonation and articulation. These symptoms constitute a pathognomonic syndrome. Their interpretation has been based largely upon experiments in animals and upon theory. The reeling, reckless gait is so familiar that description is unnecessary. The titubation or apparent constant balancing while the patient is standing is nearly as familiar. Cerebellar asynergia is the most important symptom, as it is the basis of most of the others. Babinski has shown that volitional equilibrium must be considered in two aspects: first, static equilibrium, and equilibrium of the body in a state of movement and that a lesion of the cerebellum may apparently produce the paradoxical result of very materially diminishing kinetic equilibrium. A patient with disease of the cerebellum lying upon his back can hold up one or both lower extremities for several minutes without trace of tremor or swaying, which appear within a minute in a normal person who essays to do it. Babinski has further demonstrated that persons with cerebellar disease have incapacity to make successive movements rapidly, such as pronation and supination of the wrist. The rapid succession of such movements, or of any movements for that matter, involves the faculty of arresting suddenly a motor impulse, *i. e.*, a contraction in a muscle or group of muscles, as well as sending to other muscles a new impulse which will cause them to contract. This faculty is

essential to kinæsthetic equilibrium, and for some reason not yet explicable it is disordered in cerebellar disease. The best way to distinguish this capacity is to have the patient lay his hand flat upon the table and then rapidly to supinate and pronate the hand. Patients with cerebellar disease do it very slowly or not at all. To this sign of cerebellar disease Babinski has given the name diadococinesis (formed from two Greek words, meaning successive and movement).

Tetanus-like seizures occurring in cerebellar disease, especially of the middle lobe, were first described by Jackson. They consist of exaggeration of the "cerebellar attitude" plus tonic spasm in certain groups of the bilateral muscles of the trunk and the extremities which comes on suddenly, lasts a variable time, and is not usua ly attended with loss of consciousness. But motor disorder, aside from asynergia in its various manifestations, is not an important diagnostic symptom of disease of the cerebellum. Cerebellar tremor has nothing characteristic to distinguish it from other intentional tremors. Paralysis and contracture when they occur are the result of ventricular distention or other indirect pressure. The tendon-jerks are altered in cerebellar disease, but not in characteristic fashion. In the majority of instances they are increased on the side of the lesion, but variation occurs during the course of the disease. Cranial nerve paralyses, of commoner occurrence with lesion of the superior cerebellar peduncles than of any other part of the cerebellum, and which are caused indirectly, that is by extension of the lesion, by pressure, and by action at a distance, are usually on the side of the lesion, the facial and auditory nerves being oftenest affected. The sixth, ninth, tenth, eleventh, and twelfth nerves may indicate indirect affection through impaired function. Disorder of sensibility and of the intelligence are not symptoms of uncomplicated cerebellar disease, although they are not infrequent, especially such symptoms as visual hallucinations and motor excitement.

Much animal experimentation has been done to determine the function of certain parts of the cerebellum. Pagano, working on dogs, claims to have identified in the cerebellum several motor centres, which act directly on the cord and not through the intermediation of the contralateral cerebral cortex, and Adamkiewicz is of the opinion that the true centres for voluntary movements lie in the cerebellum. But nothing of clinical importance has as yet resulted from such efforts. A certain amount of differentiation of the seat of the lesion is justifiable, especially of disease of the middle lobe and the vermis from lesion of the lateral lobe. In lesion of the middle lobe and vermis the symptoms are equally distributed on both sides of the body; head retraction and opisthotonos are especially likely to occur. Such lesion is more frequently accompanied by paralysis of the seventh and eighth nerves, the result of indirect pressure. Sudden death in cerebellar disease from pressure upon the oblongata is more likely to occur with disease of the vermis. In lesion of the lateral lobes the muscular asthenia, cerebellar ataxia, hypotonia, asynergia, loss of kinetic equilibrium, increase of static equilibrium, increase of the deep reflexes, are present on both sides, but the symptoms are most pronounced on the side of the lesion. The head is inclined to the side of the lesion, and the face looks up away from the lesion. The patient inclines to the same side in walking and tends to fall to the same side. If rotation is a symptom, he rotates from the supine to the prone position over the side of the lesion.

Vertigo, vomiting, diplopia, inequality of the pupils, nystagmus, slow pulse, exaggeration of the knee-jerks, hearing-defects on the side of the lesion, tendency to fall toward the side of the lesion, hemiataxia and hemiasynergia, and diadococinesis are common symptoms of affection of a superior peduncle. Many writers have described an association of symptoms thought to be characteristic of cerebellar agenesis, atrophy, or sclerosis; in other words, a syndrome of cerebellar deficit. The chief symptom is disorder of motor coördination, manifest in station, gait, and speech. There may be no symptoms whatsoever. Such instances are supposed to be examples of sensorimotor cortex compensation. The usual symptoms, however, are ataxia of "cerebellar" characteristics, with relative integrity of individual movements of the limbs when the patient is supine. He stands with the feet widely separated and legs and trunk betraying oscillations, titubations, which are sometimes so intense that he falls. Ataxia of the upper extremities and general muscular asthenia often exist in these cases. Speech is often scanning or otherwise seriously disordered and nystagmus usually exists. Many symptoms have been described which cannot be ascribed to the disease of the cerebellum, such as epileptic attacks, ocular palsies, and psychical manifestations.

Recently an attempt has been made to classify primary diseases of the cerebellum according to their pathological nature. Mangazzini distinguishes the following varieties: (1) Agenesis and atrophy of the cerebellum; (*a*) unilateral, and (*b*) bilateral; (2) Atrophy of the cerebellum; (*a*) in conjunction with disease of the brain, and (*b*) in conjunction with disease of the spinal cord. Holmes suggests the following: (1) Primary parenchymatous degeneration of the cerebellum; (2) olivopontocerebellar atrophy; (3) progressive cerebellar disease due to vascular or interstitial lesions; (4) acute cerebellar lesions, apart from tumor and focal lesions of vascular origin; (5) degeneration of the spinocerebellar tracts, the cerebellar being normal or small only; (6) cerebellar symptoms associated with a small central nervous system. Under the first are included familial and sporadic cases, occurring at any age and usually slowly progressive, with symptoms constituting the cerebellar syndrome. The morbid anatomy is a primary and progressive degeneration of the nervous elements of the cortex of the cerebellum, especially of the Purkinje cells, and of the fibers springing from the cortex. This reduces the cerebellum in size. There is a consecutive degeneration of the myelinated fibers, the folia and white matter of the cerebellum and proliferation of the neuroglia and the sclerosis of the ground tissue, the result of degeneration of the functional elements of the cerebellum, and complementary to the degenerative nature of the disease.

Olivopontocerebellar atrophy was first described by Thomas. It is characterized clinically by the cerebellar syndrome. It is neither hereditary, familial, nor congenital. It develops in late adult life and its course is progressive. It is characterized anatomically by atrophy of the cerebellar cortex and the bulbar olives, and of the gray matter of the pons; by total degeneration of the middle cerebellar peduncles, by partial degeneration of the restiform bodies, and by relative integrity of the central nuclei of the cerebellum.

Under the third caption are included those cases of cerebellar disease incident to arteriosclerosis and primary interstitial proliferation. There is no doubt that the cerebellar syndrome may be caused by sclerosis of the

bloodvessels of the cerebellum, focal or general, and the consecutive changes in the parenchyma. There are cases of disseminated sclerosis in which the sclerotic tissue is limited to the cerebellum, that may be included under this heading.

Under caption number four are included particularly acute cerebellar inflammation, hemorrhagic or otherwise, and hemorrhages into the cerebellum. It comprises also those cases in which the cerebellar syndrome comes on after acute infectious diseases, such as influenza, measles, and scarlet fever, especially in children with or without symptoms indicative of focal or disseminated encephalitis. This occurrence, of which there can be no doubt whatsoever, it is important to recognize. The fact that it may end in recovery must also be kept in mind. The relationship of this form of cerebellar disease to alcoholism, primary anæmia, and toxic states has yet to be established.

Under the fifth heading are some of the cases (such as those of Sanger Brown), which Marie utilized to support the existence of hereditary cerebellar ataxia, and, whatever justification there is for adhering to the designation as a clinical convenience, it is given by these cases, in the majority of which there is no disease of the cerebellum. And as the hereditary feature is lacking in some cases, the sooner the designation is given up the better the outlook for interpreting disease of the cerebellum. There are many cases in the literature that come under the sixth heading and the anatomical findings have been abundantly described.

**The Symptom Complex of Occlusion of the Posterior Inferior Cerebellar Artery.**—Occlusion of this vessel causes symptoms so sharply defined that the diagnosis can be made with much certainty. The symptoms are manifold, usually coming on abruptly without disorder of consciousness. They consist of slight motor weakness of the limbs, analgesia and thermoanæsthesia in the limbs opposite the lesion, and in the distribution of the fifth nerve on the side of the lesion, usually associated with pain or paræsthesia in the area in which objective sensory disturbances exist. Tactile and postural sensibility are usually attacked. Ataxia in the limbs on the side of the lesion, which bespeaks involvement of the restiform body or the cerebello-olivary fibers, is of very constant occurrence. With this there is often an inclination for the head to drop to the side of the lesion, and in some instances the patient tends to fall toward the same side. In addition to this there are symptoms indicating disturbances of Deiter's nucleus, the vestibular, cochlear, and vagus nerves, such as Ménière's syndrome, vertigo, vomiting, and auditory hallucinations. Paralysis of the muscles of deglutition and of the soft palate on the side of the lesion, which causes inability to swallow with impaired sensation of the pharynx, is of frequent occurrence in these cases. The larynx is sometimes paralyzed on the side of the lesion, which causes disturbance of phonation. The sixth and seventh cranial nerves are rarely affected on the side of the lesion. Symptoms indicating disturbances of the sympathetic nerve supply are smallness of the pupil, narrowing of the palpebral fissure, retraction of the eyeball, and anhidrosis, all on the side of the lesion. Occasionally obstinate hiccough and tachycardia occur. Hemiasynergy and loss of the tendon reflexes on the side of the lesion may likewise exist.

**Lesion in the Pontomedullocerebellar Space.**—Lesion of the cerebellopontine angle, or of the space above designated, usually a tumor spring-

ing from the eighth nerve, occasionally from the fifth, seventh, and other bulbar nerves, and sometimes the accompaniment of a central neurofibromatosis, produces more or less characteristic symptoms referable to a single cranial nerve, especially the eighth or fifth, which usually long antedate symptoms characteristic of disease of the brain stem or cerebellum. Such symptoms are tinnitus aurium, Ménière's syndrome, auditory paræsthesia, occipital headache, pain in the domain of the fifth nerve, and rarely facial paralysis. These symptoms are in many instances indefinite and not infrequently exist for years without incapacitating the patient. Encroachment upon neighboring organs is manifest by cerebellar ataxia, nystagmus, and occasionally paralysis of associated ocular movements, dysarthria and dysphagia. Hemiplegia may occur, usually on the side of the tumor, but it has been observed on the opposite side. The headache is by no means always occipital; frontal and crossed frontal headache have been noted. Circulatory, respiratory, and vasomotor disturbances have likewise occurred.

**Lesion of the Pons.**—Disease of the pons, both acute and chronic, is of frequent occurrence compared with disease of other parts of the brain. Inflammatory processes, acute and chronic softenings, and new-growths occur there and produce symptoms interpretation of which leads to a topical diagnosis. The symptoms vary with the site of the lesion, its size, its intensity, and the rapidity with which it develops. A slowly growing lesion, such as tumor, often exists for a long time without definite symptoms, while, on the other hand, hemorrhage produces not only grave symptoms of pons destruction, but symptoms at a distance, which complicate the clinical picture.

The dorsal part of the pons, which contains the chief nuclei of origin of the sixth, seventh, and eighth nerves, the posterior longitudinal bundle, and not far from this ventrally, the superior olivary nucleus and the motor nucleus of the fifth nerve, and toward the mid-line the fillet, is the commonest site of disease in this region. The adjacency of the pons to the corpora quadrigemina, the restiform bodies, and the superior peduncles of the cerebellum explains why symptoms indicative of disturbed function in other parts of the mid-brain are often added to those of pontile disease alone.

Disease of the pons is characterized by fairly definite symptoms, and in the books the "pontile syndrome" receives full consideration. It is true that lesion of certain parts of the pons produces a more or less typical symptom complex of crossed paralysis, *i. e.*, paralysis of one side of the face, which may include the tongue, some of the eye muscles, the abducens and motor oculi, and of the other side of the body, but disease of the pons covering a considerable area occurs without producing any recognizable symptoms. On the other hand, there are many instances recorded in which disease of the pons has been accompanied by a complex of symptoms almost impossible to interpret. The explanation of such apparent contradiction is to be found in the fact that the pons is the medium of transmission of many important pathways between the brain and the surface of the body, and of many pathways between different components of the brain, as well as the apparent origin of several of the cranial nerves and the termination of many nerve fibers (nuclei pontis) whose functions have not been definitely determined. Although these constituents are contained within a relatively small area, foci of the disease may be microscopic and produce extremely limited symptoms or no symptoms at all. On the other hand, a lesion that affects one-half

of the pons, extending from the dorsal to the ventral border to include the pyramidal tract, may produce symptoms indicative of implication of the third, fifth, sixth, seventh, and eighth nerves, disturbance or paralysis of associated lateral movements of the eyeballs indicating lesion of the posterior longitudinal bundle, disturbances of sensation, which may reach complete hemianæsthesia due to destruction of the fillet and the ascending root of the fifth nerve, and motor paralysis due to destruction of the pyramidal tract. In addition to such symptoms, lesions of the pons adjacent to the oblongata are accompanied by profound vasomotor symptoms, flushing of the skin and hyperidrosis, which indicate that this part of the pons has an important representation for the sympathetic nervous system. Experimental destruction of half the pons in lower animals, even though it does not completely destroy it, is accompanied by convulsions and coma, and similar lesions occurring from disease in man produce similar symptoms.

When we consider that in addition to such symptoms as those above enumerated, disease of the pons of slow growth, such as tumor, causes increased intracranial pressure resulting in headache, vertigo, nausea and vomiting, choked disks, and mental disturbances, and that acute lesion such as encephalitis and acute softenings are accompanied by symptoms indicative of disturbances in other parts of the brain in addition to that which manifests the focus of the disease, some idea will be gained of the difficulty of making satisfactory localizing diagnosis in many cases. The occurrence of alternating hemiplegia will naturally establish the diagnosis of pontile disease, but these localizable lesions represent only a small number of the cases of pons affection.

An alternating hemiplegia is hemiplegia of one side of the face and the opposite side of the body. It is of two types, inferior and superior. When there is paralysis of the abducens or facial nerve on one side, and of the extremities on the opposite side, it is called the inferior type, whereas if the hemiplegia is associated with an oculomotor paralysis on the opposite side, it is spoken of as the superior type. Alternate hemiplegia was first described by Millard and by Gubler, and is frequently known as the Millard-Gubler type of paralysis. Alternating paralysis of the inferior type is sometimes complicated with involvement of the tongue, and dysarthria is a conspicuous symptom. There is difficulty in articulation, and in some instances difficulty in swallowing. These symptoms constitute pseudo-bulbar paralysis. If disturbance in the associated movements of the eyes on one side is added to such a clinical picture, the topical diagnosis is fairly certain, because such symptoms indicate that the lesion involves the posterior longitudinal bundle. In such cases the lesion must be predominantly of the dorsal part of the pons.

Absence of hemiplegia, be it alternating or otherwise, does not say positively that there is no lesion of the pons. Partial or complete hemianæsthesia hemiataxia, cerebellar ataxia without hemiplegia, may occur with lesion of the pons, and may be deemed to be of such a nature when such symptoms are associated with paralysis of one of the motor cranial nerves that have their origin in the pons or the ascending root of the fifth nerve, causing alternating hemiplegia, insensitiveness of the cornea, and half-sided disturbances in the associated movements of the eyes, *i. e.*, a pure conjugate paralysis of the muscles that move the eyes laterally. Lesion of the pons may cause motor hemiplegia with no other symptoms when the lesion is in

the ventral part of the pons, of small size, and confined narrowly to the pyramids. It is said that such pontile hemiplegia causes more profound involvement of the trunk muscles than capsular hemiplegia, and that the face is usually spared.

A lesion developing ventrally in the pons may, by pressure upon the posterior part of the oculomotor nuclei, cause paralysis of associated upward movement as a result of injury to the nuclei over the superior rectus and inferior oblique muscles. Paralysis of downward associated movements depends on impairment of the inferior rectus and the superior oblique muscle, which have cells of origin in two distinct nuclei. A lesion of the nuclei of the inferior rectus muscles and of the fibers connecting them with the nuclei of the trochlear nerves causes paralysis of associated downward movements.

Conjugate deviation of the eyeballs from pontile lesion is uncommon when the lesion is confined to one side of the pons. It has been stated repeatedly that conjugate deviations of the eyeballs away from the site of the lesion is common in unilateral pontile lesion. It is probable that in most cases the side of the pons opposite to the lesion is directly or indirectly involved in these cases. Pons lesions almost always cause pupillary symptoms—narrowing of the pupils, sluggish light reaction—and tonic convulsions in all extremities, also in the face, with the exception of those areas which are innervated by nerves coming from the pons whose angle or whose intrapontile course has been destroyed or disturbed by the lesion.

# CHAPTER VIII.

## APHASIA.

By JOSEPH COLLINS, M.D.

The most interesting association of symptoms in the topical diagnosis of disease of the brain is that known as aphasia. The externalization of thought in any form, and its communication to another is a psychophysical act of great complexity. When this capacity is disordered or destroyed in an individual, the phenomenon that accompanies such disorder or destruction is likewise most complete. Aside from the higher psychical faculties, which it may be assumed lower animals do not have, speech is the distinctive possession of man in contrast with the brute. All of the other functions of the brain can be determined by experimentation upon the lower animals. The principal facts of brain localization have been so determined and later correlated with the symptoms produced by disease of different areas of the brain. In attempts to determine the localization of the speech faculty we have had to rely entirely upon the latter mode of investigation. The result has been that more has been taken for granted on inadequate evidence or insufficient proof in the localization of the speech faculty than in the localization of any other distinctive function, such as vision, audition, motion, and sensation. The present-day wondrous knowledge of the allotment of highly specialized function to definite portions of the brain cortex is the direct outgrowth of the attributed localization of the speech faculty to the third frontal convolution by Broca.

Aphasia is a term used to indicate any disturbance or perversion of intellectual expression. It includes all defects or disorders of intellectual expression, whether the result of disarrangement or destruction of the receptive or of the emissive components of the speech mechanism, or of anything which may be employed as the substitute or equivalent of speech. It may be the result of conditions by which the patient is unable to part with the expressive equivalent of an idea which has been properly formed. Or it may be caused by any condition that interferes with the reception of impulses or stimuli that enter into the genesis of ideas used in the construction of internal or external language. As movement in some form is requisite for the manifestation of all expressions, defect of this is the condition to which the term motor aphasia or aphasia of emission has been and is still often applied.

In the second form of aphasia the sufferer is unable to adapt receptive communications and make them fit the idea represented by the verbal symbol, auditory or visual; that is, he has lost the faculty of adapting the complement of the word to his own idea; it matters not whether these words be spoken or written, or communicated by some equivalent, such as music and pantomime. In a general way, this is aphasia of reception or sensory aphasia.

At the present day aphasia is earnestly being discussed by neurologists

throughout the world, and there seems to be nothing approaching unanimity of opinion as to some of the most cardinal principles of the classic teaching on the subject. Therefore a classification of aphasia at this time should not be attempted.

More than ten years ago, in *The Faculty of Speech, A Study of Aphasia*, the writer suggested that aphasia might be classified as follows:

1. *True Aphasia.*—Aphasia of apperception. Due to lesion of any constituent of the cortical speech area, constituting the zone of language.

2. *Sensory Aphasia.*—Due to lesion of the central and peripheral sensory pathways leading to the zone of language.

3. *Motor Aphasia.*—Due to lesion of the motor pathways, over which the motor impulses travel, in passing to the peripheral speech musculature.

4. *Compound Aphasia.*—Any combination of two or more of these.

If this classification were adopted, it would simplify the matter enormously, inasmuch as there would be in reality only one aphasia; moreover, it would harmonize with the clinical manifestations. It will be seen that the scheme is not unlike that which has been set forth and advocated by Marie. He denies No. 2 and calls No. 3 anarthria or aphemia. A brief sketch of the history of aphasia will facilitate an understanding of this difficult subject. There are three important epochs.

1. The publication in 1861, by Broca, of an example of aphasia and a description of the brain of the individual who had manifested the aphasia, which seemed to prove that the lesion upon which the speechlessness depended was of the posterior part of the third frontal convolution.

2. The classical work of Wernicke, in 1874, which demonstrated beyond a doubt that lesion of the first temporal convolution caused a definite symptom complex, from that time described as "sensory aphasia." Bastian had anticipated Wernicke in many of his contentions and conclusions.

3. The papers of Pierre Marie, entitled "Revision of the Question of Aphasia" in 1906.

The number of contributions since Marie's remarkable paper is enormous. Until its appearance, very little really new had been contributed since the time of Wernicke. Following Trousseau's attempts to harmonize the clinical manifestations with Broca's contention, Charcot introduced the diagram to explain the genesis of speech and the mode of its dissolution in aphasia. But it was not until the Germans took up the diagrammatic method that it became so intricate and complex, and so out of harmony with the clinical facts. During the past twenty years, as brain anatomy and physiology have become better known, diagrams have gradually been supplanted by facts determined by clinical observation and postmortem examination. Until Marie took up the subject of aphasia, the accepted teaching was that aphasia was the result of lesion of the zone of language.

**The Zone of Language.**—This is the name given to the area of the brain in which are carried on the processes essential to speech and its components. This zone is not, in all probability, strictly delimited. It varies in individual cases, and at different periods of life in the same individual, *i. e.*, it is susceptible to phylogenetic, and to ontogenetic variation, the latter depending somewhat on the speech acquisition of the individual, and on the range and number of avenues by which he receives or has schooled himself to receive information of objects of environment and of abstract knowledge.

The speech area, or zone of language, is an area made up of neurons, some of which send their axons into the Rolandic region and into the frontal regions of the brain, while others confine their distribution to the speech area itself; and, as they do not pass outside of this area, they may be looked upon as intercentral neurons.

It must still be maintained that the centres of speech in the area of language are three in number: the centre for auditory memories, the centre for visual memories, and the centre for articulatory kinæsthetic memories. The latter is often called the centre for motor memories. These three centres have a very definite localization, and their position is of great ontogenetic importance. The centre in which are stored the memories of articulation is situated in the third frontal convolution, immediately adjacent to that portion of the Rolandic cortex the cells of which give origin to the projection fibers going to the tongue, the lips, and the larynx; that is, to the parts which supply the peripheral mechanism of articulate speech. The centre in which are stored the visual images is situated in a definite part of the inferior parietal lobule, *i. e.*, its posterior portion known as the angular gyrus. The calcarine cortex is the percipient cortical visual centre. It embraces part of the dorsal lip, the bottom and the ventral lip of the calcarine fissure from the region of the peduncle of the cuneus to the posterior lip of the retrocalcarine fissure. It extends on the lingual gyrus and on the lower part of the first occipital gyrus. The geniculocalcarine path is its afferent radiation. If we have in mind the afferent radiation or central projections of the optic tract after they leave the external geniculate body, the inferior quadrigeminal body, and the pulvinar of the thalamus, until they reach the lingual and fusiform lobules bordering the calcarine fissure, we shall appreciate that the angular gyrus is the most direct, the most adjacent, and the most elective place in which the visual images could be stored. In fact, its relationship to the primary visual centre and to the fibers that convey visual impulses, the radiations of Gratiolet, is analogous to the environmental relationship of the centre for articulatory memories and the Rolandic cortex that externalizes speech. The third centre, the auditory centre, the most important of all the speech centres, is situated between these two centres in the zone of language, and occupies the first temporal convolution, and particularly that portion of the cortex which surrounds the temporoparietal sulcus. The centre for the storage of auditory memories is in a definite part of the general auditory area, in the posterior part of the first temporal convolution immediately adjacent to the gyrus in which are stored visual memories. Thus, it will be seen that the auditory and visual memories, which are contributory to the development of speech and education in general, are not widely separated; they are, on the contrary, immediately adjacent, constituting one continuous area.

Marie maintains that there is absolutely no trustworthy clinical observation that substantiates the claim that the first temporal convolution is the centre of hearing, but his views on this phase of the matter cannot be accepted. Probably there is no fixed spot in the first temporal convolution that is the sole repository of auditory memories. The cortical auditory area may vary in different persons, but anatomical researches after the method of Flechsig by that investigator, by Brodman, Meyer, and others, demonstrate conclusively that the transverse temporal gyri and the adjoining part of the first temporal convolution are decidedly richer in acoustic fibers and have a

distinct connection with the internal geniculate body. The economy of Nature is such that anatomical possession entails functional endowment.

Flechsig has shown that in the fetus, when it is about 50 cm. in length, there is a myelinated tract of fibers going from the internal capsule and the lenticular nucleus into the medullary layer of the "anterior transverse convolution" of the temporal lobe; that is, into the convolution which forms, so to say, the tip of the temporal lobe on the lateral surface. It merges into the first, second, and third temporal convolutions. This tract of medullary fibers he calls the primary projection system of the anterior transverse convolution. Corpus callosum fibers and arcuate fibers are at this period of development not medullated. They get their medullary sheaths later, and it is then very difficult, if not impossible, to distinguish the radiation of the primary bundles of the corona radiata of the anterior transverse convolution from the cortex. But at a time when the corona radiata fibers only are medullated, it is to be seen with great distinctness that the majority of them radiate into the inner two-thirds of the transverse convolution. The outer third of this convolution receives far fewer of them, and only a few scattered fibers go into the outer, free-lying portion of the first temporal convolution. Downward, this projection bundle of the corona radiata can be traced in part directly to the internal geniculate body.

The projection system of the anterior transverse convolution represents the uppermost link of the acoustic projection system, *i.e.*, of that part in which the internal geniculate body is intercalated, or, in other words, the system of the cochlear nerve. Flechsig, therefore, calls this primary projection system the "Auditory Radiation," *i. e.*, that part of the corona radiata which serves the conduction of auditory impressions to the cortex. In confirmation of this view, he adduces the clinical observation that destruction of the left anterior transverse convolution causes permanent word-deafness, whereas total anacusis has been observed only in cases of destruction of the region of the anterior transverse convolution on *both* sides.

It is folly to speak dogmatically at the present time on the relationship of sensory aphasia and its various clinical components to lesion of the auditory receiving station. Until there is a record of many cases carefully observed clinically, and until we are fortunate enough to encounter cases in which the symptoms carefully noted are found to depend upon small and single lesion, such discussion as that recently carried on by Moutier, defending the Marie theory, and Ladame and Monakow, will happen and lead to nothing. The relationship of the auditory word-perceiving centre to the word-elaborating area is probably a close one, but just what it is cannot be said dogmatically. Destruction of the left anterior transverse convolution produces in right-handed persons word deafness. Whether it might be affected to such a slight extent as to cause partial word deafness and such slight affection of internal and articulate language as to be detected only with difficulty seems quite probable.

**Clinical Classification.**—It has been customary to divide aphasia into (1) motor aphasia, (2) sensory aphasia, (3) total aphasia. Each of these are further subdivided, the second into auditory and visual aphasia. The first two varieties have always been considered distinct, although it has been generally admitted that it is not always easy to distinguish one variety from another. For instance, as said in the book above mentioned: "The lesion that causes motor aphasia is usually of the left middle cerebral artery,

which, being the principal blood supply for the remainder of the speech area, it is only in exceptional instances that destruction of Broca's convolution is not accompanied by anatomical and functional disturbance of the entire zone of language." Anatomically speaking, sensory aphasia might be defined as aphasia due to a lesion of the posterior part of the area of language, and cortical motor aphasia as due to the anterior end of this zone. The subcortical forms of each variety occur when there is lesion of the pathways which carry impressions into and away from the zone of language.

**Motor Aphasia.**—Motor aphasia has been classified as (*a*) cortical, due to lesion of the foot of the third frontal convolution (Broca's area), which abolishes the "memory images" of articulation, and (*b*) subcortical motor aphasia (pure motor aphasia of Dejerine), due to lesions of the motor pathway over which speech impulses or messages travel to be externalized. Speech is properly formed, but it cannot be produced because of the hiatus that exists in the speech pathway. These two varieties may often be readily distinguished at the bedside. But it must be said that the features, which allow us to distinguish the one from the other, are not quite so absolute and convincing as one might be led to infer from reading works on the subject. Proust and Lichtheim suggested a test for subcortical motor aphasia to prove that patients preserve the memorial notion of the word; that is, that they have in their minds the name of the object, which they are incapable of emitting. To test this deficiency of internal language, the patient who hears a polysyllabic word, or sees and recognizes an object indicated by that word, is asked to press the interlocutor's hand as many times as the word has syllables; and, if not by pressure of the hand, to indicate by some movement the number of syllables; then to indicate by similar pressings the number of letters in the word and the number of letters in the syllables.

Cortical motor aphasia, Broca's aphasia, is characterized by a loss of spontaneous and repeated speech, some disturbance of the capacity to read inarticulately, slight intellectual enfeeblement and by preservation of the capacity to comprehend articulate speech. In some instances interpretation of spoken speech is slightly impaired in true cortical motor aphasia. The peripheral speech mechanism—the tongue, lips, palate, and vocal cords—is in condition to functionate. The only justification for the use of the word motor in this form of aphasia is that the images of articulation are called into being by movement and are externalized by movement. In true cortical motor aphasia there exists the same inability to call into existence the sensory memories of articulation, and thus to make them a part of internal speech, as there is to externalize them in the shape of articulate words. Many of the cases of aphasia in the literature, which are considered to belong in this category, are not of this variety at all, but are examples of subcortical aphasia; that is, disturbance of speech dependent upon interruption of the projection tracts, which convey the articulatory impulses from the cortical area of the peripheral speech mechanism to the peripheral speech apparatus.

Associated with this loss of spontaneous speech there is an inability to repeat words and to read aloud, but the patient comprehends spoken words, oftentimes imperfectly. There is inability to express thoughts in writing, because in writing the motor word representations are always revived by the impulse which travels from the percipient centre (which is either in the visual area of the brain in spontaneous writing, or the auditory speech area

in writing from dictation) through the articulatory kinæsthetic centre to that part of the Rolandic region which guides the mobile part of the body holding the pen. Incapacity to write is proportionate to the amount of derangement of internal language, and it bears a definite relation to the amount of latent or actual visual amnesia of words which every patient with cortical motor aphasia has. In most cases the capacity to write is limited to writing the name and a few other words, such as the age, the address, and the name of the wife or parents, that have been done so habitually, automatically, and frequently that they from a part of the patient's habitual acts, and are done almost reflexly. Writing voluntarily and writing from dictation are practically impossible, yet the patient is able to write from copy.

The patient is unable to call up promptly and vigorously auditory images; that is, he has some word deafness, and in many instances amimia. This word deafness becomes conspicuous when the patient is spoken to abruptly and rapidly. The capacity for articulate expression which some motor aphasics retain is for a few words whose utterance partakes more of the nature of a reflex act or of an emotional possession than it does of a process of intellection. Although in Broca's aphasia the power to make voluntary expressions is usually entirely gone, the loss may be partial. When partial, the power of expression is limited, as a rule, to one or more monosyllabic words. Occasionally patients who are afflicted with complete motor aphasia are able to utter some words of the nature of an oath, which seem to escape from them in a rapid, uncontrollable way, or to ejaculate words expressive of the feelings. Such expressions are not the product of cognition, but of the emotions, and partake of the nature of reflex action. Other patients repeat continually some expression or meaningless word or words. Such recurring utterances are distinctive features of cortical motor aphasia and not of the subcortical variety.

Cortical motor aphasia is sometimes manifest merely by a loss of substantives, amnesia of the names of the things or objects of which the patient tries to speak.

The symptoms of subcortical motor aphasia are practically the same as those of Broca's aphasia, with two important exceptions; the patient retains the capacity to write and to read, and he responds to the Proust-Lichtheim. In other words, the patient with subcortical motor aphasia retains the capacity to talk to himself, to speak without words. He hears, sees, writes, mimics, and in other ways gives evidence of intellectual integrity and internal language. He is incapable only of articulate speech. This may not be entirely lost. Indeed, it varies through dysarthria, dysrhythmia, up to complete anarthria and arhythmia, and thus to complete speechlessness. In the conventional usage of the term this is aphasia. But it is not true aphasia, for true aphasia occurs only with lesion of the area of language, and is invariably attended with disturbance of internal language.

**Sensory Aphasia.**—Sensory aphasia is the designation given to the speech disturbances or imperfections of language due to lesion of the posterior part of the speech area, more particularly the temporal and angular gyri; that is, to lesion of the perceptive areas of the brain and the immediate incoming special sense and commissural pathways of such areas. The perceptive centres by whose functioning speech is ontogenetically developed are the auditory and the visual, and sensory aphasia is thus practically auditory and visual aphasia. It may be defined as loss of the understanding

of words due to interference with the formation of associations necessary for complete perception. Sensory aphasia differs materially from motor aphasia. In the first place it is not usually accompanied by hemiplegia unless the underlying lesion is very extensive. It is the aphasia of comparative speechfulness in contrast with the speechlessness of motor aphasia. It is characterized by logorrhœa, motor aphasia by alogia. The course of sensory aphasia is rather typical. The patient starts with senseless loquacity and gradually his vocabulary shrinks, often reaching mutism in cases in which the auditory area is completely destroyed.

When the lesion of the auditory centre is slight, there is inability to use words with their proper signification and paraphasia may be the distinctive symptom. Jargon-aphasia, or the production of a jumble of words all forged into one, the syllables of which may be articulated, but the words have no similarity to words as usually spoken, may be looked upon as an extension of paraphasia.

The degree to which spontaneous writing may be preserved or lost in sensory aphasia varies with the patient and with the seat and intensity of lesion. Incapacity to write is most striking when word blindness is prominent and total agraphia always accompanies destruction of the angular gyrus. If the lesion is predominantly of the auditory area, there is inability to write from dictation. Spontaneous writing may be preserved to a limited extent, but the output is senseless and disordered. The capacity to copy is preserved, but the patient in copying makes an exact reproduction of what is before him. This is especially true of cases in which there is lesion of the angular gyrus.

In sensory aphasia with word deafness there is inability to understand spoken words. This is dependent apparently upon the total loss of auditory verbal memories. It is one of the uncommonest forms of aphasia, and it rarely occurs independently, being frequently associated with some degree of word blindness or manifestations of Broca's aphasia. In sensory aphasia with word deafness the patient hears the voice with which the words are spoken, but they contain no meaning to him. He often recognizes the significance of other sounds, unless it be that the memory pictures of such sounds are also lost. Naturally these different degrees of word deafness depend upon the extent of the lesion or the destruction of the auditory area. The appearance of such patients is very significant. They are quiet and observant; their glance betrays suspicion or fear, and their demeanor is often one of restlessness. This alteration of manner, the inability to repeat what is said to them, and the profound diminution of spontaneous speech often cause them to be looked upon as dements.

To the form of aphasia in which there is deafness for musical notes the designation tone deafness is given. Musical deafness is almost always associated with word deafness, but there have been a few cases recorded in which it occurred apart from the latter. Many attempts have been made to classify the clinical varieties of amusia, but to no purpose.

One of the commonest manifestations of sensory aphasia is loss of the verbal memory of written and printed symbols—word blindness. It is of constant occurrence when the lesion is of the posterior part of the area of language. The visual area is made up of a perceptive centre situated on the mesal surface of the occipital lobe along the calcarine fissure, and of the centre in which is "stored" the visual memories of words, other graphic

symbols and probably of objects, situated in the posterior portion of the inferior parietal lobule, the angular gyrus, and the adjacent margin of the supramarginal convolution, which curves over the posterior extremity of the fissure of Sylvius. It must be admitted that the localization of the "visual word centre" is based chiefly on theory and slightly on facts. Destruction of this centre causes word blindness or alexia and agraphia, but no loss of visual acuteness. The primary visual area and the higher visual centre are frequently diseased simultaneously, and when this occurs homonymous hemianopsia due to lesion of the primary visual area is superadded to the word blindness. There are various degrees of intensity of word blindness in sensory aphasia. The patient may be unable to read words and yet retain the faculty of recognizing letters, or he may be able to recognize letters and be unable to join them in syllables—asyllabia it is called. The patient may not be blind to all forms of notation, graphic and symbolic representation. Thus there may be sensory amusia, sensory asymbolia, sensory amimia, etc. In many cases with word blindness there is preserved the capacity to recognize certain familiar words, such as the patient's name or words that he has been accustomed to see frequently. There are also cases in which numbers are recognized, but letters are not. Occasionally, we encounter cases in which the verbal blindness is so slight that it requires careful and persistent examination to reveal it.

If the angular gyrus is completely destroyed the patient has agraphia. In those cases in which voluntary writing is preserved, the lesion involves the primary visual centre, and the patient has right homonymous hemianopsia; the patient begins to write at the extreme left side of the sheet and stops in the middle of the page. The patients, being unable to read what they have written, are unconscious of any errors in spelling or phraseology that they may make, although they put the words on paper in an orderly fashion. Patients with word blindness are sometimes able to read written or printed words by tracing the word with the index finger or a pencil, thus substituting kinæsthetic memories for visual memories, which kinæsthetic memories revive the mental concept of the word.

There has been a very great amount of analysis of visual aphasia, as that form of sensory aphasia in which word blindness predominates is often called, and much speculation has been indulged in as to the pathological physiology and psychology of these subdivisions. There is a variety to which the name verbal amnesia or psychic blindness is given. The patient interprets letters as letters and words as words, and he can read them and copy them, but they convey no meaning to him. When they are pronounced by someone else he hears and interprets them readily, but he has no idea that they are the same words that he has been reading or copying. The lesion which it is supposed produces such a condition is one that interferes with the pathway that conveys the sensation from where the memory of the printed word or object is stored to the place where the idea is formed, if there is any such special place.

Another variety is that to which the name optic aphasia has been given by Freund. The patient, on looking at an object which he has previously seen or used, is unable to call up its name, although he is able to utter it when it is recalled for him. The lesion is supposed to be one that interrupts the pathways that unite the seat of cortical visual representation and the seat of cortical auditory memories.

A third variety is that to which the name of psychic blindness or mind blindness is given. It is the Seelenblindheit of the Germans and the cécité psychique of the French. In this condition the patient not only does not recognize the significance of letters, but he loses the power to differentiate between objects or persons and to distinguish the use of things; in other words, it is word blindness plus apraxia. The word apraxia is coming to have, or it may be said has come to have, a wider application than was formerly given to it. The term was used to indicate inability to comprehend the usage of ordinary objects and things to which one has been accustomed. It was thought to be due to the abolition of the visual memories of objects which are stored in the cortex of the parietal lobe adjacent to the centre for verbal memories. The word apraxia is now used to indicate inability to perform certain familiar purposeful movements by one who has neither paralysis nor ataxia. Apraxia may occur without aphasia. And according to the accepted doctrines of aphasia of today, aphasia may occur without apraxia, but in this connection it must be said that if Marie's ideas of aphasia are accepted, and it is likely that with some modifications and extensions they will eventually be accepted, so-called motor aphasia or the aphasia of Broca will be looked upon as a form of apraxia, that is, apraxia of the speech musculature.

It is customary to describe a subcortical form of sensory aphasia analogous to the subcortical form of motor aphasia, although its delineation is not so distinct. If we divide sensory aphasia into auditory and visual aphasia, we have to divide subcortical and sensory aphasia in the same way, for anything that causes an interruption of the visual and auditory pathways subcortically will give rise to these varieties of aphasia. Subcortical word deafness is characterized by inability to understand spoken words and by inability to write from dictation. It is distinguished from cortical auditory aphasia by the preservation of spontaneous speech and by the ability of the patient to read aloud, to copy, and to write. As in every other form of subcortical aphasia internal language is intact.

The symptoms of subcortical visual aphasia vary somewhat with the seat of the lesion, that is, with its proximity to the angular gyrus. As a rule, there is word blindness, usually partial, associated with right lateral homonymous hemianopsia. Spontaneous speech is usually well preserved. The patient is able to write voluntarily and from dictation, but he cannot read what is written either by himself or by others. There is very slight or no word deafness.

In brief, then, we have two distinct aphasias—motor aphasia (Broca) and sensory aphasia (Wernicke); two subdivisions of each of them, and total aphasia, when the two chief varieties occur simultaneously. A patient with Broca's aphasia cannot speak, or is reduced to a word or two, he writes as badly as he speaks, and he often has some word deafness. He cannot talk to himself, and he cannot read to himself. The patient with sensory aphasia, on the other hand, is a babbler. He uses words senselessly, jargon-aphasia, or in a perverse way, paraphasia. There is marked difficulty of comprehension of spoken or written language and profound disorder of reading and writing. In Broca's aphasia the trouble is predominantly of articulation and of writing, that is to say, in the emission of words; in sensory aphasia all the elements of language are disordered.

Broca's aphasia is therefore the suppression or profound alteration of the speech not dependent upon paralysis, associated with trouble of internal

language, of speech, and of writing, dependent upon lesion of the foot of the third frontal convolution, which lesion causes destruction of "motor verbal images," or upon interruption immediately beneath the cortex of the fibers or pathways that convey motor speech impulses.

Sensory aphasia is due to the lesion of the posterior two-thirds of the zone of language, and especially the first temporal convolution and the angular gyrus. Flechsig has added enormously to our knowledge of the auditory area of the human brain. At a certain period of fetal development, a myelinated tract of fibers may be traced from the internal capsule and the lenticular nucleus into the medullary layer of the anterior transverse convolution, the convolution which forms, so to say, the tip of the temporal lobe on the lateral surface. It merges into the first, second, and third temporal convolutions. This tract of medullated fibers Flechsig proposes to call the primary projection system of the anterior transverse convolution. Destruction of the left anterior transverse convolution causes permanent word deafness, whereas total anacusis follows only when the anterior transverse convolution of both sides is destroyed. Flechsig has pointed out certain myelogenetic and histological conditions which indicate that the auditory areas of the two hemispheres are unlike.

**Revision of the Aphasia Question Proposed by Marie.**—This, in brief, was the status of the aphasia question when Pierre Marie assailed it in 1906. Marie maintains that the third frontal convolution of the left side does not play any special role in the function of language; that which is called motor aphasia or Broca's aphasia is anarthria plus aphasia; that the aphasia of Broca is not a disease, not a clinical entity, but a syndrome, a superimposition of aphasia upon anarthria, or, better, a simple juxtaposition of two distinct troubles, anarthria and aphasia. As to aphasia itself, Marie holds there is only one aphasia, which he proposes to call Wernicke's aphasia, and only one speech centre diffusely localized in the left temporoparietal lobe, and that this centre is a region of intelligence specialized for language, not a centre of sensory images. The clinical splitting up of the aphasia of Broca into two elements, anarthria and aphasia, Marie maintains is verified by autopsy. One finds constantly lesion of the lenticular zone associated with lesion of the zone of Wernicke.

Marie's description of his lenticular zone is as follows: If in a horizontal section of the brain one carries a line in a transverse direction from the anterior fissure of the island of Reil as far as a corresponding point in the lateral ventricle, and another transverse line from the posterior fissure of the insula to a corresponding point in the lateral ventricle, a region is circumscribed having nearly a quadrilateral outline and containing in its territory the caudate nucleus and the lenticular nucleus, the external capsule with its different parts the cortex of the island of Reil, and the internal capsule. It is in this territory very distinctly separated from the third frontal convolution that the lesion which determines anarthria especially is situated. It is this territory that the writer designates, for the sake of brevity, the lenticular zone. The superior and inferior limits of this zone it is not yet possible to define.

Anarthria (or aphemia, the term Marie is now willing to use) is characterized clinically by the loss of speech with preservation of the understanding of words, of reading and writing. It is produced by a lesion in the lenticular zone, interfering with the coördination of movements required for the phona-

tion and articulation of words, without inducing true muscular paralysis. Broca's aphasia is produced by the combination—the proportion varying with the case—of the lesion of anarthria with a lesion of Wernicke's zone or the fibers coming from this zone.

The third left frontal convolution plays no special part in the function of speech. The true speech centre is the zone of Wernicke; which must not be considered as a *sensory* centre, but as an intellectual centre. Wernicke's zone consists of the supramarginal and angular gyri and the feet of the first two temporal convolutions. Lesion of this centre determines in proportion to its extent, and in addition to the disturbances of speech, deficient understanding of spoken words, inability to read and write, as well as the disappearance of certain concepts of a didactic character. The foot of the first temporal convolution cannot be said to constitute a sensory centre for the auditory image of words. Pure deafness does not exist. Pure alexia (pure word blindness of authors) does not occur clinically. The lesion producing it is a lesion of the posterior cerebral artery, not of the sylvian artery, as in the other aphasias. It is useless and inaccurate to drag in the angular gyrus, which cannot be recognized as the centre of visual word images.

Marie maintains that there is no reason to preserve the classification of aphasia into cortical and subcortical forms. As a matter of fact, aphasia due to focal lesions is never exclusively cortical. It is, moreover, advisable at present not to refer to the cerebral cortex the entire pathological physiology of aphasia, since the subjacent white matter seems to play a part of perhaps greater clinical importance than the gray matter. If one insists upon classifying aphasia, the varieties of which are connected by a scale of innumerable transition forms, the best division would be into (1) *intrinsic* aphasia, in which Wernicke's zone or the fibers coming from it is directly and considerably affected by the lesion (Broca's aphasia, Wernicke's aphasia), and (2) *extrinsic* aphasia, in which Wernicke's zone with its fibers is not directly involved.

For Marie there is only one aphasia, the aphasia of Wernicke. The term sensory aphasia, he thinks, should disappear. The aphasia of Wernicke has for its substratum lesion of the zone known by his name. Intrinsic aphasia is accompanied by trouble of the internal languageand intellectual deficit. The other alterations of language, anarthria and pure alexia, are extrinsic syndromes. Pure alexia is dependent upon lesion of quite another vascular territory than aphasia. Aphasia is dependent upon lesions of the sylvian artery, and alexia upon obliteration of the posterior cerebral artery. He maintains that in autopsies upon patients with Broca's aphasia there is a double lesion. One causes the anarthria, and the other the trouble of internal language, of reading, and of writing. The terms Broca's aphasia, ataxic aphasia, total aphasia, designate the progressive degrees of the same syndrome.

The three important factors in the discussion are: (1) Is Broca's area, the foot of the third frontal convolution, the centre in which is stored the memories of phonetic speech, articulatory kinæsthetic memories? In other words, is motor aphasia or Broca's aphasia real aphasia at all? If there is a well-defined, though not sharply marked, syndrome to which the name of Broca's aphasia is given, upon what is it dependent anatomically? (2) Do lesions of the so-called lenticular zone give rise to a symptom complex parallel to that of so-called subcortical motor aphasia, the anarthria or aphemia of Marie?

(3) Is the area of language the area in which memories and words seen and heard are stored, and from which alone they can be evoked by peripheral stimuli, or is the storage of such memories a function of the anterior pole of the brain (the so-called psychic sphere) or of the whole brain itself?

It must be granted that there is no adequate clinical or anatomical evidence for considering Broca's centre to be the seat of memories of articulation. There does not exist in the literature a case of Broca's aphasia clinically in which the lesion was confined narrowly to the foot of the third frontal convolution, nor does the brain upon which Broca bases his original thesis, which is now preserved in the Musée Dupuytren, which the writer has examined carefully, show it. Although there are some cases recorded in which macroscopically lesions seem to be closely confined to Broca's convolution, macroscopic examination is entirely inadequate in these cases, save that it gives an accurate idea of the extent of the lesion. Serial macroscopic sections alone can be relied upon.

F. Bernheim studied five cases of motor aphasia in this fashion and in every case there were cortical and subcortical lesions reaching far beyond the area of Broca. Moutier, in his recent book, inspired by Marie, which aims to give documentary evidence of the latter's contention, has analyzed 304 cases of Broca's aphasia. In this series there were 84 cases directly opposed to the doctrine of location of phonetic memories to the third frontal convolution; 27 of these had motor aphasia with integrity of Broca's centre. In 27 of them the foot of the third frontal convolution was destroyed, either by tumor, traumatism, or softening, but in no instance had aphasia existed. In addition there are three cases in the literature of bilateral destruction of the feet of the third frontal convolution and two cases of surgical resection of the third frontal convolution in right-handed individuals without aphasia.

It may therefore be stated that our present conception of the zone of language must be modified in so far as denying the existence of the storage of articulatory or kinæsthetic memories in the anterior pole, the foot of the third frontal convolution.

It is admitted by everyone that there is an aphasia, which is clinically distinctly characterized, to which the name motor aphasia or Broca's aphasia is given. Its symptomatic features have been described. Is it sensory or Wernicke's aphasia plus anarthria or aphemia? If it is true that the foot of the third frontal convolution is not the seat of phonetic memories, then Marie's exploration is probably the correct one. Marie admits that Broca's aphasia and Wernicke's aphasia are clinically two distinct varieties. The distinction between the two is not always very sharply drawn, but, as a rule, certain characters permit the distinction to be made. Those who have contended that the symptomatology of the two forms of aphasia were very unlike have pointed out that it is a mode of copying that distinguishes motor aphasia from the sensory aphasia. It is admitted by everyone that the motor aphasic has some word deafness and some word blindness, but it is maintained that he copies print in script while the sensory aphasic copies servilely. But there has been recent important testimony to show that this is not true (Souques).

In regard to the mental defect of aphasic patients, whether they have sensory or motor aphasia, which Marie has emphasized, this has received due consideration in every treatise on aphasia, and there can be no doubt of

its existence; considering the lesion of the brain in the majority of cases of aphasia, the wonder is that the mental defect is not more pronounced.

That lesion of the so-called lenticular zone gives rise to the symptom complex parallel to that of so-called subcortical motor aphasia there can be little doubt. The preservation of internal speech is the distinctive feature in each of them. Whether the loss of speech capacity or destructive speech capacity which results is called anarthria or aphemia is not of prime importance.

In the discussion of aphasia recently held, Marie concedes the word aphemia to express speech defect of Broca's aphasia, which he first proposed to call anarthria, making sacrifice of the word, not of the fact. The writer agrees with Marie that the term aphasia should be reserved for those cases in which internal speech is disordered. Those cases in which the internal speech is not disordered and in which there is inability to speak, pure motor aphasia (Dejerine), should be classified as cases of anarthria or aphemia. This anarthria may exist from the onset of the patient's illness, that is, there may never be any true aphasia associated with it.

The answer to the third question cannot be given positively at this time. It will be appreciated that the phrase "storage of memories," which is constantly used in writing on aphasia as a symbol, is not to be taken literally. We do not know how memories are localized, or indeed that they are localized at all. We assume that cells undergo modification from each impression that reaches them, and that this cell reacts in a different way to different impressions at different times.

A case of pure motor aphasia without agraphia, by Ladame and von Monakow, would seem to disprove one of Marie's contentions, and it seems to the writer of great value in contributing to the solution of the question. A woman, aged fifty years, became completely mute at forty-five years after a slight apoplectiform attack which produced a transitory right faciobrachial paresis. She presented all the symptoms of so-called subcortical motor aphasia: loss of consciousness, complete loss of speech. At the time of the attack she understood perfectly what was said, and manifested by mimic her impatience at inability to make herself understood. Three days after the attack she could write a few words, and at the end of the ninth day she could write fluently. Mutism remained complete. Seven months after the attack she said, with great difficulty, "Adieu, ma petite," and a few days later, by a violent effort, said, "Merci beaucoup." She said nothing thereafter. The arm rapidly regained power, and she could use it perfectly, although it always became easily fatigued. Facial paresis lasted somewhat longer. Fourteen months after the onset, she asked in writing if she might be cured by hypnotism. At this time no trace of the paresis remained. She could write perfectly, spontaneously, and from dictation she could copy, and showed in many ways that her internal language was intact. She always showed some slight difficulty in swallowing if disturbed while eating. Whenever she ate alone, this difficulty disappeared. Death occurred ten years after the onset. The capacity to write perfectly had been preserved. The autopsy showed destruction of Broca's convolution and the inferior half of the ascending frontal. This formed a funnel-shaped cyst, filled with fluid, which had undermined the rest of the frontal operculum and second frontal convolution, and had penetrated to the gyrus supramarginalis. The apex of the funnel reached through the centrum ovale to the corona radiata;

its walls were very thin and little cicatrized. The secondary degenerations corresponded exactly to the experimental lesions produced in this area on dogs and cats. They commenced in the region of the knee of the internal capsule and could be followed to the posterior planes of the corpus luysius. The degenerated fibers were divided into two branches, one of which went to the anterior part of the thalamus and the other penetrated into the middle segment of the left cerebral peduncle, between the frontobulbar bundle and the pyramid. The first terminated in those groups of cells, which were also degenerated, around the external part of the median nucleus and the median part of the external nucleus of the thalamus.

The thalamocortical fibers, forming the bundles of the corona of the thalamus, which leave this area to terminate in the cortex implicated in this case, have their origin in these parts of the thalamus. These pathways constitute corticothalamic tracts, containing centripetal elements, which conduct the impressions of the muscular apparatus for the articulation of words to the cortex; that is, centripetal paths of phonation. For the first time this tract (the bulbarcortical) has been separated from the pyramids and the frontobulbar tract, and constitutes the real bundle of the central neurons of phonation. At the internal capsule it mingles with the other fibers and tracts without forming a distinct bundle. In the tegmentum it is situated in the central portion of the ribbon of Reil and the gray substance of the pons, dorsolateral to the pyramids. In the medulla they become part of the arciform fibers to reach the different nuclei.

von Monakow and Ladame explain the clinical phenomena by cortico-bulbar diaschisis, that is, inability to overcome the disturbance of innervation of the medulla due to individual peculiarities of the patient. They admit that the symptoms are not solely the consequence of the destruction of Broca's centre, but rather the sum total of the lesion plus individual peculiarities, functional disturbances, the nature of the disease, disorders of circulation, the psychic constitution, etc.

Diaschisis characterizes a group of functional symptoms of inhibition at a distance, resulting from the brusque interruption of permanent excitations, which had formerly acted on regions more or less distant, but in direct relation by association fibers, etc., with the region destroyed.

von Monakow makes a distinction between defects of speech present soon after the onset of the lesion, and which, slight in degree, soon pass off, and those which are pronounced and permanent. The early symptoms are caused by a lowering of functional activity in a more or less distant part of the speech mechanism, due to the upsetting of balance between the several parts of this mechanism produced by the destruction of one of the integral parts by the lesion. This is the phenomenon of diaschisis.

The position taken by Marie is not so revolutionary as has been commonly supposed. In the first place, it has never been seriously denied that there is not a certain amount of dementia in practically all cases of true aphasia, This mental defect varies in different cases, but it has been generally recognized. In the second place, the anarthria or aphemia of Marie is in reality the same thing as the subcortical motor aphasia of Wernicke and the pure motor aphasia of Dejerine. The important contribution that Marie has made is that the foot of the third frontal convolution (Broca's convolution, so-called) is not the seat of articulatory kinæsthetic memories, and is not the integral part of the zone of language, and that, therefore, destruction

of it does not cause aphasia. It is not enclosed in the quadrilateral area called the lenticular zone by Marie. The discussion on aphasia held in Paris in 1908 showed conclusively that the important point in the discussion was: "Is Broca's convolution in the quadrilateral area?" Marie's position, as stated in his own words, is: "I persist in refusing radically to place in my quadrilateral area the third frontal convolution, and when I speak of lesions that are situated in my quadrilateral area, I eliminate absolutely those which encroach upon the third frontal."

It is probable that Marie is correct in assuming that the third frontal convolution is not an area in which are stored memories of articulation. The criticism that may justly be made on Marie's revision of aphasia is that he has torn down without building up. When he attempts to define his quadrilateral or lenticular area, we see that, although he gives an anterior and a posterior border to it, it includes in reality an enormous amount of brain, especially in the superior and inferior direction, and Dejerine's criticism that it is not a localization at all is a very just one. Whether we accept the classic teaching or not (and certainly it cannot be fully accepted to-day, after all the evidence has been considered), we must admit that Marie's service has been very great, for it has put us on toward fact and away from theorizing.

**Diagnosis.**—The speech faculty consists of two parts, the receptive and the emissive; either may manifest the predominance of aphasic symptoms. In true aphasia, that is, aphasia dependent upon lesion of the zone of language, neither can be the sole medium of manifestation of the speech defects. Emissive speech is manifest by articulation, by writing, and by pantomime. Integrity of the receptive side of language is commensurate with the interpretation of visual and auditory stimuli.

The attitude, the demeanor, the conduct of the patient may be of the greatest service from the very beginning of the examination. The manner and expression of one with sensory aphasia (Wernicke's aphasia) are frequently those of a person who has lost interest in his surroundings, and his attitude is that of a deaf person slightly demented. Moreover, patients who have this form of aphasia are often garrulous, and on the slightest provocation, or without provocation, emit a string of sounds that convey no meaning. This is especially true of recent cases. Patients with motor aphasia (Broca's aphasia) and with subcortical motor aphasia (anarthria or aphemia of Marie) are very different. The aphemic is often absolutely silent, but watchful, and the intensity with which he holds every move of persons around him is very striking. He may be absolutely speechless, yet capable of the fullest understanding of all that goes on about him and within his hearing and vision.

A number of schemes have been devised to facilitate the examination of aphasic patients, and the following simple plan is found most serviceable. After securing a general history of the patient's life and of his previous illness from some member of the family, and in this way getting information of the character of the disease of which the aphasia is a symptom, the patient's ability to express ideas, to receive and interpret information through every avenue should be tested. The mental processes, apart from the manifestation of mental states, and the mental capacity for the reception of sensory stimuli, then should be examined. Although a number of these may be determined simultaneously, it is best to take each one separately, such as

attention, memory, orientation, capacity for retaining impressions, for deliberation, for reasoning, and his temperament or mood.

In approaching a patient with aphasia, it is natural that endeavor should be made to elicit information by speaking to him. It becomes necessary, therefore, to determine if the patient takes note of what is said to him orally, and secondly, if he understands what is said. In other words, does spoken speech awaken in his auditory centre and in the auditory interpretative area corresponding memories? This can be done ordinarily by taking some simple question, as, "How long have you been sick?" or by addressing to him some simple command, such as, "Give me your hand." Care must be taken not to employ too conventional questions or commands, such as, "What is your name?" and "Put out the tongue." The patient may have lost the auditory apperceptive faculty and still oftentimes make reasonable reply to such questions merely from association and habit. Naturally, the patient should not get any information of what is being asked through any other avenues than those of hearing. Such patients are quick to grasp, particularly if they have been aphasic for some time, the significance of even slight emotional expression or pantomime on the part of the interlocutor. If the patient does not reply to such questions or commands, there may be trouble with the receptive or with the emissive speech faculties. If he is word deaf (that is, if the trouble is one that prevents the sound of the word from reaching the centre in which the memories of previous word sounds are said to be stored), the patient will not endeavor to respond by word or act, though in some instances he does so. Nor will the face show the slightest response or indication of comprehension. If he does respond, the diagnostic feature is that his answer, even though it be made up of articulate words, has no pertinency or bearing on the question. If the patient is not word deaf, he will make some movement, be it of the head, hand, or features, to indicate that though he understands, he cannot reply. Generally, this gesture is very significant. It consists of a despairing expression of the countenance and a touching of the lips or the throat with the fingers. Oftentimes the question can be decided very quickly, if there remains some doubt even yet, by asking some absurd or ludicrous question and noticing how the patient receives it. If, in reply to the question, "Are you one hundred years old?" he solemnly says, "Yes," or if he does not see the ludicrousness of a request to turn a somersault when he is obviously paralyzed, it is rather convincing proof that such speeches do not awaken the proper responses in his mind, and if there be no dementia, it is suggestive evidence that the patient is word deaf, and the examination should then proceed from that standpoint. It must be borne in mind that there is a varying degree of word deafness in cases of motor aphasia. Thomas and Roux suggest a method for eliciting it. The patient is shown an object and the examiner pronounces several syllables of the name of the object, the first, last, or intermediate syllable. In minor degrees of word deafness the patient recognizes some of the syllables, not all.

Although all the speech centres may be simultaneously disorganized, the symptoms attributable to lesion of one usually dominates the speech defect. If the examination so far seems to suggest the existence of the word deafness as the leading feature of the sensory aphasia, it should then be determined to what degree of completeness this exists, and the extent and kind of disturbance that it causes in the externalization of language. The amount of diminution of the patient's vocabulary, the degree

of inappropriate usage of words, the imperfections of sequence and rhythm, should all be noted. The patient should be tested for his power of recognition of simple words, short sentences, and long sentences. As he may react to conventional questions, uncommon requests should be made. The ability of the patient to interpret sounds should then be noted. Do sounds evoke previous memories of similar sounds, and do they incite the auditory centre to revive the name of the object from which sounds proceed? When a bell is sounded, or a watch is held behind the ear and apart from the stimulation of any perceptual avenue other than hearing, can the patient say "bell" or "watch?"

Then an attempt should be made to determine the various degrees of identification of sounds and words which the patient hears, his capacity for repetition, and his capacity for spontaneous thought and action. His capacity to write spontaneously and from dictation, to read to himself, should then be tested, and then his ability to name objects and to designate persons and things.

Finally, the existence of any disturbance of bone or aërial conductivity should be demonstrated or excluded.

If word deafness can be excluded and the patient still makes no reply —that is, if he remains completely speechless—the examination should be made to determine whether or not internal language is defective, for the question has then narrowed itself to a determination of whether or not the aphasia is Broca's aphasia, or whether it is subcortical motor aphasia. In other words, is the inability to speak due to a lesion of Broca's area, or is it due to lesion of the neurons that conduct the motor word impulses from the Rolandic area to the parts that externalize the word? The essential thing, then, is to determine if the patient is in full possession of the internal language. If the internal language in any of its components is disordered, then the patient has true cortical motor aphasia. If, on the other hand, there is no such disturbance, the lesion is elsewhere than in the zone of language. In some patients the differentiation will be an easy one. On the other hand, however, the task is oftentimes an extremely difficult one. It is particularly so because the test to determine if the legitimate idea of words can be evoked in the internal language (the test of Proust and of Lichtheim) is not one of universal application, because in the first place many patients have no sufficient scholarship to know anything of syllables or word construction. In the second place, there is very often associated with aphasia a slight degree of dementia. In such patients it is often extremely difficult to make them understand just what is meant by telling them to press the physician's hand as many times as there are syllables in a word. Nor is the substitute of asking the patient to make voluntary expiratory efforts as many times as there are syllables or letters in a word more applicable. But even when we cannot get the patient to respond to these tests, there is a general atmosphere about the patient with subcortical motor aphasia (aphemia, anarthria) which enables one to recognize that the patient is in full possession of his intellect and internal speech. The only shortcoming of the subcortical motor aphasia is inability to articulate. He understands everything that is said to him; he interprets information received through the visual sphere; he is capable of expressing his thoughts fully, easily, and correctly by writing and by pantomime, or, at least, he would be if it were not that the right half of the body is usually paralyzed,

and he is obliged to portray mental states by the pantomime activity of the left, the less dextrous half of the body.

Difficulty is often found in properly assigning cases of cortical motor aphasia, because the patient is still able to articulate some words. If it be kept in mind that the patient with Broca's aphasia need not be absolutely deprived of the power to articulate words; that he frequently retains the ability to say one or several words, which he uses at all times and under all conditions, and that frequently these words take the form of recurring utterances; that there is always agraphia, which is usually proportionate to the aphasia; that it is manifest in voluntary writing and in writing from dictation, but not in writing from copy, and that the patient in copying, copies print in script and script in print, showing that the copying is not a mechanical, but an intellectual act; and that there is defective internal speech, then the diagnosis of Broca's aphasia will not be so difficult.

After having tested the patient's capacity to perceive and interpret words through the auditory apparatus, he should be examined with a view to determining if there is any disability of acquiring and interpreting information through the visual apparatus. To do this requires patience and circumspection. In the first place, it should be established that the patient has no trouble with the peripheral apparatus. This can be done by an ophthalmoscopic examination. Tests should be made to determine the existence of hemianopsia. This is not an easy matter to do if the patient is aphemic or if he has word deafness; in fact, it is extremely difficult to do satisfactorily. With a patient who can understand what is said to him and who can indicate when he perceives the entrance of an object into the visual field, who can tell when the indicator of a perimeter passes beyond the range of vision, testing for hemianopsia is a simple matter. If the patient is word deaf, and if he has visual blindness, which, of course, he is apt to have if he has hemianopsia, one finds himself unable to convey to the patient by written or spoken word that which one wishes him to do or to observe. In such cases one must content himself with the information that is to be derived from forcibly and suddenly thrusting some object into the visual fields, from the right side (for right-handed patients invariably have right lateral homonymous hemianopsia when they have any), and taking note whether or not the patient blinks, as he should do if the object be perceived. If he does not, it is rather certain that he has hemianopsia.

In testing the patient to determine the integrity of the visual mechanism, one may begin by showing him familiar objects. If he does not recognize them, or shows by act or deed that he comprehends their uses or purposes, the lesion is probably of the occipital cortex. Such an individual may obtain information through the medium of other special senses, such as the tactile, gustatory, etc., that will enable him to recognize the object, the person, or the thing. If he is shown familiar objects, and he recognizes them, knows what they are for, but cannot name them, then he may have either an interruption in the pathway leading to the higher visual centre or in the centre of the intellect specialized for visual language. If it be the former, internal language will be preserved and spontaneous speech may be intact, although there is usually some paraphrasia and jargon-aphasia, and this preservation is shown most conclusively by the retention of ability to write. He may write easily and moderately well, not only voluntarily, but from dictation, but the patient is unable to read what he writes. If the

aphasia be of the latter character and complete, the patient will be absolutely agraphic. This agraphia is to be considered a part of the disorder of internal language; there is inability to arouse the visual image of the word. In such a case, an arousal must precede the transmission to the part of the Rolandic cortex that innervates the member holding the pen; there is complete agraphia. The physician then proceeds to examine whether the patient has word blindness; that is, whether the patient can read print, script, figures and other forms of notation.

We shall understand aphasia better after we have interpreted the significance of apraxia, and in every case of aphasia the existence or absence of apraxia should be established from examination of the patient's spontaneity and initiative and by a study of movements conditioned by visual, auditory, tactile, and kinæsthetic stimuli. It should be specifically noted what the patient does when objects are held up before him, what his responses are, and how they are expressed to appeals of any nature made to him through his vision, his hearing, or his senses of touch and posture. Apraxia may exist without aphasia, and vice versa, and when they occur separately the interpretation of each is much easier.

# CHAPTER IX.

## DISEASES OF THE CEREBRAL BLOODVESSELS.

By HENRY M. THOMAS, M.D.

**Anatomy.**—The blood reaches the cranial cavity by four arteries, two on each side, and these also connect with the rich plexus about the spinal cord, which in its turn receives blood at various levels by arteries which enter the spinal column along with certain of the nerve roots. It may be said, then, that there are three arterial systems supplying the brain—the internal carotids, the vertebrals, and the spinal plexus. The relative importance of these varies a great deal, especially that of the spinal plexus, which seems of but comparatively slight importance in man, although in certain animals it is competent to maintain the cerebral circulation when the other two systems are cut off.

The vertebrals which arise from the subclavian arteries pass up through the interrupted bony canals formed by the foramina in the transverse processes of the upper six cervical vertebræ, and pierce the dura mater between the atlas and the base of the skull. After entering the subdural space they converge along the lateral and anterior aspects of the medulla oblongata to the median line, where near the inferior border of the pons they unite and form the basilar artery. Each vertebral, in its intradural course, gives off a varying number of branches, which pass over the lateral to the dorsal aspect of the medulla. Any one of these branches may be much larger than its fellows and form the posterior inferior cerebellar artery which reaches the inferior surface of the cerebellar hemisphere, over which it ramifies. The lower vertebral branches are connected intimately with the spinal arterial plexus, and are connected more or less with the branches above. The posterior spinal arteries rise from this network of arteries formed from the branches of the vertebral, or directly from one branch, as the posterior inferior cerebellar artery. Shortly before the vertebral arteries join to form the basilar artery each gives off the relatively large twig which passes over the ventral aspect of the medulla to join and form the unpaired anterior spinal artery.

The basilar, in its course over the pons, gives off numerous small branches, and from it also arises the anterior inferior, or middle, and superior cerebellar arteries. When the basilar reaches the superior border of the pons it divides in a T-shaped manner, and may be said to end in the two posterior cerebral arteries, which, passing above the tentorium, apply themselves to the inferior and posterior aspects of the cerebral hemispheres.

It is evident that the vertebral arteries entering the posterior fossa of the cranium supply all the important structures lying in this fossa. This vertebral system anastomoses freely below with the arterial system of the spinal cord, and above it ends in the posterior cerebral arteries, which form

the posterior pair of the three great arteries of the cerebral hemispheres. From each of the posterior cerebral arteries, soon after its origin, is given off a vessel, the posterior communicating artery, which unites the vertebral system with that of the internal carotids and helps in the formation of the circle of Willis.

The internal carotids enter the cranial cavity through the carotid foramen of the temporal bone. Each pierces the dura mater and is continued directly into the middle cerebral artery, which, in its turn, is connected with the posterior cerebral artery by the posterior communicating artery. It also gives off the anterior cerebral artery, which passes forward and toward the middle line to be supplied to the medial surface of the cerebral hemisphere. Just in front of the optic chiasm the two anterior cerebral arteries are united by a short trunk, the anterior communicating artery, and thus the circle of Willis is completed.

There is the freest communication between the large arterial trunks at the base of the brain, and the circulation of the two sides is intimately connected. It must, however, be borne in mind that there is considerable variation in the size of the communicating branches, and that the circle of Willis is not so regular as the diagrams would indicate.

We may consider that the blood for all parts of the brain comes from a common arterial plexus or reservoir, situated in the middle line at the base of the brain. The vessels which nourish the structures at the base of the cerebral hemispheres, the basilar nuclei, etc., are given off directly from this plexus, or from large arterial trunks shortly after they leave it. These nutrient basal arteries penetrate the brain to a greater or less extent, and appear to be end arteries. The cerebral cortex gets its blood by the anterior, middle, and posterior cerebral arteries. Each of these vessels supplies a more or less definite area, but these are not absolutely distinct, as they overlap and there are actual anastomoses by arterial branches of fair size (from 0.5 to 1 mm.) between the vessels of neighboring areas.

The nutrient vessels of the cortex, so-called cortical vessels, are not given off from the larger arterial twigs, but from the smallest vessels of the pia mater, which have arisen by division of the larger branches. In the rich arterial mesh of the pia one is able to find occasional anastomoses between arterial twigs of fair size, but there does not appear to be a true pial plexus in the sense of Huebner. The nutrient cortical vessels enter the cortex at right angles and descend to a greater or less depth. They give off branches to the cortex and the white matter beneath it, and seem to be practically end arteries, although anastomoses between the branches of neighboring vessels have lately been described.

The vessels of the cortical system which pass to the white matter almost reach to the basal nuclei. They, however, make no anastomoses with the system of basal nutrient arteries, and the two systems seem quite independent. Beevor's[1] beautiful injections show as never before the area supplied by the different cerebral arteries, and his work has settled many disputed points. The drawings of the arterial supply of the basal nuclei by Aitken[2] are also most instructive.

The blood, after having passed through the capillaries, is collected by the

[1] *Phil. Tr. Lond.*, 1908, cc, 1; *Brain*, 1907, xxx, 1.
[2] *Boston Med. and Surg. Jour.*, 1909, clx.

small veins and taken to the surface of the brain, where they join in the formation of the venous plexus, which empties its blood by the way of larger veins into the various venous sinuses. The veins on the surface of the brain are less numerous than the arteries, but are of larger size and have a more superficial course. The various sinuses are large venous reservoirs, lying in folds of the dura mater. Most of them are protected on one side by the skull itself. They are intimately connected among themselves and all empty into the internal jugular veins. The intracranial venous plexus also connects with the veins of the rest of the body by other ways. Chief among these is the anastomosis between the cavernous sinus and the veins of the orbit, which, in their turn, connect with the superficial veins of the face. The other less important connections need not be especially mentioned, except to point out that there are numerous anastomoses between the veins which run in the diploë and the intracranial system on one side and the veins on the outer surface of the skull on the other. The connection of these veins with the sinuses is largely through the Pacchionian granulations, the curious little venous lakes situated in the dura close to the longitudinal sinus.

Within these granulations little folds of the arachnoid, filled with cerebrospinal fluid, are found, and it is believed by some that it is here that the cerebrospinal fluid passes out into the veins. The cerebrospinal fluid appears to be secreted by the choroid plexus, and to pass from the ventricles into the subarachnoid space by the foramen of Magendie and other openings in the pia beneath the cerebellum.

**Physiology.**—Within the skull we have, then, the brain with its membranes, blood, and cerebrospinal fluid. There is much dispute about the exact conditions which regulate the circulation of the brain. We may regard the skull as a closed box, protecting its contents from the direct action of atmospheric pressure. The total amount of blood within the skull at any instant varies but little from that at any other. This is true because the brain substance itself is incompressible, and so the small amount of cerebrospinal fluid is the only thing that can vary. Although the total amount of blood varies but little, the proportion between the arterial and venous blood may and does vary greatly.

The arteries of the brain have well-developed muscular walls, and contain numerous nerve fibers, and it seems certain that they do at times change their caliber independently of the general arterial pressure. But in spite of this, it is generally stated by physiologists that there is no definite proof that the cerebral vessels are under effective vasomotor control, and the view of Leonard Hill, that the cerebral circulation follows passively changes in the general circulation, is widely accepted. Most experimenters have failed to get any evidence of direct vasomotor action until recently, when Müller and Siebeck[1] reported experiments in which they cut and stimulated the cervical sympathetic, and obtained evidences of definite vascular changes in the brain; and Wiggers[2] has shown that the cerebral vessels, as well as the renal vessels, react to various drugs in very characteristic ways, which he believes is due, at least in a large part, to the effect of the drugs on the nerve endings. It takes, however, a much stronger solution of adrenalin, for instance, to cause the arteries of the brain to contract, than is effective on

[1] *Zeitsch. f. Exper. Path. u. Therap.*, 1907, iv, 57.
[2] *Amer. Jour. Phys.*, 1907, xx.

the vessels of the kidney. Surgeons have noticed the pial vessels change their caliber under the influence of drugs (Cushing).

Therefore, it seems probable that the cerebral vessels are under a more or less effective vasomotor control, but there is much to be done to establish this on a perfectly satisfactory basis, and in any case it seems fair to say that the cerebral circulation as a whole does follow passively that of the general circulation. When the blood pressure is raised, the blood enters the skull with increased force, and a greater quantity passes through the brain in a given time. This active hyperæmia must occur under many circumstances, but it is doubtful whether it causes any symptoms.

Consciousness depends upon a sufficient amount of blood reaching the brain, particularly the cortex, and life itself depends upon arterial blood passing through the medullary centres. To the vasomotor centre is delegated the function of maintaining an adequate blood supply to the other vital centres and to the brain in general. When there is a lack of arterial blood reaching this centre, *i. e.*, when it is anæmic, it is thrown into strong activity and causes a contraction of the arterioles within the splanchnic and skin areas, and in this manner the general blood pressure is raised, and the blood enters the brain with augmented force and floods the capillaries with arterial blood. The extent to which this regulating mechanism can counteract an obstruction through these centres has been well shown experimentally by Cushing.[1]

The intracranial pressure, that is, the force which the brain exerts against the skull, depends upon the blood pressure, and is equal to the pressure of the blood in the venous sinuses, which, under normal circumstances, is the same as the general venous pressure. It is obvious that if the intracranial pressure be raised above the general arterial pressure, no blood can enter the skull, and the animal will die. In Cushing's experiment it was shown that when this pressure became so great as to embarrass the medullary centres there was a corresponding rise of general arterial pressure. If he again increased the intracranial pressure, the vasomotor centres answered again with a rise of arterial pressure. This could be repeated until the vasomotor centres were exhausted.

If this regulating mechanism is disturbed, various results may follow, as, for instance, when the sudden removal of something that has caused continuous pressure on the abdominal vessels, as ascitic fluid, is followed by alarming symptoms. In this case the vasomotor influences controlling the splanchnic area have not been called on for some time, and the centre itself has taken part in the general weakened condition of the individual, so that when a sudden demand is made to compensate for the accustomed external support to the bloodvessels, it is entirely unable to respond, the blood collects in the splanchnic vessels, the patient becomes unconscious and may die.

While under ordinary circumstances the vasomotor mechanism and the tonicity of the muscles of the abdominal walls compensate perfectly for the change from the horizontal to the upright position, *i. e.*, for the effect of gravity upon the column of venous blood from the heart to the feet, in asthenic states, as after severe illness, the compensation may be very imperfect. When such is the case, if the patient stands, or, at times, even if he sits up in bed,

[1] *Mitt. a. d. Grenz. d. Med. u. Chir.*, 1902, ix; *Amer. Jour. Med. Sci.*, 1902, cxxiv.

his heart beats more rapidly, he becomes giddy, and may faint. The change in the pulse rate with a change in position is a fair indication of the vasomotor control, for the heart itself endeavors to make up for this incompetence.

During surgical anæsthesia induced by chloroform, and to a less extent when ether is used, a paralysis of the vasomotor centres is apt to develop, and it is for this reason that position is such an important factor for the safety of the patient, and it also explains the various procedures used in resuscitating patients, such as lifting the foot of the table, bandaging the extremities, etc. They are all directed to compensate for the loss of vasomotor control over the vessels in the splanchnic and skin areas.

Under various conditions the heart itself may become so weak as to be unable to keep the brain properly supplied with arterial blood. The ordinary fainting fit is, at least in part, an example of this. Under the influence of emotion the heart's action becomes weak, the vasomotor centre is inhibited, and, in consequence, the abdominal bloodvessels become dilated, blood pressure falls, and the heart is no longer able to drive the blood back to itself against the force of gravity; the blood accumulates in the abdominal veins, the heart empties, cerebral circulation fails, and unconsciousness occurs. The sudden loss of consciousness in epilepsy has also been ascribed to a transient paralysis of the heart (Russell[1]). In Stokes-Adams disease the cerebral symptoms, attacks of unconsciousness, convulsions, and apoplectiform seizures, are due to cerebral anæmia, caused by the temporary cessation of the ventricular systole. The extreme example of this cardiac weakness is paralysis of the heart muscle from failure of the coronary circulation, which is the usual cause of sudden death.

The unconsciousness, or, indeed, death, which at times follows forcible compression of the chest, is due to failure of the cerebral circulation caused by the inability of the heart to fill itself with blood, as well as to the damming back of the blood into the venous sinuses. Movements of the chest in respiration have an important effect upon the circulation in general and on that within the skull in particular. With every inspiration the blood is sucked into the heart from the veins, and the descent of the diaphragm, by increasing the pressure on the abdominal veins, tends to force the blood into the heart. During expiration, on the other hand, the intrathoracic pressure is increased, and so the entrance of the blood into the heart is impeded, Arterial blood pressure is influenced but slightly by the respiratory movements.

Intracranial pressure has been shown to be equal to the venous blood pressure within the sinuses and to follow every change in this. The column of venous blood between the brain and the superior vena cava is uninterrupted by competent valves, and, therefore, every change of pressure in the cava is transmitted directly to the sinuses and veins of the brain. The brain dilates with each pulse beat, that is, with every change of arterial pressure, but relatively much more with each expiration, that is every rise in venous pressure. Intrathoracic pressure is increased in expiration, and this causes an increase in the pressure of the cava, the jugular, and the brain sinuses. During prolonged expiratory efforts, the venous blood is, as it were, dammed back into the brain, intracranial pressure rises, and, as less

[1] *Lancet*, 1909, i.

arterial blood can pass through the cerebral circulation, the symptoms of anæmia may follow, as when a crying child holds his breath until he becomes unconscious.

Intrathoracic pressure makes it more difficult for the heart to fill itself with blood, and effects the circulation also in this way. Strong respiratory efforts against an obstruction may cause very marked changes in intrathoracic pressure. If the glottis be closed and forced expiration be attempted, a normal, negative pressure of the thorax becomes markedly positive, and may far exceed normal pressure in the intrathoracic veins. This is conveyed directly to the cerebral veins and capillaries, and it is not uncommon to have hemorrhages within the brain substance following such an effort. These conditions accompany a strong effort, as straining at stool, lifting a heavy weight, or coughing severely. The importance of preventing, as far as possible, any obstruction to respiration during the course of apoplexy follows directly from what has been said.

The venous outlets from the skull are so large and the anastomoses so free that they must all be obstructed to cause venous congestion of the brain. It is for this reason that thrombosis or ligature of one of the sinuses is not necessarily followed by any symptoms, and, indeed, the superior vena cava itself may be obstructed, as by a tumor, without symptoms. In this last case the blood from the vein reaches the heart through the craniovertebral veins and anastomoses through paths to the inferior vena cava. If all the veins of the neck are compressed as by a tight band or by strong flexion of the neck, the circulation may be impeded to a certain extent, and this is important under pathological condition.

**Hyperæmia.**—The brain is extremely sensitive to changes in the amount of blood which it receives. Many symptoms have been ascribed to anæmia and hyperæmia of the brain, but there is very little agreement as to the symptoms which depend upon these two conditions, and, indeed, upon just what is meant by them. Hyperæmia is sometimes divided into passive and active; passive hyperæmia meaning the damming back of blood into an organ by impeding the outflow from the veins, *i. e.*, a great increase in the venous blood at the expense of the arterial blood, and is, in fact, a venous hyperæmia and an arterial anæmia. The symptoms which follow must be largely due to a lack of arterial blood, and are, therefore, quite similar to those which depend upon anæmia.

Active hyperæmia of the brain may be due to anything that causes an increase of flow through it, *i. e.*, anything that makes the difference between arterial and venous pressure greater; for instance, an increase of the general arterial pressure or a lowering of the venous pressure in the sinuses. Such changes must be occurring all the time, and we know of no symptoms due to them, and it is hard to believe that any harm follows this increase in the supply of arterial blood.

**Anæmia.**—This may be either general or local, *i. e.*, the brain as a whole may take part with the rest of the body in a decrease of the total amount of blood, and it may be effected by a change in its quality. On the other hand, the circulation of the brain itself may be altered as a result of local conditions, by ligature of one of the vessels that enter the skull, or by disease of any of the intracranial vessels.

The symptoms due to anæmia associated with general loss of blood have been more or less known since the earliest times, for they make up most of

the clinical picture following hemorrhage, and were the symptoms upon which the older physicians relied when bleeding as a therapeutic measure. Since this procedure has become so infrequent the interest in the symptoms of hemorrhage has been largely confined to the surgeons, who study them as the chief part of surgical shock. A reference to any of the older standard works on medicine, published in the first half of the nineteenth century, will show how much attention was given to this subject. When the loss of blood is sudden and of sufficient quantity, the patient may faint. He first experiences a sense of vertigo, may have ringing in the ears, dimness of vision, and a rapidly increasing insensibility. The breathing becomes shallow and intermittent, more or less of the Cheyne-Stokes type; the pulse is feeble and rapid; the extremities are cold and covered by profuse perspiration. At times, especially when the bleeding has been rather slow, the patient may have general convulsions before loss of consciousness occurs, or, indeed, the patient may die suddenly without any very marked premonitory symptoms, having retained consciousness until the end. This accident is more apt to occur when the patient, who has lost a considerable quantity of blood in a horizontal position, stands or sits up; or even without any change of position, when the hemorrhage has been great enough to seriously embarrass the heart.

The older physicians, who consistently and conscientiously bled their patients, were well aware of the importance of position, and directed that they must be "blooded" while standing or propped up in bed, and that the amount of blood taken should be regulated largely by the symptoms of beginning anæmia of the brain. In effect, they placed the cerebral circulation in the most unfavorable hydrostatic position, and used the delicate response of the brain to its failure as a danger signal.

When the central nervous system is supplied with blood of a poor character, as in grave anæmias, symptoms of malnutrition, more or less pronounced, are apt to follow. The headache, vertigo, tinnitus, and muscular weakness, so often complained of, may, at least in a large part, be thus accounted for. On the other hand, it is quite remarkable how well and how long at times the nervous system performs its functions without apparent difficulty, even when the blood which nourishes it is far below the normal standard. In most of such cases, however, it will be found that the immunity from symptoms is only relative, and that under stress the brain gives evidence of its insufficient blood supply.

Many symptoms are ascribed to anæmia of the brain due to spasm of the vessel walls in circumscribed areas. Thus the transient paralyses in arteriosclerosis, etc., are so explained. This depends upon the assumption of an active vasomotor control, and we have seen how little definite proof there is of such an action. Should it be demonstrated, we may learn to recognize a whole group of phenomena now little understood.

**Cerebral Arteriosclerosis.**—The vessels of the brain may be diseased either alone or in association with the rest of the bloodvessels. The local occurrence of arterial disease has not as yet been thoroughly worked out, but enough has been done to establish the fact that there may be marked degeneration in one system alone, and that the condition of those vessels which can be easily examined, such as those in the arms and legs, is no absolute criterion to that which may be present in some other system, for instance, the brain.

**Etiology.**—So long as our knowledge as to the production of arteriosclerosis remains so incomplete, we can not expect to discover why, in one case, the vessels of the brain should be first affected, while in another it is those of some other vascular area, such as those of the abdomen.

Given the general causes, the wear and tear of life, the acute infections, the intoxications, and the conditions which keep up an increased blood pressure, we may perhaps assume that those organs upon which the greatest amount of stress is brought would be those in which vascular changes would first appear. We might expect, therefore, in those individuals whose activities have been largely intellectual to find the cerebral vessels affected more often than in others whose work has been almost entirely physical. In a general way this appears to be true, although there are so many modifying factors that it is of very little practical diagnostic value. The brain does not only function during so-called intellectual processes, but takes part in most of the bodily activities, and as cerebral circulation follows passively the changes in that of the rest of the body, the cerebral vessels are under constant strain, and almost as much so in the laborer as in the student.

Certain infections, syphilis for instance, appear to have a peculiar affinity for the cerebral vessels. The syphilitic endarteritis is, indeed, usually distinguished from other alterations of the vessel walls, and is apt to be associated with a gummatous involvement of the meninges, and to be followed by more or less characteristic symptoms. Syphilis, with the other infectious diseases, does seem to predispose to the ordinary arterial changes, and arteriosclerosis in infants and children is believed to be frequently due to hereditary infection.

*Age.*—In the brain, as in other parts of the body, degeneration of the arteries may occur at almost any time of life. In hereditary syphilis early arteriosclerosis is common, and at times it follows acute infectious diseases. But generally arteriosclerosis may be considered a rarity before the fortieth year, and from that time on it becomes more common with every succeeding decade, but it should be remembered that extensive arterial disease does occur before this period. Arteriosclerosis is more common in men than in women, and in those individuals who work hard and live well.

**Pathology.**—Anatomically, fairly clear-cut changes in the brain, associated with alterations in the vessels, have been described. These consist in focal areas of necrosis, which may be very small, but are at times large enough to be seen by the unaided eye. These are essentially infarcts due to obliteration and sometimes to rupture of the capillary vessels. They are most frequently seen in the gray matter of the cortex, but may be found scattered anywhere in the brain. Associated with these one finds the ordinary large areas of disease due to obliteration or rupture of one of the larger arteries, which explain the occurrence of focal symptoms. The cortical changes are distinguished from those found in general paresis by being focal and unaccompanied by the special histological elements seen in this disease. The changes in senile dementia are also much more diffuse and have their own special characteristics.

Marie[1] and his scholar Ferrand[2] have pointed out the importance of small areas of softening (lacunæ) secondary to arterial sclerotic changes, as the cause of hemiplegia in elderly people. These were often associated with hemorrhage into the brain, at times with softening, but in more than half of the

[1] *Rev. de Méd.*, 1901, xxi, 281. [2] *Thesis*, Paris, 1902.

cases they occurred alone. The symptoms which he associates with these changes are very similar to those described under arteriosclerosis.

**Symptoms.**—The anatomical changes in the arteries affect the walls so as to alter their make-up and render them less elastic, more brittle, and prone to gross alterations, such as the formation of aneurisms. The walls may be so thickened as to encroach on the lumen and decrease the capacity. As a result, the organ supplied by the diseased vessels receives its blood through vessels of a smaller capacity and with walls which are changed. Thus the amount of blood reaching the brain is decreased, and the interchange between the blood and the nervous tissue, upon which nutrition depends, is altered. This is entirely aside from the actual plugging of an artery or the rupture of its walls, the effects of which are to be the subject of a consequent section.

Nature provides lavishly, and it is probable that under normal circumstances much more blood passes through the brain than can be used. Certain it is that in not a few cases the cerebral vessels may be markedly altered and the brain continue to perform its functions so well that no defect is obvious. This is due not only to the utilization of the normal surplus of blood, but also to the reserve force of the circulatory system itself, which tends to compensate for anything that impedes the flow of blood. On the other hand, there are cases in which, although the vessels are much less altered, we ascribe the pronounced symptoms to a change in the circulatory condition; but we cannot, as yet, explain why symptoms occur in these and not in the other cases. It must be acknowledged that there is no considerable evidence upon which to base conclusions. The anatomical investigation is apt to be limited to the larger arteries at the base of the brain, and the clinical notes are far too often so meagre as to make even this of little use in correlation with the symptoms, and to render superfluous in this relation a minute study of the smaller and nutrient vessels of the brain substance. It may well be that these smaller vessels are at times much altered, when the larger vessels are but little affected, or the reverse may be true, and also the arteries of the different areas of the brain may be unequally diseased.

One would expect that as the brain began to feel the decreased blood supply the symptoms would at first, if noticed at all, be indefinite and transient, and that they would differ with the varying distribution of the vessels most diseased. Until our knowledge is much more complete any statement as to the symptoms of beginning cerebral arteriosclerosis must, of necessity, be indefinite and uncertain. There is a growing tendency, nevertheless, to explain the occurrence of a great variety of symptoms as primarily due to an altered blood supply. The following are the more important symptoms that are usually so explained.

*Neurasthenia.*—In certain cases in which arteriosclerosis has reached a relatively high degree, and which present definite symptoms referable to cerebral vascular lesions, a history is given of recurrent attacks of neurasthenia dating from a relatively early period. The conclusion that these were themselves evidences of beginning arterial disease is attractive; and when, on the other hand, one finds men of about forty, or older, suffering from the protean symptoms which we class under neurasthenia, and the physical examination reveals well-marked arterial changes, we may be excused if, in our ignorance, we explain one condition by the other.

One chief characteristic of the neurasthenic is that he is easily tired

after any effort, and that even the slightest physical or mental work is followed by many distressing symptoms. If the brain is nourished by an inadequate blood supply or receives its blood through vessels whose walls are so altered as to interfere with the free interchange of the blood and the tissues, one would expect that it would show some such evidences of distress under activity. The fact that most cases of neurasthenia can be cured by appropriate measures indicates that the condition of the brain is usually only a part of the general bodily state. Even if there be a beginning cerebral arteriosclerosis, which makes the circulation of the brain inadequate when there is a general lowering of all the bodily functions, the changes may be so slight as to be entirely compensated when the general level of bodily vigor is raised. Cerebral activities, psychical, as well as physical, are, of course, important factors in this general uplift, and one may perhaps thus understand some of the good results of mental therapeutics, even in cases of neurasthenia which seem to have been due to actual organic changes, as proved by the subsequent course.

However this may be, the practical indication is clear that we must keep in mind the possibility, in treating our cases of neurasthenia, even those in young individuals, that the chief underlying factor may be a beginning arteriosclerosis, and that we should not neglect to examine carefully for this condition, and if found we should not overlook it in our plans for treatment, no matter how enthusiastically we may regard the perhaps more alluring field of psychical therapeutics.

*Headache.*—This is often classed as one of the most common and most persistent symptoms in cerebral arteriosclerosis, and, indeed, it does occur; but the writer has been struck by the relative infrequency of this symptom, and its complete absence occurs under conditions when it might be expected. Thus, a number of elderly patients, although suffering from undoubted general arteriosclerosis, with marked involvement of the retinal vessels, and in whom the blood pressure remained persistently high, rarely falling to 200 mm. Hg., have entire freedom from any headache. Walton and Paul, in their study of 100 patients, all the subjects of arteriosclerosis, found headache in only 22 per cent., and in this connection they refer to the well-known fact that individuals who have been subject to headache in their youth, often become immune with advancing years; whereas the opposite would be expected were arteriosclerosis in itself a common cause of headache.

On the other hand, one is at times consulted by patients suffering from intense headache, who give a history of having been entirely free up to late middle life, and it is in these cases that we should carefully examine the vascular condition; but even in them it may be the associated changes that are at fault, such as involvement of the kidneys. When arteriosclerosis has advanced so far as to give focal cerebral symptoms, the onset of this is very generally associated with pain and other distressing sensations referred to the head.

*Vertigo.*—Nearly all patients complain of a sense of insecurity of equilibrium, of dizziness, or of actual vertigo. This may be more or less constant, but is more commonly transient and brought on by exertion or change of position. Some patients when placed in a certain attitude suffer at once from a sharp attack of vertigo, as was the case in two patients neither of whom could be shaved in the usual manner, for the instant the barber tilted back the chair the patient had a vertiginous attack with the sense of

impending loss of consciousness and death. In each of these cases this distressing symptom subsided under hygienic and tonic measures. Subjective auditory sensations, as well as true tinnitus, are occasionally associated with vertigo, and, indeed, may occur alone. The presumption in these cases is that there is some involvement of the middle or internal ear, but if such is the fact it escapes demonstration. It may be that some cases in elderly people, which we are in the habit of designating aural vertigo, are really to be put in this category. Vertigo, like headache, is commonly associated with the transient cerebral attacks which are very frequent in certain cases of cerebral arterial disease.

*Apoplectiform Attacks.*—As would be expected, it is not uncommon to have sudden attacks due to more or less closure of some one of the cerebral bloodvessels, or, indeed, to their rupture. The resulting paralysis may be permanent, or it may last only a very short time. In the first case we believe that there has been a destruction of nervous tissue, but in the second the loss of function is so transient that we assume it to be due to a passing stoppage of the circulation, from the effects of which the brain has recovered. It is these latter cases in which we are at present interested, although they are relatively much less common. These patients suffer from repeated, and in some cases very many, attacks of vertigo and headache, associated with aphasia, or a more or less extensive paralysis of one side or the other, from which they recover completely. These attacks may come on spontaneously or after exertion. They have been brought into relation with the intermittent loss of function which under stress occurs in other organs, whose bloodvessels are diseased—the intermittent claudication of the limbs, angina pectoris, etc., and the assumption of a transient spasm of the walls of the vessels is given as a common explanation. Even the term claudication has by some authors been extended to include these cerebral attacks, but this seems a too free use of the term. That the cerebral arteries during these attacks are in a state of spasm is an inference based upon analogy, which, however, receives strong support from observations on the retina in certain cases of transient blindness when the actual constriction and relaxation of the sclerosed central artery is to be noted (Zentmayer).

*Convulsions.*—Convulsions, either general or focal, may be associated with the apoplectiform attacks, but perhaps more frequently the subject of cerebral arteriosclerosis suffers from convulsions which have the usual characteristics of epilepsy. The occurrence of such seizures in individuals who have reached middle life always makes one suspect the presence of this condition. Typical Jacksonian attacks at times recur for years after the occurrence of a cortical lesion.

*Mental Symptoms.*—If the mental states which so frequently accompany neurasthenia are included under this heading—the inability to continue mental effort for any length of time, the forgetfulness, the irritability, the depression of spirits, etc.—we may say that mental changes occur early in cerebral arteriosclerosis. Usually, however, it is not until the disease has progressed much farther that the mental changes are so pronounced as to constitute a real alienation. On the other hand, it has been suggested that in manic depressive insanity, the varying mental states, with their associated toxic condition, are particularly favorable for the occurrence of arteriosclerosis, and Albrecht[1] found arteriosclerosis in 30 per cent. of 54

[1] *Ab. Rev. of Neurol. and Psychiat.*, 1906, p. 607.

cases examined, and he also thinks the condition of the arteries in elderly people makes them more liable to this type of insanity.

The other types of mental disease which occur late in life, the devolutional psychoses (Farrar[1]), are, as would be expected, often associated with vascular changes, and it is difficult at times to determine how much of the symptomatology is actually due to the altered circulatory condition. From these psychoses, occurring during the latter part of life, there has been differentiated a distinct type which is believed to be due to cerebral arteriosclerosis, and the symptoms have been brought into relation with fairly definite anatomical changes. Such patients who may have passed through previous attacks of neurasthenia begin to appreciate that they can no longer accomplish their accustomed mental work, that they tire easily, are unable to undertake new problems, often notice that their memory fails them, that they are unable to recall names, and frequently miss the right word. These symptoms are often associated with the attacks of vertigo and other manifestations of arteriosclerosis which have been described above, and later definite vascular lesions occur, which, although they may be recovered from, tend to leave the patient on a lower mental level. In other cases these seizures may themselves be the first indication of the process.

As the mental failure increases the patient becomes unable to carry on his work, is usually apathetic, but his mood may show marked variability, and changes quickly from that of deep depression to that of well-being. It is uncommon, however, for this to reach an actual state of exaltation, such as is not infrequently seen in general paresis. Most cases have a remarkably clear appreciation of their condition, and this is an important help in distinguishing them. The condition may progress to almost complete dementia, but is more apt to be terminated by a vascular accident.

The clinical picture at times closely resembles that of general paresis, and it may also simulate senile dementia. The diagnosis rests largely upon the presence of the more or less characteristic mental conditions, associated with definite symptoms of arteriosclerosis, and the absence of the objective symptoms which point to general paresis, or the typical, clinical picture of the senile dement. The age of the patient is also of importance. General paresis occurs, as a rule, relatively early, during the full vigor of maturity, while senile dementia occurs particularly in old age. It is between these periods that the insanities due to arteriosclerosis are apt to develop. The diagnosis may be quite impossible, and cerebral arteriosclerosis may modify the clinical picture of general paresis and of the psychoses of the devolutional period.

**Diagnosis.**—The question of the diagnosis of arteriosclerosis in general is fully considered elsewhere. The four cardinal points—(1) thickening of the peripheral vessels; (2) signs of hypertrophy of the left ventricle; (3) heightened blood pressure; and (4) a slight and variable amount of albumin in the urine—should be kept in mind and always looked for when the nervous symptoms suggest cerebral arteriosclerosis. The condition of the retinal vessels is of especial significance, for although it cannot be said that alterations in them are always indicative of changes in the cerebral vessels, their condition furnishes the best objective evidences that we have. The ophthal-

[1] *Rev. of Neurol. and Physchiat.*, 1906.

moscopic picture is quite characteristic, and has been well described by a number of authors.[1]

**Treatment.**—The treatment is that of general arteriosclerosis (see vol. iv, p. 444). It is essential that the condition should be recognized early, and that treatment should at once be directed to the vascular condition. If the causes are known, measures should be instituted to remove or counteract them as far as possible. Thus, when syphilis is an etiological factor the indication is plain. Usually, however, it is impossible to determine what has brought about the alteration in the vessel walls; whether the high arterial tension is a cause or effect, and if it be a cause, upon what it depends, and whether the alterations in the kidney are primary or secondary.

**Aneurisms of the Larger Cerebral Arteries.**—True aneurisms of the larger cerebral arteries are relatively uncommon. In Crisp's list, as quoted by Osler (vol. iv, p. 459), among 501 aneurisms, intracranial aneurisms occurred only 7 times. Beadles[2] was able to collect the records of 555 cases of such aneurisms found after death. He classified these in four groups: "(1) Those in which the first indication of a cerebral lesion has been an apoplectic attack due to rupture of the aneurismal sac. (2) Those in which a fatal apoplexy has been preceded by symptoms suggesting a cerebral tumor or other cerebral lesion. (3) Those in which there have been indications of a cerebral tumor only. (4) Those that gave rise to no symptoms whatever during life, and the aneurism was discovered accidentally after death."

In over one-half of the cases the first symptoms noted were those of apoplexy, and even in those cases in which symptoms did occur these were often trivial, or such as might have been attributed to cerebral arteriosclerosis, such as dizziness, headache, mental weakness, and even insanity. Indeed, not a few cases of cerebral aneurism have been unexpectedly found at autopsy in patients dying in insane hospitals. As aneurismal dilatations depend upon structural changes in the walls of the vessel, arteriosclerosis, or syphilitic disease, similar changes are apt to be present in the walls of the other vessels of the brain, and, as would be expected, not infrequently other lesions are present due to these vascular changes but entirely independent of the aneurism, and it is to these that many of the symptoms may be due.

The general symptoms of brain tumor, violent headache, vomiting, and choked disk, are at times present, but the frequency of their absence is remarkable. Headache particularly seems to be a rare symptom, and choked disk has been only occasionally noted, but Beadles could find but few notes of an ophthalmoscopic examination having been made. Aneurisms of the basilar artery give rise more often to focal symptoms than those on any of the other arteries at the base, these occurring in almost one-half the number of cases. This is due to the fact that aneurisms in this situation may implicate the cranial nerves or the important nervous paths which run close to the surface in the pons and medulla. Aneurisms from the posterior communicating, posterior cerebral, and intracranial portion of the internal carotid, and the middle cerebral, have given symptoms with the relative frequency in the order named, but these symptoms are almost never so characteristic as to allow of a local diagnosis. Indeed, it is only in rare instances

[1] de Schweinitz, *International Clinics*, 1907, i, 177; Marple, *Med. Rec.*, March 16, 1907, 423.

[2] *Brain*, 1907, xxx, 285.

that the diagnosis of a tumor can be made, and that such a tumor is an aneurism is a deduction that is practically not justified. The classical symptoms which were supposed to indicate an intracranial aneurism, the subjective noises in the head, and the presence of a vascular murmur which could be heard through the skull, are so rarely present as to be of little value. Beadles was particularly interested in this point and was able to find only about "a dozen cases recorded in which some more or less definite sound has been heard in the head of the patient." In none of these was there any record of the head having been examined for an objective murmur, and in all his reading he was able to find only two cases of "uncomplicated, true, intracranial aneurism in which a murmur had been heard by the examining physician. One of these was an aneurism of the vertebral, and the other an aneurism of the cavernous portion of the internal carotid." The fact that it is so rarely possible to make an accurate diagnosis of an aneurism may perhaps be partially explained by the small size which they usually reach. An aneurism larger than an ordinary marble is rare; an aneurism reaching the size of a small hen's egg is a very large one; and Beadles, in speaking of an aneurism, which he so describes, remarks that there have been probably less than a dozen larger. On the other hand, many aneurismal dilatations have ruptured relatively early, when they were no more than small, soft sacs and produced but little pressure. It is, in fact, the slow-growing, thick-walled aneurisms that have usually given symptoms. In 339 of Beadles' 555 cases, death occurred following a rupture of the aneurism, and it is apparent that their chief importance is in relation to apoplexy, and may perhaps better have been considered in connection with the miliary aneurisms found so frequently in this condition.

**Obliteration and Rupture of the Cerebral Vessels.—Thrombosis and Embolism.**—The subject of thrombosis and embolism has been fully considered (vol. iv, p. 503). The reader is also referred to Welch's article in Allbutt's *System of Medicine*, and only certain points which are of especial interest in the present connection will be referred to here.

**Etiology (Thrombosis).**—The occurrence of thrombi in the vessels of the brain, as in the vessels of the body generally, is conditioned by two factors, the first relating to an abnormal state of the vessel walls themselves, and the second, concerning the blood itself, its character, and its rate of flow.

The changes in the vessel walls are of prime importance, and are believed by many investigators to be essential factors in the production of thrombi. These changes are usually those of arteriosclerosis, and are particularly active when they have affected the intima of the artery, as in the formation of an atheromatous plaque. In arteriosclerosis all the vessel walls may be thickened, and the lumen may be markedly encroached upon, so that the formation of even a small thrombus obliterates it. This, however, is more apt to occur in that type of arterial disease associated with syphilis. It seems certain that biochemical alterations in the blood itself may predispose to thrombosis, but whether this ever occurs in a vessel whose walls are normal is a disputed question, and just what these chemical changes are is not known. In the acute infections of childhood, in typhoid fever, septic infections, influenza, etc., thrombosis of the cerebral arteries has occurred. In many of these, however, the thrombi have contained the specific microörganisms of the infection, which also have been found in the vessel walls themselves, so that even here the primary change may have been in the arterial walls.

The blood may also carry larger particles, about which, when lodged, for instance, at the bifurcation of a vessel, thrombi may develop.

The altered condition of the blood in chlorosis seems to predispose to thrombosis, but the thrombi in this condition usually affect veins, particularly the cerebral sinuses. The change in the rate of flow of the blood is of importance, for when the other conditions favoring the formation of thrombosis are present, a slowing of the blood current may be the determining factor. When this happens thrombi are more apt to occur. This seems to be especially so in those localities where the shape of the arterial channel causes eddies or little whirlpools in the general arterial current. Such places may depend on the normal, anatomical arrangement of the bloodvessels, but are more apt to be due to the changes in the vessel walls, for instance, those which produce local dilatations or definite aneurisms. In this manner it is possible, at times, to account for the frequency with which certain special arteries are apt to be occluded. Cerebral thrombosis is generally stated to be more frequent when the circulation as a whole is feeble, due to weakness of the action of the heart or to some other cause.

*Age of Onset.*—The age at which we may look for cerebral thrombosis depends upon the conditions which have been stated. As it at times occurs in association with the acute infections, we may find it in this relation at any period of life, and it is probable that many of the cases of infantile hemiplegia are due to this factor. Arteriosclerosis is usually a disease of advanced life, and so thrombosis due to this cause is uncommon before the fortieth year, but in the later periods of life the conditions favorable to its occurrence are almost constantly present.

Syphilis is a disease usually acquired in early adult life, and the involvement of the cerebral arteries, leading to their occlusion, may occur at almost any time after the primary infection. The writer has seen it occur in the first six months; and it appears that more cases develop in the first than in any other year after the primary lesion, and that they decrease in frequency with every following year. Cerebral thrombosis due to this cause is, therefore, a disease of the prime of life, and most cases occur between twenty and forty.

**Etiology (Embolism).**—The emboli which plug the cerebral arteries are derived from thrombi which have developed in the heart or the bloodvessels somewhere else in the circulatory system.

As emboli originating in the peripheral veins are usually stopped in passing through the capillaries of the lungs, those which lodge in the brain are, in the great majority of cases, derived from somewhere between the beginning of the pulmonary veins and the entrance of the arteries into the cranial cavity. The most common source is the heart itself, as from vegetations on the mitral valves, and they are more frequently associated with mitral constriction than with any other cardiac condition. Emboli are also dislodged from the aortic valves or from thrombi on the arterial walls, and at times, although more rarely, are formed of bits of calcareous matter detached from an atheromatous plaque. If we include microörganisms circulating in the blood as emboli, they may enter the circulation at any point, and pass through the relatively large capillaries of the lungs to be lodged in the capillaries of the brain; but these are not usually so considered.

*Age of Onset.*—There is no particular age at which cerebral emboli are apt to occur. This depends upon the time of life in which the conditions,

favorable to their origin, are present. These are for the most part those conditions which produce valvular heart disease, and often occur early in life, but may be present at any time. Embolism is perhaps, on the whole, most frequent between late childhood and middle life.

**Etiology (Cerebral Hemorrhage).**—The rupture of a cerebral vessel depends primarily upon the weakness of its walls, but also, to some extent, upon the pressure of the blood within them. Under normal conditions the vessel walls are competent to withstand any strain that is put upon them, and it is probable that they must be diseased before they rupture. Rupture of aneurisms, occurring on the relatively large cerebral vessels, is a not infrequent cause of intracranial hemorrhage. Various changes may so weaken the wall of a vessel that it gives way, and in rare instances hemorrhages have even taken place from arteries affected with syphilitic disease—a process which is much more apt to give rise to thrombosis.

The great majority of hemorrhages, however, occurring within the substance of the brain, are due to rupture of tiny aneurisms upon the small vessels. Since Charcot and Bouchard, in 1864, called attention to these miliary aneurism as a cause of cerebral hemorrhage, they have been almost constantly found whenever looked for. They are little dilatations, many of which can just be seen with the unaided eye, having a diameter from 0.1 to 1 mm., and very many of them may be present, scattered along the nutrient vessels of the brain, but particularly along those which nourish the basilar nuclei. There has been much dispute concerning the exact pathological process upon which they depend, which coat of the artery is first diseased and what the process is. Their relationship to arteriosclerosis and atheroma of the arteries at the base of the brain is also in dispute. This relationship is very close, although not absolute, for they may occur independently of any observable change in the larger vessels, and marked arteriosclerosis may be present in these vessels without the occurrence of miliary aneurisms on the smaller arteries. von Monakow[1] concludes that Virchow was right when he said that one could find in elderly individuals, the subjects of cerebral hemorrhages, dilatations of various kinds on the cerebral vessels of every caliber. These dilatations are not necessarily associated with any marked alteration of the vessel wall, and they may occur without atrophy of the media. von Monakow points out that it is more or less a matter of choice what dilatations are to be considered as miliary aneurisms, and that it is not always possible to distinguish them sharply from other dilatations. Rupture may also occur in vessels whose walls have undergone a hyaline degeneration, or those which have undergone alterations secondary to a mechanical injury. All these conditions he considers as exceptions, and concludes: "By far the most common and perhaps the only certain cause for *spontaneous* cerebral hemorrhage must, according to my view, be sought in the bursting of miliary aneurisms. These begin with a degeneration of the muscular coat; an endarteritic process is not essential for their formation." A very recent review of this condition has been given by A. G. Ellis,[2] who studied the cerebral arteries in 31 cases of spontaneous cerebral hemorrhage. His conclusions differ somewhat from those given above, and are, in brief, that the lesion is primary in the intima,

[1] *Gehirnpathologie*, second edition, Vienna, 1905, p. 1105. Full literature.
[2] *Proc. Path. Soc. Phila.*, September, 1909, p. 197.

beginning apparently in the elastic layer, and differing no wise from that in arteriosclerosis. The other coats may be progressively involved, and so weaken the wall that it ruptures, or the intima may give way at one point and permit blood to pass between the coats of the vessel, forming a dissecting aneurism. False aneurisms may follow both these processes, and it is in this way that the miliary aneurisms of cerebral hemorrhage occur. Spontaneous cerebral hemorrhage occurs both from the rupture of miliary aneurisms and the rupture of vessels without previous aneurismal dilatations.

All the effective causes in the production of arterial disease may be included among the predisposing causes of cerebral hemorrhage, and need not again be referred to in detail. It is interesting to note how some families show a strong hereditary tendency to the occurrence of cerebral hemorrhage at a relatively early period of life. This hereditary weakness of the vascular system may be shown by other vascular accidents—the early occurrence of general arteriosclerosis, angina pectoris, and the frequency of sudden death—even when apoplexy has not been particularly common in the family. Renal disease with hypertrophy of the left ventricle and an increased arterial pressure—a combination of symptoms which is so commonly associated with arteriosclerosis—is frequently present in cases of cerebral hemorrhage. The increased arterial tension is believed to be an important agent in the production of the vascular changes, and is also a factor in the actual bursting of the vessel. Any great increase of the pressure of the blood within a vessel whose walls have been weakened would naturally tend to cause a break, and so increased arterial pressure is given as a common, exciting cause of cerebral hemorrhage. This increase may be produced during a sudden muscular effort or a fit of anger—conditions which increase the activity of the heart. If the outflow of the blood from the cranial cavity be impeded, as when there is a great rise of pressure on the venous side of the circulation, as, for instance, during strong expiratory effort, the pressure within the veins throughout the brain is increased, and this increase is extended to the capillaries and even to some extent into the arterioles, and may produce their rupture. This is still more likely to occur when combined with an increase of arterial pressure, as during strong muscular effort. Such conditions are believed to be present during the intense respiratory spasms of whooping cough; when intense muscular efforts are made, the breath being held, as in lifting heavy weights, straining at stool, etc.—all circumstances under which cerebral hemorrhage is said to be common. It will, however, be shown later how infrequently one is able to discover any such exciting cause.

*Age of Onset.*—Cerebral hemorrhage is an uncommon occurrence before the fortieth year, but it may occur at almost any time in life. It is not an infrequent complication of difficult births, but these cases may, for the most part, be considered traumatic, resulting from the pressure on the child's head. True cerebral hemorrhage, however, does seem to occur during the first years of life, and the liability to it increases with each decade.

**Pathology (Cerebral Softening, Encephalomalacia, Thrombosis, Embolism).**—The effect of the occlusion of a cerebral vessel depends upon how completely the blood is shut off from that portion of the brain supplied by the vessel implicated. There is an extremely rich anastomosis between the vessels at the base of the brain, and theoretically the obstruction of a vessel

emptying into the circle of Willis should have no effect, but unfortunately the channels which connect the various parts are not always present, or if present are not always of the normal size. Even when the anatomical relations have been quite normal, certain of the connections may have become partially or completely obstructed. It is, therefore, impossible to be quite sure of the effect of occlusion, either by disease or ligature of the large vessels. On dogs it has been possible to tie both internal carotids and both vertebrals, provided a sufficient time has intervened between the various ligatures. In these cases the brain receives its arterial blood from the anastomosis with the spinal system. In man, on the other hand, the ligature of even one internal carotid is a procedure attended with considerable amount of risk. Serious brain symptoms have occurred in about 25 per cent. of the cases, and death has followed in nearly 10 per cent. (Jordan[1]). Certain of these results may possibly be avoided if the closure of the vessel is done slowly so as to allow the collateral circulation to be established, and it might be possible to ligate safely both common carotids in man if this procedure were followed and if sufficient time elapsed between the two ligatures (Leonard Hill), but abnormalities of the circle of Willis, congenital or pathological, must always be taken into account.

The larger arteries after leaving the circle of Willis supply different areas of the brain, but there is more or less free communication between the vessels of these areas, and it is quite possible to inject the whole brain from any one vascular trunk. The ease with which this is done depends upon the normal condition of the arteries, and in brains of older people, when arterial changes become more common, it may be impossible to do so completely, and the occlusion of one of the larger arteries, at its origin from the circle of Willis, is usually followed by more or less softening. Cases have been recorded, however, in which such occlusion has occurred without any softening having followed. When a thrombus extends, as it commonly does, so as to implicate the origin of the nutrient vessels which enter the base of the brain, the softening constantly occurs in the gray nuclei which they nourish; for these vessels are end arteries.

The changes which occur in the brain substance, following a closure of its nutrient vessels, have given rise to much discussion. The highly differentiated nervous tissue is probably more susceptible to a decrease in its vascular supply than any other tissue in the body, and at times a destruction of this tissue occurs from the blocking of an artery, which is apparently connected with other vessels by free anastomosis, as, for instance, one of the pial vessels. In general, necrosis occurs whenever the vessels which enter the brain substance, the so-called nutrient vessels, are blocked. The area of ischæmic necrosis which follows varies in position and size, depending upon the number of vessels occluded.

If the brain be examined shortly after the vascular occlusion has occurred, the necrosed area will be found to have a white or a reddish appearance, being more apt to be red when situated in the cortex of the brain, where the vascular supply is particularly abundant. There is usually marked œdema which may extend beyond the boundaries of the softened region, and be so great as to cause a definite swelling of a considerable portion of the brain. In certain cases this œdema is the only objective finding to account

[1] *Verhandl. der Deut. Gesellschaft f. Chir.*, 1907, xxxviii, 83; Becker, *ibid.*, p. 623.

for the marked symptoms which have followed a vascular occlusion, as in death after ligature of the common carotid.

The microscopic appearance of the necrosed area is quite characteristic. It is only in its centre that there is a complete disintegration of the tissue. Here one finds a diffluent mass composed of the detritus of the nervous elements, and every now and then a compound granular corpuscle. Around this the tissue shows more or less marked irritative reaction, the bloodvessels are very prominent, due particularly to proliferation of the cells of their walls and the collection of leukocytes in the surrounding tissue. Compound granular corpuscles are extremely common; newly formed neuroglial elements are present, and there is a greater or less amount of blood, either in the vessels or free in the tissue; or there may be actual small hemorrhages. The color of the infarct depends upon the number of these red blood cells. The nervous elements in this region are found swollen and in various stages of destruction. If examined after one or two weeks the processes of repair are evident. The red blood pigment is becoming altered, the phagocytes, compound granular corpuscles, leukocytes, etc., have taken up and are removing the destroyed tissue, and there is a distinct increase of the supporting tissue, the neuroglia, and the true connective tissue. If the area is a small one this process may entirely replace it by tissue which usually contains minute cavities scattered through it. In larger areas there is a tendency to cystic formation with more or less definite walls. The process which appears to have the most clinical bearing is the œdema, for it is to this that many of the symptoms may be due. It may be so great as to cause a dangerous increase in the intracranial pressure and account for definite pressure symptoms. To this also may be due many of the initial focal symptoms from which the patient recovers. It is a process which occurs frequently in the nervous tissue, and many different theories have been advanced to account for it. Cannon,[1] in a study of the effects of trauma on the brain, has paid particular attention to this point, and comes to the conclusion that the process depends upon the altered osmotic relations due to changes in the nervous tissue, secondary to vascular disturbances.

When the thrombi which occlude the vessels are infected with pyogenic microörganisms, either from having arisen secondarily to implications of the walls of the vessels by these organisms, or having been formed around thrombus from an infected source, as from ulcerative endocarditis, there may be the production of an abscess in the brain.

**Pathological Anatomy of Hemorrhage.**—When a hemorrhage occurs from the rupture of an artery of fair size within the brain, blood is thrown out under pressure, nearly equal to that of arterial pressure and much above the cerebral pressure, which is about equal to venous pressure. The surrounding nervous tissue is broken up by the mechanical impact of the blood. The extent of the destruction varies, of course, with the amount of blood thrown out and the character of tissue into which it is thrown. The white matter is more easily disintegrated than the gray matter, and a hemorrhage in it is apt to produce a larger area of destruction than when confined to the gray substance. The size of an intracranial hemorrhage varies greatly. It is usually no larger than a walnut, but it may at times occupy almost the whole of the cerebral hemisphere, or, on the other hand, be very small indeed.

[1] *Amer. Jour. of Phys.*, 1901, vi, 91.

In the tissue about the actual clot there are usually minute hemorrhages which give it a blood-stained appearance, and a hemorrhage of any considerable size is nearly always surrounded by an area of more or less intense œdema. Here, as in cerebral softening, the œdema may be an important factor in the production of the symptoms, and it is not at all uncommon to find softened areas in the neighborhood of a hemorrhagic focus. If the patient survives the initial shock, processes of repair begin very quickly. There is an endeavor to absorb the clot from the periphery and the formation of a cyst. At first the walls of the cyst are formed of a loose tissue, but this tissue becomes firmer as it gets older, and in cysts of a year or more standing it is a firm connective-tissue capsule, which has on its inner surface a layer of cells of various kinds, stained with the remains of blood pigment. The rapidity of this cyst formation varies greatly, but even in relatively small foci the blood clot can usually be found up to four or five weeks, after which time the cyst formation becomes more and more definite.

## APOPLEXY.

The symptoms following acute vascular lesions of the brain, whether the process be the rupture of a vessel or its occlusion, are in many respects identical; and clinically it is often quite impossible to determine which process has been effective. For this reason the symptomatology of these different processes is considered together under the general term of apoplexy.

**Use of the Word in Literature.**—The word apoplexy has a most interesting history. It was used by the Greek and Latin medical authors, and originally meant "to strike off or be disabled by a stroke." It appears in middle English, and Chaucer uses it near the beginning of his "Nonnes Priestes Tale," in describing the virtues of the widow. He says, among other physical characteristics:

> "The goute lette hir no-thyng for to daunce
> Napoplexye shente nat hir heed
> No wyn ne drank she, neither whyt ne reed"

It was in general literary use in the time of Queen Elizabeth, and Shakespeare makes Falstaff (Henry IV, Second Part, Act I, Scene II) use it as an interesting subject of conversation, likely to distract the attention of the Lord Chief Justice:

> "Fal. And I hear, moreover, his Highness is fallen into the same whoreson apoplexy.
> Ch. Just. Well, God mend him!—I pray you let me speak with you.
> Fal. This apoplexy is, as I take it, a kind of lethargy, an't please your lordship; a kind of sleeping in the blood, a whoreson tingling."

Apoplexy was adopted into falconry in 1614 to characterize a disease of hawks, due to too much grease or blood in the head.

Thomson in his *Castle of Indolence* (1748, Canto I, Stanza LXXVII), at the end of four stanzas, showing a remarkable medical knowledge, and, indeed, claimed by his friend Dr. Armstrong, has the following:

> "The sleepless Gout here counts the crowing cocks,
> A wolf now gnaws him, now a serpent stings;
> Whilst Apoplexy cramm'd Intemperance knocks
> Down to the ground at once, as butcher felleth ox."

Apoplexy was used in medicine to signify a set of symptoms—a more or less sudden and complete abolition of consciousness with loss of feeling and motion, respiration being maintained. As Sydenham has it: "Profound sleep, utter loss of sense and motion, with the exception of that necessary for respiration." It came to be used almost synonymously with coma, and Abercrombie constantly uses such expressions as "The patient was found in complete apoplexy." The apoplectic attack was often spoken of as a stroke or a fit. After it became generally recognized that the cause of apoplexy was commonly a hemorrhage in the substance of the brain, the pathological condition itself began to be called apoplexy, and in the early part of the last century the term was extended to signify any sudden interstitial hemorrhage, as "pulmonary apoplexy," "renal apoplexy," etc. Todd and most of his contemporaries used the term apoplexy as indicating hemorrhage into the brain, but Trousseau, in his clinical lectures, insists upon its original symptomatic meaning. At the present time the best English usage is reverting to the original meaning, although the word even yet bears with it a distinct idea of hemorrhage. In the present article apoplexy is used more nearly in its original symptomatic sense, and made to include the cases due to obstruction of the cerebral vessels as well as those following a rupture.

**Historical.**—The sudden striking down of a person in apparent health, with the abolition of consciousness, loss of all voluntary power and all evidences of sensation, this being followed promptly by death, or, after a time, a partial recovery with more or less permanent paralysis, made a clinical picture impossible to overlook. The ancient physicians recognized it and usually thought that it was due to some disturbance of the brain. Hippocrates says (*Aphorisms*, Section VI, No 57): "Persons are most subject to apoplexy between the ages of forty and sixty;" and his experience in the treatment of the condition seems to correspond with those of subsequent observers, for he says (*Aphorisms*, Section II, No. 42): "It is impossible to remove a strong attack of apoplexy, and not easy to remove a weak attack." The explanation given of the condition varied with the belief and theories in regard to the seat of consciousness and voluntary motion.

Galen (130 A.D.) had a fairly clear notion of the brain. He taught that the nerves of sensation were soft and came from the brain proper, while those issuing from the cerebellum were firm and were devoted to motion. He believed that the action of the brain consisted in inspiration and expiration of the vital spirit. The nerves he regarded as composed of hollow fibers through which the vital spirit passed. Anything that interfered with the flow of the vital spirit caused apoplexy. This he thought of as being due to some sort of pressure or a collection of phlegm that clogged up the channels, etc.

Sydenham says: "This disease (I suppose) proceeds most ordinarily from a gross, thick, phlegmatic humor, which either obstructing the capillary arteries of the brain doth hinder the free access of the blood for the supply of animal spirits, or else being protruded out of the same arteries into the cortex of the brain, doth obstruct the passage of the animal spirits. Sometimes the apoplexy is caused by an extravasation of blood out of some of the capillary arteries, and an effusion thereof upon the brain, whereby the like obstruction of the animal spirits is produced, whilst all the passages in the brain are stopped, partly by obstruction, and partly by pressure from

the load of blood lying upon it; in the like manner as apoplexies are caused by the contusions upon the brain by falls."[1]

The beginning of our modern knowledge of the pathology dates from the investigations of Wepfer (1620 to 1695), who first clearly pointed out in 1658 (de Apoplexia, Schaffhausen) the association of cerebral hemorrhage with apoplexy. He based his conclusions on four autopsies, in each of which a cerebral hemorrhage was found. He also recognized that anything which interfered with the influx of blood to the brain might cause apoplexy, and refers to certain small fibrous bodies which he found in the carotid and vertebral arteries, and also in the heart, as being able to so obstruct the circulation to the brain that an apoplexy would follow.[2] This was very shortly confirmed by a number of investigators, Valsalva (1666 to 1723), Morgagni (1682 to 1771), and especially Boerhaave (1668 to 1738) and his school, and it is from this time that the association between the words apoplexy and hemorrhage dates.

Since then a tremendous literature has accumulated on the subject, but very little advance was made in explanation of the condition until early in the last century. Cullen (1710 to 1790) may be considered as representing the best view at the beginning of this period. He says in his *First Lines of the Practice of Physic:* "Apoplexy is that disease in which the whole of the external and internal senses and the whole of the voluntary motions are in some degree abolished; while respiration and the action of the heart continue to be performed." He gives as proximate cause anything that interrupts the motion of the nervous power, either from or toward the brain. This interruption may be caused by some compression of the origin of the nerves, or by something obstructing the "mobility of the nervous power."

He believes that compression may be brought about by trauma, tumors, or "by the blood being accumulated in the vessels of the brain, and distending them to such a degree as to compress the medullary portion of the same. By fluids effused in different parts of the brain or into the cavity of the cranium, and accumulated in such a quantity as to occasion the compression we treat of." The effusion is of two kinds: either blood poured out from rich vessels, or a portion of the serum. He believed that the chief cause of apoplexy was an obstruction to the venous outflow, and gives the usual conditions that bring this about; a stooping posture, a tight ligature about the neck, obstruction of the ascending vena cava, disturbances of the heart, and forced expiration, etc. That apoplexy might follow an increased afflux of blood to the brain he believed. He points out that, as no lymphatics have been discovered in the brain, the ordinary absorption of blood is through extremities of the veins, and that if there is a resistance to the motion of blood in the veins of the brain, there may be an accumulation of serous fluid in its cavities, and consequently a condition producing apoplexy. He speaks but little of the conditions seen at autopsy. The close association between palsy and apoplexy[3] is dwelt upon, and the similarity of certain phenomena accompanying or following an epileptic fit to the apoplectic state is pointed out.

[1] This was taken from a manuscript believed to be extracts from Sydenham's *Physick Books*, and to have been dictated by Sydenham himself.

[2] See Donley, *The Johns Hopkins Hosp. Bull.*, 1909, xx, 1.

[3] Caldwell says that they are the same disease. Palsy is partial apoplexy; apoplexy universal palsy.

This he thinks could not be due to compression, but to "a certain state of immobility of the nervous power, produced by certain circumstances in the nervous system itself"—a striking modern view. His theory that the diseases of the human body were due to affections of the motions or moving powers of the animal economy fitted better here than in many other places.

As more attention was paid to pathological anatomy, the idea that the condition of the bloodvessels of the brain was an important predisposing factor in the production of apoplexy, began to make its appearance. Morgagni had already announced the belief that the usual cause of the rupture of a vessel was an aneurismal dilatation. He seems to have come to this conclusion from having observed a case of apoplexy in a patient who had an aneurism on the back of each hand, which had developed after intense palpitation of the heart, accompanied by hemicrania and loss of vision. The apoplexy he assumes to have been due to the rupture of a relatively large aneurism which had pressed on the optic thalamus. He looked for small aneurisms on the smaller vessels, but was unable, even after many dissections, to determine them.

Cheyne[1] says that even after the clot and softened brain tissue have been washed away "by means of a syringe or by a patient use of a camel's-hair pencil . . . all along the walls of the irregular cavity (the hemorrhagic focus) show many vessels not larger than a human hair ending in small clots of blood, and he (the anatomist) will sometimes find the same appearance on distant parts of the same brain." Cheyne believed these little clots to be evidences of rupture of numerous small vessels caused by a great and simultaneous action of the arteries. He did not believe that they could be associated with atheromatous changes, although Baillie (1793) had announced that in spontaneous hemorrhage "the vascular system of the brain will be almost always found diseased." Baillie gives a good description of atheroma of the arteries at the base of the brain, and concludes: "Were the internal carotid arteries not subject to the diseased alteration of structure which has been described, diffusion of blood within the cavity of the cranium would be very rare." Cheyne's view was that the hemorrhage was due to rupture of many of the smallest arteries within the substance of the brain. This rupture, he thought, depended upon an excessive activity of these vessels, and he argued that this activity would be augmented by the impulse of blood conducted to them through the larger vessels, and that this would naturally be greater if the walls of these larger vessels were normal than if they were diseased. This argument, taken with the fact that the sections, although showing atheroma frequently, did by no means do so constantly, led him to the firm belief that there was no definite relationship between alterations in the bloodvessels and cerebral hemorrhages.

One is tempted to think that Cheyne must have seen the miliary aneurisms described fifty years later. The method which he used for examining the brain was well calculated to reveal their presence, and he appears to have developed it for the purpose of either confirming or denying Morgagni's hypothesis of the presence of some such structures. That he overlooked them, or possibly misinterpreted what he actually did see, may

[1] *Cases of Apoplexy and Lethargy*, London, 1812.

be explained by the bias which his theory of multiple hemorrhages from the smaller bloodvessels gave to him.

Venous congestion, which was to Cullen the most important cause of cerebral hemorrhage, played only a very unimportant part in Cheyne's theory. He recognized that as any resistance to the circulation of an organ caused an increased activity of its arteries, "any great hindrance, therefore, to the return of blood from the brain might induce the peculiar action of the arteries, and, in a person predisposed, end in the fit." The distention of the veins, found so commonly at autopsy, he believed to have occurred during the death agony. Cheyne recognized, with most other observers, that in certain cases of apoplexy no hemorrhage could be found in the brain, and he distinguished serous from sanguineous apoplexy, although he acknowledges that he knows but little of the pathological anatomy of the former, and seems to doubt the propriety of including under this head many of the cases that are so reported.

Abercrombie[1] (1781 to 1844) wrote extensively from 1818 concerning apoplexy, and certainly more completely than any other previous author in English. He distinguished three forms—simple, in which no anatomical lesions were found; serous, in which there was a more or less extensive collection of fluid, either outside or within the brain; and sanguineous, in which there was a definite hemorrhage. He does not, however, endeavor to make any sharp clinical distinction between them, and thought that anatomically they passed from one into the other. He recognized clearly the formation of cysts from a hemorrhagic focus, and the common association of arterial disease with apoplexy in general. He described paralysis following softening of the brain, and believed that it was associated with some vascular disturbance; he records cases which illustrate this, and in one describes a condition of the bloodvessels which might very well have been thrombosis. His chief service, other than his clear clinical descriptions, was in directing attention to circulatory conditions, and he appends a chapter in which he records his "Conjectures in Regard to the Circulation of the Brain." In this he is particularly taken up in applying the newer view, that the total quantity of the blood within the skull varied but little, and the changes that occurred were simply in the relation between the arterial and venous blood. He, as did Cullen, put most stress on changes on the venous side of the circulation.

In this connection it is interesting to refer to Marshall Hall's Croonian Lectures in 1851, on "The Threatenings of Apoplexy and Paralysis, etc.," in which he calls particular attention to venous congestion as a cause of paroxysmal attacks of apoplexy, which he associates with epilepsy, and distinguishes from apoplexy due to organic brain lesion. The essential cause of this condition he believed to be a plugging of the venous outlet from the brain, due to primary, spasmodic contraction of the muscles of the neck, and he thinks mild emetics are the appropriate drugs for treatment. He points out that these spasmodic attacks of apoplexy may be the warnings of serious organic apoplexy.

Passing over many interesting articles, among which may be mentioned James Copeland's monograph (*Palsy and Apoplexy*, London, 1835), which contains many references to the literature, we shall go directly to probably

[1] *Pathological and Practical Researches on Diseases of the Brain and Spinal Cord.*

the most important English contribution at this time, the lectures of Todd.[1] He confined the term apoplexy to the hemorrhagic focus and regarded the vascular changes as the chief cause both of cerebral softening and hemorrhage. He dwelt on the association between changes in the peripheral arteries and those in the central nervous system and was well aware of the relationship between cardiac disease and apoplexy, as well as the association of both with diseases of the kidneys. He calls particular attention to the importance of disease of the smaller bloodvessels. He, however, thought it a mistake to consider that the associated cardiac hypertrophy and increased action had anything to do with the rupture. Indeed, he believed that the "actual force with which the blood circulated in the morbid arteries was most probably less than in health." Cerebral softening was due, according to him, to a lack of proper blood supply, and he divided it into white or atrophic softening, and red or inflammatory. He knew that white softening might follow the ligature or plugging of a common carotid, and refers to the recent observations of Kirkes in England and Virchow in Germany, who had described cerebral embolism associated with valvular heart disease, but he thinks that this could not be always a cause of the plugging of a vessel, and says: "I should be more disposed to refer it to a coagulum formed in the artery, promoted by an altered nutrition of its walls—arteritis, if you choose to so call it—and connected with a rheumatic or other morbid state of blood." These seem to have been later views of his, for, in general, he believed that cerebral softening usually occurred slowly and that it of itself did not cause paralytic symptoms; these being due either to rupture of the weakened nervous tissue, or the occurrence of a hemorrhage within the softened area, which he believed to be frequent.

Todd's great service, however, was his careful study of the paralytic symptoms, particularly the association of muscular spasm, and he divided hemiplegias according to the muscular tension of the limbs: (1) Those which were flaccid; (2) those showing primary rigidity; (3) those in which the rigidity developed later. The first might pass over into the third group. Hemiplegia, in which the paralysis was flaccid, he believed to be due to cerebral softening; primary rigidity was an evidence of irritation of the blood clot on the ruptured nervous tissue, or of an inflammatory condition of the meninges on the surface of the brain; late rigidity was brought about by the irritation of the contracting scar tissue formed in a softened area. He refers to the observations of Turck, who described for the first time the occurrence of secondary degeneration, and he recognizes the importance of these observations in the explanation of late rigidity.

Up to this time the interest of observers had been largely directed toward the pathological anatomy, with but little attempt to correlate the symptoms with disease of various parts of the brain, and it is obvious that no real advance could be made in this direction until the localization of functions in different parts of the brain had been established. It was obvious to these acute observers that the symptoms associated and following an apoplexy varied, and that in all probability this was due to some difference in the location of the diseased focus. As the anatomy of the brain was studied more carefully, and the distinction between sensory and motor nerves had

[1] *Clinical Lectures on Paralysis, Certain Diseases, and Other Affections of the Nervous System*, second edition, London, 1856.

been demonstrated by the experiments of Sir Charles Bell and Magendie, and the observations and theories of Gall and Spurzheim had astonished the scientific world, more and more attention was given to this part of the subject, and Romberg,[1] in his chapter on cerebral paralyses, pays particular attention to this part of the subject, but says nothing noteworthy about the pathological anatomy. He recognized that the general features of cerebral paralysis were undoubtedly modified according to the seat of the disease, but says that very little is known about it; the most firmly established fact "is the law of crucial conduction," that is, that a lesion on one side of the brain causes disturbances on the opposite side of the body. This had been noted from the earliest times, and Hippocrates said: "And for the most part convulsions seize the other side of the body, for if the wound be situated on the left side, the convulsions will seize the right side of the body." This was accepted and was used as the basis for the supposition that the tracts, wherever they might be, which conducted the influence from the brain to the muscles, crossed the middle line; but for many hundred years this belief waited for an anatomical explanation. It, however, aroused the interest of many investigators, and just two hundred years ago du Petit François-Pourfour[2] announced the results of his experiments and anatomical investigations. His experiments confirmed the general belief and his dissections demonstrated for the first time the decussation of the pyramids, of which he gives a most excellent drawing. An Italian observer, Mistichelli, made a somewhat similar observation about this time. These anatomical facts did not gain wide acceptation, and even the clinical fact that a lesion on one side of the brain produced crossed effects, was doubted by certain writers in the early part of the last century, and numerous cases were quoted to prove the contrary. Cheyne says that his dissections tend to confirm the observation that the blood is generally extravasated in the hemisphere opposite the side of the body which is paralyzed. This was the general view, but even as late as 1851, Craigie said, "Palsy of the opposite side, although frequent, is not an invariable result of injury of the brain." It is interesting that Sir Charles Bell in his earlier dissections missed the decussation of the pyramids, and that it was the anatomical demonstrations of Gall, in the early years of the last century, which called especial attention to this fact.

Romberg studied his cases carefully and endeavored to correlate the anatomical findings with the symptoms before death, but was unable to draw any definite conclusions, other than the law of crucial conduction, and he undertook some experiments which unfortunately were inconclusive, and in summing up his knowledge he could only say, "There is only one conclusion at which we have arrived which seems to be a rule with scarcely any exception, viz., that when paralysis of a leg or of an arm is the result of a cerebral affection its seat is in the cerebrum." At that time the corpus striatum and the optic thalamus were believed to be the structures from which motor impulses had their origin, and it was thought by some that the thalamus had to do with the arm and the corpus striatum with the leg. Romberg was able to find no confirmation of this.

The French observers wrote extensively on the subject. Cruveilhier

[1] *Lehrbuch der Nervenk. des Menschen; Sydenham Soc. Trans.*, 1853.
[2] *Lettres du médecine, etc.*, Namur, 1710.

made many important observations of the pathological findings in cases of apoplexy, and Trousseau's lectures may be taken as a fair representation of the views of that time. He does not particularly concern himself about the pathological anatomy, except to state that apoplexy may be the result of cerebral softening, of congestiom, of an accumulation of fluid within the ventricle of the meninges—serous apoplexy—or of an extravasation of blood, while at times no lesion may be found to cause it. He insists that the word apoplexy should not be used to mean a cerebral hemorrhage. By apoplexy he meant an absolutely sudden loss of consciousness, that is, falling as if struck down; he says that this is, in fact, a rare accompaniment of cerebral hemorrhage, and that there are almost always premonitory symptoms. He does not consider questions of exact localization in his lectures on apoplexy, but in those in which he considers aphasia he refers in detail to arguments for and against the localization of the faculty of speech in the posterior part of the third frontal convolution. It was around this point that a fierce scientific war was being waged. Under the influence of Flourens, and in revolt against the absurdities to which the phrenological school had gone, the orthodox opinion was that the cortex of the brain acted as a whole, and that there was no such thing as localization of function in it. Influenced by a number of autopsies on patients observed by himself, and the report of those observed by others, Trousseau became a staunch champion of the view which Broca had advanced, and it was largely due to Trousseau's advocacy that the belief that in the third left frontal convolution speech processes are localized became so quickly and so fully accepted.

In 1868 J. Hughlings Jackson[1] considered the corpus striatum and the optic thalamus as the highest point of the motor tract, and says that a lesion above the level of the ventricle, that is, above the upper limit of the motor tract, need not reveal itself by very definite symptoms, such as hemiplegia or loss of speech. Even later, in 1873, when he made his brilliant deduction from the study of focal epilepsies—that the movements of the different parts of the body were definitely localized in the cerebrum—it was in the optic thalamus that he thought this primarily occurred, although he assumed that the movements were again represented in the cortex, and that stimuli arising here could influence the centres in the corpus striatum and so cause focal epilepsy. Although Fritsch and Hitzig in 1870 had announced that stimulation of the cerebral cortex was followed by movements, it was not until their results had been confirmed and extended by many other observers that the motor function of the cortex was generally accepted.

As can be gathered, the morbid state of the bloodvessels had assumed a greater and greater importance in the pathological explanation of acute vascular lesions of the brain. Virchow's study of embolism, between 1846 and 1856, and the studies of thrombosis by many investigators made possible the proper understanding of cerebral softening, which had earlier been believed to be due usually to inflammation. The beginning of the full modern conception of the pathology of cerebral vascular lesions may be said to date from the discovery by Charcot and Bouchard of miliary aneurisms as the common cause of intracerebral hemorrhage.

**Occurrence.**—The frequency of apoplexy may be approximately judged from a study of mortality statistics. *The Seventh Annual Report of the*

[1] *Reynolds' System of Medicine*, Phila., 1868.

*Census Bureau* gives the figures for 1906. These cover those States whose mortality reports are deemed sufficiently reliable (a population of 40,996,317, almost one-half the population of the United States). There were 658,105 deaths from all causes, a death rate of 16.1 per thousand. Among these 29,434 were ascribed to apoplexy, and 6033 to paralysis. Many of these latter cases must have been due to previous attacks of apoplexy, although, as the report says, some were probably of an entirely different character. They are, however, considered together, and the total of 36,367 has probably as much chance of being below as above the right figures, for there are many cases included in the mortality statistics under the heading of softening of the brain and a general paralysis of the insane, which should properly be classed among the apoplexies and are not included in these figures. The total of 36,367 deaths from apoplexy and paralysis gives a death rate of 88.7 per 100,000 of the population.

In Baltimore, with a population of 553,699, there were during the year 544 deaths from apoplexy and paralysis. The expected deaths among such a population would be 490. The increased death rate depends upon the number (124) of deaths in colored individuals from these causes (29.5 per cent. of the deaths were in negroes, whereas the negro population forms not quite 19 per cent. of the whole population of Baltimore). This greater liability of the negroes to die from apoplexy and paralysis is quite parallel with their greater liability to diseases of the circulatory system. The *Census Report* shows that among 100,000 of the population of Baltimore there were 161.3 deaths from such causes among the whites and 327.4 among the blacks.

**Etiology.—Predisposing Causes.**—These are the same as those in the primary vascular lesions, and have been considered to some extent in the foregoing chapters. Whatever tends to produce these lesions has a bearing on the production of apoplexy.

**Race.**—The figures from the *Census Report* indicate that there is a distinct tendency in the negro race to diseases of the circulatory system, and also a greater liability to apoplexy. In the writer's series there were 152 negroes among 674 individuals suffering from hemiplegia in whom the race was noted.

**Sex.**—This appears to have no influence. The proportion among 1000 of the aggregate population within the Registration States was in 1900 505.8 men and 494.2 women, while among 1000 deaths from apoplexy and paralysis in 1906 there were 504.5 men and 495.5 women. In the records of the Johns Hopkins Hospital there is a marked preponderance of males, there being 489 males and 251 females, but in this hospital many more men are treated than women.

**Age.**—The vascular changes upon which the apoplexy depend are in a large proportion of cases conditioned by incidents of the life of the individual, and increase in frequency with each succeeding decade, and the frequency of apoplexy also increases with each decade.

In the mortality statistics a large number of apoplexies are recorded as occurring under one year of age. These must have been composed largely of traumatic apoplexies occurring at the time of birth, and give an undue preponderance to this time of life. The following tables, condensed from the *Census Report*, show the occurrence of apoplexy in the various decades, and gives a fair idea of the relative frequency of apoplexy at the different

periods of life. The frequency is, of course, not the same thing as the liability, for the number of cases which occur at any period of life must depend upon the number of people alive at that age. The estimated population in the Registration States in 1907 was distributed among the decades according to the ratios found in the census of 1900, and compared with the number of deaths in each decade which were reported during 1907. The error introduced cannot be great. The relative liability in the different decades is expressed in proportion, taking the liability in the second decade as 1. The increase in liability with each succeeding decade is certainly striking.

TABLE I.—*Table Condensed from the Census Report for* 1907. *Deaths from Apoplexy and Paralysis.*

| Decade. | Cases. | Proportion per 1000 of Deaths. |
|---|---|---|
| First | 670 | 20.1 |
| Second | 121 | 3.6 |
| Third | 370 | 11.1 |
| Fourth | 998 | 30.0 |
| Fifth | 2,250 | 67.7 |
| Sixth | 4,836 | 145.4 |
| Seventh | 8,491 | 255.3 |
| Eighth | 9,877 | 297.0 |
| Ninth | 5,100 | 153.4 |
| Tenth | 544 | 16.4 |
| Total | 33,257 | 1000.0 |

TABLE II.—*Table Condensed from the Census Report for* 1906.

| Population of Registration States in 1907, distributed according to ratios found in 1900. | | Deaths from apoplexy and paralysis in 1907. | Liability per 100,000 population. | Relative liability. |
|---|---|---|---|---|
| First decade | 6,984,883 | 670 | 9.6 | 5 |
| Second decade | 6,204,278 | 121 | 2 | 1 |
| Third decade | 6,260,922 | 370 | 5.9 | 3 |
| Fourth decade | 5,171,628 | 998 | 19.3 | 10 |
| Fifth decade | 3,760,575 | 2,250 | 59.8 | 30 |
| Sixth decade | 2,550,957 | 4,836 | 189.6 | 95 |
| Seventh decade | 1,579,307 | 8,491 | 537.6 | 269 |
| Eighth decade | 734,016 | 9,877 | 1345.6 | 673 |
| Ninth decade | 180,990 | 5,100 | 2817.8 | 1409 |
| Tenth decade | 13,406 | 544 | 4057.9 | 2029 |

The cases occurring in each decade differ somewhat as to their etiology, and each decade will be considered separately. In the Johns Hopkins Hospital there have been 292 cases of apoplexy, *i. e.*, patients suffering either from the acute condition or the subsequent paralysis, in the medical wards among 23,048 medical admissions. In the Out-patient Department 448 cases of hemiplegia and other results of acute cerebral lesions were

seen. The cases which were registered in both hospital and dispensary were included only among the hospital cases. The analysis of these cases is used as a basis for the subsequent discussion. The following table shows their occurrence in the various decades:

TABLE III.

| Decade. | Males. | Females. | Total. |
|---|---|---|---|
| First | 58 | 77 | 135 |
| Second | 16 | 9 | 25 |
| Third | 43 | 19 | 62 |
| Fourth | 73 | 29 | 102 |
| Fifth | 88 | 49 | 137 |
| Sixth | 99 | 44 | 143 |
| Seventh | 86 | 19 | 105 |
| Eighth | 20 | 5 | 25 |
| Ninth | 6 | 0 | 6 |
| Total | 489 | 251 | 740 |

**Decade I.**—The cases of hemiplegia in the first decade are of especial interest, and in large part are the result of processes peculiar to that period. In the *Census Report* for 1906 a large proportion of cases of death from apoplexy and paralysis in the first decade occurred in the first year, 621 among a total of 961. These must include those dying shortly after birth with symptoms indicating injury to the brain. In the Johns Hopkins Hospital records there were 135 cases of hemiplegia which had appeared in the first decade. Of these, 32 are classed as congenital, and in 16 the onset occurred during the first year. This can be only relatively accurate, as it is often impossible to determine from the history whether the paralysis was due to conditions present at birth or to those which may have occurred later. When the parents believed the paralysis to have been always present, and when nothing in the history indicated a later onset, it has been classed as congenital. In other cases the history is so clear that little doubt can be possible.

*Congenital hemiplegias* are in most instances due to accidents occurring at birth. As the head of the infant passes through the pelvic canal of the mother it is subjected to great pressure, which in the vast majority of cases the head is well adapted to withstand, but at times it is great enough to injure the brain, either directly or, as is more often the case, by means of a hemorrhage within the skull. Very rarely the paralysis is the result of a prenatal process, *i. e.*, something that has affected the brain of the fetus during its development and before labor has begun. Prenatal pathology in so far as it relates to the central nervous system is an obscure subject. Ballantyne,[1] in his large work, has but little to say about it. Gross defects of development in the brain and spinal cord have, indeed, been frequently described. These may be so great as to lead to the entire absence of the whole central nervous system, or of the brain or of a large part of it, but

[1] *Antenatal Pathology and Hygiene, The Fetus*, Edinburgh, 1902, p. 388.

as these monsters do not live for any considerable time they need not be considered in this connection.

The brains of newborn infants show at times relatively small defects, even when they may be otherwise normally developed. These consist of cavity formation involving to a greater or less extent the cortex and the white matter beneath it (porencephaly), atrophy of the convolutions (microgyria), and sclerosis in certain portions of the cortex or tracts within the central nervous system, the pyramidal tracts especially. Quite similar conditions are frequently found at autopsies performed on cases of infantile cerebral palsies, both in those which are presumably congenital and also in those which appeared during the first years of life. In fact, it is to the study of these end results of various initial lesions that our knowledge of such conditions is mostly due.

The term *porencephaly* has been used to describe a great number of conditions. It was first introduced by Heschl, in 1859, but it was not until 1880 that especial attention was drawn to it by Kundrat, from whose monograph our knowledge may be said to date. Kundrat and most other writers believe that these cavity formations are usually due to some prenatal condition, especially to some disturbance in the circulation. These defects occur most often in the central convolutions, and the bloodvessels supplying this region have often, but not always, been found diseased. The vascular obliteration has been ascribed to thrombosis and embolism occurring during fetal life, but there do not seem to be many facts to substantiate this. Inflammation of the brain substance, with secondary softening, is also given as the starting point of the process. That this is actually a cause in prenatal cases seems also to be based on very few facts. Seitz[1] after a critical analysis of the various cases which have been adduced in proof of this belief, first advanced by Virchow[2] in 1867, has come to the conclusion that the lesions which have been described under the heading of congenital encephalitis have resulted from cerebral hemorrhage, or are extra-uterine from specific infections, possibly also intoxications. He concludes that congenital encephalitis in the sense that Virchow used it has not been proved to exist. On the other hand, he believes that porencephaly found at birth may be due to a cerebral hemorrhage occurring during fetal life and records the case of a baby who died five hours after an easy birth. At the autopsy a large defect in the anterior and middle parts of the cerebrum was found. The white matter was replaced by a cavity which involved also the cortical substance. The basal ganglia, the pons, cerebellum, and medulla oblongata, were all normally formed. Microscopic examination of the walls of the cavity gave evidence of a former hemorrhage. The mother of the child had received a severe trauma to the abdomen when four months pregnant, which Seitz believed caused an intracerebral hemorrhage in the fetus.

The occurrence of hemorrhage within the fetal brain is certainly rare. Seitz was unable to find in the literature any case quite similar to his, and Ballantyne, in proof that such a condition may occur, refers to only one case, that recorded by Osler.[3] It was in the fetus of a woman six months pregnant, who died of typhoid fever. The hemorrhage was in the left

[1] *Arch f. Gyn.*, 1907, lxxxiii, 701.
[2] *Virchow's Archiv*, 1867, xxxviii, 127.
[3] *Teratologie*, 1895, ii, 13.

cerebral hemisphere and had broken through into the ventricle. There was also general œdema of the brain.

Gibb[1] records a case of a stillborn child who had a contracture of the left arm and leg and in whose right cerebral hemisphere an old clot was found above the lateral ventricle. The mother of the child had received a severe blow on the right side of the abdomen three months before labor. This was followed by repeated hemorrhages until labor was complete. These cases are sufficient to show that intracerebral hemorrhage does occur during fetal life, both following a trauma to the abdomen of the mother and during the course of an acute illness. Freud refers to a clinical case recorded by Gaudard, in which the mother received a severe blow on the abdomen in the sixth month of pregnancy. The child was born with paralysis of the right arm.

The intra-uterine causes of microgyria and lobar sclerosis are not well understood, but these conditions are generally believed to follow some disturbance of the blood supply to the developing brain. Lack of development of the pyramidal tracts, which is included among the prenatal causes of the cerebral palsies, appears to be the result of the cerebral defect, and not an independent condition.

As has been stated, the great majority of *congenital cerebral palsies* are due to accidents during birth. The association of difficult labor, asphyxia, and premature birth with the spastic paralysis of children was pointed out by Little,[2] but that these paralyses were due in most cases to hemorrhage within the cranium was first clearly shown by Sarah J. McNutt.[3] Little was well aware of the frequency of intracranial hemorrhage at the time of birth, but, as at that time the cortex was not known to have anything to do with motion, he believed that the spastic paralysis was due to hemorrhage about the spinal cord. McNutt showed that the hemorrhages incident to birth on the convexity of the brain were apt to be most intense about the fissures of Rolando, and she assumed that the atrophy of the central convolutions was due to a bilateral hemorrhage limited for the most part to this region.

In most instances these cases are associated with abnormal labor, but large and even fatal intracranial hemorrhages do occur when the birth is apparently normal and easy. Marked changes of pressure in the circulation of the infant must take place during labor, especially when placental circulation ceases and that through the lungs is established. When the child is asphyxiated the circulatory conditions present are particularly favorable for the rupture of vessels, a fact upon which Little laid great stress. On the other hand, an intracranial hemorrhage may be the cause of asphyxia.

Small capillary hemorrhages within the various tissues of the infant are an almost constant accompaniment of birth. Seitz,[4] in speaking of this fact, refers to an investigation by Paul, who made an ophthalmoscopic examination on 200 newborn children, and found retinal hemorrhages in a large proportion of cases—in 50 per cent. of those in which the mother's pelvis was contracted, in 40 per cent. when the labor was protracted and

[1] *Lancet*, 1858, ii.

[2] *Trans. of the Obstet. Soc. of London*, 1861, iii, 293; also *Treatise on Deformities*, 1853.

[3] *Amer. Jour. Med. Sci.*, 1885, clxxvii, 58; *Amer. Jour. of Obst.*, 1885, xviii, 73.

[4] von Winckel, *Handbuch der Geburts.*, 1907, iii, 49.

complicated, in 40 per cent. when the labor was premature but otherwise normal, and in 20 per cent. of normal labors.

Undue pressure on the baby's head, either by a disproportion between its size and that of the mother's pelvis, or the faulty application of forceps, may cause direct injury to the brain, but usually the pressure forces the bones of the baby's skull under each other and causes rupture of one of the venous sinuses, or of veins emptying into them. The hemorrhages which follow are usually meningeal (subdural or subarachnoidal) and are rarely within the cerebral substance itself.

Seitz arranges them in three types. The most frequent are those in which the hemorrhage comes from the longitudinal sinus or from veins entering it. In these cases the clot surrounds the convexity of the hemisphere and extends to its base, but does not reach to the other hemisphere, nor does it usually pass below the tentorium. If the rupture is of the transverse or direct sinus, the chief part of the clot is under the tentorium, surrounding the cerebellum and medulla oblongata and passing into the spinal canal, and it may extend around the cerebral hemisphere on the same side and to a less extent on the opposite side. In the third and least common class the hemorrhage is found in the ventricles. It may completely fill these and extend to the medulla oblongata and into the spinal canal without passing to the meninges about the exterior of the cerebrum or cerebellum.

Some idea of the frequency of these meningeal hemorrhages during birth can be gathered from the records of autopsies done on stillborn babies and on those which have died shortly after birth. Peterson,[1] in speaking of this point, refers to the figures given by Litzmann, who found 35 cases of meningeal hemorrhage among 161 stillborn children. Parrot, in 34 autopsies of the newborn, found 5 with blood in the arachnoid cavity and 26 with hemorrhage into the subdural space. H. R. Spencer, in 130 autopsies on stillborn children, found 53 instances of hemorrhage from the pia and arachnoid.

Although the hemorrhages occurring during labor are in the great majority of cases meningeal, they do occur at times within the brain substance. For instance, Weyhe, in an analysis of the records of the Pathological Institute at Kiel, in 959 autopsies in young infants found 122 cases in which intracranial hemorrhages had occurred. In many cases there were hemorrhages in more than one location. A clot was found 80 times in connection with the dura and 56 times with the arachnoid; in 35 instances it occurred within the substance of the brain, and in 21 within the ventricles. There were 32 instances of relatively large hemorrhages; 5 of these large hemorrhages were intracerebral, and in all but one case it seems fair to assume that the rupture took place at the time of birth, even though the author makes no mention of the character of labor in any of his cases.

**Postnatal Cases.**—Hemiplegia in children due to causes effective after birth may occur at any age, but is much more frequent in the first two years. This is well shown by Table IV, which is compiled from that given by Freud,[2] with the addition of the writer's collected cases. Of the 607 cases recorded as occurring in the first decade, 344 (56.7 per cent.) were in the first two years, and 516 (85 per cent.) in the first half of the decade.

[1] *Nervous and Mental Diseases*, Church and Peterson, 1908, p. 894.
[2] *Die Infantile Cerebrallähmung*, Nothnagel, 1897, p. 44.

TABLE IV.

| | Congenital. | Up to 1. | Up to 2. | Up to 3. | Up to 4. | Up to 5. | 5 to 10. |
|---|---|---|---|---|---|---|---|
| Osler | 15 | 45 | 22 | 14 | 1 | 3 | 9 |
| Wallenberg | 19 | 35 | 29 | 17 | 9 | 9 | 30 |
| Gaudard | 11 | 17 | 7 | 5 | 8 | 2 | 14 |
| Lovett | 7 | 5 | 12 | .. | .. | .. | .. |
| Strümpell | .. | 7 | 8 | 4 | 5 | .. | .. |
| Sachs and Peterson | 22 | 27 | 17 | 16 | 4 | 4 | 8 |
| Gowers | .. | 23 | 23 | 14 | 10 | .. | 10 |
| Freud-Rie | 2 | 3 | 11 | 9 | 3 | 3 | 2 |
| J. H. H. cases | 32 | 16 | 37 | 17 | 8 | 7 | 18 |
| Total | 108 | 178 | 166 | 96 | 48 | 28 | 91 |

The mortality statistics of the *United States Census Report* for 1906 show the same thing more strikingly, for of the 961 deaths recorded as due to apoplexy and paralysis in the first decade, 882, or nearly 92 per cent., occurred in the first five years. Although these figures are exclusive of stillborn children, they probably include many children who died shortly after birth from accidents incident to it, for 621 died within the first year and only 127 in the second.

It is also probable that a considerable number of the cases of hemiplegia included in Freud's table as developing in the first and second years were really congenital. For instance, of the 16 Hopkins cases in the first year, the histories of 5 merely stated that the paralysis was noticed at varying times during the infancy: once when the child was one month old, once when four months, twice when six months, and once when eight months; in none of these cases could the parents be sure that the condition had not existed since birth. In 5 of the 37 cases in the second year the disability had become apparent when the time arrived for the children to begin to walk, and here again no definite onset could be determined, so that these 10 cases might fairly have been added to the 32 congenital ones.

**Etiology.**—The cerebral changes which underlie the hemiplegias developing after birth and during infancy are, in general, the same as those which are present at later periods of life, but are influenced by special factors. The vascular system of a child is much less apt to show permanent changes, and on this account the spontaneous rupture or the occlusion of a blood-vessel is less apt to occur. On the other hand, acute infections seem especially prone to affect the brains of the young. The table represents the etiology of the cases occurring in the first decade. These are distributed by years in the first half of the decade and in the last half are considered together.

TABLE V.

Congenital, 32; probably congenital, 10; total, 42.

| Postnatal cases. | Year of onset. | | | | | | Total. |
|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 5 to 10 | |
| Acute infectious diseases: | | | | | | | |
| Whooping cough | 1 | 1 | 1 | 1 | 1 | 1 | 6 |
| Typhoid fever | . | 1 | 1 | . | 1 | 2 | 5 |
| Measles | . | 2 | . | 1 | . | 1 | 4 |
| Diphtheria | . | 1 | . | . | . | 3 | 4 |
| "Sore throat" | . | . | . | . | 1 | . | 1 |
| Croup | . | . | . | 1 | . | . | 1 |
| Gastro-intestinal trouble | . | 1 | 1 | . | . | 1 | 3 |
| Marasmus | 1 | . | . | . | . | . | 1 |
| Pneumonia | . | 1 | . | . | . | . | 1 |
| Vaccination | . | 1 | . | . | . | . | 1 |
| "Fever, brain fever," etc. | 4 | 4 | 3 | 1 | 1 | 2 | 15 |
| After convulsion without other cause: | | | | | | | |
| With marked brain involvement | 1 | 3 | 1 | . | . | . | 5 |
| Convulsions at onset | 2 | 12 | 3 | 1 | . | 1 | 19 |
| Developed in course of epilepsy | . | . | 1 | . | . | . | 1 |
| Congenital syphilis | . | . | 1 | 1 | 2 | . | 4 |
| Brain tumor | . | . | . | . | . | 3 | 3 |
| Trauma: Falls | . | 2 | 2 | 1 | . | . | 5 |
| Injury to head | . | 1 | . | . | . | . | 1 |
| Heart disease: Chorea and endocarditis | . | . | . | . | . | 1 | 1 |
| Congenital heart | 1 | . | . | . | . | . | 1 |
| Poisoning | 1 | . | . | . | . | . | 1 |
| Hysteria | . | . | . | . | . | 1 | 1 |
| Without determined cause | . | 2 | 3 | 1 | 1 | 2 | 9 |
| Total | 11 | 32 | 17 | 8 | 7 | 18 | 93 |

*Hemorrhage.*—The rarity of intracerebral hemorrhage in childhood is commented on by most writers on the question. Meningeal hemorrhages are more common, but even these, when not associated with trauma or with one of the acute infectious diseases, are rare. Sachs[1] thinks that hemorrhage occurs more frequently than most authors admit, and, indeed, he seems to regard it as the most common cause of these paralyses, even of the cases that develop after birth. The bleeding, he believes, is from a cortical vessel, although it may be from a vessel within the depths of the brain.

Alterations in the vascular walls may occur in childhood as an early degeneration, or more frequently as the result of some acute infection. Sachs cites von Recklinghausen's statement that fatty degeneration of the cerebral vessel is not uncommon in children. The rupture of such a diseased vessel must account for some exceptional cases of apoplexy of the first decade, and a very few of the writer's cases may best be so explained. However, there is no record in Johns Hopkins Hospital of the occurrence of a postnatal intracerebral hemorrhage in a child under ten years. This

[1] *The Nervous Diseases of Children*, second edition, 1905.

hospital admits relatively few children, and the rarity of such cases can be better judged from other records. Holt says that he has seen but one case. Among 830 autopsies at the Babies' Hospital in New York, the records of which the Staff assisted me in examining, there is no such case, and Dr. John Howland writes that "in the last 1416 autopsies at the Foundling Hospital there has been just one intracerebral hemorrhage." Sidney Phillips[1] reports the occurrence of a spontaneous hemorrhage into the pons varolii of a girl, aged fourteen months. The child, who was apparently well when put to bed at 3 P.M., was found comatose an hour afterward, and remained so for the fourteen days which she lived. No cause for the hemorrhage was discovered.

Among the 93 postnatal cases there are 9 classed as developing without known cause, *i. e.*, so far as could be discovered; the paralysis developed more or less suddenly in children who were not sick at the time, very much as we are in the habit of seeing an apoplexy occur in the late decades. The history of the onset in five of these cases is so incomplete that some or all of them may well belong to other classes. Two of the four remaining cases are of particular interest. The first was a girl, aged six years, who, while in apparent health, fell, paralyzed on her right side, but was not unconscious for any considerable time. The paralysis improved, but intense athetosis followed in the arm, for which she was admitted on six different occasions, the last time when she was twenty-five. The other case occurred in a girl, aged nine years. She was at her desk in school when she fell unconscious, and remained in a more or less dazed condition for nearly two weeks. The paralysis was on the left side and cleared up well. When twelve she had a second attack, which followed a right-sided headache, and came on while she was walking around her room. She fell, paralyzed again on her left side, but without loss of consciousness. The power again returned, but rather more slowly, and here, as in the first case, athetosis followed. She is now twenty-four, and is still under observation, having at no time shown evidence of any cardiac involvement. The onset in these cases was so characteristic of a vascular lesion that it was difficult to think of any other explanation. No sources of emboli were found, and we have been led in each case to assume the occurrence of either a rupture or a thrombus of one of the arteries in the substance of the brain.

In the other two cases the paralysis came on at the eighteenth or nineteenth month. In one case the onset was sudden; the baby was unconscious for ten days and was paralyzed on the right side. In the other case the onset was rather gradual, in the course of one or two days. The right side was paralyzed.

Hemorrhage has been the assigned pathological condition in many of the cases of hemiplegia associated with definite etiological factors. This would seem justified in certain of the cases developing after injury to the head when the skull had not been fractured, although even here the possibility of an encephalitis must not be forgotten. With fracture of the skull, hemorrhages, particularly from the meninges, are, of course, common, no matter at what time of life the accident occurs, and even when the fracture is not apparent it cannot always be excluded. In only one of the traumatic cases was the skull known to be injured, and in that there was a perforating

[1] *Lancet*, 1909, i, 1680.

wound.[1] In one case the fall may have been merely coincident with the attack of paralysis. This was in a girl, aged two years, who had been sick with some fever and who fell from a little cart. When picked up she was paralyzed on the left side. In the other four cases the falls were of sufficient gravity not to be overlooked as etiological factors.

Cerebral hemorrhage during infantile convulsions has been assumed as the cause of many of the early hemiplegias. The fact of the sequence of convulsions and paralysis is most striking, and the supposition that blood-pressure changes, incident to the fit, may have caused rupture of the blood-vessels is attractive. That the vessel walls, on the other hand, are in the vast majority of cases amply able to withstand any increase of pressure incident to the most severe convulsions, is shown by the rarity of their rupture in cases of epilepsy. Capillary hemorrhages are, indeed, frequently found in patients dying in the status epilepticus, but these do not appear to be large enough to account for paralytic symptoms. In none of the cases of this series developing at any age did the paralysis follow a fit in an epileptic, unless two cases in the first decade may be so considered. The more striking of these was that of a hemiplegic idiot seen at seven, who began to suffer from convulsions at eight months, which recurred every two or three weeks. When two years old the patient had a severe attack lasting four hours, after which his mother noticed that he was paralyzed on the left side. He did not walk again for more than a year. A single convulsion recurred six months after the attack of hemiplegia, and there was then an interval of four and a half years, after which he was subject to numerous seizures.

Including these cases, there were 54 of the 93 postnatal cases in which the paralysis was ushered in by convulsions, but in no other of these was there any record of the child having had convulsions previously. Among these 54 cases many developed in association with various etiological factors; 14 with acute infectious diseases and 10 from a variety of causes, congenital syphilis, brain tumor, trauma, etc., and the primary cause of the cerebral lesion must have varied much—hemorrhage, thrombosis, embolism, and inflammation. In the remaining 30 the cerebral involvement, as judged by the sudden onset of the convulsions in apparent health, seems to have been the primary lesion. In 14 of these there was either more or less prolonged fever, or definite evidence by protracted coma, etc., to show that the brain was seriously involved from the first. The discussion as to the pathology of infantile hemiplegia has, in a great measure, centred around cases similar to these, *i. e.*, those cases in which the paralysis developed acutely in apparently healthy children, with more or less fever and evidence of involvement of the brain. The convulsions are either completely or, for the most part, unilateral, and one must assume a cause that acts more or less locally from the first, and it is obviously unsatisfactory to explain the paralysis as the result of the convulsions without giving an adequate cause for these, especially as there is reason to doubt the efficiency of convulsions in themselves to cause permanent gross lesions. Gowers assumes, as the most satisfactory explanation of these cases, "a sudden occlusion of a small surface vein by clot, with the consequent intense congestion and hemorrhagic softening of the region of the cortex."

[1] Had I analyzed the surgical records, without doubt many more cases of infantile hemiplegia due to trauma would have been added.

The many points of similarity between the onset of these cases of infantile cerebral hemiplegia and those of acute poliomyelitis (infantile spinal paralysis) led Strümpell, in 1884, to assume as their anatomical basis a lesion in the brain analogous to that in the cord, which was believed to underlie infantile spinal paralysis, *i. e.*, an inflammation confined to the gray matter of the motor cortex of the brain (polioencephalitis) corresponding to the inflammation limited to the ventral horns in the cord. This theory was advanced at a time when the anatomical data in regard to spinal infantile paralysis were very limited and when there were practically none concerning inflammation of the brain, so that in spite of its great attraction it did not receive general acceptation. Since then, however, a great deal of work has been done on the inflammatory conditions of the central nervous system, and the views in regard to these conditions have been very much broadened. We now believe that infantile spinal paralysis is due primarily to a more or less diffuse inflammation about the vessels of the cord and meninges, involving particularly, although not exclusively, the anterior gray matter. Many cases of acute inflammation of the brain have been described which appear to be quite comparable, involving particularly the cortex. These two conditions occur separately, but at times they are found together, and some involvement of the meninges of the brain seems a constant occurrence in the fatal cases of poliomyelitis.[1]

Strümpell's view that many of the cases of infantile cerebral hemiplegia are due to an inflammation of the brain has come to be widely accepted. He himself, in the 1907 edition of his text-book, considers the cerebral palsies of children under "acute encephalitis." Oppenheim[2] accepts this view as an explanation for a number of cases. These inflammations may be caused by various agents, and encephalitis in children has been found in association with most of the acute infectious diseases. If the many cases of infantile hemiplegia which develop acutely with fever, convulsions, etc., are to be regarded as the result of inflammatory processes in the brain, the thought that they may be due to a special specific organism is evident.

Among the 93 postnatal cases there were 26 instances in which the paralysis developed in association with some one of the acute infectious diseases, 6 in relation to whooping cough, 5 with typhoid fever, 4 with measles, 4 with diphtheria, 3 with gastro-intestinal disturbances, and the other 4 distributed among various conditions. The cerebral lesions in these cases are by no means always the same. Inflammatory processes must account for a number of them, but hemorrhage, thrombosis, and embolism have been found with varying frequency with each of these diseases.

Whooping cough is accountable for more intracranial hemorrhages than any of the other conditions. A great increase in venous pressure during a paroxysm of coughing is the usual explanation, but Neurath[3] believes that changes in the vessel wall are more important than increased pressure. The hemorrhage is thought to be from a meningeal vein. Rhein, however, who analyzed the autopsy findings of 21 cases of pertussis, found the hemorrhage when present most frequently in the substance of the brain. In the case which he reports the lesion was inflammatory, and Neurath refers to a

[1] Rossi, *Nouv. Icon. de la Salpêtrière*, 1907, pp. 122 to 145.
[2] *Lehrb. der Nervenk.*, 1908, ii, 957.
[3] Pfaundler and Schlossman, Phila., 1908, ii, 427.

number of such cases and believes that hemorrhage is less common than is supposed.

Typhoid fever we believe to be accountable for 5 of our cases, although this seems a large number when we consider the relatively few cases of hemiplegia associated with this disease. Smithies was able to collect only 40 cases. The age of 32 of these cases is interesting; 9 were in the first decade, 8 in the second, 13 in the third, and only 2 beyond that time. When one considers how relatively infrequent typhoid fever is in the first decade, it is surely remarkable to find more than one-quarter of the cases of hemiplegia occurring in children under ten. Smithies was able to find the record of 5 autopsies, only one of which was in a case in a child, and the examination was made twelve years after the onset of the hemiplegia. Two of the other 4 cases occurred among our records, and have been reported by Dr. Osler. In all of the cases occlusion of an artery was found, so that from the evidence at hand we must conclude that cerebral thrombosis is the cause of most cases of hemiplegia developing during typhoid fever. It is probably secondary to changes in the artery walls.

*Diphtheria* is accountable for 4 of our cases, or, indeed, 5, if we include the case in which the history simply recorded a severe sore throat. If hemorrhage is the most common cause of the hemiplegias associated with pertussis, and thrombosis the most common cause in relation to typhoid fever, it seems that embolism plays this part in the hemiplegias developing during diphtheria. Thus, Rolleston,[1] in his collection of 65 cases, has found 15 which were completed by autopsies. Among these, hemorrhage was found in 1, thrombosis in 2, embolism in 10, embolism and thrombosis in 1, and atrophy of one hemisphere in 1 case. Escherich[2] records a case with autopsy which showed a lesion of the mitral valve and an embolus in the left middle cerebral artery.

*Measles* is represented in this list by 4 cases, but there is not any case which occurred in association with *scarlet fever*. Concerning this latter disease, Rolleston[3] speaks of the rarity of hemiplegia in relation to it, but he was able to find the records of 57 cases, 8 with autopsy. He concludes that it is usually due to cerebral embolism, but may follow thrombosis, hemorrhage, or acute encephalitis. Under this last heading he thinks uræmic hemiplegia may probably be placed.

Except in the instance of encephalitis, the involvement of the brain during the course of an acute infectious disease is secondary to involvement of the vascular system. The association between emboli and endocarditis is evident, and that the vessel walls themselves may be involved in children during the course of acute infectious diseases has been shown by a number of authors, particularly by Wiesel,[4] and that the occurrence of thrombosis is secondary to this involvement cannot be doubted. The tendency to rupture of an artery, as in whooping cough, is probably brought about by the weakening of the vessel wall through this process, as Neurath has suggested. This arterial disease appears in most instances to be entirely recovered from, but it does at times leave the artery wall permanently

[1] *Rev. Neurol. and Psychiat.*, 1905, p. 722.
[2] *Wien. med. Wchnschr.*, March 2, 1907, p. 478.
[3] *Rev. Neurol. and Psychiat.*, 1908, p. 530.
[4] *Ztschr. f. Heilk.*, 1906, xxvii, 262.

damaged, and so acute infectious disease during infancy may be the predisposing cause of later vascular accidents.

Embolism, as has been seen, is not uncommon in association with acute endocarditis occurring in the course of the acute infectious diseases (diphtheria, scarlet fever, etc.), but it is remarkable how rarely it occurs in chorea, a disease in which the heart is so generally affected. There is but one case in the series, although we have seen a large number of cases of chorea with involvement of the heart. The case was that of a little girl, aged ten years, who had had chorea a year and a half before, which was followed in the next year by rheumatic fever and endocarditis. She had been up about four weeks after this attack, when she fell from a bench, had repeated convulsions, and was paralyzed on the right side with loss of speech. The only other case in which it seems reasonably certain that the hemiplegia was due to an embolus was in a little child, aged one year, who had congenital abnormality of the heart. This organ was on the right side. The sounds at the apex were clear, but over the right second costal cartilage there was a grating systolic murmur. Four weeks before we saw the patient the mother discovered, on taking him up in the morning, that his right arm and right leg were completely paralyzed, the attack having come on during sleep.

The importance of *hereditary syphilis* in the causation of infantile hemiplegia has given rise to a great deal of discussion. Sachs believes that it has very little influence, but other authors give it prominence. In 4 cases of this series it appeared to be the most likely etiological factor and in 1 case there was hardly a doubt. This was in a child whose brother and sister had been treated for congenital syphilis, and who, himself, when twenty-one months old, was seen in the children's department with marked manifestations of the disease. Under treatment he improved, but his general development was retarded. When four years old he had an attack of right hemiplegia, with loss of speech, choked disk, and retinal hemorrhages. He was at this time passing through whooping cough, but this case is not included among those occurring in whooping cough. He improved under specific treatment, but the left optic nerve became atrophied. When five years old he had a second attack, this time on the left side, and speech was again affected. The paralysis improved, but there was a marked mental defect. There was another case in which the history seems clear. The father had syphilis, and a specific eruption developed in the baby when it was two months old. The child was given antisyphilitic treatment by the family physician. When four years old convulsions developed, followed by right-sided paralysis. When seven the child had a painful swelling of the anterior border of the tibia, which disappeared under specific treatment. The other two cases suggest this factor strongly, although perhaps not quite so definitely. The vascular lesion in these cases was presumed to be an occlusion, secondary to syphilitic changes in the vessel walls.

During the course of *brain tumor* hemiplegia is not uncommon, and three such cases occurred on the medical side; but in these the paralysis was of rather slow development, and so probably did not depend upon a hemorrhage into the substance of the tumor.

*Hysteria* may cause a hemiplegia at almost any age, and we have the record of one case, that of a little girl seen in her tenth year, suffering from

her second attack. The first attack had been a year before. She had paralysis of the right arm and leg with typical sensory disturbances. The whole disability cleared up at once under suggestion and faradic stimulation.

**Cerebral Diplegia.**—Closely allied to the hemiplegias which develop early in the first decade are the infantile cerebral diplegias. The same etiological factors, congenital abnormality of the brain, injury to the head during birth, hereditary syphilis, inflammatory conditions, etc., apply to both. In the one case the lesion is unilateral, while in the other both sides of the brain are affected. The two sides of the brain may be unequally involved, and so it is not infrequent to find one side of the body more affected than the other, and only portions of the hemisphere may be injured and corresponding symptoms result. Not infrequently the upper portions of both hemispheres are most involved, and the symptoms are largely confined to the legs (cerebral spastic paraplegia). The lesions occurring before or at the time of birth are more frequently bilateral, and diplegia is the common result of these processes. Later in the decade the reverse is true, and hemiplegia is then much more frequent.

Among 100 cases of infantile cerebral palsies in the Neurological Department of the Johns Hopkins Dispensary, and in which the process was definitely congenital, 68 were diplegia and 32 hemiplegia. There are records of only 10 cases of diplegia which developed after birth. Five of these were in the first year, 3 in association with acute illnesses, measles, pneumonia, whooping cough, and the others with indefinite illnesses. Of those developing after the first year, 2 were with fever, 2 are said to have followed falls, and in 1 no cause could be given. Besides these 78 cases, there were 4 others in which the early history was unknown, and in all the process may have been congenital.

The various *symptoms* of infantile spastic diplegia (Little's disease) are, for the most part, those of hemiplegia, which will be described in detail later, only in this case both sides of the body are involved. Certain symptoms are so pronounced that they require special mention at this time. These are particularly those depending upon the lack of development of the brain, for it is obvious that when the brain has been severely injured early in life it cannot develop normally, and so these unfortunate children show every degree of idiocy. The degree of mental retardation is, in a general way, proportionate to the motor defect, but not always so, and the intelligence of certain patients who have never been able to walk or even to sit up is surprising. It has been suggested that in such cases the lesion is not in the brain itself but in the upper part of the spinal cord. The disparity is more common in the other direction, and one often sees children who show only a slight degree of spastic paralysis, but whose mentality is very low. Indeed, every combination occurs.

As in certain cases of congenital hemiplegia, the disability may not be noticed until it is time for the child to begin to walk, but usually there are symptoms which should attract attention. Directly after the difficult birth there is often trouble in establishing respiration, and not infrequently the child has muscular twitchings or even convulsions. The increased intracranial pressure may be shown by a tense non-pulsating fontanel and a general lack of animation, a higher grade of choked disk than usual, and death may occur rapidly. In less severe cases the baby seems to recover and to perform all of its functions normally. These, it must be

remembered, are largely reflex, depending on the lower centres. Later, when the time arrives for the baby to show certain spontaneous activities, as holding up its head, sitting up, grasping at objects, supporting itself on its feet, learning to crawl, to stand, to walk, and to talk, the parents are distressed to see that these are not acquired. The spastic condition of the legs is often first noticed when the child is several months old by a resistance in the adductor muscles as the mother changes the child's napkins. When the child is held in the standing position this adductor spasm comes out more clearly and the legs are held in extension and firmly together. Indeed, at times the spasticity can be brought out only in this manner. In other cases it is so pronounced that it attracts attention very early (congenital spastic rigidity). Such babies become rigid upon the slightest cause, when manipulated in any manner or when startled by a noise. One mother complained to the writer that the movements of such a child in utero were very different from those of her other children, the child appearing to stiffen out and remain so for some time. This description was given, however, after her baby was a year old, and its truth is doubtful.

Even when the spasticity is not excessive these children learn to walk late; not usually before the third year, and often even much later. The walk is awkward and spastic, and frequently shows the typical crossed leg progression, each leg as it is advanced being thrown across the other by the strong contraction of the adductor muscles. The finer movements of the arms and hands are learned, if at all, with difficulty, and even the coarser movements are performed with marked incoördination. Such children may be entirely unable to learn to talk, and when they do, the articulation is apt to be very inexact, even when the mental development is of such a grade as to make speech possible.

In infantile cerebral palsies, both of the hemiplegic and diplegic type, abnormal spontaneous movements are common; indeed, they are rarely seen in cases which develop later in life. The characteristics of these conditions, athetosis, postparalytic chorea, etc., will be discussed later. Athetosis in its classical form occurs in diplegias, and the condition described as double athetosis is confined to this state. Lewandowsky[1] insists that double athetosis is not simply the involvement of both sides of the body with typical athetosis, but that it is a different condition and depends upon excessive associated movements. Such patients may be quite quiet when undisturbed, but when their attention is attracted, or when they endeavor to perform any voluntary act, the muscles of the face are thrown into the most startling grimaces, the arms are moved about in quick excessive choreiform movements, which suggest strongly those seen in Huntington's chorea. Double athetosis may occur in its most pronounced form when there is but little motor paralysis.

Epilepsy not infrequently develops, although perhaps this is somewhat more common in the hemiplegic than in the diplegic type. The affected limbs may be retarded in their growth, and the whole physical development be stunted. This is not always so, and even in a severe case the body may reach its normal growth.

**Decade II.**—Fewer cases of hemiplegia occur in the second decade than at any other time of life, except extreme old age, and even here the

[1] *Deutsche Ztschr. f. Nervenh.*, 1905, xxix, 339.

liability to these vascular accidents is much greater. The difference between the first and second decades depends largely upon the great number of cases in the first three years of life due to accidents at birth, and the liability of the infant's brain to inflammatory processes. The 25 cases falling in this decade were divided as follows: 6 were due to trauma, and 6 occurred in the course of acute illness, diphtheria 2, meningitis 2, typhoid fever 1, and undetermined illness 1. One other case, that of athetosis, developed after a disease that was said to be meningitis. Two were in relation to brain tumors and 2 were hysterical. The remaining 8 cases are of particular interest, for they developed suddenly in apparent health, and presumably were due to primary vascular lesions. Similar cases occurred in the first decade, but their relative frequency is much greater in the second. This is what would be expected from the increasing number of individuals who must have damaged vascular systems from congenital weakness, acute illnesses, etc. As none of the cases was completed by autopsy, the pathological basis for the paralysis must be largely a matter of inference.

**Embolism.**—Embolism seems almost surely to account for 3 of the 8 cases. The first was in a girl, aged eleven years, who had frequent attacks of sore throat and arthritis. Her paralysis came on suddenly while she was at work in the kitchen; her left leg gave way so suddenly that she fell on the stove. She did not lose consciousness, and was able to keep about for a few hours, but the next morning she noticed that her left arm and leg were weak. There was a typical left-sided hemiplegia, and the examination of the heart revealed stenosis and insufficiency of the mitral valve. The second case was quite similar. This occurred in a girl, aged fourteen years, who had for a number of years been under treatment for attacks of tonsillitis, and who for a month before her admission to the hospital had suffered from acute polyarthritis. The examination of the heart gave definite evidence of endocarditis and she had continuous fever. After being in the hospital for a month her tonsils were dissected out. On the second day after the operation she was unable to speak, and there was paralysis of the whole right side. The third case was that of a colored boy, aged thirteen years, who was awakened early one morning with intense pain in the stomach, vomited, had to be assisted back to bed on account of staggering, went to sleep, but was awakened three hours later with another attack of vomiting, after which he became unconscious and was brought to the hospital the same day. He was comatose, the right arm and leg were motionless, and the left arm and leg were in almost constant, restless movement. The head was turned to the right, and the right leg was stiff. There was mitral insufficiency. The ophthalmoscopic examination showed evidences of increased intracranial pressure, and a decompression was done. Dr. Cushing found the left side of the brain red and dry, resembling a hemorrhagic infarct. The boy recovered with right hemiplegia and aphasia.[1]

**Hemorrhage.**—This appears to account for at least one of the 4 remaining cases, which was in a girl, aged sixteen years, who, since she was nine, had been seen a number of times suffering from attacks of Henoch's purpura. While on her way home from a friend's house she had difficulty in speaking,

[1] In connection with these three cases I may refer to a case already reported in *The Johns Hopkins Hosp. Bull.*, 1901, vol. xii, of embolism of the central artery of the retina in a girl, aged sixteen years, who was suffering from chorea and mitral insufficiency.

and that evening found it hard to eat. There was, however, no weakness of the limbs until the next morning, when the right arm was affected. On the second day she had convulsions, and was admitted to the hospital aphasic and weak on the right side. She had recurrent convulsions, beginning in the right hand; the head and eyes turned to the right, the right leg was next involved, and the convulsions then became general. After each convulsion she was more comatose, and new purpuric areas appeared on various parts of the body. Dr. Cushing operated and found the dura tense and the brain and subpial space of a uniform cherry-red color. Nothing but decompression was attempted. The patient died on the day after the operation, and four days after the onset of the cerebral symptoms. No autopsy was permitted.

Hemorrhage may have accounted for the other 3 cases, although thrombosis cannot be excluded. In none of them could a source for an embolus be found. They occurred suddenly, in 2 instances the paralysis being discovered in the morning, after an undisturbed sleep, and one case, a girl, aged fourteen years, while working in a very hot schoolroom, felt nervous and fell unconscious on her desk. This was followed by paralysis on her right side with speech disturbance. The examination nine months afterward showed the signs of a typical organic hemiplegia. In this case the heart was normal and no arteriosclerosis was discovered.

While this section was in preparation a most interesting specimen was brought to the Pathological Department. This was the brain of a school boy, aged fifteen years, who seemed perfectly well upon going to bed. At 1 A.M. he aroused his roommate by loud screams, and complained of intense pain in the head. He quickly became unconscious, and died at 8 A.M. The autopsy showed an enlarged thymus, lymphatic hyperplasia of the intestine, lymph glands, and spleen, with œdema and congestion of the lungs. The brain showed a large hemorrhage in the right temporal lobe which had ruptured through the cortex. The arteries at the base of the brain appeared to be perfectly normal, and a careful microscopic examination failed to reveal any disease of the cerebral vessels.

The cases which were associated with acute illnesses differ only from those occurring in the first decade in the age of onset, except the two cases associated with meningitis. In one of these the type of meningitis could not be determined, but the other case was proved by lumbar puncture to be acute cerebrospinal meningitis. Hemiplegia is a rare complication of epidemic cerebrospinal meningitis. Castaigne and Rivet[1] report a case in a boy, aged seventeen years, and could find the record of but one other case. They ascribe the complication to encephalitis.

**Decade III.**—The liability to apoplexy in the third decade is about three times as great as in the second. This is due to the influence of syphilis and to the increased number of cases depending upon cardiovascular changes. Pregnancy and the puerperal state account for a certain number of cases, and a few are in association with various pathological conditions (pernicious anæmia, hæmophilia, etc.).

Syphilis was recorded as having occurred in 20 of 62 cases of this series, and there can be no doubt that to this number others should be added. Of 12 doubtful cases, 6 were in negroes—5 men and 1 woman—in whom

[1] *Bull. et mém. Soc. méd. d. hôp. de Paris,* 1909, xxvii, 900.

syphilis is extremely prevalent, but in whom it is always difficult to get a history of the infection, and there were 2 white women, both of whom were probably syphilitic, for one had many miscarriages and the other was a prostitute. The cerebral complication may follow the primary lesion quickly, indeed within the first year, or it may be delayed for a long time, ten or more years. One case occurred at five and one at seven months, and 4 others at about one year after the infection. The average time was approximately four and a half years.

**Thrombosis.**—Thrombosis with consequent softening of the nervous tissue, is the lesion upon which practically all the cases of apoplexy due to syphilis depend. This thrombosis depends upon a specific change in the artery walls, the so-called syphilitic arteritis. Under thrombosis should probably be grouped most of the cases occurring in association with acute infectious diseases, and the cases occurring shortly after childbirth. The hospital list contains 5 of the former and 3 of the latter in this decade. Of the 5 cases occurring during the course of an acute infection, 4 were with typhoid fever, and one with pneumonia. Two of the typhoid fever cases occurred in the hospital, and both died. The autopsy on one revealed thrombosis of branches of the middle cerebral artery. The case which developed during the second week of a severe double pneumonia was possibly due to an embolus, for when the patient was examined six months later his heart showed evidences of serious organic disease. Hemiplegia is an extremely rare complication in pneumonia. No case has occurred in the Johns Hopkins Hospital.

Hemiplegia and other focal manifestations of cerebral lesions occur in association with *pregnancy* and *childbirth*. These cases naturally fall in the third and fourth decades, and in this series 3 of the hemiplegias of the third decade were of this character. Another case, not included, was that of a young woman, aged twenty-two years, in the obstetrical ward. The patient was paralyzed in the seventh month of pregnancy, and then again three days before confinement. The paralysis was in both instances on the left side, and was complete a few days after delivery. The patient entered the hospital just before labor, which was terminated without difficulty. The other 3 cases developed after childbirth, one on the fourth day, one on the ninth, and one a month afterward.

The lesions in such cases vary a great deal, and hemorrhage, thrombosis, and embolism have all been found. von Hosslin[1] has made a careful review of this subject. Hemorrhage is the most common lesion, particularly in those cases associated with marked albuminuria or symptoms of eclampsia. In this association, however, a number of cases have been reported in which the brain showed only œdema, the so-called serous or uræmic apoplexy.

Women with diseased arteries from any cause run special risks during pregnancy and confinement. An interesting illustration of this was a woman who died in the obstetrical ward. She was thirty-five years of age, and when thirty-two had a left-sided hemiplegia, which had no association with pregnancy. When thirty-five she became pregnant, and at the eighth month she fell unconscious, apparently in acute uræmia. She was brought to the hospital, immediately delivered, but died soon after. Autopsy showed

[1] *Arch. f. Psychiat.*, 1904, xxxviii, 730, and 1905, xl, 445.

a softened area due to the old lesion, and a large hemorrhage into the pons varolii. The arteries at the base showed marked sclerosis. The liver did not show the changes characteristic of eclampsia. However, hemorrhage does occur during parturition in women whose arteries show no general disease.

**Embolism.**—von Hosslin found embolism the least common cause, although Meyer-Ruegg believes that it is the most common. The source of the emboli is endocarditis. This may be acute, developing spontaneously during pregnancy, or as an exacerbation of a chronic process; or a chronic endocarditis may be the source of an embolus. Embolic occlusion of a cerebral vessel is the reasonable explanation of the 12 cases of hemiplegia which occurred between the twentieth and the thirtieth year in individuals suffering from organic heart disease. If syphilis be excluded this is by far the most common cause of the hemiplegias developing in the third and fourth decades. Three of these 12 cases developed while in the hospital; 2 in association with ulcerative endocarditis, and the third in a case of rheumatic fever with cardiac involvement. The other 9 cases occurred while the patients were about their usual vocations, and when the cardiac condition had presumably become chronic.

**Hemorrhage.**—This is uncommon before the fifth decade, but even though syphilis may account for the majority of the 12 cases in the third decade, in which no cause could be determined, still some of them are hard to explain in this manner. For instance, a man, aged twenty-one years, who admitted only one exposure to venereal infection, and who denied all knowledge of any such accident, fell while working and was paralyzed on his right side. A physical examination revealed no lesion of the heart, and in such cases, without an autopsy, it is impossible to exclude cerebral hemorrhage. However, in 3 of the 62 cases occurring in this decade, there can be little doubt that cerebral hemorrhage did occur, and in one this was proved by autopsy. The patient was a woman, aged twenty-two years, who had nephritis, and who died eleven days after an operation for decompression. At autopsy a large capsular hemorrhage was found. The other two cases were associated with conditions which make hemorrhage almost certain. In one of these, a white man, aged twenty-eight years, with pernicious anæmia, a right hemiplegia appeared nineteen days before death. The autopsy revealed hemorrhages in the various organs of the body, but unfortunately the brain was not examined. The other case was in a hospital orderly who came from a family of bleeders, and who, was himself a hæmophiliac. A transient right hemiplegia appeared while under observation; he died three years later from carcinoma of the stomach.

**Trauma.**—Trauma accounts for 2 of our 62 cases. One of these is of interest, as the head was not directly affected. The patient, a brakeman, aged twenty-one years, was injured while coupling cars; he was crushed about the chest, and right hemiplegia developed, which may have been due to a hemorrhage secondary to great venous congestion.

**Hysteria.**—Hysteria accounts for 5 of our hemiplegias in the third decade. Four of these were in women and one was in a man.

**Decade IV.**—In this decade the liability to vascular lesions is again greatly increased, being about three times as great as that in the third decade. The influence of syphilis is still marked in the fourth. Of the 102 cases, 32 gave a definite history of syphilis, which almost equals that in the

third decade, being 31.3 per cent. in the former and 32.2 per cent. in the latter. But the group of cases in which no definite cause could be determined is much larger, making 44.1 per cent. In this group many cases must be included which are due to syphilis, although this either has not been recognized or not recorded. An illustration of this is found in a man, aged thirty-three years, suffering from diabetes insipidus, who had an apoplectic attack. Both conditions were believed to depend upon syphilitic disease, although no history of such an infection could be obtained. As other conditions which cause arterial degeneration are constantly increasing with advancing age, the proportion due to syphilis naturally becomes smaller as the age becomes greater, and, therefore, in this decade this group must contain a relatively smaller number of unrecognized syphilitic cases.

The time between the syphilitic infection and the occurrence of the apoplexy in the cases which fall in the fourth decade is, as would be expected, somewhat longer than that which was found for the cases of the third decade. In this series, as accurately as could be determined, it was a little more than eight years. Of the 32 cases, all but 1 occurred in men. This, of course, does not mean that the condition does not occur in women, but only illustrates how difficult it is to obtain a syphilitic history in women.

Acute infectious disease accounts for only 2 cases among the 102 in this decade. In these the hemiplegia developed during typhoid fever, and was presumably due to thrombosis of a cerebral artery. In this connection a private case, not included in this analysis, seen in consultation with Dr. Chrystie, of Bryn Mawr, Pa., may be noted. It occurred in a woman, aged forty-two years, during the course of a protracted fever, the nature of which was never definitely determined, the diagnosis resting between a mild attack of typhoid fever and an unusual influenzal attack. On the fourteenth day the patient had a convulsion affecting the right side, which lasted about eight minutes, and which left her paralyzed on the right side and aphasic. The left eye was blind, due to obliteration of the central retinal artery. In this case the lesion was almost surely a thrombosis of the internal carotid, implicating its ophthalmic branch as well as the middle cerebral artery. The hemiplegic attack had practically no influence on the temperature, which continued to vary between 98.5° and 101° until the twenty-second day of the disease, after which it remained practically normal.

There is one case in the fourth decade which occurred twelve days after the birth of the eighth child, in a woman, aged thirty-nine years; it was associated with a normal puerperium. Two other interesting cases were seen in private practice.[1] These were associated with evidences of toxæmia, and both had hemianopia. In one there was a transient left hemiplegia with permanent sensory disturbances. Cerebral thrombosis was the most probable lesion in these cases.

Bilateral capsular softening, due to thrombosis, was found at autopsy in a colored woman, aged thirty-nine years, who presented a typical picture of pseudobulbar paralysis.

Embolism, associated with heart disease, was the probable lesion in 11 cases—6 men and 5 women. This was demonstrated by autopsies in 3 cases.

Hemorrhage within the cranium was found in 4 cases at autopsy. Two

[1] Both of these were seen with Dr. Hiram Woods, and have been reported by him, *Jour. Amer. Med. Assoc.*, 1908, li, p. 204.

were in negro men who had had syphilis nine and ten years before death, and are included among the syphilitic cases, although this factor was probably but one of the causes of the vascular disease. They were both markedly alcoholic. In one the intracranial hemorrhage was associated with pachymeningitis and arteriosclerosis, and there was evidence of old cortical hemorrhages. In the other case there were marked cardiovascular changes and a large clot in the occipital lobe, which had broken through into the subdural space. The other 2 patients were brought to the hospital unconscious, and very little history could be obtained. In one the hemorrhage had taken place in a glioma, which apparently had given no symptoms. The other was in a man, aged thirty-nine years, who had an enormous intracerebral hemorrhage and general arteriosclerosis.

Spontaneous cerebral hemorrhage is, in fact, generally recognized as being rare before the fifth decade. von Monakow says that before the fortieth year it is extraordinarily uncommon. However, this lesion was probably present in some of the 45 cases in which the history gave no very definite indication as to the cause of the cerebral lesion, as in that of a colored man who was admitted to the hospital several times for chronic nephritis and dilated heart, and who had a number of apoplectiform attacks. In one case of hemiplegia, which had developed rather gradually, a brain tumor was found at autopsy, and in 2 cases the paralysis was definitely hysterical. Both of these occurred in men.

**Decade V.**—Apoplexy becomes much more common in the fifth decade, the liability to it being three times as great as that in the preceding ten years, and more than twice as many cases occur. In the present analysis there are 137 cases within this period. By the time an individual has reached the age of forty many things may have occurred in his life that make for vascular accidents, and it is often difficult or impossible to determine which has been to blame in a particular case.

Syphilis, such an important factor in the third and fourth decades, plays a much less outspoken part in the fifth. There are, indeed, 27 cases in which there is a definite history of a previous infection, but this, in most instances, took place many years before the apoplectic attack, the average time being between sixteen and seventeen years. In many of these 27 cases other conditions were present which may have been effective, and may or may not have been related to the specific infection. This is illustrated by the only case in this group which came to autopsy. The patient, aged forty-seven years, was infected fifteen years previously and had been in the wards on two occasions suffering from symptoms of arteriosclerosis and chronic nephritis. After staying in the hospital three months he became unconscious and a left hemiplegia appeared. There was some improvement in the cerebral condition, but he died after three weeks. The autopsy revealed chronic nephritis, hypertrophy of the heart, acute pericarditis and thrombosis of the right internal carotid artery.

Cerebral softening was found at autopsy in 7 cases dying between forty and forty-nine. It was not always determined whether the occlusion of the bloodvessels was due to an embolus or a thrombus, but in one case, that of a man, aged forty-six years, who died during typhoid fever with evidence of left-sided paralysis, there seems to be no doubt that thrombosis of the middle cerebral artery was the cause of the necrotic area in the right internal capsule. This is the oldest case of hemiplegia associated with

acute infectious disease of which we have record, and is the only one in the fifth decade. In 2 other cases which were associated with chronic nephritis and arteriosclerosis, the multiple areas of cerebral softening were believed to be thrombotic, and there were numerous cases in which the most probable clinical diagnosis was thrombosis.

In a case of diabetes mellitus a left hemiplegia developed two days before death. At autopsy pulmonary tuberculosis, chronic nephritis, acute endocarditis of the aortic and mitral valves, and infarcts in the spleen and kidney were found. There was slight sclerosis of the vessels at the base of the brain, but no macroscopic lesion was found other than a hyperæmic condition of the pia. The probable explanation of the cerebral symptoms was an undetected embolus, which for some reason, an unusual freedom of collateral circulation for instance, had not produced a definite softening. We must also think of the possibility of the case being one of toxic hemiplegia in diabetes mellitus.

**Embolism.**—The heart was noted as definitely affected in 15 cases, and was probably involved much oftener than this. That the hemiplegia was in all these cases due to embolism is, of course, not a just inference, as there were often associated conditions that might have led to thrombosis or hemorrhage.

Cerebral hemorrhage was found at autopsy in 4 cases dying in this decade.

Two cases of hemiplegia, aged forty and forty-two years, occurred after childbirth. In one the attack appeared on the tenth day. The history in the other was indefinite, simply stating that the attack occurred at the time of the last confinement, six months previous to admission.

In 80 cases the hemiplegia developed without determined cause, although in some of them arteriosclerosis was noticed, and evidences of cardiac and renal involvement. These cases must include many spontaneous hemorrhages, cases of thrombosis, and a certain number of embolism.

**Decade VI.**—In this period the liability to apoplexy and also the frequency of its occurrence again increases over the fifth decade. It is interesting that the increase in both these relations is almost exactly the same as that of the fifth over the fourth decade, being three times for the first and twice for the second. In the present series, however, there are only 143 cases as against 137 in the preceding ten years. There were 30 deaths against 19 in the fifth decade.

In this decade it is more difficult to determine the etiological factors, and 94 cases are included in the group of undetermined etiology. Most of these cases showed definite evidences of arteriosclerosis, which may have followed a variety of causes. No apology seems necessary for arteriosclerosis at this time of life.

There were 11 cases in which it was definitely stated that the patient had suffered from syphilis. This, of course, is an evident understatement of the fact. Men between fifty and sixty may have forgotten that they were infected in their early youth, or, believing that such an infection could have no possible bearing on their condition, conceal it; and, on the other hand, the examining physicians, knowing that an early specific infection is apt to be only of slight importance, have not inquired into this feature of the cases as diligently as they should. In these 11 cases the average time of infection preceded the apoplexy by 21.7 years, and it is probable that in

most of them syphilis acted only as one of the causes in producing arteriosclerosis. That syphilis may play its accustomed part at any period of life is shown in 2 of the 11 cases; in one, a man, aged fifty years, a slight hemiplegia appeared while the patient was being treated for tertiary syphilis, he having been infected two and a half years previously. In the other the hemiplegia developed at fifty-five, a year after an undoubted syphilitic infection. In these two cases there was also marked alcoholic excess.

In the only case which came to autopsy there was a history of syphilis twenty years previously. He had marked arteriosclerosis, chronic nephritis, and dilated heart, for which he had been treated in the hospital two months before. He was admitted in a comatose condition, and died in twenty-four hours. An extensive hemorrhage was found in the left cerebral hemisphere, the vessels in the brain being markedly sclerotic. There was no evidence of syphilis at the autopsy.

There were 17 cases in which the heart was noted as being evidently diseased, and in which presumably embolism accounted for the apoplexy. In 4 other cases among 7, in which intracranial softening was found at autopsy, there was a condition of the heart which could have been the source of an embolus. That it is not safe to assume that an apoplexy in an individual who presents the evidence of a cardiac involvement is always due to embolism, is shown by the occurrence of 3 cases dying of intracerebral hemorrhage, in all of which the autopsy showed valvular endocarditis.

Cerebral softening was found in 9 autopsies on patients dying in this decade. Two of these, however, were in cases which had developed in the fifth decade, one due to embolus and one to a thrombus.

Hemorrhage was found in 12 cases. Three of these have been referred to as being associated with endocarditis, and one occurred in a syphilitic patient, although there was nothing found at autopsy to suggest this infection. In one other case, in which the history gave no indication of any infection, the autopsy showed what was believed to be a definite syphilitic lesion of the abdominal viscera. In the other cases there was the ordinary vascular degeneration.

**Decade VII.**—In the seventh decade we have 105 cases for analysis, a considerable falling off from the preceding period. The liability, judged from the mortality statistics, is greater, being increased more than two and a half times, and the actual number of registered deaths in Baltimore was 95 for the seventh decade and 69 for the sixth.

At this period it is impossible to make any satisfactory division of the causes which led up to the vascular accident. Fourteen cases did, indeed, give a history of syphilis, but the average time of infection was about thirty years previous, and its influence could have been only problematical. In 2 cases syphilis did seem to have been the predisposing cause. In these, aged sixty and sixty-five years, there was an undoubted infection about three years before the onset of the hemiplegia. In neither case was there any other probable cause determined.

In the great majority of the other cases, evidences of general arteriosclerosis were found, with associated cardiac and renal conditions, states in which thrombosis and hemorrhage are common. In 6 cases the heart gave such evidences of severe valvular involvement that emboli might have been expected; but in the only case which came to autopsy marked endocarditis, general arteriosclerosis, and a hemorrhage into the left internal capsule

were found. Hemorrhage was found at autopsy in 4 cases. In the 2 cases in which cerebral softening was found it was believed to be due to embolism, once from an aneurism of the aorta and once from some unknown source, the embolism being demonstrated on section.

**Decades VIII and IX.**—In the eighth and ninth decades, although the liability to apoplexy continues to increase, the frequency of its occurrence decreases markedly in the last part of this period, owing to the relatively small number of individuals alive between eighty and ninety. In the present analysis there are 25 cases in the eighth decade and 6 in the ninth. There are 3 autopsies recorded in the eighth and 2 in the ninth. In all 3 of the eighth decade and one of the ninth, cerebral softening was found. One was due to thrombosis of the right internal carotid, secondary to embolism of the brachial artery. The other 3 appear to have been primarily thrombotic. The one case of cerebral hemorrhage occurred in a colored man, aged eighty-one, and was associated with intense degeneration of the whole vascular system.

**Symptoms.**—**Preceding.**—Slight cerebral symptoms may occur many months before apoplexy. These depend upon the vascular changes which lead up to the rupture or the occlusion of a vessel, and are important as indicating that such changes have taken place. It is obvious that an embolus whose origin is from some distant part of the circulatory system, can give rise to no cerebral manifestations until it lodges in an artery of the brain. But if the occluded artery be small, or one in which collateral circulation can be established, the symptoms may be slight or transient and premonitory of those following a subsequent larger embolus.

The liability of an artery to rupture depends upon some weakness of the vessel wall, usually upon the presence of miliary aneurisms. These minute dilatations on the smaller vessels themselves give rise to no symptoms that can be distinguished, and so in many, probably most cases of cerebral hemorrhage, there are no premonitory symptoms. Miliary aneurisms are, however, usually associated with cerebral arteriosclerosis, and the symptoms due to this condition may be present for a long time before the rupture. The symptoms in such a case are exactly those which more commonly precede an apoplexy due to thrombosis.

The rupture of a larger aneurism of one of the arteries at the base of the brain is a rare cause of cerebral apoplexy. These dilatations usually give no symptoms until their rupture, but at times they cause the manifestations of cerebral tumors. When such symptoms are present, associated with subjective noises in the head, and the presence of a vascular murmur heard through the skull, the diagnosis of an aneurism is justified. This association appears to be so rare as to have very little practical importance as a warning signal. Much more frequently the aneurismal dilatations are associated with widespread arterial changes; and here again we are apt to have the symptoms of cerebral arteriosclerosis. Hemorrhage may occur into the substance of a brain tumor, and so the symptoms of such a growth may at times be premonitory of cerebral hemorrhage.

The great majority of cases of cerebral thrombosis occur in the last half of life, and are due to more or less chronic changes of the cerebral vessels—cerebral arteriosclerosis. The symptoms which are due to this condition, and which often precede thrombosis, have already been considered. The most significant are a lowering of the mental strength, giddiness, slight

cerebral attacks, convulsive seizures, transient aphasias, slight weakness of the various limbs, paræsthesia and pain, etc., when associated with evidence of sclerosis in the peripheral arteries, especially in those of the retina, and the signs of general arteriosclerosis.

Thrombosis of a cerebral vessel, in the first half of life, is most commonly due to the effects of syphilis, or is in association with acute infectious diseases. That due to syphilis may be associated with the symptoms which are recognized as indicating specific involvement of the brain, intense periodical headaches, paralysis of various cranial nerves, etc., and these we may in the present relation regard as premonitory. There are usually no premonitory symptoms of thrombosis of a cerebral vessel occurring in the course of acute infectious diseases.

The nervous symptoms which precede an apoplexy, if cerebral syphilis be excluded, are practically all due to arteriosclerosis. They more commonly precede thrombosis than hemorrhage, but are not exceptional even with the latter.

**Exciting Causes.**—An apoplectic attack may occur under all conditions of life—during sleep, while awake in bed, upon getting up, at table, at work, at play, or while occupied in any other manner. Overexertion does not often play its assigned important role in exciting the attack, and, indeed, it is rarely possible to discover from the history any evident relation of the occupation to the stroke.

In the analysis of the cases in which the onset was after the first decade, and excluding the traumatic cases, there were 379 in which the circumstances of the attack were definitely noted. Among these 125 occurred while the patients were in bed and in most instances asleep. Fifty-four of these occurred during the course of some other illness, and 37 while the patients were in the hospital. In 36 instances the attack was noticed when the patient got out of bed, and some of these may have occurred during sleep. In contrast to this it is interesting to note that but 4 cases occurred while patients were preparing for bed. Fifty-nine attacks developed while the patients were sitting. A number of these were while the patients were eating a meal, but in most instances they were quietly reading, sewing, etc. In 3 instances they were playing cards, and it is possible that emotional effects may have been operative here and in some other cases. This seemed certainly so in one instance, that of a colored midwife, who became very angry on account of the refusal of the authorities to admit her patient to the hospital. She fell unconscious, and was herself taken into the ward, and died within twenty-four hours. This was the only case in which anger seems to have played a part. Ten attacks occurred while the patients were on more or less protracted sprees, and 54 while the patients were simply walking about, either at home or on the street.

This analysis includes apoplexies due to all causes, and it might well be that those cases which took place during rest, included, for the most part, those due to thrombosis, and that the cases of hemorrhage were among those occurring with more or less bodily activity. Theoretically, anything that increases the blood pressure within weakened vessel walls would tend to cause their rupture, and we should expect to find many cases of apoplexy due to hemorrhage occurring during muscular exertion or those emotional states associated with increased action of the heart. Increased cardiac action might also be assumed to favor the detachment of an embolus, and

so similar causes might be expected to be active in this class. Thrombosis, on the other hand, is favored theoretically by quite opposite circulatory conditions, and the predisposing causes, changes in the vessel walls or in the blood, being present, slowing of the blood current should act as an accessory factor, and might determine the time of the occlusion.

Clinically, however, the state of affairs is not so simple, and is modified by many circumstances which cannot be determined, and it is very unsafe to assume the character of the lesion from the state of activity of the patient at the time of the stroke. The study of a large number of cases in which the lesions were determined after death would show the real relation of these factors. Unfortunately, the figures available from this series are but few, but they may help to swell the total from other sources.

There were 25 cases of *cerebral hemorrhage* in which the onset was given. Twelve of the attacks took place while the patients were at rest, 10 while in bed, and 2 while sitting quietly; 6 of the 12 attacks occurred in the hospital wards, as the terminal event of various conditions; 3 other apoplexies terminated alcoholic debauches; 1 of these patients was sitting at a table, and the others were probably not very active, although as to this we have no definite data; 4 patients were struck down while walking about, and 5 while at their every-day work. In only 1 case did there seem any great amount of physical exertion, and that was in a man, aged thirty-nine years, who, after a long bicycle ride, was found at 3 A.M. on the steps of a house, paralyzed and aphasic.

The onset was noted in 23 cases of *cerebral softening*, and in 16 of these the patients were in bed, all but one being confined by some severe illness; 14 of them were in the hospital at the time of the onset. Of the other 7 attacks, 4 took place while the patients were at work, and one while occupied in each of the following ways—sitting quietly, walking about, and during an alcoholic spree.

It will be seen that of the 26 cases in which cerebral lesions developed while the patients were in bed, only 10 proved at autopsy to be hemorrhage, but that of the 19 apoplexies developing during the more or less bodily activity (including the 4 cases which occurred during alcoholic debauches), 13 proved to be due to cerebral hemorrhage.

A number of authors have interested themselves as to the importance of bodily activity in the production of the various cerebral vascular lesions. Ernest Jones[1] analyzed the records at University College Hospital, London, in relation, among other things, to the time of onset of the various vascular lesions. These records covered sixty-five years, and the available material consisted of 160 lesions, 123 cases of hemorrhage, 24 of thrombosis, and 13 of embolism. None of his cases occurred during excessive bodily activity. He compared his results with those of various other authors, and concluded: "Rest in bed, and especially sleep, protect to some extent against cerebral hemorrhage. This would seem a more accurate statement than that rest and sleep predispose to thrombosis, as I can find no evidence in favor of this, although in actual practice the two statements have a similar bearing on diagnosis. Severe exertion and time of day appear to have had too much stress laid on them in the past. A miliary aneurism that is going to rupture rarely needs the aid of severe exertion for this con-

[1] *Brain*, 1905, p. 527.

summation. Time of day is only of interest when the habits of the patient's blood pressure at different hours are known."

**The Apoplectic Attack.**—Throughout the present article the word apoplexy has been used more in its original sense as meaning an acute cerebral attack, but it has been stretched to include not only those seizures of sudden loss of consciousness with paralysis of sense and motion, but also those in which the consciousness is affected later, or, indeed, not at all. Trousseau used the word strictly as signifying an absolutely sudden loss of consciousness, and so pointed out that "apoplexy" was rarely, if ever, the initial symptom in cerebral hemorrhage, and that if the patients were under observation at the time of the onset, symptoms due to cerebral lesions could always be noted before consciousness was lost. Indeed, he states that he had never seen a man struck down as by a blow and dropping instantly in a state of unconsciousness.

Loss of consciousness as the initial symptom is indeed rare, and in the vast majority of cases patients usually complain of headache, vertigo, tingling or weakness in some part of the body, as the indications of the attack. The loss of consciousness, if it occurs, may be delayed only a few minutes, or for several hours, or, indeed, days. The manner of occurrence of the different lesions—thrombosis, hemorrhage, and embolism—differs, and one would expect the development of the symptoms to be somewhat characteristic; thus, the closure of an artery by a thrombus is a more or less gradual process, and so one would expect the symptoms to develop more or less gradually. The rupture of a bloodvessel is a sudden occurrence, and the resulting symptoms should make their appearance quickly. The occlusion of a bloodvessel by an embolus may be absolutely sudden, and in connection with it we might expect apoplexy to develop in its classical manner.

These considerations are, in general, true, but are often so modified that conclusions based upon them cannot be accepted absolutely. Although it is true that thrombosis is not in its entirety a sudden process, still the final closure of a vessel may occur suddenly, and it is not at all uncommon to have the symptoms develop with great rapidity. Jones, in this connection, says, "There is a large group of thrombosis cases, the onset of which is apoplectiform." The percentage has been put as high as 58 by some authorities. There are a number of such cases in the records of this series. For instance, a woman, aged fifty-two years, who had one previous attack from which she had recovered, while in apparent health and transacting business with a lawyer, fell unconscious. After her death, three weeks later, cerebral thrombosis was found.

Although the rupture of a vessel is an instantaneous process, the tear at first may be very slight, and only a small quantity of blood may escape. The focus may increase in size more or less gradually, destroying more and more of the nervous tissue, and many cases of cerebral hemorrhage have the onset typical of thrombosis. A man, aged fifty-nine years, while sitting with his family after supper, complained of intense headache. Ten minutes later it was noticed that his face jerked, and that when spoken to he did not answer questions. In half an hour he got up, closed the house, went up stairs, and moved about his room. He gradually became unconscious during the night, and was completely paralyzed on the right side. After his death, one week later, a large hemorrhagic clot was found in the left cerebral hemisphere. Another man, aged sixty-five years, with definite

arteriosclerosis, and who had cerebral symptoms signifying this condition, gradually lost power on the left side in the course of twenty-four hours, with increasing dulness but no actual coma. He improved somewhat, but died in eleven days from bronchopneumonia. At autopsy the brain showed marked arteriosclerosis, multiple areas of softening, and, in the right hemisphere, a large clot, 7 cm. in diameter.

The stoppage of an embolus is always sudden, but if an embolus lodges at the bifurcation of a vessel, the blood current is not completely interrupted until the formation of the thrombus around the embolus occludes one or the other of the arterial branches. This secondary thrombus formation may extend and cut off branches that had previously received blood by collateral circulation. An embolus may be caught in an artery that has free collateral circulation, or there may be more than one embolus, the first plugging a relatively small vessel and a subsequent one a more important artery. It is obvious that if any of these circumstances occur the symptoms following an embolus of the cerebral vessels may develop more or less gradually.

**Consciousness.**—Even though it is true, as Trousseau pointed out, that an absolutely sudden loss of consciousness is uncommon in association with any of the cerebral vascular lesions, still consciousness is often disturbed in association with all of them. In the records of 401 apoplectic attacks in which the state of consciousness was noted, there was no loss of consciousness in 202 cases, but in 199 it was either lost or markedly disturbed. In 107 of these it was reasonably certain that the disturbance of consciousness occurred early in the attack. As but few came to autopsy they are of no value in determining the relative frequency of loss of consciousness in relation to the different lesions. Jones analyzed his own cases, together with those which he had been able to collect from the literature, and found that among 201 cases of embolus, consciousness was lost in 47.7 per cent., and was affected in nearly 60 per cent., while in 235 cases of cerebral hemorrhage consciousness was lost in nearly 75 per cent. He gave no collected figures for thrombosis in this relation, but in his own 22 cases there was loss of consciousness in 45.5 per cent.

The mechanism of the loss of consciousness is not thoroughly understood, but since consciousness depends upon an adequate supply of blood to the cerebral cortex, a disturbance of this is the most obvious and best substantiated cause for the loss. It is easy to understand how the sudden closure of one of the large vessels would produce such an anæmia as to disturb consciousness, and, indeed, an embolus lodging in and occluding the middle cerebral or the basilar artery, both favorite seats for such an occurrence, would offer the ideal conditions for a sudden loss of consciousness. Saveliew,[1] who analyzed all the cases of cerebral emboli that occurred in Virchow's laboratory from 1856 to 1893, states that a sudden loss of consciousness is the common onset in cerebral embolism. When the artery is slowly occluded there is more opportunity for collateral circulation to be established; primary cortical anæmia is less apt to occur, and consciousness when lost is apt to be lost late. This late loss of consciousness must be due to secondary causes, and in association with occlusion of a vessel is probably dependent upon the œdema which accompanies and surrounds

[1] *Virchow's Arch.*, 1894, cxxxv, 112.

the anæmic area. This secondary œdema causes, among other things, an increase in intracranial pressure, which may be so great as of itself to produce a cortical anæmia. Destruction of the brain tissue itself must have an effect on consciousness, for it depends upon the association of various cerebral activities, and must be affected by anything that injures the structure upon which these depend. We do not know just how great the disturbance must be to abolish consciousness, or the relative importance in this relation of the different parts of the brain.

The loss of consciousness accompanying cerebral hemorrhage, although perhaps rarely the initial symptom, very commonly occurs early, and appears to be more frequent and usually of longer duration with this lesion than with either of the other two. The explanation of these facts is not clear, and many theories have been advanced. Gowers,[1] in speaking of the loss of consciousness, says: "It is probably first due to irritative inhibition of the cortex, which is maintained by increased intracranial pressure. The laceration causes powerful irritation, and when the hemorrhage is into structures that have a wide relation to the cortex, as in the pons, a small extravasation, which can have little effect on intracranial pressure, causes early loss of consciousness, which endures." von Monakow analyzes the various effects caused by a hemorrhage into the substance of the brain, and divides them into the following groups: (1) The primary local destruction of the nervous tissue; (2) the œdema in its neighborhood; (3) the decrease in the amount of blood in the vessels in the neighborhood of the clot; (4) accompanying circulatory changes in the cortex; (5) the transmission of the mechanical shock caused by the sudden escape of blood into the brain in all directions throughout the intracranial cavity, this shock, besides its physical action, acting as a physiological stimulus to various parts of the brain; and especially, he thinks, it may stimulate the vasomotor centres and cause a reflex contraction of the cortical vessels; (6) increase in intracranial pressure; (7) interference with circulation of the cerebrospinal fluid. In the last analysis he believes that the disturbance of consciousness depends upon a sudden cerebral anæmia, and that this is brought about by some or all of the various factors enumerated above, but that probably the mechanical stimulation of the vasomotor centres "plays the lion's part."

The occurrence and degree of coma does not depend upon the size of the hemorrhage, but seems to have some definite relation to its position. The exact relationship has not been definitely determined, but von Monakow states that after analyzing a great number of cases it appears to him that coma is more apt to occur the nearer the lesion is to the central gray matter of the third ventricle, or the nuclei of the optic thalamus, and that it is much less apt to occur when the white matter of the cerebral hemisphere is involved. When the hemorrhage breaks through into the ventricle consciousness is almost always lost, and Jones found that it was lost in 85.5 per cent. of this class of cases, while in the non-ventricular hemorrhages the percentage of loss was only 69.4 per cent.

The duration of the coma varies greatly, and may be very short or persist until death. It is apt to be shorter when associated with embolism and longer with thrombosis and hemorrhage; but even in hemorrhage von Monakow gives the average duration as only from a half hour to four hours.

[1] *Brit. Med. Jour.*, Lond., 1907, ii, 1.

Oppenheim puts the duration somewhat longer, from one to four hours for the lighter cases and one to two days for the more severe ones. From the actual coma, patients not infrequently pass into a state of dull consciousness, during which they have very little recollection of what passes around them, and it is not at all uncommon for such patients to insist that they were unconscious for a week or even longer.

**Pulse and Blood Pressure.**—During an attack of apoplexy the condition of the pulse varies markedly. A slow pulse, particularly with a high blood pressure, indicates an increased intracranial pressure, and is usually associated with a cerebral hemorrhage. Thus, in an old colored woman, brought to the hospital directly after the attack, and who died within four hours from a large cerebral hemorrhage, the pulse on admission was only 48, and the blood pressure was above 350 mm. Hg. In a man admitted on the third day after his stroke, the pulse rate was also 48, and the blood pressure between 330 and 340 mm. Hg. A large capsular hemorrhage was found after death. These cases are, however, rather exceptional in our experience, and the rate of the pulse is often not much lowered, and is, indeed, at times accelerated.

The character of the pulse is usually of more importance, for in association with cerebral hemorrhage it is, as a rule, hard and full, and the blood pressure may be very high, as in the cases just mentioned. Such an excessive elevation, although characteristic of acute increase in intracranial pressure, and perhaps never found clinically except with cerebral hemorrhage, is exceptional even with this condition. The blood pressure more usually ranges between 150 and 200, and even in rapidly fatal cases may be quite normal, as in a woman, aged forty-three years, brought to the hospital shortly after an attack, in whom the pulse was 100 and the blood pressure 125 mm. Hg. She died within twenty-four hours, and a large hemorrhage was found in the right hemisphere. Another patient, who died two and a half days after an extensive hemorrhage into the internal capsule, had a blood pressure of only 135.

A blood pressure in the neighborhood of 200 is frequently found in association with arteriosclerosis, and as this condition often precedes for a long time the occurrence of an apoplexy due to either rupture or thrombosis of a cerebral vessel, the significance of a high blood pressure during an apoplectic attack depends largely upon our knowledge of the previous conditions. Although it is generally stated that when an apoplexy has been due to thrombosis the pulse is quick, intermittent, and of low tension, it is not at all uncommon to find a high blood pressure associated with this condition. Janeway,[1] in speaking of his experience at the City Hospital, New York, says: "Thrombotic softening is the common cause of hemiplegia in our patients, even when they have granular kidneys and high blood pressure."

Changes in blood pressure are more significant than the actual height, and a rapidly rising pressure indicates an increasing cerebral compression, while a falling pressure is one of the most definite signs of failure of the vasomotor centres and of approaching death. A good illustration of the first condition is the case of a man brought into the ward shortly after having had a capsular hemorrhage. His blood pressure was then 220 mm. Hg. and the pulse about 90. The pressure rose steadily, and the next day reached

[1] *The Clinical Study of Blood Pressure*, 1907, p. 189.

250, when an operation was undertaken. As the dura was opened the blood pressure fell to 180 and subsequently even lower. It rose again, but never reached 200 during the three days which he lived. A most impressive example of the second sequence of events was in a man whom we had observed for a number of months. He showed the typical picture of cerebral arteriosclerosis, and had suffered from several vascular attacks. He was in the ward for nearly a month before the final accident. During the first few days of his stay in the hospital his blood pressure ranged between 230 and 240, but subsequently about 215, with a pulse rate of 95. One afternoon he complained of intense headache, for the relief of which morphine had to be given. At 6 P.M. the blood pressure was 200. In the middle of the night he was found in coma. His blood pressure at 3.30 A.M. was 240; pulse, 145. At 5.30 the pressure was 200; at 11 it was 180, and then fell quickly, reaching 80 at 1 P.M. and 60 at 1.15 P.M., the pulse remaining between 120 and 130. Death took place at 1.40 P.M. At autopsy a large cerebellar hemorrhage was found.

**Respiration.**—The respiration is usually slow and deep; it is often stertorous and irregular, and at times shows the Cheyne-Stokes type. The noisy stertorous breathing is usually due to the fact that during coma the mouth has a tendency to fall open and the tongue to fall back. This is particularly apt to occur when the patient is lying on the back. Bowles[1] has considered this very fully, and points out the importance of keeping an apoplectic patient on his side.

Cheyne-Stokes breathing during apoplexy is usually a symptom of the embarrassment of the respiratory centres in the medulla as the result of anæmia, secondary to increased intracranial pressure, and is an important danger signal. The vasomotor centres tend to keep the blood pressure above intracranial pressure, so that the medullary centres may still be supplied with blood. When the intracranial pressure remains constantly high, the blood pressure may show periodic waves (Traube-Hering waves), varying from a little below to a little above the level of the intracranial pressure. Experimentally, when such variations occur, respiration becomes intermittent. In general, it ceases when the blood pressure is relatively low and begins and increases when the blood pressure rises above intracranial pressure. If careful tracings of the blood pressure and respiration be made in a case of apoplexy showing the Cheyne-Stokes respiration, quite analogous conditions will be seen to be present. Eyster,[2] from a study of such cases, concludes that this type of Cheyne-Stokes breathing is due to the centre of respiration being intermittently supplied with blood; for when the centre has been deprived of arterial blood, it ceases to be capable of responding to its normal stimulus, *i. e.*, an increase in its supply of carbon dioxide and a reduction in oxygen, and so when the arterial pressure falls below intracranial pressure the medulla becomes anæmic and respiration ceases. Anæmia of the medulla excites the vasomotor centres to renewed activity, blood pressure is again raised, circulation is reëstablished through the medulla, the respiratory centre regains its irritability, responds to stimuli, and respiration commences. The respiratory efforts vary in force in accordance with the varying relations between the irritability of

[1] *On Stertor, Apoplexy, and the Management of the Apoplectic State*, 1891.
[2] *Jour. of Exper. Med.*, 1906, viii, No. 5.

the centre and the force of the stimulus that excites it. The groups of respiratory efforts may show the typical ascending and descending character separated by a period of complete apnœa, but they much more frequently depart from this type and consist of groups of respiration of approximately equal depth, of descending groups in which the first is the largest, or finally of groups of respiration of irregular depth (Eyster).

There are often other phenomena due to the varying blood supply of the brain. The pulse varies in rate, being slow during the period of apnœa, as a result of the stimulating effect which anæmia has on the cardio-inhibitory (vagus) centre. This inhibition is relaxed when respiration begins, and the pulse rate is increased during the period of inhibitory activity. The mental activity also varies, and the patient, who is completely unconscious when the respiration ceases, can be aroused and can speak voluntarily, during the period of respiratory activity. At times the pupils show a periodical change in their size, dilating at the beginning of apnœa and contracting when respiration returns (Cushing). The importance of this type of periodic respiration, especially when accompanied by these other phenomena, is as an indication of an increase of intracranial pressure to such a degree that the vasomotor centre has difficulty in maintaining the blood pressure above the intracranial pressure, and is a symptom of the utmost gravity.

**Ophthalmoscopic Examination.**—This may give important information, both as to the state of the bloodvessels and as indicating the condition of intracranial tension. Bordley who has examined many of the cases in the hospital, states that in most cases no other fundus changes were seen except those which preceded the stroke, *i. e.*, the changes indicating degeneration of the bloodvessels. In certain patients, however, particularly in those who were profoundly unconscious and showed Cheyne-Stokes respiration, undoubted choked disk was present. This was often of low grade, and with more dilatation and tortuosity of the veins than is usually seen with such a degree of choked disk, but it was seldom associated with retinal hemorrhages and exudates. In the cases of traumatic intracerebral hemorrhage, in which there was rapid increase of intracranial pressure, choked disk was constant.

**Temperature.**—When an apoplexy is due to cerebral hemorrhage there is usually an initial fall of from 1° to 2°. This may last only an hour or two, to be followed by a gradual rise, and may be entirely missed unless the patient is under observation at the time of the stroke. It is said that with thrombosis this initial fall does not take place. The writer has not sufficient exact data of cases in which the lesion was proved at autopsy to express any definite opinion. The cases of thrombosis which occurred in the hospital were in sick patients who usually had more or less fever and the changes in the temperature at the time of the stroke were neither constant nor remarkable.

The temperature almost always rises within the first twenty-four hours, and in rapidly fatal cases may reach great heights. Gowers associated an immediate elevation of temperature with hemorrhages into the pons or medulla. This hyperpyrexia is of great significance, and whenever it occurs is usually a sign of approaching death. In a patient already referred to, who died in about twelve hours from a cerebellar hemorrhage, the temperature rose rapidly to 106.5°. The attack took place in the night, and the initial fall of temperature, if it occurred, was not determined. On the other

hand, in certain quickly fatal cases the temperature continues to fall from the time of the attack, and may reach remarkably low levels. A rectal temperature of 95° has been recorded. The cause of these extreme temperatures seems to be a disarrangement of the heat regulating mechanism, and is one of the signs of failure of the cerebral functions. Some such disturbance seems the most reasonable explanation of the hyperpyrexia in a case of cerebral hemorrhage, in which the temperature on the second day varied between 99.5° and 100°, and rose suddenly to 106° immediately after an operation during which an unsuccessful attempt was made to evacuate the clot.

The ordinary rise of from 1° to 2° in the first few days after an apoplexy has no especially grave significance. It is the so-called fever of reaction, and is believed to depend upon some inflammatory process about the hemorrhagic focus. This fever usually lasts for only two or three days, after which the temperature remains normal. Should, however, the fever be higher than usual or last longer, it becomes of grave significance, and at any time if the temperature rises rapidly to unusual heights the prognosis becomes very grave.

**Signs of Local Injury to the Brain.**—The symptoms so far considered are the general symptoms, but even during the height of an apoplectic stroke there are in most cases signs of local injury. These depend somewhat upon the location of the lesion, but for the most part indicate that the functions of one cerebral hemisphere have been disturbed. These symptoms are usually paralytic but may be irritative, when they are revealed by an excess of muscular activity. The limbs on the side of the body opposite to the lesion may be in a state of hypertension (early rigidity) with the eyes and head deflected toward the side of the spastic limbs and away from the lesion —spastic conjugate deviation; or the affected limbs may be the seat of clonic convulsions, and if the lesion is in the motor cortex, or near it, typical Jacksonian epileptic attacks may follow.

Symptoms of paralysis are much more common, and all the movements represented on one side of the brain are usually more or less affected. During coma, when the patient is making no spontaneous movements, this is often shown by a decrease in the muscle tone on the side opposite to the lesion. The limbs are flaccid, they fall like a dead weight when raised and released, while those on the opposite side may show a certain amount of resistance. Heilbronner[1] points out that this lack of muscular tone may be determined by inspection, for if the patient be on a firm support the muscle mass on the affected side, particularly that of the thigh, acts like a sac filled with a semisolid, and takes the shape determined by gravity. It is broadened and flattened, while that on the normal side has a more rounded contour. The lack of tone of the muscles of the face on the affected side is shown by the cheek being puffed out more than on the other side during forcible respiration, the air escaping from the corner of the paralyzed side of the mouth, as if the patient were smoking a pipe. This is, as the French say, "fumer la pipe." At times the nostril on that side, instead of being expanded, is contracted during inspiration.

Not infrequently the patient exhibits a tendency to lie with the head turned toward the non-paralyzed side and with the eyes deflected strongly toward

[1] *Deutsche Ztschr. f. Nervenh.*, 1905, p. 1.

that side, *i. e.*, the patient looks toward his lesion. This is paralytic, conjugate deviation, and is due to the fact that in each cerebral hemisphere movements are represented which turn the eyes and head to the opposite side and a destructive lesion paralyzes them. The normal position of the eyes depends upon the delicately adjusted activity of their muscles, and so when the tone of the muscles associated in a definite movement is decreased, the tone of the muscles causing the movement in the opposite direction deflects the eyes. Thus a lesion in the right hemisphere causes a decrease of the activity of the muscles which turn the eyes to the left, *i. e.*, the left external rectus and the right internal rectus, together with the muscles active in the closely associated movement of the head to the same side, and it is the unbalanced tone of the muscles which opposes these movements which causes the deflection of the head and eyes to the opposite side, *i. e.*, to the right and toward the lesion.

Muscle tone depends, for the most part, upon reflex stimulation, and it seems probable that conjugate deviation in apoplexy is, to a large extent, due to a disturbance of the reflex mechanism. At any rate, the symptom is usually a transient one, occurring during coma, and lasting from a few hours to one or more days, and is coincident with disturbances of other reflexes. When it persists for a longer time it is often associated with hemianopia, a condition in which all the visual stimuli come from but one-half of the patient's surroundings and tend to keep the eyes turned toward that side. The mechanism involved is a very complicated one, and many stimuli must be active in maintaining the tone of the centres. That the visual impressions, although normally perhaps the most important, are not the only ones concerned, is shown by the interesting case reported by Dejerine and Roussy,[1] in which conjugate deviation of the head and eyes occurred in an old woman, aged seventy-one years, who had been blind since infancy.

**Pupils.**—The pupils during the apoplectic attack are usually equal and about normal size. They may be dilated, contracted, or unequal, with the larger pupil on the side of the lesion when this is in a cerebral hemisphere, but on the opposite side when the focus is in the brain stem. During coma the light reflex is absent.

**Reflexes.**—During absolute coma all reflexes are abolished, but very generally the coma is not of that grade, and one often is able to demonstrate a difference in the reflex activity of the two sides. In general, it may be stated that a sudden severe vascular lesion in one cerebral hemisphere has a tendency to abolish the reflexes on the opposite side of the body. The early conjugate deviation of the eyes and the lack of muscle tone on the paralyzed side must, in a large measure, be due to this effect. The reflexes from the skin and mucous membranes, as well as the deep reflexes, may also be lost or altered, and when such a change is demonstrated on one side it is an important indication of a unilateral cerebral lesion. Since Babinski pointed out the varying response of the great toe to plantar stimulation under different conditions, this reflex has assumed the utmost importance. If an organic lesion has occurred which implicates the pyramidal tract, the great toe, instead of being turned down, moves in the opposite direction,

[1] *Rev. Neurol.*, 1905, xiii, 161; Weisenburg, *Jour. Am. M. Ass.*, 1907, xlviii, and Debray, *Jour. de Neurol.*, October 5 and 7, 1907, have given interesting reviews concerning this subject.

*i. e.*, in extension, or more properly dorsal flexion. This abnormal response of the great toe (Babinski reflex) may make its appearance directly after a cerebral lesion, and it may be the first positive indication that such a lesion has occurred. The writer has noted this on several occasions, and in a patient whose attack occurred in the ward it was marked within twenty minutes.

As the coma lessens the reflexes return, first on the non-paralyzed side, and according to von Monakow in the following order: first the conjunctival reflex, then the pupillary light reflex, the abdominal and the other skin reflexes, and finally the deep reflexes. The return of the reflexes on the paralyzed side may be delayed for a considerable time, even, it is stated, for two or three weeks. In our experience, however, the deep reflexes return quickly, and may even be exaggerated within a short time, although the corneal and the abdominal reflexes may remain absent. The importance of the absence of these two skin reflexes in association with hemiplegia has been lately insisted upon.[1]

During coma the bladder and rectum are apt to empty involuntarily, but there may be retention, and as the patient recovers there is not infrequently some difficulty in completely emptying the bladder.

**Paralysis of Voluntary Motion.**—Unless the coma is absolute, observation, in the great majority of cases, reveals that the patient has lost certain movements. It will be noted that one arm and one leg will lie perfectly quiet, while the limbs on the other side may be moved restlessly about; that when the patient is moved in bed or irritated in any other manner, the movements which result are only on one side of the body. This lack of motion may also affect the face on the same side; *i. e.*, the patient has hemiplegia. At first, and in severe cases, all the unilateral movements are affected, but certain movements show a tendency to recover quickly or to be spared when the process is less intense. This selective paralysis in hemiplegia is of great interest and will be discussed in a subsequent section. The limbs on the non-paralyzed side are not infrequently moved restlessly about; the patient picks at the bedclothing, moves the hand to the head, draws up the leg, and may persist in these movements so constantly that at times they may suggest convulsive phenomena.

The movements of the chest during respiration are generally recorded in our histories as being equal, and we have endeavored to confirm the common statement that the movements on the paralyzed side are less than on the opposite side.

**Diagnosis.**—The diagnosis of coma due to a cerebral vascular lesion is usually made without difficulty, as when a patient who has shown evidences of the circulatory conditions which predispose to rupture or occlusion of an artery has symptoms of local involvement of the brain (numbness and weakness on one side of the body, unilateral muscular twitchings, etc.) and then passes into unconsciousness, during which definite evidences of hemiplegia are present. At times, however, the diagnosis may be extremely difficult, and, indeed, impossible, as when the coma occurs suddenly without premonitory cerebral symptoms, or when the physician sees the patient for the first time during the attack, the onset of which has been unobserved.

Coma may occur under a variety of circumstances, and, indeed, is the

[1] Milian, *Le Progrès méd.*, May, 1909.

frequent terminal state of all fatal illnesses. It is often an evidence of the toxæmias associated with nephritis (uræmia) and diabetes. It occurs as a result of poisoning by alcohol, opium, and its derivatives, illuminating gas (carbon monoxide), chloroform, ether, etc. It may be associated with sudden disturbances of the general circulation, if they are sufficient to produce cerebral anæmia, as in the ordinary fainting fit, the attacks of unconsciousness with Stokes-Adams disease, the rupture of a large aneurism, etc. It follows an epileptic attack and may be purely hysterical. It happens occasionally in malaria and heat stroke, and it is a frequent result of trauma.

In most cases the history will exclude many of the conditions in which coma may occur, and the mistakes are apt to happen when little or nothing is known of the patient or of the onset of the attack, as in hospital and police practice. Every such case should be regarded with suspicion, and the diagnosis of one of the less serious states should be deferred until the indications are clear and unmistakable.

When the coma is so deep that all special activities of the brain are in abeyance, when no movements occur and no reflexes can be elicited on either side of the body, evidences of a focal lesion of the brain are, of course, absent, and as it is upon these that the diagnosis of a cerebral lesion must largely depend, it may for a time be quite impossible to determine the cause of the coma.

*Uræmia.*—In the absence of unilateral cerebral symptoms the diagnosis must depend upon the previous history, the occurrence of chronic nephritis, convulsions ushering in the coma, which usually develops rather slowly, etc. Objectively, the condition may be remarkably similar to apoplectic coma. The high arterial tension, Cheyne-Stokes respiration, and the ophthalmoscopic picture, may be quite similar in both conditions, and when it is remembered that transient focal cerebral symptoms are not uncommon in uræmia, and that apoplexy frequently occurs in cases suffering from chronic nephritis, it will be seen how complicated the diagnosis may become. Eyster has pointed out that in the periodic respiration of uræmia the relation between the respiratory phases and the changes in the blood pressure differs from that which is found in Cheyne-Stokes respiration secondary to increased intracranial pressure, as in increasing cerebral hemorrhage, and it is probable that careful tracings of the respiratory and blood pressure changes may give important indications. Simple choked disk may be present in both conditions, but in nephritis the characteristic picture of albuminuric retinitis is much more frequent.

How confusing these conditions may be is well illustrated by the case of a young woman, aged twenty-two years, who was under observation for six months. She had chronic nephritis and a number of attacks of uræmic coma, with intense choked disk. A decompressive operation was done and seemed to give benefit. She was admitted two months later with the history of having been dull for a week. She became gradually more comatose, Cheyne-Stokes respiration developed, and she died thirteen days after admission. The only unilateral symptom that occurred was inequality of the pupils, the right being larger than the left, but as the patient was blind from retinal changes this was not regarded. At autopsy a large capsular hemorrhage was found on the right side.

Coma occurring in pregnant women at about the time of labor is usually due to toxæmia (uræmia or eclampsia), but exceptionally it depends upon

a cerebral hemorrhage, which, unless accompanied by focal symptoms, is very likely to be overlooked.

*Diabetic coma* generally causes but little confusion. The history of the preceding illness is usually obtained. The coma develops gradually, being preceded by lassitude, headache, great restlessness, and other symptoms. The pulse is apt to be rapid and of low tension, and the respirations show "air hunger," being full and noisy, but quite regular and usually not increased in frequency. The breath has a fruity odor, and so may the urine, which shows the presence of sugar, diacetic acid and acetone, and often traces of albumin with casts. Very rarely optic neuritis is present.

It must be remembered that the deep reflexes are often lost in diabetes, and their absence should not be given undue significance. Confusion may also arise from the fact that hemiplegia, in which no anatomical lesions were found, has been noted in diabetes. On the other hand, sugar has been found in the urine as the result of a cerebral vascular lesion. It is always safer to assume a definite focus when the symptoms indicate its presence.

*Alcoholic Coma.*—The mistake of regarding a case of apoplexy as simply one of acute alcoholic intoxication has been made innumerable times, and whenever this question arises the patient must be given the benefit of every doubt. The history of former alcoholism, and the marked alcoholic odor of the breath and vomitus have, of course, significance, but it must be remembered that alcohol is universally administered as a medicine under all conditions, and may have been swallowed at the onset of the attack, and also that apoplexy not infrequently occurs during an alcoholic debauch. The coma due to alcohol is almost never absolute, and the patient can usually be aroused to show some evidence of activity. This fact permits the discovery of paralytic symptoms, and it is the absence of these in such a patient, with the history, that justifies the probable diagnosis of drunkenness.

*Opium Poisoning.*—In acute opium poisoning the coma simulates intense drowsiness; the patient can be aroused by energetic stimulation, but lapses again into unconsciousness as soon as he is undisturbed. The pupils are contracted to an extreme degree; the respirations are stertorous, very slow, and may be regular, but at times occur in groups, as in the Cheyne-Stokes type. The skin is cold and of a livid hue; the pulse is rapid and of small volume. This picture is so characteristic that even without the history of administration of the poison mistakes are not apt to occur. It must not be forgotten, however, that very small pupils sometimes occur with a hemorrhage into the pons or mid-brain, and that they may be associated with some preëxisting condition, such as tabes and general paralysis, in which apoplexy is not uncommon.

Poisoning by other agents rarely gives rise to much confusion, but where nothing is known of the history of the patient one should be on the alert not to overlook them. The burning of the mouth and the intense gastro-intestinal disturbances, associated with symptoms of collapse, are usually sufficient to indicate the presence of carbolic acid poisoning. In unconsciousness from carbon-monoxide poisoning the history of exposure to escaping illuminating gas, or to the effects of incomplete combustion about furnaces, coke ovens, etc., is almost always obtained. In doubtful cases the blood must be examined. Its bright cherry-red color and the characteristic reactions of carbon monoxide are conclusive.

Coma following changes in the *general circulation* is clearly distinguished

by the associated conditions, and rarely gives rise to confusion. The ordinary fainting attack (syncope) is so well known as to require no comment. The attacks of unconsciousness in heart block are transient, and are so definitely associated with a pause in the ventricular pulse that mistakes should not occur. Rapidly occurring coma from the rupture of an aneurism, which persists until death, may, at times, be confused with that due to a large cerebral hemorrhage. The history and physical signs are usually sufficient to indicate the presence of an aneurism, and death is, in such cases, more rapid than in even the most severe apoplexies.

Absolutely sudden death, which is so often recorded in death certificates as due to apoplexy, is usually associated with a stoppage of the heart due to an obliteration of one of its coronary arteries. Apoplexy, even in the most quickly fatal cases, lasts a considerable time. Death within one or two hours is a rare occurrence, and the case recorded by Abercrombie nearly one hundred years ago, in which death occurred in five minutes, has not so far as the writer knows, been equalled. The quickest death in our records occurred in a little less than an hour, and in this case, besides the cerebral hemorrhage, there was a ruptured mesenteric aneurism.

*Epilepsy and Hysteria.*—The stupor following an ordinary isolated epileptic attack is usually easily recognized. The history of previous attacks, the characteristic convulsion which immediately preceded the coma, and its relatively short duration are significant. A case seen for the first time in the status epilepticus might easily be mistaken for one of convulsions and coma, following a cerebral vascular lesion, and the diagnosis would have to depend upon the subsequent course or the results of an autopsy. Unilateral epileptic convulsions are often followed by evidences of transient paralysis—a slight hemiplegia—even when there is no gross cerebral lesion; and, on the other hand, in patients past middle life, Jacksonian epilepsy is not infrequently the result of a previous cortical vascular lesion; and, indeed, such attacks may be the signs of a cerebral arteriosclerosis and the premonitory symptoms of a gross vascular lesion. In general paresis convulsive seizures and transient paralytic phenomena are not at all uncommon, but as gross cortical lesions often occur in this disease, one must be cautious in estimating the importance of an individual attack.

*Hysteria* at times introduces most puzzling and interesting questions of diagnosis. It should always be remembered, what is so often forgotten, that a hysterical patient has the same liability to organic disease that any other similarly situated individual has, and that in a predisposed individual hysterical symptoms may be so added to those which are dependent upon an organic lesion as to seriously confuse the picture. The definite evidence of organic disease has the same significance in a hysterical patient as in any other condition.

Hysterical coma has the appearance of deep, normal sleep, which is unduly prolonged or which has occurred during the daytime after some psychical shock. The patient can usually be aroused, at least for a time, by painful stimuli, as a faradic brush, pressure over the supra-orbital nerve or the eyeballs, water poured on the face, etc., or, indeed, by the determined command of the physician or the suggestion of some disagreeable procedure. The pulse and respiration are usually not markedly altered, although at times the respiration shows most bizarre changes with entire absence of regularity. It may have an extremely rapid rate, suggesting

the panting of an animal, or it may be so infrequent as to give rise to the fear that the patient is about to die from suffocation; and in very rare instances it shows the periodicity of the Cheyne-Stokes type. Persistent hiccoughing may be an alarming and confusing symptom, as this is also a symptom of an organic brain lesion. The temperature, unless in exceptional cases, is quite normal.

When such a condition has been ushered in by a typical hysterical convulsion one may feel reasonably sure of the diagnosis. The convulsions, however, may be absent or far from typical, and the condition so obscure that it is only after a detailed and fruitless search for some evidence of an organic lesion that a tentative diagnosis of hysterical coma is justified. The diagnosis of hysteria, especially where symptoms, apparently grave, are present, should never be more than a provisional one, and should stimulate to repeated examinations. The paralyses which are not infrequently associated with hysterical coma are to be distinguished by the absence of the distinctive characteristics of an organic type of paralysis.

*Malaria and Sunstroke.*—The sudden loss of consciousness that occurs exceptionally in malaria, and, as a rule, in heat stroke, may simulate the coma of apoplexy, and in malarial districts the diagnosis is often difficult. High fever is common in both malaria and heat stroke, and in the latter it may reach a very high point, 108° or even more. The discovery of the malarial parasite in the blood is, of course, conclusive of that disease, and the blood should always be examined in cases of coma where there is a possibility of malaria being the cause. The diagnosis of sunstroke depends upon the history of the onset under conditions that may induce it, and the absence of the evidences of paralyses characteristic of apoplexy.

*Trauma.*—In coma following injury to the skull the history of the blow is usually clear, but in a case in which the history is unknown, or the circumstances arouse suspicion of foul play, a careful examination of the head must be made. Many of the symptoms following injury to the head are due to hemorrhage within the cranium and give rise to the same localizing symptoms found in spontaneous apoplexy. It is in these cases of hemorrhage, usually from a meningeal vessel, that the symptoms of an increasing intracranial pressure are most characteristically shown. Such evidences, however, do not always mean an increasing hemorrhage, for they may be due to œdema which follows the trauma and probably depends upon altered osmotic conditions incident to injury of the brain tissue.

From what has been said above it follows that it is often difficult to determine at once whether coma is due to a cerebral vascular lesion or some other cause, but if the inquiry be made carefully and in such a manner as to reveal the presence of the various conditions most likely to cause mistakes, and the conclusion not too hastily made, grave errors can usually be avoided. The history must be gone into carefully and confirmed in every way possible, the patient examined for the evidence of any bodily injury, the urine obtained and examined, the bodily temperature, respirations and pulse watched carefully, the blood examined, and the ophthalmoscopic examination must not be omitted.

Important as these procedures are, the proof that there has been a local injury to the brain and that the coma is associated in some way with the cause of the lesion, depends upon the discovery of some irregularity in the nervous activities pointing to such a lesion. These symptoms have already been

considered in some detail, but it is at this stage in the examination that the necessity for a systematic search for them must be kept in mind.

The attitude of the patient must be observed. Is he quiet or restless? If restless, are the movements confined completely or largely to one side of the body? Are the movements purposeful or not? If convulsions occur, it is of the utmost importance to note the movements which are first involved; their march, extent, and duration. Is there any difference in the muscle tone of the two sides of the body? If there is such a difference, are the limbs abnormally flaccid or unusually stiff?

Does the patient tend to take any particular attitude? Is the head turned to one side and held there? Are the eyes in the median line or turned to one or the other side? Are they steady, or do they move and to what extent? What are the conditions of the pupils as to their size and reaction to light? Is there any asymmetry of the face during respiration? Do the two nostrils dilate equally on inspiration? Is one cheek blown out more than the other during expiration? Are the visual apertures equally wide? Are the wrinkles on one side of the face more pronounced than on the other?

The movements of the chest and abdomen during respiration should be carefully watched to see whether there is any difference between the two sides, and this should be observed both in quiet and forced breathing.

Does the patient respond to stimuli upon the two sides of the body equally, *i. e.*, is there any evidence of disturbed sensation? Care must be taken not to mistake simple reflex responses for the more elaborate movements that we believe to depend upon the activity of the cerebral cortex.

If the patient is sufficiently conscious, tests should be made to determine whether he sees objects on both sides, so as to exclude hemianopia if possible.

The reflexes must be carefully investigated, particularly as to the relative activity on the two sides. The reflex movements that follow stimulation of the cornea, the nasal mucous membrane, the skin of the abdomen, and, in man, of the inner side of the thigh, are the most important of the so-called skin or superficial reflexes. The response in regard to the movement of the great toe, following stimulation of the sole of the foot (Babinski's reflex and its modifications), has come to have a special importance, and this reflex must be tested for repeatedly and with great care. The deep reflexes —the jaw-jerk, the biceps and triceps reflex in the arms, the knee-kick and ankle-jerk in the legs—must, of course, be noted.

The condition of the skin of the patient is important. Is there any difference in the two sides as to the color of the skin, its temperature, and the amount of visible perspiration upon it?

When unilateral symptoms are demonstrated the presence of a local lesion of the brain is strongly suggested, and when the symptoms have developed in a sudden apoplectiform manner the diagnosis of some one of the vascular lesions is usually justified. At times, however, confusion may be caused by *hysterical paralysis.* In this there is often complete paralysis of the limbs on one side of the body, with marked disturbances of sensation, which may have developed suddenly after a period of real or apparent unconsciousness. At times, even with a thorough understanding of hysteria, together with a knowledge of the characteristics of organic paralysis, the diagnosis may be extremely difficult. Usually, however, the hysterical nature of the paralysis is manifest. The attack occurs in a hysterical subject after some psychical shock. It usually develops somewhat slowly

and without loss of consciousness. The loss of motion is confined, in the great majority of cases, to the arm and leg, and spares the face and the other muscles supplied by the cerebral nerves. In the limbs affected all movements are completely lost, and the paralysis does not show the selective distribution which follows a cerebral lesion. The paralyzed limbs are often quite flaccid, but contractures do not infrequently occur. There is very generally a loss of cutaneous sensation which is apt to involve the whole affected side, including the head. The anæsthesia also involves the mucous membranes, and may include the nerves of special sense.

In sharp contrast to this absolute loss of function, depending, as we believe, upon some abnormality in the field of consciousness, is the quite normal activity of the lower nervous centres. There is no disturbance in the functions of the bladder and rectum, and the pupils react normally to light. The deep reflexes, although at times very active, are not so exaggerated as to cause a clonus, and are usually equal on the two sides of the body. This is also true of the reflexes from the skin. Plantar stimulation gives the normal plantar flexion of the great toe.

It is true that exceptions to the above statement have been recorded, but they are so unusual that the practical question of diagnosis is not affected.

The hysterical loss of function is not infrequently influenced by suggestion. This is particularly true of the anæsthesia which at times can be dispelled or transferred to the opposite side by various procedures—the application of metal disks (Charcot), a mustard plaster (Adamkiewicz), or simply by suggestion in the waking state or during hypnosis. The paralysis may be similarly influenced and absolutely disappear after tetanization of the muscles by the application of the faradic current to the nerve trunks.

Under normal conditions the muscular activity involved during any strong voluntary effort spreads far beyond the muscles primarily concerned, *i. e.*, there is a synergistic action of many other muscles. This may consist of contraction or relaxation, and may even involve movements of the opposite side of the body. It seems to depend upon an automatic action of the lower centres,[1] and should not be affected in hysteria. Hoover's[2] observation seems to be an illustration of this. He has shown that when a normal person lying on his back endeavors to lift the extended leg from the couch, the opposite leg is pressed downward, and that in an organic hemiplegia this same pressure occurs when the patient tries to lift the paralyzed leg. If in such a case the patient lifts the unparalyzed leg the downward pressure of the paralyzed leg is proportionate to its remaining strength. If the hemiplegia is due to hysteria the action is different. When the patient raises the non-affected leg the opposite leg, totally paralyzed for voluntary effort, is pressed strongly into the couch, while if he be told to endeavor to raise the paralyzed leg there is no movement on the non-paralyzed side, indicating that no outgoing stimuli have left the cerebral cortex, just as if he were consciously simulating the paralysis. Risien Russell[3] has called attention to a somewhat similar phenomenon occurring in hysteria, but in this case it concerns the lack of relaxation in the antagonistic muscles.

Hysterical hemiplegia was formerly believed to affect the left side much more frequently than the right, but Ernest Jones,[4] in an analysis of 277 cases,

[1] Sherrington, *Quart. Jour. Exper. Phys.*, 1909, ii, 109.
[2] *Jour. Am. M. Ass.*, 1908, li.
[3] *Brit. Med. Jour.*, 1908, i, 608.
[4] *Rev. Neurol.*, 1908, p. 193.

found no marked difference between the two sides; indeed, the right side was a little more frequently affected than the left, the percentage being 54.2 on the right side and 45.8 on the left.

It is often stated that hysteria may simulate every type or form of organic paralysis, but this simulation is usually superficial and disappears before a detailed examination. Mistakes are much more commonly made by overlooking what is at least equally true, that many organic diseases may simulate hysteria, and that it is not at all uncommon to have a combination of the two conditions. This was well illustrated in the case of an unmarried woman, aged forty-five years, who after a long and severe emotional shock had an attack of dizziness associated with paralysis of the left arm and leg. She was admitted to the hospital in a highly nervous state, and the diagnosis of hysteria was made. The paralysis of the arm cleared up rapidly under suggestion, but that in the leg developed the typical signs of an organic paralysis. The distribution was characteristic, as was the spastic condition with well-marked ankle clonus. The patient was subsequently under my care for a number of years, and passed through numerous hysterical storms, but the paralysis of the leg remained constant and resisted all forms of suggestive treatment.

**Diagnosis of the Vascular Lesion.**—After the diagnosis of apoplexy has been made, *i. e.*, that the patient is suffering from the effects of a sudden vascular lesion, it becomes of great importance to determine, if possible, whether there has been a rupture of an artery and blood has escaped into the brain, or whether an artery has become plugged by a thrombus or by an embolus, causing cerebral softening. The diagnosis of the cause of the apoplexy in any individual case can be made only with a certain degree of probability. This degree may be so high as to almost reach certainty, or, on the other hand, so low as to amount to practically little more than a guess. Unfortunately, in the majority of cases the diagnosis has to remain very uncertain.

The brain lesion is the result of a vascular state and it is this that must be kept in mind. The vascular conditions liable to occur at the various periods of life differ, and so the age of the patient is an important point in diagnosis. In childhood and youth the spontaneous rupture of a cerebral vessel is rare, and apoplexies at that time of life, when not dependent upon inflammation, are very generally due to cerebral softening and are in association with some definite cause, acute diseases, syphilis, etc., for thrombosis, and endocarditis for embolism.

The diagnosis of cerebral embolism depends upon the determination of the source of the embolus, and when such is found the inference is usually correct. Gowers points out that the presence of endocarditis is not excluded by normal heart sounds, and under conditions in which it is common, chorea, for instance, it should be suspected even when it cannot be determined. In the later decades, when degenerative changes in the vessels have become common, the association between valvular heart disease and apoplexy has less significance as indicating the occurrence of an embolus, but even in early life one is occasionally surprised to find at autopsy a rupture of a vessel when its occlusion was confidently expected.

Between twenty and forty most cases of apoplexy are due to thrombosis secondary to syphilitic arterial disease, and then, or even before and after this period, when the cerebral accident occurs within a few years of

the primary syphilitic infection, the diagnosis of thrombosis is the most probable.

Cerebral hemorrhage, in spite of its rarity in the first half of life, should be suspected whenever the apoplexy has the distinctive characteristic of such an accident, especially when it occurs in apparent health in a subject in whom syphilis is unlikely or in conditions in which hemorrhages are common, leukæmia, pernicious anæmia, eclampsia, etc.

In the latter or degenerative period any one of the vascular conditions may occur. It would be important to know their relative frequency, but accurate knowledge could be obtained only from mortality statistics in large communities in which the cause of death was determined by autopsy.

In the cases dying in the Johns Hopkins Hospital softening was more common than hemorrhage, there being 30 of the former and 26 of the latter. Table VI shows the occurrence of these lesions in the various decades at the time of death. Ludlum,[1] who analyzed the material in Spiller's laboratory in the University of Pennsylvania, found a still greater frequency of softening, his figures being 69 of such lesions to 24 of hemorrhage. In the Royal Victoria Hospital, Montreal, the figures of which Dr. Adami put at my disposal, the relative frequency of the lesions was about that found at the Johns Hopkins Hospital, being 35 of softening and 29 of hemorrhage. These and other figures are given in Table VII.

TABLE VI.—*Johns Hopkins Hospital.—Fatal Cases.*

| | Decades. | | | | | | | | | Total. |
|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | |
| Cerebral softening | 1 | 0 | 3 | 3 | 8 | 9 | 2 | 3 | 1 | 30 |
| Hemorrhage . . | 0 | 0 | 1 | 4 | 4 | 12 | 4 | 0 | 1 | 26 |

TABLE VII.

| | Hemorrhage. | Softening. |
|---|---|---|
| Johns Hopkins Hospital . . . . . | 26 | 30 |
| Dr. Ludlum (Philadelphia Hospitals) . . . | 24 | 69 |
| Royal Victoria Hospital, Montreal . . . . | 29 | 35 |
| Montreal General Hospital, Dr. Duval . . . | 53 | 44 |
| Boston City Hospital, Dr. Mallory . . . . | 132 | 78 |
| Presbyterian Hospital, New York[2] . . . . | 112 | 48 |
| University College Hospital, London . . . | 123 | 37 |
| Total . . . . . | 499 | 341 |

From these figures hemorrhage appears to be considerably more frequent, but as Gowers points out this is probably due to the fact that as hemorrhage is more often quickly fatal than softening, more such cases would be expected

[1] *Jour. Nerv. and Ment. Dis.*, 1909.
[2] Starr, Nervous Diseases, 1909, p. 477.

to die in hospitals. He thinks it probable that softening is in general the more common lesion. Clinically, the probable diagnosis of cerebral softening, in the writer's experience, has been much more often justified than that of hemorrhage.

The character of the apoplectic stroke itself gives some indication as to its cause. Premonitory symptoms when they have been present for two or three days, especially if there is a history of several slight previous cerebral attacks, suggest the gradual onset of thrombosis. In cerebral hemorrhage slight symptoms not infrequently precede the culmination of the attack, but do so for a much shorter period, from a few minutes to a few hours, and there may be the history of a previous apoplexy of considerable severity, but not commonly of repeated slight attacks. Hemorrhage may occur, however, after several thrombotic attacks.

The occupation of the patient at the time of the stroke may give some indication, for hemorrhage is to be expected when the circulation is active, *i. e.*, when the blood pressure is high and the heart is acting forcibly, and thrombosis under the opposite conditions, when the circulation is depressed. Although it is uncommon to get the history of severe physical effort at the time of a cerebral hemorrhage, still more cases occur during the active hours of the day, and rest in bed and sleep seem to protect to a certain extent against this occurrence. Thrombosis is more common during sleep and rest in bed than hemorrhage, and this probably accounts for the preponderance of cerebral softening found at autopsy in hospitals where old chronic cases are treated. If one could determine exactly the state of the circulation at the time of the apoplexy it would be a great help in the diagnosis between these two lesions. Gowers lays particular stress on this point.

The essential thing to know is the circulatory conditions at the site of the lesion in the brain, but the condition of the peripheral circulation is an imperfect index of this, especially if nothing is known of the patient previous to the apoplectic attack. High blood pressure is an almost constant accompaniment of advanced arteriosclerosis, and may precede and accompany thrombosis as well as hemorrhage and should not be given too much weight. On the other hand, a feeble, soft pulse, with a weak, irregular heart, points strongly to thrombosis.

Coma is much more frequently absent in thrombosis than in hemorrhage, and when it develops rapidly it strongly suggests the occurrence of the rupture of a vessel. This is particularly so when the focal symptoms are not pronounced. On the other hand, a widespread paralysis, which has developed with slight or transient disturbances of consciousness, is usually due to thrombosis. However, there are many cases of thrombosis which have the typical development of hemorrhage, as well as a group of hemorrhage cases that have the onset typical of thrombosis.

A marked fall in the bodily temperature immediately after the apoplectic stroke speaks for hemorrhage, as do a rapidly rising blood pressure, Cheyne-Stokes respiration, and the other signs of increased intracranial pressure.

Convulsions, particularly if repeated, point to an irritative lesion in or near the cortex, and are much more frequently associated with softening than with hemorrhage, and more often with embolism than with thrombosis. Cerebral softening bears the same relation to localized paralyses—monoplegias, the different types of aphasia, hemianopia, etc. Hemiplegia is the usual result of hemorrhage, but is also common with softening.

The frequency with which the different parts of the brain are implicated by the different vascular lesions determines the liability to the occurrence of the various focal symptoms with each and should be kept in mind as a help in the differential diagnosis.

Hemorrhage, in the great majority of instances, is from some one of the central nutrient arteries, and involves the basal ganglia and internal capsule. This was true in 19 of the 27 lesions analyzed by the writer. The other lesions were situated as follows: twice in the frontal, twice in the occipital, and once in the temporal lobe. The cerebellum was involved twice, once with the pons, and in one instance the clot was in the pons and medulla. In Ellis' 31 cases it was in the region of the basal ganglia 26 times, not designated once, and was once each in the following situation: occipital lobe, parietal lobe and postcentral gyrus, cerebellum and pons. Hemorrhages into the pons are more frequent than those in the cerebellum (v. Monakow). True cortical hemorrhages are rare and are usually small.

Softening frequently involves the cerebral cortex; 20 of my 30 cases were so localized, and the basal ganglia and internal capsule showed softening 8 times, and in 2 instances the focus was in the lateral aspect of the medulla. Ludlum's experience was different. He found among 69 areas of softening only 9 in the cortex, all the others being in the neighborhood of the internal capsule. Thrombosis may occur anywhere, but is more frequent in the distribution of the middle cerebral artery and in that of the posterior cerebral. Emboli, in the great majority of cases, are lodged in the middle cerebral artery and its branches.

From these and other observations it appears that when the symptoms indicate a capsular lesion the probability is somewhat, although not much, greater that it is hemorrhage, but when they indicate a lesion in some other situation, the cortex particularly, it is apt to be softening.

**Prognosis.**—The diagnosis of a cerebral vascular lesion always entails a serious prognosis, not necessarily as to the outcome of the present attack, but very generally as to the underlying vascular condition, which must remain a source of constant danger to the patient. The chief exceptions to this are those accidental cases occurring in the young in relation to acute infectious diseases, parturition, etc., and of certain cases due to syphilitic endarteritis which is amenable to treatment.

The great majority of apoplectic attacks are recovered from. In my own list there are the histories of 740 patients, most of whom had recovered from the attack and applied for relief of the residual paralysis. There were 448 of these. The fatal cases in the present list were among those treated in the hospital, and number 97 out of a total of 292 hospital cases. This includes a number in which the apoplexy was only a contributory and not a direct cause of death. These figures give no idea of the relative frequency of death. The dispensary cases were all, of course, drawn from those that recovered, and there is no means of ascertaining the fatal cases of apoplexy occurring in the same community. The percentage of deaths (33.2 per cent.) among the hospital patients must be much greater than that which obtains in general, for it applies to a special group composed largely of the more severe cases or those complicated by other conditions.

In many cases previous attacks had occurred; usually one previous attack, not infrequently two and in several instances there were histories of three or more definite strokes. The time between the attacks may be short.

It is usually estimated in months, and often there is an interval of years. The longest interval was sixteen years between the first and last attack. In this case the second attack occurred in two years, but there was an interval of thirteen years between the second and third and two between that and the fourth The idea that the third attack is necessarily fatal has often been refuted. Among our cases, 79 died in the first, 10 in the second, 4 in the third, 1 in the fourth, and 3 after several attacks.

The gravity of the prognosis of any attack increases with the suddenness with which the coma has occurred, with its depth and with its duration. If the coma shows no sign of lessening after twenty-four hours the prognosis is very grave and becomes more and more grave the longer the coma persists. Patients have, however, been known to recover from coma which has lasted for a long time, even up to six weeks. The writer saw a woman, nearly eighty years old, who had been in coma for three days, return suddenly to perfect consciousness as from sleep, much to the surprise of her watching relatives and to the chagrin of her physicians, who had given an absolutely hopeless prognosis. If the coma, at first slight, becomes later more pronounced, or returns after the patient has regained consciousness, it is of more serious import than the same degree of unconsciousness at or shortly after the onset of the attack.

When the initial fall of temperature, common in cerebral hemorrhage, persists and increases, it is of very grave import. On the other hand, when the temperature rises quickly, or the fever lasts longer than the first two or three days, the outlook is grave, and if at any time the temperature reaches great heights, it is almost always a signal of approaching death.

Marked changes in the respiration, particularly when it assumes the Cheyne-Stokes type, are of grave importance as indicating embarrassment of the respiratory centre. Cheyne-Stokes respiration, however, does not always mean immediate death; it may persist or come and go during many days. It may also have been long present before the apoplexy in association with cardiac or renal disease.

Rapidly rising blood pressure as indicating increasing intracranial pressure is, of course, a serious symptom, but a constantly falling blood pressure from a previously high level is one of the surest signs of approaching death. Cardiac failure has its usual significance. Any symptoms pointing to involvement of the lungs are very important, for many cases of apoplexy die of a terminal pneumonia.

An increasing swelling of the optic nerves is a serious symptom as indicating an increasing intracranial pressure, but it should not be forgotten that it may be in association with uræmic œdema of the brain and may entirely subside.

Any estimate as to the duration of an attack is very uncertain. The most rapidly fatal cases usually live for several hours, and a patient may survive a large hemorrhage for several days, even when it has ruptured into the ventricle. The time of death varies, among other things, with the character of the lesion, and Jones[1] gives an interesting table compiled from cases proved by autopsy:

[1] *Brain*, 1905, p. 546.

| Lesion. | Number of cases collected. | Percentage dying within 24 hours. | Within a week. | Within a month. |
|---|---|---|---|---|
| Hemorrhage | 828 | 30.4 | 63.8 | 79.4 |
| Thrombosis | 158 | 15.8 | 38.0 | 74.7 |
| Embolism | 273 | 8.0 | 35.5 | 56.0 |

In our own experience the average time of death in all fatal cases when this could be determined was about eight days. For the cases proved by autopsy it was 6.2 days; for cerebral hemorrhage, 5.3 days, and for cerebral softening, 7.5 days. The few cases in which the thrombosis was secondary to embolism had a somewhat longer duration—nine days.

The age and general state of health have also an influence on the prognosis. Young, strong individuals often survive a severe cerebral shock that would be rapidly fatal at a later period of life. On the other hand, in elderly people whose arteries are badly diseased, usually with associated cardiac and kidney trouble, a very slight stroke may be the beginning of a spreading thrombosis, and instead of the patients recovering in a few days they become duller, lapse into coma, and die after a protracted illness.

**Treatment.**—**Prophylactic.**—This should begin with the prevention of the conditions liable to bring about vascular degenerations. As our knowledge increases as to the cause of arteriosclerosis, it is probable that we will know better how to ward off or delay its occurrence. The enormous part that syphilis plays in the etiology of the apoplexies of early adult life makes the importance of its prevention and treatment too obvious for comment. None of these cases should occur, and we may hope that our rapidly increasing knowledge of this disease and the extension of a higher standard of moral hygiene will make them more uncommon.

When vascular conditions are present that make the rupture of the occlusion of an artery probable, we can, unfortunately, do but little to restore the integrity of the vessel wall, except when the change is syphilitic. The treatment of the primary syphilis is so rarely thorough that a caution in this regard does not seem out of place here. The writer's belief is that even after five or six years of continuous treatment it would be wiser for the patient to take a two or three months' course of iodide of potassium and mercury twice every year. Where there is any evidence of involvement of the cerebral arteries in individuals under forty, syphilis should be suspected and thoroughly treated, even when there is no history of syphilis and its possibility seems unlikely.

Patients with damaged bloodvessels should avoid excesses of all kinds, not only those of alcohol and tobacco, etc., but also excessive mental or physical labor. The diet should be light, the bowels kept freely open, and the other measures recommended for arteriosclerosis should be employed. If such measures do not cause a reduction in the blood pressure when it is high, it is doubtful whether vasodilators are of any permanent value. Apoplexies occur during every conceivable activity of daily life, and so it is inadvisable to restrict the patient within too narrow limits.

**Apoplexy.**—Here as elsewhere effective treatment must depend upon an accurate diagnosis. In this instance similar symptoms may be due to vascular lesions of exactly opposite character—on the one hand the rupture of an artery, and on the other its occlusion, conditions which indicate quite different procedures if they are to be met directly. It is impossible in many

cases to be certain which of these lesions is present, and the treatment must be such as would be applicable in either condition. It is often of the utmost importance to decide whether active measures should be employed in the endeavor to influence directly the initial lesion, especially whether the patient should be bled or undergo a surgical operation, or whether the treatment should be conservative and symptomatic.

**Bleeding.**—If the patient be seen immediately after the stroke, and the symptoms all indicate the bursting of an artery (a rapidly developing coma, lowered bodily temperature, a strongly acting heart, with a full, bounding pulse), and we have reason to believe that the high blood pressure preceded and did not follow the hemorrhage, bleeding from a peripheral vessel might perhaps be indicated. This would be done in the endeavor to so lower the pressure inside of the ruptured vessel that the hemorrhage would stop and the extravasated blood be allowed to clot and further destruction of the nervous tissue be prevented. It must be remembered that the symptoms of apoplexy are not all dependent upon the local effect of the extravasated blood, and that many of them, coma itself among the rest, are often believed to be due to reflex effects, among which anæmia of the cortex is of great importance. If this be true, lowering of the arterial pressure could hardly be expected to help.

High blood pressure is by no means of itself an indication for bloodletting, for this may be a result and not a cause of the hemorrhage, as when the blood pressure continues to rise, and there are other evidences of undue pressure on the medulla (Cheyne-Stokes respiration, etc.). Under these circumstances bleeding would seem to be absolutely contra-indicated. If the apoplexy be due to the occlusion of a large bloodvessel, and this may be associated with high blood pressure, there can hardly be an excuse for bloodletting.

When we remember how rarely it is possible to be certain that we are dealing with a ruptured and not with an occluded vessel, and even if we be sure that a hemorrhage has occurred, how uncertain the indications for the rapid lowering of the general blood pressure are, and, indeed, how incomplete our knowledge is of the effects of bleeding on the cerebral condition, we will see how seldom we have a definite indication for bloodletting in apoplexy.

Around this question has been waged many battles, the history of which is most interesting. In the early part of the last century bleeding in apoplexy was the universal treatment, and was immediately applied to every case that was seen. Experience taught its futility in many cases, for by the middle of the last century Todd, in his clinical lectures, speaks against its general employment in apoplectic cases, and Trousseau devotes one of his lectures to this subject alone. Hughlings Jackson, writing about 1868, in Reynolds' *System of Medicine*, says: "So far as I have observed, however, the practice in this country must be exceedingly rare, as I have in the whole course of my life seen but one person bled for cerebral hemorrhage."

Shortly after this the practice seems to have fallen into almost complete disuse, but of late years there has been, to a certain extent, a revival. All the recent authors refer to bleeding, but there is very little agreement as to the benefit which follows it, and it is difficult to find exact observations. von Monakow finds little reason, either theoretically or practically, for its

employment, and Cushing speaks strongly against its use, but Gowers, Starr, Oppenheim, and others recommend it more or less strongly. Goldscheider,[1] in a recent lecture, after considering the theoretical objections, concludes that these have been pushed too far, that they do not stand on a firm basis of known facts, and that although it must be acknowledged that permanent good effects are extremely rarely seen after bleeding in severe apoplexies, still he believes that it is indicated when the diagnosis of hemorrhage is certain and when the face is congested and the pulse is full and hard. He would bleed not only at the onset of the attack, but even later if coma had persisted with symptoms of hyperæmia, or if the symptoms, which were at first slight, became more pronounced. In our own cases in which it has been used the writer has not been able to see evidence of the least permanent benefit. Even the blood pressure, although it is reduced during the operation, again rises rapidly to near its original height. Only the most urgent cases were bled, however, and this may account for the lack of good results.

*Surgical Interference.*—Spencer and Horsley suggested, in 1889, that hemorrhage from the middle cerebral artery might be controlled by compression of the carotid artery, this being applied by digital pressure, either through the skin or upon the exposed vessel, or, indeed, by permanent ligature of the vessel. This apparently has never been carried out.

Cushing[2] has urged that the clot be dealt with directly, and gives details of the necessary surgical procedure. He bases this on the theory that the hemorrhage occurs rapidly, has ceased when the physician sees the patient, and that the immediate symptoms are, for the most part, due to increased intracranial pressure, which can best be relieved by opening the cranial cavity and removal of the clot. He also says "that such a removal of a clot will, in all probability, greatly lessen the residual paralyses, which doubtless are largely due to a slow pressure atrophy of adjoining tracts rather than to their actual destruction by laceration." Cushing has operated on a number of desperate cases of capsular hemorrhage which occurred in the medical service, and also on some others, and in several instances certain of the alarming symptoms improved markedly, at least for a time. The most certain effect was perhaps on the excessively high blood pressure when present. At times the change in blood pressure is not so marked, and, indeed, a decompression may have little or no effect. This is particularly so when the blood pressure has accompanied arteriosclerosis and has preceded and not followed the apoplexy.

It is not possible at present to estimate the benefits of surgical interference in apoplexy. Too few cases have as yet been operated upon, and these have all been of the gravest character, but so far as our experience goes, it seems to teach that when the patient's life is endangered by a rapidly increasing intracranial tension, as shown by a rising blood pressure, Cheyne-Stokes respiration, etc., a decompression is clearly indicated. Whether the clot itself can be successfully evacuated without subjecting the brain to more trauma than is justified appears to await demonstration. The evidence which we now have can only lead us to hope that the operation may be so developed that the less urgent cases of cerebral hemorrhage can be successfully attacked.

[1] *Deutsche med. Wochenschr.*, 1907, xlviii, 1977.
[2] Keen's *System of Surgery*, iii, 215.

A dangerous increase in intracranial pressure may also follow thrombosis and embolism, and a decompression is equally indicated in these cases. Cushing has operated on a number of such cases, in several of which the procedure seems to have saved the patients' lives. He has also had some brilliant results in removal of the subdural clot in hemorrhages incident to birth.

The physician usually sees the apoplectic patient after the damage has occurred, and far too often hears the history of procedures that could only have done harm. The belief is very general among the laity that the early symptoms of a cerebral lesion, drowsiness, tingling or weakness in the hand or leg, etc., can best be combated by physical exercise, and that to give way to them is to court disaster. Even physicians do not seem always to appreciate the importance of absolute rest to a patient who shows the evidences of a beginning hemorrhage or other vascular lesion.

If the patient be conscious he should be cautioned to make as little voluntary effort as possible, not even to speak, this especially if there are evidences of aphasia. No endeavor should be made to arouse an unconscious patient, and the physician should be careful to make his examination with as little disturbance to the patient as possible. The patient must not be moved more than is absolutely necessary, and it may be wise not even to transfer him to another room. As illustrating how important this is regarded by some, Gowers refers to an instance in which Sir Russell Reynolds would not permit the removal of a patient who had an apoplectic stroke while consulting him, but cared for her on a couch in his office until her death ten days later. Absolute physical tranquillity of the patient is insisted upon, so that as far as possible nothing may interfere with the spontaneous stopping of the hemorrhage, or of the reëstablishment of the circulation if there be an occlusion of a bloodvessel.

The patient should be in a room protected as much as possible from noises and other sources of irritation, too bright a light, etc. Only those necessary for the care of the patient should be permitted to enter it. He should be in bed with the head slightly raised, special care being taken that the neck is not bent and that nothing interferes with the return of blood from the head. When the patient is profoundly unconscious and the breathing is stertorous, and, indeed, perhaps in general, he had better be placed on his side, so that the tongue does not fall back and impede respiration. Bowles, who has called particular attention to this, insists that the apoplectic patient should always be kept in the lateral position, the paralyzed side being down.

The state of the circulation is of utmost importance. If the heart be feeble and weak, it must be regulated. Stimulants, such as nitrous ether, camphor, caffeine, and small doses of digitalis, are recommended, and even alcohol in habitual drinkers. The opposite conditions, a strongly acting heart, high blood pressure, etc., which may indicate bleeding or surgical interference, have already been considered.

When a hemorrhage is reasonably certain, active purgation with calomel, croton oil, etc., is almost universally employed, and even in thrombosis it is the custom, although here milder purgation is recommended. Goldscheider, however, speaks strongly against this practice, for he doubts whether the good derived from the slight lowering of the blood pressure, even if this occurs, counterbalances the danger from the physical disturbances

incident to the procedure, and he would do nothing for the first day or two except to endeavor to relieve the lower bowel by the use of enemata, care being taken not to disturb the patient more than is necessary. The bladder is apt to be emptied involuntarily, but care should be taken to see that there is no retention, and if this occurs the bladder should be emptied two or three times a day by the use of the catheter.

Ice to the head has probably no influence on the circulation within the skull, and so the common habit of applying an ice cap is harmless, and as it appears to be grateful in certain cases it may be used. In hemorrhage, agents that increase the coagulability of the blood are indicated, and Gowers suggests that salts of calcium or the hypodermic injections of 0.2 gr. of arsenate of sodium may be used, and that for the opposite effect, to make the blood less coagulable, citric acid in the form of lemon juice in plenty of water may be tried in cases of thrombosis.

When the patient is in convulsions or very restless he must be kept quiet by the use of bromide, chloral, veronal, and other hypnotic drugs. Morphine, on account of its depressing effect on the respiratory centres, should be given only when absolutely necessary.

In all cases when there is a possibility that the lesion is thrombosis due to syphilitic disease of the bloodvessels, active measures must be employed —mercury by injections or inunctions. The first method requires little manipulation of the patient, and should be used first even if inunctions are applied later (Goldscheider).

Iodide of potassium in increasing doses should be added as soon as the patient can swallow readily.

Directly after an apoplectic stroke the patient should not be disturbed by attempts at feeding. The lips may be moistened with water and the mouth cleared with a swab of gauze, saturated with some cleansing mouth wash. If the patient remains unconscious for a long time and appears to be becoming weak for lack of nourishment, nutrient enemata may be given. If these are not retained, infusions of salt solution may be resorted to, although such heroic procedures are rarely justified. When the patient is able to swallow (and this ability should be carefully determined by the physician) liquids in small quantities may be given after the first few hours. Even in the mildest cases the diet must be kept very light for the first few days, and then increased very gradually.

In slight attacks it is always difficult to keep the patient quiet, but even in the mildest cases the physician must insist upon a rest in bed for at least a week, and it would be safer for two.

**Sequels.**—Destruction of any part of the brain must entail some loss of function, and when the focus is large, or so situated as to interfere with an easily recognized function, we have symptoms. Such focal symptoms commonly follow vascular lesions within the brain, and not infrequently make up most of the clinical picture, and instead of apoplexy we have palsy, as the older writers would say. The symptoms vary with the position of the focus, and a knowledge of cerebral localization is necessary for a clear understanding of this relationship.

It has been pointed out that focal symptoms follow hemorrhage more frequently than softening within the brain. This is due to the fact that hemorrhage usually occurs in the region of the internal capsule and may

cause destruction of important tracts, whereas softening may be anywhere, often in the cortex, of small extent and involve a silent area.

In most cases of apoplexy from all causes, however, focal symptoms are present. Such may be the first sign of the accident, or may be evident only when consciousness begins to return. These symptoms may be so transient that they can hardly depend upon a destruction of tissue, but must represent a loss of function due to some passing state—anæmia or œdema. Too often the disability persists as evidence of an irreparable cerebral defect.

After an attack of apoplexy the patient may show little or no *intellectual weakness*, but usually a careful inquiry will reveal a distinct lowering of the mental vigor. The patient is not the man he was before the stroke, but tires more easily, is petulant, emotional, and often depressed. He is often unable to control the expression of his emotions, and weeps tumultuously upon the slightest cause, or even when he may not be conscious of any sad feeling. Less commonly his laughter may be also uncontrolled. Similar symptoms due to arteriosclerosis are often present in a less degree before the stroke, which appears to have merely intensified them and to have reduced the patient to a lower intellectual level. When the lesion is a large one, or associated with widespread arterial disease, the defect may amount to almost complete dementia. On the other hand, especially in young subjects, a severe stroke may be survived with astonishingly little effect on the mental functions. Pasteur, who was for many days thought to be dying from an apoplexy when forty-six years old, subsequently did much of his brilliant work, and lived to be seventy-three.

If the lesion be so situated that it involves the speech area, *aphasia* follows and may be permanent. This is often associated with a marked lowering of the intellect, but at times this is not evident.

The physical defect that most often remains is *hemiplegia*, which results whenever the pyramidal tract is implicated, as in the common lesion of the internal capsule, or in any other part of its course. In severe cases the paralysis is widespread, affecting generally all of the movements of the opposite side of the body. The arm and leg are motionless, and often the facial muscles are powerless, and the common statement that one side of the body is completely paralyzed seems justified. A closer examination shows that this statement is far too general, and that there are many muscles on the affected side which are little, if at all, implicated in the paralysis, and that those affected are so to an unequal extent. This attracted the attention of the older observers, and various hypotheses were advanced to explain it. The most widely accepted one was that which Broadbent[1] advanced in 1866, when he pointed out that the muscles which escaped were those which acted more or less constantly in association with corresponding muscles of the opposite side of the body, *i. e.*, bilaterally acting muscles, and that the degree of paralysis, when present, corresponded to the degree of their unilateral activity. This he explained by conceiving a more or less intimate association between the lower spinal nuclei. When muscles acted always together the connection was so close as to practically make one nucleus, which could be stimulated equally well from the right

[1] *Brit. and For. Med.-Chir. Rev.*, reprinted in *Selections from the Writings, Medical and Neurological, of Sir Wm. Broadbent*, London, 1908.

or left cerebrum, *i. e.*, its movements, composed of both right- and left-sided muscles, were represented on both sides of the brain, and a lesion on one side of the brain would not affect them. When muscles, although usually acting together, could act unilaterally, the connection between the lower nuclei was less intimate, and their bilateral cerebral representation less complete, so that a unilateral lesion would paralyze them more or less, depending upon the degree of their unilateral activity. As entirely bilaterally acting muscles, Broadbent considered those of the eye, back, neck, and chest. He regarded the upper facial muscles as being largely, but not completely, bilaterally acting muscles; the lower facial muscles he thought were almost entirely unilaterally acting. The muscles of mastication and of the tongue he also classed among those which act both symmetrically and unilaterally. Subsequent investigations have confirmed Broadbent's observations in a remarkable way, and his explanation of the facts is very generally accepted as fundamentally true.

That movements and not individual muscles are represented in the brain (Hughlings Jackson) and that it is these which are affected in hemiplegia, is nowhere better seen than in relation to the external muscles of the eye. These muscles are never individually paralyzed, but the conjugate movements to the right or to the left are not infrequently affected, particularly in the early stage of a hemiplegia. In each of these movements muscles on the two sides of the body are involved. The rapid recovery of these movements cannot easily be explained on Broadbent's hypothesis, for although using muscles on the two sides of the body, the movements are themselves unilateral. It may be that these movements are represented in more than one part of the cortex, anteriorly in the motor cortex and posteriorly in the occipital lobe, and therefore one lesion would not be likely to destroy the pyramidal tract and the path from the occipital lobe.

The condition of the muscles of the eye in hemiplegia has been lately carefully reviewed. Mirallie and Desclaux tested the strength of the muscles by their power to overcome prisms, and came to the conclusion that in every case of organic hemiplegia, in which the face was the least involved, there was a weakness of all the muscles of the eye on that side, and also to a less extent on the sound side. S. A. K. Wilson,[1] however, working with Marie's material at the Bicêtre, and employing the same methods, came to exactly the opposite conclusion, *i. e.*, he was unable to determine any weakness of the external muscles of the eye in cases of hemiplegia beyond that which was often found in healthy individuals.

Ptosis due to weakness of the levator palpebræ superior is at times observed with lesions of the cerebral hemisphere, and the involvement of this muscle in hemiplegia is cited as an exception to the general exemption of the muscles supplied by the third nerve. Ptosis is, however, rare from a lesion within the cerebral hemisphere, and Wilbrand and Saenger, in their careful analysis of the literature, were able to find the records of very few satisfactory cases. Wilson also speaks of its rarity. When the lesion is in the brain stem and can directly implicate the nuclei of the ocular muscles, their paralysis is, of course, a common symptom.

The movements of mastication on the paralyzed side are more or less weak. This was known to Broadbent, and has lately been confirmed by

[1] *Rev. Neurol. and Psychiat.*, 1904, p. 265.

Mirallie and Gendron, who found that the weakness was particularly of the masseters and pterygoids, and was more marked the more recent the case of hemiplegia.

Although Broadbent himself recognized that all the facial muscles were implicated in varying degrees in the paralysis of hemiplegia, those about the eye being less affected, later observers applied his hypothesis more strictly, and the statement that the upper facial muscles were completely spared in a lesion of the brain, received general acceptation. A careful review of the question by a number of recent writers has confirmed Broadbent's original statement in a remarkable way. In early cases, when the face is at all implicated, it is usually so in its entirety. The movements of the upper facial muscles, in a great majority of cases, recover rapidly, and after a few days their weakness is revealed only by a careful examination. It may be noticed that the eyebrow on the paralyzed side is a little lower than on the other, that the movements of the forehead, although present, are weaker than those on the other side, and that the eye cannot be closed so quickly and firmly. The patient is unable to close voluntarily the eye on the paralyzed side without at the same time closing the opposite eye, even when he may have been able to do so before the stroke, and this may be the only remaining evidence of the weakness of this group of muscles (the orbicularis sign). The paralysis of the muscles about the mouth (the lower facial group) is much more permanent, and in some cases a slight drooping of the corner of the mouth during voluntary effort, may be the last evidence of a hemiplegia. Although this relation between the degree of paralysis between the upper and lower facial muscles holds good for the vast majority of cases, exceptional cases have been reported in which the condition is just the reverse, the upper group being more paralyzed than the lower.

The movements of the larynx are as typically bilateral as any in the body. Horsley and Beevor found that stimulation of the cortex or the internal capsule of either hemisphere caused movements of both vocal cords. In hemiplegia these movements are not permanently affected, although some involvement of them probably takes place in the difficulty in speech so common in the first few days after an apoplexy.

The movements of the soft palate are rarely referred to in detail in relation to hemiplegias, but Tetzner[1] has studied this question and finds that the condition varies greatly. In certain cases there is complete paralysis, and in others the palate moves only during gagging and then usually symmetrically. In a number of cases, fifty-two, the palate moved during phonation, and in those he found the movements symmetrical, the soft palate in front and above the anterior arch being drawn backward and toward the paralyzed side, making the picture of a tent, whose point was directed toward the paralyzed side. In a few cases this abnormal movement of the palate was the only remaining sign of the paralysis.

The tongue when protruded not infrequently deviates toward the paralyzed side. This is usually explained by the statement that the muscles of that side, being weak, are overbalanced by their opponents, and when the tongue is protruded it is pushed over toward the weak side. Beevor[2] has shown that the condition is not so simple, and he points out that stimula-

[1] *Neurol. Centralbl.*, 1909, p. 520.

[2] *Brain*, 1906, p. 487.

tion of the cortex reveals two separate centres for the movements of the tongue, one in the upper part of the tongue area, which causes deflection of the tongue toward the opposite side, and another situated lower and more anterior, the stimulation of which causes protrusion of the tongue in the mid-line. This latter affects equally the movements on the two sides of the tongue. It is some disturbance of this movement, protrusion of the tongue in the median line, that causes the deflection of the tongue toward the hemiplegic side, and not the voluntary movement of turning the tongue toward that side. This latter is a unilateral movement, and Beevor has shown that it is affected in hemiplegia, the patient having more difficulty in putting the tongue into the cheek on the paralyzed side than in moving it in the opposite direction.

The explanation of the deflection of the tongue when the patient endeavors to protrude it straight is not clear, but Beevor advanced the tentative theory that although each hemisphere contains the movements for both sides, still for a perfectly balanced movement the action of both hemispheres is required, and a lesion in one causes a greater amount of weakness in the movements of the opposite side. This amounts to an elaboration of the common statement.[1]

The movements of respiration are bilateral, and are so little affected in hemiplegia that in the records of the routine examination they are very generally noted as being equal. In a number of cases where special attention has been called to this point no difference in expansion of the two sides of the chest could be determined. At times, however, there is a marked difference, the movements on the paralyzed side being less than those of the opposite side. Hughlings Jackson[2] pointed out that the condition differed in quiet and forced breathing; during quiet breathing the movements on the paralyzed side being greater, while in voluntary or forced breathing they were less than those on the opposite side. This was confirmed by a number of investigators, but Samuel West[3] could find no such difference. In his carefully studied case the movements were less on the paralyzed side during every character of respiration, and the chest on that side actually bulged during the strong expiratory effort of coughing.

The muscles of the trunk act both bilaterally, as in forward and backward flexion of the body, and also unilaterally, in the movements toward the right and left. These movements are affected differently in hemiplegia, those acting bilaterally being but little affected, the muscles on the paralyzed side acting nearly as strongly, although somewhat later than their fellows. The lateral movements may be quite differently affected. Beevor[4] reports a case which again shows that movements and not muscles are represented in the cerebrum. The case was one of left hemiplegia. When sitting, the patient could not lean to the left with as much force as to the right, although he showed a tendency to fall toward the left, *i. e.*, there was a weakness both in the movements of the muscles which bend the body to the left (left-sided trunk muscles), and also those which keep it from falling in that direction (right-sided trunk muscles); both these movements

[1] The movements of the tongue following stimulation of the twelfth nucleus, root and nerve, are the subject of an interesting study by Mussen, *Brain*, 1909, p. 206.

[2] *Lancet*, 1905, i, 476.

[3] *Quart. Jour. M.*, 1907–1908, i, 448.

[4] *Brit. M. Jour.*, 1909, I.

Beevor considered as left-sided movements. These same muscles acted strongly in right-sided movements, *i. e.*, the patient could bend himself strongly to the right (right trunk muscles) and could from that position bring himself with normal power to the upright position (left trunk muscles). If the patient moved his body from the extreme left against the force of gravity to the vertical position and then to the extreme right against resistance, the same muscles were in action throughout the movement.

All movements of both limbs are abolished at first in a severe hemiplegia, but as the patient begins to recover, or from the first, in slight cases, the tendency for the arm to be affected more than the leg is apparent, and movements return first in the lower limbs. If the lesion be sharply localized well back in the internal capsule or involve the leg centres in the cortex, the paralysis is, of course, most marked in the leg; but the above statement is true for the great majority of hemiplegias, and it is a very uncommon case in which there is no return of motion in the leg, and very generally there is also some return of movement in the arm.

As the movements begin to return there is evidence of a certain selection. This has been the subject of a number of investigations, and here again we find that it is rarely individual muscles that are paralyzed in hemiplegia, but movements which require the action of many muscles. Of these muscular mechanisms there are some which are more apt to remain paralyzed and others which show a tendency to recover. In the leg the groups that are most affected can, in a general way, be said to be those which advance the leg during walking, *i. e.*, the flexors, abductors, and external rotators of the hip and the flexors of the knee and the dorsal flexors of the ankle; whereas the opposite group of muscles is little affected and recovers first. In the arm the condition seems to be more complicated. The movement which opposes the thumb to the little finger is most apt to remain paralyzed. Opening of the hand and outward rotation of the forearm are more affected than closing the hand and inward rotation. Extension of the elbow is more affected than flexion. The movements forward and backward of the upper arm and all the movements of the shoulder girdle are usually lost. The disability is not always proportionate to the return of muscular strength, and at times one finds an arm in which there is little or no decrease in the force of the individual movements, but which is practically useless for all its finer activities.

It is apparent that the varying degrees of paralysis following a cerebral lesion cannot be entirely explained by Broadbent's simple hypothesis of the close association of the lower centres for bilaterally acting muscles. The condition is much more complicated; the association may be between the cerebral centres themselves, as seems to be the case for the trunk muscles, which, although unilaterally represented in the cortex, as shown by stimulation experiments, act symmetrically and are little paralyzed from a unilateral lesion. Förster[1] believes that the selective paralysis of the limbs is due to an innervation of the retained muscles by auxiliary fibers which arise from the homolateral motor cortex and run as direct pyramidal fibers both in the anterior and lateral tracts of the cord. A full explanation must wait for a more complete knowledge of the physiology of motion.

The movements on the non-paralyzed side are usually somewhat affected,

[1] *Deutsche Ztschr. f. Nervenh.*, 1909, xxxvii, 349.

*i. e.*, their force is reduced, as shown by dynamometer tests. Brown-Séquard first called attention to this in 1882, and Sternberg[1] has lately reviewed the question. Often the reduction in the power is not great, but at times it is marked. The force with which a movement can be executed on the paralyzed side is generally increased, if at the same time the patient makes the corresponding movement on the opposite side. The effect of simultaneous movements on the normal limbs varies, but it is never very great.

Allied to this reinforcement during simultaneous movements of the two sides are the *associated movements* which are often seen in cases of hemiplegia. When a strong effort is made with the non-paralyzed side it is very usual to see some involuntary movement of the corresponding paralyzed limb. During efforts to make a definite localized movement on the paralyzed side, as closing the hand, flexing the ankle, etc., the whole limb may be brought into play, and, indeed, the other limb on the same side may be moved. It is also not very infrequent to see associated symmetrical movements of the opposite non-paralyzed side. These phenomena are particularly well seen in infantile hemiplegia. Strümpell pointed out that if a hemiplegic, or, indeed, any patient in whom the pyramidal tract was affected, be placed in a sitting position and asked to flex the paralyzed hip while the observer resisted the effort by making downward pressure on the knee, there will be a strong involuntary action of the tibialis anticus muscle, even though this muscle may be paralyzed to voluntary effort (Strümpell's tibialis phenomenon). This is a good example of an associated movement, and illustrates the inability of a hemiplegic to make isolated movements. During certain involuntary or reflex movements the paralyzed limbs may be moved in a surprising manner; thus, during yawning the arm may be forcibly raised and drawn across the chest.

Many explanations have been advanced to account for these associated movements. They are obviously due to a spread of the impulse beyond the centres which control the intended movements. This is believed to be due either to a hyperexcitability of the lower centres or to an increase in the stimulus, *i. e.*, voluntary effort, the direction of the spread depending upon associated paths between these centres. These connections may be the remains of primary paths laid down in the early evolutional history at a time when all movements were bilateral, and which have persisted to a greater or less extent during development.

In newborn children the tendency to associated movements is very great, and becomes less as the facility to make coördinated movements is acquired. Later in life, when new, highly specialized movements are being learned, writing, dancing, bicycle riding, etc., at first many more movements are brought into play than are required for the action. During practice the unnecessary movements are inhibited, the necessary ones are emphasized, and finally, when the movements are perfectly learned, only necessary muscles are moved, and only with the required amount of energy. The processes underlying this must be very intricate, but an essential factor is the cerebral control, acting, for the most part, through the pyramidal tracts, and an interruption of one of these tracts, as in apoplexy, causes a loss of the finely coördinated movements, and a reversion to a more

[1] *Deutsche Ztschr. f. Nervenh.*, 1908, xxxiv, 128.

fundamental type, as shown by the associated movements. The importance of the sensory factor is obvious.

The *muscular tension* in the paralyzed limbs is, in the great majority of cases, at first very much reduced, but if the lesion is situated in or near the cortex, and is irritative, the limbs may at once become rigid, or be the seat of recurrent convulsions. This is the early rigidity to which Todd called special attention. These irritative phenomena usually pass off in a few days, but are at times said to persist and to pass over into the state of late rigidity with permanent contractures.

In practically every case of hemiplegia in which there is a certain degree of recovery a condition of muscular rigidity develops in the paralyzed limbs (late rigidity of Todd). This begins when there is some evidence of returning voluntary power, and at a time when the deep reflexes have already become exaggerated. The degree of contracture is, however, not exactly proportionate to either of these factors. It is most marked in hemiplegias in which the return of voluntary power has been but slight. The limbs become rigid and tend to assume definite positions. The arm is adducted, flexed at the elbow, the thumb and fingers being flexed into the palm. The leg is straightened, *i. e.*, the contracture is most marked in the extensors of the hip and knee and in the plantar flexors of the hip. The opposite condition, the arm in extension and the leg in flexion, is very exceptional. The muscles most contracted are, in a general way, those in which the recovery of voluntary power has been greatest, and this relative overstimulation of certain muscles has been given as a simple explanation of the contracture. The condition, however, is far more complicated, and various theories have been advanced to account for it. Here, as in associated movements, we have the action of a portion of the central nervous system from which an important controlling influence has been taken.

In the *contractures* of hemiplegia there is a condition of increased muscular tension, *i. e.*, of heightened muscular tonus. Muscular tone depends on many things, but peripheral sensory impressions are necessary, *i. e.*, it is essentially a reflex phenomenon. That the cerebral hemisphere has a modifying influence on these centres is well established, and the remarkable effect upon the muscular tension of the withdrawal of these influences is beautifully illustrated by Sherrington's "Decerebrate Rigidity." In animals whose cerebrum is cut away the muscles show a remarkable state of hypertension, being maintained in a flexed position against gravity. If, however, in such an animal the posterior roots which contain the sensory fibers from a leg be cut, the limb becomes perfectly flaccid. This seems quite analogous to the fact that contracture does not occur in a hemiplegic with tabetic involvement of the posterior roots (Lewandowsky), and gives a physiological basis for Förster's procedure of cutting the posterior roots as a treatment of the hemiplegic contractures.

The positions that the limbs assume in the hemiplegic with late rigidity appear to depend upon the positions in which they are maintained, and can mechanically, by the application of splints, be altered. The attitude of the leg in bed, as determined by gravity, etc., is an important factor in its extensor contracture. When the origin and insertion of a muscle are, either actively or passively, brought closer together or separated, there is normally a reflex tendency to maintain the muscle in that position (fixa-

tion reflex). Sherrington[1] has studied this plastic tonus in animals during decerebrate rigidity and under other conditions, and gives evidence to show that it depends upon definite reflex phenomenon. This tendency is much exaggerated when the cerebral control has been withdrawn, so that when a hemiplegic contracts a muscle there is a marked tendency for the contraction to be maintained, and this explains why the contracted muscles are, as a rule, those muscles in which voluntary power has to some extent returned. At first the contracture is entirely active and subsides during sleep and under an anæsthetic, but after it has been maintained for a certain length of time actual anatomical changes occur in the muscles, so that they become permanently shortened and the contracture becomes fixed.

The *walk* of a hemiplegic patient is very characteristic, and is conditioned by the loss of voluntary power and the occurrence of contractures. The paralyzed leg is held rigidly in extension and usually rotated inward. As it is advanced the weight of the body is thrown over the sound limb and the paralyzed leg is swung forward in a curve, the toe often scraping the ground, the movement depending largely upon the muscles of the trunk. With every such movement there may be associated movements in the contracted arm.

*Exaggeration* of the *deep reflexes* is an early sign of the abnormal condition of irritability of the lower subcortical centres which follows interruption of the pyramidal fibers, and may occur soon after the apoplectic stroke. It is a constant accompaniment of the later hemiplegic state. On the paralyzed side the knee- and ankle-jerks are excessive, and clonus at both places can usually be obtained. An active adductor reflex of the thigh is common, and can be elicited, not only directly, but when the blow is struck on the opposite healthy leg, and it often accompanies the knee jerk on that side—crossed adductor reflex. On the non-paralyzed side the deep reflexes are usually increased, but not to the same degree as those on the hemiplegic side. In the paralyzed arm there is an analogous exaggeration, the biceps and triceps reflexes are intense, the scapula reflex is marked, and at times a definite clonus at the wrist may be brought out. When contractures are extreme they may so mask the reflexes that it is difficult to elicit them.

The *skin reflexes* vary. Those from the conjunctiva and the abdominal wall usually remain absent. The reflexes from the skin of the leg and foot may be excessive, any irritation causing a widespread movement of the paralyzed leg. This is usually in flexion at the hip, knee, and ankle, and the marked dorsal flexion of the great toe (Babinski reflex) appears to be a part of it. This latter movement, dorsal flexion of the great toe following irritation of the sole of the foot and the other areas of the leg, Babinski reflex and its modifications, is justly regarded as a most important evidence of a lesion in the pyramidal tract, and is very generally, although not constantly, present in cases of hemiplegia.

As we have seen, many voluntary movements of the paralyzed limbs are lost, but even those which are retained are, as a rule, performed in an awkward, inexact manner. At times, when there is but little loss of actual muscular strength, the inability to perform the finer movements is very marked, and there may be almost complete loss of function with but a slight

[1] *Quart. Jour. Exper. Physiol.*, 1909, ii, 109.

decrease of muscular strength. Ataxia in its more limited sense occurs in those cases when there is marked and permanent loss of the muscle sense.

Movements of the affected limb may be accompanied by a definite tremor, which at times has the characteristics of that seen in paralysis agitans, and at times has more the volitional type, approaching the tremor of multiple sclerosis.

Not infrequently, and more especially in cases that have developed early in life, the paralyzed limbs are in more or less constant involuntary motion. These movements are at times confined to the hand and foot, and consist in a slow, worm-like twisting and bending of the fingers, associated with flexion and extension of the wrist, analogous movements occurring in the foot. Although in a typical case of *athetosis*, as this condition is called, the movements do not involve the whole limb, the muscles of which, however, are usually spastic, still not infrequently the rest of the limb may be affected by other spontaneous movements and may even at times be jerked violently about.

Very similar movements to these more violent agitations of the limb, which are at times associated with athetosis, may occur alone, and we then have a condition that has been called *posthemiplegic chorea*. Many authors make no distinction between these two conditions, and it is undoubtedly true that it is often difficult to decide in just which category a given case should be placed. Typical, uncomplicated, posthemiplegic chorea is much less frequently seen than athetosis, but when present it has marked, distinguishing characteristics. The movements are widespread, affecting the face, the body, and all the segments of the extremities. They are manifold, violent, and have a quick, trembling character. Contractures are not common.

Both these conditions occur more frequently in infantile hemiplegias, and usually in those cases in which there has been a considerable return of voluntary power. Indeed, the limbs affected may show little or no decrease in power. Various explanations have been given to account for the condition. It appears to occur most frequently when the lesion is in the neighborhood of the basilar nuclei, particularly of the optic thalamus or the subthalamic region, and the most probable explanation seems to be that it is some disturbance of the afferent paths, particularly those from the cerebellum, which run through the red nucleus,[1] and that lesions of the motor cortex and pyramidal tracts have but little to do with it.

In hemiplegia following apoplexy the nutrition of the muscles is, as a rule, not much affected, and never to the degree that is constantly present when the lesion is in the lower motor segment. When the attack has occurred in early youth there is generally a considerable retardation in the growth of the paralyzed side, and later in life the contrast between its size and that of the opposite normal side is most striking. This general wasting or retardation in the growth of a paralyzed limb is regarded by many as depending upon disuse, and is distinguished from the degenerative atrophy which depends upon a disturbance of the lower motor neurone. Others regard it as a trophic disturbance, and point in confirmation to the lack of development of the hair and breast which occasionally occurs on the paralyzed side.

[1] Gordon Holmes, *Brain*, 1904, p. 327.

A number of observers have pointed out that it is not very uncommon to see such a degree of muscular wasting occurring so quickly, following a cerebral lesion, as to suggest the degenerative type of muscular atrophy. Indeed, Steinert asserts that some degree of this type of atrophy occurs with every hemiplegia. It usually affects the muscles of the arm more than those of the leg, and reaches its maximum in a few weeks, after which it may recede, the wasted muscle regaining most of its lost volume. Associated with the atrophy he found a definite decrease in the electrical excitability of the nerves and muscles, some slowness of the response, and a tendency for the excitability to disappear after repeated stimulation (myasthenic reaction).

The bladder, the disturbance of which is so common during the apoplectic stroke, usually regains its function quickly, but in certain cases some permanent disability remains. The patient may either have great difficulty in starting the flow, even when the bladder is full and the desire strong, or, on the other hand, he may be unable to restrain the bladder from emptying itself soon after the desire is felt.

*Loss of sensation* is much less common than motor paralysis as a permanent effect of a cerebral vascular lesion. In many cases the disturbance, if present, is so slight that it escapes notice, but it must be acknowledged that far too often the examination has been too superficial to discover the slighter grades of sensory loss. We have the records of 70 cases in the Johns Hopkins Hospital in which definite sensory disturbances were noted, and of 56 cases in which it was stated that there was no such loss. It is probable that in the majority of the other cases, where the records are silent in this regard, at least rough sensory tests were made and no disturbance found. Individual observers who have made this question the subject of special inquiry, have found some sensory loss in most cases of hemiplegia. Thus, Sandberg[1] examined 31 cases in Strümpell's clinic. In only 10 of these was he unable to discover any objective sensory disturbance, and among these there were a number who complained that the impressions were not so acute from the paralyzed as from the normal side.

At times the apoplexy has been due to a focus so situated that the sensory loss is the chief, or, indeed, the only symptom of a local destruction of the brain. Such cases are, however, uncommon, and von Stauffenberg,[2] who has very lately reviewed this subject, was able to find in the literature only 8 cases of hemianæsthesia without motor paralysis which were completed by autopsy. He included lesions of all kinds. Reference has been made to the case of a woman, aged thirty-seven years, who, about ten days after the delivery of her child, had a cerebral attack which left her with a permanent sensory loss. There was at first some motor disability, which, however, subsided quickly, and two and a half years later was evidenced only by a slight grade of ataxia. The affected side was from the first very painful, and the pain was spontaneous in the sense that it occurred independently of any unusual stimulus, but was much aggravated by even the slightest irritation. Any draught, as from a fan or from an open window, caused intolerable pain. For many months she could not stand manipulation of the arm and leg, and any quality of stimulus, particularly cold,

[1] *Deutsche Ztschr. f. Nervenh.*, 1906, xxx, 149.
[2] *Arch. f. Psychiat.*, 1909, xlv, 683.

when applied to that side, was very painful. Such a degree of pain so long continued (now six years) is a very unusual symptom of a cerebral focus. Patients frequently complain of more or less discomfort on the paralyzed side. This is most apt to be in the arm and referred to the shoulder-joint, and appears to be due to immobility and the relaxed condition of the tissues about the joint. The discomfort may, however, be widespread, and at times amount to actual pain, involving the whole side.

The *sensory loss* is rarely complete, either in degree or in the involvement of all the sense qualities. The finer sense perceptions are most often affected. A patient who is conscious of every touch may be entirely unable to properly localize it or to form an idea of the character of the stimulating substance. Deep sensibility and muscular sense are usually involved, and the power of recognizing objects felt (stereognosis) is commonly lost. Sandberg found the perception of pain, heat, and cold little, if at all, disturbed, but Schaffer describes as characteristic of a cerebral lesion the inability to appreciate heat and the painful response to a cold stimulus. The perception of pain itself is, at times, affected.

A sharply localized lesion in the brain stem, as that following an occlusion of the posterior inferior cerebellar artery, frequently gives a dissociated sensory disturbance of the spinal type, absolute loss of pain and temperature, with perfect retention of touch and muscular sense.

The distribution of the sensory defect varies greatly. It may involve the whole side, but the mucous membranes and the skin of the face are usually spared, and even when most widespread the degree of loss differs, the distal portion of the limbs being more affected, and the hand being usually more involved than the leg. The anæsthesia may be entirely confined to the distal portions of the limbs or of one limb, or it may be found in more or less isolated areas of the affected side. The division between normal and affected skin areas is not sharply marked, but one passes more or less gradually into the other. It has recently been shown that the areas of sensory loss, following a cerebral lesion, may have the distribution of those seen in lesions of the spinal cord.[1] This is particularly so when the lesion is in the cortex, and Russell and Horsley[2] suggest that this depends upon a re-representation of the spinal segments in the sensory cortex. Mills and Weisenburg[3] hold quite similar views.

*Hemianopsia* of the opposite side occurs when the visual path is involved in a cerebral lesion, and is a frequent accompaniment of organic hemianæsthesia. Total blindness of one eye due to optic atrophy, secondary to occlusion of the thalamic branch of the internal carotid artery, is a symptom of thrombosis of this vessel. The blind eye is on the side of the lesion, and not of the paralysis.

*Hearing* is not much impaired by a unilateral lesion. Impressions from each ear go to both sides of the brain, although probably in greater intensity to the opposite side. One would, therefore, expect a certain degree of deafness in the opposite ear as compared with its fellow when the auditory centre or its afferent path is implicated. Such a loss has been frequently found from a lesion in the region of one of the posterior corpora quadrigemina.

[1] Goldstein, *Neurol. Centralbl.*, 1909, p. 114.
[2] *Brain*, 1906, p. 137.
[3] *Jour. Nerv. and Ment. Dis.*, 1906.

*Vasomotor and Other Sympathetic Effects.*—During the apoplexy and directly afterward the paralyzed limbs are not infrequently warmer than those of the other side, and it is said they may be swollen and œdematous. Often the patients sweat profusely, but except in lesions of the brain stem there does not seem to be any difference between the two sides. von Bechterew[1] and others have, however, described unilateral abnormalities in the secretion of sweat following cerebral lesions, and have shown experimentally that definite effects in this secretion can be obtained by stimulating the cerebral cortex. In the chronic stage the paralyzed limbs are, as a rule, cooler than their fellows.

There appears to be no doubt that the cerebrum influences sympathetic activities, but little has been proved as to the manner and extent of this influence. The most striking case seen by the writer was in an infant sixteen months old, in whom there was a most remarkable pallor and coldness of the entire left side, which had been paralyzed during an acute cerebral attack. The vasoconstrictor symptoms and the paralysis cleared up together in about six weeks.

In three cases, in which there was every reason to believe, and proved in two by autopsy, that the lesion was a softening of the lateral aspect of the medulla, following an occlusion of the posterior inferior cerebellar artery, there were marked evidences of paralysis of the sympathetic functions on the same side—slight ptosis, a pupil that dilated poorly in dim light, and an absence of sweating on that side of the body.

*Bedsores.*—In patients who are long in bed, especially when there is lack of control of the sphincters of the bladder and rectum, bedsores tend to develop. Charcot believed this to be a definite trophic disturbance, but most observers regard it as the result of pressure, lack of cleanliness, and disturbed nutrition.

**Treatment of the Hemiplegia.**—The management of a patient after he has recovered from the cerebral attack must, in the first place, be directed against the occurrence of a new attack. These measures have already been considered under the prophylactic treatment of apoplexy.

In the treatment of the hemiplegic state itself, the paralyzed limbs should be rubbed, moved passively, and the arm supported at the elbow in the endeavor to keep the shoulder from sagging, and so prevent the pain which is so often referred to this joint. The galvanic current applied directly through the joint sometimes is a benefit. Systematic massage, passive movement of the joint, and electrical stimulation of the muscles appear, at times, to be of use in reëstablishing motor power. When voluntary power begins to return this should be encouraged by practice, and the patient should have regular gymnastic exercises. When the tendency to contracture is very pronounced, one should endeavor to see that the limbs are stiffened in the most convenient positions, and fortunately this is the attitude that is most often assumed—the arm in flexion and the leg in extension. The usual methods employed are massage, passive movements, and at times the application of splints, but of late other methods have been suggested, and Criegern[2] exercises the arm after the blood has been driven from it by the use of the Esmarch bandage, the limb held above the head. This method is not so easily applicable to the leg.

[1] *Arch. f. Anat. u. Physiol.*, 1905, p. 297; *Neurol. Centralbl.*, 1907, p. 187.
[2] *Fortschr. d. Med.*, 1909, xxvii, 2.

In cases in which the contractures in the limbs have become so intense as to prohibit their use, as not infrequently occurs in the diplegias of infants, Förster[1] has devised, and Tietze has executed, a most interesting surgical procedure. This is based on the theory that the spasticity is a reflex phenomenon, and depends largely on the afferent impressions which the lower centres receive from the limbs, and that if these could be abolished the limbs would become flaccid, and if they could be decreased in amount the spasticity would be lessened. Förster, considering the overlap in the distribution of the posterior roots, each area of the skin being supplied by at least three, and each muscle by two and usually three roots, concludes that two posterior roots supplying a definite muscle group might be safely cut if they were not neighboring roots, and that in this way the spasticity might be decreased with slight, if any, sensory loss. This operation has been carried out on a number of cases with very gratifying results. The subsequent reëducational treatment is, however, a most essential part of the procedure, and must account in great measure for the results. Other more usual orthopædic measures of cutting and transplanting the tendons are sometimes useful.

[1] *Mitteil. aus d. Grenzgeb. d. Med. und Chir.*, 1909, xx, 493.

# CHAPTER X.

## TUMORS OF THE BRAIN AND MENINGES.

By HARVEY CUSHING, M.D.

"Raison n'a que voir ny chercher
Là où l'on peut du doigt toucher."
*Ambroise Paré.* (Aphorisms.)

**Introductory.**—Under the caption "Brain Tumors" it is customary to group all neoplasms which encroach upon the enclosed space destined by Nature's intent to be occupied solely by the encephalon. In this broad sense the variety of new-growths which symptomatically compromise the brain, whether by displacement from pressure or by actual destruction, is multitudinous. They include not only the tumors originating from the tissue components of the brain and its various appendages, but those arising from the meningeal envelopes and even from the cranium as well; they include metastatic growths whose tissue elements may be foreign to the brain, and also certain infectious lesions—cysts and granulomata—which elicit the familiar pressure symptoms of tumor, though, strictly speaking, they are not neoplasms.

The characteristic symptom complex of an intracranial tumor, due to the crowding of the brain into a smaller space by a foreign growth of any sort, may therefore be produced by lesions which originate from a cranial or extracranial source and implicate the brain secondarily through metastasis or invasion; although lesions of this type are numerous, for the sake of brevity they will be given only passing consideration. One must remember to distinguish clearly between the growths which primarily occur within the substance of the brain and those which arise from its appendages or meningeal coverings.[1]

**Incidence.**—Despite their supposed rarity, the brain is actually one of the most common seats of tumor growth—their seat of predilection according to Oppenheim; and Bruns affirms that when a general practitioner says he has never encountered an instance it is an acknowledgment that patients with tumor have passed through his hands unrecognized. By far the larger number are never diagnosed; and as the progress of the disease is a lamentably slow one, it is with comparative infrequency that patients thus afflicted end their days in a general hospital, so that it is unusual for them to figure largely in pathological records.

[1] From the enormous literature on the subject of brain tumor, there are a few comprehensive monographs which should be consulted: L. Bruns, *Die Geschwülste des Nervensystems*, 2te Auflage; S. Karger, Berlin, 1908; H. Oppenheim, *Die Geschwülste des Gehirns. Nothnagel's Spec. Path. u. Ther.*, Wien, 1896, Bd. ix; H. Duret, *Les Tumeurs de l'Encéphale*, Félix Alcan, Paris, 1905.

In the autopsy reports of the Johns Hopkins Hospital to January 1, 1909, among 3150 autopsies there were 55 cases of brain tumor (1.7 per cent.). This is a larger percentage than that given by Siedel, who found, in Munich, 1.25 per cent., and by v. Beck, who found, in Heidelberg, 0.8 per cent. There are, of course, many elements to be considered, such as the average age of hospital patients, the character of the service, and the interest paid to neurological cases.

Bruns states that 2 per cent. of all patients classified as neurological suffer from brain tumor, and Blackburn's figures (29 tumors in 1642 autopsies, nearly 2 per cent.) show an equal percentage for asylum patients.

In the medical wards of the Johns Hopkins Hospital, in approximately 25,000 admissions, there have been in the neighborhood of 100 cases diagnosed as brain tumor, making an average of one tumor in every 250 patients. In the surgical service the number of patients with the diagnosis of brain tumor has risen from 0.06 per cent. in the first 5000 admissions, to 0.2 per cent. in the second 5000, to 0.3 per cent. in the third 5000, to 0.75 per cent. in the fourth 5000, and to 1.3 per cent. in the last 3000. In the last 1000 surgical admissions there have been 40 tumor cases, showing the rapid increase when particular attention is given to them.

**Etiology.**—**Sex.**—There is a general opinion that tumors occur with greater frequency in the male than in the female—twice as often, according to the following figures: Bruns found, in 63 autopsy cases, 40 males and 23 females; Gowers, in 650 clinical cases, found 440 males and 210 females. Of the 130 patients with tumor or presumed tumor seen by the author during the past six years[1] there have been 87 males and only 43 females. It is to be noted that of the 22 cases occurring in the first decade of life there was a more equal percentage, namely, 13 males and 9 females.

**Age.**—More than half the cases occur between the ages of twenty and forty years. Tumors are said to be rare in persons at the extremes of life, if tubercles and congenital growths of infancy be excluded. This, however, must be taken with a certain reservation, for in our series, with 22 cases in the first decade, there has been one dermoid cyst and one tubercle, the others being mostly cystic gliomas. Gowers limits the relative immunity of infancy to the first five months. Grouping our 130 cases according to decades, there have been 22 in the first, 13 in the second, 28 in the third, 37 in the fourth, 20 in the fifth, 8 in the sixth, and 2 in the seventh decade.

**Trauma.**—The relation between trauma and the first appearance of symptoms is a coincidence that occurs too often to be ignored. Injury is an undoubted cause of aneurism and of the so-called traumatic arachnoidal cysts. It is possible that blows on the head may serve only to bring a lesion into symptomatic prominence; for, in consequence of the blow, the rupture of a bloodvessel may occur in a vascular glioma or concussion œdema may accentuate symptoms which were previously vague. Especially in the case of a granuloma the seat of trauma may even indicate the situation of the lesion; but here we are dealing with a simple infection at a spot of lessened

[1] As an individual series of cases is more valuable, from a statistical point of view, than a collaboration of the figures from various sources, I consequently will refer from time to time to the series of 130 personal cases. Of these 130 cases, the diagnosis has been certified in 68, either by operation or post mortem. Most of the remainder are alive after palliative operations.

tissue resistance. Indeed, the latent tumor itself, through an epileptic attack or faint, may have been the cause of the fall producing the cranial injury. Thus trauma may not actually occasion, but may only serve to bring into the open, the symptoms of a previously obscure growth. This is a debated question, and naturally one of the arguments advanced in its favor is the greater frequence of tumors in the adult male, whose occupations and sports engender blows on the head, whereas in infants the two sexes are equally susceptible.

**Pathology.**—The comments on the frequency of brain tumors indicate how misleading collective evidence may be; and this applies even more to the past statistics relative to their histological nature. Few pathologists have had either opportunity or inclination to make a comprehensive study of a large series of cases, for a complete postmortem investigation entails three more or less distinct factors: (1) The gross relations and physical character of the tumor in so far as it disturbs the brain in general as well as its circulation; (2) the histological nature of the lesion itself; and (3) the secondary degenerations. The first requires the careful fixation of the tissues *in situ* by carotid injections—a precaution which is rarely taken. The second necessitates absolutely fresh tissues, for the postmortem changes which occur rapidly in the central nervous system militate against the perfect fixation essential for fibrillar stains. The third object of study—the degenerations—can only be of contributory value in exceptional cases when lesions are small, uncomplicated, situated in such areas as to have involved paths whose degenerations are profitable for study, and finally when the pathological findings can be correlated with an exhaustive clinical record. One or another of these elements is usually lacking.

Mallory's well-known studies[1] have shown that it may be impossible to make positive statements in regard to the cellular structure of many tumors, particularly of those arising in the nervous system, without operative removal and immediate tissue fixation. This goes to show with what caution one must accept earlier statistics on the frequency of glioma and sarcoma. A matter of equal importance has been emphasized by Adolph Meyer and Charles J. Lambert, namely, the desirability of a histological study of the entire growth *in situ*. This, a routine measure at the Pathological Institute at Ward's Island, has been most fruitful of results. The section of an entire growth will oftentimes betray its nature, owing to a certain characteristic arrangement of the cells which cannot be appreciated by the survey of a small fragment of tissue. This applies particularly to the recognition of certain gliomata.

The histological diagnosis in this series has been authenticated both by Dr. Mallory and by Dr. Lambert, so that with the exception of one or two tumors of very obscure nature we may speak of them with some certainty. The tissues have been available either through operation or autopsy in 69 cases. Of these, 44, or 66.6 per cent., proved to be *gliomata* or gliomatous cysts (11 cases, in 3 of which a demonstrable shell of tumor was still present), or gliopsammomata (3 cases). Many of these in the past would have been classed with sarcomata, of which there has been but a single instance (metastatic) if we exclude the 5 endotheliomas. There have been 4 tubercles and 3 syphilomas: the rest of the field is scattering. In Allen Starr's much

[1] *Jour. of Med. Research*, 1905, viii, N. S., p. 113.

quoted table,[1] based on 600 cases gathered from various sources, at a time when histological differentiation was impossible, there were 91 gliomata, only 15 per cent.; whereas there were 120 cases, or 20 per cent., tabulated as sarcoma. Hence the latter lesion has come to be regarded as by far the commonest tumor in the adult.

**Gliomata.**—These were first shown by Virchow to represent specific growths of the neuroglial tissue framework of the central nervous system; and since the discovery of a specific stain for neuroglial and ependymal cells, Mallory[2] and Stroebe have demonstrated that they may arise from the ependyma as well. A glioma consequently may arise in remote situations, as the coccygeal region, from vestiges of the primitive neural canal. Christian has shown also that ependymal epithelium and neuroglia are common constituents of teratoid tumors of ovarian origin. Thus, a glioma is a growth of ectodermal origin, whereas a sarcoma arises invariably from the mesenchymal tissues. A true combination of these forms of tumor is rare, although the difficulty of distinguishing them without special stains and close study has led to the frequent misuse of the term "gliosarcoma."

Cells are found in gliomata showing all gradations from the undifferentiated embryonal type to the characteristic neuroglial spider cells on the one hand and toward ganglion cells on the other. These growths doubtless have an embryonic origin, and thus a single cellular *anlage* suffices to explain all the types of cells which are found—neuroglial, ependymal, or ganglionic. The growth may be made up almost entirely of round, undifferentiated cells, possessing no fibrillæ, so that only by some characteristic arrangement of these cells, or by the finding of a small group which has begun to assume a more adult type, can such a tumor be distinguished from certain forms of sarcoma—the gliosarcoma of the past. Lambert is of the opinion that there is a fairly uniform tendency for the cells to arrange themselves more or less radially around a central core, which, on cross-section shows a characteristic rosette-shaped figure—the satisfactory disclosure of which may require a section through the entire growth. As the cells in this radial distribution become differentiated, those of the ependymal type remain near the core, as in the normal development of the nervous system, with the types more comparable to the stellate cells of adult neuroglia farther removed.

The variable differentiation of the primitive cells into those of a ganglionic type or into ependymal cells has led to the terms *neuroglioma ganglionare*, *ependymal glioma*, etc. The *neuroglioma* of Klebs, in which the nerve fibers themselves participate in the new-growth, becomes a somewhat doubtful lesion in view of the known persistence of normal preëxisting fibers in some of these tumors.

A glioma is usually a solitary tumor which may occur in any part of the cerebrum, cerebellum, or brain stem. They probably do not form metastases, although regionary extensions which resemble them may occur. They may be of cortical or basal origin, or may arise, although less frequently, from the ependyma. Gliomata supposedly originating in the corona radiata are common and may attain a large size. The *ependymal gliomata*, first described by Virchow, which arise from the subependymal layer of the ven-

[1] *Brain Surgery*, Wm. Wood & Co., N. Y., 1893, p. 202.
[2] *Jour. of Med. Research*, 1902, iii, N. S., p. 1.

tricles, possess unusual characteristics. Ten out of the twelve published cases, according to Martens and Seiffer,[1] have originated, as did their own, in the fourth ventricle, which, therefore, seems to be their seat of predilection. The striking feature of these growths is their multiple nature, for they have a tendency to spread—to sow themselves as it were—so that small, metastatic nodules may be found throughout the ventricles, resulting, in rare cases, in an extensive diffuse growth—*ependymgliomatosis*. Their symptomatology is obscure and they seem never to have been diagnosed clinically.[2]

In color, gliomata resemble the grayish red of cortical tissue, and their consistence is not unlike the normal brain, so that in the absence of pigmentation from hemorrhages, from cystic or other structural metamorphoses, it may be difficult to distinguish them from the surrounding tissues when exposed during an operation or even at autopsy. This is particularly true of certain forms of *diffuse gliomatosis*, often mistaken (Gowers) for *hypertrophies* of the brain and requiring a microscopic examination for differentiation. Some of the largest recorded intracranial tumors have been gliomata; in the case reported by Hochhaus the entire right hemisphere was involved. A gliopsammoma in our series weighed 496 grams.

Gliomata are usually described as infiltrating tumors with an indefinite boundary which shades off into the normal tissues without clear demarcation; but this is not always the case, for they may be encapsulated. By their character of growth it frequently occurs that the form of the brain remains unchanged, with complete absence of pressure symptoms, and also that normal functionating nerve fibers are found traversing them.[3] This explains the latency of symptoms of many gliomatous growths and also shows how their extirpation may greatly accentuate focal symptoms which were slight before their operative removal. The pia, furthermore, usually serves to check their extension, although this is not invariable, for we have seen a number of gliomata which have broken through into the subarachnoid spaces and spread over the surface in the manner of an infective process. This has been described by Grund and by Spiller.[4]

Gliomata are often exceedingly vascular (*gliomateleangiectodes*), and trauma, congestion, or operation with dislocation of position, may lead to hemorrhages from rupture of the thin-walled vessels. A spontaneous hemorrhage simulating apoplexy is not uncommon, and this may be the first symptom. Repeated small hemorrhages may occur, with marked fluctuation of symptoms. Degenerative processes with cyst formation (*cystoglioma*) are common, and the entire growth may seemingly become replaced by a single cyst (11 times in our 44 gliomata), although a careful search, sometimes requiring serial sections, will often reveal vestiges of the original growth on the cyst wall. Areas of calcification—*gliopsammoma* (3 in our 44 gliomas)—or even ossification, may occur.

Certain tumors of mesenchymal origin (sarcomata) can only be distinguished from some of the gliomata by stains of absolutely fresh and properly prepared tissue. Gliomata were claimed by Ströbe[5] to be distinguishable

[1] *Berl. klin. Woch.*, 1908, Bd. xlv, 1477.
[2] Bassoe, *Archives of Int. Med.*, 1908, vol. ii, p. 194.
[3] Byrnes, *Jour. of Nerv. and Ment. Dis.*, 1909, vol. xxxvi, p. 129.
[4] *Jour. of Nerv. and Ment. Dis.*, May, 1907, vol. xxxiv, p. 297.
[5] *Ziegler's Beiträge*, 1895, Band xviii, p. 405.

from sarcomata by their gross appearance and manner of growth, for the latter are encapsulated, are sharply distinguishable from the brain, and easily enucleated. This physical distinction is not to be relied upon, for a number of gliomata in our series have shown all the features, except histological ones, thought to characterize sarcoma.

**Endotheliomata.**—Next to the gliomata these are probably the most frequent of all true intracranial tumors, though they take their origin from the endothelial lining of the meninges much more often than from an endothelial source, vascular or lymphatic, within the substance of the brain itself. Hence, they are apt to be extracerebral or extracerebellar, as the case may be, rather than intracerebral or cerebellar, and in a strict sense are not brain tumors. In the statistical tables of the past they have been included with sarcomata or fibromata, for their spindle-shaped cells resemble those characterizing these growths. Presumably all of the so-called benign fibrosarcomata originating either from the cerebral or spinal meninges have been of this nature, and the diagnosis of fibroma of the brain is open to doubt. Mallory has demonstrated that fibrils do not occur in these tumors, so that they can be differentiated histologically from fibromata or sarcomata in which fibrils do occur.

These tumors are intrinsically benign, but ultimately cause pressure symptoms by slow indentation or moulding of the cerebral substance. They do not invade the cerebral tissues, but can be readily dislocated from their cavity or nest, leaving a smooth surface. They grow slowly and are often of long duration. In two of our cases the lesion had doubtless been present, giving local symptoms, for thirteen and nine years respectively before pressure disturbances of serious import, due in each to an obstructive hydrocephalus, became pronounced. Blackburn's figures[1] indicate that a considerable proportion of the asylum cases showing tumor after death have this lesion.

Endotheliomata may occur in almost any situation over the hemisphere and may attain a large size. A supposedly common seat of these tumors—possibly due to the unmistakable symptoms elicited—is the cerebellopontine recess, where 4 out of the 5 cases in our series have occurred. Occasionally bilateral tumors may be found in this situation. Owing to their presumed origin from the sheath of the acusticus and the early onset of auditory symptoms, they are often spoken of as *acoustic fibromata*. They are, however, to be distinguished from the intracranial *neurofibromata* of von Recklinghausen's disease, which are apt to be symmetrical and may be multiple, involving other nerves than the auditory. Endotheliomata are prone to undergo degenerative processes, and large gelatinous or cystic areas may be found. Of all tumors, they are the most favorable for operative removal.

**Sarcomata.**—We have seen that the so-called gliosarcomata are in all probability gliomata made up in part of undifferentiated cells; also that the fibrosarcoma of the past is, in truth, a meningeal endothelioma. Still, if one is to include all actual sarcomata which serve directly or indirectly to implicate the encephalon, they may perhaps continue to represent a form of cerebral growth which is very common, even though they no longer outnumber other tumors. They are nowise specific of the nervous system, and although they may originate there, are more often invasive or metastatic

[1] Intracranial Tumors among the Insane, Government Printing Office, Washington, 1903.

in origin. Owing to their variable source they may be intracranial or extracranial growths.

Although varying greatly in consistency, being in general of a firmer structure than most gliomata, they nevertheless are sharply distinguishable from the brain tissue in which they lie more or less definitely encapsulated and from which they are easily shelled out. There is usually a zone of softening of the surrounding brain due to vascular disturbances. This would make them favorable for surgical removal were it not counterbalanced by the frequent presence of secondary metastatic nodules and by the tendency to involve adjacent tissues.

These tumors, often of cranial origin, include the *alveolar sarcomata*, growths which, without fibrillar stains, it may be impossible to distinguish from endotheliomata or from true epithelial tumors. They are often of diploetic origin, the temporal bone constituting their favorite seat (Weisswanger). *Osteosarcomata* likewise take their origin from either the cranial vault or base and, penetrating the skull, lead to pressure symptoms. Sarcomata arising from the dura may also come to involve the bone and assume certain characteristics of an osteosarcoma. These so-called *perforating sarcomata* may arise from either surface of the dura, from diploe or either table of the skull, and as they infiltrate the normal tissues and even absorb the bone in their process of growth, they are apt in time to give external evidence of their situation. As the bony absorption takes place slowly, the growths are apt to assume an hour-glass shape, the intracranial portion of the tumor indenting, but usually not invading, the cerebral cortex, which remains protected by pia-arachnoid. When the scalp has become broken down the condition known as *fungus duræ matris* results.

The brain is occasionally involved secondary to a cranial lesion by the growths designated as *chloroma* or *myeloma*. The presence of the Bence-Jones body in the urine seems to distinguish the latter from all other malignant tumors of bone.

Sarcomata found within the substance of the brain are usually of metastatic origin and show the character of the original tumor. They may be cystic, or may undergo cystic degeneration (*cystosarcomata*)—a process which occurs possibly less often than is the case with gliomata. They also may become œdematous and the cells assume a mucoid character—*myxosarcomata*. Other degenerative processes may lead to necrosis or caseation, when they may bear a close resemblance to tubercle. Deposits of calcification may occur within them, giving rise to *psammosarcomata* or angiolithic sarcomata. Intracranial growths with psammomatous bodies, whether gliomatous or sarcomatous, can be localized by the *x*-rays. *Pigment-containing sarcomata* are said to occur in the brain only in metastatic form and the metastases, as a rule, are numerous. The single sarcoma in our series of 69 certified lesions was a large, coal-black, encapsulated melanosarcoma removed at operation from a subcortical situation in the parietal lobe. At the time there was no obvious extracerebral source of origin. The patient died a year later, and a primary melanotic growth of the ovary was found with numerous small, visceral metastases and two other nodules occupying silent areas in the brain.

Diffuse Sarcoma.—There is a particular form of diffuse sarcoma (pachymeningitis interna sarcomatosa, Orth) in which a primary sarcomatous thickening of the meninges may occur almost over the entire central nervous

system—brain, cord, and nerves often being encased by the nodular masses; it would seem as though a direct implantation of tumor cells had occurred through the medium of the cerebrospinal fluid. Occasionally a primary growth seems to have burst into one of the lateral ventricles, leading to the inoculation, as it were, of the cerebrospinal fluid with wide dissemination of the infective agency.[1] The process in less extensive form is more commonly met with about the base of the brain, often with bilateral involvement of the cerebral nerves, and not infrequently of the cauda equina as well.

**Carcinomata.**—Like the foregoing these tumors occur for the most part as metastases, being found most often as secondary nodules in cases of mammary cancer. The nodules occur usually in the substance of the brain; are frequently multiple, and show a marked tendency to cystic degeneration. Epithelial tumors occasionally arise from a cranial or extracranial source and invade the cranial chamber secondarily. We have seen such a tumor apparently of labyrinthine origin. Primary epithelial tumors, however, may occur in the brain from the secreting cells of the choroid plexus (Ziegler), and one of the tumors in our series, difficult to classify, is a large, seemingly benign epithelial and cystic growth, apparently of this origin. These tumors are regarded by some as a simple hyperplasia or *adenomatous papilloma* of the plexus. Epithelioma of the ependyma has been described, and the hypophysis is naturally a not infrequent seat of epithelial as of other growths. These are commonly malignant adenomata, an instance of which has occurred in our series.

**Cholesteatomata.**—Sometimes called "mother-of-pearl" tumors in consequence of their peculiar glistening external appearance, these remarkable growths have their seat of predilection in the basal meninges. They apparently never arise primarily in the brain substance itself, although this becomes excavated and compressed as the growth advances. There is no unanimity of opinion as to their histogenesis. Virchow, Frank, and others thought that the cells of the arachnoidal meshwork multiply and distend the pre-formed endothelial spaces, producing a sort of complex retention cyst rather than a true tumor growth. A later view, advanced by Ziegler, Bostrœm, and others, favors a congenital *anlage* of misplaced epidermoidal cells, a view supported by the fact that the cells become cornified and contain keratohyalin. Further, Bostrœm has demonstrated that a close relationship exists between these and the more usual forms of dermoid tumors. It is possible that both epithelial and endothelial forms may exist.

The otitic cholesteatoma of older writers, following chronic suppurations of the middle ear, apparently consists of epidermoidal masses which cause pressure absorption of the temporal bone and invade the cranial chamber secondarily. These tumors usually possess a more or less irregular flat form, modified as the growth pushes its way into the clefts and recesses between the bone and base of the brain. The surface is often covered with warty irregularities. On section these tumors consist of a grayish, friable, more or less laminated mass, made up of layers of a closely packed mosaic of flat, polygonal cells—really a dead tissue containing no bloodvessels. They are of slow growth and may run a symptomless course, being oftentimes an unexpected postmortem finding. They may, on the other hand, give definite

[1] Stanley Barnes, *Brain*, 1905, xxviii, 30.

general and focal symptoms of a basilar tumor with cerebellar, cerebellopontine, or trigeminal symptoms.

**Dermoid Tumors.**—These are among the rarest of intracranial growths. There is a single instance in our series, the cyst, containing hair, having been successfully extirpated. Bostrœm considers that various grades are recognizable, from a simple epithelial-lined sac, which he would regard as a cholesteatoma, up to the most complex embryoma which may contain examples of many tissues of the body. These tumors date from the third to the fifth week of embryonic life. They are usually basal, commonly mesial, grow slowly, and are not likely to produce symptoms. Another variety is connected with the formation of dura and bone, and it is not uncommon for them to connect, through a cranial defect, by a stalk with an extracranial dermoid. These are often found in the newborn in company with other abnormalities.

There are other lesions, possibly no less rare than the foregoing, which deserve passing mention in order to complete the enumeration.

**Teratomata.**—Teratomata have been found, chiefly in the neighborhood of the interpeduncular space, arising doubtless from some embryonal vestige with faulty development of the pituitary gland. They are apt to be accompanied by symptoms of hypopituitarism and by the characteristic local symptoms of tumor in this situation, judging from the few reported cases.[1]

**Lipomata.**—Lipomata are rare also, and those described have usually been small, basal growths. A larger form is known which has especial predilection for the upper surface of the corpus callosum.

**Fibromata.**—Fibromata are occasionally found in fairly pure form, arising from the periosteum of the cranial base or from the nerve sheaths—the latter being more properly classified as a manifestation of von Recklinghausen's disease.

**Neuromata.**—The same is true of the so-called neuromata, which arise from the perineurium of the cerebral nerves and consequently are extracerebral tumors. They usually surround the entire nerve, giving it a spindle-shaped form, and they may undergo sarcomatous or myxomatous change. They are particularly common on the acusticus, but as they are frequently multiple other nerves may be involved (multiple neurofibromatosis).

**Enchondromata.**—Enchondromata have been found arising from the choroid plexus or meninges. One especial variety, arising from the cartilaginous vestige of the *Clivus Blumenbachii* (Virchow), has its seat of predilection near the pituitary fossa.

**Angiomata.**—Angiomata, whether simple, telangiectatic, or cavernous, are congenital and usually cortical. As a rule, they produce no serious symptoms, barring the possible accident of hemorrhage or thrombosis. Typical pressure symptoms were elicited in one of our cases by a large, cavernous angioma of the temporal region. The *angioma racemosum* may occur alone or in combination with congenital vascular lesions of the scalp or face (Kalischer; Emanuel; Cushing).

**Psammoma.**—Psammoma is a term given to a number of tumors, glioma, sarcoma, and endothelioma, in which areas of calcification have taken place, making them gritty on section. Bruns is inclined to restrict the term to

[1] H. Cushing, *Jour. of Nerv. and Ment. Dis.*, 1906, xxxiii, 704; D'Orsey Hecht, *Jour. of the Amer. Med. Assoc.*, 1909, liii.

certain growths which arise in the pineal gland or choroid plexus, where, under normal circumstances, calcareous bodies—the so-called "brain sand"—occur.

**Cysts.**—We meet with (1) simple serous cysts which presumably are degenerated tumors; (2) tumors containing cysts, as cystosarcoma, cystoglioma, etc., possibly on the way to the above; (3) blood cysts, the result of cerebral or cerebellar apoplexy; (4) cysts the result of softening after embolism; (5) the cystic widenings or diverticula of the fourth ventricle; (6) dermoid cysts; (7) parasitic cysts; and (8) gas cysts.

It is probable that, in this part of the world at least, the greater number of so-called *simple cysts* are actually degenerated tumors (gliomata and sarcomata), although it may be difficult to demonstrate conclusively this association. Since surgical explorations have become frequent, it has often been found that tumors originally solid have become cystic at a later exploration or at autopsy; and one occasionally sees a tumor in what seems to be a process of cyst formation with a large area of semifluid, degenerated tissue.

Unlike those which may form as the result of absorption of an apoplectic extravasation, and also unlike the porencephalic cysts of childhood, these degenerative cysts may continue to give pressure symptoms almost as severe as those of the original growth. On evacuation they tend to refill. Their fluid content is often of a yellowish or brownish tinge, and the fluid, rich in fibrin, clots on exposure to the air. Their seat of predilection is the cerebellum. Scholz[1] has made an elaborate study of 75 published cases. There have been 11 cysts in our series, 7 of them cerebellar; and we have seen a number of borderline conditions in which a glioma seemed to be in the process of total cystic degeneration after a decompressive operation.

There are certain obscure conditions, often seen in association with an actual tumor growth, in which a large cyst of the hemisphere has an apparent communication with a lateral ventricle.

*Arachnoidal Cysts.*—Arachnoidal cysts, a consequence of trauma, may give pressure symptoms and focal signs simulating tumor. They may follow lesions which leave a defect in bone and dura over a given area, for an accumulation of the fluid which exudes through the arachnoidal membrane may become pocketed under tension, giving focal pressure symptoms.

*Parasitic Cysts.*—The *Cysticercus cellulosæ* shows an especial predilection for the central nervous system. Thus in 87 cases in Berlin the cysts in 72 instances were cerebral (Bruns). The hooked embryos attach themselves to the meninges, cortex, or ventricular wall—often of the fourth ventricle—where the larva develops as an isolated bladder-like structure, rarely exceeding a centimeter in diameter. The cysts are apt to be multiple, 111 of them having been counted by Delore and Bonhomme in a single patient. Under these circumstances, it is probable that auto-infection has occurred, a ripe segment of the parent tænia having entered the stomach from the intestine. A particular form sometimes occurs in the basal meninges, the larvæ assuming a branching form—*Cysticercus racemosus*—and a condition of so-called "cysticercus meningitis" may result. Ventricular cysts usually lead to a great increase of fluid and a distinct ependymitis may result (Lloyd).

The larvæ may be long-lived, although it is usual for them to shrink and become calcified in from three to six years. An especial study has been

[1] *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1906, xvi, 745.

made by Stern[1] of the 68 known cases of fourth ventricle involvement. The recent presence of an adult tænia solium in the intestine should arouse suspicion of the nature of the case.

*Echinococcus or Hydatid Cysts.*—Echinococcus or hydatid cysts seem to have no especial predilection for the central nervous system. The cysts are usually single and more apt to occur in the brain substance than in the meninges or ventricles. They may reach a large size, and show an especial tendency to absorb the overlying skull and thus to appear under the scalp. Suppuration may occur, or the worm may die and the contents containing the free hooklets become gelatinous and thick. The symptoms are those of a slowly growing tumor; and on exposure a differential diagnosis from other forms of cyst can at times be assured only by the demonstration of head or hooklets, or by the peculiar structure of the cyst wall.

**Infectious Granulomata.**—Formations resembling tumors, not only in their gross appearance but in the symptom complex which they elicit, are the frequent result of tuberculous or syphilitic, and occasionally of actinomycotic, processes. Although a recognized infection, and hence not neoplasms in the same sense as some of the foregoing, custom nevertheless calls for their consideration with tumors.

Encephalic Tuberculomata.—Of encephalic tuberculomata two forms are recognized: (1) Focal *tuberculous meningo-encephalitis*, and (2) the so-called *solitary tubercle*. Tuberculosis of the central nervous system is usually—perhaps always—a complication of tuberculosis elsewhere.

*Focal Tuberculous Meningo-encephalitis.*—This form consists of a local plaque of disease made up of a conglomeration of more or less caseated tubercles, usually situated on the surface of one of the cerebral hemispheres, less often a cerebellar or a basilar process. Although of meningeal origin, more or less involvement of cortical tissue is inevitable, and with the advance of the process the characteristic focal symptoms of tumor may occur, although general pressure phenomena are usually absent.

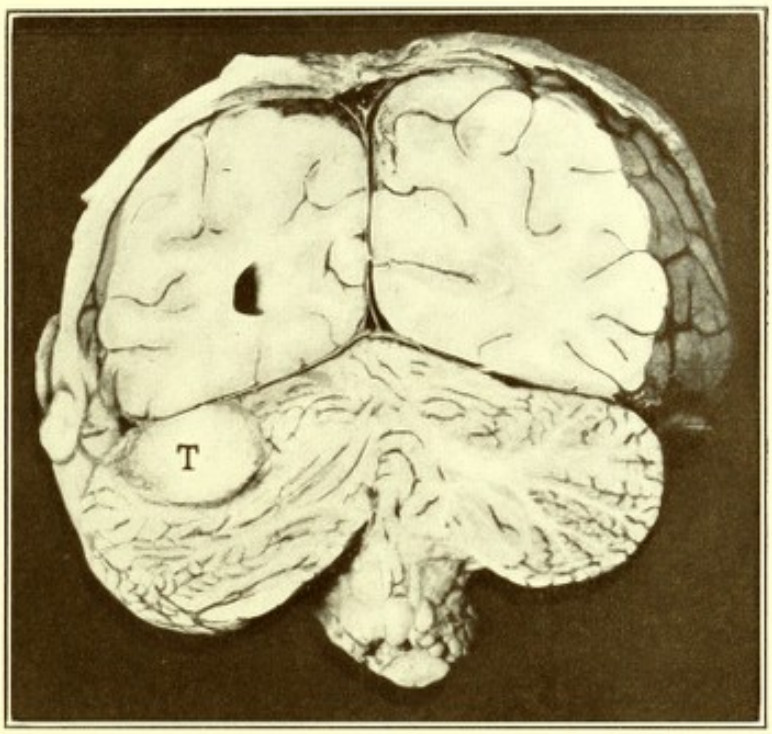
*Solitary Tubercle.*—The solitary tubercle behaves, on the other hand, in all respects like a brain tumor. According to Starr's tables, this should be the most common form of growth (32 per cent. if all ages are included), representing in the first two decades 50 per cent. of all cases, falling to 14 per cent. in adults. Our experience has been otherwise, for out of the 68 certified lesions, only 3 have been tubercles, 2 in the third and 1 in the first decade, although it must be acknowledged that a number of children in the 130 cases of presumed tubercle are alive after simple decompressions, without which these figures might have been larger. A tubercle has been found at the early age of twenty-three days in a case described by Demme.

The term "solitary" is somewhat misleading, for these individual growths are composed of many fused tubercles. "Solitary tuberculoma" is a better term, or "conglomerate tubercle," as Schmaus has suggested. The lesions are multiple, at least at an early stage, in about 50 per cent. of all cases (Bruns), becoming fused later on into an irregular conglomerate mass. This is the more likely since the originally discrete foci, in evidence of their embolic origin, are apt to be distributed on a single branch of the arterial tree.

Their form is roughly spherical, unless modified by fusion or other especial

[1] *Zeit. f. klin. Med.*, 1907, lxi, 64.

# PLATE XIX




Section of the Brain of a Patient Who Died from the Effects of a Lumbar Puncture.

Note the mould of the cerebellar tissues herniated through the foramen magnum. A tuberculoma is shown at T; there were several others scattered throughout the brain.

influence. Their favorite seat seems to be in the cerebellum or brain stem (Plate XIX). The individual lesions vary greatly in size, from a millet seed to a hen's egg on the average, although in rare cases by fusion of many isolated foci almost one entire hemisphere may become transformed into a tuberculous mass.

Coagulation necrosis with caseation takes place as the lesions enlarge, and on section the mass presents a dry, yellowish, crumbling centre, with a grayish-red, peripheral growing zone where are located viable bacilli and actively growing tubercles. As in tuberculous processes elsewhere central liquefaction with abscess formation may occur; less often the process dies out and calcification occurs. When the lesions are active the surrounding encephalic tissues are usually œdematous, and this tends to increase the pressure effects and constitutes a mechanical difference between a tuberculous and a sarcomatous tumor; for the latter, as a rule, only affects the immediately adjoining zone by pressure softening.

Syphilitic Processes.—Like the above, syphilitic processes giving a tumor symptom complex occur in the central nervous system in two especial forms: (1) As a local spreading *meningo-encephalitis*, and (2) as a solitary granuloma—the so-called *gumma* or *syphiloma*. To these may be added the basilar *meningitis gummosa*, a process often arising in the middle cranial fossa where, with notable fluctuation of symptoms, it implicates one after another of the nerves which pass through the region. Vascular lesions are common, and sudden hemiplegia may occur from occlusion of the middle cerebral artery. This basilar process is apt to spread over the sylvian region, and, as the cortex usually is more or less involved, it hardly deserves to be distinguished from the more common meningo-encephalitis syphilitica of the convexity, except for its peculiar local manifestations.

Bruns, in agreement with Oppenheim and Gowers, regards syphilis as the most frequent of the conditions producing the tumor symptom complex in adults. This opinion is based naturally on the subsidence of symptoms after antisyphilitic therapy—not an infallible test—and is contrary to postmortem statistics. It is doubtless on the same clinical basis that the cerebral cortex and the paracentral convolutions in particular are regarded as the favorite seat for gumma, owing to the definitely localizing symptoms of the lesion when in this situation. On the other hand, the well-recognized association of these lesions with trauma is possibly a sufficient explanation for their frequent occurrence under the exposed vault.

*Syphiloma.*—Unlike tubercle, syphilitic brain lesions are largely confined to adult life, and are said to occur above the tentorium more often than below. In the majority of cases, furthermore, they are surface lesions, whereas tubercles are more common in the substance of the brain. For the most part, the solitary lesion does not reach a large size, and never equals the enormous, fused tuberculoma. Somewhat like an early tubercle, however, the gumma consists of a vascular, grayish-red, granulation tissue mass of round, epithelial, and giant cells. In this tissue mass changes soon occur; through absorption or degeneration areas of coagulation necrosis appear, or through fibrosis a dense, scar-like tumor may be left—the typical syphiloma. This change is particularly common in the meningeal gummata.

In an advanced stage of the process, cortex, meninges, and even overlying bone may become adherent in a dense cicatrix which resists absorption under

the most vigorous antiluetic measures. Such a granuloma rarely undergoes the process of softening characteristic of the tuberculous lesions, although, on the other hand, disturbances of the surrounding cerebral tissue through accompanying vascular disease are much more common. Nevertheless, it is often difficult to distinguish the processes pathologically without microscopic demonstration of the agent—bacillus or spirochæte. One histological difference lies in the presence throughout the gumma of the bands of tissue undergoing fibrous change. We have occasionally experienced difficulty in distinguishing between a cortical gumma with spreading meningo-encephalitis and a superficially placed glioma which has invaded the pia-arachnoid. In all cases the serum reaction should be tried.

ACTINOMYCOMATA.—Actinomycomata are rare and have usually been secondary or metastatic lesions, although Bollinger regarded his cases, described in 1887, as primary. Howard has recently made a thorough study of these conditions.[1]

**Pathological Physiology and Symptoms.**—The clinical aspects of a brain tumor hinge far more upon matters relating to the physiology of the intracranial space than upon its mere structural features. Indeed, in view of their extraordinary variability in size, form, consistence, manner of invasion, rapidity of growth and situation, not only in so far as the latter relates to the various nerve tracts and centres, but also to the blood supply and channels of outlet for the cerebrospinal fluid, it is perhaps remarkable that any recognizable symptom complex can be accepted as in the main characteristic of brain tumors.

There are, however, in the majority of cases, as was first emphasized by Griesinger, two processes whose phenomena may be regarded as typical of the greater number of intracranial new-growths. The first embraces the symptoms produced within an intact skull by a more or less generalized increase of tension which affects the brain chiefly by upsetting its normal vascular and cerebrospinal fluid circulations—the so-called underlying or *general symptoms;* and the second, the symptoms produced by irritation or destruction of nerve tracts or centres of known function—the so-called localizing or *focal symptoms.*

When both of these processes are in full operation—a thing which is to be expected eventually in most cases—the presence of the lesion, and probably its situation also, is readily recognized. Unfortunately, at the stage when the diagnosis may be of greatest value, one or both of these elements are wanting, and owing to the fragmentary sketch instead of the finished picture, the condition may long fail of recognition even by an expert. For example, the general pressure symptoms might naturally be expected to bear some relation to the size of the growth, and yet a small tumor compressing the aqueduct of Sylvius so as to obstruct the ventricular outflow may lead to symptoms out of all proportion to its size. On the other hand, a large infiltrating glioma may be present without eliciting general or focal symptoms; for it may invade the normal tissues in such a way as not to increase tension and it may be traversed by persisting fibers which can transmit impulses. Again, a small tumor too insignificant to produce pressure phenomena may, nevertheless, lead to definite focal symptoms, shown, for example, by irritative movements

[1] W. T. Howard, *Jour. of Med. Research,* 1903, iv, 301.

if it happen to be situated near the precentral gyrus; whereas a similar lesion over a silent area need give no indications whatsoever of its presence. Thus, the factor of situation may be far more vital than that of size.

It can be easily seen that tumors which happen to arise in a so-called silent area of the brain may remain long dormant, and that the first symptoms may be those of general pressure when, in the course of time, the lesion has considerably augmented in size. Thus patients may present themselves, with (1) tumors which give absolutely no recognizable evidence of their existence and are found post mortem; (2) tumors which present focal symptoms alone with no evidences of a general increase in pressure; (3) tumors which give general symptoms alone with no focal manifestations, when they happen to occupy a silent area; (4) tumors which give typical symptoms of general pressure together with definite focal symptoms; and (5) the symptom complex (general and local) of tumor, which in the absence of a growth is brought about most often by œdema from one source or another.

With full realization that practically all tumors at their onset fall under the first of the groups cited above, and also that many brain tumors may exist for years before their symptoms become sufficiently pronounced to attract any serious attention, we, nevertheless, may wisely retain as characteristic in general of most tumors at some stage of their progress the so-called underlying or general pressure symptoms—headache, vomiting, and choked disk—to which we would like to add the external evidences of intracranial venous stasis, dyschromatopsia, the tendency to herniate, and a few other possibly less characteristic signs of tension.

**The General Manifestations.**—That the general symptoms are primarily the consequence of pressure against the brain within the closed skull is now fairly conclusive, for they may be completely set aside by making a purposeful cranial opening, just as nature may occasionally alleviate them in the young by a general diastasis of the cranial sutures. Some conception of what is taking place within the skull is necessary for their further interpretation.

Dislocation from Pressure and Tendency to Herniate.—Physiological studies, particularly those of Leonard Hill, have shown that the brain itself is practically as incompressible as water, and consequently that any considerable encroachment by a new body on the intracranial space must greatly increase the cerebral tension through the medium of its fluid contents. One of the earliest consequences of this is an engorgement of the venous circulation, leading not only to the subjective sensation of fulness, but to certain objective signs. In its most familiar aspects this appears as a dilatation and tortuosity of the retinal vessels—the premonitory features of a choked disk. There are, however, other external evidences equally characteristic, although less often commented upon, which occur; for, as in the case of the retinal veins, those from the scalp likewise normally discharge their content in large measure by way of the intracranial sinuses. Hence, the veins of the scalp, and particularly the smaller venules of the eyelids, often become a tell-tale of the intracranial venous stasis.

The cerebrospinal fluid is normally under low tension, and if there is no obstruction to its escape by usual channels the encroaching lesion at an early stage will drive the normally small amount of fluid out of the arachnoidal spaces, so that when exposed they will, relatively speaking, be found dry. Usually, however, the fluid becomes pent up in certain areas—in the interpeduncular arachnoidal space, for example, and hence the distention

of the optic sheath; or in the ventricular cavities of the brain, and hence the obstructive hydrocephalus—a factor of great moment in accentuating, sometimes abruptly, the pressure symptoms which were previously inconspicuous. The fluid, which backs up in the spinal meninges, may be demonstrated under tension by a lumbar puncture, although its complete withdrawal from this source is dangerous, for reasons to be explained. It may occasionally escape through the nares—cerebrospinal rhinorrhœa.

Another element, in addition to the mere size of the growth itself, must be taken into consideration, namely, the tendency of the brain itself to become œdematous—a condition which may lead to a sudden and marked increase of intracranial tension. These areas of œdema are often macroscopically evident on section of the brain, and are due either to vascular stasis or to local changes in osmotic pressure from chemical alterations of the cerebral fluids. They occur most frequently in the neighborhood of the growth, although they may be found at a distance, as Collier[1] has shown, and greatly confuse the regional diagnosis. Now these pressure disturbances, whether from swelling of the brain itself or from the encroachment of an enlarging tumor, affect the tension locally more than at a distance, although all portions of the intracranial cavity feel them to a greater or less extent. The cerebral chamber is partitioned in a measure into three subdivisions, by falx and tentorium, and a tumor in one of these compartments increases pressure locally to a greater extent than in the others, unless the tension is more or less equalized through the medium of cerebrospinal fluid obstruction. As a result of the unequal tension, distortion of the partitioning membranes, particularly of the falx, is common, with more or less dislocation of the hemispheres.

The brain, being a semifluid mass capable of certain alterations in form, tends to escape—to herniate, in other words—through any opening which may preëxist or be made in the dense enveloping dura. As Wolbach[2] has shown, small, actual hernias of the cerebral substance are found over the surface of the brain, corresponding with the arachnoidal tufts—particularly along the walls of the sinuses, although by no means limited to this situation, for the cerebral surface over the temporal lobes, for example, may become dotted with these minute protrusions. There is but a single normal opening of any size in the dura; namely, at the foramen magnum, and here, particularly in case the growth originates below the tentorium, a more or less marked *foraminal hernia of the medulla* and surrounding fringe of cerebellum occurs with dislocation of these structures downward into the cone of the upper spinal canal (Plate XIX). Sudden implication of the bulbar circulation from this cause is supposed to explain the unexpected fatalities which characterize some cases of cerebellar tumor; and a lumbar puncture with the removal of the supporting column of spinal fluid is particularly apt to lead to this accident, due to the wedging of the medulla in the foraminal ring.

This tendency of the brain under pressure to herniate may sometimes by natural processes serve to relieve the general pressure symptoms, for occasionally a superficially placed tumor, especially one of dural origin, may,

[1] *Brain*, 1904, xxvii, 490.
[2] *Jour. of Med. Research*, 1908, N. S., xiv, 153.

through pressure absorption, destroy the overlying bone and allow tumor and brain to protrude under the scalp—the so-called *autotrepanation*. This is apt to occur in areas where the bone is thin, as the squamous portion of the temporals; and needless to say was an element in suggesting the deliberate therapeutic establishment of such hernias in this situation.

Changes may occur in the skull other than diastasis of the sutures and local absorption. Deepening of the Pacchionian depressions is often a marked feature accompanying the formation of Wolbach's hernias. The entire inner surface of the cranium at times becomes thinned and roughened, so that it has a sand-paper feel, and the stasis in the cranial vessels may lead to great diploetic channels—an occasional source of fatal hemorrhage in the course of a craniotomy unless the openings are promptly filled with wax.

A word must be added concerning the respiratory and blood-pressure reactions. An intracranial growth, owing possibly to its relatively slow advancement, rarely shows the same respiratory and vascular reaction that characterizes a sudden or abrupt encroachment on the intracranial space, as in apoplexy, traumatic hemorrhage, or œdema. The slow pulse, rise in systolic pressure, and rhythmic respiration accompanying these states occur in their typical form in tumor cases only when there has been a hemorrhage into the growth or sudden œdema or hydrops ventriculorum. Otherwise one rarely sees the symptoms of acute compression in these long-drawn-out conditions except as a terminal event, for the rise in blood pressure and primary respiratory failure are the common phenomena antecedent to death.

**Headache.**—We are here confronted with a problem very similar to that in reference to abdominal pain. The brain itself, like the liver, spleen, and intestines, is insensitive, as is also its immediately investing membrane, the pia-arachnoid. The cranial cavity, however, is lined with and partitioned by an outer sentient membrane—the dura—the afferent fibers of which, trigeminal and vagal, are capable of demonstration by dissection. The fact that patients after trigeminal neurectomy—an operation which produces anesthesia of the dura as well as of the face—no longer suffer from the sensation of headache on the corresponding side, would favor the view that the dura plays a part at least in these subjective discomforts; but in a number of instances in which cranial operations have been performed upon conscious patients without anæsthesia it has been found that the dura is actually insensitive to touch or incision and that a sensation of pain is elicited only when traction is made upon it. It would seem, therefore, that distortion of falx or tentorium and the consequent stretching of the membrane is actually the important factor.

A good illustration of headaches which may be particularly insufferable are those which occur in association with tumors or enlargements of the pituitary gland which distend its enclosing pocket of dura; and it has been found that these discomforts may cease as soon as the capsule of the gland has been incised, even though the growth be not removed. It is even better known that the headaches of tumor may oftentimes be completely relieved by a successful "decompressive" operation, and that even when a certain amount of discomfort persists after such a measure its severity varies with the degree of tension evident in the protruding area of brain. Indeed, many headaches not associated with tumor, such as those accompanying traumatic

œdema, nephritis, and possibly even many cases of supposed migraine, may similarly be due to pressure.[1]

Headaches are not invariably present, for certain tumors need not be associated with an increase of pressure. This is notably true of many of the lesions which occur below the tentorium, such as the familiar cerebellopontine endothelioma, which may give merely local symptoms possibly for years with no notable increase of tension until it reaches such a size that it obstructs the aqueduct of Sylvius, when a rather abrupt onset of pressure symptoms may occur.

When present, headache may be persistent or occur only at fairly definite hours of the day or over certain periods of time, to be followed by an interval of partial or total remission. The sensation may be nothing more than a feeling of general fulness, or it may be agonizing. Headaches are rarely definitely localized to any particular point except when a lesion immediately underlies and possibly has caused thinning of the adjacent cranium, when local pain with associated tenderness may be present. As a rule, patients refer them indefinitely to the frontal or occipital region or to the vertex more often than to one side or the other. Discomforts primarily referred to the occiput are suggestive of a subtentorial lesion, although too much faith cannot be placed upon this, for a posterior lesion may give frontal headaches, and, vice versa, a frontal lesion discomfort referred to the occiput.

**Vomiting.**—Whether there is a centre in the medulla for this act—possibly vagal—can only be conjectured, but the symptom is thought to be more common in lesions involving the bulb. It is inconspicuous or entirely wanting in two-thirds of the cases, even after they have reached such an advanced stage that the evidences of pressure are pronounced. The typical so-called projectile or cerebral vomiting is common only in those cases in which headaches are severe, and is rarely seen until a late stage when pressure symptoms are pronounced; it hardly deserves the prominence it has been given as a cardinal symptom. When present it is often an early morning occurrence or brought on by some sudden change in position. The act may be unassociated with nausea. Unlike the other striking symptoms of pressure, it rarely occurs alone, whereas headache, choked disk, vascular disturbance, or dyschromatopsia may exist almost as the sole pressure manifestation.

**Choked Disk.**—This, the most striking objective sign, is at the same time the only one of the general pressure symptoms which lends itself to experimental reproduction. Many terms have been employed in designation of the process and many views advanced to explain the causal agency: of the two most important, one has attributed the lesion to purely mechanical agencies,

[1] It must be admitted that many points relating to headache are not entirely explained by assigning pressure as the sole cause. Thus, lumbar puncture with the withdrawal of cerebrospinal fluid from a healthy individual—a condition that is naturally supposed to bring about a lessening of the normal tension—may occasion intracranial discomfort, and the definite reflex headaches of gastric or ocular origin are difficult to reconcile with this view. Perception of pain is unquestionably a cerebral function, and yet the brain itself (with the possible exception of the basal ganglia), so far as we know, is insensitive to the stimuli which give pain at the periphery. Thus faradization of the supposed sensory cortex in two conscious patients, although giving vague tactual impressions referred to the periphery, provoked no local sensation whatsoever. (Some Aspects of the Pathological Physiology of Intracranial Tumors, *Boston Med. and Surg. Jour.*, July, 1909, clxi, 71; also *Brain*, 1909, xxxii, 44.)

the other to toxic or inflammatory ones. Hence a terminology has arisen which is confusing; and "optic neuritis" or "papillitis," introduced by von Leber, as opposed to choked disk" (Allbutt's adaptation of the German *Stauungspapille*) and "papillœdema," are variously used by different writers to express the whole process or to indicate its different stages.[1]

Influenced by the results of recent clinical and operative experiences, as well as by newer experimental investigations, the pendulum of opinion has swung away from the toxic toward the mechanical view originally advanced by von Graefe, Manz, and Schmidt. It seems in all probability a stasis œdema, in which the forcing of the cerebrospinal fluid under pressure into the sheath of Schwalbe, a meningeal expansion which invests the optic nerves as far forward as the globes themselves, plays the chief role. The sheath becomes distended, the nerve head œdematous, venous stasis occurs, and the ophthalmoscopic picture shows at this early stage tortuosity of the retinal veins with injection of the disk and more or less haziness of its outlines—the nasal margins usually being the first to become obscured.[2]

Marcus Gunn[3] has conveniently divided this process, for which he suggests the unfortunate name "tumor papillitis," into the following stages:

*Stage* 1. The earliest ophthalmoscopic signs are increased redness of disk, loss of definition in its edges, slight prominence of its surface, and narrowing of the physiological pit.

*Stage* 2. At a rate which varies much in different cases and which seems to bear a decided relation to the degree of intracranial tension, the swelling of the papilla increases, the physiological pit disappears, and the disk edges become quite obscured; along with these signs there is now slight haziness of the surrounding retina and the retinal veins show evidence of retarded circulation.

*Stage* 3. In an advancing case the next alteration consists in further swelling of the papilla, so that it becomes more prominent and occupies a larger fundus area, the venous distention becoming very marked; fine folds not infrequently appear in the œdematous retina, particularly between the disk and macula, and there may be retinal hemorrhages.

*Stage* 4. The papilla becomes more opaque and sometimes more prominent, the hemorrhages increase in size and number, and there are inflammatory exudations on the disk and surrounding retina. At this stage vision has become impaired.

*Stage* 5. The next change consists in a gradually decreasing vascularity of the papilla, parts of its surface becoming paler than normal, while the prominence either persists or slowly subsides. At this time also we first note

[1] Excellent summaries in regard to the causation of choked disk have been given by Th. Kocher in *Nothnagel's Specielle Pathologie und Therapie*, 1901, ix, 206, and by Alfred Sänger in the *Wien. med. Woch.*, 1904, liv, No. 47, pp. 2201, 2258, 2231. *Gowers' Medical Ophthalmoscopy* remains to this day the best monograph in English on this and allied subjects. References to the more important recent papers accompany Elschnig's chapter in Flatau, Jacobsohn, and Minor's *Handbuch der pathologischen Anatomie des Nervensystems*, Berlin, 1904, ii, 1165. (Cushing and Bordley, *Jour. of the Amer. Med. Assoc.*, 1909, lii, 353.)

[2] This stage is usually miscalled by ophthalmologists an "optic neuritis" if it be recognized at all as pathological and sharply distinguished from "choked disk," the term reserved for a later stage when there is a measurable swelling of the papilla.

[3] *British Medical Journal*, 1907, ii, 1126.

a change in the branches of the central artery, in the form of diminished breadth—the state of atrophy with inevitable blindness.

There is every reason for ascribing all these stages to a single process; and there are reasons for regarding the process as mechanical. For a similar change occurs in the neuroretinal tissue in cases of cerebral œdema following trauma, and although this rarely advances beyond Marcus Gunn's Stage 2, still we may see the process advance even to the stage of atrophy in persisting cerebral œdemas such as those which occur in nephritis. Then, again, it is a frequent experience to see the choked disk of tumor subside after a modern palliative operation, even though the tumor be not removed, so that the toxic elements, if any exist, should still be at work.

Choked disk, when present, is doubtless one of the most valuable signs of tumor, but it must constantly be borne in mind that it is absent in all cases at an early period, in most cases until a late period, and that many tumors have a long life. The discussion as to the percentage of cases in which choked disk occurs seems therefore to be beside the mark, for, as the diagnosis is made earlier, doubtless it will be absent in an increasingly greater proportion; and we feel a certain pride in having successfully removed the tumor in three of our cases before any evidence even of congestion of the disk had set in. Tumors allowed, uninterrupted, to run their full course will all probably show a certain stage of choked disk before the end.

The advent of a choked disk may be long delayed and yet may progress rapidly when the intracranial conditions lead to its formation. It is not uncommon to see the lesion develop while the patient is under observation, and hence frequent examinations are requisite in all suspicious cases. In one of our patients, coincident with a hemorrhage into a glioma, a swelling of four or five diopters occurred within a few hours—the condition being comparable to the experimentally induced acute lesion in animals.[1] These acute œdemas of the neuroretinal tissues may subside after pressure has been relieved almost as rapidly as they occur: it is only in the case of a long-standing lesion that subsidence of the swelling is delayed, owing to the naturally slow absorption of the new-formed tissue which fills the physiological cup and tends to perpetuate the form of the swelling.

Choked disk may be of certain localizing value, for it is apt to make its appearance, as Horsley emphasizes, earlier on the side of the lesion. Later, or when both eyes have become involved, the mere measurable height of the swelling is not of equal moment, for the older and more advanced process may project from the level of the retina less prominently than the more recent one. Attempts have been made likewise to use choked disk as an evidence of a cerebral or cerebellar lesion, the view being taken that the subtentorial growths in a large percentage of cases have a high grade of choked disk; but this, it seems, is merely a question of the time at which the diagnosis has been made, for in the majority of the cerebellar cases the symptoms, often of long standing, are rarely thought to be due to tumor until the advent of the neuroretinal change.

One particularly important fact, properly accredited to Hughlings Jackson, is the retention of visual acuity even in an advanced stage of the process. When once vision begins to fail, owing to atrophy of the nerve from the contraction of the new tissue, the loss of acuity may take place rapidly, usually

[1] Cushing and Bordley, *The Johns Hopkins Hospital Bulletin*, 1909, xx, 95.

with a concentric contraction of the visual field. Vision, indeed, may be suddenly lost. It is this which speaks urgently in favor of the modern palliative operation; for if the process has advanced to Marcus Gunn's Stage 4 the prognosis for permanent vision after operation is bad, and in Stage 5 almost hopeless.

Choked disk is a sign by no means limited to intracranial tumors, for any condition which similarly and in equal degree increases pressure will naturally lead to a like neuroretinal œdema. There are certain reasons, indeed, for believing that the so-called albuminuric retinitis is merely a modified form of the same lesion, one which cannot be distinguished at times from the choked disk of tumor;[1] and the same may be said of the neuroretinal change which accompanies cerebral concussion or contusion, apoplexy, thrombosis, and a number of other intracranial conditions. It is, comparatively speaking, rare in congenital hydrocephalus and meningitis.

**Choked Labyrinth.**—This condition—the *Stauungslabyrinthe* of Steinbrugge—thought to bear the same relation to the auditory that choked disk bears to the optic nerves, has been much debated. It is a conjectural lesion which has not been subjected to conclusive histological demonstration or reproduced experimentally. Although many cases show a diminution of auditory acuity, there are no definite clinical means of testing for the lesion —at least nothing equivalent to the ophthalmoscope in choked disk. Batten and Collier even think that stasis may affect the spinal cord and the posterior roots, especially of cervical and lumbar regions. Degenerations have been demonstrated in the entrance zone of the roots, and the condition is said to occasion pains in the root areas and loss of the tendon reflexes.

**Dyschromatopsia** (interlacing and inversion of the color fields).—Since Charcot, this has been regarded by clinical neurologists as a condition peculiar to functional or hysterical states. The perimetric examinations by Bordley and Heuer in our cases in which tumor has either been demonstrated by operation or autopsy, have shown that it is one of the most constant signs, and, inasmuch as many of the patients have at some period been treated for hysteria, one cannot escape a certain skepticism as to the correctness of many diagnoses of hysteria which have been based upon this sign.

The condition may apparently occur even before any ophthalmoscopic changes are apparent, and twice in our series the diagnosis has been correctly made through the presence of this symptom before the appearance of a choked disk. That it is in some way associated with pressure is evident from the rapidity with which the normal color field relations are restored after the relief of pressure. In its most typical form the change affects the blue field more markedly than the others, so that its boundaries may interlace with or become completely inverted within those of the red field.[2] There are numerous varieties, all conforming more or less with this primary type. Thus, we may have scotomata for blue or total achromatopsia for this color alone. Hemiachromatopsia, possibly homonymous, may occur with inversion of blue on the side of retained colors; and such a hemiachromatopsia may

[1] Byrom Bramwell, *Clinical Studies*, 1907, N. S., v, p. 1; Bordley and Cushing, *Amer. Jour. of Med. Sci.*, October, 1908, cxxxvi, 484.

[2] It is of importance that the blue disk used for the tests shall be a pure spectral color unmixed with green. Many of the disks furnished by the manufacturers are impure, and naturally a trace of green will apparently invert the blue field. Bordley and Cushing, *Archives of Ophthalmology*, 1909, xxxviii, 451.

foretell an impending hemianopsia for form as well. Thus, this symptom, which we have come to regard as almost as valuable as choked disk, may be not only indicative of an early stage of general pressure but at times of localizing import as well. Such a test of color vision is, of course, precluded in infants, in aphasic patients, and in those too ill to concentrate their attention.

**Other Pressure and General Symptoms.**—Other less important general pressure symptoms are *vertigo* and *dizziness*, possibly more common with subtentorial lesions than with those of the cerebrum and possibly also related in some way with the labyrinthine disturbances which have been described; *drowsiness*, with repeated yawning, is not infrequent when pressure is considerable and the patient in an apathetic state; *convulsions* are occasionally seen irrespective of those characterizing involvement of the precentral gyrus; and certain *psychic disturbances* with intellectual dulness are to be made out in most cases of tumor, even with no direct involvement of the frontal lobes. Rapid *loss of weight* is common, although the reverse may occur in tumors affecting the hypophysis; and *disturbances of pulse rate, respiration, body temperature,* and *urinary secretion* may also be regarded as general symptoms in certain cases.

**The Focal Manifestations.**—In evidence of the gaps in our knowledge of neurological physiology, we must still refer to large areas of the brain as "silent"—areas which may be occupied by a tumor or destroyed by wounds without leaving any discernible functional alteration. Fortunately for the sake of the regional diagnosis of tumors, these areas are becoming more and more constricted. The function not only of the cortex, but of deeper parts as well, is becoming sufficiently well determined to be of great diagnostic service. Irritation or paralysis, whether from simple pressure or from destruction of the various centres or paths of known function, gives symptoms which are available for this purpose, whether the lesions occur in the motor or sensory spheres, in those related to the special senses for taste, smell, vision or hearing, in the more highly differentiated areas associated with the speech or stereognostic mechanism, in the psychic sphere itself, or involve the cerebral nerves in their peripheral intracranial course.[1] However, the matter may not be so simple as this for *symptoms at a distance*—the *Fernsymptome* of the Germans—false localizing signs which greatly confuse the primary focal and neighborhood manifestations of the lesion,[2] are apt to occur especially late in the history of tumor.

These distant symptoms are apt to be especially confusing when the growth occupies a fairly silent area, but leads to œdema elsewhere. We have been misled by false signs in a number of instances, some illustrations of which may be given. Thus in one case a tumor of one frontal lobe so greatly indented the other as to make the latter appear to be the primary seat of trouble; a tumor of one occipital lobe in a child blind from optic atrophy, so that hemianopsia was absent, gave definite cerebellar symptoms; medullary symptoms have been far from infrequent in cases when there is a large intracranial growth situated almost anywhere; hydrocephalus has often effectually masked practically all local symptoms. A growth may become so large in a young child as to simulate essential hydrocephalus; a basilar process,

[1] For illustrative cases of tumors in the various areas Beevor's *Lettsomian Lectures* may be consulted, *Trans. of the Med. Soc. Lond.*, 1907, xxx, 150 to 233.

[2] Collier, *Brain,* 1904, xxvii, 490.

particularly when luetic, may lead to vascular thrombosis with distant symptoms; nerves situated far from the lesion may be stretched and paralyzed. This is particularly common with the sixth cerebral nerve, which has been found bent upon itself through pressure from one of the lateral branches of the basilar.

It was made apparent in discussing the general pressure manifestations that one or another of these phenomena may possess elements of localizing value. Thus, persistent suboccipital headache and vertigo are characteristic of a cerebellar lesion; choked disk is apt to occur first upon the side of involvement in lesions of the cerebral hemispheres;[1] hemiachromatopsia, which may foretell a total hemianopsia, likewise may point to the hemisphere involved; and any unusual disturbances of pulse and respiration, to a lesion in the neighborhood of the medulla. These, however, are not commonly classified under focal symptoms, which concern more specifically the disturbed function of definite lobes, centres, or paths which can be conveniently taken up *seriatim* under the heading of:

**Regional Diagnosis.**—This can be helped out by ways other than the mere study of neurological phenomena. Thus auscultatory percussion of the shaved scalp may occasionally be helpful, the audibility of the transmitted sound of the tuning fork being lessened over the tumor, particularly if it be a solid growth or one placed near the skull. The Röntgen rays may prove of value in locating growths which have undergone calcification or have eroded or displaced the cranial bones—particularly true of tumors which deform the sella turcica—or in disclosing teratoid tumors which contain bone themselves. The Neisser-Pollack method of puncture through the intact skull (*Hirnpunktion*) is not to be commended. In this procedure, after boring through the skull, a hollow needle is plunged into the brain and the fragments of tissue with which it becomes plugged are examined.

*Lumbar puncture* as a diagnostic measure has been much abused, and many sudden deaths are attributable to it. On the sudden withdrawal of the supporting column of spinal fluid the pressure from above, which continues unabated, serves to wedge the medulla and surrounding fringe of cerebellum into the cone of the foramen, with resultant anæmia of the vital centres and often sudden respiratory interruption. Three of the fatalities in the unoperated cases in this series promptly followed this measure, and death likewise in three of the patients subjected to operation occurred soon after withdrawal of the lumbar fluid during the operation, with the object of diminishing tension so as to permit of freer exploration. The procedure in tumor cases is therefore hazardous and of no particular diagnostic value; for mere measured tension of the spinal fluid is of no significance, nor is a cytological study of the fluid of great importance, now that we possess a definite serum reaction for syphilis.

**Frontal Lobe.**—Lesions in this situation lead to mental disturbances, which in a way are comparable to the symptoms one meets in paresis—indifference, unpunctuality, mental enfeeblement, loss of memory and power

[1] To quote from Marcus Gunn: "Double optic neuritis with surrounding retinal change coming on quickly suggests the cerebellum; a one-sided neuritis or marked difference suggests the cerebrum, and on the whole is in favor of the tumor being on the same side as the excess of neuritis." This also is Horsley's view. Leslie Paton's more recent studies of the same series of cases disagree with these views. (*Brain*, 1909, xxxii, 65.)

of attention, change in disposition with more or less marked irritability or taciturnity or obstinacy or jocularity, etc., often a rambling speech, lack of realization of the illness, and change in the general conduct of life with habits of untidiness. These, in greater or less degree, characterize most of the cases, although it is often astonishing to find how inconspicuous the symptoms may be with a very extensive growth. They may often be of rather abrupt onset and not until the situation of the lesion is definitely disclosed and careful interrogation made into the patient's previous mental state is it possible to learn, even from close associates, that in all probability some mental alteration has been of long standing.

There is undoubtedly a great difference between the two sides, the symptoms being more pronounced in left frontal lesions in the right-handed. It is to be noted that a tumor of one side may markedly affect, by indentation or compression, the opposite hemisphere. The symptoms also depend somewhat on the situation of the growth, whether it affects the prefrontal region, in which case they are said to be more pronounced, or whether it lies at the base or upon the external surface of the frontal lobe. Disturbances of the speech mechanism and of the motor activities of the contralateral side of the body may be expected when the posterior part of the lobe is affected. Vocal aphasia is common, and a good instance of the rarer agraphia has been recorded by McConnell.[1] There may be cortical epilepsy with movements of head and eyes to the opposite side; automatic movements are also described, and a form of frontal ataxia. Granger Stewart[2] has shown that a homolateral tremor with loss of abdominal reflex on the contralateral side of the body is of frequent occurrence. Lesions here are also accompanied by relatively early nutritional disturbances.

**Temporal Lobe.**—With the exception of its tip and of the uncinate area this constitutes a relatively silent region. Large tumors may give such vague symptoms that the first local sign of trouble may be the tenderness and bulging which foretells perforation and atrophy of the squamous wing. It is not uncommon in the usual subtemporal decompressive operation to expose an unsuspected temporal growth; this has occurred four times in our series. Left temporal lobe tumors in right-handed people may lead to disturbances of motor speech when they encroach on the frontal area, and to hemilingual or facial weakness when they similarly implicate the lower precentral gyrus. It is unusual for them to cause auditory disturbances, although inability to apprehend spoken language is supposed to follow a lesion of the first temporal gyrus, with more or less upsetting of the entire speech mechanism.[3]

Cortical lesions at the tip of the temporal lobe which implicate the uncinate and possibly the hippocampal gyri are known, largely through the papers of Hughlings Jackson in 1889, to occasion certain peculiar seizures known as the *uncinate group of fits*. Purves Stewart, in 1899, was able to find reports of six typical cases, and Mills[4] has recently collected as many more. The

[1] *Univ. of Penn. Med. Bull.*, 1905, xviii, 156.

[2] *Rev. of Neurol. and Psych.*, 1906, iv, 809.

[3] The rarity with which word-deafness occurs in cases of involvement of the superior temporal gyrus by tumor or by the protrusion of the lobe through a palliative defect makes the cortical representation of this primary word centre a matter which would seem to deserve as careful consideration and possible revision as has been given to Broca's vocal speech centre since Marie's recent criticism.

[4] *Jour. of Amer. Med. Assoc.*, 1908, li, 879.

condition, therefore, would seem to be rarely recognized, although it must be of fairly common occurrence, for we have had six typical cases in our series.

A tumor which occasions these symptoms may either arise primarily in the temporal lobe or may originate elsewhere, as in the interpeduncular space, and involve the uncinate gyrus secondarily. The uncinate seizures are characterized by subjective sensations of smell or taste—the impression usually being a disagreeable one—and often by an epigastric aura. Together with these sensations, movements often occur such as those of the acts of tasting or smelling, chewing or swallowing. Salivation is not unusual, and was pronounced in one of our patients. Consciousness is retained, as a rule, in these attacks, and they are not infrequently replaced by or associated with so-called "dreamy states," described as a distressing sense of unreality of surroundings. Respiratory phenomena have been noted. Neighborhood symptoms occur. In one of our patients there were secondary hypophyseal disturbances; in another, a fluctuating contralateral hemianæsthesia and homonymous hemianopsia due to pressure against the adjoining crux and visual pathway were present—a combination of symptoms which had led to a diagnosis of hysteria, for the tumor proved to be a benign one and had been present for many years. We have found in these uncinate gyrus lesions that disturbances of the visual fields are common, sometimes as an upper quadrantal homonymous defect.

**Paracentral Convolutions.**—For evident reasons by far the greater number of tumors which have been localized in the past have involved the motor and sensory gyri. A small growth which implicates the surface of this part of the brain almost invariably leads to irritative symptoms, beginning either with a primary motor or sensory aura in leg, body, arm, neck, face, or tongue, as the case may be. These focal or Jacksonian seizures may for a long time be the only manifestations; they may remain limited to the part involved or may spread with a typical "march," so as to involve the entire body in convulsive movements with loss of consciousness. As the lesion enlarges it is characterized by irritative disturbances to be followed by paralytic ones, shown first in the musculature of the part in which the attacks originally began. The more superficial the lesion the more circumscribed may be these paralyses; the deeper the lesion and the nearer the internal capsule the more extensive they are.

It must be borne in mind that the cortex of the central fissure dips down to a depth possibly of an inch; also that, as Sherrington and Grünbaum's observations have shown, the motor strip extends to the depth of the fissure, so that a lesion truly cortical may nevertheless lie at a considerable depth below the surface of the hemisphere. Sensory anæsthesia is much less likely to follow primary sensory fits than is motor palsy to follow either sensory or motor seizures; for although the cortical terminals for common sensation have with some definiteness been placed in the gyrus centralis posterior, still there are several relays between periphery and central end-stations, and a fairly extensive cortical or subcortical lesion is necessary before definite areas of cutaneous anæsthesia become demonstrable.

The most characteristic symptom of tumor in this situation—if one wishes to delay intervention until it occurs—is the gradual transference of a local convulsive movement into a local paralysis of movement. The paralytic features occur first as a mere exhaustion after the attack in the

affected muscular territory and then as an actual paralysis coincident with destruction of the centre. After this convulsions no longer occur in these muscles, but in those whose centres are adjoining. The first convulsion is usually local, rarely a general one, although soon local and general convulsions become interchangeable. Not infrequently between the attacks continuous twitching—"epilepsia continua"—may occur. It is the usual rule for all objective sensory disturbances to be wanting in the paralyzed area, although it is not rare to find certain disturbances of tactual sense or of the sense of localization.

**The Parietal Lobe.**—A lesion of the superior lobule, particularly if subcortical, leads to a contralateral disturbance of the stereognostic sense.[1] If the subcortical lesion be large and encroach on the postcentral gyrus, it may occasion additional sensory disturbances, such as loss of muscle sense, of position in space, or even of some forms of common sensation. Consequent upon the sensory disturbances certain irregularities of moment appear (the motor apraxia of Lœipmann). The arm, for example, although not in a strict sense paralyzed, for the patient when observing the member can make powerful though somewhat awkward movements, yet cannot be used for any purposeful act, particularly if the patient be blindfolded. More or less muscular rigidity accompanies the condition, although it is unassociated with any direct implication of the pyramidal tract.

One characteristic symptom elicited by a tumor involving the left angular gyrus in the right-handed is the inability to appreciate written language or even to read letters—word-blindness (*alexia*). A deep-seated lesion of either lobule may involve the fibers of the optic radiation and lead to a half-blindness of the corresponding halves of both retinas, either for colors alone or for form also—homonymous *hemiachromatopsia* or *hemianopsia*.

The right parietal lobe represents a comparatively silent area, regarded by Sänger as the most favorable for decompressive operations.

**The Occipital Lobe.**—A crossed homonymous hemianopsia is the characteristic, and may persist for a long time as the only symptom in addition to those incidental to general pressure. If optic atrophy secondary to a choked disk has occurred, and there is no history of half-blindness before the loss of vision, a regional diagnosis may be impossible. Occasionally the fields for color may be affected before those for form suffer any restriction, and cases have been recorded in which quadrantal blindness has been present, although these fractional defects in the field are possibly more characteristic of implication of the visual pathway farther forward as it approaches the optic tract.

Homonymous hemianopsia, of course, is characteristic of a unilateral lesion of the visual pathway in any part of its course, from occipital lobe to chiasm, and neighborhood symptoms must be depended upon to determine the seat of the lesion. When the occipital cortex, particularly the more important region of the calcarine fissure, is involved, the loss of half-vision may be ushered in by certain visual hallucinations, such as scintillating

[1] Although much discussed in its various aspects, astereognosis seems to be a fairly definite and reliable symptom of lesions of the superior parietal lobule. Cases have been recorded by Oppenheim, Bruns, C. W. Mills, and many others. A most typical instance in a case of stab wound—not tumor—has been recorded by the writer in the *New York Med. Jour.*, 1907, lxxxv, 161.

# PLATE XX

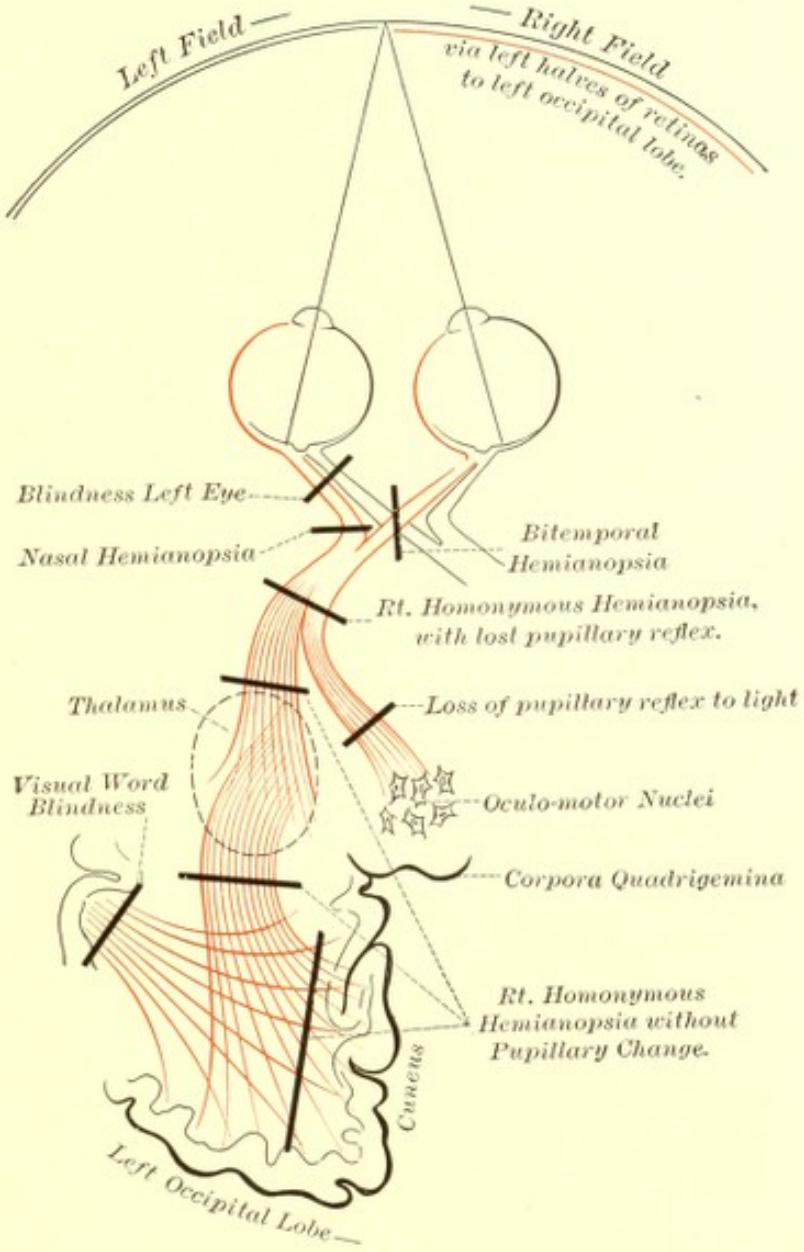



Diagram to Show various Forms of Visual Disturbance following Lesions in Different Portions of the Left Visual Pathway.

scotoma. Involvement of the optic radiation itself on the left side may be accompanied by some of the parietal lobe symptoms just enumerated—alexia or optic aphasia. Lesions farther forward, which include the fibers from the oculomotor nuclei on one side and affect the arc of the pupillary light reflex (Plate XX), lead to the hemianopic loss of this reflex on the blind side (Wernicke)—a condition which is wanting in pure occipital lesions. Occipital lobe tumors which have reached a great size may produce pontine disturbances or even symptoms indicating some implication of the corresponding cerebellar hemisphere, despite its protection by the well-hung tentorium.

**Centrum Semiovale and Basal Ganglia.**—Needless to say, lesions in any of the situations heretofore considered involve to a greater or less extent the subcortical areas, and it is often difficult to determine from the symptomatology alone whether a tumor is entirely central or whether it actually reaches the cortex. This is the more so since the cortical areas represented by the insula and by the deeper fissures dip down well into the central parts of the hemispheres, and some cortical manifestations may be present in many of these lesions, even though they lie far beneath the actual exposed surface of the brain. It is chiefly by the relatively large extent of the contralateral paralyses that one is enabled to conjecture the approximate depth of the lesion. Particularly in the left half of the brain the deeper lesions of the centrum lead to very extensive disturbances, not only in movement and sensation but in psychic activity, in the speech mechanism, and in vision.

Tumors of the *basal ganglia*, particularly those of the thalamus, are often capable of localization, the so-called *thalamic syndrome* having received much attention of late. The most characteristic symptom of a thalamic lesion is a contralateral disturbance of movement, either in the nature of a coarse tremor or of choreic or athetoid movements (Gowers, Oppenheim, Raymond, and others), often accompanied by painful subjective paræsthesias. Indeed, this is the only area of the brain a lesion of which seems to produce subjective sensations of discomfort referred to the periphery. Roussy[1] has outlined this syndrome as follows: The patients present (*a*) persistent hemianæsthesia, more or less marked in modalities of touch, pain, and temperature, but always pronounced in regard to deep sensation; (*b*) slight hemiplegia of a transient nature without contractures; (*c*) slight hemiataxia and more or less complete astereognosis; (*d*) persistent paroxysmal pains on the affected side, often intolerable and unrelieved by any analgesic remedy (*hemiplegie douloureux*); and (*e*) athetochoreic movements on the affected side.

In addition to these, the most characteristic phenomena, neighborhood symptoms occur, such as contralateral hemiplegia; oculomotor palsies, such as crossed pupillary dilatation and ptosis; emotional palsy of the face; hemianopsia; deafness from involvement of the lateral geniculate bodies, and so on.

In the hemiplegias which are due to lesions of the cortex or subcortex exaggeration of the deep reflexes and a persistent Babinski sign, together with contractions and epilepsy, are to be expected, with an absence of the athetochoreic movements and subjective painful sensations.

[1] La couche optique (etude anatomique, physiologique et clinique); le syndrome thalamique. Steinheil, Paris, 1907, p. 371.

**Corpus Callosum.**—There are only 26 recorded cases (Bruns) of tumor primarily involving this structure, and it is unusual for them to give symptoms sufficiently definite to insure a local diagnosis, although this has been correctly made in a few instances.[1] In addition to the usual pressure symptoms, these lesions are accompanied by a slowly progressive, bilateral, though not necessarily symmetrical, palsy of both extremities, without paralyses of the cerebral nerves, the latter being regarded by Raymond and others as an important negative symptom. Particularly when the anterior portion of the corpus callosum is involved, intellectual disturbances are marked,[2] often with serious alterations of the speech mechanism. There is apt to be a profound stupor, and the palsy of the extremities affects movement of the legs more than of the arms. Sensory disturbances are absent and the reflexes normal. The sphincters are uncontrolled. Tumors which involve the median portion of the corpus lead to more marked disturbances of motion, and a form of callosal ataxia has been described. Tumors which involve the splenium are apt to implicate adjoining structures with neighborhood symptoms, to be described under the following section.

In general, it may be said that a motor disturbance unequally represented on the two sides of the body with absence of sensory disturbances or evidence of involvement of the cerebral nerves is the characteristic syndrome.

**The Brain Stem (Corpora Quadrigemina and Pineal Gland, Pons, and Tegmentum).**—It is only natural that distant and neighborhood symptoms, as well as complex local ones, occur with lesions in this situation—the "cross-roads" of the nervous system. Thus pressure upon or involvement of the internal capsule or cerebral peduncles produces a more or less marked contralateral hemiplegia, possibly with some degree of hemianæsthesia; and if the growth extends outward from the mid-brain hemianopsia may occur, and likewise the muscular sense and stereognosis may be affected. The deep reflexes may thus be increased with absence of the superficial ones—this being particularly true of the Babinski phenomenon. Any growth in this situation will in the course of time lead to an internal hydrocephalus, which greatly complicates the symptom complex, in consequence of the widely distributed pressure against the hemispheres.

Involvement of the *corpora quadrigemina* leads to a staggering gait with unsteadiness and deviation sometimes to one side or the other in progression, and an especial tendency to fall directly backward; also to a failure of sight and hearing when, as the growth spreads to the side, the lateral geniculate bodies become invaded or compressed; to nystagmus; to persisting palsies of the ocular movements, particularly those in a vertical direction,[3] with preservation of lateral movements; and possibly to loss of the pupillary reflex to light and even to convergence as the centres for the oculomotor nerves become involved. The pupils may be irregular; and Bielschowsky describes a form of nystagmus with clonic twitching. Thus a combination

[1] Putnam and Williams, *Jour. of Nerv. and Ment. Dis.*, 1901, xxviii, 645.

[2] More than with tumors of the frontal lobes themselves, according to Knapp, *Brain*, 1906, xxix, 35.

[3] "Persisting paralysis of associated lateral movement indicates a lesion of the posterior longitudinal bundle; of associated vertical movement, a lesion in the vicinity of the oculomotor nucleus; paralysis of associated movement does not result from lesions of extracerebral fibers." Spiller, *Paralysis of Associated Movements of the Eyeballs* (Blicklähmung), *Jour. of Nerv. and Ment. Dis.*, xxxii, p. 417.

of ocular paralyses and ataxia (Nothnagel) especially characterizes lesions in this situation, whether they arise in the *pineal gland* or corpora quadrigemina.

When the *tegmentum* is implicated, particularly the nucleus ruber and the superior cerebellar peduncle leading to it (the *cerebello-rubral system*), a particular syndrome is apt to be called out in addition to the above, characterized by a peculiar coarse, oscillatory tremor, described by Gordon Holmes.[1] If the red nucleus of one side is involved the tremor is contralateral. We have had one or two striking examples of the condition. In addition to this tremor, which Holmes likens to that of paralysis agitans, a mask-like appearance of the face occurs; and the oculomotor palsies above mentioned, with staggering gait, etc., commonly occur. The disorders of movement with posterior thalamic lesions are more apt to be of an athetoid character than is the case with these tegmental lesions.

The *pons* is not an uncommon seat particularly of tubercles and gliomata. The latter may continue to be traversed by intact fibers, so that they may reach an astonishing size without producing such symptoms as would be expected of a lesion here. The characteristic feature, however, of most pontine tumors is the well-known alternating or crossed hemiplegia, with nuclear cerebral palsies on the side of the lesion, due to direct implication of the centres, and a contralateral palsy of the extremities with the usual evidences of pyramidal tract degeneration.

Various forms are recognized, possibly the most common combining a homolateral facial palsy with a crossed involvement of tongue and extremities. The facial territory may be the first to be affected, the brow often escaping; or, on the other hand, the palsy of the tongue and extremities may be primary, depending on the seat of the lesion. In another type the trigeminus may be affected on the side of the lesion with anæsthesia of one side of the face, and there may be a coincident crossed anæsthesia, pure or dissociated, affecting the paralyzed limbs. Another form may show itself, possibly in the absence of facial or hypoglossal disturbances, as an abducens palsy, crossed with a motor involvement of the limbs; and in these cases there is a characteristic paralysis of conjoint ocular movement toward the side of the lesion. Abducens palsies must be scrutinized carefully, however, as they are the commonest of all cerebral nerve lesions, and may, from pressure or from stretching through dislocation, accompany a tumor in almost any situation.

When a pontine growth involves the main conducting paths or the nuclei on each side, these typical crossed paralyses will be wanting, and some evidence of bilaterality of the lesion may in many cases be expected before the end. We consequently may have a multiplicity of combinations with unilateral or bilateral disturbances of facial, trigeminus or abducens, and it is even possible to have a coincident cerebral paralysis of all four extremities and both hypoglossi. The variations are too many to enumerate, although Bruns brings them into four main categories: (1) Bilateral paralysis of the cerebral nerves and extremities; (2) bilateral cerebral nerve with unilateral paralysis of the extremities; (3) unilateral cerebral nerve and bilateral paralysis of extremities; (4) bilateral cerebral nerve paralysis without involvement of the extremities.

[1] *Brain*, 1904, xxvii, 327.

Neighborhood symptoms are shown, if the growth presses forward, by oculomotor symptoms; if backward, by affections of the medullary nuclei, evidenced by disturbances of deglutition or phonation—the bulbar palsies. Salivation, albuminuria, polyuria, and extreme degrees of pyrexia have been described in association with pontine tumors.

**The Medulla Oblongata.**—The complexity of paths and centres crowded here into a small space naturally makes the symptom complex most variable; nor are the lesions sufficiently common to enable us to more than sketch their clinical picture. The centres for the cerebral nerves, from the eighth to the twelfth, may be affected so that deafness or Ménière's syndrome, palsies of the palate and pharynx with dysphasia, vocal cord palsies, disturbances of cardiac or respiratory activity, dysarthria from weakness or paralysis of the tongue, and trigeminal anæsthesias (ascending root) may occur in combination with motor and sensory, crossed or uncrossed disturbances of the extremities. The medulla may be affected and the cardiac and respiratory centres suffer as a distant symptom of most intracranial tumors, owing to the crowding of the medulla into the foramen.

Tumors of the *fourth ventricle* occasionally give symptoms sufficiently characteristic to justify a focal diagnosis. In the greater number of cases observed the lesion has been due to a cysticercus, less often to a glioma, this being the usual seat of the ependymal glioma already described. Stern[1] has collected 72 cases of the former and about 50 cases of tumor. The symptoms are suboccipital pain, a peculiar bowed position of the head, a periodic occurrence of general pressure symptoms, often of abrupt onset, accompanied by severe headache, vomiting, and cervical rigidity. In some patients a changed position of the head may cause a sudden accession of symptoms (Bruns); this is supposed to indicate an unattached and movable cysticercus. An interval of complete freedom from symptoms may follow. Sudden death is apt to occur during some one of the periods, with a slowed pulse and respiratory failure. Choked disk is rare. Polyuria, polydipsia, or glycosuria may be present. The fluctuating intensity of the symptoms is presumably due to a varying degree of obstructive hydrocephalus.

**Cerebellum.**—Inasmuch as the symptoms which they elicit bear a close relationship, all tumors situated beneath the tentorium which affect one or the other cerebellar hemispheres or the vermiform process, either through pressure or by direct involvement, will be considered together. According to the classification of Stewart and Holmes, in their paper[2] based on a series of 40 cases observed at the National Hospital, of which number the diagnosis was confirmed in 22, these growths are considered under the headings (1) of *extracerebellar*, and (2) of *intracerebellar* tumors.

Tumors here, as in other parts of the brain, lead first or last to the cardinal symptoms of pressure—headache, vomiting, and choked disk—and very frequently to vertigo or dizziness. The headache is apt to be suboccipital, often on the side of the lesion, and not infrequently is associated with tenderness on pressure. The choked disk is supposed to be found in a much higher percentage than is the case with growths elsewhere, probably due to the fact that in the course of time a cerebellar tumor will almost certainly lead

[1] *Deut. Zeit. f. klin. Med.*, 1907, lxi, 64; also *Deut. Zeit. f. Nervenheilk.*, 1908, xxxiv, 195.

[2] *Brain*, 1904, xxvii, 522.

to an obstructive hydrocephalus, in consequence of which a neuroretinal œdema is almost inevitable and may occur abruptly, often with a rapid augmentation of general pressure symptoms. In the absence of this complication, and with a slowly advancing benign lesion, choked disk may be absent for years. Vertigo is more frequently associated with subtentorial than with supratentorial tumors. When marked, the patient experiences a sensation of the movement of self or objects. According to Stewart and Holmes, in both extracerebellar and intracerebellar tumors the patient often has the impression that objects are moving in a direction away from the side of the lesion: whereas he feels himself revolving, in the case of an intracerebellar tumor, in the same direction with the objects; in the case of an extracerebellar tumor he appears to be turning in the opposite direction.

A tumor arising in the substance of the cerebellum (*intracerebellar*) leads to symptoms which affect the musculature on the side of the body homolateral to the lesion, and occasionally to the peculiar convulsive seizures described by Hughlings Jackson and known as "cerebellar fits." A tumor arising from the meninges and compressing the cerebellum secondarily (*extracerebellar*), of which the most characteristic type is the meningeal endothelioma originating in the lateral cerebellopontine recess, leads to more or less evident pressure disturbance of the cerebral nerves on the side of the lesion, in addition to the symptoms of an intracerebellar growth.

A large number of these extracerebellar tumors are supposed to grow from the flocculus or from the sheath of the auditory nerve, and as they enlarge they destroy and press to the side the pons and markedly indent the anterior and lateral portion of the adjoining cerebellar lobe. Consequently the acusticus is the nerve most commonly affected, and either subjective noises or complete nerve deafness may occur. In view of its close proximity to the eighth nerve the facial may likewise be affected with weakness of its musculature on the side of the lesion. Even in the presence of a large tumor, however, it may be found stretched out to double its normal intracranial length, with little more evidence of weakness than is shown by a less complete closure of the eye in winking. A particular triad of symptoms has been pointed out by Oppenheim, namely, the disturbance of hearing, paralysis of winking, and diminished corneal reflex. The trigeminus is occasionally affected, more especially its sensory portion, with pain or tingling or dysæsthesia over its particular skin field. The abducens may likewise suffer and more rarely the glossopharyngeal, vagus, or hypoglossus. In intracerebellar tumors it is, relatively speaking, unusual for these nerves to be affected.

Disturbances of the body musculature are common in both intracerebellar and extracerebellar forms. Tremor, loss of muscular tone, even paresis of the limb and trunk muscles, weakness of the conjugate movements of the eyes, nystagmus, and staggering gait with tendency to fall, oftentimes toward the side of the lesion, and alteration of the deep reflexes, all occur. These symptoms are most pronounced on the side of the lesion. The ataxia, which may be very marked, is a coarse movement accentuated when the patient attempts to perform some delicate act; unlike the ataxia of tabes it does not increase when the eyes are closed—an evidence of its central origin. There is an associated condition, which Babinski has termed *adiadococinesia*,[1] in which the patient cannot repeat rapidly executed move-

[1] *Rev. neurologique*, 1902, x, 1013.

ments, such as pronation and supination of the forearm, owing to the defective coördination. Loss of tone in the muscles (*asynergia*) may be present, so that the arm and the leg may be completely flaccid with retention of the deep reflexes.

The ocular movements are also affected in ways characteristic of cerebellar disease. There is apt to be difficulty in forcing and retaining conjugate movement of the eyes toward the side of the lesion, and the attempt usually elicits nystagmoid movements. It is not infrequent for *nystagmus* to be present on looking toward as well as away from the presumed site of the lesion, but as Stewart and Holmes have pointed out, the jerking movements are apt to be more rapid on one side than the other, the slower rhythm being usually upon the side of the lesion.

The position of the head is apt to be toward one side, usually facing away from the lesion, and tilted back so as to relieve the strain on the cervical muscles. Alfred Gordon asserts that forcible movement of the head toward the lesion increases the headache and vertigo in a characteristic way. Irregular *tremors* are often present in the extremities. They are accentuated with the patient erect, his arms and hands held horizontally extended and in a forced position. A staggering gait is common, with a tendency to deviate, totter, or actually fall toward one side—more often toward the side of the lesion. A method of determining the affected side is to have the patient stand on one leg or the other, the chief disability appearing on the side of the lesion. Unsteadiness is equally characteristic of a tumor involving the middle lobe or vermis and one involving the hemispheres, although with the former there is a greater tendency to fall directly backward or forward than when the lesion is laterally placed.

The reflexes are apt to be affected, disparity between the two sides being of chief importance, for there is considerable difference of opinion as to whether they are increased or are diminished on the side of the lesion. It is not uncommon for them to be diminished at one time, and at a later period increased, depending in all probability upon the degree of advancement of the process. The superficial reflexes are not greatly altered, although occasionally, in association with an increase of the deep reflexes, an extensor toe phenomenon may occur.

**The Cranial Base.**—A variety of tumors, either of meningeal or cranial origin, may arise from one or another of the cranial fossæ and produce definite focal symptoms, owing chiefly to the involvement of the cerebral nerves, in addition to the evidences of general pressure. Syphilitic processes are common here. Bony or cartilaginous tumors originating in the accessory nasal sinuses may invade the cranial chamber. Epithelial tumors may grow inward from the deeper ear; such a case occurred in our series, with the unilateral involvement of all the cerebral nerves from the sixth to the twelfth inclusive. The *cavum Meckelii* is not an uncommon seat for tumors which may give all the pain of a severe trigeminal neuralgia during the process of stretching or destruction of the Gasserian ganglion; and keratitis is a frequent accompaniment of the process. But of all these multitudinous basal and extracranial lesions, those of chief interest and moment arise in the neighborhood of and implicate the—

**Hypophysis Cerebri.**—This is a favorite seat of tumors, both congenital and acquired, and since Marie put forward the view, in 1889, that the condition he had previously named acromegaly was associated either with a

hyperplasia or adenomatous tumor of the pituitary gland, this mysterious body has become the object of close study.[1]

Two separate factors must be taken into consideration with tumors in the interpeduncular region, commonly grouped as hypophyseal tumors. First, what is their effect, if any, on the gland itself; and second, what are the chief neighborhood symptoms? The former has heretofore received scant attention, but the neighborhood symptoms are well recognized and have the following characteristics: Any mesially placed tumor in the interpeduncular space naturally presses upon the optic tracts or chiasm, leading in the majority of cases to partial amblyopia and to primary optic atrophy. This in most striking form occurs as a bitemporal hemianopsia, although by no means is this the most common type, for one nerve is apt to suffer much more than the other; so that, for example, there may be total blindness in one eye and half-blindness in the other.

Choked disk is usually absent, as there commonly is no great increase in intracranial tension, although it may occur later should the growth enlarge so as to push its way into the third ventricle and obstruct the foramen of Munro. In one of our cases a choked disk became subsequently superimposed on completely atrophic nerve heads. Headache is often a common and distressing symptom, due rather to the distention of the dural pocket of the gland than to any general increase of the tension. The *x*-rays are particularly helpful in the diagnosis of these cases, for in many of them there is a characteristic deformation of the sella turcica; or the growth itself, if a teratoma, may cast a shadow.

The glandular symptoms are quite another matter. Since Marie's discovery of the frequent association of hypophyseal enlargements with acromegaly there have been innumerable reports of cases in which a tumor of the gland or its neighborhood has been unaccompanied by evidence of "Marie's disease." These findings have usually been interpreted as contradictory of his views. An especial group of these cases, as shown by Fröhlich,[2] is characterized, in addition to the usual regional tumor symptoms, by a peculiar tendency to the deposition of fat with sexual infantilism, hypotrichosis, etc.

The hypophysis has been proved by Paulesco to be a gland essential to the maintenance of life, its total removal leading to fatality, with a peculiar symptom complex—*cachexia hypophyseopriva*. In repeating Paulesco's experiments Crowe, Homans, and the writer have found that partial removal of the anterior lobe may lead to adiposity and to atrophy of the ovaries or testes.[3] Thus an explanation is given of the syndrome of Fröhlich, which would seem to be due to a condition of hyposecretion (*hypopituitarism*). On the other hand, acromegaly, as shown by the improvement in the condition after hypophyseal operations with partial removal of the gland (Hochenegg; Cushing), would appear to be due to an oversecretion (*hyperpituitarism*).

Both of these conditions may be associated with tumor, although it is probable that in varying degrees of hyper- or hypo-activity they are of common occurrence—as common as the over- or underactivity of any ductless gland,

[1] N. C. Paulesco, *L'hypophyse du cerveau*, Paris, Vigot Frères, 1908.
[2] *Wien. klin. Rundschau*, 1901, Nos. 47, 48.
[3] H. Cushing, *Jour. of the Amer. Med. Assoc.*, 1909, liii, 249.

the thyroid for example. In the case of overgrowth (gigantism when starting early in life; acromegaly when in adult age), hypertrophy or adenoma of the gland itself is usually found. In the cases characterized by "adiposo-genital degeneration" (showing persistence of sexual infantilism when originating before adolescence; and a tendency to revert to this condition when beginning in adult life) the tumor may either implicate the gland directly or may be situated above it in the infundibular region and lead to a pressure atrophy. There are other symptoms caused by these inter-peduncular tumors, such as polyuria, glycosuria, amenorrhœa, impotence, psychic disturbances and so on, which in all likelihood are bound up with the activities of the other ductless glands. It is not unlikely, in view of Herring's discovery, that the hyaline secretion of the posterior lobe normally discharges into the cerebrospinal fluid of the third ventricle, that disturbances of hypophyseal activity may occur first or last in almost all cases of intracranial tumor.

**Differential Diagnosis.**—The three main questions which must be answered by the clinician when confronted with a suspected case of intracranial tumor in the order of their relative importance are: (1) Is a growth actually present? (2) If so, what is its presumable situation? (3) What is its nature? It cannot be emphasized too strongly that a brain tumor is rarely diagnosed in general practice until the onset of the major symptoms of pressure—the familiar triad of headache, vomiting, and choked disk. The ophthalmologist is too often the first to suspect the presence of a tumor, patients having come to him for correction of possible errors of refraction or in consequence of failing vision. It is important to appreciate the fact that tumors in the retrospect may prove to have been of years' duration before the onset of this unmistakable general pressure syndrome—an indication of the attention which must be paid in the future to the symptoms which are premonitory of these outspoken manifestations of intracranial trouble.

The early mistakes in diagnosis make a most disconcerting professional record. In our special series the majority of the cases gave a history of having undergone treatment for long periods for an astonishing variety of presumed maladies, and, it may be added, inevitably for syphilis. The usual mistakes are naturally attributable to an effort to ascribe the more or less vague disturbances antecedent to the general pressure symptoms to some cause other than the unsuspected intracranial lesion. Diagnoses of hysteria, of psychoneurosis or neurasthenia, or of gastric or ocular reflex headaches are particularly common.

**Hysteria.**—Certain of the so-called functional disturbances are inevitably superimposed on every organic lesion, and when the primary seat is obscure, and particularly if it affects the nervous system, the functional superstructure may so overtop the basal trouble as to effectually conceal it. One of our patients—the wife of a physician—for twenty years had suffered from irregular headaches, periods of amenorrhœa, intermittent grades of hemianæsthesia and hemianopsia, inversion of the color fields and peculiar dreamy periods of semiconsciousness. Although seen by many during this period there was but one diagnosis—hysteria—until a terminal choked disk betrayed the nature of the lesion—a benign tumor of the right temporal lobe and uncinate region.

In his excellent paper on brain tumor diagnosis Russell[1] says that "the signs of organic disease cannot be confounded with anything that hysteria is able to furnish." This is true of a late period in the disease, not of an early one; and we have seen, in discussing dyschromatopsia, that this symptom, supposedly pathognomonic of hysteria, proves to be one of the most definite and frequent manifestations of a new-growth.

The presence of organic changes, shown by a choked disk, cerebral nerve palsies, loss of the pupillary reflex, absence of the knee jerks, an extensor plantar response, typical Jacksonian fits, and the like, serves to eliminate hysteria; but in the absence of these definite symptoms and in the presence of the so-called functional ones, brain tumor is often most difficult to rule out. Hysterical hemiplegia may at times be distinguished from organic disease by Hoover's test, which consists of an involuntary downward pressure of the supposedly paralyzed limb when the patient attempts to raise the sound member against resistance.

**General Paralysis of the Insane.**—This is especially liable to be confounded with frontal lobe tumors, particularly when the optic nerves have become atrophied, although a choked disk is said never to occur. The symptoms of tumor are usually progressive, those of general paralysis fluctuating; and the Argyll-Robertson pupil and characteristic defects of articulation are usually conclusive.

**Disseminate Sclerosis.**—Disseminate sclerosis may simulate tumors of the cerebellum and mesencephalon. There may be headache and choked disk; optic atrophy is common. The vertigo, vomiting, cerebral nerve palsies, nystagmus, incoördination and spastic condition of the extremities may occur in both.

**Vascular Lesions.**—Vascular lesions are often most difficult to distinguish. Pressure symptoms may be present, and local signs are equally common. In arteriosclerosis the retina often proves a tell-tale should vascular disease, in the absence of choked disk, be marked in the retinal arteries. When a combination of tumor and cerebral arteriosclerosis is present a diagnosis may be very difficult. In our series there was a patient aged seventy-two, with transient aphasia and focal seizures involving the face, in whom existent vascular disease could well have accounted for the symptoms, but an exploration revealed a small primary glioma of the lower precentral region.

**Cerebral Thrombosis.**—Cerebral thrombosis may be difficult to distinguish from tumor when the process is slow, or, on the other hand, when tumor symptoms happen to be of fairly rapid onset. The vascular lesion leads to areas of softening which become œdematous and thus increase tension, so that headaches and choked disk, together with the local manifestations of trouble, make the intracranial symptoms indistinguishable.

**Apoplexy.**—Here, also, the characteristic sudden onset of symptoms speaks against tumor, although it must always be borne in mind that the hemorrhage may have occurred in the substance of a vascular growth. An apoplectic "stroke" in a young individual with no evidence of general arteriovascular disease and particularly when there has been a history of headaches, must arouse a suspicion of tumor.

[1] *Brit. Med. Jour.*, 1907, ii, 1120.

**Aneurisms.**—Aneurisms of the larger vessels, particularly of the internal carotid, basilar, and middle cerebral, are not exceptionally rare, and may reach a considerable size without symptoms; on the other hand, they may closely simulate tumor. This is particularly true of carotid aneurisms which are found in the interpeduncular space near the chiasm, which may lead to hemianopsia, optic atrophy, or oculomotor palsy, in addition to the severe headaches simulating those of pituitary tumors. They occasionally may be recognized by a murmur, but this is less characteristic of the simple saccular aneurism than of the easily recognized arteriovenous variety. They may occur at any age, even in childhood, although naturally they are more common in adult life. Usually small, they may, as in Bramwell's case, reach the size of an orange. Their blood-content tends to clot, and may organize with spontaneous healing. Gowers gives the following series in order of their frequency: Sylvian, basilar, internal carotid, artery of the corpus callosum, posterior and anterior communicating, vertebral, posterior cerebral, inferior cerebellar artery. Atheroma, alcoholism, syphilis, and senility are frequent causes.

When headache is present it is apt to be severe, and is increased by straining. Convulsions and vomiting may appear, but choked disk is rare, and a bruit is exceptionally uncommon except in the arteriovenous form, which could hardly be mistaken for tumor. The only patient in our series in whom a subjective and objective cranial bruit was present proved to have a contralateral glioma, and there was no aneurism. A bruit should be regarded as aneurismal only after the most careful consideration, for it may be produced by a vascular tumor, whether from its own circulating blood or by pressure against one of the large basal vessels. They are rarely diagnosed during life, and usually terminate in rupture with sudden death. In only 37 per cent. of the 555 cases studied by Beadles[1] did aneurism give symptoms during life, and in 67 per cent. death was preceded by an apoplectic seizure.

**Nephritis.**—The symptoms of intracranial œdema secondary to renal disease may be indistinguishable from tumor. In both, headaches, vomiting, and neuroretinal changes may be present, and focal paralyses or convulsions may accompany the nephritic œdemas—for the process is apt to be more or less restricted and need not be general in character. The so-called "albuminuric neuroretinitis" is regarded by Bramwell,[2] Bordley, and the writer as a process due largely to the same factor which produces choked disk, namely, pressure. We have seen the characteristic stellate figures due to exudates in many cases of tumor; on the other hand, they may be completely wanting in nephritis. *Uræmia* may have many features in common with the late stages of a new-growth.

**Abscess.**—Wanting a clear history of an infected cranial wound, of otitis media or infection of the accessory nasal sinuses, a differential diagnosis may be difficult in the extreme. On three occasions we have mistaken abscess for tumor. Chronic abscesses have thick walls, and when exposed may be easily shelled out from the brain. They may be of long duration, with no antecedent history, and give symptoms the exact counterpart of a new-growth. Even a high grade of choked disk, which is thought to be

[1] *Brain*, 1907, xxx, 285.
[2] Byrom Bramwell, *Clin. Studies*, 1905, iii, N. S., p. 183.

rare, may accompany them. In the retrospect, however, it becomes apparent that the symptoms are more apt to be fluctuating than is the case with tumor. Leukocytosis and fever may be absent, even though viable organisms can be recovered after some months from the cavity of the more or less dormant abscess.

**Serous Meningitis or Ependymitis.**—Serous meningitis or ependymitis leading to hydrocephalus may give a definite tumor symptom complex, although after the rather acute onset the symptoms are more apt to be fluctuating in severity, often showing long periods of abeyance. In one of our cases an obstruction of the left foramen of Munro occurred from ependymal inflammation, with symptoms indistinguishable from a tumor of the left hemisphere. Doubtless many of these peculiar cases are capable of spontaneous recovery, and they should be included in the group of false or pseudo-tumors. It may be particularly difficult to distinguish a subtentorial tumor from meningitis serosa. In addition to the pressure symptoms the latter may show nystagmus, paresis of an external rectus muscle, usually the right (Oppenheim), an inactive corneal reflex, tinnitus, and diminished hearing and cerebellar ataxia.

**Hydrocephalus.**—It seems hardly possible that a tumor could be mistaken for chronic or essential hydrocephalus, but such is the case, not only in the presence of a slowly progressive cerebellar lesion, but even with large tumors arising above the tentorium which happen to obstruct the ventricular outflow. Diastasis of the sutures occurs early in the cerebellar tumors of the young, and the cranial enlargement is symmetrical: in tumors of the hemispheres an asymmetrical configuration of the head is more apt to occur, although even in these cases an associated ventricular hydrops may lead to a condition with a symptom-complex similar to simple hydrocephalus. A careful analysis of the incidents of onset will usually serve to distinguish the two conditions.

A low grade of choked disk, rare in simple hydrocephalus, is almost always present with the tumor cases; and the latter are more apt to present rigidities with exaggeration of the reflexes and ataxia of movement than are the former. In one of our cases diagnosed by a number of neurologists and surgeons as hydrocephalus consequent upon ependymitis, an enormous psammoma of the right hemisphere was found; another child treated by ourselves for a year as a simple hydrocephalic proved to have a cystic glioma of the cerebellum.

**Hæmatoma of the Dura Mater.**—Many conditions of lesser moment must be differentiated at times from tumor, such as hæmatoma of the dura mater, characterized by a neomembrane of inflammatory origin with increasing layers of bloody extravasation. This may be found to cover the entire convexity of one hemisphere or may be merely a local process. It is usually accompanied by symptoms of progressive general paralysis.

**Traumatic Cysts.**—Traumatic cysts or even those of congenital origin may simulate tumors or may be actually associated with tumors.

**Lead Encephalopathy.**—Lead encephalopathy, according to Bramwell,[1] may present headache, vomiting, convulsions, often epileptiform in character, and double optic neuritis, and must be carefully distinguished from tumor when there is any suspicion as to lead poisoning. He affirms that

[1] *Clin. Studies*, 1908–09, N. S., vii, 91.

the two conditions so closely resemble one another that he never commits himself to a positive diagnosis of intracranial tumor without previously excluding lead poisoning.

**Pseudo-tumors.**—Under this caption Nonne,[1] Oppenheim, and Hoppe have reported a number of instances in which a definite tumor symptom complex has completely subsided under specific or general medical treatment, and others in which, on postmortem examination, there was absolutely nothing to account for the definite pressure and focal symptoms present during life. Needless to say, hysterical conditions are excluded by the presence of definite symptoms of organic disease, often with a choked disk. We have had two cases of supposed tumor with death following operation in which no cerebral lesion was disclosed; and it is possible that a number of the patients in whom all evidences of pressure completely and permanently subsided after decompression have been cases of "pseudo-tumor cerebri."

Certain of the œdemas of one origin or another, meningitis serosa and like conditions, are very difficult to recognize after death, and they represent states which lend themselves favorably to operation. The surgical cases which recover after simple decompression, even when they have been of sufficiently long standing and severe enough to have caused optic atrophy from choked disk, may in some instances represent healed tubercle, abscess, or cystic metamorphosis of a glioma. Hoppe calls attention to the possibility that some of the pseudo-tumors with negative postmortem findings may be due to the rare chronic cerebritis or cerebral hypertrophy described by Rokitansky.

**Course and Prognosis.**—Generally speaking, an intracranial tumor represents one of the most serious of maladies, dreaded by victim and physician alike, in view of the suffering to be endured by the one and the incapacity of the other to relieve. As the diagnosis is rarely certified until pressure symptoms have become full-blown, the duration of life from that time rarely exceeds six months or a year and is often preceded by mental deterioration and blindness. Speaking of the lesions in particular, on the other hand, in all their multiplicity of structure, manner of growth, situation, and possible complication, no definite statement can possibly be made. Their onset is always insidious, but it is self-evident that of tumors of the same size and type, one which occupies a silent area remains symptomatically dormant much longer than one which arises, for example, primarily in the precentral gyrus, or which early in its course has involved one of the cerebral nerves.

One thing is certain, that tumors are of much longer duration than is generally supposed, for the retrospect will often show that even malignant gliomata have doubtless been present for months or years before a diagnosis is made coincident with the appearance of general pressure symptoms, after which their course may be rapid. The symptoms from the onset are usually progressive, although there are notable exceptions, for tumors may enter a stationary phase and undergo retrograde metamorphosis. A tubercle may become quiescent, a glioma cystic or calcified; and some of the benign growths, as the endotheliomata, are of years' duration, leading in some instances to "autotrepanation" with relief of pressure, or they may even be a totally unexpected postmortem finding.

[1] *Deut. Zeit. f. Nervenheilk.*, 1907, xxxiii, 317.

There are other exceptions to the usual rule according to which symptoms slowly progress from bad to worse. Thus in cases with sudden or apoplectiform onset or when symptoms come on abruptly after a fall the entire story is apt to be a short one. Then, too, death is not uncommonly abrupt and unexpected, owing to a sudden acute œdema or to medullary anæmia from pressure, so that prognosis as to duration of life is most uncertain.

Then there remain "the unsolved riddles of pseudo-tumors," in the language of Bruns, so that the prognosis, in the presence even of a typical tumor symptom complex, need not necessarily be an utterly pessimistic one.

**Treatment.—Medicinal Measures.** — From a therapeutic aspect an intracranial tumor has in the past been regarded as one of the most hopeless of maladies. Interesting enough during life to the diagnostician, the matter has usually begun with the clinical and ended with the postmortem study. So little has treatment been able to offer that the physician has heretofore turned to the one possible therapeutic resource—antiluetic measures—on the chance that the lesion might be syphilitic.

The length of time which can justly be given over to the rigorous administration of mercury and the iodides in a case of brain tumor has been a much debated question. The period required for definite results has been placed by some as high as six months, and the dosage has been enormous. In this interval, or even in a much shorter one if the growth happen not to be luetic, the patient may rapidly fail and his choked disk, if one be present, go on to the atrophic stage. Even a person in health, put upon a similar treatment, would suffer greatly from nutritional disturbances, and much more the individual already upset by headache and vomiting.

Indeed, as a diagnostic test antisyphilitic treatment may be most misleading, for two reasons: one because with certain gliomata, as is well known, there may be a temporary amelioration of symptoms under these measures; and the other because the fibrous syphiloma, which gives the most characteristic tumor syndrome of all syphilitic processes, is exceedingly resistant even to massive doses of the usual drugs. Hence, even when the history and a positive serum reaction assure the diagnosis, the lesion must in the end be removed by operative methods if it be localizable, or if not, a palliative operation is necessary before the pressure symptoms are sufficiently relieved to allow the patient to receive full benefit from the drug administration. This too is the more urgently demanded in case a high grade of choked disk exists.

The typical basilar gummatous meningitis is relatively easy to diagnose and yields much more quickly to treatment than does a syphiloma. Fortunately the Wasserman or Noguchi reaction will usually serve to spare these patients much discomfort and their attendants long anxiety. It should always be used at the earliest moment.

To state this matter briefly, the writer would say that in the presence or absence of a positive serum reaction or a definite history of lues, antisyphilitic treatment deserves only a brief vigorous trial; if pressure symptoms have been outspoken and do not become distinctly ameliorated in the course of a few days, a palliative decompression should be performed and the treatment subsequently resumed should the diagnosis remain in doubt. One valuable criterion of the effect of the treatment and the need of early operation must rest with the condition of the eye-grounds, which

should be observed daily, for a choked disk may continue to advance, even though subjective discomfort is lessened.

In our series a syphiloma has been found and successfully extirpated in two instances. Both patients had been subjected to a long although unavailing course of treatment and were much run down. Most of the other patients had received similar treatment over long periods, oftentimes in the face of marked intolerance of the drugs.

The general treatment should be directed toward the best possible preservation of the nutrition by frequent small amounts of digestible food; for, apart from the incidental vomiting, these patients tend in later stages to lose weight rapidly. Cerebral congestion is to be avoided by proper position of the head, by ice caps, and by free daily evacuations. Alcohol is not tolerated. There is practically no effectual way of warding off the explosive attacks of vomiting; the headaches when insufferable may require large doses of narcotic drugs, antipyrine, phenacetine, codeine, combined with chloral, trional, or veronal for the night, and the hypodermic use of morphine as a last resort. Convulsions must be combated by bromides, or in desperate conditions by chloral or morphine—even by inhalation narcosis. Occasionally a threatened Jacksonian attack may be aborted by the prompt inhalation of a few drops of chloroform.

**Surgical Measures.**—It cannot be questioned that the first therapeutic indication for a tumor anywhere in the body which occasions, or is likely to occasion, symptomatic disturbances, is *removal*, provided this can be accomplished without risk and without leaving undesirable consequences; for the mere prolongation of life, whether through medicines or operation, provided it is not made worth the living, is, of course, undesirable. The second indication is the *alleviation* of symptoms, and inasmuch as the conditions which we must combat in the case of intracranial growths are almost entirely mechanical ones, they demand, for anything more than their temporary palliation, mechanical means of relief.

The first ray of hope in regard to the operative therapy for brain tumors was dimmed by von Bergmann's widely heralded views as to the practical hopelessness of these measures, owing to a long series of early surgical failures. But within the past few years new elements have served to put the subject on an entirely new and much sounder basis. Among these have been the greater precision in diagnosis and a greater perfection in technical operative methods. A further element of no little significance is the development of surgeons trained in neurological pathology and diagnosis—in other words, of operating neurologists; for it is inconceivable that these matters of handicraft can advance far or fast when the surgeon is merely the hand directed by the neurologist—a relationship comparable to that of the pre-Vesalian anatomist and his barber.

If medicine is still to be widely separated from surgery—and, in agreement with Sir Clifford Allbutt, it is a pity this divorce has ever come about—a system of medicine is no place for a discussion of surgical procedures. Nevertheless, so long as the physician continues to be the one first called upon to determine the future course of treatment for a patient with brain tumor, it is the more needful that he shall understand something of the operative methods employed for certain objects and the results which should be obtained thereby.

Familiarity and judgment in matters of medical handicraft are assets

of the greatest value, particularly for those who are disinclined themselves to practise it.

It is astonishing to learn how rapidly the number of these operations has increased. Allen Starr, in 1893, succeeded in collecting 97 cases; Chipault, in 1894, 135 cases; Oppenheim, in 1896, 140 cases; whereas today these almost represent individual figures. In our own series we have had under observation 130 cases of tumor or presumed tumor, of which number 109 cases have been subjected to operation or to repeated operations. In 67 of these 109 cases the histological and regional diagnosis has been certified at operation or at autopsy. Some of the remaining 32 cases surgically treated who remain well in consequence of palliative measures may possibly be suffering from so-called "pseudo-tumors."[1]

Surgical measures have two main objects in view: (1) To expose and remove the growth if that is possible; (2) to palliate the symptoms of pressure in case (*a*) of an impossible regional diagnosis, (*b*) of a lesion in a recognizable though surgically inaccessible region, (*c*) of irremovability on exposure, whether from excessive size, undue vascularity, or what not.

Tumor Extirpation.—Practically all of the successful cases before the year 1900 were confined to tumors involving the paracentral convolutions, but during the past ten years the number of successful removals from frontal, temporal, parietal, or occipital lobes and from the cerebellum has rapidly increased. The exposure of the hemispheres is no longer carried out by simple trephining, but by the reflection of a large osteoplastic flap with its base hinging on the thin temporal region. Thus, a wide area of exposure is secured through which the necessary manipulations can safely be carried out. As the making of the flap itself, from loss of blood, may be something of an ordeal, a two-stage operation, as advocated by Horsley, is often advisable. The tumor, if exposed, should not be scooped out, but should be removed, if possible, by careful, slow dissection, with painstaking hæmostasis. Of our 20 cases of attempted tumor or cyst extirpation in the cerebrum 10 have been successful, and the 3 operative fatalities have resulted from the effort to remove enormous encapsulated growths which, at an earlier stage would possibly have been easily removable.

A bone-flap operation is undesirable in cerebellar cases where a bilateral suboccipital exposure is essential. Subtentorial tumors are often favorable for removal. Thirty of these operations have resulted in three deaths. The lesion has been disclosed in 13 cases; considered irremovable in 2; removed or a cyst drained in the remaining 11, four of them being cerebellopontine endotheliomas.

Tumor Palliation (Decompressive Operations).—The unexpected relief which followed many of the earlier unsuccessful explorations gave birth to the idea of making a purposeful defect in the cranium and dura to

[1] The results naturally improve with greater experience, and in a series of 63 cases observed during the past ten months, all subjected to operation, there have been 8 operative deaths; marked palliative improvement in 30 cases; no improvement in 10 cases; extirpation of a tumor or evacuation of a cyst with recovery in 15 cases; making 24 per cent. of supposed permanent cures. Fifty per cent. of Oppenheim's cases succumbed to operative shock, hemorrhage, or other complications. In our series there have been 8 operative deaths, or 11 per cent.; and this does not allow for the fact that there have been over 80 operations in these 63 cases, the last and fatal ones having often been terminal procedures on patients after a long period of improvement following decompression.

give relief to pressure symptoms. Owing to the tendency of the brain under pressure to herniate through a defect and of the protruding portion to lose its function, it is desirable to make the opening over a silent area.

When it is desirable to end a fruitless exploration of the hemisphere with a palliative decompression, the osteoplastic flap may be removed *in toto*, or the subtemporal area of bone alone removed. In the case of presumed cerebellar lesions the palliative operation differs nowise from that necessary for exploration. We have had over 70 subtemporal decompressions for tumor, with two operative fatalities, both associated with a lumbar puncture. In most of the cases there has been immediate improvement, which in some instances (pseudo-tumor?) has promised to be permanent. In a few cases a tumor has finally given evidence of the location and has been removed at a subsequent operation.

These successful palliative measures have in many ways represented our chief advance in the past few years, for they are comparatively simple and free from risk. Their chief value lies in the prevention of blindness, which too often is the penalty of procrastination in cases of brain tumor. Certain lesions included under intracranial tumors, as well as actual tumors themselves, may prove to be more or less self-limited, with a tendency toward spontaneous healing, but unhappily loss of vision through optic atrophy is apt to occur in the interval of their activity; and to offset this danger, decompressive operations are particularly well adapted.

With the exception of tumors which have produced an internal hydrocephalus, alleviation of the pressure symptoms without increase in focal manifestations may be expected in most cases; and the subtemporal protrusion, if well protected by muscle, is very unobtrusive. Tumors situated in the pontine region, with or without hydrocephalus, are also an exception to this general rule, for the change of position of the parts, either after the establishment of a subtemporal or a suboccipital defect, is apt to exaggerate preëxisting local symptoms.

Punctures of the brain (*Hirnpunction*) and of the lumbar meninges as therapeutic measures, although successful in an occasional rare instance of draining an unsuspected meningitis serosa (Quincke) or the exceptional aspiration of a simple cerebral or cerebellar cyst, are nevertheless attended with such great risk of immediate death that they cannot be too emphatically condemned.

Parts of the brain, like the interpeduncular region, heretofore thought forever secure from surgical approach, have proved to be accessible in a number of ways, and tumors involving the hypophysis,[1] as well as primary hypertrophies of this gland, will in selected instances doubtless come to be regarded as surgical maladies.

So far as the cruder methods of surgery go, although we are still on the threshold, the door has nevertheless been opened, and a glance within shows room for an army of workers, but they will require a special kind of surgical training, far different from the old, the foundation stones of which must be laid in the neurological clinic and experimental laboratory for neuropathology.

[1] Archibald Church, *Jour. of the Amer. Med. Assoc.*, 1909, liii, 97.

# CHAPTER XI.

## HYDROCEPHALUS.

By HARVEY CUSHING, M.D.

"But as it never discovers itself till so much water is accumulated as, by its pressure on the sides of the ventricles, to disturb the action of the brain, we have little to hope from any medicine."—ROBERT WHYTT, *Observations on the Dropsy in the Brain.*

A PATHOLOGICAL increase in the amount of cerebrospinal fluid contained in the cranial chamber doubtless always represents a symptom of disease, not a disease of itself; although it must be confessed that pathology is at a loss to explain the undue accumulation of fluid in a certain group of cases, which, therefore, have been styled "essential" hydrocephalus. It may occur as an acute or chronic process; as one of congenital or acquired origin; as one due to inflammatory or purely mechanical causes; and finally the hydrops may be confined to the ventricular spaces or be found on the external surface of the brain.

As the cerebrospinal fluid, through retention or overproduction, is the factor chiefly concerned in the hydrocephalus, some account of the source and physiological action of this fluid is essential even to such a crude understanding of the condition as we possess.

Although possibly suspected by some of his predecessors, nevertheless Magendie, in 1842, first established beyond doubt the normal presence and rapid reformation of the fluid after withdrawal. Conjectures as to its physiological action have been many. Early writers believed in its association with the "pituitary membrane" and the nasal cavities—a view even accepted by Willis. It has been regarded merely as a surface lubricant, akin to the fluid found in the great serous cavities. Others have claimed that it serves solely as a "water-bed" to protect the brain from jar—a role which doubtless may be played by the relatively large collections of fluid in the subtentorial cisterns about the hind-brain.

Notable recent additions to our knowledge have come in two epochs: first, through the reawakening of interest in the fluid, as the result largely of Leonard Hill's physiological and Halliburton's clinical studies in the nineties, and second, through the introduction and widespread use of Quincke's lumbar puncture. Hill expressed the opinion that the fluid should be regarded as the lymph of the brain, but as Halliburton[1] has shown, it has marked chemical differences from a transudation, and must, therefore, be regarded as a true secretion, for the normal fluid is clear and colorless—like water—slightly alkaline in reaction, and of low specific gravity, 1005 to 1010; containing a mere trace of proteid (globulin) coagulable by heat, and also a reducing substance which is not sugar, but probably

[1] *Biochemistry of Muscle and Nerve*, Philadelphia, 1904, p. 70.

pyrocatechin or some derivative. Lymph, on the other hand, is like diluted blood plasma, with a specific gravity of 1012 to 1022, giving a test for sugar, containing proteids (albumin) in large amounts and coagulating spontaneously. Only in diseased conditions, such as inflammation and general paresis, does the usual character of the cerebrospinal fluid become greatly altered, with an increase of proteid, higher specific gravity, and in paresis a new substance, choline, due to the presence of degenerated nerve cells (Mott and Halliburton).

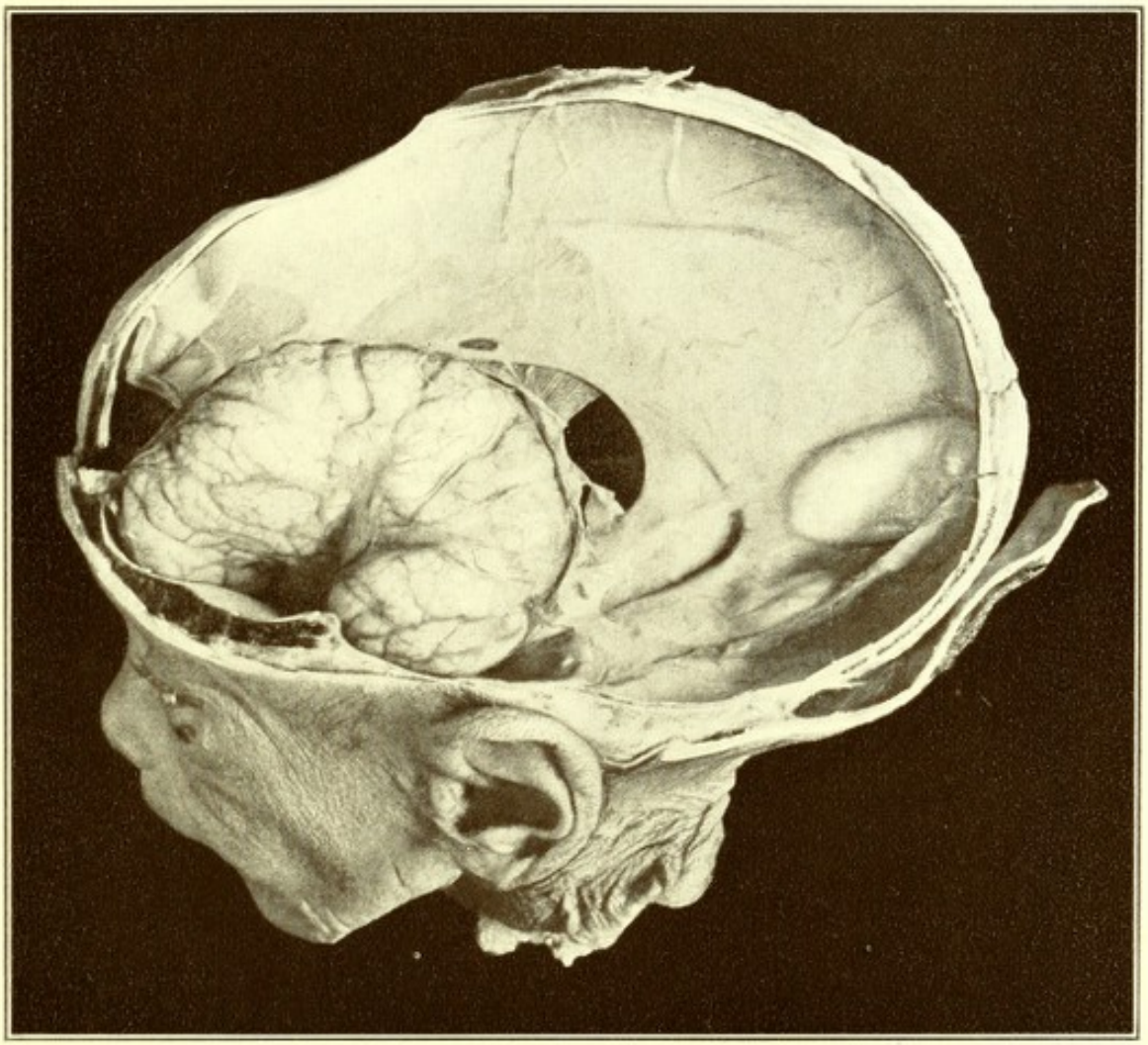
The fluid, in large part at least, is presumably the product of secretory activity of the ependymal cells lining the vascular choroid plexuses, although the precise function of these peculiar organs has not been conclusively established. Certain experiments have shown that their cells may be definitely activated; and the plexuses doubtless possess a definite glandular function.[1] The secretion, too, appears to be a fairly active and more or less continuous one, for the fluid, under certain circumstances, may form in large amounts. This is well illustrated by the rapid refilling of the hydrocephalic ventricles after tapping, but perhaps is best seen when, by accident or intent, an external fistula communicating with the subarachnoid space has been established, or in the remarkable cases of cerebrospinal rhinorrhœa such as are described in St. Clair Thomson's monograph—a condition characterized by a spontaneous escape of cerebrospinal fluid from the nose. No symptoms save loss of weight may attend such an escape of fluid continued over months, and an amount equal to half a liter may be collected in twenty-four hours. There doubtless may be a double source for the fluid, and although the larger portion comes through the ventricles, this may be added to by the cerebral lymphatics, which are said to follow the pial vessels and empty into the arachnoid spaces. It was Magendie's opinion that the vascular pia actually secreted the fluid.

It would seem, therefore, that there must be a definite circulation for the fluid; and presumably forming in large part in the lateral ventricles, it passes thence by the foramen of Munro to the mid-ventricular system and escapes into the subarachnoid spaces by way of the foramina of Magendie[2] and Luschka. From this point it bathes cord and brain, confined chiefly in the subarachnoid spaces, for only in certain pathological conditions does fluid appear in any considerable amount in the subdural space. From the subarachnoid spaces the fluid seems to escape directly into the circulation by way of the dural sinuses, rather than through lymphatic channels with the intermediation of glands before it reaches the venous system—another striking difference from true lymph. Some of it may possibly escape along the meningeal sheaths of the cerebral and spinal nerves—particularly the optic (cf. the condition in choked disk) and olfactory, but this is but a drop to the rapid escape by way of the sinuses. It was the view of Key and Retzius that the Pacchionian granulations played a certain role in this escape, but though it presumably occurs at the points where they are situated, it is probably not through their agency, for fluid may escape freely, by way of the arachnoidal connections with the dura, in infants, in apes, or in the canine in whom no Pacchionian granulations are present.

[1] Meek, *Jour. of Compar. Neurol. and Psych.*, 1907, xvii, 286.

[2] Certain unpublished investigations by Reford would make it seem that this is not a definite median opening, as described by Magendie, but rather a number of microscopic perforations in the roof of the fourth ventricle.

# PLATE XXI



Hydrocephalus Externus in an Infant with Normal Development of Skull and Meninges, but Defective Brain.

Even with so little an understanding of the cerebrospinal fluid circulation, it nevertheless can be seen how ventricular hydrops, the more important of the conditions we are to consider, can be due to obstruction of the channels of outlet from the ventricles; how infectious processes may easily occlude the small perforations in the roof of the fourth ventricle, or the valvular (if such they are) points of entry of the fluid into the sinuses; how thrombosis of the sinuses may lead to cerebrospinal fluid stasis, apart from the possible actual increase in the amount of fluid through venous congestion; how it is that the source of obstruction may be easily overlooked; and lastly, why many of the operations suggested for the relief of the condition have been based on erroneous principles of drainage.

**Hydrocephalus Externus.**—An abnormal collection of fluid outside of the ventricular cavities is rare, if we are to exclude certain pathological states accompanied by œdema of the brain and leptomeninges. It is misleading to refer to the "wet brain" of acute alcoholism, to the traumatic or angioneurotic œdemas, or to some of the cerebrospinal disturbances grouped under serous meningitis, as external hydrocephalus, although there is an excess of fluid in the meningeal spaces. It is confusing also to include here the *hydrops ex vacuo* or "compensation hydrocephalus," which is merely the natural collection of fluid required to fill the intracranial chamber when atrophic processes, such as occur in phthisis or general paresis, have led to a shrinkage of the brain.

A true external hydrocephalus, however, may be found in association with congenital anomalies, when an undeveloped brain is found filling, in part only, a cranial chamber of normal configuration (Plate XXI); and in rare instances it may occur in combination with a ventricular hydrocephalus when, through spontaneous perforation, the fluid has escaped into the subdural space. This, however, on physical principles would not be expected to occur very often, and surgical experience has shown that the distended and thinned hemisphere will not necessarily collapse and allow fluid to become extracerebral, even if a large opening be made into the ventricle through its thinned wall.

**Hydrocephalus Internus.**—It is customary to distinguish several varieties, particularly the so-called *idiopathic* form, regarded as a *primary* hydrocephalus, from those in which the condition is palpably *secondary* to some manifest lesion—the *acquired* forms. It would seem, however, that if there is any form of hydrocephalus which may justly be considered as a primary hydrocephalus, it is represented by the acute collection of fluid associated with the meningitis serosa of Quincke or with ependymitis; for here it is possible that we may be dealing with a condition in which fluid is excreted in excess of what may readily escape from the cranial chamber by the normal outlets. Nevertheless the resultant thickenings of ependyma when the acute process has subsided often lead to a persistent hydrops, so that even these states are customarily included with acquired or secondary hydrocephalus.

**Essential or Idiopathic Hydrocephalus.**—**Etiology.**—This sometimes occurs as a familial type, several members of a family being afflicted, so that hereditary influences play a part. The condition has been ascribed to fetal syphilis or rachitis, its association with congenital lues having been frequently observed. It is deserving of note, however, that the lower animals especially those in captivity, may have hydrocephalic offspring. Presum-

ably in most cases there exists some obstacle to the normal outflow of fluid from ventricle or cranial chamber, whether due to anomaly or disease. Heinecke, on the other hand, believes that the hydrops is produced by an abnormal increase in the amount of fluid secreted. Both factors possibly may be at work.

**Pathology.**—Meningeal thickenings suggestive of some prenatal inflammation are occasionally found matting together the structures, particularly about the roof of the fourth ventricle. More often, however, such an evident source of obstruction is absent, and the fluid from the ventricles can be withdrawn from the subarachnoid spaces by lumbar puncture, showing that if the process is actually an obstructive one the stasis in the fluid circulation must occur elsewhere, possibly at the foramina of entrance into the sinuses. The exact nature of these foramina is not known, and consequently the presence or absence of their occlusion cannot be demonstrated.

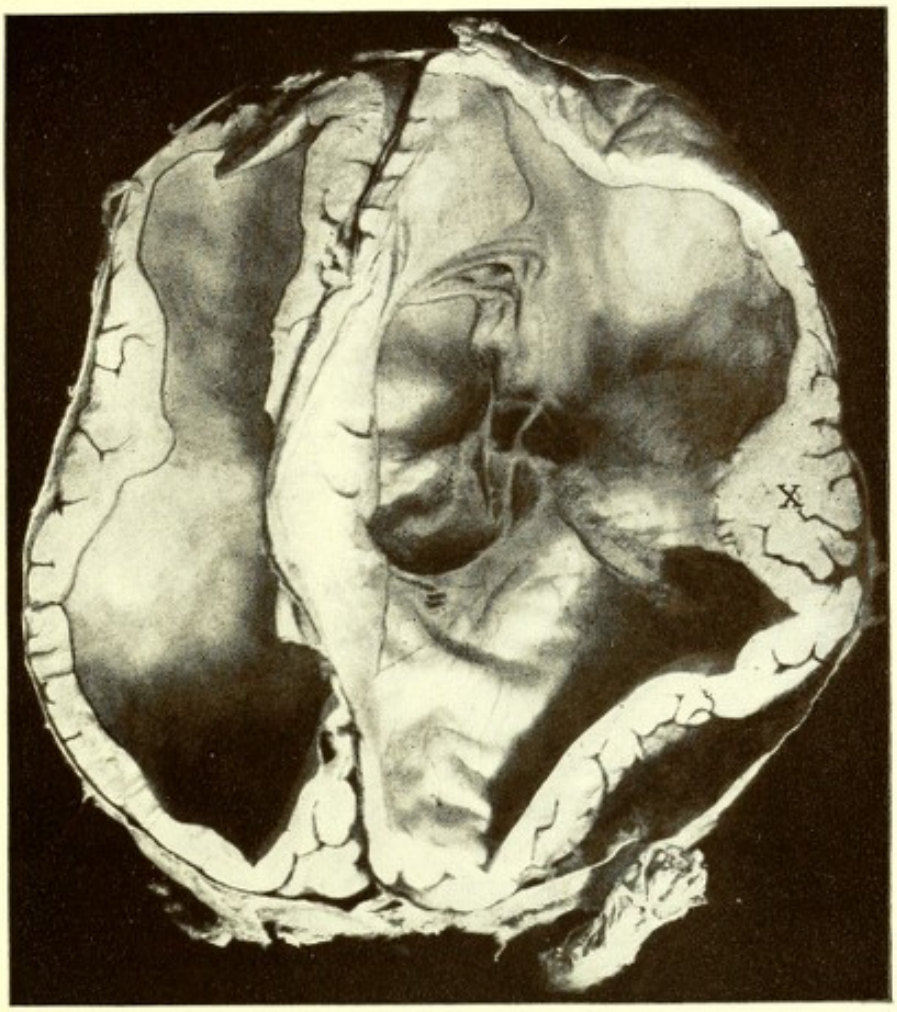
Ventricular hydrocephalus is the usual accompaniment of the structural anomalies, *cephalocele* and *spina bifida*, and it is a natural conjecture that these anomalies may possibly have been produced by some early disturbance with the normal circulation of cerebrospinal fluid which may have prevented the proper closing in by the mesoblastic tissues of the primitive nervous system. This could easily be brought about by some abnormal increase in tension of the fluid before closure was complete. Only when the obstructive lesion, whatever it may have been, has been recovered from or overcome by some operative measure can the spina bifida or cephalocele be removed without considerable augmentation in the degree of hydrocephalus already present.

"The appearance of the skull is striking, the large, thin, flaring cranial leaflets being perched on the small facial bones like the petals of a single water lily on its calyx. The bones themselves are thinned and atrophied in places, so that pericranium and dura may meet (craniotabes). There is apt to be an abnormal number of Wormian bones. The frontal, temporal, and occipital wings, instead of arising vertically from the base, overhang so that the squamous wing of the temporal, for example, becomes almost horizontal and overlies the zygoma in such a way as to almost obliterate the temporal fossa. The irregularities of the three basal fossæ are slowly pressed out, so that the sphenoidal and petrosal ridges separating them no longer project as prominent watersheds. The base, however, does not participate in the general enlargement which the other bones undergo. In extreme cases the brain itself becomes ballooned out by the gradual increase of fluid, so that in places it is of paper thinness. Not only the ventricles, but the communicating passages also are all widely dilated; the iter is distended, and the foramen of Munro may become large enough to admit three fingers (Plate XXII, Fig. 1). The corpus callosum may be stretched into a thin sheet and the much distended septum lucidum often gives way, leaving a direct communication between the lateral ventricles. The commissures between the basal ganglia usually remain intact, although they may be drawn out to a length of 2 cm. or more" (Plate XXII, Fig. 2).

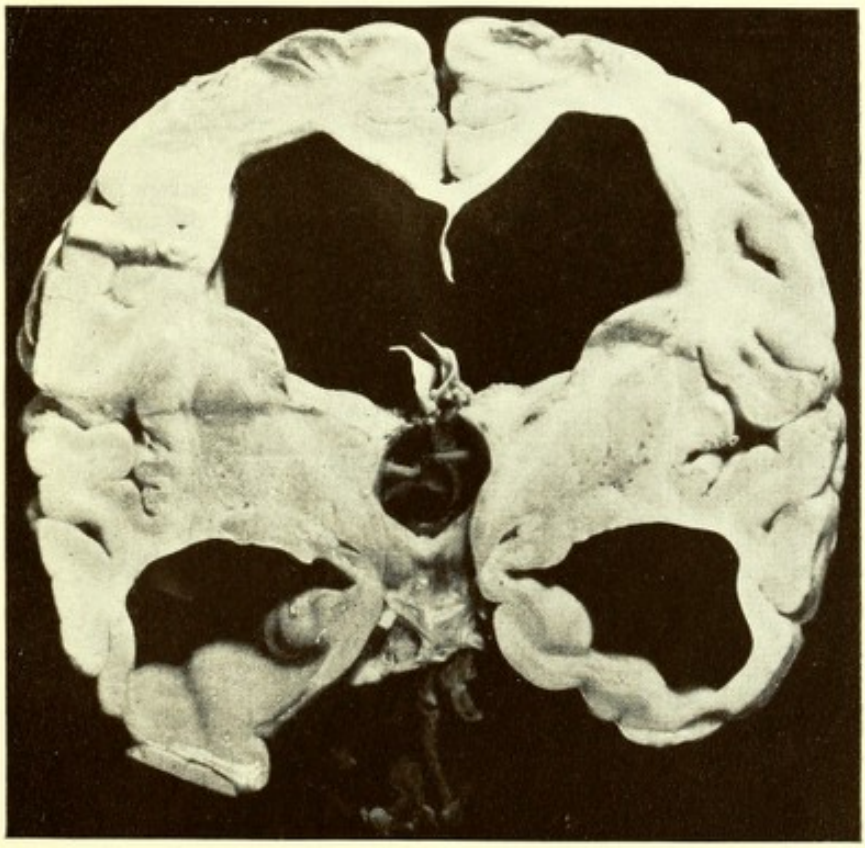
The amount of fluid may be enormous—three liters or more in extreme cases. It shows no abnormality either in saline, albuminous, or cellular elements.

**Course and Symptoms.**—In its more familiar and striking form the condition has been evident from birth, or at least has set in with insidious onset

# PLATE XXII



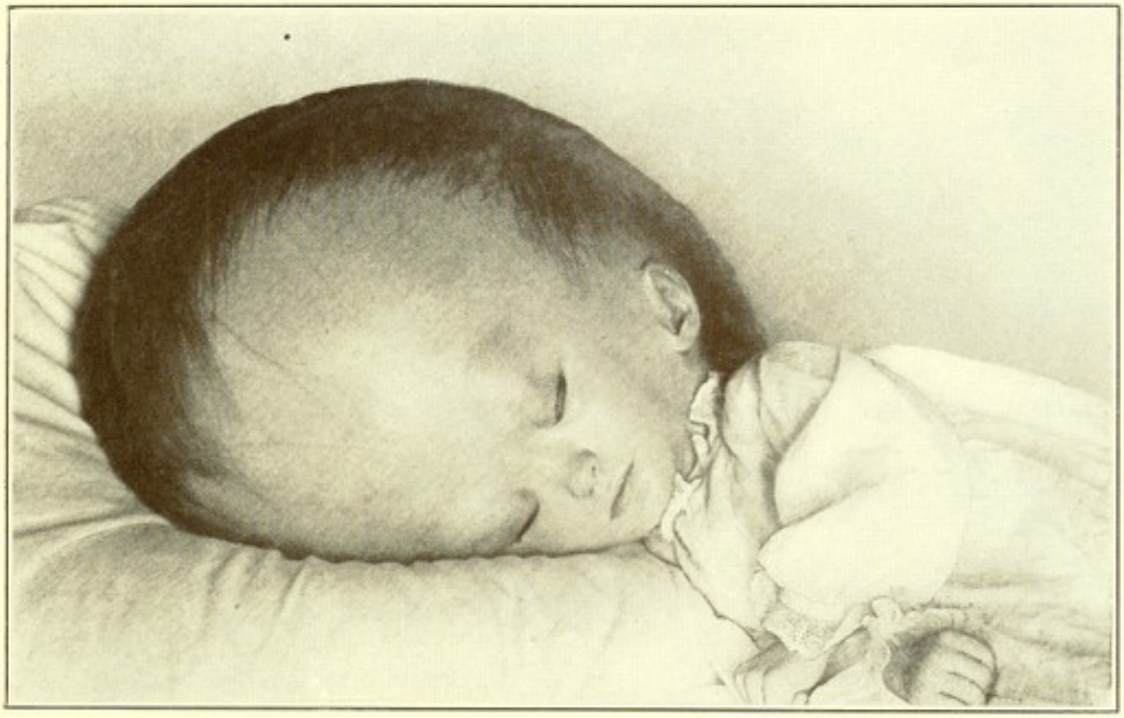

Advanced Grade of Chronic Internal Hydrocephalus, with extreme thinning of hemispheres, except at pyramidal tract (X). Note enormous size of foramen of Monro. Circumference of head, 70 cm.



Moderate Degree of Ventricular Distention in Chronic Hydrocephalus. Note rupture of septum lucidum; also great widening of iter, which is crossed by elongated commissure.

# PLATE XXIII

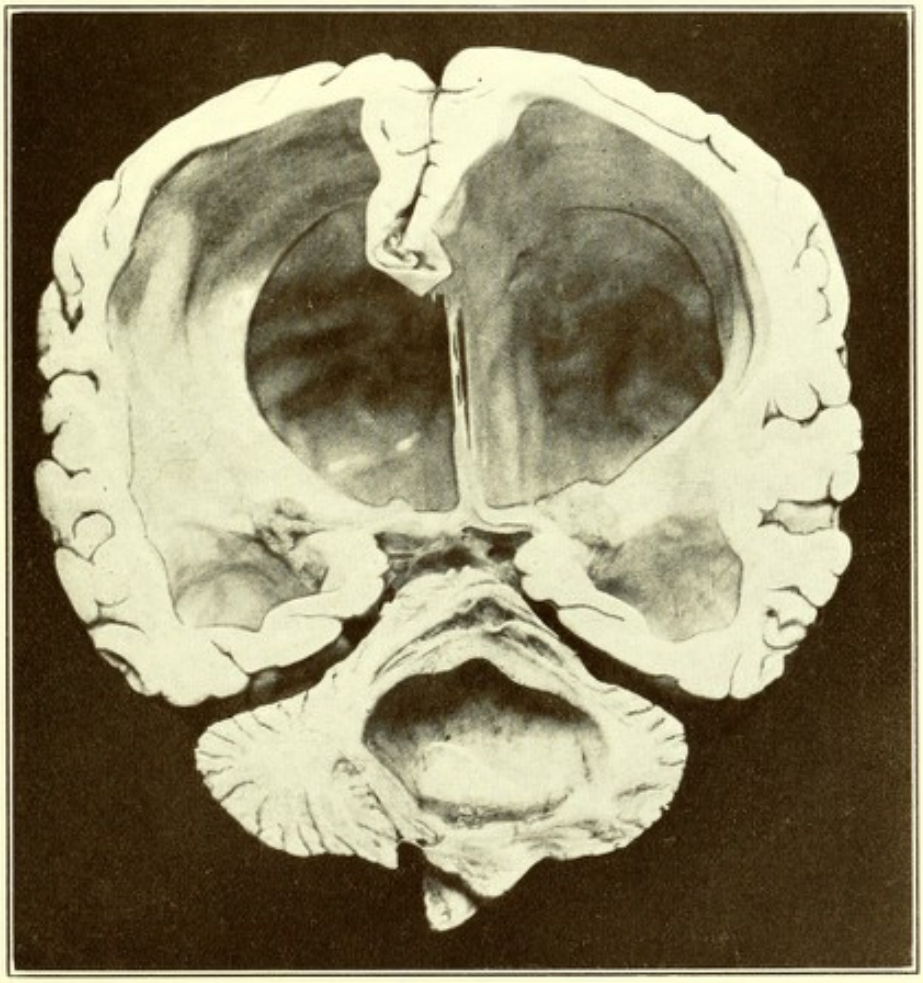
FIG. 1



Idiopathic Hydrocephalus.

An unusual degree of cranial enlargement. Circumference, 40 inches.
(Dr. Willard Knowlton.)

FIG. 2



Unsuspected Gliomatous Cyst of Cerebellum in an Infant.

Producing enormous ventricular distention from obstruction; regarded during life as idiopathic hydrocephalus.

in early infancy. Owing to the ready distensibility of the skull at this early age the condition when full-blown leads to external appearances which are unmistakable; "a cranial chamber enlarged out of all proportion to body and face; the enormous and tense fontanelles; the cranial bones of the vault widely separated, even when they should be together, so that the midparietal, midfrontal, parietotemporal and parieto-occipital sutures, bridged over by tense membrane, may all be open for 1 cm. or more; the bulging of the frontal, temporal, and occipital bones; the thin scalp, with its sparse hair and dilated vessels, which are thrown into prominence on account of the intracranial venous stasis; the expressional characteristics due to the tilting of the ears, with their wide intertragical notch and slit-like meatus; of the eyes, which are displaced by the downward bulging of the orbital roof and are covered chiefly by the lower lid, owing to the upward pull of the tight scalp upon the palpebral angles. The palpebral cleft is narrow, and the upper lid, when smoothed out, shows a network of dilated vessels."

When the enlargement is extreme (Plate XXIII, Fig. 1) these patients are unable to turn or raise the head, and pressure decubitus may result unless changes of position are frequent.

The circumference may often reach 70 and occasionally 100 cm., and the circumferential form is variable, depending upon the degree of tension and the habitual position assumed by the head, whether on the side, back, or at an angle.

The patients are, for the most part, feeble and poorly nourished, and even if the process becomes stationary after it has reached a high stage they have little resistance and are prone to succumb to intercurrent troubles. Should they live for some years they are apt to retain an infantile aspect, as well as infantile mental characteristics. Blindness, from optic atrophy, may occur, but this is not usual, and, indeed, even a low grade of retinal œdema is rare, for a pressure sufficient to produce an advanced degree of choked disk cannot well occur, as the cranium is so easily distensible. In some instances, even of the extreme type, adult life may be reached, with a certain feeble intellectual capacity combined with a traditional good nature. Rarely the process seems to have become arrested at an early age, with subsequent normal mental and physical development. Indeed, certain individuals with notable intellects are said to have shown evidences, post-mortem, of a low grade of hydrocephalus, presumably congenital.

**Diagnosis.**—There may be a close resemblance of a low-grade process to the rachitic head; the conditions of course may co-exist. When hydrocephalus is uncomplicated by this nutritional disturbance the diagnosis usually presents little difficulty. For though the cranium is large, craniotabes present, the fontanelles open, and the lines of the sutures flexible in rickets, the square shape of the head, the absence of signs of extracranial venous stasis, a fontanelle which is not tense, and rachitic changes in the bones elsewhere tend to distinguish the process. The chief difficulty lies in the differentiation of this so-called idiopathic hydrocephalus from the acquired form secondary to definite lesions. This is largely a matter of history; for in the essential form the process has been present from birth and is often associated with other anomalies; in the acquired form careful inquiry will show that a normal development has seemingly been interrupted in consequence of a cranial injury, a febrile illness, or some other unusual

incident. A choked disk and the hydrocephalic rigidities are much more common in the acquired form.

**Treatment.**—The indications are purely mechanical ones and should be directed toward the establishment of some form of permanent drainage, whether by the channels intended by nature to carry away the fluid or by the establishment of new ones. There are a great number of operative procedures in which the attempt is made to drain the fluid into the subarachnoid space, into the subaponeurotic space of the scalp, directly into the sinuses, into the abdominal cavity, into the retroperitoneal spaces, and so on. None of them have given particularly brilliant or uniform results, although there have been occasional recoveries even in advanced cases. Whatever method is used, it is necessary to determine first of all where the obstruction has taken place, for many of the older operative measures are futile if the ventricular and subarachnoid spaces freely communicate. Most of the older operations have been conducted on the view that occlusion of the neighborhood of the foramen of Magendie is the single possible lesion. Temporary withdrawal of the fluid by ventricular or lumbar puncture merely leads, as a rule, to its rapid reaccumulation, at times under greater tension than before, and is rarely of any lasting therapeutic benefit.

**Acquired or Secondary Hydrocephalus.**—This differs from the essential or primary form only in so far as it is apt to have a definite onset subsequent to birth often in association with a manifest causal agency.

**Etiology.**—Any form of obstruction to the normal circulation of the fluid or any condition which increases its production in excess of what may escape through the normal channels of outflow will produce this form. The more common agencies are tumors, meningeal or ependymal inflammations, and stasis of the venous circulation through extensive sinus thrombosis, or obstruction of the venæ Galeni.

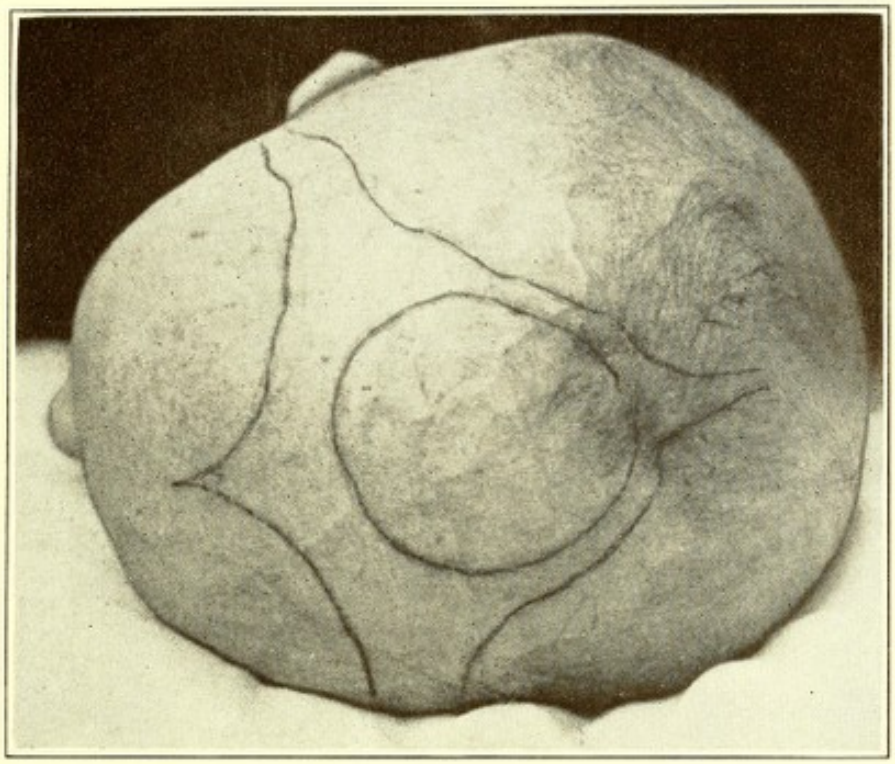
Tumors.—Tumors, particularly those confined in the subtentorial region, whether intrapontine, extrapontine, or cerebellar, may obstruct the ventricular iter or the foramen at the roof of the fourth ventricle, leading to a more or less symmetrical dilatation of third and lateral ventricles. When these conditions arise in early infancy there is a rapid secondary enlargement of the head, so that the usual pressure phenomena of tumor may be wanting, with external appearances the exact counterpart of those described under essential hydrocephalus. A tumor, a cyst, or a tubercle may be an unsuspected postmortem finding (Plate XXIII, Fig. 2). Diagnosis may be impossible without a local exploration—and not always then. A most valuable aid is the history of a previously normal state before symptoms began to appear.

The outward manifestations of idiopathic hydrocephalus, with separated sutures, wide fontanelles, etc., need not be limited to the secondary hydrocephalus of subtentorial lesions, for tumors of the cerebrum may occlude one or both foramina of Munro and produce a symmetrical cranial enlargement with general increase of pressure which serves to mask all focal symptoms. The tumors of themselves, under these circumstances of a yielding skull, may reach an enormous size.

Tumors which arise after the normal closure of the sutures and fontanelle has occurred may, through hydrocephalus, lead to a reseparation or diastasis of the sutures in the young, and even after adolescence, with an appreciable enlargement of the head, although never to such a degree as is seen

# PLATE XXIV

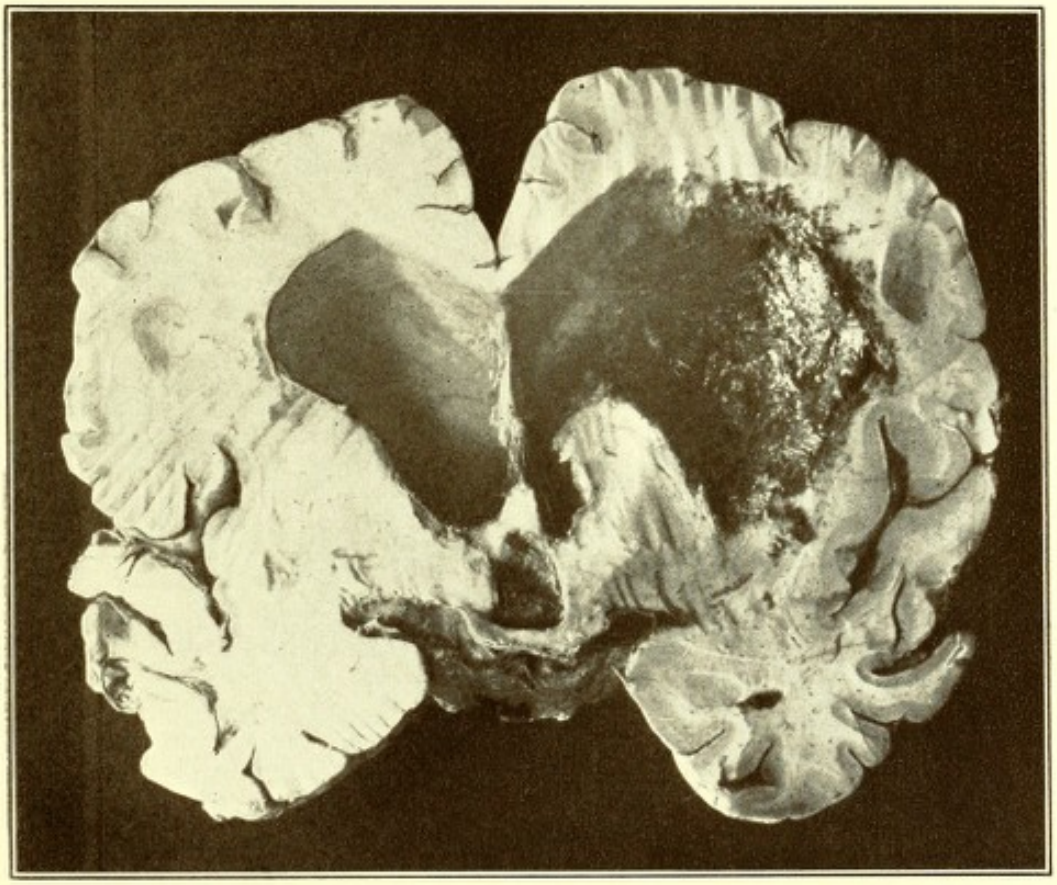
FIG. 1



**Showing Separation of Sutures and Widely Opened Fontanelle Containing Isolated Island of Bone.**

An infant with hydrocephalus secondary to meningitis; viable organisms in ventricles.

FIG. 2



**Section of Brain of Fatal Case of Capsular Apoplexy.**

In which a chronic hydrops ventriculorum with granular ependymitis was an unexpected postmortem finding.

in the infantile conditions. The "cracked-pot" percussion note which characterizes these conditions is an unmistakable evidence of a secondary hydrocephalus.

The abrupt onset of general pressure symptoms, in many cases of tumor which have been more or less dormant, is usually due to the occurrence of an obstructive hydrocephalus. This is particularly true of such benign growths as the cerebellopontine endotheliomata. The pressure symptoms, furthermore, are not necessarily constant, but may vary in intensity from time to time, depending on the degree of completeness of the obstruction. Certain lesions, like the cysticercus of the fourth ventricle, for example, may have a ball-valve action.

Inflammations.—A basilar inflammation, provided the exudate serves to occlude the minute foramina of exit in the roof of the fourth ventricle, leads inevitably to a hydrocephalus—the usual antecedent of death in these processes. Were it not for this mechanical obstruction and the resultant pressure phenomena meningitis would doubtless often be a self-limited disease.

*Cerebrospinal Fever.*—Attention has been called to the fact that in meningitis a pyo-hydrocephalus is often the terminal incident, for with the ventricular spaces not only infected but occluded the fatal termination of the malady is not remote. In infants, however, the ventricular distention need not give profound pressure symptoms, for the cranial chamber may rapidly enlarge (Plate XXIV, Fig. 1) and the process continue for months, organisms remaining viable in the cavities after they have died out from the meningeal spaces.[1] Naturally in these conditions antitoxic serum introduced by the lumbar route does not have access to the infected surfaces, and its introduction by a ventricular puncture is indicated.

*Posterior Basic Meningitis.*—In the longer enduring forms of infection, as in Barlow's posterior basic meningitis, these occlusions are common, and the disease may drag on for months with slowly enlarging head, persistent retraction of the neck, and hydrocephalic rigidities. Thus, it is not uncommon for the cases to run a chronic course, with more or less fluctuation of the symptoms according to the ease with which the pent-up fluid may escape. The occluded foramina of exit may become rechannelled in some cases, with no further advance in symptoms, although even with recovery a ventricular hydrops of this sort is apt to persist in some degree.

The ventricular complications of *tuberculous meningitis* differ nowise from those due to other infectious agencies. The patients usually die with, if not from, the secondary ventricular dilatation, graphically described for the first time by Robert Whytt.

*Ependymal Inflammation.*—A primary ependymal inflammation may similarly obstruct the passage of fluid by inflammatory closure of the interventricular foramina. This mysterious condition characterized by the presence of a granular thickening of the lining ventricular membrane occurs more often in the adult than in the young. The condition has no distinct symptomatology aside from that of an intracranial infection with definite pressure symptoms. It is not infrequently recovered from, judging from the number of instances in which traces of its former occurrence (a dilatation of the ventricle with ependymal granules) without history

[1] Cushing and Sladen, *Jour. of Exper. Med.*, 1908, x, No. 4, p. 548.

are unexpectedly brought to light when death has occurred from other causes (Plate XXIV, Fig. 2).

*Meningitis Serosa.*—The meningitis serosa of Quincke is a condition which lumbar puncture has enabled us to recognize. It is an acute process, with onset very similar to that of infective meningitis, but without the association of organisms—at least of those which can be cultivated. The condition may arise spontaneously or in the course of infectious diseases or intoxications. It is a particularly common sequel of otitis media and may be difficult to distinguish from infective meningitis or abscess. There is an excess of fluid under tension and usually a ventricular hydrops. A choked disk of low degree is commonly present in this as well as in the many definitely obstructive forms of ventricular hydrocephalus. In the absence of fever tumor may be closely simulated. Lumbar puncture shows the fluid to be under great tension, and its withdrawal may completely change the clinical picture for the better.

# CHAPTER XII.

## DISEASES OF THE PERIPHERAL NERVES.

By GORDON M. HOLMES, M.D., M.R.C.P.

The symptoms of disease of the peripheral nerves may be classified as they result from disturbance of the functions of those fibers which convey impulses to the muscles, of those which carry the sensory impressions centralward, and finally, of the fibers which are more immediately concerned with the nutrition of the tissues to which they are distributed, either directly or as part of the mechanism for the local regulation of the blood stream. There are, however, but few nerves in the body, excepting the cranial nerves, which are either entirely sensory or entirely motor. The consequence is that when any nerve is severely injured, both sensory and motor symptoms and probably, in addition, nutritive or trophic changes result. It is by the extent and distribution of these symptoms that the localization of the disease can be determined. If, however, the lesion or the disease of a mixed nerve is partial or incomplete, the functions of the different sets of fibers may suffer unequally; the general clinical experience is that in incomplete lesions, as those due to compression, the sensory fibers suffer much less than the motor fibers, or sensory symptoms may be absent, although the muscles supplied by the nerve are completely paralyzed. Lüderitz[1] has shown experimentally that the conductivity of the motor fibers is lost earlier than that of the sensory fibers when a mixed nerve is subjected to slowly increasing pressure. Evidence of trophic or vasomotor disturbance is, as a rule, little apparent in partial lesions.

**Motor Symptoms.**—The symptoms which immediately follow the complete interruption of the motor nerve fibers which supply a muscle are complete paralysis of both reflex and volitional contraction of that muscle, loss of its tone, and later, atrophy, changes in the character of its response to electrical stimulation, and finally, if recovery does not take place, contracture owing to secondary fibrosis. These features distinguish lower motor neurone or spinomuscular paralysis from paralysis due to disease in the upper motor or cerebrospinal neurones. In the latter condition movements, not muscles as such, are paralyzed, and the distribution of the paralysis does not correspond to the distribution of one or more peripheral nerves; secondly, the tone of the paralyzed muscles is increased, not diminished or lost, that is, the paralysis is spastic and not flaccid, and the reflexes which are dependent on the tone of the muscle, as the knee-jerk, are more active than normal instead of being abolished; thirdly, the paralyzed muscles do not atrophy, or atrophy only to a much less degree; and finally, there is no change in the nature of their response to electrical stimulation.

[1] *Zeitschr. f. klin. Med.*, 1880, Band ii, 97.

The amount of *loss of power* in the muscles naturally depends on the degree of the injury of the nerve; when there is complete interruption of its structure or function the paralysis is necessarily complete. When the lesion of the nerve is incomplete another factor must be considered, namely, the rate of its evolution, as when the disease is of sudden or rapid onset the symptoms may be considerably greater, for a time at least, than those produced by a similar lesion which has developed slowly. A muscle that receives its motor fibers from more than one nerve is naturally not completely paralyzed by even a complete lesion of one of them.

The *normal tone or tension of muscles* is dependent on the integrity of the peripheral reflex arc, which consists of the afferent fibers from the muscle that enter the cord by the dorsal spinal roots and terminate by synapsis around the cells of the corresponding motor neurones in the ventral horn, and of the peripheral motor neurones. When this arc is broken in any place the muscles immediately lose their tone. The atonia may be recognized by loss of the normal contour of the muscles, if this is easily visible; by their softness and flabbiness to touch, and by the lack or diminution of the resistance which they normally offer to stretching, and consequently the excess of mobility to passive movement of the joint at which they act.

The *deep reflexes or tendon-jerks* are dependent on the maintenance of the muscle tone, and it is by these so-called reflexes that this variety of tone in the muscles is most easily measured. Their disappearance or abolition is consequently an indication of the diminution or disappearance of the tone of the muscles concerned and not of their paralysis alone.

The knee-jerks may disappear early in the toxic affections of the peripheral nerves, before there is any demonstrable evidence of either motor or sensory paralysis; and during recovery from such conditions, or after regeneration of degenerated nerves, they may be absent when the recovery of power is apparently complete. The diminution or loss of tone as measured by the deep reflexes is the most sensitive guide we possess to any interference with the normal function of the peripheral nervous system.

*Atrophy of the muscles* supplied by the affected motor fibers is one of the most prominent symptoms; this is due to degeneration or regression of the muscle fibers owing to loss of the trophic influence which the motor nerves normally exert on them. It is recognizable by a diminution in the size of the muscle, and by its soft and structureless consistence. The latter symptom is frequently neglected, but it may prove valuable, especially in children, in whom it is not easy to determine the distribution of an atrophic paralysis by merely noting the movements, which cannot be performed; here by touch alone we may pick out the affected muscles. Muscular atrophy is generally recognizable within two or three weeks after a complete interruption of a motor nerve, and increases rapidly from this time until few if any fibers remain in the muscle. When the nerve lesion is incomplete the atrophy corresponds closely with the degree of paralysis; in other words, the conducting power and trophic functions of the peripheral motor nerves are lost together. When the lesion of the nerve is very slowly progressive the muscular atrophy is generally less prominent than the paralysis.

Coincident with the atrophy of the fibers of the wasting muscle an increase and proliferation of its connective tissue occur. If the muscular atrophy attains severe degrees, and if regeneration of its fibers does not soon set in, this new connective tissue slowly undergoes fibrosis and the muscle is con-

verted into a firm, inelastic band. The contracting fibrous tissue may produce contractures and deformities. Further, for the recovery of function it is necessary that the muscle fibers should regenerate, and regeneration must be evidently seriously interfered with by the presence of dense and contracted fibrotic tissue. The condition of the muscles and the changes they undergo are therefore as important as the pathological changes in the affected nerves.

One of the most valuable signs in disease of a peripheral motor nerve is *change in the electrical excitability of the nerve and of the muscles it supplies.* Muscular contraction can be normally obtained by either faradic or galvanic stimulation of the motor nerve fibers which supply it. Two or three days after section of a nerve the excitability of its peripheral portion is diminished, stronger currents are necessary to produce contraction of the muscles, and after a period of six to eight days the excitability is lost on the secondary degeneration of the nerve. The most characteristic changes are observed on direct stimulation of the muscle; they compose what is known as the *reaction of degeneration* (R. D.) and usually appear from the eighth or tenth day after section of the nerve. It must be mentioned that according to Sherren[1] increased excitability to the galvanic current is found only when the lesion of the nerve is incomplete. After some months, if recovery has not set in, the reaction of the muscles to the galvanic current diminishes slowly and finally disappears. As contraction can be obtained as long as there is any contractile tissue in the muscle, the disappearance of the galvanic excitability indicates the complete degeneration of all the muscle fibers.

In every case of peripheral paralysis it is important to test the electrical excitability of every portion of the nerve and muscle that can be reached. It occasionally happens when there is a local lesion in the nerve that muscular contractions can be obtained by stimulation below the lesion, but not from above it; the lesion may block the conduction of impulses without leading to secondary degeneration of the peripheral portion of the fiber. On the other hand, regenerating nerves may be excitable only from above the lesion; the regenerated portion is for a time unable to receive stimuli which it can conduct. This may depend on the absence of a myelin sheath (Erb). When the lesion is unilateral the excitability of both nerve and muscle may be compared with those of the normal side; when both sides are paralyzed the irritability should be compared with those of a normal person, or the tables prepared by Stintzing to show the normal limits of excitability in each nerve and muscle may be used.

*Symptoms of irritation of motor fibers* are much less frequently met with in diseases of the peripheral than of the central nervous system. Spasm, either tonic or clonic, and cramps, when due to irritation of the peripheral neurones, are generally reflex in origin and arise from excitation of the sensory fibers. Occasionally, however, intermittent or tonic spasm, in the form of rigidity, may result from irritation of a motor nerve by a neighboring focus of inflammation or by a foreign body, but even here it is difficult to exclude its reflex origin. True reflex spasms are much more frequent; the involuntary facial movements which often accompany trigeminal neuralgia, and even irritation of the cornea, are of this nature. The fixation of painful joints

[1] *Injuries of Nerves*, London, 1908.

by the tonic contraction of the surrounding muscles is also due to irritation of the sensory fibers which reflexly excite the corresponding motor neurones to excessive tonic activity; the sensory impulses may, if sufficiently intense, spread to wider reflex centres in the cord. The potency of sensory impulses from the periphery in the causation of spasm is illustrated by the severe spasms met with in strychnine poisoning and in tetanus; both these poisons only transmute an inhibitory effect into an excitation effect in the spinal reflex centres (Sherrington), and the spasms or convulsions are always directly due to a stimulus from the periphery. Cramp of a muscle may be occasionally due to influences that affect its fibers directly, as poisons like veratrine; venosity of the blood supply may also predispose to or cause spasms. This is probably the explanation of the cramps which frequently occur in states of severe exhaustion, or which may result from pressure on the larger arterial trunks of a limb.

Some forms of muscular atrophy are characterized by the occurrence of fibrillation or intermittent, more or less rhythmical wave-like contraction of some of the fibers of a muscle; this is more probably due to direct stimulation of the fibers than to excitation of the motor nerves. Myokemy, a condition characterized by constant undulating or wave-like contractions of the muscle fibers which changes from place to place, has been regarded as a symptom of abortive neuritis, but it is very rarely seen.

After complete division of a nerve and primary suture the time necessary for the return of power depends on the distance of the lesion from the periphery; Sherren lays down the rule for the upper extremities, that when a nerve is divided at the wrist perfect power may be regained within a year; but if at the elbow or in the plexus, not for two years. The muscles nearer the lesion regain their functions earlier than those distant from it. Recovery is slower after secondary than after primary suture. The muscular functions return much more rapidly after incomplete nerve lesions.

**Sensory Symptoms.**—When a sensory nerve or the sensory fibers of a mixed nerve are cut across we might expect to find absence of all sensation in the cutaneous and deep structures which are supplied by that nerve; but by the ordinary methods of testing this is rarely found, as the sensory loss which is revealed is generally much less extensive than the anatomical distribution of the nerve. This has been generally explained by assuming that the nerves overlap or anastomose, or that the neighboring end-organs take up sensibility from the anæsthetic region. The most careful investigations have failed to detect any change in sensation beyond the anatomical boundaries of the nerve which has been destroyed. The explanation has been afforded by the brilliant work of Head and his colleagues.[1] These have shown that the afferent fibers may be divided into three systems. The first system subserves *deep sensibility*, which is conveyed, as Sherrington[2] has shown, by the afferent fibers that run in the nerves from the muscles, tendons, and joints, and which consequently escape when only cutaneous sensory nerves are injured. Even when the muscular branches are involved this form of sensibility may not be quite abolished, as its fibers have wide anastomoses and often join the tendons and muscles high up in the limbs. Its function is the appreciation of pressure, of stimuli that produce deformation of

[1] *Brain*, 1905, xxviii, 99.

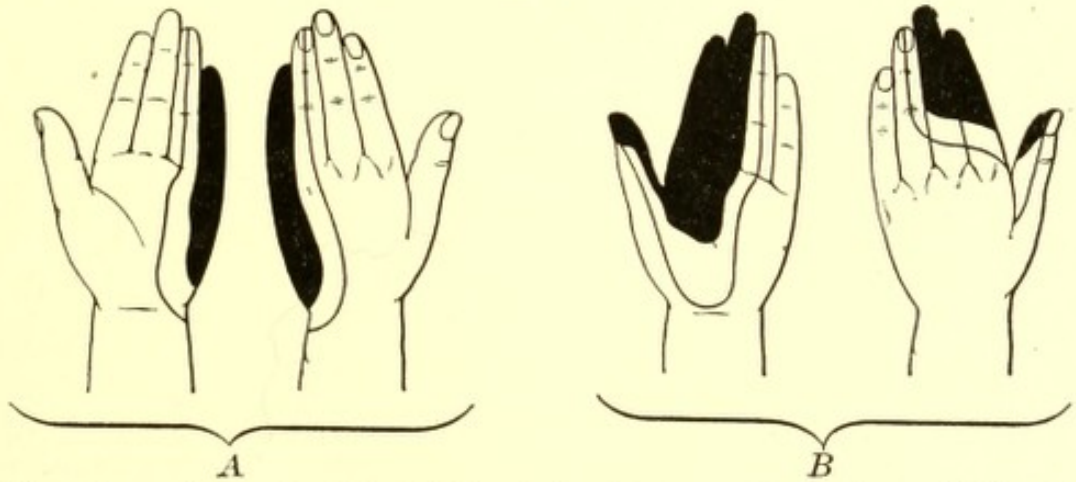
[2] *Phil. Trans. Royal Society*, 1896.

structure, and of any change in the position and condition of the joints and muscles. It is the system which subserves the sense of position. It is owing to the persistence of deep sensibility that the statement is often made that no sensory loss ensues on the section of a cutaneous nerve, as the area of distribution of the nerve remains sensitive to even the slightest pressure by a finger or pencil point; in fact, to those stimuli commonly used as a test of sensibility to touch.

The second system, to which the name *protopathic* is given, conveys painful cutaneous stimuli and the appreciation of the major degrees of temperature. The pain produced by a pin-prick or other means is not, however, localized, but radiates widely over the affected area and causes an unnatural amount of discomfort and an almost uncontrollable desire to withdraw the part from the stimulus. Although it is through this system that major degrees of heat and cold are appreciated, minor degrees of temperature cannot be recognized when it is alone present, and the appreciation of degrees of heat and cold is lost. The third system, which has been called *epicritic*, responds to light touches and to the minor degrees of temperature, which produce the sensations called "warm" and "cool." It is only when epicritic sensibility is present that the point of skin touched can be accurately localized, and that two points at a normal distance apart can be discriminated when applied simultaneously.

In the sensory disturbances that result from section of a peripheral nerve the different varieties of sensibility are lost according to their arrangement in these three systems, that is, sensibility to light touch and the minor degrees of temperature disappear together, and the appreciation of the major degrees of temperature with insensibility to painful cutaneous stimuli.

FIG. 4




*A*, loss of sensation produced by division of the ulnar nerve; *B*, that due to division of the median nerve. The areas of complete cutaneous insensibility are marked black; the skin insensitive to light touch and the intermediate degrees of temperature are enclosed by the line. (After Head and Sherren.)

When the condition of sensation on the hand is examined after section of the ulnar nerve it will be seen that there is complete loss of all forms of sensation only on the little finger and on a variable, but small, area on the ulnar border of the hand; that is, it is only here that both the protopathic and epicritic sensibilities are absent (Fig 4), but on the rest of the cutaneous

distribution of the ulnar nerve light touch and the intermediate degrees of temperature, the epicritic sensibilities, cannot be appreciated, and although painful stimuli may be recognized they cannot be accurately localized, but radiate widely and give rise to unnatural discomfort. It is evident that though there may be a considerable overlap between the ulnar and median nerves of the fibers which conduct protopathic sensibility, the loss of epicritic is practically limited by the anatomical boundary of the nerve distribution. With other nerves there may be an overlapping of epicritic sensation too. When the fibers are injured in a plexus the area of loss of protopathic sensibility may almost equal in extent that of the epicritic, while if it is due to a lesion of the dorsal spinal roots, the area insensitive to prick may actually exceed that insensitive to light touch. The more closely a peripheral nerve represents the supply of one or more posterior roots the more nearly will the loss of protopathic coincide in distribution with the loss of epicritic sensibility.

When a nerve is not completely divided, or if only its functional continuity is affected, as by bruising or compression, the condition of sensation is very variable. Sometimes every form of sensibility is lost for a time, but often pain perception remains intact and loss of epicritic sensibility may be the only sign of the injury; or after slight injuries there may be no absolute loss of sensation even to cotton wool, though the patient may be conscious of an altered sensibility in the area of the nerve.

The recovery of sensibility in the area of a divided nerve which has been sutured and is regenerating takes place in a definite and constant manner. The first change, generally observed after about two to three months, is a gradual diminution of the area of total analgesia commencing in the proximal parts of the area, and recovery of appreciation of major degrees of temperature, while the area of insensibility to light touch remains unaltered. At this stage, generally completed in about six months, there is still absolute loss of epicritic sensibility, although all the forms of protopathic sensation have recovered. The diffuseness and radiation of pain produced by a pin-prick, or rough handling, or a blow is so unpleasant at this stage that the complete absence of sensation may appear preferable to the patient. The return of sensibility to light touch and the minor degrees of temperature, as well as the power of discriminating between one and two points and of accurately localizing stimuli, rarely commences earlier than six months, no matter how favorable the nerve union may be, and is seldom complete within a year; but even for years careful examination may detect abnormalities in the sensibility of the area which had been anæsthetic.

After incomplete nerve lesions, on the other hand, the appreciation of pain and of light touch return at approximately the same time; it may be taken as an absolute rule that if the simultaneous recovery of protopathic and epicritic sensibility is observed within a few months of the injury the nerve has not been completely divided. Recovery of function is also much more rapid than after complete division of the nerve. It commences at a date which varies with the distance of the injury from the periphery, from about three weeks at the wrist to six months in the plexus, and also with its degree.

Symptoms of irritation or perversion of function of sensory fibers are often a prominent feature of disease or injury of the peripheral nerves, especially when the lesion is only partial or when the central stump of a divided nerve is involved in dense scar tissue. Pain is the most important of these subjec-

tive symptoms. Irritation of a nerve by pressure or disease may give rise to severe pain; this is undoubtedly due to the excitation of the sensory fibers which lie in the nerve at that level by the lesion; the abnormal excitation thus produced is conveyed by the ordinary paths to the higher sensory centres of the cerebral cortex, where it reaches consciousness; its origin is, however, misinterpreted and is referred to the sensory end-organs of the irritated fibers, even although the area in which they lie be completely anæsthetic—anæsthesia dolorosa. Pain of this origin is often paroxysmal, and is generally sharp, burning, or darting; at times, however, it is of a dull aching or boring character; it may be referred to the skin or the deeper structures of the limbs.

*Hyperalgesia* is met with in regions in which epicritic sensation is lost, while protopathic persists, but it often follows partial lesions where there is little or no disturbance of sensation. It not infrequently extends over the boundary of the anatomical distribution of the injured nerve, and is often associated with severe spontaneous pain.

Weir Mitchell has given the name *causalgia* to a condition of severe spontaneous pain associated with hyperalgesia and tenderness, and often with trophic disturbances. It is most frequently seen after bullet wounds; it never results from complete interruption of the continuity of the nerve, and always disappears immediately the nerve is cut across for secondary suture. In these cases it is probable that normal stimuli of peripheral origin are augmented in their passage through the injured region, or it may be that the sensory end-organs become hyperexcitable owing to a disturbance of their connection with their trophic centres. Hyperalgesia appears rarely if ever immediately after the nerve injury, generally not for a period of a few weeks.

Various *paræsthesias*, or a feeling of loss of feeling, or of weight or pressure, or, it may be, a sense of warmth or coldness, are frequently due to affection of the peripheral nerves which supply the region to which they are referred; they are generally, but not always, associated with some loss of sensibility. They are, on the whole, met with more frequently in the toxic degenerations of nerves than after traumatic or local lesions; they are probably due to a slight but persistent irritation of the sensory fibers. The most frequent form is generally described as a numbness or deadness of the part, but from the discomfort it causes it is evident that it is more than the consciousness of loss of feeling; it is rather an abnormal positive symptom, which may, even when there is no objective sensory disturbance, seriously interfere with the functions of the part; if it be the hand, for instance, everything it touches may be felt, but the sensation obtained is not natural; it is as though the object were felt through a glove. Even when paræsthesias are due to the local injury of a nerve, they are rarely referred to the whole area of its anatomical distribution; in the limbs they tend to be more intense peripheralward.

Occasionally the irritation of one of the terminal branches of a nerve gives rise to pain extending over its whole protopathic distribution, or of that of its roots, which may be accompanied by hyperalgesia, or by spasm or contractures of the muscles supplied by it. Neurotomy of the irritated branch gives instant relief.

**Trophic and Vasomotor Symptoms.**—Nutrition and conservation of all tissues of the body are to a certain extent dependent on the integrity of their connection with the central or autonomic nervous systems, and

when this is interrupted the tissues isolated from nervous influence may undergo certain changes. The most prominent of these is the atrophy of the muscles which follows the degeneration of the motor nerve fibers which innervate them, but the skin, subcutaneous tissues, and even bone may also undergo structural alterations.

It was for long discussed whether the influence of the nervous system on the tissues is exerted through special trophic fibers, or through the ordinary motor, sensory, or vasomotor nerves. The muscles are certainly dependent only on the integrity of their motor fibers, and the existence of special trophic fibers to other tissues has not been proved. The vasomotor nerves must, however, have a considerable influence on the nutrition and growth of the tissues; paretic vasodilatation follows section or injury of a nerve, and although there may be hyperæmia and a slight elevation of the local temperature for a time, the part soon becomes cold, congested, and even cyanosed, owing to the slow circulation through the dilated vessels; and its inactivity leads to lymph stasis.

It seems probable that the trophic centres for the skin and other tissues lie in the spinal ganglia, and that the centrifugal conduction of trophic influence is a function of the sensory fibers; or more probably that the normal nutritional equilibrium is in some way determined by the sensory nerves. The strongest evidence of the trophic function of the spinal ganglia is that the acute changes in the skin which characterize herpes zoster are directly due to disease of them. The innervation of the bloodvessels and of the sweat and sebaceous glands takes place through the sympathetic system; in the limbs, at least, these sympathetic fibers are intimately associated with those of motion and sensation.

Trophic changes in the skin vary with the rate of evolution and the degree of the nerve lesion; the more acute it is the more likely are trophic changes to be prominent. They may be met with either in areas of total anæsthesia, or associated with only partial disturbance of sensation. In areas of total analgesia the skin becomes thin and atrophic, and, especially in the hands and feet, inelastic and tightly stretched over the part; owing to the cessation of sweat and sebaceous secretion it is usually very dry and may be either glossy and shiny, or scaly when desquamation of the superficial layers of the epidermis is delayed. In this condition it is very liable to injury, and ulcers often develop, which, as a rule, first appear as blisters or bullæ; these often appear to be the result of the trophic disturbance alone, as they may occur apart from any assignable local cause, or follow a slight bruise. Owing to the loss of sensation the skin is liable to suffer from neglected or unobserved injuries, and to this many of the trophic changes have been attributed. Head[1] has observed that these cutaneous affections are generally co-extensive with the area of complete analgesia, and that they disappear with the return of protopathic sensibility.

Another form of cutaneous trophic disturbance which follows incomplete lesions of nerves, and is always associated with spontaneous pain and hyperalgesia (causalgia), was first accurately described by Paget[2] and Weir Mitchell, and by the former termed "glossy skin." It is seen chiefly on the fingers and hands; the skin becomes "smooth, hairless, almost devoid of wrinkles, glossy pink or ruddy, or blotched as if with permanent chilblains" (Paget).

[1] *Brain*, 1905, xxviii, 251.

[2] *Medical Times and Gazette*, 1864, i, 331.

The subcuticular tissues shrink, and the skin, which appears tightly drawn over them, is often cracked and the epithelium is partially lost, so that the cutis is exposed in places. According to Weir Mitchell, this condition is often attended with vesicles.

Changes in the growth and condition of the nails often follow nerve injuries. Retardation of growth has been frequently described, but Head has shown that this is independent of sensory disturbance, and that want of movement owing to paralysis or the fixation of the limb is the main factor. Weir Mitchell observed remarkable alteration in the nails associated with glossy skin, which consisted of curving on their long axes, extreme lateral arching, and sometimes a thickening of the cutis beneath their extremities. In other cases the nails may be thickened, corrugated, and furrowed, or thin and irregular, and lose their normal gloss.

When several nerves of a limb are damaged the bones are liable to become fragile, and if it occurs in early life their growth may be retarded. Acute and extensive nerve lesions may lead to swelling of and effusions into joints in the paralyzed regions. Another form of more chronic joint change occurs chiefly in the fingers and wrists; it commences with pain and periarticular thickening, and may ultimately produce ankylosis.

**The Treatment of Local Nerve Lesions.**—This naturally depends on the nature and the degree of the lesion; but there are certain general lines which should be invariably followed. In every case the chief aims should be to maintain the nutrition of the parts supplied by the injured nerve, to keep the paralyzed muscles relaxed, and to prevent the occurrence of contracture in their antagonists.

If the nerve has been completely divided, its two ends should be sutured together at once. The prognosis when primary suture is possible is good, but it depends to a certain extent on the nerve injured, the nature of the lesion, the condition of the wound, and the distance of the injury from the periphery; the muscular functions generally return within nine months, but sensation is rarely perfect for two or three years. Often, however, the nerve injury is not observed, or the patient does not come under treatment until after a considerable time, then secondary suture must be performed. The outlook in these cases is not so favorable as after immediate suture, but the time after the injury at which the operation is undertaken, certainly up to three years, seems to have little influence on the recovery (Sherren).

Much depends on the condition of the muscles; when these have entirely lost their galvanic irritability, complete recovery is probably impossible, and according to Warrington and Jones,[1] when the paralysis has lasted several weeks return of full power cannot be expected unless the paralyzed muscles have been kept relaxed. It is important that not merely should all scar tissue around the ends of the nerves be removed, but that these should be freshened up and brought into accurate apposition. It is occasionally impossible to bring the divided ends together; then Assaky has recommended imposing catgut threads between them to form a scaffold along which the regenerating axis-cylinders may grow, and Vanlair has introduced the method of interposing a tube of decalcified bone or a piece of a small artery or vein between them for the same purpose. Nerve transplantation, however, seems to give more favorable results in such cases, but, as Merzbacher[2]

[1] *Lancet*, 1906, ii, 1644.

[2] *Neurologisches Centralblatt*, 1905, xxiv, 150.

has shown, the portion of nerve inserted can take an active part in regeneration only when it is taken from the same animal, or an animal of the same species; if taken from another species, it immediately necroses and can consequently at the most act only as a scaffold. In other cases it is preferable to anastomose the peripheral end of the paralyzed nerve into a neighboring healthy trunk, or end to end with a flap raised from the sound nerve. In every case the exact condition of the injured nerve should be examined under an anæsthetic if there is complete reaction of degeneration in the muscles it supplies after fourteen days.

Tendon transplantation is occasionally necessary, especially when there is such complete degeneration of the paralyzed muscles that recovery after secondary suture of the nerve cannot be expected. The results obtained are often favorable. It is interesting that after nerve anastomosis and tendon transplantation nerve centres in the cord can acquire new functions or adapt themselves to new conditions.

The first and most important aim is to keep the paralyzed muscles relaxed and prevent shortening and contracture of their antagonists. If the extensors of the wrist and fingers are paralyzed, their joints must be kept fully extended by a splint along the forearm and hand; if it is the flexors of the forearm, the elbow should be held flexed in a sling. But the splint or other apparatus used should be removed frequently, and massage and passive movements of the paralyzed part systematically carried out.

The next aim should be to maintain the paralyzed muscles in as good a state of nutrition as is possible, and for this electrical treatment is usually employed. If the paralysis is not complete, and if some excitability to the interrupted current remains, faradism may be employed, but it is of little use if the muscles will not react to it, although, according to Mann,[1] regular treatment raises the excitability of both nerve and muscle and increases the flow of blood and lymph. Galvanism is more effective when the muscles will not react to faradism, but the current employed should not be too strong. Remak recommends the application of the kathodal electrode over the seat of injury where the lesion is incomplete, with the anode on the muscles or on an indifferent point, and the application of a relatively weak current without break for ten minutes or so at a time. Friedländer's[2] experiments undoubtedly showed that the regeneration of the nerve may be hastened by this method of using the constant current.

Systematic and energetic massage is probably more effective than any form of electrical treatment, and should be combined with it, but the region of the nerve injury should not be forcibly handled. Passive movements are always advisable; and the patient should be encouraged to make efforts to use the paralyzed muscles as soon as any return of power sets in.

## DISEASES OF THE SPINAL NERVES.

**The Cervical Plexus.**—Owing to its deep position among the muscles of the neck, the cervical plexus is rarely injured or affected by disease. The only one of its branches which is of clinical importance is the *phrenic nerve.*

[1] *Centralblatt f. Nervenheilkunde,* 1897, xx, 1.
[2] *Deutsche med. Wochenschr.,* 1896, xxii, 414.

Its paralysis may be due to a lesion in the ventral horns of the cord at the level of its origin, to an intraspinal hemorrhage or tumor, or to syringomyelia. Duchenne first described it in progressive muscular atrophy. It is most frequently caused by involvement of the third and fourth cervical roots in meningeal or vertebral disease, especially in spinal caries and syphilitic pachymeningitis. Owing to its deep position the nerve trunk is relatively immune from trauma, but it is occasionally injured by wounds or operations. Within the thorax it may be compressed by tumors or by aneurisms. Unilateral paralysis can be often attributed only to a local neuritis, perhaps following exposure to cold. Bilateral palsy sometimes occurs in multiple neuritis, especially in that form which follows the acute infective diseases, in lead poisoning, and in tabes dorsalis (Gerhardt).

The diaphragm is one of the most important of the respiratory muscles; when the patient is at rest its inactivity may give no trouble, but dyspnœa is easily produced by exertion or when respiration is interfered with by disease of the lungs or pleura. If bilateral paralysis sets in suddenly there may be considerable dyspnœa and even cyanosis for a time, but it is quickly relieved by the activity of the accessory muscles. Several cases have been recorded in which accidental injury of one phrenic nerve during operations in the neck has led to a fatal result, but Schröder and Green,[1] who have recently analyzed these cases, came to the conclusion that death was generally due to some other cause.

When there is complete bilateral paralysis the upper part of the abdomen is no longer protruded with each inspiration, but sinks in as the diaphragm is drawn upward by the negative pressure in the thorax; on palpation the descent of the liver and spleen can be no longer felt, and the patient is unable to expand the abdomen by taking a deep breadth. The movements of the thorax are consequently often increased, and the excessive movement of the lower part of the thoracic cage may draw the abdominal wall tense; this must not be mistaken for the effect of the descent of the diaphragm. If the paralysis is not complete the protrusion of the abdomen in inspiration can be easily resisted by pressure. Owing to inability to take a deep breath there is difficulty in coughing, and the patient cannot spit out with the normal force nor sneeze properly, and as the intra-abdominal pressure cannot be increased there may be difficulty in defecation. Pulmonary symptoms are the most important complication of diaphragmatic paralysis; owing to the relative immobility of the bases of the lungs they may have become dangerously congested, especially in patients confined to bed.

The symptoms of unilateral paralysis are generally slight and frequently escape observation. The movements of the diaphragm may be observed directly by the fluoroscope. Finally, the electrical excitability of the phrenic nerves may be examined; they are easily stimulated in the neck between the sternomastoid and scalenus anticus muscles, and above the omohyoid. The phrenic nerves also convey sensory fibers to the pleura, pericardium, and diaphragm, but sensory symptoms have been rarely referred to their disease; in a few cases, however, patients have complained of pain in the mediastinum and in the region of the diaphragm.

The *diagnosis* of diaphragmatic paralysis is not always easy; the examina-

[1] *American Journal of the Medical Sciences*, 1902, cxxiii, 196.

tion of the electrical excitability of the phrenic nerves may be very important when the other signs are not conclusive. In the majority of the cases the condition is only part of a general peripheral neuritis, or of poliomyelitis. Otherwise, bilateral paralysis is generally due to some disease in the spinal cord or meninges; it can then be rarely an isolated symptom. The vertebral column should be carefully examined, and the existence of tumors in the neck and mediastinum excluded. Paralysis must be distinguished from immobility of the diaphragm owing to diaphragmatic pleurisy or peritonitis, or to large pleural effusions. Or the diaphragm may be weak owing to secondary degenerative changes. Acute fatty degeneration of its muscle fibers frequently occurs in diphtheria; fatty degeneration may enfeeble, but rarely paralyzes the action of the muscle.

**Treatment.**—Treatment should be directed to the removal of the cause of the paralysis if it is possible; but it is rarely so unless it is due to an operable tumor either in the vertebral canal or neck. Otherwise its chief aim should be to avoid all pulmonary complications and to save the patient from exertion. If there is reason to suspect a local neuritis, warm fomentations and counter-irritation may be applied over the lower part of the anterior triangle of the neck. Electricity, especially the faradic current, has been employed with apparently some effect; the one electrode should be placed over the phrenic nerve immediately behind the sternomastoid muscles in the neck, the other over the epigastrium.

**Long Thoracic Nerve.**—Isolated paralysis of this nerve is rare, Steinhausen[1] has been able to collect records of only 29 pure cases, it is more frequently found in association with palsy of other muscles of the shoulder-girdle, and it is often seen in progressive muscular atrophy and in the muscular dystrophies. The nerve may be injured in the neck by blows or perforating wounds, or by direct pressure from a heavy weight carried on the shoulder; and it occasionally happens that it is bruised by the forcible contraction of the scalenus medius muscle through which it passes, or by excessive stretching of the nerve when the arm is raised above the head, as in painting a ceiling, climbing hand over hand, or hanging suspended by the arms. In the axilla it may be injured by a perforating wound or by operation. Paralysis of the serratus magnus has also been observed after infective disease, and with acute arthritis of the shoulder joint. In some cases it has been apparently due to a local neuritis following exposure to cold. It is much more frequent in strong muscular men than in women and owing to its usual causes it is more common on the right than on the left side. Its paralysis produces very little deformity while the arm is at rest; the scapula may stand slightly higher than normal, with its inferior angle slightly approximated to the vertebral column and separated from the chest wall. When the arm is moved forward into the horizontal position or pressed forward against resistance, the scapula, no longer held against the thorax by the serratus, is rotated on its vertical axis, so that its vertebral border projects backward and appears winged. This deformity is almost pathognomonic of serratus palsy. There is also difficulty in raising the arm above the horizontal, as while the arm is normally abducted from the side as far as the horizontal level by the deltoid

[1] *Deutsche Zeitschr. f. Nervenheilk.*, 1900, xvi, 399.

alone, its further elevation is brought about by rotation of the scapula, chiefly by the serratus magnus. The latter part of the movement is consequently lost when this muscle is paralyzed, but not invariably, as it can be sometimes carried out by contraction of the middle fibers of the trapezius. There is often diminished muscular power in the whole arm, which disappears when the scapula is firmly bound to the trunk. Slight scoliosis is frequently observed in cases of serratus palsy; it is probably due to an attempt to reëstablish the equilibrium upset by the malposition of the shoulder. When the paralysis is due to a neuritis its onset is often accompanied by severe neuralgic pains in the supraclavicular region, which may radiate up the neck, behind the scapula and even into the arm, but no loss of sensation results from an isolated paralysis of the long thoracic nerve.

**Treatment.**—Treatment must be conducted on the usual lines; it is important to prohibit work or exercise which may produce pressure or strain on the nerve. When the palsy is incurable the humeral attachment of a portion of the pectoralis major may be inserted into the serratus magnus; Tubby[1] reports one case in which this operation was performed with a favorable result.

**The Suprascapular Nerve.**—Isolated paralysis of it is very rare; Fischler[2] has been able to collect only 14 cases. In half of these it was due to direct or indirect trauma, in others apparently to a local neuritis; it may be caused by the pressure of a heavy weight carried on the shoulder, or by a fall on the shoulder or the outstretched arm. Its most prominent symptom is flattening of the infraspinous fossa and weakness of outward rotation of the humerus owing to the atrophy and palsy of the infraspinatus; this movement is not, however, completely absent, as the teres minor and the posterior fibers of the deltoid can execute it, but only very feebly. According to Duchenne, the chief function of the supraspinatus is to act as an elastic ligament in keeping the head of the humerus in close apposition to the glenoid cavity; when it is paralyzed the humerus falls away and the movements of the shoulder-joint are impeded, especially abduction and elevation in the sagittal plane. In every case of its paralysis the patient has complained of weakness and fatigue in the shoulder, and of the inability to carry weights. No definite disturbance of sensation has been detected with suprascapular palsy, but its onset may be accompanied by pain in the shoulder-girdle.

**The Circumflex Nerve.**—Paralysis is most often due to such injury as a blow or fall on the shoulder, dislocation of this joint, or fracture of the upper end of the humerus. Occasionally it is caused by the pressure of a crutch, or by lying for long on the shoulder in deep sleep or in an unconscious state; in those cases in which paralysis follows prolonged operations under anæsthesia the lesion is evidently due either to compression or undue stretching of the nerve. Similarly, paralysis of the circumflex has been frequently observed in miners who work lying constantly on the left side. Local neuritis may be due to exposure, or to extension of inflammation from arthritis or from disease in the axilla; neuritis has also been observed in infectious diseases and in diabetes. It is noteworthy that Bernhardt and Buzzard have observed isolated paralysis of the circumflex nerve in lead poisoning; in these cases there was no change in the electrical excitability of the paralyzed muscle.

[1] *British Medical Journal*, 1904, ii, 1159.
[2] *Neurologisches Centralblatt*, 1906, xxv, 444.

A complete lesion of the nerve leads to complete paralysis of the deltoid, except of a small number of its anterior fibers, which are supplied by the anterior thoracic nerves. Abduction and elevation of the arm in any plane are consequently impossible, except by rotation of the scapula by the trapezius and serratus magnus. The supraspinatus may also aid in this movement. The shoulder-joint becomes relaxed, and owing to the wasting of the deltoid its shape is altered. The paralysis of the teres minor, which depresses the arm and rotates it outward, is less prominent. The onset of the symptoms is generally accompanied by much pain. Objective sensory disturbances are not constant, but in typical cases the area of anæsthesia to light touch occupies an oval area on the outer side of the arm extending from the level of the acromion process rather more than half-way to the elbow; the loss of sensibility to pain and the extremes of temperature is less extensive. When the paralysis lasts for long there is a danger of adhesions forming in the shoulder-joint, and ankylosis may occur.

A little care and the examination of the electrical reaction of the muscle serves to distinguish primary joint disease and the secondary wasting from circumflex paralysis.

**The Musculo-cutaneous Nerve.**—It is rarely paralyzed alone. Bernhardt has collected only 14 cases, but as it is not infrequently associated with lesions of other nerves it is important to recognize its symptoms. It is generally due to a blow on, or compression of the arm, or to fracture or dislocation of the humerus. When the lesion is complete the biceps and coracobrachialis are absolutely paralyzed, as well as the greater portion of the brachialis anticus. Flexion of the elbow is consequently impossible when the forearm is supinated, but it can be carried out feebly and in limited range when the forearm is pronated by contraction of the supinator longus. Sensory disturbance is limited to the radial side of the forearm and the thenar eminence.

**The Musculo-spiral or Radial Nerve.**—The musculo-spiral or radial nerve is probably paralyzed more frequently than any other nerve in the arm. It may be injured in the axilla by dislocation or fracture of the upper end of the humerus, or it may be involved in callus formation or by the pressure of a crutch. It is occasionally compressed by the head of the humerus when the arm is kept fully abducted and extended during operations under anæsthesia (Braun). But it is much more frequently injured during its course round the humerus, very often by the pressure to which it is exposed when a person sleeps on a hard or uneven surface with the arm beneath his body, or with the weight of the head resting on the outer surface of the arm. This occurs so often in a drunken sleep that it has been assumed that chronic alcoholism predisposes to it by lowering the vitality of the nerve (Oppenheim). Gowers has pointed out that it may be injured as it passes through the triceps by a sudden violent contraction of this muscle, and, according to Adler, this is the explanation of its occasional occurrence after severe epileptic attacks. Its paralysis has followed the use of an Esmarch's bandage on the arm, and it sometimes results from the injection of ether. A local neuritis due to cold or exposure is occasionally assumed to be the cause; it is probably rarely so. When palsy develops during an acute illness it is more probably due to pressure as the patient lies in a semiconscious or delirious state. The affection of some of the fibers of the musculospiral nerve is a characteristic feature of lead palsy.

**Symptoms.**—The symptoms naturally depend on the portion of the nerve that is injured and on the severity of the injury. When the lesion is in the axilla all the muscles supplied by it are paralyzed and the patient is no longer able to extend the elbow and wrist, or the fingers or thumb at their basal joints, or to supinate the forearm except by the biceps. If the nerve is injured, as it is most frequently in the outer side of the arm, the extensors of the elbow and more rarely the supinator longus escape, but wrist-drop, which is the characteristic feature of the palsy, is present. The thumb cannot be abducted or extended, but its other movements are intact. Although the flexor muscles are not affected, the hand-grasp is considerably weakened owing to the mechanical disadvantage at which they work when the wrist is not held straight by the normal action of their antagonists; with the hand passively extended its grasp is normal. As the arm hangs by the side, the forearm is generally pronated, and becomes more fully so when the hand grasps any object, owing to the unresisted pronation action of the flexors. The power of supination with the elbow extended is completely lost, but when it is flexed the biceps can supinate the forearm. If the nerve is injured in the forearm, the supinators and even the extensors of the wrist may escape. Paralysis of the supinator longus produces slight weakness of flexion of the elbow.

Sensory symptoms are very variable; with the onset there may be subjective sensations of numbness and tingling in the cutaneous distribution, and generally most pronounced on the radial border of the hand. In incomplete lesions, as bruising or a slight neuritis, the sensory fibers are, according to the general rule, much less affected than the motor, and even with complete paralysis of the muscle there may be no loss of sensibility; when present the anæsthesia is generally most marked over the radial branch. Trophic changes are rarely prominent. In the pressure palsies there may be little or no atrophy of the paralyzed muscles. Occasionally a prominence develops on the dorsum of the hand, which is due either to the swelling of the extensor tendons (Gubler) or to overflexion of the carpus. There may be slight effusion into the carpal joints, and adhesions may form in them. The electrical reactions of the nerve and the muscles it supplies are extremely important; in the pressure palsies the nerve may be inexcitable from above the lesion, while in the portion below it and in the muscles it supplies normal responses can be obtained; the conduction of volitional and electrical impulses are thus interrupted by a lesion which is not of sufficient intensity to damage the continuity or vitality of the nerve fibers. If the lesion is more severe there must be necessarily partial or complete reaction of degeneration in the muscles it supplies.

Paralysis of this nerve is generally easily recognized, but, as Gowers points out, the fact that it produces loss of extension of the limb at all its joints may lead to error, as palsy limited to a single function suggests central disease; the absence of sensory loss, and of change in the electrical excitability of the muscles may increase the risk of error. In lead palsy it is the musculo-spiral groups of muscles which are chiefly involved, but the affection is almost invariably bilateral, the onset is usually slow and unconnected with trauma, the supinator longus, as a rule, escapes, and the reaction of degeneration appears early in the paralyzed muscles. Musculo-spiral palsy may be only a part of a general polyneuritis.

**Treatment.**—Treatment must be conducted on the usual lines. In the pressure palsies the application of the galvanic current for twenty to thirty minutes at a time is of value; the kathodal electrode should be placed over the seat of injury, the anode distal to it, and the strength of current slowly increased until the patient feels it distinctly.

**The Median Nerve.**—Owing to its deep position among the soft tissues of the arm the nerve is much less liable to injury than the musculo-spiral. In the axilla and arm it is usually injured by fractures and dislocations of the humerus and occasionally by the pressure of a crutch, but the most frequent cause of its paralysis is a wound on the palmar surface of the wrist. Occupation palsies frequently involve some or all of the hand muscles supplied by the median nerve; it has been repeatedly observed in laundresses, joiners, milkmaids, and cigarette makers. The exact nature of the lesion is doubtful; often it is undoubtedly due to a neuritis set up by pressure, but in other cases the muscular palsy and atrophy seem to be the direct result of the stress of overwork. Drummer's palsy affects chiefly the thumb muscles. A few cases have been recorded in which paralysis of the median nerve followed obliteration of the radial artery.

When the nerve is damaged above the elbow the power of pronating the forearm is lost; flexion of the wrist is feeble and incomplete, and as it can be performed only by the ulnar flexor, the hand is strongly deviated to the ulnar side. Flexion of the interphalangeal joints is also lost except that of the distal phalanges of the two ulnar fingers, which can be still bent by the unparalyzed part of the flexor profundus. The flexion of the fingers on the metacarpus is unaffected, as it is performed by the interossei. The unopposed extensor action of the latter muscles at the interphalangeal joints may lead to their hyperextension. The thumb is kept extended and adducted by the muscles which remain, and it cannot be opposed nor its distal phalanx flexed; its metacarpal bone comes to lie in the same plane as that of the fingers, like the thumb in the ape. Owing to paralysis of these movements of the fingers and thumb there is considerable difficulty in firmly grasping any object and in employing the hand in any work. When the lesion affects the nerve in the forearm, after its branches to the pronators and flexors have been given off, pronation and flexion of the wrist and fingers may be intact; then it is chiefly the thumb movements which are lost. Bernhardt and Head have pointed out that the branch which supplies the muscles of the hand may leave the main stem of the nerve in the lower part of the forearm and thus escape injury when the wound is at the wrist. When the lesion of the nerve is severe, prominent atrophy of the muscles of the thenar eminence results, and of the flexor surface of the forearm if it is above the elbow. There may be considerable paræsthesia in the cutaneous area of the nerve in earlier stages of the paralysis; the occupation palsies and neuritis are generally accompanied by troublesome pain. A complete lesion of the nerve may lead to troublesome trophic disturbance of the skin and nails in the area where protopathic sensibility is lost, and to vasomotor paresis and cessation of sweating in the radial part of the palm.

**The Ulnar Nerve.**—This may be injured alone or with other nerves in the axilla or upper arm by dislocation or fractures of the humerus, or involved in callus formation; occasionally it suffers in crutch palsy. It is much more frequently damaged at the elbow-joint by dislocations or fractures; occasionally ulnar paralysis develops slowly at long periods after elbow

injuries owing probably to the pressure of fibrous adhesions or excess of callus on the nerve, and in a few cases it has been due to traumatic or synovial cysts in this region. More rarely the curious condition of dislocation of the ulnar nerve from its groove on the posterior surface of the internal condyle is met with; it probably occurs only when the internal condyle is badly developed. Paralysis due to direct pressure from without is rare; it is occasionally due to pressure on the elbow during sleep, especially in emaciated subjects, or during infective illness. According to Braun, however, these sleep palsies are to be attributed to pressure of the head of the humerus on the nerve in the axilla when the arm is abducted and extended. But undoubtedly wounds in the wrist, which may either injure this nerve alone, or the median nerve and flexor tendons as well, are the most common cause of ulnar palsy. Primary neuritis is rare; a few cases of syphilitic neuritis have been described, and leprosy has a definite predilection for this nerve.

When the nerve is injured at or above the elbow the power of flexion of the hand is very feeble, and when attempted the hand is deviated radialward by the radial flexor; the wrist becomes hyperextended when the fingers are straightened, owing to palsy of its ulnar flexor. The movements of the little finger are lost, the middle and ring fingers cannot be flexed at their distal joints, and owing to the paralysis of the interossei the basal phalanges of all the fingers cannot be flexed or the middle or distal phalanges fully extended. Adduction and abduction of the fingers are also impossible. When the injury is situated in the lower part of the forearm the fibers to the flexor profundus digitorum escape and the interossei and thumb muscles are alone paralyzed; then owing to the unopposed contraction of the long extensors and flexors the hand becomes claw-like—*main en griffe*—with the first phalanges hyperextended and the interphalangeal joints flexed. As the first two lumbricales escape, this deformity is generally not so pronounced as it is in progressive muscular atrophy (Gowers). Adduction of the thumb is also lost. The muscular atrophy which follows severe lesions of this nerve is very typical; the hypothenar eminence disappears, the palm becomes hollow, and the interosseal spaces sink in. When the nerve is severely damaged there is complete loss of sensation in the little finger and over a variable extent of the ulnar border of the hand; on the ring finger and the rest of the ulnar cutaneous distribution sensibility to light touch and the intermediate degrees of temperature are alone affected. As the dorsal cutaneous branch separates from the nerve in the middle third of the forearm it may escape in wounds at the wrist. Dupuytren's contracture of the palmar fascia has been observed after neuritis of the ulnar nerve, and de Leon[1] has described contractures of the ulnar portion of the flexor profundus digitorum, owing to which the three ulnar fingers were bound down in the maximal flexion position, so that only the thumb and index fingers could be used—*main en prince.*

The chief danger of error in diagnosis is of confusing central and peripheral lesions, as the ulnar nerve contains almost all the root fibers of the eighth cervical and first thoracic segments of the cord. Disease of the cord is, however, generally accompanied by distinctive features.

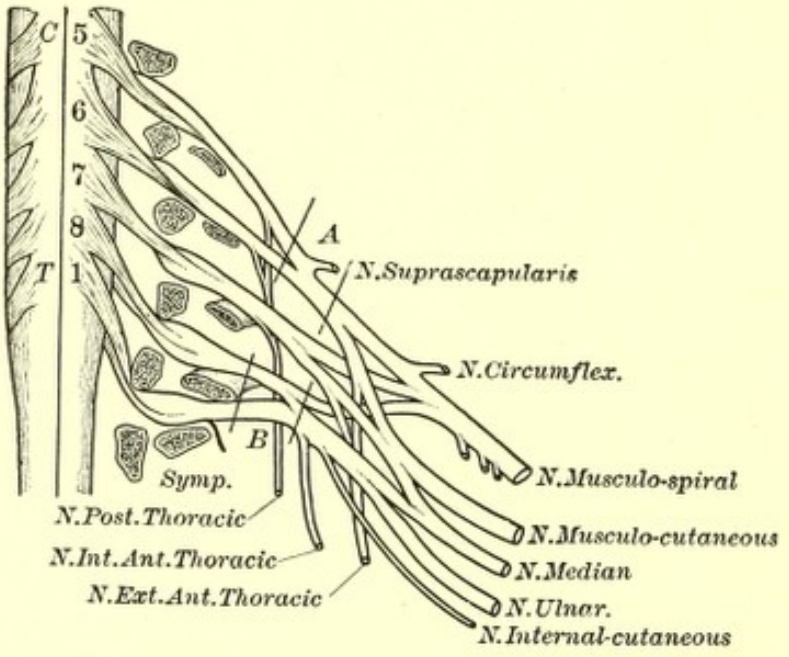
**Paralysis of the Brachial Plexus.**—The brachial plexus is formed by the anterior primary divisions of the lower four cervical and the first thoracic roots; it contains all the fibers which supply the muscles of the shoulder

[1] *Nouvelle Iconographie de la Salpétrière*, 1901, xiv, 409.

girdles and upper extremities, as well as the sensory fibers to almost the whole of the arm. By division and secondary anastomosis of these roots the three main cords of the plexus are formed. The outer cord receives the ventral trunks of the fifth, sixth, and seventh cervical roots; the inner contains the ventral trunk of the eighth cervical as well as the whole of the first thoracic root, and the posterior cord is made up of the dorsal trunk of the four lower cervical roots. This is the most common form of the brachial plexus, but it is liable to variation, for the fibers concerned in any single function do not constantly leave the cord by the same roots, but although fibers may alter their position relative to the vertebral column, they always maintain their position in relation to other fibers (Herringham). The whole plexus may be shifted up—high or prefixed type—or downward—low or post-fixed type—and the extreme variations between the high and low form of plexus may amount to nearly a whole root (Harris).[1] This fact explains to some extent the variations in the effect of lesions of the same portion of the plexus in different individuals. Paralysis of a muscle or of muscles may be due to injury or disease of the nerve fibers in the roots, in the plexus, or in the nerve trunks and their branches.

Two types of plexus paralysis merit special description; they occur as primary palsies, or as residual palsies when a more or less complete plexus paralysis has partially cleared up.

FIG. 5




A diagram of the brachial plexus and its connections with the spinal cord. *A*, represents the usual seat of the lesion in the upper arm (Duchenne-Erb) type of plexus paralysis; *B*, that in the lower arm or Klumpke type. *Symp.* is the Ramus communicans from the first thoracic root which carries the oculo-sympathetic fibers.

**Duchenne-Erb Palsy, or the Upper Arm Type.**—In this form the deltoid, biceps, brachialis anticus, and supinator longus are generally paralyzed together, and frequently also the supinator brevis and supra- and infra-

[1] *Journal of Anatomy and Physiology*, 1904, xxxviii, p. 399.

spinati; more rarely other muscles of the shoulder-girdle, and the radial extensor of the wrist and the pronator radii teres. The lesion to which it is due involves the fifth and sixth cervical roots either before or immediately after their union, or occasionally the fifth root alone; when caused, as it frequently is, by a blow or fall on the shoulder, it has been assumed that these roots were compressed between the clavicle and the transverse processes of the lower cervical vertebræ, or the first rib, but it seems more probable that the lesion is a rupture of some or all of the fibers of this portion of the plexus owing to excessive stretching and tension. This may be due to any cause which increases the distance between the shoulder and the head and neck. The vulnerability of the two upper roots of the plexus to this form of trauma is explained by the fact that they bend downward immediately on their exit from the spine, so that the extravertebral portion of each root forms an angle open downward with the portion which lies in the intervertebral foramen, while in the lower two roots of the plexus this angle is open upward and in the seventh root there is little or no bend.

In severe cases the arm hangs by the side and cannot be abducted, because of the paralysis of the deltoid, or rotated outward at the shoulder if the infraspinatus is affected. Flexion of the elbow is also impossible owing to the paralysis of the biceps, brachialis anticus and supinator longus, or it can be effected through a small range by the pronator radii teres and the flexors of the wrists.

Supination of the forearm is always weakened by the loss of power in the biceps and it may be impossible if the supinator brevis is also affected. Sensory symptoms are absent in the slighter cases, but there may be paræsthesia and loss of sensation on the radial side of the arm and forearm. Even complete section of the anterior primary division of the fifth, and sometimes of the fifth and sixth roots may produce no sensory loss (Sherren). Erb has shown that all the muscles paralyzed in this type of plexus palsy may be made to contract by electrical stimulation over a point in the neck 3 cm. lateral to the sternomastoid and the same distance above the clavicle; the loss of excitability of the nerve trunks from this point is an important sign of the upper arm type of plexus palsy.

**Klumpke Palsy, or the Lower Arm Type.**—This form of paralysis, in which the eighth cervical and first dorsal roots are involved, is generally met with only as the residue of a more extensive lesion, but it may be due to compression by a tumor in the neck or a growth in connection with the lung or vertebral column. Owing to the deeper position of these roots they are rarely injured by trauma. The palsy is characterized by an atrophic paralysis of the intrinsic muscles of the hand and generally of some of the forearm muscles, especially the flexors, with a certain amount of loss of sensation in the ulnar fingers and the ulnar border of the hand and forearm. Frequently, too, there are pupillary symptoms due to injury of the sympathetic fibers which leave the cord in the first thoracic root; but as these branch off from the root immediately outside the intervertebral foramen, they are affected only when the inner portion of the root is damaged. When they are injured, the palpebral fissure is narrowed, the pupil contracted, and there may be a slight degree of exophthalmos on the same side as the arm palsy. Vasomotor paresis on the same side of the face occurs probably only when the second and third thoracic roots are injured (Klumpke). When the small hand muscles are alone paralyzed, the typical deformity of claw-like hand

results; if the flexors of the fingers and wrist are also powerless, the wrist may become hyperextended owing to contracture of the unopposed extensors.

These traumatic plexus palsies are frequently followed by severe spontaneous pain in the arm, probably owing to the constriction of the nerve fibers in scar tissue, and often by trophic disturbances.

**Birth Palsies.**—Birth palsies due to injury of the brachial plexus generally belong to the Duchenne-Erb or upper arm type, and the limb cannot be adducted or rotated outward at the shoulder, or flexed or supinated at the elbow. The paralysis is often more extensive, and occasionally it is one or more of the lower roots which is damaged. The causation of these birth palsies has been much discussed; for the purpose of treatment it is important to recognize the nature of the lesions and to what they are due. Fracture or separation of the epiphysis of the humerus, dislocation of the shoulder, and fracture of the clavicle are occasionally found associated with birth palsies, but they are rarely the direct cause. In some cases injury of the plexus may be due to the direct pressure of the accoucheur's finger in the axilla or supraclavicular fossa, or to the blade of the forceps in the latter region; by other authors it has been assumed that its upper roots are compressed between the clavicle, which is drawn backward by the elevation and retraction of the shoulder, and the transverse processes of the lower cervical vertebræ or the first rib. It is most probable that the majority of these birth palsies are due to laceration or rupture of the fibers of the nerve trunks or roots by excessive stretching and tension. Clarke, Prout, and Taylor[1] have found rupture of the sheath and hemorrhages into the nerve, with secondary cicatricial contraction, in cases which were operated upon; complete rupture of the fifth root alone has been observed, but in most cases the lesion is found at the junction of the fifth and sixth roots. The sensory disturbances are slight and generally unimportant, but marked trophic changes may result. The paralyzed muscles atrophy quickly and lose their electrical excitability, the growth of the bones of the limbs is retarded, and owing to vasomotor disturbances the limb often becomes cold and cyanosed, and there may be swelling owing to chronic œdema of its subcutaneous tissues, and the skin may become thin and atrophy.

*Prognosis.*—The outlook in the plexus palsies naturally depends very largely on the nature of the lesion, but it is much less favorable than in disease of the peripheral nerves. Only six of the twenty-three cases recorded by Bruns recovered, and eleven of the thirty reported by Warrington and Jones.[2] Many of the cases of neuritis, but these are rare, and of those in which the loss of conductivity of the fibers is due to pressure or bruising, improve or make complete recoveries; but when a root or trunk is torn or ruptured the prognosis is very grave unless surgical treatment can be successfully adopted. This is due in the first place to the fact that the ruptured fibers become enveloped in dense cicatricial tissue, and secondly because the cells of origin of the fibers in the ventral horns of the cord are liable to degenerate completely when their axis-cylinders are forcibly torn out. The prognosis is consequently less favorable in cases of rupture due to indirect violence than when the plexus is damaged by a penetrating wound, and the chances of recovery diminish the nearer the lesion is to the spinal cord.

[1] *American Journal of the Medical Sciences*, 1905, cxxx, 670.
[2] *Lancet*, 1906, ii, 1644.

**Brachial Neuritis.**—This is a form of neuritis limited to the brachial plexus which is closely analogous to sciatica; it seems to be relatively rare. It occurs generally in late life and in females more often than in males. The pain, which is often sudden in onset, is generally of great severity and is at first referred to the back of the scapula, the forearm or hand, or to the region of the plexus itself above the clavicle or in the axilla. As a rule, it is at first intermittent, but soon becomes more continuous with paroxysmal variations, which occur spontaneously or are induced by movement. When fully developed the pain may spread over the whole arm; it is usually dull and aching, but in the severer attacks it is sharp, lancinating or stabbing. It may be accompanied by undue sensitiveness or hyperalgesia of the skin, and perhaps with tingling and numbness, but definite objective change in sensation is rare. The muscles may present slight wasting and loss of power, but it is more apparent than real, as the patient is reluctant to move the limb owing to the pain which ensues. Adhesions often develop in the joints and trophic changes in the skin and subcutaneous tissues. The condition can be mistaken for neuralgia, the chief points of distinction are the persistent tenderness of the nerves, and the influence of movement on the pain. There seems to be occasionally a danger of mistaking brachial neuritis for pain of angina pectoris or that due to an aneurism, but an accurate history and a careful physical examination should easily exclude the risk of confusion. The chief aim in treatment should be to avoid movement or anything which may excite pain. The disease is obstinate, and complete recovery cannot be always expected.

*Treatment.*—In neuritis counter-irritation may be applied over the plexus. It is much more important to maintain the nutrition of the paralyzed muscles by electrical (galvanic) treatment and massage, and to prevent the formation of adhesions in the joints and of contracture in the muscles. Warrington has laid emphasis on the importance of preventing the overstretching of the paralyzed muscles by the unrestrained contraction of their antagonists. When there is evidence that the nerves have been ruptured, or that the damaged fibers are enveloped in dense cicatricial tissue operative interference becomes necessary. It is often difficult to decide when surgical intervention should be resorted to, as we cannot be always certain of the nature or extent of the lesion, and even under favorable conditions recovery is slow. If there is no return of power within nine or twelve months of the injury in a case which has had proper treatment, it is justifiable and generally advisable to explore the plexus. The results of surgical treatment have been less favorable in cases of birth palsy than in the traumatic palsies of the adult, but a considerable number of cases of both varieties have been reported in which more or less complete recovery has followed secondary suture or grafting of the nerves.

**The Symptoms Due to Cervical Ribs.**—Attention has been only recently directed to the occurrence of nervous symptoms dependent on the presence of supernumerary cervical ribs. Thorburn,[1] who was the first to recognize the importance of the condition in England, recorded 4 cases in 1904. Thomas and Cushing[2] had already reported one case in America in the previous year; since then numerous papers have appeared, including

[1] *Trans. Royal Med. and Chirurg. Soc.*, London, 1905, lxxxviii, 109.
[2] *Johns Hopkins Hospital Bull.*, 1903, xiv.

a second contribution by Thorburn[1] and those by Lewis Jones[2] and Howell[3] in England, and Keen[4] and Colin Russel[5] in America, as well as several in German literature. Now that attention has been directed to it the condition seems to be not at all uncommon; Howell alone observed 16 cases in a relatively short period. Supernumerary cervical ribs are not rare; they are generally bilateral, but, as a rule, more developed on the one than on the other side. The symptoms they produce, however, are most often unilateral, and seem to be due either to the abnormal stretching of the lower trunk or inner cord of the brachial plexus as they pass over the rib, or to the compression of the first thoracic and eighth cervical roots by it before their junction. Occasionally this portion of the plexus may be compressed between the abnormal rib and the first dorsal one. The shape and direction of the rib are of more importance than its mere size; those which grow directly outward are not so liable to cause symptoms as those which project downward and forward into the posterior triangle of the neck.

The symptoms produced are much more common in females than in males, and generally appear in early adult life. Their appearance at this age is probably due to the late ossification of the bone, while the smaller group of cases which occur in late life may be the result of changes in the shape, or of senile rigidity of the spine and thorax (Thorburn). The right arm is more frequently affected, probably because its freer use exposes the plexus of this side to greater strain, and makes it more liable to injury.

The first symptom, and the only one in many cases, is almost invariably pain in the shoulder and arm, which is generally best localized along the ulnar border of the forearm and hand; it is always increased by vigorous or continued movement of the limb. It may be associated with numbness and tingling, or a feeling of coldness in the same areas, usually most pronounced in cold weather. In the majority of the cases some loss of sensation eventually appears in the distribution area of the first thoracic root, or on the areas of the first thoracic and eighth cervical roots, that is on the ulnar borders of the forearm and hand, and in the two ulnar fingers. As is characteristic of root lesions, there is generally a certain amount of dissociation of the various forms of sensation, the tactile loss being less pronounced than the analgesia, while of all, the thermal sense seems to suffer the most severely. In many cases there is little or no objective sensory disturbance.

A feeling of weakness or uselessness of the limb is often one of the earliest symptoms, but definite motor paralysis is much less common than the sensory disturbances. After a variable period, however, during which the pain has been more or less constant, the hand generally becomes weak and its intrinsic muscles, especially those of the thenar eminence, atrophy and lose their normal electrical reactions; the atrophy is usually most prominent in the abductor and flexor brevis pollicis. When the eighth cervical root is also affected the flexors of the fingers, and to a less extent those of the wrist, may be weak. When only its intrinsic muscles are affected the hand may assume the typical *main en griffe* position. Spasm of the flexors of the fingers and probably of other muscles frequently occurs when the palsy is not advanced. Clonic twitching has been observed.

[1] *Manchester Med. Chronicle*, 1908, xlvii, 165.
[2] *Medical Electrology and Radiology*, 1906.
[3] *Lancet*, 1907, i, 1702.
[4] *American Journal of the Medical Sciences*, 1907, cxxxiii.
[5] *Medical Record*, 1907, lxxi, 253.

The almost constant absence of oculo-pupillary symptoms, despite the paralysis of the first thoracic nerve, indicates that the lesion is situated outside the intervertebral canal where the sympathetic fibers branch off from the root. Sympathetic ocular symptoms have, however, been observed. In some cases the subclavian artery is also compressed by the cervical rib, causing inequality of the radial pulses; arterial thrombosis and even gangrene of the fingers have been observed. The cervical rib may be often palpated in the neck, and there is generally a point of tenderness to pressure over it. Cervical scoliosis convex toward the rib is frequently met with; it was present in 22 of the 61 cases collected by Schönebeck.[1]

The *diagnosis* must depend largely on a radiographic examination, by which the presence of abnormal ribs may be easily revealed; but it must be remembered that cervical ribs often produce no symptoms and that other nervous diseases are occasionally associated with them, especially syringomyelia, which has been found in several cases. Cervical ribs should be suspected in every case of isolated paralysis of the first thoracic, or of paralysis of the first thoracic and eighth cervical, roots, which cannot be otherwise explained. The presence of vascular symptoms, as inequality of the radial pulses, is of considerable value.

The only rational *treatment* is the removal of the supernumerary rib, but it is unhappily an operation attended with some danger, for though in many cases a cure has been attained, in others more extensive paralysis has resulted from operative interference with the plexus. Pain, however, seems to be always relieved by the removal of the rib. In the neuralgic cases the pain may disappear if the limb is kept at rest.

**Nerves of the Lower Limbs.**—The nerves of the lower limbs are much less frequently involved in injuries or affected by disease than those of the upper extremities.

**The Anterior Crural Nerve.**—Isolated palsy of this nerve is rare, but it may be due to compression by abdominal growths or by a psoas abscess, or to injury by fractures of the upper end of the femur or pelvis, or disease of the bones. Primary local neuritis is seldom seen, but has been occasionally observed in diabetes. This nerve, either alone or with the obturator, may be injured during parturition; this probably occurs with greater frequency than is recognized, owing to the rapidity with which it recovers from slight compression and to the fact that its symptoms may not be noticed while the patient is confined to bed. Ernst has observed 30 cases in 800 births. The psoas muscle is paralyzed only when the nerve is damaged in the immediate neighborhood of the lumbar plexus; when the lesion is situated here the thigh cannot be flexed in the abdomen, and if paralysis is bilateral the trunk cannot be flexed on the thighs when these are fixed; the patient is consequently unable to rise from the supine position. When the lesion is in the intra-abdominal portion of the nerve the iliacus alone is paralyzed, and flexion of hip is only weak. But the most prominent symptom is paralysis of the extensors of the knee, and the absence of the knee-jerk in the affected limb. Paralysis of these muscles does not make standing or walking impossible, but contraction of its flexors must be avoided, as the patient cannot resist flexion or actively straighten the joint. The paralysis of the pectineus and sartorius does not produce any other prominent symptom.

[1] *Inaug. Dissertation*, Strassburg, 1905.

Its sensory branches arise in the upper part of the thigh; the middle and internal cutaneous are distributed to the lower two-thirds of the front and inner side of the thigh, the internal saphenous to the front and inner side of the leg, and the inner side of the dorsum of the foot. Disturbance of sensation, paræsthesia, or radiating pains may be present over these extensive areas when the nerve is injured at or above the level of the groin.

The *diagnosis* of paralysis of the anterior crural nerve is, as a rule, evident; the only risk is of confusing the marked atrophy of the quadriceps extensor group in the front of the thigh which occasionally results from inflammation of the knee-joint with atrophy of these muscles, due to a nerve lesion; but in the former, although the electrical excitability of the muscles may be diminished, there is no reaction of degeneration. The ordinary lines of *treatment* must be followed; good results have been obtained by grafting the tendon of one of the flexors of the knee into the tendons of the quadriceps extensor when the paralysis is permanent.

**The Obturator Nerve.**—The obturator nerve is still more rarely injured alone. It may be injured during parturition, by intra-abdominal or pelvic growths, or by an obturator hernia. When the muscles it supplies are completely paralyzed the limb cannot be adducted, and although it can be raised by flexion of the hip, it cannot be thrown across its fellow when the patient is seated on a chair. Owing to paralysis of the obturator externus, outward rotation is enfeebled, and inward rotation of the thigh is also weak, as the adductor magnus is paralyzed. Gait is not seriously interfered with. The disturbance of sensation which results from paralysis of this nerve is limited to a small area on the inner side of the lower half of the thigh.

*Meralgia paræsthetica* is a condition characterized by paræsthesia and pain, usually with slight objective disturbance of sensation, in the region supplied by the external cutaneous nerve on the front and outer side of the thigh. It occurs chiefly in middle-aged men, less frequently in women. Its etiology is obscure; in many cases there has been a history of trauma; probably the long course of the nerve through the fascia predisposes to its injury. In one case a localized perineuritis was found (Nawratzki[1]), but in other specimens there was no abnormality. In about one-sixth of the cases reported the condition was bilateral. In 1900 Schlesinger[2] analyzed 122 recorded cases, and in the same year Musser and Sailer[3] added ten personal observations in a valuable contribution. The condition has been observed associated with flat foot. The symptoms vary greatly in intensity in different cases. The most common complaint is of abnormal sensations, of numbness, coldness, or tingling, on the front and outer surface of the thigh. In other cases pain is the chief symptom; it may be very severe, but is usually felt only after walking or standing; it is probably due to constriction of the nerve as it passes through the deep fascia when the latter is tense. Occasionally it persists even when the patient is lying down. In the majority of the cases there is a point of tenderness just below the anterior superior iliac spine where the nerve pierces the fascia. Objective sensory disturbances are very variable, but often considerable; occasionally there is hyperæsthesia. The symptoms are very intractable to treatment; rest may be necessary

[1] *Neurol. Centralbl.*, 1899, xviii, 133.
[2] *Centralbl. f. d. Grenzgeb. d. Med. u. Chir.*, 1900, iii, 241.
[3] *Journal Nerv. and Ment. Dis.*, 1900, xxvii, 16.

when there is much pain, and massage and the faradic brush over the course of the nerve may give relief. Resection of the nerve has cured some cases, but in others the pain has returned (Bramwell).

Similar symptoms have been occasionally observed in the distribution of the middle cutaneous branch of the anterior crural nerve, but generally associated with meralgia paræsthetica. Lasarew,[1] who has found the condition isolated, has given it the name *meralgia paræsthetica anterior.*

**Gluteal Nerves.**—Isolated paralysis of the *superior gluteal nerve* is uncommon; the muscles it supplies are the chief abductors and inward rotators of the thigh, and when they are paralyzed these movements are weak or lost. As the posterior fibers of the glutei rotate the limb outward, this movement becomes weak.

The *inferior gluteal nerve* is also rarely paralyzed alone; when this occurs the thigh cannot be forcibly extended, nor the trunk straightened on the thigh when the lower limbs are fixed. Standing or walking on a level are not seriously interfered with, but the limb is of little use in ascending steps, and the patient has difficulty in rising from the sitting position, as this is an action in which extension of the hip is the most important movement.

**The sciatic nerve** supplies motor fibers to the ham-strings and to all the muscles below the knee, as well as the skin in the outer side of the leg and the whole of the foot except a small part of the inner portion of its dorsum. Its main terminal branches are the external popliteal or anterior tibial nerve, and the internal popliteal or posterior tibial nerve; these generally separate in the popliteal space, but are sometimes distinct from their origin in the plexus. The symptoms due to paralysis of each of these branches will be considered before those due to a lesion of the whole nerve.

**The external popliteal nerve** may be injured by direct trauma in any part of its course, but it is especially liable to be bruised by a blow or by pressure as it bends round the fibula. It has been occasionally torn or ruptured by violent extension of the limb, and paralysis has been frequently observed in laborers who work in a kneeling or crouching position; there it is probably due to compression of the nerve between the fibula and the tense tendon of the biceps cruris. A primary neuritis of this nerve is by no means rare, and even in a general neuritis its fibers seem especially liable to degeneration. Lead palsy is occasionally limited to its distribution, especially in children (Putnam), but in these cases the tibialis anticus escapes as a rule. Isolated paralysis of this nerve is sometimes seen in tabes dorsalis. The whole limb must be unduly raised, as it is brought forward in walking, to enable the toes to clear the ground. Inversion and eversion of the foot are also weakened when there is a total paralysis. When the tibialis anticus is alone paralyzed the foot can be still flexed by the long extensors of the toes, but it is at the same time abducted; while if the function of the latter muscle alone is lost the active tibialis anticus inverts and adducts the foot. As Bernhardt points out, it not infrequently happens that the tibialis anticus escapes when the other muscles of this group are paralyzed. The paralysis of the peroneus longus is most evident when the ankle is extended, as the foot is then so strongly inverted by the unopposed action of its extensors that its outer border rests on the ground, and, as the inner part of the foot is not supported during extension, flat foot may develop. When

[1] *Deutsch. Zeit. f. Nerven.*, 1908, xxxiv, 154.

all these muscles are paralyzed talipes equinus gradually develops, owing to contracture of their antagonists; if the paralysis of all the muscles is not equal in degree the foot may be at the same time either inverted or everted, according to the degree of the paralysis of the muscles with the opposing function. The toes may be permanently flexed by the contracture of the unopposed flexors and interossei; this adds considerably to the difficulty in walking. The sensory loss which results from a lesion of this nerve is limited to the outer side of the leg, the dorsal surface of the foot, and the dorsum of the first phalanges of the toes.

**The Posterior Tibial or Internal Popliteal Nerve.**—Owing to its deeper course it is less liable to injury than the external popliteal, and its isolated paralysis is consequently much rarer. A few cases have been recorded in which it has been injured by the tendons of the flexors of the knee when these muscles are forcibly contracted (Oppenheim), and it may be compressed by or involved in tumors, aneurisms, or inflammations.

The most prominent feature is inability to extend the foot or flex the toes, so that the patient can no longer stand on tip-toe or spring from the forepart of the foot in walking; if the paralysis is of long duration the unantagonized action of the flexors of the ankle produces talipes calcaneus, while the unopposed action of the peroneus longus leads to eversion of the foot and increases the plantar arch. Flexion of the distal and middle phalanges of the toes is no longer possible, owing to paralysis of the long flexors, while the loss of the interossei and of the adductors and abductors of the great and small toes makes lateral movements of the toes impossible. The unopposed contraction of the long extensors may lead to permanent overextension of the basal phalanges and the deformity of claw foot or *pied en griffe*.

When the conduction of sensory impressions is completely interrupted there is loss of sensation on the outer side and back of the lower third of the leg, on the outer border of the foot and on the sole and plantar surfaces of the toes, as well as in the dorsum of the distal phalanges. There may be trophic disturbances in the skin and nails, and ulcers may form.

**Paralysis of the main trunk of the sciatic nerve** may be produced by fractures of the pelvis or of the upper end of the femur, or by dislocations of the hip-joint; or the nerve may be compressed by tumors in the pelvis or invaded in the extension of septic processes from the surrounding tissues. Some or all of the fibers of the nerve may be paralyzed during parturition, but the lesion is then generally situated in the lumbosacral plexus; the sciatic nerve of the child may be injured by traction on the leg in breech presentations. The symptoms of complete paralysis by a lesion near the sciatic notch are those of paralysis of its terminal branches, the internal and external popliteal nerves, with, in addition, palsy of the flexors of the knee. When the latter are powerless the limb must be held extended at the knee in walking, and it can be used only as a stilt; gait is consequently considerably impeded, especially as there is no power of movement at the ankle-joint. As all the sensory fibers of the nerve enter its terminal branches, the loss of sensation in a complete sciatic palsy includes the outer side of the leg and the whole of the foot except a small area in the inner side of its dorsum.

**The lumbar and sacral plexuses** and the extradural portions of their roots are much less commonly affected by trauma or disease than is the brachial plexus. Isolated paralysis of the lumbar plexus is extremely rare and merits no further reference, but palsy of the whole or part of the sacral

plexus is occasionally met with. It may be due to invasion or compression of some or all of its roots by tumors or inflammation, or the roots may be injured by pressure from the fetal head during birth. In the latter case it is generally only the fibers which enter the external popliteal nerve which suffer. This, it has been shown by Hünermann[1] and Thomas,[2] is due to the fact that the higher roots of the plexus from which this branch receives the majority of its fibers lie directly on the bone as they pass over the brim of the pelvis and are consequently more liable to suffer from compression than the sacral roots which are separated from the bone by the pyriformis muscle. For the same reason the superior gluteal nerve is often injured at the same time. But probably the most common causes of paralysis of these roots are malignant tumors of the pelvis, or tuberculous caries, sarcomata or metastatic carcinomata of the sacrum, which either compress or invade these roots in the intervertebral canals. The symptoms are generally those of an incomplete sciatic paralysis, but if the upper roots are involved the outward rotators of the hip and the gluteal muscles are in addition paralyzed; or if the lower, there will be probably sensory loss in the distribution of the small sciatic nerve on the back of the thigh and on the buttocks and perineum.

**Diagnosis.**—The diagnosis of disease of the nerves of the lower limbs is, as a rule, easy, but different conditions with which a partial or complete paralysis of the sciatic nerve and its branches may be confused needs further consideration. The diagnosis is often greatly dependent on the history of the mode of onset of the paralysis and of its course; when it immediately follows an injury in the region of the nerve there can be little room for doubt, if the symptoms correspond to the portion of the nerve injured. Disease of the sciatic nerve and its branches must be distinguished from:

1. *Lesions of the Sacral Plexus and of the Extradural Portions of the Lumbosacral Roots.*—When the disease is situated in the sacral plexus, muscles other than those supplied by the sciatic nerve are paralyzed, as the glutei, the obturator internus, the gemelli, and the quadratus femoris; and the anæsthesia may extend to the back of the thigh and to the buttocks if the lower portion of the plexus is involved. A careful examination of the pelvis may reveal the presence of a tumor or of other disease. The extradural portions of the sacral roots are most frequently involved by tumors or disease of the sacrum; at first, as a rule, only one root is affected and the earliest symptom is generally pain, which is often extremely severe, referred to the peripheral distribution of its sensory fibers, and paresis of the muscles which are largely supplied by it. The neighboring roots are subsequently paralyzed, and if the disease extends across the middle line, motor and sensory symptoms may develop in the opposite limb. The distinguishing feature, as contrasted with a plexus or nerve paralysis, is that the motor and sensory symptoms correspond in extent with the distribution of the root fibers. Further, if the lower sacral roots are involved before they give off their visceral branches to the sympathetic system the bladder and rectum are paralyzed; true sphincter paralysis never results from disease of the nerves.

2. *From lesions of the cauda equina*, the paralysis of the nerves which spring from the sacral plexus may be distinguished by the fact that the symptoms

[1] *Archiv f. Gyn.*, 1892, xlii, 489.
[2] *Johns Hopkins Hospital Bull.*, 1900, xi, 279.

in the former are always of radicular and not of nerve distribution, that they are almost invariably bilateral, and that when the disease has advanced sufficiently far all the roots below the level of the intrathecal disease are generally involved. The sphincter functions too are almost invariably affected. It is more difficult to distinguish between disease of the cauda equina and of the extradural portions of the spinal roots; in the latter condition, however, the symptoms are often uniradicular for a considerable time, as the sacral disease to which they are most commonly due will generally involve only one root at first. Another point of distinction is that all the roots below the level of the affected one are not paralyzed in the latter condition, no matter how long the disease lasts, unless the sacral tumor extends into the vertebral canal and compresses the cauda equina; while when this is primarily affected by tumor or meningitis, all the roots which pass through the level of the disease are, as a rule, compressed.

3. *From disease of the sacral segments of the cord* the diagnosis is easier. The symptoms, as a rule, develop more rapidly; they are almost invariably bilateral and are typically radicular in distribution, but all functions represented in the segments below the upper level of the disease are interfered with. If, however, the lower segments are not involved, the paralysis of the muscles which they supply is not associated with atrophy or change in the electrical reactions. The severe radiating pains which are an almost invariable symptom of root lesions are absent, and anæsthesia develops earlier. The sphincter functions are generally seriously affected.

**Sciatica.**—This term is commonly applied to all affections of which the chief symptom is pain in the distribution of the sciatic nerve. Such pain may be of the nature of a neuralgia and unassociated with any disease of the nerve, or it may be due to a neuritis, or to compression of the nerve or its roots by tumors or by fibrous adhesions secondary to inflammation. It is unfortunate that the one term should be used for the symptoms of these different conditions, but it is, indeed, often difficult to differentiate between them. It is, however, important to separate the cases in which there is pain without any evidence of organic disease in the nerve, from those in which sciatic pain is associated with symptoms of a nerve lesion, as anæsthesia, atrophic muscular paresis, change in the electrical reactions of the muscles and loss of the Achilles tendon-jerk.

**Etiology.**—Males are affected much more frequently than females, in about the proportion of 5 to 1. It occurs more frequently in middle life, and very rarely, if ever, under fifteen years of age. It has been attributed to almost innumerable causes, but exposure to wet and cold is generally the only apparent exciting factor; it may follow sleeping in a damp bed, or sitting on a wet or cold seat. Gowers insists that many cases develop on a gouty diathesis; others undoubtedly follow spondylitis. The disease may be also due to trauma to the nerve, as by continuous pressure on the edge of a chair, a fall on the buttock, or injury in the neighborhood of the hip-joint. Occasionally an attack sets in after severe muscular exertion, but probably only in those predisposed to the disease. It occurs frequently in anæmic and badly nourished subjects, and in the course of chronic intoxications (alcohol), without any apparent exciting cause; and often after infectious diseases. Sciatica is not infrequently a symptom of diabetes, and is then usually bilateral. Quénu has shown that the pain may be due to the pressure of dilated and varicose veins on the nerve in the neighborhood of the

sacrosciatic foramen; this form generally occurs only in those who work all day standing erect.

Sciatic pain may be also due to the presence of tumors or inflammatory processes in the pelvis, or to a loaded rectum, which may either directly compress the nerve or affect the nutrition by the venous stasis it produces. The sacral plexus is occasionally injured by the fetal head during birth, or it may be compressed by a retroflexed uterus. Finally, pain in the course of the sciatic nerve may be due to affection of its roots by disease of the sacrum or lesions of the cauda equina. Hysterical sciatica has been described.

**Symptoms.**—The chief symptom is pain along the course of the nerve, or limited to one of its chief branches. Occasionally the small sciatic and more rarely the anterior crural and perineal nerves are simultaneously affected. The onset is occasionally sudden and associated with slight pyrexia and constitutional disturbances, but, as a rule, it sets in gradually with pain in the buttock or back of the thigh in movements or in postures which make the nerve tense or cause pressure upon it. In other cases the onset of the typical severe pain is preceded by slighter diffuse pain or a feeling of discomfort during walking or after exercise. The pain increases gradually in severity; it may be either gnawing and burning, or sharp and darting in character. As a rule, it is constant, but severer paroxysms occur, either spontaneously or excited by movement of the affected limb, and its intensity generally increases at night. It may be at first limited to one portion of the nerve, generally that in the upper portion of the thigh, but as the disease develops it extends along the whole length of the sciatic trunk and its branches. Often bouts of pain occur which shoot from the buttock down the limb; such attacks may be described by the patients in similar terms to the lancinating pains of tabes dorsalis. It is usually most intense in certain points, as over the sciatic notch, in the middle of the thigh, in the popliteal region, below the head of the fibula, and behind the external malleolus; more rarely it is referred to the region of the postero-superior iliac crest, or is most severe in the foot. The seat of the chief pain is often, however, variable in any case from day to day. It is generally more or less accurately limited to the course of the nerve, but in other cases it is referred to its whole cutaneous distribution. Any movement which makes the nerve tense brings on a paroxysm. The most comfortable posture is lying on the back or on the affected side, with the thigh slightly flexed and the knee considerably bent, and when sitting the patient generally rests only on the tuber ischii of the unaffected side with the hip-joint of the painful limb extended as much as possible. In walking the hip and knee are held in moderate flexion with the foot extended at the ankle-joint, and only its toes and forepart touching the ground. Any sudden movement of the body may bring on an acute attack of pain, which, in many cases, is also increased by coughing and sneezing, and during defecation.

The disease is further characterized by the extreme tenderness of the nerve to pressure, rarely absent, except, according to Edinger, in those cases in which the sciatica is due to compression of the nerve by distended veins, and in which the spontaneous pain disappears when the patient lies at rest. In some cases the muscles of the limb are also tender to pressure. It is characteristic of the pain that it can be invariably produced by stretching the nerve; this can be most easily done by flexing the thigh with the knee

extended, or by extending the knee when the hip is flexed—*Lasegue's sign*, or, as Gowers has shown, by pressure on the nerve in the popliteal space as the patient sits in a chair with the knee flexed to a right angle; the pain which is produced by either of these means is felt not only at the point of pressure, but along the course of the nerve in the back of the thigh.

The pain is usually associated with paræsthesia. The muscles supplied by the sciatic nerve and its branches often become flabby and undergo a slight degree of general wasting, even in cases which are not due to any organic lesion, when the disease is of long duration; but in this class of cases there is no change in the electrical excitability of the muscles. Severe reflex spasms of the limb and cramps in some of its muscles, especially in the calves, are not infrequently observed.

Slight trophic and vasomotor disturbances are occasionally met with, but usually only pallor, dryness, and coldness of the skin. In other cases there may be an increase of the surface temperature, and of sweat secretion. Herpes has been observed. Scoliosis of the lumbar spine convex to the affected side is often seen, but its immediate cause is in dispute; it may be due to an attempt to spare the painful limb by tilting the centre of gravity toward the opposite side, or to a relaxation of the lumbosacral muscles of the affected side. All explanations meet with the difficulty that the scoliosis is occasionally in the opposite direction, that is, concave to the side affected. Kyphosis also occurs, but only rarely.

In addition to the pain and tenderness, evidence of organic lesion is present in a considerable proportion of the cases. There may be diminution of cutaneous sensibility, but, as a rule, it is nothing more than a blunting of tactile sensation on the back of the leg and on the foot. When there is an organic nerve lesion the hamstrings and leg muscles and occasionally the glutei may be found not merely flabby, but distinctly wasted and weak; but the feebleness of movement due to a true paresis must be distinguished from the reluctance of the patient to exert full power owing to the fear of pain. The most certain indication is the presence of qualitative changes in the electrical reactions of the wasted muscles, which must be regarded as proof of the existence of an organic lesion of the nerve. The third sign of the existence of organic disease in the nerve is the absence of the Achilles tendon-jerk. It seems very doubtful if this reflex ever disappears in the purely neuralgic cases; in fact, in many such cases it is very brisk or even exaggerated; as both the afferent and efferent paths of the reflex arc are contained in the sciatic nerve, its diminution or disappearance is one of the most delicate signs we possess of the presence of organic disease of the nerve; probably this may be excluded in all cases in which the reflex is undiminished. The reflex seems to be absent in about 30 to 40 per cent. of the cases which are clinically regarded as sciatica (Strasburger[1]).

In the great majority of the cases sciatica is unilateral, but it is occasionally bilateral, in about 7 per cent. of all cases according to Gibson's[2] statistics, though Hyde[3] has found it in 33 per cent. of his cases. This, as a rule, indicates a general and not a local exciting cause; it occurs frequently in diabetes, and may be a part of an incomplete general neuritis.

[1] *Deutsch. Zeit. f. Nerven.*, 1900, xvii, 306.
[2] *Lancet*, 1893, i, 860.
[3] *Ibid.*, 1896, i, 1281.

Sciatica has been regarded by some recent authors as a disease of the dorsal roots of the cauda equina. Dubois[1] pointed out in 1902 that the hypoæsthesia, when there is any, may correspond in extent to the distribution of one or more of the sacral roots, and more recently Lortat Jacob and Sabaréanu[2] have confirmed his observations. The sensory loss, according to these observers, most commonly coincides with the cutaneous areas of the last lumbar and the upper two sacral roots. In at least two of their six cases, however, there was an earlier syphilitic infection, and in one of these a lymphocytosis of the cerebrospinal fluid made probable the existence of a syphilitic meningitis. The proportion of cases in which definite evidence of radicular lesions can be found will be very small.

**Diagnosis.**—The term sciatica is applied by custom to cases in which the symptoms are due to an organic affection of the nerve, as well as to those in which there is no evidence of structural disease. The first step must be, however, to separate these two classes, *simple sciatica* or *sciatic neuralgia*, in which there is spontaneous pain and tenderness of the nerve to pressure and to tension, but no pronounced sensory disturbance, degenerative atrophy of the muscles, or diminution of the Achilles tendon-jerk; and *organic sciatica* or *sciatic neuritis*, in which some or all of these signs of disease of the nerve are present. Some authors, as Gowers, regard all cases of sciatica with persistent tenderness of the nerve as neuritic, and consequently make simple sciatica or sciatic neuralgia very rare; but in facial neuralgia, in which there is certainly, as a rule, no disease in the nerve, its trunks may be quite as tender to pressure as the nerve in sciatica. Oppenheim and others have rightly insisted that tenderness of the nerves is not a differential sign between neuritis and neuralgia. The pain of *hip disease* may radiate a short distance down the thigh; from sciatica it may be distinguished by the absence of tenderness in the nerve to pressure and tension, and the occurrence of pain on movement of the hip-joint, and on pressure on the trochanter. Disease of the *sacro-iliac synchondrosis*, and especially the pain which may arise from it during pregnancy, may be more difficult to recognize.

Although many cases of sciatica are due to a neuritic or morbid process in the nerve, the presence of marked sensory disturbance and degenerative atrophy of the muscles, with the absence of the Achilles tendon-jerk, should always raise the suspicion of more serious disease. If it is due to the compression of the nerve or its roots by *a tumor in the pelvis*, a rectal or vaginal examination may reveal the disease, and in these cases the sciatic trunk is not tender to pressure.

When the sacral roots are involved in *disease of the sacrum or the cauda equina*, the symptoms are generally bilateral and more irregular in distribution, and the functions of the sphincters are, as a rule, affected; the pain is also generally referred to the cutaneous distribution of the affected fibers; the sensory or motor disturbances correspond to root distribution, and the nerve trunk is not tender.

In the early stages of *tabes dorsalis* the shooting pains may be limited to the sciatic distribution, but a careful examination of the case will generally reveal characteristic signs of this disease; in it the pain is almost invariably bilateral, and the nerves are not tender to pressure or stretching.

[1] *Correspondenzblatt f. Schweizer-Aerzte*, 1902, xxxii, 366.
[2] *Revue de Méd.*, 1905, xxv, 917.

The pain of *intermittent claudication* occurs only after exercise, and is generally most intense in the distal segments of the limbs and is not limited to the course of the nerves.

**Prognosis.**—In simple sciatica this is good as regards ultimate recovery, but it is extremely difficult to predict the duration, although, as a general rule, it is proportional to the severity. Cases in which pain is associated with signs of an organic nerve lesion are less favorable than the uncomplicated neuralgic cases. The outlook is less favorable in cases of long duration and where adequate treatment is not possible. Where the sciatic pain is due to some lesion extrinsic to the nerve which compresses or injures it, the prognosis is naturally dependent on the nature of the primary disease. Relapses are unhappily not infrequent.

**Treatment.**—The first essential in all cases is *rest*. Even in mild cases this should be made as absolute as possible for some days at least, and if it is adopted early severe cases may be often converted into slight ones. The patient should be confined to bed and all movements of the affected limbs should be restricted as far as possible, if necessary by the use of a long splint reaching from the axilla to the foot if this can be borne. This rest treatment is applicable not merely to recent cases; obstinate and protracted cases which have resisted all other treatment often yield readily to it. It is often necessary to continue it four to six weeks, or even longer without break. Where it is not possible to obtain such complete rest an effort should be at least made to avoid all movements which give pain and cause stretching of or pressure on the nerve.

Constitutional conditions which are often predisposing causes should receive adequate treatment. In some of the acute cases the salicylates relieve the symptoms; and iron and arsenic often prove remarkably useful in cases with anæmia. The rectum should be emptied by mild purgatives as soon as the patient comes under treatment, and constipation should be carefully avoided.

In acute stages the application of hot poultices along the course of the nerve may ease the symptoms. Counterirritation is very generally employed, and often seems to be of distinct value, either by the application of the cautery or by blisters over the course of the nerve. Baths, douches, and especially the hot-air bath undoubtedly give relief and often influence the course very favorably, especially in its most chronic stages. When there is acute pain, symptomatic treatment may be forced into the first place. Antipyrine, phenacetin and such drugs often give relief for a time. Occasionally deep injections of morphine or cocaine into the nerve may be necessary, but they are only temporary measures and unfortunately, owing to the nature of the disease, its constant and protracted pain, and its tendency to relapse, there is always a considerable danger of the patient contracting a drug habit. Morphine is the most effective, but cocaine in doses of from $\frac{1}{8}$ to $\frac{1}{4}$ of a grain may abolish all pain for hours.

Favorable results have been recently obtained by the injection of relatively large quantities of normal saline solution (50 to 100 cc.) into the sheath of the nerve. The injection is made in the upper part of the thigh; severe pain referred peripheralward, paræsthesia, and muscular spasm of the limb, indicate when the needle enters the nerve. The acute pain disappears rapidly when the injection is commenced, which should be made slowly. More than two or three injections are rarely necessary, and often one is

sufficient. Bum[1] has recorded the result of this treatment in 73 cases; he obtained a complete cure in 42, and 14 of the others were much improved.

The galvanic current is often useful in the later stages; one large electrode should be placed over the nerve in the upper part of the thigh, the other on the leg over one of its branches, and a constant current of 3 to 5 milliamperes employed for five to twenty minutes at a time.

It is rarely advisable to use massage in acute cases, but when the muscles become flabby or atrophy, it is of service, but pressure or tension of the nerve must be carefully avoided. Acupuncture may be tried, though it is now not so fashionable a form of treatment as it was some years ago; a series of six or more carefully sterilized needles are thrust in to a depth of about two inches along the course of the nerve in the upper half of the thigh, and left there from twenty minutes to an hour. Many of the needles may pierce the nerve, but if they are inserted from above downward, only the first one causes much pain.

Nerve stretching has also fallen out of fashion, and probably rightly, but it may be necessary to have recourse to it in very obstinate cases. Bardenheuer has recently suggested cutting away the brim of the sciatic notch where the nerve passes over it, in order to leave the latter embedded in the soft tissues and free from pressure; he has claimed excellent results for this procedure.

**Diseases of the Spinal Roots.**—The spinal roots may be affected by disease either inside the dura mater, or in their extrathecal course before they anastomose to form the plexuses; the lesions of the latter portions have been considered under the plexus lesions, and the diseases of the cauda equina are dealt with in another section. The disease of the roots may be primary, or the symptoms may be caused by compression or extension of disease from the surrounding parts.

Primary root lesions are extremely rare; Farquhar Buzzard[2] has described under the term "*uniradicular palsies*," symptoms due to the affection of a single root, without evidence of any other disease. In some of these cases the onset was acute or sudden, and was attributed by the author to vascular lesions in the neighborhood of the ganglia or in the spinal nerve. One of his cases was associated with herpes zoster over the cutaneous distribution of the sensory root fibers, and was probably the result of the extension of the inflammatory process from the intervertebral ganglion to the motor fibers as they join the sensory to form the mixed nerve; such radicular palsies are not infrequently observed in the course of herpes zoster. One root alone may be injured by a tumor in the neighborhood of the vertebral column; it frequently happens with malignant disease in this region, or spinal caries. Supernumerary ribs may produce an isolated palsy of the first thoracic nerve. But as Dejerine and his pupils have pointed out, root lesions are more frequently secondary to intradural disease, and especially to compression and the invasion of them by a local tuberculous or syphilitic meningitis.

The distinguishing feature of all root lesions is the limitation of the symptoms to disturbance of the functions of the motor or sensory fibers of the root or roots affected; this distinguishes root lesions from lesions of the peripheral nerves, which almost invariably contain fibers of two or more roots. The

[1] *Wien. med. Presse*, 1907, xlviii, 1660.
[2] *Brain*, 1902, xxv, 291.

onset of the symptoms, which may be either acute or slow, is generally with pain and paræsthesia referred to the peripheral distribution of the sensory fibers involved; at this stage the skin of this region may be hyperæsthetic. The pain, which is generally at first paroxysmal but may become continuous, is, as a rule, very severe and of the same darting or shooting character as the pains of tabes dorsalis. If it is the intradural portions of the roots which are affected, sneezing or coughing may produce very acute pain in the affected root areas—*signe de l'eternuement*—owing to the effect of the sudden increase of the intradural pressure on the diseased and irritable fibers. After a variable time the pain gradually diminishes, and the skin which was previously hyperæsthetic becomes hypæsthetic as the sensory root fibers degenerate or are destroyed. When only one root is involved this diminution of sensibility may escape notice owing to the considerable overlap of the adjacent root fibers; it is largely dissociative in character, the loss of pain sensation being, in contrast to the condition found after peripheral nerve lesions, more extensive than the insensibility to light touch (Head). Simultaneously with the appearance of hypæsthesia, the muscles supplied by fibers from the affected ventral root or roots become paretic, and if the lesion is sufficiently intense they atrophy, and changes in their electrical reactions develop; but as almost all muscles receive fibers from two or more roots the paralysis of any muscle is rarely complete if the lesion is uniradicular. Cutaneous trophic changes, as herpes and purpura, have been observed in the area of sensory distribution of the affected roots, probably in cases in which the root ganglia have been involved, and the ocular symptoms of irritation or paresis of the cervical sympathetic fibers may appear when the lower cervical and upper dorsal roots are affected.

Dejerine and Egger[1] have described a case of generalized radicular neuritis of chronic course, in which the chief symptoms were violent lancinating pains, and sensory loss with paresis and muscular atrophy corresponding to the distribution of the affected roots, but the diagnosis has not yet been verified.

**Diagnosis.**—This depends on the essentially radicular distribution of the symptoms. Spinal diseases, as tabes dorsalis, syringomyelia, and local lesions, may produce symptoms of this distribution, but these can rarely offer any difficulty. From local neuritis of a peripheral nerve it may be also distinguished by the fact that the nerve trunks are never very tender to pressure. When the primary disease is a meningitis which invades or constricts the roots, lumbar puncture may aid in determining its nature.

**Treatment.**—Treatment should be in the first place directed to removing the cause; surgical intervention may be successful in the case of tumors, and if there are other symptoms of meningitis vigorous antisyphilitic treatment should be adopted if syphilitic infection cannot be excluded with certainty. In cases with persistent pain the intradural section of the dorsal roots may be necessary.

## NEUROMATA.

The peripheral nerves may be compressed and destroyed by tumors in the tissues through which they pass, or invaded by the infective granulomata, or by metastases of malignant tumors. Almost a century ago Odier recog-

[1] *Revue neurologique*, 1904, No. 11.

nized that tumors may grow from the nerves themselves, and to these he gave the name *neuromata*. This was for long applied to all tumors seated in nerves, regardless of their histological structure; Virchow was the first who attempted to separate those which are composed of nerve substance proper, which he called *true neuromata*, from those which develop from the connective tissue of the nerves and contain no newly formed nerve fibers, *false neuromata*.

*True neuromata*, as defined by Virchow, are, however, exceedingly rare; they are found practically only in connection with the sympathetic system in the thoracic or abdominal cavities. They contain, as a rule, ganglion cells and non-myelinated nerve fibers. Although benign in nature, they may infiltrate the surrounding tissues. They produce nervous symptoms only by compression of nerves, and are not tender to pressure. Two cases have been recorded, by Knauss[1] and Kredel-Benecke,[2] in which there were multiple true neuromata in the subcutaneous tissues, but apparently most of the others which have been described have been solitary. They are met with only in early life.

*False neuromata*, which develop from the connective tissue of the nerves, are much more common. Their etiology is obscure; they generally occur in the first half of life, and in many instances they have been congenital. Occasionally they are hereditary; Petrén[3] found that in 10 of the 60 cases of multiple neuromata which he collected from the literature there was definite evidence of the existence of similar tumors in the ascendant or collateral lines, while in 25 per cent. of all the cases there was an hereditary neuropathic taint. The appearance of neuromata has followed trauma of a nerve, but the false neuromata which are considered here must be sharply separated from the bulbous swellings in the ends of the proximal portions of nerves which have been divided. Other cases have been attributed to infections and to arsenical poisoning.

**Pathology.**—As these tumors grow from the fibrous tissue of the nerves they are generally fibromata and of a benign nature, but they occasionally undergo myxomatous or cystic degeneration. More rarely they become sarcomatous; this malignant metamorphosis is frequently observed when part of a tumor has been removed by operation or otherwise injured. Gliomata and lipomata have been described. The neuro-fibromata may originate from the epineurium, in which case the tumor generally lies on one side of the nerve and scarcely interferes with it, or from the perineurium or endoneurium; in the latter case it penetrates between and splits up the nerve into its secondary bundles. The structure of the nerve fibers which pass through the tumor is, as a rule, unaltered, but there may be partial or complete disappearance of the myelin sheaths, while the axis-cylinders remain intact. Neuro-fibromata may be found on any of the spinal or cranial nerves, or on the intramedullary portions of the nerve roots. They vary in size from that of a pin-head to several inches in diameter; the smaller are generally spindle-shaped and elongated in the axis of the nerve from which they grow. There may be only a solitary growth on one nerve, or thousands may be distributed on all the somatic and visceral nerves.

[1] *Virchow's Archiv*, 1898, cliii, 29.
[2] *Deutsch. Zeit. f. Chir.*, 1902, lxvii, 239.
[3] *Nordiskt Med. Arkiv*, Axel Key, Festband, 1899.

**Solitary Neuromata.**—Symptoms are more frequently produced by solitary and isolated than by multiple neuromata, but many never cause inconvenience to the patient. The most prominent feature is pain which radiates peripheralward from the tumor along the nerve and its branches; it is generally intermittent, but may become continuous and so severe as to merit Smith's description of it as "agonizing torture." Movement of the limb and palpation of the tumor, which is generally tender to pressure, may induce an attack of pain, and, as in neuralgia, the influence of the weather may be sometimes observed. It has been for long known that compression of the nerve involved proximalward to the tumor can relieve the pain. Cases have been described in which epileptic convulsions have been attributed to the irritative symptoms of neuromata, and in which the fits have ceased after their removal. Paræsthesia occasionally occurs in the distribution of the sensory fibers which are affected, but loss of sensation and muscular paresis are rarely due to simple neuro-fibromata; reflex muscular spasms are sometimes seen. When palpable, the tumors are firm and evidently circumscribed, and it is a characteristic feature that they are movable in the transverse but not in the longitudinal axis of the nerve on which they are situated. Neuromata of the sheath of the optic nerve produce loss of vision, exophthalmos, limitation of the ocular movements, and pain in the eye; and tumors on any of the cranial nerves may give rise to the symptoms of complete or incomplete lesions of these nerves.

The only satisfactory *treatment* is removal of the tumors which produce inconvenient symptoms. This is rarely a serious operation, but resection of the nerve and primary suture of its divided ends may be necessary. Rapid growth, adhesion to or infiltration of the surrounding tissues, and evidence of acute destruction of nerve fibers indicate malignancy; amputation of the segment of the limb involved may be then necessary. The spontaneous regression of neuro-fibromata has been observed.

**Tubercula Dolorosa.**—Tubercula dolorosa, or the painful subcutaneous tubercles of Wood, may be either solitary or multiple; they are generally very small and lie directly under the skin. They grow slowly and are always benign. As a rule, they are neuro-fibromata, but are occasionally lipomata or sweat-gland adenomata which develop in connection with the smaller cutaneous nerves. They are generally exquisitely tender to pressure and may give rise to spontaneous pain. When troublesome they should be excised.

**Multiple neuromata or general neurofibromatosis** is a condition in which a large number of tumors is found on many or all the peripheral nerves, and even in the sympathetic system; R. W. Smith[1] has recorded a case in which 450 were counted in one limb and over 2000 were present in the body. They are nearly always simple fibromata, but may become malignant. The condition is distinguished from the solitary neuromata, of which there may be more than one on a nerve, by the presence of a diffuse hyperplasia of the connective tissue of the nerves between the tumors, so that they can be felt or seen through the skin as thickened and beaded structures. They are rarely tender to pressure, and do not, as a rule, produce pronounced symptoms. Occasionally they give rise to local, or to vague and wandering pains,

[1] *A Treatise on Neuroma*, Dublin, 1849, reprinted by the New Sydenham Society, London, 1898.

but they scarcely ever cause paralysis or serious sensory loss. Petrén has observed one case, and has collected others in which there was motor incoordination, probably secondary to loss of deep sensibility. These cases consequently resemble progressive interstitial hypertrophic neuritis. As a rule, inconvenience results only from the size of the tumors, although when seated on the spinal roots they may compress the spinal cord or the cauda equina, and within the skull they may give rise to the symptoms of intracranial tumor. All forms of neurofibromatosis are frequently associated with cretinism, idiocy, or other mental deficiencies.

**Plexiform Neuromata.**—In this condition there is a diffuse hypertrophy of the connective tissue of all branches of a nerve, and the whole being embedded in fat or loose connective tissue gives rise to the appearance of a tumor. Occasionally they are pedunculated and hang in a sac of skin. They occur most frequently in the head and neck and are often congenital. They are not tender to pressure, and as they rarely produce nervous symptoms they belong to the domain of surgery rather than to neurology.

**Molluscum fibrosum, or von Recklinghausen's disease,** is another form of neuro-fibromatosis which is characterized by numerous sessile or pedunculated cutaneous nodules which project from the surface. They are very variable in size, and may be either soft or hard, and are occasionally lobulated. The nodules are generally most numerous in the trunk and scalp, and are very rarely seen in the hands or feet. As von Recklinghausen first demonstrated, the nodules are neurofibromata of the terminal branches of the cutaneous nerves, but the fibrous tissue of the cutaneous vessels and glands may thicken and take part in their formation. There are rarely nervous symptoms, and the nodules are not tender. The disease is generally congenital, and is most frequently seen in degenerate types. It is often associated with nævi and skin pigmentation, and these are generally neuromata on the deeper nerves.

In the condition known as *elephantiasis neuromatosa* there is, in addition to a fibromatosis of the cutaneous nerves, a diffuse thickening of the skin, subcutaneous tissues, and even of the bone, of some part of the body, generally of one lower limb. Symptoms rarely result from affection of the nerves. The condition is usually congenital, and is frequently associated with pigmentation of the skin and other abnormalities. It may be distinguished from true elephantiasis by the absence of œdema and by the thickening of the nerves which are palpable.

## HERPES ZOSTER.

**Definition.**—Herpes zoster is an affection characterized by the appearance of erythema and cutaneous vesicles on the cutaneous distribution of the fibers of one or more dorsal roots.

**Etiology.**—The researches of Head and Campbell have made it probable that idiopathic herpes is an acute specific disease of the nervous system. This view is supported by the fact that it is occasionally epidemic and may have a seasonal prevalence, but no specific organism has yet been isolated. It occurs occasionally during the course of general infectious disease and in diseases in which there is an increased susceptibility to infection, as in general paralysis of the insane. It may also result from invasion of the dorsal root ganglia by tumors, or from the extension into them of a tuberculous process

from spinal caries, or of inflammation from the meninges; it has been repeatedly observed in the course of acute cerebrospinal meningitis. In one case of herpes of the fourth thoracic segment the writer found a small metastatic abscess in the corresponding intervertebral ganglion. Herpes has been attributed to general intoxications, including arsenical poisoning.

**Pathology.**—Bärensprung, in 1861, first demonstrated changes in the intervertebral ganglia in a case of herpes zoster, but it was Head and Campbell[1] who definitely established the pathology. Their investigations showed that the herpetic eruption is always associated with acute interstitial inflammation of the dorsal root ganglia, or of their homologues on the cranial sensory nerves. In the earlier stages of the disease the affected ganglion is found infiltrated by small round cells; later part of the ganglion tissue is generally destroyed by the intensity of the inflammation, and many of its cells undergo acute necrosis; hemorrhages almost invariably occur in the inflamed area. The sheath also is generally involved, and the whole ganglion is swollen and hyperæmic. When the inflammatory products are absorbed the disease may be represented only by a small area of scar tissue, or there may be cyst formation, but in the slighter affections there may be no permanent changes in the ganglion. The affection is not, as a rule, so localized as the eruption would indicate; in one case, although the eruption was limited to the third sacral root area, cellular infiltration was present not merely in the corresponding ganglion, but all in the neighboring ones of the same and of the opposite side. The inflammation may extend a short distance into the peripheral nerves or into the dorsal roots. Attention has been recently directed to the occurrence of foci of hyperæmia and inflammation in the spinal cord associated with herpes zoster.[2] When cells of the ganglion are destroyed their processes in the peripheral nerves and dorsal roots naturally undergo secondary degeneration. There is a striking similarity between the changes in the intervertebral ganglia in herpes and the affection of the spinal cord in acute poliomyelitis.

**Symptoms.**—Idiopathic herpes often sets in with fever and general malaise, which may last for three to five days, but the rise of temperature is, as a rule, slight. Occasionally, there are gastric disturbance and other constitutional symptoms, and enlargement of lymphatic glands has been observed. The eruption, which generally occurs on the third or fourth day of the disease, is often preceded by pain and hyperalgesia in the affected area. The skin becomes erythematous and finally vesicles develop, either while there is still fever, or as the temperature falls. The special characteristic of the disease is the limitation of the vesicles to the cutaneous distribution of one dorsal root, or more rarely of two or more adjacent roots. The eruption is not, however, uniform over this region, but is made up of a series of outbursts which follow the course of the small cutaneous nerves; there is a variable extent of overlap between adjacent herpetic areas. Herpes is much more frequent on the trunk along the course of the intercostal nerves than on the limbs; according to Head those ganglia are most often affected which receive visceral sympathetic fibers. After a time the vesicles dry up and disappear, but their distribution is generally permanently marked by small, slightly depressed scars in the epidermis. On the face herpes is most

[1] *Brain*, 1900, xxiii, 353.
[2] Thomas et Laminière, *Rev. neurol.*, 1907, xv, 693.

common on the cutaneous distribution of the ophthalmic division of the trigeminal nerve; when it occurs here vesicles may form on the cornea and lead to troublesome scarring and faceting.

The most prominent symptom is pain referred to the region of the eruption. The pre-herpetic pain which usually ushers in the attack is generally of a burning or stabbing nature; it may be extremely severe, but generally abates with the appearance of the rash. The post-herpetic pain or neuralgia is much more serious; it is also referred to the region of the eruption, but frequently spreads to adjacent segments; it is so severe and so intractable to treatment that it has driven the subject to suicide more than once. In a large proportion of the cases some loss of sensibility in the affected area follows an herpetic eruption, but in others sensation is unaffected. Petrén[1] has recently shown that there may be marked or even complete loss of pain and temperature sensibility, with tactile sensation unimpaired; and he has drawn attention to the fact that post-herpetic neuralgia occurs chiefly in those cases in which there is considerable or complete analgesia. The sensory loss is generally uniform over the whole of the affected root area. A not uncommon complication is muscular paralysis of radicular distribution. Doucet[2] has recently collected 40 such cases. The palsy generally occurs in the neighborhood of the eruption, and is probably due to involvement of the ventral spinal root in the inflammatory process as it lies beside the affected ganglion; or occasionally perhaps to central lesions of toxic or infective origin. The facial nerve is perhaps the most frequently affected, generally in association with trigeminal or occipito-cervical herpes; it is, however, probably due to compression of the nerve in the aqueduct of Fallopius by a swollen geniculate ganglion which is simultaneously involved by herpes (Ramsay Hunt).[3] Paralysis of the ocular nerves has also been observed. In a few cases slight paraplegia has developed, as in a case recently published by Bruce;[4] it is probably due to extension of the disease to, or to the appearance of an independent focus in, the spinal cord.

**Treatment.**—The pain may be relieved by drugs in the earlier stage of the disease, but post-herpetic neuralgia is very intractable to all local and general treatment; relief can be promised only by section of the dorsal spinal root corresponding to the ganglion affected, or by extirpation of the Gasserian ganglion where pain follows trigeminal herpes.

## MULTIPLE NEURITIS.

*Multiple neuritis*, *peripheral neuritis*, and *polyneuritis* are the terms applied to a group of diseases which are due to affections of the peripheral nerves, or rather of the peripheral motor and sensory neurones. These conditions are distinguished from paralysis limited to a nerve, or a group of nerves in close anatomical relation, by the fact that several nerves are affected simultaneously or in rapid succession (therefore multiple neuritis), that the condition is always bilateral and more or less symmetrical, and that the longer fibers of the nerves which extend to the periphery of the limbs suffer more

[1] *Zeit. f. klin. Med.*, 1907, lxiii, 91.
[2] *Le Zona associé aux paralysies et aux amyotrophies*, Thèse de Paris, 1906.
[3] *Journal of Nervous and Mental Disease*, 1907, xxxiv, p. 73.
[4] *Revue of Neurology and Psychiatrie*, 1907, v, p. 885.

severely than the shorter fibers which are distributed to the proximal segments, or to the muscles and sensory structures of the trunk (therefore peripheral neuritis). The disease is characterized by the fact that it is limited to the peripheral neurones, while the central nervous system is intact, or any changes which may occur in it are merely a coincident effect and of no significance in the production of the clinical picture.

The term neuritis would indicate that the condition to which it is applied is an inflammatory process, but this is not so in multiple neuritis; as a rule, little or no evidence of inflammation can be found in the affected nerves even in the severest types, and where there is inflammation it probably only represents a reaction of the connective tissues to the degeneration of the nerve fibers, or the result of an attempt to remove the products of degeneration. Degeneration of the parenchyma of the nerves, due to the direct action of a poison or other noxious agent on them, is the primary and essential change.

**Historical.**—Although multiple neuritis is one of the most common of nervous diseases, it was not definitely recognized until late in the last century. Its clinical features were fairly accurately described by many of the older observers, and it is true that Jackson, of Boston, Graves, Todd, and others suspected that alcoholic and lead palsies depended on disease of the peripheral nerves and not on affections of the brain or spinal cord, but it was not until 1864 that Deménil,[1] of Rouen, demonstrated changes in the peripheral nerves in relationship with the symptoms of neuritis. Several similar observations were recorded during the next sixteen years, but it was first in 1880 that multiple neuritis as we know it now was definitely separated by von Leyden,[2] from those forms of flaccid and atrophic paralysis which result from spinal disease, and especially from acute poliomyelitis. The work of Joffroy and others in France, and of Ross and Buzzard in England contributed largely to this. Within the next few years the clinical features of almost all forms of multiple neuritis were recognized; recent work has been chiefly directed to the investigation of its causes and the essential nature of the pathological changes. It has been only lately that Landry's paralysis has been recognized as a distinct and separate form of disease, and that Raynaud's disease and erythromelalgia, in which the symptoms are also limited to, or more intense in, the periphery of the limbs, have been definitely separated.

**Classification.**—A pathological classification is insufficient, as there are only two distinct types of pathological change; in the one the primary and essential process is a degeneration of the parenchyma or functional portion of the peripheral nerves; in the other it is an inflammatory or simple hypertrophic process of the connective tissue in which the fibers are ensheathed. But primary interstitial neuritis is extremely rare, and exists practically only as part of an infective disease—namely, leprosy—while the great majority of the cases which are seen are instances of parenchymatous neuritis, and it is impossible to subdivide this enormous group satisfactorily, as there are no sharp and essential differences in the neural changes in varieties due to different causes or characterized by special symptoms.

The classification by the clinical symptoms is equally unsatisfactory. These may consist only of motor phenomena, or of disturbance of sensation,

[1] *Gazette hebd.*, 1864, p. 203.

[2] *Zeit. f. klin. Med.*, 1880, i, 387.

or vasomotor symptoms may be the most prominent feature; but we rarely find that there are only motor, or sensory, or vasomotor symptoms; in fact, there is almost invariably disturbance of all the functions of the nerves affected, and affection of any of these functions may be the dominant symptom in different cases due to the same cause. It is, therefore, inadvisable to classify cases as of the motor, sensory, or vasomotor type.

An etiological classification is usually adopted and is the most useful, as it brings into prominence the cause of each case, and when multiple neuritis has been diagnosed the next step is to ascertain its cause. But an etiological classification cannot express the prominent features of a case, for though certain poisons may have an affinity for nerve fibers with certain functions, such a selective action is not always apparent.

**Etiology.**—The wide distribution of the affection and its symmetrical regularity indicate, as Gowers points out, that multiple neuritis must be directly due to an altered blood or lymph state which has equal access to all parts of the body, and, indeed, a study of the disease brings out very clearly that its cause is invariably some poison which is introduced into the body from without, or produced within it by infective microörganisms or by a derangement of normal metabolism. It is noteworthy how many poisons different in nature and in chemical constitution possess such a strong affinity for the peripheral nerves. Many of these, especially those of inorganic nature and of simple chemical constitution, seem to have a selective action on the peripheral nerves and may produce disturbance of function in no other organ of the body; motor palsy, for instance, may be the only symptom of chronic lead poisoning. But in the majority of the cases which are due to bacterial toxins or to poisons of complex chemical nature the symptoms of multiple neuritis are only part of the result of a general infection or intoxication, or, as is the rule, in the acute specific fevers, the neuritis may appear only when the symptoms of the general infection are on the wane or passing off. This suggests that the substance that affects the peripheral nerves may not be always the same as that which produces the primary disease, but some body derived from it or a product of the disturbance of the general metabolism.

There is a group of infective diseases in which a peripheral neuritis may be the only or the most prominent symptom. Beriberi is the best known example, but there are undoubtedly other, as yet ill-defined or scarcely recognized, infectious conditions which may manifest themselves only by the symptoms of a primary multiple neuritis.

It not infrequently happens that there are two or more factors at work, predisposing as well as directly exciting causes. Neuritis occasionally develops in the course of pulmonary tuberculosis, and although it is probable that it may be at times due to the action of the tuberculous toxin alone, it is very rarely that the influence of alcohol can be excluded. Similarly, quite a series of cases of neuritis have been observed after the administration of phosphate of creasote to patients with phthisis;[1] here the tuberculosis was probably the predisposing, the drug the exciting, cause. In the extensive epidemic of peripheral neuritis in the north of England some years ago, which was caused by the contamination of beer with arsenic, it is probable that there were also two distinct but coincident etiological factors, namely, alcohol

[1] Huet, *Neurol. Centralbl.*, 1907, xxvi, p. 60.

and arsenic. Finally, there can be no doubt that most marasmic and cachectic conditions make the nerves unduly vulnerable to noxious agents.

1. **Neuritis Due to Poisons Introduced into the Body from Without.**—These poisons may be either: (*a*) *Metallic or inorganic substances*, as lead, arsenic, mercury, copper, carbon monoxide, or bisulphide of carbon; or (*b*) *organic substances*, as alcohol, dinitrobenzene, various aniline compounds and derivatives, sulphonal, and other drugs; finally, the poison may be of more complex constitution, as the toxins of ptomaine poisoning. It is noteworthy that neuritis due to any of these causes, except perhaps carbon monoxide, is generally associated with chronic intoxication and rarely follows a single administration of the poison, no matter how large the dose.

2. **Neuritis Secondary to, or Associated with, Diseases Due to Toxins Produced within the Body.**—These toxins may result from: (*a*) *Bacterial infections*, most frequently diphtheria, smallpox, typhoid, and scarlet fever. The toxins produced by septic processes may also give rise to a multiple neuritis, as is the case in puerperal neuritis; (*b*) *derangement of metabolism and auto-intoxication*, as in diabetes and other constitutional diseases. As a rule, these conditions act more as predisposing factors than exciting causes.

3. **Toxins Produced within the Body Which Have a Primary Action on the Peripheral Nerves.**—A variety of neuritis which sets in with malaise and fever and more or less severe constitutional symptoms is not infrequently observed, and probably comes into this group (acute febrile polyneuritis). It is often called post-influenzal, and although the influenza bacillus or its toxins may be occasionally the exciting cause, it seems doubtful if it is often so. These cases are distinguished by the severity of the neuritis as contrasted with the slight constitutional disturbances which precede its onset, or the complete absence of prodromal symptoms. Occasionally exposure to cold is the only apparent exciting cause in cases which probably belong here.

4. **Neuritis associated with cachexia or malnutrition,** such as occurs in anæmia, tuberculosis, syphilis, malignant disease, and in old age. These may be directly due to auto-intoxication, or in some cases, as in tuberculosis, to the action of the bacterial toxins which produce the cachexia. In other cases the general malnutrition merely leads to an increased vulnerability of the nerves to all noxious influences; or the nutrition of the nerves may be directly interfered with, as in severe peripheral arteriosclerosis.

5. **Neuritis Due to the Invasion of the Peripheral Nerves by Bacteria.**—Leprosy is the classical example of this type. Nerves involved in septic wounds may be invaded by pyogenic bacteria which occasionally extend along the trunk and may involve adjacent nerves—*ascending neuritis*—but the picture of multiple neuritis can scarcely arise. Gonorrhœa may also lead to interstitial multiple neuritis (Frisco), or to acute neuritis, either with or without arthritic disease.

**Incidence.**—Multiple neuritis is essentially a disease of adult life. It occurs rarely in childhood, and then practically only after the acute specific fevers, especially diphtheria. It may follow the treatment of chorea with "heroic" doses of arsenic. Polyneuritis is also uncommon in advanced age, and when it occurs is usually slight in degree. Hereditary influences are rarely observed. The disease is seen more commonly in females than in males, but probably this is entirely due to their greater liability to alcoholic neuritis.

There are many factors which may determine the localization or incidence

of the disease. Perhaps the most important of these is the effect of overwork or exhaustion, especially when the nutrition is subnormal or when the metabolic equilibrium is otherwise upset. Thus Edinger explains the predominant paralysis of the extensors of the wrist and fingers in lead palsy as due to the exceptional stress to which these muscles are exposed in painters; in compositors, on the other hand, who also not infrequently suffer with lead palsy, the paralysis is, according to him, often limited to the small hand muscles with which they chiefly work. Occasionally the mode of the infection determines the type of the palsy; the soft palate is generally the first organ affected in diphtheritic neuritis following pharyngeal diphtheria, while the abdominal muscles were the earliest and most severely paralyzed in a few cases in which the seat of infection was the umbilicus in infants.

**Pathology.**—The pathological changes in multiple neuritis differ from those of local neuritis not only in their distribution, but also in their nature. In the latter, the primary changes affect the interstitial tissues, and are generally of an inflammatory nature; the condition produced is consequently a local interstitial neuritis. Multiple neuritis, on the other hand, is the result of degenerative, parenchymatous changes in the peripheral nerves.

The amount of structural change is very variable; it depends not only on the intensity of the disease, but also on its cause and nature, and particularly on its duration. To the naked eye there is rarely any definite abnormality, although in the acuter cases in which the primary disease has lead to a reaction in the interstitial tissues, the nerves may appear red and swollen, and there may be distinct hyperæmia of their sheaths. The swelling is due, in the acute cases, to serous effusion into them, and they are consequently soft to touch. These changes, when present, are generally most distinct in the smaller peripheral branches. In chronic cases, the nerves may be reduced in size and unusually firm and opaque. Microscopic examination shows that the myelin sheaths are broken up and disintegrated into irregular masses which stain black with osmic acid; in cases of longer duration this fatty degeneration product may have been already removed. In the early stage the axis-cylinders may be intact; more commonly they are swollen and varicose and stain feebly, or if the neuritic process is further advanced they may be broken or completely disintegrated. While these changes in the axis-cylinder and its myelin sheath are evidently the result of a degenerative or necrotic process, those in the neurilemmal sheath are of the nature of reactionary changes. The protoplasm around the nuclei increases in amount and penetrates between the fragments of the disintegrating myelin, and the nuclei proliferate by direct division. Some of these neurilemmal nuclei form independent cells which assume phagocytic functions and help to remove the degenerated neural products, or become fibroblasts and lead to the formation of an excess of connective tissue in the degenerated nerve; but others fuse end to end to form those nucleated strands of undifferentiated protoplasm into which, when regeneration commences, the new outgrowing axis-cylinders penetrate.

Even in parenchymatous neuritis there is frequently some evidence of inflammatory or reactionary changes in the interstitial tissue; the vessels become congested and there is often some serous effusion, and occasionally a small round-cell infiltration. These are identical with what may be seen in the supporting tissue of any organ when the parenchyma or functional

tissue degenerates, and can be regarded only as a compensatory reaction of the supporting tissues.

But these neuritic changes differ from secondary or Wallerian degeneration of nerve fibers in many important particulars. In the first place, they are always most advanced in the most distal segments of the nerves—multiple neuritis has been consequently aptly called peripheral neuritis—and the intensity of the changes always diminishes steadily proximalward; indeed, it is the rule that except in the severest cases the larger nerve trunks, as the sciatic, contain a much smaller proportion of degenerated fibers than their peripheral muscular or cutaneous branches, while in the dorsal and ventral spinal roots there may be no degenerated fibers. Secondly, the morbid changes are much less uniform than in fibers which are undergoing secondary degeneration. In almost all cases of toxic parenchymatous neuritis there are patches of that form of segmental periaxial neuritis which was originally described by Gombault, in which the disease is limited, or practically limited, to a segment of the myelin sheath; in its slighter forms the axis cylinder is not interrupted and the portion of the fiber distal to the lesion does not undergo secondary degeneration, and may be perfectly normal. Numerous transition stages may be found between these local lesions and the diffuse degenerative changes. Thirdly, not only is the affection of any one fiber irregular and diffuse, but the number of fibers affected in any nerve is extremely variable; even in the severest cases it is rare to find all the fibers of even the smaller nerves degenerated.

The interstitial changes are not without importance; the extreme tenderness of the nerves has been ascribed to this, but it seems extremely doubtful if it is so, as the affected nerves are almost invariably tender to pressure, while the presence of such inflammatory changes is very inconstant. When, however, they are pronounced they may lead to the formation of an excess of connective tissue in the nerves, which ultimately replaces the degenerate fibers and may offer a serious obstacle to their regeneration, or may even interfere with the vitality of those which were originally unaffected.

Changes are occasionally present in the central nervous system; when the dorsal roots are involved degenerated fibers will be naturally found in the dorsal columns of the spinal cord; but multiple neuritis is essentially due to toxic causes, and the dorsal column fibers are extremely liable to degenerate in all forms of general intoxication. More important and much more constant are the changes in the large ventral horn cells; they were present in some degree in every case of multiple neuritis examined by the writer. They may be described as chromatolysis of the cells; these become swollen and globular in shape, the tigroid or Nissl bodies break up and disappear, vacuoles frequently form in the cytoplasm, and the nucleus is displaced from the centre of the cell. Similar changes may be generally found in the cells of the dorsal root ganglia, and not infrequently in other parts of the central nervous system, as in the cerebral cortex; the association of mental disturbances with multiple neuritis may be the clinical manifestation of the affection of the latter region (Bonhœffer). These changes are not necessarily fatal nor is the condition of the cell irreparable; in fact, it is rare to find a completely degenerated cell in any part of the nervous system in multiple neuritis. Small inflammatory foci and minute hemorrhages have been described in the spinal cord, but have been much too slight and inconstant to play any part in the production of

the symptoms. They as well as the diffuse changes in all parts of the nervous system can be only attributed to the action of the toxin.

The nature of the marked alterations in the large motor ventral horn cells, and their relation to the neuritic changes, has been much discussed. There are evidently three possibilities: In the first place that the cell changes are the primary effect of the action of the noxious agent which produces the neuritis, and that the degenerative lesions in the nerve fibers are secondary to the affection of their trophic centre; this hypothesis seems scarcely tenable, as there is no constant relation between the cellular and fiber lesions; in fact the former are occasionally absent, and it is well known that the cell disease must be very acute to produce an acute degeneration of its axis cylinder. Further, the nerves are so diffusely and irregularly affected, and the disease is so frequently limited to their distal extremities that it is extremely improbable that it can be secondary to an acute cell lesion. In favor of this view it has been argued that the portions of the neurone most distant from their trophic cells must be the first portions to degenerate when the vitality of the cell is lowered. Even though this view may not give sufficient support to the hypothesis that the cell changes are the primary lesions in multiple neuritis, it explains very satisfactorily the chief incidence of the disease on the peripheral segments of the nerves.

The second possibility is that the cell changes are secondary to the degeneration of the fibers—that they are comparable to the retrograde chromatolysis which may be seen in any cell after its axis-cylinder has been cut across. This may be an important factor, but it is improbable that it is the only one, as it must be recognized that there is no close parallel between the neural and the cellular changes, and that the cellular alterations do not always conform to the type of retrograde chromatolysis. The third possibility, and the most acceptable, is that all portions of the peripheral motor and sensory neurones are simultaneously affected, but the most prominent alterations are found in their least resistant portions, namely, the terminations of the fibers which are furthest removed from the cell body.

Degenerative changes are not infrequently found in normal peripheral nerves, or, at least, in nerves in which there was no evidence of disease during life, but only very few fibers are generally involved. Mayer,[1] who first drew attention to this fact, offered in explanation the hypothesis that nerve fibers are not stable structures, but liable to regression and capable of regeneration. Lugano,[2] however, regards these degenerative changes as the result of antemortem infections or intoxications. It is certainly beyond dispute that extensive neuritic changes may be found in cases which die in infective or cachectic states, although during life there were no signs of neuritis.

The importance of the muscular changes must not be lost sight of. The degree of change depends on the severity and the acuteness of the neuritis, but in addition to the degenerative or regressive changes, such as succeed section of a nerve, there is also often fatty, granular, or waxy degeneration of the muscle fibers, a result of the direct action of the toxin. There is frequently, too, evidence of inflammatory processes in the interstitial tissues of the muscles, as hyperæmia and congestion, serous exudation and small

[1] *Zeit. f. Heilk.*, 1881, ii, 154.
[2] *Handb. der patholog. Anat. des Nervensystems*, ii, p. 1118.

round-cell infiltration; in fact, a condition of interstitial myositis. It is probable that this vascular reaction is largely secondary to the neuritic muscular degeneration, but it may be in part independent of it. Its significance lies in the fact that it may lead to the development of an excess of fibrous tissue in the muscles and produce contractures.

**Symptoms.**—It seems advisable to describe fully one form in which all symptoms may be found in various combinations; other varieties of the disease will then need only short references.

Alcoholic neuritis is much the most frequent form—in Great Britain, at least, it is probably much more common than all the other varieties put together, and may be, therefore, taken as a type. In most cases all the functions of the peripheral nerves are affected, but occasionally the symptoms are limited to either motor or sensory disturbances, or incoördination of movement may be the most prominent feature; the latter type has been called *neuro-tabes peripherica*, or alcoholic pseudo-tabes. Alcoholic neuritis occurs practically only in middle adult life, but a few cases have been recorded in the first decade; in these the disease has generally developed after a single large dose of the poison, as in that reported by Herter. In England and America it occurs much more frequently in women than in men, according to Gowers in about the proportion of 3 to 1; in Germany, on the other hand, from the statistics of Remak, it seems to be more common in males. The social habits of the people must largely determine its incidence, but women undoubtedly seem more vulnerable to the poison.

It usually results from the use of the stronger forms of alcohol, especially from spirits, but it is seen not infrequently in beer drinkers, and especially in constant and steady tipplers. Undoubtedly, it affects chiefly those who habitually take a considerable amount of alcohol, but are rarely drunk; occasionally, however, an attack may follow a severe bout of drinking or delirium tremens. There is frequently some exciting or predisposing cause in addition to alcohol, as a general infection or insufficient nutrition; pulmonary tuberculosis is found in a considerable proportion of the cases, at least of those which come to a fatal termination. It is noteworthy that Lindl[1] found some evidence of local or general neuritis in 214 of the 300 drunkards whom he examined. The slighter forms of the disease often escape recognition.

**Onset.**—In some cases premonitory symptoms occur which may be either the result of the alcoholic intoxication or a part of the disease. To the former group belong general constitutional disturbances, as morning vomiting and other symptoms of disorder of the digestive system, or dyspnœa and symptoms of affection of the circulatory system. In other cases loss of memory or slight mental impairment may be the first indication that anything serious is wrong. But the true premonitory symptoms, when there are any, are numbness and tingling of the fingers and toes, vasomotor disturbances in the extremities, painful cramps of the muscles, especially in the calves, and vague pains. These are undoubtedly part of the disease itself and constitute its first symptoms, but as they often exist for long periods before the onset of paralysis or the other classical signs, it is important to recognize them as premonitory of a more serious affection. In this early stage there is often some fever.

[1] *Der Alkoholismus*, 1903, Neue Folge, Heft i.

The onset of the neuritis is nearly always gradual, often slow and insidious; as the patient's memory is frequently impaired, the earliest symptoms may be overlooked or attributed to the effects of drinking, and it may be difficult to ascertain the duration or the mode of onset. An acute onset is rare, but cases have been recorded in which a sudden loss of power in the limbs apparently occurred. Eichhorst has reported the case of a barmaid who fell to the ground paralyzed on attempting to leave a railway carriage, and Judson Bury refers to a man who, while smoking, found he was suddenly unable to raise his hand to take his pipe from his mouth. But these acute attacks are generally more apparent than real; they are usually seen only after acute illnesses or a bout of drinking, during which the earlier symptoms were not observed. In the common forms the first definite symptom is usually disturbance of sensation with paræsthesia in the hands and feet, vague diffuse pains which are often very severe and always increased by movement, and tenderness of the muscles to pressure; these are soon followed by motor symptoms. The symptoms are almost invariably bilateral and symmetrical from the onset and more prominent in the lower than the upper limbs, but in the acute cases one limb or the functions of one nerve may be unduly affected at first. The further evolution is variable; in the severer forms the patient is generally bedridden in a few days, but, as a rule, the disease progresses more slowly and reaches its acme in three or four weeks. Occasionally its course is much more chronic; it may develop and progress slowly for months in mild cases when alcohol is not entirely withheld. The ataxic variety has often this slow and chronic course.

**Motor Symptoms.**—Paralysis may involve either the upper or lower limbs, more commonly both, but it is invariably more marked in the distal than in the proximal segments. The legs are almost always more affected than the arms, often they alone are involved. The trunk muscles escape frequently, and any considerable degree of palsy in the muscles supplied by the cranial nerves is rare. It must always be borne in mind that true muscular paralysis may be simulated by the reluctance of the patient to perform movements with the full amount of power which is possible to him, owing to the pain which forcible contraction of the tender muscles produces; a careful examination of the electrical reactions of all muscles may be necessary to ascertain the extent of the affection. In the usual type the first symptom is a slight difficulty in walking, especially on uneven surfaces or in ascending stairs, owing to the patient's inability to raise the toes from the ground. Dorsiflexion of the feet is very feeble or impossible, and the foot when unsupported hangs in the entensor position with its dorsum almost in the line of the axis of the leg. With this condition, to which the name ankle-drop was originally given by Ross, some malposition of the toes is generally associated; in the earlier stages they are generally hyperextended at their distal and flexed at the interphalangeal joints, except the great toes, which are often extended at all their joints; later all the toes are generally fully flexed and curled into the soles; this increases the concavity of the feet and adds to the difficulty in walking.

To avoid the constant danger of the toes catching in any obstacle, or even tripping him on level ground, owing to the ankle-drop, the patient raises his feet unusually high by flexion of the hips, and throws them forward with each advancing step; the gait has been consequently called *steppage gait*. It is almost characteristic of peripheral neuritis. It occasionally happens

that all the muscles of the anterior tibial groups are not equally affected—the tibialis anticus often escapes when the peronei and extensors of the toes are paralyzed.

The symptoms of palsy are, however, very rarely limited to the muscles on the front and outer aspects of the legs; those which are usually next affected in both time and degree are the calf muscles. Walking is then more seriously interfered with, as the patient cannot spring from his toes or even fix the ankle-joints properly. The muscles of the thighs and hips suffer later and, according to the general rule, being proximal muscles, to a considerably less degree; indeed, the muscles which move the hip-joints are much affected only in the severest forms, and by that time the patient is invariably bedridden. The paralyzed muscles are soft and flabby to touch, and evidently hypotonic; when the paralysis has lasted more than two or three weeks they begin to waste, and the atrophy corresponds approximately to the degree of the paralysis. In the later stages, however, when the atrophied fibers become replaced by connective tissue, the muscles may be firm and fibrous to touch.

In the upper limbs the paralysis, as a rule, first expresses itself by affection of the extensors of the wrist and fingers. In the arms the muscles which derive their motor fibers from the musculospiral nerve are the most vulnerable. Rarely, however, does the extent of the paralysis coincide with the nerve distribution; in the arm the supinator longus and the extensor ossis metacarpi pollicis often escape, or are affected in much less degree than the long extensors. This type of paralysis and the wrist-drop that results, although most characteristic of lead palsy, is not infrequently seen in alcoholic neuritis. The loss of power in the extensors of the wrist and fingers leaves the arms almost useless; the patient cannot grasp any object firmly owing to the mechanical disadvantage at which the flexors of the fingers must work when the extensors of the wrist are paralyzed. But the flexors of the wrist and fingers rarely escape, and in the severer cases may be as powerless as the extensors. Occasionally the intrinsic muscles of the hand are affected early; according to Gowers those of the thenar and hypothenar groups always suffer later than the interossei. Of the muscles above the elbow, the triceps generally suffers more severely than the biceps, while those which move the shoulder-joints are rarely much affected. In the atrophic stage the wasting is generally most prominent on the dorsal surfaces and ulnar borders of the forearms; in the hands it may be indicated by flattening of the thenar and hypothenar eminences and by hollowing of the interosseal spaces.

The muscles of the trunk are involved in severe cases, most commonly those of the abdominal walls; this impairs the power of expelling the contents of the bladder and rectum, and coughing and sneezing. If the flexors of the thighs are weak as well as the recti abdominis, the patient becomes unable to raise himself from the recumbent position. There is considerable palsy of the muscles of the back and neck only in the severest cases. Paralysis of the diaphragm is occasionally observed; respiration is consequently severely implicated, as the intercostal muscles rarely escape completely, and the case frequently comes to a fatal termination.

The functions of the cranial nerves are sometimes involved; lingual, palatal, or pharyngeal palsies may develop in very severe cases, generally when they are progressing to a fatal termination; laryngeal paralysis is

very rare. The tachycardia, which is often a prominent symptom, is generally attributed to disease of the pneumogastric nerves; Dejerine has demonstrated parenchymatous degeneration of them.

Bilateral facial palsy, which is not infrequently seen in infective multiple neuritis, is rarer in the alcoholic form. So also are ocular palsies, though many cases in which they have occurred have been recorded; they are probably due to hemorrhagic polioencephalitis superior (Wernicke's form), which develops most often in the subjects of chronic alcoholism, and not to a neuritic affection of the ocular nerves. Nystagmus, however, is not infrequently observed. The pupillary reactions are very rarely altered, but the Argyll-Robertson phenomenon has been observed in alcoholic neuritis. Optic neuritis has also been described.

The state of the electrical reactions of the nerves and muscles is very variable. The excitability of the nerves which are affected diminishes rapidly and generally disappears completely; it is interesting that little or no reaction may be obtained from nerves the functions of which are almost intact; this is evidently the result of incomplete lesions, as periaxial neuritis limited to the myelin sheaths, which interfere more seriously with the receptivity of the fibers than with their conductibility. No contraction can be obtained, as a rule, by the direct application of the faradic current to muscles which are much paralyzed; in the less affected the reaction may be incomplete, as a small proportion of the fibers may respond. With the galvanic current all forms of the reaction of degeneration may be obtained, but the current necessary to produce any contraction is generally greater than normal. In severe cases the muscles often eventually lose their galvanic excitability.

Although paralysis is invariably the most prominent motor symptom, there is occasionally evidence of motor irritation or hyperactivity. The tonic spasms or cramps of calves and other muscles which occur as frequent premonitory symptoms occasionally persist in slighter cases throughout. Fibrillation, too, is occasionally seen, and irregular tremor of the hands, tongue, and facial muscles is a well-recognized symptom. These phenomena are probably due to irritation of undegenerated fibers. Still more important is the reflex or voluntary contraction of relatively intact groups of muscles, by which the limbs are held in the positions which are most comfortable, and by which the patient resists passive movements which give him pain. Thus the knees are almost always semiflexed by contraction of the hamstring muscles, the tendons of which may be tense. The importance of this is the danger that the shortened muscles may undergo in this position permanent organic contractures. Organic contractures may also develop in muscles which are allowed to shorten by the relatively greater palsy of their antagonists.

**Sensory Symptoms.**—The tingling, numbness, and other subjective sensations in the distal segments of the limbs generally persist throughout the course of the disease, but the pains which are an almost constant symptom are a more serious cause of suffering. They may be aching, burning, or shooting in character, and are often very severe. They are generally more diffuse and less intermittent and lancinating than those of tabes dorsalis, and, as a rule, are referred to the deeper tissues of the limbs or joints, rarely to the course of the nerves or to the skin. They may occur spontaneously, but are excited or increased by movement, either active or passive, of the limbs; severe paroxysms often occur during the night and deprive the patient

of his sleep. Not infrequently there is constant and very troublesome neuralgic pain in the soles of the feet.

But the most prominent and constant sensory symptom of multiple neuritis is the extreme tenderness of the muscles to pressure; this is probably never absent and is almost a pathognomonic sign. It is usually greatest in the calves and in the regions in which there is considerable paralysis, but muscles which are in no degree paralyzed may be very tender. In the severer cases the limbs cannot be touched without eliciting cries of pain, and even the weight of the bed-clothes may be intolerable. It becomes agonizing for the patient to lie long in any position, and yet he dreads being moved, owing to the pain which any change in posture produces. The affected nerve trunks are also often very sensitive to pressure, but this is neither so constant nor so prominent as the muscular tenderness. Occasionally the superficial nerves may be felt slightly enlarged or swollen.

Objective disturbances of sensation are nearly always much less marked than the motor palsies—in local lesion of the peripheral nerves the sensory functions are also less affected than the motor, but they are rarely quite absent. They are most marked in the distal segments of the limbs and diminish gradually proximalward, ceasing in the legs, as a rule, about the middle of the thighs and rarely marked in the arms above the elbows; but there is never a definite boundary line between the normal and anæsthetic skin, and it is characteristic of the hypæsthesia that it does not correspond in extent to the distribution of either the peripheral nerves or the dorsal roots. Usually the most definite loss is to light touches, which may not be perceived in the feet or legs and felt only unnaturally on the thighs. Sensibility to pain is less commonly lost, but there is not infrequently some delay in perception of painful stimuli. The temperature sense also may be diminished or absent in the extremities, but this is far from constant even in the severer cases. But more characteristic of neuritis is a curious combination of hypæsthesia and hyperalgesia; in areas of skin which are more or less insensitive to light touches a pin-prick or a scrape may cause intense pain, and even washing or rubbing the skin may give rise to much discomfort. Occasionally the sense of position is more affected than cutaneous sensibility.

Another symptom which results from loss of sensation, but expresses itself by disturbance of motion, is ataxia or incoördination. It is not a frequent symptom, although occasionally it may be the most prominent feature. Incoördination becomes a prominent symptom when the afferent nerves from the muscles and joint are unduly affected. Dejerine has given this condition the name *neuro-tabes peripherica*, owing to the similarity of its symptoms to those of tabes dorsalis; but in his two original cases, as well as in others, the dorsal spinal roots and the dorsal columns of the cord were intact, and the disease was limited to the peripheral nerves. The ataxia is generally apparent only in the movements of the lower limbs, and especially in walking. The gait may closely resemble that of tabes dorsalis, and the similarity is increased by the fact that the patient is more unsteady in the dark or when his eyes are closed. Ataxia of this type must be distinguished from the disorders of movement which may result from paresis alone; it may be observed in cases in which the muscular power is intact or relatively little affected. In some cases the ataxia precedes the development of paralysis; in others it may remain the chief symptom. In the latter class it often sets in rapidly, and is usually accompanied by at least some diminution of

cutaneous sensibility and frequently by severe pains and muscle tenderness. The sense of position and of movement is very diminished in the majority of these cases.

**Reflexes.**—Loss of the tendon reflexes—including the knee- and ankle-jerks in the lower limbs, the biceps and triceps jerks in the arms—is one of the cardinal signs, and the most delicate test we possess of disease of the peripheral nerves. The knee-jerk may be absent not only when there is definite palsy or loss of sensibility, but also in cases of chronic alcoholism where there is yet no suspicion of neuritis. Their absence is, however, not an absolute rule; they persist in rare cases, despite the presence of considerable paralysis, and they have been found definitely exaggerated in the early stages. This is difficult to explain; one theory assumes that it is due to the co-existence of a spinal lesion, probably also the result of the intoxication that produces the neuritis; another attributes it to a state of excessive irritability of the sensory fibers which leads to such an exaggeration of the afferent impulses to make it possible for them to overcome the obstacle offered by a slight degeneration of the motor fibers of the reflex arc. Absence of the tendon-jerks is often the most persistent sign of neuritis in cases which improve; they may not reappear for years after complete disappearance of the other symptoms. The cutaneous reflexes are generally diminished or abolished, but they occasionally persist, even when there is considerable motor palsy, especially in cases with marked hyperalgesia. The plantar reflexes, when they do not disappear, are invariably of the flexor type.

**Sphincters.**—The functions of the bladder and rectum are, as a rule, unaffected, although in cases in which there is much mental disturbance the patient may pass urine involuntarily or have symptoms of retention. Occasionally, however, dysuria and retention have been observed; such sphincter affection has been generally attributed to the co-existence of spinal disease, but this has not been demonstrated, and it is certainly possible that the neuritic affection may extend to the nerves of the bladder.

**Trophic and Vasomotor Changes.**—Apart from the atrophy of the paralyzed muscles trophic changes are not a prominent feature. In chronic cases the skin, especially of the hands and fingers, loses its natural wrinkles and becomes thin and shining, but true "glossy skin," which is frequently seen after severe local nerve lesions, is rarely met with in multiple neuritis. That of the legs is generally dry, rough, and covered with scales of desquamating epidermis. The hair and nails become dry and brittle, and the latter may be furrowed longitudinally and may grow irregularly. Bedsores are rare, and perforating ulcers and severer trophic changes are almost never seen.

Of the vasomotor changes, œdema is the most prominent; it occurs in a considerable proportion of the severer cases. It is generally localized and is most common on the dorsa of the hands and feet, and about the ankles. In the majority of the cases it certainly develops independently of vascular and renal disease, and is probably due to an affection of the vasomotor fibers. Sometimes it does not become apparent until the patient begins to walk or to sit up with his feet dependent; in other cases it may be an early or the initial sign, as it so frequently is in beriberi. Local anæmia is occasionally observed, and Ross has reported a case of alcoholic neuritis with the symptoms of Raynaud's disease. Profuse sweating is common in the early and acute stages, especially in the extremities of the limbs.

Several cases have been recorded in which neuritis set in with or im-

mediately after an attack of polyarthritis; most probably the two conditions were here due to the same cause, but in other cases one or more joints have been observed to swell and become painful during the course of neuritis; the changes may ultimately produce ankylosis. More chronic arthritic changes, especially in the joints of the fingers and wrist, which set in with pain and peri-articular thickening and ultimately produce limitation of movement, are not uncommon.

**Mental Symptoms.**—Psychical disorders are so common in multiple neuritis, especially in the alcoholic form, and so frequently of the same type, that they may be regarded as part of the disease; in fact some authorities speak of these mental symptoms as the polyneuritic psychosis. It was Korsakow who first carefully analyzed and described them in their most common form (see Vol. I, p. 196).

**Course.**—This is very variable; it depends largely on the cause, more on the stage in which the patient comes under treatment and when the action of the poison is arrested. As a rule, the more severe and acute the symptoms the longer will be their duration; they generally increase for a few weeks after the alcohol has been stopped, and then remain stationary until improvement sets in. But this can be rarely expected within one or two months. The first sign of improvement is usually diminution of the pains and cutaneous hyperalgesia, but tenderness of the muscles often persists until recovery is almost complete. The disturbances of cutaneous sensibility generally disappear before there is much return of power. This occurs first in the muscles which were last and least affected, that is, in those which move the proximal joints of the limbs, and progresses distalward, but only very slowly; in even the most favorable cases it requires months for muscles that were definitely paralyzed to regain their functions. On the other hand, complete return of power in all muscles may be expected eventually in almost all cases which improve, provided that the patient does not revert to the abuse of alcohol. As the paresis disappears the wasted muscles slowly regain their bulk, but may not attain their normal size for months. The most serious obstacle to complete recovery is the shortening and contracture of the antagonists of the most affected muscles. It is very frequently seen in the calf muscles; the ankle is then more or less fixed in the extensor position and the foot cannot be dorsiflexed, so that the patient is unable to get his heel to the ground on attempting to walk. The absence of the tendon-jerks, and alterations in the electrical reactions of the nerves and muscles are usually the last persisting evidence of the disease in these favorable cases.

The course of alcoholic neuritis is not always so favorable. In cases with acuter onset, in which the limbs become powerless within one or two weeks, the paralysis may spread to the muscles of respiration and produce a fatal result; and not infrequently death occurs from cardiac failure in the acute stage of the disease, owing either to paralysis of the vagi nerves, or to direct affection of the heart muscles. Cases have been recorded which have come to a fatal termination within a week, but probably most of these were cases of Landry's paralysis.

The chief complications are the effects of alcohol on other organs, as the heart, liver, and gastro-intestinal tract. Pneumonia and bronchitis, which occur not infrequently, are serious complications. The pneumonia may run a low and almost latent course, and its existence may not be suspected until it is detected by careful physical examination. Pulmonary tuberculosis

is perhaps the most frequent of the serious complications and probably the most common cause of death. Its relation to the neuritis may be twofold; on the one hand, the tuberculous toxin may be an etiological factor, especially when combined with other causes, as with alcohol; on the other hand, alcoholic subjects are very liable to tuberculosis, or latent foci may become active when the patient is confined to bed with neuritis.

**Diagnosis.**—When the essential features are recognized the diagnosis can present no difficulty in the majority of cases. The wide distribution of the affection of both motor and sensory functions, its rigid symmetry, and above all its greatest intensity in the distal segments of the limbs, characterize the disease in the typical cases. It is, however, very important to remember how variable the symptoms may be in different forms, and even in cases due to the same cause.

The condition with which it may be most easily confused is perhaps *acute poliomyelitis;* it may be remembered that it is little over a quarter of a century since a definite distinction was drawn between them. The difficulty is chiefly present in cases which occur in early life, as then multiple neuritis is relatively rare, while infantile palsy is common. The onset of poliomyelitis is, however, more acute, and is generally accompanied by severer constitutional disturbances; the paralysis is rarely symmetrical, and is often more marked in the proximal than in the distal segments of the limbs; further, complete paralysis of the abdominal and trunk muscles is not uncommon, while in multiple neuritis it is exceptional. Sensory disturbances and the severe and persistent neuritic pains are absent, and although the paralyzed limbs may be at first painful to pressure or movement this soon disappears and the nerve trunks are not unduly tender. Neuritis is generally progressive for a few weeks, while poliomyelitis usually comes to a standstill in a few days and the muscles least affected recover quickly; then the irregular and random distribution of the paralysis is evident.

The differential diagnosis between multiple neuritis and *Landry's paralysis* is more difficult. The course of the latter is more acute, and, as a rule, death results within ten days or a fortnight; the constitutional symptoms are generally slight and the sensory phenomena are limited to pain in the back and occasionally slight numbness in the extremities; definite anæsthesia does not occur, and neither the nerves nor muscles are tender to pressure. But its most characteristic feature as contrasted with polyneuritis is that the muscles of the trunk and of the limbs are equally affected, and that the paralysis ascends the trunk from the legs to the arms, and does not appear in the hands and feet simultaneously or successively and involves the musculature of the trunk last, as does multiple neuritis. Finally, the paretic muscles rarely waste and the cranial nerves are usually affected only when the limbs and trunk are more or less completely paralyzed.

The ordinary form of multiple neuritis, in which there is widespread palsy, can be scarcely mistaken for *tabes dorsalis*, but in the cases in which sensory loss and ataxia are the most prominent features diagnosis may be extremely difficult. Neuritis is, however, distinguished by its more rapid evolution, and by the absence of lightning pains, of sphincter disturbances, optic atrophy and alterations in the pupillary reactions. On the other hand, in tabes there is, as a rule, no true paralysis, and when muscular atrophy does occur it is usually limited to one group of muscles, and is rarely symmetrical from the beginning. But the most valuable diagnostic sign is that while in neuritis

the nerves and muscles are invariably, in some degree at least, tender to pressure, in tabes the pain reaction on squeezing the nerves and on deep pressure on the muscles is almost always diminished and frequently quite absent. The history of syphilitic infection and the presence of an excess of lymphocytes in the cerebrospinal fluid are in favor of tabes.

The symptoms of *trichiniasis* may resemble those of polyneuritis, but the acute onset with fever and gastro-intestinal symptoms, the œdema about the eyelids, and the swelling and tension of the affected muscles, as well as the character of the pain in them, are fairly characteristic. The great increase of eosinophiles in the blood and the early affection of the functions of the cranial nerves are also distinctive features.

With *general œdema* there is often a certain degree of flaccid paresis of the limbs, especially of the legs, and loss of the tendon-jerks. In some of these cases there may be a slight peripheral neuritis due to a general toxic state, but the rapidity with which the paresis disappears, and the early return of the tendon jerks as the œdema subsides, makes it more probable that the paresis is a direct mechanical or nutritional effect of the effusion into the muscles.

Polyneuritis may be diagnosed from *acute myositis* by the swelling of the extremities in the latter, owing to inflammatory œdema of the subcutaneous tissue and muscles, the rigidity of the muscles, the absence of sensory disturbances, and the erythematous rash which is nearly always present over the affected parts. Finally, in myositis the knee-jerks often persist. A form of paraplegia in old people due to contractures following chronic myositis, which has been described by Lhermitte[1] and others, may be confused with polyneuritis. But it rarely occurs before the age of seventy-five, and generally only in feeble persons confined to bed. Like neuritis, it may set in with pains, cramps, and weakness of the legs, but it can be easily distinguished by the persistence of the knee-jerks, the normal electrical reactions of the muscles, and the firm contracture of all the muscles which are affected.

**Prognosis.**—The outlook naturally depends on the acuteness and severity of the attack. In the early stages, when the paralysis is spreading rapidly, prognosis must be guarded, as there is always the danger that the affection may involve the cardiac or respiratory apparatus and lead to sudden death. But it is often surprising how patients in whom the diaphragm is paralyzed, or the heart dilated, irregular, and rapid, recover. It must be remembered that the disease almost invariably progresses for a time after the poison has been withdrawn. On the other hand, when its course once becomes stationary there is very little danger of relapse, even though improvement is very slow. The chief danger to life results from the complications, especially from tuberculosis; if these can be excluded, and the cardiac and respiratory functions are not disturbed, the outlook as regards life is very favorable and the chances of ultimate complete recovery are excellent. The restoration of function is always slow and even in cases of moderate severity four or six months must elapse before the more severely affected muscles have fully regained their power. The condition of the electrical reactions is a valuable guide in estimating the duration of the muscular paresis.

**Treatment.**—The first essential in every case of multiple neuritis is to discover and remove the cause, or stop its action as soon as possible. In

[1] *Nouv. Iconographie de la Salpêtrière*, 1906, p. 256, Thèse de Paris, 1907.

dealing with alcoholic neuritis this is often not easy, especially with female patients who take every possible means to deceive the medical attendant and obtain further supplies of alcohol. Frequently it is necessary, and when possible it is always advisable, to place the patient in a home or hospital under the care of trained and reliable nurses; if he remains in his own home he will probably succeed, even if bedridden, if it is in any way possible, in procuring stimulants. Many authorities recommend that alcohol should not be suddenly stopped, owing to the danger of collapse; but this is rarely to be feared except when the heart is very feeble, and it is certainly inadvisable to continue the administration of a drug which is producing such serious symptoms.

Rest in bed is always advisable, except perhaps in the mildest cases; the patient is thus most easily protected from exposure to cold, which may not only have an unfavorable influence on the neuritis, but also make him more liable to pulmonary complications. Complete rest is also a matter of great importance, as movement often seems to intensify the changes in the degenerating nerves, and it certainly increases the pain. In severe cases the use of a water or air bed is generally advisable, as on it the necessary pressure is more uniformly distributed, and the patient does not suffer so much as when his limbs lie on a firmer and ill-adapted surface. Much relief can be given by removing the weight of the bed-clothes by bed-cradles.

There is no specific treatment by which the progress of the disease can be arrested or recovery attained; it can be, therefore, in the acute stages at least, only symptomatic, and the most urgent and prominent symptom is usually pain. Often it is necessary to have recourse to the use of drugs; antipyrine and phenacetine are the most effective, and there is rarely any contra-indication to their use; when the patient is restless and sleepless, bromides may be combined with one or other of them. Occasionally the pains are so severe and constant that morphine is necessary, but it is evidently advisable to avoid its use in a person already addicted to a drug habit. Local applications over the chief seats of pain often give considerable relief, especially warm fomentations. Mills recommended the alternate application of very warm and cold water with a large sponge over the limbs, and Pospischill cold packs. It often suffices to keep the limbs wrapped up in a thick layer of cotton wool. Oppenheim has obtained excellent results by diaphoretic treatment; warm baths may give relief if the patient is strong enough to be moved from bed, but this should not be attempted if the heart is feeble. Local hot air baths, which can be used in bed, are sometimes very effective, and if they are not available warm packs may be employed.

Another important aim should be to prevent the development of contractures. The greatest danger is that the feet become fixed in the ankle-drop position, or the hips and knees in semiflexion. From the first, steps should be taken to keep the feet at about right angles to the legs, by the use of a large sand-bag at the foot of the bed, against which the soles of the feet may press, or by a broad, padded board placed vertically across the bed. If the calf muscles are already shortening, their contraction may be overcome by the constant tension of rubber cords stretching from the toes of shoes which the patient wears to a band around the limb at about the level of the knee. In the acute stages the knees are nearly always held semiflexed, but this should never be permitted, as the danger of shortening of

the hamstrings is very great. Full extension of the limbs may give considerable discomfort at first, but this quickly passes off if persisted in.

Of the many drugs, salicylate of soda and iodide of potassium are probably the most frequently used, but there is no evidence that they have a specific effect on the disease. Attention is better directed to general tonic treatment; strychnine in fairly large doses is certainly of considerable value when there is any tendency to respiratory embarrassment or cardiac failure. Arsenic should be rarely given, and only in small doses and under constant supervision, as it itself may be a cause of neuritis. Diet needs some attention; it should be as light and nourishing as possible.

During the stationary stage and when improvement is setting in galvanism may be employed, but it should not be adopted in the early stages when tenderness of the muscles is still very great. Large electrodes should be used, and the current should be the weakest that can produce a definite contraction of the muscle fibers and yet is not strong enough to give pain. Massage also may be of considerable service, but it should not be resorted to in the acute stages or while the limbs are still very tender. At first it should be as gentle as possible.

The patient should be encouraged to move the affected limbs as much as possible, when movement does not give him any acute pain, as voluntary movement is undoubtedly a more effective stimulus to nutrition than either galvanism or massage, and voluntary effort probably exerts a favorable influence on regeneration. If contractures have been allowed to develop, tenotomy may be necessary, but they can be generally overcome by systematic passive movements and extension.

**Acute Infective Polyneuritis.**—The group of cases included under this title has been variously described as rheumatic, infective, or spontaneous neuritis, as it is rarely possible to ascribe the attack to any definite cause; or as febrile polyneuritis, since the disease usually sets in with febrile symptoms. Many cases follow or are attributed to cold, exposure, or overexertion, but it is difficult to see how these factors can directly produce such widespread and progressive symptoms, or the extensive parenchymatous neuritis to which they are due. It is very probable that the polyneuritis is directly due to some bacterial infection, which may not be always of the same nature. Its onset is frequently preceded or accompanied by general malaise, pains in the back or limbs, a rise of temperature to perhaps 104° F., gastro-intestinal symptoms, and loss of appetite. In the severer cases the attack may be ushered in with a rigor; swelling of the spleen, profuse sweating and albuminuria, as well as vomiting and diarrhœa, have been observed. These acute symptoms have been frequently regarded as influenzal. Its infective nature is further supported by the occasional occurrence of small epidemics, of which no definite cause can be discovered.

Generally, as the febrile symptoms are subsiding the patient begins to complain of numbness, tingling, or other subjective sensations in the fingers and toes, and occasionally of sharp or burning pains, but these are rarely so severe as in alcoholic neuritis. Within a short time some loss of muscular power appears, generally in the legs before the arms, but it is more or less symmetrical and greater in the distal than in the proximal segment of the limbs. In rare cases the arms are affected before and more severely than the legs.

The disease is a typical parenchymatous neuritis. Its clinical features are practically identical with those which have been described in detail under

alcoholic neuritis, for no set of symptoms is exclusively related to a single cause; but there are a few characteristic points. The paralysis is, as a rule, more generalized than in alcoholic neuritis of corresponding severity, the trunk muscles are usually relatively more affected, and even complete bilateral flaccid facial palsy is by no means uncommon. Cases may be seen in which, though the limbs are not very severely affected, the patient is unable to close his eyes, his articulation is indistinct owing to loss of the labial letters, and as he attempts to drink, fluids dribble from the corners of his mouth owing to the palsy of the lips. Not infrequently, too, the palatal and pharyngeal muscles are weakened in some degree, so that swallowing becomes difficult and food may regurgitate through the nose. Laryngeal paresis, producing aphonia, is rarer.

There is also, as a rule, less sensory disturbance than in the alcoholic cases; spontaneous pains may be slight and even occasionally absent, and although the nerves and muscles are always tender to firm pressure, they are rarely as extremely so as in alcoholic paralysis. The amount of loss of cutaneous sensibility varies considerably.

This form of primary infective neuritis generally runs a fairly rapid course; the disease often reaches its acme in a week or ten days, and then, after a stationary period in favorable cases, the patient begins to improve. Fatal terminations, which are not infrequent, generally result from paralysis of the respiratory muscles or from heart failure. Acute cases which die within ten days may so closely resemble Landry's paralysis that distinction is difficult if there is but little sensory change or if the evolution of the disease has not been observed. There is, however, generally more constitutional disturbance than with the onset of Landry's disease, and the early affection of the cranial nerves in the neuritis is an important point in the diagnosis.

**Recurring Polyneuritis.**—Under this term Mary Sherwood[1] described two cases in 1891, although one of them, in which only the nerves of one arm were affected, certainly does not belong to this class.

These cases are characterized by a marked tendency to repeated attacks of multiple neuritis for which no definite cause can be ascertained; cases in which the successive attacks are due to renewed exposure to some poison, as alcohol or lead, are naturally excluded from this group. H. M. Thomas,[2] who reviewed the subject in 1898, was then able to collect only seven cases, and very few have been published since that date. The one observed by Thomas is most typical: A man, aged twenty-eight years, otherwise healthy, who did not indulge in alcohol to excess and was apparently not exposed to any poison, developed each June for five years an attack of typical multiple neuritis, from which he slowly recovered in four to six months. Bernhardt points out that the disease is often atypical, and that one or more of the cranial nerves have been frequently involved. It is difficult to say whether the repeated attacks are the result of an unusual predisposition, or if the first attack leaves the nerves more liable to a second. Dejerine-Klumpke and Bernhardt have observed recurrent lead palsy apart from renewed exposure to the poison. One of the cases reported by Barnes[3] as "Tonic Degeneration of the Lower Neurones," and that recorded by Williamson[4] under the same title, probably belong to this class.

[1] *Virchow's Archiv*, 1891, cxxiii, 166.
[2] *Phila. Med. Jour.*, 1898, i, 885.
[3] *Brain*, 1902, xxv, 479.
[4] *Brain*, 1903, xxvi, 206.

# CHAPTER XIII.

## DISEASES OF THE CEREBRAL NERVES.

By E. W. TAYLOR, M.D.

**General Introduction.**—The title, "Diseases of the Cerebral Nerves," is evidently somewhat misleading, first, because of the relatively brief statement necessitated by space, and secondly, because it is not possible to consider affections of the cranial nerves with any degree of completeness, irrespective of the general diseases of which they so often form a part. The attempt has, therefore, been made in this section to consider only those disturbances which are characteristic of the individual nerves, and not to infringe on the domain of the ophthalmologist, otologist, laryngologist, or the broad field of the general physician or surgeon. Such inconsistencies as may occur are necessitated by the extreme difficulty of classification. It is evident that many general diseases are in part manifested by disturbance on the part of various cranial nerves. An attempt has been made in the following pages to allude to these frequent associations without entering into undue detail. In any consideration of the cranial nerves a knowledge of anatomical relations is essential to a proper understanding of the signs and symptoms produced by their disease. As much anatomy, therefore, has been introduced as is consistent with the purposes of this work.

### OLFACTORY (FIRST) NERVE AND TRACT.

**Anatomical Relations.**—The olfactory apparatus is made up of the olfactory tract, the olfactory bulb, and the nerve proper. From the embryological standpoint the tract and bulb may properly be considered as a hollow outgrowth, which later in the course of development is obliterated, of the lateral ventricle of the brain. The olfactory tracts, dividing into roots, end in the olfactory area of the brain, localized, so far as known, in the hippocampal gyrus and adjoining regions. Further relations of the tracts to the brain remain somewhat vague. In the lower animals, in which the olfactory apparatus represents an important, constituent part of the brain, the hippocampal region is developed to a disproportionate degree, as well as the olfactory tracts. The olfactory bulb lies in the olfactory fissure beneath the frontal lobe. The lower surface of this bulb is to be regarded as a secondary sensory ganglion for the reception of fibers of the olfactory nerve proper, constituted by twenty or more fine bundles originating in the nasal Schneiderian membrane. These bundles pass through foramina in the cribriform plate of the ethmoid bone from the upper portion of the septum and the lateral walls of the nose as far down as the lower border of the superior turbinated bone. The cells of this small olfactory area, therefore, give rise to the olfactory nerves. Other portions of the

mucous membrane of the nasal cavity are supplied with common sensibility by branches of the fifth nerve.

**Etiology.**—Disturbances of smell may arise from lesions in any part of the course of the olfactory nerves or tracts from the peripheral distribution in the nose to the ultimate terminations in the brain. If, for example, the olfactory mucous membrane of the nose is affected through local irritations, swelling or any obstruction of the passage leading to this area, the sense of smell of necessity suffers, even though the nerve itself be not affected. Local disease may, however, destroy the nerve endings in the olfactory area, naturally leading to a greater or less degree of anosmia. Actual injury of the olfactory nerves at the base of the brain, where for a short distance they lie in close relation to the bone, may produce a like disturbance. Such a lesion, though probably often overlooked, undoubtedly occurs as a result of contusion of the brain, or in extensive fractures of the base involving the frontal region. Loss of the sense of smell has been observed after an attack of influenza and in senile conditions. A congenital anosmia and a rare hereditary form have also been described. Various perversions of smell are not uncommon. A condition of increased sensitiveness, hyperosmia, has been described in hysteria, usually associated with altered appreciation of odors. This condition is, no doubt, analogous to the extreme acuity of vision occasionally met with in that disease. Subjective sensations of smell, parosmia, are, on the whole, rare, but undoubtedly occur in disease of the brain, or from irritation of the nerve in its extracerebral course. In a case of brain tumor, recently under observation, in which autopsy showed an involvement of the greater part of one temporal lobe, the patient repeatedly complained of extremely unpleasant sensations of smell coming on in the form of attacks, and never twice the same. It is altogether probable in this instance that these perverted sensations were aroused by the lesion which involved the central olfactory area. Olfactory hallucinations occur in certain forms of insanity, and may possibly be regarded as having some localizing significance. As an epileptic aura, also, such perversions of smell may be significant. Gowers speaks of a case, reported by Urben, in which parosmia occurred in the course of tabes dorsalis.

**Pathology.**—The pathological anatomy of affections of the olfactory nerves is closely associated, on the one hand, with local disturbances in the nasal cavity, bearing merely a secondary relation to the nervous system, and, on the other hand, to traumatism of the cranial walls or the brain. Infections, notably meningitis, may include the olfactory apparatus in their spread, but usually in such cases other symptoms so far predominate in the clinical picture that special disturbances of smell are overlooked. It should, however, be remembered that a possible source of infection of the brain meninges may be through the cribriform plate of the ethmoid bone, particularly if it has sustained a fracture. Primary disease of the olfactory apparatus, apart from the conditions already mentioned, is rare.

**Symptoms.**—The sense of smell, broadly considered, includes the recognition of odors through the anterior nares, stimulating the olfactory area, and the recognition of flavors through the posterior nares, also through the mediation of the olfactory nerve. Whether or not the fifth nerve contains elements which subserve the special sense of smell is open to doubt. Inasmuch as only a small part of the mucous membrane of the nose, namely,

the so-called olfactory area, is supplied by the olfactory nerve, and the remainder, which is by much the larger part, by the fifth nerve, it is necessary to separate substances used for testing the sense of smell into those which stimulate the olfactory nerve and those which affect only the fifth nerve. If sensations of smell are blended with those of taste proper, such as bitter, sweet, salt, sour, a mixed sensation, termed a flavor, is produced. If the sense of smell be lost, the perception of flavors is also lost, but in spite of this fact, volatile substances, such as ammonia, acetic acid, or mustard, may so stimulate the nasal mucous membrane supplied by the fifth nerve that a distinct perception is aroused. Lacrymation may thereby be produced, due wholly to the irritation of the nasal mucous membrane through the mediation of the fifth nerve. This, however, is not a true sense of smell. Bearing these facts in mind, tests for smell must be made with non-irritating substances, such as peppermint, oil of cloves, or asafetida, rather than with substances which irritate the mucous membrane.

**Diagnosis.**—After determining the fact of loss of smell by proper tests, it is essential to exclude disease of the mucous membrane, a purulent discharge, or other local cause for the defect. The retention of the ability to recognize flavors, with a loss of the sense of smell, is very frequently due to such lesions, owing no doubt to the fact that the expiratory stream is stronger and impinges more directly upon the olfactory area than the inspiratory stream. The importance of a local examination cannot be overestimated. In the examination, apart from the tests to which allusion has already been made, electricity has been locally applied. This procedure gives rise to a phosphorus-like odor, but the test is painful and at best of doubtful value. In making a routine examination account must be taken of the intelligence of the patient, and one nostril should always be closed when the other side of the nose is being examined. As a further complication it should also not be forgotten that disease of the fifth nerve may at times cause considerable anosmia, probably indirectly, through the general nutrition of the mucous membrane, including the olfactory area.

**Prognosis.**—The outcome of affections of smell naturally depends essentially upon the character, persistence, and possibility of removal of the cause. Temporary loss of the sense of smell, due to local irritation of the nasal mucous membrane, as, for example, in ordinary rhinitis, is a matter of common experience. The function of smell is restored with the removal of the irritation. In man the sense of smell may be regarded as a luxury rather than a necessity. Hence its permanent loss, especially if unassociated with the loss of appreciation of flavors, is often not deeply felt, and in some cases no doubt not appreciated by the patient. Apart from injuries and infections of the nervous system the prognosis and treatment depend essentially on the local conditions within the nasal cavity.

## OPTIC (SECOND) NERVE AND TRACT.

**Anatomical Relations.**—The optic nerve, like the olfactory, is to be regarded rather as an outgrowth of the brain than as a nerve properly so-called. The optic vesicle appears in embryonic life as a peripheral expansion or hollow outgrowth of the forebrain. The lumen of this vesicle is later obliterated and the stalk becomes the optic nerve, the peripheral

expansion differentiating into the complex retina and its associated parts. As in the brain and cord, the axones running in the optic nerve are myelinated but devoid of a connective-tissue sheath. As in other sensory nerves, the primary neurones originate, for the most part, in ganglion cells of the retina. Passing backward toward the brain a large number of fibers, presumably twice as many as uncrossed, cross at the chiasm. This crossing is partial in man, monkeys, and many mammals, and complete in fishes,

FIG. 6

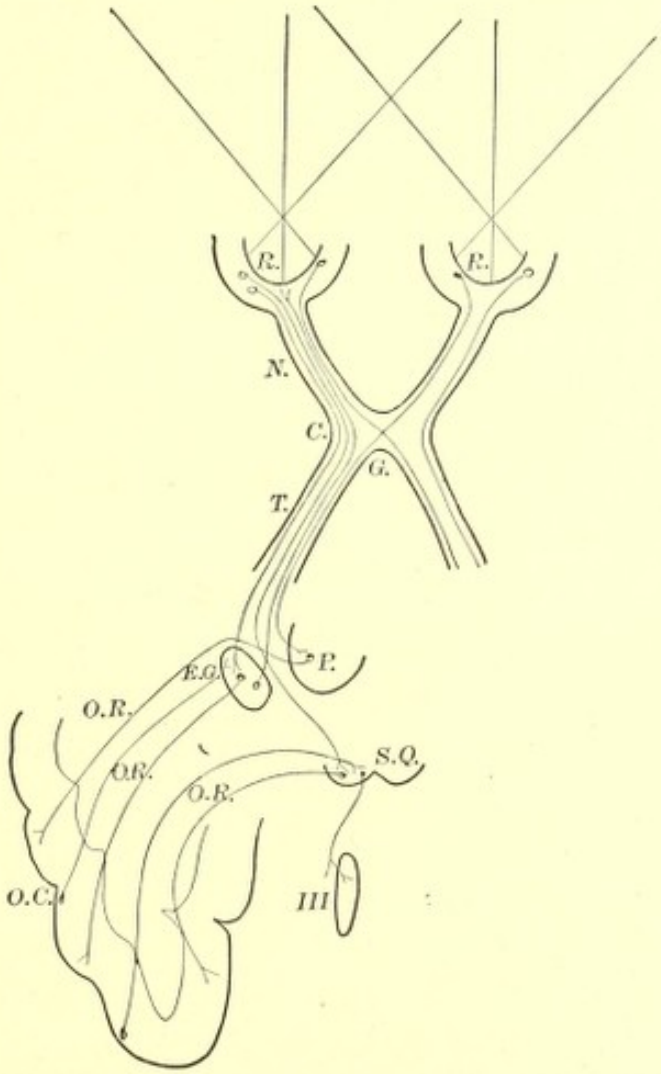



Diagram of the optic system. *R.*, retina; *N.*, optic nerve; *C.*, optic chiasm; *G.*, Gudden's commissure; *T.*, optic tract; *P.*, pulvinar of thalamus; *E.G.*, external geniculate body; *S.Q.*, superior quadrigeminum, the three ganglia constituting the primary visual centres; *O.R.*, optic radiations; *O.C*, occipital cortex; *III.*, nucleus of third nerve. Note crossing of internal fibers at the chiasm; also that most of the fibers originate in the retina, a lesser number in the cortex.

amphibians, reptiles, and birds. A possible explanation for this irregularity of crossing is to be found in the fact that those animals whose vision is wholly monocular have a complete crossing, whereas those in whom binocular vision has been established have developed a partial crossing. Behind the chiasm, the optic tracts, made up of an external fasciculus uncrossed, a middle fasciculus crossed and an internal fasciculus (Gudden's commissure) extend to their temporary termination in the lateral geniculate

bodies, the pulvinars of the thalami and the superior corpora quadrigemina, constituting neurones of the first order. The inferior corpora quadrigemina are not to be regarded as a part of the visual system. The macular bundle presumably has bilateral cerebral connections, as shown by the fact of the frequent preservation of central vision in hemianopsia. The internal fasciculus of the optic tracts, known as Gudden's commissure, connects the two internal geniculate bodies, and is not to be regarded as immediately concerned with the visual act, except through possible reflex relations. From the dorsal portions of the thalami the external geniculate bodies and the superior corpora quadrigemina fibers pass through a secondary neurone system to the cortex of the occipital lobes in the region of the cuneus, constituting the so-called optic radiations. The visual representation in the cortex is considerably greater on the mesial aspect of the hemisphere than externally.[1] Finally, there is anatomical evidence to show that certain fibers, originating in the cortex, pass to the retina through the superior corpora quadrigemina. Within the cranium the optic nerve is surrounded by pia, but at the optic foramen the dura is prolonged as a sheath, and with the pia is attached to the sclera. As elsewhere in the nervous system, between the pia and the dura lies the so-called arachnoid membrane. A lymph space, known as the supravaginal space, presumably of importance in its relation to the results of intracranial pressure, surrounds the optic nerve and its sheath. The intervaginal space of the nerve communicates directly with the intracranial meninges (Schwalbe).

**Lesions of the Retina.**—An ophthalmoscopic examination often throws light on many general diseases, and, in fact, may be a most important element in diagnosis. Such an examination should not be omitted in ordinary routine examinations, inasmuch as information of value may be derived from the appearance of the retina and of the optic disk in the absence of any noticeable disturbance of vision on the part of the patient.

**Retinitis.**—Alterations of the retina, usually manifested as an inflammation, occur in a large number of conditions, a few of which only may with propriety be considered briefly here. The general characteristics of retinitis, as seen by the aid of the ophthalmoscope, are diffuse cloudiness, particularly of the more central portions of the fundus, due to a loss of transparency of the retina and veiling of the choroid. The optic papilla shows more or less congestion with loss of clearness of outline, leading to a somewhat striated appearance. The veins are enlarged, tortuous, and engorged. Hemorrhages may occur with exudation, ultimately leading to a whitish appearance of the parts involved, through secondary changes. The color of hemorrhages, whether bright red, dark, or almost black, depends upon their age. If the nerve head or the choroid are involved in addition, the condition becomes a neuroretinitis or choroidoretinitis. Inasmuch as a retinitis is usually due to a constitutional infection, both eyes are commonly involved. The combination of the foregoing signs, together with certain characteristic groupings, make possible the recognition of somewhat

[1] Much work has been done on the cortical localization of the visual centres on the basis of clinical and pathological studies. For summaries of this work see Spiller, *The Cortical Centres of Vision*, p. 8, and Mills, *Position and the Subdivisions of the Primary Cortical Visual Area*, p. 75, in *The Eye and Nervous System*, Posey and Spiller, Lippincott, 1906. The exhaustive treatise by Wilbrand and Saenger, *Die Neurologie des Auges*, Wiesbaden, 1904, should be consulted in this and all other matters relating to the ocular nerves.

definite forms of retinitis. The most important of these to the physician are as follows:

(*a*) Syphilitic Retinitis (or syphilitic choroidoretinitis).—Syphilis manifests itself by somewhat varied lesions in the retina. The retinitis is usually associated with concurrent disease of the uveal tract, with frequent involvement of the choroid. The affection may be divided into two main types, one diffuse and the other circumscribed. In the diffuse type there is widespread but slight opacity of the retina with the occasional appearance of small grayish spots. The gradual disappearance of this opacity is accompanied by progressive changes in the pigment epithelium. The pigment cells occasionally wander into the retinal layers. In the circumscribed form, on the other hand, a characteristic feature is a yellowish-white exudate, occurring either in the macular region or along one of the larger vessels. When this occurs the ophthalmoscope will often disclose disease of the vessel wall. In the course of time these exudates are replaced by scar tissue, which predisposes to retinal detachment. Hereditary syphilis may likewise lead to a retinitis which is ordinarily observed only in the atrophic stage. There may be either small whitish areas, pigment clumps, or the connective tissue remains of former exudates. In late cases vision is affected and various scotomata, and defects appear in the visual field. Night blindness is a somewhat constant feature, and occasionally micropsia (apparent diminution in the size of objects) and metamorphopsia (distortion of the outlines of objects) may occur. The treatment of the condition is of the underlying cause, syphilis.

(*b*) Albuminuric Retinitis.—This occurs both in acute and chronic nephritis, and in the albuminuria of pregnancy. It is a frequent accompaniment of the so-called senile or granular kidney. Vision often suffers in the late stages of renal disease from the presence of such a retinitis. The ophthalmoscope discloses hyperæmia and swelling of the papilla and of the surrounding retina. Hemorrhages may occur in the nerve head and in the nerve-fiber layer of the retina. Irregular white patches may often be seen in the retina at a considerable distance from the papilla. The macula is not infrequently surrounded by a star-like arrangement of white dots, apparently converging toward it. An accompanying papillitis may be extreme, even suggesting the existence of a brain tumor. The white areas, the result of fatty degeneration, at times become confluent, forming single large white plaques. The hemorrhages vary widely both in size and number. An affection of the fundus also occurs in albuminuric conditions, which is entirely uncharacteristic. For example, simple retinal hemorrhages, retinitis hemorrhagica, or even optic neuritis and choked disk may occur.

The *prognosis* naturally depends upon the character of the cause. The outlook in the usually self-limited albuminuria of pregnancy is good, with a strong probability of a restoration of impaired vision. In cases dependent upon a more persistent cause optic atrophy may supervene with gradually failing vision. The influence of arteriosclerosis is of very great importance.

Retinitis may likewise occur in anæmia, leukæmia, and many other general and constitutional diseases which come rather within the province of the ophthalmologist than the practitioner of internal medicine.

**Lesions of the Optic Nerve.**—Inflammation, in general limited to the optic disk, producing a so-called optic neuritis or papillitis, or, in extreme

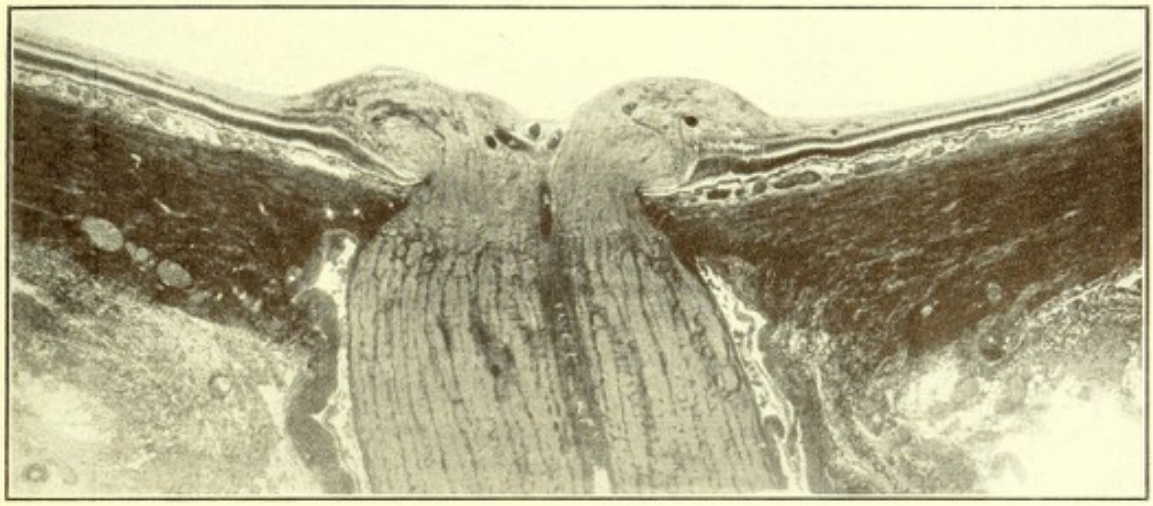
degrees of swelling, choked disk, is important to recognize. Although this sign may occur in a great variety of conditions, its usual cause is increased intracranial pressure from disease within the cranial cavity. Of such causes the most frequent are tumors, the location of which, rather than their character, is important in the production of a neuritis. Optic neuritis may also occur in other structural diseases of the brain, such as meningitis and abscess (rarely according to Bordley and Cushing), idiopathic hydrocephalus, and syphilis. In these conditions, in the absence of marked pressure, the mechanism of the production of the neuritis is often not easy to determine. Many cases of inflammation of the nerve head have also been described in connection with influenza, rheumatism, malaria, erysipelas, scarlet fever, and other general infections. It has also been observed occasionally in neuritis, anæmia, and in intoxications from alcohol and lead. Certain deformities of the skull, and particularly localized disease of the orbital region, more or less directly involving the nerves, may naturally lead to their inflammation.

**Mechanism of Papillitis, or Choked Disk.**—Various theories, no one of which is entirely satisfactory, have been suggested to account for the so-called inflammation of the nerve head. Von Graefe held to the view that the condition was due to a venous stasis, resulting from an obstruction to the return flow of blood from the cavernous sinus (Stauungspapille). On the basis of Schwalbe's work on the communication between the sheaths of the optic nerve and meninges, Manz and Schmidt-Rimpler concluded that mechanical pressure led to an œdema of the disk. Meehan regards as the important elements, œdema, congestion, and inflammation, due to a dropsy of the optic nerve lymph space induced by increased intracranial pressure. Parinaud considers choking of the nerve head to be due to an œdema of the optic nerve itself. A somewhat widely accepted theory, of which von Leber is an advocate, bases the optic neuritis upon the supposed presence of an irritant in the fluid distending the nerve sheath, produced in some way by the presence of a tumor or other source of irritation within the cranial cavity. A very significant recent investigation on the subject by Bordley and Cushing[1] tends strongly to support the so-called mechanical theories. In this paper these observers give the results of their investigation on choked disk particularly with reference to decompressive cranial operations. Of the various terms used to describe the condition of the nerve they prefer "choked disk" to designate all grades of the neuroretinal œdema. The view expressed is that early injection with stasis of the retinal vessels, œdema with elevation of the papilla, and final cellular infiltration with new tissue formation leading to atrophy are stages of the same process, due essentially to the distention of the sheath of Schwalbe by obstructed cerebrospinal fluid. It will be seen that the toxic element plays small part in this conception. The results upon the optic disk of decompressive cranial operations lead to the assumption that choked disk from practically all causes, including the changes occurring in nephritis, are due to mechanical rather than toxic causes. The evidence adduced in this article, in many ways entirely original, certainly is strongly in favor of the mechanical theory, and should do much to elucidate a long disputed pathological lesion. It must, however, still be regarded as possible that different causes may lead to similar ophthalmoscopic appearances. A true neuritis behind

[1] *Journal of the American Medical Association*, 1909, lii, 353.

# PLATE XXV

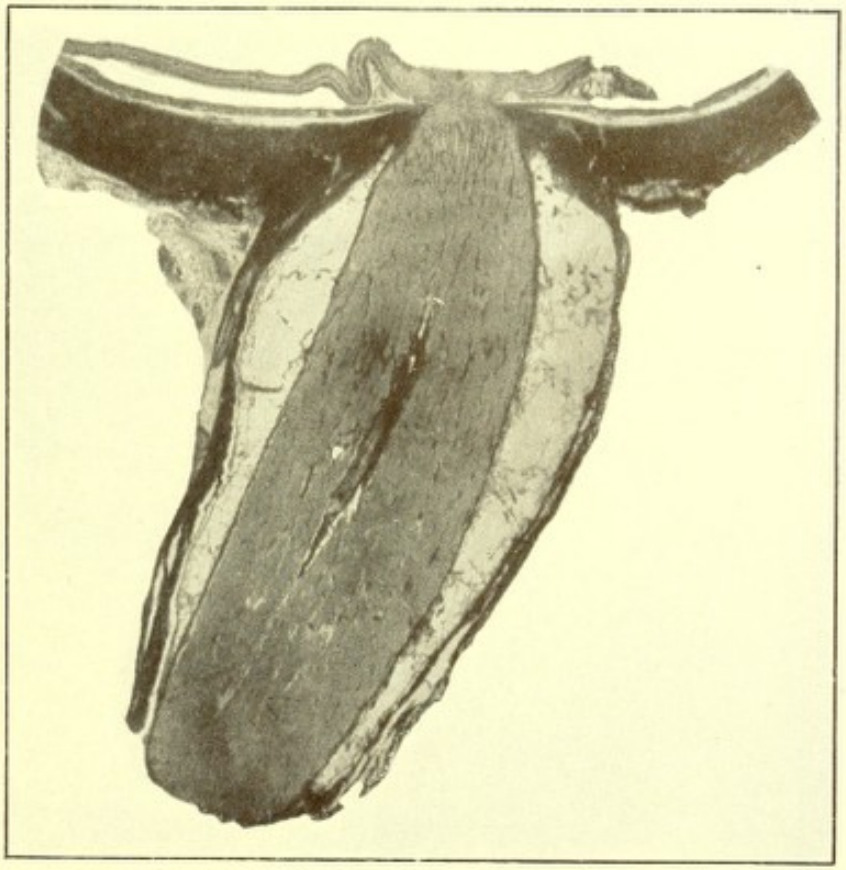
FIG. 1



Optic Neuritis (Choked Disk).

From a case of orbital tumor. Very slight œdema of subvaginal lymph space. (Reproduced through the courtesy of Dr. Frederick H. Verhoeff.)

FIG. 2



Œdema of Subvaginal Lymph Space of Optic Nerve.

With slight swelling of the disk, from a case of tuberculous meningitis. (Reproduced through the courtesy of Dr. Frederick H. Verhoeff.)

the bulb has also been observed in certain cases, and an engorgement œdema is a somewhat characteristic accompaniment of the typical picture of choked disk. If, on the other hand, a simple inflammation is the predominant factor, the elevation of the disk is less marked, and the retina is more apt to be involved. A marked swelling of the nerve head is in general to be regarded as an indication of pressure behind. The term optic neuritis is rather an unfortunate one. Papillitis more nearly describes the condition, but both terms are bad if it can be shown that inflammation plays no part. The term choked disk is useful as indicating a characteristic ophthalmoscopic appearance, associated with much swelling, but following Bordley and Cushing it may properly be given a much wider significance.

**Appearance of the Optic Disk in Papillitis.**—A somewhat artificial distinction may be made between a first stage of congestion with œdema and a second stage of true neuritis or papillitis. The general appearances are as follows: The nerve head early shows a deepening of color, with a gradual loss of outline. As the process goes on the optic disk swells, with a final complete loss of its form, so that ultimately its position can only definitely be determined by the convergence of bloodvessels toward its centre. The height of the swelling is often several millimeters. The appearance of the vessels is perhaps most characteristic. The arteries are small, and frequently concealed by swelling; the veins are dark, distended, tortuous, and appear to be divided, owing to the infiltration of the disk. Small flame-like hemorrhages are frequent, but not an essential of the picture. Vision may, for a long period, be unaffected, and the optic neuritis, therefore, go unnoticed. The visual fields show an irregular, concentric contraction. There is often an increase in the size of the normal blind spot and loss of appreciation of red and green. In brain tumors, particularly, such a papillitis may become what has been well described as a choked disk. A condition known as descending neuritis is associated with moderate swelling, and considerable exudation, producing discoloration and opacity of the papilla, but not limited to it. The retina may likewise be involved, leading to a neuroretinitis.

**Optic Atrophy.**—A degeneration of the optic nerve may occur either as a primary process or as secondary or consecutive to a papillitis. The general appearances of atrophy of the optic nerve when well marked are as follows: Alteration in color, varying from gray to greenish gray to clear white. Slight variations in color are extremely difficult to determine, and their significance should not be ventured except by those of wide experience in ophthalmoscopy. There is sinking of the centre of the disk, often with a characteristic appearance of the lamina cribrosa. The margins of the disk are usually distinct, but in secondary atrophy following papillitis they may be veiled for a long period. A broadening of the scleral ring indicates a shrinkage of the disk. The arteries, particularly in consecutive atrophies, are narrowed, in contrast with the veins which often retain their tortuosity. There is, however, finally an atrophy of the veins as well, with secondary connective-tissue changes. It is evident that the transition from papillitis to atrophy is extremely gradual, and that the foregoing appearances are clearly defined only when the atrophy has progressed to a considerable degree.

In contrast to papillitis, vision always suffers in optic atrophy. There are various defects in the field and development of central scotomata with

concentric limitation. The loss of the color field is usually greater than that of the form field. The pupillary light reflex suffers in proportion to the degree of atrophy.

*Primary atrophy* of the optic nerve occurs in tabes dorsalis, paralytic dementia, and, much less commonly, in multiple sclerosis. In the latter disease a pallor of the temporal half of the disk, as pointed out many years ago by Uhthoff, is not an uncommon appearance, presumably induced by partial lesions of the chiasm. Primary atrophy may likewise occur in chronic malaria, diabetes, syphilis, arteriosclerosis, and through the toxic action of certain drugs. A curious and inexplicable atrophy of the optic nerve on a hereditary basis, has been described, usually appearing between the eighteenth and twenty-third years. A stage of œdema and congestion, gray discoloration and pronounced atrophy, may be distinguished in this affection.

*Secondary atrophy* of the optic nerve is usually consecutive to an optic neuritis, but may occur through external causes leading to compression of the tracts and fibers of the nerve. The pathological appearances of optic nerve atrophy are similar to those of the degeneration of fibers in general.

*Diagnosis.*—The diagnosis of optic neuritis and optic atrophy depends almost wholly upon the use of the ophthalmoscope. Although lesions of this character may be regarded as probable through associated symptoms and through certain visual disturbances, an accurate determination of the condition is only to be obtained by a view of the eye grounds. The importance to the medical practitioner of such examinations has of late been increased by the significant observations of Horsley, Cushing, and others, relative to the improvement and even disappearance of an optic neuritis following decompressive brain operations. It has also been pointed out that in less grave conditions, as, for example, traumatisms of the head, leading to œdema or hemorrhage, much information may be obtained by following the changes in the nerve.

Although an optic neuritis may often be a determining point in diagnosis, its occurrence in unexpected conditions and its not infrequent failure to develop in well-defined, intracranial disease should lead to hesitation in its interpretation. Lesions of the spinal cord, for example, may occasionally give rise to a well-defined optic neuritis. Weisenburg and Thorington[1] have reported a definite optic neuritis in a case of syringomyelia, and the writer has recently observed the development of a neuritis, rapidly leading to blindness, in a case of primary tumor of the lower portion of the cord extending upward.[2] In both these instances the optic neuritis is, no doubt, to be attributed to a blocking back of cerebrospinal fluid leading to hydrocephalus. Optic neuritis occurring in the course of a myelitis has also been described, and presumably is to be attributed to the action of the toxic agent leading to the cord disease.[3] On the other hand, cases of brain tumor are in a considerable proportion of cases unaccompanied by optic neuritis, and especially in those in which pressure is not a marked symptom. The occurrence of optic neuritis in the course of intracranial syphilis is not

[1] *American Journal of the Medical Sciences*, 1905, cxxx, 1019.

[2] James Taylor and Collier, *Brain*, 1901, xxiv, 532, have discussed this matter in detail.

[3] Kernhensteiner, *Münch. med. Woch.*, 1906, liii, 802.

infrequent, particularly in those cases in which the symptoms develop in acute fashion. Such a case was observed by the writer in a man, six months after infection, with practical cure on vigorous treatment. Cases might easily be multiplied to illustrate the uncertainty of optic neuritis in various serious intracranial conditions. The fact, however, remains that the ease of its demonstration, and its definite significance when present, makes a study of the eye-grounds in all cases a matter of the utmost importance.

*Course and Prognosis.*—The course and outcome of affections of the optic disk are wholly dependent upon the character of the cause, whether it acts temporarily or permanently and whether it is capable of removal. A discussion of these matters would lead us beyond the nerve itself to a consideration of the underlying disease, and cannot, therefore, find a place here.

**Retrobulbar Neuritis.**—An acute form of retrobulbar neuritis has been described, among other causes, in influenza, multiple sclerosis, and poisoning by wood alcohol. In this condition there is rapid loss of sight in one eye, occasionally in both, with the development of a central scotoma and pain in the orbit. At first there are no changes in the optic nerve; later an optic neuritis may appear passing into atrophy, or the atrophy may apparently be primary. Complete blindness is rare, but a defective vision may remain. A chronic form of retrobulbar neuritis is more frequent. This also is rapid in onset; objects are veiled; the acuteness of vision is reduced; there is loss of color sense, with the development of a central scotoma, particularly for colors, usually oval in form. In extreme cases a central amblyopia may develop with complete loss of central vision. This is usually binocular, and often unobserved by the patient. Recovery is partial or complete. The special causes are excess in alcohol, tobacco, or both, hence it is more frequent in men than in women. The ophthalmoscopic appearances are hyperæmia of the papilla, loss of outline, with the development of white striæ along the vessels. Following this temporary phase there is apt to be a pallor of the temporal side of the disk, the nasal portion remaining normal. Rarely the whole papilla undergoes a so-called white atrophy. The essential treatment is total abstinence from alcohol, tobacco, or other agents which may be regarded as responsible for the condition.

**Amblyopia and Amaurosis without Ophthalmoscopic Changes.**—A great variety of conditions affecting sight may occur entirely without objective evidence in the fundus. Some of the more important of these are as follows: A congenital disturbance of vision for colors, or total color blindness; an amaurosis due to trauma, uræmia, diabetes, malaria, from loss of blood, from the abuse of drugs, of which methyl or so-called wood alcohol may be taken as an example; various disturbances of vision in hysteria with inversion of color field; monocular diplopia; conjugate deviation and general narrowing of the visual fields are the more important. Night blindness (nyctalopia), day blindness (hemeralopia), red vision (erythropsia), reduced size of objects (mytopsia), and increased size of objects (megalopsia) are some of the curious disturbances of vision which cannot be more than mentioned here.

**Vision.**—Apart from the ordinary tests by means of charts, it is often useful and essential to diagnosis to determine the degree of eccentric vision and its possible limitation. Such an examination may be made roughly by means of the hand or any other object brought into the field of vision from various directions. Gross defects may be determined by carefully observing

the point at which the patient first sees the object. Hemianopsia, for example, may easily be so determined. A somewhat more accurate method is to require the patient to look at a definite mark on a blackboard while seated at a distance of from one and a half to two feet. An object, as, for example, a piece of chalk, brought into the fields from various points, when recognized by the patient as a moving object, serves as an indication of the extent of the visual field in that direction, and may be so indicated on the board. In making such examinations one eye should be closed while the other is being examined. Colors are less easily seen in the order from without inward, blue, red, green. Cushing has recently called attention to an inversion of color fields as an early sign of brain tumor, similar to that seen in certain cases of hysteria. A perimetric examination is essential for an accurate determination of the visual fields.

**Disturbances of Vision.**—The general disturbances of vision are: (1) Concentric narrowing of the fields; (2) scotomata, or defects of vision elsewhere than on the periphery; (3) hemianopsia. Mention has been made of concentric narrowing and various scotomata. Hemianopsia is a disturbance of more importance for our present purpose, inasmuch as it indicates not only disturbance in peripheral vision, but also is of very great importance in determining the location of lesions within the cranial cavity.

**Affections of Optic Chiasms and Tracts.—Hemianopsia.**—Destructive lesions of the optic nerves, ventral to the chiasm, naturally give rise to unilateral or bilateral blindness and need no further mention. Owing to the partial crossing in the chiasm, however, partial disturbance of vision develops when the lesion lies either at that point or in the course of the optic tracts to their central terminations, characterized by losses of portions of the visual field in each eye. The usual lesion of the chiasm occurs in its central portion, leading to disturbance in the nasal portions of the retina with consequent affection of vision in the temporal field. This condition is known as bitemporal hemianopsia. Naturally, if the process in the chiasm extends, total blindness may result, or total blindness in one eye with retained half vision in the other. The occurrence of binasal hemianopsia, through symmetrical lesions limited to the outer chiasm bundles, is exceedingly unusual, as well as lesions of the chiasm producing inferior and superior hemianopsia, although it is perfectly conceivable that any of these disturbances may occur, and some such have been described. Syphilis, tumors, particularly of the hypophysis, and hydrocephalus are the more frequent causes of disturbances in the chiasm leading to hemianopsia.

Affections of the optic tract lead to homonymous bilateral hemianopsia, due to the fact that each optic tract contains fibers supplying the temporal side of one retina and the nasal side of the other, the fibers to the nasal side being crossed, and to the temporal side being uncrossed. The same visual disturbance is produced by any lesion either of the primary optic centres or of the occipital lobe of one side. With the exception of the so-called Wernicke hemianopic pupillary reaction the localizing significance of homonymous hemianopsia is relatively slight, apart from coincident lesions of neighboring portions of the brain or cranial nerves. Affections of the visual area in the occipital cortex, produced by tumor, areas of softening or injuries, may lead to isolated hemianopsia. The same is true of lesions of the optic radiations, which, however, if affected on the left side, usually are accompanied by aphasia. Certain facts of importance regarding

the location of the lesion producing the hemianopsia may be derived from a detailed study of the visual field, particularly with reference to the dividing line between the seeing and the blind areas. In complete lesions of the optic tract the hemianopsia is usually complete, by which is meant that it passes vertically through the fixation point. Such a complete hemianopsia is less frequent in disease of the external geniculate body, and occurs only

FIG. 7

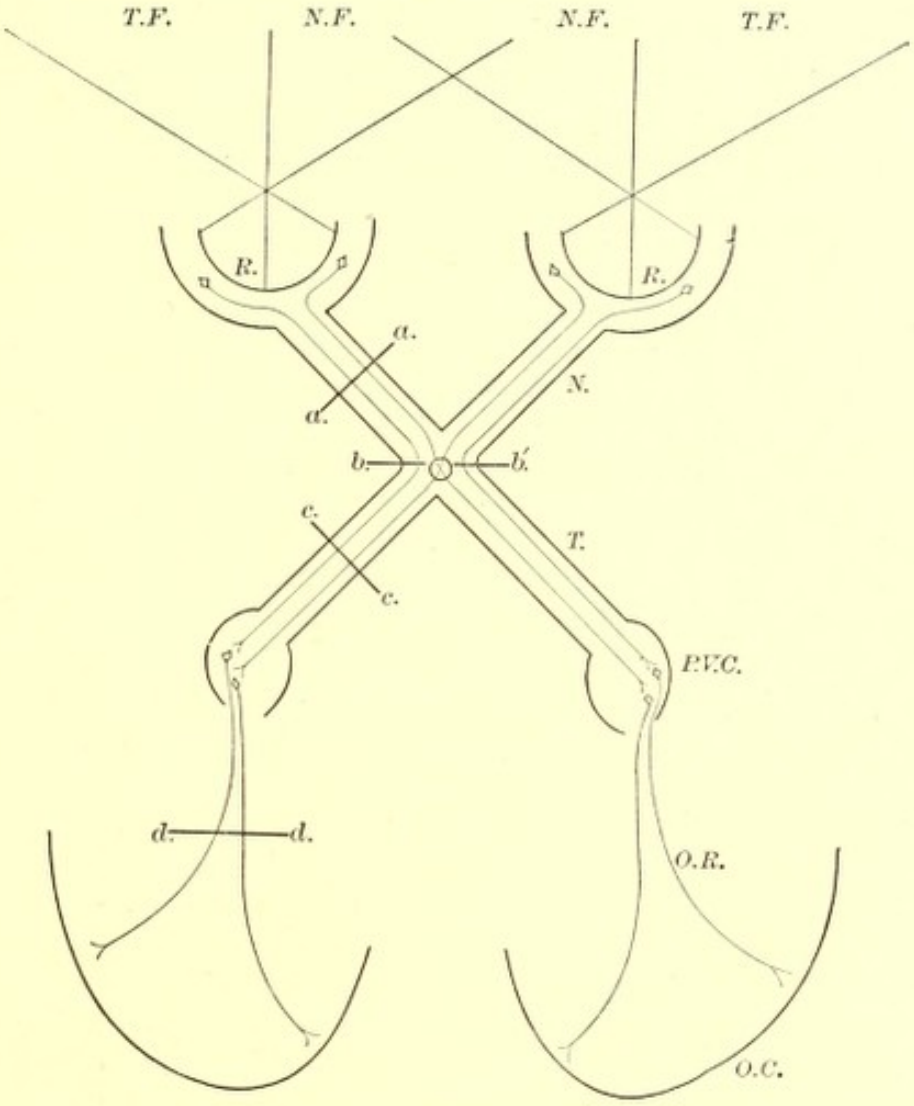



Diagram of lesions producing hemianopsia. *T.F.*, temporal field; *N.F.*, nasal field; *R.*, retina; *N.*, optic nerve; *T.*, optic tract; *P.V.C.*, primary visual centres; *O.R.*, optic radiations; *O.C.*, occipital cortex; *a.a.*, lesion producing unilateral blindness; *b.*, lesion producing unilateral hemianopsia; *b.b.'*, lesions producing binasal hemianopsia, very rare; *O.*, at centre of chiasm, lesion producing bitemporal hemianopsia; *c.c.*, *d.d.*, lesions producing homonymous hemianopsia. Hemianopic pupillary reflex in lesions anterior to primary visual centres, and behind the chiasm.

exceptionally in affections of the cortex. The incompleteness of the hemianopsia in lesions above the optic tracts must be regarded as due to the separation of the fibers and the probable separate representations of the visual fields in the cerebral cortex. The frequent sparing of the macula, for example, may be explained on the basis of a separate cortical localization for that group of fibers.

The association of homonymous hemianopsia with concentric narrowing of the visual fields is, no doubt, often to be explained, as Uhthoff has pointed out, by a complication of organic disease with a functional disturbance. Affections of the color fields, appearing in hemianopic form, have been described, and various minor anomalies of the more definite picture which is here presented. Bilateral hemianopsia is essentially identical with blindness, except that in several cases central vision has not been destroyed, and the light reflex of the pupil is retained, provided the lesion lies beyond the reflex arc.

**Hemianopic Pupillary Reaction.**—A further possibility of localizing the lesion producing hemianopsia is afforded by the so-called hemianopic pupillary reaction, to which Wernicke has called attention. If the lesion lies within the reflex arc from the retina to the central visual centres, illumination of the amblyopic half of the retina will lead to no pupillary reaction, owing to the severance of the reflex arc. Such illumination applied to the seeing half of the retina will produce the ordinary reaction. If, however, the lesion lies behind the reflex arc between the optical centres and the visual cortex, illumination of the blind side of the retina will produce a normal pupillary response. This test is no doubt of value, but it is evident that its successful performance demands much care, and that even with proper apparatus the isolated illumination of one retinal half is difficult of accomplishment.

**Mind Blindness.**—In the consideration of the disturbances of the optic nerve the condition of so-called mind blindness demands mention. In this disorder the patient sees objects, but is unable to recall their significance, or, as a patient of Wilbrands', quoted by Oppenheim, expressed it, sight is possible with the eyes, but not with the brain. Such persons fail to recognize the significance of familiar objects, and even accustomed rooms and locations appear strange to them. This state of so-called mind blindness is not to be confused with optical aphasia. In this latter condition objects are both seen and their significance recognized, but cannot be designated in words through the medium of the sense of sight. In such cases the proper designation may, at times, be aroused through another sense, as, for example, a key may be recognized as a key, but cannot be named until it is put into the patient's hand and a demonstration of its use requested.

## OCULOMOTOR, TROCHLEAR, ABDUCENS (THIRD, FOURTH, SIXTH) NERVES.

The three nerves, third (oculomotor), fourth (trochlear), and sixth (abducens), which control the movements of the eyeball and pupil, may be best grouped, although the function of each nerve is distinct and demands individual consideration.

**Anatomical Relations.—Third Nerve.**—The fibers of this nerve arise from an elongated nucleus lying ventral to the aqueduct of Sylvius, particularly beneath the ventral corpora quadrigemina. Careful studies by Starr, Siemerling, and others have demonstrated the probable association of certain fairly well-defined groups of nerve cells with definite branches of the nerve, but there is still much doubt as to the exact relationships. Tsuchida has, for example, shown wide differences in distribution. Doubt

has also been thrown upon the relation of the so-called Edinger-Wesphal nucleus and nucleus of Darkschewitsch to the main nuclear groups of the third nerve. The fibers arise for the most part from nuclei of the same side, but there is no doubt a partial crossing. Through the dorsal longitudinal bundle relations are unquestionably established with the sixth nerve, and presumably with the fourth and indirectly with the eighth and possibly others. Somewhat well determined connections with the third nerve are as follows: (*a*) With the cerebral cortex in the dorsal portion of the inferior frontal convolution, ventral to the precentral fissure (Mills); (*b*) indirectly with the cortical visual centres in the occipital cortex, presumably through the superor corpora quadrigemina, the superior brachia, and the optic radiations; (*c*) through the dorsal longitudinal bundles connections are effected with the fourth and sixth nerves, with Deiters' nucleus of the eighth and with the nucleus of the seventh, particularly with those cells which supply the orbicularis palpebrarum and the corrugator supercilii muscles for the purpose of bringing about coördination with the levator palpebræ supplied primarily by the third nerve. The third nerve proper, after leaving its nuclei of origin passes ventrally through the substance of the midbrain, appearing on the surface between the crura cerebri, thence passing along the base of the brain through the sphenoidal fissure and dividing into a superior and inferior branch. The superior branch supplies the superior rectus and the levator palpebræ superioris. The inferior branch supplies the internal and inferior rectus and the inferior oblique muscles. The fibers for the sphincter pupillæ and ciliary muscles pass through the ciliary ganglion.

**Fourth Nerve.**—The fourth or trochlear nerve arises from a group of cells caudad to the third nerve nucleus in the central portion of the gray surrounding the aqueduct of Sylvius. It lies in close proximity and in close relation to the dorsal longitudinal bundle. The two fourth nerves take a unique course in that they decussate dorsad to the aqueduct in the superior medullary velum and thence pass around the crura to appear on the ventral surface approximately at the junction of the crura and the pons. This nerve also enters the sphenoidal fissure and supplies the superior oblique muscle only. The connections of the nucleus of the nerve are presumably with the frontal convolutions of the cortex, with the nuclei of the third and sixth nerves through the dorsal longitudinal bundle, and presumably also with the cochlear portion of the eighth nerve through the superior olive.

**Sixth Nerve.**—The sixth nerve or abducens arises from nuclei lying beneath the floor of the fourth ventricle considerably caudad to the fourth nerve and in close relation anatomically to the seventh nerve, which forms a loop about its nucleus. The nerve fibers pass from the nucleus toward the ventral aspect of the pons, emerging near the median line, thence passing by way of the sphenoidal fissure to the outer side of the orbit and supplying the external rectus muscle. In addition to the relations already spoken of with the fourth and third nerves, a reflex path is undoubtedly established between the auditory apparatus and the centres for the various eye muscles primarily through the sixth nerve and secondarily through the fourth and third by way of the dorsal longitudinal bundle. The cortical relations of the sixth nerve are indefinite. They are presumably crossed, as is the case with other cranial nerves.

It is evident that the relations of the three nerves governing the movements of the eyeball are extremely close, which naturally is a development of the function of binocular vision. It is also of interest, and undoubtedly of very great clinical importance, to recognize the connection between these three motor nerves and the sensory eighth nerve. The preservation of equilibrium is undoubtedly dependent upon this relationship, together with the complex reflex connections of the eighth nerve with the cerebellum and other portions of the central nervous system.

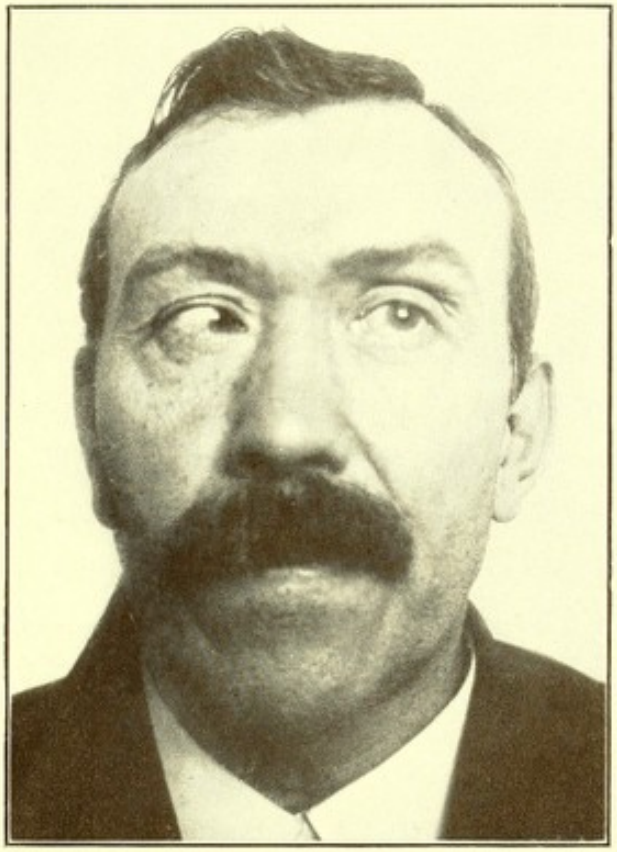
**Symptoms of Paralysis of Ocular Nerves.**—A complete paralysis of all the muscles providing for the movements of the eye gives rise to the condition known as total *ophthalmoplegia*. This may develop partially either as an internal ophthalmoplegia, in which the pupillary muscles are chiefly affected, or as an external ophthalmoplegia, in which those governing the globe are alone involved.

Under normal conditions the external rectus muscle (sixth nerve) moves the bulb directly outward. The internal rectus (third nerve) moves it directly inward; the superior rectus (third nerve) upward and inward, also with rotation of the upper axis slightly inward; the inferior rectus (third nerve) draws the globe downward and slightly inward. The superior oblique (fourth nerve) moves it downward and outward with inward rotation of the upper end of the vertical axis. The inferior oblique finally draws it upward and outward, with outward rotation of the upper end of the vertical axis.

A defect of the lateral action of the eyes, normally brought about by the internal rectus of one eye and the external rectus of the other, is usually easily observed. If the movement of both bulbs to one or the other side is interfered with, the condition is known as conjugate deviation or associated paralysis (Blicklähmung). The paralysis of eye muscles leads to the following conditions: (1) Loss or limitation of special movements of the bulb; (2) diplopia; (3) secondary contracture of the antagonists; (4) secondary deviation of the sound eye; (5) false projection of the visual field and abnormal position of the head.

From the general clinical standpoint the development of double vision is particularly important as an indication of more or less serious intracranial disease. It should be borne in mind that in congenital conditions or in persons in whom a strabismus has existed for a long time, a diplopia must exist, although often not recognized by the patient. This is due to the fact that through training and habit the retinal image of one eye is ignored after the manner of a person looking through a microscope with both eyes open. On the other hand, in a diplopia coming on suddenly the double images are at first very annoying to the patient, often leading to the semivoluntary closure of one eye to overcome the confusion of a double image. It is well to remember this fact in arriving at a conclusion regarding the length of time a diplopia has existed. A simple examination, asking the patient to fix the moving finger of the examiner with both eyes, is often enough to determine the existence of double vision. The patient should be directed to state the exact moment at which two pictures come into the field. It is sometimes useful to use a colored glass before one eye in order that the two images may be sharply distinguished. The image seen by the sound eye is spoken of as the true image, that by the affected eye as the false image. If the false image is on the same side as

# PLATE XXVI



Paralysis of Left Sixth Nerve.

Occurring in the course of tabes. (Massachusetts General Hospital.)

the eye by which it is seen, the diplopia is spoken of as homonymous, otherwise it is spoken of as crossed. If, for example, the right image disappears with the closure of the right eye, the diplopia is homonymous. In general, convergent strabismus is accompanied by homonymous, divergent strabismus by crossed diplopia. The position of the head is often altered in order to overcome the annoyance produced by a diplopia. If, for example, through weakness of the external rectus of the right eye, diplopia is produced by looking toward the right, such a patient almost inevitably will adjust his head in such a way as to obviate this possibility, namely, he will look more toward the left. Such altered position of the head is strikingly seen in cases of conjugate deviation, in which it is impossible to bring the eyeballs into the median line. The writer has recently observed a case of tumor of the pons with marked conjugate deviation in which this phenomenon was strikingly shown. The curious condition of so-called monocular diplopia or polyopia is of rare occurrence. It has been observed and adequately described in hysteria (Janet, Prince).

**Paralysis of Special Muscles.—External Rectus.**—This muscle is frequently paralyzed through lesion of the sixth nerve at some part of its long course from the pons forward. A convergent squint is produced by this paralysis, which is increased through secondary contracture of the internal rectus. The diplopia is homonymous, with a widening of the space between the upright images as the object is moved outward toward the side of the paralyzed muscle.

**Internal Rectus.**—Isolated paralysis of this muscle gives rise to a defect of inward movement with consequent divergent squint. There is a crossed visual image in that part of the field corresponding to the unaffected eye. The space between the images increases as the object is moved toward the side of the sound eye.

**Superior Rectus.**—Limitation of upward movement with rotation of the bulb, due to the tension of the inferior oblique, is induced by paralysis of this muscle. There is a crossed double image in the upper half of the field. The upper or false image is oblique and separates from the true image on looking upward.

**Inferior Rectus.**—Paralysis produces downward limitation. The eye is turned outward and also is rotated outward in the effort to look down (superior oblique). The diplopia is crossed in the lower half of the field. The false image is lower than the true and the distance between them increases downward. The images are oblique.

**Inferior Oblique.**—Movements upward and outward are limited. On looking upward the bulb is rotated inward. There is homonymous diplopia in the upper half of the field. The images are oblique.

**Superior Oblique.**—Paralysis of this muscle produces slight disturbance of motion. On looking downward there is a slight turning inward of the eye, with homonymous diplopia in the lower half of the field and convergent strabismus. In this paralysis on going down stairs the steps often appear to be double.

A slight exophthalmos is produced by a paralysis of all the external muscles of the eye, which, on account of the immobility of the globe, is not likely to be confounded with the exophthalmos of Graves' disease, hydrocephalus, or postorbital tumors. The paralyses of convergence and divergence which occasionally occur have been supposed to be due to disturbance

in a special cortical centre which presides over these associated movements. Such paralyses are apt to be accompanied by vertigo. Conjugate deviation of the eyes, due either to spasm or paralysis, is a sign of considerable diagnostic importance and is strongly indicative of a lesion within the pons having relation to the dorsal longitudinal bundle through which the associated movements of the third and sixth nerves are brought about. Spiller[1] has recently discussed this whole matter in much detail, and reaches the following conclusions: Persisting paralysis of associated lateral movement indicates a lesion of the posterior longitudinal bundle. Persistent paralysis of associated upward or downward movement indicates a lesion in the vicinity of the oculomotor nucleus. Such paralyses are not the result of a lesion of extracerebral nerve fibers. Cortical lesions may produce such paralyses, but they are temporary unless due to a bilateral brain lesion. In hysterical states such associated paralyses may likewise occur and also as a result of inflammatory lesions or others produced by alcohol and syphilis; tumor is a common organic cause, and if such associated paralyses persist operation can never be regarded as more than palliative.

*Nystagmus,* or involuntary oscillation of the bulbs on fixation, either direct or lateral, is commonly met with in conditions of muscular inequality, in multiple sclerosis, and as one of the signs of lesions in the posterior fossa, particularly in relation to the eighth nerve. Although the cause may be varied, the general explanation must be sought in a loss of coördination between the various muscles of the eye. Depending upon the movement which the eyeball takes, the nystagmus is spoken of as horizontal, vertical, or, more rarely, rotatory. The movements are very much more marked on lateral fixation, and it should be borne in mind that under these conditions considerable oscillation of the bulbs must be regarded as within normal limits. Nystagmus is often conspicuous in the blind, with whom fixation is not possible, and also in persons, especially children, with defective vision.

**Intra-ocular Muscles.**—The muscles of the iris, associated with alteration in the size of the pupil, are the so-called constrictor and dilator pupillæ. The ciliary muscle, presumably important in bringing about the act of accommodation, consists of unstriped longitudinal and circular fibers forming a circular band about one-eighth of an inch in breadth between the choroid and the iris. The nerve supply of these internal muscles of the eye is through the nasal branch of the ophthalmic division of the third nerve and the short ciliary nerve from the ciliary ganglion. The circular fibers of the iris receive their innervation from the third nerve and the radiating fibers from the sympathetic, hence paralysis of the third nerve leads to dilatation of the pupil, and paralysis of the sympathetic to its constriction. Naturally the opposite results occur when the lesion of the nerves involved is irritative rather than destructive.

The pupils narrow physiologically under the following conditions: Light on the retina; consensual reaction or the phenomenon of pupillary constriction of one eye when light is thrown into the other; narrowing of the pupil for accommodation for near objects. The path by which the reflex action of the pupil is determined is still in dispute. The so-called reflex centre has been variously placed in the ciliary ganglion, in the oculomotor nucleus, the corpora quadrigemina, the upper portion of the spinal cord,

[1] *Jour. of Nerv. and Ment. Dis.,* 1905, xxxii, 417.

and in the oblongata. Experimental and clinical studies give no uniformity of result as to the exact location of this reflex centre. Spiller is inclined to think that there is a limited area in the oblongata near the respiratory centre which is inhibitory in function, that cerebral to this there is a subordinate centre, and that the complete reflex is, therefore, conditioned by one centre having its location in the corpora quadrigemina and another at the spinal end of the fourth ventricle. It is probable that the optic nerve contains special fibers which are not identical with those fibers which subserve sight, but which constitute a part of the reflex arc partially crossing in the chiasm. In rare instances these fibers have apparently retained their reflex function when the optic nerves have been extensively diseased.

**Pathological Variations in the Pupil.**—The ordinary pathological alterations in the pupil may be summarized as follows, after Uhthoff, quoted by Weeks: (1) Loss of pupillary light reflex with retained convergence and accommodation (Argyll-Robertson pupil); (2) loss of convergence and accommodation with retained light reaction; (3) loss of pupil reflex for light and accommodation; (4) loss of all reflex movements of the pupil (ophthalmoplegia interna); (5) loss of direct light reflex in an amaurotic eye and consensual light reflex in the fellow eye due to loss of vision from a lesion peripheral to the chiasm; (6) loss of sensory or psychical reflex action; (7) abnormal miosis with retention of light and convergent reflexes arising from stimulation of the sphincter or paralysis of the dilatator; (8) abnormal dilatation of the pupil (spastic mydriasis) due to stimulation of the dilatator or paralysis of the sphincter with retention of light and convergent reflexes; (9) difference in size of the pupils (anisocoria); (10) changing anisocoria; (11) irregularity of pupil outline; (12) change in the size of the pupil under uniform illumination (hippus); (13) paradoxical pupillary reflex or dilatation where contraction would ordinarily be expected; (14) hemianopic pupillary reaction.

Of these conditions several only require special comment here. From a practical standpoint the Argyll-Robertson pupil is of very great importance. In testing for this reaction it should be remembered that in elderly persons a light reaction is naturally slow, that there are many transitional conditions between a normal pupillary reflex and an immobile pupil, and that an accompanying convergence or accommodative reaction must not be mistaken for a light reaction. Although opinions differ, syphilis is probably not always the cause of this peculiar pupillary phenomenon. It is most often met with in the parasyphilitic disease, tabes. In 166 cases Uhthoff found an Argyll-Robertson pupil associated with tabes in 67.6 per cent., with dementia paralytica in 8.8 per cent., in syphilis without tabes in 8.17 per cent. In other cerebral diseases it occurred in only 5.87 per cent., and the remaining very small percentage in other disorders of the nervous system. In two other series of statistics made by Siemerling it appeared in one that 89 per cent. of all cases of Argyll-Robertson pupil occurred in general paralytics or sufferers from tabes, and that the proportion amounted to 90 per cent. if syphilitics were included. In the other statistical summary, made on the basis of 1639 cases of Argyll-Robertson pupil, 94.7 per cent. occurred in persons suffering from general paralysis, tabes, or syphilis.[1]

As previously suggested, the location of the lesion producing this sign

[1] Bach, *Pupillenlehre*, Berlin, 1908, p. 144.

remains in doubt. The fact of its frequent occurrence in tabes has led to the supposition that the cord was primarily responsible, but the evidence for this is insufficient. Internal ophthalmoplegia, due to the paralysis of the ciliary and sphincter muscles, is in many cases presumably dependent on a nuclear lesion, with syphilis often conspicuous in the etiology. An extraordinary case with the following signs occurring in a patient with general signs of hysteria has recently been reported by J. J. Putnam.[1] The main conditions in this case were dilatation with complete immobility of the pupil of one eye associated with spasm of the ciliary muscle of the other eye, the pupil of which was small, but which responded perfectly to light and with convergence. The fundus on both sides was normal, but the vision of the eye with the spastic ciliary muscle was decidedly lessened in acuity and field, whereas the vision of the eye having the immovable pupil was normal both for distance and near objects. This anomalous state of affairs Putnam was inclined to regard as hysterical, although much doubt has been expressed as to the possibility of such phenomena in that neurosis.

Slight differences in the size of the pupils is not to be regarded as pathological. If pronounced, however, suspicion should be aroused and attention directed toward the possibility of tabes, dementia paralytica, tumor of the neck, or tumor of the upper portion of the cord.[2] Irregularity of pupillary outline, unless due to local disease of the eye, is to be attributed in most cases to lesions of the nerves innervating the iris and to a consequent imperfect action of the muscles. Such a condition of the pupils is often an early sign in tabes and dementia paralytica, and may precede the development of an Argyll-Robertson pupil.

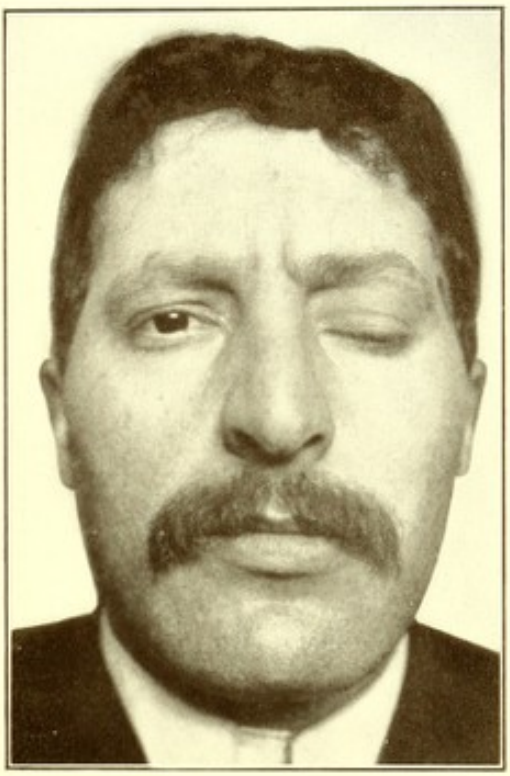
**Special Conditions of Paralysis of Ocular Nerves.**—Owing to the wide distribution of the third nerve to both the external and internal muscles of the eye, its paralysis gives rise to the most striking signs. If all branches of this nerve are affected by the lesion, the paralysis induced gives rise to external squint, ptosis, dilated and inactive pupil, and certain other less conspicuous defects in the movement of the eye from involvement of other muscles than the internal rectus. Owing also to the wide distribution of the nerve, individual muscles are often the seat of paralysis, explained in some instances at least by the separation and distinct representation of the individual muscles in the nuclei of origin. Such partial palsies are, in fact, more often met with than the complete paralysis, and occur notably in tabes, basal gummata, or meningitis, and in other conditions within the cranium which lead to pressure or partial destruction of the nerve. Among the common more or less isolated paralyses of muscles supplied by the third nerve, ptosis takes the first place. A great variety of conditions may produce this disturbance. It may, for example, occur as a congenital condition in which there is from birth a certain incapacity to open the eye widely through weakness of the levator. This affection appears at the time to have a hereditary basis. Acquired ptosis may arise from traumatism, vascular disorders, increase of intracranial pressure,

[1] *Boston Med. and Surg. Jour.*, 1907, clvi, 434.

[2] The importance of marked pupillary inequality is illustrated by a case in which a failure to attach proper significance to this phenomenon led to a failure on my part in the diagnosis of a spinal cord tumor of the lower cervical cord, later correctly diagnosed by Dr. Henry C. Baldwin and removed by Dr. C. L. Scudder, *Boston Med. and Surg. Jour.*, 1906, cliv, 623.

# PLATE XXVII

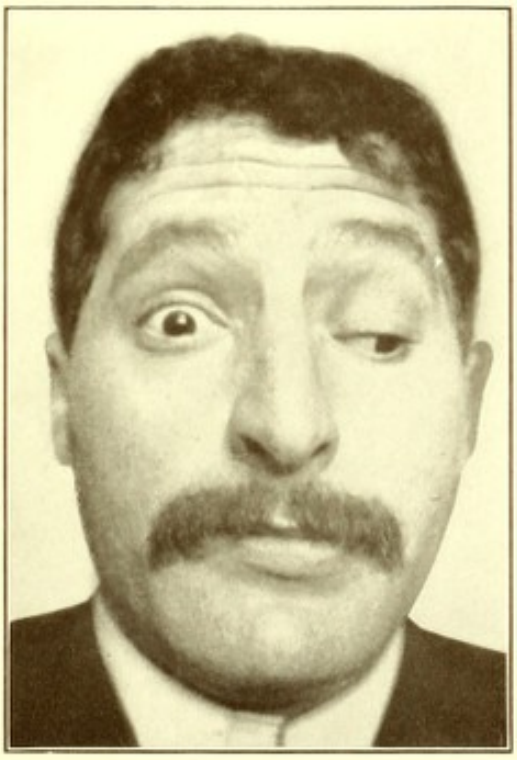
FIG. 1



Paralysis of Left Third Nerve.

Case of ophthalmoplegic migraine. (Dr. G. A. Waterman.)

FIG. 2

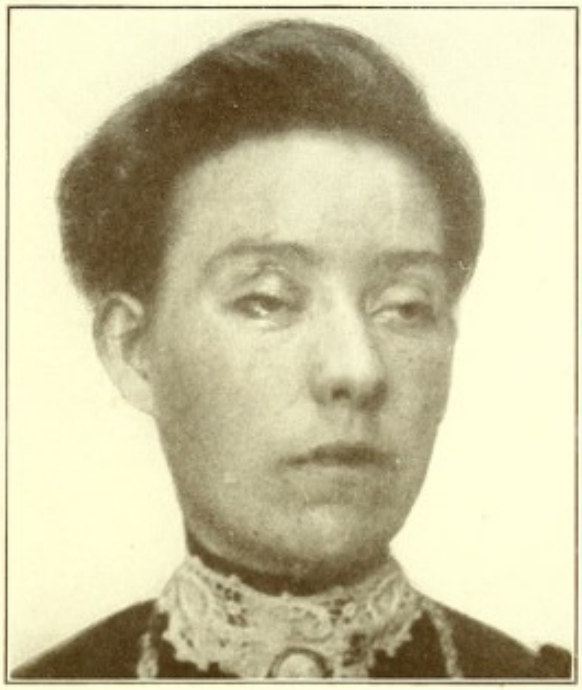


Same. Attempt to Open Eye.

Showing action of frontalis muscle. (Dr. G. A. Waterman.)

# PLATE XXVIII

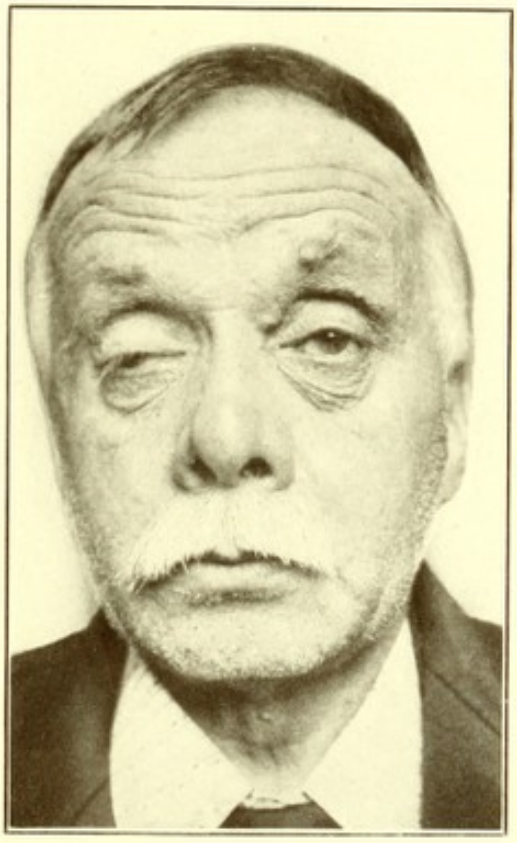
FIG. 1



Double Ptosis.

An operation to raise the lid has been attempted on the right side. (Massachusetts General Hospital.)

FIG. 2



External Ophthalmoplegia.

Involvement of both third nerves and of left sixth nerve. All movements of eyeballs imperfect except of the right outward. Normal pupillary reactions. (Massachusetts General Hospital.)

and notably in myasthenia gravis and in Gerlier's disease. Ptosis is extremely frequent as part of the more general involvement of the third nerve. In relatively young persons this sign, often associated with paralyses of other ocular muscles, is significant of syphilitic infection. The sixth nerve supplying the external rectus is likewise frequently involved in paralytic conditions, giving rise to internal squint. An association of polyuria with paralysis of the sixth nerve has been described. The causes of its paralysis are, however, in general similar to those affecting the third nerve. Paralysis of the fourth nerve is often difficult to determine and is of small practical significance. It is rarely involved alone.

**Recurrent Oculomotor Palsy.**—This extraordinary condition, as its name implies, is a recurrent palsy of one third nerve and always the same nerve, occurring usually at regular intervals, lasting days, weeks, or months, and then disappearing in whole or in part. Children are usually affected. The paralysis is accompanied by pain in the head or through the eye on the side affected, also by nausea and vomiting, in this respect having a certain analogy to migraine. Unlike migraine, however, the headache and vomiting may continue for a week. Often there is headache more or less severe at intervals of about a month, at times accompanied by oculomotor paralysis. In typical cases the whole supply of the oculomotor nerve is affected; in others, individual muscles. A somewhat doubtful distinction has been made between those cases in which there is an interval entirely free from paralysis and those in which a certain amount of paresis persists. Oppenheim, following Charcot's theory, finds the most probable explanation in a vasomotor disturbance allied to ordinary hemicrania. In general, the relations of the condition to ordinary ophthalmic migraine[1] and to the less frequent ophthalmoplegic migraine is a matter of very great interest, a discussion of which, however, is not in place here. A similar periodic abducens and trochlear paralysis has been described. Some of the cases are apparently progressive, others stationary. The prognosis is doubtful, and the treatment uncertain, owing to the lack of exact knowledge of the predisposing causes.

**Ophthalmoplegia Interna.**—This, on the whole, unusual condition, is due to a paralysis of the sphincter pupillæ and ciliary muscles, and is usually unilateral. Rarely one of these muscles may be involved without the other, and not infrequently the process may extend to the extrinsic muscles of the eye, to involvement of other muscles supplied by the third nerve, as well as to those supplied by the fourth and fifth nerve. The lesion of this affection is presumably nuclear. Syphilis is apparently to be regarded as the most frequent cause, but such a paralysis has also been observed in diphtheria.

**Ophthalmoplegia Externa.**—More commonly a paralysis of the external eye muscles, with or without a coincident disturbance of the intrinsic muscles, occurs in the form, if the onset is acute, of a so-called superior encephalitis, or, if the course is from the outset progressive, as chronic ophthalmoplegia.

**Superior Encephalitis** (Wernicke).—In 1881 Wernicke[2] described a symptom complex which he called acute hemorrhagic superior poliencephalitis.

[1] Walton has recently advanced the idea that ordinary migraine is an occupation neurosis, involving the visual centres, centre of accommodation, intrinsic and extrinsic muscles of the globe and certain muscles outside the orbit, *Jour. Amer. Med. Assoc.*, 1908, li, 200.

[2] *Lehrbuch der Gehirnkrankheiten*, 1881, Sec. 47, 229.

Alcohol was regarded as playing an important part in the etiology in two of the three cases originally described, and inflammatory changes were found in the neighborhood of the third nerve as the predominant lesion. The essential symptoms were paralysis of ocular muscles, of quick onset and rapid progress, leading finally to a practically total paralysis of the muscles involved, death resulting in from ten to fourteen days, with certain general cerebral disturbances suggestive of alcoholic intoxication. Further observation has shown that Wernicke's conception must be widely extended, and that a fairly well-defined group of conditions exists, due apparently to infection in the broad sense, which are characterized by a great variety of paralyses of ocular muscles, both external and internal. The pathological anatomy is not uniform but various nuclear and peripheral degenerations have been found. There is increasing evidence to show that the relationship between this so-called superior encephalitis, poliomyelitis, and various other acute inflammatory conditions of the central nervous system is an exceedingly close one, if, in fact, this whole group of conditions is not identical.[1]

**Chronic Ophthalmoplegia.**—Although the condition known as chronic ophthalmoplegia is also hardly to be regarded as a disease *sui generis*, its striking symptomatology justifies its separate consideration. In most cases this usually progressive form of paralysis of eye muscles forms part of a more general process, and is often associated with bulbopontine degenerations of motor nuclei and occasionally with degenerative cord lesions. The symptom complex, originally described by von Graefe, is characterized by a slow development and long course, inasmuch as no centres essential to life are involved in its progress. The affection usually begins with involvement of a single muscle, often the levator palpebræ, followed by gradual extension to the external muscles of both eyes, often sparing the accommodative mechanism. The character of the paralysis points strongly toward nuclear degeneration as a cause. Finally, the eyeball may become entirely immovable, with involvement of the orbicularis oculi. The disease is usually progressive and ultimately fatal. The usual lesion found postmortem is a chronic nuclear degeneration entirely analogous to progressive bulbar palsy and progressive muscular atrophy.

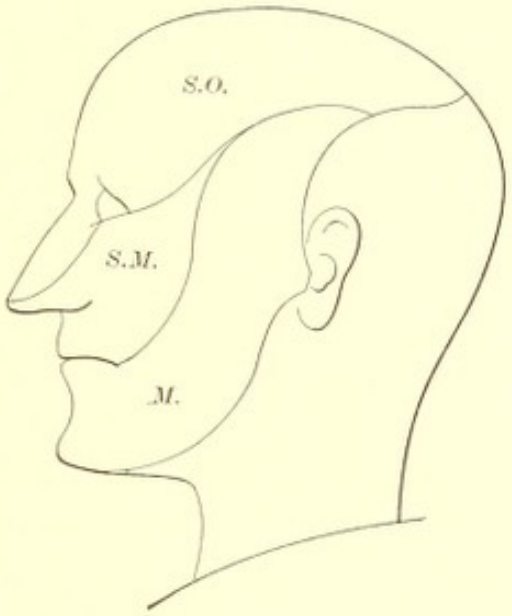
## TRIGEMINAL (FIFTH) NERVE.

**Anatomical Relations.**—The fifth, or trigeminal, nerve is made up of both sensory and motor portions, the sensory elements preponderating. Originating from cells in the Gasserian ganglion, the sensory fibers pass inward toward the pons, as a compact bundle, terminating, in part, about cells lying in the dorsomesial portion of the pons and in part extending downward as the descending root of the nerve as far as the upper portion of the spinal cord, effecting connections throughout this long course with other neurones which ultimately pass upward to the cerebral cortex. The cortical localization of the fifth nerve has not been definitely determined. The peripheral fibers leave the Gasserian ganglion as three nerves, the superior or ophthalmic branch passing through the sphenoidal fissure into

[1] E. W. Taylor, *Boston Med. and Surg. Jour.*, 1903, cxlviii, 634.

the orbit, the middle or superior maxillary division passing through the foramen rotundum, and the third or inferior maxillary (mandibular) division passing downward through the foramen ovale. These branches emerge respectively above the eye, beneath the eye, and on the chin, supplying areas of skin as represented in the accompanying diagram. The motor portion of the nerve originates from cells lying in the pons in close relation to the sensory nucleus, thence passes outward, joining the third division of the nerve peripheral to the Gasserian ganglion, thereby constituting it a mixed nerve trunk.

FIG. 8




Cutaneous distribution of fifth nerve. *S.O.*, supra-orbital division; *S.M.*, superior maxillary division; *M.*, mandibular division.

**Ophthalmic Branch.** — The first or ophthalmic branch supplies the eyeball and lacrymal glands, the conjunctiva, except that of the lower lid, the skin of the forehead and scalp up to the vertex, the mesial part of the skin of the nose and the mucous membrane of the upper part of the nasal cavity. There are also afferent pupil dilating fibers derived from the cervical sympathetic.

**Superior Maxillary Branch.** — The second, or superior maxillary division, is connected with Meckel's ganglion in the sphenomaxillary fossa which gives off the vidian nerve, important in relation to the sense of taste. The ultimate supply of the nerve is to the skin of the upper lip, side of the nose and adjacent part of the cheek, the lower eyelid, and part of the temple, also to the conjunctiva of the lower lid, the upper teeth, mucous membrane of the upper lip, upper jaw, uvula, tonsil, nasopharynx, the middle ear, and lower part of the nasal cavity.

**Inferior Maxillary Division.**—The third, or inferior maxillary division, so far as it is sensory, supplies the skin of the posterior side of the temple. adjacent part of the pinna, anterior and upper wall of the external auditory meatus, including the anterior part of the drum, a part of the cheek, lower lip and chin, the lower teeth, gums, and tongue as far back as the circumvallate papillæ, the floor of the mouth, the inner surface of the cheek, and salivary glands. The *motor portion* of the third division supplies the muscles of mastication, the masseters, temporals, both pterygoids, also the tensor tympani, the mylohyoid, and the anterior belly of the digastric. One of the most important anatomical relations of the fifth nerve is with the chorda tympani, through which the special sense of taste in the anterior two-thirds of the tongue is subserved.

As Cushing[1] has pointed out, a valuable means of determining the exact distribution and function of the trigeminal nerve in all its branches is to be derived from clinical study, particularly through the frequent operations on the Gasserian ganglion or more peripheral portions of the nerve. Cushing's

[1] *Jour. Amer. Med. Assoc.*, 1905, xliv, 773.

studies are of the greatest value, and have determined with much accuracy the fields of anæsthesia in the entire distribution of the nerve.

**Special Pathology.**—Owing to the long course and complicated ramifications of the fifth nerve, together with the fact that from origin to terminations it passes through several bony canals, it is natural that it should be damaged in various injuries or diseases of the brain and skull. It is, however, shown from Thomas'[1] statistics that the nerve is infrequently involved in fracture of the skull. In a series of 69 cases he found it affected but once, whereas the facial nerve, either alone or in combination with other nerves, was involved in 44 cases. In disease of the pons, occasioned by hemorrhage, softening, tumor, multiple sclerosis, or abscess, the fifth nerve is naturally involved. Extensive affection of the base of the brain is likewise often a cause of disturbance in the distribution of the fifth nerve. In the periphery a true primary neuritis of this nerve is exceedingly rare, and a neuritis secondary to disease in the neighborhood is also infrequent. Involvement of the nerve is naturally frequent in traumatism of various sorts, but it is rarely damaged in ordinary skull fracture. The pathological anatomy underlying the frequent neuralgia of the nerve is as yet wholly indefinite. Its association with arteriosclerosis, particularly in the neighborhood of the Gasserian ganglia, no doubt has some significance, but certainly in many cases is an insufficient and incomplete explanation of the pain. The studies of Spiller, Barker, Rusk, and Cushing have shown certain alterations in removed Gasserian ganglia, but not sufficiently characteristic to permit of their definite association with neuralgia. The situation regarding the pathological anatomy of a somewhat frequent herpes occurring in the branches of this nerve is more definite. There seems little question that an involvement of the ganglion is the direct cause of the eruption in this as in other sensory nerves, and that the somewhat fanciful term posterior poliomyelitis is not wholly misplaced. Barker's and Cushing's experiments, with the Nissl method, showed that distinct groups of cells may be involved leading to localized herpes in the same sense that groups of muscles are involved in poliomyelitis of the motor type.

**Symptoms.**—By all means the most important affection of the fifth nerve from the practical standpoint is the so-called tic douloureux, or neuralgia in other forms. In the general symptomatology of the affections of the nerve primary symptoms of irritation, manifested by pain, should be recognized. A more serious involvement of the nerve leads to anæsthesia in parts supplied, including certain mucous membranes. In such an anæsthesia of one nerve, for example, a cup from which one is drinking feels as if broken. Food collects within the anæsthetic cheek in spite of the fact that the motor power may be unimpaired. Taste is impaired over the anterior two-thirds of the tongue and somewhat at the back of the tongue, but does not remain wholly lost. The sensory loss to the facial muscles causes a peculiar awkwardness, due to an abolition, partial or complete, of the muscle sense. This disturbance not infrequently, particularly after neurectomies, may closely simulate a Bell's palsy. Cushing quotes such an instance in one of his cases of removal of the Gasserian ganglion. Such a condition is temporary and is easily determined by the persistence of normal electrical reaction. In considerable injury of the nerve the secretion of tears, of nasal

[1] *Jour. Amer. Med. Assoc.*, 1908, li, 271.

mucus and of saliva is decreased, resulting in dryness with possible secondary trophic changes. Under these conditions the sense of smell may be lost finally through such changes in the Schneiderian membrane. The corneal, lacrymal, and palatal reflexes are lost, and the tongue lies on the paralyzed side. The teeth are anæsthetic and tend to drop out. The condition of neuroparalytic ophthalmia, after operation on the fifth nerve, has been regarded as a trophic change. This Cushing is inclined to doubt, since he has found that a partial neurectomy is more often a cause of this ophthalmia than a complete one, and that if the anæsthetic eye be properly protected, such an ophthalmia in his experience does not occur. He attaches, therefore, particular importance to external sources of irritation.

FIG. 9

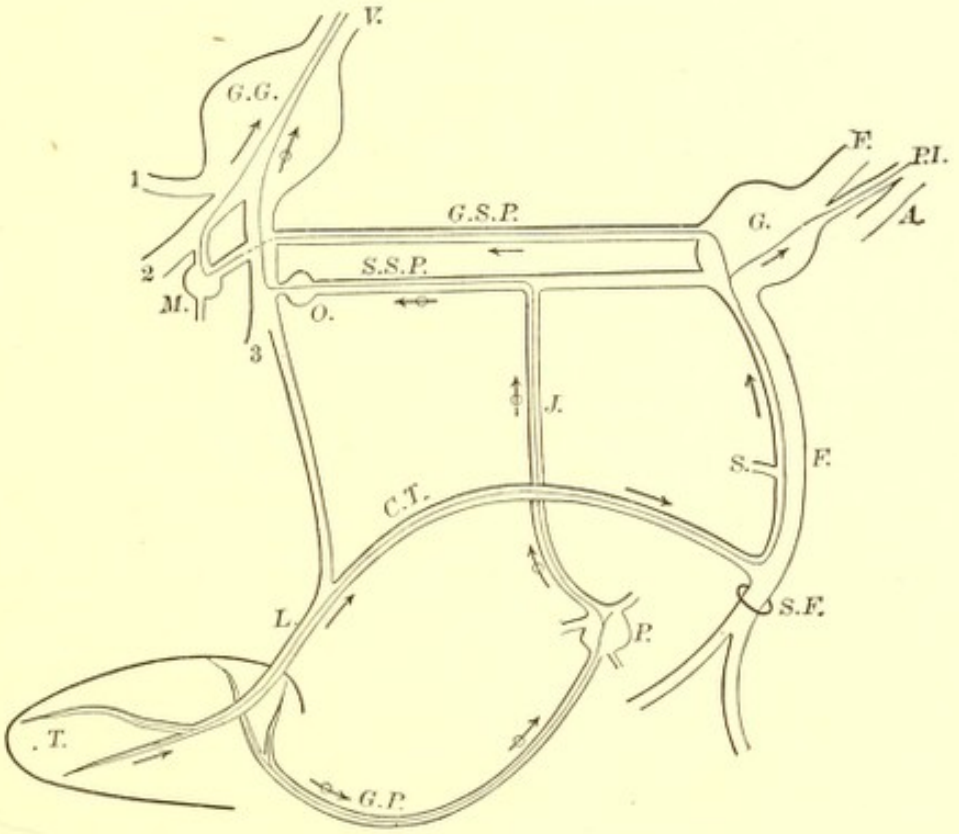



Diagram of course of taste fibers. *V.*, fifth nerve; *G.G.*, gasserian ganglion; 1, 2, 3, branches of fifth nerve; *M.*, Meckel's ganglion; *O.*, otic ganglion; *G.S.P.*, great superficial petrosal nerve; *S.S.P.*, small superficial petrosal nerve; *F.*, facial nerve; *P.I.*, pars intermedia; *A.*, auditory nerve; *J.*, Jacobson's nerve; *S.*, nerve to the stapedius muscle; *S.F.*, stylomastoid foramen; *C.T.*, chorda tympani nerve; *L.*, lingual nerve; *P.*, petrous ganglion; *G.P.*, glossopharyngeal nerve; *T.*, tongue. Course of taste fibers from anterior two-thirds of tongue represented by plain arrows, through chorda tympani, facial, great superficial petrosal, and second division of the fifth, with a possible passage through the pars intermedia. Course of taste fibers from posterior third of tongue represented by arrows with circles, through glossopharyngeal, Jacobson's nerve, small superficial petrosal, and third division of the fifth.

**Taste.**—A wide difference of opinion still exists regarding the course of the taste fibers and the part played by the fifth nerve in the transmission of gustatory sensations to the brain. There is general agreement that the glossopharyngeal nerve supplies the posterior portion of the tongue, and that the chorda tympani presumably subserves the same function for the anterior

two-thirds. A difference of opinion arises as to the connection between the chorda tympani and the brain, whether it passes by way of the trigeminus or by the facial or glossopharyngeal route. The theory that the fifth nerve is the course over which the taste fibers pass has had a large following. According to this theory the course of the fibers is through the lingual, the chorda tympani, and the facial to the geniculate ganglion, thence by the great superficial petrosal and sphenopalatine ganglion to the second division of the fifth nerve, and so to the Gasserian ganglion and the brain, or possibly through the small superficial petrosal and otic ganglion to the third division of the fifth nerve. Krause and Cushing, both surgeons of wide experience in operations on the fifth nerve, arrive at somewhat different conclusions from their neurological studies of operative cases. This no doubt is due to a number of causes, among which, as Cushing points out, the failure to make adequate preliminary tests for taste, uncertainty regarding total extirpation of the ganglion, faulty methods of making the tests, or, finally, a failure to take into consideration the loss of common sensation in the part of the tongue under examination, are no doubt important. In thirteen cases which Cushing reported in 1903, he found that in only one was there a failure to reëstablish taste perception, and this was found to be due to the fact that the chorda tympani had been divided at a previous operation and had not reunited. From this evidence it appears somewhat conclusive that the taste fibers do not, or do not exclusively, enter the skull along with the trigeminal nerve, as has been commonly supposed. The other alternative is that the fibers of the chorda tympani, after passing through the geniculate ganglion, reach the brain by way of the pars intermedia along with the facial nerve, constituting its sensory portion. The question, however, is still further complicated by the fact that lesions of the facial nerve involving the pars intermedia between the geniculate ganglion and the pons usually at least do not lead to affections of the sense of taste. The evidence, therefore, remains conflicting. The whole subject has recently been critically discussed by Davies.[1] The posterior third of the tongue and palate is presumably supplied in great measure through the glossopharyngeal nerve. Even from this part of the tongue, however, some of the fibers apparently pass to the brain by way of the fifth nerve, since disease of the Gasserian ganglion has caused impairment of taste, both in the anterior and posterior part of the tongue. The route of these fibers, therefore, has been presumed to be from the glossopharyngeal nerve to the fifth, through the tympanic plexus (Jacobson's nerve) to the small superficial petrosal, and so through the otic ganglion to the third division of the fifth. Cassirer has reported a case of complete loss of taste from disease of the glossopharyngeal nerve. From this observation the possibility is suggested that the glossopharyngeal may be the special nerve of taste. It must, furthermore, be borne in mind that individual differences may exist and that the taste fibers may conceivably take different courses in different persons.

**Motor Portion.**—Paralysis or disturbance of the innervation of the motor portion of the nerve naturally occurs only in involvement of the third division. In extirpation of the Gasserian ganglion the motor division is necessarily destroyed, but without leading to serious consequences. Paralysis of the muscles of mastication takes place on the affected side—masseter, temporal,

[1] *Brain*, 1907, xxx, 219.

and pterygoids. Paralysis of the mylohoid and anterior belly of the digastric do not lead to significant disturbance of function. Chewing is in general not seriously interfered with, owing to the preserved action of the corresponding muscles of the other side. A simple test for weakness of the muscles of mastication is to place the fingers at about the articulation of the jaw and note the differing contractions of the two sides on the attempt to close the jaws. The reaction of degeneration naturally occurs in these muscles, but is rarely tested. Atrophy later supervenes. The lower jaw can only be moved toward the injured side, either on opening or on attempted lateral movement. There is also some interference with wide opening of the mouth (digastric, mylohyoid). The paralysis which occurs in violent trigeminal pain is naturally merely apparent and due to pain inhibition. Paralysis from central lesion is rare, and the definite location of a cortical centre is as yet uncertain.

**Trismus.**—Tonic and clonic spasm of the jaw muscles, known as trismus, is rather a symptom than a specific affection. In the tonic variety the jaws are closely approximated. This occurs as a symptom of tetanus, in meningitis, seldom in tetany, as a temporary symptom in the epileptic seizure, also in certain diseases of the pons, and possibly as a result of irritation of cortical centres. It is also not very uncommon in hysteria, and may occur as a reflex from carious teeth or periostitis of the jaw. The clonic form of trismus manifests itself in rhythmic movements of the jaw, usually in a vertical, less often in a horizontal direction. It occurs in general convulsions, hysteria, epilepsy, and paralysis agitans. Oppenheim reports a case of a girl who for many years suffered from clonic contractions of the jaw muscles on the basis of hysteria. Extensive involvement of the central motor tracts may likewise lead to a jaw clonus similar to that often obtained in the ankle. The teeth grinding, both of children and adults, during sleep is worthy of mention in this connection. The prognosis of trismus is, as a rule, good, provided the underlying source of the affection can be alleviated or removed.

## FACIAL (SEVENTH) NERVE.

**Anatomical Relations.**—The seventh, or facial, nerve is ordinarily regarded as purely motor in type, a conception which must presumably be changed, not only on morphological grounds, but also because of recent researches regarding the function of the pars intermedia and geniculate ganglion, to which reference will later be made. The main distribution of the facial nerve is to the muscles of expression. Its origin in the cortex is definitely determined as occupying the inferior portion of the general motor area, ventral to the fissure of Rolando, whence the axones pass into the pons accompanying the general motor tract and crossing shortly before their distribution about the cells of the secondary nucleus in the pons. There has been much discussion regarding the superficial origin of the various branches of the facial nerve, particularly those which supply the muscles of the upper face, owing, on the one hand, to the relative freedom of the upper facial muscles in certain apparent affections of the nerve, and, on the other, to the frequent involvement of the lips in the absence of other

alterations in the supply of the facial nerve. The facts have led to the supposition that the nucleus of the upper facial branches was connected with the third nerve nucleus (Mendel) and the innervation of the lips with the upper part of the hypoglossal nucleus, of which Gowers supposes it to form a constituent part. On the basis of a very carefully studied case, both clinically and microscopically, Bruce and Pirie[1] have recently reached the following somewhat definite conclusions: That the upper facial nerve does not arise from the oculomotor nucleus; that the lip facial fibers do not arise from the hypoglossal nucleus; that the facial nerve has not crossed origin from the main nucleus; that all the fibers of the nerve arise from the recognized nucleus in the pons; and that details of the localization of function within the main nucleus are not as yet clearly established.

The peripheral nerve in its passage from the nucleus forms a loop about the nucleus of the sixth nerve, thence passes ventrally to emerge in close relation with the auditory nerve, separated from it by the pars intermedia (nerve of Wrisberg). These nerves together pass into the internal auditory meatus. The pars intermedia has no relation with the eighth nerve, but doubtless originates from the cells of the geniculate ganglion lying on the seventh nerve, and joins the seventh in the aqueduct of Fallopius. Centrally it is connected with the upper part of the glossopharyngeal nucleus, and may properly be regarded as forming the sensory portion of the seventh nerve and the main part of the chorda tympani. The seventh nerve, peripheral to the geniculate ganglion, passes into the Fallopian canal, runs between the cochlea and vestibule on the inner wall of the tympanum, separated from the tympanum by a thin lamella of bone and the lining membrane of the middle ear, finally emerging from the skull through the stylomastoid foramen. The geniculate ganglion lying at the bend of the nerve corresponds to a ganglion of a dorsal root. The chorda tympani, presumably a continuation of the pars intermedia, leaves the facial nerve before its exit from the skull. In addition to the supply of the facial muscles of expression, the facial nerve sends fibers to the platysma, posterior belly of the digastric, the stylohyoid, and a small twig within the Fallopian canal to the stapedius muscle of the ear.

**Facial Paralysis.—Etiology.**—A paralysis of the seventh nerve is more frequent than that of any other nerve in the body. In the large proportion of cases it is unilateral, and the lesion producing it is usually in its peripheral distribution. Among the apparent causes of the ordinary type of peripheral facial paralysis exposure takes a prominent place. It has been estimated that 70 per cent. of all cases show this etiological factor. Unsatisfactory as this explanation of the onset of paralysis may be, it must, in general, stand until a more definite and precise etiology is found. The significance of exposure in the absence of other underlying and unknown predisposition may with justice be questioned, but the occurrence of facial paralysis, after definite exposures to drafts or similar conditions, is too frequent to be regarded merely as a coincidence, but must be considered at least as an exciting cause. Other suggestions have been made from time to time to which due weight should be given. The theories of an infectious process, a neuritis, or a primary degeneration of the nerve within the Fallopian canal, all have advocates. In this connection a recent pathological report by André

[1] *Rev. of Neurol. and Psychiatry*, 1908, vi, 685.

Thomas,[1] in which he was able to study three cases, is of interest. The cases were all of peripheral paralysis of the seventh nerve, two of them showing hemispasm. In each of these cases an examination of the nerve and of the nucleus was made. In the first case death resulted in eighteen days and the nerve showed a parenchymatous degeneration downward in the aqueduct of Fallopius from the first bend, with almost entire loss of the axones. Above the geniculate ganglion the axones were less altered. The cells of the facial nucleus were swollen and showed chromatolysis and eccentric nuclei, whereas the cells of the opposite nucleus were normal. In the second case, following an otitis media, there was total left facial paralysis, with complete reaction of degeneration and occasional muscular spasm. Examination of this nerve showed chronic perineuritis below the first bend of the aqueduct of Fallopius. The nerve showed a neuroma of regeneration. The geniculate ganglion was somewhat atrophied, the facial nucleus normal. The value of this case is naturally impaired by the preëxisting otitis. In the third case there was almost complete right facial paralysis with occasional spasm. Examination of the nerve showed signs of regeneration. There was swelling of the nerve at the first bend of the aqueduct of Fallopius. The nucleus was normal. It is to be remarked that in all of these cases the initial lesion in the aqueduct of Fallopius appeared at the first bend of the facial nerve.

The attempt has been made to hold a neuropathic tendency responsible for facial paralysis, but this the statistics in general do not bear out. There are, however, various instances in which cases have occurred in one family, and others in which recurrences have taken place. An emotional shock has likewise been held responsible in occasional cases. Gowers, for example, reports the case of a woman who underwent a facial paralysis immediately after watching the dressing of a breast cancer, and Waterman has made somewhat similar observations. Constitutional diseases, such as gout, diabetes, leukæmia, and syphilis, have all been held responsible. It has also occurred during the puerperal state and following diphtheria. It is rare as an accompaniment of a general polyneuritis, and if it occurs in this or in allied conditions, paralysis is apt to be double-sided, as the writer has observed in several cases. A facial paralysis may also, no doubt through a nuclear lesion, occasionally occur in poliomyelitis. One such case was observed in a child during a recent epidemic, in which the only other involvement was a slight paralysis of the anterior muscles of one leg. We are on much more definite etiological ground, however, in considering the relation of trauma. Owing to its long course through the Fallopian canal, and its close proximity to a cavity which often suppurates, paralysis of a facial nerve from carious and suppurative ear disease is extremely common. Basal fractures likewise show in a large proportion of cases a facial paralysis as one of the signs. We have already quoted Thomas' statement that of 69 cases of nerve involvement in skull fracture 44 showed an affection of the facial nerve.

Naturally disease of the base of the brain, meningitis, new-growths, and affections of the medulla and pons, may readily lead to involvement

[1] *Rev. Neurolog.*, 1907, xv, 1273. See also A General Discussion and Bibliography on Facial Paralysis, A. Fuchs, *Arb. a. d. Neurolog. Inst. a. d. Wien. Univ.*, Band xvi, Part 2, 245.

of one or both facial nerves. The occasional palsy of a branch of the facial nerve supplying the orbicularis oris in cases of bulbar palsy, presumably through the degeneration of fibers passing out with the twelfth nerve, is of interest. The observation which Cushing has recently made, that certain of the cases of supposed paralysis of the seventh nerve, following extirpation of the Gasserian ganglion, are due rather to loss of muscle sense in the fifth nerve than to an actual paralysis of the seventh, is likewise worthy of mention. In a recent critical analysis of 335 cases of facial paralysis, exclusive of those due to ear disease or trauma, G. A. Waterman[1] arrives at the following significant conclusions relative to the etiology and course. He finds that sex plays no significant part in the incidence; that hereditary influence is doubtful; that exposure to cold is the most definite etiological factor as yet determined; that the affection is no more frequent in winter than in summer; that it may occur at any period of life, but is much more frequent before forty and during the third and fourth decades. Thirty-six per cent. of all these cases occurred between twenty and thirty. The disease is more severe, but less frequent in later life. Preliminary pain is not prognostic, and secondary contractures are not affected by galvanic treatment, as has at times been maintained.

**Onset and Symptoms.**—The onset of an ordinary peripheral facial paralysis is usually rapid and at times sudden. Naturally the signs are produced more slowly if the paralysis is due to a gradually increasing disease of the middle ear, or to a progressive tumor growth. Pain is a not unusual prodromal symptom, located in the ear, behind the ear, in the mastoid region, or in the neck, but is of small diagnostic or prognostic significance, according to Waterman's statistics. The explanation of this preliminary pain is not easy. It may be due either to coincident involvement of sensory branches or possibly to a neuritis which may be regarded as an accompaniment or cause of facial paralysis. At times the facial paralysis is ushered in with fever and signs of infection, especially in children. In the absence of a slowly progressive cause, a gradual onset of the paralysis is extremely unusual. The signs of facial paralysis are naturally conspicuous. The most striking is a loss of movement, often complete, of the side of the face affected, more marked in elderly persons than in young, no doubt due to the presence of wrinkles in advancing years, which when obliterated by paralysis increase the contrast between the normal and affected side. The eyelid remains open, to a certain extent even in sleep, owing to the paralysis of the orbicularis oculi. In an attempt to close the eye the globe is rolled upward, showing the sclerotic and often leading to the false idea on the part of the patient that the eye is completely closed. The partial closure of the eye often observed in the presence of very complete paralysis of other branches of the nerve is due to relaxation of the levator rather than to any active contraction of the orbicularis. In giving expression to the emotions, as in smiling, the mouth is drawn toward the unaffected side through the unopposed action of the zygomatici. The tongue appears to be protruded to one side, due to the fact that the position of the mouth is altered by the paralysis. The tongue is in reality not affected. The lips cannot be properly apposed, so that whistling is not possible. Drinking is likewise interfered with, and speech may suffer from the impossibility of properly making

[1] *Jour. Nerv. and Ment. Dis.*, 1909, xxxvi, 65.

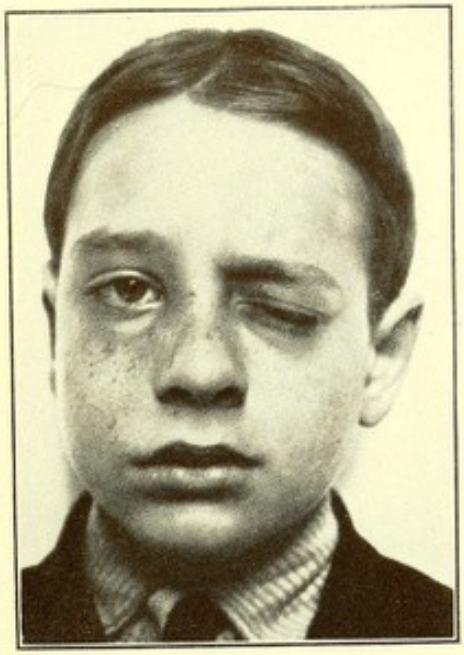
# PLATE XXIX

FIG. 1

Complete Left Peripheral Facial Paralysis.

Following operation for sarcoma of the mastoid region, with unavoidable destruction of the seventh nerve. Impairment of taste, anterior portion of the tongue on the left. Face at rest, showing slight deformity. (Massachusetts Charitable Eye and Ear Infirmary and Massachusetts General Hospital.)

FIG. 2



Same. Attempt to Close Eyes.

Left side of face immovable.

FIG. 3

Same. Attempt to Show Teeth.

Paralysis of zygomatici and other muscles concerned in raising the lips.

use of the labial sounds. From the paralysis of the buccinator, food often remains between the teeth and in the cheek until mechanically removed. The muscles of the external ear are paralyzed, as well as the platysma, so that the lower lip cannot be drawn down. The palate is not affected.

In considering the point at which the nerve is involved, a somewhat artificial distinction may be made regarding five positions (Fig. 10):

FIG. 10

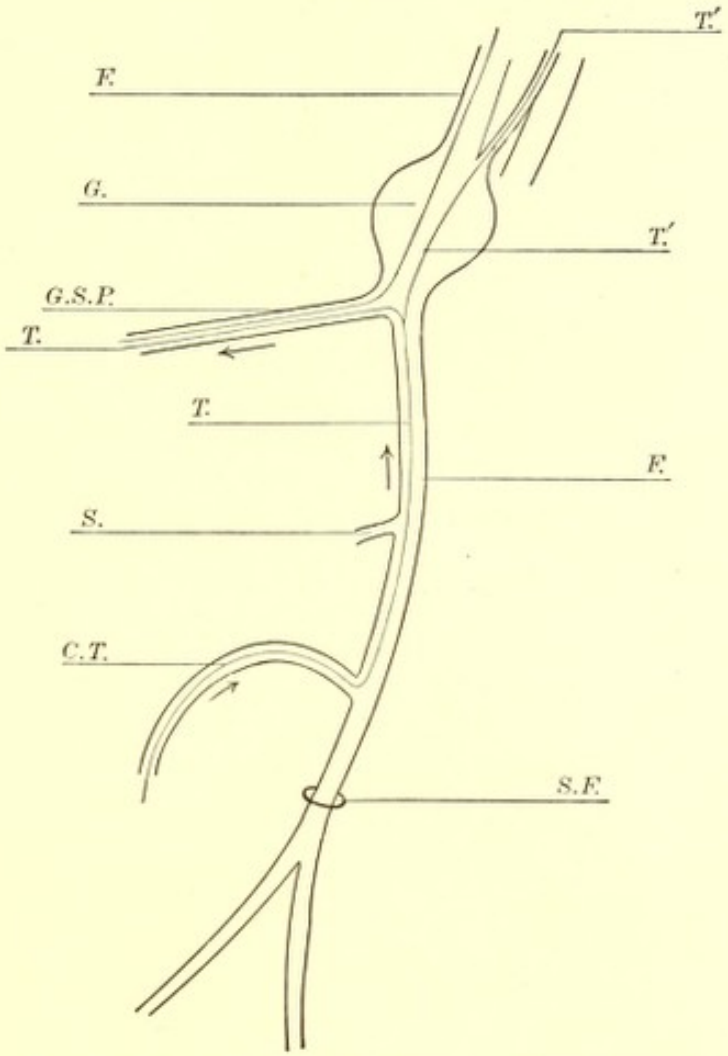



Diagram of facial nerve. *F.*, facial root; *G.*, geniculate ganglion; *G.S.P.*, great superficial petrosal nerve; *S.*, nerve to the stapedius muscle; *C.T.*, chorda tympani nerve; *S.F.*, styloid-mastoid foramen; *T. T.*, taste fibers; *T'. T'.*, possible course of taste fibers through the pars intermedia. The arrows represent the probable course of these fibers. Lesions of the nerve from the geniculate ganglion to the point at which the chorda tympani is given off lead to disorder of taste, with probable impairment of hearing (stapedius).

1. If the lesion lies below the exit of the nerve from the stylomastoid foramen, taste will not be affected, inasmuch as the chorda tympani joins the nerve above this point.

2. If the lesion be between the stylomastoid foramen and the geniculate ganglion within the Fallopian canal, taste in the anterior two-thirds of the tongue is affected through coincident involvement of the chorda tympani, which accompanies the facial nerve between these points. In addition there is also occasional slight loss of general sensibility, with possible sub-

jective taste sensations and changes in the secretion of saliva. The tongue may be furred in consequence of the altered salivary secretions. If the nerve to the stapedius muscle given off from the trunk of the facial nerve, between the geniculate ganglion and its exit from the skull, be involved, hypersensitiveness to musical notes of low pitch has been observed.

3. If the nerve is affected between the geniculate ganglion and the pons, there is in general the same symptomatology as in the ordinary peripheral type. There is no involvement of taste, which has been used as a somewhat forcible argument against the theory that the pars intermedia carries the taste fibers. Deafness is often associated with lesion of the facial nerve at this point from the fact that the auditory nerve accompanies it. A deficiency of tear secretion on the affected side has been described in many cases, and possibly an involvement of the stapedius.

4. If the lesion is in the pons itself, taste and hearing are unaffected, and there are also almost invariably lesions of other nerves, notably of the sixth, accompanying the facial paralysis.

5. If the central course of the facial nerve be involved, the symptomatology differs from all those previously considered, inasmuch as no changes in electrical reactions occur and the upper branch of the nerve is very much less involved than the two lower.

The facial nerve is a peculiarly easy one to examine with the electrical current, and the information gained from this source is of the greatest possible value in arriving at a diagnosis. The presence or absence of electrical changes may be the determining factor as between a lesion peripheral to the pons or above the pontine nuclei in the central course of the nerve. Altered electrical reactions naturally occur only in lesions of the peripheral neurones. In slight degrees of paralysis the electrical reactions may be unaffected. In severe cases, on the other hand, a complete reaction of degeneration may be established in two weeks. As between these two extremes forms of partial reactive degeneration may occur, and upon the information thus given depends very largely an estimate of the duration of the attack.

**Diagnosis.**—From the foregoing it will be seen that the diagnosis of the approximate position of the lesion is usually easy. It is especially important often to distinguish sharply between a central disturbance in the nerve and one due to a peripheral cause. The essential points in making this differential diagnosis are as follows: In the central lesion there is no change in electrical reactions; the upper branches of the facial nerve are slightly involved, either due to the fact that there are special cortical centres for different portions of the nerve, or, more probably, because muscles which ordinarily act together are presumably innervated from both sides of the brain. The facial paralysis is usually accompanied by a homolateral hemiplegia. A crossed or alternating hemiplegia indicates a lesion in the pons below the crossing of the central facial fibers and above the crossing of the pyramid. The reflex conditions of the nerve are theoretically increased, but for practical purposes may be regarded as unchanged. In contrast to this series of conditions, lesions of the peripheral nerve, irrespective of special location, are, in general, as follows: Electrical alterations are always present, except in the mildest cases. All branches of the nerve are equally involved, and reflex activity is diminished, with ultimate muscular atrophy, if recovery does not take place. A modification of the foregoing

# PLATE XXX

FIG. 1

Left Facial Paralysis of Long Standing.

Showing inability to close eye and laxity of muscles, especially evident about the mouth. (Massachusetts General Hospital.)

FIG. 2

Spasm of Left Orbicularis Oculi.

Photograph shows an attempt to open the eye, which tends merely to increase the spasm; girl, aged twelve years. (Putnam and Waterman. Studies in Neurological Diagnosis. Massachusetts General Hospital.)

general statement must naturally be made in those instances where through trauma a special branch of the nerve has suffered.

**Course and Prognosis.**—The outcome, as well as the course, varies within wide limits. Slight cases may last not more than one or two weeks, and give very little physical evidence of their presence, either by inspection or on physical examination. Other cases may continue for weeks, months, or even become permanent. As already suggested, the electrical conditions are the most important determining factor in relation to prognosis. A complete reaction of degeneration is by no means inconsistent with final recovery, but a reaction of degeneration persisting for months, and finally giving place to a quantitative diminution of electrical excitability, must always be regarded as a grave prognostic sign, indicative of permanent changes in the nerve, usually no doubt due to a greater or less degree of destruction of the pontine nucleus. A somewhat artificial distinction may be made between a mild, more severe, and a severe form of paralysis, depending upon whether the electrical reactions are normal or very slightly affected, with recovery in two or three weeks, or, in the second case, whether a partial reaction of degeneration is present, with recovery in from four to eight weeks, and finally, in the severe form, whether the reaction of degeneration is complete, with a duration of paralysis from three to six months. Naturally there are certain cases which do not conform to this classification, as, for example, those in which the paralysis lasts many months, or those in which the electrical reactions do not correspond with the apparent degree of paralytic defect.

In the ordinary case, after a varying period of complete reaction of degeneration, the electrical alterations change for the better in that the reaction of degeneration is no longer complete. The cathodal contraction increases over the anodal, and the faradic irritability of nerve and muscle is gradually reëstablished. Along with this improvement in electrical reactions voluntary control of the muscles is gradually regained until the face is restored to an essentially normal condition.

*Secondary Contractures.*—In giving a prognosis in peripheral facial paralysis the possibility of secondary contractures should always be borne in mind. Even in the cases of moderate severity a certain amount of defect may persist in the movements of the face on the affected side and in the tendency of the muscles to overact. Such secondary contractures usually come on from four to six months after the onset, and not infrequently with the inexperienced lead to a false conclusion as to the affected side. Inasmuch as the tendency is toward overaction, the affected side of the face shows a somewhat general contracture, the aperture between the lids is diminished, and on smiling or other emotional expression the affected side of the face is much more actively used than the unaffected. A further annoyance is the onset of spontaneous twitching of the facial muscles, often with isolated spasmodic contractions, especially of the zygomatici. This condition often persists for years, and, in fact, is not infrequently permanent, and may well prove to be a very much more disturbing condition than the original paralysis. Operation for its relief has been attempted with some degree of success. The cause of these secondary contractures has been a subject of much discussion, and is not yet definitely determined. A favorite theory has been that in any serious facial paralysis the nucleus of the pons is of necessity involved, the cells suffer and degenerate beyond

the point of restitution, thereby interfering permanently with adequate innervation. Nerve impulses are conceived as spreading from the damaged nucleus in an irregular and somewhat uncontrolled manner, so that an excessive degree of a normal phenomenon is produced in the contractured parts.

A lessened resistance between the cells of the nuclei has also been suggested. A plausible theory also is that which regards the contractures as due to permanent alterations in the muscles of such a character that the fibrillar substance is imperfectly reproduced, whereas the sarcoplasm maintains the muscle in a state of exaggerated tonus.

In other than the ordinary form of facial paralysis, which has been especially considered, prognosis depends essentially upon the underlying cause, and is usually a relatively insignificant matter in view of the more serious condition of which it is a sign. In tumor, for example, and similar conditions, except for its localizing and, therefore, diagnostic value, a lesion of the seventh nerve naturally depends for its outcome upon the course of the growth which has produced it. Into this field the limits of this article do not permit us to enter. Certain cases have been described as recurrent, not unlike periodic ocular motor palsy. Several cases have also been described as occurring in one family, suggesting some hereditary predisposition. Both of these circumstances are, however, too infrequent to alter the general statement that peripheral facial palsy of the so-called rheumatic type is a sporadic and benign affection of good prognosis.

**Treatment.**—This consists in various measures of assisting nature in what may be regarded as a natural process of cure. Not too much, therefore, should be expected of any definite course of medical treatment, although as an adjuvant such treatment is unquestionably often useful and at times demanded. In view of the fact that a neuritis within the Fallopian canal may be regarded as a pathological basis of the affection, fomentations and blisters may be applied in the mastoid region to serve as counter-irritants. The bowels should be kept free, the salicylates may at times be administered with advantage, and, if there is reason to regard syphilis as an etiological factor, the iodides and mercury are indicated. The chief reliance, however, in the treatment of this paralysis has been the use of electricity, and in spite of its recognized limitations, this form of treatment remains in popular favor. It is doubtful whether the electrical current has any specific effect upon the nerve, but it cannot be questioned that the stimulation of muscles incapable of movement through an act of the will is a reasonable and logical procedure. Oppenheim believes that in early, and often in older, cases striking results may be obtained by the use of electricity. In treatment by this means, presuming that a reaction of degeneration is present, the galvanic current should be employed, using over the affected muscles the pole which produces the more marked contraction. Not more than 4 to 5 milliamperes should be used, and the muscles should be stroked by an electrode for a period not exceeding ten minutes. As the faradic irritability returns, this form of electricity may be substituted for the constant current, but it is well to discontinue any form of electrical treatment when the tendency to contracture and spasm of the muscles becomes apparent. There is no adequate evidence to show that the application of electricity plays any part in the production of secondary contracture, but the treatment becomes more and more superfluous as

voluntary control over the muscles increases, and may well be discontinued before such voluntary control becomes complete.

As in many other fields of treatment, surgery has invaded this domain, and there is very considerable hope, with reason, that with surgical intervention intractable cases of facial paralysis may be very greatly benefited, if not completely cured. The first method of radical surgical procedure suggested by Ballance in 1895 was the anastomosis of the facial with the spinal accessory. In this operation a part or a whole of the spinal accessory was sutured into the divided facial, or an implantation of the facial nerve made into the split spinal accessory, the object being to facilitate the growth downward of spinal accessory fibers in the path of the functionless facial. Following Ballance, cases of this operation were reported by Faure, Kennedy, Cushing,[1] and Hackenbruch. The immediate result was a more or less permanent paralysis of the trapezius and sternomastoid muscles. The facial condition improved, but any sudden voluntary movement of the face was accompanied by a distressing involuntary movement of the shoulder and vice versa, which proved to be a decided disadvantage. To obviate this the hypoglossal nerve was selected for the anastomosis. Inasmuch as the hypoglossal nerve is closely associated physiologically with the facial, and since the cortical localization of the two nerves is near together, it seemed more rational to use the hypoglossal than the spinal accessory and a considerable number of such operations have been done.

The importance of operating as soon as possible after injury of the facial nerve can hardly be too strongly insisted upon, inasmuch as the progressive disintegration of muscle may easily go on to a point where recovery is no longer possible, however completely the nerve may regenerate. In carrying out this operation it is desirable not to divide the hypoglossal completely, owing to the consequent difficulty of mastication, deglutition, and phonation. Indication for the operation is the assurance that the facial nerve is divided or so completely injured that spontaneous recovery is impossible. Its field, therefore, lies essentially in those cases in which the nerve has been divided through trauma, particularly resulting from accidental injuries in operating on the ear, and from destructive suppurative processes within the middle ear. In this connection a case observed by G. L. Tobey is of interest. A young girl had suffered a paralysis of the face through the accidental cutting of the facial nerve within the Fallopian canal. The paralysis was complete, although certain movements of the orbicularis oculi and oris were possible, due no doubt to innervation from other nerves. The question of facial-hypoglossal anastomosis was seriously considered, but for the time being dismissed. Later, a suppuration of the ear occurred, which demanded a second mastoid operation. This was done by Dr. Tobey. In the course of this operation he found the divided ends of the facial nerve somewhat retracted within the Fallopian canal. He was able to draw these ends together and suture them, apparently with temporary favorable results regarding facial movements. A later report from him, however, and a subsequent personal examination of the patient show that the improvement was apparent rather than real. The interest of the case lies in the cleverness of the operation, in the demonstration of the possibility of suturing a divided facial nerve in the Fallopian canal, and in the conceiv-

[1] *Annals of Surgery*, 1903, xxxvii, 641, with historical and bibliographical notes.

able practical utility of this operation in place of a facial-hypoglossal anastomosis, in those cases in which the source of the injury to the nerve is manifestly within the middle ear.

**Facial Spasm and Tic Convulsif.**—Irrespective of the postparalytic contracture and spasm, of which mention has already been made, the facial nerve is peculiarly prone to spasmodic affections, due, no doubt, in part, to its complex reflex relations. There is, in the first place, an exceedingly close relation between the mental condition and the muscles of expression, and in the second place the reflexes which subserve this relation are naturally highly complex. A localized irritation of the nerve may lead to spasm, which later, when the cause has been removed, may persist as a fixed habit. The direct irritation of the nerve stem, as by a tumor, or the occasional irritation from a localized brain lesion, may produce more or less persistent facial spasm. In most of the cases no definite anatomical basis is demonstrable. Brissaud and especially Meige have distinguished between what they regard as true spasm and one of psychogenic origin. Of the latter, are those aroused emotionally, which later have become habitual. Such are various reflex spasms, the so-called *tic convulsif*. As distinguished from this type the true facial spasm is not dependent upon a mental state, but is due rather to material changes in the reflex arc either in the sensory trigeminus, facial nucleus, or the facial stem. The first type bears close relation to the psychic life, and may be externally influenced through diversion of the attention, and by similar means. The second type of spasm usually occurs in the whole distribution or in definite portions of the nerve, and is usually one-sided, and presumably is not influenced by suggestive measures. This type of spasm is that artificially induced by an electric current. A true cramp of the facial nerve may likewise occur, either tonic in character or alternating.

Patrick[1] has also recently made clear the fundamental difference between true facial spasm and tic. He states the distinction as follows: "Spasm may be said to be an anatomical, tic a physiological, disorder. A good picture of facial spasm may be obtained by faradization of the facial nerve. Tic is always the replica of a perfectly natural (though may be unusual) and physiological movement. Looking at the distortion caused by faradization of the nerve no one would mistake it for voluntary contraction of the facial muscles. On the other hand, having seen only one individual contraction of facial tic, no one could say that the movement was not a perfectly normal and natural one—possibly unusual, possibly overdone, but the legitimate result of a casual cause. Voluntary imitation of facial spasm is practically impossible. The patient himself can always repeat or imitate his tic movements, and another person can nearly always do so." In so-called blepharospasm, one of the commonest of the spasmodic affections, the subjective disturbance may be extremely slight or wholly lacking, especially in its milder forms. A case comes to mind of a young boy who had developed such a spasm as a habit of what was naturally at first purely reflex through looking persistently at the sun. This patient was almost wholly relieved by simple exercises directed to retraining of the lid muscles.

In so-called tic convulsif there are often signs of other nerve lesions and

[1] *Jour. Nerv. and Ment. Dis.*, 1909, xxxvi, 1.

even of a definite underlying hysteria. The course of these affections is chronic, the prognosis doubtful but never hopeless.

**Treatment.**—Treatment consists in the removal of any reflex cause. Drugs are, on the whole, useless, except as palliatives. Nerve stretching is possible, but should only be resorted to in very intractable cases. Injections of alcohol into the facial nerve near its exit from the skull have been used in intractable cases of facial spasm. Patrick reports three such cases, with excellent results in two and with a failure in one due to faulty technique. The treatment consists in the injection of a 40 to 75 per cent. alcohol solution into the trunk of the facial nerve. If the injection is successful, an immediate paralysis of the nerve results, naturally with a cessation of the spasm. Recovery takes place from the paralysis, and there is reason to believe that the spasm will not return even after a complete restoration of the normal function of the nerve. In this investigation Patrick acknowledges his indebtedness to Professors Brissaud and Raymond of Paris.

Occasionally a spasm of the platysma may develop. Such a case has recently come under personal observation in which the patient, a man, for two or three years has been annoyed by a distinct spasm of the right platysma when talking, without apparent involvement of other muscles supplied by the facial nerve.

**Herpetic Inflammations of the Geniculate Ganglia.**—Under this general heading J. Ramsay Hunt[1] has recently brought together a group of cases which he regards as constituting a hitherto undescribed clinical entity. He regards the facial as a mixed nerve, the nerve of Wrisberg being its sensory portion, with the geniculate as its ganglion of origin. It follows that herpes may occur in its sensory distribution in common with other ganglia of this type, as, for example, the Gasserian ganglion of the fifth nerve or the various spinal ganglia. Hunt points out that the zoster zone for the geniculate ganglion lies in the interior of the auricle and in the external auditory canal. On the clinical side the affection manifests itself in the characteristic way, with slight febrile prodromata, neuralgic pains in the neighborhood of the ear, followed on the third or fourth day by the typical herpetic vesicles, very rarely, according to Hunt, localized on the tympanic membrane. This area retains its sensation after extirpation of the Gasserian ganglion and the second and third cervical ganglia, from which it is assumed that it represents the cutaneous distribution of fibers of the geniculate ganglion.

Herpes zoster of the auricle has long been recognized, but had heretofore been regarded as belonging to the trigeminal area, and hence due to involvement of the Gasserian ganglion. Hunt's position seems well taken that this localized manifestation of herpes may be more properly explained by an involvement of the nerve of Wrisberg and its ganglia. The cases of herpes in and about the ear are uncommon. Nevertheless, in a very large collection of cases from special hospitals a certain small number has been reported for many tears. Hunt describes three clinical types: (1) Uncomplicated auricular herpes; (2) auricular herpes with facial palsy; and (3) auricular herpes with facial palsy and auditory symptoms. The

[1] *Jour. Nerv. and Ment. Dis.*, 1907, xxxiv, 73, and *Amer. Jour. Med. Sci.*, 1908, cxxxvi, 226.

latter group of cases is perhaps the most obscure and difficult of classification. On the basis of 60 personal observations and a somewhat limited pathological investigation, Hunt feels justified in establishing a characteristic syndrome divided into the three foregoing clinical groups. He also makes the statement that neuralgic affections of the geniculate ganglion and its divisions productive of an otalgia may properly be regarded as due to disorder in the sensory mechanism of the facial nerve, giving rise to earache of non-inflammatory origin. Anatomically, the geniculate ganglion stands in relation to the interior of the auricle and external auditory canal, to the tympanic plexus, to the second and third divisions of the fifth nerve, to the glossopharyngeal, and to the vagus. It is evident, therefore, that ear pain may be due to a variety of reflex causes. Hunt is convinced, however, that, although many of the neuralgic affections of the ear are no doubt dependent upon involvement of the trigeminal and occipitocervical nerves, there exists also an otalgia due entirely to involvement of the sensory branch of the facial nerve.

Orbison[1] takes issue with some of Hunt's conclusions and makes a still further division of zoster zones on the basis of a study of herpes of the tympanic membrane. He believes that herpes of this area is due not, as tacitly assumed by Hunt, to involvement of the zoster zone of the seventh nerve, but rather to zoster conditioned by disease of the petrosal ganglion of the glossopharyngeal.

## AUDITORY (EIGHTH) NERVE.

**Anatomical Relations.**—The auditory, or eighth, nerve, or, according to the older nomenclature, the portio mollis of the seventh pair, is properly regarded as two nerves subserving independent and special functions. Of these two nerves, the cochlear branch is concerned with the special sense of hearing, and the vestibular branch with the function of equilibration. The anatomical relations of the nerves, considered as a whole, are peculiarly intricate, owing to the highly developed special sense of hearing and the complexity of the neural relations underlying equilibration. The nerve has wide connections with the cerebellum, with other cranial nerves, particularly with those of sight, with the midbrain, and ultimately with the cerebral hemispheres. Like other sensory nerves, its fibers originate from ganglia lying external to the central nervous system. The cochlear division has origin from bipolar cells constituting the spiral ganglion of the cochlea. The peripheral fibers, which may be regarded as dendrites, are distributed about the auditory cells of the organ of Corti, and serve to transmit sound impulses from the internal ear. The central fibers of the cochlear division join the vestibular branch, pass through the internal auditory meatus with the facial nerve and pars intermedia of Wrisberg, and thence to the pons at its junction with the oblongata. The vestibular branch originates from cells of the vestibular ganglion (ganglion of Scarpa) at the bottom of the internal auditory meatus. The peripheral branches of the bipolar cells pass to the semicircular canals, and the central axones reach the pons in company with the facial and cochlear branches. Although this group of nerves lie in close proximity, they may be easily distinguished at

[1] *Jour. Nerv. and Ment. Dis.*, 1908, xxxv, 500.

their entrance to the pons. The details of the central connections of the auditory nerve taken as a whole do not concern the present article. The more important points may be summarized in the facts that the termination of the vestibular axones is chiefly in the auditory nucleus of the floor of the ventricle, whence connections are effected with the lateral columns of the cord, with the cerebellum, with the dorsal longitudinal fasciculus, and with the nuclei of the ocular nerves. The function of equilibration is subserved through these connections. The cochlear branch on entering the pons ends superficially in the dorsal (tuberculum acusticum) and the ventral nucleus lying in close proximity to the inferior peduncle of the cerebellum. From the dorsal nucleus the so-called striæ acusticæ pass around the restiform body, and finally reach and help to constitute the lateral fillet, ending about cells of the inferior corpus quadrigeminum. From the ventral nucleus secondary fibers pass through the superior olives by way of the trapezoid body, likewise to the lateral fillet, ending chiefly about cells of the inferior corpora quadrigemina and mesial geniculate bodies crossed and uncrossed. From these nuclei neurones ultimately reach the temporal lobe in its superior gyrus in close association with the so-called sensory speech area.

**The Cochlear Nerve.**—The function of hearing is almost undoubtedly subserved wholly by the cochlear division of the auditory nerve. Hearing may be affected through lesions anywhere in the course of the nerve, from the cortex through the various sensory nuclei to the labyrinth, but such disturbance is far from frequent in actual disease of the internal ear and its structures and in affections of the terminal branches of the nerve than in more centrally localized lesions. From this fact disturbances of hearing have come to be almost exclusively associated with that special branch of medicine which concerns itself with diseases of the ear.

1. The cortical centre for hearing is presumably localized in the superior convolution of the temporal lobe, and unquestionably stands in close relation with the cortical area for the perception of language. There is, however, still disagreement regarding its exact position. The reason that word deafness does not lead to sound deafness is that the auditory nerves naturally have relations with both hemispheres, whereas the more highly specialized function of the appreciation of language is confined wholly to one hemisphere. The observation has been made that when both superior temporal convolutions are diseased, sound deafness has also resulted. Inasmuch as this latter lesion is extremely unusual, it follows that deafness rarely results from cortical lesions.

2. Lesions of the auditory nerve from the cortex to its subsidiary nuclei may also lead, under certain circumstances, to deafness which, on the practical side, is indistinguishable from deafness due to labyrinthine disease. Lesions of the corpora quadrigemina and geniculate bodies, tumors of the substance of the pons, also certain tabetic conditions and multiple sclerosis, may lead to deafness in one or both ears through involvement of the auditory mechanism in its course through the brain stem. More practically important are lesions in the peripheral course of the nerve. Inflammatory processes, notably meningitis, are likely to involve the nerve with others at its exit from the pons, often spreading along the nerve to the ear itself. A primary neuritis of this nerve and subsequent atrophy, also the so-called rheumatic paralysis similar to that frequently observed in the facial nerve, are exceedingly rare conditions, if, in fact, they exist. A condition of very consider-

able importance, to which special attention has of late been drawn, is that of tumor growing in the cerebellopontile angle. Such tumors are not infrequently neurofibromatous in character, and may under certain circumstances be removed. They are most frequently localized on the eighth nerve, but may also involve the trigeminus and vagus. Tumors lying in this region naturally give rise to auditory symptoms, which may be of much value in determining location. Aneurism is at times a cause of deafness through pressure on the nerve.

3. In the great majority of cases, however, the lesion inducing the deafness or other disturbances in the function of the auditory nerve lies in the internal ear, either as a primary process or resulting from the extension of disease from the middle ear. Causes of such disturbances are hemorrhage, inflammations, sclerotic processes often on the basis of acute infectious disease, particularly mumps or syphilis, certain constitutional diseases, as, for example, nephritis, diabetes, and pernicious anæmia. A very common cause of labyrinthine disease is epidemic cerebrospinal meningitis, which not infrequently results in permanent deafness. Conditions of congenital deafness do not here concern us. Hysteria not infrequently shows, as one of its symptoms, a definite loss of hearing, the character of which may be determined by special tests and also particularly by associated symptoms.

**Symptoms.**—These may be classed in two groups—symptoms of irritation and symptoms of defect. Irritative symptoms or hyperesthetic conditions are unusual, but have been observed in certain abnormal functional states of the nervous system, notably in hysteria. In this condition sounds may be distinctly appreciated which are not ordinarily heard. Such hyperacuteness of hearing may also be observed at the onset of certain acute and general diseases, and undoubtedly in these instances may be attributed to a general hyperexcitability of the nervous system. In certain disorders of the conducting sound mechanism, as, for example, in paralysis of the stapedius muscle often occurring in conjunction with a general facial paralysis, low notes may be heard with unusual distinctness, due presumably to the relaxation of the stapedius muscle. The condition of so-called dysacusis, in which sounds ordinarily innocuous cause distinct discomfort, should also be mentioned. Under these conditions pain, as, for example, headache, is apparently actually increased by noise. Undoubtedly this affection, which is not an uncommon one in healthy persons, is due to a temporary general lowering of the nervous tone or to disturbance in the middle ear.

**Treatment.**—This must depend largely upon the underlying condition. If, for example, the stapedius muscle is involved conjointly with the facial nerve, the difficulty, which in any event is a trifling one, will pass off with the correction of the facial paralysis. The general nervous conditions which underlie the foregoing abnormal auditory phenomena must be carefully treated. The bromides are undoubtedly useful, but much more important is a consideration of the general nervous and physical health.

**Tinnitus Aurium.**—Under this general heading are included various subjective sound sensations referred to the ear, the character of which may be roaring, hissing, ringing, ticking, or whistling. This affection in its varied forms is extremely common and often most distressing. Its causes are manifold. In consideration of the normal sensitiveness of the auditory

mechanism, it is, in fact, remarkable that subjective sounds are not constantly being heard. One reason for this undoubtedly is that we come to ignore many sounds, which, if our attention were directed to them, would become exceedingly annoying. To city dwellers at any rate noise is practically constant, which, as we have just suggested, is, for the most part, ignored. More in detail, some of the more important causes of tinnitus may be summarized as follows:

Any morbid process in the ear may cause subjective sounds, but especially disease of the labyrinth, which is accompanied by more or less nerve deafness. Among these are alterations in circulation due to anæmia, abnormal pulsation in the carotid artery, intracranial aneurism, or possibly vasomotor palsy of the labyrinthine vessels. Osler speaks of a bruit in the ear, with systolic intensification, which was supposed, for a time, to be due to an aneurism. Cerumen in the external meatus and various affections of the middle ear may be operative in producing tinnitus. The actual involvement of the nerve endings may also be responsible, in which case irritative processes or clonic spasm of the internal ear muscles may be operative. Violent stimulation of the auditory nerve by loud and particularly by continuous sounds may produce a persistent tinnitus. Workmen in boiler factories, foundries, and similar places of employment are not uncommonly so affected. Finally, in various general nervous conditions—hysteria, neurasthenic states, in the aura of epilepsy, or in migraine—subjective sounds may be a prominent feature. A further elaboration of these simple forms of tinnitus leads often to very elaborate subjective sounds, which must then be assigned to the interposition of higher centres and probably are attributable to the action of the cerebral cortex itself. Such are the auditory hallucinations not infrequently observed in the insane. In all such conditions the relation of the mental attitude to the physical condition in the ear is naturally of very great interest and importance in modifying and keeping alive the condition. It is certainly true in this as in other neuroses that a definite cause in the ear leading to simple tinnitus may well produce a habit with which ultimately the original cause has nothing to do. If the tinnitus is double-sided the prognosis is much less favorable than in the unilateral affection.

**Diagnosis.**—In this very little information is to be gained from the character of the sounds. Although it is true that sounds synchronous with the pulse are probably directly or indirectly due to arterial pulsation, nevertheless such sounds may also be intermittent. It may, in general, be said that sounds of arterial origin are more or less continuous, but here again it is necessary to be on one's guard, since, for example, cerumen has been responsible for a constant tinnitus. The apparent location of the sound is also most varied. It may be referred to the ears, be bilateral or unilateral, or be referred to the head either at the occiput or vertex. The relation to deafness is also extremely vague, since at times the tinnitus may decrease with increasing deafness, or in other instances may persist unchanged.

From the foregoing facts it is evident that a definite diagnosis is often extremely difficult. If the sounds are elaborate it is usually safe to assume that they are of central origin, or at least that the cortex takes part in the general condition. The further question of the exact relation of the labyrinth to various subjective sounds must naturally be determined by expert otological examination, and does not concern us at this time. It is, however,

well to note whether continuous sounds are increased or diminished by the recumbent posture. If the tinnitus is the result of venous hyperæmia, it is ordinarily increased by lying down, but if due to simple anæmia, it is relieved thereby. It should also be remembered that certain drugs, notably quinine and the salicylates, may produce a tinnitus, and that persons with middle ear or labyrinthine disease are particularly susceptible. A pulsating tinnitus, due to arterial congestion, may often be relieved by pressure over the carotid artery, and this simple procedure, therefore, serves as a diagnostic hint of some importance.

**Treatment.**—This is usually unavailing unless its cause may be discovered, which is not infrequently impossible, and when possible the condition is often such that there is no available relief. The bromides and counter-irritation behind the ear may at times serve a useful purpose. If syphilis is suspected as a cause a vigorous treatment directed against that disease should be instituted. Tinnitus due to so-called functional disorders of the nervous system may be helped by the use of galvanism, using the anode as the active pole, and also by various suggestive measures.

**Nerve Deafness.**—This term is applied to the inability to hear, conditioned by an affection of the cochlear nerve with accompanying labyrinthine involvement. The details of this subject lead directly into the field of the otologist, but certain tests must be made by the practitioner which will throw light on the general character and location of the disturbance leading to the deafness. For this purpose a watch with a fairly distinct tick, or, better, a middle range tuning fork, is essential. In making such an examination it is first necessary to determine whether the external auditory meatus is clear of wax and the drum membrane intact. Having determined these points, one ear should be closed, the patient directed to close his eyes, and the watch brought from a distance toward the ear, the exact point being noted at which the tick is first distinctly heard. It should be remembered that the tick is not heard at so great a distance under these conditions as when the watch is gradually removed from the ear. The results obtained should be compared with the other ear and with a normal control. Considerable variation will be observed, but relatively speaking the test is sufficient to demonstrate the presence or absence of conduction by air. Should such an examination be negative, it indicates simply that the external ear, the internal ear, and the sound appreciating apparatus are intact; in other words, it forms the most general relative test of hearing. Under certain conditions, however, the conduction through the air and through the internal ear to the cochlea may be interfered with by disease in such a way that sounds no longer penetrate by way of the air. It is, therefore, necessary to examine bone conduction. Under normal conditions a sounding tuning fork placed either over the mastoid or other part of the skull in contact with the skin will be heard in both ears. Inasmuch, however, as the conduction through the bone to the internal ear is less direct than through the air, it happens that the tuning fork, when no longer heard over the mastoid, if placed before the ear will again be heard. This is the usual course of events under normal conditions and in nerve deafness. If, on the contrary, the sound-conducting apparatus is diseased through a plugging of the external meatus or through disease of the middle ear the sound will naturally not be heard aërially after it has ceased to be heard through the bone. If, therefore, there is an affection of the auditory nerve leading to so-called

nerve deafness, a tuning fork is not heard through the bone, inasmuch as the receptive mechanism of sound in the cochlea is diseased. Under these conditions it may or may not be heard aërially. This is known as Rinné's test. This may be summarized as follows: In labyrinthine disease both air and bone conduction hearing are diminished and bone conduction is usually lost; whereas, in middle ear disease air conduction is diminished and bone conduction is increased.

As a further associated test of that devised by Rinné, the Weber test is ordinarily employed. Under normal conditions, if a tuning fork be placed on the forehead in the middle line and one meatus closed, the sound is best heard in the closed ear, since the aërial conduction is prevented on that side, whereas bone conduction is preserved and intensified through the conversion of the ear into a closed chamber. If, therefore, the sound-conducting mechanism is affected, the tuning fork is best heard on the affected side. This is known as a positive Weber test. If, on the other hand, nerve deafness exists from involvement of the labyrinth or the auditory nerve, the fork when applied to the forehead can no longer be heard on the affected side, inasmuch as the percipient mechanism is at fault. This is known as a negative Weber test. The Weber test, in general, is much less dependable than the Rinné test. These two tests are evidently supplementary, and through their use a more definite knowledge may be obtained of the location of disease either in the sound-conducting mechanism, external auditory canal and middle ear, or in the sound-perception apparatus—labyrinth and cochlea. It is an interesting fact that in nerve deafness hearing is usually worse in the midst of noise, whereas in deafness from chronic disease of the middle ear hearing is improved by external sounds.

**Vestibular Nerve.**—Disease of this branch of the auditory nerve, so far as it is separable from affections of the cochlear nerve, leads essentially to disturbances of coördination, of which vertigo, including Ménière's complex, is the most important. Owing to the relations which the vestibular branch bears to other cranial nerves and to the spinal cord, various incoördinations of the muscles of the head, neck, and eyes may result from a lesion of the nerve. Nystagmus, for example, has been observed as one of these symptoms. Tinnitus, due to disturbance in the special sense of hearing, is often difficult to separate clinically from vertigo, of which it is often a symptom. The two conditions are very frequently associated, but it is nevertheless best to consider vertigo as a distinct disorder of equilibration in connection with the vestibular nerve.

**Vertigo.**—This may be defined as a sensation of giddiness and uncertainty resulting from a defect of equilibration, which is a complex act of muscular innervation, regulated ultimately by the cerebral cortex through the vestibular nerve and its connections in the ear and in the central nervous system. Experimental and other evidence goes strongly to show that the cerebellum is important in this function and that fibers of the vestibular nerve passing from the semicircular canals to the cerebellum are of great significance in the preservation of equilibrium. It is also clear that the semicircular canals, standing as they do in close relation to the vestibular nerve, are to be regarded as an essential part of this complex reflex. Finally, the cerebral connections directly and indirectly of the vestibular nerve bring about the cerebral control, without which preservation of equilibrium is impossible. It is perhaps too much to assume that the vestibular nerve

is invariably concerned directly in the production of the symptom of vertigo, but that it indirectly takes part in this condition through its various reflex connections is, in our present state of knowledge, hardly to be questioned. The relation of the vestibular nerve to the ocular nerves and muscles is likewise of great importance, and accounts for the vertigo frequently observed in connection with defects in ocular innervation. The production of vertigo through affections of the larynx and various portions of the gastro-intestinal tract is no doubt anatomically explainable through connections with the vagus group of nerves.

Vertigo is described as subjective when the movement seems to be in the patient himself, and objective when external objects appear to be in motion. The usual result of a feeling of vertigo is the phenomenon of staggering. Inasmuch as vertigo occurs under many conditions, it is often a matter of extreme difficulty to arrive at an estimate of its significance in any given case. On the one hand, vertigo may be one of the symptoms of organic changes in the ear or central nervous system of gravest character, or, on the other hand, may indicate a nervous disturbance of the slightest sort. As Oppenheim has pointed out, a distinct sensation of vertigo may be brought about in perfectly normal persons by closing the eyes, standing on one foot, and directing the attention to the general uncertainty of this position. This fact, however, should not lead us to minimize its possible importance, particularly if associated with other symptoms of more or less grave character. In tumor of the brain, and particularly of the cerebellum, vertigo is a diagnostic point of much significance, particularly if the staggering tendency is persistently in one direction. As an example of the relative ease with which vertigo may be induced, it has been observed that a galvanic current passed through the head leads to a distinct tendency to fall toward the side of the positive pole, whereas a breaking of the current tends to induce falling toward the negative pole. That this phenomenon has direct relations with the semicircular canals is not to be questioned.

It is not possible to give a complete list of the causes and conditions which may induce vertigo. As a general statement, it may be said that anything which alters our normal spatial relations may be productive of vertigo. Some of the more important of these conditions are diplopia, rapid changes of position, especially in a rotary direction, looking from high places, and, more indirectly, through immediate or secondary involvement of the vestibular nerve, general increase of intracranial pressure, notably through tumors especially of the cerebellum, disturbances of circulation, particularly through arteriosclerosis, acute anæmia, multiple sclerosis, migraine, epileptic attacks. Other causes, to some of which allusion has already been made, may be attributed to disorders of the stomach, intestinal tract, nose, larynx, and to certain auto-intoxications. Affections of the ear itself are naturally a prolific cause. Among the most commonplace and practically important of these is a collection of cerumen in the external auditory canal.

**Auditory Vertigo.**—In chronic middle ear catarrh a type of disturbance may occur characterized at times by sufficient vertigo to cause a fall, accompanied with nausea, vomiting, and increase of tinnitus and deafness. Such attacks have been given the name Ménière's symptom complex, to distinguish them from true Ménière's disease, which is presumably a labyrinthine affection, and which should in general be somewhat sharply distinguished from various other forms of vertigo which may under certain circumstances

simulate it. So far as possible it is well to establish a pathological basis in the various forms of vertigo, and this seems possible in true Ménière's disease.

**Ménière's Disease.**—In 1861 a French physician drew attention to the group of symptoms which have since passed under his name. The terms auditory[1] and labyrinthine may be regarded as essentially synonymous with Ménière's disease. The words, at least, are used interchangeably. In limiting the application of this affection it is desirable to realize that it is through the immediate disturbance of the semicircular canals of the labyrinth that the symptom is produced. It should not, for example, be applied to vertigo manifestly due to distant conditions in the nervous system or organs which may, in a secondary way, affect the vestibular nerve. The onset of a typical attack of auditory vertigo is, in general, as follows: Vertigo comes on suddenly, often sufficient to throw the patient to the ground. He is for a varying time stunned and possibly at times loses consciousness momentarily. The vertigo may be either objective or subjective in type. Vomiting and nausea quickly supervene, often persisting for hours, with headache. In these cases there is usually diminished hearing, with reduced bone conduction and almost always tinnitus. Occasionally nystagmus, on looking toward the affected side, diplopia, and facial palsy may supervene. The attacks vary widely in frequency, they may occur daily, weekly, monthly, or at times become practically constant (*Status Ménière*). In the earlier descriptions of the disease the onset was regarded as apoplectic in origin in a previously healthy ear. This view has been broadened by certain writers to the extent of applying the term "aural vertigo" to cases with recognized ear disease.

*Pathological Anatomy.*—Considering the affection in its narrower aspect, the lesion may be regarded as a primary disturbance in the labyrinth, usually hemorrhage, occurring in the course of syphilis, leukæmia, gout, and similar constitutional affections or from injury or from local inflammation. Disturbance in the function of the endolymph is causative of the ensuing symptoms. Deafness naturally results from a lesion of the cochlea, whereas the vertigo depends upon the invasion or disturbance of function of the semicircular canals. Parkes Weber, quoted by Osler, has made the following classification which is rather more general and inclusive than the limited conception of Ménière's disease as due only to disease of the labyrinth would justify. It is, however, of value as a comprehensive statement of the main causes of vertigo. "(1) The apoplectic form, due to hemorrhage into the labyrinth, as in leukæmia, followed, as a rule, by complete deafness in one or both ears. (2) The cases associated with progressive inflammatory disease of the labyrinth. (3) Associated with organic changes in the auditory nerves, as in tumors, sometimes in tabes, and in cases of aural vertigo associated with facial paralysis on one side. (4) Cases in which a paroxysm of epilepsy is preceded by an auditory aura. (5) The moderate attacks which are associated with the various middle ear affections, with wax in the meatus, with violent syringing of the ears, etc., all of which are probably due to increase in the intralabyrinthine

[1] Dr. Clarence J. Blake has made a very useful distinction between "auditory vertigo," due to disease of the true auditory mechanism of the internal ear, and "aural vertigo," due to affections of other portions of the ear.

pressure. Ménière's symptoms may occasionally be due to temporary excessive increase in the perilymph, possibly of angioneurotic character."

*Diagnosis.*—When occurring in typical form and when associated with manifest disturbance in the auditory function this is not difficult. In any condition of vertigo, the ear should be subjected to a careful and expert examination, to determine the range of hearing and the possible affection of the internal ear, even if the patient's attention has not been especially directed to it through tinnitus or other symptoms. The other conditions which may be indirectly the cause of vertiginous attacks, to which we have already alluded, should likewise be carefully investigated. It is, for example, not uncommon to attribute to a disordered stomach conditions which are due primarily to labyrinthine disturbance and of which the stomach disorder is an effect. From a diagnostic standpoint such cases are important as illustrating the confusion which may arise through a failure to make a proper examination of the ear and through being diverted from the source of the trouble by conspicuous symptoms elsewhere.

The greatest diagnostic difficulty lies in a sharp distinction between Ménière's disease and epileptic seizures. In the presence of marked labyrinthine disturbance manifested through deafness or tinnitus, the differentiation should not be difficult. When these symptoms, however, are slight or possibly lacking, Ménière's disease may very closely simulate an epileptic attack. For example, aural vertigo may and often is extremely sudden in onset, extremely short in duration, may unquestionably be accompanied by temporary loss of consciousness, may occur during sleep, and finally, which naturally renders its separation from epilepsy impossible, may be actually associated with that disease. All the foregoing conditions are met with in epileptic seizures in essentially the same form. Evidently, therefore, the points of distinction may be extremely vague, at least in exceptional cases. The violent sense of impulsion, as if being "hurled to the ground," is no doubt characteristic of aural vertigo, and does not occur in the same form in the epileptic seizure. Convulsive movements naturally do not occur in aural vertigo, nor is the onset so sudden that the patient is likely to injure himself. In a recent publication, Gowers[1] has described in much detail and with his usual acumen the extraordinary difficulties which may sometimes arise in the differential diagnosis between these two affections. Stress should always be laid upon the existence of ear symptoms in a supposed Ménière complex. Such symptoms naturally may occur as a complication of a true epilepsy, but if epilepsy is excluded would point strongly toward the labyrinth as the primary source of the affection.

In certain debilitated states of the nervous system, symptoms closely resembling if not identical with Ménière's disease may occur. It has been suggested that under these conditions vasomotor disturbances in the labyrinth may actually lead to a true auditory vertigo.

*Prognosis.*—The outcome of Ménière's disease is always problematical. Experience has, however, shown that with the increase of deafness the vertigo often decreases, and finally, when deafness is complete, may entirely disappear.

*Treatment.*—It is natural that in a condition of this sort a wide range of treatment has been suggested. From what has already been said it

[1] *The Borderland of Epilepsy*, 1907.

is evident that attention should primarily be directed to the condition of the ear. It is found, however, that simpler methods of treatment, such as middle ear inflation, often fail of definite result, making a resort to surgical intervention almost imperative. Eugene A. Crockett, of Boston, has drawn attention to the operation of removal of the stapes for the relief of vertigo. He regards this, however, as justifiable only in those cases in which ordinary treatment has proved futile. Less radical operations on the ossicles are suggested as preliminary measures of relief. The still more radical operation of removal of the semicircular canals necessitates the almost certain destruction of the cochlea as well, with resultant complete deafness. Even this operation, however, may in certain intractable cases be justified. The following general methods of treatment are at times serviceable: The bowel movements should be kept perpectly free, warm foot baths may be given, a residence at Karlsbad or Marienbad and similar watering places may be advised. Iodide of potash may be used to combat a possible syphilitic cause. Ocular and nasal defects should be corrected. Charcot advised quinine in spite of its known effect on the auditory nerve. The salicylates have also been prescribed and the bromides have undoubtedly been found useful at times. Nitroglycerin is indicated in high arterial tension associated with arteriosclerosis. Pilocarpine injections have been extensively used, 5 to 8 drops of a 2 per cent. solution every second day, often continued for weeks, or $\frac{1}{8}$ grain subcutaneously or by the mouth. Lumbar puncture, advised by Babinski, in which from 3 to 20 cc. of fluid are withdrawn in one or several sittings, is a somewhat doubtful procedure.

**Paralyzing Vertigo (Gerlier's Disease).**—Although vertigo is but one of the symptoms of this extraordinary affection and its relation to the auditory nerve is not understood, nevertheless it may properly be discussed at this point. Gerlier,[1] a Swiss physician, first described the affection in 1887. Since that time he, together with David, Haltenhoff, Eperon, Ladame, and Sulzer, have made various communications on the subject. In Europe the affection has been observed only in Collex, a canton of Geneva, and in certain surrounding hamlets. Cowherds and workers in the fields alone appear to be its victims, and they only during the summer months and under poor hygienic conditions. Vigorous young men are particularly likely to be attacked.

*Symptoms.*—Pain in the occipital region, muscular weakness, and disorders of the ocular nerves constitute the characteristic symptoms, combined in various ways and in varying degrees of intensity. Vertigo is a somewhat less conspicuous symptom. The pain in the neck is described as a sense of constriction often radiating into the back. The muscular weakness affects the voluntary muscles and particularly the extensors. The neck muscles may be unsymmetrically involved. An unequal ptosis is a common symptom not accompanied by paralysis of external or internal ocular muscles; the visual fields are at times diminished, and the fundus shows nothing characteristic. At the onset of an attack and preceding the ptosis a certain clouding of vision with vertigo often occurs. A typical attack may be described as follows: "The patient, previously well, is suddenly attacked by pain in the neck and back; his sight is clouded to the point of

[1] *Rev. méd. de la Suisse Romande*, 1887, vii, 1, 260; *ibid.*, 1888, viii, 22, 86; *ibid.*, 1891, xi, 201, 260.

temporary blindness; ptosis develops rapidly along with general weakness of neck and body muscles; standing erect becomes difficult; he reels and has all the appearance of drunkenness. After a period not exceeding ten minutes complete recovery takes place. During an attack the tendon reflexes are preserved, at times increased; the skin sensibility is intact, false trismus occurs, swallowing is often impossible, and general motor weakness dominates the clinical picture. The head often falls forward on the chest, due to weakness of the neck muscles, which, with the ptosis cutting off vision, renders the patient for the time wholly helpless. Nausea does not occur. Many attacks are slight and merely momentarily incapacitate the patient, but the foregoing symptoms invariably are developed in some degree."[1]

*Etiology.*—The cause remains undetermined. Considering the conditions under which the affection is most likely to occur, Gerlier presumed that it was due to an infection derived from stable soil and active only in the warm months of summer. The malady does not occur in cold weather, and the attacks are usually in the afternoon, ceasing shortly after sunset. Other explanations which have been offered, including hysteria, are even less satisfactory than Gerlier's suggestion. Although this type of vertigo, so far as known, occurs nowhere else on the Western Continent, a similar affection was described by Miura in 1897 as occurring in the northern portion of Japan, to which was given the name "Kubisagari."

*Treatment.*—Improved hygiene appears to be the most rational form of treatment. The affection is never fatal, and occurs only in summer.

## GLOSSOPHARYNGEAL (NINTH) NERVE.

**Anatomical Relations.**—The glossopharyngeal nerve forms a constituent part of the vago-glossopharyngeal-accessory group, and is mixed in function with a predominance of sensory fibers which presumably transmit, in part, the sense of taste for the posterior portion of the tongue. The motor portion of the nerve supplies only the stylopharyngeus muscle and secretory fibers to the parotid gland, whereas the more important sensory portion provides general sensibility to the middle ear, fauces, tongue, and pharynx. The more important terminal branches of the nerve are: (1) Tympanic or Jacobson's nerve, helping to constitute the tympanic plexus from which the small superficial petrosal nerve arises; (2) branches to the pharynx; (3) a muscular branch to the stylopharyngeus; (4) tonsillar branches to the tonsils, soft palate, and pillars of the fauces; (5) lingual branches, supplying the circumvallate papillæ and the posterior portion of the dorsum of the tongue, the glosso-epiglottic and pharyngo-epiglottic folds, lingual surface of the epiglottis, and sides of the tongue posteriorly. The sensory portion of the nerve arises from the jugular and petrous ganglia in the jugular foramen. The axones pass inward to end in part in the dorsal nucleus of the fourth ventricle and in part in the so-called solitary bundle. The nucleus ambiguus gives rise to the motor portion, which may also derive fibers from the dorsal nucleus. It is a matter of anatomical and practical interest that the nerve of Wrisberg ends, in part, in the same nucleus

[1] E. W. Taylor, *Paralyzing Vertigo*, p. 463, Posey and Spiller, *The Eye and Nervous System*, 1906.

as do fibers of the glossopharyngeal. Both of these nerves are possibly concerned in taste, and it may add somewhat to clearness of understanding to regard the nerve of Wrisberg as an aberrant strand of the glossopharyngeal. The central connections of the glossopharyngeal nerve so far as determined are in the neighborhood of the lower portion of the precentral gyrus.

**Symptoms.**—Isolated disease of this nerve is not definitely known, or at least the cases are too few upon which to base conclusions of value. It follows, therefore, that exact knowledge of its function is vaguer than that of any of the other cranial nerves. Clinical investigation and experiment have established beyond much doubt that the dorsal portion of the tongue and neighboring parts of the pharynx are supplied with taste fibers through it. The question has, however, been raised that possibly even these fibers finally reach the brain through the fifth nerve. Others are of the opinion that the chorda tympani fibers finally enter the central glossopharyngeal. This and similar disputed points are difficult of determination on account of the rarity of affection of this nerve without coincident involvement of its neighbors. Cassirer, working with Oppenheim, has reported a case which, in their opinion, demonstrates the fact that in certain instances all the taste fibers may run in the glossopharyngeal. In addition to the innervation through the glosso-pharyngeal, parts of the posterior portion of the tongue and neighboring parts are also innervated by the fifth nerve, which adds still further to the difficulty of exact determination of the boundaries of the two sensory supplies. It may, however, be accepted that the chief innervation of the pharynx is from the glossopharyngeal. So far as this nerve is concerned with the act of swallowing and with the motor innervation of the œsophagus in general, its relations to the vagus presumably must be held responsible. Its direct relation to salivary secretion is probable. The nerve is very frequently involved in disease of the bulb, but rarely alone, as would naturally be inferred from its extremely close central relations, particularly with the vagus and accessory nerves. Disease external to the bulb in the posterior portion of the skull, aneurism, tumors, thrombosis of the jugular vein, may implicate the nerve, but here again rarely alone, nor are injuries any more likely to affect it and not others. Oppenheim describes an interesting case following protargol insufflation, in which the symptoms were slight paralysis of the palate and throat muscles, with loss of taste in the parts supplied by the nerve. This condition he thought due to chemical action on the nerve endings. Degeneration of the nerve may occur in tabes. Disturbances of taste in affections of the middle ear due to involvement of the tympanic plexus and increase of salivary secretion have been observed. These phenomena are of particular interest in connection with the known anatomical relations of the nerve.

## PNEUMOGASTRIC (VAGUS, TENTH) NERVE.

**Anatomical and Physiological Considerations.**—The pneumogastric is to be regarded as a mixed nerve, although the exact origin, particularly of its motor root, remains in some doubt. The commonly accepted theory is that fibers from the nucleus ambiguus, together with the accessory portion of the spinal accessory nerve, constitute the motor portion of the vagus. Certain investigations, however, seem to show that the accessory nerve to

the vagus does not exist and that the motor fibers are derived wholly from the nucleus ambiguus. Another possibility is that some of the motor fibers are derived from the dorsal nucleus, as already described for the glossopharyngeal, although this nucleus is unquestionably for the most part, sensory, and concerned with the reception of fibers originating from ganglia of the root and of the trunk situated in and near the jugular foramen. The sensory axones pass into the oblongata between the restiform body and the olivary eminence, and terminate partly in this dorsal nucleus and partly in the descending root or solitary bundle in common with fibers of the glossopharyngeal nerve. In general, the vagus and glossopharyngeal nerves are not to be sharply separated anatomically in their course within the oblongata. Whether certain muscles are innervated from one or another part of these nuclei does not now concern us. The central connections of the vagus nerve are not definitely determined, and have as yet small practical significance. The general supply of the pneumogastric nerve is the most extensive of any of the cranial nerves. Its motor portion through the accessory nerve or its own fibers derived from the nucleus ambiguus supplies the muscles of the soft palate excepting the tensor palati and possibly two others, the pharynx, œsophagus, stomach, intestine excepting the rectum, the larynx, trachea, and bronchi. The sensory supply is to the dura,[1] external ear, ear, pharynx, œsophagus, stomach, larynx, trachea, bronchi, and pericardium; in addition, fibers are sent to the heart, spleen, pancreas, kidneys, suprarenal bodies, and intestinal bloodvessels. Through various ganglia wide connections are affected with the facial, trigeminal, glossopharyngeal and hypoglossal nerves, and with the sympathetic system. As has been suggested above, the confusion in determining the exact glossopharyngeal distribution is due largely to the complex relationships of the vagus and the part it plays in the innervation of the gastro-intestinal tract.

**Etiology.**—Although the vagus nerve is seldom affected by a primary isolated neuritis, it is easily involved through other causes because of its extremely long course and wide distribution. There is evidence to show that the nerve may take part in a general neuritis or analogous conditions. For example, the nerve may be affected in a general alcoholic neuritis, and its involvement in diphtheria, as described by J. J. Thomas[2] and others, is recognized as possibly one of the causes of sudden death in that disease. The nerve may also be affected in other infectious diseases and also in intoxications through lead and arsenic. Tumor, aneurism of the vertebral artery, meningitis, and similar disturbances external to the oblongata may injure the nerve, but in this case, as with the glossopharyngeal, neighboring nerves are usually affected at the same time, among which the ninth, eleventh, and twelfth are the most frequent. Sclerosis of the vessels of the posterior fossa may lead to disturbance in the function of the nerve, and it is not infrequently injured in operations for tumors of the neck and in tying the carotid artery. Conditions affecting the bulb itself, as, for example, softening, hemorrhage, bulbar paralysis in its various forms, tabes, and multiple sclerosis, are responsible for lesions of central origin. A noteworthy lesion, to which reference will later be made, is the involvement of the nerve

[1] Cushing, as the result of surgical experience in which he found the dura insensitive, is inclined to the opinion that the popularly accepted view that the membrane is sensitive is incorrect. *Jour. Amer. Med. Assoc.*, 1908, I, 847.

[2] *Med. and Surg. Reports*, Boston City Hospital, Ninth Series, 1898, p. 52.

in tabes, which presumably leads to the crises characteristic of that disease. In many of the hysterical conditions on the part of the gastro-intestinal, respiratory, and circulatory systems the nerve plays an important part.

**Symptoms.**—The symptoms produced by affections of the vagus nerve are naturally varied because of the possibility of wide location of the lesion producing them. A total paralysis of the nerve is rare, but may occur through extensive destructive processes in the posterior fossa. In this case, however, lesions of other nerves are a usual accompaniment as already suggested. In one-sided paralysis the symptoms of unilateral weakness of the palate and larynx are conspicuous. The soft palate is flaccid and hangs away from the affected side, not moving perfectly with phonation. Speech is nasal and there is more or less difficulty in swallowing. The vocal cord on the affected side does not take part in phonation or respiration. There may also be partial anæsthesia of the pharynx and larynx. In unilateral lesions of the nerve the condition of the heart is not constant. At times there is slowing of the beat, but more often an increase. Respiratory disturbance occurs usually only with a double lesion. Other general symptoms due to irritation or paralysis of the vagus are vomiting, bulimia, loss of appetite and thirst, pain in the epigastrium, and, conceivably, production of a diabetes. Further symptoms may best be considered in connection with the distribution of the individual nerves.

**Pharyngeal Branches.**—The innervation of the pharynx is through the vagus and pharyngeal nerves by way of the pharyngeal plexus. It is difficult to determine the exact part played by each nerve in the paralyses. The symptoms of disturbance in this field of innervation are difficulty in swallowing, inasmuch as the food is not properly passed on or enters the larynx, or, if the soft palate be involved, passes into the posterior nares. The so-called nasal voice is a common accompaniment. This group of symptoms is characteristic of lesions of the nuclei of origin, as seen in bulbar paralysis, in which death commonly results from actual starvation. The involvement of the pharynx with good prognosis is also common in post-diphtheritic paralysis. The difficulty in swallowing is very slight or negligible in one-sided paralysis. Spasmodic affections of the pharynx often occur without known structural basis, and are at times important as part of a more general neurosis, as, for example, hysteria. A pharyngeal spasm may at times be induced by exaggerated self-consciousness. It occurs also as one of the symptoms of hydrophobia.

**Laryngeal Branches.**—The importance of a laryngeal examination in all cases where there is a suspicion of the involvement of this region cannot be exaggerated. A palsy of the larynx may exist entirely without objective symptoms which would call attention to its existence. For example, in common abductor paralysis there is not necessarily any disturbance in vocalization, but the quality of the voice is usually slightly changed.

The innervation of the larynx is through the superior and inferior or recurrent laryngeal nerves. The superior laryngeal nerve supplies sensation to the larynx and motor fibers to one pair of muscles, the cricothyroids, which act as tensors by separating the points of attachment of the vocal cords on the thyroid cartilage from those on the arytenoids. Paralysis of this nerve, therefore, leads to relaxation of the cord or cords, which, therefore, become slack on phonation. The voice is not lost, but is weak and easily tired. The paralysis is not well marked. Paralysis of the

superior laryngeal nerve is rare because of its short course, but may be produced by wounds, tumors, or enlarged glands. It is also often obscured by the much commoner paralysis of the muscles of the cords.

The other muscles of the pharynx proper are wholly supplied by the recurrent laryngeal nerves, which also give sensory fibers to the mucous membrane below the cords. The left recurrent nerve curves around the arch of the aorta and the right passes under the subclavian artery. It is natural, therefore, that these nerves are peculiarly liable to pressure from aneurism and also on account of their long course through involvement by tumors, glands, and other processes in the upper thoracic region. Varied palsies of the laryngeal muscles may result, giving rise to a wide diversity of symptoms and to a varied laryngeal picture.

In conditions of health the edges of the vocal cords are straight on phonation, parallel and nearly in contact. Abduction takes place on deep inspiration and there is slight movement in quiet breathing. Resting, the glottis is wider than when in the cadaveric position or in complete paralysis, due to the slight tonic action of the abductors. In gradual degeneration of the recurrent laryngeal nerves the paralysis of the muscles follows a definite order (Semon's law). Abduction is first affected through disturbed action of the posterior crico-arytenoids. The thyro-arytenoids are next involved, and then the lateral crico-arytenoids. The arytenoid, unpaired, has a bilateral nerve supply, and is, therefore, not affected in unilateral cases. The analogy in this law to paralysis of skeletal muscles of the arm and leg is interesting.

**Abductor Paralysis.**—In unilateral abductor paralysis the affected cord lies in the middle line. On phonation the sound cord adducts and the larynx looks normal. The palsy becomes evident on inspiration. The voice may not be affected. Owing to the reduction of the aperture between the cords, dyspnœa occurs on exertion, but in adults ordinary quiet breathing is not affected. The symptoms of dyspnœa are more marked in children, owing to the relatively small size of the glottis. In bilateral abductor paralysis both cords lie near the middle line. On inspiration, owing to the shape of the cords, they act as a valve and are drawn closer together, causing marked inspiratory dyspnœa with stridor. In this form of paralysis sudden death is always possible from a cutting off of the air current. On failure of the thyro-arytenoids, the cords are no longer tense and hoarseness ensues.

**Total Unilateral Recurrent Paralysis.**—In this type of paralysis the cord lies in a position between abduction and adduction, the so-called cadaveric position. On inspiration the sound arytenoid is abducted and lies behind the other. On phonation the sound cord moves across the middle line, and lies at a slightly higher level than the other. The voice is not lost but is hoarse and easily tired.

**Bilateral Recurrent Paralysis.**—The severe dyspnœa which occurs in bilateral abductor paralysis does not occur when the paralysis is more complete, but the voice is completely lost. Both the cords lie in the cadaveric position, but slight inward movement from the partially active arytenoid may occur, presumably through its possible supply from the superior laryngeal nerve.

**Diagnosis.**—The diagnosis of these various forms of paralysis is made chiefly from the laryngoscopic appearances and from conditions of phonation and respiration.

**Functions of the Larynx.**—There are two main functions of the larynx—(1) phonation, and (2) respiration. The adductors are essential to the former and the abductors to the proper performance of the latter. Phonation, being a later acquired function, is under direct cerebral control, the centres controlling it presumably lying in the lower part of the precentral area, with further connections with the areas presiding over the general function of speech. Stimulation of the cortical centre for phonation in either hemisphere leads to adduction of both cords, demonstrating, as is usually the case in muscles which act under normal circumstances together, that the innervation is not entirely from one hemisphere. Respiration, which is the more fundamental function, and which in a certain sense is antagonistic to phonation, persists after the destruction of both cerebral hemispheres, as experimentally demonstrated. These facts are of interest in connection with the observation that so-called functional disorders lead to paralysis of the adductors, whereas abductor paralysis is due to structural changes. In lesions lying above the bulbar nuclei, if unilateral, paralysis of the cords does not ensue, as, for example, in hemiplegia, even though it be associated with aphasia. A bilateral lesion of the central tract, on the other hand, causes an adductor paralysis. Direct paralysis of the recurrent laryngeal nerve may result from lesions in the oblongata, at the base of the brain, in the vagus nerve, or in the recurrent laryngeal itself. In nuclear disease affection of this nerve is usually combined with that of others, often associated with a persistent rapidity of the pulse. Syphilis, meningitis, or other disturbances in the posterior fossa may lead to laryngeal paralysis. A primary neuritis analogous to that of the seventh nerve is unusual, but has been described; paralyses resulting from toxic agents, as, for example, a general neuritis arising from lead or a degenerative process in the course of diphtheria or other infectious disease, is far less unusual. The vagus nerve in the neck may be variously injured, as before described. The frequency of laryngeal difficulty in the course of tabes, although not definitely understood, is of interest particularly in relation to the laryngeal crises which at times form one of the most distressing features of that disease. Anæsthesia and various paræsthesias of the larynx are not infrequent in this affection.

The condition of the larynx in tabes has been carefully studied by Dorendorf, of Berlin, in 1903, and more recently by D. Crosby Greene,[1] of Boston, who found in 60 cases of tabes that 15 per cent. showed laryngeal complications, 10 per cent. paralysis of one or both vocal cords, and 12 per cent. were affected with laryngeal crises, figures which differ rather widely from those secured by Dorendorf on a basis of 245 cases. The type of paralysis in Greene's cases was always abductor, and this much more often unilateral than bilateral. The reason given for this fact is that the lesion is presumably a progressive degeneration of the fibers of the recurrent laryngeal nerve, and inasmuch as the abductors are the weaker group, they are the first to succumb. Greene further found that laryngeal crises are among the earliest laryngeal manifestations of the disease, from which the general practical conclusion is drawn that tabes as an etiological factor of certain laryngeal difficulties should be carefully considered. In certain cases, undoubtedly, the diagnosis of the more generalized disease may be made from the laryngeal signs.

[1] *Boston Med. and Surg. Jour.*, 1906, cliv, 97.

Perhaps the most usual causes of the involvement of the nerve are the trauma of surgical operation, the presence of aneurism and enlarged glands, cancer, goitre, effusions in the pericardium, and mediastinal disease. The involvement of the recurrent nerve is unilateral or bilateral, depending upon the extent of the causative process. Aneurism is, no doubt, the most important single cause, but tuberculosis is also often operative. Involvement of the right cord should, in fact, suggest the possibility of tuberculosis of the lung, because of the fact that the nerve in its course downward lies close to the pleura and may easily be involved in an incipient tuberculous process.

**Treatment.**—The treatment of laryngeal conditions comes almost wholly within the province of the laryngologist. The practitioner, however, is often called upon to treat the general condition, of which a laryngeal affection is one of the symptoms. Constitutional disease, for example, such as syphilis and tuberculosis or a general neuritis, must be treated by appropriate means irrespective of the laryngeal condition. The application of faradism to the affected laryngeal muscle under guidance from the laryngoscope is, at best, a doubtful procedure, and would certainly not be undertaken by any but a specialist. Surgical intervention in the various conditions which may lead to compression of the nerve in the neck and mediastinum falls within the province of the surgeon.

**Adductor Paralysis.**—As has before been said, adductor paralysis leading to difficulty or abolition of phonation forms a distinct class of functional paralysis. The great part which hysteria plays in these paralyses has been a subject of much discussion. It is probable, however, as pointed out by Cartaz, Janet, and others, that in hysterical conditions there is not much disturbance in the vocal cords, and that, as a matter of fact, the paralysis is apparent rather than real. In this, as in other hysterical paralytic conditions, the source of the paralysis is unquestionably central, and lies rather in a central incapacity for proper innervation than in any disorder of the cords themselves. In other words, in hysterical mutism, if the patient were able to utter the sound which would lead to the proper action of the cords, the cords would no doubt functionate properly. The movement of the cords, however, cannot be made simply because the words or proper sounds cannot be framed, and this naturally is purely a mental process. In general, it is probable that the so-called hysterical origin of adductor paralyses has been exaggerated, inasmuch as such disturbances are very frequently due to local disease immediately affecting the proper use of the laryngeal muscles. It may be said that adductor paralysis is never due to organic disease in the passage of the nerve from the bulbar nuclei to the larynx, but is either a pure neurosis or due to local disease. These myopathic paralyses in some degree are frequent in various forms of laryngitis. Although aphonia may be and often is a manifestation of hysteria, most of the cases are not purely hysterical in the proper sense of that term. Any state, for example, which requires greater effort than normal for proper phonation, as, for example, debilitated conditions in general or laryngeal catarrh, naturally predisposes thereto. Difficulty in phonation is especially common in tuberculosis on account of the frequent combination of extreme debility and laryngitis apart from immediate involvement of the nerve. In hysterical conditions whispering is usually possible, but even this may be at times lost, leading to the condition of so-called hysterical mutism. As suggested above, recent investigation has undoubtedly shown that these hysterical laryngeal

difficulties are usually due to an emotional cause, which not infrequently may be traced with ease in the past life of the individual.

Adductor paralysis is almost always bilateral and seldom complete. It usually consists merely in an imperfect apposition of the cords. There are three sets of adductor muscles—the lateral crico-arytenoids, the thyro-arytenoids, and the unpaired arytenoid. These muscles may be affected together or irregularly.

**Diagnosis.**—Adductor paralysis may be mistaken for total recurrent paralysis. In neither do the cords come together on phonation. In incomplete adductor palsy the cords separate widely on deep inspiration, whereas in complete paralysis of the adductors the cords lie in a position of extreme abduction, which should prevent confusion of interpretation.

**Treatment.**—Here again the treatment, except when the affection is due to a general neurosis, falls within the special province of the laryngologist. The neurologist, however, is often called upon to treat the aphonias and mutisms which depend wholly or in part upon mental, hysterical causes. Suggestion variously applied, particularly by means of a strong faradic current applied over the larynx, will frequently restore speech. Such superficial methods of treatment, however, are usually temporary in effect and relapses are the rule rather than the exception. Recognizing, as is now done, that such disturbances of speech, as analogous disturbances in other parts of the body, are usually attributable to an emotional cause or to a faulty attitude of mind, a much more rational treatment is a painstaking investigation of the past life of the patient, with particular reference to the cause or causes which may have induced the speech defect.

**Spasm of the Laryngeal Muscles.**—Spasm of the laryngeal muscles leads to adduction of the cords, as, for example, artificially induced by stimulation of the cortex. The abductors, if affected, are overpowered by the stronger adductors. Such spasmodic affections are usually bilateral, even if due to irritation of the vagus fibers on one side. The causes are irritations of the nerve in its central or peripheral course, and naturally precede the paralytic conditions due to a destruction of the nerve. Therefore, any pressure of mild degree on the nerve in its peripheral course, tumors in early stages, slowly growing aneurism, and local disease of similar sort may lead to the condition of spasm. A structural central lesion may likewise be a cause, and the condition has been observed in tabes, general paralysis, and hydrophobia. In hysterical conditions, instead of a loss of function of the laryngeal muscle, there may likewise be an overaction leading to spasm. In children the condition known as "*laryngismus stridulus*" is of practical importance. The symptoms of this latter condition are a tickling sensation in the throat, cough and closure of the glottis, leading to violent inspiratory efforts. The child clutches at objects in his neighborhood, and is evidently distressed and anxious during the period of progressive cyanosis. With or without temporary loss of consciousness there is a final relaxation of the spasm with entrance of the air. A fatal result in these cases is rare unless there be a definite and permanent source of obstruction to the inflow of air. Otherwise, relaxation of the cords inevitably follows when the cyanosis has reached the point of unconsciousness. The attacks are nevertheless distressing, and may last for hours. The *prognosis* is ultimately good. If *treatment* is demanded inhalations of amyl nitrite or chloroform will relieve the spasm.

**Phonic Spasm (Spastic Dysphonia.)**.—This condition may be classified as a rare occupation neurosis allied to writer's cramp and similar affections. It occurs particularly in voice users, and gives rise to a variety of spasmodic affections similar to those just described.

**Sensory Neuroses.**—Anæsthesia of the larynx, complete or partial, may result from injury of the nerve through disease, notably diphtheria, or from bulbar affections of varied character. Except in purely functional conditions, the motor portion of the nerve is ordinarily involved as well. The result of such anæsthesia is that food easily enters the larynx, often resulting in a fatal deglutition pneumonia. The *prognosis* is unfavorable in cases of total bilateral anæsthesia. The *treatment* must be of the underlying condition so far as is possible. Special care must be taken in feeding the patient to prevent the passage of food into the larynx. In unilateral cases the patient should be directed to swallow very slowly, lying on the sound side during the process. In complete sensory paralysis the œsophageal tube is necessary, the strictest care being taken not to pass it into the insensitive larynx. Hyperæsthesia and paræsthesia of the larynx are not uncommon in certain neuroses and in tabes.

**Cardiac Branches.**—The cardiac plexus of nerves is formed from branches of the inferior laryngeal nerve from the main trunk of the vagus and from the sympathetic system. By these means the motor, sensory, and trophic functions of the heart are subserved. In one-sided lesions of the cardiac branches the symptoms on the part of the heart are not constant. At times there is slowing of the beat, but more often an increase in its rapidity. Such results have been observed both in experimental compression and in section of the nerve. In unilateral section of the vagus at times no symptoms whatever are produced, the laryngeal muscles even not showing the effect of the lesion. This is no doubt to be explained by the fact that muscles which ordinarily act bilaterally are innervated from both sides of the brain. The phenomenon at times observed of voluntary control of the heart is of interest in this connection. It occasionally happens through some peculiar aptitude that a person is able, through an act of the will, to regulate within certain limits the rapidity of action of his heart. In one instance which has come under personal observation the experiment was discontinued because of the very considerable nervous strain which it entailed.

**Sensory Symptoms.**—A consideration of the various cardiac neuroses dependent upon the disordered function of the vagus nerve would lead beyond the limits allotted to this section. It is one of the best recognized facts in medicine that the heart is peculiarly subject to functional disorders. The effect of the emotions on this organ has always been a theme of the poets. Some of the conditions which may be attributed to a disorder of sensory mechanism of the heart are palpitation, arhythmia, tachycardia, bradycardia, angina pectoris, so-called pseudo-angina, and Stokes-Adams disease.

**Pulmonary Branches.**—Unless due to actual disease of the larynx or to organic disease of the lungs themselves, respiratory disorders are usually attributable to central causes or to bilateral lesions of the nerve. The evidence, however, thus far secured regarding the exact part played by the lesions of the nerve in the respiratory act is somewhat conflicting. Slowing, rapidity, and irregularity of respiration have all been observed. Oppenheim speaks of a case in which slowing of the pulse was associated with increase

of respiration, attributable to vagus disease. In tabes, a slowing of the respiration to three or four in a minute has been observed and has been supposed to be due to a double vagus lesion. A permanent disturbance of respiration does not occur in unilateral vagus disease. It is possible that the motor fibers controlling the bronchial vessels are concerned in the production of asthmatic conditions, and possibly also are responsible for a form of emphysema.

**Œsophageal and Gastric Branches.**—Branches of the nerve to the œsophagus are rarely diseased alone. A not infrequent condition, which must be classed among the functional disorders, is spasm of the œsophagus unassociated with organic stricture. A case under observation for many years in an elderly man without organic disease has shown marked improvement through treatment by faradism. In this instance, any carelessness in swallowing or any lack of proper mastication is liable to excite a violent spasmodic contraction of the lower end of the œsophagus, leading often to regurgitation and expulsion of food. The vagus nerve must be regarded as the sensory nerve for the stomach, through the local irritation of which the sensation of hunger is presumably induced. In its motor function to the stomach, its section lessens contractions. Vomiting is to be regarded as a sign of undue irritation, occasioned either by irritation from the presence of a foreign body, from overmuch food, or indirectly from the effect on the nerve of cerebral pressure. The gastric crisis in tabes is a phenomenon entirely analogous to the laryngeal crisis in the same disease.

**Vagal Attacks.**—Under the heading "Vagal and Vasovagal Attacks," Gowers has recently described seizures of peculiar type which he regards as due to disturbances of some of the functions of the pneumogastric. The symptoms of these seizures are, for the most part, sensory, and include subjective, gastric, respiratory, and cardiac discomfort, at times accompanied by cardiac pain and a sense of impending death. In addition to these symptoms there is frequently a slight accompanying mental change with vasomotor constriction of peripheral vessels. Slight tetanoid spasm and some sensory impairment complete the general clinical picture of a somewhat vague but no doubt important symptom complex. Seizures having the foregoing symptoms in varying degrees have a duration of ten minutes to one-half hour or more, occurring at varying intervals often for months or years. Gowers calls attention to the fact that a condition somewhat similar to these seizures has been described by Nothnagel and others, who have, however, not regarded the group of symptoms as features of a definite attack. The effect of compression of the vagus nerve in the neck, as shown by Waller many years ago, is, first, difficulty in breathing, followed by labored inspiration, uneasiness over the precordial region, and gastric discomfort at times amounting to nausea. If the pressure is continued, syncope results. These symptoms were not considered as due to compression of the carotid. In Gowers' experience women suffer more frequently from these vagal attacks than men, but he repudiates the idea that the seizures are to be regarded as manifestations of hysteria.[1]

[1] "Women suffer more frequently, but these attacks are also met in men. This and the fact that the pneumogastric and vasomotor systems are readily influenced by emotion have probably led to the frequent submergence of these attacks beneath the vague conception of hysteria, a conception which conceals whatever it covers. We must rescue from it whatever we wish to study. It can be put back again afterward if desired."—*Borderland of Epilepsy*, p. 19.

## SPINAL ACCESSORY (ELEVENTH) NERVE.

The eleventh or spinal accessory nerve is purely motor in function. The portion of the nerve accessory to the vagus has already been considered in connection with the latter nerve. The spinal portion may properly be considered independently.

**Anatomical Relations.**—The spinal accessory nerve has its origin from cells lying in the dorsolateral portion of the ventral horns of the cord, extending from the fifth or sixth cervical segment to the lower end of the oblongata. Unlike the ordinary motor spinal nerve, the fibers emerge laterally and the separate bundles unite into a common trunk, passing upward in the subdural space, entering the cranial cavity through the foramen magnum, thence after joining the vagus nerve passing outward through the jugular foramen. At the lower margin of this foramen, the spinal and accessory portions of the nerve separate, the accessory portion remaining with the vagus and the spinal portion entering and supplying the sternomastoid and trapezius muscles. Within the substance of the sternomastoid muscle the nerve unites with filaments of the second cervical nerve, which, however, play no significant part in the innervation of the muscle. Passing through the sternomastoid, the nerve enters the trapezius, sharing with the third and fourth cervical nerves the innervation of that muscle.

**Etiology.**—There are many conditions leading to involvement of the spinal accessory nerve and consequent disturbance in the functions of the two large muscles which it in part supplies. Lesions of the cervical cord itself, with destruction of the cells of origin of the nerve, are not infrequent. The more important of these are cervical myelitis, progressive muscular atrophy of spinal origin, gliosis and syringomyelia. Disease of the bone, notably tuberculous processes in the upper cervical vertebræ, pachymeningitis of tuberculous or syphilitic character, are likely to involve this nerve. Injuries of the nerve in the neck, particularly in operations for tumors, and of late the voluntary injury of the nerve to provide an anastomosis with the facial in cases of intractable facial paralysis, naturally lead to its partial or complete paralysis. A primary neuritis has also been described. In most instances, especially when the lesion lies outside the cord, the affection of the nerve is one-sided.

**Symptoms.**—The symptoms of paralysis are purely motor and consist in paralysis of the sternocleidomastoid and partial paralysis usually of the trapezius. Under ordinary conditions of rest a paralysis of one sternomastoid muscle does not lead to noticeable deformity. The defect, however, is brought out when the head, better against resistance, is turned toward the side away from the affected muscle, with slight raising of the chin. In paralysis of the right sternomastoid such a movement toward the left will be incomplete and no contraction of the muscle will take place. If the sternomastoid is paralyzed on both sides, the head tends to fall backward, and in a horizontal position cannot easily be raised. The attachments of the trapezius are such that a certain distinction may be made between the paralysis of its three portions. Under normal conditions the muscle, through its attachments in the neck and throughout the thoracic region of the spine to the upper and outer portion of the scapula, tends to raise the shoulder and approach the scapula to the middle line. The upper portion

of the muscle from the occiput to the outer third of the clavicle draws the head backward and turns it slightly toward the opposite side. The middle portion from the ligamentum nuchæ and the three upper thoracic vertebræ to the acromion and outer portion of the spine of the scapula elevates the scapula. The lower portion from the fourth and subsequent thoracic vertebræ to the inner half of the spine of the scapula draws the scapula toward the middle line. In paralysis of the clavicular portion of the muscle the shoulder does not move in respiration, but the failure of backward motion of the head is inconspicuous because of the continued action of deep neck muscles. This upper bundle of the trapezius is very apt to be spared in paralytic conditions. Paralysis of the middle portion leads to a sinking of the acromion through lack of fixation, which the trapezius ordinarily gives it, and through the continued action of the levator anguli scapulæ. The shoulder is therefore depressed forward and cannot be raised. The raising of the arm suffers somewhat in consequence. Paralysis of the lower portion leads to an increased distance of the inner border of the scapula from the middle line, with increased prominence of the scapula as a whole.

**Prognosis.**—The prognosis of lesions of the spinal accessory nerve from the causes given above naturally depends upon their individual curability. In syphilitic conditions and those resulting from transient inflammations a favorable outlook may be anticipated. In general, if the nerve be given opportunity, it will rapidly regenerate without special treatment. In hopeless paralyses much comfort and improved action of unaffected muscles may be obtained by the application of apparatus to elevate the shoulder.

**Spasmodic Affections of the Neck Muscles.**—Spasm of the neck muscles, including the innervation of the spinal accessory nerve, is perhaps the most distressing form of this type of disturbance. The variety of spasm which may develop in this general group of muscles is so great that merely an artificial classification may be made. Although the sternomastoid and trapezius frequently take part in these spasmodic affections, and often predominate in the clinical picture, we are usually not justified in regarding the spasm as wholly under the control of the spinal accessory nerve, inasmuch as other muscles supplied from other sources are almost invariably involved. Among these other muscles, the splenius, scaleni, and deep muscles of the neck, and at times the platysma and omohyoid, are of particular importance. The fact should, therefore, be recognized that, although for purposes of clinical convenience we regard certain common spasmodic affections of the neck as due to disturbance in the spinal accessory innervation, the affection is usually much more widespread and shows a distinct tendency to pass to other muscles. The importance of this in relation to treatment will receive attention later.

**Torticollis.**—Torticollis, or wryneck, occurs in various forms, some of which are quite independent of the nervous system. Of these the following types may be mentioned: Rheumatic torticollis, so-called caput obstipum, is undoubtedly due to an affection of the muscle substance which leads to a rigid position of the head, often associated with much pain. This, the ordinary stiff neck, has no relation with a true spasm, occurs chiefly in children, and is entirely self-limited in its course. A fixed torticollis due to congenital or other shortening of the muscles supporting the head is likewise

not a form of spasm, and demands no special consideration here. A type of acquired torticollis, usually of slight degree, which may lead to confusion in diagnosis, is that associated with carious processes in the upper vertebræ in the absence of external deformity. An abnormal and rigid position of the head, especially when associated with an otherwise unexplained exaggeration of the deep reflexes, should lead to the suspicion of a tuberculous or other process in the upper cervical region. The rigidity of the neck often seen in meningitis and other conditions which lead to pressure on the outgoing nerves is usually easy of diagnosis in view of accompanying symptoms.

**Spasmodic Torticollis.**—The most practically important variety of spasm of the neck muscles is the so-called spasmodic torticollis, or wryneck, in which the muscles innervated by the spinal accessory nerve play a predominant but by no means exclusive part.

*Etiology.*—The etiology is vaguely understood, hence a great variety of causes has been given, few of which are explanatory of the fundamental underlying condition which actually induces the spasm. Oppenheim, among others, is very strong in the conviction that the affection is observed particularly among persons with hereditarily unstable nervous systems, although direct inheritance is exceedingly rare. Frequent association of torticollis with other nerve disorders, either neuroses or psychoses, lends a certain weight to this view, as does also the fact that many of the cases yield most satisfactorily to treatment by psychotherapy. To make the statement, however, that the cases occur primarily in an otherwise unstable nervous system does little to explain the exact nature of the mental or physical process which makes possible this special spasmodic manifestation. In view of the fact that there is no known pathological anatomy, we are entirely justified in assuming that the cerebral cortex plays an important part in the production of the spasm, whether or not it be on a previous neuropathic basis. Various somewhat fanciful explanations of the affection have been submitted from time to time which cannot be regarded as of universal application. Irritations in the distribution of the trigeminal and occipital nerves, localized peripheral irritations as from too small a collar, disorders in the symmetrical innervation of the two sides of the body, eye-strain, organic disease of the brain, intoxications of various sorts, even astigmatism, are some of the causes which have been assigned. Perhaps in the majority of cases no adequate exciting cause whatever is to be found, and recourse must be had to the explanation that mental perturbation, overaction of the muscles, and habit are responsible. That the position of the head extending over a long period of time may ultimately lead to a spasmodic torticollis is shown by a patient recently under observation, whose work, that of a watchmaker, necessitated sitting with his head inclined and the use of a powerful glass at one eye. This work he had done for many years, and gradually a perfectly typical spasmodic torticollis had developed, which appeared unquestionably due to the constrained position in which he had been obliged to hold his head.

*Symptoms.*—The form of spasm varies in different cases from slight, hardly noticeable involuntary movements of the head to violent spasmodic contractions, painful to the patient and extremely conspicuous to the onlooker. Clonic spasms usually associated with tonic muscular contractions are the characteristic forms in which the affection manifests itself. In those

# PLATE XXXI

FIG. 1

## Torticollis.

Showing spasm of the right sternocleidomastoid muscle, and characteristic position of the head at the height of the spasm. (Massachusetts General Hospital.)

FIG. 2

## Spasmodic Torticollis.

Head drawn backward and chin up, due to involvement of the right trapezius, probably with certain deep neck muscles, in addition to the manifest spasm of the sternomastoid. (Massachusetts General Hospital.)

cases in which the spasm is clonic in character over long periods of time, one is naturally not justified in speaking of the torticollis as of the form here under consideration. The exact type of spasm depends upon the degree of involvement of individual muscles and upon the number and position of the muscles affected. The picture, therefore, becomes a very complex one, and it is often a matter of extreme difficulty to determine the precise extent of the muscular involvement. The common form and that most closely related to the spinal accessory nerve consists of a clonic and tonic spasm of the sternomastoid of one side, often accompanied by a similar spasm, particularly of the upper portion of the trapezius. In this type the chin and face are moved away from the affected side and the head bent slightly backward, due to contraction of the sternomastoid. The coincident contraction of the upper part of the trapezius likewise leads to a turning and backward bending of the head away from the affected muscle, thereby increasing in a general way and supplementing the action of the sternomastoid. Other forms of spasm, as, for example, of the splenius, rhomboids, and deep muscles of the neck, do not now concern us, particularly since they bear no immediate relation to the spinal accessory nerve. It is, however, very important to bear in mind that although a torticollis may begin with the spinal accessory innervation, it is likely to spread to other muscles, thereby complicating the treatment, especially through surgical means. As suggested above, the extent and intensity of the spasm vary widely in different cases, and are unquestionably affected for the worse by unfavorable emotional conditions. Associated psychical disturbances are of interest particularly in their etiological relationship, but it should be borne in mind that a coincidental disturbance of a mental sort is perhaps particularly likely to arise in this distressing condition.

*Pathology.*—The attempt to locate the lesion productive of torticollis of the spasmodic type has been wholly unsuccessful. To say that it is a condition of irritation in the nerve nuclei is of small help, since we are ignorant of what such irritations may consist. The most reasonable hypothesis, as maintained, for example, by Brissaud and Oppenheim, is that the cerebral cortex, particularly in the area concerned in the movements of the neck, is primarily at fault. A more mechanical explanation is, for the present, at least, wholly undetermined.

*Prognosis.*—The outcome is always uncertain, but never entirely hopeless. The unexpected not infrequently happens. In one instance, in which the condition had become so grave that an operation had practically been decided upon, a preliminary sea voyage afforded entire relief. In general, however, the affection is stubborn, resistant of treatment, and shows a tendency to spread to other groups of muscles, oftentimes with a substitution of a practically chronic spasm for the alternating clonic spasms of the earlier stages. In such cases the condition becomes almost unbearable and a resort to most radical methods of treatment is justified.

*Treatment.*—As in many affections the exact cause of which is not understood, treatment for spasmodic torticollis has been and remains largely empirical. It is not necessary, therefore, to enter into detail regarding the great number of drugs which have been advocated. Experience has unquestionably shown that, apart from their influence upon the nervous system in general, there is no agent that exerts the slightest specific action upon the spasm. The bromides and opium may be given, and as general sedatives

may have an effect, but few would venture the prescription of the opium preparations in the present state of our knowledge, unless as a last resort and under most unusual conditions. Drugs unquestionably play a very small part in the rational treatment of this affection. Electricity has been used in certain cases with good results. It has been suggested that in the application of the electrical current it is desirable to stimulate the sound muscles rather than those affected with the spasm in order to overcome, so far as possible, the overaction of the latter. However theoretically justified such treatment may be, it has relatively little practical significance. Hydrotherapy is, no doubt, useful as a general tonic measure, but it has no further significance.

The use of mechanical appliances is of interest, particularly from the fact that their effect in different cases varies widely. An arrangement by means of which the head is held absolutely fixed is never to be advised. Such constriction is extremely apt to excite the muscular spasm in an even greater degree, leading to a distressing situation for the patient, even although the head is mechanically prevented from moving. On the other hand, it has frequently been shown that a supporting apparatus which is suggestive in its operation rather than providing an absolute fixity, is often of great benefit. In one case under the treatment of F. B. Lund, a patient was remarkably relieved by a slight support at the back of the head, quite insufficient to control the muscular spasm forcibly, but enough to suggest the control through the patient's will. The common experience in these cases that in the midst of the most violent spasm the head may be restored to its normal position by gentle pressure applied to the chin is suggestive. The hint which these facts gives regarding rational treatment is that the mental side of the affection must always be seriously taken into account. The spasm is, in fact, semi-involuntary, at least in its earlier course, and should be treated accordingly. Following out this idea, the most useful treatment is certainly that which combines mental and physical means, and this is best attained through coördinative gymnastic movements. Such movements have as their underlying principle the possibility of substituting in a voluntary muscle a voluntary for an involuntary movement. The aim, therefore, is to educate the patient to again perform voluntarily movements over which he has lost control, or, otherwise expressed, to break up a bad neuromuscular habit. Systematic movements of the head are therefore advisable, and best carried out under the instruction of a skilled teacher. The positive results of this form of treatment are unquestioned in most cases, although they can rarely be regarded as wholly curative. A purely psychotherapeutic treatment in these cases is, on the whole, not efficacious, nor is it necessary. Hypnotism, particularly, is undesirable, certainly as a general procedure. A wide range of surgical operations has been suggested and surgery would appear to be at first sight clearly indicated. Results, however, have shown that surgical intervention is rarely curative and nearly always discouraging in its results. The reason for this is two-fold: first, that a surgical operation does not relieve the cerebral condition which undoubtedly lies at the basis of the spasm; and, in the second place, that more than one muscle is apt to be affected, with a strong tendency to spread to still other muscles or groups of muscles. Various operations have been suggested and carried out with a certain measure of success. Simple resection of the spinal accessory nerve is of small value, owing to its rapid regrowth. Resection

with evulsion is a more rational procedure. The operative procedure may go farther and resect upper cervical nerves if the increasing spasm demands. The muscles themselves may also be cut, thereby diminishing the strength of their contraction, and even more radical operations on nerves and muscles of the various structures about the neck may in rare instances be advisable. The results of operations have been such, however, that the unpleasant suggestion arises that the section of one nerve with cessation of spasm of the muscle to which it goes rather increases the tendency to spread. Whatever value such operations may have it is clear that their mental effect is an important adjuvant and that no operation should be undertaken which does not seriously take this fact into consideration. If the affection is primarily psychogenic in origin it necessarily follows that operation in itself is unlikely to effect a complete cure.

F. T. Murphy,[1] from the Massachusetts General Hospital clinic, has recently reported a case, in which there has been no return of the spasm for six months, operated on by a method suggested in 1890 by Keen, consisting in the section of the spinal accessory and posterior divisions of the upper cervical nerves. The operation is a tedious one, and requires the use of electricity to demonstrate the individual nerves, but in Murphy's opinion is a more useful one than others which have been attempted for the relief of this condition. Murphy also draws attention to the fact that those cases in which there is a spasmodic contracture of a more or less fixed sort are more amenable to surgical treatment than those in which the movements are clonic in character, resembling true tics. This distinction cannot, in most cases, be sharply drawn, but should nevertheless be useful in determining upon the advisability of operation in a given case.

## HYPOGLOSSAL (TWELFTH) NERVE.

**Anatomical Relations.**—The hypoglossal nerve is purely motor in function, and supplies both the extrinsic and intrinsic muscles of the tongue with the exception of the glossopalatini and the geniohyoids. The nucleus of origin of the axones of this nerve lies in the dorsal portion of the oblongata toward the lower portion of the fourth ventricle. The fibers pass ventrally, emerging from the oblongata between the olives and the pyramids, thence passing forward to their termination in the muscles in and about the tongue, establishing connections with the upper cervical ganglion of the sympathetic, with the vagus and the lingual nerves.

**Etiology.**—This nerve is very commonly affected in disease involving the bulb, notably in the various forms of bulbar paralysis, acute and chronic. In its central course from the lower portion of the cortical motor area to its secondary nucleus in the oblongata also it is not infrequently involved. Although not particularly noticeable on account of the action of the nerve of the opposite side, one hypoglossus is ordinarily affected in cerebral hemiplegia. This lesion, however, is less conspicuous, because unaccompanied with atrophic changes of the tongue. This fact, together with unchanged electrical reactions, is an important and simple means of differentiation between lesions of the central and peripheral neurone, being merely an

[1] *Boston Med. and Surg. Jour.*, 1908, clix, 865.

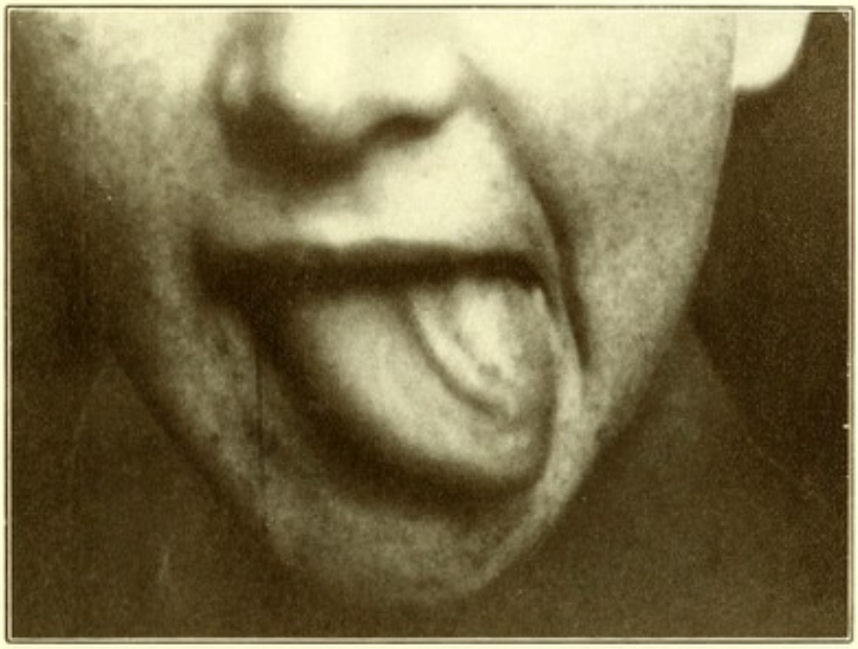
example of a general principle. Lesions in the oblongata itself, however, naturally involve the cells of origin of the peripheral axones and lead to a characteristic atrophy of the tongue. Lesions external to the oblongata itself, as, for example, meningeal exudates, tumors, or hemorrhages at the base of the skull, may involve the nerve in its peripheral course, but usually in conjunction with others, and particularly the vagus. Aneurism of the vertebral artery and syphilitic processes may involve the nerve, but these are rare occurrences. As of other cranial nerves, an isolated neuritis is rare, although such a condition has been described by various writers. Congenital atrophies of the tongue, lingual hemiatrophy, and degenerations which occur in certain central diseases of the nervous system should also be mentioned in this connection.

**Symptoms.**—Disease of one or both hypoglossal nerves gives rise to signs not easily mistakable and of much diagnostic importance. These signs are perhaps particularly noticeable when the lesion involves but one nerve. As the tongue lies on the floor of the mouth there may be no deviation of its tip, but if moved, the tendency is for it to be projected toward the paralyzed side. The explanation of this constant phenomenon is not simple, owing to the complicated relations of the tongue muscles. The attempt to move the tongue within the mouth cavity is imperfect toward the diseased side, which naturally interferes somewhat with the contact of the tongue on that side with various portions of the mouth cavity essential to proper enunciation and mastication. This defect, however, is so far compensated by the normal side that it leads to slight inconvenience. The defect becomes more apparent in the attempt to thrust out the tongue. This can be accomplished if but one side is paralyzed, but if the tongue is protruded the tip points strongly toward the affected side, its raphé describing a semicircle with its convexity toward the sound side; this deviation is due to the loss of contraction of the genioglossus. When the paralysis of both nerves is complete the tongue remains helpless and immovable in the mouth, and it becomes impossible for the patient to speak except in a most imperfect way. Mastication and deglutition are likewise interfered with through loss of action of the tongue in collecting and passing on the food. This condition is met with in the late stages of progressive bulbar paralysis, and is described under that heading. There is no sufficient evidence, as maintained by Gowers, that the muscles of the lips are innervated through the hypoglossal.

Atrophy is the constant accompaniment of peripheral lesion of this as of other motor nerves. The tongue loses its normally smooth appearance, and is thrown into folds and ridges due to the wasting of the underlying muscle bundles. To the touch it is soft and non-resistant, shows alterations in electrical reaction, and is pervaded by a fibrillary tremor.

**Treatment.**—The treatment of paralysis of the tongue, either of the spastic or atrophic type, lies almost wholly in that of the underlying condition. Electricity applied to the atrophying tongue muscle is a practical impossibility. In many conditions of unilateral disturbance of the tongue the discomfort is not sufficient to call for treatment, even if it were possible, and in the sudden or gradual involvement of the nerve from bulbar or more peripherally lying disease the affection of the tongue constitutes a relatively insignificant part of the clinical picture and demands no special treatment. The partial resection of the nerve which has lately been practised in facio-hypoglossal anastomosis for facial paralysis leads naturally to immediate

# PLATE XXXII



Unilateral Atrophy of the Tongue.

Protrusion toward the affected side, associated with movement of lower jaw toward the same side, owing to coincident involvement of the motor branch of the left fifth nerve. From a case of poliencephalomyelitis.

disturbance in its function characterized by a tendency to atrophy and slight speech defect, but this is quickly overcome, with complete restoration of function. The necessary injury to this nerve, therefore, is not to be regarded as a sufficient contra-indication to this operation.

**Spasmodic Affections.**—The occurrence of spasm in the muscles supplied by the hypoglossal nerve is common in association with more general spasmodic affections, as, for example, epilepsy, hysteria, and chorea. The impossibility of controlling the tongue observed in general athetosis, often leading to a complete loss of power of articulate speech, is a somewhat analogous condition, but is not properly included under spasmodic affections. Much more rarely the tongue muscles are affected with spasm which either does not extend beyond them or only in the immediate neighborhood. Such spasms may be either tonic or clonic in character. If tonic, the tongue is apparently reduced in size and is pressed against the teeth or other parts of the mouth cavity. Under these conditions speech and swallowing are affected. Associated or independent of these tonic spasms a clonic condition of the tongue muscles may be developed. Under these conditions the tongue is rapidly thrust out or withdrawn, or makes various involuntary excursions within the mouth cavity. Such spasms may be either bilateral or unilateral in distribution, the latter being less frequent. The relation of these cramp forms to various speech defects known as stammering and stuttering is of interest. Under certain conditions the difficult speech is apparently induced by the incapacity of the person to move the tongue properly, with a result that the word or syllable is not enunciated, or in an explosive manner. However complicated the ultimate explanation of such forms of speech disturbance may be, the relation to spasmodic affections in the distribution of the hypoglossal nerve should always be borne in mind. The muscle spasm or tic is rarely constant, but occurs rather in the form of attacks, varying widely in frequency and not always ceasing during sleep. The causes are somewhat vague, but may in general be attributed to sources of irritation in the neighborhood, on the basis of a neuropathic tendency. The relation of the mental state to cramp attacks is undoubtedly important. They may, for example, be aroused by emotional disturbances of one or another sort. The *prognosis* is, on the whole, favorable, even though the tendency to spasm may have existed for years. The *treatment* consists essentially in the removal of any exciting cause so far as it may be discovered, and psychotherapeutic procedures, with such muscle training as is possible, as indicated by the individual case. Drugs and electricity play no essential part in the treatment.

# CHAPTER XIV.

## PARALYSIS AGITANS. CHOREA. CHOREIFORM AFFECTIONS. INFANTILE CONVULSIONS.

By DANIEL J. McCARTHY, M.D.

### PARALYSIS AGITANS.

**Synonyms.**—Shaking palsy; Parkinson's disease.

**Definition.**—Paralysis agitans is a disease of the nervous system characterized by muscular rigidity and muscular weakness, with which symptoms is usually associated a definite kind of tremor, and on which are more or less dependent a deliberation in initiating and a difficulty in carrying out active movements, a characteristic facial expression, a peculiar attitude, and a distinctive gait.

**Historical.**—The disease was first described as a clinical entity in 1817 by Parkinson, who, in a thorough account of the affection, clearly separated it from the group of diseases variously designated St. Vitus' dance, chorea Sanctum Viti, epilepsia saltatoria, ballismus, skelotyrbe, etc. The condition was first sharply separated from multiple sclerosis by Ordenstein, under the direction of Charcot.

**Etiology.—Frequency.—Age.**—The disease does not seem to be at all rare in America. According to German statistics[1] there were 37 typical cases among 6000 nervous patients. Although cases of paralysis agitans, most of them doubtful, have been reported before the age of thirty years, and even before twenty years, in the majority of cases the disease does not make its appearance in an individual under forty years of age. Gowers found the average of onset to be fifty-three years in males and fifty-one years in females. Cases beginning as late as the seventy-fourth year have been reported (Gowers).

**Sex.**—The disease is about twice as frequent in men as it is in women (73 males to 42 females, Gowers).

**Heredity.**—The effect of heredity can best be judged by the figures of Gowers and of Berger, who, in 15 per cent. of their respective series, found a history of the disease in more than one member of the family. Five of 19 of Wollenberg's patients gave a family history of nervous or mental disease.

Occupation and station in life seem to have little or no influence in determining the onset, and in the majority of cases the disease does not seem related to any definite determining factor. A direct exciting cause can be traced only in about one-third of all cases (Gowers). The influence of the emotions is certainly an important etiological factor in a group of cases.

[1] *Eulenburg's Real-Encyclopädia*, first edition, Berger.

Gowers emphasizes the importance of fear or fright as an antecedent circumstance, and reports cases in which such emotion was apparently causative. Anxiety and worry may be mentioned as other predisposing emotional states.

**Trauma.**—The physical influence of trauma, apparent from the attendant psychic concomitants, is well recognized as a determining cause. Walz's analysis[1] showed in 26 cases, general concussion in 6, wounds (stabs and cuts) in 7, burning and freezing in 1, sprains, twists, and fractures in 4, and contusions in 8. Some connection between the part injured and the locality of the earliest symptoms is almost unexceptional.

Trauma may determine the spread of a paralysis agitans already begun. Walz believes that the disease can appear only in a person whose nervous system is deteriorated. "While the truth of such a theory cannot be denied, it cannot be proved, because it is rarely possible to demonstrate any predisposition in the patient" (Bailey[2]).

In a group of cases there is a history of overexertion of the muscles, especially of those first affected by the disease. Exposure to cold seems to be a determining cause in another group. The influence of the infectious fevers is probably small. Cases have, however, been reported after syphilis, malaria, and typhoid fever.

**Pathology.**—"Chaotic" is the term that adequately expresses our present knowledge of the pathology of paralysis agitans. Pathological findings have been manifold, but a definite lesion occurs so inconstantly that at the present time it may be said that the disease has no definite pathology. An exception to this statement seems to exist in the changes in the muscles found by Camp,[3] to whose excellent article on the subject the reader is referred. Whether these changes in the muscles are primary or secondary to disease of the brain, spinal cord, ductless glands, or other parts of the organism, or due to the action of a toxin, or induced by the tremor, the future alone will decide.

A peculiar difficulty at the outset of any investigation to determine the pathology lies in the fact that the changes described are not specific, and occur in a variety of conditions. It is difficult, for instance, to differentiate the various overgrowths of connective tissue and neuroglia in the spinal cord from similar changes found in old persons who never have suffered from the disease. Moreover, there is a group of cases which at autopsy shows in the cerebral nervous system no pathological changes whatever. Even the experienced eye of Oppenheim[4] could detect in one case only a "questionable" staining of the column of Goll. Most of the microscopic changes in the brain and spinal cord which have been recorded were reported when the condition was not well recognized, and when the tremor was confused with that of other diseases, especially multiple sclerosis; for instance, one of the cases reported by Parkinson himself was probably a case of multiple sclerosis. Tumors of the brain, sclerosis, cysts, and ependymitis have been reported in connection with the condition, but in these cases either the occurrence of paralysis agitans was a coincidence, or the tremor was not typical of the disease. A "symptomatic" paralysis agitans has

[1] *Vierteljahrschrift f. gerichtliche Med.*, 1896, xii, 323.

[2] *Diseases of the Nervous System Resulting from Accident and Injury*, second edition, 1908.

[3] *Jour. of Amer. Med. Assoc.*, 1909, xlviii, 1230.

[4] Quoted by Sanders, *Monatsschr. f. Psych. und Neurol.*, 1898.

been written about, but the use of the term "symptomatic" and the reason of occurrence of the tremor have not been made clear. At all events, the rarity of a specific gross macroscopic finding would preclude our regarding such finding as other than accidental. The fact that macroscopic findings occur in connection with the disease must, however, be taken into account if we should regard it simply as a symptom complex.

The microscopic changes described affect chiefly the spinal cord, and here the changes reported are many and difficult to separate from those of senility. Many authors agree as to an excess of neuroglia tissue in the cord in these conditions, but differ as to its distribution and its significance. Redlich,[1] for instance, found the chief seat of collections of glial tissue to be in the anterolateral columns, especially around the bloodvessels, while Sanders[2] finds that the neurogliar proliferation is more marked in the gray matter than in the white. Sanders especially emphasizes this as being a different condition than that found in senility. He found not only a comparatively greater increase in the amount of neurogliar tissue in the anterior horn, but also numerous spindle cells ("spinnenzellen") in cases of paralysis agitans and cases of senility with a marked tremor.

Dana describes a very moderate increase in the connective tissue within the cord. Tract degenerations have been reported, but they were absent by the Marchi method of staining in all of 14 cases examined by Camp.

Vascular changes reported include arteriosclerosis of the vessels of the cord; miliary hemorrhages into the spinal cord; proliferation of the bloodvessels, which may be surrounded by a zone of leukocytes; miliary aneurisms of the spinal cord; varicosities of the veins in the medulla, with small areas of softening in the oculomotor roots and pyramidal tracts. Numerous authors have emphasized the fact that in the arteriosclerosis found the finer capillaries were more affected than the larger.

Changes in the central canal of the cord, for instance, occlusion and widening, and the presence within it of leukocytes, have been reported; likewise the presence within the spinal cord of amyl[illegible] bodies.

Changes have been described in the cells of the spinal cord, in those of the anterior horn, but also those of Clarke's columns. Those in the former include pigmentation, chromatolysis, loss or displacement of the nucleus of the cell, atrophy of the cell, vacuolation of the cell, tumefaction of the nucleolus, etc. Dana describes especially pathological changes in the dendritic process of the anterior horn cells, with consequent diminution in number. This phenomenon was not observed by Camp in any of fourteen cases. Pigmentation of the cells of Clarke's columns has also been described.

Changes have been reported in the Betz cells in the cerebral cortex in a few cases, in connection with changes in the cells of the anterior horns of the spinal cord.

Various degenerations and scleroses have been noted in the peripheral nerves, but these are by no means constant.

In view of the striking clinical phenomenon referred to the muscles in paralysis agitans, it is not strange that a pathological basis of the disease should be sought in the muscles themselves. Certain changes were reported as early as the days of Skoda (1862), who found a fatty degeneration among

[1] *Jahrb. f. Psych.*, xii, 385
[2] *Monatsschr. f. Psych. und Neurol.*, 1898.

the deviations from the normal in the muscles. Numerous observers have since reported atrophy of the muscle fibers, hypertrophy of the same, hyaloid and fatty degeneration and proliferations of the nuclei of the sarcolemma, with smallness of these nuclei. One would naturally expect that some of these might be dependent solely on old age, yet, according to recent observers, the findings in the muscles are distinctive and can be distinguished from changes due to senility.

The findings of Camp, present fairly regularly in a series of nine cases, are quoted: "Many of the muscle fibers are swollen, and in cross-section were round instead of having the normal polygonal shape. There was a marked increase in the number of the nuclei within the fibers, many of which were smaller, denser, and rounder than the normal. These nuclei were frequently in pairs or in long chains. In several places the fibers showed atrophic changes, and in these cases, usually of long-standing disease, there was an overgrowth of the connective tissue. In many changes the fibers had a hyaloid appearance and the longitudinal striations were unusually well marked, and in many of the fibers there was a tendency to longitudinal cleavage. In one case trichina spiralis was found in the muscles, and in this case, in addition to the above described changes, there was an intense interstitial myositis and also a discoidal degeneration of the muscle fibers. Muscle spindles were hard to find in the sections from any of the cases, but in those examined the muscle fibers within them showed similar changes to those surrounding them. The nerve fibers within the spindle were normal, and the intramuscular nerves showed no degeneration when stained with the Weigert hematoxylin stain. These pathological changes were not all of the same intensity in all the muscles nor in different parts of the same muscle." On account of the statement expressed in the last sentence, little stress can be laid on the fact that in some cases of paralysis agitans pieces of muscle have been examined with negative results.

**Ductless Glands.**—The thyroid was formerly the ductless gland most viewed with suspicion as to a possible role in the causation, and of late there has been a tendency to hold lesions of the parathyroid gland responsible. It is difficult to separate the changes found in the ductless glands from those due to old age, and from changes due to diseases other than paralysis agitans. Here, again, we are confronted with the fact that in certain cases of paralysis agitans the gland assumed to be the seat of the disease may show nothing pathological. Thus, Alquier[1] examined the thyroid and adrenal glands from two cases of paralysis agitans, and found them negative. Camp, working on an hypothesis of Lundborg and of Berkley,[2] examined parathyroids from two cases of the disease, and found them pathological, the change consisting essentially in a peculiar infiltration with fat, especially in relation with the bloodvessels. That this is not specific is proved by the observation of R. L. Thomson,[3] who, after a comparison of the parathyroid glands in 9 cases of paralysis agitans with those of 39 cases of individuals dead of other diseases, found that "the parathyroid glands in individuals dying with this disease (paralysis agitans) present no change either in number, size, position, or histological structure that would serve to distinguish them from the parathyroid glands in individuals dying from other diseases."

[1] *Thèse de Paris*, 1903; *Revue Neurolog.*, 1904, p. 438.
[2] *Medical News*, 1905.
[3] *The Journal of Medical Research*, 1906, xv, 399.

Parhon and Goldstein[1] have noted a marked preponderance of hematoxylinophilic cells in the hypophysis of a woman who had suffered with Parkinson's disease.

**Pathogenesis.**—Many theories as to the pathogenesis and as to the mechanism of the production of the symptoms have been advanced, but in the absence of a definite anatomical or demonstrated physiological basis, these are little more than speculation. Gowers offers a somewhat hazy explanation by referring the tremor to a derangement of the cortical centres concerned in movements that are naturally brought about by fear. Numerous authors hold that the disease is due to an aging of the nervous system before the rest of the body, and point to the fact that the lesions found resemble those of senility. While the theory has some evidence to sustain it, it does not explain the rigidity which is characteristic of the disease, nor are the changes in the muscles explained. Dana's theory that the dendrites of the anterior horn cells are at fault has some degree of plausibility, although the lesion is not always demonstrable.

The theory that the disease depends on changes in the muscles has much to commend it, and it is possible that the fundamental lesion is some change in the muscle spindles. This latter assumption is rendered the more plausible by the fact that the muscles most severely affected by the tremor are those which contain the greatest abundance of muscle spindles, and that the tremor does not develop in muscles not supplied with the spindles. The subject is, however, rendered complex by our lack of knowledge as to the function of the muscle spindles. Even if we regard them as sensory organs concerned in muscle tone or in muscle sense, it is difficult for us to understand how lesions involving them account for the rigidity and tremor of the muscles, unless we at the same time ascribe some inhibiting influence over these organs by the cells of the anterior horn of the spinal cord. The whole subject is rendered still more complex by the fact that myotonia congenita, a disease characterized by tonic cramp of the muscles on attempt at voluntary movement, presents the pathological changes in the muscles found in paralysis agitans, except that the muscle spindles remain uninvolved. That the disease is an abiotrophy of the muscles analogous to that present in the muscular distrophies is held by some authors.

**Symptoms.**—As a rule, the disease is ushered in insidiously. There is first a prodromal period characterized by various sensations of heat, weakness, numbness, rheumatoid pains, headache, local œdema, and sensations simulating girdle pains. The symptoms usually affect the part of the body first to be affected by the disease. This period is variable in duration and is sometimes absent, the disease appearing acutely. Palz[2] reports a unique, but not typical case, in which vitiligo was the first symptom.

Tremor is the initial symptom in about two-thirds of the cases. At times it is entirely absent (paralysis agitans sine agitatione vel tremore). At first it occurs only intermittently, and usually affects the small muscles of the hand. The fingers, especially the index finger, are alternately flexed and extended at the metacarpophalangeal joints, while the thumb moves to and fro laterally at the same time that there is flexion and extension, the whole resultant movement being somewhat of a circumduction.

[1] *Revue Neurolog.*, 1907, No. 22, p. 1230.
[2] *Schmidt's Jahrbuch*, 1905, cclxxxviii, 228.

This movement, in conjunction with the movement of the index finger, produces a "pill-rolling" type of tremor, which is very characteristic. This is usually associated with a flexion and extension of the wrist-joint and a pronation and supination of the forearm. The upper arm is affected less frequently than the forearm. In the legs the calf muscles are most frequently affected. The muscles of the neck may or may not be affected, in which case nodding or rotary movements of the head are produced. The tongue and the muscles of the head and face sometimes share in the tremor; rarely, if ever, the orbicularis palpebrarum. At times the muscles of expression and of mastication are involved. The vocal cords and the muscles of the chest and of the abdomen are affected but very rarely.

The movement varies much as to range, and has a rate of about four and eight-tenths to seven oscillations per second (Gowers); it lessens in frequency as it increases in range; the fine tremor of the early stages is often distinctly quicker than the coarser period of the late period (Gowers). The rate of tremor in the arm and in the leg may vary much in the same individual.

The tremor continues during rest, is somewhat diminished by voluntary movement, and, as a rule, ceases during sleep and narcosis. It is increased by mental excitement, and may be somewhat controlled by passive movement, or momentarily by the concentration of the patient's attention, as, for instance, when he fixes an object with his eyes. When hemiplegia occurs in an individual suffering with paralysis agitans, as a rule, the tremor ceases in the paralyzed members. This cessation may, however, be transitory.

The one symptom of the disease which is cardinal, and which, with its associated symptoms, constitutes the disease even in the absence of tremor, is permanent muscular rigidity. This rigidity is at once apparent to the examining hand, and is responsible for the attitude and gait of the patient, the delay in executing voluntary movements, the facial expression, and the contractures. It affects especially the muscles of the neck and the vertebral column; but is also seen in the extremities and face. The rigidity usually is marked only when the disease has advanced rather far, but, as Wollenberg remarks, "it may be present subjectively to the patient very early, manifesting itself by a slowness and a difficulty in executing active movements." Associated with the rigidity is a more or less well-marked muscular weakness, which, however, never reaches the stage of total paralysis. This weakness, with or without a sense of stiffness, precedes tremor in about one-fifth of the cases, and occasionally is conspicuous with tremor from the first. This weakness may be manifest subjectively at a very early stage; in fact, it may be considered as one of the prodromal symptoms. Objective weakness, which is manifested, for instance, by the inability of the patient to make much of an impression on the scale of a dynamometer, must not be mistaken for the apparent weakness associated with the slowness and difficulty in executing active movements. The latter seems more or less dependent on muscular rigidity, and may consist in the slowness in the movement itself, or in a delay in the transmission of the voluntary impulse to the muscle. The delay may manifest itself even when the movement is executed with fair rapidity (Gowers).

In addition, in consequence of the muscular rigidity, there is a resistance to passive movement. This becomes greater as the disease advances, and may amount to nearly absolute fixation of a part, as, for instance, the head.

This rigidity differs from that seen in hemiplegic and paraplegic contractures, in that it is permanently present and is not increased or called forth by the movement that tests it. Owing to the rigidity, the upper part of the body is bent forward, and this is in consequence a forward, in rare cases a lateral or backward, position of the head on the trunk. J. A. Sicard and L. Alquier[1] report deviations of the spinal column in 12 out of 17 cases of paralysis agitans. These they believe to be due to the muscular rigidity; they may consist of kyphosis, lordosis, scoliosis, or a combination of these deformities. The arms are slightly abducted and the elbows somewhat flexed. The wrists are usually somewhat extended. There may be an ulnar position of the hand resembling that seen in arthritis deformans. The position of the fingers varies; in many cases they are slightly flexed at all joints in the position that they naturally assume during rest; often they are flexed at the metacarpophalangeal joints and extended at the others, from the preponderant contraction in the interossei. There may even be overextension of the last phalanx, most marked in the thumb, perhaps because the tip of the thumb is pressed against the first finger. The rest posture is especially frequent when rigidity preponderates over tremor. When the interossei contract in the tremor the "interosseal posture" is generally very marked. Usually the rigidity can be readily overcome, but in extreme cases the contracture of the interossei may go on to the degree of permanent shortening, so that the metacarpophalangeal joints cannot be passively extended beyond a right angle, just as in contraction of the palmar fascia. Occasionally only one finger (as the index) is thus affected. In the legs the rigidity involves chiefly the hip-joints and knee-joints, causing slight flexion of each and adduction of the thighs. It may extend to the feet and even cause talipes equinovarus and distortion of the toes—extension of the first and flexion of the other phalanges, so as to cause a claw-like deformity. Permanent contraction of the muscles is very rare, but the writer has known each foot to be fixed in inversion.

In consequence of the peculiar stooping posture of the patient and the lack of spontaneity in the movements, the term "statuesque" has been applied to the attitude.

The muscles of the face share in the rigidity, and show a lack of expression which has been compared to a mask. The muscles of the eyes are but little if at all affected by the slowness of movement, while nystagmus rarely, if ever, occurs as a symptom. The patient may, however, show a slowness in initiating a movement of the eyes from one point to another. A peculiar staring expression in many cases is caused by the partial or almost complete absence of winking, due to rigidity of the orbicularis.

The gait is characteristic. The patient, if seated, arises with difficulty; the first few steps are slow and shuffling, then the steps become progressively shorter and quicker, until the patient has to come to a stop to prevent his falling forward. This characteristic gait (festination or propulsion) was described by Parkinson. At times the patient, in attempting to walk, makes short steps toward one or the other side (lateropulsion), or he may make the short steps backward instead of forward (retropulsion). An individual patient may show both propulsion and retropulsion, which may be demonstrated by gently pushing the patient forward or backward, or by making slight traction

[1] *Nouvelle Iconographie de la Salpêtrière*, 1902, xv, 377.

on his clothing. The gait was formerly thought to be due to the fact that in consequence of the forward position of the upper part of the patient's body he was "running after his centre of gravity." However, many patients whose heads are turned backward show propulsion, and vice versa. The explanation of the phenomenon is probably to be found in the difficulty in overcoming the rigidity of opposing muscle groups and in the slowness of transmission of controlling impulses to the muscles.

The patient's voice is commonly high-pitched and piping, and lacks proper modulation. The patient has difficulty in shaping the first word of what he wants to say, but the succeeding words are enunciated more and more quickly. Müller[1] and also Rosenberg[2] have reported cases associated with tremors of the vocal cords. Cisler, quoted by Pelz, describes the vocal cords of 75 per cent. of his patients who were affected with the disease as approximating the cadaveric position. In very advanced cases the speech is a low "mumble," difficult, if not impossible, to understand.

The tendon reflexes may remain normal, or may be increased. Oppenheim and Frank describe a false ankle clonus due to tremor. Oppenheim asserts that a true foot clonus is sometimes present, but that the presence of Babinski's sign is to be regarded as an indication either that the paralysis agitans is symptomatic, or that some other disease exists. The paradoxical phenomenon of Westphal is often present; this consists in a long-continued contraction of a muscle on approximation of its ends; and is seen typically in the contraction of the tibialis anticus muscle on flexion of the foot.

There is a delay in the latent period of muscular contraction, and a lessening of the irritability of the muscles and nerves to the electric current (Borgherini[3]). As a rule, the function of the sphincters is well retained; occasionally incontinence of urine is present. Nystagmus is exceedingly rare. Oppenheim reports a paralysis of convergence of the eyes, which he ascribes to a probable tonic rigidity of both abductors. The same author also reports the presence of von Graefe's sign. Moczutkowsky[4] lays stress on the fact that when the patient's forehead is folded, the return to smoothness is very gradual.

Objective sensory disturbances do not belong to the symptomatology of the disease, although they may occasionally be present. An exception to the statement is the fact that in certain cases the surface temperature of the body may be increased.

Beyond a tendency on the part of some patients to adopt a whining and complaining manner, the mind remains very clear; in fact, the good nature and complaisance of most of the patients, in spite of the severity of the symptoms, is a matter of common observation. Dementia may, however, complicate a case of the disease. Subjective sensations include vague pains, vertigo occasionally, and sensations of heat or of cold.

Muscular atrophy does not form a part of the clinical picture; a slight degree may be present in long-standing cases. Trophic and vasomotor disturbances may be present; and the skin of the hand may be smooth, shiny, and swollen, the appearance being that of the "main succulente." The excretion of sweat may be increased, and salivation and drooling are

[1] *Charité Annalen*, xii, 267.
[2] *Berl. klin. Woch.*, 1892, No. 31.
[3] *Wien. med. Woch.*, 1890.
[4] *Reference in Neurolog. Centralbl.*, 1897.

present. The salivation is probably due, at least in part, to delayed deglutition.

The urine may or may not present an excess of phosphates. The sulphates have been found diminished. The total quantity of urea, as a rule, does not vary much from the normal, but it varies in proportion with the total amount of nitrogen. The urinary toxicity is normal (Vires).

**Forms.**—The modifications of the tremors and distribution of the rigidity have given rise to various clinical types of the disease, the characteristics of which have been dwelt on by certain authors. Hemiplegic, monoplegic, paraplegic, and crossed forms have been described. Charcot writes of a "forme fruste," in which the tremor is very slight and may be entirely absent. Entire absence of the tremor is not such a very rare occurrence. The rate of fiber tremor may very much exceed the usual number of produced movements. The character of the tremor may be altered by the influence of active movements, or it may even be elicited by these movements. Rigidity may be long delayed, in which case the tremor may be difficult to diagnose from that of hysteria or neurasthenia. Hyperextension of the limbs may replace the usual flexion, while a backward position of the head is not unusual. Various diseases (Basedow's disease, myxœdema, tabes dorsalis, hemiplegia) may complicate the disease and make diagnosis difficult. Spiller[1] has pointed out the similarity between arthritis deformans and paralysis agitans, and reports a case in which a tremor of the latter affection occurred in a case previously diagnosed arthritis deformans. A "forme rheumatismale" or "forme douloureuse" is spoken of by French authors. At times vasomotor or trophic phenomena (muscular atrophy, œdema, purpura) help to make up the clinical picture. Arterial hypertension has been reported.

**Diagnosis.**—In the advanced stages, as a rule, there is no other affection more readily diagnosed; occasionally, however, a case presents difficulty, as in one reported by Spiller, which had been considered arthritis deformans until a tremor appeared in one of the arms. Other tremors dependent on brain disease simulate those of paralysis agitans so closely that a group of symptomatic shaking palsies has been recognized. At times the tremor resembles that of Basedow's disease, a circumstance all the more puzzling because in certain cases paralysis agitans and exophthalmic goitre co-exist. In this case the former affection may be recognized by the presence of the muscular rigidity and associated symptoms. During the prodromal stages it is difficult or impossible to distinguish paralysis agitans from hysteria or neurasthenia. The mental make-up of the individual should be taken into account, and the possibility of paralysis agitans developing in neurasthenic or hysterical individuals should not be ignored. From the character of the pains in early cases of paralysis agitans, even tabes may be considered. The early loss of the tendon reflexes, especially the patella jerks in the latter affection, together with the other signs of the disease (loss of pupillary reaction to light, etc.), will serve to make the diagnosis clear.

Less easy in some cases is the distinction between paralysis agitans and multiple sclerosis. It is especially difficult in those cases of paralysis agitans in which the tremor is increased by active movements. The permanent character of the rigidity in the former affection and the nystagmus, sphincter

[1] *Univ. of Penna. Medical Bulletin*, 1904.

troubles, scanning speech, etc., of the latter condition will, however, be apparent. Senile tremor may simulate that of paralysis agitans closely; it is, however, not accompanied by rigidity, and is more apt to involve the head than is paralysis agitans. It develops, as a rule, in true old age, while the onset of paralysis agitans is in the presenile degenerative period. Oppenheim lays stress on the differentiation of paralysis agitans from a condition of paraplegia senilis dependent on arteriosclerosis of the brain and spinal cord, in which the body is held as in paralysis agitans, but in which true paralysis exists. In some cases the latter condition resembles a pseudobulbar palsy, and the diagnosis becomes difficult, as pseudobulbar palsy may complicate paralysis agitans.

Hereditary tremor and toxic tremors can be readily differentiated by the absence of rigidity.

At times it becomes difficult to determine whether a paralysis agitans following an injury is a true condition or simply a neurosis. Each case should be judged on its merits, but collateral symptoms of hysteria speaks against the genuineness of a complicating Parkinson's disease. Oppenheim points out that this neurosis is a persistent affection in itself, but lacks the progressive character of paralysis agitans. Finally, we must distinguish paralysis agitans from the choreas, and especially from posthemiplegic choreas, especially as Parkinson's disease may develop on the seat of a hemiplegia. The jerky, irregular character of the choreas is absent.

**Prognosis.**—The disease in itself does not seem to shorten life. In certain cases the symptoms tend to progress, both as to extent and intensity, while in others they remain relatively slight.

**Treatment.**—Little or nothing can be done to effect a permanent cure. All efforts should be concentrated to making the life of the patient as comfortable as possible. Suitable diet, bathing, exercise, and rest should be provided. The patient should be kept as quiet as possible, and should avoid all the health resorts likely to cause mental or physical excitement.

Electricity should be given as electric baths, especially the dipolar faradic (Oppenheim), otherwise it is of no service.

Friedländer[1] recommends active movements of the extensors, and also passive movements to relax the rigid muscles and to improve the power of their antagonists. This relaxation is well gained by allowing the various members of the body to fall against gravity. At first some difficulty in inducing the patient to relax sufficiently to let a certain part fall may be experienced. After a little experience in letting it fall has been gained, it should be raised against slight resistance. The movements should be practised several times during the day, but not for many minutes at a time. This treatment should extend over a long period of time.

Arsenic in the form of Fowler's solution is probably the best agent for maintaining the general tone of the patient.

Hyoscine hydrobromate (gr. $\frac{1}{200}$ to $\frac{1}{150}$ three times a day) or duboisine (gr. $\frac{1}{200}$ to $\frac{1}{150}$ three times a day), preferably hypodermically, have been recommended for the control of the symptoms, and in certain cases do exert temporary influence. Their use is attended with some danger, as they may give rise to symptoms of intoxication (vertigo, sense of pressure in the head, disturbance of vision, nausea, or dryness in the throat), which

[1] *Zeit. f. physikalische und diatätische Therapie,* November, 1907, p. 468.

is a signal for their temporary withdrawal. Neither of the drugs is a curative agent. The bromides do not seem to be of value.

Berkley recommends extract of parathyroid gland. All preparations of the gland should be kept on ice. "The initial dose of the powdered gland was gr. $\frac{1}{20}$, two to four times per day, preferably in capsules; larger doses appear to produce weakness, constipation, nervousness, and even an exaggeration of the symptoms of the disease. The first good effects in the cases treated were noted, as a rule, after fifty to seventy-five capsules had been taken, two or three weeks after beginning treatment."

Berkley's first report of the effects of the gland was on eleven patients, nine of whom were distinctly benefited, one practically cured. A later report[1] is based on notes or personal knowledge of 30 cases. Of these, 2 declined to continue the remedy, 2 had not been heard from, 5 denied any benefit, 3 showed temporary improvement only, and 18 were progressively benefited during the entire period in which they were under treatment.

"The benefit consisted in diminished rigidity, lessened pain, salivation cured, shaking diminished or cured, voluntary control of the muscles greatly increased, and restlessness or insomnia nearly or quite abolished" (Berkley). This testimony is borne out by Marogna,[2] who used the gland in a single case. Berkley does not assign to the extract of the parathyroid gland any specific effect in the treatment of paralysis agitans, but believes that it may exert an antispasmodic influence. Parhon and Urechie[3] report similar good effects from the use of a maceration of pituitary body in glycerin.

## ACUTE CHOREA.

**Synonyms.**—Sydenham's chorea; chorea minor; St. Vitus' dance.

**Definition.**—A disease occurring chiefly in children, due to the effect of an infectious agent, or its toxin, on the central nervous system, characterized by irregular, involuntary muscular contractions, resulting in movements of a purposeless nature, and associated with psychic manifestations.

**History.**—The term Chorea Sancti Viti was first used by Paracelsus, to designate an infection entirely different in its nature from that under consideration. In the sixteenth century, more particularly in middle Europe, epidemics of a hysterical dancing mania, described under chorea major, prevailed. The afflicted sought relief in religious pilgrimages to various shrines, most of all St. Vitus', whence the disease derived its name. The disease, however, was variously designated from other shrines, as St. John's and St. Anthony's dance. The infectious type of chorea as a clinical entity was first recognized and described by Sydenham. The use of the term was unfortunate in that it led to a confusion of the chorea of the type of Sydenham with the dancing epidemic above mentioned. This led to the use of the term chorea minor, to distinguish the infectious type from the original chorea major of the dancing epidemics of the middle ages. Much has been written on the subject since the time of Sydenham, and the various investigations led to the attitude that we are here dealing with a manifestation of some infectious agent upon the central nervous system.

[1] *The New York Medical Journal*, November 23, 1907, vol. lxxxvi, No. 21, p. 974.
[2] *Gazetta d. ospedali e della cliniche*, April 11, 1909, No. 43, p. 451.
[3] *Revue Neurolog.*, 1907, No. 22, p. 1230.

**Etiology.—Age.**—Chorea may develop at any time of life, but it is essentially a disease of childhood and adolescence, and is found most frequently in the later years of childhood. In Osler's analysis of 535 cases, 33 occurred in the first hemidecade, 228 in the second, 212 in the third, and 62 in the fourth. Infectious chorea is rarely seen before the fifth year.

**Sex.**—The disease is much more frequent in the female sex; according to Gowers the ratio is 3 to 1. In Osler's statistics, women are affected in the proportion of 2 to 1.

**Race.**—Mitchell called attention to the absence of chorea in the negro race, and Osler found only 5 out of 175 cases at the Johns Hopkins Hospital. The same author has noted the absence of chorea in the full-blooded Indian. It, however, may occur in the half-breed.

**Climate.**—An attempt has been made by Morris J. Lewis, in the study of cases at the Orthopædic Hospital in Philadelphia, to establish a relationship with climatic conditions (barometric and storm influences), and with apparent success. Other observers, however, working along the same lines, failed in establishing any such relationship. The frequency of attacks prevailing in the spring months can be attributed to other causes. Children are usually of poorer nerve tone and general nutrition after a winter of confinement and school work than at any other period of the year. The strain of school examinations also has an influence in children of a nervous, excitable temperament.

**Emotional Influence.**—One cannot fail to be impressed by the large number of patients with chorea who present a nervous, excitable temperament. There is, indeed, a certain type of child who might be referred to as of a choreic temperament. Such children, usually of the female sex, show an extreme condition of motor unrest. All of the movements are quick and extremely jerky. Under excitement the child is so fidgety, the movements of the arms and legs so jerky and apparently purposeless, as to constitute a condition of normal chorea. When such children have Sydenham's chorea, it is extremely difficult to see where the normal movements cease and where the manifestations of the disease begin. This is particularly true during convalescence. Weir Mitchell has called attention to a condition in adults, manifested by irregular movements, such as writing, etc., while under observation. On such a soil it takes but little intoxication of the nervous system, from a relatively mild infection, rheumatic or otherwise, to produce a well-developed case of chorea. In nervous children a decided emotion, such as fright, or excessive mental strain, may be the determining factor in the precipitation of an attack. It would be a mistake to consider emotional disturbance as a cause of a clinical syndrome running such a definite course as Sydenham's chorea. In highly emotional children hysterical manifestations or even fully developed major hysteria may complicate the clinical picture of chorea. While the association of the two diseases has nothing in common, there is a possibility of a hysterical child simulating Sydenham's chorea when there has been sufficient opportunity for observation of another case. Epidemics of chorea in schools may be explained upon this basis. Steiner, however, regarded the epidemic under his observation in Prague as a result of atmospheric influence, and excluded imitation.

**Heredity.**—The influence of a direct similar heredity in the production of chorea is very rarely seen. An unstable nervous system, inherited from ancestors suffering from epilepsy, hysteria, insanity, etc., affords a good soil for the development of various functional neuroses. Such an inheritance is, however, not sufficiently frequently met with as to be considered as an essential etiological factor in the development of chorea.

**Pathogenesis.**—This has been the subject of much discussion, and has led to extensive statistical investigation within the last sixty years. Like so many other organic and functional nervous diseases of unknown origin, it was for a long time, and is still in many text-books, attributed to fright, rheumatism, exposure to cold and wet, etc. Even the early incidence of the disease does not save it from being attributed to syphilis. As a result of careful study and observation, Sydenham's chorea, as differentiated from habit chorea, organic chorea, and electrical chorea, is now well recognized as a syndrome, the result of the actions of an infectious toxic agent upon the central nervous system. Wollenberg designated the disease as infectious chorea as differentiated from degenerative chorea and other choreiform conditions. It must, however, be admitted that a satisfactory conclusion has not yet been reached concerning the specific infecting agent or toxin. Nearly all the investigations so far recorded are either controversial or disputatious in character, and for that reason lack scientific value. Instead of proceeding from a position of complete or partial ignorance as to the cause of the disease, and advancing with an investigation along scientific lines, practically all of the investigators continue along the lines of the discussion started in the middle of the last century, as to the relationship between chorea and rheumatic fever. In nearly all of these studies, both clinical and pathological, the authors have assumed a position (necessarily one of theory), and have then written or investigated in support of their position. Were it definitely established that a specific organism was the cause of acute rheumatic fever, there might be some hopes, sooner or later, of solving the question by these means.

The evidence as presented is far from conclusive as establishing a distinct causal relationship between rheumatic fever and chorea. From a clinical standpoint, the statistics vary between 1.6 and 85.5 per cent. In the articles however, in which the subject has been studied from an unbiassed standpoint, in which there is every evidence of careful investigation and record by competent observers, there is a fair uniformity in the statistics. Thayer, Osler, Wollenberg, etc., give statistics varying from 20 to 25 per cent., and this even with a wide latitude as to what constitutes rheumatic fever. These statistics, as presented by Thayer, are of especial interest on account of the relationship between rheumatic fever and organic heart disease. Thus, 21.6 per cent. of these cases show an association with rheumatic fever, and 25.4 per cent. show evidence of organic heart disease.

When an analysis is made with the autopsy records, another difficulty presents itself. Uncomplicated chorea is looked upon as having a very favorable progress. The mortality as given by the British collective committee is 2 per cent. Sinkler found sixty-four deaths in Philadelphia in seventy-four years. Taking into consideration, therefore, the frequency of the disease, it necessarily follows that the mortality is very low. The most severe cases, if uncomplicated, usually go on to a favorable termination. The mortality of chorea gravidarum is given as only 20 to 25 per

cent., and this, be it remembered, is a very rare manifestation of the disease. Admitting, therefore, the complication of rheumatic fever in 25 per cent. of cases, it would not be at all surprising if an analysis of the deaths should show a large percentage of cases in which this complication was present.

Other septic processes, which, like rheumatic fever, may have an endocarditis or other serous membrane as a complication, may also find a place in these statistics. When, therefore, Poynton and Holmes take the position that chorea, to use the expression of Duckworth, is a "cerebral rheumatism," and support this position by finding a microörganism in six cases, this position can only be considered as scientifically correct for a single group of cases. In at least one of these cases, however, the colon bacillus was found in the blood at autopsy. Little stress was laid upon this fact, and he used this case as an argument in favor of his position. A more reasonable conclusion from the evidence of these six cases would be that when a severe type of chorea complicates a rheumatic infection, it is very likely to prove fatal. The other investigations which have been reported in cases of chorea are as follows:

Sturges: Antecedent rheumatism, 26.5 per cent.; coincident rheumatism, 4.45 per cent. Dickinson: Antecedent rheumatism, 26.75 per cent.; coincident rheumatism, 7 per cent. Peacock: Antecedent rheumatism, 28 per cent.; coincident rheumatism, 7.66 per cent. Owen: Antecedent rheumatism, 2.6 per cent.; coincident rheumatism, 8 per cent. Ogle: Coincident rheumatism, 10 per cent.

Wollenberg: Of 50 cases, antecedent rheumatism, 13 (26 per cent.); joint pains increased this to 38 per cent. Of 51 cases, coincident rheumatism, 2 (3.9 per cent.); 1 doubtful.

Steiner: Of 252 cases, coincident rheumatism, 4 (1.6 per cent.).

Pryor: In 92 cases of antecedent or coincident rheumatism, 5 (5.4 per cent.) had endocarditis.

Osler: Of 110 cases, organic heart disease in 54 (49.99 per cent.); 40.7 per cent. of these had articular pains. Of 295 cases, subacute or chronic rheumatism, or pain which might be regarded as rheumatic, in 62 (21 per cent.).

Hughes: Of 104 cases of chorea, 86 (82.7 per cent.) had rheumatism or heart disease. Sée: Of 128 cases of chorea, 61 (47.7 per cent.) had rheumatism with heart disease. Burton Brown: Of 104 cases, 89 (85.5 per cent.) had antecedent rheumatism. Chapin: 58 per cent. had a history of rheumatism, and 13 per cent. had organic heart disease. Piper: 46 per cent. had antecedent or coincident rheumatism. Herringham: Joint rheumatism (antecedent or coincident) in 26.2 per cent.; rheumatic pains in 20 per cent. Litten: In grown choreics, 42 per cent. had a history of antecedent rheumatism or exposure to cold. Meyer, P.: Rheumatism in 9 per cent.; heart lesion in 10 per cent. Sachs: Of 184 cases, 20 (10.8 per cent.) had had antecedent rheumatism.

Ziemssen, Mackenzie, Gowers: The percentage of choreic cases with antecedent or coincident rheumatism is from 24 to 26. Mackenzie: Of 439 cases, 26 per cent. had subacute or chronic rheumatism; 14.5 per cent. had vague, indefinite pains; 40.5 per cent. had a history of pains. Starr: Of 2476 collected cases, antecedent rheumatism occurred in 662 (26 per cent); coincident rheumatism in 502 (20.3 per cent.). Thayer: Of 789 cases, there was a history of rheumatism in 171 (21.6 per cent.).

All cases were classified as rheumatism in which there were pain and tenderness in the joints. Of 689 cases, there were cardiac murmurs in 235 (40.5 per cent.). About one-quarter of these were considered functional; 25.4 per cent. of the whole number were considered organic.

We would, therefore, be justified, from the evidence so far presented, in considering the close association of rheumatic fever and chorea in the maximum of 25 per cent. of cases.

It should be remembered that in chorea, like epilepsy, the disturbance of the function of the cerebral motor apparatus may occur as the result of various causes. It would be unscientific to consider that large group of cases in which convulsions begin in children of unstable motor mechanism, and which are the result of any infection, as cases of measles and scarlet fever, because these happen to be the most frequent of the infections of childhood. It seems equally unscientific to consider that chorea or similar derangements of the motor mechanism symptomatic of an acute infectious toxic process are in all cases due to rheumatic fever because a rheumatic infection occurs relatively frequently as a late infection in childhood. Macalister,[1] using Ross' method for the study of the life of the leukocyte, has shown that while the toxin in the blood plasma of chorea is toxic to the leukocytes of healthy persons, the blood plasma in cases of rheumatism is scarcely at all toxic, and that the plasma from chorea cases was toxic to the leukocytes of rheumatic cases; from which it would appear that the poisons in the two diseases are dissimilar. The writer is fully aware of the work of Dunn, of Weil, and of Hawthorne,[2] as to the slight or negative articular manifestations of rheumatic fever in childhood, but does not agree with some of the observers that every case of endocarditis, pericarditis, and myocarditis in childhood, in the absence of joint manifestations, is to be considered rheumatic. We must consider, on the one hand, that an infection in childhood is likely to be associated with vague pains, and that, on the other hand, gonorrhœa, beri-beri, syphilis, influenza, tuberculosis, erysipelas, pneumonia, and meningitis are associated with single and multiple arthritic manifestations which are described as "rheumatism," etc.

**Other Infectious Diseases.**—Measles, scarlet fever, diphtheria, typhoid fever, whooping cough, pneumonia, cholera, and smallpox have occasionally been reported as causative factors of chorea. Syphilis, and septic infections, followed by chorea, are also on record. None of these diseases, however, with the possible exception of scarlet fever, occurs with any degree of frequency. The rarity of chorea as a complication of this condition is seen from the statistics of Carslaw, who found only 3 in 533 cases of scarlet fever. The statistics of Osler are somewhat misleading unless properly interpreted. Of the 554 cases reported, the history of scarlet fever was present in 141 cases, without, however, any causal relationship between the two diseases. In none of his cases did the chorea follow immediately upon the scarlet fever. In this connection it should be noted that any condition, whether of an infectious nature or not, if it lowers the vitality of the organism and thereby lessens the nerve tone, renders the child more susceptible not only to chorea, but to other functional nervous disturbances. In this way any of the infectious diseases, anæmia, etc., may act. The toxic influence of any of these infectious diseases, however, should be borne in mind.

[1] *Proceedings of Royal Society of Medicine*, July, 1909.
[2] *British Journal of Children's Diseases*, 1907.

**Pregnancy.**—This is an important factor in the production of chorea, more particularly in the chorea of adult life. The statistics recorded show the incidence of chorea to be relatively slight after the sixteenth year. Only 16 per cent. of the cases, according to the collective investigation committee's report, occurred between the ages of fifteen and twenty. When, however, chorea occurs during adult life, pregnancy must be considered one of the most important, if not the most important factor in its production. Chorea may develop at any time during pregnancy, but is most commonly seen in the earlier months. It usually occurs with the first pregnancy, but may not appear until the second. After its development, however, with the first pregnancy, it may, as in a case reported by Féré, occur with each successive pregnancy for as many as five times. In an interesting case under the writer's observation, an attack of well-defined Sydenham's chorea was associated with a pseudo-pregnancy. The patient, a young married woman, desirous of having children, presented all the symptoms of an early pregnancy. This was associated after the first month with a typical chorea running the usual period of six weeks. At the end of this time the menstrual period returned and the enlargement of the abdomen disappeared. Two months later a real pregnancy developed, running a normal course, without other disturbance.

**Reflex Causes.**—Reflex irritation due to various causes, such as intestinal parasites, intestinal indigestion, phimosis, adenoids, and eye-strain, has been given as a cause of chorea. There is no question that irregular movements simulating true chorea are sometimes produced by such causes in nervous children. It would, however, be a mistake to consider irregular and bizarre movements as identical with a true chorea. It should be considered more in the nature of nervousness, such as is seen in neurasthenic women. A study by de Schweinitz has shown a condition of hypermetropic astigmatism in 77 per cent. of choreic children, and he concludes that a refraction error may be a determining cause of a choreic attack in a susceptible child.

**Pathology.**—Lesions either gross or microscopic have not been found in a sufficient number of cases to constitute a definite pathological basis. Minor vascular and perivascular conditions have been described in a small number of cases, and have constituted the basis for the embolic theory. Terminal emboli have been found with more frequency in the central artery of the retina than in the cerebral capillaries; Thomas collected 7 cases. It is not surprising, in view of the frequency of chorea and the large number of cases showing an endocarditis as a complication, that emboli should occasionally be found. The absence of extensive capillary emboli in the cerebral tissues and a more careful study of the bacterial infections, have led most observers to a position adverse to the embolic theory. The other vascular changes, microscopic perivascular hemorrhages, perivascular leukocytic infiltration, hyaline changes of the vessel walls, hyaline spherical bodies, have occasionally been noted in isolated cases. Inasmuch, however, as these same changes are more frequently noted in other conditions without choreic manifestations, they cannot be regarded as constituting a basic pathology.

**Bacteriology.**—The reaction of the tissues of the newborn child to bacterial invasion has been found to be somewhat different from that of the fully developed individual. The central nervous system is at birth in process of development. Relatively few of the fiber tracts have reached their

full myelinization. The functions of the nervous system are also relatively incomplete. In its reaction to varying stimuli, the nervous system cannot be regarded as complete in its development and of a stable nature until after puberty. An infectious agent or a toxin will give entirely different reactions at birth, during childhood, and in adult life. In children with a finely balanced nervous system and with a sensitive reaction to infectious agents or their toxins, a condition of disturbed function of the nervous system is more easily produced than in the more rugged type. Poynton and Paine, as above noted, considered the diplococcus found by them in rheumatic fever as the causative agent in all cases of chorea. Previous investigations have shown other organisms. Naunyn, in 1888, reported a cladothrix in the meninges and on the endocardium, in a fatal case of chorea. Steinkopff found a streptococcus, Pianese a diplococcus and diplobacillus, Mircoli a streptococcus and staphylococcus in the nervous system. Leredde, in 1891, found a streptococcus in the blood. Triboulet, in 1893, found a staphylococcus in two cases at autopsy, in one of which he had previously found a staphylococcus during life, and in another case a diplococcus during life. Meyer also reports streptococci and the staphylococcus citreus. Diplococci have been reported by Pianese, Triboulet, Coyan and Zadok, Westphal and Wasserman.

In view of these findings we are forced to conclude that the exact bacterial cause of the disease has not been conclusively proved. The correlation of all the facts at our disposal is in the support of the theory of the action of a bacterial toxin upon the cortical tissues.

**Symptoms.**—Cases of chorea divide themselves naturally into three groups according to the severity of the infection: (1) Mild chorea; (2) severe chorea; and (3) malignant chorea, or chorea insaniens.

*Mild Chorea.*—A prodromal period of a week or more is usually presented. The child is listless, somewhat depressed, nervous, with some anorexia and sometimes anæmia. The nervous irritability becomes more marked, and with it slight, irregular, purposeless, jerky movements of one or both upper extremities. This is associated with a pseudo-loss of power in the extremities affected. The pseudo-loss of power, which is dependent on lack of control, is often noticed by parents as the first symptom. A careful testing of muscular power will show usually a normal condition. The tendency to drop articles which the patient may be carrying is not due to a distinct paralytic condition, but to a relaxation of the grasp simultaneously with the development of the choreic jerk. When one arm alone is affected, the disease may progress to the lower extremities on the same side. In some cases the opposite side does not become affected, and a condition of hemichorea is presented. Usually, however, the opposite side becomes affected, following which the head and face share in the movements. Irregular bizarre forms are sometimes presented in which, for example, the arm on one side and the leg on the opposite side may be involved. Speech is usually not markedly disturbed in the mild form, although those accustomed to the conversation in the child will often notice some slight change—a slowness of the speech, with some hesitancy and irregularity. The movements in the mild form of the disease cease during sleep. An actual loss of power may develop in mild cases. The mentality is also affected, even in mild cases; this will be considered more in full under the psychic manifestations.

*Severe Chorea.*—This form of chorea, which may develop in all its intensity from the onset, or may, on the other hand, be of a gradual development from the mild form, presents a condition of constant, irregular movements of practically all the voluntary muscles. The mild form is often disregarded by parents and not considered as a serious condition, but in this form the severity of the movements and the inability of the patients to help themselves immediately demand attention. Not only are the movements more constant, but they are increased in severity and very often persist to a minor degree during sleep, and in some instances actually awaken the patient. The speech is affected in almost all cases. This varies from a hesitancy and explosive type of speech to a mumbling, and often inability to speak at all. While choreic movements of the tongue are sometimes observed, this symptom is, in the writer's experience, more frequently seen in hysterical and habit chorea than in Sydenham's chorea. Disturbance of speech is often due to irregularity of the respiratory rhythm. When the respiration becomes jerky and irregular it is not uncommon to find irregularity of the heart action with it. Motor weakness is the rule in this form, but whether it is a real loss of power or an interference with purposive action by the choreic movements is often difficult to determine. The mental symptoms are much more accentuated, the child lacks power of concentration and attention, is often irascible, and presents some failure of memory. In this form a slight elevation of temperature may be noted varying from 0.5° to 1°. A decided elevation of temperature should always suggest the presence of some complication.

*Malignant Chorea, or Chorea Insaniens.*—This may be the terminal complication of a severe form of chorea, or may develop as a distinct type from the beginning. It occurs more frequently as we approach adult life. There is often some source of intense worry or anxiety as a complicating factor in the etiology. In some cases it is a manifestation of an infectious process on a poorly balanced mentality. Pregnancy is an important etiological factor. The motor manifestations become intense, universal, and constant, may interfere with the sleep of the patient, and rapid exhaustion occurs. A confused delirium, with hallucinations, delusions, and maniacal outbreaks, ensues; the temperature rises as high as 104°, and a fatal termination is the usual result. This condition is not to be confused with simple chorea or mild types of chorea complicated by simple delusional states.

**Psychic Manifestations.**—Burr states that "although there are no sharp lines dividing the cases, they may be separated, so far as the mental symptoms are concerned, into the following groups: First (and this includes the vast majority), patients in whom there is peevishness, fretfulness, some loss of the power of fixing the attention, and a slight loss of the moral sense shown by disobedience and selfishness. Second, those showing, in addition to the above symptoms, night terrors, and transitory, visual, auditory, or other hallucinations. Third, those with distinct delirium, wild or mild, accompanied with fever. Fourth (and this group is very small when we remember how common chorea is), those showing stupor, or rather stupidity, and an acute dementia, which may follow the condition described under three, or appear without any preceding mental symptoms at all severe, and which is usually accompanied with trouble in articulation not caused by choreic movements of the lips and tongue, but the result of mental hebetude. Fever is usually present for a time at least. Patients of the first and second groups almost

always recover mentally and physically; those of the third group frequently die, and those of the fourth usually either die or, recovering from the chorea, remain demented."

This states the subject of the mental condition in chorea, practically in its entirety. While the maniacal condition has been considered by some to be in the nature of a "cerebral rheumatism," it can be more accurately stated that when this condition is associated with a high terminal fever, it represents the effect of the infection producing the febrile condition. States of mental alienation with excitement, as indicative of a grave form of chorea with an unfavorable prognosis, should be differentiated from hysteria complicating chorea. Minor hysterical stigmata, as might be expected from the neurotic nature of a large number of these children, are sometimes met with. Areas of anæsthesia, hyperæsthesia, points of acute tenderness along the nerves and vertebræ, and suggestive pains in the extremities have been described. Triboulet suggests a correlation of the symptoms to the metameric zones of Head. Rarely, hysterical contractures and convulsions, with mental excitement, may occur. The prognosis in these cases, however, is not altered by this complication.

**Paralytic Phenomena.**—A pseudo-loss of power due to the intensity of the choreic movements has been described above. In addition to this, an actual loss of power, which approaches but never arrives at a complete paralysis, has been noted by various observers since Sydenham. West gave to this condition the name "chorea mollis." The loss of power may precede the development of the choreic movements, may be coincidental with them, may remain after the choreic movements have to a large extent subsided, and may in some cases inhibit the choreic movements to such an extent as to appear to supplant them. The motor weakness may be partial in nature, a monoplegia, hemiplegia, paraplegia, or in very rare instances may be general throughout the voluntary muscles. While this condition is usually associated with very severe forms of chorea, it may, on the other hand, complicate very mild cases. A case of marked hemiplegia in a boy, aged eight years, was at first thought to be due to an organic cerebral lesion. Careful observations, however, revealed a mild chorea, and further observations confirmed the diagnosis of chorea mollis. Apart from the paralytic phenomena, the etiology, symptomatology, and progression remains the same as in the usual type of Sydenham's chorea.

Rondeau has noted muscular atrophy as a very rare complication. This atrophy exists in cases in which the chorea complicates, or is associated with, a rheumatic affection. He considers it in the nature of a reflex joint atrophy. It disappears with the cure of the chorea and the rheumatic fever.

**Reflexes.**—Much conflicting testimony has been presented as to the nature of the reflex activity in this disease. The writer's experience has shown the reflexes to be uniformly active and often quickened when properly elicited. The child, however, must be relaxed, and its attention thoroughly diverted, in order to determine the exact nature of the reflexes. In chorea mollis, when the loss of power is marked, the reflexes may be diminished in the affected part. Triboulet notes a delayed reflex activity. Other irregularities of reflex action are sometimes noted. The more constant of these is a delayed relaxation of the part to normal after the motor explosion (Gordon). The writer has never seen complete abolition of the reflexes as described by Joffroy. This author described the reflexes as diminished, as a rule,

and the normal or excited reflex as an exception. Oddo also notes, in a study of 147 choreics, normal reflex activity on both sides in 8, and on one side in 28 cases, and arrives at the conclusion that normal reflex activity only occurs in mild cases, and that usually the reflexes are diminished or suppressed (116 out of 147 cases). He also notes that an exaggeration and suppression of the reflexes may occur at different times in the same case.

**Urine.**—An excess of urine and uric acid has been found in proportion to the severity of the muscular activity. Garrod found urohæmatoporphyrin in 14 out of 20 cases. Herter reports the presence of hæmatoporphyrin in the urine not only of chorea, but likewise rheumatism. Albumin should be taken as evidence of kidney complication. Glycosuria has also been noted.

**Complications.**—The most important complication of chorea, and one which should not only be kept in mind, but carefully sought for during the course of the disease, is endocarditis. The most comprehensive statement of the relationship will be found in a study by Thayer, who found that 25.4 per cent. of his cases showed evidence of organic heart disease. Osler found evidence of organic heart disease in 72 out of 140 patients examined more than two years after the attack; and in a collection of 73 autopsies, endocarditis in 62. He further lays down, for the guidance of the physician, the following rules:

"1. In thin, nervous children a systolic murmur of soft quality is extremely common at the base, with accentuation of the second sound, particularly at the second left costal cartilage, and is probably of no moment.

"2. A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

"3. A murmur of maximum intensity at the apex, with rough quality, and transmitted to the axilla or the angle of the scapula, indicates an organic lesion of the mitral valve, and is usually associated with signs of enlargement of the heart.

"4. When in doubt it is much safer to trust to the evidence of eye and hand than to that of the ear. If the apex beat is in the normal position, and the area of dulness not increased vertically or to the right of the sternum, there is probably no serious valvular disease.

"5. The endocarditis of chorea is almost invariably of the simple or warty form, and in itself is not dangerous; but it is apt to lead to those sclerotic changes in the valve which produce incompetency.

"6. Pericarditis is an occasional complication of chorea, usually in cases with well-marked rheumatic fever."

Other complications of a rheumatic nature are erythema nodosum, subcutaneous fibroid nodules, rheumatic purpura, etc. Herpes zoster occasionally occurs, and has been attributed to the use of arsenic.

**Course and Prognosis.**—The usual case of chorea runs a course of from six to ten weeks. A mild case sometimes recovers in a shorter period. Not infrequently the course is prolonged for three or four months. Choreic movements may persist for many months—a condition termed residual chorea by Guthrie. This can be explained to a certain extent by the nervous condition of the child, the movements remaining as a habit chorea, and in rare cases may be due to the presence of some persistent peripheral irritation. Recurrent attacks of chorea are so frequent that the physician must be on the watch for them and take measures to prevent them. A boy

recently under observation has been affected with choreic movements in the last five years for a longer period of time than he has been without them. The attacks have occurred each spring and fall, lasting three or four months.

The outlook, as a rule, is favorable. Under proper treatment the patients may be assured of a complete recovery, but should also be told of the possibility of cardiac complications and the tendency of the disease to return during a period of several years.

**Treatment.**—Chorea at all times, even the milder forms, is sufficiently serious to demand the careful attention and supervision of the physician. All patients do better, the course of the disease is shortened, and the danger of complications lessened by confining the child to bed during the period of active symptoms. Anything that tends to produce mental excitement should be rigidly excluded. A nurse trained to handle nervous children is a very helpful adjunct to the treatment. The diet should be simple and nutritious, with tea and coffee excluded. In the severe cases isolation is necessary. Care should always be used in permitting visitors; strangers should be excluded from the sick room, and even members of the family when their visits or presence produce undue excitement. School duties and intellectual efforts should be avoided. Gentle massage, with bathing and a warm or cold wet pack, often have a quieting influence when properly administered. It is quite necessary in the poorly nourished, in whom overfeeding is necessary, to keep the muscles in good condition by routine massage, followed during convalescence by passive and resisted movements. These, however, should be carefully watched, and if any tendency to accentuation in the motor phenomena is manifested, they should be decreased or stopped altogether. Electricity is sometimes of value both as a body stimulant and in keeping the muscles in good condition, but often produces too much excitement to be used. Galvanism should be applied in preference to faradism.

The medicinal treatment is confined to alterative tonics and nerve sediments. In the former, arsenic, in the form of Fowler's solution, is of distinct value. It is questionable, however, whether its value is more than that of an alterative tonic. There is a possibility of doing harm if the administration of the drug is not carefully watched and the cases for its use properly selected. It is a mistake to give the drug in large doses, and it should never be given until the physiological limit is reached. Its full value may be obtained by the administration of small doses; two to three drops gradually increased until ten drops three times a day are taken. It ought never to be administered except when the patient is under the personal observation of a physician. Indiscriminate and persistent use of the drug may result in arsenical neuritis and serious disturbance of the metabolism of the child. Extensive herpes zoster has been attributed to its use. Donovan's solution may be administered in doses of two to five drops, in older children, three times daily, but here again the patient should be carefully observed. Strychnine, cimicifuga, and belladonna are sometimes used.

Sedative drugs have a decided value in the more severe and active cases. Chloral is the most valuable of these remedies, and is usually combined with one of the bromides in doses of 5 grains each, three or four times a day. In some cases it may be necessary to increase both drugs slowly, to as high as 15 grains, in older children. It is almost unnecessary to call attention to careful observation of the heart action if chloral is found necessary. In

other severe cases hyoscine may be used, either by mouth or hypodermically. Antipyrine, extract of cannabis indica, and chloretone have been found to be of value. Chloretone, like hyoscine, has a decidedly quieting influence in some cases, but the administration of both drugs must be carefully guarded. Such remedies as aspirin, the salicylates, and alkalies are of value in the control of rheumatic symptoms, but have relatively little influence on the course of the disease. Potts found apomorphine to be of distinct value in controlling the symptoms of very severe cases. In chronic chorea, when the movements persist after the general condition is improved, and in some of the milder forms of chorea, treatment by means of a combination of suggestion, at first passive movements and later by means of exercises, may be of distinct value. This treatment has been systematized by Guthrie, as follows:

1. Suggestions that the child should lie quietly should be repeated in the soothing manner used by the hypnotist. At the same time the flourishes and wriggles should be gently restrained, and the child will soon learn to lie completely relaxed and flaccid under observation.

2. Passive movements combined with suggestion. It will be found that choreic movements occur directly the child's limbs are manipulated. To correct this condition the patient's hand should be placed between the observer's hands, and raised and moved in various directions, suggestion being made meanwhile that the child should keep his own hand quite still. At first it will be snatched away and flourished as usual, but soon the patient learns to control the involuntary movement by an effort of will which makes the whole limb rigid. This is an indication of improvement, although by no means of a cure; but by degrees the child discovers that the efforts to control involuntary movements need not be so strenuous whilst his limbs are guided and restrained, and accordingly the rigidity becomes relaxed. The measure of improvement is easily ascertained by the observer. Until the limbs can be passively moved freely in all directions without exciting either rigidity or spasm no other than passive exercises should be employed.

3. Voluntary movements under guidance. The patient is then directed to perform the same movements as have been passively executed while his hand is still controlled, guided, and helped by the observer.

4. When this can be done without exciting spasms or rigidity, but not before, voluntary movements without control, in imitation of the observer's should be practised.

The movements should be of the simplest character at first, and gradually made more elaborate. They should be executed slowly and steadily, without jerks and flourishes. The chief difficulty is, at this stage, incoordination rather than spasm, and the mode of treatment is adapted from Fränkel's system in the treatment of locomotor ataxia.

Elaborate apparatus is unnecessary. Simply bringing the fingers together from a distance and touching various parts of the body with them are admirable exercises. Precision of movement can be gained and the child kept amused by various toys and games. The kindergarten supplies simple and cheap apparatus which answer the purpose. Colored balls hung by strings on a frame or strung on wires can be used. The balls can be made to swing, touched and arranged in patterns under direction. Then solid squares or cubes can be built in various shapes and forms. Such games as draughts, dominoes, or solitaire can be used for older children,

or "spilikins," easily improvised with a box of matches, pegs to be fixed in the holes in a backgammon board, can be pressed into service. A child may usually be considered cured when he can build a two-story house of cards.

The aim of exercises is to encourage freedom as well as precision in movement. For this reason it is inadvisable to allow the patient to write or to thread needles or to sew. All these require much mental concentration, fixation, and tension of the various muscles employed. There is no objection to freehand drawing on a slate or blackboard, and paper plaiting or weaving may be encouraged. It is important that all exercises should be carried out under supervision, otherwise the child becomes careless and ceases to take any trouble, provided that he can amuse himself, and if neglected often drifts into what may be called "residual chorea." The treatment in this event is by a course of drilling, marching, wheeling, and standing at attention, at word of command in particular, for the child's chief faults are inattention and carelessness, which have to be overcome. Ordinary calisthenic exercises are also useful in these cases. The important feature of "residual chorea" is that the movements are unconsciously performed, and can be immediately controlled by effort of will.

The treatment of the lower extremities is on the same principles as that of the upper. The patient should not be allowed to stand, or try to walk until all movements of the legs can be executed with fair precision while lying down. Such ataxia as still remains should be treated by making the child stand supported by the back of a chair while he places each foot separately in various positions. It is necessary finally to observe that exercises of any description are inadvisable in all acute and recent cases of sthenic chorea. They are only of use when heart, pulse, temperature, appetite, and digestion are normal, all pains and aches have disappeared, and, although general health has seemed fully restored for some weeks, choreic movements still continue.

The principles in this are: (1) That suggestion is of service where there is lack of inhibitory control over choreic movements. (2) Inhibition when acquired is often exaggerated, and has to be regulated by assistance before voluntary movements can be executed. (3) Incoördination has to be treated by exercises carefully graduated in the order of their difficulty of accomplishment.

## HABIT CHOREA, HABIT SPASM, CONVULSIVE TIC.

**Definition.**—A tic is a sudden, quick, involuntary coördinated muscular action, usually of a purposive type, and partly or entirely under the control of the patient.

Much time and thought have been devoted to the elucidation of the subject of tics. Sinkler, in using the term habit chorea, placed them in the larger group of the choreas. Objection has been made to this on the ground that, being purposive and intentional, they are of an entirely different nature from the movements of Sydenham's chorea. The term habit spasm is equally objectionable, because a large number of them are not in the strict sense of the term of a habit nature; *i. e.*, they do not develop by repetition from a volitional act. While Guthrie separates these two conditions, it is often difficult in the fully developed complex tic to decide whether it

began in the form of a habit spasm or not. All investigators agree in separating from the group of what might be termed harmless tics those cases presenting a similar clinical picture, but developing on a foundation of a serious neurosis. In Tourette's disease, which represents the most marked type of this latter group, all the voluntary muscles may be affected. The cases in this group are classed as incurable. A study of a large number of cases of tic will show a neurotic basis not only for the psychic tics (Tourette's disease), but also in the simpler forms.

It is likewise true that in many of the simple habit spasms and tics a psychic element plays an important role. Patrick has called attention to the fact that the tic movement represents a distinct psychic want—*i. e.*, a sense of relief as opposed to a very decided sense of psychic distress when the movement is inhibited. Each case of tic represents in its etiology and its essential nature some definite disturbance of the nervous balance of the child. As such, each case needs special and individual study.

**Etiology.**—An inherited or acquired neurotic temperament is the important etiological factor in most cases. In children of a nervous temperament, self-control is an essential factor in maintaining a nervous balance and proper functioning of the nervous system. Habit spasms and tics in early life and other functional neuroses in adolescence and adult life are frequently the result of a lack of discipline in the training of a child. The more vicious the heredity, the more need there is for careful guarding of the education and the self-control of the child. So-called "spoiled children" offer the best soil for the development of habit spasms and tics.

Anything which reduces the nerve tone of a child may determine the development of a tic. In some children tic may develop as a result of a loss of weight, vigor, and nerve tone, below a certain point. In these children such manifestations may be looked upon as an indication for medical supervision and treatment. In finely balanced children a nerve drain from some irritation, such as carious teeth, adenoids, phimosis, intestinal parasites, etc., may be the determining factor. These causes are usually referred to as reflex. Excessive worry, emotional excitement, or other forms of mental overstrain may in some cases be the cause.

**Symptoms.**—The movements differ from those of Sydenham's chorea in their evident purposive character and their localization to a single muscle or group of muscles. The muscles of the face are most frequently affected. A sudden, quick blinking of the eyes, which may be repeated very frequently at short intervals, or occur only a few times during the day, is the form most frequently seen. The eyebrows in other cases are suddenly elevated; the face may be drawn to one side, or the facial muscles of both sides may be affected, producing a sudden involuntary grin. The tongue may be affected, and a sudden movement as if moistening the lower lip may be so frequently repeated as to produce an inflamed condition of the skin of this area. In one child, who had been able voluntarily to produce a movement of the ears, a sudden jerky movement of both ears developed independently of the volition of the patient. Spasm of the muscles of the neck results in a jerking of the head to one side; shrugging of one or both shoulders is not infrequent.

There are usually no other symptoms apart from the motor phenomena. Hysterical outbreaks are usually met with in the type of children subject to this affection.

**Diagnosis.**—In rare cases several groups of muscles may be affected at the same time, but they can be easily differentiated from Sydenham's chorea by the purposive character of the movements.

There is a condition of the muscles of the face, most frequently localized to the orbicularis palpebrarum and due to the toxic influence of tea and coffee on the nervous mechanism, which should be distinctly differentiated from the habit spasm. The manifestations are lightning-like contractions of the individual fibrillæ, affecting all of them in rapid succession, producing at the most a slight quivering motion of the lids, but never leading to the distinct blinking of habit chorea. This is as frequently seen in adult life as in children, and yields rapidly when tea and coffee are excluded from the diet. In some of the cases eye-strain is a factor.

Habit spasms should also be differentiated from what has been described as impulsive tic (Gilles de la Tourette's disease). Some of the forms of this condition appear to be a more serious and wide-spread affection of the toxic condition above described, affecting the orbicularis palpebrarum, and due to some intoxication. It is not infrequently fatal. It begins, as a rule, in very early life, although it may occur as late as early adult life. The muscular movements may affect all the voluntary muscles, and are lightning-like in character, with marked fibrillary movements. Another group of cases described under this condition presents the same quick action of the muscles, with mental disturbance and the use of foul language. The explosive, quick character of the movements, the mental disturbance, and the coprolalia should differentiate it from either Sydenham's or simple habit chorea.

**Treatment.**—In both habit spasm and impulsive tic an underlying cause should be carefully searched for and removed. In the habit spasm about the eyes, errors of refraction and loss of muscle balance should be first corrected. The mucous membrane of the nose and the condition of the turbinate bodies should be examined to determine any cause for irritation. The ears and teeth should in the same way receive attention in all cases where the symptoms are referable to any part of the face. Irritative reflex disturbances in the genito-urinary tract, such as phimosis, etc., should be relieved. One patient with complex shrugging movements about the shoulders and twisted movements of the trunk resisted treatment until a rough woollen sweater, which he wore next to the skin, had been replaced by proper underclothing. The spasm then rapidly disappeared.

The general nutrition should be brought to a normal standard and a proper discipline infused in a routine way into the child's life. While punitive disciplinary measures sometimes succeed in early imitative cases, they frequently do harm. The child should be sent to bed at a definite time early in the evening and compelled to remain in bed an hour after the usual time of rising, both as a disciplinary measure and to secure an added amount of rest for the weakened nervous system. A period of rest in the middle of the day is also advisable. A cold sponge bath or needle bath is valuable as a tonic stimulant if the child reacts well. A simple diet without tea or coffee and with little meat, but with plenty of milk, eggs, and vegetables, is indicated. Alterative tonics, such as Fowler's solution, quinine, and strychnine, are sometimes of value; more frequently better results are secured by the use of bromides and other nerve sedatives. The child should be encouraged to inhibit the movements as far as possible.

**Electric Chorea.**—This is a rare disease, first described by Dubini, and is manifested as intensely rapid rhythmical movements in the extremities, rarely in the head and face. The movements may be very violent, and have the appearance of being produced by an electric shock. In the severer form described by Dubini as occurring in Italy, paralytic symptoms supervene, and may be associated with epileptiform convulsions. Pain in the head and neck may be present early, and toward the end of the attack atrophy and wasting of the muscles may occur. Fever may be present. The cases terminate in a few weeks or months from heart failure or coma. Jaccoud considers the affection to be due to a symptomatic chorea due to a low grade of cerebrospinal meningitis. A form of electric chorea, probably due to hysteria, has been described by Bergeron. Henoch has also described a form of electric chorea differing from both the above, and manifested by spasmodic attacks of lightning-like contractions confined to the muscles about the shoulder blade; it is probably a form of myoclonus.

**Treatment.**—This should be directed to the removal of any underlying intoxication. Free purgation, chloral, and bromides have been used, but to little effect.

## HUNTINGTON'S CHOREA.

**Synonyms.**—Chronic progressive chorea; chronic hereditary chorea; degenerative chorea.

**History.**—This affection was first fully described by Dr. George Huntington, of Long Island, in 1872. Previous mention of this disease may be found in the writing of Waters, of Franklin, New York, as early as 1841, who recognized the affection, called by the laity "magrums," as a distinct disease, and who directed attention to the onset in adult life, to the hereditary nature, and to its incurability. Lyons, in 1863, published an article in the *American Medical Times*, on chronic hereditary chorea, in which he noted all the essential features of the disease. Gorman, in an inaugural dissertation presented before the faculty of the Jefferson Medical College, stated that the affection described by Lyon existed in other portions of the country. According to him, it is circumscribed by neighborhood boundaries and is confined to sections of the country the inhabitants of which are intimately connected in their social or business relations. Huntington's description, however, was so much more accurate and served to call the attention of other investigators to the affection that the disease since then has been associated with his name. Important contributions have been made to the subject by Ewald in 1884, Peretti in 1885, Huber in 1887, Zacher, Hoffmann, Lannois in 1888, Huet in 1889, Biernacki in 1890, Jolly, Remak in 1891, Phelps, Schlesinger, Sinkler in 1892, Kronthal and Kalischer in 1892, Blankenstein in 1893, Oppenheim and Hoppe in 1893, and Facklam in 1896. A complete bibliography of the publications since that time will be found in the Huntington number of *Neurographs*, May 25, 1908.

**Etiology.—Age.**—The disease is preëminently, if not exclusively, a disease of adult life. It usually begins between thirty and forty years of age. Premonitory symptoms, in the nature of mild choreic movements, or more frequently in the nature of extreme clumsiness, or sometimes in an extra precision of movement, occur as early as the twentieth year. Cases have been reported in childhood, *i. e.*, in the second decennial period, by

Hoffmann, Peretti, and others. Stevens reports a case in which the chorea developed in infancy. The accuracy of this case as one of Huntington's chorea has been questioned. In one of the writer's patients, the history of a child born with choreic movements, which persisted for several months and until the death of the child, while suggestive, does not seem to be sufficiently conclusive to be of value in this connection. Jolly reported a case in which chorea and epilepsy developed unquestionably at nine years of age. Hoffmann reported a case in which epilepsy antedated the chorea by several years. Remak reports a case in which epilepsy was present from the twenty-third to the thirty-first year, followed by chorea at forty. Diefendorf reports a case in which epilepsy developed at seventeen and choreic manifestations appeared at fifty-three. The chorea, however, was intermittent, occurring only in the first convulsive periods.

**Distribution.**—The cases above described are practically over all the European countries—with the exception of Turkey—the West Indies, and South America. Several cases have been reported in negro families, associated with other nervous maladies, the most important of which is epilepsy.

**Heredity.**—Direct heredity has been found in practically all of the cases studied. The exact percentage, however, of those affected who developed chorea has not been determined. Huntington states that "if one or both of the parents have shown manifestations of the disease, and more especially when these manifestations have been of a serious nature, one or more of the off-spring almost invariably suffer from the disease if they live to adult life; and if by any chance these children get through life without it, the thread is broken and the grandchildren or great-grandchildren may rest assured that they are free from the disease. Unstable and whimsical as the disease may be in other respects, in this it is firm; it never skips a generation to manifest itself in another; as soon as it has yielded its claims, it never regains them." Huntington further speaks of the presence of the nervous temperament in all or nearly all of the families in which the choreic taint exists, and that "nervous excitement to a marked degree almost invariably attends upon every disease these people may suffer from, although they may not in health be overnervous." Heilbroner states that there is a tendency in successive generations for the onset of the disease to be delayed. Wollenberg states that the choreic heredity in some cases may be transformed into other neuroses, *i. e.*, epilepsy, imbecility, paranoia, grave hysteria, etc.

**Sex.**—Huntington states that it is more common among men than women. He was not aware that season or complexion had any influence in the matter. Wollenberg has collected statistics of 128 cases in twenty-two families; of which 74 were in men and 54 in women.

**Pathology.**—The pathological picture is that of a chronic diffuse cortical encephalitis. Three of the writer's cases in both the gross and microscopic picture resembled very closely that seen in paresis. There was a marked atrophy of both the convolutions of the frontal lobes and the motor area. The arachnoid was thickened relatively (due to cortical atrophy) and actually, due to a chronic proliferative process. Under the microscope there was a proliferation of the neurogliar network, with an increase of the neurogliar cells both in the cortical and subcortical tissue. The atrophic process was shown by the absence of the tangential fibers and by the marked perivascular and pericellular space dilatations with both perivascular and pericellular neurogliar nuclear proliferation. The ganglion cells of the cortex appeared

to be fewer in number, and with atrophic and pigmentary changes. There was an accumulation of subarachnoidal fluid (hydrocephalus externus), which was in all probability secondary to the atrophic processes in the brain. Throughout the basal ganglia extensive areas of perivascular proliferative gliosis were present. In other cases simple hemorrhagic pachymeningitis was found. Lannois and Paviot explain the development of the disease upon the hereditary malformation of the neuroglia of the cortex. They state that the late development of the symptoms is due to a subsequent proliferation, an accentuation of the progression of this teratological malformation.

**Symptoms.**—The heredity, the chorea, the tendency to insanity, and its development in adult life are the important features. Of the chorea, it may be stated that the movements resemble very closely, yet are distinctly different from, those seen in Sydenham's chorea. They are essentially of the same purposeless, jerky nature. There is a gradual development, at first an apparently extra precision of voluntary motion, followed by a distinct clumsy, irregular action of the muscles. They are in the beginning of minor intensity and may be confined to the upper extremities or the face. Later, the legs, and afterward the voluntary muscles of the entire body, with the exception of the eye muscles, become affected. In its final development there is a tendency to more symmetry of movement than is seen in Sydenham's chorea. This results, when the lower extremities become affected, in a shuffling, dancing type of gait, associated with irregular movements of the trunk and arms. Choreic movements of the facial muscles cause the production of grimaces somewhat allied to those seen in some cerebral types of chorea. Speech, sooner or later, becomes affected on account of the involvement of the tongue, the lips, and the respiratory muscles, and assumes a hesitating, stuttering, explosive type of articulation. The movements, as a rule, but not necessarily, disappear during sleep. The movements are under the voluntary control of the patient, even when moderately severe. A patient, for years under personal observation, although suffering severely from choreic movements, was nevertheless able to attend to his occupation as a carpenter, and to drive nails with fair precision. The motor power is, as a rule, not involved. In terminal cases, a general or hemiplegic loss of power is sometimes noted. The reflexes are exaggerated, as a rule. The electrical excitability remains normal.

**Psychic Symptoms.**—Huntington stated that "the tendency to insanity, and sometimes that form of insanity which leads to suicide, is marked. I know of several instances of suicide of people suffering from this form of chorea, or who belonged to families in which the disease existed. As the disease progresses, the mind becomes more or less impaired, in many amounting to insanity, while in others mind and body both gradually fail until death relieves them of their sufferings. At present I know of two married men, whose wives are living, and who are constantly making love to some young lady, not seeming to be aware that there is any impropriety in it. They are suffering from chorea to such an extent that they can hardly walk, and would be thought by a stranger to be intoxicated. They are men of about fifty years of age, but never let an opportunity to flirt with a girl go past unimproved. The effect is ridiculous in the extreme." Other observers have also noted the mental condition to be a severe and gradually progressive deterioration, ultimately ending in absolute dementia. In some cases the mental defect is noted from the onset of the symptoms; in others, the men-

tality may remain unimpaired for years. Mental deterioration is, however, the rule, and it is associated with a loss of memory and a tendency to self-destruction which gradually develops. When the mental degeneration is well marked, outbreaks of violence are sometimes noted. In one of the writer's patients, as the disease progressed to its termination, the clinical picture of paresis was presented. The patient, the carpenter referred to above, changed from a depressed, almost a melancholy state, to a feeling of well-being, with delusions of grandeur, a condition which persisted for a period of two years, until the time of his death. Burr,[1] records a similar observation. The chronic delusional state is more often noted than would be inferred from Huntington's description.

**Diagnosis.**—The other forms of adult chorea apart from the infectious toxic variety of Sydenham may occur, but this is the only type which presents any difficulty in diagnosis. The senile chorea associated with arteriosclerosis, and the degenerative processes of the cortex which go with it, are sometimes mistaken for Huntington's chorea. This condition, however, only appears long after middle life, and lacks the most essential feature in Huntington's chorea, the heredity. In investigating the family history it should be borne in mind that the disease is usually looked upon as a family affliction, and its presence in other members of the family is at first denied by the patient. It requires tact and careful questioning to elicit this important factor in the diagnosis.

**Prognosis and Treatment.**—The prognosis is absolutely hopeless. Nothing has been found to retard or inhibit the progressive course of the disease. The same statement is true as to treatment. Many drugs have been tried, but have been found wanting.

## CONVULSIONS OF INFANCY AND CHILDHOOD.

Convulsions occurring in infancy and childhood vary greatly in nature, in causation, in the clinical picture, and in the resultant effect upon the nervous system. They may present the symptomatic representation of some general disease or some toxic condition, or, on the other hand, they may present as an epilepsy, the disease itself. Inasmuch as epilepsy is considered elsewhere, this article will consider the subject of infantile convulsions as distinct from epilepsy.

**Etiology.—Age.**—In most cases fits occur before the completion of the first dentition. Of 300 cases studied by Cautley,[2] 44 began in the first six months of life, 52 in the second, 76 in the second year, and 44 in the third year. Thus, no less than 216 were infants under three years of age, 27 were in their fourth year, and the remainder under twelve.

There is no question of the different reaction of the nervous system in infancy, childhood, and adult life. Even when there is a definite pathological factor, as disease of the thymus, this variation is shown, for at different periods of the development of the nervous system, thymic asthma, convulsions, and myasthenia gravis are presented. At the onset of the acute infectious fevers the rigor of the adult is sometimes represented by a distinct convulsive

[1] *Journal of Nervous and Mental Disease*, 1908, vol. xxxv.
[2] *The Clinical Journal*, 1905.

seizure. This variation is more one of degree than of essential difference. Too much stress is often laid upon the term "convulsion." While loss of consciousness is the rule in convulsive seizures, it need not necessarily be absolute. In the Jacksonian attack of adult life, consciousness may be preserved during the attack, even when the convulsive seizure is of considerable distribution. The so-called convulsions of Adams-Stokes disease may be an intermediate motor reaction between the prolonged rigor at the onset of an acute infection like malaria and the more intense reaction of the convulsion seen in childhood and adult life. Thus, the motor intermittent muscular reaction, or a decided chill, which has been described by a sensation of cold, is essentially a convulsive seizure of nervous origin, and not under the control of the patient. The same grade of intoxication in infancy and early childhood produces a more acute motor reaction with disturbance of consciousness.

The work of Soltmann explains to a certain extent this variation. He found in experimental work on animals that the irritability of the motor nerves in the newborn was negative, while the irritability of the sensory nerves was low. Both varieties of nerves after birth, however, not only reach the normal irritability, but actually develop an electrical hyperirritability above that seen in the adult. The cerebral motor area is undeveloped and does not react at birth to electrical irritation. The reflex inhibitory centres and paths were also found entirely wanting. An important factor is the lack of synchronous development of the inhibitory with the motor function and the peripheral irritability. As a result of this the reflex irritability in the newborn is somewhat diminished, and thereafter shows an increase to a point above normal. Soltmann also determined, from investigations upon young animals and nursing babies, an increased reflex irritability from the fifth to the eleventh month. While later investigations have shown that in certain respects it has been necessary to modify these observations in all cases, they remain for the most part confirmed. Objection has been made to the application of the animal experiments to the human being, because of the production of convulsions on the cortical irritation of human infants. All the observers are agreed upon the absence of the inhibitory function at birth, its delayed development, and the incomplete function of the psychomotor centres.

Apart from these factors, the absence in infancy of volitional motor control, that lack of what might be termed subconscious "willing," a part of the psychic function, appears to be an important element in the production of convulsive states. The well-trained and well-disciplined nervous system is less likely to show motor unbalance under stress and insult than when the opposite is the case. In infancy, lack of such training and discipline, together with the failure of synchronous action of the different functional areas of the brain, combined with the varying progress in the development of the elemental functions, renders the nervous system susceptible to disturbances of motor and psychic correlation by toxic, traumatic, or organic influences. In other words, there is wanting not only the automatic inhibition of the nervous system, but also its reënforcement by volitional inhibition and control. The production of convulsive seizures can best be understood in the simple reflex group. Convulsions may be produced in guinea-pigs by irritation of the sciatic nerve. In a patient under personal observation at the Philadelphia General Hospital, convulsions were produced

at will by the irritation necessary to the dressing of an irritated surface on the leg. The patient, a man, aged forty-two years, had convulsions during childhood up to the sixth year, and was entirely free until the necessity arose for active treatment of the local condition. On every occasion on which the ulcerated surface was dressed without the use of cocaine, a general convulsion resulted, which could be entirely prevented by proper local anæsthesia, and ceased to occur after improvement in the part. In toxic cases the irritation is probably direct upon the central nervous mechanism. In strychnine poisoning there is both motor and psychic irritation; insomnia, as an increasing early symptom, is evidence of psychic stimulation. In convulsions of gastro-intestinal origin there is in all probability a combination of factors—a reflex exciting factor, a complete toxic factor, and a possible disturbance of the cardiorespiratory mechanism.

**Predisposition.**—The individuality in children, as regards motor balance, coördination of the psychic, motor, and inhibitory functions, and the defence of the body in resisting invading toxins is as varied as the physical character. This, in some cases, is a matter of heredity; in others, and this is more particularly true in infancy and early childhood, hereditary influences do not play an important part; in other words, a distinction can be made between an inherited predisposition and an individual predisposition. In a family of six children of good heredity under personal observation, one of the children had convulsions at the onset of measles, scarlet fever, and influenza, whereas the other children suffering from the same affections of approximately the same intensity were free from convulsive seizures. We may, therefore, assume even in infancy a predisposition to the convulsive habit. This is true both of the convulsions of a reflex nature as well as those to which the term "idiopathic" has been given. Moon, who made a careful study of 200 cases, found that convulsions in infancy associated with reflex causes were quite as serious as regards frequency and the mental and moral deterioration as those of an essential nature. In all the varieties of the convulsions of childhood a certain intrinsic condition of the brain in the nature of an instability of function is the most important etiological factor.

Convulsions in childhood almost always have a serious aspect. With proper care and attention at the time of the convulsions, and with careful attention to the life, education, and after-treatment, they may mean nothing more than an accidental complication of some underlying condition. On the other hand, they may be the starting point of a serious disturbance in the function of the growing nervous system, with mental and moral deterioration, and grave neuroses and epilepsy as the end-result.

**Reflex Causes.**—Reflex disturbances, in the writer's opinion, have been much exaggerated as a causative factor. That they are an important determining factor the writer is not prepared to deny. Except, however, for those which produce sufficient sensory irritation to amount to pain, they act, for the most part, in lowering the general health and nutrition, and causing a loss of nerve tone sufficient to bring the nervous system to a convulsive reaction. We should divide reflex factors into those which are purely reflex in their action and those associated with the disturbance of metabolism. One of the most important factors, namely rickets, which has been placed by some authors in the reflex group, probably acts in both ways. Irritation and subcutaneous lesions of the skin, such as that resulting from excessively hot baths, scalding and burning of the skin, sudden and violent varia-

tions in the surrounding temperature, painful wounds, and continued irritation from foreign bodies, often act as the determining factor. Other painful conditions, such as herpes vaginalis, phimosis, pressure on the testicles, erosion of the genitals from irritation of urine, etc., may act in a like manner. Diseased conditions from the presence of foreign bodies in the nasal or aural cavities are sometimes responsible. Carious conditions of the mastoid and disease of the middle ear may be associated with convulsions without evidence of sinus or meningeal trouble. Reflex conditions in the gastro-intestinal tract have been given as a cause in a large class of cases. The eruption of the teeth is given as an important factor by many authorities. Henke, however, as early as 1818, called attention to the fact that teething was a normal process, and that it usually occurred at about the same time as a rather rapid development both in the brain and in the general economy, and that the resultant increased sensibility was more to blame than the local process. Most observers have supported this position, which also received confirmation from the work of Soltmann. These statements, of course, refer to simple teething processes, and not to those cases in which local inflammatory and other complications exist. Ulcerative conditions of the aural mucous membrane, enlarged tonsils, and nasopharyngeal adenoids may act in a purely reflex manner, or assist in lowering the general nutrition and nerve tone.

Pathological factors in the stomach and intestinal tract present complicating factors besides the simple reflex action. Overloading the stomach of a person with a finely balanced nervous system with indigestible food may be the determining factor in a convulsive seizure not only in childhood, but at any period of life. The ingestion of irritating substances, such as alcohol, reacts in a like manner. Vogel describes convulsive conditions as the result of a gastritis from the ingestion of sulphuric acid and caustic potash; Bouchat, as the result of the various catarrhs of the gastro-intestinal tract; Romberg, from the presence of foreign bodies. In chronic constipation, intestinal auto-intoxication is considered as a factor by some. Intestinal parasites of various kinds have been blamed for the production of convulsive conditions. The number of children with convulsions is relatively small in proportion to the large number who suffer from intestinal parasites. The remarks on dentition have a like force when applied to the question of intestinal parasites. Reflex conditions in the genito-urinary tract have been mentioned. Catarrhal conditions, congenital anomalies, bladder and kidney stones (even in early childhood and more particularly in rachitic tuberculous children), and finally congenital kidney disease are factors.

**Infectious Diseases.**—Of the infectious fevers, pneumonia, scarlet fever, whooping cough, and smallpox are most frequently associated with convulsions. In the acute exanthemata the convulsion is most frequently seen as an initial symptom, whereas in pleurisy and more particularly whooping cough, it occurs in the course of, or more toward the end of, the disease. Even in the exanthemata, however, it may occur with the eruption, or more often as the terminal phenomenon. This is more particularly true of scarlet fever, in which kidney complications are not infrequent. Some authors (Hall, Vogel, etc.) even go so far as to state that convulsions in the course of scarlet fever are always of renal origin. The initial convulsion may be quite independent of the intensity of the infection or the

degree of fever. It should be stated that in some epidemics convulsions are much more frequent than in others. In some epidemics of whooping-cough, for instance, the convulsions have been absent, whereas in others a relatively large number have been affected. The initial convulsion occurring during the course of whooping cough is of much more serious import than the initial convulsions of other infections. In addition to the toxic factor, the mechanical obstruction to the return of blood from the brain often leads to capillary oozing or even gross hemorrhage. In influenza a non-suppurating encephalitis has been found.

As to other diseases, convulsions are sometimes present in typhoid fever, malaria, diphtheria, bronchitis, dysentery, cholera infantum, pyæmia, and septicæmia. In miliary tuberculosis with a meningeal complication, epidemic cerebrospinal meningitis, and typhoid meningitis the convulsions of a toxic nature should be differentiated from those of organic cerebral lesions.

**Drugs.**—Alcohol may act directly when given in overdosage or by ingestion from the milk of the mother. It is unnecessary here to call attention to the overdosage of opium and its preparations, atropine, santonin, strychnine, and other drugs.

**Emotional.**—Fright or other strong emotional disturbance may be the determining factor. Under chorea and the other neuroses, the influence of fright as a factor has been considered. In infancy and early childhood convulsions are sometimes precipitated by a strong emotional disturbance. That we are here dealing with a disturbance of metabolism as well as a sudden shock to the nervous system is evidenced by the production of convulsions in suckling children after strong emotional disturbance in the mother.

**Constitutional Conditions.**—*Rickets.*—That this is an important factor in the production of convulsions in middle childhood cannot be denied. Too much importance has, however, been attributed to it in the convulsions of infancy. It does not, as a rule, appear until the latter half of the first year of life, whereas the most serious form of infantile convulsions appears much earlier. The effect of rickets upon the general development and more particularly in retarding the development of the nervous system renders the latter susceptible to peripheral irritation and other complicating factors, owing to the lowered stage of the nutrition. It is much more common in the form of laryngeal spasm than as a general convulsion.

*Congenital Heart Disease.*—Convulsions not infrequently complicate marked cardiac weakness. These may be due to an anæmic condition of the brain, or, more frequently, to long-continued, passive congestion in the cerebral veins. Toxic factors, the result of general disturbed metabolism, may play an important part as in kidney and liver disease. Renal insufficiency as a result of cardiac inefficiency may be quite as potent as actual kidney disease in the production of disturbance of the central nervous system.

*Thymus.*—Local or laryngeal spasm and generalized convulsions with a fatal termination have been found at autopsy to be associated with enlargement of the thymus. Some of the cases were associated with rickets. Two theories have been adduced to explain the condition; the toxic theory—the most probable—and the mechanical theory, which explains the condition as due to pressure of the enlarged thymus upon the trachea, lungs, the large vessels, or the pneumogastric nerve.

**Symptoms.**—The clinical picture varies greatly, dependent upon the intensity of the convulsion and the time of life. In all cases we may assume a complete loss of consciousness, although this is difficult to prove in early infancy. The earlier the age at which the convulsion occurs the more of the tonic and the less of the clonic element is present. The relative or complete absence of tonic movements has given rise to the term "inward spasm," so much in vogue by mothers, although this term is sometimes used to designate the minor laryngeal spasms with slight rigidity. The convulsions vary in intensity from a temporary loss of consciousness with a fleeting spasm, which may be general or local, to a prolonged convulsion with both tonic and clonic movements. In the more severe forms the onset may be sudden, without previous warning, or more rarely there may be prodromal symptoms, which consist of extreme restlessness, irritability, and minor motor phenomena, such as twitching of the hands, feet, eyelids, and face. In the mild attacks a marked pallor of the face is first noticed, the eyes become fixed, at times roll up in the orbit, the breathing is shallow, the body becomes rigid, a few twitchings of the face and the extremities occur, and the attack is terminated by a sudden deep inspiration.

In the more severe forms the motor phenomena become more marked. The head is retracted, the body arched into a position of opisthotonos, the hands are clenched, the thumbs are buried in the palms, the elbows are flexed, the legs are rigidly extended, and quick spasmodic movements of the extremities occur. The respiration is shallow, but may be interrupted and spasmodic. The pulse is weak, hardly palpable, varies in frequency, and may be irregular. The contraction of the facial muscles causes a succession of grimaces, and may lead to violent contortions of the features with contractions of the eye muscles. There is some frothing at the mouth, the skin is clammy, but later the face reddens and the collection of mucus in the throat produces a coarse, rattling sound. The bladder and the rectum may be emptied; the urine during and succeeding the attack is scanty and may contain albumin. Consciousness is lost during the attack, which usually passes over into a stuporous condition, although this latter is sometimes absent. A condition of general relaxation with marked prostration may follow the attack or may be absent. In most cases the convulsions become general, even when they start as local manifestations. It is not infrequent, however, to see cases in which the convulsive movements are localized to one extremity, the face, or the side of the body. These local convulsions apparently have as deleterious an effect upon the nervous system as the generalized forms. In a recent case of Dr. Fife's, which the writer had the privilege of studying, a baby, aged eighteen months, had as many as forty convulsions a day, all of which were localized to one-half of the body. Transient paralyses are sometimes seen, and have been ascribed to exhaustion of the cortical cells. Permanent paralysis in one or more extremities is sometimes observed, and is due either to actual hemorrhage into the cortex, or in some cases to areas of encephalitis. Isolated convulsions, as a rule, last for a very short time; clonic movements may, however, persist, and in some cases may be prolonged for half an hour or longer. In exceptionally rare cases, more particularly in later childhood, twitchings of certain muscles may persist as a chronic affection. Successive convulsions may follow each other in rapid succession, and as high as eighty convulsions have been noted in a single day (Holt). One matter ought to be distinctly kept in mind with

reference to partial convulsions, namely, that isolated or Jacksonian convulsions are not to be considered as evidence of a focal lesion in infancy and childhood. In the case above recorded a slightly degenerated condition of the ganglion cells was all that was found at autopsy.

Death may result from a single attack; this, however, is a rare occurrence, and only occurs in very young infants or in those in whom the vitality is very much lowered from rickets or other diseases. Death may, however, ensue from exhaustion after long or often repeated convulsions.

The paralytic phenomenon above mentioned is the most common sequel of a convulsive attack. Posteclamptic blindness has been reported in a small number of cases; Cautley found 8 under eighteen months of age, and 3 between two and a half and three years of age. Temporary aphasia was present in one of these. Ten of the eleven collected cases recovered their sight. In none of the cases examined was there any evidence of disease of the optic nerve.

**Diagnosis.**—The diagnosis of a convulsion in infancy and childhood is not a matter for serious consideration. The differentiation into types is also one of relatively little importance. The problem before the physician is as to the cause of the condition and the possibility of its removal. In the consideration of this subject we have purposely omitted convulsions due to gross cerebral disease, but these ought to be kept in mind, and in the event of recurrent isolated convulsions careful examination of the eye-grounds, of muscle balance, of reflexes, and muscular power ought to be made. Convulsions complicating brain diseases without an acute meningitis are not, as a rule, accompanied by a marked rise in temperature. Convulsions immediately after birth, and more particularly after a prolonged and difficult labor, suggest, as a rule, the presence of cerebral hemorrhage. Convulsions usually indicate the onset of some acute disease when they occur in a child over two years of age in association with a high temperature and clinical evidence of some acute infection. A reflex and toxic factor is sometimes here intermingled on account of the associated conditions of the gastro-intestinal tract, and may to a certain extent cloud the diagnosis.

The diagnosis of reflex convulsions from worms, phimosis, etc., becomes a matter of simple observation. Rickets is frequently overlooked. These conditions should, however, always be kept in mind. In an investigation of what was thought to be a small epidemic of epidemic cerebrospinal meningitis in a home for children, those affected were found to be suffering from rickets.

One of the most difficult problems is the differentiation between eclampsia and epilepsy. This is a most difficult matter. While it is impossible to diagnose the fits of infancy as definitely epileptic, it is equally impossible to state that repeated eclamptic attacks in childhood will not persist in later life as epileptic attacks. The physician is permitted to state that in a single convulsion or group of convulsions in infants associated with a definite toxic or reflex factor, the presumption is in favor of normal nervous health in the future. If the convulsions are continued in later childhood, even with a reflex toxic or febrile factor, while the probability under proper conditions of schooling, hygiene, etc., may be in favor of freedom from convulsive seizures, serious stress or insult to the nervous system may develop and thus lead to the convulsive habit. The so-called idiopathic convulsions are more likely to be of a true epileptic nature. Convulsions during the

first year are more likely to be of an eclamptic than of an epileptic nature. As childhood advances, the probability is more likely to be in favor of epilepsy; this is more particularly true when heredity is bad and there is evidence of a degenerate nervous system, a distinct aura preceding the attack, with an initial cry and a recovery to a relatively normal condition of health immediately after the attack. It is hardly necessary to call attention to the need of a careful examination of the urine to exclude renal disease, or of the blood to exclude anæmia.

**Prognosis.**—As far as the individual attack is concerned, this must depend upon a study of the pulse and respiration, the length of the attack, the grade of exhaustion, and the degree of cyanosis and stupor. Cyanosis is an important symptom in the convulsions complicating disease of the respiratory tract. In nephritis the prognosis will depend on the severity of the renal disease and the degree to which a relatively normal efficiency of kidney function can be secured.

**Treatment.**—This depends upon a study of the causative factors, their removal, and a study of the isolated attack.

Convulsions in infancy and childhood must always be considered as a dangerous emergency condition. There should be no delay in attendance, and no procrastination in active treatment. If the convulsion is of the simple variety, and not dependent upon passive cerebral congestion, the child should be immediately placed in a quiet, darkened room, and the body placed in a mustard bath if easily accessible, or, what is more usually convenient, a mustard pack. The combination of a mustard pack and a hot foot bath, continued until there is distinct red skin reflex, will often be found to be all that is necessary in the mild convulsion. If the convulsion does not rapidly subside, the inhalation of chloroform should be immediately begun. If there is a history of constipation or overfeeding, the lower bowel should be emptied by a simple enema, or in older children by sodium or magnesium sulphate. Immediately following the emptying of the bowel, chloral and bromide should be injected. Three or four grains (gm. 0.25) of chloral and from 5 to 10 grains (gm. 0.3 to 0.6) of potassium bromide should be given to a child aged six months, and repeated in an hour if necessary; in older children a larger quantity in proportion may be administered. The injection should not exceed one to two fluidounces in bulk (30 to 60 cc.), should be injected high, and prevented from escaping by tight pressure on the buttocks. This can be repeated within an hour if necessary. If the injection is not retained, or if the convulsion should continue in spite of it when the chloroform is withdrawn, morphine should be given hypodermically (gr. $\frac{1}{40}$ at six months, repeated in an hour if necessary, gr. $\frac{1}{20}$ at one year, gr. $\frac{1}{16}$ at two years, with an increase in this dosage if the occasion requires it). If the attack has subsided, the child should be kept in a quiet room for a few days, the bowels should be thoroughly evacuated, a light diet should be administered, and small dosage of bromides given for at least a week. If the convulsions show any tendency to recur, antipyrine may be combined with the bromides. A hot bath or a hot pack is contra-indicated in convulsions complicating advanced pulmonary disease, with extensive disease or collapse, or when the fit is due to syncope. Careful search should be made of all the possible sources of reflex irritation, and these should be immediately corrected. The child should be carefully watched, and its health kept up to a normal standard.

# CHAPTER XV.

## ACUTE ENCEPHALITIS AND BRAIN ABSCESS.

By E. E. SOUTHARD, M.D.

### ENCEPHALITIS.

**Synonyms.**—Inflammation of the brain; acute red softening of brain; brain fever; cerebral fever; cerebritis; phrenitis; phrenesia; phrenetiasis; phrenicula; cerebral phlegmasia; *calenture; febbre perniciosa frenetica; suppuratio occulta cerebri.* The terms brain fever, cerebral fever, and the like frequently refer to meningitis. Conversely, discussions of meningitis frequently contain data concerning encephalitis. Papers on meningo-encephalitis, encephalomeningitis, poliencephalitis, leukencephalitis, encephalomyelitis, poliencephalomyelitis, mycosis of brain, as well as those on brain abscess, hyperæmia and congestion of the brain, pernicious malaria, central neuritis, softening of the brain, and encephalomalacia contain pertinent material.

**Definition.**—Encephalitis means inflammation of the encephalon or brain. The best usage includes under the term encephalitis exudative inflammations of the brain. This usage has been adopted in the present description, which consequently excludes all those instances of so-called encephalitis in which degeneration, atrophy, and other non-exudative processes are paramount. Many processes described in the literature as encephalitic would be more accurately described under the generic term *encephalopathy.* Accepting this nomenclature, we should regard *encephalitis* (due, for example, to the toxins of the *Staphylococcus pyogenes aureus*) as a subordinate class under encephalopathy, coördinate with, although distinct from, such other subordinate classes of brain disease as *encephalic trauma* (*e. g.*, laceration of brain), *encephalomalacia* (focal type due to vascular plugging), and *encephalic degenerations* (*e. g., encephalopathia saturnina*). The literature contains many references to the above-mentioned subordinate classes under the name encephalitis. Discussions of traumatic encephalitis, of lead and arsenical encephalitis, and (in the older writers) of encephalitis with cyst formation of vascular origin are not uncommon.

**History.**—Phrenitis is the term used by Galen for encephalitis. He calls phrenitis *delirium magnum et continuum cum febre acuta.* In fact, phrenitis appears to have been for Galen a delirium accompanied by continuous fever, whereas mania was a form of non-febrile delirium. *Phrenitis est inflammatio cerebri et meningum ejus, ob ebullitionem calidorum humorum, delirium magnum et continuum cum febre acuta inducens.* Paracelsus appears to have related both phrenitis and mania to the sublimation of mercury due to internal or external heat.

The title *De Encephalitide*[1] appears in medical literature at least as early as 1798 (T. N. Beels), and the title *De Phrenitide*[2] as early as 1615 (H. Petraeus), and these indicate the familiarity of the terms at that time. *Pseudophrenitis* and *paraphrosyne* were used in Petraeus for conditions in which the delirium is not continuous. In the seventeenth century no less than ten monographs appeared on phrenitis and allied topics, and no less than thirty-four in the eighteenth century. Monograph publication on these lines was especially frequent in the first two decades of the nineteenth century. The first systematic treatment of the subject in the modern sense was that of Hasse,[3] in 1855. But this suffers from the confusion, then prevalent, between the effects of vascular plugging and those of true inflammatory exudation.

Most of the work on encephalitis preceding the era of Virchow's work on thrombosis and embolism (1846) can be neglected for modern purposes. Virchow's work began to have wide currency in 1856, but, although it cleared the way for a definition of focal softenings (focal encephalomalacia), the status of encephalitis was no better than before. Nor can it be said that Virchow's contribution of *encephalitis neonatorum* (1867)[4] aided systematic work, since it was at once opposed by Hayem[5] (1868) and Jastrowitz (1870), and cannot yet be said to be a solved problem. Hayem (1868) developed the idea of non-suppurative or hyperplastic encephalitis. Various contributions followed in which the infectious character of encephalitis became probable. Huguenin (1876) contributed a faithful and acute discussion of encephalitis to von Ziemssen's *System*,[6] in which he was able to point out wholly characteristic differences between encephalitis and encephalomalacia, but in which he was far less successful in distinguishing between non-purulent and purulent encephalitis.

Wernicke,[7] in 1881, described the disease (*poliencephalitis acuta hemorrhagica superior*), which bears his name, as an acute inflammatory process about the Sylvian aqueduct region, and compared the process with acute anterior poliomyelitis. With the interest in infectious processes in the early eighties, Strümpell[8] (1884) was able to construct a very productive hypothesis—that many cases of cerebral palsy in children are due to encephalitis of the motor areas. In 1890 Strümpell was able to describe two cases of acute primary encephalitis in adults (autopsies by Zenker).[9]

In 1893 Macewen's work on *Pyogenic Infective Diseases of the Brain and Spinal Cord* appeared, in which the surgical importance of brain abscess and inflammation of otitic origin was developed. In 1895 Nauwerck[10] found an organism of the influenza group in encephalitis. In 1898 Councilman, Mallory, and Wright were able to describe and figure encephalitic lesions which were undeniably due to Weichselbaum's meningococcus, and were in all cases co-existent with frank meningitis.

[1] T. N. Beels, *De Encephalitide*, Lugd. Bat., 1798.
[2] H. Petraeus, *De Phrenitide*, Marpurgi, 1615.
[3] *Handbuch der speziellen Pathologie und Therapie*, Erlangen, 1855, Band iv.
[4] *Arch. f. path. anat., etc.*, Berlin, 1867, xxxviii.
[5] *Etudes sur les diverses formes d'encéphalite*, Paris, 1868.
[6] Hirnentzündung. *Handbuch d. spec. Path.* (Ziemssen), Leipzig. 1876, Band xi.
[7] *Lehrbuch der Gehirnkrankheiten*, Kassel, 1881, Band ii, Th. 3, S. 229 to 242.
[8] *Jahrb. f. Kinderh.*, Leipzig, 1884, vol. xxii.
[9] *Deut. Archiv f. klin. Med.*, Leipzig, 1890, xlvii, 53 to 74.
[10] *Deut. med. Woch.*, Berlin, 1895, No. 25, p. 393 to 397.

The infectious character of several types of exudative inflammation of the brain is well established. But the complete natural history of encephalitis does not yet exist. Ten years after Huguenin's elaborate discussion, Bernhardt, in *Eulenburg's Real-Encyclopädie* (1886), was still able to say that no unitary picture could be presented, even of that kind of brain softening due to vascular occlusion. In 1904 Friedmann stated that recent distinctions of encephalitis into various forms are erroneous, and that all the phenomena of encephalitis can be produced by a great variety of agents, among which are the meningococcus, the influenza bacillus, embolism, aseptic trauma, corrosive substances, and heat.

The interest of the present day is in the bacteriology (parasitology?) of these processes and in the possibility of differentiating cases into groups according to their infectious origins. Clinically, we are as yet unable to define groups with precision according to the different known infective agents. For some years, in Boston, an effort has been made to correlate these points of view. This work started out from the report of Councilman, Mallory, and Wright on "Cerebrospinal Meningitis," and has taken the shape of several studies dealing with the histopathological and clinical effects upon the brain of the Micrococcus aureus (Southard and Sims,[1] Southard and Keene[2]), pneumococcus (Bullard and Sims,[3] Southard and Keene[4]), streptococcus (Southard and Stratton[5]), and *Bacillus typhosus* (Southard and Richards[6]). This work has shown the association of *Staphylococcus pyogenes aureus* and of the pneumococcus with acute hemorrhagic encephalitis, uncomplicated by meningitis.

**Classification.**—We distinguish the acute from the subacute and chronic forms of encephalitis. In the acute form we find characteristically the cardinal signs of inflammation. Of these signs, redness and swelling are prominent postmortem features of acutely inflamed brain tissue. The pain which sometimes accompanies encephalitis is due not to compression of the cerebral tissue itself, but to the pressure incidentally exerted upon the trigeminal endings by the swelling of the brain. Alterations of temperature can be demonstrated under surgical or experimental conditions only. More prominent and important than these signs is the so-called fifth sign of inflammation, or *functio læsa*, which may so affect consciousness at some stages as to remove the feature, pain, which is striking in inflammation elsewhere.

In the *subacute* and *chronic* forms we meet a variety of reactions which have, until recent years, been little understood. Both the histological findings and the clinical pictures in these latter forms have much in common with the degenerative class of encephalic disease. The terminal effect of repair in several classes of encephalic disease (inflammatory, traumatic, degenerative) is atrophy or induration (sclerosis, gliosis), or even porencephaly, which may offer few or no features to determine the nature of the original disease. These points partly explain why there has been so much confusion as to what the term encephalitis precisely denotes.

[1] *Jour. Amer. Med. Assoc.*, 1904, xliii, 789.
[2] *Amer. Jour. Med. Sci.*, 1905, cxxix, 474.
[3] *Boston Med. and Surg. Jour.*, 1904, cli, 647.
[4] *Jour. Amer. Med. Assoc.*, 1906, xlvi, 13.
[5] *Ibid.*, 1906, xlvii, 1271.
[6] *Jour. of Med. Research*, 1908, vol. xix.

*Topographically*, we distinguish *encephalitis* from *neuritis* (inflammation of nerves) and *myelitis* (inflammation of spinal cord or medulla spinalis). Within the skull, we distinguish encephalitis from *(lepto)meningitis* (inflammation of the pia mater) and *ependymitis* (inflammation of the ventricular linings). Encephalitis means, then, strictly speaking, an inflammation of brain substance; but we habitually include also inflamed brain tissue in which there is little or no sign of parenchymal change and the chief sign of injury is perivascular infiltration. Within the brain substance, we might well distinguish between (and do so denominate certain diseases) *poli(o)-encephalitis* (inflammation of gray matter) and *leuk(o)-encephalitis* (inflammation of white matter).

*Chronologically*, we distinguish *acute encephalitis* (of which the types produced by staphylococci, streptococci, and pneumococci have been most studied), *subacute encephalitis* (of which the best examples are seen in syphilis and in tuberculous meningitis), *chronic encephalitis* (*e. g.*, healed cases of epidemic meningitis), and *progressive encephalitis* (perhaps of toxic character, as in dementia paralytica so often following syphilis). These latter forms are not considered in this article.

Since the differentiation of thrombotic from inflammatory softenings, the development of entities in encephalitis has been as follows:

*Encephalitis congenita*, Virchow, 1867.

Spontaneous hyperplastic encephalitis, Hayem, 1868.

*Poliencephalitis acuta hemorrhagica superior*, Wernicke, 1881.

Acute infantile poliencephalitis (infantile cerebral palsy), Strümpell, 1884.

Acute primary encephalitis (adults), Strümpell, 1890.

Pyogenic infective disease of the brain, Macewen, 1893.

Encephalitis (*Bacillus influenza*), Nauwerck, 1895.

Acute hemorrhagic encephalitis (*Staphylococcus pyogenes aureus*, pneumococcus), Southard and Keene, 1905 and 1906.

The most extensive experimental work on the pathogenesis has been by Friedmann. The most influential systematic treatise since Huguenin, 1876, has been Oppenheim's chapter in *Nothnagel's Handbuch*, 1897, now in its second edition (Oppenheim and Cassirer, 1907).

The etiological basis has, so far as possible, been adopted in the classification which follows:

**Etiologically Well Characterized.**—*Influenzal Encephalitis. Pyogenic Encephalitis. (Hemorrhagic Superior Poliencephalitis, alcoholic form.)*

**Etiologically Doubtful, Clinically Definite.**—*Acute Hemorrhagic Encephalitis, Strümpell type*, often pyogenic (?), sometimes influenzal (?), or unknown in origin. *Acute Bulbar Myelitis. Acute Encephalomyelitis. Rabic Encephalitis* (not considered in this article).

**Etiologically and Clinically Obscure.**—*Acute Non-suppurative Encephalitis, Hayem type. Encephalitis in the Newborn.*

To these must be added other forms of such rare occurrence as to forbid systematization. Thus, *Bacillus typhosus* (as well as other typhaceæ), *Bacillus anthracis*, and *Trichinella spiralis* have been found in encephalitis. The encephalitis following scarlet fever, measles, and other exanthems is probably of pyogenic origin. The relation of encephalitis to gonorrhœa, mumps, and whooping-cough is still doubtful etiologically. The encephalitis of tuberculous meningitis, of syphilitic meningitis, of sleeping sickness, and of paralytic dementia is of importance, but is not considered here.

**Influenzal Encephalitis.**—The great pandemic of influenza in 1889 to 1890 led a few writers (Leichtenstern, 1890, 1892; Fürbringer, 1892; Putnam, 1892)[1] to suspect the influenzal origin of encephalitis. Kraepelin contributed a paper on psychoses following influenza in 1890.[2] The influenza bacillus was cultivated by Nauwerck from a case of encephalitis in 1893–94. He found the bacilli in sections from a soft hemorrhagic spot in the cerebellum and in the sediment of the ventricular fluid. The organisms were cultivated from the ventricular fluid. Trouillet and Esprit[3] published in the same year a report of pure cultures from the ventricular fluid in a disease which they term *Meningo-encéphalopathie grippale*. It was suggested that influenza toxins might prepare the tissues for the invasion of other organisms, notably for the pneumococcus, which might be recovered in pure culture from the meninges or brain substance.

The atrium of infection in brain influenza may be: (1) The blood (formerly considered extremely rare); (2) the nasal mucosa and the cribriform plate; and (3) the lymphatics of the middle ear. The nasal mucosa is now considered to be the most frequent route of infection, but it is probable that influenza is frequently a blood infection. Pfuhl found three cases of influenzal encephalitis in an epidemic of 93 cases of influenza in the barracks at Hanover in 1895–96.[4]

Encephalitis is not, as a rule, part and parcel of an attack of typical influenza, but follows recovery or partial recovery from such an attack. Sometimes there is an interval of several weeks before cerebral symptoms set in. In fact, the previous attack of influenza may be quite forgotten or considered as a cold in the head. Oppenheim remarks that the premonitory symptoms of influenzal encephalitis may be hard to interpret, since the headache, vertigo, nausea or vomiting, apathy, drowsiness, and general weakness may be regarded as merely incidents in convalescence from ordinary influenza. In other instances the onset may be acute.

If there is anything which distinguishes this form of encephalitis, it is a gradually increasing loss of consciousness, taking several hours or a day for its completion. The patient can, as a rule, be roused by sharp stimuli, and the pupillary reflexes are normal. The projection system is not characteristically involved at the onset. Meningitic symptoms, such as stiffness of neck and opisthotonos, are exceptional. There is irregularity in the temperature, which is rarely high until the cerebral symptoms are well under way. Some of the febrile temperatures are perhaps due to intercurrent infections (Oppenheim). But these statements were made before the question of influenzal septicæmia had engaged attention. The pulse is likely to be slow at first, but before death quicker, small, and irregular.

The focal symptoms develop, as a rule, after coma has set in, not apoplectically, but one by one (Stadelmann's case with onset suggesting cerebral hemorrhage is a rare exception). Moreover, in a limb about to become paralyzed there are often premonitory symptoms, such as weakness, numbness, or convulsions. Sometimes the convulsions involve the whole side of the body, when the eventual loss of power is to be a monoplegia. The paralyses may be quite masked by coma, or a difference in the two sides

[1] On Multiple Neuritis, Encephalitis, and Meningitis after Influenza, *Boston Med. and Surg. Jour.*, 1892, vol. cxxvii.

[2] *Deut. med. Woch.*, 1890, xi.

[3] *Sem. Méd.*, 1895, No. 21.

[4] *Zeit. f. Hyg. u. Inf.*, 1897, Band xxvi.

may be detected by the occurrence of choreiform movements on one side only. The variety of onset and course in these cases is so great that Leichtenstern feels that there are limiting cases in which a hemiplegia may develop without coma on a truly influenzal basis. Almost every possible focal symptom has been described in cases of encephalitis developing *after* influenza; but motor symptoms far outnumber sensory ones.

It was formerly thought that the prognosis was grave, but Oppenheim is inclined to agree with Leichtenstern and Fürbringer that many patients recover. The relation of influenzal encephalitis to spastic infantile hemiplegia cannot yet be stated; probably many of these cases are the relics of early coccal infections following exanthems.

**Pyogenic Encephalitis.**—Macewen states that pyæmic abscess of the brain may arise from infective embolism originating from pneumonic areas, fetid bronchitis and empyema, fetid pericarditis, infective compound fractures, rarely acute infective periostitis, and occasionally infective ulcers of the intestines and abdominal cavity. In his experience abscesses were rarely multiple except when originating in pyæmia. Macewen makes mention of purulent encephalitis, red softening, and white softening. He describes purulent encephalitis as consisting of a swelling of the cerebral substance with extensive serous and leukocytal exudation and extravasation of red blood corpuscles, which he recognizes to be of very frequent occurrence in acute inflammations of the brain. The coalescence of minute hemorrhages gave rise to red softening according to Macewen. He made the observation that the pressure occasioned by the œdema and swelling of the brain "may *aid* in producing the pain experienced by the patient at the very outset of the encephalitis, especially when the ganglia of the fifth and its recurrent meningeal branches are included in the area of pressure."

White softening is described by Macewen as due to infective embolism. He regarded microörganisms as the chief cause of intracranial pyogenic processes, and emphasizes the middle ear as an atrium of infection. He found the Streptococcus pyogenes and the Staphylococcus aureus most frequently in brain abscesses and in suppurative leptomeningitis. Macewen does not, however, appear to have taken up non-surgical conditions systematically.

In pyogenic encephalitis there is an invasion (whether primary or secondary) of pyogenic bacteria. Although the pyogens in the broad sense include numerous bacterial species, in the writer's experience only the *Staphylococcus pyogenes aureus* and (in a few instances) the pneumococcus have produced pure examples of hemorrhagic encephalitis. Encephalitis complicated by meningitis or meningitis complicated by encephalitis (conditions usually united under the term meningo-encephalitis) are produced by the pneumococcus and streptococcus. In the writer's eight cases of pyogenic encephalitis the encephalitis was produced in six instances by *Staphylococcus pyogenes aureus* and in two instances by the pneumococcus. A novel and interesting possibility in the pathogenesis is an acute inflammatory lesion of the arachnoid villi of the longitudinal sinus.[1] If this could be shown to be the rule, one reason could be assigned for the frequent affection of the central gyri in this disease.

All the writer's cases of *Staphylococcus pyogenes aureus* brain infections

[1] Southard and Sims, *Jour. Amer. Med. Assoc.*, 1904, xliii, 789.

have shown multiple hemorrhages in the brain. In two instances a condition which might be termed bacterial apoplexy has been produced in which rupture of a vessel and solution of surrounding brain tissue has been attended with intraventricular blood. Such a case occurred in a girl, aged twelve years, who had just recovered from diphtheria.

The pneumococcus, although cultivated somewhat more frequently from the brain and meninges than the staphylococcus, is less likely to produce hemorrhagic encephalitis.

In general, it may be stated that this disease occurs at all ages. Cases ranging from five to fifty years occur in the writer's series of pure hemorrhagic encephalitis; cases of meningo-encephalitis range from four months to ninety years of age. Alcohol is an occasional antecedent factor. In children the exanthems frequently precede encephalitis, but encephalitis may appear to be a late complication of an exanthem. Arterial disease appears to have little or no relation to pyogenic encephalitis. The *onset* is more apt to be acute than gradual. The cases of gradual origin in the writer's series were more apt to be in older patients. The course is short, although variable, ranging from twenty-eight hours in a fulminant pneumococcus case to two weeks. The diagnosis of hemorrhagic encephalitis was not made in the writer's cases. The symptoms pointed to lesions of the brain in all cases except two, in which diagnoses of septicæmia and of acute disease of unknown origin were made. Meningitis was considered in two of the Staphylococcus pyogenes cases, and in one of these suspicion of poisoning was raised. In the cases of meningo-encephalitis the diagnosis of meningitis was frequent, and in 1 case a definite diagnosis of meningococcus meningitis was made. In 3 cases of meningo-encephalitis no other diagnosis than that of severe pneumonia was made. In the cases due to *Staphylococcus pyogenes aureus* the onset was sudden in 3 cases (hemiparesis and unilateral convulsions in 1; chilly feelings and frontal headache followed by ecchymoses in 1; malaise, manifold pains, ecchymoses, and cerebral symptoms in 1). In 1 case the symptoms gradually supervened upon a pleurisy and orbital suppuration. One case showed a gradual onset.

The syndromes, as a whole, were of pronouncedly cerebral type in 2 cases, of meningitic trend in 2 cases, of septicæmic character in 1 case. Septicæmia could be suspected in 3 cases in which superficial ecchymoses or suppuration became prominent. The temperature varied for the various cases; at times 103° and 104° for 2 cases; the temperature in 3 cases was never high and frequently subnormal. The pulse was in no case subnormal, and, as a rule, ranged from above 100. The respirations were, as a rule, increased.

The duration of the encephalitic symptoms cannot, in all cases, be closely reckoned; in 1 case three days; 1 case one week (after development of erysipelatoid condition) or three weeks (after injury to eye); 1 case, eight days; 1 case two weeks; 1 case fifteen days; 1 case unknown. Thus, those cases were of longer duration in which the cerebral syndromes were most clearly marked.

The distribution of the brain lesions was as follows: Four cases showed areas of frank and somewhat voluminous hemorrhage, as a rule involving primarily the subcortical region (the vertex in 1, the occipital lobes in 2, and in 1 the pons). Three cases showed multiple ecchymoses and small

abscesses of the cortex or subcortical region, with or without gross evidence of surrounding œdema. In 3 cases œdema of the pia mater was noted. In 2 cases an effusion of blood into the lateral ventricles was noted; in these cases the convolutions were flattened.

In 2 cases, in which the temporal lobe was involved, the middle ears showed no lesion. The mucosæ of the head play no such role in this kind of encephalitis as in the massive brain abscesses made familiar by Macewen.

A remarkable association of encephalitis with focal pulmonary lesions was made out in this series. Pulmonary lesions were either grossly prominent or made microscopically in all six cases. It is possible that this association is a mere matter of coincidence.

**Hemorrhagic Superior Poliencephalitis (Wernicke's Disease; Total Ophthalmoplegia; Nuclear Ocular Palsy; Alcoholic Ophthalmoplegia).**—First described by Gayet, 1875,[1] as *affection encéphalique* (*encéphalite diffuse probable*), localized in the upper parts of the cerebral peduncles and in the optic thalami, as well as in the floor of the fourth ventricle and in the lateral walls of the third ventricle, the disease owes its name to Wernicke, whose cases were published in his *Lehrbuch* in 1881. The entity was further strengthened by Boedeker, who in 1892 summed up the findings in 11 cases (3 apparently non-alcoholic, and 8 alcoholic). The resemblance of the disease to Korsakoff's polyneuritic psychosis has been brought out by Bonhoeffer (1901).[2] Schröder has recently shown that the disease, although a definite entity, is actually not a true encephalitis in the exudative sense of the term.

Wernicke defined the disease as an acute inflammatory disease of the nuclei of the eye muscle nerves, leading to death in from ten to fourteen days. The disease was characterized by palsy of eye muscles of rapid onset, quickly progressing to almost complete ophthalmoplegia (sphincter iridis and levator palpebrarum excepted). The patient's gait recalled that of alcoholism, showing a combination of stiffness and ataxia. Somnolence was a striking feature, either at onset or toward the close. Where somnolence set in late, the disease at first recalled delirium tremens. Optic neuritis occurred in all Wernicke's cases. The course is not always so acute, for Gayet's case lasted five months, and other writers[3] described cases with recovery. The non-alcoholic and the alcoholic forms of Wernicke's disease are considered separately.

(*a*) *Hemorrhagic Superior Poliencephalitis, Non-alcoholic.*—These cases are very few. In Gayet's case the symptoms followed three days after a boiler explosion, in which, however, the patient, a man, aged twenty-eight years, sustained no bodily injury. Paraphasia and paragraphia were the first symptoms. General weakness, apathy, and marked *drowsiness* followed. The face became mask-like, and there was double ptosis with a general atony of the facial musculature, so that in speech there were no expressive facial movements. General weakness was such that the patient could scarcely stand or grasp objects. Pupils, vision, and all kinds of sensation were quite normal. There was almost complete *paralysis* of both *oculomotor nerves*. There was later transitory hemiplegia, which disappeared, leaving the patient in a state of excitement, with more facial expression and pain in the right leg. Conditions varied thereafter. Intelligence

[1] *Archives de Physiologie*, 1875, vol. vii.
[2] *Monatsschr. f. Psych. u. Neur.*, 1899, v, 265.
[3] Wolfe, *Jour. of Nerv. and Ment. Dis.*, 1894; Wiener, *Brain*, 1897, xx, 458.

was maintained, and there were never any considerable changes in the eye grounds. Emaciation followed with incontinence and decubitus; death occurred five months after the accident.

Morbid sleepiness also characterized Wernicke's first case, a young girl, who had attempted suicide with sulphuric acid. The sleepiness, ataxia, optic disturbances, and vertigo came on two months after the suicidal attempt. Oculomotor palsy, disorientation, and hebetude were the main features of the attack, which led to death twelve days after the onset. Salomonsohn's case showed loss of knee-jerks and slowing of the pulse, in addition to other characteristic symptoms. Luce's case was incidental in miliary tuberculosis. Murawieff's case followed three months after influenza, and showed also sarcoma of one precentral gyrus. Zingerle's case had chronic digestive disturbances and an abdominal tumor of long standing.

(*b*) *Hemorrhagic Superior Poliencephalitis, Alcoholic.*—Alcoholic ophthalmoplegia is a disease closely allied in symptomatology with Korsakoff's disease and with alcoholic polyneuritis. Some writers believe that the symptoms in these three conditions scarcely represent entities, but are all incidents in chronic alcoholism. If this be true, it is doubtful whether Wernicke's disease is truly encephalitis in the exudative sense, and, in fact, Schröder was able to find in a typical case, merely, (*a*) destructive lesions due to minute hemorrhages, and (*b*) reparative changes (phagocytic cells, vascular and neuroglial changes) like those following any non-inflammatory injury. Schröder inclines to the belief that alcoholism sets up some kind of vascular change such that, under conditions of unknown character involving severe brain injury, capillary hemorrhages ensue in various parts of the brain. The region beneath the corpora quadrigemina is for unknown reasons the locus of election for such hemorrhages. It is possible that this tendency is related to the characteristic hemorrhages in anaphylactic intoxication.

Subjects having this symptom complex are often brought to a hospital as cases of delirium tremens. The victims are usually male whisky drinkers of middle age, who have been suffering for months or years from chronic alcoholism. For some days or weeks, headache, pains in various parts of the body, vertigo, and vomiting may precede the ophthalmoplegia. As a rule, the ophthalmoplegia is incidental in a characteristic alcoholic delirium, which otherwise differs from the ordinary delirium tremens in the well-marked mental and bodily weakness of the patient.

The ophthalmoplegia is not absolute nor always an associated palsy. Sometimes the pupil and the levator palpebræ superioris are involved as well. Nystagmus is frequent. Wilbrand and Sänger[1] have pointed out that bilateral ptosis occurs in about one-half the reported cases, although it is not an essential feature. One case in six shows normal vision and unaltered eye grounds. The eye ground changes when found, consist either of a slight neuritis with retinal hemorrhages, or of a pallor of the temporal halves of the papillæ (sign of an alcoholic amblyopia due to chronic neuritis of the papillomacular fiber bundle of the optic nerve.

It is difficult to distinguish the rest of the picture from that in chronic alcoholism. *Muscular weakness*, indistinct *schlaftrunken* (Wernicke) speech, and a *gait* recalling the cerebellar gait are characteristic.

[1] *Die Neurologie des Auges, Handbuch*, 1900, vol. i.

In the differential diagnosis, brain tumor (especially of the posterior cranial fossa, particularly in the cerebellum) is suggested by the gait and the eye-ground changes. The course of the disease is perhaps more likely to suggest apoplexy or focal encephalomalacia. Not only the gait and the non-febrile course, but also the alterations of consciousness may support such a diagnosis; and, in the light of Schröder's recent histological observations, the disease may perhaps be best regarded as a peculiar kind of apoplectic disease in which multiple minute hemorrhages affect a particular area. The optic neuritis and the symmetry of the ophthalmoplegia eliminate any question of the classical forms of apoplexy. The ocular palsies of botulism (bilateral paralysis or paresis of accommodation, mydriasis, ptosis) present some resemblances to those of Wernicke's disease.

**Acute Hemorrhagic Encephalitis, Strümpell Type.**—It is probable that many of the cases of acute hemorrhagic encephalitis which Strümpell predicted in 1884 and described in 1890 are of pyogenic origin. Strümpell's first cases showed at autopsy a bilateral bronchopneumonia and a markedly enlarged spleen, in addition to the encephalitis described below. Cultures from the brain lesions proved negative. But Strümpell, after dismissing the hypothesis that his cases belonged to the epidemic meningitis group, assumes that there must be a specific agent for what he conceived was a primary local infection. The possibility of an influenzal origin was not considered by him.

It seems justifiable to put all primary cases of unknown etiology provisionally into this group, which further investigation will undoubtedly resolve. The newer ideas about septicæmia (*e. g.*, in typhoid fever) have so altered our point of view that one naturally hesitates to classify any encephalitis as primary, on the ground that it may result from a blood infection. Since Landsteiner and Popper, and Flexner have successfully transferred anterior poliomyelitis from man to monkeys, there is a possibility that not only anterior poliomyelitis, but also acute encephalitis and Landry's paralysis, may be due either to an ultramicroscopic organism or to protozoa.

Préobrajensky has described the lesions in hemorrhagic encephalitis. In 10 cases isolated foci were found, in 5 other cases multiple foci. The cases with multiple foci were of longer duration. The frontal and occipital lobes are more often attacked than the others. The central ganglion and the temporal lobe are least often injured. No focus was ever observed on the inferior surface of the hemisphere. The cerebellum is more often affected by hemorrhagic encephalitis than by hemorrhage, softening, and syphilis. The foci of encephalitis showed vascular congestion and especially inflammatory changes in the walls of the vessels. Thromboses of the veins are found frequently not only in the brain substance, but also in the meninges, and are more diffuse than the hemorrhagic condition. Valdonio finds inflammatory lesions with thrombi and hemorrhagic lesions and regards the former as primary. He recovered no organisms in two cases.

Orazio D'Allocco has described six cases of acute poliencephalitis in children (one with autopsy). The onset in these cases was sudden, attended with general infectious symptoms. There was a continuous generalized tonic spasm of all the muscles, including those of respiration and of the head, with continual uniform almost rhythmical spasms. The limbs were affected symmetrically, as a rule, but the spasm may be confined to one limb. Spasms are more marked near the head. Muscular excitability is increased.

Anatomically, an endophlebitis and an endarteritis leading to vascular occlusion and degenerative or inflammatory lesions were found.

**Acute Bulbar Myelitis (Acute Inferior Poliencephalitis; Acute Inflammatory Bulbar Palsy).**—*Acute inferior poliencephalitis* has more verbal than substantial resemblance to *acute superior poliencephalitis*, since the non-inflammatory hemorrhagic character of the latter (Schröder) is replaced in the inferior form by acute inflammatory lesions. These lesions recall closely those of anterior poliomyelitis; in fact, many cases of anterior poliomyelitis show bulbar involvement, so that some writers believe that acute inferior poliencephalitis is a disease closely allied to anterior poliomyelitis—possibly an identical infection with an unusual localization. W. Pasteur's[1] epidemic of seven cases of paralysis occurring in children (ranging from eighteen months to eleven years of age) in one family is often cited in this connection, since some of the cases were cerebral and some spinal. At all events, whether the analogy of anterior poliomyelitis is accepted or the frequent association of bulbar myelitis with influenza epidemics be taken as a hint at the etiology, the disease acute bulbar myelitis is very different from Wernicke's disease both in site and in character. Histologically, there is evidence of acute destruction of nerve tissue, not at all confined to the gray matter, together with vascular congestion and lymphocyte accumulations in the perivascular sheaths. Not only do these lesions affect white matter as well as gray, but the meninges are frequently involved.

The *onset* is rapid, but not apoplectiform. General malaise, headache, vomiting, and sometimes fever precede the local signs of bulbar involvement. The characteristic delirium or sleepiness of Wernicke's disease is not found. As a rule, in from three to seven days there is difficulty in swallowing, with paralysis of the tongue and palate, facial paralysis, and paralysis or paresis of vocal cords.

The *course* depends upon the locus of the inflammatory foci, which may directly involve a greater or smaller number of the gray nuclei (nuclear form of palsy), in which there is least hope of recovery, or may affect the nerves in their intrabulbar course (infranuclear form), in which the prognosis is less grave. On account of the ill-defined limits of the inflammatory process, any given case is somewhat likely at its height to show a combination of nuclear, infranuclear, and even supranuclear involvement. If the subject does not become unconscious in his attack, the course is said to be more favorable. The prognosis is, in general, not so unfavorable as was formerly thought, but death occurs in about one-half the cases. The possible relation of certain cases of recovery to multiple disseminated sclerosis has been mentioned (Oppenheim).

In differential *diagnosis*, syphilis, hemorrhage, and softening of the bulb must be strongly considered, whereas tumor, tubercle, and abscess are less likely to cloud the issue. Syphilis is ordinarily attended by basal meningitic symptoms. Hemorrhage and softening have a sudden or rapid onset which is not characteristic of encephalitis.

Some cases with persistent bulbar phenomena have been attributed to encephalitis; thus, for example, Huet and Lejonne[2] attribute a facial palsy and hemiatrophy of the tongue to the effects of a former acute inferior

[1] *Clin. Soc. Trans.*, London, 1897, xxx, 143.
[2] *Rev. Neurol.*, Paris, 1906, xiv, 105.

poliencephalitis attacking the nuclei of the facialis and hypoglossus of the right side. The onset three years before the time of observation had been with convulsions and fever.

**Acute Encephalomyelitis and Poliencephalomyelitis.**—These names have sometimes been given to cases of anterior poliomyelitis complicated by lesions higher than the spinal cord; but there are other cases in which the element of anterior poliomyelitis is not so prominent. Possibly cases of Wernicke's disease with a number of hemorrhages below the level of the quadrigemina have yielded so complex a picture as to deserve this name. Cases of acute bulbar myelitis have been not infrequently complicated by lesion higher than the bulb.

A tendency of the symptoms to *descend*, possibly beginning with ophthalmoplegia, going on with bulbar symptoms, and ending with limb disorders, has been noted. There is great irregularity in the degree to which the very various portions of the involved apparatus are diseased. Whereas total ophthalmoplegia and glossopharyngolabial palsy, associated with spinal palsy, are the symptoms of the disease in its full development, yet in cases in which the ophthalmoplegia is complete the bulbar trouble may be limited to slight facial palsy; and in cases in which the glossopharyngolabial palsy is complete there may be no more than ptosis to express the suprabulbar lesion. Oppenheim has endeavored to reduce these diseases to some kind of system, but as yet with little success.

When the cerebral white matter and the included nerve paths are involved, it seems better to name the disease *encephalomyelitis*. The disease has foci as widespread in some instances as to lose its usual purely motor character (Taylor[1] and Oppenheim). These cases vary extremely in outcome, some dying, some recovering, and some remaining with defects. The probable course of several weeks or longer should possibly align them rather with the slower degenerative processes than with acute encephalitis.

General cerebral symptoms are few or confined to the drowsiness and weakness characteristic of Wernicke's disease. Fever sometimes occurs. The facies is said to be characteristic (Hutchinsonian; eyeballs immobile, ptosis, elevated eyebrows), together with relaxation of the lower face, flattening of nasolabial folds, loose-hung limbs.

A case of Combe's[2] illustrates the peculiar combinations of symptoms possible in encephalitis with multiple foci. It is said to have followed influenza. That form of alternate paralysis known as *Weber's syndrome*, viz., right hemiparesis and left ophthalmoplegia, appeared in a boy, aged two years. Weir Mitchell's *posthemiplegic choreiform movements* followed on the right side, but associated with spasmodic movements in the field of the left facial and abducent nerves, amounting to an alternating hemichorea. Later there was weakness of the right oculomotor nerve. Such hemiparesis with crossed paralysis of the oculomotor nerve with tremors of the paralyzed parts corresponds with *Benedikt's syndrome* (due to a lesion of the cerebral peduncle). But the involvement of the left facial and abducent nerves, together with the right-sided hemiparesis, amounts to the *Millard-Gubler syndrome*. The patient, therefore, suffered from a combination of Benedikt's syndrome and the Millard-Gubler syndrome due to a lesion

[1] *Boston Med. and Surg. Jour.*, 1903, cxlviii, 634.
[2] *Syndrome de Benedikt inférieur, Rev. Mens. d. mal. de l'enfance*, 1904, I, xxii, 1.

of the pons near the left pyramidal tract (this new syndrome Combe proposes to term the *Inferior Benedikt syndrome*), but in addition showed a complication by incomplete ophthalmoplegia, due to a separate lesion situated in the oculomotor nuclei.

It is obvious that the diagnosis of such multiple circumscribed lesions and the determination of their encephalitic nature is a matter for the future. At present the general diagnosis must be made largely by elimination of vascular and other focal possibilities and on the ground of the course of the disease. The poliencephalitis here mentioned evidently has considerable alliance with the frankly degenerative diseases of these loci.

It is under this head that much difference of opinion holds concerning distinguishable entities. E. W. Taylor distinguishes the following in this field: Encephalitis, encephalomyelitis, acute ophthalmoplegia, acute superior and inferior poliencephalitis, acute or apoplectiform bulbar myelitis, poliencephalomyelitis, and poliomyelitis.

It may be mentioned at this point that Batten[1] proposes to group anterior poliomyelitis with encephalitis, and neglecting the preëmption of the term "superior poliencephalitis," or Wernicke's disease, Batten classifies as follows: Acute poliencephalitis superior, including cases with frontal, central, occipital, and cerebellar localization; acute poliencephalitis inferior, including cases in which cranial nerve nuclei are affected; acute poliomyelitis anterior. Batten regards both brain and cord disease as due to thrombosis of fine terminal vessels due to an unknown organism or toxin. Batten's classification is of doubtful value, although there are some cases in which there is a thrombosis of the fine terminal vessels (a prominent feature in pneumococcus and streptococcus brain infections demonstrated by Southard and Keene, and Southard and Stratton). It is not proved that thrombosis plays any part in the disease anterior poliomyelitis.

Comby[2] has described an infection of a seven-year-old girl secondary to influenza. The pharynx was the supposed atrium of infection. This case showed phenomena of poliencephalitis, of poliomyelitis, and of polyneuritis. Such occasional instances of generalized intoxication bring out the possible union in a given case of many neurological phenomena, but they do not prove the kind of unity which Batten and Guthrie[3] have asserted.

Alice Hamilton,[4] in 1902, in connection with a case of Patrick's, has discussed the literature of poliencephalomyelitis, and seems to believe in the unity of Wernicke's disease with more diffuse affections, such as bulbar myelitis, encephalomyelitis, and non-hemorrhagic disease. It is doubtful whether the conclusions of Batten and Hamilton as to the unity of all these conditions can be maintained.

**Acute Non-suppurative Encephalitis (Acute Hyperplastic Encephalitis, Hayem).**—Among non-hemorrhagic forms, Hayem's hyperplastic type is least well recognized and is probably very rare, except in the sense that hyperplastic or proliferative phenomena are the invariable concomitants of all not too severe destructive injuries in the central nervous system.

**Encephalitis in the Newborn (Encephalitis Congenita; Encephalitis Interstitialis Congenita; Encephalitis Neonatorum; Stéatose Interstitielle Diffuse de l'Encéphale).**[5]—Encephalitis in the newborn occurs at times as the result

[1] *Lancet*, 1902, vol. ii.
[2] *Bull. Méd.*, 1906, xx, 41.
[3] *The Clinical Journal*, 1905, p. 176.
[4] *Jour. Med. Research*, 1902, vol. viii.
[5] *Archives de Physiologie*, 1868, vol. i.

of septic infection of gastro-intestinal or pulmonary origin (Fischl). A case is on record in which a pathogenic strain of *Staphylococcus pyogenes albus* was grown from the focal lesions (Fischl). Undoubtedly, also, encephalitis may complicate the picture of syphilis in the newborn, although spirochetæ are seldom demonstrable in the nervous system, even in congenital syphilis.

Aside from pyogenic, syphilitic, or other recognized forms of encephalitis occurring in the newborn, there is another form, the *encephalitis congenita* of Virchow, which is still *sub judice*. Virchow believed that he had learned the cause of death in a considerable proportion of stillborn and newborn infants, attributing it to encephalitis and myelitis of congenital origin. The only analogies for this form of encephalitis were findings in the brains of children of variolous and syphilitic mothers. For eight or nine years prior to 1865 smallpox raged in Berlin, and there were many stillborn infants, showing little or no sign of gross lesion, but microscopically characteristic foci of fat-laden cells in the white matter. On these changes in the children of variolous mothers Virchow based his claim for a new entity, which he thought would attain much medicolegal importance. In general, he regarded congenital encephalitis as a hitherto unobserved brain disease analogous with the parenchymatous changes of the liver and kidney in the exanthemata.

In histological detail Virchow compared the fat-cell foci—termed by him neuroglia cells undergoing fatty metamorphosis—with precisely similar foci described by him in congenital syphilis (1858) and with the syphilitic hepatic foci of Gubler and interstitial fatty changes in the rare disease known as syphilitic interstitial nephritis. Virchow was also much influenced in his interpretation by his finding of primary interstitial changes in the retina in the amblyopia following Bright's disease.

Hayem, 1868, promptly criticised Virchow's findings, noting the extreme frequency of fat-laden cells in the brains of the newborn. Virchow replied that he considered fat cells of neurogliar origin alone of importance in interstitial encephalitis, and printed a plate from a case of *encephalitis congenita variolosa*, showing great numbers of fatty cells between the vessels.

In the same year Parrot published a piece of work, since largely neglected, on what he termed diffuse interstitial steatosis of the encephalon, and offered clinical histories which proved to Parrot's satisfaction that these changes were due to lack of food. Parrot found similar changes in two starved cats. Parrot's results were reprinted without marked alteration of views in his monograph on athrepsia in 1877.[1]

Jastrowitz regarded the fatty cells figured by Virchow and by Parrot as incidental in the development of the brain. From an extensive material (65 cases), Jastrowitz stated that these cells are found in small numbers in the third month of fetal life, in greater numbers in later months, but as a universal finding in the cerebral white matter only in the newborn. The locus of election for the granule cells is the white matter, and especially the corpus callosum and its processes. The occurrence of groups of granule cells outside these usual loci, *e. g.*, in the gray matter of the cortex or in the basal ganglia, was regarded by Jastrowitz as pathological. The pathological fat-cell groups were thought by Jastrowitz to be due rather to

[1] *Clinique des nouveau-nés. L'Athrepsie. Leçons recueillies par le Dr. Troisier*, Paris, 1877.

infections and various maternal diseases than to developmental errors or atrophy (Parrot).

Virchow,[1] 1883, returned to the discussion with fresh data. He was unable to find the fatty metamorphosis constantly (6 of 9 fetuses, 11 of 27 newborn, 14 of 17 children dying after birth showed the change). Moreover, in the focal form of congenital encephalitis he found evidence of nerve fiber lesions.

Modern opinion inclines to the view that the focal form, in which not only masses of fat-laden cells, but also injury of nerve fibers, can be demonstrated is the true congenital encephalitis. A case of the writer's has shown how streptococci may bring about in the newborn a reaction of the mononuclear cells rather than of the as yet ill-developed polynuclear cells in the meninges. Possibly this finding explains some of the cellular peculiarities of congenital encephalitis.

Giraud[2] concluded from a case of athrepsia with convulsions and other brain symptoms that the disease, contrary to Parrot's opinion, is sometimes curable.

**Summary.**—There is a form of encephalitis in the newborn which is at times a cause of stillbirth or of death soon after birth, and is due to various factors (sometimes to pyogenic infection, sometimes to syphilis, and sometimes to unknown intoxications), perhaps more often maternal than non-maternal. The disease is histologically characterized by focal lesions of the cerebral white matter (sometimes visible in the gross) showing nerve fiber alterations and accumulations of fat-containing cells. Protracted cases may yield a neurological picture of irregularly distributed convulsions and pareses, respiratory rather than cardiac disorder, and initial, not continued, fever.

**Experimental Encephalitis.**—The field of experimental encephalitis has not been thoroughly worked. Several men in Ziegler's laboratory have investigated the results of trauma in the brain. Coen[3] demonstrated mitotic figures in the nerve cells surrounding the region of necrosis. Friedmann, in several papers, has described varying phenomena of experimental encephalitis, which in 1904 he summed up by saying that "former distinctions of encephalitis into 'forms' are unnecessary," since sufficiently strong stimuli (meningococcus, influenza bacilli, embolism, aseptic trauma, corrosive substances or heat) can bring about all the different conditions which have been termed encephalitis. In the more severe cases, according to Friedmann, all histological combinations of encephalitic processes may be found ranging from small-cell infiltrations to areas of softening with granule cell deposits and extensive areas of proliferation with giant cells. According to Friedmann, various stages and degrees of these processes have been hemorrhagic, parenchymatous, and hyperplastic encephalitis.

After small traumatic lesions fibrillary sclerosis may follow; after more extensive lesions cellular proliferation is prominent. In cases of hæmatogenous origin, Friedmann emphasizes a primary infection of the small bloodvessels. These have been shown by Southard and Keene, and Southard and Stratton, to characterize pneumococcus and streptococcus infections of the brain. Freidmann, 1904, has given a series of plates illustrating the appearances

[1] *Ueber Encephalitis Congenita, Berl. klin. Woch.*, 1883.
[2] *Thèse de Montpellier*, 1906–1907, No. 56.
[3] *Ziegler's Beitr. z. pathol. Anat.*, 1887, Band ii.

of encephalitis in rabbits. Koeppen[1] has described one form of end results as a kind of *état criblé*, in which the fibrillar gliosis has been insufficient to fill the space of the destroyed tissue. In other cases a dense sclerosis follows.

Farrar has described the histopathological process in the rabbit's brain after introduction of elder pith. He described a passive period, occupying about twenty-four hours after operation, a proliferative period, and a terminal or involutional period (four weeks). Farrar is inclined to disallow the remarkable phenomena of regeneration described by Borst, in which there was said to be observed a new-growth of medullated fibers into small, celloidin blocks placed in the cortex of the rabbit. The phenomena of pia arachnoiditis in the rabbit after application of glacial acetic acid have been described by FitzGerald.

Experimental work upon bacterial encephalitis as yet scarcely admits systematization. Work upon the peripheral nerve roots and the method of invasion of the central nervous system by bacteria has been done by Homèn and Laitinen, and by Orr and Rows.[2] It is evident from the work of these men that more attention must be paid to the lymphatic route of possible infection. The writer and associated workers have produced experimental brain infections in a variety of ways, using several varieties of pyogenic bacteria. They have produced meningitis, encephalitis, and ependymitis, but so far neither a pure encephalitis nor the important lesions of the small vessels found in many cases of adult encephalitis has been produced in the guinea-pig by these workers. The time relations of the experimental brain infections in the guinea-pig are: *Staphylococcus pyogenes aureus*, intrapulmonary injections, after six hours, focal polynuclear exudation in the meninges; after twelve hours, ependymitis; after twenty-four hours, slight polynuclear leukocytosis in the meninges; after forty-eight hours, subependymal leukocytosis and moderate encephalitis (leukocytes in apposition with nerve cells). After three days meningitis, encephalitis, and ependymitis at their height; after four and five days lymphocytosis and phagocytosis; after six days, phagocytosis for leukocytes; after nine days very slight polynuclear leukocytosis, slight evidence of phagocytosis. After one month the tissues are negative, except for slight fibrillar gliosis, pigment-bearing phagocytes, lymphoid and plasma cells, and eosinophilic polynuclear leukocytes.

In work upon the *pneumococcus*, postorbital inoculations were employed. Many strains of pneumococcus gave no lesion. Polynuclear leukocytosis was noted as early as six hours. The exudate is at its height in three, four, or five days, and is largely confined to the meninges. Some of the strains produced lesions in which mononuclear cells were more prominent than polynuclear cells. Work with the *streptococcus* produced results quite analogous to those with the pneumococcus.

Southard and Richards have investigated guinea-pig brain tissues after postorbital inoculation of *Bacillus typhosus*. In contradistinction to the early crisis of the aureus brain inflammations (three days) and the pneumococcus inflammations (four days), it was found that the *Bacillus typhosus*

[1] *Arch. f. Psych.*, 1898, Band xxx.

[2] *Brain*, 1904, p. 460; *Review Neurol. Psych.*, 1906, iv, 25; *Brit. Med. Jour.*, 1907, i, 987; *Review Neurol. Psych.*, 1907, v, 345.

produced a critical phase from the fifth to the seventh day. Mononuclear elements appear in the pial meshes in the typhosus inoculations by the seventh day. By the tenth day fresh exudative cells have ceased to appear. By the fourteenth day the disease has virtually passed and traces only of the old exudate are found.

It is obvious from these pieces of work that, although Friedmann's account of the non-specificity of various described "forms" of encephalitis is, in general, justifiable, it is probable that variations in the rate as well as in the degree of experimental brain inflammations may be determined, and that further qualitative differences may be expected.

Special work has been done by Cantani on the effect of influenza bacilli on the central nervous system of rabbits. He was able to kill rabbits by brain injection of influenza bacillus in proper amount and of proper virulence. The first symptoms appeared nine to ten hours after injection. Paraplegia of crural type was gradually followed by paralysis of all extremities and convulsions. The autopsies showed evidence of severe intoxication throughout the trunk, as well as meningitis, encephalitis, and ependymitis, with demonstrable influenza bacillus. Injections of killed bacilli into the brain in larger amounts produced the same results. It is possible that the rabbit has a special susceptibility of the brain to the influenza bacillus, such as the spinal cord seems to show to the toxin of the Shiga bacillus.

In connection with experimental encephalitis more work is very desirable in animals. Encephalitis in horses has been long recognized; but it is commonly held that equine encephalitis has a very manifold etiology.

**Diagnosis.**—The diagnosis of acute encephalitis is not often successfully made, and often not made at all. Perhaps the diagnosis is rendered more accurately in children's clinics than elsewhere, owing to the interest attaching to the Strümpell type. Nevertheless, in the majority of cases the course is either so fulminant or acute as to offer little chance of diagnosis, or else is such as to warrant a diagnosis of "cerebral disease." In a large number of the writer's series of cases of encephalitis and meningoencephalitis the diagnosis of cerebral disease was made without attempt to differentiate between hemorrhagic encephalitis, massive cerebral hemorrhage, meningitis, and brain syphilis. The attempt of Oppenheim to systematize encephalitis has been so successful that the diagnosis will undoubtedly be rendered more frequently *ante mortem* in the future.

There are few practical difficulties connected with the diagnosis of *hemorrhagic superior poliencephalitis, alcoholic form*, since the history of alcoholism, the acute onset, the associated ocular palsy, the somnolence, delirium, the general weakness (and, as a rule, optic neuritis), stamp the disease. Although Oppenheim has sought to reduce hemorrhagic superior poliencephalitis, alcoholic form; hemorrhagic encephalitis, Strümpell type; and influenzal encephalitis to a single entity on clinical grounds, maintaining that there are intergrading forms uniting all three, yet it does not appear that this position is warranted. Bonhoeffer's original clinical claims and Schröder's recent histological observations go far to show that Wernicke's disease is a rare but perfectly definite disease with definite antecedents and a fairly definite course.

Although Wernicke's disease presents few or no difficulties in diagnosis, the *Strümpell type of acute hemorrhagic encephalitis*, the form termed in

this article *pyogenic encephalitis*, and *influenzal encephalitis* give considerable difficulty.

The endeavor should be made in all doubtful acute "cerebral" diseases to establish or eliminate an influenzal origin. Influenzal encephalitis, it must be remembered, occurs *after* rather than during an attack of influenza. Cases occurring in epidemics are more frequently diagnosed than sporadic cases. Lumbar puncture proves of considerable aid. The data of Cohen's recent work on *septicæmic meningitis* must also be considered.[1]

Since many of the cases may turn out to be of pyogenic origin and of pyæmic nature, it is probable that blood cultures will in the future aid extremely in the diagnosis. The etiological data of cultures from lumbar puncture fluid and from the blood will not point unequivocally to encephalitis. On theoretical grounds the positive lumbar puncture culture will point rather to meningitis or to meningo-encephalitis than to a pure encephalitis. Close attention must be paid to all the clinical signs. If we group together influenzal encephalitis, pyogenic encephalitis, and hemorrhagic encephalitis of unknown origin (termed in this article the Strümpell type), the chief conditions to be considered in differential diagnosis are: (1) Other intracranial infective diseases, such as meningococcus meningitis, tuberculous meningitis, pyogenic meningitis, and sinus thrombosis; (2) brain tumor and brain syphilis; (3) cerebral hemorrhage and focal encephalomalacia; (4) certain conditions of cerebral intoxication resembling meningitis but anatomically negative; and (5) rarely acute poisoning, hysteria, and other diseases.

The differential diagnosis in the first group, *intracranial infective diseases*, is greatly aided by lumbar puncture with microscopic examination of the cells in the centrifuge deposit, and with bacteriological examination. This, in doubtful cases, should include an attempt to grow the influenza bacillus on blood media. The finding of blood in the lumbar puncture fluid may suggest chronic internal hemorrhagic pachymeningitis and cerebral hemorrhage as well as acute hemorrhagic encephalitis. Nor will every case of hemorrhagic encephalitis show blood in the cerebrospinal fluid. Blood obtained through technical accident from the vessels at the point of puncture must not be regarded as indicating true intraventricular hemorrhage. A careful cytological examination, preferably by Alzheimer's[2] or some similar method, should be performed. Examinations for the tubercle bacillus or meningococcus and other pyogenic bacteria should be made. A lumbar puncture fluid without leukocytes, blood, or bacteria must not be considered as excluding the possibility of hemorrhagic encephalitis.

Stiff neck, retraction of the abdomen, and hyperæsthesia are not common in encephalitis. Herpes has not been reported. Paralysis of eye muscles is exceptional. Nevertheless, on account of the extensive variation in foci affected by the multiple lesions of hemorrhagic encephalitis, it is improbable that there is any sign of meningitis which may not also occur in some cases of hemorrhagic encephalitis. The diagnosis frequently depends on the association of a monoplegia or hemiplegia with successive involvement of the paralyzed parts or aphasia coming on early in the disease.

[1] *Annales de l'Institut Pasteur*, 1909.

[2] *Centralbl. f. Nervenheilk.*, 1907, xxx, 499, and Cotton and Ayer, *Review of Neurol. and Psych.*, 1908, vol. vi.

Similar phenomena in meningitis are frequently complications or sequelæ rather than central factors. The acute onset, the drowsiness or tendency to coma, vomiting, convulsions, and optic neuritis offer no points in differential diagnosis against meningitis, nor can much weight be placed upon the condition of the pulse or respiration.

As against *sinus thrombosis* the stasis of blood in the veins of the face has been mentioned by Macewen, but this is not always present. Sinus thrombosis runs a very acute course. The differentiation often fails, except in middle-ear disease.

With respect to the differential diagnosis from *brain tumor*, the onset, the fever, and rapid course are important. Hemorrhagic encephalitis rarely shows an altogether characteristic tumor syndrome. Oppenheim especially warns against the immediate diagnosis of brain tumor in cases showing attacks of cortical epilepsy. Similar remarks apply to brain syphilis when it takes the form of gumma. Brain syphilis in the form of chronic meningitis also runs a quite different course from that of typical hemorrhagic encephalitis. Those rare cases of brain syphilis in which the disease is essentially an encephalitis must give rise to difficulty, except when a history of syphilis can be established. Since these cases occur in secondary syphilis, the differential diagnosis should not prove difficult.

Cerebral hemorrhage and focal encephalomalacia were formerly frequently diagnosed in cases of hemorrhagic encephalitis. In young persons with healthy cardiovascular systems the diagnosis of cerebral hemorrhage or thrombosis may not come into question; but, since hemorrhagic encephalitis occurs in older persons, there is a possibility of confusion. The cases termed by the writer *bacterial apoplexy* occurred in subjects twelve and twenty-two years of age. It is easy to see that in such cases the differential diagnosis between hemorrhagic encephalitis and cerebral hemorrhage would be impossible on theoretical grounds. Practically the paralyses which characterize hemorrhagic encephalitis do not occur simultaneously with the onset of drowsiness or coma. The signs of severe cerebral disease are obvious long before paralyses or convulsions have set in. The paralyses of hemorrhagic encephalitis may increase in intensity from time to time in the course of several hours or a day, presumably owing to the gradual development of inflammatory lesions with oozing of blood. The absence of initial or antecedent fever and of other signs of sepsis will indicate rather a cerebral hemorrhage or focal encephalomalacia due to arterial disease.

Cerebral *intoxication without anatomical signs* (the so-called pseudomeningitis of Krannhals[1]) gives rise to the diagnosis of encephalitis, although more often to the diagnosis of meningitis in some cases. Krannhals' cases occurred in the influenza pandemic of 1889 to 1890. The occurrence of focal symptoms would ordinarily exclude this diagnosis. Poisoning was considered in one of the writer's cases (*Staphylococcus pyogenes aureus*) of bacterial apoplexy, which ran an acute course of three days. Attention to the acute infective feature of encephalitis will usually exclude the diagnosis of *hysteria* with certainty. *Uræmia* and *acetonæmia* may need to be considered.

**Encephalitis in Advanced Years.**—There is neither predisposition nor lack of predisposition to hemorrhagic encephalitis in old age. The diagnosis

[1] *Deut. Arch. f. klin. Med.*, 1895, liv, 89.

as against focal encephalomalacia or cerebral hemorrhage is naturally difficult, especially since febrile conditions (bronchopneumonia) may apparently lend infective features to focal encephalomalacia. Hoppe-Seyler[1] mentions hemorrhagic encephalitis as a rare complication of pneumonia in the aged, and Siemerling, in the same *Lehrbuch*, mentions difficulty in differential diagnosis between sinus thrombosis and encephalitis in the aged. The case of circumscribed hemorrhagic encephalitis reported by Mills, in which there are no data as to etiology, occurred in a woman, aged eighty-three years.

**Encephalitis in Infancy and Childhood.**—The possible medicolegal significance of congenital interstitial encephalitis was mentioned by Virchow. Possibly this question should be raised more often in cases of "overlying." Pediatrics practically distinguishes acute encephalitis in infants and a form of encephalitis in older children which shows more tendency to recovery.

Acute and frequently fatal encephalitis of infants is regarded as a primary encephalitis with origin unknown except in certain pyogenic cases. These cases show high fever, stupor or coma, convulsions at the onset as well as later, superficial breathing with occasional Cheyne-Stokes episodes, a rapid, feeble pulse, and sometimes bulging of the fontanelles. The neck and limbs are stiff and flexed. Strabismus sometimes occurs. The occurrence of slight paresis or monospasm of a limb or of the facial nerve is characteristic. The endeavor should be made to determine or eliminate a pyogenic origin. Lumbar puncture findings are helpful as against the diagnosis of meningococcus meningitis.

The encephalitis of older children is often secondary to exanthemata, diphtheria, or pertussis, and has an insidious origin sometimes suggestive of brain tumor. Recovery is the rule in these cases, which sometimes precede the development of frank epilepsy. The child is immobile except for tetanic spasm; the neck is stiff, and opisthotonos may suggest meningitis. Tremor of the limbs, facial palsy, monoplegia, aphasia, convergence or conjugate deviation of the eyes, and optic neuritis may occur. James Taylor[2] has given valuable data, and distinguishes the following varieties of infantile cerebral palsy:

| | |
|---|---|
| Hemiplegic forms, resulting from encephalitis, vascular lesions and injury. | Acute encephalitis, infantile hemiplegia, infantile monoplegia, idiocy with cranial asymmetry, choreiform hemiparesis, hemiathetosis. |
| Diplegic forms, resulting from death of cortical cells. | Generalized rigidity, idiocy with general rigidity, paraplegic rigidity, spastic facial diplegia, amaurotic family idiocy, bilateral athetosis, choreiform diplegia, epileptiform myoclonus. |

Only the diseases under hemiplegic forms are considered by Taylor to be encephalitic in origin, and of these, infantile (cortical) hemiplegia in the most important. The conditions found, in order of frequency, are acute encephalitis, thrombosis (arterial or venous), hemorrhage, and embolism. The case of Limbeck suggests how porencephaly may be due to

[1] *Schwalbe's Lehrbuch der Greisenkrankheiten*, 1909, p. 236.

[2] *Paralysis and Other Diseases of the Nervous System in Childhood and Early Life*, 1905.

encephalitis. Alzheimer has called attention to the relation between idiocy and encephalitis. According to Taylor the victims of infantile hemiplegia are healthy and without hereditary stigmata at onset, and undergo between the ages of one month and six years severe symptoms, consisting of fever, convulsion, and often vomiting and coma. After a period varying from a day to a week the attack becomes less severe, but hemiplegia comes on first in the face, then in the arms, and lastly in the leg. This hemiplegia is flaccid for a few days and afterward spastic. The paralysis clears up to a considerable extent, but, as a rule, not completely. Such are the primary cases. A second group is incidental in certain specific fevers, and probably corresponds to the group of pyogenic encephalitides. Taylor holds that thrombosis is found in certain cases, and calls attention to the possibility of embolism from endocarditis complicating this specific fever.

Obstetrical injury or other forms of trauma and mechanical rupture of vessels in paroxysms of whooping-cough or in convulsions and meningeal hemorrhage occurring at birth are less important factors. The prenatal cases of infantile hemiplegia are likely to be of thrombotic origin as a result of maternal syphilis. The intrapartum cases are due to obstetrical injury, and the postnatal cases are in general encephalitic.

According to Taylor the following anatomical changes have been found in cases of infantile hemiplegia: Circumscribed atrophic sclerosis (in cases where the nerve elements perish, but the neuroglia survives), cyst formation (in cases where both nerve elements and neuroglia are destroyed), superficial shrunken patches resembling wet wash leather with adjacent atrophic sclerosis (of thrombotic origin), and porencephaly. The neurological phenomena of surviving cases (persistent facial paresis, stunting of growth of arm, choreiform and athetotic movements of arm, characteristic brachial contracture, distortion of bones and joints, posthemiplegic disorders of voluntary and involuntary movements, trophic disturbances and arrest of development, and epileptic attacks) will not be more particularly considered here. About one-half of all cases of infantile hemiplegia are said to be subject to recurrent attacks of epilepsy which begin long after the onset of the hemiplegia.

**Treatment.**—The febrile nature of hemorrhagic encephalitis indicates certain general measures. Although the hemorrhages are, as a rule, of an oozing character, and the progress of focal symptoms depends rather upon spread of inflammatory lesions than upon massive hemorrhages, it is, nevertheless, desirable to avoid any drug treatment which may stimulate the heart. The patient should rest in bed with the head higher than the trunk, and shielded from light and noise. In the somnolent or comatose condition especial attention must be paid to the administration of food and the evacuation of the bladder and intestine. Incontinence occasionally occurs. Cold compresses or ice bags may be applied to the head. Except in cases of chlorosis, venesection or leeches in the temporal or mastoid region are said to have given good results (Oppenheim). Drug treatment is unsatisfactory. Restless patients may require various narcotics or moist packs. Oppenheim warns against surgical treatment and even against lumbar puncture. It appears that there have been few surgical attempts at alleviation of this disease. It might seem that, were accurate localizing diagnosis possible in certain cases of massive hemorrhage, surgery would be helpful. A case of traumatic subcortical hemorrhage reported by Cush-

ing, 1907, gives a hint in this direction; and cases are undoubtedly seen at autopsy in which evacuation of a clot would have been possible. In general, however, surgeons are doubtful of the value of surgical intervention in encephalitis. Auvray speaks of the difficulties in the differential diagnosis of non-suppurative encephalitis and brain abscess, but would not intervene in encephalitis. Operation in meningo-encephalitis (Macewen's case) has been done, and Oppenheim calls attention to Hahn's case (1896), in which trephining was performed under the false impression that abscess or hemorrhage of traumatic origin was present.

Since in the future our diagnoses of encephalitis must improve in the line of etiology, it is possible that especially in the influenzal and pyogenic groups the possibilities of immunization and opsonic treatment may be exploited. The somewhat rare chance of a syphilitic encephalitis must be considered.

## BRAIN ABSCESS.

Brain abscess is a focal destructive disease of brain tissues, characterized by the presence of pus, and is ordinarily distinguished from tubercle, gumma, neoplasm, and cyst of softening, despite the fact that cavities with puriform contents may be found in these conditions. As in the rest of the body, artificial aseptic abscesses can be produced by chemical or physical means within the cranium. Non-experimental abscesses are now generally stated to be invariably due to microörganisms. It is useful to bear in mind, however, that even in man a variety of destructive aseptic lesions can be produced by violence from falls or blows, and that only months or years later these quiescent lesions may be occupied by bacteria. Recent knowledge concerning the frequency of bacteriæmia giving no clinical sign has explained the otherwise mysterious occurrence of latent traumatic abscess.

**History and Sources.**—References, both monographic and periodical, to the subject of brain abscess are very scattering before 1850. Thereafter the work of the otologist Toynbee,[1] of Lebert,[2] of Biermer (particularly contained in R. Meyer's dissertation,[3] 1867), excited interest which was brought to a focus in Huguenin's systematic work of 1876.[4] Surgical interest arose which culminated in the classical work of Macewen (1893). The otological development was maintained by Körner[5] in various publications. The best systematic treatise since Huguenin is by Oppenheim in Nothnagel's *System*, 1897, greatly developed in a second edition of over 300 pages by Oppenheim and Cassirer,[6] 1909. The most striking recent observations are derived from otology, especially from the diagnostic point of view. The bacteriology of brain abscess remains a comparatively neglected field, and most writers feel that cases cannot be clinically differentiated according to microörganisms.

[1] *Med. Chir. Trans.*, London, 1851, xxxiv, 239.
[2] *Virch. Arch. f. path. Anat., etc.*, 1856, x, 78, 352, 426.
[3] *Zur Pathologie des Hirnabscesses*, 1867.
[4] *Von Ziemssen's Handbuch d. spec. Path. u. Therapie*, 1876, Band ii.
[5] *Die Otitischen Erkrankungen des Hirns*, 1902; *Nachträge*, 1908.
[6] *Der Hirnabszess*, 1909.

**Etiology.**—A great variety of organisms has been demonstrated in brain abscess, not all of which are surely concerned in the original production of the lesion, but may settle in preëxistent lesions. Malinowski, 1891, showed that suppurative processes could sometimes be induced in the normal brain of the dog by injections of pyogens, but constantly in brains that had been injured ten days previously.

**Pathology and Pathogenesis.**—Abscesses of the brain occur in all sizes from approximately half the size of a hemisphere down to the barely visible. The smallest abscesses are, as a rule, pyæmic in origin and multiple; they intergrade with encephalitis, the non-suppurative form of which may be so named merely because no pus is visible, whereas, polynuclear leukocytes may be easily demonstrable microscopically. Nevertheless, there is good reason to distinguish such interstitial inflammatory conditions from frank abscess, as in other organs and tissues. The larger abscesses are usually single and are frequently objects of surprise at autopsy, because the volume of "silent" brain tissue and the chance of physiological compensation for destroyed mechanisms are not precisely remembered by the anatomist. The larger single abscesses are apt to be subcortical rather than cortical, and are likely to break through to the surface or into the ventricles late in the disease.

The pathogenesis is by extension from external infected wounds (acute traumatic abscess), by extension from sinus thrombosis, by extension through diseased bone or by lymphatic paths (otitic, rhinogenic, orbital), by blood infection (pyæmic abscesses, abscesses metastatic from bronchiectasis, empyema). The pathogenesis of latent brain abscess is dubious, although there can be no doubt of the part played by blood-borne infection. Occasionally there is an actual demonstration of pigment derived from lung lesions in the metastatic cases.

The abscesses contain, as a rule, a green or yellowish-green, or yellowish pus, generally watery, and in long-standing cases sometimes almost clear. The abscess wall after some days (five or six, according to Friedmann's experimental data, eight or ten days in man) begins to show a membrane. This membrane shows a large amount of collagenous tissue, intermingled with neuroglia in the outer zones. The development of mesodermal reaction in these abscesses is greater than in most other intraneuraxial lesions (solitary tubercle excepted), but for all that there is no tendency to obliteration of the cavity. Such obliteration must be the rarest of events. The investing membrane may be so dense and coherent that the whole abscess may be stripped out, especially from a brain somewhat softened by postmortem change. Frequently death, from rupture into the ventricles with generalized pressure symptoms or rupture into the meninges with a terminal leptomeningitis, intervenes before such membrane formation.

**Symptoms.**—The general symptoms are those of heightened intracranial pressure, and in the main resemble those of brain tumor. Deeplying abscesses tend to heighten intracranial pressure more than superficial abscesses. Subtentorial abscesses produce a general heightening of pressure by means of hydrocephalus. The general symptoms to be considered are:

1. *Headache.* It is rare for a case of brain abscess to run a complete course without headache. Even in the latent period of chronic abscess headache may occur periodically. The onset of fever, attacks of coughing and sneezing, the use of alcohol and coffee are likely to increase the pain.

The headache is apt to be of the dull, bursting type, and is inclined to localize itself upon the side of the abscess. Cerebellar abscesses yield, as a rule, a regionary headache, but there is a surprising number of cerebellar cases with frontal headache. A certain number of cases, perhaps the majority, show localized sensitiveness upon pressure or percussion over the seat of the abscess. During the period of encapsulation the headache of abscess is perhaps less severe than that of tumor; but rapidly spreading abscesses yield headaches of maximal intensity.

2. *Vomiting.* Vomiting occurs in practically all cases, as a rule at the time when headache is most marked, certainly without relation to the condition of the stomach, and often apparently excited by a change of position. Subtentorial abscesses are perhaps more prone to show this symptom.

3. *Vertigo.* Vertigo, unlike headache and vomiting, is inconstant in brain abscess, and perhaps characterizes only a minority of cases. Moreover, since the majority of cases with abscess symptoms give at least a suspicion of being otitic in origin, the vertigo runs a chance of confusion with aural phenomena.

4. *Slowing of Pulse.* At the height of the disease the pulse is usually subnormal (perhaps 50 to 56). Pulse rates of 40 and 30 occur, and rarely it may diminish to 10. A slow pulse is especially helpful in the differentiation of brain abscess and local ear disease and sinus thrombosis.

5. *Respiratory disorder*, usually with tendency to slowing, is not infrequent, particularly in cerebellar abscesses.

6. *Eye-ground Changes.* These are less common and severe in brain abscess than in tumor. They are found, as a rule, when the other symptoms of brain abscess are pronounced.

Generalized convulsions occur in a minority of the cases. Cerebral abscesses in children appear to cause convulsions more often than brain abscesses in adults. Late in the disease and in cases in which the pus bursts into the ventricles convulsions are more common.

Hebetude, sleepiness, indolence, and an appearance of fatigue are characteristic. Absolute coma occurs late and may occur repeatedly in a given case. Episodic attacks of restlessness, confusion, or excitement may set in at any time, and are characteristic of the initial stage in cases having an acute onset. They are apt to accompany attacks of fever. Cases suggestive of the psychiatric diagnosis of delirium are rare. The latent cases may suggest melancholia.

*Temperature.*—Normal temperature may persist throughout, and periods of subnormal temperature characterize some cases. The initial stages of cases having acute onset usually exhibit fever. As Oppenheim suggests, the insidious onset of many cases and the subsidence of fever before the physician is called in many others have led to the idea that brain abscess is characteristically non-febrile. Naturally the ventricular and meningeal complications of the terminal stages may lead to fever. Macewen's data show that the majority of cases throughout observation show normal or subnormal temperatures. Perhaps the latent encapsulated abscesses of a chronic type are those which most characteristically fail to show fever. Certainly a persistent high temperature or a typically intermittent fever is not characteristic of uncomplicated brain abscess, that is, abscess without meningitis or otitis.

**Focal Symptoms.**—Although cases of brain abscess sometimes fail to yield focal symptoms, yet the majority yield fairly characteristic symptoms which often betray the true nature and origin of the lesions. Thus the occurrence of motor symptoms is most characteristic in traumatic and metastatic abscesses; whereas frontal abscesses are characteristically rhinogenic; temporal and cerebellar, otogenic. A few metastatic and traumatic abscesses have been found localized in the occipital region. Abscesses of the pons, the medulla, and the brachium conjunctivum are decidedly uncommon. Local variations in the relatively normal tissue surrounding abscesses are commonly stated to account for rapid modifications in the symptom picture, and for the immediate diminution in symptoms following surgical drainage of abscess cavities.

1. *Focal Motor Symptoms.* These are: (*a*) *Epileptic symptoms* of the Jacksonian type whose distribution may easily serve to indicate the spread of the abscess. These convulsive phenomena naturally occur more often with the cortical than with deeper lesions. The possibility is always to be borne in mind that the convulsions are due not to destructive cortical lesions, but to encephalitic or meningitic changes overlying abscess. (*b*) *Paralytic symptoms* tend to be monoplegic, and, if hemiplegia eventuates, the parts are successively and not simultaneously involved. *Disorders of sensation* are often masked by the patient's mental state.

2. *Frontal Symptoms.* These are frequently absent or hard to differentiate. The vicinity of the motor zone permits, in the midst of indeterminate symptoms, the development in later stages of brachial or facial symptoms. Conjugate deviation of head and eyes toward the side of lesion has been noted in connection with epileptic attacks. The mental symptoms which one might expect and which occasionally arise in the brain tumors so localized have generally failed of demonstration.

3. *Temporal Symptoms.* Temporal abscess is practically never found unexpectedly at autopsy in clinical cases properly observed. The data concerning contralateral and homolateral disturbances of hearing are still equivocal, both on account of the sensorial state of the patient and on account of the frequent involvement of the peripheral auditory apparatus in these cases. Aphasic symptoms are characteristic in left-sided lesions. The sensory aphasia of Wernicke, as Wernicke himself pointed out, does not ordinarily develop from the temporal lobe abscess as it usually occurs, since the cortex and white matter of the superior temporal gyrus are not usually involved until late. Oppenheim has pointed out that the lesion characteristically cuts the association paths rather than the auditory centres themselves, so that an amnestic aphasia supervenes in which the patient speaks little and has lost command of many words. The understanding of terms for simple things remains intact. Slight paraphasia and inability to name objects, with maintenance of power to repeat words, are shown. The abscess may disconnect the auditory and the optic centres and thereby effect a partial word-deafness with maintenance of the understanding of words without visual content. Hemianopsia may be produced by deeper lesions. Contralateral motor phenomena are paralytic rather than epileptic in character, and are probably best regarded as due to involvement of the internal capsule. Muscular rigidity, increased deep reflexes, diminution in the abdominal reflexes, development of the Babinski reflex occur. Macewen and Körner have pointed out that oculomotor palsy is frequently

found on the side of the lesion. Oculomotor palsy and contralateral facial and brachial monoplegia, with other abscess symptoms, point to temporal localization.

4. *Cerebellar Symptoms.* The most important cerebellar symptoms cannot be precisely spoken of as focal; but cerebellar ataxia, Babinski's cerebellar asynergy, general muscular weakness, occasionally hemiataxia, Babinski's adiadochokinesis, forced movements, peculiar attitudes of head and body, vertigo and vomiting, nystagmus, convulsions, and various effects of compression of lower structures may be found. Pain in the back of the head and nuchal region, retraction of the head, and stiff neck are more characteristic of cerebellar abscess than of other types.

5. *Occipital Symptoms.* Beyond hemianopsia and possibly word-blindness there are few characteristic symptoms from occipital abscess.

Further localizing points are mentioned under the diagnosis of abscesses classified below as of various origin: (1) Traumatic. (2) Otitic. (3) Metastatic. (4) Rhinogenic. (5) The so-called idiopathic brain abscess.

**Diagnosis.—Traumatic Brain Abscess.**—Traumatic brain abscesses form about one-quarter of reported cases (Church and Peterson). They are likely to be superficial and situated directly below the locus of trauma, and naturally affect the convexity, particularly the frontal and parietal regions, more often than other parts of the brain. In contradistinction to localized traumatic leptomeningitis, which may develop very shortly after the trauma, traumatic brain abscess begins to show symptoms only after several days (*acute traumatic brain abscess*) or after a long latent period of weeks to years (*latent traumatic brain abscess*).

In the *acute traumatic form*, the interval between injury and development of frank *symptoms of abscess* may be occupied by signs of brain laceration or by certain meningeal symptoms, followed by a somewhat sudden development (several days to two weeks after trauma) of the signs of heightened brain pressure with fever. Whereupon, with the development of focal signs, the picture of abscess becomes complete. Involvement of motor parts of the brain is obviously more often the rule in traumatic than in other forms of brain abscess, so that both the history of trauma, with local appearances, and the course of the disease, with its focal signs, somewhat simplify the diagnosis. Nevertheless, it may not be easy to tell such cases from cases of suppurative meningitis. It is said that muscular hypertonus and generalized hyperæsthesia are less apt to develop in abscess cases. The cortical epilepsy or monoplegic symptoms, which are not infrequent in encephalitis or with cortical cysts of softening, sometimes occur in traumatic brain abscess, but speedily develop into hemiparesis or hemiplegia, possibly with aphasia. Sometimes the hemiplegia comes on suddenly. Again, there are rather characteristic short remissions which indicate that the process of abscess development is by no means so free as that of a meningitis of similar localization. Oppenheim states that the focal signs of brain abscess are less likely to be masked by general signs than are the focal signs of meningitis, an observation which probably signifies that abscess may proceed for many hours or days without interfering with intracranial pressure. The fever which may characterize the initial onset of pressure symptoms need not be a prominent feature in the later course. In fact, absence of fever as well as slow pulse are commonly thought to favor the diagnosis of abscess when the symptoms of the disease are fully developed. The

frequent co-existence of abscess and localized meningitis, as emphasized by Huguenin, must not be forgotten in the analysis of symptoms. In the absence of surgical interference, death may ensue within three weeks.

In the *latent traumatic form* we find a similar preference for frontal, central, or parietal localization, but, although these latent abscesses are certainly related to overlying local trauma, yet they are not due, in the opinion of most writers, to direct infection from superficial injured tissues. Possibly subinfection with organisms, circulating in the blood but derived from some quite different source, may account for the lighting up of an acute inflammation in the mechanically injured brain tissue. These foci would otherwise, and doubtless often do, gradually heal and in the end produce quite imperceptible functional changes. A careful inspection of the latent periods in such cases may often reveal suggestive symptoms, as a rule, of an irritative or "functional" character. The frank onset of the disease is attended with headache, perhaps localized and accompanied by pain on percussion of the adjacent bone. Vomiting, vertigo, tremors, slow pulse, are characteristic, and a large number of cases fail to show fever, but may even exhibit a subnormal temperature. Such abscesses are consistent with but slight alterations of consciousness, but investigation usually discovers some change, if nothing more than unusual somnolence; such changes when observed are often variable. Changes in the eye-grounds are not constant. Emaciation and gastro-intestinal disorders are frequent. There is great possibility of confusing such latent abscesses with brain tumor; in the differential diagnosis we are aided by the occasional febrile attacks, the rapid progression, and the lack or late development of eye-ground changes in abscess.

A recent instance of blood-borne infection of intracranial abscess with Bacillus typhosus in a traumatic case under treatment for typhoid fever has been reported by Gurd and Welles.[1]

**Otitic Brain Abscess.**—One-third to one-half of all cases of brain abscess are said to follow purulent otitis media. Of course, only a small proportion of such otitides give rise to brain abscess, and then often many years after the onset. Mastoiditis, temporal bone disease, and the so-called cholesteatoma (of the otologists), and cases of otitis media which do not secure free drainage (either naturally or surgically) are especially dangerous. Less frequently brain abscess follows immediately upon an acute otitis media. A somewhat rare complication is brain abscess ensuing upon the temporal bone disease found in diabetes. Otitic abscesses of the brain are rare in cases which have never undergone perforation.

Like the traumatic forms, otitic brain abscess is usually solitary. Its characteristic locus is the temporal lobe, and if in the right temporal lobe it may yield no localizing sign, but if in the left, may yield word deafness or sensory aphasia, permitting accurate local diagnosis. Naturally, the general symptoms may hinder the detection of this feature. Let such an abscess spread inward, and a spastic hemiparesis, possibly associated with hemianæsthesia and hemianopsia, will ensue, as a result of pressure upon, or destruction of, the underlying white paths. Oculomotor and abducent disorders, especially ptosis, may be associated with the other symptoms through pressure upon the corresponding nerves.

[1] *Annals of Surgery*, 1908, xlvii, 4 to 9.

Temporal lobe abscess is about twice as frequent as cerebellar abscess following ear disease (Heimann,[1] 428 temporal, 198 cerebellar; Neumann,[2] 336 temporal, 196 cerebellar). Cerebellar otitic abscesses in about nine cases out of ten (Neumann) produce symptoms. One group of symptoms (*vestibular nystagmus, vertigo,* and *vestibular ataxia*) is dependent upon involvement of Deiters' nucleus. Inasmuch as labyrinthitis and cerebellar abscess often co-exist, the diagnosis of vestibular nystagmus may be difficult. The ataxia of cerebellar otitic abscess is, according to Bárány, a characteristic compound of vestibular and cerebellar features. Peculiar correlations between the direction of nystagmus, the position of the head and trunk, and distribution of ataxia have been described by the otologists. Nystagmus to the diseased side, described as Neumann's sign, has been confirmed in three of five cases of cerebellar abscess by Hegener. Another group of symptoms (hemiparesis and hemiataxia) depends upon interruption of white paths in the cerebellar tissue. Rapid emaciation and anæmia have characterized many cases (Okada). Uncomplicated cases are apt to run an afebrile course. A slow pulse, without correspondence with such fever as may occur, as in other processes, shows heightened intracranial pressure. Occipital pain is *not* especially characteristic of cerebellar abscess; on the contrary, frontal pain is not infrequent (spread of irritation from a recurrent branch of the ophthalmic division of the trigeminal nerve, supplying the tentorium, to other branches of that division, Krause).

Temporal lobe abscess and cerebellar abscess are often difficult to distinguish. Hemianopsia is uncommon in cerebellar abscess. The speech disturbances of cerebellar abscess are rather bulbar than cerebral in character. The hemiparesis of cerebellar abscess is homolateral; of temporal abscess, contralateral. Palsies of isolated eye muscles are the rule in cerebellar abscess, whereas ptosis, external strabismus, and oculomotor palsy characterize the temporal cases. The pain of temporal lobe abscess cases is temporal or parietal, as against the occipital or frontal pain of cerebellar abscess. Nystagmus, stiff neck, and sensitiveness of neck tissues are characteristic of cerebellar cases.

The law of Toynbee, according to which disease of the tympanic cavity gave rise to cerebral abscess, whereas disease of the external meatus threatened rather the lateral sinus and the cerebellum, and labyrinthine disease was likely to lead to bulbar involvement, has been much modified by more recent knowledge as to possible paths of infection. Huguenin, for instance, pointed out that the cerebellum could be involved directly along the facial and auditory nerves.

Cases of Stokes, of Habermann, and of Bloch and Hechinger indicate that the sense of smell should be tested in this group of cases. Twice the anosmia was homolateral with abscess, once contralateral. Dench[3] has published an analysis of 102 recorded cases of cerebellar abscess (33 recoveries) and 100 cases of cerebral abscess (52 recoveries) of otitic origin.

**Metastatic Brain Abscess.**—The diagnosis of metastatic brain abscess (about 10 per cent. of all cases, Gowers) must take into account the predominant sources for embolism. Bronchiectatic abscesses, pulmonary gangrene, lung abscess, pleural empyema, ulcerative endocarditis, pyæmic

[1] *Arch. f. Ohrenh.*, 1905, lxvi, 67.
[2] *Der otitische Kleinhirnabszess*, 1907.
[3] Otitic Brain Abscess, *Amer. Jour. Med. Sci.*, 1907, cxxxiv, p. 692.

processes of very various origin, often permit the production of brain abscesses, and, as in other hæmatogenous brain diseases, the territory of the Sylvian arteries is especially prone to involvement. It is also stated that the left Sylvian region is the locus of election for metastatic abscess. Metastatic brain abscess is more often multiple than single and the symptoms more closely resemble those of the acute traumatic form than those of otitic abscesses. Embolic in origin, this disease is apt to have an apoplectiform onset, but the effects of the septic emboli are likely to continue and create sudden fresh attacks, due either to extension of local processes or to the accession of more emboli. The emboli are usually small and carried to the small branches supplying the cortex cerebri or its subjacent white matter.

The Sylvian predilection of such emboli secures a predominantly motor set of symptoms for these cases. But in some instances the foci are so many that localization is impossible. Eye-ground changes usually fail to develop in the acute course of these cases, nor are vomiting and vertigo such prominent symptoms as in most other forms of brain abscess.

**Rhinogenic Brain Abscess.**—Gerber[1] has collected from the literature 66 cases of brain abscess following frontal sinus disease. In the majority of cases the posterior wall of the sinus was diseased, and the abscesses are usually found involving the gyri of the orbital surface, and vary in size up to that of an orange. The characteristic absence of focal symptoms, the occurrence of frontal sinus symptoms, and the complicating presence of meningitis symptoms render the diagnosis difficult. Eye-ground changes are not uncommon, so that a diagnosis as against brain tumor has to be guarded. If, after frontal sinus operation, the headaches continue or increase, and fever, loss of appetite, alteration of general attitude, slowness of speech, restlessness alternating with apathy set in, orbital brain abscess may be suspected (Gerber). Later, several of the general symptoms of brain abscess may set in.

**Idiopathic Brain Abscess.**—Huguenin was forced to the opinion that idiopathic suppuration of the brain does not exist. Gowers states that in about one-sixth of the total number of cases no cause for the abscess can be discovered. He believes that forgotten trauma is an important feature of some of these cases, but regards the whole matter as an open question. Oppenheim also suggests forgotten trauma, and thinks that infectious diseases may at times produce suppurative disease in the brain without foci elsewhere (possibly through an otitis?).

**Course and General Diagnostic Features.**—The symptoms of brain abscess may all occur separately with meningitis, sinus thrombosis, and brain tumor, especially if these be complicated by extension of purulent or destructive processes. Moreover, syphilis sometimes yields a similar picture, and the difficult differentiation of symptoms of labyrinthine and central origin may often leave the nature of a case in doubt. In the absence of ear disease, disease of the nose and its communicating sinuses, bronchiectasis, empyema, or other peripheral source of metastasis, the chances are against the diagnosis of brain abscess. The history of trauma is unfortunately often dubious of interpretation.

As against *meningitis*, abscess more often produces hemiplegia, monoplegia, or aphasia, whereas irritative symptoms are more common in

[1] *Beitr. zur Anat. Path. und Klinik der Stirnhöhlen*, 1909.

meningitis. Cerebellar abscess, however, may produce the same retraction of the head and stiffness of the neck as meningitis. The absence of fever speaks against meningitis. The presence of fever favors neither diagnosis.

As against *sinus thrombosis*, the lack of high fever, of rapid pulse, and of tenderness and swelling over the internal jugular vein at its origin may be decisive for abscess. The occasional co-existence of the two lesions must be remembered. As against *brain tumor*, double optic neuritis is less frequent in abscess. Bramwell states that he never commits himself to a positive diagnosis of intracranial tumor unless suppurative ear and nose disease can be excluded.

In the majority of cases three or four stages may be distinguished: (1) The initial stage (lasting a day to a week), characterized by headache, vomiting, slow cerebration, confusion, delirium, fever, and rapid or slow pulse, with possibly stiffness of the neck, pupillary differences, convulsive phenomena, and the relative *absence* of focal symptoms. (2) The latent period (lasting weeks, months, or years, on the average one to three months), either absolute, when the symptoms are completely lacking, or relative, characterized perhaps by episodes of headache, vomiting, vertigo, and convulsions, and failing to show any constant phenomena save, possibly, depression. (3) The stage of abscess symptoms properly speaking (lasting some days or weeks), characterized by the signs of heightened intracranial pressure and the extension of suppuration. (4) The terminal stage.

**Prognosis and Treatment.**—Left to itself, brain abscess kills, despite the extraordinary periods of latency often reported. It may kill through heightening of intracranial pressure, perhaps effected by spreading of suppuration into the ventricles or the meninges. The treatment is essentially surgical, and Oppenheim remarks how unfavorable seemed the surgical results as depicted by Huguenin in 1876, and how relatively favorable as shown by Wernicke in 1881.

The excellence and extent of modern otological work should decrease the number of cases of brain abscess of the temporal and cerebellar type. Oppenheim counsels opening the skull and searching for suppuration in all cases in which the diagnosis of traumatic brain abscess is certain or even probable. The indications for operation in otitic cases are scarcely less decisive. Investigation of the cerebral and cerebellar conditions in the course of otological operations must be left to the judgment of the otologist or surgeon. Every large clinic has cases in which an abscess cavity in these regions has been "just missed" by the operator. Oppenheim goes so far as to state that purulent pachymeningitis, sinus thrombosis, or even beginning pyæmia do not contra-indicate operation. The onset of coma does not prove that surgery would fail. Obviously operations upon metastatic abscesses are less likely to prolong life, and multiple pyæmic abscesses are beyond our present range of treatment.

The mechanical increase of intracranial pressure either directly or through the effects of drugs or foodstuffs upon the vascular system should be avoided. Therapeutic measures against headache (narcotics, morphine, ice bags) and attention to the possibility of bedsores and the condition of the bladder and rectum, to say nothing of general nursing and hygienic measures, are obvious indications.

# CHAPTER XVI.

## EPILEPSY.

By WILLIAM P. SPRATLING, M.D.

**Synonyms.**—The antiquity of epilepsy is demonstrated by its great variety of synonyms, of which the following are good examples: *Morbus sacer*, the sacred disease; *Morbus Herculeus*, the Herculean disease; *Morbus commitialis* and *Morbus mensalis*, the disease of assemblies, and the disease of the table; *Morbus sonticus*, the serious or dangerous disease; *Morbus sideratus*, the star-struck disease—so-called because the ancients thought the epileptic had received a blow from a star or was blasted by some heavenly body; *Morbus deificus*, the god-making disease (epilepsy acquired this designation and distinction because the ancients thought it increased the priest's reputation for sanctity). It is also called *analepsia*, a grasping upward, and *apoplexia parva*, or small apoplexy. The Italians refer to it as *passio caduca et perdito*, meaning "the falling sickness combined with a tendency to destructiveness;" the Germans as *epilepsia, fallsucht;* the French, *épilepsie, grand mal, haut mal*, and *petit mal*. There are numerous other synonyms which scarcely need be mentioned here.

**Definition.**—Except for the purpose of neurological instruction and occasionally in medical jurisprudence, a definition of epilepsy is rarely necessary. If we carefully analyze a good definition, we can acquire a fair idea of the disease; and a physician called to testify in a court of law in which the question of epilepsy is an issue, and who cannot define the meaning of the word, may find himself in an embarrassing position. The following definition by the author embraces the more notable characteristics of the disease in all its types: "Epilepsy is a disease or disorder affecting the brain characterized by recurrent paroxysms which are abrupt in appearance, variable in duration, but generally short, and in which there is impairment or loss of consciousness together with impairment or loss of motor coördination, with or without convulsions."[1]

All phenomena that comprise an epileptic convulsion group themselves about two essential fundamental facts, to wit, the loss or disturbance of motility and the loss or disturbance of consciousness. There may be more of one and less of the other expressed in a given attack; or the reverse may be true, but if neither be present in any degree there can be no true epileptic convulsion.

**Classification of Types of Convulsions.**—We are not yet at a point in the study of epilepsy where we can formulate a classification based upon etiology. At present we can do no better than use the classification based upon symptomatology, which, however unscientific it may be, is practical

[1] From *Epilepsy and its Treatment*, by the author, 1904.

and readily applied. The four primary forms of epilepsy are: (1) Grand mal; (2) petit mal; (3) Jacksonian; and (4) psychic.

*Grand mal* is the most frequent and most pronounced of all the epilepsies, occurring in more than three-fourths of all cases. During such attacks consciousness is lost and motor coördination wholly destroyed. The patient falls or is thrown violently to the ground, often sustaining severe bruises, incised wounds, and fractures, death resulting in rare cases and being generally due to fracture of the skull.

*Petit mal* attacks partake of the same general characteristics as grand mal, save that they are not so severe. Consciousness is not always lost, and when it is, it disappears more gradually. Motor coördination passes in the same gradual way, and its loss is not complete.

*Jacksonian epilepsy* that remains as such throughout the seizure is rare, and does not occur in more than 3 to 4 per cent. of all cases. In this form the convulsive movements are confined to one leg, or one arm, to one group of muscles, or to one side of the face. It is the most easily diagnosed of all the epilepsies, and acquired its name in 1867 from Hughlings Jackson, who wrote a classic description of it at that time. Prior to 1867 it was known under the name of *partial epilepsy*, or the *parcella epilepsy* of the French writers, having been given this designation by Bravais in 1827.

*Psychic epilepsy* involves but one faculty, and that is the mind. In pure form it is not readily confused with any other type or with any other condition. Psychic epileptics suddenly lose consciousness, remain motionless a few seconds, if standing do not fall, and presently the normal state recurs. If the patient is talking when the attack occurs, the thread of conversation is merely interrupted, not lost, for speech goes on after the attack as before it. Usually not a word is lost or misplaced, and it is rare for more than momentary confusion to be apparent. Such attacks are barren of physical accompaniments, save perhaps a slight flushing or pallor and usually some drawing down of the corners of the mouth, with very fine fibrillary tremors of the muscles of expression. Psychic attacks vary in duration from three or four up to fifteen or twenty seconds, and in rare instances they last longer. They may occur several hundred times in twenty-four hours, or there may be but a few in the course of a year or more. Instances have come under the writer's observation in which 280 to 300 psychic attacks—all perfectly distinct and isolated in character—have been observed and recorded in twelve hours. There is no apparent reason why these "flashes" of lost consciousness should not occur several thousand times in twenty-four hours, as some writers claim to have witnessed, but any number in excess of three hundred or so must be estimated and not counted.

These, then, are the four chief forms of epilepsy which there should be no difficulty in recognizing, and which serve as a groundwork upon which to base a more elaborate classification of subtypes and varieties should one be desired. Brief mention may be made of some of the less frequent and more bizarre forms which are so rare as to demand but little more than passing attention from a diagnostic standpoint, since pathology, prognosis, and treatment will be satisfied by what will be said relative to the four primary forms.

**Irregular Forms.**—*Tetanoid Epilepsy.*—This type was first described by Pritchard in 1822. It is so infrequent that it has never been given a place of its own in the literature. When we consider the pathopsycho-

logical changes that must occur in the brain during a seizure, it is difficult to understand why there should be two distinct forms of spasm in an ordinary grand mal fit.

Tetanoid epilepsy may be described as constituting one element only of the ordinary attack—the tetanic or tonic state being present to the exclusion of the clonic or the alternating contraction and relaxation period. Care must be exercised not to confound this form of the disease with tetanus, for they have no relationship whatever. A boy, aged nine years, had a tetanoid seizure in my presence. He was of good physique, strong and robust, a pupil in school making good progress. He exhibited none of the stigmata of degeneration, and, so far as could be determined, his ancestry was clear of transmissible diseases. He was standing when the attack appeared. The initial movement was suddenly to spread the legs wide apart as though involuntarily to brace the body against falling. Instantly he grasped with both hands the clothing of a person standing near, uttered a single sharp, impulsive cry, his face flushed almost to cyanosis, the eyes were staring, the pupils widely dilated, and the eyeballs bulging as in strangulation. On grasping the muscles of his arms and legs and feeling those of his body generally, all were found as rigid as wood. He remained in this stiff and motionless position approximately ten to twelve seconds, when suddenly he became generally limp, his knees gave way, and he would have fallen to the floor had he not been supported. Within twenty seconds or so he was able to get on his feet, to walk, and answer questions intelligibly.

Tetanoid epilepsy is far more dangerous to life than ordinary grand or petit mal, the danger lying in the long-continued rigidity of the muscles of the trunk which are so intimately concerned in respiration. A woman, aged thirty-two years, went to a faucet for water, when a tetanoid attack occurred. Her feet were placed wide apart and the palms of both hands laid flat on a marble slab. She made no sound of any kind. Five minutes later she was discovered in this position, the face and neck were deeply cyanotic, the eyes wide open, fixed and staring, and the entire body so rigid that on supporting the back of the head with the heels resting on the floor she was laid full length without difficulty. Death occurred within two minutes after the onset of the attack, and was due to the sudden, sharp, and complete inhibition of respiration.

*Partial Epilepsy.*—This incomplete type deserves no place of its own, because attacks which might come under it can be better classified under Jacksonian Epilepsy, although there are some cases in which a *bona fide* attack once begun is not completed, and so it is customary to consider such rudimentary beginnings under the head of a partial epilepsy.

Partial epileptic convulsions are due to such causes as meningitis, acute or chronic, syphilitic infection, uræmic poisoning, excessive emotional states, alcoholic intoxication, brain trauma, embolism, thrombosis, and extrinsic causes which act in a reflex manner, such as injuries to peripheral nerves, old cicatrices, and various forms of irritation in different viscera. The cry is usually absent and consciousness rarely lost. An aura is lacking, although there may be indefinite sensations, such as tingling, numbness, and the like. As a rule, the spasm begins in the face and frequently goes no farther. Occasionally it begins in one thumb or in one of the lower extremities. To students of epilepsy the diagnosis of this type of the disease possesses no difficulty whatever.

*Myoclonus epilepsy* is a rare association disease that was first described by Unverricht in 1891 under the designation of family epilepsy. In about a fourth of the cases it occurs sporadically. It is characterized by paroxysmal, asynchronous, bilateral, lightning-like contractions of the trunk and the proximal muscles of the extremities, with varying intervals of entire freedom from such movements, and accompanied by a more or less persistent grand mal type of epilepsy. Prout and Clark[1] report 57 cases of this disease, 4 being cases studied by them at the Craig Colony, the rest collected from the literature. Shanahan has also reported a case from the Craig Colony.[2] Other writers in the United States have made mention of cases since the disease was first carefully studied at the Craig Colony in 1902–1903.

Hereditary factors are generally more pronounced in this association disease than in ordinary uncomplicated epilepsy. All the cases observed by the writer were in well-marked neuropathic families. An immediate excitant is of far less moment in the myoclonus type than in ordinary epilepsy. The condition usually appears in adolescence, and in about one-half the cases epilepsy comes first—the myoclonus condition being engrafted upon it later. In a third of the cases the onset of the two diseases is simultaneous. The seizures are more or less infrequent, usually grand mal in type, and preceded by increased myoclonic movements. The diagnosis is rarely difficult, provided too much stress is not laid on a single symptom. The prognosis is unfavorable. The disease shortens life more frequently than does ordinary epilepsy. The pathogenesis appears to consist of a faulty chemotaxis due to an inherent organic anomaly not fully demonstrable at this time.

*Epileptic equivalents* signify something that is "like or equal to" an epileptic convulsion, and may be regarded as a substitute attack. By some neurologists the existence of epileptic equivalents is denied, such attacks being looked upon as vagrant manifestations of some well-recognized form of the disease. After having witnessed almost every conceivable form of epileptic attacks with definite variations, the writer is not prepared to assert that we have justification for referring to certain phenomena as "equivalents." Bizarre forms of epilepsy are sufficiently common to permit us to believe that what might be termed an "equivalent" is in reality an incomplete attack, or the accentuated variation of an ordinary attack.

Epileptic equivalents, if they are to be considered, last from a few moments to hours or days, varying in the meanwhile in intensity of expression. It is not essential that we have a fit implicating the entire body in gross muscular commotion to have it followed by mental disorder. Any kind of an attack may have such a sequel. Epileptic equivalents may alternate with ordinary grand or petit mal seizures so far as the period between the attacks is concerned. The writer has observed repeated instances in which they appeared three, six, nine, and even twelve months apart with almost unvarying regularity, partaking in this respect of the "folie circulaire," or the "circular insanity" of the French school, which appears, runs its course, disappears, and recurs time and again within a given period, until more than a score of alternations have marked the progress of the disease, when the symptoms of terminal dementia begin to be apparent, and sooner or later

[1] *American Journal of Insanity*, vol. lv, p. 585.
[2] *Journal of Nervous and Mental Disease*, 1907, xxxiv, 504.

it is definitely established. Epilepsy that is complicated by alternate equivalent attacks is far more difficult to influence by treatment or to cure than ordinary seizures, the psychic element giving a degree of obstinacy against forms of treatment that usually avail when the equivalent is not to be combated.

**Method of Examination.**—Prior to the founding of special colonies, sanatoria, and hospitals for epileptics, incomplete examinations were the rule rather than the exception in the effort to establish a diagnosis of epilepsy, the reason being that the patient could rarely be kept under the physician's control long enough to complete such examinations in the manner required. Under the old system the complete physical and mental examination of a supposed epileptic was not regarded as essential. Now both are always indispensable. It is rare that a single examination suffices to cover the points that need to be gone over time and again with scrupulous exactness and care. The examination must reveal the condition of every organ in the body, and should include careful physical measurements to determine the possible existence of atrophy due to an early palsy. Thus, frequently patients come under observation in whom an epilepsy that did not appear until puberty or later was due to an infantile palsy that occurred before the third year.

**Family History.**—Minute and systematic inquiry into this is essential, for the reason that epilepsy is so often due to heredity. Not only must we consider the immediate relationship, but we must inquire into the collateral branches as well. We must make a cautious and not infrequently a tedious search for epilepsy, insanity, alcoholism, syphilis, tuberculosis, migraine, cancer, chorea, "spasms" or "fainting spells," hysteria, and other evidences of constitutional or neurotic taint that in similar or modified form might have been transmitted directly or indirectly to the patient, remembering always that these things are skeletons in the family closet only dragged forth in most instances by adroit and persistent inquiry. Similar heredity—that is, where the epilepsy in the child is due to the same disease in one of the parents—is responsible for approximately 16 per cent. of all cases. Dissimilar heredity—meaning the presence of insanity, alcoholism, or some other constitutional disease in the ancestors—is responsible for fully 50 per cent. of the remaining cases.

**Personal History.**—This is not less important, and includes some twenty to twenty-five carefully selected questions framed to show birth accidents, which play no inconsiderable part in the production of epilepsy, delivery at full term or premature birth, spasms in infancy, difficult dentition, prolonged fits of crying, evidences of rickets or scrofula, serious injuries to head or spine, and the acute infectious fevers of childhood, especially scarlet fever, the sequelæ of which lead to many cases of epilepsy before the tenth year of life.

**History of Epilepsy.**—This should begin with a thorough description of the first attack, provided it was witnessed by a competent observer. This is of exceedingly great importance, and no effort should be spared to secure the most reliable account it is possible to obtain. No fact in this connection is too trivial or insignificant to deserve record.

The aura, if any, the type or types, if there be more than one kind, the anatomical location at which the attack begins and the order in which it progresses, evidence of false sight or false hearing just before, during, or

following an attack, the effects of the disease on the mind temporarily and permanently, the greatest number of attacks in twelve or twenty-four hours, the tendency to "serial attacks" or to the far more serious condition of status epilepticus, the tendency to gluttony in any form, any noticeable change in temperament, manifestations of disobedience, of moral obliquity, vicious personal habits, and other factors that help to complete the personal history should all be objects of careful inquiry.

The *personal examination* includes a study of the physiognomy. In many cases the skilled observer can make a diagnosis of epilepsy by an analytical scrutiny of the patient's face alone. A confirmed epileptic who repeatedly falls during seizures is apt to have multiplied or overlapping scars on prominent points, such as the nose, the supra-orbital ridge, the chin, the forehead, and the occiput; and if much bromide of potassium has been taken over a long period and elimination has been sluggish, the patient will have a disfiguring acne. Epilepsy that is distinct in character, that begins early in life, and expresses itself with frequent and systematic regularity leaves a stigma on the physique in the way of a more or less stunting of growth. Careful measurements, made at the Craig Colony, of 954 epileptics grouped in different age periods showed that epileptics are shorter in stature and weigh less on an average by 12.5 per cent. than do persons of normal health and free from this particular disease.

The *physical examination* shows scars due to falls and to operations for the relief of the epilepsy, the two generally being distinguishable. Certain anatomical stigmata of degeneration are frequently encountered, such as malformed and asymmetrical heads, irregular hard palate conformations, misshapen ears, and other features too numerous to mention in detail.

The *mental examination* is most important; it shows the effects of the disease on the mind, gives indication as to whether the epilepsy is true or false, and, most important of all, it furnishes a basis on which to establish a prognosis in a way in which nothing else can do.

The aid of the laboratory should frequently be employed. This is more essential as the resources of the laboratory become more helpful. Chemical examinations of the urine, blood, sweat, gastro-intestinal contents, and the cerebrospinal fluid are essential in most cases, while repeated examinations of the urine and blood are called for in all cases.

All superficial and deep reflexes should be carefully tested, anæsthetic and hyperæsthetic areas located if suspected, and the mouth, teeth, ears, eyes, and nose subjected to the most careful scrutiny for disease or abnormality of any kind. The nose should be examined for polyps or hypertrophic tissue which may obstruct breathing. Evidences of stigmata of degeneration in connection with the hard palate, the ears, or of the cranium generally or of any of its parts should be systematically noted.

Few epileptics can be properly examined for the first time in less than two hours; while a second examination is not infrequently required. In just proportion to the completeness of these original studies will be the value of the treatment that follows. It is the author's practice to reduce to writing all instructions, suggestions, and diet regulations formulated for the patient. A record card of all seizures should be kept by the patient or his immediate attendant.

**Etiology.—Age.**—Epilepsy is essentially a disease of early life. In 2523 cases observed at the Craig Colony during thirteen years, 84 per cent.

were found to have originated before the twentieth year. There is no disparity in this respect between the sexes prior to the twentieth year. After that the disease is more common in males than in females, in the ratio of 100 of the former to 80 of the latter. Three factors are responsible for this difference—alcoholism, trauma, and syphilitic infection, which are relatively common in male adults, and very seldom observed in childhood, and then it is mostly trauma and not syphilis or alcohol that caused the disease. Inherited syphilis is occasionally a factor in infancy, alcohol never.

More cases of epilepsy occur during the first year of life than during any subsequent period of equal length. The following table clearly shows the age influence in 1162 cases: First year, 163; second, 67; third, 48; fourth, 38; fifth, 30; sixth, 43; seventh, 48; eighth, 54; ninth, 48; tenth, 56; eleventh, 48; twelfth, 71; thirteenth, 56; fourteenth, 84; fifteenth, 59; sixteenth, 43; seventeenth, 44; eighteenth, 32; nineteenth, 33; twentieth, 26; twenty-first, 27; twenty-second, 16; twenty-third, 15; twenty-fourth, 6; twenty-fifth, 7.

After the twenty-fifth year the number grows progressively smaller until about the sixtieth year, when epilepsy arising *de novo* becomes a rarity. Occurring after that age it is more frequently due to arteriosclerosis than to any other single factor. This cause is rare in women. The oldest epileptic that ever came under personal observation was eighty-nine years of age when the first seizure occurred, the patient dying in his ninety-second year as a result of serial attacks, simulating status epilepticus.

*The influence of heredity on age* is interesting, for it shows that when heredity is present it almost invariably manifests itself early in life, although cases have been recorded in which heredity was the assigned cause and in which the first attack did not occur until after the thirty-fifth year.

**Race.**—Racial differences or characteristics play no part in the causation. So far as can be ascertained, all races suffer alike and in relatively the same proportions. Since 1895 the writer has studied the disease in natives of the United States (including Indians and negroes in the South), Germany, England, Russia, Ireland, Scotland, Wales, Canada, France, Italy, Austria-Hungary, Japan, Armenia, Arabia, and Belgium. Reports received by the National Association for the Study of Epilepsy—*Transactions*, 1905–1906—show its existence in Turkey, India, Brazil, Chili, Australia, and the Fiji Islands. There is every reason to believe that no race under the sun is exempt from "the strangest disease in human history."

**Occupation.**—This does not offer any advantage or disadvantage in the etiology. The only reason it is found more frequently among persons of certain vocations is because of the relatively larger number of persons engaged in such callings.

**Heredity.**—Carefully prepared statistics of any magnitude on this subject do not exist, and even if they were available, the difference due to the personal equation would be so considerable as to make any deductions of but little value. Gowers makes the unqualified statement that "epilepsy is an inherited disease," adding, "there are few diseases in the production of which inheritance has a more marked influence, and the traceable influence is always far less than that which exists." Voisin, Féré, Nothnagel, Dana, Peterson, Starr, Church, and others express similar opinions, although none with the positiveness of the English author, whose opportunities for observation have been exceptional.

The writer's position in the matter is that epilepsy is more frequently due

to heredity than to any other single factor, one-sixth of all cases being due to this cause alone. In a careful study of 2523 epileptics under his care in a given period, it was definitely established that sixteen out of every hundred were due to heredity. In most of these it was possible to establish some other cause in addition to heredity, although such other secondary cause was often so indistinct as to render it a very questionable factor, leaving heredity the supreme agency to which the disease could positively be ascribed. This percentage was arrived at after the most painstaking inquiry in every case; the family and antecedent history of the patient being carefully gone into time and again. Only the statements of persons competent to testify were considered, and all the data procured were in writing.

Heredity is of two kinds—similar and dissimilar. As has been stated, the former means the direct transmission of the same disease from parent to offspring. The latter means that insanity, alcoholism, or some constitutional disease has been converted in transmission from parent to offspring so that it appears as epilepsy in the latter. The 16 per cent. previously referred to were due to similar heredity only. If we include both types of heredity, the percentage is increased approximately to 60 or 65 per cent. In making this assertion we may be asked to explain how far heredity is actually responsible, and a safe reply would be that if such influence had not been present, in all probability there would have been no epilepsy.

The *modus operandi* of heredity is an unsolved problem, and while greater reference will be made to this part of the subject under pathogenesis, it may be said now that it is the function of the cortical brain cells to produce and retain energy. This function is continuous, and the cells must be ready to release such energy at a moment's notice to satisfy any demand that may be made. In normal nervous action the balance between the income and the outgo of the nervous force is constantly maintained. When this balance is disturbed there is evidence of irregularity or disease, and a discrepancy is at once apparent between the manner in which such energy *is* released and the manner in which it *should be* released.

Motor neurones have power to inhibit action. If this power is impaired, it follows clearly that action will be irregular, disjointed, abnormal, unsound, and the cause of such impairment is the cause of disease. In the first place there may be—and in all probability there usually is—a generic cause for such impairment. The fault is assumed to lie either in a structural malformation, in the congenital undersize of the cell, or in the overproduction of nerve force combined with defective nerve fiber insulation which permits nervous energy to go to unchecked and indiscriminate waste.

*Insanity in the parent* as a predisposing cause of epilepsy in the offspring is less effective than alcoholism. In 660 males insanity was found in the parents in 49 (13 per cent.); while in 410 women, 42 of them (7 per cent.) had insane parents.

**Alcohol.**—This is a positive and fruitful factor in the etiology of epilepsy in both similar and dissimilar heredity. In 1070 cases, 111 males and 51 females—equal to 15 per cent. of the entire number—had alcoholic parents. Very few women acquire epilepsy through excessive drinking, but it is a distinct factor in men of middle age and who, as a rule, live sedentary lives.

**Consanguinity, syphilis, and tuberculosis** all play a part, although just how far tuberculosis does so is difficult to decide. It can safely be doubted whether it does more than undermine the resistance, making the individual

more susceptible to any constitutional disease. The writer does not believe that tuberculosis is ever an efficient and sole cause of epilepsy.

It is different with consanguinity and syphilis. Numerous instances have come under personal observation in which the intermarriage of blood relations so vitiated the general stamina as to induce epilepsy. Syphilis is a more powerful influence than blood marriages in this respect. It has the power to cause epilepsy through general brain infection, or the infection in the parent at a certain time may induce hereditary syphilitic epilepsy in the offspring. Three well-defined cases due to the latter cause have come under personal observation. One was a boy, aged eight years, whose father was a physician and admitted syphilitic infection, which was at its height at the time of the boy's conception.

Among other predisposing agencies of doubtful potentiality are rheumatism, cancer, scrofula, and such parental intoxications as lead, opium, diabetes, and the like. A high degree of conservatism should be displayed when seeking to ascribe epilepsy in the offspring to any of these constitutional maladies in the parents.

**Organic Causes in Early Life.**—It will make the study of the immediate causes less difficult if we take them in serial order and base them more or less upon certain age periods.

*Cerebral Palsies.*—One of the greatest causes in the young is the birth palsies, and fully 8 per cent. of all cases are due to the present or remote influences of brain hemorrhage. The seat of lesion in these cases is the hemisphere of the brain involving the central motor neurones and that part of the motor cortex as far as the anterior horn, making such palsies corticospinal, in contradistinction to those due to disorders of the neurospinal neurones, which give rise to the forms of infantile spinal palsies without involvement of the brain. Most cerebral palsies occur before the third year, and fully one-half of them are at birth. In the latter the cause is found in some injury to the mother during pregnancy or an injury to the child through the use of instruments. The writer considers the last an important factor, to which insufficient attention is given. During the first two or three months of life reflex action is poorly developed, and convulsions at that period are, as a rule, primarily cerebral and due to hemorrhage.

Repeated incomplete or superficial examinations may fail to show that a hemorrhage occurred in early life and that convulsions result years later; whereas, had *careful examinations* been made to identify the degree of atrophy present, we would have light on an otherwise obscure problem. One means of detecting a palsy, inconspicuous, but yet sufficient to perpetuate convulsions, is to test the degree of motion and power in the arm or leg supposed to be palsied, immediately after a convulsion. If the member examined did not bear the brunt of the seizure, but shared it in equal degree with other parts of the body, it would be in a state of exhaustion and lacking in muscular power, as may be shown by a dynamometer if the palsy is confined to the arm and hand. This condition constitutes "exhaustion paralysis," from which recovery is usually made in a few days or weeks at most. Should this exhaustion paralysis be more widespread, it will be marked in the arm or leg involved in the palsy.

*Birth Accidents.*—These are responsible for epilepsy in some cases, but in just what proportion cannot be accurately stated. In studying the etiology of a large number of cases, we hear now and then of a "blue baby"

who later developed epilepsy. Such cases come under asphyxia. The use of forceps may give rise to epilepsy in cases in which prolonged and severe pressure causes an extrameningeal hemorrhage, the pressure of the clot that forms giving rise to the disease. It should be the duty of the obstetrician to guard against the possibility of future epilepsy by the prevention of birth accidents through the exercise of great care in pressure and traction and in early drawing the surgeon's attention to a cerebral hemorrhage in the newborn. We seem justified in anticipating beneficent results from surgical intervention in selected cases.

*Difficult Dentition.*—This probably never acts as an unqualified cause when not influenced by another agency, but there is little doubt that when dentition becomes pathological, in a *strongly tainted neuropathic* subject it may cause a type of convulsion which, if permitted to go unchecked, may eventually pass into a more or less true form of the disease. No one would think of designating as epilepsy a single convulsion caused by painful dentition in a rachitic child, accompanied by high temperature, irritability, and great restlessness. But should there be several convulsions which continue with more or less regularity, showing the essential characteristics of the true disease, including the aura, the epileptic cry, and the various postconclusive phenomena—common reason would lead us to designate them as epileptic. So, while dentition may never be an unqualified factor, it has under special conditions the power to incite a type of convulsion that may pass into epilepsy so far as the general results, the prognosis, and the treatment are concerned. In thirty cases of epilepsy originating between the sixth and eleventh months of life, and all personally known to the writer, difficult dentition was the assigned cause.

*Infectious Disease.*—Some of the infectious fevers of childhood are rather frequent causes of epilepsy, scarlet fever being the most conspicuous. The disease does not usually appear until some weeks after the fever has subsided and scarlatinal nephritis appeared. A peculiarity of such convulsions when they first appear in the seventh or eighth year is that they frequently subside to recur from some trivial irritation with the advent of puberty, always a critical epoch in the life of any person with a tendency to epilepsy or any other neuropathic disease. Whooping-cough, measles, typhoid fever, insolation, pneumonia, influenza, malarial fever, diphtheria, and cerebrospinal meningitis are all infrequent causative agents. Cerebrospinal meningitis has greater power in this respect than any of the others.

*Fright and Shock.*—Too little stress is laid on emotional causes in the etiology of epilepsy, especially in young persons of prominent neuropathic temperament. A distinguished English author declares that "of all the immediate causes of epilepsy, the most potent are psychical—fright, excitement, anxiety." In 814 males and 591 females studied by the author at the Craig Colony emotional shock or fright was the given cause in 22 of the former and 40 of the latter, equal to 3 per cent. of the males and 8 per cent. of the females. This disproportionate percentage sustains the established fact of the greater instability of the nervous system of the female. Heredity plays a conspicuous part in practically all cases due to these causes, the invariable rule being that the more pronounced the hereditary taint, the less powerful the psychic factor need be to cause epilepsy. The reverse is equally true. Psychical causes are more active in early than in adult life, for it is during this period that hereditary influences are most strongly felt.

*Overwork* has been given as a cause of epilepsy—a proposition to which we cannot assent. An epileptic subjected to very marked and prolonged bodily fatigue may have his attacks increased as a result of such condition.

*Trauma.*—Injury to any part of the central nervous system may cause epileptic convulsions, while injuries to peripheral nerves may produce reflex convulsions which, under long continued and sufficient intensity, may finally partake of the essentials of the true disease, with the exception that they will necessarily be lacking in the obscure basic lesion in the central nervous system that we must assume to be present in the idiopathic disease.

Brain trauma produces epilepsy without the associated influence of heredity of any kind in any degree. Convulsions usually follow traumatic lesions that have a distinct anatomical foundation, although they may follow a blow on the cranium which produced concussion only. Convulsions of this character are generally quite innocent, and are recovered from without evil after-effects. When there is anatomical injury, when the skull is broken and the bone depressed in any way to interfere with the cerebral function, the case is obviously more serious. Such injuries demand prompt surgical treatment to the end that established epilepsy may be prevented. It is when such wounds are neglected and the surface scar allowed to heal, or when the blow causes a small portion of the inner skull plate to split off and be driven through the meninges into the brain, there being no external evidence of brain laceration, that obscure traumatic epilepsy may develop.

A blow which fractures the skull may not be followed by convulsions until considerable blood effusion has taken place, the irritation resulting from a clot causing the convulsions, as may be demonstrated by its removal. Blows over the motor area are most likely to cause epilepsy, while a bullet in any part of the brain is capable of producing the same result.

All traumatic epilepsies are largely motor in character, and the mental deterioration induced by them is not usually very marked, nor does such deterioration generally appear until some years after the epilepsy has become established. Some purely motor epileptics never show mental impairment in any conspicuous degree. The length of time that intervenes between the injury to the head and the appearance of epilepsy varies from a few days or weeks up to several years, developmental processes later on giving a pernicious character to a lesion apparently innocent when first received.

More males than females suffer from traumatic epilepsy because vocation renders them more liable to accident. Trauma is responsible for 7 to 8 per cent. of all the epilepsies, and while the types vary in expression, grand mal is the most common. It is not settled that mechanical injury to the brain can induce psychical attacks—although such cases have been reported. Traumatic cases are most active in the young; in 73 out of 88 cases in both sexes seen by the writer, the injury was received before the twentieth year. The sexes were divided as follows: Males, under ten years, 32 cases; ten to twenty years, 25 cases; and over twenty years, 14 cases; females, under ten years, 10 cases; ten to twenty years, 6 cases; and over twenty years, 1 case.

**Miscellaneous Causes.**—Given a marked hereditary tendency, there are numerous agencies capable of causing epileptic convulsions in addition to those mentioned. Gastro-intestinal disorders have not received the consideration they deserve. Intestinal toxæmia may cause convulsions in two ways, either by proximately irritating the peripheral (visceral) nerves in

that locality or by the absorption of certain toxic substances. Poisoning from lead occasionally causes *saturnine epilepsy*. A boy, aged four years, ate fresh white lead from a wall, and violent convulsions resulted; these persisted for days, then disappeared, and recurred ten years later coincident with the stress of puberty. He became a confirmed epileptic. It is questionable whether *tobacco in excess* or in any form can induce epileptic convulsions in a normal person. Six cases have come under the writer's care in which excessive cigarette smoking—sixty to seventy cigarettes in twelve hours—was the alleged cause of the disease. All were boys under sixteen years of age. It cannot be doubted that the influence of *excessive* nicotine poisoning in *selected subjects* may induce epilepsy.

*Shock from electricity* has been known to produce the disease. A clear case due to this cause was witnessed in 1896. A man, aged thirty-five years, grasped the "live wire" of a street railway car. Chronic epilepsy followed the shock, and he recovered damages from the company. Ten years later he was still epileptic, his attacks being mostly psychic in character and followed by long periods of automatism.

*Renal disease* in the form of scarlatinal nephritis has been mentioned as a factor. Nephritis in late adult life may also produce epilepsy, which should be differentiated from uræmic convulsions.

*Disorders of menstruation*, functional or organic, may induce epileptic attacks in women possessing a neuropathic predisposition. Convulsions usually appear in such cases between the twelfth and sixteenth years. There is a lay tradition to the effect that the establishment of womanhood favorably influences epileptic seizures. The writer's observations in fourscore cases of the kind have been to the contrary. The same is true of marriage in most cases. Rarely in women of certain types favorable effects may follow, but the reverse is oftener the case. The *menopause* is a comparatively frequent exciting cause of epilepsy in persons possessing a high degree of neuropathic taint. We should constantly remember that convulsions in infancy are very apt to recur at one of the physiological epochs—the second dentition, puberty, maternity, and the menopause or senility, when changes are marked.

*Pregnancy and maternity* are occasionally complicated by epilepsy. Three cases due to the latter have been noted. Such convulsions are usually designated "eclamptic." It requires patient and prolonged study to make a differential diagnosis between temporary eclamptic convulsions and those of true epilepsy. Precipitate study in such cases may lead to false conclusions.

It is contended by eminent authorities that masturbation can be demonstrated as a cause of epilepsy from the fact that circumcision checks the convulsions. True epilepsy is seldom due to this practice, although Gowers declares it may cause "habit spasms" during early puberty. Nine boys, from twelve to seventeen years, in whom the alleged cause was masturbation, have been treated by the writer.

The frequency with which *heart disease* of some form is encountered in epilepsy would appear to give it some weight as an etiological factor. In 1070 cases of epilepsy the following cardiac disease or irregularity was noted: Mitral regurgitation in 77, irregular cardiac force and rhythm in 75, cardiac hypertrophy in 50, roughened first sound in 13, systolic murmur at apex in 10, aortic regurgitation in 9, mitral stenosis in 2, tricuspid regurgitation in

1, and a diastolic apex murmur in 1, making a total of 238 out of 1070 presenting evidences of some more or less pronounced cardiac abnormality. It must not be supposed that these conditions caused or even influenced the origin of epilepsy in every case. They simply existed with the epilepsy. On the other hand, there is but little question that a cardiac lesion of a specific kind may rarely cause epileptic convulsions which bear a striking resemblance to syncopal attacks, and may be mistaken for them.

*The influence of sleep* in the production of seizures is a most puzzling question. "Predormitial" epilepsy is applied to attacks that occur at the precise moment that sleep begins; while "postdormitial" is applied to attacks that appear coincident with the waking period in the early morning, usually between five and seven o'clock. More than a score of cases have been noted by the author in which true epileptic seizures—wholly devoid of aura of any kind—never appeared except as the last trace of consciousness was lost in sleep. The experiment was repeatedly made of having the patient change the hour of sleeping from night to day, the result being that the attacks changed also and continued to follow sleep. It would appear that in such cases the extremely delicate psychic stimulation of consciousness alone is sufficient to inhibit some epileptic convulsions. If this be true we must marvel at the high degree of sensitiveness possessed by the central nervous system, since it can be so easily thrown into the greatest confusion by the ordinarily salutary condition that characterizes the brain during normal sleep.

Some nocturnal epileptics who also have diurnal attacks have unpleasant dreams which excite convulsions. Such dreams apparently play upon subconscious emotions, exciting the brain to violence as though full consciousness were present. The mechanism of the fit in such cases is doubly obscure. When the two sexes meet in an assembly hall for dancing or other amusement, the emotional stimulation induces convulsions. This fact has been repeatedly verified by denying the right of one sex to be present.

*Hydrocephalus* is occasionally a gross organic cause of epilepsy before the fifth year. Such cases are incurable. Focal lesions of the central nervous system independent of external brain injury play a conspicuous part in the production of epilepsy. Tumors, local areas of softening, abscesses, atrophy, sclerosis, are all causative agencies now and then. Middle-ear disease underlies a type of convulsion that closely simulates true epilepsy. A close study of aural symptoms should be made in all such cases to determine the true nature of the disease. Mastoid disease induces a type of motor epilepsy difficult to cure.

*Eyestrain.*—This question has been an active battleground on which neurologists and ophthalmologists have contended in efforts to class this condition as one of the established causes of epilepsy. A vast amount of disjointed work and many wholly misleading opinions, due to lack of any intelligent system of examination, can be found in the literature prior to 1901. In that year two ophthalmologists of distinguished ability made a thorough test of the matter at the Craig Colony that should be accepted as conclusive. With more than 900 epileptics of all types, ages, and conditions, and with every known etiological cause to select from, after a studied and prolonged examination, all save 100 were rejected as being unsuitable for the test. A week was spent by the two oculists in making careful examinations of the 100 patients, at the end of which time 32 more were set aside, leaving 68 with whom no fault could be found that would be likely to impair

the value of the test. Individual prescriptions were written for glasses for every patient—for the epilepsy was to be cured, if at all, by the correction of refractive errors through the use of proper lenses, and not by the more radical use of the knife. A manufacturing optician of repute personally fitted the glasses, and an assistant physician was delegated to keep a close oversight over the 68 patients for one year. All seizures, including the type, duration, hour of occurrence, and all uncommon phenomena in any way connected, were recorded. Whenever glasses were broken or lost, they were quickly replaced; and, all in all, no fairer or more judicious test could possibly have been made; the plain, unequivocal truth of the value or lack of value of the method in question was carefully sought.

The results were unsatisfactory. The total number of attacks that occurred during the year following the trial of glasses was no less than the number during a similar period before glasses were adopted. One man whose epilepsy was motor in type, and who had a persistent motor aura which lasted fifteen minutes before each attack, had his seizures modified by the glasses. He had a seizure six weeks after being fitted with glasses, and was then free from them for one year, when his glasses were broken. Immediately the attacks recurred in the same form as before. Resuming glasses, the attacks again disappeared, and this was repeated four times within the following three years. He is still epileptic, seven years after the effort to cure him with glasses was first undertaken.

It appears that in some cases proper glasses—just as the bromides—have the power to *hold attacks in abeyance*, but they do not possess the power to cure. Such is the writer's unqualified belief.

As a result of this experiment, so carefully and conscientiously made, it is the author's firm opinion that cases of *epilepsy due to eyestrain* are of extreme rarity, if, indeed, they are ever encountered; while cases due to this cause that can be cured by the use of glasses alone, not counting those whose attacks may be *suppressed*, are more infrequent still. We have no moral right to hold to the fallacy that true epilepsy can arise from a restricted point or organ, like the eye, for it is a disease of the *entire organism*, and to cure it we must apply treatment on a broad and rational basis to that end.

**Pathology.**—The changes in epilepsy are best studied under two divisions, gross and minute, or organic and microscopic. The first includes the anatomical states found in the cerebrospinal nervous system, in the meninges, and in the bony walls.

Among the organic brain conditions are atrophy and maldevelopment due to hemorrhage, thrombosis, embolism, trauma, localized or general sclerosis, tumors, cysts, abscesses, meningeal thickening resulting from inflammatory exudates, and various congenital and acquired deformities of the skull. Such gross lesions do not of themselves cause epilepsy. They are only effective in co-existence with an unstable nervous organization, one predisposed to convulsive manifestations. Skull anomalies include alterations in its texture, the bone being usually thickened, although there are cases in which the bone is not only unusually thick, but extremely brittle, the latter condition, as a rule, being found in certain localities only.

We must not mistake pathological conditions of the skull present since birth for those due to falls occasioned by repeated epileptic convulsions. Congestion of the skull and coverings of the brain and spinal cord is constant in epilepsy, being especially marked when death follows serial attacks or

the more fatal condition of status epilepticus. The younger the patient the greater the degree of congestion in such cases. Deformities of the skull and facial asymmetries in epileptics are common. Binswanger, Bourneville, Sollier, Féré, and other notable writers have called attention to this. Stigmata of degeneration affecting the hard palate, teeth, and ears are also frequently noted.

Pathological changes to some extent are usually found in the meninges in cases of long standing, and are most marked in the infantile palsies of the cerebral type. Here the dura is frequently so closely adherent to the skull that its separation is difficult and occasionally it must be torn away piecemeal. In other cases it has undergone calcareous degeneration and shows similar deposits within its substance. The pia mater presents frequent changes in chronic epilepsy, but none that are constant. They consist mostly of œdema or congestion, the latter being a sequence to severe seizures covering a long period of time. A certain amount of œdema is quite common in old insane epileptics, and it is nearly always associated with an increase of cerebrospinal fluid. Thickening and opacity of the pia mater are also met with in infantile hemiplegic cases, but with nothing like the frequency with which they are seen in the insane.

Of all pathological conditions observed, infantile cerebral hemiplegia is by far the most common. The proportion of cases of epilepsy due in some measure to this cause is variously stated by different observers. Osler noted 35 cases in a total of 120. At the Vanderbilt clinic there were 87 cases in 264, and Sachs and Petersen found 62 in 140, and Wallenberg 66 in 160 cases. Our own studies in nearly 4000 cases yielded 34 per cent. due to this cause alone. On the whole, we are justified in stating that 40 per cent. of patients with the cerebral hemiplegia of infancy sooner or later become epileptic. To produce epilepsy the size of the hemorrhage in such cases is of little consequence. Convulsions may follow one so slight as to escape notice. On the other hand, it may be so extensive as to make it difficult to understand how the patient was able to survive its destructive influence.

Some neurologists hold that *sclerosis of the cornu ammonis* is one of the most frequent gross conditions found in epilepsy. It was first described by the French writers, who laid much stress upon it. Others regard it as merely the local expression of a widespread sclerotic change that may be noted throughout the cortex of the brain. Tumors, cysts, and vascular lesions in the brain rarely cause true epilepsy, although when they occur in the motor cortex they may produce epileptiform seizures. As a rule, they are non-malignant and are much more liable to occur in the meninges than in the brain substance itself. Sarcomata and gliomata are more common than the rest, and may develop in the brain substance in the first instance or follow secondarily. Old epileptics not infrequently present at autopsy small cysts in various portions of the brain which probably occur secondary to vascular changes in the arterioles in the cortex.

When death follows a number of severe seizures the entire cerebrospinal system frequently presents numerous small punctate hemorrhages which are most marked in the cortex. They are especially distinct in status epilepticus and some authorities regard them as the direct cause of death, others as the cause of an exhaustion paralysis. Personally we accept neither view, but regard them as the physical results of seizures. Similar

extravasations are seen on the face and neck after grand mal attacks in some individuals.

We now have to consider the most striking changes presented by the *cortex* of the epileptic when the disease has existed for a long time. When death occurs during status epilepticus these changes are the most decided. The cells are swollen in many instances to twice their normal size, the nucleus being large and granular and possessing an indistinct outline. The limitations of the nucleus are often difficult to determine, the chromatic substance has almost entirely disappeared from the body of the cell, and it assumes an irregular outline.

The most striking changes are found in the nucleolus, which, in addition to being granular, swollen, and poorly outlined, is often entirely absent, having been abstracted during the process of section making. This condition is found two or three hundred times more frequently in cases of status epilepticus than in the normal brain. The most important alterations are observed in the nucleus of the cells of the second cortical layer and other cells of that type. The change affects the intranuclear network, destroying it and rendering the nucleolus a loose body within the nucleus, so that it is readily lost during the process of cell division. The ultimate disappearance from the cortex of cells so seriously altered is to be inferred. The particular involvement of the nucleus in this process is a fact of the first importance. Biological facts teach us that "the formative power of the cell centres in the nucleus," and for that reason it must be "regarded as the essential organ of inheritance;" that "it plays an essential role in chemical synthesis;" that "digestion and absorption of food and secretion cease with its removal from the cytoplasm," and that "fragments of protoplasm deprived of the nucleus die" (Wilson, Verworn). And so it is that when morbid processes attack the nucleus, the one portion essential to its life is threatened, and if this continues too long the cell is destroyed. We seem justified in assuming that the essential poison in epilepsy is one which attacks the centres of cell life and which shows a special predilection for certain delicately constituted cells of the cerebral cortex, typical of the second layer.

Many authors have described chromatolysis following the epileptic seizure. It is always present after serial fits, and is especially marked in status cases, where it is often so decided that the large Betz cells of the motor cortex appear like denuded cell remnants, the body of the cell being greatly shrunken. Changes of this character are always general. In the light of recent investigations (Marinesco, Lugaro, Ewing), we are to regard these changes as nutritional in character, brought about chiefly by the suspension of functions on the part of the nucleus which, as we have previously mentioned, presides over the processes of nutrition and assimilation within the cell.

The conclusion seems warranted that epilepsy is a disease state of the sensory elements of the cortex, and that the impulses constituting the discharge phenomena are peculiar to such disease states and are transmitted over other than the ordinary motor paths. The elements of the cortex most seriously involved are certain sensory cells of the second cortical layer, some of which are destroyed during the epileptic process.

**Symptoms.—Status Epilepticus.**—This distinctive and most accentuated expression of the epileptic state was the first described by Bouchet and Casauviehl in the Saltpêtrière and Bicêtre Hospitals in Paris in 1825

under the term "État de Mal." It is the maximum development of epilepsy. In it one paroxysm follows another so closely that the coma and exhaustion induced by it are continuous.

Sooner or latert he condition is attended by a marked rise in temperature, with increased pulse beat and respiration. A fatal termination follows in 25 per cent. of all cases. It usually occurs in idiopathic epilepsy, in which grand mal seizures predominate. In very rare instances it follows psychic seizures, which are exempt from gross muscular commotion of any kind, and death usually occurs within a few hours. When following psychic seizures the condition is known as "masked status." Sex has no influence in causing it, and the mortality between the two sexes is equal. It may appear at any age, but is most common at puberty.

The precise cause of status epilepticus remains undetermined. The state is divided into two stages—stuporous and convulsive. Sometimes the attacks are an hour apart until the first five or ten have occurred; after that they may follow each other with only a few seconds intervening. At the outset consciousness is completely regained between the paroxysms, but later, as the interparoxysmal periods become progressively shorter, the degree of consciousness is lessened, and finally continuous coma supervenes, generally to terminate in death. Sometimes the pulse reaches 160 to 200 a minute; while a temperature of 106° to 107° F. is by no means rare. One patient under the writer's care recovered from a very severe attack by being kept for some time in a cold bath, the temperature reaching 109° F.

After several hundred seizures have occurred and a high temperature has been reached, the convulsions abate and the stuporous stage is ushered in with complete coma or collapse. The mouth is foul, the teeth covered with sordes, the tongue parched, the skin bathed in a cold, clammy sweat, swallowing becomes difficult or impossible, and the urine and stools pass involuntarily. While the patient may die in convulsions, some hours are usually passed in coma first. During this period all reflexes are abolished, the respiration is stertorous, and death may supervene at any moment. The final stage is not unlike that of deep uræmic coma just prior to death.

The number of attacks varies from fifty to two hundred or more in twenty-four hours. In one case there were five hundred and nineteen sharply defined grand mal seizures in forty-nine and one-half hours, after which the patient died in deep coma. In this case the initial spasm throughout was confined to the first phalanx of the left thumb.

The *diagnosis* of status should never be difficult. The frequent attacks, the coma, the high temperature, and hurried respirations combine to produce a picture that has no parallel. Without exception the prognosis is extremely grave. As a rule, the more gradual, deliberate, and unremitting the increase in the cardinal symptoms, and that, too, in spite of the most prompt and vigorous sedative treatment, the more unfavorable the prognosis.

The *prophylactic treatment* of status is important. If a patient is habituated to serial attacks, sedatives must be employed early to check them before the more fatal state of status supervenes. In case it has already appeared, bromides in large doses combined with chloral and opium must be continuously employed. Any sudden withdrawal of bromide in the treatment of status is almost sure to hasten death.

**Forms of Epileptic Aura.**[1]—In its literal meaning "aura" signifies a "vapor or emanation from a body which surrounds it like an atmosphere." The term was first used by Pelox more than twenty centuries ago, who thought the fit began as a "spirituous vapor in the veins of the extremities and ascended to the head, when the patient became unconscious."

Aura are classified as sensory, psychic, motor, and irregular. The first signifies a disturbance in common sensation; the second, disturbance in mentality; the third, disturbance in motor apparatus; while the fourth embraces features of two or more, even of all, of these. All aura possess clinical importance when carefully studied and their significance understood and applied. In 815 men and 515 women individual aura were found in 36 per cent. of the total number. In the same series 4 per cent. had psychic and 2 per cent. motor aura. Of the total number, 45 per cent. had aura of some type, leaving 55 per cent. free from such preconvulsive phenomena.

Sensory aura are more common than all the rest combined, and show a wide diversity in character. Phenomena affecting vision predominate in the form of "flashes of lights." In some cases the colors of the rainbow seem to pass in quick succession across the visual field. Sudden blindness alone may constitute all there is of an attack of this character. Such cases are not infrequent. The very curious condition of objects seeming to increase or diminish in size is occasionally encountered as the forerunner of a fit, while now and then pain is felt during the same period in the eyeball, and is supposed to be due to irritation of the fifth nerve. A girl, aged seventeen years, of more than average intelligence, and whose petit mal attacks had been appearing for two years, declared that just before she "had a spell" her mother, who might be standing a few feet away, would seem to "grow taller than the door of the room, and keep getting taller and taller until suddenly everything becomes perfectly black."

*Auditory aura* usually partake of the character of "roaring and voices" heard shortly before the convulsion, the "sound of sea waves," or "roaring and buzzing" in both ears. Such aura occur in 2 to 3 per cent. of all cases. Some epileptics fancy they hear bells ringing, trains rushing by, or the crash of falling trees.

Abnormal sensations of taste and smell occasionally precede attacks and partake of a wide range. Some experience a taste like quinine; others a metallic taste; others "numbness of the tongue," as though it had been anæsthetized. The entire tongue or only half of it may be affected.

*The epigastric aura* is more common than all the other sensory aura combined. It is present in 15 per cent. of all cases, and its type once established is fairly constant. Patients refer to it as a "gnawing, burning, indescribable sensation" at the pit of the stomach that rises to the throat and head before they lose consciousness. Nausea and vomiting are not infrequently experienced just before or after an epileptic seizure. In some cases the patient's breath immediately following an attack is so strikingly foul as to contaminate the entire atmosphere of a large room.

*Psychic aura* appear as sudden abnormal mental states which leave a definite impression of some extraordinary intellectual sensation. Dastoi-

[1] It would be better English to employ the plural "Auræ," but long usage of the singular "Aura" has set the seal of approval so firmly on it that it seems unwise to attempt a change now.

effsky—the Russian novelist—declared he experienced a state of great ecstasy, of supreme joy, just before his attacks occurred, his postconvulsive state being one of the most painful mental depression. A well-educated priest, who was under the writer's care for some years, regularly experienced a similar state of well-being before and a marked melancholy extending over several days after his rather infrequent grand mal attacks, which invariably followed dietetic indiscretions committed late at night. In walking along the street he would suddenly feel "transported" to a state of marvellous joy, to find himself a little later seated on the curb of the sidewalk or in the middle of the street.

The most common psychic aura is a sudden acceleration of the imagination, a quick overflowing of the process of thought, in which "the train of the imagination is urged ahead with trembling, excited haste until the thread is snapped and unconsciousness occurs." Occasionally a fixed sentence, the frequent repetition of a word, or some phrase will present itself to the mind time and again just before the fit. A young male Hebrew of excitable disposition, who was accustomed to have several grand mal attacks a month, rapidly repeated what seemed to be "kammacatchme" half a score of times before unconsciousness supervened. Later, when his attacks became lighter and less abrupt, he changed this pre-convulsive utterance to "Come and catch me," declaring that he felt the seizure coming and that he was asking for help. The earlier expression was then known to be the latter corrupted into one word by too rapid articulation.

*Motor aura* are rare, and it is always difficult to determine whether they constitute a true aura or whether they are the initial motor manifestation of a fit. It is better, as a general rule, to regard them as part of the seizure.

*Irregular aura* are infrequent, and combine several of those mentioned above. They may take the form of whistling, singing, or a series of grunts. An aura rarely changes its type and manner of expression, but any aura may do so.

**Sequelæ.**—About 40 per cent. of epileptics who suffer grand mal seizures bite the tongue, causing injuries—the scars of which ordinarily disappear within forty-five to sixty days. It is a wound of unusual severity which leaves a scar on the tongue lasting more than sixty days. As a rule, injuries of this organ heal rapidly. Scars on the face and scalp, fractures, and dislocations are not uncommon. The nose and clavicle suffer fracture most often. Occasionally the skull, either at the point of the blow or by "contrecoup," the tibia, humerus, and ribs are broken in a fall, or through the severity of muscular contraction; this latter force is exceedingly rare. Repeated dislocations of the shoulder and jaw are not infrequent, and in some subjects they habitually recur.

*Burns* are common, causing ugly scars and disfiguring contractions. Now and then the fingers of both hands are lost by fire. A woman, aged forty years, while automatic after a seizure, grovelled with her fingers in the live coals in a cooking stove. She lost all her fingers at the third joint on both hands. It seems that complete anæsthesia is present during periods of automatism.

Hemorrhagic extravasations about the face and neck are noted now and then after a severe seizure, and are due to a constricting band of clothing about the neck that interferes with the circulation of the blood. As a rule, they disappear in a few days, changing in color like a bruise.

A characteristic sequel is *exhaustion paralysis.* After a series of attacks the patient shows a complete exhaustion paralysis of one leg or arm, or one side of the face, or perhaps one-half of the body. The condition generally subsides and normal motion is restored in most cases in a few weeks, although occasionally, when caused by attacks of long duration and unusual severity, it may persist for months. Exhaustion paralysis was first described by Bravais in 1824. More recent studies of the conditions were made by Clark at the Craig Colony in 1902.[1] Ordinarily it can be diagnosed without the previous history by noting the absence of spasticity, atrophy, or vasomotor changes, all of which are common in old organic hemiplegias and for which exhaustion paralysis might be mistaken.

*Temperature Changes.*—Bourneville and Lemoine first studied the temperature in epilepsy in 1869. The writer's[2] studies of 1000 seizures showed that the temperature is increased in 60 per cent. of all grand mal seizures. Single attacks of great severity increase the temperature from 0.5° to 4°. Purely psychic seizures may cause an elevation of temperature to 102° F. or more. Now and then the thermometer may aid in distinguishing epileptic from hysterical convulsions.

*The eye* may be involved in epileptic seizures, showing dilatation or contraction of the pupil just before, during, or after seizures. Different types of attacks produce different results in this respect, and down to this time the matter has not been sufficiently studied to warrant any conclusions of value.

The so-called "dreamy states," to which writers allude as being characteristic of the epileptic state, are merely the condition of automatism to which reference has been made, and which derives its chief interest from the medicolegal aspects its presence is always likely to assume.

The effects of seizures *on the circulation* was studied in 284 epileptics. In these the pulse ran uniformly above the normal in 64 per cent. In 20 of them it was below 70; in 74 it ranged from 70 to 80; while in 161 it varied from 80 to 100. In the remaining 29 cases it was 100 or over. So far but little reliable work of sufficient extent has been done in studying the blood pressure in epilepsy, and, as with the eye, there are no conclusions to be drawn at this time. It is a promising field for investigation.

**Diagnosis.**—If the physician has opportunity to witness grand mal or petit mal seizures, there is rarely any difficulty in determining the nature of the malady. But when the attacks are psychic or Jacksonian, the diagnosis is more difficult, and inexperienced observers may easily fall into an error. When an epileptic seizure is not witnessed by the physician until the coma stage is established, the cause of the coma may be difficult to understand. In such instances the first question should be, Is the person an epileptic? The determination of this should solve the problem at once; but if the person is not known to be an epileptic, the possibility of alcoholism, apoplexy, diabetic coma, hysteria, or some allied state or condition must be considered.

If *alcoholic coma* is present, the patient's breath will very likely settle the cause. The face will be somewhat congested and red, the eyes watery and inflamed, and there will be an entire absence of acne or scars from falls.

If the coma follows a *cerebral hemorrhage,* the breathing will be stertorous

[1] Clinical Studies in Epilepsy, *Archives of Neurology and Psychopathology,* 1899, ii.

[2] *Medical News,* September, 1901.

and noisy, the face drawn to one side, and the pupils unequal. In such cases the physician should proceed with great caution, relying upon time to help him reach a correct conclusion.

Convulsions due to *paresis* and occurring during the first stages of the disease may be mistaken for epileptic manifestations, but a close study of the mental and physical symptoms will serve to establish a correct diagnosis in most cases. Delusions of grandeur, a hyperexalted state of well-being, delicate fibrillary tremors about the mouth, and progressive physical decline are not characteristic of epilepsy.

*Toxic convulsions* due to lead and certain blood states associated with nephritic diseases may occasionally be mistaken for epilepsy, although the fact that there is usually but one convulsion, and knowledge of the absence of the disease in the subject, should be sufficient to exclude epilepsy.

*Vertigo* may rarely be confused with lesser epileptic convulsions.

The convulsions of *tetanus* are not apt to be mistaken for epilepsy of the commoner types, although in rare cases the tetanoid form of the disease may fail to be properly diagnosed.

Occasionally persons will *simulate* epileptic convulsions to escape punishment for crime, or to gain the sympathy of witnesses who might give alms. The falsity of the disease in such cases can only be determined by a close study of the subject held under confinement. Soap in the mouth in such cases is used to produce a lather to simulate the froth present in genuine epilepsy. The simulator chooses the time and place for an attack; the epileptic does not. The simulator falls in a manner not to suffer injury, while the epileptic is frequently injured in falling. In simulated attacks the pupils suffer no change; in genuine epilepsy they are dilated after most seizures. The urine and fæces are not voided in simulated attacks, while in real epilepsy they are. In closing the hand in contraction the epileptic shuts the fingers tightly *over* the thumb, which lies firmly embedded in the palm, while the simulator closes the thumb *over* the fingers. The true "epileptic cry" cannot be produced by a malingerer, nor can such a person successfully suppress the corneal reflexes. After hard convulsions and rarely after very quiet attacks (psychic), an *exhaustion paralysis* is not infrequently established. The simulator is wholly unable to produce this condition at will, nor can he increase the body temperature, which in true seizures may be increased from 0.5° to 3° F. or more.

A notable point of differentiation is the *tone* and *degree* of muscular contractions. The simulator can reproduce tonic better than clonic contractions, and if a skilled observer will grasp the bare forearms of the patient during the period of clonic contractions, he will find that in true epilepsy these contractions are short, sharp, regular, almost electric-like, and generally powerful in degree as compared with those produced by the simulator at will; here they are irregular, mild, and lacking in the force that characterize the former. Pressure over the supra-orbital nerve will cause pain in simulation, while in genuine epilepsy such is not the case.

**Prognosis.**—Epilepsy is curable in 8 to 10 per cent. of all cases. This statement applies to a result we may expect to attain in the treatment of a large number of individuals representing all types, ages, causes, and periods of duration from the very recent to the most chronic. The first prognosis in epilepsy of which we have record was given by Hippocrates, who, while failing to give definite figures, made this declaration: "If (it attacks)

youths and young adults recovery may take place." After this early pronouncement medical literature seems to have remained devoid of reference to the subject for centuries, and not until the latter part of the seventeenth century do we find it again referred to by Tissot, a French physician, who stated that he had cured a great number of epileptics, and believed that much good could be done toward the arrest of the malady.

Following Tissot and during the next half century, 1803 to 1851, Maisonneuve, Pinel, Esquirol, Valleix, and others gave very gloomy opinions, none seeming to believe there was any cure for the disease. Then came a reaction, and a second French school took a much more optimistic view, declaring that cures could be obtained in 10 to 50 per cent. of all cases. Chief among the more hopeful later writers were Herpin, Russell Reynolds, and Trousseau, the work of the latter (1861) coming almost down to our own time.

In some cases we cannot give a satisfactory prognosis in epilepsy until the patient has been observed for some time—never less than weeks, and more often for months. This is especially true when the convulsions are infrequent or of nocturnal type only. There are conditions in each case that must be studied before we can form an opinion of much value as to the final outcome, and they are as follows: (1) The influence of heredity; (2) the influence of age at the onset; (3) the duration; (4) the frequency, character, and time of occurrence of the seizures; (5) the influence of marriage, of the catamenia, of pregnancy, parturition, and the menopause; (6) the influence of sex; and (7) the influence of accidental factors, such as intercurrent diseases and senility.

1. **Heredity.**—As a rule, when similar heredity is an ascribed cause, the fact does not make the prognosis any less favorable. While this statement seems difficult to accept, it has the approval of the best students of the disease both in this country and in Europe, especially in England, where Gowers was first to call attention to it. Of course, it would be folly to say that anything in the individual's condition acquired by transmission from an ancestor could exert a beneficial influence in the treatment of the offspring, more than, knowing of this cause, greater care is exercised in the supervision of the multitude of small things that mean so much in the treatment of this disease.

2. **Age.**—The epilepsies which occur during the first year of life are nearly always due to heredity or to trauma, if we include in the latter the cerebral hemorrhages of this period, the birth accidents, and asphyxia. Many are also due to the gastro-intestinal disorders and the acute infectious fevers.

Occurring at an age when developmental processes are active, we are more apt to reach a satisfactory conclusion as to prognosis by a little delay than by stating our belief of the outcome at once. The palsy cases can be set down at once as incurable, although marked improvement may be possible in not less than 75 to 80 per cent. of all cases.

In the epilepsies of the second dentition period in children who had no convulsions prior to that time the prognosis is good.

The *influence of puberty* in both sexes is marked in almost every case which develops some years prior to that important epoch, and, as a rule, it is not favorable. Usually the epilepsies that manifest themselves at this time are secondary to others that occurred earlier in life.

In considering the influence of age we may group the adult epilepsies

together. When luetic infection of the brain is the cause the prognosis is generally good, if not for complete recovery, at least for long arrests, which virtually amounts to the same thing. When an adult epilepsy is due to alcoholic intoxication, and proper treatment is early instituted, the prognosis is good. If food poisoning of any kind occurs in conjunction with alcoholism as a cause, the prognosis, while being less favorable than under either alone, is still by no means hopeless.

On the whole, the most favorable age, including all types, is covered by the period between the tenth and thirtieth years, with the exception of cases in men due to syphilis of the nervous system, to alcoholism, and to trauma. When the cause is found in trauma to some part of the central nervous system, the extent of the injury and the condition of the patient generally must determine the prognosis. If the injury is recent and surgery gives prompt and complete relief, the prognosis is good. If such relief is delayed until the convulsive habit is established independent of the primary cause, the outlook is less favorable.

3. **Duration.**—Regarding this little is said, for the reason that all depends upon the character of the seizures. If they are psychic only, the prognosis is always bad, no matter how long the attacks have manifested themselves. If the seizures are Jacksonian, the earlier treatment is instituted the more favorable the prognosis. Attacks of grand mal and petit mal are curable, irrespective of the duration, in all cases in which the fit is more motor in character than it is psychic. Indeed, it can be laid down as a safe proposition that all epilepsies whose expression is more motor than psychic are more often curable than when the reverse is true.

4. The *frequency* of seizures is of value in reaching a prognosis, and each type must be studied separately under this point. It is never wise to give a favorable prognosis in psychic cases, no matter how few seizures have occurred. On the other hand, cures are wrought in grand mal, petit mal, and Jacksonian cases after thousands of attacks have occurred, provided —as was noted above—the fit is more motor than psychic. The next point—the character and time of seizures—is all important. We can lay it down as a proposition that cannot be controverted that psychic epilepsy, pure and simple, that begins as such and terminates as such, is always incurable.

Among the thousand cases which have come under the writer's observation there is but one in which the disease was of this type and in which a cure will probably occur. The attacks were in a girl, aged seven years, and occurred at the outset at the rate of seventy to eighty in twenty-four hours. Two years after she had been under treatment her attacks were reduced to seven or eight in twenty-four hours. After she had been under treatment five years she reached a point where there was one attack in sixty to ninety days. Shortly after this the influence of puberty was felt, and its effect was bad. The attacks again increased in number, occurring thirty to forty times in twenty-four hours. When she had reached her seventeenth year another decrease had occurred, the attacks manifesting themselves once a month to once in six weeks, and because of the natural development, mental and physical, that had taken place during the entire time of her illness, a favorable prognosis was given.

5. **Marriage.**—This is a debatable question which can only be determined in each case. Even though we leave out of consideration any effect or

influence of the marriage state itself, there are sometimes other factors that make a favorable impression and serve to give a better prognosis. So far, however, as the marriage state alone is concerned, and especially because of the stress that accompanies childbirth, with the dangers of infection thereafter, the influence of marriage can never be held to be for good.

The influence of the menopause does not tend to give a better prognosis; the reverse is more apt to be true. In women whose mental stamina is not good we may expect an increase in the mental enfeeblement at this period. Sometimes the attacks just at this time will suffer a radical change in type, and instead of appearing as grand mal or petit mal or in some other motor form, they will occur as psychical epileptic equivalents, and whenever this transition takes place the prognosis is extremely unsatisfactory. When the disease follows childbirth, especially in cases in which there were eclamptic attacks prior to that period, the prognosis is not good.

6. **Sex.**—The influence of sex is practically nil, with the exception, possibly, of a slight percentage in favor of males, this increase occurring after adult life is reached, during which attacks due to syphilis, alcoholism, and trauma are favorable as to the outcome under treatment.

7. The influence of other diseases on epilepsy is an interesting field for study. Measles, scarlet fever, tuberculosis, and other diseases not infrequently serve to check, sometimes permanently, all epileptic convulsions. The philosophy of this is difficult to explain, but that it is true is, nevertheless, a fact. A number of cases have come under personal observation in which the individual suffering from chronic epilepsy contracted tuberculosis, after which no further epileptic convulsions occurred.

**Treatment.**—Not infrequently new cases of epilepsy present so many irregular manifestations that we must study them for some time before adopting a regular course of treatment. A physical examination of every patient should be made, and not infrequently repeated. It should include every organ of the body—especially the nervous system, the blood, the urine, and the gastro-intestinal contents. The condition of the stomach should be an object of special investigation, for we know that nutritive disorders and errors in metabolism play a most important part in the etiology, and the physician who undertakes to treat the disease and fails to make a thorough examination of the entire digestive system does not do all for his patient that should be done.

It must not be inferred that we expect to find the cause of epilepsy in some abnormality of function in the organs examined. At the same time it is well known that such abnormalities do play a role in the production of convulsive manifestations, and this we cannot afford to ignore.

The control of the patient is a matter of very great importance. Epileptics frequently represent various states of degeneracy. Many of them possess will powers weakened by disease, and many are unable to resist temptations which if yielded to are always detrimental. Unless the patient possesses sufficient intelligence to care for himself properly, he should be placed in a sanatorium, or a companion or nurse should exercise supervision over him. For children a nurse should always be employed. It is never possible to satisfactorily treat an epileptic child so long as he remains in his own home. Many influences serve to defeat the purpose in view, and unless the nurse can have full authority in the child's home, it is best to remove the patient to a different environment.

The duration of the treatment is a matter that can never be determined in advance. As a general proposition no patient with epilepsy can be cured under two to three years, and many require a much longer time. In some cases the treatment must be kept up during the lifetime of the patient. In these cases where a cure is not possible long arrests are secured by means of appropriate treatment and through the influence of hygienic living.

**Medical Treatment.**—Before mentioning in detail the drugs to be used, it is well to state that no two patients with epilepsy are amenable to precisely the same treatment, and no two required to be treated the same length of time. Results that may be obtained in one patient in a year cannot be attained in another, precisely similar so far as we can see, in double that time, and the point which we desire to emphasize is the individuality of each patient.

The one drug that has enjoyed the greatest reputation in the treatment of epilepsy, since it was first used for this purpose by Laycock in 1847, is bromide of potassium. At the same time, the one drug that has done more harm in the treatment of this disease because of its indiscriminate use has been bromide of potassium. Of late years we have come to understand that while this drug probably does not possess the power to actually cure the disease, it is capable of suppressing epileptic phenomena for indefinite periods, and that each patient is capable of absorbing and eliminating just so much and no more. Any amount beyond this is only an irritant thrown off by the kidneys and through the skin, to the detriment of both.

It is never necessary to produce a bromide acne. When such occurs it is distinct evidence that the patient is receiving too large a dose. By careful trial we can determine exactly the size of dose it takes to control the seizures, and once this point is established the amount should not vary unless there is a change in the patient's condition which makes it desirable that his serial attacks or status epilepticus may be deferred or may be arrested, in either of which events the size of the dose can, for the time being, be materially increased.

The disadvantages due to the use of bromide of potassium are as follows: On the alimentary canal it acts as an irritant, and interferes with the reflex activity of the stomach in a way to check the normal secretion of the gastric juice, resulting in an impairment of digestion. It causes obstinate constipation, which should always be avoided in epilepsy whenever possible. It produces a heavy coating of the tongue, foul breath, bad taste in the mouth, loss of appetite, and sometimes an obstinate diarrhœa and nausea. On the general nutrition of the body it acts unfavorably, decreasing metabolic changes. It has a deleterious action on the entire muscular system, producing a condition of enfeeblement, causing an unsteady gait, a depression, and often complete loss of sexual vigor, loss of memory, a slight degree of aphasia, and a tendency toward the misuse of words and sentences. It lowers bodily temperature by depressing the heart's action and by stimulating the vasoconstrictors. Its most marked effect, however, is on the skin, producing widespread, unsightly eruptions. In skins that are especially sensitive these eruptions may take the form of pustules and even of ulcerations of large extent.

The bromide salt is eliminated very slowly, and escapes from the body unchanged. It is found in all of the secretions, including the sweat, urine, semen, milk, and fæces, which demonstrates the extent to which the system

becomes bromidized, and raises the question as to whether its action on the brain alone is the only manner in which it demonstrates its value in the treatment of epilepsy. The drug can be administered to secure the best possible results without causing unsatisfactory manifestations.

It is well to begin with a small dose—in an adult 5 grains (0.3 gram) three times a day, and in a child under ten or twelve years, 2.5 to 3 grains (0.2 gram) three times a day, always giving the drug in plenty of water and in conjunction with some suitable adjuvant. A course of calomel in broken doses, followed by a saline cathartic or a course of castor oil, should be administered before the bromide is begun, and it is well as a routine practice to give one or the other of these every two or three weeks during the entire period of bromide administration.

Especial pains must be taken to keep the emunctories in the best possible action. The skin can be kept so by means of steam or hot water baths and by massage two or three times a week. As soon as any evidence of eruption on the skin appears, the size of the dose should be decreased. So long as this does not occur and the attacks are held in check the small dose should be adhered to. If the skin does not show an eruption, and if the attacks show a tendency to increase, the size of the dose must be increased also. Every epileptic has a point of tolerance that should be studiously sought, and once found, always observed afterward. There is no other drug whose administration has the same peculiarity, with the exception of potassium iodide.

The bromide of soda is useful in some cases in which the bromide of potassium does not meet the indications. As a rule, it is also better in women than in men. It possesses distinctly sedative properties on the reproductive organs in women, and it can be pushed to a larger dosage without deleterious effects than is the case with the potassium salt. In women epileptic attacks are prone to express themselves with far greater frequency around the menstrual period, and we must often largely increase the dose of sedative drugs at that time. The bromide of soda is the best drug we have for the purpose.

*Bromipin.*—Of late years efforts have been made to find substitutes for the bromide salts that would possess their advantages but none of their disadvantages. One of the newer remedies which meets these requirements in many cases in a safe way is bromipin, which is pure bromine in the oil of sessamum. It is best administered in the form of an emulsion composed of simple syrup, spirit of peppermint, and gum arabic. This formula is more useful in feeble and asthenic individuals than in cases of any other type. It generally causes a gain in weight.

The chief mistake in the use of bromipin is to give too small a dose. This should be regulated on a par with the bromide salts and an amount given equal to a similar amount of the salts.

Of the vegetable preparations used in epilepsy, the fluid extract of adonis vernalis has been used extensively. It possesses distinct motor depressant properties. The dose is 2 to 3 drops three times a day, and is best administered in conjunction with the bromide salts.

Borax was first used in the treatment of epilepsy about twenty-five years ago, and while it may be of value now and then in doses ranging from 15 to 20 grains (1 to 1.3 gram) three times a day, it is generally much less useful than the bromide salts.

Chloral hydrate is not much used during the interparoxysmal periods. It is useful only in the treatment of status epilepticus when heroic measures are required. It is always of use in states of mental excitement and insomnia, which occur so frequently in epileptics. In status epilepticus, when the patient becomes unconscious and evidences of exhaustion appear, chloral should be withdrawn.

Nitroglycerin is frequently of great value in cases of senile epilepsy presenting evidences of arteriosclerosis. The effect of the drug on the heart and circulation is not infrequently most positive. Its action is of short duration, which requires that it be frequently administered.

Zinc is one of the oldest remedies used in epilepsy, and was in evidence long before the bromides had been discovered. Its action is similar to that of these salts, although milder.

Another of the newer vegetable preparations which was brought forth by a physician in South Carolina some years ago is that of solanum carolinense. At our hands it has given, on the whole, very satisfactory results, especially in the treatment of the severer forms of epilepsy. It is not a drug for use in the regular types.

Tincture of simulo was first recommended by Eulenburg in 1888. It comes from a plant of the hyssop family in South America, and in doses of 2 to 3 drams three times a day has attained considerable reputation. While its action is not positive in some cases, it is worthy of a trial when the better-known remedies have not been successful.

Trianol was very generally adopted some years ago, but it has fallen into complete disuse at the present time. It was administered in aërated water, which practically doubled the effects attained.

The coal-tar derivatives, including antipyrine, phenacetin and acetanilide, have all been used with more or less effect, although none of them can be said to possess distinct properties of value. They are useful, however, in allaying postconvulsion headaches, which are frequently of extraordinary severity and refuse to yield to ordinary measures.

Various preparations of iron are useful in some cases. A different type of case is found in chlorotic girls and women about the time of puberty and later. It is well to combine with the iron small doses of arsenic, the purpose being to build up the blood system as rapidly as possible. Iron in this way does not have a distinct effect on the nervous system, but affects it through the vascular system.

Electricity has been tried in one form or another, and while it possesses value as an alterative in some cases, it has no power in the immediate control of attacks.

Chloroform is useful in suppressing attacks that threaten to run into status, and it is also useful in checking status itself. Sometimes, however, it will fail in this respect. It should never be pushed to the extreme.

*Hydrotherapy* has been used extensively in one form or another, and it has a place of value in the treatment of the various epilepsies we had no conception of ten or even five years ago. Since we have come to see and fully understand the value of rapid elimination, the removal as quickly as possible of all toxic matters from the body, we have come to use therapy more and more. It is a remedy that is suitable for use in 30 to 40 per cent. of all cases, and it has a great value in the treatment of the alcoholic epilepsies. Usually it is best administered in the form of steam baths

or hot packs, the essential purpose being to procure the greatest possible activity on the part of the skin.

Various serums have been used, but they have apparently been discontinued entirely, as nothing is heard of them at the present day.

*Systematic treatment* is essential. Once it is begun it should not be interrupted if possible under two to three years. The control of the patient is a matter of great importance. In cases of intelligent adults supervision by another person may not be necessary, but when the mental faculties are impaired in adults, and always in the cases of children, supervision by another person is essential. No epileptic child can be satisfactorily cared for in its own home, especially where it is under the control of the parents. The best plan is to place the child in a private home, preferably in the country, or in a private sanatorium. The education of epileptic children should be postponed until health is regained, which is of much greater consequence than a little learning. Smoking, as a general rule, should be forbidden, especially in all minors.

Exercise out of doors and as much out-door life as possible are desirable in every case. Exercise must never be carried to the point where it induces more than a healthy fatigue.

The marriage of epileptics, as a general rule, should be forbidden. There may be rare cases, and these are governed usually by domestic considerations, in which marriage is permitted, provided there be no offspring. This, however, is a matter which cannot always be regulated, for which reason it may be best to lay down the hard and fast rule that no epileptic should ever marry.

The *surgical* treatment of epilepsy is beginning to assume an importance not heretofore understood, for the reason that we have expected results too soon. We now know that a surgical operation may be only the beginning of improvement which does not reach its height for some years. Many such cases have come under personal observation. Other forms of treatment should not be discontinued when an operation is performed, and the operation should be regarded as supplemental. About 8 per cent. of all cases are amenable to surgical treatment.

# CHAPTER XVII.

## SYPHILITIC AND PARASYPHILITIC DISEASES OF THE CENTRAL NERVOUS SYSTEM.

By B. SACHS, M.D.

The recognition of these disorders is not easy, but during the past two decades great advances have been made in the differential diagnosis between syphilitic and other affections of the central nervous system. The discovery of the Spirochæta pallida (Schaudinn) and the serodiagnostic investigations of Wassermann have helped to clear up the etiology of many doubtful conditions. It is impossible to overestimate the support which these methods give to the accuracy of diagnosis. Nevertheless, laboratory methods are an addition to, and not a substitute for, clinical observations.

Among the syphilitic and parasyphilitic affections of the nervous system we may class the following: (1) Cerebrospinal syphilis; (2) acute and chronic specific myelitis; (3) syphilitic spinal paralysis (Erb's type); (4) tabes dorsalis; (5) general paresis.

### CEREBROSPINAL SYPHILIS.

Almost every manifestation of brain or spinal cord disease might be enumerated under this heading. At least, there are very few conditions pointing to focal disease in the brain or in the medulla spinalis which might not at one time or another be taken to be due to preceding syphilitic infection. It is not the intention to enumerate all such diseases, but to select for especial mention a few of the clinical conditions which are so distinctly specific that they can by clinical examination alone be recognized as a form of syphilis of the nervous system. More than eighteen years ago the present writer[1] attempted to impress this fact upon the medical public by helping to establish a condition known as multiple cerebrospinal syphilis. The resemblance in name to multiple cerebrospinal sclerosis was favored because, as a matter of fact, the differential diagnosis between these two conditions must frequently be made.

By *multiple cerebrospinal syphilis* we mean an affection which involves distant parts of the cerebrospinal axis and gives rise to a multiplicity of symptoms, some of them due to a lesion of parts as widely apart as are the brain axis and the sacral segments of the cord.

**Etiology.**—Under this heading nothing need be said except that this form is frequently due to a more or less recent infection and may appear at any time within a few months to a few years after the initial lesion. It

[1] *New York Med. Jour.*, 1891, liv, 309.

is also well to insist again and again that the disease may often be preceded by a history of soft chancre, as well as of hard chancre, and that the absence of a satisfactory history of preceding syphilitic infection need not militate against the diagnosis when the clinical picture is distinctly in favor of such. It is in these forms of disease that the examination of the cerebrospinal fluid, as well as of the blood, may give valuable assistance, although, as Nonne[1] has shown, in these very cases of cerebrospinal lues, the blood may give a positive Wassermann reaction, whereas the cerebrospinal fluid may be negative. To a certain extent the writer's experience supports this view, although he has not found a negative reaction in as large a proportion of the cases as Nonne has. On the other hand, let it be stated distinctly, as Wassermann himself pointed out in his first publications, that a negative reaction cannot be taken to prove the absence of syphilitic poison.

**Pathology.**—This form of specific disease may safely be said to be due in a considerable number of the cases to a chronic syphilitic involvement of the meninges of the brain and spinal cord. According to the predominance of the symptoms we may, therefore, have a pachymeningitis or leptomeningitis cerebri specifica, or a pachymeningitis or leptomeningitis specifica spinalis. That from this meningitis the process may extend into the brain or spinal cord need not be insisted upon, and if some writers speak of meningo-encephalitis specifica or of meningomyelitis specifica, the morbid process itself unquestionably remains the same. With this widespread involvement of the membranes of the brain and spinal cord there may be special gummatous deposits, and this accounts for the fact that in so many instances we have symptoms pointing with special force to one particular locality, and associated with these other symptoms pointing to involvement of remote parts. A gumma in the vicinity of the crus may be associated with a general leptomeningitis, and we can well understand, therefore, that in such a case there may be complete ophthalmoplegia interna and externa, without other cranial nerve symptoms, associated with a spastic paraplegia of the lower extremities and involvement of vesical and rectal functions, since the dorsal and lumbar portions of the cord are sites of predilection for the specific spinal pachymeningitis and leptomeningitis. Aside from involvement of the brain and spinal cord substance and the meninges, we may have a specific endarteritis as the sole expression of a more or less chronic syphilitic process. In such conditions attacks of hemiplegia due to thrombosis are very apt to occur, and there is no doubt, although these conditions have not been so well observed, that specific disease of the spinal cord bloodvessels with subsequent softening may give rise to various clinical groupings pointing to involvement of the spinal cord itself.

It is characteristic of syphilitic disease of the nervous system that the granulation tissue (granular proliferation) starts from the smallest capillaries, either from the capillaries of the connective tissue or from the capillaries (vasa nutrientia) of the larger bloodvessels. Oppenheim was one of the first to lay special stress upon the gelatinous character of the specific thickening of the spinal and cerebral meninges. Mention must also be made of the fact that a primary degeneration of the cranial nerve nuclei may be an expression of general syphilitic infection.

[1] *Syphilis u. Nervensystem*, second edition, Berlin, 1909 (contains full literature to date).

Many years ago Jürgens insisted that cerebrospinal syphilis began in the brain and descended into the spinal cord. So many reports have been made of syphilitic changes affecting either the brain or spinal cord simultaneously, or beginning in the spinal cord and affecting the brain secondarily, that this proposition of Jürgens can no longer be maintained.

**Symptoms.**—The disease may come on in acute or subacute fashion. The onset of the localizing symptoms may be preceded by general symptoms such as are known to be due to a general specific disease, particularly of the brain, and it may be noted that in a very large majority of the cases cerebral symptoms precede those of a spinal character. The earliest symptoms of this condition are apt to point to involvement of the base of the brain and particularly to involvement of the various cranial nerves, and among these the third nerve and the sixth are most frequently affected, while in others the fifth, seventh, eighth, and twelfth nerves may be involved, according to the special distribution of the lesion. An early ptosis, an ophthalmoplegia externa and interna, unilateral deafness, abnormalities of sensation and even dissociation of sensation within the distribution of the fifth nerve, paralysis of one or both vocal cords, paralysis and atrophy of the tongue with accompanying disturbances of deglutition and of speech, and, of course, unilateral facial paralysis—all these symptoms may appear early in the course of a multiple cerebrospinal lues. Moreover, cranial nerve symptoms may be associated with paralysis of the upper and lower extremities. Not infrequently the upper extremities escape, so that we have the peculiar association of cranial nerve palsies with a paraplegia of the lower extremities. In the vast majority of the cases this paraplegia, whether of the upper or lower extremities, is of the spastic order, due, no doubt, to the more frequent involvement of the lateral portions of the cord, or to the fact that when there is a meningeal infiltration the invasion from the meninges affect first the tracts running through and near the lateral columns. That the spastic paraplegia would be associated with increase of the deep reflexes and with a disappearance of the superficial reflexes in the regions affected goes without saying. The vesical and rectal functions may or may not be disturbed. The paraplegia may at times be flaccid, and only very recently Hoffman[1] has reported cases of the order of poliomyelitis anterior unquestionably due to syphilis, while Spiller[2] has reported a syphilitic acute anterior poliomyelitis due to thrombosis of the cervical anterior median spinal artery.

The clinical picture may, therefore, involve one or more parts of the central nervous system, and these parts may be remote from one another, so that the mere fact that a single lesion cannot explain all the symptoms in a given case, and that there must be a multiplicity of lesions, points with strong probability to a general luetic process, for, as will be seen later on, there are but few other processes that give rise to similarly widespread affections.

In the article on tabes reference is made to the pseudo-tabes syphilitica. We need not, therefore, at present refer to the possible involvement of the posterior columns of the cord giving rise to symptoms resembling tabes, and to their association with symptoms pointing to distinct involvement of the brain axis. But cases of this sort can be differentiated from true tabes

[1] *Neurol. Centralbl.*, October 16, 1909.
[2] *Journal of Nervous and Mental Disease*, 1909, xxxvi, 601.

by the very multiplicity of symptoms, by the association of tabic symptoms with a number of ocular nerve palsies, and not merely by the association with ptosis or with rectus externus palsy, as happens in some cases of tabes dorsalis. Whatever the localization may be that is indicated by the clinical symptoms, there are other peculiarities which lend support to the diagnosis of a multiple cerebrospinal lues. The writer summarized these so long ago as 1893.[1] They are: (1) The unusual distribution of the disease over the greater portion of the brain and spinal cord axis; (2) the relatively slight intensity of the morbid process as compared with the extensive area involved, evidenced by the preservation of some of the functions with complete loss of others; (3) a rapid disappearance of some of the symptoms and a very chronic persistence of others; and (4) the frequent history of other cerebrospinal accidents. Thus, some of the patients give a history of a preceding hemiplegic attack with more or less complete recovery. Others give a history of temporary paralyses, of temporary aphasia; still others, of a preceding attack of paraplegia which may have lasted a few weeks or a few months, followed by satisfactory recovery. It is the remissions and exacerbations, the recoveries and relapses, that are more characteristic of a cerebrospinal syphilitic process than of any other condition.

**Diagnosis.**—The chief task is to differentiate multiple cerebrospinal syphilis from *multiple cerebrospinal sclerosis*, which can be distinguished, however, from the latter by the absence of intention tremor, of nystagmus, and of scanning speech, and, above all, by the frequent remissions in the course of the syphilitic affection. And yet, as Oppenheim has pointed out again and again, remissions are characteristic also of disseminated sclerosis. However, in disseminated sclerosis there is rarely such complete recovery as one not infrequently sees in multiple cerebrospinal syphilis. At the present day the examination of the cerebrospinal fluid and of the blood will give some assistance, and if the findings in the blood are positive, disseminated sclerosis may be excluded, since it is tolerably certain that it is the rarest thing in the world to have disseminated sclerosis develop on the basis of a preceding syphilitic disease.[2]

The only other widespread affections of the central nervous axis which might resemble multiple cerebrospinal syphilis are a *cerebrospinal tuberculosis* and a general *carcinomatosis* or *sarcomatosis*. From tuberculous disease the syphilitic affection may be distinguished by the more rapid course of the former and by the fact that the tuberculous trouble very often leads to a strictly limited basilar or spinal meningitis with formation of one or more solitary tubercles. Moreover, the tuberculous process affects the lower portion of the brain axis and the upper portion of the spinal cord much more frequently than it does the dorsolumbar region. A general sarcomatosis or carcinomatosis may give rise to symptoms somewhat resembling those of multiple cerebrospinal syphilis, but, as a matter of fact, a difficulty in diagnosis rarely arises, for the preceding history of primary neoplasm in some other organ, particularly in the breast or in some of the glands or bones, puts the physician on his guard. The distribution of the symptoms may be very similar, but the greater gravity of the disease is

[1] Syphilis of the Spinal Cord, *Brain*, 1893, xvi, 405.

[2] Nonne's monograph contains an excellent chapter on cyto-diagnosis and the Wassermann test.

easily recognized. As a rule, the symptoms are steadily progressive, although, as in a patient recently seen, the symptoms may be stationary over a period covering six to eight months. The autopsy, however, showed a general carcinomatosis extending all the way up and down from the cervical to the sacral region, involving the vertebræ and pressing upon the spinal coverings, but the spinal cord itself had only been affected by compression. The tissue itself had not been invaded by the carcinomatous process. In other cases, however, the carcinomatous process may cause complete destruction of the cord. To quote from a previous article of the present writer, we may state "that there is a frequent combination of symptoms that can be recognized as a special form of disease—a multiple cerebrospinal syphilis due chiefly to a specific meningitis of the brain and cord, which is apt to form special deposits in different parts of the central nervous system, or by proliferation to make inroads into the brain or cord, and that the association of cranial nerve affections with special symptoms, most frequently of a spastic type, in the presence of a distinct history of syphilitic contagion and of repeated remissions, gives strong evidence of this special form of disease."

**Treatment.**—This does not in any wise differ from the specific treatment which is discussed in the case of tabes dorsalis, excepting that in this disease, particularly when it affects vital parts, as it sometimes does when the symptoms point to involvement of the centres in the pons and medulla, treatment should be pushed so energetically as to give prompt relief. When the vital centres are in danger good judgment and great therapeutic energy are in order.

**Acute and Chronic Syphilitic Myelitis.**—The frequent localization of the syphilitic process in the dorsal and lumbar regions of the spinal cord gives rise to a clinical picture pointing to an acute, subacute, or chronic myelitis. The absence of root symptoms (pains, paræsthesia, etc.) indicates that the spinal meninges have escaped, but the association with distinct cerebral luetic symptoms, such as pupillary immobility or strabismus, goes to show that the specific myelitis is an especially restricted form of the cerebrospinal syphilitic disease. In the variability and incompleteness of the symptoms we have the same earmarks of the luetic process that were dilated upon in the section on cerebrospinal syphilis. The features of syphilitic myelitis are discussed elsewhere (page 240), and require no extended discussion here. According to the special areas of the cross-section of the cord which may be involved in the luetic process the symptoms will naturally vary. The clinical pictures of acute or chronic poliomyelitis anterior or of amyotrophic lateral sclerosis may be presented when the disease is, in reality, an acute or subacute incomplete luetic myelitis. Almost any form of myelitis may occur due to syphilis. In making the diagnosis, it is of the utmost importance to consider other evidence of constitutional syphilitic disease which the patient may have presented at the time of examination or in preceding years. The writer has seen patients treated at one time for a transitory ocular palsy, at other times for an attack of hemiplegia, and then again for a spastic paraplegia of the lower extremities. One patient, who has passed through these three phases, in addition has developed a condition of specific dementia bearing a close resemblance to general paresis. The previous occurrence of such accidents, the frequent recoveries from more or less serious paralytic conditions, are features common only to syphilitic spinal

disease. It should also be remembered that a specific meningitis or meningomyelitis may go hand in hand with the formation of one or more gummata, and the difficulties of diagnosis may therefore be increased by the fact that symptoms pointing to tumor of the cord may either precede those of myelitis or be superadded to them.

**Specific Spinal Paralysis (Erb's Type).**—This is discussed elsewhere (page 248), and is included here for completeness. Erb called attention to a series of symptoms which he considered pointed indubitably to syphilitic spinal paralysis, but Oppenheim and the writer, while recognizing the frequent occurrence of Erb's type, were more inclined to consider it *a* syphilitic spinal paralysis and not *the* syphilitic spinal paralysis. The experience of later years has strengthened this view. Erb's type was characterized (1) by the usual symptoms of spastic paraplegia with its peculiar gait, carriage, and movements; (2) by marked exaggeration of the deep reflexes; then by muscular contractures which are slight as compared with the exaggeration of the reflexes. Involvement of the bladder, a slight, yet distinct, disturbance of sensation, gradual onset of the disease, and a decided tendency to improvement, were further characteristics. This same series of symptoms had previously been recognized by Rumpf, and later Friedman described their occurrence even in children. Erb was inclined to attribute this special form to a diffuse meningomyelitis, but Nonne has shown very conclusively in several cases which came to autopsy that Erb's type of syphilitic spinal paralysis may be the expression of a combined systemic degeneration involving the pyramidal and cerebellar tracts and the tracts of Goll.

Before concluding, a quotation is taken from the chapter in the writer's book on the *Nervous Diseases of Children:* "One may suspect specific disease of the cord if the patient presents symptoms of paralysis, whether they be of the spastic or flaccid character and whether the contractures be slight or not, provided the patient furnishes evidence of a morbid process affecting a very large part of the cord and yet showing a relatively slight intensity at any given level of the cord. He may, for instance, exhibit the symptoms of extreme paralysis, spastic or atrophic, with partial or slight anæsthesia of the parts paralyzed, with little or no involvement of the bladder, or, as often happens, he may present traces of specific disease in other parts of the central nervous system. The chief difference, according to this, between the ordinary forms of myelitis and the specific diseases of the cord can be understood if we remember that the symptoms of an acute or subacute myelitis prove that the entire cross-section of the cord is affected almost simultaneously and to an equal degree, whence it follows that in such cases severe paralysis is likely to be associated with severe anæsthesia, with marked contractures, with absolute loss of vesical and rectal control, with serious trophic disturbances, and so on; whereas, in the cases of spinal syphilis the morbid process invades the cross-section of the spinal cord partially and slowly. We may, therefore, find symptoms which point to a very marked affection of one or more of the systems of the spinal cord and to relative immunity from disease of the gray matter or other portions of the cord. Thus, we may have extreme paralysis, but only slight anæsthesia; or extreme loss of power with relatively slight rigidity, as Erb pointed out. Furthermore, in the ordinary cases of myelitis the symptoms point to a certain portion of the spinal cord at which the disease is most intense, while

in cases of syphilis of the spinal cord the clinical symptoms show that the disease involves a very large portion, if not the entire spinal cord, yet affects each single segment relatively little. A still further aid to differential diagnosis is the very frequent involvement of the brain, at the same time that the majority of the symptoms point to disease of the spinal cord, or if these two sets of symptoms do not set in simultaneously, we frequently have in a patient who presents symptoms of a chronic specific myelitis the history of a preceding illness in which the symptoms were of a cerebral, rather than of a spinal, character. . . . The rapid and often unexpected recovery, as well as the relapses, help also to distinguish these cases from the usual forms of myelitis."

## TABES DORSALIS.

**Synonyms.**—Tabes dorsalis is also known as locomotor ataxia, posterior spinal sclerosis ("*Ruckenmarkschwindsucht*" of the Germans).

**Definition.**—Tabes dorsalis is a chronic, more or less progressive disease of the central nervous system, exhibiting its chief morbid changes in the spinal ganglia, in the posterior roots and posterior columns of the spinal cord; it is characterized clinically by a very definite series of symptoms, among which the Argyll-Robertson pupil, the lightning pains, the girdle sensation, the loss of the deep reflexes, and the ataxic gait are the most prominent.

**Etiology.**—During the last third of the nineteenth century the neurologists were divided into two camps with reference to the etiology of tabes dorsalis. The one faction, led by Fournier and Erb, claimed that tabes dorsalis was a true syphilitic affection of the spinal cord; the other faction, led by Leyden, believed that syphilis was only one of a number of different causes that might produce this disease. The latter accused the adherents of the opposing theory of attaching undue importance to syphilis. As a matter of fact, the statistical data gathered by Erb and his pupils proved beyond a doubt that in about 95 per cent. of the cases of tabes dorsalis there had been preceding syphilitic infection, and we may add to this that when the disease afflicted women, as it does much more rarely than men, the percentage of preceding syphilitic infection is even higher. The writer has for years paid the closest attention to this point, and in his own private practice, as he has stated repeatedly, he has rarely seen a case of tabes in which the etiological factor of syphilis could be excluded. In only one case was he certain of this, and that was in a patient who had been entirely well until the age of thirty-five, and had never exhibited the slightest sign of syphilis, nor had he contracted any infection. He was struck by a piece of shell after a dynamite explosion, and within six weeks of that time developed the first symptoms of what proved to be a rapidly progressive form of locomotor ataxia. More recently the serodiagnostic researches with the Wassermann method have revealed the tremendous importance of syphilis in the etiology of the disease, the percentages of positive findings varying from 90 per cent. (Nonne, Wassermann, and Plaut) to 66.6 per cent. (Sachs, Castelli). The lower percentages recorded by the latter may, perhaps, be due to the established practice in this country of subjecting tabic patients to prolonged

antisyphilitic treatment. Erb's[1] dictum of years ago, that a person who has not had syphilis was not likely to have tabes dorsalis, has been fully substantiated by later experience, and while trauma and possibly extreme exertion[2] may be occasional factors, we are safe in stating that syphilitic infection is by far the most common cause of tabes dorsalis.

Locomotor ataxia appears, as a rule, five to twenty years after the initial infection. If syphilis is acquired late in life, syphilitic forms of spinal disease may appear within a very few years. The writer has seen the first symptoms of tabes dorsalis, including optic atrophy, in a man, aged fifty-five years, appear within one year of the initial infection. The preceding lesion is almost invariably an ulcus durum, but patients with tabes dorsalis not infrequently give a history of ulcus molle.

**Pathology.**—The chief anatomical change is a degeneration of the posterior columns of the cord. Even the gross specimen reveals a distinct grayish discoloration of these parts. Under the microscope it is seen that the degeneration begins in the columns of Burdach at the level of the upper lumbar segment. It must be noted that this is the area through which the posterior roots pass. At higher levels the columns of Goll are also involved at a very early period of the disease. At a later stage the entire posterior columns of the lumbar and dorsal segments are completely degenerated. In the cervical segments the degeneration may be limited to the columns of Goll; later on here, as elsewhere, the greater part of the posterior columns is degenerated. In the morbid process the nerve trunks are degenerated, while the glia may remain either normal or may proliferate. The disease is not strictly limited to the posterior columns. The columns of Clarke and the posterior horns (particularly Lissauer's root zone) are frequently involved. The posterior roots are diseased so often that many investigators (Leyden, Redlich, Oppenheim) believe the changes in the spinal cord to be due to early involvement of the posterior roots. Oppenheim,[3] and Thomas and Hauser[4] have traced this degeneration to the spinal ganglia, the trophic centres of the posterior roots. The morbid process may extend into the medulla oblongata, involving the spinal root of the trigeminal nerve (thus accounting for the rare and interesting forms in which trigeminal symptoms appear early in the disease). The solitary bundle, the sensory motor nuclei of the fifth nerve, the auditory nucleus (rarely), the hypoglossal nucleus, and the nucleus of the vagus nerve may become involved. In addition to all of these and much more frequently, we may find a degeneration of the optic nerve (simple white atrophy). Other cranial nerves may undergo simple atrophy. Oppenheim found a degeneration of the vagus and recurrent laryngeal and the glossopharyngeal nerves. Obersteiner and Schiff accounted for the hemiatrophy of the tongue by a degeneration of the hypoglossal. The auditory nerve is, of all cranial nerves, the one most rarely affected. Oppenheim's finding of a degeneration of the Gasserian ganglion is related to the involvement of the sensory root of the fifth nerve. Atrophy of the sensory nerves of the skin has been shown to occur by the researches of Westphal, Dejerine, Siemerling, Oppenheim, and others.

[1] *Zeit. f. Nervenheilk.*, xxxiii; also Fischler, *Zeit. f. Nervenheilk.*, xxviii. See also Erb's article contributed to the *Deutsche Klinik*, vi, 807.

[2] Poisoning by lead and ergot produces clinical and anatomical conditions similar to tabes dorsalis.

[3] *Archiv f. Psychiatrie*, vol. xviii.

[4] *Nouv. Icon.*, etc., 1904.

All the changes enumerated hitherto refer to the sensory fibers of the periphery or of the spinal cord. French writers (Charcot, Pierret, Raymond, and others) have shown that in some cases the anterior gray horns are also involved. This will account for some of the cases of tabes with muscular atrophy, although it is doubtful whether the peripheral nerves may not be responsible for some of these muscular wastings. In conclusion, we may state that while the morbid process of tabes dorsalis is essentially a posterior spinal sclerosis, it may involve other parts of the cord and of the central nervous system, as well as the peripheral nerves. The toxic agent may exert its effects upon almost any or every part of the nervous system, although it has certain definite sites of predilection; but to make tabes dorsalis a cortical disease because cortical fibers undergo atrophy (according to Jendrassik) is carrying the argument a little too far. All these changes may be secondary to changes in the central sensory neurones (Strümpell). The opponents of the Fournier-Erb doctrine of the specific origin of tabes dorsalis have urged that there is nothing characteristically syphilitic about the morbid process; there is some truth in this, but the force of this objection is lessened by the findings of Hoffmann, Kuh, Minor, Nonne,[1] Sachs, and others, who have shown that tabes dorsalis does occur in association with a typical specific meningitis.

**Symptoms.**—With the possible exception of general paresis, there is no disease presenting such a multiplicity of symptoms, and it is doubtful whether any one patient ever presents even a bare majority of the symptoms. The clinical picture will be described as it is outlined in the writer's mind from a large individual experience, even if there be some departure from the description as it is so commonly taken over from one text-book into another. First of all, there are two distinct groups, one the ataxic and the other the ophthalmic. The ataxic group represents the classical type. In this group of cases the optic nerve symptoms are developed very late or not at all, while all the ataxic and sensory symptoms attain to fullest development. In the ophthalmic group optic nerve atrophy is one of the earliest and most pronounced symptoms of the disease, leading to early blindness, while the ataxic symptoms in this group are very imperfectly exhibited. While the latter group presents fewer symptoms, the early development of amaurosis makes it the form most to be dreaded. Both groups have, however, a majority of symptoms in common. It has been customary to divide the symptoms into (1) those of the prodromal or pre-ataxic stage, and (2) those of the ataxic stage. The prodromal stage may last for months or years, and yet the final diagnosis cannot be safely established until several of the cardinal symptoms have appeared. The presence of three of the cardinal symptoms is sufficient for this purpose, and among those cardinal symptoms we must rank subjective as well as objective signs.

The cardinal symptoms of tabes dorsalis (given approximately in their order of importance) are:

1. Lancinating pains.
2. Argyll-Robertson pupil.
3. Loss of deep reflexes, particularly of the knee-jerks and of the Achilles tendon reflex.

[1] See Nonne's monograph and the writer's article in the *New York Medical Journal*, 1894, lix.

4. Romberg symptom (swaying of the body with the eyes closed).
5. Girdle sensation in various forms; hyperæsthesia as well as delayed sensation.
6. Hypotonia of the muscles.
7. Bladder disturbances (rectal insufficiency is rare).
8. Ataxic movements of the lower extremities. (Ataxia of the upper extremities is relatively rare.)
9. Sexual weakness.
10. Cranial (more particularly ocular) nerve palsies, strabismus, double vision, etc.
11. Optic nerve atrophy, single white or primary white atrophy.
12. Visceral crises.
13. Trophic disorders.

Any combination of these symptoms is possible, but the majority of cases are characterized for a long period of time by lancinating pains, some forms of hypæsthesia, pupillary symptoms, and by the loss of the deep reflexes. After the lapse of some considerable period of time the ataxia and the bladder symptoms appear. We must enter in detail upon the description of these various symptoms.

The *lancinating pains* occur, according to Erb, in 90 per cent. of the cases, and in 70 per cent. of all cases of tabes they constitute the earliest symptom. They are lightning-like, severe, darting, or boring pains occurring in any part of the body, most frequently down one or both legs within the distribution of the sciatic, around the calves, and into the heel or toes. Sometimes they are particularly annoying within the distribution of the crural nerves. These lancinating pains occur less frequently in the upper extremities, are localized often enough in the thorax and sometimes even in the face (tabes superior). The lancinating pains are so commonly sciatic in distribution that persistent sciatica, particularly if bilateral, is suggestive of tabes. These lancinating pains come on without rhyme or reason when the patient least expects them. Any slight change of temperature or any change in the weather may be sufficient to produce them. It is not astonishing therefore, that the pains are often misjudged as "rheumatic" or "neuralgic." If the patient complains of vague and severe pains, it is well to examine the pupils, and if lightning-like or lancinating pains are associated with Argyll-Robertson pupils, or with loss or impairment of the deep reflexes, one may suspect the presence of tabes dorsalis.

Hyperæsthesia may be noted within the special areas of pain, so that the pressure of the clothing or of the bedclothes becomes intolerable. Closely allied to the subjective sensory disturbances just recorded are various forms of paræsthesia, such as formication, a feeling of "pins and needles," a feeling as if the patient were walking on velvet or cotton, peculiar feelings of tension about the perineum and the genitals. Formication in the distribution of the ulnar nerve is the commonest among those occurring in the upper extremities. Disturbances of sensation around the thorax give rise to the well-known girdle sensation—a tolerably early and very characteristic symptom of the disease. These sensory disturbances are often the initial symptoms, and can be accounted for by the early involvement of the posterior root fibers and their prolongations into the cord. Tabic pains are, to a degree, early root symptoms.

The *Argyll-Robertson pupil* occurs in at least 80 to 90 per cent. of all cases.

It implies a failure of reaction to light, whereas the pupils react promptly during convergence and accommodation. Simple as the phenomenon is, experience is necessary to determine the exact manner of pupillary response. It is well to examine the patient in a darkened room with artificial light (many dark pupils cannot be examined by ordinary daylight). Unless every attempt at convergence is excluded, a false inference may be drawn. Also remember that the phenomenon applies only to eyes with normal or nearly normal vision.

The *immobility of the pupil* to light is generally bilateral. Differences in the reaction of the two pupils may be observed in the earliest stages. At other times it is strongly suspicious of an active luetic process. Tabic pupils are generally miotic ("pin-hole pupils"). Even the smallest pupil may contract still further during convergence, but will not do so under the stimulation of light. The contours of the pupil are apt to be irregular. Oculists lay less stress upon this, but the neurologists are generally agreed with the present writer in holding this sign to be one of importance in tabes, and particularly in those cases in which the luetic virus is still active. Mydriasis occurs in those cases in which there is complete immobility of the pupil. Such mydriasis may be unilateral. Examination of the pupil, it will be seen, tells a powerful tale. In the writer's experience the Argyll-Robertson pupil is the earliest objective, and by far the most constant, symptom of tabes. A close second to it is the loss of the deep reflexes.

The *loss of the patellar tendon reflex* (Westphal's phenomenon) is one of the earliest and most constant symptoms. It is present in fully 95 per cent. of the cases, although in a few instances, particularly in the earlier stages, the knee-jerk may be either present in one or both legs, or may still be elicited by reinforcement (Jendrassik's method). It is not unusual to observe a gradual disappearance of the knee-jerks with the development of other symptoms. Of the method of eliciting the knee-jerk little need be said except that the muscles should be relaxed and that the attention of the patient should be diverted from the purpose of the examination. As a rule, it is sufficient to have the patient sit with the knees crossed, the examiner placing the left hand between the patient's knees and striking the tendon with his right hand, at the same time asking the patient to close his eyes and to pull his own hands with the fingers interlocked. For this examination the patient should be tested when the knees are bare, else mistakes are bound to occur; and if there is any doubt it is wise to put the patient in a recumbent position with the knees semiflexed, or else to lay the patient flat on a couch or bed with the legs hanging down over the edge. At times a diminished knee-jerk can only be discovered by placing one hand on the quadriceps while tapping the tendon with the other. Slight contractions of the muscle, which may, however, not be sufficient to produce an excursion of the leg, may thus be detected. The greater the physician's experience, the more readily he will be able to determine whether a knee-jerk is utterly or only apparently absent. A hypotonic condition of the quadriceps is a more or less constant accompaniment of the loss of the knee-jerk. It is a well-known fact that many persons, particularly physicians and nurses, who know the value of this symptom, inhibit the reflex more or less unconsciously. Physicians and students who test their own reflexes often have great difficulty in eliciting them because of their inability to remove the inhibition.

Of late years we have come to regard the *loss* of the *Achilles tendon reflex*

as being of equal importance with the loss of the knee-jerk. Some writers even claim that it often precedes the loss of the patellar tendon reflex. This is surely not the rule. The loss of the Achilles tendon reflex is of especial value if the reflex is found to be present on one side and absent on the other. This symptom is not elicited as easily as is the knee-jerk. In testing for the Achilles tendon reflex the patient may be examined in the recumbent posture, knee bent, and foot flexed dorsally, so as to put the Achilles tendon slightly on the stretch, and then tapping the tendon sharply with a hammer. If the reflex is present a slight plantar flexion of the foot is the result. Still better, if the patient is able to stand, let him place one knee on a chair and then strike the tendon. Methods of reinforcement may be employed, but they are not often required.

Loss of the deep reflexes of the upper extremities is not as valuable a symptom, for the simple eason that these reflexes are not invariably present in normal individuals. The triceps tendon reflex is the most constant, but in spite of Frenkel's statement that it is invariably present in healthy persons, failure to elicit it cannot be considered a diagnostic aid unless other deep reflexes are also wanting, except possibly in the few (and rare) cases of cervical tabes. The unilateral loss of the triceps or wrist reflex would be significant.

By contrast the superficial reflexes remain unaltered, disappearing in those cases only in which there are very grave disturbances of cutaneous sensation. The plantar, the cremasteric, the gluteal, and the abdominal reflexes can be elicited in tabic patients easily enough. The Babinski phenomenon is never present, as it is absent also in all normal persons.

The next most important and very constant sign is the *Romberg symptom*—the swaying of the body with the eyes closed. This is due chiefly to the loss or alteration of superficial and deep sensibility. To elicit this symptom the patient is asked to stand with eyes closed and with the feet touching one another at the toes. Erb has shown that this necessitates careful balancing of the body, and can be carried out only if the patient appreciates exactly his position in space and his contact with the floor. The sensation of the skin of the feet must be normal and the patient must also have full knowledge of the tension of his muscles and ligaments, or else he cannot maintain his proper static equilibrium. So long as the patient is able to control by vision his whereabouts in space, he may be able to stand perfectly; with the removal of visual control the sensory defects become evident. This test should be repeated a number of times; even normal individuals on a first examination sway considerably when asked to close their eyes, often a purely psychic phenomenon. It is rare to find the Romberg symptom in tabic patients with optic nerve atrophy and blindness. Some years ago the writer reported the case of a blind tabic individual who swayed when he closed his blind eyes. This was evidently a psychic reminiscence of his early tabic period.

In connection with the Romberg symptom brief reference may be made to the *sensory disturbances*. In the vast majority of tabic patients there is, at least in the earlier stages of the disease, a very slight change of tactile sensation. At all events, nothing more than a moderate tactile hypæsthesia exists, as determined by touching with cotton, with a camel's-hair brush, or with a faradic electrode. Such changes as do occur are most common in the soles of the feet, on the inner or outer margin of the feet, and within

the distribution of the ulnar nerve. In his last edition Starr[1] has given excellent illustrations, taken from Bonar, of areas of anæsthesia in cases of locomotor ataxia. They bear all the earmarks of true segmental anæsthesia, but the fewest cases present this symptom in such marked form.

The *pain sense* is more often affected. Hypalgesia and delayed pain sensation are the rule. The latter is a very striking phenomenon, the patient perceiving it as only a mere touch (although pricked with a pin), and after a second or more perceiving (at times) intense pain. Erb has shown that faradic sensibility is much diminished. On the whole, this diminution is proportionate to the loss or diminution of the general pain sense.

The *temperature sense* is apt to be altered. The perception of heat may be diminished and cold felt more acutely than by normal individuals. Exceptions to this rule are not infrequent. Many tabic patients are loath to give up their cold morning bath, thus showing no unusual intolerance toward cold. Allochiria, reference of a sensory stimulus to the opposite side of the body, the writer has observed a number of times; polyæsthesia, the perception of several sensory impacts when touched but once, has also been recorded, but the writer has not seen it except as a species of self-deception. Astereognosis occurs in the advanced stages, also analgesia of the genitals in both sexes; both are, however, rare phenomena.

Sensory disturbances of the trunk of the body are present in a very large number of tabic patients, and have been studied carefully by Hitzig, Dejerine, and others. These hypæsthetic and anæsthetic zones are developed early in the disease, giving rise to various forms of girdle sensation, showing a distinct root and segmental distribution. These forms of disturbed sensation are symmetrically developed, and if they are asymmetrical they correspond to the anatomical distribution of the earliest tabic (root) changes in the dorsal segments. If the lower part of the spinal axis is involved, we are apt to find similar paræsthesia and hypæsthesia around the perineum and genitals.

Disturbances of *deep muscular sensibility* are largely responsible for the Romberg symptom. They may be revealed by other tests. The patient has lost all power to appreciate the exact position of his limbs. If he is in the recumbent position and is asked to elevate his leg to the exact level at which the examiner has placed the other leg (the patient's eyes being closed), he is unable to do this. If sitting in a chair (with eyes closed) and asked to cross his legs, he overestimates the movement needed to clear the knees. He may also fail to appreciate simple passive movements of the joints. All these tests can be carried out more readily with the lower than with the upper extremities.

Tabic patients are supposed to have an impaired sense of fatigue (a very inconstant symptom). It is certain that they have a diminished osseous sense, a diminution or loss of vibratory sensation (as determined by placing a tuning fork on the long bones of the extremities).

A most valuable, reliable, and early symptom of tabes is a distinct *muscular hypotonia*. Erb, Leyden, and Frenkel have studied the phenomenon carefully (Frenkel introduced the term). It may and often does precede the ataxia, and is, therefore, of great diagnostic importance. Not infrequently it helps to establish the differential diagnosis. It consists in an abnormal

[1] *Organic and Functional Nervous Diseases*, third edition, 1909, p. 352 et seq.

diminution of the tension of the muscles and ligaments, so that excessive passive movements of the joints are possible. The normal individual can flex the thigh upon the trunk with the leg extended 50 or 60 degrees; the hypotonia of tabes permits this flexion to 90 degrees or more. Hyperextension of the knee in the recumbent position and genurecurvatum in the erect position are constant phenomena. The same freedom of joint movement (according to Frenkel) is to be observed in the spinal column (what an opportunity for the osteopaths if they knew it!), also in the joints of the hands and fingers. The abdominal and spinal muscles also are in a condition of hypotonia. The condition is independent of the degree of ataxia and of the loss of deep sensibility. It has a considerable influence on the locomotion of the patient.

We pass now to the consideration of the *ataxia* which is so prominent a symptom of the disease that it justifies the term *locomotor ataxia*. We have hinted above that there are some cases that are not ataxic from the beginning to the end of the disease. Ataxia implies no loss of gross muscular power, but an inability properly to coördinate muscular movements. In more than nine-tenths of the cases ataxia appears first in the lower extremities, and very insidiously at that. The patient notices at first that he has become a little unsteady on the legs, that he walks as though he were intoxicated, that his legs "give way" under him. He experiences great difficulty in going up or down stairs; he stumbles in jumping off or on a car; cannot cross the street in safety; has to watch his movements; must look at his feet or else he does not know where they are; if he is to walk at all, he must walk with a broad base and can walk with a stick only; finds much more difficulty in walking at night than during the day, and stumbles or falls if he tries to get out of bed in the dark. By degrees he has developed the typical tabic gait, which even the layman nowadays recognizes, and which the medical man can distinguish easily from the spastic gait (with stiff and more or less closely locked knees).

These earlier stages of tabic ataxia are much more manifest on examination. If the patient is asked to stand on one foot, he topples or falls; if asked to turn about on his heels quickly, he sways badly; if asked to walk a straight line, placing one foot in front of the other, he is utterly incapable of doing it; in all movements requiring coördination of the muscles he goes wide of the mark. In the recumbent posture he cannot elevate the leg when extended without performing zigzag movements; he has difficulty in touching one knee with the opposite heel, and with the eyes closed all these defects in movement are much exaggerated, particularly if there is considerable impairment of deep muscular sensibility.

Many patients learn to accommodate themselves to a certain degree of ataxia, and for years (particularly under the influence of modern therapeutic methods) get about with considerable ease. As the disease progresses the increasing ataxia renders the limbs useless and the patient becomes chair or bedridden. In extreme cases the patient, even if supported, has lost control over his legs so completely that, on attempting to walk, the legs are thrown about in the wildest, often grotesque, fashion. Ataxic movements of the upper extremities are, on the whole, very rare, and there are few who have lost the ability to write or to feed themselves. In that rare form known as *tabes superior* the ataxic movements of the hands are more pronounced. The ordinary movements of daily routine (tying a cravat, cutting with

scissors or a knife, piano playing, writing, etc.) become impossible. On examination (putting the finger to the nose with the eyes closed, touching the examiner's finger with eyes open), the defect of movement is brought out distinctly. Ataxic movements of the facial muscles, of the lips, of the tongue, of the laryngeal muscles, are rare; if present, they can be recognized by special disturbances.

In the preceding pages we have described the most typical and most constant symptoms, but there are a number of others to be enumerated, the student remembering that their absence need not militate against the diagnosis of tabes.

*Vesical Disturbances.*—Important and often most troublesome to the physician are the vesical disturbances. These are so common, even in the earliest period of the disease, that they not infrequently constitute the first symptoms that arouse a suspicion of serious organic nervous disease. In the genito-urinary service of a large hospital the neurologist is often consulted for the purpose of determining whether or not vesical symptoms, which appear to be independent of disease of the bladder itself, are the expression of a latent (?) tabes. At first dysuria (neuralgic pain in the region of the bladder), hyperæsthesia of the neck of the bladder, dribbling, frequent micturition or moderate retention, are the usual forms of disturbance. As the disease progresses the amount of residual urine increases; catheterization may be necessary, and whether this be done by the patient himself or a nurse (we will not add, by the physician), cystitis is only too apt to be superadded. This increases the discomfort of the patient, and if it leads to pyelonephritis, as it may do readily enough, there is distinct danger to life. It is one of the intercurrent disorders that may cut short the duration of the disease, which might otherwise extend over twenty or thirty years.

*Rectal insufficiency* is rare. In some patients with hypæsthesia of the anal region inconvenience is experienced after taking purgatives, but otherwise the rectal sphincter continues to perform its function satisfactorily. The patient is more depressed over the loss of sexual desire and of sexual power, the latter often preceding the former. Female tabic patients are reported to experience a loss of sexual desire, but the point is not easily established. Among male patients, the loss of virility is one of the earliest symptoms that cannot be relieved by the modern remedies resorted to in cases of non-organic disturbance of the sexual function.

*Cranial nerve* disease is an important factor in the series of tabic symptoms. Oculomotor palsies, partial or complete, abducens paralysis, rarely trochlearis palsy, each and all are known to occur. The palsies of the earlier period—ptosis and strabismus—are particularly significant, because they occur even more frequently as the symptoms of cerebral lues. The differential diagnosis between tabes dorsalis and cerebral syphilis is established by the association of other characteristic symptoms of locomotor ataxia. In the more advanced stages of this disease we may find paralysis of associated movements of one or both eyes, pointing to involvement of the various nuclei. Complete ophthalmoplegia externa and interna are observed in rare instances.

The *facial nerve* is so rarely involved that mention of this fact would not be necessary if facial nerve symptoms and those due to involvement of the motor branch of the fifth nerve did not occasionally occur in rare forms of tabes complicated by bulbar disease. The sensory branch of the fifth

nerve is not as exempt as is the motor division of this nerve. It has been my peculiar experience to see a number of patients (three are now under observation) in whom a dissociation of sensation, at first unilateral and later on bilateral, within the distribution of the trigeminal nerve, has been the earliest symptom of tabes. In two of these patients this fifth nerve dissociation preceded all other symptoms, even the pupillary phenomena and the loss of knee-jerks, by many years. During this period the diagnosis of the disease was much in doubt. The suspicion of an unusual form of syringomyelia was entertained until the appearance of the cardinal symptoms of tabes left no doubt.

Palsies due to involvement of the tenth and eleventh nerves also occur, but rarely, if at all. The most frequent form among these is characterized by dyspnœa and inspiratory stridor. One of Erb's patients had to have tracheotomy done, and wore a cannula for six years. Hoarseness and aphonia, due to paralysis of the recurrent laryngeal nerve, have also been recorded. Atrophy or paralysis of the trapezius and sternocleidomastoid muscles, due to involvement of the eleventh nerve, is excessivly rare. A little more frequent is the atrophy of the tongue due to disease of the hypoglossal nerve, but this condition is associated too, as a rule, with other symptoms of bulbar paralysis. It may be stated here that progressive cranial nerve palsies, if not a typical progressive bulbar paralysis, may occur in association with the other symptoms of tabes. The involvement of one or more, if not of all of the nuclei of the cranial motor nerves, may cause an endless variety of symptoms. The olfactory and auditory nerves escape, as a rule, although cases have been described with loss of the sense of smell, with delusions of smell, and still others in which there has been continuous tinnitus with vertigo. Ménière's symptom group has also been recorded.

But by far the most important cranial nerve symptom is the *optic nerve atrophy*. This has been referred to above, where it was said that it gives rise to a distinct group of cases known as amaurotic tabes.

Among the orthodox symptoms, although often wanting, are the *visceral crises* and the *trophic disorders*. We owe our knowledge of the visceral crises largely to the accounts given by Charcot. In his inimitable way he described gastric crises, and nowadays every neurologist recognizes the condition, which is quite as distressing as any other of the many troubles that befall the tabic patient. But they still seem to be a *terra incognita* for the general practitioner and for the specialist in gastric and intestinal disorders. These gastric crises consist of severe neuralgic pains in the epigastrium or in some other part of the abdomen, appearing suddenly, lasting for hours or days and then disappearing quite as suddenly. The intense pain is accompanied by uncontrollable vomiting. It will be readily understood why this condition leads to errors in diagnosis. Many a troublesome gastric condition that has occasioned an infinite variety of medicinal and even surgical measures has in reality represented the gastric pains of tabes. Only a short time ago a patient was seen who had passed through the hands of a number of practitioners and specialists, and was suspected of all sorts of gastro-intestinal disturbances, when the loss of the pupillary reflex to light and the absence of the knee-jerk would have given the examining physicians a hint as to the true nature of the disorder.

Entirely similar to the gastric are the intestinal crises. One patient with

trigeminal dissociation also suffered from frequent inexplicable diarrhœa with intense abdominal pain, until the proper interpretation of both was made manifest by the appearance of a series of tabic symptoms.

Among the curiosities of tabetic symptomatology we may class the vesical, the renal, the testicular, and even the clitoris crises; the exact symptom of each may be inferred from the known function of these organs. Special mention must be made of the laryngeal crises which Téréol described in 1868. These attacks begin with a tingling sensation in the throat, a feeling of constriction leading to a condition of temporary asphyxia with stridor, cyanosis, and occasionally to a convulsive seizure. The spasm lasts but a few minutes, when the patient is out of imminent danger. Oppenheim has described pharyngeal crises with pain in the pharynx and frequent movements of swallowing.

Cardiac crises are referred to because the writer has seen them in several cases, characterized by intense pains, like those of angina pectoris; but as these patients were at an age when angina pectoris could not be excluded, their exact value as a symptom of tabes is a matter of dispute.

Lastly, a few words about *trophic disorders*. The best known among them is the mal-perforant or perforating ulcer, which is generally situated below the big toe at the level of the metatarsophalangeal joint, but it may occur in other parts of the sole of the foot. It is a small, circular ulcer, extending through the soft parts to the bone. It does not yield readily to treatment. Erb claims that it is cured more often by mercurial salve than by anything else, and for this reason he believes it to be specific in character. Herpes zoster has been observed often enough in cases of tabes to make it worthy of special mention. Its known dependence upon changes in the nerve roots and in the spinal ganglia explains its occurrence in locomotor ataxia. Tabic patients are prone to repeated attacks, whereas others rarely have more than one attack, at least at an interval of many years.

Charcot and his pupils, who contributed so much to the full understanding of the symptomatology of tabes, were the first to dilate upon the *arthropathies*. The knee-joint is the one most frequently affected; next in frequency come the hip- and shoulder-joint , but marked and similar trophic changes occur in the elbow- and finger-joints. The chief characteristic is an enormous swelling of the joint with little or no increase of synovial fluid. The joint is, however, much enlarged, remains normal in color, and absolutely painless. The arthropathies may appear early, and if they become chronic lead to grotesque deformities, to luxations and subluxations, to the formation of osteophytes; the bones become unusually porous and the slightest accident may cause fracture in and around the joint. Painless fracture of the long bones on slight exertion or slight injury is tolerably frequent. In the ankle-joints and in the smaller joints of the foot similar structural changes occur that Charcot described as the tabic foot—a grossly misshapen foot. A limb or joint so affected becomes thoroughly useless, and yet it is astonishing how long patients with these arthropathies manage to get about. Not unlike these arthropathies are the alterations of the alveolar processes leading to a loss of teeth. All the teeth may drop out in this way. It is, however, so rare an occurrence that in hundreds of cases of tabes the writer has not seen a single instance of this condition.

Much has been made of the occurrence of *cardiac disease* in tabes. When it does occur it is an accidental complication, and it is due to the preceding

syphilitic infection. In the course of some investigations of cardiac conditions, by Collins and the writer,[1] with the Wassermann method, the specific basis has been well established, as was done before them by Citron and others.

Lastly, we must take note of the *psychic* manifestations of tabes. Conditions of depression, of neurasthenia, of morbid irritability, are not unusual or difficult to account for in view of the distressing condition. Sufferers from locomotor ataxia may develop paranoia or paranoid states. Some of them, as a recent patient has done, may pass through states of acute maniacal excitement or through states of transitory dementia with and without aphasia, probably an expression of brain syphilis. But all these psychic manifestations are relatively less frequent in tabes than the occurrence of symptoms characteristic of general paresis. Both diseases have a common etiology—syphilis. It is not astonishing, therefore, that some patients should develop both conditions. As a matter of fact, tabic patients do develop often enough the symptoms of general paresis (delusions of grandeur, loss of memory, paretic speech, etc.). Still more frequently do patients with general paresis develop a series of tabic symptoms (lightning pains, ataxia, loss of knee-jerks, etc.). Some symptoms, such as the pupillary reflex, immobility, loss of vesical control and of sexual power, both diseases have in common. In spite of this community of symptoms, we need not go to the length that Möbius, Raymond, Mott, and others have, in declaring that tabes and general paresis are due to one and the same process, and that both are metasyphilitic diseases, and that it is only the difference in localization that determines whether the morbid process gives rise to the clinical picture of tabes or to that of general paresis. That there is an intimate etiological relation between the two diseases cannot be denied, but the simultaneous occurrence of the two diseases is, after all, relatively rare. Tabes followed by general paresis is distinctly rarer than general paresis followed by tabes. The association is rare enough to caution the physician not to make the diagnosis of general paresis whenever a tabic patient presents slight psychic abnormalities.

Before concluding this endless record of symptoms, it may be well to recall the fact that the disease may last fifteen to twenty or even thirty years, unless life is terminated earlier by some intercurrent disease. Of all the symptoms, blindness, lancinating pains, cystitis, and the visceral crises are the most distressing. If the disease drags on, the ataxic limbs may become paralyzed; in this helpless condition the occurrence of bedsores, the incontinence of urine and fæces, the increasing marasmus, make a truly pitiable picture. Fortunately patients are usually relieved of their misery by some intercurrent disease before these extreme conditions appear.

Bearing in mind the great multiplicity of symptoms, one can readily see that there may be a vast difference in the grouping of symptoms of various cases of tabes, and also of the general course of the disease in one or the other individual. In some instances the course is unusually slow, so that the disease appears to be stationary for ten to fifteen or twenty years; in others the disease may come on insidiously and suddenly take a turn for the worse, developing within the course of a few months a number of the most distressing symptoms. As a rule, the prodromal stage covers a period of months or

[1] *Amer. Jour. Med. Sci.*, 1909, cxxxviii, 344.

years. By slow stages a slight difficulty in walking progresses into a marked ataxia. Only now and then, as in a recent case, the ataxia is developed with startling rapidity. A gentleman, aged forty years, a lawyer, who had attended to his work without any difficulty and been able to plead in court and stand on his feet all day long if necessary, suddenly found himself beginning to sway, and within forty-eight hours was markedly ataxic. Of course, the pupillary symptoms and the loss of the deep reflexes had preceded the onset of this symptom, but of these latter the patient was not aware.

It has been customary to designate various types of the disease according to the predominance of some one set of symptoms or according to the localization of the disease as indicated by the chief symptoms. The terms explain themselves; thus, various writers choose to speak of tabes dolorosa, of tabes paræsthetica, of tabes atactica, of tabes paralytica, and tabes amaurotica; other designations are tabes visceralis, and tabes superior (meaning cases in which the upper extremities are chiefly involved). Tabes juvenilis applies to those rare forms in which all the symptoms appear early in life.

Erb has made special studies of the cases to which he applies the term *tabes incompleta*. These are practically identical with the formes frustes of the French writers. In this special group of cases only a few of the cardinal and characteristic symptoms are present. While this may be the rule in the earlier stages of many cases, the term should, of course, only be applied to those in which this paucity of symptoms has been noted for a number of years. There are individuals known to every neurologist, who, for years, have had the Argyll-Robertson pupil, others who have had visceral crises, still others who have had slight vesical disturbances, and every one of them suggestive of tabes, and yet in the absence of corroborative symptoms the physician may well hesitate to make a final diagnosis of tabes. In all such forms the presence of the Wassermann reaction and the cytodiagnostic examination of the cerebrospinal fluid will help to put the diagnosis of tabes on a firm basis.

**Diagnosis.**—From the preceding enumeration of the symptoms it is evident that the fully developed forms of the disease can hardly be mistaken for any other affection of the nervous system. If two or three of the cardinal symptoms are present the diagnosis can be made without any hesitation, but it is well also to bear in mind that the disease may begin with one or the other of the more unusual symptoms, with visceral crises, with an arthropathy, with vesical and rectal disturbance. These rather unusual symptoms may precede the cardinal symptoms for a number of months or years, and in such cases the exact nature of the disorder cannot be diagnosed until one or more of the cardinal symptoms have appeared. The early recognition is so important that the physician may be pardoned in venturing the diagnosis of tabes on mere suspicion and of subjecting the patient to such treatment as may possibly help to inhibit the progress of the disease. In the recognition of the earlier stages, as has been intimated before, evidence furnished by the Wassermann reaction and by examination of the cerebrospinal fluid will be of the greatest importance.

In spite of all precautions, mistakes will happen, and the question will often arise whether the condition is one of tabes dorsalis or one of those now to be mentioned. The other chronic systemic affections of the spinal cord, such as *spastic spinal paralysis, amyotrophic lateral sclerosis*, and the *spinal amyotrophies,* need hardly be considered. In the latter the pupillary

phenomenon are wanting, the deep reflexes may not be absent, the knee reflex may be increased, all sensory disturbances are wanting, and the entire clinical picture is wholly different from that of tabes. Some of the symptoms of tabes dorsalis may occur now and then in *tumors* of the cord, invading first the posterior half of the cord, and in *multiple sclerosis*, if the sclerotic areas happen to involve the posterior columns and the posterior gray matter rather than the pyramidal tracts, in which they generally occur. But in such cases in which there is a doubt as to whether it is one of tabes dorsalis or disseminated sclerosis, the occurrence of nystagmus, of altered speech, and the ataxic tremor will help to indicate the true nature of the disease. Much more difficulty will be encountered in differentiating between a *syphilitic meningomyelitis* invading the posterior half of the cord and genuine tabes dorsalis. Many years ago the writer dilated upon the subject, to show that some cases which were considered to be genuine instances of tabes dorsalis were actually cases of specific infiltration of the meninges, starting in the meninges but invading the cord. The presence of such symptoms which point strongly to the true syphilitic nature of the process, such as complete immobility of the pupils, paralysis associated with the ataxia at an early stage of the disease, marked remissions and exacerbations of the symptoms, would help to indicate an active luetic process rather than a typical posterior spinal sclerosis. *Syringomyelia* would hardly come into question. In rare instances this disease, beginning in and affecting chiefly the lumbar portion of the cord, may, for a time, give rise to symptoms suggestive of tabes dorsalis, but the dissociation of sensation and the absence of typical tabic pupillary symptoms will help to establish the differential diagnosis.

Much more difficulty will be experienced in differentiating between tabes dorsalis and the *combined scleroses* of the spinal cord. In this condition we are apt to have an ataxic paraplegia, and at one or the other stage of the disease the ataxia may preponderate to such an extent that the patient may present so few of the spastic and paralytic symptoms that there will be little reason to suspect anything else than tabes dorsalis. If the ataxic symptoms are associated with increase of reflexes, and if the pupillary phenomena are not typical of tabes dorsalis, the diagnosis of ataxic paraplegia or of combined sclerosis of the cord may safely be made. *Friedreich's ataxia*, which was formerly thought to be intimately related to locomotor ataxia, is recognized by its occurrence early in life, by its family or hereditary disposition, by the absence of the characteristic pains and sensory disturbances of tabes, by the absence of vesical and rectal disturbances, and by the existence of normal pupillary reflexes. The ataxia of Friedreich's disease is also coarser and of a more awkward type, and it involves the upper extremities much more frequently than is the case with true tabes. The "heredo-ataxie cerebelleuse" of Marie is recognized still more readily by the exaggeration instead of the absence of the deep reflexes; by the presence of club foot, and a number of other signs which do not occur in genuine tabes.

From *cerebellar tumors* tabes dorsalis will be differentiated quite easily, for in the former the form of the ataxia, the vertigo, vomiting, double optic neuritis and the absence of pupillary immobility, of the Romberg symptom, and of the lancinating pains, and the much more rapid development of all the symptoms, will lead to the correct diagnosis. The greatest difficulty of all will be experienced on attempting to differentiate between tabes dorsalis and *alcoholic multiple neuritis*, or, as it was formerly called, alcoholic

pseudo-tabes. The resemblance to tabes lies in the ataxic movements of the lower extremities and in the absence of the deep reflexes, also in the occurrence every now and then of marked sensory disturbances. On closer examination the difference becomes much more marked, for in alcoholic pseudo-tabes the pupillary phenomena are not so constant, although there is often a sluggish reaction to light. There is almost invariably a considerable degree of paralysis associated with the ataxia, often also some muscular wasting. Lancinating pains and girdle sensation are not the rule in multiple neuritis due to alcoholism, and the entire development of the symptoms is more rapid than is the rule in tabes dorsalis. Alcoholic pseudo-tabes leads more commonly to a paralytic condition of the lower extremities, more often also than is the case in tabes to an involvement of the upper extremities, while the physical symptoms are less pronounced, and the inquiry into the habits of the patients will give the physician a hint as to the origin. In not a few instances the writer has been convinced that a moderate alcoholic excess was sufficient to produce a pseudo-tabes alcoholica which would not have been developed if the patient had not previously been infected with syphilis. It is the old story of two poisons being more powerful than one; and after all that has been said and written on this subject it is safe to say that if one is acquainted with the symptoms of tabes dorsalis, there is very little difficulty in differentiating between it and other diseases of the central nervous system.

**Prognosis.**—This is either favorable or unfavorable, not so much according to the gravity of the symptoms, as it is proportionate to the general optimism or pessimism of the physician. It is true that it is practically an incurable disease. The pupillary phenomena and the loss of the deep reflexes are rarely if ever recovered from, and in this sense, after the symptoms of tabes have appeared, the patient is afflicted with the disease for all time to come. On the other hand, the disease is often very slowly progressive; it may at times be said to come to a standstill, and many a patient is able to attend to his routine duties and to enjoy a tolerably comfortable life for many years. Every neurologist will concede that there is much to be done for the tabic patient, and the latter may be given the assurance that there are many ways of making him fairly comfortable.

Some years ago the neurologists of Paris, especially Brissaud, Marie, Raymond, and Babinski, claimed that tabes dorsalis seemed to have taken on a milder form than in former years; they were inclined to attribute this to the more thorough specific treatment which the patients received. There is some doubt as to the truth of this, but the fact remains that we do not see as many of the distressing forms of extreme ataxia as in former years, and tabic amaurosis does not seem to be as common as it was some twenty-odd years ago. On the whole, cases of tabes in which lancinating pains are the earliest and severest of the symptoms, are not the most unfavorable. While these lancinating pains predominate, the other symptoms are slow to appear. The cases in which a marked ataxia appears at a very early period represent a group of cases which are apt to advance rapidly and to lead to a large number of complications, but even these can be favorably influenced by various forms of treatment, The writer has been particularly struck by the fact that the amaurotic type of tabes, while it is perhaps the most unfortunate one, is often more slowly progressive and many of these patients would have been tolerably comfortable for fifteen or twenty years

if it had not been for the unfortunate loss of vision. The prognosis is most unfavorable in those cases in which the early occurrence of vesical symptoms has led to a cystitis or a pyelonephritis, in which other syphilitic phenomena, such as convulsions and apoplexy, have occurred as an early complication. The combination of tabes and general paresis is bound to lead, sooner or later, to a fatal termination. But it is well to insist once more that not every psychic disturbance occurring in a tabic patient is necessarily a form of general paresis.

**Treatment.**—The question of prophylaxis looms up prominently. By the time the diagnosis is safely established the spinal cord changes are so considerable that one cannot expect to do much more than to arrest the disease. No one has yet been able to claim that he can in any way influence by treatment sclerotic tissue in the spinal cord after it has been once fully developed. As regards prophylactic measures, all that can be expected is possibly to lessen the syphilitic contagion. Such a Utopian state of affairs is not within our reach, but we can at least demand of everyone who has contracted syphilis that he subject himself to prompt and sufficient treatment. While it would be difficult to prove by statistics that prompt and proper antisyphilitic treatment following upon the initial lesion prevents the development of syphilitic and parasyphilitic diseases of the central nervous system, it is at least probable that these serious manifestations could be to a degree lessened if energetic measures were resorted to in every instance at the outset of the disease. There is at least no reason why the central nervous system should not be influenced as the osseous system is, and surely we do not see at the present time as much bone syphilis as half a century ago. Whatever one's views may be regarding the possibility of influencing the development of tertiary symptoms by early treatment, it is certain that when the first signs of any syphilitic or parasyphilitic affection of the central nervous system appear, energetic measures should be resorted to. It is safe, therefore, to establish the general principle that in every case of tabes dorsalis in which thorough antisyphilitic treatment has not been administered, such treatment should be given before anything else is attempted. At the present date, and in view of recent experiences, this statement may be modified to mean that if a positive Wassermann reaction has been obtained, the patient should receive prompt antisyphilitic treatment. If the reaction is negative, the same treatment may be given if there are clinical signs pointing to an active luetic process.

By appropriate antisyphilitic treatment the writer means mercurial injections,[1] either of some insoluble salt, say 1 grain of mercury salicylate, once a week, repeated during ten or twelve weeks, or else sublimate injections of $\frac{1}{6}$ of a grain (0.01 gram), repeated every other day until ten, twelve, fifteen, or twenty have been given, according to the severity of the case. A tablet (suggested by the late Dr. Jones) containing $\frac{1}{6}$ of a grain (0.01 gram) of bichloride of mercury, $\frac{1}{4}$ of a grain (0.016 gram) of sodium chloride, and $\frac{1}{16}$ of a grain (0.004 gram) of muriate of cocaine, has been found particularly useful. In spite of the well-known germicidal properties of sublimate, it has been the writer's custom to dissolve this tablet in a sufficient quantity of water and to boil it in a test-tube before making a deep intramuscular

[1] The writer is not opposed to mercurial inunctions if they are properly administered.

injection. In this way all possibility of infection or abscess is avoided, and in more than a thousand such injections not a single abscess has occurred at the site of injection. It has also been the writer's practice never to give iodides and mercurials at the same time, in order to avoid the formation of insoluble salts. The better practice is to give a course of subiimate injections, then to wait a week or two, and then to give a thorough course of iodides. The writer has every reason to be thoroughly satisfied with the use of the sodium iodide in saturated solution, beginning with ten drops three times a day, well diluted, and increasing the daily dose by one drop until thirty to forty or fifty drops three times a day are reached. Beyond this it is hardly necessary to go, and the writer does not feel warranted any longer in supporting the late Dr. Seguin's claim that satisfactory results in many cases cannot be obtained unless very large doses, such as 150 grains three times a day, are administered. It is questionable whether such very large doses are properly absorbed. Incidentally it may be well to remark that by adopting this course one rarely gets salivation and that the patient's digestion is upset very much less than if the mercurials and iodides are administered at one and the same time. In the treatment of syphilis of the nervous system the writer has abandoned entirely the so-called mixed treatment. Those who prefer the use of a 10 or 25 per cent. solution of iodopin may safely substitute it for the iodide of sodium.

The question has often been raised whether antisyphilitic treatment is of any distinct benefit in tabes dorsalis. One cannot claim that the disease has ever been cured. The pupillary reflexes do not return to normal, nor is an absent knee-jerk restored, but there is no doubt that a condition which seemed to be rapidly progressive has been arrested, that patients who have been tormented by severe shooting pains have suffered less and less as the treatment was continued, that vesical and rectal insufficiency has been lessened, and in not a few instances impending mental trouble has been averted. The writer has no hesitation, therefore, in favoring the energetic use of mercurial salts in the initial treatment of tabes dorsalis, and of urging that mercurials rather than iodides be given the fairest possible test.

In addition to this specific treatment much else can and should be done for the patient. Above all, he should be induced to lead a life free of all excitement and free of all excesses. Alcohol and tobacco and every other form of indulgence should be restricted to very narrow limits. Physical exercise should be maintained within sensible limits so long as it is at all possible for the tabic patient to get about unaided. In view of Edinger's theories, fatigue should be avoided, but the ordinary tabic patient must be encouraged to keep his muscles in good condition and to use them in the best possible way so long as it is convenient for him to do so. The tabic patient who lies down, as it were, under his disease, seems to succumb much more rapidly than he who endeavors to make the best of his disease. Among general therapeutic measures, hydrotherapeutic procedures have met with great favor. While hot baths should be avoided, the tepid full bath with slightly cooler ablutions can be safely recommended. Cold bathing, particularly sea bathing, is not tolerated well, although among our better classes there are many tabic patients who feel thoroughly comfortable while continuing the cold water habit, and so long as they do not complain of increasing neuralgic pains it need not be discouraged. This is a point which may well be determined according to the needs of the indi-

vidual patient. To those who can indulge in the luxury of a course of treatment at Nauheim this line of treatment may be recommended, although it is undoubted that the excellent results obtained there are due not only to the influence of baths, but to the combination of hydriatic procedures with the other physical and mechanical measures. It would be eminently desirable to create similar opportunities for appropriate treatment in this country.

Galvanism of the spine and general faradization are still employed. Many a patient feels comforted if not distinctly improved by such treatment, and the improvement that is attributed to mild faradization can be explained by the effect which it has upon the peripheral sensory neurones. General massage, particularly in connection with hydriatic procedures and with electrical treatment, is of distinct benefit. But there is no doubt that more has been accomplished in the successful treatment of tabic patients during the last decade by systematic exercise than in any other way. To Frenkel,[1] of Heiden, belongs the credit of having developed a system of exercises so designed as to reëducate the ataxic muscles. It has been clearly shown that with the exception, perhaps, of the most advanced cases, patients who are so ataxic as not to be able to stand or walk unassisted can regain the use of their muscles and can be improved to such an extent that they can follow their ordinary occupations. Those who are specially interested in the details of these exercises can find full particulars in the monographs of Frenkel and Goldscheider,[2] also in the writings of Gräupner,[3] and in a more recent one of O. Foerster.[4]

The treatment of locomotor ataxia has baffled physicians for so many years that it is natural that all sorts of cures are reported from time to time; that such cures should be lauded to the skies for a limited period, to be abandoned forever after. Thus, about ten years ago the suspension treatment, as suggested by Motschutkowsky, was in vogue. Every one tried it; every clinic installed the apparatus; every patient was subjected to it. At the present day it has been entirely eliminated from practice and even from theoretical discussions. It was tried and found wanting. A similar fate has befallen the practice of the bloody and bloodless stretching of the sciatic and crural nerves, as practised some five and twenty years ago.

In addition to the general measures, purely symptomatic treatment must often be resorted to. So far as the pains are concerned, nitrate of silver has been endorsed by many. Its good effects are questionable, although it is one of those drugs which every neurologist will employ in the course of this protracted disease. If the pains cannot be diminished by the use of pyramidon or aspirin, in combination with codeine, nothing will influence them. A distinct caution should be given regarding the use of opiates, particularly the use of hypodermic injections of morphine. Many a tabic patient has also become a marked morphine habitué and the morphine habit has thus made conditions ten times worse than they would otherwise have been.

The treatment of the gastric crises also calls for good judgment on the

[1] *Die Behandlung der Tabische Ataxie*, etc., Leipzig, 1900.
[2] *Anleitung zur Hebungs Behandlung*, etc., second edition, Leipzig, 1904.
[3] *Allg. Med. Centralztg.*, 1898, No. 38.
[4] *Physiologie u. Pathologie der Coördination*, Jena, 1902.

part of the physician. It is best to give antineuralgic remedies, but during the period of the crises the stomach and bowels should have complete rest, hot applications should be applied over the abdomen, the patient kept in a recumbent posture, and, if drugs must be given, a combination of cerium oxalate and codeine may be used. In not a few cases special attention has to be directed to the condition of the bladder. Cystitis, with all its attendant dangers, is a common occurrence. This condition is to be treated according to well-received surgical principles, and particularly in those cases with considerable residual urine, the bladder should be emptied at regular intervals and washed with mild antiseptic solutions. Permanganate of potash and boric acid are the ones commonly employed. There is danger in allowing the patient himself or even a nurse, unless he be specially trained, to carry out these procedures. It means much for the comfort of the average tabic patient if his cystitis is held in check. In spite of all precautions there is greater danger to the patient suffering from locomotor ataxia from his cystitis than from almost any other condition. The constant use, for a prolonged period of time, of urotropin need only be mentioned.

If one were to enumerate every possible form of symptomatic treatment, it would be necessary to run the gamut of almost every drug in the Pharmacopœia. In passing, however, a word should be said regarding the attempt to influence a rapidly progressive optic nerve atrophy by specific treatment. From a large experience it is difficult to claim that amaurosis has been completely averted in any one instance. At the same time the claim can be put forth that the development of total blindness seems to have been postponed for a considerable period of time by the discrete use of antisyphilitic measures. It is, however, in these very cases that one should be most careful not to push the measures to the extreme, although the writer is not able to endorse the opinions of some that the progress of optic nerve atrophy is made more rapid by the use of these measures. It is wise, however, to have the vision tested carefully during the course of the treatment in any cases of amaurotic tabes, and if the vision is growing rapidly worse to desist from this therapeutic plan.

Quite as important as, if not more important than, drug treatment, is the psychic treatment of tabes dorsalis. No patients are more liable to general despondency, to marked hypochondriasis, than are those suffering from locomotor ataxia. They should, therefore, be given every possible consideration, should be encouraged in every way, and should be told that in spite of having an incurable disease, the symptoms may remain stationary for a long period of time, and that life can be made tolerably comfortable in spite of the disease.

## GENERAL PARESIS.

General paresis of the insane, or dementia paralytica, represents one of the severest forms of nervous disease, and is characterized by a long series of physical and mental symptoms. From the insidious beginnings the disease progresses, during a period of several years, to a condition of complete dementia, affecting both men and women in the prime of life. The majority of cases occur in persons between the ages of thirty and fifty, although

its occurrence is not unknown in children (juvenile general paresis) and also in persons entering upon the senile period. In the former there is generally a history of hereditary lues, while in the latter, syphilitic infection has been acquired late in life.

**Etiology.**—General paresis is a disease of civilization, although civilization alone can hardly be held responsible. Krafft-Ebing's famous dictum that it is due to "civilization and syphilization" has hit the nail on the head. The Chinese, Turks, and the Irish were supposed to be tolerably free of the disease. The same was at one time supposed to be true of the negro race, but in this country we have had ample opportunity to discover that neither the Irish nor the negroes enjoy immunity. It is well established that in only 40 or 50 per cent. of the patients is there any family history of mental or nervous disturbances, of dipsomania, or of any other serious form of degenerative nervous disturbance.

The disease affects men very much more frequently than women, the proportion, according to some, being nearly 7 to 1, according to others, 2 to 1. Among 70 cases in my private practice, 5 were women (7.1 per cent.). In all large cities general paresis, and particularly general paresis in the female, is far more frequent than in the country. Among men the disease is supposed to be commoner among the well-to-do and among those who have indulged in all sorts of excesses. The clergy are known to be relatively free from the disease, while the German statistics show that army officers yield an unusually large percentage. Among women the lower classes are distinctly more frequently the victims than the better classes. All these facts point to the well-established conviction that the chief etiological factor of general paresis is a preceding syphilitic infection. For many years we have had to be content with statistical proof, and those of us who have had a large experience, particularly in private practice, have regarded it as almost axiomatic that the man who presents symptoms of general paresis must also present some symptoms of constitutional syphilis. In the writer's series in private practice there have not been more than two or three in whom there was any doubt as to the preceding specific infection, Of late years, however, we have fortunately been made independent of statistical proof. The examination of the cerebrospinal fluid obtained has shown a pleocytosis which occurs only in syphilis or in a disease of syphilitic origin. Moreover, he introduction of the Wassermann reaction has shown an unusually high percentage of positive reactions in the blood of general paretics. Some of the figures have been above 85 and none much below 70 per cent. In 31 cases of the writer's the reaction was positive in only 67.7 per cent. It is safe to say that the syphilitic origin of general paresis cannot be denied. At all events, this infection forms the basis upon which the disease develops. There may be special exciting causes, but without syphilis general paresis does not occur. The frequent occurrence of general paresis in husband and wife teaches the same lesson. The writer has seen a number of such, and only very recently was asked to see a widow, who, more than ten years after the death of her husband who died of general paresis, exhibited the first symptoms of this disease.

A period varying from five to twenty years or more may elapse between the initial infection and the first development of the disease. It is doubtful whether thorough or negligent treatment shortly after the initial infection has much influence upon the development of general paresis, but the mere

fact that this is still a matter of doubt makes it incumbent upon every physician not only to treat the initial symptoms of syphilis, but to subject every person who has been infected with syphilis to repeated courses of anti-syphilitic treatment, whether such person presents symptoms or not. With the Wassermann reaction as a guide, there is no sufficient reason why any patient should be allowed to take his chances. The least the physician can do is to demand that the patient have an examination of the blood made at least twice a year, and if there be any positive reaction, then surely a course of treatment is necessary, whether that patient present any specific symptoms or not.

Given a syphilitic infection, any excesses, particularly alcoholic and venereal, may play the part of further exciting causes. Overwork and worry have been assigned as additional etiological factors. Worry and emotional excitement play an important part in the development of many psychic conditions, and may have a bearing upon the development of general paresis, but overwork alone, and more particularly overwork without excesses, is rarely followed by any serious results. Excesses are harmful; work, and even overwork, as a rule, beneficial. Of the many additional exciting causes which have been enumerated, the author feels that trauma of the skull is the one which deserves greatest consideration. Among the laboring classes several cases have been seen, in which the first symptoms of general paresis appeared within a few weeks following serious head injury, but in not one of these cases could the additional factor of a syphilitic infection be excluded. Ziehen is of the opinion that the syphilitic virus is apt to exhibit itself in the brain after its resistance to disease has been diminished by severe trauma. Possibly the shoe fits the other foot, and it would be safer to say that syphilis reduces the resistance of the brain and makes the effect of an injury more evident than it otherwise would have been. There seems little doubt that alcoholism is an etiological factor of some importance. When it is able to exhibit its effects upon the system previously weakened by constitutional syphilis, the damage it does is doubly great. Allowing for the close resemblance between alcoholic pseudo-paresis and dementia paralytica, there are some undoubted cases of genuine paresis in which alcoholism has been a distinct and exciting cause.

**Pathology.**—The morbid changes prove it to be a disease not merely of the brain, but of the spinal cord as well. The popular designation of the disease as "softening of the brain" is wrong in every particular. Macroscopically we may note thickening of the skull, with loss of the diploe, adhesions between the dura and the skull, a pachymeningitis, a condition of external and internal hydrocephalus, a cloudy and thickened appearance of the pia, particularly over the frontal portion of the brain; adhesions between the pia and cortex, a granular ependymitis, and, above all, a very marked atrophy of the cortex. The fissures appear unusually deep and the gyri small. Arteriosclerotic changes are noticeable, more especially in the basilar arteries. The weight of the brain is considerably reduced, atrophy of the frontal and parietal lobes being responsible for this reduction, whereas the temporal and occipital portions of the brain are more nearly normal. On section of the cortex the gray matter appears very considerably reduced in volume.

There has been considerable discussion as to whether or not one could recognize general paresis on the postmortem table. From macroscopic

appearances alone this would hardly be safe, although we could make a fortunate guess in perhaps 50 per cent. of the cases. The researches of Cramer, Binswanger, and Nissl and Alzheimer, have thrown a flood of light on the histological changes. The pia is invariably diseased, with distinct infiltration with plasma cells and lymphocytes. All the bloodvessels show either proliferating or degenerative processes. The cortical substance itself contains diseased bloodvessels. There is a proliferation of the endothelium, the elastic tissue is increased, the adventitia proliferates, and the adventitial lymph spaces are dilated and infiltrated. As the disease progresses the bloodvessels undergo regenerative changes. The ganglion cells suffer the greatest change. Many of them undergo complete atrophy, and those that are retained and at all recognizable exhibit every variety in change of the cell architecture. The changes in the interstitial tissue and in the bloodvessels are so great that the question has arisen whether the cells are primarily or secondarily affected. At all events, it is certain that in general paresis the nuclei and the neuroglia fibers are increased, that there is a proliferation of spindle cells, and that these are associated with marked changes in the bloodvessels, some claiming that the entire process may be conceived as a neuroparalytic hyperæmia ending in a secondary lymph stasis, and that this lymph stasis is responsible for the degeneration of the nerve elements. Binswanger maintained that the disease involved the nervous elements primarily. Alzheimer, the most recent and the most thorough investigator of the subject, claims that there is a proliferation of the glia in the entire cortex. He lays great stress upon the inflammatory process, and believes that the chief changes are due to destruction of the parenchyma. Much is made of late of the fact that the changes in the nerve elements are not unlike those occurring in other diseases as the result of primary disease of the parenchyma.

In spite of recent advances, it is well to hark back to the important fact that Tuczek first insisted upon—that the tangential fibers are destroyed in general paresis. It was supposed by some that these tangential fibers disappeared, particularly in the association areas of Flechsig. Others maintain that the atrophy is diffuse over the entire cortex. While each one of the pathological features of general paresis may occur in other conditions, it would seem fair to conclude that the disappearance of the tangential fibers, proliferation of the glia, marked vascular infiltration, together with the atrophy of the gyri, particularly in the frontal and parietal lobes, would enable one to make a diagnosis of general paresis from pathological appearances. It should also be stated that other portions of the brain do not escape disease in general paresis; degenerative changes are to be found in the basal ganglia, particularly in the optic thalamus. Westphal, Siemerling, and others have found degenerative changes in the nuclei of the ocular muscles, and such changes have also been noticed in the nuclei of the pons and the medulla oblongata. Westphal was also among the first to note a degeneration of the various systems in the spinal cord. The pyramidal tracts and the posterior columns are those most frequently affected. The degeneration of the posterior columns has been held by many to be identical with the changes in tabes dorsalis; others have thought it to be distinct therefrom. The frequent association of tabes with general paresis may be more readily explained if we presume these changes to be identical in both diseases. Degenerative changes have also been found in the cerebellum

and even in the peripheral nerves. Such a wealth of material has been accumulated with regard to the pathology of general paresis that the student who wishes to investigate this special part of the subject is referred to the articles of Nissl and Alzheimer,[1] Cramer,[2] Schaffer,[3] Kaes,[4] and Joffroy et Leri,[5] from which he may gather the more recent literature.

**Symptoms.**—There are few diseases so easily recognized as general paresis when the symptoms are well developed; there is, on the other hand, no disease of the central nervous system which begins so insidiously and which is diagnosed so often on mere suspicion. If once suspected, in nine cases out of ten the suspicion is corroborated by the further progress. The disease is far reaching, not only for the effect that it has upon the patient himself, but often enough it has important bearings upon business and family ties. Its early recognition constitutes one of the most important duties of every physician, whether he be general practitioner or specialist. Nine times out of ten the patient who is brought to the physician's office with a blank countenance, who enters the room with an air approaching indifference, or at times in a state of excitement, who by the first word he utters reveals the tremor of speech, whose attitude shows that he fails to appreciate the importance of a medical examination, may be recognized as a sufferer from paretic dementia before he has taken a seat in the consulting room. The blank stare, the paretic speech, and the indifferent manner are the first conspicuous signs the patient presents. The disease is characterized chiefly by a progressive form of dementia, by a peculiar tremulous speech, by epileptiform and apoplectiform attacks, and by disturbances in the pupillary reaction.

The symptoms may be divided into psychical and physical. The *mental* symptoms are so much more important than the physical that they must be described first. Many a patient is aware of a change coming over him; others do not appreciate that they are in any wise different from their former selves, and resent the suggestion of illness. In the earliest stage the patient may have what appear o be ordinary neurasthenic symptoms, such as mild depression, a feeling of pressure on the top of the head, irritability, sleeplessness, vertigo, and easy fatigue. In association with these a change in the patient's emotions and general behavior is noticed by those about him. The man who has been extremely punctilious about all his business engagements neglects them and can give no special reason why he has neglected them; in fact, he fails often to see the importance of having done so. The patient who has practised a rational economy becomes wasteful; the man who has been sober in his dress takes to the wearing of garish colors; fancy socks, bright colored cravats, a flower in the buttonhole at all times of the day, are some of the changes noticed in patients whose dress during health was of the sober kind. The family man who has been respectful toward his wife and children, toward his employees and his servants, shows an entire lack of respect, and instead of the decent treatment which he was accustomed to accord them, behaves tyrannically, if not brutally. Such changes denote a marked defect of judgment as to the relations between the patient

[1] *Histol. und histopathologische Arbeiten von Nissl*, Band i, 1904.
[2] *Handb. der path. Anat. des Nervensystem*, Band ii, chap. 41.
[3] *Neurol. Centralbl.*, 1903, p. 802.
[4] *Monatsschr. f. Psych. u. Neurol.*, 1902, Band xii.
[5] *L'Encéphale*, 1908, No. 4, p. 322.

and his fellow-beings. The same patient will begin to squander money, to sign checks recklessly, to grow irritable over small trifles in business, neglecting larger and more important duties, thus proving that his judgment has become defective. Impaired judgment may lead the patient into criminal acts. Petty thefts committed by a person who has all the worldly goods that anyone would require, indecent exposures, improper relations toward subordinates, are some of the acts committed in the earlier stages of general paresis.

With this defective judgment *defective memory* is associated at an early date, so that the patient forgets the facts that everyone is expected to know —the day of the week, the month, the year, the name of the President or Governor, even the names of his family. The paretic at an early stage may be unable, offhand, to give the names of his children, or of his partner; his memory for recent events is often more impaired than his memory for things that have happened long since. It is curious to see how easily the physician may get at the evidence of impaired memory. As a rule, it is best to begin by questioning the patient as to his age. If he states, for instance, that he is fifty-two years of age, and you ask him the year of his birth, he is unable to give it; ask him how long he is married, he fails to state it correctly; if the wife in his presence supplies the answer, he is unable to state the year of his marriage. A simple sum of subtraction is quite beyond his mental powers. If asked to write his name and address, he will be able to do it correctly in the majority of instances, unless he already be in a very advanced stage. Ask him to write the names of some cities which have not come within his daily correspondence, and you will soon detect his inability to do so. The things which he has known most thoroughly he may be able to do; those which require a little reflection he fails in. In the early stage the patient may write New York, Boston, and Buffalo correctly, he is apt to fail on Philadelphia, Rochester, and Constantinople, which last is the stumbling block for many. In every instance, of course, it is well to assure one's self that the patient spelled correctly before the disease came on. His memory may also be tested by putting before him the simplest sums in arithmetic. The ordinary additions he may be able to do, but ask him to multiply 8 by 13 or 7 by 8, or ask him to divide 120 by 15, and one quickly discovers the limitations of his knowledge. Far from being annoyed at his mistakes, the average paretic shows either an indifference to the test or considers it a huge joke.

The difference in the response on the part of the neurasthenic and the paretic is so marked in his answer to these simple tests, that whatever suspicions one may have entertained as to a mere neurasthenia are soon set aside by the results of this short examination. The outcome of these tests is that the first signs of dementia are quickly discovered, and as the disease progresses the dementia becomes more and more marked, so that finally the patient has completely forgotten the most elementary facts and his memory will grow so feeble that in the course of time he is practically nothing but a vegetating organism. So much for the rapidly progressive dementia.

Associated with this dementia are other psychic symptoms. We have alluded to the unusual irritability of the patient out of all proportion to the provoking cause. Of great import are the *delusions of grandeur*, which were by many considered almost pathognomonic of general paresis, but which seem to many of us to play a less important part in the diagnosis

than they did fifteen or twenty years ago. What the cause of these changes in the general clinical picture may be, whether it is the sobering influence of our times or not, cannot be discussed at this point, but every now and then we still hear patients give expression to their exalted state of feelings, and the point that is characteristic of the delusions of grandeur of general paresis, as compared with the delusions of grandeur of paranoia, is that in the former the delusions are extravagant, beyond all reason, and wholly unsystematized. They are the transitory and fleeting expressions of the moment, or mere extravagant utterances, and do not constitute the basis for purposive actions as they do regularly in paranoia.

The delusions of grandeur of the paretic are in general coincident with the euphoria, and vary with the station in life which he occupies. The paretic laborer may exhibit his exalted state of mind by travelling about, as one poor fellow did, all day long on trolley cars to prove that he owned the company. An architect conceived the notion that he had been commissioned by the King of Bavaria to fill the city of Munich with huge palaces, and that the windows, instead of being of ordinary glass, should be of diamonds. The merchant in the earlier stages of paresis may imagine himself doing millions instead of thousands of business per year; to export his goods to Europe he has engaged a fleet of vessels which reaches from New York to Liverpool. The stock broker imagines himself commissioned to corner steel or some other stocks; and thus there is practically no limit to the absurd notions that the paretic may develop. Unlike the paranoiac, although he conceives himself to be one of the richest men in the world or one of the most powerful potentates, he does not exact from others the respect and the homage due to his position.

In relatively few cases the delusions are of a hypochondriacal and even of a melancholy tinge. The belief of an entire absence of stomach or bladder, the idea that the food instead of being digested passes into the head and not into the stomach, or the notion of a change in the genital organs, these and other equally absurd delusions occur in general paresis. Maniacal and deeply melancholic conditions, states of extreme anxiety and fear, also constitute a peculiar feature of the psychical condition of the paretic. The true value of these is easily recognized, first of all by their absurdity and by the association of these delusions with some one of the other physical symptoms characteristic of general paresis or with the distinct evidence of a rapidly developing dementia.

It is worth noting that in the early stages of general paresis many a patient realizes the impending trouble. He may be conscious of the lapses of memory or of the difficulties in transacting ordinary business, and some notice the difficulty of speech. The writer has had a number of patients who made the diagnosis of general paresis on themselves, and in whom the suspicion has unfortunately come true. This was particularly striking in the case of a young lawyer, who had lost a brother from general paresis some years previously, and who had a distinct recollection of the symptoms as the brother exhibited them; at the time he consulted me he recognized that he himself was exhibiting the symptoms of the paretic brother. After the initial stages of the disease have passed the patient soon lapses into a condition of apathy, and many of them become entirely oblivious to everything going on around them and are bedridden until released by death.

The *physical signs* are, if not as numerous, quite as helpful in establishing the early diagnosis during the incipient stages of the disease. Reflex immobility of the pupils is one of the earliest symptoms, and often precedes the onset of other signs by many years. In some patients we have the typical Argyll-Robertson pupil; in others there is an immobility both to light and during accommodation. Irregularity in the contour of the pupil, either when the pupil is dilated and particularly when contracted, together with complete immobility, suggest an active specific virus. Inequality of the pupils and paralyses of the external ocular muscles also occur, the latter more frequently in the cases that are associated with tabic symptoms.

*Speech disturbances* have an unusual significance in this disease. The suspicion of general paresis rests more often upon this one symptom than upon any other. The disturbance consists in a tremor of speech and in the inability to pronounce words with labials and consonants, particularly with *l* and *r*, and in the inability to repeat test sentences consisting of words joined together rather by sound than by their inherent meaning. Ask the paretic to say "truly rural," "artillery cavalry brigade;" ask him to repeat such sentences as "Around a rugged rock a ragged rascal ran;" ask him to pronounce "circocebus griseo viridis" (a pet test of Meynert), and you will find not only difficulty in articulation, but also a memory which is so defective that the patient forgets some of the words or some of the syllables and makes a jumble of the entire sentence. Neurasthenic and other non-paretic patients may hesitate and make some few mistakes when asked to repeat these test sentences, but on the second or third trial they correct their mistakes and pronounce the sentences well; above all things, they show no such defective memory as the paretic patient does. The paretic often after repeated trials makes the same mistakes and finds it impossible to pronounce the consonants and words like "artillery," "electricity," and the like.

In connection with the speech disturbance, *tremors* of the facial muscles, of the tongue, and of the hand are particularly noteworthy. The tremor of the facial muscles is, perhaps, the most significant. When the paretic is asked to show his teeth, the entire face, including the lips, quivers; when asked to protrude the tongue, he exaggerates the movement, the tongue is jerked to and fro; and in most characteristic fashion the paretic, instead of pushing out the tongue once or twice, as the ordinary patient would do, keeps on doing it until he is told to stop. The facial tremor has a further significance, inasmuch as it is seen practically in no other condition except in acute and chronic alcoholism. The existence of this tremor alone necessitates the differential diagnosis between general paresis and alcoholism. The tremor of the hands is of a coarse kind, very different from the tremor of multiple sclerosis, and is more an awkwardness of movement than anything else. This suggests another disturbance so common in the general paretic, viz., a general awkwardness of movement associated with the tremor. In tying the cravat, in buttoning or unbuttoning a vest, in fastening suspenders; in the case of women, in the attempt to sew on a button, to hem a handkerchief, even to fasten a hairpin or a breastpin; in short, in attempting to do the most accustomed acts of every-day life,[1] the patient fails to do

[1] I can recall a musician who exhibited, as a first sign of general paresis, an inability to use the lip muscles properly in playing the flute.

these successfully, and spends an unusual amount of time in accomplishing this. He fumbles and grows excited over the most trifling actions. The same tremor and awkwardness are shown in the attempt to write. The paretic may retain the power to write his name and the words which he has been accustomed to write again and again. Oftener his writing is tremulous, his memory is so defective that he omits letters and syllables in the most ordinary words, and as the disease advances he forgets even to write his name, and is no longer able to sign a check or any document of importance. The difficulties in writing may occur so early that the patient is startled at his inability, and worries about this more than about any other symptom.

The *deep reflexes* may be either lost or exaggerated. In keeping with the changes which are known to occur in the spinal cord, it is not astonishing that this difference should occur. In some we have, together with the immobility of the pupils, a loss of the deep reflexes, of the knee-jerks, and of the Achilles tendon reflexes—the tabic series of symptoms. In others the reflexes are exaggerated, the knee-jerks are very much increased, and ankle clonus may be present. The exaggeration of the reflexes must be considered, particularly in those cases in which there is a question as to the condition being one of neurasthenia or of general paresis, although in neurasthenia it is most unusual to have an ankle clonus, whereas the knee-jerk may be exaggerated.

A general *muscular weakness* is also a frequent symptom, particularly in the second half of the disease, and offers excuse for the term dementia paralytica, which, in perhaps more than one-half of the cases, would seem to be unwarranted. With this muscular weakness, particularly if the patients are kept in bed, a general flaccid, atrophic condition of the muscles is apt to be associated, but it is an atrophy that is due more to disuse than to anything else.

Among the physical symptoms we must also include convulsive and apoplectiform *seizures*, which occur during the progress of the average case. While the rule holds good that these seizures occur in the second half of the disease, they sometimes constitute one of the earliest signs. The writer has always insisted that if a patient past middle life, who has neither cardiac nor renal disease, develops epilepsy or has an apoplectic attack, the suspicion is justified either of active syphilis or of general paresis, if not of both.

Not infrequently the neurologist is asked to see a patient who is supposed to be in a uræmic convulsion. Examination of the urine, however, fails to show any trace of kidney affection, and the suggestion that the convulsion may be the first symptom or an early symptom of general paresis comes as a surprise to the general practitioner. In the writer's experience, patients who exhibit apoplectiform or convulsive seizures at an early period run a very rapid course, the disease often ending fatally within six months or a year after such convulsions or apoplectic attack. The convulsive attack is of the ordinary epileptic character, and can in no wise be distinguished from the typical seizure, unless it be that the patient recovers very rapidly and appears, possibly for a week or more thereafter, to be in unusually good health. A little later, however, the first signs of the dementia set in, and on closer examination the patient is found to present other characteristic signs of general paresis. The apoplectic seizure may result in a temporary hemiplegia, from which the patient also may recover with unusual

rapidity, and in other patients there may be, with or without hemiplegia, a transitory aphasia. The symptoms of these apoplectic or apoplectiform seizures disappear with unusual rapidity, but they leave the patient, as a rule, in a disturbed state, and with every recurrent attack—some paretics have six or even more such seizures—the mental condition becomes more and more apathetic and there is a steady deterioration. The rapid disappearance of the apoplectic symptoms leads to the suspicion that these attacks are not due to the ordinary vascular accidents, but that they must be due to some temporary interference with the circulation. In patients who have died of general paresis after apoplectic seizures no sufficient cause has been found for the apoplexy.

*Vasomotor* and *trophic* disturbances are not unknown. Among these we need merely enumerate bedsores, perforating ulcer of the foot, arthropathies, gangrene. There is also at times an unusual fragility of the bones, so that fractures occur most easily and not infrequently lead to the suspicion or to the accusation that the patient has been severely handled by an attendant. In asylums hæmatoma of the ear is looked upon with especial suspicion as a symptom of unusually grave import. It occurs not only in general paresis, but also in many other psychic disorders. While it may be due to slight trauma, it surely would not occur if it were not for the marked tendency to trophic disorder. In view of the grave importance and of the grave significance attached to this one symptom, it is worth noting that every now and then there is recovery from grave mental derangement is spite of the occurrence of hæmatoma, and the writer has seen one such recovery even in a case in which the diagnosis of general paresis had been made. Pruritis, salivation, and universal cyanosis occur in rare instances, but have no pathognomonic value.

**General Course.**—Enough has been said to indicate that the disease begins in insidious fashion, that there is often a suspicion of serious disease impending before the definite diagnosis can be made, and that in the majority of instances, if the suspicion has once been entertained, the worst fears, unfortunately, prove to be well founded. After the initial period of excitement or depression, accompanied either by hypochondriacal delusions or delusions of grandeur, after the development of the typical mental and physical symptoms, the disease passes into the stage of progressive dementia. After the lapse of months or of one or two years, during which time the patient, fortunately for himself, had been in a state of euphoria, and, as a rule, is the only one in the entire circle ignorant of the severity of the symptoms, he passes into the terminal stage, in which he is bedridden, a mere vegetating organism, soiling himself, developing bedsores, becoming more and more emaciated, and dies either from sheer exhaustion, from an apoplectic seizure, of from intercurrent diseases, such as cystitis, enteritis, or septicæmia following upon bedsores. Only a few die during or after an epileptiform or apoplectiform seizure.

**Duration.**—It is commonly supposed that the disease runs its fatal course in two, three, or five years, but every alienist has seen cases of undoubted paresis that have run a course of ten, fifteen, or even twenty years. Many of us have obtained the impression that general paresis does not run as rapid a course as seemed to have been the rule years ago. It is difficult to claim whether this is due, on the one hand, to earlier recognition, so that the period of observation is longer, or to the more careful and more

persistent antisyphilitic treatment which these patients now receive. But that careful treatment has much to do with the prolongation of life in general paresis is unquestionably true. Some of the longest-lived paretic patients are among those who have been nursed at home, where better attention surely can be given than in the average asylum. There is also a further possibility that some of these long-lived cases of general paresis are, after all, cases of syphilitic pseudo-paresis, and in this connection it is necessary to call attention to the marked remissions occurring in the course of the disease. Two and three such periods of remissions amounting to months and even years are not unknown. The most troublesome remission, however, is the one which frequently sets in after the disease was first suspected and after a proper course of treatment has been instituted; the improvement may be so great that doubt arises not only in the minds of members of the family, but of the physician himself, as to whether the case was, after all, one of genuine general paresis. This doubt, however, is soon dispelled by the development or continuance of the typical physical symptoms and by the recurrence of the psychic disturbance.

**Prognosis.**—This is most unfavorable if we allow for the few questionable cases which are supposed to have recovered. It is safe enough to predict an early fatal termination in almost every case of general paresis, and yet the experience of each individual alienist will undoubtedly bear out that of the present writer, who, in spite of his knowledge of the grave prognosis, has insisted that recovery is not altogether impossible, or at least that such pronounced remissions may occur that the patient may be able to resume his duties and be a useful member of his family and of society for a number of years. This is supported by the fact that several patients in whom, more than fifteen years ago, at least three and four prominent alienists made the diagnosis of general paresis are at this day mentally well, although they still present some of the physical signs of the disease. There are disorders in which the diagnosis of general paresis is made for the want of better knowledge, and until we learn to discriminate better than we can at present between the various forms of general paresis, it is well to know that some cases may behave contrary to the ordinary rule, and that because the diagnosis of general paresis has been made, particularly in the earliest stages, a fatal termination is not necessarily inevitable.

**Diagnosis.**—A disease presenting as manifold a series of symptoms will naturally give rise, at times, to great difficulties in diagnosis. When the mental condition is in doubt the presence of the physical signs enumerated in the preceding pages is of the greatest possible import. Thus, if a patient presents what would ordinarily be supposed to be symptoms of neurasthenia, and if he, in addition, has small pupils and either diminished or exaggerated reflexes and a slight peculiarity of speech, the probability of his having more than a mere neurasthenia becomes very great. In attempting to differentiate between neurasthenia and general paresis stress must be laid on the fact that while the neurasthenic may complain of lack of concentration, of his inability to recall things, of pains in the head resulting from the effort of thinking, etc., he presents no signs of actual mental deterioration. Yet the distinction cannot always easily be drawn. In the writer's experience a patient was suspected of suffering from neurasthenia until the occurence of an epileptic attack helped to clinch the diagnosis of general paresis. Paretic speech can hardly be mistaken for any other speech disturbance,

but the hypochondriac and the neurasthenic are apt to notice slight hesitancy of speech which may occur within normal bounds, and noticing these may accentuate them; but even if there is slight hesitancy in articulation, it is very rare, indeed, to have any such evidence of defective memory associated with defective speech as occurs in general paresis. Moreover, the neurasthenic is much more conscious of his disturbed condition, and is much more likely to attach great importance to it than the paretic does. Marked as the difference between the two diseases is, every now and then cases will occur, particularly in those who have been known to have had syphilis, in which for a period of a few weeks or even longer there may be a doubt as to whether neurasthenia or general paresis is in process of development. Such cases are much more apt to occur in private practice than in hospital experience. If the difficulties of diagnosis seem to be insuperable, the examination of the cerebrospinal fluid and the Wassermann reaction of the blood will give further help.

The occurrence of epileptiform and apoplectic attacks is of great importance in doubtful cases; also when the question arises whether the case is one of general paresis or of multiple cerebrospinal sclerosis. The writer has seen few cases in which there was any reason to hesitate with reference to *disseminated sclerosis*. The tremor, the scanning speech, the marked exaggeration of all the deep reflexes, the nystagmus, are symptoms which are never or rarely as marked in general paresis as in disseminated sclerosis, nor does the mental condition accompanying multiple sclerosis at all suggest dementia paralytica. A difficulty may occasionally arise if one sees a patient with disseminated sclerosis in or near the terminal stage. In that case, without previous history, a condition of dementia paralytica might be suspected, but the protracted course of the disease, the antecedent existence of physical signs before the mental symptoms developed, and the entire aspect will help to distinguish between the two diseases.

Far more difficulty is experienced in attempting to differentiate between general paresis and *syphilitic pseudo-paresis*, or lues cerebri. The difficulties are increased by the fact that the two conditions are very similar, and that even the pathologist cannot easily establish the line of demarcation. Clinically it may be said that in lues cerebri there is always complete immobility of the pupils both to light and during accommodation. The onset is often preceded by other specific accidents, as previous paraplegias or hemiplegias that have ended in more or less recovery. The dementia is not as rapidly progressive as in the genuine form of dementia paralytica. Above all, we may argue the presence of a lues cerebri from the instantaneous effect of antisyphilitic treatment. This is never, or rarely, as prompt in general paresis as it is in this special class of cases. After all has been said with regard to this differential diagnosis, the fact remains that cerebral syphilis often gives rise to a combination of symptoms which it is impossible to differentiate from genuine dementia paralytica. Furthermore, everyone has seen cases which have been diagnosed as syphilitic pseudo-paresis, pass into genuine forms of the disease, so that it has been the writer's habit, whenever he has been compelled to make the diagnosis of a pseudo-paresis syphilitica or of brain syphilis with mental symptoms, to state that the case is more hopeful regarding the possible effects of treatment than a typical general paresis would be, but that these specific forms often terminate in typical genuine paresis.

There is also an *alcoholic pseudo-paresis* which may lead to some confusion in diagnosis, but the difficulties are not nearly as great as in the case of syphilitic disease simulating general paresis. The psychic symptoms due to alcoholism may result in a demented condition, but, as a rule, there has been a history of delirium tremens, there are very marked delusions and hallucinations, and, more than this, there are the distinct physical symptoms of alcoholism, possibly even a partial peripheral neuritis, and the pupillary reflexes are rarely as much involved as they are in syphilis or in general paresis. Some day the examination of the cerebrospinal fluid may possibly lead to a safe differentiation between these various conditions.

There is also an *arteriosclerotic dementia* which we see in older persons, which may have some resemblance to general paresis, but, first of all, the age of the patient argues rather in favor of an arteriosclerotic process than general paresis; the dementia is not as rapid and rarely as complete; and, as a rule, before any suspicious mental condition is reached there has been a history of hemiplegia, transitory attacks of aphasia, vertigo, and of numerous other conditions which we all know to be associated with a continued arteriosclerosis.

After giving due consideration to all these points of diagnosis, the fact remains that when general paresis is suspected it is present in fully 90 per cent. of the cases, and that in the remaining 10 per cent. it will be most difficult, even for the most experienced practitioner or specialist, to state whether or not the diagnosis of general paresis or of some form simulating general paresis must be maintained.

**Treatment.**—Granted that in the vast majority of instances general paresis is an incurable disease, there is, nevertheless, much to do if the patient is to be properly cared for. In the initial stages the proper treatment may bring about a cessation of the symptoms or a very marked remission, and the patient may be enabled to return to his family and to his work for an indefinite period. The first essential is that the patient be kept free of all worry and excitement, and that he be subjected to as few new mental impressions as possible. To send a paretic travelling with an attendant for a change of environment, as is so frequently done in other psychoses, would be a grievous error. The paretic brain needs rest above all things. Such rest cannot be obtained by a change of environment unless that change be to a quiet country resort or some special sanatorium or institution. Many writers insist that all paretics shall be committed at once to institutions for the insane. It is well to do this, in view of the fact that the paretic is wholly irresponsible and may suffer bodily harm and inflict injury to others because of his lack of judgment and responsibility. The initial symptoms are often so slight that the family are loath to believe that the patient is a source of danger to himself or to others. In the crowded streets of our cities a man in the initial stages of general paresis is scarcely able to take care of himself properly. At a crowded crossing, getting on and off crowded conveyances, he is apt to become confused, and is fortunate if he is not injured. The paretic surely needs supervision at a very early stage. On the other hand, among the well-to-do classes, if a reliable attendant can be secured, it is often feasible to retain the paretic in his own home very much longer and with far less difficulty than in the case of patients suffering from other forms of mental derangement. Other things being equal, the writer cannot support the contention that every patient suffering from general paresis

must at once be confined in an asylum. Each case should be judged upon its individual merits and according to the conditions of the environment.

The close relationship between syphilis and general paresis, the fact that it is often difficult to differentiate between brain syphilis, general paresis, and so-called syphilitic pseudo-paresis, makes it incumbent upon the physician in every instance to give the patient the benefit of mercurial treatment, particularly if the Wassermann reaction be positive, or if the cerebrospinal fluid shows a marked lymphocytosis. Even in the absence of these evidences of active syphilis, mercurial treatment should be given a fair trial. As in cases of tabes, so in general paresis, it has been the writer's practice to give a course of at least twelve injections of bichloride of mercury ($\frac{1}{6}$ grain each, 0.01 gram), to interrupt treatment for a few weeks during this period, placing the patient on mild tonics, and then to give a moderate course of iodides (15 to 30 grains, 1 to 2 grams, of the saturated solution of sodium iodide three times a day). The iodide and the mercury should never be administered at the same time. According to the condition of the patient after the lapse of weeks or months another course of treatment should be given. The marked improvement seen in many cases encourages one to advocate strongly this treatment. During the last two years the writer has been guided by the presence or absence of a positive Wassermann reaction. If the reaction has been found positive at one stage of the disease, and if it becomes negative after a course of treatment, as it almost invariably does, treatment may be stopped for a time and may be resumed if the reaction again becomes positive, and even if it remains negative, another course of mercurial treatment after four or six months should be attempted.

German writers claim that general paresis is very apt to be made worse by mercurial treatment, and that it is the syphilitic pseudo-paresis which is benefited by it. The present writer and others in this country are not so firmly convinced of this, and there seems little reason to discard discriminating mercurial treatment in general paresis. The more thorough investigation of the blood and of the spinal fluid has led to the conviction that paretic patients harbor syphilitic antibodies, and some believe that they are still carriers of the spirochæta. Some have recommended the use of atoxyl, which has proved so efficient in sleeping sickness, in the hope that the syphilitic virus might be influenced by this drug. Ehrlich has discovered an arsenic preparation which is certain to cure every animal suffering from sleeping sickness. This arsenophenylglyzin has been recommended by Alt[1] in the treatment of dementia paralytica. It is a light-yellow powder, easily soluble in water, easily oxidized, and, therefore, supplied in vacuum tubes. The powder is to be used immediately after opening. One gram of this is to be injected intramuscularly on two successive days in the case of a healthy adult. Weaker persons are to receive 0.8 gram. The only contra-indication is the presence of complicating cardiac disturbance. Alt reports that he has treated 31 paretics who yielded decidedly positive Wassermann reactions. In 7 of these the reaction disappeared completely; in 1 of them it returned after five weeks; in the other patients the Wassermann reaction was distinctly weaker in the course of treatment, but returned after a short period of time. In view of the hopelessness of other forms of treatment, this recommendation of Alt deserves notice. It should be

[1] *Münch. med. Woch.*, July 20, 1909.

mentioned here that in this country, so far as the present writer knows, no serious attempt has been made to use this drug.

In every case of general paresis there is need of purely symptomatic treatment. The ordinary states of excitement may be influenced by the rational use of bromides, codeine, and the hydrobromate of hyoscine ($\frac{1}{100}$ grain, by mouth or hypodermically). The insomnia, which is a very troublesome symptom in many cases, is to be combated by the use of bromide, chloral, veronal, and trional, in the ordinary doses. The epileptic and apoplectiform attacks are to be treated as these conditions would be treated if they occurred independently of general paresis. Mild hydriatic procedures are in order, but extreme forms, such as cold spinal douches and the like, should be avoided. In the chronic and bedridden cases the function of the bladder has to be carefully watched; the tendency to bedsores must be carefully considered. If the patient cannot feed himself, the food must be administered carefully and discriminatingly. In the advanced stages of general paresis treatment is at best unsatisfactory and purely symptomatic. The rational medicinal treatment and careful nursing in the earlier stages of the disease are often followed by encouraging improvement in the condition of the patient.

# CHAPTER XVIII.

## NEURASTHENIA. THE TRAUMATIC NEUROSES AND PSYCHOSES.

By CHARLES W. BURR, M.D.

### NEURASTHENIA.

The word neurasthenia is used with both an indefinite and a precise meaning. Unfortunately it is frequently used to designate very unlike conditions, sometimes as a cover for ignorance, and sometimes as a euphemism for a more serious word—insanity. It is often confused with hysteria and sometimes regarded as mere laziness. It may be so severe as to cause permanent disability or so slight as to be recovered from in a few weeks. Its symptoms are numerous, and its true primary cause is still a matter of dispute. The difficulties of its study are, therefore, manifold, and many of the apparent differences of opinion among writers arise from using the same word to designate different things. Notwithstanding the frequent use, indeed abuse, of the word, primary neurasthenia really is infrequent, and the diagnosis is becoming more rare as the methods of study improve, as our knowledge of the chemistry of disease grows, and as we examine patients with greater and greater thoroughness. Yet it is a really existing thing, and not a mere word. Strictly defined it includes only a condition of pathological weakness without discoverable lesion, showing itself by too rapid and too great fatigue, physical or mental, or both, emotional unbalance, and undue irritability (too great response to stimuli) of the nervous system. It means disability or inability more than perversion of function. The machine runs too slowly and weakly rather than wildly or pervertedly, although this also happens.

One thing always to be remembered is that there are two distinct types of the condition, one a primary disease and the other a secondary state. From the examination of the patient on any one day, and in ignorance of his past history, it is often impossible to determine which of the two we are dealing with. Time will always show, but it is important to make the correct diagnosis early and not late, and therefore it is important to learn the patient's personal and family history. It is more than probable that future discoveries in biological chemistry will decrease the number of cases diagnosed as primary and increase the number diagnosed as secondary, but there will remain a certain number prenatal in origin and not due to external causes arising after birth.

**Etiology.**—We shall first consider the cause of primary neurasthenia and then some of the diseases causative of secondary neurasthenic conditions. Primary neurasthenia is prenatal in origin and, in the majority of cases, inherited. These two words, prenatal and inherited, are usually

used by medical men as synonymous, but the former includes the latter. Hereditary influences are those carried by the germ cell and the sperm cell, from generation to generation, and are to be kept separate from the influences arising and acting after fusion of the cells. Prenatal influences include the hereditary plus those which, arising after conception, act upon the fetus from without. We know little about the effects of pathological prenatal influences, save in the case of gross disease, and nothing about the mechanics of heredity. We know but little of the late results (results appearing in childhood or later) of mild illness of the mother or of the embryo on the future mental and physical condition of the offspring. It is very possible, for example, although it has never been proved, and the obstacles in the way of proof are almost insuperable, that an acute febrile attack in a pregnant woman, not severe enough to cause abortion or anything noticeable at the child's birth, may yet so weaken it that the power of resistance to the stress of life will be greatly decreased. It is possible that alcoholic intoxication of either parent, otherwise healthy, at the time of conception, may have a permanently ill effect on the offspring which yet may not show itself till long after birth. It has been claimed that this has been clinically proved by the exclusion of all other possible causes, but we do not know, nor can we learn, whether acute alcoholism can affect human spermatozoa and ova, and to exclude all other causes is, to say the least, difficult. It has been claimed to have been experimentally proved that acute intoxication does affect the spermatozoa in certain lower animals. Chronic alcoholism may, by the widespread and serious tissue changes it produces in the parent, with consequent disturbance of function, undoubtedly affect the offspring. We know, as said, only the grossest facts in prenatal pathology, and minute changes caused by apparently trifling disturbances may have lasting effects which may not appear till far on in adolescence or even maturity.

The *fundamental* symptom of neurasthenia is inability to withstand the normal amount of stress without breakdown, shown by excessive fatigue and irritability. Stress is the sum of all the forces which act upon the individual organism and its constituent cells, and therefore includes disease, the wear and tear of life, everything affecting the emotions, mechanical work, and intellectual effort. In the primary form of neurasthenia this weakness is congenital; we come into the world with unequal powers of resistance to emotional, intellectual, physical, or chemical stress. We are only beginning to know something of the causes of inequality of ability to resist, and what we do know concerns almost altogether chemical immunity in mycotic disease. Of the causes of congenital muscular or mental weakness apart from the cases in which there is manifest gross disease we know nothing. It is possible that congenital abnormalities of function of one or several of the ductless glands may be at the root of much neurasthenia rather than any disease arising primarily in the nervous system itself, but to-day this is a theory rather than a proved fact. This is certain, that while the great majority of people are able to successfully resist the ordinary stress of life, there are a number who succumb, whatever the reason may be, under a burden which the majority carry well. The chief predisposing cause seems to be, in fact is, a bad heredity. In almost all cases of serious primary neurasthenia, if the family history be known, there will be found "a bad strain in the blood." By this, of course, is not meant moral viciousness, but biological weakness. There is present some nervous instability in the

family. It is a tendency which is inherited, not neurasthenia itself. Indeed, it is a general law that not disease but a tendency to disease is inherited. Thus, neurasthenia does not necessarily beget neurasthenia, nor hysteria hysteria, but nervous or mental instability of any kind produces either itself or some allied weakness.

Further, since in nature there is always an endeavor (the limitations of language compel us to personify nature and speak of it as if it willed) to regain the normal, a very bad ancestry may beget a good posterity. Most neurasthenics, then, show something wrong in the family history. It may be general nervousness, eccentricity, or genius, at least pseudo-genius, or it may be some definite disease, as hysteria, insanity, epilepsy, gout, or alcoholism. General nervousness or mere invalidism is a more frequent ancestral trait than the various neuroses, psychoses, or organic nervous disease. This may be merely because invalidism is more frequent in general than serious organic or functional nervous disease, *i. e.*, the greater frequency may be merely arithmetical and not one of percentage. The writer is well aware that there is a tendency to-day among certain writers and in the popular mind to minimize the effects of inheritance and to magnify those of environment, to believe that education and training can overcome all inherent evil tendencies, and that all of us could be well, strong, brilliant, and altogether normal if only we had been taught. Education can do much, and a bad environment may injure the good just as a good environment may help the bad, but an inheritance of health is still better than vast riches.

Some primary neurasthenics break without any discoverable exciting cause; in most, however, some unusual external stress is necessary, however slight. The average age of onset of marked symptoms is late adolescence or early maturity, but some subjects retain fair health till middle life and a few succumb in early childhood. Puberty is a critical period. Some of the boys and girls who in childhood appear bright and strong, in every way normal, at about fourteen or fifteen begin to complain of headache, backache, and mental tire, no longer do well at school, cease to take pleasure in active muscular play, and are fretful and peevish. They are really entering on a life of neurasthenia. Some die of senility while they are still mere children by the calendar. Only the healthy man's age is measured by the courses of the sun, the weakling's clock runs more quickly. The amount and nature of the stress necessary to cause breakdown vary with the individual. Some can carry a burden almost equal to the average, others reach the straining point far short of this. Mental work, even if carried to excess, provided it is not accompanied by worry, is not a frequent cause, but when worry is present, neurasthenia is frequent, and often the first symptom is inability to think consecutively and continuously. Many intellectual men are neurasthenic, but not often because of their work, and much of the world's work has been done by invalids. Darwin was neurasthenic for years, not because of his hard thinking, but because of his almost continuous seasickness while on the "Challenger." Carlyle's whole writings are tinged with invalidism. Much pessimism is due to what one may call neurasthenic dyspepsia, and the reader, especially if he be young and ingenuous and take the printed book too seriously, may, in turn, get a mental dyspepsia leading to physical neurasthenia. It is probable that the reading of books of a certain type has caused more neurasthenia than the writing of them.

*Emotional stress* is a much more important factor than physical overwork. In women the countless little troubles of life and family care are very important. The common notion that only the rich and socially well placed among women have neurasthenia is absolutely false. It is more common among working women than among those who are relieved of the burden of work. This is becoming more noticeable since women have invaded the work formerly monopolized by men. School teachers and workers in stores and factories are especially susceptible. The mill demands and gets an awful tribute in the broken health of countless girls. It is not alone, not so much, the actual work that causes breakdown as the whole manner of life and the associations of the average mill girl. A few, one had almost written a great many, physicians have objected to the so-called higher education of women on the ground of injury to health, but they are regarded as old fogies and no one listens to them. Perhaps they will be listened to some time in the future. To-day it is wisest not to endeavor to oppose the current of popular opinion and desire, for that is impossible, but let us not try to make Portias out of common mortals nor forget that motherhood is rather more important than science, literature, or even politics. Professional life, especially the practice of medicine, has done but little good to many of the women who have gone into it. It is dangerous for a medical man to venture to advise medical women, but considering the relatively small number of women who are physicians, the percentage of neurasthenic wrecks among them is not small. The reason of this is not far to seek. The emoluments are small, the work severe, the worry great, the responsibility heavy, and the opportunities for success few. Only the strong can survive.

Child-bearing should in no way be injurious to normal women, but among those under par it is an occasional cause, as are the infectious fevers. Women are more prone to primary neurasthenia than men, but secondary, symptomatic cases seem more frequent in men. This is probably because men are still exposed to more stress of different kinds. Bad education is a great predisposing cause. The most important purpose of education, far more important than book learning, is to teach self-control, and none needs to be taught this as much as the offspring of the weak. Often, on the contrary, the neurasthenic that is to be receives the poorest education; he often learns unconsciously, from his elders in the family, to whine, to grumble, to be unwisely selfish. His ego is hardened, his altruism destroyed. He is petted and scolded and spoiled and soon made worthless. His little aches and pains are made much of, and obedience is unknown to him. The writer is not preaching brutality to children, and has no sympathy with the puritanical dogma that pleasure is sin and happiness a preparation for damnation, but one must learn to obey before he can command himself. Undue severity and lack of sympathy may do as much harm as the reverse. There are children, and often they are of the best, who are so out of tune with their human surroundings that they are driven in on themselves and live their own internal lives in a subjective world all of their own creation. This, however, more often leads to insanity than to neurasthenia.

*Occupations* are important causative factors. Those causing great emotional stress are most dangerous. Thus, actors, business promoters, speculators (financial, not philosophical), and clergymen of the intense type are especially liable to break down. The fault is not altogether with the work. It is partly due to the fact that emotionalists are prone to be

drawn to it. Climate, or rather the change from a temperate to a hot country, has an influence. Woodruff, for example, has described the neurasthenia occurring in white people living in the tropics. Race, too, is important. The American negro never suffered from neurasthenia till recently. But now it is of not infrequent occurrence, especially in the half-breeds. Alcohol, syphilis, and moral degeneration partly account for this, but more important is the fact that a lower race is trying to compete with a higher, and is unfit to do so. The strain is too great, and, although it seems a hard saying, the fittest to survive in the moral sense are often the unfittest in the biological sense. Apart from organic disease and bad morals, the mere struggle of the best specimens of an inferior race to attain the plane of a superior leads often to their downfall. Among Caucasians the Jews seem to be the most prone to neurasthenia. Of course in them it is not a question of racial inferiority, rather the reverse. The usual explanation given is that years of oppression have at last affected their stamina, but the cause, whatever it may be, is deeper than that. It certainly plays no part in this country, because several generations of living here does not seem to decrease the tendency. Those of us who were taught to read the Old Testament can recall some evidence that even in the days recorded there the disease was not unknown among them.

Much has been written to prove that some one thing is the cause of primary neurasthenia, indeed of all neurasthenia—floating kidney, ptosis of one or all of the abdominal organs, diseases of the genitalia in women, aberration of the sexual function, even lateral curvature and eyestrain, have each been upheld as the one and only cause of the condition. All these things are evil, and any one may be an exciting cause of nervous symptoms, and each should be looked for and if present attended to, but no one is a primary cause. That is inherent in the patient himself. The influence of trauma as a cause will be discussed elsewhere. Probably the most frequent exciting cause is the mere burden of life, the worries and frets, the great griefs and responsibilities, the dull routine, the lack of ability to get interested in the game of life, and, on the other hand, playing the game too hard. If we could be taught, and could obey the lesson, that everyone must live his life within the limitations of strength nature has put upon him, much neurasthenia would be prevented, but unfortunately few are taught till too late, and many who know are so enmeshed in the net of adverse circumstance that they cannot obey the lesson.

In *secondary* or *symptomatic* neurasthenia the cause can usually be found if sought for, but not a few serious organic diseases may, for a time at least, hide their own proper symptoms and show only weakness. The writer well remembers a man under his care when a hospital resident. The chiefs, distinguished and skilled men, diagnosed his case as neurasthenia. He complained of great general weakness and nothing more. After a rest cure he was persuaded and mildly browbeaten into taking more and more gentle exercise, longer and longer walks. He himself thought he was getting better. One day he told me he was too tired and weak to get up, and I, with the superior air of the young interne, rather scolded him. He died before I had walked the length of the ward. The necropsy showed marked chronic myocarditis. He was not a neurasthenic, but died because his heart muscle could no longer do its work. Another patient, a man beginning to be old, lost his wife, to whom he had been devoted. He began

to complain of pain in the back of his head and neck, and of general weakness. He slept poorly, ate capriciously, lost interest in all things, and began to pass large quantities of urine. He then took to his bed. Examination revealed nothing. He improved a great deal for several months, when a sarcoma of the thigh appeared, ran a very rapid course, and he died. The sarcoma had nothing to do with the early symptoms, the association of the neurasthenia and the sarcoma was purely accidental, but it was hard for the family doctor to make the patient's people believe so.

The later stages of syphilis may, for quite a long time, present nothing but neurasthenic symptoms, and, without a history of chancre or of previous organic symptoms, an accurate diagnosis is impossible. Given a man who presents symptoms of weakness without manifest cause, especially if his previous life has been healthy and he has borne stress well, it is important to inquire very closely into the possibility of luetic infection, and if there be any evidence whatever it is well to put him to the therapeutic test. The following is an example: A young man complained of loss of memory and inability to fix his attention, with general weakness. Examination revealed no sign of organic disease. His history, however, cleared the matter up. He had had a chancre about five years before, and later suffered from a transient apoplectic attack with aphasia, still later an epileptiform seizure with a temporary left-sided hemiplegia, and at another time an ocular palsy lasting only a short time. It is remarkable that such serious symptoms should have occurred and left no trace in the way of residual palsies, disturbance of the reflexes, etc., but such was the fact. The diagnosis in his case was easy because a clear history was readily obtainable. His symptoms were not neurasthenic at all, but really due to a diffuse brain degeneration.

Sometimes, and with increasing frequency since the appearance of the present custom of surgeons in getting patients out of bed at the earliest possible moment after an operation, severe neurasthenia follows operation. The writer has not infrequently seen months lost out of the lives of patients and much suffering ensue because surgeons were stingy of a few more weeks in bed. This is true not only of women, but of men patients. Profound neurasthenia often follows typhoid fever and influenza, and in the latter especially there seems to be no relation between the severity of the primary infection, or rather of the symptoms during the fever, and that of the neurasthenia. It is notorious that two or three days of influenzal fever may be followed by weeks, even months, of convalescence, the patient feeling as miserable and being as weak as if he had passed through some long and dangerous illness.

There is a distinct type of neurasthenia, commoner among young men than young women, due to vicious sexual habits in adolescence. Not only does the habit itself do harm, but a large part of the literature about it does more harm. There is scarcely a book on the subject, and they are all sold to and read by youths, which does not do the boy reader harm and, if he is neurotic, irreparable injury. Many a boy has been permanently injured in mind by fright after being made to believe, by reading pseudo-scientific literature, that he is congenitally perverted, when really he is only the victim of a vicious habit which can be cured. Some authors claim that sexual abstinence may be a provocative cause, but there is no foundation for such belief.

**Symptoms.**—It is very difficult to give a clear picture of the symptomatology of neurasthenia, because cases vary so in intensity and severity and because what symptoms or group of symptoms predominate depends so much on the personality of the patient. While the cardinal symptoms of rapid fatigue and undue response to stimuli are always present, the organs which show the irritability vary most markedly in different patients. This variability in the apparent seat of the disease is so great that it has even been proposed to differentiate spinal, cerebral, gastric, and many other kinds of neurasthenia, but this is too great refinement in diagnosis, or rather it confuses the place of manifestation of symptoms with the place of origin of the disease. One organ may present the greatest number of symptoms, but the disease is general. It is impossible to give an account of the disease that will cover and include all cases. The best which can be done is to relate the symptoms and indicate the commonest combinations.

An attack is never sudden in onset, although there may be an acute outburst of severe symptoms. The physician may be told that the illness came on acutely, but investigation will always show that really the onset was slow. The great symptoms are physical and mental tire. The patient finds that she, for the disease is more frequent in women, is very easily fatigued. Molehills are mountains and the grasshopper a burden. Things that she could do before without effort now become burdensome. At first she does not think that there is much the matter. If the condition is severe she becomes unable to make any continuous muscular effort, and soon is bedridden. At the same time mental effort becomes difficult. In the beginning there is no difficulty in starting a train of thought, but in carrying it on for any length of time and in keeping the attention fixed. Finally, continuous thought becomes impossible. There are rare cases in which the weakness is entirely physical and not at all mental. There are men and women who can do continuous mental work when physically they are bedridden. But no matter how difficult it may be to think, there are never any profound perversions of thought. There are no delusions of grandeur, of self-accusation or of persecution, or, indeed, of any kind. The patient may be depressed and even fearful, being startled by the inability to think, and fearing it is the percursor of mental breakdown. Frequently there are morbid fears which may take many shapes—a sense of impending evil, fear of closed or open places, etc.

There is usually some *emotional depression;* the patient is, as a rule, unhappy, but this is a natural though exaggerated result of the weakness. It arises occasionally from hyperconscientiousness, the patient feeling she is neglecting her duty, that she is not properly looking after her children or caring for her husband, that she ought to rise up and do her work, and that it is her own fault she does not do so. Or if she is of a different temperament she becomes peevish, fretful, faultfinding, and supremely and childishly selfish and unreasonable. When depression becomes profound and unsurmountable the patient is bordering on, if she has not reached, melancholia. A few patients maintain emotional equilibrium throughout a long attack. There is never any serious change in the moral sense, and the neurasthenic is never led by illness to acts of shameless crime, or even to slight infraction of the moral law. In severe cases which have lasted a long time there is frequently a great increase in selfishness, and this is not to be wondered at, because there are few Jobs in the world.

*Pain* or rather unusual feelings (paræsthesias) are always present in the head. Occipital ache is the most usual complaint. This may come on apparently spontaneously or follow muscular effort, thought, or worry. There may also be brow ache or pain in the vertex. Some patients complain only of queer indescribable sensations in the head. They speak of them as dragging, drawing, or straining feelings, or compare them to a tight band around the head. Slight vertigo is common. There may be hyperæsthesia of the scalp, and pain may be referred to the hair. After the head the spine is the most frequent seat of pain. It may be localized or diffuse, and, as a rule, a mere touch on the skin over the backbone causes more pain than deep pressure. Although there is never anæsthesia, the patient frequently suffers paræsthesias in the hands and feet—pins and needles, a feeling of a tight glove, cold or hot flashes. Curiously enough, pain due to organic disease, *e. g.*, gallstone colic, may be fairly well borne. Paræsthesias are often more distressing than pain. *Circulatory disturbances* are always present. The hands and feet are cold, moist, and bluish or bluish-red, or the skin is dry and scaly. There may be attacks of cardiac palpitation, a choking sensation as if the heart were in the throat, or the heart may seem to stop beating for an appreciable time. Sudden movement may cause vertigo. Although the skin is washed out and pasty looking, and the lips have lost their color, there is no true anæmia; the circulation, not the blood, is at fault. There may be, and probably is, a decrease in the total quantity of blood in the body—that is a thing that cannot be measured—but there is never any great decrease in the hæmoglobin percentage or in the number of red and white corpuscles. Leukocytosis is never present unless there is some complication. The explanation of the cases of apparent plethora, the red-cheeked patients, probably is a reduction in the quantity of the plasma of the blood, causing an increased blood count from concentration. This cannot be proved, but it is probable.

Loss of appetite may be present from the onset, come on later, or the appetite may be capricious—to-day ravenous and to-morrow gone. The sight of food may produce nausea, and its presence in the mouth may fail entirely to stimulate the salivary glands to action. This is one of the causes of difficulty in swallowing in neurasthenics. Food in the stomach may almost entirely fail to excite the gastric glands to secrete. After eating there may be a sense of weight in the epigastrium, a feeling of smothering and even pain. The intestines may become distended with gas. Sometimes eating increases the headache. The bowels are, as a rule, constipated, or constipation may alternate with diarrhœa. Pain may be referred to the appendix in the absence of any inflammation in it. Often there are annoying feelings in the rectum after a stool, as if the bowel had not been emptied. The gastric atony may go on to catarrh and eating be followed by vomiting. When anorexia and indigestion is marked, emaciation always follows, and may be so great as to reduce the patient to a mere skeleton and make one fear the existence of some malignant disease or visceral tuberculosis.

Sleep is, as a rule, poor; either there is more or less insomnia or the patient dreams much and nightmare is common. The patient does not awake rested and refreshed, but weary and depressed. The morning is the worst period of the day with all neurasthenics. In mild cases there is no disturbance of menstruation, but in the more severe amenorrhœa occurs and leucorrhœa is common. There may be nervous irritability of the bladder

and consequent frequency of micturition, and, on account of the habit of neurasthenics of taking too little liquid, the amount of urine voided may be below the normal and its specific gravity too high. Of course, the presence of albumin and casts indicates something other than neurasthenia. Endeavors have been made to find distinctive substances in the urine which would throw some light on the origin of the disease, but so far without success.

The deep reflexes are, as a rule, increased, but genuine, persistent ankle clonus can never be obtained, although quite often there is an abortive or spurious clonus shown by a few coarse and irregular vibrations of the foot when it is passively dorsally flexed. The existence of a persistent and rhythmical ankle clonus means organic disease. A tap on the knee often causes a start of the entire body. Although perhaps increased in amplitude, the knee-jerk is never spastic. It often diminishes under repeated stimulation, and when the patient is extremely weak it may be so slight as to be obtainable only on reinforcement. If the knee-jerk is absent it is well to look for other signs of organic nervous disease, for surely the absence is not caused by neurasthenia. Its absence is congenital and significant of nothing in one person in five hundred.

Vision and hearing are the two special senses most often affected. True deficiency of vision is rarely present at the beginning of an examination, but retinal tire soon comes on. The patient can read for a short time, but soon the print becomes blurred and she complains also of pain in the head and mental fatigue. Sometimes there is retinal hyperæsthesia, and the patient desires to be kept in a dark room, saying light causes intense pain. Reversal of the fields of vision is a symptom of hysteria, not of neurasthenia, but rapid contraction of the fields as the examination proceeds, caused by retinal exhaustion or possibly by inability to hold attention acutely enough to appreciate slight stimuli, is not uncommon. The auditory symptoms are somewhat similar. Either the patient appears to be somewhat deaf, which is really a matter of lack of attention, or she is extremely sensitive to sounds of all kinds. There are no visual, auditory, or other hallucinations. Ringing in the ears is not very infrequent, but the subjective sound is never interpreted as bells or voices.

**Diagnosis.**—The diagnosis of neurasthenia is largely by exclusion. It is necessary to exclude all possible gross organic disease. One of the diseases most often confused with neurasthenia and frequently associated with it is *hysteria*. The error is of no great practical importance from a therapeutic point of view when it concerns physically weak hysterics, because the same methods of treatment are then useful in both diseases. As a matter of intellectual accuracy it is, however, well to keep them apart. Hysteria is as much abused a word as neurasthenia, and it would be well if it were used to designate only that group of symptoms including certain palsies, convulsive attacks, anæsthesias, and respiratory and circulatory disturbance, with or without a certain type of purely mental symptoms. Convulsions, paralyses, and anæsthesia are not symptoms of neurasthenia under any circumstances, and their presence indicates some other disease.

The early stage of chronic *insanity*, especially paresis, may look very much like simple neurasthenia. By the time a paretic comes under the care of an alienist, or is so ill that the question of sending him to a hospital is being considered, the diagnosis is usually not difficult, but in the early, the

prodromal, stage it may be impossible. In the cases of paresis in which there are physical signs of organic disease from the outset, inequality of the pupils, abnormality in the iridic reflexes, scanning speech, spastic or absent knee-jerks, and intention tremor, the diagnosis is easy, because none of these occur in neurasthenia. Tremor is often seen, but it is an emotional tremor and not like that present in paresis. When the grandiose state is present the problem is easy, because no neurasthenic could be grandiose, but some paretics are depressed and even hypochondriacal. Moral degeneration, as shown by shamelessness or criminal acts, always excludes neurasthenia. The neurasthenic is often very selfish, but never morally degenerate. That is to say, the disease never causes moral degeneration. It may have existed before, and of course the neurasthenia will not cure it. In paresis there is a great lack of power of intellectual judgment even very early. In neurasthenia there is not so much lack of judgment as inability to form judgments continuously for any length of time. So far as the neurasthenic judges at all, she is prone to judge correctly. Notwithstanding all these points of differentiation, it may need several weeks' study before a correct opinion can be formed. When distinct delusions of any kind persist in a patient, he or she is insane and not merely neurasthenic. The best rule to follow is to regard every patient who shows pupillary and other reflex abnormalities as seriously ill.

Mild *hypochondria* is sometimes called neurasthenia, but when there are delusions, when the patient believes he is suffering from some nonexistent disease, he is mentally diseased, and needs treatment appropriate for that and not for neurasthenia. It is sometimes very difficult to differentiate the very early stage of adolescent insanity from neurasthenia. Indeed, the onset is often so insidious that it is impossible to foretell whether the patient is going to be seriously ill or not. Always, however, there is some distinct mental perversion even at the start. The patient is a little queer, his manner changes, his behavior alters, his ideas show perversion, and there occurs some manifestly insane act. When hallucinations and delusions appear, the diagnosis is certain. Cases of mild melancholia may seem to be neurasthenia at the outset, but the emotional depression is always greater than the physical weakness. There is always, even in the mildest cases, a sense of personal unworthiness. So soon as delusions of self-accusation are established there is no doubt the patient is insane. Certain cases of *Graves' disease* may be mistaken for neurasthenia. The classical case, with exophthalmos, goitre, and rapid heart is easy enough to diagnose, no matter how many neurasthenic symptoms may be present, but cases occur in which the weakness seems to be, before careful examination, the only symptom.

One of the difficult problems in medicine is the diagnosis of neurasthenic *headache*. There is a tendency among some physicians to jump to the conclusion that if a woman—with men they are more careful—complains of constant persistent headache it is not so severe as she imagines and is caused by a slight neurasthenia or "a run down system." This is rather dangerous, because, while head pain, or at least some strange feeling in the head, is common in neurasthenia, yet headache from other causes is much more frequent. Counting all kinds of headache, that from neurasthenia is not common. There is little in the headache itself which is characteristic except that it is apt to be described rather as a dull, queer, or strange feeling

than as a sharp pain. It may be located in any part of the head, but most frequently in the occiput, the base of the brain the patient will say, and the nape. The next most frequent seat is the frontal region. When at all severe it is apt to greatly decrease ability to think and to bring on emotional depression, but these same effects follow headache from other causes. The eyes should always be examined, not only for refractive errors and muscular trouble, but also for optic neuritis and albuminuric retinitis. The writer has more than once seen a brain tumor in the early stage mistaken for neurasthenia. Typical migraine is easily diagnosed, but aberrant cases may require careful study. The nose should always be examined, for disease of it is a not very infrequent cause of headache, although not of paræsthesia. Headache should never be regarded as neurasthenic unless all other possible causes are excluded and other symptoms of neurasthenia are present.

It is possible at first sight to mistake myasthenia gravis pseudo-paralytica for neurasthenia, but in it the muscular weakness is at first, if not throughout the course of the disease, local. It begins with drooping of the eyelids, weakness of the muscles of mastication, and trouble in swallowing. The later picture is definite.

**Prognosis.**—The severity of neurasthenia varies greatly, and so much depends upon whether the patient can afford proper treatment and is so situated as to regulate and control the manner of her life that it is impossible to make any general statements as to prognosis. Taking all neurasthenics together, the outlook as to both life and cure is good. Many men and women are mildly neurasthenic for many years, and yet not only live, but do their duty in the world and not infrequently a great work. Much of the world's work has been done by invalids, many of them congenital neurasthenics. Throwing out the mild cases and counting only the severe ones, the patients who become bedridden and need strict rest cure, the danger to life is not great, but the likelihood of recurrence is extreme. If the patient be so situated that she can control the manner of her living, it is often possible for her to lead a fairly happy and quite useful life by living according to rule, but should stress come to her, and sometimes even without stress, she may break again. Again, if the stress which caused the attack was very severe and the patient reacts well under treatment, the danger of recurrence is much less than if the stress was slight and the reaction slow. Thus, many busy, hard-working, and much worried men pass through a very severe attack, recovering completely and remaining well. The prognosis in secondary neurasthenia, that occurring in the course of and caused by some organic disease, depends entirely upon the nature of the primary disease. Cases following the acute infectious fevers, even though symptomatically severe, are usually entirely and permanently recovered from.

**Treatment.**—The means used to combat neurasthenia must vary with the severity of the attack. Some cases are so slight as to require nothing more than a change of scene, outdoor play, and, if possible, removal of the exciting cause, while others are so severe that they test the skill of the wisest physicians, who sometimes fail utterly in bringing about a cure. For patients seriously ill the Weir Mitchell rest treatment is the best, indeed, the only, method, and it will be described in detail. Its important elements are absolute rest in bed, food, massage, isolation, baths, and electricity. The patient is best treated away from her own home, in order to get rid of all association with the worries of housekeeping and family life. A well-

regulated rest house, where there are but few other patients and where all the servants and nurses are well trained, is the ideal place, but it is also the most expensive. A not too large special hospital is the next best place, and home, as a rule, the worst. For working people, in the narrow meaning of the term, a bed in a ward of a general hospital ward is infinitely better than home, and much can be and is done in general hospitals in the way of cure. A very small number of patients, those in whom, although the physical weakness is great, emotional control is good, can be successfully treated at home. A well-trained and sensible nurse, not a chatterbox or a scandal-monger, but a woman of brains, tact, and strength, is in any event a necessity, because amateur or family nursing of a neurasthenic always spells failure. She must perform not only the official duties of a nurse, but by her presence relieve the monotony of isolation, for to leave a seriously neurasthenic person entirely alone for hours at a time is disastrous. To have ever with her a nagging nurse, who in her wisdom thinks neurasthenia a polite name for laziness, is still more disastrous. A nurse who is not loyal to the physician in charge is, to use the language of one of the newer pseudo-sciences, a potent source of "malignant animal magnetism." Her psychological influence is evil. Even a good nurse and admirable woman may be ill adapted for a particular patient. If the patient takes a dislike to her, even though to do so be entirely irrational, her usefulness is done. Friction between patient and nurse makes cure more difficult. Patients who need treatment for a long time should, for several reasons, have a change of nurses after a few months. The nurse, no matter how good, is prone to lose interest after a time or become "stale" or even nervous herself, for the work is hard and the strain severe. She may get too well acquainted with the patient, and sometimes, but this is extremely rare, an unwholesome friendship arises. Having obtained the nurse and got the patient to the rest house or hospital, the treatment begins. At first rest should be absolute. The patient should not leave the bed except to go to the toilet. Very rarely the muscular weakness is so great that the bed-pan must be used. The bed should be neither too soft nor boardlike in hardness. Feather beds are the worst, woven wire beds with hair mattresses the best. The patient should be put on a couch for an hour each forenoon while the bed is aired and freshly made up.

Food is an important element in the treatment. Such patients very soon after treatment is begun need large quantities of easily digested food, but at first it is best to use skimmed milk alone, giving the first day a few ounces, say three, every two hours from seven in the morning till nine at night. Next day the dose may be increased to four ounces, and soon the patient should be given two or two and a half quarts of milk daily. Many patients do not like milk, or believe that it makes them bilious, but a tablespoonful of limewater in each glass will often overcome this, or, if need be, it may be predigested. No positive rule can be laid down as to how long milk is to be the only food. Some patients thrive on it, and can be kept upon it for several months, but the majority need some other food after two or three weeks. It is then well slowly to enlarge the diet. The seven o'clock milk may be replaced by a glass of cocoa and at 8.30 she may have a breakfast consisting of a soft-boiled or poached egg, bread and butter, fruit, and milk, and after a few days dinner, in the middle of the day, never at night, may be added. It should consist of soup, some hot meat, beef, lamb, chicken

or squab, vegetables, bread and butter, and some simple dessert. About a week later a light evening meal may be added. By this time the daily dietary consists of three meals, the midday one being quite large, and a glass of cocoa in the early morning and milk in the middle of the forenoon, middle of the afternoon, and at bedtime. The kind of bread used is important. Fresh bread and hot, moist rolls should never be taken by a neurasthenic. Bread a day old, toast, the crusty part of crisp rolls, or zwiebach are the best. Water is an important part of the diet, and as milk is withdrawn should be supplied in its place. Red meat should not be eaten more than once a day. Eggs may be eaten raw, poached, or boiled, but not fried. In fact, all fried food should be prohibited. Coffee and tea should be prohibited in the earlier period of the treatment. Later, one cup of coffee with cream may be allowed at breakfast. Alcohol in all forms is harmful to all neurasthenics, and should not be given in any dose at any time.

The next important element in treatment is *massage*. If a neurasthenic is put to bed and given no physical exercise, evil is bound to result. She will become constipated and digestion, even if fairly well performed before, will be disturbed. The great good of massage is that it gives the patient exercise, and the muscles work without any fatigue on the patient's part. Of course, the work done is nothing like so great as in active exercise, but there is no need that it should be so. Massage also does good in increasing the peripheral capillary circulation and possibly in actually squeezing poisonous products out of the muscles. The best time to give it varies in different patients, but the treatment should never begin or end within an hour of a meal. Some women who sleep badly are helped to sleep by it, and with them it should be given just before settling down for the night. For most patients about 3 P.M. is the best time. Whatever time is chosen, it should be the same hour every day, and not one day in the forenoon, the next in the afternoon, and the next at night. While the treatment is being given the patient should, during the first weeks, at any rate, remain quiet and not talk to or be talked to by the nurse. After the treatment is over she should remain quiet for one hour and not be disturbed for any reason. Some masseuses like to use cocoa oil or some other substance during massage. They are harmless, but of little if any benefit. There are a few women whom rubbing makes intensely nervous. Some have an abhorrence of being handled by anyone, and some do not dislike the treatment but get cold hands and feet and bluish lips after it. Oftentimes after a few days and by beginning with only a perfunctory rubbing abhorrence can be overcome. Rarely the treatment must be stopped altogether. At the beginning the treatment should not last more than half an hour, later on forty-five minutes to an hour. At first it should be very gentle, later more severe. Toward the middle of the course resistive movements should be added, and when the patient is up several hours a day she should begin light gymnastics. It is better to have massage given by some one other than the nurse, because she has enough to do in other ways.

*Electricity* is useful but not imperative. It, like massage, gives muscle exercise without work on the patient's part and has some good effect on the circulation. It is a mild stimulant to some patients, but this effect is largely, if not altogether, psychical. A few patients have such a nervous fear of it that it cannot be used to advantage. It should be given several hours after or before massage. A good way is to give it at eleven in the morning

and massage in the afternoon. An ordinary nurse's faradic battery is all that is required. The two electrodes should be freshly covered with absorbent cotton for each treatment, wet with warm water, placed only a few inches apart over the bodies of the muscles of the arms and legs, and, the slowly interrupted current being turned on, each muscle be made to contract three or four times. The electrodes should not be placed on the tendons or the bony prominences. Care must be taken not to use a current so strong as to cause pain. The entire body should be gone over in about forty-five minutes. Sometimes the patient is made to feel much better by ending the treatment by using the rapidly interrupted current to the neck and heels. A small electrode is put on the nape of the neck and an electrode large enough to cover both feet placed on the soles. The rapid current is then turned on for ten minutes.

*Isolation* is imperative in all severe cases, but not necessary in the milder ones. Mental rest is needed quite as much as physical, and a neurasthenic is injured by the anxious faces of near relations and the useless sympathetic talk of friends who too often discuss her troubles with her. The kind friend who tells her how well she is looking and that she will recover right away if she will only make an effort is a very disturbing factor. The ills of life and petty annoyances which to her are great burdens are brought constantly before her by family talks. Sometimes more serious troubles in the home life right themselves after a time by a judicious absence of talk and argument about them. Sometimes more wholesome marital relations, which have been lost, may be regained by the temporary separation of husband and wife. Sometimes people who care a great deal for each other react very badly on each other when one or the other is sick. For a shorter or longer time, then, it is best for the patient to see only the nurse, the doctor, and the masseuse. Notwithstanding the isolation, there is no need for the patient, as a rule, to lead a silent and entirely passive life. Soon the nurse should read to her for a little while each day. Later they may play games, and when she begins to get up she may read, write letters, see her relations and friends, sew, embroider, make baskets, model in clay, and so gradually resume her life.

At first she should have a warm sponge bath every day, preferably in the morning after the cocoa and before breakfast. Each extremity and the trunk should be bathed separately. Later, she can be given a warm and still later a cool plunge bath daily, and later she may bathe herself. In women the hair should be shampooed twice monthly. This is a little thing, but it gives comfort, and comfort hastens cure. All possible causes of so-called reflex irritation should be removed. Ocular troubles especially should be remedied. Local treatment of the genitalia, unless there be real disease present causing local symptoms, usually does more harm than good. Vaginal examination in young, unmarried women in the hope of finding some hypothetical cause for the disease is never justifiable. Such an examination is a great shock, and should never be made unless local symptoms imperatively demand it, and then it should be done under an anæsthetic.

*Drugs* play little part in the rest treatment. Sometimes insomnia is so great as to require an hypnotic. Veronal is probably the best, although sometimes trional or sulphonal does better. Hyoscine in doses of $\frac{1}{150}$ gr. at night is sometimes useful, but, with the frequent perversity of active and powerful drugs, it occasionally excites instead of quieting the patient. A warm pack is often of service. Opium and its derivatives are always to

be avoided. The old compound sumbul pill three times daily often quiets the nervous unrest. When there is much gastric atony ascending doses of nux vomica, beginning with 10 drops before meals, are useful. When intestinal indigestion and catarrh are present a pill of nitrate of silver, gr. $\frac{1}{12}$, immediately after meals is of great use. Constipation is often overcome by abdominal massage. When it fails the fluid extract of cascara in 15 minim (1 cc.) doses three times a day, or a single larger dose at night, is useful. Phosphate of soda before breakfast or a corresponding dose of the effervescent salt sometimes acts better. The combination of senna leaves stewed with prunes is homely but useful. Enemata are sometimes required. Many distressing abdominal symptoms are relieved by a binder.

Let me say one word more about the remedying of peripheral irritation in neurasthenia, and first as to the pelvic organs. Any serious disease should be treated in the same way as if the patient were not a neurasthenic. Surgical treatment of an infantile uterus will do no good. It is not a cause of the neurasthenia, but simply one of the signs of an ill-developed organism. Tears of the perineum and cervix should be repaired if they cause local symptoms, but no immediate amelioration of symptoms is to be expected. Even the wildest doctrinaires have ceased to remove normal ovaries. If the ovaries are seriously diseased and in themselves need surgical treatment, they must be removed, but such removal usually has but little effect on the neurasthenia and sometimes is followed by very serious nervous symptoms lasting months or even years. This, of course, refers to operations before the menopause. After the pelvic organs the eyes are the organs most treated in neurasthenia. Any disease of them should be treated. It is undoubtedly true that eye-strain in persons prone to neurasthenia may cause many annoying and even distressing symptoms which can be remedied by proper treatment. Whether a floating kidney should be operated upon depends upon the circumstances and the symptoms in each particular case.

How long should a rest treatment last? No definite time can be set, for it depends upon the progress. There certainly should be some improvement in six weeks. The good signs are increase in weight or, if the patient has been fat always, a hardening of the fat and firmness of the muscles, and improvement in the color of the skin and mucous membranes. The pasty, oily, dry skin becomes clear, a little pink, and the oiliness passes away. The tongue becomes clean and the breath ceases to be foul. The appetite improves, and, the strength returning, the patient wants to get out of bed and do things. When the patient begins to get out of bed it should at first be only for a half hour, then an hour, and every few days thereafter a longer time. She should gradually do more and more for herself and should be interested in something outside herself and outside the serious affairs of life, *i. e.*, she should have a hobby. She should also begin to go out of doors, and finally take up her duties again.

There is one type of woman whom a rest cure ruins. Through very wilfulness and indolence such women want to stay indefinitely in bed, not because they are sick, they are not, but because they enjoy it. They are lazy and selfish, and love the luxury of having doctors and nurses. They enjoy the importance of being ill, and are too empty headed to want to do anything in life except exist. They must be forced to get up—they never should have been put to bed—and slowly resume ordinary life. It is as hard, as Weir

Mitchell has said, to get some patients out of bed as it is to make others remain there. Each needs what the other wishes.

Recently there has arisen, or, rather, is now arising, an opinion among physicians that rest treatment is not as useful as many of us believe. The reason for this is not hard to discover. After it became popular it began to be used in many entirely unsuitable cases, and often was not carried out in proper detail. Sometimes the physician forgot that there is necessarily a psychic element in all treatment, and thought that all his duty was done when he mechanically set the machinery of treatment in order and then ceased to exercise any personal influence in the matter. Really the personality of the physician is a very important element in success. He needs to be a wise man, to know much, and to have great sympathy with the frailities of human nature. He must know when to be stern and when to be sympathetic, and, above all, he must never lose or, at any rate, show that he has lost interest in the case. It is sometimes dangerous to show too much interest in the patient. Another reason is that in therapeutics, as in everything else, the pendulum of opinion is apt to swing first to one extreme and then to another, never reaching a place of stable equilibrium. Hence to-day there is coming into vogue a work cure for neurasthenia. It has a place and a large place in the treatment of nervous people. It has long been used by many men as the last stage of the rest treatment (Weir Mitchell has so used it for years), and with many patients is a most excellent thing. Its fundamental idea is that much nervous disturbance depends upon the loss of interest in life and that work outside the things the patient has been in the habit of doing and along new lines will resurrect that interest and produce a saner, healthier view. All this is undoubtedly true.

The question at the outset, then, is, Does the patient need rest or work? The answer depends largely upon the physical strength or weakness of the patient and upon the character of the symptoms. When there is great neuromuscular weakness, when all physical effort causes rapid and great fatigue, when there is emaciation and serious digestive disturbance and any mental effort causes distress, the first, the imperative need is bed rest. On the other hand, when without much or any true physical weakness there is mere loss of the joy of living, mere boredom of life, with some, even great, nervous irritability and a little mental queerness, some doubt as to the affection of near relations, not very severe mental tire, and some but not severe gastric and intestinal disturbance, then work in the therapeutic sense is much more needed than rest. A total change in environment and the development of new interests are very necessary. Cases a little more severe need increased rest but not rest treatment. On the contrary, one hour of rest each afternoon is often quite sufficient. The important thing is to re-arouse interest in life, and this can best be done by inducing the patient to take up some new line of work, giving her at the same time much out-door exercise and compelling her to take enough food at regular times. Finally, there is a large class of neurasthenic patients who are not severely ill, but yet suffer a great deal and need only modified rest treatment. Removal from home is almost always necessary for cure. No absolute rules can be laid down for the conduct of every patient. The matter must be left to the wisdom and discretion of the attending physician.

In the last few years a great deal has been written concerning the so-called *psychic* treatment of functional nervous diseases, and claims have been

made that neurasthenics can be cured by such means. Indeed, the word *psychasthenia* is coming more and more in use to replace neurasthenia. Now, if neurasthenia be made to include all kinds of nervous symptoms and several types of mental aberration, if it means palsy of the will, and laziness and general nervousness, then certain cases of it can be cured by purely mental treatment. But the writer has not been discussing that sort of thing, but a serious and well-defined disease, the causes of which are deeply rooted in the patient. What part, then, can so-called psychotherapy play in its cure? Both much and little. No physician can help a neurasthenic if the patient doubts his skill or thinks he is treating her in a merely mechanical way without human interest. He must have her confidence to get good results. She must believe he can cure her or, at any rate, help her. This is, of course, purely a psychical matter dependent upon his personality. But when we come to the technical side of psychotherapy the question is different. For example, the use of *hypnotism* as a therapeutic means is in the writer's mature and deliberate opinion productive of more harm than good, not only in neurasthenia but in almost all diseases. It is a great shock to a woman to learn that anyone can make her unconscious and, so she believes and has learned by reading popular books and articles, subservient to another's will. If often repeated it certainly tends to weaken will power. Quite recently suggestion under hypnotism has been replaced by suggestion without hypnotism. This is really not such a mysterious process as some of the papers written about it would lead one to suppose. It really is nothing more than the acceptance on the patient's part of the truthfulness and accuracy of what the physician says and willingness to be led by him. Susceptibility to it indicates only ingenuousness and childlikeness of mind. The newest form of psychotherapy consists in an appeal to the religious sense of the patient. No harm and much good may and does come from this when properly and conservatively used, but unwisely and emotionally employed, and to the exclusion of proper medical treatment, it must do harm. It sometimes leads to very serious results because there are certain people who need not stimulation of the religious sense, but sedation. Dabbling in psychology, the occult, and the mysterious has injured a great many people and made not a few insane. It is wiser and safer for physician and priest each to keep within his own sphere. Somewhat allied to religious psychotherapy, but advised by men of a very different kind, is the treatment founded on the theory that neurasthenia and hysteria are based on the sexual function, and that if a patient be induced to make a confession of errors and sins in the long ago, she will be helped by the confession and speedily recover. No good need be expected from such a theory nor from practice based on it.

Neurasthenia in youths resulting from sexual abuse is not helped by the rest cure. As a rule, they are injured by it. Even though they seem to be physically very weak, it is far better to compel them to take out-door exercise, cold baths, the simplest of food and not too much of it, than to put them to bed, stuff them, and give them time to day-dream about sexual pleasures. Often the hard life of a ranch among men who will not sympathize too much with them is the best means to a cure. They always need encouragement, and a quiet sensible talk does them a great deal of good.

A great deal of neurasthenia would be prevented if people were taught how to live properly. The robust and healthy, hard fibered, and with good ancestry, can get through this world with little care and less training, but

those who come into the world handicapped need much of both. Although we suffer from the despotism of our ancestors, we can to a degree avoid their rule if we learn our own weakness and what we can do and what to avoid. So far environment may overcome heredity. The weak-fibered boy or girl can often escape neurasthenia if he or she be well environed and taught self-control and sensible habits of living; how much to work, how much to play, what responsibilities to assume and what to decline. Further, good training may strengthen a weakling so that as the years go by he can do more and better work and stand more and more stress.

## THE TRAUMATIC NEUROSES AND PSYCHOSES.

The use of steam and more recently of electricity in industrial life has created a new cause of disease, or, rather, has made an old cause, accidental injury, much more frequent. Traumatic neuroses and psychoses are not new diseases; it is probable that some of the slaves suffered from them at the building of the pyramids, but the tremendous development of machinery has increased the frequency of accidents and the horrors accompanying them. The rapid growth of the very modern belief in the responsibility of employers and the increase in the legal duties of common carriers toward their passengers and the general public have drawn great attention to these diseases and led to their close study. We shall not consider the effects of severe direct trauma to the brain, spinal cord, and nerves, but only the nervous and mental conditions resulting from accidents in which, although there may or may not have been injury to some other part of the body, the nervous system has escaped gross trauma. We shall exclude discussion of injury and fright as exciting causes of paralysis agitans, exophthalmic goitre, and Sydenham's chorea, and consider only traumatic neurasthenia, hysteria, and insanity not ascribable to serious local cerebral injury. We shall include the late results of concussion of the brain and strain of the lumbar muscles. Any discussion of treatment is unnecessary, because it is the same whether the illness be caused by trauma or not.

A great deal has been written and several theories propounded to explain the mechanism of the traumatic neuroses and psychoses. It is pretty generally accepted that they are in some way connected with disturbances in the central nervous system. Some metaphysical authors have assumed that mind exists independently of the brain, but works in association with it, the doctrine of parallelism, and attribute the nervous and mental disorders to psychic, as distinguished from cerebral, shock. Other writers hold that mind is merely a function of the brain, but that the brain is affected purely in its function without organic change, and still others believe that there are organic changes present but so minute that we cannot see them even under the microscope. But, although much has been written, we do not yet know what happens to and in the brain to produce traumatic neuroses and psychoses; we know nothing of their morbid anatomy. It may be assumed that whatever pathological lesion does exist is in the brain. There are probably other, secondary, lesions in the abdominal viscera in neurasthenia, but they are hypothetical. The theory that neurasthenia is caused by spinal concussion is not accepted by many authorities at the present time.

The possibility of fraud on the part of the person alleged to be ill makes

the study of the traumatic neuroses and psychoses more difficult than that of most diseases. In ordinary practice the question of malingering is rarely important; in illness following an accident it is always to be thought of. Another difficulty is due to the fact that some writers are, unconsciously of course, biassed in their opinions, because most of their work consists in examining claimants for defendant corporations or the clients of lawyers who have claims. Sometimes the bias is not altogether unconscious, and, indeed, might be called by another name. Apart from downright fraud, there may be exaggeration of really existing symptoms, and a pessimistic physician may scare the patient to his increased hurt. Indeed, it has been claimed that there are no traumatic neuroses or psychoses, but that the conditions so named are the direct and sole result of suggestion on the part of the physician treating or examining the patient. It is not, however, the prevalent opinion, and is not likely to become such. A last difficulty is the fact that there is no complex of symptoms pathognomonic of trauma as a cause. There is no clinical picture from which one can conclude, knowing nothing of the history of the case and judging only from the physical examination and the patient's account of his symptoms at the time of the examination, that the illness is caused, and only can be caused, by trauma. In every case other causes might have produced the same effect. We can only say that the shock of an accident can produce this condition and cannot produce that.

The accidents most liable to be followed by serious results are those in which the accompanying circumstances are horrifying and those which occur suddenly, unexpectedly, and without any forewarning. Accidents which cause very severe physical results very often are followed by less severe nervous results than others in which the physical injury is slight, and those occurring during sleep, and especially during drunkenness, are the least likely to be followed by serious so-called functional nervous or mental troubles.

*Traumatic neurasthenia* is probably the most frequent of all the nervous conditions which follow accidents. It may be mild and transitory or severe and permanent. Its symptomatology differs in no way from that of neurasthenia from any other exciting cause. A frequent history is about as follows: The patient has fallen from a car which started as she got off or on, or has been knocked about in a collision, or thrown to the ground from a wagon or cart. She is dazed for a moment, or even made unconscious for a short time. There are also some bruises, or, it may be, a broken rib, a cut scalp, or a broken arm. She goes, or is taken, home and finds herself much shaken up, but with no fear of any serious results. The physical injuries heal well, but she finds that there is something wrong with her. She sleeps badly, her appetite is capricious, and digestion poor. Attacks of cardiac palpitation occur and the peripheral circulation is sluggish. She may at times faint, or fear she is going to do so. She is downhearted, cries easily, is peevish and fretful. She was a useful and sensible housewife; now she is fit for nothing. She tires very easily, and has lost interest in everything. She may blame herself for this loss of interest and struggle against it, or she may become wholly selfish and demand to be treated better by her family. She gets the feeling of being a martyr and ill used. Power of mental concentration goes. Headache comes on, especially the curious occipital ache which patients often describe as a feeling of unbearable strain rather than

as pain. She becomes physically weak, and can scarcely drag herself around, and finally, it may be, is bedridden. The onset, instead of being slow, may be very acute and date from immediately after the accident. If she is a working woman or a workingman's wife, her illness often leads to great financial difficulties in the family, and brooding over this and comparing it with former times increase her illness. If the case is of a kind that leads to a lawsuit, the expectation of appearing in court, the interviews with lawyers, the examination by experts all add to the illness. Many patients are improved or, indeed, cured soon after a verdict, no matter what it may be, not because the patients were malingerers, but because the horror of the trial is over. Some cases are made more severe by the long delays which occur before legal settlement is reached, and sometimes among the poor, in cases in which there is no doubt of the genuineness of the symptoms, proper treatment cannot be carried out because of the lack of money. Some patients present great physical weakness and rapid mental fatigue with little or no emotional unbalance.

Some cases which are commonly classified as traumatic neurasthenia ought rather to be designated by the term general nervousness. The patients suffer from general nervous unrest rather than real weakness. They often describe their condition by saying they "feel in a hurry all over." They fidget and fret and cannot get anything done because they do not know where to begin. The condition is, of course, not serious, but it is annoying and may last a long time.

*Traumatic lumbago*, which logically should not be studied here because it is caused by direct muscular strain, and hence is neither a neurosis nor psychosis, may be, because it so frequently accompanies and, indeed, aggravates neurasthenia and hysteria. It really is the result of strain of the muscles and ligaments of the back caused by violent and involuntary muscular effort made to save one's self in falling or being thrown. Force directly applied, as by a blow or by striking an object, is a less important causal factor. The pain of traumatic lumbago must not be confounded with the painful points in the spine which occur in hysteria. The lumbago involves more or less of the whole mass of lumbar muscles on one or both sides, and is shown objectively by rigidity when the patient twists upon the spine, or bends to either side or backward. In a severe case the attitude is characteristic. The spine is held stiff and straight, or bent forward from the pelvis. The lumbar muscles are in more or less rigid spasm. The patient moves slowly, not only in walking, but in using the hands or moving the head. On rising from a chair he supports the back by placing the hand on the thigh. The gait is slow and hesitating, the feet being dragged on the ground or barely raised from it. In very severe cases the pain is so great that the patient cannot walk, and this may lead to a suspicion of palsy from spinal cord disease. But there is no true palsy; the bladder and rectum are under control; there is no anæsthesia; indeed, there are no signs of cord disease.

**Hysteria.**—Any one of the possible combinations of hysterical symptoms seen in non-traumatic cases may follow and be caused by trauma. No one combination proves a traumatic origin. It must never be forgotten that the presence of hysterical symptoms does not disprove the existence of organic cerebral or spinal disease. This association of hysteria and organic disease, whether it is a mere coincidence or whether, as is thought by some, a gross lesion may be an exciting cause of hysteria, is seen apart from trauma.

Thus, often in brain tumor, in women especially, in the early stages, hysterical symptoms, or symptoms taken to be hysterical, are so pronounced that the organic disease is entirely overlooked. It is especially important, for therapeutic and prognostic reasons, to carefully exclude organic disease before the diagnosis of hysteria is made. To mistakenly diagnose cerebellar disease as hysterical astasia-abasia might lead to serious though different evils to both patient and doctor.

The most frequent type of serious hysteria following an accident is that of palsy with anæsthesia, but any type may develop. The palsy may be monoplegic, hemiplegic, or paraplegic. Sometimes the symptoms remain unchanged throughout the course of the illness, sometimes new ones are added, until finally the patient presents or has presented all or almost all the symptoms of the disease. A hysterical fit or series of fits may begin the illness or appear during its course. Sometimes the patient complains of only one symptom, but in such a case great care must be used in diagnosis, because monosymptomatic hysteria is very rare. Thus, the tremor of fright may become permanent. For example, a woman, aged thirty-two years, was knocked down by a trolley car as she was crossing the street. She was unconscious for a few minutes and somewhat bruised, but no severe physical injury was discovered. On regaining consciousness, she began to tremble violently, and some months later the tremor was still present and was never absent except during sleep. She also complained of feeling scared all the time, and of anorexia, pain in the left breast, and general nervousness. The tremor was not that of paralysis agitans, multiple sclerosis, or alcoholism, nor was there any other symptom of any of these conditions. She had the very excessive knee-jerk found often in nervous and excited men and women (the knee-jerk that causes a sudden jerk of the whole body), but no ankle clonus. There was a little tenderness over the spine and pain on pressing the left breast, but not the right. Tremors of this kind are sometimes followed by, or are the first symptom of, paralysis agitans. Among the rarer symptoms are the hysterical breast, bleeding breast, and true hysterical insanity. More or less neurasthenia is present in a great many hysterical patients, although many neurasthenics are not hysterical.

A common history is as follows: After an accident, in which the patient has been shocked, and sometimes quite a little time after, the patient finds trouble in walking or using the hands. There is associated with this headache, especially in the occiput, backache, points of tenderness along the spine, and a general feeling of weakness. The motor disability may go on to complete inability to use the affected parts. The palsy, instead of slowly increasing, may come on suddenly immediately or quite a little time after the accident. On examination the ordinary signs of organic palsy are not found. There is no true ankle clonus, or spasticity of the knee-jerk, but there is frequently anæsthesia, it may be to slight touch only, or all sensibility may be absent. The anæsthesia may involve one entire side, the palsied one if there is hemiplegia, and stop abruptly at the middle line, or both legs, or be in scattered spots of more or less regular geometrical shape, but having no relation to the distribution of the sensory nerves, or involve the hand or foot alone, having a clear-cut circular boundary anywhere above the wrist or ankle.

Sometimes single symptoms are complained of or are so predominant that the patient pays little attention to others which medically may be more

important. For example, a woman was thrown to the floor of a car during the excitement following the blowing out of a fuse. She was taken to a hospital, where she was found to be dazed and confused, but not unconscious. She was bruised on the head and arms. After a few hours she went home by herself and went to bed. When seen by the writer, some months later, she complained only of loss of the sense of taste and smell, which she dated as arising after the accident, but could not remember how long after. Careful examination revealed no organic cause for the loss, and the general examination of the nervous system brought to light no other signs of disease, either functional or organic. More frequently anosmia and ageusia are merely part of a complex symptomatology. Thus, a woman, aged thirty years, was thrown from a car, striking her back and head. Immediately after the accident she had pain in the right arm and loss of use of the right arm and leg, and later lost feeling in both. When seen she had a hysterical right hemiplegia, curious jerking movements of the head coming on every few minutes, accompanied with spluttering speech and lasting a minute or two—it seemed to be a true tic—and loss of taste and smell. She claimed to have monocular diplopia in the right eye, but this was not subjected to any test. There was complete anæsthesia to touch, pain, and temperature on the right arm, leg, and trunk, and tactile anæsthesia on the right side of the face.

A bleeding breast in a man who had had a penetrating wound of the chest seemed to have been caused by some slight local trouble in the nipple rather than by hysteria. When examined he complained of pain at the seat of the old wound, saying that the pain caused by bending forward prevented him doing manual work. Many months after the accident he suddenly became numb in the right arm and leg. During the examination, on milking the left breast, or rather pinching the nipple, about a quarter of a dram of blood spurted out. He had no other sign of anything more than general nervousness. A jury gave him $5000 damages, but were said to be more influenced by the fact that he had actually sustained a penetrating wound of the chest than by his nervous complaints. Vomiting of blood, especially immediately after the accident, is much more frequent than bleeding from the breast. It may recur at intervals for some time, and is probably caused by rupture of some of the small veins of the œsophagus. It does not resemble blood arising from a hemorrhage in the stomach. Malingerers sometimes suck blood from cut gums to simulate this symptom.

The following is a case regarded as probably one of mixed hysteria and organic disease: A woman, aged thirty-five years, was thrown to the ground and dragged some distance as she was getting on a street car. She was dazed, but apparently not badly hurt, and soon continued on her journey. The same evening her physician found her in a state of nervous collapse. When examined by the writer, several weeks later, she was in bed and the movements in the right leg and arm were weak. She could and did move them, but not with any force. Further, on lifting the right arm there was a marked coarse tremor in it, and it soon fell, probably because, as she said, it became weary. There was tactile anæsthesia and analgesia on the right side. During the examination she had a violent cramp spasm of both legs, but more severe in the right, which lasted about two minutes. The feet were flexed strongly, the legs flexed on the thighs, the thighs on the abdomen. Severe pain was evident. Both knee-jerks were increased and ankle clonus

was present. She complained of transitory visual hallucinations, occurring especially at night and apparently in the half-waking state. A month later the hemiplegia had become complete and the other symptoms, except the hallucinations, which had ceased, remained the same.

**Mental Disturbances.**—Several types of *mental trouble* may follow accident. In fact, in a sense, there are always some mental symptoms associated with the nervous, because, even in neurasthenia, there is always rapid mental fatigue, an inability to do any continuous mental work, and oftentimes some, and sometimes very marked, decreased ability to form correct mental judgments, with disability or inability to decide any question. As to hysteria, it is a mental affection having physical symptoms, although it is not a true insanity. Under this heading, therefore, are considered the purely mental symptoms that may follow accident. The most frequent, probably, is mere mental *dulness*, resembling congenital stupidity. It may occur alone or be accompanied by the physical symptoms of neurasthenia. Alone it constitutes a true psychasthenia. There may be all degrees of severity. The man or woman who, before, was mentally quick, alert, and accurate in thought is now slow, dull, and inaccurate. The attention cannot be fixed for any length of time. There is marked mental inertia. The man who read much and high class literature now does not read at all or very little. The clerk who could rapidly add up a column of figures, calculate percentage quickly, almost automatically, now can scarcely do any arithmetic, not because he cannot do it if he tries hard enough, but because he cannot try hard enough for more than a few minutes. The foreman of a gang of laborers, who before could control them well and instantly give the proper orders, now overlooks more than he oversees, and himself recognizes his deficiencies. All grades of this condition occur, from the slightest to the apparent profound dementia in which mental power is lost entirely, for the time being, at least, and the patient must be cared for. The extreme cases are very rare, unless there has been some gross organic lesion resulting from direct and serious injury to the brain or skull, which, of course, puts them out of the class of psychoses. The milder cases are very common. Frequently it is not so much the direct result of the accident as a secondary consequence dependent upon the worry and grief resulting from inability to work. It may be unaccompanied by true physical weakness, although the mental inertia may render it impossible for the patient to make muscular effort for more than a few minutes. There is sometimes added to the mental dulness emotional depression, a condition of melancholy. This, too, may be slight or severe, and is increased by the distressing financial conditions so often consequent upon inability to work or to attend to the duties of life.

Let us now take up the *insanities* in the more limited and restricted sense of the word. What types of insanity can and what cannot be caused by the shock of accident? Of course, the accident is merely the exciting cause; the predisposing cause is in the man, else everyone would become insane after an accident that would make anyone insane. The predisposing causes are the soil, the exciting causes the seed which grows in it. One of the most frequent questions which comes up is whether a given case of *paresis* has resulted from an accident. Now, of course, the classical cause of paresis is the combination of alcohol, syphilis, and strain and stress of life. There is no doubt that, given these as already existing, and given

a concussion of the brain or a fracture of the skull, the brain injury may precipitate the occurrence of symptoms which did not exist before and would never have come into being had not the patient been subjected to some unusual stress. The question is rather different in those cases in which no gross injury to the skull or brain has occurred, but it appears that a severe shock to the nervous system can light up the disease. A patient with an already existing paresis, but in the early stage, may surely be made worse by an accident. In the later stages no harm is done because the mental powers are already too low to be influenced by shock. It is very important to determine whether the patient had the disease established before the accident. When a period of many months of good health has elapsed between the accident and the very beginning of the symptoms of paresis, and there has been no gross injury to the brain, the probabilities are that the two have no correlation.

When elderly people have sustained an accident the claim is often made that it has resulted in *senile dementia*. As a rule, elderly people do not stand sudden physical shock well, and frequently there is permanent loss of mental alertness in consequence of it. There is no doubt that occasionally it is the exciting cause of a true senile dementia. The cerebral arteries, although thickened, may be healthy enough to nourish the brain under ordinary circumstances, and the kidneys, although sclerosed, may also excrete fairly well and yet both fail, never to recover, under the stress of shock. There may be serious difficulty, however, in determining the causal relation of the accident in a given case. It is frequently not easy to discover what was the true mental state of the patient before.

*Hysterical insane* states may be caused by accidents, as they may be caused by shock of any kind. The melancholic condition spoken of above may, in a susceptible person, pass into a true melancholia. States of acute confusional insanity and dementia præcox also occur. Drunkards bear shock worse than any other class of people, and may have an acutely oncoming insanity in consequence. So much for the positive side. Now as to the negative. True congenital *paranoia* is never caused by accident. It is a purely developmental disease and needs no external exciting agent for its production. It is doubtful also if true *acute mania* is ever so produced. The cases of *epileptic insanity* usually follow some direct injury to the brain, although the convulsions may not occur for a long time after the accident, but there are usually some physical signs of the injury even at the beginning. This is not always the case, or rather at the beginning the physical signs may be slight. Thus, a man in a railroad car was struck on the side of the head with the metal end of a bell rope, which had snapped off when the car parted from the rest of the train. He was knocked down by the force of the blow and was senseless a few minutes. He recovered consciousness quickly and complained only of the pain in the bruise at the spot where he had been hit. The bruise quickly healed, but he continued to have pain in that region. Some months later he began to have general, not local, epileptiform convulsions. They were alleged by the railroad company to be hysterical, but his family physician claimed they were organic, and he was trephined at the seat of the injury and the inner table of the skull was found fractured, a spicule of bone pressing against the dura. There was a marked adhesive inflammation of the dura in the neighborhood. He had no recurrence of the fits for several months, and was then lost sight of.

In his case there were no mental symptoms, but only the convulsions. Whenever true epileptiform, not hysterical, fits follow an accident it is always well to hunt for a local cerebral trauma.

**Diagnosis.**—The diagnosis of accident as a cause of nervous and mental disease is difficult, because in so many cases the question arises, Is the patient a malingerer? There is no short and easy way to a determination of the question. Mental alertness, knowledge, and skill on the part of the examiner, and care in investigation will always uncover fraud. Bias on the examiner's part may lead to great injustice, and this is a very important matter. The man who believes that no attention should be paid to the mere complaints of the claimant, the merely subjective symptoms, and that a diagnosis should be based solely on the objective signs, will often reach a different conclusion from him who thinks the mere statement of the claimant should be accepted at its face value. The middle path is always the safest. Undoubtedly there are many fraudulent claims, and just as undoubtedly perfectly legitimate claims are sometimes opposed in ways that are not fair. Some defendants are as tricky as some claimants.

Before a correct diagnosis can be made all the important facts in the case must be known. Especially important are the state of health of the claimant just before the accident and the nature of the accident. When a merely trifling accident has occurred, unaccompanied by any cause for shock, it is doubtful if the accident is the cause of the illness. For example, a man, about thirty-three years of age, had undoubted hysteria, which he claimed was due to striking his head against the roof of a bus which gave a sudden jolt, because of an inequality in the road. The man made no complaint at the time, continued his journey, attended to his affairs for about a month, and then had a hysterical convulsion followed by persistent hysterical symptoms. It was found that he never had had any permanent occupation, never had kept at any work in any one place for more than a few weeks, had caused his family, who were in good circumstances, much trouble by his laziness and shiftlessness, and had always excused himself on the plea of illness, making hypochondriacal complaints all the time, while, as a matter of fact, he was well nourished and strong. The trifling nature of the accident, together with his temperament and character, made one disbelieve that the accident and the hysteria had anything to do with each other. Therefore, one important point is to determine whether the accident was or was not enough to have caused the illness.

The next question is to determine the presence or absence of other causes, for example, preëxisting physical disease. Thus, a man complained that a few days after being violently thrown from a trolley car and sustaining many bruises his left knee suddenly gave way under him and he could not walk. He claimed, and no doubt honestly believed, that it was caused by the fall. As a matter of fact, he had locomotor ataxia, and the knee trouble was due to a Charcot joint. Although he had very slight ataxia of motion and station, the knee-jerks were absent, Argyll-Robertson pupil was present, and he said he had had slight pains in his legs for several years. Of course, the degeneration of the spinal cord necessary to produce all these symptoms could not have taken place in a week, no matter how severe the injury and shock of the accident might have been. Another patient complained of neurasthenia, but on examination marked signs and symptoms of chronic and serious mitral valve disease were found quite sufficient to account for

her condition. The jury gave her a verdict, and very properly, because undoubtedly the shock of the accident had increased her sufferings. The question sometimes arises in the case of men, whether their illness is not really caused by alcohol or syphilis, but both conditions usually present quite definite objective signs.

How long a time may elapse between the accident and the illness and yet the illness be the result of the accident? No time can be arbitrarily set down. Many persons who at first suffer little, later develop very serious symptoms, but the time between the onset of symptoms and the accident is never very long. This, of course, applies only to the neuroses and psychoses and not to the late effects of serious physical injury, such as concussion or fracture of the skull. In both these conditions there may be apparent recovery, and a long time after symptoms may arise as a direct consequence of a disease process in, say, the meninges, the direct result of the primary trauma. External conditions may bring on severe and sudden symptoms in these cases of old and quiescent injury. For example, a severe concussion may make it impossible for a man to do work in the heat, although in a cool place he may work very well. Traumatic epilepsy may begin years after the injury to the skull.

Certain diseases may come on suddenly at the time of the accident, and yet not be caused by it. For example, an old man fell as he was getting off a car. The evidence was that it was motionless, but nobody could tell whether he had stumbled or not. When he was picked up he was unconscious. When consciousness returned he was paralyzed on the right side and aphasic. He was old, his arteries very hard, his urine contained tube casts and a trace of albumin, and was of low specific gravity. He had no local signs of fracture of the skull, and the writer's opinion was that he most probably fell as a result of an apoplexy. In cases similar to the above, in which death occurs and an autopsy is held, if there is no fracture of the skull and there is a hemorrhage from the striate artery, it is very strong evidence that the patient fell in an apoplexy. On the other hand, the shock of an accident may, itself, produce an apoplexy.

The differential diagnosis between senility and the effects of trauma is often difficult. Shock may produce a premature senility, and, given a man who presents the signs and symptoms of senility, the diagnosis must depend upon the history of his condition preceding the accident. If it be shown that, notwithstanding the existence of arteriosclerosis, the man was in good condition, and that dating from the accident mental and physical decay began, it is reasonable to suppose that the injury was the exciting cause.

Can an accident without severe physical injury to the head affect the *mental development* of a child? Now and again it is claimed that such an accident has caused imbecility. A nervous child is often dull and stupid and emotionally apathetic or unbalanced for some time after an accident in which there has been a large element of fright, but in the great majority of cases there is no permanent ill effect, the patient regaining emotional and mental equilibrium after a variable period. Permanent mental impairment may result in already pathologically high-strung children, but is very rare. When there has been serious concussion of the brain very severe and permanent effects may follow, but even in cases of concussion the natural resiliency of youth tends to bring about complete recovery. Slight concussion surely causes no permanent ill effects in healthy children.

Assuming that the patient is ill, and ill because of an accident, the next question is the exact nature of the illness. Is it a pure neurasthenia? If so, the patient will present more or fewer of the symptoms described of that disease. For our present purpose the most important thing is to exclude organic disease of the abdominal and thoracic viscera. This can only be done by careful and, it may be, repeated examination. Defendants sometimes claim in court that all the symptoms are subjective and that no one can tell how ill the claimant is. This is not altogether correct. A physician can tell much by the behavior, conduct, and appearance of a person as to whether he is ill or pretending. Although hysteria and neurasthenia are different and distinct diseases, they are often combined in one picture, or a patient largely neurasthenic may have a few hysterical symptoms.

The differential diagnosis between *hysteria* and *gross organic diseases* is important. A patient has convulsions, palsy, ataxia, or neurasthenia. Are the symptoms hysterical or organic? The classical hysterical fit is not hard to recognize. The absence of the initial cry, the unbitten tongue, the retention of control of the sphincters, with, after a fit is over, the passage of a large quantity of clear urine, the preservation of consciousness or rather its alteration, an aberration of consciousness without its abolition, the state in which the patient knows more or less well what is going on but is unable to communicate with anyone, the slow onset of the fit, permitting the patient to reach a place of safety, the absence of momentary rigidity before the clonic spasm comes on, the dramatic element, the seeming purpose in the movements, the long continuance, the absence of deep sleep after, the presence of signs of hysteria between the attacks, all point to hysteria.

The diagnosis of hysterical palsy may be difficult. This may be paraplegia, monoplegia, or hemiplegia. Authorities differ as to which is the more frequent. In the writer's experience brachial monoplegia, or rather hemiplegia, with much greater involvement of the arm than of the leg, has occurred most frequently, and paraplegia least. Although at first sight it may look like organic disease, the picture is never perfect. The condition of the reflexes is important. Hysteria never causes a loss of the knee-jerk. As a rule, it is much increased, but it does not possess the qualities of the increase present in disease of the motor cortex or lateral tract. There is not the spasticity, and when the patellar tendon is tapped the whole leg is jerked, often the whole body, and the patient becomes excited. True ankle or patellar clonus is never present. There may be a few irregular and unequal to-and-fro movements of the foot when the examination is made, but true clonus, with the regularly periodical, equal, and continuous vibration of the foot, never occurs. The plantar jerk may be absent or very marked even in an anæsthetic foot; the true Babinski reflex is never present. On stroking the sole the whole foot or leg may be forcibly withdrawn and the toes may fly in any direction, but there is never the slow, deliberate extension and lateral separation of the toes, as is often seen in organic disease.

*Anæsthesia* is an important diagnostic symptom. It is sometimes claimed to be non-existent because the patient did not know of its existence till examined, when, it is alleged, it has been brought into being by the suggestion of the physician. Now, anæsthesia may be due to suggestion, but the explanation of many cases is that it is so slight as not to have made it difficult for the patient to feel objects. A patient may have tactile anæsthesia sufficient to make him insensitive to a wisp of cotton or the light touch of a

match stick, and yet feel perfectly when an object is pressed against or put in his hand. There are then multitudinous points of contact which may reinforce each other. A pretended analgesia can easily be discovered by sudden and unexpected pricks with a sharp instrument. This should not be done during the formal sensory examination, but before it and while the patient is off guard. Examiners sometimes forget that there may be preservation of the pain sense with tactile anæsthesia. To jab a patient with a pin and because he says it hurts conclude he necessarily has no tactile anæsthesia is to form a hasty judgment on insufficient evidence. The Mannkopff test, dependent upon the fact that a sudden painful stimulus frequently increases the pulse rate, is of some value in testing sensibility to pain, but sudden fright from fear of pain may do the same, and the amount of pain necessary to affect the pulse varies with the individual.

*Contractures* are frequent and may be lasting. The only muscular wasting is that from disuse. It is always diffuse, and there is never any change in the electrical reactions. When marked local wasting is present there is organic disease. It is necessary to be on guard not to mistake the general wasting of disease for trophic changes. The contractures, especially if the patient is very thin, may make the joints, notably the knee and elbow, so prominent as to create a suspicion of trophic joint trouble. Such changes do not occur, but long-continued immobility of the limb may cause a kind of adhesive inflammation around the joint and in the muscles, causing stiffness in it and shortening of the muscles. In such a case the contracture does not relax under the most profound anæsthesia. The hysterical joint, which curiously is usually the knee, is swollen and painful, but shows no other sign of inflammation. There is no local heat, redness, or swollen tortuous veins. It comes on suddenly and may pass away as suddenly. Hysterical joints are sometimes claimed to be cases of tuberculous arthritis due to direct injury, and hence the importance of a differential diagnosis.

The *palsy* in hysteria may be either flaccid or rigid. In organic hemiplegia the patient in walking lifts the leg from the hip and swings it; in hysteria the leg is dragged. Hoover has studied a sign of great value in differentiating the two. In organic hemiplegia, if the patient, lying supine, attempts to lift the palsied leg, the other is pressed down against the bed, If he lifts the non-palsied leg, the other is pressed down. This does not occur in hysteria. There is never, in hysteria, a true palsy of the bladder and rectum, but temporary retention is common.

The *visual symptoms* are interesting. Contraction of the fields of vision with reversal, although common, is not pathognomonic. It occurs in brain tumor, fracture of the spine, and, indeed, not a few other diseases. A gradual contraction of the fields during the examination is characteristic rather of neurasthenia and states of exhaustion, but tubular vision apparently occurs only in hysteria. Monocular diplopia is hysterical unless due to dislocation of the lens or some other local trouble, but it must be remembered that unintelligent people often complain of monocular diplopia when really they mean they have dimmed or blurred vision in one eye. Hysterical blindness can only be diagnosed by exclusion, and must be separated from mind blindness. Since the patient avoids obstacles in walking, he may be accused of hysteria or even fraud. The condition is always accompanied by other signs of brain disease.

*Hysterical ataxia* is differentiated by the absence of the signs which accompany organic ataxia. Occasionally it is mistaken for cerebellar disease, the examiner being misled by the fact, common in the latter disease, that the patient moves the legs well in bed.

*Hysterical mutism* must be separated from aphasia and fraud. As a rule, the presence of other symptoms makes the diagnosis quite easy. The hysterical mute, as a rule, manifestly makes no real attempt to speak; the aphasic, no matter how speechless, always tries. In armies soldiers are cured of pretended mutism by the inhalation of chloroform. When mutism is the only symptom, careful watching will soon settle the question.

**Prognosis.**—The danger to life is very small in any of the traumatic neuroses and psychoses. Pure neurasthenia never in and by itself leads to death, nor does it seem to increase very much the patient's susceptibility to infection if he is so situated that he can take proper care of himself. There is some dispute as to whether hysteria ever ends fatally. A few cases have been reported, but the probability of there having been an error in diagnosis is quite great. Myasthenia gravis and Landry's paralysis are sometimes mistaken for it, especially in the early stages, and occasionally patients who have had severe hysterical paralysis for years develop sclerosis of the cord and die from its secondary consequence. Whether a larger percentage develop such lesions than can be accounted for by mere arithmetical chance, or whether long-contiuned hysteria does predispose to sclerosis, is an unsettled question. If it does, then hysteria may indirectly be a fatal disease. Personally, if a hysterical patient should die under my care, unless from some intercurrent disease, my belief would be that there had been an error in diagnosis.

The outlook as to the duration of any given case of traumatic hysteria or neurasthenia depends on many factors. The age of the patient, the severity of the illness, the length of time it has already existed, the ability of the patient to obtain proper treatment, are all important elements. Although some cases of hysteria are recovered from suddenly and the cure of neurasthenia is never sudden, the latter disease probably has a somewhat higher permanent recovery rate than the former, because hysterical patients are very prone to relapse when subjected to any stress or even without it. In general, old age is of bad prognostic import, because when the arteries begin to thicken and the kidneys are sclerosed the patient's power of resistance to shock decreases. Usually youth increases the chance of recovery, but hysteria may so upset the mental and nervous equilibrium as to lead to permanent disease or a life of semi-invalidism. This is, of course, especially true in those patients in whom there is a strong hereditary or congenital predisposition to hysteria. One of the most important elements in prognosis is the ability of the patient to get proper treatment early in the illness, especially if it be neurasthenia. It is rarely safe to venture any prophecy as to how long illness will last. It is never safe to do so unless the physician knows all about the case. The duration of traumatic lumbago is very variable. Enough pain usually lasts after the acute stage is over to give the patient some trouble for a long time.

The prognosis of abnormal mental states is the same as when not caused by shock, except that, in general, in any mental trouble directly resulting from an external cause the patient has a better chance for recovery, other things being equal, than if it had arisen without such external cause.

# CHAPTER XIX.

## MIGRAINE. NEURALGIA. PROFESSIONAL SPASMS. OCCUPATION NEUROSES. TETANY.

By SMITH ELY JELLIFFE, M.D.

### THE MIGRAINES.

Under this head will be discussed a series of characteristic phenomena, which are among the oldest affections known in literature, and which are here brought together arbitrarily, with the full recognition that divergent opinions are held as to their essential relationships.

### OPHTHALMIC MIGRAINE.

**Synonyms.**—Sick headache; megrims; hemicrania; bilious headache; Hemikranie, Migräne. Latin, cephalalgia biliosa; suffusio dimidians; migraine; emicrania.

**Definition.**—At the very outset the difficulty of defining this protean affection is met with. Inasmuch as, with other biological phenomena, migraine may be a simple or an extremely complex condition, shading at times insensibly into other disease pictures, it is possible only to attempt a definition of the more classical types of the affection, neglecting for the time the numerous related forms which may be bound up with other morbid manifestations. Taking these limitations into consideration, migraine may be defined as a periodical abnormal state in which the patient suffers from a peculiar oppressive pain in the head, unilateral or bilateral, localized or general, which develops very gradually from heaviness to dulness, to pain that is splitting, and is accompanied or more often preceded by characteristic visual signs, such as scotomata, flying specks, or partial blindness. Chilliness, depression and sensory disturbances, particularly in the stomach, and which may lead to nausea or vomiting, are also usually present. An attack may be terminated, after a few moments, by vomiting, or it may persist hours or even days. After a variable length of time, usually following a heavy sleep, the patient regains his previous condition of well-being.

**History.**—A rich literature exists on the subject of migraine. A heritage of the rich and the poor, the great and the small alike, it has numbered among its sufferers many of the master minds of all times, and no disorder can vie with it in richness of description from medical writers who have been themselves subject to its vagaries.

Hippocrates seems to have missed it, but since his time its more stable features have been slowly crystallizing out. Even at the present time it cannot be said that we have accurate knowledge concerning its intimate

nature. Although, historically considered, Aretæus is credited with having given the first description of migraine, in Celsus one finds a description which, while not corresponding in many details with what is now understood to be migraine, is, nevertheless, very suggestive.

Aretæus made a very definite separation of various kinds of headaches which previously had been in a chaotic state, and he is entitled to be known as the first to set off the group of heterocrania, or migraine.

Galen not only gives us the name for the affection, but essays a description and an explanation as well: "How constantly do we see the head attacked with pain when yellow bile is contained in the stomach; as also the pain forthwith ceasing when the bile has been vomited." He also gives his adherence to the doctrine of consensus, or sympathy between the head and the stomach, thus outlining the crude beginnings of the humoral (autotoxic) and sympathetic (reflex) hypothesis.

Cælius Aurelianus has given an excellent description of a migraine attack, noting for the first time that the Greeks called it hemicrania, and was the first to note a preponderance in the female sex, and mentions cold, heat, and insomnia as causes.

The stomach is made to bear its etiological burden all through the Byzantine period, Serapion and others faithfully transcribing the doctrines of Galen. In the long interval between, up to the appearance of Tissot's classical monograph (1784), many writers busied themselves with this affection. Fernel adopted the very modern view that headache was not a disease but a symptom, and maintained that migraine had its seat in the brain, but he followed the Galenic humoral doctrines in his explanation. Lepois, in the seventeenth century, gave his own personal experiences through fourteen years, and called attention to the fact that the usual after effects of vomiting and sopor might come on without the presence of the headache. Wepfer seems to have more clearly appreciated the eye symptoms.

Tissot is credited with having given the first classic on migraine. His description is that migraine is a severe pain, which occupies only one-half of the head, principally the brow, the eye and the temple. This characteristic—to attack one side of the head alone—is sufficient to distinguish it from ordinary headache. He later showed that the pain might be bilateral, and, although not like typical migraine, may yet show the other signs, *i. e.*, sleepiness, etc. He was among the first to speak of the hereditary features of the affection. He noted its periodicity, ascribed it as coming primarily from the stomach, and treated it largely by diet and laxatives.

Tissot's description remained authoritative up to the appearance of Liveing's monograph, "On Megrim, Sick Headache, and Some Allied Disorders" (1873), although in the interim the symptomatology was becoming richer and the case analyses more exhaustive. Thus, Vater, Hennicke, and Heberden made observations upon the scotomata. Plenck, Parry, Wollaston drew from personal experiences the picture of half-sided blindness. Schönlein and Romberg introduced the neuralgic theories, while Du Bois-Reymond, influenced by the newer work of Claude Bernard, developed the hypothesis of arterial spasm which Möllendorf controverted, and postulated a sympathetic paralysis, both of which views were conciliated by Jaccoud and by Eulenberg (1867), who described angiotonic and angioparalytic conditions.

**Etiology.**—A great number of hypotheses have been advanced which consider it as either arising on a healthy substratum, on a condition engrafted upon a constitutional anomaly of structure or of function, or as nothing but an essential anatomical constitutional anomaly. The older neuralgic theories are of historical interest alone.

The hypothesis which maintains that the disorder arises autochthonously, affecting that portion of the brain which is the seat of the constitutional anomaly and acts here locally through the specific cause of the attack has been termed by Spitzner the *central theory*, and is supported by such authors as Romberg, Leubuscher, Liveing, Jackson, Gowers, Möbius, and Spitzner. Another group of hypotheses may be termed the *centripetal theories*. These assume that some cause, toxic, reflex, etc., originates in a peripheral organ, which, in turn, acting through the circulation or the sensory nerves, or both, affects the brain cortex and gives rise to irritative processes, and this causes the attack.

The *vasomotor* hypothesis assumes that the essential feature is a vasomotor spasm followed by vasomotor dilatation, thus causing anæmia and hyperæmia, which act on the nervous tissues and cause the headache.

The *toxic hypotheses* have many advocates, but all suggest different poisons, and these do not seem able ever to cause the condition when given under non-vital conditions.

The followers of the *reflex hypotheses* successively call on the uterus, ovaries, foreskin, adherent clitoris, gastroptosis, enteroptosis, the eyes, the ears, the nose, the throat, etc. Each specialist here advocates his own particular organ as the essential factor, just as each chemist claims his own chemical, uric acid, xanthin, paraxanthin, etc., to be the real cause.

The combined hypotheses next come into review, the toxin reflex, the vasotoxic, the vasotoxic central theories, etc. The organs which have been declared at fault have been the vasomotor centres, the sympathetic, the gastro-intestinal canal, secretory, excretory, ductless glands, organs of generation, male and female, the various organs of sense, the eye, ear, nose, tongue, the muscles of the body and the general metabolism of the entire body, the brain in general, the cortex, the pons, the thalamus. Many authors pick out several factors; others confine themselves to one. Some faddists say it is eye strain; others ear strain; others masturbation; while others blame corsets, tight collars, or the wearing of high hats.

Spitzner[1] has recently subjected these ideas to a thorough criticism, and has advocated the hypothesis that migraine is due to a constitutional anatomical defect, namely, an absolute or relative stenosis of the foramen of Monro. This is the fundamental substratum of the migraine constitution. The occasional causing of a passive or active hyperæmia of the brain leads to a hyperæmia of the choroid plexus. This causes a more or less complete plugging of the foramen of Munro, with the production of an increase of pressure in one or both of the ventricles. The increased pressure on the vessels causes more distention and more pressure on the walls of the ventricles; a vicious cycle is established, and the migraine mounts to its height, until the pressure is relieved either by a spontaneous reduction or by the sudden let down in tension due to a shock reaction—such as occurs in the act of vomiting, from the use of various vasodilators, etc.

[1] *Ueber Migraine*, 1907.

The general hypothesis of a vasomotor disturbance seems to account for most of the facts, and the general position here taken is that such disturbance may be conditioned by a host of causes. The view advocated then admits that a certain amount of fact exists in practically all of the hypotheses, but maintains that a one-sided mode of interpretation is inadmissible.

**Relation of Epilepsy to Migraine.**—This has been widely discussed. To those who look upon a word diagnosis of many dissimilar conditions as symbols of entities, such a discussion is, perhaps, pertinent, even if on an illogical basis, but when the standpoint is maintained that there are many epilepsies and many migraines, and that both words may stand for things at times very much related and at other times not related at all, discussion is more or less fruitless. To assume that a migraine attack, which may undoubtedly originate as a result of a simple attack of constipation, has any relation whatever to an epileptic attack, which may be due to a splinter of bone in the brain, or to general paresis, or to a gumma, or to a miliary tubercle, or to a chronic gliosis, or to a multiple sclerosis, etc., is sheer nonsense; but it is not impossible to understand that a profound change in the character of the cerebral circulation, which may be due to transitory causes, and which may give rise to an attack of migraine, may also, in rare instances, be an exciting cause of a motor disturbance sufficient to set free an epileptiform convulsion. In such a sense the subject may be discussed. An increasingly greater number of atypical and borderland conditions are coming into review, and in the more recent analyses convulsive attacks indistinguishable from the hysterical and psychasthenic motor reactions are being described. Until a clearer conception is gained of the mutual relations of cerebral circulation and motor discharge it is folly to attempt to review the interesting, though fallacious, suggestions of Féré, Möbius, and others, who claim a necessary relation between migraine and epilepsy. The coexistence of migraine attacks and of epileptic attacks is not uncommon, but recent search has not shown that migraine is more common among epileptics than among people at large (Spratling). From the standpoint also of organic brain disease as a factor in the causation of a migraine the discussion is even more relevant.

**Abortive Attacks.**—It is difficult to be certain of the character of many conditions in patients who suffer from migraine, as there exists an involuntary tendency to drag every symptom within the net of this one great malady. Experience shows, however, that sufferers from migraine possess a keen insight into these undeveloped or abortive attacks, and after many years of close observation they are able to recognize them with considerable precision. Incomplete or abortive attacks may be said to be the rule rather than the exception, and attempts to classify the disorder according to the number of symptoms present offer no help in the understanding of the complete picture.

Möbius suggests that the parents of patients suffering from migraine with scotomata often have suffered from migraine without scotomata, but he also speaks of the reverse as happening. The extreme prevalence of migraine makes many of our conceptions regarding its necessary hereditary nature very dubious, and the extreme variability of the individual attacks in the same patient makes general hereditary features extremely improbable. It is by no means infrequent that many patients will show at one time or

another almost every symptom mentioned in the voluminous literature of migraine. Thus, one patient under personal observation had about two attacks weekly for a year. He then went two years without a single attack, and he then had several severe ones with aphasia and psychical symptoms, interspersed with abortive attacks, with hardly any two alike. He was a veritable museum of migraine attacks in the fifteen years that he was under observation.

Many families are known in which both parents have been sufferers from chronic migraine for years, and yet none of the children, now in some instances over forty years of age, have ever had an attack. In one family seven children, and in another five, have never had any attacks, and yet two generations of ancestors have suffered. The high percentage of incidence makes it almost impossible to calculate a hereditary factor. Again, it may be borne in mind that, like the epilepsies, there are many kinds, so with the migraines they are undoubtedly many. Some are due to hereditary anomalies, while others have nothing whatever to do with anything of an hereditary character. Thus, one can speak of migraines that are probably hereditary and others that are not.

The commonest abortive attacks are those that begin in the classical manner, with chilliness, perhaps with pinched face and cold extremities. The patient then has the scotomata and wretchedness, depression and apprehension, and then while waiting for the headache the patient notices that it does not come, and, although many may still have heaviness and a sense of discomfort, the feeling of relief is sufficient to make them feel well.

Others have added the sensation of pricking in the fingers, numbness in the hand or arm, or other sensory disturbances without the headaches. In some the entire attack will consist of a disturbed painful sense of discomfort, without sensory symptoms, scotomata, or headache, but they feel sick at the stomach, and have an attack of what they term "biliousness," which clears up after vomiting. This feeling will recur with sufficient frequency, and at times be combined with such other symptoms of a migraine attack, in its varying aspects, as to stamp the whole process as a variant of a true attack. Isolated attacks of vomiting, as the sole expression of a migraine, are known.

Attacks of scotomata occur alone, without antecedent distress, and no after-effects are noted. These are most uncommon, and historically it may be noted that Parry and Airy had such attacks. It is highly probable that the majority of patients who have had many migraine attacks will have had some of this nature. Attacks of scotomata and vomiting occur without headache. In many, on the contrary, headache is the only symptom.

Some patients have attacks of hemiparæsthesia, without any other symptoms of migraine. Such generally occur at night, and usually follow severe mental exertion; in one patient under observation a severe ordeal in playing a difficult piece of music will bring on such an attack without other signs. This patient's severe attacks are very extreme, being associated with hemi-œdema, hemiparesis, hemi-anæsthesia and marked hysteromaniacal outbursts.

Under the heading of equivalents, Liveing speaks of stomach attacks associated with some of the vascular phenomena of migraine; glossal spasms are also mentioned by him. Attacks of giddiness, vertigo, intestinal colic, mental anxiety and depression which occur at periodic times in partial asso-

ciation with migraine symptoms, are also noted as equivalents. There is need of further study of these isolated phenomena associated with vasomotor disturbances.

Attempts have been made to determine the relative frequency of migraine attacks with and without the *visual signs*. These are not over reliable, because of the vast preponderance of abortive attacks over those of the complete classical type. It may be recalled that many authors have attempted to create a special type of eye migraines which are autonomous, but the gradations are so numerous when different individuals and differing attacks in the same individual are considered that such a view receives little support.

Möbius expresses the opinion that the percentage of visual accompaniments with the attacks is usually overstated. His statistics show 130 cases, with 14 visual aura. In Liveing's 60 patients, 37 suffered from scotomata. Gowers says that the cases are about half and half, with and without eye signs. Galezowski maintains that the visual aura migraines appear later in life, thirty to fifty years, than ordinary migraines.

It is difficult to state an individual position, the results of personal inquiries having been so diverse. Close questioning has revealed the fact that at some time or other in the course the majority have had visual symptoms, and it is not improbable that the usual statistics are largely derived from studies of too few attacks, *i. e.*, largely from the severer attacks only. Some notes on individual histories are of interest. Several patients have kept fairly accurate records of their migraine attacks for several years. One shows 168 attacks in a period of about ten years; of these, about 100 were abortive attacks, the vast majority of which, 60 per cent., consisted of scotomata alone. Of the 68 remaining attacks, about 50 per cent. were ordinary hemicrania, lateral or bilateral, without scotomata, the others ophthalmic migraine, usually unilateral and with scotomata. Not one of the attacks was ever accompanied by vomiting. Two were associated with aphasia, fifteen with sensory tactile associations; there were five or six attacks of hemiparæsthesia, one in the day-time, the rest at night. Spasms of the orbicularis were a common accompaniment. Every attack sufficiently severe to require an analgesic was promptly relieved by from 5 to 10 grains of either antipyrine, acetanilide, or phenacetin.

Similar results in about a dozen other cases show records of from 50 to 200 attacks. It is rare that a patient suffering from migraine of any severity goes through life without some one or more of the sensory phenomena, and notably the scotomata. But a great many individuals have their attacks, both mild and severe, while asleep, and may never know of the occurrence of scotomata. Many of these patients, who have given no history of scotomata, have had frequent night attacks with sore eyeballs and sore spots on the scalp, two symptoms of frequent, although not necessary, relationship with other sensory symptoms. Féré's[1] interesting case, of the dreaming of seeing a fire, is in point in this connection.

**Classical Migraine.—Early Symptoms.**—These may be termed precursors to a full attack of migraine, or they may constitute the symptoms of an abortive attack. The most striking are a sense of heaviness, with yawning, chilliness, dizziness, or depression, motor twitching, even sharp

[1] *Revue de Méd*, 1903, p. 127.

spasms, closure of the eyelids, sensory phenomena, chiefly paræsthesia, occasionally anæsthesia, and affections of the eyes or other sensory organs, ringing in the ears, blowing, whistling, modifications of taste, of smell, of touch, etc. The temporal arteries are often smaller, and the saliva diminished.

The premonitory signs, which show a great degree of variability in different individuals, and also in different attacks in the same individual, may be felt several minutes before the attack, in some rare instances even days. This is frequently the case in women in whom the onset of the menstrual function seems to bear some relation to the attack. The ordinary depression felt at this time is a thing apart from this special type of depression that pervades them. At times such attacks of depression and anxiety, combined with a sense of chilliness and dizziness in the head, will constitute the entire picture of an abortive attack. Many attacks come unheralded.

Many patients having attacks at night find themselves tired, and heavy, with sore spots on the scalp in the morning. Experience tells them of an attack during sleep, which is often accompanied by unpleasant dreams, even nightmares, with excruciatingly painful sensations in the head. Möbius relates a case in which the patient dreamed of having swallowed a rabbit, which ate its way out through the stomach wall. After this unpleasant dream the patient had a severe migraine on awakening.

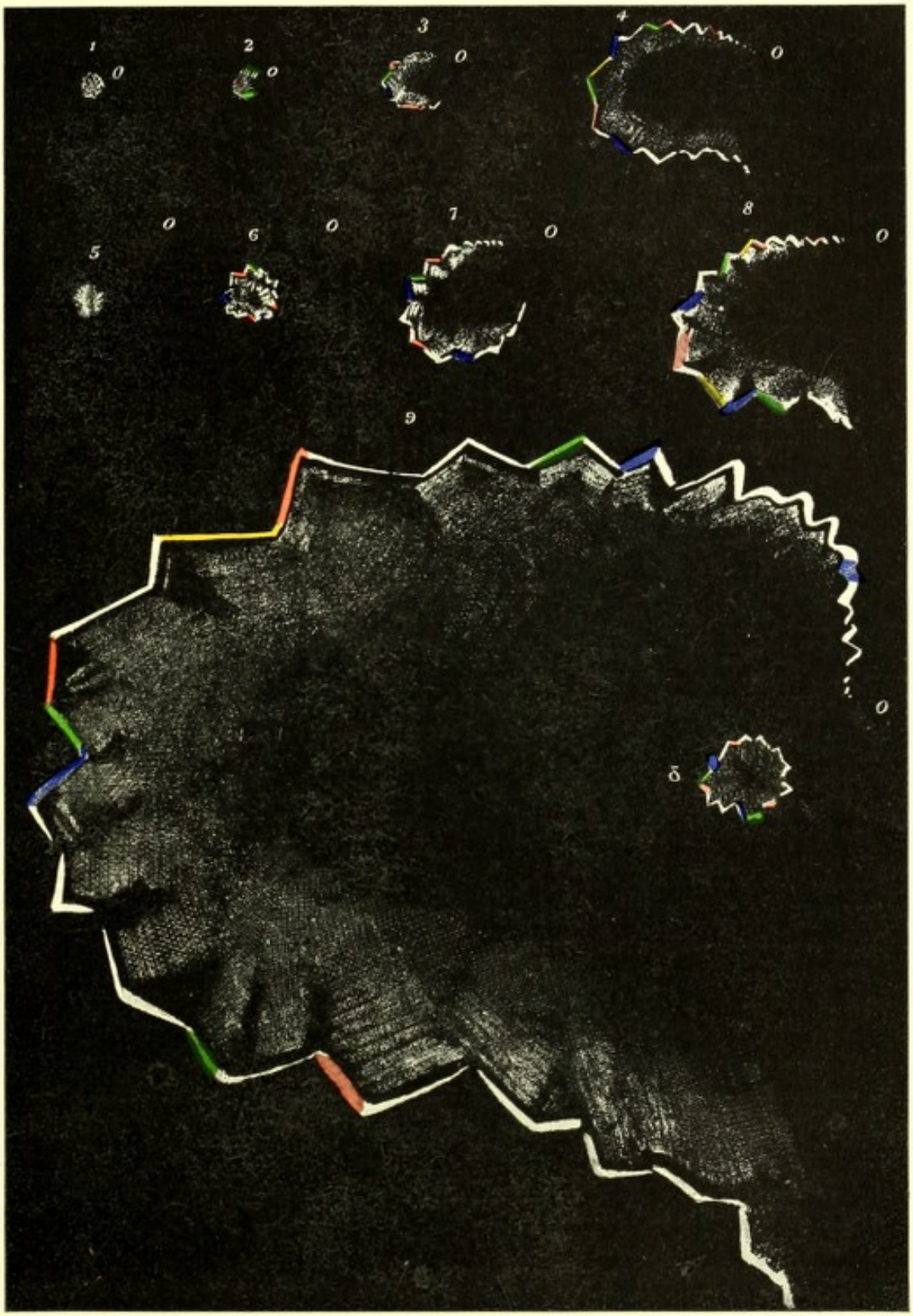
In all probability premonitory symptoms of some type are invariably present; when thought to be absent it is because the patient has overlooked them, either by reason of the mild character, or because of naturally poor powers of observation. Many patients, who have had headaches for years, have never noticed their one-sided localization, or the well-known fortification spectra, until their attention has been directed specifically to them. Many patients will deny ever having had zig-zags of light, etc., until shown Airy's pictures, when they remember having seen such phenomena. It is because of such poor observation that many cases of true migraine are overlooked, which fact lends further support to the personal belief that this disorder is very much more prevalent than is usually supposed.

**Sensory Symptoms.**—In the more classical attacks the patients have preliminary sensory symptoms. These are spoken of by Möbius in the sense of an aura, but as the essential relation of the migraines to the epilepsies is at least far from proved, the term aura, if used in a sense comparable to an epileptic aura, is objectionable; if one chooses to regard it as an early symptom in a migraine or in an epilepsy, well and good. If the term aura be used as, for instance, the term "fever" is used, there can be no objection, but if by aura is meant a restricted phenomena essentially related to an epileptic aura, the term should be eliminated.

A sense of coldness and chilliness is one of the commonest sensations. This is usually general, and is associated with a pale countenance, goose flesh, perhaps clammy hands, and a sense of misery. Cases are known, and are by no means uncommon, in which the chilliness has been one-sided, and is accompanied by other phenomena involving the entire half of the body, including the face, of the same side. Yawning is a common early sign.

Unilateral paræsthesia is not an uncommon early sensory sign. Many patients note a tingling or numbness in the fingers of one hand; this may spread up the arm, and in rare instances general unilateral paræsthesia of a very uncomfortable nature may be present. In some instances such

# PLATE XXIII




From Dr. Hubert Airy's Paper, On a Distinct Form of Transient Hemiopsia, Philosophical Transactions, 1870, p. 247.

Figs. 1 to 4.—Early stages of Fortification Spectra as seen in dark. O = sight point.
Figs. 5 to 8.—Similar series, beginning lower.
Fig. 9.—Fully developed. $\vartheta$ = secondary attack within.

unilateral paræsthesia has constituted the sole symptoms of an attack, save for the heaviness and usual discomfort. Occurring at night, such attacks are often extremely wearing, keeping the patient awake. Photophobia, flow of tears, strange sounds, tinnitus, peculiar odors, queer tastes, peppery or flat, may be noted.

Anæsthesia is less often observed, largely because of the negative character of the symptom. When involving the face or mouth it is complained of. Anæsthesia frequently follows the tingling of the early paræsthetic disturbances. Franz[1] has shown that there is a very evident decrease in the pain threshold, especially after the headache has set in.

The *visual phenomena* are the most striking, and hence held to be of the most frequent occurrence. The ease of observation in part accounts for the usually accepted opinion that they are the commonest of the early symptoms. Very few individuals have been subjected to a careful sensory analysis. If more were investigated, it is probable that other slight sensory signs would be found to be equally prevalent and as evanescent. The visual signs have been described by many writers, and many illustrations have been made showing their chief characteristics. The extreme uniformity of their general character is striking, as well as the variations of the same pattern.

As a rule, the patient notices a slight blurring of his vision if reading, or a slight flicker of light located in one eye, to one side of the centre. Closer observation reveals either a slight cloudy spot, which seems to follow the eye in reading, cutting out the after-images a letter or so from the point of the centre of vision. The slight subjective sense of difficulty in reading may precede the discovery of a scintillating spot, which becomes visible on closing the eyes. Little by little this spot spreads out, usually in a crescent-like fashion. General statistics are thus far unavailable, but personal experience has shown that the majority of these scotomata have begun in the left eye, are situated to the left of the middle line, with the convexity of the crescentic border to the left. As the crescent gradually grows larger, the difficulty in seeing clearly becomes more marked, especially on the periphery of the visual field. For most, the scotomata is in constant motion, flashing in its spectral zig-zag fashion, thus causing the classical name "fortification spectrum," from the play of colors, and the fortress-like "ins and outs" of the outline.

After a variable time, from five to twenty minutes, the scotomata gradually subside, or suddenly disappear, to be followed by the headache. Not infrequently the headache never comes, and the preliminary sensory phenomena of chilliness, heaviness, and scotomata constitute an abortive attack. A description of the scotomata of migraine might fill a volume. The classic of Liveing reproduces the excellent illustration of Airy's, which is here shown.

Occasionally the right half of the field is involved. Sometimes it is the upper half, one of Möbius' patients saying that everybody seemed headless; occasionally, it is the lower. In rare instances the patient complains of total blindness, *i. e.*, central scotomata. Berbez reports an interesting case of ring-like scotomata—the patient, on looking at his watch, could see only the central pin where the hands were united; the figures on the dial were all obscured by the scintillating scotomata.

[1] *Amer. Jour. of Physiology*, 1905.

These scotomata are usually bilateral phenomena, they may begin in one eye before appearing in the other, and be somewhat different in the two eyes, and may disappear in one eye sooner than the other. Scotomata limited to one eye are probably rare.

The point of view of Möbius, that the term scotoma should be applied to migraine and the word amaurosis reserved for a different class of phenomena, is sound, since real blindness is probably never present in any case. There is dimness of vision, but not blindness.

The retinal changes during the time of the occurrence of these scotomata are uncertain. Blanching of the papillæ has been observed by some (Galezowski); pulration of the retinal arteries with dilatation by others. Personal experience has shown similar dilatation in a few cases, but, as a rule, a normal fundus is found. The picture seen will depend upon the stage of the attack and upon its severity.

Pupillary dilatation occasionally occurs. Slight irregularity of the pupils during a severe attack of an ophthalmic migraine, dilatation being usual on the affected side, is not unusual. Bilateral pupillary contraction is the rule in the stage of the headache.

During the onset of the fortification spectra, it not infrequently happens that mild motor phenomena occur in the eyelid of the side to be affected. The eyelid droops a particle, and Gowers and others report double vision, interpretable as a sign of paresis in an ocular muscle. As these phenomena are not rare in the severe types of migraine, called by some ophthalmoplegic migraine, and which others, Möbius and Stirling, for instance, claim are not to be classed with the migraines, their consideration will be deferred.

**Motor Disturbances.**—*Speech.*—This may be considered as both a motor and sensory phenomenon, for the most frequent type of change is sensory aphasia in most instances. Anarthrias are known, especially in the ophthalmoplegic variety, but for ophthalmic migraine the type of aphasia observed is very characteristic. As described by Charcot, it is an intermittent, halting aphasia. At one moment the patient can get the right word, at the next he cannot. He stumbles on a word; uses madame for monsieur, etc. In Liveing's cases, 15 out of 60 had speech disturbances; one, on hearing clock bells, was unable to inquire what they were. Féré cites the case of a coachman who forgot where he was going to drive his passengers; Berbez, a like case in which a pedestrian lost his way, as he could not read the street signs understandingly. Gowers speaks of a case of word deafness. Cases of agraphia are also known. Möbius reports a case with typical scintillating scotomata at one time on the right side, and at another on the left. When the patient suffered from a right-sided scotoma he had sensory aphasic signs, but they were not present when the scotoma was on the left side.

Other observers have noted the same phenomenon, while contradictory observations are also recorded. The speech disturbance sometimes resembles a paraphasia, the patient using a jumble of words. In a personal case the patient could not sing a well-known tune correctly, his sense of musical values having been interfered with.

The onset of the aphasic disturbance may vary greatly. It is usually temporary, persisting at times not over a few minutes, again persisting a few hours. It frequently antedates the headache, or is coincident with it. In a recently reported case of Meige the aphasia persisted as long as the headache, and disappeared, as a rule, when that disappeared. The patient

showed a loss of ability to say certain words and a tendency to the employment of incorrect words. There was no anarthria.

Charcot attempted to explain these dysarthrias or aphasias as due to a vessel spasm in the region of the sylvian artery, and Meige follows him. The symptoms in one of Meige's patients were closely similar to those of a case reported by the writer.[1]

*Paralytic Phenomena.*—Attention has already been called to the rare occurrence of hemiparesis, which may even involve the facial muscles. Up to the present time no instances of crossed hemiplegic type have been found in the literature. This is of interest in connection with the hypothesis of the bulbar origin of migraine, especially of the ophthalmoplegic variety.

*Cerebellar Symptoms.*—Oppenheim has called attention to a cerebellar hemicrania in a patient in whom every attack of migraine was accompanied by typical cerebellar symptoms. The patient was uncertain in his gait, walked like a drunken man, was dizzy, and had the sensation that the body or individual parts of it were doubled. The sense of equilibrium was disturbed in each attack.

Dizziness and loss of the sense of equilibrium are not infrequent, but such a complete syndrome has been described only by Oppenheim.

**Headache.**—This is the most common feature, and exhibits a great amount of variability as to location, quality, intensity, and duration. In the more classical attacks the headache begins on the average about fifteen to thirty minutes after the appearance of the scotomata or other sensory phenomena. It frequently begins on one side, and may remain so or become bilateral. As a rule, it is frontal, or occupies the vertex, but may involve the temporal regions or the occiput, sometimes as low down as the neck. Gowers' experience points to the parietal region as being oftenest affected, and usually over a small area. Henschen, in 123 patients, shows the pain to have located 110 times in the forehead, 100 times in the parietal region, and 54 times in the occiput. There is usually pain over the eyes, and the eyeballs are usually painful to pressure. In a few instances pressure over the malar bones is painful, and occasionally there is a well-marked jaw ache.

Statistics of the percentage of location are difficult to present, since the individual will have all the different varieties. Thus, in a case already cited, in which the abortive attacks were so frequent, the headaches comparatively rare, the strictly unilateral headaches were only 5 per cent. of the entire number. In others the hemicranic type runs much higher. In Henschen's records of 123 cases, 56 had one-sided attacks, in 67 both sides were involved. In Liveing's 61 patients, 17 had one-sided attacks, in 7 the attacks were variable, while in 34 both sides were involved. Möbius and others note that the headache often begins on the side opposite to the beginning of the sensory aura. Personal studies do not confirm Möbius' statement. Averages one way or another do not occur if a summary of all attacks is attempted instead of picking out those which are most vividly present in the memory of the patient. It does seem, however, as first noted by Liveing, that one-sided sensory symptoms are oftener accompanied by one-sided than by bilateral headaches. With bilateral sensory phenomena, scotomata, etc., bilateral pains are the commonest.

In many attacks the pains are limited to the eyes, the feeling of soreness

[1] *New York Med. Jour.*, 1906, lxxxiii, 33.

of the eyeballs being so very marked that it is painful to move them. Pain in the neck may also cause the desire to hold the neck rigid.

The character of the pain defies analysis, since descriptive phrases are used in such various ways by different observers. In some attacks the head simply feels slightly sore, or heavy, or dull, or thick; "like a block of wood," is a frequent expression. "Filled with sawdust," one patient says. Again, the pain is agonizing, impossible to describe. Some patients shriek with the pain, become hysterical, and roll about the floor, grasping the head between the hands, wishing to beat their brains out. Between these extremes numberless gradients are found among different individuals, and in different attacks in the same individual. Nearly all patients will say that the severe pains are throbbing or thumping, usually indicating great pressure from within or without; as Möbius has said, "some patients think the head will burst, others that it is being squeezed in a vise." Descriptions of bursting are more common. The pain is an all-pervading one, gradually mounting to a maximum, then running along continuously without any break, with, at all times, sudden accessions, especially on movement, if one leans over, or is forced to sudden exertion. In but the rarest instances is it described as lancinating in quality. It is the type of pain apparently seen in cerebral tumor, in acute hydrocephalus, in cerebrospinal meningitis, and allied to the pain of opium poisoning, or of sea-sick headache; all pointing in the direction of a modification of intracerebral pressure, at times an increase, or it may be a decrease, either of which may cause severe pain. Occasionally the phenomenon of a bilateral headache with marked predominance of one-sided pain will be observed.

The severity of the pain may be conditioned by a number of factors. Movement uniformly increases it. Bending over becomes impossible. The first movement on lying down is usually accompanied by a sudden rise in severity, but this gradually subsides. The taking of alcohol usually increases the severity of the pain, as does also the use of tobacco. Eating, if possible, may help somewhat, but usually augments the pain, and is avoided. Strong sensory impressions invariably increase the pain. Noises of various kinds often aggravate the pain tremendously and cause certain patients marked distress. The "Fourth of July" invariably drives several migrainous patients to some quiet spot in the country, free from crackers and bombs. Möbius notes that the rage of migrainous parents directed toward their noisy children often resembles a pathological hatred. Strong light is invariably avoided, because of its tendency to increase the pain. The movements of the eyeball and attempts at visual accommodation cause an increase in the pain.

Psychical effort is often impossible; in milder attacks the awakening of a strong mental stimulus may make one forget the pain. Möbius says that his attacks, usually light ones, are frequently forgotten during an interesting visit to the hospital, to be once more prominent afterward. The writer has frequently begun a lecture with a severe migraine to find it almost forgotten until the close, when it reappeared, usually with renewed vigor.

The movements of straining at stool, and vomiting, coughing, etc., invariably cause a rapid and sharp rise in the severity of the pain. Sensory stimuli may have an unpleasant effect on the psyche. Thus, certain odors cause distress; the smell of cooking acts much as it does on shipboard—it

accelerates vomiting. Certain skin phenomena, such as painful sore spots, are frequent after the headaches.

In certain personal experiments with drugs the following have invariably increased the headache within a few moments: A few whiffs of chloroform or of ether, adrenalin by mouth, digitalis, strophanthin, and ergot. Drugs that raise the blood pressure, in general, increase the pain when taken, especially at the beginning of the headache.

The headache may clear away very suddenly after an attack of vomiting, or it may pass without vomiting; in some it fades away gradually. It may last a few minutes, a few hours, or a few days. Some cases of what Möbius chooses to call *status hemicranicus* are recorded.

**Vasomotor Disturbances.**—Practically all attacks of migraine are accompanied by visible vasomotor disturbances. The battle of the adherents of the vasoconstrictor *vs.* the vasodilator hypothesis lends point to this observation. In most cases vasoconstrictor phenomena (coldness, paleness, goose-flesh, etc.) precede, to be followed later by vasodilator changes. Thomas and Cornu both point this out as a result of their experiences. Thomas contributes a statistical study of 107 cases in support of the early pallor, small pulse, and coldness, which pass over into the phenomena of warmth, red, flushed face and skin, and full pulse. The period of initial constriction may be unnoticed by reason of its transitory character. In some instances this initial vasoconstriction may be very marked and give rise to the phenomena of localized cyanosis, even advancing to the picture of the constriction phase of the Raynaud disease type.

In the same manner the secondary vasomotor dilatation may pass the bounds ordinarily observed and lead to localized œdema, to the erythromelalgia type, or, exceptionally, to hemorrhagic phenomena in the conjunctiva, ocular tissues, or even in the walls of the stomach.

**Secretions.**—Alteration in secretory functions are frequently observed early or late in the attack. Reference has been made to the excess of secretion of tears as a frequent precursor. Vomiting of frothy mucus, liquid diarrhœa, increase of sweat, coryza (Calmeil), or incessant salivation (Liveing, Tissot) are common phenomena.

The changes in urinary secretion have attracted careful attention and given rise to many hypotheses. The early vasoconstriction of the periphery, coldness, lack of secretion, of perspiration, etc., account in a purely mechanical way for the increase of urinary secretion in the early stages, and also for most of the phenomena which have been interpreted so one-sidedly by Haig and others. Metabolic studies by Bioglio lead him to exclude the general hypothesis that fundamental disturbances of metabolism are responsible for the migraine. He was unable to show constant changes, and although it is not possible to exclude metabolic disorders as a primary cause for some of the changes in the intracerebral circulation, yet the causal relation remains as yet unproved.

**Trophic Disturbances.**—These have been reported by several observers. Cornu says that nearly all of his cases of migraine show facial assymetry, and facial atrophy is recorded. These instances are largely coincidences, and are not necessary attributes of the migraine. A facial atrophy which could be interpretable only on the basis of a migrainous disturbance of the vasomotor apparatus is very problematical, and certainly Cornu's results are not confirmed by others. Loss of weight in the severe rapidly recurrent

cases is due to disturbances in general nutrition due to gastric, rather than to other causes. Herpes is a not infrequent accompaniment of some cases, but the recognition of its infectious nature successfully disposes of its essential relationship to migraine.

**Psychical Disturbances.**—These have been noted by many observers, Liveing being one of the first to point out the relationship of disturbed psychical states to the attacks of migraine. Möbius, however, takes the position that such psychical disturbances are largely due to the pain, and are epiphenomena, as it were, in many instances being related to hysteria. Möbius' position, however, is not tenable in view of the many observations pointing in other directions.

In the vast majority of migraine attacks there are no mental changes, either before, during, or after the attacks. Mild depression, hopelessness, despondency with clear consciousness, are frequent mental states. With very severe pains Möbius admits the clouding of consciousness, and is not sure that the severe stuporous states are not due to pain as well. Mingazzini, on the other hand, believes there is justification in erecting a special group, which he has termed the hemicranic dysphrenias (disfrenie emicraniche), and he distinguishes a transitory and a more permanent variety. Recent observers are practically in accord in showing that severe mental disturbances varying in character and intensity may be part of a migraine attack.

Guidi has amplified these observations by reporting the history of a number of cases in which the patients suffered in the day or hours before the onset, in a much more decided manner than the feelings of anxiety or of depression described by Liveing. Thus Guidi calls attention to grave alterations in the psychical state of a number of his patients. In one the entire character of the personality would change preceding the attack. A patient who had always been calm, reserved, quiet, and modest, suddenly became much agitated, was forward, noisy, and loquacious, and told salacious stories, which was far from his usual behavior. While in health a spare eater, preceding an attack he suddenly became very hungry, and hankered especially for starchy foods. During the attack the patient had glycosuria, which disappeared later.

With the onset of pain the picture is less clear, yet there is little doubt that many patients suffer from profound psychical disturbances, which arise independently of the pain. One such case which is typical, under personal observation, would be interpreted by Möbius, and rightly so, as one in which the pain is the first link in a hysterical reaction. But there are other cases which do not belong to this group. Mingazzini's hemicranic dysphrenias may be cited as examples, in part, at least. In others severe disturbances have occurred, such as states of anxiety, rising to actual anguish (Charcot); phobias of inability to perform acts (Cornu-Charcot); terror (Liveing, Féré, Kraft-Ebing); hallucinations of sight (phosphenes, colored lights, animals) and hearing, with mental confusion (Forli, Mingazzini); maniacal excitement (Mingazzini, Jelliffe); and stupor, unconsciousness (many authors).

Liveing reports that 25 per cent. of his cases showed psychical symptoms. The Italian observers record fewer, but it appears that at least from 10 to 15 per cent. of the cases of grave hemicrania show some distinct mental disturbance in some one or more of their attacks which is more significant than the usual depression which is so universal.

**Symptomatic Migraines.**—The occurrence of migraine-like attacks accompanying, or due to, definite disease condition, notably organic disease of the brain, is well known. The association of migraine with gout and malarial affections has been noted. So far as gout as an etiological factor is concerned, Möbius is inclined to see nothing more than a coincidence; while, as for malaria, he holds it to cause an orbital neuralgia, not a migraine. As for the latter, it seems clear that the well-known effects of malarial infection on bloodvessel tonus are entirely sufficient to cause a typical migraine attack. It is known that attacks of migraine may be very frequent during the continuance of a malarial infection. Such may disappear for months after quinine therapy, and then reappear at the time of a later malarial infection.

Migraine-like attacks are not infrequent in cerebral tumor; they may appear periodically, as in cases fully reported by Abercrombie and Möbius, or they may be continuous and distinguishable with great difficulty from the pain of tumor, as in cases reported by Wernicke, who has said that such attacks may be quite readily confused with those more typical of tumor. In tumors, however, vomiting brings little or no relief; quiet gives less relief, and the fluctuation in the intensity of pain is less prominent. A primary onset of migraine-like attacks in adult life should always awaken the suspicion of an organic brain lesion.

Oppenheim has called particular attention to the occurrence of migraine-like attacks in the onset of tabes; Möbius is inclined to think it a rare combination, and regards such either as a pure coincidence or a migraine-like neuralgia. In general paresis, migraine-like attacks may be an initial symptom. Migraine attacks are not infrequent throughout the early stages of the disease, but the anatomical correlations are still hypothetical.

**Diagnosis.**—The difficulties appear in the consideration of ordinary headaches and in neurasthenic headaches; in distinguishing between the scotomata of migraine and other scotomata; the paræsthesia of migraine and other paræsthesias; the aphasia, the vomiting, etc., as seen in migraine, and the same as due to other causes. In most individuals abortive and incomplete attacks are the rule, and it is often extremely difficult to determine the precise significance of these abortive attacks particularly.

Möbius has suggested that the problem is not only whether the case is one of migraine or not, but whether it is migraine alone, and not something additional. This author's contention that migraine is hereditary and begins in youth, would seem to make it a simple matter, but clinical experience shows that real migraines do appear in later years, apart from other affections, and as for the heredity factor, the extreme prevalence of the affection makes it difficult to accurately weigh this factor. If to heredity we strictly apply the Mendelian criterion, that the absence of the disorder in either progenitor negatives its appearance in the descendant, then the Möbius doctrine falls; if, however, by going into the collaterals, as is so frequently done, then almost every disease having a large incidence may be proved to be hereditary.

The periodic recurrence is a difficult criterion. There is usually no difficulty in diagnosing the classical attacks from simple headache, but at times such a differentiation is impossible. Many chronic sufferers from migraine know well their real attacks, are able to distinguish abortive

attacks, and also have headache of an entirely different nature. The simplest test separating abortive migraines from simple headache is the occurrence of sensory phenomena, other than pain, which have their main seat in vasomotor disturbances. It is on this account we would ally the severe headaches following the use of alcohol, ether, chloroform, opium, or related drugs to the migraines, rather than to simple headaches. The headaches of neurasthenia, anæmia, syphilis, lead poisoning, nasal sinus involvement, supra-orbital neuralgia, nephritis, eye strain, glaucoma, etc., should present little difficulty.

**Treatment.**—The treatment of the migraine attack is, for the most part, fairly satisfactory. There are few patients for whom some relief cannot be obtained, both with reference to the diminution in the number of attacks, and to the mitigation of the severity of the attacks themselves.

The migraine habit, constitution, or liability—call it what one will—exists in very varying degrees; in some a very slight disturbance is sufficient to set free those forces which culminate in an attack; for others it requires a very much greater mal-adjustment. If the general reflex vascular hypothesis be taken as a tentative explanation, it is very readily understood why the taking away of various forms of peripheral irritation may result in eliminating one or more, and in certain instances all, of the causes which set the migraine reaction in operation.

It is folly to shut one's eyes to the very evident clinical fact that certain migraines are relieved, if not entirely wiped away, by the correction of some peripheral disorder, sometimes more than one, which has had definite action on the nervous system. Just what the differential relation may have been between the severity of the irritant and the mildness of the attack is impossible to judge, but certainly the relief from eye-strain, from diseased turbinates, from adenoids, from constipation, from dysmenorrhœa, from a number of minor yet definite peripheral irritations, will relieve a certain number of patients. Perhaps they are the very slight ones, perhaps not; we are not yet in a position to say. One should, therefore, eliminate at the outset such of these structural defects as are shown to have some influence on the nervous system. In denying any possibility to these influences in the causation of migraine one errs almost as badly as when maintaining some one of them to be the only element in the case, as faddists are doing and always have done.

Gastro-intestinal factors are closely analogous to those just mentioned. In the minds of most clinicians, and certainly as generalized in the feelings of most of those afflicted, it is in the stomach, liver, or intestines that the main seat of the trouble is to be sought. The gastro-intestinal factor is undoubted in many cases; it may be exclusively gastric or colonic; perverted chemism, perverted bacterial action (primary or secondary factors, no one can yet say). As to the significance of chemical features, resulting from altered gastric secretions or from toxic bacterial products, we are entirely in the dark. It is certain that none of the products which have been held responsible as auto-intoxicants are universal causes. At any rate, the general features of gastro-intestinal hygiene should be carried out. Constipation is to be avoided and such diet taken as experience has shown is individually applicable. Excesses in certain articles of diet are held by many as exciting causes; in many, such empirical feelings should be respected; the patient often knows himself better than does the physician.

In some, excessive carbohydrate intake acts disastrously; in others, wine, whisky, or gin. In one patient under observation the taking of even minute quantities of gin invariably causes an attack. The history of inability to eat fatty food, particularly sausages, is not infrequent. The significance of the nightmares and headaches after mince pie, cake, or cheese should not be idly dismissed.

In rarer instances, one notes that certain auditory stimuli may bring on a migraine. To attend certain fatiguing and thrilling operas is followed in some by migraine attacks.

For such attacks conditioned by recognized outside factors, such as malaria, gout, lead poisoning, etc., it is evident that these should receive attention.

If the varying elements mentioned have any real relation, it is evident why such a variety of measures will be of help to a few, and why so many more will be worthless for many but useful for some. Medication between attacks is largely useless, save naturally in the symptomatic migraines. General medication, for no definite purpose but just in hopes that it may do some good, as iodides, bromides, strychnine, etc., is senseless. If definite factors are found that need correction, and can be so modified by drugs in the desired direction, then they will prove useful. Thus iodides will undoubtedly help many presenile arteriosclerotic migraines; bromides are useful for sleepless and irritable conditions which provide a good foundation for the nervous instability that permits an attack; laxatives are called for if persistent constipation bears any causal relationship.

Complicated systems of diet have been devised. Usually such are more prolific in engendering semi-invalidism than useful for migraine. Here and there a patient derives benefit from a strict dietary régime, but unless there are real reasons why a patient should not eat red meat, or tomatoes, or other sundry articles, as determined by actual experience and under repeated experimental trials, in order to eliminate faddist's errors, the patient is better off without a diet card. The reasons sought for are not those contained in many treatises on dietetics, in which mediæval notions concerning differences in red meat and white meat, vegetables growing under the ground and those above the ground, are foolishly perpetuated. The only satisfactory manner to attack the metabolic problem is to carry out a complete metabolism analysis. Haphazard attacks here and there lead only to premature and insecure judgments.

Complete formulas for attacking excessive bacterial putrefaction are applicable only when it is proved that such excessive bacterial action exists and has a relation to the migraine. The hypothesis cannot be excluded ex cathedra, but it remains unproved for most cases, and of doubtful applicability in a few.

The avoidance of alcohol and tobacco, while advisable, is so only relatively. The individual's reaction to all influences should be rigidly estimated before those usually self-evident restrictions are imposed in the name of health.

In certain individuals a change of occupation may be absolutely necessary, but here again one must be wisely conservative, and not consign all migraine patients to an outdoor life, especially when outdoor workers are by no means exempt and ought to be clerks. The character of the work is to be borne in mind. The elements of haste, of pressure, and of lack of leisure are to be thought of in this connection.

For the treatment of the attack itself one finds that a like fitting of remedies to the individual is called for. In the initial phase of vasoconstriction a number of vasodilators are of service, although their action is extremely unequal. The nitrites and nitrates have been employed for years, and usually with a fair degree of success if the dosage and individual member of the group be correctly chosen with reference to the severity of the attack. A mixture is of greatest value; nitroglycerin and erythrol tetranitrate give the best combination, for following the very evanescent and powerful action of the former, the more prolonged and steady action of the latter maintains the effect. The slowly acting nitrates are practically useless. Nature's readjustment, vasodilatation by vomiting, etc., has already reduced the cerebral pressure, and the stage has passed when the dilating remedies might be useful. It is practically only in the vasoconstriction stage that the nitrites are worth much; and in many they are inefficient, the reasons, therefore, being as yet unappreciated. Given too late, they overdo the dilatation and increase the difficulty.

The analgesic vasodilators have come to occupy the front rank. The precise significance of each must be appreciated in order to obtain the best results. Solubility, time of absorption, slight differences in the chemical formula and in action, continuance of effect with minimum by-effects, are all to be studied. The list is a long one and is constantly on the increase. Antipyrine, acetanilide, phenacetin, and the related salicylic acid (aspirin, etc.) compounds are the chief members. It is to be remembered that while their general action is closely related, there are specific differences in the working of each, and the measure of success that one has in mastering the majority of migraines depends upon a knowledge of these factors. Antipyrine, by reason of its rapid solubility and quick action, occupies an important place, but is not always applicable. Acetanilide, alone or in combination with other analgesics of related type (salicylic acid derivatives), bromides, and caffeine are also valuable. The dosage should be graded according to the usual severity of the attacks. Tolerance is established in the quickly recurring attacks, and changes must be made. It is not yet certain what part is played by the respective analgesic and vasodilatation actions of this group. They have robbed migraine of most of its terrors, and tended to diminish the use of morphine and its derivatives very markedly.

Caffeine is a much overrated drug. In the abortive attacks and in the morning remains of a migraine it is useful; but for a full-fledged attack it is not efficient. Similarly, bromides alone, chloral, and other widely used drugs are valuable only in mild attacks. They should be used in preference to other more potent remedies, which should be reserved for the severer attacks, in order that one's therapeutic measures may more correctly approximate the needs of each individual occasion.

The use of aconite and cannabis indica is more restricted now that really efficient analgesics are known. Aconite is rarely called for, while cannabis indica or cannabis americana has a limited, although no less definite, place. In attacks associated with much mental depression the addition of cannabis is often useful. The often experienced inefficacy of this latter remedy is largely due to its extreme variability. Great care is therefore to be exercised in the selection of a proper preparation. Tablet preparations are usually worthless. This is equally true of the volatile

nitrite preparations. Opium, or its main derivative morphine, should be used only as a last resort. It is rarely really needed.

Lying down in a quiet, darkened room—a brisk saline laxative taken as early as possible, the patient being undressed and well covered—these are essential in the severe exhausting attacks. A very hot bath often aids very materially in restoring the patient to comparative freshness. Cold is to be avoided.

The greatest folly of all is to treat all patients and every attack alike.

**Ophthalmoplegic Migraine.**—Attention has been called to the fact that in the ordinary attack of ophthalmic migraine there may occur various sensory or motor phenomena, among which anæsthesias or paralyses are the most marked. These sensory and motor changes are extremely diverse when the entire range of the migraine symptomatology is brought into review, but there is one symptom grouping which, by reason of its comparative frequency and close similarity, was set apart from others occurring in this affection and named by Charcot ophthalmoplegic migraine, in order to distinguish it from its more classical relation. It consists in a paresis or a paralysis of one or more muscles of the eye, innervated chiefly by the oculomotorius, which comes on either following or during a migraine attack.

Inasmuch as oculomotor pareses or paralyses may occur from a great diversity of causes, apart from a migraine, and may appear periodically, it has been held by many that the term ophthalmoplegic migraine has no particular right to exist, but the evidence is too great to eliminate migraine as a competent producing cause for these periodic oculomotor paralyses.

**Etiology.**—Whether heredity plays any greater part here than in migraine in general is difficult to decide. The present attitude is to restrict the influence of heredity in migraine; certainly there is very little evidence of direct transmission of ophthalmoplegic migraine as such. The most striking personal case was devoid of any migraine heredity in any direct branches. Yet if there is any truth in the Spitzer hypothesis, heredity should be much in evidence in the ophthalmoplegic variety. Perhaps it is, but the cases are few and far between.

There has been much speculation concerning the central or peripheral nature of this third nerve palsy. The present view taken for migraine in general, that it is due to a disturbance in cerebral pressure, secondary to vascular modifications, is sufficient to account for the oculomotor palsies as well, in view of the location of the peripheral branches of the third nerve in regard to the cerebral vascular plexuses. In fact, the occurrence of the ophthalmoplegic type is one of the strong arguments for the general pressure hypothesis, as Spitzer has well argued. If, as has been shown by several autopsies, to these considerations additional local causes be added, which increase or permanently maintain such pressure effects, the interpretation is comparatively simple. Thus, exudates, fibrous processes, swelling in the cavernous sinuses, swelling of the hypophysis, tumor formation, gummata, etc., have been found in patients suffering from periodic oculomotor paralyses associated with migraine.

It is true that some of these are to be interpreted as symptomatic migraines, in which the foreign body acts primarily as an irritant to cause the vascular disturbance, which sets free the migraine reaction, and secondarily serves as an additional cause of pressure to bring about the palsy. In a personally observed case, with basal gummata, the periodic oculomotor palsy and

migraine attacks have occurred for a period extending over four or five years, usually with every menstrual period. Here were three interplaying factors, and the exact part played by each can only be inferred. The slight disturbance of menstruation, usually adjusted, in this case, by reason of the exudate, was not. A migraine was set up, the acute pressure of which, added to that of the exudate, caused the ophthalmoplegia. This ophthalmoplegia had become fairly persistent in the intermigrainous interval in recent years.

**Symptoms.**—Leaving aside for the moment the atypical and symptomatic periodic oculomotor paralyses due to other causes than migraine, one finds in these patients, usually during or after a severe attack of unilateral migraine, with headache, nausea, vomiting, etc., a ptosis of the eyelid on the same side, and a loss, partial or complete, of the upward, downward, and inward movements of the eye of the same side. This eye is usually directed outward and downward, and the patient sees double. This may or may not be accompanied by sensory disturbances in the superior branch of the trigeminus, just as may be observed in ordinary ophthalmic migraine.

After a variable length of time, a few days, a week or more, the paralysis disappears, usually gradually, and the patient suffers no inconvenience from the ocular palsies or the ptosis. In some individuals such palsies accompanying a migraine have come on comparatively young in life, almost with the beginning of the migraine attacks; for the majority, however, they follow several years after the establishment of a migraine, in some instances as late as thirty years. In some only a very severe attack will be accompanied by the oculomotor signs, or only slight palsies; transitory ptosis may occur frequently. But in others the palsies develop with each attack of migraine and often in increasing severity. The effects may persist longer and longer between the attacks, until in a few they become permanent palsies. This type, however, often permits of other interpretations.

A double lesion can be understood, although it rarely occurs. Isolated abducens palsy has been described, also isolated trochlearis; and complete ophthalmoplegia is reported in a single case, but in view of the many contributory factors it perhaps is preferable to view such a case from another standpoint.

**Diagnosis.**—Every patient should be regarded as one suffering from something more than the migraine, until all accessory causes are excluded. What these may be has been mentioned already.

**Treatment.**—Little needs to be added to the therapy outlined under migraine. Syphilis as a cause for both a migraine and an exudate should be treated, and the Wassermann reaction utilized to clear up the diagnosis and therapeutic indications.

## NEURALGIA.

**Definition.**—A painful affection of the nerve trunk or its branches, characterized by remittent or intermittent rapid flashes of pain, with free intervals, not usually accompanied by trophic disturbances of the muscles, unless its severity limits the activities of an organ, occasionally associated with painful nerve trunks and with disturbances in the skin structures.

Neuralgias are but the expression of many diverse lesions which may involve the body in general, as toxic and infectious states, a nerve trunk itself, the sensory ganglia, contiguous structures, or they may be the reflex expression of a disorder in a viscus, apparently unrelated to the nerve fibers, which are the site of the pains. Neuralgia is, therefore, to be considered solely as a symptom, a syndrome, or a purely reflex condition. It is a mooted point whether a pure idiopathic neuralgia occurs apart from other conditions which give rise to neuralgic pains. Those who adopt such a standpoint include those so-called functional cases for which a cause cannot be found.

**Etiology.**—An extraordinarily wide range of causative factors may determine mild or severe neuralgias in very diverse regions of the body. The most frequent causative factors are:

(*a*) Anæmias, such as follows severe hemorrhages; chlorosis, pernicious anæmia, and anæmia due to various parasites. Anæmia is a potent factor in reducing resistance to pain and in determining the outbreak of a painful reflex disturbance.

(*b*) Toxins of exogenous origin, inorganic and organic, or purely endogenous toxins. Thus, poisoning by lead, mercury, arsenic, copper, especially mild cases, often conditions severe neuralgias. Alcohol and tobacco are frequent organic poisons, while the toxins of many infectious disorders are especially prone to bring about neuralgias. The morphinist or cocainist suffers severely from neuralgia as an abstinence symptom. Influenza, tonsillitis, and smallpox are familiar illustrations, while the malarial organism causes a toxæmia with a pronounced aptitude to induce neuralgia. Typhoid fever, measles, gonorrhœa, possibly syphilis, and streptococcus infections are frequently accompanied by neuralgias. The endogenous toxæmias of diabetes and latent nephritis frequently cause severe neuralgia.

(*c*) Inflammation of the sensory ganglia, which may be either of infectious or non-infectious nature, gives rise to some of the severest forms, as seen in herpes zoster. According to the ganglion involved, there may result an ear neuralgia—otitic neuralgia, a zoster of the seventh nerve (Hunt), in one or more of its branches—zoster of intercostal ganglia, or even down to the lower extremities.

Ganglion involvements of non-infectious types give rise to neuralgias such as tic douloureux, while tumors of the sensory ganglia may condition persistent and obstinate neuralgias in the distribution of the affected sensory nerves.

(*d*) Involvement of the nerve trunks themselves, either by mild neuritic processes, perineuritis, pressure from anatomical structures, pressure from lesions, cuts, bullet wounds, tears, tumors, aneurism, exostoses, fractures, or displacements, may cause severe neuralgias, which by the advance of inflammation, the persistence of pressure or the continuance of the lesion finally show the neuritic reaction.

(*e*) Reflex neuralgias are numerous and puzzling. Pulmonary, cardiac, gastric, hepatic, renal, ureteric, intestinal, vesical, uterine, ovarian, prostatic, testicular, and affections of other viscera give rise to herpetic eruptions, with painful, sensitive skin areas and neuralgias; in many instances the neuralgia is not accompanied by herpes. Head's complete analysis of this class of cases is of paramount importance.[1] Thus, a persistent sciatica may be

[1] *Brain*, xvi, 1; xvii, 339; xix, 153.

the reflex of a prostatic disturbance. An anæmic woman may not suffer from pain, but on menstruation her referred neuralgic pains may become very severe over the tenth dorsal nerve, and pain and tenderness are frequent over the areas of the sixth dorsal (heart), seventh dorsal (stomach), and there may be occipital and midorbital neuralgia (Head).

(*f*) Organic disease of the nervous system. General paresis, tabes dorsalis, spinal or cerebral disease, thalamus involvement, syphilitic meningomyelitis, etc., are often accompanied by neuralgic pains.

(*g*) Hereditary predisposition. The neuropathic constitution, hysterical personality, physical and mental overwork may be added to the conditions mentioned as responsible. Some individuals have neuralgic pains on the slightest provocation. The arthritic, gouty, rheumatic, and scrofulous may be said to be predisposed. Unknown factors thought to be related to atmospheric pressure, humidity, high electrical tension, etc., play a role in many of these cases.

(*h*) Chronic vascular disease, and especially arteriosclerosis, is a frequent cause, particularly in the aged, the senile, and the presenile. Syphilitic neuralgias are frequently conditioned by vascular disease.

(*i*) Last, but not least, exposure to cold is an important factor. It is not certain that all neuralgias caused by cold are not really mild types of neuritis or perineuritis, but since a neuralgia may be the sole expression of an extremely mild neuritis or perineuritis, discussion of the distinction is fruitless. The older writers found cold a predisposing cause in from 25 to 40 per cent. of the cases. In damp, cold countries this is particularly noticeable.

**Symptoms.**—Pain is the main factor in neuralgia. For the most part, it is the only expression of the nerve disturbance. The character of the pain varies considerably, but in general it may be described as unilateral and paroxysmal. It is characteristic of most neuralgias that they are not primarily localized in the periphery. The pain seems to begin beneath the surface, and may then shoot out to the periphery. It may be described as biting, boring, tearing, darting, cutting, like an electric shock, like a hot iron, etc., each patient having his own pet expression. It may come and go in lightning-like flashes or throbbing pulsations, persisting for a shorter or longer time, then stopping for minutes, hours, or days, then recurring. When continuous, the pain varies considerably in its intensity. At times it is agonizing; then again it is growling and grumbling beneath the surface.

The painful area in most neuralgias conforms to the peripheral distribution of the sensory nerves. In the herpetic and referred neuralgias the root zone area is involved; this supplies an important differential in determining these types. At times strictly localized to a more or less definite spot, again the pain radiates through an entire limb, over one-half of the face, or the trunk. The radiating character of the pain in some neuralgias is very striking. It spreads out along the peripheral branches of a nerve stem. As in the trigeminal neuralgias, one may find the upper, middle, or lower branches involved; alone, in combination, or all three. A dental neuralgia may suddenly jump to other branches of the fifth nerve and the pain become general. Individual peculiarities are constantly met, in which case one theoretically assumes differences in irritability of the nerve substances, in the gray matter of the central structures. In some patients with sciatica,

the pain may suddenly shift from the typical distribution to perhaps the intercostals or other structures closely related centrally.

Certain *points* seem to be foci from which the pains start. These are usually situated along the nerve trunks, and pressure upon them is often sufficient to cause an exacerbation in a mild attack, or to provoke an attack in a period of intermission. Valleix attached considerable importance to these points. They are found, according to him: (1) At the point of emergence of the nerve trunks; (2) at such situations where a nerve trunk transverses a muscle to reach the skin; (3) at points where the nerve fiber breaks up into branches; (4) at points where the nerve becomes very superficial; and (5) at Trousseau's apophyseal points. Valleix's points play a secondary role in present day interpretations of neuralgias, and Romberg has given a severe critique of Valleix's claims. They are of interest particularly in the neuritic types, but are absent in other forms of neuralgic affections.

Accompanying phenomena are frequent. In some patients a sense of apprehension may precede the coming on of an attack; vague sensations of discomfort often antedate the neuralgic outbreak. Ripples of pain, like pin pricks, short twinges, etc., announce the advent of a more serious attack, or may be the sole evidence of an abortive one. Such mild phenomena are extremely frequent in certain of the so-called predisposed or neuralgic individuals. Some feel that they cannot live at high altitudes; others fear rain, or an east wind; a thunder storm causes others to have twinges; while, again, certain dietary indiscretions make others complain of painful twinges for days. Just what conditions are at the basis of these features is unknown, yet they are none the less real. One must always bear in mind the indubitable influence of purely mental influences; this does not make the pains any the less real, however, but opens a view as to possible therapeutic resources.

Skin hypersensitiveness is frequent. It may precede or accompany an attack, and persist after the pain has ceased. Epicritic sensibility is mostly implicated. Light touch, a pin prick, or slight degrees of heat and cold are magnified. Deep pressure and extremes of heat and cold are usually palliative.

*Anæsthesia* is not infrequent following an attack of pain, and the exact topographical distribution of the sensory modifications on the skin throw considerable light on the possible causation of a neuralgia (Head). Trousseau's *point apophysaire*, or painful spinal point, should be recalled in this connection.

*Paræsthesia* is very frequent, and certain distributions seem to show it more than others. Thus, in the cutaneous branches of the femoral they are not infrequent. Here they take on the character of a "meralgia paræsthetica."

*Motor disturbances*, either as cramp-like contractions or as paralyses, are not infrequent in accompanying conditions. The painful contractions of tic douloureux and the oculomotor paresis of ophthalmoplegic migraine are familiar examples of these. Gowers reports general convulsions, vomiting, and opisthotonos in severe neuralgias. Brissaud has called attention to a spasmodic type of sciatica with increased knee-jerks, increased Achilles reflex, and muscular contractions, in which central involvement seems unquestioned.

*Vasomotor* and *secretory* symptoms are frequent. The bloodvessels are frequently contracted in the early stages of a neuralgic attack, with resulting blanching and cooling of the skin. Following this a period of warmth, of redness, of free perspiration may result due to the secondary dilatation of the vessels. In many cases of trigeminal neuralgia other secretions may be modified. Crying, coryza, or salivation are not infrequent, while in general neuralgic attacks an increase in the amount of urine and of milk secreted is frequently found.

Premature graying of the hair, loss of hair, thickening of the skin, erythemata, eczema, pemphigus, herpes, thickening of the bones, and, occasionally, muscle atrophy are among the rarer trophic by-products.

During an attack irregularities of the pulse are not unusual; slowing is the rule. The pupils are frequently dilated, at times unequally.

The general physical and psychical reactions are extremely important. Loss of sleep and anorexia cause the patient to lose strength and flesh; and anxiety, irritability, and petulance are almost inevitable. Mental depression, sufficient to lead the patient to make suicidal attempts, is not infrequent, especially in severe cases of trigeminal and sciatic neuralgia. The contracting of a drug habit is not unusual.

**Course.**—This depends naturally upon the underlying condition. Many are acute and transitory, persist for three or four days, and never reappear. Such are the herpetic types. On the contrary, certain neuralgias are persistent, chronic, and show a tendency to grow worse. Tic douloureux is an example. Many reflex neuralgias run an acute recoverable course, but show a marked tendency to recurrence. The neuralgias which accompany the chronic cachexias of nephritis, carcinoma, brain or spinal cord disease usually progress in a markedly chronic manner. In those hereditarily disposed individuals the tendency to chronicity with longer and shorter periods is proverbial. Earlier French writers attempted to distinguish benign and severe forms. Most neuralgias in which the causative factor is undiscoverable (the so-called idiopathic or primary neuralgias) run a benign course, while the neuritic types are less amenable to treatment.

The subdivisions of neuralgic neuroses, subacute neuritic neuralgia, and chronic neuritic neuralgia offer a grouping referable to the course which has only clinical convenience to warrant it.

In the first type one finds the disorder limited more or less to the neuropath. The attacks come on without appreciable cause, or follow a nervous shock, exposure to cold, or dietary indiscretions. The pain comes on with great suddenness and usually without great violence; it comes and goes apparently without rhyme or reason, and is not accompanied by painful nerve trunks nor trophic disturbances. It recovers at times, to recur at intervals of a year or years.

In the subacute neuritic neuralgic type, exposure to cold or to pressure, especially in arthritic patients, determines an attack. The attack develops gradually; the pain, at first mild and intermittent, gets worse and worse and more continuous. Finally, after a day or more the paroxysms become extreme, the intervals between marked by dull pain; Valleix's points are characteristic findings. When involving a mixed nerve, muscular atrophy or other trophic signs appear, signalizing the occurrence of a neuritic process. Local œdema and herpes zoster are frequent accompaniments. This type usually commences to recover in from two to three weeks, and an

ultimate recovery is to be expected. Recurrences occur, however, and leap to the third type of chronic neuritic neuralgia. This form is frequent in the aged. The history is usually that of several subacute attacks with increasing tendency to chronicity. Here the trophic disturbances in muscle and in skin are more marked. The paroxysms run a remittent course.

**Diagnosis.**—Enough has been said to emphasize the need for a searching analysis of the causative factors in every neuralgia. They are many, and presumably the most widespread diagnostic error is the overlooking of an early tabes dorsalis in young to middle aged adults. Children are not prone to neuralgias—in the narrower sense—and a neuralgic affection in childhood calls for close scrutiny.

Since unilateral pain, of special localized type, occurring in irregular attacks, is almost the sole criterion of neuralgia, it is very frequent that organic disease of a viscus will show precisely similar accompanying features. In the majority of cases the underlying organic lesion may be detected—occasionally it remains difficult to locate. Not infrequently the diagnosis of a persistent neuralgia may be cleared up by the finding of malarial organisms in the blood, or more rarely the presence of a marked eosinophilia will call attention to trichina as the cause of an obstinate neuralgia; or the eggs of an intestinal parasite in the fæces (uncinaria) may direct attention to an anæmia which underlies a severe neuralgia. Syphilitic neuralgias, either toxic or vascular, are by no means infrequent.

The diagnosis of *myalgia* from true neuralgia is not often difficult, but occasionally, especially in the intercostal and lumbar regions, the diagnosis becomes uncertain. These neuralgic-like myalgias are usually isolated in their location, are not, as a rule, accompanied by acute exacerbations, nor are the regions usually painful on pressure. Motion, on the contrary, usually aggravates myalgia. Motion of the jaw—it may be recalled—is enough to start a severe tic douloureux.

Neuralgic pains about the jaw are at times the precursors or accompaniments of new-growths of the jaw or parotid (Gowers). Neuralgic affections of the cranial nerves are not infrequently due to intracranial growths.

*Neuritis* of a mild grade offers a specially difficult problem. As already stated, mild neuritis is a neuralgia. The question to be solved concerns the likelihood of a more severe degree of neuritis. In this case the usual signs of neuritis appear—painful swollen nerve trunks, trophic disturbances, more continuous pain, Lasègue's phenomenon, weak, flabby muscle fibers, and electrical changes. New-growths pressing upon or involving the nerve trunks within or without the spinal canal, in the early stages particularly, begin their disturbance by a pure neuralgia. Minute analysis of the sensory phenomena will usually clear up the diagnosis early, although in the earliest phases at times it may be impossible.

A neuralgic affection may be one of the earliest signs of multiple sclerosis. Oppenheim has found a severe tic douloureux to have been the earliest sign of this disorder. Syringomyelia may begin as a localized neuralgia. Minute hemorrhagic lesions of the spinal cord of traumatic origin give rise to neuralgias.

In the diagnosis of hysterical neuralgia great caution should be exercised. Hysterical neuralgias partaking of the nature of a pseudo-neuralgia are extremely diffuse, and react very rapidly and markedly to suggestive

influences. Timme has reported a case of general spinal carcinosis, which for a long time was masked under the guise of a hysterical neuralgia. Hysterical neuralgias are almost invariably accompanied by other signs of the hysterical personality.

*Neurasthenic pains* need to be differentiated. The many mixed forms of neurasthenic, hypochondriacal, and hysterical origin bear their characteristic sidelights. The diagnosis of these types of neuralgia should not be lightly made, for it is not to be forgotten that these syndromes of themselves may be the reaction on the part of the nervous system to some more fundamental organic lesion. Thus, patients suffering from severe neurasthenia, with cachexia, and severe intercostal neuralgic pains may have an undiscovered carcinoma of the stomach, mediastinum, etc.

In *tabes dorsalis* the pains resemble those of neuralgia very closely, but, as a rule, have a wider range, are not localized in a peripheral nerve distribution, and are more apt to be radicular in their distribution. Pain on pressure of the nerve trunk is usually absent.

The pains, cramps, and muscular weakness of *intermittent claudication* sometimes bear some resemblance to a severe neuralgia. Aortic aneurism gives rise to reflex neuralgic pains, which are usually very severe, burning or boring in character. Aneurisms in other regions are to be carefully excluded.

In the diagnosis of complicated intractable cases of neuralgia, in which some lesion of the nerve is suspected, Pitres' procedure of comparing the analgesic effects of cocaine injected into the skin of the terminal filaments, along the nerve trunk, or into the root zone of the spinal cord is to be borne in mind. In this manner one may be able to distinguish whether the site of the disturbance is extrafascicular, fascicular, or medullary.

In reflex neuralgias the use of cocaine or other local anæsthetic may determine, by exclusion, the site of the original lesion. An orthoform suppository pressed well against the prostate has been known to relieve a severe sciatic neuralgia. Tumors of the pelvis frequently give rise to sciatic and crural neuralgias, and persistent neuralgic pains of the knee are often a reflex from hip-joint disorder.

**Prognosis.**—This is conditioned by the pathological process that is responsible. The more chronic of the neuralgias, which in years gone by tended to bring about chronic invalidism or inveterate drug habits, have ceased to have such a sinister import by means of a better understanding of the underlying conditions, and by a much more resourceful therapy. The younger and stronger the individual, and the less the tendency to hereditary disposition, the better the prognosis in those neuralgias which apparently are idiopathic, as well as those due to alcohol, lead, or other toxic agent. In the more chronic forms, which are not due to removable conditions, the prognosis is bad. With increasing insight, however, into the many intricate disturbances of nerve metabolism many of the intractable forms may be conquered.

**Treatment.**—The chief indications are to quiet the pain and root out the cause. A painstaking study of the history and an exhaustive physical examination are necessary in all cases. The therapy will vary, therefore, widely if the cause be ascertained; a course of quinine will cure one patient, a surgical operation may be called for in another. General rules, therefore, are largely illusory.

Taking up the general therapeutic indications, the analgesics which have proved serviceable may be discussed first. From the true therapeutic standpoint they are solely palliative. Many cases of severe neuralgic pain may be temporarily subdued by the synthetic analgesics and the dangers of a morphine habit averted. Phenacetin, acetanilide, antipyrine, aspirin, pyramidon, lactophenin, and phenocoll are among those that have proved valuable. New ones are constantly being added, and among them some are certain to be of value. The salicylic acid group combinations are at times useful, especially in the milder cases and in patients with arthritic tendencies. In influenza and tonsillitis neuralgias the salicylates are useful. Combinations of these with soporifics, such as chloral, paralydehyde, sulphonal, trional, or veronal, are useful in procuring sleep, and thus prevent the reduction of the patient's resistance.

If any of the opium group be necessary, it is better to give such in sufficient doses. Usually smaller doses may be given when combined with the analgesics mentioned. Aspirin, gr. vii (0.5 gram), codeine, gr $\frac{1}{3}$ (0.02 gram), and trional, gr. vii (0.5 gram), for instance, is a useful combination in mild cases to be taken at night. Other combinations are equally effective. In the chronic neuralgic pains morphine is to be avoided as long as possible. This does not apply to a very old patient, or one in whom the neuralgia is simply the expression of some chronic incurable disorder—carcinoma, for example. The gradual immunity acquired, with the need for larger doses, and the pernicious effects of a habit apply to all the members of the opium group.

It is doubtful if chloroform or ether is to be recommended by inhalation in severe neuralgias. They are useful temporarily in great crises.

Other drugs are quinine, which in combination with the salicylates is specially valuable; arsenic, which is serviceable in the neuralgias due to anæmia, especially in combination with iron. Atropine and aconitine were used widely before the days of the antipyritic analgesics. Their definitely poisonous qualities have driven them into the background. The unreliability of Cannabis indica has done the same for this otherwise useful analgesic. The iodides are called for in the syphilitic neuralgias, and are useful in many neuritic neuralgias.

Counterirritation is of great service in most cases of severe neuralgia, especially after the acute onset is over. The Paquelin cautery is the best means; mustard paste, cantharides, turpentine, chloroform, ether, and acupuncture all have their place. Local freezing may be carried out by ethyl chloride, methyl chloride, ether, or other volatile substances. Menthol, or other similar derivatives may be used for mild neuralgic pains to advantage.

Direct applications of local analgesics, either to the nerve trunk or within the spinal canal, are valuable in many obstinate deep-seated neuralgias, especially of medullary origin. Cocaine, tropacocaine, eucaine are all useful given by the Corning or Quincke method.

Local applications of heat are grateful and valuable. Hot water bags, hot sand, electrical pads, etc., may be utilized. General or local hot water baths, or hot air baths (baking), are at times desirable. In acute neuritic processes Gowers has found heat disadvantageous.

General hygienic treatment is imperative. A generous diet, full sleep, healthful occupation, and freedom from mental worry are essential. Cod-

liver oil, nitrogenous diet, with iron, arsenic, strychine, calcium salts, are indicated. Faddy dietaries should be avoided. Even in arthritic neuralgias it is doubtful if meat does any particular harm when not taken to excess. Alcoholic beverages are to be denied.

Hydrotherapy may be added to other hygienic means, if it proves invigorating and causes an increase in appetite and sleep. Otherwise it may prove disadvantageous.

Climatic changes are often advisable. Low-lying, damp, and humid atmospheric conditions seem least desirable. The general stimulus that comes from a drier, higher atmosphere, even if colder, works to the general advantage, even if not directly valuable for the relief of pain.

Electrotherapy, when well managed and properly selected, is of great value in some neuralgias. It cannot be said that it is clearly recognized just what forms of current are best utilizable in what types of neuralgia, hence most efforts must follow the method of trial and error. In general, however, Leduc's modifications of the d'Arsonval's rapidly interrupted current offer the readiest and most widely applicable form of electrical current for the relief of neuralgic pain. The effect in certain intractable forms of sciatica is sometimes miraculous. It is doubtful if any other form of electrical application is known at the present time that is as valuable as this. It is, in fact, a type of electrical anæsthesia, solely palliative, but very grateful. Newer applications are being brought out, and other forms may replace the Leduc currents, but at present they seem to give the most reliable results.

Faradic currents as heretofore employed act for the most part simply as counterirritants, and seem to possess little superiority over the actual cautery. Galvanism with mild currents is useful for many topalgias.

*Surgical* intervention is called for in all cases in which pressure is demonstrable and the cause removable. Tumors and new-growths, involving or pressing upon nerve structures, if removable, should be taken away. Surgical interference may be of radical service in many of the reflex neuralgias of obscure origin probably related to visceral ptoses. Nerve stretching needs mention mostly to be condemned.

## SPECIAL LOCALIZED FORMS OF NEURALGIA.

While any nerve fiber in the body may become painful, there are certain regions which show a greater tendency to involvement than others. Bernhardt has collected the statistics of localized distribution in some 685 cases, with the following results: Trigeminal, 124; occipital, 42; brachial, 108; intercostal, 45; lumbo-abdominal, 12; crural, 25; obturator, 2; sciatic, 303; anterior femoral, 11; Achilles, 3; tarsalgia, 4; metatarsalgia, 4; and coccygeal, 2. In 613 cases collected in the clinic of Dr. M. Allen Starr during four years (1902 to 1906) the distribution was as follows: Trigeminal, 315; occipital, 28; brachial, 31; ulnar, 1; intercostal, 19; lumbo-abdominal, 19; crural, 2; sciatic, 194; coccygeal, 1; peroneal, 2; and plantar, 1.

Thus, with the New York population the affections of the three branches of the fifth stand first, the sciatic next, while the brachial, occipital, intercostal, and lumbo-abdominal follow. Bernhardt, Lachnit, and Conrad have found sciatica to be the commonest type, with trigeminal neuralgias second.

Eulenberg has found, with the author, that the trigeminal affections have been more frequent. In the Vanderbilt Clinic (New York) the trigeminal neuralgias have been more common in women, while the sciaticas have predominated very markedly in men.

**Facial Neuralgia.**—Neuralgias of the branches of the fifth nerve are among the commonest of all the neuralgias. Fothergill's studies on "A Painful Affection of the Face," published in 1773, is a classic. The inferior and superior branches preponderate in frequency of involvement. Most frequently these neuralgic pains are due to some affection of one of the branches. Inflamed teeth play a predominant role. Affections of the ears, the eyes, iritis, cyclitis, iridocyclitis, the skin of the face or head, inflammation within the accessory sinuses of the nose, forehead, antrum, mastoid, all of these may produce diffuse neuralgic pains, at times clearly separable from a simple neuralgia of the fifth, at other times not. The characteristic type of trigeminal neuralgia is tic douloureux; trigeminal herpetic neuralgia, involving one or more branches, is not uncommon.

Cold and wet are very important agents in facial neuralgia. In certain countries, notably England and the north of Germany, facial neuralgias from this cause are extremely common; they seem to be much less frequent in the United States, and notably so in southern countries.

Tabes dorsalis may make its first appearance as a trigeminal neuralgia. Reference has been made to Oppenheim's case of multiple sclerosis beginning as a facial neuralgia. The other factors of general causation already noted may be of primary significance in facial neuralgia.

Neuralgia of the superior branch is seen more commonly by physicians, although the dental branches are involved much more frequently. These patients go to dentists, and hence do not enter into medical statistics. This is a reason why it is incorrectly stated, by most writers, that the superior branches of the fifth are most often involved. For the most part the milder types of facial neuralgia are induced by irritation of some of the terminal filaments, while in the severe form, tic douloureux, which is the more classic, a lesion of the Gasserian ganglion is uniformly present. Mild cases of tic douloureux may be indistinguishable clinically from other types of facial neuralgic pain.

Enough has been said on neuralgia in general to indicate the character of the simpler forms of neuralgia of the fifth. One type, however, by reason of its severity and its fairly definite pathological anatomy, needs more extended consideration. It would be desirable to restrict the term tic douloureux to a definite and, if possible, limited type of neuralgia of the fifth nerve, particularly to the form due to changes of a chronic degenerative nature occurring in the Gasserian ganglion. This is not yet possible, and clinically the neuritic and peripheral cases are either not at all separable from the ganglion cases, or with considerable difficulty.

*Tic douloureux* affects one side of the face only, as a general rule. In certain generalized neuralgias, such as those due to diabetes or nephritis, both fifth nerves may become involved, but this is unusual. In the majority of cases selection occurs among the branches, one or two being involved, rarely all three; the ophthalmic branch the oftenest, the inferior maxillary the least. Avicenna knew tic douloureux and described it with great accuracy.

The more classical tic douloureux neuralgias are characterized by the

extreme severity of the pain, usually preceded by paræsthetic prodromata, and widely accompanied by sympathetic or irradiating pains in other branches than the one chiefly involved, or in other nerves. The pain may be paroxysmal or continuous, with marked exacerbations. Oppenheim has compared them to the piercing pains of a sharp knife or the burning of a red-hot wire. The patient remains for a shorter or longer period, a few minutes to several hours, under the grip of the pain, unable to move a muscle of the face or fearful of stirring, lest a spasm more fearful than the others should occur. The slightest touch is avoided, even the air pressure of a suddenly closed door may bring about an exacerbation. The longer attacks are rarely as vicious as the shorter ones.

Valleix's points are relatively constant. In the ophthalmic involvement the sore parts are found above the supra-orbital notch, at the external angle of the upper lid, the upper outer aspect of the nose, and the globe of the eye; in the superior maxillary branch the inferior orbital notch is the chief point of pain; the malar bone, and opposite the last upper molar are other less frequently found points, while the outer angle of the mouth and the roof of the mouth are rarely their site. In the inferior maxillary distribution the points are chiefly just in front of the auditory canal, the side of the tongue, the border of the chin, and Trousseau's points over the first and second cervical vertebræ.

Vasomotor and secretory disturbances are usual. The skin is, as a rule, hot and swollen, occasionally pale and frigid; tears, nasal secretions, and saliva flow in abundance. The eyelids may be swollen, the conjunctiva reddened, to the point of ulceration at times; within the nose and mouth extravasations occur, and ulcers are not uncommon. Herpetic attacks are also not infrequent, and in some of these attacks grave injury to the eye structures may take place. Glaucoma is one of the severe complications. Other trophic disturbances have been noted, such as skin eruptions, acne, erysipelatous reddening, graying of hair, and blackening of the tongue. In continued cases hemiatrophy is known to occur. Changes in the sense of taste, of touch, of hearing, are at times present. Photophobia is frequent, while diminution in the visual fields and accommodation cramps are noted. Gellé has described a neuralgic deafness, due possibly to labyrinthine effusion in protracted cases.

Severe mental disturbance, amounting at times to hallucinatory confusion, may be present, and profound states of depression, which have resulted in suicide, in isolated cases, are to be guarded against.

The motor disturbances consist in convulsive movements of the facial muscles (convulsive tics, spasmodic epileptiform neuralgia, Trousseau), sudden forced closing of the eyelids, drawing of the mouth to one side, or sudden turning of the head. At times the convulsive movements extend to the arms. Paralytic phenomena in the third nerve are noted. The general physical disturbances noted are prone to occur in this type.

**Course.**—In the majority of cases the attacks appear in series and attain a periodicity which comes to be dreaded by the sufferer. The free intervals usually become shorter and shorter; but many patients may have only one attack a year, especially in cold weather, or even at longer intervals. A single attack may last a few days, or in the severe forms several weeks, the patient not being free from pain day or night, save under the influence of morphine. Some patients have a few attacks in a lifetime, others are

not free from the disease for years. The severer convulsive forms are prone to occur late in life, when the natural resistance is low.

**Diagnosis.**—Ordinarily the classical form of tic douloureux is recognized without difficulty. Patients have had all their teeth extracted, however, under the mistaken diagnosis of a dental disease, while some intractable facial neuralgias have been cured by proper attention to diseased teeth. Aneurism of the carotid, tumors pressing upon the nerve or upon the Gasserian ganglion, may be difficult to determine as the exciting cause. These, however, are usually accompanied by accessory symptoms, palsies, eye-ground changes, aneurismal murmurs, pain within the head, cerebellar syndromes, ear pains, etc. The otalgias (tympanic neuralgias) usually considered in this connection are possibly due to geniculate ganglion disorder, and have been referred to by Hunt as neuralgias of the seventh nerve, a view point that Mills combats.

**Treatment.**—It is as essential to endeavor to find and treat the cause of a facial neuralgia as for neuralgia in general. The various remedies given under the heading of neuralgia may be tried, and as malarial neuralgias are very frequently facial, energetic quinine therapy should be given; the absence of blood findings is not contra-indicative, especially in malarious regions. Arsenic is a useful adjuvant in these patients, and also valuable in non-malarial neuralgias. Gelsemium, the tincture in 10 minim doses, gradually ascending, aconitine in doses of $\frac{1}{100}$ grain, Cannabis indica (fresh) in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain, are reputed as specially valuable in the facial cases. Any of the analgesic antipyretics may suit individual cases, and avoid the use of morphine, which alone is reliable in many severe cases. Local applications of cocaine to the conjunctiva, nasal mucous membranes, and buccal surfaces are sufficient to repress some mild attacks.

Injection methods have been tried for years. In the beginning the peripheral branches were injected by various analgesic drugs, in early days chloroform, and in later times particularly cocaine and its allies or derivatives. The effects were valuable but temporary. Osmic acid was used later (Billroth and Neuber, 1884; Eulenberg, 1884; Schapiro, 1885); this set up a degenerative process, and in many instances relief was obtained, but regeneration took place even when the injections were practised within the main trunks. Pitres and Vaillard, in 1887, and Schlösser,[1] in 1900, took up a series of experiments with alcohol, and the latter has perfected a method of injecting alcohol within the substance of the Gasserian ganglion, which has given excellent results. The chief feature in the deep alcohol injection method is the introduction of a long, dull, cutting needle into the foramen ovale and there injecting in situ the branches of the trigeminus. Schlösser advocated piercing the cheek behind the last molar, running up alongside of the pterygoid plate of the sphenoid to the foramen, while Ostwalt claims he can obtain the same results by going up within the mouth. The Levy method is also extrabuccal. Narcosis is not necessary; 80 per cent. alcohol is used. In three to four hours following the injection pain is relieved, and two or three more injections are given within a week to complete the treatment. Immediately following the injections, which should be done only after extended practice on the cadaver, there is a marked anæsthesia on one side of the anterior part of the head, including

[1] *Münch. med. Woch.*, April 30, 1907.

the nostril, palate, and one-half the tongue; a slight paralysis of the muscles of mastication, which may persist for some time, but usually disappears in a few hours; a degenerative process is set up in the nerve trunk, which is recoverable, and general sensibility usually returns, but the pain is absent. Relief extending over a year in a number of cases is reported by numerous observers. Some patients have been relieved for four or five years. Œdema of the posterior eye structure and hemorrhage are among the discomforts and even dangers of the operation, especially in the use of the intra-orbital methods devised by French operators. In Schlösser's analysis of 209 cases, 123 had injections in the trigeminus. Recurrences were noted on an average of ten months in the majority of these. Repeated injections, however, delay the recurrences more and more, and thus the patients may be held immune to the pain. Ostwalt claimed better results from his injections, saying that 90 per cent. may be benefited; in 10 per cent. the disease is probably intracranial. Relapses occurred in 30 per cent. of his cases, and then within five months. Numerous modifications and later methods have been devised.

As regards surgical means, three procedures have been seriously advocated. The first and earliest consisted of peripheral section. Oliver Wendell Holmes, writing in 1836, says that Dr. J. C. Warren, of Boston, was the first to divide the facial nerve at its exit from the stylomastoid for tic douloureux. Section of the fifth may be employed to advantage in those cases in which the disease is undoubtedly peripheral. As modified by more recent procedures, the older objection that regeneration takes place is partly done away with, and in selected cases peripheral section may be preferred to the more severe and serious operations.

Rose,[1] in 1890, first successfully extirpated the ganglion for tic douloureux, although MacEwen and Horsley unsuccessfully attempted the operation a few years previously. Horsley, Hartley, and Krause further perfected the operation, and the modified Hartley-Krause operation by the temporal route has been largely the method of choice. Cushing's modifications are of lasting value. The operation still remains one of much difficulty and seriousness. Recurrences are known even with this method, and the efficiency of the newer devices for preventing this by capping the ends of the divided nerve trunks with metallic laminæ is too recent to pronounce upon.

Van Gehuchten,[2] in 1903, suggested a third departure, namely, what he termed the physiological section of the roots of the Gasserian ganglion by tearing. His extensive studies on regeneration in nerve fibers showed him that the central side of the ganglion must be attacked if the problem of recurrence was to be solved, and, furthermore, his method took into account the small number of intracranial cases not reached by the more peripheral methods. Spiller, as early as 1898, had suggested the surgical expedient of cutting the sensory root, which he claims is safer than the operation of tearing, a procedure tried in 1881. The method of division of the sensory root as reported by Frazier and Spiller promises to be one of the most valuable surgical procedures thus far devised.

**Cervico-occipital Neuralgia.**—This occurs in the distribution of the sensory nerves of the cervical plexus, consisting chiefly of the occipitalis major, the occipitalis minor, auricularis magnus, cervicalis superior, supra-

[1] *Lancet*, 1892, i, 295.

[2] *La Nevraxe*, 1903, v, 201.

clavicularis and phrenic. Neuralgia in this general region seems to be rare. In Remak's summary of 15,000 cases only 50 were in the cervico-occipital regions. Valleix has given one of the most complete monographs on neuralgia in this area and little has been added to his description, save in the finding of rare etiological factors.

**Etiology.**—The several causes of neuralgia are operative here, and need not be repeated. Special determining features seem to be the carrying of heavy weights on the shoulders (a more frequent cause seen in brachial neuralgias), arthritis deformans of the upper cervical vertebræ, caries, syphilis, tuberculosis, tumors, pachymeningitis, falls and blows wrenching the cervical vertebræ, enlargement of the cervical lymphatics, and aneurisms of the vertebral artery. Oppenheim refers to the great frequency of hysterical neuralgia in this general region; neurasthenic neuralgia of the back of the neck and occiput is common.

The pains occupy the regions mentioned, being particularly localized in the neck, below the occiput, and running up to the vertex, occasionally behind the ears. The Valleix point found most frequently is the occipital point between the mastoid apophysis and the first cervical vertebra; points between the sternomastoid and trapezius (cervical), the anterior border of the mastoid, and the middle of the ear are of less frequent occurrence.

The pain is very frequently bilateral. Dull pain on pressure, with tender skin, is usual as an interparoxysmal occurrence. This tends to make the sufferer hold his head in a stiff position, which in time may cause a characteristic attitude. The tenderness may be so acute that ruffling of the hair will start a paroxysm. Graying of the hair, loss of hair, with other trophic signs, may be present. Sudden pulling back of the head, or other muscular involvement, is an occasional symptom.

**Diaphragmatic Neuralgia.**—This form of neuralgia, also known as phrenic neuralgia, is of rare occurrence. Falot and Peter have written upon it. The pain is usually present near the free border of the ribs, or within the chest, between the ribs, occasionally as high as the chin and in the neck, beneath the clavicle, and in the scalenus anticus muscle. Trousseau's points are located over the second to the fifth cervical vertebra. The pain frequently runs down the arm, especially in certain complex cases of mixed brachial neuralgia.

Breathing may be seriously interfered with, the breath coming fast and short; longer excursions of the diaphragm are impossible. It is a common experience to have a sharp, short stitch in the side, with inability to breathe for fear of pain. This is the type of distress encountered in phrenic neuralgia. In the majority of cases the pain is in the left side.

Anæmia, affections of mediastinum, heart, and pericardium,and aneurism of the aorta are the most frequent attending features. An intractable phrenic neuralgia may complicate an exophthalmic goitre, or be present in carcinoma of the neck region.

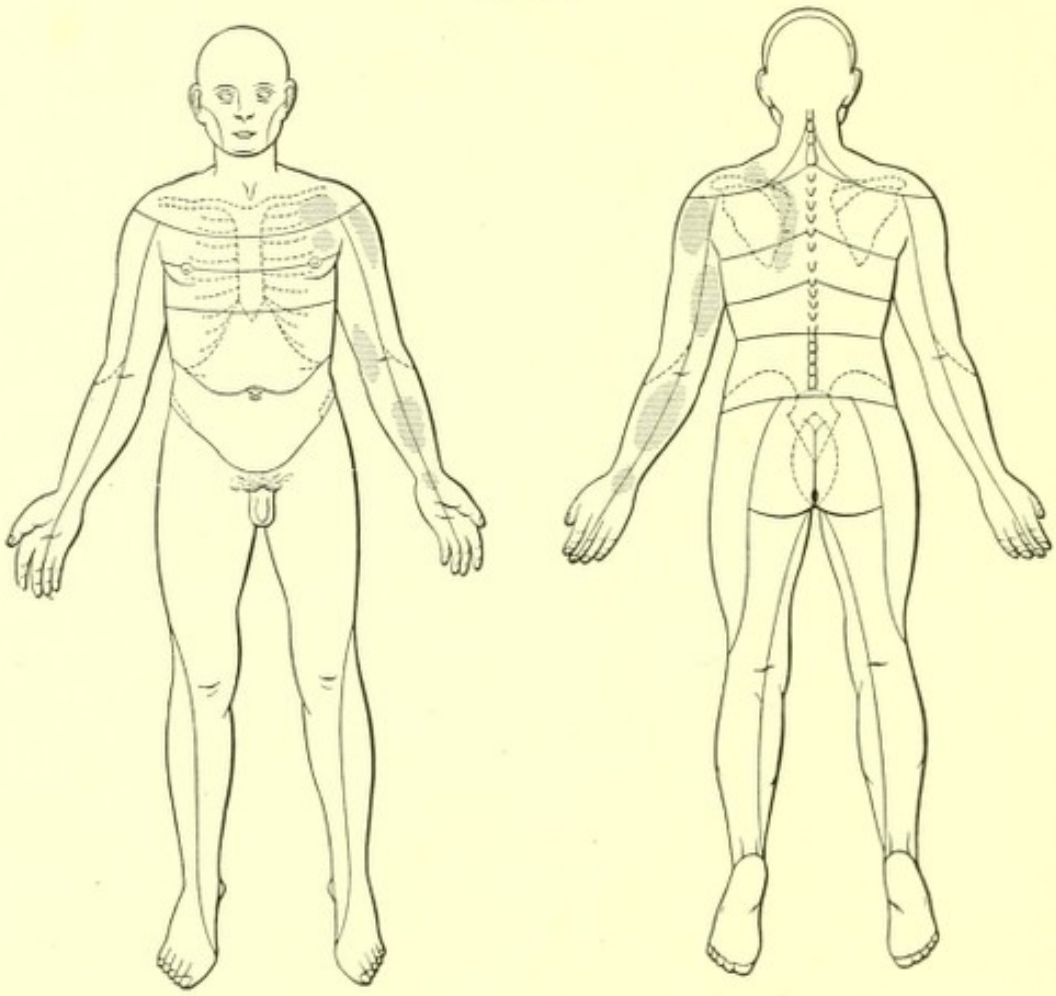
Idiopathic or pure phrenic neuralgias seem to be unusual, whereas temporary or more permanent types are seen as symptoms of the affections named. In the latter case the prognosis depends on the initial difficulty.

**Brachial Neuralgia.**—In this general form the components of the brachial plexus, from the four lower cervical, or some of its filaments, and first dorsal roots, are those involved. The chief nerves carrying sensations from the skin area of the arms and shoulders are the circumflex, radial,

internal cutaneous, and musculocutaneous. These enter, for the most part, the upper and middle cords of the plexus. In the majority of cases the pains of brachial neuralgia are located in the upper arm and about the shoulder, *i. e.*, in the area of the circumflex, radial, musculocutaneous, and internal cutaneous nerves.

Bernhardt's statistics show that men are more frequently affected than women, but the reverse shows true in the figures of other observers (Romberg, Erb). More women come to the Vanderbilt Clinic for brachial neuralgia than men, and in most instances it seems that excessive sweeping is the

FIG. 11



Painful points in brachial neuralgia.

attributed cause. In piano players neuralgias in this area are frequent. Perhaps these should be relegated to the occupation neuroses with the pains of hair dressing, skirt carrying, telegraphy, writing, etc. At any rate, arm and shoulder pains are frequent in their mild grades at least, and very variable. Dana[1] has suggested, perhaps not very seriously, that among the well-to-do women the arms, from lack of use, are becoming vestigial, and the wide occurrence of mild shoulder and arm pains in women may be looked at from this point of view.

The usual causative factors all come into play here. The neuropathic

[1] *Medical Record*, 1907, lxxi, 297.

constitution is put in the foreground by Oppenheim; Bernhardt lays considerable stress upon the importance of bone injuries with callus formation in the formation of many arm neuralgias. Small punctured wounds about the wrist, forearm, and arm are responsible for many symptomatic neuralgias, as Weir Mitchell has so well shown. More remote causes are found in vertebral disease, tumor formation, aneurisms, syringomyelia, multiple sclerosis, and tabes. The frank neuritic processes in their beginnings must be borne in mind, and cervical rib should not be overlooked

**Symptoms.**—Cervicobrachial neuralgias are extremely variable in distribution, extent, and severity. The onset is usually sudden, especially in those patients in whom an antecedent history of exposure to cold and wet is obtainable (motormen, policemen, etc.); at times the beginning is preceded by twinges and slight distress. On awakening in the morning sharp pain is felt in the shoulder and arm. The pains in brachial neuralgia are less apt to be the sharp shooting variety so dreaded in tic douloureux, but sudden accession of sharp pains, varying in their intensity, are frequent. As with most neuralgic pains, movement increases them. Toward evening the pains are apt to increase and the patient, although obtaining relief by lying down, rarely sleeps well. Soreness of the skin, slight swelling, and general reduction in tone are the usual accompaniments. With increasing disuse slight atrophy is common, and swelling is usual. The tendon reflexes are usually more irritable and active. More marked atrophy paresis with vasomotor trophic symptoms and altered tendon reflexes indicate a definite neuritic process. Herpetic eruptions occur with non-infectious, as well as infectious involvement of the sensory ganglia.

Tender points are very variable. They are most frequently in the middle of the back; about the level of the second or third dorsal there is usually a sore Trousseau point. Gowers notes that the inferior ulnar point in front of the wrist is the commonest sore point. Babinski has called particular attention to a radial neuralgia due to a mild or severe neuritis of the radial. The pains occupy the posterior portion of the arm, and are unusually severe. Neuritic changes are not infrequent. The chief causes seem to be exposure to cold and the rheumatic constitution.

**Diagnosis.**—In the diagnosis particular care is needed in excluding affections of the spinal cord, meninges, and vertebræ, as well as angina pectoris and pseudo-angina. Disease of the joints and bones should be excluded at the outset, although it may be impossible in some cases of periostitis. In tumors and other organic affections of the cord the painful points are usually absent, but the earliest and only symptom of spinal cord tumor, intramedullary or extramedullary, may be a brachial neuralgia. In tabes the pains are apt to be bilateral. The exhaustion neuralgias are also apt to be bilateral. Valleix's and Trousseau's points are usually absent. The general indefinite features of a myalgia, plus the muscular rather than the nerve soreness, are usually sufficient to exclude it.

The occupation neuroses involving the arm and shoulder are many. The history of the protracted exercise of certain groups of muscles is usually sufficient to identify the proper cause for the neuralgic pains. Occupation neuralgias, like neuritic neuralgias, are neuralgias none the less, the sole diagnostic question arising as to the cause, and through this the proper mode of therapeutic attack and the probable outcome. Alcoholic neuritis in its mild grades offers particular embarrassments. Lead poisoning

neuralgias are to be borne especially in mind, while diabetes is of prime importance.

Brachial psychalgia is a possibility, but the diagnosis must be made with extreme caution after a rigid exclusion particularly of organic factors. Hysterical and neurasthenic neuralgias occur in this distribution as well.

**Treatment.**—Rest is a necessity, and is primarily insured by the use of a sling. The diagnosis of the cause being assured, treatment should be begun to remove it, either by medical or surgical means. Nerve suturing for injury has come to occupy an important part, and is usually attended with good results, even after long periods of loss of function. In the early stages active mechanical treatment is to be avoided. Hot applications are useful in most acute neuralgias. In later phases massage, particularly the Nägeli movements, are valuable. Galvanism, 3 to 6 milliamperes, is well adapted to these neuralgias, but usually much better results are obtained by the Leduc rapidly alternating currents. Salicylates (especially in analgesic combinations), iodides, quinine, arsenic, and large doses of strychnine are of value.

**Intercostal Neuralgia.**—The twelve dorsal nerves constitute the plexus involved, although the upper series, especially of the left side, are most frequently concerned. Bernhardt says that the site of election is mostly from the fifth to the ninth. Since the dorsal nerves divide into internal and external branches the site of the neuralgia may be on the surface or within (pleurodynia, etc.). The two upper nerves send branches to the internal surface of the arm, and pain is occasionally felt there. The abdominal involvements are rarer, and may extend down to the genitals.

Women more often show this form of neuralgia than men, and the disorder is much more common in cold weather.

The pains are usually less severe than in other regions, although their sharp, sticking character distresses the chest movements, especially since all motion tends to aggravate them. Tender points are found at the site of perforations near the spine. Skin hyperæsthesia is extreme at times. Herpetic neuritic neuralgias are relatively common in this distribution.

Among the causes to be diagnosed may be costal caries, affections of the spinal cord and meninges, disorders of the pleura, particularly carcinoma and tuberculosis, aortic aneurism, dilatation of the stomach, carcinoma of the liver, angina pectoris, pericarditis, local trauma, fractures, etc.

*Mammary neuralgia* or *mastodynia*, which is frequent in the later phases of nursing, and in some women at the menstrual epoch, is a special form. The pain is usually deep within the gland, and may be accompanied by a slightly increased secretion. The whole skin may be sensitive, especially the nipple, when the superficial nerves are mostly involved. Local glandular induration occasionally occurs. This has led to the mistaken diagnosis of carcinoma, but a neuralgia may occur due to a carcinoma of the breast. Tabes may give rise to an intercostal neuralgia.

**Treatment.**—Local applications are useful, especially the ethyl chloride spray. Blisters are efficacious. Bandaging affords marked relief. General measures already described call for no further mention.

**Lumbar Plexus Neuralgias.**—These are most conveniently arranged as (1) lumbo-abdominal, (2) ilioscrotal or testicular, (3) crural, (4) femoral, and (5) obturator, involving in each case certain of the branches of this plexus. Mixed and indeterminate forms are not infrequent.

1. **Lumbo-Abdominal.**—These occupy the lower half of the trunk, and are extremely variable. The chief nerves involved are the iliohypogastric and its branches, the inguinal, and genitocrural. Strict localization to one trunk is rare, and men are more frequently affected than women. The chief causes in addition to those of general moment are local inflammatory conditions or new-growths involving the plexus or some of its branches. The pains are usually unilateral, occasionally bilateral, involve the region of the back below the ribs, the gluteal region, the abdominal and inguinal areas, the scrotum, or the labia. The chief Valleix's points are over the lumbar vertebræ, the hip or iliac point, hypogastric point, and the scrotal point. Lumbo-abdominal pains are usually accompanied by intercostal pains above or thigh pains below.

2. **Testicular Neuralgia.**—Astley Cooper termed this neuralgia the "irritable testicle." The pains are usually unilateral, pass into the testicle, which may be swollen and tender to the touch. The pain not infrequently passes into the leg and back, and the patient may have an attack of vomiting. Bernhardt notes that the pain may be so intense as to cause the patient to seek castration. The affection is an obstinate one, and is not helped, as a rule, by removal of the testicle. Diagnosis involves a rigid exclusion of organic disorder of the testicle, although many affections (gonorrhœa, tuberculosis, chronic prostatitis, etc.) are not infrequently accompanied by persistent neuralgic pains.

3. **Crural Neuralgia.**—The crural or femoral nerve is here implicated. The pain extends in the upper front and inner side of the thigh, to the knee, and further through the saphenous distribution to the ankle and inner aspect of foot, extending as far as the big toe. It is almost entirely confined to men, and shows considerable variability as to the branch involved. It not infrequently accompanies a sciatica. Special etiological features are found in fæcal impaction, or even chronic constipation, disease of the hip or knee bones, enlargement of the inguinal glands, aneurism of the iliac artery. Charcot called attention to the frequent association of crural neuralgia and diabetes. Spinal arthritis is an obscure cause.

Movements of the thigh usually are painful, and the patient comes to bend his body forward in a strained postion. The painful points of greatest frequency are just below Poupart's ligament, just within the inner condyle, over the malleolus, inner side of the instep, and one over the great toe. Neuro-atrophic changes usually occur in the quadriceps, but the patellar reflex is rarely affected, save when a definite neuritis is present. Herpes, reddening, hyperæsthesia are not infrequent. In the diagnosis, disease of the inguinal vessels is to be looked for, as well as intrapelvic disorders, new-growths, etc. Crural neuralgias have a fairly good prognosis.

4. **Femoral Neuralgia.**—Here the cutaneous femoris lateralis, arising higher up in the pelvis, is involved. The pain is felt in the upper and outer aspects of the thigh, extending to the knee. A painful point over the anterior spinous process of the ilium is usual. Paræsthesia in the distribution of this nerve has been extensively studied (Meralgia paræsthetica.) The relation of the pressure of corsets in the causation of this type of neuralgia has been pointed out by Freud, and much sitting in adipose individuals is frequently associated with this neuralgia. The prognosis is favorable.

5. **Obturator Neuralgia.**—Lesions of this nerve are fairly constant as a result of the pressure of the intestinal loops of a hernia. The pain is located

in the inner side of the thigh, and is accompanied by a feeling of stiffness, creepy, crawly feelings of the skin, and inability to bring the thigh toward the middle line of the body.

**Neuralgias of the Pudendal Plexus.**—A large number of neuralgias of the general plexus are recorded. The median hemorrhoidal branches, distributed to the rectum, bladder, and vagina, the inferior branches to the arms, and the pudendal nerve supplying the testicular sac, the labia, penis, urethra, and clitoris, are the chief nerves involved. The general terms, spermatic neuralgia, anal neuralgia, perineal neuralgia, rectal neuralgia, vesical neuralgia or cystalgia, urethralgia, prostalgia, penis neuralgia, irritable uterus, ovarian neuralgia, are utilized to describe these different affections. These neuralgias are very rare, but often very obstinate. Spermatic neuralgias are among the most frequent, and are not infrequently accompanied by painful priapism, perhaps ejaculation.

Since the advent of bicycle riding neuralgias of this general region have been on the increase. The ovarian neuralgias are complex, and more often come within the domain of the gynecologist, as structural defects are usually the underlying causes. Localized herpetic eruptions accompany neuralgias of this plexus. Lesions of the cauda equina are to be carefully excluded in neuralgias of this region.

**Neuralgias of Coccygeal Plexus.**—Coccygodynia, painful coccyx, is a not infrequent disorder in women, especially in multiparæ and in the badly constipated. Trauma and caries are frequent causes. The hysterical coccyx is not infrequent, and referred coccygeal pains are common. The pain is so intense at times that defecation is rendered impossible; the patient cannot sit, and a grave neurasthenic condition supervenes. The medico-legal significance of coccygodynia is real, appearing frequently as a local symptom of a general traumatic neurosis. Surgeons frequently lay considerable stress on a freely movable coccyx in accident litigation. A just estimate of the true bearing of an injury to the coccyx can only be arrived at by a careful survey of all of the factors of the particular case.

Local treatment is seldom efficacious save in the truly neuralgic types. Resection is rarely a justifiable procedure. Nägeli's massage movements are particularly efficacious in remedying the mechanical obstructions often conducive to persistent constipation.

## OCCUPATION NEUROSES.

**Synonyms.**—Professional neuroses; professional cramps; crampes fonctionelles et professionelles; spasmes professionelles; impotence professionelles; Beschäftigungsneurosen; Beschäftigungskrämpfe.

**Definition.**—These are abnormal nervous modifications of voluntary muscular coördinations involving certain muscle groups, which are subjected at frequent intervals, by reason of a definite occupation, to fine, complicated, and coördinated movements. They are known under a great variety of names, such as writer's cramps, pianist's cramps, violinist's wrist, telegrapher's cramp, milker's cramp, tennis elbow, baseball pitcher's glass arm, dancer's cramp, etc. For the most part, they consist of tonic contractions, with or without pain, intermingled at times with clonic shocks or tremors. The associated movements become more and more difficult to carry out,

until a motor paresis terminates the attempt, the muscles all being tied up, as it were, into a painful tonic contraction. Muscular atrophy may appear later. A great diversity of objective manifestations is to be observed. In general, two features are characteristic. The spasms occur in muscle groups which carry out some habitual or professional synergistic act, and the cramp occurs only when the act itself, *i. e.*, the bringing together of the entire willed mechanism, is performed. When the individual muscles are acting independently in some other less frequently performed movement no such cramp occurs. The implication of mental impulses is highly probable.

**History.**—These disorders have probably existed in certain of their forms for ages. So long as there have been cows to milk, milker's cramp has occurred, but it has only been within more recent years that the essential kinship of a number of these affections has been definitely realized. It is usually stated that Sir Charles Bell was the first to give a clear description of these disorders when, in 1830, speaking more particularly under the general caption of Partial Paralysis of the Muscles of the Extremities,[1] he writes: "I have found the action necessary for writing gone, or the motions so irregular as to make the letters be written zigzag, whilst the power of strongly moving the arm, or fencing, remained." It will be seen that Bell's observation is hardly more than a note. No research has told of earlier notes, although it is highly probably that such exist. It was by Duchenne, of Boulogne, that the first clear descriptions and explanations were given. He first pointed out clearly the chief, if not all of the features of these disorders. He says, "These accidents manifest themselves only during the exercise of certain voluntary or instinctive movements, and are localized in those muscles which enter into synergistic action." Duchenne showed that the cramps were not confined to any particular muscles; any group in the body which subserved a frequently repeated coördination might be involved. The predominance of hand cramps is due entirely to the fact that they are more frequently employed. Thus, the arm, neck, leg, trunk, and shoulders have certain professional cramps. Duchenne even laid special emphasis on the mental origin of these affections, a standpoint which is widely held at the present time.

Bruck (1832), speaking of stammering and stuttering, makes some critical remarks on a similar condition occurring in writing, namely, that "when writing the pen goes involuntarily over the paper, just as a runaway horse would go with its rider." Gierl, in the same year, in an excellent history of a case, speaks of a characteristic tremor of the fingers of the right hand when occupied in the act of writing. Numerous observations are recorded in the next ten years, and Strohmeyer,[2] in 1840, opened the way to the folly of extreme surgical treatment by tenotomy which still prevails in many of the analogous conditions, *i. e.*, tics, wry neck, etc. The earliest monographs on the occupation neuroses are those of Zuradelli, in 1857, and Haupt, in 1860.

The recent monographs of most value are those by Remak,[3] 1894, Bernhardt,[4] 1898, and the article on Crampes Professionelles by Meige.[5] Chap-

[1] *The Nervous System of the Human Body*, Washington, 1833, p. 221.
[2] *Bäyer, Correspondenzblatt*, 1840, No. 8.
[3] *Eulenberg's Real-Encyclopädie der gesammten Heilkunde*. New edition, 1909.
[4] *Die Erkrankungen der peripherischen Nerven*, Tiel, ii.
[5] *Bouchardt's and Brissaud's Traité de Médecine*, second edition, 1905.

ters by Gowers, Oppenheim, and Grasset in recent editions of their text-books may also be cited.

**Etiology.**—The chief factor beyond all others is the frequent and continuous use of certain muscular movements in an occupation beyond the natural capacity of the individual to stand the strain. Added to this are usually certain minor or major features which determine why that particular individual should develop the neurosis. In many instances an antecedent pressure neuritis reduces the capacity of certain groups of muscles involved in a coördination, and this throws increased stress on others. In others, faulty methods of adjustment, as in writing in a cramped position, or with improper light, may prove a determining factor for one individual. Mention may also be made of definite physical ills which contribute to reduce nervous tone. These are not unimportant; anæmia, lead poisoning, alcoholism, excessive use of tobacco, venery, diabetes, tuberculosis, the beginnings of serious nervous troubles, paresis, tabes, etc., may be distal causes. Accident, with strain to muscle, tendon, or joint, may be the straw which breaks down the adjustment. Mental causes may show in others; anxiety, worry, financial stress may cause persistent insomnia, which may contribute to an occupation neurosis.

The influence of *heredity* is difficult to trace, for no family is free from some disorder or disease, and the evidence is too flimsy to build upon. When a careful study of a whole community shows that 50 per cent. of the healthy inhabitants have antecedents who have suffered from some mental trouble alone—such as Koller's study of several cantons in Switzerland shows—it is about time to stop prating about the influence of heredity in such affections, especially when it is known that so many minor difficulties constitute the turning point in the development of the disorder. To find an epileptic aunt or uncle, a nervous father or mother, etc., and to conclude that that is the most important reason why one has an occupation neurosis is nonsense. Even in a case of Gowers', in which father and child suffered from writer's cramp, it may be suspected that some faulty structure had been handed down, which if corrected might have cured both.

In well-developed cases, more particularly since there is such a mental element, it is not unnatural that *psychical* causation factors should be postulated. Duchenne advocated the view that mental factors were important. Meige follows more closely the original psychic germ sown by Duchenne, and places the mental factors in the foreground. He believes that the occupation neuroses are essentially allied to the tics, resembling them not only in their objective features, but also in their mode of appearance and evolution in the disposed. In the vast majority of these cases, he holds that the motor trouble appears very distinctly as a consequence of the mental trouble. They may be considered, following him, as a result of mental aboulias or motor amnesias, limited strictly to a determined functional act, and showing themselves by the incapacity to regulate the synergetic contractions necessary to a good execution of this act. Furthermore, it may develop that the act itself gives rise to the fixed idea that the act is impossible to execute. It is well known, he says, if the regular putting in action of a function is favorable to its performance, its overactivity predisposes to perturbance of that function. But even more than the exaggerated repetition of the act itself, the exaggerated concentration of attention on the idea that the act will be badly executed leads to ineffectual

carrying out of the properly balanced function. Thus, a series of motor defects spring up which troubles the mind of the patient, makes him believe in his incapacity to perform the act, and thus leads to the inability to coördinate his efforts. Thus, one sees the irregular muscle developments that enter into play in this disturbance. Certain muscles contract to excess, others not at all, or not at the necessary time. At one time the flexors override the extensors; again, it is the reverse. Meige aptly terms it "a functional anarchy;" the disoriented cortical control gives its commands at the wrong time and at the wrong place; thus, the objective phenomena (chronic contraction, tremors, impotence and pain) receive their interpretation.

The multiplicity of forms described—contracted, spasmodic, ataxic, tremulous, paralytic, etc.—all arise on this common mental basis; they may be found isolated in one patient, or may exist in various combinations. All distinctions between professional and functional spasms, cramps, etc., Meige holds, are arbitrary.

**Occurrence.**—Those coördinated actions of most frequent performance will naturally present themselves the more often as disordered. The practitioner in the large city will practically see more writer's cramp than other types. The laryngologist will see the preacher's, singer's, auctioneer's, etc.; the practitioner of rural districts will see milker's palsies. The medical man in miners' camps will be called more often to treat the pick cramp and miner's nystagmus. Thus, according to locality, relation to definite occupations, etc., characteristic forms will be observed. In every large city there are workers in certain occupations who cluster in communities; in all of these fine manual work is carried on for long hours, under adverse circumstances. Many of these workers develop very severe grades of occupation neuroses, but their study has not been at all systematic.

Many writers say that the disorder is more common in men. This statement is made largely on the evidence afforded by the sufferers from writer's cramp, but since women have taken up telegraphy, stenography, and typewriting, and are engaged so widely in the manufacture of small articles for the trimming of clothing, hats, etc., it would seem that they now show more forms of occupation cramps than men do. Gowers' statement regarding stenographers not developing writer's cramp is erroneous. A limited experience has revealed at least a dozen stenographers, male and female, who have had to give up their work because of occupation cramps.

The *age* of onset is usually after twenty years. Making a combination of the reported cases (194) of Berger, Poore, Gowers, Remak, and Bernhardt, the distribution is as follows: Ten to twenty years, 4; twenty to thirty years, 67; thirty to forty years, 63; forty to fifty years, 45; fifty to sixty years, 10; and sixty to seventy years, 5. To which may be added 46 personal cases: Ten to twenty years, 4; twenty to thirty years, 12; thirty to forty years, 26; and forty to fifty years, 4.

**Occupation.**—Practically every known occupation has its neurosis. To describe them in detail is impossible. Even new sports introduce new occupation neuroses, such as the golfer's "wrist," the tennis "elbow," baseball "glass arm," etc. Blacksmiths, coopers, carpenters, masons, ironers, tailors, and washwomen show the peripheral neuralgic types, usually implying pressure. Among cigar rollers, those who crochet, knit, sew, or cut, rinsers, cabinetmakers, planers, machinists, locksmiths, weavers, chemists (holding beakers and test-tubes), players of musical instruments,

occupation neuralgias, which may never advance to occupation palsies, are known.

**Symptoms.**—Irrespective of the occupation, the development of a professional neurosis seems to proceed along fairly definite lines. Individual variations are found, however, and certain persons, by reason of unknown factors, do not travel as far as others in their journey to the completed stages. It is for this reason that the etiological features upon which Meige lays so much stress may enter into the mastery of the situation. In the majority mild or severe occupation neuralgias are first observed; many patients never have anything else, and a few never have these. Occupation palsies, or impotence, as the French call it, present another phase; these may represent the results of a general or local asthenia and bear no relation to the fully developed neurosis, such as is seen in a typical writer's cramp.

In those forms in which a general asthenia is at the basis of the motor loss, anæmia, cachexia, alcoholism, diabetes, tuberculosis, etc., are often present. Thus, in many occupations which call for localized, restricted, muscular adaptations involving considerable muscular power, as in cigar rollers, engravers, and glassblowers, localized atrophies develop, sometimes as a result of pressure on an exposed nerve, sometimes from other causes. These pressure palsies and pressure atrophies should receive very careful scrutiny. Some of the more characteristic are seen in waiters, in butchers who carry heavy pieces of meat over the shoulder, in others who carry packs, in steel workers, in steersmen who use the arm to push the rudder, in polemen (as seen in the European canals) who push the boat by their long poles against an elbow. Potato peelers, vegetable preparers, drummers, polishers, planers, etc., all show such muscle pareses, accompanied usually by neuralgic manifestations.

For many of those patients in whom atrophies develop, a local cause may be found. Some nerve stem is caught between the structures of the hand and a surface pressed upon; in rare cases perhaps a nerve may suffer from pressure of tendons when such tendons are constantly on the stretch. Hunt[1] describes such a local condition for the ulnar distribution in certain occupations. These local atrophies, when seen apart from the more definite development of muscular incoördinations in a complicated motor act, play a minor role in the true occupation neuroses, and are mentioned solely because such atrophies are sometimes contributory factors in the production of the sense of fatigue, and are the cause of paretic phenomena in others. In the sense that these show pure peripheral origins they differ from the typical cases of occupation neuroses (writer's cramp), although it seems undeniable that transitional stages are to be observed between the peripheral cramp-like states and the centrally induced phenomena.

Remak has called attention to the cramps of sewing girls, which, after preliminary neuralgic-like pains, come on at night. These patients relate that they are awakened out of their sleep by the severity of the painful cramps. He has also laid stress upon certain cramps of milkers, which originate in an apparently different manner from the most classical milker's cramp. Perhaps they are of the same nature, seen either at a different stage of development or in an individual of different neural tendencies. Cases of atrophy of the thenar, hypothenar, and interossei muscles are known

[1] *Journal of Nervous and Mental Disease,* 1908, xxxv, 673.

in milkers, and it is certain that peripheral influences cannot be rejected entirely in the interpretation of certain milker's cramps.

By reason of these and other factors it is not possible always to draw sharp lines between the stages in the development of the professional cramp. Each case seems to travel a slightly different path, and one sees the patients all tending in the same general direction, but at different stages and with varying syndromes. The accidental occurrence of pressure neuritides constitutes one of the commonest accessory factors.

Inasmuch as *writer's cramp* (mogigraphie, graphospasmus, cheriospasmus, crampe des ecrivains, fingerkrampf, schreibekrampf, etc.) is the most frequent of these occupation neuroses, it may serve as an example of the other forms. In the complicated muscular coördination of writing every individual follows his own methods, and no two are ever alike, but, although the actual writing may be different in a million individuals, the general mechanism is largely the same. In general three trends are manifest in writing. There are those who make great use of the smaller movements of the fingers, those who write largely from the wrist, and those whose movements are still wider, employing the whole arm, freehand, as it were. The general grasp of the pen is about the same with all. Those who write a great deal rapidly, such as newspaper men, usually acquire a very large flexibility in their writing, and use all mechanisms in turn. Thus, they can rest certain groups of muscles, and still carry on the writing movements.

Most observers are in accord in ascribing a greater tendency to the development of writer's cramp in those who make greater use of the finer finger movements. Those who write from the wrist and forearm are less liable, while the large freehand writer is usually immune. Gowers is especially emphatic on the harmfulness of resting the hand on the fingers. While these factors of finger position do play a part, they are not *the* most important, and the large arm writer is not necessarily immune. It is by reason of other factors that the training to large arm movements in a "cramper" is often futile.

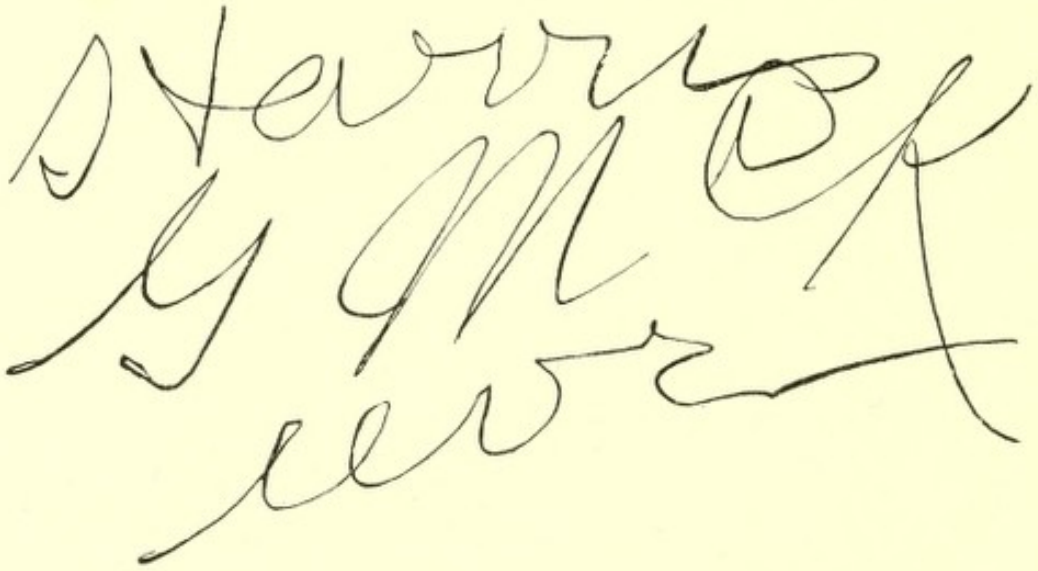
Writer's cramp is no recent disease; above all, it is not a creation of the steel pen, as has been assumed by some good observers, nor of any special type of penmanship. That its clearer description should have coincided more or less with the introduction of the steel pen is accidental, for in the early days the disease was described as occurring among those who still adhered to the use of the old-fashioned quill, and it is known among the Chinese, who still use a brush. The writer has seen one case in a Chinaman, a professional letter writer. The mere fact of the widespread occurrence of so many different kinds of occupation neuroses is enough to negative any such type of inference. Nor can full adherence be given to the view that the disorder originates entirely from a faulty manner of holding a pen or of the use of certain muscles in a definite way in writing. The trouble lies farther back, although it is readily conceded that certain faulty methods of writing are more arduous than others, involve more muscular action, and hence are more likely to cause greater fatigue.

Clinicians have described four chief types—the spasmodic, the tremulous, the neuralgic, and the paretic forms—but these signify little, since nearly all cases show more or less of all of these symptoms. Writer's cramp, in its strictest sense, shows the spastic type in its purest forms.

In a well-developed case the patient, after perhaps years of gradually

increasing difficulty, after writing for some minutes, or perhaps hours, feels a sense of stiffness in his writing. He has neuralgic pains in certain muscles, at the shoulder-joint, the wrist, or in the thumb. A certain rigidity seems to pervade the strokes, and he notes a marked increase in tension in the thumb which grasps the pen. The index finger becomes unruly, the pen is liable to slip, and firmer grasping is resorted to to hold the pen in place. After a short time of rest he may be able to resume his writing for some time before this grade of difficulty again becomes manifest. With increasing severity the troubles become more marked; the firmer grasping of the pen becomes a spasm, at times one of flexion, but more often an extension (Canstatt's two types), the index finger straightens, the pen becomes

FIG. 12



Spastic type, writer's cramp.

more upright, the thumb is strongly contracted to hold the pen against the hand, the spasm extends, the thumb straightens out, and the pen can no longer be held. The writing becomes more and more irregular, scratchy, and scrawly until it becomes illegible.

Other fingers may be involved; in some cases all the fingers are extended, the wrist flexed, the arm brought down to the side of the body, while severe pains radiate throughout the entire arm. Brusque pronation or supination of the arm may then follow. The whole writing apparatus becomes fixed in a tonic (sometimes clonic) battle between agonist and antagonist, all acting out of concert, and mutually disrupting the harmonious relations of a well-planned coördination. Many patients write for years, with but the sense of stiffness and some pain. Others, on the contrary, develop acute spasms, and no sooner commence to write than the cramp develops. In most of the fully developed cases considerable mental excitement is present. Vascular disturbances (perspiration) may be distinct.

The character of the spasms is usually tonic, and they develop slowly. Some patients have jerky movements somewhat resembling clonic contractions, but these seem less common. Tremor is an almost invariable accompaniment, and there are writers for whom only tremors exist.

It is usually conceded that of the predominant types, the spastic form is most frequent; the feeling of great weakness and soreness in the muscles, which is antecedent to all forms of writer's cramp, is not considered here. The spastic form alone, or in combination with tremors makes up about 40 per cent. of the cases. Berger,[1] in his analysis of 64 cases, found that in 24 there was a pure spastic type; 10 showed the paralytic type, 8 had tremors, while the remainder, 22, had combinations; over 50 per cent. had cramps. In Remak's 42 cases there were 9 cases of true writer's tremor; while 32 showed more or less spastic symptoms. He had observed only one pure paralytic case. Gowers has also noted the rarity of the paralytic forms.

It is highly significant that the moment the pen is laid aside the cramp ceases, the patient can move his hand or arm in any desired direction, and apart from, at times, a mild sense of tire in the affected muscles, all signs of previous difficulty vanish. The patient can carry on coördinated movements involving the very muscles which were in such a painful state of contraction while performing the single act of writing.

Benedikt is stated to be the first observer to record the tendency that exists for the disorder to spread to other frequently repeated intricate coordinations. This is true for those particularly in whom the psychical element plays a major role, but is less apparent in other types. Thus, patients with well-developed writer's cramp may still be able to play the piano, do fine sewing, embroidery, etc. On the other hand, in rare instances the patients become more or less helpless, and cannot carry out any fine manual coördinations.

In the *tremulous type*, which in its pure state is comparatively rare, the patient's hand begins to shake with the act of writing, the writing is tremulous and shaky, like that of an old person, the whole arm may be involved in the tremor, and patients are known who, without pain or other disturbance, have written such a tremulous hand for thirty or forty years. They have never lost the power of writing, but must always write with a distinct tremor.

In the *paralytic type*, which is rare in the pure form, the patient loses the power to grasp the pen. There is a stage of rapidly advancing fatigue and weakness, up to a point when paresis without spasm or tremor, and often without severe pain, save that of tire, is reached. The fingers become stiff and inactive, as though nailed to the table. After a few moments' rest the patient can resume his writing, to be again interrupted by the rapid advance of paresis. The localization of the paretic muscle groups may vary. Berger reports a case of isolated shoulder and upper arm type. Simple abducens pollicis paralysis is known. Duchenne's case of infraspinatus palsy is classical; here the patient could write if the paper was pulled along by the left hand, but the carriage of the arm across the page was impossible.

*Sensory Disturbances.*—Such are usually present if only as a distressed sense of tire, of pressure, of tension. Enough has been said of the neuralgic pains and the pains of tonic contractions. Anæsthesia and paræsthesia are present, although infrequent; and hyperæsthesia with painful nerve trunks may be present, especially in the neuralgic types. Such painful

[1] *Beschäftigungsneurosen, Eulenberg's Encyclopädie,* 1885, vol. ii.

nerve trunks with the occurrence of Lasègue's symptom in the lower extremities may clear up the diagnosis in mild alcoholic cases.

Vascular disturbances, either local, as angiospastic phenomena with cold hands, or as paralytic phenomena with localized sweating, are rare. General anxiety with vascular disturbances is not as infrequent.

Electrical changes are not of frequent occasion; when present they indicate the neuritic nature of an occupation palsy, and thus aid in the diagnosis.

**Course and Prognosis.**—The outlook is bad if one has in mind only those cases which are construed to be occupation palsies in the narrow sense. When one takes into consideration, however, all of the forms, especially those of widely varying etiology, the prognosis is good. This is especially true for the alcoholic and pressure types, while in the purely mental types the reverse is true. Most patients with fairly well-developed professional neuroses run an up-and-down course with fairly well-marked chronicity. With improvement in general strength and rest, the professional movements are better carried out, and vice versa. Some patients rest for a year or more, and develop their neurosis within a week after resuming their occupation. In such cases the mental factors are predominant. These patients need education, not rest. In some patients cure seems impossible, either due to defect on the part of the patient to grasp the rationale of a course of therapeutic effort, or a lack of proper analysis of the case on the part of the attending physician. In severe cases of writer's cramp the course is especially obstinate, but not always hopeless.

**Treatment.**—Personal factors enter so largely into this that the individual must always be borne in mind. Occupation neuroses develop on the one hand on a basis of overwork of certain muscular coördinations, without any other complicating factors; at the other extreme patients will be seen in whom the psychical factors are predominant or have become so, and the fatigue element is practically nil; occupying a partial middle ground are those in whom constant, minute maladjustments are to be borne in mind. Few individuals will present examples of the extremes; the vast majority will present combinations in varying proportions of overwork and mental attitudes with certain intermediary asthenia. These should receive the fullest attention.

There is little doubt that most patients—save those in whom the psychical features are markedly developed—will receive benefit from the general methods of increasing physical vigor. A sea trip, change, etc., are here indicated. Alcohol and tobacco are to be reduced to a minimum.

Abstinence from writing, in the writer's cramp form, is the first requisite. Many clinicians, Gowers among them, recommend the advisability of teaching their patients to write with the left hand, thus affording the right hand an opportunity to rest. This may effect a comparative cure, but not infrequently such patients develop double writer's cramp. Such an evolution is presumptive evidence of a large psychic element, and for such the methods of Meige are particularly advisable. Methods of reëducation of the writing are also desirable in other cases; changes in the form of pen, using a large penholder, stub pens, changing the slant of the writing, learning to rest one's entire arm on the writing table, and to utilize the larger muscles in the writing act—these, with general hygienic tonic procedures, are at times sufficient to afford sufficient variation for an affected individual to just steer clear of an actual breakdown. Great care must be exer-

cised in this direction. With definite mental features in the case, such schemes are a waste of time, and may be bad psychotherapy.

In the large class in which accessory elements play a part, *i. e.*, congenitally weak muscles, pressure and toxic neuritides, prophylactic measures combined with direct methods of nerve and muscle treatment are needed.

Strychnine, calcium salts in the form of hypophosphites, iron, atropine, galvanization, mechanotherapy, massage, etc., are useful. Bier's hyperæmic procedures are helpful.

Special electrical methods may be desirable. Many patients are benefited by the Leduc oscillatory currents. The role played by complicated methods of electrical stimulation is difficult to estimate. In those patients for whom such electrical stimulation acts as a general tonic, its action is evident; in others the suggestive feature should not be overlooked; in others it is useless or worse. A few of the specific modes of utilizing electricity may be recommended; for instance, the stable galvanic current of medium intensity, the positive pole being applied to the neck, the negative pole to the supraclavicular fossa, to the brachial nerves, and to the affected muscles in turn for periods of from five to ten minutes daily. These applications may have to be continued for months. Eulenberg praises the use of local galvanization to the muscles affected by the tonic cramp-like contractions, the positive pole being affected to the involved nerve stem. He found that in those cases in which there was marked tremor the best results were obtained by applying the negative pole to the spinal column and the positive pole to the involved muscles and nerves at the periphery. Erb even recommended passing the galvanic current through the head. Others stimulate the sympathetic. All the writers who find electricity beneficial report that it must be used for months, which is largely indicative that it is solely a general measure and really has little direct value. Local hydrotherapeutic, mechanotherapeutic measures seem to promise more; they are frequently combined with galvanization to advantage.

Active and passive *gymnastics* combined with vibration may be practised in most cases to advantage. These should always have for their object the bringing about of new associations in the muscular activities, which can counteract the cramp movements. It becomes imperative to work out in minute detail the specific cramp movements, and then have the patient practise with great regularity the opposing mechanisms to such movements. Thus, if the extension of the index finger is one of the first of these cramp movements to be set in motion in the sufferer from writer's cramp, frequent practice of a contrasting or an opposite movement is desirable, until the entire cramp mechanism has been learned, as it were, in its opposite form. This specially developed type of muscle training, with its implied training of the cortical impulses, promises the most permanent results. The brain writing mechanism must be trained out of a habit of incoördination, and into a new coördination. The older methods of learning new methods of writing only took the second factor into consideration; it is imperative to deal with the whole faulty mechanism.

## TETANY.

This peculiar motor manifestation has had a variety of names since Corvisart, in 1852, termed it "tetanie." Other terms applied by various

writers have been tetanilla (Comte), intermittent tetanus, spasmodic muscular retraction, spasmodic myelomeningitis, essential contractions, tonic occupation cramps, and idiopathic muscular spasms. Frankl-Hochwart, in 1887, clarified the conception, and since his striking descriptions the term tetany has had universal recognition.

**Definition.**—It cannot be said at the present time just what tetany is, or rather, perhaps, it might better be expressed, that so many different tetanies are known that it is evident that, like epilepsy, it is not a single affection but many; a symptom grouping, very variable as to completeness, which is best regarded as a more or less localized manifestation of a convulsive nature, due to a large number of exciting causes, possibly with some single and unique underlying factor. Whether such a position can be maintained for the so-called idiopathic epidemic form remains to be seen. If the present view be maintained, however, the term tetany must be rejected from nosology as a disease conception in the vast majority of the conditions which now go by that name, or perhaps preferably a binominal nomenclature might be adopted which would specify the type under consideration. Thus, it might be advisable to advocate the use of the terms tetany idiopathica, tetany gastro-enterica, tetany thyroidea, tetany toxica, including bacterial and chemical toxins, tetany gravida, etc., until it may be shown just what underlying factor is responsible. Inasmuch as it appears that a certain grouping of cases occurs about some definite etiological factors there are probably some definite neural paths of preformed diminished resistance which permit of the similarities of reaction from many different sources of irritation. The similarities of muscular reaction are more often hypothetical than real, but this is not to be wondered at considering the intrinsic difficulties of observation, particularly in children.

It is highly probable that tetany must be regarded as an example of hyperfunctioning of certain parts of the motor mechanism in which altered muscular excitability is primary, while special neurological features play a secondary role, and yet these latter give to the muscular manifestations their special trend. Here particular attention is paid to the etiological factor, since the clinical manifestations alone are not always sufficient to distinguish the form of toxin present.

Frankl-Hochwart,[1] speaking more particularly of the adult forms, is more didactic in his presentation. Tetany, he considers, has as its main symptoms tonic, intermittent, bilateral, often painful cramps, which, without, for the most part, any loss of consciousness, involve the muscles of the upper extremities, particularly the hand, which is held in the obstetrical position. The muscles of the lower extremities may also be involved, those of the larynx, of the face, and of the jaw, seldom those of the chest, abdomen, neck, diaphragm, or tongue. In rare cases the eyeball muscles are implicated, as is also the bladder. In the sensory sphere paræsthesia and pains are present, while hyperæsthesia occurs now and then. Pressure upon the brachial plexus may give rise to an attack (Trousseau); hyperexcitability to electrical currents is present (Erb); mechanical hyperexcitability of the muscles and motor nerves is observed (Chvostek), while the sensory hyperactivity to mechanical and electrical stimuli is also present (Hoffmann). The psyche is usually uninvolved, mental disturbance being

[1] *Die Tetanie der Erwachsenen*, 1907.

found only now and then. In the chronic and repeating forms secretory and trophic disturbances occur, such as increased perspiration, reddening of the skin, swellings of the joints, mild œdema, falling out of the hair and nails, discoloration of the skin, urticaria, and herpes. Dyspnœa may supervene; polyuria and glycosuria are rare accompanying symptoms. Abortive and incomplete forms this author designates as tetanoid.

**Historical.**—The early history of tetany is lost in antiquity. Some French writers are of the opinion that in the writings of Hippocrates may be traced the earliest mention of this affection, and quote the following: "The wife of Philistidi Eraclides was attacked by an acute fever, with redness of the face without any manifest cause; after that day she stiffened, and spasm appeared in the fingers and toes; gradually this increased and more stiffening appeared; soon after, the redness became less hot, and the convulsions became more moderate." Later Latin writers are also cited.

De la Motte,[1] de la Roche, Tissot, and Ramel all described cases at the end of the eighteenth century. In Etmüller's writings (1708) one finds the phrase "Morbus hungaricus spasmus extremorum," which is very suggestive of the probable greater frequency of this type of affections in these lands, while Wolf, in 1717, describes similar affections in Saxony. Among English writers, Clarke, in 1815, was the earliest to call attention to laryngeal spasms, accompanying the spasms of the extremities.

The modern era sets in with the studies of Steinheim, a physician of Altona who noted symptoms following removal of the thyroids (1830), and of Dance in 1852. The term intermittent tetanus was used by them for the first time. In the period immediately following were many studies; Tournelle, Constant, Murdoch, Tessier, Imbert-Gourbeyre, were among these earlier workers. Corvisart, in 1852, gave us the name as applied to-day. Trousseau, about the same time, began his active researches, and in the years from 1851 to 1860 described and elaborated the symptom which bears his name.[2]

The work of the German school, which began with Keyler in 1837, in 1872 brought forward by Kussmaul[3] the relations of stomach tetany to other types. Erb,[4] in 1874, described the electrical phenomena. Chvostek,[5] in 1872, described his symptom, and gave the great impetus to the Viennese students of this affection which has culminated in the monographic and complete researches of Frankl-Hochwart.[6]

For didactic purposes Frankl-Hochwart divides tetany into simple acute forms and chronic recurring forms. A further division of forms occurring in children and in adults is made. Tetany of the adult he groups into eight classes: (1) Tetany idiopathica—tetany of otherwise healthy individuals —workman's tetany. This is the form which seems to occur epidemically as an acute, or acutely, recurring affection in certain cities, notably Vienna, Heidelberg, etc., principally in the early spring months, and among certain handworkers—tailors, shoemakers, etc. (2) The tetany of gastric and intestinal affections. (3) The tetanies of acute infectious diseases, typhoid

[1] *Lehrbuch der Geburtshülfe.*
[2] *Gaz. des Hôp.*, 1856, 1858; *Bull. de Thérap.*, 1860.
[3] *Berl. klin. Woch.*, 1872, No. 37.
[4] *Archiv f. Psychiat.*, 1874, No. 271.
[5] *Wien. med. Presse*, 1876, p. 1201 et seq.
[6] *Die Tetanie* 1907. An exhaustive monograph in its second edition.

fever, cholera, measles, scarlet fever, etc. (4) The tetanies of acute poisoning, chloroform, morphine, ergot, phosphorus, lead, renal and genital substances. (5) The tetanies of maternity (pregnancy, parturition, and nursing). (6) The tetanies of parathyroid involvement. (7) The tetanies of other nervous diseases, exophthalmic goitre, brain tumors, cysticerci, syringomyelia, etc.

Whether all of these have a common etiological factor is a problem that has not yet been definitely solved. It would seem that from the work of MacCallum a deduction might be made allying the various forms—namely, that a disturbance of calcium metabolism of the body is responsible for the hyperactivation of the neuromuscular responses which results in the tetany spasms.

**Incidence.**—Tetany in its different manifestations is undoubtedly rarely seen and is even less frequently reported. In undeveloped phases the tetanoid reaction is comparatively frequent in children. The instances of gastric tetany are probably the most frequent, while the pure epidemic form has not been encountered in the United States. In Griffith's study only 77 cases were found recorded, while Howard's later collection brings the American cases to 154 in 1907. The incidence in the Johns Hopkins Hospital has been 0.05 per cent. Thus, so far as clinicians in English-speaking countries are concerned, tetany may be considered as being infrequently seen, but even in countries in which it is thought to be epidemic it is rarely observed. It is undoubtedly often overlooked and at times confused with hysteria. Mattanschen[1] has shown that in the various garrisons of Austria the disease is extremely rare. Thus, from 1895 to 1905, only 90 cases of the condition have been reported in a total population of 100,000 soldiers.

**Etiology.**—At least two factors are concerned in the general hyperexcitability of the neuromuscular apparatus and the neural trend that determines the manifestations of this hyperexcitability in certain groups of muscles and for certain combinations. So far as the first factor is concerned, it would seem that a fairly clear concept has been gained, particularly through recent work, while for the second no reasonable explanation is forthcoming.

As regards the epidemic tetanies, Schulz and Hoffmann advocated the thesis that they were due to disturbances in the thyroid gland; this was in the time when the parathyroids were unknown. The fact of the close anatomical relationship of the two glands in man and many carnivora served to hide the deeper truth of the separation of their physiological functions first brought out by Gley in herbivorous animals, in which the glands are anatomically separable and thus capable of exact experimentation. It is unnecessary to go through all the steps which have led to the knowledge that the parathyroids play an important role in the functions of the body, and that the peculiar muscular hyperexcitability seen more particularly in tetany is in some manner related to these glands, especially in regard to their insufficient action. It is possible that other anomalies of neuromuscular activity are correlated with these glands. Lundborg has advocated wide hypothetical possibilities; even the peculiar motor manifestations of a group of mental disorders, katatonias and their allies,

[1] *Wien. klin. Woch.*, 1907, xvi, 470.

may have some light thrown on them through this avenue. Berkley[1] has begun such a research.

More recent workers have not rested on the apparently certain foundation that tetany is essentially a manifestation of perverted parathyroid activity, but have sought to bring the anomalous forms into conformity with this conception, and, further, to obtain a more fundamental insight into the essential features of the disturbed neuromuscular reaction. Whether the work of MacCallum and Voegtlin[2] has definitely solved this problem is to be determined, but it would appear that the essential factor has been found in the relation of the parathyroid to the calcium metabolism of the body. The hyperexcitability of the neuromuscular apparatus is primarily due to a lack of calcium in the blood, and this is due to a relative or absolute insufficiency of the parathyroidal glands. Just how the calcium exchange of the body is controlled by the parathyroids, and what role in such control is played by other glands, is not yet known. The conclusions of MacCallum and Voegtlin are so important that they are worth quoting freely:

"Tetany occurs spontaneously in many forms, and may also be produced by destruction of the parathyroid glands. These glands are independent organs with a definite specific function; whether this function is intimately related to that of other organs of internal secretion is not as yet proven. The number and distribution of the parathyroids varies greatly. Failure to produce tetany experimentally is probably due to the fact that some parathyroid tissue remains after an apparently complete extirpation. A small amount of parathyroid tissue is sufficient to prevent the development of tetany.

"In tetany there is apparently some disturbance of the composition of the circulating fluids ordinarily prevented by the secretion of the parathyroids, which disarranges the balance of the mineral constituents of the tissues. Possibly this consists in the appearance of an injurious substance of an acid nature, for such tetanies may be relieved by extensive bleeding, with replacing of the blood by salt solution. No actual poisonous material has, however, been demonstrated by the transference of the blood of a tetanic animal to the veins of a normal one.

"Numerous researches have shown the important relation of the calcium salts to the excitability of the nervous tissues. Their withdrawal leaves the nerve cells in a state of hyperexcitability which can be made to disappear by supplying them with a solution of a calcium salt.

"Tetany may be regarded as an expression of the hyperexcitability of the nerve cells from such cause. The injection of a solution of a salt of calcium into the circulation of an animal in tetany promptly checks all the symptoms and restores the animal to an apparently normal condition.

"Studies on the metabolism in parathyroidectomized animals show: (*a*) A marked reduction in the calcium content of the tissues of the blood and brain during tetany; (*b*) an increased output of calcium in the urine and fæces on the development of tetany; (*c*) an increased output of nitrogen in the urine; (*d*) an increased output of ammonia in the urine, with an increased ammonia ratio in the urine; (*e*) an increased amount of ammonia in the blood.

[1] *Amer. Jour. of Insanity*, 1909, lxv, 415.

[2] See *Jour. of Exper. Med.*, 1909, vol. xi, for the most recent summaries along the lines of chemical research.

Much of this affords evidence of the existence of some type of acid intoxication. Its effects are, however, not neutralized by the introduction of alkaline sodium salts, and may perhaps be regarded as especially important in producing a drainage of calcium salts from the tissues which can be remedied by the re-introduction of calcium salts.

"Emphasis must be laid on the remarkable difference which exists between the alterations in metabolism following thyroidectomy and those following parathyroidectomy, and in all experimental work great care must be exercised in separating the glands." Berkley has seemed to neglect this important point in his work on katatonics.

"In general, the role of the calcium salts in connection with tetany may be conceived as follows: These salts have a moderating influence on the nerve cells. The parathyroid secretion in some way controls the calcium exchange of the body. It may be supposed that in the absence of the parathyroid secretion, substances arise which can combine with calcium, abstract it from the tissues, and cause its secretion, and that the parathyroid secretion prevents the appearance of such bodies. The mechanism of the parathyroid action is not determined, but the result, the impoverishment of the tissues with respect to calcium, and the consequent development of hyperexcitability of the nerve cells, and tetany is proven. Only the restoration of calcium to the tissues can prevent this. This explanation is readily applicable to spontaneous forms of tetany in which there is a drain of calcium for physiological purposes, or in which some other condition causes a drain of calcium. In such cases the parathyroids may be relatively insufficient."

This sums up a series of studies which have been pointing in this direction since Gregor, in 1900, and, more particularly, Sabbatini, in 1901, called attention to the action of calcium salts on nervous irritability. The results of Parhon and Uréches' work with dogs largely confirms the studies of MacCallum and Voegtlin here quoted, although Parhon has also found nonconfirmatory evidence.

It has been shown, moreover, that salts of barium and of strontium are capable of controlling the spasms of experimental tetany. While this does not constitute a real difficulty in the interpretation of the results just cited, it remains to be shown just what pharmacological relationships these salts have on nerve cells. In view of their close relation to calcium, it may be that they temporarily take on the function of calcium.

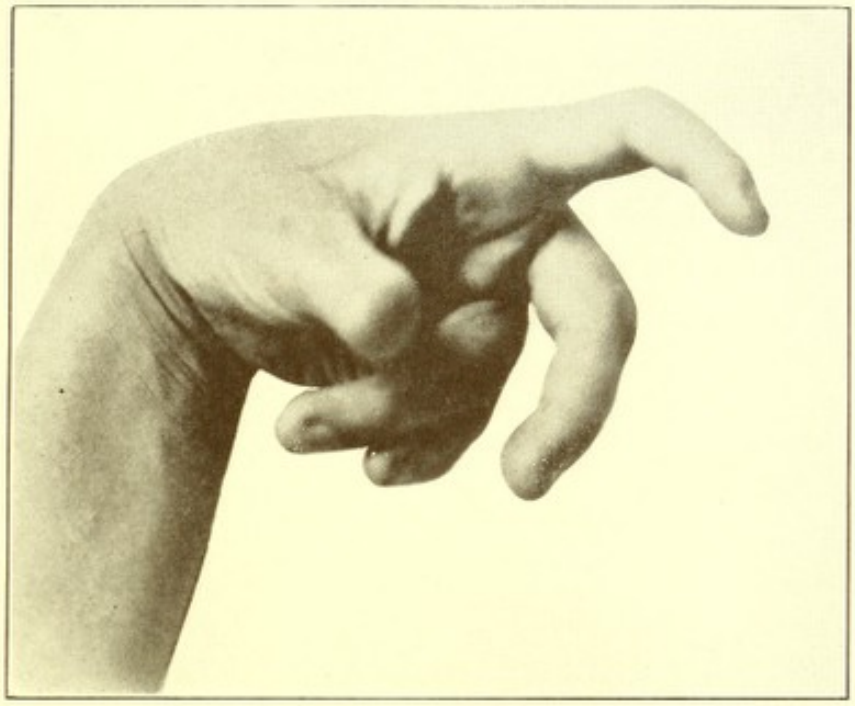
The second series of factors still remains to be solved. Why is it, if the facts just quoted bear an essential relation to tetany the disease reaction seems to be so strictly localized? Is this simply bad observation on the part of the clinicians, or are there local conditions of diet, water supply, etc., that bring about changes in the intake of calcium? Such conditions are known to prevail for certain infants fed on cow's milk, and have been extensively studied.[1]

Why do certain workers, tailors, shoemakers, etc., suffer and not others? Is Frankl-Hochwart's suggestion regarding the peculiar position adopted by these workers of value in relation to the blood supply of the organ? What have gastric dilatation, gastro-intestinal disorders, etc., to do with the calcium intake? Are there perversions of gastric chemism that favor calcium exclusion? This must be determined in order to explain these cases, and why

[1] See Escherich's Monograph, *Tetanie der Kinder*, 1909, for most recent summaries.

# PLATE XXXIV

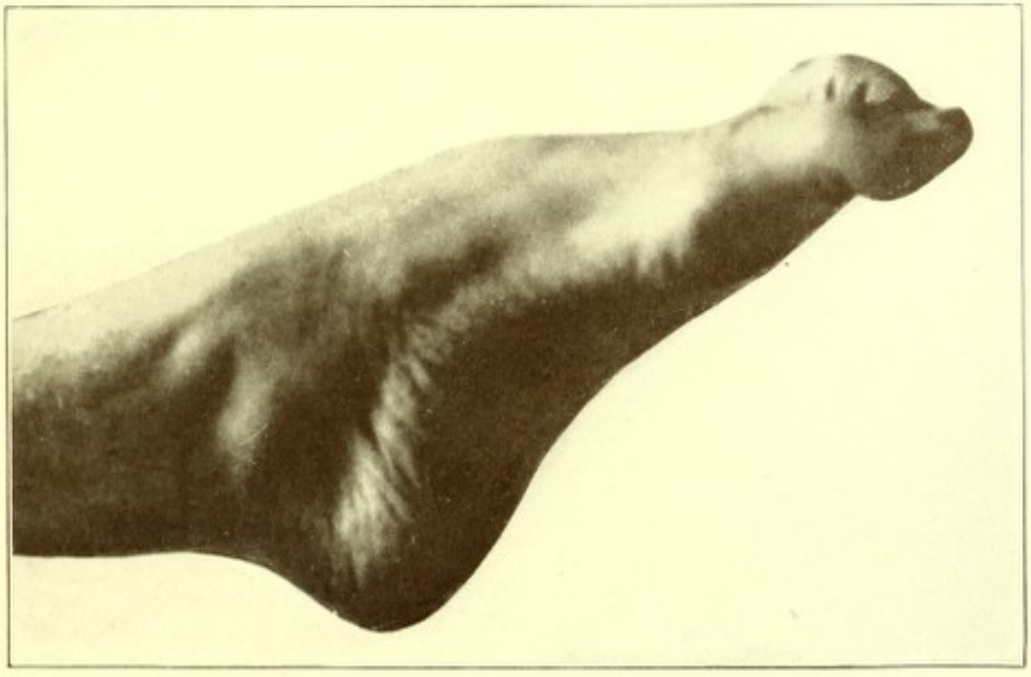
FIG. 1



Tetanic Spasm.

Showing flexion of wrist.

FIG. 2



Tetanic Spasm.

Showing plantar flexion of foot and toes.

should certain gravid women develop a parathyroid insufficiency? The observations of Lange, Jeandelize, and others on thyroid activity at this period are only suggestive.

Still further, why should the hyperexcitability not be more generalized, and bring about general epileptiform conditions instead of very localized spasms, and further only in certain groups of muscles. These must be more thoroughly explained before the general hypothesis can meet with unqualified recognition, for the symptoms seem to call for a specific rather than a general type of poisoning. The MacCallum hypothesis takes this into account, but the answer is still to be sought.

**Pathology.**—Concerning the histological changes the present view excludes a specific pathology. The insufficiency of the parathyroids, be it relative or complete, may be brought about by a great variety of lesions. Such have been described by various observers, and each, in turn, has been considered of specific significance. These in reality offer evidence in favor of the parathyroid insufficiency hypothesis, but go no further. In the minor grades of tetany in children, particularly in so-called spasmophiles which Frankl-Hochwart regards as tetany, the findings of Yanase in Escherich's clinic are illuminating. Here hemorrhages in the parathyroids seemed fairly constant findings, and offer an explanation of the galvanic hyperexcitability. At the other extreme one finds the absolute insufficiency tetanies in experimental parathyreopriva and in the rarer cases, such as Pool's in man. Here the parathyroid insufficiency is established, and the calcium treatment restorative. In acute epidemic forms thyroid (and probable parathyroid) involvements are known. Tumors, tuberculosis, and a host of other changes in the thyroids have been described. It will probably be found that in most of these the parathyroids are likewise implicated. Thus, in exophthalmic goitre a combination of thyroid and parathyroid symptoms is often present. In many tetanies pure thyroid symptoms appear.

The relations of the ganglion cells in the spinal cord which stand in direct connection with the sympathetic system are in need of analysis. Only a few of these groups have been localized, for instance, Jacobson's localization for the Klumpke syndrome.

**Symptoms.**—Considerable variation is to be found, but in general four types of symptoms are observable in the fully developed attack. These are the muscular spasms, which may go on to an exhaustion paralysis, or paresis; the Trousseau phenomenon; increased electrical excitability, or the Erb's symptom; and mechanical hyperexcitability of the muscles—Chvostek's sign. In some patients one or more of these may be missing. Abortive forms, so-called, may present even fewer signs. On the other hand, a richer combination of symptoms, apparently closely related to the general disorder, may be encountered. Sensory disturbances, anomalies of circulation with œdema, of respiration with cyanosis, and of temperature are sometimes found. True psychoses, perhaps indistinguishable from the hysterical confusions, are found. Trophic disorders of the skin, hair, and nails occur. In some rare instances widely confused phenomena resembling epileptic seizures occur.

**Muscle Cramps.**—The most persistent feature consists in a characteristic form of muscular spasm. It occurs chiefly in the extremities, mostly involving the flexors. The spasm is tonic, generally bilateral, and is usually

induced by some irritation, mechanical or electrical. Overexertion, exhaustion, changes in temperature, acute diarrhœa, or emotional excitement may precipitate an attack. Consciousness is involved only in certain forms (parathyroid tetany); pain may or may not be present. The small muscles of the hand are usually first implicated, perhaps after tingling-like prodromata. There is marked adduction in the interossei and the thumb. The hand usually takes a very significant position—that of the "obstetrical hand." It is also described as a "penholding" position. In many mild attacks only the thumb may be involved, and in others the hand alone. Sometimes the hands are closed, making a fist. Flexion at the wrist may follow, the arms then being folded across the chest, or they may be held up in the air or down at the side. Such wider movements occur in the more severe attacks only. A somewhat similar series of flexor cramp-like movements may occur alone or in combination in the lower extremities. Talipes equinovarus, inversion of the foot, and plantar flexion may be present. The legs may be flexed on the thigh, and the thigh on the pelvis in the most severe instances. The contractions in the lower extremities are rarely as severe as in the upper, and frequently are lacking altogether. Escherich considers the classical carpopedal spasms to be rare in children.

In Frankl-Hochwart's series of 122 cases, only 70 showed cramps in the lower extremities. Occasionally a patient has cramps in the upper and paræsthesia in the lower extremities. Frankl-Hochwart makes the assertion that there are no cases on record of tetany confined to the lower extremities. In Howard's series,[1] however, and these are from the United States, all four extremities were involved in 61 out of 77 cases; in 14 the arms alone were involved, while in 2 the spasms were confined to the legs, in which latter respect Frankl-Hochwart's claim is negatived. Other spasms may occur. Thus, risus sardonicus, 11 in 122 in Frankl-Hochwart's series, and trismus, in 10 of Howard's, may both be found, although, as a rule, these rarely occur spontaneously, but are invoked by tapping over the facial nerve region in bringing out the Chvostek or Hoffmann phenomena. The back muscles were involved in 24 of Howard's series; at times to such an extent as to cause opisthotonos.

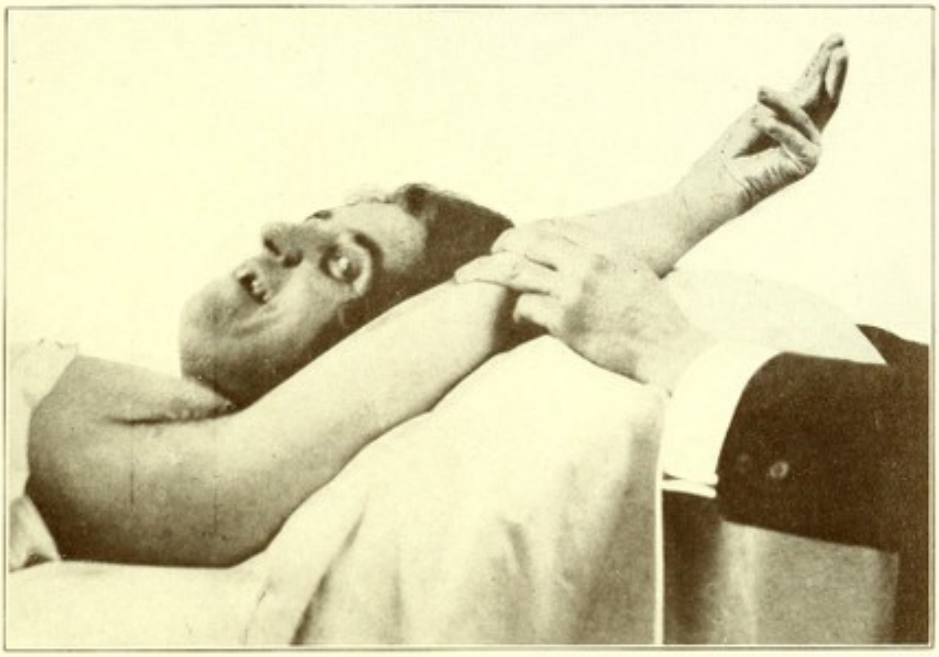
Other muscular spasmodic contractions are rare, although it may be said that any muscular contraction may be expected. It is highly probable that sharper delimitation of the tetanoid from the hysterical and epileptoid spasms is needed, and that closer observations will negative the broader statements now usually accepted. Some of these rarer contractions occur in the bladder, the rectum, the diaphragm (which has caused asthmatic attacks), and the muscles of the tongue. *Laryngospasm* is one of the commonest forms, especially in children, where it has been held as specific for the rachitic. Whether it is to be regarded as a specialized symptom in tetany, or as an individual expression of other conditions, is not at present definitely determined. Spasms of the eye muscles, sufficient to cause diplopia, occur. In 7 of Frankl-Hochwart's series these eye muscle cramps were present. They occur spontaneously. Generalized convulsions are reported in 13 of Howard's cases; 9 of these, however, were in children.

Considerable emphasis may be laid on the bilateral character of these

[1] *Amer. Jour. Med. Sci.*, 1906, cxxxi, 301.

# PLATE XXXV

FIG. 1



Method of Producing Tetanic Spasm of Hand by Stretching the Brachial Plexus by Forcible Abduction of the Arm. Note "Obstetrical" Hand.

FIG. 2

Method of Producing a Tetanic Spasm in the Feet by Stretching the Sciatic Nerve by Forcible Flexion of the Trunk on the Thighs.

muscular movements. It is extremely rare in the true tetany reaction to find one side alone involved; Frankl-Hochwart reports two cases only.

An incidental feature of some of the hand cramps is the occurrence of an intervention cramp in the same sense as it occurs in Thomsen's disease. The co-existence of the two conditions has been reported.

These muscular cramps persist for a very variable length of time. Not only do they vary in different individuals, but the same patient in different attacks, or at different times in the same attack may show marked variation. In the majority of cases reported the spasms persist for from fifteen minutes to an hour, and two or three hours is not an excessive period. Hoffmann has reported a persistent cramp which lasted for ten days. In fatal cases the contractions pass over into a lethal continuous spasm.

Clonic spasms are rare, but are known as blepharospasm, spasms of the tongue, etc. Postconvulsive paralysis or paresis is an uncommon outcome. Excessive muscular tire, however, is not rare.

**Trousseau's Phenomenon.**—To be able in a free interval to induce an attack, or to increase the attacks in force and frequency during their continuance by pressure upon a nerve trunk, or upon a bloodvessel, is the essential feature discovered by Trousseau. Frankl-Hochwart has almost annihilated the specificity of the conception by stating that pressure anywhere on the body is capable of inducing the attack. This is true in a relative sense only, since sites of election are of primary importance. These are the bicipital sulcus and over the crural artery; a strong squeeze of the bones of the hand may be sufficient. One grasps the arm with the thumb on the outside and the fingers pressing upon the region of the brachial plexus. The pressure must be distinct, and not fleeting. It may be necessary to press for at least five minutes. In Howard's American series this phenomenon was present in about 80 per cent. of 45 cases; in Frankl-Hochwart's, in 62 per cent. of 122 cases. The significance of this symptom is unknown. Is it a result of the combined pressure of artery and nerve, as claimed by Trousseau, or is it a sign of anæmia, as claimed by Kussmaul and others? Inasmuch as a bilateral spasm is set up by a unilateral anæmia, it seems to call for a wider interpretation, and Schlesinger[1] has arrived at the hypothesis that it is a reflex.

Ferenczi[2] upholds the anæmia etiology, since he has demonstrated that by simply holding the arms high extended over the head this reduction of blood supply will result in spasmodic contractions. If Frankl-Hochwart is correct in his broadening out of Trousseau's concept, since pressure on a bone may bring about the spasm, the anæmia hypothesis seems to lack support. Furthermore, his studies on animals would seem to require in explanation a direct nerve stimulus, rather than an alteration in the blood supply. The real difficulty lies behind in the hyperexcitability of the nerve impulse. Trousseau's phenomenon is often well marked in hysterical patients, and in the more classic types of hysteria.

**Electrical Hyperexcitability.**—This is present in (*a*) motor nerves (Erb), (*b*) sensory nerves (Hoffmann), and (*c*) nerves of special sense (Chvostek, Jr.).

(*a*) *Motor Nerves.*—Erb first showed that minimal electrical stimulation (0.5 to 2 milliamperes) brought out the tetany spasm reaction. Later

[1] *Neurol. Centralbl.*, 1892, p. 66.

[2] Ibid., 1904, p. 294.

studies of Weiss and Frankl-Hochwart showed the constant hyperexcitability to the galvanic current, while the reactions to the faradic stream were inconstant. A large number of anomalous electrical reactions occur in the motor nerves of these patients. Thomas[1] has described a paradoxical "catelectrotonus tetanus." With cathodal stimulation to the nerve, with currents too weak to be recorded by the galvanometer, one could notice fibrillary contractions in the muscles, which, with the increase in current, became more marked until tetanus developed. This spread to all the muscles supplied by the nerve and ceased on the closure of the current. Anodal stimulation was negative.

Peters[2] has described a "jumping jack" sign brought about by galvanism of the spine, the anode being applied to the sternum, the cathode over the cervical or thoracic vertebræ; minimal currents (3 to 4 milliamperes, even at times 0.5 to 1 milliampere) may cause the arms and legs to jump at each closure of the current. He found that the contractions may be induced on both sides if the cathode and anode are placed directly on the middle line, whereas, if they are placed on one side of the spinous processes the contractions will be limited to that side. In the mild cases the phenomenon is not induced. In such cases the use of an Esmarch bandage will aid in bringing it out. After lumbar puncture it is impossible to bring out the sign.

(*b*) Hoffmann's researches showed a marked hyperexcitability of the sensory nerves; mild currents causing paræsthesia or pain; while (*c*) Chvostek, Jr.,[3] showed that for relatively mild currents distinct auditory sensations were elicited by opening and closing the current. Taste perceptions react in a similar manner, while the optic apparatus does not show any reactions. Weiss reports cases in which there is no electrical hyperexcitability, but Frankl-Hochwart says that he knows of no acute case that shows normal electrical reactions.

4. **Mechanical Irritability of Nerves and Muscles.**—Chvostek first showed that simple mechanical stimulation of a nerve trunk is sufficient to induce a spasm. Sharp tapping over a nerve trunk, or even over a muscle, is capable of inducing a typical flexor spasm. The favorite site for bringing out Chvostek's sign is over the facial nerve in front of the ear. It is not always present, and in 40 of Howard's patients tested, only 50 per cent. responded positively, but the figures of Chvostek and of Frankl-Hochwart are much more definite. The latter author classes it as one of the most important signs of the tetany reaction, although it is not always present. He further distinguishes three grades in the Chvostek phenomenon. In the more severe reactions (1) a tap in front of the ear will cause contractures in the entire facial innervated musculature. Even light stroking may set free the muscular contractures (Schultze's phenomenon). In the middle grade (2) a stroke of the hammer causes contraction of the nasolabial folds, while in the lightest grades (3) the corner of the mouth alone contracts after percussion in the classical spot. In his series Chvostek, No. 1, was found in 39, No. 2 in 58, and No. 3 in 19; in 11 patients it was entirely absent. This is particularly true for some of the more chronic patients in whom

[1] *Johns Hopkins Hosp. Bull.*, 1895, vi.
[2] *Deutsch. Arch. f. klin. Med.*, lxxvii, 69.
[3] *Wien. klin. Woch.*, 1890, No. 43.

the symptoms are much less definite. Chvostek's phenomenon has been found in other conditions, and its frequent occurrence in enteroptosis is of particular significance in reviewing the relation of gastric disturbances to the tetany reaction.

Schlesinger's observations of its occurrence in tuberculosis, chlorosis, neurasthenia, hysteria, and severe gastric disturbances are of interest, since Frankl-Hochwart agrees with him largely in his findings, and even goes further and states that he has found the phenomenon in a number of normal individuals. In these, however, it exists in his grades Nos. 2 and 3. He raises the suggestion, however, that possibly many of Schlesinger's patients really were mild or abortive cases of tetany. Its occurrence in myxœdema (Kraepelin), cretinism (Eiselsberg), and related trophic disturbances is recorded.

A sensory hyperexcitability has been described by Hoffmann and confirmed by many. It manifests itself in a manner analogous to that of the Chvostek mechanical excitability of motor nerves. Percussion over a sensory nerve trunk gives rise to painful sensations or to widespread paræsthesia in the entire distribution of the nerve percussed. Alone it has less diagnostic significance than Chvostek's sign, but in conjunction with the latter its importance should not be overlooked, especially in the diagnosis of simulators, aggravators, and hysterica.

**Mental Phenomena.**—Although it is considered characteristic that little or no involvement of the mental functions should be present, certain patients with well-developed tetany show mental symptoms. Frankl-Hochwart has called attention to this, and Kraepelin has observed it. Such mental symptoms have varied from the marked hyperexcitability of the chronic tetanoid patient to acute transitory confusion and delirium—quite in the sense of a toxic delirium—to a fairly advanced degree of deterioration in the sense of secondary dementia, as seen in myxœdema, epilepsy, and related types. The cases reported are still too few and imperfectly studied to permit any generalizations. It cannot yet be decided whether the tetany is to be regarded as primary or secondary in many of these cases, and hence the relationship of the psychosis to the tetany or to an accompanying condition is far from being clear. If the general viewpoint be maintained, that the tetany reaction is simply an indication of a more or less definite type of poisoning (parathyroid insufficiency, diminished calcium metabolism), then it is apparent that an underlying toxic feature is to be sought in explanation of the mental disturbance apart from the tetany.

**Special Senses.**—Modification of *sight* is a rare complication. Changes in the general eye apparatus seen as a whole are of not infrequent occurrence. Thus, ocular palsies, nystagmus, anomalies of light and accommodation reactions are recorded. Retinal changes are occasional, hyperæmia and neuritis having been seen. These are to be regarded as largely incidental in the development of the disorder, and not an intrinsic part. The trophic anomalies known to cause cataract are probably more fundamental; they are an impress of the general trophic disturbance which also sets up the modifications in the excitability of the neuromuscular apparatus, setting free the tetany spasms. *Hearing* and *taste* are rarely involved. Buzzing in the ears is to be regarded as a general widespread disorder, and has nothing to do with tetany itself.

**Sensory Phenomena.**—In addition to the purely motor and electrical phenomena, certain sensory changes are often present. These are more fleeting and less constant, as a rule. Pains during the contraction are usually present. They may be severe, or consist of a slight feeling of tension and drawing. Paræsthesia is irregularly distributed and extremely common. Such sensations Hoffmann claims may be induced by tapping on the sensory nerves or by electrically stimulating them. They are described as the crawling of ants on the skin, or the hands "fall asleep." Such paræsthesia is especially prevalent in the incomplete tetany cases. Anæsthesia is occasionally found. In such instances one should bear pseudo-tetany or hysterical tetany in mind.

**Temperature changes** have been noted for many years, but are infrequent, and probably the result of some infectious process, which is a possible primary cause for the metabolic disturbance in the individual case. Respiratory and circulatory changes are to be regarded from a similar standpoint. Involvement of the muscles of respiration naturally cause primary disturbances. The genito-urinary findings have been inconstant and are largely coincidental. Blood changes are of considerable interest from the experimental side. MacCallum and Voegtlin have shown a marked reduction in the calcium content of the blood in experimental tetany. Similar findings are reported by Parhon.

**Trophic Phenomena.**—These are very variable, and one suspects that some bear very little relation to the disorder. Certain of them are probably fundamental. Attempts have been made to explain all these changes on the general hypothesis of a vasomotor disturbance. The development of a cataract, which is not uncommon, is thus explained by Peters; but the general disturbance of parathyroid activity is more important. Falling out of the hair, loss of the finger-nails, irregular sweating, œdema, joint and tendon effusions, cyanois, redness, various eruptions of an erythematous, scarlatiniform, measly, herpetic, or urticarial nature, are all seen. Many of the skin eruptions occur in the gastric cases. Here the general disturbance in chloride metabolism may play a role. The skin reflex has nothing to do with the tetany, but is consequent on the same disturbance which may give rise to tetanic spasms. The trophic disturbances in tetany strumipriva (parathyroidectomy) are to be seen from the same standpoint.

**Tendon Reflexes.**—These are known to be modified in a certain number of patients, particularly in the direction of hyperexcitability. Ankle clonus has already been recorded.

**Course.**—Clinicians have recognized arbitrarily three groups of cases in adults, and most modern authors are inclined to follow Trousseau in his classical description. In the *benign* form the sensory phenomena, such as formication or a simple sensation of heat, may precede the spasms. These are confined for the most part to the hands or occasionally to the feet. The contractures may be fleeting, persisting for from five to fifteen minutes, or they may persist for an hour or more. Often the attack terminates by a recurrence of the sensory symptoms. A period of repose lasting for a quarter of an hour to two or three hours supervenes, and the spasms recur. In some instances two or three attacks a day may persist for several months. These mild attacks may recur at yearly intervals, or, as in some of Frankl-Hochwart's cases, several years may elapse, and then they will recur. These

benign cases are usually unattended with much pain. Consciousness is not disturbed, and there are usually no sensory, trophic, or temperature disturbances. Again, more *severe* attacks are observed. These occur more often in young adults, the benign forms having been mostly observed among children. Here the contractions are more violent and more painful. The preceding sensory phenomena are usually more pronounced, and with the increase on the severity of the condition other symptoms may be noted. Headache, malaise, and a rise in temperature of 1° to 3° may be noted. The affected muscles may show signs of congestion, and localized œdema of the hands and feet may be observed. Other muscles than those of the extremities may be involved. The muscles of the abdomen may be frequently contracted or the sternocleidomastoid and the pectorals; strabismus may be noted, either the external or the internal rectus, particularly the latter, being involved. Trousseau first noted the spasms of the pharynx, the larynx, the bladder, and the muscles of respiration, a severe dyspnœa supervening in the event of the latter muscles being affected.

These severe attacks are rarer than the benign ones. Frankl-Hochwart has shown that there is a distinct tendency for the well-marked lighter cases in many instances to become graver, and the good prognosis which most writers have given is now seriously doubted by this observer.

In the *grave form* there is no addition of symptoms. The attacks occur with greater and greater frequency and become more and more intense, and the patients die as a direct result.

**Diagnosis.**—The diagnosis of a classical case offers few difficulties. In English-speaking countries it is apt to be overlooked, although the more frequent reports of recent years point to the fact that it is being recognized more often, especially in its milder forms. The presence of cramps in the upper extremities, alone or in conjunction with the lower limbs, with the classical obstetrical hand and the additional evidence supplied by the Chvostek, Trousseau, and Erb signs, is usually sufficient to determine a diagnosis. Frankl-Hochwart would separate the different forms, saying that gastric tetany often offers particular difficulties, especially as a group of individuals exists in whom with gastro-intestinal disturbances there is a tendency to cramp-like contractures with the typical hand position. He groups them with the hysterias, rather than with the tetanies, however.

*Hysterical pseudo-tetanies* offer perhaps the most difficult diagnostic problems, especially as mixed and combined forms are undoubtedly known. It is not to be forgotten that some authors enlarge the hysterical group and limit the tetany group, and when it is considered how clumsily at times physicians conduct their examinations for the Chvostek, Trousseau, and Erb signs, especially overlooking the opportunities for suggestion in their examinations, it is perfectly understandable why such variances exist. Apart from tactless suggestive influences, however, Curschmann, as well as many another, has shown that the so-called cardinal symptoms are in reality not absolutely trustworthy. It is at times almost impossible to make a diagnosis from pseudo-tetany on a hysterical basis. The mental influence of accident insurance, litigation, simulation, aggravation, etc., should receive careful consideration. Especially is one liable to cast too much discredit upon the patient's reliability if these factors appear. The extensive literature quoted by Frankl-Hochwart should be consulted in this connection.

*Tetany strumipriva*, or, better, *parathyreopriva*, as suggested by Erdheim,[1] offers the most classical manifestations of the disorder, throwing, as well, considerable light upon some of the possible underlying and fundamental features of this peculiar reaction type. Insufficiency of the parathyroids results in convulsive phenomena of the tetany type. Pineles[2] has collected all the cases of tetany parathyreopriva, the study of which is one of the most important of the steps which led to the conception of tetany as a metabolic disorder, in which parathyroid activities are chiefly concerned. Since it is more clearly realized that the parathyroids are embedded in the thyroid tissue, and that the disorders of both may be present, the real difficulties in diagnosis consist in the separation of the symptoms due to disturbances in function of each gland.

**Prognosis.**—The point of view here maintained precludes the possibility of the statement of a general prognosis. Very little is known definitely of the prognosis in infants and children. Most authors agree in giving a fairly good prognosis, although Frankl-Hochwart says that healthy children rarely acquire convulsions, and that the prognosis is not good. In many of these children only one tetany-like spasm has been noted. In others the cramps may persist for weeks and even months. In simple cases the prognosis is much better than in those complicated especially with gastric or intestinal affections. Bronchitis, pneumonia, and occasionally an ascaris infection also determine a less favorable prognosis. Dangerous signs appear with glossal cramps, which may cause death. Recurrences are frequent in those who recover.

Tetany coming on during pregnancy and childbirth usually has a good prognosis. The hyperexcitability of the nervous system may persist for weeks after delivery. In succeeding pregnancies the recurrence of the phenomenon may be looked for. Frankl-Hochwart notes that in succeeding pregnancies the attacks are apt to be milder. It should be borne in mind, however, that sudden death may occur in these cases. The classical cases of Trousseau and Szukitz are cited.

In the cases apparently due to disturbances of the stomach surgical interference has brought about distinct amelioration. Sudden death may occur, and apart from surgical intervention the prognosis is admittedly bad (70 to 80 per cent.). The cases are comparatively rare, however. In severe cases associated with marked gastric dilatation, operation, if only exploratory, is advisable. The mortality after operation in some dozen or more cases now reported is as low as 30 per cent. The subsequent history of these patients remains to be reported.

Tetany following infectious diseases and acute or chronic poisoning seems to present a favorable prognosis, perhaps the best of the various forms.

The prognosis of tetany thyreopriva depends upon the amount of thyroid gland removed and whether the parathyroids are included. Total extirpation of the entire thyroid tissue and parathyroid is recognized to have a fatal outcome. The entire after-history of thyroid operations, so far as tetany and its prognosis as consequent on the operative procedures are

[1] *Mitt. a. d. Grenzgebiet. d. Med. u. Chir.*, 1906, vol. xvi.

[2] *Sitz. b. d. Akad. d. Wiss. in Wien.*, 1904, 113, Abt. 3, p. 190; and *Mitt. a. d. Grenzg. d. Med. u. Chir.*, 1904, xiv, 120. Also consult Pool, *Annals of Surgery*, 1907, xli, for a complete discussion.

concerned, must be entirely rewritten, since it has been in recent years only that the parathyroids have been reckoned with as an etiological factor. Tetany appears after total removal of the parathyroids, not only in man, but in lower animals. What the outcome of complete parathyroidectomy in man may be with the retention of thyroid tissue is not yet satisfactorily determined. In Pool's case, in which it is possible that the entire parathyroid was removed at the second operation, the patient recovered from the operation and was able to carry on her work for a time. What the ultimate history may be is undecided. We are as yet not in a position to decide as to the definite fate of these parathyroid removals or insufficiencies under calcium or surgical therapy.

The most important class numerically are the so-called epidemic tetanies. Frankl-Hochwart, in his earlier papers, gave a fairly good prognosis in this form, but in later contributions he presents further histories of these patients, and finds that the prognosis is far from being good. Many of them have died, directly or indirectly as a result of their tetany or its causative factor, in from four to eleven years after the onset. Intercurrent disease caused death in many, so that this author only holds that the tetany has perhaps reduced the resistance. Of his many cases, 264 in all, he was able to trace 55 some ten or more years after. Of these, 11 had died early; of the 44, only 9 were healthy; 37 came under personal investigation; 7 had chronic tetany, 19 suffered from tetanoid symptoms; in fact, two-thirds had persistent tetany reactions. This leads him to the conclusion that the prognosis without treatment is in reality very bad, which would be more in consonance with the prevailing point of view that regards the tetany reaction as a result of diminished parathyroid activity.

**Treatment.**—From the standpoint here outlined it may be readily deduced that a general treatment is not a rational mode of approach. The parathyroidectomized individual would not be benefited by a gastric operation. The essential feature in the general treatment is the search for the irritant, and this cannot always be successful.

In approaching any specific instance the first question is as to the validity of the phenomenon. This is placed first, because it should be the simplest factor to exclude. If no reason, remote or apparent, can be found which would account for simulation, aggravation, or suggestion, it may be assumed that the excitation of the nervous system has other than a purely psychogenic origin. In rare instances simulation has been a factor; thus, patients have simulated tetanoid contractures in order to obtain the pleasure of chloroform seminarcosis, and continue a habit once acquired. Hospital parasitism counts among its devotees a number who find the tetanoid spasm a source of interest and advantage; but these cases are not common.

With a positive diagnosis established, the organ involved should come into review. Inasmuch as parathyroid insufficiency is the most general cause, it is rational to treat those cases, many in children, the whole group of so-called idiopathic tetanies, many tetanies of pregnancy and of thyroid disease, by thyroid and parathyroid preparations. Parathyroid preparations seem to fulfil most of the conditions, yet occasionally the combined thyroid and parathyroid involvement renders the giving of the combined products of more service.

The use of foodstuffs rich in calcium and of calcium salts follows as a natural corollary from the studies enumerated. For the most part, it

would seem that such medication might entirely replace the use of the glandular substances themselves. In experimental tetanies the successful effect of the calcium salts has been very striking, and in tetanies in children calcium therapy has given almost uniformly good results. Such therapy apparently renders the older means unnecessary, such as curare, opium, hyoscyamus, the bromides, chloral, belladonna, chloroform, galvanism, sweat baths, etc. Up to the present time therapeutic experience is not sufficient to definitely prove the durability of calcium medication in the cases in which it seems needed.

The surgical expedient of transplanting parathyroid tissue has proven successful in animal work;[1] its successful application in persistent chronic tetanies in man is clearly foreshadowed by the experimental work on dogs. The technical difficulties do not seem insuperable in view of the ready transplantation of these structures to indifferent parts of the body.

Inasmuch as comparatively definite light has been thrown on the whole subject of the tetany reaction by the researches of MacCallum and Voegtlin in particular, the stomach tetanies, heretofore a particularly obstinate group, offer opportunity for a combined treatment, radical as well as conservative. Useless gastric operations need not be performed, yet at the same time the lines are laid down that point to the time when such operations may be of life-saving service. When obvious gastro-intestinal conditions point not only to such irritants in the cycle, it were folly to persist in a line of medication which, notwithstanding its theoretical possibilities, does not give results. Gastrostomy, gastrectomy, gastroduodenostomy, etc., according to the gastric lesion, may be demanded, since the gastric and intestinal mucosa seems to be an important factor in the possibility of calcium intake.

The best methods of administration of calcium salts are matters for individual experience. Foodstuffs rich in calcium, milk, eggs, whey, etc., are clearly useful forms. Injections of calcium chloride, which have been useful in animal work, may be adopted in man. Calcium hypophosphites or other salts to be taken by mouth are available, and intestinal enemata are indicated if other avenues of medication are contra-indicated. The exact dosage remains to be determined.

[1] Leischner, *Arch. f. klin. Chir.*, 1907, lxxxiv, 1, 208.

# CHAPTER XX.

## HYSTERIA.

By SMITH ELY JELLIFFE, M.D.

The synonyms for this affection are legion. The term hysteria was in common use in the time of Hippocrates; Pliny spoke of suffocatio mulierum, Willis and Sydenham of affectio hysterica, Lorry of melancholio nervea, Sauvage and nosologists of his time of hysterie, Van Helmholt of asthma uteri, Piorry of neuropallie, while Babinski's pithiatism is the latest term proposed.

**History, Etiology, and Psychopathology.**—No abnormal manifestation of human conduct is so well documented as that which is usually termed hysteria. From the very earliest times to the present, it has been recorded, in some of its phenomena, in all of the forms of human expression. It entered into legends and folk lore before historic records were known; it can be traced in the most ancient books of the East and West, in the poems and plays of the latter lyrical or dramatic writers, in the records of historians of events, chroniclers of action, and the makers of laws; can be seen preserved in marble in the works of the Greeks and Romans, in wood in the early Christian centuries, and in the forms of decorative art of the middle ages to the present. Priests, lawyers, philosophers, and physicians have occupied themselves with it in all ages and in all lands, and it still remains the great enigma of human personality. At no time in the past can it be said that any unanimity of opinion was reached concerning its essence; every age has known conflicting opinions, not only concerning its causes, but also its manifestations. Current notions of the times and customs of the different countries have each in their turn contributed to give it a varying presentation, but its underlying features have remained the same throughout.

With such a record and such a uniformity of expression, it hardly seems consistent to doubt its existence—yet doubters there have always been and always will be—because no one single formula has yet explained all of the phenomena. Nor is one likely to be found. Agnosticism cannot, however, lessen the interest in its records, nor dim the ardor of the oncoming student in attempting to reach a final generalization.

A complete historical analysis cannot be given here. In the summary of Laehr[1] and the special works of Nasse,[2] Friedreich,[3] Landouzy,[4] Dubois (of

[1] *Die Literatur der Psychiatrie, Neurologie und Psychologie,* XIV–XVIII Jahrhundert, four vols., 1904.

[2] *De insania commentatio secundum libros hippocraticos,* Bonn, 1830.

[3] *Vesuch eine Literärgeschichte der Pathologie und Therapie der psychischen Krankheiten,* 1830.

[4] *Traité complet de l'hystérie,* 1846.

Amiens),[1] Pommes,[2] Raulin,[3] Wier,[4] Brachet,[5] Briquet,[6] Richer,[7] Calmeil,[8] Trélat,[9] Legrand de Saulles,[10] Gilles de la Tourette,[11] Tornéry,[12] Pitres,[13] Bernheim,[14] Binswanger,[15] Steyerthal,[16] Voss,[17] Abricosoff,[18] Cesbron,[19] Janet,[20] and Savill,[21] the student interested in its history will find ample material; the more recent works of Voss, Steyerthal, Binswanger, and Cesbron are especially valuable. By reason of the many conflicts of opinion in the different ages a systematic sketch of the development of the theories is practically impossible, for many of the most ancient of these ideas still flourish in a modified form, while others are lost and forgotten, and will probably never come into service. For this reason the mode of presentation set forth by Brachet, and repeated by Cesbron, will be followed.

*Legendary Period.*—The sacred books of the East—particularly the Vedas (1800 B.C.)—contain passages relative to nervous diseases and particularly to disorders of menstruation. The delay of this function was considered a cause of nervousness, and was treated by thrashing the woman if the delay was more than nine days, the object being to chase the devil that was afflicting her. In the sixteenth century there were "complete manuals of thrashings" with endless details. Bernheim records the use of magical formulæ for the treatment of nervous disorders in Persia, in the seventh century, B.C.

In the works of Herodotus one finds that a hundred years previous to the time of Hippocrates there were specialists in nervous disorders, and the papyrus of Leyden gives full instructions how to dislodge a devil that has entered the stomach. Ancient Hebraic history—in the Talmud—while giving emphatic accounts of epilepsy, gives no very clear picture of hysteria. The later apostolic writings are replete with details of a variety of hysterical disorders, paraplegias, monoplegias, trance states, etc. Chinese legends and documents show its presence from its earliest times, and the discoveries of magnetism, of Perkins' tractors, and similar methods of treatment were antedated by the Chinese by many centuries. Hysterical epidemics were known from the earliest times in China, and in later ages a Chinese traveller,

[1] *Histoire philosophique de l'hypochondrie et de l'hystérie*, 1833.
[2] *Des affections vaporeuses du sexes*, 1752, sixth edition.
[3] *Traité des affections vaporeuses du sexes*, 1758.
[4] *Histoires, disputes et discours, etc.*, 1579, new edition, 1885.
[5] *Traité de l'hystérie*, 1847.
[6] *Traité clinique et thérapeutique de l'hystérie*, 1859.
[7] *Etudes cliniques sur l'hystéro-épilepsie, ou grande hystérie*, 1881.
[8] *De la folie-historique*, 1845.
[9] *Récherches historiques sur la folie*, 1839.
[10] *Les hystériques*, 1883.
[11] *Traité clinique et thérapeutique de l'hystérie*, 1895.
[12] *Les maladies nerveuses pendant l'antiquité gréco-romaine*, 1892. (Paris Thèse.)
[13] *Lecons cliniques sur l'hystérie et l'hypnotisme*, 1891.
[14] *Hypnotisme, Suggestion, Psychotherapie*, 1891. (See translation by C. A. Herter, New York; see also historical chapter in second edition, 1903.)
[15] *Die Hysterie*, 1904. The most complete work on hysteria up to the time of its appearance.
[16] *Was ist Hysterie?* 1909.
[17] *Klinische Beiträge zur lehre von der Hysterie*, 1909.
[18] *L'hystérie aux XVII et XVIII siecles, Études historique*, 1898. (Paris Thèse.)
[19] *Histoire critique de l'hystérie*, 1909. (Paris Thèse.)
[20] *Mental State of Hystericals, Major Symptoms of Hysteria*, 1908.
[21] *Lectures on Hysteria*, 1909.

seeing the famous *convulsionnaire* manifestations of St. Medard in Paris in 1740, compared them to the actions of a Chinese sect whose origin extends back into legendary history.

Homer, Herodotus, and Appolodorus all tell of Melampus, a physician, son of Amythaon, King of Pelos, who cured the three daughters of Proteus, King of Argos, of their ideas that they were cows. Melampus' fee, it is true, was one of the daughters and half the kingdom, but he evidently did not retire from practice, for there was an epidemic of hysterical frenzy among the women of Argos, and Melampus cured them also. Homer further tells of the temples erected to his name. To be inspired of Apollo was popular in those early Greek days, but among the many devotees, if one sees things as Strabo saw them, the Sibyllæ made up a special group of women given to major hysterical manifestations.

Æsculapius himself, on Galen's authority, was one of the earliest of psychotherapeutists. The whole cult of the Asclepiades, judging from the construction of their temples, their rites, and ceremonies, was proficient in the treatment of hysterical disorders, and the "temple sleep" of these earlier days was a highly colored psychotherapy, remnants of which still persist in ecclesiastical ceremonial and ritual. Then as now the hysterical formed but a small percentage of the afflicted seeking relief, but even in these legends the hysterical nature of many of the characters is clearly portrayed.

It is usually stated that Hippocrates was the first to suggest the uterine causation of hysteria which was given to him in a letter from Democritus, but Steyerthal denies that the father of medicine really teaches this doctrine, it being found only in one of the Hippocratic works of disputed authenticity. Cesbron notes that Pythagoras, Empedocles, and Democritus all taught the uterine etiology hypothesis before Hippocrates.

That blondes are more lymphatic, and, therefore, more humid and, hence, more readily disposed to hysteria, while brunettes are drier and less disposed, is also a presumably Hippocratic idea, which curiously enough has its modern advocates, many of whom are pleased with having discovered this so-called law. Hysterical mutism, hysterical hemiplegia (without involvement of the face), hysterical crises, facial, crural, and sciatica neuralgias, cardialgia, hysterical dyspnœa, hyperæsthesia, gastric attacks with nausea and vomiting, hysterical coughing, all are described or alluded to in the various Hippocratic works as given in the Littré edition.

Down through the medical historical records one can trace in almost unbroken succession the development and modifications of the germ idea of an unsatisfied uterine longing as bearing a genetic relationship to hysteria, which, in the modern expressions of Freud and his disciples receive its most refined and psychologically subtle rendering as the "psychic conversion of forgotten sexual trauma."

Celsus was content to reproduce the earlier doctrines, while Arætius has given a rich detail description. Apparently Arætius was the first to mention apparent death as a hysterical phenomenon. With Galen the scene shifted somewhat; uterine wanderings and unsatisfied sexual longings were partially abandoned, for he showed that the uterus could not wander, but on observing the cure of hysterical crises in a widow following a second marriage, and explaining it by his humoral doctrines, the new hypothesis of menstrual (leucorrhœal) retention came into vogue. Galen's discussion of the use

of the word hysteria should not be overlooked, since its application concerns much modern discussion of the unfitness of terms as symbols for disease processes. He sententiously remarks that if physicians would pay more attention to the things themselves, and less to idle discussion of the names, medicine would be benefited.

Abricossoff infers that Galen recognized hysteria in the male sex, although most historians have quoted Gilles de la Tourette in saying that Carolus Piso was the discoverer of male hysteria. Steyerthal also disputes Tourette's statement. For Galen, as for Hippocrates, hysteria was a proteus, and judging from the statistical importance given to it by early writers even to Sydenham's day, it may be readily assumed that much was included by these earlier writers in hysteria which to-day would be relegated to various psychoses on the one hand, and to organic disorders on the other.

During the Arabian period the Galenic doctrines held sway, but just when the doctrine of a uterine vapor or aura arose is not certain. Paulus Æginata speaks of an aura, but Fernels (1699) is more explicit in his description of a "vapor that commences in the uterus; that strikes the viscera and causes nausea," "that rises in the diaphragm and the thorax and renders the respiration short and frequent;" "when the vapor rises to the brain it causes a uterine furor." Many have assumed this to be the first complete expression of the theory of uterine vapors, and this general name "vapors" was very prevalent, expecially in French and English writings up to the time of Charcot. Ambroise Paré lent it the weight of his authority.

The uterine etiology died a lingering death, and had its advocates as late as the middle of the eighteenth century when the nervous system of the uterus became the chief criminal in the new pursuit. Cullen first enunciated this transition doctrine according to Cesbron.

*Humoral Doctrines.*—Attention has been called to the humoral doctrines of Galen, but a later humoralism may be touched upon, namely, that of Astruc, Cheyne, Lange, Brisseau, Blackmore, Pressavin, and many other eighteenth century writers. Naturally there were many with mixed humoral and uterine doctrines.

*Nerve Theories.*—To consider the nerves as a primary seat of hysteria was a natural outgrowth, and Boerhaave sought to explain the hysterical crises by a sort of intoxication of the abdominal nerves, due to a stagnation or an obstruction of the corrupting humors. Boerhaave was in reality one of the earliest suggestors of the splanchnic origin of hysteria. He did not give any better or worse definitions of these corrupting humors than many modern auto-intoxication advocates. He did not believe that the uterus was the site of the manufacture of these humors.

Although, for English-speaking students, the works of Sydenham are justly regarded as offering a remarkable picture of hysteria, yet he introduced ideas of retrogression, the influence of which is seen even at the present day. It has been noted that Galen may have believed in hysteria in the male, but, if so, such a belief was forgotten for many hundreds of years, and only about Piso's time did the presence of hysteria in the male obtrude itself upon the thoughts of clinicians. How to handle it, in view of the uterine humoral theories, was evidently difficult, and Sydenham cut the knot by saying that hypochondria was for the male what hysteria was for the female, thus claiming the two diseases as practically synonymous.

Sydenham may also be regarded as the forerunner of the doctrine that there was no such disease as hysteria; hypochondria was everything in this century.

*Cerebral Localization Hypothesis.*—Cesbron makes the rather startling statement that from the days of Hippocrates to the nineteenth century practically only four writers located hysteria as an affection of the brain. From the uterus, to uterine humors, to uterine nerves, to the nerves in general, and thus to the centre of the nervous system; these were the successive steps in the evolution of the ideas of etiology. An Arabic physician, Pratis, first called hysteria a "cold cephalalgia." Carolus Piso, in 1618, said that all the symptoms of hysteria came from the head and not from the uterus or intestines, and he also was one of the first to describe cutaneous anæsthesia, deafness, blindness, hysterical aphonia, and hysterical tremors. Willis, in his *Cerebral Pathology*, followed Piso.

*Modern Period.*—With the development of the Charcot school the hysteria hypotheses commenced to take on more definite shape, and in the past thirty years the views show a marked similarity even though couched in different terminologies. These modern hypotheses may be grouped about three general centres, the psychological, the physiological, and the biological, in all of which the psychogenic factor is prominent.

The chief *psychological* hypothesis, namely, that of dissociated personalities, received its first great impulse from Charcot himself, and has been most attractively elaborated and made popular by his pupil Janet, and even more intricately analyzed and extended by Freud and his school. For Charcot, hysteria was a psychosis, and Gilles de la Tourette is largely responsible for the prominence of the factor of suggestion in the aftercoming presentations; the extreme position of this feature alone having been advocated by Babinski, which author would seek to dismember hysteria further and give us a new grouping within this large medley, a task that Janet has also attempted, as well as Freud. Pithiatism, psychasthenia, and the anxiety neuroses are the new entities partly separated out of the hysteria conglomerate, partly from the neurasthenic mass, and partly from the initial stages of the more frank psychoses, notably dementia præcox.

Of the Charcot followers, Möbius suggested the line of many later definitions. He called those morbid phenomena hysterical which were induced by ideas, and the physical as well as the psychical reactions had a common psychogenic origin. For him every one was more or less hysterical. Every one has hysterical small coin in the bank of his personality. Gilles de la Tourette's large monograph, published in 1891, is the most faithful elucidation and amplification of the Charcot doctrines. It, with Briquet's classic, has served as the modern fount of symptomatology.

At the present time it is recognized that the Charcot teachings were too fixed; they regarded hysteria in the light of an indivisible entity, almost in the formal light of a "species" within the limits of which were dragged a vast cohort of symptoms. Charcot described as an entity—a morbus—where present-day psychiatry sees a cohors morborum, having really little in common, save a tendency to similar emotional reactions. The stigmata of Charcot are not alone the appanage of a definite disease, they are very widely distributed; they are not the *stigmata diaboli* but the *stigmata necessitatis* (Steyerthal).

Probably no studies of hysteria in modern times have attracted so much attention as those of Pierre Janet, who has been so very prolific that it

becomes almost impossible to give a short résumé of his standpoint. His own abstract, given at the Amsterdam Congress in 1907, is perhaps the best available. The study of somnambulism is his starting point. For him there exists in consciousness a region below, if such a term be permissible, the normal waking or personal consciousness, which is called the subconscious. Groups of ideas may exist in this, so to speak, twilight region without being at all clearly perceived by an individual, in fact, without being known at all, and yet they may operate to produce results very much as if they were the subject of voluntary attention.

The hysteric in an access of delirium lives through fancied experiences about which he knows nothing when he "comes to"—he has an amnesia for all of these events. The hysterical amnesia does not confine its manifestations to such conditions, but invades the details of life. The person who is sent on an errand forgets what she is sent for before she gets half way to her destination. This is a simple but common example. Janet would explain this by a disorder of attention. The directions are imparted to the patient but are not acutely attended to, and drop at once into the region of the subconscious and are forgotten by the waking, personal consciousness. The anæsthetic arm is so because the patient does not attend sufficiently to the sensations from the arm to perceive them. There is a narrowing of the field of personal consciousness, which is but another way of expressing the defect in attention.

The synthesis of mental processes into a coherent whole constitutes the personality or ego, and the hysterical process causes a splitting, a disintegration, or a doubling of the personality. Janet's definition is that "hysteria is a form of mental depression, characterized by the retraction of the field of personal consciousness and by the tendency to the dissociation and the emancipation of systems of ideas and of functions which by their synthesis constitute the personality." For Janet the hysterical and the hypnotic states are identical, based upon the common factor of suggestibility.

The theories of Sidis, of Breur and Freud, and those of the Freud school are modified dissociation theories in which one finds more stress laid upon etiological factors. This is practically true of Freud's ideas, which are of paramount interest to the student of mental problems since this author's work with that of Vogt's has proved a great stimulus leading to the interpretation of mental mechanisms.

Freud's hypothesis presupposes three features: (*a*) The role of psychogenesis, (*b*) Janet's ideas upon dissociation and psychical automatism, and (*c*) Binswanger's formulation of the etiological importance of the affect. As early as 1880 Breur advanced the interpretation that the individual roots of the hysterical symptoms were to be sought in ideational complexes with marked feeling tone which came about as a result of psychical or physical trauma, and in 1893 to 1895 Breur and Freud further formulated the idea that the psycho-neurotic symptoms originated from these complexes either by a process of (*a*) conversion, whereby the emotional (affect) excitement brought about abnormal physical innervation—this caused hysteria; or (*b*) by transposition of the affect through indifferent ideas (anxiety neuroses). The principle of overdetermination expressed the heaping action of an affect sufficient to determine a symptom. This same overdetermination is more concretely and physiologically expressed by Cajal's concept of avalanche action.

The ground of the conversion or the transposition lies in the immiscibility of the traumatic complex with the personality. The patient refuses to accept it, and instead of ab-reacting—and thus normally disposing of the complex—converts it or transposes it. The affect thus remains shut in or hidden in the subconscious.

Gradually Freud's attention became centred, and perhaps somewhat one-sidedly, upon the sexual nature of the original trauma. His general hypothesis is extremely intricate, and no short abstract does justice to the results obtained by his psycho-analysis, yet his present attitude may be expressed somewhat as follows: There develops, usually on a constitutional basis, in the period before puberty definite sexual activities which are mostly of a perverse nature. These activities do not, as a rule, lead to a definite neurosis up to the time of puberty, which in the psychic sphere appears much earlier than in the body, but sexual fantasy maintains a perverse constellated direction by reason of the infantile sexual activities. On constitutional (affect) grounds the increased fantasy of the hysteric leads to the formation of complexes which are not taken up by the personality and by reason of shame or disgust remain buried. There therefore results a conflict between the characteristic normal libido and the sexual repressions of these buried infantile perversions. These conflicts give rise to the hysterical symptoms. It is in his contributions to the sexual theory that Freud develops his later thoughts of the sexual origin of the hysterical reaction. By sexual it is important to remember Freud is not speaking of sensual.

Inasmuch as these sexual traumata are forgotten, buried in the subconscious, it becomes necessary to dig them out by the process of psycho-analysis, either using Freud's method, or by the association tests so minutely and painstakingly elaborated by Jung and Riklin particularly. In practice it may take months or years to fully analyze some hysterical cases. When fully analyzed the patients become cured—the analysis has been a catharsis.

White[1] has expressed the whole matter very clearly. He writes: "The characteristic of the psychic traumata that produce hysteria is their large content of painful affect. A painful affect, fully reacted to at the time, may produce no harm, but if for any reason reaction fails, the feelings become repressed and the possibilities of dissociation are created. Failure of reaction may be due to the failure of conditions that make efficient reaction impossible, as for instance, an insult "is swallowed," a dear friend or parent is lost, and no compensation is possible. This gives rise to "retention-hysteria." Again, ideas usually of a sexual nature, which are incompatible with the personal consciousness, are repressed—ab-reaction is not permitted, no effectual catharsis takes place. This condition produces the "defence" hysteria. Finally, experiences occur in a hypnoid state—*i. e.*, in a split-off, dissociated, or dreamy state. They produce the so-called "hypnoid-hysteria."

"The final principle of the Breur-Freud hypothesis is the principle of conversion. The strangulated affect, the unreacted-to emotion, belonging to the dissociated state which has been repressed, finds its way into bodily innervation, thus producing the motor phenomena of hysteria. In this

[1] "Current Conceptions of Hysteria," *Interstate Medical Journal*, January, 1910.

way the strong idea is weakened by being robbed of its affect—the real object of conversion."

"The significant feature of Freud's theory is the tracing of every case to sexual traumata during early childhood. Sexual experiences differ, however, from ordinary experiences—the latter have a tendency to fade out, while the idea of the former grows with increasing sexual maturity. There results a disproportionate capacity for increased reaction which takes place in the subconscious. This is the cause of the mischief.

"There must be, however, a connecting link between the infantile sexual traumata and the later manifestations. This connection Freud finds in the so-called "hysterical fancies." These are the day dreams of erotic coloring, wish-gratifications, originating in privation and longing. These fancies hark back to the original traumatic moment, and, either originating in the subconscious or shortly becoming subconscious, are transformed into hysterical symptoms. They constitute a defence of the ego against the revival, as reminiscences, of the repressed traumatic experiences of childhood."

It is premature to pass judgment on Freud's ideas. They have their warm advocates and bitter opponents—they suggest the psychical archeologist grubbing about in the fragments of old, crumbling, and mutilated memories with perhaps a tendency to romantic reconstruction. The method of the archeologist who reconstructs the entire animal from a single tooth is not called in question, but it would seem not improbable that psychoanalysis, so-called, might find the same type of "sexual fragments" in non-hysterical individuals, but Freud's "strangulated affects," "conversions," and "ab-reactions" have not yet been sufficiently subjected to critical tests to determine their real value.[1]

It had already been intimated that the superstructure built on the Charcot foundations had become a little top heavy, and the time had arrived for a more searching critique. There have been hosts of destructive critics, and the dismemberment of hysteria has been going on *pari passu* with its growth. Of the more recent serious studies looking toward a limitation of the concept and a sharpening of the diagnostic boundaries, the monograph of Steyerthal and the articles by Babinski are the most noteworthy. Steyerthal's position is extreme. For him there is no such thing as hysteria. The vast majority of the cases may be reduced to neurasthenia and mental inferiority. The stigmata of Charcot, Janet, and others are nothing but signs of tire or exhaustion. Steyerthal's exposition is ingenious and scholarly, and he has detected a number of historical errors which have been repeated for many years, but his general standpoint is too narrow. Babinski's chief position is equally restricted, but in another direction. His dismemberment of hysteria is the most complete contribution that he has given to date.[2] He has seen the hysterical problem grow, and with many another, he has sought to carve away from the mass the trivial and unessential and present some definite entity. This new grouping of old facts under a new

[1] Freud's ideas are available in Brill's very excellent translation, "Selected Papers on Hysteria and Other Psychoneuroses," *Nervous and Mental Disease Monograph Series*, No. 4, New York, 1909. The method of Psycho-analysis by Association Tests is given best in the same writer's translation of Jung's "Psychology of Dementia Præcox," same series, No. 3, 1909.

[2] *La Semaine Médicale*, January 6, 1909, No. 1, p. 1.

head he has christened pithiatisme; it consists of those hysterias of tradition which respond to his formula, namely, those patients whose ills are brought into existence by the power of suggestion, and the cure of which takes place by persuasion. And suggestion, for Babinski, should express the action by which one endeavors to make another accept or realize an idea which is manifestly unreasonable, while persuasion ought to be applied to ideas that are reasonable, or which, at least, are not in opposition to good sense. All that class of phenomena which falls within the limits of this concept are truly pithiatique, or, using the old term which Babinski would avoid, are hysterical.

Babinski has attacked the unconscious and great extension of the idea of hysteria. The reasons for this are numerous, but may be conveniently assembled in three groups as follows: They are due to (1) errors in diagnosis; (2) to defective observation and the including in the hysterical camp of a number of simulated symptoms which do not belong there, and finally, (3) the confusion of nervous states which should be considered separately—neurasthenia, hypochondria, etc.

While all of the errors in diagnosis cannot be reviewed, Babinski calls particular attention to hemiplegia and hemianæsthesia. The plantar reflex of Babinski, the combined movements of trunk and thighs, the Hoover thigh phenomenon, the sign of Grasset, the hypertonicity of paralyzed muscles, the phenomena of pronation, have come to aid in diagnosis. As for hemianæsthesia, if the examination be sufficiently artful, it disappears entirely. If the more careful methods of Babinski, Stern, and Ziehen are employed it would appear that Babinski's contention is fairly true. Babinski further has pointed out that in the mistakes have been included hæmoptysis, hæmatemesis, hæmaturia, and fever.

The phenomena of his second group, simulation and deceit, are difficult of classification. These, again, may be subdivided. One sub-group consists of the imitators of true pithiatic signs, those who simulate true hysteria—these show the various accidents such as paralyses, contractures, anæsthesia, etc. This sub-group is one of the most difficult to understand, and it cannot be said that Babinski has thrown any real light on it. These have been dealt with under the heading of aggravation and exaggeration by the followers of the more classical schemes. Inasmuch as persuasion does not seem to cure them, they are, ergo, not pithiatiques, but simulators. Surely a very weak position. A second sub-group comprise those patients whose phenomena are truly fictitious. They cannot be induced even by suggestion. Fever and anuria are examples.

A third sub-group comprises real phenomena but consciously brought about for purposes of deception. These are the erythemata, phlyctenulæ, ecchymoses, ulcerations, sphacelæ, œdemas, etc. This group Dupré has already termed "mythomanies."

Finally, in discussing the third category, Babinski narrows down his conception to those cases which can be influenced by suggestion, and in a significant line he asks of the symptomatic aspect of the phenomena which suggestion can give rise to: Is it specific; does it belong to troubles related possibly to an organic affection, or to a functional malady having a mechanism different from suggestion? Here Babinski begs the whole question. For if we have a suggested chorea so like the real thing as to be inseparable by present methods, it hardly seems that his criteria help us much.

Babinski in his third group separated the true pithiatiques from the "emotives"—in whom moral shock can cause phenomena to appear or disappear. He contends that in these cases an imitative factor is greater than the emotional one; finally, he erects a third sub-group in which exaggerated tendon reflexes, vasomotor phenomena, dermographia, etc., are present and usually allied with hysteria.

True hysteria then falls into three large sub-groups—the "pithiatiques," "the emotifs," and the "reflex," and these constitute a well-limited neuropathic state distinguished from all other neuroses. One can deduce that the hysterical or pithiatique phenomena depend essentially in their appearance, duration, form or disappearance, on the psychic milieu of suggestible subjects whose predisposition is susceptible of being put in play by such and such a spectacle, or such and such a proposal.

Babinski's practical consequences, which he has briefly outlined, and which belong really in the chapter on therapeutics, are worthy of reproduction. He concludes that: (1) A physician should guard himself from maladroit suggestions and poor methods of examination. (2) Knowing the influence that surroundings play with these patients, these should be so modified as to get the desired effect. (3) One must avoid contrasuggestive treatment. (4) One should limit one's self in diagnosis and not make too many promises, especially in the combined cases, *i. e.*, where there are hysterical signs due in reality to organic disease. (5) From a medicolegal standpoint it is important to differentiate hysteria from simulation in order not to deprive those really suffering from just recompense on the one hand, and secondly, to limit the abuse of accident litigation in fraudulent claims. Babinski holds that his formula is useful in separating the conditions.

*Physiological Theories.*—The modern physiological hypotheses of chief interest are those of Sollier, Binswanger, and the English school. They hardly do more than restate the fundamental psychological hypotheses in physiological terms. Sollier defines hysteria as a psychical functional disturbance of the brain, consisting of a torpor or a sleep of the cerebral centres, localized or generalized, temporary or permanent, and manifesting itself consequently according to the centres affected by vasomotor, trophic, visceral, sensory, motor, and final psychic disturbances, and according to its variations, its degree, and duration, by transitory crises, permanent stigmata, or by paroxysmal accidents. Confirmed hysterics are only vigilambulists whose state of sleep is more or less profound, more or less extensive. This is a dissociation hypothesis pure and simple, and inasmuch as little is known of sleep physiologically, one makes no advance by defining hysteria in terms of sleep.

Binswanger may be reckoned among the adherents of a physiological hypothesis. For him, although he claims it to be true that all hysterical phenomena can be influenced by psychical processes, yet there is no proof that hysteria originates without exception by means of psychical processes exclusively. The disturbances of sensation and of motion which are reckoned among the most characteristic of the stigmata of hysteria are not necessarily of psychical origin. The same holds true for many of the visceral disturbances. The trend of recent study tends to confirm Binswanger's view.

The hysterical change consists in the disturbance of the orderly correlation between the psychical and material processes, and in a double direction.

On the one hand for a definite series of material cortical stimuli, the parallel psychical processes are either lacking or only incompletely awakened, while, on the other hand, an excess of psychical accomplishment results in response to minimal cortical stimulation, producing a variety of back actions on the entire innervation processes which originate in the cortex, or are ruled by it. The pathological superabundance of psychical work consists not only in an increase of emotional reactions, in a sharpening of the sensations, or in the propping up of overvalued ideas, but also in the very striking facility by which the psychical processes, above all, ideas, can influence function to produce hysterical reactions.

It must be considered that in individual functional areas of the cortex, entirely different variations from the normal conditions of stimulation, both quantitatively and qualitatively, exist. At times over-stimulation, at times under-stimulation come into play, or dissimilar activities may show side by side. Furthermore, it is to be borne in mind that sharply localized disturbances of the cortex secondarily involve the capacity of other functional cortical areas, either enhancing or depressing their activities. Such functional dependencies show themselves most markedly in the disturbance of the corticomotor regions, where, for example, a "falling out" of the functional capacity of the path from cortex to bulbar or spinal motor centres causes paresis or paralysis, the incoming stimuli, influenced by anæsthesia or analgesia, not being sufficiently active to cause the requisite nerve discharge leading to motor activity. This disturbance of dynamic proportion is not confined to the cortex alone, but may extend to the entire nervous system, showing itself particularly in correlated physiological functional groups.

Binswanger further holds that similar dynamic disturbances are found in epilepsy, hypochondria, neurasthenia, etc., but that the distribution of the disturbed mechanism varies, and, although the entire group may be thrown into one, the psychoneuroses, yet each has its distinguishing features notwithstanding much overlapping, combination, and mixed forms.

Such a physiological statement does not go far enough, however; practically the same defects may be postulated for the psychoses in general, certainly for dementia præcox at least. According to Binswanger, however, the hysterical changes are characterized by the abnormal activity of psychical influence on the innervation processes. Proportionately slight, of themselves almost negligible, cortical stimuli set up intense sensations, with pathologically increased feeling tone (pain) and conversely corticoperipheral stimuli processes, in spite of moderately strong peripheral stimuli, do not give rise to the parallel psychical processes. The intercortical or association processes show analogous disturbances. The importance of the pathological affect action, simply stated by Binswanger, has received much extended analysis in the work of Breur and Freud, of Bleuler, Jung, Ricklin, and others; and Binswanger adopts Freud's general method of interpretation, especially with reference to the mechanism of conversion, laying less stress upon sexual traumata as the exclusive agents in the production of the reaction.

*Vasomotor Hypothesis.*—Such a view has been adopted by numbers of English neurologists, the most recent expression of which is that of Savill. For this author hysterical syncope is one of the cardinal symptoms in the disease. It represents a hysterical cerebral attack, and is due to the activity of a special reflex vasomotor centre situated in the solar plexus of the abdomi-

nal sympathetic. This is usually started into activity by an emotional stimulus from the brain, but may be started to activity and produce exactly the same kind of attack by pressure upon the groin, the stimulus passing through the iliohypogastric nerve, or by other peripheral stimulus from other areas in the skin or mucous membrane. Hysterical cerebral attacks may also be induced by variations in the vascular condition of the splanchnic area.

It is a further feature of Savill's hypothesis that the vascular conditions within the abdomen, skin, and brain oscillate among themselves, and he further drags in a modern humoralism by assuming that the symptoms are conditioned by the state of the purity of the blood. Hysterical motor disorders, paralyses, etc., are due to localized or general vascular changes in the brain; they differ from organic lesions only in extent, duration, and intensity, and in the fact that they are primarily conditioned through the sympathetic. Hysterical anæsthesia is due to the same types of vascular lesion. Other sensory symptoms, hyperalgesia, etc., are due to peripheral vascular lesions, and psychogenesis may play a leading role.

As to the psychogenic element, Savill admits that the mind plays a prominent part in many hysterical phenomena, but hysteria is a psychosis only in the following respects:

1. Hysterical persons throughout life present certain inherent peculiarities of mind—*e. g.*, a tendency to mental abstraction, to autohypnotism, to mental dissociation, and to dual consciousness—which render them more liable, especially on the occurrence of any disturbance of the cerebral circulation or nutrition, to exhibit abnormal mental phenomena.

2. A certain proportion of hysterical symptoms are purely mental; the mental faculties are unstable and easily disturbed, particularly the various commemorative faculties; various tricks and habits of body and mind are constantly arising.

3. The emotional side of the mind is strongly developed, and emotional outbursts which we call "hysterics" are frequent. Emotion is also a frequent determining cause of other hysterical disorders by producing vascular changes. By the well-known influence of the emotions on the vasomotor centres, vasomotor phenomena are common; and since the vasomotor centres are themselves unstable, many surprising effects and symptoms are produced, apparently as the direct but really as the indirect effect of emotion.

4. The mind plays an important part in *exaggerating* symptoms which have a slight physical basis, such as a vascular derangement of a part.

5. The mind also plays an important part in the *perpetuation* of symptoms after the physical basis which had initiated them has passed away.

Thus, Savill comes to a poly-etiological conclusion. No single lesion or hypothesis is capable of explaining all the symptoms. The vasomotor system, however, plays the most important role, and Savill states that fully 90 per cent. of all of the symptoms are conditioned by instability of the vasomotor centres throughout the body and a want of coördination among these, associated with more or less emotional instability.

Although much is said of emotional instability, Savill makes no attempt to clear up the conception of the role of the emotions in this disease—a conception which practically all agree is of primary importance. If we are to succeed in analyzing hysteria we must approach the subject from the side

of emotional reactivity. This is best considered here, as its final analysis will probably come from the physiological and pathological data furnished by the study of certain cellular alterations found in those psychoses with prominent disorder in the affective sphere.

A logical mode of approach is hardly possible, but in considering some of the well-known psychical reactions which accompany organic visceral disease, a breach may be made in the walls of the problem. For some it is true such phenomena are not hysterical, but if the ground is so shifted, the significance of the word is lost. These types of phenomena furnish a clue to the nature of similar phenomena which it is desirable to understand.

Head has clearly presented certain mental changes which accompany visceral disease; the more patent of these may be dismissed from present consideration. In considering a special type of reflex pains of visceral disease and the influence that they consciously or unconsciously play in conduct, one approaches very closely, if not enters, the hysteria confines. Many of the phenomena called hysterical are almost constant accompaniments of organic disease of the viscera. Hallucinations of sight, of hearing, of smell, haptic hallucinations, rarely as completely developed as seen in the major psychoses, are extremely frequent. Many of these partake more of the nature of illusions, but the difference is at times an artificial one, in which connection Möbius' term of pain illusion as descriptive of the psychogenic pains of hysteria is to be considered. Moodiness is constant and leads to sleeplessness. Restlessness, attacks of crying, ideas of impending ill, feelings of exaltation, impulses to suspicion, though rarely approaching the grades usually termed delusions of reference or of observation, are extremely frequent. In all of these cases there is a type of pain which is produced by impulses passing from the affected organ up the fibers of the sympathetic, through the ganglion of the posterior root, into the central nervous system. From this result the reflex pains of the peripheral distribution of continuous segments so well described by Head. The intensity and duration of these visceral reflected pains are factors of considerable importance in the production of mental changes. Slight and evanescent pains rarely have any reaction, but the continuous, oftentimes only slight pains, are more likely to lead to the psychical effects. Similar results result from continuous though perhaps mild affect conditions, as will be seen later. Women are more prone to these reflex visceral pains, and menstruation increases the severity of the mental phenomena.

Individuals thus afflicted often acquire a depressed moodiness which is, to use Head's phrase, non-projected, by which he seeks, and rather inadequately, to distinguish the state from an *emotional* state by calling the latter projected. Consciousness is dominated by a feeling tone, but the reasoning state is not projected. The opposite side of this moodiness is an exaggerated sense of well-being. The moodiness of organic disorders may be associated, or even conditioned, by the ill-defined feelings of suspicion already mentioned. Such feelings are usually under the influence of persuasion, in which respect, again, their relation to like hysterical states is evident.

A further consideration concerns changes in attention and memory. Their analysis is extremely difficult by reason of the complexity of the phenomena. Head has reported classical examples in mild and advanced tuberculosis and the literature of organic disorders allied with hysterical reactions is replete with similar cases; some of Binswanger's unusually

ample histories in his great monograph on hysteria are instances in point. Notwithstanding the difficulty of analysis, several points stand out. Fatigue is certainly one, and the presence of the feeling due to the visceral sensations is another. Thus, attention is divided, and memory is less complete.

The individual capacities for reproducing—*i. e.*, by associated processes to bring up in consciousness memory images of feeling tone—is extremely variable, and here visuals, auditives, olfactives, affectives, etc., follow their own laws of association.

While these considerations may, at first sight, seem to have but indirect bearing upon the subject of hysteria proper, it is very evident that they do bear directly upon the mechanism by which many manifestations, usually termed hysterical, are brought into existence, and furthermore they are of importance in any attempt to sift out those cases which depend upon some definite organic factor, though extremely variable as to its extension and its intensity, seen either from the standpoint of a direct visceral irritant factor in producing mental states of altered adaptation, or as causative in reducing mental resistance. The significance of Babinski's insistent critique of hysterical phenomena is amply justified by these considerations.

The next step is to ascertain how far such similar mechanisms may be set in play by lesser and lesser amounts of organic change and greater and greater amounts of mental presentation of the same class of phenomena. For it is practically coming to be conceded that the term hysteria should be restricted to the more purely psychogenetically induced disturbances of these mechanisms just considered. Not that these cover the entire series of phenomena ranged as hysterical, but they are those least capable of being understood as possibly arising as purely psychical reactions. No attempt is made to present the theories of the emotions, but it is accepted that inadequacies to emotional reactions are primary factors in the phenomena of hysteria.

Stated in very bald and primitive form, when one is brought face to face with an experience—say the oncoming of an excited animal—one's primary sensations of recognition and feeling tone of fear cause the adaptive motor mechanism of self-protection. In the healthy animal it may be to flight, but an inadequate reaction may result in inability to do anything, *i. e.*, in a paralysis, the individual components of which cannot be here analyzed. One may reason that so long as the reaction remains inadequate the individual suffers from an emotion, fear, and the recognition of the inadequacy, *i. e.*, the paralysis, constitutes the emotion. Thus the emotion considered biologically is an evidence of maladaptation.

Head's work seems to point in the direction of the *localization* of the affected mechanisms, *i. e.*, the sympathetic, but turning to Sherrington's recent discussion of the proprio-receptive system one finds indications that the question is a much wider one and progress is being made that is eminently suggestive, at least, in its bearings on the hysteria problem.

*Biological Hypotheses.*—One terms these biological largely for the sake of convenience. They regard the hysterical reaction in the light of an adjustment to experience, or interpret it in biological terms, or perhaps in the terms of comparative psychology. The hypotheses mentioned do not conform to hard and fast classifications; thus Freud's latest interpretations could as well be classed here as under the head of psychological hypotheses. Here one can conveniently place the views of Dubois, and more particularly

his pupil Schnyder, Hellpach, and Claparéde, not to mention a host of others. Schnyder regards hysteria as the persistence in adult life of the childish type of reaction to the facts of life. It is a mode of reaction in persons of naïve, simple, and elementary mentality. It is a mentality lacking in development and defective in judgment and critique. Such mentalities, when placed in new environments to which they cannot adapt, or adapt with difficulty, develop the hysterical reaction. They, so to speak, go back to primitive methods of escaping difficulties. Hysterical paralysis, or anæsthesia of an arm or a leg, is the analogue of an animal "playing possum," the more one pokes them the quieter they lie. While, on the other hand, hysterical diarrhœas represents an inadequate—*i. e.*, emotional reaction to painful ideation.

Schnyder also speaks of a racial hysteria as a manifestation of the infancy of a nation, of people who are primitive and simple. It is as commonplace to note that conduct which for a white man would be called hysterical is normal for the colored race, and Kraepelin's observation of the great amount of hysteria among the natives of Java is in line with this general argument.

Modern methods of expression, the press, strikes, boycotts, unions, etc., are the safety valves of much that otherwise would be retained and repressed, and in Freud's terms "be converted" into hysterical phenomena.

Claparéde lays special stress upon the fact that the hysteric shows a marked resistance to the recall of painful memories. This, viewed biologically, he considers to be a defense reaction. Suggestibility is also a protective adaptation against excessive development of personal peculiarities and tendencies, which, should they be given free play, would isolate the individual from the social world in which he lives. Instead of yielding to these impulses the suggestion of another is followed because adaptive. Suggestion thus becomes a biologically rational process—not as Babinski would have it, a response to irrational thoughts. Claparéde's ideas are interesting in view of Bleuler's very clear-cut presentation of suggestibility in the light of an adaptive mechanism with negativism as its physiological corrective, producing severally in its excess hypersuggestibility and hypernegativism in both the hysterical and dementia præcox reactions.

An attempt at a biological definition may be hazarded. Thus understood, hysteria is the carrying over into adult life of a primitive adaptive type of nervous and mental reaction to psychical influences due to congenital anomaly, acquired development, or diminished resistance.

A few words may be said concerning the *anatomical* and *pathological* data. Here, although a definite correlation has not yet been effected, there are indications which show that the factors of congenital anomaly and diminished resistance may have some demonstrable anatomical substratum. The analysis of cortical function and cellular structure is yielding results. Flechsig's data have been partly verified, corrected and amplified by the systematic study of structural variations in the cortex by the cytological methods pursued by Campbell, Vogt, and Brodmann. Criteria are being established for the determination of the physiological value of cell layers and cell groups.

The layers of cells functioning for the reception of different types of stimuli are becoming fairly well delimited, but perhaps more important than all of these, pathological studies of the psychoses, dementia præcox particularly, show us, though as yet but faintly, that certain definite cell groups are

affected. Here there is a most profound disturbance of the affective states with relative integrity of sensation, motion, memory, and association processes. The work of Cajal on the functional physiology of different neurones is giving more point to the ideas of dynamic polarization and some tangible anatomical details are coming to hand which give warrant for the terms "condensors" and "accumulators" of nervous energy. There is some hope of explaining the mechanism of increased discharge, lengthened discharge, and avalanche action on anatomical grounds, and problems which have always been considered as purely functional and unanalyzable by microchemical methods seem to offer some hope of solution by these more tangible and objective procedures.

**Symptoms.**—Before attempting a systematic presentation of the symptoms of the hysterical reaction a few remarks may be made regarding the value of this motley collection of observations in which real fragments are mingled with those born of credulity and mental laziness. The golden period of hysteria which reached such a high point as a result of the stimulus of the Charcot teachings has been followed by one of analysis in which the careful sifting of the enormous material has become an imperative necessity. One of the first striking facts of this reëxamination is the astonishing frequency of mistakes in diagnosis. It was possible for Landouzy in 1846 to make a collection of all the published observations of hysteria—such a task is now impossible, and would be fruitless, for it has only been within recent years that enough finer methods of examination have been at hand to decide some of the vexed diagnostic points.

Thus, the tubercle bacillus has shown the real character of many heretofore diagnosed hysterical hæmoptyses; gastric chemistry has similarly relegated many intestinal disorders into their proper position; microscopic and cryoscopic methods enable one to determine the essential features of supposed hysterical hæmaturias; methods of blood examination have reduced the number of hysterical fevers almost to a minimum.

In the field of the paralyses, hemiplegias, monoplegias, paraplegias, etc., mistakes have been especially frequent. With the newer signs of organic involvement of the nervous system many organic disorders are recognized which, heretofore, were called hysterical, and are even called so at the present time in their early stages. Literally thousands of sick individuals suffer from complaints which a lazy diagnosis dubs hysteria. The falsity of their position would be revealed by a searching and intelligent analysis.

Another feature concerning the revaluation of symptoms and one of the most difficult to adequately present, is simulation. Unconscious simulation, if there be such, and aggravation are not included here. These are features of what must be termed the hysterical mentality. Simulation as here restricted is probably of rare occurrence, yet it is constant and serves to discredit and discourage careful study of many deserving patients.

What symptoms are due to deception and deceit in the non-hysterical and in the hysterical? It is highly probable that many of those who practise such deceptions, seen from the psychiatric view point, are either debiles or mild precocious dements. As for deliberate frauds, their symptomatology may be disregarded. There still remain a large number of symptoms which are held to be characteristic of hysteria, and which, however, are variously estimated by different observers. Here one is on the most difficult ground of

the hysteria problem and each individual observer looks at the symptoms from entirely different points of view. Each reflects certain aspects of subjective truth rendering a combination impossible, hence the hopelessness of a definition. Thus no philosophic unity has as yet been offered which gives a standard scale of measurement of these various symptoms.

The imperfect scales have been many. Thus, restricting the discussion purely to modern symbols, the striking motor phenomena of the "crises des nerfs," the major attacks, are the only real phenomena which should be considered as hysterical, according to the present view of Bernheim. All others are epiphenomena and are the results of education. This point of view would considerably simplify the symptomatology.

A subdivision of symptoms into two groups as stigmata and as accidents has long been current, but it is certain that in the word stigmata is found a relic of the methods of the middle ages in their search for trace of the presence of the Devil in accused individuals. These marks of Satan are handed down still as the most important of the symptoms of hysteria. Here, again, one finds discord and strife. Whereas theologians, magistrates, medical men were in practical accord in the year 1600, at the present time there are hardly two neurologists agreed as to what shall be considered as stigmata or essential features of the hysterical phenomena. One knows that for Janet, anæsthesiæ, amnesiæ, abulias, paralyses and changes of character represent the stigmata, while under the head of accidents, he groups hysterical attacks, somnambulism, subconscious acts, and fixed ideas, whereas Babinski claims that careful examination shows that anæsthesia never exists, and between these extremes one finds all possible variations. Some authors, Charcot, for example, lay great stress on restriction of the visual fields, others, as Oppenheim, practically deny its existence.

Thus for almost every symptom these conflicting attitudes of mind are found. A few uphold the absolute lack of existence of many phenomena, while others are in supreme discord as to the interpretation of phenomena which to them are subjectively true. It becomes, therefore, a difficult matter to know just how to present the symptoms. We have chosen the purely clinical method, grouping the observed symptoms according to the methods of clinical neurology and clinical psychiatry. Inasmuch as anatomy teaches the absolute correlation of neuronic structures within the entire human body, it is an idle distinction to say that hysteria is a psychosis or a neurosis. Splitting the difference by calling it a psychoneurosis simply implies that the nervous adjustments of the different parts of the body, one to another, as well as the adjustment of the individual as a whole to his environment, show various types of disturbances called hysterical symptoms.

Hysteria is a general tendency to certain reactive expressions. The difficulty in description is an evidence of the instability of the concept, its width, and its fluctuating outlines. There lies in every person the possibility of the hysterical reaction, and it all depends either on the stimulus or the change in the resistance to bring it out. Pitt's satirical comment that "every man has his price" has its analogue in the truth that every individual has his hysterical Jack-in-the-box. Whether it will go off or not depends on the strength of the spring and the force put on the lid. When one finds a malady due to thousands of causes, it makes one conclude that either none of them has anything whatever to do with it, or that each may play a minimal role, and so it would seem with the hysterical personality. Whereas one man braves the

loss of a million dollars with patient forbearance and quiet dignity, another has a hysterical dream state after losing a purse. It may take the loss of a husband or a child to unloose the hysterical mechanism of one, while the death of a pet parrot is sufficient to keep some women in bed a month with an attack of hysterical paraplegia.

The aphorisms of a people reflect the aspirations of their affective lives. In the one to whom the phrase "Still waters run deep" applies, it will take a severe shock to unhinge the mechanism of affective control. So it is with the many small difficulties of life, by the one digested and forgotten, by another ever rising like the garlic of a bygone dinner and producing its disturbance in the affective sphere.

The difficulty that all students of the hysteria problem have encountered is to pick out those phenomena which are of the most significance, and which perhaps offer the most tangible lines of explanation. The various theories just discussed represent these master keys, which have seemed, for their authors at least, to have unlocked all the doors of the hysterical crypt.

So far as present clinical experience goes, one enters the hysteria domain by the same roads that one has entered the field of internal medicine, the field of psychiatry, the field of epilepsy. The time has long since gone by when four juices and their perturbations explained all the phenomena of internal medicine. Excitement, depression, and dementia no longer constitute the high roads through the kingdom of psychiatry. Epilepsy no longer remains a unity, explicable through any single cause, and so it is with hysteria. It is a vast hierarchy, almost a social organism in the psychical life of the individual, and clinically must be so regarded.

**Hysterical Character.**—For didactic purposes, the symptoms of hysteria may be divided into two main classes. The foundation is a psychically abnormal character—the hysterical character, hysterical temperament—engrafted upon this are a variety of nervous and psychical phenomena due to a pathological suggestibility. Suggestibility is used here in the biological sense already outlined, not in the restricted sense as used by Babinski.

The hysterical character is chameleon-like in its variations, appearing in all classes, in the intellectual as well as the weak-minded, and baldly expressed, shows remarkable similarities to the childish type of mind. Leaving aside the minor variations, it shows itself chiefly in emotional instability or lability, in its ability to be influenced readily, in negativism and impulsiveness, in a tendency to make sensations, a remarkable egotism, desire to confabulate, to fabricate, and to simulate. Seen from the standpoint of the psychiatrist, these mental attributes stand in the foreground; the neurological manifestations are dependent upon them, and the tendency of modern analyses of hysteria is to push the psychical anomalies forward.

In the child, one finds the prototype of this character, hence some authors speak of hysterics as grown-up children, others, again, as educated savages. Students of comparative psychiatry find these features widespread in lower races. Naturally in the weak-minded one looks for much the same types, just as one expects to find them in those grown-up individuals widely classed as degenerates or, better, as Walton suggests, deviates. The female represents in the main the physiological type of such character summaries, and Janet has made a lasting contribution to the subject in advocating his idea of a "defective synthesis," as a word picture of what occurs in those who show these psychological traits to excess.

From this standpoint the advent of hysterical signs in comparatively healthy individuals under the influence of fatigue, of old age, disease, intoxications, of shock, and of trauma becomes comprehensible. Here, again, Janet's phrase "reduction of the mental level" is illuminating. Under such circumstances synthesis, which has been adequate to meet most of the exigencies of life, breaks down—becomes dissociated by reason of a sinking of the mental level, and permits physiologically younger and simpler types of reaction to dominate conduct. The use of the concept subconscious seems unnecessarily confusing.

Naturally the complete exhibition of these features is rarely seen in any individual case, save in the severe degenerative hysterias, and those well on the road to a hysterical psychosis. Practically one may encounter patients with well-developed hysterical symptoms, with almost complete absence of the features just enumerated, barring perhaps the element of heightened suggestibility.

**Emotional Instability.**—Excessive lability of the emotional life is most striking. The mood is constantly changing. They are readily hurt, and break into tears on the slightest reproof; again, they show a passionate scorn and relieve themselves by violent bursts of anger. Gaiety changes into depression, and prolonged periods of happiness are followed by equally moody depressions. This changeability of mood shows itself in their activities. They are happy and busy or discontented and idle, sympathetic one moment and antipathetic the next; their mood can rarely be counted on. Smiles are succeeded by frowns and friends and relatives are at a loss to explain this change, or are constantly on their guard to avert its consequences. Thus many such patients develop into the "spoiled darlings" of the household. Sydenham expressed it well: "All is caprice. They love without measure those whom they will soon hate without reason."

In male hysterics particularly, one observes a greater tendency to prolonged depressions, which anomaly undoubtedly had its effect in influencing the earlier writers, particularly Sydenham, to term these patients hypochondriacs.

**Suggestibility.**—Volumes have been written analyzing the pathological suggestibility of hystericals and science is in practical accord in acknowledging this trait as one of the most fundamental in the genesis of this mental state. It is well known that many claim it to be the master key to the whole problem, and the classical description of Babinski, who would set apart a special group in the hysterical medley under the term pithiatism, is founded upon this feature alone. True pithiatism (hysteria) for Babinski consists in those conditions which can be brought about by suggestion and cured by persuasion.

This peculiarity is to be sought in a special type of idea associations so well studied by Jung and Ricklin of recent years. In the foreground of the hysterical reaction type—for one can speak of general types only—are to be found more or less independent active idea complexes of great affect value whose development is enormously greater than in normal individuals. The association test reactions are riddled through and through with disturbances due to these complexes, many of which are only slightly suppressed by apparently harmless reactions.

Up to the present time no adequate notion of what is meant by suggestion and suggestibility has been presented. Fortunately, psychiatry has freed

itself from the notion that it is due to Zeus, Juno, Apollo, or other deity; the middle-age witchcraft and devil possession has disappeared, that is, in so far as the name is concerned. The essential ideas, however, are still compelling in the minds of the populace, but set in phrases, such as telepathy, mind influence, absent treatment, and the like. It is almost commonplace to remark that 90 per cent. of the populace (certain cynics put it higher) do not and cannot think for themselves; they must, therefore, be led by the few, and adopt the phrases, formulæ and ideas of the few. This simply means that practically all people are more or less easily influenced or led by the suggestions of others. What is common to so many is found greatly exaggerated in a few—these are, for the most part, the hystericals, but it is only to certain types of suggested action that they are suggestible. Janet speaks of them as showing the type that leads to dissociation as a result of the narrowing of the field of consciousness.

Suggestion and suggestibility pervade the entire hysteria problem in terms of varying vagueness that are inexhaustible.[1]. They cannot be discussed further here.

**Hysterical Negativism.**—Many studies in so-called hysterical negativism have concerned themselves with the graver psychosis, dementia præcox, but the same mechanism is met with in the hysterical. One meets it in the frequent antipathic moods, when the patients are mute, or give only monosyllabic answers, when they refuse to eat at the table, or only toy with their food, but eat large quantities of sweets in their rooms, or steal down to the cupboard late at night. One sees it in an exaggerated spirit of Christian resignation and renunciation. The extreme capriciousness and impulsiveness of the fully educated hysteric shows most exquisite negativistic phenomena.

**Hysterical Sensationalism.**—To be constantly in the limelight, be it for purposes of attracting attention or to invite pity, is another feature. Romantic accusations, sensational confabulations, self-mutilations, and refined theatrical attempts at suicide—hysterical dream state suicidal accidents are not meant here—these have their motive power in this desire to be the observed of all observers. Lies, slandering, disfigurations, and stealing are not too much to bring this about. In the present state of journalistic enterprise in certain countries, opportunities are not wanting to gratify these ambitions. The thoughtful see in such hysterical journalism the source for much hysteria of the proletariat.

The exaggerated ego of the hysteric has always given rise to comment from the earliest times. The attention is fastened pathologically on the ego, and the constant search for sensations within the patient's own body on which to hang complaints is pursued with a definite pleasure and refined meditation. The slightest sensation is caught hold of and magnified. Somatic pains, for instance, are not created by the neurosis, but are merely utilized, aggravated, exaggerated, and retained by it. Careful examination, psycho-analysis, as well as physical, of so-called hysterical pains will nearly always find a nucleus of organic foundation. Freud makes the acute observation that it is always the common, the most widespread pains of humanity that seem to be most frequently called upon to play a part in hysteria.

[1] Consult Bleuler, *Suggestibilität Negativismus und Paranoia;* also Sidis, *Psychology of Suggestion*, New York, 1909, 2d ed.

The pains of others, however, excite but very little interest and frequently one sees jealousy and envy arise if others' pains are considered of moment in comparison. To be sick and the centre of the stage becomes a lifework, a theatrical apprenticeship, which with masterly tutelage by many physicians, usually produces a finished artist in the end. Such were the famous Charcot cases, which the neurologists of Paris, the pupils of Charcot, now admit were the products of a too fervid culture. The negativistic phase, martyrdom, is a self-evident corollary.

Special attention should be directed to the tendency to confabulation. Originating in day dreaming, in the delights of fantasy, poetic fiction gradually passes through the stage of unconscious warping of truth to deliberate falsification. The will to believe becomes pathological, and a facile imagination soon supplies all of the gaps of actual observation even if it does not supply amnesia itself. Ganser's syndrome, as well as the confabulations of Korsakoff's psychosis, afford opportunities for studying the mechanism of hysterical confabulations of the most pronounced types.

**Motor Disturbances.**—*Attacks.*—The convulsive attacks have always attracted great attention and contributed the most striking phenomena. The capacity for this type of conduct has existed from the earliest days and it is one of the most remarkable and persistent of the features of hysteria. This transmission down through the ages, of precisely the same positions, attitudes, cries, and contortions, shows that fundamental human mechanisms are being played upon by the most primitive agents. The ancient representations of major hysterical attacks by artists do not vary from modern photographic studies of similar cases.

The striking major hysteria attacks are, however, the exception rather than the rule, and have varied so little in their manifestations that little can be added to the older descriptions. The matter of absolute frequency is a question that cannot be resolved, since there are so many factors as to make generalizations hazardous. Some physicians never see any such phenomena. It is well known how German students maintained the absence of major hysteria, while the Charcot school was richest in observations. The studies on hysteria from Germany are triple in number of those of France of to-day. Epidemics come and go, local agitations and general excitements give rise to the proper milieu for major hysterical manifestations; the possibilities seem always there in all peoples; the environmental factors may only be lacking. The religious revivals of one people are considered as manifestations of major hysteria by another and there are plenty of contemporary movements that offer excellent opportunities for studying the manifestations of major hysteria.

Statistics are notoriously unreliable in matters of this kind. Briquet, who made the first large enumeration, found that 72 per cent. of his hysterical patients (practically all women) had major hysterical attacks. Pitres, who has given a similar numerical summary, found 81 per cent. of 69 women and 22 per cent. of 31 men had major attacks. These were in the great Charcot times and under the influence of active imitative and suggestive factors. Binswanger's later figures show that 25 per cent. of 52 male patients and 54 per cent. of 80 female patients with clearly defined hysterical symptoms suffered from convulsive attacks. Gotz in his 75 patients found 3 per cent. to be affected. Voss gives higher figures for the men, 65 per cent. of 22 men, and 57 per cent. for 101 women. The latter author

makes statements regarding race, none of which can stand a thoroughgoing critique.

On the surface of things Slavic and Celtic races are more often affected, but in any such summary due recognition should always be given to the things compared. A polyclinic population, for example, will show absolutely different statistics from a clinic or hospital population, and conditions of education, of environment, and emotional stress have more relation to these manifestations than questions of race. Behind all such statistical summaries there is a marked lack of critique of the essential determining factors, and at present there are no trustworthy factors regarding the racial relations to hysteria as seen in major hysterical attacks. The Jewish race has always shown a higher incidence than other races, but the explanation is not apparent. Personal statistical studies do not confirm the usual idea so far as the frequency of major hysterical phenomena are concerned.

During periods of intense excitement the curves of incidence of certain affections of the nervous system are bound to show marked fluctuations. Famine, war, local or general disturbance, financial distress—all of the social factors which make an impress upon the emotional nature of man, cause great perturbations in the incidence of major hysteria.

With the beginning of major hysterical phenomena in an individual the tendency to recurrence is very strong. Data are wanting as to the number of attacks, but with young individuals daily, weekly or monthly (menstrual) attacks are not uncommon. There is a natural tendency for such major manifestations to gradually become less common with advancing years. Weeks or months and finally years will go by—even in untreated individuals—with few attacks and usually only the weak-minded or trained convulsionists remain. Other manifestations may grow up, constituting the interparoxysmal or postparoxysmal modes of the same capacity for reaction.

Notwithstanding the persistence in type of the grand attacks there are countless modifications. Those accompanied by contortions may be estimated as among the severest. Many authors with the example of the epileptic convulsion in mind have sought to erect a similar march for the major hysterical attack. Such attempts are not in accord with the clinical factors, and lack of uniformity is universal. If purely general lines are sought the hysterical attack may be described as showing three stages—a prodromal stage, one of muscular convulsions, and a poststadium.

The *prodromal stage*—one of extreme multiplicity, often, though badly, termed an aura—is most frequently one of mental unrest, and is shown either by restlessness, irritability, crossness, or depression; there is frequently some mental as well as motor retardation and a feeling of general distress, or a sense of tension which may lead to crying, fits of anger, or moodiness. With the fuller development a certain increase in intensity in a more restricted number of these phenomena takes place, anxiety increases, the sense of tension localizes itself more definitely, as clutchings at the throat, as palpitations, as the feeling that the skin might burst, the stomach explode, and similar feelings of tense constriction. Things dance before the patients' eyes, they hear sounds in the ears, become dizzy, and then begin to show convulsive movements. All of this may take a few days or a few minutes to develop; as a rule, the development of the earlier major attacks is longer than the later attacks, when, at times, apparently the slightest cause will precipitate an attack. In some patients the development is slow and ap-

parently very quiet. They will lie very still for a long time—perhaps all day—in bed with the head in the pillow, and then sudden respiratory spasms, yawnings, passing of gas, etc., will indicate the presence of the severe muscular spasms. Many apparently-to-be major hysterical attacks only get to the period of the motor outbreak. They are all aura and never show the discharge phenomena.

*Convulsive Stage.*—Here the usual initial phenomena are those of extreme muscular tension, which is tonic in character. It is most frequently a slow extension movement, the head being thrown back, the arms extended, fingers extended or in tight fist, legs straight and stiff, and toes extended. The face, rarely mask like, gives more often the impression of one in a day dream or phantasy, and the general tonus of the whole musculature is in marked contrast to that of an epileptic convulsion. The position of the body often assumes certain very characteristic attitudes, and the crucifix position is extremely common, especially among those whose associations have been extremely devout. The development of the position in point of time also shows considerable variation. Very frequently there is a progression from the head and shoulders, then to the waist, and, finally, the lower extremities, of a sort of snake-like extension that is like a gigantic overdrawn stretch. Local groups of muscles may show isolated extension positions. Some attacks consist entirely of these sinuous movements of extension with slow relaxations, and then slow extensions. The ancients, and many moderns, always interpreted these as having a strong sexual element.

In others the extension period is of but a few seconds' duration. Then the muscles relax, and a period of irregular contortional movements follows. It is not a true clonic spasm, the movements are too irregular, with rolling, tossing, kicking, and frequently screaming, with frequent recurrences of the tonic position, with opisthotonos, and then a recurrence of the rolling and individual clonic muscle movements. The tonic phases are much more coördinated than in those of relaxation—clownism is the proper application. Here a vast variety of individual muscular positions are manifest, boring of the head into the pillow, shaking it from side to side, threshing the arms backward and forward, rocking the hips, sudden sitting up and rocking backward and forward, or side to side, with hands tightly clasping the strongly flexed knees. Sometimes again the patient stands up and hops from foot to foot, or glides about, sometimes slowly and dreamily, with eyes widely opened, turned upward and arms extended, or suddenly breaks into a wild dance. In most of these cases the dramatic or theatrical character of the impersonation is very remarkable. The individual variations are too numerous to mention. It should not be forgotten, however, that some of the antics of dementia præcox (katatonic) patients have been described as being those of major hysteria.

The facial expression often shows in these various phases a similar changeability, sometimes devoid of any expression, again it takes on the character of one in great joy or hatred, or anger; it leers and squints, pouts and disdains. It is dramatic or ecstatic, repulsive or enticing, and psycho-analysis often reveals that the idea associations passing in the mind are such as are expressed by such facial movements.

This period shows extreme variability not only in the form of muscular expression, but also in point of time. There is constant change in many

patients, while in others monotony is the characteristic stamp. Some attacks come and go, others are over with in one explosion, but most attacks last some time—rarely less than fifteen to thirty minutes, more often one to two hours, frequently several hours, and in rare instances, days. Some hysterical dream states extend over months—if psychiatry is advanced enough to assure the differentiation from some abortive forms of dementia præcox or certain confused manic-depressives.

Notwithstanding the statements of many of the Charcot school that the major hysterical attack begins with a cry, it would appear that such a mode of onset is comparatively rare. It probably was an imitative phenomenon in France when the great cultivation of hysteria was in progress. The hysterical cry has rarely the same sound as that of the initial cry of the epileptic attack. Accidents not infrequently happen in the stormy attacks; the tongue may be wounded and a cyanotic color of the lips is frequent. Not much dependence can be placed on the presence or absence of mucus. Absolute increase in the production of saliva is rare.

Ordinary hysteria usually stops here. The patient gradually quiets down, the movements become less and less assertive, and a crying fit or a sudden scream may terminate the affair. After the attack there is usually a condition of great fatigue and depression. Post-paroxysmal mutism is a frequent sequel, and mild aphasic or paraphasic disturbances are frequent. Voss reports the finding of a myasthenic reaction.

A neurological examination during a quiet stuporous interval, or post-paroxysmal stupor, usually reveals no marked changes. The pupils are usually widely dilated and react to light for the most part; Voss and others report slow reactions; loss of pupillary light reaction is known; Redlich's studies show considerable variability; sensory tests are obviously impossible, analgesia being usual. Signs of organic paralysis are absent, and the deep reflexes are not markedly altered, but the skin and mucous reflexes may show marked diminution. In rare instances only will one find the patient passing urine and fæcal matter.

*Postconvulsive Period.—Dream States.*—The multiplicity of paths along which the major hystericals will travel is enormous, but about one-half of them, according to Pitres, less according to Binswanger's and Voss's observations, will pass into a condition of lethargy, in which they may remain for hours, days, in some instances, weeks. These are the patients concerning whom one reads so much in the yellow press—the "living dead" and "buried alive" cases.

These patients lie with closed eyes, immobile. The respiratory movements are hardly appreciable, and they often go for days without attending to the wants of nature. Careful examinations shows that the urine leaks away little by little in some patients; passing the fæces in bed is also reported. In others there is a condition of more active delirium; they talk, coherently or incoherently, to imaginary personages about them, a sort of semi-dream, spoken aloud, abounding in phantasy, and showing on analysis much of the characteristics of a dream, at times a typical delirium. Some patients in hysterical dream states show almost all of the characteristics of a katatonic dementia præcox, while, again, others behave like mildly confused manic-depressives. Ziehen's description of this general group of cases is particularly useful.[1]

[1] *Klinische Psychiatrie*, 1908, 3d ed.

The varieties of mental disturbance which may be arbitrarily arranged in this third group are countless. The attack often serves as the point of departure for many of the interparoxysmal phenomena. The patients wake up, lost, as it were, to their surroundings. They may have gone away from home in a somnambulistic state, and have little memory for what has happened, or only very hazy or isolated bits of memory concerning their actions. Patient analysis may bridge over the gaps and reconstruct a history of the mental wanderings, but unless the most severe self-critique is invoked, many of these attempts will reflect the subjective romantic dispositions of the investigator more often than they do the memories of the investigated.

It was a common experiment in the Paris hospitals in the days of Charcot, to press upon the ovarian region, mammary region, etc., to produce such major hysterical attacks, and as a result an extensive literature collected regarding these so-called hysterogenic zones; Charcot had his patients so trained that pressure on an ovary would cause an attack, pressure of the mamma would bring it to a close, and pressure on the two areas at the same time would cause no reaction. All this was nothing more than pure training. Scientifically one calls it suggestion; a great deal of it was humbug; not a malicious sort of deception, but a type of superabundant, uncritical faith shared in by both physician and patients. Hysterogenic zones there are none, if one restricts the meaning to the direct influence of definite peripheral irritations as capable of inducing hysterical attacks without the influence of psychical, suggestive factors on the part of the investigator. Again, it has been denied that major hysterical attacks could be consciously induced by the mere willing, but there is evidence that negatives this view.

**Course and Length.**—No two major hysterical attacks are alike. In general they are prolonged, usually lasting an hour or so, occasionally days, while lethargic states are known to persist several weeks. Some attacks are as short as a minute or two. Status hystericus lasting for several days is described.

*Varieties of Attacks.*—Kaleidoscopic varieties are to be expected. Monophase attacks are seen in those that show only the contortions, or others who fall directly into a lethargy or narcolepsy; more rarely mono-epileptoid phases are observed, and in these differentiation is difficult. Isolated cataleptic phases were frequent in the earlier descriptions; most of these are now relegated to the katatonias of the dementia præcox group, although apparently true hysterical catalepsies with flexibilitas cerea and hallucinatory ecstatic postures are known.

**Rhythmic Movements.**—*Tremors.*—The full description of the innumerable varieties of tremor in patients with complete or fragmentary hysterical reactions is due to the labors of the Charcot school. The three-fold classification that they offer (*a*) trepidation, (*b*) vibratory, and (*c*) intentional tremors has the merit of simplicity even if incomplete. Simple tremor when the hands are at rest is extremely frequent. Static tremor, when the fingers are strongly extended and spread apart, is less often seen, while intention tremor is comparatively rare. Mixed forms are the rule, and the polymorphic character of the tremor is diagnostic. All these types of tremor are usually rhythmic, but irregular, ataxic, choreic-like tremors are observed. The localization is manifold, eyelids (Rosenbach), shoulders, fingers, legs, tongue, mouth, etc. When involving the muscles of the mouth

or tongue one obtains the characteristic stuttering, or irregular pseudo-paralytic speech disturbances. Horizontal axis tremors of the head may resemble early paralysis agitans tremor.

In the hands they may be unilateral or bilateral, local or general, quick, vibratory tremors—8 to 12 per second, slower, 5 to 12 per second (pseudo-mercurial), or even slower, 4 to 5 (paralysis agitans like). From another point of view the tremors may be peripheral, radicular, or spinosegmental. The chief diagnostic feature of the hand tremors is that they rarely conform to a single type; they are polymorphous. Hysterical tremors are absent during sleep. Attention directed to these hand tremors and attempts at restraining them usually result in their increase, and sudden emotionl shock usually increases them greatly. In the act of eating they usually diminish or disappear. Isolated attacks of tremor are of further interest; such may persist for a week or so.

*Intention tremor* undoubtedly takes place in hysterical affections. It may resemble that of multiple sclerosis very closely, and is not infrequently met with in traumatic cases, especially in litigated ones. The combination of absent tremor in the supported hanging hand with intention tremor in hysteria is seldom met with. It is not to be forgotten that true multiple sclerosis tremor is all too frequently diagnosed as hysterical. The coincidence in similar localization of tremor with changes in sensibility is diagnostically especially significant.

Further rhythmic motor phenomena are hysterical hiccough, hysterical coughing, often extremely confusing, abdominal spasms, and hysterical shaking (shakers). Hysterical stuttering is a particularly obstinate type. Hysterical asthma, spastic hysterical aphonia (laryngismus hystericus) hysterical tachypnœa, dyspnœa, all fall within the rhythmic affections of the respiratory apparatus. Just which of these may be attributable to the influence of suggestion and which fall in the group of faulty diagnosis in Babinski's classical dismemberment time will decide.

*Coördinated Impulsive Movements.*—Hysterical crying and laughing are among the most ordinary explosions of hysteria, occurring for the most part in young women about the time of puberty. The middle grades of the hysterical temperament are prone to this type of expression, and in the histories of most hysterics one reads that such emotional expression was common in the earliest phases.

Binswanger offers certain diagnostic and prognostic signs concerning these attacks and the severity of the hysterical neuropsychosis in general. In one class one observes them after emotional shock in the absence of other factors. They are then to be considered as the expression of a pathologically emotional irritability, and the explosion is out of all proportion to the affect shock both with reference to its intensity and its duration. They betoken constitutional inferiority, usually on a definitely hereditary basis, particularly of the same type of disturbance in the ascendants. Binswanger is inclined to regard them, when symptomatic, of less grave importance than those of a second group in which an acquired or congenital neurasthenia lays the foundation. In the former group they represent perhaps the only disease feature of the personality, while in the second they seem to indicate a general instability and are more frequently accompanied by the development of a more complete hysterical symptomatology.

Other types of coördinated impulsive movement attacks are described,

a number of which are characterized by great uniformity; grimacing, hopping, etc. The trend of modern investigation is to ally many of these cases with the milder and even severer dementia præcox reactions. A characteristic failure of many of the earlier investigations of these movements was in not keeping the patients under observation for a sufficient number of years. Had this been done the development of dementia præcox would have been recognized. The symptomatology of hysteria might have been poorer, but a more correct understanding of the process would have been gained. Furthermore, the haziness of the whole concept would have been immensely cleared up.

Wernicke, in his masterly studies of what he has termed the motility psychoses, has directed observation in the right line.

**Arhythmic Movements.**—*Choreiform.*—While the tremors are usually rhythmical, irregular, incoördinated, unwilled, and large movements of the choreic type are occasionally met with, which offer particularly difficult diagnostic problems. This is all the more striking since combinations of hysteria with chorea are observed. Imitative choreas, which may attain epidemic proportions, are matters of history. Charcot further complicated the whole question by assuming the essential relationship of chorea and hysteria, a view that has support only on superficial grounds.

In all choreiform affections the important factors of infection or excessive growth with mental or physical fatigue should not be overlooked. To call a chorea hysterical in the absence of rheumatic fever is nonsense. Hysterical hemichorea is known. All patients with chorea are entitled to a rigid neurological examination, especial stress being laid upon the reflexes, and the more recent symptoms of involvement of the cerebellar and cerebrospinal systems. Spinal puncture should not be omitted in the severe cases. Chorea hysterica in the face of rigid neurological and psychiatric examination is becoming rarer and rarer.

Chorea electrica, Voss claims in many cases is hysterical.

*Myoclonus.*—Here the movements are larger, involving whole, functionally related muscular groups, and the contractions are rapid, tic-like in character. Whether there is a hysterical myoclonus is as difficult to decide as to assume practically all myoclonic reactions to be hysterical. To refer them to the tics is simply to push the psychogenic factor farther back in the past. This entire group of affections needs revision.

*Tetany.*—The difference between a true tetany and hysterical tetany reaction is difficult to state. The presence of Chvostek's, Erb's, and Trousseau's phenomena, with the special factors of occupation and of etiology, go far to establish the diagnosis of essential tetany. The test of calcium therapy affords further information. From the clinical point of view the signs known are only generally indicative; they are not positive, not even Erb's phenomena. In many hystericals electrical hyperexcitability of the muscles is marked. It is highly probable that further study of the tetany reactions may throw considerable light on the mechanism of the motor reactions in hysteria itself.

**Akinetic Motor Phenomena.**—*Paralyses.*—Weakness or loss of muscular power is among the most frequent of the more marked manifestations. There is the greatest variety, with reference both to extent and grade of akinesis. For many years clinical neurology was without definite criteria of the psychic nature of these pareses and paralyses, and although the

phenomena of hemiplegia and of monoplegia are found in the records of the early Greek and Latin writers, it has been only within recent times that the essential differences between organic and functional paralyses have been definitely formulated.

Statistically considered, about 25 per cent. of hysterics show definite akineses. In Briquet's series there were 125 in 400. Landouzy reports 40 paralyses in 370, while Pitres' figures are less than 17 in 100 with paralyses of the extremities. Binswanger reports 4 in 109, remarking that the paralyses are less frequently seen in Germany than in France. Voss, working largely with Russians, finds nearly as many as did Briquet, 27 in 123. Percentages will depend largely upon the type of clientele, and the special suggestive factors. Personal figures drawn from three types of clientele show less than 5 per cent. in what might be called private practice, perhaps 10 per cent. in hospital practice, and over 90 per cent. in litigation railroad accident work. Statistics are largely fortuitous.

Muscular weakness, myasthenia, of varying grades is found in the majority of hysterics; it may remain without developing farther, or be but the beginning of a paralysis. Maladroit suggestions can convert a paresis into a paralysis with surprising facility. These myasthenias, which are purely of psychogenic origin, due to the sense perhaps of helplessness, of anxiety, of dread, fear, displeasure, pain, etc., which affect influenced psychical processes and exert a depressing effect upon the motor innervation, are to be distinguished from the neurasthenic myasthenias. Clinically considered, however, they are mostly hysteroneurasthenic in character. Difficulties in diagnosis from organic myasthenias are often extreme, especially in early multiple sclerosis and certain obscure spinal-cord lesions, small circumscribed myelitis, slowly developing tumors, low-grade pressure neuritis, certain intoxications, etc.

Of the most outspoken paralyses, hemiplegia, paraplegia and monoplegia are the more striking; mixed types, triplegias, quadriplegias, etc., are also to be met with.

*Hemiplegia.*—This occurs in about one-half of the total paralyses (Ziehen). It may be incomplete—diplegic, arm and leg without cranial nerve involvement—the commoner form—or rarely with cranial nerve additions, or mixed hemiplegic with the opposite arm or leg. Still more rare are the quadriplegias, and alternate hemiplegias are curiosities. The onset of the hemiplegia is nearly always acute, often accompanied by a sense of weakness or giddiness, and almost invariably accompanying an affect shock; many develop after a major hysterical attack. The paralysis is at once manifest, and only rarely advances slowly. Occasionally pain, nausea, and vomiting accompany the development of the paralysis.

Clinically considered, these paralyses lack the signs of organic disease. The absence of involvement of the cerebral neurone is demonstrable by the absence of pathologically increased tendon reflexes, clonus, increased tonus, of Babinski's phenomenon, of Oppenheim's reflex, or modification of Schaffer's paradoxical reflexes, of Van Gehuchten's inguinal cutaneous reflex, the femoral reflexes of Remak, of Grassett's, Peaucier's, Hoover's, and Babinski's hip-thigh, associated movement signs, etc.

The integrity of the peripheral neurone is evidenced by the presence of knee-jerks and of the Achilles jerk, the absence of atrophy (until contractures may develop after long disuse), no nerve tenderness (unless

psychogenic), or unaltered electrical excitability, hypotonus, trophic skin disturbances, etc.

These are the usual diagnostic signs separating functional from organic cases, yet a review of recent literature emphasizes the need of excessive caution in attaching too much or too little importance to variations which undoubtedly exist. Thus while it is clearly realized that the tendon reflexes should be normal, Babinski maintains always, the knee-jerks are usually very active. Ankle clonus, called by some pseudo-clonus, has been reported by Binswanger, Sternberg, Westphal, and Voss. Oppenheim describes foot tremor, and Dejerine has demonstrated that patellar and foot clonus are exceptionally present in hysterical cases. Of recent years considerable discussion has centred about the possibility of the presence of inferior cutaneous reflexes, particularly Babinski's phenomenon in hysteria, heretofore considered absolutely diagnostic of organic involvement of the pyramidal tracts. Roth, Van Gehuchten, and others, however, report the presence of a Babinski toe phenomenon of occasional occurrence in hysteria. Since modern conceptions of the many factors which may cause a Babinski toe phenomenon have been so amplified, definite judgment is premature concerning this point.

Involvement of the motor cranial nerves seldom occurs in hysterical palsy. The cases reported of oculomotor and abducens paralysis are extremely doubtful (Borel, Parinaud), and the differentiation between a paresis and a spasm is extremely difficult. The cases of ophthalmoplegia externa should also be judged with the idea of hysterical contractures in view. Hysterical ptosis is not infrequent.

*Facial palsy* is of particular interest as a hysterical sign. All three branches may be involved, although participation of the supra-orbital branches is exceedingly rare. The whole subject of facial palsy, direct or crossed, is in need of review, and special attention must be directed clinically to the danger of seeing a palsy on one side when there is in reality a hysterical contracture on the opposite side.

*Glossolabial* involvement is reported (Remak), and considerable variation exists regarding the position of the tongue and the palate. Most of the anomalous cases seem to consist of a mixture of spasm and of paresis. In the hysterical aphonias the movements of the palate have been observed to be less ample. Inferior alternate paralyses with crossed facial are reported. They need to be carefully weighed (Voss).

Laryngeal palsy may also be reckoned among the paralyses; more frequently bilateral than unilateral it is rarely complete. Laryngoscopically, the picture is usually negative.

*Paraplegia.*—Paraplegia superior is extremely rare, while paraplegia of the lower extremities is more frequent, although statistics concerning its occurrence are not available. Inferior paraplegia is found either as a typical paralysis of the lower extremities, with inability to move the limbs, complete or in part, or as an abasia (astasia-abasia), in which there is conservation of the power to move the limbs while in a horizontal position, but inability to use them in the vertical position. Both the flaccid and spasmodic types of paraplegias have their analogues in the hysterical paraplegias, and contractures with these paraplegias are comparatively often met with. Transitional types are the rule.

Trauma, especially in young girls, or even children, is the most frequent

exciting cause. Railway accident hysterical paraplegias are of comparatively frequent occurrence. The onset is, as a rule, brusque, although its gradual development is observed, and Voss states that excessive tire of the lower extremities in anæmic young girls may be considered as a contributory cause. The distribution is apt to be extensive, although restrictive movements may be preserved. The patellar reflex is apt to be exaggerated in both types, but the absence of the signs already mentioned in the discussion on hemiplegia is indicative of the psychogenic nature of the disturbance. The functions of the bladder and rectum are usually normal, although several authors note involuntary defecation and urination. Mistakes in diagnosis, or imperfect observation, are here to be particularly guarded against, since the indefinite symptoms arising from small spinal-cord hemorrhages are not infrequently diagnosed as hysterical.

The distribution of the anæsthesia will largely depend upon the method of examination. A maladroit examiner can find almost anything he wishes, and since the usual methods for examining for sensory disturbances are largely tinctured with clumsy suggestions, the beautiful figures of the Charcot School students are better evidences of such methods than scientific documents. In examining for anæsthesia, the real point should always be guarded. The patient should never be asked if she feels, but should be questioned to distinguish between kinds of sensations. A consistent anæsthesia to all the types of possible test has never been encountered. The classical representations show a sensory distribution practically coinciding with the extent of the motor loss, but a great variety of irregular distributions has been figured.

Atrophy may follow contracture, but without reaction of degeneration, although Dubois has well shown that a diminution to both galvanic and faradic irritability in the majority of cases is possible. Steinert's study shows that the previously held ideas concerning peripheral lesions and electrical changes are too narrow and in need of revision. After years of inactivity, paraplegic contractures may be absent.

*Astasia-abasia.*—This is an irregular incomplete type of paraplegic disturbance, first described by Blocq, a pupil of Charcot, in 1888, differing from true paraplegia briefly in that there is no paralysis or ataxia of the limbs in the sitting or reclining position, but only when the patient tries to stand or to walk. Even complex motor acts may be performed, dancing, etc. Oppenheim, Binswanger, and others regard it as a syndrome met with in other conditions than hysteria. It is undoubtedly of psychogenetic origin, and, hence, should be considered as a hysterical complication of the other psychoneuroses, neurasthenia, psychasthenia, etc., rather than as an integral portion of these psychoneuroses.

*Pseudo-tabes.*—Pitres has given a full analysis of this allied disturbance. Pains, incoördinated ataxic movements, disturbance of the eyes, analgesia, etc., make a differential diagnosis extremely difficult at times. It is an interesting fact that Charcot made a diagnosis of true tabes in a patient who went to Lourdes and recovered. Hysterical symptoms in a tabetic are frequent, but with present clinical aids the diagnosis should not be difficult.

*Monoplegia.*—Hysterical monoplegias are extremely frequent, and very irregularly distributed. Mention has been made of the paralyses of the third, fourth, or sixth nerves, of the facial and of the muscles of the pharynx

and larynx. Even individual muscles of a nerve innervation group may be involved as in hysterical ptosis, single, or double, spastic or flaccid, internal or external rectus, etc., with horizontal nystagmus. Brachial monoplegias are perhaps the most frequent, leg monoplegias are rarer.

**Disturbances of Sensation.**—A searching study of hysterical anæsthesia is still lacking. Attention has already been called to the stigmata diaboli of the middle ages. During the sixteenth and seventeenth centuries hysterical anæsthesia was counted one of the signs of the influence of the Devil, and in the works of this period can be found nearly all of the various forms of anæsthesia which the investigators of the nineteenth century have made classic, after Gendrin's first definite expression in 1846 of the relation of alteration of cutaneous sensibility to hysteria.

Believing it to be premature to accept *in toto* the dictum of Babinski, that there is no such thing as hysterical anæsthesia, a brief recapitulation of the accumulated knowledge concerning these phenomena is desirable.[1]

The general mode of presentation as given by Ziehen in his recent monograph is the most practical:

*Disturbances of Touch.*—These consist of anæsthesia, hypæsthesia, and hyperæsthesia. Anæsthesia may be general, unilateral, isolated in spots or patches, or involving symmetrical regional areas. Hypæsthesia, *i. e.*, incomplete anæsthesia, is much commoner than anæsthesia. Total general anæsthesia is probably extremely rare. Pitres' statement that 20 per cent. of the cases show it, is one of the extravagances of the Charcot period. Binswanger considered only six of the many reported cases to be valid, and Voss has added six from literature as probably authentic examples, and two of his own observation. Some of these patients showed "dream" state conditions, and it is extremely difficult to correctly estimate their value.

Hemi-anæsthesia or hemi-hypæsthesia is not infrequent, although the usually accepted teachings of Charcot are certainly defective. The left side appears to be much more often involved, and considerable variation in intensity exists in the different portions of the affected side. Crossed anæsthesia, *i. e.*, face of one side, and body of opposite side, is also reported. Bizarre groupings, such as involvement above the waist on one side, and below the waist on the opposite side, are known. It is not impossible that some of the crossed anæsthesias have been really due to minute thalamic lesions. Patch-like anæsthesia or hypæsthesia is common. The breast and forearm are especially involved, but the irregularity in position, form, size, constancy, etc., is bewildering.

Geometrical distribution, the well-known stocking-and-glove type, which rarely corresponds to any anatomical nerve area, is classic. The border of the anæsthetic area is apt to be very indefinite, not only during an examination, but particularly on comparison with preceding examinations. The patch-like anæsthesias show a similar, if not greater, variability. As a rule, the patient is unaware of the anæsthesia, and in but few cases does it cause any inconvenience. Other patients complain of pain (anæsthesia dolorosa) or pricking, or compare the sensation to the crawling of ants, or the feeling of a limb that has gone to sleep.

Anæsthesia develops suddenly, as a rule, either following a major attack,

[1] See Voss (Schmidt's *Jahrbucher*, February, 1909), for a critical summary of Babinski's teachings concerning anæsthesia.

or without such a precedent. It would appear that few old hysterics are free from anæsthetic concomitants. The increasing opportunity for suggestive action is here manifest.

*Temperature Sense.*—Loss of ability to distinguish heat and cold appears to follow the modification of tactile anæsthesia, but cold nearly always has a striking effect upon the hysteric, especially when applied *en masse*, as by a shower bath or cold pack. A few, not truly convincing, cases of dissociation of temperature and tactile sense are on record, also cases of dissociation of warm and cold. Patients with loss of temperature sense rarely burn themselves, although burning in hysterical thermanæsthesia is recorded. There are no complete studies as yet on the behavior of these tested in accord with Head's teachings regarding epicritic and protopathic sensibility.

*Deep Sensibility.*—In a few isolated instances deep sensibility seems to be affected, but it does not necessarily impose its pathological corollaries—ataxia, etc. Hyperæsthesias and hyperalgesias are more frequent than deep anæsthesias. Dissociations with intact tactile and lost deep sensibility, or vice versa, are among the hysterical anomalies recorded that require more corroboration and more exact observation.

*Mucous Membranes.*—The sensibility of the mucous membranes may be modified in a manner quite analogous to that already noted for the skin. Thus anæsthesias of the mucous membrane of the mouth, nose, urethra, rectum, ear-drum, epiglottis, etc., are recorded. Thaon makes the startling statement that in one-sixth of all hysterics there is anæsthesia of the epiglottis, yet respiration pneumonia does not occur as a result.

*Pain Sensibility.*—Analgesia and hyperalgesia are both present. General diminution of pain sensibility, hypalgesia, is about as frequent as general hypæsthesia, and is found under similar conditions. Absolute analgesia is found in a few hysterical dream ecstatic states. Hemi-analgesia and hemi-hypalgesia run *pari passu* with the hemi-anæsthesia and hemi-hypæsthesia, and crossed varieties are described. The usual pupillary response to painful stimuli is obtained in many of these cases, thus the integrity of the reflex mechanism is assured even if there may be repression of the psychical pain associations.

Hyperalgesia is a common hysterical symptom. In one form or another it enters into almost every hysterical history. Hysterical points or zones of tenderness on deep pressure, usually unilateral, are extremely frequent. The most common of these are the iliacal, or so-called ovarian, pressure point, also present in men, inguinal pressure point, just above Poupart's ligament, epigastric point, mammary point, jugular point, the supra-orbital and infra-orbital points, and the vertebral points. There are extended discussions of the location of these points and their relation to the so-called hysterogenic zones. Suggestion is the important element, but there are certain areas where pressure with suggestion seems to have more influence than other zones; the same phenomena may be brought about, however, by other suggestive means. After all, the point has no real relation to the disease. The most classical points are usually those upon pressure of which physiological stimuli are more readily set up in the normal individual. Pressure upon other zones, or even the same point, may bring about the cessation of an attack. Pressure on these points may also determine localized pains, paralyses, contractures, ataxias, etc., and vice versa, it can be used to cause these symptoms to disappear.

*Spontaneous Pains.*—Such are found in perhaps 80 per cent. of all hysterics. They are exceedingly variable, but may be roughly classed as follows:

(*a*) *Topalgias* or circumscribed painful areas having no anatomical relation to any known nerve distribution. Unquestionably the study of these has been much confused by lack of careful consideration of Head's zones of reflex visceral disturbance and vice versa, hysterical topalgias have been often considered as evidence of visceral disease. The occurrence of topalgias as isolated phenomena should be interpreted more as evidence of reflex visceral disturbance than as indicative of hysteria, and the modern tendency is to recognize more and more the strength of Head's position, that a great mass of symptoms which have been regarded as hysterical are in reality but the peripheral reflex disturbances due to visceral disorder or disease. Further, many topalgias are found in the neurasthenic. Mastoid topalgias and appendical topalgias have given occasion for over-zealous surgical interference.

(*b*) *Neuralgias.*—In the earlier studies on hysteria these have been described as occurring frequently, but more careful inquiry has revealed faulty diagnosis, so that, at present, a neuralgia, even when apparently not conforming to the known anatomical distribution of a nerve trunk, should be diagnosed as hysterical only after careful elimination of organic features. Examination may reveal a cervical rib to account for a persistent brachial neuralgia or the presence of sugar in the urine may reveal the diabetic origin of another.

(*c*) *Arthralgia.*—Pains in the joints offer particularly difficult problems. Hysterical coxalgia should never be so diagnosed in the absence of a radiographic examination. Since the advent of such methods they have been much less frequently diagnosed, even when the arthralgias are accompanied by hypæsthesia or hyperæsthesia, contractures and disturbances of gait, apparently hysterical. Certain cases are so quickly relieved by suggestive therapeutics that the occurrence of true hysterical arthralgias seems certain.

(*d*) *Enteralgia.*—Many striking cases of visceral pains of undoubted psychical origin are in the literature. Mention has been made of hysterical appendicitis, concerning which A. Dubois has written an extensive monograph. Hysterical ileus, with pain and fæcal vomiting, is held by Babinski to belong to the group of "simulation," in which opinion Strümpell shares, but Bergman has collected the older cases, Voss the newer ones, and Naunyn believes it a possible hysterical condition. Recent studies on antiperistalsis by Muhsam[1] seems to point to the possible psychogenic origin of such reversed peristalsis. Vomiting is a frequent hysterical symptom, and even the hyperemesis gravidarum is held by E. H. Müller to be of possibly psychical origin.

(*e*) *Cardialgias.*—Binswanger describes cardialgia as a frequent hysterical symptom. In its severest forms—angina pectoris hysterica—it is undoubtedly very rare, but as palpitation, sense of oppression, irregular pains, quick pulse, dropped beats, etc., it is frequent. Hysterical bradycardia has been described by Triboulet.

In addition to the false cardiopathies of both hysterical and neurasthenic origin, there are the false gastropathies, mental anorexias, false enteropathies,

[1] *Mitt. a. d. Grenzgebiete d. Med. u. Chir.*, 1900, vi.

false genitopathies, etc., on which Dejerine has cast such a wholesome illumination. They have often been indiscriminately herded with the hysterical symptomatology, or with hysteroneurasthenia; many are found in Janet's psychasthenic group. In their purest expression they should be kept in a group by themselves as the false pathies. They frequently are complications of hysteria, and are too largely the product of bad medical suggestion.

*Disturbance of Vestibular Apparatus.*—Dizziness of vestibular origin as a pure hysterical reaction is undescribed, although Frankl-Hochwart's and Bouyer's cases of pseudo-Ménière's disease have been referred to this category, and hysterical aural vertigo has been described. Voss has also given an apparently clear case of aural vertigo of hysterical origin, operated upon without finding any organic cause. A close reading of these cases shows many deficiencies of examination sufficient to exclude them from the category of well-known but transient or even mild organic vestibular affections. It is better perhaps to acknowledge them as due to sufficient causes yet unknown than to shut ones eyes to careful methods of examination by calling such hysterical. The possibility of the occurrence of the hysterical reaction due to cerebellar (vestibular) disturbance is not to be overlooked. The same is true for a large number of the dizzy spells frequently met with. Binswanger has correctly estimated these as common complaints of mankind arising from a vast variety of transitory causes, weakness, tire, associated fatigue from eye movements, gastro-enteric disturbance, etc.

*Smell.*—The percentage of normal variation in smell is not yet definitely established, hence the work of Lichtwitz, who found complete and half-sided anosmias and other anomalies of smell, is not conclusive. Irregularities of smell in the non-hysteric are very commonly found by Ziehen, who makes a systematic investigation of smell in every case in his clinic. Gilles de la Tourette is inclined to regard such disturbances as of diagnostic value. Babinski fails to find any marked anomalies unless the methods of investigation are palpably suggestive. Voss finds 30 per cent. of his cases with anomalies. This is about the average run of variations found in the psychiatric clinic at Berlin for all cases. Voss finds the left side more often involved.

*Taste.*—Variations in taste for sweet, sour, bitter are not frequent, but are recorded by numerous authors. Hyperagusia is especially pronounced. Agusia is a frequent accompaniment of the facial anæsthesia. Hemilateral agusia and hyperagusia are described.

*Hearing.*—Hysterical deafness is rare, yet recorded by Binswanger, Habermann, Magnus, Barth, Walton, and others, yet changes in modulation of the voice, so characteristic of organic affections of the ear, are practically absent. Wieber makes some observations to the contrary which are striking. Bone conduction is also modified. Voss has described double-sided hysterical deafness in combination with hysterical paraplegia.

*Sight.*—Double-sided blindness is rare, and few reported cases are free from scepticism. Unilateral blindness is frequent. Normal eye grounds, unmodified pupillary reactions, acute onset under psychical shock are the usual diagnostic features, taken in connection with the other features of the hysterical personality. The test with colored glasses and colored word stimuli, with later association tests, reveals the fact that the patients see, and the image is not in the subconscious. It is voluntarily repressed.

The lid reflex to sudden illumination is a valuable diagnostic sign. The consensual light reaction has not been carefully observed. Voss reports a case with its diminution in a unilateral amaurosis.

Diminution of vision as a subjective complaint is extremely common; it usually involves the left eye, while restriction of the visual fields has become a classic since the work of Féré. Babinski denies its presence absolutely, and claims it to be the product of maladroit suggestion. Such has been largely the writer's experience. Such limitations are described as concentric and bilateral, and are claimed for a third of all the cases; Voss gives 25 per cent. Increased fatigue, inability to concentrate the attention, etc., are among the usual etiological factors. It is subject to great variation, and simulation and exaggerations are very frequent.

Central scotomata, sector and ring-shaped defects, are among the rarer anomalies on record. Disturbances in color perception are also on record. Achromatopsia for single colors is the most frequent, although apparent true color blindness is described. Seeing double with one eye (polyopia monocularis) was first described by Parinaud. Within recent years Voss reports four cases and negatives the ciliary accommodation cramp hypothesis in favor of its being dependent on an amaurosis. Ziehen suggests a hallucinatory basis, especially for Ulrich's patient, who saw things six times with one eye.

Pupillary disturbances may originate from psychical influences. Pupillary immobility is claimed as a hysterical symptom, but most authors maintain that even in the severest hysterical attacks absolute rigidity is absent. Voss believes he has seen slowed reactions during an attack—attention has also been directed to this author's observation regarding unequal pupillary reactions in one-sided amaurosis. Spiller has reported irregular pupillary phenomena in a case of hysterical chorea. Micropsia and macropsia are rare phenomena (Janet, O. Fischer).

**Vasomotor and Trophic Disturbances.**—Here may be conveniently grouped a number of phenomena which have excited great discussion; hysterical fever, changes in the skin, œdema, urticaria, with modifications of the secretions, and metabolic disturbances serving as the chief examples.

*Hysterical Fever.*—That hysteria may, without other complicating factors, give rise to hyperthermia is the conclusion of Manahiloff, who, in a *Montpellier Thesis* of 1903, has gone over the entire field critically and thoroughly. The fever is due to a disturbance of the heat regulatory centres. Kausch,[1] in a recent collected criticism, comes to the same conclusion. There are not wanting, however, those who contest the validity of a hysterical fever. Oppenheim believes in its possibility, and has recently reported personal observations, and in Voss' latest monograph the evidence is confirmatory.

The temperature curve is not characteristic, and the fever is found usually only in the severest forms, especially those with major convulsive phenomena. The grade of rise is not great, as a rule, yet temperatures of 104° to 106° F. have been recorded. In certain cases with fever the accompanying pulse and respiratory changes have not taken place, while in others, dyspnœa and tachypnœa occur. Curious anomalies are reported by several observers, such as an axillary temperature 2.3° F. higher than the rectal; differences

[1] *Mitt. a. d. Grenz. der Med. u. Chir.*, 1906.

on the two sides of the body, etc., Such variations are known to exist in organic lesions of the nervous system. A diagnosis of hysterical fever should be made only after every possible cause of hyperthermia had been excluded. A diagnosis of hysteria founded on fever alone is untenable.

*Skin Changes.*—Notwithstanding the excellent review of Cassirer[1] the question of the occurrence of hysterical lesions of the skin has not reached a unanimous conclusion. Babinski classes the usual skin eruptions, bullæ, gangrene, ulcers, etc., among the deceits or artefacts. There is no doubt that many are such, and there is further evidence to support the contention that hysteria may be solely a complicating factor in an individual with peculiar trophic skin phenomena, but there also seems to be no reason for doubting the emotional and psychogenic origin of many skin lesions.

Among the more recent contributions to the literature of multiple gangrene of the skin thought to be of hysterical origin are those of Kreibich and Matzenauer, who observed markedly increased vasomotor irritability in hystericals, in some of whom the slightest skin irritation would lead to gangrenous necrotic ulceration. Bettmann has reported a case of gangrene in a hysterical individual, due to the application of an extremely weak lysol solution, and he and Ziehen, Bayet, Thomer, and others find that in many hystericals the skin may be excessively delicate in its reaction to external irritants. It is not a far cry to interpret an acceleration of a reparative process to the same hysterical type. At any rate, in spite of the practically unscientific aspect of the Lourdes "marvels," certain interesting rapid cures of ulcerous processes are worthy of more careful consideration than has hitherto been given them.

Bullous dermatitis is not infrequently observed as a hysterical phenomenon, while among the rarer types of so-called hysterical skin disorders are reckoned erythema, urticaria, dermographia, ecchymoses, chromidrosis, vicarious bleedings, etc. They are to be accepted with considerable reserve for the vast majority of these skin changes have been artefacts. Dupré has suggested the term mythomania to cover a number of these chronic self-mutilators. Yet there remain a few cases in which this factor cannot be proved. Urticaria is so common in general that its necessary relation to hysteria may be dismissed.

**Œdema.**—The instructive discussion of the Paris Neurological Society did not settle the debated point of hysterical œdema, which was first noted by Sydenham, nor could any unanimity be reached concerning the relation of suggestion to œdema. The cases reported have been many, and a general classification into white and blue œdema is made, the former being soft, the latter hard. These come on rapidly, usually following an affect shock, and disappear as quickly. Pitres has made them disappear apparently under the influence of a magnet. They may be associated with sensory or motor symptoms and be distributed as a hemiplegia, a paraplegia, or a monoplegia. In some instances of blue œdema the skin is infiltrated and pits on pressure. In other cases there seems to be a local vasomotor paralysis, with no infiltration and no pitting. Andemach[2] has recently reported an unusually well-authenticated case, also with loss of the knee-jerks.

[1] *Die vasomotorisch-trophischen Neurosen*, Berlin, 1906.
[2] *Münch. med. Woch.*, October 26, 1909, p. 2222.

The exclusively hysterical nature of these œdemas is not yet conclusively established; but the preponderance of evidence is in favor of the possibility of their psychogenic origin.

**Bleeding.**—Hysterical phthisis has been, for the most part, relegated to the mistakes in diagnosis, as well as practically all of the gastric and kidney hemorrhages, but a number of observations of bleeding from the skin, mucous membranes, or viscera, often termed vicarious menstruation, etc., remain, after the deceits are exhausted, as bona fide hysterical phenomena. Many of these patients show a number of related vasomotor disturbances. The bleeding may be scanty or profuse, in which latter case, however, the general health does not seem affected. The locations of the bleedings described have been legion.

**Secretory Disturbances.**—The influence of hysterical emotional states on the secretory activities is evident, and a number of anomalies are recorded. The sweat glands usually functionate normally, but lack of perspiration, which may be unilateral, has been observed. Local hyperidrosis has also been described. Under artificial conditions, such as medication with pilocarpine, the sweat secretion has been made symmetrical, even in a hysterical individual who was evidencing unilateral secretory anomalies. The salivary secretion is more often diminished than increased. Yet a striking case of sialorrhœa has been described by Matthieu and Roux, in which there was a secretion of two-thirds of a liter daily. This case was complicated by excessive vomiting. Increased flow of tears or absolute dryness may be met with. Increased bronchial secretion, accompanied by coughing, or excessive swallowing of laryngeal or pharyngeal mucus, may also be noted, as well as increased nasal secretion with sneezing attacks. From the diagnostic side it may be borne in mind that unilateral and even bilateral disturbances of these secretions diagnosed as hysteria have proved to be cases of tabes, of multiple sclerosis, or of syringomyelia.

The urinary secretion is frequently very variable; at the present time one meets with fewer cases of absolute anuria than in the Charcot days, especially since ureteral catheterization has been made possible, yet in a few instances one finds relative anuria in association with hyperidrosis or excessive stools. Simulation is the usual explanation, for Babinski the sole explanation, but there are definite cases of relative anuria. Polyuria is more frequent. Matthieu has reported a case in which 30 to 40 liters of urine a day were excreted. During major attacks an increase of urine may be expected. Incontinence is rare, but occasionally occurs either as a paroxysmal or interparoxysmal phenomenon. In hysterical trance or ecstatic states attention must be directed to the bladder in order to avoid overdistention.

The intestinal secretions may be profoundly modified by the mental state. This has been known from the Hippocratic days, but made experimentally objective by the classical researches of Pawlow and his followers. Cannon has shown for the intestines what his predecessors have shown for gastric motility and secretory activity. Diminished or increased gastric and intestinal secretions are among the most frequent anomalies in the hysterical. Naturally their diagnostic significance is very slight, since such anomalies are frequent in other conditions. Hysterical diarrhœa is not infrequent.

**Metabolic Disturbances.**—The loss of flesh in some hysterics is classic. They may be true hysterics or the mental anorexias of Dejerine. A notable

example of the "human skeleton" cured in recent years at Lourdes belongs to this group. The loss of flesh is usually the result of not eating, but some patients grow thin in spite of fairly normal feeding. A few cases of hysterical adiposis are recorded, but their exact significance is doubtful.

The classical researches of Gilles de la Tourette have shown that in the interparoxysmal period there are no marked deviations from the normal metabolism. During the attacks, however, a modification of the usual relationship of calcium and magnesium to the phosphates of potassium and sodium may take place, *i. e.*, there may be an inversion of the formula of the phosphates. The most recent studies carried on in Binswanger's clinic have yielded no striking conclusions.

**Blood.**—No generalizations of value have yet come from blood studies. A recent work of Schultz shows that the oft-noted anæmia may be associated with even an increased hæmoglobin content; that the red blood cells are usually normal, and further, that as a result of the vasomotor disturbances in hysteria it is necessary to take blood for examination from different parts of the body. The conflicting results of previous studies are partly explicable because of lack of attention to this detail.

**Reflexes.**—The corneal reflex, conjunctival and palpebral reflexes are frequently diminished. The corneal reflex is rarely absent, and the secretion of tears is not modified, as a rule, even in the presence of diminished corneal and palpebral reflexes. Irregular distribution in the sneezing reflex is occasional. It has little diagnostic significance, and loss of the faucial and pharyngeal reflexes, on which much stress has been laid, is of minor value. They are frequently very sluggish in the non-hysterical, especially those who have had much treatment of the throat. In the hysterical, irregularities in the faucial reflex are more apt to be present than in the non-hysterical. Little is known of the jaw-jerk.

The triceps and radial periosteal reflexes are usually increased. In the presence of contractures they may not be obtainable. At times they are lost, but their diagnostic value is minimal. Marked irregularity would point to organic lesions. The abdominal, epigastric, and cremasteric reflexes are subject to considerable normal variation, but when unequal, organic affection should be searched for; mistakes occur by the contraction of the abdominal muscles. Even in the presence of anæsthetic skin areas the cremasteric reflex is rarely modified. The anal reflex may be absent; occasionally it shows hyperæsthetic sensibility in much the same manner as one finds in vaginismus—also a rare hysterical reflex. Anal hypæsthesia is not infrequent, especially in women, but what relation it has to the anal erotics of Freud is not yet determined.

The knee-jerks are usually increased, but, as a rule, symmetrically. Loss of the knee-jerk is undoubtedly present as a hysterical sign, although contested by Babinski. Many observations leave little doubt as to the actual occurrence of loss of knee-jerk during and after hysterical attacks. They are extremely rare, and may later permit of other interpretations. A similar position may be taken for the Achilles jerk. There is a known variability here. However, if tested by the kneeling method its absence is strongly indicative of organic lesion. In the observations of Nonne and Willroth, the interesting finding was a periodic loss of the tendon reflexes. In the interparoxysmal periods they were normal or increased. Attention has already been directed to the Babinski sign.

**Psychoses.**—The use of the term is to a certain extent tautological, since from one point of view hysteria itself is primarily a mental disorder, but putting aside a too strict interpretation of the word psychosis, under this head can be grouped a series of phenomena that bear at times a close relation to the somatic disturbances.

The most striking of these profound mental alterations, and one which has been understood only within comparatively recent years, is the so-called hysterical dream state (Dämmerzustand). For years it has been known that following an epileptic attack or during an alcoholic debauch a patient may show a condition of dreamy delirium, which may not seriously interfere with his general orientation or conduct, but during which ordinary normal consciousness is not operative. Similar attacks are known to follow major hysterical seizures, or may originate without any convulsive antecedents. These are of shorter or longer duration, persisting from a few moments to perhaps a few weeks. Occasionally such a dream state may be terminated by convulsive seizures. The most striking of these states may be observed during major hysterical attacks, but there are other types during which the patients seem perfectly quiet and contained, or they are only dazed. If consciousness is deeply clouded, one finds these patients in a continuous slumber, with closed eyelids, sunken head, and relaxed muscles. As previously noted, they are the "living dead" frequently commented on by the lay press.

In another type one finds the patients about, even attending to their daily affairs, but doing them in a mechanical automatic manner, engaged, as it were, in a deep reverie all of the time. Patients in this condition have taken long voyages, and on coming to themselves are at a loss to account for their actions. Many fugues, or flights, are due to these hysterical attacks. Occasionally such people commit criminal acts, and important and difficult medicolegal problems arise. Somnambulistic performances belong to this general type of phenomenon, although here the clouding of consciousness is rarely as profound as in the dream states following convulsive attacks. In certain of these dream states the patient rehearses in his delirium occurrences that produced the initial affect shock.

Whether the dream state be accompanied by motor restlessness, agitation, or by stupor and lethargy, there is usually after the attack complete or partial retrograde amnesia.

The grade of amnesia cannot be determined on a priori grounds. It is rarely a complete amnesia, but it may be.

A further feature of considerable importance is the occurrence of a peculiar type of conduct which was first clearly set forth by Ganser, of Dresden, as "Vorbeireden." Many of the patients, especially the younger ones, show a peculiar foolish behavior—quite like young boys of from eight to ten years of age. They are very short and snappy in their answers, burst out into uncontrollable laughter, imitate the cries of animals, and are extremely difficult to manage. In answer to questions they give indirect or inverse answers, half answers, contradict themselves—very obviously at times, apparently not caring just what they say. At first sight their answers seem like deliberate attempts at falsehood or simulation, but the great similarity in this type of case is so striking as to permit the recognition of the Ganser symptom. This type of answer is not peculiar to hysteria; it is found in alcoholism, in epileptic dream states, and is very common in

dementia præcox. It is an important diagnostic aid in many difficult medicolegal cases, but is largely regarded by ignorant jurists as "fake."

Hysterical dream states may be confused with similar dämmerzustände of alcoholic, epileptic, or of traumatic origin. The diagnosis of a hysterical dream state is founded on the grounds of a dreamy disturbance of consciousness, suggestibility, an extreme changeability of the conduct, on foolish, childish conduct, and the presence of somatic hysterical signs. Diagnosis may be almost impossible in the presence of alcoholism.

It is of extreme medicolegal importance to recall that the motives of normal consciousness may be carried over into the dreamy state, and that criminal acts which seem to have a perfectly well-recognized motive may be performed in a state of consciousness in which the disordered mind does not know the difference between right and wrong. The question of legal responsibility in hysteria is extremely knotty. It has rarely entered into the courts in the United States or England, but is well-recognized by Continental jurists.

Closely related to the Ganser syndrome is that of pseudologia phantastica, or pathological liars. This condition is well studied in the monographs of Binswanger and Voss, and particularly in the communications of Delbrüch. Pathological hysterical swindlers have been made the subject of an important monographic treatment by Kraepelin.[1]

**Course and Prognosis.**—Ziehen has coined the word "delective" to express the multiplex combinations of the hysterical manifestations. By this is meant that any symptom in the broad group may be chosen by the individual, and that in successive attacks the picture may be quite dissimilar. This renders it almost impossible to describe the course of this psychical reaction. In general, it is a chronic affair, no consideration being given at this time to the purely symptomatic hysterical outbreaks which accompany other disorders.

It has been only recently that the suspicion has become a conviction that many of the symptoms which mark the course of hysteria are of medical manufacture, and the varied groupings of the picture come into existence in response to suggestions on the part of the attending physician, nurses, or anxious friends. Psychical contagion is one of the most difficult features to combat in limiting the symptomatology. Too great solicitude that the patient should not tire herself, or exercise her limbs too much, may be the point of departure of a paresis or paralysis. This factor plays a great part in modifying the course of the disorder, and especially in varying its manifestations.

Although the course in ordinary hysteria may be regarded as chronic, much will depend upon the element of causation, the subsequent affect shocks, and naturally the factor of most importance, the mental foundation of the individual. In a young individual, subjected to a single affect shock, other things being equal, one expects a less protracted course with fewer variations than in an older individual subjected to the vicissitudes of constantly recurring causes for emotional disturbance.

The prognosis in general, so far as the somatic signs are concerned, is good, but here a general rule will not apply to the individual case. There are certain hysterical individuals who never recover. The hysterical constitution is so definitely established that, although the individual paralysis,

[1] *Allg. Zeitsch. f. Psych.*, 1906.

analgesia, or anæsthesia is recovered from, the patients go on to other manifestations. They become chronic invalids, the despair of all those connected with them. The severe degenerative hysterias—usually superior feeble-minded deviates, with hysterical complications, rarely ever recover. In the hysterical complications of organic disease one does not look for recovery. The neurasthenic hysterias present a hopeful prognosis if the affective incidents can be regulated and the struggle for existence better adjusted.

The traumatic hysterias, barring the other factors, *i. e.*, mental inferiority, psychopathic constitution, alcoholism, neurasthenia, etc., have a fairly good prognosis. The continuance of litigation, with its emotionally disturbing factors of expectancy, the desire to get even, and the constant suggestion to make one's self out as sick as possible in view of the effect on the jury, are the most pernicious features of the traumatic hysteroneurasthenias. It is not true, however, that these patients get well as soon as they are paid. In the long period of waiting, due to the incessant delays of the law, they often develop a true psychotic state that takes many years to efface.

In the type of hysteria met with in the well-bred and intelligent, the prognosis is fairly good in the young; with advancing years it becomes less hopeful. The most favorable types are those seen in childhood.

A few cases of fatal hysteria have been reported. The exact significance of these cases is far from being settled. The longer a hysterical symptom has persisted, the more sinister the prognosis, but paraplegias, hemiplegias, of three or even five years' duration, have been known to recover.

**Diagnosis.**—If hysteria may simulate almost every known combination of symptoms, its diagnosis manifestly presents features of more than usual difficulty. Year by year, however, the analysis becomes more and more searching, and it is only with reference to certain conditions that a differential diagnosis becomes complicated.

**The Epilepsies.**—In estimating the character of the major convulsive attacks, the epilepsies come into review, although the analogies are but superficial. Much has been written of the borderland states between these two conditions, but the more they are studied, and the more known of the hysterias, for both are not entities, the less real resemblance is found. There is really little doubt that the two conditions may be present in the same individual, and further it may occur that the same pathological process may be the determining moment for both conditions. Thus, a cerebral tumor may cause epileptic convulsive seizures, and the reduction of resistance due to the growth of the neoplasm may determine in an otherwise insusceptible individual a hysterical outbreak.

Sommer suggests a fourfold division of the complex cases: (1) Epilepsy, which symptomatically resembles the severe forms of hysteria; (2) hysteria, which symptomatically resembles genuine epilepsy; (3) epilepsies to which hysteria is added, as a result of the organic cause of the epilepsy; and (4) hysteria in which epilepsy occurs as a pure complication. The term hystero-epilepsy only renders the concept hazy, and is best avoided entirely, even in the Richer sense as a name for the major hysterics, although both Binswanger and Apelt believe in a hystero-epilepsy, in the sense of the co-existence of the two diseases.

Even though it be known that the positive signs fail in the differential diagnosis of particularly difficult cases, yet with continued observation a diagnosis is possible in the sense of the Sommer divisions just quoted.

In epilepsy the signs usually relied upon to make a diagnosis in the ordinary cases are loss of consciousness, complete amnesia, injury to the tongue or body, involuntary loss of urine or fæces (the former more frequent), immobility of the pupils (not invariable), clonus or Babinski phenomena at the end of the attack (also not always), exhaustion, and sleep. In the hysterical attack the loss of consciousness is nearly complete, the amnesia is island-like, or may be cleared up entirely by association experiments, or in hypnosis, the biting of the tongue or injury to the body in falling is apt to be slight, if present at all, the urine and fæces are rarely discharged involuntarily, only in rare cases are the pupils immobile, and ankle clonus and the Babinski phenomena are not seen. After a hysterical convulsion the patient is rarely sleepy, even if fatigued.

**Meningitis Cerebrospinalis Acuta.**—The occurrence of lymphocytosis and bacteria in the lumbar puncture and exact observation of the temperature have disposed of most of the pseudo-meningitis attacks of former authors.

**Organic Brain Disease.**—A number of organic brain affections may be the exciting causes of the outbreak of typical hysterical attacks. Hemorrhagic meningitis, brain tumors, cerebrospinal carcinosis, septic encephalitis, syphilitic meningitis, gummata, trauma with microscopic lesions, are among the causes known to have occasioned true hysterical convulsive outbreaks. It is of moment to realize that a number of such patients with organic affections have in their early stages masqueraded as hysterical —and under the eyes of clinicians of high standing.

**Multiple Sclerosis.**—Of all organic neurological affections this offers the most difficulty in differentiation from hysteria, especially in its early stages, and the problem is further complicated since the two disorders may co-exist in the same patient. The regressive character of many of the symptoms of multiple sclerosis further complicates the picture. Nystagmus, ankle clonus, Babinski's sign, and temporal disk pallor when present speak almost indubitably for multiple sclerosis. Confusion has been introduced by Van Gehuchten, however, in his reported cases of hysteria with the Babinski phenomenon. Most patients with multiple sclerosis can be markedly helped by suggestive treatment. This constitutes another occasion for question in the diagnosis. The disturbances in sensibility in the two affections may be identical. The newer studies on the knee-jerks by Weiler have shown important differences, and the use of the association test after the methods of Jung and Ricklin throw more light on the psychical mechanism and give further aid in the differentiation. When the two conditions seem to co-exist, the greatest care is necessary not to attach too little importance to the organic signs mentioned as being extremely rare in hysteria.

**Hemi-anæsthesia.**—The differential diagnosis of organic lesion of the sensory cortex, or the posterior third of the internal capsule, is often not simple when the picture is one of a pure hemi-anæsthesia. Here the search for astereognosis and for disturbances of deep sensibility must be made with great caution, avoiding the slightest intimation of suggestion in the examination. In the hemi-anæsthesia of the thalamic syndrome, the accompanying pain, slight paresis, disturbance of deep sensibility, of postural sense, slight choreo-athetoid movements, increased reflexes, no Babinski sign, all on the same side, are the chief signs pointing to an organic lesion. Thalamic lesions, when small, have been diagnosed as hysterical hemiplegias with hemi-anæsthesia, largely because of the absence of the Babinski phenomenon.

**Syringomyelia.**—Schlesinger, in his monograph on syringomyelia, has called particular attention to the vasomotor disturbances occurring in syringomyelia when limited to the posterior horn, which may resemble very closely those of hysteria. Such a localization is rare, but difficulties arise in the initial stages.

**Chorea.**—Hysterical choreas offer many points of confusion. In the more frankly organic choreas, with spinal fluid findings and slight hypotonus, tendency to adiadokokinesis and altered plantar response, the diagnosis is not difficult; nor in those patients with acute infectious disease, notably streptococcus infections. There remain a number of choreiform affections that recover rapidly on suggestive treatment, but even they should not necessarily be classed as hysterical. Choreas to be considered hysterical should be accompanied by other signs of that psychoneurosis.

**Neurasthenia.**—Ziehen has given the following rather didactic, yet practically useful differentiation:

| Hysteria. | Neurasthenia. |
|---|---|
| Mostly unilateral, patch-like or regional anæsthesia, hypæsthesia, and hyperæsthesia, and analgesia, hypalgesia, and hyperalgesia. | Sensibility intact or generally increased. |
| Pressure points with hysterogenic and eventually also hysterophrenic characters more marked mostly in one side, usually combined with hyperæsthesia. | Pressure points without such characters, usually symmetrical and with intact skin sensibility. |
| Paralyses not unusual. Headache localized in spots, rarely a sense of pressure. | Nearly always abnormal tire. Headache often band-like and sense of pressure frequent. |
| Visual fields diminished. | Visual fields only diminished under the influence of fatigue. |
| Smell, taste, and hearing often involved. | Smell, taste, and hearing involved symmetrically and usually in the sense of a hyperæsthesia. |
| Skin reflexes often unequally modified. | Skin reflexes rarely different on two sides of body. |
| Often typical attacks. | No attacks—or rarely attack-like emotional movements set up by affect action, mostly depressive ideas. |
| Mood excessively variable. | Mood irritable or hypochondriacal. |
| Intellectual activity disturbed, especially the attention. | Intellectual activity disturbed by reason of early fatigue. |
| Marked suggestibility. | Suggestion influences slight. |
| Sleep often excellent. | Sleep mostly bad. |
| Course polymorphous. | Course rarely polymorphous. |

**Neuralgias.**—The frequent attacks of pain of the hysterical are often confused with neuralgic pains of reflex or direct origin. The lay ideas of nerve distribution offer a clue to the diagnosis.

**Psychoses.**—Since hysteria is accompanied by, or founded upon, a modification of the entire personality, it is not surprising that hysterical psychical symptoms should be found in the non-hysterical psychoses. Nissl has stated that in from 10 to 12 per cent. of the psychoses in women this is so. It is particularly true for the manic-depressive group and in the initial stages of dementia præcox, particularly those with preponderating katatonic signs. Occasionally one finds typical hysterical pictures in general paresis, and the hysteria of senile and presenile depressions is classical. The hysterical dream states are diagnosed with great difficulty from similar conditions in epilepsy, alcoholism, manic stupor, catatonia, and traumatic dream states.

So far as *manic-depressive insanity* is concerned, the accompanying signs of divertibility, flight of ideas, of psychomotor activity and the press of activity are usually sufficient to make a diagnosis. Mistakes are common, since a knowledge of psychiatry is not widespread in the profession and the injudicious advice of marriage (the panacea for hysteria) has been a hideous mistake in view of the subsequent development of a marked psychosis.

*Paresis.*—Paresis should offer little difficulty. Irregular pupils, characteristic memory defect, speech disturbances, tremors, spinal puncture findings and the Wassermann reaction give definite signs.

*Dementia Præcox.*—The entire hysteria problem centres about this psychosis, and diagnostic problems arise, which at the present time are almost impossible of solution. Thus the differentiation of a catatonic stupor from a hysterical dream state may be impossible. Often the only help lies in the history. The sudden onset, after a psychical shock, speaks for a hysterical dream state, while a longer and slower course is indicative of catatonia. The foolish conduct, negativisms, catalepsy, amnesia, grimaces, analgesias, etc., are common to both conditions. A negativism that lasts for days and weeks is evidence against hysteria, and energetic refusal of nourishment points in the same direction. Hysterics even in a dream state are less liable to soil themselves than catatonics, yet in the deeply clouded states of consciousness such extreme grades of uncleanliness may be present. Unilateral pressure points and disturbances of sensibility are more indicative of hysteria, but not definitely so.

A stuporous state preceded by hypochondriacal ideas, by ideas of influence, and changes in character in the direction of emotional stupidity, or disinterestedness and menstrual disturbances, points to catatonia.

The remarkable analogy drawn by Jung[1] between hysteria and dementia præcox explains in large part why such a difficulty arises, and the present author[2] has attempted to show that the symptoms of the two conditions, although identical, are, nevertheless, representative of quite different stages in the synthesis and analysis of personality. As one in going up a mountain obtains the same view at any one spot as when coming down, so in the peculiar dissociations of personality in its still loose synthesis (hysteria)

[1] *Psychology of Dementia Præcox*, translated by Peterson and Brill, New York, 1909. Nervous and Mental Disease Monograph Series, No. 3.

[2] *Predementia Præcox, Amer. Jour. of Med. Sci.*, 1907, cxxxiv, 157.

and in the dissociation of a disintegrating personality (dementia præcox) one may obtain quite similar symptom pictures. The emotional apathy of the dementia præcox patient shows a striking resemblance to the indifference of the hysterical. Jung has shown that in both there are emotional complexes, which are covered up and hidden, or repressed. In the hysterical the mood does not last long, but is suddenly interrupted by an explosion, a crying spell, muscular contortions, and the like. The impulsive acts of the apathetic dementia patient are similar, although here the protective mechanisms are harder to penetrate and the painful buried complex more difficult to reach by reason of the mental disintegration.

The tendency to repress the unpleasant and to bury it as deeply as possible, which is a normal psychological means of adjustment, is seen in its diseased phases in both disorders. In the hysterical, following Freud's interpretation, one finds the conversions, the transmutations, whereas in the dementia præcox cases one finds the peculiar transpositions and blocking, which prevents adequate reactions to reality. Although Jung deprecates the use of the phrases "hysterical character" and "dementia præcox character," yet they are useful concepts, if one does not push the words too far. All types of temperament may be found among hystericals, yet it is characteristic of these personalities that a powerful emotional complex is present which is incompatible with the ego-complex. One encounters certain embellishments on the part of the dementia patients which show the influence of such emotional complexes, which are also present in hystericals. It is frequently seen in forms of studied and pretentious behavior, aristocratic gaits, philosophical enthusiasms, religious originalities, etc. These are frequent expressions of either the hysterical or dementia præcox reactions. Delusions of social elevation in dementia præcox frequently manifest themselves in exaggerated manners, studied speech, bombastic expressions, affected eloquence, and high-sounding phrases. One sees in the quasi-religious schemes, such as Christian Science, both hysterical and dementia præcox features.

**Treatment.**—No pathological manifestation is in such urgent need of therapeutic individualization as hysteria, and to lay down general laws of treatment is to invite failure. The vast majority of patients suffering from the hysterical reaction can be benefited, but it requires a great nicety of adjustment of means to ends, and a refined tact and at times almost superhuman ability to choose from the various resources at hand in order to accomplish permanent results. Without a fairly complete insight into the chief etiological factors in the individual case little can be accomplished beyond the mere removal of some of the more striking or annoying symptoms.

In no nervous disorder is *prophylaxis* more important and so universally neglected. For this latter reason it should occupy the first place in therapeutic considerations. It should include a knowledge of and a contest against all of the various influences that conduce to the development of the hysterical character or that encourage its further development in a susceptible individual. Such a survey of the modes of prevention must not overlook the importance of modifying the social milieu of the hysterical if special phenomena are to be avoided.

The facts concerning heredity teach that syphilis, tuberculosis, and above all alcoholism form the soil in which the best hysterical products are raised. Accidents during childbirth are also fruitful sources for the development

of the nervous constitution that falls a prey to the hysterical reaction. Thus, an enlightened eugenics would avoid the marriage of blood relations in whom nervous affections are present, and the marriage of syphilitics and the tuberculous should be most rigidly scrutinized. Only under the best of circumstances can one minimize the real dangers in the probable progeny of such marriages.

The hygiene of the pregnant mother, so essential from all other points of view, is doubly important from that of nervous inheritance. The general laws concerning hygiene of the nursling and child cannot be entered into here. Statistics would seem to show that children nursed by their own mothers run less risk of developing hysterical accidents, yet with improved pediatric methods the application of this must not be too rigidly insisted on, especially when the mother's health—not her comfort alone—suffers from the strain of continued nursing.

An unwise and unreasonable desire to stimulate the very young child is a prolific source of the nervous disposition. The nursing child should be left very much alone, and not made the plaything of every passing stranger, much less the centre of admiration of doting relatives and friends. Much has been written concerning the nourishment of the growing child, with the unfortunate result that there is a great deal of rampant faddism, and the boy or girl often gains that little knowledge which is a dangerous thing. Discussion of what the children should or should not eat, if at all necessary, should be carried on behind closed doors, and not made the tridaily subject of admonitive conversation with the family at the table. The exaggerated fancies concerning what is digestible and what is indigestible gained by the children from such discussions at meal-time form the basis of a multiform hysterical dyspepsia in after-coming years. There is probably no single means whereby pernicious suggestions play havoc with the mental health of children, and adults as well, than the continued discussion of digestive physiology at meals. Hot biscuits and other items of diet play a minor role in comparison with half-baked dietetic ideas constantly set forth by anxious parents and friends.

The merest common-sense notions of eating rationally and slowly are all that it is necessary for a child to know about his diet. If children do not care to eat certain things, it is extremely unwise to make a fuss about it. It only concentrates their minds on the implied importance of it. If left alone they grow out of the notions sooner than when a scene is made over every childish whim. Such commands fix the child's distastes, and prevent the free play of the important law of forgetting.

Certain articles of food should, however, be tabooed. These are alcohol and all alcoholic containing liquids. Tea and coffee are not good for growing children, and an excessive meat diet as well as an excessive fat diet is harmful. A mixed diet with well-cooked carbohydrates and abundant fresh vegetables is desirable. Avoidance of monotony in the meals will give appetite and zest to eating as well as a wider experience that tends to correct one-sided notions concerning food and food values.

The *mode of life* of the child is in need of careful regulation, yet the machinery of such control must not always be on view. An orderly day without interruption in its specified tasks and an abundance of sleep are imperative in the ages up to about eight, or the ordinary beginning of school period. The afternoon nap for the child up to six is desirable. Naturally, children's

parties, with rich food, candy, and the excitement of imitating the social whirl of their elders, are bad. Anything that overstimulates the child is disadvantageous. It has been found that single children are much more apt to be hysterical than those growing up in a large family, probably because the hotbed culture due to constant contact with older minds often develops immature fruit, expecially if the younger mind is overstimulated or is spoiled by excessive indulgence. It is also a striking fact that a single brother in a large family of sisters comes out a good candidate for the hysterical reaction.

The young child of nervous organization will need special physical and mental training. Gymnastics and games in the open are advantageous; hydrotherapy is useful if not overdone. The daily cold plunge is a useful tonic, but such methods may be very readily carried to extremes. The child's enjoyment of such procedures, as an index of his vim and energy, is a better guide to their usefulness than a rigid scheme of "hardening" the children. Summer sea baths or lake baths in the open give the ideal hydrotherapy.

Play is much vaunted, but it is in need of careful scrutiny. As a rule, the childish mind needs little stimulation of the imaginative faculties. The child lives in a sort of disconnected dream most of the time, and what is needed is not forcing in this quality, but direction of it. Just how this direction shall be carried out in the individual case must be left to the tactful mother. If she be hysterical, the most difficult problems arise, for then one has both heredity and constant suggestion to battle against. Under such conditions most children do better away from home, unless the mother has gained by hard experience and careful study the lessons that help her to remedy her own anomalies of inheritance or defects of bad training.

The factor of suggestion is practically identical with that of imitation. Here enter both the advantages and the dangers in the bringing up of the children. The child imitates its surroundings and adapts its imitations in accord more or less with its primary inherited likes and dislikes. These are, however, not immutable, and are made so only by the constant imitation of the parents. The children profit or suffer from the daily examples set before them, and by influencing the parents to the soundest methods of training one arrives at a scientific pedagogy that can eliminate the most pernicious hysterical weeds that a bad heredity can sow. The ability on the part of parents to be cheerful under all circumstances; to bear petty annoyances of life with equanimity; to keep their worries and angers to themselves, is a constant stimulus in the right direction for developing children.

The *school* question soon obtrudes itself, and here a host of difficulties arise, concerning which unanimity of opinion is obviously lacking. Too often one regulates the going to school by chronology, but experience teaches that no two minds are alike, and that there is a physiological age that may be different from a chronological age. In the mental sphere this is very striking, and it is sound pedagogy to attempt to teach a child this or that not according to his years or months or days, but according to his individual development. It is for this reason, if no other, that parents should know the teachers in a school, if outside schooling seems to offer the best resources, and a constant interchange of thought should take place between the school and the home. The stimulus in natural history that is everywhere apparent

in school work is to be furthered, as it leads to an intelligent appreciation and enjoyment in the open air. It is to be hoped that an extension can be made of the system of the German "mountain and forest trips," whereby children from ten and over can spend a few days in the open together under the supervision of trained teachers.

In connection with the education of the hysterically susceptible child, particular attention should be directed to the *emotional life*. Here is the weak point in the hysterical character—the lack of emotional control; and especial emphasis should be laid upon the training of this pathological lability of the affective life. Children should not be overindulged; their minds should be directed from their little aches and pains; they must understand what danger is without fearing it. They should know what hunger and cold are, and learn to disregard both. The observance of the wishes of others, and the need for accommodating their desires to the interests of others should be tactfully taught. Simply telling children these things is not real education. The opposing affective states must be called up, and thus self-control taught. The process of physical hardening is of no value if the emotional self-control is not acquired at the same time; fortunately the necessity for action and precision required in outdoor games, or indoor athletics, is a most helpful aid in acquiring control over the activities of the sympathetic nervous system, which is the most important link in the emotional chain. A proper sympathetic nervous system hygiene does not overlook the importance of regularity in defecation and urination.

A close control of imaginative literature is essential. Highly exciting tales are to be excluded, but they are rendered obnoxious, as a rule, by an early cultivation of the young boy's or girl's ideas of style in language and good writing, as the defects soon become so obvious that such kind of writing fails to attract by reason of its vulgarity or poor style. Hysterical adults would do well to avoid reading works that stimulate day dreaming, castle building, and especially the erotic.

The young boy or girl should be encouraged to build, to dig, to carpenter, to swim, to skate, to ride horseback, and works of phantasy should be kept in the background. Much of the handwork done by girls is bad, especially that which can be done mechanically, as it encourages day dreaming. If the stream of consciousness be directed in productive lines there can be no objection to such work. The cultivation of efficiency and productivity are in direct antithesis to the dreamy, hazy, inefficient, slipshod features of the hysterical character.[1]

Isolation and solitary pre-occupation are bad features for the predisposed individual, hence the question of the child's companions should be made a matter of special moment by the parents. Leaving these matters to adjust themselves by chance is usually a lazy method, yet it may be less harmful than the setting up of hypothetical, overdrawn, and impossible ideals. The real rub with companions, whereby the child must make the necessary adjustments and find himself, is more helpful if the wise parent sees the faults in adjustment and will take the trouble to work on them, than when he tries to seek an impossible degree of perfection in the environment. Young children who are wrapped in cotton wool invariably suffer from this fancied protection.

[1] See Payot, *Education of the Will*, Funk-Wagnall, 1909.

Hence good public schools and the large private schools are often much preferable to the smaller schools, where, unfortunately, much snobbery is rampant and special privileges are granted. These schools may not harm the normal child, many of them are highly desirable for special training, but they should not be selected for the child with hysterical, syphilitic or alcoholic antecedents, or for those who show the development of great emotional instability. The so-called "tender" or "sensitive" child must not be handled too tenderly, nor yet be left absolutely to his own resources.

The avoidance of sexual factors even for the young child is a *sine qua non*. In practice, however, it is much more difficult than in theory. For this reason nature study has an added claim for recognition. It not only brings the young bodies into the open air, but also brings the mind into contact with real things and actual forces. Their sexual education thus becomes natural and gradual, and sexual ideas are less liable to be distorted as the period of puberty comes on.

Special difficulties arise in the training of children who show a marked tendency to morbid anxiety, fears, and self-consciousness. Those who are afraid of being left with strangers, of sleeping in the dark, of going into the woods alone, of doing anything alone for the first time, are rarely helped by harsh measures. Such methods only tend to suppress their expression and force the children into expedients of lying and even hysterical conversions. Supposed headaches or other physical ailments are complained of in order to avoid exposure to disagreeable experiences, and thus the patient who should be really trained is forced into a hysterical defence reaction.

The principle of prophylaxis continues just as operative in the years after puberty as before, although the personality becomes more fixed and is modified with greater difficulty, but at no time can it be said that one's ideas become so firmly settled as to be incapable of some change, at least if only the correct method of approach be found.

**Etiological Therapy.**—Emphasis has been laid upon the fact that in many individuals possessed of varying degrees of the hysterical constitution physical factors may play an important part in determining a breakdown in resistance, and the development of hysterical symptoms. Many of these individuals are not really hysterical in the narrow sense of the word. They possess only that common fund of hysterical reactions with which all are born. With them, however, the stress is too much by reason of an added physical or social burden. To ascertain just what this is, and to relieve it is the problem of the attending physician. The diagnosis is often not simple, since considerable doubt may arise as to whether the physical disorder is primary or secondary.

The organs of generation in women call for special scrutiny, and although disturbances here rarely have any relation to the hysterical constitution, nevertheless, in those with such a predisposition gynecological disorders may break down an acquired resistance to hysterical manifestations. Even in the average woman pelvic disease may bring about hysterical outbreaks. Under any condition grave gynecological disorders require proper treatment.

It should not be overlooked that unnecessary gynecological interference in the typical hysterical approaches the criminal, or that major gynecological operations are a frequent and direct cause of hysterical breakdowns. The gynecologist has a stony path to travel in the hysteria domain. Attention might be directed here to the clinical group that Dupré has dubbed "mytho-

manias," since so many of them have close hysterical relationships. Many of these patients are obsessed with the idea of operation, and they invent their symptoms in order to gain their point. They invariably consult gynecologists, and—sad to say—succeed in gratifying their own wishes.

In hysterical virgins gynecological interference, barring definite and precise indications, is to be avoided. It almost invariably aggravates the condition, and by suggestion may become the nucleus of a "mythomanic" obsession. Too much stress cannot be laid upon this subject, for there is a great amount of harm done by gynecological tinkering in many cases of hysteria.

Another consideration with even less to recommend it is that of gastric disturbance. Of all hysterical manifestations, those of the stomach are among the most common, and numerous are the attempts made to influence the mental state by way of the gastro-intestinal canal; and, strictly speaking, without results. The good results are obtained through suggestion alone, and the bad results, also due to suggestion, are legion in number. Gastrotherapy, with its faddy dietetics—speaking only from the hysteria standpoint, be it understood—has contributed more to the actual production of hysteroneurasthenic stomach disorders than almost any other form of bad medical suggestion. This may seem an extreme position, but a clarified judgment forces one into it. No one has better shown this to be one of the great evils of extreme gastrotherapy than Dejerine, who constantly has under psychotherapeutic treatment the victims of "dietetic" therapeutics.

One special etiological factor demands an emphatic expression. It concerns the sexual life, and more particularly *marriage*. The hoary age of that etiological factor known as the unsatisfied uterine longing, and its supposed remedy, marriage, both of which are still popular, serve to show how extremely subtle the gradations are in the type of disorder under discussion. Its justification seems to rest on the universal fact that flighty young people settle down after marriage. But there is a great difference, from the standpoint of psychiatry, between the hysterical constitution and the irregular or often so-called hysterical conduct of young people, women particularly. Marriage does not cure hysteria, and whatever judgment may be reached with reference to Freud's ideas of juvenile sexual traumata as the cause of hysteria, it is certain that marriage on general grounds is dangerous therapeutic counsel for the hysteric. The solution of the problem of a contrary position, should marriage be denied the hysteric, is also difficult. Certain writers take the position that the physician should not advise under these conditions; he should simply state all the situation objectively and let the family decide for themselves. As an actual matter of fact, the specialist *is* called upon to advise, but his advice should be given in the form of a free discussion after all the facts of the case are available—the personality of the contracting parties weighed, their burden of heredity, their social opportunities, and attitude toward, and capacity for, meeting their responsibilities.

**Treatment of Developed Hysteria.**—Psychotherapy expresses in a word the most profitable mode of treatment. But the word conveys but a very inadequate idea of what is meant by the general notion. It here means all those mental methods, with or without physical adjuncts, whereby the personality of the individual may be influenced to a healthier degree of self-control. Psychotherapy includes all mental influences, from the use of the cheapest charlatanism to that of the highest pragmatic philosophies.

That which may be accomplished by a cheap trick for the ignorant coal heaver may have to be worked out with much labor by means of the most tactful dialetics with an educated college professor. A word, a command, may relieve a child of a hysterical paralysis, but it may require years of careful psycho-analysis to eradicate the same in a woman of intellectual and refined cultivation. The use of the command and "harsh method" for the latter would be as ridiculous as the psycho-analytical method in the former.

Psychotherapeutics is so wide that it is no wonder that it finds its practitioners in all spheres of life. Such have come into existence as a more or less direct outgrowth of the social milieu, and their teachings are more or less adapted to individual needs and ignorance. General laws are inapplicable, but there must be a fundamental principle of moral reëducation in order to obtain permanent results. One may have to run the gamut of bullying, and cajoling, of hypnotic hocus pocus, and appeal to social or religious prejudices; one must learn to play upon conceit and vanity, on love of family pride and desire for social prestige; the entire armamentarium of suggestive influences will be found necessary if one would conquer all cases, and the man is rare who can command them all. Hence in practice one falls back upon his or her natural bent, and invariably develops a one-sided or many-sided psychotherapy according to his natural endowment and acquired method.

Such positive psychotherapeutic treatment falls naturally into a few large groups, for the exclusive use of which one finds many special pleaders. The most important of these may be conveniently grouped as *hypnosis*, *suggestion*, and *reëducation*. In using these categories it is evident that they are not to be understood as different things or as mutually exclusive, nor as exhausting the subdivisions of the subject. They simply indicate general tendencies, rather than separate methods.

**Hypnosis.**—The concept of hypnosis has changed considerably since the early days of Liebault, Charcot, and Bernheim, yet the word is still frequently understood in the sense of an appeal to the miraculous, the superhuman, or the subconscious. Under its modern definition as only a form of suggestion it loses much of its significance. As used here it is understood as a mode of impressing certain ideas on an individual's mind after having, by trick methods, induced a condition of modified consciousness, known as hypnotic sleep, the hypnoidal state, etc., not to mention the numberless variants insisted upon by various students.

First, as to its applicability in hysteria, it is generally known that some are refractory. They cannot be hypnotized; they usually represent a higher level of intelligence than those who may be hypnotized. In such its efficacy is nil. Among those who are most readily hypnotized one finds the weak-minded, the mental inferiors of modern German classification. The appeal to the marvellous and the apparently supernatural has a great hold upon these, but since their hysteria is due to their real constitutional mental inferiority, the hypnotic suggestions are of very little permanent value; they simply reinforce and repeat the type of suggestibility that is an essential feature of the disorder.

Another type, how numerous they are it is difficult to decide, responds to hypnotic suggestions, not on a basis of weak-mindedness, at least not in the sense of general averages, but rather as a pure expression of the hysterical personality. To obtain a foothold with these patients it may be necessary

to start them with hypnosis, but with hypnosis alone one rarely cures a severe hysteria. It may be of advantage as an entering wedge, but if continued it only perpetuates the type of reaction we are trying to eradicate. Hypnosis seems to make some startling cures, but alone it does not modify; in fact, it renders the hysterical personality more susceptible. It therefore does harm as an exclusive mode of treatment, even when most carefully guarded. One is making real progress in the treatment of a hysteric when the patient has learned to be uninfluenced by hypnotic passes.

In the hands of the unscrupulous or the unpractised a great deal of damage can be done. The fancied influence for absolute evil that hypnotists are believed to exert on their subjects can in reality be accomplished only in the weak-minded. Such individuals do not need any particular pressing of hands or looking into mirrors to produce their lack of will.

It is a striking commentary on the value of hypnosis in the treatment of hysteria to find it practically rejected by its warmest advocates of twenty years ago. With the limitation thus outlined, however, it will always persist as a useful adjunct in the beginning stages of treatment for some patients.

**Suggestion.**—A definition of suggestion as used in psychotherapy is as unsatisfactory as that of hypnosis; and the difficulties have become more manifest since Babinski uttered his terse definition of hysteria as "that series of phenomena that can be produced by suggestion and cured by persuasion." It may be recalled that in 1891 Bernheim claimed that suggestion could do away with the hemi-anæsthesia of a hemorrhage in the optic thalamus; it could cure a multiple sclerosis, or overcome the palsy of a lead neuritis. Such a suggestive faith could move mountains, but it has no such import in the present section. On the other hand, there is the idea of suggestion which says that it is "some kind of an idea that enters into the mind in some sort of a manner," as Janet puts it. Such a use reduces it to a level with credulity, or belief, or unreasoning faith. Babinski has defined his own use of the word as "suggestion should express the action by which one endeavors to make another accept or realize an idea which is manifestly unreasonable, and that persuasion ought to be applied to ideas that are reasonable or which, at least, are not in opposition to good sense." Suggestion thus becomes a one-sided mechanism; it can bring about hysterical phenomena, but can do nothing to relieve them.

For our restricted purposes of psychotherapy, suggestion consists in bringing about an affect state by influences, the import of which are not apparent to the individual. It consists in inducing mental associations leading to the modifications of the patient's emotional and, therefore, psychical state that will cause actions which make for a better adjustment. Suggestions enter consciousness, either perceived on the threshold of consciousess, or on its margin, awaken ideas, associations, and in a manner similar to an endless chain bring about what Cajal has well termed "avalanche" action, which has a compelling force on the individual, who may be, and usually is, unmindful of the origin of the influences. The wise suggestor knows how, by little hints and side remarks, by appeal to fear, to jealousy, or to praise, to cause a summation of impulses which have an impelling force far exceeding that of a command. Such suggestions may thus have a very extended action, they may govern the activity of the glands of the heart or vasomotors, they may split definite idea complexes, and may

so affect the senses that illusions and positive and negative hallucinations may take place.

Suggestion used in this sense, then, is different from command or direct action, and it also differs from persuasion. A loose application of the word suggestion confuses these three procedures. Perhaps, after all, the distinctions are superfluous in practice, and the resourceful therapeutist makes use of all three. One may command a monobrachial hemiplegic to raise his arm; if uttered suddenly and in emphatic tones, success may crown the effort. One may urge and urge a patient day by day to walk, telling him he is getting stronger and stronger and will soon be well; this is persuasion or mediate suggestion. The hysterical bedridden paraplegic may suddenly get up and run in response to the cry of a child who is in danger; this is suggestion (indirect suggestion). The child's cry, its helplessness, the need for relief, make a continual emotional appeal of such power that the forces of inhibition are stampeded, as it were, and effective action results. By casually commenting on the erect carriage, and graceful, easy walk of a fellow patient, an astasic-abasic may be immensely helped, provided the praise and commendation be wisely apportioned and carefully administered. The vanity, the desire for praise, the egotism, the ideals of a patient must be correctly estimated in order to bring about corrective suggestions. Such suggestive treatment is usually combined with a purposeful neglect of the chief appearance in the disease, *i. e.*, so far as direct attention or questioning is concerned, and later, persuasion or complete discussion of the disorder can be added to reinforce the advance started by the suggestive ideas.

Command, suggestion, or persuasion are only symptomatic remedies; they do not change the chief factor which is responsible, namely, the hysterical character. Following the opening which they afford, a more fundamental method is necessary in order to permanently modify the personality and to make a recurrence of the manifestation less probable or impossible.

**Reëducation.**—This is the ideal psychotherapeutic goal. It aims to reconstruct the individual on a firm basis of reasonable and helpful philosophy that permits him to understand his weakness and his strength, his limitations and his powers. Mills[1] has well summarized a part of its aims in saying: "This method contemplates teaching the patient what he has, what he has not, what he seems to have, what he can do, what he cannot do, and what he simply believes he cannot do." But it is more than this. It concerns itself less with the patient's illness than it does with the steady cultivation within him of that individual mental and moral stability that makes true men and women, and not molluscs.

The exposition of the aims to be accomplished needs a short summary of the methods to supplement it. In a quick review of the individual patients, one is struck at once with the fact that they are very dissimilar and require quite different treatment. One recognizes that for the imbecile and weak-minded types, for whom perhaps only a cure of symptoms can be hoped for, hypnotism may be justifiable. An appeal to the miraculous relieves for a time, but the patients usually go on their way with ever-changing symptoms. Reëducation for such is a waste of effort, of time, and may squander a pittance which might better be utilized to keep the patient isolated and away from those particular parasites who feed upon the

[1] *Monthly Cyclopædia and Medical Bulletin*, July, 1908.

credulity of this type. The manifold fads, cults, and quasi-religions derive their followers and endowments largely from this class.

Granted a normal mental endowment in a comparatively young individual, one has a number of expedients. If neurasthenic factors appear large in the history, an *isolation* technique is advisable. Known under various names, it matters little who first invented it, but certainly Weir Mitchell did much to give it definiteness and point, nor did he neglect the reëducation features. The patient should be isolated, separated from home and family, preferably in a suitable hospital room or ward, or private house. Isolation at home rarely cuts off all the old ties, and certainly does not introduce the new scenes that pique curiosity and stimulate interest. The choice of a suitable nurse is of paramount importance. One's chances of success, in the vast majority of cases, depend more on the coöperation of the physician and nurse than on the particular methods pursued.

The daily regime must be rigid or pliable according to the case. The majority need a rigid schedule, the very strictness is a part of their moral rehabilitation. For food one commences with milk, three to four quarts daily—keeping close watch on the weekly body weight, then rapidly rising to five, six, or more quarts, as indicated by the character of the stools and the digestion. The patient is not permitted to see anyone; she cannot write or read, but must lie in bed. The physician's visits must at first be rare, but must be made an event in the day. One must bring to the sick room each and every time some new mental pabulum which can be worked over by the patient. It may be a chapter from Job, a fragment of natural history, a bit of vital human experience, a thought from Epictetus, perhaps a problem of conduct to be solved—something that remotely or directly bears on the subject in view, *i. e.*, the gaining of a new and healthy point of view of life, and the stimulation of moral fiber. The nurse throughout must have the same point of attack—not obtrusively so—and above all a cheap and boisterous optimism is to be suppressed.

Little by little, as the weight increases, reading may be introduced, but the books read need care of selection. One makes a great mistake in advising a book one does not know well. It may contain ideas that have a special affinity for the intense emotional complexes which are closely related to the patient's hysterical reactions. After four, five, or six weeks the patient may be permitted more latitude, both with reference to meals and associations with the outside world. This technique is to be found in many special manuals as worked out by hours, minutes, and seconds. To follow such too rigidly for all cases would be an absurdity. A regular schedule should be devised for each case and rigidly held to.

In addition to the general psychotherapeutic talks, which should not leave out of sight the emotional training as well as the more narrow intellectual, special days should be set aside for the discussion of some of the more intensely colored emotional features of the patient's history. In certain types of patients this may have to take on the character of a prolonged psycho-analysis in the sense elaborated by Breur and Freud. The tactics of these conversations require individual adjustment, and are a purely personal acquirement. Its principles may be reduced to writing, but its practice is acquired only by force of natural endowment and studied skill. The refined dialecticism of a Dubois, the charm of personality and resource of a Weir Mitchell, the hearty emotional appeal and camaraderie of a

Dejerine, are individual expressions of great masters in their particular line of therapeutic resource.

The use of religious ideas and ideals in psychotherapy calls for special mention. Since religion is an expression of the emotional response of the individual to the most ancient of all suggestive factors—namely, the riddle of existence and the hereafter—it is not surprising that it should play an immense role in the affect life of the individual. The evolution of the customs and usages of the priestly class symbolizes the expression of this emotionalism and thus sways it. Religious belief and its foundations should never be lost sight of in an enlightened psychotherapy, and the use of religious motives, of clericals themselves, is often of inestimable service. To the medical psychotherapeutist, however, trained as he alone is trained to properly estimate the etiological factors, physical, mental, and moral, which cannot be understood without a firm grasp of biological medicine, it is a sad sight to see the pitiful reversion in modern times to the pre-Hippocratic priestly mode of treatment of hysteria and kindred psychoneuroses. Emmanuelism, Christian Science, Mental Healing, and the like are but faint and futile grasps at the larger truths that medicine has made its own in the progress of twenty-five centuries. The successes of such practitioners are unfortunately offset by the manufactured hysterical products which such systems invariably produce. But it has already been pointed out that the practitioner of medicine is not free from responsibility in this respect.

Hydrotherapy, electrotherapy, pharmacotherapy, physicotherapy are necessary adjuncts, according to etiological requirements, but must be considered in the light of more or less temporary assistance. As exclusive mediums for suggestive influences they fall short of the ultimate aims of a complete psychotherapy; but as rational means to give physical tone, muscular activity, to correct disturbances of visceral adjustments, etc., they are necessary. The opium and hyoscyamus narcotics, analgesic antipyretics, and alcohol hypnotics are to be given with great caution, and never over any extended period of time. Iron, arsenic, and strychnine are not specifics, but are useful tonics. The salts of the alkaline earths, usually in combination with the phosphates, are indicated as quieting in excessive muscular irritability.

Valerian and allied drugs have no specific value, but it is not impossible to find combinations of the volatile oil class of drugs that have very definite tonic action on a dilapidated sympathetic nervous system, and as such are symptomatically valuable. The search for remedies having definite and selective action on the sympathetic nervous centres, on the cord, or their representatives in the cortex has only just begun. The treatment of hysterical delirium may require the bromide hypnotics—it is only under rare circumstances that hyoscine is necessary. Paraldehyde is, by reason of its extremely unpleasant taste, one of the most useful of the alcohol hypnotics.

**Analytical or Cathartic Method.**—No therapeutic consideration of hysteria can claim completeness without definite mention of the method originally termed the cathartic method by Breur, and later amplified by Freud as the analytical method. In its rough form it may be termed the "talking it out" procedure in psychotherapy, and the usefulness of the confessional in church practice is due, in large part, to the same principles. Freud's claim that the analytical method is one which acts most penetratingly and carries farthest

is undoubtedly well justified, but unquestionably its application has its limitations. Although at first practised with partial hypnotic addenda, Freud now practically abandons such methods. He quotes Leonardo da Vinci as saying "that the art of painting consists in placing little heaps of paint on uncolored canvas where before there have been none, while sculpturing, on the other hand, takes away from the stone as much as covers the surface of the statue contained therein." The method of suggestion acts like the former, that of analysis like the latter.

Freud's technique has slowly evolved, and a presentation that is valid for the present may change in the near future. His general procedure is to place the patient in the recumbent position, the physician sitting behind the patient's head at the end of the lounge. The physician thus remains practically out of sight of the patient, who is then asked to give a detailed account of his troubles, and to say everything that comes to the mind irrespective of its seeming logic or sense and apart from disturbing, mortifying, or unnice suggestions. In all such histories gaps are inevitable. These the patient is urged to fill in by thinking closely of the attendant circumstances, speaking aloud all of the flitting thoughts that pass during this search ("free association"). All the thoughts are requested to be uttered, notwithstanding their disagreeable nature. The patient must exercise no critique and remain passive. It will be found that the disagreeable thoughts are pushed back with the greatest resistance. This is made all the more striking since the hysterical reaction, *i. e.*, the symptom, is the symbolic expression of the realization of a repressed wish and gives the patient some gratification. A great effort is made to retain the symptom, especially as its origin is not really perceived, and since it represents, in symbol, the individual's former conscious strivings. In psycho-analysis one attempts to overcome all of these resistances and by a series of judicious and tactful probings reconduct into the patient's consciousness the hidden thoughts which underlie these symptoms. Every symptom has some meaning; behind it there lies some associated mechanism, the origin of which the patient unconsciously or partly consciously represses. In the psychoneurotic symbol may be read the cryptic expression of the original thought driven back and hidden.

To slowly analyze and pick apart the mechanism is the object of the analytical method. One needs not only special tact for such excursions into the subtleties of the mental life of some individuals, but also a developed method of interpretation. Every act, every symbolic expression or action, lapse in speech, mannerism, needs to be carefully noted and its bearing coördinated. Freud lays particular emphasis on the analysis of dreams, since he believes that in the dream the subconscious, or the "repressed conscious," is more apt to reveal itself. Hence a careful reading of Freud's *Significance of Dreams* is of the greatest value in this study, also his *Psychopathology of Every-day Life.* In his work on dreams he has developed to the full the chief directions along which his mind has travelled in the psychoanalytical method.

It is of the utmost importance to trace back into the earliest years the striking emotional influences that have come into experience, for, for Freud, the hysterical reaction consists in a perverted type of reaction to these experiences. As is known, the blurring, or loss of an emotional influence—an affect, in short—is due to a number of factors. In normal life forgetting

is the commonest type of a corrective adaptation, and forgetting is carried out with special ease if the emotional stress has not been excessive. Forgetting, however, is only a secondary phenomenon, and usually is more successful if the immediate reaction has been an adequate one. Such immediate reactions express themselves as tears, as anger, as impulsive acts, etc., and in such reaction the affect is discharged. In every-day life one calls it giving vent to one's feelings. If, however, the reaction is suppressed, the affect becomes united to the memory of the experience, and an emotional complex, or, to use a rather broad simile, a psychic boil results, which must heal by absorption, by discharge, or by other means. Freud uses the term ab-react (abreagieren) to signify the adequate reaction, or discharge, of such affects or their resulting complexes. Talking the whole thing over, giving vent to one's secrets and confessions, are well-known abreactions.

In hysteria certain of these complexes remain prominent; they are neither reacted too promptly, nor is their unpleasant feeling tone diminished by the blurring process of forgetting, although it is characteristic of the Freud point of view that the actual experience which gives rise to them becomes forgotten and the cause of the affect disturbance which becomes later converted, it may be into physical signs, remains apparently unknown to the patient. It must be dug out by psycho-analysis, and when once discovered catharsis takes place and the patient becomes cured.

It is further of importance to realize that abreaction is impossible for certain types of psychical shock or trauma. The inevitable, in the loss of a beloved person, is not overcome by frank discussions; social relations may make it impossible to mention the shock; or, again, it may be that it may concern itself with things which the person wished to forget, and which were intentionally inhibited and perhaps repressed from conscious memory.

In the limited space the full value of Freud's cathartic method cannot be presented. His original works must be consulted, as most of his critics apparently have not taken the trouble to read them. If truth be often stranger than fiction, certainly the charge of their being romances does not preclude their value. That Freud's cathartic method is very valuable for certain types of cases, especially in a class of the well-educated and cultivated, admits of no controversy. It is not improbable that Freud's particular clientele contains a large number of these very intractable cases which have resisted the easier, hit-or-miss method of suggestive therapeutics. It must not be forgotten that Freud is constantly applying the Dubois principle of reëducation in his analyses.

# CHAPTER XXI.

## AMAUROTIC FAMILY IDIOCY (TAY-SACHS DISEASE).

By B. SACHS, M.D.

The name, amaurotic family idiocy, was applied by the present writer to an interesting family disease of infancy, characterized by an entire lack of mental development, by a progressive weakness of all the muscles of the body, and by rapidly developing blindness, associated with typical changes in the macula lutea. The disease is generally fatal, the patients dying, as a rule, in a condition of complete marasmus before the end of the second year of life.

**History.**—In 1881 Waren Tay[1] published an article describing "symmetrical changes in the region of the yellow spot in each eye of an infant." The child was twelve months old. It could not hold up its head or move its limbs; there was weakness but not absolute paralysis of any part. Its entire brain development was slow and poor. At the first examination, March 7, 1881, the optic disks were apparently healthy, but "in the region of the yellow spot of each eye there was a conspicuous, tolerably diffuse, large, white spot, more or less circular in outline, and showing at its centre a brownish-red, fairly circular spot contrasting strongly with the white spot, and this central spot did not look like a hemorrhage, nor as if due to a pigment, but seemed a gap in the white patch through which one saw healthy structures." Tay thought that these appearances were similar to those of embolism of the central artery of the retina, and supposed the changes to be "possibly congenital." Five months later another examination was made, which showed that the disks had become atrophied, but that the changes in the macula lutea were the same as before. In the same family, according to Tay's reports, three similar cases have occurred, each one of the children presenting ocular symptoms and exhibiting physical conditions that were similar in all respects, and all three dying before the age of two years.

This peculiar ophthalmoscopic finding was noted by Magnus, Goldzieher, Wadsworth, of Boston, Hirschberg, H. Knapp, and others, but it was regarded merely as a rarity and was not suspected as being one of the symptoms of a distinct family disease.

Without any knowledge of these ophthalmoscopic reports, the present writer,[2] in 1887, published the history and the postmortem record of a patient who had suffered from a peculiar form of idiocy associated with blindness. The family character of the affection was not evident until a sister of the first patient, born several years later, had become similarly affected.[3] In the

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, 1881, i, 55: also 1884, iv.

[2] On Arrested Cerebral Development, with Special Reference to its Cortical Pathology, *Journal of Nervous and Mental Disease*, 1887, xiv, 541.

[3] *Journal of Nervous and Mental Disease*, 1892, xvii, 603; also 1896, xxi, 475.

course of the next six or seven years the author had occasion to see several instances of the same disease and to obtain the history of a family in which four children had been afflicted with this disease and all had died of it.

Kingdon, of Nottingham, was the first oculist to call attention to the fact that the condition which Tay described was practically part of the disease to which the writer had referred. In 1894 Carter collected all the cases of this disease known up to that time, and in 1896[1] the writer was able to give a list of 19 cases, of which 8 had come under personal observation, and by that time the chief symptoms of the disease had impressed themselves so clearly upon his mind that he felt warranted in giving the disease the name Amaurotic Family Idiocy. Since that publication numerous cases have been described by American and by some European writers. Among those who have contributed most to a complete understanding of the disease may be mentioned Kingdon and Russell,[2] Higier (who proposed the name Tay-Sachs disease), Falkenheim, Shaffer, Fry, Vogt, Spiller, Treacher-Collins, Apert, Holden, Poynton, Schumway, and Buchanan. An excellent review of the entire subject, with the complete literature[3] to 1909, and with a careful analytical study of 58 cases, is contained in the work of Wilbrandt and Sänger (*Die Neurologie des Auges*).

**Etiology.**—Up to the present time it has been impossible to assign any definite reason for the development of this disease in special families. In some of those examined by the writer there has been blood relationship between the parents, and in one of the families there had been innumerable psychoses among the relatives of both parents. Injury to the mother during pregnancy has been noted in at least one of the writer's cases. The family predisposition is evident from the fact that 28 cases occurred, to the writer's knowledge, in fifteen families. Carter was the first to call attention to the fact that all of the cases reported had occurred among Hebrews,[4] and even up to the present day not a single indubitable case has occurred among any other people. The racial feature of the disease is all the more astonishing because other diseases to which it is closely allied, and above all the juvenile form of amaurotic family idiocy, have been observed and recorded among all races and all nationalities.

It is important to note that syphilis is not an etiological factor. This is of interest not only because other family affections have been attributed to syphilitic infection, but also because the disease bears a distinct resemblance to specific disorders which are characterized by various forms of dementia and by ocular disturbances (Batten). Moreover, there is an hereditary optic nerve atrophy occurring late in life which is supposed to be due to hereditary syphilis, and with this optic nerve affection the present disease might possibly be confounded.

**Pathology.**—In 1887, before I had fully recognized all the characteristics of this striking affection, I described the condition as one of arrested

[1] *New York Medical Journal*, 1896, lxiii, 697.

[2] *Royal Medical and Chirurgical Society Transactions*, 1897, lxxx, 87.

[3] Robert Bing, in the *Ergebnisse der innere Medizin u. Kinderheilkunde*, 1909, iv, 82, has made a careful study of this disease in connection with other family affections; Apert (*Sem. méd.*, 1908, p. 25) collected 106 cases; of these, 73 cases occurred in twenty-five families.

[4] Bing is in error in restricting the disease to Polish Hebrews. My first two cases were of German Hebrew extraction.

cortical development and supposed that this was due to what I termed an "agenesis corticalis." I was much impressed at the examination of the brain of the first patient with the confluence of the central and Sylvian fissures and a complete exposure of the island of Reil.[1] The brain was unusually hard, even in the fresh state, and the knife actually grated on removing a small section of the cortex. On microscopic examination the most important changes were found in the cortex, in sections taken from the frontal lobes, the motor areas, from the base of the third convolution, from the first temporal, and from other parts of the cortex. The same changes were found in all the cells. I was struck by the fact that hardly a single pyramidal cell presented anything like a normal appearance. The contour of the cells was either round or elongated, and the cell protoplasm presented every possible change from slight to complete degeneration. In some cells the nucleus and nucleolus were entirely wanting or were relegated to the margin of the cell. A few years later Hirsch found that the same cellular changes occurred not only in the gray matter of the cortex, but in the gray matter of the entire central nervous system; not only in the cortex of the brain, in the cell ganglia, and in the gray matter of the spinal cord, but even in the spinal ganglia. This was corroborated by a later examination of one of my own cases,[2] and in 1903 I felt warranted in declaring amaurotic family idiocy to be a disease chiefly of the cortex and of the gray matter of the entire central nervous system. These findings have been fully corroborated by all recent investigators, among whom I wish particularly to mention Spiller, Kingdon, and Schaffer.[3] The last-named writer has, with astounding patience and accuracy, devoted himself to the study of the pathology of this disease, and has given a most thorough anatomical study of 8 cases. All of his findings have been splendidly corroborated by a recent study of my own, which was presented at the International meeting in Budapest. The findings of the disease are so characteristic that the diagnosis of amaurotic family idiocy can be made without hesitation from an inspection of the microscopic specimens. The figures show the characteristic changes.

Schaffer has pointed out that the chief characteristics are: (1) A widespread cytopathological process; an unusual swelling of the cell protoplasm and of the dendrites; a swelling of the hyaloplasm, which causes a mechanical destruction of the cell fibrils; ultimately the cell body is a mass of detritus. Curiously enough, the axis-cylinder is not involved in this general swelling. (2) Every cell of the entire central gray matter, both of the brain, of the spinal cord, and of the spinal ganglia, is similarly affected.

If we proceed to an examination of histological details we find, first of all, an unusual swelling of the cell body and a more or less complete disintegration of the cell protoplasm. The fibrillæ disappear, so that in many of the cells there are only slight traces left of the endocellular network. The fibrils near the periphery of the cell body are preserved somewhat longer than are those in the central part of the cell mass. The nucleus and nucleolus, if at all preserved, are, as a rule, pushed nearer to the periphery of the cell body, and as the disintegration of the mass goes on the nucleus

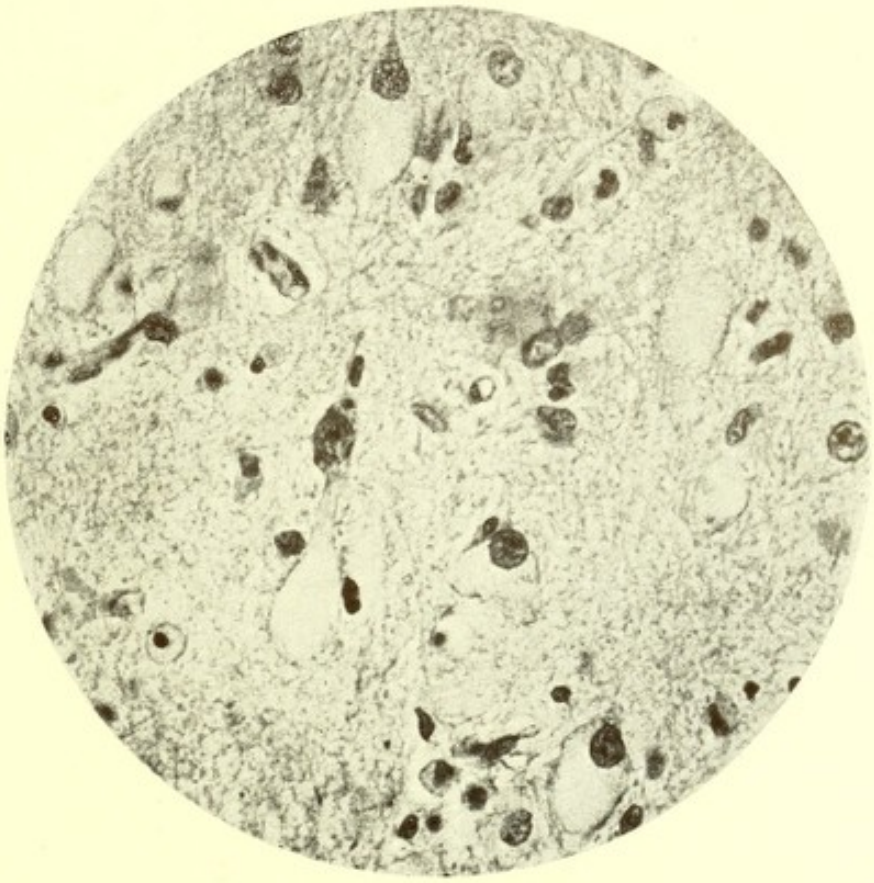
[1] I do not attach much importance to the confluence of fissures which I described in my first cases.

[2] *Journal of Nervous and Mental Disease*, 1903, xxx, p. 1.

[3] *Zeit. f. die Erforschung. u. Behandlung. d. Jugendl. Schwachsinns*, 1909.

# PLATE XXXVI

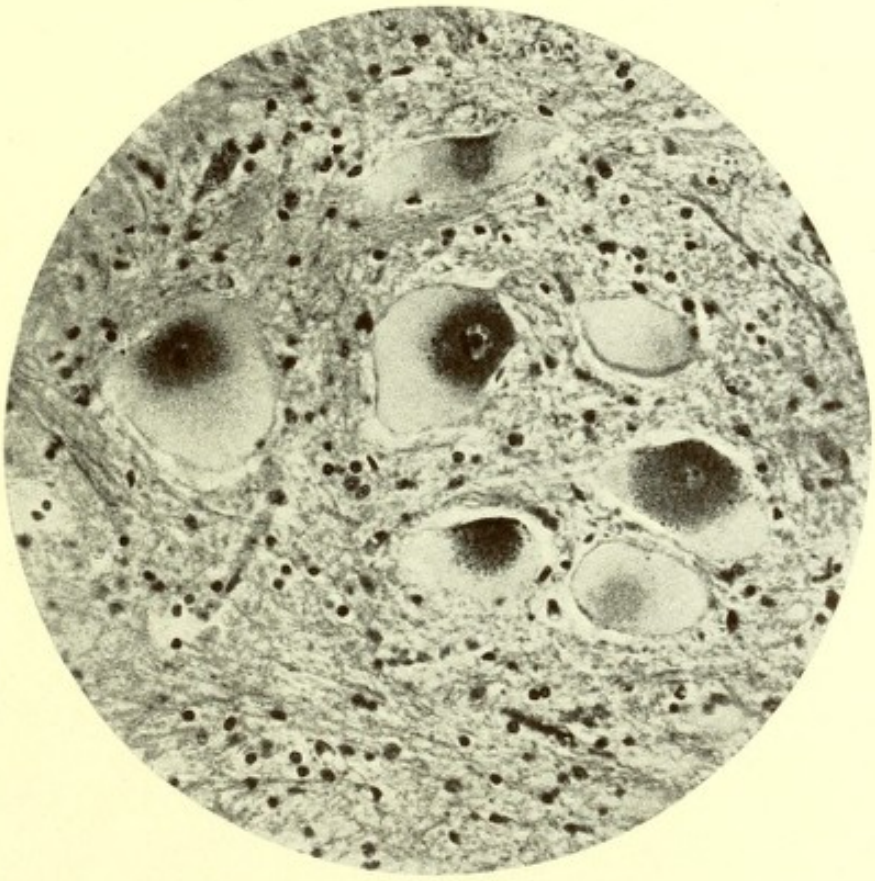
FIG. 1



Brain Cortex Cell.

Showing degenerating ganglion cells.

FIG. 2

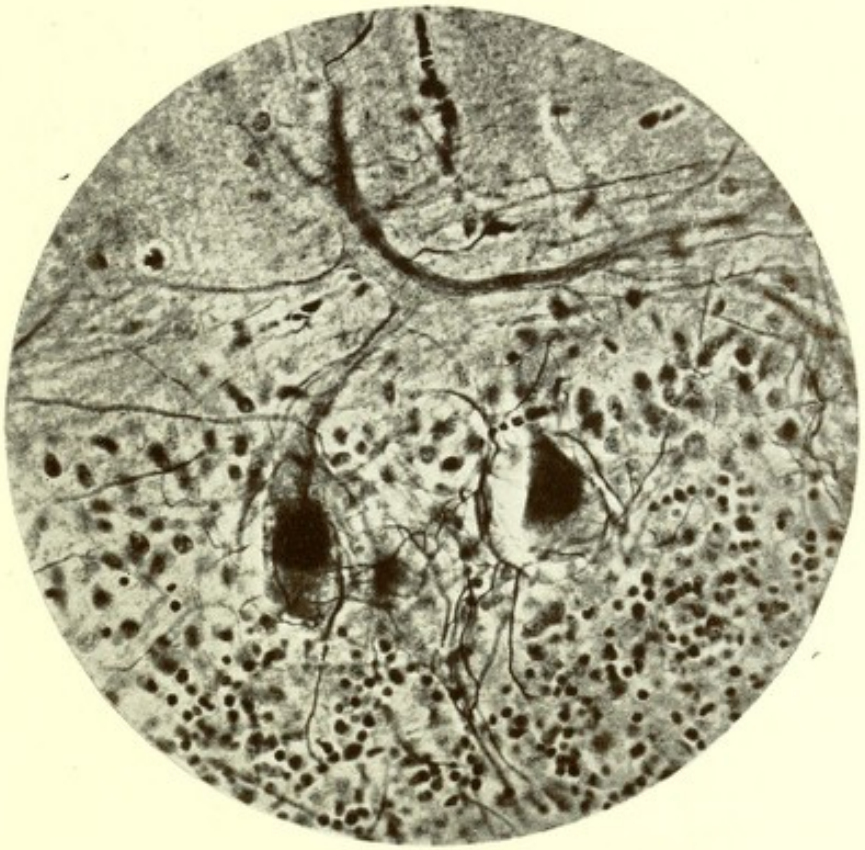


Ganglion Cells of the Anterior Horn of the Cord.

Showing the excentric nucleus surrounded by the remains of the chromatophilic substance and the marked increase in size [illegible]

# PLATE XXXVII

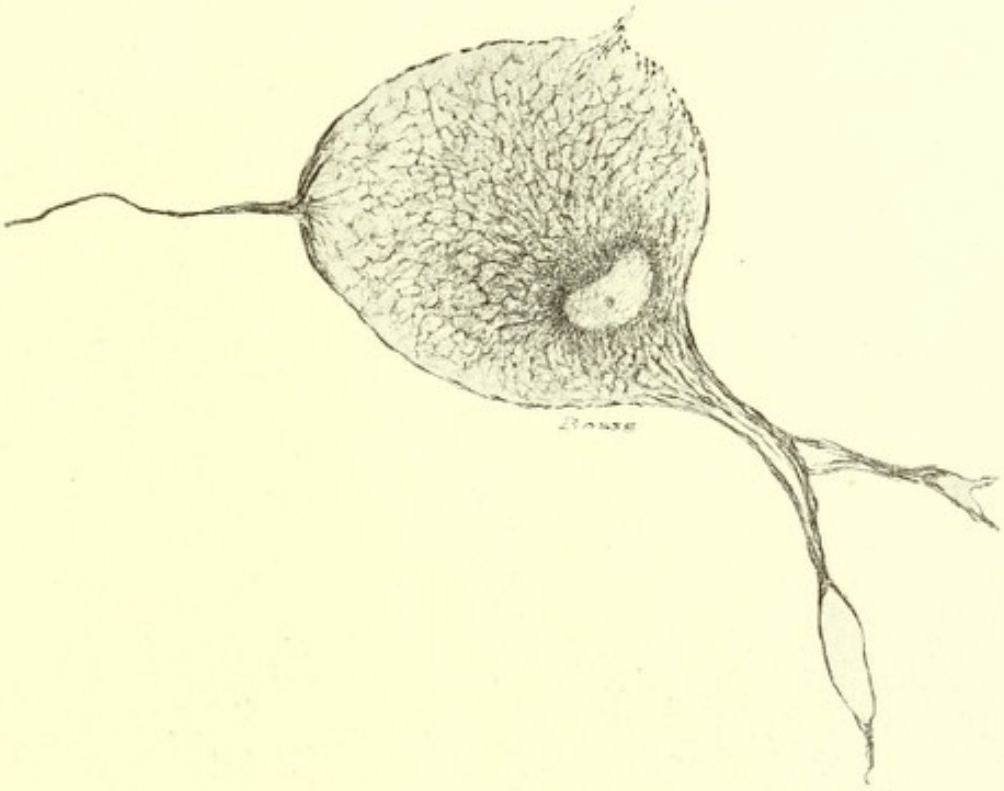
FIG. 1



Purkinje Cell.

Showing the swollen dendrites.

FIG. 2



Large Anterior Horn Cell.

Showing very well-preserved endocellular network and swelling of dendrites. The fibrils have commenced to disappear in the dendrites. (Bielschowsky.)

and nucleolus also disappear. A proliferation of the glia, particularly in the cortex, with the development of giant glia cells, has been noted. There is no evidence of any inflammatory process. Changes in the white fibers are unimportant and unessential.

It is Schaffer's merit to have laid stress upon the swelling of the dendrites, which appears to be so constant a condition as to be almost pathognomonic of the disease. The deterioration of the cell body and the swelling of these cell processes, exclusive of the axone, have been noted in all of Schaffer's and in my own recent case. This is not the place to discuss the bearing which these studies had upon a knowledge of the cell fibrils, but it need only be hinted that Schaffer has utilized the pathological appearance in this disease in his careful study of morbid cell structure. The disappearance of the fibrils in the dendrites and in the cell body is especially worthy of note.

Holden, as well as Schumway and Mary Buchanan, has made careful histological examinations of the eyes in amaurotic family idiocy. All these writers agree that the essential changes in the eyes are a degeneration of the ganglion cells of the retina and of the nerve fibers of the optic nerves and tracts, which are genetically a portion of the central nervous system. They also believe that the white area in the fundus is the result of the swollen and degenerated ganglion cells, which are present in much greater numbers in the macular region than elsewhere, and do not consider this white area to be due to œdema of the tissue as was first supposed.

**Pathogenesis and Theory of the Disease.**—Writers on amaurotic family idiocy have been divided into two groups. Some, among whom the writer wishes to class himself, have maintained that amaurotic family idiocy is, in a sense, a congenital disease, even if it is not manifest at the time of birth. The tendency to the disease is unquestionably born with the child. In the writer's opinion children afflicted with amaurotic family idiocy are possessed of a nervous system so inadequate to the demands imposed upon it that its cells, after having performed their function for a few weeks or months, undergo complete disintegration. In other words, these children have a very limited capacity for normal development. Their central nervous system is not equal to the functions that they are expected to perform for more than the first three or four months of life, and from that time on a rapid deterioration of all functions is established.

Even Schaffer, who was inclined for a time to class amaurotic family idiocy among acquired diseases, is bound to concede that the entire nervous system of such a child is so delicately organized that it is not able to repair the ordinary physiological waste of its cells. These delicately constructed nerve cells are so easily exhausted that they undergo a progressive deterioration, which leads to the death of the ganglion cell with a consecutive hyperplasia of the glia substance. These views are so thoroughly in accord with those of the writer that there seems no radical difference between them. Only a few authors now adhere to the view which was held by Hirsch, that the disease is altogether an acquired condition and that it is possibly due to some toxic agent. Hirsch believes that it might be due to some toxic substance contained in the mother's milk. From the very outset I was loath to accept the explanation of a toxic origin for any profound family disease, and in the case of these amaurotic children Hirsch's view falls to the ground because several of those seen by the writer were not

nursed by their own mothers, but by wet-nurses of a different race and different nationality. However, a child born with a defective nervous system may be more subject to toxic influences than otherwise normal children would be.

**Symptoms.**—The children afflicted with this disease have been born at full term and apparently in perfect health. Difficult labor or instrumental delivery has no bearing upon its development. The children do well during the first three to six months of life, when they become listless and apathetic, move their limbs very little, show no interest in their surroundings and show the first signs of visual disturbance which ultimately leads to blindness. As time goes on, the child is unable to hold up its head or to sit up. Its muscles are generally flaccid, rarely spastic. The reflexes may be normal, a trifle subnormal, or somewhat exaggerated, but marked spasticity, such as we find in the spastic diplegias of childhood, is not common. In some instances there is an unusual sensitiveness to touch and to sound (hyperacusis), the child being startled by the slightest noise. Convulsions occur in some cases, but are not a frequent or integral symptom. All the functions of the body are in a low state of activity. The children are subject to frequent bronchial attacks, and often show gastro-intestinal disturbances. An examination of the fundus reveals the peculiar condition described by Tay. The "cherry-red spot in the region of the macula lutea" has become a familiar phenomenon to those who are on the lookout for this disease. There is a gradual increase of all the symptoms, the mental defect becomes more and more noticeable, the palsy more extreme, complete blindness follows, and the child, from having been a plump, well-developed infant, lapses into a condition of marasmus in which it dies, as a rule, before the end of the second year.

To sum up briefly, the chief symptoms are: (1) A mental impairment during the first months of life leading to absolute idiocy. (2) Paresis or paralysis of the greater part of the body, which may be either flaccid or spastic. (3) The reflexes may be normal, deficient, or increased. (4) A diminution of vision terminating in absolute blindness (the cherry-red spot in the region of the macula lutea and later a simple optic nerve atrophy). (5) Marasmus and a fatal termination, as a rule, before the age of two years. (6) The occurrence of the affection in several members of the same family.

In some but not in all of the cases nystagmus, strabismus, hyperacusis, and convulsions may be added to the above cardinal symptoms A loss of the sense of hearing was noted in a few cases. Falkenheim referred to "explosive laughter," but the writer has not been able to corroborate this finding in any of his own cases. If we allow for slight variations in the symptoms, it must be said that there are few diseases of childhood or of adult life that are as uniform in their clinical manifestations as this. In a few cases, such as the one reported by Koller, the optic nerve changes have preceded the characteristic changes in the macula lutea, and in one of Higier's cases the optic nerve atrophy was much more marked than the changes in the macula lutea.

**Diagnosis.**—The clinical symptoms are so distinct that a physician who is acquainted with the disease can hardly fail to make the diagnosis. The direct recognition of the disease must depend upon the concurrence of the various symptoms. Thus, there are innumerable forms of brain defect that do not belong to this category. There are also disturbances of vision, often syphilitic and of congenital origin, that have nothing to do

with amaurotic family idiocy. But if a child that appears to have been well at birth loses its vision in the course of the first six months of life, if the child becomes listless, and if, on examination, the "cherry-red spot" can be found, there can be no doubt whatever about the character of the disease. Some difficulty will occasionally be experienced in differentiating between amaurotic family idiocy and the congenital *diplegias* and *paraplegias*, but the preservation of considerable intelligence and the presence of normal vision ought to be sufficient to prevent mistakes in diagnosis. Incidentally, it might be remarked that children suffering from amaurotic family idiocy may have either flaccid or spastic limbs. On the whole, the flaccid limbs are fully as common. It is necessary to insist upon this since the writer has found that others seem to consider spasticity a regular feature of the disease, which it surely is not. It is important in every suspected case to inquire into the character of the mother's labor at the time the child was born. Difficulties in labor have nothing to do with amaurotic family idiocy, but are commonly associated with congenital spastic palsies and with various forms of congenital idiocy and epilepsy. Convulsions may occur in amaurotic family idiocy, but do not constitute an integral symptom of the disease.

It is not always easy to distinguish *syphilitic* cerebral disease of the infant from amaurotic family idiocy, and if several members of the same family are afflicted, as they well might be in the case of an hereditary syphilitic disease, the difficulties become still greater, but if all the symptoms of the Tay-Sachs disease are borne in mind the question will finally be solved.

Amaurotic family idiocy as described by the present writer is to be differentiated from the *juvenile form* to which Spielmeyer[1] and Vogt[2] have called special attention. This form bears a close resemblance in many respects to the Tay-Sachs (infantile) type, but it occurs much later in life, often appearing not until the eighth, tenth, or twelfth year, is associated with blindness (without the changes in the macula), and leads to a fatal termination after a period of a few years. The writer has been struck by the resemblance between these two forms, and will not deny that they are akin to one another, without, however, being identical. This kinship must also be upheld in view of Schaffer's investigations, which reveal a great resemblance between the histological findings of the juvenile and the infantile forms of amaurotic family idiocy. Such differences in histological structure as do occur might be due to the fact that the juvenile form represents a less violent process, attacking a brain that had advanced farther in development than the infantile brain of typical amaurotic family idiocy. It is also astonishing that the juvenile form is not restricted to the Hebrew race, and that the infantile form invariably occurs among the Hebrews and among them only. While I would not deny that these two forms of disease are somewhat related to one another, I cannot for the time being consider them identical.

**Treatment.**—Under this heading there is, unfortunately, little to be said. As in the case of other family affections, we have no means of staying the disease. I have been asked a number of times, by the parents of these "amaurotic" children, whether the birth of normal children can be expected. In several of the families that I have seen, normal children have grown up

[1] *Neurol. Centralbl.*, 1906, xxv, 51; also *Monograph Gotha*, 1907.
[2] *Monatsschr. f. Psych. u. Neurol.*, 1906, xviii, 161.

side by side with the unfortunate victims of the disease. In such families it has not been restricted to one sex or the other. From my own experience I can only advise that the dangers of blood relationship be seriously considered; that marriage between persons closely related be discouraged; and marriages between persons in whose families there are marked neurotic or psychical taints are also undesirable. If the disease has made its appearance in the child, purely symptomatic treatment is the only kind that can be adopted in the case of the victims of this fatal family disease.

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