An atlas of the differential diagnosis of the diseases of the nervous system : analytical and semeiological neurological charts.

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AN ATLAS

OF THE

DIFFERENTIAL DIAGNOSIS

OF THE

DISEASES OF THE NERVOUS SYSTEM

ANALYTICAL AND SEMEIOLOGICAL NEUROLOGICAL CHARTS

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To
Thomas Hun
a loving father
a learned physician
a man of wisdom and wit
this book is dedicated
in most grateful remembrance

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PREFACE

The diagnosis of diseases of the nervous system is generally regarded by medical students as one of the most difficult subjects in their course of study. It is so difficult that many students become discouraged and after a few attempts make no strong, continued effort to master it and, perhaps in consequence, physicians generally are weaker in this than in other phases of their work. In the hope of making this task less difficult for both physicians and students this book has been written. If the student can be taught to make the diagnosis of these diseases with comparative ease, it may happen that he will be led to undertake those further studies in the finer anatomy and physiology of the nervous system, which are essential for a full understanding of this difficult but fascinating department of medicine.

A careful physical examination and history of the case, as complete as can be obtained, are, of course, the essential basis of every diagnosis; but the commonly employed method of comparing the combination of symptoms thus obtained in any case with the various syndromes characteristic of the different diseases until a similar combination can be found, is not altogether satisfactory. More scientific and instructive is the analysis of each important symptom and the consequent ascertaining of the disease which must cause it under the circumstances (the other symptoms) existing in any individual case which may present itself.

In spite of its apparent complexity, the diagnosis of nervous diseases lends itself better than that of the diseases of most of the other organs to exact pathological analysis. Just as a chemist in analyzing a substance of unknown composition by a series of appropriate tests eliminates from consideration one group of chemical bodies after another until he finally discovers its class and name, so the neurologist subjects a patient to one test after another in definite sequence. As the result of each test he throws out of consideration one or more groups of diseases and assures himself that he has to do with a disease belonging to another definite group. With each successive test the number of diseases constituting a group becomes less, until finally one definite individual disease stands revealed among the few most closely related to it by a comparison of the remaining symptoms characteristic of each, which are given in the final abstracts. This analytical method is used, I think, by all great teachers of neurology in demonstrating cases of disease before their classes of students. It is the crystallization of this teaching into the tabular form which this book attempts to present.

In using this book for diagnostic purposes it is important that the "Introduction to the Diagnostic Charts" on page 119 should be carefully studied. By means of these charts it is possible to diagnosticate easily and rapidly any disease

of the nervous system and to localize the lesion, when any lesion exists. If the examiner makes a mistake at any point, the next step in the process or the abstract of the other symptoms of the disease will probably show him that he is in error and that it is necessary for him to retrace his steps.

For the sake of completeness certain trophic diseases are included, which, although causing a number of functional disturbances in the nervous system, are not really nervous diseases.

As might naturally be expected, the same disease, in so far as it presents many symptoms, appears a number of times in the different charts and even in the same chart; so that, in order to get a more complete idea of its symptomatology, it is essential that the different abstracts of it should all be read. To facilitate this, cross references by numbers within brackets are placed in the text.

Many diagnostic and technical terms are used which may not be familiar to the student; therefore these terms are classified, defined and their significance stated, as far as it is known to the author, in a series of semeiological charts preceding the diagnostic ones. Cross reference to these terms also is facilitated by the numbers within the brackets. A very full index, in the preparation of which the author has received much assistance from his friend, Dr. Dawes, also serves this same purpose.

The peculiar characteristic of this book on diagnosis is that it gives to the student or physician a key by which, in a comparatively easy manner from one or more important symptoms, he can arrive at a diagnosis. It also has the advantage that it divides the diseases into groups, the members of which have a definite relationship with each other; so that in the process of using the charts the student is constantly catching glimpses of the natural relationships between the different diseases of the nervous system. Although the symptoms of different diseases have often been contrasted in tables of parallel columns, in no other book, known to the author, has the subject been presented as it is here, and this must be his excuse for publishing it and for any defects which it may show, as there was no model which could be followed in preparing it.

In the preparation of the charts the author has received valuable suggestions and aid from several friends and especially from Drs. Mosher, Gordinier and Archambault, while for the plates he is greatly indebted to Drs. Streeter and Hawn. To these, his present friends and former students and assistants, he gratefully acknowledges his indebtedness and returns his thanks.

It is very gratifying to the author that the first edition of two thousand copies has been exhausted in eighteen months, giving him an opportunity of making a complete revision of the text, and of adding a few plates and also introductions both to the semeiological and to the diagnostic charts, which seem to him to add much to the value of the work.

The criticisms of Dr. Mosher and his untiring aid in putting both editions through the press are large factors in whatever success the book may have.

HENRY HUN.

Albany, N. Y.,
August 1, 1914.

PART I

SEMEIOLOGY

THE EXAMINATION OF PATIENTS

AND A

PHYSIOLOGICAL AND PATHOLOGICAL ANALYSIS

OF THE

RESULTS OBTAINED FROM SUCH EXAMINATION

AN ANALYSIS OF THE

SUBJECTIVE AND OBJECTIVE SYMPTOMS OF DISEASE

INTRODUCTION TO THE SEMEIOLOGICAL CHARTS

The diagnosis of nervous diseases, if it is to be at all satisfactory and accurate, must be based on anatomy and physiology. The practitioner is supposed to have some acquaintance with these subjects, and the curriculum of the medical college is so arranged that the student is taught them before he commences clinical work. It seems, however, desirable to make here an attempt to present a brief, but comprehensive, outline of the physiology of the nervous system, including some statements as to its anatomy, which latter can be supplemented by an inspection of the plates at the end of the book.

The fundamental element, or unit, of the nervous tissue is the *neuron* (461-4) a cell with many processes projecting from it; some short and branching (dendrons), one (rarely two) which often extends a long distance and usually becomes the axon of a medullated nerve fiber, and which, in some cases, gives off a few collateral branches. Both axons and dendrons are composed of delicate fibrillae which pass directly without interruption through the cell body. Of these neurons, varying in form and size and supported by the delicate framework of the neuroglia, the entire nervous system is composed.

The fundamental physiological characteristics of the nervous tissue are *excitability* and *transmission:* the power of receiving an excitation and transmitting it from one end of the neuron to the other and of transmitting it to other neurons with which the first is in anatomical and physiological relationship or contact. By its dendrons the nerve cell receives nervous impulses and by its axon it sends out its own impulses. There is experimental evidence which tends to prove that the activity of a nerve cell is the result of chemical reactions (consumption of chromatophilic substance, etc.), while the conduction along nerve fibers is mainly a physical process. The transmission of energy from one neuron to another in contact with it seems to depend upon differences in the tension of this energy in the two neurons. The cellular activity is, therefore, easily exhausted, while the activity of the nerve fiber is not easily exhausted.

Of the numerous forces and forms of energy in the world only a portion can be perceived by man. It is certain that some animals perceive things imperceptible to him. The various forms of energy in nature cannot act directly upon the nerve to produce sensory impulses, but intermediate organs, "end-organs," are necessary to transmute the external energy into nervous energy. In virtue of chemical changes the potential energy stored in the end-organ becomes active; the inciting cause of this being the external or foreign irritation. There are, doubtless, many forms of energy in the world which cannot be perceived, because there are no suitable end-organs to bring about this transmutation. Sometimes this can be accomplished by adding to the end-organs some mechanical contrivance suitable to bring about this transmutation; as for instance, the fluoroscopic screen for X-rays. The universal ether is doubtless in vibration far beyond the limits of about four hundred million million per second, which constitute for us the color red, and about seven hundred and sixty million million, which constitute violet; and indeed we have reason to believe that the ultra-violet rays have some effect upon our body, but beyond the above limits the vibrations of the ether are not recognizable by our eye and brain. The absence of a sensory end-organ limits the number of perceptions and consequently the content of consciousness, but this content is, or can be, much larger at the present time than in times past. Many new forces (X-ray, etc.) are now perceptible by the organs of sense which were before inperceptible. The sensory apparatus also is not absolutely perfect. Things moving very rapidly cannot be seen. The spokes of a rapidly revolving wheel cannot be seen.

Cutaneous and muscle-joint sensation (Chart VIa). The surface of the body and the cavities connected with it contain organs: the terminal organs of sense (the sensory "end-organs"), which bring the body into connection with some, but probably far from all, of the forces of nature, and which "end-organs" are so constructed as to transmute physical forces (light in the eye; sound in the ear; heat, cold, touch, pressure and pain in the skin, etc.) into nervous excitations in the terminal filaments of the peripheral nerves. The skin contains many of these isolated terminal sense organs and, therefore, sensibility is not spread uniformly over the skin, but is located in individual points. From these points of greatest sensibility, its acuteness diminishes concentrically. In every square centimeter of skin there are, on the average, 12 to 13 points for cold, 0 to 3 for heat, and 24 for pressure, impressions; although these figures vary very greatly for different parts of the skin, the points being most numerous on the finger tips and fewest on the back. Most observers maintain that there are distinct points also for painful sensibility. Where the skin is stretched over bone (the malleoli), sensation is less distinct and is more distinct where the skin is hairy; a point for tactile sensibility being situated at the base of most, if not of all, hairs. There may be a delay of several seconds in the conduction of painful impressions, and there may be a summation of painful impressions; so that with repeated pin-pricks the pain becomes more acute. After section or injury of a nerve, the anesthesia and analgesia are never so extensive as is the area of distribution of the nerve, and sensibility often returns before the regeneration of the nerve has taken place. This is partly due to peripheral anastomoses and partly to recurrent fibers of the sensory nerve.

Head and his colleagues, after much experimental work on the subject, arrived at the following conclusions:

There are in the peripheral areas three kinds of sensibility, due to there being three different kinds of nerve fibers supplied to each area.

1st. *Deep Sensibility.* Muscle sense, pressure sense, pressure pain and localizing sense. This sensibility is conveyed by sensory nerve fibers, more or less deeply situated, beneath the skin and usually running with the motor nerves. When the motor nerves of the muscle tendons are cut, these forms of sensibility are lost.

2nd. *Epicritic Sensibility.* Tactile sensibility for slight impressions, form and space sense, sense of moderate, not extreme, temperatures (22° to 40° C.) and the precise localization of pain and temperature sense. This area of sensibility is very constant for each individual nerve. The restitution of this form of sensibility is very slow and is not complete until after several years.

3rd. *Protopathic Sensibility.* Pain and sense of extremes of temperature (below 22° or above 40° C.). These symptoms are accompanied by paresthesiae and a false localization. This form of sensibility is best tested on the periphery of the affected area, where the anesthesia is not complete, or over the whole area while regeneration is taking place. The restitution of this form of sensibility is relatively rapid (7 to 10 weeks).

These researches of Head are of great interest and value and have attracted much attention and discussion, but they are not, in their entirety, accepted by all neurologists.

Sensory impulses of all kinds are carried to the central nervous organs by the sensory nerves. Of these, the spinal enter the cord through their cell bodies in the spinal ganglia and through the posterior nerve roots (Fig. 26); while the fibers of the trigeminal, the great cranial nerve supplying sensation to the face, after passing through their cell bodies in the Gasserian ganglion, enter the trigeminal sensory nucleus in the pons (Fig. 19). The fibers from the posterior nerve roots, on entering the spinal cord, are sorted according to their physiological function into three great parts (Fig. 26). One part, which conveys tactile, pressure and muscle-joint sense impressions, ascends, mainly without decussating, in the posterior columns to the nuclei of the columns of Goll and Burdach, and thence is continued by a new set of neurons (the internal arcuate fibers), which decussate and pass through the median lemniscus (Figs. 20-3) to the optic thalamus, whence it is continued, also by another set of neurons (relays), to the parietal cortex. The second part, which conveys impulses for co-ordination, passes to the cells of the column of Clarke and

thence, mainly without decussating, through the direct cerebellar tract in the outer part of the lateral column and through the restiform body to the cerebellum. The third part, which conveys temperature and painful impressions, passes through cells in the posterior horn, decussates in the central gray matter of the cord and passes upward in the antero-lateral column through the spino-thalamic tract and lateral portion of the formatio reticularis to the optic thalamus, and thence to the cortex.

A destructive lesion either in the terminal end-organ, or at any point of these sensory tracts or neurons, causes a corresponding paralysis of sensation (anesthesia); while a slight, irritative lesion may cause hyperesthesia, paresthesiae or pain in the distribution of the nerve.

In addition to the anesthesia, which occurs in organic disease of the nervous system, there is an anesthesia which occurs in hysteria: hysterical anesthesia. This hysterical anesthesia, occasionally but rarely, involves the organs of special sense. It more commonly involves cutaneous sensibility and then the anesthesia is not limited to the distribution either of a peripheral nerve or of a nerve root. It may instantaneously disappear. It may recur in the same place, or in some other locality. It does not prevent the use of the part in performing acts, in which sensibility is essential. Upon excitation of the anesthetic parts vascular reflexes occur, which is never the case in anesthesia due to organic disease, peripheral or spinal. It is evident that this hysterical anesthesia is the result of imagination or delusion. It is purely psychic.

Pain (374) is an unpleasant sensation which never occurs in health, but only when the body is injured, either mechanically or chemically. It is a signal of warning that the body needs protection. Its intensity depends not only upon the intensity of the mechanical or chemical irritation, but also upon the condition (inflammatory, etc.) of the peripheral nerves and of the cerebral cortex. It is more intense when accompanied by fear and apprehension. An unexpected wound is less painful than an anticipated one. Pain is often associated with the allied perceptions called "paresthesiae" (375) which at times precede, at times accompany, and at times follow, the pain, and which are usually of central origin and are due to irritation of the sensory fibers at some part of their course through the central nervous organs.

Gustatory sensation (Chart VIa). The mucous membrane of the mouth (in addition to the terminal organs for tactile, pressure, thermic, painful, etc., impressions) contains also the terminal organs of the nerves of taste: the taste buds or bulbs, so called from their form, embedded in the epithelium of the mucous membrane of the mouth, especially of the tongue. Excitation of these taste bulbs gives rise to four distinct gustatory sensations: sweet, acid, salty and bitter, to which may, perhaps, be added alkaline and metallic. Many so-called tastes are really a combination of gustatory and olfactory sensations. The nerve fibers arising from the taste bulbs on the posterior portion of the tongue pass by the glosso-pharyngeus nerve in a direct manner through the petrous ganglion to its nucleus in the medulla, whence they ascend with the other sensory fibers of the lemniscus to the optic thalamus, and thence to the cerebral cortex posterior to the gyrus hippocampi (Fig. 16); while the nerve fibers arising from the taste bulbs on the anterior portion of the tongue pass at first in the lingual nerve and, soon leaving this, form the chorda tympani, which joins the facial nerve and runs with it through the Fallopian canal to the geniculate ganglion. Here the fibers divide; a part continuing alongside the facial nerve and forming the nervus intermedius, which runs to a nucleus in the medulla close to the glosso-pharyngeal nucleus; while the rest of the fibers run through the petrosal nerves and join the fifth nerve and pass to the Gasserian ganglion (Fig. 36), and thence to the cerebral cortex (Fig. 16). A destructive lesion at any point of this course will cause unilateral loss of taste (ageusia). When the lesion is in the Fallopian canal, the ageusia may be associated with facial paralysis on the same side.

Olfactory sensation (Chart VIa). The mucous membrane of the nose, analogous to that of the mouth (in addition to the terminal organs for tactile, thermic, pressure, painful, impressions), contains also the terminal organs of the nerves of smell. The nerves terminating in these organs pass upward through the cribriform plate to the olfactory bulb, and thence backward through the olfactory tract: some to the anterior perforated space and sub-thalamic region

(olfactory reflexes) and some to the cortical center for smell in the gyrus hippocampi (olfactory perceptions) (Fig. 16). Each olfactory bulb is connected through the anterior commissure with both cortical centers.

Hearing (Chart VIa.) The terminal organ for hearing is the organ of Corti in the cochlea, within the petrous portion of the temporal bone. In this organ there is a long series of vibratory structures of unequal lengths; so that on them can be reproduced every possible tone with its over-tones or harmonics. The auditory nerves terminating in this organ pass to the ventral and dorsal auditory nuclei in the pons. From these nuclei fibers pass upward, some decussating and some not, through the lateral fillet to the corpora quadrigemina, and thence through the sub-lenticular region of the internal capsule, posterior to the fibers for cutaneous sensibility, and reach the cortical auditory center in the anterior part of the superior temporal convolution (Fig. 15). A destructive lesion of one auditory nerve will cause unilateral deafness on the same side, but a lesion of the tract connecting the sub-cortical with the cortical centers, since this tract contains both crossed and uncrossed fibers, will not cause any deafness. Deafness results only from a bilateral central lesion affecting the corpora quadrigemina or the sub-cortical tracts described above. Even destruction of the auditory cortical center in both hemispheres does not seem to cause complete deafness.

Sight (Chart VIa). The terminal organs for sight are the rods and cones in the retina within the eyeball. The rods seem to be concerned in seeing in dim, the cones in bright, light. They vary in relative numbers in different animals, according as they roam by night or by day. From these structures start the terminal filaments of the optic nerves, which run backward from the eyeballs. In the optic chiasm the fibers from both maculae luteae and from the nasal half of each retina decussate; so that in the left optic tract are collected all the fibers from the left half of each retina (right visual field) and those from both maculae luteae; while in the right optic tract are collected all the fibers from the right half of each retina (left visual field) and those from both maculae luteae. The fibers of the optic tract on each side terminate in the external geniculate body, the pulvinar and the anterior quadrigeminal body of the same side, and are thence continued through the posterior portion of the internal capsule and the fasciculus of Gratiolet to the lips of the calcarine fissure on the median surface of the occipital lobe of the same side (Fig. 37).

A destructive lesion of the optic nerve causes blindness of the corresponding eye, but a lesion of any portion of the optic tract, or geniculate body, or fasciculus of Gratiolet, or of the lips of the calcarine fissure will cause homonymous hemianopia of the field of vision of the opposite side; while a lesion of the central portion of the optic chiasm will cause binasal hemianopia.

Internal or general sensation. In addition to these sensory impulses which convey to the brain excitations from the special sensory organs and hence from the external world, there are others which come from the different organs of the body and, in case they reach the cortex, give rise to what is called internal or general sensation. Some of these internal excitations remain entirely peripheral and affect mainly the blood vessels; others reach no further than the spinal cord or ganglia at the base of the brain and incite those automatic acts which preserve the nutrition and the life of the individual; while others reach the cerebral cortex and at times affect profoundly the processes taking place in it. These general sensations have much influence on a person's emotions, moods, thoughts and actions. Hunger may entirely alter the normal acts of a man or beast.

Such internal or general sensations are for the most part ill-defined and ill-localized. They seem to depend upon the blood supply and upon the activity of the different organs and upon the state of contraction of the hollow organs; and they appear to have much to do with our feeling of comfort or discomfort, which latter may amount to even severe pain. The sensation of hunger seems to be caused by contraction of the empty stomach, and the various colics by contraction of the circular muscular fibers of the intestine, the ureter or the bile duct. But the best understood of all these internal or general sensations is the composite one called muscle-joint sense, which is mainly made up of impulses from the muscle and its tendon and the articu-

lating surfaces and also from impulses from the skin and other tissues in the neighborhood, as these are stretched or relaxed in motions of the joint. The muscle-joint sensory conduction we have already considered in connection with the conduction of tactile sensory impulses. To these internal sensations must be added also, probably, the cortical innervation feelings (see later) inasmuch as a person often feels that he is moving, or feels that he knows the position of, a paralysed or even amputated limb.

Sensation (Chart VI)

When these various impulses have passed along the various tracts and have traversed, and been interrupted by, several masses of gray matter, they reach the sensory area of the cerebral cortex and there give rise to a new form of energy called sensation. That is to say, a physical force (as for instance, ether in rapid undulation) is converted in a terminal organ into nervous energy, and as such, having traversed the sensory tracts, reaches the cerebral cortex. It is there transmuted into a new form of energy (as for instance the sensation of light). The sensation of light takes place in the brain, not in the eye, and has no similarity to the undulations of ether from which it normally originates, and it may, indeed, be caused not only by these, but also may originate, in perfect darkness, from mechanical irritation of the eye (as by pressure from the finger upon the eyeball) or of the optic nerve. Sensation is, therefore, rather a symbol than a picture of the external object, with which by experience it is associated.

Sensation is thus a special, individual force, similar to electricity, light, etc., which is produced in the cerebral cortex and which has its special, individual characteristics. A complex manifestation of this force constitutes consciousness and personality. Sensations originating from the different organs of sense are located, as we have seen, in different and special portions of the cortex (Figs. 15 and 16) and do not at all resemble the external phenomena causing them. A clap of thunder and a flash of lightning are very different external phenomena, but the reactions in the cortex, which constitutes these sensations, probably vary in topography rather than in quality. We know nothing more of the essence of this form of energy, which we call sensation, than we do of the essential nature of electricity, or of contractility in the muscle fiber or in the amoeba. We know something of its effects and we know something of the locality of the cerebral cortex in which it occurs (Figs. 15 and 16) and that it is very dependent upon abundant blood supply and that it must result from chemical actions taking place in the cortex. Conscious sensation, probably occurs only in those animals which possess cerebral hemispheres.

Sensation and all other forms of mental activity are absolutely dependent upon a fairly healthy cerebral cortex and a fairly abundant blood supply to it. When the cerebral hemispheres in an animal are removed, or when the cerebral cortex in man is entirely or mainly destroyed by disease, or in a child the hemispheres are absent or very defective, or when the blood supply is cut off from the cerebral cortex altogether or in large part, then sensation, perception, memory, thought, emotion (and its corporeal expression), ethics, association of ideas, voluntary motion, inhibition, intelligence, personality and consciousness are all lost.

Sensation is the simplest manifestation of consciousness (see later) or cognition. For its production a certain degree of intensity of the nervous impulses is essential, below this point of intensity the cortex may be in activity, but sensation will not result; the activity will be sub-conscious. A series of these slight impulses quickly repeated may by summation cause sensation. There is, therefore, a minimum of intensity necessary for sensation; just as electricity passing through a wire must have a certain intensity before the wire glows and light is produced. There is also a maximum beyond which, no matter how great the irritation, there is no increase of sensation, but rather a diminution from exhaustion of the nerve cells. Between this minimum and maximum point, sensibility increases, or diminishes, not continuously, but by little steps; a definite ratio to the stimulus (Weber's law).

Furthermore, a weak or moderately strong excitation may reach the cortex at a time when other portions of the cortex are in such strong excitation that this weak irritation may produce no sensation, but remain sub-conscious. The line between the conscious and the sub-conscious cannot be sharply drawn.

Perceptions and Concepts (Chart VI)

A perception consists of a combination of sensations, which are obtained from various sensory end-organs, but all of which proceed, usually simultaneously, from the same external object. A perception of an apple is composed of several sensory impulses: of visual sensations from the retina, representing the outline and markings and color of the apple; of muscle sensation from the ocular muscles, representing its distance from the eye, its position in relation to other objects and to some extent its form; of tactile sensations from the hand, representing its form, firmness and texture; and of gustatory sensations from the mouth, representing its taste. The various physico-chemical changes, thus set in activity in the cortex, combine to produce the full perception of the apple. For a full and complete perception, consisting as it does of so many elementary sensations, quite an appreciable time, or frequent repetition, is needed. The development of a perception is found by experiment to proceed from generalities to details. A combination of the full perceptions of many apples, each resembling and at the same time in some respects differing from the other, produces the idea or *concept* of an apple, with which is associated its written and spoken name and any other experiences or knowledge which have become associated during our life with apples. (See also under *Associations.*)

Perceptions occur in the cerebral cortex in immediate proximity to the cortical termination of the corresponding projection fibers. Each cortical center consists of a smaller portion, in which the projection fibers terminate and a larger portion, in which perceptions take place and in which their memories are stored. Thus, the optic fibers terminate in the lips of the calcarine fissure, while the rest of the median and convex surface of the occipital lobe is devoted to optical perceptions and memories (Figs. 15 and 16). When sensations only, but not perceptions, can occur, as when that portion of the center in which the projection fibers do not terminate is diseased, the condition is called in general *agnosia*. When there is a failure of tactile perceptions the condition is called *astereognosis;* in failure of optical perceptions *soul-blindness*, or *psychic blindness*, and in failure of auditory perceptions *soul-deafness*, or *psychic deafness*, or *auditory or sensory aphasia*. When that portion of the cortex in which the sensory fibers terminate is diseased, both perception and sensation are abolished.

Emotions (Chart III)

Certain activities of the brain are accompanied by feelings of pleasure or discomfort or even pain, and usually also, if these feelings are moderately intense, by changes in the functional activity of the internal organs, especially in the circulatory and respiratory systems, but also in the alimentary canal and in the other viscera and glands of the body. These feelings are due in great part to the internal or general sensations. When the bodily functions are disordered we have a general feeling of discomfort and when all is working well we have a sense of buoyancy and exaltation; all moves smoothly without friction, as in a well oiled machine. These internal sensations, as was mentioned on a previous page, are ordinarily the dominant factor in our feelings and emotions and greatly influence also our voluntary actions, which for instance may be altogether different in a state of hunger from those in a state of satiety. Indeed the internal sensations, such as hunger, etc., are very often themselves the cause of extensive voluntary acts, which have for their aim relief from this sensation. As these internal sensations vary from time to time, our moods change, and perceptions, which at one time are pleasant, may at another time be unpleasant. Irrespective of our moods, however, some perceptions are almost always pleasant, others are not. Things which tend toward the preservation and health of one's self and his family are usually pleasant, and vice versa. Perceptions to which we have become accustomed are usually pleasant, and even unpleasant perceptions by frequent repetition at times become bearable and even pleasant.

Not a few perceptions are accompanied with relief of discomfort, as when hunger is assuaged, or we accomplish something desired or in some way contribute to our well-being or success and thus give pleasure; while other perceptions act in a contrary manner. These feelings of pleasure and pain may be due in part to the intensity of the sensation or perception, in part to heredity, as a result of evolution in case of objects desirable for the health of the body; but in greater part

to associations (see *Associations*) with previous similar perceptions, and in greatest part with the feeling of satisfaction or dissatisfaction with the result of one's actions.

Sensations of moderate intensity are usually pleasant; while sensations of very great intensity, which produce abnormally strong reactions in the nervous tissue, are usually unpleasant. Sensations originating from sets of vibrations having a simple ratio to each other are usually pleasant, while those which have a complicated ratio are usually unpleasant. Foods which nourished our ancestors usually taste good to us. The child probably acquires a taste for sweet things from the sugar in its mother's milk. Most of our pleasant and unpleasant sensations are the result of our education. They are, therefore, much more pronounced in adults, especially educated ones, than they are in children. A perception which is associated with, or followed by, pleasure or pain will always, or for a long time, as often as it occurs actually or in memory, be accompanied by a pleasant or painful emotion, whether the memory of the original pleasant or painful result associated with it is present in consciousness or not.

Mankind does not find itself in this world with all its needs and wants satisfied; on the contrary everyone must acquire food, clothes, habitation, warmth and a hundred other necessities. A man who sees his neighbor with something good, which he has not, desires it, or something similar. These wants and desires are the great, almost the only, incentives to voluntary action. When this voluntary action results in success we have in it our greatest pleasure and when it results in failure, our greatest unhappiness. All things connected with our success receive an associated emotion of happiness; while those things connected with our failure receive an associated emotion of grief.

In these various ways a certain number of our perceptions have associated with them an emotion (204), or tone, of pleasure or pain, greater or less, and a series of such emotions, or one long continued, will make us happy or unhappy for a considerable length of time and will constitute what we call our "mood."

In certain abnormal states of the cerebral cortex (exhaustion, circulatory irregularities, poison and other less well known disorders) the emotions become dissociated from the ideas with which they are normally associated; so that all cerebral activity is accompanied by one emotion; in some cases, sadness; in others, fear; in others, joy and in others apathy or absence of all emotion. An emotion is often so strong and so occupies the patient's consciousness that it is impossible, or nearly so, to attract his attention.

Memory (Chart III)

When perceptions take place, chemical changes are occurring in a definite portion of the cerebral cortex, which not only produce the perception, but also leave thereafter a permanent alteration in the cortex. The force derived from the chemical changes taking place in the cortex during an active perception may result in a structural, physical or chemical change in the nervous elements, or more likely in the storing in them of potential energy, which can be liberated and become actual later. Memories are dynamic changes in nerve cells and fibers which reduce the resistance to subsequent similar impressions or excitations. Certainly, a definite change is brought about which registers a permanent memory of the object perceived and subsequently this memory can be latent (sub-conscious), or active (conscious), from time to time. Consciousness, the actual perception of an object and its associated active memories (active attention), is a very exhausting, energy consuming process for the cerebral cortex. Subconsciousness, the preservation of memories, not present in consciousness, is not exhausting to the cortex, even though the memories be preserved for many years.

In virtue of this change in the cortex, a memory of this perception always results from irritation of this altered cortex. This memory may be aroused, or enter into cognition, by the external force which originally caused it and, then, the object will be *recognized* (re-known), because the actual perception corresponds perfectly with its memory; or the memory may be aroused by way of those association fibers which it had previously set into activity. Memories become associated with each other in accordance with the relationship of the objects causing

them, as the result of our experiences with these objects. In perception, then, a trace of the cortical excitation remains in the cerebral cortex as a memory, in a sense analogous to the persistence and after image in the retina after strong excitation (looking for some time at a bright light).

These memories are, however, very different qualitatively from the original perceptions. The former have no actuality. Even though they may be at times very vivid, they never seem real to a normal personality.

The whole cortex of the brain is in great part a huge store-house of memories. These memories are grouped together; so that those which are derived from the same organ of sense lie together in the cortex. This localization of memories has been worked out with great care and is to a considerable extent known. It is shown in Figs. 15 and 16. A local cortical lesion may thus produce a loss of a group of allied memories.

All memories are sensory in character with exception of one doubtful group. This group consists of memories of so-called "innervation feelings." When a muscle is contracted the person to whom it belongs has a feeling of this contraction and can estimate its strength. This feeling in called an " innervation feeling " and its memory is stored away in the cortex of the anterior central convolution and of the neighborhood in front of it. This innervation feeling seems to be essential for the voluntary performance of the corresponding act. When, in consequence of a cortical brain lesion in the area in which these memories are stored, a person loses the power of performing certain acts, he often says, " I have *forgotten* how to do it." These innervation feelings and memories do not obtrude themselves strongly into our consciousness. They have rather to be sought for, but they usually can be observed, except in those actions which follow very rapidly upon the perception, or memory, causing them. There are many physiologists and psychologists, however, who question whether there are any so-called innervation feelings or memories in consciousness. However that may be, whether true innervation memories exist or not, the function of this cortical area is an actuality and whenever a portion of the motor cortex is sufficiently excited by a perception, or a memory, from the sensory cortex, a so-called voluntary, or association, action results. (See *Voluntary Movements.*)

Association

The essential physiological characteristics of nervous tissue are: first, its excitability, its reaction to stimulation by the discharge of nervous energy stored within it; and second, its transmissibility, this nervous energy, whenever produced, does not long remain localized, but tends to pass along nerve fibers, throughout its own neurons and to other neurons. The channels along which it will pass depend upon the anatomical arrangement of the fibers. In consequence of heredity and evolution, certain channels are easier for the passing of this nervous impulse than are others. This is especially true of certain reflexes present at birth, such as breathing, sucking, etc. Other channels are made easy later in life by the constant passage of impulses along them. The more frequently an association fiber is used the better conductor it becomes. The way that has once been traversed and that has often been traversed becomes the easiest way. It is the way of least resistance and it is a universal rule, whether it be a foot-path, or a conductor of electrical or of other force, or a nerve fiber or cell, that the way of least resistance is the easiest way: the way usually followed. When not used for a long time, like a deserted garden path, the channel may be obliterated and the association lost.

When a perception occurs, impulses radiate out along the association fibers from that portion of the cortex which produces it. If at the same time another perception, or a vivid memory of a perception received a moment before, takes place in another portion of the cortex, the association fibers connecting these two portions of the cortex, where perceptions are occurring, or have just occurred, being acted upon at both ends, will convey impulses to and fro more readily than the other association fibers. The longer and more frequently the association fibers are traversed by these impulses the better conductors do they become and these two perceptions become more and more easily excited the one from the other. The activity

in the cortex does not long persist; so that when the associated idea is in consciousness, the original perception which awoke it is already, or soon will be, sub-conscious. Yet they are firmly associated together; so that whenever in the future one enters into activity it may excite the other. Thus, association between perceptions of the events and objects received simultaneously, or immediately before or after each other, are formed in a never-ending stream and the events and objects are considered as contemporaneous and often as related to each other. Subsequent experiences may verify and strengthen some of these associations and may disprove and unmake others. Associations with any one perception may be, and usually are, extremely numerous. There is also an association of words as well as of perceptions, and the associations of words have no necessary relationship to the associations of the objects which they represent. Associations may be at first very imperfect and very difficult to form, but with repetition and practice become easy. The work of a child in school is difficult until by repetition he has learned thoroughly his lesson. Then the recitation is easy. Addition, subtraction, etc., are at first performed slowly and with difficulty but later, in consequence of frequent repetition, rapidly and easily.

When a number of perceptions are produced which are very similar and yet show more or less individual variations, as for instance perceptions of men or dogs, from a comparison of them and of memories of others, more or less similar, a *concept* or *idea* of a man or a dog is formed, which includes all the individuals. From many examples of individual freedom of action, the abstract idea, or *abstraction*, of liberty is formed. A great many such abstract ideas are gradually formed and this process is facilitated by the use of language. But each idea is the result of experience: the result of a conglomeration or generalization of one or more perceptions and their associations, and, by the aid of language, is given a name. It has been said that "we can understand only so much of an abstraction as we know individual cases which sustain it." Thinking and reasoning are much simplified and made more rapid by the employment of these abstractions.

Ethics

Inasmuch as the sensory and motor areas of the cortex are intimately connected together, some perceptions lead to voluntary action, which may result in pain, either directly as physical pain, or indirectly as mental pain, the result of punishment or condemnation; so that the action and the perception which led to it will become associated with these unpleasant sensations or perceptions, and these associated unpleasant sensations will tend to restrain further similar actions. Such acts, bringing with them a penalty, will be called wrong and there will gradually be formed a large number of associations which will be identified with the ideas of punishment and condemnation and which tend to prevent the performance of wrongful acts; just as another combination of associations which have become associated with pleasure, reward or praise, will be associated with good or right. A person's idea of what is right or wrong will depend upon his education, the result of experience and of teaching, and is the basis of emotions and ethics, and of that ill-defined function, the so-called *conscience*, and may evolve into very elaborate and very controlling feelings and habits of thought. According as education has developed one set of these associations rather than the other, a good or bad character, not from his own but from the community's standpoint, is formed. These ethical ideas can of course be imparted from one person to another by language and, indeed, frequently are so imparted, but such ethical ideas are rarely so firm and convincing as those obtained from experience.

Concentration and Attention

It seems to be a general law in the physiology of the nervous system that when there is a strong activity in one part, the activity of the rest of the nervous system is inhibited. Thus, reflex activity can be inhibited by strong pain; and the reflex activity of the spinal cord is more or less inhibited when the brain is in activity. In the brain itself, when a portion of the cortex or a group of nerve cells, is in activity, the activity of the other cortical areas, as well as that

of the lower centers, is inhibited. The stronger the local activity, the greater and more extensive will be the general inhibition, and the more this active portion will have a free and uninterrupted field. Naturally, consciousness remains limited to this strong activity for a long time. When an unusual or very vivid perception or idea is in consciousness it occupies the center of the stage. Consciousness is limited to this one vivid idea and its associations; so that milder activities occurring in the cortex at the same time, which should produce, ordinarily, perceptions and associations, remain sub-conscious. This phenomenon is called *concentration* and is a very important function in nervous physiology. When the cause of this concentration is a perception, in addition to this inhibitory influence, impulses from the active sensory cortex radiate to the motor cortex and out to the perceiving organ and cause a change in its musculature in the form of greater tension, tonicity, change in its position, etc., which local change heightens the power of the organ for the perception of stimuli. Concentration is only another name for attention and has been regarded as an effort, and an expression, of the will or will-power, but the primary and essential factor seems to be the cortical activity. (See *Will.*) Not infrequently the concentration is centered about an unpleasant idea, from the thought of which we vainly try to escape; yet it is forced upon our attention and we cannot free ourselves from it in spite of every effort of our so-called will. We are at times in a state of "expectant attention" in regard to some possible perception, which state we cannot prevent, try as we may.

Reverie and Thought

The steady stream of perceptions originating from the excitation of the various sensory organs is constantly awakening associated memories, and these memories other associated memories, and so, while consciousness remains passive, an ever-varying series of memories, visions, day dreams, etc., flow by. But consciousness may be active, and just as cerebral activities may cause either action or inhibition in the sub-cortical centers, so the whole mass and content of consciousness may to a certain extent strengthen certain memories and weaken others. This action of consciousness, like other nervous actions, grows stronger by use. So that a trained, educated, intellectual man, is able to keep one set of memories present in consciousness (attention), to call up associated memories, to reject some, to keep others active, to compare them all together. This is called the *act of thinking* or *reasoning.* The process of thinking is thus independent of speech; although speech is essential to its clear expression and certainly facilitates it, especially in its deep and profound forms. The question of *attention* is one which seems to require a more or less external *will* to keep the cerebral activity limited to one subject. Attention is, however, in part a manifestation of the association of ideas. If many associations at the same time bring into strong consciousness the desirability of investigating some one perception, this idea which we may call "a" and which will have widespread associations, will bring into consciousness this perception to be investigated, which we may call "b" with its various associations. If one of these associations leads to others remote and unrelated, and away from "b", it will not go far before it will awaken some of the associations connected with "a;" even the absence of "b" from consciousness will do this, and "a" will be brought into consciousness and through "a" the investigation will be brought back to "b" again.

When we have forgotten a name, we often cannot by an effort of "will," however strong, recall it. The desire for the name starts series after series of associations in some way related to the name, which finally bring it into consciousness. Or the attempt may fail and the desire may be unsatisfied at the time. Hours or days afterwards the name may enter consciousness by some chance association and be recognized as the desired name.

Imagination: Creative Faculty

Although usually one perception calls up its associated memories and keeps repeating them, in recalling again and again past events; yet unrelated perceptions and ideas may be present simultaneously, or nearly so, in consciousness and may be artifically associated together so that by such combinations ideas and scenes may present themselves, which are not the result

of our experience; or our former experiences may be changed or modified out of all relationship with themselves. This is called imagination. It is the creative faculty which shows itself actively in prose and poetic works of the imagination, or passively in day dreams; in contradistinction to true facts or real history.

Consciousness (Chart III)

Consciousness consists, at any instant of time, of the then present perceptions and of those past memories which are directly or indirectly associated with these present perceptions and which have been awakened by them into activity. Consciousness is thus a form of energy resulting from activity of the cerebral cortex. The other memories, not at that instant active, constitute sub-consciousness and may at any time become conscious memories. The content of consciousness embraces only a small fraction of those activities which take place in the brain and indeed only a fraction of those activities which take place in the cerebral cortex. The activity within the cerebral cortex must reach a certain intensity in order to produce sufficient energy to constitute consciousness. When this cortical activity, although existent, is less intense, we call the result of this activity sub-consciousness. Such sub-conscious activity may leave an ill-defined memory as the result of its action. The conscious and sub-conscious content of the brain together constitute a *personality.*

A new born babe has, probably, no consciousness. An infant attains consciousness slowly as he gradually obtains perceptions and memories and forms a large number of associations of all kinds. When an infant has his first perception, this one perception, together with certain rudimentary sensations he may have acquired, constitutes his entire consciousness and his entire intelligence. It is all he knows. As other perceptions are obtained and associated together his intelligence and his consciousness become larger, more distinct and more complete. The child in his development increases with great rapidity the number of his perceptions, less rapidly and subject to many subsequent corrections, that of his associations, still less rapidly his concepts, even more slowly his ethical and aesthetical ideas, and yet more slowly his abstractions; but at the end of a few years any perception or memory suggesting action is subjected to the interplay of all these activities before the action is done or left undone. In other words, consciousness and the act of thinking and reasoning on which action is based (see *Voluntary Motion*) are already, even in childhood, very complicated, perhaps more so than later in life, when action is mainly determined by habits of thought: by judgments firmly established by numerous experiences.

Consciousness is a form of energy or force, such as are light and electricity, which is transmuted from chemical action taking place in the cerebral cortex. Consciousness embraces all these chemical activities which have a certain intensity; below this degree of intensity these activities constitute sub-consciousness. The process is somewhat analogous to that in an electric-light bulb through which an electric current may be constantly flowing, but which only gives forth light when the current has attained a certain intensity.

Consciousness is constantly being newly formed and is dependent upon the perceptions, memories, feelings and ideas, ethical and others, present at any one instant. These phenomena themselves constitute and are consciousness. None of them, usually, continues long in consciousness. Others are constantly forcing them out. None remains constant. The continuity of consciousness is preserved by the mingling of memories of past perceptions with present ones, and by memories of past states of consciousness.

Consciousness is an active process and depends upon the integrity and the blood supply of the cerebral cortex. This blood supply is in constant ebb and flow throughout the different areas of the cortex; being at any instant most abundant in those areas which are in activity. Consciousness is a condition which, as yet certainly, we do not understand, although in a general way we regard is as the result of chemical changes taking place within the cerebral cortex. *The chemical changes themselves are not consciousness,* but they produce this form of nervous energy very much as a steam boiler and a dynamo, or a galvanic battery, produce electricity. We

are as ignorant of the exact nature of consciousness as we are of that of electricity. These chemical changes produce consciousness; a form of nervous energy; just as the chemical changes taking place in the muscles produce muscular force; a form of mechanical energy; just as chemical changes taking place in any living cell or tissue of the body produce a form of energy peculiar to itself. Consciousness thus locally produced, and thus continually produced, in the brain, passing according to definite channels to other regions of the cortex, surges through the brain, as memories and ideas are awakened and cause action and reaction. The subject is a most difficult one and is made, in a sense, more difficult by the faculty of language, which allows us to replace an idea by a word of somewhat uncertain definition and thus leads to uncertain and faulty reasoning, or to a high sounding sentence which means nothing. Consciousness, or cognition, seems to be something added on to the essential processes taking place in the brain. The various association-reflexes occurring in the brain could take place and do take place quite as accurately without consciousness, as for instance in the automatic acts of the somnambulist, or in the epileptic trance.

The brain is very abundantly supplied with blood, especially the cortex, and the latter is very sensitive to any interference with its blood supply. Loss of consciousness, which occurs normally in sleep and pathologically in many conditions, is caused much more frequently by a change in the quantity (anemia) or quality (drugs and poisons, including sepsis and other autogenetic toxic products) of the blood supply than by all other conditions combined. Perversions of consciousness, on the other hand, seem to depend less upon the quantity of the blood supply than upon its quality (poisons) and upon changes, organic or functional, in the cerebral cortex, especially upon its exhaustion.

Personality (Chart III)

Personality is the sum of the conscious and sub-conscious content of the brain. It expands as this content grows larger and better systematized. It becomes greater as during life a larger stock of energy is accumulated in its associated memories. It is stronger as the consciousness is more intense (virility). The "Ego" is the result of a long series of experiences (former perceptions) by which the body is differentiated from the external world (corporeal ego), and by which the complex of memories and ideas which the cortex has accumulated is differentiated from that of other individuals and is peculiar to itself (mental ego).

The totality of one's memories constitutes his experiences. Many similar memories, or experiences, are gradually combined into a general idea or principle which becomes a guide, or association channel, for future judgments and actions and may persist after the individual memories or experiences upon which it was founded have been lost. Cortical excitations of this nature are followed at once by actions which are almost involuntary (habits) and are not the result of a balancing of many former memories and ideas (thought). In this way one's character or personality is built up. Ideas firmly fixed by tradition, education and habit, acquire an overwhelming emotional value. They not only exist in spite of experience but even mould experience into conformity with themselves. Personality is the result of the manifold working of natural forces. Had the natural forces been different the personality would have been different. Each personality has its own history founded on its own personal experiences. *A man's personality has been created during and by his life, in a brain whose organization and capacity have been modified by heredity.*

Personality, being founded on, and consisting of, personal experiences, is strongly individual; but inasmuch as the large majority of men in the same community have very much the same experiences, and as they discuss these experiences with each other, there springs up between them a friendly feeling as beings of the same nature and with the same interests, needs, desires and aims. According to its education from its experiences a personality may keep itself apart from others and strive only for its own well-being and may thus be selfish (an egotist), or it may merge itself into the social life of the community and strive for the well-being of its fellow men as well as its own and thus be generous (an altruist). In spite of much in common,

each personality differs from others. Some by their educational experiences become con emplative men, others close observers, others men of action, etc. Some men are of weak character, who have always been indulged and have always followed the path of least resistance; some are of strong character, who have had to endure privation and have learned to control their desires. These different kinds of men cannot by any effort of will change suddenly their character, which has been formed slowly by countless past experiences, acting upon a brain the anatomical structure and physiological activity of which has been modified by heredity. The personality of a child has the potentiality of developing in the future, but the kind of development depends more upon the kind of future that is before it, than upon its heredity.

Personality seems to be the energy resulting from chemical changes which have taken place and are taking place in the cerebral cortex and to depend absolutely upon the integrity of the latter. When the cortex is exhausted, or diseased, personality may be changed under some unusual experience, resulting from the undue dominance of some local excitation of the cortex, either permanently or temporarily (double personality), or it may become completely lost (automatism).

Intelligence (Chart III)

Intelligence consists of the content of object consciousness. It is absolutely dependent upon memory, without which it cannot exist. The greater the number of memories and the more perfect and easier the recalling of associations, the greater is the intelligence. A person's memories depend primarily on perceptions derived from his sensory organs and on his experiences. The greater the number and the variety of his experiences the greater will be his intelligence, other things being equal. If any one sensory organ is absent or diseased from birth, memories of this sense will not be present and the intelligence will be diminished, unless this defect is in some way compensated for.

An increase of intelligence, though unusual, is not abnormal. In some cases this increase is due to a greater number of perceptions and ideas (the learned man); in some cases to better and wider associations throughout the entire sensory cortex (the wise man), and in some cases one portion of the cortex is functionally developed at the expense of others (the genius).

A diminution of intelligence may be due to imperfect development, to impaired nutrition or to destructive lesions of the cortex.

Perversions of intelligence, although they may, in part, be caused by peripheral lesions, are fundamentally due to disease, or poisoning, or malnutrition of the cerebral cortex.

Soul and Mind

All of these physiological activities of the cerebral cortex, which we have considered and which are popularly called "mental" or "spiritual," although they depend upon an inherited anatomical structure of the brain, are not present at birth. They are acquired, or created, during life by experience and by education in its broadest sense. They are the result of physiologico-chemical activity within the cerebral cortex. There is no scientific or trustworthy evidence of the existence of any further factor in the form of any ethereal essence, a "mind" or "soul" as distinguished from what has been described above as "personality." Indeed this assumption rather complicates than simplifies the matter, inasmuch as it is contradictory to one of the most firmly established principles of natural philosophy: "the law of the conservation of energy." This law has not only been established by irrefutable proof in the inorganic world, but also has been proved by experiment to be valid in animals and even in man. If the soul can produce or stop a cerebral activity of its own volition, thus creating or annihilating force which normally proceeds in an endless chain from one manifestation of force to another, then the law of the conservation of energy is no longer valid.

Mankind has been unwilling to allow that the causalities and laws, which prevail in physical activities, can be potent also in the body and still less in the brain and "mind," because this controverts all their preconceived notions of the soul and its relation to God. The prejudices (pre-judgments) of most men will not permit them to regard the mental activities as the

result of the physiological activity of the cerebral cortex, in the same way that the physiological activities of the other internal organs of the body produce and cause the functions of these organs. They are unwilling to regard psychology as identical with the physiology of the cerebral cortex. In earlier historic times, the winds from the cardinal points of the compass, rippling streams, cascades, the waves of the sea, growing trees and shrubs, etc., were each supposed to be animated by an indwelling spirit. At the present time, Naiads, Nereids, Dryads and other Nymphs: charming creatures of the imagination, have all been banished by the advance of knowledge. Only the indwelling, animating spirit of man: the soul, remains.

Whether an individual believes in a soul or not, depends upon his traditions, his education, his experiences and upon the personality which has been gradually created and developed during his lifetime by the combination of all those forces constituting consciousness and sub-consciousness, and which may, in a sense, be likened to the older conception of a soul. It seems probable that consciousness, intelligence, personality, etc., are forms of energy and force and may, perhaps, be called *spiritual* in contradistinction to *material*. Whether this energy or force, which is gradually accumulated during life, persists after death, we do not know. It certainly is absolutely dependent upon the blood supply of the cortex, and when this is arrested, personality with every other manifestation of consciousness ceases; but it still exists potentially and may be reanimated, if the circulation be restored after an interval of only a few minutes. If, however, the arrest of the circulation is so long that the cortex begins to degenerate or die, the personality is lost permanently, both actively and potentially.

Our knowledge in comparison with the wonders of the Universe is infinitely small. We know the relation of things, not their essence. But our knowledge is increasing and it is to be hoped that our children's children may have a higher point of view and a clearer vision.

Insanity (Charts III and XVI)

Whether we believe in the existence of a soul within the body or not, certainly insanity is no longer regarded as the possession of the body by an evil spirit (demoniacal possession). This was a well established belief for ages, but it has long since been abandoned and we now regard insanity as caused by abnormal cerebral action. The control of the body by a spirit, which we have finally rejected as regards insanity, the vast majority of mankind still retains for the healthy body, possibly because most of the few men who really think have not studied cerebral physiology.

We have considered briefly the actions taking place in the sensory area of the normal, healthy, cerebral cortex. In an abnormal cortex these actions are deranged. Local disordered cortical function produces local paralysis or apraxia or convulsions or even hallucinations; while general disordered function produces coma, neurasthenia, or insanity. Abnormal structure, whether the alteration be slight or great, and consequently abnormal function, of the cerebral cortex may be either congenital or acquired. The congenital form may be manifest in early infancy or may become apparent at any stage of the individual's development, as he successively meets tasks which require more and more intellectual power, when it becomes evident that his intelligence and ethics fall below the commonly accepted standards of the race or community of which he is a member.

The greatest degree of absence of intelligence is *idiocy* (1081), which shows itself almost at birth. In this disease the brain is so functionally incapable that it cannot produce perceptions, or register memories, or form associations, except of the most rudimentary kind. These persons, then, have no material for intelligence, consciousness or ethics and are incapable of speech. Next to this extreme degree is *imbecility* (1088), which may show itself at any time from birth to early childhood. In this class, simple perceptions, memories, associations and speech are possible, but only very imperfectly, and there is very limited material for consciousness, intelligence or ethics. A still slighter degree of this condition is only manifested when a considerable degree of intelligence or ethics is imperatively required, especially at the so-called critical periods of life, as at puberty (some forms of adolescent insanity—1096). Although individuals of this

class have memories, associations and consciousness, their intelligence, ethics and judgment are found to be inferior to those of their fellows educated under the same conditions and they are called *feeble-minded* and *defectives* (1092) in varying degree. In the slighter forms of this class only the higher and more complicated ideas, such as altruism and morality, are absent or impaired (moral insanity and some criminals). In the severer forms, the loss is more profound and involves all the cortical functions.

In other cases of congenitally defective brain the defect is very slight, but some of the association channels are more patent than others, and than is normal; so that certain associations and ideas are constantly being presented to consciousness and are called up by all kinds of unrelated associations and cannot be corrected, and consequently the cortical actions are distorted and twisted and irrational. To this class belong the *paranoiacs* (1113).

Education and training have much to do with the development of the activity of the cerebral cortex and consequently with the individual's intelligence and ethics. There are individuals who, partly in consequence of a defective brain, but mainly in consequence of a defective training and education, do not have normal experiences and form a number of abnormal associations and ideas, especially ethical. Such individuals comprise the majority of criminals and cranks. Such cases bridge over the separation between the congenital and the acquired forms of insanity. Of course, it is possible that a person with a normal brain, who is isolated from his fellow beings and receives no training or education, will be feeble-minded or even an imbecile.

All these congenital forms of insanity may be broadly classed under the term *amentia* (211, 1076) *in its widest sense:* the mind never having fully developed. On the other hand all the acquired forms of insanity may be broadly classed under the term *dementia* (212, 1077) *in its widest sense:* since there is always present a certain mental weakness not previously present, a falling off in greater or lesser degree from the previous more perfect cortical activity.

Acquired, disordered activity of the cerebral cortex resulting in insanity primarily also depends upon a defective brain, either hereditary or acquired, but secondarily upon many inciting causes. It may be due to a general deficiency in the blood supply consequent upon atheromatous arteries, as in *senile dementia* (1105); or may be due to an irregular cortical circulation consequent upon chronic meningitis; the most striking example of this class being *paresis* (1104). Various poisons (alcohol, etc.), endogenous or exogenous, are responsible for other forms of insanity which are usually, but not always, of comparatively short duration. Local lesions of the brain, such as abscess, tumors, etc., may in some cases alter the circulation of blood through the cortex generally and thus cause insanity. Exhaustion of the cerebral cortex from worry, anxiety, shock and other causes may cause insanity in persons with an unstable brain, as may also an anemic and altered condition of the blood. It is, of course, possible that several of the above causes act simultaneously, or in sequence, and as a matter of fact they frequently do so.

In most of the forms of insanity the altered cortical activity manifests itself in certain striking and unusual phenomena. One of these is hallucinations (213, 1078), which are abnormal perceptions. In hallucinations the symbol occurring in the cortex does not correspond to any external phenomenon, but is purely subjective, and is due to disturbances in the cortex itself, not in the peripheral sense organs. The hallucinations may occur in any of the special sensory regions of the cortex and hence may be either olfactory, gustatory, visual, auditory, tactile, or even visceral. The process in the brain which produces an hallucination must be similar to that which produces a perception. An hallucination is much more vivid than a memory and an hallucination is not a complete and correct reproduction of a former memory, but usually is something strange and bizarre. In some cases the hallucinations do not have the vividness of true perceptions, but seem to be internal voices of suggestions, telephonic communications or electrical action, etc.

An illusion (214) is also a false perception, but it originates from an external reality which is misinterpreted in the brain: the symbol in the cerebral cortex is not such as is usually associated by the average man with the external object, but rather a symbol usually associated

with a quite different object. Hallucinations and illusions may occur as the result of a local disturbance in a brain which may not be for the moment entirely normal, although the individual is certainly not insane. In such cases, hallucinations and illusions can be quickly dispelled by reason and by proof of their abnormal character. Insane persons, however, in consequence of a diffuse cortical disturbance, cling to their hallucinations and illusions with great tenacity in spite of strong proof to the contrary. These hallucinations and illusions occurring in a brain weakened by nature, poison or disease, naturally lead to abnormal associations and consequently to abnormal ideas. Abnormal associations will result not only from the strength and vividness of these hallucinations, but also because from patches of meningitis, or other cause, some areas of the cortex have more blood than others and, therefore, respond more readily to association impulses, near and remote. Moreover these abnormal ideas entering into consciousness and coming into conflict with former long established ideas lead to a condition of consciousness which we call bewilderment, clouded, befogged, confusion, distrust, apprehension, fear, etc. In these cases, impulses reaching the cortex normally from the organs of sense are so much weaker than the excitations already there, that they cannot enter into consciousness, but remain sub-conscious. They may, although sub-conscious, be registered and may be recalled to consciousness after the attack of insanity is past, but they have no present value and are inadequate to correct the abnormal activities and no sane judgment can result.

A cortex in which normal perceptions can occur only imperfectly, or not at all, and in which abnormal perceptions, associations and ideas are dominant, will naturally produce abnormal association reflexes, or actions. The simplest of these is *delirium* (217, 1107-8), in which the patient responds by word and act to the many false perceptions and ideas in his clouded and weakened consciousness. When the intensity of the process is less the false perceptions and ideas will produce *delusions* (215, 1079), which will cause abnormal and often dangerous association reflexes or acts. These delusions may remain isolated, unsystematized or may be woven in with all the real experiences of the individual life; so that a systematized delusion, founded upon more or less evidence or reasoning, acting upon a weakened or limited general cortical activity or judgment, results.

Often in justifying or explaining a delusion a patient will give reasons or cite experiences which we call false, but which are doubtless experiences, the symbols of which have occurred in his abnormally acting cerebral cortex. These delusions, or false and uncorrectable judgments, naturally lead to acts which are incompatible with an unconstrained life in a reasonable community. Naturally with all these abnormal cortical activities not only the ethical ideas of the individual are changed, but also the normal emotions associated with normal cortical activity are profoundly altered, whether in the form of exaltation or depression, either continuously or in alternation with each other.

In all forms of insanity, in consequence of its abnormal content, consciousness is altered and personality may be changed. There may be a double personality or the individual may imagine that he is dead, an animal, a king, or God, or, in extreme degrees of dementia, the patient may show no consciousness or personality at all. The emotions are also altered (morbid temperaments) in accordance with the ideas in consciousness, or may be entirely dissociated from the ideas with which they are normally in harmony, or may be entirely absent in extreme dementia, or may be feebly carried over from former highly emotional states. The emotion most frequently present, especially in the early stages of the disease is fear (phobias).

Fear and apprehension are prominent, even dominant, symptoms in the early stages of almost every case of insanity. The unusual, often monstrous, phenomena occurring in the cerebral cortex are so different from those previously present and so out of harmony with former memories and ideas, that the patients naturally become distrustful, apprehensive and full of fear. Many can hardly believe the information supplied by their own senses, much less the words of their friends. Some regard themselves as persecuted and as the victims of conspiracies. Fear is the dominant emotion within them.

The association reflexes are always altered in insanity in consequence of the abnormal cortical activity. In extreme dementia, voluntary motion is completely abolished. In profound melancholia, voluntary acts, as well as thought, are inhibited; while the reverse is true in mania, in which cortical activity, although abnormal, is greatly exaggerated. In consequence of prominent, compulsory ideas, so frequent in insanity, compulsory acts result.

Movement (Charts IV and V)

When a sensory surface is irritated the animal often responds immediately by a comparatively simple movement, or the movement may occur only after a considerable space of time and may be very complicated, or it may never occur. Movements may also occur spontaneously, apparently not being preceded by any sensory irritation in the immediate past; although on careful analysis these spontaneous movements can always be referred back, indirectly, to some sensory irritation. All these different kinds of movements are divided into three great classes: reflex, voluntary and automatic.

Reflex Action and Inhibition (Chart V)

A reflex act is a reaction from an irritation, which under like conditions always takes place in exactly the same way; it seems purely mechanical, as if a machine were working. The irritation may be a usual (normal or adequate), or an unusual (abnormal or inadequate), one; the former being much more effective, and it may affect the skin, mucous membrane, or muscle, tendon or fascia, or any of the organs of special sense. The impulse starts in the end-organ of a sensory nerve supplied to the sensory organ affected and passes centripetally along the peripheral sensory nerve fiber, or fibers, to the spinal or cranial ganglion, situated on the posterior spinal, or the cranial, nerve root. Thence it passes forward through the posterior horn to the anterior horn of the spinal cord, or through the brain stem to a cranial motor nucleus, and thence along a motor nerve root and peripheral nerve to a muscle or muscles, striated or unstriated, in which it causes a contraction, or to a gland in which it causes an alteration in its secretion (Fig. 24). Muscular tonicity is a variety of reflex action (240).

The various nervous elements traversed by the nervous impulses, as just described, constitute what is called "the reflex arc" (296). Slight irritative lesions of the reflex arc cause exaggeration, while destructive lesions cause abolition, of reflex action. This is the simplest form of nervous reaction and this is the simplest expression of it. Such simple reflex acts are the only ones occurring in the body during the early months of life and are unconscious acts. Similar reflex acts cause the respiratory and cardiac movements, the flow of saliva and other secretions, the vascularity of organs and the warmth of the body, and in general regulate the physiological actions of the body.

When the nervous impulse reaches the gray matter of the central nervous organs so many ways of transmission are open to it that it can pass by longer or shorter arcs or by several of them. The shortest possible reflex arc is through a peripheral ganglion (as in the vasomotor reflexes). The next shortest is through the spinal cord. A longer one is through the spinal cord and the ganglia at the base of the brain. The longest is through the cerebral cortex. Some reflex acts, such as the pupillary (302), remain unconscious acts throughout life. On the other hand, most of the sensory impulses described above, when they have traversed the sensory nerves and have reached the sensory ganglia, pass in part as described above to the motor nuclei, but pass also in part up the central sensory conducting tracts to the higher ganglia at the base of the brain; where they may cause more complicated reflex acts and pass still higher to the sensory cerebral cortex; where they may give rise to sensations and perceptions (Fig. 35). Here the impulses may apparently stop, or they may be continued from the sensory cortex to the motor cortex and thence a new impulse may pass downward along the pyramidal tract, and thus the involuntary reflex act may be increased or replaced by a voluntary act, or may be abolished (inhibited), voluntarily.

This voluntary abolition of reflex activity (inhibition) may be brought about by a contraction of those muscles which antagonize the muscles taking part in the reflex act, or this latter act may be "inhibited" by a direct action upon the sub-cortical motor cells taking part in it. In addition to this voluntary inhibition, a great variety of nervous activities taking place in almost any part of the nervous system (especially strong, painful impressions), and even the normal process of cerebral activity, will cause a more or less complete inhibition of reflex activity. It seems as though two impulses acting upon a cell at the same time under certain conditions may mutually counteract each other. Therefore, reflex activity is more active in animals in which the brain (or even other parts of the nervous system) is separated from the spinal cord or reflex centers and in human beings when the same result is accomplished by disease (isolation). Curiously enough, when the human spinal cord in its upper part is completely destroyed the reflex activity of the lower parts of the cord is abolished. This phenomenon has not been satisfactorily explained and is in marked contrast to the exaggerated reflexes found in incompletely destructive lesion of the upper portion of the cord.

Conduction of reflex or other impulses along the peripheral nerves is equally rapid whatever may be the intensity, or quality, of the irritation, but conduction through the gray matter is much slower and varies greatly with the intensity and quality of the irritation. The gray matter also possesses the power of summation; so that inactive excitations may become active ones by repetition. The gray matter immediately following its activity shows a "refractory period" of longer or shorter time during which is it inexcitable or exhausted. This indicates that the gray matter accumulates energy during rest, which it discharges when in activity. This refractory period may play its part in rhythmical action. Most reflex acts are purposeful and healthful in their nature. Many of them are absolutely essential for life. They may be divided into the offensive and the defensive.

A destructive lesion of any portion of the reflex arc causes abolition of the reflex acts, as does also a strong irritation of the higher nervous centers. Slight irritative lesions, such as slight inflammations, will cause an exaggeration of the reflex act, as will also and more commonly a lesion which interferes with conduction of nerve impulses (inhibitory impulses) through the central motor (or cortico-spinal) neurons. An irritation, especially a continuous one, even if not very intense, will often cause a tonic spasm or contracture.

Voluntary Action, Association Reflexes (Chart IV)

The anterior portion of the cerebral cortex, which contains innervation (kinesthetic) memories, or motor centers, is connected through bundles of association fibers with the posterior portion of the cortex, in which are memories obtained from the organs of sense. Activity never normally originates directly, or spontaneously, in the motor cortex, but comes to it from the sensory cortex. When a very strong excitation arises in this sensory cortex, as for instance perceptions which are associated with the idea of imminent danger of death, this excitation passes to the motor cortex and thence down through the internal capsule and pyramidal tract and causes movements of flight and self-preservation. This act is as inevitable and as machine-like as is the simplest reflex act. A good swimmer bent on suicide cannot drown himself unless he is weighted or the action of his knees or legs is restricted. Escape from imminent death is for most men an imperative voluntary act. If, however, the danger is less great, as on a battle-field, the excitation leading to flight may be still there, but it may be inhibited by excitation from other associations, such as the idea of shame, love of country, etc., and the two excitations may neutralize each other. It is a question which idea: the fear of death or the love of country and honor, is based on stronger perceptions and wider and stronger associations. Whichever is the stronger prevails.

Ordinarily, when a perception, or memory, suggesting action has sufficient intensity to enter consciousness, the excitation is sufficiently strong to pass along the association fibers and awaken into activity the corresponding innervation memories and, if no other counteracting excitation comes to this latter portion of the cortex, the irritation passes through the great

motor cells in the anterior central convolution and down through the internal capsule and pyramidal tract and the action takes place. Actions resulting from memories are usually weaker than those resulting from the original excitation or perception. When a number of more or less conflicting memories and ideas are in consciousness, some for and some against the action, impulses will be constantly coming to the motor cortex to be either immediately inhibited, or strengthened. The play of the different perceptions, memories and ideas: *the play of motives*, may continue a long time as the person *deliberates* and exercises his *free-will*. This merely means that the different memories, together with the ideas derived from the mass of associated memories which constitute our ethics and those which constitute our emotions, have sufficient intensity to act upon the motor cortex, some as excitants, some as inhibitors. Fresh, allied memories constantly enter consciousness, because of their association with those already in it, and take part in this phenomenon. It is like a debating society in which arguments for and against are presented almost simultaneously, and the stronger argument rather than the will of the judge is the decisive factor. It is probable also that sub-conscious activities may play some part in this process. Eventually the stronger excitation will prevail and the act will be either done or left undone.

A voluntary act, depending upon, and being the result of, the association of ideas, may be described as an *association reflex*. The idea of the apparent freedom of will depends upon the absence of external compulsion and also upon the fact that the action takes place, or does not take place, in accordance with the relative strength of our ideas and desires. The more perceptions and ideas a person has in his memory, the more learned and intelligent he is, the greater, wider and more protracted will be this "play of motives" and the more difficult will be the choice of the resulting action: the association reflex, the victory of any one set of motives. The very learned man is not the man of action. In a child or in an ignorant man, with fewer elements of a choice, the association reflex may be more prompt. When a decision under the same or similar conditions is made a second time, and especially when frequently repeated, the association reflex takes place more and more promptly. In the frequent repetition of acts: practice, the same association conducting channels are being constantly traversed and consequently become better conductors, and the acts become easier to perform: become more or less automatic. It becomes a habit. A large part of our voluntary acts are habitual. It is to be remembered also that the internal sensations, which dominate our "moods," exercise a strong influence over voluntary movements which, under changing moods and altered internal sensations may be very different at different times, although the causal external sensation is the same.

The gray matter: the point of union of the motor and sensory neurons, is in small compass in the sub-cortical centers and hence is well fitted for direct transference for reflex and automatic actions. In the cortical centers the gray matter is spread over a large surface and permits separate, local action, and consequently permits a large number of different memories and ideas, some positive and some negative, to act simultaneously upon the motor cortex and thus either cause or prevent a voluntary action. Both voluntary and reflex acts are for the benefit of the individual. The reflex acts depend upon heredity and evolution. They are the result of the experiences of the individual's ancestors, of the experience of the race (phylogenetic). Voluntary acts depend upon the individual's personal experience (ontogenetic). The difference between the two depends mainly on differences in anatomical structure. In conditions which are new and in which no experience can guide him, an individual's voluntary acts are quite as likely to be detrimental as salutary. His reflex acts almost without exception are salutary.

The innervation memories stored in the motor cerebral cortex are originally acquired from reflex acts. The first voluntary acts of the child (sucking, opening and closing eyes, closing of hand, etc.) are adopted reflex acts, either unmodified or but slightly modified. A young infant does not will to suck milk from his mother's breast. It is a reflex act. But after the infant has experienced the result of this act a sufficient number of times, the sight of his mother, or hunger, may awaken his desire and he will suck the breast voluntarily in consequence of this

active memory. Voluntary motions, or association reflexes, occur early and develop rapidly in infantile life, but occur much earlier, though they develop much slower, in young animals. Chickens run almost as soon as they are born to their mother when she "clucks" for them. The lower animals thus possess at birth, by heredity, a more perfect nervous system; while human infants possess at birth by heredity, one less perfect, but capable of a wonderful development, which results in greater part from personal experiences.

A normal voluntary motion depends not only upon a fairly healthy cerebral cortex (which implies a certain degree of intelligence) and a normal muscle, but also upon the integrity of the two motor neurons: the central (*cortico-spinal*) and peripheral (*spino-neural*), or the upper and lower (461-2). The impulse causing the voluntary contraction starts (as regards its purely motor function) in the cell body of the central motor neuron lying in the anterior central convolution (Fig. 15). It passes along the axon of the cell, which becomes the axis cylinder of a nerve fiber, through the corona radiata, the anterior portion of the posterior limb of the internal capsule (Fig. 17), and emerges from the cerebral hemisphere at the base of the brain in the pes cerebri. It is then covered by the transverse fibers of the pons Varolii, from the lower edge of which it emerges to help form the anterior pyramids of the medulla oblongata (Figs. 20-22). At the junction of the medulla with the spinal cord, these fibers of the anterior pyramids in great part decussate; the completeness of this decussation varying somewhat in different individuals (Fig. 23). Usually the great majority of the fibers decussate and run down through the spinal cord in the posterior part of the lateral column (crossed pyramidal tract), a small minority of the fibers running down in the anterior column of the same side as the pyramid and close to the anterior median fissure (direct pyramidal tract) (Fig. 26). In certain extremely rare cases no decussation takes place. The fibers from both the direct and the crossed pyramidal tracts pass to the groups of motor nerve cells lying in the anterior horns (Fig. 26), and to these cells they communicate their impulses. (The course of these central motor neurons is shown in Fig. 34.) From the group of nerve cells in the anterior horns of the spinal cord, these impulses, thus communicated, pass out along the axons of the cells, which axons become the axis cylinders of the anterior nerve roots, and thus pass along the peripheral motor nerve fibers to the group of muscles innervated by this group of nerve cells in the anterior horn. These impulses enter the muscles by the motor end plates and cause a muscular contraction.

Although the cortical motor centers represent almost exclusively muscles lying on the opposite side of the body, it appears from clinical observation and physiological experiment that the muscles of the body have a bilateral cortical representation. By electrical stimulation of the cortex the muscles on the same side of the body may be made to contract, although a much stronger irritation of the center is needed than is necessary to cause a contraction of the corresponding muscles on the opposite side of the body. Those muscles on both sides of the body which usually act together have especially well marked bilateral representation; so that these muscles are rarely completely and permanently paralysed in unilateral cerebral lesions. A cortical paralysis may affect motion only and may be very circumscribed; two or three fingers, or the thumb only. The actions which are especially lost in the cortical lesions are the purposeful actions which have been slowly acquired as the result of experience and training: actions which are peculiarly voluntary and skilful.

A destructive lesion of either of these types of motor neurons will cause a motor paralysis. If the peripheral motor neurons are destroyed there will be a paralysis both of voluntary and of reflex acts: a flaccid paralysis (252), while if the central motor neurons are destroyed there will result a paralysis of voluntary acts only; the reflex acts persisting and being even increased: a spastic paralysis (253). (For explanation of the increase of reflex activity just mentioned see page 22.)

The Will and Will Power

The term "voluntary" motion implies "volition" or "will": some attribute of the individual or personality which controls or regulates the cerebral activities. The will and the freedom of the will stand, however, in direct contradiction both to the law of preservation of force or energy

and to the law of causality. Moreover, we have just learned on page 23 that voluntary motion results from the play of motives and that the strongest sensory cortical activity prevails and causes the resulting motion which seems to be due to our will power only because the perception or memory in consciousness resulting from this "strongest sensory cortical activity" is, in virtue of its strength, in accordance with our so-called will or desire. It seems, therefore, better to call these actions which result from the strongest sensory cortical activity "association reflexes" rather than voluntary acts. As the result of experience in life we acquire desires (see page 11) and each desire can only be satisfied by action. The presence in consciousness of a desire to obtain a certain end or result; which desire is itself the result of cortical activity will by this intense cortical activity excite, influence and usually control those cortical activities, which produce action, thought, study, etc. It is the cortical activity causing the desire which does this, not some external entity: the will.

The act of concentration or attention seems also to call for a will power, but we have learned on page 14 that concentration depends primarily on a strong cortical activity, which the so-called will power and our desires are often incapable of controlling. What has been called the will is probably the dominant cortical activity present in consciousness at any given moment. It depends upon the personality which rests upon many experiences and upon many crystallized experiences in the form of judgments, habits, prejudices, etc., which give our actions stability and consistency; and this sum of consciousness, or even one strong idea in consciousness, the result of a strong cortical activity, may modify the association of ideas and may control both them and the resulting action.

Nevertheless, both in voluntary actions and in efforts of attention, the individual, or the personality, does not seem to himself to be a mere passive spectator, but feels that, although he may be somewhat bound down by experience and habit and prejudice, yet he does exercise a very real and decisive influence upon both these processes, and at least modifies if he does not actually control them. Everyone feels conscious of this power, and it is not altogether satisfactory to dismiss this feeling as a delusion existing in the mind of every member of the human race. It is indeed quite possible that the personality, which is a force produced by chemical process taking place in the sensory cerebral cortex: the result of cellular activity, may in the motor cortex be transmuted back into cellular activity, and thus may promote or inhibit motor action. Just as an electric light, produced by chemical activity within a battery, may be transmuted again upon a photographic plate into chemical action; or as the light of the sun acting upon the chlorophyl in green leaves may break up the molecule of carbon dioxide into its constituent parts: carbon and oyxgen.

Personality is a force, and it is hard to conceive of an immaterial spirit as anything else than a force. The primary and essential element in the process still remains the activity of the neuron, and we have already seen that the force generated by one set of neurons may in turn generate, or modify, the activity of another set of neurons. Thus, the personality, which is gradually built up during the life of an individual, may play an active, not a passive, role in the phenomena constituting his conscious life and activity. This statement of what constitutes the will and will-power is in harmony with the subjective feelings of mankind; and yet it is but a restatement in other words of what has been said in the preceding paragraph that " the will is the dominant cortical activity present in consciousness at any given moment," because the personality is also the expression of the sum of the cortical activities present at any given moment.

Spasms and Convulsions (Chart IV)

Spasms and convulsions consist in involuntary muscular contraction. They depend mainly upon irritation of the central gray matter, especially the cerebral cortex, and partly upon peripheral irritation.

Passive contracture and Thomsen's disease alone are purely of peripheral (muscular) origin. Many of the tonic spasms are reflex, some are the result of nerve root irritation (meningitis, tumors, etc.) and many are associated with degeneration of the pyramidal tracts.

The result of pathological and experimental investigation makes it evident that epileptic and epileptiform convulsions originate from irritation of the motor cortex. When a slight, but lasting, local irritation of the motor cortex occurs, there results a local spasm, clonic and tonic, which extends from one extremity to another and finally becomes a general convulsion, accompanied in some cases by coma. When the irritation is stronger and especially when it affects both hemispheres there results first a tonic followed by a clonic convulsion and coma. Irritation of other parts of the cortex can also produce epileptic convulsions, if the irritation be strong enough and the motor cortex be intact. Tonic spasms, without clonic ones, may be obtained by irritation of many parts of the central nervous system. The epileptiform convulsion caused by cortical irritation may be accompanied by alterations in the cardiac action in the respiration and in the activity of other internal organs, as in cases of ordinary epilepsy. Indeed, these changes in the cardiac action and in the circulation through the brain may be more essential factors than is the cortical irritation in the actual production of an epileptic attack.

The contractures which accompany cerebral paralyses are due to contraction of the stronger muscles, partly in efforts for voluntary movements from the brain, but mainly reflexly from the spinal cord.

The pathogenesis of many spasms and the localization of their origin, especially of the irregular spasm, are given in the chart.

The various forms of spasms are at times quite difficult to recognize. It requires much experience to be able always to differentiate clonus, tics, athetoid and choreic spasms from one another and from the perversions of motion: tremor, ataxia and apraxia. This is unfortunate because the diagnosis by these diagnostic charts requires that the symptoms be correctly observed and named. The student should compare carefully what he sees with the definitions in the book and should observe as many cases as possible.

Apraxia, Ataxia and Tremor (Chart IVc)

When an impulse from a cortical motor center passes down to a group of nerve cells in the anterior horns of the spinal cord, it causes a definite synergic contraction of a number of muscles to produce the movement over which this group of nerve cells presides. As soon as this movement commences, a number of sensory impulses pass from the muscles and joints involved to the co-ordinating centers, especially to the cerebellum, and the movement is consequently co-ordinated and orderly. This co-ordination of movements is not inborn. It is acquired by experience and practice. The movements of a new born baby are always ataxic. When the function of the cortical center is impaired there results a paralysis or an *apraxia* (loss of skill) according to the degree of the impairment and when the co-ordinating apparatus is functionally impaired there results ataxia. In either case awkward, ill-adapted and uncertain movements result. There is *asynergy* the muscles taking part in the movement do not act together at the proper time and with the proper relative force to produce an orderly movement.

The motor apparatus, together with its sensory regulation, may be called the executive apparatus and it may be disordered in various ways.

1st. If the motor portion of this apparatus be injured there results a *paralysis* or *paresis*. See Chart IVa.

2nd. If the sensory or regulating apparatus be injured there results *ataxia*. See Chart IVc.

3rd. If what has been learned has been lost or impaired there results *apraxia* or *dyspraxia*. See Chart IVc.

Ataxia: inco-ordination of movement, always depends upon some disturbance of the sensory or regulating apparatus. It occurs in several distinct varieties, depending upon the portion of the sensory nervous system affected.

1st. Peripheral, or dynamic, due to lesion of the peripheral sensory neurons.

2nd. Cerebellar, or static, due to lesion of the cerebellum or its tracts, including the termination of the auditory nerve in the semi-circular canals.

3rd. Cerebral ataxia, to a lesion of the cerebral hemispheres.

1. *Peripheral*, or *dynamic, ataxia* (280, 644) is caused by an impairment or loss of the complicated sensations conveyed by sensory fibers from the muscles, joints and other tissues which is known by the name of muscle-joint sense (42 and 352). It affects all movements of the parts involved. It is associated with hypotonia (240), which allows an abnormal excursion in passive movements without the resistance normally offered under sudden stretching, and which may allow of abnormal positions of the extremities. The loss of the muscle-joint sense can to some extent be replaced by the sense of sight, which allows the patient to guide his movements by his eyes.

Cerebellar, or *static, ataxia* (281, 642) is caused by impairment of the function of the great co-ordinating organ: the cerebellum. It affects mainly, or only, walking and standing, which acts resemble those of a drunken man, or become absolutely impossible. The sense of sight gives very little aid in such cases. Movements of the extremities while the patient is recumbent are fairly normal. With cerebellar ataxia is usually associated vertigo; although this latter symptom may not be pronounced.

Cerebral ataxia is due to a lesion of the sensory tracts and centers within the brain. If this lesion involves the sensory tracts in the medulla or pons or crura cerebri, the cerebellar tracts may also be involved and the ataxia may be either cerebellar or dynamic or both. In cases of cerebral hemianesthesia where the lesion is either in the optic thalamus, the internal capsule or the parietal cortex, the ataxia which invariably results is of the dynamic variety and is associated with hypotonia. Such cases of ataxia may be slight in degree and may show great and relatively rapid improvement. When the lesion is in the parietal cortex, the centers for cutaneous and muscular sensibility, ataxia results, because of the loss of those sensations which are essential for the proper guidance of voluntary movements. In tumors of the frontal lobe, whether cortical or sub-cortical, ataxia is a common symptom and is of the cerebellar type; being doubtless due to involvement of the fronto-cerebellar tract. In cerebral ataxia it is evident that the patient is trying to execute the movements and knows what he wants to do, but he executes them awkwardly.

Apraxia (282) may result from the loss of the purposeful idea which should prompt a given action. In lesions of the posterior central convolution or of the supra-marginal gyrus this idea cannot be formed (sensorial apraxia or agnosia), in which case the action which should follow the idea cannot originate; or when this idea is formed the memory is quickly lost (amnestic apraxia), in which case the appropriate action is begun, but never completed. In lesions of the anterior central convolution, or of the area immediately anterior to it, the purposeful idea may be present, but the innervation memories necessary for the production of the appropriate action are lost; so that the action cannot be performed (motor apraxia). When the association fibers connecting the anterior and posterior central convolutions are the seat of lesions, the appropriate action will not occur, or a somewhat similar action may be substituted for it (associative apraxia).

In any organized society much results from imitation and from instruction. Certain complexes of innervation feelings become by practice so firmly united, that what was at first done with difficulty and imperfectly, becomes easily and perfectly done. These innervation complexes are not inborn (although their anatomical sub-structure may well be), but are learned, are acquired by practice. These innervation complexes become memories (kinesthetic memories). Innervation memories may be conscious in early life when first learned, but may be unconscious later. Many of them may never enter consciousness. As long as these memories persist the corresponding action may be performed, consciously or unconsciously, as the final result of sensory impulses exciting them. Many complicated acts are not performed often enough to form an innervation complex, but must be performed consciously and with constant sensory guidance from many parts of the brain simultaneously (sight, muscle sense, touch, etc.).

Tremor (250) may be caused by rapid rhythmical interruptions of the innervation impulses passing to the muscles or by a failure of a proper proportion or equilibrium in the innervation of the muscles and their antagonists. Tremor usually ceases during sleep and is usually increased

by mental excitement; although a very powerful emotion may arrest the tremor temporarily. It seems to be always of central origin. Clonic spasm from exaggerated reflexes must not be confounded with a coarse tremor.

Speech (Charts IVc and XIII)

The most complicated and important of all voluntary acts is speech. Speech and the allied functions: reading and writing, are peculiar to human beings and are the result of much instruction in the line of imitation and study. These functions, therefore, depend upon a healthy brain. If a child has such an imperfectly formed brain that he is an idiot (743), he consequently cannot speak. The perfection and content of speech, reading and writing depend upon education; being more imperfect the less the education and training and are, therefore, often quite abnormal, or even absent, in the defective and feeble-minded (750, 1088, 1092) and in hysteria (747-8) and insanity, especially in adolescent insanity (1096), in dementia (1103), in coma (745) and in insanity with diffuse cortical changes in the speech area (1104).

The power of speech is of enormous importance in the development of the race. It is the one factor which has enabled the human race to so far outstrip all other animals that it seems to form an entirely different order of beings from them. The spoken, and still more the written, word allows man to make his own all the experience, knowledge and wisdom of his ancestors and contemporaries, and raises him, thus, far above his own limited individual experiences.

Like all knowledge, the art of speaking, reading and writing is acquired from sensory impressions. The art of speech is derived from the sense of hearing; so that when a child is born deaf, or acquires deafness in the first two or three years of life, he is also dumb: a deaf mute (744). A deaf mute can be taught to speak only very imperfectly, and then only by the sense of sight, or much more rarely by touch (Helen Keller). The art of reading and writing is derived partly from the sense of hearing and mainly from the sense of sight. If a child is born blind, or acquires blindness in the first few years of life, he can learn to read only books printed in a peculiar way, and then only by the sense of touch and hearing; the sense of touch replacing the sense of sight in these cases.

It is evident, then, that the perceptions and memories of spoken words are of fundamental importance in the art of speaking. These perceptions take place and these memories are stored, in right handed persons, in the posterior half of the left superior temporal convolution and in the posterior portion of the left island of Reil (Fig. 15); so that lesions of this area cause a profound disorder of speech: sensory aphasia (772). From this portion of the cortex impulses pass along association fibers (the fasciculus uncinatus) lying in the external capsule to the base of the left inferior frontal convolution (Fig. 15) and to the anterior portion of the island of Reil. A lesion in this region also causes a profound disorder of speech: motor aphasia (771).

The distinction between motor and sensory aphasia is not always easily drawn. In some cases when a patient is unable to speak a desired word it may be very difficult to decide whether he has forgotten the innervation memories necessary to speak the word (motor aphasia—221), or has forgotten the word itself (sensory aphasia—222). In the latter case he may be able to repeat the word when he hears it spoken. Lesions of the external capsule, in which run the association fibers connecting the centers of sensory and motor speech (the fasciculus uncinatus), also cause a profound disorder of speech (conduction aphasia).

Perceptions of written or printed words are formed and their memories are stored, in right handed persons, in the cortex of the left occipital lobe, and from this area impulses pass along the association fibers lying beneath the angular gyrus to the base of the left inferior frontal convolution and the base of the left middle frontal convolution, where are stored the innervation memories of speech and writing respectively. Therefore, deep lesions in the region of the left angular gyrus in right handed persons will cause a complete alexia (773) and an incomplete agraphia (776). The area of the cortex in the left hemisphere described above, including the bases of the middle and the inferior frontal convolution, the island of Reil, the posterior half

of the superior temporal convolution and the angular gyrus is called "the zone of language" and is the cortical center, or psychic center, for the faculty of language.

In addition to its cortical center, speech depends upon the integrity of the muscles and nerves which move the lips, tongue, soft palate, larynx and those concerned in respiration. In lesions of these muscles and nerves and of their nuclei in the medulla and pons and of the pyramidal tract, speech may be abolished (anarthria) or pronunciation impaired (dysarthria), whether in consequence of paralysis or of inco-ordination, or of spasm (as in stuttering). Reading and writing may be similarly abolished or impaired in lesions of the peripheral nerves or of their nuclei in the optic thalamus or in the anterior horns of the cervical enlargement of the cord or of the fasciculus of Gratiolet or of the pyramidal tract.

Dysarthria might also be due to a cortical paralysis of the pneumogastric nerve, but the laryngeal muscles have a bilateral cortical representation; so that if one cortical area be injured the corresponding area of the other hemispheres can carry on the function of speech perfectly. There is, therefore, no laryngeal paralysis, or consequent dysarthria, due to any lesion within the cerebral hemispheres, unless the lesion be very extensive and involves both hemispheres (pseudo-bulbar paralysis).

Automatic Movements (Charts III and XVI)

This term is applied to two quite different sorts of actions. In one sense automatic, or autochthonus, acts are reflex acts which originate, not from external, but from internal, or organic, excitations or irritations. One of the best examples of this activity is the respiratory act. Such acts are very numerous and carry on the nutritive activities of the body.

The name is also applied to voluntary acts which have been learned with more or less difficulty, but which have been enacted so often that they can be performed without consciousness. Such acts are walking, writing, piano-playing, smoking and many others which can be very perfectly performed unconsciously, although each one can also be enacted consciously and usually is so done.

Trophic Influences (Chart XVII)

The nervous system exercises an important trophic influence over many of the tissues of the body, in addition to influences over their blood supply through the vaso-motor system. This trophic influence can be divided into two great divisions, motor and sensory. When the motor nerve cells of the central or peripheral motor neurons are degenerated or destroyed (as in lesions of the nerve fibers or of the motor cells, of which these nerve fibers are the axons), the nerve fibers springing from such degenerated cells undergo a rapid degeneration, as do also the muscles, in which these nerve fibers terminate; and in early life when there is motor paralysis, or immobility of parts of the body from any cause, these parts fail to grow normally.

When the sensory nerves are degenerated, as in syringomyelia, myelitis, tabes, lesions of the spinal ganglia or of the ganglia at the base of the brain, etc., in consequence of the anesthesia, the body is no longer protected, by reflex and voluntary acts, from the many traumatisms to which it is frequently subjected and therefore ulcerations, arthropathies, ulcerations of the cornea and other trophic lesions result.

Some of the ductless glands, especially the pituitary and the thyroid, when hypertrophied or atrophied as regards their glandular structure, also produce widespread trophic disorders.

The Cerebro-Spinal Fluid (Charts VIII and XIX)

The central nervous organs (brain and spinal cord) are bathed in a fluid called the cerebro-spinal fluid. This fluid is secreted or transudes from the choroid plexus within the ventricles of the brain and thus may contain substances which are in the blood. It passes out of the ventricles at the inferior angle of the fourth ventricle, through the foramen of Magendie. If from any cause (tumor, meningitis, etc.) the foramen of Magendie is occluded, this fluid, constantly secreted, cannot excape from the ventricles and dilates these cavities more or less ac-

cording as the sutures of the skull are ossified less or more completely; thus producing internal hydrocephalus. The cerebro-spinal fluid passing out of the foramen of Magendie becomes the sub-arachnoid fluid, which lies in the meshes of the tissue forming the deeper layers of the arachnoid. In this situation it can receive products of any inflammation of the meninges: albuminous substances (globulin) and cellular structures (leucocytes in acute, and lymphocytes in chronic, inflammations); so much so as to be cloudy or even purulent. The specific germs of the various forms of meningitis can often also be detected, as well as blood in hemorrhage and pus in abscess. In tertiary and quaternary syphilitic meningitis the Wasserman reaction is usually positive.

The cerebro-spinal fluid is obtained by lumbar puncture and the rapidity of its escape is evidence of the tension which it is under, which tension can more accurately be measured by a manometer. When the cerebro-spinal fluid is increased in amount, as in meningitis, or when a foreign body, as a tumor, is within the cranial or spinal cavity the tension of the fluid is usually increased. The examination of this fluid is, therefore, of much importance in disease of the cerebral and spinal meninges and in other intra-cranial and intra-spinal conditions.

Electricity and the Nervous System

Nervous conduction, although it has some analogies with electrical conduction, is due to an entirely different form of energy. But when nervous action takes place, whether in a peripheral nerve or in a central ganglion, there always occurs an electrical current through the nerve or ganglion in the opposite direction. So constant and delicate is this reaction, that it has been used to prove the presence of nervous activity. Moreover the electric current, both Galvanic and Faradic, can be conducted along nerve fibers, and changes in the tension of electricity so conducted in the nerve fibers cause contraction of the muscles in which they terminate; as is shown in Chart VII. The muscle fibers also respond directly to changes in intensity of a galvanic current, but not to those of a Faradic current.

All forms of electrical energy are excitants for all the sensory organs, acting not so much upon the end-organs as upon the nerves themselves.

Other forms of electricity, especially static electricity and high frequency currents, are used as therapeutic measures but have no diagnostic value.

CHART I

Case-Taking

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES

Errors in diagnosis result more frequently from imperfect observation than from faulty reasoning.

Methods of Examining and Testing Patients — Data derived from

- QUESTIONING ..... see chart I a.
- INSPECTION ..... see chart I b.
- PALPATION ..... } see chart I c.
- PERCUSSION ..... }
- ELECTRICITY ..... }
- LUMBAR AND BRAIN PUNCTURE. }
- OPHTHALMOSCOPY ..... } see chart I d.
- LARYNGOSCOPY ..... }
- THERMOMETRY ..... }

CHART I a

Questioning

Comprising Numbers 1 to 18

(Note)—The examination of every patient, who is conscious and intelligent, begins with a history of his health and of that of his ancestors. This is an important source of information, although usually less so than are the results of the physical examination. The taking of a reliable clinical history is something of an art, but at best we are absolutely dependent upon the truthfulness of the patient, as we rarely have means to check his statements by information from other sources. It is important to put the patient at his ease and to gain his confidence. The patient is vitally interested in his own case and it is best to let him tell his own story of his illness in his own way, without interruption; then to question him further about his illness, more especially and fully in regard to the organ probably affected, but also concerning the function of the other organs of the body. This done, he should be questioned as to his previous illnesses, occupations, etc., and finally as to any special prevailing illness in his ancestors or relatives. It is important to ask as few leading questions as possible. Questions in regard to personal habits and venereal diseases should only be asked when absolutely alone with the patient, and then in a manner which assumes that all men are guilty of indiscretions. During our taking of the clinical history we should have the patient under close observation and can thus form a good judgment as to his manner and general mental and physical characteristics.

QUESTIONING

Methods of Testing

1 History of present illness. (Chart II)

Allow the patient to tell the story of the illness without interruption. Then ascertain the exact date and manner of onset (sudden or slow, prodromata, etc.) and the exact sequence of symptoms. Inquire into all details which may concern the case (headache, pain, paresthesiae, vertigo, insomnia, mental condition, emotions, memory, special senses, paralyses, spasms, fits, disturbances of organic reflexes, loss of weight and strength, etc.), whether of recent or of old date. Seek for any possible cause (injury, poisons, drugs, infections, worry, mental or physical overstrain, shock, etc.). Be careful not to suggest answers to nervous people. Inquire into previous treatment and its effect.

2 Family and personal history. (Chart II)

Ascertain the occurrence, in the present, or a past, generation of the family, of any nervous diseases, especially the neuroses (neuralgia, epilepsy, hysteria, insanity, suicide, drunkenness, etc.), or of syphilis, or tuberculosis. Inquire as to consanguineous marriages. Note patient's age, full address, race, his mental and physical development in school life, occupation, habits (alcohol, drugs, venery, masturbation, etc.), dwelling and previous illnesses, such as rickets, infectious diseases, chorea, fits, tuberculosis, syphilis (use discretion in this inquiry: ask about sore throat, skin rashes, miscarriages, etc.), and injuries at birth or later. Ascertain the condition of other organs (cancer and tuberculosis).

3 Consciousness. (Charts III & XVI)

Patient may lie in a stupor and make little or no response to questions, noises, shaking, pin pricks, or strong sensory irritations of any kind. He may appreciate neither his surroundings, nor his acts, nor the time and place, nor his own individuality. He can remember, after recovery, nothing of what happened while he was unconscious. There are all possible grades in impairment of consciousness from complete coma to a slight lack of attention and an inability to collect his thoughts. This can be learned by conversation with him.

4 Sanity. (Charts III & XVI)

Patient's conversation and manner may show that his brain acts in an abnormal way and that he entertains abnormal perceptions and ideas (hallucinations, delusions, compulsory acts and ideas, etc.). Ascertain if a change has taken place in the patient's normal mental state, and when. Note whether patient is elated, active, loquacious; or dull, inattentive, sluggish, distracted, evasive, suspicious, and why. Some cases may require prolonged observation. At times irritating questions may be desirable, in order to excite the patient.

5 Intelligence. (Charts III, XIII & XVI)

In testing a patient's intelligence, we test his *general knowledge* by asking him to name the different days and the different months and by arithmetical, geographical, political and historical questions. His *power of observation* by showing him a number of things and asking him later to describe them. His *power of attention* by asking him to add a long column of figures or underscore a letter wherever it occurs in a page of print. His *power of comprehension* by asking him to explain something he has read or heard. His *association of ideas* by giving him a word and asking what other ideas it suggests to him. His *mental reaction time* by the time he takes to solve problems, or to name an object, the picture of which is shown to him. His *moral sense* by questions in ethics.

6 Memory and understanding. (Charts III, XIII & XVI)

An apparent defect in intelligence may be due to lack of attention, or may be shown by further questioning, by having him repeat long phrases, execute verbal and written commands and name objects shown to him, to be due wholly or in part to a loss of memory; either general (amnesia), or local (aphasia), especially to a failure to understand what is said to him (sensory aphasia); while reason and judgment are normal. Test memory for remote, as well as for recent, occurrences. Test his memory of statements made a few minutes previously, or of events of the day before, or of years before.

7 Emotions. (Charts III & XVI)

Patients may show by their conversation, if suitably guided, or by their manner, or by both, whether they are emotional or not. The emotional state of the patient and the mental characteristics discussed just above, can often best be learned from the statements of friends and relatives. Curious fears, the so-called "phobias," (235) are often present.

8 Speech. (Charts III, XIII & XVI)	Patient's speech may be entirely absent (anarthria) or altered and very defective, i.e., rational or irrational; there may be limited vocabulary or use of wrong word (aphasia), poor articulation (dysarthria), tremor in voice, monotonous, scanning speech, omissions of syllables and words. Speech is tested by interrogation and spontaneous (voluntary) speech. Test also patient's understanding of letters, words and phrases spoken to him, his executing spoken and written commands, his picking out objects named; and have patient name objects, give sequences, i.e., numbers, days of week, months, etc., and repeat catch phrases, as "Round the rough and rugged rock the ragged rascal ran," etc.
9 Reading. (Charts III, XIII & XVI)	Ask the patient to read aloud, even short sentences, words, or letters only. Note any defect either in utterance or understanding.
10 Writing. (Charts III, XIII & XVI)	Ask the patient to write, spontaneously, from dictation and from copy. Have him write the names of objects shown him. Note any defect in the character of the writing or in the ideas expressed.
11 Stereognosis. (Charts III, VI & XXII)	Ask the patient to name objects placed in his hand, his eyes being shut, after excluding anesthesia. Even without feeling them all over and without moving them about in his fingers, a normal person should be able to recognize many objects (metals, cloth, etc.) merely laid against the skin of his hand, face, foot, lips, etc. Stereognosis may, therefore, in exceptional conditions, be tested, although less perfectly, in other parts than the hands.
12 Sight. (Charts VI & XIV)	Ask the patient to read small print or Jaeger's test type at reading distance (10 to 16 inches, according to age, refractive conditions, etc.) and Snellen's test letters at twenty feet. If patient cannot read the appropriate line at twenty feet the loss of vision is expressed by the number of feet from the chart at which he can read this line divided by twenty. Thus at ten feet the vision would be expressed by $\frac{1}{2}\frac{0}{0}$. In great defect of vision the patient may be able to see only dimly the hand moved before his eye, or may only be able to distinguish between light and darkness.
13 Color sense. (Chart VI)	Ask the patient to match different colored worsteds.
14 Field of vision for white and colors. (Hemianopia) (Charts VI & XIV)	Place the patient with back to the window or light and have him close his left eye and with his right gaze at the observer's left eye. Then let the observer move his hands about in a plane mid-way between himself and the patient; so that each should see the hand at the same instant as it comes into the field of vision. The observer can see if the patient's eye wanders from his own and recall it. Test left eye in same way. If any defect in field of vision is suspected, use a perimeter. With a perimeter not only the field of vision, but also, by using different colored papers, the color field can be mapped out. Normally the color field is largest for blue, then for yellow, orange, red, green, etc., in the order named. If this order is changed there is said to be an "inversion of the color fields" (849). Normally the lines limiting the different color fields are everywhere separate from each other. If they touch or cross there is "interlacing of the color fields" or "dyschromatopsia" (849).
15 Hearing and tinnitus aurium. (Charts VI & XIV)	The patient's hearing may be tested by voice, watch, or tuning fork. Be sure there is no wax in the ear. Galton's whistle should be used for testing high and low notes. Each ear should be tested separately. Bone conduction is tested by holding watch or tuning fork firmly on skull. Normally a tuning fork, which, held on mastoid ceases to be heard, can still be heard when held close to meatus (Rinne's test). Normally a vibrating tuning fork, held on center of forehead, is heard equally in both ears. If heard best in the deaf ear (positive) the lesion is in external or middle ear. If heard best in the normal ear (negative) the lesion is in inner ear or in auditory nerve (Weber's test). We also ask about ringing in ears (tinnitus aurium).
16 Smell. (Charts VI & XIV)	Ask patient to name from its odor any fragrant substance (such as asafoetida, cloves, peppermint, etc.) held for a moment beneath each nostril in turn, the other being closed. Ammonia and acetic acid should not be used in this test.
17 Taste. (Charts VI & XIV)	Ask the patient to point to the name on a printed card of the taste of a strongly bitter, sweet, salt or sour solution touched from a medicine dropper, or a camel's hair brush, to one side after the other of the protruded tongue. The tongue should be well washed between each test.
18 Sleep.	The amount of sleep which the patient gets in the twenty-four hours is always an important question. Insomnia (agrypnia) is present in many nervous diseases and is apt to be exaggerated by patients; so that their statements should be controlled, when possible, by those of the nurses or relatives. Many symptoms, especially fears, are worse at night: "Pavor nocturnus of children."

CHART Ib

Inspection (mainly)

Comprising Numbers 20 to 42

INSPECTION

Methods of Testing

20 Facial expression and general appearance and behavior. (Charts XVI & XVII)	The expression of the patient's face indicates, in most cases, the degree of his intelligence and his emotional state (sad or gay or anxious), and also may suggest the presence of certain diseases and conditions; such as myxedema and cretinism (1163–4), acromegaly (1183), scleroderma (1165), exophthalmic goitre (1193), paralysis agitans (677), myasthenia (553), nasal obstruction, atheroma of temporal arteries, notched teeth, hazy cornea, and the saddle-back nose of syphilis, etc. His general appearance and behavior often indicate his power of self restraint (inhibitory power, breeding), or the existence of hallucinations (213) of sight, hearing, touch, or of compulsory acts (218).
21 Walk. (Chart XIII)	The walk of the patient may suggest the presence of hemiplegia (254), paraplegia (257), local paralysis (259), ataxia (motor or cerebellar) (248), spasm (242), atony (252), paralysis agitans (677) and other tremors (250), pseudo-hypertrophic paralysis (500), hysterical paralysis (527), foot drop, (bilateral in multiple neuritis and lead palsy, unilateral in acute anterior poliomyelitis), weakness, exhaustion, etc.
22 Skull. (Chart XVI)	The skull should be observed as to type (brachy- or dolicho-cephalic, round or long heads), size (microcephalic—small, macrocephalic—large), rickets (box shaped), general or local hydrocephalus (bulging—posterior or anterior), fontanelles and sutures, asymmetry, tumors, etc.
23 Vertebral column. (Chart X)	The spinal column should be observed as to curvature (angular or lateral), scoliosis, kyphosis, spina bifida (occulta), deformity (dislocation), Pott's disease, tumor tenderness (by palpation), etc.
24 Eye. (Charts V, VI & XIV)	Note the existence of arcus senilis, the condition of pupils (unequal, anisocoria (341), myosis (340), mydriasis (339), and irregularity), the presence of keratitis or iritis, prominence of eyeballs, nystagmus, squint, ptosis, paralysis, etc.
25 Pupillary reflex to light. (Charts V & XIV)	Note whether each pupil, the other eye being covered, dilates and contracts as the eye is alternately shaded by the hand and exposed to light, or an electric light is flashed into it; vision being constantly fixed upon some distant object. When a pupil contracts to light (direct reflex) the pupil of the other eye also contracts (consensual reflex).
26 Hemiopic reflex. (Charts V & XIV	Note whether the pupil contracts as light is flashed on each half of the retina alternately. A ray of light collected by a lens should be used in this test. This reflex is difficult to obtain, and not entirely reliable.
27 Pupillary reflex to accommodation. (Charts V & XIV)	Note whether the pupil dilates when the patient looks at a distant object and contracts when he looks at one so near his face as to require convergence of the eyes. This test can be made on a blind man by having patient first converge his eyes and then make the axes of his eyes parallel, by imagining that he is looking at a near and then at a distant object.
28 Double vision, diplopia. (Charts VI & XIV)	Note which eye deviates, however slightly, from the direct axis of vision and which eye lags more or less on movement of eyeballs in following the moving finger. Place a colored glass before the affected eye, move a bright object (candle) throughout the field of vision and have the patient note the relative position of the two images. The colored image will of course be the one seen by the affected eye.
29 Secondary deviation of the sound eye. (Chart XIV)	Hold a card close in front of the sound eye. Have the patient look at an object so held that the weakened muscle must be brought into action. The sound eye covered by the card will be observed to move too far and when the card is removed the sound eye will quickly move back into proper position.
30 Nystagmus. (Charts IV & XII)	The oscillation of the eyeball which constitutes nystagmus is often plainly to be seen. Extreme deviation of the eyeballs in one direction or the other makes it more evident, and at times demonstrates a nystagmus not otherwise apparent. If present, nystagmus is usually recognized while making the two tests 28 and 29. It should not be confounded with the irregular jerky motion of a weakened ocular muscle attempting to move the eyeball.

31 Tremor. (Charts IV & XII)	Note any tremor of lips, tongue, or other parts of the body. Note its frequency, amplitude, its relation to voluntary movements and whether it is associated with muscular rigidity. In testing for tremor, ask patient to hold arms extended before him or over his head with fingers spread and motionless.
32 Convulsion and spasm. (Charts IV, XI & XII)	Note any convulsion (269), spasm (245–6), contracture (263–4), athetosis (271), choreiform movement (272), etc., which may be present. These various forms of spasm are often difficult to recognize and differentiate from each other.
33 Paralysis (motor). (Charts IV, X & XIII)	Note any obvious paralysis, such as ptosis. Note the naso-labial fold and the height of the angle of the mouth on each side. While under close inspection, patient should be requested to execute every possible motion: i.e., wrinkle forehead (look upward, or open eyelids held closed by observer), frown, open and shut each eyelid, move eyeballs up and down and to either side (note whether upper eyelid follows eyeball well downwards), whistle, laugh, distend cheeks, raise upper lip and each angle of mouth, protrude tongue straight and move it in all directions, raise uvula in phonation, close jaws and move chin forwards and jaw laterally, contract strongly all muscles of face at once, move head backwards, forwards and towards each shoulder and shake it, bend body in all directions, raise arms vertically, raise shoulder, adduct and abduct arm, flex and extend elbow, wrist and each finger, spread fingers, adduct, abduct, flex and extend thumb, pronate and supinate forearm while elbow is flexed, stand on each leg, raise body on tiptoes, adduct and abduct thigh, flex and extend thigh, leg, foot and toes.
34 Paresis. (Charts IV, X & XIII)	Make strong resistance to above mentioned movements while patient is executing them: i.e., pull on eyelids, on one angle of mouth, resist movements of jaw, or of bending head or body, or of flexing, extending, adducting and abducting joints, compare the strength of the paretic muscle with that of a similar healthy one, when possible, with its fellow of the opposite of the body. For future comparison, etc., the strength of the paretic muscle can be registered by dynamometers, of which the most practical is the one for the hand grasp. Or sufficient weights may be placed on hand, foot or head to overcome the attempted movement.
35 Myasthenia. (Chart IV)	Note whether patient tires easily on repeated or continuous activity of any set of muscles.
36 Diadocokinesia. (Chart IV)	Note whether patient can alternately extend and flex joints quickly and repeatedly. Test especially rapid alternate supination and pronation.
37 Ankylosis.	Note whether any joint is rigid, so that it cannot be moved. Ascertain the cause of the rigidity, whether bony union, contracted muscle or contracted scar tissue (muscle, ligament, skin, etc.).
38 Contracture. (Charts IV & XI)	Note whether any muscle is contractured with consequent impaired motility of the joint and whether this contracture can be overcome by force, with or without etherization (active contracture), or not (passive contracture).
39 Muscle tone. (Charts IV & X)	Note whether muscles are firm or flabby, and whether or not resistance is offered to rapid passive motions of joints while the patient tries to make no voluntary resistance. Normally there is slight resistance. In disease the resistance may be altogether absent (atonia), or weak (hypotonia), or strong (hypertonia).
40 Trophic lesions. (Chart XVII)	Note whether any muscle shows atrophy or hypertrophy, or fibrillary contractions, or if there is any arrested development or trophic lesions of other tissues (especially ulcers, herpes, glossy skin, abnormalities of nails, etc.).
41 Co-ordination (synergy). (Charts IV & XII)	Note whether complicated movements are executed in an orderly manner while the patient's eyes are closed. Ask patient to walk, touch point of nose with finger tip, pick up objects, write, touch knee with heel of other foot, hold foot steady in one position, trace a circle in the air with foot, walk backwards, walk along a line, stand on one foot alone, or on both feet close together, either side by side or one in front of the other (Romberg's symptom), stand on tiptoes or on heels, stand on one foot and trace a circle on the floor with the toe of the other foot. All these tests should be made both with eyes open and shut.
42 Muscle and joint sense. Deep sensibility (bathyesthesia, kinesthesia). (Charts VI & XII)	Note whether patient, with his eyes shut, can tell whether his joints are flexed or extended, or can duplicate with one extremity the position in which his other is placed. Note whether he can estimate weights correctly or can grade by weight loaded balls correctly. Note whether he can locate his extremities in space. To test this, his eyes being shut, an extremity after being moved about is held in one position and he is told to turn his head and eyeballs so that when he opens his eyes he shall be looking directly at his thumb or great toe. When he opens his eyes it will be plain to see whether they are directed right or not.

CHART I c

Palpation and Percussion

Comprising Numbers 45 to 68

PALPATION AND PERCUSSION

Methods of Testing

45 Circulation and respiration. (Chart XVII)

Note the color of the skin, the pulsation of arteries in neck, the condition of the jugular veins and the frequency and regularity of respiration, especially Cheyne-Stokes' respiration (435), whether respiration be costal or abdominal, or diaphragm be immobile, unilaterally or bilaterally.

46 Pulse. (Chart XVII)

Note pulse of patient as to frequency, volume, tension (best tested by tonometer or sphygmomanometer) and irregularity in rhythm and force.

47 Difficulties in sensory testing. (Chart VI)

The result of all sensory tests (and the same is true in regard to tests for many mental symptoms) depends upon the patient's truthfulness. Deception is always possible and even with the most truthful patients the tests require much time and the results are often contradictory, especially so in excitable and in uneducated patients, who cannot fix their attention continuously. Nothing should be present to distract the patient's attention and his skin should be warm. In some nervous diseases the patient has occasional, spontaneous sensations which interfere with the tests. Most patients under the education of repeated tests become more acutely sensitive. On the other hand, tests too long continued tire the patient and give rise to contradictory results. It is to be remembered that the sensibility of the skin both for tactile and painful impressions varies greatly in different parts of the body and in different individuals.

48 Tactile sensibility. (Charts VI & XIV)

With the finger tip (or with a smaller and lighter object, such as the head of a pin, a camel's hair brush, a pledget of cotton, a hair, etc.), touch the patient's skin lightly, having told him to say "yes" every time he feels the slightest touch. Or the patient may describe figures (space sense) traced on his skin with ink (to prevent dispute or doubt). A pledget of cotton is better for accurate testing than is the finger tip or a pin, because with the cotton the pressure sense (49) is eliminated. Of course, during all sensory tests the patient's eyes must be closed or covered. In some cases of hysterical anesthesia, if the patient is told to say "no" when she does not feel the touch, she will say "no" only at the instant she is actually touched within the anesthetic (?) area; showing that sensation is not abolished, although it may well be abnormal. Tactile sensibility, or, more properly, "space sense," or "localizing sense," (53), may also be tested with the esthesiometer; a pair of blunt dividers, by which it is noted how far the points may be separated and yet be felt as one. This distance varies greatly in different parts of the body (at the point of tongue it is one m.m., at finger tips two m.m., along back and on upper part of arm and thigh it is sixty-five m.m. The distance is smaller transversely than longitudinally on the extremities. Neither this compass aesthesiometer, nor Herring's aesthesiometer gives more valuable results than the pinhead tests. When mapping out an anesthetic area commence in the anesthetic area and work towards the normal skin. Do the reverse in mapping out hyperesthesia; i.e., from normal skin to hyperesthetic area. The electro-cutaneous test can be more accurately measured, but is of little practical value.

49 Pressure sense. (Chart VI)

Note whether patient can estimate correctly the amount of pressure exercised by the finger pressed against the skin, or by weights laid upon it.

50 Painful sensibility. (Charts VI & XIV)

Note whether patient feels pain when pinched, or when skin is pricked by fingernail, pin-point, or other sharp substance. Many instruments have been devised for measuring more or less accurately the intensity of the painful impression.

51 Retardation of conduction. (Chart VI)

Note whether the painful sensation is felt immediately upon, or some seconds after, the painful contact.

52 Persistence of sensation. (Chart VI)

Note whether the painful sensation persists a longer time, after the painful contact has ceased, than is normal.

53 Localization. (Chart VI)

Note whether the point of contact, tactile or painful, can be localized correctly by the patient either by description or by pointing; his eyes, of course, being shut.

54 Double sensation and polyesthesia. (Chart VI)

Note whether a single tactile or painful contact causes two (double sensation) or more, sensations (polyesthesia).

55 Temperature sense. (Chart VI) — Touch the skin at numerous points alternately with small test tubes, one filled with hot, the other with cold, water, or with hot and cold bodies (spoons) of the same size and form. Certain points of the skin are especially sensitive to heat; others to cold. It is well, therefore, to test for heat and cold separately.

56 Pallesthesia. (Chart VI) — Note whether the patient feels the vibration of a tuning fork (vibration sense) pressed so firmly on the skin that the vibration can be transmitted through the underlying bone (osseous sense).

57 Cutaneous reflexes. (Chart V) — Stroke or scratch, as softly as will suffice, with finger nail or head or point of pin, the skin of the sole of the foot (plantar and Babinski), or a buttock (gluteal), or the inner side of thigh (cremasteric), or the side of abdomen (umbilical), or the hypochondrium (epigastric), or interscapular region (interscapular), or stroke firmly along the postero-internal border of the tibia (Oppenheim's reflex) and note the resulting movement. The muscle itself must be felt and watched in cases where the resulting contraction is too slight to move the part.

58 Mucous membrane reflexes. (Chart V) — Touch with finger, straw, brush, or probe, the cornea or conjunctiva (conjunctival), or mucous membrane of nose (nasal), or palate (uvular), or pharnyx (pharnygeal), and note the resulting movement.

59 Vaso-motor reflexes. (Charts V & XVII) — Note the pallor or redness of the skin, also rapid changes and flushings with or without irritation, such as scratching with a pin or fingernail (dermographia).

60 Ankle-clonus. (Charts V & X) — With leg relaxed, semi-flexed and well supported, strike or press the sole of the foot quickly, firmly and continuously upwards and note whether the foot oscillates or not. This clonus occurs at times spontaneously when the toe and not the heel rests on the floor ("spinal epilepsy").

61 Knee-jerk. (Charts V & X) — While patient is sitting on a chair with legs crossed, or better on a table with legs hanging free, or is lying in bed on his back with knees flexed, strike the ligamentum patellae a sharp blow with the finger, edge of hand, book or percussion hammer and note whether the foot flies forward. The amplitude of the excursion of the foot is not alone a safe guide to infer increase of knee-jerk, but rather its vigor, its quickness, and the presence of two or three additional oscillations as the foot falls back again. Even a continuous oscillation, or clonus, occurs in some cases (the so-called "spinal epilepsy"). More common than this clonus is a simultaneous contraction of the adductors of the other thigh when the knee-jerk is exaggerated. In order to obtain this reflex the observer must make sure that the muscles of the legs are completely relaxed. The extensor femoris muscle must be observed and felt in those cases where the resulting contraction is too faint to move the leg. Knee-clonus may be obtained in suitable cases by grasping the patella from above and pulling it sharply downwards.

62 Achilles reflex. (Charts V & X) — While patient is kneeling in a chair with his feet projecting free, the tendo-Achillis should be strongly struck with a percussion hammer and the movement of plantar flexion noted. Where the patient cannot kneel the leg may be supported in any position which relaxes it and the tendo-Achillis struck.

63 Dorsal foot reflex. (Chart V) — When the dorsum of the foot is struck sharply over the 4th or 5th metatarsal bones, note the dorsal (normal) or plantar (pathological) flexion of toes (Mendel-Bechterew's reflex—320).

64 Elbow and wrist reflexes. (Chart V) — The arm being relaxed, well supported and semi-flexed at elbow the tendons at elbow or wrist are sharply struck.

65 The jaw reflex. (Chart V) — The patient's chin is firmly grasped with finger and thumb or a flat stick is placed in the patient's mouth resting on his lower teeth, the mouth being half open, and then the stick or the hand holding chin is struck sharply downward and the closure of the mouth noted.

66 Kernig's reflex. (Charts V & X) — With thigh flexed at hip and leg flexed at knee, the patient either sitting or lying, the leg should be quickly extended at knee joint and a strong resistance to such extension noted, if present.

67 Mechanical irritability. — Strike the nerve or muscle sharply with the finger or percussion hammer or press the nerve trunk or its tender points.

68 Reinforcement. — The tendon, and to some extent the cutaneous reflexes, can be made stronger and can be often made to appear when apparently absent, by diverting the patient's attention in any way, usually by having him pull strongly on his clasped hands, his eyes being turned to the ceiling or to a picture at the instant the reflex is tested (Jendrassik).

CHART I d

Electricity, Lumbar Puncture, Brain Puncture, Ophthalmoscopy, Thermometry, Caloric Reaction

Comprising Numbers 70 to 80

ELECTRICITY AND LUMBAR PUNCTURE

Methods of Testing

70 Faradism. (Chart VII)

The electrodes should be kept well moistened with warm salt water during the testing. The larger electrode is placed on sternum or back of neck or sacrum; while the smaller electrode, provided with an attachment for making and breaking (opening and closing) the current, is placed over the motor point of nerve or muscle. The secondary current of a faradic battery should be employed and the current should be gradually increased in strength by methods which vary in different batteries, until the faintest distinct contraction of the muscle occurs whenever the current is suddenly closed, the negative electrode being over the motor point. The test should be repeated several times. As the skin becomes moister a less strong current becomes necessary. It is important to make sure of the exact position of the motor point in each case by some preliminary tests and not to let the electrode slip away from this point during the testing.

71 Galvanism. (Chart VII)

With the electrodes arranged as above, first the negative, later the positive, electrode should be placed over the motor point of nerve or muscle and the strength of the current slowly increased by means of the rheostat until the faintest distinct contraction of the muscle occurs whenever the current is closed. The strength of the current causing this contraction, with each electrode in turn over the motor point, should quickly be read from the galvanometer, even before the needle has quite ceased its oscillations. In the same way read from the galvanometer the strength of the weakest current which will cause the faintest distinct contraction, when each electrode in turn is on the motor point and the current suddenly opened.

72 Muscle and nerve. (Chart VII)

In all cases both the muscle and the nerve supplying it should be tested both by faradism and galvanism.

73 Character of the contraction. (Chart VII)

Note the character of the muscular contraction, whether quick or sluggish (degenerative), or showing any peculiarity, and whether it is unusually persistent (myotonic), or whether it rapidly grows feebler under repeated tests (myasthenic).

74 Lumbar puncture. (Charts VIII & XIX)

The patient's body should be bent strongly forwards. Patient should, if possible, sit, but may be lying down. The skin having been thoroughly washed with alcohol, a horizontal line should be drawn from the posterior spine of one ilium to the other and a sterilized fine needle three or four inches long, preferably of platinum and with rather a short bevel, should be inserted between the laminae of the vertebrae immediately below or above this horizontal line. The needle may be inserted in the median line or a little to one side of it and pushed steadily forward and slightly upward until it enters the arachnoid sac when usually the cerebro-spinal fluid will escape in drops. If the needle be pushed too far it can be felt as it strikes the body of the vertebra and it should then be withdrawn about half an inch. It is rarely necessary and sometimes dangerous to attach a syringe and aspirate the fluid. If the needle becomes occluded clear it out with the stylet. It is better not to withdraw more than half an ounce of the fluid. Note the rapidity of escape, whether by drops or in a fine stream (tension), its appearance (cloudy, bloody, purulent). The fluid may be examined chemically (for albumen, sugar, cholin, etc.). A portion of the fluid, especially that containing the fine coagulum which frequently forms, is centrifuged, the clear fluid is carefully poured off and the bottom of the tube scraped and aspirated with a capillary pipette, the content of which is spread on a slide, fixed, stained and examined for cells (lymphocytes, leucocytes, bacteria, etc.). The cerebro-spinal fluid should also be tested for an increase of globulin, indicative of the presence of a syphilitic infection, of ancient or recent date, or of a meningitis, according to the method suggested by Noguchi (419). After lumbar puncture patients should remain quiet in bed during twenty-four hours. Even so, they are apt to suffer from headache, especially if much fluid has been withdrawn, or withdrawn too rapidly. Sometimes the nerve trunks of the cauda equina are injured, causing pain in the legs, but such pains are rarely severe and are of short duration. In some cases, in consequence of the withdrawal of the cerebro-spinal fluid, the medulla and cerebellum have been drawn down into the foramen magnum and death has resulted promptly. Such an accident is only possible in cases of cerebral tumor situated in the posterior fossa of the skull, and therefore lumbar puncture should not be performed in such cases.

BRAIN PUNCTURE, OPHTHALMOSCOPY, LARYNGOSCOPY, THERMOMETRY, AND THE CALORIC REACTION

75
Brain puncture

This operation consists in trephining (with avoidance of the sinuses and large arteries) a small button from the scalp and bone, inserting a very thin needle canula and aspirating a small quantity of the brain substance, or tissue of a tumor, or fluid from a cyst. It has been many times performed and the results have been somewhat encouraging, but it is an operation which should be performed only by an experienced surgeon or neurologist and its detailed description is hardly in place here.

76
Ophthalmoscopy.
(Chart XIV)

Examine the eyes for choked disc or optic neuritis, and for optic atrophy, retinitis, miliary tubercles, etc.

77
Laryngoscopy.
(Chart XIII)

Examine the larynx for evidence of paralysis of one or more or of all its muscles.

78
Thermometry.

It is often necessary to ascertain the temperature of the patient. The thermometer should be well washed in cool water both before and after taking the temperature. In taking the temperature in the mouth, the bulb of the thermometer should be placed well under the tongue and it should be noticed that the lips are held tightly closed during the two minutes that the thermometer is left in the mouth. In taking the temperature in the axilla, the axilla should first be wiped dry from sweat and care should be taken that the thermometer be surrounded by skin and not at all by clothes; the patient should be rolled over on his side in order to press arm firmly against chest and the thermometer should be left in position eight minutes. In taking the temperature in the rectum, a little vaseline or soap-suds should be put on the bulb before inserting it into the rectum, where it should remain two minutes. Instruments have been invented for taking the surface temperature of the skin of any part of the body, but they have not proved to be of much practical value.

79
Caloric reaction.
(Chart XII)

When one ear of a normal person, with head held upright, is syringed out with cool water there results a horizontal and rotatory nystagmus towards the other ear; when water warmer than the body is used, the nystagmus turns towards the syringed ear. This reaction does not occur in cases of destruction of labyrinth, or of paralysis of the vestibular nerve.

80
Cerebellar nystagmus.
(Chart XXI; 1272)

In lesions of the right cerebellar hemisphere, nystagmus to the right may only be seen, or may be made more marked, when the patient lies on the left side, and vice-versa.

CHART II

Analysis of the Subjective Symptoms of the Case

Comprising Numbers 81 to 190

List of diseases most likely to occur as the result of the etiological factors obtained from the history of the case.

- **81 Heredity, including consanguineous marriages in neuropathic families (Predisposing cause)**
  - **84 Inherited Diseases** — Organic Diseases
    - 101 Idiocy and Imbecility
    - 102 Spina Bifida and Meningocele
    - 103 Hereditary (Huntingdon's) Chorea
    - 104 Hereditary (Friedreich's) Ataxia
    - 105 Myatonia Congenita
    - 106 Myotonia Congenita (Thomsen's Disease)
    - 107 Muscular Dystrophies
    - 108 Syphilis of the Nervous System
  - **85 Inherited Tendencies** — Neuroses
    - 109 Insanity
    - 110 Epilepsy
    - 111 Hysteria
    - 112 Chorea
    - 113 Neurasthenia
    - 114 Neuralgia and Migraine.
    - 115 Drunkenness (Alcoholism)
- **82 Personal Factors (Predisposing causes)**
  - **86 Age**
    - Infancy and Childhood
      - 116 Cerebral Palsy of Childhood
      - 117 Acute Anterior Poliomyelitis
      - 118 Meningitis (tuberculous, etc.)
      - 119 Hydrocephalus
      - 120 Tetany
      - And all the inherited diseases except 103 and 106
    - Childhood and Youth
      - 121 Caries of Spine and Compression Myelitis
      - 122 Meningitis (tuberculous, etc.)
      - 123 Hereditary Ataxia
      - 124 Glioma
      - 125 Chorea
      - 126 Epilepsy
      - 127 Muscular Dystrophies
      - 128 Hysteria
      - 129 Insanity
    - Adult — All other forms of Nervous Diseases and many of those above given
  - **87 Sex**
    - More common in women
      - 130 Hysteria
      - 131 Exophthalmic Goitre
      - 132 Neuroses
    - More common in men
      - 133 Locomotor Ataxia (Tabes)
      - 134 Paresis
      - 135 Injuries
      - 136 Organic Diseases
  - **88 Race**
    - Jewish & Latin — 137 Neuroses
    - Anglo-Saxon — 138 Organic Diseases
  - **89 Dwelling Place, Habitation**
    - Tropical
      - 139 Beri-Beri
      - 140 Leprous Neuritis
      - 141 Sleeping Sickness
    - Dampness — 142 Neuritis
  - **90 Occupations**
    - Overstrain — 143 Occupation Neuroses
    - Poisons — 144 Neuritis

- 83 Etiological Factors (Inciting causes)
  - 91 Traumatism
    - Physical
      - 145 Wounds and Injuries
      - 146 Hemorrhage in Brain, Cord or Membranes
      - 147 Meningitis
      - 148 Myelitis
      - 149 Disseminated Sclerosis
      - 150 Neuritis
      - 151 Tumors
      - 152 Abscess
    - Psychical, Acute & Chronic
      - 153 Hysteria
      - 154 Insanity
      - 155 Neurasthenia
      - 156 Traumatic Neuroses
  - 92 Poisons Toxic
    - Metallic
      - 157 Arsenical Neuritis
      - 158 Lead Palsy, Colic, etc.
      - 159 Mercurial Tremor
    - Alcoholic
      - 160 Multiple Neuritis
      - 161 Neurasthenia
    - Tobacco, Tea or Coffee
      - 162 Tremor
      - 163 Neurasthenia
    - Narcotic
      - 164 Drug Poisoning; Acute or Chronic
  - 93 Infections
    - Germs and Toxines
      - 165 Neuritis
      - 166 Meningitis
      - 166 Myelitis
      - 167 Acute Anterior Poliomyelitis
      - 168 Landry's Paralysis
      - 169 Neuralgia
      - 170 Tetanus
      - 171 Hydrophobia
  - 94 Syphilis
    - Tertiary Syphilis
      - 172 Gumma
      - 173 Meningitis Gummosa
      - 174 Neuritis Syphilitica
      - 175 Endarteritis Syphilitica
    - Post-Syphilitic Infections
      - 176 Locomotor Ataxia
      - 177 General Paresis
  - 95 Exhaustion
    - From Illness, Overstrain, Worry
      - 178 Neurasthenia
      - 179 Hysteria
    - From Venery and Masturbation
      - 180 Neurasthenia
  - 96 Extension of Inflammation
    - Caries of Skull or Vertebrae
      - 181 Cerebral or Spinal Abscess
      - 182 Sinus Thrombosis
      - 183 Meningitis
      - 184 Myelitis
      - 185 Neuritis
  - 97 Arterial Disease
    - 186 Apoplexy
  - 98 Metastasis from Other Organs
    - 187 Tumors
    - 188 Tuberculous and Suppurative Meningitis
  - 99 Disease of Other Organs
    - Bright's Disease
      - 189 Uremia
    - Diabetes Mellitus
      - 190 Diabetic Coma
  - 100 Cold is a doubtful direct, but probably an auxiliary etiological factor

CHART III

Disturbances of Mental Activity

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease.

200 Disturbances of Mental Activity.

201
CONSCIOUSNESS
The appreciation of one's existence and individuality as separate from the rest of the universe (Subject consciousness). The content of consciousness is the sum of the present perceptions of the various sensations (Object consciousness), together with the memories of past perceptions and judgments (Experience) (Chart XVI)

202
INTELLIGENCE
The power of ascertaining facts and reasoning upon them. The power of discovering the relation of things and of acquiring knowledge (Chart XVI).

In disease, consciousness and intelligence may be either diminished or perverted as is set forth in Chart III a.

Neither intelligence nor consciousness is exaggerated or increased in disease, although the latter may be apparently so (Self-consciousness). In such cases, however, there is a concentration or limitation of consciousness rather than an increase of it; an exaltation of the subject, with a lowering of the object, consciousness.

203
MEMORY
The power of retaining in the mind and of recalling at will perceptions and ideas formerly received. The more striking the perception and the more frequently it is repeated or recalled, the better becomes its memory (Chart XIII).

204
EMOTIONS
An emotion is a state of consciousness accompanied by a feeling of pain, pleasure, fear, anger, wonder, scorn, etc. In health a person's emotion is usually in harmony with his environment, but in disease it may be quite independent of the environment (Chart XVI).

In disease, memory may be diminished in whole or in part, and the emotions may be either diminished or exaggerated as is set forth in Chart III b.

Memory is never increased in disease, although certain memories may be accentuated and others lost.

CHART III a

Disorders of Consciousness and Intelligence

Comprising Numbers 201 and 202, and 205 to 218

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

CONSCIOUSNESS

Diagnostic Symptoms	Definition	Significance
201 CONSCIOUSNESS		
DIMINISHED		
205 Coma	The patient lies in a profound stupor from which he cannot be aroused by irritation of any sensory organ (eye, ear, skin, mucous membrane, etc.). No voluntary acts are performed and the reflexes are abolished or diminished, except the circulatory and respiratory, which are often, but not always, deranged. Patient is unable to swallow. Lips and cheeks puff out during expiration.	These three conditions are not always sharply differentiated, but may merge into each other. They are due to loss or diminution of brain function in consequence of pressure upon the brain or of circulatory disturbances in it, or of poisons, etc. Occur in traumatism, and in many organic diseases of the brain and its membranes and especially of its blood vessels; also when toxic substances (morphia, etc.) or toxins (fever, etc.) are in the blood; also in Bright's disease and diabetes mellitus. Rarely the condition is functional.
206 Semi-coma or Stupor	The patient is apparently in a coma but by strong sensory irritation can be aroused to some manifestation of consciousness. No voluntary acts are performed, but the reflexes are usually present. Patient can swallow. Patient may lie apparently awake, but really unconscious, with a low muttering delirium (Coma vigil).	
207 Dazed, Bewildered, Somnolence or Sopor	The patient lies in a deep sleep or moves about automatically. Can be rather easily aroused, but does not fully appreciate his surroundings. Can speak more or less intelligently.	
PERVERTED		
208 Erroneous personality	A mental condition in which a person imagines himself to be different from what he really is; sometimes an animal, sometimes a famous character in history, sometimes God, etc.	Occurs in insanity (functional).
209 Double personality	At intervals the patient is in a sort of somnambulistic state and presents an abnormal consciousness and personality. His memory at times changes with his personality, in which case he remembers only occurences in former similar conditions and not those of his normal state, and vice versa. This is a very rare condition and offers much opportunity for deception, and in some cases of hysteria may well be suggested by the examining physician.	Occurs in hysteria and epilepsy (functional).
210 Automatism Somnambulism	A person performs complicated and apparently intelligent acts, while suffering from loss, or great impairment, of consciousness, and retains little or no memory of the acts done.	Brain is probably anemic or exhausted, or the patient is under the influence of a great emotion (fright). Occurs in epilepsy, insanity, hypnotism, and rarely in hysteria (functional); not uncommon in childhood during sleep.

Diagnostic Symptoms		Definition	Significance
202 INTELLIGENCE — DIMINISHED	211 Amentia	Absence or defect of intelligence, which is congenital or is acquired in infancy before the intelligence has developed.	Due to a malformed or diseased brain. Occurs in idiots, imbeciles and feeble minded persons.
	212 Dementia	Absence or defect of intelligence, which is acquired in later life in a person previously intelligent.	Due to atrophy or functional failure or diminution of blood supply of cerebral cortex. Occurs in insanity and is often its terminal stage.
PERVERTED	213 Hallucinations	Vivid perceptions of sensations (visual, auditory, olfactory, tactile, painful, etc.) not directly dependent upon any external corresponding reality; a sensation without an external object. They are usually regarded as real and are then associated with defective judgment and mental impairment, and therefore cannot be corrected.	Due to disease of the cerebral cortex, whether functional, circulatory, toxic or organic. Usually symptoms of insanity, or of extreme degree of neurasthenia, are also present. In insanity these perversions of intelligence cannot be corrected by reason and demonstration, and in neurasthenia only rarely and imperfectly.
	214 Illusions	Erroneous perceptions. A false interpretation of an actual sensation, which is really of a different nature from that which the patient believes it to be. Frequently occur in rational persons, especially in those with defective terminal sensory organs. In such cases easily corrected.	
	215 Delusions	Erroneous judgments (often, but not always, dependent upon hallucinations) which can be corrected neither by reason, nor by the evidence of the senses and which are not in accord with universal human experience, and are the consequence of mental enfeeblement. Delusions are systematized or unsystematized according as they are supported or explained by more or less coherent reasoning, or not. The systematized delusions are of much more serious prognosis.	
	216 Hypochondriasis	Delusions of imaginary symptoms and illness formed on an insufficient basis of abnormal sensations, which cannot be corrected and are associated with much mental depression.	
	217 Delirium	Irrational talk in persons with diminished consciousness. Probably due in most cases to hallucinations, illusions and mental confusion; consequently its irrationality may be in part only apparent. Often occurs in fevers.	
	218 Compulsory ideas and actions (275)	Certain thoughts or questions or doubts, which are forever in the patient's mind and cannot be removed. They may be of any nature. Patients are irresistibly compelled by an unknown force to do certain acts or to say certain words, usually quite trivial. Patients recognize the abnormal character of these ideas and acts and are made very unhappy by them, but are quite unable to prevent them.	

Methods for the detection of disorders of consciousness and intelligence are described in Chart I a.

For further discussion of these symptoms and the diseases in which they occur see Chart XVI.

CHART III b

Disorders of Memory and Emotions

Comprising Numbers 203 and 204 and 220 to 237

MEMORY

Diagnostic Symptoms	Definition	Significance
220 Amnesia	Inability to recall former perceptions and ideas. Loss of memory in general. May be more or less extensive. May affect memories of the immediate, or of the remote, past.	Functional or organic disease of the cerebral cortex, often anemia, sometimes the result of fright.
221 Motor aphasia	Inability to express by words some idea in the patient's mind, although there is no paralysis of the vocal organs and the patient can usually express the idea by gesture. A loss of memory of how to speak (innervation memories), especially names. A limitation of the vocabulary.	Lesion in or near base of left inferior frontal convolution and anterior portion of left island of Reil in right handed persons, and of the right side in left-handed persons.
222 Sensory or Auditory aphasia (word deafness)	Inability to understand (although not deaf) spoken words formerly intelligible. Loss of memory of words formerly heard. Hence inability to recognize them when spoken (233).	Lesion in or near posterior part of left superior temporal convolution and posterior portion of left island of Reil in right handed persons.
223 Optic aphasia	Inability to name objects, which the patient sees clearly, although he can name them after feeling them. Loss of visual memories (232).	Lesion of left occipital lobe or of association fibers from this lobe in right handed persons.
224 Mixed aphasia	A mixture of the three forms of aphasia just described.	Any one or a combination of the above lesions, or a lesion of the island of Reil, or of external capsule in right handed persons, in whom the above lesions are always in the left cerebral hemisphere, or, in slight degree, may result from carelessness.
225 Paraphasia (Jargon speech)	The use of a wrong word, or the omission of a word, or the placing of the right word in the wrong place, in speaking, with consequent incoherent speech. Jargon speech is an extreme degree of this.	
226 Paragraphia	The use of a wrong word, or the omission of a word, or the placing of the right word in the wrong place, in writing.	

DIMINISHED MEMORY 203

227 Agraphia	Inability to express in writing the idea in the patient's mind, although he formerly could do so and his right arm and hand are not paralysed.	Lesion in the base of the middle left frontal convolution, cortical or subcortical.
228 Alexia (Word blindness)	Inability to read words patient could formerly read, although he sees them clearly and there is no paralysis of his vocal organs.	Sub-cortical lesion beneath left angular convolution in right handed persons.
229 Astereognosis	Inability to recognize objects by the sense of touch, although there is no anesthesia present in sufficient degree to prevent it.	Lesion in or near cortex, or sub-cortex, of contralateral posterior central convolution.
230 Apraxia	Inability to execute a desired act. Loss of skill in executing acts, although there is no motor paralysis present. Loss of innervation memories necessary to perform these acts.	Cortical, or sub-cortical, lesion of motor area of contralateral hemisphere.
231 Agnosia	Inability to recognize objects through some organ of sense which is itself normal. This may be due to failure of full perception or to loss of special memories.	Cortical, or sub-cortical, lesion of sensory area of cortex of contralateral cerebral hemisphere.
232 Psychic blindness	Inability to recognize well known objects or to comprehend familiar things by sight, although the patient is not blind. Loss of visual memories, optic aphasia (223).	Cortical, or sub-cortical, lesion of left occipital lobe, except in region of calcarine fissure.
233 Psychic deafness	Inability to recognize and comprehend well known words and sounds, although the patient is not deaf. Loss of auditory memories. Includes sensory aphasia (222).	Cortical, or sub-cortical, lesion in left superior temporal convolution in right handed persons.

	Diagnostic Symptoms	EMOTIONS Definition	Significance
204 EMOTIONS — EXAGGERATED	234 Sadness (Melancholia)	Without adequate cause the patient is depressed and unhappy. There is a great repression of mental and physical activity usually. He can be influenced little, if at all, by reason; difficult to get his attention.	
204 EMOTIONS — EXAGGERATED	235 Fear (Phobias)	Without adequate cause the patient is in constant fear of an impending calamity, or has an unformulated fear. He dreads to cross an open space (agoraphobia), or to enter a small room or confined space (claustrophobia), or fears a storm (astrophobia), or syphilis (syphilophobia), or ill-timed urination (urophobia), or everything (pantophobia), etc. Can be influenced little, if at all, by reason. Frequently has a more or less unconscious sexual basis.	Functional or circulatory disturbance of cerebral cortex, especially cerebral exhaustion. Occurs in neurasthenia and especially in insanity. Fears and apprehension seem to be the basic symptoms of many forms of incipient insanity (Mosher).
204 EMOTIONS — DIMINISHED	236 Joy (Mania)	Without adequate cause the patient is exhilarated. There is great exuberance of mental and physical activity. Careless and destructive. Can be influenced little, if at all, by reason. Difficult to get his attention.	
204 EMOTIONS — DIMINISHED	237 Apathy	Without adequate cause patient is in a dull stuporous condition. No expression of physical or mental activity. An automaton, submitting passively to whatever is done for him.	

Methods for the detection of disorders of memory and emotion are described in Chart I a.

For further discussion of these symptoms and of the diseases in which they occur see Charts XIII and XVI.

CHART IV

Disorders of Voluntary Motion

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)
Definition, Significance and Relationship of the Symptoms of Disease.

240
DISORDERS OF VOLUNTARY MOTION

The power of executing movements by an effort of will is acquired in early life. The process is quite obscure, but seems to depend upon the existence of innervation memories of past acts, primarily reflex. Voluntary motion depends upon the integrity of the central motor neurons (461) and of the peripheral motor neurons (462). In disease the power of voluntary motion may be diminished, exaggerated or perverted.

MUSCULAR TONICITY

Closely connected with the power of voluntary and involuntary action is the fact that the muscles of a normal person are in a condition of constant, slight, but varying, contraction. This is called muscular tonicity or tone. It is really a reflex act caused and maintained by many slight irritations, and can be abolished by cutting the posterior nerve roots. Muscular tonicity is increased (hypertonia) in destructive lesions of the central motor neurons and in some functional disorders. It is diminished (hypotonia,) or abolished (atonia,) in destructive lesions of the peripheral motor or sensory neurons, in lesions of the cerebellum, in sleep and in narcosis.

241
DIMINUTION
also called
AKINESIS and
HYPOKINESIS

244
PARALYSIS
A condition in which the muscles cannot be concontracted by the strongest effort of the will. As commonly used the term includes:

PARESIS
A condition in which the muscles can be contraced only feebly by the strongest effort of the will.

The conditions under which paralysis or paresis occur are set forth in Chart IV a.

242
EXAGGERATION
also called
HYPERKINESIS

245
TONIC SPASM
A continuous, involuntary, muscular contraction of longer or shorter duration (572).

246
CLONIC SPASM
More or less rhythmical alternations of involuntary, coarse, violent muscular contractions and relaxations (571). Must not be confounded with a coarse tremor.

247
IRREGULAR SPASM
Involuntary acts of various kinds (292, 573-4).

The conditions under which the various forms of spasm occur are set forth in Chart IV b.

243
PERVERSION
also called
PARAKINESIS

248
ATAXIA
Disorderly movements due to loss of power of coordination (638). Asynergia. Associated with hypotonia (252)

249
LOSS OF SKILL,
APRAXIA
Awkwardness.

250
TREMOR
Involuntary rhythmical oscillation of some part of the body or of a muscle. Less powerful, more rapid and more rhythmical than a clonic spasm but similar in appearance, especially when coarse. Tremor may be slow (3 to 6 per second) or rapid (8 to 12 per second). It may be coarse or fine (639).

The conditions under which the various forms of perversion of motion occur are set forth in Chart IV c.

CHART IV a

Motor Paralysis

Comprising Numbers 244 and 251 to 260

244 PARALYSIS { CHARACTER / EXTENT

MOTOR PARALYSIS

PARALYSIS — CHARACTER

Diagnostic Symptoms	Definition	Significance
251 Spastic, or hypertonic, paralysis. (473). (Figs. 24–6)	A paralysis in which the muscles show increased tone and offer much resistance to passive motion, especially rapid motion. The normal excursion of the joint is restricted. The muscles have their normal volume and under the microscope their fibers show a normal appearance. The electrical reaction of muscle and nerve is normal (396). The tendon reflexes are increased.	Destructive lesion of central motor neurons (461). It occurs in diseases of the brain or spinal cord, or may be functional. Rarely a reflex spasm (268), especially preputial irritation in children, or pain, may simulate this condition.
252 Flaccid, or hypotonic, or atonic, or atrophic paralysis (472). (Figs 24–6)	A paralysis in which the muscles have lost their tone and offer little or no resistance to passive motion, even when rapid. The joint has a normal or even increased excursion. The muscles exhibit a great and rapid atrophy, and under the microscope their fibers show a loss of their transverse striation and various forms of degeneration (fatty, hyaline, etc.). The electrical reaction of degeneration is present (399). When muscles are completely degenerated (404) passive contractures (263) may occur. The tendon reflexes are abolished or diminished.	Destructive lesion of peripheral motor neurons (462). It occurs in diseases of the muscles, peripheral nerves, anterior horns of cord, or motor nuclei in brain stem. It is never functional, but may be somewhat simulated by joint disease. Hypotonia without muscular paralysis or atrophy occurs in cerebellar lesions, tabes and other ataxic conditions (240).
253 Myasthenic paralysis (553)	A rapid tiring of muscles upon exercise. A myasthenic reaction to electricity (401). Muscles show small foci of small round cells.	A lesion of the muscles and often of thymus gland.

PARALYSIS — EXTENT

Diagnostic Symptoms	Definition	Significance
254 Hemiplegia (478-9) (Figs. 17–24)	A paralysis with exaggerated tendon reflexes, of one lateral half of the body and extremities limited by the median line in front and behind. It is partial, if limited to arm and leg; complete, if arm, leg, tongue, palate and face are all involved. In some cases of hemiplegia there are slight weakness and exaggerated reflexes on the other side of the body also, especially in the leg. Symmetrical, bilateral muscles, which have a common function and a bilateral cortical innervation, are not paralysed; at most temporarily weakened. Such are the ocular, masticatory, laryngeal, respiratory, bladder, rectal, etc., muscles. In cerebral hemiplegia certain muscles are, in most cases, more completely paralysed than others. These "predilection muscles of Wernicke" are the trapezius, the external rotators and adductors of the upper arm, the triceps, the supinators and abductors of thumb, the extensors of the thigh, the flexors of the leg and the dorsal flexors of the foot.	A lesion of the contralateral central motor neurons (461). In extremely rare cases the lesion may be homolateral (homolateral hemiplegia), in which cases the pyramidal tract may not decussate in the medulla. Hemiplegia is usually due to a cerebral lesion, but the partial form may be due to a bulbar or spinal lesion, very rarely. Very rarely, there may be no lesion, except an extreme local anemia or edema of brain as in nephritis (hemiplegia sine materia).
255 Diplegia (478)	A double hemiplegia involving both sides. May be complete or partial and not infrequently is limited to the legs, or the face (facial diplegia), etc.	A lesion, usually but not always cortical, of the central motor neurons or basal nuclei on both sides.
256 Crossed paralysis (537-42) (Hemiplegia alternans) (Figs. 20–1)	A paralysis of one or more homolateral cranial nerves and of the contralateral arm and leg.	Always due to a lesion involving the pyramidal tract with other structures in the brain stem (460); either in the medulla (hypoglossal hemiplegia alternans (1268)), the pons (facial hemiplegia alternans (1269)), or in the crus cerebri (motor oculi hemiplegia alternans (1270)). The nuclei, or the neurons, peripheral or central, of the cranial nerves are involved below the decussation of their central neurons.
257 Paraplegia (480) (Figs. 24–6)	A symmetrical paralysis of both sides of the body. Usually only involves the legs and lower part of body, but may involve the arms and even both sides of the face.	May occur in lesions of the muscles (dystrophies) (477), or of the peripheral nerves (neuritis (488-9)), or of the spinal cord or brain stem, or even of the cerebral cortex (bilateral lesion). The distinction between paraplegia and diplegia (255) is not always sharply drawn. In general diplegia is applied to paralyses of cerebral origin, paraplegia to those of spinal or peripheral origin.
258 Monoplegia (479) (Fig. 15)	A paralysis of one extremity only, or of one half of the face only.	May be due to lesion of motor cerebral cortex, or of the motor nuclei, or of the peripheral nerves.
259 Local paralysis (481) (Fig. 15)	A paralysis limited to one or more muscles of the face, eye, mouth, neck, body or extremities. Less than a whole extremity.	May be due to lesions of muscles or of peripheral nerves, or of spinal cord, or rarely of motor cerebral cortex, or functional.
260 Aphonia (737-8)	Inability to produce vocal sounds. Absence of voice, but whispering is possible.	A variety of local paralysis.	Laryngeal paralysis, organic or functional.

Methods for the detection of paralysis and paresis are described in Chart I b.

For further consideration of these symptoms and of the diseases in which they occur, see Chart X.

CHART IV b

Spasm

Comprising Numbers 245 to 247 and 263 to 276

SPASM

	Diagnostic Symptoms	Definition	Significance
245 TONIC SPASM	263 Passive contracture (Figs. 24–6)	A continuous contraction of long duration in which the muscles, tendons and ligaments have become anatomically shortened and cannot be extended by force, even under etherization. The muscle fibers are degenerated, while the connective tissue of the muscle is hypertrophied and usually secondarily contracted, as in other newly formed connective, or scar, tissue.	Due to muscular lesions and to degeneration of the peripheral motor neurons (462).
	264 Active contracture (Figs. 15, 17, 24–6)	A continuous contraction lasting weeks, months, or years, which can be overcome by force, either with or without etherization. Muscles are in a normal condition of nutrition. Most common in the arms, or legs, or neck muscles (torticollis). The active contracture of a hemiplegia is usually that of flexion in the arm and of extension in the leg.	Active contractures occurring in hemiplegia affect the muscles not absolutely paralyzed. When the contracture is overcome by the application of a plaster of Paris splint, the muscles often show a surprising degree of voluntary motion, when the splint is removed. These contractures depend in part, on attempts at voluntary movements and on associated movements, but in greater part on reflex action from sensory irritation; the inhibitory action of the brain being cut off by the lesion. They never occur in hemiplegia in tabetics and in any case can be relieved by section of the posterior nerve roots. Such contractures are always of very bad prognosis as to recovery.
	265 Myotonia (613)	An active contracture of brief duration but much longer than a convulsive tic. It may occur at the commencement of voluntary motion (Thomsen's disease, or myotonia congenita) or may be excited by cold (Eulenberg's disease, or paramyotonia congenita). It is frequent in meningitis and tetanus in which it takes many forms, viz: "retraction of head;" "trismus," strong closure of jaw; "opisthotonus," arching of body backwards; "pleurosthotonus," bending of body to one side; "emprosthotonus," arching of body forwards and "orthotonus," holding of body rigid and straight.	
	266 Rigidity	An active contracture of such mild degree that it does not prevent passive, or even voluntary, motion of the part, although rendering it difficult (paralysis agitans (612)), etc.	Active contracture is sometimes due to paralysis of antagonist muscles or to muscle lesions.
	267 Convulsive tics (601)	A violent spasm of momentary duration. If rapidly repeated it must be classed under myoclonus (270 and 601-4). If painful it is called 'tic douloureux" (602).	All tonic spasms (not including passive contracture) are due to a functional disorder, or are reflex (especially in children), or are due to irritation (chemical, sensory or vascular) of central motor neurons (461). Painful cramps, especially in legs, of the nature of myotonia or tics, may be due to a deficiency of water in the system.
	268 Reflex spasm	A spasm, usually tonic, caused by irritation of some sensory tissue.	

	Diagnostic Symptoms	Definition	Significance
246 CLONIC SPASMS	269 Convulsion (571)	Violent clonic contractions of many, or of all the, muscles of the body.	Clonic spasms are usually due to irritation of the cerebral cortex, but may also result from very exaggerated reflexes (clonus).
	270 Myoclonus or convulsive tics	Successive clonic contractions of one, or of a few adjacent muscles. Repeated convulsive tic. Most common in the face muscles (blepharospasm (601)).	
247 IRREGULAR SPASMS	271 Athetosis or mobile spasm (574)	Slow, worm-like, rhythmical movements, often associated with transitory contractures (spasmus mobilis), of fingers and wrists and more rarely of toes and ankles. Hyperextension is the predominant action. Usually unilateral, but may be bilateral. Much more common in children than in adults. Muscles of the neck, face and of other parts of the body are not infrequently involved.	Lesion is usually in posterior part of optic thalamus or corpus striatum of contralateral hemisphere and not causing complete paralysis. Lesion may involve the fibers connecting the optic thalamus with the cerebral cortex. May occur in diffuse cortical lesions.
	272 Choreic movements Chorea minor (573)	Rapid, irregular, co-ordinated, but purposeless movements caused by contraction now of one group of muscles, now of another, throughout the body; bilateral or unilateral (hemichorea). Cease during sleep. They often render voluntary movements ataxic and are usually associated with a mild degree of paralysis of the muscles involved.	
	273 Chorea major or magna (628)	Patient performs involuntarily and uncontrolably a complicated and apparently purposeful movement. Also applied to a coarse tremor or violent oscillation of a part of the body.	Functional disorders, occuring in the neuroses and in insanity.
	274 Habit chorea (626)	Patient frequently performs involuntarily, and usually unconsciously, the same act. Usually a small act.	
	275 Compulsory acts (218)	Patient is compelled by some power within him which he cannot understand or explain to perform certain acts against his will.	
	276 Associated movements	Muscular contractions, occurring when movements are executed or attempted, in muscles not directly concerned in the movement attempted; often the corresponding muscles of the opposite side of the body, often those of the face. Such associated movements are Bell's phenomenon (444), Strümpell's tibialis phenomenon (445), Babinski's associated movements in unilateral paralysis (446).	In such cases movements often associated together, but which can be easily dissociated voluntarily in health, cannot be dissociated in disease which cuts off voluntary action.

Methods of detection of spasm are described in Chart I b.

For further discussion of these symptoms, and of the diseases in which they occur, see Charts XI and XII.

CHART IV c

Perversions of Motion
Ataxia, Loss of Skill, Tremor

Comprising Numbers 248 to 250 and 280 to 293

ATAXIA—LOSS OF SKILL

Diagnostic Symptoms	Definition	Significance
248 ATAXIA		
280 Motor ataxia (644) (dynamic ataxia) (Figs. 24–6)	Voluntary movements are executed in an irregular and disorderly manner, which is due to a loss of the co-ordinating power. Rarely associated with decided vertigo.	Is due to a loss of muscle sense (42) (deep sensibility). May be due to lesions of peripheral sensory nerves, or of posterior columns of cord, or of brain stem, or of cerebral cortex posterior to fissure of Rolando, or may be toxic (alcohol), or functional.
281 Cerebellar ataxia (642) (static ataxia) (Figs. 19–26)	Walking and standing are inco-ordinate, but other acts are not, or only slightly so. Patient executes simple movements of his legs fairly well when lying in bed, but in walking and standing he lacks synergy of the muscles and staggers and sways like a drunken man. Usually associated with vertigo (392).	Is due to a lack of muscular synergy (41) (asynergy). Due to lesion or functional disorder of the cerebellum or its tracts, including the direct cerebellar tract in brain stem or cord, or to tumors in frontal lobe of brain, or to disease of ears or eyes, or to poisons (alcohol, etc.). In lesions of the cerebellar hemisphere the disorder is transitory; in lesions of the worm it is more permanent.
249 LOSS OF SKILL		
282 Apraxia (Fig. 15)	Inability, or difficulty, in performing a desired and accustomed act because of loss, or derangement, of the innervation memories concerned in that act. Loss of skill.	Loss of innervation memories, general or partial, due to cortical or subcortical lesions (anterior or posterior central, or supra-marginal convolution) or to functional or anemic disorders of cerebral cortex. (See page 26-7).
283 Anarthria (737)	Absence of speech. Speech may never have been acquired, as in idiocy, or it may be voluntarily restrained for a purpose; or it may be more or less involuntarily restrained, as in insanity or hysteria.	May be either functional or organic and, if the latter, may or may not be due to lesions in the organs of speech. If not, it is called pure motor aphasia or aphemia.
284 Dysarthria (738)	Such difficulty in articulation that speech becomes indistinct and blurred, but is probably never so great as to cause complete anarthria (283).	Occur in lesions of the medulla and pons (bulbar paralysis, Figs. 21–3) and of the cranial nerves. Also in diphtheria, hydrophobia, myasthenia gravis, rarely in trichinosis and frequently in hysteria (globus hystericus).
285 Dysphagia	Difficulty in swallowing.	
286 Dysmasesis (553)	Difficulty in mastication.	
287 Astasia and Abasia (653 and 792)	Complete inability to stand or walk but legs can be moved freely, even strongly, when lying or sitting.	A delusion or auto-suggestion, which occurs in hysteria. May occur rarely in cerebellar lesions.
288 Diadocokinesia (36)	Difficulty in repeating a movement rapidly, especially supination.	Occurs in lesions of a cerebellar hemisphere, or is functional.

TREMOR

250 TREMOR

Diagnostic Symptoms	Definition	Significance
289 Passive tremor (646 and 647)	Involuntary, rhythmical oscillation or trembling of a part which is otherwise at rest.	Functional. Occurs in paralysis agitans, weakness, etc.
290 Intention tremor (645)	An involuntary tremor which only occurs when a voluntary motion is made, or is willed and is about to be made.	Functional and organic. Occurs in neuroses and in organic diseases (disseminated sclerosis)
291 Nystagmus (640)	An involuntary trembling or oscillation of eyeball, usually horizontal, rarely vertical, very rarely rotatory. Increased, or only occurs, on voluntary motion of eyeball, especially on extreme deviation. The rapidity of the oscillations varies from 60 to 200 per minute. Their amplitude from 2 to 4 millimeters. Nystagmus may be oscillatory when the motion in each direction is equally rapid, or rhythmic when it is quicker in one direction than in the other.	Occurs especially in lesions of the vestibular and other nuclei in the pons, Deiter's nucleus in the cerebellum, the posterior longitudinal bundle in the brain stem, in disturbances in the semi-circular canals, and in weakness of ocular muscles, and in lesion of ponto-cerebellar angle; also in the caloric reaction (79), and in cerebellar disease (80).
292 Fibrillary contraction or fibrillation (641)	An involuntary contraction of a bundle of fibers of a muscle of short duration. When many occur in adjacent bundles at short intervals, waves of contraction run over the muscle, but do not cause it to contract as a whole.	Degeneration of those multipolar nerve cells in the anterior horns of the spinal cord and brain stem of which the motor nerves supplying the muscle are the axons. Rarely occurs in traumatic neuroses.
293 Myokymia (697)	A fibrillary twitching of the muscles occurring in healthy persons.	Normal. Exhaustion. Following excessive muscular contraction or exposure to cold.

Methods of detection of perversions of motion are described in Chart I b.

For the further discussion of these symptoms and of the diseases in which they occur, see Chart XII.

CHART V

Reflex Activity

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the symptoms of disease.

296 REFLEX ACTS

An involuntary movement caused by irritation of a sensory nerve or terminal organ. Although not the result of conscious intention, yet these acts seem purposeful and usually tend towards the protection of the body. In order that a reflex act may take place there must be a comparatively healthy reflex arc, consisting of a motor nerve, a sensory nerve and some gray matter connecting the two; or, in other words, a motor neuron and a sensory neuron connected together directly or by a bridging neuron. Reflex acts are inhibited and modified by inhibitory impulses passing down from the brain along the so-called inhibitory fibers, which are also the central motor neurons (the pyramidal tract) (472-4, 810). (Figs. 19, 24).

297 CUTANEOUS OR SUPERFICIAL REFLEXES

A reflex act which originates from an irritation of the skin (57).

298 MUCOUS MEMBRANE REFLEXES

A reflex act which originates from an irritation of a mucous membrane (58).

299 TENDON OR DEEP REFLEXES

A reflex act which originates from the sudden stretching of the fibers of a muscle (60-6).

300 ORGANIC REFLEXES

A reflex act affecting one of the viscera of the body (1), especially the bladder or rectum.

301 VASO-MOTOR REFLEXES

A reflex act affecting the arterioles (59).

The conditions in which reflex acts are disordered are set forth in Chart V a.

302 PUPILLARY REFLEX

A reflex act affecting the pupil (25-7).

The conditions in which the pupillary reflexes are disordered are set forth in Chart V b.

CHART V a

Cutaneous or Superficial Reflexes, Mucous Membrane Reflexes, Tendon or Deep Reflexes, Organic Reflexes, Vaso-Motor Reflexes

Comprising Numbers 303 to 326

CUTANEOUS REFLEXES

Diagnostic Symptoms	Definition and Location of Reflex Centers
303 Plantar	Plantar flexion of the toes when the sole of the foot is irritated. (1st and 2nd sacral segments.)
304 Babinski's	Sluggish extensive dorsal flexion of the great toe when the sole of the foot is irritated.
305 Gordon's	Dorsal flexion of the great toe when deep pressure is made through the calf muscle on the deep flexor muscles beneath; the leg being completely relaxed.
306 Oppenheim's	Dorsal flexion of the great toe elicited by firm stroking with a hard object, or finger, just behind the postero-internal border of the tibia from above downwards; the leg being completely relaxed.
307 Gluteal	Contraction of the buttocks when the skin covering them is irritated. (4th and 5th lumbar segments.)
307a Anal	Contraction of sphincter ani upon pin pricks of anus. (5th sacral segment.)
308 Cremasteric	Drawing up of the testicle when the inner side of the thigh is irritated. (1st to 3rd lumbar segments.)
309 Umbilical	Sudden movement of umbilicus towards the side of abdomen irritated. (8th to 12th dorsal segments.)
310 Epigastric	Sudden retraction of epigastrium when the hypochondrium is irritated. (7th to 9th dorsal segments.)
311 Interscapular	Drawing inwards of the scapula when the skin of the interscapular space is irritated. (5th cervical to 1st dorsal segments.)
312 Corneal or conjunctival	Closing of the eyelids when the cornea or conjunctiva is irritated. (5th to 7th cranial nuclei.)
313 Nasal	Sneezing when the nasal membrane is irritated. (5th to 10th cranial and upper cervical nuclei.)

Significance

The abnormal reflexes, Babinski, Gordon and Oppenheim reflexes and ankle-clonus, always indicate disease of the central motor neurons (461), except in infants, in whom these reflexes (except ankle-clonus) may be present normally, and in some cases of hysteria in which an imperfect ankle-clonus may rarely be obtained. The Babinski reflex is most reliable in a diagnostic sense. The Oppenheim reflex is sometimes present when the Babinski is absent and vice versa. Kernig's sign indicates meningitis or meningismus; it is an important, but not a certain, diagnostic sign.

Alterations in the tendon reflexes are of very much greater diagnostic value than are those of the cutaneous (except the Babinski) reflexes, which are in many cases inconstant, probably because the cutaneous reflex impulses may even pass through the gray matter of the brain (cerebellum) as well as through a wide area of that of the spinal cord.

Diminution of reflexes is usually of little diagnostic value, but their abolition is of great value and may be due to a destructive lesion of any part of the reflex arc (a peripheral motor neuron, a peripheral sensory neuron, or a central bridging neuron). When there is a lesion of the peripheral motor neuron, atrophic motor paralysis is present in addition to the loss of the reflex. When there is a lesion of the peripheral sensory neuron there is usually a sensory paralysis (anesthesia, etc.), in addition to the loss of the reflex. Diminution or abolition of reflex activity (cutaneous or tendon) may occur, at least temporarily, in acute diseases or other forms of irritation of the central motor neurons; also in cases of shock, exhaustion, coma, narcotism and after epileptic fits, (except Babinski); also by will power and by voluntary movements of the muscles concerned; also (except Babinski) in cases of complete separation of the brain from the spinal cord, and, rarely, of increased intracranial pressure, also frequently in fevers.

The abolition of the knee-jerk is of great diagnostic importance. It is absent in tabes, neuritis (multiple and crural), acute anterior poliomyelitis involving the extensor cruris, Landry's paralysis, lesion of the cauda equina or of the lumbar enlargement, during the attack of family periodic paralysis, when exhausted after an epileptic attack and in cases of muscular dystrophy involving the extensor cruris muscles. It is usually abolished in Friedreich's ataxia and combined sclerosis except in the early stages when it may be increased. It may be absent in cerebral compression (tumor or meningitis) and in some cases of cerebellar disease, and may then be unilateral. It may be absent also in the conditions mentioned in the preceding paragraph.

Diagnostic Symptoms	Definition and Location of Reflex Centers	Significance
314 Uvular	Raising of the uvula in phonation or upon irritation of its mucous membrane. (9th to 10th cranial nuclei.)	Exaggeration of the reflexes may be due to a mild inflammation, or to any irritation, of any part of the reflex arc. Strychnine increases reflex activity by irritating the nerve cells in the anterior horns. More commonly the reflexes are increased by any lesion of the motor central neurons, thus cutting off the normal inhibitory influence of the brain, and are then associated with paralysis of voluntary motion. The presence of ankle-clonus, the Babinski reflex and the dorsal foot reflex indicates a lesion of the pyramidal tract much more certainly than does an exaggerated knee-jerk, unless the latter is associated with an adductor contraction. Very commonly the reflexes are increased in functional diseases (hysteria) and in nervousness.
315 Pharyngeal	Retching or gagging when the pharynx is irritated. (9th to 10th cranial nuclei.)	
316 Ankle-clonus	Oscillation of the foot when the ball of foot is pressed quickly and continuously upwards. (5th lumbar and 1st sacral segments.)	
317 Achilles reflex	Sudden plantar flexion of foot when the tendo - Achillis is sharply struck. (1st to 2nd sacral segments.)	
318 Knee-jerk	Sudden extension of knee when the ligamentum patellae is sharply struck. When this reflex is exaggerated it is usually accompanied by a contraction of the adductors of the opposite thigh, or even by knee clonus (61). (2nd to 4th lumbar segments.)	Innervation of the muscles not concerned in the reflex act and diverting the attention increases reflex activity (reinforcement, 68).
319 Kernig's sign	Resistance to sudden extension of the knee.	The paradoxical reflex is of no diagnostic importance. It consists in a contraction of the tibialis instead of the calf muscles when ankle-clonus is tested for; also of a contraction of the flexors instead of the extensors of the thigh when the knee-jerk is tested for.
320 Dorsal foot reflex	Sudden plantar flexion of the toes when the dorsum of the foot over the 4th and 5th metatarsal bones is struck. (5th lumbar and 1st sacral segments.)	In the dorsal foot-reflex (Mendel-Bechterew) normally there is either no reflex or a dorsal flexion of the toes, but in cases of pyramidal tract lesions a plantar flexion of the toes occurs.
321 Elbow and wrist reflexes	Sudden extension or flexion of elbow or wrist when the corresponding tendons are sharply struck. (5th to 7th cervical segments.)	
322 Maxillary reflex	Sudden closure of jaw when it is sharply struck downwards. (5th cranial nucleus.)	Inability to void urine, or to retain it, is sometimes due to nervousness and sometimes to mechanical obstruction (enlarged prostate or stricture), but any other serious disturbance of the organic reflexes indicates organic disease of the nervous system. It never occurs in diseases limited to the peripheral nerves, except in lesions of the cauda equina, and rarely in cerebral disease. It is most common in spinal disease, sphincter paralysis with empty bladder and constant dribbling of urine in lesions of lumber enlargement, and detrusor paralysis with distended bladder and often with dribbling of urine in lesions above the lumbar enlargement. (Fig. 28.)
323 Bladder or vesical reflex	The retention of urine in the bladder by the sphincter reflex, and the expulsion of urine by the detrusor reflex and the synchronous relaxation of the sphincter. (Hypogastric sympathetic ganglia.)	
324 Rectal reflex	Similar to that of the bladder. (Hemorrhoidal sympathetic ganglia.)	
325 Ischemic reflex	A sudden pallor of the skin following an irritation and limited to the area of irritation.	
326 Paralytic, hyperemic reflex (dermographia)	Congestion of the skin following the ischemia due to irritation; (tâches cérèbrales and dermographia).	Vaso-motor disturbances cause a disturbance of the nutrition of a part. Diseases which result from, or are associated with, disturbances of the vaso-motor reflexes are discussed in Chart XVII.

The methods of eliciting the various reflexes are described in Chart I c.
Diseases in which the reflexes are altered are discussed in Charts X, XIV, XVI, XVII.

CHART V b

Pupillary Reflexes

Comprising Numbers 302 and 330 to 341.

PUPILLARY REFLEXES

Diagnostic Symptoms	Definition and Location of Reflex Centers	Significance
330 Pupillary reaction to light (25)	Pupil contracts when light is thrown on retina of the same eye (direct reflex), and when light is thrown on retina of opposite eye (consensual reflex), and dilates when retina is shaded from light (ciliary ganglion).	The pupillary reaction to light is diminished or absent in lesions of the reflex arc (optic nerve, corpora quadrigemina, the Westphal-Edinger cell group of the motor oculi nucleus, third nerve and ciliary ganglion), especially in lesions of the ciliary ganglion. When the optic nerve or corpora quadrigemina are involved the consensual reflex can not be obtained from the other (healthy) eye. It is absent in blindness, deep sleep, narcosis, shock, coma, epileptic, and occasionally in hysterical, attacks; also absent in tabes, in many cases of paresis and in rare cases of syphilis alone; absent also when eye is under the influence of mydriatics or myotics.
331 Pupillary reaction to accommodation (27)	Pupil dilates when patient looks at a distant object and visual axes are parallel and contracts when patient looks at a near object and eyes converge.	The pupillary reaction to accommodation is absent (cycloplegia) in lesions of the third nerve, sometimes after diphtheria, occasionally in alcoholism and when the eye is under the influence of mydriatics or myotics, also in myopia and in cases of deficient convergence.
332 Argyll-Robertson's phenomenon (447, 891)	Pupil does not respond to light, but does respond to efforts at accommodation.	The Argyll-Robertson's phenomenon occurs in almost all cases of tabes and paresis (in many of these cases a degeneration of the posterior columns of the cord has been found at autopsy) and very rarely in cases of syphilis in which there are no manifestations of either tabes or paresis for years afterwards. The reverse of the Argyll-Robertson's phenomenon, i.e., the preservation of the light reflex and the loss of the accommodation reflex, occurs occasionally in diphtheritic paralysis and has been found associated with syphilis, basal meningitis, tumors of corpora quadrigemina and myelitis. It is extremely rare.
333 Immobile pupil (545)	The pupil responds neither to light nor accommodation, but in some cases may still dilate slightly on irritation of cervical sympathetic.	Immobile pupil may occur in lesions of the optic nerve or tract or in its nucleus or in that of the third nerve or in the ciliary ganglion or its nerve. It may also be associated with ophthalmoplegia externa or interna or both. When it occurs alone it is due to a lesion in the nucleus. Immobile pupil also occurs in tabes, in epilepsy, in some forms of hysteria, in fainting, and in katatonic stupor.
334 Hemiopic reflex (26)	Pupil contracts when light is thrown on the unparalysed half of retina, but does not contract when light is thrown on paralysed half.	The hemiopic reflex occurs only in lesions of the optic tract or geniculate bodies (homonymous hemianopia) or of the central part of the optic chiasm (bitemporal hemianopia). The existence of this reflex is disputed by many observers.

PUPILLARY REFLEXES (Continued)

Diagnostic Symptoms	Definition and Location of Reflex Centers	Significance
335 Cilio-spinal reflex (465, 1191–2)	Pupil dilates when neck on same side is irritated or when cocaine is dropped in the eye. (Cervical sympathetic ganglion.)	The cilio-spinal pupillary reflex is absent in lesions of the cervical sympathetic, and in many lesions of the medulla and lower cervical and upper dorsal region of the spinal cord (cilio-spinal center—465).
336 Hippus	When the eye is suddenly exposed to light, there occurs a series of alternate contractions and dilatations of the pupil, gradually growing less in degree.	Hippus is usually associated with a general exaggeration of reflexes.
337 Westphal's pupil reaction	When patient's eyelids are held forcibly apart and he attempts to close them he not only turns the eyeball upwards (Bell's phenomenon) but also the pupil contracts.	Westphal's pupillary reaction occurs in some cases of tabes and in paresis.
338 Paradoxical pupillary reflex	Pupil dilates instead of contracting upon exposure to light or upon efforts of accommodation.	The paradoxical pupillary reflex is of no diagnostic significance. It has been observed in tabes and in paresis and is the result of fatigue.
339 Mydriasis	Dilated pupils.	Mydriasis may be irritative or spasmodic, due to irritation of the cervical sympathetic ganglion or nerve; or may be paralytic, due to paralysis of the third cranial nerve or the ciliary ganglion; or may be due to both causes. It occurs in children, and on taking certain drugs (mydriatics). It occurs also from irritation of the cervical sympathetic *directly* by incipient lesions in the cervical enlargement of the spinal cord and its membranes, or by tumors in the neck, or by excess of carbonic acid in the blood as in dyspnoea; and *indirectly* by strong emotions and especially by pain; also in paralysis of the sphincter pupillae (iridoplegia) from lesions, such as optic atrophy, glaucoma, lesions of the third nerve, or ciliary ganglion, which break, or impair, the reflex arc, and which usually cause more or less diminution of vision and a deficient perception of light; also in coma, in cases of increased intra-cranial pressure, and in some other cerebral and meningeal lesions, especially in their later stages.
340 Myosis	Contracted pupils.	Myosis may be irritative or spasmodic, due to irritation of the third nerve or ciliary ganglion; or may be paralytic, due to paralysis of the cervical sympathetic ganglion or nerve, or may be due to both causes. It occurs in old age, in deep sleep, or on taking certain drugs (myotics); also from irritation of the third nucleus or nerve, as in meningitis in early stages and especially in hemorrhage into the pons; and from excessive use of accommodation, as in watchmakers, etc.; also from paralysis of the sympathetic in lesions of the neck and of the spinal cord (syringomyelia). It occurs often in tabes, paresis, iritis, irritation of cornea and, temporarily, after excision of the Gasserian ganglion.
341 Unequal pupils or anisocoria	One pupil is larger than the other when the eyes are at rest.	Aniscoria occurs in many conditions and is of little or no diagnostic value.

The methods of eliciting the pupillary reflexes are described in Chart I b.
Diseases in which these reflexes are altered are discussed in Chart XIV.

CHART VI

Disorders of Sensation

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease.

344 DISORDERS OF SENSATION

The power of receiving perceptions of the external world and of the occurrences in our own body (the basis of all knowledge) is acquired early in life. The nature of the process is entirely unknown, but it rests upon the power of storing up memories and of recalling them at will. It depends upon the integrity of the central and peripheral sensory neurons (463-4), as well as upon that of the terminal sensory organs and of the cerebral cortex (47 to 56). This power may be diminished, or exaggerated, or perverted in various diseases.

345 DIMINUTION

Either no perception or an abnormally feeble one follows a sensory irritation adequate in health to cause a perception (805, 810).

346 EXAGGERATION

An unusually strong perception, as compared with health, follows any sensory irritation (806).

The conditions under which sensation may be diminished or increased are set forth in Chart VI a.

347 PERVERSION

The occurrence or modification of a perception such as never occurs in health (930).

The conditions under which sensation is perverted are set forth in Chart VI b.

CHART VI a

Diminution and Exaggeration of Sensation

Comprising Numbers 345 and 346, and 348 to 372

SENSATION

345 DIMINUTION

Diagnostic Symptoms	Definition	Significance
348 Anesthesia (complete) or Hypesthesia (partial). (Superficial sensibility)	A loss, or diminution, of the normal sensibility to touch upon adequate irritation. Normal sensibility varies in acuteness in different parts of the body and in different individuals.	Diminution of sensibility may be due to disease of the terminal end organs, or to a destructive lesion either of the peripheral sensory neurons (464), (in which case all forms of sensibility are abolished over an area usually coinciding with, but smaller than, the distribution of a peripheral nerve, and the reflex acts in the same part are also abolished); or of the sensory central neurons (463), (in which case frequently all forms of sensibility are not abolished, and the anesthetic area does not correspond to the area of distribution of a nerve, and the reflex acts in the part are not abolished). Sensibility is abolished in coma, narcosis and often apparently in hysteria. A broad zone of analgesia and, more rarely, of anesthesia also, about the body occurs in locomotor ataxia: "tabetic cuirass." The anesthetic area may coincide with the distribution of a peripheral nerve or with that of a nerve root (peripheral lesion); or with the distribution of several nerve roots (spinal lesion); or the area may involve one-half the body: called hemianesthesia (cerebral lesion and hysteria). Anesthesia of one side of the face and of the opposite arm and leg, "crossed hemianesthesia," occurs in lesions in the tegmentum of the pons. Anesthesia may involve some portion of the body supplied by small branches of many different nerves, such as a hand, a foot, a leg, a forearm, etc., and be sharply limited "stocking and glove variety" (hysterical). (Fig. 33.)
349 Analgesia or Hypalgesia	A loss, or diminution, of the normal sensibility to pain, which in health varies in different individuals and in different parts of the body.	
350 Thermic Anesthesia or Hypesthesia	A loss, or diminution, of the sensibility to variations in temperature. This loss may be more marked for cold than for heat and vice versa.	
351 Loss of pressure sense	Inability to distinguish differences in the amount of pressure made on the skin.	
352 Loss of muscle and joint sense or Akinesthesia. (Deep sensibility)	Inability to tell how strongly a muscle is contracted, whether a joint is flexed or extended, or where an extremity is situated in space. A very complex sensation.	
353 Apallesthesia or loss of osseous sense or vibration sense.	Inability to feel the vibration of a tuning fork pressed firmly on the skin.	
354 Astereognosis	Inability to recognize objects by the sense of touch; anesthesia not being present.	Analgesia, thermic anesthesia and apallesthesia may be due to lesion of the central gray matter, or of the antero-lateral ascending tract, of the cord. (Fig. 26.)
355 Deafness or Anakusia or Hypakusia	Loss, or diminution, of sense of hearing.	Astereognosis always indicates a lesion of the cerebral cortex. (Fig. 15.)
356 Anosmia or Hyposmia	Loss, or diminution, of sense of smell.	Anakusia, anosmia, ageusia and blindness, may be due to a lesion of the sensory terminal organ, of the sensory nerve or tract, or may be functional. But these symptoms may occur in so many conditions unconnected with the nervous system that they may have very little diagnostic value in nervous diseases.
357 Ageusia or Hypogeusia	Loss, or diminution, of sense of taste.	
358 Blindness or Anopsia or Amaurosis	Loss of vision.	Hemeralopia associated with a central scotoma for green and red is not uncommon in tobacco smokers; so that when the pupil is dilated in a dim light the healthy part of the retina can act. This condition is quite different from snow-blindness, where the retina is exhausted by too bright and too long continued light.
359 Amblyopia	Decided impairment, but not complete loss, of vision, especially for colors in the early stages. Usually in such cases the field of vision is made small by the loss of more or less of its periphery or by scotomata.	Nyctalopia is at times associated with congenital retinitis pigmentosa, with cortical (peripheral) cataract and with other defects in the eye.

	Diagnostic Symptoms	Definition	Significance
DIMINUTION (Continued)	360 Hemeralopia	A condition in which the patient sees better in a dim light than in a bright one (day blindness).	Homonymous hemianopia is due to a lesion of the optic tract posterior to the chiasm, of the geniculate bodies, the optic fasciculus or the median surface of the occipital lobe of the opposite side of the brain (lips of calcarine fissure). (Fig. 16)
	361 Nyctalopia	A condition in which the patient sees well in a bright light but is almost blind in a dim one (night blindness).	Bi-temporal hemianopia is due to a lesion of the central part of the optic chiasm. Nasal hemianopia is due to a lesion of the lateral margin of the optic chiasm. Bi-nasal hemianopia cannot result from one lesion.
	362 Hemianopia	Loss of one-half of the field of vision.	
	Homonymous	Loss of the same half in both fields.	Tetartanopia is due to a lesion of the upper lip of the contralateral calcarine fissure if it be a lower quadrant of the field of vision and of the lower lip of this fissure if it be an upper quadrant; very rarely to a partial lesion of the geniculate bodies or optic fasciculus. (Fig. 16.)
	Nasal	Loss of the nasal half in each or either field.	
	Bi-temporal	Loss of the temporal half in both fields.	
	363 Tetartanopia or Quadrantic Hemianopia	Loss of an homonymous quadrant of both fields of vision.	
	364 Achromatopsia or color blindness. Hemichromatopsia	Inability to distinguish the different colors from each other either throughout the whole, or in one-half the field of vision.	Achromatopsia may be due to a congenital defect or to defective education or may be the early stage of a gradually developing blindness or amblyopia. Due to mild, not completely paralysing, lesions of any portion of the visual tract in the broad sense.
	365 Dissociation of sensation	Loss of some forms of cutaneous sensibility (usually for pain and temperature) with preservation of others (tactile). (Figs. 24-7.)	Dissociation of sensation always indicates a lesion of the central gray matter (syringomyelia) or of the lateral columns of the spinal cord, or more rarely a lesion in the ponto-cerebellar angle of the pons at the level of the auditory nerve. It occurs associated with motor paralysis of the opposite side of the body in some cases of Brown-Séquard's paralysis.
346 EXAGGERATION	366 Hyperesthesia	Increased tactile sensitiveness. An unusually slight touch can be perceived. A very rare and even doubtful condition. It is usually employed when a touch causes an unusually great, even painful sensation, where hyperalgesia or haphalgesia (380) would be a better term.	Exaggeration of sensibility of all kinds is usually functional. More rarely it is the result of an irritative, rather than a destructive, lesion of the central or peripheral sensory neurons. It occurs in strychnine poisoning and tetanus. Hyperesthesia occurs as a zone at the upper limit of the anesthesia in many spinal lesions, and on the same side of the body as is the lesion in Brown-Séquard's paralysis. It is usually associated with increased reflex activity.
	367 Hyperalgesia	Increased sensitiveness to pain.	
	368 Thermic Hyperesthesia or Hyperalgesia	Increased, even painful, sensitiveness to heat or cold, or both.	
	369 Hyperosmia	Increased, even painful, sensitiveness to odors.	
	370 Hypergeusia	Increased and unpleasant sensitiveness to taste.	Photophobia is functional, or due to eye strain, or to inflammation of some part of the eye, or optic nerve, or cerebral meninges.
	371 Photophobia	Increased and painful sensitiveness to light.	
	372 Hyperakusia	Increased, even painful, sensitiveness to sounds.	Hyperakusia is functional, or due to ear diseases affecting the labyrinth, or to cerebral conditions causing hyperemia of the labyrinth (meningitis, encephalitis, tumors, etc.) and to spinal affections.

Methods for the detection of these conditions are described in Chart Ic.
Diseases in which these conditions occur are discussed in Chart XIV.

CHART VI b

Perversions of Sensation

Comprising Numbers 347 and 374 to 392

SENSATION

Diagnostic Symptoms	Definition	Significance
374 Pain (Figs. 33, 38)	Is an unpleasant sensation not felt in perfect health, except in cases of injury. It varies greatly in intensity. It presents different qualities, such as: tearing, cutting, burning, throbbing, darting, etc. It may be diffuse, or felt in a small area (localized), or may run along a nerve trunk (radiating), or may run half way or entirely about the body or an extremity (girdle), or it may be felt in an area which is itself anesthetic (anesthesia dolorosa). Pains may vary as to time of occurrence, some showing a distinct periodicity (malaria, neuralgia and migraine), some occur at menstrual epochs. Some headaches occur in morning (uremic), others in afternoon (ocular) and others towards evening and at night (syphilitic). Some pains are increased by pressure (neuritis and neuralgia) while some are diminished by it (lead colic).	Perversions of sensibility, especially pain and paresthesiae, are often functional and are often due to irritation (pressure, chemical, inflammatory, etc.) of central or peripheral sensory neurons. Radiating and girdle pains are usually due to lesions of the nerve roots. Anesthesia dolorosa is due to a lesion of the central end of a sensory neuron which has been destroyed below this point and therefore can conduct no sensations from below. Although pain may be felt as peripheral it may be of central origin and due to lesions of central neurons within the brain or cord. On the other hand pains due to lesions in the abdominal viscera may be referred to remote parts of the body or the head (referred pains 952).
347 PERVERSION		
375 Paresthesiae	Curious sensations rarely felt in perfect health, usually unpleasant but not severe enough to be called pain. They are numbness, tingling, formication, heat, cold, heaviness, tired feeling, hunger, etc.	Failure of localization may be functional but usually results from lesions of the peripheral sensory neurons (tabes).
376 Failure of localization (Topoanesthesia)	When a cutaneous sensation is felt but cannot be localized.	Allocheiria occurs in hysteria, very rarely in tabes, hemiplegia and sclerosis. Polyesthesia occurs only in tabes and in hysteria.
377 Allocheiria	When an irritation is not felt at the point of contact, but at a corresponding point on the opposite side of body.	Paradoxical sensation has been met with in a number of spinal and cerebral diseases, but is without diagnostic significance.
378 Double sensation and Polyesthesia	Where one contact gives rise to two distinct sensations (double sensation) or more (polyesthesia).	Haphalgesia occurs in hysteria.
379 Paradoxical sensation	The quality of thermic sensation is reversed, a hot body feels cold and vice versa.	Retardation of conduction of pain occurs only in lesions of peripheral sensory neurons (tabes or multiple neuritis).
380 Haphalgesia	A slight tactile impression from certain objects, but not from others, is felt as intense pain.	Persistence of sensation occurs in lesions of the peripheral sensory neurons (tabes).
381 Retardation of conduction of pain	The sensation of pain is not felt until an appreciable interval after the time of contact.	Binocular diplopia is due to a weakness of one or more of the external muscles of one eye, or to displacement of one eyeball; so that the image does not fall on identical spots in the two retinae.
382 Persistence of sensation	The sensation continues an unusually long time after the irritation causing it has ceased to act.	

PERVERSION (Continued)

Diagnostic Symptoms	Definition	Significance
383 Binocular Diplopia (816)	Two separate visual perceptions of the same object, the perception from the normal eye (true image) being more distinct than that from the abnormal eye (apparent image).	Monocular diplopia may occur in hysteria, in cases of double pupillary opening, in anomalous refraction (incipient cataract), and irregularities in the cornea.
384 Monocular Diplopia or Polyopia (878–82)	A condition in which objects appear double or multiple, even when looked at with one eye alone.	Metamorphopsia may occur in hysteria, also in astigmatism (refractive) and in displacement of the retinal elements (retinal) which may occur in retinitis, choroiditis, and in detachment, or tumor, of retina.
385 Metamorphopsia	A condition in which objects appear distorted.	
386 Micropsia	A condition in which everything looks much smaller than normal.	Micropsia may occur in hysteria, in paralysis of accommodation and, with distortion, when the retinal elements are spread apart (recent choroiditis or retinitis).
387 Macropsia	A condition in which everything looks much larger than normal.	Macropsia may occur in hysteria, in spasm of accommodation and, with distortion, when the retinal elements are crowded together (atrophic stage of retinitis and choroiditis).
388 Tinnitus Aurium	A sound of ringing, roaring, whistling, etc., in ears or head.	Tinnitus aurium, parakusis, parosmia and parageusia occur in lesions of the terminal organ and in insanity and functional disorders. They may constitute the aura of an epileptic attack.
389 Parakusis	Perversions of hearing, such as hearing tones incorrectly or hearing better when other loud noises are present at the same time, or hearing sounds or words for which there is no external cause (hallucination).	
390 Parosmia	The perceptions of abnormal odors or of those for which there is no external cause (hallucination).	
391 Parageusia	The perception of abnormal tastes or of those for which there is no external cause (hallucination).	
392 Vertigo	A feeling as if the person (subjective) or as if surrounding objects (objective) were whirling about, or both.	Vertigo may be functional (hysteria, neurasthenia, traumatic neuroses); or may depend on changes in the cerebral circulation, especially anemia and hyperemia (cardiac and arterial diseases, congestion in portal or systemic circulation, galvanism of head or neck), or toxic (tobacco, morphine, alcohol, some digestive disturbances, etc.); or may depend on diseases of the cerebellum and its tracts, or of the ear or eye. It is the principal symptom in Ménière's disease (aural vertigo). Vertigo is closely associated with vomiting. In vertigo associated with lesions in, or pressing upon, a cerebellar hemisphere, external objects seem to whirl in the direction away from the injured hemisphere in both conditions, but the subjective vertigo, usually, is away from the injured hemisphere in case the lesion is within it and towards it when the lesion is external and presses upon the hemisphere.

Diseases in which these conditions occur are discussed in Charts XIV and XV.

CHART VII a

Electrical Examination

Comprising Numbers 395 to 405

Definition, Significance and Relationship of the Symptoms of Disease

	Name of the Reaction	Tissue Tested	Reaction to Faradism	Reaction to Galvanism and Formula of Galvanic Reaction	Character of the Contraction	Significance of the Reaction
395 ELECTRICAL REACTION OF MUSCLES AND NERVES (70-3)	396 Normal excitability (473)	NERVE AND MUSCLE	Contraction present to a strength of current which is normal for the nerve and muscle tested.	Neg.Cl.C. Pos.Cl.C. Pos.Op.C. Neg.Cl.Tet. is the normal formula, or in other words Neg.Cl.C. occurs with the weakest current that will cause any contraction. Neg.Cl.C. Pos.Cl.C. with a little stronger current. Neg.Cl.C. Pos.Cl.C. Pos.Op.C. with a still stronger current. The explanation of the above formula is as follows: The weakest current that will cause any contraction of the muscle will do so when the negative electrode is on the motor point and the current is closed. (Neg.Cl.C.) A more powerful contraction will take place when a stronger current is used and then there will also be a contraction when the current is closed and the positive pole is on the motor point (Pos.Cl.C.). A still more powerful current causes a contraction when the current is opened and the positive electrode is on the motor point (Pos.Op.C.). With such powerful currents and the negative pole on the motor point there results a tetanus or continuous contraction when the current is closed, (Neg.Cl. Tet.); so that the muscle cannot relax to contract again when the current is opened. There is, therefore, in health no reaction corresponding to " Neg.Op.C."		Normal excitability shows a normal condition of muscle and nerve.
Nerve fibers respond to changes in intensity of both the faradic and the galvanic currents. The changes in intensity are best brought about by making and breaking the current.	397 Diminished excitability	NERVE AND MUSCLE	Contraction present but it requires an unusually strong current to produce it.		Quick.	Diminished excitability occurs in many diseases and conditions, especially in lesions of the central motor neurons and is not of much value in diagnosis.
Muscle fibers respond only to the galvanic current. The muscle responds to the faradic current only in virtue of the nerve fibers supplied to it. When these nerve fibers are degenerated the muscles can no longer respond to the faradic current. Both nerves and muscles have points on the body surface; the so-called motor points (see figures 1 to 5) from which they are most readily excitable. Therefore, in testing a nerve or muscle by electricity the electrode (positive or negative) is placed on the corresponding motor point (70-3).	398 Exaggerated excitability	NERVE AND MUSCLE	Contraction present to an unusually weak current.			Exaggerated excitability is a rare condition. It occurs in nervous persons with moist skins and in tetany.
	399 Reaction of degeneration (472)	NERVE	Gradual loss of excitability which becomes complete in about two weeks after injury or onset of the disease.	No reaction.	None.	The reaction of degeneration proves that the peripheral motor neurons are degenerated and that recovery will either never take place, or will be very slow. The lesion must be either in the peripheral nerves, or nerve roots, or in the anterior horns of the spinal cord, or in the motor nuclei in the brain stem.
		MUSCLE	Gradual loss of excitability which becomes complete in less than two weeks after injury or onset of the disease.	After the first two weeks the muscle responds to unusually feeble galvanic currents and the normal formula is reversed; the positive pole being more potent. Pos.Cl.C. Neg.Cl. C. Pos.Op.C. Neg·Op.C. (which last reaction never occurs in health). It is usual to express the formula for the normal reaction and for the reaction of degeneration in the German language in which Kathode means the negative electrode and Anode means the positive electrode. The usual normal formula is K.C.C., A.C.C., A.O.C., K.C.Te. The reaction of degeneration is A.C.C., K.C.C., A.O.C., K.O.C. The essence of the normal formula is K.C.C.>A.C.C. The essence of the formula of the reaction of degeneration is A.C.C.>K.C.C.	Sluggish. The sluggish character of the muscular contraction is the most characteristic thing in the reaction of degeneration.	

ELECTRICAL REACTIONS (Continued)

	Name of the Reaction	Tissue Tested	Reaction to Faradism	Reaction to Galvanism	Formula of Galvanic Reaction	Character of the Contraction	Significance of the Reaction
395 ELECTRICAL REACTION OF MUSCLES AND NERVES	400 Partial reaction of degeneration	Nerve	Contractions present, but require unusually strong currents, whether faradic or galvanic.		Either the normal formula, or the formula of the reaction of degeneration, or a combination of the two may be present. A.C.C. may equal K.C.C.	Quick or sluggish	The significance of this reaction is the same as that of the reaction of degeneration, except that it indicates the lesion is less severe and that all the nerve fibers are not degenerated.
		Muscle	Contractions present only to unusually strong currents.	Contractions present to unusually weak currents		Sluggish.	
	401 Myasthenic reaction (553)	Nerve and Muscle	Contractions quickly grow less strong and soon cease under rapidly repeated excitation.	Normal	Normal.	Quick, grows rapidly weaker and ceases.	Occurs only in myasthenia gravis (553).
	402 Myotonic reaction (613)	Nerve and Muscle	Continuous tonic contraction lasting some time after the electrical stimulation has ceased.	Curious wave-like contractions occur, and last after electrical stimulation has ceased.	Positive pole is about equally as potent as the negative. Hence the formula A.C.C. = K.C.C.	Continues usually a long time and has a wave-like character.	Occurs in Thomsen's disease (613).
	403 Neurotonic reaction	Nerve	Unusually excitable. Tetanic contraction persists after electrical stimulation has ceased.		Normal.	Continuous	Occurs in hysteria, amyotrophic lateral sclerosis and chronic bulbar paralysis.
		Muscle	Normal.	Normal.			
	404 Reaction of completely degenerated muscle (70 to 73)	Muscle	None.	None.	None.	None.	Muscle fibers are entirely degenerated and recovery is impossible.

405 Electrical reaction of the Optic and Auditory Nerves

The optic nerve responds to the galvanic current with a sensation of light, the color of which varies with the pole employed.

The auditory nerve responds with a loud sound when the negative electrode is placed in or near the meatus and the current closed and with a faint sound when the positive pole is used and a stronger current broken. These reactions are without diagnostic importance.

The negative electrode placed in front of the ear causes a nystagmus towards the ear tested when the current is closed and in the opposite direction when the current is broken. The positive electrode causes nystagmus in exactly the reverse direction.

In cases of disease in which the caloric test (79) is absent and in which the electric test is present, it is fairly certain that the lesion is in the labyrinth and not in the nerve. If there is no response to either the caloric or the electric test the lesion is in the nerve or its nucleus.

CHART VII b

Erb's Motor Points for Electrical Examination of Nerves and Muscles

The motor points are the areas upon the surface of the body at which the individual nerves and muscles can be most easily excited by electricity. For the nerves, these points coincide with those at which the nerve lies most superficially or where it can be pressed against a resisting tissue; for the muscles, they lie over the point of entrance of the nerve into the muscle.

Comprising Figures 1 to 5

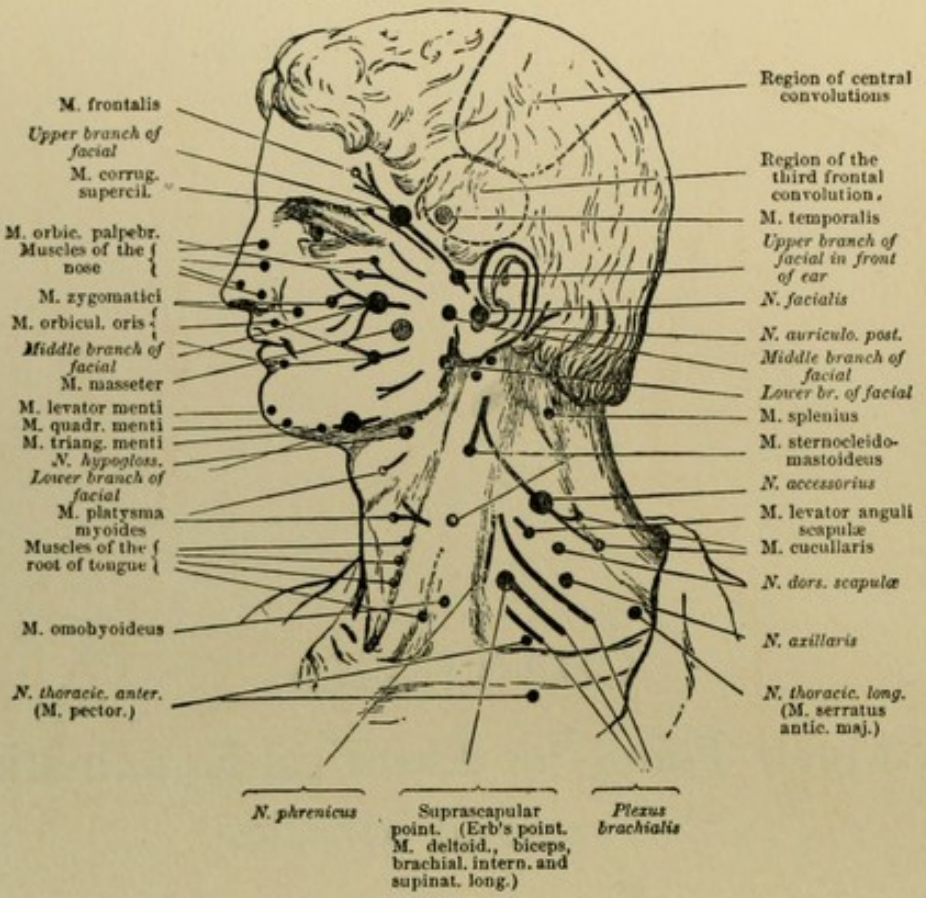
M. frontalis
Upper branch of facial
M. corrug. supercil.
M. orbic. palpebr.
Muscles of the nose
M. zygomatici
M. orbicul. oris
Middle branch of facial
M. masseter
M. levator menti
M. quadr. menti
M. triang. menti
N. hypogloss.
Lower branch of facial
M. platysma myoides
Muscles of the root of tongue
M. omohyoideus
N. thoracic. anter. (M. pector.)
Region of central convolutions
Region of the third frontal convolution.
M. temporalis
Upper branch of facial in front of ear
N. facialis
N. auriculo. post.
Middle branch of facial
Lower br. of facial
M. splenius
M. sternocleido-mastoideus
N. accessorius
M. levator anguli scapulæ
M. cucullaris
N. dors. scapulæ
N. axillaris
N. thoracic. long. (M. serratus antic. maj.)
N. phrenicus
Suprascapular point. (Erb's point. M. deltoid., biceps, brachial. intern. and supinat. long.)
Plexus brachialis

Fig. 1

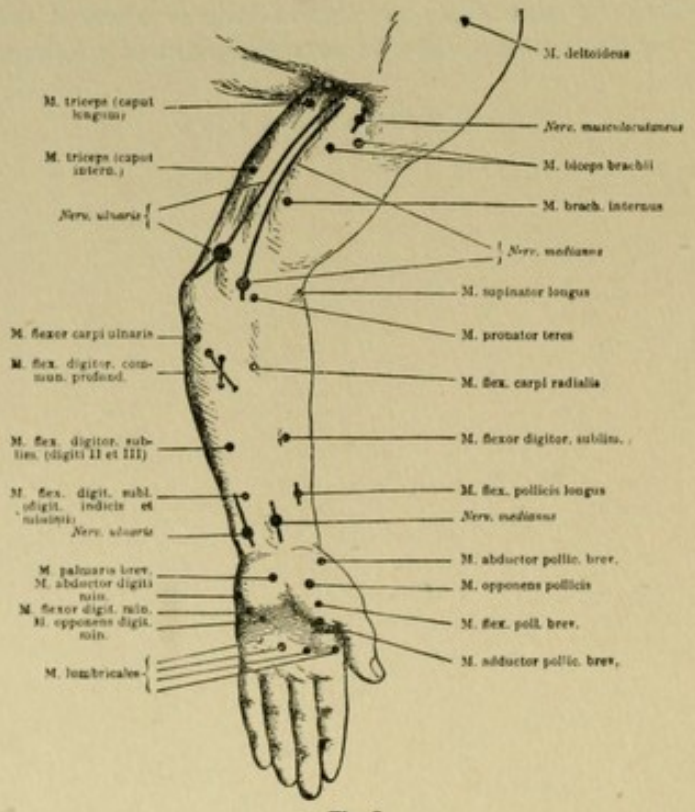
M. deltoideus
M. triceps (caput longum)
M. triceps (caput intern.)
Nerv. ulnaris
M. flexor carpi ulnaris
M. flex. digitor. commun. profond.
M. flex. digitor. sublim. (digiti II et III)
M. flex. digit. subl. (digit. indicis et minimi)
Nerv. ulnaris
M. palmaris brev.
M. abductor digiti min.
M. flexor digit. min.
M. opponens digit. min.
M. lumbricales
Nerv. musculocutaneus
M. biceps brachii
M. brach. internus
Nerv. medianus
M. supinator longus
M. pronator teres
M. flex. carpi radialis
M. flexor digitor. sublim.
M. flex. pollicis longus
Nerv. medianus
M. abductor pollic. brev.
M. opponens pollicis
M. flex. poll. brev.
M. adductor pollic. brev.

Fig. 2

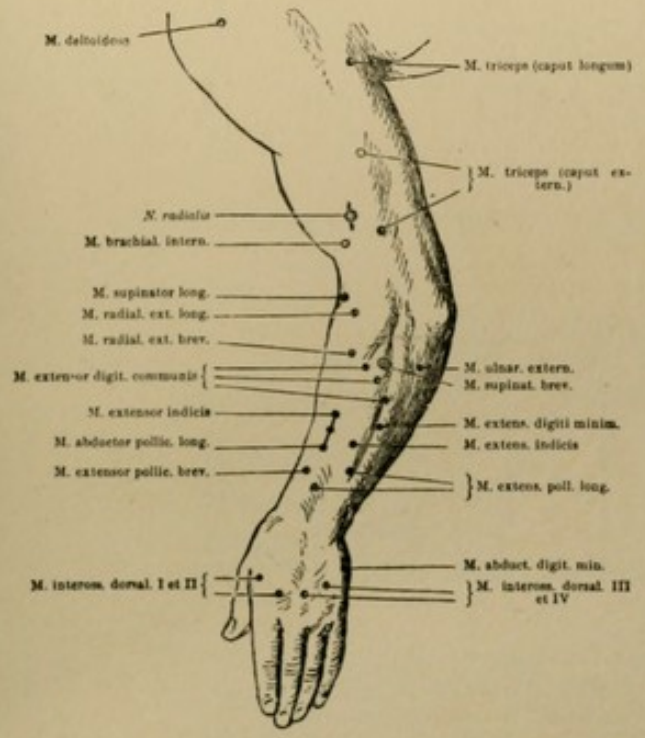

Fig. 3

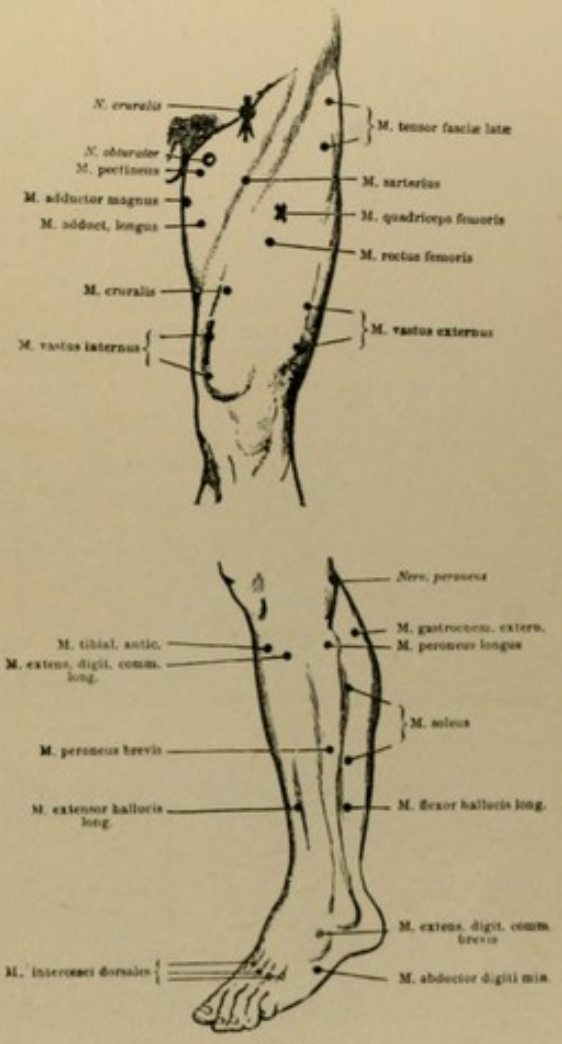

Fig. 4

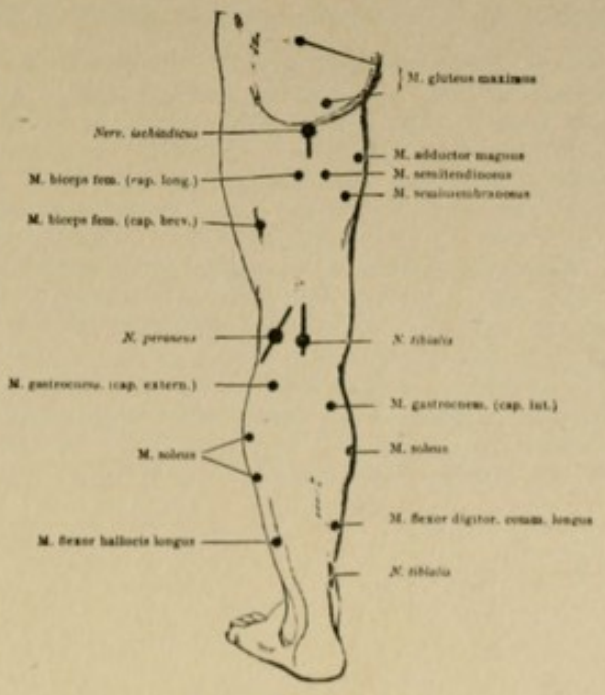

Fig. 5

CHART VII c

ERB'S DIAGRAM SHOWING THE EFFECTS OF INJURY OF A NERVE

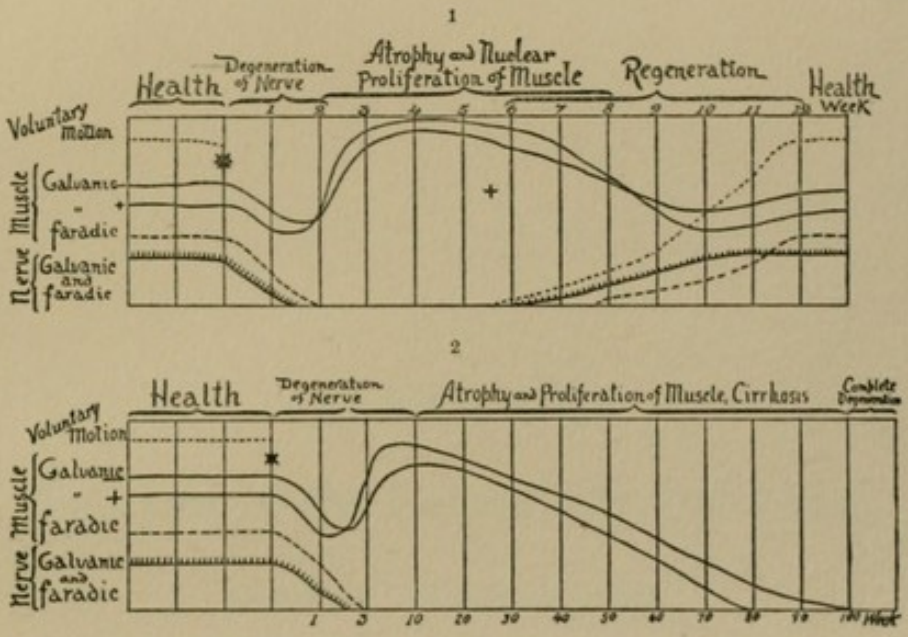

FIG. 6

Charts Illustrating the Reaction of Degeneration

The star (*) indicates the incidence of a paralysing lesion in the domain of the peripheral neuron. Voluntary motion is lost at once. During the first two weeks there is slight diminution of the galvanic excitability of muscle; there is also rapid diminution of the faradic excitability of muscle and of the galvanic and faradic excitability of nerve, which are completely lost at the end of the second or third week. During the second week there is rapid increase in galvanic excitability of muscle and the response to the positive pole becomes greater than to the negative.

Chart 1 represents the reaction in a case terminating in recovery. During the sixth week (indicated by the cross X) regeneration begins. The increased galvanic excitability of the muscles gradually diminishes until it becomes normal and the poles are reversed so that the negative response is again greater than the positive. Voluntary motion returns first, then the galvanic and faradic excitability of the nerve, and last of all, the faradic excitability of the muscles.

Chart 2 represents the reaction in a case terminating in atrophy and cirrhosis of the muscle. The galvanic excitability of the muscle is increased and the poles are reversed, as before. The decline in galvanic excitability continues, however, until the end of the second year, when it is entirely lost. Voluntary motion, and the electrical reactions of both muscles and nerve are thus permanently destroyed.

CHART VIII

Analysis of the Cerebro-Spinal Fluid

Comprising Numbers 410 to 420

ABNORMAL CEREBRO-SPINAL FLUID

	Characteristics	Method of Testing	Significance
410 ABNORMAL CEREBRO-SPINAL	411 Tension	Can be roughly estimated by the rapidity of flow of fluid through the canula, whether in drops or a stream, more accurately by the height to which the fluid rises in a vertically held glass tube connected by a short rubber tube with the canula. A stopcock on the canula adds to the accuracy by preventing the escape of much fluid and the consequent lowering of the tension. The fluid in the tube rises and falls with the respiration. An additional more rapid and stronger pulsation indicates a basilar aneurism.	A low or very rapidly diminishing tension has no diagnostic meaning, except as indicating an obstruction to the communication of the fluid in the ventricles with that of the vertebral canal, as in closure of the foramen of Magendie. A high tension means increased intra-cranial, or intra-spinal, pressure caused by an increased amount of cerebro-spinal fluid or by a foreign body within the cerebro-spinal cavity. It occurs in tumors, abscess, hydrocephalus, hemorrhage, acute, subacute, some cases of chronic, and serous meningitis, also in cerebral edema (nephritis, anemia, etc.), acute infectious diseases and some other conditions.
	412 Red color or 413 reddish yellow color	By sight. Hematoidin crystals may be seen under the microscope.	Fresh blood in the fluid may be the result of puncture of a blood vessel, in which case it is most abundant in the fluid first drawn, usually coagulates, and settles quickly on centrifugalization. Or, May be the result of hemorrhage into the ventricles or membranes. Hemorrhage, haematoma, aneurism, etc.
	414 Cloudy	By sight. Pus cells under the microscope. Polymorpho-nuclear leucocytes.	An increase of cellular elements in the fluid is usually the result of an acute or sub-acute meningitis. In some cases of acute meningitis, however, the fluid may be clear.
	415 Clear with delicate coagulum	By sight.	Tuberculous meningitis, usually.
	416 Cellular elements and bacteria	Fluid soon after withdrawal should be centrifugalized. Tube should be emptied quickly and from its walls and bottom sediment should be sucked in and out of a capillary tube, well mixed and spread on two clean slides. One slide should be stained by Gram's method for bacteria, and the other by Wright's blood stain for cellular elements. Or, The fluid (not centrifugalized), 10 parts, can be mixed with 1 part of a solution consisting of methylene blue 0.2%, glacial acetic 4.0%, and water to 100 %, and counted in a Thoma-Zeiss chamber.	The normal cerebro-spinal fluid shows under these conditions 1 to 3 cells in a field of the microscope. If there are more than 4 to 6 cells in a field it indicates a meningitis. If the cells are mainly leucocytes it indicates epidemic cerebro-spinal, or purulent meningitis, or rarely an acute tuberculous meningitis. Broadly speaking, an acute infectious meningitis. If the cells are mainly or entirely lymphocytes it indicates a tuberculous meningitis, or cerebro-spinal syphilis, or paresis, or tabes, or acute anterior poliomyelitis, or convalescence from any form of acute meningitis. Broadly speaking a chronic infectious meningitis. If echinococcus cysts or hooklets are present, they indicate the presence of an echinococcus cyst.

ABNORMAL CEREBRO-SPINAL FLUID (Continued)

	Characteristics	Method of Testing	Significance
F L U I D	417 Sugar	By Haines' test or other tests.	Not of much significance, but the sugar normally present is diminished usually in meningitis and in some other conditions.
	418 Albumen	Two c.c. of the fluid mixed with 10 c.c. of Esbach's fluid is centrifugalized during one hour in a conical tube graduated to 0.1%.	Normally not more than ½% is present. Usually increased in meningitis and tumors. A diminution in the amount usually indicates a progressive space-occupying disease. Of little diagnostic significance.
	419 Globulin	Two c.c. of a saturated solution of chemically pure neutral ammonium sulphate should be placed in a test tube and one cc. of the cerebro-spinal fluid should be gently run upon its top. If the reaction is positive, within 3 minutes, a grayish white ring should form at the junction of the two fluids. At the end of one-half hour, the surface of the ring should show a delicate network. Best seen by indirect illumination. Or, Boil slightly 1 volume of the cerebro-spinal fluid with 5 volumes of a 10% butyric acid solution, add 1 volume of a normal solution of sodium hydroxide, reheat and allow to cool. If a flocculent precipitate forms, the reaction is positive. (Noguchi test).	Indicates meningitis, acute anterior poliomyelitis, cerebro-spinal syphilis, paresis, tabes, rarely a brain tumor.
	420 Positive Wassermann reaction	This test can only be performed in a laboratory by an expert.	The reaction is positive in 90% of cases of paresis and in 60% of cases in tabes. In cerebro-spinal syphilis both the cerebro-spinal fluid and the blood usually give a positive reaction. In other cases of syphilis (without meningitis) the reaction is usually negative with the cerebro-spinal fluid, but positive with the blood.

CHART IX

Special Syndromes and Anatomical Terms

Comprising Numbers 425 to 465

Syndrome	Definition	Significance
425 Hysterical symptoms (1074)	Occur usually in self-conscious females of an emotional nature. Lack of inhibition and great susceptibility to suggestion. Desire to excite admiration and sympathy and wonder. Mental instability. Globus hystericus (426). Spinal, inguinal (or ovarian) and other tenderness. Great variety of symptoms (especially subjective) which cannot be explained by any organic lesion. Glove and stocking form of anesthesia or hemianesthesia and concentric contraction of the field of vision are common symptoms, but the patient is usually ignorant of their existence until they are discovered, or more probably suggested, by the physician. Exaggerated reflexes but no ankle-clonus or only pseudo-clonus. Never a Babinski reflex. Motor paralyses, tremors, contractions and convulsions are not uncommon. Transference of hemianesthesia can be effected in some cases. The anesthesia of the fingers does not prevent delicate acts being done by them with eyes closed. Such patients when tested and the anesthetic area is touched often answer "no" at the moment of contact (48). Many other symptoms do not seem to be real but rather seem to be imaginary and may result from hallucinations or delusions or more probably are the result of suggestion: auto-, or foreign. Probably many of the hysterical symptoms come into existence as the result of the physician's careful and minute examination or repeated examinations (foreign suggestion).	Hysteria (1074)
426 Globus Hystericus (1074)	The feeling of a lump or ball behind the upper end of the sternum which interferes both with swallowing and breathing. The sensation often commences in the epigastrium and rises to the base of the neck and remains there; the patient not being able to get it up or down. It may be caused by a spasmodic contraction of the muscles of oesophagus or throat.	
427 Hysterogenic areas (1074)	Spots scattered over the body, but usually in the left inguinal region, where light pressure or irritation will cause more or less violent hysterical attacks	
428 Hysterofrenic areas (1074)	Spots scattered over the body, but usually in the left inguinal region, where firm and continued pressure will cause the arrest of an existing hysterical attack.	
429 Lasègue's symptom (1074)	A condition in which the patient cannot move an anesthetic extremity when her eyes are closed, but can move it readily when she opens her eyes and looks at it.	
430 The epileptic aura (575, 846, 1058)	The aura is a symptom (warning) which occurs before the attack in about half the cases of epilepsy. It may be remote or immediate. The former is often called "a prodromal symptom" and occurs hours and days before the attack. It consists usually in an emotional change (irascibility, etc.), changes in the amount of sleep, of food taken, in sexual desire and vasomotor phenomena. Much more characteristic and important is the immediate aura which occurs a fraction of a minute before the attack. This aura may be "psychic" (anxiety, anger, joy, dreamy states, special thought or memory, etc.), or a "sensory hallucination" which may be visual (blindness, lights, colors (red), elaborate false visual perceptions, etc.), or auditory (deafness, noises, and false auditory perceptions), or olfactory or gustatory hallucinations, or cutaneous paresthesiae (the feeling of a wind blowing on some part of the body is quite common) and pains, or visceral paresthesiae, especially epigastric. Vertigo is a common immediate aura; or the immediate aura may be motor and consist in twitching of a group of muscles, (Jacksonian epilepsy), or in more complicated automatic movements of the body, or in hiccough, sneezing, yawning or swallowing. Vasomotor disturbances, flushing or pallor with secondary paresthesiae, are not uncommon immediate aurae. Usually the aura is always the same in the same individual; rarely it varies. In rare cases the aura may not be followed by an attack and in still rarer and always doubtful cases it may be the only symptom of epilepsy.	Epilepsy (575, 846, 1058)

Syndrome	Definition	Significance
431 Jacksonian epilepsy (587-8, 605)	A clonic spasm of one or more muscles in one side of the face or in one arm or leg, which may remain local, but usually rather rapidly extends to other muscles of the same side of face, or of the arm or leg in which it commenced. It then may extend to an adjacent extremity in the same order in which the cortical centers are placed: thus from the face to the arm and then to the leg, from the leg to the arm and then to the face, from the arm to the leg and face nearly or quite simultaneously, but never from the face to the leg, or vice versa, without involving the arm. When the spasm has extended over the whole half of the body it may remain so or may pass across and involve the other side. As long as the spasm is local or limited to one-half of the body consciousness may or may not be lost, but when the spasm involves both sides of the body consciousness is always lost.	Local cortical lesion (587-8, 605) (Figs. 15–16)
432 The prodromata of apoplexy (504, 1060-3)	In many cases of apoplexy, especially in cases of cerebral thrombosis, the apoplectic attack is preceded by a number of more or less definite and characteristic symptoms which may be remote, preceding the attack by months or years; or immediate, occurring immediately before the attack. These prodromata are both *general*, such as headache, vertigo, drowsiness and stupor, irritability, forgetfulness, hypochondriacal feelings, ringing in the ears, flashes before the eyes, etc.; and *local*, such as temporary attacks of aphasia, diplopia, achromatopsia, dysarthria, temporary paralysis of arm or paresthesiae. None of these symptoms is so characteristic that an attack of apoplexy can be confidently predicted from its presence. The most constant prodromal symptom of apoplexy (except of embolism) is high arterial tension.	Apoplexy (504, 1060-3)
433 Tabetic or visceral crises (661)	Paroxysmal attacks of pain in, and functional disturbances of, some viscera, occurring in the course of locomotor ataxia. These attacks recur after irregular intervals, persist during an hour, or a day or two, and are analogous to the paroxysmally occurring lightning-like pains in the legs. "Gastric crises" are the most frequent and consist in severe pain in the epigastrium together with uncontrollable vomiting and retching. At times attacks of gastric pain or of vomiting occur separately. "Hepatic crises" resemble gallstone colics, even being accompanied by slight jaundice at times. "Laryngeal crises" consist in attacks of coughing and dyspnoea. "Laryngeal vertigo" (Ictus laryngeus) consists in a sensation of tickling and burning in the larynx, a stridulous inspiration with a feeling of suffocation and a falling to the ground unconscious for a few minutes. "Pharyngeal crises" consist in repeated acts of noisy swallowing. "Renal crises" resemble attacks of renal colic. "Vesical crises" consist in pain in region of bladder and prostate, and constant desire to urinate. "Urethral crises" consist in attacks of pain in urethra and desire to urinate. "Rectal crises" consist in attacks of pain in the rectum and tenesmus. "Vulvo-vaginal crises" consist of attacks of pain in vagina. "Clitoridian crises" consist of attacks of pain in vulva with sexual desire and discharge of mucus. "Anginal crises" resemble angina pectoris. Occasionally "crises" of several kinds occur simultaneously.	Tabes (661) (Fig.) 27)
434 Bulbar symptoms (546)	A combination of several or all of the following symptoms, dysarthria or anarthria (283-4), dysphagia (285), drooling of saliva from mouth, propulsive speech, and puffing of lips. Paralysis of the 7th, 9th, 10th, 11th, and 12th, and at times of other cranial nerves. Spastic paraplegia or hemiplegia of extremities. Sensory paralysis and ataxia. Respiratory difficulty, and in severe cases rapid, irregular pulse and Cheyne-Stokes' respiration.	Lesion or disorder of medulla (546). (Figs. 21–2)
435 Cheyne-Stokes' respiration	Long pauses in the respiration. After a pause the respiration commences slow and deep and rapidly becomes quick and superficial and as rapidly becomes slow and deep again and terminates in another long pause (lasting from five to sixty seconds, or more) and so on; each cycle being completed in a few minutes. A somewhat similar respiratory disturbance which is called Biot's respiration consists of frequent pauses in the respiratory act, lasting many seconds. Biot's respiration occurs in Bright's disease, etc., but has no particular significance in nervous diagnosis.	

Syndrome	Definition	Significance
436 Stokes-Adams' phenomenon	Slow pulse with long arrests (one half to one minute or more) during which the patient becomes pale, unconscious and may show a more or less pronounced convulsion.	Lesion of bundle of His in the heart, or irritation of pneumogastric nerve.
437 Babinski and Nageotte's bulbar syndrome (1268)	Paralysis of the tongue, diaphragm and larynx with ataxia of the homolateral side; analgesia and thermic anesthesia with motor paralysis of arm and leg of the contralateral side, myosis and pseudoptosis, dysphagia and dysarthria.	Lesion of medulla. (Figs. 21–3)
438 Ponto-cerebellar angle lesions (1363)	Homolateral deafness and contralateral analgesia and thermic anesthesia with preservation of tactile sensibility, nystagmus and weakness of conjugate deviation of the eyes towards the side of the lesion, anesthesia and abolition of reflexes in the distribution of the trigeminus on side of lesion, adiadocokinesia on the same side, optic neuritis, cerebellar ataxia and occipital pains, all more marked on side of lesion.	Lesion of ponto-cerebellar angle. (Fig. 20)
439 Millard-Gubler's syndrome (1269)	Homolateral facial paralysis with contralateral paralysis of arm and leg.	Lesions of pons. (Fig. 20)
440 Weber's syndrome (1270)	Homolateral oculo-motor paralysis with contralateral hemiplegia.	Lesion of crus cerebri.
441 Benedykt's syndrome (1270, 1325)	Homolateral oculo-motor paralysis associated with a tremor of the contralateral arm and leg.	Lesion of red nucleus or of rubro-spinal tract.
442 Brown-Séquard's paralysis or spinal hemiplegia (509, 840)	Below the point of lesion there are motor paralysis, exaggerated tendon reflexes, Babinski reflex, elevation of temperature, vaso-motor disturbances, and at times more or less hyperalgesia, ataxia, and loss of deep sensibility on the homolateral side, together with analgesia, thermic anesthesia, apallesthesia (353) and more or less tactile anesthesia, on the contralateral side. The anesthesia is bounded above by a narrow zone of hyperesthesia or hyperalgesia. Brown-Séquard's paralysis is more often atypical than typical.	Unilateral spinal lesion. (Figs. 24–6)
443 Spinal epilepsy (60–1 and 520)	Violent and continued tremor of the leg after it has been struck or shaken.	Greatly exaggerated tendon reflexes.
444 Bell's phenomenon	A turning upward of the eyeballs when an attempt is made to close the eyelids in peripheral facial paralysis.	Facial paralysis (peripheral).
445 Strümpell's tibialis phenomenon	When a patient, with spastic paralysis of a leg, lying on his back, attempts to flex the paralysed leg at the knee against light resistance, a dorsal flexion of the foot also occurs. Strümpell has found similar phenomena in the radial and pronator groups of muscles in the forearm.	Lesion of the pyramidal tract. Figs. 24–6)
446 Babinski's associated movements of trunk and thigh	When a patient with spastic paralysis of one leg, lying on a hard surface without a pillow, with legs slightly abducted and hands folded across chest, attempts to raise the body to a sitting posture, the paralysed leg is involuntarily raised from its support while the normal leg lies at rest.	

Syndrome	Definition	Significance
447 Argyll-Robertson's pupillary reflex (891)	Loss of the pupillary reflex to light, while the reflex persists with efforts of accommodation and the consequent convergence and parallelism of eyeball (332).	Tabes, paresis and syphilis (661).
448 Romberg's symptom (static ataxia)	A wavering, staggering and even falling when attempting to stand still with eyes shut and with the feet in contact, either laterally or the one before the other (41).	
449 Biernacki's sign	A loss of the normal sensitiveness to pressure of the ulnar nerve behind the elbow.	Tetany (614).
450 Trousseau's sign	Pressure on the nerve trunks of the extremities causes a tetanic spasm of the muscles supplied by them.	
451 Chovstek's sign	The facial nerve shows extreme irritability to percussion or pressure.	
452 Erb's sign	Muscles and nerves are unusually excitable both to galvanism and to faradism.	
453 Quinquand's sign	Patient spreads his fingers and presses their tips against the palm of the observer's hand which is held vertically. After a few seconds a series of slight shocks are felt as if the phalanges of each finger were knocking together.	Chronic alcoholism.
454 Erb's paralysis. Combined shoulder and arm paralysis (490)	A paralysis of the deltoid, biceps, brachialis anticus and supinators, long and short. In some cases the supra- and infraspinatus muscles are also paralyzed, and to a less extent the extensors of the wrist and fingers. Anesthesia of outer aspect of forearm and hand is occasionally present. Paralysis of 5th and 6th cervical nerve roots.	Lesion of the brachial plexus.
455 Klumpke's paralysis (490)	A paralysis of the small muscles of the hand and fingers. There is anesthesia of ulnar side of forearm and hand. In some cases the muscles of the forearm, except the supinator longus, are also paralyzed, and the eye on the same side exhibits myosis, retraction of the bulb and narrowing of the eyelid opening. Paralysis of nerves arising from the 7th and 8th cervical and 1st dorsal nerve roots.	Erb's paralysis. May be due to injury at birth (obstetric paralysis or Duchenne's palsy).
456 Brudzinski's neck sign	When the arms and legs are flexed fully on the trunk and the head is passively bent forward the patient shows signs of pain.	Meningitis.
457 Brudzinski's leg sign	When one leg is passively fully flexed on the trunk the other leg is drawn up by the patient into a similar position.	
458 Grasset and Gaussel's phenomenon	Inability of a patient when lying on his back to raise both legs simultaneously although he is able to raise either leg separately.	Organic hemiplegia (incomplete)

460
Brain stem — Comprises the medulla oblongata, pons varioli and crura cerebri. (Figs. 18–23.)

461
Central motor neurons (upper motor neurons) — Motor cerebral cortex, corona radiata, internal capsule, pyramidal tracts at base of brain, motor decussation and crossed and direct pyramidal tracts in spinal cord. Figs. 15–26.)

462
Peripheral motor neurons (lower motor neurons) — Motorial end plates, peripheral nerves, anterior nerve roots, nerve cells in the anterior horns of spinal cord and the motor nuclei in the brain stem. (Figs. 19, 26.)

463
Central sensory neurons (upper sensory neurons) — Sensory cerebral cortex, corona radiata, internal capsule, cerebellum and its peduncles, lemniscus and sensory decussation, nuclei of columns of Goll and Burdach, antero-lateral ascending (Gower's) tract, direct cerebellar (Flechsig's) tract and column of Clark. (Figs. 15–26.)

464
Peripheral sensory neurons (lower sensory neurons) — Sensory end organs, peripheral nerves, posterior nerve roots, spinal ganglia, posterior horns and columns of Goll and Burdach in the spinal cord and nuclei of columns of Goll and Burdach. (Figs. 22–6.)

465
Cilio-spinal center (335, 1191-2) — Situated in the lateral horn of gray matter in the last cervical and first dorsal segment of the spinal cord and is connected with a higher center in the medulla. Destructive lesions of this center and its nerve roots cause (1st) a paralytic myosis, (2d) a narrowing of the eyelid opening, (3d) an enophthalmus; while irritative lesions (rare) of this center and its nerve roots cause (1st) a spasmodic mydriasis, (2nd) an exophthalmus.

PART II

DIFFERENTIAL DIAGNOSIS

A CLINICAL DIAGNOSTIC ANALYSIS OF THE SYMPTOMS

OBTAINED FROM THE EXAMINATION OF PATIENTS

INTRODUCTION TO THE DIAGNOSTIC CHARTS

DIRECTIONS FOR THEIR USE

In using this book for diagnostic purposes the student, or practitioner, having made a complete examination of the patient according to the scheme presented in chart I, should make note of the more important symptoms. Then, selecting any one of these symptoms, he should turn to the table of contents and see which chart treats of the disorders which include this symptom. Finally, turning to the commencement of the chart indicated, he should apply one test after another until he reaches the diagnosis.

At the left margin of each chart is placed the symptom to be analyzed; on the right margin are placed all the possible diseases in which this symptom can occur. Proceeding from left to right, in each column a number of alternatives are offered, and by selecting the one appropriate to the case the diagnostician proceeds from one column to the next, until he reaches the correct diagnosis. In the column immediately preceding the diagnosis is an abstract of most of the symptoms which may occur at different stages of the disease. The great majority, but not necessarily all, of the symptoms given in the abstract should be present in the history or found in the examination of the case, if the diagnosis be correct.

A few practical examples will illustrate the method much better than a long general description. Let us then consider a few cases as they occur in actual practice. Only the essential symptoms are noted.

Case I. Male, aet. 51.—He smoked and chewed tobacco and drank to excess for years. About two months ago he began to have pains at various points in both legs. His legs became slowly weaker and his flesh became tender, but he is able to walk a mile. Organic reflexes normal. Feet cold, and the legs have lately grown smaller. On physical examination the muscles of the lower legs, and less so those of the thighs are weak, tender and somewhat atrophied. The legs, and especially the feet show slight anesthesia, marked analgesia and well marked retardation of the conduction of pain. Achilles reflex absent. A slight knee-jerk can be obtained with difficulty. In walking toes drop a little and the knees are raised abnormally high.

Important symptoms: PARALYSIS (weakness), HIGH-STEPPING GAIT, ANESTHESIA and PAIN.

The chief symptom in this case is weakness, and we, therefore, turn to chart X, which discusses "diseases causing motor paralysis." The paresis in this case is continuous and the reflex acts are diminished or absent. We, therefore, have to do with a flaccid paralysis and turn to chart Xa. The presence of muscular atrophy following the paralysis, together with the absence of any apparent hypertrophy, guides us in the second column away from the functional diseases and the muscular dystrophies and to the degenerative atrophies; while the normal organic reflexes guide us in the third column away from the spinal cord, and to the peripheral nerve, diseases. The presence of anesthesia, pains, muscle tenderness and other sensory symptoms guide us in the fourth column to the class of neuritis of the spinal nerves. In the fifth column the fact that there are many spinal nerves affected guides us to the diagnosis: Multiple Neuritis, which the history of alcoholic abuse confirms.

We can approach this case in another way by considering his abnormal walk. In the table of contents we find that disorders of gait are treated in chart XIII and indeed in chart XIIIc. The walk in this case is evidently "paralytic and flaccid," the tendon reflexes being diminished. Furthermore it is a high-stepping gait. A comparison of the three possible abstracts with the symptoms of our case makes it evident that the case is one of Multiple Neuritis.

We can trace the case also by means of the anesthesia and analgesia: symptoms which are discussed in chart XIVa. The tendon reflexes being diminished and the organic reflexes normal in this case, we are led to three abstracts, only one of which fits our case, and thus the diagnosis

of Multiple Neuritis is again confirmed. Finally we may take up, the initial symptom in the case: pain in the legs. Pain is discussed in chart XV and pain in the extremities in chart XVc. In our case the pain is bilateral and is associated with anesthesia; so that we are again brought to three abstracts, of which the one of Multiple Neuritis most nearly fits our case.

Case II. Female, aet. 23.—Ten years ago she and her brother had simultaneously an attack of headache, backache and fever. Her brother died and she recovered with a paralysis of both legs, which has since improved, rapidly at first, then more slowly. Her legs are still somewhat weak, especially the left one, but she can walk fairly well. No sensory disturbances, organic reflexes normal. On physical examination there is a decided weakness, slight atrophy and slight shortening of left leg. Knee-jerks are absent in both legs. No objective sensory abnormalities.

Important symptoms: PARALYSIS.

In the analysis of this case we follow the same path traced in case I until we reach column four in chart Xa. In this case there are no sensory symptoms, the paralysis involves neither the cranial nor the extensor nerves exclusively, and is acute in its origin; so that the diagnosis must be Acute Anterior Poliomyelitis.

Case III. Male, aet. 48.—Had a chancre followed by a cutaneous rash twenty-two years ago. During the past ten years has had "lightning pains" in legs and a girdle sensation, also gastric, vesical and urethral crises. During the past six months, his walking has become difficult and awkward and is much worse, practically impossible, in the dark. Organic reflexes normal, except for some delay in micturition. On physical examination there is no loss of muscular power, but all movements of legs are awkward, violent and excessive. There is marked ataxia, anesthesia in areas and well marked retardation of conduction of pain from feet. Complete absence of knee-jerk. Argyll-Robertson's pupillary reflex, Romberg's symptom and loss of muscle sense in legs. Lumbar puncture gave fluid showing the presence of globulin and lymphocytosis and a positive Wassermann. In walking the patient does not stagger, but flings feet out widely.

Important symptoms: ATAXIA, ATAXIC GAIT, ANESTHESIA, PAIN, ABDOMINAL CRISES, and LYMPHOCYTOSIS IN CEREBRO-SPINAL FLUID.

In this case there is no loss of motor-power but well marked ataxia in legs. From the table of contents we learn that disease causing perversion of motion, including ataxia, are treated in chart XIIa, to which we turn. As the patient does not stagger in walking and the movements of the legs are ataxic, not only in walking, but also in other movements, it is certain that the case is one of "motor ataxia." The ataxia is bilateral and the knee-jerks are absent; so that it is evident that we have to do with tabes or multiple neuritis (pseudo-tabes). We differentiate these two diseases by comparing the abstracts of their symptoms. As in this case there is no muscular weakness, atrophy and tenderness, it is plain that the diagnosis is Tabes.

We may also reach a diagnosis in this case by studying the patient's walk with the aid of chart XIIIc. The gait is ataxic, rather incoordinated than staggering, the knee-jerks are abolished and there is Argyll-Robertson's phenomenon, so that the diagnosis of Tabes is confirmed. Furthermore we may trace the case by the symptom of anesthesia with the aid of chart XIVa. The tendon reflexes are abolished. The organic reflexes are not much disordered, but they are slightly. There is no motor paralysis and thus we are led again to Tabes. If we consider the symptom named "Argyll-Robertson's phenomenon," which is present in this case, we shall find it discussed in chart XIVd and here again we are led directly to Tabes. If we consider the pains in the legs or the girdle sensations about the body or the abdominal crises, we find them discussed in chart XV and in either case are led to Tabes. If we consider the results of the examination of the cerebro-spinal fluid with the aid of chart XIX, we find the butyric acid test positive, the existence of lymphocytosis, a positive Wassermann, a clear fluid and ataxia, and thus the diagnosis of Tabes is again confirmed.

Case IV. Female, aet. 19.—Patient's father and mother were first cousins. They had eight children, of whom three died in infancy and four are healthy. Child learned to walk late and with difficulty, frequently stumbled and fell. Was backward at school and when she was nine years old it was evident to all that she was not normal. Patient's movements became gradually and steadily more awkward. Now she cannot walk without aid. General movements are slightly ataxic and simulate somewhat a tremor. Movements of the legs are more ataxic and weaker than those of the arms. Her walk is extremely ataxic and staggering. No knee-jerks, Babinski present. Organic reflexes normal. Internal strabismus. No loss of muscle sense.

Important symptoms: Ataxia and Ataxic Gait.

The most characteristic symptom in this case is ataxia and so, as in case III, we turn to chart XIIa. In this case, the ataxia is mainly on walking and there is no motor paralysis and no loss of muscle sense. We are, therefore, brought to the alternative as to whether the disease occurs in an adult or a child. This case doubtless dates from early childhood. There are no similar cases among her brothers and sisters, but she comes certainly from a tainted family. She has no nystagmus, but has strabismus. As this case began before puberty and has no knee-jerks it is doubtless a case of Friedreich's Ataxia. The strabismus points to Marie's hereditary cerebellar disease and indeed these two diseases are so closely related that there is some question as to whether they are separate entities.

We may approach this case from a different angle. The chief symptom is difficulty in walking. We turn, therefore, to chart XIIc and note that the walking is ataxic. The staggering gait which is permanent, the bad heredity, the absence of knee-jerk and the commencement of the disease in infancy confirms the diagnosis of Friedreich's Ataxia. It may be noted in passing that this case does not show a distinct tremor, or nystagmus, or the blurred speech which symptoms are often present in this disease.

Case V. Male, aet. 62.—His disease commenced with difficulty in speaking and swallowing about a year ago, and has slowly and steadily progressed. His speech has become so bad that it is unintelligible and he has the greatest difficulty in swallowing, and chokes over his food. There is constant drooling of saliva from his mouth. Cannot protrude his tongue beyond his teeth, cannot raise his arms because of weakness of muscles about the shoulders. His legs are somewhat weak. Fibrillary contractions and great atrophy of muscles of tongue and of shoulder girdle (deltoid, pectorals, etc.). Muscles of hands are not involved. Absence of tendon reflexes in arms. Knee-jerks lively, ankle-clonus and Babinski are present. There are no sensory disturbances.

Important symptoms: Paralysis, Fibrillary Contraction and Muscular Atrophy.

The principal symptom in this case is a motor paralysis. We turn, therefore, to chart X. The paralysis certainly is a continuous one and of the three alternatives next offered us we must select the third, inasmuch as we have a flaccid paralysis with muscular atrophy in the head and arms and a mild spastic paralysis in the legs. We turn, therefore, to chart Xc. In this case the cranial and spinal nerves are involved, next there are no sensory symptoms, next the disease is chronic, and finally the lips, tongue, larynx and pharynx are involved; consequently the diagnosis is Progressive Bulbar Paralysis. But this diagnosis does not explain the paralysis and atrophy of the muscles of the shoulder which are supplied by spinal nerves. We turn, therefore, to the next sub-division, where spinal nerves are alone involved, and follow through, no sensory symptoms and through a paralysis involving the shoulder girdle muscles, and reach the diagnosis of Amyotrophic Lateral Sclerosis. The diagnosis is, then, a combination of two diseases: Progressive Bulbar Paralysis and Amyotrophic Lateral Sclerosis, and we find in the abstracts of these two diseases that they often occur together in combination.

If we consider the symptom "fibrillary contraction" with the aid of chart XIIb, it is evident that this is an organic and not a functional disease, that there is a marked muscular atrophy and that there are no sensory symptoms, and thus the diagnosis of both Progressive Bulbar Paralysis and Amyotrophic Lateral Sclerosis is confirmed. Finally if we consider the symptom

"muscular atrophy" with the aid of chart XVIIa we find that the atrophy is considerable and of a relatively rapid course, that there are no muscular hypertrophy and no sensory symptoms and thus we are led again to the same diagnosis.

Case VI. Male, aet. 12.—During the first year of his life the child had great difficulty in retaining his food. At the end of his first year he began to have convulsions with unconsciousness, and with the exception of an interval of two years these have continued up to the present time; the last attack having occurred three weeks ago. The child has a very small head and an idiotic expression of face. He apparently understands most of what is said to him, but he can talk only a very little and only a few words are intelligible. There are no contractions or deformities, and he uses his arms and legs well.

Important symptoms: Anarthria and Idiocy.

The most striking symptom in this case is that a boy of twelve years can scarcely speak intelligently. Turning to the table of contents we find that disorders of speech are treated in chart XIII, to which we turn. The loss of speech in this case is so nearly complete that it can be called anarthria, which is discussed in chart XIIIa. The disease is evidently congenital, and the expression of the face is idiotic, and reading and writing are impossible; so that the diagnosis is Idiocy. Had we on the other hand decided that the child could speak, but very imperfectly and unintelligibly we should have sought for the disease in the same chart XIIIa, under the heading of dysarthria. Here the congenital nature of the defect and the absence of cleft palate, etc., would have led us directly to Imbecility. In order to trace the case further let us follow the cross reference after idiocy which is 1081 and which we find in chart XVIc. This case on account of his convulsions might be classed under Epileptic Idiocy or on account of his small head under Microcephalic Idiocy, or under both.

Case VII. Female, aet. 53.—Complains of trembling and that she cannot execute any movements quickly, because her arms and legs are stiff and rigid. When walking she has a decided tendency to pitch forward. Feels warm at times when the room seems cool to others. Expressionless face, passive tremor of hands. Propulsion and retropulsion when walking or standing. Rigidity of arms and legs. Difficulty in rising from a low chair. Knee-jerks rather increased.

Important symptoms.: Muscular Rigidity, Tremor and Abnormal Walk.

The most characteristic symptom in this case is the rigidity of the arms and legs which is a mild tonic spasm. From the table of contents we learn that diseases causing spasm are treated in chart XI, to which we turn and find that general tonic spasm is discussed in chart XIb. In this case there is no fever and of the five sub-divisions under this head, this case clearly falls in the second: "rigidity which does not prevent passive or voluntary motions." Of the two alternatives next offered it is evident that we must choose the second, in the abstract of which we find all the symptoms present in our case. The diagnosis is, therefore, Paralysis Agitans.

If we follow the symptom "tremor," we find this treated in chart XIIb. It is a passive tremor and, whether it be increased or diminished on voluntary movements, if it be slow, the abstracts show that it is a case of Paralysis Agitans, because the other abstracts do not fit this case at all.

If we consider the difficulty in walking in this case we turn to chart XIIIc. In the three great divisions offered this case evidently falls in the third: "paralytic and spastic;" and of the two sub-divisions next offered we must take "general rigidity" which leads us again to the diagnosis of Paralysis Agitans.

Case VIII. Male, aet. 59.—During the past 34 years has had at times attacks of asthma. During the past four years has been troubled by a great many paroxysmal attacks of vertigo, at irregular intervals; some are slight, some are so severe as to throw him from a chair half way across the room to the floor, where he must lie for several hours, because when he raises his head from the floor he vomits violently and the dizziness becomes worse. He often has slight attacks of vertigo, which make him stagger when walking. During these four years he has been slowly

growing deaf in his left ear; the deafness being now extreme. He has also had during the same time in the same ear, a buzzing and a ringing which is most intense just before an attack of vertigo. He has no paralysis and no loss of muscle sense. Bone conduction is absent. He also is much troubled by gastric flatulence, to which he attributes his vertigo, but when he takes digestive medicine and the digestive disturbances are relieved, the vertigo remains unchanged. His eyes were found to be astigmatic and proper glasses used, but no improvement in the attacks of vertigo followed.

Three years after the above record was made the attacks were milder and less frequent, but at that time his left ear was totally deaf and deafness was advancing in his right ear.

Important symptoms: Paroxysmal Vertigo, Staggering Walk and Deafness.

In this case the principal symptom is paroxysmal attacks of vertigo; diseases causing which, we learn, are treated in chart XVd. We see from this chart that vertigo may be caused by digestive disturbances and disease of the eye, both of which were present in this case, but the vertigo persisted when these abnormal conditions were relieved; so that they could hardly be the cause. On the other hand, we find that vertigo is associated with deafness: a prominent symptom in this case, and in looking over the abstract of this form of vertigo we see that it fits the case exactly; so that the diagnosis is Ménière's Disease.

If we consider another symptom "the occasional staggering in walking," we find this treated in chart XIIa. This patient has no loss of muscle sense and no muscular paralysis. He is an adult and his hearing is abnormal and thus we are led again to Ménière's Disease. Finally if we trace the symptom "deafness" with the aid of chart XIVe we find that the deafness, at any rate at first, was unilateral, that bone conduction is absent, that there is no facial paralysis and that severe paroxysmal vertigo and tinnitus aurium is present; thus confirming again the diagnosis.

Case IX. Female, aet. 17.—Heredity good. Was well until about three years ago when, at the time of commencing menstruation, she began to have attacks of clonic convulsions with unconsciousness, which have continued up to the present time and in which she has occasionally bitten her tongue. Has also lesser attacks of unconsciousness, or very cloudy consciousness in which she automatically prays, or says foolish things. Has no memory of any of her attacks. She has an immediate aura of fire before her eyes and of wheels revolving in her head. Some headache follows the attack. The convulsions occur only, and the lesser attacks mainly, at night. Physical examination is negative, urine normal. Fundus of eye normal. Knee-jerks equal. Much acne on face.

Important symptoms: Coma and Convulsions.

The constant symptom in all her attacks is unconsciousness, or coma, of short duration. This symptom is treated in chart XVIa. There is no history of recent injury, of brain disease, of poisoning, of heart disease, of paralysis, of kidney disease or of fever. Therefore, we are led at once to the diagnosis of Epilepsy or of Eclampsia. The latter can be excluded by the frequently recurring attacks at long intervals.

If we next take the symptom of clonic convulsion with the aid of chart XIa, we find that there is no fever and the convulsion is a universal one, and not local at the onset. There is coma and there are no symptoms of disease of the brain or cord, or of the kidneys, heart or blood, or of poisoning and thus we confirm the diagnosis of Epilepsy.

Case X. Female, aet. 34.—Nine years ago one morning, her left arm, leg and side of face felt numb and she could not see things on her left side without turning her head. These symptoms steadily increased during the day and she could not use her leg, and especially her arm, well. She could always walk, but at first she could walk only with difficulty. This difficulty in walking gradually passed away. She could use her arm, but could not use it well for more than a year, and it is not quite right even at the present time. The numbness of the left side and the inability to see things on her left still persist. On physical examination there is found anesthesia and analgesia of the left arm and leg and of left side of body and face, (left hemianesthesia and hemianalgesia), also blindness in each eye for all objects to the left of central

vision (left homonymous hemianopia). The left arm and leg are a little awkward and a trifle weak; strength of left hand grasp to that of right is as 80 to 105. Knee-jerks lively, perhaps stronger on left side: neither ankle-clonus nor Babinski. Organic reflexes normal.

Important symptoms: HEMIANESTHESIA, HEMIANALGESIA and HOMONYMOUS HEMIANOPIA.

The principal symptoms of this case are hemianesthesia, hemianalgesia and homonymous hemianopia. These are sensory symptoms and indeed symptoms of a diminution of sensation. We turn to the table of contents and find that "diseases causing a diminution of sensation" are considered in chart XIV, which we next consult. Starting with disorders of sensation in the first column, we have five alternatives offered us in the second column, among which we ought, without doubt, in this case to select diminution of sensation and following this division we have in the next column three alternatives, among which, undoubtedly, we should select anesthesia and analgesia and turn to chart XIVa. In this chart we have the alternatives of the tendon reflexes being either absent or present. In the above case they are present. The dilemma in the next column is quickly decided because the organic reflexes are normal. The history of a motor paralysis lasting a year or more and still slightly persisting directs us to the first alternative in the next column, especially as there are no hysterical symptoms present; while the unilateral nature of the symptoms and next the acute onset (one day) brings us to the diagnosis of Cerebral Hemorrhage or Softening.

To determine which lesion is present, we follow the first cross reference, No. 503-6, which we find in chart Xb. In looking over the abstracts differentiating cerebral hemorrhage, embolism and thrombosis, our case, with its relatively slow onset, its absence of any coma, its absence of any source for an embolism, is probably one of cerebral thrombosis and certainly one of cerebral apoplexy.

The next question is as to the locality of the softening. To ascertain this we turn to the table of contents and find that "localization from symptoms of paralysis" is discussed in chart XXII to which we turn. The reflexes being present in our case, we are brought to the question whether sensory or motor paralysis is dominant. In our case sensory paralysis is dominant and we turn to chart XXIIc. Of the first alternative offered us in this chart we must choose the first: anesthesia and analgesia. In regard to the next column, the distribution of the anesthesia in our case evidently falls into the class: "the (left) arm, leg and face are anesthetic." In our case there is no Jacksonian epilepsy and there is hemianopia, so that the localization of the softening is in the posterior part of the right internal capsule. If we now turn to fig. 17 we can easily see how a lesion in the posterior portion of the internal capsule can easily involve the sensory fibers from one-half the body and also the optic fibers; the continuation of the optic tract. It is also easy to understand that on account of the wide-spread circulatory disturbances in the early stages of the disease, before a collateral circulation had to a degree reestablished itself in the periphery of the lesion, the motor fibers lying directly anterior should be involved and a more or less temporary hemiplegia should occur, as was indeed the case. It seems strange that deafness did not occur in this case as it is certain that the auditory fibers also must have been involved in the lesion, but it is well known that central lesions only produce deafness, even unilateral deafness, when the lesion is bilateral (see page 8).

Thus we have arrived by means of the charts to the diagnosis of this case of "thrombosis of the artery supplying the posterior portion of the internal capsule," but in order to make this diagnosis doubly sure, let us take another one of the prominent symptoms, such as homonymous hemianopia, and follow it through the charts. This symptom is also a diminution of sensation and therefore we turn again to chart XIV. Disregarding this time diminution of sensation we follow "disturbances of vision" and "limitation of field of vision" to chart XIVb. Here we find homonymous hemianopia and in the next column there can be no doubt that we must choose the path which hemianesthesia indicates and by it are led to the diagnosis of hemorrhage, or softening, in the posterior part of the posterior limb of the opposite sided internal capsule, which is the diagnosis to which we had already reached by another road.

CHART X

Motor Paralysis

DIAGNOSTIC ANALYSIS OF SYMPTOMS.

Symptoms Analyzed	Tests: Permanence of Paralysis	Tests: Reflexes in Paralyzed Muscles	
469 MOTOR PARALYSIS OR PARESIS (244)		Abolition or diminution of both voluntary and reflex acts in the muscles involved.	
		472 FLACCID PARALYSIS (252)	The differential diagnosis of those diseases in which FLACCID PARALYSIS occurs is set forth in CHART X a.
		Lesions of peripheral motor neurons.	
		There are hypotonia and changes in the electrical reaction of the nerves and muscles involved in very varying degree from simple diminution in excitability, to complete reaction of degeneration.	
After a careful examination has shown that the paralysis is a true one and is not simulated by any ankylosis or by pain on motion.	470 CONTINUOUS PARALYSIS	Abolition or diminution of voluntary, with persistence or even exaggeration of reflex, acts in the muscles involved.	The differential diagnosis of those diseases in which SPASTIC PARALYSIS occurs is set forth in CHART X b.
		473 SPASTIC PARALYSIS (251)	
		Lesions of central motor neurons.	
		There is hypertonia without alterations of electrical reaction of the nerves and muscles.	
		474 A combination of FLACCID PARALYSIS in the upper part of the paralyzed area, and of SPASTIC PARALYSIS in the lower part.	The differential diagnosis of those diseases in which there is a combination of FLACCID and of SPASTIC PARALYSIS, and of those in which INTERMITTENT PARALYSIS occurs is set forth in CHART X c.
	471 INTERMITTENT PARALYSIS.	All the muscles of the body and head.	
		The muscles of one or both legs, rarely of arms.	
		Commencing in legs, extending to arms	
		Associated with a cervical rib.	

CHART X a

Flaccid Paralysis

Comprising Numbers 475 to 477 on left side of Chart
and 482 to 500 on right margin

472 FLACCID PARALYSIS

475 No muscular atrophy, except rarely in chronic cases. Reflexes may be diminished only, not abolished.

- Paralysis beginning in the feet and ascending to the head in *adults*. — The paralysis is in the form of a paraplegia, commencing in the feet a ing bulbar symptoms (434), and causing death usually in the second are pronounced the disease is probably a neuritis (488). The spleen dry's paralysis and in addition hematoporphyrinuria. (Compare care
- No true paralysis but great hypotonia in *infants*. — Occurs usually congenitally, rarely during the first year of life. No he in very abnormal positions. The child cannot use the slight power ances, no disturbances of organic reflexes. Electrical reaction much di

476 Muscular atrophy, usually great, following the paralysis after the second week of the disease.

Paralysis primary.

The Degenerative Atrophies. (See also Syringomyelia —552, 837–9.)

- The organic r flexes a r e permanently disordered (1 and 323–4).
  - Marked sensory symptoms, such as pain, paresthesiae, anesthesia, etc., are present with the motor symptoms. The legs only are paralysed and exhibit trophic disturbances. There is incontinence of urine and the bladder is empty or nearly so.
    - Very acute onset. Sympton thesia. May be blood in c
    - Acute, sub-acute or chronic other evidence of syphilis
    - Very chronic and progressiv
    - Very acute, acute or sub-acu of the cord. Fibrillation c perineum, genitals and mo
  - Sensory symptoms, such as pain, nerve and muscle tenderness, paresthesiae, anesthesia, etc., are present.
    - Many spinal (very rarely cerebral) nerves are affected. — There is usually a history of acute, sub-acute and chr and arms (long neurons) a weakness, atrophy and te duction of pain and loss o form of Korsakow's psych form rarely runs an acute ical form exhibits many s
    - The paralysis is coincident with the distribution of one, rarely of a few spinal nerves. — The motor and sensory paralysis (anesthesia) m a y be slight and the pain great. The motor paralysis is usually greater than the sensory.
      - Nerve Atr
      - The m han
      - One o
  - In very exceptional cases sensory symptoms may be practically absent.
- The organic reflexes are normal or show only transitory disturbances (1 and 323–4).
  - The paralysis is confined to the distribution of one or more cranial nerves.
    - Occurs most commonly in inj For special forms of neuriti
    - The paralysis is usually mor
  - Extensor muscles are alone affected. — Usually confined to the exte lead poisoning. Previous a
  - No sensory symptoms, except rarely pain and tenderness over the nerve trunks in early stage.
    - A paralysis of acute onset, usually confined to the arms and legs, generally to a portion of one or both; in rare cases involves the cranial nerves. — The paralysis (which is alway sions. Rarely there is sligh so in children. The paraly In infants and young chil neuritis with predominatin toms are common and th of functionally related mu The muscles of the trunk and scurvy (Barlow's disea (4) encephalitic form, (5) Chronic atrophic paralysis (
    - A chronic form, may occur exceedingly rarely. — (Compare carefully with 4
    - A paralysis of chronic onset commencing in peronei muscles and extending symmetrically. Intrinsic muscles of the feet affected. — The di muc app out

477 A combination of muscular atrophy and apparent hypertrophy.

Paralysis secondary.

The Muscular Dystrophies.

A chronic disease commencing in childhood or youth and usually showing marked heredity. It exhibits a progressive muscular atrophy, usually combined with some hypertrophy, hence called muscular dystrophy. In time all the muscles become atrophied. The organic reflexes are normal and there are no sensory symptoms whatever and no motor paralysis, except such as would result from the muscular degeneration. Even the apparently hypertrophied muscles are weak. Tendon reflexes are early much diminished and finally absent in the affected muscles. There are no fibrillary contractions. The course of the disease is progressive, but very chronic, lasting many years. From its point of commencement the atrophy extends throughout the body. It produces a marked lordosis. Although the muscular dystrophies are divided into three groups, there are many transitional and mixed forms, and the examination of the excised muscles also shows mixed forms.

- The disease is extremities. muscles show with a mixtu trophy in dif ent parts of t More or less
- The disease is tremities. E cles shows de mixture of a and especiall terstitial fat.

DIAGNOSTIC ANALYSIS OF SYMPTOMS

ABSTRACT OF SYMPTOMS

	DIAGNOSIS	
r ascending the legs and body. Before the legs are completely paralysed the paralysis appears in the hands and rapidly ascends the arms. The ascending paralysis reaches the medulla and pons; pro-luc- ough some rare cases may recover. The organic reflexes are usually normal, except as affected by weakness of the abdominal muscles. There is little pain and little or no anesthesia. If sensory symptoms enlarged. There is rarely fever, and no electrical reaction of degeneration (399). Certain drugs, especially the prolonged use of trional or sulfonal, may cause symptoms closely resembling those of Lan- 488 and 495.)	Acute Ascending (Landry's) Paralysis.	482
luscles exhibit extreme want of tone and flabbiness. The legs are always, the arms are often, affected but in less degree. The mobility of the joints is greatly increased and the extremities can be placed ary muscular contraction still remaining to any advantage. The child can neither sit, stand, nor walk without assistance. Tendon reflexes absent, no muscular atrophy, no fibrillation, no sensory disturb- but no reaction of degeneration. The tendency of the disease is towards improvement, but not to complete recovery.	Myatonia Congenita. Amyotonia Congenita.	483
st extensive in first few days and may improve later. May be deformity in lower dorsal region of the vertebral column. The anesthesia always present is not preceded by hyperes- inal fluid.	Lumbar enlargement of spinal cord. (Figs. 24–7.) — Injury of, or hemorrhage in,	484
ymptoms continue to extend for some time and are fairly symmetrical. Fibrillation may be present. The anesthesia usually present is not preceded by hyperesthesia. A history or often found in syphilitic myelomalacia (1211) which is not as frequent in the lumbar enlargement as it is higher in the spinal cord.	Lumbar enlargement of spinal cord. (Figs. 24–7.) — Acute or chronic myelitis or myelomalacia of (791, 825, 1148a, 1309).	485
Symptoms at first mainly unilateral, becoming bilateral later. Cerebro-spinal fluid is usually under increased tension.	Lumbar enlargement of spinal cord. (Figs. 24–7.) — Tumor in, or compressing, (826, 1309).	486
usually sub-acute. Much pain in perineum and genitals. May be deformity in lumbar region of spine. Bed-sores are less common and symptoms less symmetrical than in lesions n the peronei muscles. When the lesion is low down the muscles on anterior aspect of thigh are not paralysed and knee-jerks are normal. The anesthesia always present in the extensively over legs, is often preceded by hyperesthesia.	Lesions of the cauda-equina (1007, 1308). (Fig. 29.)	487
lcohol, etc.) or infection (diphtheria, etc.). The paralysis is usually in the form of a paraplegia and increases in intensity during the first few weeks, but in some cases reaches its height in a few days; s. The extensors are usually weaker than the flexors and the consequent foot-drop causes, when walking is possible, a high stepping gait. The paralysis commences at the distal extremity of the legs ds towards and often involves those of the body. The cranial nerves are rarely affected and then especially the long ones (phrenic, pneumogastric, facial, etc.). The most characteristic symptoms are of the muscles affected, at first the small muscles of the hands and soles of the feet. In addition, there are usually pain, paresthesiae, mixture of hyperesthesia and anesthesia, ataxia, retardation of con- sense. Edema and trophic disturbances are common. The atrophy and emaciation are often extreme. Not infrequently there is fever, and in alcoholic cases there are mental disturbances usually in the 0). No globulin or leucocytosis in cerebro-spinal fluid. Great variations in type occur. Some cases may be purely motor (diphtheria, etc.), others mainly sensory and others mainly ataxic. This last acute ataxia." The diphtheritic form may involve the muscles of the extremities, but it always first appears in, and is usually limited to, the uvular and ciliary (accommodation) muscles. The arsen- sory disturbances and a peculiar bronzing or pigmentation of the skin. (Compare carefully with 482 and 495.)	Multiple Neuritis. Polyneuritis. Pseudo-tabes. (662, 787, 823, 1008, 1147, 1307). (Figs. 33, 38).	488
when it can be felt and is usually the seat of various paresthesiae and of intense pain. In severe cases a complete, or partial, electrical reaction of degeneration and trophic disturbances are present. he muscles involved and subsequent active or passive contractures of the antagonists may occur. For description of special forms of neuritis or injury, see Chart XII c.	Neuritis or injury of a spinal nerve (sciatica, etc.), (822, 941, 1146–7, 1173, 1303–5–7). (Figs. 33, 38).	489
sensory symptoms coincide with the area of distribution of one or more nerves entering into the brachial plexus. Muscles of shoulder and upper arm, 5th and 6th cervical (Erb's paralysis); or muscles of earm with oculo-motor symptoms at times, 7th and 8th cervical and 1st dorsal nerves (Klumpke's paralysis, 454–5)).	Neuritis or injury or tumor of brachial plexus (Erb's or Klumpke's paralysis), (454-5). (Figs. 33, 38).	490
nder tumors can be felt on one or on many nerves, which are usually the seat of paresthesiae and of more or less pain, often severe.	Single or multiple Neuromata, (938). (Figs. 33, 38).	491
sure to cold, extension of inflammation, syphilis and tumor at base of brain. The paralysis is not accompanied by symptoms of central disease. All the muscles supplied by the nerve are usually paralysed. y see Chart XII c.	Injury or inflammation of one or more motor cranial nerves (facial paralysis, etc.), (1301-7). (Fig. 19.)	492
ead and accompanied by other symptoms of central disease. Only a portion of the muscles supplied by the nucleus may be paralysed. May be associated with atheromatous arteries or infection.	Inflammation, softening or hemorrhage involving one or more motor cranial nuclei (motor oculi paralysis, etc.), (1304). (Figs. 18-21.)	493
he wrist and fingers causing wrist-drop. Supinator longus not involved (716). Rarely the muscles of dorsal flexion of foot are involved, causing foot-drop. Blue line on gums. History of exposure to lead colic, arthralgia, etc. Lead can be detected in the urine after the administration of iodide of potassium.	Lead Palsy, (584, 788, 1050).	494
motor and never sensory, except temporarily in the legs in very rare cases) may rarely come on abruptly, but is usually preceded by fever and signs of meningeal irritation, exceptionally even convul- and muscle tenderness. Lymphocytosis and globulin in cerebro-spinal fluid in the acute stage. In a small percentage of cases the disease rapidly terminates fatally. The onset is always acute, especially atest during the first week or two and may improve rapidly at first and later more slowly, but recovery is usually only partial; some or many muscles remaining paralysed and atrophied throughout life. parts may remain more or less undeveloped. The vast majority of cases occur in the first three years of life, but the disease may occur in adults and then may, in rare cases, closely resemble multiple symptoms. Deformities, contractures, subluxation of joints from relaxed or contractured muscles and ligaments, and arrest of growth are common in young children. Trophic and vaso-motor symp- ar atrophy is usually extreme. The paralysed parts are usually cold and cyanotic, and the paralysed muscles exhibit the reaction of degeneration. The paralysis may have any distribution, but groups always affected. The more widespread the paralysis, the greater will probably be the improvement. The legs are more frequently affected than the arms and the extensors more than the flexors. paralysed. Cranial nerves rarely affected (acute encephalitis). An infectious disease which may occur in epidemics, especially in warm weather. In young children the gums must be carefully examined be carefully excluded. Poliomyelitis has been divided into a number of different forms which are partially described by their names (1) spinal form, (2) Landry's paralysis form, (3) brain-stem form, ebellar form, (6) neuritic form, in which form slight muscle tenderness may occur, (7) meningitic form, (8) abortive form. Some cases of porencephaly (501) may have the same etiology and pathogenesis. possibly be regarded as a chronic form of this disease with a very different etiology and pathology. Whether there is a true chronic anterior poliomyelitis is very doubtful. If it exists it is certainly very rare. 8.)	Acute Anterior Poliomyelitis. Myelitis of the Anterior Horns. Infantile Paralysis. Acute Atrophic Paralysis. Heine-Medin's Disease, (789, 1148, 1233). (Figs. 24-6.)	495
ally commences before the age of twenty years and was formerly considered one of the muscular dystrophies as there is often a well marked heredity. Usually the paralysis and atrophy do not extend the knees. In the later stages of some rare cases symptoms of atrophic paralysis have developed in the hands and arms. The muscles obtained by harpooning show simple degenerative atrophy, and no ertrophy. In rare cases there may be pain. Fibrillary contractions and reaction of degeneration are present. Club-foot is common. Leyden has described a form of hereditary muscular atrophy with- phy, attacking the legs in the first ten years of life, in which the atrophy involves especially the muscles attached to the pelvis.	Spinal or Neuritic Muscular Atrophy. Peroneal type, and Charcot-Marie-Tooth's type, of muscular atrophy, (696).	496
ked in the upper ation of exsed eration of fibers ophy and hyper- ers and in differ- degenerated fiber. ial fat is present.	Simple Idiopathic Muscular Dystrophy — The disease usually commences between the ages of 15 and 35 years. The atrophy begins in the orbicularis oris and extends to the other muscles of the face, to the shoulder girdle and finally to the legs. Patient cannot whistle, nor speak well, nor drink easily. Lips are thin and protrude; "tapir mouth." When smiling the mouth elongates only, its corners are not raised. Some muscles of shoulder are often apparently hypertrophied at first and later atrophied, or may be atrophied from the start. — Facio-scapulo-humeral type, or Landouzy-Dejerine type, of dystrophy, (1152).	497
	Simple Idiopathic Muscular Dystrophy — The disease usually commences between the ages of 12 and 15 years. The pectorals, trapezoid, deltoid, etc., are often hypertrophied at first and later atrophied. The biceps and especially the triceps are atrophied from the start. Motion of shoulder and arm much impaired. The face muscles are not involved until late in the disease, if at all. — Scapulo-humeral type, or Erb's juvenile form, of dystrophy, (786, 1152).	499
rked in lower ex- on of excised mus- on of fibers with a and hypertrophy, quantities of in-	The disease usually commences between the ages of 2 and 7 years, but sometimes much later. The calf muscles first show apparent hypertrophy, followed by the extensor cruris, infra-spinati, deltoid, etc. Other muscles are atrophied. All muscles finally become atrophied. Even hypertrophied muscles are weak, but not so weak as those which are atrophied. Marked lordosis and weakness of serratus magnus muscle. In rising from the ground patient has to push himself up with his arms and crawl up upon himself. Waddling gait. Deformities and contractures occur in the later stages and the lordosis is later replaced by a marked kyphosis. — Pseudo-hypertrophic Paralysis, (786, 1156).	500

CHART Xb

Spastic Paralysis

Comprising Numbers 478 to 481 on left side of Chart
and 501 to 527 on right margin

473 SPASTIC PARALYSIS

478 Hemiplegia or Diplegia or Monoplegia (254–5, 258)

Congenital or acquired in infancy. There may be fever or apoplectic symptoms at onset.

A motor paralysis of one (infantile hemiplegia) or both sides (Little's dis... are common and may mask the exaggerated reflexes. In walking there... and at times idiocy or insanity. Frequently there is a partial arrest of... of cerebral diplegia, bulbar symptoms (434) are present without fibrill... expressions, etc., can occur involuntarily, but no voluntary motions. T...

479 Hemiplegia, or Monoplegia (254, 258) (See also Syringomyelia—552, 837-9.)

Almost always in adults and after middle life. Sudden onset, or stroke (ictus), usually with coma (205 and 1037), or with headache or vertigo and mental confusion. Not infrequently the attack commences with a hemiplegia which may or may not be followed by coma.

Symptoms of irritation (convulsions, rigidity, etc.) are more pronounced than are symptoms of paralysis.

The disease i... ache and n...

Symptoms of paralysis are more pronounced than those of irritation (convulsions may occur, especially in cortical lesions and in hemorrhage into the ventricles, in which case lumbar puncture may yield a bloody fluid). The paralysis is in part temporary and in part permanent in varying degree. Slow improvement with almost perfect recovery in rare cases. More or less permanent mental impairment, often very slight. Usually patients are more emotional than previously. Exaggerated reflexes and ankle-clonus are present after coma has cleared up. Babinski is present from the start. Puffing, stertorous respiration is common. Cheyne-Stokes' respiration (435) and tracheal rales are very unfavorable symptoms.

A sudden att... the upper, ... balls, away... arterial dis... face, then ... hemiplegic ...

Similar to th... arterial te... mon than ...

Similar to th... syphilis. ... basilar art... hemorrhag...

Gradual onset without coma, except as a terminal symptom.

Sensory symptoms are always present. Organic reflexes are normal or only slightly disordered.

Brain symptoms. Steadily increasing psychic disorder, and local motor and sensory disturbances over the same area.

Headache, v... mon. Cho... more rarel... mental du... usually pr... increased t... compressio...

Spinal symptoms. Paralysis of motion and sensation on opposite sides of body.

Characteristi... Symptoms ... spinal epil...

Choreic symptoms.

The paralysis is only slight and follows or accomp... a paralysis (chorea mollis).

480 Paraplegia (257) (See also Syringomyelia—552, 837-9.)

There is paralysis always of motion and commonly of sensation, usually in the form of paraplegia, more rarely in the form of a spinal hemiplegia (442), which later may become a paraplegia. The reflexes are exaggerated. Ankle-clonus and Babinski are present. Spasms and contractures and bed sores are often present. The organic reflexes are disordered. The motor paralysis is permanent or lasts a very long time. Sensory paralysis may be slight and transitory and may be altogether absent. The anesthesia is often limited above by a narrow zone of hyperesthesia.

Cranial and spinal nerves are involved.

Intention tremor, nystagmus, scanning speech, ata...

Arms and legs are paralysed. Priapism is common, also respiratory difficulty and early death. Radiating pains are common.

There may be a history of injury and a fracture o...

No history of injury. Little or no pain. Sensor... syphilitic myelomalacia (1211).

May be history of remote injury. Much pain radia... spinal fluid.

Legs only are paralysed. Girdle sensation and pains radiating into the extremities are common.

There may be a history of injury and a fracture o...

No history of injury. Little or no pain. Sensory ... tic myelomalacia (1211).

May be a history of remote injury. Much girdle pa...

Evidence of Pott's disease or tumor compressing t... pain. In cases of compression due to Pott's dis... under increased tension and may contain globul...

History of working under increased atmospheric p...

Old age, atheromatous arteries, arterial tension usu...

Tumor can be seen or felt on back replacing the ... is involved, or not. Club-foot is common.

Signs of irritation predominate over those of par... unless the cord is also involved. Usually a hist...

Legs mainly involved. Arms involved later and slightly, if at all. These diseases may occur in severe anemia.

No ataxia. — Paralysis purely motor, a paresis rat... passive motion, especially when ... a multiple sclerosis (659). Erb's s... This disease may be simulated, in ...

Ataxia. — There is a combination of motor pa... In some cases, especially toward ...

481 Paralysis of any extent: local, monoplegia, hemiplegia, or paraplegia

Paralysis limited by some prominent anatomical landmark.

The motor paralysis is usually accompanied by a great variety of sens... the physician (imaginary or delusional paralysis). A paralysed limb ... retention of urine is common. Hysterical symptoms (425). Impossi... this is sometimes of value in diagnosis.

DIAGNOSTIC ANALYSIS OF SYMPTOMS

ABSTRACT OF SYMPTOMS

the body, or of one extremity, without anesthesia and with very little muscular atrophy (from disuse). A squint is common. The legs are often mainly or alone paralysed. Rigidity and contractures
n adductor spasm, "scissors gait" (795–9). Extensor spasm is also frequent. Epileptiform convulsions are common, both at onset and during the course of the disease. There is much mental weakness
of the paralysed part. Athetosis and post-hemiplegic chorea and other motor disorders are common. Speech is commonly affected (dysarthria). The organic reflexes are not disordered. In some cases
atrophy, and automatic and mimetic actions are preserved. In such cases the eyeballs will follow a light or other object, but cannot be turned by any effort of the will, and laughing, weeping, emotional
is often smaller on the side of the affected cerebral hemisphere.

the result of injury. When not so, prodromal symptoms (432) are usually present. Convulsions are common, both at the onset and during the course of the disease, which is often intermittent. Head-
ispairment are frequent. The onset of paralysis is apoplectiform. It is usually motor only, is rather mild in degree and is usually transient. Organic reflexes normal. Cerebro-spinal fluid often bloody.

ralysis usually of one-half of the body, usually of motion only, rarely of sensation only, sometimes of both. The lower branch of the facial nerve is much more completely and permanently paralysed than
ay be normal. Onset is usually accompanied by profound coma (205) of several hours or days duration, but not always. In the coma there is often turning of the head, and conjugate deviation of eye-
e hemiplegic side. Pulse is slow. Slight variations of temperature about the normal point; when the variation is extreme the prognosis is bad. Prodromata are rather rare (432). There is often
d nephritis and usually high arterial tension. The disease usually occurs in advanced life. If death does not occur in the coma, there is usually more or less improvement in the paralysis; first in the
leg and lastly in arm. Contractures, causing flexion of joints of arm and extension of joints of leg, are frequent and of bad prognosis as regards recovery from the paralysis. Athetosis and other post-
isturbances occur in rare cases.

but onset is more often instantaneous and coma is usually less profound and long and frequently is entirely absent. There are no prodromata. There is cardiac disease or other source for an embolus. The
usually low. The disease usually occurs in youth or middle age. In general the symptoms are less severe and less permanent than in hemorrhage. Convulsions, aphasia and monoplegias are more com-
rrhage, because the lesion is more apt to be cortical. Multiple (more than two) recurrences are more common in embolism than in hemorrhage.

but onset may be more gradual, although still sudden and even at times instantaneous. Prodromata (432) are common. Coma is more frequently absent or less profound. There is often a history of
erial tension is usually high. Arterial disease is common. The disease usually occurs in advanced life. Bulbar symptoms are more common than in hemorrhage or embolism, because branches of the
more frequently affected. Simultaneous paralysis of many cortical functions is more common than in hemorrhage. Multiple (more than two) recurrences are more common in thrombosis than in either
olism.

omiting and convulsions, general or local, are com-
or optic neuritis is usually present in tumor, much
cess. Steadily increasing inertia and more or less
Focal symptoms, both spasmodic and paralytic, are
specially Jacksonian epilepsy (431,605). Markedly
f cerebro-spinal fluid and slow pulse in stage of
ially in tumor.

No fever. No infection, or origin for abscess. Chronic course. Steady progression of all symptoms. Often history of previous remote injury. No increase of cellular elements found in the blood or in cerebro-spinal fluid obtained by lumbar puncture. Frequently symptoms irritative, rather than paralytic. Percussion of skull over region of tumor often shows tenderness. Headache is very rarely absent and is usually intense. It may be general or local, but is of little or no value in localizing the tumor. The paralysis commences as a monoplegia and very slowly (weeks or months) extends.

Often fever. A source of infection, especially suppurative diseases of the ear. Rapid course, except that a latent period in the progress is common, followed by a rapid termination. Delirium is common. Often a history of a recent injury. Leucocytosis may be found in the blood and in the cerebro-spinal fluid obtained by puncture (74). Frequently symptoms rather paralytic than irritative. The paralysis commences as a monoplegia and rather slowly (days) extends.

ons are motor paralysis, loss of muscle sense and ataxia on one side of the body; with analgesia, thermic and sometimes tactile anesthesia of other side (442). Stationary, or steadily progressive chronic course.
ng bilateral. Reflexes vary with the position of the tumor. They may be absent, but are usually increased on the side of the motor paralysis, and are so greatly increased frequently as to cause
3).

attack of hemi-chorea, involving the same side of the body. Symptoms may be bilateral, but are then difficult of recognition. In many cases the hypotonia (almost invariably present) may simulate

gularly distributed motor and sensory paralysis.

rae with deformity. The knee-jerks may be abolished in the early stages, later increased. There may be blood in the cerebro-spinal fluid.
ms and disorders of the organic reflexes are almost always present. A history or other evidence of syphilis (1205) is often present in cases of
o arms. Much pain and rigidity and spasm of back. Symptoms at first more unilateral. Local pains. May be increased tension of cerebro-

Very acute onset / Acute onset / Chronic onset / Very chronic onset } Cervical region of spinal cord above cervical enlargement. (Figs. 24-6.)

rae with deformity. The knee-jerks may be abolished in the early stages, later increased. There may be blood in the cerebro-spinal fluid.
s and disroders of the organic reflexes are almost always present. A history or other evidence of syphilis (1205) is often present in cases of syphili-
idity and spasm in back. Symptoms at first unilateral. Local pain. The tension of the cerebro-spinal fluid may be increased.

Very acute onset / Acute onset / Chronic onset / Very chronic onset } Dorsal region of spinal cord. (Figs. 24-6.)

. Rigidity and spasms in muscles of legs and back are frequent. Usually intense pain when spine is bent or moved and especially on getting out of bed in the morning. Much girdle pain and radiating
ere may be no sensory symptoms. Reflexes may be so exaggerated as to constitute spinal epilepsy (60–1, 443), but vary according to seat of lesion. Contractures may occur. Cerebro-spinal fluid is
ymphocytosis.

Headache, vertigo and vomiting in early stage of disease.

a, loss of memory. Emotional and exhibits mental impairment. Paralysis is not severe, resembles that of paralysis agitans without tremor, and reflexes of all kinds are not much altered.

f the vertebrae, or the cleft can be felt in spine without any visible tumor (occulta). There may or may not be paralysis. Reflexes may be present or exaggerated according as the lumbar enlargement

Much rigidity, girdle and radiating pains and spasm in back muscles. There is usually spasmodic retention of the urine in early stages. Paralysis is of sudden onset, slight in degree and disappears soon,
injury. Reflexes exaggerated. Lumbar puncture may yield a bloody fluid. Symptoms vary with position of hemorrhage.

n a complete paralysis, very slowly progressive and often stationary during long periods. Spasm, rigidity of leg muscles and later contractures. Greatly exaggerated reflexes. Legs offer great resistance to
Ankle-clonus, Babinski and spinal epilepsy (443). Organic reflexes little if at all disordered. Arms usually not affected, but may be slightly so after years. This clinical picture is at times the early stage of
spinal paralysis, although in it the reflexes are not always greatly exaggerated and the posterior columns are at times involved, cannot be distinctly separated from this disease, except perhaps by its etiology.
mild form, with exaggerated knee jerks, but without ankle clonus or Babinski, and with marked stiffness of legs, as the result of reflex action from irritation, especially from the genitals.

nd marked ataxia which develop very gradually and slowly. Some moderate pain and rarely there may be anesthesia and analgesia. The tendon reflexes are exaggerated. Ankle-clonus and Babinski.
d of the disease the reflexes may be abolished, but the Babinski persists. It is a disease of adult life, but is analogous to Friedreich's ataxia (652).

nptoms. The disease is usually of sudden onset and often follows some powerful emotion. It is usually permanent until cured by another strong emotion, which may be often artificially produced by
ffers great resistance to passive motion, even to slow motion. Contractures are common. Knee-jerks are usually increased, but no true ankle-clonus or Babinski. Organic reflexes rarely disturbed, but
explaining all of the symptoms by any one organic lesion. The paralysis is not limited to one muscle, or to the distribution of one nerve. Associated movements do not occur in hysterical paralysis, and

DIAGNOSIS

Cerebral Palsy of Childhood. Porencephaly. Acute encephalitis. Infantile hemiplegia or diplegia (Little's disease). (577, 630, 798, 1048, 1086.) (Figs. 15-16.) 501

Meningeal Apoplexy. Hemorrhage in cerebral meninges. Pachymeningitis Interna Hemorrhagica. Aneurism, etc. (588, 1060.) (Figs. 15-16.) 502

Cerebral Hemorrhage. 503

" 504

Cerebral Embolism or acute softening. 505

Cerebral Apoplexy. (432, 588, 832, 856–7, 1043, 1060–3.) (Figs. 15-7.)

Cerebral Thrombosis or acute softening. 506

Cerebral Tumor, including Cyst. (536–42, 578, 587, 833, 849, 855–61, 892, 908, 960, 1047.) (Figs. 15-17.) 507

Cerebral Abscess or localized Meningitis. (578, 587, 907, 960, 965, 1045–6.) (Figs. 15-17.) 508

Spinal Tumor or unilateral spinal lesion. Brown-Sequard's paralysis. (442, 486, 515, 519, 542, 551, 826, 836–40, 975, 981, 1000.) (Figs. 24-6.) 509

Choreic Paralysis. (622.) 510

Disseminated Sclerosis. (580, 659, 668, 688, 756, 765, 799, 913, 1051.) 511

Injury or hemorrhage in, 512
Acute myelitis or myelomalacia of, (795, 828.) 513
Chronic myelitis or myelomalacia of, 514
Tumor in, 515

Injury or hemorrhage in, 516
Acute myelitis or myelomalacia of, (795, 829.) 517
Chronic myelitis or myelomalacia of, 518
Tumor in, 519

Compression Myelitis. (795.) (Fig. 27.) 520

Caisson disease or Diver's paralysis. 521

Senile Paraplegia. (791a) 522

Spina-bifida. 523

Hemorrhage in spinal membranes. Hematorrhachis. (973.) 524

Spastic Paraplegia or lateral sclerosis (usually symptomatic and part of a more extensive lesion, spinal or cerebral.) (797.) (Figs. 24-7.) 525

Ataxic Paraplegia or postero-lateral sclerosis. (660, 796.) (Figs. 24-6.) 526

Hysterical Paralysis. (747–8, 759, 793, 878, 1074.) 527

CHART X c

Combined and Intermittent Paralysis

Comprising Numbers 471 and 474 on left side of Chart
and 535 to 556 on right margin

474 COMBINED PARALYSIS

- Cranial and spinal nerves involved. (Figs. 18-23, 33, 38.)
  - Sensory symptoms present usually.
    - Bilateral symptoms. — If the patient does not promptly die, one or mor… gia (525). There are usually dysarthria, dyspl… toms at first may be more unilateral.
    - Crossed paralysis (256) and bulbar symptoms (434).
      - Paralysis of one or more eye muscles of one side
      - Paralysis of facial (both upper and lower branch…
      - Paralysis of hypoglossus of one side and of arm
  - No sensory symptoms.
    - Acute. — The onset of paralysis is sudden. If the patient … sive. They are usually unilateral, but may be… paralysed, while there is a spastic paralysis m… May be due to acute inflammation, hemorrha… myelitis. Often due to syphilitic endarteritis o…
    - Chronic—The chronic forms of these diseases, with the spinal form (547), constitute the progressive muscular atrophies and resemble the muscular dystrophies in that the paralysis and atrophy advance together slowly, and it is difficult to say which is primary. They also constitute a group of chronic degenerative atrophies.
      - The paralysis involves the eye muscles. — A g… n… p…
      - The paralysis involves the lips, tongue, pharynx and larynx. — The u… A… t… n…
- Spinal nerves alone involved. (Figs. 24-7, 33, 38.)
  - No sensory symptoms. — Symmetrical paralysis commencing in the small muscles of hands or in shoulder girdle muscles. — The muscles affected show progressive weakness, … or, more rarely, in the muscles of the shoulder … thumb cannot be brought across hand to touc… but not always. There are secondary contract… of muscles is increased. Often associated w… umns are involved or not. It is difficult to di…
  - Marked sensory symptoms are present, such as pain, paresthesiae, anesthesia, etc., with the motor paralysis. — Both arms and legs are paralysed. There are trophic disturbances in the arms and not in the legs. Pupils are often unequal. Reflexes are abolished in the arms and increased in the legs. Babinski and ankle-clonus are present. The bladder is usually more or less distended; its detrusor being paralysed. Contractures may be present in the legs.
    - Very acute onset. Sympto… may yield a bloody fluid…
    - Acute, sub-acute or chroni… litic myelomalacia (1211…
    - Chronic course, intense pa… common. Cerebro-spina…
    - Very chronic onset and a…
  - Dissociation of sensation (365) is present. — Dissociation of sensation is the most characteristic symptom and is co… Trophic lesions are usually prominent. Pemphigus, ulceration and … trophic symptoms predominate over motor symptoms in the arms; … present the symptoms may be both in arms and legs, and the moto… aggerated in central gliosis in the cervical or dorsal regions. The cer… fairly rapid course, and may exhibit a unilateral, spastic, muscular p…

471 INTERMITTENT PARALYSIS

- All the muscles of the body and head. — The characteristic sign of the disease is the rapid tiring of the muscles when in activity. Pati… (401). There is no muscular atrophy and no reaction of degeneration. In the domain of c… usually held retracted on account of the ptosis. The symptoms are slight in the morning …
- Muscles of one or both legs, rarely of arms. — Intermittent attacks of painful muscle cramp, and weakness of leg or legs, caused by walkin… Rarely the disease occurs in one or both arms. No sensory disturbances except painful cram…
- Commencing in legs extending to arms. — Recurrent attacks of paralysis of the muscles of the legs usually first and then of arms, las… nerves are not attacked. There is usually well marked heredity, or the disease occurs in f… but in some groups of family periodic paralysis these negative symptoms are not present.
- Associated with a cervical rib — A cervical rib can be felt and can be seen with the X-ray. In some cases of cervical rib, att… skin which comes on after the arm has been used a short time. and, if use of the arm is co… be caused by a cervical rib, and then is often relief by elevation of the arm and is made w…

DIAGNOSTIC ANALYSIS OF SYMPTOMS

ABSTRACT OF SYMPTOMS

		DIAGNOSIS	
al nerves are paralysed. There is more or less marked spastic paraple- taxia, anesthesia and often vertigo. (Bulbar symptoms—434.) Symp-	Acute onset. Regressive course. No optic neuritis. A variety of apoplexy (504). No increased tension of cerebro-spinal fluid.	Hemorrhage, softening or acute inflammation in brain-stem (543-4, 656).	535
	Chronic onset. Progressive course. Optic neuritis. May be increased tension of cerebro-spinal fluid.	Tumor in, or compressing the brain-stem (656).	536
rm and leg of opposite side.	Acute onset and regressive course. No optic neuritis. No increased tension of cerebro-spinal fluid.	Hemorrhage or softening in crus cerebri (543).	537
	Chronic onset and progressive course and optic neuritis. May be increased tension of cerebro-spinal fluid.	Tumor in, or compressing crus cerebri (656).	538
rigeminal nerve on one side and of arm and leg of opposite side.	Acute onset and regressive course. No optic neuritis. No increased tension of cerebro-spinal fluid.	Hemorrhage or softening in pons (543).	539
	Chronic onset and progressive course and optic neuritis. May be increased tension of cerebro-spinal fluid.	Tumor in, or compressing pons (656).	540
of opposite side.	Acute onset and regressive course. No optic neuritis. No increased tension of cerebro-spinal fluid.	Hemorrhage or softening in medulla (544).	541
	Chronic onset and progressive course and optic neuritis. May be increased tension of cerebro-spinal fluid.	Tumor in, or compressing medulla (656).	542
ot die promptly, later the symptoms are regressive rather than progres- ral. A number of cranial nerves, either motor or sensory, or both, are ess pronounced in the arms and legs. Vertigo is a common symptom. embosis, embolism, or compression. May occur in acute anterior polio- itic neuritis.	There is more or less extensive paralysis of the motor nerves of eyeball (3rd, 4th and 6th); so that ptosis, squint, double vision, nystagmus, etc., may result.	Acute or Apoplectiform Polioencephalitis Superior (495, 535, 1064).	543
	There is paralysis of the lips, tongue, pharynx and larynx (7th, 9th, 10th, 11th and 12th nerves), with consequent dysar- thria and dysphagia and usually ataxia and respiratory disturbances.	Acute or Apoplectiform Polioencephalitis Inferior. Acute Bul- bar paralysis (495, 535, 1064).	544
onset of weakness of ocular muscles. It may be steadily progressive or, having progressed to a certain point, it may remain stationary. Muscles may be attacked in any order, ptosis, squint, im- yeballs, immobile pupils. The disease may attack only the external muscles of the eyeball (ophthalmoplegia externa), or only the internal muscles (ophthalmoplegia interna), or both, (ophthalmo- mpleta). Disease may be complicated by bulbar paralysis (546) and is usually associated with amyotrophic lateral sclerosis.		Progressive Ophthalmoplegia. Polioencephalitis Superior Chronica (often symptomatic of a steadily progressive, more widespread disease, such as tabes, tumor, etc.).	545
s bent forward. There are bulbar symptoms (434). There are drooling of saliva, dysarthria, dysphagia, and aphonia. Paralysis, tremor, atrophy, fibrillary contraction of muscle of tongue, lips, harynx, larynx, etc. Both facial nerves are involved in some cases. The paralysis very slowly progresses. There are symptoms of a mild spastic paraplegia in legs with ankle-clonus and Babinski. e of advanced life. Often associated with amyotrophic lateral sclerosis and at times with progressive ophthalmoplegia (545). In addition to the pseudo-bulbar paralysis of myasthenia gravis (553) nother form due to lesions in both cerebral hemispheres in which there is no muscle atrophy, or fibrillation, and no change in the electrical reaction, but all the other symptoms of bulbar paralysis less marked. There is more mental impairment and greater emotional excitability than in true bulbar paralysis.		Progressive Bulbar Paralysis. Polioencephalitis Inferior Chronica. Labio-glosso-pharyngeal Paralysis, (694. 761, 1150).	546
y, fibrillary contractions and all degrees of alteration in electrical excitability from simple diminution to complete reaction of degeneration. The process commences in the small muscles of the hands, scapulo-humeral type), is usually fairly symmetrical and extends to the other groups of muscles in arms, body and even legs. The muscular weakness follows and is dependent upon the atrophy. The ittle finger The fingers cannot be spread apart, nor can their last two phalanges be extended on the first. The legs show a mild degree of spastic paraplegia, with ankle-clonus and often Babinski, specially the "claw hand." The head is usually bent forward and there is much deformity about shoulder and other parts. A disease of adult life, and of very chronic course. Mechanical irritability ogressive bulbar paralysis (546). Some authors divide this symptom complex into two groups according as to whether the atrophy or the paralysis is primary, and as to whether the lateral col- such distinction clinically. The one form may be an earlier stage of the other.		Amyotrophic Lateral Sclerosis. Progressive Spinal Muscular Atrophy. Aran-Duchenne type of muscular atrophy. Chronic Atrophic Paralysis, (695, 797, 1149).	547
most extensive in first few days and may slowly improve later. May be deformity in cervical region of spine. Knee-jerks may be absent in early stages. Lumbar puncture	Cervical enlargement of spinal cord. (Figs. 24-6.)	Injury of, or hemorrhage in,	548
. Symptoms continue to extend for some time and are fairly symmetrical. Organic reflexes disordered. A history or other evidence of syphilis (1205) is often found in syphi-		Acute or chronic myelitis or myelomalacia of, (795, 835, 1310).	549
spasms in arms and neck precede the paralysis and contractures and muscular atrophy in hands, "claw hand." Cutaneous eruptions (herpes, pemphigus, etc.) are not un- may be under increased tension and show lymphocytosis. Most of these cases are the result of chronic syphilitic meningitis.		Pachymeningitis hypertrophica cervicalis.	550
ssive course. Symptoms at first mainly unilateral, becoming bilateral later. Cerebro-spinal fluid may show increased tension.		Tumor in, or compressing, (836)	551
with pain and paresthesiae and more or less motor paralysis and atrophy. The muscular atrophy has often the location and characteristics of that of progressive spinal muscular atrophy (547). ion of the hands and trophic lesions of bones, muscles, and other tissues occur in the type called Morvan's disease. In the most common form the cervical region is alone affected and sensory and he legs show a mild spastic paraplegia (525), and scoliosis or kyphosis occur in more than half the cases. Paradoxical (379) and spontaneous sensations have been noted. When a diffuse glioma is otoms are about as prominent as the sensory and may be unilateral in the early stages.. All forms of reflex action are abolished when the cervical and lumbar enlargements are involved, but are ex- rm of the disease runs a chronic course, extending at times over decades, but slowly progresses and the anesthesia at first slight steadily becomes more marked. The diffuse form often runs a s, at least in the early stages.		Syringomyelia. Central gliosis. Morvan's Disease, (693, 837-9, 1009, 1150a, 1170, 1187, 1357, 1359).	552
n walk well at the start, but after a few (or a few hundred) steps is tired out. The same is true of all other voluntary acts. Examination of the muscles with electricity gives the myasthenic reaction nerves (in which the case usually commences) there may be ptosis, diplopia, ophthalmoplegia, diplegia facialis, dysarthria, dysmasesia, etc., and all the spinal nerves may be affected. The head is w worse during the day. No sensory disturbances except painful cramps. Organic reflexes normal.		Myasthenia gravis. Pseudo-bulbar Paralysis.	553
ng the attack the feet are cold, and there is diminished or absent pulsation in arteries of feet, associated with marked arterio-sclerosis of arteries of leg as shown by palpation and by the X-ray. rganic reflexes normal. Angio-spastic hemiplegia in which temporary attacks of hemiplegia, sometimes associated with aphasia, occur, is probably a variety of this disease.		Intermittent Limping or Claudication. Dysbasia Angio- sclerotica, (1199).	554
few hours or days. The attacks usually occur in the morning or after rest. During the attack the left cardiac ventricle may become temporarily dilated and a murmur may be heard. The cranial groups. During a severe attack there is often a diminution or absence of the reflexes and of the faradic and galvanic excitability of the nerves, and of the mechanical excitability of the muscles, of these cases are apparently due to malaria and can be cured by the administration of quinine.		Family Periodic Paralysis.	555
cur, but only after use of the arm. These attacks are usually unilateral, even though the extra rib is on both sides. The attack consists of numbness, tingling, feeling of congestion, redness of the d, the arm shows a decided paresis, which passes off if the arm is kept at rest. An ununited fracture of the clavicle will rarely cause similar symptoms. Pain, in the form of a brachial neuralgia, may y motion. In rare cases this paralysis, at first intermittent, may become permanent and may be associated with atrophy of the muscles of the hand and even of the forearm.		Pressure of cervical rib upon sub-clavian artery.	556

CHART XI

Convulsion or Spasm

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptom Analyzed	Tests: Character	Tests: Extent	
570 CONVULSION OR SPASM (242)	571 CLONIC mainly (246)	GENERAL CONVULSION	Diseases in which convulsions occur are set forth in Chart XI a.
		LOCAL CLONIC SPASM	Diseases in which local clonic and all forms of tonic spasm occur are set forth in Chart XI b.
	572 TONIC mainly (245)	GENERAL TONIC SPASM	
		LOCAL TONIC SPASM	
	573 CHOREIFORM (272)		Diseases in which choreiform and athetoid spasm occur are set forth in Chart XI c.
	574 ATHETOID (271)		

CHART XI a

General Clonic Convulsion

Comprising Numbers 571 on left side of Chart
and 575 to 596 on right margin

571 GENERAL CLONIC CONVULSION

- **Apyrexia.**
  - The convulsion commences in all the muscles at about the same time (epileptiform convulsion).
    - Loss of consciousness, (coma or semi-coma) (205). Frequently biting of the tongue or other injury. Short duration.
      - No other symptom of disease except the convulsion.
        - Repeated attacks. — The conv… followe… tion of … tile, au… tendon … spots a… pressur… spasm … around … attack … Such p… observa…
        - One attack or one series of attacks. — A convul… and nc… periphe…
      - Symptoms of serious brain disease.
        - Congenital or in infancy. Often fever at onset of first convulsion. — The disea… rarely … are rar…
        - In youth or more often in adults. — Between … are foll… of the … sympto… disease … psychic… vance t…
      - Symptoms of cerebro-spinal disease. — Intention…
      - Symptoms of disease of other organs than the brain
        - Kidney disease. — There ar… and th…
        - Cardiac disease. — Slow pul…
        - Blood disease. — Rapid fe… culatio…
      - Symptoms of poisoning.
        - Blue line on gums, lead in urin…
        - Alcoholic odor of breath and g…
    - Apparent, but no true, coma (shown by susceptibility to suggestion). No biting of tongue or other injury. Long duration. — Symptoms of hysteria (425). Such attacks have been called hystero-epilepsy. — The attac… very ir… usually … orbital … toms (…
  - The convulsion always commences in one group of muscles and later extends over the whole or part of one side of the body and often over both sides. Jacksonian epilepsy (431). — If the convulsion remains unilateral, consciousness may or may not be lost, usually not, but it is always lost when the convulsion becomes bilateral. — There a… present … of serious … ease, … always.
- **Hyperpyrexia.** — Epileptiform convulsion. — Coma during and after the convulsion. — Occurs s…
- **Pyrexia.** See also 577.
  - Epileptiform convulsion. Coma during and usually after the convulsion. — Headache, backache and radiating pains, delirium, vertigo and vomiting, especially on change of posture, hyperalgesia (spinal and elsewhere), photophobia, etc., are early symptoms. Retraction of head, opisthotonus, etc. (265). Paralysis of cranial nerves (squint, etc.), cutaneous eruptions (herpes), tâches cérébrales and Kernig's symptom (319). Tonic spasm and paralysis are more common in basic inflammations, and clonic spasm in cortical inflammations.
    - Lumbar puncture gives a clear … bloody or purulent, fluid, unde… pressure containing globulin … polymorphonuclear leucocytes…
    - Lumbar puncture gives a clear f… increased tension and contai… ulin and many mononuclear … cytes, and if the disease … polymorphonuclear leucocytes…
    - Lumbar puncture gives a clear f… increased tension, but no i… cellular elements.
  - Coma during the convulsion.
    - May occur in children at the onset of any infectious disease, especially in …
    - Is the result of some unusual metabolic changes within the body, and espe…

DIAGNOSTIC ANALYSIS OF SYMPTOMS

ABSTRACT OF SYMPTOMS

r the major attack (le grand mal), is at first tonic with arrest of respiration. The face, at first pale, soon becomes flushed and cyanotic. The pupil is dilated and inactive. This tonic state is quickly
minute) by clonic spasm of longer duration (4 to 5 minutes) with noisy respiration, and froth on lips often bloody from the tongue, which is often bitten during the attack. There is often lateral devia-
and eyeballs and nystagmus. At times urine and feces are passed during the attack. The attack is often preceded by twitching of certain muscles, "motor aura," or by a sensory hallucination (tac-
visual, olfactory, etc.) called the "sensory aura" (430) and is often ushered in by a cry, "the epileptic cry." In some attacks the temperature of the body is raised and albuminuria is frequent. The
are occasionally absent during and immediately after the attack. A deep sleep and some muscular and sensory weakness (stage of exhaustion) often follow the attack. After a violent attack purpuric
onally seen on the forehead and face. Complete amnesia in regard to it always follow an attack. There are recurrences at varying intervals, but attacks can neither be brought on nor arrested by
ny part of the body. In some cases the attacks occur only at night or while asleep by day (nocturnal epilepsy). The attacks vary greatly in character. At times there is merely rigidity without clonic
a media). At times there is merely loss of consciousness without action or with some automatic act: the minor attack (le petit mal). At times the patient is unconscious and runs forwards and turns
und (epilepsia procursiva rotatoria) or performs a series of very complicated acts (automatism). At times the patient is unconscious and performs an act of violence (epileptic mania). At times an
ty follows or replaces a convulsive attack (psychic equivalent). In old cases of epilepsy and in those in which the attacks are very frequent there is more or less mental impairment (epileptic dementia).
re usually dull and stupid, often destructive and apparently vicious. Malingerers have been known to imitate an epileptic convulsion closely. The diagnosis of malingering can only be made by long
the purposeful and timely nature of the attack by sweating and other evidences of conscious effort and by the confession of the malingerer

ck, especially common in syphilitic and rachitic children, but occurring also in adults, especially in pregnancy, altogether similar (epileptiform) to the above, but occurring only once, or in one series,
ed. Associated often with digestive disturbances and abdominal distension and may be due to poisoning (ptomaine, alcohol, etc.), or to the status lymphaticus. May be due in some children to
tion and at the onset of an acute infection. No sharp line can be drawn between these convulsions and those of uremia (581), or lead (584), or alcohol (585), or auto-intoxication (596).

genital or begins in early infancy. Unilateral or bilateral convulsions occur, associated with monoplegia, hemiplegia or diplegia, with considerable mental impairment. There is usually dysarthria,
often post-hemiplegic motor disturbances: athetosis, rigidity and contractures. Frequently there is more or less complete arrest of development, physical and mental. The shape and size of the skull
al. Cases of epileptic idiocy may belong to this class, even though they present no paralysis.

vulsive attacks, which
a temporary weakness
s involved, there are
steadily-progressing brain
ially motor, sensory and
oms, and a steady ad-
coma or dementia.

Headache, vertigo and vomiting. Choked disc or optic neuritis. More or less motor and sensory paralysis. Mental inertia (apathy) and increasing mental weakness and loss of memory. Localizing symptoms are sometimes present. Convulsions are often local.

Loss of will power, restlessness, delusions (usually of exaltation) and symptoms of insanity. Poor judgment. Good natured but irascible. Childish. Characteristic blurred speech. Tremor of lower facial muscles, lips, tongue and hands, and awkwardness (ataxia and apraxia). Unequal and irregular pupils, Argyll-Robertson's pupil, optic neuritis or atrophy. History of syphilis. Lumbar puncture shows lymphocytosis in cerebro-spinal fluid, globulin and a positive Wassermann reaction (419-20).

, scanning speech, nystagmus, unsteady gait, motor and sensory paralyses, many symptoms of local lesion, etc.

a series of convulsions, accelerated respiration, restlessness, cyanosis and contracted pupils. Often edema of lungs and of other tissues. The arterial tension is usually high, the heart hypertrophied,
contains albumen and many casts and is usually scanty. There may be albuminuric retinitis, headache, vomiting, Cheyne-Stokes respiration and somnolence.

g intermission in heart beats associated with coma and convulsions, which pass off after the heart begins beating again. Arteries atheromatous. Advanced life usually.

e of small volume. Insufficient amount of blood or of red cells and hemoglobin. Pallor, dyspnoea on exertion and strabismus are common. Often severe digestive disorders with congested portal cir-
esent. Often the result of one large or of repeated hemorrhages.

aking K.I. Wrist-drop. History of lead colic and of exposure to lead.

emor and nervousness. Muscular tenderness. History of alcoholic abuse.

ays in the presence of an audience. There is always a warning in the form of globus hystericus, palpitation, etc. Patient may fall or glide to the ground but does not hurt herself. The convulsion is
and violent and many of the movements seem purposeful and to be theatrical posing (crucifixion, etc.) and assumed attitudes, (attitudes passionelles). Patients often "rave" during the attack, which
long time, especially if the audience be excited. Eyes are usually closed and the eyeballs turn upwards if eyelids are forced open. Attack can usually be arrested by pressure on ovarian region, supra-
etc., especially if the audience be sent from the room. Moderate pressure upon these parts may cause an attack. Great variety of sensory symptoms. Anesthesia is usually present. Hysterical symp-
bundant limpid urine after the attack, but urine and feces are never passed involuntarily during an attack. The reflexes are not abolished during the attack as may occur in epilepsy (1058).

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The convulsion often commences with a motor (twitching), rarely with a sensory (tingling), aura. It spreads first through the whole of the part first attacked and then in a definite order from face to arm, or from arm to leg or face, or from leg to arm, according to the anatomical arrangement of the cortical motor centers; hence never from leg to face without the arm being involved. The convulsion is followed by a hemiplegia, sometimes transient, sometimes permanent and progressive. There may be muscular rigidity in the intervals between the convulsive attacks.

Occurs in youth or middle age and often after traumatism of old date. Headache, vertigo and vomiting. Usually choked disc or optic neuritis. Symptoms steadily progressive. Similar attacks without choked disc may occur in localized meningitis or other irritative lesions in the cerebral cortex.

Occurs in old age and often after recent injury. Alcoholism, atheromatous arteries and insanity are common. Sudden apoplectic attack with improvement later. No choked disc. Symptoms progress paroxysmally, and after each paroxysm there is marked improvement

r, after some prodromal symptoms such as headache, poor eyesight, etc., in persons who have been exposed to high temperatures. Delirium is usually present.

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By lumbar puncture, Weichselbaum's diplococcus almost always, or rarely the pneumococcus or other germs, may be found in the cerebro-spinal fluid. There is often an epidemic of the disease (Weichselbaum's diplococcus). Strong retraction of head.

By lumbar puncture, pyogenic bacteria may be found in the cerebro-spinal fluid. Suppuration or an infected wound may be present, especially in the head. Suppuration in middle ear or mastoid, or in nasal sinuses, carbuncle or erysipelas of head or neck. Retraction of head may be less marked.

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By lumbar puncture tubercle bacilli may be found in the cerebro-spinal fluid. A tuberculous process may be found in some other part of the body. Grinding of teeth and hydrocephalic cry. Choked disc and choroid tubercles may occasionally be seen by ophthalmoscopic examination. Tuberculin skin test will be positive.

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By lumbar puncture no bacteria can be found in cerebro-spinal fluid. Many, if not all, of the general symptoms of meningitis may be present, but they are not so severe and are not so constant as in the other forms and are promptly relieved by the withdrawal of a moderate amount of the cerebro-spinal fluid.

ecting the nervous system.

of intestinal putrefaction and other abnormal processes, often started by the ingestion of tainted meat and other poisons. Vomiting, tympanites, diarrhoea and foul smelling feces.

DIAGNOSIS

Idiopathic Epilepsy (including the major attack, the minor attack, epilepsia media, nocturnal epilepsy, epileptic automatism, epileptic mania, psychic equivalent, epileptic dementia), (110, 126, 430, 846, 1027, 1058, 1071, 1083, 1102). 575

Eclampsia, (1059). 576

Cerebral Palsy of Childhood. Porencephaly. (501, 630, 798, 1048, 1086). 577

Cerebral Tumor (including abscess and cyst) not in or near motor area of cortex (507-8, 587). 578

Paresis. General Paresis. Paralytic Dementia, (134, 177, 416, 419-20, 675, 763, 895, 1049, 1104, 1216, 1230). 579

Disseminated Sclerosis, (511, 659, 668, 688, 756, 765, 799, 913, 1051). 580

Uremic convulsion, (576, 850, 956). 581

Stokes-Adams' Disease, (436, 1057). 582

Anemic convulsion. 583

Lead convulsion (404, 576, 1050). 584

Alcoholic convulsion, (576, 658, 663, 764). 585

Hysterical convulsion, (1074). 586

Cerebral Tumor (including abscess and cyst) in or near motor area of cortex. Jacksonian Epilepsy. (431, 507-8, 605.) (Figs. 15-16.) 587

Hemorrhage in or near motor area of cortex (pachymeningitis, etc.). Jacksonian Epilepsy. (502, 1060.) (Figs. 15-16.) 588

Sunstroke or Heatstroke, (966, 1068). 589

MENINGITIS

Cerebro-spinal Meningitis, (608, 1214, 1226). 590 591

Purulent Meningitis, (1227). 592

Tuberculous Meningitis (1228-9). 593

Serous Meningitis. Meningismus. (1239). 594 (608, 1045)

Febrile or toxic convulsion 595

Auto-toxic convulsion, (576, 1067). 596

CHART XI b

Clonic or Tonic Spasm

Comprising Numbers 572 on left side of Chart
and 600 to 621 on right margin

(Note)—Many of the spasms, especially the tonic spasms, are associated with pain, and are then called "cramps."

571 CLONIC SPASM

LOCAL

Pyrexia in very acute cases. — Shock-like spasms similar to that produced by an electric shock. — Begins in one arm and side ... side and then to opposite ...

LOCAL CLONIC SPASM

Apyrexia

A single or many times repeated spasm, rarely contracture, of one muscle or of a group of muscles, occurring in paroxysms which rather tend to subside on voluntary movements. Myoclonus. (270.)

- Occurs in face and more rarely in neck and arms. — The ... and ...
- Begins in arms and may extend to legs, but almost never to face. Often the tendons play as in subsultus tendinum. — The ... me... are... are... mu...
- Begins in side of face or in one arm or leg and may extend over one, or even both sides of body. — The ... fir...

572 TONIC SPASM

GENERAL TONIC SPASM

Pyrexia

- Spasm commencing in jaws. — There is the history of an infected wound, or septi... of jaws, occurring in paroxysms; also rigidity o... body being held in position of opisthotonus, em... becomes very high. The disease varies greatly ...
- Spasm commencing in pharynx and oesophagus. — There is history of a bite by an animal (usually ... especially on sight of water. Spasmodic closur... cough, opisthotonus and general spasm are cor... light and accommodation. The stage of excite... diagnosis must be made in such cases by the p...
- Spasm commences in back of neck and in back. — There may be more or less ... symptom. Lumbar pun...

Apyrexia. If unconsciousness is present. See also epilepsia media (575).

- Cerebellar ataxia is present (281). — A tonic spasm of sudden onset, the face not being affected. — The ... de... Retr...
- Rigidity rather than spasm, not strong enough to prevent passive or voluntary movements (266). — Extremities and trunk rem... attack may last minutes ... Rigidity of all muscles, ma...
- Spasm only at commencement of any action. — Spasm passes away as the action is continued, to ... the muscles of the face usually escape altogeth... fibers show marked hypertrophy. Closely allie... low exposure to cold with consequent reflex v... The so-called acquired form, " myotonia acqui...
- Spasm mainly confined to hands and feet, paroxysmal. — Bilateral painful tonic spasm of muscles of hands ... tended. Increased mechanical (Trousseau's ph... ciated with rickets or digestive disorders and i... It occurs in infectious diseases, in poisoning a...
- General painful clonic, followed by tonic, spasm. — Spasm very general and v... nine poisoning. Death u...
- General permanent contracture. — Paralysis is coincident wit...

LOCAL TONIC SPASM

Apyrexia.

- Spasm only occurs when performing some accustomed act. — Occurs usually in small muscles and in those tha... gradual onset, steadily grows worse, and render... rather than spasm. Atrophy of the muscles i... the voice of singers, public speakers, etc.
- Rather brief spasm of one or more muscles. — A spasm lasting minutes c... cough, oesophageal spas...
- More permanent spasm. — A contracture of a few or many muscles usually ... efforts are made to overcome it. No muscle a... bling (674) or may consist in jumping or ski...
- A permanent or, at least, a long continued, contracture. The muscles are anatomically shortened, in later stages.
  - A hemiplegic contracture. — Tendon reflexes are increa... and lasts for years. Us...
  - A paraplegic contracture. — Tendon reflexes are increa... follows an attack of par...
  - A local contracture. — Absence of reflexes. Muc... traction of tendon and f...

ABSTRACTS OF SYMPTOMS

				DIAGNOSIS	
ck and extends to leg of same	Much pain in head and neck at onset. Weakness slowly follows the spasms. At times there is wasting of muscles and loss of faradic excitability. The spasms become violent and more continuous. Epileptiform attacks are common. Death results in a few months.			Dubini's Disease or Electrical Chorea. (627).	600
s are almost always unilateral, n bilateral are not symmetrical.	The spasms seem like mimic gestures and appear to be rather purposeful. May have originated from local irritation, but have persisted after the irritation has ceased.	Psychic disorders of an emotional or neurasthenic nature often present.	The contractures are accompanied by no pain. See also reflex spasm (617).	Convulsive Tic (blepharospasm, torticollis, etc.), (267, 270, 617, 726).	601
			The contractures are accompanied by sharp darts of pain.	Tic Douloureux, (267, 726, 947).	602
s are bilateral and fairly sym- but not synchronous. They ular in force and rhythm, and ost always limited to one	The spasms never appear to be purposeful. No movement results, merely individual muscles spring forth in strong contraction. Irritation of the skin or tendons causes paroxysms.	Little or no emotional disturbances. Trembling of muscles between the paroxysms. The spasms become less, or entirely cease, during sleep. Reflexes exaggerated.	The muscles involved are attached by one end to the trunk of the body. Muscles of the face, hand and forearm, foot and lower legs almost never involved. No heredity. A disease of adult life.	Friedreich's Paramyoclonus Multiplex.	603
			Many cases occur in the same generation of a family. The disease begins in early life and is associated with epilepsy and dementia.	Unverricht's Family Myoclonus Epilepticus.	604
ns are always unilateral at	The spasm may extend to adjacent muscles and so over one half the body and then pass across and involve both sides, or it may remain a local spasm and pass away in a short time. After many such local spasms, one may occur which will pass into a general convulsion. A general convulsion can sometimes be averted by tying a band tightly around the extremity as soon as the local spasm appears. Consciousness is always lost when the convulsion extends to both sides of the body; but usually persists when spasm is limited to one-half the body or to one extremity.			Jacksonian Epilepsy, (431, 587, 1282-3, 1291).	605
lbirth, within a month, usually within two weeks. The infection may occur through the navel in new born babies (tetanus neonatorum). The characteristic symptom is rigid spasmodic closure l muscles, producing "risus sardonicus." In the onset a gradually increasing stiffness of masticatory and other muscles followed and accompanied by paroxysms of painful tonic spasms; the botonus, pleurosthotonus or orthotonus (265). The spasms are associated with profuse sweating. There are no mental symptoms and no coma. Towards the fatal termination the temperature xiety. The longer the incubation period the milder the disease. Local tetanus and head tetanus with local paralysis have been described.				Tetanus, (170). Tetanus traumaticus. Tetanus rheumaticus. Tetanus puerperalis. Tetanus neonatorum.	606
r cat) within a year, usually within six months. The most striking features are tremor, rapid pulse, fever, mental depression, fright, horror and extraordinary emotional excitement, even mania, harynx and œsophagus, making swallowing, especially of fluids, impossible. Saliva cannot be swallowed and is expelled from the mouth with difficulty. Spasm of muscles of respiration, hic- Reflex acts from cutaneous or special sensory surfaces are greatly increased, especially that of inspiratory dyspnœa; and priapism occasionally occurs. The pupils are dilated and respond to is at times followed by a stage of paralysis and is often preceded by a prodromal stage of malaise and of pains, especially in the scar. Hysterical persons at times simulate hydrophobia. The e of hysterical symptoms (425), absence of fever and by time.				Hydrophobia. Lyssa. Rabies, (171).	607
rbance, or loss, of consciousness. Tonic retraction of neck, opisthotonus and boat-shaped retraction of abdomen. Slight irritation will cause spasm. Headache, backache, delirium, Kernig's shows increase of cells in cerebro-spinal fluid, except in serous meningitis. For different varieties see 590-4.				Meningitis, Cerebral and Spinal, (508, 590, 831, 974, 1005, 1032, 1045, 1208-9-13).	608
nities on the same side as the lesion are adducted, on the opposite side abducted. Head, trunk and extremities each rotate about long axis fro side of lesion to the opposite side and the eyes towards the same direction.				Lesion of cerebellar hemispheres, (648, 686, 783, 1016, 1272).	609
of the head and opisthotonus, flexion of elbows, supination of hands, extension of legs with pointing of toes.				Lesion of vermis of cerebellum, (648, 686, 783, 1016, 1272).	610
any position in which they may be placed for a surprisingly long time. Wax-like resistance to passive motion. Difficult positions maintained indefinitely without apparent effort. The s or days. Anesthesia, abolition of reflexes, and apparently more or less complete loss of consciousness are usual symptoms. Other hysterical symptoms are often present, (425).				Catalepsy, (1096-8).	611
e face, speech monotonous, passive tremor of hands and legs, characteristic attitude, festinating gait. Tendency to fall backwards or forwards (677).				Paralysis Agitans. Parkinson's disease, (677, 766, 800).	612
n on first movement after a rest or when action is done faster. Patient cannot hurry or execute rapid movements. Is liable to lose equilibrium. The arms are less affected than the legs and Marked heredity. Myotonic electrical reaction (402). Increased mechanical excitability of muscle, even slight pressure with the finger-tip causes a sluggish long continued contraction. Muscle This disease is "paramyotonia congenita" (Eulenberg's disease) also on an hereditary basis (it has attacked twenty-eight members of a family in five generations), but these paroxysmal attacks fol- otor spasm and nutritive disturbances in the muscles. A myotonia congenita intermittens and a myotonia congenita atrophica have been described with the characteristics implied in their names. is probably an altogether different disease.				Myotonia Congenita. Thomsen's disease, (265, 1155), including Paramyotonia Congenita (Eulenberg's disease).	613
ometimes of feet, lasting minutes, hours, or rarely days. Hands and feet drawn into smallest volume possible with hollow deepened (obstetric) hand). Joints of arms flexed, those of legs ex- enson (450)) and electrical (Erb's sign (452)) excitability of nerves. Facial nerve very irritable; so that slight blows on it cause spasm of facial muscles (Chvostek's sign (451)). Usually asso- al parasites, or dilated stomach; often follows extirpation of parathyroid glands. Sometimes occurs as a symptom of hydrocephalus, cerebral tumor or other serious brain disease in children. pregnancy and as an occupation neurosis (616). In some cases this disease may be due to destruction of the parathyroid glands and may perhaps be cured by the administration of these glands. results in a couple of hours. contracture. Convulsions, mental defect and partial arrest of growth, are common. The contracture and motor paralysis may be unilateral or bilateral.				Tetany, (120, 616).	614
inful, mainly in the form of opisthotonus (265). Periods of intermission with relaxed muscles lasting several minutes. Cutaneous and tendon reflexes increased. History or evidence of strych-				Strychnine convulsions, (314-7, 366).	615
e been overworked or improperly worked in doing the same act many times. The spasm is often painful, and in some cases pain may be the only symptom (neuralgic form). The spasm is of accustomed act difficult or impossible. It occurs only when the muscles are used. In some cases there is tremor, in others incoordination and in rare cases, paralysis or paresis (paralytic form) ed is common. Patients are usually neurasthenic. Many varieties: writer's, telegrapher's, pianist's, violinist's, seamstress', shoemaker's, etc., cramp. A similar neurosis occasionally affects				Cerebral palsy of childhood, (116, 501, 577, 630, 798, 1048).	615a
				Occupation Neuroses, (143, 614).	616
rs, due to local irritation in neurasthenic patients. Functional in its nature. Many varieties. Blepharospasm, torticollis, spasmodic croup, laryngismus stridulus, bronchial asthma, whooping rethral spasmodic stricture, vesical spasm, tenesmus, etc. See also the convulsive tics (601).				Reflex spasm, (601, 637, 1194).	617
iated with anesthesia of the part. Usually there is a combination of spasm of flexors and extensors, such as is not seen in organic disease. A functional spasm, which becomes greater the more y. At times may be cured by ovarian pressure or by faradization. Other hysterical symptoms (425). Hysterical spasms are not always in the form of contracture. They may consist in treen- (saltatory spasm).				Hysterical contracture, (1074).	618
hen the spasm does not prevent their occurrence. Little or no muscular atrophy. The spasm is limited to the arm and leg of the same side as follows an attack of apoplexy, by a few weeks there are flexion of elbow, wrist and fingers, and extension of knee. Is associated with a sclerosis of the pyramidal tract and indicates a hopeless prognosis as to recovery from the hemiplegia.				Post-hemiplegic contracture, (501, 504, 577, 615a).	619
hen the spasm does not prevent their occurrence. Little or no muscular atrophy. The spasm, which is often not so continuous as in the hemiplegic form, consists in flexion of both knees, and a. It is of very bad prognosis.				Post-paraplegic contracture (512-20, 548-51, 795).	620
scular atrophy. Is limited to the distribution of one or more nerves. Follows disease of nucleo-peripheral motor neurons. Occurs in fingers. Dupuytren's contracture seems to be due to con- rather than of muscle.				Post-neuritic contracture. Dupuytren's contracture.	621

CHART XI c

Choreiform and Athetoid Spasms

Comprising Numbers 573 and 574 on left side of Chart
and 622 to 631 on right margin

CHART XII

Perversion of Motion and Local Palsies and Spasms

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptom Analysed	Character	
635 PERVERSIONS OF MOTION (243)	638 ATAXIA (248)	The diseases in which ataxia occurs are set forth in Chart XII a.
	639 TREMOR (250)	The diseases in which tremor, nystagmus, or fibrillation occurs are set forth in Chart XII b.
	640 NYSTAGMUS (291)	
	641 FIBRILLARY CONTRACTION OR FIBRILLATION (292)	

LOCAL PALSIES AND LOCAL SPASMS

636 LOCAL PALSIES	See Chart XII c.
637 LOCAL SPASMS	See Chart XII d.

CHART XII a

Ataxia

Comprising Numbers 638 and 642 to 644 on left side of Chart
and 648 to 664 on right margin

63 ATAXIA (248)

642 Ataxia mainly upon standing or walking. Staggering gait. Static ataxia. Cerebellar ataxia. (281).

No loss of muscle sense. No motor paralysis, except in late stage of 651-2.

- Occurs at any age, usually in adults. Usually sensory symptoms
  - Sight and hearing normal. — Patient ex… nystagm… Hypoto… very m…
  - Sight or hearing abnormal. — Staggering … mally. … at time… normal.
- Occurs in youth. No sensory symptoms. — Occurs in family groups and sh… though less pronounced. A ge… mus is common and speech of…

643 Inability to stand or walk. More or less complete.

- Bilateral. — Many sensory symptoms. — Evidently functional. — Legs can … (230) r…
- Unilateral. (Hemiataxia.) — Often analgesia and thermic anesthesia. — Evidently organic. — No loss of … lying do…

644 Ataxia of all movement. Dynamic ataxia. Motor ataxia (280).

- Unilateral. (Hemiataxia.) — Loss of muscle sense and sensory symptoms usually prominent. Knee-jerk usually increased. — When of acute course the condi… ally follows an apoplectic atta… arterial disease is present. … chronic course choked disc … present.
- Bilateral
  - Knee-jerks normal. — No ankle-clonus. — History o…
  - Exaggerated knee-jerks, ankle-clonus and Babinski.
    - Great variety of local symptom… are intention tremor, scanning … ness. Rarely the disease run… essential point is the presence …
    - A combination of symptoms of … usually lost before any anesth… Knee-jerks may be abolished …
  - Knee-jerks and ankle-clonus absent. No Babinski. Often loss of muscle sense and retardation of conduction of pain.
    - Rarely any permanent motor p… ball) are not uncommon early … held well apart and feet are fl… symptom (448), Argyll-Robert… sensations and paresthesiae a… gesia in patches and in cuiras… in cerebro-spinal fluid. The … Positive Wassermann also us… mainly affected. In the ordi… and the diagnosis must rest m…
    - Slight motor paralysis is presen… normal. Cranial nerves rarel… never so chronic as tabes. N…
- Irregular distribution. — Knee-jerks usually exaggerated, but no Babinski or ankle-clonus. Evidently functional (pseudo-ataxia).
  - History of …
  - Emotional …

ABSTRACT OF SYMPTOMS	DIAGNOSIS	
s the staggering, irregular gait of a drunken man. Little ataxia of movements of hands, or of legs when lying down. Vertigo, vomiting and headache are often present. Choked disc or may be present (tumors). Knee-jerks may be present or absent (usually present). Cerebellar fits (609-10) may occur. Symptoms may be bilateral or unilateral (same side as lesion). present, also a diadochokinesia (36) due to a long continuance of muscular contraction. Patients can lie on back with legs flexed at hips and knees much longer than a normal person and onger than patients with dynamic ataxia.	Lesion of cerebellum or its tracts; if acute in onset, apoplexy; if chronic, tumor, (609-10, 654, 686, 783, 1016, 1272).	648
t, constantly or paroxysmal; headache, vertigo and vomiting; choked disc. Knee-jerks — Diplopia or other disorders of sight. Vertigo ceases when eyes are closed.	Ocular ataxia or vertigo, (1020).	649
t, constantly or paroxysmal; headache, vertigo and vomiting; choked disc. Knee-jerks — Deafness and ringing in one ear. Paroxysmal attacks of intense vertigo and defect in bone conduction are frequent symptoms.	Aural ataxia or vertigo. Ménière's Disease, (685, 918, 1019).	650
well marked heredity usually. Staggering gait, but ataxia also in arms, coarse, irregular tremor, simulating jerky choreiform movements. Nystagmus defective. Symptoms present a mixture of weakness and ataxia. — Occurs after puberty. Knee-jerks present. Ocular paralysis, loss of pupil reflex and optic atrophy common.	Marie's hereditary cerebellar ataxia, (669, 782).	651
well marked heredity usually. Staggering gait, but ataxia also in arms, coarse, irregular tremor, simulating jerky choreiform movements. Nystagmus defective. Symptoms present a mixture of weakness and ataxia. — Occurs before puberty. Knee-jerks absent, except in early stage and in exceptional cases. Babinski reflex usually present. Optic atrophy rare. Club-foot common.	Friedreich's Disease. Hereditary Ataxia, (670, 687, 762, 781).	652
oved easily in all directions without ataxia when patient is lying down, but collapse when she tries to walk, apparently from lack of confidence and will power. Simulates an apraxia than an ataxia. Often has an emotional cause and hysterical symptoms (425) are present. Both legs are involved.	Astasia and Abasia, (287, 792).	653
cle sense. May or may not be motor paralysis of same side with analgesia and thermic anesthesia of contralateral side. One leg only involved. No ataxia when moving leg while but marked ataxia while walking	Lesion of lateral column of spinal cord, involving direct cerebellar tract, (648, 1356, 1360, 1396). (Figs. 24-7.)	654
usually Often in of be — Marked anesthesia without analgesia. No motor paralysis. No other symptoms. Very rare. May be the earliest stage of a spinal tumor. Unilateral later becoming bilateral.	Lesion of posterior column of cord, (785, 1350-1 1347, 1396). (Figs. 24-7.).	654a
usually Often in of be — Apoplectiform attack followed by hemiplegia (sensory oftener than motor). Other post-hemiplegic motor disturbances are often present. The ataxia occurs in convalescence in cases where the motor paralysis was slight.	Post-hemiplegic ataxia (lesion in or near posterior part of optic thalamus (1275). (Fig. 17.)	655
usually Often in of be — There are often ataxia and loss of muscle sense on one side of body and analgesia and thermic anesthesia on the other side. There may be crossed paralysis. Dysphagia and dysarthria and paralysis of various cranial nerves are usually present.	Softening, hemorrhage or tumor in brain stem, (535 et. seq., 830, 1268-71). (Figs. 19-22.)	656
usually Often in of be — Signs of cortical irritation (convulsions). Anesthesia, especially loss of muscle sense, is often present. Headache common. May be some mental disturbance.	Softening or tumor of contralateral parietal cortex. (1355, 1362). (Fig. 15.)	657
cholism. Blurred and foolish speech. Ataxia and other symptoms. Temporary tremor.	Alcoholic intoxication, (663, 673, 764, 780).	658
Usually both motor and sensory symptoms. Irregular, jerky, ataxic movements of both arms and legs, and movements are slow. Gait is often both spastic and ataxic. Very characteristic ech, nystagmus, especially on motion of eyeball, and atrophy of optic nerve. In some cases bulbar paralysis (434) is an early symptom. Patients are often emotional and exhibit mental weakacute course and has been called "acute ataxia," of which there are several varieties (662). In its early stages the diagnosis of this disease is often exceedingly difficult. The most ymptoms only explicable on the assumption of the existence of several, separate, small lesions.	Disseminated Sclerosis. Myelitis Disseminata. Encephalomyelitis, (511, 580, 668, 688, 756, 765, 799, 913, 1051).	659
motor ataxia (661) and spastic paraplegia (525) in varying proportions. Little or no pain. Weakness, stiffness, ataxia, paresthesiae and anesthesia of legs. Vibration sense (56, 353) is can be detected. In later stages arms may be somewhat involved. A rare disease. Occasionally some involvement of cranial nerves. Organic reflexes slightly, or not at all, disordered. rds the end of the disease, but Babinski persists. This disease may be caused by pernicious and other severe anemias.	Ataxic Paraplegia. Combined Sclerosis, (526, 796). (Figs. 24-6.)	660
sis; but hypotonia (252), allowing hyperextension and extreme mobility of joints, is common, and temporary paralyses in the domain of the cranial nerves (especially the muscles of eyeptoms. Movements are ataxic, quick, violent, excessive and constantly controlled by eyesight. The affected parts cannot be held motionless in one position long. In walking, legs are too far outward and too far forward and are brought back hard on heel. Ataxia much worse when eyes are closed. Walking in the dark or backwards is usually impossible. Romberg's phenomenon (447), myosis and optic atrophy with concentric limitation of field of vision are common. Lightning pains of great intensity in small areas followed by hyperalgesia, girdle vomitent symptoms. Organic reflexes, especially the vesical, are disordered. Arthropathies (1186), or perforating ulcer or other trophic disorders may be present. Anesthesia and analnar hyperthesia). Retardation of conduction of pain. Visceral crises (987) are usually present. Towards the end of the disease motor paralysis may appear. Lymphocytosis and globulin se is sometimes divided into three stages—(1st), the neuralgic; (2nd), the ataxic; (3rd), the paralytic stage. History or other evidences of syphilis, or of venereal disease is usually present. present. There are several forms of tabes. In the cerebral form, atrophy of the optic nerve is the prominent symptom and the legs show little ataxia. In cervical tabes the arms are form the legs are mainly affected. In all forms the knee-jerks are absent. Babinski is present in rare cases, complicated by lateral sclerosis. In many cases of tabes the ataxia is slight on the absence of the knee-jerk, the Argyll-Robertson phenomenon and the cerebro-spinal lymphocytosis, together with whatever other symptoms may be present.	Locomotor Ataxia. Tabes Dorsalis, (433, 756, 784, 827, 894, 979, 987, 1004, 1217, 1231) (Figs. 24-6).	661
Muscles tender and atrophic. Pains rarely very severe and partial reaction of degeneration and retardation of conduction of pain often present. Organic reflexes normal. Pupil reaction volved. Usually sensory symptoms. Usually history of alcoholic abuse. Mental disturbances in many cases. Occasionally the disease runs an acute course, "acute ataxia," (659) and is bulin or lymphocytosis in cerebro-spinal fluid. Prognosis is good.	Multiple Neuritis. Polyneuritis. Pseudo-tabes, (488, 787, 823, 1008, 1147, 1307)	662
ing drugs, alcohol, etc. — The ataxia is usually associated with tremor and neurasthenic symptoms. Failure of memory and other signs of mental impairment. Patients are emotional and excitable.	Drug habit (toxic), (482, 658, 764, 780).	663
se. — The ataxia may appear only when eyes are closed and is usually associated with cortical anesthesia. Hysterical symptoms (425) are present. Rarely fall, but usually collapse on reaching a place of safety.	Hysterical Ataxia, (1074).	664

CHART XII b

Tremor, Nystagmus, Fibrillation

Comprising Numbers 639 to 647 on left side of Chart
and 668 to 697 on right margin

639 TREMOR (250)

- 645 Intention Tremor (290).
  - Coarse, irregular tremor; 4 to 8 per second.
    - Tremor is usually associated with scanning speech, nystagmu… Usually a great variety of motor and sensory symptoms d… sionally with their loss, over a very variable area. The … Vertigo is a very common symptom.
    - Occurs in family groups and shows well marked heredity. Staggering gait. Ataxia. Nystagmus is common and speech often defective.
      - Occurs after pub…
      - Occurs before pu…
  - Fine tremor.
    - Tremor is associated with general weakness or convalescence …
- 646 Passive Tremor. Increased on voluntary motion and excitement (289).
  - Fine, rapid tremor; 8 to 12 per second.
    - Exophthalmus, goitre, tachycardia, vascular throbbing. flus… when patient looks downward (Graefe's symptom). The g…
    - History of addiction to alcohol or drugs. Mental symptoms … greatly at different times.
    - Presence of hysterical symptoms (425). Tremor is worse wh…
    - Tremor is marked in face, lips and tongue. Progressive me… cytosis and globulin in cerebro-spinal fluid. Wassermann …
    - Slow tremor of hand and foot of same side, associated with c…
  - Slow tremor; 3 to 6 per second.
    - Tremor, which is associated with muscular rigidity and mask-li… involves the other side. The tremor is most marked in the … acteristic attitude (head and body bent forward, elbows fl… ilar tendency to run backwards (retropulsion). The attitu… The disease often commences with a stiffness and slowness … no sensory symptoms except the sensation of rigidity and …
- 647 Passive Tremor. Diminished on voluntary motion (289).
  - Slow, fine tremor; 3 to 6 per second.
    - Tremor begins bilaterally. Head is early affected. Nodding …
  - Slow, coarse tremor.
    - Rotatory or nodding tremor of head occurring suddenly in ri… involved. The tremor ceases when the child's eyes are clo…
    - A series of jerky tremors limited to the back, or involving al…
- Either Intention or Passive Tremor.
  - Not associated with other nervous symptoms. Hereditary b…

640 NYSTAGMUS (291)

Always a symptom of organic disease. Very rarely, an hysterical clonic spasm may simulate true nystagmus (pseudo-nystagmus). This is often vertical and is more rapid and more violent than nystagmus and is associated with other hysterical symptoms (425).

- No weakness of any rectus muscle.
  - Impairment of sight.
    - Defective vision from whatever cause, …
    - Due to lack of pigment in iris, choroid …
    - Workers in mines. Due to working i…
  - No impairment of sight.
    - Vertigo is a prominent symptom.
      - Paroxysmal attac… violently, or ga…
      - Vertigo, cerebella…
    - Coarse, jerky tremor is a prominent symptom. Ataxia is also present.
      - Occurs in early y…
      - Occurs at any ag…
    - Cerebral symptoms present. Occurs in mening…
    - Rickety baby in winter. Most marked wh… ing of the hea…
    - Congenital. Lateral oscillatin…
- Weakness of one or more of the recti muscles. Nystagmus occurs in convalescence fr…

641 FIBRILLARY CONTRACTION OR FIBRILLATION (292).

- Evidence of organic disease. Degeneration of peripheral motor neurons.
  - Marked muscular atrophy with muscular weakness.
    - Marked sensory symptoms. Analgesia and the …
    - No sensory symptoms.
      - Muscular atrophy …
      - Muscular atrophy …
      - Muscular atrophy …
- Evidence of functional not organic diseases.
  - No muscular atrophy or weakness. Occurs usually in …

ABSTRACT OF SYMPTOMS	DIAGNOSIS	
phy of optic nerve and ataxia. Reflexes are usually exaggerated and ankle-clonus and Babinski are present. In some cases the deep reflexes are early abolished and the organic reflexes disordered. any local lesions, although all these may be absent. The motor symptoms commence as fatigue, slowly becoming paresis, rarely paralysis, and are usually associated with exaggerated reflexes, occa- symptoms are usually in the form of paresthesiae, more rarely pain followed by irregular patches of anesthesia. Epileptiform or apoplectiform attacks followed by transitory paralysis are common.	Multiple Sclerosis. Disseminated Sclerosis, (511, 580, 659, 688, 756, 765, 799, 913, 1051).	668
Knee-jerks are present. Ocular paralysis, loss of pupillary reflex and optic atrophy are common.	Marie's Hereditary Cerebellar Ataxia, (651, 782).	669
Knee-jerks are absent except in early stage or in rare and exceptional cases. Babinski is present. Optic atrophy rare. Club-foot common. Rarely sensory symptoms are present.	Friedreich's Hereditary Ataxia, (652, 687, 762, 781).	670
n acute disease. Anemia is usually present, but no evidence of any organic disease of the nervous system. Exhaustion or holding a limb in a strained position for a long time causes tremor.	Asthenic Tremor or weakness, (790).	671
reating, diarrhoea, much nervousness, tremor especially marked on excitement, polyuria and at times albuminuria, insomnia and vertigo are common symptoms. The upper lid does not fall normally of the parenchymatous variety and need not be large.	Exophthalmic Goitre. Baseclow's Disease. Graves' Disease, (1193).	672
mmon, moral sense and judgment impaired and speech blurred. Patient is restless and emotional. Pupils contracted in opium cases. Quinquand's sign (453) in alcoholic cases. Symptoms vary	Toxic Tremor (alcohol, opium, nicotine, mercury, etc.), (658, 663, 780).	673
ntion is directed to it. Irregular tremor. Evidence of great exhaustion of the nervous system. Often history of injury associated with fright.	Hysterical or Neurasthenic Tremor and also Traumatic Neuroses, (1072-5).	674
airment. Restless and childish. Speech is slurred by elision of syllables and letters. Apoplectiform and epileptiform attacks may occur. History of syphilis. Lumbar puncture shows a lympho- usually positive. Argyll-Robertson's phenomenon.	Paralytic Dementia. General Paresis. Paresis, (579, 763, 895, 1049, 1104, 1216, 1230).	675
otor paralysis of the opposite side. Other symptoms of a severe brain lesion. May be associated with hemiplegia, sensory or motor. Tremor ceases during sleep and is increased in excitement.	Lesion of the Crus Cerebri or Pons involving the Rubro-spinal tract, (441, 1270, 1325). (Figs. 18-20).	676
, consists of "pill rolling" movements of fingers and general tremor, which commences in one extremity, later extends to the other extremity of the same side (rarely to that of the opposite side), and finally and hands; the head and body escape, except in very rare cases. Disease commences after forty years of age and progresses slowly. The tremor usually becomes coarser in the later stages. Char- ees slightly flexed) and festinating gait or propulsion: a tendency to fall forward which compels patient to walk faster and finally to run until he falls forwards or against an obstacle. He has a sim- and mask-like face are all due to muscular rigidity which is more characteristic of the disease than is the tremor, and in rare cases occurs alone without any tremor (paralysis agitans sine agitatione). ement of one arm or hand, simulating a mild paralysis, the tremor appearing later. Voluntary movements are slow, much restricted and feeble, but are never completely paralysed. There are g of heat, which are frequent and distressing symptoms. Patient slowly becomes entirely helpless.	Paralysis Agitans. Parkinson's Disease, (612, 766, 800).	677
ient. No rigidity. General weakness. Atheromatous arteries.	Senile Tremor.	678
abies in the winter time and passing off in the summer time. Usually accompanied by nystagmus which grows worse when the child's head is held still. Occasionally other muscle groups are	Spasmus Nutans. Nictitatio Spastica, (690).	679
les, caused by cold (physiological) or infection (pathological) and resulting in an increase in the body's temperature and may be followed by high fever.	Chills. Rigors. Shivering.	680
coholism in ancestors, etc.). May be local or general. Usually in advanced age.	Essential Tremor.	681
ital or acquired, in early childhood. Often associated with blepharospasm and oscillation of head.	Amblyopia, (350).	682
Usually associated with photophobia.	Albinism.	683
light and looking sideways constantly while at work.	Miner's Nystagmus.	684
ertigo associated with deafness and tinnitus aurium and due to disease of inner ear. Nystagmus in the opposite direction from the labyrinth involved occurs occasionally when head is rotated ed, or ear syringed, or labyrinth in any way irritated. The caloric reaction (79) is absent.	Ménière's Disease. Aural vertigo. Labyrinthine Vertigo, (650, 918, 1019).	685
n and other symptoms of disease of the cerebellum. The nystagmus is usually towards the side of the lesion.	Cerebellar Disease, (609-10, 648, 783, 1016, 1272).	686
Strong heredity. Knee-jerks absent, except in early stage. Babinski is present. Optic atrophy rare. Muscular weakness and contractures are not uncommon.	Friedreich's Hereditary Ataxia, (670).	687
heredity. Intention tremor. Exaggerated knee-jerk, Babinski, optic atrophy and scanning speech are common symptoms. Vertigo is usually present (932).	Disseminated Sclerosis, (668, 765).	688
d in local lesions (tumors especially). Nystagmus is commonly present in bulbar lesions, especially in those involving the cerebellar peduncles and tracts and the posterior longitudinal bundle.	Cerebral Disease (especially of the brain stem).	689
child's head is held still and the tremor prevented (679). Not to be confounded with the deep bowing (Salaam eramp) which occurs in some idiots and epileptics, with or without nystagmus. Roll- occur in otitis media and in rickety children.	Spasmus Nutans. Nictitatio Spastica, (679).	690
gmus associated with jerking movements of the limbs or trunk, aggravated by cold and by percussion. Associated with other congenital defects.	Nystagmus-myoclonus.	691
ular palsies or when weakened muscles are strongly exerted.	Ocular Muscular Insufficiency, (816). (Figs. 14, 18).	692
nesthesia with only slight anesthesia, or none at all. Trophic disturbances and mutilation. Slight tactile impressions are often painful.	Syringomyelia, (552, 837-9, 1009, 1170, 1187, 1357-9). (Figs. 25-7.)	693
ibrillary contraction of tongue and lips, dysarthria, dysphagia and spastic paraplegia.	Chronic Bulbar Paralysis, (546, 1150). (Fig. 21-2)	694
ibrillary contraction of small muscles of hands and of shoulder girdle combined with spastic paraplegia.	Amyotrophic Lateral Sclerosis, (547, 797, 1149). (Figs. 24-6.)	695
ibrillary contraction of the peronei muscles. Rarely there are pain, muscle twitching and anesthesia.	Spinal or Neuritic Muscular Atrophy, (496).	696
ularis palpebrarum but may occur in any muscle. It consists in a quivering of the muscle fibers and occurs in neurasthenic and exhausted persons.	Myokymia. Myoclonia, (203).	697

CHART XII c

Local Palsies

Comprising Numbers 700 to 721 on right margin

(Note)—The anesthesia accompanying these palsies can be seen from the areas of cutaneous distribution of these nerves depicted in the plates at the end of the book (Figs. 33–8). In mild lesions of the nerves anesthesia is either absent or much less marked and less extensive than the motor paralysis.

LOCAL PALSIES

	Abstract of Symptoms	Diagnosis
Inability to Move, More or Less, Muscles of the EYEBALL	There are ptosis and strabismus divergens and the pupil is dilated and immobile both to light and accommodation (this condition of the pupil may occur as an isolated paralysis,—333). The eyeball can be moved in no direction except outward (abducens), and outward and downward with rotation of eyeball (superior oblique). For symptoms characteristic of the isolated paralysis of each ocular muscle see Chart XIV c, 816. When the superior oblique muscle is paralysed the levator palpebrae superioris is paralysed with it and ptosis results.	Paralysis of Motor Oculi. (Figs. 14, 18). 700
	The ocular muscles, except the levator palpebrae superioris, have a bilateral cortical representation. Hence ocular paralyses, except ptosis, almost never occur in lesions above the oculomotor nucleus, except in bilateral lesions. The cortical representation of the ocular muscles seems to be very diffuse or multiple. Conjugate deviation may result from supra-nuclear lesions.	
	For the symptoms of paralysis of the trochlearis (patheticus) nerve and of the abducens nerve, each of which produces a strabismus convergens, see 816.	Paralysis of Trochlearis and of Abducens. 701
JAW	The muscles of mastication of one side, rarely of both sides, are paralysed and in severe cases atrophied. The temporal and masseter muscles cannot be felt firmly contracting when efforts are made to chew. The jaw cannot be closed tightly or opened strongly or moved laterally towards the healthy side (external pterygoids), or the chin pushed forwards (internal pterygoids). Mastication of food is difficult or impossible; dysmasesis (286). The jaw reflex (322) is abolished. In some cases one side of the soft palate (tensor veli palatini) is paralysed and in some the hearing of low tones is unpleasant (tensor tympani).	Paralysis of motor branch of Trigeminus. 702
	In trigeminus lesions there is unilateral abolition of the conjunctival, corneal, sneezing and palatal reflexes; and the secretion of tears is at times affected. There is no irritation, or tears, from inhaling ammonia or acetic acid. There is also loss of sense of taste, and dilatation of the pupil, narrowing of the eyelid slit, even enophthalmus, are present. Heat and redness of skin in recent cases and coldness and cyanosis of skin in old cases. The salivary secretion and taste are affected when either the proximal or the distal end, but not the middle, of the nerve is affected.	
FACE	The muscles of expression of one side (rarely of both sides) of the face are paralysed. The forehead cannot be wrinkled and the eye appears larger than normal and cannot be closed (lagophthalmus, hare's eye). When attempts are made to close the eyelids the eyeball turns upward, the cornea disappearing behind the upper lid (Bell's phenomenon). The angle of the mouth is lower than normal and cannot be raised. The naso-labial fold is obliterated. The lips cannot be firmly closed; so that whistling is impossible and speech is impaired. Mastication is difficult because the weakened buccinator muscle allows food to collect between the jaws and the cheek. The platysma is also paralysed; so that the angle of the mouth cannot be drawn downwards. Tears may flow from the eye and irritate the cheek and saliva from the angle of the mouth. The conjunctiva may become inflamed and the cornea ulcerated, because the eyelid cannot wink and keep the conjunctiva clean. In some cases the facial paralysis may be preceded and accompanied by pain. In severe cases the paralysed muscles exhibit the electrical reaction of degen-	Facial Paralysis. Bell's palsy. Prosopoplegia. Facial Monoplegia. Facial Diplegia, (751, 928, 1317). 703

	Abstract of Symptoms	Diagnosis
Inability to Move, More or Less, Muscles of the Face (Cont.)	eration. Hearing and taste are frequently impaired and disordered. When taste is affected the salivary secretion is also affected. In the early stages of the disease the face is drawn over toward the healthy side by the unantagonized healthy muscles. In the later stages the face may be drawn back again permanently towards the paralyzed side by the contracting newly formed connective tissue in the degenerated muscles. Also in the early stage of recovery the face may be drawn towards the paralyzed side by over-innervation of the muscles formerly paralyzed, and may exhibit temporary contractures and spasms, possibly "associated movements." These spastic symptoms may be due to irregular regeneration of the nerve. The upper fibres of the facial nerve have a bilateral cortical representation as do the laryngeal nerves. Hence lesions of the cerebral hemispheres paralyse mainly the lower branch of the facial; the eye on the paralysed side can be closed but is easily forced open. For the localization of the different forms of facial paralysis, see 1317.	
Larynx	Paralysis of the pneumogastric nerve is discussed under 760. In addition to the laryngeal paralysis there is often present disorder of the respiratory act and of the heart beat (tachycardia).	Pneumogastric Paralysis (760). 704
Neck	When the tip of the shoulder sinks downwards and forwards and the arm cannot be easily raised, there may be a paresis of the trapezoid muscle. When this muscle is paralysed on both sides, the head tends to fall forward. When the head is drawn towards one shoulder and the chin turned upwards and towards the other, the sterno-cleido-mastoid muscle is paralysed on that side toward which the chin turns. This posture is called caput obstipum spasticum, when the muscle is atrophied and secondarily contracted and the deformity can no longer be corrected by passive motion. Caput obstipum spasticum occurs also and is more pronounced in torticollis from spasm of the muscle (730). When the sterno-cleido-mastoid muscle is paralysed on both sides, the head tends to fall backwards.	Paralysis of the Spinal Accessory. 705
Tongue	The tongue when protruded turns towards the paralysed side. When both sides are paralysed the tongue cannot be protruded at all, and in such cases, speech, mastication and deglutition are difficult and imperfect. In lesions of the *nucleus* of the hypoglossus nerve there is also a mild paresis of the orbicularis oris muscle. Intracranial lesions involving the hypoglossus and other nerve roots at the base of the brain may cause Avelli's syndrome: pharyngo-laryngeal or glosso-pharyngo-laryngeal paralysis (546); or may cause Schmidt's syndrome: the above and also sterno-cleido-mastoid and trapezius paralysis.	Hypoglossus Paralysis. (546, 752). 706
Diaphragm	The diaphragm is paralysed on one or both sides, causing dyspnoea on exertion and sinking in of the epigastrium on inspiration, especially on deep inspiration. The lower part of the lung is drawn upwards and atelectasis and pneumonia may occur. Besides the usual causes of compression and neuritis, this paralysis may also occur in pleurisy, peritonitis, trichinosis and in bulbar and spinal lesions. The paralysed diaphragm shows Litten's phenomenon.	Phrenic Paralysis. 707
Arm	The supra-and infra-spinatus muscles are paralysed; so that rotation of the arm outward and raising it in abduction are impaired. Muscles involved are atrophic and ulnar side of hand is turned forwards.	Supra-Scapular Paralysis. 708
	The serratus anticus major is paralysed; so that when the scapula is raised, its lower angle approaches the vertebrae and the inner margin of the scapula does not lie close to the thorax and, on movements of the arm upwards and forwards, stands from the thorax like a wing. The arm cannot be raised beyond a horizontal line.	Long Thoracic Paralysis. Serratus Paralysis. 709

	Abstract of Symptoms	Diagnosis	
Inability to Move, More or Less, Muscles of the	Motion of the arm inward and forward is impaired. Hand cannot be placed on opposite shoulder.	Anterior and Posterior Thoracic Paralysis.	710
	Rotation of the arm inward and motion of the arm backward are impaired.	Sub-Scapular Paralysis.	711
Arm (Cont.)	The deltoid and teres minor are paralysed: so that the arm cannot be raised.	Axillary Paralysis.	712
	The combined paralyses of the brachial plexus: Erb's and Klumpke's paralysis, are discussed under 454, 455 and 490.		
	The biceps, brachialis anticus and coraco-brachialis muscles are more or less completely paralysed; so that flexion of the arm at elbow is more or less impaired, especially in supination (very rare).	Musculo-Cutaneous Paralysis.	713
Hand	The pronators and flexors of the hands and fingers, the muscles of the ball of the thumb and the first and second lumbrical muscles are paralysed. The hand can neither be flexed nor pronated. The thumb cannot be brought across hand to touch the little finger, but remains close to the index finger (ape's hand). The first (proximal) phalanges of fingers can be flexed, but not the second and third phalanges.	Median Paralysis.	714
	The interossei, the third and fourth lumbricals, and the muscles of the little finger are paralysed. The proximal phalanges cannot be flexed, the other phalanges cannot be extended and the little finger cannot be moved. The fingers cannot be spread. When muscle atrophy and contracture occur "claw hand" results.	Ulnar Paralysis.	715
	The extensors and supinators of the hand and fingers, and the abductor pollicis longus, are paralysed. The thumb is adducted and can neither be abducted nor extended. Wrist-drop and slight pronation. Wrist and fingers cannot be extended completely. The wrist-drop differs from that of lead palsy (494) in that the supinator longus is paralysed. Therefore, if the forearm is held midway between supination and pronation and the elbow strongly flexed against a resistance offered, the belly of the supinator longus will not stand out firmly contracted as it will in lead paralysis and in health.	Musculo-Spiral and Radial Paralysis.	716
Leg	The extensor femoris is paralysed; so that flexion of the thigh on the body and extension of leg on thigh are impossible or difficult. Standing and walking are difficult, and ascension, jumping and running impossible.	Crural Paralysis (997).	717
	The adductor muscles of thigh are paralysed; so that adduction of leg, pressing of thighs together and crossing of legs are impossible.	Obturator Paralysis.	718
	The glutei muscles are paralysed; so that walking, ascending stairs, straightening up of body, abduction and rotation of thigh are impaired. Generally much muscular atrophy.	Gluteal Paralysis.	719
	Foot and toes are paralysed; the leg cannot be flexed on thigh and rotation of the thigh is impaired. In cases of isolated tibialis paralysis there is absence of plantar flexion of foot, and of plantar, flexion, spreading and adduction of toes (Pes calcaneus et valgus). In cases of isolated peroneal paralysis there is absence of dorsal flexion and abduction of foot and its adduction impaired—absence of dorsal flexion of toes. There are foot-drop, high stepping gait and Pes equino-varus.	Sciatic Paralysis. (996)	720
	For paralysis from lesions of the cauda-equina, see 487, 1007 and 1308.	Cauda Equina Paralysis. (Fig. 29).	721

CHART XII d

Local Spasms

Comprising Numbers 725 to 733 on right margin

LOCAL SPASMS

	Abstract of Symptoms	Diagnosis
Spasm of Muscles of Jaw	The jaws are held tightly shut and the masseter and temporal muscles can be felt to be contracted (lock jaw), usually bilaterally. The spasm may be "tonic," as in tetanus (606), tetany (614), irritation of teeth (wisdom teeth) and certain unilateral lesions of the pons and medulla; or "clonic," as in chills and in rare cases of paralysis agitans and hysteria. When the pterygoid muscles alone are in spasm the mouth is held open and cannot be closed.	Trigeminal Spasm or Cramp. Trismus. 725
Face	Spasms of one or more muscles of expression of the face, unilateral or bilateral, are relatively common, as in convulsive tic (601) and tic douloureux (602). These spasms are often a mixture of tonic and clonic contractions, the clonic predominating. They may affect all the muscles or only one, as in tonic spasm of the orbicularis palpebrarum (blepharospasm) (601, 617), or in clonic spasm of this muscle (spasmus nictitans: nictation). The platysma myoides often takes part in these spasms and very rarely the muscles of the soft palate and the internal and external ear muscles. Very rarely spasm of some of the facial muscles about the mouth constitute an occupation neurosis or cramp, as in the "Auctioneer's cramp" and "Cornet player's cramp." These facial cramps may be symptomatic directly of lesions of the cortical facial center, of the facial nerve in its course, and reflexly of the trigeminal nerve or its terminal filaments in the eye, nose, mouth or ear. There is also to be remembered the passive contracture of the degenerated muscles and the active contracture due to over-innervation of the convalescing muscles in facial paralysis. Causeless and uncontrollable laughter must also be classed among the facial spasms. This condition, similar to the allied state of causeless and uncontrollable crying, occurs especially in hysteria and in lesions of the optic thalamus.	Facial Spasm or Cramp (267, 601). 726
Pharynx	Spasm of the pharynx of a tonic nature preventing swallowing and of a clonic nature repeating the act of swallowing with great frequency occur. The former occurs in hydrophobia (607) and somewhat also in tetanus (606); while the latter, associated with coma, frequently occurs in mild epileptic attacks. The spasm also occurs from irritation of the pharynx in hysteria and very rarely, as one of the crises in locomotor ataxia (433). Spasm of the oesophagus is not uncommon in hysterical persons and makes the swallowing of food very difficult.	Glosso-Pharyngeal Spasm or Cramp. 727
Larynx	Spasm of the muscles of the larynx (spasmus glottidis, false croup, laryngismus stridulus), causing noisy and difficult breathing, is a not uncommon and occasionally a dangerous condition. It occurs almost exclusively in children and is often associated with rickets and with digestive disorders. Occurs also in general diseases such as hydrophobia, hysteria, epilepsy, chorea, tabetic crises, etc. Sneezing (sternutatio spastica, ptarmus) and coughing, reflex acts implicating both the pneumogastric and the intercostal nerves, are often due to pathological conditions and irritation of the nervous system. Bradycardia, Cheyne-Stokes' respiration and cerebral vomiting are symptoms of irritation of the pneumogastric nucleus, but are not characteristic and are of little diagnostic value.	Pneumogastric Spasm or Cramp. 728
Tongue	Spasm of the tongue is very rare, especially so the tonic form. During the attack speaking and swallowing is impossible. Very rarely a tonic spasm of the tongue occurs when the patient attempts to speak (stuttering and aphthongia). Spasm of the tongue is sometimes associated with facial spasm and with spasm of the submaxillary muscles. These spasms may be due directly to lesions of the cortical tongue center, of the hypoglossus nerve in its course, or reflexly, especially from lesions of teeth, mouth and nose.	Hypoglossus Spasm or Cramp. 729

LOCAL SPASMS (Continued)

Spasm of Muscles of	Abstract of Symptoms	Diagnosis
NECK	Spasm of the neck muscles, especially the sterno-cleido-mastoid: caput obstipum (spastic wry neck), is sometimes congenital and is sometimes acquired in later life. In these cases the head is drawn toward the shoulder of the affected side and the chin is turned toward the other side and slightly elevated and the sterno-cleido-mastoid muscle can be felt to be firmly contracted. When the trapezius is the seat of the spasm the occiput is drawn backwards and turned toward the shoulder of the affected side and the edge of the muscle can be felt to be firmly contracted. Spasm of the muscles is sometimes tonic, sometimes clonic and often both. The cause of these spasms is often neurotic and often rheumatic. Rarely it is some disease of the eye or of the ear (torticollis ab oculo laeso, ab aure laesa) or of the cervical vertebrae. Usually many muscles are involved, although one or two more prominently than the others.	Spinal Accessory Spasm or Cramp (601). 730
DIAPHRAGM	Tonic spasm of the diaphragm, either unilateral or bilateral, occurs very rarely and produces dangerous dyspnoea. It sometimes occurs as one symptom of a general disease: tetanus, hydrophobia, hysteria, etc. Clonic contractions are common and cause hiccough (singultus), always a distressing and at times a dangerous symptom, which occurs occasionally in brain and spinal cord lesions and frequently in irritation of the pneumogastric nerve, especially from the gastric mucous membrane. A similar but slower contraction of the diaphragm associated with facial spasm (opening of mouth) causes the act of yawning (oscedo, chasmus) which is sometimes frequently repeated as an aura of apoplexy or epilepsy and occurs also in hysteria, digestive disorders, drowsiness, etc.	Phrenic Spasm or Cramp. 731
ABDOMEN	Tonic and clonic contractions of some or all of the abdominal muscles occur with extreme rarity, and are usually, if not always, hysterical.	Intercostal Spasm. Abdominal Spasm. 732
ARM AND LEG	Tonic and clonic spasms of the muscles of the arm and shoulder or of the leg, with the exception of the secondary contractures due to lesions of the pyramidal tract and of the peripheral nerves, are very rare. They usually are due either to deficiency of water in the system, and often occur in disease in which much water is lost, as cholera, diarrhoea, etc., or to hysteria, or to rheumatic factors, or are reflex. The deformity resulting in each case can be predicted from the function of the muscle involved.	Brachial, or Lumbar, or Sciatic Plexus, Spasm or Cramp. 733

CHART XIII

Disorders of Speech and Gait

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptoms Analysed	Character of Disorder	
735 DISORDERS OF SPEECH, READING AND WRITING.	737 ANARTHRIA (283) Inability or unwillingness to speak. No disease of vocal organs or peripheral nerves. This condition may result from a complete aphonia (260) or complete aphasia (221) or complete dysarthria (284).	The diseases in which Anarthria and Dysarthria occur are set forth in Chart XIII a.
	738 DYSARTHRIA (284) Ability to express thought by speech but articulation is defective.	
	739 APHASIA (221) Articulation normal but expression of normal thought is defective.	The varieties of Aphasia and the conditions under which they occur are set forth in Chart XIII b.
736 DISORDERS OF GAIT.	740 ATAXIC 741 PARALYTIC AND FLACCID 742 PARALYTIC AND SPASTIC	The diseases in which Disorders of Gait occur are set forth in Chart XIII c.

CHART XIII a

Anarthria and Dysarthria

Comprising Numbers 737 and 738 on left side of Chart
and 743 to 768 on right margin

737 ANARTHRIA (283)

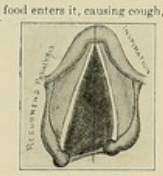
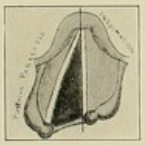
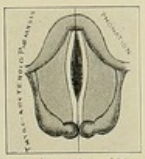
- Result of disease in infancy, or congenital.
  - Auditory memories necessary for understanding spoken words were never acquired, or were early lost through disease; hence innervation memories necessary for speech were never learned.
    - May make noises but cannot speak.
    - Can be trained to speak through the sight.
- Result of disease in adult life.
  - Innervation memories necessary for speech have been acquired but are not available. No hysterical symptoms. — Complete absence of speech, and readi is impossible. Patient makes no speak or to communicate by gestures.
  - Hysterical symptoms and etiological factors present, although not always prominent.
    - Will neither whisper nor speak.
    - Can whisper faintly but distinctly.
  - Apoplectic symptoms (504). — May mutter but cannot articulate dist

738 DYSARTHRIA (284, 388)

- Congenital.
  - Vocal organs defective. — Words imperfectly formed, also a nasal voice. An ex
  - Vocal organs normal. — Words imperfectly formed and usually a very limited
- Defective Education. — Vocal organs normal. — Substitution of one letter for another. An examinati speaks the vowels correctly but has difficulty in sp
- Paralytic. — The labials, the linguals or the vowel sounds or all of them cannot be properly pronounced. A careful examination reveals a paralysis or a paresis within the domain of the facial, the hypoglossal or the pneumogastric nerve.
  - No symptoms of any central disease.
    - Patient cannot whistle or close lips tightly.
    - Tongue is not protruded straight but deviates to paraly
    - Soft palate is not raised (bilateral) or not raised symme
    - Anesthesia of larynx. Paralysis of crico-thyroid muscl on lower level) and of thyreo-ary-epiglottis muscle (
    - Immobility of one or both vocal cords from paralysis of veric position of cords (between extreme adduction in cases of unilateral paralysis, the healthy cord mo paralysed cord.
    - Immobility of one or both vocal cords from paralysis o cord or cords lie near the median line (extreme add become smaller on inspiration.
    - Immobility of one or both vocal cords from paralysis arytenoid lateralis muscles) and in some cases the aryte and hoarse. Cords are wide open (abduction) in all, or
  - May be symptoms of central disease. — Unilateral or bilateral paralysis of the soft palate, of all the laryngeal muscles and anesthesia of larynx.
- Tremor and Ataxia.
  - Slow and clumsy speech. — Cerebellar gait. — Speech sounds as if a foreign bod
  - Tremulous and slovenly speech, words are badly formed, letters and syllables are left out both in speaking and writing. — Evident mental deterioration.
    - Argyll-Robertson's phenomenon attacks may occur. Childishn
    - Alcoholic history, appearance, tre
  - Scanning speech. — Intention Tremor. — Great variety of widespread mot
- Rigidity. — Monotonous speech. — Passive Tremor. — Rigidity of muscles and slight fl
- Spasm.
  - Certain letters (consonants) are spoken with difficulty and are repeated many times imperfectly be
  - Utterance is arrested by a spasm of one or more of the muscles concerned in speech, such as the hyp is directed to the speech the worse it becomes. Singing is usually not at all affected.

DIAGNOSTIC ANALYSIS OF SYMPTOMS

ABSTRACT OF SYMPTOMS

				DIAGNOSIS	
	Expressions of face and actions are idiotic.	Reading and writing impossible.	Patient exhibits little or no intelligence and history shows that he never had any. Incapable of any but the most elementary education, if of any. Soils himself with urine and feces.	Idiocy, (1081).	743
e of	Expressions of face and actions are normal.	Reading and writing are possible after much training.	Patient shows a good intelligence, but can express himself only by gestures and that only as the result of careful training. Some deaf mutes can be taught to speak, generally very imperfectly.	Deaf Mutism.	744
loud t to	Absence of facial expression and of all voluntary actions.	Patient lies in a deep sleep and cannot be aroused to any expression of consciousness or intelligence by stimulation of any sensory surface.		Coma, (205, 1037).	745
	Facial expression and actions are abnormal.	Patient is evidently insane and has delusions. When recovery has taken place it may be learned that his silence was due to a delusion, usually either of fear or of a divine command (delusional insanity). Anarthria is also common in profound dementia.		Insanity, (1041).	746
	Hysterical appearance. Placid and contented.	Probably only a severe form of hysterical aphonia. It is a rare condition. Most cases can be persuaded to whisper a few words.		Hysterical Mutism, (1074).	747
		Can, by hard urging, be made to whisper some words faintly but distinctly.		Hysterical Aphonia, (759, 1074).	748
ly.	Sudden onset, usually followed by speedy death.	Paralysis of motion or sensation or of both in the extremities.		Apoplexy in pons or medulla.	748a
	ation of the vocal organs shows no paralysis, but a developmental defect; such as cleft palate and similar malformations.			Cleft Palate, etc.	749
	abulary. Intelligence very defective.	Paralyses, usually of the spastic variety, may be present in the extremities.		Imbecility, (1088).	749a
	the vocal organs shows no defects or paralysis. Patients exhibit a rather childish or affected form of speech. Some substitute "W" for "R", others use "TH" for "S", etc. In idioglossia a child ng certain consonants and substitutes others for them; thus apparently invents a new language. This speech usually becomes normal as the child grows older.			Lisping and Lalling and Idioglossia.	750
		Labials cannot be clearly spoken, especially when the paralysis is bilateral.	Other facial muscles are paralysed.	Paralysis of Facial nerve, (703, 928, 1317).	751
	ide, or cannot be protruded at all.	Linguals cannot be clearly spoken. This difficulty is usually temporary. Nasal voice. Liquids may regurgitate through nose.	May be evidence of injury or pressure upon hypoglossus nerve.	Paralysis of Hypoglossus nerve, (706).	752
	lly (unilateral) in phonation.	Nasal voice. Indistinct articulation, which is improved when head is thrown backwards.	May follow diphtheria. Liquids may regurgitate through nose.	Paralysis of Levator Palati.	753
	cal cords not tense in phonation and ottis immobile).	Voice hoarse. In swallowing, larynx is not well closed; so that food enters it, causing cough, dyspnœa and possibly deglutition pneumonia.	May follow diphtheria.	Paralysis of Superior Laryngeal Nerve.	754
	ost all the laryngeal muscles. Cadaextreme abduction). In phonation, cross the median line to reach the	Aphonia, weak cough and snoring breathing if bilateral. Weak, hoarse voice if unilateral. If bilateral the cords are sucked together during inspiration, giving rise to a slight stridor.	 	Tumor or lesion along course of, or in, recurrent laryngeal nerve, especially lesions of the aorta or lungs.	Paralysis of the entire Recurrent Laryngeal Nerve (Recurrens Paralysis).	755
	rico-arytenoid posterior muscle. The on) leaving narrow slit which may	Voice very little altered. If bilateral, there is great inspiratory dyspnœa with stridor.	 	May be a tumor or lesion along the recurrent nerve. May be the first symptom of an entire recurrent paralysis, or one symptom of a more general disease (bulbar paralysis, tabes, multiple sclerosis, etc.).	Paralysis of Crico-Arytenoid Posterior Nerve (Abductor or Posticus Paralysis).	756
	one or both the adductors (thyroIs as well. Aphonia, or voice is weak part of, their course. Edges concave.		  	Evidence of inflammation of the larynx.	Laryngitis.	757
				History of over use of the voice.	Exhaustion.	758
				Hysterical symptoms.	Hysterical Aphonia, (748).	759
	here are aphonia or hoarseness and nasal speech and some dysphagia. There may be respiratory dyspnœa and deglutition pneumonia. If both nerves are slightly affected there may be rapid and irregular cardiac action and slow and irregular respiration; if severely affected, death occurs.			May be evidence of injury or of pressure upon pneumogastric nerve.	Paralysis of Pneumogastric Nerve trunk, (704).	760
				Bulbar symptoms (434) with hemiplegia or paraplegia and exaggerated tendon reflexes. In pseudo-bulbar paralysis (546, 553) the speech is also thick and indistinct.	Bulbar Paralysis, (434, 544, 546, 694, 756, 1150).	761
	as in the mouth (hot potato speech) and frequently changes in pitch. Symptoms of hereditary ataxia.				Friedreich's Hereditary Ataxia, (652, 670, 687, 781).	762
	7). History of syphilis. Lumbar puncture shows globulin and lymphocytosis. Patients are restless and especially irritable and violent, although usually amiable and steadily progressive dementia. Wassermann usually positive.				Apoplectiform and epileptiform Paresis, (134, 177, 579, 675, 895, 1049, 1104, 1216, 1230).	763
	r and breath. Temporary nature. Foolish speech and actions.				Alcoholism, (585, 658, 663, 673, 780, 953, 1030, 1053, 1101, 1107, 1109).	764
	and sensory symptoms. Nystagmus, exaggerated knee-jerk, Babinski. The speech is jerky and somewhat resembles the scanning of verse.				Multiple or Disseminated Sclerosis, (511, 580, 659, 668, 688, 756, 799, 913, 1051).	765
	on of most joints. Attitude is that of bending forward. Mask-like face. Festinating gait. Speech also shows festination at times.				Paralysis Agitans, (612, 677, 800).	766
	e they are finally uttered explosively. Speech is explosive and full of repetition of certain sounds.				Stammering (Anarthria Literalis).	767
	lossus (aphthongia). There is difficulty in relaxing the muscle spasm so that words can be uttered. The same word is repeated several times before others can be spoken. The more the attention				Stuttering (Anarthria Spasmodica or articulative tics).	768

LOCAL PARALYSIS OF PERIPHERAL NERVES

CHART XIII b

Amnesia and Aphasia

Comprising Numbers 739 on left side of Chart
and 769 to 777 on right margin

739

AMNESIA AND APHASIA (221 to 227)

None of these conditions constitutes a disease, but is rather one symptom of a more complex disease. Each is a form of dementia in the broad sense of the term and consists in a loss of general or special memories. See also Anarthria and Dysarthria (738-8).

- Patient is capable of normal speech but exhibits a decided loss of memory.
  - The loss of memory may not be accompanied by any, o of the names of persons is rather common and of no or less distant past, are referred by the memory to t cerebral concussion and compression (1042-3), especi little time immediately previous to the injury and fri
  - Examination of the patient shows a loss of memory, es in old people and in the insane, and is usually associ
- Patient is incapable of normal speech for want of innervation memories of a few or many spoken words.
  - Can express ideas by gestures, but cannot name object when it is spoken to him and can often then pronou one or two words, or even to none (anarthria). Use and often even when it is not. Cannot construct sente sing songs. When his arm is not paralysed patient Can usually read but not aloud. The condition is u
- Patient is incapable of normal speech for want of auditory memories of spoken words.
  - Patient fails to understand more or less of what is said what he repeats. Cannot execute verbal commands, conscious of this mistake even when his attention is
- Patient is incapable of normal speech for want of visual memories of written or printed words.
  - Patient cannot name objects seen, or read written or p Cannot execute written commands, but readily execu Can write from dictation imperfectly, but not at all
- Patient is incapable of normal speech from loss of innervation memories and of auditory memories of spoken words.
  - Patient at a lo called
- Patient is incapable of normal speech from loss of proper associations and of appreciation of the memories concerned in speech.
  - Patient exhibi

AGRAPHIA. Patient's speech is normal, but his writing is abnormal.

- Patient is incapable of writing for want of the necessa condition uncomplicated by motor aphasia.
- Patient omits words in writing, uses the wrong words, n

Broca, in 1861, published a case of motor aphasia with a lesion at the base of the left inferior frontal convolution and thereby laid t

Wernicke (whose studies have contributed greatly to the comprehension of aphasia) divided motor and sensory aphasia into three

1st. Cortical Motor Aphasia, in which the patient is unable to speak, write or read aloud correctly, or to speak or write correc

2nd. Sub-cortical Aphasia, in which the patient can neither speak spontaneously nor from dictation nor read aloud correctly,

3rd. Transcortical Motor Aphasia, in which the patient can neither speak nor write correctly, but can speak and write from

1st. Cortical Sensory Aphasia, in which the patient can speak (with paraphasia) and copy, but can neither write, nor speak,

2nd. Sub-cortical Sensory Aphasia, in which the patient can speak quite perfectly, write, copy, read aloud and understand wr

3rd. Transcortical Sensory Aphasia, in which the patient can speak (with paraphasia) and write (with paragraphia), can copy

Wernicke also recognizes a Conduction Aphasia, in which the patient can speak, write and read and understand correctly, but exhi

Marie considers all forms of aphasia as resulting from a greater or less degree of a general intellectual impairment rather than to lo aphasia with difficulty of articulation (anarthria or dysarthria). Whether he is altogether right in this or not, certainly our conceptions o ideas, neither of which is probably altogether false.

LYSIS OF SYMPTOMS

HASIA AND AGRAPHIA

T OF SYMPTOMS	DIAGNOSIS	
nly by very little, intellectual impairment in other respects. To a certain degree the loss of memory gnostic or prognostic value. "Retroactive amnesia" is where events, which occurred in the more immediate past, as in Korsakoff's psychosis (1100). "Retrograde amnesia" occurs in some cases of those associated with fright. In it, memory is lost of those events which occurred during some	Amnesia.	769
ially for recent events, impaired judgment and a general failure of mental powers. Very common l with mental depression.	Dementia (1077).	770
ll, or at all. Can use verbs better than nouns and proper names. Recognizes the desired word it. In speaking, the patient is frequently at a loss for a word. His vocabulary is limited often to wrong word (paraphasia—775) but is often conscious of his mistake if his attention is called to it correctly (agrammatismus), but can often repeat sequences of numbers, days, months, etc., and can usually write from copy, but makes many mistakes in spontaneous writing (paragraphia—777). ly associated with right-sided hemiplegia in right-handed persons and vice-versa.	Motor Aphasia or Aphemia (221, 1390).	771
him. Cannot repeat what is said to him, or if in rare cases he can do this, he does not understand t readily executes written ones. In speaking, the patient frequently uses a wrong word and is not d to it. Can write spontaneously and from copy but not from dictation. He can read well.	Sensory Aphasia. Auditory Aphasia. Word Deafness (222, 1345).	772
ed letters or words, but may at times recognize and name objects which he touches and feels. verbal ones. In speaking, patients rarely use a wrong word and are conscious of their mistakes. n copy and make many mistakes in spontaneous writing. Cannot read what they have written.	Visual or Optic Aphasia. Alexia. Word Blindness (228, 1391).	773
neither name objects nor understand words spoken to him. In speaking, patient is frequently or a word or uses a wrong one and is then unconscious of his mistake, even when attention is t. He may or may not be able to read and writing is impossible or very defective.	Mixed Aphasia (224).	774
s words in speaking, uses the wrong word, puts words in a wrong place in the sentence and coherent, even jargon speech.	Paraphasia (225).	775
nervation memories. His arm and hand are not paralyzed for other movements. A very rare	Agraphia (227, 1389).	776
s up words in the sentences so that writing becomes incoherent.	Paragraphia (226).	777

foundation, not only of the modern ideas about the faculty of speech, but also of cerebral localization.

divisions each:

rom dictation, or to read with full understanding, but can copy correctly and understands what is said to him.

t can read, write and understand what is said to him.

tation, can copy, can read aloud, and can understand speech and writing.

copy from dictation, nor read aloud perfectly, nor understand speech or writing.

g, but cannot speak or write from dictation, nor understand speech.

rite, and speak from dictation, and read aloud, but all without understanding, and cannot understand either speech or writing.

paraphasia and paragraphia.

erebral lesions, especially not to those of the left inferior frontal convolution. He considers motor aphasia to be a combination of sensory hasia previously to Marie's article had been growing too schematic. The truth probably lies somewhere between Marie's and Wernicke's

CHART XIII c

Disorders of Gait; Ataxic, Paralytic and Flaccid, Paralytic and Spastic Gaits

Comprising Numbers 736 to 742 on left side of Chart
and 780 to 800 on right margin

(Note)—In addition to the diseases mentioned in this chart, pain, whether in the joints (rheumatism, gout, arthritis, morbus coxae, etc.), or in the muscles (rheumatism, myositis, etc.), or in the bones (caries, etc.), or in the nerves (sciatica, etc.) will cause a limping gait. The gait of a patient suffering from weakened arches in the feet is very characteristic in advanced cases and this common disease should always be thought of in any disturbance of gait and in any painful affection of the legs or lower back.

DIAGNOSTIC SYMPTOMS AND TESTS

ABSTRACT

726 DISORDERS OF GAIT.

740 Ataxia. (Incoordination is the most prominent symptom.)

- Staggering Gait. (Reeling gait.)
  - The disorder is of a temporary nature. Patient's speech is blurred and foolish. Marked ment
  - Disease of permanent nature. Patient sways from side to side and lurches like a drunken man. The ataxia is almost entirely limited to walking and standing.
    - There is a strong heredity and disease occurs in family groups and in youth. Nystagmus.
      - Occurs befor
      - Occurs after optic atro
    - No heredity. Occurs at any age. Retraction o
- Incoordinated Gait. (Stamping gait.) Patient does not walk like a drunken man, but throws his legs about in an awkward and exces manner. All movements of legs are ataxic. In well marked cases legs are raised high, fl outwards and forwards excessively and brought back and down to ground with hard stamp heel. The eyes are employed to control the movements and walking in the dark is very perfect or impossible.

741 Paralytic and flaccid. (Weakness is the most prominent symptom.)

- Waddling Gait. Muscular atrophy and pseudo-hypertrophy.
  - In walking patient throws body from side to side lik weak. In rising patient pushes himself up with h tumors.)
  - Muscles normal. Similar walk. Congenital. Usually bilateral but ma of hip and absence of acetabulum.
- High-stepping Gait. General weakness, especially of extensors. Bilateral. May be some ataxia in the walk. Mu
  - Weakness of extensors only. Bilateral. Blue line on gums. Wrist-drop as well as foot-drop
  - Variable distribution. Weakness, especially of extensors. Often unilateral. Muscular atrop
- Feet drag over ground. In walking all muscles of legs seem too weak to raise feet. No tremor or spasm. Steps short.
  - Temporary condition following illness. Organic and
  - Permanent condition. Organic and peripheral reflexes disease.
  - Advanced Age, atheromatous arteries. Loss of memo
- Inability to stand on one or both feet. Hysterical symptoms present. Lack of will power. Knee-jerk may be increased. Ankle-clonus usually, Babinski always absent.
  - Both legs. Legs can be moved freely and normally when lyi walk or has forgotten how to walk.
  - One leg. The weak leg is drawn along after the strong one strength in leg than would be necessary for wa (Schüller's side gait).

742 Paralytic and spastic. (Stiffness is the most prominent symptom.)

- Toes scrape along ground. Legs rigid and frequently tremble. Tendon reflexes increased. Ankle-clonus and Babinski present.
  - Unilateral. The weak leg is usually swung forwards and outw leg is usually strongly extended at the knee and laterally) along a line the patient moves well to
  - Bilateral. The legs are rigid and offer resistance to forward walking, body and shoulders must often be be legs forwards. Legs frequently show trembling forward. Thighs are adducted so that knees a even crossed in walking (scissors gait). Pernici of severe, anemia may be present.
- General rigidity. Patient is slightly bent forwards and all his joints slightly flexed. Festin ency to stagger backwards. Passive tremor.

OF GAIT

SYMPTOMS	DIAGNOSIS	
disorder and history of alcoholic abuse.	Alcoholic Intoxication (658, 663, 673, 764).	780
uberty. Knee-jerks usually present. Contracture and deformity of feet. Babinski present.	Friederich's or Hereditary Ataxia (652, 670, 687, 762).	781
iberty but in youth. Knee-jerks usually present and exaggerated. Oculo-motor paralysis and .	Marie's or Hereditary, Cerebellar Ataxia (651, 669).	782
ad, cerebellar fits and other cerebellar symptoms may be present.	Lesions of Cerebellum or its tracts (609-10, 648, 686, 1016, 1272).	783
Knee-jerks abolished. Argyll-Robertson's phenomenon, optic atrophy. History of syphilis usually. A common disease.	Tabes (661, 756, 827, 894, 979, 987, 1004, 1217, 1231). (Figs. 24-7.)	784
Knee-jerks are usually present. May be no other symptoms than ataxia and anesthesia, or may be all the spinal symptoms of locomotor ataxia, but none of the crania, especially no eye symptoms. A rare disease.	Lesions of posterior columns of spinal cord (654a, 1347, 1350-1, 1396). (Figs. 24-6.)	785
a duck. Marked lordosis. Atrophy of some muscles, apparent hypertrophy of others, but all are ands and crawls up his own legs. (A similar gait is seen at times in pregnancy and in abdominal	Muscular Dystrophies (477, 1152).	786
e unilateral. No change in the muscles. Hip joints unusually mobile. X-ray shows dislocation	Congenital Dislocation of the Hip.	786a
lar weakness, tenderness and atrophy. Knee-jerks absent. Many sensory symptoms.	Multiple Neuritis (488, 662, 823, 1008, 1147, 1307).	787
History of colic and of exposure to lead.	Lead Palsy (494, 1050).	788
without tenderness. Electrical reaction of degeneration. No sensory symptoms.	Acute Anterior Poliomyelitis (495, 1148, 1233). (Figs. 24-7.)	789
pheral reflexes normal. No sensory paralysis.	Weakness (671).	790
ordered (lost). Sensory paralysis. Patients, even with crutches, are rarely able to walk in this	Myelitis or Myelomalacia in lumbar enlargement of cord (485, 825).	791
and mental impairment. Reflexes normal or increased.	Senile Paraplegia (522).	791a
r sitting Patient apparently makes no effort to walk. Legs collapse. Apparently is afraid to	Astasia and Abasia (287, 653).	792
t never advances beyond it. In some actions when taken unawares the patient shows more g. In walking sideways (stepping laterally) along a line patient moves badly in each direction	Hysterical Hemiplegia or Monoplegia (1074).	793
ls about the normal leg as a pivot and is set down in advance of this latter (mowing gait). The whole side of the body is rigid and swings forward as a whole. In walking sideways (stepping ds the paralysed side, but badly towards the healthy side (Schuller's side gait).	Organic Hemiplegia or Monoplegia. (Apoplexy, Cerebral or Spinal, Tumor or Abscess.)	794
Organic reflexes are disordered, and sensory symptoms are present. No ataxia.	Myelitis or Myelomalacia above lumbar enlargement, including Compression Myelitis (517-20, 829). (Figs. 24-7.)	795
ovements; so that, in ar backwards to pull lonus) when brought held tight together or , or some other form — Organic reflexes may or may not be disordered, sensory symptoms. Marked ataxia.	Ataxic Paraplegia (526, 660). (Figs. 24-7).	796
Organic reflexes not disordered. No sensory symptoms. No ataxia. — Adult.	Spastic Paraplegia (525), including Amyotrophic Lateral Sclerosis (547).	797
Youth. Scissors Gait.	Cerebral Diplegia (478, 501, 577, 1048).	798
Dissociation of sensation (365).	Syringomyelia. (552, 693, 837-9, 1009, 1150a, 1170, 1187, 1357-9).	798a
Intention tremor, marked ataxia, at times staggering gait.	Disseminated or Multiple Sclerosis (511, 580, 659, 668, 688, 755, 765, 913, 1051).	799
on and propulsion—a tendency to go forward at ever increasing speed; also retropulsion—a tend-	Paralysis Agitans (612, 677, 766).	800

CHART XIV

Disorders of Sensation

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DISORDERS OF GENERAL SENSATION AND OF THE SPECIAL SENSES

Symptom Analysed	Alterations in Sensation		
804 Disorders of Sensation. (See also Perversion of Sensation Chart XV).	805 Diminution of Sensation.	810 Anesthesia and Analgesia.	See Chart XIV a.
		811 Dissociation of Sensation.	
		812 Loss of Muscle Sense.	
	806 Exaggeration of Sensation.	813 Hyperesthesia.	
	807 Disturbances of Vision.	814 Perversion.	See Chart XIV b.
		815 Limitation of field of vision.	
		816 Double vision.	See Chart XIV c.
		817 Conjugate Deviation of Eyeballs.	
		818 Pupillary Abnormalities.	See Chart XIV d.
		819 Ophthalmoscopic Examination.	
	808 Disturbances of Hearing.	820 Deafness (anakusia).	See Chart XIV e.
	809 Disturbances of Taste and Smell.	821 Hyperakusia (oxyakoia) or Parakusia.	

CHART XIV a

Disorders of Sensation

Comprising Numbers 805 to 812 on left side of Chart
and 822 to 841 on right margin

805 DIMINUTION OF CUTANEOUS SENSIBILITY (345).

- 810 ANESTHESIA usually combined with some ANALGESIA and THERMIC ANESTHESIA, especially in severe cases of the disease (348-50). (Figs. 26, 33).
  - Tendon reflexes diminished or absent (lesion of peripheral sensory neurons—472).
    - Organic reflexes normal (300). (Figs. 24-6).
      - The
      - And
      - The
    - Organic reflexes disordered (300). (Figs. 24-7.)
      - Ass d
      - No fi
  - Tendon reflexes normal or exaggerated in arms or legs or both (lesion of central sensory neurons—473).
    - Organic reflexes disordered; it may be only slightly (300). (Figs. 19-30.)
      - Ass l
      - Ass
      - No
    - Organic reflexes usually normal, very rarely disordered (300).
      - Ass mot
      - Ass hyst tom
  - Tendon reflexes absent in arms; exaggerated in legs. Lesion both of peripheral and of central sensory neurons.
    - Organic reflexes slightly disordered (300).
      - Ass di
- 811 ANALGESIA and THERMIC ANESTHESIA with little or no TACTILE ANESTHESIA (DISSOCIATION OF SENSATION) (365).
  - Tendon reflexes usually exaggerated in legs (473). Organic reflexes little or not at all disordered (300).
    - Arms affected.
      - Pain of V
    - Legs affected.
      - Pain in re
    - Motor paralysis and hyper opposite side of the body
- 812 AKINESTHESIA.
  - Loss of muscle sense is usually associated with ataxia and an parietal cortex.
- 812a NUMBNESS.
  - Unilateral numbness of hand and foot, steadily progressive, Bilateral numbness is of no diagnostic importance. It may

806 EXAGGERATION OF CUTANEOUS SENSIBILITY.

- Hyperesthesia and hyperalgesia are of little or no diagnostic myelitis.

ANESTHESIA AND ANALGESIA

ABSTRACT OF SYMPTOMS

ABSTRACT OF SYMPTOMS	DIAGNOSIS	
esthesia corresponds to the distribution of a nerve or to that of one of its branches, though usually less extensive. In case of spinal nerves there is also a paralysis of motion, more ss pronounced, in the distribution of the nerve (Figs. 33-38).	Neuritis or Nerve Injury (489, 492, 941, 1146-7, 1173, 1301-7).	822
esia, pain and muscular paralysis, tenderness and atrophy widespread and symmetrical in the distribution of spinal nerves. Usually a history of alcoholic abuse.	Multiple Neuritis (488, 662, 787, 1008, 1147, 1307).	823
esthesia corresponds to the distribution of a nerve root, but is less extensive. Central symptoms often present (Figs. 33-38).	Lesion of Posterior Nerve Root or of Spinal Segment (1302).	824
ted with flaccid paralysis, muscular atrophy and trophic disturbances in legs. Bladder empty and sling. Incontinence of feces. Bedsores. — Symptoms bilateral. Acute or sub-acute.	Myelitis or Myelomalacia in Lumbar Enlargement (485, 791, 1309).	825
— Symptoms mainly unilateral, at least at first. Very slow progressive course.	Tumor in Lumbar Enlargement (486, 1309).	826
tor paralysis, but marked ataxia and loss of muscle sense. Romberg's symptom, Argyll-Robertson's pupil. Tabetic cuirass. Retardation of conduction of pain. Optic atrophy ent. Ulnar hypesthesia and paresthesiae.	Tabes. Locomotor Ataxia (661, 755, 784, 894, 979, 987, 1004, 1217, 1231).	827
ted with spastic paralysis, without muscular atrophy, in arms and legs, or in legs alone. Bi- al anesthesia bounded above by a zone of hyperesthesia. — Spastic paralysis in both arms and legs. Priapism. Disturbances of respiration.	Myelitis or Myelomalacia in Upper Cervical Region (513-4).	828
— Spastic paralysis in both legs.	Myelitis or Myelomalacia in Dorsal Region (517-8).	829
ted with paralysis of cranial nerves, ataxia, symptoms unilateral, at least in early stages, dysarthria and dysphagia.	Lesion in Brain Stem (535, 653).	830
tor paralysis, anesthesia limited to anal and genital region and vicinity. Incontinence of urine and feces. Impotence. Reflexes in legs normal.	Lesion of conus terminalis of Spinal Cord.	830a
ted with ymptoms. — Symptoms bilateral and mainly irritative. — Motor spasm (retraction of neck and opisthotonus) and convulsions. Acute onset with fever. Kernig's sign. Lumbar puncture shows globulin and increase of cellular elements in cerebro-spinal fluid. Herpes facialis.	Cerebral Meningitis (508, 590, 608, 1032, 1045, 1208-9, 1228-9).	831
— Symptoms unilateral. Mainly paralytic. — Acute onset. — Motor paralysis, which may be temporary. Often hemianopia. Usually ataxia and loss of muscle sense. Cerebral symptoms. Post-hemiplegic motor disorders.	Cerebral Hemorrhage or Softening (504, 588, 832, 856-7, 1043, 1060-4).	832
— Chronic onset. — Usually motor paralysis. Convulsions, local or general. Jacksonian epilepsy (587, 605). Mental inertia and impairment. Choked disc or optic neuritis.	Cerebral Tumor (507, 536-42, 578, 587, 833, 849, 855, 861, 892, 908, 960, 1047).	833
ted with al symp- 25). Symptoms usually unilateral. Anesthesia usually in form of hemianesthesia, which may be transferred in some cases. Anesthesia often bounded by a prominent anatomical landmark. The anesthesia is usually unknown to the patient and is discovered upon physical examination, but when discovered is usually more complete than that present in cases of organic disease. The anesthesia is not real. The patient can button clothes, etc., with anesthetic hands without looking. No evidence of any organic disease.	Hysterical Anesthesia (1074).	834
ted with flaccid paralysis and muscular atrophy in arms, with spastic paralysis in legs. Bladder dded and dribbling. Constipation. Pupils are unequal often. — Symptoms bilateral and acute or sub-acute.	Myelitis or Myelomalacia in Cervical Enlargement (549-50, 1310).	835
— Symptoms mainly unilateral, at least at first. Very slow progressive course.	Tumor in Cervical Enlargement (551, 1310).	836
nd paresthesiae in arms and hands are prominent symptoms. Motor weakness and tremor of arms. Muscular atrophy, with fibrillation ands and arms in late stages. Scoliosis and kyphosis in neck and upper dorsal region. Spastic symptoms in legs. Organic reflexes normal. Tropic disturbances and mutilation in hands. chronic course.	Syringomyelia or Morvan's Disease (cervical type) (552, 633, 1033, 1150a, 1170, 1187, 1357-9). (Figs. 24-7.)	837
tabetic-like) and paresthesiae in legs and feet. Legs exhibit a steadily asing paralysis, which may be unilateral in the early stages. Organic es are more or less disordered. Chronic or sub-acute course. — Reflexes increased spasm predominates over paralysis in early stage. Babinski usually on both sides, but in unequal degree. Organic reflexes slightly disordered.	Syringomyelia or Central Gliosis in Dorsal Region. (552).	838
— Reflexes early abolished. Tropic disturbances in legs. Organic reflexes early and greatly disordered.	Syringomyelia in Lumbar Enlargement. (552).	839
nesia on one side of the body and extremities; analgesia and thermic anesthesia and at times also tactile anesthesia on the d extremities. Much girdle pain and spinal epilepsy are common symptoms.	Brown-Sequard Paralysis (442, 509, 981). (Figs. 24-5.)	840
hesia. It occurs in multiple neuritis, tabes, and in lesions of posterior columns of spinal cord, of brain stem, of posterior third of posterior limb of internal capsule and of the		
h slowly progressive mental dulness is suggestive of cerebral tumor. So much so that "choked disc" and other symptoms of cerebral tumor should be sought for in such cases. either of nervous, or of vascular, origin.	Numbness.	841
alue, with the exception of the zone of hyperesthesia, limiting above the anesthesia in transverse myelitis or myelomalacia. In such cases it marks the upper limit of the		

CHART XIV b

Disturbances of Vision

Comprising Numbers 807, 814, 815 on left side of Chart
and 842 to 866 on right margin

807 DISTURBANCES OF VISION

814 PERVERSION.

- A yellow color of all objects seen irrespective of their true color; xanthopsia (yello
- A red color (erythropsia) of all objects seen irrespective of their true color (red vis
- A green color of all objects seen irrespective of their true color (green vision).
- Muscae volitantes, twisted threads and irregular spots moving about in field of vi
- Flashes of light and dark spots surrounded by a bright zone (glittering scotomata)
- Achromatopsia (364) and hemichromatopsia occur in slight lesions of the geniculate
- An inversion (red having a larger field than the blue—14) and an interlacing, of the color fields (Dyschromatopsia).
  - Hysterical symptoms (425) are present.
  - Choked disc and other symptoms of br creased intra-cranial pressure is relie

815 ABSENCE OR LIMITATION OF FIELD OF VISION (358 to 364).

- Blindness (358, 1318). No lesion within orbit.
  - Bilateral. No lesion in eye. Pupillary reflexes no
  - Unilateral or Bilateral.
    - No lesion in eye. Optic neuritis may b
    - No lesion in eye. No optic neuritis. shown that the blindness is not real.
- Homonymous Tetartanopia or Quadrant Hemianopia. No hemiopic pupillary reflex. No hem or other paralysis. May or may not disc. Very rarely occurs in lesions of or optic fasciculus of opposite side.
- Homonymous hemianopia (14, 362, 1321). May very rarely be bilateral, due to double lesion.
  - No hemianesthesia. No hemiopic pup (26). No othe
  - Hemianesthesia.
  - May or may not be a hemiplegia. pupillary reflex. Paralysis of moto abducens nerve or both.
- Bitemporal hemianopia (362, 1319).
- Nasal hemianopia (362, 1320).
  - Slow onset, progressive course, terminat in complete blindness. Choked disc. pupillary reflex.
- Horizontal hemianopia. Occurs in lesions of the retina, or of opt
- Homonymous scotomata. These may occur as the result of small neighborhood of the calcarine fissure.
- Concentric limitation of field of vision, even to complete blindness.
  - Increased tension of eyeball. Excavati Cuppi
  - No increased tension of eyeball. On opht
  - Hysterical symptoms (425) are present.
  - Symptoms of tabes are present, especiall no ataxia. History of Syphilis. Glob

OF VISION

SYMPTOMS		DIAGNOSIS	
ision).		Jaundice, or Santonin, Amyl Nitrite, Cannabis Indica or Picric Acid Poisoning.	842
).		Neurasthenia, Hysteria, great emotional excitement and after cataract operations; also after the eye has been exposed for a long time to an electric or other bright light (snow-blindness).	843
		Diseases of optic nerve and retina and after cataract operations.	844
. Seen especially when eyes are turned towards a bright light.		Neurasthenia, circulatory disturbances in brain and digestive disturbances.	845
ddenly appearing and disappearing in the field of vision.		Migraine, and Aura of Epilepsy, and circulatory disturbances in brain.	846
lies, of the optic fasciculus and especially of the calcarine cortex.		Achromatopsia	847
		Hysteria (1074).	848
disease are present. The color field becomes normal after the in- (Cushing.)		Cerebral Tumor (833).	849
. Uremic amaurosis may be in this class (edema).		Lesion or edema of both occipital lobes.	850
esent. Pupillary reflexes absent.		Lesion of optic nerve or chiasm.	851
llary reflexes normal. Hysterical symptoms. By tests it may be		Hysterical Amblyopia	851a
esthesia choked ic tract	Upper homonymous quadrant of each field of vision.	Lesion of lower lip of contralateral calcarine fissure.	852
	Lower homonymous quadrant of each field of vision.	Lesion of upper lip of contralateral calcarine fissure.	853
y reflex ralysis.	Sudden onset and of short duration. Often more marked in, or limited to, one eye. No other symptoms except nervousness. Circulatory disturbances.	Aura of migraine.	854
	Choked disc. Slow onset. Progressive course of the disease.	Tumor involving median surface of contralateral occipital lobe or fasciculus of Gratiolet (1364).	855
	No choked disc. Rapid onset. Permanent, not progressive, or rarely shows a regressive course.	Hemorrhage or softening in or near contralateral calcarine fissure or optic fasciculus of Gratiolet (1364).	856
	No hemiopic pupillary reflex. No choked disc. Regressive course.	Hemorrhage or softening in the posterior part of posterior limb of contralateral internal capsule.	857
emiopic culi or	Choked disc. Slow onset. Progressive course.	Tumor involving contralateral optic tract or geniculate bodies (893, 1321).	858
	No choked disc. Rapid onset. Symptoms of meningitis may be present.	Neuritis or lesion of contralateral optic tract (893, 1321).	859
usually emiopic	Bilateral.	Tumor compressing central part of optic chiasm (892, 1319). (Enlarged pituitary.)	860
	Unilateral	Tumor compressing homolateral outer part of optic chiasm (892, 1320).	861
erve or chiasm, involving their upper or lower portion.		Horizontal Hemianopia	862
ns in the geniculate bodies, in the optic fasciculus or in the		Homonymous Scotomata	863
and final atrophy of optic nerve. Pupils dilated and unequal. f disc.		Glaucoma (943).	864
hoscopic examination the optic papilla shows atrophy.		Optic atrophy. (898.)	865
		Hysteria (1074).	866
gyll-Robertson's phenomenon and absenc eof reflex. Little or and lymphocytosis in cerebro-spinal fluid.		Tabes (661).	867

CHART XIV c

Disturbances of Vision

DIAGNOSTIC ANALYSIS OF SYMPTOMS

807 DISTURBANCES OF VISION

816 Double vision. Diplopia (383-4). (Fig. 18)

817 Conjugate deviation of eyeballs.

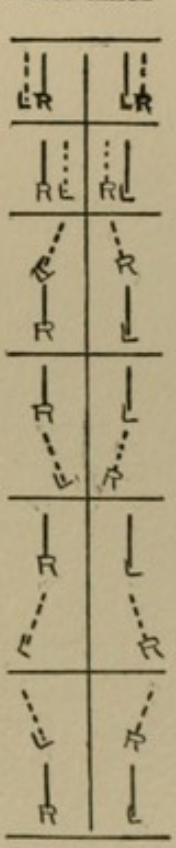
Character of the Diplopia	Secondary Deviation of Sound Eye (29)	Displacement of Visual Axis (28)	Limitation of Motion	Position of False Image (See 28)	Graphic Representation of the Diplopia. Broken Line is the False Image	Diagnosis
BINOCULAR — The images separate and come together again when the eyeballs are turned from one side to the other, or upward or downward and back again.	Inward.	Inward. Strabismus convergens.	Outward.	On the same side as the affected eye.		PARALYSIS OF — External Rectus. 870
	Outward.	Outward. Strabismus divergens.	Inward.	On the opposite side to the affected eye.		Internal Rectus. 871
	Upward.	Downward. Strabismus deorsum vergens, slightly divergens.	Upward and somewhat inward.	Above and on opposite side to the affected eye, image tilted top inward.		Superior Rectus. 872
	Downward	Upward. Strabismus sursum vergens, slightly divergens.	Downward and somewhat inward.	Below and on opposite side to the affected eye, image tilted top outward.		Inferior Rectus. 873
	Downward and inward.	None or slightly upward and inward. Strabismus sursum vergens, slightly convergens.	Rotation downward and somewhat outward.	Below and on same side as the affected eye, image tilted top inward.		Superior Oblique. 874
	Upward and inward.	None or slightly downward and inward. Strabismus deorsum vergens, slightly convergens.	Rotation upward and somewhat outward.	Above and on same side as the affected eye, image tilted top outward.		Inferior Oblique. 875

Fig. 14

The images do not separate and come together again as eyeballs are turned.	Absent	May be variable.	The limitation of motion and the position of the false image are the reverse of those in paralysis. There may be present some irritation, especially in the nose or teeth, which would cause a reflex spasm. The spasm is usually more transient than a paralysis. The muscles usually affected are the internal rectus and the inferior oblique.		Spasm of the ocular muscles. 876
The whole eyeball can be seen to be displaced.					Displacement of eyeball. 877
MONOCULAR — No changes visible in eye.			Hysterical symptoms (425) are present.		Hysterical diplopia. 878
MONOCULAR — Changes visible in eye.			Two openings can be seen in pupil.		Double pupillary opening. 879
			By oblique illumination the lens can be seen to be opaque in patches.		Cataract. 880
			Examination shows dislocation of lens.		Dislocation of lens. 881
			Examination shows astigmatism and an irregular contour of the cornea.		Irregularities of cornea. 882
Associated with other symptoms of lesions in the pons. Eyes turned away from the side of the lesion. Deviation is usually not present when the eyeballs are at rest. A vertical deviation of the eyeballs occurs very rarely. It is associated with a lesion of the corpora quadrigemina. (1271).				Lesion near the anterior portion of the pons (cephalad) to the abducens nucleus, involving posterior longitudinal bundle.	883
Associated with other symptoms of lesions of the brain above the pons. — Eyes turned to the side of the lesion.				Paralytic lesion in almost any part of brain, especially, in posterior part of frontal lobe.	884
Associated with other symptoms of lesions of the brain above the pons. — Eyes turned away from the side of the lesion.				Irritative lesion in cerebral cortex.	885

CHART XIV d

Abnormalities of Pupil and Optic Papilla

Comprising Numbers 818, 819, 890, 891, 897 and 898 on left side of Chart
and 890 to 914 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

ABSTRACT OF S

818 ABNORMALITY OF PUPIL

- Disordered pupillary reflex to light and accommodation (330-1). Mydriasis, myosis or unequal pupils (339-41). — These phenomena occur in too
- 890 The hemiopic pupillary reflex. (26).
  - Bitemporal hemianopia (362, 1319). — Choked disc. Symptoms prog
  - Homonymous hemianopia (362, 1321). — Often hemiplegia or paralysis history of syphilis. Very ra
- 891 The Argyll-Robertson's phenomenon (447). — History of syphilis. Lymphocytosis in cerebro-spinal fluid. Positive Wassermann.
  - Ataxia. Absence of knee-jerk.
  - Mental impairment. Blurred s
  - Rarely occurs. No ataxia. Kn

819 ABNORMALITY OF PAPILLA.

Result of Ophthalmoscopic Examination.

- 897 Optic neuritis. Choked disc.
  - Bilateral.
    - Retinitis. — No marked symptoms of cerebral disease.
      - Albumen and casts in urine.
      - Sugar in urine and in blood.
      - Lead in urine.
      - Examination of the blood shows a condition of severe anemia.
      - Urine and blood normal.
    - No retinitis. — Marked cerebral symptoms.
      - Well marked history of traumatis
      - Increased size of head, and font
      - Retraction of head. Cerebro-sp
      - General convulsion of Jacksonia lepsy is common. May be paralysis. Reflexes usuall creased.
  - Unilateral. — Local inflammation can usually be made out by examining the eye and
- 898 Optic atrophy.
  - Bilateral.
    - Secondary. — It may be the terminal stage of a neuritis and Traces of the active inflammation (old hemorr
    - Primary. No signs of a former inflammation.
      - Old age. Usually atheromatous arteries and hig
      - Loss of knee-jerk. Myosis. Lightning pains.
      - Unequal pupils. Impairment of speech. Tremo
      - Childishness.
      - Characteristic tremor or other symptoms of this
  - Unilateral. — Local inflammation or lesion can usually be made out on careful exam

SIS OF SYMPTOMS

OPTIC NEURITIS AND ATROPHY

PTOMS	DIAGNOSIS	
any conditions to be of much diagnostic importance. ussed in Chart Vb.		
ive, terminating in blindness. Often associated with acromegaly.	Tumor compressing the optic chiasm (851, 860-1, 1319-20).	892
ranial nerves. Optic neuritis or symptoms of meningitis. At times a a quadrant hemianopia in partial lesions of the geniculate bodies.	Lesion of contralateral optic tract or geniculate bodies (858-9, 1321).	893
ightning pains. Girdle sensation and tabetic cuirass.	Tabes (661, 827). (Figs. 24-7.)	894
ch. Apraxia. Restlessness. Childishness. Uncontrollable.	Paresis (1104).	895
rks present. May be no mental impairment. Normal speech. No apraxia.	Syphilis (1205).	896
Headaches, especially in morning. Usually edema of some part of body. Dyspnoea on exertion and loss of strength.	Bright's Disease.	899
Progressive emaciation and loss of strength. Great thirst and polyuria. Large appetite. Dry skin.	Diabetes Mellitus (1175).	900
Blue line on gums. History of lead colic. Wrist-drop. History of exposure to lead poison.	Lead Poisoning (494, 576, 584, 788, 988, 1050).	901
Dyspnoea on exertion and progressive weakness. Pallor of skin and mucous membranes.	Anemia or Leukemia.	902
History of syphilis. Argyll-Robertson's pupillary reflex. Lymphocytosis in cerebro-spinal fluid. Positive Wassermann.	Syphilis (1205).	903
which the nerve has been injured. Usually complicated with facial paralysis.	Injury.	904
es and sutures open in the young.	Hydrocephalus (960).	905
lymphocytosis. Fever.	Meningitis (590, 608).	906
May or may not be fever. At times a latent period. Primary suppuration of bones of skull or elsewhere. Optic neuritis present in about 53% of cases.	Cerebral Abscess or Sinus Thrombosis (508).	907
No fever. Usually steady progression. Optic neuritis present in about 80% of all cases; almost invariably present in tumors in the posterior fossa. Tumors in pituitary gland, corpus callosum and in the central convolutions, especially extra-cerebral tumors, often show no optic neuritis.	Cerebral Tumor (507, 578).	908
it.		
e follow any of the causes of neuritis mentioned above. s and exudates, etc.) can usually be seen.	Terminal stage of Optic Neuritis (865).	909
terial tension.	Senile Optic Atrophy.	910
der disturbance.	Tabes (827). (Figs. 24-7.)	911
Mental impairment. Restlessness. Unreasonableness.	Paresis (1104).	912
se can usually be made out on careful examination.	Disseminated Sclerosis (668).	913
ion.	Disease of the eyeball and orbit (1322).	914

CHART XIV e

Abnormalities of Hearing, Taste, and Smell

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Diagnostic Symptoms and Tests						Abstract of Symptoms	Diagnosis	
808 DISORDERS OF HEARING	820 DEAFNESS AND AKUSIA (355)	WORDS AND SOUNDS	Usually unilateral. May be bilateral. A permanent .. symptom.	Bone conduction impaired.	No facial paralysis.	Severe paroxysmal vertigo and tinnitus aurium.	Ménière's or Labyrinth disease (650, 685, 1019).	918
						No vertigo. May be heredity. Locomotor ataxia or disseminated sclerosis may be present.	Atrophy of auditory nerve.	919
				Bone conduction not impaired.	May be associated with facial paralysis.	May be history of syphilis, symptoms of meningitis, symptoms of tumor at base, optic neuritis, etc.	Tumor or inflammation involving auditory nerve trunk.	920
						Disease of, or injury to, middle or outer ear; cerumen.	Lesion of ear.	921
		SOUNDS	Usually bilateral. Very rarely unilateral, and then only a transitory symptom.			Associated with symptoms of lesion of the pons or crura cerebri.	Bilateral lesion of the lemniscus. (Fig. 20.)	922
						Associated with symptoms of lesion of the cerebral cortex. Complete deafness does not always occur in a bilateral lesion of the temporal cortex.	Lesion of the temporal cortex on both sides. (Fig. 15.)	923
						Hysterical symptoms (425). No symptom of organic disease.	Hysterical deafness (1074).	924
		WORDS ONLY. Sensory aphasia (222) is present.					Lesion of left superior temporal convolution. (Fig. 15.)	925
	821 HYPERAKUSIA, OXYAKOIA OR PARAKUSIA (372, 389).					Hysterical symptoms are present.	Hysteria (1074).	926
						Inflammatory lesions of ear or its neighborhood are present.	Hyperemia of inner ear.	927
						Facial paralysis is present. Low notes are especially painful. Tinnitus aurium is present.	Facial paralysis (1317).	928
809 DISORDERS OF SMELL AND TASTE.	Very little, if any, diagnostic significance can be attached to disturbances of smell and taste.							

CHART XV

Perversion of Sensation: Pain and Vertigo

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DISORDERS OF SENSATION—PERVERSION

Symptoms Analysed		Location of Pain	
930 PERVERSION OF SENSATION IN NERVOUS DISEASES (306).	931 PAIN (330).	933 PAIN IN NERVE. Pain limited to the trunk and branches of one nerve in any part of the body, except that at the height of the attack, there may be a mild radiation of the pain into corresponding nerve of opposite side or into adjacent nerves.	See Chart XV a.
		934 PAIN IN HEAD. HEADACHES IN NERVOUS DISEASE. After a careful examination with suitable instruments has proved the absence of glaucoma, iritis, muscular insufficiencies and other diseases of the eye, of the nose and its sinuses, of the teeth, of the ear, of the scalp (rheumatism), or of the cranial bones (periostitis, caries).	
		935 PAIN IN TRUNK IN NERVOUS DISEASE. After a careful examination has proved the absence of Pott's disease, rheumatism of spine or trunk muscles, arthritis, disease of breast, pericarditis, pleurisy, aneurism, pleurodynia, periostitis, cancer and other tumors, colic (intestinal, uterine, biliary, renal) dyspepsia, pancreatitis, appendicitis, peritonitis, gastric ulcer, gastritis, enteritis, hernia, floating kidney, tubal pregnancy, pelvic inflammation, intestinal obstruction, etc.	See Chart XV b.
		936 PAIN IN EXTREMITIES IN NERVOUS DISEASE. After a careful examination has proved the absence of any disease of the bones, muscles, joints, blood vessels or skin of the arms and legs. Flat foot must be excluded.	See Chart XV c.
	932 VERTIGO		See Chart XV d.

CHART XV a

Pain in Nerve, Pain in the Head, Headache

Comprising Numbers 933 and 934 on the left side of Chart
and 937 to 966 on the right margin

933 PAIN IN NERVE

The differential diagnosis between neuritis and neuralgia cannot always be made clinically. The diagnosis is aided by the experience that certain nerves, such as the sciatic, are more prone to neuritis; while others, such as the trigeminal, are more prone to neuralgia. (Figs. 33, 38).

Paroxysmal pain with free intervals.	Never any motor paralysis or persistent anesthesia or loss of reflexes.	May be some v	
		of the hair.	
		or neurastheni	
Continuous pain with exacerbations.	May be motor paralysis or anesthesia or loss of reflexes or all combined.	May be both v	disturbances
		the electrical	
		tion. General	

934 PAIN IN THE HEAD

APYREXIA

LOCALIZED PAIN

A history of neurotic heredity or other evidence of a neuropathic predisposition, congenital or acquired, is common. Pain is unilateral and is increased by movement and by exposure to cold or wind, and is sometimes associated with muscle spasm. Vasomotor and trophic disturbances are often present.	Pain limited to the whole or a portion of the trunk and distribution of the trigeminal or occipital nerves. Diseases of the eye, the nose and its sinuses, the teeth, the ear, the scalp and the bones must first be excluded. (For the diagnosis between neuritis and neuralgia see 933.)	The pain is felt above the eye in the forehead, in the ten If tension of eyeball be increased, examine eye for glauc
		The pain is felt below the eye in the cheek and side of nc
		The pain is felt in the lower jaw and its teeth and gums :
		The pain is felt in two or three of the situations describe
		The pain is momentary in duration and is associated with
		The pain is felt in the occipital region running up along o and early symptom in neurasthenia and nervous breako
	Pain strictly limited to one-half the head.	Periodical attacks (often occuring at menstrual epoch) of crania angio-paralytica) or pallor and dilated pupils (h there are vomiting and nausea. The disease usually c
	Pain as if nail was being driven through the skull.	Pain of great intensity in a small spot anywher
Pain, nocturnal, in small area and spreading.	History or other evidence of syphilis.	Pain may be felt at any time but is worse in evening or Cranium is often tender at points. Pain may be due t
Pain localized in small area.	Disease exists in organs within the head or body.	Frontal headache may be due to gastric dyspepsia and co to pelvic disease. These referred pains are associated v

DIFFUSE PAIN

Evidence of poisoning.	Exogenetic.	Occurs after the ingestion of narcotics. Does not come on
		Occurs as the result of breathing for hours foul air in unve
	Auto-genetic.	Occurs as the result of constipation, especially where the b
		Occurs in Bright's disease, usually is worse when patient
Evidence of circulatory disorder.	Cerebral hyperemia.	Headache with fulness and throbbing in head, aggrava aches may be followed by a cerebral hemorrhage. Hea
	Cerebral anemia	Headache, most commonly at vertex, with fainting. Da In this as in other forms of headache several etiologica
Evidence of nervous exhaustion	Headache associated with phobias and tremors and insomnia and other symptoms pressure within the skull, especially pressure in occipital and cervical region. H
Evidence of serious brain disease.	Optic neuritis or choked disc.	Progressive symptoms, motor or sensory or both, first of i cussion over the seat of the lesion. Lumbar puncture sl
	May follow traumatism.	Intractable, incurable, more or less constant headaches. stretching of the dura mater by tumor, hydrocephalus,
Chronic headache. Pain constant with exacerbations.	Evidences of rheumatism elsewhere.	Diffuse pain and tenderness of scalp. Pain on movement
PYREXIA. Evidence of infection.	Headache.	Temporary. Occurs during the first few days or first
		Permanent. Occurs throughout the disease and is a
		Permanent. Suppuration elsewhere in head or body
HYPERPYREXIA. Evidence of exposure to high temperature.		History of exposure to high temperature. Headache oft

ABSTRACT OF SYMPTOMS			DIAGNOSIS	
-motor but no trophic disturbances, except rarely a slow blanching ver any electrical reaction of degeneration. Patient usually anemic		Certain points on the nerve are usually tender (points of Valleix). Frequently the parts supplied by the nerve are hyperesthetic and local spasms occur. The neuralgia may be only one symptom of a more general disease (symptomatic neuralgia) or independent of any other disease (idiopathic neuralgia).	Neuralgia.	937
motor and trophic there may also be ction of degenera- alth usually good.	Nerve neither swollen nor tender.	A tumor may be felt or rarely seen with X-ray on nerve.	Neuroma, (491).	938
		A tumor or a displaced bone or other substance may be felt or seen with X-ray near, and compressing, the nerve.	Compression Neuritis.	939
		Rash of herpes limited to distribution of nerve.	Herpetic Neuritis.	940
	Nerve wherever it can be felt is swollen and tender. There may be an inflammatory focus near to and involving the nerve.		Neuritis, (489, 492, 822).	941
and as far back as the vertex. It is most severe along the nerve trunk but extends also beyond it and on each side. The tender point is at the supra-orbital notch. The eyeball may be painful and tender. (864).			Supra-orbital Neuralgia or Neuritis.	943
nnd radiates into the teeth of the upper jaw. The tender point is at the infra-orbital foramen.			Infra-orbital Neuralgia or Neuritis.	944
l in the side of tongue, in the ear and in the temporal region The tender points are at the mental foramen and in the temple.			Infra-maxillary Neuralgia or Neuritis.	945
bove.			Trigeminal Neuralgia or Neuritis.	946
			Trigeminal Neuralgia or Prosopalgia.	942
clonic or a series of clonic spasms of a facial muscle.			Tic Douloureux (602).	947
ide of the scalp to the vertex. The neck is stiff. The tender points are behind the mastoid process, behind the middle of the sterno-cleido-mastoid muscle and on the parietal eminence. This is a common m. In many cases the pain is dull and is a sense of strong pressure rather than pain.			Occipital Neuralgia or Neuritis.	948
ere pain, sometimes on one side, sometimes on the other side of the cranium. Skin is very hyperalgesic and vaso-motor disturbances, either in the form of flushing, sweating and contracted pupils (hemi- crania angio-spastica) are often present. Often ushered in by visual hallucinations in the form of flashes of light, etc., or by paralytic phenomena, such as hemianopia. Towards the end of the attack nences in early life, ceases in old age and often shows a direct inheritance. "Symptomatic" migraine is not infrequent in tabes, paresis, brain tumor and epilepsy.			Migraine or Hemicrania. Idiopathic and symptomatic, (846, 854, 1028).	949
n scalp with the feeling as if a nail was being driven through the skull at this point. This region is tender. Hysterical symptoms (425) are present.			Clavus. Hysteria, (1074).	950
nt, or occurs only at night, or in the early morning hours. It follows no nerve distribution but is felt over a small area and extends over a wider and wider circle. Argyll-Robertson's phenomenon is frequent. periostitis. Lumbar puncture may show lymphocytosis or positive Wassermann. Optic neuritis may be present.			Syphilitic Neuralgia (meningitis).	951
igation, as well as to disease of the eye or caries of teeth. Occipital headache and temporal headache may be due to disease of the eye, teeth, pharynx or ear. Vertex headache may be due to anemia or n hyperalgesia of skin of same region, which may be as constant and important a symptom as is the pain. The pain of eye strain grows worse towards evening, or follows reading.			Referred Pains or Symptomatic Neuralgia, (374).	952
mediately, often not till the next day.			Alcoholic or Morphine Headache.	953
lated rooms, especially if patient is accustomed to pure air. Transitory.			Foul Air Headache.	954
els usually act freely. This headache is usually most marked in the frontal region.			Constipation Headache.	955
t wakes up in the morning. Urine is usually scanty and contains albumen and casts. Edema and gastric disturbances are common. Albuminuric retinitis is often present.			Uremic Headache.	956
by cough. The congestion may be active (after taking amyl nitrite) or passive (heart disease). Vertigo and vomiting may be present. High arterial tension. Tinnitus aurium. A series of such head- hes occurring at the time of puberty or of menstruation may well be congestive.			Congestive and High Tension Headache.	957
ess before eyes. Cold hands and feet. Cardiac or arterial disease present. A series of such headaches may be followed by a cerebral thrombosis. The headache may be relieved by the recumbent posture. actors may be present.			Anemic Headache.	958
neurasthenia. Pain grows less towards evening and is usually felt in the occiput or vertex. Feeling as if a tight band or cap were upon the head (casque neurasthenique). Often a sense of fulness and aches resulting from overstrain (mental or physical, especially eye strain) may well be of this nature.			Neurasthenic Headache, (1072).	959
ation, later of paralysis. In case of abscess there may be a latent period and, in the active stage, fever. Headache is constant with intense exacerbations. In rare cases the skull may be tender on per- s greatly increased pressure of cerebro-spinal fluid. Such headaches can be relieved by the operation of "decompression."			Cerebral Tumor, or Abscess, or Hydrocephalus, (507, 508, 965).	960
cause can be assigned. May be some mental deterioration or other cerebral symptoms. No optic neuritis. Lumbar puncture will usually show lymphocytosis. Apopiectic attacks may occur. Irritation or Fever may be present in rare sub-acute cases.			Pachymeningitis and Chronic Meningitis, (588, 590).	961
fronto-occipital muscle. Relieved often by application of warmth. At times small tender nodules can be felt, and hence the condition has been called "indurative headache."			Rheumatism of Scalp.	962
eek of almost any fever (typhoid). Such headaches may be due to the action of the toxic substance directly upon the brain or indirectly by causing vaso-paralytic congestion.			Infection or Toxic Headache.	963
ciated with muscle spasm and paralysis and local edema of the scalp (sinus thrombosis). Coma. Lumbar puncture will show lymphocytosis and globulin in cerebro-spinal fluid.			Acute Meningitis and Sinus Thrombosis, (500, 1044).	964
Latent stage. Convulsions and coma.			Cerebral Abscess (960).	965
followed by coma and convulsions.			Sunstroke, (589, 1068).	966

CHART XV b

Pain in Trunk

Comprising Numbers 935 on left side of Chart
and 970 to 990 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

935
PAIN IN TRUNK IN NERVOUS DISEASES.

- Pain in back.
  - Evidence of neurotic temperament. No evidence of organic disease.
    - Pain and tenderness of spinous processes.
      - Phobias and nervous exhaustion, pain and sense of pressure most m
      - Hysterical symptoms (425). Much tenderness of spinous processes,
    - Pain and tenderness of coccyx. — Severe pain in coccyx without evidence of any disease of it. Pain i symptoms (425) are present.
  - Evidence of organic disease.
    - Pain, tenderness and rigidity of spine.
      - May follow traumatism.
        - Severe and constant pain in back and radiating about body and i Much spasm of spinal muscles. Exaggerated reflexes. Little or n if any, it is of a transitory nature. Hyperes hesia and hyperalges
        - Slowly increasing motor and sensory symptoms, irritative and paraly paralytic symptoms are more prominent, the tumor is in the cord. and pain radiating into extremities than in meningitis usually.
- Pain in thorax and abdomen.
  - Girdle pain (374).
    - Vertebral column is ankylosed. — It may be possible to feel exostoses on vertebrae. Unilateral or bil other parts of the body. X-ray examination makes the diagnosis p
    - Unilateral.
      - No other symptoms. — Pain shoots around chest, following the course nerve, or may be limited to a small area of the pericarditis, pneumonia, pleurodynia, periosti been excluded by a careful examination.
    - Bilateral usually.
      - Many other symptoms.
        - Loss of knee-jerk. Argyll-Robertson's phenom litic infection.
        - There is a zone of hyperesthesia where the gir severe.
      - No other symptoms. — History or other evidence of syphilis. Lumba
    - At first unilateral and later bilateral. — Slowly increasing motor and sensory symptoms
  - Local pain.
    - In mammary gland.
      - Hysterical symptoms. — Paroxysmal attacks of pain in one mammary g can be detected. Pain is usually in the left
    - In precordia and arm.
      - Old age. Arterial disease. — Paroxysmal attacks of pain in precordia shooti region, of suffocation and impending death.
      - Any age. No arterial disease. — Pain similar to the above, but no arterial disea tobacco, overwork, etc.
    - Along attachment of diaphragm. — Pain felt in lower anterior part of chest, also in same side of neck, Tender points are along the attachment of the diaphragm and bel tremely rare disease.
    - In abdomen. In all these rare forms of neuralgia organic abdominal disease must be carefully and thoroughly excluded.
      - Paroxysmal attacks of pain in epigastrium often occurring at the sa ach or neighboring viscera, especially no gall stones. May be ass
      - Similar paroxysmal attacks of severe pain, occurring irregularly at p
      - Paroxysmal attacks of severe pain in abdomen occurring with some Pain relieved by pressure. Blue line on edge of gums, wrist-drop
      - Pain in hip, groin, hypogastrium and genitals. Tender points near
    - In genitals. — Neuralgic pains and irritability in the pelvic viscera, the bladder, rec pains at times occur during years in one testicle or one labium

PAIN IN TRUNK

BSTRACT OF SYMPTOMS	DIAGNOSIS	
ed in cervical spine and occiput.	Neurasthenia (1072).	970
cially in mid-dorsal region; ovarian tenderness is also common.	Hysteria. Spinal Neuralgia (1074).	971
ased by motion, touch, defecation, etc. In most cases there is a history of injury. Often hysterical	Coccygodynia.	972
extremities. aralysis, and — Injury. Very sudden onset. Lumbar puncture may show bloody fluid. Retention of urine.	Hematorrhachis (524).	973
extremities. aralysis, and — History of infection (septic, syphilis, etc.). Lumbar puncture shows globulin and increase of cellular elements in cerebro-spinal fluid.	Meningitis Spinalis, acute (febrile) and chronic (afebrile) (608, 1005).	974
(paraplegia dolorosa). When irritative symptoms are very prominent the tumor is meningeal, when ymptoms at first usually unilateral, later bilateral. Less pain and spasm in back, more girdle pain	Spinal Tumor (509, 826, 836-40, 981, 1006). (Figs. 24-7.)	975
al girdle pains at level of the disease. Rarely any paralytic symptoms. Usually bone lesions in ive.	Spondylitis Deformans. Arthritis Deformans.	976
n intercostal rve; pleurisy, etc., having — Tender points of Valleix: one, two inches from posterior median line; another, two inches from anterior median line; and a third, in mid-axillary line. Other points on nerve may also be hyperalgesic. Pain is paroxysmal. Respiration, cough, sneezing, etc., are painful.	Intercostal Neuralgia.	977
n intercostal rve; pleurisy, etc., having — Rash of herpetic vesicles along course of nerve.	Herpetic Neuritis (940).	978
n. Lumbar puncture gives lymphocytosis. Ataxia. Lightning pains in legs. History of syphi-	Tabes (827, 661). (Fig. 27.)	979
pain is and below a bilateral anesthesia, which may be slight, and a motor paralysis, which may be	Transverse Myelitis (Figs. 24-7)	980
uncture gives lymphocytosis. Pains worse at night.	Syphilitic Meningitis.	980a
first irritative, later paralytic. Brown-Séquard's paralysis at first (442).	Spinal Tumor (975).	981
l, and, at times, radiating beyond the limits of the breast. No tumor or other disease of the gland st.	Mastodynia.	982
p to left shoulder and even down left arm and, at times, both arms. Sense of oppression in sternal rial tension is usually high. Area of cardiac dulness usually increased.	Angina-Pectoris.	983
Neurotic individual who has an overstrained heart. At times the result of gastric indigestion,	Pseudo-Angina-Pectoris.	984
t frequently on left side. Breathing, sneezing, coughing, etc., painful. Pain occurs in paroxysms. sterno-cleido-mastoid muscle. No signs of pulmonary, pleural, cardiac or other disease. An ex-	Phrenic Neuralgia.	985
hour, especially in the early morning. No digestive disturbances or evidence of any disease of stom- ted with contraction of the empty stomach and consequent feeling of hunger.	Gastralgia.	986
rus or neck of bladder or anus, associated with symptoms of tabes (661).	Tabetic Crises (433, 827).	987
odicity, when biliary, renal and other forms of colic, appendicitis, diverticulitis, etc. have been excluded. ad in urine after administration of K. I.	Enteralgia (Lead Colic, etc.).	988
e, on crest of ilium, inner part of groin, etc.	Lumbo-abdominal Neuralgia.	989
a, uterus, vagina and urethra, but these are rare and relatively unimportant conditions. Neuralgic is. From this point the pain may radiate.	Pelvic Neuralgia.	990

CHART XV c

Pain in Extremities

Comprising Numbers 936 on left side of Chart
and 995 to 1012 on right margin

936
PAIN IN EXTREMITIES IN NERVOUS DISEASES.

DIAGNOSTIC SYMPTOMS AND TESTS			ABSTRACT
		Pain in arm.	Pain radiates along one or all of the nerves of the arm. Te other points where nerves are superficial. Vaso-motor di paralysis; but movements of arm are impaired by the pain. pressure on nerves, must be carefully excluded.
	Unilateral. Any of these forms of neuritis may be associated with, or precede, or follow a rash of herpes: herpetic neuritis. (Figs. 33, 38).	Pain limited to the trunk and distribution of the sciatic, anterior crural or obturator nerve.	Pain shooting along the trunk, or over small areas in the distr but the pain may prevent motion. Patient holds knee of the opposite side and bears his weight on the healthy leg. major (trochanteric point) and in popliteal space (poplitea may be decided muscular weakness and atrophy. Sciatica tion for any possible pressure upon the nerve should alway
			Pain along the trunk and distribution of the anterior crural ankle. Tender points on anterior aspect of the hip joint, paralysed and atrophied and knee-jerk lost and anesthesia be secondary to diabetes and injury. There may be an er
			Pain along inner side of thigh, along course of obturator ner neuralgia and is usually associated with paralysis of the a
		Pain limited to outer surface of thigh.	Pain is associated with paresthesiae (especially numbness an The paresthesiae are more characteristic of this disease tha
		Pain in a joint.	Pain in a joint, usually the knee-joint, increased on motion. dence of any disease of the joint. Many hysterical symp
		Pain at insertion of Achilles tendon.	Severe pain at insertion of Achilles' tendon on walking and s
		Pain in heel	Pain in lower surface of heel, especially when walking or sta removal of the sub-calcaneal bursa, or of exostoses, others
		Pain in toe.	Pain in the metatarso-phalangeal joint, especially of the fourt is lowered from "breaking" of the arch transversely.
	Bilateral.	With girdle pains, and lumbar puncture gives lymphocytosis.	With Romberg's symptom, Argyll-Robertson's phenomenon, lymphocytosis and lightning pains over small areas in legs,
			With pain and rigidity in back and in extremities. Exagger
		With anesthesia.	Steadily progressive motor and sensory symptoms, at first m Brown-Sequard's paralysis (442).
			Motor paralysis and anesthesia over whole of both legs, exce peripheral and organic reflexes. Muscular atrophy and tr in lower back and radiating into legs.
		With dissociation of sensation.	Motor and sensory paralysis commencing at the distal end o and tenderness. The disease usually commences with pain
			Pain and paresthesiae, analgesia and thermic anesthesia wit symptoms are usually limited to arms with symptoms of s are milder.
		With vaso-motor disturbances.	Extreme pain in soles of feet associated with redness and swell foot must be excluded.
			Pallor and coldness of fingers and toes followed by cyanosis In extreme cases a larger or smaller slough forms and is c
		With fat.	Marked increase in fat, either diffuse or in separate tumors, i with it, and the fatty masses are tender, especially in the ea

TREMITIES

SYMPTOMS	DIAGNOSIS	
r points in supra-clavicular fossa, in axilla at head of radius and at rbances. Fibrillary contractions at times occur. There is no motor umors at base of neck and in axilla, and a cervical rib (556), causing	Cervico-brachial Neuralgia or Neuritis of Ulnar, Median Radial, etc.	995
ion of the sciatic nerve. Little, if any, anesthesia or motor paralysis. affected side semi-flexed, thigh slightly abducted, inclines his body to der points over the sciatic notch (gluteal point), above the trochanter oint). In neuritis, the nerve, wherever felt, is tender, and then there much more frequently a neuritis than a neuralgia. A rectal examina- e made.	Sciatica (720).	996
ve on the anterior surface of the thigh and inner surface of leg to the r side of knee and at internal malleolus. Extensors of thigh may be be on anterior surface of thigh and inner side of leg in neuritis. May on of herpes along the course of the nerve.	Crural Neuralgia or Neuritis.	997
after hernia and other diseases have been excluded. A rare form of ctors.	Obturator Neuralgia.	998
gling) and is frequently associated with, and is caused by, flat foot. the pain, which is often entirely absent.	Meralgia Paresthetica.	999
he skin is much more sensitive than the articular surfaces. No evi- s (425).	Arthralgia or Hysterical Joint.	1000
ding. May follow gonorrhea, malaria, gout, broken arches or injury.	Achillodynia.	1001
g. Some cases are cured by rheumatic medicine, others by surgical supporting the weakened arches.	Talagia or Calcanodynia.	1002
e, usually following an injury. Usually occurs in women. The joint	Metatarsalgia or Morton's Toe.	1003
ia, history of syphilis usually, always loss of knee-jerk, cerebro-spinal erficial and deep, often followed by hyperalgesia over same area.	Tabes. Neuralgic stage (661).	1004
l reflexes. No ataxia. No Argyll-Robertson's phenomenon.	Spinal Meningitis (608, 974).	1005
unilateral, later bilateral. Increased pressure of cerebro-spinal fluid.	Spinal Tumor (509, 826, 836-40, 975).	1006
n some cases the domain of the anterior crural nerves. Abolition of e disturbances. Anesthesia in perineum and genitals and much pain	Lesions of Cauda Equina (487). (Fig. 29.)	1007
tremities and extending towards body. Muscular weakness, atrophy paresthesiae in toes and fingers and often with fever.	Multiple Neuritis (488).	1008
t tactile anesthesia. Trophic disturbances and mutilations. These e paraplegia in legs. The pains often resemble the pains of tabes, but	Syringomyelia (552, 693, 837, 1150a, 1170, 1187, 1357-9).	1009
and later with pallor, shrinking and wrinkling of the same parts. Flat	Erythromelagia (1198).	1010
congestion; so that fingers and toes become purplish and even black. ff.	Raynaud's Disease (1195).	1011
rms and legs, but not elsewhere. There is considerable pain associated tages when they are forming.	Adiposis Dolorosa. Dercum's Disease (1176).	1012

CHART XV d

Vertigo

Comprising Numbers 932 on left side of Chart
and 1015 to 1033 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS			ABSTRACT
VERTIGO (392)	Motor Ataxia is present.		In these cases the vertigo is not a prominent symptom. In some of falling and fears that he will fall and experiences some verti lesions in the brain stem and elsewhere. The diagnosis is made
	Cerebellar Ataxia is present.		Any disease of the cerebellum, especially tumors, may cause vertig the hemispheres. The diagnosis is made from the absence of p and, in tumors, the optic neuritis and failure of sight.
	Crossed Paralysis.		Lesions of the brain stem may involve the tracts from the cerebell made by the motor or sensory paralysis or both, which occur i paralysis in the domain of the cranial nerves (crossed paralysis
	Vertigo and movement of head.		Cysts and tumors suspended free in the fourth ventricle cause int the diagnosis is extremely difficult or impossible. The vertigo n held. Choked disc is common.
	Deafness and symptoms of aural disease.		A steadily, progressive deafness of one ear associated with tinnitu may throw patient to the ground. Raising the head from the g or loss of bone conduction and loss of power of hearing high no the paroxysmal attacks. Suppurative and other diseases of the the ear is completely deaf, but then may commence in the othe cause vertigo by affecting the semi-circular canals or vesti laesa). It is difficult to draw the line between these cases of au all these conditions. Strictly speaking, Ménière's disease appl flammation of the labyrinth causing vertigo is called Voltoni's
	Diplopia and symptoms of ocular disease		Double vision and weakness of ocular muscles and eye strain may is relieved by closing the defective eye, even when it is not caus
	Symptoms of circulatory disturbances.	Position and moving.	When patient's head is bent down for a long time and then is sudd vertigo. A blow on the head will cause vertigo, probably in c back of head or moving head quickly may cause vertigo. A s to the head.
		Exhaustion.	Great weakness, especially in the convalescence from disease, is a
		Digestive disorders.	When, in consequence of the congestion due to digestive disorder are anemic. These digestive disorders may also produce abno diagnosis is made by the presence of the digestive disorder and
		Cardiac and hemic disease.	In all forms of cardiac disease the brain may receive an insufficien frequent in aortic disease. The diagnosis is made from the pre to the altered quality than quantity of the blood supply (1029)
		Atheromatous arteries.	Atheromatous arteries interfere with the normal blood supply bot cause vertigo. This is especially common in elderly people. T usually, an increased arterial tension.
		High blood tension.	Fulness of head, headache, mental confusion, tinnitus auriu tension.
		Apoplexy.	Vertigo is a common initial symptom of apoplexy of all forms (cere rhage) and may be the only symptom of a slight attack. Usua
		Epilepsy.	Vertigo may constitute the aura which may or may not be followe In some cases a severe subjective sensation of vertigo, frequently Vertigo is a not uncommon symptom in the interval between t
		Migraine.	Vertigo may be the initial symptom or may accompany an attack makes the diagnosis plain.
	Toxic.		Abnormal conditions of the blood, as in the early stages of the in Various toxic substances, such as tobacco, alcohol, coffee, morphia, of the cerebral or cerebellar cortex. The diagnosis is made by the A disease endemic in Switzerland and occurring only in men work ness of vision, ptosis, often diplopia without strabismus, and a Pain in back of neck. Attack lasts a few minutes.
	Symptoms of cerebral disease (headache, etc)..	Organic.	In addition to apoplexy, any irritation of the meninges (tumors, lo ciated with severe vertigo, especially on change of position. T pressure on the cerebellum, or, when situated in the frontal lob is made by the numerous other symptoms of these diseases: co with the vertigo, which is less severe in the recumbent posture
		Functional.	Vertigo is a not uncommon symptom in those functional nervous such as neurasthenia, the traumatic neuroses and hysteria. The vertigo is never very severe and often resembles rather syncop

OF SYMPTOMS	DIAGNOSIS	
es, in consequence of the incoordination, the patient is in danger while in other cases the vertigo may be the direct result of the m the presence of motor ataxia	Tabes, Disseminated Sclerosis and other disease with ataxia.	1015
which is more permanent in lesions of the vermis than in those of lysis, the presence of cerebellar ataxia, headache, and vomiting	Cerebellar Disease (609-10, 648, 686, 783, 1272).	1016
and cause ataxia and, less frequently, vertigo. The diagnosis is ne form of hemiplegia with increased reflexes, and also of local e). (Figs. 19-22.)	Lesions of the brain stem (460, 535-46, 656, 830, 1268-74, 1301-4, 1375, 1378, 1382-4, 1388, 1398).	1017
e dizziness only when head is moved. Except for this symptom vary greatly in intensity with the position in which the head is	Lesions within the fourth ventricle. (Fig. 19.)	1018
n that ear, and with paroxysmal attacks of severe vertigo which nd may cause vomiting. Attacks vary in severity. Impairment are usually present. Vertigo is usually entirely absent between may be present, but usually are not. Disease usually ceases when r. Almost any disease or functional disturbance of the ear may ar nerve directly or indirectly (aural vertigo or vertigo ab aure vertigo and Méuière's disease, which latter is often used to cover only to cases of hemorrhage into the semi-circular canals. In- ease.	Ménière's Disease. Voltoni's Disease. Aural Vertigo. Vertigo ab aure laesa (650, 685, 918).	1019
use vertigo. Occurs sometimes on railway trains. The vertigo by the diplopia alone.	Ocular Vertigo. Vertigo ab oculo laeso (649).	1020
raised, or when patient's body is rotated rapidly, he experiences equence of vaso-motor reflex disturbance. Lying on one side of ar vertigo may result from the application of a galvanic current	Acute Cerebral Anemia.	1021
mon cause both of vertigo and ataxia.	Exhaustion Vertigo.	1022
he portal circulation is engorged with blood, the cerebral vessels l chemical substances which may produce a toxic vertigo. The he cure of the vertigo when the indigestion is cured.	Acute Cerebral Anemia from digestive disorders, hemorrhage, etc.	1023
d irregular supply of blood and vertigo may result. This is most ce of cardiac disease. In hemic diseases the vertigo is due rather	Chronic Cerebral Anemia from blood and cardiac diseases.	1024
s to amount and as to uniformity of distribution and hence may diagnosis is made from the presence of atheromatous arteries with,	Chronic Cerebral Anemia from atheromatous arteries (syphilis).	1025
palpitation of heart, dyspnoea on exertion, and high blood	Cerebral Congestion.	1025a
l hemorrhage, embolism and thrombosis, and meningeal hemor- the sequence of other symptoms makes the diagnosis clear.	Apoplexy (504).	1026
a full attack. The diagnosis is made from the epileptic attacks. llowed by vomiting, may be the equivalent of an epileptic attack. ttacks, and may continue during minutes or hours.	Epilepsy (575, 1058, 1071).	1027
migraine. The hemicrania, the much more prominent symptom,	Migraine (846, 854, 949).	1028
ious diseases and in leukemia, melanemia, gout, diabetes, etc.	Toxic Vertigo (1024).	1029
nine, etc., will cause vertigo, probably by affecting the circulation of the ingestion of the substances before each attack of vertigo.	Drug Vertigo.	1030
in hot cow stables. It consists in attacks of vertigo, with dim- alysis of some function or act of the arms, simulating hysteria.	Gerlier's Vertigo. Vertige Paralysant.	1031
lesions and especially inflammations and syphilitic lesions) is asso- rs may act both by irritation of the meninges and by transmitted by direct irritation of the cerebro-cerebellar tract. The diagnosis lsions, vomiting, slow pulse, etc., which are frequently associated	Cerebral Meningitis and Tumor (Syphilis) (508, 536-42).	1032
ses which are the result of psychic traumata, acute and chronic, ferential diagnosis of these diseases is made in other charts. This ttacks.	Neurasthenia, Traumatic Neuroses and Hysteria (1072,-5).	1033

CHART XVI

Disorders of Cerebral Activity

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptoms Analysed	Alterations in Mentality	
1036 Disordered Mentality.	1037 Coma.	See Chart XVI a.
	1038 Pseudo-Coma.	See Chart XVI b.
	1039 Double Personality.	
	1040 Weakened Mentality.	
	1041 Insanity.	See Chart XVI c.

CHART XVI a

Coma

Comprising Numbers 1037 on left side of Chart
and 1042 to 1068 on right margin

103. COMA OR SEMI-COMA (205, 745).

See also pseudo-coma, (1038).

History, or other evidence, of recent injury to head.	Convulsions rare.	Pupils usually contracted and respond feebly to light. — Patient may be completely unconscio… toms follow the injury immediately
		Pupils dilated, often unequal, and usually do not respond to light. — Symptoms usually follow the injury … symptoms. Often conjugate devi…
History of a previous brain illness, of which the coma is only one symptom, and often the terminal one, or the presence of an inflammation of the scalp (eryispelas, suppuration), or of the bones of the skull (caries and especially suppuration of the bones of the ear).	Convulsions are frequently present.	The symptoms are those of a local meningitis (507, 1045) or abscess (508, … the coma are common. There may be local symptoms, both irritative a… be edema of eyelids and conjunctivae, choked disc, prominence of the ey…
		Retraction of neck and opisthotonus. Fever, headache, delirium. Convul… tation followed by those of paralysis.
		Headache, vertigo and vomiting. Often mild delirium. A recent, infecte… general and resemble those of a rapidly growing tumor. Choked disc o…
		Headache, vertigo and vomiting. May be a history of former injury. No… tumors at the base are more likely to cause paralysis of one or more cr…
		A motor monoplegia (anarthria, etc.), rare, hemiplegia or diplegia occurrir… defect. Epileptiform convulsions, unilateral or bilateral, are frequent.
		Progressive mental impairment, childishness, restlessness, amiable but irri… syllables and letters are left out and letters doubled. Apoplectiform an…
		History of lead poisoning, of lead colic, of wrist-drop, etc. Blue line on g…
		Intention tremor. Scanning speech. Many motor and sensory symptom…
	No convulsions.	Headache, increasing fever, difficulty in walk and speech, tremor, increasi…
History or other evidence of poisoning.	Convulsions rare.	Patient can usually be aroused from his coma sufficiently to speak and his … alcoholic abuse.
	Convulsions absent.	Pupils are contracted and do not respond to light (if opium). Patient is … other narcotic.
		Patient is in a confined space or room in which there is a strong smell of il…
Evidence of a cardiac inadequacy.	Often a slight spasm or rigidity during the attack.	Sudden attack of coma with pallor and weak or absent cardiac action of sl… extremities cold, restlessness, yawning. Low arterial tension, steadily g…
		Sudden intermission of heart beat during a considerable fraction of a min…
Sudden attack of coma of short duration with or without a convulsion. Usually a history of similar attacks and often of remote injury.	Convulsions almost always present and are usually the most striking symptom of the disease, but not so characteristic as unconsciousness, which is at times the only symptom of the attack.	The attack usually commences with a convulsion, as described in Chart X, 5… sometimes trivial, sometimes a deed of violence (post-epileptic insanity) … sciousness and no convulsion and either no action or some trivial or fool… the convulsive attack and is called the "psychic equivalent" and is alto… and take journeys and are lost to their friends and to themselves. After a… an epileptic attack is the complete or almost complete unconsciousness … some mental weakness which may slowly increase to mild or extreme de… epileptics have attacks during many years and yet show little, or no, …
		The attack is altogether similar to the major attack of epilepsy, but it does… distended abdomen, foul smelling feces, vomiting, diarrhoea, etc., and c…
Sudden attack of unilateral paralysis. Rarely the paralysis comes on slowly; steadily increasing during hours or days, "Ingravescent apoplexy." In such cases the coma may be slight or absent	Convulsions are rare, but both tonic and clonic spasms may occur, involving one-half of the body when the lesion is cortical, or involving both sides of the body when the hemorrhage occurs in the brain stem (460) or ruptures into a ventricle.	The coma comes on instantly or in the course of a few hours. There are stert… in the form of hemiplegia. The bilaterally innervated muscles (upper fa… tory, abdominal, micturition, defecation, etc.) escape permanent paralysis. … reflexes may, during the coma, be abolished, later exaggerated (Rosenba… pass away after several hours or days. The lower branch only of the fa… the paralysed side. Usually there is also at first a sensory hemiplegia w… in which latter case the motor paralysis greatly improves or entirely dis… in leg and finally in the arm. In case recovery does not take place flexc… in leg. Post hemiplegic motor disturbances may occur, especially in h… toms such as aphasia may occur. A reactive inflammation about the les… second week. Repeated attacks at varying intervals are common.
		After uncertain prodromata, coma and paralysis with fever appear and de… encephalitis superior hemorrhagica acuta) or may be in the form of a he…
Albumen and casts, or sugar, or all three, in urine.	No convulsions.	Patient emaciated. Acetone odor of breath. Pulse is small and rapid, ski…
	Convulsions usually.	Onset usually gradual. Some edema, cyanosis, restlessness, rapid noisy res… albumen and casts.
Decided fever. Hyperpyrexia.	Convulsions frequently.	Occurs at the onset of acute infections, especially in children. Often associ… but in these cases convulsions are rare, and the cause may consist in a fa…
		History or evidence of exposure to great heat — Absence of perspiration. T… symptoms (paralytic) occasionally occur

ABSTRACT OF SYMPTOMS	DIAGNOSIS	
r a short time, after which he remains in a dazed condition for a time, or he may be only dazed from the start. Pallor, low blood tension, vertigo and vomiting are common symptoms. May be contusion of scalp. Symp- rely any paralysis. Often retrograde amnesia (769).	Cerebral Concussion.	1042
diately, but not always; there may be a "lucid interval," (especially in extra-dural hemorrhage). Coma, profound stertorous breathing, pulse slow, reflexes abolished, increased tension of cerebro-spinal fluid are the usual of head and eyes. Often paralysis in the form of more or less complete hemiplegia, together with Babinski reflex and some spasm. Often retrograde amnesia (769).	Cerebral Compression (contusion and hemorrhage).	1043
) with usually high fever and chills (both may be absent) occurring in a cachectic, anemic, or infected patient, especially in one with caries of bone of skull (otitis). Headache, vomiting, restlessness and delirium preceding ralytic. A very characteristic symptom is a localized edema of the scalp. In thrombosis of superior longitudinal sinus there may be epistaxis and edema at root of nose. When the cavernous sinus is involved there may and oculo-motor and abducens paralysis. When the transverse sinus is involved there may be edema over mastoid and palpable thrombosis of internal jugular vein in upper part of neck.	Sinus Thrombosis, (964).	1044
and retraction of abdomen. Paralysis of cranial nerves. Kernig's reflex. Lumbar puncture and examination of the fluid, which may be cloudy, give globulin and increased cellular elements in it. Symptoms of cerebral irri-	Meningitis, (590, 608).	1045
nd or other cause, or origin, for suppuration. Moderate, irregular, often absent fever. Course is progressive but may be very acute or extremely chronic and often shows a latent period. The symptoms are both local and rather rarely. The general symptoms are more prominent than the local. Suppuration of the middle ear and of the mastoid cells must be carefully excluded, especially in children (see 508).	Cerebral Abscess, (152, 181, 508, 578, 587, 907, 960).	1046
. Course is progressive. Mental deterioration, but local symptoms are often as prominent as the general. Convulsions, especially Jacksonian epilepsy, are common, especially when tumor is in or near the cortex; while nerves. Choked disc is very common (see 507, 897).	Cerebral Tumor, (151, 507, 536-42, 578, 587, 833, 849, 855, 858, 861, 892, 960).	1047
genitally, or in early infancy are common. Usually some arrest of development of paralysed part and of skull. Little or no muscular atrophy, and reflexes exaggerated. Ankle-clonus and Babinski. Usually some mental athetosis and hemichorea frequently complicate the disease and contractures occur in almost every case.	Cerebral Palsy of Childhood. Porencephaly, (116, 501, 577, 630, 798, 1086).	1048
steadily increasing dementia. Blurred speech. Tremor of lips, tongue and hands. Terminal dementia. Inability to repeat difficult phrases, due partly to paraphasia, partly to loss of memory. Writing imperfect: words, vulsive attacks. Abnormalities of pupil. Argyll-Robertson's reflex. Lumbar puncture gives globulin and lymphocytosis in cerebro-spinal fluid. History of syphilis. Positive Wassermann reaction.	Paresis, (1104).	1049
Convulsions. Lead can be found in the urine, especially after the administration of K. I.	Lead Palsy, (158, 494, 584, 788).	1050
aggerated reflexes. Ataxia. Nystagmus. The convulsive attacks may be epileptiform, apoplectiform or myotonic. Headache, compulsory acts and slight dementia are not uncommon symptoms.	Disseminated Sclerosis, (668).	1051
wsiness, passing into coma and death. Trypanosomes are found in blood and in cerebro-spinal fluid.	Trypanosomiasis or African Lethargy.	1052
is characteristic of intoxication; being indistinct, blurred and foolish. Pupils are dilated and respond to light. Flushed face and conjunctiva, and stertorous respiration. Often tremor or twitching. History or evidence of	Alcoholic Coma, (764).	1053
rowsy, and mentally incapable. Respiration is slow and stertorous. Face is congested. Pulse is at first slow, but later, especially in fatal cases, becomes rapid. History or evidence of patient's having taken morphine or	Narcotism from opium, etc.	1054
ating or coal gas. He is cyanotic with rapid, irregular pulse. He often vomits and exhibits more or less tonic or clonic spasm.	Illuminating or coal gas poisoning.	1055
uration. Often preceded by tinnitus aurium, dimness of vision, cold sweats, and nausea. Slight spasm or rigidity is frequent during the attack. In internal hemorrhage the onset of the coma is more gradual. Pulse is small, g lower.	Syncope. Internal Hemorrhage.	1056
more with coma and slight spasm. Slow pulse, atheromatous arteries. Usually occurs in advanced age.	Stokes-Adams' Disease, (436, 582).	1057
The convulsion lasts only two or three minutes and is accompanied and followed by a coma which gradually passes into a sleep. This post-epileptic coma is sometimes absent and is sometimes replaced by unconscious action, h an attack of general convulsions is called "the major attack" or "le grand mal." At times such an attack follows another immediately throughout a long series (status epilepticus). At other times there is only uncon- tion; unconscious movements of lips and jaws being common. This is called "the minor attack" or "le petit mal." Sometimes the attacks occur only at night (nocturnal epilepsy). At times an attack of insanity replaces similar to the post-epileptic insanity. These attacks may last minutes, hours, or days, and in them the patients lose their former individuality. In some of these attacks (ambulatory automatism) the patients wander about cks there is amnesia. A rare form of epilepsy is one in which the patient falls suddenly to the ground and gets up again immediately without any appreciable loss of consciousness (vertiginous). The essential characteristic of it and the complete or almost complete absence of any memory of it. In the interval between the attacks, which at times extends over months and years, the patient may be entirely normal. Frequently he is irritable and shows a, especially if the attacks are frequent and rest on a strong hereditary basis. This condition is not to be confounded with the transitory mental impairment due to bromide given therapeutically. On the other hand, many l impairment. Epilepsy beginning in a patient over forty years of age should suggest the possibility of a cerebral tumor.	Epilepsy, (110, 126, 430, 575, 846, 1027, 1071, 1083, 1102).	1058
ecur. There is only one attack or a series of attacks at short intervals. It can usually be referred to an irritation of some part of the body or to some form of poisoning. It is often associated with digestive disturbances, when these conditions are removed. It is most common in children.	Eclampsia, (576).	1059
respiration and a slow full pulse. Usually paralysis ocular, masticatory, deglutition, laryngeal, respira- nski reflex usually present from the start. The other ign). Patient may die in coma or the coma may e decidedly paralysed. Tongue protruded towards quickly disappears, but which may be permanent, rs. Improvement usually begins in the face, next atractures appear in arm and extensor contractures Some mental impairment persists. Local symp- ay cause an exacerbation of the symptoms in the	Prodromal symptoms (headache, vertigo, etc.) common. Convulsions, especially Jacksonian epilepsy (431), at onset and later in disease. Hemiplegia disappears quickly and completely.	Pachymeningitis Interna Hemorrhagica, (502, 588). 1060
	Prodromal symptoms may or may not be present. Profound and long coma usually. High arterial tension (associated with cerebral miliary aneurisms). Permanent, or long continued hemiplegia. Aged patient. Presence of interstitial nephritis.	Cerebral Hemorrhage, (146, 411-13, 503, 588, 832, 856-7). 1061
	No prodromata. Youthful patient. Cardiac or pulmonary disease, sepsis or aneurism. Existence of embolism elsewhere in body. Slight or absent coma. Pulse not so slow. Hemiplegia not so permanent. Paralysis of some cortical function; aphasia, hemianopia, etc. Spasms not infrequent.	Cerebral Embolism, (505, 832). 1062
	Prodromal symptoms present. Slight or no coma. Atheromatous arteries or history of syphilis. Paralysis of some cortical function; aphasia, hemianopia, etc. A mild, transitory form, with tendency to relapses and without contracture, is the so-called "lacunar hemiplegia," in which not one large focus, but numerous minute foci of softening occur in the cortical area involved.	Cerebral Thrombosis, (506, 832, 1207). 1063
esults in two or three weeks. The disease usually occurs in young persons and seems to be due to poisoning, especially alcoholic. Optic neuritis may be present. The paralysis may attack the eye muscles (Wernicke's polio- gia, partial or complete.	Acute Multiple Encephalitis, (495, 543-4).	1064
y. Breathing is labored and may show "air hunger." Sugar and almost always albumen and casts also in urine.	Diabetic Coma.	1065
on. Pulse of high tension. History usually of headache, vomiting, dyspnoea and dimness of vision. Ophthalmoscope may show albuminuric retinitis. In rare cases there may be hemiplegia or other paralysis. Urine contains	Uremic Coma, (576, 581, 850, 956)	1066
with acute indigestion with foul smelling feces. High fever is common. Often history of improper food. The coma which occurs in the course of, and especially towards the end of, acute infectious fevers is probably of this nature, circulation (cerebral anemia).	Toxic or Auto-Toxic Coma, (596)	1067
oma comes on rapidly but not instantaneously and is preceded by many prodromata; such as headache, mental confusion, marked disturbances of vision, paresthesiae, weakness, etc. Delirium is a common symptom. Local	Sunstroke or Insolation, (589, 966).	1068

CHART XVI b

Pseudo-coma, Double Personality and Weakened Mentality

Comprising Numbers 1038 to 1040 on left side of Chart
and 1069 to 1075 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS			ABSTRACT
1038 PSEUDO-COMA.	Hysterical symptoms (425).	Convulsions and Spasms frequent.	Occurs usually in girls and women of an emotional t be stopped by a strong and continued pressure o vation will usually show that she is attentive to from external causes, or auto-suggestion (hypnoti
1039 DOUBLE PERSONALITY AND AUTOMATISM (209).	Hysterical symptoms (425). Epileptic symptoms (575).	Convulsions frequent.	Patient seems at times to be in a hypnotic state, o and in that state to lead a life carried on from fo states from auto-suggestion, patients often act like tion and in some hysterical patients may well be d While in an unconscious state patient often perform has no memory. Whether in such unconscious st doubtful. While unconscious, epileptics often pe
1040 WEAKENED MENTALITY. Patients appear to be intelligent, but incapable of long sustained effort, and of self-control, often foolish and unreasonable. The different diseases in these groups merge into each other and no sharp line can be drawn between them. They all rest on a neurasthenic basis, and in all suggestion plays a great part.	Apprehension and various phobias are prominent symptoms. In consequence of their weakened mentality these patients cannot rid themselves, by reasoning, of their unreasonable apprehensions and fears.		The symptoms are those of a general exhaustion of t especially of the lower centers. It is common in easily. Every task looms as a mountain before t will power are both poor. They feel nervous, ir phobia, claustrophobia, etc.). Almost as charact The patient also suffers much from palpitation, tive disturbances. The essential symptoms of ne
	Abnormal and greatly increased suggestibility is the prominent symptom. Symptoms are various, inexplicable and incredible. No certain evidence of any organic disease; although almost every disease can be more or less perfectly simulated (425).		The disease occurs almost exclusively in women and system, are probably all really cerebral and seem adopted by the patient as the result of impressio patients are usually so dominated by the desire t of accomplishing this. Too much reliance cannot, stimuli varies from day to day and is often quite (425). Anesthesiae, paresthesiae, hyperesthesiae, disturbances occur alone or combined, transitory o often has as its cause a psychic trauma, either ac nervousness, theatrical posing, irritability and incr ance of remarkable and startling symptoms of the life. In general these patients show lack of self-c may show wonderful will power or obstinacy. S mata of hysteria;" others occur only rarely. The catalepsy (611), globus hystericus (426), emotion oesophagus, torticollis and other spasms (618), he (527), ovarian tenderness, photophobia, tremor (67 itation, anorexia and fasting, tympanites, phanto fever, flushing, sweating, angio-neurotic edema (1 field of vision, (866) somnambulism (1069), doubl
	The result of an accident.		The disease occurs as the result of traumatism asso injury. It very rarely occurs when a *severe* physi cases where pecuniary compensation may be obta ceiving any compensation. The disease is closely scribed above under hysteria. Tremor, fibrillary paralyses (motor and sensory), palpitation and va are insomnia, especially in the early morning hou symptoms can be simulated, and as many of these scious and unconscious simulation. Simulation, h in "suggestion," as in hysteria.
	All the various forms of insanity described in the next chart exhibit, and are in part dependent		

TY, AND WEAKENED MENTALITY

SYMPTOMS	DIAGNOSIS	
ıre. Eyelids are closed and resist attempts to open them. Coma can usually varies. Even in the apparent coma the patient is suggestible and close obser- surroundings and therefore not truly comatose. Such a condition may result somnambulism, trance).	Hysterical Coma (1074).	1069
an allied condition from auto- or foreign suggestion, or from wilful deception, er similar states quite distinct from the normal life. In the hypnotic or allied omatons. This is a very rare condition and offers much opportunity for decep- o unconscious suggestion on the part of the physician.	Hysteria (1074).	1070
mplicated acts and leads a life, during hours, days or weeks, of which he later he can remember what happened in previous similar states is, to say the least, n automatic acts.	Epilepsy (1058).	1071
ıervous system, especially of the brain, associated with an increased irritability, ı but more so in women. The patients are either incapable of exertion or tire ı; so that they are discouraged before they undertake it. Their memory and ble, apprehensive and have a number of peculiar fears: phobias (235—agora- tic of neurasthenia as are the phobias, are indecision and lack of will power. o-motor disturbances, paresthesiae, headache, backache, neuralgias and diges- sthenia are apprehension and fear (phobias).	Neurasthenia, Psychasthenia (113, 155, 161, 163, 178, 180, 671, 674, 843, 845, 959, 970, 1033).	1072
e neurasthenia and is greatly depressed by reason of an abnormal state of self- rmanently fixed upon the condition of his body or of his mind. Patient is de- disease or abnormality of some organ of his body, generally the viscera, which nedical examination no abnormality can be discovered adequate to justify the patient's mind. These false judgments are very various and are often mon- like an exaggeration of the neurasthenic phobias. The patients are anxious on their ills. The essential symptom of hypochondriasis is a fixed, constant, is diseased.	Hypochondriasis (216).	1073
dren, and the symptoms, which may apparently affect any part of the nervous e imaginary: to be the result of a false idea (delusion—215), or of suggestions ceived from others or from some abnormal sensations within the body. The cite wonder and admiration that they are not very scrupulous in their means refore, be placed on their statements. The reaction of the patients to external ormal in its results. The symptoms of the disease are both many and variable otor paralyses, convulsions, spasms, contractures, vaso-motor and secretory rmanent, producing a confused and constantly varying picture of disease, which or chronic, or more frequently both. In addition to the chronic condition of d suggestibility, the course of the disease is interrupted by the sudden appear- atest intensity, which render the patient helpless and often apparently threaten rol, but in the production and maintenance of some prominent symptom they of these symptoms occur so frequently that they have been called the "stig- t important of these acute hysterical attacks are convulsion (586), coma (1069), attacks of laughing or crying, aphonia (748, 759), mutism (747), stricture of anesthesia and its transference (425, 834), astasia, abasia (653, 792), paralysis spinal irritation, clavus (950), cough, dyspnoea, palpitation, vomiting, regurg- umor, false pregnancy, peritonitis, anuria, polyuria, melanuria, hemorrhages,), blindness (851a), deafness, (924), anosmia, ageusia, concentric limitation of onsciousness (1039), etc.	Hysteria (111, 128, 130, 153, 179, 345, 425-6, 527, 586, 618, 628, 664, 674, 747-8, 759, 793, 834, 843, 848, 866, 878, 924, 926, 950, 971, 1000, 1033, 1069-70, 1075).	1074
ed with great fright, or in some accidents from fright alone without physical injury has been received. It is especially common in railroad accidents and in l for the injury; although it occurs also in cases where there is no hope of re- ied to neurasthenia and hysteria and it may present any of the symptoms de- traction, especially after exertion, vertigo, paresthesiae, neuralgic pains, local notor disturbances are common symptoms. Quite characteristic of the disease and a melancholic, hypochondriacal, mental state. Most, if not all, of these tients are seeking to recover damages, there is naturally more or less of con- ver, is far from explaining the traumatic neuroses, the key to which lies rather	Traumatic Neuroses. Sometimes called Traumatic Hysteria (156, 616, 674, 1033).	1075
on, a weakness of the mental powers, varying in degree, but always decided.		

CHART XVIc

Insanity

Comprising Numbers 1041 on left side of Chart
and 1076 to 1117 on right margin

EXTREME DEFECT IN INTELLIGENCE

1076 Amentia (211). More or less complete

condition in which the mind has not developed with advancing age, due to a disease of the brain, either congenital or acquired in infancy. Besides the mental defect, these patients often present many and various physical defects and deformities such as: deformed skull, posterior hydrocephalus, high palatine arch, coarse body, deformed ears, etc. The amentia may be either general or partial, and some of its slighter degrees may be due in part to defective training.

- Patients show little or no intelligence. Are unable to urine and feces. About their only desire is that Most of these patients exhibit frequent and violent
- Patients can talk and are more or less cleanly in their They are incapable of much education. They can and the sexual instinct is often strongly developed.
- Patients show a degree of intelligence approaching th children develop up to a certain point, but then st ing eyes, thick fissured tongue, and short, stubby fin tobacco. Simple infantilism is often called "atele
- Certain feeble-minded persons seem incapable of app

1077 Dementia (212) More or less complete.

A condition in which the mind has developed to a certain, even a high, degree of intelligence and then, in consequence of disease of the brain (functional or organic), all mental development has not only ceased, but there has been a distinct retrogression, which may go on to a complete loss of intelligence. Memory, emotions and interest are all lost. Patient becomes apathetic, reacts to no stimulation, soils himself and does not even eat.

Occurring in youth, at puberty or before 25 or 30 years.

- Complete apathy, coming on more or less acutely. C tion. Appears to be anesthetic and analgesic. The cases recover after several months.
- Partial apathy. Patients are dull and stupid but cons is an absence of emotions and of inte:est in anythin They perform frequently spontaneous, impulsive, sil of a phrase which they have just heard or spoken (e The varieties under this head merge into each other

Usually occurring in adult life after 25 years, but may occur in youth.

- History of alcoholism and usually associated with mult
- History of alcoholism extending over many years. T amounts of alcohol. The symptoms at times resemb
- History of very numerous epileptic seizures. Gradua
- History of a previous psychosis which has gradually p (apathetic dementia) but some cases show great rest
- History of syphilis. Lumbar puncture shows globuli inability for continuous mental concentration, rec at least cheerfulness, in spite of the illness which pa dementia. No paralysis, but much paresis and esp tabes, more rarely with various forms of spinal scle

Occurring in old age.

Associated with physical weakness and with atheroma loss of memory, especially for recent events, with re Blood tension often high.

1078 Hallucinations are abundant and dominant. Hallucinatory Insanity (213).

A condition in which the patient is constantly receiving false perceptions from his different senses: either visual, auditory, olfactory, gustatory, tactile or painful, or from several or all combined. Associated with this is always a certain degree of impairment of consciousness, which weakens his judgment and does not permit him to decide that these hallucinations are false.

- Patient is overwhelmed by a large nu intentions towards him. He does fo
- History of alcoholism. Patient's hall Great fright. Violent attempt to e
- Many other poisons besides alcohol: produce a mild hallucinatory insanity
- Disease commences with fever, headac

1079 Delusions are present and dominant. Delusional Insanity (215)

A condition in which the patient has formed a false judgment about things which concern him. The basis of these false judgments is partly a congenitally defective brain and partly hallucinations. Associated with these delusions there is always present a varying degree of impairment of intelligence, which prevents the patient from recognizing the falseness of the delusion when evidence is presented to him which would be adequate for a normal man; although many of these patients in their own way reason shrewdly. These delusions lead to irrational conduct on the part of the patient which would not be irrational were the delusions true.

- History of alcoholism. Patient has for men is that their wives are unfaithfu
- Patient has delusions upon which he b is not built upon them. Curable in
- Patient has a number of delusions, u systematized or nearly so but whi are strong enough to influence his co duct and bearing. Curable in most cas
- Patient has many delusions which are and are woven in with the delusions i sions are thus systematized and some himself as a most remarkable person, delusion of persecution (every happe

1080 An exaggerated emotional state is the dominant symptom. Emotional Insanity (204).

- Exaggeration of the sometimes natural feeling of sadness or discouragement with life.
  - Patient is constantly in a depressed and painful frame o by the patient to explain the melancholy: the comm are very prone to suicide. Their circulation and th
- Exaggeration of the natural feeling of joyousness.
  - Patients are constantly in a joyous and excited mood w matic and due to delusions (1111). Mania is divided i
- Alternations of mania and melancholia.
  - Alternations at long intervals of mania and melancholia
  - Attacks of excitement or of depression may recur (recurr Kraepelin has incorporated all of these forms under the te

The insanities of the neuroses have been considered under epilepsy, hysteria and hypochondriasis.

ABSTRACT OF SYMPTOMS

DIAGNOSIS

ak intelligibly, but are often noisy. Many are unable to walk. Usually soil themselves with ood. They can do no act requiring intelligence and are incapable of sustained attention. reaks of anger. Many of them are undersized.

- Associated with hydrocephalus. Fontanelles and sutures are unusually open and broad. Brain often shows a tuberculous sclerosis with nodules.
- Associated with epilepsy.
- Associated with a very small skull. Brain usually weighs less than 1000 grammes, or 33 ounces.
- Occurring in family groups, with blindness and optic atrophy and dark red spot in place of macula lutea.
- Associated with congenital hemiplegia or diplegia and with rigidity and convulsions (501).
- Associated with no special characteristic.

Hydrocephalic Idiocy. 1082

Epileptic Idiocy. 1083
Microcephalic Idiocy. 1084
Amaurotic Idiocy. 1085
Porencephalic Idiocy. 1086
Idiopathic Idiocy. 1087

(1081 Idiocy (743))

ts. They exhibit in their acts an evident though low degree of intelligence. few abstract conceptions and no high moral ideas. Masturbation is common

- All the classes above given under idiocy but with less extreme mental defect.
- Very defective intelligence in general, but in certain directions (usually music or mathematics) may show great skill.
- Associated with destruction or atrophy of the thyroid gland. Arrest of growth. Thick, dry and scaly skin. Coarse features (1164).
- Associated with no special characteristics.

Imbecility 1088
Idiot Savants. 1089
Cretinism. 1090
Idiopathic Imbecility. 1091

(1088 Imbecility.)

rmal but are evidently below the standard. They cannot be educated beyond a certain point. They are often obstinate and of violent temper upon provocation, even though in general amiable. Some nd never advance further (infantilismus). In some cases of infantilismus, there is a defective growth of the body with a small head and a peculiar characteristic Tartar-like expression of the countenance, slant- They possess great faculty of imitation (Kalmuck idiots or mongolismus). This disease presents many resemblances to cretinism (1090, 1164). Infantilism may be due in some cases to poison, such as wine or " Infantilism with premature senility is called "progeria."

Defectives and Infantilism. 1092 The Feeble-Minded 1093

dding the simplest moral ideas, although their mental defect is not so obvious in other directions. On closer examination, however, a general mental defect becomes evident.

Moral Imbecility or Habitual Criminals. 1094

ousness is obscure. Patient incapable of any mental effort. Is in a dream-like state, immobile and does not appreciate what is going on about him. Will answer no questions and will respond to no stimula- e no hallucinations or delusions. The cutaneous reflexes are lost and the tendon reflexes are exaggerated. The course of the disease is interrupted at times by periods of excitement. More than half of such

Primary Dementia and Stupor. 1095

ness is fairly clear and they appreciate what is going on about them. Hallucinations and delusions are common. There Anesthesia and analgesia. They are unwilling to perform any act and offer resistance when forced to do it (negativism). ts. They often repeat the same act or the same words many times (stereotypy). They also often repeat the last word lia), or repeat obscene words (coprolalia), or repeat an act (echopraxia), or remain in any given posture (catalepsy). all show, as prominent symptoms, impulsive ideas and acts. The prognosis is uncertain.

- Simple progressive mental deterioration, at times permanent, often temporary, commencing at puberty.
- Alternating conditions of stupor and excitement, negativism, stereotypy, echolalia with steady mental deterioration, at times permanent, often temporary.
- Hallucinations and partially systematized delusions with steady mental deterioration.

Hebephrenic form. 1096 Adolescent Insanity or the so-called Dementia Precox (a term of very doubtful value). 1097
Catatonic form. 1098
Paranoid form. 1099

neuritis, except in very rare cases. A peculiar loss of memory with a bringing of memories of the remote past up to the immediate past (retroactive amnesia—769), failure to appreciate the relations of time and space.

Korsakow's Psychosis. 1100

r. Dysarthria. Loss of memory and power of attention and mental power. Delusions are frequent. Normal and ethical ideas blunted. Irritable, sentimental and often very susceptible to the effects of small ase of paresis (1104) (pseudo-paresis), but the dementia is less pronounced, the cerebro-spinal fluid is normal and improvement follows abstinence from alcohol.

Alcoholic Dementia, (658, 1107, 1109). 1101

of memory and mental power. Masturbation is common. Usually steadily progressive and incurable, but this dementia may occur in childhood and may be transitory, in which case it may be cured by bromide.

Epileptic Dementia, (575, 1058). 1102

into a condition of apathy and more or less complete loss of intelligence. Patients may carry over into this stage some traces of the emotions and delusions of the former psychosis. They usually sit motionless ess (agitated dementia).

Terminal or Secondary Dementia. 1103

l lymphocytosis in cerebro-spinal fluid. Wassermann usually positive.. Argyll-Robertson's phenomenon and often unequal pupils. Tremor of lips, tongue and hands. Loss of memory, loss of self-restraint, ess and change in character are early symptoms. Speech blurred, slovenly and tremulous. Words, syllables and letters left out in speaking and in writing. Restless, excited and irritable, with exaltation or rarely recognizes as existent. Grandiose delusions, silliness and inconsistency in striking contrast with inordinate pretensions. Gradually increasing physical and especially mental weakness up to complete y apraxia (230, 282). Careless, inconsiderate, slovenly clothes, etc. Apoplectiform and epileptiform seizures are usually present in the course of disease. Incurable, but remissions. Frequently associated with

Paresis. General Paresis. Paralytic Dementia, (134, 177, 416, 419-20, 579, 675, 763, 895, 1049, 1216, 1230). 1104

arteries. The dementia varies from day to day greatly in degree and may lead the patient to do very foolish things (often erotic), while apparently sane. Loss of will power (drunkenness). Depression and n of past memories. Dread of impending poverty. Desire to go home, imagining himself to be in a strange place. Restless at night, associated with hallucinations and delusions. Attacks of excitement.

Senile Dementia. 1105

of hallucinations which cannot be reconciled with his previous experiences. He becomes confused, frightened and timid. Doubts his own identity and that of those about him, and especially doubts their acts which might be rational were his hallucinations true. May be the early stage of other forms of insanity. Probably a form of moderate delirium.

Confusional Insanity. 1106

ions are of all kinds but are usually visual and concern snakes, spiders and other small grotesque and repulsive animals. Tactile hallucinations, shown by fumbling of the fingers, also are usually very evident. from his enemies. Pronounced, continuous tremor and insomnia. Disease usually lasts less than a week and usually terminates in recovery.

Delirium Tremens (1101, 1109). 1107

r exogenetic, such as belladonna, salicylic acid, etc.; or autogenetic, such as uremia, cholestrin, ptomaines; or septic (post-febrile insanity), especially when the poison acts upon an exhausted nervous system, may delirium, which may last a few hours or days or may continue during weeks, months or years.

Toxic, Septic or Post-febrile Insanity. 1107a

lazed feeling and delirium, followed by violent excitement and violent actions. Many hallucinations. Speech is continuous, violent and incoherent. Disease usually terminates fatally in two or three weeks.

Acute Delirium or Delirium Grave 1108

, on the basis of hallucinations, more or less permanent delusions, especially of jealousy and persecution. These are often so exaggerated and monstrous as to be grotesque. A very common delusion in Patients often act violently. Some alcoholic tremor. Curable, and usually lasts less than a month after alcohol has been withheld.

Acute Alcoholic Mania. Alcoholic Hallucinosis. (1101, 1107). 1109

his actions. These delusions are not associated with any decided emotional manifestations and are at times permanent, at times changeable; but they are not reasoned upon shrewdly and a systematic theory t cases.

Simple Delusional Insanity. 1110

These delusions are of a pleasant exhilarating nature impelling to action, to talk and incidently to the disregard of the right of others. These patients are usually violent and dangerous to themselves and others.

Symptomatic or Delusional Mania. 1111

These delusions are of a depressive nature and tend to inhibition of action and speech, to self-abasement and to self-destruction. The patients sometimes seem to be in actual stupor (melancholia cum stupore) or merely profoundly depressed with inhibition of all action (melancholia attonita). At times the intensity of their grief drives them to frenzy and breaks through their inhibition and impels them to deeds of violence (raptus melancholicus). Occasionally there is restlessness (melancholia agitata). All these conditions seem to be the direct result of delusions. Such patients are very prone to suicide.

Symptomatic or Delusional Melancholia. 1112

the result of hallucinations. They are fixed, permanent and are reasoned upon. Newspaper paragraphs on indifferent subjects and various circumstances in the entire life of the patient are more or less distorted attempt at an explanation of the curious things which are happening to the patient. The patients reason very clearly and if their premises are granted it is almost impossible to answer their arguments. The delu- d theory is evolved to explain them. The most trivial happenings, even in the remote past, are fitted into this system in the most surprising and ludicrous way. The general result is that the patient regards ause so many persons busy themselves about his affairs. In typical cases the patient evolves first a delusion of observation (all things, even the most indifferent and remote have reference to him); next, a is to injure him or to try his character), and finally a delusion of grandeur (he must have a great destiny when so many persons seek to injure him or to tempt him).

Paranoia. 1113

nd for which there is no discoverable adequate cause—"a cloud settles over the mind." A pessimist. No hallucinations, no delusions, except such as are secondary to the depressed frame of mind and invented of an unpardonable sin, etc. There is an inhibition of mental and physical activity. Patients withdraw themselves as much as possible from the world. They sit quiet and are pictures of woe. They odily functions are greatly disordered. Curable. The secondary form of melancholia, due to delusions, has been described under 1112.

Primary Melancholia. 1114

ut any discoverable adequate cause. Optimistic. They have no hallucinations or delusions. They are impelled to constant speech and constant activity. They are violent and dangerous. Mania is often sympto- a mild form (maniacal excitation) and a severe form (frenzy). Curable.

Primary Mania. 1115

h usually, but not always, a comparatively normal period between the two. The duration and the intensity of the attacks and the duration of the interval are very varying.

Circular Insanity. 1116

insanity), or may alternate, after a longer or shorter interval (circular insanity). In some cases the excited and depressed phases are commingled, or the alternations are momentary, giving a "mixed form." "maniacal depressive insanity" but the permanence of this term is doubtful. His classification involves a recasting of the nomenclature of mental diseases, and it is doubtful if the time is yet ripe for such an attempt.

Maniacal Depressive Insanity 1117

CHART XVII

Trophic and Sympathetic Disorders

DIAGNOSTIC ANALYSIS OF SYMPTOMS

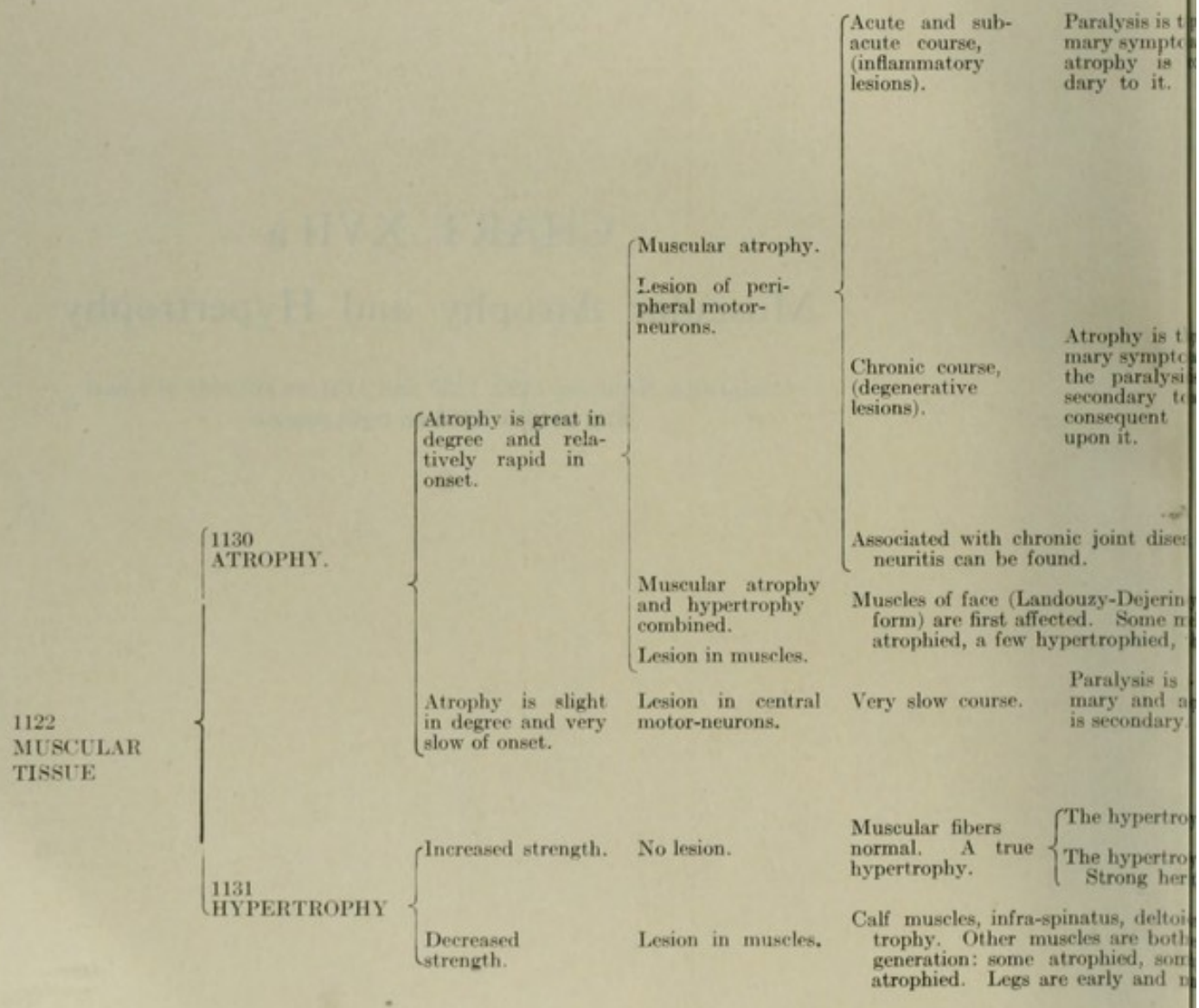
TROPHIC DISORDERS AND DISORDERS OF THE SYMPATHETIC SYSTEM

Symptoms Analysed	Tissues Involved	
1120 Trophic Lesions.	1122 Muscular Tissue.	See Chart XVII a.
	1123 Cutaneous and Sub-Cutaneous Tissue.	See Chart XVII b.
	1124 Fatty Tissue.	See Chart XVII c.
	1125 Bone Tissue.	
	1126 Joint Disease.	
	1127 Other Trophic Lesions.	
1121 Disorders of the Sympathetic System.	1128 Ganglionic Disorders.	See Chart XVII d.
	1129 Vaso-Motor Disorders.	

CHART XVII a

Muscular Atrophy and Hypertrophy

Comprising Numbers 1122, 1130 and 1131 on left side of Chart
and 1146 to 1156 on right margin


1122 MUSCULAR TISSUE
1130 ATROPHY.
1131 HYPERTROPHY
Atrophy is great in degree and relatively rapid in onset.
Atrophy is slight in degree and very slow of onset.
Increased strength.
Decreased strength.
Muscular atrophy.
Lesion of peripheral motor-neurons.
Muscular atrophy and hypertrophy combined.
Lesion in muscles.
Lesion in central motor-neurons.
No lesion.
Lesion in muscles.
Acute and subacute course, (inflammatory lesions).
Chronic course, (degenerative lesions).
Very slow course.
Muscular fibers normal. A true hypertrophy.
Associated with chronic joint dise
neuritis can be found.
Muscles of face (Landouzy-Dejerin
form) are first affected. Some m
atrophied, a few hypertrophied,
Paralysis is t
mary sympto
atrophy is
dary to it.
Atrophy is t
mary sympto
the paralysi
secondary t
consequent
upon it.
Paralysis is
mary and a
is secondary
The hypertro
The hypertro
Strong her
Calf muscles, infra-spinatus, deltoi
trophy. Other muscles are both
generation: some atrophied, som
atrophied. Legs are early and m

IS OF SYMPTOMS

ND HYPERTROPHY

SYMPTOMS			DIAGNOSIS	
		History of injury, wound, bruise or scar.	Injury of nerve (489, 822).	1146
		Limited to distribution of one nerve (simple neuritis) or many nerves, (multiple neuritis). Usually associated with sensory symptoms: pain and anesthesia, nerve and muscle tenderness.	Neuritis (488-92, 822, 940-8). (Figs. 33, 38.)	1147
ori- nd on-	Complete or partial electrical reaction of degeneration.	Groups of muscles attacked not corresponding to the distribution of any nerve. No sensory symptoms, except some pain at onset in back, joints and muscles. Very rarely nerve and muscle tenderness. Globulin and lymphocytosis in cerebro-spinal fluid in acute stage.	Acute anterior poliomyelitis (495, 789). (Figs. 26-7.)	1148
		Atrophy affects either the arms or the legs. Sensory and other symptoms of myelitis are present. Organic reflexes are more or less disordered. Superficial and deep reflexes are abolished in the paralyzed area.	Myelitis of Cervical or Lumbar Enlargement (485, 549).	1148a
ori- nd nd	Diminution of the electrical excitability, but no reaction of degeneration:	Atrophy commences in the small muscles of hands, or muscles of shoulder girdle, and extends and is associated with fibrillary contractions. Mild spastic paraplegia (525, 797) in legs.	Amyotrophic lateral sclerosis (547, 695, 797). (Figs. 26-7.)	1149
		Atrophy affects the muscles of tongue and lips and is associated with fibrillary contractions. Mild spastic paraplegia (525, 797) in legs.	Chronic bulbar paralysis (546, 694). (Figs. 21-2.)	1150
		Atrophy affects the hands usually. Is associated with dissociation of sensation and often with ulceration and mutilation.	Syringomyelia (552, 693, 837-9, 1009, 1170, 1357-9).	1150a
especially with ankylosis. Many of these cases are neuritic, but in some no			Arthritic atrophy.	1151
pe), or of shoulder girdle (Erb's juvenile type), or of legs (pseudo-hypertrophic es apparently hypertrophied. Excised muscle fibers show degeneration: some increase of interstitial fat. No fibrillary contractions.			Muscular dystrophies (477, 786, 1156).	1152
hy	The atrophy is due entirely to disuse. No electrical reaction of degeneration.	The reflexes are exaggerated. Ankle-clonus and Babinski are present when legs are affected, unless prevented by contractures.	A paralysis of long standing, especially one from infancy.	1153
is the result of much exercise.			Strong man or athlete.	1154
is due to muscle spasm, occurring at the commencement of voluntary motion. y.			Thomsen's disease (613).	1155
d some other muscles appear large, but are weak: a false or apparent hyper- ak and atrophied. No fibrillary contraction. Excised muscle fibers show de- ypertrophied and much interstitial fat. Slow course. All muscles are finally ly affected.			Pseudo-hypertrophic paralysis (500) and the muscular dystrophies (1152).	1156

CHART XVII b

Cutaneous and Sub-cutaneous Trophic Disorders

Comprising Numbers 1123 and 1132 to 1135 on left side of Chart
and 1160 to 1173 on right margin

1123 CUTANEOUS AND SUB-CUTANEOUS TISSUE

1132 Atrophy.

The skin is unusually smooth and thin. The fingers become pointed. change occurs quite frequently in nervous diseases, especially in th

The hair falls out, either all over head, face and body (as in syphilis) skin is not changed in appearance. Allied to this condition is the consequence of severe pain, or psychic shock, or unknown cause

Atrophy of the normal pigment of the skin; so that patches of clear persons of dark complexion. The edge of the patch is more deepl
See also facial hemi-atrophy, 1179.

1133 Hypertrophy.

The skin and mucous membranes everywhere appear thickened, as if i slightly, on pressure. The skin is sallow, dry and scaly. Patient body and features are enlarged. Nails, teeth and hair break a heavy. Voice is slow and hoarse. Response is slow and intellect disordered. The thyroid gland is atrophied, or destroyed by removal of the thyroid gland. Arterio-sclerosis and interstitial n common in women than in men, and frequently occurs at the time in children they become dwarfs. The cause of the disease is th thyroid gland and it can be cured by the administration of the thy

The skin is thickened, generally or locally, infiltrated, very firm an especially at their ends, and the fingers become much shortened in women than in men and seems to be allied to myxedema. The dis ocdematosum) and ends with an atrophy of the indurated patch (

1134 Eruptions.

Clusters of vesicles filled with clear fluid, each cluster upon a patch or two nerve roots and strictly limited to their distribution. Th It is usually accompanied, preceded and followed by severe pain in pain may continue for months after the rash has disappeared.

In some forms of nervous disease (especially in hysteria) elevated pa always when the skin is irritated (urticaria scripta dermographia sometimes do not.

Successive crops of bullae, which are at first small vesicles and increas Several vesicles may coalesce. There may or may not be fever. may be intense. A very fatal disease.

1135 Ulcerations.

With much loss of tissue.

Ulcerations larger and smaller with sloughing and loss of phalanges and even whole fingers and toes. The whole process is painless and may in part be the result of traumatism in the analgesic parts.

No spa the a sensil nerv

Spastic turba ture over symp

Large, deep, sloughing ulcers commencing wi patients usually suffering from motor and s subjected to much pressure (sacrum, trocha pulously clean.

With small loss of tissue.
See also Raynaud's disease (1195).

An ulceration usually commencing on the bal and painlessly extending deeper, until in m on its dorsum. Such an ulcer very rarely which ulcerates and the pus escaping form lary reflexes and other symptoms of tabes a in the urine in a small minority.

Ulcerations more or less severe, the result o skin is often bronzed. Symptoms of neurit

S TROPHIC DISORDERS

SYMPTOMS		DIAGNOSIS	
e nails are excessively curved and are striated. This in which the peripheral neurons are degenerated.		Glossy skin.	1160
only in patches, usually on the head and face. The rning white of the hair in patches, or universally, in s of hair dye).		Alopecia, (general or areata).	1161
ite appear. They are, of course, most noticeable in igmented than the surrounding skin.		Vitiligo and Leucoderma.	1162
rated, and do not pit, or pit but e very sensitive to cold. The all out. The movements are ity very sluggish and at times ase. The disease may follow itis may be present. Is more he climacteric. When it occurs osence of the secretion of the l gland.	Occurring in adults.	Myxedema.	1163
	Occurring in children.	Cretinism and Dwarfs (1090, 1177).	1164
ard. The bones of the phalanges become absorbed, abnormally movable. The disease is more common often commences as a local patch of edema (stadium lium atrophicum). At times patches are pigmented.		Scleroderma and Sclerodactyly.	1165
ddened skin; the clusters following the course of one uption dries up and disappears after a week or two. nerve, along the course of which it is situated. The		Herpes Zoster. Herpetic Neuritis.	1166
s, white or red, appear, at times spontaneously, and 200). Such patches of urticaria sometimes itch and		Urticaria (1201).	1167
any size, appear on the skin and mucous membranes. re are always some burning sensations and the pain		Pemphigus.	1168
symptoms in legs. The disturbances are limited to of distribution of one or more nerves. All forms of y are abolished. Small tumors may occur along the nks, together with other manifestations of leprosy.		Leprous Neuritis.	1169
nptoms in legs, when, as is usual, the trophic dis- s are limited to hands and arms Pain and tempera- se lost, with persistence of tactile sensibility, usually ected area. Kyphosis and spondylitis are common us.		Syringomyelia or Morvan's disease (552, 693, 1187). (Figs. 25-7.)	1170
edness of the skin and occurring only in bed-ridden ory paralysis, and occurring almost always on parts rs, etc.), especially when the parts are not kept scru-		Bed Sores. Decubitus.	1171
the foot, not growing larger superficially, but slowly cases it extends quite through the foot and appears curs on the hand. It usually commences as a corn, sinus. Loss of knee-jerk, Argyll-Robertson's pupil- resent in the majority of cases, while sugar is present		Perforating Ulcer of Tabes and (rarely) Syringomyelia and Diabetes.	1172
ight traumatism. In cases of arsenical neuritis, the 933) are present.		Neuritis (488-92, 822, 940-8, 1147).	1173

CHART XVII c

Trophic Disorders of Fat, Bone and Joints

Comprising Numbers 1124 to 1127 on left side of Chart
and 1175 to 1188 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS ABSTRACT C

1124 FATTY TISSUE.

1136 Atrophy. One of the earliest symptoms of diabetes mellitus is an inability of the b excess of fat had been deposited. Patients lose weight and if the dise urine shows the constant presence of sugar. Atrophy of fat and emac

1137 Hypertrophy. Large and tender deposits of fat, in lumps or in layers, widely diffused c Arms and legs painful and tender, especially in the acute stage when th frequently in middle aged women (often alcoholic or syphilitic).

1125 BONE TISSUE.

Failure in development.

Many cases occur, either congenitally or acquired in early life, in which so that the individuals remain throughout life of abnormally small sta due to atrophy or loss of function of the thyroid, or pituitary, gland. mally formed (simple dwarfs or decidedly undersized men), while oth described elsewhere, under infantilism and mongolism (1093), cretinis (chondro-dystrophia foetalis) there is a dystrophy of the epiphyseal c normally in length; so that dwarfism results. The head is relatively lc especially their proximal segment, the hand is short, the fingers broad, lordosis, pelvis contracted, legs often bowed or knock-kneed and joi developed. Adults, as well as children, not infrequently become short in the legs, as in rickets, osteitis deformans (1182), osteomalacia (118 kyphosis, etc.

In cases of extensive acute anterior poliomyelitis and of cerebral palsy of cl or very slow growth of the part from disuse.

1138 Atrophy. One side of the face is much smaller than the other, due to atrophy of al fat. The process is usually progressive. It seems to be caused by inju neuritis. Dryness, scaliness and loss of color of the skin are common small area atrophies, which atrophy gradually extends laterally over th process continues until the entire half of the face is atrophied and, in parts of the body. One side of the tongue is usually atrophied. Pa the atrophy.

1139 Hypertrophy.

One side of the face is much larger than the other, due to enlargement of progressive, and seems in some cases to be due to a periostitis.

The bones of the head and face are enlarged, diffusely or nodulated, and them. Headache, neuralgia, blindness, deafness and facial paralysis are not enlarged. Forehead is bulging and head is often of great size

Disease commences late in life with slight pains, especially in legs. Th jaw is not enlarged. The head enlarges, the legs and vertebral colun patients become shorter (even as much as a foot or more) and their w

Symmetrical enlargement of all the tissues, but especially the bones of th etc. It comes on gradually, patient requiring larger and larger gloves shouldered" (kyphosis). These changes are often associated with bite head and joints is a common symptom. The disease is caused by hy in early life, before the epiphyses are joined by bone to shaft, gigantis

The hands and feet are enlarged, and the fingers and toes "clubbed." Th by the X-ray. These symptoms are associated with chronic pulmonary toms vary greatly in degree and extent; the mildest form being "clubb

1140 Fragility. In some persons the bones are unusually brittle and break upon the sligh of these cases occur in old age (senility), others occur in middle life, du malacia), while others occur in children. The disease causing it ha thyrosis, etc.

1126 JOINT DISEASE.

Joints painless, enlarged, abnormally movable, especially hyperextension, cartilages eroded, effusion of synovial fluid, exostoses of bone. The exciting cause for these changes is often painless traumatism, at least in part.

Joint involvement not uncommon. Usually in legs. Knee-jerk Bladde

Joint involvement rare. Usually in arms. Knee-jerk Loss o

1127 OTHER TROPHIC LESIONS.

1141 Atrophy and hypertrophy. Atrophy or hypertrophy of different organs (mammary glands, tongue, etc quently met with and may be due to disordered nervous action, but the

F FAT AND BONE

SYMPTOMS	DIAGNOSIS	
to deposit fat in the tissue, although previously often an is long continued become emaciated. Examination of the ion occur in fevers and in many other conditions.	Diabetes Mellitus (900, 1172).	1175
arms and legs. Face, feet and hands not much involved. it is being deposited. Locomotion impeded. Occurs most	Adiposis Dolorosa. Dercum's Disease (1012).	1176
bony framework of the body does not develop normally; . There is reason to believe that some of these cases are ne of these individuals are merely small but otherwise nor- show many physical deformities. Some cases have been 090, 1164) and microcephaly (1084). In *Achondroplasia* lages, in consequence of which the bones do not increase the bridge of the nose depressed, the arms and legs short, almost equal length and divergent (trident shape), lumbar abnormally lax. The muscles are rather unusually well consequence of excessive bowing of weakened long bones etc. and in consequence of curvature of the spine, as in	Dwarfism, Microsmia, Nanosmia, Achondroplasia (1164).	1177
hood occurring in infancy there is often an arrest of growth	Disuse from Paralysis.	1178
e tissues, even of the bones, and especially of the skin and infection, or cold and in some cases is due to a trigeminal nptoms. The process commences in the skin, of which a kin and inward to the fat, muscles and even bones. The cases, extends beyond the median line and even to other n the trigeminal nerve usually precedes and accompanies	Facial Hemiatrophy.	1179
the tissues, especially of the bones. The process is usually	Facial Hemihypertrophy.	1180
y cause pressure symptoms on the nerves running through , thus, common symptoms. Lower jaw and extremities	Hyperostosis Cranii or Leontiasis Ossea.	1181
nes of the body become enlarged and soft, but the lower become bent and bowed (spondylitis and kyphosis). The is affected.	Osteitis Deformans. Paget's Disease.	1182
nds and feet, lower jaw, and sternum, also ears, tongue, d shoes. Thorax is much enlarged and patient is "round oral hemianopia, followed at times by blindness. Pain in trophy of the pituitary body. If the disease commences stead of acromegaly results.	Acromegaly and Gigantism.	1183
nes of the forearms are also often enlarged, as can be shown ease of a septic or tuberculous nature usually. The symp- fingers."	Hypertrophic Pulmonary Osteoarthropathy.	1184
violence, even on turning the patient over in bed. Some softening of the bone and diminution of lime salts (osteo- sen variously named: osteogenesis imperfecta, osteopsa-	Fragilitas Ossium. Osteopsathyrosis.	1185
re absent. Pains in legs. Ataxia without paralysis. mptoms. Argyll-Robertson's pupil reflex.	Arthropathy of Tabes (661). (Charcot's Disease.) (Figs. 24-7.)	1186
re exaggerated. Pains in arms. Paralysis of arms (slight). inful and thermic, with persistence of tactile, sensibility.	Syringomyelia (552, 693, 1170). (Figs. 24-7.)	1187
or other parts of body (hands, fingers, etc.), are not infre- re of obscure significance and are without diagnostic value.	Localized Hypertrophies and Atrophies, symmetrical and asymmetrical.	1188

CHART XVII d

Ganglionic Disorders, Vaso-Motor Disorders

Comprising Numbers 1128, 1129 and 1142 to 1145 on left side of Chart
and 1191 to 1203 on right margin

1128 GANGLIONIC DISORDERS.

1142 Paralytic.

Ptosis of eyelid, although patient can raise it perfectly by an effort of will (
not dilate when shaded, although it contracts briskly when eye is exposed
sure with retraction and lowering of eyeball (enophthalmus). Intra-oc
abolished, flushing of skin and absence of sweat on the affected side of fac
third rib.

1143 Irritative.

The symptoms are exactly opposite to those of paralysis of the cervical sym
widening of the palpebral fissure (Stellwag's sign) and delayed descent of u
The Boston-Kocher's sign, an amplification of Graefe's sign, may occur in

Exophthalmus, tachycardia, goitre, flushing, sweating, tremor, nervousness,
ward (Graefe's sign), widening of the palpebral fissure (Stellwag's sign), th
The disease occurs much more frequently in women than in men and altho
the cervical sympathetic ganglia, yet it is really due to excessive secretion
the reverse of those of myxedema (1163), can be produced by the admini
extirpation of the thyroid.

Paroxysmal spasm or congestion of the bronchioles, often reflex from nasal
nervous temperament of most asthmatics, together with the very rapid on
may be due to a disturbance of the thoracic sympathetic. The paroxysm
prolonged expiratory murmur, make the diagnosis easy. Asthma is associ
be in part voluntary, in part reflex; also is usually associated with bronch

1129 VASO-MOTOR DISORDERS.

1144 Vascular.

Paroxysmal attacks of coldness and pallor ("dead fingers," "local syncope")
of all together. These attacks may last a few minutes or hours and then
the same parts become dusky blue, or purplish black ("local asphyxia or c
This attack may pass off, after several hours, with abundant sweating, or
grenous and finally slough off. The necrosis does not usually involve the
metrical. It is more common in cold weather and is often brought on by
Hematuria and evidence of congestion of other internal organs may occur

Analogous to Raynaud's disease is gangrene of extremities occurring in many
old age; either without the local syncope or local asphyxia, or with only s

Paroxysmal attacks of formication, tingling, numbness and other paresthesi
intervals and exclusively in women. They seem to be brought on by ove
cases during the attack the skin becomes pale and blue. Similar symptoms

Paroxysms of severe pain in one foot, rarely in both, rarely in hands and ver
creased by allowing foot to hang down, or by motion of it, or by cold. Th
redness and swelling of the whole, or part, of the sole of foot. Usually a
to a simple vaso-motor neurosis. The neuritis, when present, is often asso

Occurs in middle aged or elderly persons and is associated with arterial disease
walk and increases so that walking becomes impossible. It passes off after
attack the feet are cold and there is absent or greatly diminished pulsation i
alcohol and tobacco and injury seem to be common causes of this condition
feet. The arms are rarely involved.

In many diseases if lines or writing be traced on the skin with a sharp point, t
to lines of bright redness, which persist for minutes or hours.

1145 Exudative or Secretory.

Paroxysmal attacks of localized edema of sub-cutaneous or sub-mucuous tissu
a few hours or days. The extent of the edema varies greatly. It may be
extremity, or even more. It may cause death when occurring in the larynx.
sure. They occur in hysteria and are usually associated with a neurasthen
are associated with symptoms of digestive disorder, they are called urticaria
except the itching. The disease often shows a strong heredity and at times

Edema of the legs, unilateral becoming bilateral, bad heredity. The edema
being a sudden demarcation at the level of the joint. The edema may be a

Some cases present paroxysmally or constantly a profuse sweating, usually loca

OTOR DISORDERS

YMPTOMS	DIAGNOSIS	
ido-ptosis). Contraction of pupil (myosis), which does light and on convergence. Narrowing of palpebral fis- tension diminished. The cilio-spinal reflex 335) is d also on side of neck, or of arm and thorax above the	Paralysis of Cervical Sympathetic.	1191
hetic. Dilatation of pupil (mydriasis), exophthalmus, r eyelid when eye is turned downward (Graefe's sign). s disease and in exophthalmic goitre (1193).	Irritation of Cervical Sympathetic.	1192
yed descent of upper eyelid when eye is turned down- and systolic murmur in vessels of neck and in thyroid. many of its symptoms may be referred to disorder of the thyroid gland. Many of its symptoms, which are tion of thyroid gland, and the disease can be cured by	Exophthalmic Goitre.	1193
ease. Freedom from symptoms in the interval. The and cessation of the attack, indicates that the disease ttacks of dyspnoea, with the abundant dry rales and with strong contraction of the diaphragm, which may	Asthma (617).	1194
tingling of fingers or toes or tip of nose or of ears or y pass off, or may be followed by an attack, in which osis"), from congestion. This is associated with pain. parts, or a small portion of them, may become gan- le of the cyanotic area. The disease is usually sym- tting hands in cold water, or by working with hands. ome attacks.	Raynaud's Disease. Symmetrical Gangrene (1011).	1195
mbers of a family at varying ages from childhood to t indications of these conditions in some of the cases.	Family Gangrene.	1196
n fingers and hands. The attacks occur at irregular rk and by having the hands in cold water. In some metimes occur in the early stages of acromegaly (1183).	Acroparesthesia.	1197
rely in face, lasting a few minutes or a few hours, in- ain, except in the earliest attacks, is accompanied by ks men only, and is generally due to a neuritis, rarely ed with atheromatous arteries.	Erythromelalagia (1010).	1198
A painful cramp occurs in muscles of legs after a short ort rest to return if walking is resumed. During the e dorsalis pedis or posterior tibial artery. Syphilis, The disease not infrequently precedes gangrene of the	Intermittent Limping or Claudication. Dysbasia Angio-Sclerotica (554).	1199
ines appear for a few seconds white, but soon change	Dermographia (326, 1167).	1200
ausing localized swellings, either white or red, lasting e-half inch in diameter, or may extend over an entire These swellings are not tender and do not pit on pres- ondition. If the swellings are red in color, itch and No sharp line can be drawn between the two diseases ems to be malarial.	Angio-Neurotic Edema and Urticaria. (1167). Quincke's Disease.	1201
be limited above by the ankle, knee or groin; there iated with pyrexia or gastric disturbance.	Milroy's or Meig's Disease. Trophedema.	1202
d, sometimes general.	Hyperhidrosis. Excessive Sweating	1203

CHART XVIII

Syphilis of the Nervous System

Comprising Numbers 1205 to 1217

DIAGNOSTIC SYMPTOMS AND TESTS

1205
SYPHILIS OF THE NERVOUS SYSTEM.

History of personal, or hereditary, syphilis. Physical evidence of syphilis; such as Wassermann reaction, a chancre or its scar, induration, mucuous patches, a syphilitic rash or its copper colored scars, hazy cornea, notched teeth, furrows about angle of mouth, saddle nose, ptosis, iritis, enlarged glands, periosteal nodes, etc.

- Syphilitic Nervous Diseases.

  Symptoms of syphilis of the nervous system are very variable from day to day, transitory and manifold. They consist of paresis, rather than of complete paralysis. They usually show rapid improvement under K.I. and Hg. or Salvarsan. Nocturnal headache is common, as are also the Argyll-Robertson's pupillary reflex, unequal pupils and optic neuritis.

  - Cerebral symptoms.

    Although these symptoms can be divided into several, more or less well defined, groups, yet a combination of several or all of the lesions, in varying intensity, is not infrequent; so that a combination of the symptoms of several or all of the groups may be present in one case. Pure, uncomplicated cases of each type are, however, commonly met with.

    - Little or no lym in cerebro-spinal lumbar puncture.
    - Globulin and decide ocytosis is found i spinal fluid from puncture.

  - Spinal symptoms.

    (Both forms of spinal syphilis may occur together.)

    - No globulin and lit lymphocytosis foun cerebro-spinal fluid lumbar puncture.
    - Globulin and decide ocytosis found in spinal fluid from puncture.

  - Cerebral and spinal symptoms.

    - Globulin and decide ocytosis in cerebro-s fluid.

  - Local peripheral symptoms.

    - Wassermann reactio blood. Normal cereb fluid.

- Post-, or Meta-, syphilitic nervous disease.

  - Cerebral symptoms.
  - Spinal symptoms.

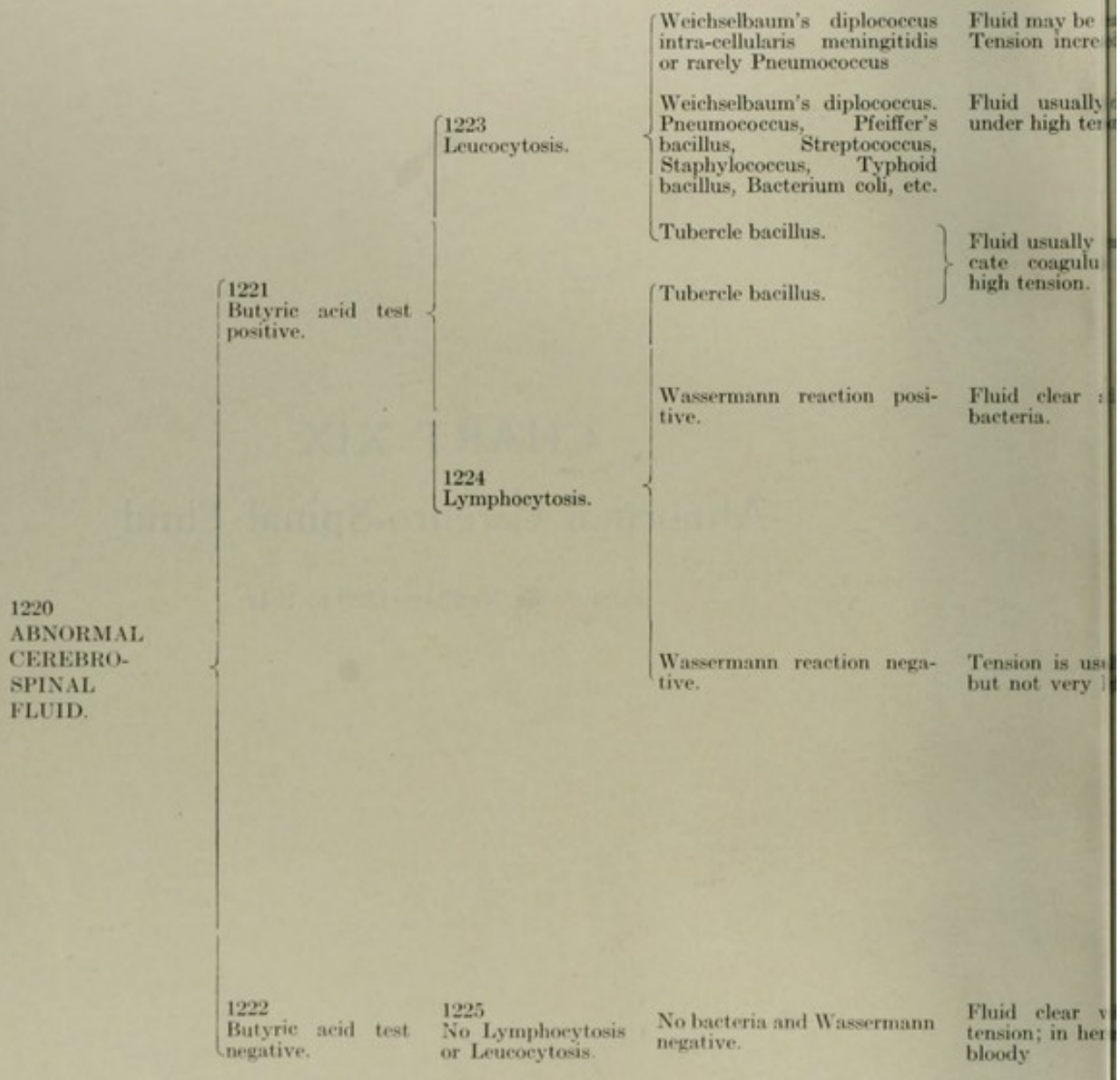
    - Increased lymphocy cerebro-spinal fluid.

ABSTRACT OF SYMPTOMS		DIAGNOSIS	
Symptoms of cerebral tumor (507, 536). Other syphilitic symptoms may be present. Rapid course with irregular remissions and intermissions. The symptoms of cerebral compression are much less pronounced than in non-syphilitic tumors. Very amenable to anti-syphilitic treatment.		Isolated Cerebral Gumma.	1206
Symptoms of cerebral thrombosis (506). The attacks occur rather early in adult life. There are many prodromata. Nocturnal headache is common. The paralysis is moderate in degree, variable in intensity and often temporary. Mental derangements, often in the form of trance-like states, frequently occurs. Branches of the basilar artery are involved most frequently, and the attack often occurs during sleep, or without coma during the day.		Cerebral Syphilitic Endarteritis and Thrombosis.	1207
Symptoms of meningitis (590, 608), which may be very slight and very variable. With severe headache (nocturnal) there may be some nausea and vomiting. Little or no elevation of temperature or retraction of neck. No tuberculin reaction or evidence of tuberculosis. This disease is rare in children.	Symptoms of cortical irritation (Jacksonian epilepsy, local headache and tenderness) and paralysis of cortical functions (aphasia, monoplegia, etc.). Mental derangement is common, and often takes the form of paresis (pseudo-paresis), but is amenable to anti-syphilitic treatment.	Syphilitic Meningitis of Convexity of Brain.	1208
	No symptoms of cortical irritation or paralysis of cortical functions. Paralysis of cranial nerves (especially the oculo-motorius), at times, of irregular distribution and in varying degree. Drowsiness and stupor are common.	Syphilitic Meningitis of Base of Brain, including Kahler's Disease.	1209
Symptoms of Brown-Séquard's paralysis, or later of paraplegia (442, 509, 840, 981).		Isolated Spinal Gumma.	1210
Symptoms of myelomalacia (485, 513-4, 517-8, 549-50).		Spinal Syphilitic Endarteritis and Thrombosis.	1211
Symptoms of lateral sclerosis (525). (Fig. 26.)		Erb's Syphilitic Lateral Sclerosis.	1212
Symptoms of spinal meningitis, or of pachymeningitis (550, 608, 974, 1005). Rigidity of back. Girdle pains and radiating pains, exaggerated reflexes in legs. Some of these cases present the symptoms of progressive spinal muscular atrophy (547).		Syphilitic Meningitis of Cord and of Nerve Roots. (Meningo-myelitis, Pachymeningitis Cervicalis Hypertrophica.)	1213
A combination of the above symptoms (1208-9, 1213) in very varying extent and intensity. A clinical picture comprising cerebral and spinal symptoms and presenting great variations, which are impossible to describe in a few words.		Cerebro-Spinal Syphilis.	1214
Symptoms of neuritis (488-92, 822-3, 940-8).		Syphilitic Neuritis.	1215
Symptoms of general paresis (1104).		Paresis.	1216
Symptoms of locomotor ataxia (661).		Locomotor Ataxia. Tabes. (Fig. 27.)	1217

CHART XIX

Abnormal Cerebro-Spinal Fluid

Comprising Numbers 1220 to 1242


1220
ABNORMAL
CEREBRO-
SPINAL
FLUID.
1221
Butyric acid test positive.
1222
Butyric acid test negative.
1223
Leucocytosis.
1224
Lymphocytosis.
1225
No Lymphocytosis or Leucocytosis.
Weichselbaum's diplococcus intra-cellularis meningitidis or rarely Pneumococcus
Fluid may be
Tension incre
Weichselbaum's diplococcus. Pneumococcus, Pfeiffer's bacillus, Streptococcus, Staphylococcus, Typhoid bacillus, Bacterium coli, etc.
Fluid usually
under high te
Tubercle bacillus.
Fluid usually
cate coagulu
high tension.
Tubercle bacillus.
Wassermann reaction positive.
Fluid clear
bacteria.
Wassermann reaction negative.
Tension is us
but not very
No bacteria and Wassermann negative.
Fluid clear
tension; in he
bloody

RO-SPINAL FLUID

GNOSTIC SIGNS			DIAGNOSIS	
ar or cloudy. l usually.	Occurs in epidemics.	Symptoms of epidemic Cerebro-spinal meningitis (591).	Epidemic Cerebro-spinal Meningitis.	1226
cloudy and n.	Occurs sporadically.	Symptoms of sporadic or purulent cerebro-spinal meningitis (592).	Sporadic Purulent Meningitis.	1227
ar with deli- and under	Acute course.	Symptoms of tuberculous meningitis (593).	Acute, or sub-acute Tuberculous Meningitis.	1228
	Chronic course.		Chronic Tuberculous Meningitis.	1229
free from	Tremor and mental symptoms.	Symptoms of Paresis (1104).	Paresis.	1230
	Ataxia.	Symptoms of Tabes (661).	Tabes.	1231
	Symptoms not typically characteristic of paresis or tabes, being due to a cerebro-spinal meningitis.		Cerebro-spinal Syphilis (1208-9, 1213-14).	1232
	Motor paralysis.	Symptoms of acute anterior poliomyelitis (495). (Figs. 26-7.)	Acute Anterior Poliomyelitis.	1233
	Herpetic rash.	Symptoms of herpes zoster (1166).	Herpes Zoster.	1234
y increased	Epidemic. High fever.	Symptoms of Typhus.	Typhus Fever.	1235
	Choked disc usually present.	Symptoms of cerebral or spinal tumor (507, 578, 587).	Tumor.	1236
	Choked disc may be present.	Symptoms of cerebral or spinal abscess (508, 578, 587).	Abscess.	1237
		Symptoms of hydrocephalus (411, 905, 960).	Hydrocephalus.	1238
	Headache.	Symptoms of serous meningitis (594).	Serous Meningitis.	1239
	Apoplexy.	Symptoms of cerebral or spinal hemorrhage (503, 524, 1060-1).	Hemorrhage.	1240
increased hage often	Albumen and casts.	Examination of the urine shows albumen and casts. Edema, headache, dyspnoea, etc., usually present.	Uremia.	1241
	Anemia.	Examination shows anemia, pallor, etc., or acute infections, or some similar conditions.	Anemia.	1242

PART III

LOCALIZATION

OF

LESIONS WITHIN THE NERVOUS SYSTEM

BY

A CONSIDERATION OF THE
PARALYTIC AND IRRITATIVE SYMPTOMS
RESULTING FROM THEM

CHART XX

Spinal Localization

Comprising Numbers 1250 to 1267

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1250 V Sacral	None.	Coccygeus.	Elevation of coccyx.	Anal.	None.	Skin over sacrum and anus.
1251 IV Sacral	Coccygeus.	Levator ani. Sphincter ani. Detrusor urinae. Transversus perinei. Erector penis. Compressor urethrae.	Elevation of coccyx. Elevation of anus. Sphincter ani. Ejection of urine. Vaginal constriction.	Erection of penis diminished.	None.	Slightly larger area than above extending over inner portion of gluteal region.
1252 III Sacral	Sphincter ani. Levator ani. Detrusor urinae. Transversus perinei. Erector penis. Compressor urethrae.	Rectum.	Defecation disturbed. Retention of urine, later followed by dribbling. Ejaculation lost. Erection possible but paretic.	Ejaculation lost. Erection diminished. Tendo-Achillis.		As above, and perineum, genitals and upper part of inner surface of thighs. (Testicle sensitive to pressure.)
1253 II Sacral	Sphincter ani. Levator ani. Detrusor urinae and other muscles as in 3d sacral.	Pyriformis. Obturator internus. Gemellus superior. Gluteus maximus. Biceps femoris. Gastrocnemius. Soleus. Tibialis posticus. All the small muscles of foot.	Outward rotation of thigh. Retraction of thigh. Flexion of knee. Plantar flexion of foot. Standing on the toes. Raising inner margin of foot. Defecation and Retention of urine as in 3d sacral.	Ejaculation. Erection. Plantar weakened.	None.	As above, and the posterior surface and outer surface of thighs.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1254 I Sacral	Muscles of anus. Muscles of bladder. Muscles of genitals. Pyriformis. Abductor hallucis. Flexor hallucis brevis. I-IV dorsal interossei. I-III plantar interossei. III-IV lumbricales. Abductor minimi digiti. Opponens minimi digiti.	Gluteus maximus. Obturator internus. Gemellus superior. Gluteus medius. Gluteus minimus. Biceps femoris. Semi-membranosus. Semi-tendinosus. Popliteus. Gastrocnemius. Soleus. Tibialis posticus. Peroneus longus. Peroneus brevis. Flexors of toes. Extensors of toes.	Retention of feces. Retention of urine or dribbling. Erection and ejaculation impossible. Outward rotation of thigh impaired. Internal rotation impaired. Flexion of knee difficult. Plantar flexion of foot. Raising inner margin of foot. Raising outer margin and dorsal flexion of foot. Flexion and extension of toes, adduction of great toe, abduction of little toe, etc.	Plantar weakened. Achilles-tendon reflex. Ejaculation. Erection. Micturition. Defecation. Gluteal.	None.	As above, and a strip on posterior and outer surface of lower legs and of dorsum of foot and especially of toes.
1255 V Lumbar	Muscles of anus and rectum. Muscles of bladder. Muscles of genitals. Pyriformis. Biceps femoris. Flexors of toes. Peroneus longus. Peroneus brevis.	Gemellus superior. Gemellus inferior. Gluteus medius. Gluteus minimus. Semimembranosus Semi-tendinosus. Gluteus maximus. Tensor fasciae femoris. Gastrocnemius. Soleus Extensors of toes. Tibialis anticus.	Defecation. Micturition delayed, dribbling. Erection and ejaculation impossible. Outward rotation of thigh very difficult. Inward rotation impaired. Flexion of knee difficult. Retraction of thigh very difficult. Flexion of foot barely possible. Flexion of toes impossible. Extension of toes weak, except great toe, which may be dorsally flexed. Raising inner margin of foot difficult. Raising outer margin of foot impossible.	Ejaculation. Erection. Micturition. Defecation. Gluteal.	Plantar. Tendo-Achillis.	As above, and back of thighs and legs and inner and outer margin and sole of feet.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1256 IV Lumbar	Muscles of rectum and anus. Muscles of bladder. Muscles of genitals. Obturator internus. Pyriformis. Gemelli. Gluteus medius. Gluteus minimus. Gluteus maximus. Biceps femoris. Semimembranosus. Semitendinosus. Popliteus. Gastrocnemius. Soleus. Flexors of toes. Extensors of toes. Peroneus brevis. Peroneus longus. Tibialis anticus.	Obturator internus. Rectus femoris. Vastus externus. Vastus internus. Adductor magnus. Adductor brevis. Adductor minimus. Gracilis.	Defecation, with fecal incontinence. Micturition, with dribbling. Erection and ejaculation impossible. Outward rotation of thigh weak. Inward rotation impossible. Retraction of thigh impossible. Flexion of knee lost. Plantar flexion of foot lost. Flexion and extension of toes lost. Raising outer margin of foot. Raising inner margin. Extension of thigh weak. Adduction difficult.	Patellar may be wanting.	Plantar.	As above, and inner side of lower legs and dorsum of feet, and strip on outer posterior surface of thighs.
1257 III Lumbar	Muscles of anus, bladder and genitals. Outward rotators and thigh. Inward rotators of thigh. Retractor (flexor) thigh. Flexors of knee. Plantar flexors of foot. Flexors of toes. Extensors of foot. Vastus externus.	Vastus internus. Rectus femoris. Crureus. Adductors of thigh. Flexors of thigh at the hips.	All movements of legs are lost, except that extension of legs is barely possible and that the thigh can be flexed on body by the psoas and iliacus. Defecation and micturition are destroyed. Urine and feces dribble and cannot be retained.	Patellar and cremasteric.	Ankle-clonus may exist.	As above, and whole of legs except a triangular area on front of thigh with base at Poupart's ligament.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1258 II Lumbar	Paralysis of all muscles of lower extremity, except psoas.	Psoas.	Complete paralysis of legs, rectum and bladder. As above.	Patellar, Achilles, and cremasteric.	Achilles may be increased. Plantar.	Whole of legs and pelvis. (Testicles not sensitive to pressure.)
1259 I Lumbar	Total paralysis of whole lower extremity, psoas included.			Cremasteric and Achilles.	Patellar absent or increased.	As above, and groins and front of scrotum and penis.
1260 XII to III Dorsal	Paralysis of lower extremity, and gluteal region. Paralysis of abdominal and and dorsal regions, gradually added as the site of the lesion ascends.		As above, and paralysis of muscles of respiration causes diaphragmatic breathing and dyspnoea.	Epigastric and umbical reflex.	Patellar, cremasteric, Achilles and Plantar.	As above, and a band running around body about two segments below the one involved and limited above by a narrow zone of hyperesthesia.
1261 II Dorsal	As in 3d dorsal.		As above.	All below lost in complete division of cord.	All subjacent reflexes.	As above, and a strip on the inner side of the upper arms.
1262 I Dorsal	All muscles of trunk and lower extremities.	Flexion of fingers. Muscles of the little finger. III and IV interossei. Lumbricales. Pronator quadratus. Lower part of pectoralis major. Lower part of pectoralis minor.	As above and weakness in flexion of fingers. Pronation disturbed.	Oculopupillary symptoms. All below lost in complete division of cord.	All subjacent reflexes.	As above, and a strip on the inner side of the forearms.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1263 VIII Cervical	Paralysis of muscles of trunk and lower extremities. Abductor of little finger. Adductor of thumb. Flexor of the little finger. Opponens minimi digiti. III and IV interossei. Lumbricales.	Flexors of the little finger. Opponens minimi digiti. Flexor subl. digitorum. Flexor profun. digitorum. Flexor carpi ulnaris. Extensors of the thumb and fingers. Triceps (slight). Latissimus dorsi (lower part). Pectoralis major. Pectoralis minor. Scalenus medialis. Scalenus posterior.	As above. Hand weak. Extension of arm. Int. rotation and retraction of arm. Adduction of arm.	Oculo-pupillary symptoms. All below lost in complete division of cord.	All below.	As above, and the fingers, except volar surface of the thumb and the ulnar surface of the little finger. The cervical sensory nerve roots supply the same area of the skin in common, especially in the hands and fingers. Hence the anesthesia is slight and uncertain.
1264 VII Cervical	Lower extremities and trunk. Flexor profundus digitorum (ulnar side). Flexor carpi ulnaris. Small hand muscles. Pronator quadratus.	Extensors, Flexors and Abductors of thumb. Extensor indicis. Extensors of the fingers (movements barely possible). Supinator longus. Biceps (very slightly paretic). Triceps. Pectoralis major. Serratus magnus (slight). Latissimus dorsi. Teres major.	As above and Hand very weak. (Winged scapulae.) Retraction and inward rotation of arm.	Arm reflexes. Forearm reflexes. Palmar reflex. All below lost in complete cord division.	All below.	As above, and most of the hands and a small strip on the anterior, another on the posterior, surface of the forearm.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1265 VI Cervical	Muscles of lower extremity and trunk. Muscles of fingers (including thumb) and hand. Triceps. Pectoralis major. Latissimus dorsi. Teres major. Infraspinatus. Serratus magnus.	Coraco-brachialis. Biceps. Brachialis anticus. Supinator brevis. Deltoid. Scaleni. Splenii. Deep head and neck muscles.	As above and movements of fingers and thumb impossible. Extension of forearm. Flexion of forearm weak. Supination very weak. Adduction of arm and inward rotation. Adduction, retraction and external rotation. "Winged" scapulae. Raising of arm. Rotation of head. Fatal in a few days or weeks.	Arm reflexes. Extensor forearm reflexes. All below lost in complete cord division.	All below.	As above, and whole of hands and fingers and radial side of forearm.
1266 V Cervical	Muscles of lower extremities and trunk. All the muscles of the arm, forearm, hand and fingers; even the deltoid, coraco-brachialis and brachialis anticus. Deep cervical muscles. Intercostals.	Levator anguli scapulae. Scaleni. Diaphragm (because of filaments from V cervical segment to phrenic nerve), or spread of injury from 5th to 4th cervical segment. Trapezius and sterno-cleido-mastoid are intact.	As above and shoulders raised with difficulty. Rotation and flexion of head. Dyspnoea. Fatal in a few hours or days.	Scapular and tendon reflexes of paralysed muscles in arms. All below lost in complete cord division.	All below.	As above, and whole of arms, except tip of shoulder.
1267 IV-I Cervical	Total cross-lesions from the fourth cervical segment upward are rapidly fatal, because of complete paralysis of the diaphragm and intercostals. Total cross-lesions of the brain-stem are rapidly fatal for the same reason.					

CHART XXI

Cerebral Localization

Comprising Numbers 1268 to 1286

CHART XXI a

Cerebral Localization in the Medulla and Pons: Ganglia at Base

Comprising Numbers 1268 and 1269

Seat of Lesion			Paralysis of Motion	Paralysis of Sensation	Action Lo Impair
1268 Lesion involving lateral half of the Medulla Oblongata. Babinski and Nageotte's Bulbar Syndrome (437). Rare because of the small transverse area of the medulla. Thrombosis of Posterior Inferior Cerebellar Artery causes very similar symptoms. (Figs. 21-3.)			Crossed paralysis: hemiplegia alternans hypoglossica. Homolateral half of tongue, diaphragm and vocal cord, contralateral arm and leg. In some cases arm and leg may be paralysed on both sides, but not equally so. Extremely rarely leg on one side and arm on the other are paralysed.	Taste in posterior part of homolateral half of tongue. All forms of sensation in pharynx and throughout the respiratory tract. Analgesia and thermic anesthesia of homolateral half of face and contralateral half of body. Anesthesia of one side, or of both sides of the body.	Articulation, deglutition, r cardiac action, vomiting, use and of arms a one or both sid
1269 Lesion in lateral half of the Pons Varolii. (Fig. 20.)	Lesion in Lower (Caudad) Third.	Confined to the bridge portion.	Crossed paralysis: hemiplegia alternans facialis. Muscles of expression of homolateral half of face and the external rectus at times, and contralateral arm, leg and half of tongue (Millard-Gubber's syndrome—439).	None, unless indirectly from pressure and then contralateral hemianesthesia.	Articulation, mastication. M of homolateral face, and of co arm and leg.
		Confined to the tegmentum.	Muscles of expression of homolateral half of face and of external rectus (Foville's paralysis). Contralateral arm and leg may be slightly involved.	Contralateral hemianalgesia and thermic an ethesia and at times hemianesthesia. Anesthesia, and especially analgesia, of homolateral half of face (Hemianesthesia alternans). Very rarely, deafness. Rarely dissociation of sensation.	Articulation, m winking. Mov homolateral hal
	Lesion in Middle and Upper (Cephalad) Third.	Confined to the bridge portion.	Complete contralateral hemiplegia.	Usually of all forms of sensation in homolateral half of face. Occasionally also hemianesthesia of contralateral half of body.	Chewing and articulation. M of contralateral body.
		Confined to the tegmentum.	Conjugate deviation of eyeballs toward the side of the lesion. May be complete hemiplegia of slight degree from pressure.	Paralysis of all forms of sensation on homolateral half of face. Contralateral hemianalgesia. May be contralateral hemianesthesia	Chewing and articulation. movement of toward the sam the lesion.

NSVERSE LESIONS OF BRAIN-STEM

MEDULLA AND PONS

OR	Reflexes Altered	Vertigo	Ataxia	Muscle Sense	Secretory and Other Disturbances
nation, ration, ughing, tongue leg on	Tendon reflexes increased with Babinski and ankle-clonus on opposite side. Cutaneous reflexes may or may not be increased.	Usually present.	Usually present and of both motor and cerebellar type. Homolateral.	Usually lost, especially if motor ataxia be present.	Myosis and pseudo-ptosis (ophthalmoplegia sympathica) and salivation are common. Cheyne-Stokes's respiration (435).
inking, ements alf of lateral	Tendon reflexes increased with Babinski and ankle-clonus on opposite side. Cutaneous reflexes may or may not be increased.	Often present.	No motor, but there may be cerebellar, ataxia.	Normal.	Conjunctivitis is frequent in eye of same side. May be a tendency to fall or to turn to one side. Salivation.
cation, nts of f face.	Normal or slightly exaggerated as above.	Usually present.	Usually present on the same side as the lesion.	Lost on the same side as the lesion.	Conjunctivitis is frequent in the eye of the same side. Salivation.
sually ements alf of	Tendon reflexes increased with Babinski and ankle-clonus on the opposite side. Cutaneous reflexes may or may not be increased.	Often present.	No motor, may be cerebellar, ataxia	Normal.	Ulceration of cornea may occur. May be a tendency to fall or turn to one side.
sually jugate eballs ide as	Normal or may be slightly exaggerated.	Present.	May be motor and cerebellar atxaia.	Lost on the same side as the lesion.	Ulceration of the cornea may rarely occur. A slow rhythmic tremor of the arm and leg of opposite side may be present.

CHART XXI b

Cerebral Localization: Ganglia at Base

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF BRAIN STEM AND CEREBELLUM

Seat of Lesion	Paralysis of Motion	Paralysis of Sensation	Actions Lost or Impaired	Reflexes Altered	Vertigo	Ataxia	Muscle Sense	Secretory and Other Disturbances
1270 Crura Cerebri — Lesion confined to the pes or foot.	Some, or all, of the ocular muscles (except external rectus) on the same side, combined with a contralateral hemiplegia, usually complete. Hemiplegia alternans oculomotoria. (Weber's syndrome, 440).	None. Contralateral hemianesthesia, or hemianalgesia and thermic hemianesthesia, or both. Deafness may be present, if lesion be bilateral.	Movement of eyeball. Use of contralateral half of the body.	Tendon reflexes increased, with Babinski and ankle-clonus, on opposite side. Cutaneous reflexes may or may not be increased.	Usually absent.	None.	Normal.	Tremor resembling that of paralysis agitans (Benedikt's syndrome). (441)
1270 Crura Cerebri — Lesion confined to the tegmentum.	One or more ocular muscles, except the abducens.		Movement of eyeball.	Tendon reflexes normal.	Present.	Cerebellar type.	Impaired.	A slow, rhythmic tremor of arm and leg of opposite side may be present.
1271 Corpora Quadrigemina. — Lesion confined to anterior pair (nates).	Bilateral, more or less extensive, of all ocular muscles, except the abducens.	May be blindness without choked disc or other lesion.	Movement of eyeball.	Pupil reflex lost to both light and accommodation.	Usually absent.	May be absent.	Normal.	Nystagmus (at times vertical), squint, pupils often unequal.
1271 Corpora Quadrigemina. — Lesion confined to posterior pair (testes).	None or may be slight paralysis as above, or of trochlearis.	May be deafness, if lesion be bilateral.	None, except chewing at times.	Normal.	Usually present.	Present. Of cerebellar type.	Normal.	May be slow, rhythmic tremor of arm and leg of opposite side, especially on voluntary motion.
1272 Cerebellum.	None.	None.	Walking and standing.	Normal or slightly exaggerated. Rarely abolished	Usually present.	Cerebellar ataxia with hypotonia almost always present.	Normal.	Nystagmus (80), tendency to fall to one side, occipital headache is frequent, cerebellar fits may occur.
1273 Middle cerebellar peduncles.	None.	None.	Walking, standing and sitting.	Normal or slightly exaggerated.	Usually present.	Usually present with hypotonia of the cerebellar type.	Normal.	Tendency to fall or to turn eyes, head or body to one side. Rotatory movements, more or less pronounced, choreic-spasms in homolateral half of body, and vertical divergence of the eyeballs sometimes occur.

Lesions of inferior cerebellar peduncles cause lateropulsion; those of the superior cerebellar peduncles cause choreiform movements and cerebellar ataxia.

1274 Base of Cranium. Fractures, tumors, etc., at base of skull may cause many of the above symptoms according to their position, but their early and characteristic symptom is paralysis of one or more of the cranial nerves. Symptoms of paralysis predominate over those of irritation.

Small lesions, not so extensive as to involve the entire lateral half of the brain stem, may occur at any point. The symptoms of these lesions depend upon the function (physiology) of the part affected and will naturally vary greatly. The location of such a lesion in a transverse section will depend upon what longitudinal fiber tracts are involved, and in longitudinal section upon what cranial nuclei and nerves parts are involved, as shown by the symptoms present in any case. A study of the figures at the end of this book is essential for the localization of such lesions and will serve this purpose better than a long verbal description.

CHART XXI c

Cerebral Localization: Ganglia at Base

LOCALIZING SYMPTOMS IN LESIONS OF GANGLIA AT BASE OF BRAIN

Seat of Lesion	Diagnostic Symptoms
1275 Optic Thalamus. (Fig. 17)	Symptoms are variable and uncertain. May be hemianopia (pulvinar, and external geniculate involvement) with hemiopic pupillary reaction, contralateral hemianalgesia. Rigidity, choreiform movements, athetosis, and incoordination of contralateral leg, arm, and half of face may be present. The above mentioned motor disturbances occur also in lesions just external to the optic thalamus which involve the fibers connecting the thalamus with the cerebral cortex. Sensory disturbances (pain, hemianesthesia dolorosa, anesthesia, loss of muscle sense) may be present in the same parts. Occasionally a slight irritation of the skin is not felt at all, while a stronger one is felt inordinately. Absence of emotional expression in face, even when not paralyzed. Vaso-motor disturbances may occur in opposite side of body. Isolated analgesia or thermic anesthesia does not occur in lesions above the optic thalamus, but other forms of anesthesia do.
1276 Corpus Striatum. (Fig. 17) — Nucleus Lenticularis and Nucleus Caudatus.	No diagnostic symptom except the hemiplegia due to the involvement of the internal capsule. In rare cases a lesion of the nucleus lenticularis may be of such a form as to injure the anterior and posterior part of the posterior limb of the internal capsule, while its middle part escapes. In such cases there results a hemiplegia which involves the leg and face more than the arm. Dysarthria is a not uncommon symptom and in some cases the symptoms of sensory irritation: muscle spasm and incoordination described under lesions of the optic thalamus have been present. When the ganglia on both sides are affected, voluntary voiding of urine may be impossible, while automatic involuntary voiding may occur at regular intervals.
1276 Corpus Striatum. (Fig. 17) — Internal Capsule.	Lesions in the anterior limb of the internal capsule cause either no symptoms or a paralysis of contralateral half of face. There may be ataxia and athetoid movements. Lesions in the anterior two-thirds of the posterior limb of the internal capsule cause a total contralateral hemiplegia of the body. This hemiplegia consists purely of a muscular paralysis and never produces a paralysis of the cortical functions such as aphasia, alexia, etc; but may produce dysarthria. Lesions in the posterior third of the posterior limb of the internal capsule cause hemianesthesia and loss of muscle sense on the opposite side of the body. Lesions at the extreme posterior end of the posterior limb of the internal capsule, in addition to hemianesthesia, cause contralateral hemianopia, deafness only if the lesion be bilateral and often the symptoms of motor irritation, described under lesions of optic thalamus.
1277 Corpus Callosum.	No diagnostic symptoms.
1278 Island of Riel, Claustrum and External capsule. (Fig. 17)	Lesions in this area produce disturbances of speech, grouped under the general term paraphasia, and may produce anarthria, the result of complete aphasia.
1279 Pituitary Gland.	Hypertrophy, tumor, hemorrhage and some other lesions of the gland associated with excess of secretion may cause acromegaly or gigantism, in addition to a progressive bi-temporal hemianopia, terminating in blindness. A defect or atrophy of the gland associated with a diminution of secretion in early life may cause dwarfism and may produce pituitary eunuchismus or adiposogenital degeneration with excess of fat and a defect in the formation of the genitals. In any case of pituitary disease there may be polyuria, polydipsia and occasionally glycosuria and very rarely an escape of cerebrospinal fluid from the nose (hydrorrhoea nasalis). In some cases of pituitary disease there are no symptoms.
1280 Pineal Gland.	Abnormal growth of hair and deposition of fat. Abnormalities of genitals (at times with attacks of sexual excitement). Excessive growth in height of body (dyspinelismus). In consequence of involvement of adjacent tissue, bilateral ocular paralysis, nystagmus, pupil abnormalities, ataxia, and perhaps disturbances of hearing may be present.

CHART XXI d

Cerebral Localization: Lobes of Brain

LOCALIZING SYMPTOMS IN LESIONS OF CEREBRAL HEMISPHERES

Seat of Lesion		Diagnostic Symptoms
1282 FRONTAL LOBE Contains the centers for all the skilled acts, especially the left lobe. Large lesions in the frontal lobes may cause a change in character and disposition of the patient. Many lesions, especially tumors, cause Jacksonian epilepsy, especially when situated in posterior part of lobe; while lesions in anterior part of lobe may cause epileptiform convulsions. Ataxia sometimes occurs in tumors in the frontal lobe. (Fig. 15)	The ascending frontal convolution.	Lesion in the upper fourth of this convolution may cause Jacksonian epilepsy commencing in, and motor paralysis of, the contralateral leg. Very large lesions (hemorrhage, tumors, etc.) in this region may cause also paralysis of the homolateral leg in a lesser degree.
	Lesions in this region may cause awkwardness (cortical ataxia, apraxia) rather than paralysis.	Lesions in the middle half of the convolution may cause Jacksonian epilepsy commencing in, and awkwardness of or loss of skill or complete paralysis of, the contralateral arm. Very minute lesions in the upper part of this region may affect only the shoulder; in the lower part, only the hand.
		Lesions in the lower fourth of this convolution may cause Jacksonian epilepsy commencing in, and paralysis of, the contralateral half of face and neck. Very minute lesions in the upper part of this region, may affect only the eyes; in the lower and anterior part, the tongue and larynx.
	The base of the middle left frontal convolution.	Small lesions in this area may cause in right-handed persons, argaphia, and in many cases Jacksonian epilepsy, commencing in the contralateral arm.
	The base of the inferior left frontal convolution.	Small lesions in this area may cause, in right-handed persons, motor aphasia, and in many cases Jacksonian epilepsy, commencing in the right side of the face.
1283 PARIETAL LOBE Contains the centers for cutaneous and muscular sensation. Many lesions, especially tumor, cause Jacksonian epilepsy when situated in the anterior portion of this lobe; while lesions in posterior portion may cause epileptiform convulsions. (Fig. 15)	The ascending parietal convolution.	Lesions in the upper fourth of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral leg and foot.
		Lesions in the middle half of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral arm and hand.
		Lesions in the lower fourth of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral half of face.
	The left angular gyrus.	Deep lesions in this region, in right-handed persons may cause alexia and hemianopia.
	The rest of the parietal cortex.	Lesions in this region may cause loss of muscular sense and motor ataxia in the contralateral arm and leg.
1284 TEMPORAL LOBE Contains, on the left side, the centers of sensory speech. Lesions may cause epileptiform convulsions. (Fig. 15)		Lesions in the posterior portion of the left superior temporal convolution in right-handed persons, may cause sensory aphasia (psychic deafness).
1285 OCCIPITAL LOBE Contains the centers of sight. Lesions may cause epileptiform convulsions. (Fig. 15)	Neighborhood of calcarine fissure.	Lesions in this area cause contralateral homonymous hemianopia. A lesion limited to the superior lip of this fissure causes quadrantic hemianopia or tetartanopia of the contralateral lower quadrants of field of vision. A lesion limited to the inferior lip of this fissure causes loss of contralateral upper quadrants of the field of vision.
	Rest of occipital lobe.	Lesions in this area may cause loss of power of recognition of persons and things (psychic blindness).
1286 Cortical Lesions. (Fig. 15)		Many lesions cause a mixture of paralysis and convulsions over a limited area which in some cases may slowly grow larger. The intelligence of the patient is always more or less impaired.

CHART XXII
Cerebro-Spinal Localization

TOPICAL DIAGNOSIS

LOCALIZATION OF LESIONS FROM ANALYSIS OF SYMPTOMS

1290
PARALYSIS

The most important of all localizing symptoms.

- 1292 The reflexes in the paralysed area are abolished (except in 1310 and 1329) A lesion of the peripheral neurons.
  - 1294 Sensation alone, in all its forms is lost or impaired.
  - 1295 Motion alone is lost or impaired. — See Chart XXII a.
  - 1296 Both motion and sensation are lost or impaired.
  - 1297 Special forms of peripheral paralyses. — See Chart XXII b.
- 1293 The reflexes are present (except in 1357 and 1359) A lesion of the central neurons.
  - 1298 Sensory paralysis dominant. Little or no motor paralysis. — See Chart XXII c.
  - 1299 Motor paralysis dominant. Little or no sensory paralysis. — See Chart XXII d.
  - 1300 Both motor and sensory paralysis well marked. — See Chart XXII e.

1291
Jacksonian Epilepsy, together with other symptoms of cerebral disease.

For diseases and lesions accompanied by *motor paralysis* see 469, by *motor spasm* see 570, by *ataxia* see 638, by *tremor* see 639, by *nystagmus* see 640, by *fibrillation* see 641, by *local paralysis* see 636, by *local spasm* see 637, by *disorders of speech* see 735, by *disorders of gait* see 736, by *anesthesia and analgesia* see 810-12, by *disorders of special senses* see 807-9, by *pain* see 931, by *vertigo* see 932, by *mental disorders* see 1036, by *trophic disorders* see 1120, by *vaso-motor disorders* see 1129, by *ganglionic disorders* see 1128, by *syphilis* see 1205, by *abnormal cerebro-spinal fluid* see 1220.

CHART XXII a

Cerebro-Spinal Localization Paralysis with Abolished Reflexes

TOPICAL DIAGNOSIS

LOCALIZATION OF LESION FROM ANALYSIS OF SYMPTOMS

REFLEXES ABOLISHED

	Diagnostic Symptoms and Tests			Localization	
1294 Sensation alone, in all its forms, is lost or impaired.	Area of anesthesia, etc. lies within the area of distribution of one or more nerves.	Onset acute or sub-acute.	Nerve involved, if palpable, is tender on pressure. No symptom of disease of central organs usually, unless nuclei are affected.	Lesion is in one or more sensory cranial nerves or nuclei or sensory end-organ; the nerve affected depending upon its anatomical distribution (822). (Figs. 19-21, 33, 38).	1301
	Area of anesthesia, etc. lies within the area of distribution of one or more nerve roots.	Onset acute or chronic.	Nerves involved, if palpable, are not tender. May be symptoms of disease of central organs.	Lesion is in corresponding sensory nucleus in the brain stem, or in the posterior horn of spinal cord, or in column of Burdach, or in posterior nerve root. (Figs. 19-21, 24-6).	1302
1295 Motion alone is lost or impaired.	The paralysis is limited to muscles supplied by one or more nerves. (Figs. 19-21.)	Onset acute or sub-acute. No fever at onset.	Nerve involved, if palpable, is tender on pressure. No symptoms of disease of central organs. All the muscles supplied by the nerve are paralysed, usually.	Lesion is in one or more motor cranial nerves, or a mild lesion of mixed spinal nerves; the nerve affected is the nerve supplying the paralysed muscles (489-93). (Figs. 19-21, 33, 38).	1303
	The paralysis is limited to muscles supplied by one or more nerve roots. (Figs. 19-21)	Onset acute or chronic. May be fever at onset.	Nerve involved, if palpable, not tender. May be symptoms of disease of central organs. Often only a portion of the muscles innervated by the nucleus are paralysed.	Lesion is in corresponding motor nucleus within brain stem, or in anterior horn of spinal cord, or in the anterior nerve root (493-5). Figs.19-21, 24-6).	1304
1296 Both motion and sensation are lost or impaired. — UNILATERAL	Motor and sensory paralysis is within the area of distribution of one spinal nerve.	Onset acute or sub-acute. No fever at onset.	Nerve involved; tender on pressure. No symptoms of disease of central organs.	Lesion in one spinal nerve (489). (Figs. 33, 38).	1305
	Motor or sensory paralysis is within the area of distribution of several nerves from one plexus.			Lesion in brachial or lumbar plexus (490). (Fig. 32, 38).	1306
1296 Both motion and sensation are lost or impaired. — BILATERAL	Motor and sensory paralysis extends over legs or arms or both, or even more generally.	Onset acute or sub-acute. May be fever at onset.	Nerves involved not tender. There are disturbances of organic reflexes and other symptoms of organic disease of central organs.	Muscles show weakness, tenderness and rapid atrophy.	Lesion of many spinal and (rarely) cranial nerves also (multiple neuritis) (488).	1307
				Legs alone are paralysed and exhibit trophic disturbances. Anesthesia of rectum and bladder.	Great pain. May be deformity of lumbar spines. Symptoms less symmetrical and bed-sores less common than in lumbar lesions. Domain of anterior crural nerve may be normal when lesion is low.	Lesion of cauda equina (487). (Fig. 29).	1308
					Little pain. May be deformity of lower dorsal spines. Symptoms symmetrical. Bed-sores always present. No portion of legs escape.	Lesion of lumbar enlargement of spinal cord (484-7). (Fig. 24-6).	1309
				Both legs and arms are paralyzed. There are trophic disturbances in arms but not in legs. Reflexes are abolished in arms, exaggerated in legs (548-51).		Lesion of cervical enlargement of spinal cord (Fig. 24-6).	1310

CHART XXII b

Cerebro-Spinal Localization

Comprising Numbers 1297 and 1315 to 1317 on left side of chart
and 1318 to 1336 on right margin

TOPICAL I

LOCALIZATION OF LESION FR

PERIPHERAL PARALYSIS W

DIAGNOSTIC SYM

- 1297 SPECIAL FORMS OF PERIPHERAL PARALYSIS. REFLEXES ABOLISHED IN PARALYSED AREA, EXCEPT IN 1329.
  - 1315 DISTURBANCES OF VISION. (807.)
    - Blindness of entire field of vision of one eye is present. light.
    - Bitemporal hemianopia is present. The outer half of eac is present.
    - Nasal hemianopia is present. The inner half of field of is present.
    - Homonymous hemianopia is present. Identical halves (r pupillary reflex is present i.e. reflex is absent when para
  - 1316 PARALYSIS OF OCULAR MUSCLES (700).
    - All muscles of one eye paralysed. Eyeball protruded or
    - All muscles supplied by third cranial nerve are paralysed at once.
      - No hemiplegia. Other crani
      - Paralysis of arm and leg of op
      - Tremor of arm and leg of op causing ataxia.
    - Partial or progressive paralysis of muscles supplied by thir
    - Paralysis of external rectus muscle.
      - No hemiplegia. Other crani
      - Hemiplegia often combined v of conjugate deviation of be involved.
  - 1317 FACIAL PARALYSIS (703).
    - Lower branch of facial only, or mainly, paralysed. Other symptoms of disease o tion never present. Reflex
    - Both lower and upper branches of facial nerve equally paralysed.
      - Paralysis of arm and leg of op
      - No hemiplegia. Chronic cou and abducens, may be affec
      - Associated with unilateral dea
      - No deafness but hyperakusis notes, and often the high n times absence of secretion
      - Hyperakusis. Loss of taste i
      - No hyperakusis. Loss of tast
      - No hyperakusis. No loss of t

GNOSIS

M ANALYSIS OF SYMPTOMS

H ABOLISHED REFLEXES

OMS AND TESTS	LOCALIZATION	
tic nerve is atrophied. Pupil does not respond to	Lesion in optic nerve (897-8).	1318
eld of vision is blind. Hemiopic pupillary reflex	Lesion is in the central part of optic chiasm (362, 815, 860, 892).	1319
on of one eye is blind. Hemiopic pupillary reflex	Lesion is in outer margin of optic chiasm (362, 815, 861).	1320
or left) of each field of vision is blind. Hemiopic d half of retina is excited by light.	Lesion is in the optic tract or external geniculate body of opposite side (858, 893).	1321
er evidence of disease within orbit.	Lesion within the orbit (914).	1322
nerves paralysed.	Lesion of 3rd cranial nerve trunk or nucleus (700). (Fig. 18.)	1323
site side.	Lesion involving crus cerebri (676).	1324
te side present at rest and exaggerated on motion,	Lesion of red nucleus or rubro-spinal tract on same side as motor oculi paralysis (441, 676).	1325
anial nerve (700).	Lesion of 3rd cranial nucleus, in whole or in part (700). (Fig. 18.)	1326
nerves paralysed, especially the facial.	Lesion of 6th cranial nerve or nucleus (1330-1). (Figs. 19, 20.)	1327
hemianesthesia of opposite side. Loss of power to right or left. Facial or auditory nerve may	Diffuse lesion of Pons Varolii (539, 883). (Figs. 19, 20.)	1328
e brain present. Electrical reaction of degenera- resent.	Lesion above nucleus of facial nerve in cerebral hemispheres or in crura cerebri. (Fig. 15, 19).	1329
ite side. Often abducens paralysis.	Lesion in Pons Varolii. (Figs. 19, 20.)	1330
usually. Other cranial nerves, especially auditory.	Lesion of nucleus of facial nerve. (Figs. 19, 20).	1331
s and vertigo without disease of the ear.	Lesion of facial nerve trunk at base of brain (Fig. 19).	1332
tinnitus aurium, due to stapedius paralysis. Low also, are painful to hear. No loss of taste. At ears.	Lesion of nerve above geniculate ganglion (928). (Fig. 36).	1333
terior two-thirds of tongue of same side.	Lesion of facial nerve between geniculate ganglion and stapedius branch. (Fig. 36).	1334
anterior two-thirds of tongue of same side.	Lesion of facial nerve between stapedius and chorda tympani branches. (Fig. 36).	1335
. Tenderness near stylo-mastoid foramen.	Lesion of facial nerve below chorda tympani branch. (Fig. 36).	1336

CHART XXII c

Cerebro-Spinal Localization

Comprising Numbers 1298 and 1340 to 1346 on left side of Chart
and 1347 to 1369 on right margin.

DIAGNOSTIC SYMPTOMS AND TESTS

1298 SENSORY PARALYSIS DOMINANT. LITTLE OR NO MOTOR PARALYSIS. TENDON REFLEXES PRESENT OR EXAGGERATED.

- 1340 ANESTHESIA with or without ANALGESIA.
  - Limited to one or both legs.
    - Marked ataxia. — Anesthesia marked, bilateral. of muscle sense.
    - Slight ataxia. — Anesthesia slight and most ma be cerebral symptoms, Jacks
  - Limited to one arm. — Slight ataxia. — Anesthesia slight, most marked cerebral symptoms (Jacksoni
  - In both arms and both legs. — Marked ataxia. — May be other spinal symptom in arms and legs.
  - In arm and leg of same side.
    - Marked ataxia. — May be other spinal symptom in arm and leg.
    - Slight ataxia. — Anesthesia slight, most marked i be other cerebral symptoms,
  - In arm and leg of one side and in other side of face. — Moderate ataxia. — May be paralysis of other cran the eyeballs.
  - In arm, leg and face of same side. — Slight ataxia.
    - No Jacksonian epilepsy. Hemia
    - Jacksonian epilepsy common.
- 1341 ANALGESIA with THERMIC ANESTHESIA, but little or no tactile anesthesia, is present. DISSOCIATION OF SENSATION.
  - In one or both legs.
    - Usually unilateral. — No trophic disturbances. No d
    - Usually bilateral. — Trophic disturbances in legs. usually abolished, especially in
  - In one or both arms.
    - Usually unilateral. Leg of same side also involved. — No trophic disturbances. Often
    - Usually bilateral. Legs of normal sensibility. — Trophic disturbances in arms. especially in advanced cases.
  - In arms, or legs, or both.
    - Bilateral usually, marked ataxia. — May be other spinal symptoms. plegia).
    - Unilateral, slight ataxia.
      - Hemianopia and anesthesia usua
      - Jacksonian epilepsy and other c present.
      - In contralateral arm and leg with
- 1342 HOMONYMOUS HEMIANOPIA. — Identical halves of each field of vision (right or left) are blind. No hemiopic pupillary
- 1343 HOMONYMOUS TETARTANOPIA, QUADRANT HEMIANOPIA. — Identical quadrants of each field of vision (right or left) are blind. No hemiopic pup anesthesia or other paralysis. May be other cerebral symptoms.
- 1344 PSYCHIC BLINDNESS. — Patient is not blind, but cannot recognize things by sight, though he may by touch or h
- 1345 SENSORY APHASIA. — Patient is not deaf, but cannot understand words spoken to him, although he understa memory for spoken words.
- 1346 ASTEREOGNOSIS. — Patient is not anesthetic, or very slightly so, but cannot recognize objects by the sense

IAGNOSIS

OM ANALYSIS OF SYMPTOMS

GGERATED REFLEXES

	LOCALIZATION	
be other spinal symptoms, especially loss	Lesion in one or both posterior columns of cord in dorsal region. Same side if unilateral (654a, 785). (Figs. 24-6.)	1347
in foot. Almost always unilateral. May epilepsy, etc.	Lesion in upper one-fourth of posterior central convolution in contralateral cerebral cortex. (Fig. 15.)	1348
and, astereognosis marked. May be other ilepsy). Usually some motor paralysis.	Lesion in middle one-half of posterior central convolution in contralateral cerebral cortex. (Fig. 15.)	1349
Dyspnoea common. Loss of muscle sense	Lesion of posterior columns of cord in cervical region (654a, 785). (Figs. 24-6.)	1350
Dyspnoea common. Loss of muscle sense	Lesion of posterior column of cord on same side, in cervical region (654a, 785). (Figs. 24-6.)	1351
d and foot. Astereognosis marked. May cially Jacksonian epilepsy.	Lesion in upper three-fourths of posterior central convolution of contralateral cerebral cortex. (Fig. 15.)	1352
rves. Paralysis of conjugate deviation of	Lesion in tegmentum of pons Varolii on same side as the facial anesthesia (883). (Fig. 20.)	1353
common.	Lesion of posterior part of internal capsule of contralateral hemisphere (857, 1276). (Fig. 17.)	1354
mianopia. Mental deterioration.	Lesion of superior parietal lobule of contralateral hemisphere (657). (Fig. 15.)	1355
bance of organic reflexes. Usually ataxia.	Lesion in periphery of opposite lateral column of cord in dorsal region (1360). (Figs. 24-6.)	1356
nic reflexes disordered. Tendon reflexes anced cases. (Figs. 24-6.)	Lesion in central gray matter (anterior commissure) of cord in lumbar enlargement. In central gliosis the lesion may extend upwards to the cervical enlargement and involve the arms secondarily (837-9, 1359.)	1357
xia without loss of muscle sense.	Lesion in periphery of the opposite, or of both, lateral columns of the cord in the cervical region (1360). (Figs. 24-6.)	1358
don reflexes usually abolished in arms,	Lesion in central gray matter (anterior commissure) of the cord in cervical enlargement (Syringomyelia) (552, 693, 837,-9 1009, 1170, 1187, 1357). (Figs. 24-6.)	1359
ways some motor paralysis (spastic para-	Lesion of lateral columns of cord (654, 1212, 1356, 1358, 1396). (Figs. 24-6.)	1360
resent. Other cerebral symptoms.	Lesion of posterior part of contralateral internal capsule (857). (Fig. 17.)	1361
l symptoms usually present. Anesthesia	Lesion of inferior parietal lobule of contralateral hemisphere (657). (Fig. 15.)	1362
fness.	Lesion of ponto-cerebellar angle on side of deafness (438).	1363
. Other cerebral symptoms.	Lesion of edges of calcarine fissure of occipital lobe, or of fasciculus of Gratiolet of contralateral cerebral hemisphere (362, 815, 890, 1285). (Fig. 16.)	1364
y reflex. No hemi- Lower quadrant of field of vision.	Lesion of upper lip of contralateral calcarine fissure (363, 815, 1285). (Fig. 16.)	1365
y reflex. No hemi- Upper quadrant of field of vision.	Lesion of lower lip of contralateral calcarine fissure. (Fig. 16.)	1366
g. Has forgotten what he has seen.	Lesion of cortex of occipital lobe of left cerebral hemisphere (232, 1285). (Fig. 15.)	1367
hem when he sees them written. Has no	Lesion of cortex or sub-cortex of posterior part of left superior temporal convolution (222, 772). (Fig. 15.)	1368
uch, although he can by the sense of sight.	Lesion in cortex or sub-cortex of the posterior central convolution of contralateral hemisphere (229, 354). (Fig. 15.)	1369

CHART XXII d

Cerebro-Spinal Localization

Comprising Numbers 1299 on left side of chart
and 1372 to 1391 on right margin.

DIAGNOSTIC SYMPTOMS AND TESTS

1299
MOTOR PARALYSIS DOMINANT. LITTLE OR NO SENSORY PARALYSIS. TENDON REFLEXES PRESENT OR EXAGGERATED

- Limited to one or both legs. Organic reflexes not disordered.
  - Symptoms bilateral usually. — May be other spinal symptoms. Oft of sensation in legs.
  - Symptoms unilateral usually. — May be other cerebral symptoms, esp
- Limited to both arms and both legs Organic reflexes not disordered.
  - No sensory paralysis. No cerebral symptoms. Often ataxia and arms and legs.
  - Usually some sensory paralysis. Dysarthria and dysphagia. Paral ing with position of lesion.
- Limited to one arm. — Occasionally some slight sensory paralysis. Jacksonian epilepsy an common.
- Limited to arm and leg of same side.
  - Dissociation of sensation and ataxia may be present. Organic re cerebral symptoms.
  - Usually some sensory symptoms. Dysarthria and dysphagia co cranial nerves frequent.
  - Usually some sensory symptoms. Jacksonian epilepsy and other syn
- Limited to lower branch of facial nerve.
- Limited to arm and lower branch of facial nerve of same side. — Jacksonian epilepsy and other sym common. Often complicated with
- Limited to arm and leg of same side and hypoglossus nerve of opposite side.
- Limited to arm and leg of same side and lower branch of facial nerve of opposite side. — Usually some sensory symptoms. L Paralysis of some other cranial n abducens paralysis.
- Limited to arm and leg of same side and motor oculi nerve of opposite side. — Usually some sensory symptoms. I nerves common.
- Limited to arm and leg and lower branch of facial nerve on same side.
  - Symptoms of paralysis rather than of irritation. Not progressive.
    - Usually other cerebral symptoms pr toms.
    - Often sensory symptoms present. P pression on opposite side of face, a
  - Symptoms of irritation. Jacksonian epilepsy. — No objective sensory symptoms. Oft
- DYSARTHRIA and DYSPHAGIA — Paralysis of some of the cranial nerves and usually of arm and leg also.
- AGRAPHIA — Loss of power of writing, although arm is not paralysed.
- MOTOR APHASIA — Loss of power of speaking some or all words. Limited vocabulary. muscles of speech not paralyzed.
- ALEXIA — Inability to read, although patient can see and can speak.

AL DIAGNOSIS

FROM ANALYSIS OF SYMPTOMS

TH EXAGGERATED REFLEXES

	LOCALIZATION	
ataxia and dissociation	Lesion of homolateral, or of both lateral, columns of cord in dorsal region (1356, 1358, 1360). (Figs. 25-7.)	1372
ally Jacksonian epilepsy.	Lesion of upper part of anterior central convolution of contralateral hemisphere, cortical or sub-cortical (leg center). (Fig. 15.)	1373
ociation of sensation in	Lesion of lateral columns of the cord in the cervical region (525). (Figs. 25-7.)	1374
of cranial nerves vary-	Lesion of the brain stem (involvement of pyramidal tract in the medulla, pons or crura cerebri). (Figs. 19-22.)	1375
her cerebral symptoms	Lesion in cortex or sub-cortex of middle one-half of anterior central convolution of contralateral hemisphere (arm center). (Fig. 15.)	1376
s not disordered. No	Lesion of contralateral lateral column of cord in cervical region (1131, 1141). (Figs. 25-7.)	1377
n. Paralysis of some	Lesion in the brain stem (involving the pyramidal tract). (Figs. 19-22.)	1378
ms of cortical disease.	Lesion in cortex or sub-cortex of upper three-fourths of anterior central convolution of contralateral hemisphere. (Fig. 15.)	1379
ns of cortical disease or aphasia.	Lesion in cortex or sub-cortex of inferior part of anterior central convolution of contralateral hemisphere (face center). (Fig. 15.)	1380
	Lesion of cortex or sub-cortex of lower three-fourths of anterior central convolution of contralateral hemisphere (arm and face centers). (Fig. 15.)	1381
rthria and dysphagia. s common, especially	Lesion of medulla on same side as the hypoglossus paralysis (rare condition). (Fig. 21.)	1382
	Lesion in bridge portion of pons on same side as the facial paralysis. (Fig. 20.)	1383
lysis of other cranial	Lesion in pes cruris cerebri on same side as the motor oculi paralysis. (Fig. 19.)	1384
. No sensory symp-	Lesion in anterior part of posterior limb of internal capsule of opposite hemisphere. (Fig. 17.)	1385
ysis of emotional ex- sis, etc.	Lesion in posterior part of optic thalamus and corpus striatum of opposite hemisphere. (Fig. 17.)	1386
motor aphasia.	Lesion throughout anterior central convolution of contralateral hemisphere (cortex or sub-cortex). (Fig. 15.)	1387
	Lesion in tegmentum of pons or medulla (284-5). (Figs. 20-1.)	1388
	Cortical or sub-cortical lesion at base of middle frontal convolution of left cerebral hemisphere in right handed person (227, 776). (Fig. 15.)	1389
ids can be made and	Cortical or sub-cortical lesion at base of inferior left frontal convolution in right handed person (221, 771). (Fig. 15.)	1390
	Sub-cortical lesion of left angular convolution in right handed person (228, 773). Fig. 15.)	1391

CHART XXII e

Cerebro-Spinal Localization

TOPICAL DIAGNOSIS

LOCALIZATION OF LESION FROM ANALYSIS OF SYMPTOMS

MOTOR AND SENSORY PARALYSIS WITH EXAGGERATED REFLEXES

JACKSONIAN EPILEPSY

		Diagnostic Symptoms and Tests	Localization	
1300 Both motor and sensory paralysis well marked. Reflexes present or exaggerated, except in 1396.	Limited to both legs.	Paralysis severe. No ataxia. Organic reflexes much disordered. Some of the trunk reflexes are lost. Vertical extent of lesion is shown by the absence of the different trunk reflexes. Upper limit of lesion shown by the zone of hyperesthesia, limiting the anesthesia above.	Transverse lesion of spinal cord in dorsal region. (Myelitis.) (516-9, 829).	1395
		Paralysis not so extreme. Marked ataxia. Loss of muscle sense. Organic reflexes not at all, or slightly, disordered. Trunk reflexes not abolished. Knee-jerks and other leg reflexes may be increased or abolished.	Lesion both in lateral and posterior columns of cord. (Ataxic Paraplegia.) (526, 660, 796) (Figs. 25-7.)	1396
	Limited to both arms and both legs.	No involvement of cranial nerves. Priapism. Dyspnoea. Very dangerous, usually fatal.	Transverse lesion of spinal cord in cervical region. (512-5, 828) (Figs. 25-6).	1397
		Involvement of some cranial nerves. Dysarthria and dysphagia. Very dangerous, usually fatal.	Lesion on both sides of brain stem (medulla, pons or crura cerebri, according to cranial nerves involved). (Figs. 19-21).	1398
1291 JACKSONIAN EPILEPSY		Spasmodic twitching of head and eyes to one side. Twitching may remain limited to these muscles or may extend to other muscles of face and neck and arm and later to leg of same side or may finally extend to muscles of both sides of body.	Lesion in or near base of middle frontal convolution of contralateral hemisphere. (Fig. 15).	1399
		Spasmodic twitching commences in one side of face. Twitching may remain limited to these muscles or may extend to others as above.	Lesion in or near lower quarter of the central convolutions of contralateral hemisphere. (Fig. 15).	1400
		Spasmodic twitching in hand or arm. Twitching may remain limited to these muscles or may extend to face or to leg or to both simultaneously of same side and may later extend to muscles of other side of body also.	Lesion in or near middle half of the central convolutions of contralateral hemisphere. (Fig. 15).	1401
		Spasmodic twitching of foot or leg. Twitching may remain limited to these muscles, or may extend to arm and later to face of same side and later to muscles of other side of body. (Figs. 15, 16).	Lesion in or near upper quarter of central convolutions or paracentral lobule of opposite hemisphere.	1402
		Spasmodic twitching, commencing simultaneously, in arm and face of same side, which later extends to muscles of the leg of the same side and still later to muscles of the opposite side of the body.	Lesion near and equally distant from motor area of face and arm in contralateral hemisphere. (Fig. 15).	1403
		Spasmodic twitching commencing in arm and leg of same side, which may later extend to face of same side and may later extend to muscles of the other side of body.	Lesion near and equally distant from motor area of arm and leg in contralateral hemisphere. (Fig 15).	1404
		Spasmodic twitching commencing in face and arm and leg of same side, which may later extend to muscles of opposite side.	Lesion in inferior parietal lobule of contralateral hemisphere. (Fig. 15).	1405

PLATES

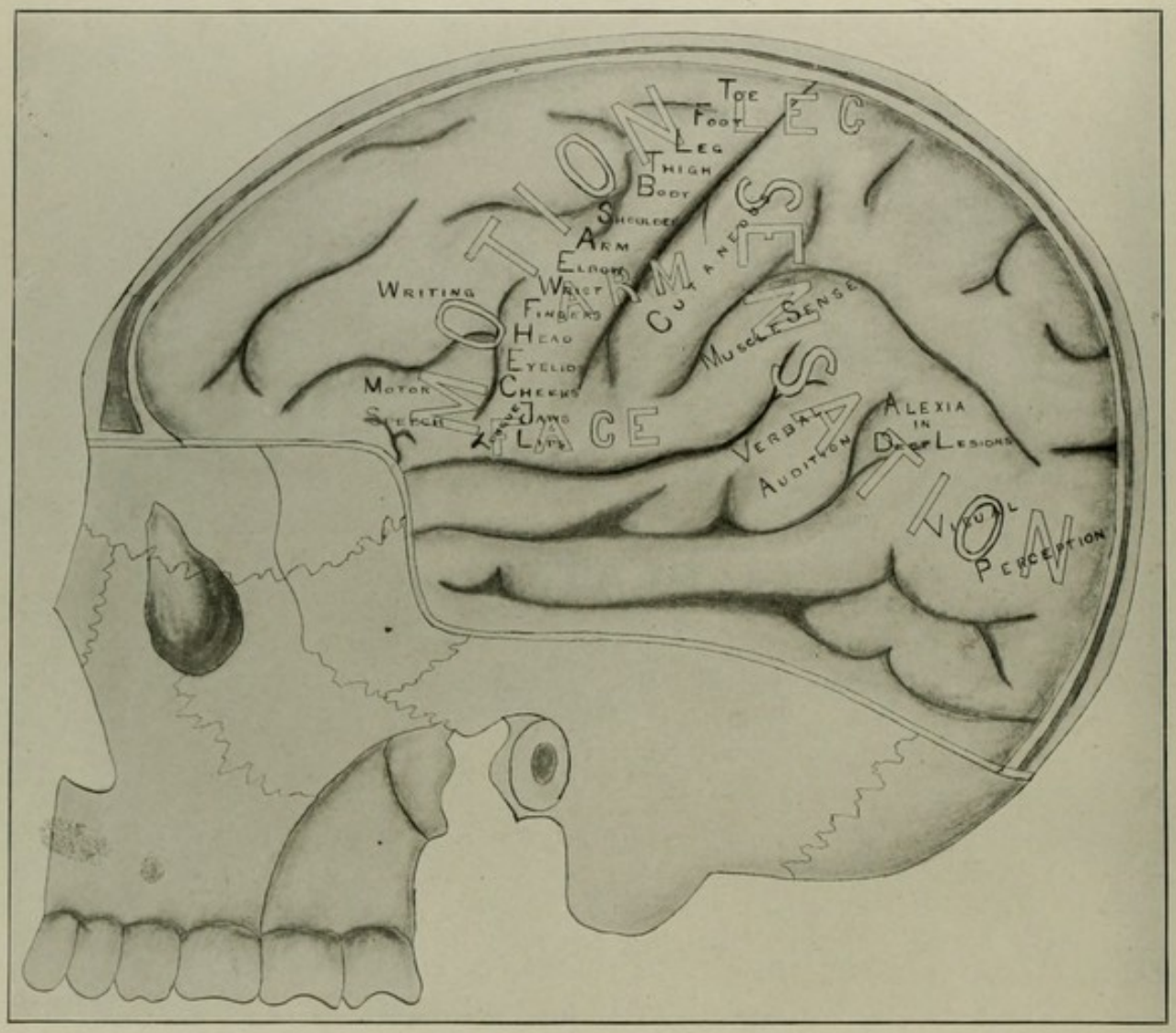


Fig. 15

Schematic representation of the convex surface of the left cerebral hemisphere, showing the motor and sensory areas, and the location of the cortical functions.

See 1282–6, 1348–9, 1352, 1355, 1362, 1367–9, 1373, 1376, 1379, 1380–1, 1387, 1389–91, 1400–5.

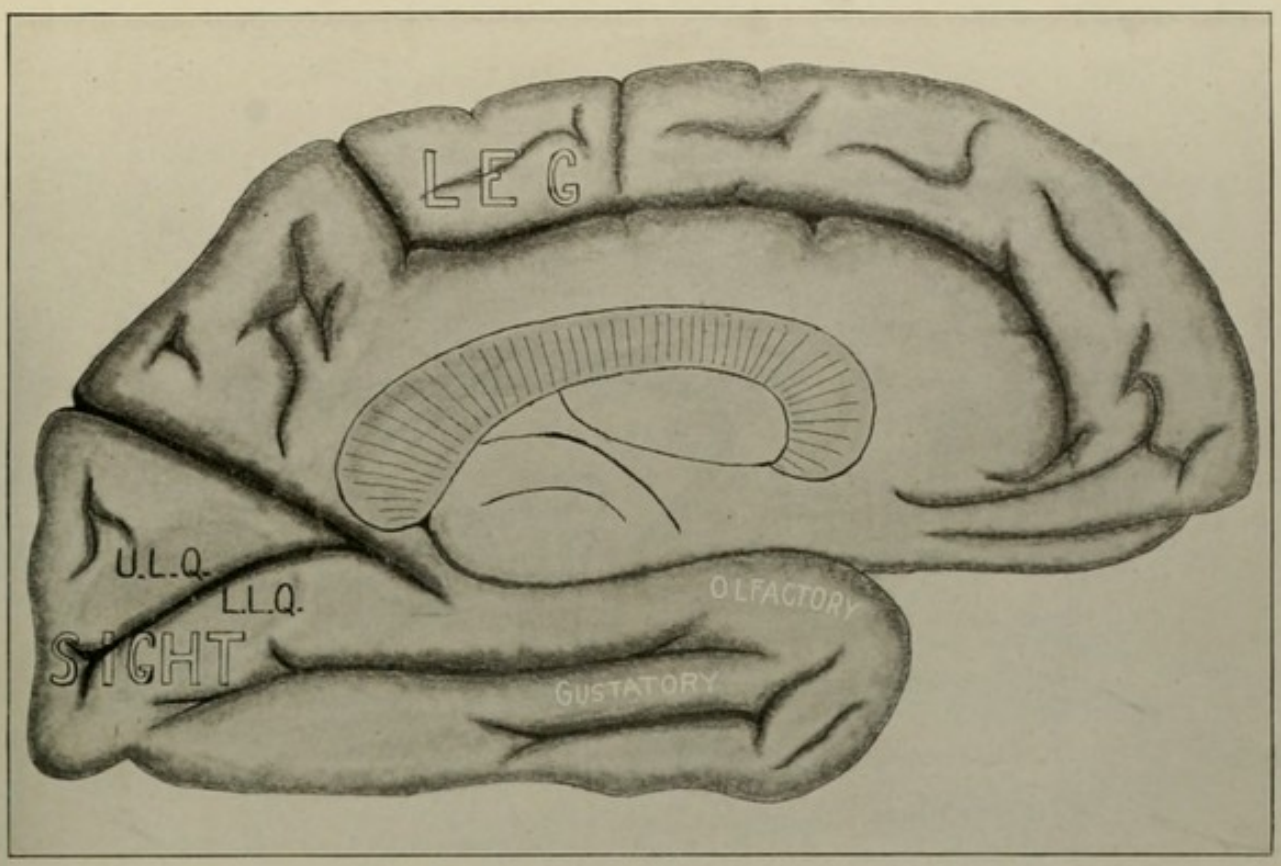


Fig. 16

Schematic representation of the median surface of the left cerebral hemisphere. U. L. Q.=Upper left quadrant of retina. L. L. Q.=Lower left quadrant of retina.

See 852-3, 856, 1285, 1364-6, 1402.

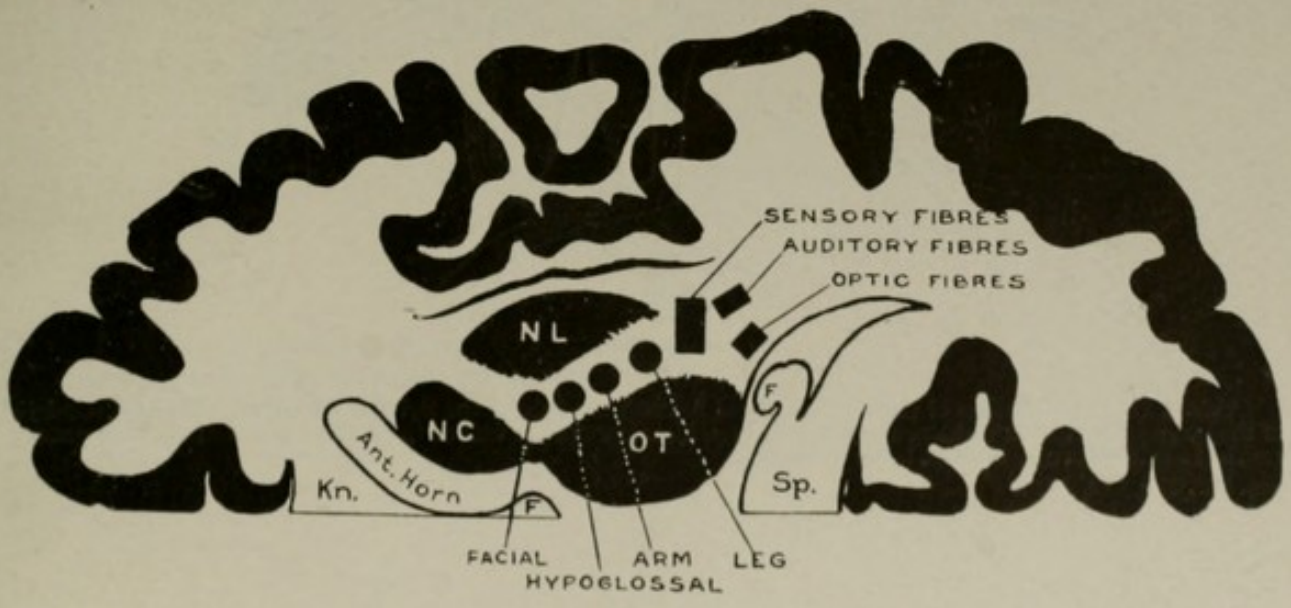


Fig. 17

Horizontal Section through Right Hemisphere showing the principal tracts situated in the Internal Capsule; Kn, Genu of Corpus Callosum; F, Fornix; NC, Caudate Nucleus; NL, Lenticular Nucleus; OT, Optic Thalamus. Sp, Splenium of Corpus Callosum.

See 1275-6, 1354, 1361, 1385-6.

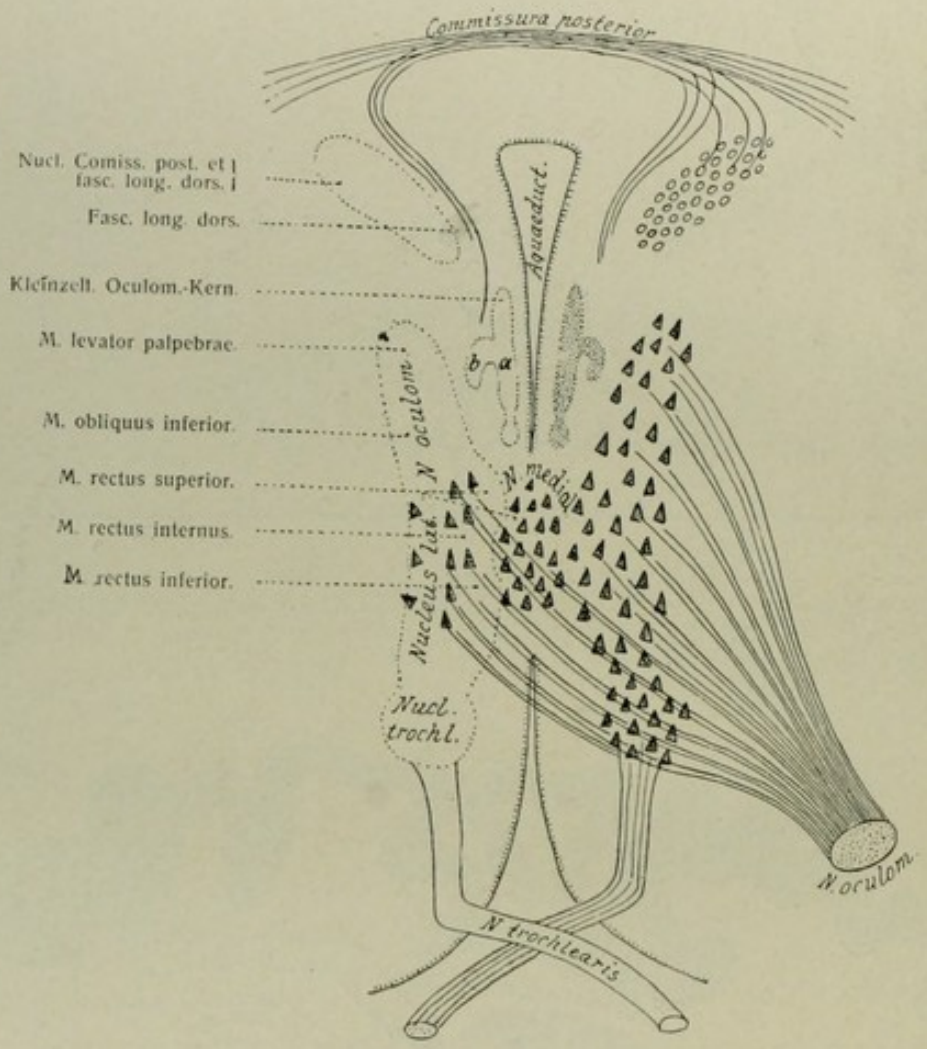


FIG. 18

Schematic representation of the nuclei situated beneath the floor of the Sylvian aqueduct, showing the origin of the posterior commissure, the oculo-motor and trochlearis nerves, as well as the nuclear localization of the centers for the individual ocular muscles (after Edinger).

Sec. 692, 700, 816, 1316.

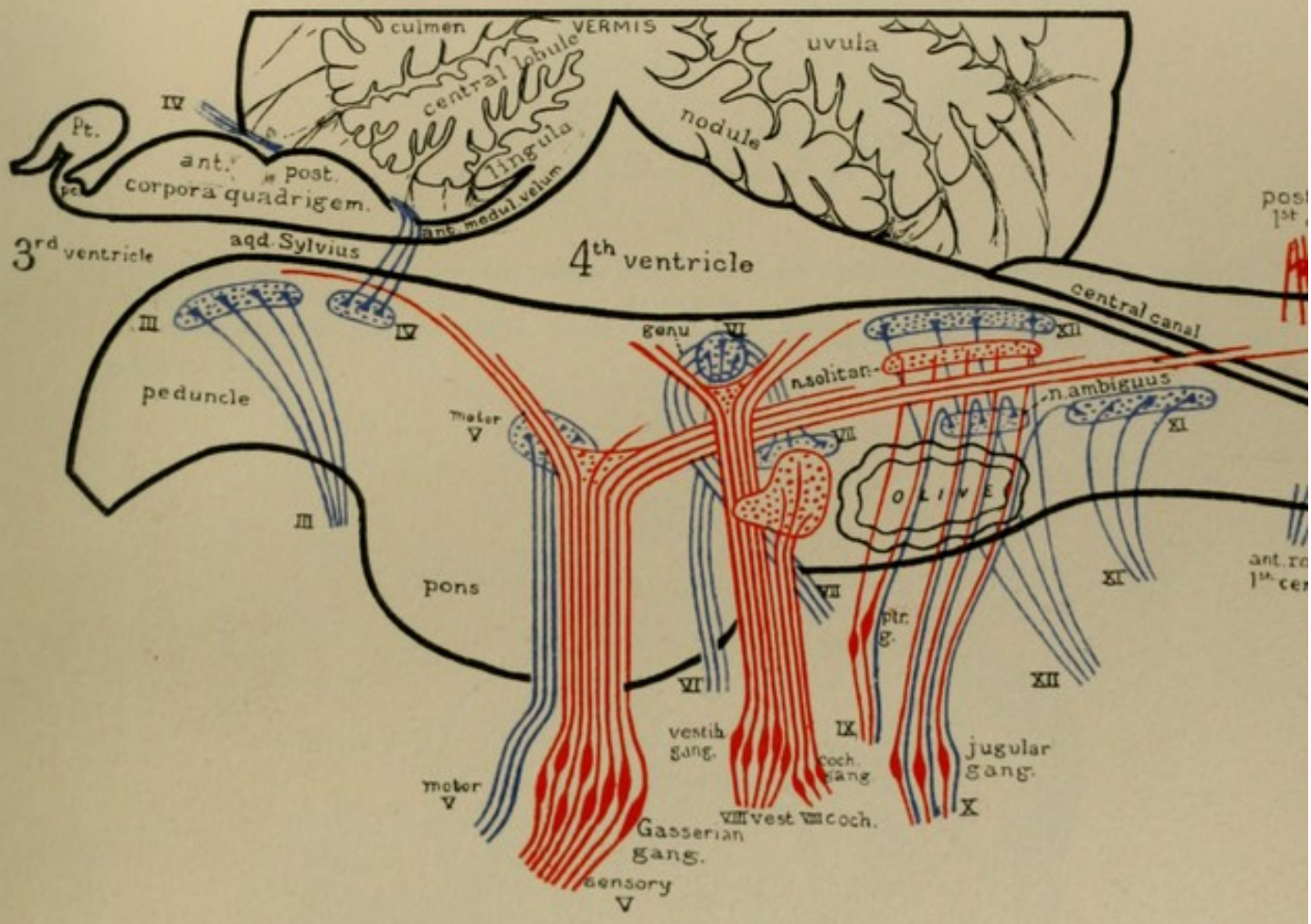


Fig. 19

Schematic representation of brain stem; showing nuclei and nerve roots. The sensory nuclei and nerve roots are colored red, the motor blue. See 1301-4, 1323-32, 1353, 1375, 1378, 1398.

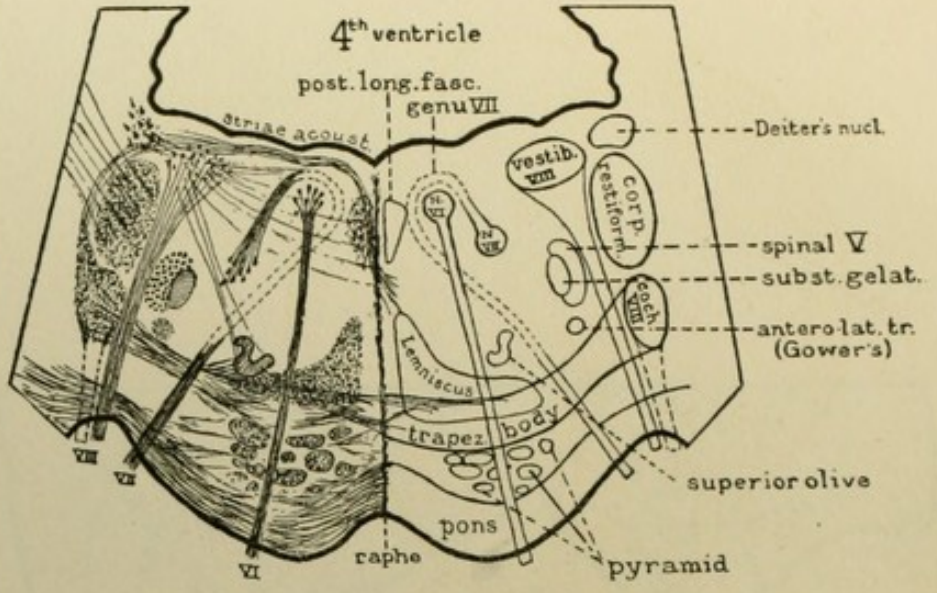


FIG. 20

Diagrammatic transverse section through the pons at a level slightly posterior to the superficial origin of the trigeminus.

See 1269, 1301-4, 1327-30, 1353, 1383, 1388, 1398.

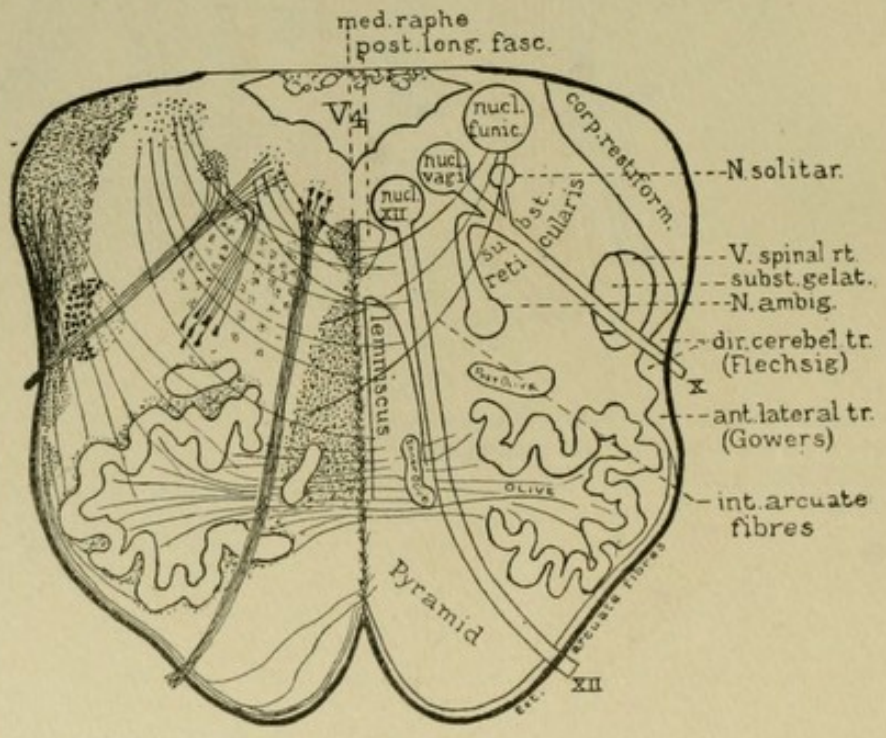


FIG. 21

Diagrammatic transverse section through the medulla, approximately near its middle.

See 1268, 1301-4, 1382, 1388, 1398.

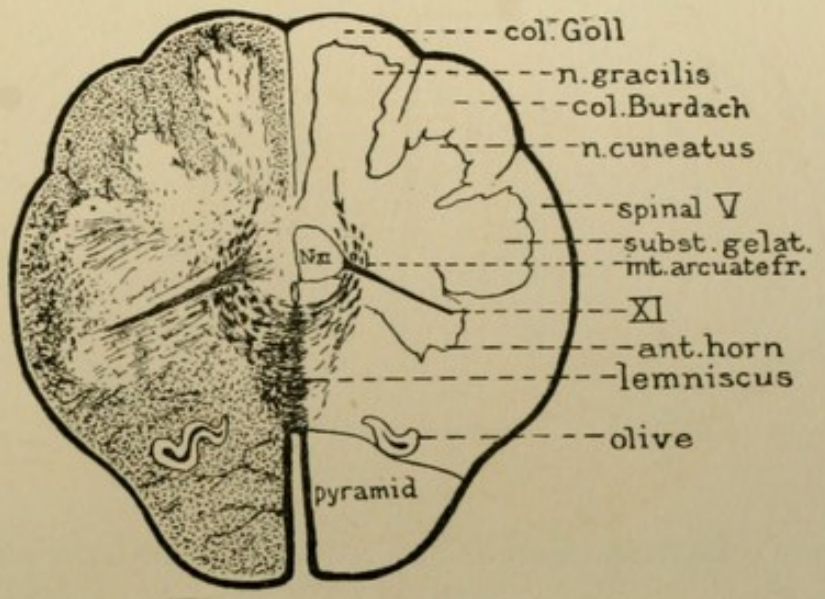


Fig. 22

Transverse section of medulla just above motor decussation and just above line of junction with the cord, showing the sensory decussation and the topography of the lowest level of the medulla.

See 1268

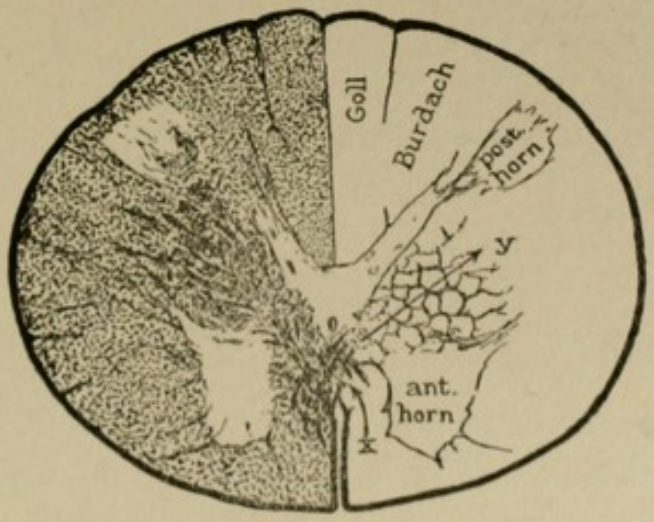


Fig. 23

Transverse section of the cord just at the line of junction with the medulla, showing the motor decussation and the topography of the uppermost level of the cord.

See 1268

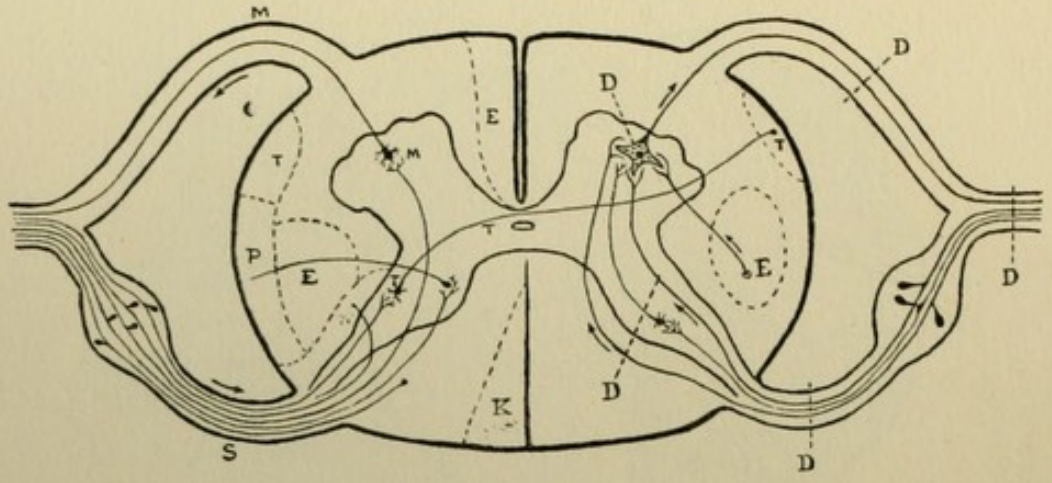


FIG. 24

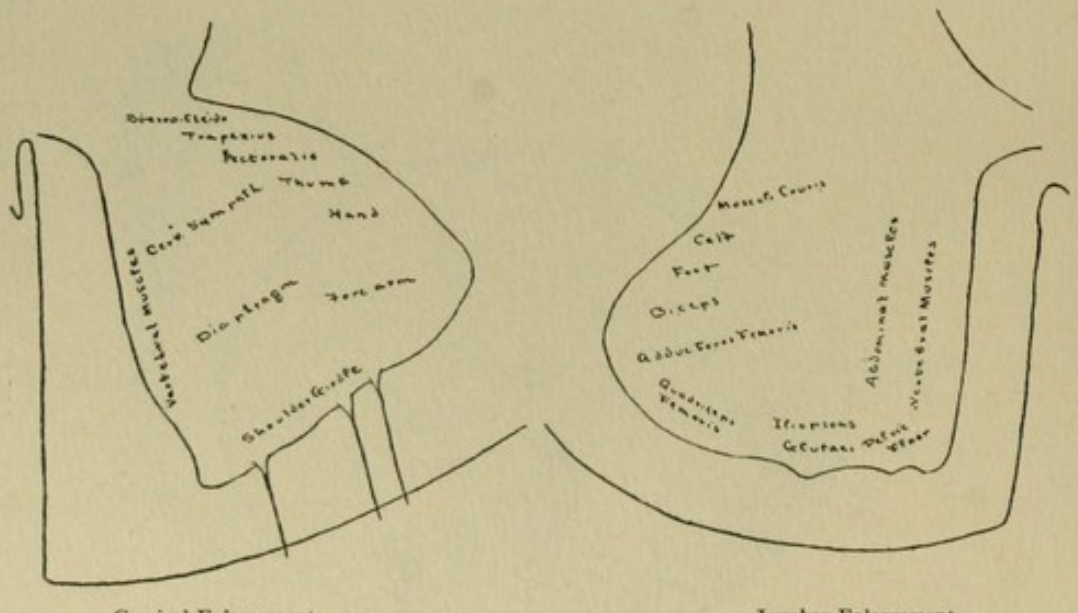
DIAGRAMMATIC SECTION OF THE SPINAL CORD TO ILLUSTRATE ITS PHYSIOLOGY

Left side shows situation of lesions causing disorders of motion and sensation.

Right side shows situation of lesions causing disorders of reflex activity.

Destructive lesions at M or E cause diminution, slight irritative lesions, exaggeration, of motion. Destructive lesions at S cause permanent anaesthesia, analgesia, thermic anesthesia and loss of muscle sense. Destructive lesions at T cause analgesia and thermic anesthesia. Destructive lesions at P cause ataxia. Destructive lesions at K cause loss of muscle sense, ataxia and anaesthesia. Irritative lesions at S, K, T, or P, may cause exaggeration, or perversion, or both, of sensation. Destructive lesions at D cause diminution, and at E, exaggeration, of reflex activity. Slight irritative lesions at D cause exaggeration, and at E diminution, of reflex activity.

Symptoms of lesions at M are described in 252, 263, 495, 547, 789; 1148-9, 1233 and 1304 at E in 251, 254, 256, 525-6, 796-7 and 1212, 1372-4-7; at S in 824; at T in 1356-8-60; at P in 281, 654; at K in 280, 654a, 785, 1302, 1347 and 1350-1, 1396. The results of lesions at D and E are discussed in Chart V a.



Cervical Enlargement

Lumbar Enlargement

Fig. 25

LOCALIZATION OF NUCLEI IN THE ANTERIOR HORNS OF THE SPINAL CORD

(After Edinger modified from Sano.)

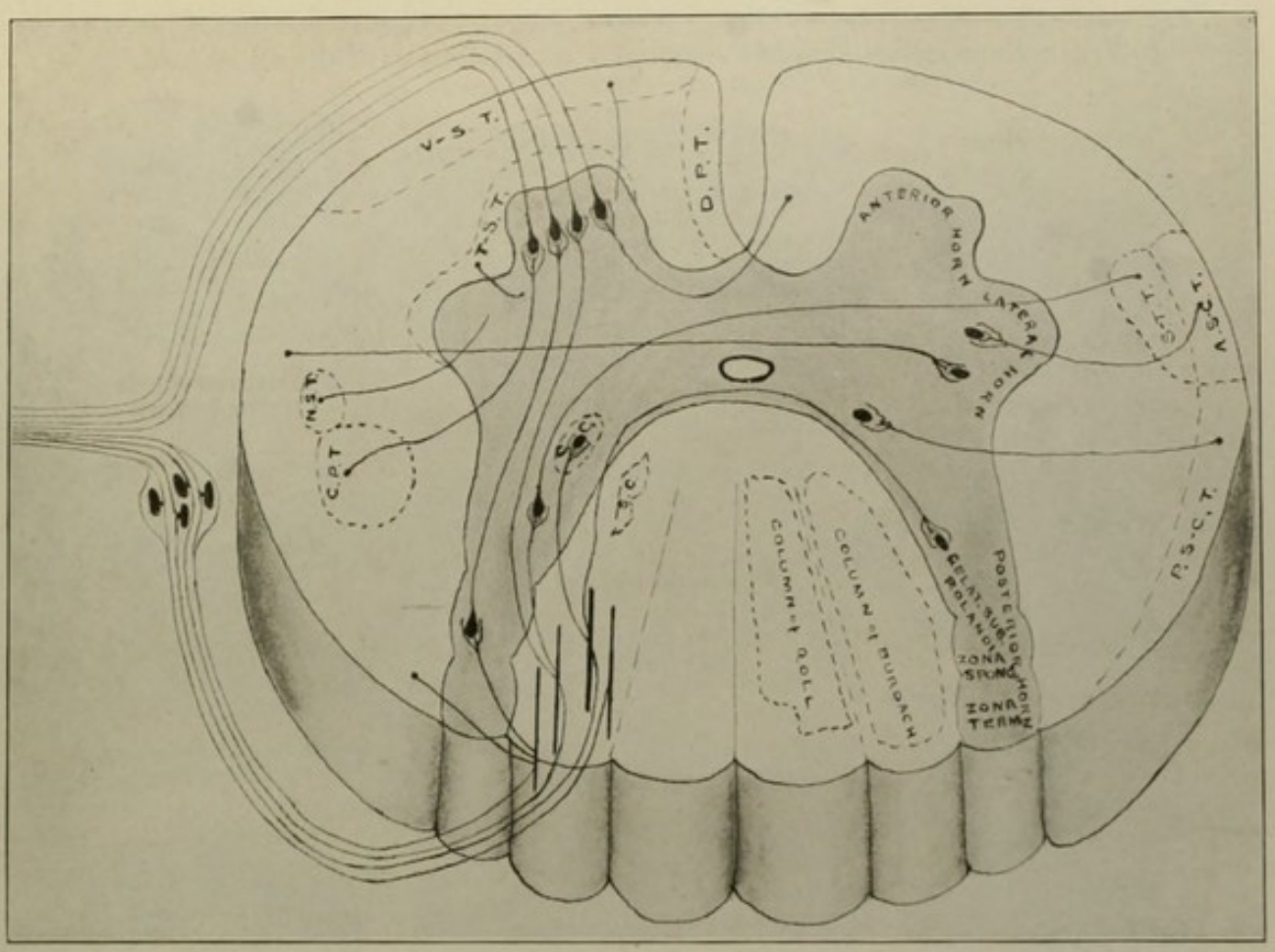


Fig. 26

A SCHEMATIC REPRESENTATION OF A TRANSVERSE SECTION OF THE SPINAL CORD; SEVERAL LEVELS BEING COMBINED INTO ONE

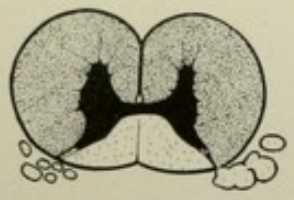
Descending Tracts		Ascending Tracts	
V.S.T.=vestibulo-spinal tract		S.T.T.=spino-thalamic tract	
T.S.T.=tecto-spinal tract		A.S.C.T.=anterior spino-cerebellar tract	(Gowers' tract)
D.P.T.=direct pyramidal tract	cortico-spinal tract	P.S.C.T.=posterior spino-cerebellar tract	(Flechsig's tract)
C.P.T.=crossed pyramidal tract		C.C.=Clark's column	
N.S.T.=rubro-spinal and thalamo-spinal tracts			
S.C.=Schultze's comma			

On the left side of the cord are represented the nerve roots and those bundles of long fibers in the white columns which carry impulses downward from the brain to the spinal cord, and on the right side are represented those bundles of long fibers in the white columns which carry impulses upward from the spinal cord or spinal ganglia to the brain. It hardly needs to be stated that, although in this figure these long bundles of fibers are represented on one side only, they are really situated symmetrically on each side of the cord. The short fibers which connect different levels of the cord together are not represented in the figure.

Lesions involving the pyramidal tract give rise to a spastic paralysis described under 251, 254, 525-6, 796-7, 1212 and 1372-4-7. Lesions involving the anterior horns give rise to atrophic paralysis, the acute forms of which are described under 495, 789, 1148, 1233 and 1304; while the chronic forms are described under 547, 695, 1149 and 1304. Lesions involving the posterior horn give rise to symptoms described under 1302. Lesions of posterior columns give rise to symptoms described under 785, 1302, 1347, 1350-1 and 1396. Lesions of the spino-cerebellar tract give rise to symptoms described under 281 and 654. Lesions of the spino-thalamic tract and of the anterior commissure of the gray matter give rise to symptoms described under 365, 811 and 1356-60. Lesions of the whole of one lateral half of the cord give rise to symptoms described under 442, 509, 840 and 981; while lesions of the whole transverse section of the cord give rise to symptoms described under 485, 513-4, 517-8, 520, 549-50, 791, 795, 825, 828-9, 835 and 980, 1148a, 1309-10, 1395-7.

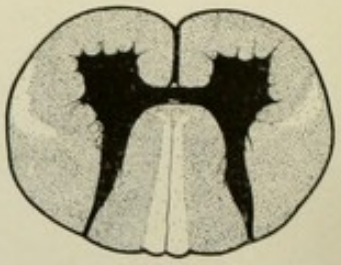
Fig. 27

Schematic representation of the more important diseases of the spinal cord.

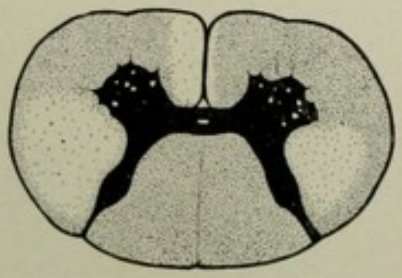


Locomotor Ataxia
(lumbar region)

See 345, 416, 419–20, 433, 661, 756, 784, 827, 891, 894, 911, 979, 987, 1004, 1015, 1172, 1186, 1217 and 1231

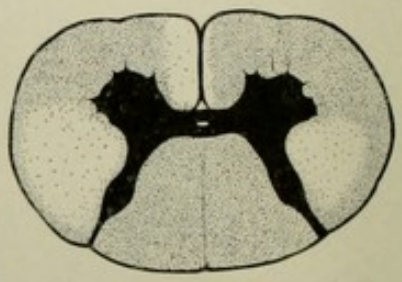


Locomotor Ataxia
(cervical region)

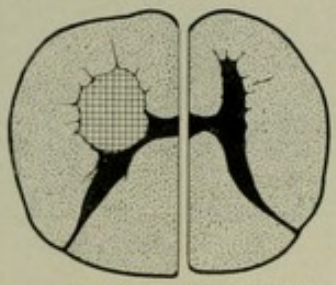


Amyotrophic Lateral Sclerosis

See 547, 695, 797, 1149; and 525, 670 and 797

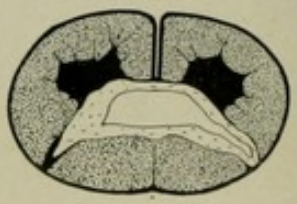


Descending Degeneration of
Pyramidal Tracts

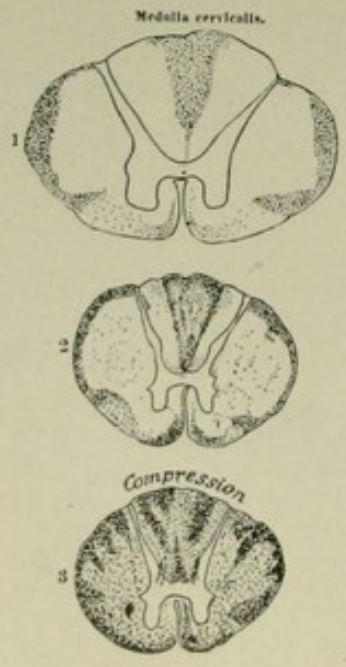


Acute Stage Chronic Stage

Anterior Poliomyelitis
See 416, 419, 495, 789 1148 and 1233



Syringomyelia
See 552, 693, 837-9,1009, 1150a 1170, 1187, 1357 and 1359



Compression Myelitis with the consequent Ascending and Descending Degenerations. See 520, 795.

No. 3 shows the point of the compression with the whole transverse section of the cord the seat of an inflamma

No. 1 shows ascending degeneration of the columns of Goll, of the spino-thalamic tracts, and of the anterior and posterior spino-cerebellar tracts.

No. 2, close to the lesion, shows in addition a slight degeneration of the columns of Burdach.

Nos. 4–6 show degeneration of the crossed and direct pyramidal tracts of the vestibulo-spinal, rubro-spinal, and thalamo-spinal tracts and of Schultze's comma.

The upper series face up and the lower down.

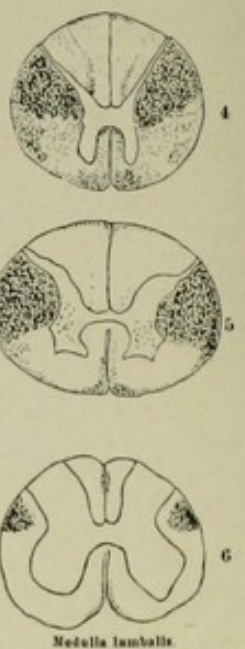


Fig. 28. Diagram to illustrate the mechanism of the bladder reflex

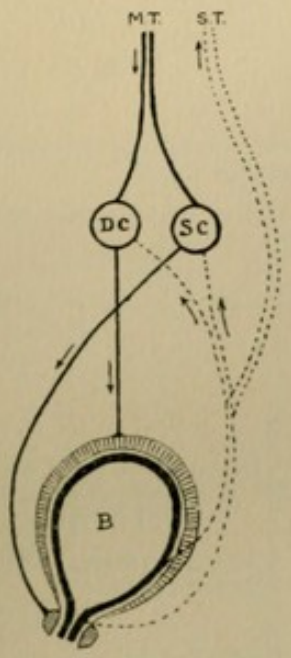


FIG. 28

B represents the bladder. S C represents the reflex centre, with its motor and sensory neurons, for the sphincter of the bladder, which is excited to action by urine in the neck of the bladder or in the prostatic urethra. D C represents the reflex centre, with its motor and sensory neurons, for the detrusor of the bladder, which is excited to action by the distention of the walls of the bladder. These two reflexes are antagonistic and the sensory surface irritated being much larger in the latter (D C), than in the former (S C), reflex, the detrusor reflex will eventually overpower the sphincter reflex under normal conditions. S T represents the sensory tract connecting the bladder with the brain, by means of which the individual is informed as to the degree of fulness of the bladder. M T represents the motor tract connecting the cerebral with the spinal centre by means of which the individual can inhibit the activity of either centre (up to a certain degree) and increase the activity of the antagonistic centre.

FIG. 29 illustrates effects of lesions of cauda equina.

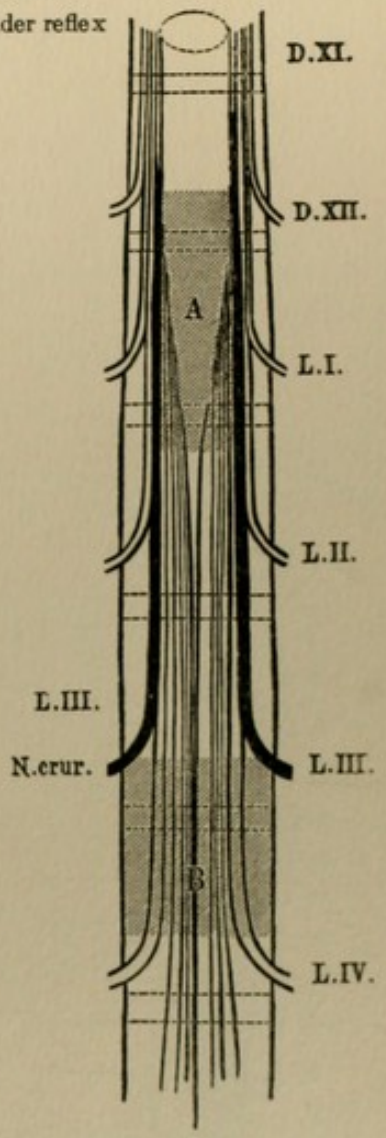
If the lesion is at "A" there is complete motor paralysis of both legs, and complete anesthesia of the whole of both legs and of the perineum, buttocks, scrotum and penis, and all reflexes of the legs are abolished.

If the lesion is at "B" there is complete motor paralysis of both legs, except the flexors of the thigh and the extensors of the leg, and complete anesthesia of the perineum, buttocks, scrotum and penis, and of the posterior surface of the thighs, the posterior and lateral surfaces of the legs, and all of the foot, except a small area on its inner surface. All the reflexes of the legs except the knee-jerks are abolished.

In both cases the muscles atrophy, there is no zone of hyperesthesia above the anesthesia and the bladder and rectum show a motor and sensory paralysis.

If the lesion is limited to the conus medullaris there is a paralysis of the rectum and bladder and an anesthesia of the penis, scrotum, perineum, one inch about anus, and the upper two-thirds of the posterior surface of the thighs. Otherwise there is no paralysis of motion or sensation.

See 487, 721, 1007, 1308.



(After Fr. Schultze-Köster.

FIG. 29

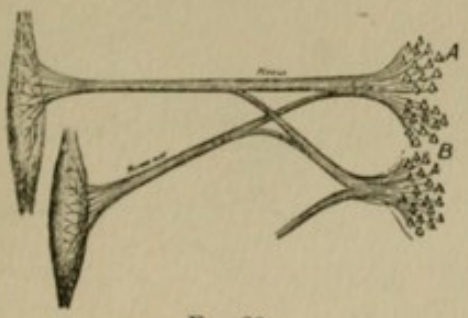


FIG. 30

Showing the innervation of muscles through more than one nerve root, so that the destruction of one nerve root or of one group of nerve cells does not cause a complete and permanent paralysis.

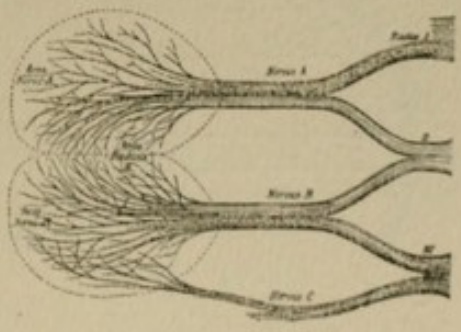


FIG. 31

A diagram showing that a given sensory area of the skin is supplied by filaments from several nerve roots; so that division of one root does not necessarily produce total anesthesia. It also shows the peripheral overlapping; so that the area supplied by one nerve can be almost completely supplied by neighboring nerves.

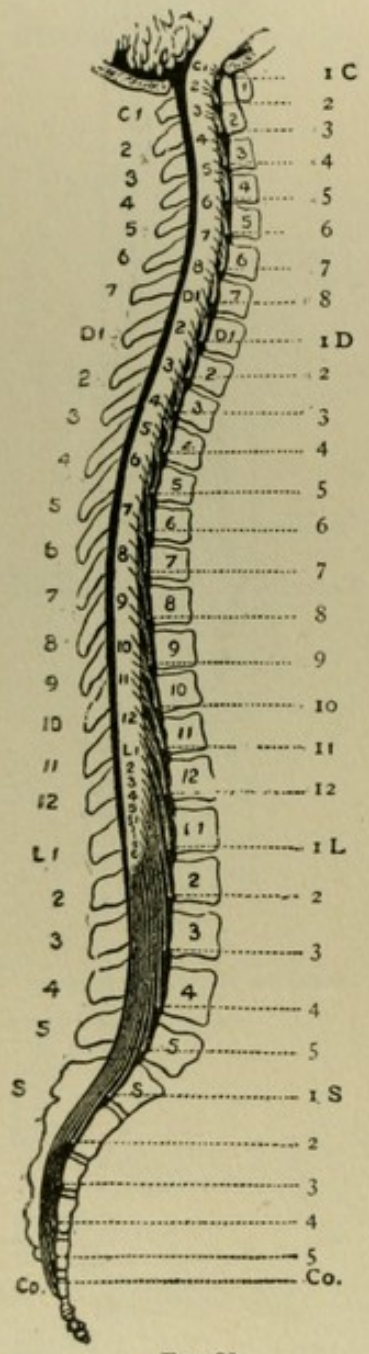
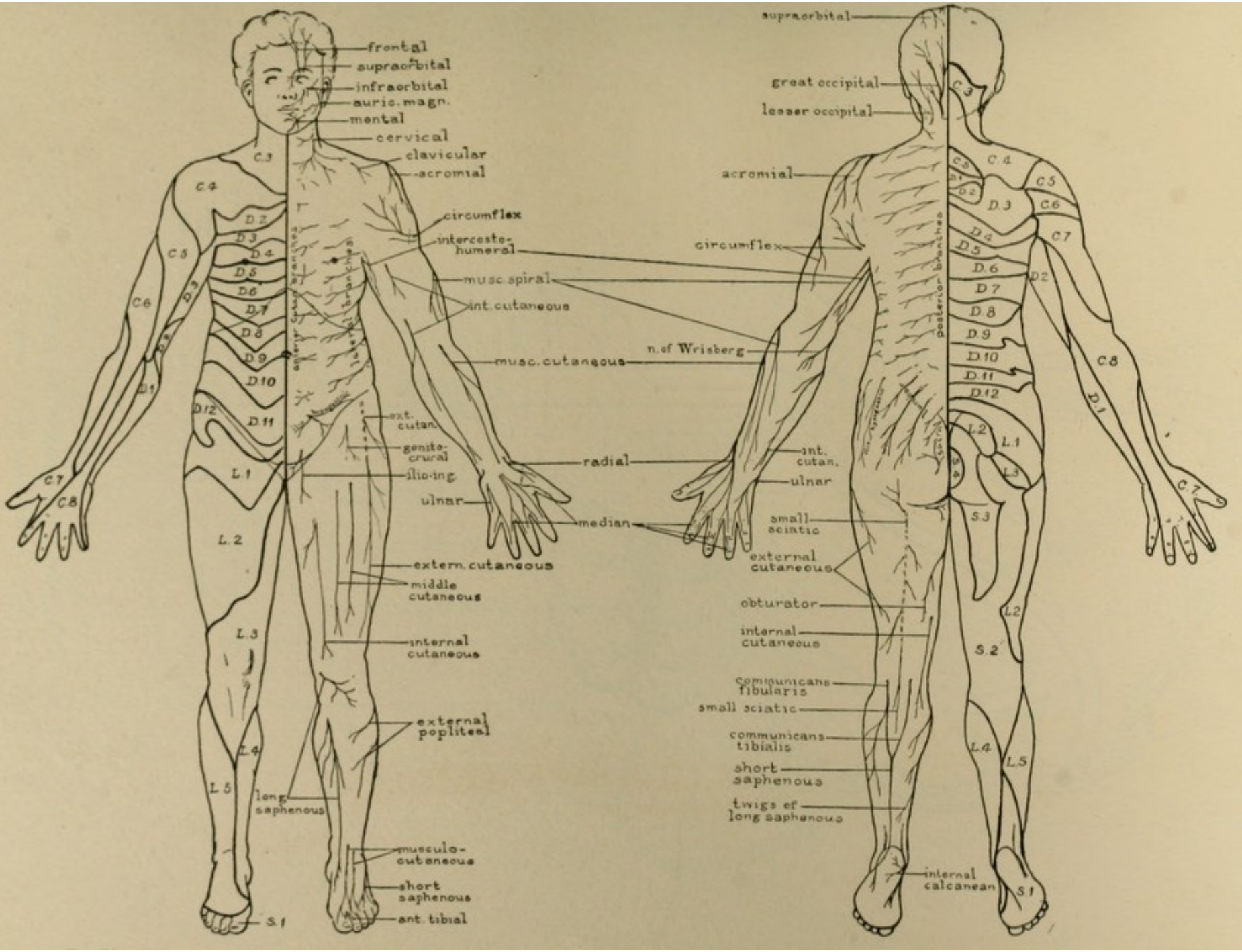


FIG. 32

MOTOR AND REFLEX FUNCTIONS OF THE SPINAL-CORD SEGMENTS (MODIFIED AFTER STARR AND EDINGER)

SEGMENT	MUSCLES	REFLEXES
Cervical	Sternomastoid	
	Trapezius	
2–3	Scaleni	
	Small rotators of head	
	Diaphragm	
	Lev. ang. scap.	Dilatation of pupil by irritating side of neck, 4 cervical to 1 dorsal
	Rhomboids	
4	Spinati	
	Deltoid	
	Supinat. long	Scapular reflexes, 5 C–1 D
	Biceps	Supinat. long., 5 C
5	Supinat. brev.	
	Serrat. mag.	
	Pectoralis (clav.)	Biceps, 5–6 C
	Teres minor	Triceps, 6 C
	Pronators	Posterior wrist, 6–8 C
6	Brachialis ant.	Scapulo-humeral, 7 C
	Triceps	
	Long extensors of wrist and fingers	Anterior wrist, 7–8 C
7	Pectoralis (costal)	
	Latiss. dorsi	Palmar, 7 C–1 D
	Teres maj.	
	Long flexors, wrist and fingers	Epigastric, 4–7 D
8	Extensors of thumb	
Dorsal 1	Intrinsic hand-muscles	Abdominal, 7–11 D
2–12	Dorsal and abdominal muscles	
Lumbar	Abdominal muscles	Cremaster, 1–3 L
1	Iliacus	
	Psoas	Patellar, 2–4 L
2	Sartorius	Bladder, 2–4 L
	Flexors of knee	
	Quad. femoris	
3	Int. rotators of thigh	
	Adductors of thigh	Rectal, 4 L–2 S
4	Abductors of thigh	
	Tibialis ant.	Gluteal, 4–5 L
	Calf-muscles	
5	Ex. rotators of thigh	
	Extensors of toes	Achilles, Ankle-clonus, 1–3 S
Sacral	Peronei	
1–2	Long flex. of toes	Plantar, 1–2 S
	Intrinsic foot-muscles	Anal, Virile, 3–5 S
3–5	Perineal muscles	


frontal
supraorbital
infraorbital
auric. magn.
mental
cervical
clavicular
acromial
circumflex
intercosto-humeral
musc. spiral
int. cutaneous
musc. cutaneous
radial
ulnar
median
ext. cutan.
genito-crural
ilio-ing
extern. cutaneous
middle cutaneous
internal cutaneous
external popliteal
long saphenous
musculo-cutaneous
short saphenous
ant. tibial
supraorbital
great occipital
lesser occipital
acromial
circumflex
n. of Wrisberg
int. cutan.
ulnar
small sciatic
external cutaneous
obturator
internal cutaneous
communicans fibularis
small sciatic
communicans tibialis
short saphenous
twigs of long saphenous
internal calcanean

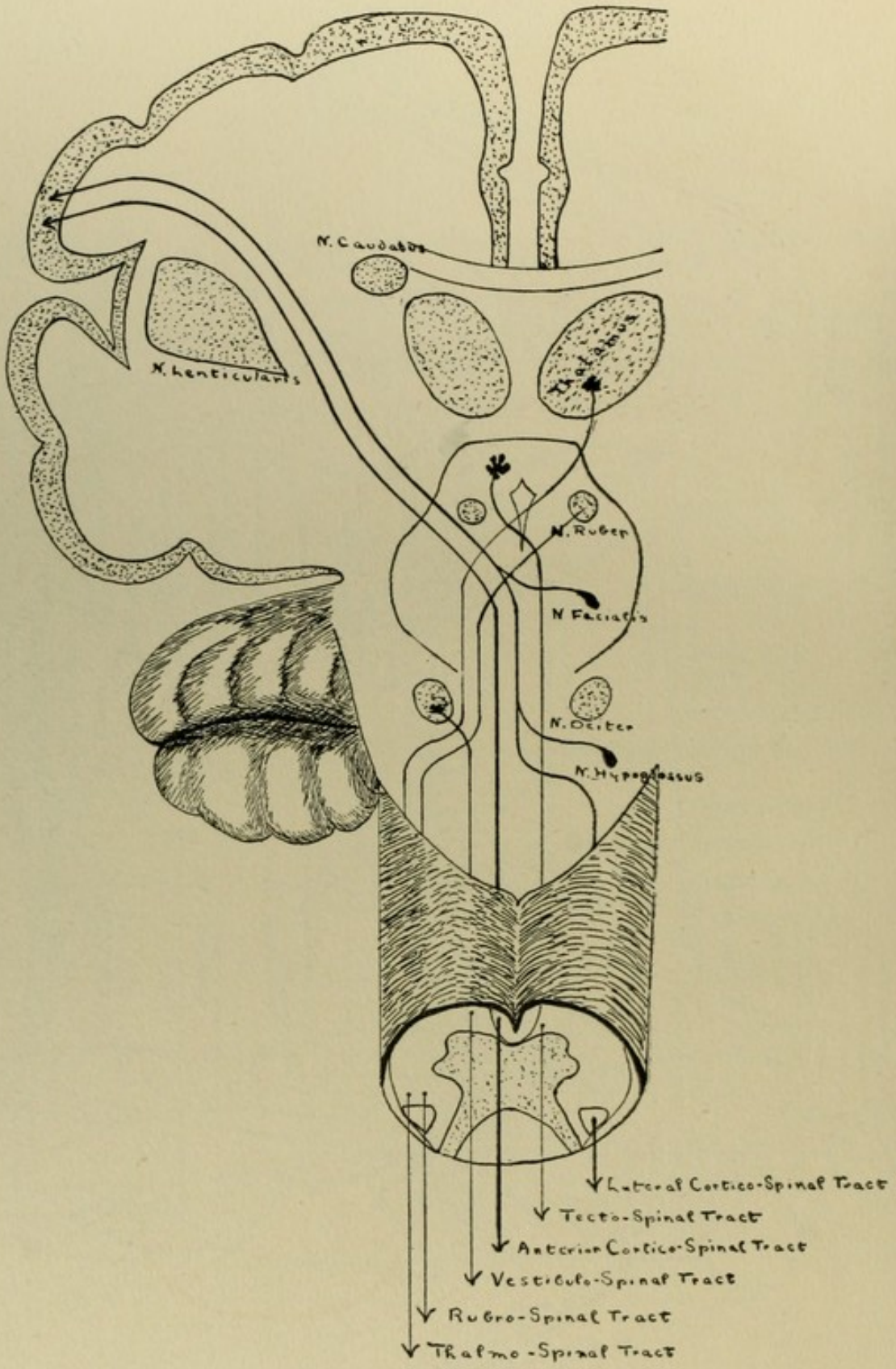


Fig. 34

LONG MOTOR PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26.

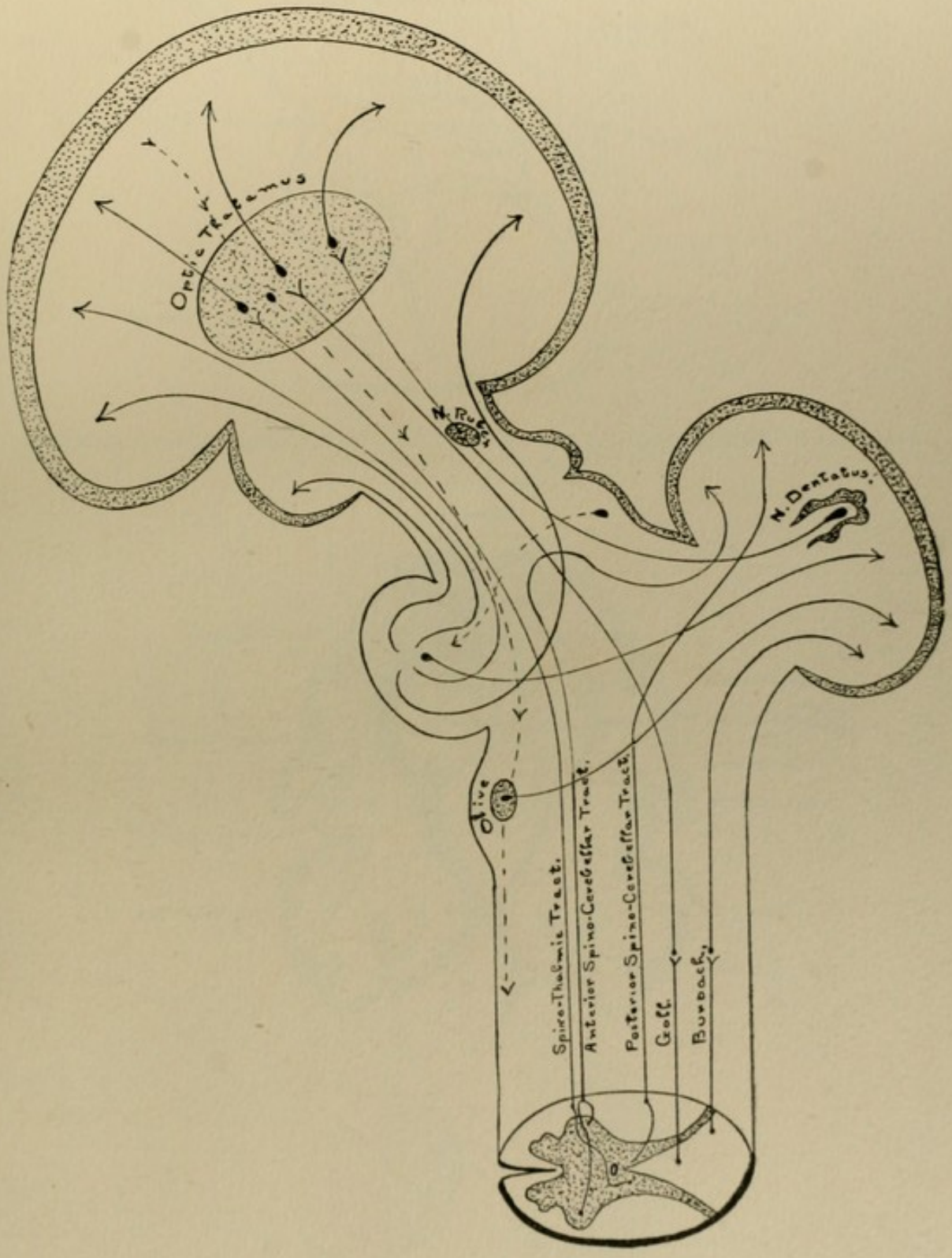


Fig. 35

LONG SENSORY PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26.

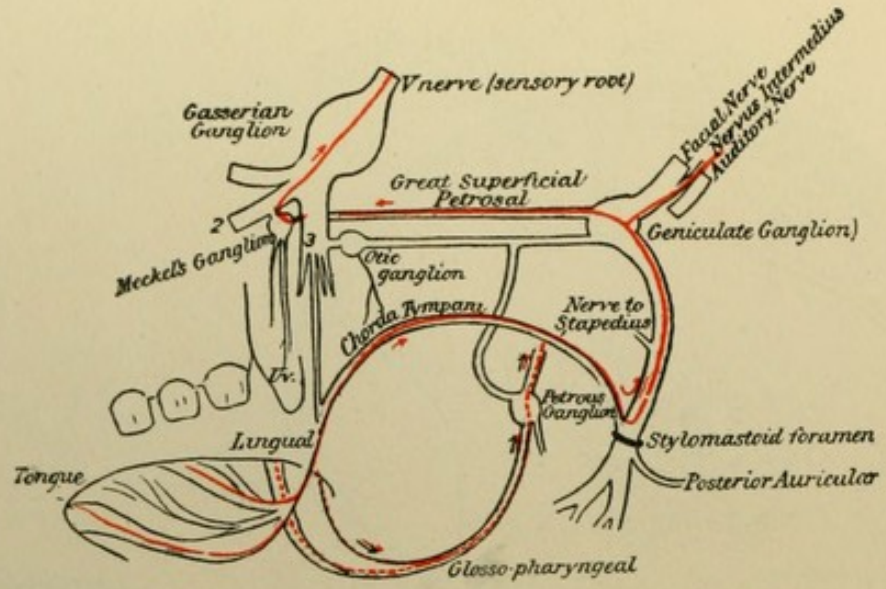


Fig. 36

DIAGRAM OF TRIGEMINAL, FACIAL AND GLOSSO-PHARYNGEAL NERVES, SHOWING COURSE OF TASTE FIBRES.

(After Purves Stewart)

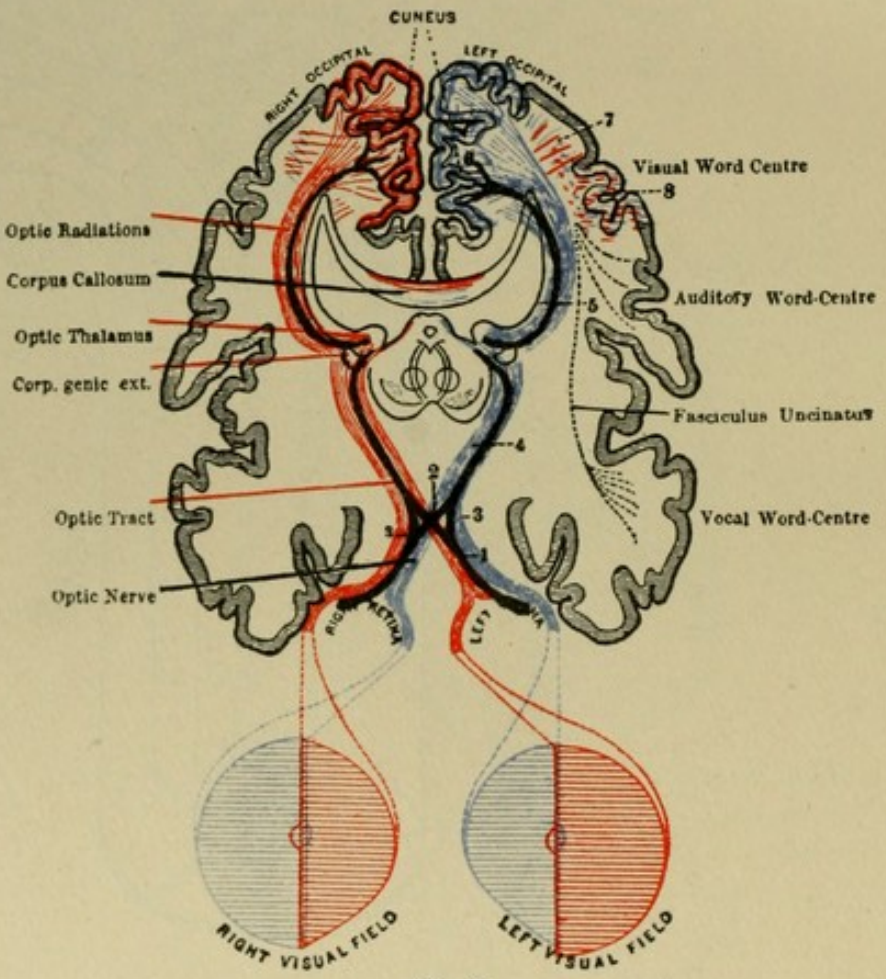


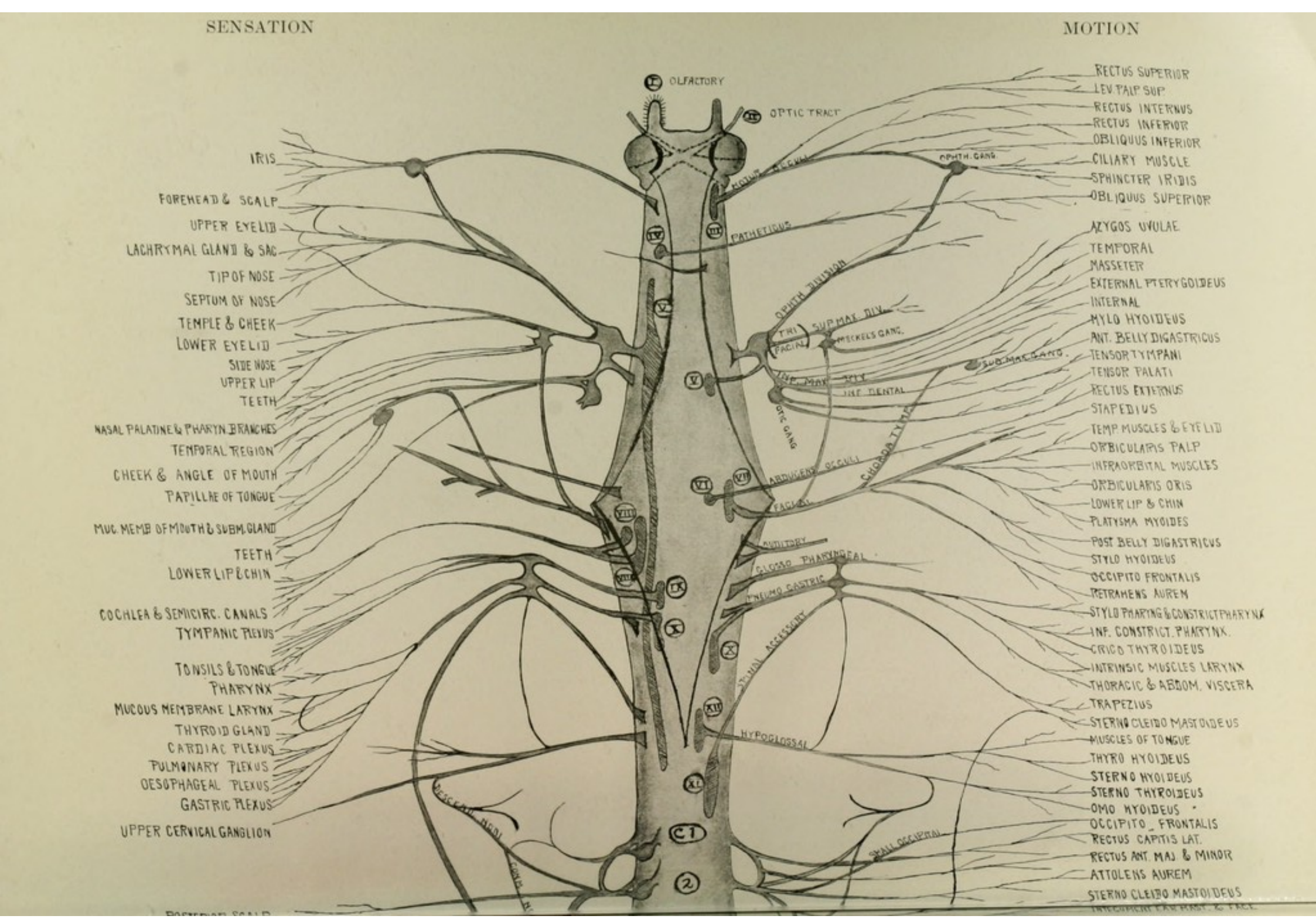
Fig. 37

DIAGRAM ILLUSTRATING HEMIANOPIA

(Modified from Vialet)

Lesion at 1 produces blindness of one eye.
Lesion at 2 produces bi-temporal hemianopia
Lesion at 3 produces bi-nasal hemianopia
Lesion at 4 produces R. hemianopia with hemiopic pupil reaction.
Lesion at 5 produces R. hemianopia with normal pupil reaction.
Lesion at 6 produces R. hemianopia with normal pupil reaction.
Lesion at 7 produces psychic blindness.
Lesion at 8 produces Alexia.

The heavy black lines represent the fibers from the macula lutea in each retina, the point of central, or clearest vision.


SENSATION
MOTION
I OLFACTORY
II OPTIC TRACT
III MOTOR OCULI
IV PATHETICUS
V OPHTH. DIVISION
TRI FACIAL
SUP MAX DIV
MECKELS GANG.
INF MAX DIV
INF DENTAL
OPHTH. GANG.
SUB MAX GANG.
OTIC GANG
CHORDA TYMPANI
VI ABDUCENS OCULI
VII FACIAL
VIII AUDITORY
GLOSSO PHARYNGEAL
PNEUMO GASTRIC
SPINAL ACCESSORY
IX
X
XI
XII HYPOGLOSSAL
C1
2
IRIS
FOREHEAD & SCALP
UPPER EYELID
LACHRYMAL GLAND & SAC
TIP OF NOSE
SEPTUM OF NOSE
TEMPLE & CHEEK
LOWER EYELID
SIDE NOSE
UPPER LIP
TEETH
NASAL PALATINE & PHARYN. BRANCHES
TEMPORAL REGION
CHEEK & ANGLE OF MOUTH
PAPILLAE OF TONGUE
MUC. MEMB OF MOUTH & SUBM. GLAND
TEETH
LOWER LIP & CHIN
COCHLEA & SEMICIRC. CANALS
TYMPANIC PLEXUS
TONSILS & TONGUE
PHARYNX
MUCOUS MEMBRANE LARYNX
THYROID GLAND
CARDIAC PLEXUS
PULMONARY PLEXUS
OESOPHAGEAL PLEXUS
GASTRIC PLEXUS
UPPER CERVICAL GANGLION
RECTUS SUPERIOR
LEV. PALP. SUP.
RECTUS INTERNUS
RECTUS INFERIOR
OBLIQUUS INFERIOR
CILIARY MUSCLE
SPHINCTER IRIDIS
OBLIQUUS SUPERIOR
AZYGOS UVULAE
TEMPORAL
MASSETER
EXTERNAL PTERYGOIDEUS
INTERNAL
MYLO HYOIDEUS
ANT. BELLY DIGASTRICUS
TENSOR TYMPANI
TENSOR PALATI
RECTUS EXTERNUS
STAPEDIUS
TEMP. MUSCLES & EYELID
ORBICULARIS PALP
INFRAORBITAL MUSCLES
ORBICULARIS ORIS
LOWER LIP & CHIN
PLATYSMA MYOIDES
POST BELLY DIGASTRICUS
STYLO HYOIDEUS
OCCIPITO FRONTALIS
RETRAHENS AUREM
STYLO PHARYNG & CONSTRICT PHARYNX
INF. CONSTRICT. PHARYNX.
CRICO THYROIDEUS
INTRINSIC MUSCLES LARYNX
THORACIC & ABDOM. VISCERA
TRAPEZIUS
STERNO CLEIDO MASTOIDEUS
MUSCLES OF TONGUE
THYRO HYOIDEUS
STERNO HYOIDEUS
STERNO THYROIDEUS
OMO HYOIDEUS
OCCIPITO_FRONTALIS
RECTUS CAPITIS LAT.
RECTUS ANT. MAJ & MINOR
ATTOLENS AURUM
STERNO CLEIDO MASTOIDEUS

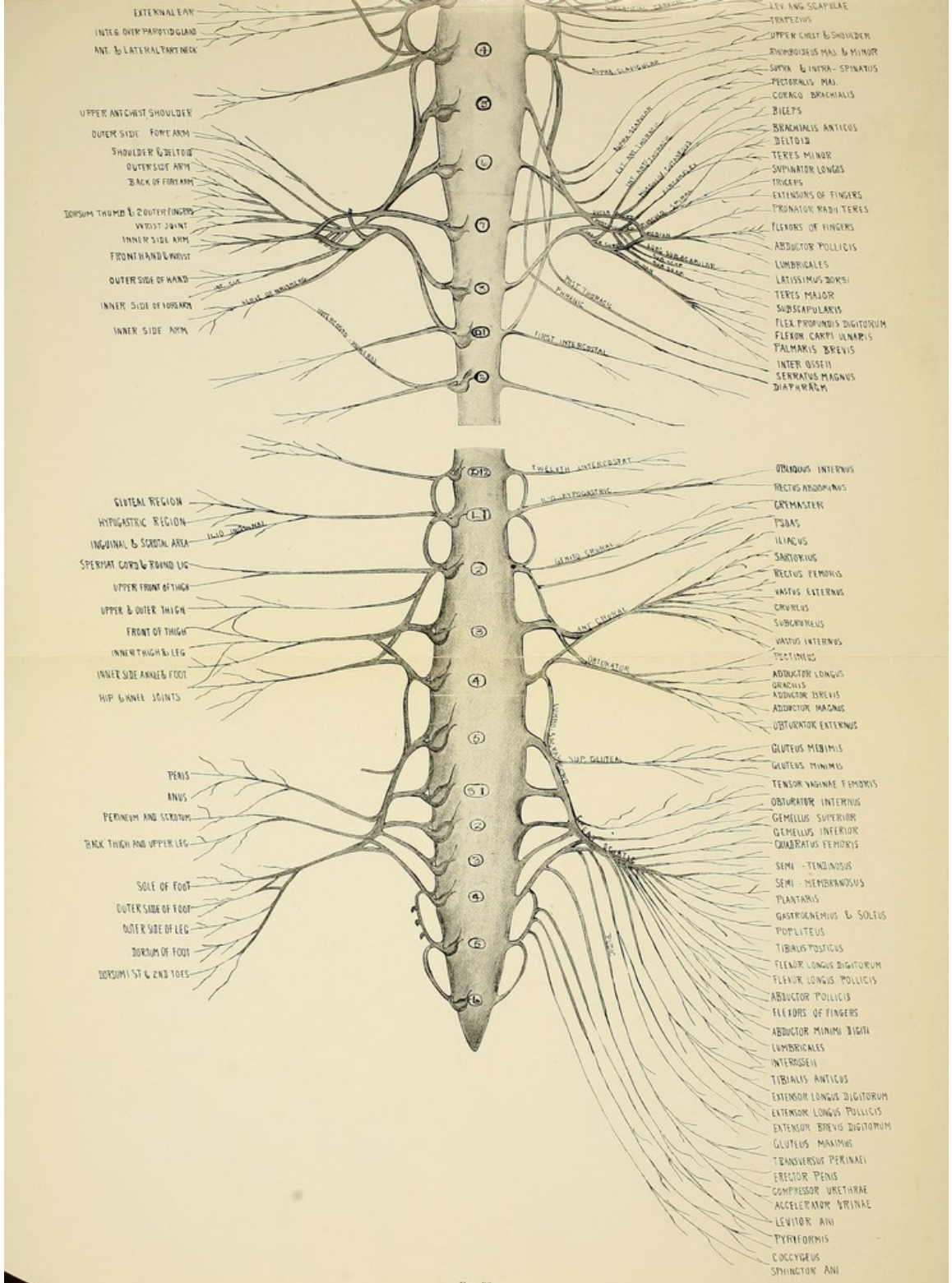

EXTERNAL EAR
INTEG. OVER PARTOTID GLAND
ANT. & LATERAL PART NECK
UPPER ANT CHEST, SHOULDER
OUTER SIDE FORE ARM
SHOULDER & DELTOID
OUTER SIDE ARM
BACK OF FORE ARM
DORSUM THUMB & 2 OUTER FINGERS
WRIST JOINT
INNER SIDE ARM
FRONT HAND & WRIST
OUTER SIDE OF HAND
INNER SIDE OF FOREARM
INNER SIDE ARM
GLUTEAL REGION
HYPOGASTRIC REGION
INGUINAL & SCROTAL AREA
SPERMAT CORD & ROUND LIG
UPPER FRONT OF THIGH
UPPER & OUTER THIGH
FRONT OF THIGH
INNER THIGH & LEG
INNER SIDE ANKLE & FOOT
HIP & KNEE JOINTS
PENIS
ANUS
PERINEUM AND SCROTUM
BACK THIGH AND UPPER LEG
SOLE OF FOOT
OUTER SIDE OF FOOT
OUTER SIDE OF LEG
DORSUM OF FOOT
DORSUM 1ST & 2ND TOES
LEV. ANG. SCAPULAE
TRAPEZIUS
UPPER CHEST & SHOULDER
RHOMBOIDEUS MAJ. & MINOR
SUPRA & INFRA-SPINATUS
PECTORALIS MAJ.
CORACO BRACHIALIS
BICEPS
BRACHIALIS ANTICUS
DELTOID
TERES MINOR
SUPINATOR LONGUS
TRICEPS
EXTENSORS OF FINGERS
PRONATOR RADII TERES
FLEXORS OF FINGERS
ABDUCTOR POLLICIS
LUMBRICALES
LATISSIMUS DORSI
TERES MAJOR
SUBSCAPULARIS
FLEX. PROFUNDIS DIGITORUM
FLEXOR CARPI ULNARIS
PALMARIS BREVIS
INTER OSSEII
SERRATUS MAGNUS
DIAPHRAGM
OBLIQUUS INTERNUS
RECTUS ABDOMINUS
CREMASTER
PSOAS
ILIACUS
SARTORIUS
RECTUS FEMORIS
VASTUS EXTERNUS
CRURUS
SUBCRUREUS
VASTUS INTERNUS
PECTINEUS
ADDUCTOR LONGUS
GRACILIS
ADDUCTOR BREVIS
ADDUCTOR MAGNUS
OBTURATOR EXTERNUS
GLUTEUS MEDIMUS
GLUTEUS MINIMUS
TENSOR VAGINAE FEMORIS
OBTURATOR INTERNUS
GEMELLUS SUPERIOR
GEMELLUS INFERIOR
QUADRATUS FEMORIS
SEMI - TENDINOSUS
SEMI - MEMBRANOSUS
PLANTARIS
GASTROCNEMIUS & SOLEUS
POPLITEUS
TIBIALIS POSTICUS
FLEXOR LONGUS DIGITORUM
FLEXOR LONGUS POLLICIS
ABDUCTOR POLLICIS
FLEXORS OF FINGERS
ABDUCTOR MINIMI DIGITI
LUMBRICALES
INTEROSSEII
TIBIALIS ANTICUS
EXTENSOR LONGUS DIGITORUM
EXTENSOR LONGUS POLLICIS
EXTENSOR BREVIS DIGITORUM
GLUTEUS MAXIMUS
TRANSVERSUS PERINAEI
ERECTOR PENIS
COMPRESSOR URETHRAE
ACCELERATOR URINAE
LEVITOR ANI
PYRIFORMIS
COCCYGEUS
SPHINCTOR ANI

FIG. 38

INDEX

INDEX

Roman numerals indicate charts. Arabic numerals indicate margin numbers.
Arabic numerals preceded by "p" indicate pages.

Hun.

Atlas of differential diagnosis of the diseases of the nervous system.

~~Cole~~