THE DIAGNOSIS
OF
NERVOUS AND MENTAL DISEASES

PERSHING

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BY

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PREFACE.

The object of this book is to facilitate the recognition of nervous and mental diseases by physicians who are not specialists in neurology. It makes no attempt to add to the facts of medicine but aims simply to set forth a practical method of diagnosis in as convenient and compact a form as possible.

Neurological diagnosis presents peculiar difficulties for those who have not been specially trained in it. A thorough examination must be followed by a complicated train of reasoning in which there are many possibilities of error. To avoid the difficulties by leaving them all to the neurologist is impracticable, for cases of nervous disease, often simulating other maladies, are sure to come to every one who practices medicine, even to the specialist who has least to do with neurology.

In trying to teach an available method of diagnosis, medical writers may well adopt a plan extensively used in other sciences. The student of botany does not have to carry in mind the characters of all the species he may possibly meet in order to find the name and classification of any specimen in which he may be interested. He is provided with a table, or "key," in which there is a condensed description of all the classes, orders, genera and species, so arranged that, having made a proper examination of the specimen, he can rapidly find the divisions and subdivisions to which it belongs. The same method is used with great advantage in zoölogy, chemistry and mineralogy. Of all the branches of medicine, neurology is the one to which it is most applicable.

Some years ago, for the use of my classes, I began the construction of a series of diagnostic tables, under such general heads as Hemiplegia, Paralysis of Ocular Muscles, Optic Neuritis, Headache, etc., adopting the form of a botanical key. These tables have grown until they pretty fairly cover the field of neurological diagnosis and they are offered to the profession in the hope that they will be practically useful as an outline map of the field. They aim to show, explicitly but briefly, what symptoms are the most important in a given case of nervous disease, and how an analysis of the symptoms should lead to the recognition of the disease; in other words they attempt to show on paper what a neurologist would have in mind in confirming his diagnosis, step by step. As an intelligent use of the tables presupposes a knowledge of the essential features of the case under consideration and the ability to recognize certain general conditions, they are preceded by a description of the methods of examination and a brief discussion of the signs of organic disease, hysteria and neurasthenia.

It is of course understood that such an outline of diagnosis should be used as an adjunct to some complete treatise on nervous diseases and one of the hopes entertained by the writer is that it will make the fuller knowledge of the best text-books more readily accessible to the busy physician.

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EXAMINATION OF THE PATIENT AND THE GENERAL SIGNIFICANCE OF SYMPTOMS.

In order to be thorough the neurological examination must follow an orderly routine. In any case involving difficulties of diagnosis or treatment it is true economy of time and labor to get all the available data and accurately record them at the beginning, making such additional notes from time to time as new facts may require. For this purpose loose sheets of paper with printed headings, properly arranged, are most convenient, although an ordinary record book may be used.

FAMILY HISTORY.

After getting a general idea of the patient's complaint and making the usual notes of the name, age, race, domestic condition and occupation, inquiry is to be made as to the family history. The state of health, if living, or the age at death and cause of death, should be noted of each of the parents, grandparents, brothers and sisters. Care should be taken not to overlook the existence, in any of the near relatives, of nervous or mental disease, tuber-

culosis, rheumatism or gout, and a general denial on the part of the patient should be supplemented by specific inquiries, extending often to the history of the uncles and aunts.

PERSONAL HISTORY.

The salient points in the personal history prior to the present illness are first to be noted in chronological order, after which the history of the present illness is to be obtained with special reference to the first symptoms and their mode of onset, whether sudden, rapid or slow, the order in which the subsequent symptoms have appeared and the patient's complaints at the time of examination. Skilful questioning will be necessary to bring out the full history and it should be definite in regard to the following subjects:

1. Injury at birth.
2. Damage to the nervous system by previous diseases, such as scarlatina, influenza, meningitis, etc., or by injury.
3. Convulsions or nervous spells of any kind.
4. Syphilitic infection.
5. Other diseases of the sexual organs, real or imaginary, and sexual excesses.
6. Habits as to alcohol, tobacco and other possible poisons.
7. The uric acid diathesis.
8. Exhausting influences and signs of exhaustion.
9. Abnormal suggestibility, morbid fears, hypochondriacal worry and other signs of mental instability.

Possible injury at birth is of especial importance when paralysis, spasm or arrest of development dates from infancy.

If convulsions or nervous spells of any kind have occurred care is to be taken to ascertain when they began, how often they have recurred and under what circum-

stances, as well as to get an accurate description of them. It must be remembered that some symptoms, such as attacks of *petit mal*, are not likely to be mentioned unless the examiner asks specifically about them and, on the other hand, that leading questions will often elicit totally incorrect answers.

Syphilis is so common a cause of headache, neuralgia, and many grave organic affections and so much depends on early specific treatment that it is difficult to exaggerate the importance of its prompt recognition. There need be no hesitation about asking any male patient whether venereal infection has occurred and cross-examining him if the answer is negative. When the direct question cannot be asked, as in the case of most women, or when the answers are inconclusive, the examiner must be alive to the significance of the symptoms of syphilis, such as the eruptions, mucous patches, nasal ulcers, alopecia, keratitis, iritis, choroiditis, nodes on bones or cartilages, enlarged glands, nocturnal headache, etc. When hereditary syphilis is suspected inquiry is to be made as to snuffles, emaciation, eruption and parchment-like character of the palms and soles in early infancy.

The history as to sexual disorders other than syphilis is important because they occasionally cause reflex pains and very often are a source of exhaustion or worry. Worrying about an imaginary disease may be far more harmful than the physical effects of the real disease, so patients should be induced to express themselves freely. Many a man carries a mental burden for months or even years because he has an occasional seminal emission or has discovered a little mucus at the meatus, which he fancies to be an exhausting seminal discharge, or because he has discovered the normal epididymis or the veins of the spermatic cord or that the left testicle hangs lower than the

right. That a nervous woman's mind is often fixed upon her pelvic organs to her great detriment is well known to every physician. A morbid idea of this kind often constitutes a mental traumatism which is the most important fact in the case, and the removal of the idea may be the most difficult as well as the most essential part of the treatment.

A true estimate of the effects of sexual excesses and of masturbation requires careful discrimination. In many cases these vices are an important physical cause of exhaustion, but in many other cases the fear and remorse occasioned by past errors are of serious import while the physical effects are insignificant.

The intemperate use of alcohol is generally freely confessed by men but often denied by women, who are more apt to drink secretly if at all. When excessive drinking is not confessed, alcoholism may be revealed by a history of gastric catarrh with morning vomiting, delirium tremens or multiple neuritis with the mental state characteristic of the alcoholic form.

The possibility of poisoning by lead, arsenic, mercury, carbon disulphid or other toxic agents may have to be considered, especially in connection with the patient's occupation.

The uric acid diathesis is a very important factor in the etiology of headache, neuralgia, sciatica, neurasthenia and mental depression, as well as of ordinary gout and rheumatism. No uncertainty as to its essential cause should prevent its full recognition in diagnosis and treatment. A history of muscular or articular pains without organic cause, or of repeated attacks of tonsillitis, pharyngitis or eczema, or of so-called bilious attacks, or even a history of headache or despondency habitually worse in the early morning, ought always to suggest the possibility of this condition

being the cause. If further inquiry shows that the urine is often dark and sometimes deposits urates the possibility becomes a probability.

The causes of nervous exhaustion, such as the infectious diseases, repeated pregnancies, lactation, overwork, anxiety and grief, are generally clearly indicated in the patient's account. The signs of exhaustion, such as early fatigue, irritability, lack of zest for work and a loss of the former ambitions and interests, are also likely to receive prominent mention. More often overlooked is the fact that moral delinquencies, such as neglect of plain duties, cowardice, addiction to stimulants and narcotics and offenses growing out of irrational antipathies or unrestrained impulses, may have their origin in exhaustion, especially in patients of neurotic inheritance.

In functional nervous disorders it is of the greatest importance for diagnosis and treatment that the facts in the personal history which reveal the patient's mental peculiarities should be fully noted. If the patient tells of most extraordinary effects of medicines, of queer sensations felt on the approach of a thunder storm or while riding in an electric car, or of severe pains or profound emotional disturbance caused by some trivial occurrence, the physician, instead of controverting the statements at this time, should be an interested and sympathetic listener, and so lead the conversation as to get a full understanding of the degree of emotional instability and susceptibility to suggestion. For the same reason, if the patient is afraid to go into the street alone, or is absurdly convinced that his heart is seriously diseased, he is not to be laughed at, but encouraged to tell the whole of his troubles.

Failure to elicit this part of the history often causes failure in the management of the case when better methods would insure success. Even if the diagnosis be correct, a

plan of treatment which is perfect on the physical side may be rendered useless by a mistaken idea on the part of the patient that he cannot stand some of the remedies or that his first temporary backset, after a period of improvement, shows the futility of continuing on the same plan. On the other hand, the mental peculiarities which are a source of danger may be made the means of securing confidence, obedience and unfaltering constancy by a physician who understands them. The psychologic management of neurotic patients is a difficult art which can hardly be taught on paper, but it is so important a means of success that every young practitioner should make a conscious effort to acquire it. The three questions to be kept in mind when taking a history of such a patient are: "What light do the mental characteristics of the patient throw upon the diagnosis of his case?" "What is there in his mental constitution which may endanger the success of treatment?" and "How can his peculiarities be made to contribute to a successful result?"

It is hardly necessary to remind even the beginner in neurology that he should not express an opinion based on the history alone, but should always proceed to a thorough physical examination.

THE PRESENT CONDITION.

The objective examination should be begun by observing the general appearance as to vigor, color, state of nutrition and expression of the face. Any abnormality in the size or conformation of the head or any of its parts will be observed at the same time. The actual bodily weight should be noted for comparison with its amounts in the past and in the future. At least a cursory examination should be made in any case into the condition of the heart, lungs, abdominal organs and urine, and elaborate investigations

may be necessary in special cases. The urine, in particular, must often be examined with the greatest care, for uremia causes a great variety of nervous symptoms and it is by no means excluded by the failure to find albumen. In all doubtful cases the chemical tests should be supplemented by microscopic examination and a consideration of the total quantity and specific gravity of the urine passed in 24 hours.

MOTOR DISORDERS.

The motor disorders to be looked for are paralysis, ataxia, spasm and tremor. As a general test of motility let the patient walk forward and backward, turn and stand on either foot, first with eyes open and then with eyes closed. Next let him grasp the examiner's hands as strongly as possible and afterward hold out both hands with the fingers spread apart, then, with eyes closed, touch the tip of the nose with each forefinger and bring the tips of the forefingers together before the face, above the head and behind the back. Then have him draw up the corners of the mouth as though to show the upper teeth, close the eyes tightly, wrinkle the forehead, so as to open the eyes as widely as possible, and protrude the tongue. If all these actions are readily performed without any abnormality being observed motor disorders of the limbs, face and tongue are excluded and the examiner may proceed to test the reflexes. But if any abnormality is revealed a complete investigation will be necessary.

An abnormal gait may conform to any one of a number of widely differing types which can best be understood by comparison with a normal gait. In normal walking, while the weight of the body rests upon one leg, the other is swung forward like a pendulum hanging from the hip. The swinging foot is kept clear of the ground partly because the hip is slightly raised by the motion which throws

the weight of the body upon the fixed leg, but mainly because the moving limb is shortened by flexion of the knee and dorsal flexion of the foot. All pathological changes of gait are caused by something which prevents the proper support of the body by the fixed leg or the proper forward swing of the moving one.

In the gait characteristic of simple weakness, as in extreme exhaustion or after typhoid fever or in old age, the steps are short and slow. The knees feel as though about to give way and may visibly bend under the weight of the body. In extreme cases the patient grasps surrounding objects for support.

The gait of ordinary hemiplegia owes its peculiarity to the fact that on the paralyzed side flexion of the knee and dorsal flexion of the foot are especially weak so that, as the paralysis is spastic, the limb is held in rather rigid extension. When it should swing forward the heel is raised with difficulty and it is still more difficult to sufficiently raise the toe. It is as though the limb were too long and the foot would remain planted on the ground were it not for two extra motions, by which the patient, as far as possible, obviates the difficulty. He leans excessively toward the sound side, thus elevating the hip on the paralyzed side, and then gives the paralyzed limb an outward swing so as to bring it around in advance of the sound one. The motion resembles the swing of a scythe, the foot pointing outward and representing the blade. There is generally little difficulty in supporting the body while the sound limb is brought forward, as the extensors of the paralyzed limb are relatively strong. Recognition of this form of paralysis is made especially easy by the posture of the paralyzed arm which, in a typical case, is held close to the body and flexed at the elbow, wrist and fingers. Weakness of the lower part of the face may also be apparent.

The gait characteristic of hysterical paralysis of one leg is very different from that of organic hemiplegia, just described, and resembles that of children who imitate lameness in their play. The paralyzed limb is dragged after the sound one, not swung around it nor placed in advance. The foot is often held at right angles to the leg so firmly as to show that there is no weakness of its dorsal flexors.

In the gait of spastic paraplegia both lower limbs are in nearly the same condition as the paralyzed one in organic hemiplegia and the difficulty is to get one foot in advance of the other. To accomplish this the patient leans forward and to the side opposite the limb about to be moved which is then stiffly swung forward as one piece, the foot, especially the toes, dragging on the floor. Spasm of the adductors may cause the thighs to rub together and even to cross. The accompanying exaggeration of the tendon reflexes may be so great as to cause ankle clonus to be elicited as the foot is planted and the weight of the body thrown upon it.

The steppage gait, characteristic of multiple neuritis, is caused by a flabby paralysis of the dorsal flexors of the foot on both sides. When the heel is raised the foot hangs down of its own weight. In order that the toe may clear the ground the heel must be raised by an exaggerated flexion of the hip and knee, much higher than it normally is and then the dangling foot is flung forward, the toe coming down first. The action is somewhat suggestive of that of a high-stepping horse. Poliomyelitis often causes a similar defect but it is usually unequal on the two sides.

Weakness of one leg, especially if there is no rigidity, causes a limp, because the weight of the body is not trusted on the weak leg longer than necessary, and the patient hastens to plant the sound foot and bring the weight on it, thus exaggerating the foot-fall on the sound side. The

same kind of limping is caused by any condition of one leg which makes it painful for it to bear the weight of the body.

If an abnormal gait or a defective movement in the upper part of the body indicates any form of paralysis, each movement should be tested separately.

The power to move a joint may be tested by three methods: 1. The patient is told to move the joint vigorously in each of its possible ways, flexion, extension, adduction, abduction, etc. If motion is not free or vigorous in any direction, care should be taken to make sure that the limitation is not due to mechanical conditions, such as disease of the joint or shortening of the opposing muscles. 2. The patient is told to make the same motions as strongly as possible while the examiner resists them and thus estimates their force. 3. The patient is told to hold the joint in each position while the examiner tries to force it into the opposite position. In many cases the third of these methods gives the most reliable results. These tests may be varied and extended by having the patient stoop and rise on one leg, stand on tiptoe, mount a chair, pick up an object from the floor, arise from a recumbent position, form a ring with the thumb and forefinger, etc.

Paralysis of one side of the face is indicated by comparative smoothness of that side in repose and by less movement than occurs on the sound side in whistling, showing the upper teeth, closing the eyes and wrinkling the forehead. When the weakness of the lips is slight it may best be detected by putting the tip of a finger between them on either side and, while the patient tries to compress it as firmly as possible, pushing upward and downward so as to estimate their muscular power. Slight weakness of one *orbicularis palpebrarum* may be detected by trying to open the lids with the fingers while the patient tries to keep them closed

and also by noting the patient's inability to close the eye on the weakened side without at the same time closing the other. Bilateral paralysis of the face is not so easily detected but when considerable in degree it is revealed by lack of expression and defect of movement on both sides.

Unilateral paralysis of the tongue is shown by deviation to the paralyzed side when it is protruded, bilateral paralysis by inability to protrude it and difficulty in pushing solid food from the mouth into the pharynx in the first part of deglutition.

Paralysis of the palate causes a muffled, nasal sound of the voice, inability properly to utter the explosive consonants (b, p, d, t, g, k), or to puff as in blowing out a candle, and regurgitation of liquid food through the nose. The absence, on one or both sides, of the elevation of the soft palate which normally occurs on saying "Ah," can be seen by direct inspection.

Paralysis of the pharynx causes difficulty in pushing food into the esophagus after it has passed the fauces.

Paralysis of the laryngeal muscles causes alterations of voice and sometimes difficulty of breathing and is recognized with certainty by the observation, with the laryngoscope, of defective movement of one or both vocal cords in respiration and phonation. For details the reader is referred to a text-book of laryngology. Unilateral paralysis of any of the laryngeal muscles is always caused by organic disease.

Thus far the directions for examination have presupposed the coöperation of the patient, but it is often of the greatest importance, especially after apoplectic attacks and injuries of the head, to recognize paralysis while the patient is unconscious. In such cases the paralysis is almost always in the form of a partial or complete hemiplegia which will be revealed by a careful comparison of the two sides of the

body. The eyes and head are often turned away from the paralyzed side and if the head is forcibly turned to this side it soon goes back to its former position. This is called conjugate deviation of the head and eyes. The corner of the mouth is drawn down, the naso-labial fold is partly or wholly obliterated and the cheek may flap more in expiration on the paralyzed side.

On inspecting the limbs it may be noticed that those on one side are moved while the opposite ones are quiet. Moreover, the superficial reflexes and even the tendon reflexes on the paralyzed side are likely to be lost or diminished in this early stage, although later the tendon reflexes will be increased. On raising the arms and letting go of them at the same time the paralyzed one is seen to drop more quickly and limply than the other, and the same test may be applied to the legs.

Ataxia, or incoördination, may exist with or without paralysis, and sometimes causes a striking abnormality of gait when no paralysis can be detected. There are two distinct forms of ataxic gait, that due to disease of the sensory tracts of the spinal cord, as in tabes dorsalis, and that due to disease of the cerebellum or of structures adjacent to it.

In the ataxic gait of spinal disease the incoördination is due to the fact that interference with the sensory impulses from the moving limbs prevents the patient from being aware at each instant of their posture and the force of their muscular contractions and so from having a correct idea of the motion which should follow. The result is that he sways more than is normal, walks with the feet far apart, steps too high, brings the foot down too hard, the heels striking first, and keeps the eyes fixed on the ground ahead of him, so as to make vision compensate as far as possible for the defect in posture sense. Closing the eyes

greatly increases the difficulty in walking and a degree of ataxia too slight to be detected when the eyes are open may be quite apparent when they are closed. The ability to stand steadily with the eyes closed is even more affected than the ability to walk. When the patient attempts it he sways much more than he would if in health. This swaying is known as Romberg's symptom, or ataxia of station. A very delicate test for ataxia of station is to have the patient try to stand on either foot with the eyes closed; if he can do this without difficulty ataxia in the lower limbs is excluded.

The gait of cerebellar ataxia may sometimes appear to be like that of spinal ataxia, but is generally clearly distinguished by a tendency to reel from side to side, as in ordinary drunkenness, a tendency which is but slightly increased by closing the eyes.

Ataxia of the upper limbs is indicated by an irregular deviation of the hand in trying to touch the nose with the tip of the forefinger or to bring the tips of the forefingers together. It is much increased by closing the eyes.

Ataxia of the muscles of the trunk is shown by swaying on trying to sit steadily erect without such support as the back of a chair would give.

Tremor is a symptom so familiar that it calls for no special description, but care should be taken to note what parts tremble, the character of the tremor, whether fine or coarse, rapid or slow, regular or irregular, and under what conditions it occurs. When it occurs only on attempting a movement, as in disseminated sclerosis, it is called intention tremor. The flickering tremor of small parts of a muscle, seen when motor nuclei are degenerating, and the facial tremor of paretic dementia are of especial importance.

Spasm, or abnormal involuntary muscular contraction, is generally easily recognized. The most important things

to note concerning it are whether it is tonic or clonic, and whether it affects the whole or part of a single muscle, a group of muscles or the muscular system generally.

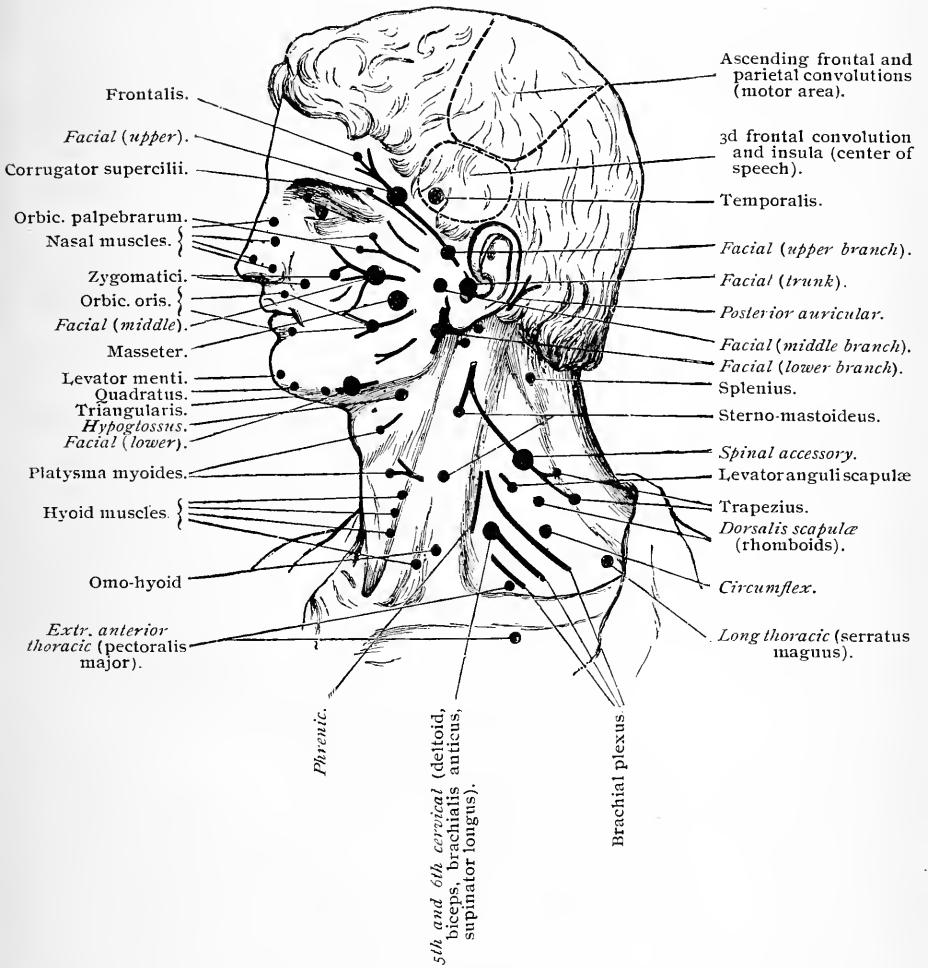
ELECTRICAL REACTIONS AND TROPHIC CONDITIONS OF MUSCLES.

The most useful electrical test is that of faradic irritability. It is easily made by placing one electrode of a faradic battery over the motor point of the muscle (the point where the principal nerve enters) and, after placing the other electrode on any convenient part of the body, turning on the current. If the muscle is in a healthy condition and the current of moderate strength a contraction will at once occur. This contraction is tetanic if the primary current is rapidly interrupted by the automatic vibrator common to all such batteries. In testing, however, it is better that the interruptions should be at longer intervals so that single contractions may be observed and a minimum of discomfort caused. If the battery is not specially provided with a slow interrupter the same result may be obtained by first starting the battery and then turning back the adjusting screw of the vibrator just far enough to prevent its working in the ordinary way, after which the electrodes are to be applied and the vibrator flicked with the finger so as to momentarily close the primary circuit and immediately open it again. Each time this is done there is an isolated secondary shock to which the muscle responds with a single contraction. The exact location of the motor points is easily learned by practice with the aid of the accompanying diagrams.

Where the nerve supplying a group of muscles is readily accessible, for example, the ulnar where it passes the elbow or the external popliteal where it passes around the head of the fibula, the whole group may be made to contract at once by applying the electrode over the nerve.

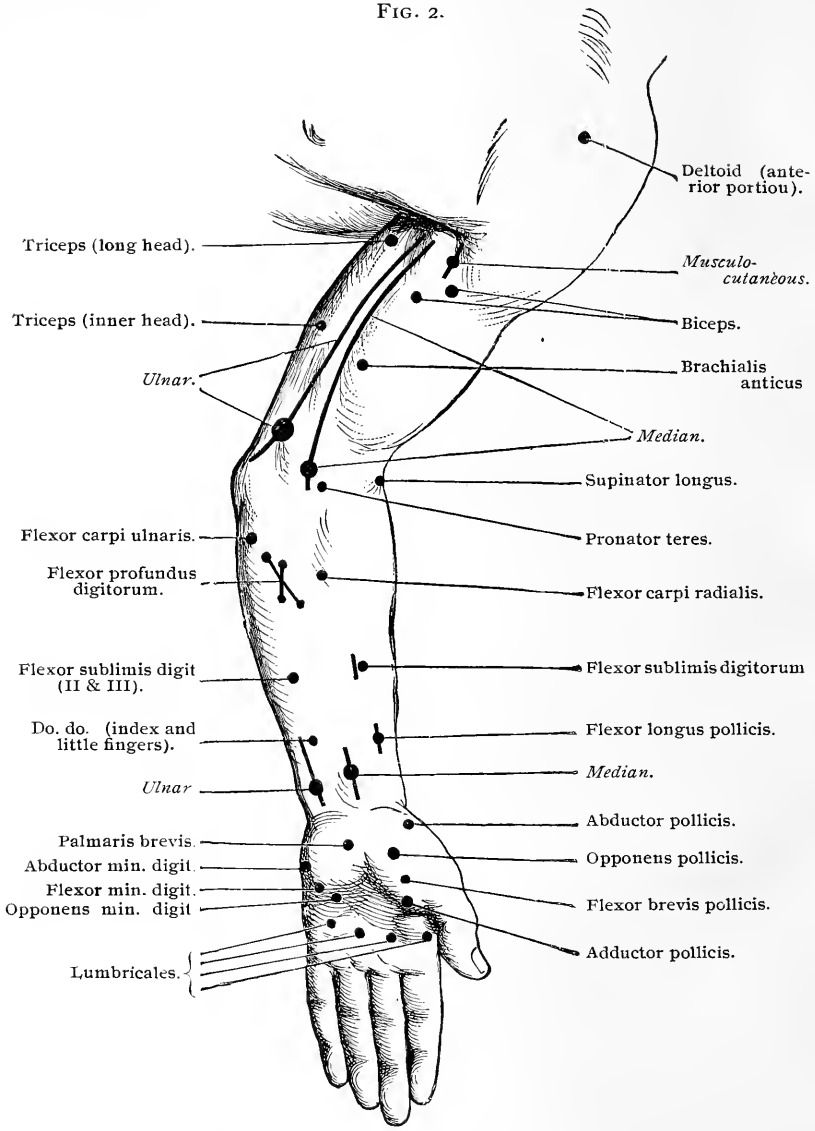
MAPS OF MOTOR POINTS.

FIG. 1.



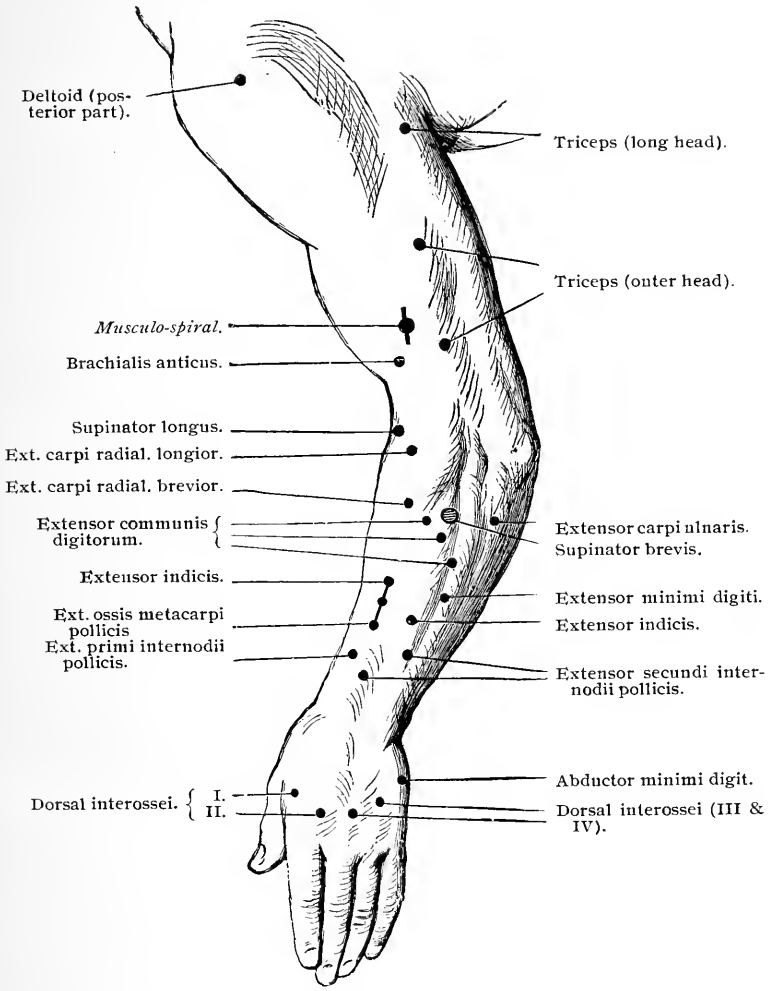
MOTOR POINTS ON FACE AND NECK.
(*From Ormerod, after Erb and DeWatteville.*)

FIG. 2.



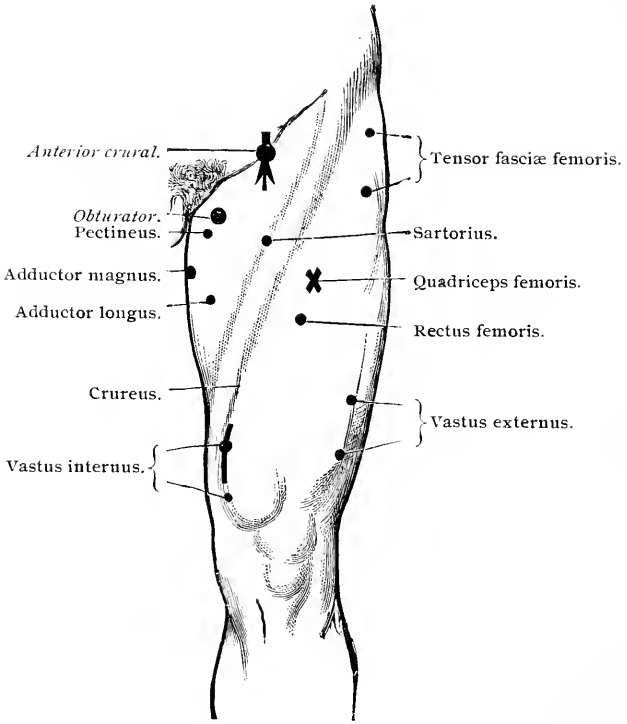
MOTOR POINTS ON UPPER LIMB, FLEXOR SURFACE.
(*From Ormerod, after Erb and De Watteville.*)

FIG. 3.



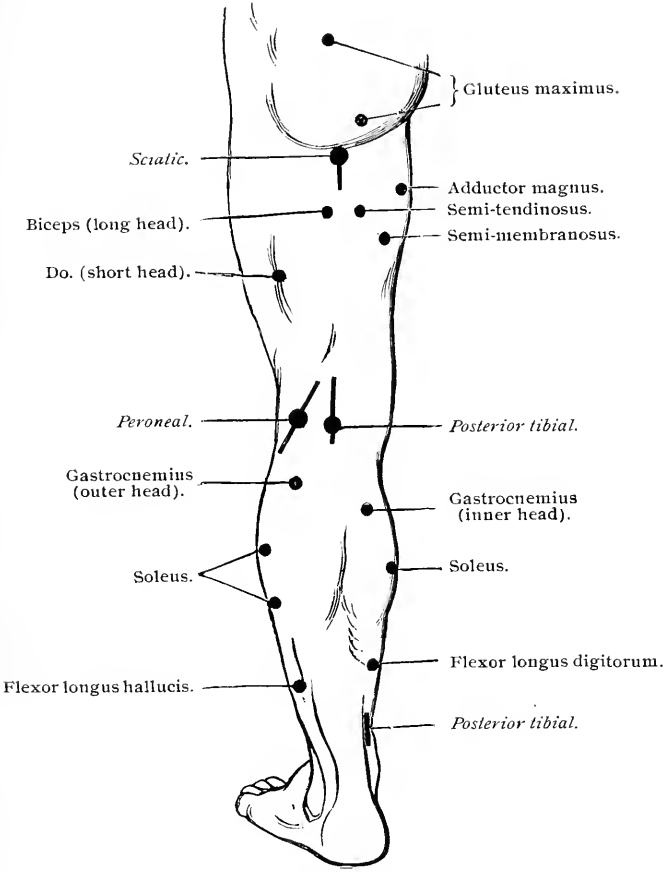
MOTOR POINTS ON UPPER LIMB, EXTENSOR SURFACE.
(*From Ormerod, after Erb and De Watteville.*)

FIG. 4.



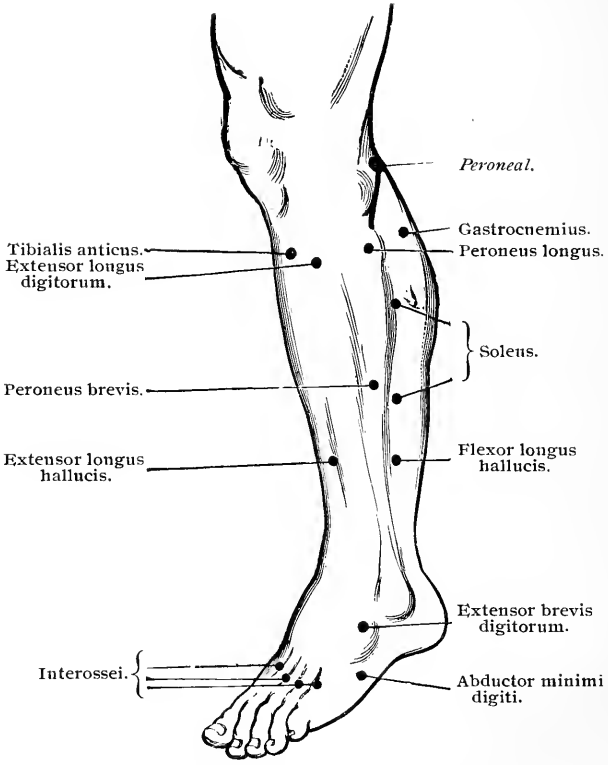
MOTOR POINTS ON THIGH, ANTERIOR SURFACE.
(*From Ormerod, after Erb and DeWatteville.*)

FIG. 5.



MOTOR POINTS ON LOWER LIMB, POSTERIOR SURFACE.
(*From Ormerod, after Erb and De Watteville.*)

FIG. 6.



MOTOR POINTS ON LEG, EXTERNAL SURFACE.
(*From Ormerod, after Erb and De Watteville.*)

Muscles that are paralyzed by functional disease or by organic disease of the upper motor segment (that is of the

FIG. 7.

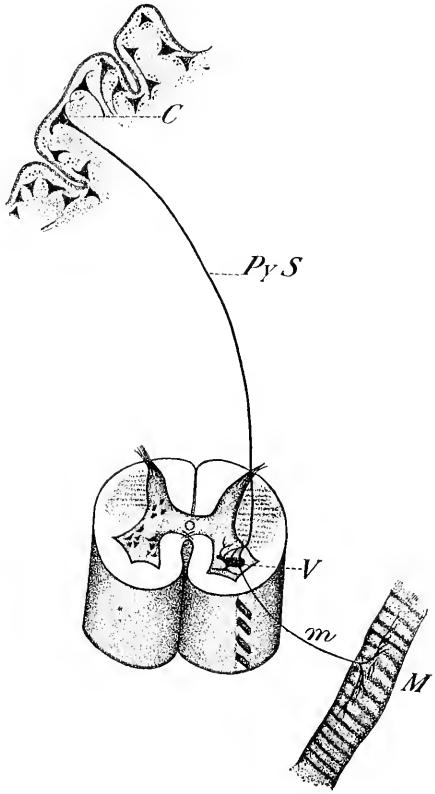


Diagram of an Element of the Motor Path.—(*Tyson after Strumpell.*) *C*, motor ganglion cell in the cerebral cortex; *Py S*, lateral pyramidal tract, central or upper motor neuron; *V*, ganglion cell of anterior horn; *m*, motor nerve, peripheral neuron; *M*, muscular fiber.

cortical motor centers or pyramidal tracts, *C* or *Py S*, Fig. 7) retain their faradic irritability unimpaired and, with very rare exceptions, they do not waste. But muscles paralyzed

by organic disease of the lower motor segment (that is of the anterior horns of the spinal cord, the motor nuclei at the base of the brain or the peripheral nerves, *V* or *M*, Fig. 7), as in poliomyelitis, bulbar paralysis or neuritis, are in a very different condition. Within about two weeks of interruption of this part of the motor tract their faradic irritability is lost or impaired, according to the severity of the disease. This is because the faradic current can make a muscle contract only by exciting its motor nerve fibers and in this class of diseases the nerve fibers are degenerated. This degeneration also causes the muscles to waste by preventing them from receiving the normal influence of their trophic nerve centers. Moreover, they lose their tone and their tendon reflexes. When the faradic irritability of a muscle is merely diminished, the diminution may be detected by first applying the current to the affected muscle and then to another known to be healthy, preferably to the corresponding one on the opposite side.

The tests of the galvanic irritability of a muscle are not so simple as the Faradic test because the positive and negative electrodes have an unequal effect on the muscle and a reaction is sometimes caused by opening as well as by closing the circuit. Only the most elementary account of the galvanic reactions will be given here, but it will suffice for practical diagnosis.

If the negative electrode, or kathode, be placed over the middle of a healthy muscle, the positive one being on any convenient part of the body, and a current of about five milliampères gradually turned on, there will be no visible effect. But if the current be interrupted and then suddenly closed, the muscle will give a single quick contraction at the moment of closure. This is called the kathodal closure contraction and is often abbreviated to K. C. C. If the current now be reversed so that the positive pole, or anode, is

on the muscle, and closure be made after an interruption, there will be no visible contraction or a weaker one than that obtained with the kathode: only by using a somewhat stronger current can the anode be made to cause a contraction equal to the previously obtained kathodal closure contraction. Hence it is said that kathodal closure contraction is greater than anodal closure contraction, or, in abbreviated form, K. C. C > A. C. C.

In functional paralysis and in paralysis due to organic disease of the upper motor segment the galvanic reactions of the muscles remain as in health. But in muscles whose lower motor segment (*V* or *M*, Fig. 7) is diseased, especially if the disease has advanced rapidly, there is a striking change. We have already seen that such muscles lose their faradic irritability. Galvanic irritability, on the contrary, is commonly increased, so that a contraction is caused by a weaker current than is necessary for the normal muscle. This contraction, however, instead of being quick, as in the normal muscle, is sluggish and suggests the contraction of a worm. Moreover, closing the current causes a greater contraction with the positive pole on the muscle than it does with the negative pole, which is expressed by the formula A. C. C. > K. C. C., just the reverse of that for the healthy muscle. These two peculiarities, the sluggishness of the contraction and the excess of A. C. C. over K. C. C. constitute the reaction of degeneration, which is often abbreviated to R. D. The reaction of degeneration appears within about two weeks of complete and rapid interruption of the lower motor segment and persists for some months, giving way to the normal reaction if the motor tract be restored and passing into complete loss of galvanic irritability if there be no restoration. In very gradual destruction of the lower motor segment, as in the slower cases of spinal muscular atrophy, it does not appear at all and there is a

simple loss of galvanic irritability. Muscles showing the reaction of degeneration are flabby, have lost their tendon reflexes and are soon conspicuously wasted.

THE REFLEXES.

All reflex actions are alike in that their occurrence is proof of the organic integrity of the reflex arc, consisting of a sensory tract, nerve center and motor tract. They differ greatly, however, in diagnostic import and are generally divided into three classes: (1) Tendon, or deep reflexes; (2) superficial, or skin reflexes, and (3) visceral reflexes.

A tendon reflex is the process by which a sudden increase in the tension of a muscle, usually brought about by tapping its tendon, evokes a muscular contraction in response.

Of all the tendon reflexes the knee-jerk is by far the most important and its utilization in diagnosis was one of the great achievements of modern neurology. It tells so much and tells it so quickly that every one who practices medicine should use it as habitually as he does percussion or auscultation.

The knee-jerk is usually elicited by having the patient sit with one knee crossed over the other and then striking a recoiling blow upon the patellar tendon with the ends of the fingers. In ordinary cases the foot will promptly be jerked forward by a contraction of the quadriceps extensor muscle. If the patient is in bed the test may readily be made, without disturbing him, by raising the knee so that the leg and thigh form an angle a little greater than a right angle, the foot resting easily on the bed, and then striking the tendon. In this case the foot may not move, unless the reflex is exaggerated, but the contraction of the quadriceps is easily seen or felt.

After some practice the examiner can tell at once whether the knee-jerk is normal, exaggerated or diminished, especially if the responses on the two sides are carefully compared. In exaggeration not only is the range of motion increased, but a response is evoked by a lighter blow on the tendon than is necessary in normal cases. When the response is slight it may be made more distinct by Jendrassik's method of reinforcement, which consists in having the patient make some effort with the upper part of the body, such as pulling on his clasped hands or pressing the examiner's hand, at the moment the tendon is struck. The eyes should be closed at the same time.

If the knee-jerk is not elicited by the tests so far described it is not to be regarded as absent until further trials show that it can not be elicited by any means whatever. Perfect relaxation of all the muscles of the knee is essential in all doubtful cases and the examiner should not only tell the patient to relax and let the leg hang limp, but he should feel of the flexor tendons and quadriceps to be sure that they are lax. Having the patient sit on the edge of a table with the legs hanging free may be necessary to secure full relaxation. A single distinct response is to be taken as outweighing all previous failures. But if relaxation has been secured and reinforcement employed and striking the tendon still evokes no response, especially if tests have been made on different days, the very significant note is to be made that the knee-jerk is absent.

The reflex arc for the knee-jerk consists of sensory fibers of the anterior crural nerve passing from the quadriceps extensor muscle to the second, third and fourth lumbar segments of the spinal cord, of these segments themselves and of the motor fibers passing from them back to the muscle. Organic disease interrupting this arc at any point must obviously prevent the occurrence of the reflex and, for some

unexplained reason, traumatic destruction of a dorsal or cervical segment of the cord also abolishes it, although less severe injury or disease in the dorsal or cervical part of the cord exaggerates it. Accordingly we find the knee-jerk to be absent in all organic diseases of the anterior crural nerve, of the corresponding nerve roots or of the second, third and fourth lumbar segments of the cord (such as tabes, neuritis, poliomyelitis and myelitis) and also in the severer cases of fracture-dislocation of the spine. Conversely, the persistent absence of knee-jerk is proof of some such organic disease.

Exaggeration of the knee-jerk occurs in all organic diseases which impair the integrity of the upper (cortical) motor segment for the quadriceps extensor, provided the reflex arc is intact, or that only a moderate proportion of its motor or sensory neurons is degenerated, and that complete destruction of any cervical or dorsal segment of the cord has not occurred. Such diseases are vascular lesions, inflammations and degenerations in the brain, cervical or dorsal myelitis, lateral sclerosis, postero-lateral sclerosis and amyotrophic lateral sclerosis. They cause exaggeration of the knee-jerk and other tendon reflexes by interrupting the normal cerebral control over the spinal centers and, in proportion to the degree of interruption, the exaggeration may be slight or very great. In the latter case if the patella is grasped and quickly pulled downward a series of rapidly recurring contractions, constituting patellar clonus, may occur. In hysteria, neurasthenia and other functional diseases the knee-jerk is often exaggerated but not to the degree which is common in organic disease. Very great exaggeration, therefore, especially if accompanied by ankle clonus, is to be taken as presumptive proof of organic disease but moderate exaggeration may be due to either organic or functional disease.

The presence of knee-jerks that are equal on the two sides and normal also has great significance; their presence and equality exclude any disease seriously affecting either of the reflex arcs and the absence of exaggeration excludes any disease of the brain or spine involving the upper motor segment.

What the knee-jerk tells may be summed up thus:

1. Its absence is evidence either of organic disease of some part of the reflex arc or of complete destruction of a cross-section of the cord.

2. Its great exaggeration along with ankle clonus is proof of organic disease affecting the upper motor segment for the leg, but its moderate exaggeration may be due to either functional or organic disease.

3. Its presence in normal and equal degree on the two sides is proof of the absence of any organic disease of the reflex arc and of any organic disease of the brain or cord affecting the upper motor segment for extension of the knee.

The Achilles tendon reflex, or heel-jerk is elicited by supporting the foot lightly, the knee being slightly flexed, and, after seeing that the limb is passive, striking the tendon a recoiling blow with the ends of the fingers. Ordinarily the blow is followed in about a tenth of a second by a contraction of the calf muscles and a corresponding movement of the foot. This movement is normally much less conspicuous than the knee-jerk but it may be much exaggerated in disease. When thus exaggerated a form of the reflex called ankle clonus may be produced by supporting the leg with the knee slightly flexed and, after securing relaxation, making an abrupt but not too forcible attempt to passively flex the foot. The sudden tension of the calf muscles causes a reflex contraction and, if pressure is maintained on the sole of the foot, the tension is instantly re-

newed so that a series of contractions occurs, making the foot vibrate at the rate of five to nine times a second. In typical ankle clonus this vibration continues for a considerable time if the proper degree of pressure is maintained. Ankle clonus may also be elicited by having the patient sit with the toe resting lightly on the floor, the heel being an inch or two above it, and then smartly pressing the knee downward so as to flex the foot. In some conditions clonus also appears when the patient attempts to walk.

The reflex arc for the heel-jerk consists of the fifth lumbar and first sacral segments of the cord together with sensory and motor fibers of the spinal cord connecting them with the calf muscles. The presence of heel-jerks that are normal and equal on the two sides excludes organic disease (such as neuritis, tabes and myelitis) affecting the reflex arc at any point and also organic disease of the brain or upper part of the cord affecting the upper motor segment for the calf muscles. The absence of the reflex, on the other hand, is not to be taken as proof of disease unless corroborated by other signs. When exaggeration is so great that typical ankle clonus can be elicited organic disease of the upper motor segment certainly exists; the spurious clonus occasionally seen in severe hysteria has a slower rate, and generally ceases after a few vibrations. Moderate exaggeration, however, is often seen in functional as well as in organic disease.

Although the knee-jerk and heel-jerk are by far the most important tendon reflexes, there are others which should always be tested when disease of the corresponding sensory or motor tracts is in question. In the upper limb reflex muscular contractions may often be evoked by tapping the tendons of the pectoralis major, triceps, biceps or any of the muscles moving the wrist or fingers. The limb should be passive

in such a posture that the muscle to be tested is but slightly stretched. The centers for these reflexes are at various levels from the fifth to the eighth cervical segments. Under any conditions the presence of a reflex is proof of the integrity of its reflex arc. If a muscle is paralyzed absence of the tendon reflex, except in rare cases of hysteria, is proof of lesion of the lower motor segment, that is of the cord or nerves, while great exaggeration is proof of organic lesion in the upper motor segment. Moderate exaggeration may be a symptom of either organic or functional disease. Absence of the reflex in a muscle otherwise normal has no positive significance.

A tendon reflex of the muscles of mastication, which has received the uneuphonious name of jaw-jerk, may sometimes be elicited by downward tapping on the half-dropped lower jaw. When exaggerated it indicates disease of the upper motor segment for the muscles of mastication.

The superficial reflexes are muscular contractions caused by irritation of the skin or mucous membrane. The most important of these are the plantar, gluteal, cremasteric, lower abdominal, epigastric, palmar and scapular, having their centers at various levels of the spinal cord, and the conjunctival, having its center at the base of the brain.

The plantar reflex is elicited by scratching or tickling the sole of the foot, and consists first of a movement of the toes, if the irritation is slight, followed, if the irritation is stronger, by flexion of the hip, knee and ankle so as to withdraw the foot. Its center is in the first three sacral segments. It has recently been proved that except in infancy the normal response of the toes is flexion and that if their first movement, particularly that of the great toe, is extension, without dorsal flexion of the foot, a lesion of

the upper motor segment for the foot is almost always present.

The gluteal reflex is a contraction of the gluteal muscles in response to an irritation of the skin of the buttock. Its center is in the fourth and fifth lumbar and first sacral segments.

The cremasteric reflex is a contraction of the cremaster muscle, drawing the testicle upward, caused by irritating the skin on the inside of the thigh. Its center is in the first three lumbar segments.

The lower abdominal reflex is a contraction of the abdominal muscles in response to an irritation of the skin in the iliac region. Its center is in the lower five dorsal and first lumbar segments. The epigastric, or upper abdominal reflex is a dimpling of the epigastrium in response to an irritation of the skin over the lower anterior margin of the chest and has its center in the fourth to seventh dorsal segments.

The palmar reflex is a flexion of the fingers caused by irritating the palm. It is generally absent except in young children and has its center in the lower two cervical and first dorsal segments. The scapular reflex is a contraction of the supraspinati and infraspinati muscles in response to an irritation of the skin over them. Its center is in the lower four cervical segments. The conjunctival reflex is the well-known closure of the eyelids caused by irritation of the conjunctiva. Its center is in the pons.

In testing the superficial reflexes it is best to make the irritation of the skin rather sharp so as to get a response at the outset, as the reflex irritability is rapidly dulled by the repetition of gradually increasing irritations.

The importance of the superficial reflexes consists in their presence being proof of the integrity of the respective reflex arcs; the exaggeration or absence of most of them

is of little significance because it may be caused by many trivial variations from health as well as by severe ones.

The visceral reflexes to be kept in mind in a neurological examination are the palatal, pharyngeal and laryngeal and the anal and vesical. The three first named may be elicited in health by touching the respective parts with a feather or probe but are absent or diminished in bulbar paralysis, diphtheritic paralysis and some cases of hysteria. The anal and vesical reflexes are, of course, always normal when micturition and defecation are normally performed; they are abolished by disease of the third and fourth sacral segments or the corresponding nerves, in which case there will be inability to retain urine or feces and digital examination will find the sphincter ani loose and flabby instead of contracting on the finger; these reflexes, although not lost, are disturbed by disease of the cord above the centers and by bilateral brain disease, in which case there is both retention and incontinence of urine and feces, that is inability to secure voluntary evacuations and inability to place restraint upon involuntary ones, but the sphincter ani is firm and contracts on the examining finger.

Taking the reflexes as a whole, it will be seen that they furnish a means of testing an extensive series of reflex arcs whose centers form an almost unbroken line reaching from the lower sacral segments through the entire cord into the medulla and pons. As it is highly improbable, in any organic disease of the nerves, cord or brain, that all the reflexes should remain normal, their presence in a normal degree is often a strong reassurance when organic disease has been feared, and in medico-legal cases it may disprove the claim of an hysterical, neurasthenic or malingering plaintiff that he is suffering from an irreparable injury.

TESTS OF CUTANEOUS SENSIBILITY.

The sensibility of the skin to touch, pain and temperature may demand careful investigation in certain cases. In making the tests the patient is blindfolded or required to keep the eyes closed. Sensibility to touch is tested by lightly touching various points with a camel's hair pencil, a bit of absorbent cotton or the end of the finger and noting whether the patient feels the touch and can locate it accurately. To test pain the quill end of the pencil may be sharpened or the point of a pin used to prick the skin; when the pain sense is defective pricking the skin gives an impression of touch instead of a sharp sting. The temperature sense is tested by applying bottles or test-tubes, one filled with warm and the other with cool water, the temperatures being such as to cause a distinct feeling of warmth or coolness to the normal skin.

These tests are quite decisive and easily made when the cutaneous sensibility is normal or is greatly impaired, but when the defect is slight the investigation is tedious and perplexing. In such a case it is important to compare the surface whose sensibility seems to be diminished with a part known to be normal, especially with the corresponding part on the opposite side of the body, and for a delicate test the two parts should be touched or pricked at the same time. In all cases in which sensory loss is an important factor in diagnosis the limits of the insensitive area should be carefully marked on the body and then transferred to a diagram so as to determine as accurately as possible whether it corresponds to the area of one or more nerves or spinal segments or merely to the external configuration of some part of the body or to a geometric area. (Figs. 8 to 11, 28 and 29.)

FIG. 8.

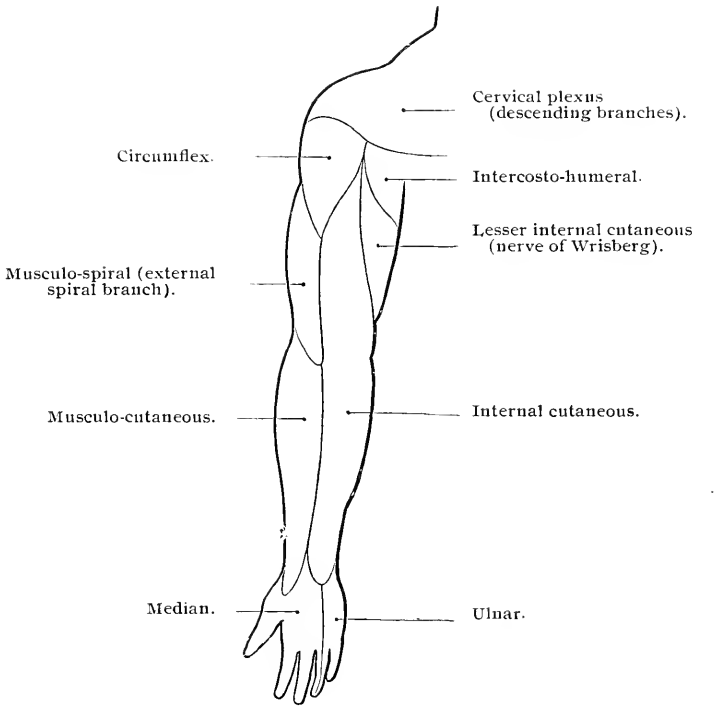
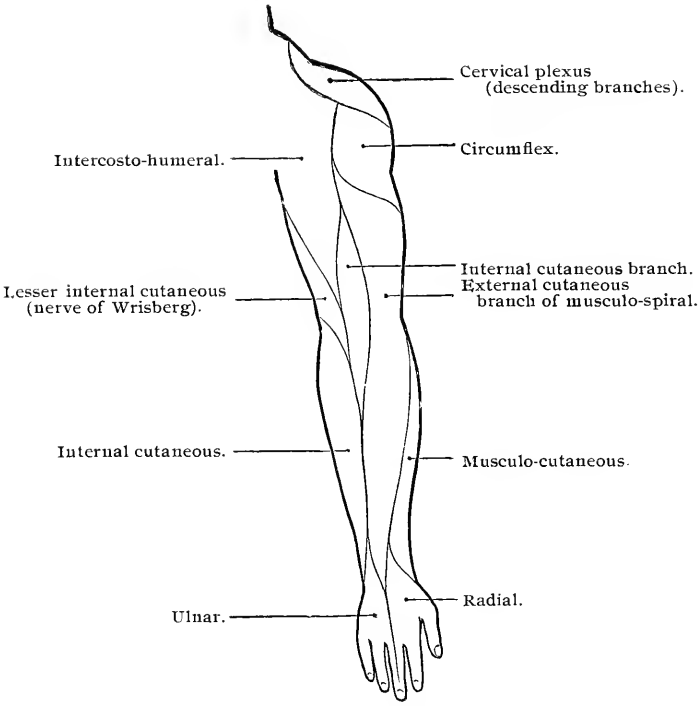


DIAGRAM OF CUTANEOUS NERVE-SUPPLY OF UPPER LIMB.
(*From Ormerod, after Flower.*)
Anterior Surface.

FIG. 9.



Posterior Surface. (*From Ormerod*)

FIG. 10.

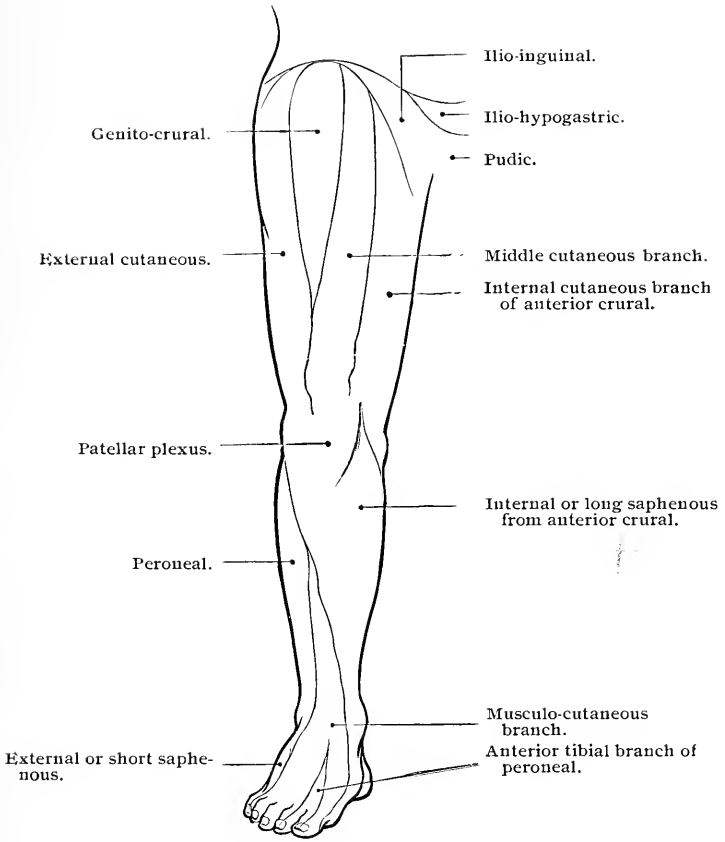


DIAGRAM OF THE CUTANEOUS NERVE-SUPPLY OF THE LOWER LIMB.

(*From Ormerod, after Flower.*)

Anterior Surface.

FIG. 11.

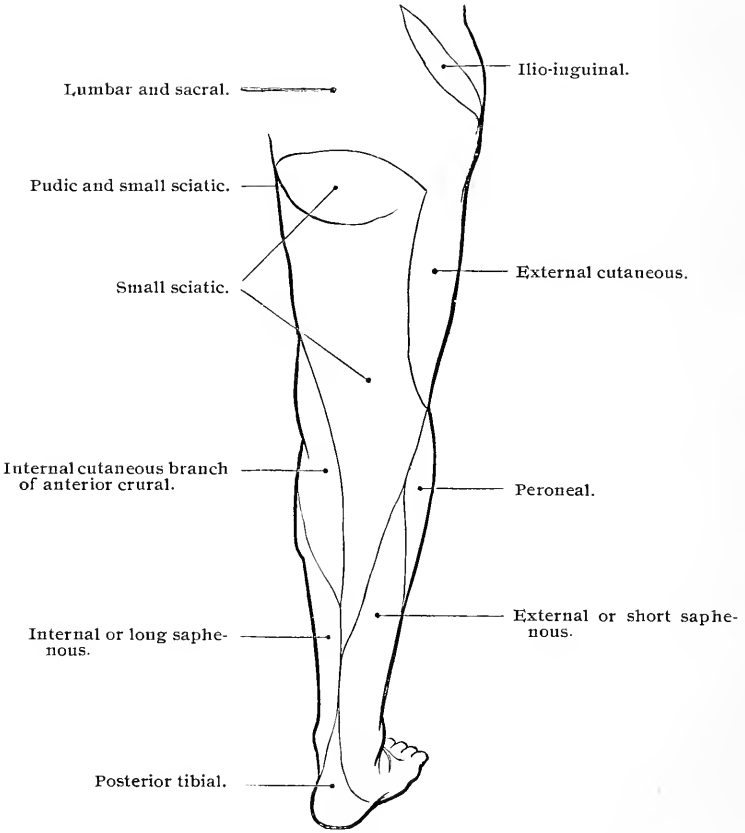


DIAGRAM OF THE CUTANEOUS NERVE-SUPPLY OF THE LOWER LIMB.
(*From Ormerod, after Flower.*)
Posterior Surface.

THE POSTURE SENSE.

The recognition of the posture of different parts of the body, without the aid of sight or touch, depends mainly on muscular sense, but is aided by the sensibility of the various parts of a joint and of the skin over it. It is tested by having the patient's eyes closed and then firmly grasping the parts on one or both sides of a joint and putting it in different postures, telling the patient to imitate each posture with the opposite limb or to tell what is being done.

TASTE AND SMELL.

For testing taste one should have on hand solutions of sugar, common salt, citric acid and quinine, to be used in the order named. The tongue is protruded and held by the examiner while some of the solution on a brush or wisp of cotton is gently rubbed in on either side. The patient should nod assent or dissent to the examiner's questions without having the tongue released. Sometimes, however, the taste is not perceived until the tongue is replaced, when it immediately becomes quite distinct. This as a rule has no clinical significance.

Smell can easily be tested by dropping some perfume of any kind on a little cotton and holding it to either nostril.

EXAMINATION OF THE EAR.

Inspection of the external ear may reveal anomalies of form indicating degeneration and predisposition to neurotic affections.

Hearing is most easily tested by holding the watch opposite either ear and noting the maximum distance at which it can be heard. This distance is to be put down as the numerator of a fraction of which the normal distance is the denominator; thus if the watch is heard at 20 inches

and no farther, and the normal ear hears it at 30 inches, hearing is recorded as 20/30.

If the watch shows deafness on one side the next question is whether it is due to disease of the external or middle ear or to disease of the internal ear, auditory nerve or brain. This question can be answered, aside from inspection of the external auditory canal and tympanic membrane, by placing the handle of a vibrating tuning fork on the top of the head or on the upper teeth and noting in which ear the sound seems louder. If it is louder in the deaf ear then bone conduction is not impaired and the disease is in the external or middle ear and has no significance from the strictly neurological point of view. But if it is louder in the sound ear bone conduction is impaired in the deaf one so the disease must be in the inner ear, auditory nerve or brain and the defect is called nervous deafness.

The result of this test may be confirmed by comparing the patient's bone conduction with the examiner's in the following way: The tuning fork while vibrating strongly enough for the patient to distinctly hear it by bone conduction is pressed on the temporal bone just above and behind the ear. The sound gradually becomes fainter and the patient makes a signal as soon as he no longer hears it. The examiner then immediately transfers the fork to the same position on his own head and if he still hears it the patient's bone conduction is impaired and the deafness is nervous. This method is especially valuable when hearing is impaired on both sides.

In certain cases of nervous deafness it may be important to determine the limits of the auditory field for high notes by means of Galton's whistle.

EXAMINATION OF THE EYE.

A mere inspection of the eyes will often reveal impor-

tant symptoms, such as drooping of the upper lid, protrusion of the ball, difference in the pupils, strabismus and nystagmus.

Proceeding to a more minute examination, the pupils should be compared with each other when exposed to light and again when shaded. Then each pupil should be tested for light reaction while the other eye is closed, the patient looking at some distant object so as to eliminate the effect of accommodation. In health each pupil contracts when a near object is looked at. This is called the reaction to accommodation, or convergence, and is tested by observing the pupils while the patient looks first at a distant object and then at the point of a pencil close at hand, the line of vision remaining the same so as to exclude variations in the light.

Inequality of pupils without loss of light reaction may be due to so many conditions of the eye, of the nervous system and even of other organs, that it has no definite significance when considered alone. But when it appears in addition to cerebral symptoms, such as headache, vomiting and disturbance of consciousness, and cannot be accounted for by disease of the eye or of the chest or neck, it is strongly indicative of organic cerebral disease. Absence of light reaction, if not caused by a drug or by disease within the orbit, is proof of cerebral or cerebrospinal disease, which is almost invariably organic. Absence of light reaction together with preservation of reaction to accommodation constitutes the Argyll-Robertson pupil, a sure sign of degenerative disease of the central nervous system, often seen in tabes and sometimes in paretic dementia.

A general idea of the condition of the motor apparatus of the eyes may be obtained by observing them while the patient looks to the right and left, upward, downward and

obliquely. Nystagmus, a tremulous or jerky oscillation of the eyeball, may be apparent in the extreme positions although entirely absent when the eyes look straight ahead.

Paralysis of the ocular muscles is of the greatest significance and must be looked for whenever organic disease within the cranium is suspected. Paralysis of the levator palpebræ is easily recognized as it causes ptosis, or drooping of the upper lid, which can be due to no other cause except spasm of the orbicularis. This spasm is rare and can readily be distinguished as an active resistance to the separation of the lids which causes fine, concentric wrinkles in the skin over them.

Paralysis of any of the external muscles of the eyeball is indicated by four symptoms: (1) Displacement of the eye at rest in a direction opposite to that in which the paralyzed muscle should move it; *e. g.*, in paralysis of the external rectus the eye is turned inward. (2) Limitation of movement in the direction in which the affected muscle acts; *e. g.*, in complete paralysis of the external rectus the eye cannot be moved outward beyond the mid-position. (3) Diplopia, the image of the affected eye, called the false image, being displaced in the direction in which the paralyzed muscle should turn the eye; when the position of the object requires an effort to look in this direction the two images are farthest apart, but when the object is carried to the opposite side, so that no effort is required of the paralyzed muscle, the two images come together; *e. g.*, in paralysis of the right external rectus the image seen by the right eye (false image) is displaced to the right of the true one when the object is directly in front of the patient; when it is carried to the patient's right the images separate still farther but when it is carried to his left they come together again. In paralysis of the right internal rectus the false image is displaced to the left of the true

one, the two images coming together when the object is taken to the patient's right. It will be noticed that the diplopia is crossed (image of right eye to left and vice versa) when the axes of vision diverge and that it is homonymous (image of right eye to right and left to left) when the axes of vision are crossed.

4. Secondary deviation of the sound eye: When an attempt is made to look at an object in the direction in which the paralyzed muscle should move the eye, the sound eye, if covered, will move too far in this direction, but will immediately move back to its proper position when uncovered.

The displacement of the eye at rest and the limitation of movement are readily observed by the examiner except when the paralysis is slight or affects one of the oblique muscles alone. It must be remembered, however, that in coma and even in deep sleep the eyes may diverge and be turned upward and yet be perfectly normal when consciousness returns.

To study the diplopia let the patient look at a candle or a vertical strip of white paper and, in order to distinguish the two images, place a colored glass before one eye, preferably the sound one. If both images are not readily seen or if no colored glass is at hand, have the patient close first one eye and then the other, carefully noting the apparent change in the position of the object.

To test for secondary deviation hold a card between the sound eye and the point of a pencil held in such a position that to fix it with the affected eye the muscle in question must act. Then if the sound eye moves too far it can be observed and on quickly removing the card it will be seen to move back to its proper position.

The following table gives a condensed description of the signs of paralysis of each of the individual muscles:

TABLE OF SIGNS OF PARALYSIS OF EXTERNAL OCULAR MUSCLES.

Muscle.	Displacement of visual axis.	Limitation of movement.	Position of false image.	Secondary deviation of sound eye.
External Rectus.	Inward.	Outward.	To the side of affected eye.	Inward.
Internal Rectus.	Outward.	Inward.	To the side opposite that of affected eye.	Outward.
Superior Rectus.	Downward.	Upward.	Above and to side opposite that of affected eye.	Upward.
Inferior Rectus.	Upward.	Downward.	Below and to side opposite that of affected eye.	Downward.
Superior Oblique.	Difficult to detect.	Difficult to detect.	Below and to side of affected eye. Image tilted, top inward.	Downward and inward.
Inferior Oblique.	Difficult to detect.	Difficult to detect.	Above and to side of affected eye. Image tilted, top outward.	Upward and inward.

The two internal muscles of the eye, the sphincter of the iris and the muscle of accommodation are also liable to paralysis, which is indicated by dilatation of the pupil and loss of light reaction and reaction to accommodation in the case of the sphincter and by inability to focus the eye on near objects when the muscle of accommodation is paralyzed.

Individual ocular muscles may be paralyzed alone or in combination with others, according to the seat and extent of the disease. Such a paralysis is one of the strongest proofs of organic disease of the nervous system; it may in rare cases be due to toxic influences without organic change or to a functional neurosis like migraine or epilepsy but it is practically unknown in hysteria.

The external rectus is frequently paralyzed alone, being the only muscle supplied by the sixth nerve, which from its long course is especially exposed to danger in disease at the base of the brain. Isolated paralysis of the superior oblique is also not rare because it is the only muscle supplied by the fourth nerve. Either of these muscles may also be paralyzed by disease of the corresponding nucleus. All the other ocular muscles, external and internal, are supplied by the third nerve and, in a lesion of the nerve trunk, generally suffer together, so that the eye is turned outward, motion is limited inward, upward and downward, the lid droops, the pupil is dilated, accommodation is lost and there is crossed diplopia. When only a part of the muscles supplied by the third nerve are paralyzed the disease is more commonly in the nuclei beneath the aqueduct of Sylvius, but it may possibly be limited to the corresponding branches of the nerve.

So far paralysis of individual muscles or muscle groups has been spoken of. But there is another form in which the muscles, tested separately, give no characteristic sign of paralysis or even act normally, while there is distinct loss of power to perform one or more of the associated movements of both eyes: viz., convergence, divergence, looking to either side or upward or downward. As these associated movements are normally under voluntary control, it will readily be understood that they may be lost, as a result of suggestion in hysteria. They may also be lost in organic central disease, particularly of the cortex.

Spasm of ocular muscles, especially of the internal recti, may occur and may be mistaken for paralysis of the opponents. In spasm the limitation of movement, especially if the eyes be separately tested, is less marked and less constant, the double images do not separate on looking in

one direction to come together again on looking the opposite way and there is no secondary deviation.

Lack of proper balance of the muscles moving the eyes, not sufficient ordinarily to cause diplopia, may be of considerable importance in hysteria and neurasthenia, although the reports of remarkable cures from the correction of such a defect alone are to be received with much allowance for the patient's susceptibility to suggestion. The balance of the internal and external recti when the eyes are at rest can be tested by placing a prism of eight or ten degrees, with its base downward and accurately horizontal, in the trial frame before one eye, and then having the patient look at a candle or a vertical strip of paper or a horizontal scale at the other side of the room. The prism causes vertical diplopia and if the muscle balance is normal the upper image will be exactly above the lower or (on account of the slight convergence necessary in looking at an object only a few yards distant) very slightly displaced to the side opposite the prism. If the upper image is displaced a few inches or more to the side opposite the prism then there is a relative insufficiency of the internal recti. If the upper image is displaced to the same side as the prism there is a relative insufficiency of the external recti. The balance of the muscles which turn the eye upward and downward may similarly be tested by placing a prism of about ten degrees with its base inward and accurately vertical before one eye. If the double images of the test object are in the same horizontal plane the balance is normal. If the image of one eye is above that of the other and the vertical distance between them increases as the object is raised, the superior rectus of that eye is relatively weak. The converse, of course, indicates weakness of the inferior rectus.

The relative power of the muscles in action may be

measured by finding the strongest prism that may be placed before one eye and still permit the two images of the object to be fused into one. The internal recti should be able thus to fuse the two images with a prism of about thirty degrees placed before either eye, base outward. The external recti are much weaker, but should overcome a prism of about eight degrees, base inward. The superior and inferior recti should overcome a prism of about three degrees, base upward or downward. These figures are to be taken as merely approximate and considerable allowance is to be made for individual variations within physiologic limits, especially when there are no symptoms attributable to muscular strain.

Acuity of vision for distant objects is tested in each eye separately by having the patient read the smallest letters possible for him on a well-lighted test card across the room. The result is expressed by a fraction of which the distance of the card is the numerator and the distance at which the same letters can be read by a normal eye the denominator. Thus if the smallest letters read at 20 feet are read by the normal eye at 50 feet the vision is recorded as 20/50. Acuity for near objects and the range of accommodation can be measured together by finding the smallest type that the patient can read and the limits within which it is read.

When acuity of vision is impaired we must find whether the defect is due to an error of refraction, to disease of the media or to disease of the retina, optic nerve or brain. Any defect that can be fully corrected by a spherical convex lens is due to either hypermetropia or presbyopia. A defect which is limited to distant vision and can be fully corrected by a concave spherical lens is due to myopia. A defect which is different in degree for lines at the same distance, running in different directions, and can

be corrected only by the use of a cylindrical lens, with or without a spherical one in combination, is due to astigmatism. Disease of the media, retina or head of the optic nerve will be visible on ophthalmoscopic examination. Retrobulbar disease of the nerve or disease of the brain is to be inferred from the exclusion of other causes and the combination of symptoms present.

The fields of vision are of great importance in neurological diagnosis. To test them one eye should be covered and the other kept fixed on a small object straight ahead. Then a piece of white paper, two-fifths of an inch square, is brought forward on the outer side of the eye until it is just visible, which should be when a line from the paper to the eye makes an angle of about 90° with the line of direct vision. The paper is next brought forward on the nasal side until just visible, which should be at an angle of about 55°. When brought into view from above, it should be visible at about 50°, and from below at about 70°.

As the paper is brought in from the point at which it first becomes visible toward the line of fixation it should grow more and more distinct, except when it passes through the normal blind spot, whose center is about 13° to the outer side of the point of fixation. If it disappears in any other part of the field or grows indistinct the defect is called a scotoma.

After testing the fields for white, colored papers of the same size should be used, noting the greatest distance from the point of fixation at which the color can be recognized. Of the three colors most employed, blue has the largest field, red the next and green the smallest. The color fields may be defective in spots although no corresponding defect for white may exist; such spots are called color scotomata.

The foregoing rough method will suffice for ordinary cases, especially if it indicates a normal condition of the fields, but much more accurate results can be obtained by mapping them by the aid of some form of perimeter, as described in the text-books of ophthalmology.

The defects in the fields that are of most importance from the neurological point of view are hemianopia, or blindness in one half of the field, concentric limitation, or blindness in a peripheral zone, scotomata, or blindness in isolated spots, and reversal of the normal relations of the color fields, *e. g.*, that for green being larger than that for red.

Hemianopia is generally bilateral and homonymous, that is, both fields are affected and the right or left half of each is blind. In such a case the patient looking directly at any object with both eyes open sees only the right or left half of it. This symptom is conclusive proof of organic intracranial disease except when it is part of an attack of migraine and in extremely rare cases where an hysterical patient may have become so familiar with it as to make its acquirement by suggestion possible. The lesion may be in the optic tract, in the region of the anterior corpus quadrigeminum, in the optic radiation or in the cortex about the calcarine fissure; it is always on the side of the brain opposite the blind side of the fields. Peripheral limitation of the field occurs in hysteria, in which it is usually regular, and in atrophy of the optic nerve, in which it is apt to be irregular. Scotomata occur in diseases of the retina and of the optic nerve. Reversal of the normal relation of the color fields is generally believed to occur only in hysteria.

Skill in the use of the ophthalmoscope is of the greatest importance to the neurologist unless he can constantly command the services of an ophthalmologist, which, of

course, is seldom practicable. It should by all means be acquired through personal instruction but it is possible for the persevering to acquire it alone. For directions as to practice the reader is referred to any good text-book of ophthalmology. The student of neurology should especially practice the direct method and first learn to recognize the normal appearance of the disk and the changes caused in it by optic neuritis and optic atrophy. (Figs. 21 to 24.) He should then become familiar with the appearance of the various forms of retinitis and choroiditis. The colored lithographs, commonly furnished in the atlases and text-books, are invaluable for the student who cannot have personal instruction in a large clinic.

EXAMINATION AS TO SPEECH.

The various ways of using language are functions of a complicated nervous mechanism, easily deranged by disease, and the consequent defects are not only of great interest psychologically but are of great practical importance in diagnosis. In conducting the examination four lines of inquiry should be followed in regular order.

1. The patient's talk. Are words badly uttered? If so, is the defect due to spasmodic arrest of utterance or to the omission, replacement or wrong arrangement of difficult sounds? Are the intonation and accent normal to the patient? Entirely distinct from the mode of utterance is the question whether the words used (considering the patient's age and education) are sufficient in number, appropriate and properly arranged. Is the stock of words used very small? Is the same expression used over and over without regard to meaning? Are wrong words similar in sound or associated in meaning substituted for the right ones? Are correct and incorrect words mingled in a confused jargon? Can he name familiar objects? Can

he repeat the words he hears? If he does not talk at all may it be due to mental disease?

2. The comprehension of oral speech. Do the patient's answers to questions show that he understands them? Can he be made to comply with simple but unexpected requests, such as to raise the right hand or close the left eye, made in an ordinary tone, without gesture or change of facial expression? Can he select familiar objects when named? If comprehension is defective can it be explained by ordinary deafness?

3. The ability to read. Does the patient occupy himself with books and papers as he formerly did? Can he read aloud? Can he show that he comprehends what he reads? Does he comply with written requests to perform simple actions? Can he read and understand figures? Does he comprehend the nature of ordinary objects when he sees them? If there is any defect is it explained by simple loss of vision?

4. The ability to write. Can the patient write his signature? Are the letters properly formed or is there tremor or incoördination? Can he express himself in writing? Can he write from dictation? Can he copy? Can he write figures and perform arithmetical operations? Can he make even the crudest drawings of familiar objects? If there is any defect is it due to paralysis, and if he cannot write with the right hand can he with the left, or can he trace words with the foot?

EXAMINATION AS TO THE MENTAL CONDITION.

Before examining a person supposed to be insane, the family and personal history should, whenever possible, be obtained from a relative or friend, paying special attention to nervous or mental disease in other members of the family, to any change in the character of the patient and

the time at which the change began, and to any facts indicating failure of memory, depression, exaltation, hallucinations or delusions. Except in the rarest cases, where danger of violence or of obstinate refusal to be examined makes deception necessary, the physician should appear in his true character, but he should treat the patient as though sane and should examine as though looking for some purely physical ailment.

The usual questions as to age, place of residence, occupation, domestic relations, previous illnesses and present sufferings should be asked; the answers may not be reliable, but they are valuable as indications of the state of memory and the general mental condition. Sleep and dreams are important in themselves and as affording an easy and natural transition to hallucinations.

The questions are to be followed by a thorough physical examination of the nervous system and the most important organs, for much depends on the recognition or exclusion of organic nervous disease, infectious diseases and intoxications. Special attention is to be paid to the condition of the pupils, tremor, twitching of the face, articulation, pulse, temperature and the urine. Incidentally the physical examination will very probably cause the patient to reveal some of his mental peculiarities.

Proceeding to the psychological problems of the case, the patient is to be led into conversation about himself. If he talks freely it is seldom difficult to recognize the emotional exaltation and rapid flow of ideas characteristic of mania, the hopeless depression and slow ideation of melancholia or the weakened judgment and loss of memory indicative of dementia. Delirium (with its lack of consciousness of the present surroundings and incoherent hallucinations) and mere stupor are as a rule not hard to distinguish from the types of mental disease just mentioned, but transition forms may be perplexing.

The symptoms of paranoia are sometimes very difficult to detect. Hallucinations are to be inquired for, when suspected, as though they were ordinary symptoms, but with as smooth a transition as possible; thus if the patient is annoyed by steam whistles it is easy to ask whether the noise ever seems to take the form of words, then what the words are and what motive anyone could have for saying such things. Or, if there are unpleasant dreams, it is natural to ask what is seen in the dreams and then whether such things ever appear during the waking moments. If delusions are suspected but not revealed the conversation should be made to turn indirectly upon religion, politics, inventions, electricity or persecutions, according to the history previously obtained, and this will generally succeed. If a delusion be revealed the next question is whether it is systematized, that is, whether the patient can give logically connected reasons, however absurd they may be, for his belief.

Sometimes, especially if a reliable history has not been obtained, all our efforts will elicit nothing decisive. The patient answers quietly and naturally, showing no undue emotion and no defect of judgment or memory, and betraying no hallucinations or delusions. Such a person is either sane or a paranoiac who is skillfully concealing his delusions and it is generally better to postpone the examination to another time than to prolong it unduly and to seem too anxious to find out the patient's secret. In the meantime inquiries can be made and the best method of leading up to the probable delusion considered.

If the patient will not talk at all attention to his attitude and expression will almost always discover the reason. A dull, vacant or silly expression indicates idiocy, dementia or stupor. A sad or anxious look with an attitude of profound dejection indicates that speech is inhibited by mel-

ancholia. A rapt, ecstatic or keen and vigilant look, with an attitude of alertness, betrays the paranoiac who has a command from heaven not to talk or a delusion that talking will give his persecutors an advantage over him.

Cases of delirium or stupor, in which the patient is manifestly insane for the time being, but which are not typical of mania, melancholia, dementia or paranoia, cannot be elucidated by psychological methods alone. On the contrary, the results of the entire examination are to be considered in an effort to recognize the physical disorder of which the mental disturbance is but one symptom. Here, as in many other parts of the field of neurology and psychiatry, is an illustration of the fact that no one can be a competent neurologist or alienist without first being a competent general physician.

THE RECOGNITION OF ORGANIC DISEASE.

For clinical purposes all nervous disorders are divided into two classes, organic diseases, whose symptoms are caused by a visible alteration in the structure of the nervous system, and functional diseases, in which impaired nutrition of the neurons is assumed but in which the most refined methods of investigation fail to reveal visible structural changes. This is a somewhat crude division, but it has been of the greatest practical advantage and in the present state of knowledge is of fundamental importance.

It is generally necessary, before proceeding to the more special diagnosis to answer the general question whether the disease is organic or functional or a mixture of both. This question is often the most difficult one connected with the case and, as an incorrect answer is likely to lead to a useless or even disastrous plan of treatment, the symptoms which may be decisive are worthy of the most careful study. The presence or absence of signs indicating organic disease must alone be considered at first. Until this is done symptoms of functional disorder, however characteristic, should be allowed no weight, for hysteria or neurasthenia may complicate the gravest organic conditions. The significance of many of the signs of structural change has already been mentioned in discussing the examination of the patient, but it is desirable to consider them together.

Paralysis of external ocular muscles not due to local conditions within the orbit, unless limited to the conjugate motions of both eyes which one may voluntarily make or refrain from making, is almost absolute proof of organic disease within the cranium; the exceptions are certain extremely rare cases of migraine, epilepsy or toxemia. With the same qualifications failure of light reaction is equally significant, provided the influence of such drugs as atropine or physostigmin can be excluded. Mere inequality of pupils is not quite so important, but if it is known to have occurred during the progress of the disease in question and is not due to peripheral irritation on the side of the dilated pupil it is almost as conclusive. The Argyll-Robertson pupil is invariably due to degeneration of the central nervous system. Optic neuritis or optic atrophy is in itself organic nervous disease, and in connection with cerebral symptoms is proof of intracranial organic change, provided we can exclude certain forms of intoxication, particularly uremia, alcoholism, plumbism and tobacco poisoning. Well-defined and persistent hemianopia is always organic. As a transient symptom it may occur in migraine.

Paralysis of the face is practically always organic unless, as may rarely happen, it is caused by uremia.[1] Paralysis of the palate, except when a part of adynamic bulbar paralysis, is organic. It is especially significant as one of the earliest symptoms of an oncoming diphtheritic paralysis. Paralysis of laryngeal muscles if bilateral may be either organic or functional, but if unilateral it is always organic. Loss of control of the bladder and rectum

[1] Genuine cases of hysterical facial paralysis are so excessively rare that their possible occurrence may be disregarded in ordinary diagnosis. Glosso-labial spasm is more frequent.

is generally organic, and paralysis of the sphincters is always so.

The mowing gait or steppage gait is proof of organic paralysis. Paralysis, with or without corresponding sensory loss, which is definitely limited to the muscles supplied by nerve trunks or spinal segments is organic.

Atrophy of muscles with loss of faradic irritability is due either to organic nervous disease or to idiopathic disease of the muscles. Reaction of degeneration or fibrillation in a wasted muscle proves the existence of organic disease of the lower motor segment.

Genuine absence of knee-jerk is almost, but not quite absolute proof of organic disease of the nervous system or muscle. It is never a symptom of hysteria or neurasthenia, but may with extreme rarity be found in apparent health and it has been successfully simulated. Ankle clonus which persists for a considerable time and has a steady rate of five to seven vibrations per second is probably always organic. A clonus of very brief duration or a persistent one at an inconstant rate of two to four vibrations per second certainly may be hysterical. A spastic condition of the lower limbs, so extreme that when the examiner lifts one leg from the bed the pelvis and the other leg move with it, is always organic. The allied condition, called spinal epilepsy, in which a paroxysm of tonic spasm of the legs passes into clonic spasm, is also invariably organic. Localized convulsions of the Jacksonian type are proof of a correspondingly localized irritation of the cortex which is almost always caused by organic disease. A localized sensory aura is generally caused in the same way but there is a greater possibility of its being merely functional.

If no evidence of organic disease is found it is still not to be positively excluded, especially if the symptoms are

equivocal, until the time has elapsed in which organic change would necessarily reveal itself. Thus, if a delicate and possibly tubercular child has suffered for a week from headache, with occasional vomiting, the absence of all signs characteristic of organic disease requires the diagnosis of meningitis to be withheld, but does not exclude it, for within the next few days, pupillary changes, strabismus or some other conclusive symptom may appear. In the meantime the special diagnosis that would follow in either case must be considered in the light of all the facts already known. Conversely, the longer a disease has lasted without proof of its organic nature the greater the assurance that it is only functional. If a partial hemiplegia has lasted for months without such proof, and the gait and other symptoms are characteristic of hysteria, organic disease may almost certainly be excluded.[1]

It will probably be inferred from the foregoing statements that the recognition or exclusion of organic disease in general is sometimes very difficult or even, for the time being, impossible; in fact it is generally this part of the diagnosis which calls for the greatest caution and the highest skill in weighing evidence.

The organic nervous diseases are divided into four groups: vascular lesions, including hemorrhage, embolism and thrombosis, inflammations, effects of pressure and morbid growths, and degenerations. These groups are clinically distinguished mainly by the time of onset, that is, the time that elapses between the appearance of the first symptoms and their attaining a considerable degree of intensity. The following table, from Gowers, shows the relation between the time of onset and the nature of the disease.

[1] Even then disseminated sclerosis might possibly underlie the hysteria.

Disease	*Onset*	*Disease*
	Sudden (few minutes)	Vascular lesions
	Acute (few hours or days)	
Pressure and growths	Subacute (one to six weeks)	Inflammation
	Subchronic (six weeks to six months)	Degeneration
	Chronic (more than six months)	

From this table it will be seen that, excepting injuries and merely functional disorders, a sudden onset always denotes a vascular lesion, as in a case of apoplexy. An acute onset is most frequently due to an inflammation, but it may be caused by a slowly developing vascular lesion, especially thrombosis or hemorrhage. A subacute onset denotes inflammation or the effect of pressure or morbid growth; it is too slow for a vascular lesion and too rapid for a degeneration. A subchronic onset denotes chronic inflammation, the effect of pressure or growth, or degeneration. A chronic onset denotes pressure or growth or a degeneration; it is too slow for even a chronic inflammation.

The pathological diagnosis is carried still further by a consideration of the causes of disease that may be found, the indications afforded by the accompanying symptoms and the seat of the disease, as shown by the localizing symptoms. These indications are applied in the tables which form the main portion of the book.

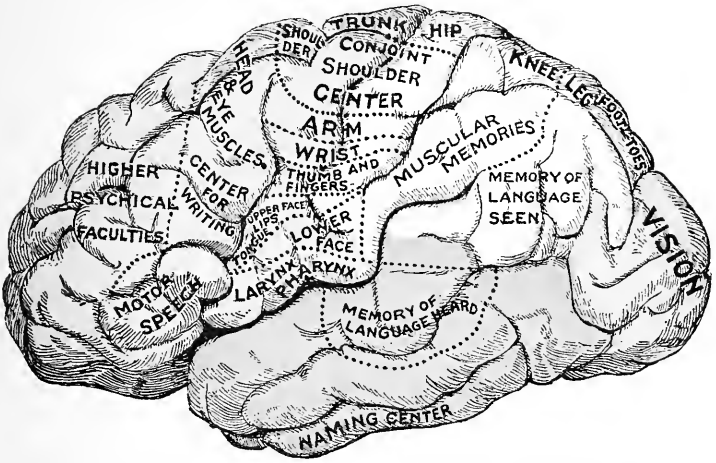
THE PRINCIPLES OF LOCALIZATION.

It has already been said that the kind of organic disease is to be inferred mainly from the time of onset; the seat of the disease is to be inferred from the part of the body whose function is disturbed and the character of the disturbance. If the hand is paralyzed it is known that the disease attacks some part of the motor tract for the hand, either the cortical center, the pyramidal fibers connecting it with the gray matter of the cervical enlargement of the cord, this part of the cord itself or the nerves connecting it with the muscles. A complete knowledge of localization would require a correspondingly complete knowledge of the anatomy and physiology of the nervous system in relation to all the rest of the body.

In applying this knowledge to clinical localization it is necessary to distinguish the comparatively limited symptoms caused by overaction or loss of function at the seat of the lesion alone from the more widespread disturbance which may be caused by pressure or shock transmitted to adjacent and even distant parts of the nervous system. Only the direct symptoms are available for a precise localization, although the indirect ones are important as a general indication.

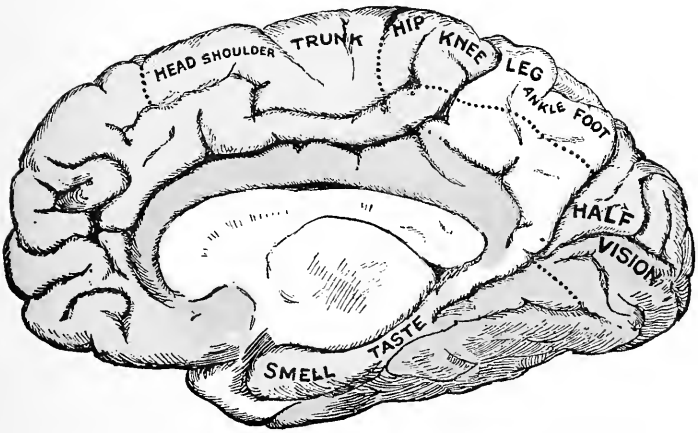
This distinction is made by comparing the extent of the symptoms at different times. Thus, a hemorrhage limited to the cortical center for the right arm, at first not only causes spasm and paralysis of the arm, but, by pressure and shock affecting the entire left hemisphere, it may cause complete hemiplegia, aphasia and unconsciousness.

FIG. 12.



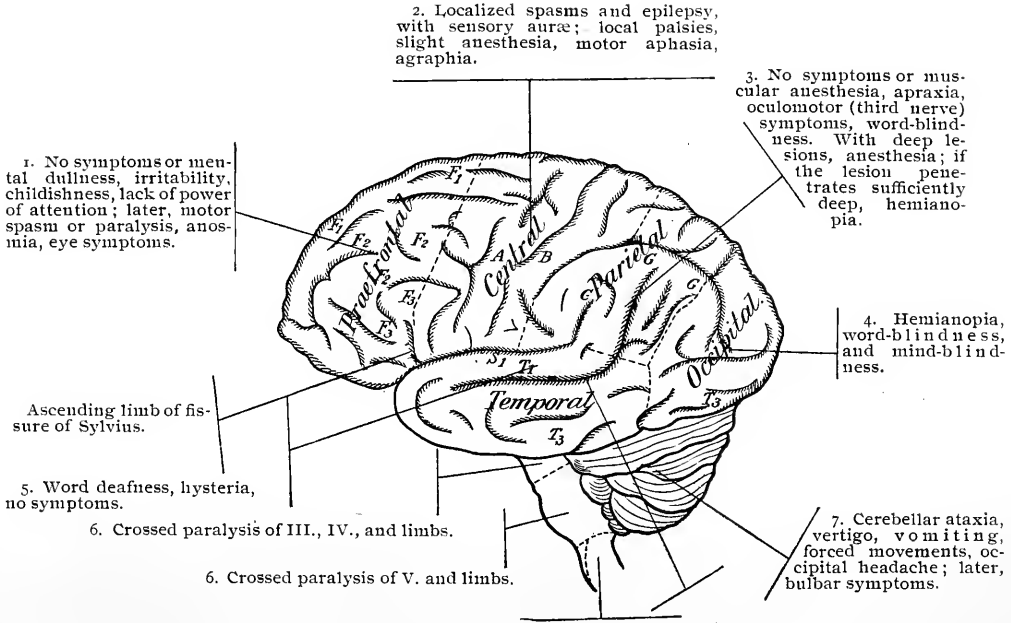
A DRAWING OF THE LEFT CEREBRAL HEMISPHERE (HUMAN).
Showing the different localizable areas on the external surface. (*From Gordinier.*)

FIG. 13.



A DRAWING OF THE RIGHT CEREBRAL HEMISPHERE (HUMAN).
Showing localizable areas on the median surface. (*From Gordinier*)

FIG. 14.



Showing Focal Symptoms of Brain Tumor. (*Tyson after Dana.*)

After a few days, however, consciousness and speech will have returned, the paralysis of the face and leg will have disappeared and only the paralysis of the arm will remain as a precise indication of the seat of the lesion. Hence, when the symptoms are of sudden onset, the later and more permanent condition is the one on which to base the localization. But, conversely, a rapidly increasing lesion, say a tumor, in the region of the cortical center for the right arm, may at first cause a spasm of the arm which precisely indicates the seat of disease; later, although the growth still occupies but a small part of the left hemisphere, its pressure and irritation may be transmitted to the entire brain, causing hemiplegia, aphasia, general convulsions and coma. Hence, in the case of a gradually increasing lesion the earlier symptoms are the ones available for localization. It will be noticed that while the important symptoms in one case are the late ones and in the other the early ones the time for localization in both cases is when the symptoms are most limited in extent.

The problem of localization may be very much confused by the existence of two or more lesions. The only way to solve it is to be thoroughly familiar with the effect of each. Multiple lesions are mostly syphilitic or tubercular.

THE SIGNS OF HYSTERIA.

Hysteria is a diseased condition in which perverted ideas and emotions cause the bodily symptoms. In its milder and more familiar forms its mental origin and true nature are generally obvious, but it has a great variety of rarer and more severe manifestations, which, on account of their close superficial resemblance to other diseases, often lead to the most serious errors of diagnosis. Any part of the body, whatever, may be affected in severe hysteria, hence, although it is a purely nervous disease, every practitioner of medicine or surgery must deal with it, and failure to recognize it often leads to treatment that is unnecessary and even disastrous.

A trustworthy diagnosis of hysteria must depend first on the absence of the symptoms that would prove the existence of any organic or other functional disease. The signs of organic disease in general have already been discussed; the special form which is most likely to be mistaken for hysteria is disseminated sclerosis. The functional disorders which it is most important to exclude are epilepsy, migraine, chorea and various effects of uremia. The positive indications of hysteria are of the most varied character and may appear, often quite unexpectedly, at any point in the history or physical examination.

The family history is often doubly significant, for the example of an hysterical mother or sister may greatly increase whatever predisposition is inherited. The history of the patient's past illnesses may give strong evidence of the abnormal susceptibility to emotional disturbances and

to suggestion which is the primary cause of hysteria. It must be remembered, however, that every one is more or less susceptible to disturbing emotions and ideas; it is only morbid susceptibility that is to be taken into account.

The history of the patient's mental experiences immediately preceding the onset is of great importance. If the symptoms closely follow a strong emotion, or the observation of similar symptoms in others or anxious thought about disease, they are probably hysterical. This is only a probability, however, until other than mental causes have been excluded by a careful consideration of the age, previous illnesses and present condition; for the onset of an organic paraplegia may possibly coincide with the shock of bad news, the monoplegia or partial hemiplegia that follows an apparently trivial injury is sometimes due to meningeal hemorrhage or syphilitic thrombosis, a convulsion following excitement may be epileptic or toxic, the palpitation that follows a talk about heart-disease may be primarily due to a valvular lesion, the chorea that seems to be an imitation is sometimes genuine, and so on.

The character of the paroxysms of which the patient complains often affords conclusive evidence of hysteria. Fits of laughing or sobbing and globus (the feeling of something rising into the throat like that which precedes sobbing) when they follow trivial causes, are proof of hysteria. Convulsions in which consciousness is partially retained and words are uttered, or in which the movements and attitudes express purpose and emotion and friends vainly try to hold the struggling patient, are always hysterical. Retraction of the head and arching of the body in a convulsion whose nature is doubtful, make hysteria probable, but meningitis, tetanus, strychnia poisoning and hydrophobia must be excluded. The occurrence of a paroxysm whenever a certain definite area (hysterogenic zone) is ir-

ritated, is evidence of hysteria, even when the paroxysm in itself is of doubtful character. States of trance, catalepsy and hypnotism are forms of hysteria. Alternating states of consciousness (double consciousness) are generally hysterical, sometimes epileptic. Spasm causing slow, rhythmical oscillation of any part of the body is hysterical, providing that organic disease has been excluded.

The state of the reflexes is of minor importance. The knee-jerk and heel-jerk are generally considerably exaggerated in hysteria while the plantar reflex is often diminished or lost. As this contrast is very rare in other conditions it affords probable evidence of hysteria. A nervous start when the knee-jerk is tested, especially if accompanied by a complaint of peculiar sensations, is also significant.

The hysterical character of a paralysis is recognized by the absence of organic disease and the presence of other symptoms of hysteria, rather than by anything peculiar in the motor loss itself. With but rare exceptions (chorea, migraine, occupation neurosis and toxic conditions) any paralysis that is not organic must be regarded as hysterical and symptoms characteristic of hysteria will generally be present. Nevertheless, there are certain forms of paralysis which are in themselves peculiar to hysteria.

In a case of partial hemiplegia, if the paralyzed leg is dragged after the sound one, instead of being swung past it, and the foot is held stiffly at right angles to the leg, instead of showing a tendency to drop of its own weight, the paralysis is hysterical. Inability to flex or extend a joint on request, although the same thing can be done automatically when attention is distracted, is proof of hysteria. Restraint, by contraction of the opponents, of a movement which the patient has been urged to make

and has actually begun is also hysterical. Paralysis of the adductors of the vocal cords, as indicated by aphonia, is always hysterical when coughing and sneezing are normal.

Among the most characteristic stigmata of hysteria are the sensory disturbances. All of these, whether sensory loss, paresthesia, hyperesthesia or pain, are alike in that their location never corresponds definitely to the areas of nerves or spinal segments, but is in areas of a quite different shape or in isolated spots. Sensory loss is the most important, although it is probably not so frequent in other countries as in France. It occurs in one of the following forms:

1. Hemianesthesia, or loss of sensibility, generally including all kinds, in one lateral half of the entire body. It is often accompanied by impairment of the special senses on the same side, the impairment of sight not being hemianopia but amblyopia with contraction of the visual field of the affected eye and perhaps a reversal of the relative size of the color fields. The left side is affected about three times as often as the right.

2. Anesthesia in so-called geometric areas, as in the form of a glove, stocking or sleeve, or in a circle, ellipse or triangle. Such a loss usually includes all kinds of sensibility and is especially apt to be found over a paralyzed or contractured part.

3. In scattered islets of variable shape.

Any of these forms, if the loss is great and well defined, may be confidently regarded as hysterical, even without the variability under suggestion or emotional change which may usually be observed. It is true that on theoretical grounds we might expect organic disease of the cortex to cause sensory loss in similar areas, for the hysterical anesthesia is probably caused by the inhibition of cor-

responding cortical centers, but, as a matter of fact, the sensory loss of cortical organic disease is slight and ill defined compared with that of hysteria. Hemianesthesia may also be caused by organic disease of the internal capsule, but in that case the visual defect, if any be present, is hemianopia and other signs of organic change are generally unmistakable.

Hyperesthesia or tenderness in any of the geometric areas just mentioned is almost as significant of hysteria as sensory loss.

The pain known as clavus, which is sharply localized in a small area near the vertex, is almost invariably hysterical, but as a general rule hysterical pains are not to be distinguished by their character or location. It is rather the circumstances under which they appear and disappear, the absence of certain conditions which accompany ordinary pains and the presence of other signs of hysteria that are significant.

If a patient complains of severe and long-continued pain, especially if it is said to prevent sleep, and yet there is no loss of weight or disturbance of pulse and the facial expression is not indicative of suffering, the pain is probably hysterical.

When a patient complains of intense pain awakened by the lightest touch or slightest change of posture and yet makes no complaint when considerable pressure is indirectly applied to the same part, the hysterical character of the pain is certain. For example, in the hysterical imitation of hip disease the slightest visible disturbance of the joint may appear to cause great agony, but the foot may be pushed upward so as to press the head of the femur firmly into the cotyloid cavity without causing any sign of pain; and in the hysterical imitation of Pott's disease there is the same superficial tenderness yet the patient may experience

a sudden jar of the spine or a downward pull on the shoulders without wincing. In many cases of this kind simply calming the fears and diverting the attention of the patient to other things will cause all signs of pain to disappear.

A visual defect may sometimes be the first convincing sign of hysteria. Dimness of vision which may be corrected by appropriate suggestion accompanying the use of a plain glass, or two glasses which neutralize each other, must of course be hysterical. Many of the cases in which eye-strain and various nervous disturbances appear to be relieved by very weak glasses or by a slight change in the balance of the ocular muscles are of the same nature. Concentric contraction of the visual field of one eye with reversed relation of the color fields (red field greater than blue, or green greater than red) is generally regarded as proof of hysteria, but unless there are other signs of hysteria its significance is doubtful. Monocular diplopia or polyopia, in the absence of a gross error of refraction, is almost certainly hysterical. Intense photophobia in the absence of any inflammatory condition that could cause it is proof of hysteria.

THE DIAGNOSIS OF NEURASTHENIA.

Neurasthenia is a state of functional weakness of the nervous system generally, especially of the higher centers.

An uncomplicated case is recognized:

1. By the absence of symptoms characteristic of organic disease of the nervous system, heart, lungs or other organs, and of those characteristic of hysteria or any of the psychoses such as mania, melancholia or paranoia.

2. By a history of some of the causes of nervous exhaustion, particularly a neurotic inheritance, exhausting diseases, injuries, emotional strain, overwork, sexual excesses, toxic conditions and insufficient food.

3. By signs of irritable weakness of the higher cortical centers. Among the most important of these are (*a*) lack of zest for work, feeling of inability to concentrate thoughts or arrive at a decision, complaint of failing memory but with a good recollection of events, early onset of mental fatigue; (*b*) morbid sensations in the head, not amounting to actual pain but consisting of rather vague feelings of soreness, dizziness, weight, constriction, increased volume, vacancy, confusion, etc.; (*c*) feelings of soreness or distress in the spine, heart, stomach and other organs; (*d*) excessive sensitiveness to trifling annoyances; (*e*) irrational fears, consisting of a vague sense of impending evil or dread of bodily or mental disease or occurring in paroxysms analogous to the stage fright of normal individuals; these may be excited in a great variety of ways, *e. g.*, being in the presence of a crowd, being alone, in a closed room, in an open space, near a

building, in the dark, crossing a bridge, etc.; (*f*) inability to divert the mind from a certain thought or phrase, usually of an indifferent or unpleasant character, which keeps recurring like a too-familiar tune and may impel to fatiguing and perhaps dangerous actions, such as counting, touching the posts that may be passed, reading all the signs on the street, following an absurd train of reasoning, etc.; if the impulse is resisted a feeling of distress occurs and often compels the patient to resume the tiresome performance; these morbid thoughts and impulses are not so common as the other symptoms of neurasthenia, are more difficult to cure and in rare cases pass into actual insanity; (*g*) impaired vasomotor control, cardiac palpitation, nervous indigestion, sexual debility, increased tendon and muscle reflexes and tremor.

MIXED FORMS OF DISEASE.

In studying a disease we necessarily learn it first in its uncomplicated form, but in practice we often find a patient to besuffering from two or more diseases at the same time. Thus organic disease of the brain is often complicated by epilepsy or hysteria; hysteria and neurasthenia are often mingled, and either may be added to epilepsy or migraine; myelitis may complicate a cerebral disease, and so on almost indefinitely. Such complications are very likely to lead to an incomplete diagnosis, for it is natural to feel that the diagnosis is made as soon as one disease is recognized, and the mind has a strong tendency to ignore symptoms which do not harmonize with an opinion already formed.

The only way to avoid being misled by such cases is not only to know the symptoms, course and possible complications of each disease, but also to form the habit of investigating anomalous symptoms with especial care.

THE RECOGNITION OF SPECIAL DISEASES.

EXPLANATION OF TABLES.

The use of the following diagnostic tables is so simple that any explanation may seem superfluous. After a thorough examination of the patient a prominent symptom is selected, an objective one like paralysis, spasm or optic neuritis, if it be present, and the corresponding table is found. Then the general descriptions marked by Roman numerals are compared in regular order with the symptoms of the case, stopping at the one which agrees with them. Under this division will be found a number of coördinate subdivisions, each marked by a capital letter. These are to be compared with the case in regular order until the right one is found, and so on through the various subdivisions until the individual disease is reached. Whenever the general description does not harmonize with the symptoms of the case it is, of course, necessary to pass over all that may be included under it and go on to the next coördinate division, which is always marked by the next character of the same kind. It will sometimes occur that divisions in the table are made according to the presence or absence of symptoms which have been entirely overlooked in the examination; in such a case the only remedy is to complete the examination at the next opportunity.

The most critical point in the diagnosis will generally be the decision as to whether or not there is organic disease. Of the symptoms mentioned in the tables as indi-

cating it, some are absolutely conclusive, but others, taken singly, denote only a strong probability of its existence. The weight to be given to these symptoms has already been considered. It must, of course, happen in some cases that the existence or absence of organic disease cannot be determined with certainty. In such a case the physician may assume each possibility in turn and by comparing the resulting diagnoses come to a probable conclusion, or he may have to wait for further symptoms to develop.

Whenever feasible both the pathological and the localization diagnosis are given together, but where necessary a supplementary table on localization has been added.

HEMIPLEGIA.

Paralysis of the leg, arm and tongue on one side and of the lower part of the face on the same side or, very rarely, on the opposite side.

I. Occurs at birth. Onset often marked by convulsions. Epilepsy and mental defect common in survivors.

Cerebral Birth Palsy, usually due to meningeal hemorrhage.

II. Occurs in infancy, usually in the course of an infectious disease. Onset sudden or very rapid, in most cases marked by convulsions. Aphasia accompanies right hemiplegia if the child has learned to talk. Epilepsy and mental defect common in survivors.

Infantile Cerebral Palsy, usually due to thrombosis in cortical veins.

III. Occurs after infancy.

A. Onset sudden or very rapid (in a few seconds to a few hours), generally marked by loss or disturbance of consciousness, with or without convulsions. Aphasia accompanies right hemiplegia but is often transitory.

1. The attack is preceded by evidences of uremia, particularly a great diminution in the amount of the total urinary solids, albuminuria and casts and a characteristic pallor, edema or retinitis. The paralysis quickly disappears if free secretion of urine can be secured. Rare. *Uremic Paralysis.*

2. The attack is preceded by symptoms of chronic, organic, mental or nervous disease. Onset often marked by convulsions. Temperature rises rapidly to 101° or more. Paralysis generally passes away in a few days if the patient survives.

a. History of gradually increasing dementia and often of delusions of grandeur, indistinctness of speech, incoördination or weakness of limbs, etc.

Apoplectiform Attack in Paretic Dementia.

b. History of intention tremor, transient paralysis or amblyopia, nystagmus, scanning speech or other symptoms of disseminated sclerosis.

Apoplectiform Attack in Disseminated Sclerosis.

3. The attack is not preceded by symptoms of chronic, organic, mental or nervous disease. The paralysis is permanent or disappears slowly. Within a few days of the attack the tone of the affected muscles and their tendon reflexes are increased.

a. No prodromata. Endocarditis or pulmonary abscess present at time of onset. No marked initial fall of temperature. Age usually less than 40.

Cerebral Embolism.

b. Prodromata (nocturnal headache, transient weakness, tingling, mental dullness, etc.) common. Syphilis present or at least not excluded. No other cause of arterial degeneration. No cause of embolism. No evidence of increased blood pressure at time of onset nor marked initial fall of temperature. Age usually less than 40.

Syphilitic Thrombosis.

c. The onset follows an injury to the head, either immediately or after a short interval, and is very rapid rather than sudden. The paralysis is often preceded by convulsions on the same side and by severe headache on the opposite side, followed by sopor, coma and slowness of pulse.

Meningeal Hemorrhage.

d. Arteries weakened by atheroma or infectious disease. Evidence of increased blood pressure at time of onset, such as a tense pulse, cardiac hypertrophy, muscular exertion, excitement, etc. Tem-

perature usually falls one degree or more within a few hours. Age usually more than 40.

Cerebral Hemorrhage.

e. Evidence of lowered blood pressure at the time of onset together with arterial disease (especially the atheroma of advanced age), or with increased coagulability of the blood, as in the puerperium, phthisis, gout, cancer, etc. Fall of temperature slight if any. Coma generally not profound. Prodromata common; onset often rapid rather than sudden. *Cerebral Thrombosis.*

B. Onset gradual in a few hours or longer. Headache, often accompanied by vomiting, usually precedes any disturbance of consciousness.

1. Onset rapid in a few hours to a few days.

a. Attack follows an injury to the head without intracranial infection, or occurs in a patient who is alcoholic or chronically insane. The paralysis is often preceded by convulsions on the same side and by severe headache on the opposite side, followed by sopor, coma and slowness of pulse.

Meningeal Hemorrhage.

b. There is an infected wound of the head or caries of the skull, with correspondingly localized headache, on the side opposite the paralysis. Fever with gradual development of delirium, sopor and coma. *External Pachymeningitis.*

c. A source of intracranial infection or irritation is present, *e. g.*, purulent otitis media, tuberculosis, pneumonia, epidemic influence or sunstroke. Onset marked by headache and fever, often accompanied by vomiting. General hyperesthesia exists at first, but is soon followed by delirium which, in severe cases, merges into stupor and coma, headache persisting as long as the patient can answer questions. Retraction of the head, localized twitching and general convulsions are

common. Other paralyses may occur, especially in the ocular muscles. Optic neuritis, rarely intense, is common in the more protracted cases when the disease is at the base, rare when it is over the convexity.

Meningitis, possibly *Cerebritis* or *Abscess.*

2. Onset acute or chronic, in a few days to weeks or months. A source of purulent infection is present, *e. g.*, otitis media, empyema, abscess in any part of the body, infected wound of the head, etc. Temperature irregular, generally elevated, but sometimes depressed. Rigors followed by fever and sweating common. Optic neuritis frequent, but rarely intense. Focal cerebral symptoms (Fig. 14) common and cranial nerve symptoms uncommon in comparison with meningitis. Duration may be short or it may be very long with a period of latency. *Intracranial Abscess.*
3. Onset gradual and slow, in six weeks to six months or longer. No evidence of suppuration. Temperature mostly normal.
   a. Headache usually intense, often accompanied by vertigo and vomiting. Optic neuritis in four-fifths of all cases, often intense. Pulse often slow. Convulsions or any form of focal (Fig. 14) or cranial nerve symptom may occur. Course usually long and for the most part steadily progressive, with mental failure toward the end. Inherited or acquired predisposition to new growths sometimes apparent.

      Intracranial Tumor, *Aneurism or Cyst.*

   b. Headache chiefly nocturnal. Optic neuritis absent or slight. Syphilis present or at least not excluded. Focal or cranial nerve symptoms may occur as in tumor. *Syphilitic Meningitis.*
   c. Age usually more than 60. Headache often absent, rarely intense. No optic neuritis nor other cranial nerve symptom. Convulsions very rare.

Aphasia and dementia may gradually occur. Course slowly progressive up to a certain point, but then may be arrested for a time. Duration from onset to fatal ending varies from a few months to two years. Rare.

Chronic Progressive Cerebral Softening.

FIG. 15.

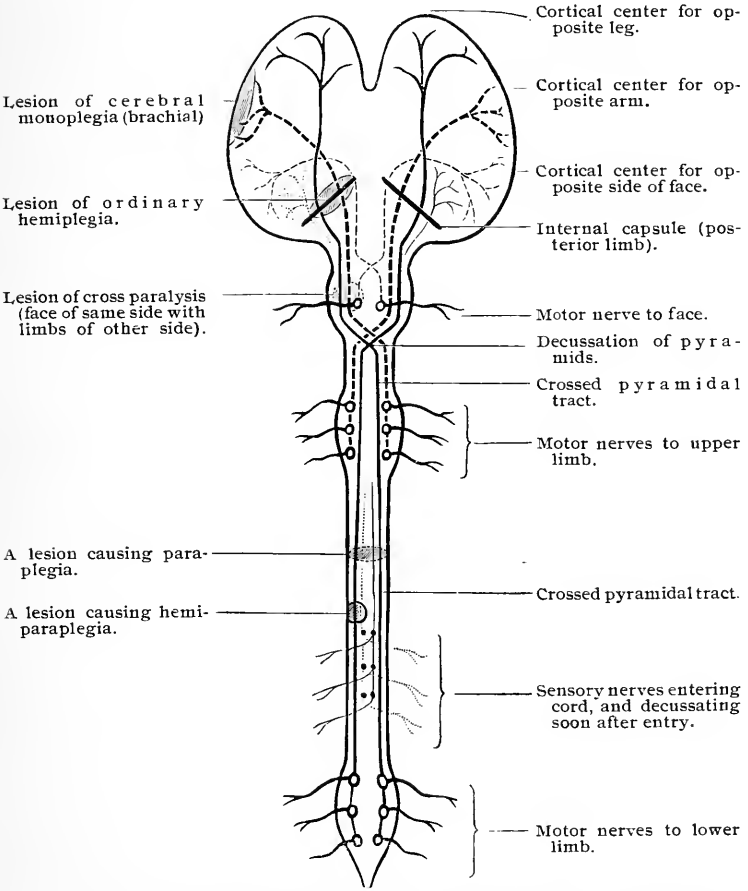


DIAGRAM TO SHOW THE GENERAL ARRANGEMENT OF THE MOTOR TRACT, AND THE EFFECT OF LESIONS AT VARIOUS POINTS. (*From Ormerod.*)

LOCALIZATION DIAGNOSIS IN HEMIPLEGIA.

I. The hemiplegia is preceded or accompanied by a symptom characteristic of involvement of the cortex, particularly Jacksonian epilepsy, epileptic attacks having a definitely localized sensory aura, a definite form of aphasia or an intellectual defect which cannot be regarded as a temporary or indirect result of the lesion. If the symptoms are gradually developed the paralysis begins in a single limb or one side of the face (monoplegia) and becomes a hemiplegia by extension to the adjacent parts. (Figs. 14 and 15.)

Lesion in or immediately beneath the Cortex, beginning in the part corresponding to the earliest disturbance of motion, sensation or speech.

II. The paralysis begins as a monoplegia and gradually extends to hemiplegia. There are no Jacksonian attacks or localized sensory auræ and no aphasia or intellectual loss except such general defect as may result from the pressure or irritation of a rapidly growing tumor or abscess. There may be slight sensory loss in the paralyzed limbs, but there is no definite hemianesthesia. *Lesion of the Centrum Ovale.*

III. Hemianesthesia, with or without hemianopia, accompanies the hemiplegia. There may be pains in the paralyzed limbs. *Lesion in or near the Internal Capsule.*

IV. There is mobile spasm (athetosis) or incoördination (post-hemiplegic chorea) of the paralyzed hand. Pains in the paralyzed limbs common. *Lesion of the Thalamus.*

V. Paralysis of all or a part of the muscles supplied by the third nerve or of the superior oblique occurs at the same time as the hemiplegia, but on the opposite side. May be accompanied by hemianopia, especially in cases of tumor.

Lesion of the Crus or uppermost part of the Pons.

VI. The paralysis of the limbs is accompanied by paralysis of the face, external rectus or muscles of mastication, or by anesthesia of the face, on the opposite side. Swallowing and articulation likely to be impaired. Trismus and general rigidity may occur. In acute lesions hyperpyrexia is common and death generally occurs in a very short time.

Lesion of the Pons.

VII. The symptoms are not characteristic of any definite location.

Lesion of the Internal Capsule to be assumed as the most probable.

PARTIAL HEMIPLEGIA—MONOPLEGIA.

Paralysis of arm and leg or of arm and lower part of face on one side, or paralysis of one limb or of lower part of one side of the face.

I. There is organic disease of the nervous system, shown by such signs as paralysis of ocular muscles, face, tongue or one side of larynx; paralysis and sensory loss both corresponding to the function of definite nerve trunks or spinal segments; loss of faradic irritability; absence of knee-jerk; typical ankle clonus; optic neuritis or optic atrophy; failure of light reaction; Jacksonian epilepsy, etc.

A. The paralyzed muscles are flabby and soon waste, their tendon reflexes being lost and faradic irritability lost or impaired. Control of bladder and rectum retained.

1. Onset acute, in a few hours to a few days, and resembles that of an eruptive fever, possibly marked by convulsions, often attended by rheumatoid pains. No sensory loss. Six-sevenths of all cases in children under ten years of age. *Poliomyelitis.*

2. Onset acute, subacute or, rarely, chronic, in one to six weeks or longer. Paralysis, together with numbness, tingling pain and more or less sensory loss, is in the distribution of certain nerves. Muscles and nerve trunks generally tender.

a. An arm affected. The apparent cause is gout, rheumatism, exposure, fracture of humerus, pressure of new growths or wounds of the arm or shoulder.

Neuritis involving brachial plexus, nerves or nerve roots.

b. A leg affected. The apparent cause is vertebral caries, pelvic inflammation, pressure of new growths, an infected wound of a nerve or an injury about the hip.

Neuritis involving lumbar and sacral plexuses.

B. No wasting nor loss of faradic irritability. Paralysis spastic; tendon reflexes exaggerated.

1. No cerebral or cranial nerve symptoms. Leg or leg and arm paralyzed. Loss or diminution of sensibility to pain, temperature and touch in an area corresponding to that of the paralysis but on the opposite side.

Unilateral Lesion of Spinal Cord.

2. Cerebral and cranial nerve symptoms common. Sensory loss, if any, is on the same side as the paralysis but is usually slight in comparison with it and does not correspond to the distribution of nerve trunks or spinal segments.

Diagnosis same as in Hemiplegia.

II. All positive signs of organic disease absent.

A. The paralysis occurs as a complication of chorea and is generally a monoplegia, sometimes a partial hemiplegia. No reaction of degeneration. There may be some sensory loss. Always passes away in the course of a few weeks. *Choreic Paralysis.*

B. Onset variable; if sudden it may suggest apoplexy, especially when it occurs in an hysterical fit; if gradual the paralysis may advance in steps from one joint to the next.

An emotional cause is often distinctly apparent. Susceptibility to suggestion is apparent in the history or at the examination. History of hysterical attacks common. Hysterogenic zones may perhaps be found. Symptoms often vary greatly with emotional changes.

When the patient is urged to attempt a movement the opposing muscles may often be seen to resist it. If attention be diverted acts requiring the use of the paralyzed

muscles may perhaps be performed automatically. Under encouragement, especially if electricity or other local treatment has been used, a far greater movement may be made than at first seemed possible. When the leg is involved and the patient walks, the toes do not, as in organic paralysis, keep to the ground from the mere weight of the foot, necessitating an exaggerated motion of the hip by which the lame foot is swung in advance of the sound one; on the contrary, the foot is often held in a fixed position and is usually dragged after the sound one instead of advancing before it.

Sensory loss is often more extensive and profound than the paralysis and its distribution is characteristic of hysteria, being most commonly in the form of hemianesthesia with corresponding impairment of the special senses, or in areas bounded by the external lines of the body or by circular lines around a limb, or in geometric areas, but never corresponding exactly to the distribution of particular nerves or spinal segments.

Hysterical Paralysis.

LOCALIZATION DIAGNOSIS IN PARTIAL HEMIPLEGIA AND MONOPLEGIA.

For most cases the seat of the lesion has already been sufficiently indicated. In cases of spastic partial hemiplegia or monoplegia of intracranial origin, localization depends on the same principles as in hemiplegia (which see), but the lesion is almost always in the cortex or centrum ovale, because there the motor tract is so spread out as to admit of the fibers for the face, arm or leg, being damaged separately; in the internal capsule, crus or pons, these fibers are so close together that a lesion of any considerable size must affect all of them and so cause a complete hemiplegia.

PARAPLEGIA—DOUBLE HEMIPLEGIA.

Both legs are paralyzed, with or without paralysis of the arms and face.

I. Organic disease of the nervous system is shown to be present by some positive sign, *e. g.*, paralysis and sensory loss corresponding definitely to the distribution of nerves or the function of spinal segments, angular deformity of spine, girdle sensation or other root symptoms, degenerative atrophy of muscles, loss of faradic irritability, absence of knee-jerk, typical ankle clonus, paralysis of face, tongue, ocular muscles or one side of larynx, optic neuritis or atrophy, absence of light reaction, etc.

A. There is no paralysis, spasm or sensory loss, except in parts innervated by the spinal cord and in such form as to be accounted for by disease of the cord itself.

1. There is more or less sensory loss in addition to the paralysis, the upper limit of both corresponding to a segment of the spinal cord, generally combined with disturbed action of the bladder and rectum. Not preceded by localized spinal pain and rigidity.

a. Onset sudden in a few moments.

i. Simultaneous with severe injury to spinal column.

Fracture or Dislocation of Vertebræ, Wound of or Hemorrhage into Cord.

ii. Without external violence.

Hemorrhage into Cord.

b. Onset gradual, acute or chronic. Vertebræ not diseased. Pain rarely a prominent symptom, although some dull pain is usually present.

Myelitis.

2. Paralysis preceded by localized spinal pain and rigidity; corresponding radiating pains common. Paralysis of legs spastic with exaggeration of knee- and Achilles-jerks, unless the disease be low enough to involve the lumbar enlargement, in which case wasting and loss of tendon reflexes will be found. Sensory loss and disturbance of bladder and rectum may or may not be added to the other symptoms.
   a. Deformity or swelling, often with deep-seated tenderness, indicates disease of the vertebræ.
      i. Patient most commonly a child, sometimes an adult, rarely an elderly person. The tubercular diathesis is almost always manifest, but very rarely syphilis may be the cause. The pain, generally of moderate severity, is increased by motion or jars and diminished by rest of spine. Prominence or lateral displacement of one or more spinous processes the characteristic deformity.
      Spinal Caries.
      ii. Patient generally in second half of life. There may be a history of tumor elsewhere or of predisposition to new growths or to aneurism. Pain very intense and greatly aggravated by motion.
         § Radiating pain on left side of thorax, mainly in the areas of the fifth and sixth dorsal segments. Thrill and murmur at seat of deformity.
         Aneurism eroding Spine.
         §§ Pain on both sides. No thrill or murmur. *Tumor of Spine.*
   b. Nothing to indicate disease of vertebræ.
      i. Onset sudden or rapid after passing from an air pressure of three atmospheres or more to the ordinary pressure. Headache, giddiness, abdominal pains and vomiting common.
      Caisson Disease.

ii. Onset sudden. No fever at first.
Spinal Meningeal Hemorrhage.

iii. Onset gradual but rapid, marked by chill and fever. *Acute Spinal Meningitis.*

iv. Onset gradual and slow.

§ History of alcoholism, syphilis or exposure to cold.
Chronic Spinal Meningitis.

§§ Evidence of predisposition to new growths. No other cause of meningitis.
Intraspinal Tumor.

B. Symptoms in parts innervated by the spinal cord greatly predominate over such cerebral or cranial nerve symptoms as may occur and often exist alone. No localized spinal pain, rigidity or radiating pains.

1. Paralysis always flabby with loss of tendon reflexes. Onset acute or subacute.

a. Onset in a few hours to a few days and resembles that of an eruptive fever; often marked by vomiting, sometimes by convulsions; attended by rheumatoid pains. Paralysis is usually unequal on the two sides and is rapidly followed by wasting and loss of faradic irritability. Anterior tibial group and peronei generally most affected. No disturbance of bladder or rectum. No sensory loss. Six-sevenths of all cases occur in children under ten years of age. *Poliomyelitis.*

b. Onset acute. Paralysis begins in legs and ascends rapidly through trunk to arms. No wasting or change in electrical reactions. No severe pain. There may be slight and ill-defined sensory loss and perhaps disturbance of bladder and rectum. Fever generally absent. Most cases occur in early adult life during convalescence from some acute infectious disease or after exposure to cold.
Acute Ascending Paralysis.

c. Onset acute or subacute, in one to four weeks or

more. Paralysis, numb, stinging pain and more or less sensory loss are in the distribution of peripheral nerve trunks, especially the external popliteal and musculo-spiral. Muscles and nerves tender. Bladder and rectum very rarely involved. Usually a history of alcoholism, metallic poisoning, diphtheria, septicemia, extraordinary exertion or exposure to cold. *Multiple Neuritis.*

2. Paralysis spastic with exaggeration of tendon reflexes. Onset of disease always slow, although in disseminated sclerosis paralysis and other symptoms may come on rapidly. Bladder and rectum not affected until disease is far advanced.
   a. Paralysis is accompanied by intention tremor, nystagmus, scanning speech or other signs of scattered lesions. *Disseminated Sclerosis.*
   b. No evidence of disseminated sclerosis. No sensory loss.
      i. The spastic paraplegia is accompanied by degenerative muscular atrophy and fibrillary twitching in the upper part of the body, generally beginning in the hand, shoulder or back. *Amyotrophic Lateral Sclerosis.*
      ii. Paraplegia accompanied by ataxia. *Postero-lateral Sclerosis.*
      iii. Paraplegia without muscular atrophy or ataxia. *Lateral Sclerosis.*

C. Cerebral symptoms predominate over any signs of spinal disease that may accompany them.
   1. Onset acute, marked by intense headache and fever. Delirium occurs early and is often followed by sopor and coma. Retraction of head occurs in almost all cases. Spinal rigidity and pain, both localized and radiating, together with cranial nerve symptoms, common. An eruption of herpes, purpura, urticaria or erythema occurs in most cases. No apparent cause except epidemic influence. Whole course

from onset to fatal ending or established convalescence varies from a few hours to a few months.

Cerebro-spinal Meningitis.

2. Onset of disease chronic. The paraplegia is either spastic or ataxic and may be of slow or rapid onset. Progressive mental failure (often with delusions of grandeur), inequality of pupils, stumbling speech and slight facial twitching are the most characteristic symptoms. *Paretic Dementia.*

3. Onset usually sudden or very rapid, occurring in most cases at birth or in infancy, but it may be slow and occur at any age. No conclusive sign of spinal disease but cerebral symptoms (disturbance of consciousness, mental defect, aphasia, convulsions, etc.) and cranial nerve symptoms are common. Paralysis spastic with exaggeration of tendon reflexes except for a short time immediately after a sudden onset. Control of bladder and rectum sometimes impaired and involuntary evacuations may also occur through inattention.

Bilateral Lesion of Hemispheres or, possibly, a single one of Pons, kind of lesion determined as in Hemiplegia, q. v.

II. All positive signs of organic disease absent, although an atypical ankle clonus may be found, especially in cases of long standing with contracture of the calf muscles.

A. Onset variable. An emotional cause is often apparent. Susceptibility to suggestion is generally manifest in the history or at the examination. History of hysterical attacks common; hysterogenic zones may perhaps be found. Symptoms often vary greatly with emotional changes. When the patient is urged to attempt a movement the opposing muscles may often be seen to resist it. If attention be diverted movements of the paralyzed limbs sometimes occur automatically and under encouragement, especially if electricity or other local treatment has been used, a far greater movement may be made

FIG. 16.

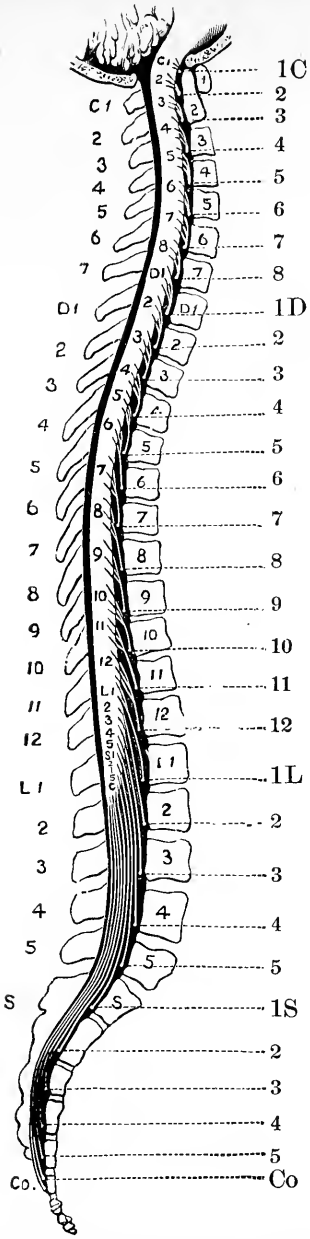


DIAGRAM FROM AN ORIGINAL INVESTIGATION BY W. R. GOWERS, SHOWING RELATION OF VERTEBRAL SPINES TO THEIR BODIES AND TO THE NERVE ROOTS. (*From Tyson.*)

The ends of the vertebral spines are opposite the middle of their own bodies only in the lumbar region. They correspond to the lower edge of their own bodies in the cervical and last two dorsal, and to the upper part of the body below in the rest of the dorsal region.

than at first seemed possible. Sensory loss is common, never corresponding to the distribution of particular nerves or spinal segments, but in areas bounded by the external lines of the body or by circular lines around a limb, or in geometric areas. *Hysterical Paraplegia.*

B. Paraplegia does not appear when the patient is lying down but only on attempting to walk or stand or, rarely, while sitting. The affection is usually hysterical but not always certainly so. *Astasia Abasia.*

LOCALIZATION DIAGNOSIS IN PARAPLEGIA AND DOUBLE HEMIPLEGIA.

Spinal Localization.—The spinal cord is hypothetically divided into segments, each one being numbered like the pair of nerves connected with it; thus we have eight cervical, twelve dorsal (or thoracic), five lumbar and five sacral segments. Each of these segments is higher than its corresponding vertebra; the relation of segments, nerve roots and vertebræ to each other is shown in the accompanying diagram from Gowers.

The upper limit of a coarse spinal lesion may be determined in three ways:

1. By comparing the upper limit of the sensory loss with the sensory areas supplied by the several spinal segments as given in Figs. 28 and 29; sometimes the limit of anesthesia is made especially distinct by a zone of hyperesthesia immediately above it.

2. By comparing the upward limit of the paralysis and loss of faradic irritability with the table on the next page showing the relation of muscles to spinal segments.

3. By comparing the loss of reflexes with the part of the same table showing the relation of reflexes to spinal segments.

In determining the lower limit of the lesion it is obvious that sensory loss and paralysis cannot be utilized but the good condition of segments below the lesion will usually be indicated by the absence of atrophy, the presence of faradic irritability and the reflex response of the corresponding muscles. For example, in a case of gunshot

Table Modified from Gowers, Showing the Approximate Relation of Spinal Segments to Muscles and Reflexes.

Muscles.	Segments.	Muscles.	Segments.	Reflexes.
	C 1	Small rotators of head.	C 1	
Sterno mastoid.	2	Depressors of hyoid.	2	
Upper neck muscles.	3	Lev. ang. scapulæ.	3	
	4		4	
Upper part of Trapezius.	5	Diaphragm.	5	
		Serratus. Flexors of elbow. Supinators. (Shoulder Muscles.)		Biceps-jerk.
Lower neck muscles.	6	Extensors of wrist and fingers.	6	Triceps-jerk. Wrist-j. (ext.) Wrist-j. (flex.) (Scapular.)
	7	Ext. elbow. Flex. wrist and fingers.	7	
Middle part of Trapezius.	8	Pronators.	8	
	D 1	Intrinsic muscles of hand.	D 1	Palmar.
	2		2	
	3		3	
	4		4	
	5		5	Epigastric.
Lower part of Trapezius and Dorsal muscles.	6	Intercostals.	6	
	7		7	
	8		8	
	9		9	
	10	Abdominal muscles.	10	Abdominal.
	11		11	
	12		12	
	L 1		L 1	
	2	Cremaster.	2	Cremasteric.
		Flexors of hip.		
	3	Extensors of knee. Adductors of hip.	3	Knee-jerk.
Lumbar muscles.	4	Ext. and abductors of hip.	4	
Peronei. Flex. of ankle.	5	Flexors of knee.	5	Gluteal. Heel-jerk.
Ext. of ankle.	S 1	Intrinsic muscles of foot.	S 1	Plantar.
	2		2	
	3	Perineal and anal muscles.	3	Perineal, anal and vesical.
	4		4	
	5		5	
	Co.		Co.	

wound of the cord there was sensory loss up to the line between the first and second lumbar areas; there was paralysis of all joints of both lower limbs with corresponding loss of faradic irritability and reflexes. This indicated an extensive lesion in the lumbar enlargement and that the second lumbar was the uppermost segment involved. But the anal and perineal reflexes were lively and the faradic irritability of the perineal muscles was retained; there was complete retention of urine until a large quantity had accumulated and then there was a purely reflex evacuation. This indicated that the lower sacral segments were not involved. Consulting Fig. 16 it will be seen that the damaged portion of the cord, extending from the second lumbar to the second sacral segment inclusive, was contained mainly by the twelfth dorsal vertebra, not extending as high as the upper border of the eleventh or as low as the lower border of the first lumbar. An operation was performed and the bullet found within the spinal canal just above the lower border of the eleventh dorsal vertebra.

Cerebral Localization.—The cerebral lesions that cause double hemiplegia or paraplegia act in precisely the same way as those which cause hemiplegia, the only difference being that both sides of the brain are affected instead of one, and they are localized by applying the same principles as in hemiplegia.

The history of the onset must be carefully studied. If paralysis has occurred in two distinct attacks, first on one side and then on the other, the symptoms attributable to each must be considered separately, for in such a case there are two lesions, having no connection except that of a common cause, such as syphilis or arteriosclerosis.

Assuming that a single lesion has caused paralysis on both sides, the localizing symptoms may be arranged as follows:

LOCALIZATION DIAGNOSIS IN DOUBLE HEMIPLEGIA OR PARAPLEGIA OF INTRACRANIAL ORIGIN.

I. Spastic paralysis of both legs, perhaps also involving the arms but to a lesser degree, has its onset at birth or in infancy. Epilepsy, delayed acquisition of speech or intellectual defects almost invariably present.

Cerebral Birth Palsy or Infantile Cerebral Palsy, due to bilateral meningeal hemorrhage or cortical thrombosis.

II. Spastic paralysis of both legs occurs along with symptoms indicating an intracranial tumor.

Growth near the Vertex, pressing on both paracentral lobules.

III. There is double hemiplegia along with paralysis of the third or fourth nerve on either side. Hemianopia may be present. Optic neuritis and other signs of a tumor likely to be present. *Lesion of both Crura.*

IV. Double hemiplegia is accompanied by paralysis of the seventh, sixth or motor branch of the fifth nerve, on either side, or by anesthesia of the face. Swallowing and articulation generally impaired. Hyperpyrexia and a rapidly fatal course common in acute lesions. *Bilateral lesion of Pons.*

V. Paralysis on both sides of the body (perhaps irregularly distributed) is accompanied by paralysis of the lips, tongue, palate, pharynx and larynx. Acute lesions rapidly fatal. (See Bulbar Paralysis.) *Lesion of the Medulla.*

PARALYSIS OF OCULAR MUSCLES.

I. The paralysis is not limited to the associated movements of both eyes which can be made voluntarily by normal individuals, such as convergence or conjugate movement to the right or left, but when the eyes are tested separately a motion which is not separately under the control of the will is found to be lost on one side independently of the action of the other. Paralysis of the levator is revealed by ptosis; of the external muscles of the eye-ball by limitation of movement in the direction of the affected muscle, secondary deviation of the sound eye and diplopia; of the internal ocular muscles by the absence of accommodation or of pupillary action. Spasm simulating ptosis, spasmodic strabismus or spasm of the muscle of accommodation must not be mistaken for paralysis.

A. The paralysis is caused by hemorrhage, inflammation or new growth within the orbit.

B. The paralysis is not caused by disease within the orbit.

1. The paralysis is congenital, often associated with malformation of various kinds but not with signs of active disease.

Error of Development of nuclei, nerve trunks or muscles.

2. The paralysis is congenital or occurs in early infancy, associated with globular enlargement of the cranium. Convulsions and other paralyses common.

Hydrocephalus.

3. The paralysis occurs at birth or afterward.

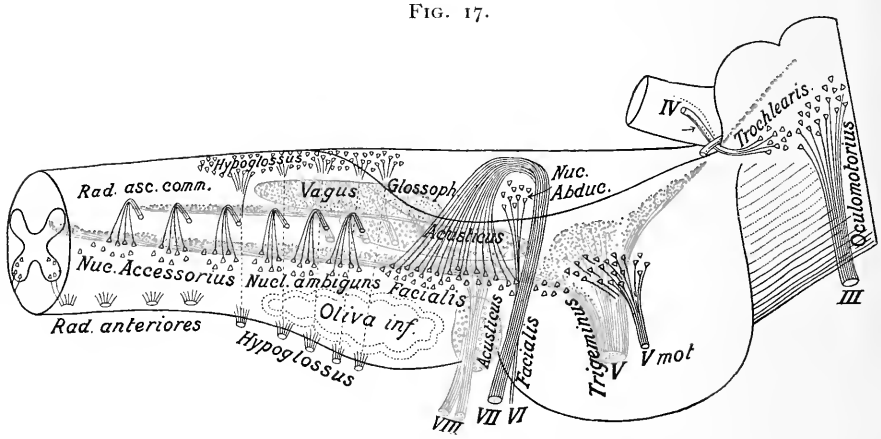
a. The combination of other symptoms, especially hemiplegia, headache, dizziness, vomiting or mental impairment, with the ocular paralysis indicates the existence of coarse organic disease within the cranium.

i. The onset of symptoms is sudden and

§ Occurs at birth. Hemiplegia, diplegia or monoplegia accompanies the ocular paralysis. Epilepsy and mental defect common in survivors.

Cerebral Birth Palsy, generally due to meningeal Hemorrhage.

FIG. 17.



SITUATION OF THE CRANIAL NERVES. (*From Tyson, after Edinger.*)

Cranial nerve nuclei, oblongata, and pons represented as transparent. Motor nuclei, black; sensitive nuclei red.

§§ Immediately follows a blow on the head.

Damage to nerve trunks or nuclei by Concussion, Hemorrhage or Fracture.

§§§ Is not related to injury of the head.

Hemorrhage or Thrombosis in the region of the nuclei or, very rarely, hemorrhage into nerve sheath.

ii. Onset of cerebral symptoms acute in a few hours to a week.

§ A source of intracranial infection or irritation is present, *e. g.*, purulent otitis media, tuberculosis, pneumonia, epidemic influence or insolation. Onset marked by headache and fever, often accompanied by vomiting. General hyperesthesia exists at first, but is soon followed by delirium which merges into stupor and coma, headache persisting as long as the patient can answer questions. Rigidity of the neck with retraction of the head, localized twitching and general convulsions are common. Other paralyses may occur. Optic neuritis, rarely intense, is common when the disease is at the base, rare when it is over the convexity.

Meningitis, possibly cerebritis or abscess.

§§ The eye-lids and temple on the affected side are edematous and distended by venous blood and the eye-ball is prominent. The patient is depressed by an exhausting disease or there is a source of infection. Temperature normal or only slightly raised, unless meningitis also exists.

Thrombosis of Cavernous Sinus.

§§§ Ocular paralysis preceded or followed by atrophic paralysis of limbs or of lips, tongue and throat. No sensory loss. Onset like that of an eruptive fever.

Polio-encephalitis, acute inflammation of nuclei analogous to poliomyelitis.

§§§§ Onset of cerebral symptoms rapid in a person who gives a history or the

signs of chronic alcoholism. Paralysis of the limbs accompanied by tenderness of muscles and nerves and sensory loss (alcoholic neuritis), often by characteristic delirium, usually, but not always precedes the ocular paralysis.

Alcoholic Polio-encephalitis.

iii. Onset acute, subacute or chronic. A source of purulent infection present, *e. g.*, suppurative otitis media, infected wound of the head, empyema or abscess, in any part of the body. Temperature irregular, usually elevated, but sometimes depressed. Rigors followed by fever and sweating common. Optic neuritis frequent, but rarely intense. Duration may be short or very long with a period of latency.

Intracranial Abscess.

iv. Onset chronic.

§ No source of purulent infection. Personal or family predisposition to new growths may be apparent. Temperature normal or only slightly disturbed. Headache generally intense, often accompanied by giddiness and vomiting. Optic neuritis in four-fifths of all cases, usually intense. Pulse often slow. Course long and mostly progressive. Toward the end mental failure tending toward stupor and coma.

Intracranial Tumor, including aneurism and hydatid cyst.

§§ Patient alcoholic or syphilitic. Optic neuritis absent or slight. No fever or slowness of pulse. There may be spasm or paralysis in the domain of other cranial nerves, rarely in the limbs. *Chronic Meningitis.*

b. Proof of the existence of coarse organic intracranial disease is lacking, but there is evidence of an intoxication or infection.

i. The paralysis is limited to the muscles concerned in light reaction and accommodation. The patient, intentionally or by accident, is under the influence of atropia or a drug of similar action.

ii. Syphilis is active, as shown by the history or the presence of characteristic symptoms.

Syphilitic Inflammation of nerves or nuclei or pressure upon them by a gumma.

iii. The ocular paralysis follows diphtheria or, rarely, comes on during its course. Commonly limited to loss of accommodation together with paralysis of the soft palate but any or all of the external ocular muscles may be affected and the muscles of the limbs may show a wasting paralysis with loss of tendon reflexes. In severe cases there is a corresponding sensory loss. *Diphtheritic Neuritis.*

iv. The ocular paralysis occurs as a symptom of influenza, pneumonia, scarlatina, measles or typhoid fever or of poisoning by metals, ptomaines or gases, without other signs of meningitis.

Probably Inflammation of Nuclei, possibly Neuritis.

§ History of exposure to lead. Dark line at junction of gums and teeth. Lead often present in the urine. Characteristic colic and other paralyses, especially of the extensors of wrists and fingers, almost always precede ocular paralysis.

Plumbic Neuritis or degeneration of nuclei.

v. Sugar in the urine, thirst, excessive appetite and general weakness indicate the presence of diabetes. *Diabetic Neuritis.*

c. There are signs of degenerative disease of the

central nervous system. Individual muscles supplied by the third nerve often paralyzed alone, indicating disease of the nuclei. The ocular palsy is often transient but may be permanent and sometimes progresses to complete paralysis of all the external muscles of both eyes, accommodation and contraction of the pupil usually being preserved. Optic atrophy and the Argyll-Robertson pupil are common. The ocular paralysis may precede all other symptoms.

i. The knee-jerks are absent. A history of lightning pains in the legs is common, also of loss of virility and slight difficulty in voiding or retaining the urine. The patient sways on standing with eyes closed and in the later stages the gait is ataxic. Argyll-Robertson pupil in most cases. *Tabes.*

ii. Failure of judgment and memory, often combined with monstrous and unsystematized delusions of grandeur, accompanies signs of organic disease, among which inequality of pupils and stumbling speech are common. *Paretic Dementia.*

iii. Intention tremor with nystagmus or scanning speech is associated with various signs of scattered lesions, such as isolated paralyses, contraction of the visual fields with impairment of color sense, loss of smell, nervous deafness, etc. *Disseminated Sclerosis.*

iv. Ocular paralysis associated with spastic paraplegia of gradual onset accompanied by ataxia. No sensory loss. *Postero-lateral Sclerosis.*

v. Ocular paralysis associated with wasting paralysis and fine fibrillary twitching in other muscles.

§ Wasting paralysis begins in muscles of hand, shoulder or upper arm.

! Spastic weakness of legs. Tendon reflexes of paralyzed muscles exaggerated.

Amyotrophic Lateral Sclerosis.

!! No weakness of legs until latest stages. Tendon reflexes of paralyzed muscles abolished.

Spinal Muscular Atrophy.

§§ Wasting paralysis begins in muscles of lips, tongue, pharynx and larynx, interfering with speech and swallowing.

Bulbar Paralysis.

vi. Ocular paralysis associated with loss of pain and temperature sense in areas where touch is retained, together with paralysis and trophic disturbances of variable and irregular distribution. *Syringomyelia.*

d. There is no evidence of coarse organic disease, infection or organic degenerative disease of the central nervous system. The ocular paralysis occurs periodically, usually associated with migraine or severe neuralgia, at first completely disappearing after each attack but tending to become permanent.

Periodic Ocular Paralysis. (*Migraine Ophthalmoplégique.*)

e. The ocular paralysis is a complication of a general nervous condition in which rapid heart action, enlargement of the thyroid gland and exophthalmos are prominent symptoms.

Exophthalmic Goitre.

f. The ocular paralysis exists alone or together with facial paralysis.

i. Muscles of one eye affected after exposure of that side of the head to a draft.

"Rheumatic" Ocular Paralysis, due to neuritis and analogous to the ordinary form of facial palsy.

ii. No cause can be found.

Diagnosis necessarily uncertain but in most cases a symptom of Syphilis or the first symptom of a degeneration such as Tabes or Paretic Dementia.

II. The movement lost is one of the associated movements of both eyes normally under control of the will, viz., convergence, divergence or motion to either side or upward or downward. When either eye is tested alone all the muscles may act. To be distinguished from spasm of the muscles opposing those appearing to be paralyzed, although an error in this respect might not affect the pathological diagnosis.

A. The combination of other symptoms with the ocular paralysis, especially of hemiplegia, headache, dizziness, vomiting or mental impairment, indicates the existence of coarse organic disease within the cranium. Ocular paralysis generally transitory.

Diagnosis to be made as in Hemiplegia or as in I, B of this table, q. v.

B. Proof of the existence of coarse organic intracranial disease is lacking but there is evidence of an intoxication (alcohol, opium, chloral, lead, diabetes, ptomaines) or an infection (syphilis, diphtheria, influenza, pneumonia, scarlatina, typhoid fever).

Inflammation or functional impairment of cortical centers, subcortical tracts or nuclei.

C. There are signs of degenerative disease of the central system. Optic atrophy and the Argyll-Robertson pupil are common.

Diagnosis to be made as in Optic Atrophy or as in I, B, 3, c of this table.

D. Signs of organic disease, intoxication or infection absent.

1. There is weakness and lack of balance of the ocular muscles but no actual paralysis. Errors of refraction are commonly associated. The patient's nervous energy has been exhausted. Disagreeable sensations in the head and along the spine which shift their

location within a very short time, irritability and morbid fears are common. *Neurasthenia.*

2. Ocular paralysis occurs and may vary or disappear under the influence of emotion or direct or indirect suggestion. History and other symptoms indicate the presence of hysteria, while no other cause can be found. Rare. *Hysteria.*
3. The ocular paralysis is a complication of a general nervous disorder in which rapid heart action, enlargement of the thyroid gland and exophthalmos are prominent symptoms. *Exophthalmic Goitre.*
4. The ocular paralysis exists alone and no cause can be found.

Diagnosis necessarily uncertain but in most cases a symptom of Syphilis or the first symptom of a degeneration, such as Tabes or Paretic Dementia.

LOCALIZATION DIAGNOSIS IN PARALYSIS OF OCULAR MUSCLES.

The nature of the lesion causing ocular paralysis affords some indication of its situation. Meningitis and syphilis are especially likely to damage the surface of the brain and the nerve trunks at the base; they rarely attack the nuclei. Degenerative diseases, such as tabes, spinal muscular atrophy, bulbar paralysis, and the acute inflammation of alcoholism or poliomyelitis attack the nuclei, usually on both sides. Paretic dementia may cause paralysis of conjugate movement (inability of both eyes to look in a certain direction) by damage to the cortex, or paralysis of definite muscles by damage to the nuclei. More precise indications are given by the distribution of the paralysis, as shown in the following table:

I. The paralysis is limited to the muscles of one eye. Swelling and venous congestion of the lids and protrusion of the eye-ball appear along with the paralysis or soon after it. Optic neuritis or atrophy may occur. No signs of disease within the cranium. *Lesion within the Orbit.*

II. All the muscles supplied by the third nerve are paralyzed or weakened at the same time. Other cranial nerves may be involved. The pathological condition is hemorrhage, meningitis, syphilis or tumor.

Lesion at the Base of the Brain, damaging the nerve trunk.

A. The paralysis of the third nerve is accompanied by hemiplegia on the opposite side, with or without corresponding hemianopia. *Lesion of the Crus.*

III. The paralysis is bilateral and may be limited to only a part of the muscles supplied by the third and fourth nerves, the others retaining their function or becoming paralyzed at a considerably later time. The pathological condition is usually a vascular lesion, an intoxication, acute inflammation or a degeneration. *Lesion of the Nuclei.*

IV. The external rectus of one eye is paralyzed, the motions of the other eye being normal. The pathological condition is hemorrhage, meningitis, syphilis or tumor.

Lesion at the Base of the Brain, damaging the nerve trunk.

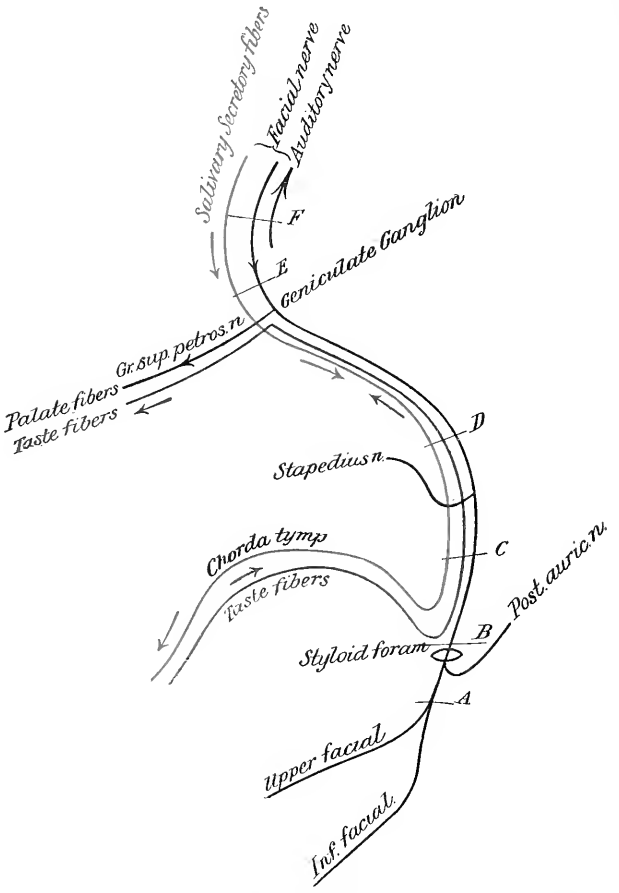
V. The external rectus of one eye is paralyzed and the other eye cannot voluntarily be turned inward. Facial paralysis may occur at the same time, or paralysis of the muscles of mastication, or facial anesthesia. Nystagmus is common. Swallowing and articulation are likely to be impaired.

Lesion of Nucleus of Sixth Nerve.

VI. There is inability to look in a certain direction, both eyes being affected alike. Usually occurs in acute cerebral lesions along with hemiplegia, the eyes and head being turned away from the paralyzed side. May be preceded by spasm in which the eyes and head are turned toward the convulsed (afterward paralyzed) limbs.

Lesion of Frontal Cortex or subcortical tract.

FIG. 18.



SIMPLIFIED DRAWING OF THE PERIPHERAL DISTRIBUTION OF THE FACIAL NERVE.
(*From Tyson, after Sahli.*)

FACIAL PARALYSIS.

Muscles supplied by all branches of the seventh nerve paralyzed with wasting and loss of faradic irritability in all but the mildest cases.[1]

I. Paralysis follows external injury.
   A. Injury occurs at birth, forceps having pressed upon nerve near its exit from the skull. *Pressure Neuritis.*
   B. Paralysis follows wounds or operations about the ramus of the jaw.
      1. Occurs immediately. *Section or Crushing of Nerve.*
      2. Occurs after a few days. *Neuritis.*
   C. Follows fracture of the skull, nervous deafness usually occurring at the same time.
      1. Occurs immediately. *Section or Crushing of Nerve.*
      2. Occurs after a few days. *Neuritis.*

II. No external injury.
   A. Onset sudden, in a few moments.
      1. Limbs on opposite side paralyzed (crossed hemiplegia) or sixth nerve on same side. *Hemorrhage or Thrombosis of Pons.*
      2. Nervous deafness and vertigo occur at the same time. *Hemorrhage pressing on Seventh and Eighth Nerves.*
      3. Taste lost in anterior two-thirds of tongue on same side. *Hemorrhage into Facial Canal above origin of Chorda Tympani.*
      4. Face alone affected. *Hemorrhage into Canal below origin of Chorda.*

[1] Paralysis of the lower part of the face without wasting or loss of faradic irritability is included under the head of Monoplegia.

B. Onset gradual but acute, in a few hours to a few days.

1. A source of intracranial infection or irritation is present. Onset of disease marked by headache and fever, often accompanied by vomiting. General hyperesthesia exists at first and is soon followed by delirium which in severe cases merges into stupor and coma, headache persisting as long as the patient can answer questions. Retraction of the head, localized twitching and general convulsions are common. Other paralyses may occur, especially in the ocular muscles. Optic neuritis, rarely intense, is common in the more protracted cases. *Meningitis.*

2. Suppurative otitis media present; no other cause. No other paralysis or spasm; mind clear.

Inflammation of Nerve Trunk.

3. Face has been exposed to cold especially in a wind or draught; no other cause. Eye muscles on the same side very rarely paralyzed. The most common form; often called rheumatic.

Inflammation of Nerve Trunk.

C. Onset of disease chronic.

1. Accompanied by headache and often by vomiting.

a. Optic neuritis present, generally intense. Other cranial nerves affected, especially the eighth and sixth. Convulsions may occur. Pulse often slow.

Intracranial Tumor, Aneurism or Cyst.

b. Patient alcoholic or syphilitic. Optic neuritis absent or slight. No fever or slowness of pulse. There may be paralysis or spasm in the domain of other cranial nerves, rarely in the limbs. Nervous deafness and vertigo generally present.

Chronic Meningitis.

c. Intention tremor is present together with nystagmus, scanning speech or other evidence of scattered lesions. *Disseminated Sclerosis.*

d. Absence of knee-jerk and other symptoms indicates tabes. Rare. *Tabes.*

BULBAR AND PSEUDO-BULBAR PARALYSIS.

Two or more of the following organs are paralyzed: lips, tongue, palate, pharynx and larynx.

I. Onset sudden in a single attack. Paralysis commonly bilateral, rarely unilateral. Palatal, pharyngeal and laryngeal reflexes usually impaired. Atrophy and reaction of degeneration may occur in affected muscles.

Apoplectiform Bulbar Paralysis.

A. Not immediately fatal.

Thrombosis of Medulla Oblongata or, very rarely, Hemorrhage or Embolism.

B. Immediately fatal.

Hemorrhage or Thrombosis of Medulla, very rarely Embolism.

II. Onset sudden but in two attacks, paralysis of the muscles supplied from the medulla not occurring until the second attack and the larynx usually escaping. Double hemiplegia or other symptoms show that each hemisphere is involved. Nutrition and electrical reactions of the paralyzed muscles not affected. Throat reflexes preserved. Very rare.

Pseudo-bulbar Paralysis due to vascular lesion in each hemisphere.

III. Onset acute, in a few hours to a few days. The affected muscles waste and lose faradic irritability. The throat reflexes are lost. There is usually also atrophic paralysis of the eye muscles, face or limbs.

A. The paralysis follows or possibly accompanies diphtheria, beginning in the ciliary muscle and palate.

Diphtheritic Bulbar Paralysis.

B. Paralysis secondary to influenza, typhoid fever or other infectious or toxic disease. Very rare.

Neuritis or, possibly, Inflammation of Bulbar Nuclei.

C. Paralysis is part of the primary disease whose onset is attended by headache, fever and perhaps vomiting or convulsions. Very rare.

Inflammation of Bulbar Nuclei, analogous to poliomyelitis.

IV. Onset generally chronic, sometimes acute or subacute.

A. There is disease in the upper part of the neck capable of affecting the ninth, tenth, eleventh and twelfth nerves at their exit from the skull, *e. g.*, tumors, cellulitis, vertebral caries, wounds, etc. No signs of intracranial disease. *Neuritis.*

B. No disease of neck. Signs of intracranial disease present.

1. Paralysis unilateral, at least at first. Lips usually escape. Headache, vomiting and mental dullness common.

a. Optic neuritis present, often intense. Pulse often slow. *Tumor or Aneurism pressing on medulla.*

b. Syphilis active; headache chiefly nocturnal. Optic neuritis absent or of slight intensity. *Syphilitic Meningitis.*

2. Onset always slow, in an adult. Paralysis bilateral from the start, beginning in the tongue, involving the lips and tending to spread to the palate, larynx and pharynx. Headache and optic neuritis absent but optic atrophy may occur. Paralyzed muscles often waste and show altered galvanic reactions even while retaining faradic irritability. Palatal, pharyngeal and laryngeal reflexes usually lost. May exist alone, but the possibility of its being only part of a widespread degeneration, such as spinal muscular atrophy, amyotrophic lateral sclerosis, tabes, disseminated sclerosis or paretic dementia, should always be carefully considered.

Typical Bulbar Paralysis, due to degeneration of nuclei.

3. Onset acute or subacute, usually in a young person. Muscles excessively fatigued when used, but recover

temporarily after rest. Electrical reactions retained but may be quickly exhausted by repetition of test. No reaction of degeneration, atrophy or sensory symptoms. May extend to masticatory, facial or ocular muscles. Symptoms may vary greatly from day to day. Paralysis of respiration or swallowing may occur, but recovery is possible.

Asthenic Bulbar Paralysis, no anatomical basis known.

LARYNGEAL PARALYSIS.

Paralysis of one or more laryngeal muscles without involvement of lips, tongue, palate or pharynx and without other indication of organic intracranial disease.

I. The neck or mediastinum is so diseased as to damage one or both pneumogastric nerves or their laryngeal branches, *e. g.*, stab wound, operation, goitre, enlarged glands, tumor, aneurism, cellulitis, etc.

A. The vocal cords move normally in breathing but remain lax on phonation, the glottis forming a wavy line. Food enters the larynx because the epiglottis does not properly cover it. Upper part of larynx more or less anesthetic. Very rare.

Bilateral lesion of Superior Laryngeal Nerves.

B. One or both cords immovable in cadaveric position (between abduction and adduction). Breathing not impeded. Phonation and explosive cough equally difficult or (in bilateral lesion) impossible.

1. Heart action disturbed; pulse rapid. Respiration may be slow. Vomiting may occur.

Lesion of Trunk of Pneumogastric.

2. Pulse and respiration not affected.

Lesion of one or both Recurrent Laryngeal Nerves.

C. Vocal cords can be adducted in phonation but only partly separated in inspiration. Breathing stridulous, especially during sleep.

Partial lesion of Recurrent Nerves.

II. There is no disease in the neck or mediastinum, but the larynx is directly injured by the swallowing of injurious substances, by pressure from a lodged bolus, or from adja-

cent growths, by syphilitic or catarrhal inflammation, excessive fatigue or the like.

Non-nervous Laryngeal Paralysis.

III. No nerve lesion or adequate local cause can be found.

A. An emotional cause can be traced and symptoms may vary much with emotional changes. History of hysterical attacks common and various other signs of hysteria may be present. Paralysis always bilateral.

1. Voice is reduced to a whisper. Vocal cords do not approach on attempted phonation but move normally in breathing and approach perfectly in explosive coughing or sneezing. May precede or follow hysterical mutism.

Hysterical Aphonia due to paralysis of adductors.

2. Voice not impaired. Inspiration stridulous; expiration normal. Very rare.

Hysterical Paralysis of Abductors.

B. No cause can be found. Paralysis sometimes unilateral.

Probably the first symptom of bulbar paralysis or of a widespread degeneration, such as spinal muscular atrophy, amyotrophic lateral sclerosis, tabes, disseminated sclerosis or paretic dementia.

PARALYSIS OF PARTIAL OR IRREGULAR EXTENT.

I. The paralysis appears only on attempting certain coördinated movements which have been performed to great excess in the patient's occupation, such as writing, telegraphing, playing a musical instrument, etc.

Paralytic form of Writer's Cramp or other Occupation Neurosis.

II. All motions requiring the action of the paralyzed muscles equally affected.

A. Onset acute, in a few hours to a few days, resembling that of an eruptive fever. Paralyzed muscles waste and lose faradic irritability. No sensory loss. Rheumatoid pains of onset usually quickly subside but may persist. Most cases in later infancy; six-sevenths of all cases under ten years of age. *Poliomyelitis.*

B. Onset acute or subacute, very rarely chronic. Paralysis affects muscle groups supplied by spinal nerves or plexuses and is accompanied or soon followed by more or less sensory loss in corresponding areas (Figs. 8 to 11). Affected muscles waste and lose their tendon reflexes and faradic irritability. Numb, tingling or burning pain in the affected area with tenderness of the muscles and often of the nerve trunks. Skin often edematous and glossy.

1. Paralysis, pain and sensory loss in the area supplied by a single spinal nerve.

a. The apparent cause is a wound, pressure on the nerve trunk, exposure, infectious or toxic disease or the extension of adjacent inflammation. *Neuritis.*

b. No traumatic, infectious or toxic cause apparent. Nodular swelling can be felt in the course of the nerve. *Neuroma.*

2. Symptoms in the area supplied by the brachial, lumbar or sacral plexus of one side.
   a. Symptoms in the arm with severe pain, greatly increased by movement, in the plexus itself as well as in the arm. Wounds or contusions affecting the plexus, forcible retraction of the shoulder, gout, rheumatism, exposure and (in the new-born) obstetrical manipulations are the most common causes.
      i. Paralysis of deltoid, flexors of elbow, supinators and often the spinati. Pain and sensory loss in shoulder and outer side of arm.

         Neuritis in upper part of plexus or, rarely, in fifth and sixth cervical roots.

      ii. Paralysis in triceps, forearm muscles and hand muscles. Pain and sensory loss in hand, forearm and inner side of arm.

         Neuritis in lower part of plexus or, rarely, in seventh and eighth cervical and first dorsal roots.

   b. Paralysis of flexors of hip, adductors of thigh and extensors of knee. Pain and sensory loss in lower part of abdomen, front and sides of thigh and inner side of leg and foot. New growths in abdomen, caries of vertebræ and ascending infection the common causes.

      Neuritis of lumbar plexus or roots.

   c. Paralysis of flexors and outward rotators of thigh, flexors of knee and muscles moving the foot and toes. Pain and sensory loss in back of thigh and all of leg and foot except inner side. Pelvic growths and inflammation, parturition and ascending infection the common causes.

      Neuritis of sacral plexus.

3. Symptoms in the areas of a number of spinal nerves on both sides, especially the external popliteal and musculo-spiral nerves. Alcoholism is the most com-

mon cause; other causes are pyemia, diphtheria and other infectious diseases, metallic poisons, exposure and ever-exertion. *Multiple Neuritis.*

C. Onset of disease chronic (except very rarely in paralysis agitans when it follows a nervous shock), but some symptoms, including paralysis, may come on rapidly, especially in disseminated sclerosis.

1. Paralysis follows tremor which begins usually in one hand and gradually spreads. Voluntary motion is not entirely lost, but becomes more and more difficult. The face becomes expressionless; the head and shoulders are bent forward, the elbows, hips and knees are slightly flexed and the hands are held in the "pill-rolling" position by a tonic spasm mainly affecting the flexor muscles (Fig. 20). The tremor usually persists during rest, but ceases temporarily during attempts to arrest it or to use the hand; in rare cases it is entirely absent and the diagnosis must be made from the weakness and the characteristic posture. Tendon reflexes normal. No sensory loss or severe pain, but much distress and unrest. No positive signs of organic disease. Patient in second half of life. *Paralysis Agitans.*

2. There is intention tremor (well seen when the patient attempts to drink) which is generally associated with nystagmus, scanning speech or other signs of multiple lesions. The paralysis may begin in individual muscles or in muscle groups, varying greatly in situation in different cases. The affected muscles may waste and lose faradic irritability, but as a rule they do not. Tendon reflexes generally exaggerated. Begins during first half of life.

Disseminated Sclerosis.

3. The paralysis is preceded by progressive dementia, often accompanied by delusions of grandeur, stumbling speech, facial twitching and pupillary changes. A disease of middle life. *Paretic Dementia.*

4. Onset of paralysis always gradual and slow, in months or years. The paralyzed muscles slowly waste and lose faradic irritability.
   a. Atrophic paralysis begins in an adult patient, usually in the hand muscles, deltoid or upper arm muscles and after spreading more or less to others on the same side commonly attacks the other side in the same order. Fibrillation is common. Reaction of degeneration may occur in the cases of more rapid onset but not in the slower ones. The muscles are never enlarged and similar cases are very seldom found in the same family.
      i. There is loss of pain and temperature senses, often of greater extent than the atrophic paralysis, with retention of touch sense in the same area. Other trophic changes such as glossy or thick and horny skin, whitlow, ulcerations, gangrene or degeneration of joints may occur. There may also be a spastic weakness of the legs without wasting.

         Syringomyelia.

      ii. There is no sensory loss and no other trophic symptom than muscular atrophy.
         § The atrophic paralysis in the upper part of the body is accompanied by spastic weakness of the legs without wasting. The tendon reflexes are exaggerated in both arms and legs.

            Amyotrophic Lateral Sclerosis.

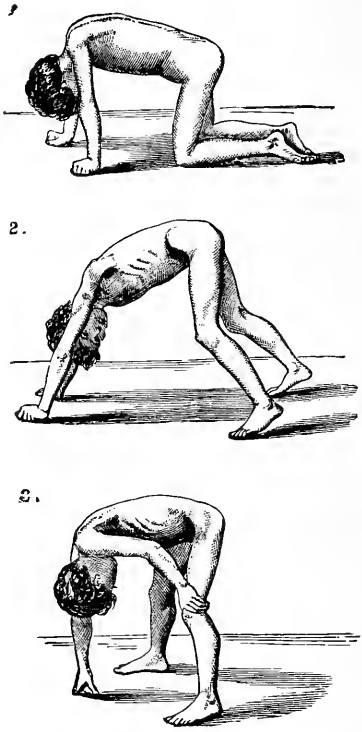
         §§ Lower limbs not affected until late in the disease when their muscles are wasted and flabby. Tendon reflexes of the affected muscles diminished or lost.

            Spinal Muscular Atrophy.

   b. Atrophic paralysis begins in childhood in the distribution of the peroneal nerves, causing talipes equinovarus. The disease slowly extends to the

muscles of the calves and thighs, and some years from the onset may involve the upper extremities (beginning in the intrinsic muscles of the hands) and the trunk. Fibrillation is common. Sensory

FIG. 19.



MODE OF RISING FROM THE GROUND IN PSEUDO-HYPERTROPHIC PARALYSIS. (*From Gowers.*)

loss may be observed. Other cases generally occur in the same family.

Progressive Neural Muscular Atrophy. (Also called Peroneal Form, Charcot-Marie Type.)

c. Paralysis begins elsewhere than in hand muscles,

deltoid or muscles supplied by the peroneal nerves. No fibrillation. No reaction of degeneration, galvanic and faradic irritability diminishing together. Other cases generally occur in the same family.

Idiopathic Muscular Atrophy.

i. Some of the weakened muscles are enlarged, usually the calves, infraspinati, quadriceps extensors, glutei or lumbar muscles. Onset usually in childhood (Fig. 19).

Pseudo-hypertrophic Muscular Atrophy.

ii. None of the weakened muscles is enlarged. Disease begins in childhood, youth or early adult life and usually first attacks the biceps, triceps, pectoral, latissimus, face, extensors of knee or flexors of hip.

Simple Idiopathic Muscular Atrophy.

ATAXIA.

The patient is unable to stand or walk steadily, not on account of weakness alone, but because of incoördination of muscular action. There may also be disordered movement in the trunk and arms.

I. A reeling gait and the associated symptoms, such as headache, vomiting, optic neuritis, paralysis in the domain of the cranial nerves or hemiplegia, indicate coarse intracranial disease directly or indirectly affecting the cerebellum or the region of the quadrigeminal bodies. The ataxia is not greatly increased by closing the eyes.

A. Onset of symptoms sudden or very rapid, usually with loss or disturbance of consciousness. The arteries are atheromatous or syphilitic or there is endocarditis. No signs of suppuration.

Hemorrhage, Thrombosis or Embolism.

B. Onset rapid or slow. There is a source of purulent infection, *e. g.*, an infected wound of the scalp or cranium (especially compound fracture), otitis media, empyema or abscess in any part of the body. Rigors followed by fever and sweating common. Temperature irregular, mostly elevated but sometimes depressed. Duration may be short or it may be long with a period of latency. Optic neuritis is common but is rarely intense.

Abscess.

C. Onset chronic. No source of infection but a personal or family predisposition to new growths may be apparent. Temperature runs a normal or nearly normal course. Headache generally intense, often accompanied by vertigo and vomiting. Progressive mental failure occurs toward the end. Optic neuritis is the most char-

acteristic symptom; it is present in four-fifths of all cases and is usually intense. Course long and mostly progressive. *Tumor.*

II. The associated symptoms, such as absence of knee-jerk, slight urinary difficulty, Argyll-Robertson pupil, optic atrophy, lightning pains, nystagmus, stumbling or scanning speech, or mental impairment of the paretic type, are of slow onset and indicate a degenerative disease affecting the spinal cord or both cord and brain.

A. The ataxia of gait and station is greatly increased by closing the eyes. The knee-jerks are absent except in rare cases in which advanced optic atrophy is an early symptom. A history of lightning pains in the legs is common, also of loss of virility, slight urinary difficulty and ptosis or diplopia. Argyll-Robertson pupil in most cases. *Tabes.*

B. A jerky or reeling gait begins in childhood or youth in members of certain predisposed families and is sooner or later accompanied by nystagmus, stumbling or blurred speech and incoördination of neck and arm muscles. Knee-jerks generally but not always absent, in rare cases increased. Paralysis and corresponding deformities may occur, especially in the later stages.

Hereditary Ataxia (*Friedreich's Disease*).

C. Weakness of legs and ataxia of gait and station begin in adult life and gradually increase. Knee-jerks and other tendon reflexes almost always increased, very rarely lost. Lightning pains very rare. Slight difficulty of articulation common. *Ataxic Paraplegia.*

D. Intention tremor with nystagmus or scanning speech is associated with various signs of scattered lesions, such as isolated paralyses, contraction of the visual fields with impairment of color sense, loss of smell, nervous deafness, etc. Knee-jerks generally exaggerated.

Disseminated sclerosis.

E. Failure of judgment and memory, often combined with monstrous and unsystematized delusions of grandeur,

accompanies signs of organic disease, among which inequality of pupils, facial twitching and stumbling speech are common. *Paretic Dementia.*

III. The associated symptoms (such as paralysis, wasting, loss of faradic irritability, tenderness of muscles and nerves, numb stinging pain and some sensory loss, chiefly in the distribution of the external popliteal or external popliteal and musculo-spiral nerves), together with evidence of a toxic influence, indicate multiple neuritis. Knee-jerk lost except in the rarest cases. Argyll-Robertson pupil absent. Onset of ataxia usually acute or subacute, always more rapid than in the average case of tabes. Other symptoms may not be marked.

A. History of prolonged alcoholic excess. Various signs of alcoholism may be present. Pain a prominent symptom. *Alcoholic Neuritis.*

B. The ataxia is preceded by diphtheria. Onset of neuritis marked by loss of ocular accommodation and paralysis of the palate. Pain absent or slight. *Diphtheritic Neuritis.*

C. History of acute or chronic poisoning by arsenic, or arsenic is found in the urine. Both arms and legs usually affected. Pain a prominent symptom. Herpes zoster common. *Arsenical Neuritis.*

D. The ataxic form of neuritis may sometimes be due to various acute infectious diseases, malaria, septicemia, diabetes, beri-beri or leprosy.

IV. There is no evidence of organic disease. The ataxia appears first after an emotional disturbance and may increase or diminish in correspondence with emotional changes or suggestion. It may exist in any degree and sometimes prevents either standing or sitting (astasia-abasia). Various signs of hysteria may be present. *Hysterical Ataxia.*

TREMOR.

I. The tremor is due to emotion, exposure to cold or extreme fatigue in a healthy person. It is fine; rate about 10 per second. *Physiological Tremor.*

II. The tremor is an expression of the weakness of an exhausting disease, such as typhoid fever, or of neurasthenia. It is fine and rapid and ceases during rest. *Asthenic Tremor.*

III. The tremor is due to poisoning by alcohol, mercury, lead, arsenic, opium, chloral, tea, coffee or tobacco.

Toxic Tremor.

IV. The tremor is a symptom of organic disease of the nervous system, but is of slight diagnostic importance compared with other symptoms, as in hemiplegia, cerebral tumor or abscess, cerebellar disease, paretic dementia, tabes, hereditary ataxia, etc.

Diagnosis as in Hemiplegia, Optic Neuritis, Headache, Ataxia, etc.

V. Tremor is a prominent and important symptom.

A. The tremor begins after forty years of age, usually in one hand, rarely in one leg and slowly extends to the other limb on the same side and then to the limbs on the opposite side. Its rate is five to seven oscillations a second and the range is generally small. As a rule it continues during rest and is lessened or stopped by effort; rarely, in an early stage, it is elicited by effort. Following the tremor, beginning in the same part and spreading in the same order, muscular weakness and rigidity appear, the muscles, especially the flexors, gradually contracting so as to cause a characteristic posture with absence of facial expression. The hands assume the "pill-rolling" position; the knees, hips, wrists, elbows and shoulders, are somewhat flexed; the head

and spine are bent forward (very rarely the head is bent backward) and the face stares straight ahead. Patients are restless, uncomfortable and unhappy and often complain of painful sensations of heat or cold. The tendon reflexes are generally normal, rarely exaggerated and there is no nystagmus. *Paralysis Agitans.*

FIG. 20.

PARALYSIS AGITANS. (*From Gowers, after St. Leger.*)

B. The disease generally begins in early youth or adult life but may begin at any age. Weakness of the limbs or of some of the muscles supplied by the cranial nerves is usually the first symptom. The tremor is characteristic and is called intention tremor because it occurs when the hands are used and subsides during rest. It is coarse, jerky and irregular, the average rate being about 6 per second. Nystagmus is common. Speech is usually stacatto or scanning at first, in a monotonous voice, later becoming

slurred and indistinct. The tendon reflexes are generally exaggerated. A great variety of other symptoms, such as might be caused by scattered lesions, may occur, including impairment of the visual fields for form and colors, optic neuritis, optic atrophy, nervous deafness, and ataxia. The patient is often unduly complacent.

Disseminated Sclerosis.

C. The tremor is part of a general nervous disturbance, generally in a woman, of which rapid heart action, enlargement of the thyroid gland, protrusion of the eyeballs and general vaso-motor dilatation are prominent symptoms. The tremor may be very fine and rapid or it may be so coarse and irregular as to suggest chorea. It is usually elicited by voluntary action.

Exophthalmic Goitre.

D. Tremor occurs only on attempting to perform a certain habitual and highly coördinated movement of the hands such as writing, engraving or playing on a musical instrument, which has been performed to excess. May be accompanied by painful sensations, spasm or weakness. *Occupation Neurosis.*

E. The tremor begins in old age, is mostly in the hands and head and is fine and rapid, diminishing during rest and ceasing during sleep. Rigidity and weakness are absent. *Senile Tremor.*

F. The history and symptoms indicate hysteria and no other cause is found. The tremor is very irregular both in time and amplitude and may shade into the rhythmical movements of hysteria described under localized spasms.

Hysteria.

GENERAL SPASMS.

Spasmodic muscular contractions not limited to one region of the body.

I. Contractions occur at very short intervals, not in distinct paroxysms, and involve different groups of muscles in irregular succession.

A. The movements are jerky and irregular, at first appearing to be such as might be made voluntarily by a fidgety or perverse child, *e. g.*, grimaces, thrusting out the tongue, jerking of the hands so as to upset objects or sudden relaxation of grasp so as to drop them, grotesque changes in the posture of the head, trunk and limbs. The movements usually affect the two sides of the body unequally and are sometimes limited to one side. In a severe case they may become very violent. Speech is often made indistinct or entirely arrested. Often associated with rheumatism and may be complicated by endocarditis. Most cases occur between five and fifteen years of age, nineteen-twentieths before twenty. Two-thirds of the patients are girls. *Chorea.*

B. Movements bear a general resemblance to those of chorea but are less jerky and more rhythmical. A mental cause can usually be found, especially imitation of other patients. Other evidences of hysteria generally present. *Hysterical Chorea.*

C. Movements generally coarser and more pronounced and postures more extravagant than those of chorea. Begins usually between thirty and forty years of age, rarely before twenty, in members of certain families. Ends in dementia. *Hereditary Chorea.*

D. Movements like those of chorea or hereditary chorea. Disease begins in middle or advanced life, is not mark-

edly hereditary and rarely ends in dementia or shortens life. *Senile Chorea.*

E. Spasmodic contractions occur mostly in the large muscles near the trunk, especially in the quadriceps, flexors of the knee, calf muscles, deltoid, biceps, triceps, supinator, lower face and neck, the distal part of the limbs being comparatively unaffected. The spasm usually appears first in the shoulders, upper arms or face, and is clonic or a combination of clonic and tonic, sometimes with tetanic exacerbations. The clonic contractions are abrupt, as though due to electric shocks, and irregular, recurring from ten to fifty times a minute. They may affect single muscles not separately under control of the will or successive parts of a muscle and are usually not sufficient to cause movement of the limbs.

Paramyoclonus Multiplex.

II. Contractions occur in a distinct paroxysm or convulsion which may or may not recur.

A. Consciousness is lost or obscured during the paroxysm.

1. There is evidence of organic disease of the brain or its membranes, consisting of such signs as globular enlargement of the cranium, inequality of pupils, absence of light reaction, paralysis of the face or of ocular muscles or of one side of the larynx, optic neuritis or atrophy, localized spasm of the Jacksonian type or a localized beginning of the general spasm, typical ankle clonus without hysterical contracture, etc. Convulsion epileptiform. Persistent headache and vomiting common in the intervals.

Organic Cerebral Disease.

The differential diagnosis is made on the same principles as in hemiplegia, whether this symptom is actually present or not.

2. Signs of organic disease are absent.

a. Paroxysms recur, irrespective of any definite toxic or reflex cause, forming a series.

i. Aura, if it occur, short; absolute loss of con-

sciousness; a single cry; fall, regardless of danger; dilatation of pupils; tonic spasm; cyanosis; clonic spasm; frothing at mouth, often bloody from tongue having been bitten; involuntary evacuations may occur; gradual lengthening of interval between jerking movements followed by cessation of spasm and return of consciousness. During the convulsion words are never uttered and the movements never express any emotion or purpose whatever. Convulsion lasts only a few minutes and is generally followed by deep sleep.

Epilepsy (*grand mal*).

ii. An emotional exciting cause frequent. Prodromes often long; if there is a cry it may be repeated; if a fall it is not utterly regardless of danger; rigidity; contortions, especially arching the body and placing the head or limbs in grotesque positions; the patient may struggle with the attendants; countenance and movements express emotion and purpose; excited exclamations may occur; tongue not bitten though lips or hands may be; no involuntary discharges. Attack may last a long time and contortions be many times repeated; may often be arrested by pressure on sensitive parts or by an emetic. *Hysteria.*

iii. An emotional exciting cause frequent. Prodromes often long; actual onset usually sudden. The limbs on both sides become rigid and may remain so without jerking, the arms in flexion or extension, the lower limbs in extension with the feet usually inverted. The force of the spasm may vary greatly under attempts to overcome it. The attack may last a long time but may be arrested by an emetic. Other signs of hysteria present. *Hysteria.*

iv. Onset sudden after an emotional exciting cause. Tonic spasm affecting the muscles throughout the body maintains it in a fixed posture. The face is usually expressionless but may express an emotion. At first there is great resistance to a change of posture but in a few minutes the limbs yield to moderate force and they then remain for a time in the posture given them; but if a limb is unsupported within fifteen or twenty minutes its weight begins to prevail and it slowly descends. The duration of the attack varies from minutes to days but it may be arrested by an emetic. It is almost always a symptom of hysteria and may form only one phase of an hysterical convulsion; occasionally it is associated with insanity. *Catalepsy.*

b. Paroxysm solitary or, if more than one, each may be attributed to a definite and temporary cause. The convulsion is not in itself distinguishable from that of epilepsy and similar attacks may afterward be repeated in series so as to constitute true epilepsy.

i. Patient an infant or young child, generally having, along with the predisposition to convulsions incident to its stage of development, some additional predisposing cause, particularly an inherited neurotic taint or rickets.

§ Digestion disordered by excessive or improper food, intestinal parasites or gastro-enteritis.

Eclampsia of Gastro-enteric Irritation.

§§ Convulsions are associated with irritation due to eruption of the teeth; no other exciting cause. *Eclampsia of Dentition.*

§§§ Convulsions mark the onset of an acute

infectious fever, such as scarlatina, measles or poliomyelitis, corresponding to the rigor of an adult patient.

Eclampsia of Acute Fever.

ii. Patient generally an adult, but may be of any age.

§ Convulsion immediately follows a blow on the head. *Eclampsia of Concussion.*

§§ There has been a serious loss of blood or an exhausting discharge. Pulse very small and weak. If the fontanelle is still open it is depressed.

Eclampsia of Cerebral Anemia.

§§§ There is evidence of a toxic substance in the circulation.

! The urine contains albumen or casts or its specific gravity and total quantity indicate defective elimination. Signs of uremia common, *e. g.*, edema, pallor, albuminuric retinitis.

Uremic Eclampsia, including Puerperal Eclampsia.

!! History of excessive consumption of alcohol, perhaps of delirium tremens, or of other forms of alcoholism. Signs of alcoholism common, such as characteristic odor of the breath, bloated face and bleary eyes. No other cause. *Alcoholic Eclampsia.*

!!! History of exposure to lead. Signs of plumbism present, *e. g.*, blue line on the gums, dry colic, bilateral wrist-drop. *Plumbic Eclampsia.*

B. Consciousness not essentially obscured during the paroxysm.

1. The spasm is tonic and begins in the muscles of mastication, extending gradually to those of the neck, spine, chest and abdomen. Paroxysms of general

tonic spasm then occur and cause risus sardonicus, opisthotonos and rigidity of limbs, during which the eyes are open and the patient suffers severely. Between the paroxysms there is continuous tonic rigidity, especially of the jaws and neck. History of a wound that may have been infected through earth or manure, or of exposure to cold, or, in the new-born, an infection of the umbilicus. *Tetanus.*

2. Paroxysms of bilateral tonic spasm begin in the muscles of the hands, or of the hands and feet, after prodromal tingling and stiffness; spasm extends toward the trunk which is also involved in severe cases. Hands in obstetric position, wrists and elbows slightly flexed, arms adducted, toes flexed, feet in equino-varus position, knees and hips extended or rarely flexed, thighs adducted. The jaw is not affected until late in the attack if at all. In the intervals spasm may be excited by pressure on nerve trunks or arteries (Trousseau's symptom). Galvanic, faradic and mechanical irritability of the affected muscles enormously increased. Occurs chiefly in children often associated with rickets and sometimes with laryngismus stridulus and eclampsia. In adults it may occur in conditions of exhaustion or after exposure to cold or removal of the thyroid gland. Very rare in North America and rare in England; more common in continental Europe. *Tetany.*

3. History of surroundings may indicate probability of poisoning. Patient at first exhilarated. Spasm comes on rapidly, beginning in the limbs and extending to the trunk so as to cause opisthotonos. Eyes open. Relaxation in intervals. Jaw affected later in the attack than other parts and relaxes earlier.
Strychnia Poisoning.

4. Onset from twelve days to several months after the bite of a rabid animal. Spasm begins in pharynx, causing dysphagia and horror of liquids. A quick

inspiratory jerk and general tetanoid convulsions are soon added to the pharyngeal spasm. Mind at first clear, but delirium or frenzy may supervene. Excessively rare in North America. *Hydrophobia.*

III. Continuous tonic spasm holds two or more limbs in a fixed position, the arms in rigid flexion or extension, the lower limbs generally in rigid extension with the feet inverted but sometimes in flexion. The onset is often sudden after an injury or an emotional disturbance but may be gradual. At first the force of the spasm varies greatly when attempts are made to overcome it and under variations in the patient's attention to it, while under an anesthetic it relaxes completely; but later there may be structural shortening of the muscles which cannot be overcome even in profound anesthesia. An atypical foot clonus can sometimes be obtained but unequivocal signs of organic disease of the nervous system are absent. Other symptoms, obviously hysterical, are generally present. *Hysterical Contracture.*

IV. Permanent tonic contraction of the flexors generally and of the muscles of the face comes on very slowly, after forty years of age, and causes a characteristic posture with absence of facial expression. (Fig. 20.) The hands assume the "pill-rolling" position; the knees, hips, wrists, elbows and shoulders are somewhat flexed, the head and spine bent forward, or very rarely the head bent backward; the face is blank and stares straight ahead. Generally but not always preceded by a characteristic tremor, which begins in one hand (or rarely in one leg) and gradually extends to the opposite side. Patients are restless, uncomfortable and unhappy and often complain of painful sensations of heat or cold. Tendon reflexes generally normal, rarely exaggerated. There is no nystagmus. *Paralysis Agitans.*

LOCALIZED SPASMS.

Spasms limited to one region of the body in voluntary muscles that are not paralyzed.

I. The spasm occurs only in distinct paroxysms.

A. The attack begins in a definite part of the body commonly with a feeling of numbness. Tonic or clonic spasm then occurs and may spread to other parts, always in an order corresponding to the arrangement of the cortical motor centers, *e. g.*, beginning in a foot it passes up the limb to the trunk, then to the arm and only later to the face; beginning in the arm it may next affect either the face or the leg. Consciousness is not lost unless general convulsion ensues. The convulsed part is weakened, or even paralyzed, for a time subsequent to the attack, varying from a few minutes to many hours. For the twenty-four hours following a paroxysm the total quantity of urinary solids is increased. When attacks occur many times daily the temperature is elevated above 101° and there is rapid mental deterioration. Other signs of organic disease usually present.

Jacksonian Epilepsy. Caused by organic disease irritating the cortex; nature to be determined by the accompanying symptoms and their mode of onset.

B. The attack begins and may spread as in Jacksonian epilepsy but the convulsed part is not weakened, there is no mental deterioration and prolonged observation reveals no other indication of organic disease. For the twenty-four hours following an attack the total quantity of urinary solids is diminished. The spasm may often

be excited by observation or direct suggestion and there are stigmata or other proofs of hysteria.

Hysterical Simulation of Jacksonian Epilepsy.

C. The spasm is in the adductors of the larynx.

1. The patient is usually a child with rickets, most commonly from six to eighteen months of age. Respiration is suspended by sudden closure of the glottis; the head is thrown back, the face at first pale then livid. Spasm of other respiratory muscles and of hands and feet may be associated.

Laryngismus Stridulus.

2. The patient is a child with catarrhal laryngitis. Partial closure of the glottis comes on during sleep, causing dyspnea with inspiratory stridor.

Spasmodic Croup.

3. The patient is a youth or an adult.

a. There is absence of knee-jerk, together with Argyll-Robertson pupil, ataxic gait or station when eyes are closed, lightning pains or other symptom of tabes. *Laryngeal Crisis.*

b. The paroxysms are traceable to an emotional disturbance or to suggestion. Signs of organic disease absent; stigmata of hysteria present.

Hysterical Spasm of Glottis.

c. Paroxysms occur mostly at night in neurotic patients, but are not traceable to suggestion or emotion. Stigmata of hysteria absent.

Neurotic Spasm of Glottis.

D. The spasm is in the constrictor vaginæ and levator ani, the paroxysms occurring on attempted coitus. The patients are newly married, neurotic women.

Vaginismus.

II. The spasm occurs only on attempted use of the affected muscles.

A. The spasm occurs on attempting a particular movement which is necessary in the patient's occupation and has been excessively repeated, as writing or playing a

musical instrument, but does not occur during rest or when performing any other action. Usually accompanied by a feeling of fatigue or pain.

Occupation Neurosis.

B. On attempting to stand spasm causes an alternating flexion and extension of the lower limbs. Rare, probably hysterical. *Saltatoric Spasm.*

C. Tonic spasm comes on when a movement is attempted after a period of rest, passing away if the attempt is persisted in. Especially affects the legs, but may also appear in the hands or face. Hereditary and usually congenital. Rare. *Myotonia (Thomsen's Disease).*

D. On attempting to speak or perform some other voluntary action of the larynx spasm of the adductors occurs, although all involuntary and automatic functions are properly performed. Rare, probably hysterical.

Phonic Laryngeal Spasm.

III. The spasm causes persistent and rhythmical movements of alternate flexion and extension of the limbs, nodding or rotation of the head, protrusion and retraction of the tongue, etc. *Hysteria.*

IV. The spasm has no relation in time to voluntary movements, which are not seriously impaired. The spasmodic movements, such as winking, grinning, nodding the head, shrugging the shoulders, jerking a limb or jumping, have the appearance of being voluntary although inappropriate, but they are repeated in spite of efforts to control them.

A. The spasm begins in early life, often as a voluntary or automatic movement due to a definite exciting cause, and persists as a habit after the original cause has ceased to act. *Habit Spasm.*

B. The movements are more abrupt and rapid than those of habit spasm. Bodily and mental stigmata of degeneracy commonly present. Often accompanied by an impulsive tendency to utter certain senseless expressions, to repeat the words of others (echolalia) or their gestures (echokinesis) or to utter obscene words (coprolalia). Efforts

to control the impulsive actions cause fatigue and distress.

Convulsive Tic.

V. Spasms not included under the previous headings. May be tonic or clonic or both, with or without paroxysmal exacerbations.

A. In muscles within the orbit. Not accounted for by errors of refraction. Positive signs of organic disease absent.

1. Farthest point of distinct vision coincides with the normal near point. Monocular polyopia, micropsia, or macropsia often associated with the myopia. Onset usually sudden. The symptoms are dispelled by the use of an appropriate concave glass but may not yield promptly to atropia. Often shown to be hysterical by the presence of hysterical amblyopia or other hysterical stigmata or by the curative effect of suggestion. *Spasm of Accommodation.*

2. There is convergent (or very rarely divergent?) squint. The double images are at a constantly varying distance from each other, but do not approach or separate when the object is moved from one side to the other as in ocular palsies. The field of fixation of either eye taken separately is normal in recent cases and in cases of long standing is only flattened on the side opposite the contracted muscle. Onset usually sudden, often after an hysterical seizure.

Spasm of External Ocular Muscles.

B. Tonic spasm of the muscles of mastication prevents the jaws from being separated.

1. There is evidence of organic intracranial disease. Headache, vomiting and fever common.

Organic Disease. Nature to be determined from the accompanying symptoms and their mode of onset; usually meningitis.

2. Signs of organic disease absent.

a. After an infected wound, especially one infected by earth or the refuse of stables, tonic spasm begins in the muscles of mastication and gradually

spreads to the muscles of the neck, spine, chest and abdomen. Paroxysms of general tonic spasm, causing opisthotonos and severe pain, occur spontaneously or on slight provocation. Temperature may be normal or elevated. The mind is clear.

Tetanus.

b. There is a source of reflex irritation such as caries or faulty eruption of a tooth, a wound of the inferior maxilla, inflammation of the temporo-maxillary joint or suppurative tonsillitis. The spasm does not spread and ceases when the source of irritation is removed. *Reflex Trismus.*

c. The jaws remain closed after an hysterical attack or an emotional disturbance or in response to suggestion. No infection; no reflex cause. Other signs of hysteria generally obvious.

Hysterical Trismus.

C. Spasm in the distribution of the facial nerve.

1. Limited to the orbiculares palpebrarum, usually clonic and bilateral but may be either tonic or clonic, bilateral or unilateral. Tonic closure of the lids may simulate paralytic ptosis but is readily distinguished by concentric wrinkles in the skin, by the closure being unaffected by posture and by active resistance when the physician attempts to open the lids. Pressure points of excitation or arrest common. Disease may be reflex, neurasthenic or hysterical.

Blepharospasm.

2. Especially affects the zygomatici and orbicularis palpebrarum, the orbicularis oris being generally unaffected although the platysma, depressor anguli oris and levator menti are sometimes involved. Mostly unilateral but may extend to the other side. Sometimes there are brief paroxysms of tonic and clonic spasm, sometimes single contractions occurring at irregular intervals. Generally begins in the second half of life, very rarely before thirty. *Facial Spasm.*

3. Tonic spasm, to which fine clonic contractions are added, retracts one angle of the mouth while the tongue is spasmodically turned to the same side. The orbicularis palpebrarum is generally unaffected but the platysma may be involved. The sound side appears to be paralyzed but it is not relaxed and the mouth opens more widely on the side of the spasm. Other signs of hysteria generally present. Rare.

Hysterical Glosso-labial Spasm.

D. Spasm limited to the tongue. It may be tonic, clonic, rhythmical or mixed, paroxysmal or continuous. Patients mostly hysterical or epileptic. Rarely caused by reflex irritation. *Glosso-spasm.*

E. The spasm affects the rotators of the head, especially the sterno-mastoid and trapezius. It is tonic or clonic or tonic and clonic combined. The ear of the affected side is usually brought nearer the inner end of the clavicle, the chin being elevated as well as turned to the opposite side, but the head may simply be rotated.

1. Begins in the second half of life, only rarely before thirty. Not amenable to mental treatment. Signs of hysteria absent except very rarely when the two diseases simply co-exist. *Spasmodic Torticollis.*
2. Occurs in youths or young adults predisposed to hysteria. The influence of mental changes in exciting or restraining the spasm is apparent. Other signs of hysteria present. *Hysterical Torticollis.*

F. The spasm is in the retractors of the head.

1. There are signs of organic disease such as inequality of the pupils, impaired light reaction, paralysis of eye muscles or of the face or tongue or one side of the larynx, optic neuritis, optic atrophy or Jacksonian epilepsy, together with headache and often vomiting or with severe pain at the back of the neck.

Organic Disease within the cranium or upper part of the spinal canal, most commonly meningitis.

2. Signs of organic disease absent.
   a. The disease is chronic and begins usually in the second half of life, only very rarely before thirty. The spasm is like that of spasmodic torticollis. Exacerbations are made more frequent and intense by emotion but the disease is not amenable to mental treatment. Stigmata of hysteria absent except in rare cases in which the two diseases co-exist.

      Spasmodic Retrocollis (*Bilateral Torticollis*).

   b. The spasm occurs as a complication of a severe fever, *e. g.*, typhoid. It may easily be mistaken for a symptom of meningitis from which it is distinguished by the absence of any unequivocal sign of organic disease and by the subsequent course.

      Functional Retrocollis of Infectious Fever.

   c. Retraction of the head persists after an hysterical convulsion or occurs in response to suggestion.

      Hysterical Retrocollis.

G. Tonic spasm causes persistent rigidity of some part of the spine and is liable to exacerbations, perhaps with the addition of clonic spasm. Pain is felt in the affected part of the spine and there are also radiating pains in the course of the nerves issuing from this part, often causing girdle sensation. At first there is hyperesthesia in the distribution of these nerves but later there may be anesthesia and atrophic paralysis. Paraplegia and sensory loss up to the level of the affected part, together with disturbance of the bladder and rectum, may eventually appear showing that the substance of the cord has become diseased.

1. Deformity or swelling with deep-seated tenderness indicates disease of the vertebræ.
   a. The patient is usually a child, sometimes a young adult, rarely an elderly person. The tubercular diathesis is almost always manifest but very rarely syphilis may be the cause. The pain is generally

of moderate severity, increased by motion or jars and diminished by rest of the spine. Prominence or lateral displacement of one or more spinous processes is the characteristic deformity.

Spinal Caries.

b. The patient is in the second half of life, often with a history of tumor elsewhere or of predisposition to new growths or to aneurism. The pain is very intense and is very greatly aggravated by motion.

§ The radiating pain is on the left side of the chest. Characteristic thrill and murmur at the seat of pain and deformity.

Spinal Aneurism.

§§ Radiating pains on both sides. No thrill or murmur. *Spinal Tumor.*

2. Nothing to indicate disease of the vertebræ.

a. Onset sudden. No fever at first.

Spinal Meningeal Hemorrhage.

b. Onset acute, marked by chill and fever.

Acute Spinal Meningitis.

c. Onset chronic.

i. History of alcoholism, syphilis or exposure to cold. *Chronic Spinal Meningitis.*

ii. Evidence of predisposition to new growths. No other cause of meningitis.

Intraspinal Tumor.

H. The spasm is in the muscles of a single joint, most frequently the hip, knee, shoulder or ankle.

1. The joint is persistently rigid, the spasm not being relaxed when attention is withdrawn nor even during sleep, nor is it overcome by moderate, persistent force. Any attempt to move the joint causes pain which is not superficial and not markedly affected by attention. Complaints of pain are often involuntary and accompanied by the facial expression of suffering; night startings and cries are common. The muscles, espe-

cially the extensors, are wasted more than disuse would account for, and their electrical reactions may be diminished. The local temperature is almost always persistently elevated one-half a degree to three degrees; very rarely it is normal or subnormal. The posture is never one of complete flexion or complete extension but is that of greatest ease. The symptoms do not undergo marked change in a short time. Under profound anesthesia the spasm relaxes and then additional signs of joint disease are apparent.

Reflex Spasm of Joint Disease.

2. The spasm relaxes more or less during sleep and may often be entirely overcome by persistent moderate force if the patient's attention is withdrawn. The pain complained of is far greater than the other signs of disease would lead one to expect, but is often superficial and is markedly affected by attention. Night startings, involuntary cries and facial signs of suffering are absent. The muscles are not wasted more than disuse would account for and their electrical irritability is normal. The local temperature is generally normal or subnormal; rarely it is temporarily elevated, never persistently so. The posture often differs widely from that of greatest ease and in many cases automatic actions which would cause great pain in real joint disease are performed without complaint. The symptoms often change greatly in a short time. Examination under anesthesia reveals no signs of joint disease.

Hysterical Simulation of Joint Disease.

I. Continuous tonic spasm of one arm or one leg or of arm and leg on one side, leaving the face free, fixes the arm in rigid flexion or extension, the lower limb in rigid extension with the foot usually inverted. The onset is often sudden after an injury or an emotional cause. The force of the spasm varies greatly when attempts are made to overcome it and under variations in the patient's attention to it. Anesthesia

of the hysterical type often exists over the affected muscles, sometimes over the whole limb or the affected half of the body. An atypical foot clonus can sometimes be obtained, but unequivocal signs of organic disease are absent. Other symptoms of hysteria are generally present.

Hysterical Contracture.

OPTIC NEURITIS.

The optic papilla is swollen and its margin is obscured by inflammatory exudate. Some vessels may be hidden here and there by the exudate and the veins may be tortuous and overdistended. Venous hemorrhages may occur. Where the veins pass over the inclined margin of the disk they appear darker because, not being at right angles to the line of vision, their central light reflex is lost. The neuritis is said to be slight when the papilla is but slightly swollen and its margin not altogether obscured; it is intense when the papilla is much swollen and appears to extend far beyond its normal margin, which is entirely obscured. In such cases the veins are usually distended and hemorrhages are common, giving rise to the term choked disk. This intense neuritis without retinitis is highly characteristic of intracranial tumor. When both the papilla and the retina are inflamed the condition is called neuro-retinitis.

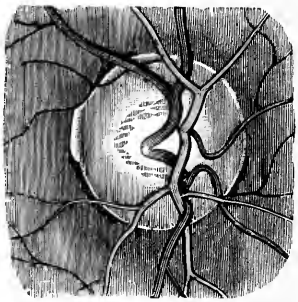
I. The neuritis is almost always unilateral and is caused by inflammation, hemorrhage or new growth within the orbit.

II. Neuritis almost always bilateral, not caused by disease within the orbits.

   A. Confirmatory signs of the existence of organic intracranial disease (such as paralysis of ocular muscles, face, tongue or one side of larynx, paralysis of limbs that is not spinal, peripheral or functional, Jacksonian epilepsy, typical ankle clonus, coincident mental impairment, etc.) are absent. The optic neuritis is almost always slight and may be accounted for by a systemic condition or by the presence of disease outside the cranium.

1. The blood is very greatly impoverished as in severe simple anemia, pernicious anemia or leucocythemia. Hemorrhagic retinitis is generally present.
2. There is a systemic infection as in variola, scarlatina, measles, severe malaria, influenza, diphtheria, typhus, typhoid or puerperal fever or syphilis. Optic neuritis without other signs of organic disease is rare in all of these diseases and when it occurs is usually part of a neuro-retinitis.
3. There is a condition of toxemia as in uremia, alcoholism, plumbism, chorea, gout, hydrargyrism or tobacco poisoning. Optic neuritis is very rare except in uremia, where there is usually in addition a characteristic retinitis, and in chorea, where very slight neuritis is said to occur in from eight to ten per cent. of all the cases.

FIG. 21.



HEALTHY OPTIC DISK. (*From Ormerod, after Nettleship and Jäger.*)

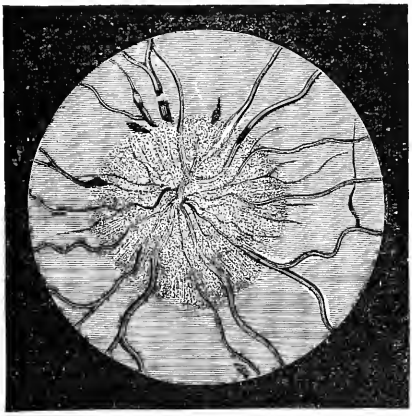
4. There is disease outside the cranium with which the optic neuritis is connected in a way not now understood, *e. g.*, inflammation or injury of the upper part of the spinal cord, suppurative otitis media or acute suppression of menstruation.

B. Confirmatory signs of organic cranial or intracranial lesion are present or appear in the course of the disease.

Headache and vomiting are common. There is no disease outside the cranium sufficient to account for the optic neuritis and the accompanying symptoms. The optic neuritis is often intense in tumor, very rarely so in other conditions.

1. The cranium is injured or diseased in such a way as to affect the optic nerve directly or to cause meningitis, which in turn causes the neuritis, as in fractures

FIG. 22.



INTENSE OPTIC NEURITIS. (*From Ormerod, after Nettleship and Jackson.*)

at the base, caries of the sphenoid bone or chronic thickening of the cranial bones.

2. There is no external evidence of cranial injury or disease; the lesion is intracranial.
   a. The disease is congenital or develops gradually in infancy. There is globular enlargement of the cranium. *Hydrocephalus.*
   b. The onset of cerebral symptoms is sudden or very rapid, in a few seconds to a few hours. Optic neuritis slight.
      i. Onset coincident with a blow on the head

which causes loss or disturbance of consciousness. No evidence of a vascular lesion.

Cerebral Concussion.

ii. Onset coincident with, or follows a blow on the head, or occurs spontaneously during a weakened condition of the cerebral arteries. Hemorrhage is indicated by headache on the side of the lesion (usually the side of the more intense neuritis) or by cranial nerve palsies on this side or by paralysis of face or limbs, often preceded by spasm, on the opposite side; also by sopor, coma and slowness of pulse. *Meningeal Hemorrhage.*

iii. Onset sudden, marked by loss or disturbance of consciousness and hemiplegia. There is no traumatic cause but an infectious endocarditis or a pulmonary abscess is present. *Cerebral Embolism.*

c. Onset acute or subacute, in a few hours to a few weeks, with irregular fever. A source of intracranial irritation or infection is present, *e. g.*, suppurative otitis media, tuberculosis, pneumonia, epidemic influence, wound of the head or sunstroke. General hyperesthesia in the earlier stage is followed by delirium which merges into stupor and coma. Spasm of the neck, general and local convulsions and cranial nerve palsies are common.

Meningitis, or in rare cases abscess or diffuse cerebritis; differential diagnosis not always possible.

d. Onset chronic, in six weeks or longer.

i. There is a source of purulent infection, *e. g.*, an infected wound of the scalp or cranium (especially compound fracture), otitis media, empyema or abscess in any part of the body. Rigors followed by fever and sweating com-

mon. Temperature irregular, usually elevated but sometimes depressed. Duration may be short or it may be long with a period of latency. *Intracranial Abscess.*

ii. No source of infection but a personal or family predisposition to new growths may be apparent. Temperature normal or nearly so. Headache generally intense, often accompanied by giddiness and vomiting. Progressive mental failure occurs toward the end. Optic neuritis the most characteristic symptom; it is present in four-fifths of all the cases and is usually intense. Course long and mostly progressive.

Intracranial Tumor, including aneurism and hydatid cyst.

iii. Patient alcoholic or syphilitic. Fever absent or slight. Optic neuritis not intense. Organic disease indicated by spasm or paralysis in the distribution of cranial nerves, rarely in the limbs. *Chronic Meningitis.*

iv. Optic neuritis occurs very rarely in paretic dementia, disseminated sclerosis and chronic cerebritis, but is not likely to be an important factor in the diagnosis of any of these conditions. Paretic dementia is usually easily recognized by its peculiar mental symptoms together with some sign of organic disease; disseminated sclerosis by intention tremor, nystagmus, scanning speech, inconstant and irregular paralyses, etc. Chronic cerebritis can not now be distinguished during life from tumor or chronic meningitis.

OPTIC ATROPHY.

The optic disc is sharply defined and abnormally white, having lost its normal rosy tint, owing to the disappearance of the capillary vessels. The retinal arteries and veins are generally smaller than normal.

I. The atrophy is secondary to optic neuritis, which may be shown either by an earlier ophthalmoscopic observation or by the presence of exudate in the nerve head, concealing the lamina cribrosa and giving a "filled in" appearance to the disc and sometimes extending outward along the retinal vessels. The appearance of the disc alone is not always sufficient to distinguish post-neuritic atrophy from simple atrophy.

Diagnosis to be made as in Optic Neuritis, q. v.

II. The atrophy is non-neuritic, or simple, which may be shown by the absence of an earlier observation of neuritis and the absence of exudate in the nerve head and along the retinal vessels. The lamina cribrosa is more exposed than normal.

A. The atrophy is almost always unilateral and is caused by hemorrhage, inflammation or new growth within the orbit.

B. The atrophy is almost always bilateral and is not caused by disease within the orbits.

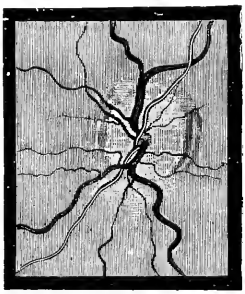
1. Confirmatory signs of the existence of organic disease of the central nervous system (such as paralysis of the ocular muscles, face, tongue or one side of the larynx, paralysis of the limbs that cannot be explained as functional, Jacksonian epilepsy, typical ankle clonus, absence of knee-jerk, Argyll-Robertson pupil, coincident mental impairment, etc.) are absent. Although only rarely found apart from other evidence

of organic nervous disease, the atrophy may be explained as the effect of a systemic condition, an inherited influence, or a severe neuralgia, or migraine, or as the first symptom of an organic disease.

a. The blood is very greatly impoverished as in severe simple anemia, pernicious anemia or leucocythemia.

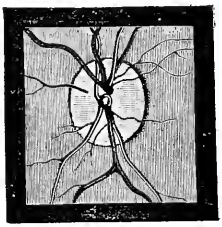
b. There is a systemic infection, as in variola, scarlatina, severe malaria, diphtheria, typhus fever, typhoid fever or syphilis.

FIG. 23.



ATROPHY OF DISC AFTER PAPILLITIS.
(*From Ormerod, after Nettleship.*)

FIG. 24.



ATROPHY OF DISC FROM SPINAL DISEASE.
Lamina Cribrosa Concealed, Vessels Normal.
(*From Ormerod, after Nettleship and Wecker.*)

c. There is a condition of severe toxemia, as in uremia, gout, diabetes, alcoholism, plumbism, hydrargyrism, chorea or poisoning by tobacco or carbon disulphide.

d. The atrophy follows long-continued and severe ophthalmic neuralgia or migraine. Very rare.

e. The atrophy occurs in youths or young adults of certain families with no apparent cause except hereditary influence. Very rare.

f. The atrophy occurs without any associated symptoms or evidence that it is hereditary.

Probably Tabes or Parctic Dementia, of which other symptoms will appear later.

2. Confirmatory signs of the existence of organic disease of the central nervous system are present.

a. The cranium is injured or diseased in such a way as to interrupt fibers of the optic nerves or optic tracts, as in fracture at the base or chronic thickening of the bones.

b. There is no evidence that the cranium is primarily affected; the lesion is intracranial or spinal.

i. The disease is congenital or develops gradually in infancy. There is globular enlargement of the cranium. *Hydrocephalus.*

ii. The onset of the disease which causes the atrophy is sudden and—

§ Coincident with a blow on the head and loss of consciousness.

Cerebral Concussion.

§§ Without external violence. Hemiplegia occurs as consciousness is lost.

! Age usually less than 40. There is a septic endocarditis or a pulmonary abscess. *Cerebral Embolism.*

!! Age usually more than 40. The arteries are degenerated. There may be evidence of increased blood pressure at the time of onset.

Cerebral Hemorrhage.

iii. The onset of the disease which causes the atrophy is gradual and slow.

§ The knee-jerks are lost and the Argyll-Robertson pupil is present. A history of lightning pains in the legs is common, also of transient diplopia, ptosis, loss of sexual power or difficulty in voiding or retaining the urine. Ataxia of station and gait is usually present in some degree but is especially apt to be slight in cases where optic atrophy occurs early. This disease causes simple optic atrophy more frequently than all the other causes combined.

Tabes.

§§ Failure of judgment and memory, often combined with monstrous and unsystematized delusions of grandeur, accompanies the signs of organic disease, among which inequality of pupils and stumbling speech are common.

Paretic Dementia.

§§§ Intention tremor with nystagmus, transient spastic paralysis or scanning speech is present. Knee-jerks usually exaggerated. Optic atrophy slight.

Disseminated Sclerosis.

§§§§ The course of the disease is marked by severe headache, often accompanied by vomiting and vertigo. Pulse often slow. The state of the visual fields and other localizing symptoms indicate a progressive lesion affecting one optic nerve or the chiasm. *Intracranial Tumor.*

§§§§§ Optic atrophy may occur in lateral sclerosis and in bulbar paralysis but so rarely that the mere mention of the possibility is sufficient.

TROPHIC AND VASO-MOTOR SYMPTOMS.

I. There is atrophy of one or more muscles.

A. The atrophied muscles are those which move a diseased joint and the atrophy is secondary to more or less fixation of the joint. Faradic irritability usually retained, but may be diminished.

Arthritic Muscular Atrophy.

B. The muscles are wasted independently of joint disease or disuse and are paralyzed. Faradic irritability almost always lost or diminished and reaction of degeneration is common when the wasting is rapid.

Diagnosis as in Paralysis.

II. There is hypertrophy or atrophy of bones.

A. There is a gradual and symmetrical enlargement of the hands and feet, including the bones and soft parts. The fingers are sausage-shaped and the nails broad and flat. The lower and upper jaw, orbital ridges, nose, tongue and ears also become enlarged, causing a characteristic deformity of the face. Headache and ocular symptoms very common. Patient an adult, more frequently a woman. Autopsy and, in many cases, the symptoms during life reveal intracranial disease affecting the pituitary body. *Acromegaly.*

B. The bones of the hands and feet, especially the terminal phalanges, are enlarged. The fingers and toes are excessively clubbed and the nails much curved. The ends of the long bones are also enlarged and the joints are involved but the face and skull are not affected. The patient is an adult, more frequently a man, with chronic pulmonary disease. There is nothing to indicate intracranial disease. Very rare.

Hypertrophic Pulmonary Osteo-arthropathy.

C. The bones of the cranium and face are enlarged. Various cranial nerve symptoms may occur, owing to pressure or obstruction of foramina. Disease usually begins in childhood or at puberty. Inferior maxilla and extremities not affected. Called *Leontiasis ossea* by Virchow. *Megalocephalie* by Starr. Very rare.

Hyperostosis Cranii (*Putnam*).

D. There is progressive atrophy of the bones and soft parts of one side of the face. Hair and teeth fall out. Very rare. *Facial Hemi-atrophy.*

III. There is trophic disease of one or more joints. The cartilages are eroded and the ends of the bones wasted or perhaps enlarged by irregular bony deposits. There may be edema around the joint and copious effusion into it.

A. The knee-jerks are absent. A history of lightning pains in the legs is common, also of loss of virility and slight difficulty in voiding or retaining the urine. The patient sways on standing with eyes closed and in the later stages the gait is ataxic. Argyll-Robertson pupil in most cases. *Tabes.*

B. There is loss of sensibility to pain and temperature in areas where touch is retained. There is usually paralysis of somewhat variable and irregular distribution, most commonly atrophic in the arms and spastic in the legs. *Syringomyelia.*

C. The joint disease is on the same side as hemiplegia and secondary to it. No other cause.

Hemiplegic Arthropathy.

D. The joint disease is secondary to paraplegia which is accompanied by loss of control of the bladder and rectum and more or less sensory loss, indicating some form of myelitis. *Myelitic Arthropathy.*

IV. The disturbance is in the skin and subcutaneous tissue without marked involvement of the bones.

A. Sloughing occurs in a paralyzed part, especially where the skin is subjected to pressure or the irritation of heat or a blister. *Diagnosis as in Paralysis.*

B. A painless, indolent ulcer appears in the sole of the foot or, very rarely, in the hand. It resists treatment and tends to penetrate deeply. Absence of knee-jerk and various other symptoms indicate the existence of tabes.

Perforating Ulcer in Tabes.

C. The disturbance is in the peripheral distribution of certain nerves (most frequently the musculo-spiral and external popliteal) which are generally tender. The skin is at first reddened and gradually becomes smooth and glossy. When the hand is affected the fingers become pointed. Adhesions may form in the joints. There is numb, stinging pain, usually quite severe and more or less sensory loss in the affected area. The corresponding muscles are paralyzed and tender and waste and lose their faradic irritability. A local or general cause of neuritis is present. *Neuritis.*

D. Vesicles appear in the area supplied by one or more spinal segments or in one or more of the cranial sensory areas described by Head. (Figs. 25, 26, 27, 28 and 29.) Numb, stinging, generally very severe pain is felt in the same area. The vesicles ulcerate and leave scars.

Herpes Zoster. A symptom of irritation of the posterior root ganglia or of the Gasserian ganglion.

E. The skin and subcutaneous tissue throughout the body gradually become thickened. The swelling is firm and does not pit on pressure. The face has a coarse, puffy, round appearance and the hair is thin. The mind becomes dull, simulating dementia. The thyroid gland is always diseased and its function diminished but it may be either atrophied or enlarged. Administration of thyroids causes marked improvement. The disease may be congenital in goitrous dwarfs especially in Switzerland and other mountainous countries (thyroid cretinism), or it may be acquired in adult life, six women being affected to one man, or it may be caused by operative removal of the thyroid (cachexia strumiprava).

Myxedema.

F. There is arrest of circulation, usually in the extremities (fingers, toes, nose, ears) and symmetrical, but it is sometimes unsymmetrical and may affect the trunk and proximal parts of limbs. The disease begins with local pallor and a feeling of icy coldness (local syncope), which, after a variable time, is followed by cyanosis (local asphyxia) and later by gangrene. In the cyanotic and gangrenous stages there may be intense pain.

Raynaud's Disease.

G. The skin, either generally or locally, is first thickened and hardened, later wasted, appearing to be stretched over and bound down to the underlying parts. The hands (sclerodactylie) and face are most often affected. Motion may be impeded by the hardened skin. Localized patches probably correspond to the distribution of spinal segments or nerve trunks. The disease generally begins in youth or early adult life, three-fourths of the patients being females. It may be associated with Raynaud's disease. *Scleroderma.*

H. There are destructive trophic changes in the fingers, especially in the terminal phalanges, like those of a felon, but painless. Neuralgic pains may occur independent of the trophic changes. Sensibility to temperature and pain is lost in areas where touch is retained. Various other cord symptoms, especially paralysis of the type found in amyotrophic lateral sclerosis, may confirm the diagnosis of syringomyelia.

Morvan's Disease, a special form of syringomyelia.

I. There are symmetrical fatty tumors in the subcutaneous tissue which are tender and painful. Patients usually alcoholic or syphilitic. *Adiposis Dolorosa (Dercum).*

J. There are subcutaneous and perhaps submucous swellings affecting principally the face, lips, tongue, pharynx, genitals and limbs. The swelling is well defined, tense, not tender, does not pit on pressure and may be either white or pink. It comes on rapidly, in a few minutes to a few hours and after lasting hours or days may

rapidly disappear, recurring at regular or irregular intervals. Gastro-intestinal pains are common. Neuropathic heredity is very marked in many of the cases.

Angioneurotic Edema.

K. There is pain in one foot, very rarely in both feet or in a hand, associated with redness and often with swelling and increased local temperature, sometimes with hyperhidrosis and local hemorrhages. All the symptoms tend to subside when the patient reclines and rests the foot in an elevated position; they are also relieved by cold and aggravated by warmth. Standing and letting the foot hang brings on a paroxysm of pain accompanied by a rose-red flush and arterial throbbing; there is no cyanosis or gangrene. Occurs almost exclusively in men, either alone or in association with various other nervous affections. *Erythromelalgia.*

L. There is an edematous swelling of a limb which is hysterically paralyzed, or contractured, or exhibits the hysterical simulation of joint disease. The swelling does not pit on pressure and is greatest in the morning. The part may be unchanged in color and temperature, or may be red and warm, but is mostly cyanotic and cold.

Hysterical Edema.

V. There is a general vaso-motor dilatation with rapid heart action and, in most cases, goitre and protrusion of the eyeballs. The carotids pulsate strongly, there is a characteristic thrill and murmur in the enlarged thyroid and the heart is often dilated. The skin is moist and diarrhea is common. A fine, rapid tremor of the hands is almost always to be observed. Mental changes (irritability, mania, melancholia) may occur. Far more common in women than in men. The disease is apparently dependent upon excessive function of the thyroid gland, so it may be regarded as the opposite of myxedema.

Exophthalmic Goitre.

THE PAINS OF NERVOUS DISEASE.

I. Pain definitely referred to the distribution of one or more nerves.

A. Accompanied by signs of organic disease of the nerve, such as a tumor or tumors on the nerve trunk or nerve endings, atrophic paralysis, loss of tendon reflexes or of faradic irritability, tenderness of the nerve and the muscles supplied by it, sensory loss in the distribution of the nerve, glossy skin, etc. Pain of a peculiar stinging or burning character and usually persistent in the interval between exacerbations.

1. No local cause of neuritis, such as wound or pressure. A tumor or tumors may be felt on the nerve trunk or nerve endings. *Neuroma.*
2. A single nerve is affected and a local cause of neuritis, such as pressure, stretching, a wound or local infection, is apparent. *Localized Neuritis.*
3. The areas affected are bilateral and symmetrical, especially in the distribution of the musculo-spiral and external popliteal nerves. A toxic cause, such as alcoholism, arsenical poisoning, general infection or exposure with extreme exertion is present. *Multiple Neuritis.*

B. No proof of organic disease of the nerve, although the skin supplied by it and certain definite points along its course may be tender. Pain paroxysmal, usually with intervals of complete freedom. Toxemia, anemia or general nervous depression and some local irritation the most common causes. *Neuralgia.*

II. Headache. Pain in the head not definitely referred to the distribution of particular nerves.

A. There is organic disease of the brain or its membranes,

shown, not merely by the severity and persistence of the headache, but also by the occurrence of some more positive sign, *e. g.*, paralysis of ocular muscles, face, tongue or one side of larynx; inequality of pupils or failure of light reaction; optic neuritis or optic atrophy; typical ankle clonus; Jacksonian epilepsy, etc. If delirium or stupor occurs headache still continues. Vomiting common.

1. Onset sudden or very rapid, in a few minutes to a day, following an injury or occurring in an aged or insane person, without fever or other evidence of infection. Rigidity, perhaps convulsions, followed by paralysis, on the side opposite the headache. Sopor and coma with slowness of pulse supervene.

   Meningeal Hemorrhage.

2. Onset acute or subacute, in a few hours to a few weeks. Delirium, general or local convulsions and cranial nerve symptoms (such as ptosis, strabismus, inequality or immobility of pupils, paralysis or twitching of face, etc.) common. Optic neuritis occasional, rarely intense.

   a. Patient, more commonly a child or an old person, depressed by an exhausting disease. Temperature normal or slightly elevated. Venous distension and edema of the forehead and sides of the head or of the eyelids and temple.

      Marantic Sinus Thrombosis.

   b. A source of intracranial infection or irritation is present, *e. g.*, otitis media, tuberculosis, pneumonia, epidemic influence, wound of the head, sunstroke, etc. Fever attends the onset; afterward the temperature is irregularly elevated or, at times, depressed. Retraction of the head often a prominent symptom. Cranial nerve symptoms common. Optic neuritis occasional, rarely intense. General hyperesthesia present in the early stage, followed by delirium which, in severe

cases merges into stupor and coma. Whole course from onset to death or convalescence short, a few hours to a few weeks.

Meningitis, rarely Encephalitis or Abscess.

i. The source of infection is purulent otitis media, which is followed by signs of mastoid disease. Then come rigors, rapid and extreme rises of temperature with equally rapid falls and profuse sweating, indicating pyemia. The obstructed internal jugular vein may sometimes be felt in the neck as a cord.

Thrombosis of Lateral Sinus. (May be associated with meningitis or abscess.)

3. Onset rapid or slow, in a few days to weeks or months. Source of purulent infection present, *e. g.*, otitis media, empyema, abscess in any part of the body, infected wound of the head. Rigors, followed by fever and sweating common. Temperature irregular, usually elevated but sometimes depressed. Optic neuritis frequent but rarely intense. In comparison with meningitis focal cerebral symptoms are common and cranial nerve symptoms uncommon. Duration may be short or it may be very long with a period of latency. *Intracranial Abscess.*

4. Onset slow. No signs of suppuration. Temperature normal or nearly so. Headache usually intense, often accompanied by vertigo and vomiting. Optic neuritis in four-fifths of all cases, often intense. Pulse often slow. Course usually long and for the most part steadily progressive with mental failure toward the end. Convulsions or any form of focal or cranial nerve symptom may occur. Inherited or acquired predisposition to new growths sometimes apparent.

Intracranial Tumor, Aneurism or Cyst.

5. Onset slow, without fever and without intense optic neuritis or slow pulse. Patient alcoholic or syphi-

litic. Organic disease indicated by spasm or paralysis in the domain of cranial nerves, rarely in the limbs. *Chronic Meningitis.*

B. Positive signs of disease of the brain or membranes absent but there is evidence that the cerebral vessels are diseased.

1. The patient is past forty. The arteries are atheromatous and pulse hard. Headache is throbbing and is increased by exertion or excitement. Heart often hypertrophied and some albumen is usually found in the urine. *Arteriosclerosis.*
2. Headache chiefly nocturnal and accompanied by insomnia. Age and general condition exclude senile degeneration. Syphilis can not be excluded and is usually manifested by some of its characteristic signs. Premonitions of focal symptoms and rapid mental deterioration, without other assignable cause may show that the cerebral circulation is greatly disturbed. *Syphilitic Endarteritis.*

C. Headache follows a blow on the head. No positive signs of organic disease. *Traumatic Headache.*

D. The headache forms part of the periodic attack of migraine or epilepsy. Complete freedom between attacks. Family history of similar attacks or of other neuroses common.

1. Pain unilateral, at least at first, and accompanied by nausea and intolerance of light or noise, often by partial darkening of the field of vision or subjective sensations of light or color. Often complicated by hysteria. *Migraine.*
2. Headache precedes or follows an epileptic convulsion or an attack of petit mal. *Epilepsy.*

E. There is no organic disease of the brain, membranes or vessels and the headache is not traumatic nor a part of a periodic neurosis.

1. Headache appears at the same time as fever and usually disappears if delirium supervenes. There is

evidence of a general infection, usually one of the specific fevers. Meningitis may sometimes be closely simulated, especially as the pupils may be unequal (*e. g.*, in pneumonia or tonsillitis), the head retracted (*e. g.*, in typhoid fever), or even optic neuritis be present in very rare cases, but other indications of organic disease are absent. *Infectious Fever.*

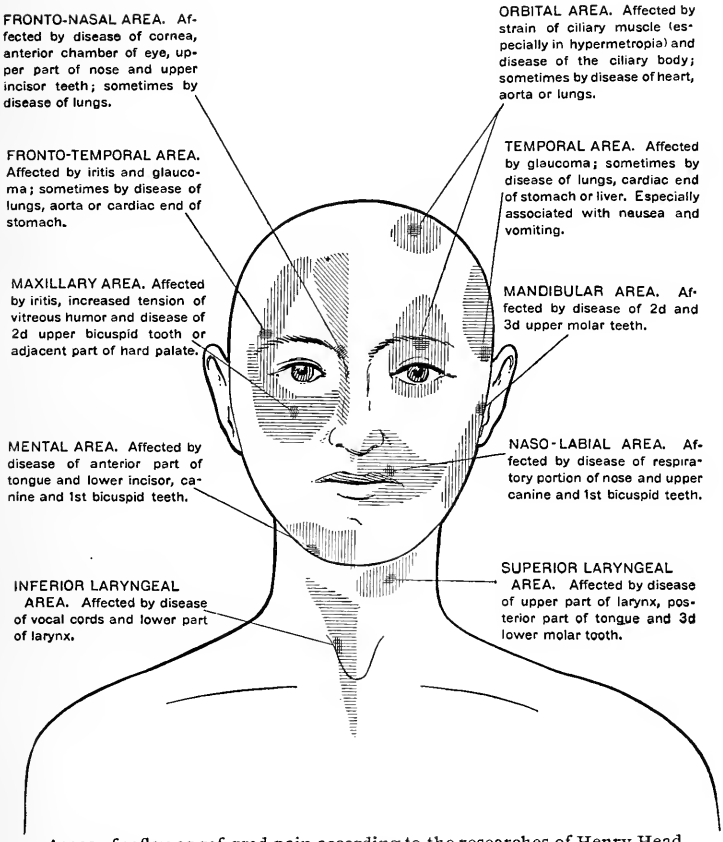
2. A toxic substance is active in the system. Pain may be diffuse and dull or in definite areas and of a neuralgic character.
   a. The urine contains sugar and the patient suffers from polyuria, thirst, weakness, etc. *Diabetes.*
   b. The urine contains albumin or casts or its specific gravity and total quantity indicate deficient elimination of urea. Pallor and edema are often manifest and there may also be a characteristic retinitis. *Uremia.*
   c. Urine free from albumin and sugar, but at times is dark and deposits urates. Headache worse in early morning and tends to wear off during the day; often accompanied by depression of spirits. Uric acid accumulations indicated by attacks of rheumatism, gout, tonsillitis, gravel, etc. *Uric Acid Diathesis.*
   d. History of excessive consumption of alcohol. There may be gastric catarrh, morning vomiting, characteristic odor of breath, tremor, insomnia, etc. *Alcoholism.*
   e. Digestion is perverted, as shown by abdominal distress, eructations, flatulence or other symptoms. Headache is usually dull, comes on soon after eating or is a sequence of eating some particular food, and is temporarily relieved by purgatives and intestinal antiseptics. To be distinguished from the reflex pain of gastric or intestinal disease. *Indigestion.*
   f. Patient has been exposed to lead which may per-

haps be found in the urine. There may be the blue line on the gums, colic with constipation or bilateral wrist drop. *Plumbism.*

3. No infective or toxic cause but the cerebral circulation is disturbed.
   a. Headache throbbing, follows excitement or exertion and may be associated with a flushed face or injection of the conjunctivæ. Increased by coughing, sneezing, straining or lowering the head. *Active Hyperemia.*
   b. Headache dull and heavy. Return of venous blood obstructed by mitral regurgitation, tumor of the neck, constriction or other cause. *Passive Hyperemia.*
   c. Heart action weak or blood impoverished, as shown by pallor of skin and mucous membranes, faintness, low percentage of hemoglobin, paucity of red corpuscles, etc. Pain may be of a neuralgic character in definite areas. *Anemia.*
4. No infective, toxic or circulatory cause but there is a reflex disturbance. The pain, accompanied by superficial tenderness, is felt in definite areas and is generally called neuralgia.
   a. There is an error of refraction with overwork of the ciliary muscle, disease of the eye-ball or rarely a loss of muscle balance alone. The pain is mainly in the orbital region (*e. g.*, strain of ciliary muscle), the fronto-temporal region (*e. g.*, cyclitis) or the temporal region (*e. g.*, glaucoma), sometimes back of the eye. In strain of the ciliary muscle the pain appears in the morning, is aggravated by near work and is relieved by rest of the eye. Often a complication of neurasthenia. *Ocular Headache.*
   b. The pulp of one or more teeth is diseased or the teeth are crowded by a faulty eruption. Reflex pain is felt in the naso-frontal area (upper in-

cisors), the temporal area (second upper bicuspid) or one or more of the other pain areas of the face and neck, according to the tooth affected.

FIG. 25.



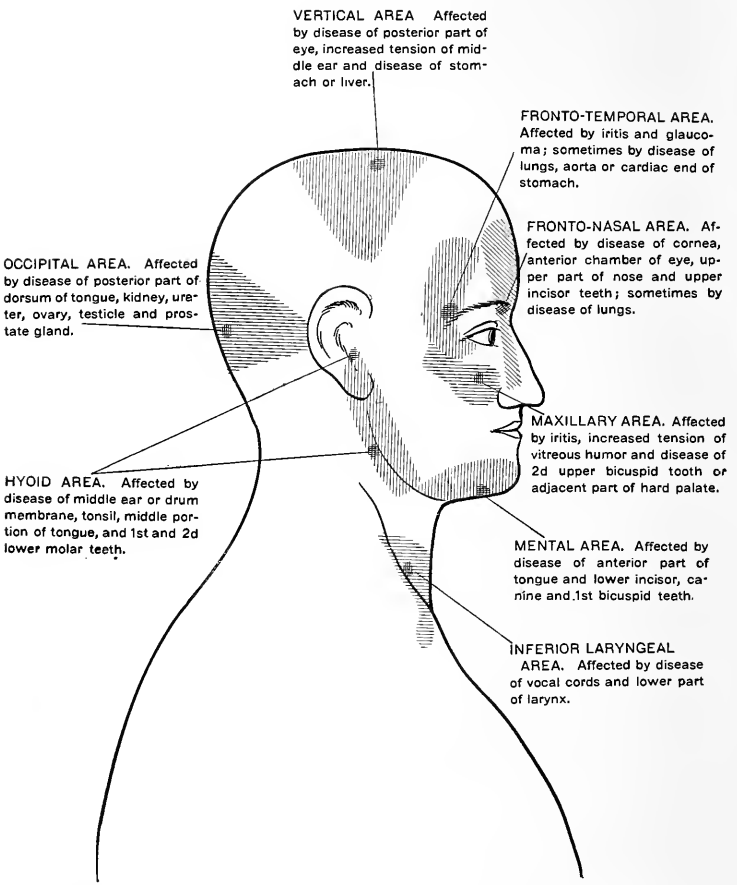
Areas of reflex or referred pain according to the researches of Henry Head.

Generally described as neuralgia, although the areas of pain do not strictly correspond to the distribution of individual nerves. *Dental Headache.*

c. There is disease of the tympanic cavity or drum

membrane. The pain is most intense in the ear and back of the angle of the jaw, but may extend

FIG. 26.



Areas of reflex or referred pain according to the researches of Henry Head.

over the vertical and parietal areas.

Aural Headache.

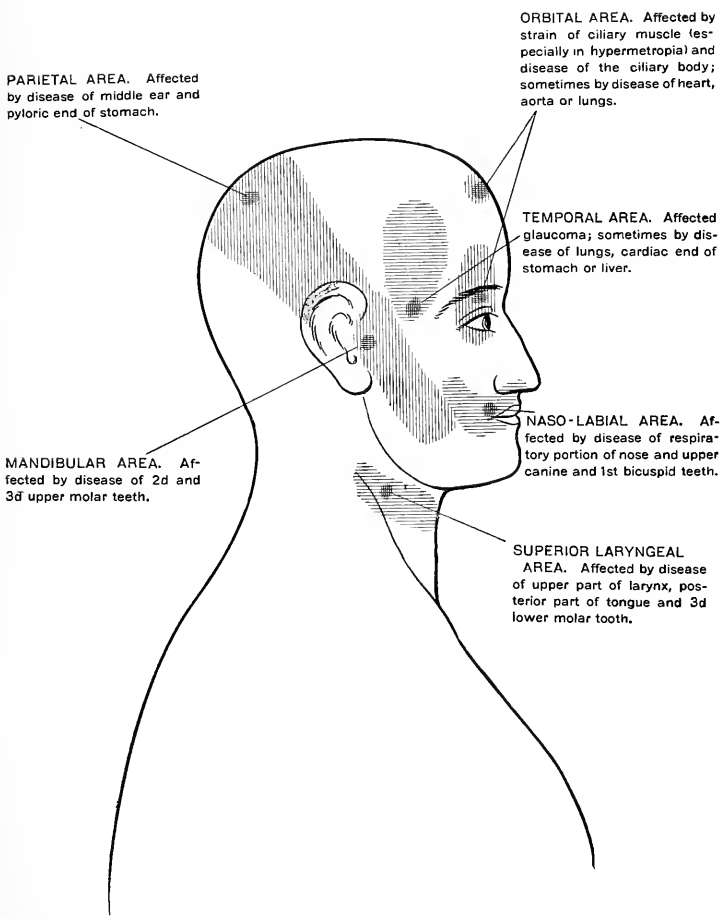
i. Purulent otitis is followed by mastoid disease and then by signs of pyemia. The obstructed

jugular vein may sometimes be felt in the neck as a cord.

Thrombosis of Lateral Sinus. (*May exist with or without meningitis or abscess.*)

d. The pain is felt in the fronto-nasal or orbital area

FIG. 27.



Areas of reflex or referred pain according to the researches of Henry Head.

and is associated with disease of the nose or its adjacent sinuses, increasing or diminishing with it. Headache may sometimes be markedly increased by touching the middle turbinated bone with a probe and relieved by the application of cocaine. Rare. *Nasal Headache.*

e. The headache is added to pain and tenderness in the sensory areas of certain spinal segments (Figs. 28 and 29) as a secondary reflex pain, due to disease of some of the viscera within the trunk.

i. Aortic regurgitation, aortic aneurism or mitral stenosis with regurgitation is present. Headache in the forehead or temple, mainly on the left side. Pain and tenderness in some of the areas of the first six dorsal segments, often subject to severe exacerbations (angina pectoris). *Cardiac Headache.*

ii. Active pulmonary disease exists. Headache may be in any part of the cranium, except the occiput, mainly on the side of the disease. Pain and tenderness in some of the areas of the first seven dorsal segments. *Pulmonary Headache.*

iii. A painful disease of the stomach exists. Headache in the temporal, vertical or parietal region. Pain and tenderness in some of the areas of the sixth to ninth dorsal segments. *Gastric Headache.*

iv. An irritating disease of the intestine exists. Headache parietal or occipital. Pain and tenderness in some of the areas of the ninth to twelfth dorsal segments. *Intestinal Headache.*

v. There is disease of the liver or its appendages. Headache temporal, vertical, parietal or occipital. Pain and tenderness in some of the areas of the seventh to tenth dorsal segments. *Hepatic Headache.*

vi. There is a painful disease of the kidney or ureter, most frequently calculus. Headache occipital. Pain and tenderness in some of the areas of the tenth to twelfth dorsal segments.

Renal Headache.

vii. There is disease of the prostate. Headache occipital. Pain and tenderness in some of the areas of the tenth to twelfth dorsal, first lumbar or first to third sacral segments.

Prostatic Headache.

viii. The ovary or testicle is diseased. Headache occipital. Pain and tenderness in area of tenth dorsal segment.

Ovarian or Orchitic Headache.

5. No infective, toxic, circulatory or reflex cause apparent.

a. Nervous energy exhausted from any cause, particularly by prolonged worry with overwork or other excess combined with impaired nutrition. The patient may for a short time exert normal mental and bodily powers, but soon becomes fatigued and irritable. Sensations in the head are often described as queer and disagreeable rather than painful, such as tightness or looseness of the scalp, lightness, heaviness or increased volume of the head, inability to think, etc. A lack of zest for ordinary affairs and morbid fears are common. Painful and superficially tender spots are usually found along the spine as well as in the head.

Neurasthenia.

b. Headache appears and disappears in accord with emotional changes or in response to suggestion. Often limited to a small spot at vertex or in temple (clavus). Various signs of hysteria may be present. *Hysteria.*

III. Spinal pain. Pain felt in or near the spinal column, often extending into the sensory areas supplied by the corresponding segments of the spinal cord.

A. Accompanied by signs of organic spinal disease, such as deformity of the spine; absence of knee-jerk; typical clonus; degenerative atrophy of muscles with loss of faradic irritability (without signs of neuritis); the combination of paraplegia with more or less sensory loss, the upper limit of both corresponding to the function of a spinal segment; impaired control of the bladder, etc.

1. Paraplegia, more or less sensory loss and impaired control of the bladder and rectum appear early. The upper limit of the motor and sensory loss corresponds to the function of a segment of the spinal cord and is often marked by a zone of hyperesthesia immediately above it. Spinal rigidity and radiating pains generally absent.

   a. Onset sudden.

      i. Simultaneous with severe injury to the spinal column.

         Fracture or Dislocation of Vertebræ, Wound of or Hemorrhage into the Spinal Cord.

      ii. Without external violence.

         Hemorrhage into Cord.

   b. Onset gradual, acute or chronic. Vertebræ not diseased. Pain dull, not a prominent symptom.

      Myelitis.

2. Spinal rigidity accompanies pain. Corresponding radiating pains common. If paraplegia, sensory loss and impaired control of the bladder and rectum occur, it is later in the course of the disease.

   a. Deformity or swelling and deep-seated tenderness indicate disease of the vertebræ. Paraplegia, sensory loss and impaired control of bladder and rectum eventually occur unless the disease is arrested.

      i. Patient most commonly a child, sometimes a young adult, rarely an elderly person. The tubercular diathesis is generally manifest but very rarely syphilis may be the cause. Pain

FIG. 28.

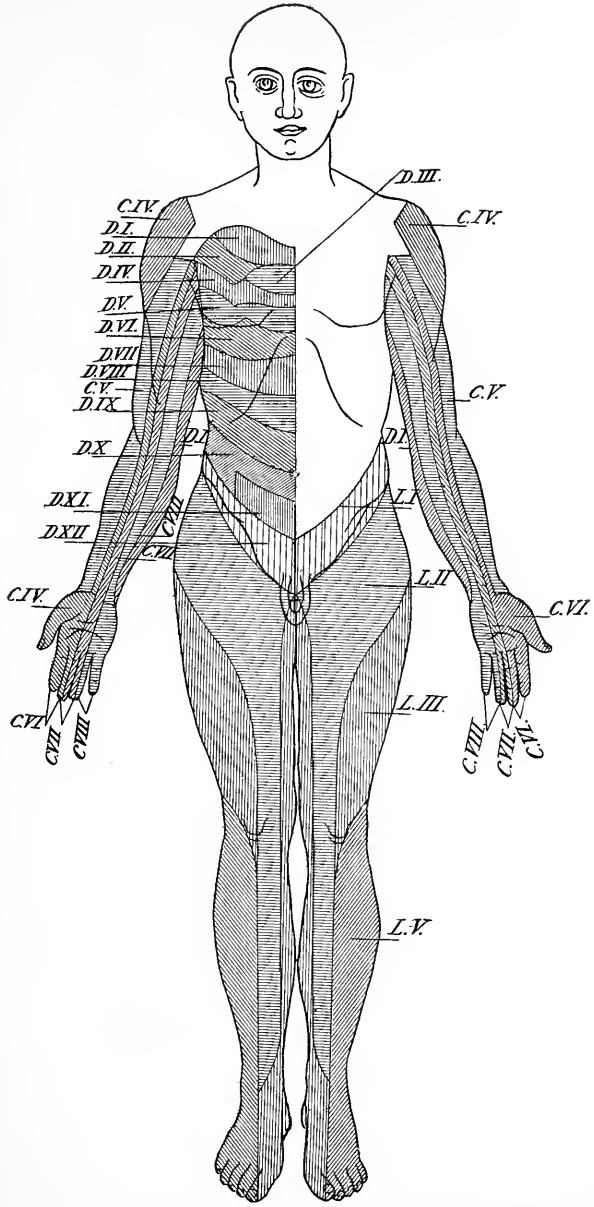


DIAGRAM OF SKIN AREAS CORRESPONDING TO DIFFERENT SPINAL SEGMENTS
(*From Tyson, after Starr. Trunk Areas from Head.*)

FIG. 29.

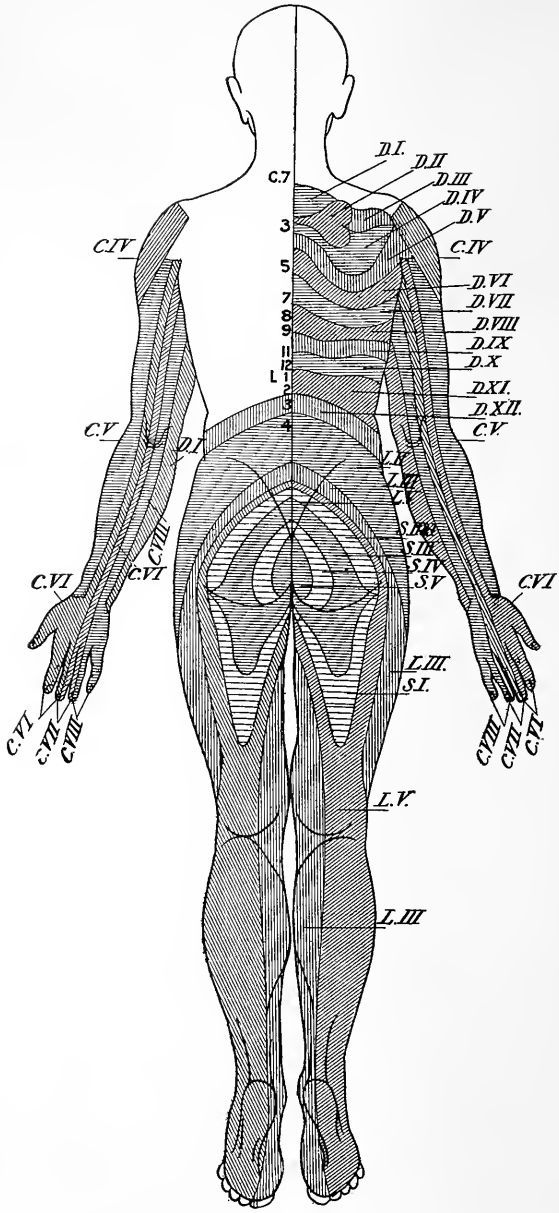


DIAGRAM OF SKIN AREAS CORRESPONDING TO DIFFERENT SPINAL SEGMENTS. Arabic Numerals refer to Vertebræ. (*From Tyson, after Starr. Trunk Areas from Head.*)

usually of moderate severity, increased by motion or jars and diminished by rest of the spine. Prominence or lateral displacement of one or more spinous processes is the characteristic deformity. *Spinal Caries.*

ii. Patient generally in the second half of life, sometimes with a history of tumor elsewhere or of predisposition to new growths or aneurism. The pain is very intense and is very greatly aggravated by motion.

§ The radiating pain is on the left side of the chest. Characteristic thrill and murmur at the seat of pain and deformity. *Aneurism Eroding Spine.*

§§ Radiating pain usually on both sides. No thrill or murmur. *Spinal Tumor.*

b. Localized spinal pain and rigidity gradually occur, most frequently in the cervical region, in a patient predisposed to arthritis deformans, evidence of which may be apparent in other parts of the body. Radiating pains and other root symptoms may occur but are not followed by paraplegia or other cord symptoms. Rare. *Vertebral Arthritis Deformans.*

c. Nothing to indicate disease of the vertebræ.

i. Onset sudden. No fever at first. *Spinal Meningeal Hemorrhage.*

ii. Onset acute, marked by chill and fever. *Acute Spinal Meningitis.*

iii. Onset chronic.

§ History of alcoholism, syphilis or exposure to cold. *Chronic Spinal Meningitis.*

§§ Evidence of predisposition to new growth. No other cause of meningitis. *Intraspinal Tumor.*

B. Not accompanied by signs of organic spinal disease.

1. Pain and superficial tenderness in the back and in the

sensory areas of corresponding segments appears in connection with visceral disease and increases or diminishes with it, constituting the referred or reflex pain of visceral disease. The correspondence of the painful areas to the particular organ diseased is shown in the following table, which is taken from the work of Dr. Henry Head. It must be remembered, however, that febrile and toxic conditions without demonstrable localized disease may cause the same areas to become painful. Often called neuralgia.

ORGAN DISEASED.	SPINAL SEGMENTS WHOSE SENSORY AREAS ARE PAINFUL.	CRANIAL AREAS IN WHICH PAIN MAY ALSO BE FELT.
Heart,	1, 2, 3 dorsal (angina pectoris),	Orbital.
Lungs,	3, 4 cervical, 1, 2, 3, 4, 5 dorsal,	Fronto-nasal, orbital, fronto-temporal, temporal.
Ascending Aorta,	1, 2, 3, 4 dorsal,	Orbital, fronto-temporal.
Arch of Aorta,	5, 6 dorsal,	Fronto-temporal.
Stomach, cardiac,	6, 7 dorsal,	Fronto-temp., temporal.
Stomach, pyloric,	8, 9 dorsal,	Vertical, parietal.
Liver and appendages,	7, 8, 9, 10 dorsal,	Temporal, vertical, parietal, occipital.
Intestine,	9, 10, 11, 12 dorsal,	Parietal occipital.
Kidney and ureter,	10, 11, 12 dorsal,	Occipital.
Prostate,	10, 11, 12 dorsal, 5 lumbar, 1, 2, 3 sacral,	Occipital.
Ovary or testicle,	10 dorsal,	Occipital.
Rectum,	2, 3, 4 sacral.	
Epididymis,	11, 12 dorsal.	
Oviduct,	11, 12 dorsal, 1 lumbar.	
Bladder, mucous membrane and neck,	1, 2, 3, 4 sacral.	
Bladder, over-distension and ineffectual contraction,	11, 12 dorsal, 1 lumbar.	
Uterus, in contraction,	10, 11, 12 dorsal, 1 lumbar.	
Uterus, os,	1, 2, 3, 4 sacral (5 lumbar very rarely).	

2. The pain is most frequently in the lumbar, sometimes in the dorsal or cervical region, and is generally worse in the morning, tending to wear off during the day. There is evidence of a rheumatic condition, such as rheumatism in other parts of the body, a history of previous rheumatic attacks, marked variations corresponding to changes in the weather, the alternation of scanty and excessive elimination of urates, great relief from the administration of salicylates, etc. Not a nervous disease, but included here because it must often be carefully distinguished from more serious diseases which are likely to be mistaken for it. *Rheumatism.*

3. There are moderately painful and superficially tender spots along the spine and often in the head. Nervous energy is exhausted from some cause, usually by prolonged worry with overwork or other excess combined with impaired nutrition. The patient may for a short time exert normal mental and bodily powers but soon becomes fatigued and irritable. Queer sensations in the head are often complained of, such as tightness or looseness of the scalp, lightness, heaviness or increased volume of the head, inability to think, etc. Morbid fears and a lack of interest in ordinary affairs are common.

Neurasthenia.

4. The pain comes and goes in accord with emotional changes or in response to suggestion. Although it and the accompanying superficial tenderness may appear to be intense, both disappear or are greatly diminished when attention is strongly engaged by something else. Various other signs of hysteria may be present. Often combined with neurasthenia.

Hysteria.

IV. Pain felt in the trunk or extremities not accompanied by corresponding spinal pain nor definitely referred to the distribution of certain nerve trunks.

A. There are signs of organic disease of the brain or cord.

1. The patient has suffered a cerebral vascular lesion, most frequently softening in the region of the basal ganglia and the pain is due to irritation of the sensory tract. *Post-hemiplegic Pain.*

2. The pain consists of lightning pains in the lower limbs or trunk, or has the character of "crises" (gastric, laryngeal, vesical, rectal, etc.). Absence of knee-jerk with Argyll-Robertson pupil, ataxia or urinary difficulty makes the diagnosis clear. *Tabes.*

3. Pain may be like that of neuralgia or that of tabes. There is loss of sensibility to temperature and pain with preservation of touch. More or less paralysis occurs, usually atrophic in the arms and spastic in the legs, together with various trophic symptoms, the whole group of symptoms being such as might be caused by chronic disease mainly affecting the gray matter of the cord. *Syringomyelia.*

B. There are no signs of organic disease of the brain or cord.

1. Pain caused by arrest of circulation, which is usually in the extremities (fingers, toes, nose, ears) and symmetrical, but is sometimes unsymmetrical and may affect the trunk and proximal parts of limbs. The disease begins with local pallor and a feeling of icy coldness (local syncope), which, after a variable time, is followed by cyanosis (local asphyxia) and later by gangrene. The symptoms are aggravated by cold and relieved by warmth. In the cyanotic and gangrenous stages the pain may be intense.

Raynaud's Disease.

2. Pain in one foot, very rarely in both feet or in a hand, associated with redness and often with swelling and increased local temperature, sometimes with hyperhidrosis and local hemorrhages. All the symptoms tend to subside when the patient reclines and supports the extremity in an elevated position; they

are also relieved by cold and aggravated by warmth. Standing erect and letting the extremity hang brings on paroxysms of pain accompanied by a rose-red flush and arterial throbbing; there is no cyanosis or gangrene. Occurs mostly in men either alone or in association with various other nervous affections.

Erythromelalgia.

3. The pain comes and goes in accord with emotional changes or in response to suggestion. Although it and the accompanying superficial tenderness may appear to be intense, both disappear or are greatly diminished when attention is strongly engaged by something else. Various other signs of hysteria may be present. Often combined with neurasthenia.

Hysteria.

a. The pain is felt in a joint, most commonly the hip, knee, shoulder or ankle, and the complaints are out of all proportion to any objective signs of disease. Night startings, involuntary cries and facial signs of severe suffering are absent. The joint may be held in a fixed position by spasm of its muscles, but this spasm relaxes more or less during sleep and may often be entirely overcome by persistent moderate force if the patient's attention can be distracted. The muscles are not wasted more than disuse would account for and their electrical irritability is normal. The local temperature is generally normal or sub-normal; rarely it is temporarily elevated, never persistently so. The posture often differs widely from that of greatest ease and in many cases automatic actions which would cause great pain in real joint diseases are performed without complaint. The symptoms often change greatly within a short time. Examination under anesthesia reveals no sign of joint disease.

Hysterical Simulation of Joint Disease.

VERTIGO.

The patient has a false sense of motion of his own body or of the objects about him. The sensation may be so strong as to cause actual motion of the body in the same direction.

I. There is coarse organic disease of the brain or its membranes, generally shown by headache and vomiting (not dependent on the vertigo), together with such symptoms as hemiplegia; paralysis of ocular muscles, face, tongue or one side of larynx; inequality of pupils or failure of light reaction; optic neuritis or optic atrophy; typical ankle clonus; Jacksonian epilepsy; coincident mental impairment, etc. The localizing symptoms generally indicate the cerebellum, pons or quadrigeminum as the seat of lesion.

Vertigo of Organic Intracranial Disease, special diagnosis to be made as in cases of hemiplegia, optic neuritis, headache, etc.

II. There is degenerative disease of the central nervous system, shown by its very slow onset and the presence of such symptoms as ataxia, intention tremor, ocular paralysis, nystagmus, optic atrophy, Argyll-Robertson pupil, loss or great exaggeration of knee-jerks, slight urinary difficulty, stumbling or scanning speech and characteristic mental impairment.

A. The gait and station are ataxic, especially when the eyes are closed, and the knee-jerks are absent. A history of lightning pains in the legs is common, also of loss of virility, slight urinary difficulty and ptosis or diplopia. Argyll-Robertson pupil in most cases. *Tabes.*

B. Intention tremor with nystagmus or scanning speech is associated with various signs of scattered lesions, such as isolated paralyses, contraction of the visual fields with

impairment of color sense, loss of smell, nervous deafness, etc. *Disseminated Sclerosis.*

C. Failure of judgment and memory, often combined with monstrous and unsystematized delusions of grandeur, accompanies signs of organic disease, among which inequality of pupils, facial twitching and stumbling speech are common. *Paretic Dementia.*

III. Positive signs of organic disease of the brain or membranes absent, but there is evidence that the cerebral vessels are diseased.

A. The patient is past forty. The arteries are atheromatous and pulse hard. Vertigo often accompanied by throbbing headache which is increased by exertion or excitement. Heart often hypertrophied and some albumen may be found in the urine.

Arteriosclerosis.

B. Age and general condition exclude senile degeneration. Nocturnal headache and insomnia generally present. History or physical condition or mental deterioration without other assignable cause gives evidence of syphilis. *Syphilitic Endarteritis.*

IV. There is no organic disease of brain, membranes or vessels.

A. The vertigo follows a blow on the head and may persist after all other symptoms have disappeared. *Concussion.*

B. The vertigo follows rotary, swinging or vertical motion of the body, as in sea-sickness. *Mechanical Vertigo.*

C. There is a toxic cause, as in cases of over-consumption of alcohol, tobacco, caffein or other drugs, or of auto-intoxication as by uric acid, urea or ptomaines.

Toxic Vertigo.

D. Vertigo occurs in paroxysms, in association with tinnitus and deafness which are shown to be nervous by impairment of bone conduction and limitation of the auditory field for high notes. Lesions of the middle or external ear may coexist and in some cases they are the primary cause of the labyrinthine disease. Paroxysms are sometimes excited by coughing, sneezing, a loud noise, indi-

gestion or any depressing influence, and may be severe enough to hurl the patient to the ground. Between paroxysms vertigo may be entirely absent or present in a slighter degree, but deafness and usually some tinnitus persist. Nystagmus, diplopia, vomiting and severe prostration may be secondary consequences of an attack. A disease of adult life, twice as frequent among men as among women.

Labyrinthine Vertigo (including Ménière's Disease). Subacute and chronic inflammation and degeneration due to cold, gout, syphilis or senility are the common conditions; hemorrhage and acute inflammation are very rare.

E. Weakness of one or more ocular muscles causes an erroneous projection of the visual field. Vertigo disappears on closing the eyes or on restoration of the muscle balance. Usually transient owing to the re-education of the sensory centers involved. Comparatively rare. *Ocular Vertigo.*

F. Vertigo is preceded by indigestion and disappears when the indigestion is cured. Organic disease of the labyrinth excluded. *Gastric Vertigo.*

G. Vertigo is associated with disease of the nose or adjacent sinuses and increases or diminishes with it, sometimes being relieved by an application of cocaine. The labyrinth is not organically diseased, but is probably disturbed owing to an intimate relation between the nasal and labyrinthine vessels. Very rare. *Nasal Vertigo.*

H. The cerebral circulation is disturbed.

1. Vertigo associated with throbbing in the head, perhaps with flushing of the face and injection of the conjunctivæ. Increased by coughing, sneezing, straining or lowering head. *Active Hyperemia.*
2. Associated with dull, heavy feeling in head. Return of venous blood obstructed by mitral regurgitation, tumor of the neck, constriction or other cause. *Passive Hyperemia.*

3. Heart action is weak or impoverishment of blood is shown by pallor of skin and mucous membranes, faintness, low percentage of hemoglobin or paucity of red corpuscles. *Cerebral Anemia.*

I. Vertigo occurs as part of a periodic nervous attack, disease of the labyrinth being excluded.

1. The vertigo occurs as the aura of an epileptic convulsion or, together with a brief interruption of consciousness, constitutes an attack of petit mal.
Epileptic Vertigo.

2. The vertigo is part of periodic attacks in which unilateral headache, nausea, intolerance of light and noise, and partial darkening of the visual field or subjective perception of light are common. Rarely it may constitute the whole of an attack.
Migrainous Vertigo.

3. The earlier attacks have an emotional cause and afterwards may occur or be suppressed in consequence of an emotional change or a suggestion. Various signs of hysteria may be present. Hysterical deafness and tinnitus may be so associated as to closely simulate labyrinthine disease. *Hysterical Vertigo.*

J. The patient's nervous energy has been exhausted, especially by prolonged worry with or without overwork. He can generally exert normal bodily and mental powers for a short time but is soon fatigued and is more or less irritable. Unreasonable fears are common. Painful or tender spots in the head or along the spine (found to be of shifting location if accurately marked) are very common. Other morbid sensations in the head are often described as queer rather than painful, such as tightness or looseness of the scalp, heaviness, lightness, emptiness or dullness of the head, etc. *Neurasthenia.*

K. No sufficient cause can be found.
"*Essential Vertigo*," most probably a symptom of beginning disease of the labyrinth or of an undetected accumulation of uric acid.

COMA.

The patient is unconscious and a satisfactory history cannot be obtained.

I. The head has been injured.

A. Skull generally unfractured. No paralysis. Coma not profound. Patient tosses about. Pupils contracted. Pulse rapid. Breathing quiet. Reflexes retained.

Cerebral Concussion.

B. Skull often fractured. There is unilateral paralysis often with conjugate deviation of head and eyes, or there is relaxation on both sides of the body. Coma profound. Pupils dilated, often unequal. Pulse slow. Breathing stertorous. Superficial reflexes lost or diminished, especially on the paralyzed side, and all reflexes may be abolished.

Cerebral Compression, due to hemorrhage, usually meningeal.

II. Careful examination reveals no evidence of injury to the head.

A. There is unilateral paralysis, often with conjugate deviation of head and eyes. Pupils may be unequal.

Diagnosis as in Hemiplegia.

B. No paralysis or other sign of unilateral cerebral lesion.

1. Heart very feeble. Respiration shallow. Skin pale and cold. There may be evidence of loss of blood. Consciousness quickly returns if the cerebral circulation is restored. *Syncope.*
2. Urine albuminous and contains casts or is scanty and of low specific gravity. There may be characteristic pallor and edema or retinitis. Eclampsia may occur. Breath may have a urinous odor.

Temperature generally depressed except in puerperal cases. *Uremia.*

3. Urine contains a considerable quantity of sugar. Breath may have the odor of acetone. *Diabetes.*
4. Diminution of hepatic dullness, enlargement of spleen, sallowness, emaciation and ascites or other sign of portal obstruction present. No other cause. *Cirrhosis of Liver.*
5. Pupils contracted. Respiration weak and slow. Pulse generally strong. Narcotic odor of breath or the surroundings may indicate that opium or one of its preparations has been taken. *Opium Poisoning.*
6. Respiration weak and slow. Pulse weak, perhaps rapid or irregular. Contents of stomach heated with caustic potash or soda give odor of chloroform. *Chloral Poisoning.*
7. Odor of carbolic acid. Mucous membrane of mouth whitened and shrivelled. Urine may be greenish black. *Phenol Poisoning.*
8. Coma not profound. Odor of alcohol on breath. Temperature normal or subnormal. *Drunkenness.*
9. Patient has been exposed to extreme heat in a moist atmosphere. Bodily temperature very high. *Heat-stroke.*
10. All the above causes absent. Tongue may be bitten. Convulsion may be known to have accompanied onset of coma, which does not last long unless convulsion recurs. Patient generally in first half of life. *Epilepsy.*

N. B. Many other conditions cause coma, especially just before death, but the history is generally known and the diagnosis made from earlier symptoms.

DISORDERS OF SPEECH.

I. Words are badly uttered owing to defect in the movements of the vocal organs, but, considering the age and education of the patient, are not defective in number or arrangement. The understanding of spoken words and reading and writing are not affected.

A. The patient speaks only in a whisper and the vocal cords cannot be voluntarily brought together, although they approximate perfectly in coughing or sneezing. Whispered articulation is generally good, but the aphonia may pass into mutism. Onset usually sudden in an hysterical attack or after violent emotion, but may be gradual. Other signs of hysteria always present.

Hysterical Aphonia.

B. Utterance is suddenly arrested by spasm of the lips, tongue, glottis or respiratory muscles, causing some of the muscular contractions of normal speech, particularly those which momentarily stop the flow of expired air, to be unduly intensified and prolonged. The difficulty is not in making any of the elementary sounds taken separately, but in relaxing the muscles needed for one sound in time to produce the next. Singing may be normal. Always worse when the patient feels himself to be under observation.

Stuttering (Anarthria Spasmodica). Generally a functional neurosis, but may complicate organic brain disease.

C. Certain literal sounds are formed with difficulty or not at all, or are misplaced, so that words are slurred, distorted or mutilated.

Stammering (Anarthria Literalis).

1. The defect is due to paralysis of two or more of the

following organs: lips, tongue, palate, pharynx and larynx.

Bulbar or Pseudo-bulbar Paralysis, which see.

2. The impairment of utterance appears along with hemiplegia and is often accompanied for a time by some degree of motor aphasia, especially in right hemiplegia.

Diagnosis as in Hemiplegia, commonly a vascular lesion in the region of the internal capsule.

3. The defect in speech is accompanied by gradual impairment of judgment and memory, often by monstrous and unsystematized delusions of grandeur. It consists mainly of syllable-stumbling, especially over words containing l and r. A facial twitch often accompanies the effort to speak. Pupillary changes and other signs of degenerative cerebral disease are common. *Paretic Dementia.*

4. The defect in speech appears along with intention tremor, which is often accompanied by nystagmus, amblyopia, various paralyses or other signs of scattered lesions. At first speech is abnormally distinct and slow in a monotonous voice, suggesting a schoolboy's scanning of Latin verse, but later words are slurred and indistinct. *Disseminated Sclerosis.*

5. There is no evidence of organic nervous disease.

   a. The mode of onset and accompanying symptoms indicate hysteria. The stammering varies greatly in character and may be combined with stuttering. *Hysterical Stammering.*

   b. The defect is due to the incoördination of the vocal organs in chorea. May pass temporarily into complete motor aphasia. *Choreic Stammering.*

   c. The stammering is due to a toxic state as in typhoid fever, uremia, gout, diabetes, narcotic poisoning or snake bite. *Toxemic Stammering.*

II. The patient's vocabulary, in at least one of the ways in which language may be used (speaking, understanding, reading or writing), is greatly diminished and the words used in speaking, writing or reading aloud are often badly arranged. *Aphasia.*

A. Organic intracranial disease is indicated by the accompanying symptoms, particularly paralysis of the face, a sudden onset with disturbance of consciousness (the causes of a vascular lesion being present), hemianopia, Jacksonian epilepsy or optic neuritis. The form of the aphasia indicates the seat of the lesion, which is in the left hemisphere in the right-handed and vice versa; the nature of the disease is to be inferred as in hemiplegia, since any organic change in a cerebral hemisphere may be a cause of hemiplegia or of any form of aphasia. Softening from vascular occlusion is the most common cause of aphasia because it is the most common change in the cortex.

1. Speech is heard and understood.

a. The words spoken are very few or perhaps none at all, although the vocal organs are not paralyzed. Those that can be spoken may be used on all occasions without regard to their meaning, but the patient knows when he has used them incorrectly. Oaths and songs may be retained without the power to use any of the words separately.

Motor Aphasia.

i. Writing abolished and the comprehension of written or printed language greatly impaired after all indirect effects of the lesion have passed away. Disability usually permanent.

Cortical Motor Aphasia. Lesion of the cortex at the foot of the third frontal convolution.

ii. Writing and quiet reading are regained when the indirect effects of the lesion have passed

away. The aphasia usually accompanies right hemiplegia, due to a lesion of the internal capsule and often improves rapidly.

Subcortical Motor Aphasia. Lesion of the white matter beneath the third frontal convolution.

b. The number of words correctly spoken is large, but the patient may be unable to name objects seen and nouns may be lacking in spontaneous speech. The characteristic symptom is the inability to read aloud or to understand written or printed words, although they are seen (word-blindness). There is always some defect in the right half of each visual field (left half in left-handed persons), usually hemianopia, but acuity of vision is not sufficiently impaired to account for the alexia. Mind-blindness (the inability to recognize familiar objects when seen) may be added to the word-blindness, indicating a lesion in each hemisphere.

Visual Aphasia.

i. Writing is abolished, owing to the inability to recall the appearance of written words.

Cortical Visual Aphasia. Lesion of the angular gyrus.

ii. Spontaneous writing is retained, although copying is impaired or lost, but the patient cannot read what he has written.

Subcortical Visual Aphasia. Lesion beneath the angular gyrus so placed as to interrupt its communication with the right visual centers.

c. Speaking and reading are normal, but there is inability to write with the right hand, owing to incoördination without paralysis, while writing with the left hand is retained. Rare. To be carefully distinguished from the agraphia complicating

motor, visual or auditory aphasia, which is much more common.

Graphomotor Aphasia. Probably a lesion of the foot of the second frontal convolution.

2. Speech is heard but not understood, the words of the patient's mother tongue sounding to him like those of an unknown language (word-deafness). There is also more or less inability to recall words, especially nouns, although the corresponding ideas are distinct (verbal amnesia). The word-deafness and verbal amnesia sometimes exist alone but in a typical case, while some words are correctly spoken, wrong words of similar sound or meaning are often substituted for the right ones (paraphasia) and others are mutilated so that speech becomes a jargon without the patient being aware of it. Writing suffers in the same way as speech (paragraphia) but to a greater degree and may be entirely abolished. Reading aloud is changed in the same way (paralexia) and the comprehension of written or printed words is generally lost.

Auditory Aphasia. Lesion of posterior part of first temporal convolution.

B. There is no evidence of organic intracranial disease.

1. Owing to deafness occurring in early life the patient has not learned to talk or has forgotten how.

Deaf-mutism.

2. The loss of words and voice is generally absolute, yet the patient is intelligent and writes readily. In very rare cases writing may be lost or recurring utterances occur so as to cause a strong resemblance to organic motor aphasia. Other evidence of hysteria always present. *Hysterical Mutism.*

3. The aphasia takes one or more of the forms described as due to organic disease, most commonly motor aphasia or a mild degree of auditory aphasia.

a. There is motor aphasia following the stammering

of severe chorea. Disappears as the chorea improves. *Choreic Aphasia.*

b. The aphasia is one of the phenomena of epilepsy or migraine, lasting from a few minutes to a few hours. *Epileptic or Migrainous Aphasia.*

c. The aphasia appears in the state of profound exhaustion toward the end of an infectious fever, especially typhoid, but disappears as strength returns. *Aphasia of Infectious Fever.*

d. The aphasia is due to a toxic state of the blood, most frequently uremia or narcotic poisoning, occasionally diabetes, gout or snake-bite.

Toxemic Aphasia.

INSANITY.

The patient's mind is so undeveloped or so changed from its normal condition as to unfit him for the domestic, social or business relations appropriate to his age and station. The delirium and stupor of acute fevers and acute intoxications are not included, unless unusually prolonged.

I. The mental defect is due to arrested development of the mind caused by disease or injury of the brain in early life. All the later acquired powers, such as sustained attention, abstract reasoning, self-control and observance of the social proprieties, are strikingly defective and in all but the slightest cases it is easy to show a marked defect in memory. Results or accompaniments of the cerebral disease are often conspicuous, such as cranial malformation, hemiplegia, monoplegia or paraplegia of the spastic type, disorders of speech, epilepsy and various stigmata of degeneracy. A history of difficult birth or of convulsions in infancy is common. If the arrest occurs at or before birth or in early infancy and the defect is very great, so that the patient is unable to take proper care of his person or to express his wants intelligibly, it is always called idiocy; but if the arrest occurs later or is incomplete, so that the mental defect is not so great, it is often called imbecility or merely feeble-mindedness, although it is really a milder degree of idiocy. Imbeciles can generally care for the person and talk fairly well and most of them are capable of considerable improvement by education, while a few show exceptional power in certain limited fields, such as music or arithmetical calculations. *Idiocy.*

On account of its convenience for clinical purposes the following classification of idiocy is taken from Frederick

Peterson with but slight modification. The same case may be in more than one class.

A. The arrest of development is caused by hydrocephalus. *Hydrocephalic Idiocy.*

B. The cranium is abnormally small. *Microcephalic Idiocy.*

C. The disease arresting mental development has caused hemiplegia, paraplegia or monoplegia. *Paralytic Idiocy.*

D. The arrest of mental development is due to repeated epileptic attacks. *Epileptic Idiocy.*

E. The arrest is caused by injury to the head. *Traumatic Idiocy.*

F. The arrest is caused by meningitis. *Meningitic Idiocy.*

G. The arrest is caused by scarlatina, measles, diphtheria or other infectious fever. *Post-febrile Idiocy.*

H. Arrest of physical and mental development is caused by disease of the thyroid gland which is generally enlarged. The disease is common in the mountainous regions of Europe and Asia, but rare elsewhere. The patients are dwarfs having the characteristic appearance of cretins, the skin being thick, coarse and yellow, the hair scanty, eyelids puffy, eyes small, nose flat, lips thick, tongue large, teeth deficient and limbs deformed. The administration of thyroids causes improvement which, in some cases, is very great. *Myxedematous Idiocy.*

I. Degeneration of the retinal neurons, causing blindness, and degeneration of the neurons of the cortex and central nervous system generally, causing idiocy, occur together in young infants of certain families. *Amaurotic Idiocy.*

J. The patients show a special aptitude in some limited field, such as music, arithmetical calculations, drawing or buffoonery, which is far in excess of their other powers and may greatly exceed that of the average normal individual. *Idiots Savants.*

K. The lack of mental development is due to deprivation of

sight and hearing and may be remedied by appropriate education as in the cases of Laura Bridgman and Helen Kellar. *Sensorial Idiocy.*

II. The disease occurs in a mind already developed and consists in either a perversion or a loss of faculties.

A. There is a state of emotional exaltation or depression, accompanied by a corresponding acceleration or repression of ideation, speech and action. There is no real failure of memory, although it may be impossible to secure the patient's attention sufficiently to test it. Delusions, if present, are secondary to the emotional change and in harmony with it and are not systematized. There are no signs of organic disease.

1. After an initial stage of mental depression the patient becomes excited and exhilarated. His talk is rapid and shows a ready, though superficial, association of ideas. There is an excessive tendency to act in accordance with any idea that may occur and all the ordinary restraints on speech and action are absent or are effectual for a few moments only. Appetite and the animal instincts generally are excessive. Delusions, if they occur, are of a pleasant, often of an ambitious nature and are inconstant and often incoherent. In general, the patient appears as though in the excited stage of alcoholic intoxication. All gradations exist from the slightest noticeable exhilaration to furious madness, marked by incoherent raving and blind destructiveness. *Mania.*
2. The patient gradually passes into a state of mental depression, in which the association of ideas is retarded. Delusions or hallucinations, if they occur, are painful and include the idea of personal disrepute or guilt. As a general rule food is refused and speech and action repressed or almost abolished, the patient staring ahead in profound dejection, perhaps automatically performing some destructive action such as picking clothing to pieces. Exceptionally

there are frantic efforts to escape from imaginary evils (melancholia agitata). The tendency to suicide is strong and attempts at suicide may be preceded by homicide, under the delusion that the victim is benefited by being removed from a wretched world.

Melancholia.

3. Attacks of mania and melancholia alternate with each other, with or without a normal interval, so as to form a regularly recurring cycle. Incurable.

Circular Insanity.

B. The patient is subject to delusions which are logically coherent or systematized and are mainly limited to one subject. There is no such profound emotional change as in mania or melancholia and memory is not impaired, being exceptionally accurate for occurrences associated with the delusions. There is an hereditary predisposition to insanity and stigmata of degeneracy are common.

In a typical case, after a period of morbid introspection, there gradually develops a delusion of persecution, based partly on hallucinations, especially of cutaneous sensibility and hearing, and partly on misinterpretation of actual occurrences. The means believed to be employed by the persecutors varies with the environment, education and imaginative power of the patient; electricity, hypnotism, mind-reading and X-rays being common now, while witchcraft and demoniac possession were the common means a couple of centuries ago. The imaginary persecution becomes more persistent and systematic and is usually supposed to be carried on by some powerful organization, such as the Free-masons, the Catholic Church or a political party. Later, apparently as an explanation of the persecution, there generally appears a delusion of great personal importance, such as being the heir to a throne, a political or religious reformer, a great inventor or even Jesus Christ.

The delusions may vary from the typical form (paranoia querulans, paranoia erotica, paranoia hypochon-

driaca, etc.), and hallucinations may be especially prominent (paranoia hallucinatoria), while in many cases the disease is but partially developed (cranks), but systematization is characteristic of all cases. The patient can give elaborate reasons for his beliefs, however absurd, and can exercise his mental faculties normally on subjects not connected with the delusions. Formerly called monomania. Incurable. Some cases end in a mild dementia. *Paranoia.*

C. There is an intellectual loss shown most readily by a failure of memory for recent events, but always involving impairment of the judgment, attention, abstract reasoning and esthetic, social and ethical feeling. Emotional changes, hallucinations and delusions may occur, but they are secondary and delusions are never systematized.

1. There has been no preëxisting mental disease or epilepsy and there is no evidence of organic cerebral disease. The patient, a youth or young adult with an hereditary predisposition to insanity and perhaps given to masturbation, rapidly becomes depressed, stupid and apathetic, often reaching an extreme degree of degradation. As a rule recovery takes place, leaving no recollection of the period of stupor, but death may occur or true dementia supervene. Most nearly related to melancholia, from which it is distinguished by greater impairment of consciousness and memory and absence of the profound depression with sense of guilt and of delusions. Originally called acute dementia, but differs from true dementia in the absence of organic cerebral disease and the possibility of recovery. *Stuporous Insanity.*

2. The mental defect is caused by some form of organic cerebral disease and is permanent. *Dementia.*

   a. There is coarse organic disease of the brain, such as tumor, abscess or a vascular lesion.

      Dementia of Coarse Organic Disease. Special diagnosis as in Hemiplegia.

b. The dementia is preceded by mania or melancholia or, rarely, by paranoia and at first is mixed with the exaltation, depression, or delusions characteristic of the cause. Later these vestiges of the original disease vanish and the dementia may reach an extreme degree of mental degradation.

Terminal Dementia.

c. The faculties are lost as age advances. Differs from the normal loss of mental vigor in old age only in degree. *Senile Dementia.*

d. The dementia is preceded by prolonged excess in the use of alcohol, often by attacks of delirium tremens and by delusions of suspicion and fear, based on mental depression and hallucinations, somewhat resembling the delusion of paranoia, but less systematized. Onset may be very gradual or rapid during an attack of delirium tremens.

Alcoholic Dementia.

e. The dementia gradually supervenes in epilepsy. No other cause. *Epileptic Dementia.*

f. The patient is almost always a man, in early or middle adult life, who has been syphilitic or alcoholic or who is nervously exhausted by a fast life or some prolonged strain. After a neurasthenic stage there is a gradual alteration of character and loss of mental power. Along with the mental defects common to all forms of dementia there are various signs of degenerative cerebral or cerebro-spinal disease. Speech is marred by stumbling and perhaps stuttering, especially in words containing l and r. The effort to speak or to protrude the tongue is often accompanied by a facial twitch. The writing is badly formed and more or less incoherent on account of repetitions and omissions. Tremor of the hands and a fine fibrillary tremor of the tongue and lips are common. The pupils are generally unequal but the

difference is inconstant. Argyll-Robertson pupil may be found. Weakness or incoördination of any or all of the limbs may supervene and the knee-jerks, although usually exaggerated, may be lost, so as to cause a strong resemblance to tabes. Epileptiform and apoplectiform attacks are common. In most, but not all cases, the neurasthenic depression is succeeded by a stage in which there are monstrous, unsystematized delusions of wealth, power or personal excellence. Remissions, even apparent cures, may occur, but the disease always advances again and ends fatally, generally within two or three years from the onset of symptoms.

Paretic Dementia.

D. The insanity presents a superficial resemblance to mania but differs from it in the more rapid onset and greater violence of the symptoms, absence of the characteristic exhilaration, greater impairment of consciousness and absence of any subsequent recollection of the attack.

1. The patient is almost always a man in early or middle adult life. There is no prodromal period and the onset is exceedingly rapid. There is a swift succession of incoherent ideas, revealed by words, shouts or inarticulate cries, and accompanied by uncontrollable rage. There is intense and general motor excitement which culminates in a blind destructive fury. The face and conjunctivæ are flushed, the circulation and respiration accelerated and the temperature raised. The symptoms rapidly subside within a few hours to a few days of the onset, generally within twelve hours, and end in sleep, from which the patient awakes with no recollection of the attack. Recovery is complete and the attack does not recur. Rare.

Transitory Frenzy.

2. The patient is of neurotic inheritance, more frequently a woman. The exciting cause may be a fever, alcoholic excess, puerperal disease or emotional shock.

After a prodromal period of not more than a few days which is marked by depression, headache. insomnia and irritability, a violent delirium sets in, Under the influence of constantly changing, incoherent hallucinations and delusions the patient sings, shouts or swears and, if unrestrained, attacks attendants and smashes furniture. Anger or dread usually predominate; exaltation is rare. Pulse and respiration are rapid and feeble and temperature irregularly elevated. Food is generally refused. Involuntary evacuations occur. Ends within a few weeks, sometimes within a few days, in extreme prostration, which is followed by coma and death, by dementia, or rarely by recovery without recollection of the attack. Second attacks do not occur. *Acute Delirium.*

E. The insanity is caused by injury to the brain or by a definite constitutional condition. The mental symptoms generally differ from the typical forms of insanity in being of an incomplete, mixed or inconstant type and are better described as delirium than as mania, melancholia or paranoia. If maniacal excitement occurs the typical exaltation is apt to be lacking, while unpleasant hallucinations, incoherence and confusion are more prominent than in true mania. Although there is often a marked depression it is not likely to be so constant or to involve so great a sense of guilt as in melancholia. Delusions of suspicion or persecution frequently occur but they are neither so constant nor so coherent and systematized as in paranoia. Temporary stupor may simulate dementia and genuine dementia is not an uncommon termination.

1. Insanity is caused by a blow on the head and consists in confusion with hallucinations and delusions which are generally of an unpleasant character. Headache, vertigo, irritability and various physical defects are common. May end in recovery or in dementia.

Traumatic Insanity.

2. Insanity is caused by sunstroke, the symptoms being like those of traumatic insanity.

Insanity of Insolation.

3. Insanity results from the excessive consumption of alcohol, not necessarily causing drunkenness, but eventually causing some of the signs of chronic alcoholism, such as gastric catarrh, cirrhosis of the liver, morning vomiting, characteristic odor of the breath, bloated face, tremor, etc.

   a. After an unusual excess or on the occurrence of some local inflammation or injury, or perhaps without any exciting cause, the patient becomes dejected, timid and restless. Sleep is at first disturbed by horrible dreams and within a few days these are replaced by insomnia with illusions and hallucinations, especially of seeing loathsome and dangerous animals, accompanied by corresponding terrifying delusions. For a time the visions may be dispelled and the patient reassured, but they return in constantly changing forms and he becomes continuously delirious, talking and acting as though at his ordinary work, reaching for imaginary objects or trying to escape or to protect himself. Hallucinations of touch, hearing or smell may also occur. Speech becomes more and more incoherent and may be reduced to an unintelligible muttering.

   The temperature is elevated, the pulse rapid and soft and there is tremor, especially of the hands, face and tongue.

   Within a week convalescence begins or there is increasing prostration which generally leads to coma and death. May be complicated by pneumonia, hepatic or renal disease, alcoholic eclampsia or neuritis. *Delirium Tremens.*

   b. The mental symptoms are a complication of alcoholic multiple neuritis and are less acute than de-

lirium tremens, consisting mainly in loss of memory for recent events and a corresponding delusion of living in the past. The patient usually says that he has been out of doors, engaged in his usual occupations, during the period of illness. The manner may be perfectly rational. Recovery may be complete, but there is a tendency to dementia.
Insanity of Alcoholic Neuritis.

c. Following incomplete recovery from delirium tremens or independent of it, there is a gradual deterioration of character and impairment of judgment and of memory for recent events. Hallucinations, especially of hearing, occur and delusions of being the victim of marital infidelity or the object of a conspiracy to mutilate or kill are very common. Desperate attempts at escape or defense may result in serious injury to self or in homicide. Differs from melancholia agitata in the prominence of hallucinations and in the emotional depression being secondary and sense of guilt being less profound; from paranoia in the inconstancy of the delusions and their lack of systematization. Improvement generally occurs when alcohol is withdrawn, but there is a strong tendency toward increasing dementia. *Chronic Alcoholic Insanity.*

d. The disease consists in a periodically recurring, uncontrollable craving for alcoholic drinks which impels the patient to degrading debauches. During the interval there is no abnormal desire for drink and conduct may be quite rational, but evidences of a neurotic inheritance and stigmata of degeneration are common. *Dipsomania.*

4. The patient is syphilitic. Beginning, as a rule, with insomnia and severe nocturnal headache, he passes into a state of melancholic depression or excited delirium, often accompanied by delusions of suspicion, or, more frequently, into a dazed, stupid

condition resembling dementia. The diagnosis may often be confirmed by the presence or history of an eruption, mucous patches, nodes on bones, a cerebral vascular lesion in the absence of atheroma or heart disease, or by some other indication of syphilis; it may, however, depend solely on the nature of the mental symptoms and the absence of any other adequate cause. Tends to end in dementia, but under appropriate and timely treatment complete recovery may be secured.

Syphilitic Insanity.

5. Insanity comes on during or soon after parturition or during pregnancy. The form of the mental symptoms varies greatly in different cases and is often changeable in the same case. Hallucinatory delirium with confusion is the most common condition and with this there may be depression simulating melancholia, excitement simulating mania, but generally without exaltation, and delusions of suspicion suggesting paranoia. Profound apathy may occur and either pass away or merge into true dementia. Sleep is poor or absent. Appetite is generally poor and food is often refused. The duration may be only a few days or very long. Many patients recover, but some die and in some the insanity is permanent.

Puerperal Insanity.

6. Symptoms like those of puerperal insanity come on after the puerperal period, owing to a profound state of exhaustion which culminates during lactation. *Lactational Insanity.*

7. Insanity occurs as a consequence of the nutritive disturbance of pulmonary tuberculosis. It is marked by apathetic depression resembling melancholia or dementia, delusions of suspicion or a confused hallucinatory delirium.

Phthisical Insanity.

8. The symptoms are like those of phthisical insanity but are caused by the cachexia of carcinoma.

Carcinomatous Insanity.

9. In the course of an attack of articular rheumatism (or possibly of chorea without rheumatism) delirium comes on and is accompanied by violent choreic movements. Delusions of suspicion are common. Signs of organic disease of the central nervous system may appear and cardiac complications are common. May last weeks or months.

Rheumatic or Choreic Insanity.

10. Insanity is caused by an acute infectious disease, such as typhoid fever, pneumonia, influenza, scarlatina, septicemia or malaria. It consists at first in confused, hallucinatory delirium which may pass into a stupor or into delusions of suspicion. Ends mostly in recovery, sometimes in idiocy or dementia.

Insanity of Infectious Fever.

11. Symptoms like those just described are caused by starvation. *Insanity of Inanition.*

12. Symptoms like those just described are caused by lead, mercury, cocaine, morphine or other poison or by auto-intoxication as in nephritis or diabetes.

Toxic Insanity.

13. The patient is epileptic and the mental disturbance is a part of the epileptic attacks or a result of their frequent occurrence. To be distinguished from those cases in which epilepsy and insanity have a common cause, as in various forms of organic cerebral disease.

    a. The mental disturbance is transitory, occurring after the epileptic attack or just before it or constituting the entire attack. It may consist of an hallucinatory delirium, leading sometimes to impulsive acts of great violence, or of a dazed, stupid condition, or perhaps of a condition appearing normal to the casual observer, in which the patient performs accustomed actions in the

ordinary way, but is liable to be impelled to any foolish or criminal act by ideas over which he has no control. The insane state may last but a few minutes or for days. When it passes away the patient usually has absolutely no recollection of it and at best his recollection of it is very imperfect. *Transitory Epileptic Insanity.*

b. Owing to incomplete recovery in the intervals, the insane state, which was at first limited to the time of attack, becomes nearly constant and ends in epileptic dementia.

Chronic Epileptic Insanity.

14. The patient is subject to severe hysteria and the mental symptoms of hysteria merge into a delirium which is changeable, highly emotional and marked by profound egoism and a desire to attract attention. Illusions are frequent, hallucinations also occur and there may be erotic or persecutory delusions. To be distinguished from other forms of insanity not caused by hysteria but occurring in the hysterical.

Hysterical Insanity.

15. The patient is neurasthenic and the morbid impulses, fears, imperative ideas, hypochondriacal fancies, indecision or inability to fix the attention, which are characteristic of neurasthenia, have gradually become so intensified and so dominate conduct as to constitute true insanity. The exaltation of mania, the characteristic depression of melancholia, the systematized delusions of paranoia and the intellectual defect of dementia are entirely absent. The patient is aware of the irrational action of his mind and deplores it. Rare; very many neurasthenics fear insanity for every one who actually becomes insane. *Neurasthenic Insanity.*

16. The patient has myxedema and the mind becomes dull, tending toward stupor and dementia. A confused delirium with hallucinations and changing

delusions may occur and may be of either the melancholic or the maniacal type. On the administration of thyroids all symptoms improve, at least for a time. *Insanity of Myxedema.*

17. The mental disturbance complicates exophthalmic goitre and may be of the melancholic, maniacal or hysterical type. *Insanity of Exophthalmic Goitre.*

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