Familiar forms of nervous disease.

J. Bentley Squier Jr.
July 10th 1893.

FAMILIAR FORMS

OF

NERVOUS DISEASE

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BY

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WITH ILLUSTRATIONS, DIAGRAMS, AND CHARTS

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PREFACE.

ADVANCES in knowledge in the department of neurology have been of late more rapid than in any other branch of medicine. The result has been to render diagnosis in many affections, previously obscure, both more precise and less difficult; and to open to successful surgical treatment many diseases formerly considered fatal. It is the object of this volume to make available to the general practitioner some of the results of later investigations which have a direct and practical bearing upon the commoner forms of nervous disease. The facts which have been chiefly emphasized are those which enable one to make an accurate diagnosis of the nature and of the location of lesions in the central nervous system; for it is evident that such a diagnosis is the essential preliminary both to medical and to surgical treatment.

This work is not a treatise upon nervous diseases. It is a series of clinical studies of the more familiar types. The chief source of all medical knowledge is the careful study of groups of cases by those who have at their disposal a large supply of clinical material. The data which have been utilized in the preparation of these studies have been selected from American sources, chiefly from the Nervous Clinic of the College of Physicians and Surgeons. My assistants in that Clinic have joined heartily in the work of collecting and analyzing the material, and have prepared several of the most valuable chapters. I take pleasure in acknowledging my indebtedness

to Dr. Frederick Peterson, the Chief of Clinic; and to Dr. Walter Vought, Dr. Winslow W. Skinner, Dr. Edwin E. Swift, and Dr. M. L. Goodkind, the clinical assistants, for their co-operation.

The cases which are recorded have been carefully chosen from a large number because they present typical features and indicate the possibilities of surgical as well as of medical treatment. It is hoped that as a contribution to clinical neurology they may be useful and that these studies may stimulate similar work in other large clinics of this country.

M. ALLEN STARR.

NEW YORK, May 1st, 1890.

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CHAPTER I.

THE LOCALIZATION OF CEREBRAL FUNCTIONS.

The development of the doctrine of cerebral localization.—A statement of the facts now established.—The proofs of the doctrine derived from anatomy, embryology, pathology, experimental atrophy, comparative anatomy, and recent exact researches in physiology.—Its application to cerebral surgery.

THE theory of the localization of brain functions is now established. It is no longer a matter of doubt that each part of the organ has its own work to do. And already the practical application has followed, and cerebral surgery has its accepted place in therapeutics. The brain is no longer an organ exempt from surgical interference.

It becomes, therefore, a necessity for every physician to be in possession of those facts which are essential to the diagnosis of local cerebral disease; to be able to decide upon the possibility of surgical interference; to be able to direct the surgeon where to trephine.

The facts which must be known are not difficult to understand, and, when they are brought together, the conclusions which they warrant are evident at once. It is the collection of the facts which is necessary. But they are scattered through medical literature and are not easily accessible to those who are far from libraries. Their study is tedious, yet the conclusions to be reached are of the greatest importance.

In the present chapter, the progress of the discovery of the facts of localization, the various proofs of the doctrine, and

its chief data are presented for the purpose of bringing into general knowledge the results of the special work of neurologists during the past ten years.

Physiological experiment has led the way to much of this knowledge. But it is only in so far as it has been supplemented and confirmed by clinical and pathological observation on man, that the facts which are of practical importance can be utilized. Hence, greater attention is paid here to cases of disease than to the supposed effects of experiments upon animals. And illustrative cases of the various cortical lesions are presented, in order to demonstrate the possibility of diagnosis and of surgical treatment.

THE DEVELOPMENT OF THE DOCTRINE OF CEREBRAL LOCALIZATION.

While it was known in the first century of this era that each hemisphere of the brain is in functional relation with the opposite half of the body, the first facts which had a bearing upon the modern theory of the localization of the functions of the brain were not observed prior to 1822. At that time Thomas Hood, in England, and three years later Bouillaud, in France, recorded cases to show that disturbances of speech were produced by disease in the frontal lobes of the brain. In 1836, Marc Dax published a case of aphasia caused by a lesion in the left hemisphere: but this excited little notice until, in 1861, Broca attempted to locate more definitely the faculty of language. This he assigned to the left third frontal convolution, basing his assertion upon two cases of loss of speech, and supporting his position, two years later, by fifteen cases, in fourteen of which aphasia was due to disease in that region. The subsequent publication of numerous cases of aphasia with autopsies, and the discussion of the subject before the French Academy of Medicine in 1864, did much to stimulate the study of cerebral diseases, and to bring to notice the causal relation between certain nervous symptoms and certain brain lesions.

Up to that time the teachings of Flourens, that the brain

acts as a whole, its various parts not possessing different powers, had been widely accepted. But the facts regarding aphasia threw doubt on these teachings; and when, between 1862 and 1867, Hughlings Jackson and Meynert advanced the opinion that the anterior part of the brain only was concerned in motor acts, the one reaching this conclusion from the study of localized spasms, the other from the observation of the lesions of general paresis, the authority of the French physiologist was seriously undermined. It was evident that the physiological experiments of Flourens needed confirmation; and in 1869 and 1870, Fritsch and Hitzig, of Berlin, undertook their verification. From the beginning of their experiments, the modern doctrine of the localizaton of functions may be said to date; for by those experiments it was proven that in animals the anterior portion of each hemisphere is motor, its irritation by electricity causing co-ordinated movements, and its removal causing paralysis in the limbs of the opposite side. These conclusions were confirmed by Ferrier, in 1873, who enlarged them greatly by his experiments: for he showed that the posterior portions of the brain are sensory and distinguished between various sensory areas assigning each sense to a cortical region.

It is not necessary to enter here upon the discussions between different physiologists to which their discoveries gave rise. Nothnagel (1877), Munk (1881), Luciani and Seppilli (1884), Ferrier (1887), François Franck (1887), Beevor, Horsley, and Schäfer (1888), and Thompson and Brown (1889) have published conclusions based upon experiments upon different animals, with somewhat varying results as regards the exact localization of the different functions. But whatever minor differences may be noticed, all agree upon the principle that the functions of the brain are localizable. Even the strongest and almost sole remaining opponents of the theory, Goltz, of Strassburg, and Brown-Séquard, of Paris, admit that the results of destruction of various parts of the brain are different. On all sides, therefore, the doctrine of Flourens has been discarded and the modern view has prevailed.

The physiological discussions have, from the first, stimu-

lated clinical and pathological observations. For it was at once evident that in the case of man it is only by the careful collation of symptoms and lesions that conclusions regarding cerebral localization can be reached. From 1864 until the present time, the publication of more or less careful records of cases of cerebral disease in the medical journals of every country has gone on. From time to time, these cases have been collected and analyzed; special symptoms being brought into relation with limited lesions, and general conclusions being reached from the numerous data. From time to time, previous conclusions have had to be modified, finer distinctions between local functions have appeared to be warranted, and obscure phenomena have been cleared up by the observation of some rare case, or by the insight of some gifted observer.

The work of Charcot, Pitres, Boyer, Brissaud, Féré, and Bernard, in France; of Ferrier, Ross, Bastian, and Gowers, in England; of Nothnagel, Exner, Wernicke, Benedikt, Petrina, Vetter, Wilbrand, Lichtheim, and others in Germany; of Luciani and Seppilli in Italy; of Seguin, Mills, Dana, and the author in America, warrants mention in any historical summary; though to these names others might well be added. For by the careful collection and comparison of individual cases—a work which requires much laborious research and much critical insight—in pursuance of a strictly inductive method of investigation, these observers have reached well-founded conclusions regarding the application of the doctrine of cerebral localization to man.

THE FACTS OF LOCALIZATION NOW ESTABLISHED.

It has, by this method, been fully established that various powers of perception, memory, and volition can be definitely assigned to various parts of the brain; that each sense has its corresponding area in the cortex and each voluntary movement its point of departure from the cortex.

It has been proven that perceptions leave behind them a physical trace inseparably connected with the area in which

they were originally received; that irritation of this area produces a renewal in consciousness of the original perception as a memory, which may be so vivid as to appear real, being then a hallucination; that destruction of this area causes a loss of the power of recalling the previous perception or of recognizing it when repeated.

It has been proven that movements which are acquired by practice, and are therefore the result of conscious effort, are always initiated from a definite area; that this area is inseparably related to movements; since its irritation, by any cause, results in the production of forcible movements of the nature of spasms, and its destruction produces a loss of the power of movement or paralysis of voluntary action.

It has been proven that the use of language involves both the recollection of auditory and visual symbols and the initiation of impulses of speech and writing, thus calling into play not only the motor portion of the brain, destruction of which therefore causes a loss of speech, as Broca held, but also the sensory portion of the brain, a lesion of which will also cause aphasia, as the opponents of Broca clearly proved by the cases which they cited in opposition to his theory. The progress of the investigation, therefore, has helped to reconcile facts supposed to be incompatible, while it has added definiteness to diagnosis.

THE VARIOUS PROOFS OF THE DOCTRINE.

While thus from the investigations of clinical observers and of pathologists facts have been accumulating which place upon a sure and reliable basis the doctrine of cerebral localization as relating to man, in other lines of research further evidence, in favor of this doctrine, has been elicited.

The study of brain anatomy, as well as that of physiology, has been revolutionized during the past twenty years. By the introduction of new methods of research, the complex structure of the cerebral hemisphere has been gradually brought to light. In the adult brain, as it is seen when removed from the body, the structure of the white matter lying beneath the cor-

tex is not suggestive of its functions. But the microscopic study of this portion of the brain has shown it to be made up of nerve fibres passing in various directions, and the admitted function of such fibres is the transmission of nervous impulses. In the healthy adult brain it is impossible to separate these fibres from one another or to detect the existence of bands of fibres functionally related and running together in tracts. But, in 1877, Flechsig made the important discovery that such tracts could be differentiated from each other in fœtal life, by the fact that fibres which were functionally related developed simultaneously and in the definite direction in which they were destined to transmit impulses; each tract having its own time and direction of development. It thus became possible to separate different tracts from one another and to trace their exact course and destination; and in the hands of Flechsig, Bechterew, and Edinger this work has yielded a brilliant confirmation of the doctrine of localization.

It had already been observed by Türck, in the study of the spinal cord, that as a result of transverse lesions, certain columns degenerated downward and others upward. The researches of Flechsig showed that these separate columns, distinguished from one another by the degenerative method of Türck, corresponded in situation to the separate tracts which developed at different times in the fœtus. Thus the developmental and degenerative methods combined to show the existence of separate tracts in the white matter of the spinal cord. It was but a step to apply these methods to the examination of the brain: and, in 1878, Brissaud showed that after certain limited brain lesions, certain tracts in the white matter showed evidences of degeneration. These tracts coincided, in situation, with the functional tracts simultaneously differentiated by the method of Flechsig, which thus received an important verification.

The differentiation of tracts through the white matter was also aided by the application of the atrophy method of von Gudden to the investigation of brain anatomy. Von Gudden had discovered, in 1870, that when an organ, like the eye, is extirpated from an animal at birth, the nerve connected with

the organ fails to develop, and hence, by its atrophic appearance in the grown animal, can be distinguished from the corresponding nerve of the opposite side, and the course of each can thus be traced. Von Gudden also investigated the results of natural atrophies from deficient development of certain parts in idiots. It remained for his pupils to apply the method fully to the tracing of intra-cerebral tracts. This was done by von Monakow (1884 and 1889) in some experiments which resulted in tracing the visual tract and showing its connections; and it has been applied by Babinsky (1886), by Spitzka (1886), and by Forel (1889) to the tracing of the auditory tract. Tracts thus traced are found to correspond to those whose situation had already been determined by the other methods mentioned. And hence *the harmony in the results of these totally different methods of investigation carries conviction that the doctrine of localization is absolutely true.*

It is found that areas of the brain whose function is established by the clinico-pathological method, may be shown by these anatomical methods to be joined to the organs with which they are functionally related. Hence the results of these parallel lines of research harmonize.

And further, the results of a series of observations made by Gegenbaur, Spitzka, Osborn, Zuckerkandl, Hervé, and Edinger upon the comparative anatomy of the brain have already furnished an independent mass of testimony regarding the localization of function. This last-named method is as yet in its beginning; but it has produced the evidence needed regarding the course of the auditory tract in the brain axis (Spitzka, 1886), and has confirmed the hypothesis of the location of the olfactory sense (Zuckerkandl, 1887) and of the motor region concerned in speech (Hervé, 1888). It may be said to be the correlative of the atrophy method of von Gudden. For in that the loss of an organ was associated with a deficient development of its related cerebral parts; while in this the excessive development of certain parts is associated with an extraordinary power and use of some special organ. And in the future this line of research will doubtless furnish a mass of facts of the greatest value.

Lastly, it may be mentioned, that exact methods of electrical measurements have been applied to the solution of questions of localization by Horsley, Beevor, and Gotch (1888). When an electric current is applied at one end of a nerve tract its arrival at the other end can be detected and the time of its passage can be measured. If its passage is direct, the time will be short; if indirect, longer. It is thus found that an electric message sent from the motor area of the brain travels in the motor tract and never spreads to other tracts; that the message starting from the motor area of the arm does not pass below the upper dorsal region of the cord; while a message from the motor area of the leg reaches the lumbar part of the cord; a valuable confirmation, therefore, of the respective locations in the cortex of the areas for arm and leg. Again, it had been found that electric excitation of various areas of the cortex might produce the same movement; thus, the application of currents to two widely separated regions gave rise to a turning of both eyes to one side; and it was impossible to determine in which of these areas the motor centre for conjugate movements of the eyes was located. The investigation of the time elapsing between the irritation and the movement has shown that after irritation in the anterior of these two regions the reaction-time was less than that after irritation of the posterior region. Hence it is argued that the actual motor impulse starts from the anterior region; and that when it is the sensation which induces the movement, as by the attraction of a light both eyes turn to the side, the impulse starts from the posterior region, but must go first to the anterior region before it passes to the oculo-motor centres. Thus the more exact methods of research in physiology are being used to confirm or to correct the earlier statements and to finally determine disputed questions.

It is therefore evident, from the summary of the results reached by different independent methods of investigation, that cerebral localization is no longer a tentative theory. It is an established fact.

As long ago as 1876, the practical bearings of the doctrine

of localization were appreciated by the early investigators, especially by Ferrier. It was even then evident that if disease in the brain could be exactly located during life, and was of a nature to be removed by surgical means, diagnosis might lead the way to therapeutics. It has only been within the last five years, and chiefly in America and England, that this great result has been attained; and even now the application of the doctrine to the development of cerebral surgery meets with hesitating acceptance upon the Continent of Europe. Caution cannot be excessive, and there are indeed still but few parts of the brain which can be said to be open to surgical treatment. But the continuous progress of discovery in the past, and the actual success of well-conducted aseptic surgical interference in appropriate cases, warrants the expectation of future results in the domain of cerebral surgery, which will rival in brilliancy those of any other department of that most practical art.

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CHAPTER II.

THE FUNCTIONS OF THE CEREBRAL CORTEX.

The general functions of the brain cortex; perception; association of perceptions into mental images; memory; expression; self-control. —The special functions of the brain cortex; criteria of evidence; general vs. local symptoms.—The brain fissures and convolutions; the cortical areas.—Cranio-cerebral topography.

THERE is a unanimity of belief among physiologists and psychologists that all conscious mental activity has its physical basis in the cerebral cortex. Reflex and automatic acts may be performed in response to sensory impulses by the agency of the masses of gray matter collected in the spinal cord, in the cerebral axis, or in the basal ganglia; and some automatic action, such as acts of somnambulism and in a state of hypnotism, may possibly be cortical yet unconscious. But conscious perceptions of sensation and conscious efforts require for their performance the existence and activity of the gray matter spread out in a thin layer upon the surface of the cerebral hemispheres. This is proven not only by the facts that the degree of development of the cortex in extent, thickness, and condition of nutrition and blood supply varies directly as the mental powers of the animal, as the capacity of the race, and as the mental status of the individual; but also by the facts that its deficient development leads to idiocy, and its actual disease produces mental as well as physical symptoms.

GENERAL FUNCTIONS OF THE CORTEX.

The general functions of the cortex of the brain may be enumerated as follows:

(1) It receives impressions from the various sensory organs of the body as conscious perceptions.

It is true that every impression is not consciously perceived, as is shown by the familiar fact of one's reading down a page without knowing what has been read. Yet all impressions are received and semi-consciously preserved, as is proven by the fact that one may become conscious that the clock has struck and be able to recall the hour, even though the striking has been apparently unnoticed. Impressions which are consciously perceived come in through different senses and hence reach different parts of the cortex, since each organ is joined to its own area by a distinct tract. Hence the conscious perception of impressions may be arrested by cortical disease, and the location of the disease will determine which set of perceptions is cut off.

But impressions are not exclusively sensory. They may be motor. For motion is performed reflexly at first, is then perceived consciously as motion, is remembered, and finally imitated by effort. The impression of a motion made, acquired through muscular sense, is therefore one of the conscious perceptions of the cortex; and hence it too may be obliterated by disease. It must be held that motor memories lie at the basis of all adaptive movement. These are acquired by observation and practice. The combination of known efforts into new adaptations, like the combination of various perceptions into a single concept, or the combination of memories into imaginative fancies, is a power of the cortex dependent upon but additional to its power of conscious perception.

(2) The association of various perceptions into mental images is also a function of the cortex. Perceptions rarely come singly. As one looks at a rose one perceives its delicate color and its fragrance, one handles it and gets the sensation of its soft, velvety leaves and thorny stem. These various perceptions are associated together into a mental image of the rose, which is a thing of parts and not a simple perception. The various perceptions occur in various regions of the cortex, and these regions are joined with one another both by a fine network of fibres within the cortex and by bands of association fibres running beneath the cortex. It is by means of these fibres that various cells, whose function it is to receive

the impressions, are joined together. Hence, the physical basis of the mental image of an object is a complex set of cortical cells joined together by fibres.

(3) This physical basis of each mental image remains after the object ceases to be perceived. It is, therefore, the function of the cortex to retain perceptions as memory pictures so that consciousness may recall them and recognize them when they appear again. Such recollection may occur of only one perception, but this is likely to lead to the recollection of the entire mental image, or of other images associated with the first. Disease of the cortex will, therefore, impair the powers of recognition and of recollection either by depriving the mental image of some of its parts, or by interfering with the process of association which is necessary to its completeness.

(4) It is also a function of the cortex to give expression to thought in action and in speech. Thought may be considered as the play of consciousness among the mental images along the lines of association, in part voluntary, in part automatic. Such thought leads eventually to expression either in sign language, in speech, or in action. Each of these is at first imitative but eventually voluntary; and each is directed by its appropriate cortical area. If these areas are diseased, arrest of the powers of expression ensues, as in all cases of paralysis.

(5) But thought does not lead immediately to expression in all cases. Expression may be restrained; the impulse may be arrested. This restraint of the flow of thought outward in expression has been termed inhibition; and inhibition, or the act of control, is the highest of all cortical functions.

In this summary of cortical action, it will be noticed that no mention has been made of the higher mental acts, of judgment, reason, imagination, or of those qualities which determine talent and character. These cannot, as yet, be assigned to any particular region of the cortex, and no physical basis, no mechanism for such purely mental acts can, as yet, be pictured to the mind. As Hughlings Jackson has well said, "Psychical states are not functions of any centre but are simply concomitant with functioning of the most complex nervous arrangements." It is certainly true that all mental activ-

ity has a physical basis in the brain; but there are numerous problems regarding the mutual relation of thought and cerebral action upon which physiology has thrown no light.

SPECIAL FUNCTIONS OF SPECIAL AREAS OF THE CORTEX.

It is possible, from the evidence of clinical cases and of pathological examination, to assign *special functions* to different parts of the cortex. These are motion, tactile, temperature, pain and muscular sensations, sight, hearing, smell and taste, and the use of language.

But, in reviewing this evidence, it seems necessary to consider critically its value; and to exclude all questionable facts, so as to make conclusions reliable.

According to Nothnagel,[1] those cases only are of use in determining the function of limited areas of the cortex in which there is a single lesion of small extent, accurately located and of considerable duration. If lesions are multiple, it is impossible to assign different symptoms to each. If the lesion is of large extent, the functions of two or more areas involved cannot be distinguished. If the patient dies soon after the invasion of the disease, especially if that disease be a cerebral hemorrhage, it is impossible to separate temporary symptoms due to shock, from permanent symptoms due to the destruction of brain tissue. If, however, the function of limited areas is fairly determined by such carefully-weighed evidence, corroborative proof furnished by multiple and extensive lesions may be admitted.

Cases of brain disease afford two distinct kinds of information regarding the function of cortical areas which have been termed by Exner[2] positive and negative, and each is of value. The positive evidence is the symptom produced by the lesion of a definite area. The negative evidence is the lack of symptoms referable to other organs. Thus if a lesion in one region produces uniformly a special form of blindness but never causes any disturbance of motion, it is allowable to conclude positively that this region has a functional relation to sight;

[1] "Topische Diagnostik der Gehirnkrankheiten," p. 6.

[2] "Localisation der Functionen in der Grosshirnrinde," p. 4.

and, negatively, that it has no functional relation to motion. Each of these lines of evidence is important.

In the study of cases of cortical disease, it is necessary at the outset to distinguish general from local symptoms, for it is the latter only which enable one to locate the lesion. *General Symptoms* are those which are common to various kinds of brain disease in whatever region they are located. They are headache, vertigo, digestive disturbances, general convulsions, optic neuritis, with or without blindness, delirium, and coma. These are due either to an increase of the intra-cranial contents (as by the growth of a tumor) and consequent pressure, or to some other interference with the normal condition in the brain. They give no evidence as to the exact region of the brain which is affected. They merely indicate that the brain is diseased.

Local Symptoms, on the other hand, depend entirely for their production upon the region of the cortex which is invaded. They are disturbances of motion and of sensation of various kinds, and of sensory perception; disturbances of memory; and loss of speech. They may be divided into symptoms of irritation and symptoms of destruction; and it is not infrequently the case that the former precede the latter in the course of a disease. An irritative lesion produces symptoms due to an increased activity of the area affected; *e.g.*, spasms, pain, tingling and numbness, flashes of light, sounds, or hallucinations of smell and taste. A destructive lesion produces symptoms due to a loss of function in the area involved; *e.g.*, paralysis, anæsthesia, blindness, deafness, loss of smell and taste and of the use of language. Each of these points to a different location of the disease, and is essential to its localization.

THE BRAIN FISSURES AND CONVOLUTIONS.

Before proceeding to study the special functions of different parts of the cortex in detail, and the symptoms of their diseases, a brief recapitulation of the fissures and convolutions may be given for reference.

Brains differ widely from one another in their appearance on the surface, great variety being the rule. Even the two hemispheres rarely resemble each other exactly either in the arrangement of fissures or of convolutions. Hence no one figure or diagram is satisfactory. It is necessary to establish certain landmarks on the brain surface, and this may be done by means of the chief fissures. The convolutions which are separated by these fissures may be small or large, or may

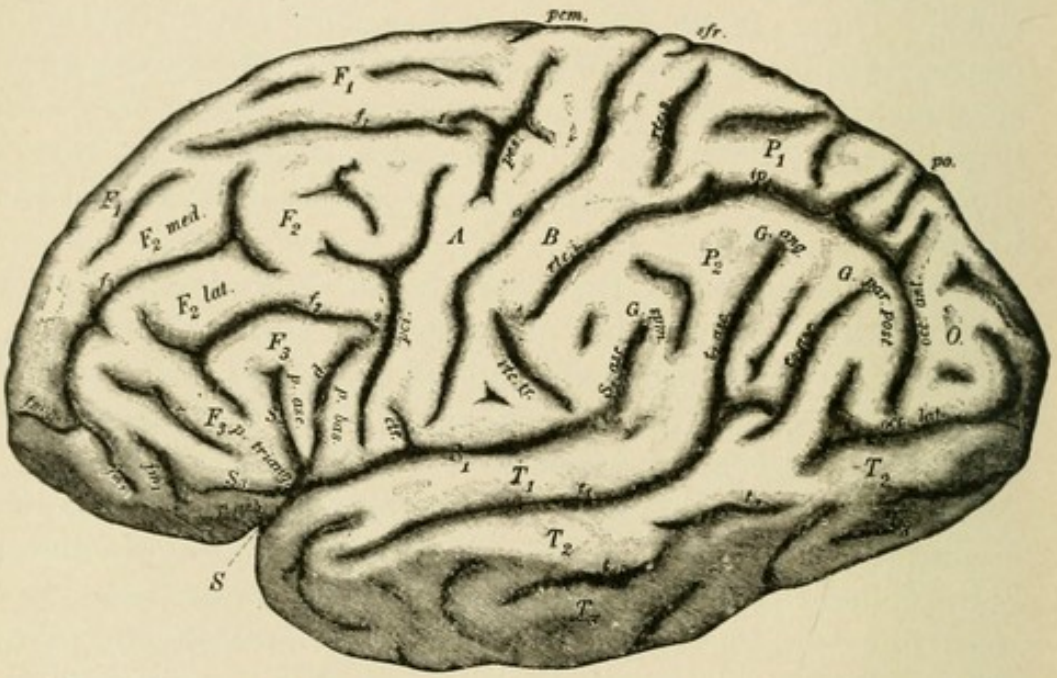

FIG. 1.—The Lateral Surface of the Left Hemisphere. (Eberstaller.) *S*, fissure of Sylvius; *c*, fissure of Rolando, or central fissure; *pci*, sulcus præ-centralis inferior; *pcs*, sulcus præ-centralis superior; *rtc*, sulcus retro-centralis; *sfr*, sulcus sub-frontalis; f_1, f_2, f_3, sulcus frontalis, sup., inf. et medius; *ip*, interparietal fissure; *po*, parieto-occipital fissure; *occ. ant.*, *occ. lat.*, sulcus occipitalis, ant. et lat.; t_1, t_2, t_3, sulci temporales; *A*, *B*, ant. and post. central convolutions; F_1, F_2, F_3, Frontal convolutions, sup., med., infer.; P_1, P_2, parietal convolutions, super. and infer.; *G. spm*, gyrus supra-marginalis; *G. ang.*, gyrus angularis; *O*, occipital lobe; T_1, T_2, T_3, temporal convolutions.

even be reduplicated, presenting by the indentation of very small fissures, the appearance of two or even more irregular convolutions. Yet, however complex the folding, it is, in every case, possible to distinguish the chief primary fissures easily; and this being done, the convolutions can be assigned to their diagrammatic equivalent and named.

The figure (Fig. 1) is drawn from a photograph of the brain by Eberstaller.

The diagrams (Figs. 2 and 3) are those of Ecker—which are widely known. The prominent features of the convexity

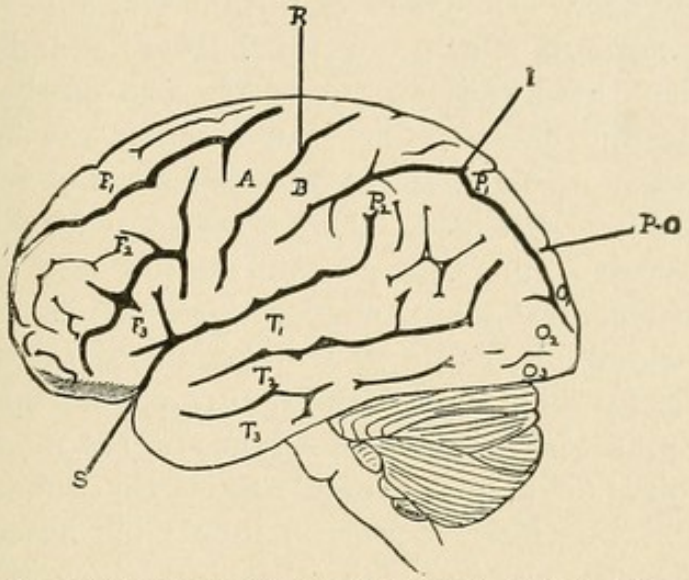

FIG. 2.—Fissures and Convolutions of the Left Hemisphere. (Ecker.) *S*, fissure of Sylvius; *R*, fissure of Rolando; *I*, Interparietal fissure; *Po*, parieto-occipital fissure; *F*1, *F*2, *F*3, frontal convolutions; *A*, *B*, ant. and post. central convolutions; *O*1, *O*2, *O*3, occipital convolutions; *T*1, *T*2, *T*3, temporal convolutions.

are the fissure of Sylvius (Figs. 1 and 2, S); the fissure of Rolando (Figs. 1, *c*; 2, R); the interparietal fissure (Figs. 1 and 2, I); and the parieto-occipital fissure (Figs. 1 and 2, P-O), which is seen on the median surface to run downward to join the calcarine fissure (Fig. 3, C). On the median surface the calloso-marginal fissure is the most prominent (Fig. 3, M) and the occipito temporal (Fig. 3, T-O) is also deep. Secondary and tertiary fissures may be distinguished from these primary fissures by their shortness and shallowness.

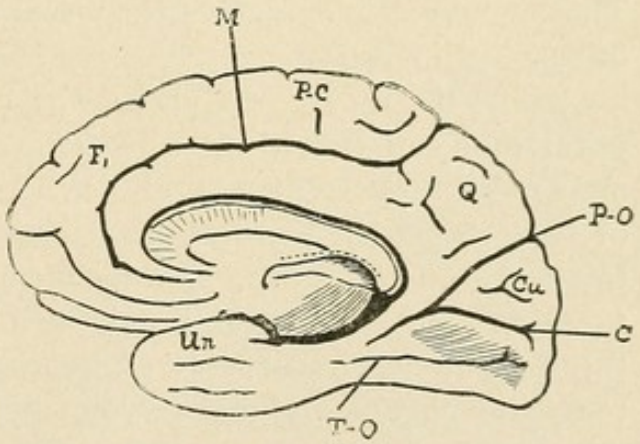

FIG. 3.—The Fissures and Convolutions of the Brain. The median surface of the right hemisphere. *M*, Calloso-marginal fissure; *P-O*, parieto-occipital fissure; *C*, calcarine fissure *T-O*, temporo-occipital fissure (inferior); *F*, frontal convolution; *P-C*, paracentral lobule; *Q*, quadrate lobule; *Cu*, cuneus; *Un*, uncinate convolution.

The convolutions of the convexity are divided into five groups. The frontal convolutions, whose general direction is from before backward, are three in number: superior or first, middle or second, inferior or third (Figs. 1 and 2, F_1, F_2, F_3). Behind these and lying on each side of the fissure of Rolando are the anterior and posterior central convolutions (Figs. 1 and 2, A, B), which join together at the vertex in the para-central lobule (Fig. 3, P-C). These are of the chief importance surgically, as they contain the motor centres. Posterior to the central motor convolutions, lie the superior and inferior parietal lobules (Figs. 1 and 2, P_1, P_2), separated from one another by the inter-parietal fissure. The superior parietal lobule presents a square appearance on the median surface of the brain, and hence is there named the quadrate lobule (Fig. 3, Q). The inferior parietal lobule has numerous short convolutions, the chief of which lies just above the fissure of Sylvius and is known as the supra-marginal convolution. The angular gyrus is at its posterior limit. At the posterior extremity of the hemisphere lie the short occipital convolutions (Figs. 1 and 2, O_1, O_2, O_3); and on the median surface, the cuneus (Fig. 3, Cu), which is a part of this region, is seen. These convolutions receive the visual impressions and are also of surgical interest.

Below the fissure of Sylvius, the convolutions of the temporal lobe run from before backward, being numbered from above downward (Figs. 1 and 2, T_1, T_2, T_3). This region is of importance in the left hemisphere, since in the first two temporal convolutions, speech memories are located. Its function on the right side is unknown, though somewhere near this region lie the centres of hearing. In the apex of the temporo-sphenoidal lobe and on its median surface are the convolutions which receive impressions of smell and taste (Fig. 3, Un). There are several convolutions lying on the under surface of the hemisphere and passing from the occipital to the temporal region whose function is not known. And the small convolutions of the basal part of the frontal lobe which lie on the orbital plate, are also unimportant physiologically in view of our present knowledge.

It must be distinctly understood that the cerebral fissures are not real boundaries of cortical areas, since the gray cortical layer is not cut through anywhere by a fissure, but is merely thrown into folds by its existence. In referring, therefore, to functional areas, it is not possible to refer to different convolutions as synonymous with these areas. Thus the frontal convolutions make up portions of three distinct functional areas: the frontal, the motor, and the speech areas. While, therefore, precision of description of lesions is attained by the naming of convolutions, the facts of localization prove that functional areas are not bounded by cerebral fissures, or limited to particular convolutions.

In considering diseases of the cortex, therefore, the functional areas are considered successively, rather than the anatomical lobes of the brain.

CRANIO-CEREBRAL TOPOGRAPHY.

The fact that the brain may be exposed for the removal of diseased parts in appropriate cases has made it necessary to ascertain the relation of its different fissures and convolutions to the cranial sutures, or to certain landmarks upon the surface of the head. These relations are well shown in the figure (Fig. 4), which is a photograph of a cast of a head made immediately after death by Dr. Cunningham, of Dublin.[1] Numerous rules have been laid down for the determination of the location of various parts of the convex surface of the hemisphere upon the head. The most important are the following, which may be compared with the diagrams of Reid (Fig. 5). To find the fissure of Rolando, lay down a line from the root of the nose to the occipital protuberance over the top of the head, and take a point 0.557 of the distance back upon this line. This point will correspond to the upper end of the fissure. The fissure makes an angle of 67° with the median line just measured. Hence if two strips of metal fixed to one another at

[1] See Dublin Journ. Med. Sci., 1888, p. 157. For an opportunity of photographing this cast I am indebted to Dr. F. Ferguson, Curator of the Museum of the New York Hospital.

this angle be placed on the head with their junction upon the upper end of the fissure, when one strip is on the median line, the other strip, pointing forward and downward, must lie over the fissure of Rolando. In its lower third the fissure becomes a little more vertical than the strip. The fissure is about

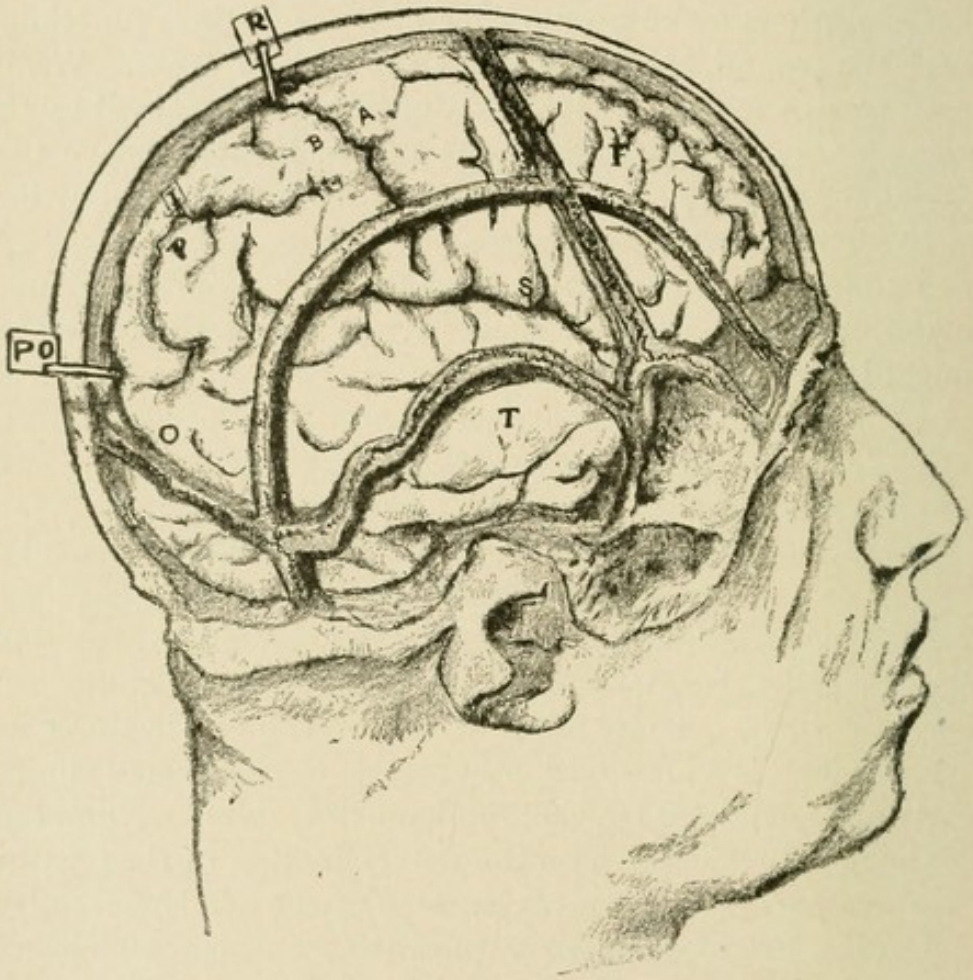

FIG. 4.—Photograph of a Cast of the Head showing Relation of Cranial Sutures to the Cerebral Fissures and Convolutions. *F*, Frontal; *P*, parietal; *O*, occipital; *T*, temporal lobes; *S*, fissure of Sylvius; *R*, fissure of Rolando; *I*, interparietal fissure; *P*,*O*, parieto-occipital fissure; *A*,*B* ant. and post. central convolutions.

three and a half inches long. To find the fissure of Sylvius, lay down a base line from the lower margin of the orbit to the auditory meatus. Lay down a second line parallel to the base line from the external angular process of the frontal bone backward one inch and a quarter; this gives point one. Find the most prominent part of the parietal eminence and

from it draw a line downward perpendicular to the base line, and on this take a point three-quarters of an inch below the eminence; this gives point two. Join these two points and the line will lie over the fissure of Sylvius. The anterior limb of the fissure will be two inches behind the external angular process. The fissure of Sylvius is about four inches long. To find the parieto-occipital fissure, continue the line of the fissure of Sylvius to the median line. At their junction lies this fissure. Since all areas now open to surgical operation can

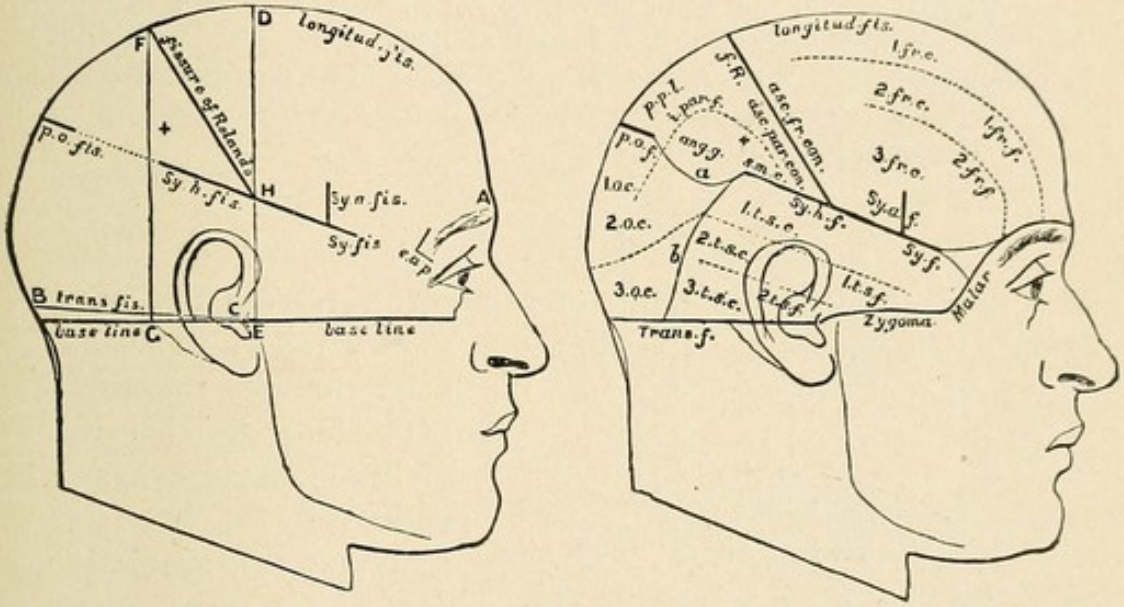

FIG. 5.—Guiding lines Reid and Relations of the Lines to the Fissures, (Reid.) (See text for explanation.

be located with a definite relation to these three fissures, no further rules are necessary. Since in opening the skull it is customary to make a fenestrum of at least an inch in diameter, and it is frequently necessary to enlarge the opening much more, a procedure in no way dangerous under aseptic conditions, there is no difficulty in recognizing the fissures and convolutions exposed if the rules are closely followed. Prior to the large incision of the scalp it is well to mark certain points upon the skull by the sharp point of a scalpel, so that when the bone is laid bare surface landmarks may still be kept in view.

CHAPTER III.

THE MOTOR AREA AND ITS AFFECTIONS.

The limits of the motor area. The functions of the motor area.—Jacksonian epilepsy or localized spasms; symptoms and cases.—Case I., Brachial spasm and aphasia.—Case II., Brachial and facial spasm with aphasia.—Case III., Brachial monospasm.—Case IV., Crural monospasm.—Case V., Crural monospasm.—The diagnosis of the nature of the disease and of its location.—Surgical treatment of Jacksonian epilepsy.

THE MOTOR AREA.

THE situation and limits of the motor area are now accurately determined and are shown in Figs. 6 and 7. This has been done by the study of two independent series of phenomena — spasm and paralysis.

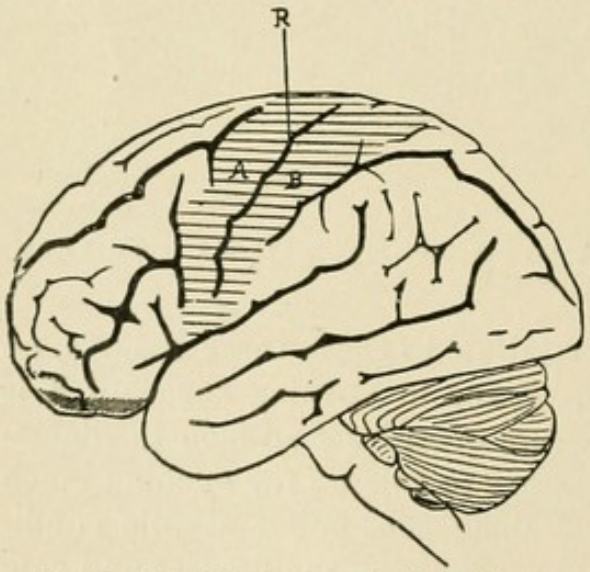

FIG. 6.—The Motor Area on the Lateral Surface of the Hemisphere. This includes the anterior and posterior central convolutions (*A*, *B*) on each side of the fissure of Rolando (R).

Electrical stimulation of the cortex in monkeys, within certain limits, produces spasmodic contraction of groups of muscles. If the stimulation be very weak, the resulting movement is limited in extent. If the stimulation be increased or kept up, the movement becomes greater. Thus weak stimulation of a given spot will cause the hand to close. Increase the stimulus and the wrist and arm will be flexed. Increase it again or continue it for some time, and the entire limb will be moved and even face and leg may be-

come tense and give evidence of slight motion. It is thus established that each cortical district presides over and initiates some movement, so that definite movements can be assigned to different areas within the motor region.

Very precise statements now appear to be warranted, based upon the recent physiological experiments of Horsley,[1] Schäfer, and Beevor and on the careful clinical studies of cortical spasms by Rolland.[2]

If we study actions as they are performed, we find that every act involves a succession of movements by different joints, each movement being produced by a number of motions in different muscles. The act of carrying a glass of water to the mouth, may involve perhaps the same muscles as the act of lifting a heavy vase from a high shelf and placing it on the floor; but there is a great difference between the order and sequence of movements in the two cases and the relative force of these movements.

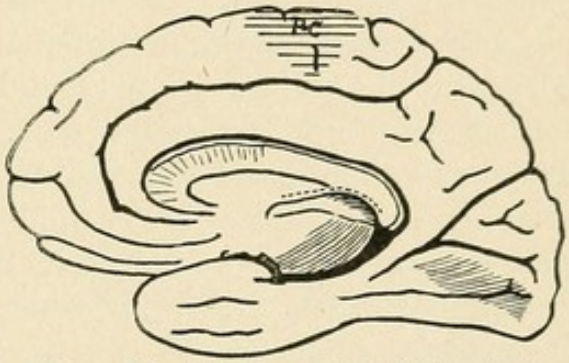

FIG. 7.—The Motor Area on the Median Surface. This includes the paracental lobule (*PC*) into which the central convolutions run.

In the first, the delicate balancing of the glass by the fingers and thumb is the most noticeable: in the second, it is the firm action of the arm and shoulder which is necessary to support the weight, and the act is aided by balancing movements of the entire body and legs to maintain the equilibrium. In both acts the entire muscular system of the upper extremity is involved, but in each there are some muscles whose action is greater in degree and precedent in time than in the other. It seems necessary, in view of recent researches, to adopt the view that the motor centres of the cortex govern motor acts rather than special muscles; that it is the act of grasping, or of opening the hand, or of separating the fingers, or of pointing at an object, which is produced by

[1] Philosophical Transactions, 1888, B. 20, B. 28.

[2] "L'Epilepsie Jacksonienne," Paris, 1888.

cortical action, rather than the contraction of the flexors or of the extensors as separate muscles of the hand. When any act is carefully examined it is found to involve a large number of muscles in its production. One cannot point one's finger quickly and forcibly at an object without feeling that almost the entire muscular system of the body is really being called into play. The motor area of the upper extremity is to be thought of, therefore, as containing an enormous number of centres of action, arranged in groups, so that it cannot be divided by sharp lines or circles into flexor or extensor centres, since flexor and extensor action occur simultaneously in many different acts; as, for example, in suddenly closing the hand. If it be divided at all, it must be into regions for various acts, each having a predominate feature.[1] Those acts in which the shoulder movements take the chief part are represented in the upper forward part of the arm area; those in which the thumb and finger movements take the chief part are represented in the lower hinder part of the arm area, and the elbow acts may be placed between these: so that if, in a motor spasm, the first thing which occurs is a motion of the thumb and a closure of the hand, and then pronation and flexion of the wrist and elbow and forcible adduction of the arm follow, while finally the shoulder is raised as if to aid the act of forcible seizure of an object, it is proper to conclude that the cortical irritation began far back in the thumb region of the arm area and gradually increased in intensity. While if the spasm comes on in the reverse order, the point of initial irritation must be located in the shoulder region at the anterior part of the arm area. In both of these areas all of the muscular movements performed are represented; but in the hinder one, it is the hand motions which predominate, or may be said to be primary: in the forward one it is the shoulder motions which are prominent and the others are quite secondary.[2]

[1] Jackson says, "The convulsion is a brutal development of a man's own movements. A severe fit is nothing more than a sudden, excessive and temporary contention of many of the patient's familiar movements, such as winking, articulating, singing, manipulating, etc."

[2] Philosophical Transactions, B. 20.

If, therefore, a very slight irritation occurs in the anterior part of the motor area, it will cause only a shrugging of the shoulder; if it becomes more intense the arm and forearm may begin to move; and if it is at a maximum, the hand participates in the convulsion. If very slight irritation occurs in the posterior part of the motor arm area, the fingers only may twitch, or the thumb be turned in; and as the intensity of the irritation increases, the number of motions made by the extremity as well as their force increases. The spasm does not cease in the muscles first set in motion as other muscles become involved, but continues in all till a maximum of motion is reached. Examples of these varieties of spasm, and of the order of extension of the motions, are found in the first three cases of brachial spasm, soon to be studied. In one of these cases both of these forms of spasm were observed, indicating clearly that the lesion was not a small focus of irritation, but a wide one covering a considerable area, or else that these were two separate foci of disease.

The area governing the movements of the head and the conjugate motion of the eyes, is thought to lie in front of the arm and face areas. Distinctions in the order of centres in the face area are not yet well established clinically, though the upper forward part is known to govern the upper facial muscles, the lower part the muscles of the mouth and tongue. The motions of the legs are so few and simple in man, as compared with monkeys on whom physiological experiments are made, that little need be said regarding them except to note that the centres of movement for the foot and great toe lie farthest back, those for the knee and hip further forward, and those for the trunk and back appear to be limited to the median surface of the hemisphere in the para-central lobule. If a spasm, therefore, begins in the abdominal muscles—as in Case V.—the lesion causing irritation is to be sought on the median surface of the brain, and here it was found at the operation on this case; if it begins in the toes and advances up the leg, the irritating lesion lies in the convexity far back in the motor area.

Electrical stimulus has been frequently applied to the brain

of man, with the result of producing the same effect in respect to motion, which is produced in monkeys. Hence it is interesting to observe the result of such stimulus in monkeys. The accompanying figure (Fig. 8) shows the various areas of the motor zone mapped out by Horsley on the monkey's brain; the subdivisions of the motor area being thus demonstrated. The phenomena of spontaneous spasms in man are identical in kind with those produced by electrical stimulation of the cortex. In both the movement is of the voluntary character; that is, it occurs in harmoniously related muscles

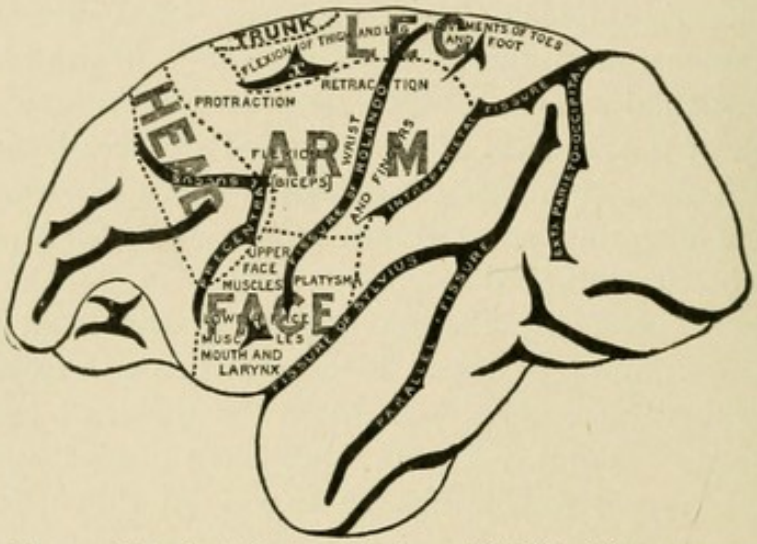

FIG. 8.—Diagram of Monkey's Brain. (Horsley and Schäfer). The motor area and its various functions.

acting together for an end. In both it may be limited to a small movement, as the turning inward of the thumb upon the hand. In both it may be increased in intensity, involving more and more groups of muscles, producing more and more complex acts. In both it may extend to parts of the body represented in adjacent areas. In both opposing movements may be alternately excited, giving rise to what Horsley calls confused movements, such as are seen in a severe convulsion.

JACKSONIAN EPILEPSY—LOCALIZED SPASMS.

It was the study of localized spasms which led Hughlings Jackson long ago to the conclusion that a portion of the

brain cortex is excitable and is motor; and that in the motor area distinct divisions for the control of different movements exist. Such limited spasms have been termed *Jacksonian epilepsy;* and the study of cases has shown that the *location of the source of irritation is the determining element in the form of spasm.*[1]

The symptoms of Jacksonian epilepsy are as follows:

1. *A sensation of numbness* or tingling, or a feeling of motion in some particular part of one side; usually the side of the face, or the thumb and fingers, or the great toe. This has been called the "*Signal symptom*" by Seguin, as it ushers in the attack. It remains during the attack. It is of the greatest value in the diagnosis of the seat of the lesion, and may determine the point at which the trephine is to be placed, if an operation be undertaken. Its situation is usually the same in all the attacks of one patient.

2. *A spasm or convulsion* beginning uniformly in one part of the face or of an extremity and slowly invading other parts. The eyes or face may begin to twitch; or the thumb may turn in and the fingers close; or the toes may move and the ankle become stiff. The spasm commences soon after the signal symptom and increases in intensity. It continues sometimes for three or four minutes, or longer when the convulsion extends to other parts. The movements performed are usually complex movements out of their natural order and sequence and with an irregular degree of force; both tonic and clonic spasms occur.

3. *The spasm extends* from the part in which it begins to other parts in a definite order of extension. Thus if it starts in a part of the face, the entire face is involved and then the shoulder, arm, forearm, and hand, possibly the leg also from the trunk down to the toes. Or if it starts in the hand, the

[1] Hughlings Jackson's early articles are to be found in Medical Times and Gazette, 1861–1863; Aug. 13th, 1864; May 28th, 1868; Aug. 26th, 1871; Jan. 6th, 1872; March to August, 1873; January and October, 1874. May to September, 1875; Jan. 1st, 1876. Lancet, May 16th, 1868; Dec. 13th, 1873. Brit. Med. Journal, May 10th, 1873. His latest lectures have appeared as this volume was in press. The Lumleian Lectures on Convulsive Seizures, Brit. Med. Jour., March 25th and April 5th, 1890.

forearm, arm, and shoulder become successively affected, then the face or the leg. If it starts in the trunk, the thigh and leg and foot follow in the motions. Or it may begin in the foot and gradually creep up the body. As it extends, the muscles first affected become more and more convulsed until the entire limb or the entire side is working. Occasionally the opposite side of the body is affected and if this occurs consciousness may be finally lost.

4. *Consciousness* is almost always perfectly *preserved* during the attack.

5. *After the convulsion* ceases the *parts convulsed*, and especially the part in which the spasm began, and consequently continued longest, *are found to be partially paralyzed* and very awkward, and not infrequently the numbness persists for a time and is found to be associated with a moderate degree of tactile or thermal anæsthesia. This partial paralysis may remain from a few minutes to several hours, depending on the severity and duration of the convulsion. If it remains permanently, it is evidence of organic destructive changes in the cortex. Thus a growing tumor may cause local spasms at first; then local spasms with temporary paralysis; then the duration of the attacks of post-epileptic paralysis increases, and finally the paralysis becomes total and permanent with partial anæsthesia. Usually, if the lesion is only one of irritation, the patient is free from symptoms between the attacks.[1]

These symptoms are absolutely characteristic of Jacksonian epilepsy, and when they occur are absolute evidence of a source of irritation of some kind in the motor area. The exact location in that area is indicated by the "signal symptom" in each case.

The following cases, seen at the Vanderbilt clinic, illus-

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[1] Hughlings Jackson explains the attack as due to a liberation of energy by the nervous elements during rapid decomposition of some material in or a part of the motor cells, probably due to chemical changes. As glycerine may become explosive by the addition of nitric acid forming nitro-glycerine, so he thinks the cells may become ready to discharge by imperfect nutritive changes. For an interesting presentation of this theory and many acute observations, the reader is referred to the Lumleian lectures for 1890. Brit. Med. Jour., March and April, 1890.

trate the phenomena of Jacksonian epilepsy in several of its forms, and the localization of the lesion is shown in each case:

CASE I.—JACKSONIAN EPILEPSY.

Aphasia and Brachial Monospasm.—The patient, a man of 32 years, who had had a chancre in 1882, suffered from vertigo, headache, and insomnia during November and December, 1885, and on December 15th while at work, suddenly felt a numbness in his right hand and let fall a pail which he was carrying. At the same time, he found himself unable to speak. He did not lose consciousness; he understood what his companions said to him, but he could not reply and he could not move his arm. These symptoms passed off in half an hour. Two days later another attack occurred, beginning with a numb feeling at the tips of the fingers; then this extended up the hand and arm; then the fingers became flexed and stiff, though he found that by effort he could straighten them. There were no clonic spasms of the fingers or hand or arm; but the numbness and stiff feeling extended up the arm, shoulder, and neck to the face, which was drawn to the right side, and then speech was lost. This attack lasted twenty minutes without loss of consciousness. Similar attacks occurred every day or two for three weeks, and during this time his headache and insomnia increased. The attacks were not all alike in their commencement. Sometimes they began with aphasia; sometimes with brachial numbness; but in all the distribution of the symptoms was finally the same. The leg and trunk were never involved. The hand often felt cold during the attack, though it was warm to the touch, and was always weak for some hours after an attack. On examination January 3d, 1886, there was no aphasia or facial paresis, but there was slight tactile, and temperature anæsthesia in the hand, some paresis and slight ataxia of movement. He was put at once upon inunctions of mercury and large doses of iodide of potash. The attacks ceased after a week of treatment; and in the course of a month all symptoms had sub-

sided. Six months afterward he reported that on the day previous he had felt a tingling in the right side of his face for a few moments; and had had, for a few days, some headache. He was again put upon specific treatment and no further symptoms developed. He was shown at the clinic in October, 1888, having then been two years without treatment and in good health. All paresis and anæsthesia had disappeared. The diagnosis in the case was a syphilitic gumma in the meninges over the lower posterior part of the second frontal convolution, causing occasional cortical "discharges of energy," resulting in the brachial, facial, and aphasic symptoms. The fact that these symptoms never extended to the leg showed that the area affected was not extensive and did not include the upper third of the motor area. The fact that the symptoms began occasionally with aphasia and occasionally in the arm showed that the lesion was at a point about equidistant between speech and arm areas. The greater frequency with which attacks began with aphasia, and the very slight anæsthesia permanent for a time in the arm showed that the middle third of the motor area was not involved to as great a degree as the aphasic area. The pathological condition was probably a thin, flat, and rather diffuse infiltration of the membranes over the area (Fig. 9). A very similar case was reported by Seguin[1] in which a limited meningitis was found in this area after death (Fig. 14, 1).

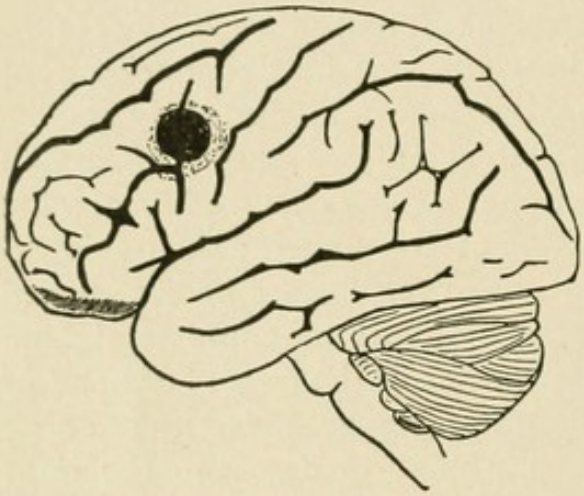

FIG. 9.—Diagram of Left Hemisphere. Situation of lesion in case I.

The co-existence of tactile and temperature sensations with the spasm and the subsequent blunting of these senses with the existence of ataxia is interesting as an indication of the mixed function of the motor area.

[1] Jour. Nerv. and Ment. Dis., June, 1887.

CASE II.—JACKSONIAN EPILEPSY.

Brachial Monospasm Extending to the Face and followed by Aphasia.—Five years ago, when 43 years old, this woman had a carbuncle on her back which was opened. A short time afterward her attacks began, and have continued ever since, sometimes at intervals of several weeks, sometimes as frequently as several in a day. She first notices a prickling sensation in the right hand, which then begins to jerk, and the spasm extends up the arm and involves the right side of the face. As soon as the prickling begins she becomes speechless, and recovers her speech very slowly after the attack passes off. Occasionally her right leg is stiff during the spasm, but it rarely jerks. She sometimes loses consciousness but not as a rule. She suffers from severe occipital and frontal headaches, which are not, however, constant, and at times has a very intense itching in the right side of the face. There are no symptoms of paresis or anæsthesia between the attacks, and the only objective signs are an increase of the tendon reflexes on the right side and slight lateral nystagmus. Her optic discs are normal. She passed the menopause at 37 and is not hysterical. She was not benefited by treatment, and soon ceased to attend.

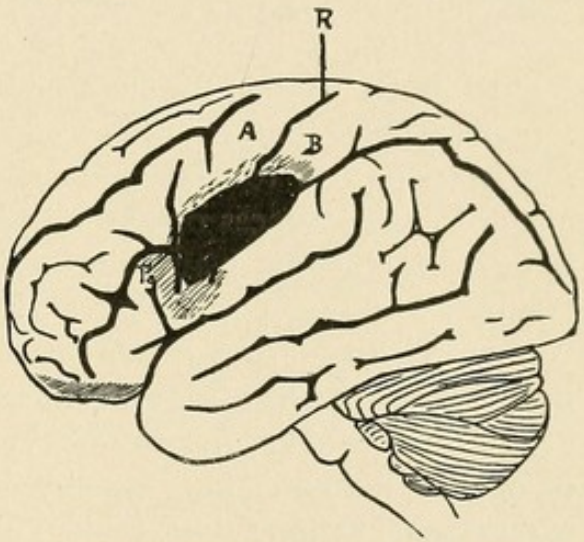

FIG. 10.—Lateral Surface of Left Hemisphere. Situation of lesion in Brachial Monospasm. Case II.

The probable diagnosis was a chronic pachymeningitis over the left hemisphere, affecting the middle of the anterior and posterior central convolutions. The supposed situation of the meningitis is shown in Fig. 10. The lesion is located in this case posterior to that in Case I., because the spasm uniformly began in the hand, speech being lost later.

CASE III.—JACKSONIAN EPILEPSY.

Brachial Monospasm.—The patient, a man of 34 years, had a chancre thirteen years ago, but had been in good health until April, 1887, when attacks began for which he applied to the clinic in April, 1888. These attacks began by a trembling and numb sensation, usually in the fingers of the left hand, but occasionally in the left shoulder. This was followed at once by spasm of the entire left upper extremity. When the tingling began in the fingers the spasm began in the hand and extended up the arm, the wrist and elbow being successively invaded, and finally reached the muscles of the left side of the neck. When it commenced in the shoulder the neck was moved first, and then the spasm extended down the arm to the fingers. Several attacks were observed of both varieties, lasting from one to four minutes, never producing any disturbance of consciousness or extending beyond the muscles of the left upper extremity and neck. The rate of muscular contraction was four per second. Just before the spasm power was 115°. Just after, it was 90°. There was some awkwardness of movement after an attack not noticed before it; but sensation was never impaired. When he applied he had been having as many as five attacks daily. There were no other symptoms at first; but under observation a gradually increasing weakness of the left hand and awkwardness of movement developed, with a sensation of numbness, until power was reduced to 60°, one-half that of the right hand, and finer movements, such as buttoning his clothing, were done clumsily. He had no headache, no tenderness of the head, no optic neuritis, and no cerebral sensations. Under treatment by inunctions of mercury and increasing doses of iodide of potash with bromide of potash his attacks diminished in frequency, so that he had but four during May. In June, after drinking hard and neglecting treatment, he had a return of the attacks, having forty-one in five days. He was sent into Roosevelt Hospital, and the same treatment was carried out with more care than he had probably given it when at work. He was kept in the hospital till August, when he was dis-

charged, as he had had no attacks for three weeks. In October he reported at the dispensary that there had been no return of his attacks. Power was then found to be perfect, 145° to 165° in both hands; sensation perfect, but a slight tremor of the left hand was noticed, and he admitted that he was a little awkward in using it, but said that he had no numbness.

The diagnosis was a syphilitic tumor of the meninges pressing on the middle third of the right anterior and posterior central convolutions, which was absorbed by the specific treatment. The location of the tumor was thought to be at the point indicated in Fig. 11. Had the specific treatment failed, an operation would have been undertaken. In a very similar case reported by Dr. Keen,[1] the removal of a portion of the hand centre was followed by a recovery from attacks whose frequency had been six a day.

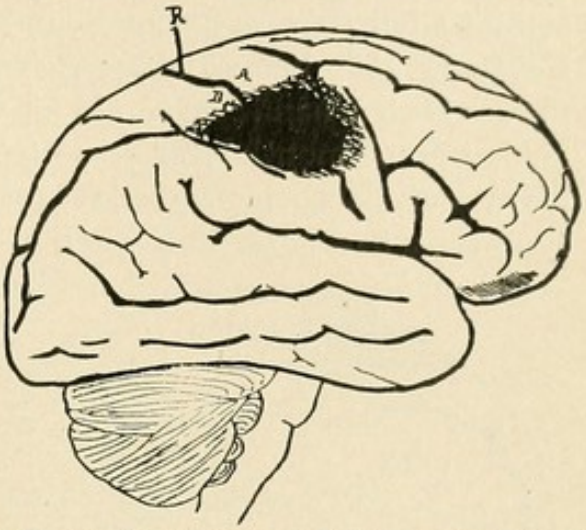

FIG. 11.—Lateral surface of Brain. Situation of lesion in Brachial Monospasm with Paresis. Case III.

CASE IV.—JACKSONIAN EPILEPSY.

Crural Monospasm.—For a year and a half prior to his appearance at the clinic (March, 1889) the patient, a man of 50 years, had been subject to attacks of the following kind. He felt a tingling and numbness in the left foot followed immediately by twitching of the muscles of the toes, which then extended up the leg to the hip until the entire limb felt numb and was in motion. Sometimes this ceased after a few minutes; at other times he felt it ascend the left side of his body to his arm and then he would lose consciousness; and on coming to his senses was told that he had had a general convulsion, in which, however, he never bit his tongue. These

[1] Keen, Med. News, April 12th, 1890, vol. lvi., p. 383.

attacks occurred at first at intervals of a month, then became much more frequent, and then again subsided so that he had had but one in three months prior to March 8th, 1889, when his last attack occurred. Early in his illness he had had vertigo, headache, especially in the right parietal region, and insomnia; but these symptoms subsided after a few months. In February, 1889, he had noticed a beginning weakness and numbness in his left hand; he would let things fall unless he watched his hand. A month later he noticed some difficulty in talking, his articulation being indistinct for a few days; but both these symptoms had passed off when he was examined. On March 8th he was not found to present any abnormal symptoms excepting a slight increase of knee-jerk on the left side. It being probable that he had had a chancre, he was put upon mercury and iodide, and this treatment, with occasional tonics, has been continued for a year, during which time he has had no attacks and has been in good health. It is therefore probable that the attacks were due to a small focus of irritation in the paracentral lobule, either a small gumma or a localized syphilitic meningitis, which had at one time extended downward along the fissure Rolando; and that this was absorbed under the antisyphilitic treatment. The slight, transient paresis of the arm and tongue were probably due to the formation of thrombi of small extent in cortical arteries having free anastomoses but in a state of endarteritis syphilitica; they could hardly have been due to the slight pressure of the syphilitic exudation. The situation of the lesion is shown in Fig. 12. In a very similar cases reported by Hun[1]

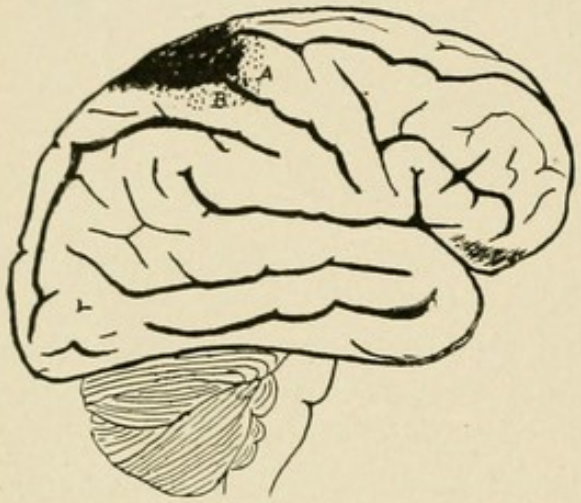

FIG. 12.—Lateral surface of Right Hemisphere. Situation of lesion in Crural Monospasm. Case IV.

[1] Henry Hun, Amer. Jour. Med. Sci., January, 1887, Case IV.

and Seguin[1] a tumor was found in this region. Fig. 14, 4 shows the situation of the tumor in Seguin's case.

CASE V.—JACKSONIAN EPILEPSY.

Monospasm of Trunk and Leg.—The patient, a man of 46 years, had suffered from attacks of a peculiar character for several years. He felt a numbness and tingling in the great toe of the left foot, followed by a twitching of the muscles. This sensation and spasm then extended up the leg, usually stopping at the hip, but occasionally invading the muscles on the left side of the abdomen, which would then contract rhythmically causing him much discomfort. It never extended to the arm, and never produced a loss of consciousness, but was always followed by a sensation of weakness in the leg. There were no other symptoms. This patient continued to have spasms once a week, or oftener, for months. He put himself under the care of Dr. Seguin and was finally induced to go to the New York Hospital and was operated upon by Dr. Weir, who found a local focus of meningitis over the right para-central lobule, with adhesions between the falx, the pia, and the brain on the median surface of the hemisphere; thus establishing the diagnosis of a focus of irritation in the cortical centres for the leg and trunk. The case will be reported in full elsewhere. The situation of the lesion is shown in Fig. 13.

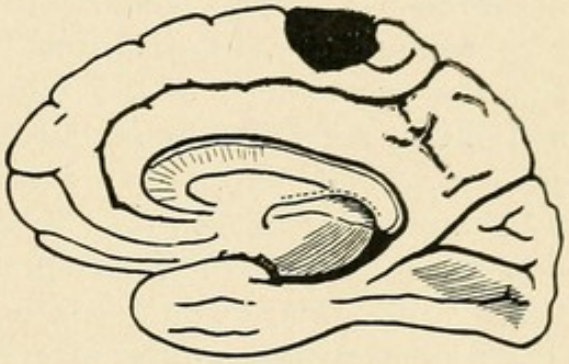

FIG. 13.—Median surface of Right Hemisphere. Situation of lesion in Spasm of Trunk and Leg. Case V.

The location of the lesion in these cases is very evident in the light of the experience of the past ten years, numerous cases exactly similar to these being on record where an operation or an autopsy revealed the exact seat of the disease. It is impossible to cite such cases here, but in the articles of

[1] Jour. Ment. and Nerv. Dis., June, 1887. Case II.

Hughlings Jackson, to which reference has already been made, and in the books of Rolland on Jacksonian epilepsy and of Bastian on Paralysis, cerebral and spinal, as well as in a recent article of Lowenfeld,[1] numerous illustrations of the same form of spasm with autopsies may be found. It is with these cases and those already cited in view that the lesion in each of the preceding cases has been definitely located.

The Nature of the Lesion in these Cases.—In three of the preceding cases the diagnosis of a syphilitic exudation upon the meninges was made. It is well known that the most frequent syphilitic affection producing cerebral symptoms is an obliterating endarteritis, and it is certain that on the occurrence of a thrombosis within such a diseased artery sudden monoplegia may occur, temporary in character, owing to the fact that the anastomosis of the arteries supplying the cortex is so full that a collateral circulation is soon set up and prevents permanent softening. Such a monoplegia is rarely preceded by monospasm; hence in the cases here recorded, since there was a spasm, the lesion was probably not endarteritis with thrombosis. The diagnosis made was meningitis syphilitica. The most convincing point of differentiation between meningitis and thrombosis is the recurrence in the former of the same form of spasm in the same extremity over and over again. When the lesion is the formation of a thrombus, on each occasion of the attack the situation of the spasm and of the subsequent paralysis differs. For when a syphilitic endarteritis invades the vessels it is scattered widely through them and is not confined to one alone. It seems evident, therefore, that in these cases the nature of the lesion was a syphilitic exudation in the meninges or a gumma. That the exudation was not of sufficient thickness to produce much pressure is seen by the very slight symptoms remaining between the attacks. And its comparatively small amount is also proven by the rapid response to treatment in all of the cases.

In the second of these five cases the probable diagnosis was a slowly increasing pachymeningitis with adhesion to the

[1] Arch. f. Psych., xxi., s. I., s. 411, 1890.

cortex not syphilitic in origin. This diagnosis could not be established as the patient was not under observation, for a sufficient time.

In the last case the nature of the lesion was obscure, but on operation a limited meningitis with adhesion of dura, pia, and brain surface was found. Such localized meningitis has been found after death in a number of cases as the cause of the epilepsy.

There are numerous cases of Jacksonian epilepsy in which it is impossible to determine the nature of the disease, and in which, even after death, no gross changes are found. It is well known that a uræmic convulsion may simulate a localized spasm. Therefore some caution is to be exercised in the diagnosis of this disease. When, however, the symptoms occur in the order named, and attacks are repeated, each resembling the others, and no other cause is found, a diagnosis of a source of irritation in the cortex in a particular area is proper. This source of irritation is most frequently tumor, next a small area of softening or of sclerosis, lastly meningeal adhesion.

SURGICAL TREATMENT OF JACKSONIAN EPILEPSY.

Many operations have been performed for the relief of Jacksonian epilepsy with varying success, and it is still too soon to make any definite statements regarding the advisability of surgical interference, although the safety of the operation under antiseptics makes it legitimate in any severe case where the spasms occur frequently. If this is undertaken, the trephine should be applied over the part of the motor area which presides over the movements with which the spasm begins. If on exposing the dura or pia mater, or the brain surface, some lesion is found, such as a localized meningeal thickening, a tumor, an adhesion between membranes and brain, or an abnormal appearance of the gray matter, it is proper to remove it. If nothing is found, it is proper to apply to the cortex exposed the irritation of a faradic current of electricity, by means of two poles made of blunt-pointed probes, held within a quarter of an inch of one another, the current being

increased gradually until motions are elicited in the limb of the opposite side. When the point of the cortex is found whose irritation causes a motion similar to that with which the spasm begins, a small wedge-shaped piece of the cortex may be excised.[1]

This surgical treatment has been carried out successfully in a number of cases by various operators; among whom Macewen, Horsley, Bergmann, Weir, Keen, and Park may be mentioned. In a number of cases the spasms have ceased, the patient has remained well, and has not suffered from permanent paralysis. In some of the cases, however, the spasms have returned subsequently to the operation, so that it is not possible to promise a cure in any case.[2]

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[1] For successful cases see Mills: Trans. Cong. Amer. Phys. and Surg., vol. i., p. 223, 1888. Lloyd: Jour. Nerv. and Ment. Dis., vol. xiv., p. 350, 1889. Keen: Medical News, vol. lvi., p. 381, April 12th, 1890.

[2] For an account of the surgical procedure the reader is referred to the following articles upon cerebral surgery: 1. Brain Surgery, V. Horsley, M.D., Brit. Med. Jour., Oct. 9th, 1886, April 23d, 1887; also Amer. Jour. Med. Sci., April, 1887. 2. Cerebral Surgery, by W. Macewen, M.D., Brit. Med. Jour., Aug. 11th, 1888. 3. A Contribution to the Diagnosis and Treatment of Tumors of the Cerebrum, by R. F. Weir, M.D., and E. C. Seguin, M.D., Amer. Jour. Med. Science, 1888, July, Aug., and Sept. 4. Three Successful Cases of Cerebral Surgery, by W. W. Keen, M.D., Amer. Jour. Med. Science, 1888, Oct. and Nov. 5. Surgery of the Brain, by Roswell Park, M.D., N. Y. Med. Jour., 1888, Nov. 3d, 10th, and 17th. 6. Die Chirurgische Behandlung von Hirnkrankheiten, by E. von Bergmann, Berlin, 1889, 2te Auflage. 7. Cerebral Surgery, by W. W. Keen, M.D., Ref. Handbook Med. Sci., vol. viii., 1889.

CHAPTER IV.

THE MOTOR AREA AND ITS DESTRUCTION.

Cortical Paralysis.—Monoplegia and Hemiplegia.—Case VI., Cortical Hemorrhage producing Aphasia and Hemiplegia; Removal of the Clot; Recovery.—The Centres of Muscular Sense.—Tactile Centres.

CORTICAL PARALYSIS. MONOPLEGIA AND HEMIPLEGIA.

WHEN any portion of the motor area is destroyed by disease, paralysis results. The extent of this paralysis is determined by the location of the destruction. If this is in the lower third of the motor area, the face (except the act of closing the eye) will be paralyzed. If it is in the middle third, it will be the movements of the upper extremity which are impaired. If it is in the upper third, it will be the lower extremity which is rendered useless.

Hemiplegia does not often occur from a cortical lesion. For such a lesion must be very extensive to involve the areas for face, arm, and leg. The ordinary form of hemiplegia is due to a small lesion tearing through the motor tract in the internal capsule, where the fibres from the separate areas are collected in a narrow band, as seen in the diagram, Fig. 26.

Cortical paralysis is usually in the form of a monoplegia, one part only being paralyzed. Hence, in tumors of the cortex, monoplegia rather than hemiplegia is the rule. In Fig. 14 the situation of four such tumors is shown which produced monoplegia in arm or leg. As a tumor becomes larger the paralysis may extend gradually from one limb to another, giving rise to associated monoplegiæ (as in Case II., Fig. 14): the face and arm or the arm and leg are associated together in such paralysis: the face and leg are never paralyzed together when the arm escapes. The diagnosis of a brain tumor can usually be made from the general symptoms, and

when it is localized in the motor area it should be removed. For examples of such cases in which tumors were successfully extirpated, the reader is referred to the articles mentioned at the end of the last chapter.

In small areas of softening in the cortex, due to emboli or to thrombi forming in vessels which are diseased or obstructed by syphilitic deposits, the paralysis is almost always limited to

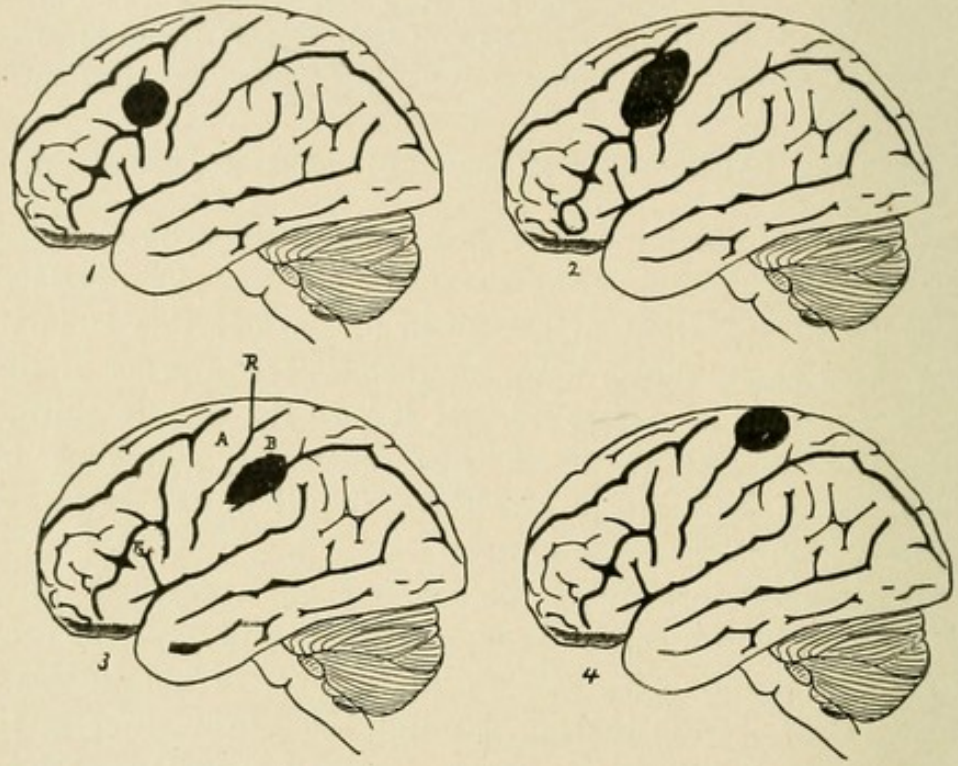

FIG. 14.—Diagrams of Lateral Surface of Brain showing Lesions. 1, Situation of tumor producing brachial spasm and paralysis, successfully removed by Weir, Amer. Jour. Med. Sci., xcvi.; 2, Situation of tumor producing brachial paralysis, and finally hemiplegia, successfully removed by Keen, Amer. Jour. Med. Sci., xcvi., p. 337; 3, Situation of adhesion of membranes producing brachial spasm and paralysis successfully removed by Keen, Amer. Jour. Med. Sci., xcvi., p. 330; 4, Situation of tumor producing crural spasm and paralysis, with autopsy; Seguin, Jour. Nerv. and Ment. Dis., June, 1887.

one limb. These do not admit of relief. Hemorrhages in the meninges causing paralysis, though common in children, are rare in adults. They are usually due to traumatism, as in the remarkable case soon to be studied, which illustrates the character of cortical paralysis when this is very extensive. They may also be due to chronic pachymeningitis. The lesion in chronic pachymeningitis is permanent and progressive; and

when a hematoma forms, the condition of the patient is, as a rule, such that operative interference could not arrest the disease or result in permanent benefit.

The prospects of recovery in cases of cerebral hemorrhage are so bad, and the utility of medical treatment by drugs, electricity, or apparatus is so doubtful, that in any case where surgical interference is possible it affords the only definite hope. When a hemorrhage has occurred within the cerebral hemisphere, lacerating the tracts and destroying the tissue, operative interference is out of the question. For tissue once destroyed in the brain is not repaired by nature, and even were repair possible, the surgeon could not reach and remove the clot without producing a further laceration of very important cerebral structures or serious cerebral hemorrhage. It is, therefore, in cases of cortical or surface hemorrhages only that operations can be done. When such hemorrhages occur as a complication of depressed fractures, they may be treated coincidently with the fracture, and when the cranial bone is elevated the hemorrhage may be stopped and the clot removed.[1] When, however, there is no external evidence of injury, the propriety of trephining becomes a problem of a most serious character. If the symptoms point to a hemorrhage upon the base of the brain, involving by pressure the cranial nerves, the clot cannot be reached by the surgeon. If, however, it is upon the external surface of the hemisphere and produces symptoms of aphasia, hemiplegia, or hemianopsia, it can be removed.

Soon after the onset of an apoplexy, it is rarely possible to determine whether a clot lies upon or within the brain. It is only by observing the character and order of the onset of symptoms, by watching their progress, and determining which are permanent direct local symptoms and which are temporary indirect general symptoms, that a definite diagnosis can be reached. It becomes evident, therefore, that in but a few cases of cerebral hemorrhage can the surgeon accomplish

[1] In an article by Turner, Lancet, 1887, i., p. 116, may be found a number of cases of aphasia produced by wounds and fractures of the skull, and relieved by removing clots, or pieces of bone.

anything, and that only after some careful observation of a patient is he warranted in making an attempt.

In the following case (for the opportunity of seeing and recording which I am indebted to Dr. Chas. McBurney) the conditions for success seemed favorable and the result demonstrates the propriety of the course taken:

CASE VI.—CORTICAL HEMORRHAGE AFTER INJURY.

Right Hemiplegia. Aphasia. Operation. Partial Recovery.—A physician was thrown from his carriage on the 17th of August, 1889. He was slightly stunned by the fall, but was able to help his wife, who seemed badly hurt, to reach home. For some hours he appeared to be suffering merely from bruises, and though lying down was able to converse with a patient and give him a hypodermic injection. In the course of the evening, however, he became delirious and excited, then rather stupid, and regarding the occurrences of the three following days, he has no recollection; though he was able during all this time to recognize his wife. On the morning of the 18th he was found to be completely hemiplegic on the right side and completely aphasic; although the power of understanding what was heard was never lost. The condition of hemiplegia with complete motor aphasia remained until December, 1889, at the time of the operation. At that time he was able to move his leg a little and to drag it forward when held up by two persons. He could not move his arm or hand at all. His face was slightly flat on the right side, but his tongue protruded straight. The only sounds he could make were the vowel sounds; no formed words were possible. The right pupil was one-third smaller than the left one. Sensations of touch and pain and of location were somewhat impaired, but not lost on the affected side. To cold and hot objects he seemed extremely sensitive. The tendon reflexes were extremely exaggerated, wrist and finger clonus, as well as patella and ankle clonus, being easily elicited; and ankle clonus could not be stopped and often persisted for hours when once started. The hand was flexed and pronated; the

leg extended; both were rigid. He controlled his sphincters perfectly. Mentally, he appeared to be less active than previous to the accident; though understanding what was said, it was difficult to determine the reliability of his assent or dissent in the sensory tests. He was emotionally unstable, laughing very easily and being at times depressed. Sometimes the laughter was like that of a demented person, causeless and silly. Sight was good, and the optic discs appeared normal. Hearing was unaffected.

The chief and most permanent symptom was, therefore, the motor aphasia. The hemiplegia had somewhat improved during three months, but the arm was still totally helpless. The inequality of the pupils, the mode of onset, and the permanence of the symptoms pointed to the occurrence of a cerebral hemorrhage in or upon the left hemisphere. The total aphasia could not be explained by a lesion in any other position than in the posterior part of the third frontal convolution; for while aphasia may occur as a temporary symptom in lesions of the left internal capsule, it is not one which persists. The paralysis, greater in the arm than in the leg, was consistent with the idea of a cortical lesion in the middle half of the anterior central convolution. The escape of face and tongue from the paralysis would not have been possible had the lesion causing the aphasia and brachial paralysis lain in the internal capsule, or in fact anywhere within the brain, because the speech tract and motor arm tract are separated from each other by the motor tract to the face and tongue: and the three, as a rule, are injured together. The hemorrhage was, therefore, thought to be cortical and to lie in the position indicated in Fig. 15, with two areas of greatest intensity and an area of less intensity between them. Inasmuch as the aphasia was the most prominent symptom and had shown no tendency to subside, it was thought that the posterior part of the third frontal convolution was the point of greatest pressure.

Operation.—On December 13th Dr. McBurney undertook the operation of trephining. The trephine was applied at a point one inch and seven-eighths behind and seven-eighths of

an inch above the external angular process of the frontal bone; the line backward being parallel to the zygoma. The dura did not pulsate, and when it was incised the pia was found to be œdematous, and the surface of the brain was seen to be without pulsation and to be covered with a clot of blood over the posterior part of the area laid bare, which was the third frontal gyrus.

The opening in the skull was accordingly enlarged backward and upward by the use of rongeur forceps. As the lower part of the anterior central gyrus was exposed, it was seen to be also covered by the clot of blood, which was continuous with a thicker layer which extended into the fissure of Rolando. This fissure was consequently laid bare by an increase in the cranial opening, and was followed up to the junction of its middle and upper thirds, where the clot was found to cease. The pia was then incised as the clot lay beneath it, and the clot was gently removed by sponging. It was found to fill up and greatly distend the fissure of Rolando, and in the *cul-de-sac* at the lower limit of the fissure a mass of blood had collected, making pressure forward as well as inward, thus adding to the compression of Broca's speech centre by the thin surface clot already described. The clot was partly organized and came away in small shreds upon the fine aseptic sponges used. The brain cortex was slightly discolored, and at no point of the area exposed was it seen to pulsate. The veins in the pia, which had been cut, were tied, drainage-tubes were put in place, the dura was stitched, the scalp-wound was united by sutures, and the bandages were adjusted.

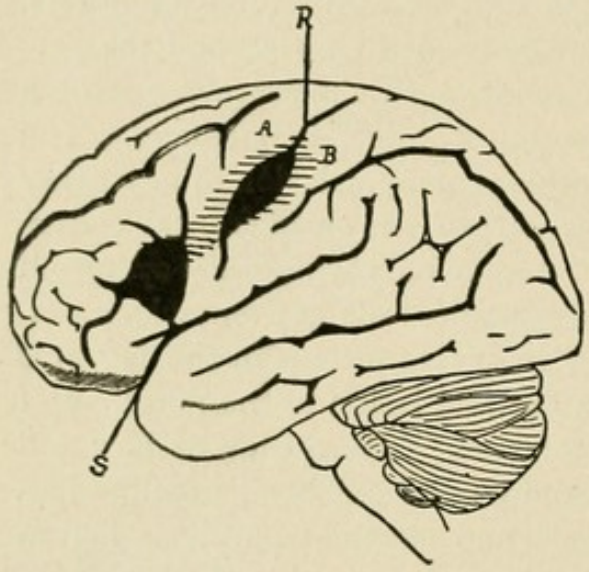

FIG. 15.—Diagram of Lateral Surface of Left Hemisphere. Situation of clotssuccessfully diagnosticated and removed. Case VI.

The entire operation was conducted according to the most strictly aseptic method, with success, as the subsequent lack of suppuration and the normal temperature demonstrated. At the third dressing, on the fourth day, the brain was seen to pulsate under the entire opening. The drainage-tubes were removed in a week, and in the course of a month the wound had entirely healed.

From the first day after the operation, a decided change was noticed in the mental condition of the patient. He was much more intelligent and no longer silly or emotional. In the course of the first week, it was evident that an improvement in his power of movement was beginning. At the end of three months, he was able to walk with the aid of a cane and to perform many of the larger motions of the arm and forearm, and flexion but not extension of the fingers. Speech had been also returning slowly under instruction and constant practice. At present, six months after the operation, he can repeat many words after another person; can say all the letters of the alphabet and reply simply to ordinary questions. It appears as if he were learning, like a person studying a foreign tongue, to articulate strange and unfamiliar sounds. But his efforts now are successful, while formerly they could not be begun. It is very evident that the removal of the clot has been followed by an improvement in his mental condition, motor power, and speech. The irregularity of the pupils subsided after three months; the rigidity of the arm and leg and the increased tendon reflexes remain, but his general condition is certainly far better than prior to the operation, and there is every reason to hope for recovery. A marked deficiency of tissue and depression of surface remain partly filled by dense connective tissue; but as the hair covers this completely it is not very noticeable and is easily concealed by a cap.[1]

[1] C. B. Ball (Brit. Med. Jour., April, 1888, p. 748) and Mouisset (Lyon Médical, Oct., 1889, p. 204) report successful operations for aphasia, with removal of a cortical clot and recovery of speech.

THE CENTRES OF MUSCULAR SENSE.

The localization of the centres appreciating the position of a limb (the so-called muscular sense) has been a matter of controversy. It may therefore be interesting to note that in the preceding case, in which the clot compressed the motor centres, muscular sense was impaired. This was more strikingly evident in another patient, also seen with Dr. McBurney. In this man a bolt had been driven through the left parietal bone over the hand centre and had produced an almost complete paralysis of the right hand and arm, with only partial affection of the face and leg. In this patient, tactile and pain and temperature senses were slightly impaired, and muscular sense was markedly deficient. He could locate accurately the point touched upon the hand, but was unable to place his left hand in positions given to the right hand with his eyes closed. If the right hand was placed in a fist position and he was told to put his left hand in the same position, he was just as likely to open as to close it; and other positions also were not appreciated nor imitated. An operation was performed to elevate a depressed portion of skull in this man, and extensive injury of the parietal cortex was found, with an abscess under the depressed bones. The exact location of the lesion was therefore certainly in the posterior central region. These two cases would therefore appear to indicate a coincidence in situation of the motor and muscular sense centres in the cortex.

THE TACTILE CENTRES.

The existence of slight anæsthesia in both of these cases and the uniform occurrence of sensations of numbness in cases of Jacksonian epilepsy when the irritation occurs in the motor area seems to show quite clearly that the tactile and motor centres coincide. Horsley has suggested that in the three chief layers of cells which make up the motor cortex the three functions of motor power, muscular sense, and tactile sense are located, so that each part of the motor area has a sensory function as well. When one considers how nicely muscular

and tactile impressions regulate motor efforts, this theory gains in probability. The efforts to lift a bit of cotton or a lump of lead differ greatly, and are directly determined by the perception of weight and contact.

Whether there are tactile centres separate from the motor area in the hippocampal region, as Ferrier alone holds, is not yet determined for man. Dana has shown [1] by a large collection of cases that lesions in the motor area are very frequently associated with anæsthesia. And since the sensory tract in the internal capsule lies just behind the motor tract, it is not unlikely that tactile centres do lie (as I showed in 1884 [2]) in the parietal region just behind the motor area or else coincide with it. It is certain that lesions in the posterior half of the motor area are frequently productive of anæsthesia, while lesions in the anterior half of the motor area are rarely productive of anæsthesia. In the posterior part lie the motor centres of the smallest joints, fingers, and toes, and, since the finer movements of these are guided by touch, it is not unlikely that about these centres tactile areas lie. The question of the localization of tactile temperature and pain senses is still an open one; and more precise statements are not yet warranted.

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[1] Dana: Jour. Nerv. and Ment. Dis., 1888, p. 650.

[2] Starr: Jour. Nerv. and Ment. Dis., July, 1884.

CHAPTER V.

THE VISUAL AREA AND ITS AFFECTIONS.

The limits of the visual area.—Hemianopsia; the visual tract.—Sensory epilepsy from irritation of the visual cortex.—Case VII., Visual hallucinations and temporary hemianopsia.—Case VIII., Visual hallucinations and temporary hemianopsia.—Case IX., Hemianopsia of cortical origin.

THE VISUAL AREA.

THE visual area of the brain is located in the occipital convolutions and the cuneus. The limits of this area are shown in Fig. 16 (1 and 2) and are no longer a matter of dispute. Since 1884, when I was able to collect[1] 27 cases of lesion in the occipital lobes, and to prove by their analysis that the visual area was located in this region, about 20 additional cases have been published. These seem to confirm the conclusion reached by Seguin, in 1886,[2] that the cuneus is the part of the occipital lobe in which the visual tract ends. That the angular gyrus has

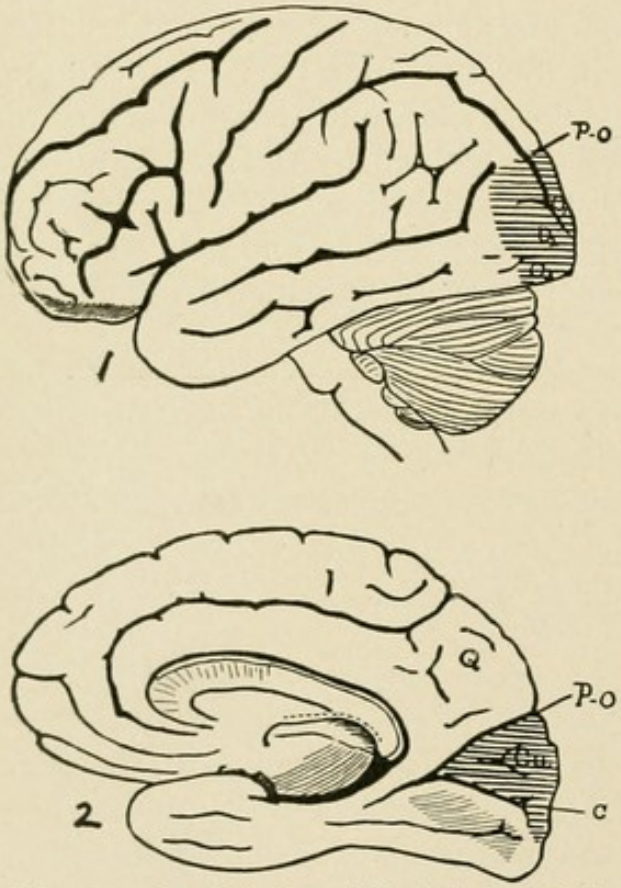

FIG. 16.—1, Lateral Surface of Hemisphere; 2, Median Surface of Hemisphere. The shading shows the visual area.

[1] Starr: "Visual Area of the Brain," Amer. Jour. Med. Sci., Jan., 1884.

[2] Seguin: "Hemianopsia," Jour. of Nerv. and Ment. Dis., Jan., 1886.

nothing to do with vision in man (whatever its function in monkeys) is not only proven by these cases of lesion in the occipital region, but is also determined by a collection [1] of 23 cases of lesion in the angular gyrus without disturbance of vision. The visual area is therefore limited in man to the convolutions of the occipital lobe.

The character of the blindness produced by the destruction of this cortical area is peculiar. It is termed bilateral homonymous hemianopsia, that is, a blindness in the like-named halves of both visual fields. When the right occipital lobe is affected the patient cannot see any objects lying to the left side of the middle line as he looks forward. When the left occipital lobe is affected the patient can see nothing to the right side of the middle line. This peculiar form of blindness is easily understood by a consideration of the anatomy of the visual tract as shown in the diagram (Fig. 17). It is seen that each optic nerve divides into two parts at the optic chiasm. The larger part crosses to the opposite optic tract; the smaller joins the optic tract of the same side. Each optic tract then contains fibres from both eyes. It is found that the fibres from the temporal half of the retina, which receives impulses from the nasal half of the visual field, do not decussate. Hence a lesion of the right optic tract will cause blindness in the nasal half of the visual field of the right eye. Fibres from the nasal half of the retina, which receives impulses from the temporal half of the visual field, do decussate with those of the opposite side. Hence a lesion of the right optic tract will cause blindness in the temporal half of the visual field of the left eye. Therefore a lesion of the right optic tract causes a loss of function in the right half of both retinæ, and as a consequence the blindness is limited to the left half of both visual fields. Both eyes are affected, the blindness is bilateral; it is but one half of the vision which is lost, the blindness is therefore called hemianopsia; and like-named halves of the two visual fields are blind, hence the term bilateral homonymous hemianopsia.

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[1] Starr: "The Lesions of Sensory Aphasia." Brain, July, 1889.

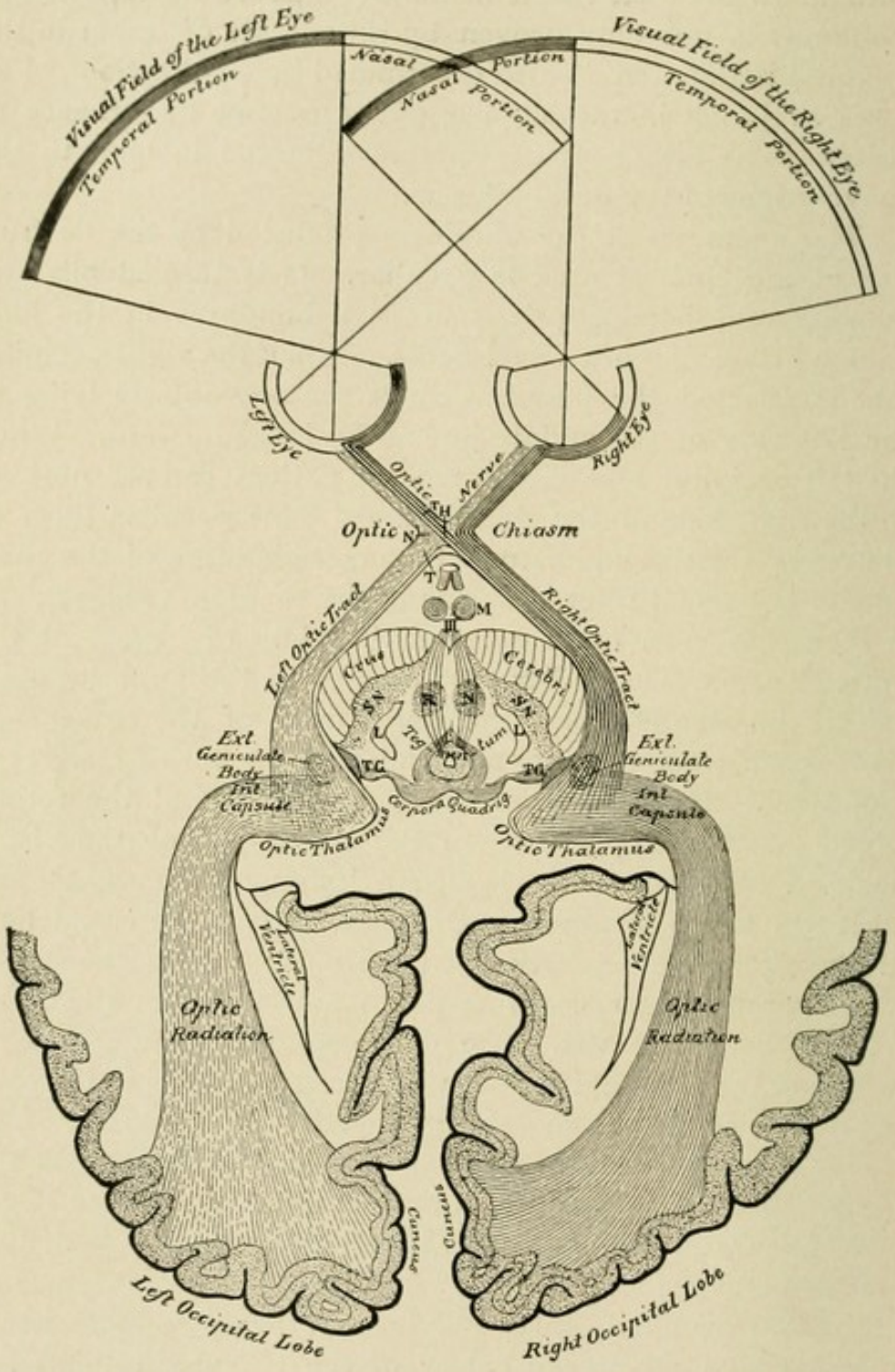

FIG. 17.—The Optic and Visual Tracts. *N*, Lesion causing nasal hemianopsia, unilateral; *T*, lesion causing temporal hemianopsia, unilateral; *H*, lesion causing bilateral heteronymous hemianopsia. Any lesion lying behind the chiasm causes bilateral homonymous hemianopsia; *RN*, red nucleus of tegmentum; *SN*, substantia nigra of crus; *L*, lemniscus; *IG*, inter. geniculate body; *III*, third nerve exit; *I*, infundibulum; *CM*, corpora albicantia.

From the optic chiasm, each optic tract passes around the crus cerebri and terminates in the external geniculate body, in the optic thalamus, and in the anterior corpus quadrigeminum, as seen in the diagram. From these three masses of gray matter new fibres start out which issue from the outer side of the optic thalamus, enter the internal capsule, and curve backward through the occipital lobe, lying on the outer side of the posterior horn of the lateral ventricle, and thus reach the occipital convolutions, ending chiefly in the cuneus. It is evident that a lesion anywhere in the course of the visual tract from the optic chiasm to the cuneus will cause bilateral homonymous hemianopsia. This is the only form of blindness produced by a lesion of the visual area, or caused by disease of one cerebral hemisphere. Blindness in one eye alone is not produced by cortical disease. When it occurs it is usually either from disease in the eyeball or in the optic nerve, or it is hysterical.

Since the visual tract is a long one, it is desirable to be able to locate a lesion in its various parts. This can easily be done by a study of the symptoms associated with the hemianopsia. Thus if the optic tract is involved, as it curves around the crus cerebri, the same lesion which causes hemianopsia will be likely to affect the motor tract in its passage through the crus, or to involve the third nerve at its exit from the crus (compare the diagram Fig. 17 with the diagram Fig. 26). In this case hemianopsia and hemiplegia of one side will be combined with oculo-motor paralysis of the other side. If the optic tract is involved at its termination in the thalamus, the lesion, unless very small, will affect the sensory tract from the opposite side of the body in its passage along the internal capsule at the side of the thalamus; and in this case hemianæsthesia will be found as well as hemianopsia. If the visual tract in the internal capsule is involved by disease of the capsule or basal ganglia, the proximity of both sensory and motor tracts may render a combination of hemianopsia, hemianæsthesia, and hemiplegia possible. If the visual tract is invaded within the occipital lobe or in the cortex, none of these other symptoms will be present; hence their absence is

an aid to the localization of the lesion. As will be shown later (Chapter VII.), when the left visual tract is invaded in the occipital lobe, word-blindness may be found. Such a condition does not occur when the right hemisphere is diseased.

SENSORY EPILEPTIC SEIZURES FROM IRRITATION OF THE VISUAL AREA.

Since "nervous discharges" occurring in the sensori-motor area give rise to spasm followed by paralysis distinctly limited to a single limb, it is not surprising that attacks of an homologous nature should be observed when other regions of the cortex are invaded.[1] The two following cases indicate the existence of "discharges" in a purely sensory area of the cortex; namely, in the visual area, which lies in the occipital lobes. The initial symptom in both cases was the sudden appearance of a bright light to one side of the middle line in both eyes, followed by a blindness in the corresponding visual field. The hallucination of light may be regarded as homologous to the motor spasm; the blindness as homologous to the paralysis. The situation of the initial irritation must have been in the occipital convolutions, since they receive impressions from the eyes; each occipital lobe being functionally related to one half of both eyes, so that the hallucination or blindness was necessarily one-sided. These cases are of importance, as they might be easily mistaken either for migraine or for ordinary epilepsy of the *petit mal* type. But a consideration of the symptoms shows that either of these diagnoses would be erroneous.

CASE VII.—CORTICAL DISCHARGE IN THE LEFT OCCIPITAL LOBE.

Visual Hallucination followed by Temporary Right Hemianopsia.—The attacks from which the patient suffered began in 1886, when he was 39 years old, after a day of severe in-

[1] Hughlings Jackson: Brit. Med. Jour., Jan. 11th, 1879; Charcot: Leçons, Progrès Méd., 1887; Pitres: Revue de Méd., Aug., 1888; Löwenfeld: Arch. f. Psych., xxi., p. 423; Anderson: Brain, 1886, p. 385.

tellectual work. While reading he felt a queer sensation in his head and found himself blind in the right half of both visual fields. He called his wife to apply cold water to his head, and thus arrested the attack at once. Similar attacks recurred at intervals of six or eight weeks for two years. Sometimes the hemianopsia is preceded by an aura of blue or red light or of "smoke" in the right visual field, and then the blindness follows. The attacks last from three to ten minutes and are not attended by loss of consciousness or followed by headache. There is no vertigo with them, but his head feels full and sometimes his right upper eyelid twitches.

He is a physician and observes and describes the attacks intelligently. He has no other symptoms. Bromide reduces the frequency of the attack. His V = $\frac{20}{20}$; pupils are equal and react normally; he has no insufficiency of the ocular muscles; his visual fields are normal to light and colors; but his optic discs are rather pale. When relieved of a condition of extreme neurasthenia the attacks become less frequent but they still occur.

CASE VIII.—CORTICAL DISCHARGE FROM THE OCCIPITAL LOBES.

Visual Hallucinations followed by Hemianopsia.—The patient has had attacks since the age of 21. They begin with a sudden vision of yellow sparks or flashes of light in one half of the visual field of both eyes, either to the right or to the left, never in the middle line, and then in a few seconds the entire half of the visual field in which he has seen the flashes becomes dark for a few minutes. He feels dizzy and bewildered at the time, and as if about to faint, but he has never lost consciousness or fallen or had a twitching of the muscles. The attacks are often followed by severe unilateral headache. During the attacks his face flushes and his head feels full. He can arrest the attack by putting his feet in hot water. Bromides diminish the frequency of the attacks, which ordinarily come about once a week. He has no other symptoms. V = $\frac{20}{20}$; pupils are dilated and react normally; insufficiency of

the externi, ½°; adduction, 9°; abduction, 6°; visual fields normal and discs clear.

In the first case the left cuneus (Fig. 16) must have been irritated, since the hallucinations and blindness were uniformly in the right half of the visual field.

In the second case the irritation affected sometimes one side and sometimes the other. Since the cunei lie adjacent to one another, separated only by the falx cerebri, it seems probable that in this case the irritating focus of disease may have been in the falx between the cunei, and variations in the cerebral circulation caused the irritation to be manifested at times on one side and at times on the other. It is well known that hemorrhages in the falx at this point, by pressing simultaneously on both cunei, have caused total blindness by producing double hemianopsia. Such cases have been recorded by Chauffard and Bouveret (1888).[1]

The following case, which was referred to me by Dr. John E. Weeks, Chief of the Ophthalmic department of the Clinic, and the history of which he has kindly prepared, well illustrates the form of blindness due to cortical lesion:

CASE IX.—LEFT HEMIANOPSIA.

A woman was forced to fly from a burning house within a week of her confinement, and on reaching the street became weak and noticed a sudden blindness in both eyes. She stumbled but did not fall or lose consciousness, and she had no convulsion. For a few hours subsequently she felt a weakness in her left side, but this soon passed off. The blindness remained. Seven months after the onset she was found to have no symptoms of paralysis or anæsthesia, but a well-marked left homonymous hemianopsia. She was blind in the left half of both visual fields; the blindness, however, not involving the point of distinct vision, but passing, as the diagram (Fig. 18) shows, 3° around it. When she looked straight forward she saw everything to the right side, nothing to the left side. Her optic nerves were normal, and vision was $\frac{20}{30}$ + in

[1] Revue de Médecine.

both eyes. There were no symptoms of cranial nerve paralysis to indicate a lesion of the optic tract on the base. There was neither hemiplegia nor hemianæsthesia, one or both of which would have been present had the lesion been in the

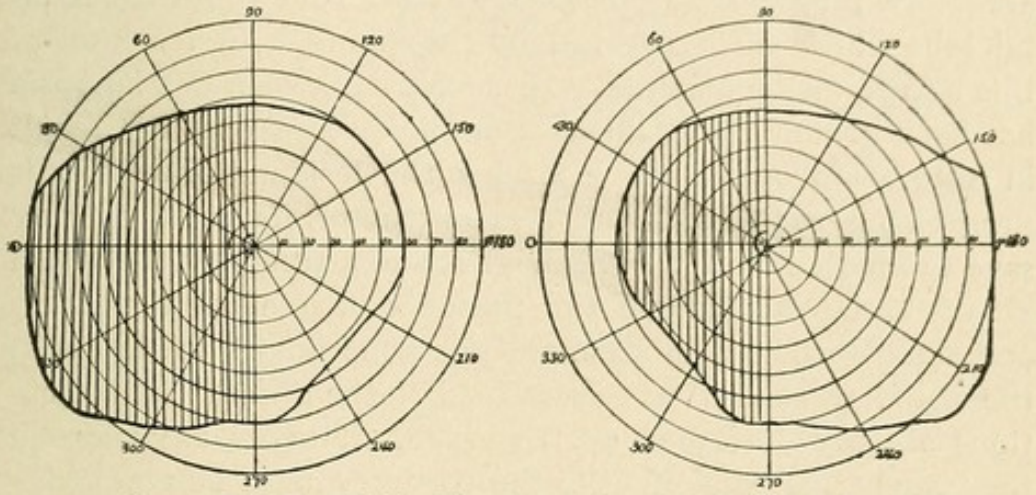

FIG. 18.—The Visual Fields. The shading shows the blind field in Case IX.

optic thalamus or internal capsule. The lesion, therefore, must have lain in the right occipital lobe or its cortex (Fig. 19).

The diagnosis of the nature of the lesion was as evident as that of its location. A sudden cerebral apoplexy in a puerperal woman is almost certainly due to the lodgment of a clot in some artery of the brain, and the origin of such a clot, acting as an embolus is always from a venous thrombus in the uterus when, as in this case, there is no cardiac disease or phlebitis elsewhere. The only artery which supplies the occipital convolutions exclusively is the posterior cerebral, and although thrombi and emboli in this artery are rare, there is a sufficient number of cases on record in which they have been found to warrant the diagnosis in this case.

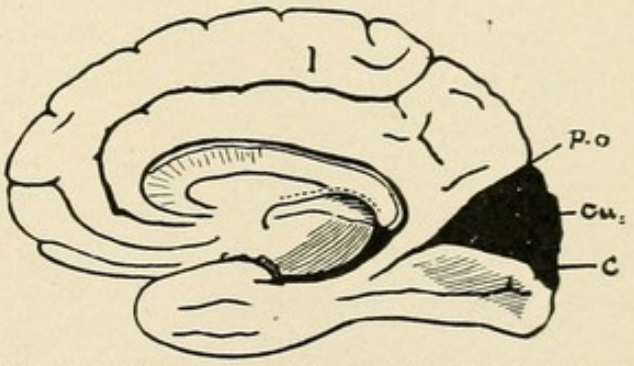

FIG. 19.—Median Surface of Hemisphere. Lesion causing left hemianopsia.

There are a number of cases upon record in which lesions of a destructive character have been removed from the occipital lobe. In 1882 Wernicke and Hahn reported [1] a case of abscess of this region successfully diagnosticated and evacuated. In 1887 Birdsall diagnosticated a tumor in the occipital lobe which Weir removed by trephining.[2] Other cases of this nature might be cited. In such cases, when hemianopsia has been produced the removal of the disease does not result in restoration of vision. But when the lesion is one which is progressing, such as an abscess or tumor, its removal may save the patient's life. The diagnosis of a tumor in the visual area is to be made from the presence of general symptoms combined with hemianopsia, and the situation of the lesion in the visual tract can be ascertained if the differential indications already described are regarded. Tumors anywhere in the visual tract, excepting in the cortex, cannot be reached.

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[1] Virchow's Arch., vol. lxxxvii., p. 335.

[2] Med. News, April 16th, 1887.

CHAPTER VI.

THE CORTICAL AREAS GOVERNING LANGUAGE.

The varieties of aphasia.—Mental images or concepts and their physical bases.—Apraxia; Inability to recognize objects; Case with successful operation.—The musical sense and its loss.—The seat of the lesion in apraxia.—Word images and their physical basis.—Aphasia; Inability to recognize or to use language.—Word deafness; Word blindness; Motor aphasia; Agraphia.—Paraphasia.—The tests for aphasia.—Illustrative Cases.

BEFORE entering upon the study of aphasia, it may be of service, in understanding affections of speech, to look at the mental and physical mechanism which regulates the use of language.

The accepted notion to-day is that every word in ordinary use has a complex mental substratum, which may be termed the word-image, made up of a number of memory-pictures. The memory of the sound of a word as spoken, the memory of the appearance of the word as printed, the memory of the muscular movements needed to write the word or to pronounce it, are known to be distinct from one another and yet to be associated together. Loss of one of these memory-pictures, or disturbance in their association, impairs the integrity of the word-image, and produces such defects in its use as are indicated by the names given to the respective varieties of aphasia. For the division of disturbances of speech into the two great classes of sensory and motor aphasia—the first due to defect in the receptive and the second due to impairment of the emissive functions of the brain—has been followed by a further subdivision into several varieties. These are word-deafness, word-blindness, agraphia, motor aphasia, and paraphasia. But, before considering these defects, let us look at the physical basis of the thought which lies back of

the word. For the word is only a convenient means of expressing a thought, and thinking precedes language, as every intelligent observer of animal life or of the education of a child knows.

MENTAL IMAGES AND THEIR PHYSICAL BASIS.

Objects and actions are the chief subjects of thinking, and their mental substratum is not difficult to discover. Take an example. A steel pen has a certain shape and size and consistence which I see and feel. The shape and hardness perceived by eye and finger are associated and are remembered, and the mental substratum or idea of the pen consists of these two memory-pictures joined together. Other pens have forms of their own, and of them, too, I have a series of memory-pictures. But the idea of the object is only made complete when its use is known. And so to the memory-pictures of the pen is added another of the act of writing; itself a complex image, for that act has been watched and practised before being acquired, and its recollection involves the recalling of those memories of activity of which I have been a spectator or a participant. The mental image of the action is associated with the various mental images of different kinds of pens, so that it forms a common basis for the for-

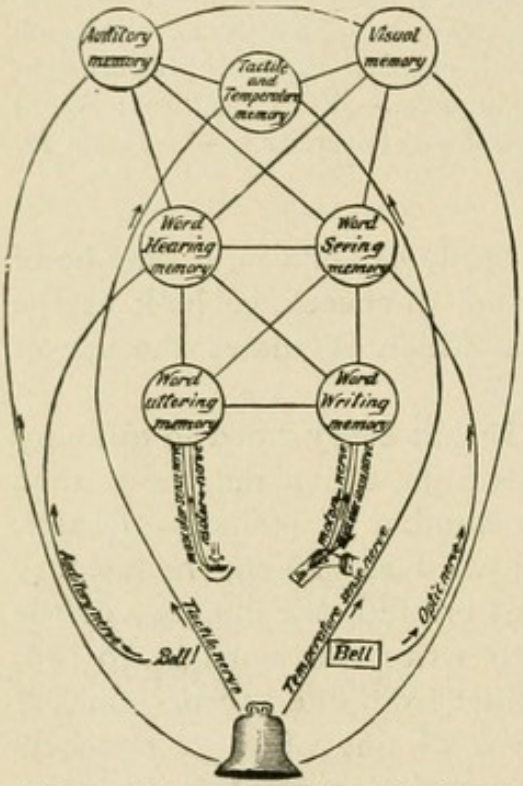

FIG. 20.—Diagram to illustrate the Concept "Bell," and to show the Varieties of Apraxia and Aphasia. The memory pictures are relics of past perceptions received through different senses. Their association makes up the mental image bell. The word image is made up of the memories of the sound and appearance of the word, and of the uttering and writing-effort memories; these are joined together. The mental image and the word image are also joined with one another, making up the concept "bell."

mation of a general idea of a pen as an object used for writing. And this idea, complex as it is, made up of numerous memory-pictures, is acquired by the mind through observation long before language is used. The baby may try to write before it can say "pen," or before it can describe the act of writing. The words, then, are added to the mental image to make it communicable to others, but are not essential to the idea.

A bell has, as its mental basis, four distinct memory-pictures; it has weight, form, a cold smooth surface, and sound; in fact, every object, if thus analyzed, will be found to vary from every other in respect of the memory-pictures which form its mental substratum. The figure shows the various kinds of memory-pictures and word-images making up the concept "bell." What is true of these single words will be found to be true of every other, if the trouble be taken to analyze one's subjective knowledge of them—an analysis full of interest.

APRAXIA.

It is a fundamental position involved in the accepted theory of cerebral localization that memories are the residua of perceptions and are therefore localizable in the regions of the brain concerned in perception. It follows that these memories forming the idea of an object or an action, being distinct from one another, may be lost by disease of the brain having a limited extent, and that the character of the memories lost will depend on the location of the disease. Now, cases have been recorded in which persons acted as if they no longer possessed such object-memories, for they failed to recognize things formerly familiar. A fork, a cane, a pen may be taken up and looked at by such a person, and yet held or used in a manner which clearly shows that it awakens no idea of its use. And this symptom, for which at first the term blindness of mind was used, is found to extend to other senses than that of sight. Thus the tick of the watch, the sound of a bell, a melody of music, may fail to arouse the idea which it formerly awakened, and the patient then has deafness of mind; or an

odor or taste no longer calls up the notion of the thing smelt or tasted; and thus it is found that each or all of the sensory organs, when called into play, may fail to arouse an intelligent perception of the object exciting them. For this general symptom of inability to recognize the use or import of an object the term *apraxia* is now employed. And since apraxia is a symptom which is very frequently associated with aphasia and which, in fact, may lie at the basis of aphasia, it should always be looked for in a patient. To test for apraxia it is only necessary to present various objects to a person in various ways and notice whether he gives evidence of recognition. Have him watched by his friends, and they will be able to tell whether he still chooses his articles of food at the table intelligently; whether he still knows how to put on his clothes, to use various toilet articles; to sew or knit or embroider if the patient is a lady; to admire pictures, or flowers, or perfumes, as before the illness began. The patient may or may not be able to name these objects; that, at present, is not the question. But is it evident that the object awakens an idea in the mind?

The variety of apraxia known as blindness of mind is the one most commonly found, and it may not be uninteresting to recount one case of it, especially as that case affords the first example of its successful treatment by operation.

It is a case recorded by Macewen,[1] of Glasgow, and is as follows:

A man who had received an injury a year previously to his applying for treatment suffered from deep melancholy and strong homicidal tendencies which were relieved by paroxysms of pain in the head. There were no motor phenomena, but it was discovered that immediately after the accident, and for two weeks subsequently, he had suffered from psychical blindness. Physically he could see, but what he saw conveyed no impression to his mind. An object presented itself before him which he could not make out, but when this object emitted sounds of the human voice he at once recognized it to be a man. In attempting to read, he saw what he consid-

[1] British Medical Journal, Aug. 11th, 1888.

ered must be letters and words, but they were unknown symbols to him; they conveyed no impression of their meaning; the memory of their signs was gone; the book was a sealed book to him. These phenomena gave the key to the hidden lesion in the brain. On operation the angular gyrus was exposed, and it was found that a portion of the internal table of the skull had been detached from the outer and had produced pressure on the posterior portion of the supra-marginal convolution, while a corner of it had penetrated and lay embedded in the anterior portion of the angular gyrus. Removal of the bone resulted in complete recovery from the pain and mental symptoms.

The variety of apraxia known as deafness of mind has recently been studied by Oppenheim,[1] of Berlin. He noticed that while some aphasics retain their musical faculties, others may lose the power to follow melodies, to appreciate music, or to hum or sing the tunes which they formerly knew. Thus, he relates that a patient of Finkelnburg, a flutist, noticed an impairment and finally a loss of power to follow notes, to play or to write notes, at the same time that he lost his speech. Ballet relates the case of a musician who suddenly lost his power to read music, and Kast one of a musician who suddenly became note-deaf; and Oppenheim gives histories of eleven such patients seen by him. That this is not a part of aphasia in any sense is proved by the fact that in many patients no such loss of musical power accompanies aphasia; and in many cases of aphasia, it is expressly stated that the musical faculties were unimpaired. Granet tells of a patient who could say only one word, "pardi," and yet who could follow music, and not only sing the tune of the Marseillaise, but, while singing, pronounce several words, "patrie," "enfant," etc., which he could not say voluntarily; and Proust relates the case of a musician who could neither speak nor write, yet who could write a musical melody which he heard sung.[2]

In view of these observations, it is worth while to test intelligent patients as to their musical faculties—their power to

[1] Charité Annalen, xiii., 1888.

[2] L'Encéphale, 1888, No. 5.

recognize tunes, hymns, melodies, their power to sing or hum a tune, and to read or write music or play on various instruments.[1]

Investigations of the power of a person to recognize and name an object by touch or taste or smell would be interesting, but have not often been undertaken. The loss of the muscular sense and consequent inability to perform delicate motions of the hands has rendered deaf and dumb persons who use their peculiar language of motion incapable of conveying their ideas; and if cases of this kind were more closely investigated, some light might be thrown on the exact location of muscular sense. But all of us, by the feeling of many objects, can recognize them at once with our eyes closed, and thus arouse the memory-pictures of an object through this channel of sensation. Recently a case has been recorded by Farjes, in France, of a gentleman afflicted with aphasia, who, though unable to name an object when he saw it, could do so when he felt it, thus proving that when one association is broken another may persist and be put to use. And in this connection mention may be made of cases, now several in number, of persons who, though unable to read letters which they saw, were at once able to name these letters correctly when they traced the shape of the letter with the finger, the muscular sense thus acting for sight and arousing the idea, and enabling the person to read, though he had true "blindness of mind."[2]

All of these facts are of great interest, not only as throwing light on mental processes of association, but as indicating that those processes are carried on in definite areas of the brain and in tracts which connect those areas, and can be arrested by disease in these parts; for in some of these forms of apraxia the situation of the lesion has been found. It is as yet too soon to make definite statements regarding such lesions. It seems likely that they lie in that *terra incognita*, the

[1] A patient of my own, suffering from pure motor aphasia and agraphia, but without any paralysis, had lost her power of playing on the piano though she could read and enjoy music. She could sew perfectly well.

[2] See Bernard: "De l'Aphasie."

white matter beneath the cortex. And if the problem of the differentiation of cortical from subcortical lesions is ever to be worked out, it will only be after a number of cases, thoroughly examined as to all these processes, have been recorded, with autopsies to supplement the clinical record.

One fact, however, of great interest, not previously mentioned to my knowledge, has been brought out by a study of the cases of apraxia which I have been able to collect and which are here tabulated, namely that the lesion has always been found in the left hemisphere in right-handed and in the right hemisphere in left-handed persons; in other words, in the same hemisphere lesion of which has produced aphasia. It is true that apraxia, like aphasia, has been noticed in cases of paretic dementia in which the disease is diffuse and is situated on both sides. This fact would not, however, imply that a unilateral lesion alone was insufficient to produce the symptom, and the nine cases with autopsy here brought together for the first time seem to establish the statement made.

Apraxia has as many varieties as there are organs of sensation. The variety which existed in the nine cases tabulated was that of psychical blindness, and accompanied the form of aphasia known as word-blindness. In all the cases the lesion involved the cortex of the supra-marginal and angular gyri, or the tracts posterior to these parts in the white matter beneath them. It is probable, from the association of psychical deafness with word-deafness, that it is due to lesions in the upper temporal convolution, but autopsies are wanting. It is noticeable, however, that this symptom of apraxia is always associated with some form of aphasia. Hence all cases of aphasia should be examined for apraxia. It is well known that lesions in the right hemisphere in right-handed, and in the left hemisphere in left-handed persons do not produce aphasia. In the accounts of cases of such lesions, I have not been able to find any account of the discovery of any form of apraxia. It seems, therefore, that at present we must locate our memory-pictures of objects on one side of the brain, and on the same side which presides over speech. This appears to be very

TABLE I.—*Cases of Apraxia with Word-Blindness.*

No.	Author.	Reference.	Power to recognize objects seen.	Power to read.	Power to write.	Power to understand speech.	Power to speak.	Lesion.	Location in left hemisphere.
1	Bateman.	On Aphasia, 1870.	Impaired.	....	Lost.	Good.	Fair; misplaced words.	Cyst and softening.	P_2, A.
2	Ball.	Arch. of Med., 1881.	Impaired.	Impaired.	Impaired.	Impaired.	Fair; misplaced words.	Softening.	P_2, T_1.
3	Claus.	Irrenfreund, 1883, p. 82.	Impaired.	.....	Lost.	Impaired.	Good.	Softening.	Within T-O lobe.
4	Amidon.	N. Y. Med. Jour., 1885, p. 113.	Impaired.	Lost.	Lost.	Lost.	Good.	Softening.	P_2, A, T_1, T_2, O_2.
5	Seppilli.	Functions Localization, p. 182.	Impaired.	.....	.....	Lost	Talked jargon.	Softening.	Entire T-O lobe.
6	Monakow.	Arch. f. Psych., 1885, xvi., 166.	Impaired.	Lost.	.....	Lost.	Good.	Softening.	Within T-O lobe.
7	Bernheim.	Hecht. Thèse de Nancy, 1887.	Impaired.	Lost.	Lost.	Good.	Good.	Softening.	Within O lobe, also O_1, O_2, and A. (Right hemisphere, pat. left-handed.)
8	Laquer.	Neurol. Centralbl., 1888, p. 340.	Impaired.	Lost.	Never learned.	Good.	Talked jargon	Softening.	T_1, P_2, O_2.
9	Macewen.	Brit. Medical Jour., August 11th, 1888.	Impaired.	Lost.	....	Good.	Good.	Pressure.	P_2, A.

TABLE II.—*Cases of Pure Word-Blindness without Apraxia.*

No.	Author.	Reference.	Power to recognize objects.	Power to read.	Power to write.	Power to understand speech.	Power to speak.	Lesion.	Location in left hemisphere.
1	Broadbent.	Med.-Chir. Trans., 1872.	Good.	Lost.	Good.	Good.	Good.	Softening.	Under T_1 and A.
2	Jastrowitz.	Centralbl. f. Augen., 1877, p. 284.	Good.	Lost.	....	Good.	Good.	Softening.	O lobe.
3	Henschen.	Neurol. Centralbl., 1886, p. 424 (2).	Good.	Lost.	....	Impaired.	....	Softening.	A.
4	Hun.	Amer. Jour. Med. Sci., January, 1887.	Good.	Lost.	Lost.	Good.	Slightly impaired.	Softening.	P. C., P_1, P_2, A.
5	Wilbrand.	Seelenblindheit, 1887, p. 180.	Good.	Lost.	Never learned.	Good.	Good.	Softening.	Within T-O lobe.
6	Sigaud.	Progrès Médical, 1887, p. 177.	Good.	Good.	Lost from lack of power to recall appearance of words.	Good.	Good.	Softening.	P_2, A.

NOTE.—P. C. = posterior central; P_1, superior parietal; P_2, inferior parietal, including supramarginal gyrus; A, angular gyrus; O, occipital gyri; T, temporal gyri.

strange, since the bilateral connection of each sensory organ with both hemispheres is proved. There appears to be no reason why the appearance of things, their sounds, tastes, odors, etc., should not be received and remembered in both hemispheres instead of in one. But, as a fact, they are not. And the fact remains as mysterious as the corresponding fact regarding speech-memories.

APHASIA AND ITS VARIETIES.

Turning now from this investigation as to the condition of the memory-pictures of objects in the brain which together form the idea of a thing, let us look at the second set of memory-pictures which make up the word-image. As already stated, the word makes the idea available for thought and for its communication. The word "pen" or "bell" brings to a focus a number of various sensory images. But the word itself, in its ordinary use among intelligent persons, involves several of the senses. Words are heard, read, spoken, and written; and each of these processes goes on in its own brain-area, in which area, therefore, distinct residua of the process or memory-pictures of the word lie. Hence each word is composed, as far as its own image goes, of four separate memories, each of which may be lost separately.

1. If the memory of the sound of the word is lost, the word cannot be called to mind and cannot be recognized when heard. Show the patient a watch and he is unable to name it; tell him it is a stone, a match, a watch, and notice whether he dissents from the former and gives signs of satisfaction at the last. If he does, he has only auditory amnesia but not word-deafness. If not, he is word-deaf, and is unable to understand what is said to him. The lesion lies in the posterior half of the first and second temporal convolution in the left hemisphere in right-handed; in the right, in left-handed persons[1] (Fig. 21). This is one variety of aphasia whose lesion is well known, though it rarely occurs alone.

[1] I have collected 50 cases of pure sensory aphasia with autopsies on which this statement is based. Brain, part xlv., July, 1889.

2. If the memory of the appearance of the word is lost, the visual image of it cannot be called to mind or recognized, and then the patient will be unable to write spontaneously, for he cannot remember how the letter looks which he wishes

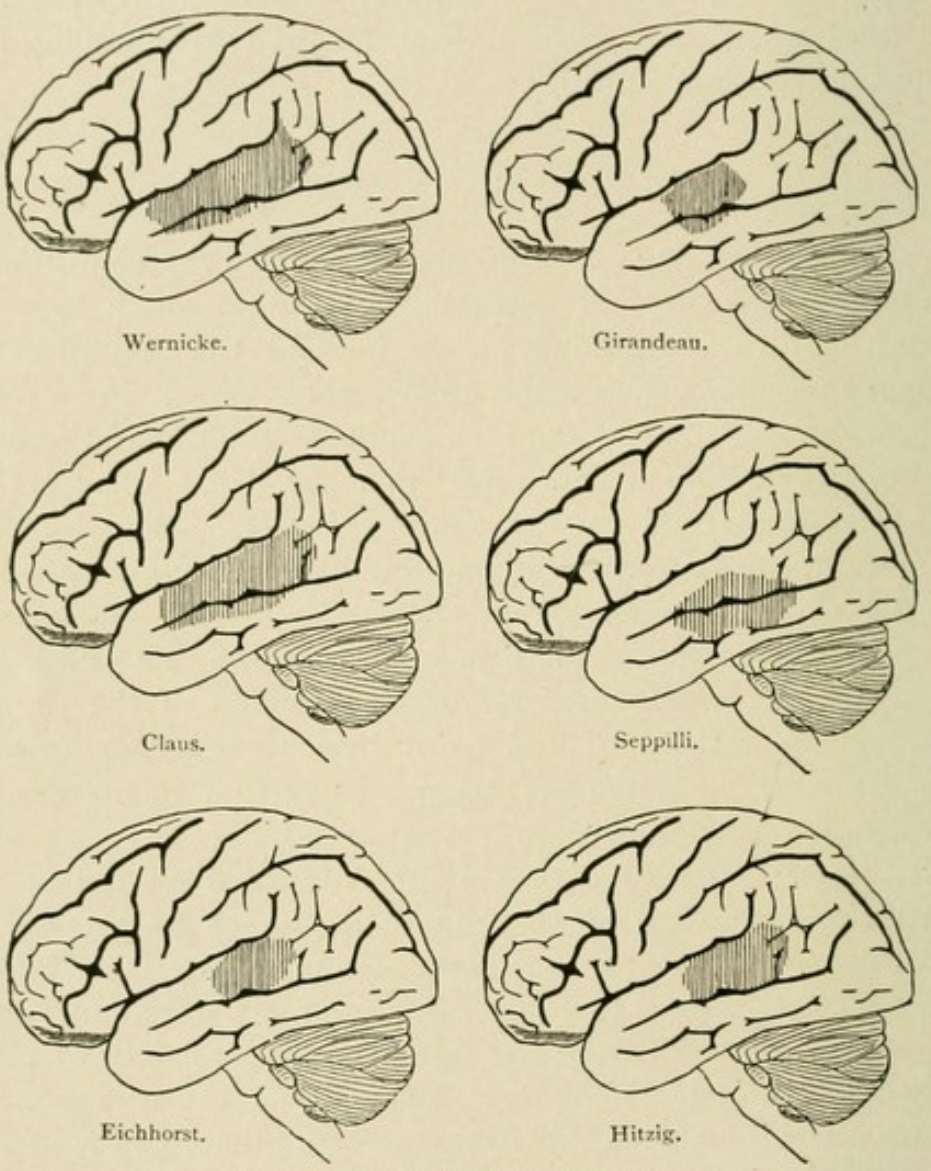

FIG. 21.—The Situation of Lesions causing Word Deafness only.

to write; and he will be unable to read because the shapes of the letters and words seen arouse no recollection. As a matter of fact, words are forgotten more easily than letters, and if a patient is to relearn to read he must begin with letters and go on to words. Figures are sometimes recalled when

words are forgotten, and many a patient can do mathematical calculations on paper who cannot read or write ordinary words. I have seen a case in which the reverse was true: the patient being able to read and write, but being unable to understand figures or calculate. Such patients may also play cards

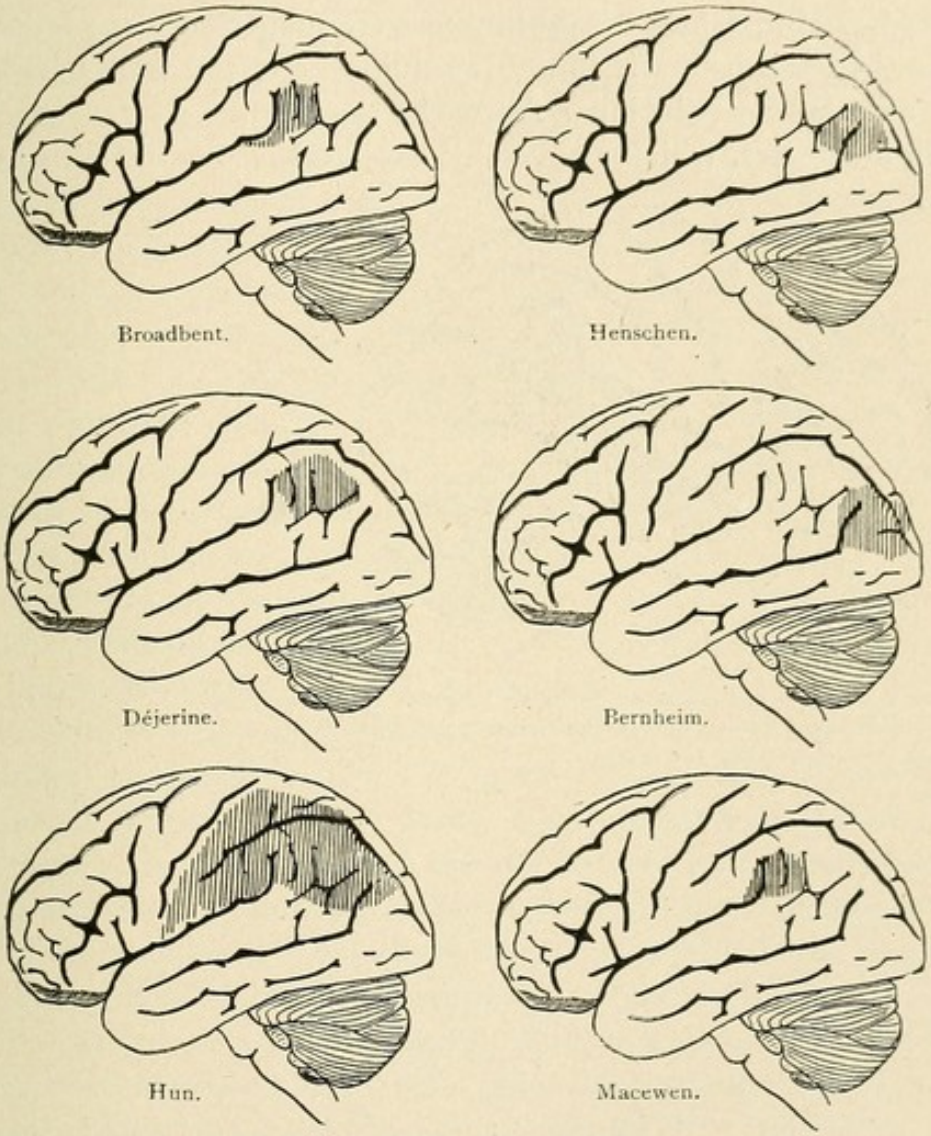

FIG. 22.—The Situation of Lesions causing Word Blindness only.

or other games if they are not psychically blind. It is not infrequently the case that persons who are thus word-blind can read aloud, can write at dictation, or copy, and yet show no evidence of understanding what has just been read or written. A distinction must be made between those who can and cannot do

these things, though its pathological basis is still obscure. The condition of visual amnesia with word-blindness is due to a lesion involving the inferior parietal convolutions and angular gyrus, and is often associated with psychical blindness, but may occur independently of it (Fig. 22). It was present in all the cases of psychical blindness enumerated in Table I. Word-deafness and word-blindness frequently occur together, and then the lesion is found involving both the temporal and the angular convolutions. About forty cases are now on record, with autopsies, which prove this assertion. Two of the cases here recorded illustrate this combination.

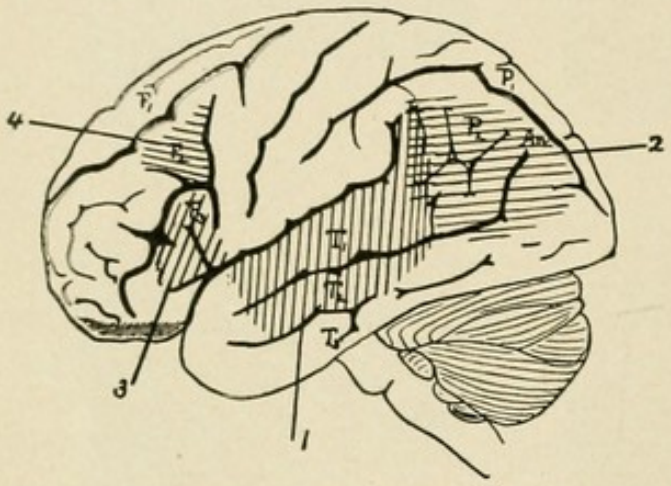

FIG. 23.—The Situation of Lesions causing Aphasia. 1, Lesion of word deafness; 2, lesion of word blindness; 3, lesion of motor aphasia; 4, supposed lesion of agraphia.

3. If the memory of the effort needed to pronounce a word is lost, a true paralysis of active speech occurs, though the muscles may not be weakened. This is the ordinary form of motor aphasia, due to a lesion of Broca's centre (Fig. 23, 3). It is to be noted that such a loss of speech involves a loss of the power of repeating words after another, as well as of voluntary speech, and is not accompanied by any inability to understand spoken or written language. In the uneducated, as in children, the acts of talking and writing are closely joined, as may be seen by watching the lips, which move in the act of writing. But among those accustomed to write much these acts are independent, and it is probable that many educated aphasics may be able to answer questions in writing when their efforts at speech fail. Reading aloud will also be lost in motor aphasia, for here, too, the inability to articulate hampers the patient.

4. The independence of the effort-memory necessary for

writing and connected with movements of the hand from the effort-memories of speech has been alluded to. When these are lost alone the condition is known as agraphia. In such a state the pen cannot be used. Copying, writing at dictation, and voluntary writing are all lost. It has been noted already that when a word cannot be called to mind it of course cannot be written. But words can often then be written at dictation, if the person is one who writes much. Hence sensory agraphia and motor agraphia must be distinguished; the former being a part of word-blindness, the latter not at all associated with inability to read. The lesion of motor agraphia is unsettled, though a few facts point to the posterior part of the second frontal convolution as the probable seat of this function (Fig. 23, 4). It is not unlikely, however, that the more exact localization of fine movements of the thumb and fingers in the posterior central convolution by Horsley may be followed by the discovery of the writing centre in this vicinity. As it is still unsettled, it becomes incumbent to test every patient as to his powers of writing voluntarily and at dictation and of copying.

These forms of aphasia are due to a loss of distinct memory-pictures. These several memory-pictures which are united in the word-image may thus be reasonably regarded as separate from one another in their location in the brain. But since they *are* joined together to form the word-image, it follows that the association-fibres joining the various areas are as necessary to the use of even a single word as the various areas with their memories. It is really by association only that an object or a word becomes a subject of thought or use. To a man who understands English only, the word "cloche," as seen or heard, conveys no idea. Form an association with that word by telling him that it is the French for "bell," and he at once possesses an idea. Or, to put it in another way, cut off from the auditory word-memory "bell" all its connections with sight, sound, feeling, and effort-memories—isolate it completely—and when the word bell is heard it no longer means anything. Such complete severance of association-fibres is the probable explanation of some cases of psychical

blindness and deafness, including word blindness and deafness. And when the condition is complete it will, of course, be impossible to distinguish between a loss of the memory-picture and an isolation of the memory-picture. The condition may, however, be incomplete. A man suddenly unable to read at sight is able to recognize letters by tracing them with his forefinger. What does this mean? It is evident that he has a visual memory of the letter, for he can, under certain circumstances, arouse that memory and recognize the word. But he cannot arouse it directly by sight. He has to do so indirectly through muscular sense. Is it not evident that the association of the visual memory-picture with the general visual area is cut off, but its connection with the muscular sense area is preserved; and so, by way of the latter, it can be reached? The association with the word-hearing and word-writing memories in these cases is commonly preserved, for the man could write at dictation or write spontaneously, yet was unable to read what he had just written. All the associations with that visual memory, then, were intact but one, and that one the one commonly used in reading, namely, from the common sight centre in the occipital lobe to the angular gyrus. Thus analyzing the condition subjectively we reach the conclusion that in such a case some defect in definite associations was present. In four such cases now on record a deep-seated lesion within the occipital lobe underneath the cortex has been found after death,[1] lying in such a position as inevitably to affect seriously the association-fibres passing to the angular gyrus.

Again, it has been noted that the power to recall the name of objects was often lost when the patient was able to recognize that name when heard. It is in a serious and permanent degree the same trouble which we are all liable to when we fail to recall the name of a man whom we know. In such a condition of auditory amnesia the memory or mental echo of the word is not lost, because when the word is heard it is recognized. The memory-picture remains, but is inaccessible by the ordinary way. The sight of the object does not call it

[1] Wilbrand: "Seelenblindheit," p. 180; Claus: Irrenfreund, 1883, p. 82; Déjerine: Progrès Médical, July 31st, 1883; Broadbent: Med. Chir. Trans., 1872, p. 162.

up. But, in such a case, if the object be handled or smelt or tasted—or heard, if that is possible—the name may come to mind. Thus the voice of the man may cause us to recall his name when his face has not done so. In other words, if one association-tract is impervious others may be open, and before it can be stated that the memory-picture is lost all the ways to it must be tried. The theory that auditory amnesia is due to lesion in the association-tracts from the occipital to the temporal convolutions, in distinction from word-deafness due to lesion in those convolutions, has a pathological basis. There are eight cases on record with autopsies in which this effect has resulted from a lesion of the occipital-temporal association-tract.[1] In collecting facts we are met with the difficulty that previous examinations of cases have been incomplete even when autopsies have been secured, hence the importance of more careful examination of patients with aphasia.

5. There is one form of aphasia in which the lesion is known to affect a great association-tract, viz., paraphasia, or the aphasia of conduction of Wernicke. In this condition the word-hearing and word-uttering process is perfect, but their connection is broken. The patients can understand what is said to them and can pronounce many words well. But they misplace words, use one word for another, *e.g.*, call, as one of my patients did, the card four of spades the "five of telephone." Such patients may even talk jargon. In such a case it is impossible for the person to repeat a word after another, yet he may be able to speak the word, like the patient who, after trying in vain to say no at the request of the physician, finally gave it up and, shaking his head sadly, said, "No, no." The lesion in these cases usually involves the island of Reil and severs the fibres beneath it which join the temporal and frontal lobes with one another.[2] This power of repetition should, therefore, be tested invariably. A somewhat similar condition of defective writing and copying, in

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[1] See Brain, part xlv., l. c.

[2] Cases of Wernicke, Aphas. Sympt. Compt. 1874; Déjerine: Revue de Méd., 1885, p. 174; Starr: Amer. Jour. of the Med. Sci., 1884, Case 99.

which words are misspelled or are unintelligible, has been recognized clinically, but its lesion is not yet known.

It is, therefore, evident that the processes of association should be most carefully tested. Such acts as reading aloud, writing at dictation, or copying, may be mentioned as involving the function of a few of the association-tracts, and these should be investigated, as well as those already named.

In many cases of aphasia the symptoms are not such as to point to one of these pure forms of aphasia; a combination of the characteristics of these forms will be found. The patient then has a mixed form of aphasia in which both sensory and motor elements are combined. But in such cases a thorough examination will demonstrate that one or the other pure form is present in excess, and this will indicate in which direction the lesion lies. The fact that all the various speech areas are supplied with blood by the middle cerebral artery, and the frequency with which an embolus lodges in this artery instead of plugging a small branch of it, explains the frequency of mixed aphasia and the rarity of pure forms—which are due to small areas of softening.

To examine an aphasic thoroughly it is necessary to test—

1. The power to recognize objects seen, heard, felt, tasted, or smelt, and their use.

This will determine whether the condition of apraxia is present.

2. The power to recall the spoken name of objects seen, heard, handled, tasted, or smelt.

3. The power to understand speech and musical tunes.

4. The power to call to mind objects named.

This will test the integrity of the auditory speech area and of the association-tracts between other sensory areas and the temporal convolutions.

5. The power to understand printed or written words.

6. The power to read aloud and to understand what is read.

7. The power to recall objects whose names are seen.

8. The power to write spontaneously, and to write the names of objects seen, heard, etc.

9. The power to copy and to write at dictation.

10. The power to read understandingly what has been written.

These tests will determine the condition of the visual word-memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

11. The power to speak voluntarily, and if it is lost, the character of its defects.

12. The power of repeating words after another.

This will test the integrity of Broca's centre and its association-tracts.

The practical application of the localization of lesions in aphasia hardly requires more than a mention. It is obvious.

The regions of the brain concerned in speech are especially accessible to the surgeon, and experience has shown that subcortical tumors and abscesses are as open to operation as cortical lesions.[1] It is evident that in cases of sensory aphasia the trephine should be applied, not over Broca's centre in the frontal region, but over the temporo-parietal region; in word-deafness over the posterior temporal region; in word-blindness over the angular gyrus; in both combined over the inferior parietal region, especially if verbal amnesia is present.

When aphasia is purely motor, the understanding of language being preserved but the power of talking being lost, the lesion is in Broca's centre and the trephine should be applied over the posterior part of the third frontal convolution, as in Case VI., already described.

The lesion of agraphia is not yet so absolutely determined as to warrant its surgical treatment when it is the only symptom present.

The following cases illustrate some of the varieties of aphasia here described, and present interesting features:

CASE X.—WORD-DEAFNESS AND WORD-BLINDNESS.

S. B., aged sixty-five, right-handed, was suddenly seized with aphasia during the night of April 20th, 1888. His wife on

[1] See cases of Seguin and Weir: Amer. Jour. Med. Sci., 1888; Ferrier and Horsley: Brit. Med. Jour., 1888, i., p. 530; Roswell Park: Trans. Cong. Amer. Phys. and Surg., 1888.

waking found him on the floor, acting strangely, saying, "What is the matter with me?" and in distress, but conscious and not paralyzed. For the next three days his wife thought that he was out of his head, for he paid no attention to what she said, and could only say one word—"hook." Then the use of words began to return, and he talked better and better. He complained of numbness in the right side, and said that he could not understand her. He seemed himself, but she was unable to get him to answer questions, as he could not comprehend what she said to him. His general health has been about as usual, but he has kept quiet, as going about among people bewilders him. He is always worse in the morning on rising.

Examination on May 14th failed to reveal any sensory or motor disturbance, excepting a slight deviation of the tongue to the right. Reflexes equal on both sides; pupils equal and react normally. The existence of hemianopsia could not be determined, as he could not be made to understand what was sought, and experiments were ambiguous in results, as he was very watchful of all tests. Hearing good; central vision good. Loud aortic direct murmur, with hypertrophy and dilatation of the heart, which was rapid and irregular. The aphasia was tested carefully. As far as the power of articulation and of the spontaneous use of words was concerned he had no loss. He talked freely and rapidly when he could be made to talk at all, and often made remarks of his own accord; *e.g.*, while I was testing his sensation to cotton, he said, "I feel you touch me, if that is what you want to know." As far as the power of writing spontaneously or handling the pen was concerned there was little loss, for he wrote his name clearly. He therefore did not present the symptoms of motor aphasia nor of agraphia.

His power of understanding spoken or written language was, however, almost entirely abolished. He had a bewildered look, and turned to his wife constantly for aid when spoken to, though he did not seem to understand her any better than me. To all such commands as "Shut your eyes," "Show your tongue," "Stand up," "Sit down," "Put on your glasses,"

he remained wholly indifferent, listening attentively, but not obeying until the idea was conveyed by gestures, when he at once understood and acted accordingly. To ordinary questions he gives no appropriate answer; sometimes, however, when he sees that a reply is awaited, he says, "I don't understand you;" "I would answer you if I knew what you wished to know." The only method of arousing any comprehension is to repeat a single word over and over, when he sometimes understands. Thus when the word "name" is repeated he at last seems to get the idea, and says, "Oh, my name; yes, my name is S. B." To several subsequent questions he gives the same reply, and seems puzzled that I should ask, as he supposes, the same question again. The word "age" repeated, elicits the answer, "Sixty-five, sixty-seven," and he appears uncertain as to the exact number. When asked his address he does not reply, but when it is repeated gives his name again, then his age, and only when the words "Long Island" are repeated several times does he get the idea. Then he gives his full address at once correctly. There is, therefore, almost total word-deafness. His auditory memory of the names of objects is also impaired, for when various objects were shown him he failed to name any of them, though after several attempts he occasionally succeeded. He appeared much perplexed at his failure to name objects, and so much distressed that tests were not pursued. The power to recall names, as well as the power to recognize words, was therefore much impaired. Since it was impossible to obtain replies to spoken questions, written questions were put to him. And this elicited a remarkable symptom. He read to himself or aloud all that was written, easily and rapidly, but failed utterly to understand what he read. To very few of the written questions did he show any evidence of intention to reply. In one or two, a single prominent word read several times aloud awakened an idea; thus, "When did this begin?" was answered by, "I don't know whether I can tell you; it was the twentieth of April, I think." "Where do you live?" he read three times aloud, and then answered by giving his name. Other questions were equally lost to his understand-

ing. It was evident that word-blindness as well as word-deafness was present, and in about an equal degree. This condition of word-blindness appeared to prevent his writing freely. The only thing he could be got to write was his name; and this he wrote again and again when attempts were made to make him write at dictation or copy. In fact, he could do neither of the last two even when the idea was conveyed to him by gestures. He succeeded a week later in copying a few figures, but was unable to add them up, though formerly a good accountant. The word-blindness was therefore associated with visual amnesia.

The condition was therefore one of pure sensory aphasia, word-deafness and word-blindness being combined, but no disturbance of utterance, no misplacing of words, no hesitancy in reading aloud being present.

The diagnosis of embolism of the terminal branches of the left Sylvian artery, producing anæmia and loss of function in the first temporal and angular convolutions and the intervening cortex, seemed justifiable in view of the existence of a cardiac obstruction at the aortic valve, and in view of the post-mortem findings in similar cases in the brain (Fig. 23, 1, 2).

During the following months he improved slowly but steadily, the irregular cardiac action being corrected by the use of digitalis. He soon recovered the power to name objects and to write after a copy, but was still unable, in July, to write at dictation or to add and divide. His wife thought that he understood many things he heard, but she admitted that he did not comprehend what he read, for he had given up reading his Bible on this account. It was evident that he understood much that was said to him. Thus it became possible to test his visual field, which was found to be intact on all sides though slightly contracted in the right eye. Ophthalmoscopical appearances were normal from the first.

In October he was very much better. He now understood what was said to him, if it was said slowly, but it was difficult for him to follow conversation, as the change from one topic to another confused him. He would go on talking on a previous subject even when questions on a new one were asked,

and his wife says that she has to indicate to him that the subject is changed by clapping her hands. He is able to name objects quickly and correctly and speech is as fluent as ever. He reads without hesitation aloud and says that he understands. His wife says that he now reads the papers and tells her the news. So she is sure that he does understand what he reads if he reads very slowly. Rapid reading, like rapid speaking, he is still unable to follow, and for this reason he cannot yet go to church.

His writing is still, at the end of two years, imperfect. He figures and calculates perfectly, but makes mistakes in writing at dictation and in copying. During an attempt to write he stopped and said, "I can't think how that letter looks." It was a capital H which he wanted; and a little later in the test, when a word beginning with it was given him to copy, he failed to make it properly, writing Nurrison for Harrison. And it is this lack of memory of the appearance of words which prevents his writing with any freedom. There is therefore still a moderate degree of sensory aphasia, the word-blindness being now more prominent than the word-deafness.

CASE XI.—WORD-DEAFNESS AND WORD-BLINDNESS; TEMPORARY APRAXIA.

T. D. C., aged forty-two, right-handed, suddenly became unconscious on March 11th, 1888, and on coming to himself a few minutes later felt bewildered, did not recognize his surroundings or his wife or father, and could not talk intelligibly or understand what was said to him. He therefore presented symptoms of aphasia with psychical blindness. His alarm led him to desire to see his physician, Dr. Walker, to whom he at once wrote the following note, which he apparently read over after it was written, and sent off with the idea that it was a request for an immediate call. The note is written in his natural handwriting and reads: "Please person me personally per me Dr. personally me personally me. My love. T. D. C." Dr. Walker saw him on the following day and found a condition of almost total aphasia. He tried to talk, but could not be understood, the words used being discon-

nected. He said, "Crazy, I am crazy!" and he was unable to reply to questions, as he could not understand anything said to him. He was slightly paralyzed on the right side of the face, not elsewhere, and he had pain over the left eye and in the forehead and head on the left side. For a week this condition remained and then he began to improve rapidly, recognized his family and friends, regained the use of words, and on March 24th wrote a letter of some length quite correctly to Dr. Walker. I saw him March 25th. He then talked freely and rapidly, so that no one would have noticed any difficulty of speech, articulation being distinct and words used correctly. He was able to write spontaneously freely, but has difficulty in writing at dictation and in copying. The words dictated had to be repeated if more than three words were given, and in copying he scanned closely each letter and word. But writing thus slowly he made no mistake. There was, therefore, no motor aphasia or agraphia.

There was, however, marked impairment in his power of understanding what he heard or read. My questions were only understood when spoken very slowly and repeated, and any sudden change of subject caused evident bewilderment. His wife had found it very difficult to make him understand, and as she spoke to him in French it was evident that this, as well as his English memory of words, was impaired. He had been to the theatre and occupied a front seat the day before I saw him, but had been unable to understand the words of the play, so that, as he said, "The actors might as well have spoken in Greek." Unfamiliar words were not as readily understood as common ones, and the bewilderment increased as he was examined; so that at the end of half an hour he did not understand as quickly as at first. He was able to repeat single words after me, but if more than two or three were given him at a time he failed, and in a long phrase only occasional words were remembered. His recollection of familiar sentences—*e.g.*, "Peter piper"—was, however, good. The same trouble was noticed in writing at dictation as in repetition. He recalled the name of various objects shown to him readily. He therefore had partial word-deafness.

When asked to read he did so to himself and aloud accurately. But he only understood short sentences, and if long ones were read through they conveyed no idea to him. He was spending most of the time in reading, but confessed that he only got an indefinite idea of what he read. The single words, if dwelt upon long enough, were understood, but when combined into sentences were not fully comprehended. He remembered having read the note to Dr. Walker, and supposed it was written correctly. When given paragraphs of a newspaper to read he was never able to tell subsequently the contents of the paragraph when he had read it rapidly. In writing from a copy he could only recollect the appearance of a word or two of the copy and had to consult it frequently. There was therefore partial word-blindness.

Hearing and sight were perfect, the visual field being equally extensive in both eyes and normal. No trace of the psychical blindness remained. He had a systolic murmur, heard at the apex and conveyed to the back. The lesion in this case was probably like that in Case X. He did not recover from his sensory aphasia, but went abroad, and died in Paris of acute encephalitis about the middle of April.

In both of these cases the power to speak, the power to write spontaneously, the power to read aloud were preserved; the power to understand spoken or printed language was lost, and the power to repeat sentences, to write at dictation, or to copy was impaired. In both, the power to recall the names of objects seen was much better than the power of recognizing those names when heard. In both, the things which were read aloud correctly were nevertheless not comprehended. In both, the condition present was pure sensory aphasia without any affection of articulation, or of the use of the pen; and in both a very marked degree of recovery had followed the attack. In both, the word-deafness and word-blindness were about equally marked. In neither was motion, sensation, sight, or hearing markedly impaired.

CASE XII.—PARAPHASIA PROGRESSING TO TOTAL APHASIA.

H. A. T., aged fifty-four, right-handed, after a severe mental strain gradually developed a difficulty of speech during a period of three weeks preceding the time when I saw him, in consultation with Dr. O'Dwyer, on January 9th, 1885. He first misplaced words, using one word when he meant to use another, both in speech and in writing, and this increased until his speech became almost unintelligible. At the same time mental failure was thought to be present, as he seemed less able to understand what was said to him than formerly, and had forgotten many words and names. On examination it was found that he understood fairly well all words and short sentences, but failed to follow long sentences or complex orders. When told to walk, to stand still, etc., he did so; but when told to pick up a pack of cards and put them on another table, he did not catch the idea until it was repeated slowly. His power to recall the names of objects was very much impaired, but he dissented from wrong names and recognized the right names when told. He often recognized the fact that he had not said the word which he meant to say, but he did not succeed in this always; so that he often became indignant with his wife because she failed to understand him. He could not recall the names of presidential candidates, of the day of the week or month; and even when told he forgot them at once. There was, therefore, considerable auditory amnesia and word-deafness. There was also some word-blindness. He read single words, but sentences seemed to convey no idea. When he attempted to read aloud he uttered jargon. When he attempted to write, either voluntarily or at dictation, or after a copy, he only succeeded in making a series of badly-formed letters, few words being properly spelled and the result being illegible. Though an expert accountant, he failed to add or to subtract correctly, putting down and saying out loud the wrong figures every time. There was evidently a considerable degree of word-blindness. That his mental powers were not impaired was evident from the fact that he would not allow me to take the paper on which these attempts at

writing had been made until I tore off and gave to him the part containing his name.

His power of articulation was good, words being well pronounced and his speech rapid and fluent. Like the power of handling the pen, the power of forming words was unimpaired. There was no motor aphasia and no motor agraphia.

But the chief defect consisted in a substitution of some word for the one which he wished to use. Thus when asked to name the card of a pack with which he had been playing, he called the four of spades "five of telephone;" "trying to do" he pronounced "trayling to dool." His alphabet was said as follows: "a, m, w, x, w, m, x, y, w," the constant recurrence to "m" and "w" being noticed; he also substituted m for four in counting from one to ten. The days of the week were not said correctly, Friday coming next to Tuesday, but this he noticed and corrected after saying them all over from the first. He made mistakes in repeating words after me, as well as in talking and in reading aloud. This difficulty of speech became worse when he was excited, and then not a single sentence of a long harangue could be comprehended. This was especially unfortunate, as he was anxious to give directions regarding financial affairs and about his will; and though he knew what he wished, it was impossible for others to learn his desires, either by his speech or in writing.

Vision and hearing good; slight paresis of right side of face and tongue, none elsewhere; no anæsthesia; optic discs normal; no cardiac disease. Syphilis was suspected, but no history was obtainable, and specific treatment, on which he was placed, failed to modify the course of the disease, which progressed to total aphasia and right hemiplegia, and he died comatose on February 9th, 1885. No autopsy was obtained.

In this case a partial degree of word-deafness and word-blindness was accompanied by a very marked condition of paraphasia with paragraphia.

CASE XIII.—PARAPHASIA AND PARAGRAPHIA.

W. E. V., aged forty-five, right-handed, became unconscious suddenly in March, 1884, and on recovering conscious-

ness was unable to talk, had much tingling and numbness in his entire right side, but no paralysis. For a month he could only say, "How many," but then the use of words returned, and improvement has been steady until February, 1885, when I saw him. He is an intelligent physician and interested in his own case. He understands perfectly whatever he hears or reads, and has no evidence of psychical blindness or deafness, though for a few weeks after the attack he could not play cards or enjoy music as he had formerly done. His pronunciation and his power of using the pen are perfect, and his only complaint is that both in speaking and in writing he uses wrong words. In talking he chooses his words carefully and speaks slowly, but makes occasional mistakes—*e.g.*, "when" for "well," "lant" for "lance," "misplate" for "misspell." He says his alphabet and counts perfectly. He says that when unable to say the word he is thinking of, if he calls up to his mind the appearance of the word as written he can usually pronounce it correctly; and it is found that he makes fewer mistakes in reading aloud than he does in talking. In writing he often misspells words, *e.g.*, "unforable" for "unfavorable," "sencotts" for "senate," but he copies freely, remembering several words easily, and he writes well at dictation. He says that he has sometimes written the name of one drug in a prescription for another, and that he is also apt to write the quantities wrong, so that he never fails to read his prescriptions several times. He finds much difficulty in keeping accounts, addition and subtraction being very much impaired, and the results being very apt to be wrong. The condition is therefore one of paraphasia with paragraphia. He has cardiac disease, which makes it probable that the lesion is embolic in origin. Vision and hearing unimpaired. Six months subsequently a slight improvement had taken place, but he never recovered entirely, and died of cerebral embolism three years later.

Such an uncomplicated condition of paraphasia is rare, and it is especially interesting to notice that conduction of impulses from the word-hearing centre to the word-uttering centre was more interfered with than from the word-seeing

centre to the word-uttering centre. He therefore got over the obstacle by calling to mind the appearance of the word, and then could say it when the recollection of its sound was insufficient to initiate the movement. The lesion therefore affected the association-tract between the temporal and frontal lobes more seriously than the one between the angular gyrus and the frontal lobe.

For a good illustration of the symptoms in pure motor aphasia, the reader is referred to Case VI., already described, in which the condition was relieved by a surgical operation.

CHAPTER VII.

THE CORTICAL REGIONS WHOSE FUNCTION IS UNDETERMINED.

The frontal convolutions; mental defects.—The parietal convolutions.—The temporo-sphenoidal convolutions.—The uncinate convolution and the sense of smell.—The median cortex of the hemispheres.

THE REGIONS OF THE CORTEX WHOSE FUNCTION IS UNDETERMINED.

IF diagrams of the two hemispheres are prepared in which the motor, visual, and speech areas are laid down (Fig. 24), it will be seen that there remain extensive regions of the cortex to which, as yet, no function can be ascribed.

There is (1) the frontal region, including all the cortex of the frontal convolutions, except the posterior half of the third frontal on the left side.

In lesions of this region mental symptoms are quite likely to be prominent. These do not conform to any type of insanity. They are rather to be ascribed to a loss of self-control and a decided change of character. The mind exercises a constant inhibitory influence upon all action physical or mental; from the simple restraint upon the lower reflexes, such as the knee-jerk and the action of the sphincters, to the higher control over what may be termed psychical reflexes, such as emotional impulses and their manifestation in expression, speech, and conduct. This action of control implies the recognition of the import of an act in its relation to other acts, and involves the highest mental powers, judgment and reason. By inhibiting all but one set of mental acts, it enables one to fix the attention on a subject and to hold it there.

It is the possession of these mental powers which chiefly

distinguishes a normal man from an imbecile or an idiot. And the brain of a normal man differs from that of the lower animals and from idiots chiefly in the greater and more perfect development of the frontal lobes. It seems probable, therefore, that the frontal lobes have some necessary relation to the highest mental powers. In about half of the cases on record of destruction of the frontal convolutions by disease, the patients have manifested mental symptoms, such as an inability to fix the attention, to follow a continuous train of thought, or to conduct higher intellectual processes; to exercise the power of judgment and reason and to maintain that self-control which regulates conduct. Various forms and degrees of dementia have been observed after such lesions.[1] And when such mental symptoms are present, the diagnosis of lesion in the frontal lobes is warranted when lesions elsewhere can be excluded. Unfortunately such cases are not yet open to surgical treatment, for any precise localization of functions in the large frontal regions is impossible; and in the absence of symptoms of aphasia or paralysis we have not even a clue as to which hemisphere is diseased.[1]

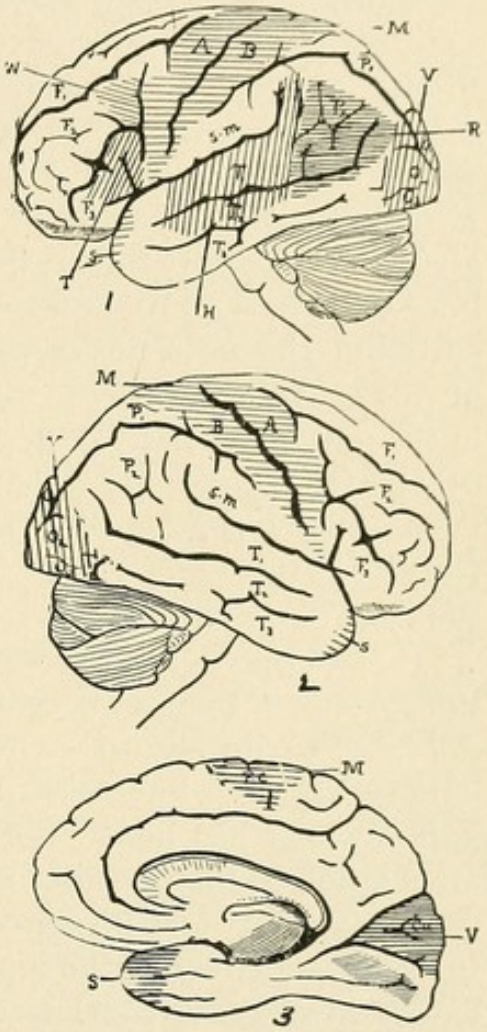

FIG. 24.—The Unknown Areas. 1, Lateral surface of left hemisphere; 2, lateral surface of right hemisphere; 3, median surface of right hemisphere. *T*, Talking; *W*, writing; *M*, motor; *V*, visual; *R*, reading; *H*, hearing; *S*, smelling areas. The functions of the unshaded areas are unknown.

[1] See "Cortical Lesions," Amer. Jour. Med. Sci., April, 1884, where 23 cases are collected. Compare the remarks on Psychical Epileptic Equivalents in chap. xvii.

There is (2) the region of cortex lying between the posterior central convolution and the occipital convolutions, and including the parietal convolutions except the left inferior parietal lobule.

It has already been stated, in discussing the motor area, that lesions of the posterior central convolution are more likely to produce disturbances of tactile and muscular sensations than lesions in the anterior central convolution alone. In 1884 I attempted to show that in the parietal convolutions were the centres of tactile sense, and Dana's collection of cases gives some support to this theory. I am now inclined to believe that the sensory and motor areas coincide, including the cortex of the posterior central convolution, and that lesions of this convolution are found to be associated with anæsthesia in the paralyzed part, rather because the motor and sensory centres for the parts in which the appreciation of sensations is most acute (viz., the thumb, fingers, and hand, the toes and feet) lie in this region, than because this region contains tactile centres only.

It is an open question whether the muscular sense perceptions occur in the motor centres, as I am inclined to believe. A few cases on record would indicate their location in the parietal region. But these cases are open to the same explanation which has been offered for the anæsthesia observed after lesion in the posterior central convolution.

It may therefore be stated that we do not know the functions presided over by the superior and inferior parietal lobules, except in so far as the latter on the left side is concerned in the memory of the appearance of printed words.

There is (3) the region lying in the entire temporo-sphenoidal lobe on the right side, and in the temporo-sphenoidal lobe on the left side, not concerned in speech. Abscesses in this region are very common after otitis media, and are often successfully operated upon. But the diagnosis in such cases has to be reached entirely by the existence of otitis, together with the discovery of the general symptoms of brain disease (see page 15); as no distinctively local symptoms are produced by lesions in this area. It is to this region that physi-

ologists assign the function of hearing. There are two cases on record, in which bilateral destruction of the three temporal convolutions has been followed by total deafness. But deafness of cerebral origin is an exceedingly rare symptom, and there is not as yet a sufficient number of facts to warrant any localization of the auditory sense, or to determine the exact functions of the temporo-sphenoidal convolutions. There are several cases on record of tumors in this area producing convulsions preceded by an aura of sound. These, however, do not prove this region to be auditory. The same lack of knowledge regarding the regions of the cortex lying on the base of the temporo-sphenoidal lobe must be admitted.

There is (4) the apex of the temporo-sphenoidal lobe, including the uncinate convolution (Fig. 24, S). To this region the olfactory tract has been traced, and in it physiologists located the sense of smell. Three cases are recorded which favor this localization, by Hamilton,[1] by Anderson,[2] and by Hughlings Jackson.[3] In Hamilton's case there were symptoms of epilepsy, and each fit was preceded by a sensory aura of a bad smell. The lesion was a meningitis and sclerosis of this convolution. In the other cases there were epileptiform attacks preceded by an aura of a bad smell, and this region was destroyed by a tumor. In neither case had smell been tested. The theory that this area governs the sense of smell has therefore some pathological facts in its favor, and though it is hardly allowable to draw a definite conclusion from three imperfectly observed cases in man, the conclusions of anatomical, physiological, and pathological evidence, taken together, make it probable that the sense of smell is perceived in the uncinate convolution.

There is (5) the entire median surface of the hemispheres with the exception of the paracentral lobule (motor) and the cuneus (visual). With regard to the functions of this region nothing is known (Fig. 24, 3). This includes the gyrus fornicatus and the hippocampal cortex, no cases being on record

[1] Allan McL. Hamilton : N. Y. Med. Jour., June, 1882.

[2] J. Anderson: Brain, 1886, p. 385.

[3] Brain, part xlvii., 1889.

to support the theory derived from physiological experiment that the tactile sense is located in the hippocampus.

When the extent of these regions is contrasted with the extent of those regions whose functions are determined, it is found that they include about two-thirds of the entire cortical area. It becomes evident, therefore, that as yet but a third of the cortex is open to surgical interference—since in this extent only can an exact diagnosis of a lesion be made.

The functions of the brain, whose situation is still undetermined, are hearing and taste.

CHAPTER VIII.

THE TRACTS WITHIN THE BRAIN AND THE DIAGNOSIS OF SUB-CORTICAL LESIONS.

The projection tracts; frontal; motor; tactile; visual; auditory.—The commissural tracts.—Mirror writing.—The association tracts.

THE TRACTS WITHIN THE BRAIN.

THE diagnosis of lesions involving the mass of white substance lying beneath the cortex of the brain and above the level of the basal ganglia has been hitherto almost impossible. Such authorities as Nothnagel,[1] Charcot,[2] and Strümpell[3] admit that few rules for guidance can be given, and at present there are few definite symptoms assigned to diseases of the centrum semi-ovale.

The subject of lesions of this part of the brain has an immediate practical bearing. In the journals of the past few years are recorded several cases of operations upon the brain for the evacuation of cerebral abscesses, and the excision of tumors, some of which were sub-cortical. This is, doubtless, but a beginning of a brilliant advance in the department of cerebral surgery. But unless the diagnosis of sub-cortical disease can be made with some degree of accuracy, such operations cannot be safely undertaken.

The natural manner of approaching a subject of this nature is to follow the inductive method, and, by a collection of cases in which an autopsy has demonstrated the existence of a lesion, to collate the common symptoms and thus arrive at

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[1] Nothnagel: "Topische Diagnostik der Gehirnkrankheiten," 1879.

[2] Charcot: Revue de Médecine, 1883, "Localisations cérébrales."

[3] Strümpell: "Lehrbuch der inneren Krankheiten," 1884.

the diagnostic conclusions. But while this method does afford a certain number of facts of importance, it is by no means as satisfactory as in the case of cortical lesions, in the investigation of which it has given such brilliant results. For lesions of the centrum ovale are not very common, and, therefore, are not often suspected during life. An examination directed toward the symptoms which such lesions might cause is therefore frequently omitted, and hence facts are wanting in the recorded histories which would serve to establish a symptomatology of these diseases. There are many symptoms in every case which must be sought for by the examiner, as they are of a character to escape the notice of the patient. Disturbances of the temperature sense, of vasomotor activity, of the power of locating the position of the extremities, or of the exact limitation of the visual field, are all instances in point. It is evident, therefore, that some of the symptoms of lesions in the centrum ovale may have escaped notice because they were not properly investigated. And it follows that we must ascertain the probable nature of those symptoms from other data than those furnished by the recorded cases.

It seems to have been a constant fact in the history of progress in clinical medicine that, just in proportion to our knowledge of the anatomy and physiology of a part, does our knowledge of the symptomatology of its diseases advance. To bring symptoms into connection with lesions is the aim of scientific investigation, and this has been done chiefly along the lines of anatomy and physiology.

Hence, in investigating lesions of the centrum ovale, we may turn from pathology to anatomy for the information needed to establish the symptoms of such forms of disease.

The anatomy of the centrum ovale can be studied to the best advantage upon torn brains which have been properly hardened in alcohol. A dissection made in this manner with care enables one to establish the fact that this part of the brain is made up of fibres only, and to distinguish easily three sets of fibres passing through it. These three sets have been named the *projection*, *commissural*, and *association* systems of fibres.

THE PROJECTION FIBRES.

The *projection* system includes those fibres which join a definite area of the cortex with parts of the nervous system lying below it. It therefore follows that but one termination of a projection fibre is found in the cortex, and hence the fibre, in passing through the centrum ovale, is either on its way to, or on its way from, some nervous mechanism in the basal ganglia, brain axis, or spinal cord. Indirectly, through the medium of such mechanisms, the external world is projected upon the brain and reaches consciousness, and voluntary impulses originating in the brain are sent to the muscles. A dissection of the centrum ovale, prepared so as to show the projection system, demonstrates the existence of fibres from the entire cortex passing downward and gathering together within the hemisphere at the upper level of the basal ganglia, and either ending in the optic thalamus, or going on between these ganglia to the brain axis and spinal cord.

The majority of these fibres end in the optic thalamus, which is thus connected with all parts of the cortex of the brain. They are not shown in Fig. 25. Of the function of these we know very little. Two large bundles, however, are separable from the mass. One of these passes inward and forward from the occipital lobe, and joins the pulvinar of the thalamus and the external geniculate body (Fig. 25, D). This is now known as the visual tract, and conveys impulses received by the optic thalamus from one-half of each retina to the like-named occipital lobe, as has been already described (page 51). A second bundle passes from the temporal lobe upward to the thalamus, internal geniculate body, and posterior corpus quadrigeminum, in which the auditory tract from the acoustic nucleus ends (Fig. 25, E). This probably conveys impulses of sound from both ears to each temporal lobe, as the investigations of von Monakow [1] and Babinsky [2] by the atrophy method, and of Spitzka [3] by the method of compara-

[1] Arch. f. Psych., xii. [2] Virchow's Arch., cv., 28.
[3] N. Y. Med. Journal, Sept. 18th, 1886.

tive anatomy have shown. These are the only bundles of the thalamic radiations whose function is determined.

Some of the projection fibres pass on through the internal capsule without communicating with the basal ganglia, and of these we know three distinct bundles.

The first is collected from the three convolutions of the

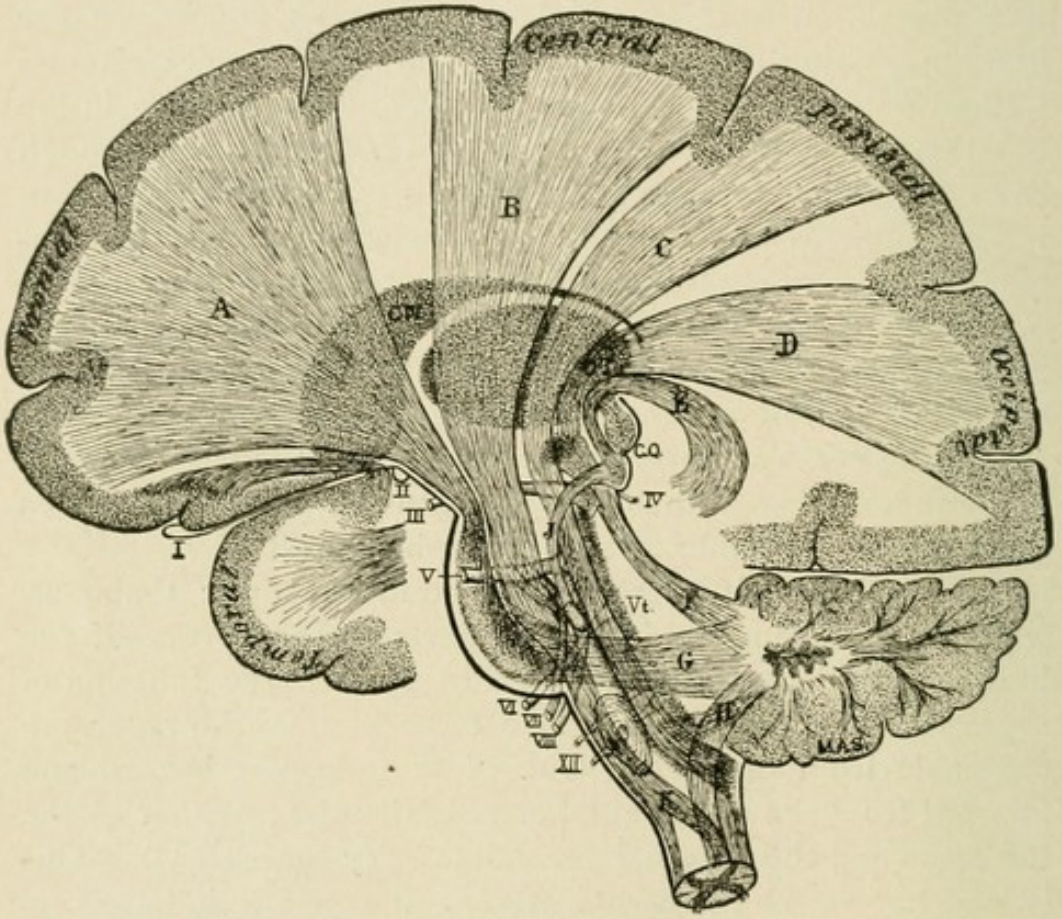

FIG. 25.—The Projection Fibres within the Brain. Lateral View of the Internal Capsule. *A*, Tract from frontal convolutions to pons nuclei thence to cerebellum; *B*, motor tract; *C*, sensory tract for touch; *D*, visual tract; *E*, auditory tract; *F*, superior cerebellar peduncle; *G*, middle cerebellar peduncle; *H*, inferior cerebellar peduncle; *J*, tract from auditory nucleus to corp. quad. inf.; *K*, motor decussation in medulla; *N*, red nucleus of tegmentum; *O*, olivary body; *O T*, optic thalamus: *C N*, caudate nucleus; *Vt*, fourth ventricle; numerals refer to cranial nerves.

frontal lobe, and passing between the caudate and lenticular nuclei in the anterior division of the internal capsule, descends in it to the base of the brain, and issuing in the inner third of the foot of the crus cerebri, passes down to the pons, where it terminates in nuclei lying in the ventral half (Fig. 25, A). The nuclei thus reached by these fibres are also joined by

other fibres from both hemispheres of the cerebellum, which enter the pons at its lateral surfaces in the middle peduncles (Fig. 25, G). Thus it is evident that a connection exists between each frontal lobe and both cerebellar hemispheres, the crossed connection being greater than the direct one. In Flechsig's case of deficient cerebellum, the pons nuclei and the fibres to them from the frontal lobe were found.[1] In my case of deficient cerebrum the pons nuclei and the fibres to them from the cerebellum were found.[2] Therefore each half of this tract, if it is a continuous tract, may develop independently of the other.

Of the function of this tract we know nothing, and of the functions of the frontal lobes and cerebellar hemispheres we know very little. Therefore we cannot as yet connect any symptoms with its destruction.

The second bundle of the projection system is the motor tract. It comes from the posterior part of the third frontal convolution, the two central convolutions, and from the paracentral lobule, and passes out of the base through the middle third of the crus cerebri. Its fibres collect at the middle portion of the upper surface of the internal capsule, those from the lower parts of the cortex passing straight inward, those from the upper parts curving outward and downward to pass around the side of the lateral ventricle. Thus within the centrum ovale these fibres, if looked at from in front (Fig. 26), appear like the sticks of a fan, and like those sticks their relative position is altered in the point of junction, where those passing inward from the lowest part of the cortex lie in front of those which pass downward from its upper part (Fig. 26). Thus in the capsule the order from before backward is, *first*, the fibres conveying speech impulses to the pons and medulla; *second*, the fibres conveying facial motor impulses to the pons; *third*, the fibres destined to the arm centres of the cord; *fourth*, the fibres transmitting impulses to the leg centres in

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[1] Flechsig: "Plan des Mensch. Gehirns," Leipzig, 1884.

[2] Starr: "Sensory Tract in Central Nervous System," Jour. of Nerv. and Ment. Dis., July, 1884.

the cord. The fibres conveying impulses to the muscles of the trunk probably lie behind those to the leg.

This is the great motor tract, whose course through the anterior half of the posterior division of the capsule is well known, and which can be traced through the middle third of each crus, through the pons (where the division to the facial

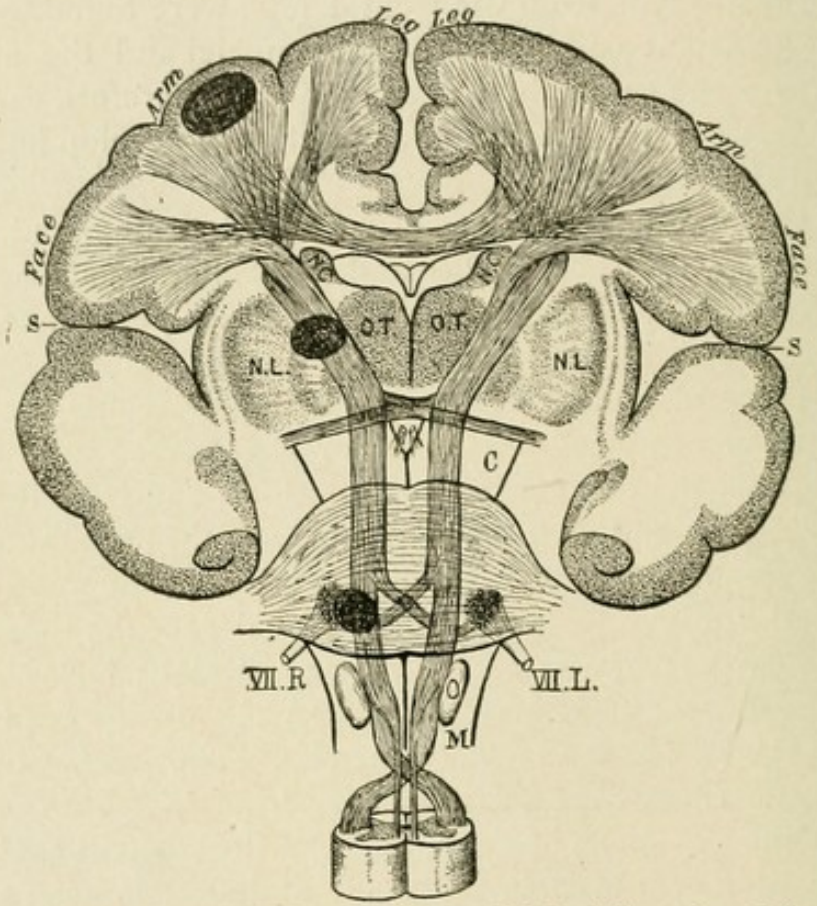

FIG. 26.—The Motor Tract. *S*, Fissure of Sylvius; *N L*, lenticular nucleus; *O T*, optic thalamus; *N C*, caudate nucleus; *C*, crus; *P*, pons; *M*, medulla; *O*, olivary body. The tracts for face, arm, and leg gather in the capsule and pass together to the lower pons, where the face fibres cross to the opposite VII. nerve nucleus, while the others pass on to the lower medulla where they partially decussate, to enter the lateral columns of the cord; the non-decussating fibres pass from the anterior median columns. The effect of a lesion situated at three points in the tract is shown on the left side of the figure; at *x*, *y*, *z*.

nucleus crosses to the opposite side and ends), and thence by way of the pyramids of the medulla to the crossed pyramidal and anterior median columns of the spinal cord. It is evident, however, that the concentration of this tract is much greater in the capsule than in the centrum ovale, where the individual fibres are scattered among the other systems and occupy but

a small area from before backward. Lesions beneath the third frontal convolution of the left side produce motor aphasia. Lesions beneath the central convolutions in the centrum ovale produce paralysis, which will vary according to the position of the lesion. The nearer the lesion lies to the cortex the more will the symptoms resemble those of cortical disease, monoplegia being the rule (Fig. 26, x). The nearer the lesion to the point of junction at the capsule the more will the symptoms resemble those of capsule lesion, hemiplegia being the rule (Fig. 26, y). There are numerous cases recorded which support this statement. When the motor tract is injured by a lesion in the lower half of the pons (Fig. 26, z) the result is paralysis of the face on the side of the lesion and of the arm and leg on the opposite side (alternating paralysis).

The third set of fibres of the projection system includes those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions (Fig. 25, C). These take a similar course to those of the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, toward the occipital lobe. They are mingled with fibres which pass to the optic thalamus, but are separable from them in fœtal brains,[1] as Edinger has shown, and may be traced down through the capsule to the tegmentum of the crus, where they divide into a portion going to the lemniscus, and a portion going to the formatio reticularis. The fibres can be traced through these tracts downward to the medulla and through the sensory decussation to the posterior pyramids of the medulla in which the posterior columns of the spinal cord begin. This set of fibres conveys the sensations of touch, pain, temperature, and muscular sense,[2] and lesions in its course will cause disturbance of these sensations. Like lesions in the motor tract, the rule obtains that the nearer the cortex the more likely is the lesion to cause an affection of a single limb, while the nearer the capsule the more likely is the symptom produced to be hemianæsthesia.

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[1] Edinger: "Vorlesungen über den Bau der Nervösen Centralorgane," Leipzig, 1889.

[2] Starr: "Sensory Tract," loc. cit.

Looking, then, at the dissection of the projection system of fibres, it is evident that a lesion which lies in the centrum ovale at any point posterior to the præcentral fissure of the frontal lobe, may produce recognizable symptoms, for it must affect either the motor, or the sensory, or the visual, or the auditory tracts, or individual fibres of those tracts. In the latter case it will be necessary to examine carefully for symptoms, as they may escape a superficial examination.

THE COMMISSURAL TRACTS.

The second system of fibres in the centrum ovale is the *commissural* system. This joins corresponding areas of the two hemispheres with one another. The commissural fibres between the frontal, parietal, and occipital lobes of the two sides pass in the corpus callosum; the view of Hamilton, of Aberdeen, that these fibres are not commissural being disproved by the researches of Spitzka and Beevor.[1] Those from the temporal lobes pass in the anterior commissure. The function of these fibres is to harmonize the action of the two hemispheres. This may be proven with regard to the fibres which join the central convolutions with each other, and harmonize motor acts (Fig. 26). Every one knows that simultaneous movements of like nature can be made with great facility with both upper extremities. An attempt to swing Indian clubs is more difficult if each hand is executing a different motion. So, too, movements which are difficult when attempted with the left hand alone become easy when associated with corresponding movements of the right hand—as, for example, drawing a circle, writing one's name. But the motion, in order to be associated by the commissural fibres, must involve the corresponding muscles on both sides of the body. Hence, if the right hand moves to the right the left must move to the left to a similar degree in order to bring into play the aid of commissural association. Hence, such writing with the left hand will be backward, and can only be read by the aid of a mir-

[1] See Hamilton: Brain, July, 1885; Beevor: Brain, Oct., 1885; Spitzka: Amer. Jour. Neurol., Aug., 1884.

ror. Now, it is a very simple thing to test this power of symmetrical movement in persons, but I venture to state that it is very rarely done; so that no clue is usually obtained as to the condition of conducting power in the commissural fibres in cases of brain disease. It is the commissural fibres joining the motor convolutions only which can be thus tested. Failure to perform easily corresponding bilateral motions in face, hands, or feet would indicate some obstruction to conduction in them. With regard to the fibres lying in the anterior part of the corpus callosum and joining the frontal lobes, little can be said except that in numerous cases of idiocy these fibres have been wanting. The cases of McBride (*Amer. Jour. Neurol.*, May, 1884) and of Erb (*Virchow's Arch.*, Bd. 98) of lesion of the corpus callosum presented no peculiar local symptoms. Nor have we any power of testing the integrity of commissural fibres lying posterior to the motor tract, though a theoretical statement may be made, that those between the occipital lobes are very important, inasmuch as each occipital lobe receives impulses from but one-half of each retina. Hence, integrity of both occipital lobes and simultaneous, connected, and harmonious action in both is necessary to the *perfect* perception of the *whole* of any object when the eyes are fixed upon one point of that object. The function of the anterior commissure which connects the apices of the temporal lobes is probably that of conducting sensations of smell and taste, but this is not certain. Theoretically, a lesion of the commissural fibres should produce a lack of harmony in the simultaneous action of the two hemispheres, and if the lesion in the centrum ovale lay opposite to the two central convolutions, this could be detected by appropriate tests.

THE ASSOCIATION TRACTS.

The third system of fibres in the centrum ovale is the *association* system (Fig. 27).

It can be shown by careful dissection that each convolution is joined to the two adjacent convolutions by fibres which pass around the separating fissures. Also, that bundles of

fibres exist which pass from each convolution to the convolution next but one, and so on (Fig. 27, A). Hence, it may be stated that each convolution has a possible connection with every other. Besides this association of convolutions by small bundles of fibres, it is possible to find a distinct set of association tracts which pass between more or less distant regions. One such tract passes from the frontal lobe, collecting its bundles from all three convolutions, backward to the occipital

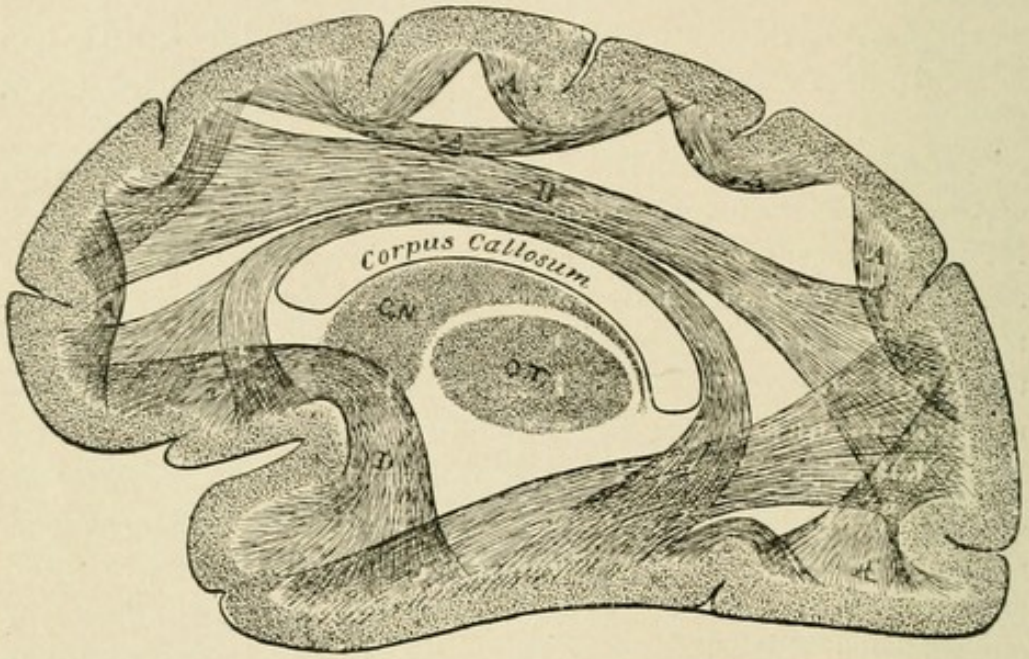

FIG. 27.—The Association Fibres. *A*, Between adjacent convolutions; *B*, between frontal and occipital areas; *C*, between frontal and temporal areas, cingulum; *D*, between frontal and temporal areas, fasciculus uncinatus; *E*, between occipital and temporal areas, fasciculus longitudinalis inferior; *C N*, caudate nucleus; *O T*, optic thalamus.

lobe (Fig. 27, B). Another tract joins the occipital with the anterior part of the temporal lobe (Fig. 27, E). Another passes from the upper two temporal convolutions forward to the third frontal convolution, passing beneath the island of Reil (Fig. 27, D). And a tract from the frontal to the posterior temporal area may also be found (Fig. 27, C).

What can be the function of these association fibres? Without going very deeply into the subject of psychology, it may be suggested that they form the physical basis for the association of concrete memories and of psychical acts.

Our concrete memories are reproductions of former sensations. But if the sensations are received in various areas of the brain, as we must admit from the facts already stated regarding the function of the projection fibres, it is evident that the reproduced sensation which depends on the revival of the original sensation will occur in the same part of the brain in which the original sensation was received. A sight memory is located in a different part of the brain from a sound, or touch, or motor, or speech, or writing memory. And this fact will be readily admitted by those who have seen cases in which a single set of memories have been lost while others remain. The symptoms of apraxia, word-deafness, word-blindness, aphasia, agraphia, alexia, are all dependent upon loss of a definite set of memories. And they are due to definite lesions of the cortex in different areas, as has already been stated in the discussion of aphasia (Chap. VI.). But no concrete memory can be said to exist without associations. The sight of a dog leads us at once to think of the form and appearance of other dogs, of animals resembling dogs, and of quadrupeds in general. Thus the impulse received in a memory area excites other memories of a *like* quality. Further, the sight of a dog might easily lead us to think of his bark, or, if we had once felt it, of his bite; and that because originally the simultaneous occurrence of the sight and sound of the animal had associated the two memories forever. Thus an impulse received in a memory area may excite other memories of an *unlike* quality. In fact, any one of our concrete ideas may be said to be made up of a number of associated memory pictures, all of them united into a definite whole. My idea of an orange is made up of my memory of the appearance of the orange, its color and form, of the memory of its odor and taste, of the peculiar feeling of its surface, of the sound which I know as its name, of the motion necessary in my throat and lips to pronounce the word which designates it, and of the movements necessary to write the word orange. And further, each one of these memories is joined not only to the next, but also to all the others, for if you show me an orange and ask me to say or to think or to write its name, I can do either at your

will, and need not say it before I write it, or taste it before I can call to mind its odor.

These facts of concrete memory gained by introspection show how intimate is the connection between our various kinds of memories, and demonstrate *à priori* the need of just such sets of association fibres in the brain as may be found by anatomical dissection. It may be stated, therefore, that the association fibres of the centrum ovale have as a function the harmonizing of the activities of the various cortical areas. They are necessary to the union of different memories in a whole, to the formation of a concrete idea. I do not now allude to abstract ideas, or to processes of reasoning. I have spoken only of concrete ideas, for it is only regarding these that our investigation of cases of localized brain disease will allow us to speak. But if these association fibres pass through the centrum ovale, it is evident that the integrity of the centrum ovale is necessary to their proper action, and that lesions of the centrum ovale will lead to a separation or failure of association of ideas and memories formerly joined. If the fibre is broken which joins my memory picture of a familiar face with my memory picture of the sound or name associated with that face, I cannot call the person by name on seeing him. If I have lost the power of copying a page that is placed before me and which I can see, it is possibly because I no longer associate the sight of a letter with the motion of my hand necessary to write that letter, an association originally made by practice. If I can understand what is said to me, and can pronounce words distinctly, but talk jargon or replace one word by another, it is because the fibres which join corresponding auditory and motor speech memories are unable to perform their function.

Instances of such loss of function in the association fibres between the temporal and occipital lobes have already been given in the chapter on aphasia.

It is evident, therefore, that we have not only a knowledge of the anatomy and physiology of the association fibres of the centrum ovale, but also a set of symptoms referable to their destruction. But here, again, it must be emphasized

that careful examination of cases in a proper way is necessary in order to detect a lesion of such fibres.

Brill has recorded [1] a case of lesion of the cuneus associated with color-blindness to green, and he states that the patient had difficulty in naming various colors on account of the presence of a slight degree of amnesic aphasia. This aphasia was most marked in regard to names of colors, not in regard to contours, for he would compare colors to the tints of common objects. The aphasia was limited to the names of those colors which were imperfectly perceived—green and violet.

These facts seem to emphasize the importance, already insisted upon, of careful examinations, especially in cases of aphasia.

THE DIAGNOSIS OF SUB-CORTICAL LESIONS.

It is evident, therefore, that a lesion of the centrum ovale may give rise to three sets of symptoms, referable to projection, commissural, and association fibres. The important fact at once appears that these symptoms will be inevitably associated in any case of its occurrence. It is upon the fact of such *association of symptoms* that the diagnosis of lesions of the centrum ovale must depend. Monoplegia alone may be caused by cortical or centrum ovale disease. Hemiplegia may be caused by capsular or centrum ovale disease. But if in either case it is the centrum ovale which is diseased, there will be in addition to the paralysis a suspension of action of commissural and association fibres. The commissural fibres being destroyed, simultaneous bilateral symmetrical motions will be suspended. The association fibres being destroyed, certain mental associations will be affected. Take as an example a lesion in the centrum ovale of the occipito-temporal region. Such a lesion will produce hemianopsia because it involves the visual tract of the projection system. It may also produce word-blindness, the patient being no longer able to associate a word or letter seen with its corresponding sound, or with the motion necessary to write it. Charcot has re-

[1] Amer. Jour. of Neurology, Feb., 1883.

ported a case of this kind, and as I had an opportunity to see and examine the patient under his direction, I may be excused for mentioning it in detail. The man, who was a very intelligent merchant, was suddenly seized with right hemianopsia while playing billiards, and was surprised to find that he saw but one-half of the ball and of the table. Soon after he had occasion to write a letter, and after writing it was surprised to find that he could not read what he had just written. He found, however, that on tracing individual letters with the pen or fingers he became conscious of the letter—a few letters (r, s, t, x, y, z), however, being exceptions to this rule. When a book was given him to read, he would trace out the forms of the letters with some rapidity and thus manage to make out the words. If his hands were put behind him and he was asked to read, he would still be observed to put his fingers in motion and to trace the letters in the air. Speech was in no way interfered with, but reading aloud was only accomplished, like reading to himself, by the aid of muscular sense. Here, then, was an example of a lesion which had separated entirely the tract associating sight with speech, viz., the occipito-temporal tract; but had left intact the tracts associating sight with muscular sense and muscular sense with speech.

The first tract, the occipito-temporal, lies side by side with the visual, and its implication in the lesion which caused the hemianopsia is not to be wondered at. The second tract, the occipito-central, lies much higher in the centrum ovale and evidently escaped. Now, had this patient been an unintelligent laborer, seldom called upon to read or write, such a peculiar symptom might have escaped notice, and although it might have been discovered that he had alexia, the possibility of his being able to read by means of his muscular sense would only have been elicited by proper investigation. This case also illustrates a possible point of differential diagnosis between lesions of the centrum ovale and lesions of the cortex. If the lesion is in the cortex, some one of the cortical functions—*e.g.*, sight or motion—will be suspended together with their memories; while in lesion of the centrum

ovale it is only the association of two or more memories which will be impaired. It is possible that in cases of word-blindness the symptoms are due to such a break of the association tracts when sight and speech are preserved. Certainly, in numerous cases of word-blindness and in the condition of dyslexia[1]—*i.e.*, a great reluctance with some inability to read—the lesion found was in the centrum ovale just beneath the cortex, at the posterior end of the Sylvian fissure, and involved the centrum ovale where the occipito-temporal tract lies.[2]

There is a psychological deduction which occurs naturally from the study of the case just related. The man could see the letter so as to be able to trace its form, and yet the sight alone conveyed to him no idea. We have already seen, in studying apraxia, that a concrete idea must be considered as a complex collection of memory pictures. Is it not evident from this case that a single one of these pictures isolated from the rest is incapable of awakening a concrete idea? The patient only obtained the idea when he put into action the motor memory, whose connections with sight and sound and speech were all intact. We have spoken only of concrete ideas. If we conceive of abstract ideas as made up of a numerous assemblage of concrete ideas, as they may possibly be, brought into consciousness as a single concept by the aid of the name or word, is it not probable that lesions of the centrum ovale will give rise to a confusion of abstract as well as of concrete ideas? And is it not possible that the symptoms of mental confusion of that peculiar kind which occurs in diseases of the frontal lobes of the brain, may be ascribed to a suspension of action of association and commissural fibres which pass in all directions through the frontal lobes?

Nothnagel, in his great work, "Topische Diagnostik," has said that there are no diagnostic local symptoms of lesion of the centrum ovale. Such symptoms as headache, vertigo, vomiting, mental inactivity, and optic neuritis being general rather than local symptoms, and common to affections in any part of the brain, are frequently present in disease of the cen-

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[1] Berlin: "Ueber Dyslexie."

[2] Brain, part xlv., p. 91.

trum ovale. It is not to these that reference is made, but rather to symptoms whose presence would lead inevitably to the diagnosis of disease below the cortex and above the basal ganglia. Nothnagel's assertion may be repeated to-day, notwithstanding the labor that has been expended since 1879 on the study of localized brain disease. It is evident to me that the way to solve the problem here is to reason from anatomy and phyisology to the probable results of lesion in the centrum ovale, and then in every case to examine for the symptoms which theoretically might occur. In that manner it will be evident that no latent symptoms will be overlooked, and it is not impossible that, after a time, so many new symptoms now unsuspected may be educed and brought into connection with lesions found by post-mortem examination, as to warrant the formulation of certain diagnostic signs of diseases of the centrum ovale.

CHAPTER IX.

AFFECTIONS OF THE BASE OF THE BRAIN.

I. Paralysis of the ocular nerves.—II. Unilateral cranial nerve paralysis.—III. Staggering to one side as a symptom.

I. PARALYSIS OF THE OCULAR NERVES.

In affections of the parts of the brain which lie upon the base, one of the most common symptoms is some form of paralysis of the ocular muscles. There are a number of forms of such paralysis, each of which implies a lesion in a different position.

Internal strabismus with inability to turn the eyeball outward and an accompanying contraction of the pupil is most frequently seen. This implies an affection of the sixth or abducens nerve, which has the longest course upon the base of any cranial nerve, and hence may be affected by a lesion anywhere from the sphenoidal fissure in front to the junction of the pons and medulla behind (see Fig. 28).

External strabismus, with inability to move the eyeball upward or downward, and with ptosis, as well as dilatation of the pupil and loss of pupil reflexes, is also often observed. These symptoms together imply an affection of the third nerve or oculo-motorius. This nerve issues from the inner side of the crus and passes forward to the orbit. A lesion in its course implies that the disease producing it lies in front of the pons Varolii. If the disease is a tumor or meningeal thickening situated between the two crura, both third nerves are often involved together, as they there lie side by side.

Paralysis of the fourth nerve is extremely rare and is only to be detected by a careful examination for double images with a red glass over one eye. The upright image is then

seen to be upright by the normal eye, but appears to be displaced downward and turned obliquely by the affected eye. Such a conditon indicates disease upon the back of the cere-

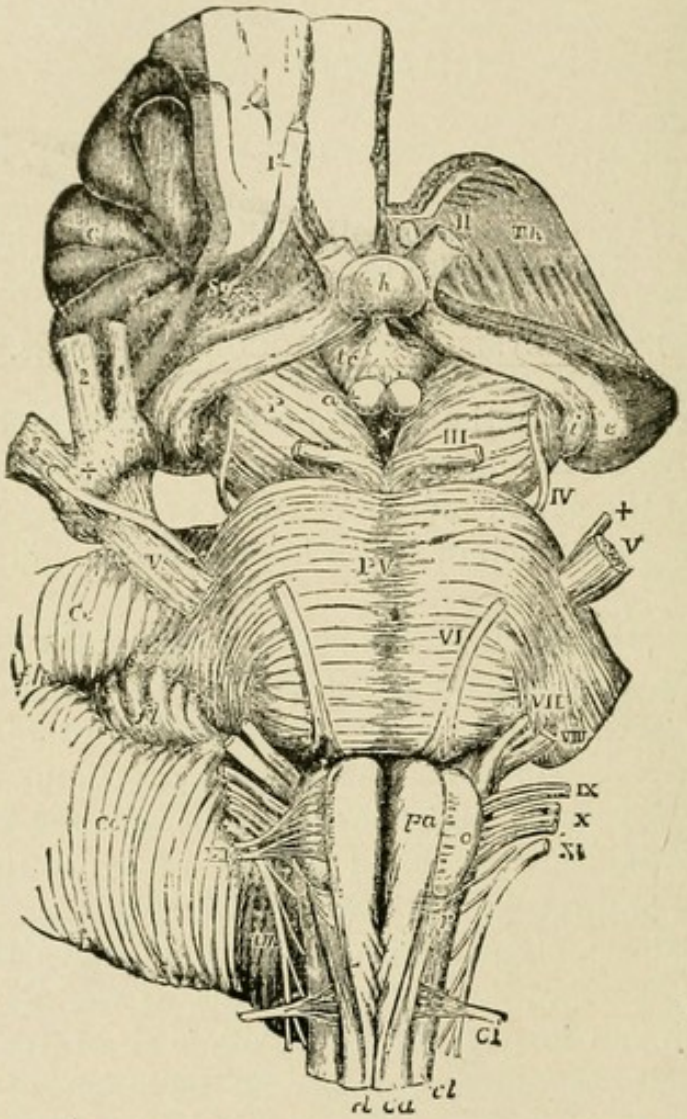

FIG. 28.—The Base of the Brain and the Cranial Nerves. Crura, Pons and Medulla (Allen Thompson). *I* to *XII*, the cranial nerves; *Th*, optic thalamus; *h*, pituitary body; *tc*, tuber cinereum; *a*, corpora albicantia; *P*, pes pedunculi; *i*, interior, and *e*, exterior, geniculate body; *PV*, pons Varolii; *pa*, anterior pyramid of medulla; *o*, olive; *d*, decussation of anterior pyramids; *ca*, anterior column of spinal cord; *cl*, lateral column of spinal cord; *Ce*, cerebellum; *fl*, flocculus of cerebellum.

bral axis or in the anterior part of the cerebellum, since the fourth nerve does not pass out on the base but curves around the outer side of the crus lying between the corpora quadrigemina, and the cerebellum (see Fig. 29). In a case present-

ing the symptoms of cerebellar disease, the existence of fourth-nerve paralysis shows that the anterior portion of the cerebellar hemisphere on the side of the paralysis is the part of the cerebellum affected. This statement is based on a case with autopsy seen in Meynert's clinic in Vienna.

It is very common to find the sixth and third nerves of one

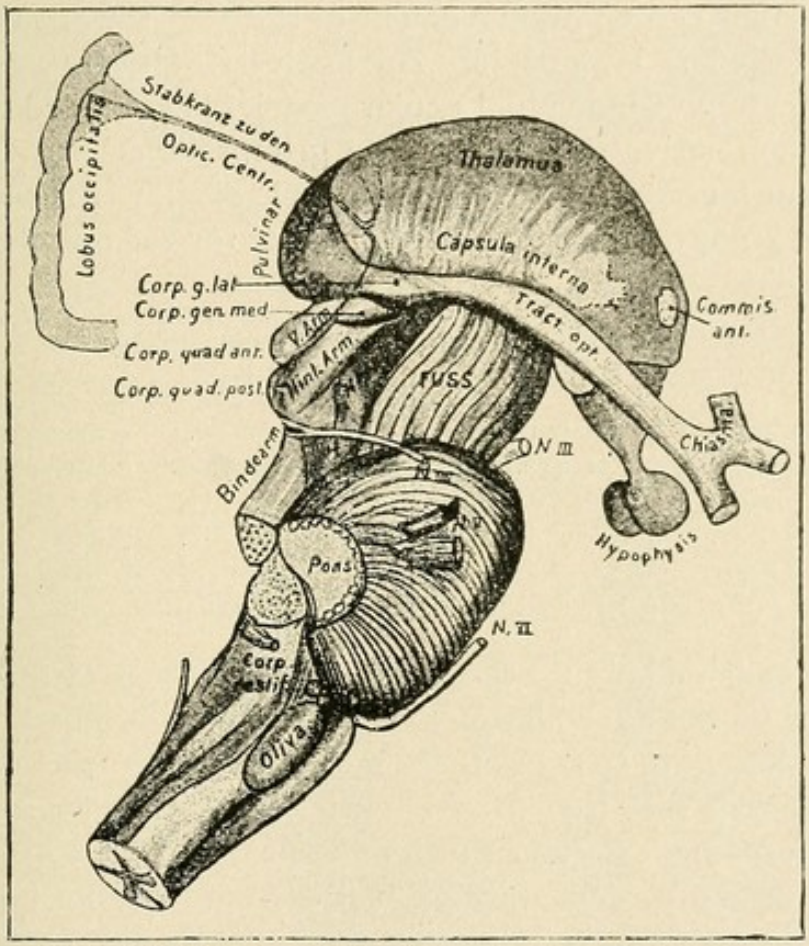

FIG. 29.—The Cerebral Axis, Lateral View (Edinger). The exit of the ocular nerves are shown; N. III., N. IV., N. VI. Fuss = pes pedunculi. Bindearm = superior peduncle of cerebellum. *Corp. g.* = Corpora geniculata.

or both sides affected together in varying degrees in basilar affections, especially in the forms of syphilitic disease of the base.

It may be laid down as a rule that when one of the ocular nerves is affected all of its functions are about equally impaired. This condition is not, however, always found. There are cases observed in which a single ocular muscle or two

muscles become weak, the others not being affected. Under these circumstances the conclusion is warranted that the lesion does not lie in the course of the nerve supplying the muscles, but is located in the group of cells from which various sets of fibres making up the nerve arise. If it is the external rectus which is paralyzed alone, a lesion of the sixth nucleus can be diagnosticated from one of the sixth nerve by the fact that conjugate movement of the eyes toward the side of the lesion is impossible. An analysis of twenty cases of such affection of individual ocular muscles has enabled me [1] to locate the relative situation of the groups of cells governing the ocular muscles. The following table displays the situation of these groups, each group being named for the muscle which it governs:

ARRANGEMENT OF THE GROUPS OF CELLS GOVERNING THE OCULAR MUSCLES.

Right.		*Left.*	
2 Sphincter iridis.	1 Ciliary muscle.	1 Ciliary muscle.	2 Sphincter iridis.
5 Levator palp. sup.	3 Rectus internus.	3 Rectus internus.	5 Levator palp. sup.
6 Rectus superior.			6 Rectus superior.
7 Obliquus infer.	4 Rectus inferior.	4 Rectus inferior.	7 Obliquus infer.
8 Obliquus super.			8 Obliquus super.
	9 Rectus externus.	9 Rectus externus.	

The cells making up these groups lie in the gray matter of the floor of the aqueduct of Sylvius and of the fourth ventricle, all but the last group being underneath the corpora quadrigemina (see Fig. 30). Disease of a single group results in the permanent paralysis of its muscle, just as the disease of a single group of cells in the spinal cord in infantile paralysis results in the paralysis of a single muscle. A number of groups may be affected together, and Wernicke has named such a disease when it occurs suddenly polio-encephalitis superior, and he considers it quite homologous to polio-myelitis. When all the groups except the sphincter iridis are simultaneously or successively diseased, the disease is named ophthalmoplegia externa. The result is a double ptosis, inability to move the eyes in any direction, but preservation of sight

[1] "Ophthalmoplegia Externa Partialis," Jour. Nerv. and Ment. Dis., May, 1888.

and of the pupil reflex to light. The appearance of the patient suffering from ophthalmoplegia externa in its terminal stage

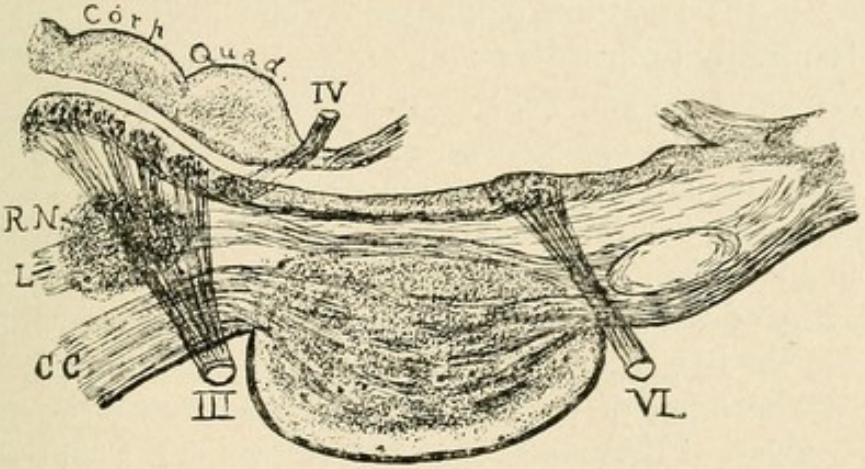

FIG. 30.—Sagittal Section through the Cerebral Axis to show the Nuclei of the Ocular Nerves in the Floor of the Aqueduct of Sylvius and Fourth Ventricle and the Course of the Nerves to their Exit. The various groups of cells from which the III. N. arises are seen. *R N*, Red nucleus of tegmentum; *L*, lemniscus (sensory tract); *C C*, motor tract in the crus cerebri seen to traverse the pons and enter the ant. pyramid of the medulla.

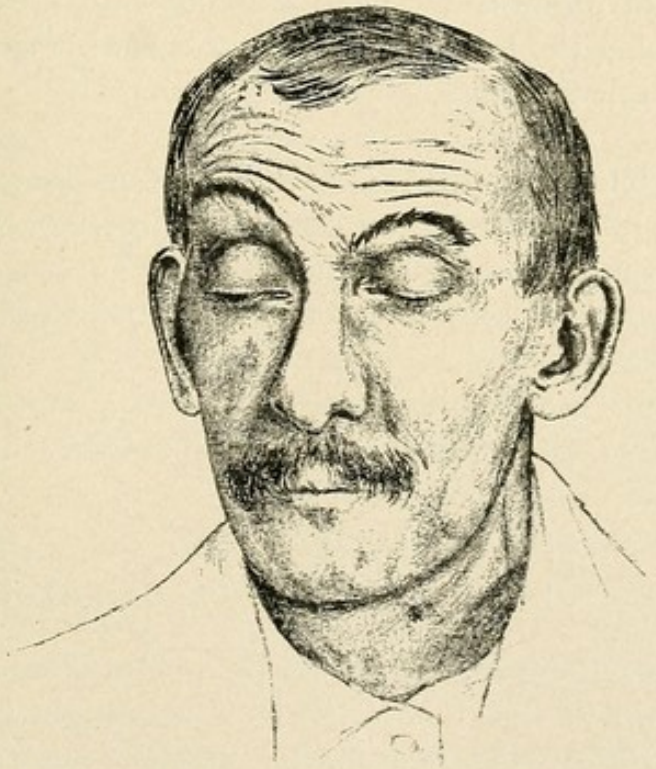

FIG. 31.—Photograph of Patient with Ophthalmoplegia Externa. Double ptosis, external strabismus. The muscles of mastication are also atrophied.

is shown in Fig. 31, a photograph of a patient under Dr. Peterson's care at Ward's Island Hospital. I am indebted to

him for the use of the photograph. The great effort of the patient to open the eyes by contracting the frontal muscles in place of the levatores palpebræ is well shown. Such a disease is usually progressive and cannot be arrested by treatment.

Ophthalmoplegia interna, in which the movements of the iris alone are paralyzed, the external muscles of the eye not being affected, is a rare condition seen occasionally as a symptom in hydrocephalus and paretic dementia, and due to a thickening of the ependyma of the third ventricle with implication of the two anterior groups of cells just mentioned, which lie at the entrance of the aqueduct of Sylvius into the ventricle. Paralysis of the pupil reflex to light, while the contraction in accommodative effort is preserved, is called the Argyll Robertson pupil, and is due to some interference with the passage of impulses along the fibres from the corpora quadrigemina to the cells controlling the iris and not to disease in the group of cells governing the iris. It is a symptom of locomotor ataxia and of multiple sclerosis.

It is evident from this review of symptoms referable to the ocular muscles, that it is not difficult to distinguish between lesions on the base affecting the ocular nerves and lesions within the cerebral axis affecting their nuclei in the floor of the fourth ventricle.

The following cases illustrate the latter condition, and examples of the former will be considered subsequently.

CASE XIV.—PARTIAL OPHTHALMOPLEGIA EXTERNA—SOFTENING IN THE CRUS CEREBRI DUE TO EMBOLISM.

S. D., a Frenchman, aged fifty-six, a painter by occupation, and a resident of Providence, R. I., was brought to the clinic April 18th, 1887. He had been a healthy man all his life, with the exception of occasional attacks of rheumatism and frequent attacks of migraine. He had never contracted syphilis, and denied all symptoms of pulmonary, cardiac, gastro-intestinal, and renal disease, although a physical examina-

tion revealed the existence of slight aortic obstruction producing a systolic murmur heard at the base and associated with slight ventricular hypertrophy.

He stated that about the 1st of April, 1887, he had been seized very suddenly with double vision and vertigo, objects appearing to move up and down constantly, so that he was much bewildered and unable to stand or to walk alone. He managed with help to reach his home, but has no recollection of what occurred during the three following days, during which, according to the statement of his family, he lay in a somnolent condition, but not comatose nor paralyzed. He was then able to get up, but felt stupid, dizzy, and walked with difficulty, it being impossible for him to fix any object with his eyes; all objects being seen double and in motion. These symptoms have improved slightly, but he still feels weak, has vertigo and double vision. He has never had headache, nor has he felt any sensation of numbness or cold or pain in his body, and he has had no paralysis, tremor, or spasm. He staggers in walking and the staggering is not increased by closing his eyes.

Examination shows a well-nourished, intelligent, active man, whose facial expression is rendered peculiar by the position of his eyes. When at rest, they diverge slightly, and the right eye is turned upward, and the right pupil is slightly larger than the left. When the eyes are moved it becomes apparent that the motion is defective. The eyes can be turned from side to side together perfectly, but such motion soon produces lateral nystagmus of the right eye. They cannot be converged to an object nearer than two feet, because of slight weakness of the right internal rectus muscle. They cannot be turned downward below the horizontal line either together or when tested separately. When asked to look up, the right eye follows the object above the horizontal line, but the left eye does not. Both eyes, however, turn up and in, though this motion produces rotary nystagmus. The reaction of the pupils to light and in accommodation is prompt, though the right pupil contracts in accommodation more slowly and less completely than the other, and remains slightly larger. Tests

by secondary deviation and double images confirm the conclusion reached by this examination, viz., that in the right eye there is paralysis of the inferior rectus and paresis of the internal rectus; and that in the left eye there is paralysis of the inferior rectus and superior rectus. There is no ptosis. There is no paralysis of the oblique muscles or of the external recti. One week later the paresis of the right internal rectus, the difficulty in convergence, and the difference in the size of the pupils had disappeared, but all the other conditions remained, and persisted for fourteen months. He then had a stroke of apoplexy due to embolism of the right middle cerebal artery with left hemiplegia, which still remains. He is still suffering from general weakness, vertigo, and double vision, although the latter symptom no longer troubles him excepting when he tries to read. Attempts to turn the eyes up or down, or upward or inward, produce nystagmus of a rotary kind, more marked in the right eye, and this always makes him dizzy. The diagnosis made at first was embolism, from the aortic valve, in the small arteries entering the posterior perforated space between the crura cerebri, and resulting in one or more small foci of softening in the tegmentum cruris; a diagnosis which the subsequent development of hemiplegia confirmed.

This diagnosis was reached by exclusion; for it is impossible for the symptoms to have been caused by a tumor or a meningitis upon the base of the brain in the course of the third nerves. Such a lesion would not have come suddenly or have remained stationary, and would have involved the oculo-motor nerve as a whole, impairing all its functions and not affecting merely a part. Nor is a condition of acute inflammation with hemorrhage in the floor of the aqueduct of Sylvius possible, nor such a condition as occurs in a true ophthalmoplegia externa; for there is no tendency manifest toward an extension of the symptoms, or to complete immobility of the eyes.

It is true that a hemorrhage in the same region is with difficulty distinguished from an embolism, but in hemorrhage some evidence of pressure upon the adjacent sensory or motor tracts is usually shown which was wanting here at first; and

in this case the existence of an aortic murmur and the subsequent occurrence of hemiplegia make embolism probable.

CASE XV.—THROMBOSIS OF AN ARTERY IN THE TEGMENTUM OF THE CRUS CEREBRI.

Henry B., aged forty, applied for examination at the New York Polyclinic February 27th, 1886, complaining of numbness in the right side of his body and of double vision. He denied syphilis, but admitted having had gonorrhœa twice, and having suffered from severe sore throat and nocturnal headache. Has never had any eruption. No scars on genitals. It is, therefore, uncertain whether he has had syphilis.

He was fairly well until five months ago, when, on waking one morning, he noticed that he saw everything double, and had a peculiar sensation of numbness in his entire right side. This condition has been permanent.

Examination shows a well-nourished man, able to walk naturally. There is slight ptosis of the left eye, and a marked sinking downward of the eyeball when at rest. The pupil is slightly dilated, but reacts to light and during accommodation. Voluntary motion of the eyeball is good, except upward, the superior rectus being weak. When his eyes are at rest, he sees double, the new object appearing just above the old one. From the consequent misinterpretation of the position of objects, he lifts his feet higher than necessary in walking or in going up-stairs. When he turns both eyes to the right or to the left, the double vision persists, but the two images maintain the same relative position. There is no apparent deviation to either side, and no secondary lateral deviation can be noticed when one eye is covered. Examination by Dr. Webster: R.V. $\frac{20}{30}$, L.V. $\frac{20}{30}$. Hm $\frac{1}{50}$ in both eyes. No insufficiency at 20′ nor at 1′. Ophthalmoscopic examination negative. The condition of the eye is one of paresis of the levator palpebræ and superior rectus muscles; all other muscles of the left eye and all muscles of the right eye being normal.

He complains of a constant feeling of numbness and tingling in the right half of the body; face, arm, body, and leg

being equally involved. Examination shows a slight degree of tactile anæsthesia in the entire right half of the body, excepting a small area back of the ear and on the neck, where sensation is normal. There is also a difference between the perception of temperature sensations in the two halves, heat and cold being perceived less intensely on the right side. The sense of pain is also somewhat impaired on the entire right side. These sensations are not delayed in transmission. Muscular sense is normal. There is no incoördination, all motions being accurately and promptly performed. The reflexes are normal and equal on both sides. There is no loss of power. There are no other symptoms. Vertigo and headache are absent. Hearing normal. The examination indicates a lesion in the course of the sensory tracts of touch, temperature, and pain from the right half of the body, not sufficient in extent to destroy completely that tract, but only to impair its action.

Since the sensory and ocular symptoms began simultaneously, it is probable that they were due to one cause. The fact that but two of the branches of the left third nerve were involved pointed to a lesion in the nucleus of the nerve rather than to a lesion in the nerve trunk. In this case, the fifth and sixth groups of cells (page 108) were affected. But just at the side of the gray matter lining the aqueduct of Sylvius, in which these groups lie, the formatio reticularis of the crus cerebri is situated. In the formatio reticularis, the tracts conveying touch, temperature, and pain pass from the opposite side of the body toward the internal capsule. A lesion lying in the formatio reticularis on the left side of the tegmentum, if small in size, might at once interfere with the transmission of sensations from the right side of the body, and affect the nuclei of the left third nerve. The lemniscus, lying outside of the formatio reticularis, would not be affected if the lesion were a small one, and hence no ataxia or incoördination would be present. A lesion, therefore, in the formatio reticularis of the tegmentum of the left crus cerebri would explain perfectly the symptoms present in this case (see Figs. 17 and 30).

As to the nature of the lesion, its sudden onset and the

stationary character of the symptoms indicated that tumor and hemorrhage were equally improbable. If a small thrombus had formed in one of the twigs of the posterior cerebral artery which supplies the crus cerebri, it would have led to a small area of softening, the symptoms of which would have been sudden in onset and stationary in character. An embolus would produce the same effect, but the patient's freedom from cardiac disease threw doubt on the hypothesis of embolism. The diagnosis of thrombosis was strengthened by the fact that syphilis was suspected, a specific endarteritis being the usual cause of thrombosis.

The case is interesting, as it demonstrates the possibility of local diagnosis from the peculiar combination of symptoms present; and also as it is rare to find an affection of but two of the nuclei of origin of the third nerve. The failure of the patient to return a second time prevents any statement regarding the termination of the case.

II. UNILATERAL CRANIAL NERVE PALSIES.

The diagnosis of affections of the base of the brain is rarely difficult, and cases of these affections are very frequently met with. From before backward upon the base the cranial nerves lie in a definite order, and no serious lesion can be present there without involving them. In any case, therefore, in which the cranial nerves are affected, attention is at once directed to the base of the brain as the seat of the disease. Very frequently the nerves upon both sides are involved in the lesion, especially when this is a syphilitic meningitis or gumma, or some other form of tumor. Such cases are so common as to need no special description. For in addition to the general symptoms of brain disease, such as headache, vertigo, insomnia, restlessness, or even convulsions, the local symptoms of cranial nerve lesion enable a diagnosis to be reached at once.

The lesion may also be strictly unilateral, the cranial nerves upon one side only being affected. And as such unilateral cranial nerve palsies are rare, some of the cases on record are here collected for comparison with those presented.

I. Bamberger (*Wien. Medicin. Wochenschr.*, 1883, p. 114) reports the following case:

A woman, aged fifty-three, previously healthy and without syphilis or tubercular disease, began to suffer in the spring of 1880 from pain in the entire right side of the head and face which was sufficiently intense to keep her awake. In the fall a right-sided facial paralysis developed, and soon after an internal strabismus of the right eye. During the following year the right eye became inflamed, and ulceration of the cornea rendered sight imperfect. She came under observation in 1882, and was shown by Bamberger at the Medical Society of Vienna in January, 1883. It was then demonstrated that the right eyeball was completely immovable, and the upper lid could not be raised; that the entire right side of the face, including the conjunctiva and mouth, was anæsthetic, and that the cornea had become ulcerated and opaque; that the right half of the face was completely paralyzed, taste being affected on the right anterior part of the tongue and the uvula also being paretic; the muscles of mastication were also weak. The patient could not hear a tuning-fork placed upon the mastoid process as well on the right side as on the left; although the watch held before the ear was heard better on the right side, a fact which Bamberger explained by supposing an increased tension of the drum membrane due to the facial paralysis. There was anæsthesia of the right half of the fauces and paralysis of the right vocal cord, also paralysis of the trapezius and sterno-mastoid muscle. There were no symptoms of vagus paralysis and the hypoglossal nerve was not involved, as the tongue was freely movable.

Bamberger concluded that the lesion was one of the nuclei of these nerves along the floor of the fourth ventricle.

II. In February, 1884, Nothnagel presented to the Vienna Medical Society a patient with paralysis of the fifth, sixth, seventh, eighth, ninth, tenth, eleventh, and twelfth nerves, on the left side. She had paralysis of the tongue, of the left trapezius and sterno-mastoid; difficulty in breathing, swallowing, and talking, the left vocal cord being paretic; loss of taste and anæsthesia of mouth and face and eye on the left side; a cen

tral deafness of the left ear; internal strabismus as well as left facial paralysis, including the muscles of mastication. There was neuro-retinitis, but no paresis of the extremities. The third and fourth nerves escaped. Nothnagel thought the lesion was a unilateral chronic tubercular meningitis, or a tubercular tumor involving the cranial nerves after their exit from the side of the cerebral axis (*Centralblatt für Nervenheilk.*, 1884, p. 147).

III. Brière reports a case in the *Gazette des Hôpitaux*, 1884, p. 883, of paralysis of the fourth, fifth, sixth, and seventh cranial nerves on the right side which occurred in a girl aged twenty-one, who had been infected with syphilis a year previously. She had a growing tumor in the mouth, and it was supposed that a similar tumor on the base of the brain had caused the symptoms. She died a year later.

IV. Dr. Hughlings Jackson reported a case in the *Med. Times and Gazette* for January 8th, 1870, of a male, aged twenty-eight, who had had syphilis and who was found to have paralysis of the fifth, sixth, seventh, and twelfth nerves on the left side and paralysis of the vocal cord also on that side. He was slightly deaf in the left ear. There was marked weakness and anæsthesia in the right limbs, which led to the diagnosis of a syphilitic affection of the base involving these nerves and compressing the pons.

V. Mader, of Vienna, reports in the service of the Rudolph Hospital for 1882, a case of sudden paralysis of the right face with anæsthesia, loss of taste, and paresis of the right half of the tongue. The electric reactions of the facial nerve were those of degeneration. The patient recovered.

VI.–IX. Möbius recounts five cases of multiple cranial nerve paralysis in the *Centralblatt für Nervenheilkunde*, 1887, in four of which the paralysis was unilateral, and in all of which recovery followed specific treatment.

X. A case was reported by Dr. Ramskell, seen at the London Hospital in May, 1868 (*Med. Times and Gazette*, May 23d, 1868), of a man aged twenty-seven, syphilitic, who had suffered from ptosis and paralysis of the fifth, sixth, seventh, and eighth nerves on the left side. Taste was destroyed on the left side.

The cornea was ulcerated. There was no paralysis of the limbs and no difficulty of speech or of swallowing. He suffered much from nocturnal headache, and after some months developed optic neuritis. The termination of this case was not reported.

XI. A most remarkable and indeed unique case is reported in the *Fortschritte der Med.*, December 15th, 1887, of a gradually advancing total paralysis of all the cranial nerves on both sides, without other cerebral symptoms. The autopsy failed to reveal, at first sight, any lesion; but careful examination showed a sarcomatous growth beneath the dura covering the base of the brain in the bone, which had narrowed all the foramina of the skull and had thus compressed all the cranial nerves at their exit.

These cases demonstrate the possibility of an affection of the base of the brain producing unilateral cranial nerve paralysis. When the lesion lies outside of the cerebral axis, the motor and sensory tracts passing through it are less likely to be affected than when—as in Case XV.—the lesion lies within it. In the latter condition the characteristic symptom is the alternating character of the paralysis or anæsthesia, which can only occur from lesions in the pons Varolii or crus.

III. STAGGERING TO ONE SIDE.

In the following cases an additional symptom of a unilateral affection of one part of the brain lying on the base has been noticed, viz., *a persistent tendency to stagger toward one side* in walking, or to fall toward one side in standing. This symptom is not very often mentioned, but it is not infrequent and is of great value in determining the exact location of the seat of the disease, for, as the cases here collected show, it indicates that the lesion has involved the fibres of the pons Varolii as they pass off into the cerebellum. Since several of the cranial nerves pass out of the side of the pons, it is not to be wondered at that the symptoms of unilateral cranial nerve palsy and staggering should occur together.

The new cases here recorded for the first time show three

phases or degrees of this symptom of staggering. In one case the patient fell to one side on attempting to stand. In all three the patient staggered to one side in attempting to walk. In one the patient ran around in a circle when he walked, and in his epileptic attacks he ran about in a circle at the beginning of an attack, a symptom which has received the name of procursive epilepsy. It is interesting to observe that in all these cases the staggering was toward the side of the lesion—that side being indicated by the side of the cranial nerve palsy. In the two cases of procursive epilepsy with autopsy reported by Bricon,[1] the lesion was found in one half of the cerebellum, and in a recently published case of Mairet[2] a sclerotic patch was found in one hemisphere of the cerebellum.

CASE XVI.—UNILATERAL MULTIPLE CRANIAL NERVE PARALYSIS.

Syphilitic Basilar Meningitis or Gumma—Staggering Toward One Side.—The patient, a man of thirty, has been under my observation since February, 1884, and has presented very interesting symptoms. When first seen he was suffering from severe headache in the occipital region, worse at night, from insomnia, from vomiting, and from paresis of the *right abducens* and *facial nerves.* In the course of three months the *right trigeminus* had also become involved, so that there was some anæsthesia, analgesia, and disturbance of temperature sense in the entire right half of the face, as well as slight paresis, and internal strabismus with contraction of the right pupil. Then vision began to be affected and a slight swelling of the optic discs was seen with marked venous congestion, a beginning optic neuritis. Under mercurial inunctions and one hundred and fifty grains of iodide of potash daily these symptoms entirely disappeared in three months, but after six months of good health he again began to suffer from insomnia and headache, and soon after a paresis of the *right third nerve* with external strabismus, dilatation of the pupil and slight ptosis, and slight left hemiplegia developed. At this time the knee-jerk

[1] Bourneville et Bricon: "Recherches Cliniques sur l'Epilepsie," Paris, 1888.

[2] Mairet: Revue de Méd., 1889, pp. 147, 641, 741.

was absent from the left knee and exaggerated in the right knee, and the right half of his face was numb, but not anæsthetic. A renewal of the treatment produced a subsidence of the symptoms, so that in two months his eyes were straight and the left half of his body was as strong as the right. But three months after this he again applied for treatment, this time complaining of very great difficulty in walking owing to vertigo, with a marked *tendency to stagger and fall to the right side*, and almost total *deafness in the right ear*. The intensity of the symptoms increased, and at the end of a month it was found that the *right third, fifth, seventh, and eighth nerves* were all paretic, and *polyuria* had developed so that he was passing nearly three quarts of urine daily, which was, however, free from sugar. At this time there was a slight weakness of the left arm and leg, a very typical cerebellar gait, and a continued tendency to fall to the right. Specific treatment was again prescribed, and in the course of two months all the symptoms, except the polyuria and the tendency to stagger toward the right side, had disappeared. Since November, 1886, he has had no return of the cranial nerve symptoms. In January, 1890, when shown at the clinic, he still had a staggering gait with marked turning toward the right side when walking with his eyes closed, a loss of knee-jerk in the left side and exaggeration on the right side, and he complained of polyuria. All the cranial nerves were found to be normal, and there was no evidence of hemiplegia. The urine was free from sugar or albumin. The optic discs were clear. He had no headache and he slept well.

The Situation and Character of the Lesion.—This man must have had a syphilitic exudation upon the base of the brain limited to the right side, advancing from one part to another and invading at the outset the sixth and seventh, and later the sensory part of the fifth and the third nerves; and lastly, after subsiding in these two localities, appearing further back and involving the eighth nerve, and affecting the right middle peduncle of the cerebellum (Fig. 32). The fourth, motor fifth,

ninth, tenth, eleventh, and twelfth nerves were at no time involved. The entire recovery from all cranial nerve symptoms shows that the mass has been entirely absorbed. And the permanence of the symptoms of polyuria and staggering gait indicate rather a permanent destruction of tissue in the medulla and middle peduncle of the cerebellum than the existence of continued pressure. Such a destruction of tissue, limited in extent, might easily have been produced by the ob-

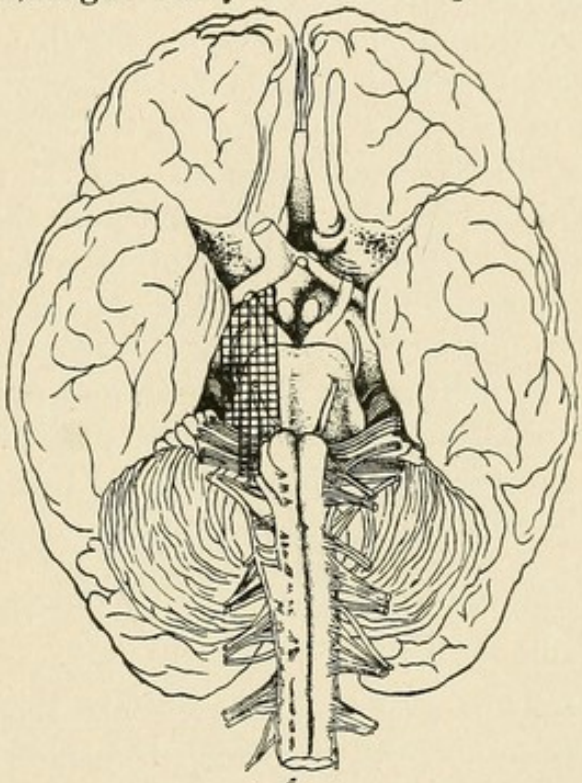

FIG. 32.—The Base of the Brain. Unilateral syphilitic meningitis causing cranial nerve paralysis in Case XVI.

struction of one or more branches of arteries entering the lateral surface of the medulla in close contiguity with the seventh and eighth nerves and resulting in an area of local anæmia and softening. Such a condition was found post-mortem in cases reported by Eisenlohr [1] and by Dr. Delavan,[2] the local softening in the latter case having been limited to a small area of the lateral part of the medulla, and causing in that case a unilateral paralysis of the vocal cord.

[1] Eisenlohr : Arch. f. Psych., xix., p. 314.

[2] N. Y. Med. Rec., 1885, Feb. 14th (autopsy subsequently).

Polyuria may be due to lesions in the middle third of the medulla, near the floor of the ventricle and in the lateral part of the formatio reticularis, Dr. Flatten having collected eleven cases of this kind.[1] It has been observed frequently by Oppenheim[2] as a complication of cerebral syphilis. Staggering gait may be due to lesions of the cerebellar peduncles, as well as of the middle lobe of the cerebellum in which these peduncles terminate. It is interesting to note that the tendency to stagger was *toward the side of the lesion.* This was also observed in a case reported by Buzzard[3] in which a tumor in the right side of the pons invading the right middle and inferior peduncles caused a staggering toward the right side. It was also present in a patient with brain tumor seen by me with Dr. Peabody at the New York Hospital, in which the autopsy showed the tumor to be located on the side of the pons.

In the following case a marked tendency to fall and to stagger toward the left side was present, and the concomitant symptom of alternating anæsthesia proved that here again the lesion was in the pons Varolii on the side toward which the staggering occurred.

CASE XVII.—HEMORRHAGE IN THE LEFT MIDDLE CEREBELLAR PEDUNCLE AND PONS.

Tendency to Turn and Fall Toward the Left Side—Alternating Anæsthesia.—A healthy but hard-working engineer was suddenly seized with vertigo, headache, and vomiting, and with double vision, difficulty in swallowing and speech, and numbness in the left side of the face and right side of the body and right limbs. These symptoms gradually passed off to some extent, but when seen two weeks after the attack, he was found to have partial anæsthesia of the left half of the face, and of the entire right side of the body from the level of the neck downward, and paresis of the left sixth nerve causing a partial internal strabismus. These symptoms seemed sufficient to locate the lesion in the lower half of the pons Varolii

[1] Arch. for Psych., xiii., 671.

[2] Oppenheim: Berl. klin. Woch., 1889, No. 48.

[3] Brain, 1888.

upon the left side, and in the absence of cardiac or arterial disease the lesion was thought to be a hemorrhage[1] (Fig. 33).

The chief feature of interest was the disturbance of his gait and equilibrium, a symptom which had been present from the onset. He staggered much in walking, although not paralyzed, and he turned toward or fell toward the left side whenever he started to walk. He had no vertigo, but on rising from a seat would fall over to the left unless supported. This

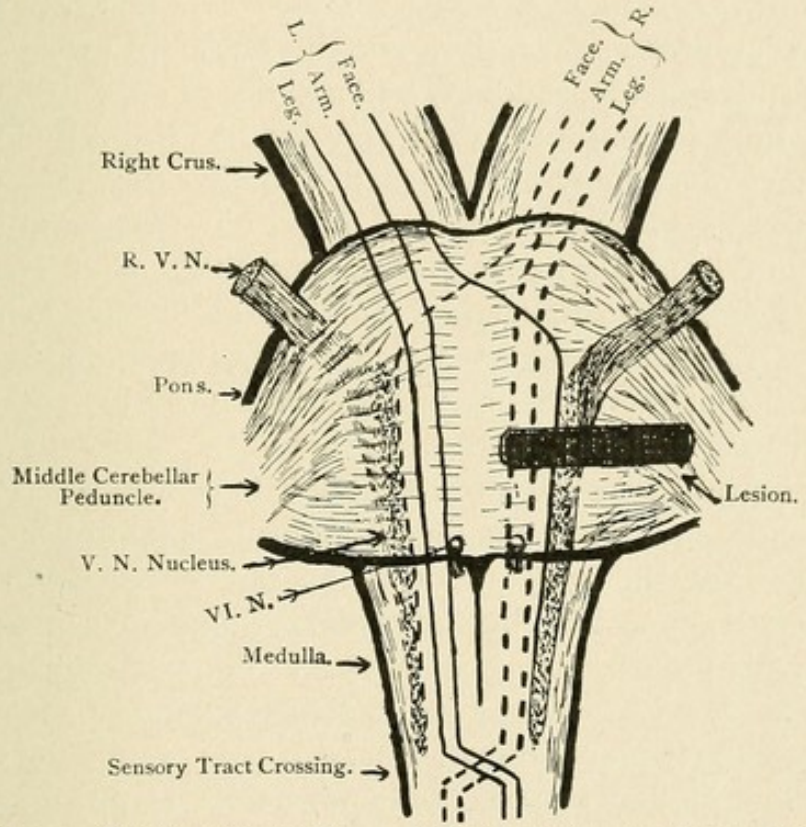

FIG. 33.—The Crura Cerebri, Pons, and Medulla showing the Sensory Tracts, and a Lesion producing Alternating Anæsthesia and Staggering toward the Left. Case XVII.

symptom did not appear to be increased by the closure of his eyes. There was no ataxia of the limbs. In the course of two months after the attack the diplopia had disappeared; the anæsthesia, though present, was less; but the gait was still very unsteady, with a marked tendency to stagger to the left. At present, a year after the onset, he is able to walk, but has still some traces of the anæsthesia and a tendency to stagger to the left.

[1] A case almost identical with this one is reported by Senator: Arch. f. Psych., xi.

In this connection the following case may be recorded, as it presents the same feature prominent in the preceding case viz., a tendency to turn to one side in walking, but in a much more marked degree. It also offers an example of procursive epilepsy.

CASE XVIII.—PROCURSIVE EPILEPSY IN A DEAF-MUTE.

Rotation to the Left in Walking.—A boy now (January, 1890) aged seven years was apparently a healthy baby until the age of seven months, when he suddenly had a series of convulsions, since which time he has been deaf and dumb. For a month after the convulsions he was paralyzed upon the left side of his body, but this hemiplegia passed off within two months, and no trace of it now remains except that the arm and leg upon the left side are smaller and not as well developed as those on the right, all movements, however, being perfect, without rigidity or increase of reflexes. He still has occasional attacks of epilepsy, and these are of the procursive variety; for he runs forward and to the left in an attack. He is still deaf and dumb and is manifestly wanting in intelligence, though he has not the appearance of an imbecile. He notices everything about him, but puts everything he can get hold of into his mouth, eating all sorts of things without regard to taste. He cannot be taught to keep himself clean. The feature of particular interest, however, is his tendency to move about in a circle whenever he is not held. If put down on the floor he begins at once to run around toward the left side. Even when standing he will turn about toward the left from time to time. This tendency is so uniform that at home he is kept tied to a chair. It has been present ever since he began to walk. The condition is very similar to that observed in animals after a unilateral operation upon the cerebellum.

The nature and position of the lesion in this case cannot be determined, there being no other symptoms of a localizing nature, but when compared with other cases it seems probable that the symptom of turning to the left indicates that the left middle peduncle of the cerebellum is diseased.

In concluding this chapter upon affections of the base, one additional set of symptoms, sometimes overlooked, may be mentioned. They are due to a disturbance of vaso-motor control.

The vaso-motor centre lies in the medulla and lesions in the upper half of the medulla produce marked vaso-motor symptoms. They consist of a general vaso-motor paralysis with flushing of the surface and sensation of heat, and of abnormal sweating. Polyuria and glycosuria may also be caused by its destruction. The vaso-motor centre is bilateral, and each centre controls the circulation on its own side of the body. Lesions in its area are so likely to cause sudden death that but few cases can be found in which it was affected. But the symptoms of a vaso-motor character are to be looked for in any case of medullary lesion, and when such symptoms are limited to one lateral half of the body, and are associated with other symptoms of bulbar disease, they are valuable as signs of the situation of the lesion.

There are a few general symptoms which have been not infrequently observed in disease of the base which require mention. General convulsions is the most constant of these. The majority of sudden lesions (hemorrhage, embolism) in the pons are ushered in by general convulsions, followed by coma. Nothnagel established the fact that in animals irritation of the pons produces general convulsions, and hence authors have spoken of a convulsive centre in the pons. This centre will be alluded to in discussing epilepsy. When the lesion is a tumor or a sclerosis—*i.e.*, a gradually increasing lesion—convulsions rarely occur. Headache, disturbance of vision, vertigo, and psychical changes have been frequently observed in connection with diseases of the pons and medulla, but they are to be ascribed to changes in the circulation or internal cranial pressure, and not to any special local lesion. Hemiplegia and hemianæsthesia often occur in lesions on the base when the motor or sensory tracts which pass through the cerebral axis are affected. Such lesions have already been considered in Chapter VIII.

CHAPTER X.

THE LOCALIZATION OF SPINAL CORD DISEASES.

The functions of the spinal segments. The relation of the segments to the vertebræ.—Diagnostic symptoms of local spinal lesions at various levels; (1) extent and character of the paralysis; (2) condition of reflex action; (3) distribution of sensory disturbance; (4) position assumed by the limbs.

THE FUNCTIONS OF THE SPINAL SEGMENTS.

THE localization of the functions of the gray matter of the spinal cord has been determined within the past six years. The first observations upon the subject were made by Gowers and Ross in England, and by Erb and Remak in Germany; they were based on but a few cases of disease. The numerous facts bearing upon the accurate localization of functions were first collected in 1884.[1] The careful microscopic study of the structure of each spinal segment in man, as compared with the structure of corresponding segments in fishes, birds, rabbits, dogs, oxen, and monkeys, furnished much valuable information. The review of the physiology of the various segments as investigated by Ferrier and Yeo, offered another set of facts. The study of numerous cases of congenital malformation of the cord and of the atrophy occurring in it after amputation produced a further line of evidence. And lastly, the pathological data derived from a collection of forty cases of anterior poliomyelitis, with autopsies, and of a still larger number of cases of syringo-myelitis and of transverse lesions at different levels, afforded ground, when compared with the

[1] Starr: Amer. Jour. of Neurol. and Psychiatry, August, 1884.

data furnished by these other methods of investigation, for some definite conclusions which were embodied in a table.

The accuracy of these conclusions has been attested by a large number of cases published during the last six years in various countries by different observers.[1] In only a few particulars has it been necessary to modify or to correct them. Such corrections have been embodied in the table presented on pages 128 and 129.

RELATION OF SEGMENTS TO VERTEBRÆ.

The relation of the various segments of the cord to the bodies and spines of the various vertebræ, must be known as a preliminary to the consideration of spinal surgery. As the cord extends only to the level of the second lumbar vertebra, its various segments do not lie opposite to the vertebræ from which they are named. The accompanying diagram of Gowers displays the mutual relation between the segments and their nerves, and the bodies of the vertebræ, and no further description is needed.

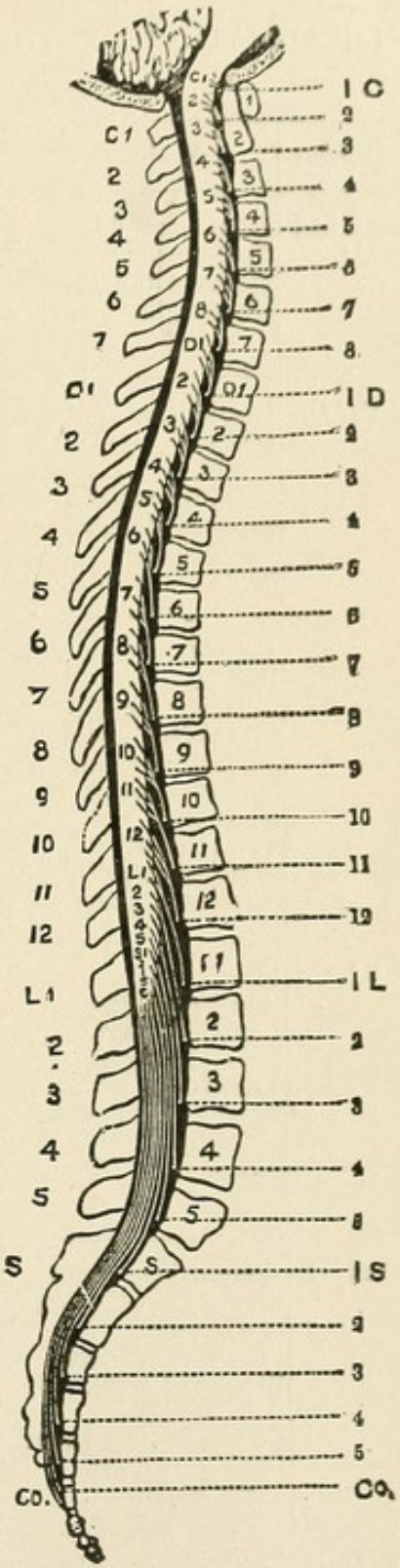

FIG. 34.—The Mutual Relations of the Vertebral Bodies and Spines to the Segments of the Cord and to the Exit of the Nerves. (Gowers.)

[1] The table has been reproduced in Pepper's System of Medicine, in Schmidt's Jahrbuch, in Edinger's Vorlesungen, 2d edition, in Mill's Monograph on Spinal Localization, and elsewhere.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
II. and III. C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra and infra spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V. C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex.	Sensation.
I. D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II. to XII. D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
I. L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg.	Outer side of thigh.
III. L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I. to II. S.	Flexors of ankle. Long flexor of toes. Peronei. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III. to V. S.	Perineal muscles.	Foot reflex. Achilles tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perineum. Genitals.

SYMPTOMS OF SPINAL LESIONS.

In any case of spinal disease in which the location and extent of the lesion are to be determined, four sets of symptoms are to be studied.

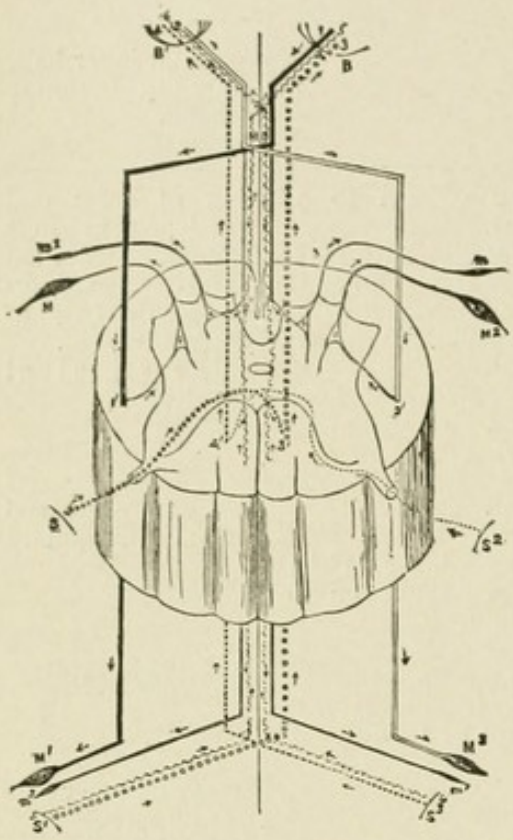

FIG. 35.—Diagram of a Spinal Segment as a Spinal Centre and as a Conducting Medium (Bramwell). *B*, Right; *B'*, left hemisphere; *M O*, medulla oblongata; 1, motor tract from right hemisphere; at *M O* it divides into a direct and a crossed tract, the former passes down the right ant. median column, the latter passes down the left lateral column, giving off at 1' fibres to the ant. gray horns of the cord, in which the motor cells lie, from which nerves pass to muscles, *M*; 2, motor tract from left hemisphere; *S*, *S'*, sensitive areas on the left side side of the body; 3, 3' the main sensory tract from the left side of the body; it passes into the post. gray matter of the segment, then decussates, and passes up the right half of the cord in the post. columns, thence to the right hemisphere of the brain; 4, 4', the main sensory tract from the right side of the body; 5, 6, the tracts of muscular sense from the legs, passing up upon the same side of the spinal cord in which it enters, in the column of Goll, decussating at the medulla, above the motor decussation. The arrows indicate the direction of nerve currents.

First, the extent of the paralysis, and the exact muscles which have atrophied and have lost their faradic reaction. Secondly, the condition of the reflexes: those which are lost; those which are exaggerated; those which are normal. Thirdly, the exact distribution of the region of anæsthesia. Fourthly, the position instinctively assumed by the limbs.

I. In regard to *the extent and character of the paralysis*, it is always to be remembered that a muscle may be paralyzed from two separate spinal lesions: first, because voluntary impulses from the brain to the spinal motor centres are cut off; secondly, because the spinal motor centres are destroyed. In the first case, when the disease is confined to the lateral pyramidal columns of the cord, and when impulses cannot reach the healthy spinal centres, the muscle, though paralyzed, does not atrophy to any marked degree, and

preserves its power of normal response to the faradic and galvanic currents. It has a firm consistence and may indeed be quite rigid, and its contractility to percussion may be exaggerated. This is the first type of spinal paralysis. In the second case, when the disease involves the gray matter and destroys the group of cells which governs the muscle, the muscle is not only paralyzed but also much atrophied, and loses its power of contraction when the faradic current is applied, either entirely or in great part. Its power of contraction to galvanic stimulus may be changed, both diminished,

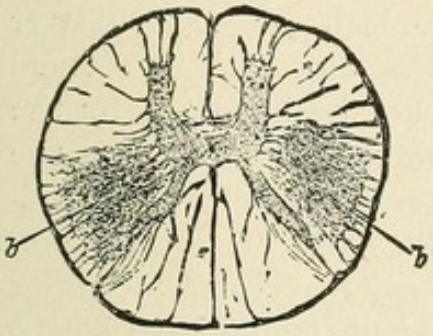

FIG. 36.—Lateral Sclerosis (Gowers.) The situation of the lesion producing the first type of spinal paralysis.

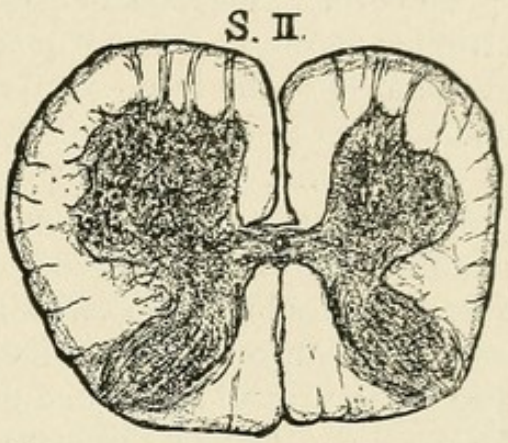

FIG. 37.—Ant. Poliomyelitis. The atrophy of the right anterior horn produced paralysis of the second type in the peroneal and post. tibial groups of muscles. Patient died at age of 28, paralysis came on at age of 7.

so that the contractions obtained are slow and require a stronger current to produce them, and also altered, so that the muscle responds more actively to the positive than to the negative pole (the so-called *reaction of degeneration*). The muscle feels flabby and is relaxed, its muscular tone is diminished and it does not contract on percussion. This is the second type of spinal paralysis.

The importance of the careful separation of these different types of paralysis cannot be exaggerated, since the line of treatment may be determined by the condition found. Thus in one case studied at the clinic, it was found that all the muscles below the waist were partially paralyzed; but the muscles of the abdomen and anterior surface of both thighs

were atrophied, flabby, and failed to react to faradism, while all the other muscles of the thighs and legs were of natural size, were rigid, their muscular contractility to percussion was increased, and the electric contractility was normal. The patient had fallen from a wagon and injured his dorsal spine, there being a marked deformity from the ninth to the twelfth dorsal vertebræ. The question to be decided, nine months after the injury, was the possibility of recovery from the paralysis by a surgical operation on the spine which should correct the deformity and remove pressure from the spinal cord. This was decided in the negative, because the atrophic paralysis of the quadriceps femoris indicated that at the level of the second and third lumbar segments the cord had been so completely crushed as to destroy the gray matter of the anterior horns, and this fact being certain, it seemed probable that the condition could not be benefited by the removal of pressure. The rigid non-atrophic paralysis in the other muscles indicated that the lateral pyramidal motor columns were thrown out of function, a symptom possibly produced by pressure and not necessarily associated with their destruction. Hence this symptom alone might have led to an operation for the relief of the pressure, had not the other shown that a part of the gray matter was destroyed, and hence that the columns were in all probability destroyed as well and not merely compressed. Thus the electrical condition of the different paralyzed muscles may determine the question of surgical treatment.

In another case, to be described more fully later, in which there was no paralysis of voluntary motion, although there was an area of total anæsthesia and a loss of bladder and rectal control, the absence of electrical changes in the muscles in connection with the other symptoms was sufficient to locate the lesion in the lowest sacral segments and exclude one in the cauda equina which had at first been suspected. And this fact also determined the question of surgical interference. Hence it is absolutely necessary in every case of supposed spinal lesion to test both the power and the electric reaction in each muscle separately.

II. In regard to the tendon reflexes, it is to be remembered that the normal or increased state of the reflex implies the integrity of the segment of the spinal cord governing that reflex. Thus if the knee-jerk is present the lumbar segment is in a normal state, is not destroyed by the disease. If the knee-jerk is exaggerated, it must be either because the controlling action of the brain is cut off, as by an injury to the cord higher up than the second lumbar segment, or because the second lumbar segment is irritated by contiguous inflammatory disease. The former will be ascertained by the existence of other motor and possibly of sensory symptoms. The latter will be evident from the history of the progress of the disease, which must show evidence of acute or chronic myelitis. In the latter condition the exaggeration of the reflex will soon be succeeded by its diminution and its loss.

When a reflex is lost it may imply destruction of the segment of the cord governing that reflex; but it does not necessarily do so. For if impulses to or from that segment do not pass along the nerves, the reflex act will not be performed. Hence it is necessary to examine for an area of anæsthesia corresponding to the sensory nerve along which the impulse is carried to the segment; or for a condition of paralysis with atrophy and reaction of degeneraton in the muscles supplied by the nerve which conveys the impulse from the segment. If neither of these is found alone, the segment is probably diseased.[1] The condition usually found in an injury at one level of the spinal cord is a loss of reflex acts performed by the injured segments, and an exaggeration of the reflex acts governed by the segments below the level injured. Thus in the case just mentioned of destruction of the second and third lumbar segments the knee-jerks were lost, but ankle-clonus was excessive on both sides.

What has been said regarding the knee-jerk is true of all the tendon reflexes, and also of the automatic action of the bladder and rectum.

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[1] For an interesting study of tendon reflexes the reader is referred to "Lectures on Diseases of the Nervous System," by Dr. Thomas Buzzard.

III. In regard to the exact extent of the anæsthesia in lesions at various levels the following diagrams will convey a better idea than any description.

The first diagram (Fig. 38) shows the area of anæsthesia when the lowest extremity of the cord or the nerves to it are involved, viz., the coccygeal and fifth and fourth sacral segments and nerves. This area includes the perinæum and genital organs. When these segments are involved the action of

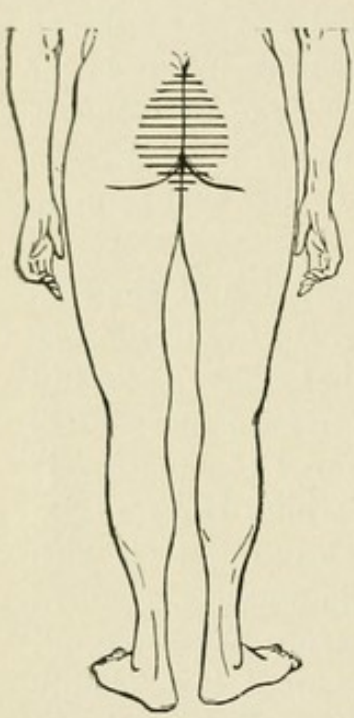

FIG. 38.—Area of Anæsthesia in Lesion of V. and IV. Sacral Segments.

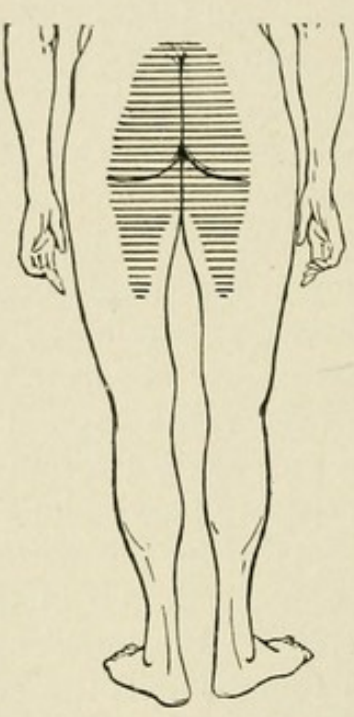

FIG. 39.—Area of Anæsthesia in Lesion of V., IV., and III. Sacral Segments.

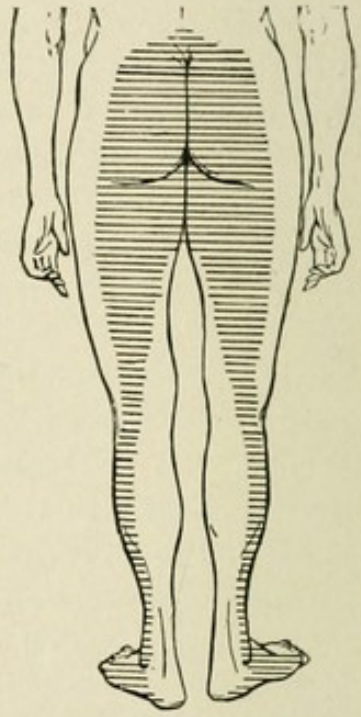

FIG. 40.—Area of Anæsthesia from Lesion at V. Lumbar Segment.

the bladder and rectum is always imperfect, and if they are destroyed all control is lost.

The second diagram (Fig. 39) shows the area of anæsthesia when the lesion involves the third sacral segment as well as those below it.

The third diagram (Fig. 40) shows the area of anæsthesia when the last lumbar and all the sacral segments are destroyed. It will be noticed that up to this level all anæsthesia is on the back of the limbs.

The fourth and fifth diagrams (Figs. 41 and 42) show the area of anæsthesia when the lesion lies as high as the third lumbar segment. The inner side of the leg and thigh as well as the back are anæsthetic.

From this point upward to the second dorsal segment the band of anæsthesia about the trunk will correspond to the nerve arising from the highest segment invaded. Thus in a lesion of the second lumbar segment, the level of anæsthesia

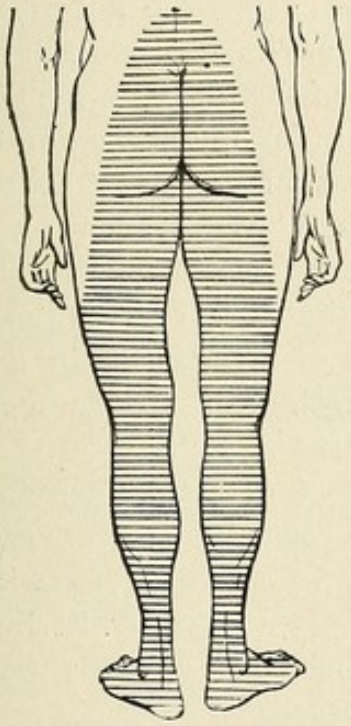

FIG. 41.—Area of Anæsthesia from Lesion at III. Lumbar Segment. Back.

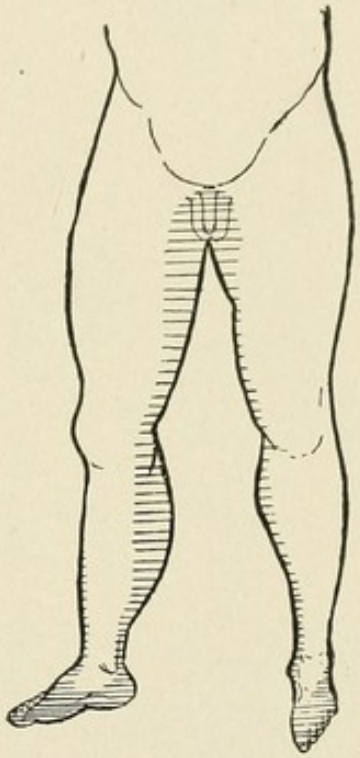

FIG. 42.—Area of Anæsthesia from Lesion of III. Lumbar Segment. Front.

follows the ilio-inguinal nerve, and the entire front of the thigh is included in the insensitive area. At the dorsal segments it corresponds to the level of the intercostal nerves (Fig. 43).

The areas of anæsthesia produced by lesions of the cervical portion of the cord are shown in Figs. 44, 45, and 46.

When the lower portion of the cervical enlargement is involved the area of anæsthesia reaches the armpits on the trunk at the sides, and follows the line of the second dorsal intercostal nerve around the back. In front it is lower than behind, because the upper part of the chest is supplied by the supra-

clavicular nerves from the upper cervical segments. The inner side and back of the arm in its lower half is anæsthetic, and the zone of anæsthesia extends down the inner side and back of the forearm and over the back of the hand, and of the little, ring, and one-half of the middle fingers. In Fig. 44, which represents the area of anæsthesia in a case of lesion of the seventh and eighth cervical segments and the first dorsal segment described by Thornburn,[1] the anterior surface of the arm and the radial side of the forearm, the thumb and index finger had not lost sensation. A case recently reported by Macewen[2] presented identical symptoms. In Fig. 45 is represented the area of anæsthesia in a case of lesion of the same level described by Herter.[3] In a case of cervical pachymeningitis reported by Ross (*Brain*, Vol. VII., p. 68, 1884) without autopsy, the same distribution of sensory disturbance was observed.

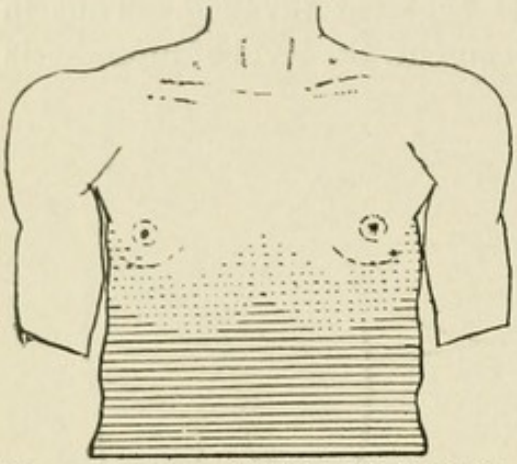

FIG. 43.—Transverse Myelitis at VI. Dorsal Segment; Area of Anæsthesia. Case XXV.

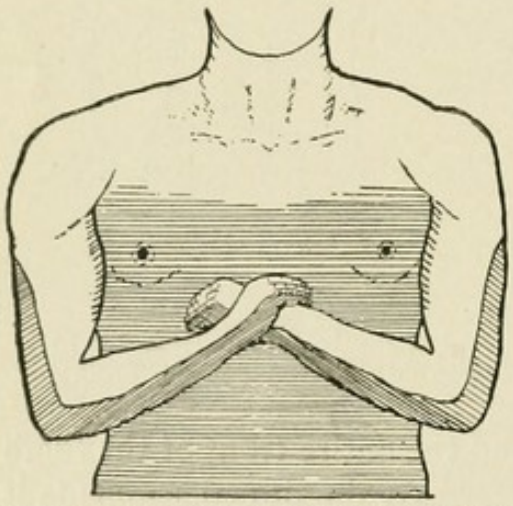

FIG. 44.—Injury at VII. Cerv. Segment; Area of Anæsthesia. (Thorburn.)

When the middle of the cervical enlargement is invaded and the sixth segment of the cord is destroyed, the area of anæsthesia is about the same upon the trunk, but invades the entire arm below the insertion of the deltoid. This is shown in Fig. 46. This case was one of Brown-Séquard paralysis

[1] Thornburn: "A Contribution to the Surgery of the Spinal Cord," p. 39.

[2] Macewen: Brit. Med. Jour., April 5th, 1890.

[3] Herter: Jour. of Nerv. and Ment. Dis., January, 1890. These cases confirm the theoretical statements of Ross, Brain, 1887, p. 342.

from caries of the fourth cervical vertebra with displacement and pressure upon the left side of the spinal cord. It is fully described in the next chapter (Case XXVIII), and the lesion was as high as the sixth segment. The patient died of respiratory paralysis, when the disease invaded the fourth and third segments of the cord, and as death usually occurs from this cause

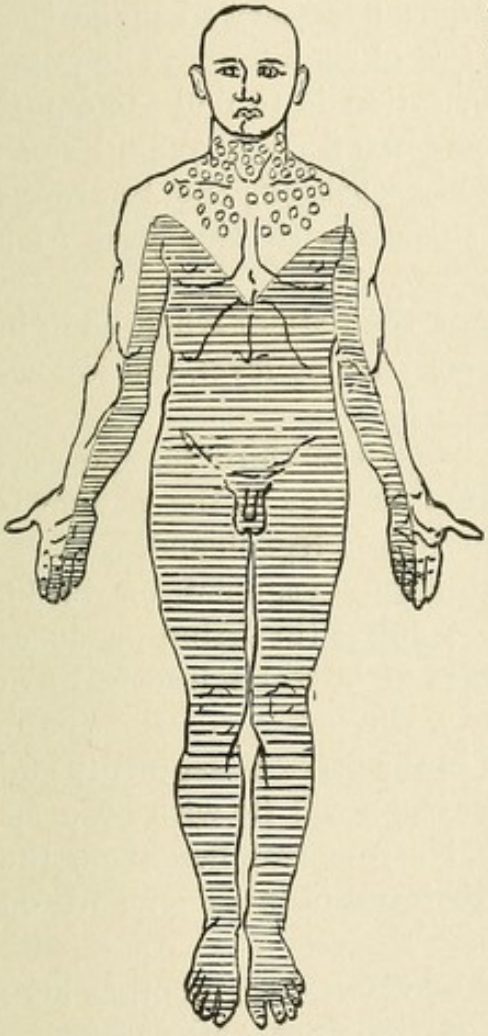

FIG. 45.—Injury at VII. Cerv. Segment; Area of anæsthesia in lines; of hyperæsthesia, dotted. (Herter.)

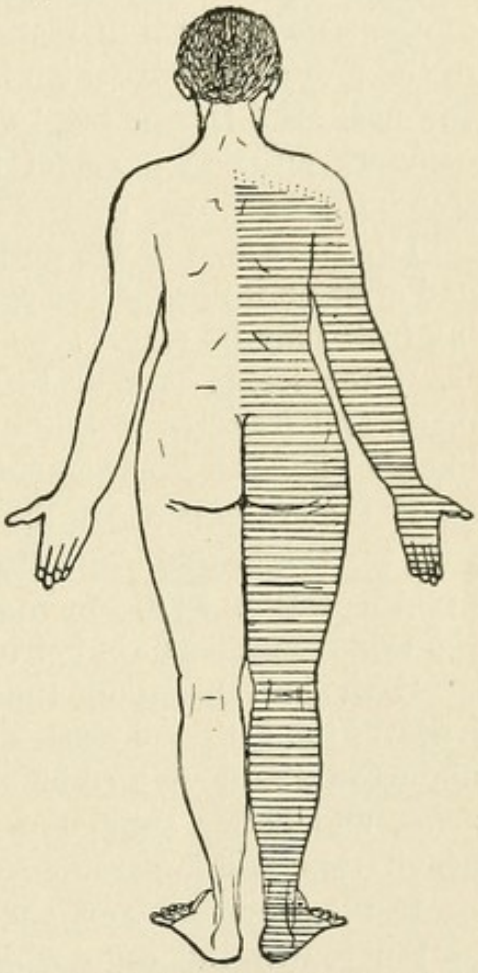

FIG. 46.—Injury at VI. Cerv. Segment, on one side only. Area of anæsthesia in lines; area of hyperæsthesia dotted. Case XXVII.

when the disease is situated at this level, no opportunity is offered for the determination of sensory disturbances in such cases.

It will be seen from a review of these diagrams that the investigation of the exact area of anæsthesia is of great importance in determining the level of the cord which is involved. The tests are to be made by touching the surface simul-

taneously on both sides at symmetrical spots with cotton wool, by pricking it with a needle, and by touching it with a test-tube containing both cold and hot water. The great individual variations in the perceptions of the points of a compass—distinguishing two points from one—and the careful attention on the part of the patient necessary to make this test, has led me to discard it as a test of sensation. The point of chief importance in testing sensation is to find some part of the patient's body which is unaffected (*e.g.*, in spinal lesions the neck, the face being too keenly sensitive), and to always compare affected parts of the surface with this part as a standard.

IV. In regard to the position of the patient when lying in bed after an injury of the spinal cord at one level, the following facts may be stated:

If the lesion is near to and below the third lumbar segment, and irritates it but does not destroy it, the patient is likely to lie with the thighs drawn up and legs flexed.

If the lesion is above the third lumbar segment, he will lie with the thighs and legs extended; unless the lesion produces great irritation, as in the last stages of lateral sclerosis, when the thighs and legs are flexed and adducted.

If the lesion is about the two lower cervical segments and irritates the fifth and sixth segments, he will lie with his arms abducted about at a right angle, his forearms flexed on the arms, and hands supinated or their position dependent on gravitation, and fingers flexed.[1]

If the lesion is about the middle cervical segment, as high as the fourth, the arms will lie at the sides and cannot be moved, as occurred in my second case of Brown-Séquard paralysis (Case XXVIII.).

A word of explanation will show the reason for these positions. Supposing two muscles of opposing action to be set in action at one and the same time, the stronger will overcome the weaker. Suppose one of these to be paralyzed, as soon

[1] Examples of this position are given by Thorburn, l. c.; Herter, l. c.; and Macewen, l. c.

as the other acts once voluntarily or when by irritation of its cells it is kept in action, the resulting position cannot be corrected, for its opponent will never contract again voluntarily. In either case the result is a fixed position not usually the normal one.

In cerebral paralysis, when voluntary movements are cut off from the arm, the arm is adducted, the forearm is held in a flexed and supinated position, and the fingers are flexed; because the balance of power between the abductors, extensors, and pronators of the forearm and the adductors, flexors, and supinators is in favor of the latter; all the impulses to these muscles originating in the spinal cord. For the same reason the leg is held rigid, somewhat adducted and extended, in cerebral paralysis and in lateral sclerosis and in transverse lesions of the cord when these are above the lower dorsal region. Therefore the position of the affected limbs in cases of hemiplegia, and of paraplegia from lateral sclerosis, is the same and becomes a diagnostic symptom of an interference of conduction of voluntary impulses from the brain to the spinal cord—a symptom of cerebral paralysis, and of what has been termed the first type of spinal paralysis.

In spinal paralysis from destruction of the gray matter, that is, in the second type of spinal paralysis, the balance of power between muscles holding the limb in position is interfered with, because one or both sets of muscles have entirely lost their tone. If one set alone is paralyzed the other acts unopposed. Thus in a transverse lesion of the lower half of the cervical enlargement, the arms are abducted, flexed, and supinated, because the muscles producing these movements are not paralyzed, and contract voluntarily and reflexly, while their opponents, the adductors, extensors, and pronators, are totally paralyzed and do not act. Such a condition in the leg leads to various forms of talipes. If both sets of muscles are paralyzed together, as in many cases of infantile spinal paralysis, or in general myelitis, the limb hangs flaccid and not rigid, and falls into any position from the action of gravitation. The ligaments in such a limb soon become overstretched, and hence extraordinary positions can

be given to a limb passively in such cases. Examples are found in the partial dislocation of the head of the humerus in infantile paralysis of the shoulder muscles; and in the abnormally free motion of the hip and knee when the muscles moving them are paralyzed. When firm contractions with malpositions—such as occur in the forms of talipes—are produced in infantile paralysis, it is because one of the muscles moving the joint is not entirely paralyzed. A contracture does not result when there is total paralysis of all the muscles moving a joint.

* * * * * * *

In the majority of cases of spinal injury with crushing of the cord or with acute transverse myelitis, and also in cases of slow compression of the cord, the four classes of symptoms here described are present together. It is thus possible to confirm or to rectify a diagnosis formed from the study of one class of symptoms by that of the others; and thus to reach a certainty in the diagnosis of the location of the spinal lesion.

THE FUNCTIONS OF THE SPINAL SEGMENT.

Thus far the consideration of the localization of functions in the spinal cord has been directed to the various segments as they lie one beneath another throughout the length of the organ. This study would be incomplete were the functions of the various portions of each segment not mentioned briefly. The appearance of a cross-section of the cord in the cervical region is shown in Fig. 47, and the division of the white columns into tracts and of the gray matter into anterior and posterior horns and central or intermediate gray substance is familiar.

In the gray matter the anterior horns contain the groups of cells whose various functions in controlling both the action and nutrition of different muscles has already been studied. It is thought that the anterior and lateral groups of cells in the horn preside over the muscles controlling "fundamental motions" common to all vertebrates, such as flexion and extension of joints; while the median and central groups of cells

preside over "accessory motions" peculiar to monkeys and to man, such as the finer movements of the hand, fingers, and thumb, the nice motions of the ankle and toes in walking.[1] In the central and intermediate gray matter of the cord lie the vaso-motor centres which control the circulation and nutrition of the skin, nails, joints, bones, and viscera. The separate existence of motor and trophic cells is still unproven, but disease in the anterior or posterior horns alone is not followed by trophic or vaso-motor changes in the parts

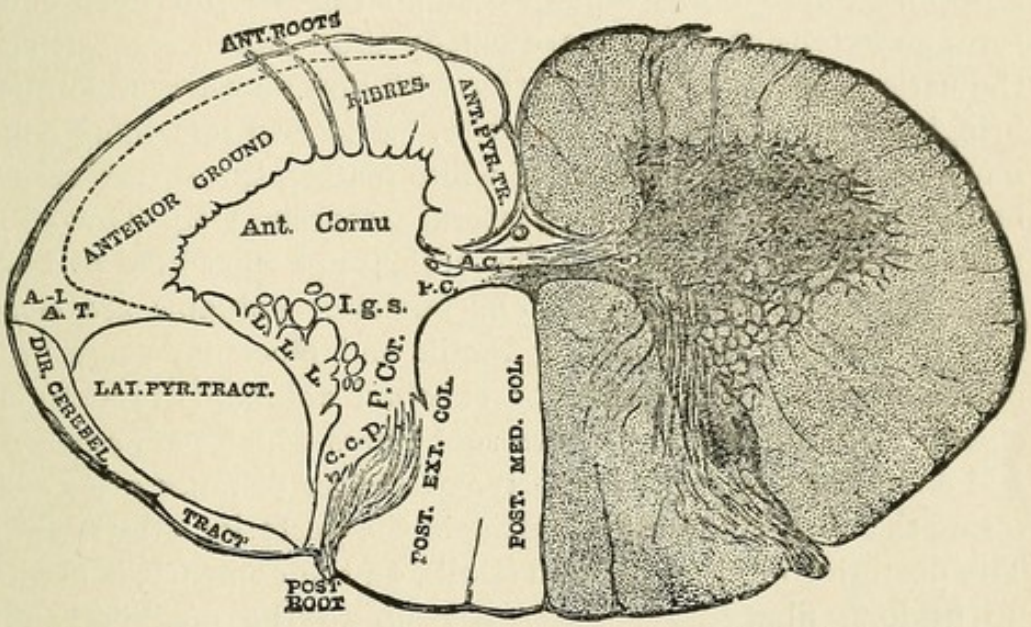

FIG. 47.—Diagram of a Section of the Spinal Cord in the Cervical Region. (Gowers.) *A.C.*, Ant. commissure; *P.C.*, post. commissure; *I.g.s.*, intermediate gray substance; *P.Cor.*, post. cornu; *c.c.p.*, caput cornu post.; *L.L.L.*, lateral limiting layer; *AL–AT*, antero-lateral ascending tract.

just named, while in disease limited to this intermediate gray matter (syringo-myelitis) the vaso-motor and trophic changes form a very important group of symptoms. And the definite group of cells known as the vesicular column of Clarke, which lies at the junction of the posterior horn with the intermediate gray matter, has been proven by Gaskell to have vaso-motor functions. In the posterior gray horns the cells do not lie in groups, but are scattered through a dense felt-like structure of fine fibres in which the sensory nerve roots terminate.

The white columns of the cord have been separated into

[1] This distinction is made by Ross and supported by the observations of Spitzka.

tracts by means of the embryonal studies of Flechsig, and of the pathological studies of numerous investigators from Türck to Gowers.

The anterior median column, which comes from the anterior pyramid of the medulla of the same side on which it lies, and the lateral pyramidal tract, which comes from the anterior pyramid of the medulla of the opposite side, serve to transmit voluntary impulses and inhibitory impulses from the motor region of the brain to the motor cells of the cord. The anterior ground fibres which surround the anterior horn and form an extensive column are chiefly short fibres associating the various levels of gray matter with one another and thus bringing their action into harmony. The two remaining columns of the lateral portion of white matter, the direct cerebellar column, and the antero-lateral ascending tract (Gowers) transmit impulses upward to the brain; but what kind of impulse is still undetermined. The posterior columns are sensory in function, transmitting tactile and muscular sense impulses upward. The posterior external column (Burdach) is chiefly made up of short nerve root fibres which pass upward and downward in it for a shorter or longer distance after entering the cord before terminating in the posterior gray horns. The posterior internal column (Goll), on the contrary, is made up of long fibres passing directly up to the medulla, and thence to the brain by way of the lemniscus. In this column the sensations from the legs go up in the fibres lying near the periphery of the cord; while those from the arms go up in fibres lying near to the posterior gray commissure.

It is of course possible that each of these tracts may be diseased alone. As a matter of fact, there are but two spinal affections in which such a limitation of disease to the white tracts is found, viz., lateral sclerosis and locomotor ataxia. The symptoms of lateral sclerosis have been incidentally described already under the title of spinal paralysis of the first type (p. 130). Those of locomotor ataxia will receive attention in a subsequent chapter.

It is also evident that all these tracts will be diseased together in transverse lesions of the cord, whether due to hem-

orrhage or to slow compression or to any other cause. The chief symptoms of such lesions have been already considered and need not be restated. It is always to be remembered, however, that in a transverse lesion of the cord at any level, the paralysis and anæsthesia in the parts below the lesion are due to the destruction of the white columns which should transmit voluntary impulses and sensory impressions to and from those parts, but which are cut off by the lesion.

In any case of spinal disease where it is desirable to localize accurately the lesion, it is suggested that a written summary of the symptoms be compared with the table of localization of the functions of the cord, when it will become evident, by contrasting the normal with the abnormal conditions, what part of the cord is affected. As Bramwell justly observes, "the essence of the clinical examination of the spinal cord consists in the systematic and separate examination of each spinal segment, by observing the motor, sensory, reflex, vasomotor, and trophic conditions of its body area." Such an examination will lead to accurate diagnosis of local lesions.

If that lesion is one which can be removed, such as compression by a tumor, or by a displaced vertebra, it is important to remove it as soon as possible. For experience has shown that a long-continued compression is followed by ascending and descending degeneration of the white columns of the cord; and this condition is believed to be permanent, and remains after the pressure is removed. Hence in spinal surgery, prompt action is of great importance. The fact is illustrated by the history of the first case of Brown-Séquard paralysis (Case XXVI.), in which an earlier operation might have resulted in a greater degree of improvement than was obtained.

CHAPTER XI.

THE LOCALIZATION OF SPINAL CORD DISEASES (CONTINUED).

Cases of lesions at various levels of the cord.—Lower sacral.—Sacral and lower lumbar.—Upper lumbar.—Cases of lesion of the cauda equina contrasted with those of the lower segments of the spinal cord.—Case of Brown-Séquard paralysis at the ninth dorsal level. Operation. Improvement.—Cases of transverse lesion in the ninth and sixth dorsal regions.—Cases of transverse and unilateral lesions at the lower and upper cervical levels.

CASES OF TRANSVERSE LESIONS AT VARIOUS LEVELS OF THE CORD.

In the last chapter the principal facts which enable one to locate a spinal lesion have been stated. In this chapter a number of typical cases of spinal lesion at different levels, from below upward, are described in order to illustrate the possibility of locating a spinal lesion when the foregoing facts are kept in mind.

CASE XIX.—HEMORRHAGE INTO THE SPINAL CORD INVOLVING THE COCCYGEAL AND FIFTH AND FOURTH SACRAL SEGMENTS—SECONDARY MYELITIS ADVANCING UPWARD TO THE SECOND SACRAL SEGMENT.

A young woman, previously healthy, was suddenly seized, after long standing and walking, on May 1st, 1889, with severe pain in the sacral region and down the back of the legs; with retention of urine and fæces, so that catheterization was necessary from the first, and the bowels only moved after enemata; and with total anæsthesia of the vulva and vagina and

perinæum, and of the back over the area shown in Fig. 38. For six months this condition remained. The pain became dull and less severe in the back, but in an area on the back of the left thigh, corresponding to the region shown in Fig. 39, it was very intense. The urine was retained if not drawn, or would dribble away when the bladder had become full. Defecation was always attended with pain. The anæsthesia was permanent. She felt rather weak but was not paralyzed, and could perform all the motions of the feet and walk well. There was no atrophy of the limbs, no girdle sensation, and no electrical changes.

I saw her, with Dr. F. J. Quinlan, in October, 1889, and in addition to the symptoms mentioned, found the knee-jerks exaggerated, the plantar and gluteal reflexes normal and no ankle-clonus. A careful exploration of the pelvis failed to reveal anything abnormal. The diagnosis was made of hemorrhage into the spinal cord, and its situation was evident from the distribution of the anæsthesia. The pain in the left thigh indicated that some inflammatory or sclerotic process was advancing from the fourth into the third sacral segment on the left side.

I saw her again, in February, 1890. Then the subjective symptoms had increased. She had less pain but more numbness, and the anæsthetic area was found to have extended down both thighs and corresponded quite exactly to the area shown in Fig. 39, being a little larger on the left side. In addition, a slighter degree of anæsthesia was found on the outer side of the left leg in the area shown in Fig. 40. In the peroneal group of muscles on this leg, the faradic contractility had diminished from 45 to 60°.[1] It was therefore evident that the inflammatory changes had advanced upward in the cord, had involved the entire third sacral segment and a part of the second sacral segment on the left side. As the area of the thigh had become insensitive, it had also become less painful, showing that the irritative lesion had been fol-

[1] An arbitrary scale. This fact would show that the peronei were the muscles represented lowest down in the cord at the second sacral.

lowed by a destructive lesion in the third sacral segment. The same objective symptoms persisted. In spite of the anæsthesia over the sacrum, there was no tendency to bed-sore, and in spite of the paralysis of the bladder, there was no cystitis. She could walk well and there was no paralysis, but she found her left leg grew weary more quickly than before, a fact which corroborated the weakened state of the peroneal muscles found by the electric examination. Counter-irritation by cautery and cups, galvanism to the spine, iodide of potash, and ergot had been faithfully employed but had not arrested the progress of the disease. The possibility of a growing tumor—which might have been removed—was ruled out on account of the sudden onset and the absence of signs of pressure upon the cauda equina, whose nerves lie all about the sacral region of the cord. And the case was therefore thought to be hopeless.

CASE XX.—COMPRESSION OF CAUDA EQUINA FROM POTT'S DISEASE—SUBSEQUENT MYELITIS OF LUMBAR AND SACRAL REGIONS.

The patient[1], a lady in middle life, was seen in June, 1887, in consultation with Dr. V. P. Gibney, who was treating her at that time for Pott's disease with marked deformity and projection of the first lumbar vertebra.[2] Her legs were paralyzed, toe, ankle, and knee joints could not be moved voluntarily at all, and the motions of the hip joints, though possible in all directions, were weak, especially upon the left side. There was much atrophy and constant fibrillary twitchings of the paralyzed muscles; and in all the muscles of the legs and back of the thighs, including the glutei, there was a reaction of degeneration. There was loss of tendon reflexes at the knee and ankle, and of the abdominal and gluteal reflexes. The area of anæsthesia was that shown in Figs. 41 and 42,

[1] Cases quite similar to this one have been recorded by Kirchoff, Arch. f. Psych., xv., p. 607; by Bernhardt, Berlin Klin. Wochen., Aug. 8th, 1888; by Osler, Medical News, Dec. 15th, 1888; by Oppenheim, Arch. f. Psych., 1889, xx., p. 298, with autopsy; and by Mills, Medical News, March 1st, 1890, Case II.

[2] See Med. Record, Oct. 1st, 1887; Dr. Gibney's report of this case.

tactile sensations being lost and temperature and pain senses being much impaired. On the front of the thighs, to the knees and for a short distance below the knees on the front of the leg, in the exact area which is not shaded in Fig. 42, she was very hyperæsthetic, the least touch or cold application giving her great pain. She had no girdle sensations and no symptoms above the level of the hip joint in front. She lay with legs extended, unable to move them. Her bladder and rectum were under control for the first two months of her illness, during which time these symptoms had gradually developed, but then retention of urine and constipation set in, and soon after a bed-sore developed over the sacrum. She gradually grew worse during the summer and died of exhaustion in February, 1888. The interesting feature in this case is the existence of a hyperæsthetic area on the front of the thighs. This is perfectly homologous to the band of hyperæsthesia found around the trunk above the anæsthetic band in cases of dorsal myelitis, and is evidence of implication of the cord as distinct from a lesion compressing the cauda equina. When pain is present in cauda equina lesions, it is felt in the anæsthetic area, *not* in the area supplied by the nerves whose origin is higher, as in myelitis.

Other points of differentiation manifest in this case were the great atrophy, fibrillary twitchings, and reaction of degeneration in the muscles paralyzed, which indicate spinal cord disease and are less marked in cauda equina compression. Fibrillary twitching is not frequently seen in nerve injuries. It is quite common when the gray matter of the cord is diseased. The loss of bladder and rectal control is also very common in spinal cord lesions and uncommon when the cauda equina is compressed.

CASE XXI.—FRACTURE OF THE LOWER DORSAL SPINE—PARAPLEGIA FROM SPINAL INJURY AT THE SECOND LUMBAR SEGMENT—THE QUESTION OF OPERATION.

The patient fell off a load of hay and broke his back in November, 1888. For eight months he was confined to bed, with total paralysis of motion and sensation below the waist

and of bladder and rectal control. Then he gradually improved, and in December, 1889, was able to walk with help, and came to the city to ascertain whether an operation upon the spine would relieve his paralysis. The question was referred to me for decision by Dr. McBurney.

Though able to stand and to drag his feet along, his powers of motion in the lower extremities were very bad. The anterior thigh muscles were atrophied, relaxed, flabby, and showed the reaction of degeneration. The other muscles of the thighs and legs were rigid and moved with difficulty, though their size and electric response were good. The legs were cold and cyanotic. His abdominal and patella reflexes were lost. Ankle-clonus was very active on both sides.

His urine dribbled away constantly, the sphincter being paralyzed and his rectum could only be emptied by enemata. His sensation was much impaired below the level of a band passing around the trunk along the course of the ilio-hypogastric nerves, above which a zone of hyperæsthesia one inch wide was found. All sensations below this band were impaired, those of touch and pain to a considerable degree. He was hypersensitive to cold, which was painful, but he did not feel hot objects as hot. When any part of his leg was touched he referred the sensation to some part of the thigh. His knowledge of the position of his limbs was good. In walking, his gait was spastic, the feet were dragged, the knees overlapped, and the trepidation from the feet would often become so great as to necessitate his stopping for a moment.

There was a marked deformity with projection backward of the spines of the ninth, ten, and eleventh dorsal vertebræ.

The symptoms pointed to a compression or a destruction of the spinal cord at the second lumbar segment, which lies opposite the eleventh dorsal vertebra, and their duration made it probable that secondary degenerations had occurred. The important question was whether there was a compression only or also a destruction of the cord. The atrophy and reaction of degeneration in the anterior thigh muscles, the loss of knee-jerk and the zone of total anæsthesia in the region of the ilio-hypogastric nerve, were thought to show the existence of

the destruction of the cord at that level. Hence the mere removal of compression was not thought advisable, since a recovery from the paralysis was not possible, as the cord was destroyed.

In contrast with these cases, in which the sacral and lumbar segments of the spinal cord were diseased, the following cases are of interest. They are cases of injury of the cauda equina, in which, however, the spinal cord was not affected. Thorburn[1] and Mills[2] have directed attention to the possibility of differentiating spinal from caudal injuries. The chief features of difference between the two classes of cases are shown in the following cases.

CASE XXII.—HEMORRHAGE INTO THE SPINAL MEMBRANES OF THE CAUDA EQUINA—NO LESION OF THE SPINAL CORD.

A healthy man awoke, on November 10th, 1889, with tingling and numbness in the back of both legs, especially in the left one. The sensation was that of a foot asleep, and it extended up the back over the sacrum, down the back of the thighs, and over the outer part as well as the back part of the legs and soles of the feet. The perinæum and posterior part of the scrotum were also numb and the rectum was partially insensitive. This condition had remained about stationary, when I saw him, in January, 1890, and was the only symptom from which he had suffered. An examination showed an area of hyperæsthesia corresponding to that shown in Fig. 41, the anterior part of the thigh and the inner part of the leg and foot having escaped. In the hyperæsthestic area, there was much pain and tingling produced by the slightest touch, and a very great hypersensitiveness to pain and to heat and to electricity. He was not paralyzed, his motor power and coördination were good, his muscles showed normal electrical reaction, and had not atrophied. His reflexes were normal and he had no loss of bladder or rectal control and no pain.

[1] Thorburn: Brain, 1888.

[2] Mills: Medical News, June 15th, 1889, and March 1st, 1890.

He had suffered from bleeding piles, which had been cured just before the onset of these symptoms.

The tingling and hypersensitiveness certainly indicated an irritation of all the sensory nerves of the sacral plexus. But the absence of paralysis, atrophy, and bladder symptoms and the preservation of normal reflexes negatived any spinal cord disease. A careful pelvic examination by the rectum failed to show anything abnormal there. And the sudden onset of the symptoms and their stationary character ruled out the possibility of either a pelvic or a spinal tumor. The posterior roots of the sacral plexus must have been involved in a sudden pressure, and the only diagnosis remaining was a hemorrhage in the meninges about the cauda equina, and chiefly on its posterior surface; a diagnosis which was aided by the probability of unusual venous congestion of the caudal veins in consequence of the suppression of a customary hemorrhage from the piles.

This case is interesting, since it serves to indicate the necessity of some care in diagnosis. The sensory symptoms alone might have been due to spinal cord disease and have led to its diagnosis; but the absence of motor and reflex disturbances and the integrity of the automatic action of the bladder and rectum showed conclusively that the cord itself was not the seat of disease.

CASE XXIII.—TUMOR (?) COMPRESSING THE CAUDA EQUINA.

The patient had strained his back in lifting a heavy weight in August, 1889. He soon began to suffer from pain in the sacral region, which has gradually increased to the present time (April, 1890). This pain has been extending (always being greater on the left side) at first into the area represented in Fig. 39, then down the entire back of the thighs and legs; and now he has considerable pain over all the region indicated in Fig. 41; a girdle sensation running along the ilio-inguinal area and a moderate degree of anæsthesia in the region represented in Fig. 40. During his illness he has become progressively weaker and thinner in his lower limbs and now

walks with a shuffling gait. The muscles do not show any change of electrical reaction. His knee-jerks are increased. His bladder and rectum act slowly, but he has not lost control over them.

The gradual onset indicates either a tumor or chronic meningitis or a chronic myelitis. The position of the girdle sensation is such as to locate a myelitis as high as the second lumbar segment—a point opposite the eleventh dorsal vertebra—a point much higher than the seat of pain. The hypothesis of a myelitis here is negatived by the preservation of the reflexes, by the preservation of electrical reaction and absence of much atrophy, and by the absence of pain and anæsthesia about the front of the thigh and leg, which are related to the upper lumbar region, and by the fact that the symptoms are more on the left side. Thorburn says that if symptoms are asymmetrical we have to do with an affection of the cauda rather than of the cord. The slow progress

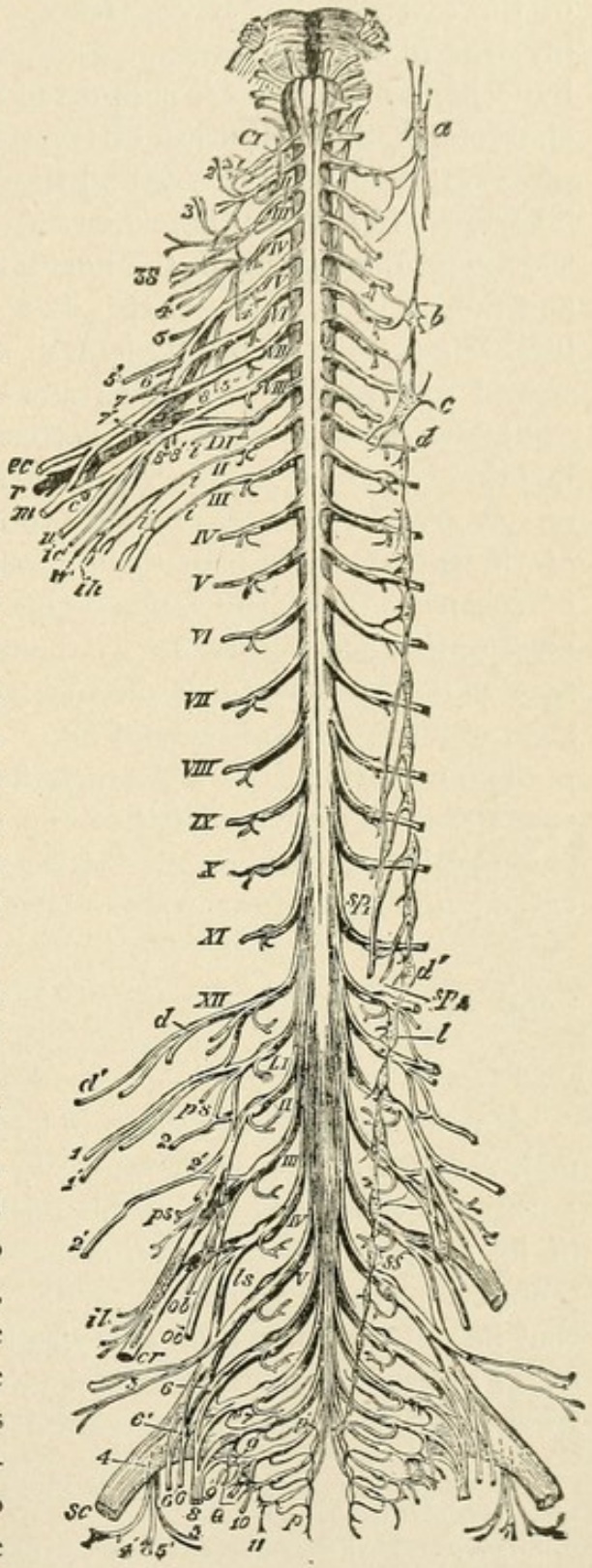

FIG. 48.—The Spinal Cord, the Spinal Nerves, the Cervical, Lumbar, and Sacral Plexuses, and the Cauda Equina. (Ferrier.)

of the case also negatives the hypothesis of a myelitis. In myelitis of such duration bed-sores would be expected. The hypothesis of a tumor is supported by the progress and by the character and distribution of the symptoms, especially by the preservation of the reflexes and the control over the sphincters. Thorburn[1] has shown that when the cauda equina is compressed the symptoms appear in the nerves which lie within the cauda and not on its surface—that is, in the sacral rather than in the lumbar nerves. This statement is supported by the cases observed by Osler[2] and by Mills.[3] When, therefore, the area of anæsthesia is as extensive as that shown in Figs. 40 and 41, but the degree of paralysis is too slight and the preservation of reflex and automatic activity too great to indicate a lesion of the spinal cord in the third lumbar segment, the diagnosis of compression of the cauda equina by tumor or by chronic meningitis seems warranted. This will be confirmed if the seat of pain is low down on the back, much lower than the level of a spinal lesion would be.

The importance of differentiating tumors compressing the cauda equina from myelitis of the spinal cord cannot be exaggerated, since operations for the former condition are indicated, while operations for the latter are impossible.

CASE XXIV.—ACUTE MYELITIS OF THE LUMBAR AND SACRAL REGIONS.

Martin M., aged twenty-two, had been a perfectly healthy man until the onset of his illness in April, 1881. While lifting a heavy weight he suddenly felt a sharp pain in the lower part of his spine, a numb sensation beginning at the hips and descending to the feet, and a feeling of weakness in his legs so that he fell to the ground and was unable to rise or move his lower extremities. He was picked up and taken to a hospital, where all the symptoms of complete paraplegia were found.

[1] Thorburn: Brain, 1887, vol. x., p. 381; see especially Cases II. and III., which resemble this case in every particular.

[2] Osler: Medical News, 1888, vol. iii., p. 669.

[3] Mills: Medical News, 1890, vol. vi., p. 217.

For a week after the attack he had total loss of sensation and of motion in the lower half of his body, and incontinence of urine and fæces; and within three weeks large bed-sores had developed over the sacrum and on both trochanters. There was a slight return of sensation during the second week, but no recovery of motor power; and within a month a constant fibrillary tremor in the muscles of the legs was noticed, which has continued.

He was transferred to Bellevue Hospital in November, 1881, seven months after the onset of the paraplegia, in a condition in no way different, according to his own account, from that present the day after the attack.

Examination revealed the following facts:

The patient is a large, fat man, well nourished, and shows no traces of suffering. He is totally paralyzed in the lower extremities and trunk below the middle of the spine. The muscles are relaxed, there is no rigidity or contracture, there is a marked fibrillary tremor, and the muscles are much atrophied, though the development of fat preserves the contour of the limbs. All reflexes are abolished. There is a total loss of faradic contractility, and reaction of degeneration in all the muscles of both legs.

Sensations are almost wholly lost below the level of the ninth dorsal nerve, although he is aware of deep pricking in the right leg, feeling slightly the touch of the sharp instrument, but no pain. Temperature and pain sensations are wholly lost, but he is aware of the tremors in the muscles of the legs, and can estimate imperfectly the position of the limbs. A zone of hyperæsthesia two inches wide extends around the body above the ninth dorsal nerve.

There are no vaso-motor or trophic disturbances in the legs, but the large bed-sores over the thighs and sacrum remain. These have exposed the bones, but the parts are insensitive. There is incontinence of urine, and a chronic cystitis is present. The fæces are passed involuntarily.

The patient remained in a stationary condition for a month. He then developed an acute purulent myocarditis—described by Dr. Wm. H. Welch before the American Associ-

ation of Physicians and Pathologists, 1886—and died suddenly of heart failure.

Autopsy.—There was considerable opposition to a post-mortem examination by the friends, so that it was impossible to obtain the entire spinal cord. The portions below the first dorsal vertebra were, however, secured. The pia mater covering the cord was congested. At the level of the tenth dorsal segment of the cord a region of marked softening was found about half an inch in length. Section at this point showed such a degree of disintegration that the gray matter was not to be distinguished from the white columns. There was no evidence of hemorrhage into the cord, and no deposit of pigment. Microscopic examination of the fresh cord at the lesion showed the presence of Gluge corpuscles in large numbers. An ascending degeneration was evident to the naked eye. No descending degeneration was to be seen, but the entire cord below the lesion was somewhat softened and the limitation of the anterior cornua was indistinct.

The cord was hardened in Müller's fluid and preserved in alcohol. Microscopic examination of sections made at various levels revealed the characteristic lesions of an acute myelitis.

There was a great distention of all the intra-spinal vessels and a marked thickening of their walls. No hemorrhages were found. There was a total destruction of the gray matter of the anterior cornua and central portion of the cord from the level of the tenth dorsal segment downward. The posterior cornua were destroyed at the tenth dorsal segment, but had escaped in the parts below. The normal tissue was replaced by a dense mass of connective-tissue in which numerous nucleated cells were found. There was no trace of nerve cells. The columns of the cord were sclerotic, and no trace of nerve fibres was to be found at the level of the lesion. Below the lesion the posterior columns and posterior nerve roots were normal, but all other parts were sclerotic. Above the level of the lesion an ascending degeneration was found in the column of Goll and in the direct cerebellar column, and for a short distance in the column of Burdach also. This also involved a zone of fibres extending forward nearly to the anterior median fissure around

the periphery of the cord. And inasmuch as there was no evidence of meningitis, it must be concluded that the ascending degeneration had involved a portion of the cord not included in the direct cerebellar column but lying anterior to it, viz., the antero-lateral ascending tract of Gowers.

The history of the case shows the difficulty in distinguish-

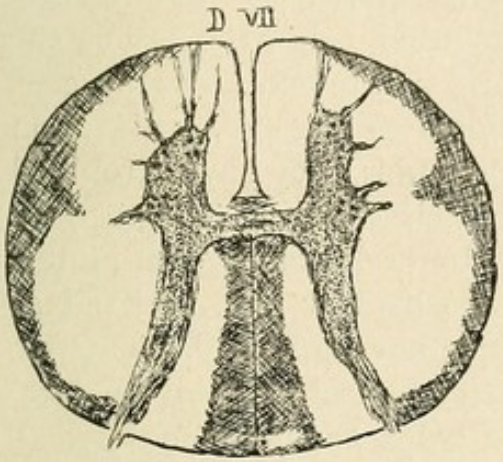

FIG. 49.—Ascending Degeneration in Case XXIV. Col. of Goll, direct cerebellar col., and col. of Gowers were degenerated on both sides.

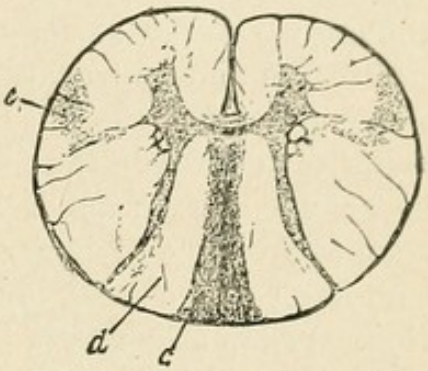

FIG. 50.—Ascending Degeneration in Gowers' Case. *c*, Col. of Goll; *d*, col. of Burdach; *e*, antero-lateral ascending tract or col. of Gowers. (Gowers.)

ing clinically between an acute myelitis and aspinal hemorrhage.

The total paralysis with atrophy, the reaction of degeneration, and the fibrillary tremors in *all* the muscles of the legs were sufficient proof during life of an extension of the lesion to the anterior cornua of the cord in their entire longitudinal extent below the level of the lesion—thus distinguishing the case from simple transverse myelitis; and the zone of hyperæsthesia showed the exact level of the upper limit of the disease.

CASE XXV.—HEMORRHAGE IN THE SPINAL CORD AT SIXTH DORSAL SEGMENT—LOCALIZED TRANSVERSE MYELITIS AT THIS REGION.

The patient, a male, forty-six, was seen in October, 1888, with Dr. L. A. Sayre. On December 4th, 1886, he was suddenly seized with a very severe pain in the middle of the back

and became absolutely paralyzed from the waist down, both motion and sensation being entirely abolished below the sixth intercostal nerve, and the bladder and rectum being beyond control. He has remained in this condition ever since, suffering greatly from pain in the spine between the shoulders, and being incapable of moving a muscle or of feeling any sensation whatever below the level of the sixth rib. His reflexes are all preserved and are exaggerated below the lesion, the only reflex lost being the epigastric skin reflex. The muscles, though paralyzed as far as volitional motions go, contract on percussion and on irritation of the skin; they have atrophied to some degree from disuse, but their electrical contractility is perfect except in the intercostal muscles of the sixth interspace, where reaction is lost. His bladder empties itself as soon as a couple of ounces of urine have collected. The rectum has to be cleared out by enemata or by digital exploration. When any part of his body below the lesion is touched, he says he feels it at the level of the sixth vertebra in the back, but the strongest faradic current causes no pain. The vaso-motor condition is interesting; stroking the skin, or touching it with a hot or cold object or with a faradic brush, causes, immediately, intense redness; so does pressure of any kind, so that he lies naked on a sheet and is turned over every half-hour by two attendants in order to prevent the formation of bed-sores, which the pressure of his limbs have frequently produced and which have been healed with difficulty. Around his body from the sixth to the fourth intercostal space, over a band two inches wide, there is extreme hyperæsthesia (Fig. 43). The absence of any tendency to contractions or to rigidity in the muscles is remarkable. The position of his limbs is governed wholly by gravitation.

The diagnosis in this case was that of a hemorrhage dividing completely the spinal cord and distending the membranes at the level of the sixth dorsal segment.[1]

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[1] Bastian has stated that in complete division of the spinal cord there is no increase of tendon reflexes and no rigidity. This case contradicts the first statement and confirms the second. See Med. Chir. Soc. Rep., Lancet, March 1st, 1890.

BROWN-SÉQUARD PARALYSIS.

The term Brown-Séquard paralysis is given to a condition in which one-half only of the spinal cord is affected at one level. This lesion gives rise to a very characteristic set of symptoms below its level. These are paralysis of motion on one side with preservation or exaltation of sensation, and paralysis of sensation on the opposite side, with preservation of motion. The explanation of these symptoms is evident from Fig. 51. A lesion on the left side of the cord (as in Case XXVI.) cuts off, from the parts below it, all voluntary impulses which come from the right half of the brain and pass down the left lateral column; hence it causes paralysis of motion in the left leg. The same lesion cuts off from the left half of the cord below it, all sensory impulses going up to the brain. But these sensory impulses have crossed over the cord as soon as they have entered it below. The sensations from the left (paralyzed) leg ascend in the right half of the cord and are, therefore, not cut off by the left-sided lesion; while those from the right (unparalyzed) leg ascending on the left side of the cord are arrested at the point of the lesion. At the level of the lesion, all sensations coming in by the posterior nerve roots will be arrested, and this gives rise to a band of anæsthesia around the body; the band being, how-

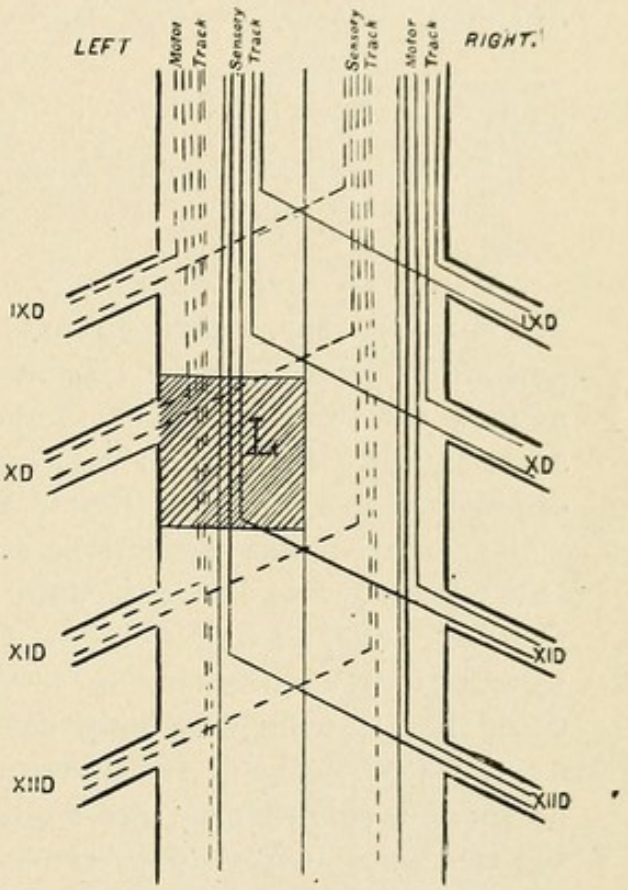

FIG. 51.—Diagram of Lesion causing Brown-Séquard Paralysis. *L*, Lesion in left half of cord cuts off motor impulses to left leg, sensory impulses from right leg, and sensory impulses from *XD* nerve. It irritates *IXD*, causing hyperæsthesia. Case XXVI.

ever, one segment higher on the side of the lesion than upon the other side, as shown in the diagram. The two following cases illustrate this condition. The rapid course and fatal termination of the second case make it remarkable. The negative result from an operation for the relief of pressure in the first case—the first case of this kind on which an operation has been performed—makes it interesting and shows the limits of spinal surgery.

CASE XXVI.—FRACTURE OF NINTH DORSAL VERTEBRA—BROWN-SÉQUARD PARALYSIS—OPERATION, WITH PARTIAL RECOVERY.

A teacher, twenty-four years old, had a fall upon his back, in February, 1887, which resulted in fracture of his dorsal spine and total paraplegia with loss of sphincter contol, which remained for some months and then gradually diminished to some extent. From the very first he noticed that power was diminished to a greater extent in the left leg than in the right; while sensation was affected more in the right leg than in the left. He had a bed-sore over the right buttock. When seen, October, 1888, he had a typical spastic paraplegia: the legs were rigid, the voluntary power much impaired, so that he could hardly walk with help, and the tendon reflexes much exaggerated. The motor symptoms were much more marked on the left side than on the right. The differences between the two sides in regard to sensation was very great. While on the right side he was quite insensitive to cold, heat, pain, and touch, on the left side he was extremely sensitive to these impressions, the slightest contact giving rise to pain. Any irritation of the surface on the left side left a broad red streak on the skin, an indication of vaso-motor paralysis, which was confirmed by the fact that the surface temperature was perceptibly lower on this side. The disturbances of sensation ceased at the mid-dorsal region, where a zone of partial anæsthesia existed around the body, and above this a zone of hyperæsthesia was found about two inches wide on both sides. The anæsthetic zone followed the line of the ninth dorsal

nerve on the left side and the tenth dorsal nerve on the right side (see Figs. 51 and 52). The skin reflexes were increased upon both sides, but more so on the right side. He could control his sphincters. Motions of the trunk were painful and limited; the pain being felt in the spine at the region from the sixth to the tenth dorsal vertebræ, which were very tender to pressure. A deflection of the ninth dorsal spine to the right was quite evident. There were no symptoms in the arms.

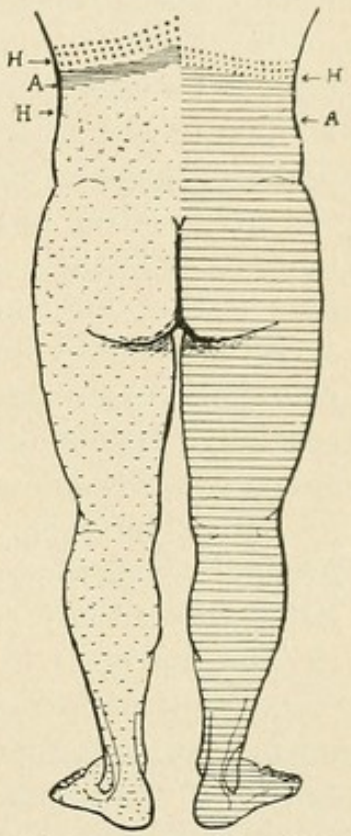

FIG. 52.—Areas of Anæsthesia and Hyperæsthesia in Case of Brown-Séquard Paralysis. Case XXVI. Lines indicate anæsthesia; dots indicate hyperæsthesia.

Diagnosis.—The questions of interest in diagnosis were, first, as to the exact nature of the lesion, and, secondly, as to the possibility of operative interference.

The nature of the lesion might have been either a mere compression of the cord by the displaced and fractured vertebræ, or a serious injury to the cord at the time of fracture with myelitis, or a hemorrhage into the cord, or a hemorrhage outside of the cord in the membranes.

A hemorrhage into the membranes usually infiltrates them and extends downward, filling the spinal canal if severe, and forming a local clot if slight. Such a clot usually extends around the cord, producing general pressure of slight degree. If severe enough at the outset to produce a total temporary paraplegia, it would hardly have been so strictly localized upon one side as to have caused the Brown-Séquard paralysis which remained. A unilateral clot within a membrane whose structure is not such as to limit the extent of a bloody effusion, seemed improbable. A hemorrhage in the cord itself is often produced by injury and is not infrequently unilateral. At its occurrence the initial pressure might have produced the temporary paraplegia, and after the contraction of the

clot the unilateral destruction of the cord would give rise to Brown-Séquard paralysis. The clot, however, must inevitably have caused an actual destruction of spinal tissue. In this case, however, there was no evidence of a destruction of the cord, to any extent, upon the side of the lesion; for the abdominal, epigastric, scapular, and dorsal reflexes were not lost and the zone of anæsthesia about the left side was not total, as is usual when a part of the cord, and consequently its sensory gray matter, is destroyed. The reaction of the abdominal and intercostal muscles to faradism was also perfect, which indicated an integrity of the anterior gray matter at the dorsal segments opposite the site of injury. If the hemorrhage were not in the gray matter it might certainly be within the white columns; but in that case the motor symptoms should predominate if the clot were in the lateral column, or the sensory symptoms should predominate if the clot were in the posterior column. As a matter of fact, neither of these symptoms was specially predominant; hence it was concluded that an intraspinal hemorrhage was impossible.

An extensive myelitis subsequent to injury was also improbable, since the permanent symptoms indicated a unilateral lesion without any great destruction of gray matter.

The diagnosis by exclusion was therefore a compression of one-half of the spinal cord by displaced bone, a diagnosis warranted by the existence of deformity and tenderness with restricted movement of the vertebræ. The possibility of some movement in all directions and the character of the deformity, a slight displacement of the spinous process to one side, made it improbable that any dislocation of the vertebræ had occurred. Pressure of the bone on the cord would surely suspend its functions at the point of pressure and might give rise to degenerations upward and downward. The pressure suddenly produced would at first cause a general bruising of the entire segment affected, with total paraplegia, and later these symptoms would give place to those of local pressure only, viz., Brown-Séquard paralysis. The pressure, though sufficient to suspend the transmission of impulses through the white matter, might not be sufficient to destroy the gray matter or

to arrest its reflex action. And the fact that some transmission of impulses was still possible through the compressed part, was shown by the performance of slight voluntary motions in the left leg and the perception of strong irritations in the right leg. Hence it was thought that no part of the cord was absolutely destroyed. Thus the symptoms corresponded with considerable accuracy to the hypothesis of unilateral pressure with secondary degenerations, and negatived other hypotheses.

The question of operation next presented itself. The time which had elapsed since the injury was eighteen months, and the symptoms indicated a probability of a descending degeneration below the level of the lesion. It is still a matter of uncertainty whether regeneration occurs in the spinal cord. Experimentally after section in animals it does not. But many cases of paraplegia from Pott's disease, in which rigidity and increased reflexes are present, recover to a considerable degree when the spine is straightened and the pressure on the cord removed. It seemed, therefore, that an operation in this case would offer the only possible relief and might be followed by improvement. It was accordingly undertaken by Dr. McBurney.

The details of the operation will be published by him elsewhere. When the spine was exposed and the spinous process of the ninth dorsal vertebra removed, it was found that a bony deposit, probably callous, on the inner surface of the left lamina of the vertebra had made lateral pressure upon the left side of the cord, which appeared to be pressed over toward the right side in the spinal canal. This lamina was, therefore, removed. A probe passed upward and downward outside of the dura failed to encounter any obstacle. The dura did not pulsate. Its resistance at all points seemed equal. It was not opened.

The patient made a satisfactory recovery from the operation, though for a few days after it all his spinal symptoms were increased, the bladder being irritable and much pain being felt in the umbilical region and around the body. After the eighth week the improvement in his spinal symptoms

began to appear. Six months after the operation an examination showed that the actual voluntary power in the left leg was greater than prior to the operation, but it was still rigid, spastic, and its reflexes much exaggerated. The vaso-motor paralysis had ceased, the temperature of the leg was the same as on the other side, and irritation did not leave as marked a line. All hyperæsthesia and anæsthesia of this side of the body had ceased. He could walk with aid and balanced himself better than before the operation. There was, therefore, a slight improvement in the motor symptoms, and a relief of the vaso-motor and sensory symptoms on the side of the lesion. In the right side there was also a change. He perceived sensation of touch, temperature, and pain more acutely and more quickly than before the operation, but was still unable to locate them correctly, occasionally referring touch to his groin when it was made below the knee, a symptom which had been present from the beginning. The result of the operation was, therefore, some relief of the symptoms, but not a great degree of improvement. The pressure of the bone had probably caused degenerations in the cord, which were not affected by the removal of their cause.

The question of the possibility of regeneration has been decided negatively; for at the end of eighteen months after the operation the condition remains about the same. He walks better than before the operation, but the spastic rigidity of the left leg remains, and it is slightly cooler to the touch than the right one. The sensation in the right leg is better than before the operation. He can feel all sensations on the outside of the leg normally, but on the inside of the leg and upon the foot warmth is not felt at all, and touch and pain are not accurately located. There is no girdle sensation and no area of absolute anæsthesia anywhere.

This case therefore appears to corroborate the statement made by Thornburn [1] that "a certain amount of improvement may follow a crushing lesion, due probably to the recovery of those portions of the cord structure that have only been compressed and not destroyed."

[1] "Surgery of the Spinal Cord," p. 159.

Thornburn's conclusion, from a study of sixty-one cases, is that "the operation of trephining the spine for traumatic lesions as compared with the condition which it is intended to relieve does not present any very great dangers, and appears unlikely to increase the gravity of the prognosis, but as both *à priori* argument and the results of published cases show that it is unlikely to be of service, it should be abandoned, except in cases of injury to the cauda equina, and in the latter, on the other hand, it will probably prove to be an eminently practicable and serviceable procedure."

This case indicates that the removal of pressure from a degenerated cord is not followed by complete recovery. This patient, however, is so much improved that he declares that he is very glad that the operation was performed.

With these facts in mind, I have refused to recommend an operation in three cases, one of Brown-Séquard paralysis and two of traumatic transverse myelitis, which were seen at the clinic within a year, although in two of these manifest deformity was present. I do not think, however, that when there is evident pressure upon the cord without absolute signs of a destruction of tissue in it, it is right to refuse the patient the benefit of the operation.

The record of cases successfully treated by Macewen,[1] Horsley,[2] Abbe,[3] and others where pressure has been removed and a considerable degree of recovery has ensued, seems to warrant this conclusion.

CASE XXVII.—CASE OF CERVICAL PARAPLEGIA.—AUTOPSY.

The following case, reported by Dr. Christian A. Herter[4] and kindly placed at my disposal by him, is here inserted as a typical illustration of a transverse lesion of the lower cervical segments:

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[1] Macewen: Brit. Med. Jour., 1888, vol. ii., p. 308.

[2] Horsley: Med. Chir. Trans., 1888, vol. lxxi., p. 402.

[3] Abbe: N. Y. Med. Jour., Nov. 24th, 1888; and N. Y. Med. Rec., Feb. 9th, 1889.

[4] Jour. Nerv. and Ment. Dis., January, 1890.

The patient fell and broke his back on October 9th, 1889. He was taken to the Presbyterian Hospital, in this city, and there found to have a tender spot without deformity over the sixth and seventh cervical vertebræ. His respiration was purely diaphragmatic. There was complete paralysis of motion and of sensation below the arm-pits. In the upper extremities there was complete loss of power in the intrinsic muscles of the hands and interossei, in the flexors and extensors of the wrist, and in the triceps. There was slight loss of power in the pronators and supinators and in the biceps and pectorals. The deltoids, trapezii, and muscles of the neck and head were normal. All reflexes except the left plantar were lost, and there was retention of urine. The area of anæsthesia is shown in the diagram (Fig. 45, page 137) and involved the corresponding regions upon the back of the arms as well as upon the front. The dotted area around the neck was hyperæsthetic, and here a girdle sensation was felt. The posture assumed was that described by Thorburn in his cases: arms abducted, forearms flexed and supinated. The patient died in forty-eight hours. The autopsy showed the cord to be crushed, by a dislocation of the sixth cervical vertebra, at the eighth and seventh cervical segments.

CASE XXVIII.—CARIES OF CERVICAL VERTEBRÆ—BROWN-SÉQUARD PARALYSIS—DEATH.

A girl of fifteen, previously in good health, noticed in August, 1887, that her neck was becoming stiff and painful on motion. The pain was in the course of the great occipital nerves, and by October her head had become so rigid that no active or passive motion was possible. During October weakness in the arms, especially in the left arm, began, and soon the left leg also became weak. She was sent to me on the 22d of October by Dr. G. A. Kretchmar, and then presented the following symptoms. The *head* was held rigid, turned slightly to the right, the chin slightly elevated, and passive motion was prevented by the rigidity of the deep muscles of the neck as well as by a deformity of the spine, which was

evident both by a deep hollow in the neck at the fourth and fifth cervical vertebræ and by a projection at the back of the pharynx. Pain in the neck and over the back of the head was a prominent symptom, but was not associated with anæsthesia (Fig. 46, page 137). The *left upper extremity* was partially paralyzed, all movements being very much impaired; those at the shoulder and elbow joints more than those at the wrist and fingers. No motion of the arm was possible, it hanging helpless. Motions of the forearm were very weak, extension being impossible; the forearm was semi-flexed upon the arm. While all the fingers could be moved, she had no grip. The triceps and wrist tendon reflexes were greatly exaggerated, a triceps clonus being obtained. All sensations were perceived equally in the left side and in the face; and the muscular sense was keen and quick.

The *left lower extremity* was weak, both at hip, knee, and ankle joints, and the tendon reflexes were much exaggerated. The sensation was normal—that is, the same as in the face.

The *right upper extremity* was freely moved, but was markedly anæsthetic up to the shoulder. Tactile sensations, sensations of pain, heat, and cold were not perceived by any means as promptly, as accurately, or as intensely as in the corresponding part of the opposite limb. There was marked ataxia of movement in the right hand when her eyes were closed; and the perception of its position was less rapid and accurate than on the left side. There was no motor paralysis and no increase of tendon reflexes.

The *right lower extremity* was not paralyzed, the knee-jerk was normal, no ankle-clonus was obtained. The same kinds of anæsthesia were found as on the right arm, though sensations were rather better in the lower than in the upper extremity. The area of anæsthesia is shown in Fig. 46, page 137.

The *gait* was much impaired. She staggered in standing and walking, with a tendency to fall toward the right when her eyes were closed, and her gait was decidedly ataxic.

The *control* of the sphincters was somewhat impaired. She had to strain a long time before her water would pass, and a

loss of voluntary control of the bladder soon ensued, so that a catheter had to be used.

There was no hyperæsthesia of the paralyzed side and no band of anæsthesia about the trunk on that side.

By the end of November, 1887, all of the symptoms had increased in intensity, the paralysis of motion of the left side being complete and the anæsthesia on the right side being increased; she was unable to sit up or to control her sphincters. She had distressing attacks of dyspnœa.

The condition finally became one of total paraplegia, and in January, 1888, she died.

Diagnosis.—It was evident from the first that a caries of the fourth and fifth cervical vertebræ had resulted in a displacement of these bones, which had produced pressure upon the left half of the spinal cord at the level of the fifth and sixth cervical segments That these segments or their nerves were seriously implicated was evident from the fact that a marked atrophy, as well as paralysis and a reaction of degeneration, was found to exist in the deltoid, biceps, triceps, supinator longus, supra and infra spinatus, and pectoral muscles of the left arm, while no such atrophy or reaction was found in the other muscles in spite of their paralysis. In the right arm the anæsthesia was more marked over the back of the shoulder and arm and on the outer side of the arm and forearm than elsewhere. The pressure was sufficient to cut off to some extent motor impulses downward to the left leg on the side diseased, and sensory impressions of all kinds from the right side of the body below the level of the lesion.

Operative interference appeared to be impossible, and the disease made rapid progress, producing finally total paraplegia and death from respiratory paralysis. An autopsy was not made.

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The cases brought together in this chapter demonstrate the differences in the symptoms produced by lesions at various levels of the spinal cord. They also show that a study of local symptoms in spinal lesions results in exactness of diagnosis and may lead the way to successful surgical treatment.

CHAPTER XII.

LOCOMOTOR ATAXIA.

WITH SOME REMARKS ON THE TREATMENT BY SUSPENSION.

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LOCOMOTOR ataxia, or posterior sclerosis, or tabes dorsalis, as it has been variously named, is a chronic disease of certain limited regions of the spinal cord characterized anatomically by a typical sclerotic degeneration of the posterior external columns and adjacent parts, and clinically by motor disturbances, chiefly incoördination, and by remarkably diverse derangements of the various forms of sensibility.

The *principal symptoms* of the disease found in the patients applying for treatment at the clinic were as follows:

(1) A feeling of weakness, often strangely in contrast with the actual power of the muscles when the latter was measured.

(2) A hesitation or feeling of insecurity and unsteadiness when standing or walking in the dark, or with closed eyes.

(3) A sensation as of walking on springs, on a cushion, or on velvet, confined to the soles of the feet and due to anæsthesia of those parts.

(4) Fulgurating or lightning pains, often described as stabbing, darting, or tearing pains, which were said to shoot through the limbs, generally the lower extremities, causing excruciating suffering during a few seconds or minutes, then disappearing, only to return after a short time.

(5) A feeling of constriction, generally about the trunk, called the girdle sensation.

(6) The so-called crises which occur suddenly in attacks and are infrequent. They may be manifested as spasmodic

pains in the viscera, or as exaggeration of the function of a viscus. Crises may take the form of severe griping pains in the region of the stomach, accompanied by flatulence, generally by vomiting, rarely by nausea (gastric crises); they may resemble attacks of renal colic (nephralgic crises), or consist of pain in the bladder, urethra, or rectum (vesical, urethral, rectal crises); or paroxysms of coughing or of dyspnœa (bronchial and laryngeal crises). Crises connected with the sexual organs have not been observed.

(7) Various disturbances of cutaneous and muscular sensibility of all degrees of intensity and of very various forms. These merit a partial enumeration. They consist of insensibility of the skin to simple touch or contact (*anæsthesia*); abnormally increased sensibility to touch (*hyperæsthesia*); sensations referred to the skin and not corresponding to the causes producing them, as formication, a velvety or furry feeling, crawling, tingling, etc. (*paræsthesia*;) an exaggerated and abnormal sensibility to cold felt all over the whole body, obliging patients to dress unusually warmly and to live in superheated rooms for comfort (*cryæsthesia*[1]); insensibility to pain from pricking, pinching, etc. (*analgesia*); abnormally increased sensibility to pain—*e.g.*, great pain felt from a slight prick or even from the touch of a soft object (*hyperalgesia*); two new symptoms which will be explained further on (*hyperthermalgesia* and *hypercryalgesia*); loss of the sense of the position of the members (disturbance of muscular sense); and retardation of pain—*e.g.*, the pain of the prick of a needle may not be felt until several seconds after the contact of the needle has been received (Remak's sign).

The above-named symptoms, however, most of which are dependent upon an alteration of the various forms of sensibility, while very numerous, varied, and exceedingly interesting from a clinical and even from a pathological point of view, are subjective, are not uniformly present, and are not essential to the diagnosis of the disease.

The diagnosis is based upon *three objective signs*, the pres-

[1] Cryæsthesia, from κρίος, icy cold. Dieulafoy, Gaz. hebdomadaire de Méd. et de Chir., June 25th, 1886, p. 426.

ence of which together may be considered quite pathognomonic of locomotor ataxia, viz.: Westphal's symptom—that is, loss of patellar reflex; Romberg's symptom—that is, swaying widely and falling on standing with eyes shut; and Argyll-Robertson's symptom—that is, a loss of the pupil reflex to light, the reaction in accommodation being present. With these data, a diagnosis of tabes dorsalis may unhesitatingly be made, even in the absence of any or all of the others. On the other hand, the absence of any one of these three signs throws a doubt upon the accuracy of the diagnosis unless the latter be confirmed by a number of other symptoms; while the absence of Westphal's symptom alone is a quasi-proof that the disease is not pure tabes.[1]

In addition to these diagnostic signs, the following *objective phenomena* are found in the fully developed disease:

(1) Incoördination or ataxia in standing (*static ataxia*), which is more marked when the eyes are closed and the base of support is diminished in area, as when the feet are placed as closely together as possible.

(2) Ataxia in movement of any of the extremities; as in walking (*locomotor ataxia*, ataxic gait), when the patient may not be able to control the movements of the feet properly, or to put them down naturally even after repeated efforts, so that the gait is uncertain and staggering, the feet being placed wide apart and often being lifted too high and coming down to the floor with a noise; or ataxia in fine movements, such as seizing an object, placing the fingers on the ear and nose with the eyes closed, buttoning the clothing, writing, etc., in which the irregular jerky, swaying motions, quite unintentional, are very characteristic.

(3) Temporary or permanent paralysis of one or more of the muscles of the eyeball; paralytic strabismus or ptosis.

(4) Atrophy of the optic nerve, causing diminution of the visual field to colors and then to light, followed by blindness, which may become total.

(5) Disturbance of the action of the bladder, either incon-

[1] In a few cases when the disease begins in the cervical region the knee-jerk persists for some months after the onset.—M. A. S.

tinence or difficulty in beginning the act of urination; constipation; temporary increase followed by loss of sexual power.

(6) Trophic disturbances, such as painless swelling and dis-

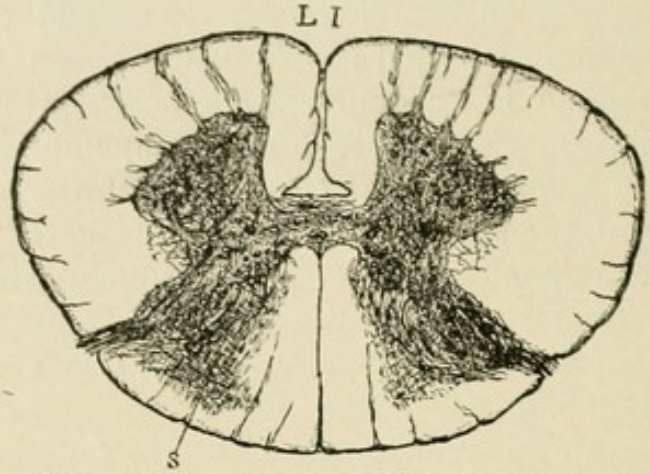

FIG. 53.—Area of Sclerosis in the First Stage of Locomotor Ataxia. Patient died one year after the onset of the first symptoms. The knee-jerk was lost on the left side where the sclerosis (*S*) was found to be much more intense than on the right side. The sclerosis extended as high as the IX. dorsal segment. No secondary sclerosis was found.—M. A. S.

integration of joints (Charcot's joint, ataxic or *tabetic arthropathy*), deformities of the feet, perforating ulcers, loosening and falling out of the hair, teeth, and nails.

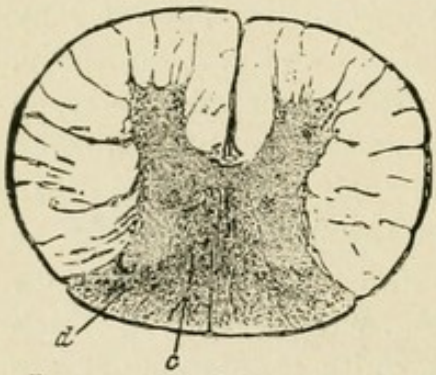

FIG. 54.—Area of Sclerosis in Last Stage of Locomotor Ataxia (Gowers). Both of the posterior columns are completely sclerotic, the lesion being more intense in the col. of Burdach (*d*) than in the col. of Goll (*c*).

Without entering more fully into the general description of this disease, it may here be mentioned that the seat of the lesion characteristic of tabes dorsalis is not exclusively in the posterior columns of the spinal cord, as was once believed, but has been shown to invade other regions of the cord, either primarily or secondarily. Thus the column of Goll, the direct cerebellar tract of Flechsig, and the antero-lateral tract of Gowers are usually affected secondarily; and the analgesia tract of Spitzka and the border-zone (Randzone) of the posterior horn (the tract of Lissauer)[1] are sometimes primarily affected by sclerosis.

[1] "Beiträge zum Faserverlauf im Hinterhorn des menschlichen Rückenmarks." Arch. für Psychiatrie, 1886, p. 377.

Symptoms closely resembling those of the disease in question, if not identical with them, have been described in cases in which the posterior columns were healthy and only peripheral neuritis was found at the necropsy.[1]

From the date of the establishment of the Vanderbilt Clinic in February, 1888, to January 1st, 1890, there have been 24 cases of tabes dorsalis treated at the institution. Of this number 2 were women, a proof of the greater frequency of the disease in males. Of this number 6 denied syphilis and presented no evidences of that disease.

An endeavor has been made to ascertain the first symptom manifested by the patient in these cases. In 17 this was determined. It was pain in 6 cases; a feeling of general weakness and fatigue in 2 cases; ataxia in 4 cases, chiefly locomotor; paræsthesia in 2 cases; numbness in both hands in 1 case; enuresis in 1 case; and swelling of the great toe in 1 case.

In the 24 cases, on examination the following symptoms were found: Incoördination (other than static) was recorded in 13 and was absent in 7 cases. An ataxic gait was observed in over one-half of the patients. Romberg's sign was present

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[1] Déjerine: "Altérations des Nerfs cutanés chez les Ataxiques," Arch. de Physiol., 1883, vol. ii., p. 72. The first case was reported at a meeting of the Société de Biologie, Feb. 18th, 1882. Déjerine et Sollier: "Nouvelles recherches sur le tabès périphériques (Ataxie locomotrice par névrites périphériques)," Arch. de Méd. expérimentale, 1889, vol. i., p. 251.

The differential diagnosis between locomotor ataxia and peripheral neuritis is thus described by Prof. Starr in his lectures on multiple neuritis: "Ataxia, loss of knee-jerk, pain, and sensory disturbances, including loss of muscular sense, Romberg's symptom, and optic neuritis, are common to the two diseases. In multiple neuritis the relatively rapid onset of the ataxia, which follows closely upon the sensory symptoms; the prominence of numbness and anæsthesia rather than of lightning pains; the extreme degree of anæsthesia and analgesia; the tenderness of the muscles and nerves; the usual occurrence of some degree of actual paresis with atrophy and the reaction of degeneration; and absence of bladder and sexual symptoms will point inevitably to the diagnosis. Furthermore, the ataxic form of neuritis only occurs after poisoning with alcohol or arsenic, or as a sequel of diphtheria, and the establishment of the causation will aid the diagnosis. Here, too, the course of the case toward recovery with return of the knee-jerk will decide in favor of neuritis, if the diagnosis has not been reached in the early stage."—"The Middleton Goldsmith Lectures for 1887," Med. Record, Feb. 5th, 1887.

in 22 cases, including both women, or ninety-two per cent. In 4 cases there was or had been some paralysis of the intra-orbital muscles. No monoplegias, hemiplegias, or paraplegias were noted.

Symptoms involving reflex activity were, of course, found in every case. The knee-jerk was invariably absent. The Argyll-Robertson pupil was noted seventeen times. In 2 cases there was complete immobility of the pupil both to light and to accommodation. In 2 cases the reaction to light was slow and slight.

The sexual power was recorded as good in 6 cases, as lost in 3, as diminished or incomplete in 2.

Sensory symptoms, those apparently spontaneous as well as those elicited by methodical examination, were found in their usual variety and abundance. Fulgurating pains were complained of in 19 cases (seventy-nine per cent), eighteen times in the lower extremities, three times in the upper, and once in both lower and upper extremities. Girdle sensation was present in 9 cases. In one of these the "girdle" had moved upward, during the progress of the disease, from the abdomen to the level of the nipple. Paræsthesiæ were complained of by the majority of the patients. Pricking, tingling, or numbness in the hands and feet, especially the latter, with a sensation in the soles of the feet as if the patient were walking on a cushion or velvet, was present in 13 cases, and *cryæsthesia* was quite marked in eight per cent of the cases. One of these patients felt continually cold, could not dress warmly enough for comfort, and kept his room at the temperature of 32° C. (90° F.) and still felt chilly all the time. Another had felt continual chilliness during five years. This symptom, as well as hyperthermalgesia and hypercryalgesia, soon to be described, has not been mentioned, at least to my knowledge, as one of the morbid sensory phenomena due to tabes dorsalis. It has been noted especially in chronic parenchymatous nephritis and also in chronic alcoholism, and it is interesting to note in this connection that in both of these diseases peripheral neuritis is not infrequently present. It is possible, therefore, that in all three of these affections this symptom is due to

chronic inflammation or degeneration of the cutaneous nerves. Complete anæsthesia of the feet was found in 1 case. Analgesia was observed in 4 cases, in 3 of which the insensibility to pricking and pinching was limited to the lower extremities. In 1 case it extended as high as the umbilicus.

Alterations in the muscular sense were not often noted. In one patient this sense was lost in the left leg and diminished in the right arm.

Remak's sign was present in 3 cases (twelve per cent), two of the patients having had this disease four years, the other, a woman, two years. The retardation of sensory conduction was noted as being of ten seconds' duration in one case and twenty seconds in another, which is not far from that of fifteen seconds, as in one of Dr. Remak's patients.

Hyperthermalgesia and *hypercryalgesia*, which are here put forward as new symptoms of tabes dorsalis, are two painful subjective phenomena due to hyperæsthesia of the sense of temperature. They represent sensations that are quite *out of proportion* to the degree of heat or of cold that is applied to the surface of the skin. Hyperthermalgesia (θερμη heat) means hyperalgesia produced by the application of heat to the cutaneous surface, hypercryalgesia (κρύος, great cold) signifies hyperalgesia to cold applied in the same manner. The former is present when the patient feels pain and starts as though hurt upon brief contact with a body having a temperature of + 50° C. (about 120° F.) or a few degrees less, a temperature which to a normal nervous system is quite supportable; the latter when the patient is similarly affected by contact with a temperature of + 10° C. (50° F.) or a few degrees higher.[1] The tests are made by heating or cooling water in a test-tube and holding the tube against the skin. Hyperthermalgesia was

[1] The terms *thermalgesia* and *cryalgesia* would be properly used to express normal sensations, the first meaning a painful sensation of heat, the second of cold, which can be elicited from any normal organism by the application of a certain temperature which would correspond to the sensations felt. It is only when the sensation is not proportional to the exciting cause, thus becoming a hypersensation —if we may be allowed the expression—that it becomes pathological, as in the cases here reported.

present in four cases, hypercryalgesia in three. All the latter cases were also hyperthermalgesic, and these two symptoms were manifest in the same regions of the body in the three cases (abdomen, thighs, and legs). In one case they were very marked, the patient starting and uttering an exclamation of pain whenever the warm or cool body (a test-tube filled with water) was applied to the bare skin of the above-mentioned regions. The following is a brief history of this case:

CASE XXIX.—J. E., hair-dresser, French, thirty-seven years of age, of medium height, strongly built and muscular, had a hard chancre at the age of nineteen, which was treated in France by mercury in large doses during a long period of time. Six years ago he began to have fulgurating pains of great intensity in the calves of the legs and in different regions of thighs both in front and behind. These pains were so severe that they forced him to cry out. They came and went suddenly and were not of the nature of muscular cramps. He has followed treatment for these pains during several years without relief.

At present, the knee-jerks are lost; he has moderate myosis with Argyll-Robertson pupils; no ataxia, either static or locomotor, but constipation and various disturbances of sensibility. The latter consist of analgesia of both lower extremities and an abnormally painful sensibility to moderate heat or cold when applied to the surface of the legs. The hyperthermalgesia and hypercryalgesia are observed in the regions of analgesia (absence of pain from the prick of a pin, from pinching, etc.), and only in these regions. There is no false perception of the *quality* of the temperature, as heat is aways recognized as heat and cold as cold, but there is false perception of its *quantity*. Remak's sign is absent.

These observations bring additional evidence of the independence of the different forms of cutaneous sensibility, and show that one form (as tactile sensibility) may remain normal, while another (the thermic in the last case) may be impaired. They also demonstrate that the rapidity of conduction of sensation may still remain intact when the sensation itself is impaired. The two subjective phenomena of hyperthermalgesia

and hypercryalgesia have not, so far as the writer has been able to discover, been mentioned before in the symptomatology of spinal diseases. In the remarkable article of Dr. Remak,[1] of Berlin, upon the disturbances of sensibility in tabes dorsalis there is no reference to them, although certain electric phenomena and the extraordinary retardation of painful sensations are there described. In one of our patients hyperalgesia to temperature was felt in the analgesic regions; in another there was no disturbance of cutaneous sensibility except hyperalgesia to temperature, and the sensitiveness to cold (which was the greater) was so painful that he begged that the tube be removed at once, although the temperature of the water in it was but + 15° C. (58° F.).

In regard to ocular symptoms, there was diplopia in 2 cases, polyopia in 1, optic atrophy in 3, and unilateral mydriasis due to paralysis of the ciliary muscle in 1 case (a woman). In 4 cases the color-field was diminished and in another case there was right nasal hemiopia.

Enuresis was present in 11 cases (forty-six per cent).

Tabes was, in one instance, associated with general paresis of the insane, which manifested itself by the patient's general optimism, by marked tremor of lips and tongue, and by hesitation in speech. His memory was preserved to a certain extent, for he remembered (or at least gave immediately, as if he remembered) various dates, as that of his birth, of his marriage, of his children's birth, etc. He could also calculate fairly, although he was said to have been losing this faculty of late.

In 2 cases the disease has lasted thirteen years, which is the maximum duration of the disease observed thus far in our patients; in 3 it was only of two years' duration. The two patients who have had it the longest do not show so many troublesome symptoms as many who have acquired the disease more recently. Sexual power is still good in one of them.

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[1] "Sensibilitätsstörungen der Tabes dorsalis," Arch. f. Psychiatrie, 1877, vii., 496. In one patient he found hyperalgesia (to touch) on the right thigh with anæsthesia of opposite thigh to all kinds of excitation—*i. e.*, analgesia, thermo-analgesia, electro-analgesia.

The treatment of these 24 cases was varied and was generally constitutional as well as symptomatic. Mercurial inunctions followed by potassium iodide were prescribed in most of the cases which presented a syphilitic history, but rarely with any marked effect. One patient before coming to the clinic had been taking nitrate of silver and reported unfavorable effects from it. It was not continued. Fowler's solution was ordered in a number of cases, and is in Dr. Starr's opinion of as much service as any medicinal remedy. Tonic remedies, as iron and strychnia, were administered to several patients as needed. As an anodyne to the fulgurating pains, antipyrine and antifebrine were commonly employed and usually with success. Occasionally the faradic brush relieved an attack of pain. In one case the pains were relieved by a teaspoonful of the following mixture, taken every four hours:

℞	Potass. iodidi, ...	2.0
	Tinct. opii, ...	2.0
	Ext. gelsemii fl., ...	2.5
	Ext. cimicifug. fl., ...	5.0
	Syr. sarsap. co.,	
	Aq., ...	āā. ad 60.0

Treatment by Suspension.—As soon as this method of treatment was put forward by Charcot, it was applied to nearly all of the patients systematically, the number then treated being ten. The disuse into which it has fallen after three months' trial is proof of its inability to relieve the symptoms. It is now rarely employed. Notwithstanding its discomfort to the patient and its occasional dangers, to one of which I had occasion last year to call attention,[1] and notwithstanding the disappointment in the method after so much benefit had been hoped for, there was still a certain small amount of good done by it in a few cases. One patient, tabetic during nine years, averred that suspension "took all his pains out." Another, a woman, showed much improvement in walking after submitting to suspension frequently during several weeks. A third

[1] "A Danger to be Avoided in the Treatment of Nervous Affections by Suspension," N. Y. Med. Jour., June 29th, 1889, p. 715.

obtained an apparatus and suspended himself daily for six months, thinking that it helped him in walking: he has recently given it up. The two symptoms, pain and ataxic gait, are the only ones we have seen ameliorated by this mode of treatment. The conclusions derived from our experience harmonize with those on the subject expressed at a recent discussion in the Neurological Society of London,[1] that suspension fails to benefit patients in the large majority of cases. On the other hand, one patient thought that suspension did him harm, and the majority to whom it was applied found that they derived no permanent benefit from it. The method has been faithfully tried by Prof. Starr upon six private patients without any favorable result.

One patient, a healthy-looking married man of thirty years of age, who has had tabes for the past four years, gave up all medicinal treatment about one year ago, and has sought gay society and amusements and now reports himself improved. There can be no doubt of the exactness of the diagnosis in his case for he shows Romberg's, Westphal's, Argyll-Robertson's, and Remak's signs. He suffers from fulgurating pains, cryæsthesia, and hyperthermalgesia.

As regards cures, there can, of course, be none reported. Four cases have improved, three appear stationary; in the remainder the disease is progressing. One patient has died.

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[1] Reported in La Sémaine Medicale, Feb. 26th, 1890.

CHAPTER XIII.

THE PARALYSES OF INFANCY.

I. Traumatic paralysis of the brachial plexus, Erb's paralysis.—II. Infantile spinal paralysis, atrophic paralysis (anterior poliomyelitis).—III. Infantile cerebral paralysis.

THERE are three forms of paralysis commonly seen in infants; and since each of these is dependent upon a different lesion and each has a very different prognosis and requires a different manner of treatment, they are grouped together in order that the contrast may be evident. These various forms are (1) *Erb's paralysis* of the arm, or *obstetrical paralysis*,[1] due to an injury during delivery of the fifth and sixth cervical nerves at their junction in the brachial plexus; (2) *Infantile spinal paralysis*, due to an acute inflammation of the groups of cells in the anterior gray horns of the spinal cord; and (3) *Infantile cerebral paralysis*, due to hemorrhage or embolism with subsequent sclerosis and atrophy, or possibly to an acute inflammatory process in the motor area of the brain cortex.

(1) ERB'S PARALYSIS—OBSTETRICAL PARALYSIS—BIRTH-PALSY.

This form of paralysis is noticed within a few hours or days of the birth of the infant. It is usually limited to the deltoid, biceps, brachialis-anticus, infra-spinatus, and supinator longus muscles, but occasionally involves the extensor muscles of the hand. The arm hangs loosely at the side of the body, its position as a whole being governed by gravita-

[1] Described by Duchenne as "paralysie obstétricale infantile du membre supérieur," and by Erb as "Entbindungslähmung." Ziemssen's Cyclo., Amer. Ed., vol. xi., p. 562.

tion only, with the forearm extended and pronated and the wrist and fingers flexed. If the arm be lifted and then let go it falls at once into this position. The muscles which are paralyzed are relaxed and their opponents are not rigid, so that the joints are all freely movable and motion does not give the child pain. In response to surface irritation the unaffected arm is seen to move freely, while in the paralyzed arm the only movements made are those of extension of the forearm upon the arm, of flexion and extension of the wrist and of the fingers. Sometimes when the extensors of the wrist are involved, there is slight extension of the two distal phalanges of the fingers only. A few days after birth, if the paralyzed muscles are examined electrically, it will be found that they have lost their contractility to the faradic current, and that on applying the galvanic interrupted current contractions occur more forcibly under the positive than under the negative pole (the reaction of degeneration). In these tests the unaffected arm should be taken as a standard for comparison with the other. If sensibility is tested by a needle, it will usually be found to be very much impaired over the area of the arm corresponding to the distribution of the cutaneous branches of the circumflex nerve, an oval area whose centre is the insertion of the deltoid muscle. It is best to test the sensibility of an infant by pricking the surface of this area on the unaffected side and noticing how promptly reflex acts of movement and of crying are produced by slight irritation. Then, when the babe has become quiet, the affected side may be tested with the same amount of irritation, when the reflex acts will not be produced so promptly or in some cases at all.

After the condition has remained for several weeks or months without much change, as it often does, the muscles which are paralyzed become considerably atrophied, and feel unduly soft and yielding. The actual size of the limb, however, may not be very perceptibly reduced, for the deposit of fat in a fairly healthy baby is much greater than the substance of muscular tissue, and therefore there is not the rapid wasting of the arm and forearm which is noticed in adults suffering from paralysis of the brachial plexus. When the

condition has lasted for three or four months there sometimes appears a slight stiffness of the unparalyzed muscles, so that bending the forearm at the elbow or opening the little closed hand is not as easy as at the beginning. A permanent rigidity, however, rarely if ever develops.

Some of these cases go on to spontaneous recovery. Thus in two cases seen at the Polyclinic, this occurred within six months. Many of them may be benefited by treatment, and recover within a year. This has been the case in three patients under my observation. Others, however, do not recover rapidly: the condition remains for three or four years, and only recovers when the child is old enough to be taught systematic gymnastic exercises. Several such cases have been observed, and I have seen a girl of fourteen who still had some disability of movement at the shoulder-joint due to the occurrence of birth-palsy. Erb gives a rather unfavorable prognosis in such cases.

The varying course of the disease, from complete recovery within four months to partial recovery after several years, depends upon the severity of the lesion and upon the line of treatment pursued—as in all cases of traumatic neuritis.

The lesion in cases of birth-palsy is a traumatic neuritis of the upper two nerves of the brachial plexus. Erb has shown that it is possible by careful faradic examination to find a spot which corresponds to the point of emergence of the sixth cervical nerve between the scaleni at which the irritation of the faradic current will produce a contraction in the deltoid, biceps, brachialis anticus, and supinator longus muscles. Stronger irritation will also cause the extensors of the wrist to contract. It is here, therefore, that the brachial nerves pass. Any pressure at this spot will easily injure them on their way to the muscles named. Such pressure is often made during delivery. This is particularly the case when the child presents by the breech and when it is necessary to extract the head. The common method of delivery in such cases is to put two fingers of the right hand upon the maxillary bones of the child's face and to place the index and middle fingers of the left hand, opened like a fork, upon the shoulders of the

child and thus make traction. In the so-called Prague method the same position of the left hand is employed. The left hand is at the same time used as a fulcrum around which the head is rotated, so that pressure over the shoulders is made forcibly and for some time. It is the pressure of the obstetrician's fingers which causes the injury of the brachial plexus in the majority of cases; and I have noticed that in over seventy-five per cent of the cases seen the paralysis was in the left arm, which finds its explanation in the greater length of the middle finger of the hand which is doing the damage. In the act of traction there is a tendency for the obstetrician to flex the fingers, and thus the tip of the finger is pressed deeply into the side of the child's neck. The cause being thus evident, this complicating affection may be avoided by a little care. In some cases injury of the brachial plexus is produced in other ways. Thus when it is necessary in a hand or arm presentation to replace the limb and bring down the head; or when it is necessary to bring down a misplaced arm; or when in version traction is made on the arm it is very easy to injure the brachial plexus. The application of forceps in an awkward manner to the head may also produce pressure on the neck and thus injure the plexus. Thus this form of paralysis is rightly termed obstetrical.

Children do not appear to be at all liable to injuries to the nerves, in spite of the many accidents which befall them. I have not seen a case of traumatic neuritis of the extremities in a child below the age of fourteen excepting of the form just described.

The treatment of Erb's paralysis is of much importance, as recovery can undoubtedly be hastened if proper means are employed. The arm should be kept as far as possible with the elbow flexed and not allowed to hang down, since its weight often suffices to over-stretch the ligaments of the shoulder, which are no longer assisted by the action of the deltoid. The arm should be rubbed daily, the mother being taught the kneading process of massage. Unintelligent people will be more likely to keep up the necessary rubbing if some simple lotion or ointment be ordered. The circulation

and nutrition of the limb will also be aided by sponging alternately with hot and cold water followed by brisk friction. And as soon as any voluntary motion can be made the child should be encouraged to make it, even if it be necessary to tie the unaffected limb to the side of the body. As the child grows older, systematic exercises of a gymnastic kind should be insisted upon daily.

The application of electricity probably aids the process of regeneration in the nerves and certainly helps to keep up the proper nutrition of the paralyzed muscles. For both purposes galvanism is required; for the faradic current has no catalytic action and fails to produce any contraction in the paralyzed muscles in these cases.[1] Galvanism is to be used in two ways: first, it is to be applied as a continuous current; secondly, it is to be applied as an interrupted current. In the first method a flat electrode is placed over the back of the neck or between the shoulders and the other electrode is stroked gently along the shoulder and down the arm, not being lifted from the skin. The current employed should not exceed six or eight milliampères,[2] and the duration of the application should not exceed five minutes daily or every second day. The poles may be used alternately. The object sought by this method is to promote the chemical processes which lie at the basis of all nutrition and regeneration in both muscles and nerves. The second method has another object. It is to strengthen the muscles by inciting them to action. A muscle which acts improves in nutrition, as the blacksmith's arm is usually cited to prove. When voluntary action is impossible, artificial action must be substituted. When an electric current is suddenly sent through or is interrupted in its passage through a muscle, a sudden change of electrical state is produced in the muscle, irritating it and making it contract. Such a contraction is called the closure contraction or the opening contraction, according as the current is suddenly sent

[1] See Chapter on Electricity as a Therapeutic Agent.

[2] Batteries differ so much in power and the skin of patients differs so much in resistance that no number of cells can be specified to be used. It is absolutely necessary to control the amount of galvanism given by means of a galvanometer.

through or is suddenly stopped while passing. The muscle does not contract during the passage of the continuous current. It only contracts when it receives a shock—that is, when the current is interrupted. To secure the desired end, therefore, in the second method, one of the poles used should have a key by means of which the finger by pressure can make or break the circuit, and during the application the circuit should constantly be closed and broken. A current should be used just strong enough to cause a contraction in the paralyzed muscles, and since in cases where the reaction of degeneration is present, as in birth-palsy, the muscle reacts more promptly to the positive than to the negative pole, it is the positive which should be placed upon the muscle.[1] The daily use of this artificial exercise is recommended, and after a little instruction the parents will easily learn how it should be done. The only dangers to be avoided are the application of too strong a current and too long an application. The mildest current which will cause contractions is to be used, and each muscle should not be exercised more than half a minute at a time, and not more than three times at one application. Let any one try to keep up rapid voluntary movement of any muscle—for example, the index finger—for more than a half a minute, and he will realize that further motion results in undue fatigue rather than in stimulation. Hence electric fatigue should be avoided in the application when electric exercise is the object.

When the child grows to an age when gymnastic exercises can be taught it, these should be systematically employed to exercise the muscles which are weak.

(2) INFANTILE SPINAL PARALYSIS—ANTERIOR POLIOMYELITIS.

Etiology.—This form of paralysis occurs with greatest frequency between the ages of one and four years. Thus of 31

[1] In order to be sure which pole is negative, dip the wires in water and let a current pass; notice from which wire bubbles begin to come off: that is the negative pole. The negative pole is also the most painful one when applied to the skin.

cases seen at the Polyclinic and Vanderbilt Clinic, in 20 the age of onset was between these limits. Four cases began before the age of one year; others as late as the age of thirteen. I believe there is no case on record of a child born with anterior poliomyelitis, though in some cases of spina bifida a total destruction of the lower lumbar cord has been observed, and subsequently paralysis with talipes has been present. But in such cases sensory symptoms as well as motor paralysis are to be found, and therefore they need not be mistaken for infantile paralysis. The age of onset will, therefore, prove of value in differentiating infantile spinal paralysis, on the one hand from Erb's paralysis, which is always congenital, and on the other from cerebral paralysis, which may occur at birth.

The onset of the disease in infantile paralysis is usually sudden, with febrile symptoms, and not infrequently with convulsions and delirium. The fever leads to vomiting, diarrhœa, and general prostration, and as the age of onset coincides with the age of teething, the parents usually suppose that the disease is due to this physiological process.

One case was brought to the clinic on the day after the onset and was found to have a temperature of 103°. Antipyrine was given, and also ergot; the spine was blistered over the lumbar region, and subsequently the child entirely recovered the use of its legs, though for six weeks it could not walk. Since acute congestion of the spinal cord is known to be present at the onset, too much importance cannot be paid to counter-irritation early. In 13 of these cases such a febrile onset was known to have occurred. In some of them it had no known cause; in others it followed the occurrence of measles, scarlet and typhoid fevers, and severe malarial poisoning.

One child tumbled into a pail of ice-water on the morning of the onset. Another noticed the paralysis soon after a violent exertion in lifting a heavy load, and it appeared in the right arm, on which the chief weight had rested. The effort of learning to walk has been supposed to be an occasional cause of paralysis in the legs, and though no cases can be cited to support this theory, the last case mentioned lends probability

to it. The cases at the clinic did not show any uniform relation between the season of the year and the onset.

Table Showing Age at Onset.

5 months	in	2	cases.
7 "	"	1	case.
8 "	"	1	case.
12 "	"	2	cases.
14 "	"	1	case.
16 "	"	1	case.
18 "	"	1	case.
20 "	"	2	cases.
22 "	"	1	case.

Between 2	and 3	years,	10 cases.
" 3	" 4	"	2 "
" 4	" 5	"	2 "
At age of 5		"	1 case.
" " 7		"	1 "
" " 11		"	1 "
" " 13		"	1 "

Males, 15. Females, 16.

The Character of the Paralysis.—The onset of infantile paralysis is not only sudden, but the distribution of the paralysis is extensive and its character severe; far more so than it is ever destined to remain permanently. Thus it is not uncommon for both legs to be totally paralyzed at the onset, but after a few months to find the paralysis limited to a few muscles of one leg. This occurred in four of the cases observed. Again, where the arms are paralyzed it is usual to find both arms affected seriously for several days, so that none of the joints can be voluntarily bent; and then finally the paralysis passes off, excepting in three or four muscles of one arm. This was the history in three cases. Or the extent of the paralysis is very alarming, all the extremities being involved and the little patient being utterly helpless. In one case both legs were at first affected, and six months later both arms were invaded. In such a case—of which I have seen two other examples—the final extent of the paralysis is usually in one arm and one leg.

In the terminal stage of the disease it is very uncommon to find any limb totally paralyzed or to find both arms or both legs equally affected, though at the onset both of these conditions may have been present. This feature in the course of the disease has given origin to the name "regressive paralysis," an unfortunate name, since there is rarely if ever a total disappearance of all symptoms.

The usual form of the paralysis is flaccid atrophic paralysis. Each muscle affected is seriously paralyzed; is soft and yielding, never rigid; it has lost all mechanical irritability and its tendon reflex is never preserved; it soon suffers in its nutrition, so that a rapid diminution in the size of the limb is noticeable within two weeks; and it presents a reaction of degeneration. The entire limb at the outset and for some weeks thereafter is relaxed, and its position depends entirely on the force of gravitation. It falls into any position which the posture of the child allows. In these respects the symptoms in individual muscles are identical with those of Erb's paralysis, but present a very marked contrast to the symptoms of cerebral palsy.

The extensive distribution of the initial paralysis has already been mentioned. After a month, when the effects of the severe spinal congestion have passed off, it is possible to ascertain just what muscles are to be affected for some time. The distribution of the permanent paralysis in the 31 cases is shown in the following table:

Both legs	in 9 cases.	Both arms	in 1 case.
Right leg	" 6 "	Arm and leg	" 2 cases.
Left leg	" 6 "	Trunk with both legs	" 1 case.
Right arm	" 3 "	Trunk with left leg	" 1 "
Left arm	" 1 case.	Both legs and both arms	" 1 "

The records of the muscles affected in each case are too extensive to warrant their reproduction. It is sufficient to say that in the large majority of the cases in which one or both legs were affected (26 cases) the paralysis was finally limited to the muscles below the knee; the anterior tibial group, the posterior tibial group, and the peroneal group being affected in the order of frequency named. In but 4 cases were the muscles above the knee paralyzed. In no case were all the muscles of the limb equally affected. When the anterior tibial group was totally paralyzed, the posterior tibial group, though often partially paralyzed and somewhat atrophic, usually preserved some power and pulled the foot downward into a position of talipes equino-varus. When the peroneal

group was paralyzed, the persistence of power in the others favored the development of deformities. It is the unequal distribution of the paralysis which leads to the conditions of late rigidity and malpositions which are so often seen. For it is found that the flaccid paralysis in these cases is frequently followed after a year or more by a condition of rigidity and contractures with resulting deformities. Such contracture has this characteristic, that it develops in the muscles which are not the ones most paralyzed; thus contrasting sharply with the rigidity found in cerebral paralysis. For example, in the patient whose picture is presented (Fig. 55) the inability to flex the first phalanges or to extend the second and third phalanges in the hand on account of paralysis of the interossei and lumbricales, while the long flexors and extensors are not paralyzed excepting the extensors of the radial side of the wrist, has resulted in a permanent state of deformity in the hand as shown in Fig. 55. But the contracture is easily overcome by passive motion. In another patient suffering from cerebral paralysis, whose picture is given in the next section (Fig. 60), the flexion of the elbow and of the wrist are due to the fact that all the muscles of the arm are suffering equally from voluntary paralysis, but the balance of reflex spinal action being in favor of the flexors, these predominate over the extensors. Here it is difficult to overcome the spastic rigidity. If any doubt should arise as to the nature of the paralysis in a case of secondary rigidity, the spinal form

FIG. 55.—Infantile Spinal Paralysis of Left Upper Extremity. The deformity of the hand and the inequality in the size of the scapulæ are noticeable.

can always be diagnosticated from the cerebral form, by the presence in the latter of mechanical and reflex activity, which in the former is always lost.

When the arms are affected by paralysis the entire arm is rarely involved, though many of the muscles may be affected, as is seen in the patient, whose picture is given (Fig. 56). Usually the paralysis is limited in extent and the cases here collected bear out the observation of Remak that it is easily possible to separate an upper-arm type from a lower-arm type. In the former the muscles affected are the deltoid, biceps, brachialis anticus, and supinator longus. In the latter the muscles affected are the extensors or the flexors of the wrist and fingers—rarely both together. Cases have also been recorded in which the only muscles affected were the intrinsic muscles of the hand. Remak calls attention to the fact that the supinator longus and the long extensors of the fingers, though lying side by side, are very rarely paralyzed together.

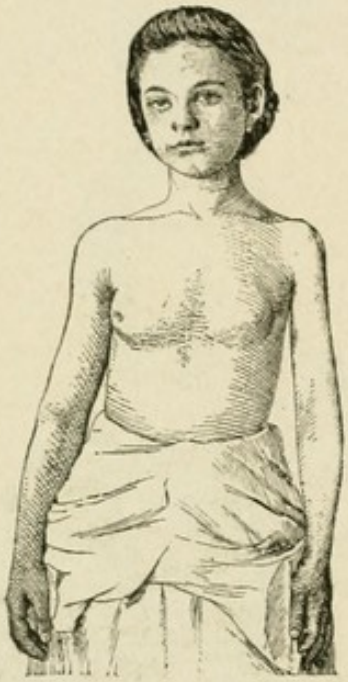

FIG. 56.—Infantile Spinal Paralysis of the Left Upper Extremity. The atrophy of the entire extremity, including the bones of the shoulder, is evident. The girl is now 13; the onset occurred at the age of 3.

The reason for the peculiar distribution of the paralysis in cases of spinal paralysis will be seen at once if reference is made to the facts embodied in the table of localization of spinal functions.[1] It is there made evident that the groups of cells which govern the motion and nutrition of the different muscles lie in various segments of the spinal cord, and that an acute inflammation limited to one or at most two segments (as the lesion in anterior poliomyelitis usually is) will affect certain groups of cells only, while other groups lying above or below the affected segments will escape. Thus when a lesion

[1] Chapter X., pages 128 and 140.

lies in the upper cervical region it will naturally produce the upper-arm type of paralysis, and the supinator longus will be necessarily involved because its group of cells lies side by side with those of the biceps and brachialis anticus, whose function of flexion it materially assists; while if the lesion is situated low down in the cervical cord in such a place as to affect the group of cells related to the extensor muscles on the forearm, the supinator longus necessarily escapes, since its group is far above the lesion.

In any case of infantile spinal paralysis, if the exact muscles paralyzed are determined, a reference to the table will indicate at once the extent of the lesion in the cord (see Fig. 37, page 131).

Another fact is brought out by reference to the table which explains the varying degree of the paralysis in many cases, and its great extent at first and subsequent regression. Each group of cells in the cord has a considerable longitudinal extent,[1] and may be seen to lie not merely in one segment, but often to form a part of two or three segments.[2] And such groups are not of equal length nor arranged side by side at all levels, but one may overlap, as it were, another, as is seen in the diagram (Fig. 57). The consequence is that if one segment only of the spinal cord is destroyed, one entire group may be destroyed if it is a short one, but others are only partly destroyed, the parts of the group which extend into adjacent

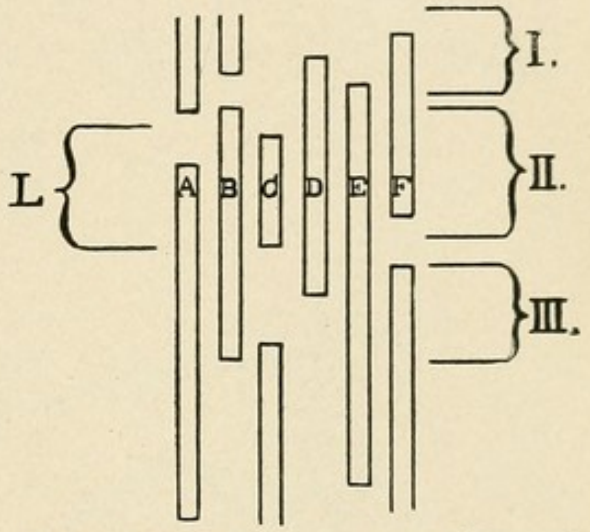

FIG. 57.—Diagram of the Arrangement and Extent of Groups of Cells in the Spinal Cord. Some groups are much longer than others, and lie in several segments.

[1] For an exact description of these groups and their extent, the reader is referred to my article on the localization of spinal functions, Amer. Jour. of Neurology and Psychiatry, Aug., 1884.

[2] This fact seems to be overlooked by Thorburn in his remarks on localization of motor groups.

uninjured segments escaping. Hence the symptomatic result of the lesion will be a total paralysis of the muscle corresponding to the short group, and only a partial paralysis of the muscles corresponding to the long groups. The latter may be at first entirely put out of action by the shock or intense collateral congestion, but subsequently, since a half or even more of their spinal cells remain intact, the function is gradually resumed, and, parallel with this, there is some recovery

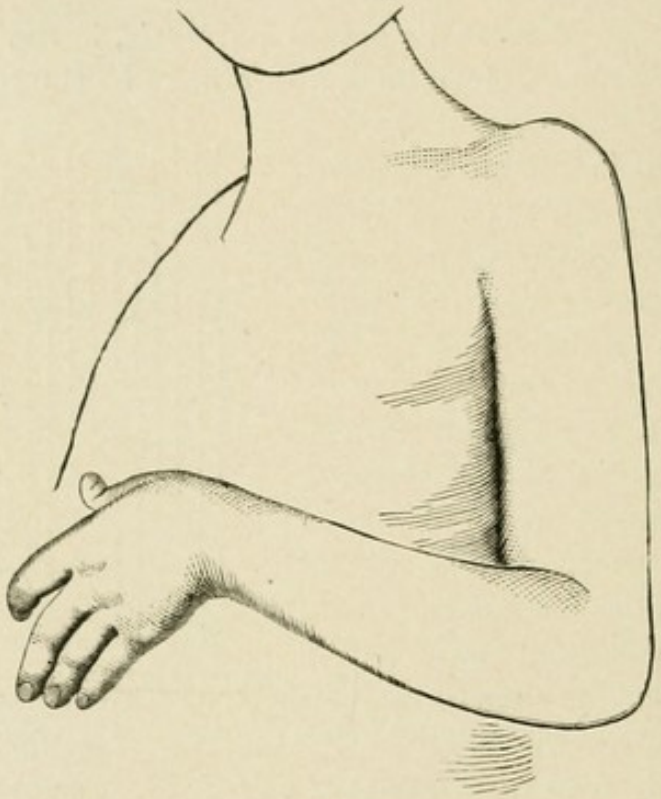

FIG. 58.—Infantile Paralysis of the Left Hand and Forearm. The position is due to paralysis of the extensors on the radial side of the forearm.

of power in the muscles. In some cases of anterior poliomyelitis the lesion found after death has been limited exclusively to a single group of cells in one segment, the groups lying about it being unaffected. This was the case in a patient, observed by Prevost, who had a paralysis of the muscles of the thenar eminence only. In such a case the recovery of the majority of the muscles after the acute onset is due to the fact that as the congestion attendant upon that onset subsides, the groups which are uninjured resume their function.

This pathological explanation of the symptoms in infantile spinal paralysis applies, of course, to all cases, both those of paralysis of the arm which have been cited as examples and also those of paralysis of the trunk and of the leg. The rarity with which the trunk and abdominai muscles are found to be paralyzed is due to the fact that the groups of cells governing them are very long and very narrow and extend through several segments of the dorsal region of the cord. A lesion which is usually of short extent will, therefore, not involve the long groups seriously. That the trunk does not, however, always escape is shown by the picture of one of the cases (Fig. 59), and by the case that Birdsall has reported in which the abdominal muscles of one side were involved alone. In such a case the longitudinal extent of the lesion in the cord must be considerable.

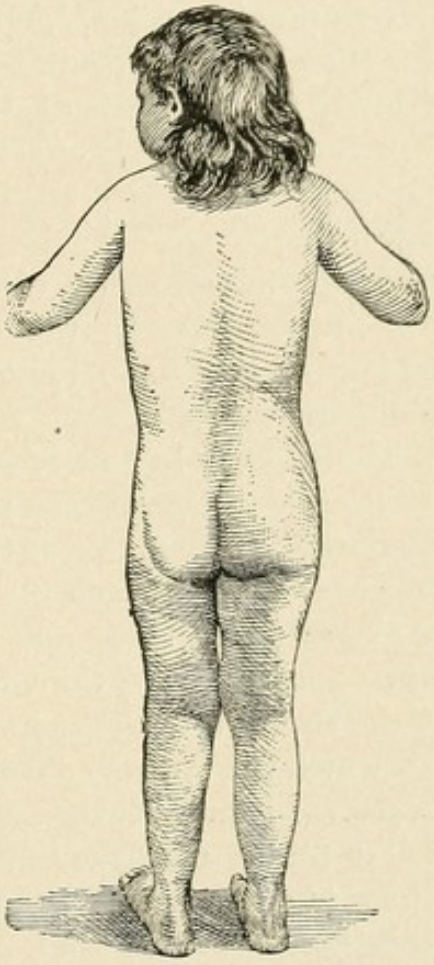

FIG. 59.—Infantile Spinal Paralysis. The atrophy of the left side of the trunk and of the entire left lower extremity is evident. The child is now 3 years old; the onset occurred at the age of 1 year.

The atrophy in infantile spinal paralysis is a very marked feature. It has led to the nam *atrophic paralysis*, which has been adopted by Gowers. It develops rapidly, and it is usually permanent in some of the muscles. In none of the 31 cases did the corresponding limbs on the two sides present the same measurements in circumference. In several cases of such paralysis which have recovered the power of motion to a very great degree, the difference in size remains. The atrophy may be less evident in fat children, especially before the age of four. In many cases there is also a decided difference

in the length of the limbs. The lesion affects the rapidity of growth in the paralyzed limb, so that the unaffected one soon outstrips it. Thus in the case shown in Fig. 59, the left leg is half an inch shorter than the right one, two years after the onset of the disease. This difference gives rise to much deformity in some cases, marked scoliosis resulting from the shortening of one leg. This has been observed in two cases. It is therefore important to bear this in mind in ordering appliances for support of the limb. In cases in which the growth of the bone is affected, the lesion lies deep in the gray matter nearer to the central canal than in other cases; see page 141.

A secondary effect of the atrophy and paralysis is to weaken the support given by the muscles to the ligaments of the joints; hence the joints become abnormally movable and subluxation may occur. This is shown in Fig. 56.

The change in the response of the muscles to electrical irritation is of importance in regard to prognosis as well as to diagnosis. Duchenne was the first to observe that those muscles recovered in which the faradic reaction persists. It may also be said that those muscles will recover in which the faradic reaction returns, though lost, within six weeks. The test for the reaction of degeneration therefore affords the information which the parents chiefly desire as to the prospect of recovery. In one private case under my care the left leg was at first totally paralyzed, but the faradic reaction did not disappear in any muscle except the tibialis anticus. After four weeks it returned in that muscle, and at the end of one year all muscles, except the tibialis anticus, had regained their natural size and power, and after two years the recovery was perfect in the entire limb. In the muscles which present at the end of six weeks the reaction of degeneration, I have never seen a perfect recovery, although I have seen a great progressive improvement. Thus in all the cases of which pictures have been given, the persistent use of galvanism (applied in the same manner as has been described in the treatment of Erb's paralysis), in the one case for two years, in the other case for one year, has been followed by a steady improvement in both

power and nutrition. Yet I am convinced that the degree of improvement possible in any case is dependent entirely upon the extent of the lesion. If the entire group of cells governing a muscle is destroyed, no treatment will avail to restore the muscle. If, however, some part of the group remains, a part of the muscle is capable of recovery, and this part may be so developed by exercise, voluntary or electrical, as to in the end nearly do the work of the normal muscle. The electrical application does not affect the lesion, but it counteracts its effects.

The total absence of sensory symptoms is characteristic of infantile spinal paralysis, and in the absence of a history of the onset might be the only means of differentiating it from Erb's paralysis. In the majority of cases, however, the manner and time of the beginning of the two diseases present such strong contrasts that they cannot be confounded. And the distribution of the paralysis in anterior poliomyelitis rarely resembles exactly that in Erb's paralysis.

This is the only form of spinal paralysis observed with any frequency in childhood; hence it is not necessary to consider its differential diagnosis from other varieties of myelitis, a diagnosis not always easy in adults.

In the treatment of infantile spinal paralysis, the chief object is to increase the nutrition of the limb and to protect it from injury. It is always cold, hence it should be well wrapped up in winter. It is always heavy and in the way, hence it should be carried in a sling or aided by a brace, a measure which also counteracts the tendency of gravitation to stretch the ligaments. The means of aiding nutrition are rubbing, massage, hot and cold douches, and electricity applied in the same manner as in Erb's paralysis. Such treatment must be kept up faithfully for several months, or even for years, and therefore may be intrusted to intelligent parents, and regular attendance enforced upon unintelligent clinic patients. Great aid is afforded by the application of proper braces, and the ingenuity of the physician must be exerted to devise the exact form of apparatus which shall supply the lacking muscular strength without overloading the weak limb.

A great aid to recovery is the constant practice of systematic, well-planned gymnastic exercise, so adapted to each case as to bring out all latent power in weakened muscles.

(3) INFANTILE CEREBRAL PARALYSIS WITH RETARDED DEVELOPMENT.

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Under this title are included all those cases resulting from lesions situated above the level of the pons Varolii. Such lesions may exist in the cerebral meninges, cortical motor centres, or pyramidal tracts of either hemisphere as they traverse the internal capsule, crus, or pons (Fig. 26, p. 94). But in the majority of the cases the lesion is cortical in location.

Cerebral paralyses are easily differentiated from those of poliomyelitis, inasmuch as in the latter there are found, concomitant with the flaccid paresis, a loss or diminution of the reflexes, rapid atrophy, no rigidity, and a degenerative electrical reaction. Cerebral paralyses, on the other hand, usually begin in children suddenly, with convulsions or spasms and with fever and delirium; there is manifested more or less brain disturbance; the paralysis is of the hemiplegic type; there is much tendency to rigidity in the paralyzed limb; the reflexes are exaggerated; there is neither rapid nor extreme wasting, and the electrical reactions are normal.

Of the 36 cases of infantile cerebral disease examined at this clinic from February, 1888, to January, 1890, 20 were males and 16 females; 11 were left hemiplegias, 9 were right, 4 were paraplegias, 1 was a monoplegia, 1 a diplegia, and there were 10 cases of maldeveloped brain without paralysis.

Etiology.—In nearly all of these cases, the onset could be dated before the age of four years. Seven date from birth, in 2 of which a distinct history of injury was obtained; these were delivered with instruments; one was a complete hemiplegia and the other a monoplegia. The latter presents not only a brachial palsy but a pear-shaped skull. The child's intellect is, however, bright.

In none of these cases could a syphilitic history be obtained and in none could maternal impressions be proven.

Among the cases which dated from birth, it is probable that meningeal injury was the cause of paralysis. Dr. McNutt, in her complete monograph on this subject,[1] has shown how this can occur. When labor is protracted and the head presents, the mechanical compression congests the basal meninges and rupture of the vessels often ensues. In breech presentations the parietal meninges are also congested and suffer a like injury. Such a rupture is predisposed to by the existence of fatty degeneration in the vessels, a condition which Hübner has found not infrequently in childhood.

The accompanying damage to the brain is the cause of the convulsions, of the paralysis of the limbs, and of the permanent symptoms. These cases vary much in degree, and it is only in the more severe forms that all of these symptoms are present.

One of the patients with hemiplegia dating from birth was a twin, the other child being asphyxiated during delivery, thus showing the greater danger of paralysis in double births.

Cerebral tumors were diagnosticated in 2 cases, but these were not detected until a few months after birth.

Eight hemiplegic cases dated from convulsions, and four from teething. One case of paraplegia followed a fright which occurred at the age of four years, when the child was left alone at night.

Four dated from scarlet fever and cerebro-spinal meningitis and two from diphtheria. Strümpell and Bernhardt have noted the relations of these palsies to acute diseases.

In the remaining cases no direct causes could be ascertained.

Symptomatology—Paralysis.—Among those cases with no pronounced onset it was noticed, first, that one arm or one leg did not move; sometimes none of the limbs were under the full command of the will. These latter cases, if slight, are apt to receive less attention than the one-sided cases, because

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[1] Amer. Jour. of Med. Sci., Jan., 1885.

no comparison can be made between the two sides. A sudden onset with convulsions had occurred in the majority of the cases. The convulsions were usually confined to one side; in a few cases they were general. The head was turned backward, the thumb was in a position of inversion, and there was persistent rigidity of the part convulsed. In many cases the convulsions were followed by loss of consciousness, which lasted from a few hours to many days. In some there was present a rise of temperature which was only transient, but this was often quite high when the convulsion ceased. Delirium occurred in about half of the convulsive cases.

The paralysis which was discovered when the child recovered consciousness was generally a complete hemiplegia; less frequently it was incomplete, the paresis being more marked in the arm and resulting gradually in total loss of power; and in some instances the entire loss of power was established only after repeated convulsions.

Among the facial muscles the frontalis and orbicularis palpebrarum usually escaped, and the inferior muscles which were paretic made a rapid recovery as a rule. If the paralysis is in the right side—as it was in 3 cases—it may be accompanied by distinct motor aphasia in children who have already acquired the power of speech. Aphasia does occur rarely with left-sided paralysis in children.

The residual paralysis resembles the hemiplegia of adults, being most marked in the arm and next in the leg (Fig. 60). It differs, however, in the more rapid recovery of the leg. In all infantile cases the leg improves much more speedily than the arm and the paralysis may completely disappear, an event which rarely happens in the upper extremity. The atrophy in the leg is not so marked and the growth is not frequently seriously arrested. When the leg is of fair size and the muscular development is good, there may be only a slight dragging of the limb perceptible upon rapid walking, instead of the hemiplegic curve so noticeable in an undeveloped limb and in extreme paralysis. The arm manifests much atrophy and weakness. The power sometimes returns partly in the shoulder and elbow and to a less extent in the hand.

In the four cases of paraplegia the atrophy and arrested development were manifested alike in each leg.

Reflexes.—The reflexes at the knee were exaggerated in 11 of the cases. Ankle-clonus was obtained in four instances. The wrist reflex was also exaggerated. In the diplegia both reflexes were absent. In paraplegia the rigidity of the muscles sometimes prevents the examiner from obtaining a knee-jerk.

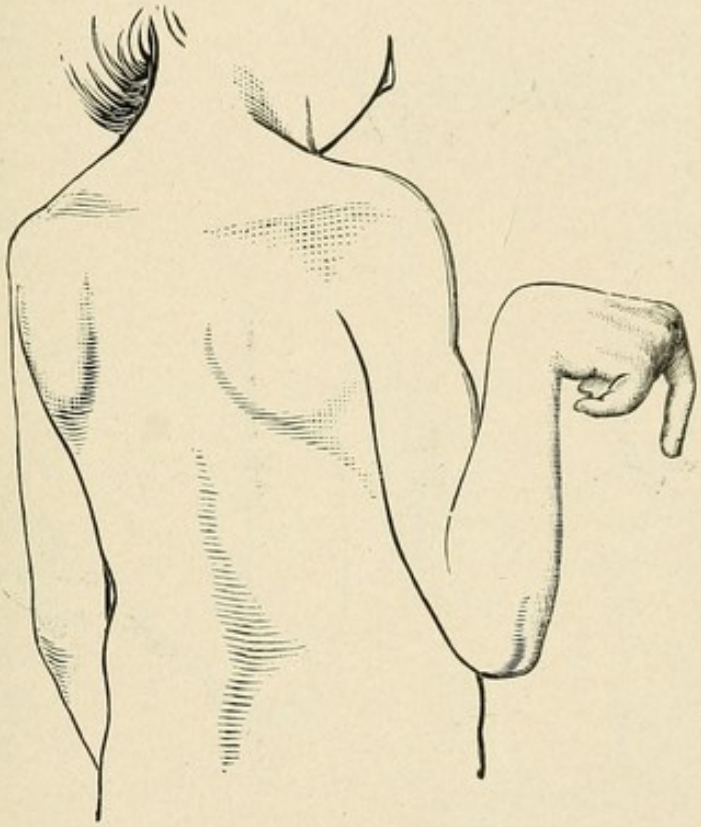

FIG. 60.—Infantile Cerebral Paralysis of the Right Arm with Athetoid Motions. Girl of 8; hemiplegia at age of 2. Face and leg have recovered; arm still paralyzed.

Rigidity and Contracture.—In 14 cases the rigidity was well marked. In the extreme cases where this rigidity had lasted for years, contractures had resulted in which condition relaxation was no longer possible on account of the structural changes which had taken place in the muscles. Unless contractures have set in, the rigidity disappears during sleep, and is increased by emotion and aggravated by any forcible attempt to overcome the spasm. The face never participates in any form of late rigidity. The position which the upper limb as-

sumes is that of adduction of the shoulder, flexion of the elbow, flexion and pronation of the wrist, and marked flexion of the fingers. The leg assumes the position in which the extensors and flexors are each rigid, the heel is elevated, foot inverted, producing a talipes equino-varus.

Athetosis.—Athetoid movements were present in four of the cases. The character of the movements was very slow

FIG. 61.—Infantile Cerebral Paralysis of the Right Arm with Athetoid Motions.

(Figs. 60, 61, and 62). They were usually limited to the arm or hand. It was adducted, the elbow was flexed, and occasionally the arm was carried backward by the spasm and rotated inward, so that the hand was held against the lumbar region with the palm turned outward; usually the hand was carried forward against the chest with the palm turned downward. Three successive positions are shown in Figs. 60, 61, and 62. The girl was eight years old, and her hemiplegia had occurred at the age of two. The motions were constant during her

waking hours, and were increased by any attempt at voluntary motion.

In an athetoid hand the interossei and lumbricales which flex the metacarpo-phalangeal and extend the phalangeal joints are affected; rarely are the long extensors and the long flexors affected. Therefore the hand is usually in the so-called interosseal position, with flexion of the proximal and extension of the middle and distal phalanges. The illustration shows these athetoid movements of the hand (Fig. 63).

The athetoid movements of the toes correspond to those of the fingers in point of action. In severe cases the movements continue during rest and may persist during sleep.

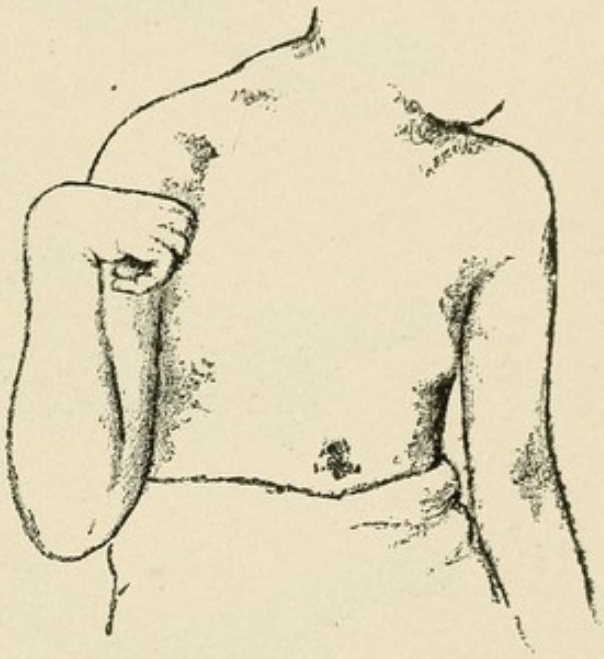

FIG. 62.—Infantile Cerebral Paralysis of the Right Arm with Athetoid Motions.

Post-Hemiplegic Chorea.—This was present in three cases. It occurs in partially but never in completely paralyzed limbs, and usually appears simultaneously with a marked diminution of the paralyzed symptoms. The spasms as a rule become gradually established as motor power returns, although they may appear suddenly or seem to be induced by an effort to move the paralyzed limb. The movements affected by choreiform spasm are greater in the fingers and toes and are less as the shoulder and hip are reached. These movements are of a wider range than those of hemiplegic tremor; they cease during sleep and are aggravated by voluntary effort. The tremor which succeeds hemiplegia is fine and is limited to the seat of paralysis. In one case of right hemiplegia and in the case of diplegia, which was of the flaccid variety, there were choreiform movements of the head alone. The latter case was one of typical chorea nutans.

One interesting case, from a diagnostic point of view, was that of a right hemiplegia and a left hemichorea in a boy of nine years of age. On examination it was ascertained, how-

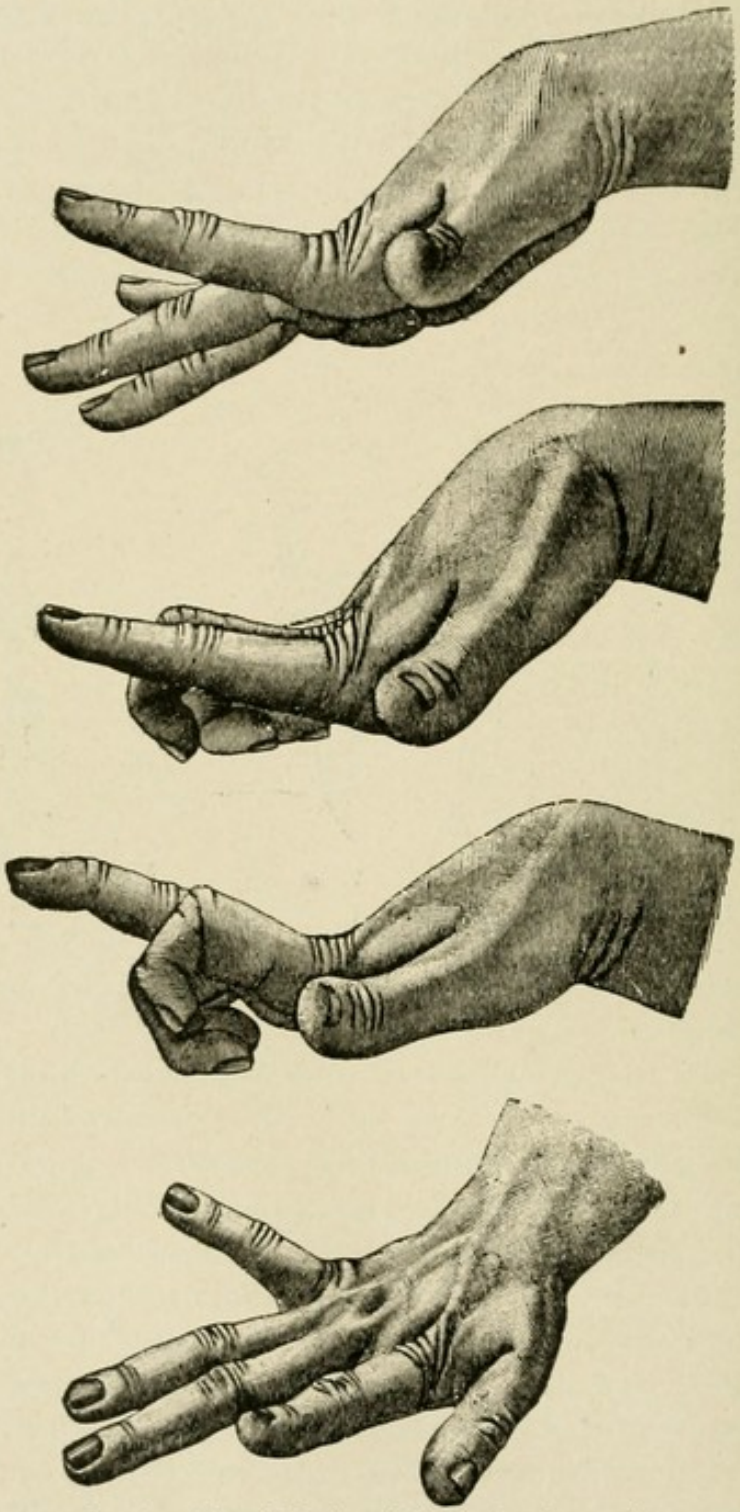

FIG. 63.—Athetoid Motions of the Hand. (Strümpell.)

ever, that the hemiplegia was of four years' duration, while the hemichorea had lasted but a week and was idiopathic.

Associated Movements.—The face, while presenting no apparent rigidity, will often, during a smile, overact on the side which was paralyzed, and this is a most valuable diagnostic indication in cases of old infantile hemiplegia, of which little trace may remain elsewhere.

A strong effort to grasp with the unaffected hand will sometimes cause a similar movement in the paralyzed hand, and *vice versa*. This was present in 3 cases.

More frequently the paralyzed part is affected during the act of yawning and stretching.

Epilepsy.—This was present in 5 cases. The epileptiform attacks commenced simultaneously with the hemiplegia in 3 cases, and occurred subsequently in 2. They are far more frequent after infantile than after adult hemiplegia. They were as a rule confined to the paralyzed side. In one case they were general. Aura was present in the majority of the cases, and in four of them it began deliberately in some portion of the paralyzed side.

In hemiplegia of sudden onset due to vascular obstructions, softening or sclerosis follows, the brain tissue adjacent is usually damaged by collateral changes, and the nerve cells suffer a change in the nutrition and function, causing instability which is continued by their repeated discharge. It is probable that in idiopathic epilepsy a sudden discharge of nerve cells or the violent liberation of nerve energy occurs in the cortex, a theory to which this class of cases lends support.

Mental Defects.—These were very common and presented every conceivable degree, from a mere tendency to some functional disturbance up to pronounced idiocy. The cranium was misshapen. The palate was arched high and the teeth were in many cases defective.

Pathology.—The pathological conditions which produce infantile cerebral hemiplegias may be assumed to be of two kinds: one is vascular occlusion, or rupture, and the other, as maintained by Strümpell, is inflammation of the gray mat-

ter of the cortex—a polio-encephalitis analogous to polio myelitis.[1]

The vascular theory has much to support it. It is the common cause of cerebral cavities known as porencephalus. Vascular rupture is a known complication of such general diseases as frequently precede the cerebral palsies.

Whether the lesion is a result of embolism or of thrombosis is uncertain; the former has generally been assumed, and in support of this Hübner reports a case of bilateral palsy with rigidity and trismus developed during bronchitis in a child one year old. Cavities were found in both hemispheres and in the pons with clots in the middle cerebral arteries; but these clots were canalized and the arteries beyond were pervious. In the majority of cases of proven embolism there has been an obvious source for the occluding plug—usually endocarditis.

It is known that primary thrombosis occurs in the sinuses of children, and infantile hemiplegia may be the result of it.

In cases with no evident softening in which there is no cavity, but only shrinking and induration of a part of the cortex, Gowers suggests that the lesion is probably thrombosis in a surface vein, and that the reason why the lesion is not found more frequently at autopsies is because in fatal cases the clot usually spreads into a sinus before death, and so the case is regarded as one of sinus thrombosis. The closure of a vein does not cause softening of the entire cerebral tissue from which the blood should pass into the vein, but merely venous congestion, minute extravasation, and punctiform softening—a condition that may well leave the state of atrophy and induration met with in some cases. Lastly, in many cases, a large clot due to the rupture of a vein in the pia compresses the brain cortex and prevents its development. This is probably the most common cause of infantile hemiplegia. The theory of Strümpell, that an acute inflammation limited to the motor

[1] For a complete discussion of this subject, the reader is referred to an excellent little book on "Cerebral Palsies of Children" by Prof. William Osler, and to a monograph, with bibliography, by Drs. Sachs and Peterson; Jour. of Nerv. and Ment. Dis., May, 1890.

area of the cortex may occur, has no pathological basis as yet; though the analogies observed between the course of the disease in brain and cord cases offers some ground for its acceptance.

Whatever the original lesion in cerebral palsies may have been, the final result in more than one-half of the cases is a condition of atrophy and sclerosis of the convolutions. The most common seat of this atrophy is the motor area of the brain, and hence the frequency with which paralysis is found in such cases; but other areas may be involved, and many cases of imbecility from sclerosis of the frontal region, or of blindness from atrophy of the visual region, or of deafness and consequent dumbness from atrophy of the convolutions about the Sylvian fissure are on record. Such atrophy may be of sufficient extent and depth to produce a cavity in the brain substance. In addition to atrophy and sclerosis it is common to find cysts, and the remains of old clots in the cortex. In but a few of the recorded cases could embolism or thrombosis be actually proven. If the condition described by Strümpell occurs—a true inflammation of the cortical gray substance limited to the motor zone—it is a rare affection.

Prognosis.—The recovery of the face is usually assured. The leg is nearly always greatly improved and often recovery is complete. There is generally a permanent disability of the arm, including atrophy and slight contracture.

Treatment.—This at the onset during the active symptoms demands the use of sedatives, cathartics, cold to the head and leeches to the temples, or counter-irritation to the neck.

Subsequently, as the temperature of the paralyzed parts is usually lowered and the circulation is sluggish, the limbs should be wrapped in wool. Massage should be practised daily for half an hour, with the use of olive oil or other unguenta. Faradic electricity should be resorted to and persevered in, with more frequent applications than are wont to be employed. It should be applied especially to the extensors to overcome flexion.

When the rigidity and contractures are marked, warm

baths with massage and with passive flexion and extension are of service.

The palsied arm of hemiplegia cannot be much benefited, but in cases of spastic diplegia and paraplegia, manipulations and orthopædic measures may enable a crippled child to walk.

The following cases illustrate the disease and demonstrate the most common forms observed.

CASE XXX.—INFANTILE CEREBRAL PARALYSIS—ONSET AND PROGRESS.

J. C., aged six, was seen by Dr. Starr in consultation with Dr. Gibney February 11th, 1886.[1] He had been feverish and restless for two weeks, and on the 4th of February had been seized with a severe convulsion attended by high fever (106°), which had yielded to baths after lasting all night. During the following four days a condition of right hemiplegia with hyperæsthesia and with aphasia developed, the arm being affected first and most seriously, and occasional spasmodic twitching being observed during this time in the affected limbs without loss of consciousness. During these days the temperature varied from 100° to 104°.

When examined a typical condition of right hemiplegia was found with slight hemianæsthesia and motor aphasia, but no disturbance of consciousness or of understanding. His pupils were normal. The face, tongue, arm, and leg were all involved, the paralyzed parts being relaxed, not hot, not cyanotic or œdematous, and reflexes being increased on the paralyzed side. The evacuations occurred involuntarily.

During the following month a gradual improvement occurred, and by the end of three months the aphasia was passing off so that he said a number of words; the face and tongue no longer showed evidence of paralysis; the arm was seriously affected, but he could raise it slightly and move the fingers, which showed some tendency to contracture in a flexed position; the leg had so far recovered that he could walk. Reflexes were still exaggerated on the paralyzed side and slightly

[1] Reported by Dr. Gibney, N. Y. Med. Record, 1886.

so on the other. Hemispasms continued at intervals. During the past four years growth and improvement have been steady, but the paralysis in the arm remains and he has not entirely recovered his speech.

CASE XXXI.—INFANTILE CEREBRAL PARALYSIS—TERMINAL CONDITION.

Mary C., aged nine, came to the Vanderbilt Clinic February 18th, 1889. She was found to be a healthy child, excepting for the presence of a weakness of the left arm and leg, the arm being chiefly affected. There was a decided inability to use the hand; only the large motions of the joints could be performed, fine adaptations being impossible; there was a general imperfect development of the limb, the arm and forearm being three-quarters of an inch in circumference less than those of the right side, but the atrophy was not limited to any one muscle or group of muscles. The affected limb was cooler, bluer, and the skin rougher. It was very stiff to passive motion. Sensation was equal on both sides. The length of the arm was one and one-half inches less than that of the right one, and the size of the hand was less. The same differences were noted in the leg to a less degree. The face was not affected, nor was speech involved. The parents stated that this condition had been present from birth.

CHAPTER XIV.

MULTIPLE NEURITIS.

The discovery of the disease.—Pathology.—Etiology.—Symptoms: sensory, motor, electric changes, deformities, vaso-motor, trophic, mental.—Course and duration.—Diagnosis.—Types of the disease.—Beri-beri.—Prognosis.—Treatment.

MULTIPLE neuritis is a disease of the peripheral nervous system, consisting of a more or less widespread, symmetrically distributed degeneration in the nerves.

This disease has only recently been generally recognized and diagnosticated. The symptoms which it presents had been for many years known and classified under the terms "alcoholic paralysis," "general spinal paralysis," "acute ascending myelitis," and had been erroneously referred to disease of the spinal cord. In 1864, Duménil,[1] of Rouen, recorded a case of paralysis in which lesions of the nerves were found after death, and two years later he published another similar case.[2] But no notice was taken of these cases until 1876, when Eichhorst, of Berlin, recorded a case[3] of widespread disease of the peripheral nerves. This article of Eichhorst, and those of Joffroy,[4] in 1879, Leyden,[5] 1880, Lancereaux,[6] 1881, and Grainger Stewart,[7] 1881, attracted attention to the new disease, and many observations followed: so that in 1884 Buzzard[8] was able, in the Harveian lectures, to present a clinical picture of the affection, and the writer in 1887,[9] in the Middleton Goldsmith lectures, was able to collect a large number of facts bearing upon the pathology, etiology, and diagnosis of this disease. Ross has published[5] a series of articles on the disease recently (1890) which sum up concisely our present knowledge of the affection.

Pathology.—The lesions in multiple neuritis are limited to the peripheral nerves, and are found to be more intense in

the peripheral branches of those nerves than in the nerve-trunks. The condition found is one of degeneration, similar in all respects to the degeneration present after injury of a nerve. The medullary sheath of the nerve-fibre is swollen, is divided into segments of semi-fluid consistency and fatty appearance, its continuity is broken, and its insulating function suspended. This may be the only lesion in a very mild case, and regeneration may follow the degeneration (Fig. 64). In more severe cases the degeneration proceeds, the medullary sheath is broken up into fine granules of fat or into a molecular débris and is absorbed, and the axis cylinder is not only swollen, degenerated, and divided, but may also be changed into a granular mass and completely absorbed, so that an empty sheath of Schwann alone remains as a trace of the former

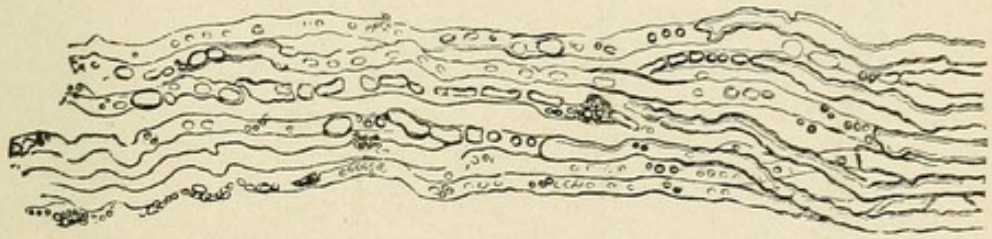

FIG. 64.—Multiple Neuritis. Lesion in the medullary sheath in a mild case. (Mayer.)

nerve-fibre. In all the process the sheath of Schwann participates, its connective-tissue nuclei being swollen and segmented, and new nuclei appearing inside and outside of it. All these processes may be present simultaneously in various fibres of a nerve (Fig. 65).

After the degeneration has proceeded for a time, regeneration begins. This takes place either by a development of new axis cylinders and medullary sheaths from nuclei in the old sheath of Schwann, as Neumann [10] holds, or by the growth of new axis cylinders outward from the undegenerated part of the old fibre into the old sheath, as Ranvier teaches.[11] This form of neuritis, in which the changes are especially marked in the nerve-fibres, is termed parenchymatous neuritis, in distinction from the second form, in which the connective-tissue elements take a more active part in the inflammation. Interstitial neuritis appears to have the same origin, and to occur under the

same circumstances, as parenchymatous neuritis, but in it the endoneurium and perineurium take part in the process, and the increase of connective-tissue elements in the affected

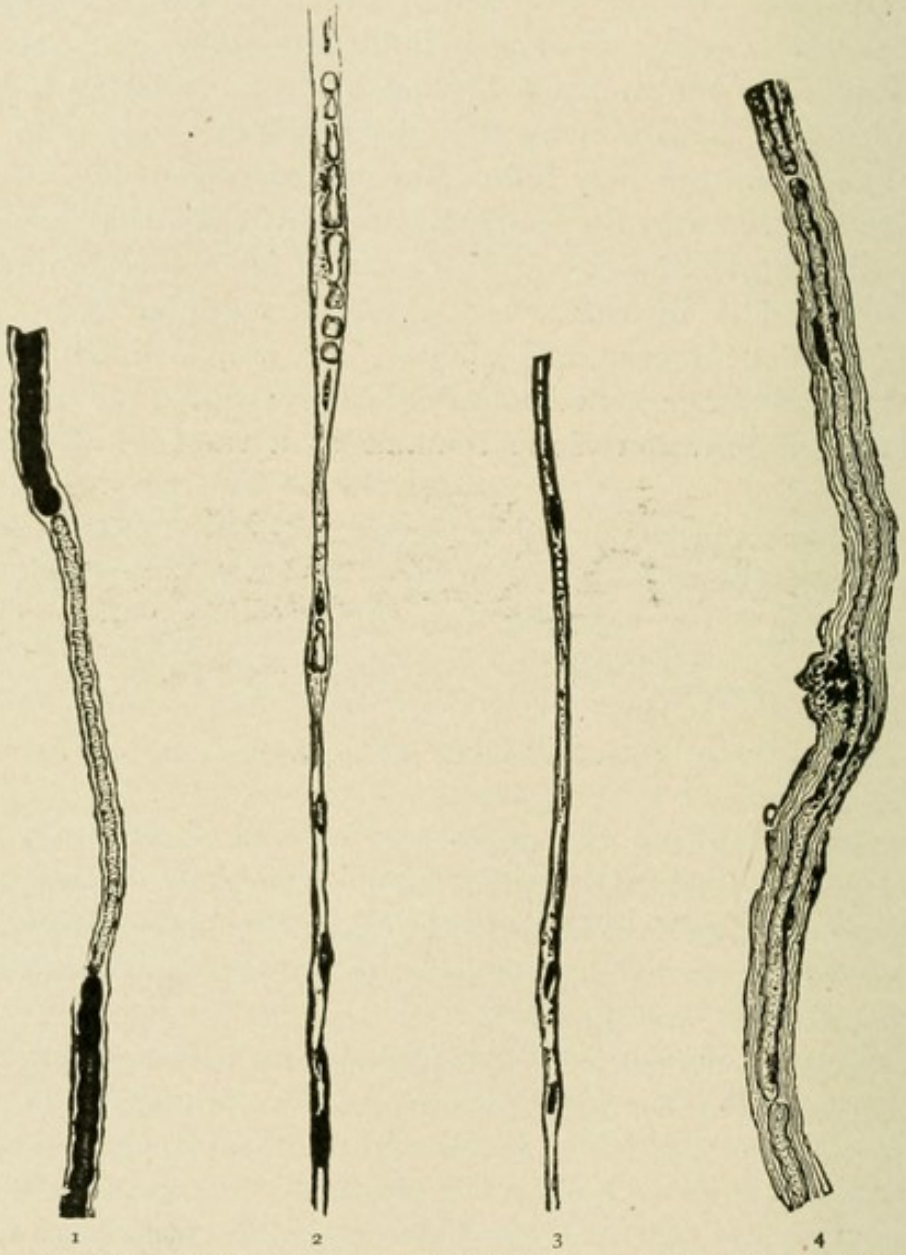

FIG. 65.—Multiple Neuritis (Mayer). 1, Swelling and granular appearance of axis cylinder; 2 and 3, absorption of débris leaving empty sheath; 4, regeneration beginning.

nerves is very noticeable.[14] As far as is known, the process of regeneration does not differ in the two varieties. Lastly, a diffuse neuritis may occur in which both parenchyma and interstitial tissue are affected together (Fig. 66).

Etiology.—There are many causes which produce multiple neuritis, and the cases may be classified according to the etiology.

There are, first, *cases due to toxic agents;* of which alcohol, lead, arsenic, bisulphide of carbon, and illuminating gas are the most active. All these agents produce widespread and serious effects upon the nervous system, but in many cases those effects are chiefly manifested in life and at the post-mortem table in changes in the peripheral nerves. The ma-

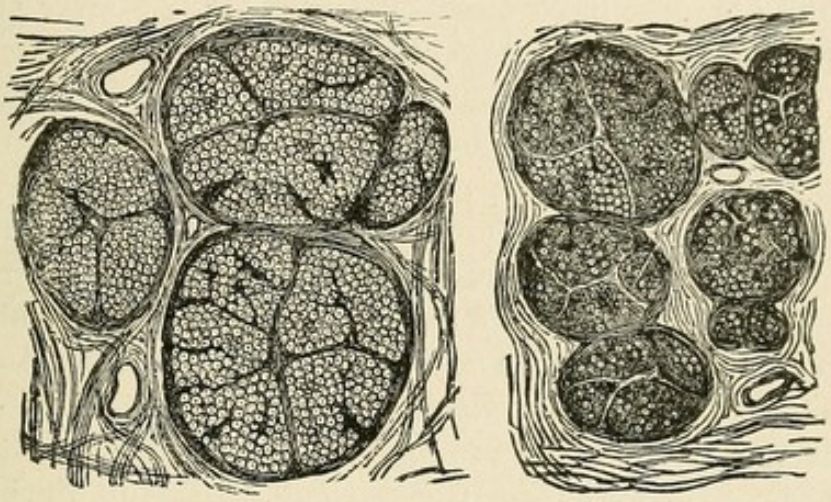

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FIG. 66.—1, Cross Section, Normal Nerve; 2, Cross Section, Diffuse Neuritis. (Joffroy)

jority of cases of "alcoholic paralysis," "lead palsy," and "arsenical pseudo-tabes" are due to multiple neuritis.

There are, secondly, *cases due to infectious agents*, of which those producing diphtheria, variola, typhus, typhoid, malaria, and tuberculosis have been hitherto recognized. In the course of all these diseases, or as a sequel to them, multiple neuritis may develop, and its occurrence is too frequent to be accidental.

There are, thirdly, *cases occurring in epidemics* in many tropical countries, notably Brazil, India, Japan, Africa, and in the East and West Indies. Sporadic cases of this description developing among sailors on shipboard occasionally find their way to our seaports. The disease is called kakké in Japan;

beriberi in India and Brazil. It presents certain features which differ from those of the ordinary form. It has been ascribed to dampness, to a diet of rice or of fish, and to an infectious micro-organism.

There are, fourthly, *cases supposed to be due to exposure to cold* and dampness and to *over-exertion*, or in which no cause is known.

In all these forms the clinical features of the disease are essentially the same, although different cases present a variety of combinations of symptoms.

Males are more liable than females. All ages after puberty are equally represented, excepting in diphtheritic cases, which occur chiefly in childhood.

Symptoms.—The individual symptoms of multiple neuritis are very numerous.

The *sensory symptoms* are the earliest to appear and the last to pass away. In the majority of the cases on record, from whatever cause, numbness, tingling, or formication ushers in the disease. These forms of paræsthesiæ begin in the feet and hands, and extend to the knees and elbows. They may be associated with burning, stretching, boring, or tearing sensations, which distress the patient especially during the onset. But all such sensations usually subside as the affection reaches its height. Their recurrence, as the case goes on, may be regarded as a favorable symptom, however annoying, for they frequently precede recovery, and are among the last evidences of the disease to disappear. Pain is usually present as well as paræsthesiæ. It may occasionally be sharp in character, but is usually moderate and not continuous. At times it may be lancinating, and so severe as to necessitate the use of morphine. But it is rarely as distressing as in cases of locomotor ataxia. Tenderness in the nerves and muscles is a constant symptom. It may be so marked that the limbs cannot be moved or handled, and thus it may interfere with the application of electricity and massage. When the tenderness and pain are referred to the joints, as not infrequently occurs in the early stage of the disease, the case may be mistaken for one of acute articular rheumatism, and if the joints are swollen

or the limbs œdematous, the difficulty of diagnosis is greatly increased.

In addition to these subjective feelings, some demonstrable disturbance of the various sensations is usually present. Hyperæsthesia to touch, and also to electricity, is not infrequently observed during the first few weeks. It is usually followed by some anæsthesia, although this rarely becomes complete. In some cases the loss of tactile sense is quite evident from the onset, either limited to the cutaneous distribution of some special nerve, in which case oddly shaped areas of insensibility will be found; or, as is most often the case, about uniformly distributed over the distal parts of the extremities. When the anæsthesia is at its height the patient has difficulty in locating a touch upon the limb, even though he feels it. The transmission of pain and temperature sensations may then be delayed, but the impressions are usually felt quite acutely. The sense of pressure has been tested in only a few cases, and in those it was decidedly impaired. The muscular sense escapes being involved in some cases, but in others it is the most profoundly disturbed of all the senses. When it is involved, the incoördination and ataxia are well-marked symptoms, and, as already stated, some of the cases have been mistaken for locomotor ataxia, because of the predominance of the disturbance of muscular sense.

These sensory symptoms are usually limited to the forearms and hands and to the legs and feet. In a few cases they have involved the entire extremities, and even the trunk; and one case of facial tingling, with anæsthesia, has been recorded.[12] The skin reflexes are usually preserved. In a very few cases the sensory symptoms have been entirely wanting.

The special senses are rarely affected in multiple neuritis. It is true that optic neuritis has occurred in a few cases, and in two cases hearing as well as sight has been affected. These cases prove that no nerve can be said to be exempt from implication in this disease, but the liability to affection seems to be slight in the case of the nerves of special sense.

The *motor symptoms* are as marked and as important as the sensory. Paralysis, beginning as simple weakness, with a

feeling of fatigue on any exertion, gradually increases in severity, until at the height of the disease it becomes complete. It usually comes on rapidly, so that within two weeks the patient is helpless; but it may be less sudden, and not deprive him of the power of walking and of using his hands for two or three months. In a few cases a very acute onset is recorded, all the symptoms developing within three or four days. The distribution of the paralysis is not uniform at the outset. It may develop in the muscles supplied by a single nerve, and advance to others; it may begin in all the muscles of the legs, and then involve those of the forearms; it may commence in all four extremities at once. It is always more severe in the muscles which move the joints of the feet and hands and the ankles and wrists. It rarely invades those which move the knees and elbows. When the disease is fully developed, all the muscles below the knees and elbows are much weakened or totally paralyzed. In a few cases those of the thighs and arms are also involved, and occasionally the muscles of the trunk and those of respiration become affected and the patient dies. In some cases of multiple neuritis the motor cranial nerves become involved; those of the eye and of the face being most liable to invasion. It is only in fatal cases that the action of deglutition has been affected; and when the pneumogastric is invaded and the heart becomes rapid and irregular, the prognosis is always grave. Spasms rarely occur.

The paralyzed muscles are relaxed, flabby, and atrophied; they may or may not lose their mechanical irritability, but their normal tone is always lost, and hence the so-called tendon reflexes are abolished. To the electric current their excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost, and then the muscles react to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and this fact is quite pathognomonic of neuritis. For in anterior poliomyelitis, where the muscles re-

spond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion. The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found, and the negative pole produces stronger contractions than the positive pole. If the muscles that are not paralyzed be tested, the same electrical changes may often be discovered in them. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle and nerve are, therefore, important symptoms of multiple neuritis. And as the disease goes on to recovery a gradual increase in the galvanic irritability occurs, a fact which is often of much aid in prognosis, if careful measurements of the strength of current used be made by the galvanometer.

As a result and accompaniment of the paralysis, *abnormal positions* are assumed by the limbs. The dropped wrist and dropped foot are quite characteristic of multiple neuritis. But other deformities may be present. In a few cases there have been extreme contractures of all the extremities in flexed position. And not infrequently the appearance of the claw-shaped hand and some one of the various forms of talipes indicates a serious shortening of one set of muscles and corresponding weakness of its opponents. These deformities usually subside as the power returns, or if they do not, they can be corrected by proper manipulation and by apparatus. In a few cases it has been necessary to resort to tenotomy, but a permanent deformity has not been recorded.

The *vaso-motor and trophic symptoms* are less constant than those already described. They are present in the epidemic form in all cases, and are especially severe. In some cases marked œdema has been an early and a permanent symptom. This may develop in the feet and hands, or may appear about the joints. It usually is temporary. The circulation is not impaired any more than is customary in a limb whose muscles are inactive, and coldness and cyanosis are rarely sufficient

to attract attention. Sometimes profuse perspiration is a noticeable symptom, being limited to the paralyzed parts. It may be offensive, and by its evaporation always causes a complaint of coldness. In other cases glossy skin makes its appearance early, and remains until regeneration of the nerves begins. Its disappearance in one of my own cases was the first sign of recovery in the lower extremities. Other forms of trophic disturbance are rarely met with in multiple neuritis. And this is quite remarkable in view of the fact that it has been the tendency of late to refer such trophic affections as ulcerations, bed-sores, gangrene, pemphigus, and various eruptions to lesions of the nerves. It is true that inflammations of the joints, resembling those appearing in acute rheumatism, sometimes occur at the onset of neuritis; but as they disappear quickly, while other symptoms remain, it is improbable that they are to be traced to the changes in the nerves. They may be due to the infectious agent, or to the same obscure cause which sets up the neuritis, or they may be evidence of an attack of acute articular rheumatism, which is in turn followed by neuritis; but they cannot be described as trophic symptoms of the disease, otherwise they would be more constant in their occurrence and more permanent in their duration.

One feature of alcoholic paralysis remains to be noticed, viz., the cerebral symptoms. These are hardly ever wanting. There is at first excitement rising to the degree of active delirium, with illusions and hallucinations of the various senses; there is insomnia, which so soon exhausts the patient if it is not remedied; there is the loss of memory, especially of recent occurrences; and the lack of power of attention or concentration, which prevents intelligent conversation. The indifference to bodily wants may be so great as to lead to uncleanliness, and since paralysis of the sphincter is the rare exception, incontinence is usually to be ascribed to the mental state. It is useless to attempt to get any reliable history of their illness from these patients. Their statements are unintelligible or unreliable. And here it may be well to notice a symptom first remarked by Strümpell. These patients will relate occurrences as having happened recently, with much

elaboration of detail, when, as a fact, the story is entirely a product of their imagination. Thus one patient of my own, who had been confined to bed for many days, told me one afternoon that she had been out to see an eminent gynecologist during the morning; had gone to his office and waited for him several hours; had seen other patients there; and finally had been told by the doctor's brother that he would not return in time to see her, so she had come home again. And this was all related in apparent good faith, so that I have no doubt that she believed that what she said had occurred. With the possibility of such delusions in view, it is evident that the statements of these patients cannot be accepted regarding anything, especially as to their own history.

One patient, who was admitted to Bellevue Hospital during my service there, told me a different history of her case every day for a week, and it was only by interviewing her friends that the correct account was obtained.

A negative symptom of some importance is the absence of any interference with the automatic acts controlled by the sphincters, in the majority of cases.

The onset is usually sudden, and is sometimes, in toxic and spontaneous cases, accompanied by a marked febrile movement, with chill, and temperature of 103° to 104.5° F. The fever may persist for several days, but usually subsides spontaneously, and does not recur. In a few cases there has been a constant elevation of temperature from one-half a degree to one degree and a half above the normal; and an increase in the rapidity of the pulse throughout the disease has been noticed. A pulse of ninety need give no alarm, but if it runs up to one hundred and forty and becomes irregular, there is reason to believe that the disease has attacked the vagus nerve, and then the prognosis becomes serious, though not by any means hopeless.

The duration of the disease varies considerably in different cases. An average of 25 cases gives seven months as the probable time required for complete recovery. But in these cases the duration varied from two months, in the most favorable, to sixteen months, in the most refractory.

Diagnosis.—It is by reference to the etiology and to the course of the case, as well as to the combination of symptoms present, that the diagnosis of multiple neuritis must be reached. The most important diagnostic symptom is tenderness along the course of the peripheral branches of the nerves and in the muscles. The cases most liable to be mistaken for multiple neuritis are anterior poliomyelitis, locomotor ataxia, and diffuse central myelitis. The following tables present the differences usually found:

Anterior Poliomyelitis.	Multiple Neuritis.
Sudden onset with fever and development of paralysis in all limbs, followed in from three to five days by subsidence of paralysis, which remains in a few muscles of one limb; or, if two are affected, the paralysis is very rarely symmetrical. If onset is subacute, four weeks is the duration of onset. Muscles not tender.	Fatigue for some weeks, then sudden onset and progress for two weeks with or without fever. Legs first affected, then arms, then body, and paralysis has no tendency to subside for a month; the limbs are affected symmetrically, and the muscles affected are very tender.
Sensory symptoms are rare, and when present soon subside.	Sensory symptoms are constant and severe, and increase; anæsthesia becoming well developed.

Paralysis, atrophy, reaction of degeneration, coldness of the limbs, loss of tendon reflexes, and preservation of bladder control, are common to both diseases.

Locomotor Ataxia.	Multiple Neuritis.
Pains are lightning-like in character.	Paræsthesia is more marked than pain.
Nerves are not tender.	Pain is produced by pressure on nerves.
Ataxia developes slowly and after a period of pain.	Ataxia is an early symptom, develops rapidly, and may exist without pain.
Anæsthesia and analgesia slight.	Anæsthesia in patches total.
No actual paralysis, no atrophy.	Paralysis with atrophy.
No change in electric reactions.	Change in electric reactions.
Argyll-Robertson pupil present.	Pupils react normally.

There are very few symptoms of diffuse myelitis which are not found in cases of neuritis. But cases of diffuse myelitis of the type of Duchenne—paralysie générale spinale subaiguë ascendante—are very rare, and, indeed, by Leyden it

has been affirmed that all such cases are multiple neuritis. Other authorities dispute this assertion and leave us to establish points of difference. These are as follows: In neuritis affections of the functions of micturition and defecation do not occur. Girdle sensation is very rarely mentioned as a symptom; bed-sores and cystitis have not been observed. The advance of the paralysis is rarely from the legs to the thighs and trunk, and then to the arms; it is usually from the legs to the forearms, the trunk and thighs escaping, and, as a rule, the distal portions only of the extremities are paralyzed. If the muscles of the abdomen and respiration are involved, it is only in rapidly fatal cases. In neuritis there is usually some ataxia, and loss of muscular sense is quite evident; while in some, at least, of the cases of myelitis of Duchenne, there were no sensory symptoms at all. Finally, the tenderness of muscles and nerves, and the absence of tenderness to pressure or to heat in the spine, would decide in favor of neuritis. The diagnosis from meningitis of the cord, from tumors or hemorrhages into the cord, or from general paralysis of the insane, would rarely present any difficulty to one who was familiar with the symptoms in those affections and who knew the prominent features of multiple neuritis.

There are certain common types of the disease, a familiarity with which may aid in the diagnosis. If a patient, known to be alcoholic, complains for a time of tremor and muscular weakness, and then is suddenly paralyzed in the legs and arms, develops fever, becomes delirious, and shows great tenderness of the limbs and rapid loss of faradic electric reaction in the muscles, the diagnosis of multiple neuritis can be made. If a patient, who has been taking arsenic medicinally for some time, begins to complain of paræsthesiæ, clumsiness, and fatigue, and soon shows a marked ataxia in hands and feet, with tenderness along the nerves, some atrophy in the weak muscles, and a change in their electric reactions, the diagnosis of multiple neuritis can be made. Lead-palsy is easily recognized. Diphtheritic paralysis is usually limited to the muscles of the pharynx, or of the eyeballs; but the sudden onset of marked ataxia without a preceding stage of pain after diph-

theria, warrants the diagnosis of neuritis. If a tuberculous individual suddenly develops pain and tenderness and weakness in both arms or both legs, and this is followed by paralysis and atrophy in a few symmetrically situated muscles, the diagnosis of multiple neuritis can be made. And lastly, if any one of these clinical pictures is presented by a patient who has been exposed to cold or to wet, or has overstrained himself severely, and a careful examination fails to reveal the characteristic symptoms of general diffuse myelitis, while the limbs are very tender to pressure, multiple neuritis may be suspected.

Cases of *beriberi* may be slight or severe.

There are, first, slight cases, in which the onset is gradual, being usually preceded by a little fever, coryza, and conjunctivitis, which cease when the actual symptoms commence. The patient first notices a weak and heavy feeling in his legs, and finds that he tires so easily that he cannot walk as much as usual. The tired feeling is soon associated with numbness and pain in the legs, and with a slight œdematous swelling. Then, if not before, palpitation of the heart, oppression and weight in the epigastrium, loss of appetite, and general malaise are felt, and the patient finds it necessary to apply for treatment. An examination then shows some diminution of power in the feet and legs, and also in the hands, with loss of tendon reflexes, and much tenderness in the muscles, which show a diminished electric excitability. There is never any ataxia, though the patient sways when his eyes are closed. There is discovered a slight degree of anæsthesia, of irregular distribution, chiefly in the legs and in the radial-nerve region on the forearms. Though the patients look pale, it is usually impossible to find anæmia by examination of the blood. The circulation in the extremities is sluggish. The heart is irregular and rather rapid, and the œdema of the extremities indicates a failure of its power. Dr. Wallace Taylor[13] finds that a sphygmographic tracing is characterized by a sudden high upstroke in ventricular systole, by a precipitous descent from the apex of the percussion-wave, and by dicrotism. Beyond this point these cases, which make up the majority, do not ad-

vance. They usually recover in a few days, or at most a month; although a few become chronic and require several months before the cure is complete.

There are, secondly, severe cases. These may present three different types. There is the atrophic or dry type, in which, after an onset similar in nature to that in the slight cases, but much more rapid, the weakness develops into a true paralysis, associated with marked wasting of the muscles and reaction of degeneration, with great diminution of galvanic excitability. Within a week the patient has to go to bed, and then the paralysis soon spreads from the legs to the arms, and may involve the trunk and even the face. The entire muscular system wastes away till the patient is a mere skeleton. In addition to the motor symptoms there is great sensory disturbance. The suffering from pain, paræsthesiæ, and general muscular tenderness is extreme, and the patient lies totally helpless, and unable to tolerate the lightest touch. The skin may be glossy. There is usually some anæsthesia, but it is never complete, although it may involve the entire body. The temperature sense is seldom affected. Pain may be delayed in transmission. There are no gastric symptoms, and no œdema. Some cases prove fatal from general exhaustion, or intercurrent disease, but the majority recover after a convalescence which lasts a year or more, during which the muscular system is rebuilt.

There is, secondly, the hydropic or wet type. In these cases heart-failure appears early, and is associated with a marked decrease of arterial tension and much œdema of the entire body, effusion into the cavities being added to that beneath the integument. The swelling of the œdematous parts conceals the atrophy which is going on in the muscles, but this is indicated by the paralysis, which is as severe as in the preceding form, and it becomes evident during recovery when the œdema has subsided. I have seen an example of this in the captain of a vessel who came to this city from the Philippine Islands. The disease developed during the voyage.

There is, thirdly, the acute pernicious type. In this all the symptoms of the two former types appear in rapid succes-

sion, and, in addition, gastro-intestinal symptoms and a suppression of urine combine to make the condition an alarming one. Effusions into the pleura and pericardium appear early. The pulse becomes small and irregular, and cyanosis indicates the heart-failure which precedes death.

Prognosis.—The prognosis of multiple neuritis is good, excepting in severe alcoholic cases, and in the very severe forms of beriberi just described. The progress of the recovery may be slow; in some cases two years may elapse before it is complete, but seven months may be considered the average duration, and slight cases may get well in three months.

Treatment.—The majority of the cases being toxic in origin, the most important indication is to remove the cause. Total abstinence from alcohol is necessary for at least two years after recovery to prevent a relapse. Lead and other poisons must be eliminated from the system. Such hygienic measures as may be used to counteract influences of an infectious kind must be employed. The direct treatment of the disease is aided by the spontaneous tendency to recover in a person whose constitution is sound, and whose strength is maintained by general tonic treatment. It is possible to hasten the process of regeneration by the use of full, nutritious, and especially fatty diet, including cream and cod-liver oil; and by the administration of strychnine, phosphorus, iron, and arsenic, no one remedy being continued for more than three weeks at a time. The use of warm and cold douches to the paralyzed limbs, of skillful massage when the tenderness has sufficiently subsided, and of mild (10 ma.) constant stabile galvanic currents along the limbs (see page 182), probably aids the regeneration by stimulating the circulation and increasing metamorphosis of tissue. The daily use of the interrupted galvanic current to the muscles, of sufficient strength to produce contraction in them, gives them proper exercise to maintain their tone (see page 183). Rest is enforced by nature for some time, but moderate exercise is indicated during recovery, but should not be carried to the point of fatigue.

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CHAPTER XV.

PARALYSIS AGITANS.

Analysis of twenty-three cases.—Etiology.—Symptoms.—The varieties of tremor.—Rigidity and contractures.—Gait and its peculiarities.—Treatment.

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TWENTY-THREE cases of Parkinson's disease have been under treatment in the Nervous Department up to January 1st, 1890. Of these 10 were females and 13 were males. This is over one per cent of the whole number of nervous cases recorded (2,070). Ordenstein ("Sur la Paralysie agitante et la Sclérose en plaques généralisée," Paris, 1868) places this disease fifth in order of frequency among nervous disorders, stating also that it is more common than locomotor ataxia—facts which are not borne out by our statistics. Berger noted 37 cases of paralysis agitans among 6,000 patients suffering from diseases of the nervous system, a little over one-half of one per cent.

ETIOLOGY.

Age at Onset.—In the majority of our cases the disorder developed between the ages of fifty and sixty years, next in order between forty and fifty, and in all between thirty and seventy years. Eighteen patients were between forty and sixty years of age at the time of onset, agreeing in this respect with the statistics of all other observers. The periods of life in which the tremor began in this series of cases are classified in the following table:

Age at Onset.	Male.	Female.	Total.
30 to 40 years	2	1	3
40 to 50 "	3	1	4
50 to 60 "	7	7	14
60 to 70 "	1	1	2
Total	13	10	23

Sex.—The fact that men are more frequently affected by this disease than women is borne out by our own figures.

Heredity.—It is a moot question whether heredity plays any particularly important rôle in its development or not. In only two of our own cases could any such factor be suspected. These two cases were brother and sister. In the former it is still mild in its manifestations, while in the sister the disorder has led to irremediable contractures in both hands and both feet and to such general rigidity that locomotion is almost impossible and even speech difficult.

Occupation.—All of the patients were from the common walks of life, but a few of them followed pursuits in which they were especially subjected to exposure to extremes of heat or cold. Thus one was a night-watchman, one a coachman, one a messenger, one an engineer, one an out-of-door laborer, and one had worked as a tobacconist in a damp basement for thirty years.

Exposure to Cold and Wet as a Cause.—In five cases the immediate cause given for the tremor was working in the wet and cold. Two cases, both men, date the onset of the disease from the famous "blizzard" of March 14th, 1888, when New York City was snowed in to such an extent that all travel was suspended, and several people were lost and frozen to death in the streets. These two men were both out in the storm, and the tremor appeared almost immediately after the exposure to the cold and the exertion required to reach their homes.

Worry and Anxiety as Causes.—One of our cases was an illicit distiller of whiskey, and the disease appeared soon after his discovery and trial and the confiscation of all his property.

In 2 cases domestic infelicity was an etiological element. One woman developed it during an anxious period of nursing her dying mother; and another woman during a period of worry over her drunken son.

Traumatic Causes.—In one woman the tremor began in the right arm soon after a fall from a step-ladder. (This patient is the one whose brother is also afflicted with the disease; and the brother has been made much worse by his constant fear and worry, lest he should some time become as helpless as the sister.)

An excellent example of trauma as an exciting cause was that of one man who, at the age of fifty-two, was driving a refractory horse. The horse ran away and threw him out of the vehicle upon his left shoulder. No immediate harm was done, but paralysis agitans soon became manifest, the tremor beginning in the left arm. In such a case as this fright may have also had a share in its production.

Miscellaneous Causes.—In one case fever and ague of three months' duration immediately preceded the development of Parkinson's disease. In another articular rheumatism in the left foot was antecedent to the appearance of tremor in the same extremity.

Among causes given by other authors in their collections of cases are to be mentioned gout, by L'Hirondel (Thèse de Paris, 1883) and typhoid fever, by Berger.

SYMPTOMATOLOGY.

Tremor.—This is one of the cardinal symptoms of paralysis agitans, although, paradoxical as it may seem, quite a number of cases have been recorded of true Parkinson's disease without tremor. Thus Charcot observed 2 such cases, Berger and Wienskowitz 2, Buzzard 1, Hardy 1, Amidon 1 (*N. Y. Med. Record*, November 24th, 1883), and Beevor has very recently descibed 4 (Med. Chir. Soc. Proceed., 1889, Vol. VIII., page 8). The other chief symptom, rigidity, is always present when the tremor is wanting.

Tremor was noted as present in every one of our 23 cases.

The extremities in which it made its first appearance, as related by the patients, are tabulated as follows:

In the right hand	in	10	cases.
" left "	"	6	"
" left foot	"	3	"
In both hands	"	3	"
" both feet	"	1	case.
Total		23	

The extent of the tremor at the time of examination may be gathered from the following table:

Table Showing Extent of Tremor.

In one extremity,	3	cases.
In two extremities,	11	"
In three extremities,	3	"
In four extremities,	6	"
In head as well as limb,	5	"

Charcot's statement that the head never takes part in the tremor, but is only moved by the contiguous shaking of the upper extremities, has been proven to be unfounded in fact.[1]

It will be observed that there was tremor of the head in 5 of our 23 cases, and in all of these it was possible to determine the participation of the cervical musculature in the tremor.

With the exception of the shivering from cold or terror, the tremor of paralysis agitans is almost the only one developed when the body is in a condition of rest. Almost all

[1] The following are some of the authorities who have disproved Charcot's assumption:

OPPOLZER: Spital Zeitung, No. 17, 18, 1861.
CLEMENT: Lyon Médical, No. 26, 1869.
JONES: Brit. Med. Jour., 1873.
WESTPHAL: Charité Annalen, iii. u. iv. Jahrg.
DEMANGE: Revue d. Méd., ii., 1882.
BUZZARD: Clinical Lectures on Dis. of the Nerv. Syst., 1882.
HUBER: Virchow's Arch., vol. cviii., p. 45.
GOWERS (8 out of 37 cases): Dis. of Nerv. Syst., 1888.

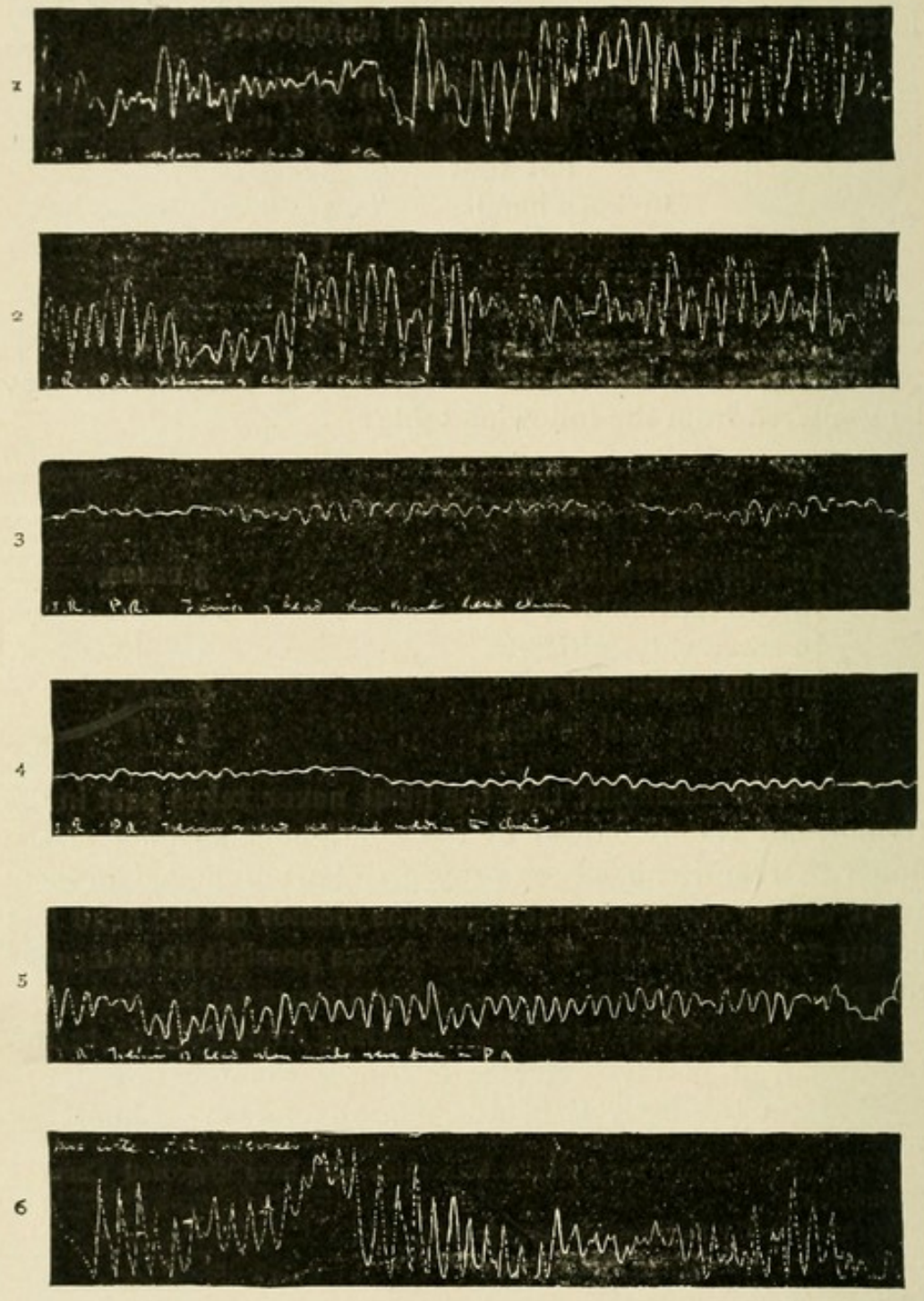

FIG. 67.—Tracing of Tremor of Paralysis Agitans. 1, Tremor of extensors of carpus, right hand, 5.3 per second; 2, tremor of extensors of carpus, right hand, 5.1 per second; 3, Tremor of head while hands held chair, 4.4 per second; 4, Tremor of head while hands held chair, 4.6 per second; 5. tremor of head, no effort with hands, 4.8 per second; 6, Tremor of single fingers, 4.5 per second.

others belong to the class of intention tremors, or to such as are originated by the extension of limbs without support. Furthermore, the tremor varies greatly in extent and rate of rhythm at different times, and even in different parts of the body of the same individual. We noticed in some cases that there was a cessation of the tremor completely for an hour or two daily, or in others great diminution or increase for an indefinite period.

By means of an Edwards sphygmograph, numerous tracings of tremors in various diseases have been taken at the clinic, some of which were made the subject of a short contribution on muscular tremor by myself at the meeting of the American Neurological Association in Washington, September 20th, 1888.[1] I determined the average rate of vibration of this tremor to be from 3.7 to 5.6 per second, thus agreeing with all other investigators (excepting Gowers), as will be noted by glancing at the following table:

Author.	Publication.	Rate per Second.
Marie	Contrib. à l'étude, etc.	5
Charcot	Mal. du Système Nerv.	4–5
Ewald	Berl. klin. Woch., 1883, No. 32	5
Grashey	Arch. für Psych., 1885	4.14–5.34
Huber	Virchow's Arch., Vol. 108, p. 45	3.43–5.57
Gowers	Dis. of the Nerv. Syst., 1888, p. 1001.	4.8–7
Wolfenden & Williams	Brit. Med. Journ., May 19th, 1888	5.1
Peterson	Journ. Nerv. & Ment. Dis., Feb., 1889.	3.7–5.6

It is probable that all tremors are a modification of the rhythmic discharges of energy from the motor ganglion cells, which as is well known occur at the rate of ten per second. Consequently when we have fewer per second, it is because of the fusion of two or three impulses. The dicrotic character of the oscillations in paralysis agitans has been demonstrated by Wolfenden and Williams by means of specially-constructed myographic apparatus.

Illustrations of the tremor of paralysis agitans taken from various portions of the body, and also a series of myograms from different diseases for comparison, are here inserted.

[1] See Journal of Nerv. and Ment. Dis., Feb., 1889.

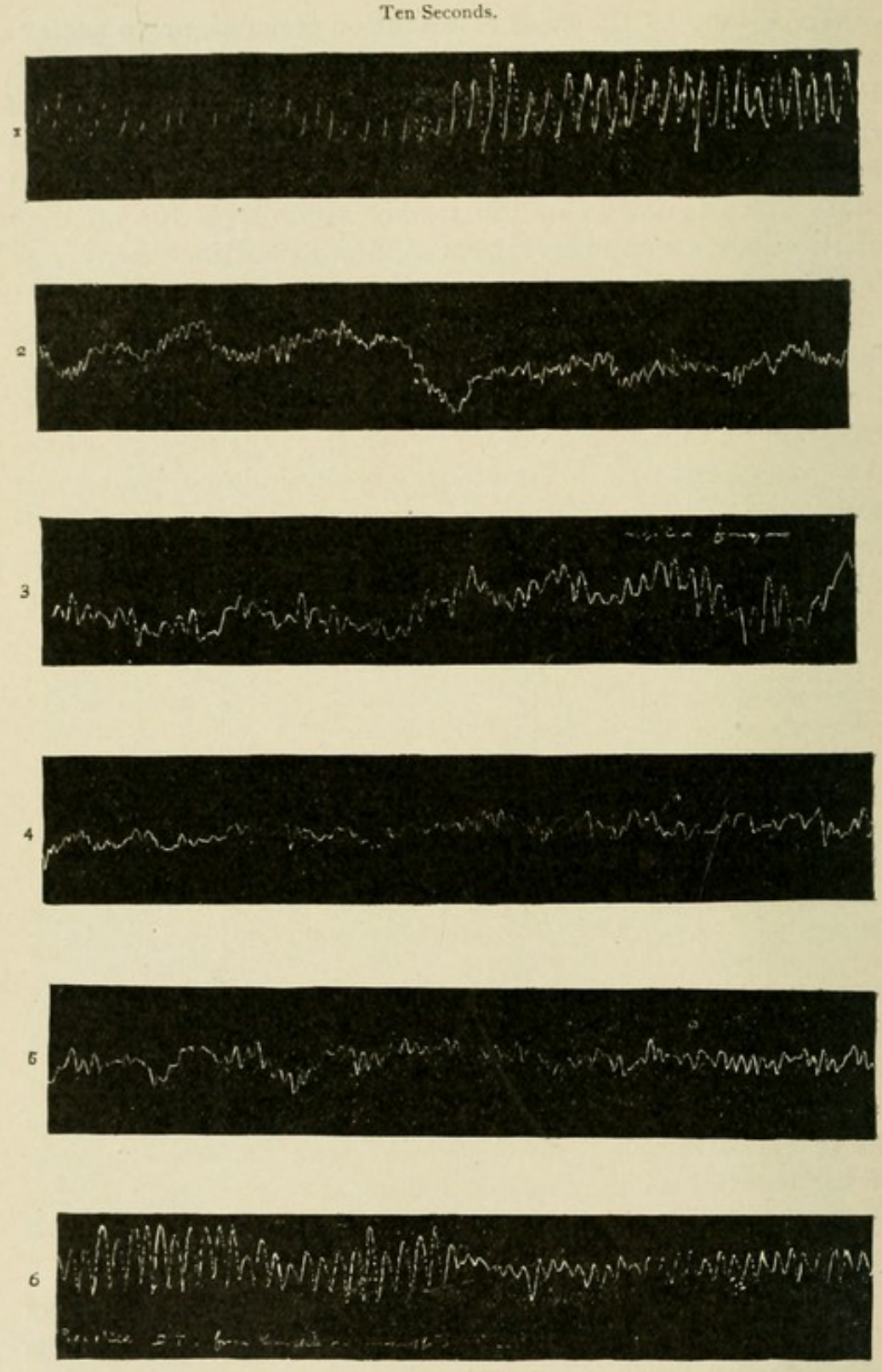

FIG. 68.—A Comparative Series of Myograms of Tremor. 1, Paralysis agitans; 2, Basedow's disease; 3, multiple sclerosis; 4, hysterical tremor; 5, neurasthenic tremor; 6, delirium tremens.

Rigidity.—This cardinal symptom was present in every one of the 23 cases studied, although more marked in some than in others. As is well known, rigor musculorum manifests itself in the extremities, trunk, neck, and face. The muscles of the eyes are extraordinarily seldom affected. Debove has reported cases where there was rigidity of the ocular muscles (*Le Progrès Médical*, 1878). In one of our cases the orbicularis oris was so inflexible that the patient had no control over it and driveled constantly. Rigidity of the lingual musculature was observed in a few cases; and probably a certain amount of stiffness of the muscles concerned in articulation and phonation accounts for peculiarities of speech noted in some.

In one case, a woman aged sixty-five, with a "hemiplegic type" of paralysis agitans affecting only the left side of the body, the rigidity was limited with remarkable precision to the muscles of the left side of the face and neck, left arm and left leg, and even to the left sides of the tongue and orbicularis oris. There was no history or symptom of hemiplegia.

Contractures.—Over eighty per cent of the cases presented the typical position of Parkinson's disease as figured in the text-books. The bowed head, flexed elbows and knees and flexed metacarpo-phalangeal joints are to be looked upon as a species of contracture. The flexors of the elbow were often so contracted that complete extension could not be made, and almost always an attempt to stretch the biceps proved painful.

One case, a woman, aged fifty-seven, was completely helpless with the most advanced degree of contracture that I have ever seen in this disease, with one exception. There was a double talipes-equino-varus and absolute anchylosis of the joints of the ankles and feet, and of all the joints of the hands, of the fingers, and of the wrists, in the characteristic position, but of an extremely exaggerated type. This patient could move her thighs when placed in a standing position, so that she could walk when supported by another; but it was impossible for her to turn in bed, or to rise up from a chair or from a recumbent position.

Propulsion, Retropulsion, Lateropulsion.—Eleven cases presented no peculiarity of gait as evidenced by "running after the centre of gravity." Festination, or propulsion, alone was observed in 6 cases. Both propulsion and retropulsion were of frequent occurrence in 5 cases, and lateropulsion was noted in but 1. Anton Heimann, who reports in detail 19 cases of Parkinson's disease in his exhaustive monograph ("Ueber Paralysis Agitans," Berlin, 1888), noticed also the presence of lateropulsion in but 1 case. A tabular view of these phenomena o locomotion in our 23 cases is here appended:

Form of Movement.	Males.	Females.	Total.
Propulsion only	3	3	6
Both pro- and retro-pulsion	4	1	5
Lateropulsion	1		1
No peculiarity of gait	5	6	11
Total	13	10	23

Tendon Reflexes.—In only 5 cases were the knee, wrist, and elbow jerks exaggerated. In 2 they were hypertypical and in all the rest normal. The exaggeration was not so marked as in cases of organic disease of the cerebro-spinal segment of the motor tract, and indeed no more than is commonly observed in people of advanced age, in whom we ordinarily expect an increase of the deep reflexes. Ankle-clonus was not elicited in any case.

Electrical Changes.—In one case of eight years' standing, in which the disease was limited wholly to the left side, I was enabled to conclusively demonstrate diminished neuro-muscular contractility to faradism upon the affected side. This corroborates Benedikt's statement, who many years ago noted diminution of electrical irritability in the affected extremities of old cases. His further statement that neuro-muscular contractility is markedly increased in such parts in recent cases I had no opportunity to confirm.

The Voice and Speech.—Buzzard in a clinical lecture on shaking palsy (*Brain*, January, 1880) called attention to the

high pitch and piping quality of voice in some cases of the disease, and other authors have mentioned the occasional peculiarity of a sort of halting ejaculation of words.

There were distinctive characteristics of articulation and phonation in no less than 9 of the 23 cases. There is probably no question that these changes depend almost wholly upon a certain amount of rigidity in the muscles concerned in speech and vocalization. The particular features I have noted in the 9 cases are, firstly, a condition of monotonia, as though there were difficulty in adjusting the vocal cords for the purpose of varying the pitch; secondly, a high pitch and piping quality of tone, which may possibly depend upon a certain minute degree of contracture in the crico-thyroid, posterior crico-arytenoid, and internal thyro-arytenoid muscles (a laryngologist might make an interesting study of vocalization in this disease); thirdly, there is often what has been well termed a species of festination in speech. There will be some difficulty in beginning a sentence, a hesitation upon the first word, but that word having been articulated, the patient rapidly repeats the whole sentence if a short one; if it be long he pronounces quickly five or six words, and then stops, apparently to readjust his muscles previous to the ejaculation of another series of words.

Thermal Paræsthesia.—A feeling of excessive heat over the whole body usually, sometimes over limited areas, has been mentioned by various writers as frequent in Parkinson's disease. Charcot found no alteration in temperature in such cases, while a later observer, Berger, claims that there may be marked peripheral increase although the general temperature is normal. This subjective sensation of heat was noted in 5 of our cases, in 4 general, over the whole bodily surface, in 1 limited to the abdominal surface. This last case, a man aged sixty, had such a continual feeling of intense heat over his abdomen that he was constrained to keep constantly lifting his clothing from that surface. I placed an Immisch surface thermometer, carefully covered, upon his abdomen for seven minutes, and an ordinary clinical thermometer under his tongue simultaneously for the same interval of time.

Temperature in the mouth was 98½°, upon the abdominal surface 97°.

At the summer meeting of the French Society for the Progress of Sciences (*Centralb. für Nervenheilk.*, November 15th, 1889), Mossé, of Montpellier, reported his observations upon this condition in 2 cases of the disease. He discovered no actual increase of peripheral temperature. In 1 case the thermal paræsthesia was coincident with broad patches of superficial redness on the back of the hands and inner surfaces of the forearms. He regards the sensation of heat, as well as the exanthema and œdema sometimes observed in shaking palsy, as phenomena due to disturbance in vaso-motor centres.

Paræsthesia of Cold.—This symptom was present in but 2 of the 23 cases. One woman, aged sixty-five, whose tremor began in the left arm, has always had a subjective sensation of cold in that arm A man, aged sixty-eight, who has the disease confined to both arms, complains of a feeling of great cold in these extremities. But in neither case was any particular coldness discoverable upon examination.

Miscellaneous Paræsthesiæ. — Patients often complain of numbness and prickling, sometimes of pains, in the affected extremities. They often speak of the pains as rheumatism. One man had shooting pains in his legs; another a dull aching pain in the three members involved; another numbness in the hands and soles of the feet; another much rheumatoid pain in his two arms, which were the seat of the disease. Two women also suffered from pain in the limbs affected.

Restlessness.—Very many patients have a feeling of general discomfort, making them exceedingly restless, especially at night. It is a species of anxietas tibiarum, only it affects the whole organism. Five of the 23 cases made particular mention of this trying symptom.

Hyperidrosis.—If it is present, this symptom is, as a rule, associated with thermal paræsthesia, and in all likelihood depends upon similar vaso-motor disturbances. Hyperidrosis existed in but one of the series of cases here cited, and in him the perspiration was very profuse and the sensation of heat extreme.

Tachycardia.—Although Marie and Azonlay (*Progrès Méd.*, 1885, No. 49) mention the frequency of this symptom in cases of paralysis agitans, it did not exist in any one of the cases here collected.

Mental State.—Although in many cases diminished intelligence or veritable psychoses have been described in connection with Parkinson's disease, there was no affection of this character in any of our patients.

TREATMENT.

The exact pathology of shaking palsy not yet having been determined, all treatment has thus far been more or less empirical, or symptomatic. Treatment has been particularly directed toward diminishing the tremor and lessening the bodily discomfort. The following list of some of the more important therapeutic agents that have been employed in the disorder has a historic interest as well as pessimistic significance:

Potassium bromide and iodide.
Tincture of veratrum—veratrin (Féris).
Chloride of barium (Brown-Séquard).
Carbonate of Iron (Elliotson).
Strychnine (Trousseau).
Ergotine.
Calabar bean (Ogle).
Chloral hydrate.
Opium.
Morphine (Heimann).
Atropine.
Gelsemium.
Curare.
Hyoscyamine (Charcot).
Hydrobromate of Hyoscine (Peterson and Langdon, *N.Y. Med. Rec.*, September 19th, 1885).
Fowler's solution (Eulenburg).
Arsenite of potash, subcutaneously (Eulenburg).
Coniine (Berger)
Eserine (Riess).

Heimann is an enthusiast as regards the use of morphine in these cases. He says (*loc. cit.*): "It is the only remedy which can for at least a short time make the patient comfortable."

Probably hyoscyamia has been employed more than any other drug in the treatment of these cases at the Vanderbilt Clinic. In at least two patients it has had a decidedly beneficial effect, almost causing the tremor and restlessness to disappear. In one or two patients the results were so bad after its exhibition for a considerable time that the remedy had to be discontinued. We are accustomed to prescribe one $\frac{1}{100}$ grain tablet morning and evening, increasing to thrice daily if desirable, but only in very rare instances giving as much as $\frac{1}{60}$ grain at a dose.

Recognizing the value of opiates as a remedy or antidote for the discomfort, restlessness, anxietas tibiarum, etc., from which these patients so often suffer and by which they are so often made continually miserable, I have lately employed codeia with considerable benefit, especially when combined in the form of a pill with hydrobromate of hyoscine (codeia gr. i. – ij., hyos. hydrobrom. gr. $\frac{1}{100}$) and administered twice or thrice daily; while codeia possesses many of the useful attributes of morphia, it is less deleterious in its influence upon the system.

The use of warm baths of half-hour duration deserves mention, as the best means of allaying the muscular discomfort, weariness and restlessness, of which these patients complain.

CHAPTER XVI.

CHOREA.

Analysis of One Hundred and Twenty-four Cases.—Etiology.—Month of Onset.—Relapses.—Relation to Rheumatism, Endocarditis, and Malaria.—Duration of Attacks.—Treatment.

By WALTER VOUGHT, M.D.,
First Assistant to the Nervous Clinic.

THE data for this chapter have been obtained from 75 cases of chorea seeking treatment at the Vanderbilt Clinic from the time of its opening in February, 1888, to January 1st, 1890, together with the records of 49 additional cases which Professor Starr has kindly placed at my disposal.

The whole number of cases of nervous diseases under observation during this time was 2,070, so that the proportion of cases of chorea to all nervous diseases would be as 1 to 28.

Sex.—Of the 124 cases 36 were males and 88 females; the proportion being as 1 to 2.16.

Age		Age		Age	
Five years	3	Thirteen years	14	Twenty years	4
Six "	5	Fourteen "	9	Twenty-eight years	2
Eight "	16	Fifteen "	8	Thirty-two "	1
Nine "	6	Sixteen "	4	Thirty-five "	1
Ten "	9	Seventeen "	7	Thirty-seven "	1
Eleven "	14	Eighteen "	5	Forty-nine "	1
Twelve "	9	Nineteen "	2	Fifty-four "	1
Age not stated	2				

From the above table it is seen that the greatest number of cases occurred between the ages of eight and fourteen years, and that after fourteen years of age there is a gradual decrease until twenty, after which but few cases are found and these are apt to be chronic cases.

Extent.—In 76 cases, more than fifty per cent, the disease

was general, and of these the right side was more affected than the left in 5, and in 3 cases the opposite of this obtained. Twenty cases, or sixteen and one-eighth per cent, were limited to the left side alone and thirteen, or ten and two-fifths per cent, to the right side. Four cases were of the "habit chorea," 1 of them limited to the shoulders, 1 to the shoulders and head, 1 to the head, and 1 to the face. In 11 cases the extent is not stated.

Number of the attack.—Of the 124 cases, 46, *i.e.*, thirty-seven per cent, were first attacks and 54, *i.e.*, forty-three and five-tenths per cent, were relapses; of these 54, 30, *i.e.*, twenty-four and one-fifth per cent of the whole number of cases, were second attacks; 6 were third, 5 were fourth, 4 were fifth attacks, 1 was the sixth, 1 the seventh attack and 4 patients are recorded as having had several preceding attacks, 1 was the eighth, 1 the ninth attack, 2 cases were chronic, and in 24 cases the number of the attack is not stated.

Frequency of Relapses.—Examination regarding the frequency shows that 1 case had nine attacks, 1 case seven attacks, 2 cases four attacks and 2 cases three attacks in a corresponding number of years. One case had five attacks in fifteen years and on each occasion the affection was limited to one side of the body; in 1 case there were four attacks in fourteen years, and in 1 case exacerbations occurred twice yearly for five years.

Month of Onset.—In 74 cases where it was possible to accurately determine the month of onset, it was found that 11 each began in September and October, or nearly thirty per cent, and in the months of April, May, and June, 21 cases, or more than twenty-eight per cent. By reference to the chart it will be seen that, beginning with February, the frequency rises steadily to May, then falls to July. and again steadily rises to September, remaining up through October, when it suddenly drops to remain down through December, rising slightly to January when it again falls to February. The curve of the months of applying for treatment will be found on the same chart and undergoes almost the same variations as the months of onset (See Fig. 69).

An interesting fact was presented by 3 cases; 1 of them had had nine attacks in as many years and each attack began in September, 1 had 3 attacks in as many years, each beginning in February, and the third had 4 attacks in as many years, each beginning in the spring of the year. Dana's[1] observations correspond closely to these, he finding, from 130 cases, that the disease was most frequent in the spring months, next to these in the fall months, then in the winter and least frequent in summer. When the different statements of different authors on this point are examined, it seems probable that the disease

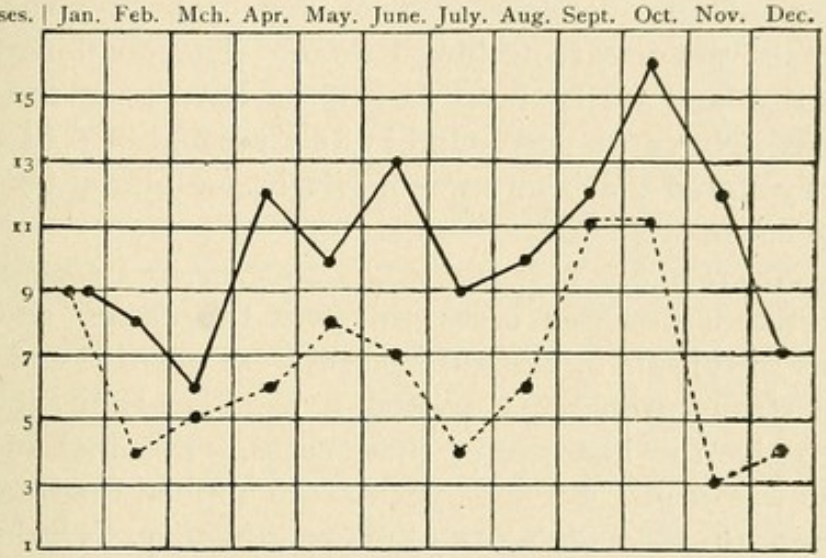

FIG. 69.—Diagram to show Relation of the Development of Chorea to Months. Line = applicd for treatment; dotted line = disease began.

does not occur everywhere with equal frequency during the same seasons of the year. Thus Lewis,[2] of Philadelphia, found that March had the highest number and October and November the lowest. Koch[3] found that most of his cases occurred in December. Gowers[4] says, "in this country (England) the influence of season does not seem to be great." Meigs and Pepper,[5] quoting Gerhardt and Weir Mitchell, say that they have observed that chorea occurs more frequently and in a more severe form in the spring than at any other season and that relapses of the disease are most apt to take place at that time.

[1] N. Y. Med. Jour., Apr. 1st, 1889.
[2] Polyclinic, Jan., 1887.
[3] Deut. Arch. Klin. Med., Bd. xl., Heft 5–6.
[4] Dis. of the Nerv. Syst., vol. ii., p. 549.
[5] Dis. of Children, 7th Ed., p. 611.

From the above we may conclude that there exists a marked tendency for the disease to relapse; that this is more marked during the first year after the first attack, and that with each following year the liability to a return diminishes progressively. In addition to the tendency to relapse, there is also shown a tendency to relapse at certain times of the year.

Etiology.—Rheumatism was present or had previously existed in 21 cases, being assigned as the direct cause in 10. Fright was the cause in 14 cases, overstudy at school in 2, over-excitement, overwork, measles, and pregnancy each in 1. In 2 cases the disease followed a fall. The combination of rheumatism and scarlet fever is said to have been the cause in 1 case. No cause was found in 81 cases and in 6 cases it is distinctly stated that there was no fright. Malarial poisoning was the cause in 3 cases.

The Relation between Malarial Poisoning and Chorea.—As above stated, in three cases this was the cause, and inasmuch as the proofs in 2 of these cases were positive and in the third a strong probability existed, it has been thought worth while to give the histories of these cases. The first of these patients was a girl æt. nine years, who applied for treatment October 12th, 1889, giving the following history. During two weeks in August preceding she had been at College Point, Long Island; while there she was in her usual health; soon after she began to complain of general pains, headache, poor appetite and to have some fever in the afternoons, and she lost her previous good color. Two weeks before applying for treatment she had been much frightened by burglars in the house, and the muscular twitchings began a few days afterward. She had never had chorea or rheumatism. Examination showed a well-marked chorea, the spleen enlarged, and an anæmic bruit at the base of the heart, and examination of the blood showed the presence of the crescentic and pigmented intracellular bodies characteristic of malarial poisoning. She was put upon arsenic and quinine and on January 20th, nine weeks after, was completely well.

The second patient was a lad æt. ten, who had had an attack

five years previously. He had never had rheumatism. His trouble began three weeks before application for treatment. Examination showed marked chorea. The patient was unable to feed himself. There was heard over the cardiac apex a soft blowing systolic murmur, no cardiac hypertrophy. Examination of the blood showed the presence of a few pigmented intra-cellular bodies. The patient was treated with Fowler's solution, and was completely well in four weeks.

The third patient was a lad æt. eleven, who four years previously had had a similar attack lasting three weeks. Present attack began one week ago. No fright; never had rheumatism. Physical examination was negative. Put on Fowler's solution for two weeks; at the end of that time, the patient being no better, sulphate of quinine three grains, three times a day were given. He was completely well at the end of three weeks, improvement being marked at the end of one week.

The attention of the writer was called to the possibility of a relation existing between malarial poisoning and chorea, by Dr. A. B. Pope of the Children's Department of the Clinic, in the summer of 1889, he having observed that some patients would recover in a short time when treated with quinine, other lines of treatment having been tried in vain. The blood from six different patients was examined with negative result, but in all of these treatment had been begun before the examination. The seventh patient examined was the first above cited; she was examined at the time of first application for treatment, as were the second and third patients. Since this time other patients have been similarly examined but with negative results, examination being made at the time of application for treatment. It would be desirable to continue the examination of the blood from cases of chorea to see what connection, if any, exists between malarial poisoning and it, it being the writer's belief that some relation exists between the two diseases in this city; whether it be any more than that malarial poisoning acts as a predisposing cause is at present difficult to say. It is the hope of the writer that others may follow out this line of observation.

Eichhorst[1] says that the disease may follow intermittent fever.

Weir Mitchell[2] says: "There is, however, no evidence to favor the view that chorea can have a malarial origin except that it arises in spring with as much certainty as ague."

That malarial poisoning gives rise to neuralgias and headaches is acknowledged. In the blood from a case of beriberi where well-marked neuritis was present the writer found the malarial organism, and in a case of multiple neuritis with distinct history of chronic malarial exposure the examination of the blood showed the presence of the malarial organism.

The depreciated physical condition which patients are often brought to from prolonged malarial poisoning certainly predisposes to the development of chorea. Chorea is not as common in the negro as in the white. Mitchell says that the weight of evidence is in favor of the opinion that the black is less liable to chorea than the white, and the infrequency of malarial poisoning in the negro is well known. It is in the spring and fall that we see the most manifestations of malarial poisoning, and in the fall months especially do we find most of the cases of chronic malarial poisoning. Of 74 cases of chorea, where the beginning of the disease could be accurately determined, 8 began in May and 11 each in September and October. We know that one attack of malarial fever predisposes to other attacks; the liability of chorea to relapse at certain times of the year has been spoken of.

The treatment of malarial poisoning by the administration of arsenic is the one that meets with the most success, likewise this is the standard treatment of chorea.

Relation to Rheumatism.—The possibility of chorea being the result of some infectious material in the system has been put forward by the following authors, Joffroy,[3] Leube,[4] Eulenburg,[5] Prior,[6] Strümpell.[7] One writer, Koch (l. c.), considers

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[1] Pathologie und Therapie, Aufl. 3, Bd. 3.

[2] Dis. of Nerv. Syst., 1881, p. 141. [3] Le Progrès Médical, 1885, No. 22.

[4] Deutsches Archiv für klin. Med., Bd. xxv., S. 242.

[5] Real Encyc., iv., 269. [6] Berl. klin. Woch., 1886, No. 2.

[7] Lehrb. d. Inn. Krankh., Bd. ii., S. 441.

that "a definite cause is demanded for chorea with its characteristic and constant train of symptoms, its typical course, and necessarily well-defined localized anatomical seat." That a relation exists between rheumatism and chorea and between endocarditis and chorea writers on this subject are at present generally in accord, and the most convincing proofs of this are given by Osler,[1] who found that of 110 cases of chorea re-examined at periods varying from two years to sixteen years after the attack for which they had sought treatment, 54 cases presented signs of valvular disease; that in 22 cases, forty and seven-tenths per cent, there was a distinct history of articular trouble sometimes with the chorea, but in 6 cases, from one to five years after the attacks. The larger proportion, fifty-nine and three-tenths per cent, of the cases were without any history of rheumatic trouble. Of the 21 cases which had had three or more attacks of chorea, only 7 had had rheumatism.

In the author's cases endocarditis was present in 12 and existed without rheumatism in 6, and dependent on it or at least coexisting with a distinct history of it in 5. In 1 case, examined one year after an attack, endocarditis had developed.

Koch having previously considered the relation existing between chorea, polyarthritis, and endocarditis says: "Thus we see how chorea, polyarthritis, and endocarditis may occur in varying order. The primary affection has in the vast majority of the cases its seat in the nervous system, less often in the joints, and least of all in the endocardium. It is caused by the characteristic choreaic virus, but often indeed by that of polyarthritis closely allied to it, and perhaps in isolated cases other infectious germs may give rise to a like train of symptoms."

When we consider that an injury may give rise to tuberculosis, exposure to cold and wet to rheumatism, and to a new attack of intermittent fever (in one predisposed to it), as the writer himself has not infrequently seen, why is it not in perfect accord with the theory of an infectious disease that the nervous system being a locus minoris resistentiæ may, by a

[1] Am. Jour. Med. Sci., vol. xciv., p. 372.

sudden psychic effect, as fright or a fall, or by more gradual ones as over-study, over-work, or systemic weakening from pregnancy, become the seat of invasion of an infectious material or predisposed to its development?

Complications.—Acute articular rheumatism developed in 4 cases during the existence of the attack of chorea.

Endocarditis existed in 12 cases and in 6 of these it was independent of rheumatism, in 5 cases a rheumatic history was found, and in the other case no mention was made either of the presence or absence of this disease. The mitral valve was affected in 6 cases, and in the other 6 no mention is made on this point. In 93 cases the positive[1] absence of endocarditis is noted and in the remaining 19 no mention is made.

Hæmic or functional murmurs were present in 10 cases.

One case examined one year after the cure of a first attack had in the interval developed chronic endocarditis.

Melancholia coexisted in 1 case, speech was markedly affected in 2 cases, and 2 patients were feeble minded; night terrors were present in 2 cases, irritability of temper was present in several, and 1 case was of such severity that there was complete inability to perform any voluntary motion, the patient being confined to bed unable to move or to speak.

Electrical Reaction.—In 5 cases of hemi-chorea examined, there was an increased irritability of the muscles of the affected side in response to the faradic current. In another case no difference could be detected.

THE DURATION OF THE DISEASE AND ITS TERMINATION.

The Duration of the Disease in Cases Cured.

2 weeks,	. . . . .	in	1 case.
4 "	. . . . .	"	13 cases.
6 "	. . . . .	"	4 "
8 "	. . . . .	"	4 "
12 "	. . . . .	"	4 "
16 "	. . . . .	"	4 "
20 "	. . . . .	"	1 case.
Unknown,	. . . . .	"	2 cases.

[1] This is, of course, as far as diagnostic means will permit.

Cases Improved.

in 3 weeks,	. . . . .	2 cases.
in 4 "	. . . . .	11 "
in 6 "	. . . . .	1 case.
in 12 "	. . . . .	2 cases.

Three cases were unimproved after one, five, and twenty-one months respectively.

In 1 case where the disease had lasted for one year, cure was affected in two months.

Four cases of adult chorea occurring in patients of thirty-two, thirty-seven, forty-nine, and fifty-four years of age respectively, and lasting from five months to four years before application for treatment was made, were not materially benefited.

Three cases of chronic chorea occurring in patients of thirteen, fourteen, and seventeen years of age and existing four, five, and two years respectively, were also not benefited by treatment, but it seems probable that the inability to attend regularly, bad hygienic surroundings, and the early discouragement which dispensary patients show if they do not improve rapidly, was a large factor in the failure of a cure or improvement.

TREATMENT.

In all cases the patient's general health has been attended to, the withdrawal of all bad influences and surroundings secured, and the correction of any errors of refraction in the eye made. Rest of body and mind as far as possible, and the administration of baths and good food; the administration of arsenic and chloral; and the use of quinine and iron for the anæmia are the therapeutic measures employed in all the cases. Arsenic is given in as large doses as the patients can bear, beginning with a small dose and increasing gradually. When symptoms arise from its administration, cessation for a few days and beginning at the dose left off has been the routine treatment. One of the greatest difficulties to contend against in dispensary practice is the withdrawal of the patient from school. This is in many cases a very difficult thing to

accomplish, parents not appreciating the value of this as a therapeutic measure. Parents are in all cases warned of the liability to relapse and are requested to bring their children for examination at the slightest symptoms of any return of the disease.

PARAMYOCLONUS MULTIPLEX, AND ITS DIAGNOSIS FROM CHOREA. WITH A REPORT OF A CASE.

By DR. STARR.

Paramyoclonus multiplex is a spasmodic affection of the muscular system of peculiar character, distribution, and course, dependent upon irritation of the nervous motor mechanisms, and somewhat resembling chorea, hence it is appended to this chapter.

The chief features of this disease are illustrated by the following case:

John D., aged thirty-three, of Kingston, Canada, a grocer, of good family history, but of nervous temperament, was in his usual health until September 15th, 1886, when he strained his back and right shoulder by lifting a box, sixty pounds in weight, while in an awkward position. The pain, below the right shoulder blade, resulting from this strain was so severe that he went at once to his physician, Dr. H. J. Saunders, in whose office he had a peculiar attack. He began to cry and scream with pain and soon felt a choking sensation and was unable to get his breath; then followed convulsive movements of the body and legs, the latter being drawn up and thrust out forcibly. These lasted an hour, after which he was taken home. They returned at short intervals, for three days, being attended by pain in the back; and then they began to involve the muscles of the upper extremities and neck; and a few twitching movements of the face also occurred. The spasms were always very rapid, but were chiefly confined to muscles attached to the trunk, it being noticed from the first that the muscles of the forearms and hands and of the legs and feet did not take part in the spasms. The diaphragm was affected

usually, so that dyspnœa and exhaustion attended the attack, and it was occasionally followed by vomiting, when occurring after a meal. He had at first ten or twelve such attacks in twenty-four hours, each lasting nearly an hour. After two or three weeks, the attacks became less severe, the legs being less violently moved, and their duration decreased. But they had continued until May 2d, 1887, when I saw him, varying in severity and duration, but constantly diminishing, so that then he was having but three or four a day, though sometimes they were more frequent. He had several attacks in my presence, each lasting from one to five minutes, and leaving him much exhausted. The muscles first affected were those of the back and abdomen, a series of quick, alternate contractions of the dorsal muscles and of the recti abdominis, resulting in a rapid protrusion and retraction of the abdomen. As this became more rapid and forcible, the body and head were thrown backward and forward, without any spasm of the muscles of the neck, and in one attack which occurred while he was standing, similar complementary movements to preserve his balance produced alternate slight flexion and extension of the hips. In a much more severe attack which was observed when he was stripped, the muscles involved were first those of the back and abdomen already mentioned; then the neck muscles, so that the head was not only nodded, but was turned from side to side; then the muscles of the upper arm, the pectorals, deltoid, biceps, and triceps, being all in action, and lastly, the muscles of the thigh, the quadriceps femoris, biceps, and semitendinosus and semi-membranosus, with the glutei, acting with such force as to cause movements of both hip and knee-joints. The contractions of these muscles were rapid, the rate rising to ninety per minute. The spasm in the arms was not severe enough to move the shoulder or elbow joints, but the muscles named were seen to contract and raise the skin. He said that the wrist and fingers, ankles and toes, had never participated in the spasm. He had noticed that formerly there was some movement in the forearms, probably of the supinator longus muscles, but this I did not see. There was also, in a severe attack, a spasm of the diaphragm resulting in a long inspira-

tion, accompanied by a sound, and while this was tonic, rapid movements of the intercostal and accessory respiratory muscles to supply the lack of inspiratory action of the diaphragm were made. Only at the very first had there been seen any spasm of the facial muscles, and for some months these had not been affected.

The spasms were so severe as to make me fear that he would fall while standing, as he said he had done several times, and would nearly throw him out of an arm chair when sitting. They came on suddenly, but were usually preceded by a peculiar sensation which ascended from the legs to the head, and ceased as suddenly, leaving him in a state of considerable exhaustion, panting and perspiring, and looking badly as if about to faint. During the interval between the spasms an occasional fibrillary twitching in the muscles of the back and pectorals was observed, but not elsewhere. He could not stop the spasm voluntarily or in any way limit its course. Nor could he start it voluntarily. But any exposure of the skin to cold, any irritation of the skin by electricity, any tapping of the tendons at the knee or attempt to elicit ankle clonus was sufficient to start a spasm at once. He said it often came on after muscular effort, such as a long walk. It was also more likely to occur after mental excitement, and it usually came on when he began to talk about his condition or went to see a stranger. From the beginning of the affection, pressure on the right shoulder blade, where he had the pain, produced a spasm at once. It ceased during his sleep and never woke him by occurring at night. He thought that a drink of whiskey arrested it sometimes.

His motor power, sensory perception, and voluntary coordination were not in any way impaired. His skin and tendon reflexes were exaggerated, a marked ankle clonus being present on both sides. His muscles responded normally to both electrical currents. Mentally, he was perfectly clear, but somewhat excitable, and after each attack in his exhausted condition, tears came to his eyes as he spoke of his trouble. He did not appear, however, to be of an hysterical temperament. Treatment had been somewhat successful, as he con-

sidered his condition much better than it had been six months ago, for he said that the attacks were less severe, shorter, and less frequent. Dr. Saunders reported his cure four months after I saw him.

This spasmodic affection, limited to the muscles of the body and proximal portion of the limbs and only occasionally affecting the neck and face, has been termed by Friedreich, who described it first, as paramyoclonus multiplex. Since its first description, a number of cases have been reported by Lowenfeld, Marie, Silvestrini, Bechterew, Seeligmüller, Homen, Remak, Fry, and Vanlair. It must be regarded as a distinct disease, since it is different from chorea, from hysterical spasms, from epileptiform convulsions, from convulsive tremor or tetanilla, and from tic convulsif.

Its causation is uncertain. In 3 cases fright, in 1 case severe hemorrhage, which may have caused fright, in 1 case the shock of a cold bath, and in my own case an overstrain causing pain have preceded the development of the spasm. In Fry's case over-exertion was the cause. Vanlair thinks it may be due to severe peripheral irritation. In 2 of the cases, a chronic spasmodic affection had been present for several years. It has been observed twice in a female, the remaining cases being males. The age of the individual has little to do with its development, for persons of all ages from ten to fifty-two have been affected. Its symptoms are quite characteristic. The spasms are bilateral and symmetrical. They are limited to certain muscles. In 8 cases, the quadriceps femoris and flexors of the leg, and the so-called "upper arm group of muscles," the deltoid, biceps, and supinator longus, were affected. In 7 cases, the muscles of the back were involved. In 6 cases, the muscles of the neck contracted; in 5 cases, the glutei. In 4 cases, the face and the diaphragm were involved. In no case have the muscles of the hands or forearms, of the feet or legs been affected. The usual limitation of the spasm to the body muscles with those of the thighs and arms is very noticeable.

The character of the spasm is also characteristic. It is a rapidly repeated clonic spasm occurring at intervals. In 6

cases, the rate of contraction has been counted. It has varied from 50 per minute to 180 per minute. In my own case, it was about 90. It is not a sudden, single, irregular muscular contraction, like that of chorea, but appears to be always bilateral and to involve several muscles of a physiological group at once, thus resulting in a series of movements, any one of which can be voluntarily made. In several cases, a tonic contraction has occurred in one or more of the muscles affected, before or during the clonic seizure. In my own case, the spasm of the diaphragm was tonic for one-eighth to one-quarter of a minute during each attack, and in the early attacks, the spasms of the back were tonic for some seconds. The clonic contractions continue, when once set up, for a varying time, from half a minute to ten minutes, and are succeeded by a complete interval of freedom from spasm. In my own case, this interval had varied from half an hour to about one week. And the fact that the free quiet intervals were getting longer had encouraged him to hope for a recovery. During the spasm itself, the resulting movements were of a very violent nature. The head was thrown about by the movements of the body, rendering the patient dizzy. The body was tossed about in the chair, so that there seemed to be danger of his being thrown out upon the floor. If the spasm occurred while he was walking, he was quite liable to be thrown down, and had hurt himself several times. But this violence is not always present—for in two cases, the spasms were never severe enough to cause a movement of the joints, and were only observed when the patient was stripped—being then of the nature of a fascicular twitching. In my own case, such a fascicular muscular twitching was occasionally seen during the intervals in the muscles of the back and the pectorals.

In the majority of the cases, any tapping of the tendons or any irritation of the skin was sufficient to produce a spasm. This seems to be an important point, for I am not aware that it has been observed in hysterical or choreic spasms. It is true that, in hysterical cases, certain zones or areas can occasionally be found on the body, irritation of which may cause

or may arrest the attack. But in this condition the spasm is produced by irritation anywhere on the skin—or by tapping the tendons at the knee and ankle—and was not associated with disturbances of sensation, which are characteristic of hysterical zones. The knee-jerk has been increased in 4 cases, was less in 1 case, and was not tested in the remainder. The skin reflexes were also increased in 4 cases, and are mentioned as normal in but one of the remainder. Mental excitement seems to have predisposed to the onset of the spasm in 3 cases. Had the disease been hysterical in nature, this would probably have been observed in a larger proportion. Voluntary effort stopped the spasm in 4 cases and made it worse in 3 cases. Had the disease been hysterical, volition would probably not have influenced it favorably in the majority of cases. The spasm has ceased during sleep in 4 cases, but has continued in 1 case.

In none of the cases have consciousness, motion, sensation, co-ordination, or electric excitability been in any way affected —an important negative fact, since it proves at once that the condition is a functional neurosis, and makes it very unlikely that it is of an epileptic or an hysterical nature. In one case, which died of phthisis, a careful exmination by Prof. Schultze, of Heidelberg, failed to reveal any lesion of the nervous system.

It is evident from this review of the symptomatology that the characteristics of the disease are quite distinct; that it can be differentiated from chorea, from hysteria, and from epilepsy.

These characteristics may be summed up as follows: Paramyoclonus multiplex is a spasmodic affection of the muscular system, occurring bilaterally in symmetrically situated muscles attached at one or both ends to the trunk, and in muscles whose function is associated with these, consisting of a series of violent clonic spasms of considerable rapidity and severity, occurring only at intervals; and associated with fascicular tremors of the affected muscles, persisting during the interval between the spasms. It occurs after some mental or physical strain, and is not accompanied by any disturbance of sensory or motor functions, excepting by an increase of the superficial

and deep reflexes. It can be excited by irritation of the skin or tendons.

In regard to the prognosis, it may be said that this is favorable. The majority of the cases have recovered quite rapidly under treatment. In 2 cases, however, relapses have occurred.

The treatment which has been of most service has been the application of strong galvanic currents to the spine and neck, and the application of the anode to sensitive points in case these exist. Under this treatment Fry's case recovered in three weeks. Many nerve sedatives have been used, and also nerve tonics. The exact effect of these seems to be doubtful. In my own case, sedatives, tonics, and electrical applications had all been equally futile to arrest the attacks, but the patient had improved to a considerable degree under the varied treatment. I prescribed galvanism to the spine, arsenic and chloral with some beneficial effect. Hammond has advised the hypodermatic use of arsenic.

It is useless to discuss the nature of the disease from so few cases as are at our disposal. It has been regarded as a functional neurosis, and to this all must agree, both on account of the absence of any lesion, in one case examined by the most competent neuro-pathologist in Germany, and on account of the absence of symptoms of organic disease and the recovery of the cases. Whether it has a central origin and is produced by a hyper-excitability of the brain or spinal cord, induced by the sudden vaso-motor spasm accompanying fright or mental or physical strain, as Friedreich believed, or whether it may be a reflex spasm due to some peripheral irritation which, being conveyed to the spinal and medullary centres, produces the spasm reflexly, as another author has suggested, remains for the future to decide. The case here reported would seem to favor the latter view.

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CHAPTER XVII.

EPILEPSY.

A classification of epileptics.—The causes of the disease.—The frequency of attacks.—The aura.—Psychical epileptic equivalents.—Reflex neuroses of an epileptiform type and their diagnosis.—The treatment of epilepsy.

EPILEPSY is such a common disease, two persons in every thousand being epileptics, that every fact which can be established regarding its causation, its phenomena, its nature, or its treatment is of importance. The study of a large number of cases enables the physician to divide epileptics into certain groups—such as those suffering from convulsive attacks (grand mal), and those suffering from slight attacks without movements (petit mal); or, those in whom the attack is preceded by an aura, and those in whom no aura occurs; or those in whom an organic nervous lesion, or a source of peripheral irritation, or no ascertainable cause can be found. Such attempts at classification are found to lead to the conclusion that epilepsy is a protean affection, presenting very different features in different groups of cases. The study of these groups results in a division of all epileptics into three classes —the first, in which the disease is distinctly cortical in origin; the second, in which the disease is medullary in origin; the third in which it is reflex in origin.

In *the first class of cases* the epileptic attack presents many features which resemble those found in one or other of the forms of Jacksonian epilepsy already studied (Chapter IV.). The attack begins with a conscious sensation, either a sensation of motion, or of tingling and numbness, or of light or color, or of sound or smell or taste, or some visceral feeling such as nausea, suffocation, palpitation, etc., etc. Such spontaneous conscious sensations imply necessarily, as we have

already seen, cortical irritation. Or the attack begins with peculiar abnormal states of consciousness which will be described further on as the psychical epileptic equivalent. The attack goes on to a more or less general series of movements. These may be merely a twitching of the eyes, smacking the lips, irregular balancing motions of the body to prevent a fall, or awkward movements of the hands; such as occur in many light attacks. They may on the other hand present all the features of a severe general convulsion, with a cry, a biting of the tongue, and enormous and powerful motions in every muscle of the body. In Jacksonian epilepsy, as we have seen, the spasm may be limited to one limb, or may invade an adjacent part, or may become unilateral, or may finally become general. In Jacksonian epilepsy the rate of extension is slow. In this first class of epileptics the extension of the movements is very much more rapid, but it is found that there is such an extension and that the attack, as a rule, does not begin, as in the second class of cases, at once in all the parts. The attack is succeeded by a condition of deep sleep or of great mental confusion. All the functions of the cortex appear to be for a time suspended. The patient is unable to think clearly, to give any account of himself, or to realize what has occurred. In Jacksonian epilepsy the spasm is succeeded by paralysis, the hallucinations of vision by blindness, the tingling by anæsthesia, the movement by ataxia. Thus in both Jacksonian epilepsy and in this first class of epileptic cases there is evidence of severe involuntary cortical irritation, beginning in one region and extending to others, and followed by a suspension of cortical activity for a time. The analogy between the phenomena is very striking. It seems reasonable to conclude that since Jacksonian epilepsy is known to be due to irritation in the cortex, in the first class of epileptic cases the seat of the disease is in the cortex of the brain. In this first class of epileptics are included, therefore, all cases of petit-mal and of grand-mal with an aura, and all cases of so-called psychical epileptic equivalent, soon to be considered, as well as all cases of epilepsy from traumatic affections of the cortex and from organic cortical disease.

In *the second class of cases* the epileptic attack presents a different series of phenomena. There is no conscious warning. There is a sudden loss of consciousness only (petit-mal) or a sudden general convulsion with loss of consciousness. Following the attack there is, in some cases, the same condition of confusion and coma which occurs in the first class, but in other cases this is absent. Such attacks resemble the attack with which a gross lesion in the pons Varolii and medulla oblongata usually begins. Furthermore, such attacks can be experimentally produced by irritation of these parts of the brain in animals, as Nothnagel showed in 1872. Epileptic attacks have been long ascribed to a discharge of nervous energy from the medulla, or to vaso-motor spasm whose starting-point is supposed to be from the medulla. The arguments urged by Nothnagel[1] and recently restated by Ziehen[2] in support of this view have much strength and afford a satisfactory explanation for this second class of epileptic cases. They do not explain the first or third classes of cases at all. In this class are included all cases without aura, commonly called genuine idiopathic epilepsy.

In *the third class of cases*, which is very small in comparison with either of the other two classes, the origin of the epileptic attack is found in a source of peripheral irritation. It has long been known that an injury to any part, especially if that injury causes a division or compression of a nerve trunk, may be followed by epileptic attacks, in a person previously free from epilepsy. It is sometimes noticed in these cases, that a sensation in the scar precedes the attack, or that pressure on the scar will bring an attack on. It has long been known that, in nervous little children, persistent irritation of the genital organs, such as may be due to phimosis, to concretions beneath the prepuce, or to foreign bodies in the vagina, or such as may be produced by masturbation, gives rise to attacks of an epileptiform kind. It has long been known that in some women the function of menstruation is uniformly attended by seizures of a nervous character resembling epi-

[1] Nothnagel: Ziemssen's Cyclo. of Med., Amer. Edit., vol. xvi., pp. 185 to 221.

[2] Ziehen: Arch. f. Psych., 1890, vol. xxi., p. 863.

lepsy. It has long been known that, in nervous children, gastro-intestinal irritation may bring on convulsions. It has recently been argued that irritation arising from the eye, chiefly from imperfect muscular action and consequent unconscious ocular effort,[1] and also that irritation produced in the naso-pharyngeal cavity may cause nervous attacks of an epileptiform nature. The attacks occurring in this class of cases differ from the attacks in the other two classes in the fact of the great frequency of their occurrence from the outset, in the fact that between the attacks more or less subjective discomfort or pain, or objective signs, attract attention to the irritated part, and in the fact that removal of the source of irritation is followed by very prompt cessation of the attacks. In all such cases the attack may be explained by supposing that a long-continued irritation of moderate degree has a cumulative effect upon the gray matter constituting primary sensory centres in the spinal cord or brain axis, that this state of irritation extends to the medulla oblongata, which finally takes on a condition of unstable equilibrium, such as is present in the second class of epileptic cases. The medulla is then ready to set free a sudden nervous discharge upon any sudden peripheral irritation. This relieves its strain. After the discharge, the cumulative action again begins. The continued peripheral irritation thus produces occasional epileptiform attacks. In the third class of cases, therefore, are included all the cases in which a source of peripheral irritation can be found whose removal is followed by cure.

It will be observed that in discussing these classes of cases the character of the convulsion and the condition of consciousness during it have not been particularly emphasized. This is because, in all three classes, the various phenomena characterizing epileptic attacks may present themselves, so that, from a consideration of the attack at its maximum of intensity, no distinction between these classes of cases can be reached. When, however, single cases are carefully studied in all their details, it is found that characteristic symptoms,

[1] G. T. Stephens, M.D.: "Functional Nervous Diseases," 1888. Wigglesworth, Brain, No. xlv.

additional to the mere fit, enable the observer to assign the case to its proper category. And the importance of such a classification of cases will be appreciated when the subject of treatment is discussed.

The statements in this chapter are based upon the records of 167 cases of epilepsy, of which 94 were observed at the Vanderbilt Clinic from February, 1888, to January, 1890; 32 at the Polyclinic between 1885 and 1888; and the remainder in my private practice.

ETIOLOGY.

There were 84 males and 83 females. The following table shows the age of the patient at the onset of the attacks.

Age of Onset.

Birth to age of 5 years,	29 cases.
Age of 5 to age of 10 years,	22 "
" 10 " " 15 "	30 "
" 15 " " 20 "	16 "
" 20 " " 30 "	30 "
" 30 " " 40 "	9 "
" 40 " " 50 "	8 "
" 50 " " 65 "	3 "
Age unknown,	18 "

It thus appears, that nearly sixty per cent began before the age of twenty, and that the development of the disease after the age of thirty is rare—thirteen per cent.

Convulsions had occurred in infancy in 30 cases in addition to the 29 in which the disease began before the age of 5; a fact which demonstrates that infantile convulsions are the most common of all the predisposing causes of this disease—or, to state it in a different way, that a congenital irritable nervous system predisposes the child to the occurrence of convulsions from any slight cause and may be the underlying foundation for the development of the disease. This is true in one-third of the cases. Convulsions had occurred in some very near ancestor in 16 cases. An injury to the head by a

blow or a fall had occurred in 11 cases. Among other causes assigned by the patients were fright, worry, great anxiety, sunstroke, alcoholism, scarlet-fever, measles, "brain-fever," masturbation, poisoning by copper, uterine and ovarian disease, and syphilis. When convulsions occurred as a symptom of syphilitic disease of the brain or its membranes, the case was not classed among the epileptics in the table of cases, and in the few cases in which syphilis was ascribed as a cause, no other symptoms of syphilis were present. In 7 of the female patients the attacks were distinctly menstrual in their occurrence, and had been so from their beginning. In 4 cases a source of peripheral irritation was found, once in the eye, once in the nose, twice in the genital organs.

In the large majority of the cases, careful questioning failed to elicit any ascertainable cause for the disease.

CHARACTER AND FREQUENCY OF ATTACKS.

In a large majority of the cases the interval between the first and second attacks was a long one, from two years to four months; the interval between the second and third attacks was shorter, from one year to three months, and the greatest frequency of attacks was only attained after the disease had been established for several years.

In a few cases epileptiform convulsions began with great frequency, several occurring in a day and a number within the first two weeks. In all of these cases some distinct cause was ascertained. This was either irritation of the brain by hemorrhage, meningitis, or from an injury to the skull; or irritation of some internal organ, as constipation, chronic gastro-intestinal irritation, genital irritation, nasal irritation, or defects of vision. Nine of the patients had attacks of petit-mal only. Forty had both petit-mal and grand-mal. One hundred and nine had grand-mal only. In the large majority of the cases the disease had been in existence several years at the time the patients were seen. The greatest frequency with which attacks of grand-mal occurred is shown in the following table.

Table Showing Greatest Frequency of Epileptic Attacks.

1 in 1 year or over,	9 cases.
1 " 6 months,	2 "
1 " 3 "	6 "
1 " 2 "	10 "
1 " 1 month,	25 "
1 " 2 weeks,	22 "
1 " 1 week,	7 "
1 per week, less than 1 per day,	28 "
Over one attack daily,	28 "
Frequency not recorded,	30 "

It becomes evident that in less than twenty per cent of the recorded cases did the attacks exceed one a day. In these cases such frequency was by no means permanent. It had occurred at some time in the course of the disease and therefore was recorded. But it may be said to be unusual in epileptics under treatment—for in but 6 of these cases did the attacks fail to be reduced by treatment to less than one a day—and in many they were reduced to one a week or less. Thus in numerous cases the record shows a reduction of daily attacks to one in two or three weeks; of weekly attacks to one in three months; of monthly attacks to one in six months. From a study of these cases it would seem that about two per cent of epileptics continue to have very frequent fits in spite of medicinal treatment. In these cases evidences of marked cerebral disease—shown by mental deterioration—was usually found. And it may be noted incidentally that in the cases classed under mal-development of the brain, in many of which epilepsy was present (but which are not included in this list), the attacks were very frequent in their occurrence. It would, therefore, appear that very great frequency of attacks in epilepsy, persisting permanently, may be taken as evidence of organic cerebral changes.

In all the cases in which attacks of petit-mal were present the number of these attacks was much greater than those of grand-mal. Usually several such attacks were occurring daily when the patients applied.

The patients were always questioned regarding the possibility of attacks occurring during the night. The result was

Nocturnal attacks only in,	22 cases.
Nocturnal attacks as well as diurnal in,	17 "

It is evident, therefore, that nocturnal attacks are comparatively rare, being present in but twenty-five per cent of the cases—a fact which may be explained by the exemption of the nervous system from external sources of irritation during sleep.

Procursive attacks, that is, an attack which consists of or begins with rapid running either forward or in a circle, avoiding obstacles, and not falling, occurred in 2 cases only —one of these a case already recorded (p. 124) as an illustration of a tendency to turn to one side in walking.

THE AURA IN EPILEPSY.

In 65 cases the attack was preceded by a conscious sensation which was so constant in its occurrence and was so definitely remembered, as to be considered by the patient a positive warning of the recurrence of an attack.

In each patient the aura was always the same—a variety of auræ in the same patient not having been observed.

The character and relative frequency of the auræ were as follows:

1. Epigastric sensations, which the patients likened to the feeling produced by a voluntary forcible contraction of the abdominal muscles, or to a ball rising from the stomach, occurred in 19 cases. This was the aura most frequently observed.

2. Visual sensations, of light, or sparks, or a blue color, or a red color, or a purple color, occurred in 14 cases. One patient saw a coffin before her as the attack began, another saw a face, another several faces advancing toward her.

3. Tactile sensations, in the form of tingling or numbness in the body or limbs occurred in 10 cases. One patient complained of a sudden sense of cold or chilliness all over her

prior to the attack. Another of a coldness in the feet, which ascended to the trunk before she lost consciousness. Three patients could not describe the sensation felt, but were conscious of an indefinite sensation coming over the entire body. Several perceived the convulsive movements of the head or arms before losing consciousness.

4. Vertigo was complained of as an aura in 7 cases.

5. A sensation of fear without known cause occurred in 8 cases.

6. Palpitation of the heart, a sensation of suffocation, a drowsiness, heaviness of the eyelids, a sudden inability to speak, were mentioned by different individuals as auræ. And in other cases the psychical auræ soon to be discussed were present.

In 47 cases the existence of an aura was distinctly denied. In the remainder, 55 cases, no record of an aura was made, but as the aura was inquired into as a matter of routine, it is probable that in these cases none was felt. It may be stated that in the majority of epileptics there occurs no warning whatever of the occurrence of the attack.

In view of the recently developed theory[1] that the beginning of the nervous "discharge" in an epileptic attack is frequently in the cerebral cortex, the study of the aura becomes interesting. It resembles in an epileptic fit the "signal symptom" occurring in Jacksonian epilepsy as the beginning of a local spasm. And since the "signal symptom" is of great value in determining the point of beginning cortical irritation, it is not unlikely that the aura in epilepsy may have a similar significance. A visual aura might indicate that the nervous discharge began in the occipital lobes; a theory supported by the fact that in three patients the aura of light was always seen to one side of the middle line (compare also p. 52); a vertigo might indicate that it began in the cerebellum; a sensation of numbness or of movement that it began in the sensory-motor area of the cortex. And in view of the fact that in localized spasm excision of the cortex at the seat of irritation has, in a few cases, resulted in recovery, the practical value

[1] Gowers: "Diseases of the Nervous System," vol. ii., p. 697.

of the study of the aura becomes evident. It is not impossible that patients may appreciate a previously unnoticed aura if their attention be directed to it. I have found this to be so in a few cases. And it is evident that the existence of such varieties of auræ is in itself an argument of no little strength, that the nervous discharge in an epileptic attack is not of uniform nature nor of uniform situation.

It is well known that "*intellectual auræ*" have been described by Hughlings Jackson,[1] and attacks which he has called a "dreamy state." He thinks that they afford evidence of the origin of a nervous discharge from the highest cerebral centres. In one of the cases at the Polyclinic, the patient told of certain "queer thoughts" which he refused to disclose, always coming into his mind before an attack. A private patient of my own, a professor of mathematics, has as an invariable aura, the sudden coming into consciousness of an algebraic formula which arrests his attention no matter what he is thinking of.

THE PSYCHICAL EPILEPTIC EQUIVALENT.

In some cases of epilepsy, there is evidence of the existence of a state of consciousness, not only differing from the normal consciousness, but also differing from the state of unconsciousness usually occurring in an epileptic attack. These attacks are termed psychical epileptic equivalents. Those who find the essence of an epileptic attack in the loss of consciousness, find difficulty in assigning such attacks to a proper place in their definition of the disease. Those who regard the whole epileptic phenomena as evidence of sudden involuntary nervous discharge from the cortex, see in these cases examples of discharge from the highest cerebral centres. To explain a psychical epileptic equivalent it is necessary to suppose a discharge in the areas which preside over the higher conscious actions of the mind. This may give rise to involuntary mental processes sufficiently distinct to arrest and

[1] Hughlings Jackson: On a particular variety of Epilepsy, "Intellectual Auræ." Brain, 1888, vol. xi., p. 179. See also Chas. Mercier: "The Nervous System and the Mind," 1888, chap. i., "The Nervous Discharge."

hold the attention of the individual. Any of these discharges may be so light as not to extend to adjacent brain areas, in which case it is a single idea which comes to mind. But if they are severe and the irritation extends to neighboring areas, the mental impression may rise to such a height that maniacal acts are performed; the thought goes on to a confusion of many ideas and a loss of consciousness. The analogy between the cases of cortical disease already considered and some of these cases of epilepsy is evident. The following cases illustrate these phenomena.

CASE XXXII.—PSYCHICAL EPILEPTIC PHENOMENA.

A gentleman in middle life, of rather nervous disposition, and mentally an active man, was attacked at the age of forty-two with grand-mal; and during the past three years since the first attack has had four severe nocturnal general convulsions at intervals of fourteen months, ten months, two months, and eight months. After each convulsion he is acutely maniacal for about three hours, during which time he has to be restrained from injuring himself and others, and from doing much damage. The mania is succeeded by a deep sleep from which he wakes without any memory of what has occurred since the onset of unconsciousness with which the convulsion begins. (Such maniacal attacks are not very uncommon, and occasionally it is the case that the attack of mania occurs in place of a convulsion rather than succeeding it. It is then termed the psychical equivalent of a convulsion.) The interesting feature of this case is, however, the development during the past year of peculiar attacks, which occur almost daily, of the following kind. While the patient is talking, reading, walking, or at his professional work, he will suddenly cease what he is about, and his face will assume a broad grin for about ten seconds, then he looks about in an absent-minded way, and immediately resumes his occupation. At these times there is no apparent loss of consciousness; he never staggers if standing, nor lets anything drop which he is holding, but when questioned as to what has amused him he has

no recollection of having been amused, or any notion that anything unusual has occurred. These attacks are undoubtedly of the nature of petit-mal, but the fact of the change of mental expression seems to indicate that when they occur there is present a second state of consciousness, in which the patient perceives something funny, just as in the maniacal condition there is a second state of consciousness in which the mind is excited by frightful perceptions or notions. The recollection of neither of these second states remains. While the severe attacks are now under control by the bromides, the minor attacks are not at all affected by treatment.

CASE XXXIII.—PSYCHICAL EPILEPTIC EQUIVALENT. DOUBLE CONSCIOUSNESS.

A young man of fair intelligence and good physique was first seized with an epileptic convulsion after a severe fall, in which he hit his head, five years ago. Since that time he has had numerous attacks of grand-mal at irregular intervals.

He was put under treatment by bromides in July, 1884, and up to January 10th, 1885, had had no return of the convulsions. On January 10th he came home from work as usual, ate his supper, and went to bed. He slept with his brother, who is sure that during the night he had no convulsion. The patient says that when he woke up on the 11th of January, he found himself in Bellevue hospital and learned to his surprise that it was evening. He has no recollection of anything which occurred between going to bed on Friday night and waking in the hospital on Saturday night. From his family, however, it was ascertained that he got up as usual on Saturday morning, and while it was noticed that he acted a little strangely at breakfast nothing was said to him and he went as usual to work. His employer thought that his eyes looked brighter (possibly his pupils were dilated) and that he did not appear natural, but he took no special notice of this and soon after his arrival at the shop in 24th St. he sent him up to a house in 46th Street (about a mile) on an errand, to obtain a carpenter's bit and brace. He went up to 46th St., did the

errand, and evidently explained himself intelligibly for he was given the bit and brace. The next trace he has of his movements was at 8th Avenue near Bleecker St. (about two miles away), though how he got there he does not know. He there went into a plumber's shop, and asked to be allowed to sit down and rest. He had nothing in his hands, so must have lost the bit and brace on the way. He soon got into a lively talk with the plumber, and became quarrelsome, so that he was told to go out. He went away, but in an hour came back, entered the shop, and tried to strike the plumber. This was for him an unusual proceeding, as he is of a mild and gentle disposition. An officer was summoned, who took him to the police station, where it was evident that he was out of his head; so they sent him to St. Vincent's Hospital, whence he was at once transferred to Bellevue. He fell asleep soon after admission to Bellevue, and on waking in an hour or so was surprised to find where he was. His manner of talking made it at once evident that nothing was the matter with him, and he was discharged at once and went home, rather indignant at having been sent to a hospital and anxious to find out the reason. Being a very intelligent lad he became interested in his own case at once, on learning a few of the facts, and ascertained by inquiry the various occurrences which have just been described. Soon after this I lost track of him and do not know whether any similar attacks have since occurred.

The case must be classified among cases of double consciousness, or psychical epileptic equivalent. The development of this state is known to be most frequent among epileptics. It is of interest to notice that three states of consciousness followed one another in this patient; first the normal state, second, the abnormal state, third, the normal state. In the second abnormal state he had a memory of things which occurred in the first state, for he went to work as usual; and must have had a recollection of his usual habits, of his place of business, etc. In the third state he had a recollection of what had occurred in the first state, but not of the events of the second or abnormal state. A certain degree of consciousness and continuity of recollection were, therefore, continu-

ous from the first state into the second, and from the first state into the third. The absolute break lay between the second state of abnormal consciousness and the third state of normal consciousness. I have called it a state of abnormal consciousness, and there is evidence that he acted during it in a rather unnatural manner. That the entire abnormal state was an epileptoid phenomenon is only an assumption based on the lack of memory of its occurrences, and the existence of epilepsy in the patient. It gives no explanation of the peculiar occurrence at all, unless the theory of a discharge and subsequent period of feeble activity in the higher cerebral centres be accepted.

CASE XXXIV.—PSYCHICAL EPILEPTIC EQUIVALENT.

A small messenger boy came to my dispensary class one day in considerable genuine distress. He said that on two occasions while out on errands in the city, he had suddenly lost his way, and had become so completely bewildered that he had to get a policeman to take him home. He was perfectly familiar with New York, had always lived here, knew the streets and buildings well, and never, except on these two occasions had had the slightest difficulty in finding his way about. But at both these times it seemed to him as if he were in a perfectly strange city, and he had no idea in which direction, to turn. There was no loss of consciousness, and he had never had a convulsion. He was such an honest-looking little fellow that I could not doubt his story, and further inquiry proved it to be true. Similar cases have been recorded by a French authority which had occurred in epileptics, and I suppose that this boy had on each of these occasions a peculiar attack of a psychical character—an epileptic equivalent with subsequent loss of memory.

CASE XXXV.—PSYCHICAL EPILEPTIC EQUIVALENT.

A teacher of considerable eminence consulted me some time ago about a peculiar phenomenon which troubled him greatly. On several occasions, which had recently been in-

creasing in frequency, he found his attention arrested, no matter what he was doing, by a strange and unfamiliar set of ideas which suddenly came up to his mind. He was obliged to think of these at once, and to keep his attention upon them about as long as it took him to walk 200 feet, when they would as suddenly fade out of consciousness, leaving no memory of their contents. He was sure that there was no cessation of consciousness at any time, though he admitted that he could not go on talking, or notice a question, or do anything during the attack, but follow the idea presented to the mind. And though he has no recollection of the idea after it is gone, he recognizes the same idea as occurring again in the next attack. So that now, as soon as this peculiar notion of which he is unable to give an account comes into his thought, he fumbles in his pockets for his bottle of amyl nitrite and usually succeeds in uncorking it and inhaling the drug, thus arresting the attack. Frequently the attacks are succeeded by a headache which lasts a day. Bromides decrease the frequency of their occurrence. Mental work increases their frequency markedly. The sudden occurrence of an attack of a definite character, the inability to carry on a normal mental process during the attack, and the total lack of memory of the forcible idea which presents itself in the attack, seem to indicate a powerful involuntary cerebral action, of such intensity as to command attention, and so completely unrelated to the normal train of thought as to leave behind it no associations in memory by means of which it can be reached by consciousness. I should consider this a nervous discharge in the higher cerebral centres presiding over mental action, corresponding in kind to the nervous discharge in the motor centres which give rise to monospasm. It is a sort of psychical epileptic phenomenon, and the irritation probably has its location in the frontal lobes, since speech is suspended during its progress.

Such cases, then, offer a strong argument for the possibility of "nervous discharges" from those parts of the cortex whose activity is synchronous with mental processes. Cases with an aura of sensation indicate a discharge from the sensory

areas of the cortex. Cases with a conscious spasm of limited extent, indicate a discharge from the motor areas of the cortex. Cases with conscious visceral sensations indicate a discharge from those parts of the cortex presiding over the internal organs, the location of which is not yet known. It thus becomes evident that the study of the aura in every epileptic case is of the greatest importance. For it may indicate the exact seat of the disease, organic, functional, or nutritive, not only in the cortex but in some definite cortical area. It also will enable cases of epilepsy of cortical origin to be differentiated from so-called genuine idiopathic epilepsy.

THE DIFFERENTIAL DIAGNOSIS BETWEEN EPILEPSY AND EPILEPTIFORM ATTACKS DUE TO PERIPHERAL IRRITATION.

In some cases of supposed epilepsy the removal of some source of peripheral irritation is followed by complete recovery. The assertion has, therefore, been made that peripheral irritation may produce true epilepsy. This I believe to be erroneous, since the careful study of the two classes of cases shows that a differential diagnosis between epilepsy and convulsive attacks of reflex origin is possible. This diagnosis can in some cases be made by a study of the attack. In a reflex case, the attack is often attended by an incomplete disturbance of consciousness, the patient knowing what is going on during the attack and being able to tell, when the attack is over, what has been said or done to him during its progress. This is illustrated in the second case here appended. In epilepsy, as a rule, the loss of consciousness is complete and the patient has no memory of anything between the onset and his recovery.

There are some cases of reflex neurosis, however, in which the diagnosis cannot be made from the nature of the attack. It is truly epileptiform in character. In these cases much may be learned if the mode of onset of the disease, the frequency of attacks at the outset, and the concomitant symptoms are studied. In a reflex case the onset usually occurs at a time when, from some general constitutional disease, the nervous system is in a state of irritable weakness. Girls who

are anæmic, or are just developing, and boys who are suffering from the exhaustion of sexual abuse or from malarial poisoning, are particularly susceptible. The frequency of the attacks of whatever kind is very great from the very beginning. Thus in a case of reflex neurosis from genital irritation, seen with Dr. O'Dwyer in a boy of three, within a week of the onset, the attacks had reached the number of eight or ten daily, and within three weeks the number had risen to sixty a day. The attacks were of a petit-mal type. He would stop playing, turn pale, stand with eyes fixed and pupils dilated, unconscious but not falling, for about thirty seconds, and then recover consciousness and look about in a frightened manner and cry a little, and then go on playing. This case was cured by circumcision. In another case, due to irritation in the pharynx, the attacks of great mental irritation, almost maniacal, occurred several times daily from the outset. This case was cured by excision of the pharyngeal tonsil. And in the first case here appended, due to eye strain, the attacks occurred daily from the outset. This great frequency of attacks at the onset does not occur in idiopathic epilepsy. The interval between the first and second fits is often one or two years, usually over three months, rarely a month. The intervals then become shorter, but I have never known a patient with genuine idiopathic epilepsy[1] to have a fit daily within a year of the onset. In the 167 cases of epilepsy, less than twenty per cent had attacks daily, and these were all cases in which the disease had been present for several years. In none had the interval between the first and second fit been less than a month.

Lastly, in reflex neuroses, there are usually other symptoms which indicate the seat of the peripheral irritation. If that irritation is in a scar, there is pain or numbness in it, and pressure on it may cause an attack. If the irritation arises from the genitals, there is evident irritability and undue redness of the organs, tendency to handle them, especially during an attack, difficulty of micturition, and evident phimosis, as in the second case appended, or vaginal irritation. If the

[1] By this term I mean to include the second class of cases.

irritation is from the uterus or ovaries, symptoms of disease of these organs other than the attacks will be discovered and tenderness in the region will be found. In this list of cases are two of genuine epilepsy, from whom the ovaries had been removed without any effect on the frequency of attacks. If the irritation be from the digestive tract, indigestion, constipation, and flatulence will be complained of. If the irritation arises from the respiratory tract, a careful examination will probably be made because the imperfect nasal breathing or nasal voice, or the existence of catarrh, will call attention to these parts. And if the irritation is from eye strain the patient will complain of headache, frontal or occipital, aching in the nape of the neck, or discomfort about the eyes after using them for near or far objects as the case may be. Impacted wax will cause a cough, or tinnitus or deafness. Thus, in any case of peripheral irritation of sufficient severity to produce a reflex neurosis, nature will indicate the source of the trouble by discomfort or abnormal sensations in the part which is the seat of irritation.

In genuine epilepsy, on the other hand, the patient usually feels perfectly well between the attacks and presents no symptoms of nervous irritation.

Since, in every patient suffering from epilepsy, the nervous centres are always ready to discharge their reserve energy upon slight external excitement, it is a matter of common necessity to remove all possible causes of serious peripheral irritation. Such causes must necessarily be looked for and prevented. In a true epilepsy, this may decrease the frequency of the attacks to some extent. In a reflex neurosis, recovery immediately follows the removal of the exciting cause.

The following cases which were seen at the Clinic are good examples of reflex neuroses. I may add that, in my experience, reflex neuroses of an epileptiform type are exceedingly rare.[1] I have records of 6 cases only in a series of 3,500 cases of nervous diseases.

[1] For a confirmation of the statement that reflex neuroses from eye strain are infrequent, the reader is referred to an exhaustive article on this subject by D. B. St. John Roosa, Med. Record, April 19th, 1890.

CASE XXXVI.—REFLEX NEUROSIS FROM EYE STRAIN. EPILEPTIFORM ATTACK.

A boy of twelve began to have attacks in September, 1888, of the following kind, without known cause. He feels a numbness and tingling in the right hand which ascends the arm, then he clasps his hands, utters a cry, and loses consciousness, has a general convulsion of moderate intensity and short duration "two to three minutes" (?) without biting his tongue or frothing at the mouth, after which he is sleepy for an hour. From the beginning he had had at least one attack daily, usually at night, and within two months of the onset he was having several daily as well as several at night. No intestinal or genital irritation was discovered, but his eyes troubled him after reading, and an ocular examination showed marked hypermetropia and astigmatism, and hyperæmia of the left optic disc. His attacks had been in no way influenced by the use of nitroglycerine or by moderate doses of bromides; but after proper glasses were given, the attacks ceased at once, and a year subsequently he reported that they had not returned.

CASE XXXVII.—REFLEX EPILEPTIFORM ATTACKS FROM GENITAL IRRITATION.

The boy, now aged nine, has had attacks since the age of five. He has no aura, but while at play suddenly straightens his body, becomes rigid, does not fall, twitches in all his muscles slightly, laughs in a silly manner, seems dazed, but will make a random reply to questions at any time during an attack, and when it passes off he goes to sleep at once and sleeps for several hours. These attacks have been very frequent from the outset; he had several in the first week, and had six attacks the day before I saw him. He always has an attack soon after getting into bed at night, which attracts attention because he laughs aloud. The attacks have diminished slightly under bromides. An examination led to the suspicion of masturbation, which he freely admitted, having

discovered the possibility of orgasm and never having spoken of it to any one. His penis was large, the prepuce tight so that it could not be retracted, the meatus was quite red, and the examination produced erection at once. He was told to stop the habit and was referred to his physician, with a recommendation that circumcision be done; this was followed by complete cessation of the attacks at once.

THE TREATMENT OF EPILEPSY.

In regard to the treatment of epilepsy, it may be stated at once that when causal factors are removed so far as possible, and all sources of irritation in the various organs are eliminated, the chief reliance in the treatment must be in the use of the bromides. A combination of the bromides of potash, soda, and ammonia, to which an alkaline potash salt is added, with a small amount of arsenic, is the form used at the Clinic. This is given largely diluted in a tumbler full of water, either before meals or two hours after eating. In cases of nocturnal epilepsy, it is given at bed time only and in these cases belladonna (Tincture ♏ v.-x.) or sulphate of atropia in full dose ($\frac{1}{120}$ gr.) is added. In each case the amount of medicine given is regulated wholly by its effect upon the attacks, each patient keeping a register, and his daily or weekly amount being varied in accordance with his condition. The daily amount given at the outset is one drachm of the mixed bromides and this is increased or decreased as the case goes on. If proper attention is paid to the condition of the bowels and of the skin and of the kidneys, there appears to be little danger of the occurrence of objectionable symptoms of brominism.

The effect of bromide upon the occurrence of attacks is in many cases very marked. This is true of the first and second classes of epileptics, but less of the third class. In several cases the number of attacks was reduced from one in a month to two in a year. In one case monthly attacks ceased and the patient has been nine months without an attack. In several cases weekly attacks have been reduced to one in two

months. In one case weekly attacks were reduced to one attack in six months. The addition of chloral to the bromide mixture was found very serviceable in many cases. The most favorable result was in a boy aged 16, who had had convulsions since infancy and who was having three or four fits daily when put upon treatment. There was a progressive diminution in the attacks upon the use of one drachm daily of bromides, so that at the end of ten months his record showed that he had been ten weeks for several periods without an attack. In this case twenty grains of chloral was given daily in addition to the drachm of bromide. There was little or no mental depression produced, and for the past six months he had been able to work in a store, and his improvement has been maintained for two years.[1]

There are some cases in which bromides do not appear to have any marked effect. This is often true of those cases in which it produces severe gastro-intestinal catarrh This was observed in five per cent of the cases. In these cases it appears to be useless to continue it if its combination with other remedies has no effect. Other remedies, such as borax, iodide of zinc, valerianate of zinc, antifebrine, sulphonal, and nitroglycerine have been tried in these cases without any success, and the conclusion is reached that they are incurable. One patient, in whom a history of a fall was obtained and a scar on the scalp and a groove in the bone over the parietal region were found, was trephined by Dr. McBurney without any permanent result, although for two weeks after the operation his fits ceased; they then returned with the former frequency, four daily. It is to be noticed that in this case there was no aura. The addition of digitalis or strophanthus to the bromide is made when there is a weakened heart or when the use of bromide produces an indefinite sensation of fear, a symptom not infrequently observed.

During the past two years a number of new remedies have been tested with some care at the Clinic, and in private prac-

[1] The lectures of Seguin on the Treatment of Epilepsy (N. Y. Med. Jour., March and April, 1890) contain most complete and valuable suggestions on this subject.

tice. Tincture of simulo was tried in 14 cases during three months, severe cases being selected in which daily or weekly attacks were present. The remedy showed some tendency to reduce the number of attacks of grand-mal, but had no effect whatever upon petit-mal. Thus in one case daily attacks of grand-mal were reduced to two per week; in another case attacks occurring every two weeks were reduced to one in two months; in another, monthly attacks were reduced to one in three months; thus the effect of the remedy was shown to be a decided one. In two cases, in which the attacks always occurred at the menses only, simulo had no effect, though used in daily half-ounce dose for two weeks at a time, before the attacks were due. In four cases it failed to have any perceptible effect.

The combination of simulo with the bromides was found to be more efficacious than either drug alone in four cases—but it failed in one. Its high price prevents its routine use in dispensary practice. Antifebrine has been tried in a number of cases without any favorable result. It does not seem to have any power of influencing the frequency of attacks.

Every patient who has an aura is furnished with a small bottle containing cotton wool saturated with amyl nitrite, and is instructed to inhale the remedy as soon as the aura is perceived. In about one-quarter of the cases it aborts the attack; such cases are also often improved by the use of nitroglycerine $\frac{1}{100}$ gr. t. i. d., or of codeine $\frac{1}{2}$ gr., t. i. d. given in addition to the bromide and chloral mixture.

The treatment of petit-mal is less satisfactory than that of grand-mal. The only remedy of any service is nitroglycerine. In one case, a male, the success of the drug has been so marked as to warrant mention. Daily attacks have ceased and for six months he has had no attacks at all. In a number of cases it has produced about the same relative diminution in the number of attacks which has been produced by bromides in the attacks of grand-mal. In many cases, however, it fails to affect the disease, and as no rule can yet be laid down as to the character of the cases in which it is successful, a trial is always made of it in every new case

Should the further study of epileptic cases establish the classification here proposed, and should further experience in the surgical treatment of Jacksonian epilepsy show it to be efficient, it is not impossible that surgical treatment of the first class of cases of epilepsy may become possible. It is, however, not yet time to urge its trial.

CHAPTER XVIII.

SOME PAINFUL FUNCTIONAL AFFECTIONS AND THEIR TREATMENT.

Trigeminal Neuralgia.—Tic Convulsif.—Reflex or transferred pains.—Headaches, their varieties and treatment.

I. TRIGEMINAL NEURALGIA.

Of the 151 cases of trigeminal neuralgia seen in two years at the Vanderbilt Clinic, the large majority were of malarial origin and yielded to treatment by a single mercurial purgative, followed by Warburg's tincture or quinine, the latter drugs being given in three full doses at intervals of an hour, beginning six hours before the attack was due. Others due to chronic malarial poisoning were found to yield to arsenic, with tonics and cod-liver oil. A considerable number not malarial have been successfully treated by aconitia, beginning with $\frac{1}{200}$ gr. twice daily and increasing daily one $\frac{1}{200}$ gr. until the physiological effect of tingling of the extremities, face, and tongue and a chilly feeling in the back was reached. This dose was then reduced slightly and kept up for some days, usually with favorable results, the condition of the heart being watched. The combination of drugs included in the neuralgic tablet (page 326) has also been of great service. In three cases, one of infra-orbital and two of dental neuralgia, surgical interference was found necessary. These were referred to Dr. Hartley and were operated upon by him in Roosevelt Hospital. In the first case the infra-orbital nerve was reached by a curved incision following the natural line in the face below the eye; a portion, a centimetre in length, was exsected, with immediate relief which has continued for over two years. In the second case the inferior dental nerve was reached by trephining the ramus of the lower jaw, the superficial inci-

sion being along its posterior border. In this case also the relief of the neuralgia has been permanent for two years.

In the third case the inferior dental nerve was reached through the mouth, the mucous membrane being anæsthetized with cocaine and the operation being done without ether, as the patient was an old man with feeble heart and atheromatous arteries. In this case the relief from pain has been continued for three months, and at present he has no return of the symptoms.

It is interesting to find that, although all the patients complain of tingling and numbness in the area of distribution of the divided nerve, and all have a considerable degree of anæsthesia in this area, this anæsthesia is not total in any case —a fact which demonstrates that the anastomosing network of nerve fibres under the skin suffices to convey impressions from the domain of one nerve trunk into that of another. This fact has been utilized in the treatment of several cases of traumatic neuritis in the arm. For it has been found that by stroking the surface with a fine faradic brush from the anæsthetic area over into the sensitive area, sensation gradually returns in the borders of the anæsthetic area, so that by the application the area of anæsthesia may be decidedly diminished before the union and regeneration of the divided or injured nerve in the arm has been complete.

II. TIC CONVULSIF.

A spasmodic twitching of the facial muscles has been observed in several cases. In one case ocular defect was the cause and the correction of it by glasses brought relief. In two cases the tic had been of long duration and had given the patient much discomfort. In both of these cases all medicinal remedies had been tried in vain. In tic convulsif the spasm is usually a reflex motor act in consequence of sensory irritation. Sometimes the seat of the irritation is evident because a local pain is present, and then division of the painful nerve will give relief. In neither of the cases mentioned was there any definitely localizable pain. It became necessary, therefore, to

determine (1) whether the spasm were reflex; (2) if so, in which branch of the trigeminal nerve the irritation began. This was arrived at by injecting cocaine beneath the skin of the face successively at the point of emergence of the supra-orbital, infra-orbital, and dental branches of the trigeminal nerve. In the first case the injections had no effect on the spasm when the upper two branches were anæsthetized, but the injection at the mental foramen in the lower jaw was succeeded by immediate stoppage of the spasm, which cessation lasted half an hour, until the local effect of the cocaine had subsided. This was interpreted to mean that the spasm was reflex, and that the irritation began in the lower branch of the trigeminal nerve. The patient was then operated upon by Dr. Hartley, who exsected this branch at its exit from the jaw with the effect of permanent cure of the tic. In the second case, referred to me by Dr. R. H. Derby, injection into the infra-orbital branch of the nerve stopped the spasm, and division of this branch was followed by permanent cure of the tic. In a few cases when no cause was found a labile galvanic current, 2 to 4 ma. applied for ten minutes daily, has given relief.

III. REFLEX OR TRANSFERRED PAINS.

There are many nervous individuals who complain of either sharp neuralgic pains or dull aching pains in various regions of the body for which no cause can be found on examining the painful region. Much light has been thrown upon these forms of pains by Dr. C. L. Dana. His observations are practical and of much value, and with his permission the following diagrams and table are introduced.

So called reflex pains are really transferred pains. For example, an irritation in the stomach may cause a pain and tenderness between the shoulders, particularly from the fifth to the eighth dorsal vertebræ. The explanation is as follows: the branches of splanchnic nerves from the stomach enter the spinal cord at the level of the fifth to the eighth dorsal segments; irritation along these nerves excites sensory impulses in these segments; those impulses are sent to the brain, and

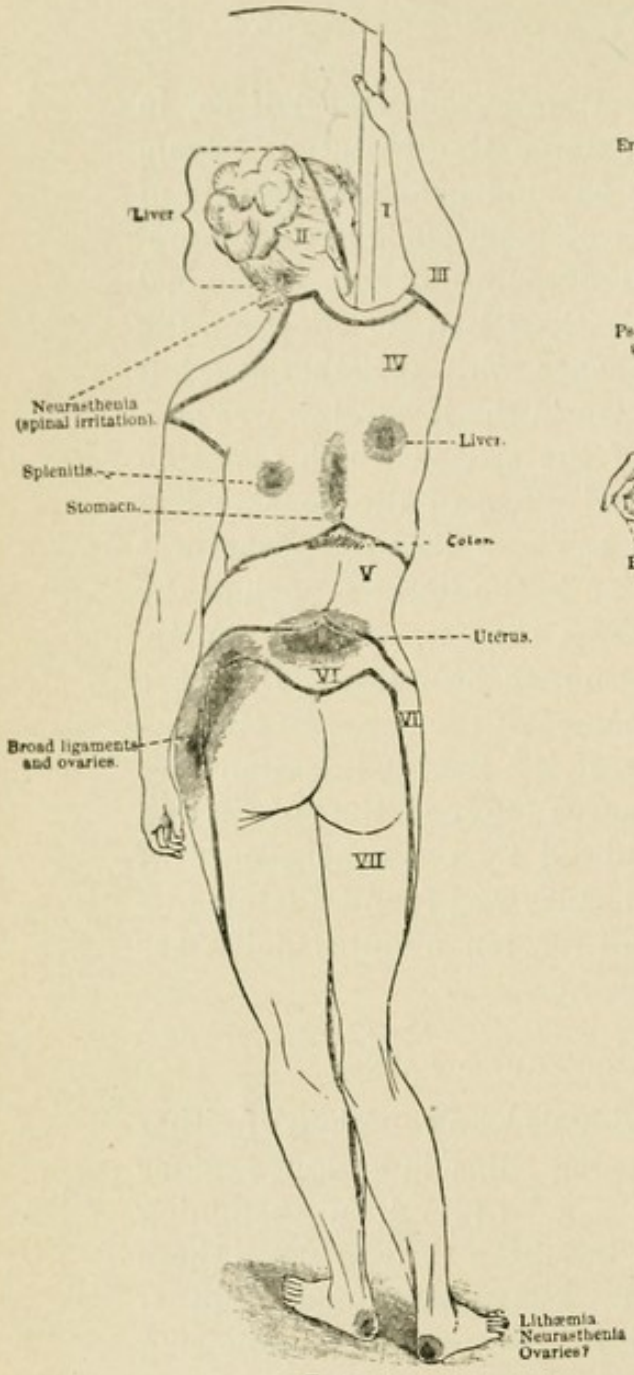

FIG. 70.

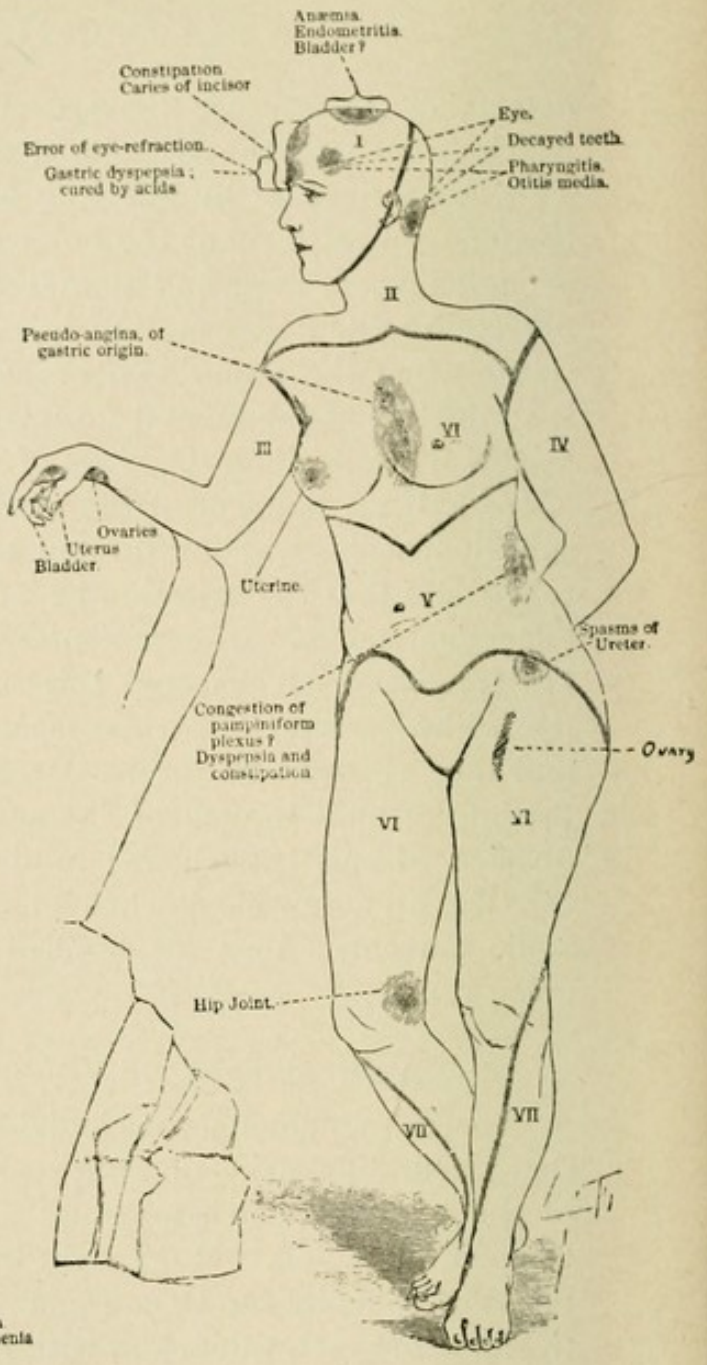

FIG. 71.

Area.	Strands of cerebro-spinal nerves.	Distribution.	Associated ganglia of sympathetic.	Main distribution.
I. . . .	Trigeminus, facial, etc.	Face and anterior scalp.	4 cerebral.	Head.
II. . . .	Upper 4 cervical.	Occipital region, neck.	1st cervical.	Head (slightly to ear).
III. . . .	Lower 4 cerv. and 1st dor.	Upper extremities.	2d and 3d cerv., 1st dor.	Heart.
IV. . . .	Upper 6 dorsal.	Thoracic wall.	1st to 6th dorsal.	Lungs.
V. . . .	Lower 6 dorsal except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.	5th to 12th dorsal.	Abdominal viscera, testes, ovary, fundus uteri *via* renal plexus.
VI. . . .	12th dorsal, 4 lumbar.	Lumbar region, upper gluteal, ant. and inner thigh and knee	1st to 4th lumbar.	Pelvic organs.
VII. . . .	5th lumbar and 5 sacral.	Lower gluteal, posterior thigh, leg.	1st to 5th sacral.	To pelvic organs, sympathetic supply being small.

Table showing distribution of the seven cerebro-spinal strands of nerves, and location of reflex pains.

are there referred by consciousness to the dorsal segments of the spinal cord. But since, in general experience, sensations and pain coming from those segments have been due to irritation in the surface of the body corresponding to them, these splanchnic sensations are referred to this surface. Hence the discomfort which should be referred to the stomach is referred to the back. The mistake is perfectly comparable to that which often occurs after an amputation when irritation in the stump leads the patient to refer sensations to the amputated hand or foot. Dana has studied the location of these reflex or referred pains very carefully and his figures demonstrate, more concisely than description, the area of pain in the diseases of the various viscera of sympathetic origin[1] (Figs. 70 and 71).

It will be seen, from their inspection, that there is hardly any viscus in which disease may not give rise to some referred pain. It is also evident that without some knowledge of the localities of these referred pains, the presence of such pains might easily mislead the physician. I have had several patients brought to me for supposed spinal disease in the dorsal region; the symptoms being pain in the back and sometimes in the epigastrium, tenderness of the dorsal spine, general weakness and neurasthenic complaints; in which the correction of a chronic gastric dyspepsia, a condition which had been supposed to be secondary to the more serious spinal affection, has been followed by permanent relief of the symptoms. It is not necessary to enumerate cases illustrative of these reflex painful affections. They are familiar to every practitioner. A study of the diagrams may, however, throw much light upon some hitherto obscure cases.

In reflex pains, treatment must be directed to the relief of the functional or organic disease in the viscus affected. It may be added that not infrequently sharp counter-irritation over the seat of the pain is followed by relief not only of the pain, but also of the disturbance of function in the viscus, which is the seat of disease.

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[1] N. Y. Med. Jour., July 23d and 30th, 1887.

IV. HEADACHES.

The More Common Forms and Their Treatment.

By DR. M. L. GOODKIND,
Fourth Assistant at the Nervous Clinic.

Headache is a symptom for whose relief, during the past year, at the nervous department of the Vanderbilt Clinic, 287 patients applied, and in presenting a brief description of the different varieties, I shall classify them in accordance with their frequency, avoiding all superfluous considerations, and confining myself strictly to the essential and inherent characters of each.

I. *Anæmic.*

118 Females ; 1 Male.

The first class in number and importance constitutes that variety of headache found almost exclusively in women, and which, from its etiological factor, it is usual to call *anæmic* headache.

This condition was found, as a rule, in women of middle age, who had become enervated by multiple cares of maternity ; and in young women living and working in a vitiated atmosphere and unable through poverty to regard proper hygienic rules. In both of these classes, poor living and a bad selection of food and drink (diet of cakes and pies, excessive tea drinking, etc.) were potent causative agents in the production of this form of cephalalgia.

The majority of these patients complained of a boring, frontal, or vertex pain, accompanied by ringing in the ears, palpitation and constipation; and presented usually a pale appearance. In more severe cases, there were amenorrhœa, vertigo, dyspnœa, œdema of the feet, and cardiac oppression causing fear and causeless apprehension. The violence of these various symptoms was so marked, in some cases, as to simulate organic disease; and especially prominent was the disposition of these patients to refer their symptoms to the

heart. Upon auscultation of this organ it was often possible to detect a functional, systolic murmur at the base, with accentuation of the pulmonary second sound; and a venous hum in the large veins of the neck.

All these patients, ranging in age from fourteen to seventy years, received treatment, which consisted in correcting their dietary (excluding cakes, candy, pies, tea, etc.) and directing sanitary regimen, with proper exercise in the open air; and in the administration of iron, arsenic, strychnine, cod-liver oil, and other tonics. The persistent use of aloin, in accordance with the suggestion in a valuable paper by Sir Andrew Clark,[1] was found to be of the greatest service in the treatment of these cases.

II. *Gastric.*

29 Females; 18 Males.

This class, like the preceding, presented many phases, but the following were very constant symptoms.

The patient complained of a headache, referring the pain to the occipital region, or to the forehead, or less often to the vertex, and gave a history of constipation, with flatulence, eructations, vertigo, nausea, and occasionally vomiting; and more rarely, in severe cases, cough, dyspnœa, hypochondriasis, and a feeling as though there were a weight in the orbit pressing upon the eyeball. They often complained of pain in the back.

These symptoms were present in various degrees of severity and duration—from hours to weeks.

Our first object in the treatment of these patients was to relieve them of the accumulation of fæces and products of decomposition, which generated noxious gases in their alimentary tract, causing a sort of individual sewer gas poisoning, and from which they were only relieved by emptying the intestines promptly and efficiently and several times. If enemeta were of no avail, resort was had to calomel or to the compound aloin tablet of the dispensary, followed by artifi-

[1] Lancet, Jan. 1st, 1887.

cial Carlsbad salts or a rhubarb and soda mixture, together with carminatives. Alkaline drinks were prescribed and in some cases charcoal, as they were indicated. These had, as a rule, the desired effect. As a prophylaxis against recurrence, we enforced upon our patients the necessity of regular habits, the avoidance of overfeeding, and the need of the constant use of large draughts of water. Much gastric headache with constipation may be relieved by enforcing the use, especially upon females, of a sufficient amount of water daily. Hot water before each meal was found of great service.

III. *Malarial.*

19 Females; 10 Males.

Under this heading we include those headaches, characterized by a train of periodically recurring symptoms, which are sufficiently distinctive to warrant the diagnosis of malarial origin, and which differed in form and location from the pain of trigeminal neuralgia.

The pain was usually supra-orbital in location, though it was sometimes occipital and not infrequently involved the entire side of the head.

It was variously described as a dull, heavy, or sharp, shooting pain. The time of its greatest severity was usually midday or in the afternoon. At the time of the severe paroxysms, the patient usually suffered from a feeling of chilliness, flashes of heat and cold, sometimes profuse sweating, malaise, and languor. Anorexia and constipation were frequent accompanying symptoms. Unless subjected to treatment, the headaches recurred day after day, or every third day or once a week. This tendency to periodicity was very marked in this form of headache.

The treatment pursued in these cases was the administration of a mercurial purgative, followed by 20 grains of quinine three hours before the attack was expected, or three doses of ten grains at intervals of an hour, beginning five hours before the onset. In many cases Warburg's tincture or extract was found to have a more prompt effect than quinine. This course

of treatment was usually of avail in the management of these cases, but in a few intractable cases, quinine failed to have the desired effect, when arsenic was used with satisfactory results. In a single case both arsenic and quinine failed, when given separately, while their combination was followed by subsidence of the headache, and other symptoms, which were very severe in this instance.

IV. *Syphilitic.*

6 Females ; 14 Males.

In cases of this form of headache, a history of syphilitic disease could often be obtained by direct admission of the patient, but when this was denied, either designedly or from ignorance, the concomitant symptoms (periostitis of tibia and frontal bone, enlarged glands, loss of hair, eruptions, scars on skin and mucous membranes, complaint of sore throat and rheumatic pains, etc.) generally elucidated the nature of the headache.

The symptoms, which this type of headache presented, were quite characteristic. The pain was usually most severe at night. Even though the patient had continuous pain, he would suffer an exacerbation as night approached. The pain was frontal or occipital, or diffused over the whole scalp, or the scalp would present small areas exquisitively sensitive and painful, so that a person ordinarily of good temper and equable disposition would become peevish and ill-tempered, and some deteriorated rapidly in health from the insomnia which ensued.

This class of headache is very persistent, unless subjected to treatment, to which it is ordinarily very amenable. The treatment consisted in the administration of the saturated solution of iodide of potash, beginning with ten drops t. i. d., well diluted with water or milk, and increasing one drop a dose until they took ʒ ss. to ʒ i. t. i. d. If this treatment proved inefficient, after two weeks' trial, inunctions of mercurial ointment, used in addititon to the potassic iodide, generally secured a favorable result.

This treatment was resorted to not only in the well-identified cases of syphilis, but also in those obscure cases giving a history of headache, only present in the evening or worse at night, and with no other evident cause, and the results in a few such cases, where every other form of treatment failed to relieve, justified this suspicion.

V. *Eye Strain.*

7 Cases.

In 7 of the patients treated, no other cause of headache could be discovered than an error in refraction of the eyes. In nearly all these cases hypermetropia with astigmatism existed, and from the strain imposed on the ciliary muscles, in increasing the convexity of the lens, to counteract the effects of this error of refraction a more or less severe headache—usually frontal in location—ensued. Until the proper methods for the rectification of the eye trouble were made use of, viz., the fitting of proper glasses, these cases were not relieved.

VI. *Traumatic.*

8 Cases.

In 8 cases, histories of blows or falls on the head were obtained with no evidences of fracture of the cranial bones.

These were treated by applications of cold cloths to the head, and by the bromides and iodide of potash. They were usually intractable cases.

VII. *Plethoric.*

12 Cases.

In 12 cases, all males, no other etiological factor could be acsertained than general plethora. These were men, for the most part, of short stature and florid habit, indulgent to themselves in the way of rich living, addicted to alcoholic excesses and of indolent disposition. They were very prone to sudden alterations in the cerebral circulation, following emotions of any kind, and this they manifested by outbursts

of violent temper and irascibility, or at times by becoming very despondent and melancholy.

During the attacks they described themselves as being subject to sensations of vertigo, with spots before the eyes, tinnitus aurium, feeling of suffocation, and intense frontal headache.

The treatment to be followed in these cases is sufficiently evident; changing the mode of life, sedatives to allay the vascular and cerebral excitement, aconite, chloral, bromides, etc., depletion of the overburdened vessels by active saline catharsis, and the use of ergot.

VIII. *Miscellaneous Cases.*

40 Cases.

In 40 cases the following causes were detected: nephritis; headaches, frontal and worse in the early morning, high tension pulse: carious teeth; headache temporal, and unilateral: gout; headaches usually relieved by a full meal: rheumatism and lithæmia; dull general headaches: arterio-sclerosis; headache severe, dull pain, worse about 4 A.M., when the patient was awakened by pain.

No causative factor could be found to which the headache might be attributed in 17 of the patients.

The treatment in these cases of headache was causal, while in the last class of cases, to which no cause could be assigned, treatment was conducted empirically, and the drugs which were of the greatest utility were antipyrine, antefebrine, and phenacetine. Exalgine was tried for a time, but was given up as inferior to the former drugs in these cases as well as in those of neuralgia.

Almost every form of headache, even including headache from cerebral tumor, was found to yield promptly to antipyrine in 15-grain doses or to phenacetine in 10-grain doses. The dose rarely had to be repeated, once after an hour. No untoward symptoms have been observed from the use of these drugs. The only form of headache which they usually failed to relieve was that due to general neurasthenia. They are invaluable for the relief of pain during the slow removal of the cause by other measures.

CHAPTER XIX.

THE TREATMENT OF NEURASTHENIA.

The vaso-motor origin of neurasthenic symptoms.—Mental recreation.—Overfeeding.—Baths.—Vascular stimulation.—Exercise.—Tonics vs. Sedatives.—Alkalies.

THE symptoms of neurasthenia are so well known as to require no comment. The causes of the disease are very numerous, but by far the most frequent one is a strain of an emotional kind, some worry, anxiety, grief, fright, or mental depression involving a constant irritation of the feelings, and a consequent excitement of the vaso-motor portion of the nervous system. Instability of vascular tone, irregularity in the circulation of the blood in one or more of the organs of the body and in the brain, seems to be the most constant element in the production of the symptoms of neurasthenia. This has been so fully demonstrated by the experiments of Mosso and of Angel, and so clearly shown by the pulse tracings of Webber, as to need no additional proof. In a large majority of the cases observed at the clinic this element has been clearly recognized. Without dwelling upon it fully, I desire to note that it is this instability of vascular tone which in neurasthenics prevents the maintenance of functional hyperæmia of the brain during work, thus preventing a concentration of attention during mental labor and hence producing the impairment of memory, the incapacity for continued reading, or study, or writing, or listening or conversation so often complained of. It is also manifested in other organs and causes the undue muscular fatigue, the imperfect gastro-intestinal digestion, the imperfect hepatic action, and possibly the imperfect assimilation of food and excessive excretion of uric acid so often observed. For while the coincidence of

neurasthenia and lithæmia cannot be denied, their mutual etiological relation is by no means finally determined. It is not a constant cerebral hyperæmia or a continuous cerebral anæmia from which neurasthenics suffer, but from an unstable vascular tone. The necessary sequel of such irregularity of circulation is an impairment of nutrition in the nervous system.

In the treatment of neurasthenia, the removal of the cause of mental distress is a sine qua non, and if this cannot be accomplished treatment can only be palliative. Time is the healer of much anxiety, grief, and worry, but while it is slowly soothing the cares away, mental occupation of sufficient variety and novelty to divert and amuse is a great aid. Mental rest is not secured by an absence of mental activity, but by directing the mind into new channels, and calling into play new departments of the organism, thus incidentally leaving those previously acting and now exhausted to recuperate. This is the secret of the success of travel, diversion, and novel employment in the treatment of neurasthenia. And these are therapeutic agents not to be despised.

In neurasthenic persons there appears to be an entire lack of surplus energy available for special efforts. They live on what they make. They are always on the verge of nervous bankruptcy. It is necessary for their recovery, therefore, to secure a supply of nerve force sufficient not only to carry them along from day to day, but to accumulate a credit on account for extra demands. Hence the need of what appears to be overfeeding in such persons. Three good generous meals, including all varieties of food, supplemented by three light lunches of easily digested fluids, such as milk, kumyss, weak punches, bouillon, chocolate with whipped cream, cocoa, egg-nog, or wine whey, are needed. And if artificial digestives are given, they should be given generously after each repast. Each person is capable of ascertaining the diet best suited to his needs, for what is one man's food is another man's poison, and the object is rather to feed the patient than to pursue any special theoretical scheme of dietetics. Hot water may or may not aid his digestion. Vichy, lithia, and seltzers waters

may or may not increase his powers of assimilation. As a rule an excess of fluid as well as of food seems to be of service, helping in the rapidity and completeness of the assimilative process throughout the body. Stimulants are, as a rule, to be avoided, but in a few cases, especially where heart stimulants are indicated, they may be allowed.

Next to mental diversion and overfeeding, come those means which aid the building up of new nerve tissue by securing a rapid circulation of the blood in all the organs. Material of repair must be carried to the seat of damage. To secure a sudden flushing of the neurasthenic brain and spinal cord with blood, is like bringing water to a thirsty man, or giving air to one nearly suffocated. Experimentally it can be shown that peripheral irritation produces vaso-motor reflex effects. This is the fact which hydrotherapeutic measures are based upon. It is of less importance what kind of water is used or at what temperature it is used, than that it should be so used as to produce a sudden change of state on the surface of the body. It is the shock which stimulates, not the continuous application. Therefore, whether hot or cold packs or baths or douches are employed, let either be followed by the other. My favorite methods are a warm spinal douche applied from the back of the head down to the coccyx for three minutes, followed by a cold application, by douche or sponge or damp towel for a half minute; or a warm pack in sheet and blankets for twenty minutes, followed by cool sponging for a minute; both being followed by brisk rubbing, or, if needed, by massage, and both being used at night as they prove most efficient hypnotics.

Such measures increase the force and frequency of the heart, dilate the vessels of the central nervous system, increase the strength and depth of respiratory efforts, and move the venous blood along the limbs—all important factors in the acceleration of the flow of blood. Massage aids the flow by promoting venous return, and, if combined with movements involving effort against resistance, by stimulating functional hyperæmia of muscles. General faradism does the same in less degree. Central galvanization may have the same effect

and possibly an additional effect in increasing chemical change. In a Turkish bath the douche, massage, and electrical applications may be combined, provided their duration be not so long as to weary the patient. In every case such measures should be succeeded by a period of rest, the patient being instructed how to relax his muscles and being advised to lie in any other than the dorsal position.

Regular exercise both in-doors with Indian clubs or a house gymnasium, and out-of-doors by walking, driving, riding, or bicycling, are essential, and even if this exercise has to be begun when it can only be pursued for a very short time, five or ten minutes, it must be enforced and the time gradually lengthened.

In all these measures the ability of the patient is to be considered, and the rule followed never to pursue any means of occupation, of exercise, of diversion, or of treatment, long enough to produce weariness. Nature furnishes a time limit by the sense of fatigue.

It having been for so long the custom to order sedative mixtures—such as bromides—in cases of nervous excitement, it may awaken surprise when the medicinal means employed at the clinic are reviewed. Bromides, chloral, hyoscyamus, antipyrine, phenacetine, and sulphonal will produce a temporary relief in many cases of neurasthenia. But while this fact is admitted, I do not hesitate to say that I discountenance their use. It is like quieting the fear and hallucinations of one in delirium tremens by giving him more alcohol, to give bromides to a neurasthenic. The last state of the man is worse than the first. There are cases of delirium tremens where heart failure threatens life, and whiskey must be used with the food which combats the state of starvation present. So there are cases of extreme nervous irritation and anxiety with sleeplessness in which phenacetine, sulphonal, and the other drugs mentioned are necessary while other means are being employed. Such cases are the exception. The best medicinal treatment in neurasthenia is one directed to building up the nervous system and to combating the irritable nervous weakness by giving strength rather than by suppressing

irritation. Iron, strychnia, arsenic, phosphorus, quinine, the alkaline hypophosphites, and cod-liver oil are the drugs most frequently prescribed in these cases.[1] No one remedy is to be used more than ten days. A succession of different tonics acts better than a combination of all or than any one used continuously. And an occasional intermission in the medicinal treatment serves to enforce the idea that the general measures are of far greater importance than drugs. The regulation of gastro-intestinal digestion may be borne in mind when the nerve tonics are administered; the strychnine may be given as Tr. nucis vomicæ before meals as a stimulant to the appetite, or the iron as the albuminate with some form of pepsin after meals. Phosphoric acid will aid the gastric juices, or the alkaline hypophosphites will counteract gastric acidity, as well as other acids or alkalies. Thus one means may accomplish two results. I regard arsenic as one of the best nerve tonics, especially in those forms of neurasthenia which are characterized by a loss of vascular tone.

The lithæmic state is one which so often coincides with neurasthenia that it requires notice. An occasional dose of calomel, the use of salicylate of soda, phosphate of soda, and carbonate of lithia, with or without small doses of opium, is not to be neglected when the two conditions are combined, and in such conditions a non-starchy diet is employed.

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[1] The formulæ used at the clinic are given in the last chapter.

CHAPTER XX.

THE ORDINARY FORMS OF INSANITY.

Analysis of seventy-one cases of insanity.—A practical grouping of cases of insanity.—I. Defective brains.—II. Diseased brains.—Varieties of each group.—Home *vs.* asylum care.

By FREDERICK PETERSON, M.D.,
Chief of the Nervous Clinic.

PHYSICIANS in general practice are continually brought into contact with the same classes of mental infirmity or disease as those which frequent the Vanderbilt Clinic. They are as a rule cases of congenital brain defect, or of the quieter psychoses, or incipient forms of brain disease; such as the various degrees of feeble-mindedness, imbecility, idiocy; and hypochondriasis, melancholia, paranoia, general paresis, and, more rarely, some of the maniacal conditions, and terminal dementia.

Certain points of interest may, therefore, be of value in connection with the diagnosis and treatment of these cases, and it is the object of this study to call attention to these practical features.

CASES AT THE VANDERBILT CLINIC.

There have been received and treated in the Nervous Department of the Vanderbilt Clinic, up to January 1st, 1890, 71 cases of insanity, 41 males and 30 females, which may be simply classified, according to the diagnoses entered in the clinic records, as follows:

Form.	Males.	Females.	Total.	
Hypochondriasis	17	1	18	
Melancholia	1	10	11	
Paranoia	4	5	9	
Imbecility	5	4	9	17
Idiocy	4	4	8	
General Paresis	7	1	8	
Hallucinatory Mania		2	2	
Senile Dementia		2	2	
Secondary Dementia	1		1	
Hysterical Mania		1	1	
Acute Masturbational Insanity	1		1	
Heboidophrenia	1		1	
Total	41	30	71	

The relatively large number of *hypochondriacs* in proportion to other cases is of course due to the fact that it is in the nature of the patient afflicted with this disorder to consult numerous doctors and to frequent successively most of the dispensaries of the city. Of the 17 males, the majority were suffering from this psychosis as a result of various abuses or maladies of the sexual apparatus. Some of the cases improved under treatment directed both to their general nervous condition and to the local etiological element; one or two recovered; but the majority disappeared from the clinic without much change in their mental state.

The cases of *melancholia* were mostly recent and simple, and owed the origin of their disorder to the usual dual cause, viz., physical ill health and mental strain. Two cases were puerperal and one was brought on by religious excitement. One of the puerperal cases was suffering from her second attack. In only one case was heredity ascertained. Two recovered under the opium treatment, which in my experience is the most advantageous, and the rest either were ordered to be sent to institutions or were lost sight of.

In most of the cases of *paranoia* a hereditary history was readily elicited. All had either fixed systematized persecutory delusions alone, or these in combination with delusions of a grandiose character. Two of the women were sisters, and presented an excellent example of *folie simultanée* (in their case *folie à deux*). Their mother was a paranoiac with the fixed

delusion that she was the Virgin Mary. One of the young women wrote for me an autobiographical sketch and the other a series of letters which were used as a basis for a study of their cases in the *Alienist and Neurologist*, January, 1890. The two sisters were brought before the students with other cases in a clinical lecture on paranoia, and naïvely exhibited their mental infirmities very clearly and perfectly, describing their continual persecution by an organized band of conspirators; their hallucinations of sight, hearing, smell, taste, and feeling; their delusions of poisoning by the introduction of deleterious substances into their food and water, and of noxious vapors into their rooms; and their continual moving from one house to another, and their applications to the police for assistance, in order to escape the machinations of their persecutors. Both sisters were eager to narrate their story, and what one failed to include the other supplied, until the mosaic of their psychosis was perfected.

Another paranoiac presented himself at the clinic with a large broad strip of thin sheet lead rolled up in his pocket, and an electro-telephonic instrument of his own invention which he called a "stethoscope." He was persecuted by a "gang" which made use of telephony and electric apparatus to annoy him. At night he wrapped the lead sheet about him for protection from currents. Each member of the "gang" had a "stethoscope," and he also had made one in order to ascertain the schemes of his enemies, and thus be better able to escape them. He explained the derivation of the word "stethoscope" as from "stetho," *stealthy*, and "scope," *to scoop*, for by its use the "gang" expected to "stealthily scoop him into their clutches!"

There was nothing especially noteworthy in the 17 cases of *idiocy and imbecility* received. We made an effort to secure the proper treatment for hopeful cases, and by this is meant of course mental and industrial training. One or two we were enabled to send to the State Asylum for Idiots at Syracuse. Several cases of partial or complete mutism were sent to institutions in the city for the education of the speech faculty.

The 8 cases of *general paresis* seen by us were mostly in

the early stages of the disease. Two remained under our observation until their death by convulsions, without exhibiting further mental changes than a progressive dementia, presenting none of the characteristic grandiose delusions or conditions of melancholy, or outbursts of maniacal excitement not uncommonly met with. Three were sent to asylums for the insane and the rest are still under treatment. One case was unusual on account of the youth of the patient. He was twenty-one years of age and had the typical somatic and psychic symptoms of the disease. (A number of younger cases have been recorded in literature.) One case developed rather suddenly after a fall upon the head, and at first presented symptoms which led us to consider his malady a pachymeningitis hemorrhagica. Later developments proved it to be unmistakable paretic dementia of traumatic origin.

One case was a fine example of *heboidephrenia* recently described by Kahlbaum (*Allg. Zeitschr. für Psych.*, 1889, part iv.). The rest of the insane patients presented no unusual features.

A PRACTICAL GROUPING OF CASES OF INSANITY.

A student or a general practitioner unfamiliar with the various phases of insanity, if brought into contact for a time with a large number of insane people, for instance, by a week's visit to a public asylum, would, in a short time, unconsciously arrive at a simple grouping of the insane into classes.

In a walk through the wards he would probably be at first struck by a diversity of shape in the heads of patients, for in every public asylum a certain number of cranial malformations are to be encountered. He would thus be able to classify the insane there met with into those with normally-shaped heads and those with heads abnormally formed—or, in other words, into such as had brains properly developed but fallen a prey to disease in adult life, and such as had brains defective from birth. Thus the first distinction he would make would be between the following two groups:

I. Defective Brains.

II. Diseased Brains.

Upon further examination of the first class he would distinguish certain degrees of defect, and would divide this class into such as were most infirm—*idiots;* such as were only moderately defective—*imbeciles;* and such as were manifestly only congenitally weak in mental capacity—*the feeble-minded.*

In the second class he would be able to distinguish greater diversity. From mere observation of physiognomy he would select the gloomy visages of the *melancholiacs*, establishing at once a distinct class of patients in a condition of mental depression (Fig. 72). The opposite extreme of great mental and motor excitement—an exalted or *maniacal state*—would be quickly noted.

He would furthermore see large numbers of patients who, though apparently idiotic, imbecile, or feeble-minded in their speech and demeanor, were yet distinguishable from such cases by the possession of normally-shaped heads, and in these he would soon learn, by inquiry into their histories, to recognize a class whose brains had once enjoyed normal faculties and ordinary activity, but had been weakened intellectually by some acute antecedent brain-storm, usually either mania or melancholia—a condition of mental enfeeblement, or *dementia* as it is generally termed. It is a dementia consequent upon a prior attack of insanity, and hence, as a rule, is called secondary or terminal dementia.

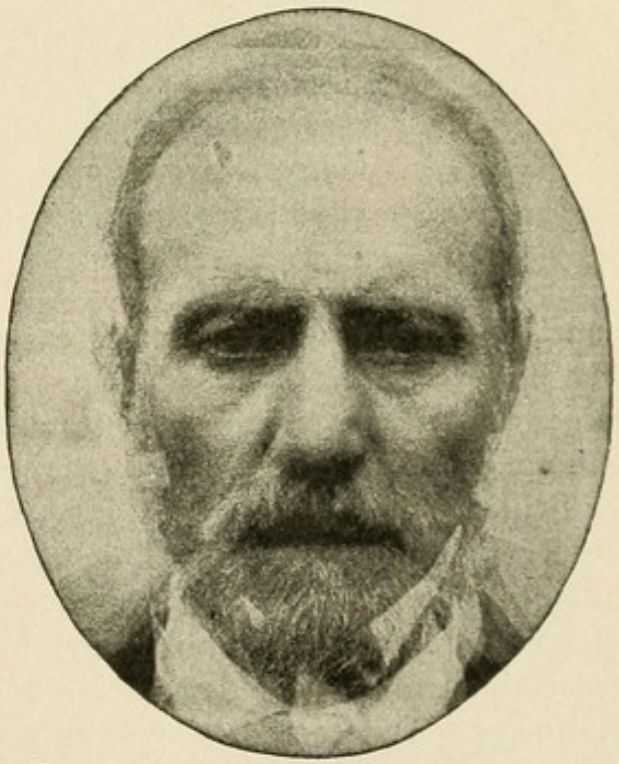

FIG. 72.—Composite Photograph of Six Patients suffering from Melancholia. (Noyes.)

It would not be long before a certain number of the pa-

tients would appear to possess a positive symptom-complex, identifying them as a class by themselves—a clinical entity

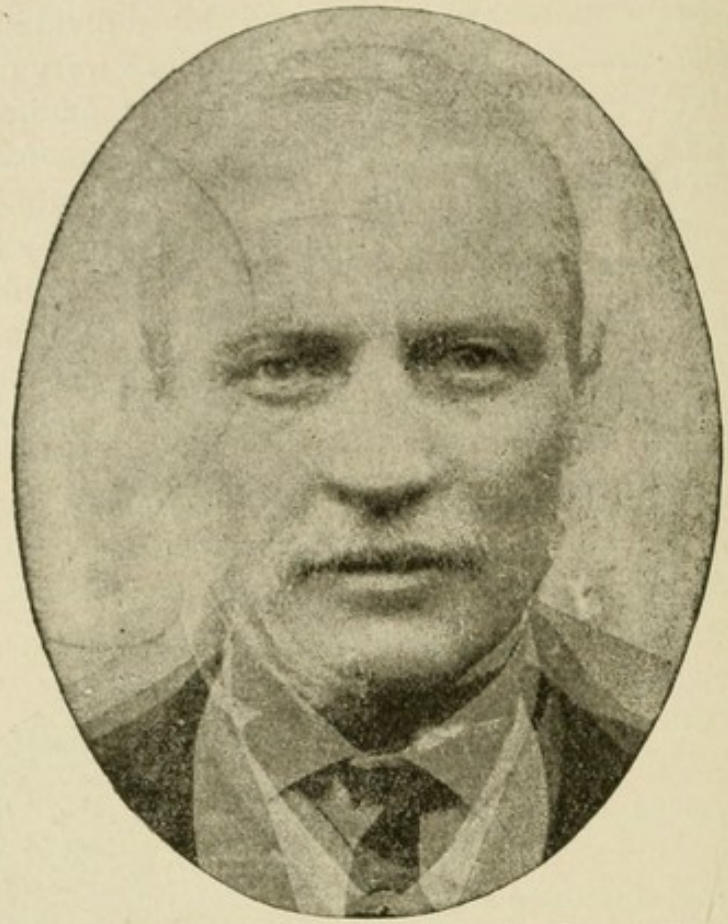

FIG. 73.—Composite Photograph of Eight Patients suffering from Dementia Paralytica. (Noyes.)

consisting of a progressively increasing paresis or paralysis of the body together with a progressive enfeeblement of the mind, and therefore correctly termed *dementia paralytica*, or general paralysis of the insane (Fig. 73).

Still another class would now appeal to the systematic observer—patients with fixed delusions, generally of persecution, often of personal aggrandizement, and in whom by careful inquiry and examination a hereditary taint was to be ascertained and some degenerative stigma (such as cranial malformation) detected. They have congenital instability of the intellectual centres, and at the same time their actual insanity may not have developed until the attainment of adult life; therefore it is difficult to determine whether they belong in the first or second great categories into which the novice

has attempted to classify the insane. He may decide, like Magnan, to consider them as congenitally deficient, yet prone to degenerate, a higher class than the idiots or imbeciles, and higher even than the feeble-minded—a sort of superior degenerate class. He will learn to call them cases of psychical degeneration or *paranoia*, which is the name now generally adopted.

This is my own early experience of the crystallization of insanity groups in the mind; I will not dignify it by the title of a classification, but I will call it a grouping of the various forms of insanity, which is so simple and natural as to better serve the requirements of the student and general practitioner than the studiously-elaborate classifications of the text-books, which are more adapted to the needs of the specialist.

The grouping arrived at, then, is as follows:

I. Defect of Brain.
   - (*a*) Idiocy.
   - (*b*) Imbecility.
   - (*c*) Feeble-mindedness.
   - (*d*) Psychical degeneracy (paranoia).

II. Diseases of Brain.
   - (*a*) Mental depression (melancholia).
   - (*b*) Mental exaltation (mania).
   - (*c*) Mental enfeeblement (dementia).
   - (*d*) General paresis (dementia paralytica).

The student or general practitioner needs no further classification. As his experience ripens, he will of course learn to recognize associated conditions or causes—hysterical, toxic, pubescent, puerperal, lactational, climateric, senile, hypochondriacal, epileptic, organic, cerebral—and to distinguish acute, subacute, chronic stages and primary, secondary, periodical, recurrent, circular, stuporous, or frenzied forms.

PARANOIA.

A typical case of paranoia exhibits certain positive features which make this form of insanity a clinical syndrome. There are hereditary taints; some eccentricities in childhood; more

marked peculiarities during youth, often associated with a degree of hypochondriasis; and at about the age of thirty, sometimes earlier, sometimes later, the growth and systematization of delusions of persecution, which may in turn be combined with or give place to systematized delusions of an exalted character (either religious, philosophic, patriotic, or erotic). Such delusions completely dominate their entire mental action without impairing every faculty.

This is the typical form as exemplified in the case so well described by Dr. William Noyes (*Am. Jour. of Psychology*, May, 1888) of the brilliant but "unfinished" artist whose delusions of an exalted religious nature were wonderfully expressed in his own drawings and paintings reproduced to illustrate Dr. Noyes' papers.

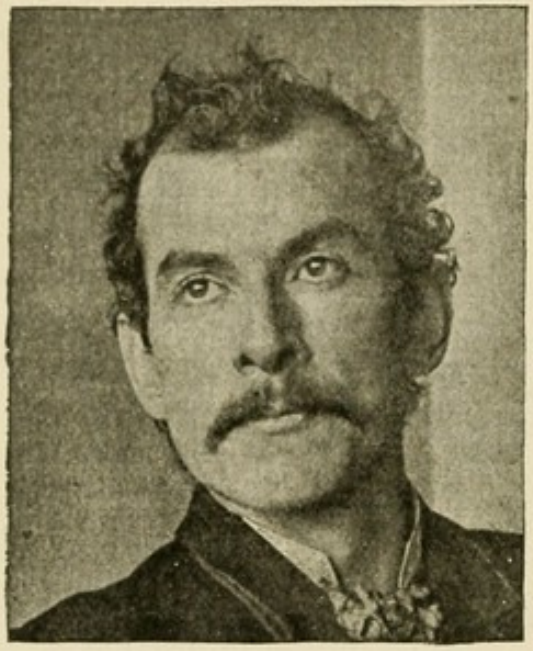

FIG. 74.—Photograph of a Paranoiac. (Field.)

Another perfectly typical case, a man of unusual intellectual culture and literary ability, was he who, under the title of "The Piling of Tophet" wrote an autobiography, now in my possession, extracts from which, with his history, will be found in my paper in the *Am. Journ. of Psychology*, January, 1889. He was a religious paranoiac, and had a paranoiac uncle.

The two sisters already referred to are excellent examples of cases with systematized delusions of persecution.

Some time ago, through the courtesy of Dr. M. D. Field, of this city, I had the opportunity of seeing and examining an erotic paranoiac, viz., the man who followed the actress, Miss Mary Anderson, over this country and Europe, for several years, never gaining an audience with her, but always deluded with the idea that she loved him, as he did her, with a pure, Platonic affection. He had hallucinations of hearing her

make love to him. Besides this purely mental love for her, he had expansive delusions of personal grandeur, claiming to be a great astrologer and the writer of a book explaining the cosmogony of the universe. I reproduce here his photograph (Fig. 74), together with the cranial measurements and drawings I made at the time of my examination (Figs. 75 and 76). There was an abnormal increase of height of the skull. A complete description of the case, together with transcripts from his own writings, will be found in Dr. Field's paper in the *Journ. of Nerv. and Ment. Dis.*, for September, 1889 (*Cf.* also *N. Y. Med. Journ.*, December 7th, 1889).

To illustrate the dangers to which

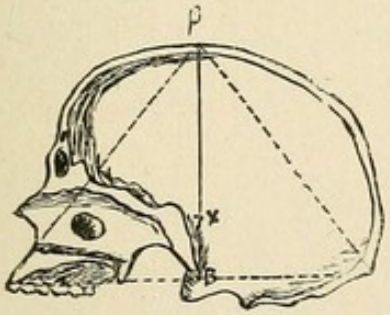

FIG. 75.—Measurements of the Cranium in Jas. D. The pathological variation is in β X and β B, and hence, although below the average in most of its measurements, the head of this paranoiac is above the average in the naso-occipital arc and binauricular arc, owing to the pathological height of the skull.

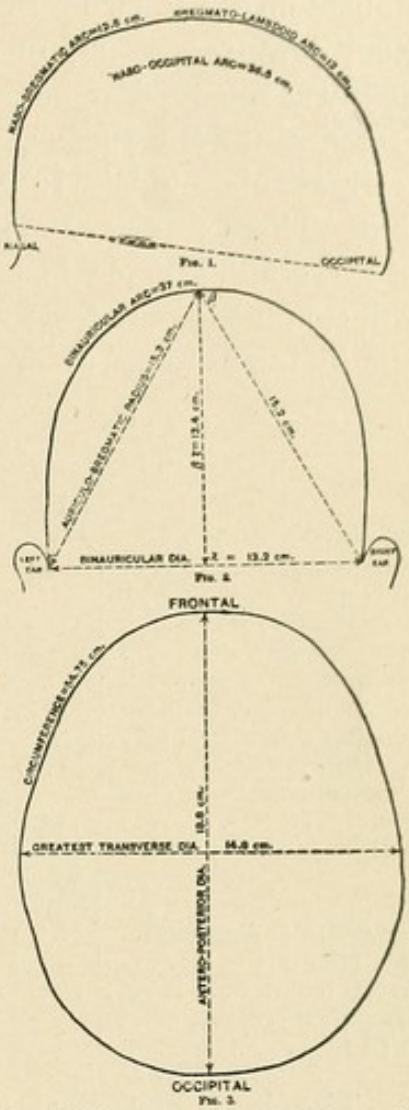

FIG. 76.—Measurements and Shape of Cranium in Jas. D.

Cephaloscopic Drawings of the Head of an Erotic Paranoiac. Fig. 1.—Departure from the normal proportion in the naso-occipital arc, owing to great vertical height of skull. Fig. 2.—Vertical measurement β χ is excessive, and a pathological variation from the normal. Fig. 3.—Shows asymmetry of the skull in horizontal circumference, the right frontal segment being greater than the left, and the left parietal segment greater than the right.

society is sometimes exposed by the presence among us of paranoiacs with persecutory delusions I need but refer to the case of Ernst Duborgue, who some years ago ran though

Fourteenth Street in this city stabbing right and left with steel compasses. Upon examination the diagnosis of paranoia was made, and he was sent to the Hudson River State Hospital for the Insane. He died there of pulmonary and intestinal tuberculosis, July 28th, 1886. I made an autopsy upon him. There was very marked asymmetry of his skull, sclerosis of the cranial bones, increase of cerebro-spinal fluid, and convolutional asymmetry. His brain weighed forty-three and one-quarter ounces avoirdupois.

TABLE OF CRANIAL MEASUREMENTS.

	Average of normal male skull.	Limits of physiological variation.	Jas. D.		Remarks.
			Head.	Skull.	
Circumference	52	48.5—57.4	54.7	51.5	Below average.
Volume (rough approximation)	1500	1201—1751		1390	Below average.
Naso-occipital arc	32	28—38	36.5	34.3	Above average.
Naso-bregmatic arc	12.5	10.9—14.9	12.5	12.5	Average.
Bregmato-lambdoid arc	12.5	9.1—14.4	12	12	Below average.
Binauricular arc	32	28.4—35	37	34.7	Above average.
Antero-posterior diameter	17.7	16.5—19	18.8	17.8	About average.
Greatest transverse diameter	14.6	13–16.5	14.8	13.8	Below average.
Length-breadth index	82.2 Brachycephalic	76.1—87	78.7 Mesaticephalic	77.4 Dolichocephalic	Below average.
Binauricular diameter	12.4	10.9—13.9	13.2	12.5	About average.
β X (Bregma to X)	11.7	10–12.65	13.4	12.9	Pathological.
Facial length	12.37	10.5—14.4	12.5	12.5	Above average.
Empirical greatest height (β B)	13.3	11.5—15	15.5	15.5	Pathological.

The best historical and critical survey of the subject of paranoia has been written by J. Séglas. It has been translated into English by Dr. William Noyes and published in the *Journ. of Nerv. and Ment. Dis.* of March, April, May, June, 1888. For studies of paranoiacs, famed in national annals and in bellestristic and æsthetic domains, such as the rulers of Russia, Austria, Bavaria, Spain, the cases of Louis Riel, Guiteau, Swedenborg, William Blake, etc., the interested reader is referred to Dr. W. W. Ireland's "The Blot upon the Brain" and "Through the Ivory Gate."

Although it would be well to restrict the term paranoia as much as is possible to the class of cases I have cited, which were formerly called "monomania" by Americans, *primäre*

Verrücktheit by the Germans, and even still by the all-too-conservative English "monomania" or "delusional insanity" (see Bevan Lewis' "Text-book of Mental Disease," 1890, page 192), yet there are undoubtedly cases not so typical as those I have described to which the term paranoia often appears to be equally applicable. These are what may be considered *formes frustes* of paranoia; some for instance which break down altogether at puberty, some which pause at one of the stages of evolution to progress no farther, and some again which may be conjoined with other varieties of insanity. The eccentric individuals popularly known as "cranks" are without doubt imperfectly developed cases of paranoia.

As regards the treatment of these patients, little can be done except perhaps in the way of moral methods, and these are of most value in the earliest stages. They may abort the psychosis. The asylums are the destination of most paranoiacs, and in the best of these institutions the discipline, employment, recreations, and regularity of eating and sleeping, exercise a beneficial influence upon the course of the disease.

Paranoiacs should be very carefully examined, especially those with persecutory delusions, as to the presence of ideas of retaliation and vengeance upon their persecutors. If there is any suspicion of their possessing dangerous tendencies they should of course be deprived of their liberty as soon as possible.

Melancholia.

This form of insanity is so well-known, that a definition is unnecessary here. But I would call attention to the necessity of early isolation of melancholic patients from their relatives and friends, and to the particularly careful examination requisite to determine the existence of suicidal inclinations. Suicide is more frequent among the quiet cases of mild melancholia who seem not to need asylum care than among those actually sent to asylums; and I am sure I am correct in saying that many cases go unrecognized as real insanity by the family physician, until suicide, or possibly both homicide and suicide, suddenly awaken him to his irremediable error.

Thorough investigation, unceasing vigilance, speedy separation from friends, and early commitment to an institution, should be the physician's rule in all of these cases.

With regard to medicinal treatment, the use of opium in the form of tincture or extract, in gradually increasing doses, I have found, in common with many other asylum physicians who have carefully tried its utility upon large numbers of patients, to be productive of the best results; morphia is not so efficacious, but I have at times employed successfully the

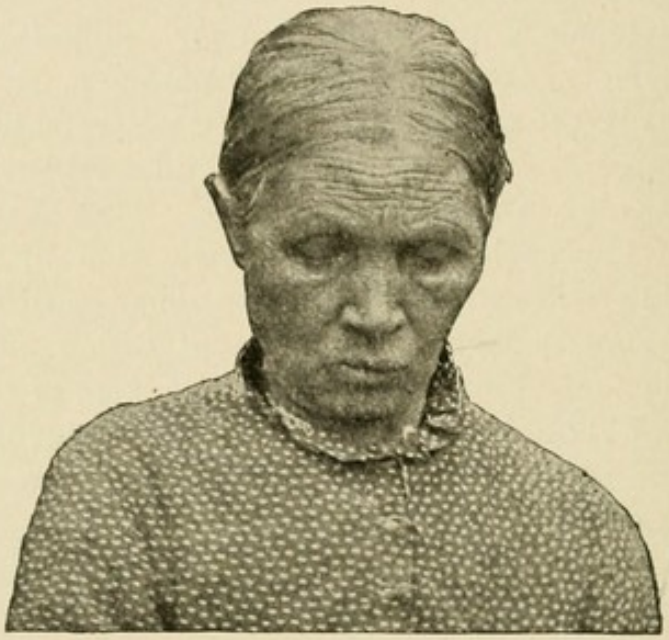

FIG. 77.—Photograph of Patient suffering from Melancholia. (Peterson.)

bromide of morphia. Opium should be administered without the knowledge of the patient (indeed it is safest that no one but the physician himself should know what is prescribed) and carried to the extent of forming a habit, if need be, later gradually diminished until it can finally be withdrawn altogether.

A valuable feature of the opium treatment is, that instead of increasing the constipation coincident with this disease, I have found it to counteract this condition. After a short time the bowels move with regularity every day.

I have much faith in the use of the prolonged warm bath (one-half to two hours) as a hypnotic agent in melancholia.

Frequent feeding, even forcible feeding if necessary, is of the greatest importance in the treatment. It combats the condition of anæmia and physical prostration which is often the cause of the disease.

The excellent composite photograph of six cases of melancholia made by Dr. William Noyes is, with his kind permission, reproduced (Fig. 72); and also a photograph of one of my own patients (Fig. 77).

Mania.

Maniacal conditions are so familiar and so readily recognized that they need not be described here. Most cases of this kind are preferably treated in hospitals or asylums, rather than at home. But when for some reason immediate removal is impossible, the motor and mental excitement are temporarily allayed best by the hypodermic injection of hyoscyamia or hydrobromate of hyoscine (doses of from $\frac{1}{100}$ to $\frac{1}{60}$ at first), and exhaustion prevented by the wet pack and overfeeding. In feeding maniacal patients, liquid food only can as a rule be used, whether by ordinary forcible methods, or by the nasal or stomach tube. This liquid food should consist of milk, raw eggs, Valentine's meat juice, sugar, and, where indicated, whiskey. I think the employment of the tube only rarely necessary; in fact I finally abandoned it in my asylum practice, because I found few patients to resist more peaceful methods for any dangerous length of time, and in the worst cases I was able to feed without the tube on making the attempt just as they were passing under the influence of the hypodermic injections. While non-restraint is the rule of asylum practice, the long-sleeved camisole is often absolutely necessary in cases treated at home.

General Paresis.

Dementia paralytica in its earliest stages first comes into the hands of the general practitioner, and is usually treated by him for months before pronounced insanity develops. I

need not dwell upon the importance of early recognition of this disease, not indeed for purposes of treatment, which is rarely if ever of any avail to stay its fatal progress, but on account of the gravity of the prognosis which is to be made. The onset of this disorder is protean in its manifestations. The earliest symptoms may be either physical or mental or both. Tremor of the fingers and of the hand in writing; fibrillary tremor of the tongue and lips; slight difficulty in the pronunciation of certain words such as occur in the phrase "Grief brings frightful dreams"; overaction of the occipito-frontalis muscle; slight irregularity or inequality of pupils, myosis, or loss of the pupillary reflexes; exaggeration of the wrist, elbow, and knee-jerks (rarely diminution); all these are among the earliest somatic characteristics. Later these become more and more pronounced until diagnosis is inevitable.

The mental symptoms are particularly variable. There may be at first but a melancholy or a hypochondriacal condition. This is more common as an early symptom than that feeling of well-being and personal aggrandizement which almost always makes its appearance at some epoch in the disease. Loss of memory, temporary amnesia, and, as a result of these, a certain amount of incongruity of action; emotional susceptibilities; the inappropriate or misplaced use of words; these are among the most frequent of the psychic prodromi. Sometimes unexpected maniacal outbreaks occur.

Convulsions, epileptiform or apoplectiform, generally usher in the later stages of general paresis, but these may also make their appearance very early and very unexpectedly.

A limited number of cases will progress to a fatal termination without manifesting symptoms necessitating their removal from home, but the great majority ultimately require the special supervision and care of the infirmary ward of an institution.

Where syphilis has a share in the etiology, treatment should of course be directed to this factor, but it will seldom alter the fatal course of cortical dissolution. Physostigmine and ergot in small doses may be administered. These drugs occasionally modify some of the symptoms, but no agent as

yet known will do more than slightly postpone the lethal termination.

On two occasions of late, trephining seems to have caused a temporary remission in general paresis, possibly, as claimed, by lessening intracranial pressure (*Brit. Med. Jour.*, Nov. 16th, 1889). If surgical interference could be depended upon to produce even a remission, it would certainly be justifiable.

Remissions, with considerable improvement at times, and lasting for months, rarely for a year or more, may be anticipated in a restricted number of cases; but such episodes have no influence upon the unfavorable prognosis.

HOME AND ASYLUM TREATMENT.

While it is apparent from the foregoing that I believe in the early removal of most cases of insanity from the environment in which the psychosis has developed, I am by no means an admirer of asylums in general as now conducted. The large public institutions are hampered in their treatment by the enormous number of patients and by the lack of a corresponding number of physicians and attendants. The superintendent is seldom a thoroughly trained alienist, and in some States these charities of the people are unfortunately made to subserve the interests of the great political machine. Patients cannot derive that benefit which they should from the medical superintendent's long experience, because their individual requirements must be sacrificed to the many demands upon his time in the general management. His assistants are too few and too pre-occupied with their clerical and office duties to carefully individualize and treat the patients. Finally there are too many patients. A State asylum containing but two hundred patients has but an indifferent public standing. It must have a thousand or two thousand patients in order to satisfy the ambition of the community and of the managers. Small wonder, then, that the actual object of the institution should so often be lost to sight!

Until therefore these great charities, now little more than warehouses for the storage of articles unnecessary or in

society's way, conform more to the character of a hospital, with its modern equipment, its attending physicians, and its consulting specialists, the higher classes of private asylums in the hands of men who are known for their professional attainments and probity of character will always be more desirable as places for the reception and treatment of such insane patients as are so fortunate as to be able to enjoy their advantages.

It is to be hoped, however, that at some future day our general hospitals will provide special wards or pavilions for this particular class of cases, so that in every city one or several places will be at all times ready to receive the acute insane and care for their sick brains in the highest scientific manner.

CHAPTER XXI.

ELECTRICITY AS A THERAPEUTIC AGENT.

Elementary physics.—I. Static electricity.—II. Voltaic electricity or galvanism.—Catalytic effects.—Cataphoric effects.—Electro-tonic effects.—III. Faradism or the induced current.—Summary.

THE modern view of electricity differs widely from the older views often reproduced in medical literature. To follow them closely would lead one to consider electricity as an entity, possibly a fluid, which can be administered like a drug. The term "current" is partly responsible for this fallacy, it being difficult to deal with this term without imagining a flow of something in a definite direction. Yet just as the older term caloric is now discarded, and the old notion that "heat is the result of caloric being poured into a body" raises a smile, so similar fallacious notions regarding electricity should at present meet with disapproval.

The sooner it is realized that electricity, like heat and light, is merely a form of activity in matter, or, to be somewhat more precise, in the imponderable ether surrounding each molecule of matter, and the sooner the term "electrical current" gives place to the more accurate phrase *state of electrification*, the sooner will the mystery surrounding this agent be cleared away.

When electricity is applied to the body a change is produced in the normal condition of the molecules making up the body, a new molecular condition is produced, but nothing is added to or subtracted from the body itself.

Between the two poles of the battery this molecular state is more intense than elsewhere, and under one pole it is somewhat different from that under the other. The intensity of the change is measured roughly in terms of the strength of

its pull upon a balanced needle—but this should not lead to the adoption of such terms as a dosage of electricity, which, like the term current, is sure to mislead.

When electricity is thus regarded as a mode of energy capable of estimation, like other modes, in terms of work, it becomes evident that a valuation of the various forms under which this energy appears is possible.

In common usage these forms are static or frictional electricity, voltaic electricity or galvanism, and induced electricity or faradism. It should be distinctly understood that all these forms are really the same mode of energy displayed under varying conditions. An illustration may make this plain. Suppose that you have a dry sponge and a tumbler of water. If you throw the water over the sponge the greater part of it will run off, leaving only the surface of the sponge wet. You have applied a quantity at once, and your effect is short and soon over. Suppose, on the other hand, you let the water fall on the sponge drop by drop. It will soak in and wet it thoroughly but gradually, until in the end all the water will remain in the sponge, which is now soaking. The effect is slow but intense. Water and sponge are the same in both cases. The conditions vary under which they have been brought together.

This illustration, which is not to be taken literally, may convey some notion of the difference between static and voltaic electricity. By the former sudden transient effects are produced very limited in intensity. By the latter, slow, permanent effects are produced of much intensity.

I. STATIC ELECTRICITY.

It seems strange, when the history of the uses of electricity is reviewed, to find how much credence is still placed in the effects of frictional electricity. In the first place, the change of electrical state produced by it is limited exclusively to the surface of bodies charged. Take a metal ball insulated by a glass stand over which two hemispheres are fitted, and charge this with static electricity. If the hemispheres be now re-

moved from the surface, they are found to carry all the electrical charge, and no change of electrical state has been produced in the metal ball beneath them. There is absolutely no proof that the human body acts in any way differently from the metal ball. Any effects obtained by static electricity must, therefore, be from surface stimulation; and, hence, wholly reflex and indirect in character.

When the surface of a body is charged with static electricity, it discharges itself gradually into the air by diffusion, or directly into the ground if the body be not insulated. The human body cannot be permanently charged with electricity.

The state of electrification on the surface of the body is communicated to the air about, which is thus put in a similar electrical state. But bodies in a like state of electrification repel one another. Hence the hair, when charged, is repelled from the body, and stands on end; air, when charged by diffusion, moves away from the body, and the air in motion may be felt as a breeze; hence, when a person is charged with static electricity this breeze is perceived.

If all effects of static electricity are reflex in their nature, due to irritation of the skin, then the agent differs in no respect from any mild form of counter-irritation—such as whipping with twigs, lomi lomi of the Sandwich Islanders, the needle douche of a Turkish bath—excepting, possibly, in the convenience of application. Stimulation of the circulation, contraction of muscles as from a blow, agreeable sensations, or sharp pain, may all be produced in such a reflex manner, if they are considered desirable. But no further distinctly curative effects can be produced. And, as we shall see later, the same effects can be secured by faradism.

It is possible to produce muscular contractions by the use of static electricity through the clothing, and therefore the necessity of undressing a patient is obviated by the use of this form of electricity, which is, therefore, convenient in large clinics. But it is not easy to limit its action to definite muscles, and as in the majority of cases of paralysis it is desired to obtain a tonic nutritive action as well as mere exercise, the other forms of electrical application are preferable. Another

objection to the use of static electricity is the fact that an efficient apparatus is cumbersome, takes up a large amount of space, cannot be transported, and requires an assistant to revolve the wheels.

There is something so startling in the application of electric sparks to the body that static electricity has been employed for producing sudden mental impressions.

The elements of mystery and of expectant attention doubtless play a part in the treatment of some functional nervous affections. It is an open question how far they may be legitimately employed. When static electricity relieves hysterical affections, it must be admitted that it is largely by means of the mental effect produced.

II. VOLTAIC ELECTRICITY OR GALVANISM.

A frictional machine does all its work suddenly, in a fraction of a second. It is not capable of producing a constant electro-motive force. This can, however, be obtained from a galvanic battery; and very soon after the discovery by Volta, the use of static electricity was largely superseded in medicine by that of galvanism. The fact that a voltaic battery furnishes a definite amount of electro-motive force capable of doing a measurable amount of work and producing definite effects on the body suffices to explain its continued use. Yet, when definite effects from galvanism are considered, there are few physicians to be found who will contend for a moment that there is any such certainty regarding the therapeutic effects on the body as there is regarding the use of drugs. Opium will quiet pain; aconite will depress the heart. But there is no statement which can be made with equal definiteness regarding electricity.

As far as the effects of galvanism are now known, they may be classified under three distinct heads, viz., catalytic, cataphoric, and electro-tonic effects.

a. Catalytic Effects.—The splitting up of a compound element into its constituent parts is one of the ways in which the work done by electricity is measured. The compound

body is put into a state of electrification which makes the condition of its molecules so unstable that they fly apart, and as they burst asunder those which have taken on a state like that of one pole of the battery are repelled from it and appear at the opposite pole. A strong current passing through the body produces intense catalytic effects, disintegrating and destroying the tissues, and raising blisters under each pole, the one (negative) containing acid, and the other (positive) an alkaline fluid. Even with a weak current, the taste produced in the mouth differs at the different poles, one being sour, the other salty. There is no doubt, then, that marked chemical changes may be produced by the galvanic current.

This power, then, may be used like that of any other destructive agent, to dissolve body tissues. For some purposes —such as elimination of hairs from the face—it is convenient. How successful it may prove in the destruction of tumors—especially of the internal organs—it is hardly time to judge. In the resolution of strictures its use is strongly combated by eminent authorities after thorough trial. In any case the difficulty of limiting its action, the serious nature of the wound which is left, the slow healing and deep scars which remain, combine to make it less certain, less definite, less satisfactory, than ordinary surgical procedures—and, consequently, it is not indorsed by surgeons generally.

The use of mild, non-destructive currents has been urged by some, and it has been claimed that a catalytic effect of a moderate kind may aid nutrition. Nutritive processes are dependent upon chemical and molecular changes. Is it possible to stimulate these by a constant galvanic current? Theory here is aside from the point. Experience must furnish facts. The only scientific observation which I have been able to find is furnished by Prof. James K. Thacher, of Yale College, and it is certainly extremely valuable and interesting. In a case of bilateral paralysis of all the extremities, due to diphtheritic multiple neuritis, Dr. Thacher made a series of therapeutic experiments upon the arms, each arm being treated alone for a week at a time, and its relative gain under treatment being compared with that of the opposite untreated arm. The re-

sults were measured in degrees of strength of grip as determined by a dynamometer.

It was found that the arm to which a labile constant galvanic current was applied daily for ten minutes, gained in strength in a degree far greater than that of the untreated arm. So that the gain under galvanism could be exhibited as follows:

	Galvanized arm.	Untreated arm.
1. Ten days' treatment to left arm,	17° gain	12° gain.
2. Seven " " to right "	15° "	10° "
3. Seven " " to left "	7.4° "	0.9° "

The galvanized arm made almost double the progress of the other (1 : 1.72).

The gain under faradism was much less perceptible.[1]

I know of no other accurately measured, scientific test under proper conditions which demonstrates so perfectly the power of the constant galvanic current to promote nutrition. It would add greatly to our knowledge if similar observations were made. And it is undoubtedly the catalytic effect of the electricity which is responsible for the nutritive changes.

It is very important to produce nutritive changes in many nervous cases, especially in all forms of paralysis, where it is necessary to keep the muscles strong, until the lesion is repaired by nature. It is undoubtedly through its catalytic action that galvanism produces many of its most beneficial effects in nervous diseases.

This is particularly true of its effects in neurasthenic conditions due to imperfect nutrition of the general nervous system. A neurasthenic person usually feels refreshed and invigorated by an application of galvanism to the spine or to the body generally. This should be made by means of large sponge electrodes applied to different parts of the spine and moved up and down during the application; or by means of central galvanization, first described by Beard, one pole being placed on the epigastrium and the other moved about over

[1] Medical News, April 18th, 1885.

the entire body; or, by another method of Beard in which the patient's feet are placed on one electrode and the other is placed upon the spine. In such treatment the strength of the current must depend somewhat upon the sensations of the patient. A daily application for ten minutes of a current of twenty milliampères (ma.) is to be employed. Twenty milliampères is usually as strong a current as can be borne when sponges six inches in diameter are used. If the size of the sponge is increased, the current, being spread over a larger area, must be increased in strength to produce the same effect at any part of this large area as it produced in any part of the smaller area. Thus the strength of current has to be regulated by the size of electrode as well as by the varying resistance of the patient's skin, which latter factor is extremely variable. Hence the absolute necessity of using a galvanometer in all galvanic applications.

In diseases of the brain, such as cerebral hemorrhage, sclerosis, abscess, tumor, or embolic softening, as well as in all forms of insanity, electrical treatment is useless. It cannot affect the pathological process which causes the symptoms. In diseases of the spinal cord it is usually recommended and often applied. Roughly, we may divide these diseases into (1) inflammatory destructive lesions of the gray matter, and (2) sclerotic processes. In the first class, of which disseminated myelitis and infantile paralysis may be taken as types, there may be an object in reducing congestion; but it is not certain that this can be accomplished for any length of time by electricity, as it can by counter-irritation or by cupping. After the acute stage is over, spontaneous recovery will occur in greater or less degree in many cases. It is possible that this may be aided by the application of electricity to the spinal cord. In the second class, of which locomotor ataxia is a type, it is theoretically difficult to assign any effect upon the sclerotic process of a change of molecular state. And, practically, few definite results can be affirmed regarding the effects of its application. Ataxia and spastic rigidity do not appear to be removed either temporarily or permanently, and the analgesic effects of electricity are very often disappoint-

ing. In spinal affections, therefore, it seems to me that experience does not confirm the statements of some text-books and of those whose interests lie in the direction of its use.

This experience conforms to that of such a careful observer as Gowers, who, in his recent text-book, states that electrical treatment fails in his experience to affect the progress of organic nervous diseases.[1]

Another question of importance is, to what degree the electrical state is induced in deep-lying tissues such as the brain and spinal cord. The body is not homogeneous and the power of conduction of different tissues varies greatly. If two pipes lead out of a reservoir, one free, the other plugged with rubbish, the water will seek the easy outlet to the exclusion of the other. To rely upon the older diagrams of the diffusion of electric currents in the body is to be misled.

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[1] The following quotations from Gowers are cited in support of the statement in the text.

"There is no evidence that the application of electricity to the spinal column has any influence on the process of recovery in acute myelitis. Its chief value is to maintain, as far as possible, the nutrition of any muscles of which the nerves have undergone degeneration."—Vol. i., p. 235.

No mention of electricity is made by Gowers in dealing with the treatment of chronic myelitis.—Pp. 245–6.

"Electricity has been strongly advocated and largely used in the treatment of infantile paralysis, and there is reason to believe that it is useful, although its influence has been much exaggerated. In no sense is it a curative agent, and there is no evidence that its application to the spine is capable of increasing the degree or accelerating the course of the recovery of the nerve elements. Nor is it easy to obtain evidence of its influence over the muscles. If the wasting is rapid, this progresses in spite of daily and sedulous applications. Nevertheless, its demonstrable effect on the muscles in causing their contraction must have an influence in the right direction upon their nutrition. This is of no avail if no recovery takes place in the spinal cord, but in most cases some recovery in the cord does occur."—P. 270.

"Electricity has little influence on the chief symptoms of tabes. The voltaic current is powerless over either the pains or the ataxy, whether it be applied to the spine or to the limbs."

After alluding to faradization of the skin in pain, and in incontinence of urine, Gowers adds:

"The central cause usually renders local treatment ineffective."—P. 326.

"Electricity is useless in spastic paraplegia."—P. 340.

"The most sedulous and skilful use of electricity, voltaic or faradic, fails, as a rule, to produce any effect on the course of progressive muscular atrophy."—P. 380.

Muscle conducts easily, bone less so, least of all the nervous tissue, which is peculiarly insulated by its fluid surroundings. The result must be that but a small fraction of the current reaches the nervous organs in comparison with the amount applied. Just how much does reach them has never yet been accurately determined, and cannot be from the nature of the case in a state of health.

It is, therefore, necessary to be guided still in the use of electricity in central nervous diseases by the teachings of experience and to use it in a somewhat empirical manner if at all.

In peripheral nerve lesions many of the objections urged against the use of electricity in affections of the central organs are obviated. It is possible to localize the application and to determine by the sensations and motions produced just what nerves are affected. It is certain that favorable nutritive effects are produced by the increased circulation and more rapid chemical changes induced by the agent. It would be interesting to ascertain definite time results in the treatment of these diseases, but facts are still to be collected. Is the duration of a lead palsy shortened by the application of a constant current? Is the course of a facial paralysis in any way affected by its use? I have been much disappointed by the lack of rapid results in the former. I have seen the latter go on to the most severe form, with a permanent paralysis, in spite of the most careful applications of electricity. Many cases of both diseases get well. It is still impossible to estimate the exact value of electrical applications in such affections, but that they are of great use both in aiding nutrition in the muscles paralyzed and by giving those muscles proper exercise is certain.

In neuralgia the galvanic current is also of service, the palliative effects of the positive pole being proven. The rule proposed by Dr. Gibney, of placing one pole over the nerve trunk and increasing the current until the patient has peripheral sensations in the area of distribution of the nerve treated, seems to me of great value, for unless such sensations are perceived it is unlikely that the nerve is really brought

into a state of electrification. If the spinal cord is really excited by the current, why should not general peripheral sensations be produced? It is rarely that I have succeeded in causing them, and I do not think they are elicited in the usual method of application.

b. Cataphoric Effects.—The power of electricity to promote osmosis is well proven. A transfer of substances from the positive to the negative pole through intervening organic tissue can be made. This is termed a cataphoric action. It has been used of late[1] in the production of local anæsthesia, and for the quieting of neuralgic pain; cocaine, morphia, and aconitia having been brought into direct contact with painful nerves in this manner. As a method of treating neuralgia this is of distinct service, and it may also be used in painful affections of the muscles.

The method of the application is simple. A short glass cylinder is covered at one end with parchment securely tied and sealed with collodion. Into this tube, filled with the solution of the drug to be used, the positive pole is introduced through a cork which serves to retain the fluid. The parchment surface is then applied over the painful spot and the current allowed to pass, the negative pole being placed at any part of the body desired. The strength of current used should be from eight to twelve milliampères, and the duration of the application from four to ten minutes. The drug is carried by osmosis through the parchment and through the skin.

Medicated electric baths, which might be arranged on this principle, cannot be at present commended, since the dosage of the drug thus employed is wholly uncertain.

c. Electrotonic Effects.—It has already been stated that the application of electricity produces a state of electrification in the molecules of the body. This state varies under the different poles of the battery, and each pole is supposed to produce an effect the exact reverse in kind from that under the other pole. These opposite states are known as anelectrotonus and

[1] "Electric Cataphoresis as a Therapeutic Measure." F. Peterson, M.D., N. Y. Med. Jour., April 27th, 1889.

katelectrotonus. It is not necssary for us to determine whether the state produced is due to a parallelism of molecular vibration, comparable to that occurring in polarized light, or whether it is a state induced in the dielectric, the impalpable ether surrounding the molecules. Suffice it to say that two electrotonic states exist, one at each pole, and that the tissues between the poles share in these states in the direct ratio of their nearness to the poles.

The sudden production of either state excites violent molecular movements in the tissues, and these become doubly violent if either state is suddenly replaced by its opposite.

Each tissue manifests its irritation in its own manner, the muscle by contraction, the motor nerve by sending a motor impulse to the muscle, the sensory nerve by transmitting a sensory impulse to the brain. Hence to excite tissues to functional activity sudden changes in their electrotonic state are produced by suddenly closing or opening or reversing the constant galvanic current. The last-named procedure, reversing the current, or using voltaic alternatives, is necessarily the most active excitant of all. No one can deny the beneficial effects produced by exciting the functional activity of the various organs, especially when, as in paralysis, the organs cannot be voluntarily set to work; for exercise aids nutrition, promotes the circulation, assists metabolism. It is probable that no other method equals this in these respects, although massage has a parallel effect. The electrotonic effect upon the vessel walls is also proven, for a decided dilatation of the smaller arteries can be seen after an application; and when this condition has passed off, if the part be immersed in warm water, the local redness becomes again manifest. This has been ascribed to the chemical changes set up by the catalytic action of the current, but the fact that it is more intense at the negative pole demonstrates that the result is due, in part at least, to the electrotonic state produced; and the fact that it is caused by a faradic current, which has no catalytic effect, confirms this statement.

Galvanism or voltaic electricity properly administered with accurate measurements is undoubtedly of great service in

the treatment of nervous diseases. For it produces chemical changes which aid nutrition; it transfers substances from place to place in the body, thus possibly stimulating the natural movement of fluids in vessels and lymphatics; and it excites organs whose function is suspended by disease to functional activity. It thus meets many of the needs which the physician appreciates in the treatment of nervous affections. As has been shown, however, it is of much greater service in functional disorders of nutrition and in peripheral nervous diseases, such as neuritis and neuralgia, than in grave lesions of the central nervous system. When used in the latter it is merely to combat individual symptoms, such as paralysis, and not to affect the process of disease to which the symptoms are due.

III. FARADISM, OR THE INDUCED CURRENT.

It has been stated that when the galvanic current is closed or opened, a sudden change of state is produced in the body. The same change of state is produced in an independent wire which lies at the side of the wire conducting the galvanic current. The current in the independent wire is said to be induced by magnetism. If such a wire be wound in a coil, each layer of the coil reinforces every other by magnetism, and when the coil is large the induced current produced is very strong. The strength of a faradic current can be increased by increasing the number of layers in a coil; or more conveniently by using coils of different thickness, or by pushing a single large coil further and further over the original galvanic coil, thus bringing more and more of the secondary coil over the primary one. While a galvanic current is passing continuously the induction of electricity does not occur in the secondary coil; hence the presence in every faradic battery of a vibrating rod which constantly interrupts the passage of the primary weak galvanic current, making and breaking the circuit and inducing the secondary current.

If electrodes are attached to the ends of the secondary coil of wire and the electricity is applied to the body, it is evident

that a series of rapidly recurring shocks will be given. The physical effect is so far exactly the same as in the application of a galvanic interrupted current. But a very strong effect of an electrotonic kind is produced by such a faradic application and a very weak or hardly perceptible catalytic and cataphoric effect.

The effects of faradism are, therefore, wholly electrotonic effects, never catalytic or cataphoric. Its convenience in being produced in a portable apparatus is its only advantage over voltaic alternatives. The difference of reponse to the faradic current from that to voltaic alternatives in conditions known as the reaction of degeneration, is not to be ascribed to any difference in the nature of the current, but only to the fact that the degenerated muscle will not respond to changes of state rapidly produced in it. This has been proven by increasing by a mechanical device the rapidity of voltaic interruptions, when the degenerated muscle finally ceases to respond to them, while the healthy muscle on the contrary becomes tetanized.

Tetanic contraction is produced in a healthy muscle by voltaic alternatives, whose rapidity is over twenty to the second. In a degenerated muscle, as the rapidity of the alternatives rises from five per second, the degree of contraction becomes progressively less, and when a rate of twenty per second is reached the muscle no longer contracts.[1] The rapidity of faradic interruptions is usually above twenty per second. Since muscular contractions are desired in the treatment of paralysis, it follows that faradism is useless unless the muscles act to it. In any case of paralysis, therefore, in which faradism produces contractions, viz., in all forms of paralysis from brain lesion, and in many very mild cases of spinal and peripheral paralysis, faradism may be used. If, however, as in the last named diseases, a nutritive and stimulant to chemical action is desired as well as mere muscular exercise, the galvanic current must be employed.

Admitting the great use of interrupted currents in exciting

[1] I am indebted to Mr. Charles Young, of Princeton College, for these facts and for a demonstration of them.

tissues to activity by producing changes of electrotonic state, can further claims be allowed? There is one which has already been alluded to in dealing with static electricity, namely, the reflex effect of surface irritation. It is not unlikely that changes of circulation or of molecular state—very beneficial in character—may be excited in central organs by such reflex irritation. It is evident, then, that the only effect which occurs from an application of static electricity can be produced equally well by interrupted currents.

Faradic electricity has one other use. In cases of anæsthesia from any cause, the faradic current applied by means of a wire brush over the anæsthetic surface is sometimes followed by a decrease in the degree of anæsthesia. And it has had also a reputation for quieting pain if applied in the same manner. Its limits, however, as is evident, are many; and for general use the galvanic current is of far greater service when properly applied.

To sum up the facts here presented, we may say that:

1. Static electricity offers nothing beyond an interrupted galvanic current, and fails to furnish those effects which are most desirable in the treatment of disease.

2. A constant galvanic current can produce chemical changes which aid nutrition, or destroy tissue according to the strength employed.

3. A constant galvanic current can transfer medicines into the body from without.

4. An interrupted galvanic current, or a faradic current, can excite various organs to functional activity, thereby aiding their nutrition.

5. It is questionable whether the pathological state causing organic diseases can be in any way influenced by electricity.

6. If functional diseases are benefited, it is in an uncertain manner; it being undecided whether it is by influencing the molecular condition, the chemical changes, the circulation, or the electrical state of the organ affected; or by the state of mental expectation induced. The agent is, therefore, used empirically, and the physiological indications for it are as yet

uncertain. As a therapeutic agent its use is very limited, and carefully balanced scientific observations are still needed to establish its proper sphere.

I cannot, however, close this chapter without stating that after the constant use of electrical treatment for the past six years in dispensary and in private practice, I have been disappointed in the results obtained. And I cannot ascribe this lack of result to any defect in the applications, for they have been made with care, with the best of apparatus, and with exact galvanometric measurements, according to the methods described by such authorities as Erb, De Watteville, Benedickt, and Beard. My experience coincides with that of Gowers, that the therapeutic effects of electrical applications have been much exaggerated and are really quite limited.

CHAPTER XXII.

PRESCRIPTIONS IN USE IN THE NERVOUS DEPARTMENT OF THE VANDERBILT CLINIC.

MIST. AC. NITRO-MURIAT.

℞	Ac. nitro-mur. dil.	♏ iiss.
	Tinct. nuc. vomic.	♏ i.
	Tinct. calumbæ	♏ x.
	Syr. limon	♏ xx.
	Aquæ q. s. ad	fl. ℥ i.

M. Dose ℥ ss.

MIST. ACID. TONIC.

℞	Tinct. nuc. vomic.	♏ v.
	Ac. nitro-mur. dil.	♏ x.
	Tinct. cinchon. comp. q. s. ad	fl. ℥ i.

M. Dose ℥ i.

MIST. ALKALINA.

℞	Magnes. sulph.	gr. viiss.
	Magnes. carb.	gr. iv.
	Spir. ammon. arom.	♏ iv.
	Aquæ q. s. ad	fl. ℥ i.

M. Dose ℥ i.

TABLET ALOIN, BELLADON. ET STRYCH.

℞	Aloin	gr. $\frac{1}{5}$
	Ext. belladon	gr. $\frac{1}{8}$
	Strych. sulph.	gr. $\frac{1}{120}$

M. One tablet.

TABLET BROMIDE COMP.

	Morphia brom.	gr. $\frac{1}{8}$
	Hyoscin. hydrobrom.	gr. $\frac{1}{100}$
	Camph. monobrom.	gr. i.

TABLET BROMIDE COMP. NO. 2.

Morphia brom	gr. $\frac{1}{16}$
Hyoscin. hydrobrom	gr. $\frac{1}{200}$
Camph. monobr	gr. $\frac{1}{2}$

MIST. "BROWN-SÉQUARD."

℞ Pot. iod	gr. vi.
Pot. brom.,	
Ammon. bromid āā	gr. xv.
Sod. bicarb	gr. iv.
Tinct. nuc. vomic	♏ vi.
Tinct. gentian. co	♏ viij.
Aquæ q. s. ad	fl. ℥ i.

M. Dose ℥ i.

MIST. CAPSICI.

℞ Tinct. capsic.,	
Tinct. zingib āā	♏ v.
Tinct. cinchon. comp q. s. ad	fl. ℥ i.

M. Dose ℥ i.

MIST. CASCARÆ COMP.

℞ Ext. cascar. fl.,	
Tinct. nuc. vomic.,	
Tinct. belladon āā	♏ iv.
Glycerin q. s. ad	fl. ℥ i.

M. Dose ℥ i.

MIST. CEPHALALGIACA.

℞ Ammon. chloridi	gr. iss.
Morph. acetat	gr. $\frac{1}{8}$
Caffein. citrat	gr. $\frac{1}{20}$
Spir. ammon. aromat	♏ $\frac{1}{3}$
Aq. menth. pip	fl. ℥ ss.
Elix. guaran q. s. ad	fl. ℥ i.

M. Dose ℥ i.

MIST. CHOREA.

℞ Liq. pot. arsenit	♏ iij.
Aq. menth. pip q. s. ad	fl. ℥ i.

M. Dose ℥ i.

MIST. CHOREA COMP.

℞ Liq. pot. arsenit..... ♏ iij.
Chloral. hydrat..... gr. v.
Aq. menth. pip.....q. s. ad fl. ʒ i.
M. Dose ʒ i.

PIL. COLITIS.

℞ Strych. sulph..... gr. $\frac{1}{40}$
Pulv. ipecac.,
Ext. belladon.,
Ext. colocynth. comp.....āā gr. $\frac{1}{4}$
Ext. gentian..... q. s.
M. One pill.

PULV. "DIGESTIVE."

℞ Pepsin..... gr. v.
Bismuth subnit..... gr. x.
Strych. sulph..... gr. $\frac{1}{100}$
Carb. ligni..... gr. v.
Thymol..... gr. $\frac{1}{4}$
M. Dose gr. xx.

MIST. "D. T."

℞ Pot. bromid.,
Sod. bromid.....āā gr. xv.
Chloral. hydrat..... gr. x.
Tinct. zingib..... ♏ x.
Tinct. capsic..... ♏ v.
Spir. ammon. aromat..... fl. ʒ i.
Aquæ.....q. s. ad fl. ʒ ij.
M. Dose ʒ ij.

MIST. EPILEPTICA.

℞ Pot. bromid.,
Sod. bromid.....āā gr. x.
Ammon. bromid..... gr. v.
Sod. bicarb..... gr. ij.
Liq. pot. arsenit..... ♏ i.
Aquæ.....q. s. ad fl. ʒ i.
M. Dose ʒ i.

MIST. EPILEPTICA COMP.

℞ Tinct. belladon..... ♏ ij.
Pot. bromid..... gr. xv.
Chloral. hydrat..... gr. v.
Aquæ.....q. s. ad fl. ℥ i.
M. Dose ℥ i.

TABLET EUONYMIN CO.

℞ Euonymin..... gr. ¼
Podophyllin,
Aloin.....āā gr. ⅛
M. One tablet.

PIL. FERRI CARB.

"Blaud's Pill."
℞ Pot. carb..... gr. iiss.
Fer. sulph. exsic..... gr. iss.
M. One pill.

MIST. FERRI CHLORIDI.

℞ Tinct. ferri chlor..... ♏ xv.
Glycerin..... ♏ xx.
Aquæ.....q. s. ad fl. ℥ i.
M. Dose ℥ i.

TABLET IRON, ARSENIC, AND STRYCH.

℞ Ferri redact..... gr. i.
Acid. arsenios..... gr. $\frac{1}{100}$
Strych..... gr. $\frac{1}{60}$.
M. One tablet.

MIST. FERRI ET STRYCH.

℞ Strych. sulph..... gr. $\frac{1}{32}$
Ferri pyrophosph..... gr. x.
Tinct. zingib..... ♏ i.
Glycerin..... ♏ v.
Aquæ.....q. s. ad fl. ℥ i.
M. Dose ℥ i.

MIST. FERRI SALICYLATIS.

℞ Acid. salicylic..... gr. xx.
Fer. pyrophosph..... gr. iv.
Sod. phosph..... gr. l.
Aquæ.....q. s. ad fl. ℈ ss.
M. Dose ℈ ss.

VINUM FERRI AMAR.

℞ Fer. et quin. cit ..... gr. ij.
Tinct. aurant. amar ..... ♏ iv.
Elixir. simpl ..... ♏ xv.
Vin. xeric ..... fl. ʒ ss.
Aquæ ..... q. s. ad fl. ʒ i.
M. Dose ʒ i.

PULV. LAXANS.

℞ Sod. chlor ..... gr. vi.
Sod. bicarb ..... gr. xxiv.
Sod. sulph. exsic ..... ʒ i.
Magnes. sulph ..... ʒ ss.
M. Dose ʒ ij.

PIL. LAXATIVA.

℞ Pulv. aloes ..... gr. iss.
Ext. nuc. vomic ..... gr. $\frac{1}{4}$
Ext. belladon ..... gr. $\frac{1}{6}$
Ext. colocynth. comp ..... gr. ij.
M. One pill.

MIST. MAGNES. ET FERRI SULPH.

℞ Magnes. sulph ..... gr. xv.
Ferri sulph ..... gr. i.
Acid. sulph. dil ..... ♏ xv.
Aquæ ..... q. s. ad fl. ʒ i.
M. Dose ʒ i.

TABLET "NEURALGIC."

℞ Quin. sulphat ..... gr. $\frac{1}{2}$
Morph. sulphat ..... gr. $\frac{1}{10}$
Strych. sulphat ..... gr. $\frac{1}{120}$
Acid. arsen ..... gr. $\frac{1}{80}$
Ext. aconit ..... gr. $\frac{1}{8}$
Sig. One tablet repeated in 1 hour.

MIST. POT. IODID. ET COLCHICI.

℞ Pot. iodid ..... gr. vi.
Tinct. cimicif ..... ♏ iv.
Tinct. opii camph.,
Vin. colch. rad. ..... āā ♏ v.
Aq. cinnam ..... q. s. ad fl. ʒ i.
M. Dose ʒ i.

SOL. PHOSPHORI, THOMPSON.

℞ Phosphori..... gr. i.
Alcohol. absolut..... fl. ʒ v.
Dissolve with the aid of gentle heat and add, previously heated:
Glycerin..... fl. ℥ iss.
Alcohol..... fl. ʒ ij.
Spir. menth. pip..... fl. ʒ i.
M. Dose ♏ xij. ʒ i. contains gr. $\frac{1}{20}$ phosphorus.

PIL. QUIN., FER. ET STRYCH.

℞ Strych. sulph..... gr. $\frac{1}{60}$
Quin. sulph.,
Fer. redact.....āā gr. i.
M. One pill.

MIST. RHEI ET SODÆ, I.

℞ Pulv. rhei..... gr. ij.
Sod. bicarb..... gr. v.
Pulv. ipecac..... gr. $\frac{1}{4}$
Aq. menth. pip.....q. s. ad fl. ʒ ij.
M. Dose ʒ ij.

MIST. RHEI ET SODÆ, II.

℞ Pulv. rhei..... gr. i.
Sod. bicarb..... gr. x.
Aq. menth. pip.....q. s. ad fl. ℥ ss.
M. Dose ℥ ss.

MIST. RHEI ET SODÆ COMP.

℞ Pulv. rhei..... gr. ij.
Sod. bicarb..... gr. x.
Pulv. ipecac..... gr. $\frac{1}{3}$
Tinct. nuc. vomic..... ♏ v.
Aq. menth. pip.....q. s. ad fl. ʒ ij.
M. Dose ʒ ij.

MIST. SALINA.

℞ Sod. et pot. tart..... ʒ i.
Sod. bicarb..... ʒ ss.
Pot. acetat..... gr. xv.
Aquæ.....q. s. ad fl. ℥ ss.
M. Dose ℥ ss.

TABLET "SCIATIC."

℞ Tinct. colchici,
Tinct. cimicifug.,
Tinct. aconit.,
Tinct. belladon.....āā ♏ ¾
M. One tablet.

MIST. SEDATIVA.

℞ Morph. sulph..... gr. $\frac{1}{48}$
Acid. hydrobrom. dil..... ♏ viiss.
Spts. chloroform..... ♏ v.
Syr. scillæ..... ♏ xv.
Syr. tolutan..... q. s. ad fl. ℥ i.
M. Dose ℥ i.

MIST. SOD. SALICYLAT.

℞ Acid. salicylic..... gr. x.
Sod. bicarb..... q. s.
Ext. glycyrrhiz..... gr. iij.
Glycerin..... fl. ℥ ss.
Aquæ.....q. s. ad fl. ℥ ij.
M. Dose ℥ ij.

MIST. TONIC.

℞ Tinct. nuc. vomic..... ♏ iv.
Ac. phosph. dil..... ♏ viij.
Syr. hypophoph. cal. et sod.....q. s. ad fl. ℥ i.
M. Dose ℥ i.

REGISTER OF CASES TREATED AT THE NERVOUS DEPARTMENT OF THE VANDERBILT CLINIC FROM FEBRUARY, 1888, TO JANUARY, 1890.

Diseases.	No. of Cases.
Acroneuroses	19
Alcoholism	72
Anæmia	25
Anterior Poliomyelitis	22
Aphasia	6
Aphonia hysterica	2
Athetosis, functional	2
Ataxic paraplegia	3
Atrophy of shoulder muscles	3
Atrophy of arm and leg from joint disease	1
Arteritis chronica	10
Brachial Spasm	6
Brown-Séquard paralysis	2
Bulbar paralysis	2
Cerebral hyperæmia	2
Cerebral tumor	3
Chorea	75
Coccygodynia	1
Concussion of spine	1
Crutch paralysis	1
Congenital contraction of muscles	1
Defect of speech	2
Debility	3
Dementia senilis	2
Dementia, terminal	1
Dyspepsia	17
Endarteritis deformans	3
Epilepsy	94
Epilepsy, cortical	7
Erb's paralysis	3
Exophthalmic goitre	9
Facial paralysis (Bell's palsy)	12
General paresis	8
Headache	298
Hemiplegia	46
Hydrocephalus	2
Hysteria	39
Hystero-epilepsy	5
Hypochondriasis	18
Hydrosis of extremities	1
Idiocy	8
Imbecility	9
Insomnia	20
Lateral sclerosis	5
Lead palsy	14
Lithæmia	4
Locomotor ataxia	26
Lumbago	3
Malaria	8
Mal-development of brain	29
Mania	5
Melancholia	11
Menière's disease	5
Meningitis, basilar syphilitic	2
" " tubercular	3
Migraine	22
Monoplegia, brachial	5
" crural	2
Multiple sclerosis	15
Myalgia	9
Myelitis, transverse	11
" general	11
Neuralgia, trigeminal	151
" occipital	10
" brachial	12
" crural	6
Neurasthenia	209
Neuritis of circumflex n	7
" " brachial plexus	32
" " median n	3
" " musculo-spiral n	34
" " peroneal n	2
" " radial n	2
" " ant. tibial n	4
" " ulnar n	1

Diseases.	No. of Cases.
Neuritis multiple, alcoholic	6
" " arsenical	1
" " diphtheritic	2
" " scarlatinal	1
Neuroses, traumatic	6
" toxic, from tobacco	5
" vesical	2
" climacteric	6
Nocturnal terrors	2
Occupation neuroses	9
1 cigarmaker's, 1 plasterer's, 1 pianist's, 3 telegraphers', 2 writers', 1 sewing.	
Ophthalmoplegia externa	1
Pachymeningitis, cerebralis	3
" cervicalis	1
Paralysis, of third n	3
Paralysis agitans	23
Paranoia	9
Paraplegia	1
Pons lesion (alternating paralysis)	1
Pseudo-musc. hypertrophy	1
Psychoneuroses from sexual excess	8
Progressive muscular atrophy	4
Rheumatism	28
Sciatica	42
Senility	12
Somnambulism	3
Somnolence	1
Spasm of head (rotary)	1
Spasm (habit)	2
Spinal irritation	2
Syphilis of the brain and membranes	36
Tic convulsif	2
Tic douloureux	5
Tinnitus aurium	4
Torticollis	2
Tremor post-hemiplegic	3
" senile	4
Unclassified	82
Vaso-motor neuroses	2
Vertigo	23

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