3.—

A TREATISE

ON

NERVOUS DISEASES;

THEIR

SYMPTOMS AND TREATMENT.

A TEXT-BOOK FOR STUDENTS AND PRACTITIONERS.

BY

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trust that students and general practitioners who have little time to read will find what they most need for diagnosis and treatment of the cases occurring in practice. In some parts, especially the introductory chapters, the condensation may seem to have been carried too far; but more extensive descriptions would have required an increase in bulk.

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A TREATISE

ON

NERVOUS DISEASES.

CHAPTER I.

GENERAL INTRODUCTION.

EULENBURG, A., Lehrbuch der functionellen Nervenkrankheiten. Berlin, 1871.—ZIEMSSEN, Cyclopædia of the Practice of Medicine. Vols. XI, XII, XIII, XIV.—ROSENTHAL, M., A Clinical Treatise on the Diseases of the Nervous System. Trans. by L. Putzel. New York, 1879.—HAMMOND, WILLIAM A., A Treatise on the Diseases of the Nervous System. New York, 1881.—HAMILTON, A. McL., Nervous Diseases : their Description and Treatment. Philadelphia, 1881.—CHARCOT, J. M., Lectures on the Diseases of the Nervous System, delivered at La Salpêtrière. Trans. by George Sigerson. New Sydenham Society, 1877, 1881.—GRASSET, J., Maladies du système nerveux. 1881.—BUZZARD, T., Clinical Lectures on Diseases of the Nervous System. Philadelphia, 1882.—AXENFELD, A. (Huchard), Traité des névroses. Paris, 1883.—ROSS, JAMES, A Treatise on the Diseases of the Nervous System. 2 vols. New York, 1883.—STRÜMPELL, A., Krankheiten des Nerven-systems. Leipzic, 1884.

IT will be an advantage to consider in a general way the methods of examining sensation and motion in patients affected with nervous diseases. It will prevent the necessity of repetition if certain symptoms or complications are also considered in this introductory chapter.

METHODS OF TESTING SENSATION.

Changes of *sensation* of *touch* may be recognized by touching the patient as lightly as possible with the

finger or a feather. The finger should be kept still, on the spot first touched; if it is moved about, rubbing the skin, the patient will much more readily recognize that he has been touched.

The temperature of the finger should be as nearly as possible equal to that of the body; otherwise the difference in temperature is felt.

Changes in the sensation of *pressure* may also be recognized with the finger by varying the amount of pressure and asking the patient whether it is greater or less. There are various apparatus for more delicately testing this sense by means of adding graduated weights, but these are unnecessary for ordinary purposes.

In testing the sense of *pain*, a pin or a knife can be used, or the patient can be pinched with varying degrees of severity. If one or two hairs on the patient's limbs are pulled, the sensation will be very nearly the same as if a pin were stuck into that place.

Changes in the sensation of *temperature* may be examined by means of spoons dipped in tumblers of water of different temperature. More delicate methods of examining the variations in the sense of temperature may be found in having small bottles full of water of different temperatures. The patient is then asked whether the temperature of one is higher or lower than that of another. But these are rather refinements of examination, which are rarely of any great practical value.

The *muscular sense* may be tested by asking the patient, with his eyes shut, to move his limbs in different directions, or to find his feet in the bed, or, when the limbs are widely separated, to raise one foot and put it down by the side of the other.

The *æsthesiometer* is used in testing the ability of the patient to recognize whether one or two points touch the surface of the body. A rough form of æsthesiometer may be contrived by holding two pins in the fingers and, varying the distance of their points, touching

the patient with them; or needles may be stuck in a piece of wood, and then the patient touched. There are, however, many simple instruments for this examination, as compasses with a graduated scale, or points sliding on a graduated rod. In making this examination, it is necessary that both points should touch the skin together with as nearly as possible equal force. In examining the opposite sides of the body, the points should be either transverse on both sides, or parallel with the axis of the limb on both sides. They should not be transverse on one side and longitudinal on the opposite.

The various sensations may be more acute than normal—hyperæsthesia; or less acute—anæsthesia; or there may be a perceptible delay in their recognition. Occasionally, when only two points are applied to the skin, the patient will have a sensation as if three or more touched him.

The *vision* may be affected in several ways. There may be partial blindness (amblyopia), or there may be entire blindness, or various other changes;—diplopia, if the axes of the two eyes are not parallel. This defect may be present only when the patient looks in certain directions, if one muscle is weaker than the corresponding muscle on the opposite side, so that the diplopia may not be noticed unless the patient looks to the right or left, or upward or downward. Vision may be limited in extent, so that the patient sees only objects placed directly in front of him. Or, more rarely, central vision may be wanting, and only objects around the periphery of the field of vision may be recognized.

The extent of vision for white light may be normal, or nearly normal, but the field of vision may be circumscribed for colors.

The extent at which colors can be seen is naturally not so great as the extent at which white can be seen; thus, the field of vision for green is the most contracted, next in extent is the field for red, then blue, then yel-

low. In some cases, the field of vision, instead of being limited concentrically, is lost on one side, so that the patient sees only that part of an object which is to the right or left of the median line. This is called *hemiopia*, as referred to the retina, or *hemianopsia*, as referred to the field of vision.

If accuracy is required in an examination, it is necessary to use a blackboard with a point of fixation, the patient being placed at a distance of a foot from that point, with one eye covered; a bit of white paper or chalk is then moved from the outside inward until the patient can see it, and a mark is made on the board. This is repeated at short intervals around the central spot until we have mapped out roughly the field of vision of the eye.

It is generally sufficient, however, with the patient sitting in front of the physician, with one eye covered, the other eye fixed upon the center of the physician's face, for the physician to move his fingers first one side and then the other, above and below, and inquire whether the patient sees the motion of the fingers. Sometimes the field of vision for white is natural, or nearly natural, but colors can be recognized only in one lateral half of the field of vision—hemianacropia; for testing such a change, the blackboard, with bits of colored paper, would be necessary.

The *ophthalmoscope* is absolutely necessary in order for a satisfactory examination of patients with diseases either of the brain or of the spinal cord, and it is better to use it in every case, no matter what the disease may be supposed to be.

To give any satisfactory description of the use of the ophthalmoscope would require altogether too much space, a short description being very unsatisfactory. It is better to refer to books on ophthalmology for such description.

It is very necessary to examine the pupils, to note changes in their reaction to light and to accommoda-

tion, to notice whether both pupils act alike, or whether there are differences between the two. In examining the pupils, it is necessary to cover the one not under observation, as otherwise the light, falling upon the sound eye, may cause contraction, when, if examined separately, the pupil would remain immovable.

METHODS OF TESTING MOTION.

The *motor power* may be examined by simply watching the movements of the patient. With children, it is a good plan to allow them to roam about the room, playing with whatever attracts their fancy.

With adults, one can watch their gait as they enter the room, their manner of taking a seat or executing other motions; if it is necessary for them to undress, the way in which they take off their clothing should be noted. Much can be learned by thus carefully watching the ordinary motions of patients.

If it is desired to recognize clearly slight losses of power in the limbs, the dynamometer may be used. There are several different forms of this instrument; the simplest is the best. It is not necessary to estimate the exact power, in pounds, of pressure, but it should be possible, from the scale of the dynamometer, to estimate the relative power of the two sides of the body. A rough idea of the relative strength of the two hands can be gained by allowing the patient to squeeze one's hand.

Slight fibrillary twitchings of the muscles may sometimes be excited, if they do not occur spontaneously, by snapping the muscles with the finger. The natural tone of the muscle is also indicated by the energy with which a single contraction takes place under the snapping of the finger.

The most delicate test of muscular power is electricity. Under some circumstances the muscle loses its power of responding to the irritation of the faradic or

the galvanic current. Any muscle which is separated from its centers of nutrition in the spinal cord undergoes the above change of reaction, whether the disease is a destruction of those trophic centers or of the nerve-fibers passing from them to the muscle.

The first change noticed in the muscle may be a slight and temporary increase of irritability for both forms of electricity. A day or two afterward the muscle begins to lose its power of reacting to the faradic current. By the end of the second or third week it no longer responds to this stimulus. After a slight diminution of reaction to the galvanic current, during the second week the irritability of the muscle for this current increases until it becomes much greater than normal. This increased irritability for the galvanic current may continue for many weeks or months, but at length is gradually lost, until, if the paralysis is permanent, the muscle responds to neither current.

With this change in the amount of irritability there occurs a difference in the manner in which the muscle reacts to the galvanic current. Instead of the natural, rapid, spasmodic contraction, the muscle contracts slowly, the contraction reaching its height only after a perceptible interval of time, and then slowly relaxing—a change that is very evident, even to the unskilled observer.

There are also changes in the quality of the reaction to the galvanic current, according as the positive or negative pole is placed upon the muscle, and the current closed or opened. These changes are of less practical value, and can be learned from works on electrotherapeutics.

This "*reaction of degeneration*" is of great importance in diagnosis.

Reflex actions play an important *rôle* in the internal economy of the body, and in its relation with the external world.

An impression made upon an afferent nerve is car-

ried to a group of nerve-cells; by means of these, alone or in connection with other groups, it is transformed into a motor impulse, which is carried by efferent nerves to muscles, and causes them to contract. This is the simplest form of a reflex action. The muscles excited are usually those nearest the point receiving the irritation. If, however, the irritation is severe, distant muscles may be brought into action, even those on the opposite side. The muscles may not be voluntary; they may belong to internal organs, as stomach, intestines, bladder; the impression may be made upon these viscera, not on the surface of the body; the groups of nerve-cells may not be in brain or cord, but in one of the sympathetic ganglia, or in the walls of the viscus.

The impressions upon the special senses may give rise to reflex actions confined to the organ receiving the impression, as movements of the iris, contraction of the intrinsic muscles of the ear; or the impression may be so strong as to call into action other muscles of the body, as when a loud noise or bright flash causes the head to turn, or sets the heart beating violently.

Reflex actions control, in a very large degree, the secretions of the different glands and the supply of blood to various parts of the body.

Thoughts and emotions have a great influence upon the circulation, the secretions, and the nutrition of the body; this influence is generally reflex in its nature, and may often be utilized in the treatment of disease.

It is not necessary that the brain should take cognizance of the impressions which excite reflex action; indeed, these actions are often more powerful when the subject is unconscious of the impression exciting them, as when the spinal cord is divided.

The reflex centers for the different groups of muscles in the limbs are situated at about the level whence the nerves supplying those muscles arise. There are reflex actions which follow an irritation of the skin—cutaneous reflexes; also actions which follow irritation

of deeper parts, of which the tendon reflexes are examples.

The various *reflexes* require a separate description. Gowers has given the clearest account of these. By gently irritating the skin, by tickling, scratching, or pricking, the superficial or cutaneous reflexes may be excited. Gowers mentions the plantar (from the sole of the foot), depending on the lower part of the lumbar enlargement; the gluteal, by irritating the skin of the buttocks, depending on the cord at the level of the fourth or fifth lumbar nerve; the cremaster, by which the testicle is drawn up when the skin on the inner aspect of the thigh is irritated, depending on the first and second lumbar pairs; the abdominal, by irritating the skin at the side from the ribs downward, depending upon the eighth to the twelfth dorsal nerves; the epigastric, produced by irritating the side of the chest in the fourth to the sixth intercostal spaces, depending upon the fourth to the seventh dorsal nerves. There are sometimes reflexes on the back, caused by irritating the skin along the edge of the erector-spinæ muscles; and when the skin between the scapulæ is irritated, some of the scapular muscles contract, the scapular reflex.

The deep reflexes, as they are sometimes called, include the *clonus* and *tendon reflexes*. The more common are the *patella tendon reflex*, and that developed just above the elbow. To obtain these, the limb should be semi-flexed, should hang free without voluntary muscular tension; a sharp blow just below the patella, on its ligament, or on the tendon just above the olecranon process, produces a sudden contraction of the muscle and partial extension of the limb. To examine for the patellar tendon reflex, the patient should sit on a high chair or table, with the legs swinging free; or, if the feet rest on the floor, one leg may be thrown over the opposite knee, or the physician can pass his arm under the knee, resting his hand on the other knee, the

leg hanging free over his arm. For the examination of the triceps humeri tendon reflex, the patient's arm, semi-flexed, should be supported so that the forearm can move with moderate freedom. Tendon reflexes are occasionally found with other tendons.

Ankle-clonus is excited by holding the patient's leg extended, or very slightly flexed on the thigh; then, by suddenly flexing the foot, and perhaps the toes too, the Achilles tendon and the flexors of the toes are put on the stretch; then a rhythmic contraction and relaxation of the calf muscles occur, which continue as long as the proper degree of flexion of the foot is maintained. Sometimes a strong pressure on the sole of the foot is needed to develop this, and sometimes only a very light pressure, the stronger checking the clonus. The clonus can sometimes be excited by a similar flexion of the wrist-joint. When the reflex excitability is much exaggerated, it may be possible to obtain a clonus in the toes or fingers.

The *front tap contraction*, which Gowers says is a very delicate test of increased irritability, is obtained by keeping the leg nearly extended on the thigh, the foot moderately flexed so as to keep the tendo-Achilles slightly tense, then a gentle tap with the ends of the fingers is made over the edge of the tibia. The calf muscles contract, drawing the foot down; the action is usually very slight, and is the stronger the nearer the ankle the tap.

A tap over the head of the tibia will sometimes produce a contraction of the rectus femoris; or a tap over the radius at the wrist will cause contraction of the biceps; over the ulnar at the wrist, of the triceps.

Tâche cérébrale is developed by drawing the fingernail or the finger across the skin, especially over the abdomen. A red line appears after a variable length of time, corresponding to the tract of irritation. This line can be produced in a large majority of patients.

It is of value as a symptom only when it appears very quickly and is of a deep color; the line made with the nail is brighter than the broader line made with the finger. At the side of the red line the skin seems to acquire a paler tint, as if the smaller vessels were contracted. This sign is of less value than was formerly supposed.

Cheyne-Stokes respiration, named from those who first described it, consists of a peculiar rhythmical change in the breathing. After a pause, in which two or three respirations are lost, the lungs are again filled, the breathing slowly increases in rapidity until a limit is reached; the frequency then diminishes until there is another pause. This succession of respiratory acts is then repeated. This symptom is a very serious one, and almost always indicates a fatal termination.

Bed-sores form with extreme rapidity in some cases of lesion of the spinal cord, as the result of irritation of the cord; at other times they are slow in appearance. They are among the most annoying complications, and sometimes give great discomfort.

Perfect cleanliness, bathing the skin after every passage, changing the bedding when wet or soiled, are absolutely necessary to prevent their occurrence. When the patient can be moved, his position should be changed to relieve pressure. The whole back, especially the sacral region, should be bathed daily with strong alcohol.

When the skin becomes discolored and a bed-sore seems imminent, even greater care should be taken in bathing, that spot be relieved from pressure, and, if the skin is broken, zinc ointment may be used. Further mischief may sometimes be thus avoided. If a slough forms, the best treatment is the alternate use of ice and poultices—ice for two or three minutes, then poultice for two or three hours. Of course, the offensive discharges must be removed; where the edges are undermined, a gentle stream of water from a fountain syringe

must be used to wash out the pockets holding the discharge. Carbolic acid, thymol, phenol, or similar substances, may be sprinkled on the poultices. A charcoal poultice is sometimes of benefit.

Various water-beds, air-beds, and fracture-beds are in use for these cases, and may answer a good end.

A mild galvanic current has been recommended, obtained by a silver plate on the sore, connected by wire with a zinc plate over the sound skin, and a piece of wet cloth placed between the zinc and the skin.

Constipation accompanies many nervous affections, whether organic or functional. Violent means to remedy this are rarely desirable. Hyoscyamus, belladonna, nux vomica, can be added to prescriptions or given independently, and may relieve the bowels sufficiently. If these are not sufficient, small doses of compound extract of colocynth may be added. Unless there is some contra-indication for strychnia, the following may prove serviceable:

℞ Ext. colocynth. comp., gr. ½ to 2;
Ext. belladonnæ, gr. ⅛ to ¼;
Ext. nucis vomic., gr. ¼ to ½.
M. Fl. pil.

A half-grain or grain of ipecac may be added to this with advantage if there is slight gastric disturbance. This should be given from once to three times daily, as may be necessary.

Drinking copiously, especially a large supply of water, hot or cold, early in the morning, will many times be all that is necessary.

In lesions of the spinal cord, more energetic cathartics may be needed; aloin or podophyllin, with belladonna or hyoscyamus, can be given in small pills. Extract of colocynth may be combined with these. Enemata of soap-suds or castor-oil, or both, may be needed to assist the drugs. It is very desirable to avoid impaction of the fæces.

If a stool has been long delayed, an enema of six to sixteen ounces of olive-oil, retained two or three hours, then followed by a pint or more of soap-suds, will often produce a motion with comparatively little discomfort to the patient. Of course, the physician should judge of the size of the enemata by the condition of the patient. A "fountain syringe" is much preferable to others.

Cystitis is frequently a troublesome complication in cases of paralysis. It is most frequently associated with lesions of the spinal cord, and sometimes is rather early in its appearance. Whenever there is retention of urine, there is danger lest, being only partially evacuated, the residue should become alkaline, and phosphatic sediments form; the decomposed urine is a source of irritation, and inflammation of the bladder is set up; this inflammation may extend to the ureters and kidneys, giving rise to much distress, and being itself a source of exhaustion and danger.

The bladder should be emptied by catheter twice a day. A long rubber tube attached to the end of the catheter, hanging into a vessel on the floor by the side of the bed, helps to keep the bed dry, and acts as a siphon to more thoroughly empty the bladder. If the urine becomes alkaline, benzoic acid, five grains thrice daily, or boracic acid, or salicylic acid, may be given. The bladder should be washed out at least once a day with a double catheter, using a weak solution of carbolic acid or nitrate of silver; the latter can be used of a strength of three or four grains to the ounce every third to sixth day.

The diet should be easily digestible with mild drinks.

The *nutrition* of patients is often below normal. It is a task to eat when there is no appetite, and the patient, yielding to his aversion for food, eats little; gradually the whole system suffers, yet there is no demand for more food; the system becomes accustomed to the lowered standard of nourishment.

Many times the greatest patience and tact is needed to restore the lost strength. It is generally better to give food frequently in small quantities than to try to increase the amount taken at one time. The intervals may be as near as every half-hour or hour.

Milk is the most convenient food. It can be made more palatable by adding salt, and more digestible by adding lime-water, half an ounce to six ounces of milk, or five grains of bicarbonate potassa or soda to the same amount. It should be slowly sipped, or taken with a teaspoon—not drank: one to three quarts a day, according to how much else is taken. Koumiss is a pleasant and easily digestible form of milk; directions for making it are given in the Dispensatory.

Eggs, if perfectly fresh, are usually acceptable prepared in various ways. It is very hard to obtain perfectly fresh eggs in a city; most city eggs are somewhat stale, and sometimes, if eggs are laid in musty hay, they acquire a disagreeable flavor; in either case patients may not be able to enjoy them. Eggs should not be fried.

Fat is an important article of diet for nervous patients. Often too little is taken. Cod-liver oil is excellent if it can be taken; if not, cream and butter may be used as substitutes.

Butter should not be heated above the boiling-point for water, and *should not be used for cooking.*

A powder of beef, which is made by J. Féré, imported by E. Fougera & Co., New York, forms a valuable article of diet in many cases. It can be mixed with water or milk; it is already cooked. The ordinary beef-teas and essences are of very small value as food.

Sometimes it may be necessary to feed with a stomach-tube. This may be passed twice or even three times a day, and it is much better to use it than to have a patient live half starved. In hysteria and insanity it is sometimes absolutely necessary to thus feed a patient.

When there is obstinate vomiting, it may be well to let the stomach rest a few days, giving only small pieces of ice; then begin with small amounts of food, gradually increasing.

Patients may be fed by the rectum, using partially digested meat, milk, or, better than these, an egg beaten up with ten grains of pepsin. Generally an egg or two can be given in this way every five or six hours; it is better to wash out the bowels once in twenty-four to forty-eight hours with a warm-water enema. Nutrient enemata should have a temperature of about 95° to 100°. If not too much exhausted at first, patients can be sustained for a fortnight or more in this way.

DISEASES OF THE BRAIN.

CHAPTER II.

INTRODUCTORY.

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ANATOMY.

The accompanying diagrams from Ecker, with explanations, will illustrate better than any verbal description the nomenclature of the convolutions.

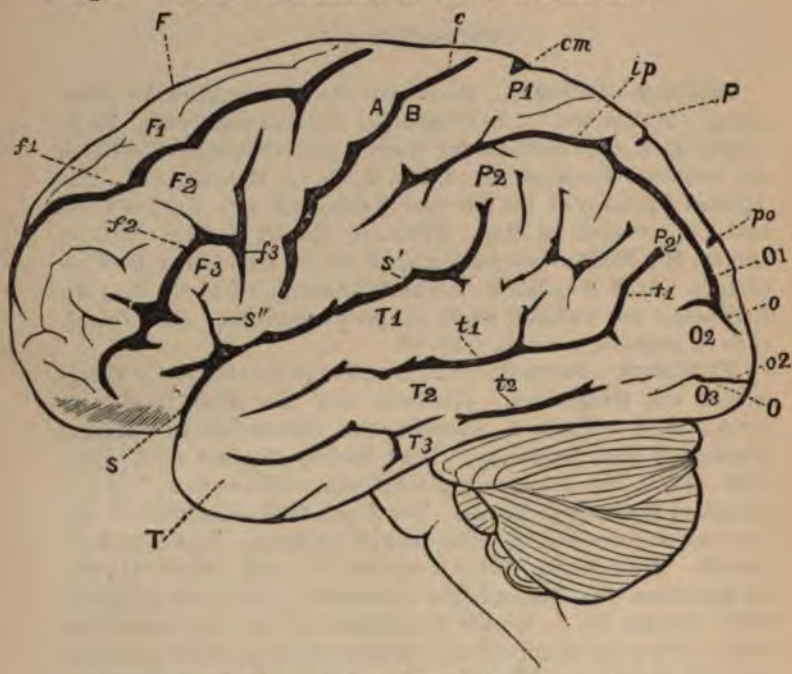

FIG. 1.—View of brain from the side. (ECKER.)

F, frontal lobe; *P*, parietal lobe; *O*, occipital lobe; *T*, temporal lobe; *S*, fissura Sylvii; *S'*, horizontal, *S''*, ascending branch; *c*, sulcus centralis, fissure of Rolando; *A*, anterior central convolution, ascending frontal convolution; *B*, posterior central convolution, ascending parietal convolution; F_1, upper (or first), F_2, middle (or second), F_3, lower (or third) frontal convolution. (Occasionally these are numbered from below upward. The above is the more generally received nomenclature.) f_1, upper, f_2, lower, f_3, vertical (præcentral) frontal fissure; P_1, upper, P_2, lower parietal lobule; P_2, gyrus supramarginalis (*s'* is on the same gyrus); P_2', gyrus angularis. (This passes around the posterior end of the first temporal fissure, uniting the first temporal and supramarginal convolutions with the second temporal convolution.) *ip*, sulcus interparietalis; *cm*, end of the sulcus calloso-marginalis; O_1, first, O_2, second, O_3, third occipital convolution; *po*, fissura parieto-occipital, internal perpendicular fissure; *o*, sulcus occipitalis transversus; o_2, sulcus occipitalis longitudinalis inferior; T_1, first, T_2, second, T_3, third temporal convolution; t_1, first, t_2, second temporal fissure. (t_2 is generally bridged, and so interrupted.)

In these diagrams only the important sulci are given; these vary somewhat in different brains; the convolutions between these sulci are subdivided by secondary sulci, whose arrangement is less constant. Occasionally one of the principal sulci may be bridged over by a convolution, causing an apparent irregularity.

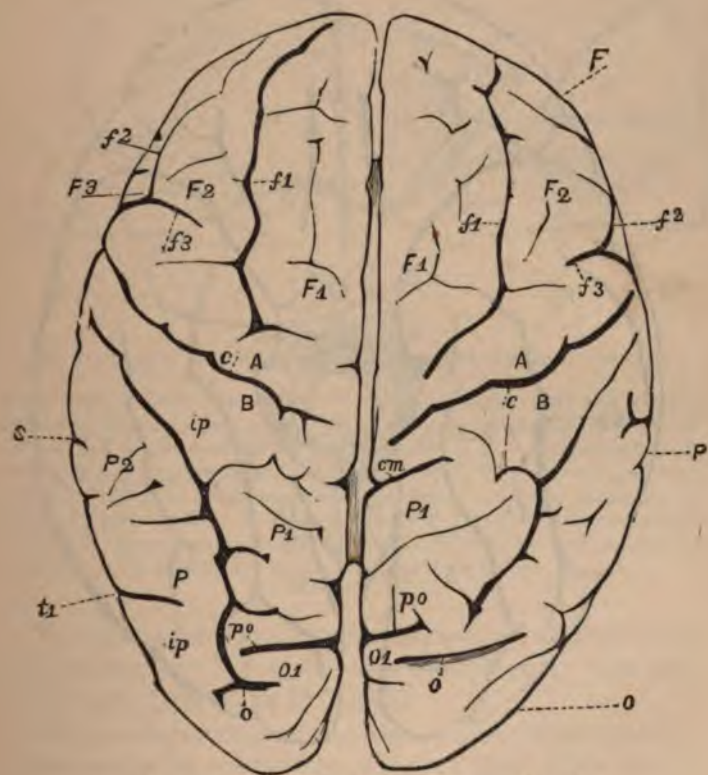

FIG. 2.—View of the brain from above. (ECKER.)

Lettering same as in Fig. 1.

From the cortex, medullary nerve-fibers pass through the white substance, the centrum ovale, converging toward the basal ganglia—*corona radiata*. They converge from all parts toward a tract of white substance

which separates these basal ganglia from each other—the internal capsule.

This capsule is one of the regions whose physiology is best known, whose lesion gives the most definite and permanent symptoms.

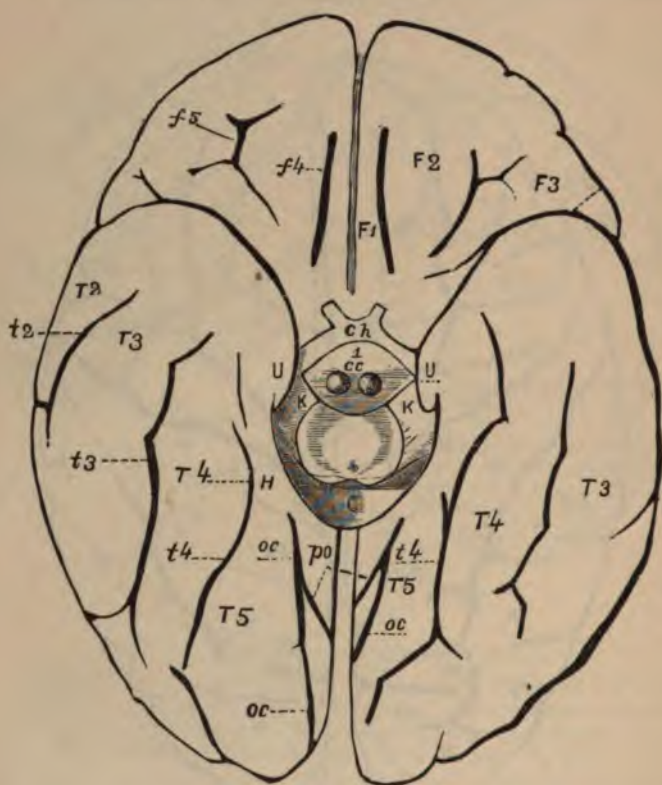

FIG. 3.—View of the brain from below. (ECKER.)

F_1, gyrus rectus, the prolongation of the first frontal convolution; F_2, middle, F_3, lower frontal convolution; f_4, sulcus olfactorius; f_5, sulcus orbitalis; T_2, second, or middle, T_3, third, or lower temporal convolution; T_4, gyrus occipito-temporalis lateralis (lobulus fusiformis), T_5, gyrus occipito-temporalis medialis (lobulus lingualis); t_2, middle, t_3, lower temporal fissure; t_4, sulcus occipito-temporalis inferior; *po*, fissura parieto-occipitalis; *oc*, fissura calcarina; *H*, gyrus hippocampi; *U*, gyrus uncinatus; *Ch*, chiasma; *cc*, corpora albicantia; *KK*, pedunculi cerebri; *C*, corpus callosum.

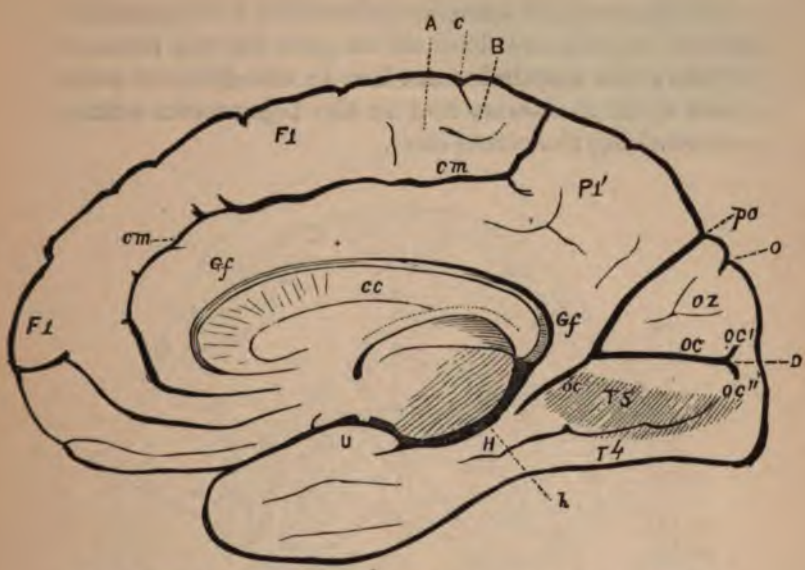

FIG. 4.—View of the medial surface of the right hemisphere. (ECKER.)

CC, corpus callosum, cut through the middle; *Gf*, gyrus fornicatus, *H*, gyrus hippocampi, *h*, sulcus hippocampi, *U*, gyrus uncinatus; *cm*, sulcus calloso-marginalis, F_1, first frontal convolution, its medial side; *c*, end of sulcus centralis, *A*, anterior, *B*, posterior central convolution; *Oz*, cuneus; *P'*, præcuneus; *po*, fissura parieto-occipitalis; *o*, sulcus occipitalis transversus; *oc*, fissura calcarina; *oc'*, upper, *oc''*, lower branch; *D*, gyrus descendens; T_4, gyrus occipito-temporalis lateralis; T_3, gyrus occipito-temporalis medialis (lobulus lingualis); around the central fissure is a quadrilateral lobule, *A*, *B*, called the paracentral lobule.

The accompanying representation, an outline from a photograph by Bitot, will give a sufficiently clear idea of the more important divisions.

The caudate nucleus and the outer or third member of the lenticular nucleus receive few fibers from the corona radiata, nearly all of whose fibers pass into the internal capsule. Fine bundles of white fibers pass from the lenticular nucleus into the internal capsule, and seem to pass on to the pyramidal tract. From the caudate nucleus many bundles of fibers pass into the anterior limb of the internal capsule; others cross this and enter the lenticular nucleus.

The fibers which have been described as entering the internal capsule are destined in part for the basis of the crus; the remainder are lost in the different parts of the optic thalamus, and in the tegmentum cruris, some reaching the cerebellum.

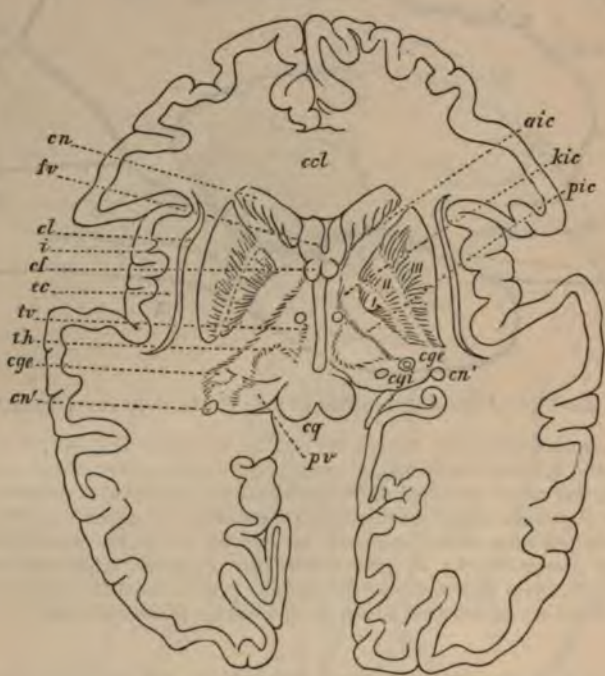

FIG. 5.—Horizontal section of the brain.

ccl, corpus callosum; *cn*, caudate nucleus; *fv*, fifth ventricle; *cl*, claustrum; *i*, island of Reil; *cf*, crura of the fornix, which, turning upon themselves, form the corpora albicantia; *ec*, external capsule; *tv*, third ventricle; *th*, optic thalamus; *cge*, external corpus geniculatum; *cn'*, the lower part of caudate nucleus; *pv*, pulvinar; *cq*, corpora quadrigemina; *cgi*, internal corpus geniculatum; I, II, III, the three divisions of lenticular nucleus (on the left only two divisions are seen); *aic*, *kic*, *pic*, the anterior limb, knee, and posterior limb of the internal capsule.

The following diagrams, in some respects slightly modified from Wernicke, will help to an understanding of the course of the most important bundles of fibers, and the relations of the ganglia.

Charcot has shown that only a part of the fibers of the internal capsule passes beyond the pons. The capsule is divided into an anterior and a posterior limb, the angle formed by the two being called the knee of

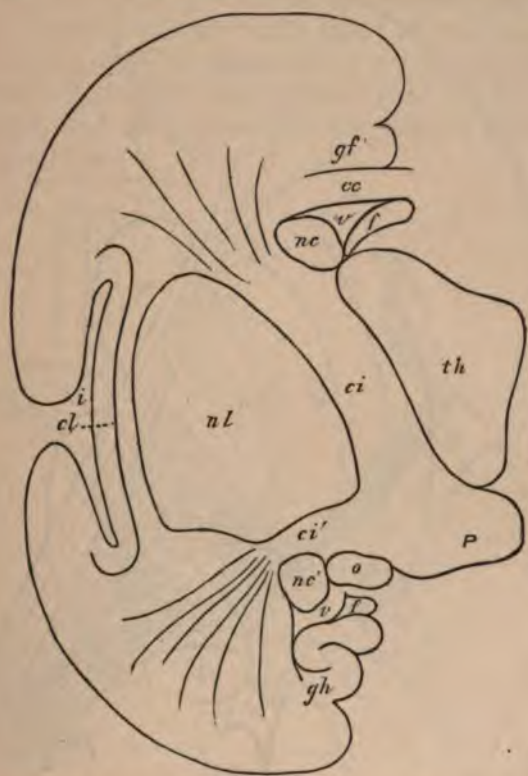

FIG. 6.—Diagram of a perpendicular section of the brain, showing the internal capsule and its relations.

gf, gyrus fornicatus; *cc*, corpus callosum; *v*, ventricle; *f*, fornix; *nc*, caudate nucleus; *th*, optic thalamus; *ci*, internal capsule, upper (anterior) limb; *p*, peduncle; *ci'*, internal capsule, lower (posterior) limb; *nc'*, lower part of caudate nucleus; *o*, optic tract; *gh*, gyrus hippocampus; *i*, island of Reil; *cl*, claustrum; *nl*, lenticular nucleus.

the capsule. The fibers from the anterior limb pass through the inner portion of the *basis cruris*. When a lesion implicates only these, the descending degener-

ation can be traced as far as the pons, but not beyond. That portion of the fibers of the basis which arises from

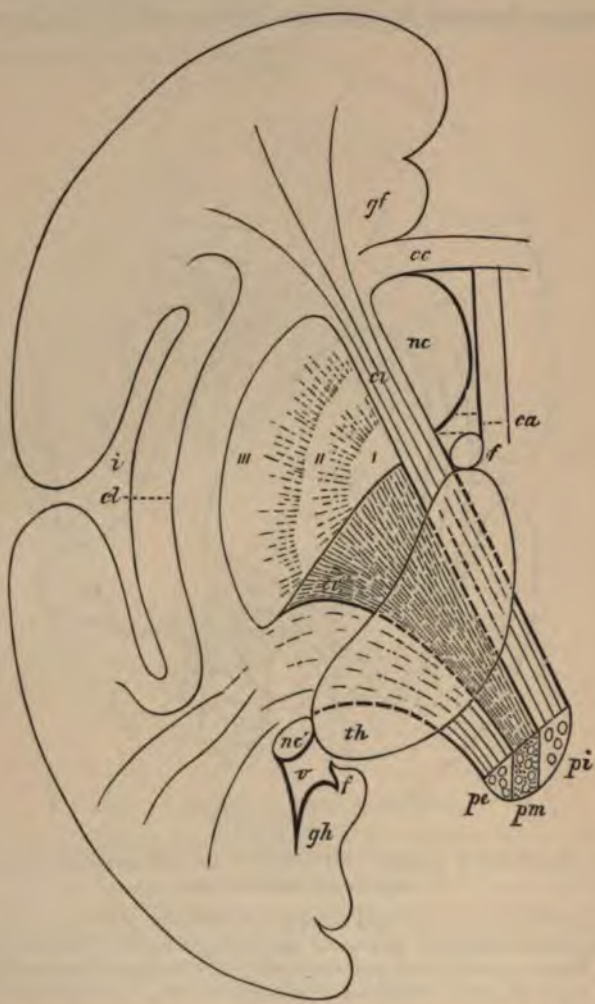

FIG. 7.—Diagram of a horizontal section of the brain, showing the course of the peduncular fibers.

ca, anterior commissure; I, II, III, the three divisions of lenticular nucleus; *pi*, *pm*, *pe*, the internal, middle, and external portions of the peduncular fibers passing below the optic thalamus. Other letters as in Fig. 5.

the inner two thirds of the posterior limb passes through the middle third of the basis, and secondary degeneration of these fibers can be followed through the pons and medulla into the pyramidal tracts of the cord. In Fig. 7 this portion of the internal capsule is shaded. Lesion of the outer third of the posterior limb of the capsule is followed by no descending degeneration; hence it is supposed that its fibers are centripetal, and they pass into the corona radiata of the occipital lobe.

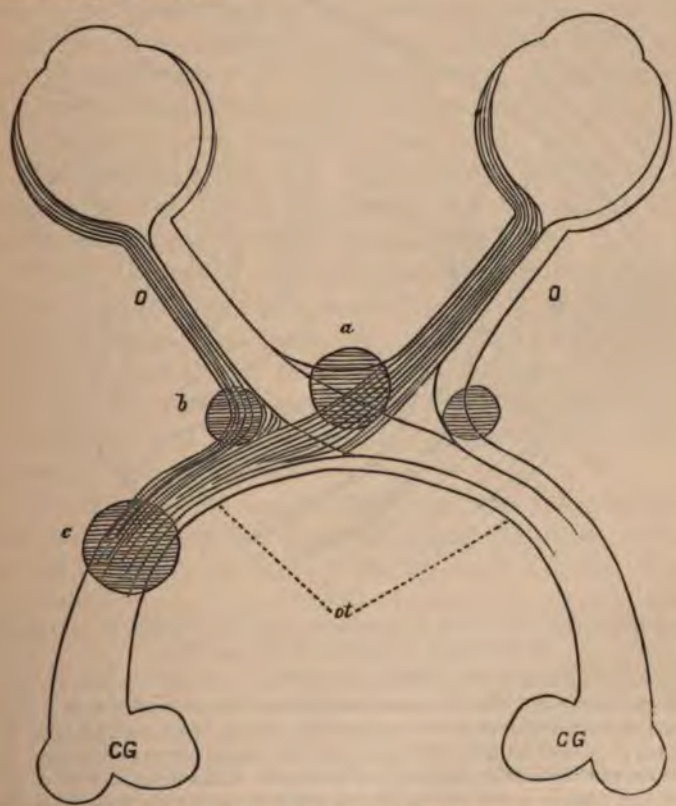

FIG. 8.—Diagram illustrating decussation of optic nerves in the chiasma, and the effect of lesions of portions of the optic tract.

o, optic nerves; *ot*, optic tracts; *cg*, corpora geniculata; *a*, *b*, *c*, lesions in front, at side of chiasma, and on optic tract.

The decussation of the optic nerves at the chiasma is only partial. The diagram on page 25 may give an idea of the arrangement of the fibers. The fibers from the right optic tract pass to the right side of both retinas, the larger portion decussating with those of the opposite side; and *vice versa* for those of the left optic tract.

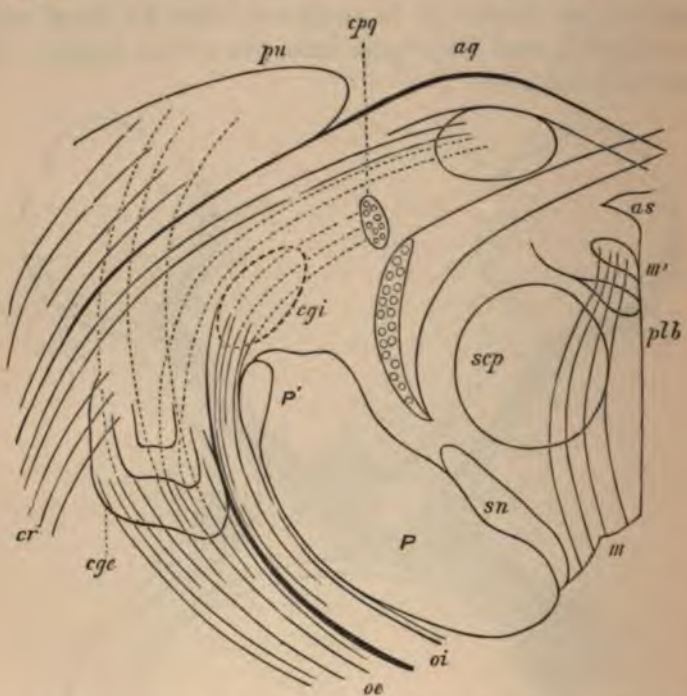

FIG. 9.—This is modified from Huguenin, to agree with Wernicke's description of the origin of the fibers of the optic tract.

aq, anterior corpus quadrigeminum; *pu*, pulvinar; *cpq*, crus of posterior corpus quadrigeminum; *cgi*, internal corpus geniculatum; *cge*, external corpus geniculatum; *cr*, posterior fibers of the corona radiata, passing to the occipital lobe; *oe*, external division of the optic tract, passing to the external corpus geniculatum, the pulvinar, and the anterior corpus quadrigeminum; *oi*, the inner division of the optic tract, passing to the internal corpus geniculatum, and the posterior corpus quadrigeminum; *p*, peduncular fibers; *p'*, the hemispherical bundle seen in transverse section of the crus cerebri; *sn*, substantia nigra; III, third nerve; III', its nucleus; *scp*, superior cerebellar peduncle (red nucleus); *plb*, posterior longitudinal bundle; *as*, aqueduct of Sylvius.

When an attempt is made to study the optic tract, tracing its fibers to their central origin, we meet some opposing statements made by different authors. Wernicke describes the optic tract, passing from the chiasma backward, as dividing into two portions—an external, which is much the larger, and a smaller internal; the former can be traced to the external corpus geniculatum, the posterior portion of the optic thalamus called the pulvinar, and the anterior corpus quadrigeminum; the internal portion, he says, arises from the internal corpus geniculatum, and the posterior corpus quadrigeminum; this division, he says (following v. Gudden), has no connection with the optic nerve. A bundle of fibers seems to pass directly from the corona radiata of the occipital lobe to the optic tract.

The preceding diagram, Fig. 9, which follows Wernicke, is modified from Huguenin, and will aid in understanding these divisions of the optic tract.

The advantage of following the different bundles of fibers through the crura cerebri, pons, and medulla would be, to say the least, very doubtful. Huguenin, Wernicke, and Duval have given us studies of this region. A few outline drawings or diagrams, with brief explanation, will serve to localize the important nerve-centers at different levels.

The third nerve enters the crus near the anterior border of the pons, not far from the median line, forming the internal boundary of the pyramidal fibers. The nerve splits up into bundles of fibers which diverge, the inner bundles following a slightly waving course backward to the nucleus; the outer bundles, forming a curve, pass through the outer edge of the cerebellar peduncular fibers (the red nucleus), then converge to the nucleus, which is situated on either side of the median line just anterior to the aqueduct of Sylvius. Anterior to the nucleus the nerve passes through the posterior longitudinal fasciculus. The nucleus of the third is irregularly pear-shaped, the smaller end pointing forward,

lying near the median raphé. Curving around from the posterior longitudinal fasciculus can be seen the descending (motor) root of the fifth nerve. Posterior to these is the anterior corpus quadrigeminum, and external to this the internal corpus geniculatum.

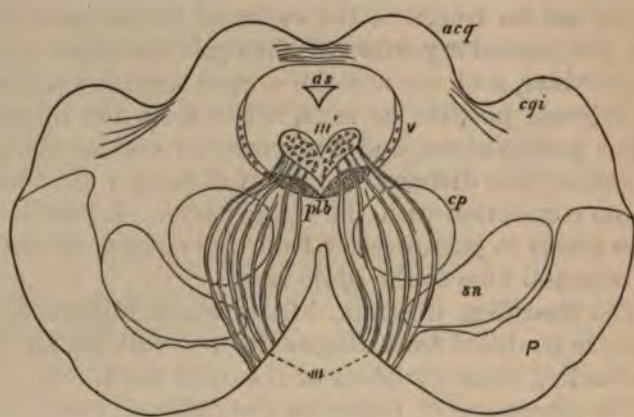

FIG. 10.—Origin of third nerve. (After WERNICKE.)

as, aqueduct of Sylvius; *plb*, posterior longitudinal bundle; III, third nerve; III', its nucleus; *p*, basis of crus cerebri (foot of the peduncle); *sn*, substantia nigra; *cp*, superior cerebellar peduncle (red nucleus); *cgi*, internal corpus geniculatum; *acq*, anterior corpus quadrigeminum; V, descending (motor) root of trigeminus.

The fourth nerve enters the valve of Vieussens just behind the posterior corpus quadrigeminum, decussates in the valve, curves around the aqueduct of Sylvius, and enters its nucleus, which is situated just behind (below) the nucleus of the third nerve in relatively the same position.

Both the third and fourth nerves receive a few fibers, ascending from the sixth nerve, from the posterior longitudinal fasciculi, which do not enter their nuclei.

The nucleus of the third nerve receives fibers from the anterior corpus quadrigeminum, and probably similar fibers pass to the nuclei of the fourth and sixth nerves.

The sixth and seventh nerves are closely connected at one point in their course. The sixth enters the anterior aspect of the pons just at its junction with the medulla oblongata not far from the median line. It crosses the pons, changing its direction several times so that no one section can follow its whole course, and enters its nucleus just external to the eminentia teres near the median raphé on the floor of the fourth ven-

FIG. 11 represents sections through the pons, so as to show on the left the seventh and sixth nerves, with their nuclei; on the right, the sixth and eighth nerves, half schematic.

VII, seventh nerve; VII', eminentia teres, where the seventh nerve turns downward; VII'', the proper nucleus of the seventh nerve; VI, sixth nerve; VI', the common nucleus of sixth and seventh; VIII, eighth nerve; VIII', its nucleus; V, ascending root of the fifth nerve; *r*, restiform body; *so*, the superior olive; *p*, the peduncular fibers.

tricle. This nucleus is also the origin of some of the fibers going to the seventh nerve, which enters the pons just anterior (forward) to the eighth, crosses diagonally in a gentle curve to this nucleus, from which it receives some fibers; then it can be followed to the eminentia teres, where it turns downward; it soon turns again to pass forward and outward, slightly downward, to its inferior nucleus. The fibers, in passing from the eminentia teres to this nucleus, divide and separate more or

less widely from each other, and form a wide network rather than a compact bundle. The nucleus is composed of three or four groups of cells, in each section, which are each surrounded by separate bundles of fibers. Just outside the facial nerve, and anterior to its inferior nucleus, is the ascending root of the fifth nerve.

From the nucleus of the sixth nerve thin bundles of fibers pass forward (upward) in the posterior longitudinal fasciculus to the third and fourth nerves. These communicating fibers decussate in their course quite near the point where they unite with the other fibers of the third and fourth nerves.

The course of the eighth nerve is not as yet of so

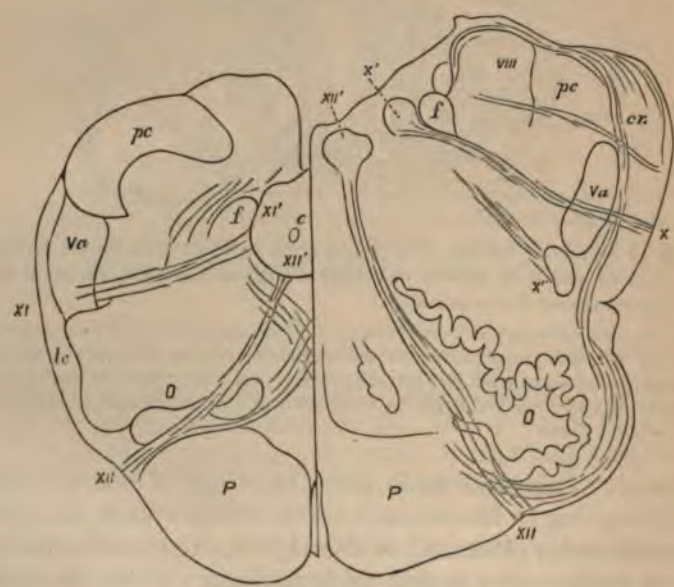

FIG. 12.—Transverse section of the medulla on the right at a higher level than on the left. (After WERNICKE.)

P, the anterior pyramidal fibers; *O*, the inferior olivary body; *lc*, the lateral column; *cr*, the restiform body; *pc*, the posterior columns; *f*, longitudinal fibers; *c*, central canal; *Va*, ascending root of the fifth; X, XI, XII, the corresponding nerves; X', XI', XII', their nuclei; X'', anterior vagus nucleus.

much practical interest. It enters the pons just behind (below) the seventh, and divides into three bundles, which are distributed to separate nuclei. It is scarcely necessary to give the particulars of its deep origin.

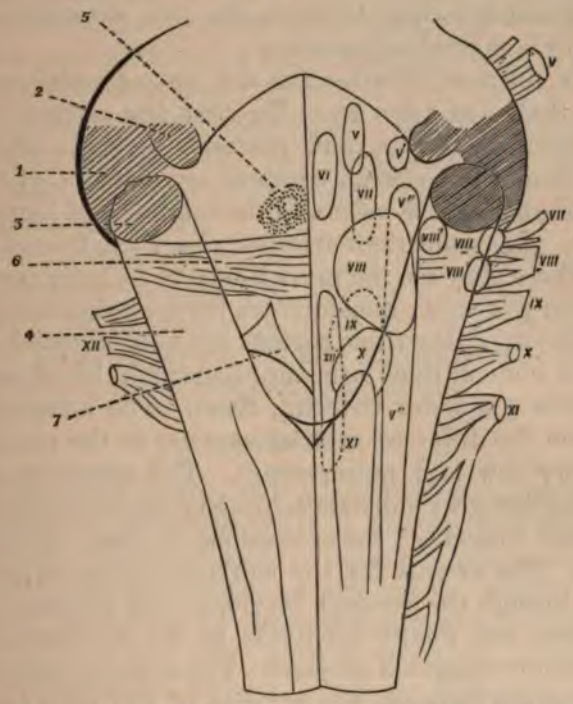

FIG. 13.—Schematic view of the relative situations of the nuclei in the medulla. (After ERB.)

V–XII, the nerves or their nuclei; 1, middle, 2, superior, 3, inferior cerebellar peduncle; 4, restiform body; 5, eminentia teres; 6, acoustic fibers; 7, ala cinerea.

The relations and origins of the nerves of the medulla oblongata can be easily learned by a study of the accompanying figures (12 and 13), and it is scarcely necessary to give more detailed descriptions.

The distribution of the blood-vessels in the brain has been studied especially by Duret. The results obtained are of much practical value. The circle of Wil-

lis at the base of the brain furnishes rather free communication between the carotids and the vertebrals of the same side, and between the arteries at the posterior part of the brain on opposite sides, unless there are anomalies in the size of the arteries. Anteriorly, the only communication between the two carotids is the anterior communicating artery.

The nutrient arteries for the corpus striatum and optic thalamus arise from the first few centimetres of the anterior, middle, and posterior cerebral arteries; those from the posterior cerebral are distributed to the optic thalamus, the others to the caudate and lenticular nuclei. These nutrient arteries arise, then, from a position where they are the more likely to feel any increase of blood-pressure; anastomoses between the secondary arteries supplying these regions are very unusual.

The convolutions are supplied with blood-vessels from the pia mater covering them. The anastomoses between the different arterial systems in the pia mater are very few and unimportant. The arterioles, after entering the gray substance, quickly subdivide into the minutest branches; these anastomose freely with each other. The vessels for the subjacent white substance pass through the cortical layers, giving off only a few branches, and finally subdivide in the medullary substance into elongated meshes. There are no important anastomoses between the arteries of the convolutions and those of the large ganglia at the base, even where the two come very near together, as in the corpus striatum opposite the *insula*.

The veins anastomose more freely, and are so disposed with reference to the arteries of the convolutions that the blood is delayed, especially in the arterioles in the gray substance, and the nerve-cells are continuously bathed in arterial blood.

As the arteries branch at nearly right angles, and subdivide rapidly into very small vessels, the blood-pressure is diminished; but the absence of communi-

cations between the larger branches does not allow of the removal of pressure from weakened arteries.

Most of the first and second frontal convolutions, and the convolutions in the median fissure as far back as the termination of the sulcus calloso-marginalis, are supplied by the anterior cerebral arteries. The third frontal convolution, the anterior central and posterior central, and very nearly all the parietal and the upper part of the temporal lobes and the insula, are supplied by the middle cerebral. The lower portion of the temporal and the occipital lobes are supplied by the posterior cerebral artery.

One of these arteries, or any of their branches, may be obstructed mechanically, or may be subject to temporary reflex spasms, so as to interfere with the proper flow of blood. Hyperæmia may also affect any one district, leaving the others nearly unaffected.

PHYSIOLOGY.

Fritsch and Hitzig (1870) first called attention to the irritability of certain districts of the cerebral cortex as suggesting the localization of motor functions in separate and distinct regions of the brain. Ferrier soon after (1873) published observations made in the same direction, and has since greatly extended our knowledge. During the last ten years the literature of the subject has increased wonderfully.

The region of the cortex immediately anterior and posterior to the fissure of Rolando has been found to be excitable; an irritation applied to this region causes motion in the voluntary muscles, according to the locality of the irritation. Other regions may be excitable, and probably are, but the above central region is the one which seems to act most directly upon the limbs. When motion is produced by irritation of other regions, it is probably indirect. Other regions than these may, when irritated, cause various sensations or give rise to mental actions; but these are not revealed to us by

motor phenomena. A careful study of Ferrier's plate, with the motor centers marked on Ecker's diagram of the convolutions, will be all that is needed to fix these centers in the mind.

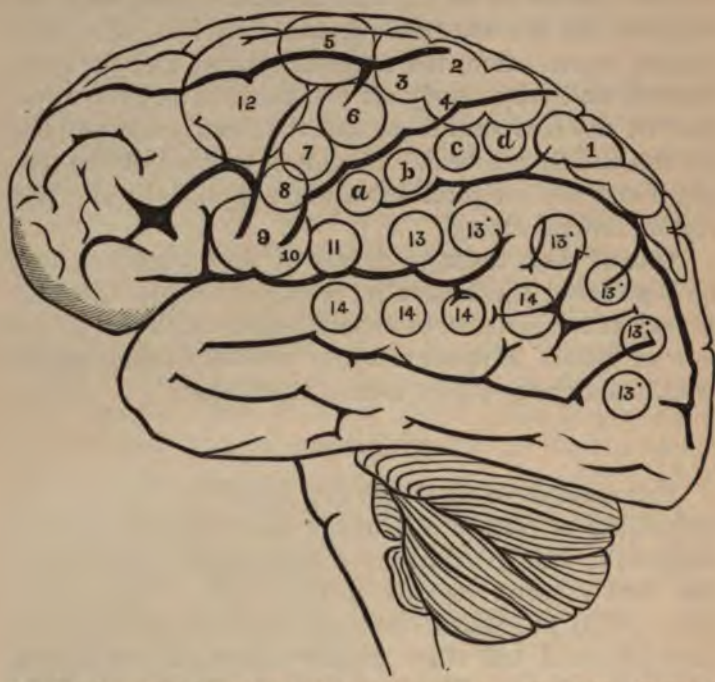

FIG. 14.—Location of motor and other centers in the cerebral cortex. (FERRIER.)

1, on the upper or superior parietal lobule center for the opposite leg; 2, 3, 4, around the upper end of central fissure, centers for opposite leg, arm, and trunk; 5, centers for motion of opposite arm and hand forward; *a*, *b*, *c*, *d*, on the posterior central convolution, centers for motions of fingers and wrist of opposite hand; 6, supination and flexion of the opposite forearm; 7, 8, 9, 10, 11, on the anterior central convolution and around the base of central fissure, motions of mouth, lips, and tongue (9, 10 are called oro-lingual centers by Ferrier); 12, elevation of eyelids, dilatation of pupils, conjugate deviation of eyes, and turning of head to opposite side; 13, 13', centers which seem to have relation to vision and cause motions of the eyes; 14, centers which seem to be concerned with hearing, and give rise to motions expressive of attention.

The views as to the sensory centers are still unsettled. It is much more difficult to locate these; and, indeed, the facts as yet known rather tend to show that

the centers for different sensations are much less clearly defined than those for motion.

Ferrier locates the centers for sensation in the parieto-temporal region; sight in the supra-marginal and angular convolutions (13, 13′); hearing in the superior

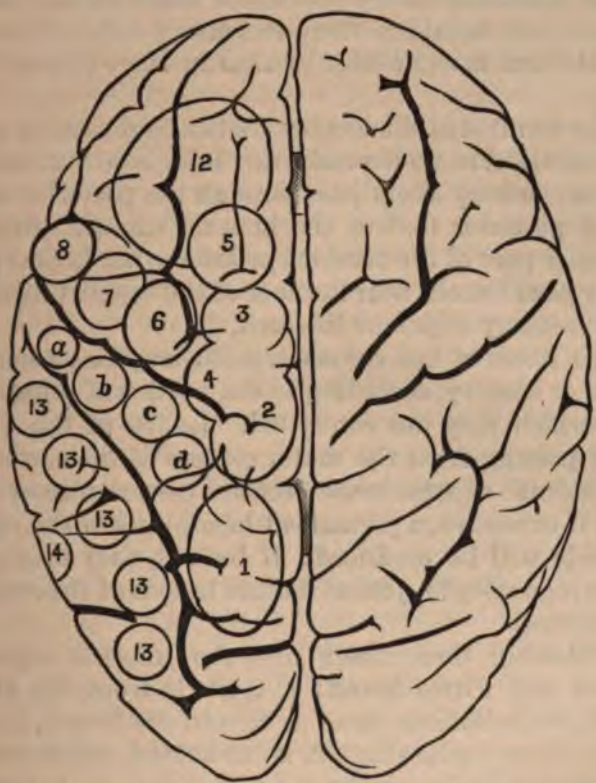

Fig. 15.—Location of motor and other centers in the cerebral cortex. (Ferrier.)
Lettering same as Fig. 14.

or first temporal convolution (14); taste and smell in the lower extremity of the temporo-sphenoidal lobe—region of the subiculum cornu Ammonis; tactile sensation in the region of the hippocampus. The centers for sight and hearing are probably correctly located, but there are also centers for sight in the occipital lobe

quite as important as those in the angular and supra-marginal convolutions. There is some uncertainty as to the other sensory centers.

Petrina is of opinion that the faculty of sensation is more generally diffused, and that at least the fibers for tactile sensation follow the motor fibers to the motor centers, and terminate there in sensory cells. Thus he regards each motor center as also in some degree sensory.

The location of the centers for tactile sensation must be considered as undetermined. It is, however, settled that the sensory fibers pass through the posterior third of the posterior limb of the internal capsule, through the outer part of the cerebral peduncle, the lateral part of the pons Varolii near the floor of the fourth ventricle, to the sensory region of the cord.

The fibers of the corona radiata may be classed as motor or sensory, according to the portion of the cortex with which they are connected. Lesion of the white fibers passing from the motor centers of the cortex to the internal capsule causes serious disturbance of motion; if extensive, a permanent hemiplegia of the opposite side will be produced; if limited, they may give rise to monoplegias, just as limited lesions of the cortical motor zone.

Following these fibers into the internal capsule, Franck and Pitres found: 1. Quite in front the fibers which, on irritation, cause motion in the face and eyelids on the opposite side. 2. Next behind, the fibers for the anterior limbs on the opposite side. 3. A bundle of fibers which move both limbs on the opposite side. 4. A very small bundle for the opposite hind limb. 5. In the posterior part of the caudate nucleus those which cause elevation of the opposite ear.

These experiments were made upon animals; clinical observations and post-mortem examinations must finally show how nearly the same order is followed in man.

The functions of the ganglia at the base are not yet well determined. We know nothing in regard to the functions of the caudate and lenticular nucleus; they do not seem to have any direct control over either motion or sensation, which has as yet been discovered.

The functions of the optic thalamus are still a subject for investigation and discussion. Nothnagel, from experiments on animals, concludes that motor impulses, which are excited, or depend upon peripheral sensory impressions, take their origin in the optic thalami.

Wernicke's conclusions agree very nearly with this view. "They serve, 1, for acquiring consciousness of motions through 'muscular sense' or innervation's sense; 2, for the involuntary adaptation of our motions to external relations by means of the reflex mechanism which they contain; 3, as a way for certain sensory tracts, which, according to Meynert, serve for transmission of muscular or innervation's sense."

The posterior portion of the optic thalamus, the pulvinar, forms a part of the visual centers; it stands in intimate relations with the corresponding halves of both retinas, as is shown by the symptoms in cases where it is destroyed. Exactly how these relations are maintained is not yet definitely known.

The physiology of the pons, medulla, and neighboring parts can be inferred from the anatomical descriptions given above, or will be mentioned more conveniently in connection with the symptoms due to their lesion in the following sections.

GENERAL SYMPTOMATOLOGY.

Lesions affecting the brain may be divided into two classes, destructive and irritative. These may act only upon the nerve-fibers which they immediately affect; or they may exert an influence at a distance, which may be different in its nature from their immediate influence. A hæmorrhage will destroy nerve-fibers, which

are torn across; it will also, if of sufficient size, interfere with the function of others by compressing them, and it may give rise to symptoms of irritation. A tumor may irritate certain fibers, and, by compression, prevent the action of others, or it may destroy those among which it grows, and irritate others at a distance. As a rule, a lesion which occurs suddenly, as a hæmorrhage, will give rise to more symptoms depending upon interference with distant regions than those lesions which slowly increase in size. It may be necessary, then, in forming an opinion as to the seat of a lesion, to wait until the commotion produced by the first shock of the disturbance has subsided and the remote symptoms have disappeared.

Destruction of the motor centers, or of the white fibers of the corona radiata underlying these centers, is revealed by paralysis of the limbs or muscles over which they preside. Irritation of these parts gives rise to spasm of those muscles. It may happen that there is first an irritation due to the destruction of fibers, shown by a spasm; then follows the paralysis dependent upon the permanent lesion. If the irritation is very strong, it not only excites to activity the center on which it first acts, but neighboring centers, and perhaps the whole motor area of both hemispheres, may be thrown into commotion; then general convulsions will result.

By a careful study of the seat of the paralysis or convulsion, and a comparison of these with the motor centers, a reasonably accurate conclusion as to the seat of a cortical lesion may be formed. Spasms confined to one limb are much more clearly diagnostic of cortical lesion than paralysis. When convulsions are general, the diagnosis is less certain; but, by watching the commencement of the attack, noting that invariably the same limb is first affected each time, and that the convulsions follow the same course, a correct diagnosis may often be made.

The caudate nucleus and the lenticular ganglion may be nearly or quite destroyed without any disturbance of motion or sensation. Generally, however, especially if the destruction is produced by a hæmorrhage, there is more or less compression of neighboring parts, the internal capsule is thereby disturbed, and symptoms result therefrom. Often, also, a portion of the capsule is destroyed, and then the symptoms are permanent. If the destruction is limited to the gray ganglia, as the clot is absorbed the pressure upon the capsule is removed, and the symptoms may entirely disappear.

The *optic thalamus* may be partially destroyed without special symptoms; those which occur seemingly depend upon pressure or implication of neighboring parts. At other times there are disturbances of sensation which seem to arise directly from the lesion of the thalamus. Injuries to the optic thalamus may cause more or less disturbance of vision. When the anterior or middle portions are injured, this disturbance, if present, is temporary; if the posterior part, the pulvinar, is injured, the loss of vision is permanent, and generally affects only one half the visual field—hemianopsia.

There is a small region, *carrefour sensitif*, lying external to the pulvinar, which serves as the course for sensory fibers. Lesion of this region will give rise to hemianæsthesia; and when there is disease of the optic thalamus or pulvinar, this region would very likely be disturbed.

A peculiar motor disturbance is associated with injury of the posterior and outer part of the optic thalamus, and perhaps adjacent parts, though some observations seem to show that lesion of the thalamus alone is sufficient to produce the phenomena. The partially or entirely paralyzed limbs are in a state of unrest; they keep up a constant motion, which may be simply a tremor, or a slow, irregular motion in all directions

without co-ordination or rhythm, sometimes resembling chorea, sometimes peculiar and unlike any other motor phenomena. This has been called *athetosis*, or *post-hemiplegic chorea.*

The *internal capsule* is the most important part of the base of the brain, so far as relates to the symptoms arising from its destruction.

When the anterior limb of the internal capsule is destroyed, secondary degeneration affects only the internal segment of the base of the crus, and this degeneration can not be followed beyond the pons. When the anterior two thirds of the posterior limb are destroyed, there is descending degeneration, which can be followed through the middle segment of the crus, the pons, medulla, and anterior pyramids, where it crosses to the opposite side of the cord, excepting a small portion, the anterior pyramidal fibers, which pass downward on the same side. When the outer third of the posterior limb is destroyed, there is no descending degeneration. Hence we conclude that the most, if not all, of the motor fibers for the trunk and limbs from the motor area of the brain pass through the anterior two thirds of the posterior limb of the internal capsule, and follow the course taken by the descending degeneration.

When the anterior limb of the internal capsule is destroyed, there is no paralysis of the limbs; the facial nerve may be paralyzed.

When the anterior two thirds of the posterior limb are destroyed (or pressed upon so as to interfere with function), there is always paralysis of motion in the opposite side of the body. This paralysis is permanent, and is followed by late contraction, due to secondary degeneration of the pyramidal tract.

When the posterior third of the posterior limb is destroyed, there is anæsthesia of the opposite side; Charcot calls this cerebral hemianæsthesia, and says it is a faithful reproduction of the characteristics of hysterical hemianæsthesia; the insensibility extends to the

profound parts, muscles, mucous membranes; it involves also the sensory apparatus, the senses of taste and hearing, of smell and sight. The sight is lost by a concentric narrowing of the field of vision; the perception of colors is lost—first green, then red, orange, yellow, and blue, until everything has a grayish hue.

When disease of one hemisphere causes amblyopia or amaurosis, the disturbance of vision is observed in the opposite eye; a slight defect may also be discovered in the corresponding eye if carefully sought, provided the intelligence of the patient is not too much disturbed. The lesion is then situated in the posterior third of the posterior limb of the inner capsule, or in some portion of the cortex which is not yet exactly located, but which probably corresponds to Ferrier's centers (13, 13′) in the angular gyrus, or it may be in some portion of the occipital lobe.

Many times the loss of vision affects only one lateral half of the visual field. This is called *hemiopia*, in speaking of the loss of power in the retina, or more usually *hemianopsia*, as referring to the visual field.

When there is double temporal hemianopsia—the loss of sight affecting the temporal half of both visual fields—the lesion must be in the chiasma, probably near its anterior border, at *a*, Fig. 8.

When the nasal half of the visual field is lost—nasal hemianopsia—the lesion must be on the corresponding side of the chiasma, *b*, Fig. 8; both sides are rarely thus affected. If there is amblyopia or amaurosis of one eye and nasal hemianopsia of the other eye, the lesion must be on the side of the chiasma corresponding with the amblyopic eye, and penetrate deep enough to affect the decussating fibers.

When there is loss of vision for corresponding lateral halves of both visual fields, the defect is called lateral homonymous hemianopsia. This may be caused by lesion of the opposite optic tract (*c*, Fig. 8), of the pulvinar, of the occipital lobe, or possibly of the

angular gyrus, or of the fibers passing from the pulvinar to the centers of vision. If there is no reaction of the pupil when light falls upon the affected half of the retina, the lesion must be in the optic tract or the anterior corpus quadrigeminum—i. e., in right lateral hemianopsia the left optic tract would be affected.

If the pupil reacts when light falls upon the affected half of the retina, the lesion is either in the pulvinar, in the corona radiata, or the cortex. When the pulvinar is the seat of a hæmorrhage, there may be other symptoms of apoplexy, motor or sensory; these will disappear in a short time, but the hemianopsia will be permanent. There may be photopsia.

When the corona radiata leading to the centers of vision is the seat of hæmorrhage, the hemianopsia will at first be complete, but soon the vision will be partially restored; there will be other apoplectic symptoms. If the loss of sight is caused by tumor, it will come on gradually, and will be accompanied by other symptoms —paralysis of cranial nerves, etc.—showing the nature and seat of the lesion. Photopsia may be present.

When the cortical centers of vision are affected, there will be no photopsia; the hemianopsia may be complete or only partial; there may be loss of perception of color in one half of the visual field, with power to perceive white. There will be no other paralysis except vision.

Charcot and his followers claim that lesion of the hemispheres is more frequently followed by amblyopia, contraction of the field of vision, and that hemianopsia is rare. Féré thinks the lesion causing hemianopsia must be situated between the angular gyrus and the fissure of Rolando, or in the medullary fibers leading from that region. We do not yet know why in one case there is hemianopsia and in another amblyopia.

When the lesion is situated below the internal capsule, so that the *crus cerebri*, or parts below, are implicated, the cranial nerves may be affected on the same side with the lesion. The symptoms may then be in

part on the same side, in those nerves directly affected; in part on the opposite side, for the nerves arising below the seat of the disease. This gives a crossed or alternate paralysis. A comparison of the symptoms with the physiology and anatomy of this part of the mesencephalon will lead to as correct a diagnosis of the location of the lesion as can be made.

Lesions of the *anterior corpora quadrigemina* are attended with partial or total loss of vision. If the lesion is unilateral, the loss of vision is also unilateral. Opinions differ as to whether there is simple unilateral amaurosis or lateral hemianopsia.

Lesions of the *posterior corpora quadrigemina* are associated with inco-ordination, which Nothnagel says is like that found in lesion of the cerebellum, and which is possibly dependent upon a coincident lesion of the superior cerebellar peduncle.

These symptoms alone would not be sufficient to locate the lesion in the corpora quadrigemina. There will often be found other symptoms due to extension of the lesion to neighboring parts.

The third and fourth nerves, with their nuclei, lie just below the corpora quadrigemina, and may be implicated; then there would be partial or entire paralysis of these nerves. The motor and sensory tracts pass through the crura cerebri, and may be implicated, giving rise to corresponding symptoms.

The third nerve supplies many different muscles. Its most anterior fibers are for accommodation; just behind these are the fibers which innervate the sphincter of the iris; then are arranged the fibers for the internal rectus, the superior rectus, the levator palpebræ superioris, the inferior rectus, and the inferior oblique.* When the

* Kahler and Pick suggest that the posterior bundles supply the levator palpebræ, rectus superior, and obliquus inferior—muscles which are employed together in looking upward; that the posterior median bundles innervate the internal rectus and the inferior rectus; the anterior, the pupillary, and accommodation muscles.

nucleus or the nerve in its course through the peduncle is affected, the symptoms will guide to a diagnosis of the seat of the disease. Kahler and Pick say a disassociated, partial paralysis of the oculo-motor nerve points primarily to a lesion of the territory through which the roots of this nerve run.

Most significant for lesion of the cerebral peduncles is the alternate paralysis of the motor-oculi nerve. The limbs and face, perhaps also the tongue, are paralyzed on one side, opposite to the lesion; the ocular muscles are paralyzed on the other side, the same side with the lesion. Great disturbance of sensation would indicate that the outer portion of the crus was affected.

In case of disease of the *pons Varolii* a few symptoms are important; among these, alternate paralysis, the limbs affected, as to motion and sensation, on the side opposite the lesion, the cranial nerves on the same side with the lesion; whether the facial, the fifth, the sixth, are all or only in part paralyzed on the side opposite the limbs, depends upon the height at which the disease is situated. If there is thus alternate paralysis, the disease is almost sure to be in the pons, sometimes it may be in the medulla oblongata.

A conjugate deviation of the eyes to one side is diagnostic of lesion of the pons, when, in case of paralysis, the eyes are turned toward the hemiplegic side; in case of spasm, away from that side. This symptom points to the vicinity of the nucleus of the sixth nerve as the locality of the lesion.

Conjugate deviation of the eyes is found, associated with deviation of the head to the same side as the eyes, in cases of hæmorrhage into the hemispheres. Then the symptom is of only short duration; in a few hours or a few days the motions of the eyes and head become free. Under these circumstances it is of little value in locating the lesion; it may accompany superficial lesions, or those which involve the cerebral substance, becoming more frequent as the lesion is situated nearer

the corpus striatum and the fibers radiating from the cerebral peduncle. The deviation is away from the paralyzed side, toward the affected hemisphere; when there are convulsions, the deviation is toward the side convulsed.

When conjugate deviation of the eyes occurs in disease of the pons, it is usually independent of any deviation of the head, and is persistent.

In examining the eyes, it is necessary to examine them together, and each one separately. In paralysis due to disease of the pons, the abducens on the same side with the lesion is paralyzed alternately with the limbs. The internal rectus then draws the affected eye toward the opposite side. The internal rectus of the other eye, on account of the union of the sixth nucleus with the third nerve, being paralyzed, so far as acting in harmony with the opposite abducens, allows that eye to be drawn outward by its external rectus. As the fibers of the third nerve, which control the direction of the eyes for near vision, are not paralyzed, being independent of the sixth nerve, both eyes will turn inward naturally when an object is slowly approached to the eyes. When examined separately, the eye whose abducens is paralyzed can not be turned outward beyond the median line; the other eye can be turned inward, though, it may be, only with considerable effort. In this separate examination of each eye some nystagmus may be noticed.

When the abducens alone of the ocular nerves is paralyzed, the pons must be the seat of the disease, unless other symptoms show that the lesion is peripheral.

When only one pupil is persistently contracted, lesion of the pons on the same side is indicated.

When speech is affected, it is in the form of anarthria.

The sensory fibers are said to pass through the lateral portions of the pons, and great or total loss of sen-

sation in disease of the pons would point to that portion as its seat.

Compulsory motions and ataxia would be rather indicative of lesion of the cerebellar peduncle than of the pons, though Penzoldt noticed compulsory motion backward when the middle portion of the pons was affected.

When the *cerebellum* is the seat of a tumor, many symptoms will be caused by pressure upon neighboring parts; also in case of hæmorrhage the earlier symptoms may be in part due to the effects of influences acting upon other regions. In disease of the cerebellum, vomiting is a very common symptom; optic neuritis is frequent; the ventricles may be distended with serum. The most characteristic symptom is motor disturbance, a general weakness and a disturbance of co-ordination, with vertigo and uncertainty of gait. This disturbance of gait is found only when the vermiform process or the middle lobe of the cerebellum is affected. When the middle lobe is diseased, there is likely to be priapism. The cerebellar hemispheres may be extensively diseased without causing characteristic symptoms.

The symptoms at a distance due to pressure, etc., will generally be a great aid to diagnosis, and without these it may be impossible to locate the disease.

The symptoms caused by lesion of the nuclei in the medulla oblongata will be described under Bulbar Paralysis.

DISTURBANCES OF SPEECH.

Speech may be interfered with in several ways: 1. The power of transforming thought into words may be lost (*amnesic aphasia*); the patient may yet understand what is said and be able to read. The loss of speech may be absolute, or, as is more common, the patient may retain the power of speaking certain words or phrases; these expressions are usually "yes" and "no," which may be used correctly, but quite as often

incorrectly; certain expressions which are of the nature of exclamations and oaths may be retained. The extent to which speech is affected varies greatly; sometimes patients can repeat a sentence spoken in their hearing, sometimes can read aloud, yet immediately after are unable to say the same words; sometimes they can spontaneously speak a part of a sentence, or they may occasionally be at a loss for a word, and recognize it when suggested. Sometimes only one class of words is lost, as nouns, or words expressing relations or adjectives.

When the loss of the faculty of recollecting words is marked, there is also a loss in the power of expressing thought by gestures (*amimia*), and the patient can not write, even if the motions of the hand would permit writing (*agraphia*). They are unable to write with the left hand. In attempting to write, sometimes only lines are made, sometimes separate letters, but not words; or words may be written, but not to form sentences.

2. The patient may have the power to change thought into words, but the connection between the center where this is done and the vocal organs is interrupted, and the patient can not express himself. This is called *ataxic aphasia*, or *anarthria;* the lesion may be at any point between the speech-centers and the vocal organs. When, however, the nuclei of the nerves in the medulla are diseased, peculiar difficulties of utterance are produced which are not properly included under this title, and which will be considered in connection with bulbar paralysis.

Under the name *dysarthria* may be included certain disturbances of speech, as stuttering, explosive speech, scanning speech, trembling speech, etc.

When there is anarthria, ataxic aphasia, the patient is unable (according to the degree of loss of power) not only to speak, but he is also unable to read; he may, however, be able to express himself by gestures and by writing.

3. A peculiar disturbance of the faculty of speech has been attracting attention lately. The patient is able to hear sounds or see objects; he is able to originate thought and give expression to his thought in words more or less appropriate; but words spoken convey to him no ideas, or words written are not recognized as words. There is, as named by Kussmaul, "*word-deafness*" or "*word-blindness.*" The patient may also lose the power of recognizing tunes, or of reading musical notes.

It is rare to find word-deafness or word-blindness without other affection of speech.

4. Kussmaul gives two other divisions of speech disturbance which are closely allied, *paraphasia*, and *agrammatism* or *akataphasia*. The former is "the inability to properly connect word-images and the corresponding conceptions, so that, instead of the ones corresponding to the sense, misplaced or entirely incomprehensible word-images present themselves"; the latter is "the inability to form words grammatically and to arrange them in sentences syntactically."

These peculiar defects of speech may accompany ordinary amnesic or ataxic aphasia; they rarely occur alone. It may be convenient to retain the term paraphasia, which is the more frequent of these two forms of disturbance.

5. There may be entire loss both of power to comprehend what is said and of the power of expressing thought.

The lesions which cause these different forms of disturbance of speech are situated in the vicinity of the posterior part of the third frontal convolution, the island of Reil, and the first temporal convolution, or in the white medullary substance just below these parts, or in the course of the motor fibers leading to the vocal organs. The amnesic aphasia depends on lesion of the posterior part of the third frontal convolution and the island of Reil near that spot, or the white substance

immediately below; anarthria depends upon interruption of the centrifugal fibers passing to the vocal organs; word-deafness and word-blindness occur when there is lesion of certain portions of the auditory or visual centers. The exact seat of these changes is not yet well determined. Paraphasia is found in lesion of the island of Reil, the adjacent parts of the first temporal convolution, or the commissural white fibers immediately below.

In all forms of speech disturbance the lesion is found, in the great majority of cases, on the left side, and is associated generally with more or less fully developed right hemiplegia. In left-handed persons the lesion is usually on the right; there is left hemiplegia. In a few cases this rule is not observed.

When the centrifugal fibers are destroyed near the base of the brain, in the cerebral peduncles, pons, or medulla, there may be anarthria with either right or left hemiplegia; such cases do not follow the above rule, and it may be that in some of the anomalous cases the distinction between true aphasia and ataxic aphasia or anarthria has not been sufficiently noticed.

CHAPTER III.

DISEASES OF THE MEMBRANES.

VIRCHOW, Das Haematom der Dura Mater. *Verhandl. d. phys. med. Gesell. in Würzb.*, 1857, vii, p. 134.—HUGUENIN, in Ziemssen's Cyclopædia, vol. xii.—GRIESINGER, W., Haematom der Dura Mater. *Arch. d. Heilkunde*, 3. Bd., 1. H.—KREMIANSKY, JACOB, Ueber die Pachymeningitis interna haemorrhagica bei Menschen und Hunden. *Virch. Arch.*, 42, 1868, pp. 129 and 321.—DUJARDIN BEAUMETZ, A Lecture on the Treatment of Meningitis. *New York Med. Journal*, Aug. 11, 1883, p. 141.—BERTALOT, H., Ueber Meningitis tuberculosa bei Kindern. *Jahrbuch f. Kinderheilkunde*, ix, 1876, p. 227.—SEITZ, JOHANNES, Die Meningitis tuberculosa der Erwachsenen, Berlin, 1874.—DAWSON, Y., Tubercular Meningitis in an Infant; Death; Necropsy; Remarks. London *Lancet*, April 12, 1884, p. 660.

PACHYMENINGITIS.

Inflammation of the dura mater is not very common as an independent disease. The dura mater is composed of two lamellæ which are loosely united with each other; either of these may be the seat of disease; hence arise external and internal pachymeningitis.

EXTERNAL PACHYMENINGITIS

is caused by disease of adjacent bony structures or by injuries. The symptoms are so combined with those arising from the primary affection, and the indications for treatment depend upon the nature of that affection to such a degree, that it is not necessary to delay longer on this variety of the disease.

INTERNAL HÆMORRHAGIC PACHYMENINGITIS.

There are two different views as to the origin of hæmorrhagic pachymeningitis: one proposed by Virchow and generally adopted, according to which there is first a congestion of the dura mater, which gives rise to a thin membrane on its inner surface; under this membrane occur hæmorrhages; the clot becomes organized and forms a membrane of greater or less thickness, under which more blood is poured out, and this process may be frequently repeated.

Huguenin states that he has not been able to discover any initial inflammation of the dura mater, nor any dilatation of the middle meningeal artery. He says the first stage is simply an extravasation of blood, which becomes organized, forming a false membrane, and from this arise new hæmorrhages. The difference between Virchow and Huguenin relates only to the first stage of the disease.

The brain may be much compressed, and, as the affection occurs chiefly in patients suffering from other diseases, the changes due to those diseases will be found.

ÆTIOLOGY.—The larger proportion of patients are advanced in age; many have been hard drinkers; sometimes an acute disease immediately precedes the meningeal affection; the insane, or those suffering from other cerebral disease, may be attacked. Huguenin finds a cause in cerebral atrophy, whether the result of old age, alcoholism, or other cerebral changes; he also believes the blood comes from the veins of the pia mater. Injuries have been sometimes the cause.

SYMPTOMS.—Many times the hæmatoma gives rise to no characteristic symptoms.

Headache is the most common symptom; this may be severe, or there may simply be a sense of heaviness or pressure. Sometimes the patient has a sensation as if something moved back and forth in his head, which

Griesinger refers to the sensation caused by a large cyst moving with the motion of the head. This is, however, felt under other circumstances where there can be no cyst, and therefore is not pathognomonic.

When the hæmorrhage first occurs, there will be the symptoms of meningeal hæmorrhage, more or less fully marked; the symptoms of cerebral compression, abolition or dullness of intellect; motor disturbance, irregular spasmodic motions, contractures, or loss of power, sometimes paralysis; slowness of pulse, sometimes with irregularity; the opposite pupil more dilated, the corresponding pupil contracted.

These symptoms may be very marked if the hæmorrhage is large, and the paralysis may even be bilateral, though one side is affected first, the other later.

The patient regains after some days his former condition of health, or may remain dull, drowsy, and paretic; usually the headache persists. After a longer or shorter interval, another attack occurs. With the repetition of the phenomena the diagnosis becomes more certain.

Diagnosis.—When the hæmorrhage is insignificant, a correct diagnosis may be impossible; when the hæmorrhage is large, the symptoms are those of meningeal hæmorrhage, and it is only by waiting for the subsequent course of the case to develop that a diagnosis can be made.

Prognosis.—The disease is always serious and generally fatal; recovery may occur. Griesinger reports a case in which the hæmorrhagic cyst was found nine years after the attack, the patient having recovered his health. Often, however, the disease with which the pachymeningitis is associated is one which renders recovery hopeless.

Treatment.—The treatment should have reference to preventing the hæmorrhage, to favoring absorption. Quiet, rest, cold to the head without intermission, leeches behind the ears, and a light diet, may fulfill the

first indication immediately after the acute attack. In the interval everything which would favor increase of blood-pressure should be avoided. Iodide of potassium and mercury in moderate amount may be used.

INFLAMMATION OF THE PIA MATER.

LEPTOMENINGITIS.

By *cerebral meningitis*, inflammation of the pia mater is commonly understood. Cerebral meningitis may be either simple or it may be dependent upon the deposit of tubercles. The latter is the more frequent, and the disease is much more common with children than with adults. In adults, considering all cases, simple meningitis is more common than among children; indeed, it is so rare in childhood that some authors have denied its existence.

ÆTIOLOGY.—Non-tubercular meningitis may be primary or secondary. When primary, it is often impossible to discover the cause; at other times there has been exposure to the sun, mental anxiety or overwork, great physical exertions, the influence of cold and dampness; alcoholic excess has also been mentioned as a cause.

Meningitis may be secondary to disease of the bones enveloping the brain, as the temporal bone in diseases of the ear. An obstinate or long-persisting otorrhœa should therefore never be neglected. Never neglect purulent otorrhœa under the impression that it is comparatively harmless.

Injuries to the cranial bones and fractures may give rise to caries and to meningitis; so also may syphilitic disease of the bones, and some intra-cranial affections, as tumors and abscesses.

Meningitis may be secondary to acute diseases, as pneumonia, pleurisy, cardiac lesions, eruptive fevers, erysipelas of the head, rheumatism, dysentery, peritonitis, and diphtheria; Bright's disease is occasionally complicated with meningitis.

Pathological Anatomy.—The lesion may occupy either the base of the brain or the convexity, or it may be limited to a circumscribed area only. The smaller vessels are distended with blood, the large veins are full. There is more or less serum exuded from the vessels into the meshes of the pia mater. Along the vessels are collections of pus, mixed with fibrinous exudation. If this is in comparatively small amount, it is found only in the sulci; otherwise it may be spread over the convexity. At the base of the brain it is collected also in the depressions, by preference around the pons; under the posterior lobe, near the crura cerebri; between the crura; and around the optic nerves. This exudation is not free fluid, but is contained in the meshes of the pia mater, which is swollen, or a fibrinous exudation may contain pus and serum; it may be large in amount, especially at the base, and the nerves may be surrounded by a thick, comparatively firm fibro-purulent sheath.

In very chronic cases there may be found thickened patches in the pia mater, composed of fibrous tissue among whose fibers the microscope shows pus-cells.

In cases of moderate duration the membranes, on being stripped off, may carry away portions of the gray substance.

Sometimes the ventricles are distended with serum; but this is less common than in tubercular meningitis. If they are distended, their lining membrane, the ependyma, is found thickened or covered with granulations, and it may be softened.

Symptoms.—The commencement of the disease is usually acute. Occasionally, for a few hours or days, there are headache, vomiting, and dizziness; but these prodromata are not common; there is not, as a rule, the preceding ill-health which is so often seen in tubercular meningitis.

The initial symptom is a severe headache; occasionally a chill, soon followed by vomiting; the temperature

rises quickly, even to 104°; the pulse is rapid; there is delirium; sometimes there are convulsions; toward the latter part of the first stage motor disturbances appear, either paralysis or contractions. The second stage is characterized by coma and the destruction of all the vital powers.

The *duration* of the disease is from a few hours to several days. When the disease is secondary to some other affection, the duration may be very short. When a large part of the convexity is affected, life is usually not prolonged beyond a few days; when the disease is limited to the base, the patient may live several weeks.

If the patient lives but a few days, the initial symptoms will be quickly followed by the more serious, and the final stage will almost immediately succeed the earlier symptoms. If the disease is protracted, there is a more regular progression, and it may be possible even to divide the disease into stages.

Headache is the most constant symptom, and is noticeable so long as consciousness continues, and even during delirium; or in a child too young to speak, the knitted brows, the restless motion of the head, the hands carried to the head, the moaning and crying, clearly show that there is headache. The pain is more frequently general, but sometimes is confined to the frontal or occipital regions.

The temperature is high at the beginning of the disease. When there is meningitis of the convexity, the temperature remains high, and shows less variation than when the base alone is affected.

The pulse may be at first rapid; subsequently it becomes less rapid, and fluctuates independently of the temperature; toward the close of life it may become excessively rapid again, but, if there is compression of the brain by exudations, it may fall even as low as 50.

Respiration is not especially affected in the earlier

stages, excepting that it may be very rapid in children; but toward the close of life it may be irregular, and the peculiar rhythm, which has been named Cheyne-Stokes respiration, may be observed. (See page 10.)

Vomiting may be an early symptom; at first the ingesta are vomited, later it is bilious. Sometimes the vomiting does not appear until a later period of the disease. There are very few cases in which vomiting does not occur. The appetite frequently does not suffer, and the patients take food readily, though they soon reject it again. The vomiting is often very obstinate.

Constipation almost invariably is extreme, and it is sometimes nearly impossible to obtain a motion.

Delirium is one of the earlier symptoms when the convexity is affected, beginning suddenly with delusions or hallucinations; the patients may try to rise and wander about, or they may become furious at restraint. In other cases the delirium shows itself at first only as the patient is falling asleep; it is more quiet, muttering, or talkative.

In meningitis of the base, delirium is less constant; but, when the convolutions adjacent to the base are affected, it may be present.

In the later stages the mental powers are destroyed, the patient passes into coma, from which there may possibly be a slight awakening before death; but this is less common than in tubercular meningitis.

Convulsions are almost always present in meningitis of the convexity. When the base alone is affected, there is generally at least local spasmodic action of some of the muscles supplied by the cranial nerves.

Contractions also occur; there is rigidity of the neck, the muscles of the face may be affected, or there may be strabismus. It is not common to have permanent contraction of the limbs; when they are affected, there is usually partial flexion. Attacks of opisthotonos, with tonic contraction of the extensors

of the limbs, may be present and last for a few minutes.

The abdomen is less frequently retracted than in tubercular meningitis; yet this is sometimes seen, especially when the disease is chiefly at the base of the brain.

That disturbance of the vaso-motor nerves which leads to the easy production of the *tâche cérébrale* is less common in simple meningitis than in tubercular.

The patient gradually passes into a state of coma, the delirium alternates with stupor, the convulsions diminish in frequency, the contractions are now relaxed, again reappear, until finally coma is fully established; then there is dilatation of the pupils, relaxation of the sphincters, the pulse becomes slow and irregular, but the temperature rises, the insensibility is profound, and the patient dies without a struggle. Death may, however, occur at an earlier stage during the delirium and convulsions.

The headache is explained by the affection of the membranes, by the exudation and fullness of the vessels. Leucocytes wander from the vessels into the cortical layers of the brain, the circulation is interfered with, there is irritation of the nerve-cells; hence the delirium, the convulsions. If the cortex is affected, these are more marked and more general; if the base only is affected, there is less probability of delirium, and the convulsions and contractions are more likely to affect the muscles supplied by the cranial nerves. From over-stimulation may follow exhaustion; hence the convulsions intermit. When the disease has produced destruction of the nerve-cells, which is rare, or when effusion into the ventricles has produced compression of the brain, or the exhaustion has become extreme, the last stage sets in, and there is coma. This sketch of an explanation of the symptoms is rather brief, but, by keeping in mind the physiological relations of the

parts affected, it will often be easy to explain the varying symptoms.

When meningitis occurs in the course of acute diseases or as the result of injuries, necrosis of bone, etc., the symptoms are less clearly defined. The previous primary disease obscures the beginning of the meningeal affection. (For a complete discussion of the possible variations, Ziemssen's "Cyclopædia," vol. xii, may be consulted.)

After acute meningitis certain symptoms remain; there is a chronic thickening and change which is permanent.

Local changes in the membranes and cerebral cortex may give rise to spasms of a few muscles or a limb, which may spread to all the limbs, becoming general convulsions. Spasms confined to one set of muscles, or which always begin in the same muscles, are characteristic of cortical changes. The situation of the disease can be localized in the motor centers corresponding with the affected limb.

When the motor center is destroyed, there will then be paralysis of the limb instead of spasm.

Meningitis is present in the beginning of these cases, but soon the brain-substance is affected; there is then really encephalitis. Many times syphilis is the cause of these local changes in the membranes and cortex.

DIAGNOSIS.—The diagnosis from tubercular meningitis will be considered under the latter disease. Cerebritis commences less suddenly, and the febrile symptoms are less severe; the symptoms are generally more circumscribed.

The eruptive fevers may be complicated with meningitis, but it is only occasionally that this is so; typhoid fever is more frequently thus complicated than the others. The diagnosis is rendered more difficult because head symptoms occur without meningitis. The delirium is more active when there is meningitis, the

headache is more severe, and there are more frequently convulsions.

The course of the pulse and temperature is less regular than is normal in the eruptive fevers. The character of the delirium, the sudden change in the temperature and pulse, a sudden increase of headache, the character of the convulsions—these data will aid in diagnosis, but there will often be a doubt.

Cerebral symptoms during the course of acute rheumatism are not always due to meningitis; frequently an autopsy shows no change of the membranes; occasionally, however, there is inflammation. The diagnosis is extremely difficult, and usually the patient dies without a decision being reached. Yet more difficult are those cases in which severe cerebral symptoms occur on the first outbreak of rheumatic fever before the joints are affected. These cases are rare; sometimes the cerebral symptoms subside, and the articular phenomena appear to clear up the diagnosis.

Once in a while pericarditis is accompanied with delirium and other cerebral phenomena; there is not necessarily meningitis; the primary disease is shown by physical examination, and is the more important in treatment.

The coma after epilepsy and in the course of Bright's disease may be recognized by the aid of the early history, the thermometer, and a careful examination of the urine, keeping in mind, however, that albumen may be present in the urine passed after an epileptic attack or in meningitis; the presence of marked œdema, ascites, or other serous effusions would also aid in diagnosis. If the previous history can not be learned, a diagnosis may be impossible. It should be kept in mind that meningitis may arise during the course of Bright's disease, as purulent inflammation of other serous membranes may occur.

The possibility of poisoning should be remembered.

The diagnosis from alcoholism will be considered under the latter affection.

PROGNOSIS.—The prognosis is very unfavorable; a recovery is rare, and is almost never complete. The patient, if life is not lost, is liable to have headaches, to have partial loss of memory and diminished intellectual power. "When a natural sleep takes the place of coma and the patient awakes without fever, without paralysis, with a clear mind, though acting slowly," the prognosis is favorable.

TREATMENT.—When the disease is uncomplicated, and the patient is seen at the beginning of the attack, the treatment can be vigorous, and there should be no delay. If the patient's strength justifies, general bleeding is proper. If the patient is feeble, leeches may be applied over the mastoid bones; blood may be drawn by cups from the nape of the neck. Cold to the head, uninterruptedly, and smart purgation, are to be employed. Some advise blistering the scalp; but this is of doubtful value.

In this disease, as in others, a high temperature may be met by cold baths, repeated as the temperature again rises, or a lukewarm bath, and showering the head with cold water has been advised. Of course, the patient's strength should be taken into account and all precautions taken, as in other acute diseases. Among drugs to lower temperature are salicylic acid and quinine in large doses; antipyrin, in doses of thirty or forty grains, repeated, if necessary, in an hour or two, may lower the temperature. Iodide of potassium in large doses, repeated at short intervals, is of great value. If there is a suspicion of syphilis, mercurial inunctions should be used; this has been strongly urged by some, even when there is no reason to suspect syphilis. Ergot has been recommended; but its value is doubtful.

The headache and delirium are exhausting; they should be met by chloral and bromide of potassium in large doses, from twenty to fifty grains of each (one and

a half to three grammes), repeated after half an hour or an hour if necessary. These can be given by enemata if the stomach will not retain ingesta. Small doses of chloral, under fifteen grains (one gramme), are of no value, but, even when frequently repeated, often seem to increase the delirium. Subcutaneous injections of morphia may be of great value to check convulsions and stop the obstinate vomiting.

The patient's strength should be sustained by a proper diet; if there is much vomiting, by nutrient enemata. Milk two parts, and lime-water one part, in very small amounts, beginning with a teaspoonful every half hour, is sometimes most valuable in vomiting. Sometimes it is necessary to give the stomach entire rest.

Trousseau speaks most discouragingly of all treatment, and emphatically disapproves of all active and depressing measures. He says that in the two cases of recovery which he had, Nature deserves the credit rather than his art.

TUBERCULAR MENINGITIS.

PATHOLOGICAL ANATOMY.—This disease, as is well known, consists in a more or less extensive growth of miliary tubercles in the pia mater and the inflammation excited thereby. In a large majority of cases the bacilli of tuberculosis are carried by the blood to the spot where they develop; then the tubercles form upon or within the vessels, and the normal epithelial cells undergo a change by which a large number of smaller, more round, cells are formed by subdivision, until a small nodule is formed, by the continued growth of which the vessel is closed, and then fatty degeneration of these cellular elements gives rise to the yellow, caseous centers which are seen in the larger nodules. These growths are found, not merely upon the vessels of the membranes, but also on those which enter the cortex of

the brain. The irritation of these nodules, and their interference with the normal nutrition of the membranes, give rise to inflammation; hence there are added to the miliary tubercles the injection and exudations, the fibro-purulent deposits of meningitis.

When the tubercles are many and of moderate size, it is easy to recognize them; but when small and few in number, a careful examination will be necessary.

The inflammation excited by these deposits of tubercle is not confined exclusively to the vicinity of the morbid growths, though it is generally more marked there. Pus may be found at a distance, especially along the course of the vessels.

The ventricles are frequently filled with serum, sometimes so distended as to cause compression of the brain and flattening of the convolutions.

The pathological changes are fully considered in treatises upon pathological anatomy and upon children's diseases.

In the vast majority of cases there are tubercles or cheesy deposits in other organs, whence the bacillus is carried to the brain.

When there seems to be an exception, the question arises whether the disease may not have existed undiscovered.

Ætiology.—Tubercular meningitis is much more common in early life—between two and seven years. Among adults it is the more frequent between the ages of twenty and thirty or thirty-five. Seitz found that, in every thousand deaths among adults, tubercles were present in the pia in fifteen cases. Males are more frequently affected than females.

As tubercular meningitis is generally secondary to tubercular or cheesy deposits elsewhere, all those influences which favor the development of such changes in other organs will act as predisposing causes of this disease.

Symptoms.—Many times there has been a gradual

failure of health preceding the outbreak of the symptoms strictly due to the meningeal affection. The preceding disease has undermined the general health. As the tubercles alone probably have little influence in producing the symptoms, which appear only when inflammation arises, it is not possible to determine how long the morbid growths have existed. Whether some of the earlier symptoms, as heaviness, languor, change of disposition, occasional headaches, are caused by the tubercular deposit, or by inflammation localized around these, is not absolutely certain, but probably by the latter.

Sometimes, owing to the primary disease, it is difficult to recognize the beginning of the meningeal affection; at other times the onset is as sudden as in simple meningitis. There may be a chill, but headache is more frequently the initial symptom. In children, convulsions may occur early. Vomiting occurs sooner or later in almost all cases, but may cease after a few days; there is constipation. There may be general hyperæsthesia of the skin, also an over-sensitiveness to noise and to light. The patient will perhaps bury his face in the pillow to shield his eyes from the faintest ray of light.

The temperature is very irregular. Sometimes there is high fever at the beginning, but less regularly so than in simple meningitis. Not unfrequently the temperature is normal for several days; then its course is irregular. A very high temperature is not often found until toward the close of life. The pulse also varies, but does not vary with the changes of temperature.

Convulsions may occur in the later stages of the disease, and partial convulsions—as of the muscles of the face, of the eyes, or of mastication—are seen in many cases, producing contortions of the face, strabismus, etc. The head may be drawn back, or at least the neck be stiff; the abdomen may be retracted. The pupils may react imperfectly or be unequal. Paralysis of one

or more limbs, as well as of certain muscles supplied by cranial nerves, occur after a few days. The tâche cérébrale can very generally be produced.

The mental symptoms are sometimes delayed until a late stage, sometimes appear early; irritability, moodishness, fretfulness, and lack of mental vigor, may be among the earlier phenomena. Delirium is common, especially on falling asleep; it is not generally very violent. Toward the close of the disease all mental function is abolished, there is complete coma; then the more active symptoms—as spasms, contractions, cries, restlessness—cease. The temperature generally rises quickly, once in a while becomes lower. The pulse may be very slow. Respiration becomes irregular, the Cheyne-Stokes respiration may appear, and soon the patient dies. A short time before death intelligence may return; there seems to be a very great improvement, which may well deceive the friends, but the physician should be on his guard.

Optic neuritis is quite common in tubercular meningitis, unless the disease is confined to the convexity. The neuritis is not often intense; is sometimes so slight as to not be easily recognized; it is rarely accompanied with hæmorrhages. Both eyes are affected.

It has been the custom to divide the progress of the disease into three stages: 1. Irritation; 2. Pressure; 3. Paralysis or coma.

Occasionally these stages are well defined, but more frequently they run into one another, and the symptoms occur with so little regard to orderly succession, that it is not possible to define the various stages. Rather than to describe the disease thus, I have preferred to give a general description, which, it seems to me, more nearly corresponds with what is seen in common practice.

The following explanation of the symptoms, condensed from Huguenin, will assist to a better understanding of the disease: The earlier prodromal symp-

toms, if there are any, may depend upon disturbance of the nutrition of the brain, caused by the tubercles, possibly by irregular and variable irritation of the nerve-cells. Inflammation of the pia mater is excited by the tubercles; the cortex is affected secondarily; occasionally slight hæmorrhages occur. The mental symptoms are those of excitement during the rise of the inflammation, the character of the mental disturbance varying with the locality of the inflammation, as different nerve-centers are affected. The hydrocephalic effusion will explain the symptoms of compression which are seen toward the close of the disease. The convulsions may be referred to irritation of nerve-centers in the cortex or to an irritation of the medulla oblongata, the latter especially if the convulsions are general. Rigidity of the muscles of the neck, and the weakness and peculiarities of gait, are not easily explained, but may be due, not simply to the cerebral lesion, but rather to spinal complications. When paralyses of individual limbs or of one side occur early, there is usually a local cause, easily recognized at the autopsy (as hæmorrhage or local softening). When these occur late, the cause may escape detection. Local paralysis of individual cranial nerves may be explained by the effusion around the roots of these nerves, the neuritis or other degenerative changes set up in them.

When the cortex is chiefly or exclusively affected, mental symptoms will predominate, with local spasms and paralyses; when the base is chiefly affected, mental symptoms may be almost entirely absent; general weakness, general spasms, paralyses of cranial nerves, and at length hydrocephalus, will be the principal symptoms.

Diagnosis.—The first question is naturally whether there is meningitis; the second, whether, if present, it is simple or tubercular. The following enumeration of the more important symptoms is given by Seitz: "Headache; vomiting; constipation; absence of roseola; stu-

pefaction; confusion; delirium; obstinacy; carphologia; stiffness of the neck, back, or muscles generally; general hyperæsthesia; retracted abdomen; paralysis of the pupils, eyelids, eyes, of the face, of the limbs; trembling; twitching; convulsions; old phthisical affection of the lungs, and other chronic inflammatory, suppurating, and caseous processes; duration of the disease from two to four weeks with fatal termination."

The diagnosis from simple meningitis is not by any means always easy. The diagnosis will be facilitated by bearing in mind, that in children tubercular meningitis is much the more common, while in adults simple meningitis is the more prevalent.

The condition of health preceding the attack is an important element in diagnosis. The tubercular form, as a rule, begins less acutely, the duration is longer, and the course more irregular.

The temperature is less steady, may be moderately elevated only for a few days, may be below normal, is rarely very high, excepting toward the close of life. In simple meningitis the temperature is higher at the commencement, and is more regular. The pulse is frequently slow in tubercular meningitis, and does not necessarily follow the variations of the temperature.

Tubercular meningitis is the more often accompanied with paralyses of the cranial nerves, and even of the extremities.

Convulsions, retracted abdomen, tâches cérébrales, contractions, vomiting, constipation, and the variations in respiration, give but little aid in forming a diagnosis between the two forms of meningitis.

It is very important to distinguish the pseudo-hydrocephalic state found in infants, especially during diarrhœa, dysentery, and other exhausting diseases. This will be considered under Cerebral Anæmia.

It will sometimes be necessary to guard against being deceived by the possibility of reflex irritations, as teething, ingestion of improper food, and worms, which

may give rise to convulsions and other doubtful symptoms.

Wilks makes the following statement: "If you meet with an obscure case of recent disease, to which you can only apply the term cerebral, without being able to declare the existence of any special lesion, it will generally turn out to be a case of meningitis." This is rather too sweeping, but contains considerable truth.

PROGNOSIS.—Trousseau mentions two cases of partial recovery in children, one with paralysis. The child died five months later from dysentery, and the traces of the old disease were found at the autopsy. In adults it is very doubtful whether recovery is possible, and even in children the recoveries are so few that practically there is no reasonable hope of a favorable termination.

TREATMENT.—It is almost hopeless to try to do anything, but we can refrain from being too meddlesome. The active treatment, which might be of advantage in simple meningitis, would not be called for in the tubercular. The cases reported as recovering were treated, some by ergot in large doses, and some by iodide of potassium in large doses frequently repeated.

As in many cases a diagnosis is doubtful, and as simple meningitis is amenable to treatment, it will often be most wise, excepting depleting measures, to treat a case of suspected tubercular meningitis as if it were simple meningitis.

CHAPTER IV.

CHANGE IN BLOOD-SUPPLY.

JONES, C. H., Studies on Functional Nervous Disorders. London, 1870, p. 64, 86.—FOTHERGILL, J. M., Cerebral Anæmia. *West Riding Hospital Reports*, vol. iv, 1874, p. 94.—MOTTA, E. A., Ueber Hirnanämie im Algemeinen und insbesondere über Blutleere des Gehirns und über dessen consecutive Erweichung. *Deutsche Klinik*, 43, 1874.—MITCHELL, WEIR, Fat and Blood. Philadelphia, 1877.—HEWITT, GRAILY, Chronic Starvation. *London Lancet*, Jan. 11, 1879, p. 38.—BALL, BENJ., On Certain Cases of Functional Ischæmia of the Brain. *Brit. Med. Jour.*, Oct. 30, 1880, p. 693.—JACCOUD, S., Traité de pathologie interne, 1870, t. i, p. 106.—MOXON, WALTER, Influence of the circulation upon the nervous system. *Brit. Med. Jour.*, 1881, I.

CEREBRAL ANÆMIA.

Among the conditions of the brain most difficult for diagnosis are those in which there is irregular blood-supply. There are several reasons for this: 1. An overworked brain is an exhausted brain; not only are the proper cerebral nervous elements exhausted, producing irregular action, but the vaso-motor nervous supply also may become irregular from exhaustion of the vaso-motor nerves. 2. Another cause for mistake is that a badly nourished brain is irritable. Hence many of the symptoms of exhaustion and of malnutrition resemble those found in congestion or stimulation from excess of blood. 3. Poor blood, that which is not suitable for nourishment, may be sent in large amount to the brain, and there will still be practically anæmia; the nervous structures may have lost their power of absorbing the

nutrient materials from the blood, and the same results follow.

Under anæmia it is intended to comprise all those conditions wherein there is insufficient nutrition of the brain, owing to defect in quantity or quality of the blood.

ÆTIOLOGY.—When there is general anæmia or chlorosis, the brain usually suffers more or less from an insufficient supply of healthy blood. Those conditions, then, are causes of cerebral anæmia which give rise to general anæmia, as hæmorrhages, deficient supply of food, bad hygienic surroundings, exhausting discharges, whatever causes a poor appetite or poor digestion, etc.

A hindrance to the flow of blood to the brain, as pressure on the arteries, obstruction of their caliber by atheroma, arteritis, spasm, embolism, thrombosis, may give rise to a deficiency of blood-supply in the brain as a whole, or their influence may be local, and limited to a small area. The same result follows a weakening of the heart's action, so that the blood is not sent with sufficient force to fill the cerebral vessels.

Deficient oxidation, absorption of poisons, overheating by sun or artificial heat, hyperpyrexia in fevers, etc., cause a defect in the quality of the blood, rendering it unfit for the healthy nutrition of the brain.

Though not immediately acting upon the supply of blood nor its quality, it is necessary to consider the conditions under which the patient has lived. Not only do anxiety and worry, but overwork, mentally or bodily, and loss of sleep, by exhausting the nervous system, have a tendency to give rise to an irritability of the brain, which causes it to show the effects of deficient blood-supply more quickly.

There are very few cases where diminished blood-supply alone is the cause of the symptoms. It is only in the acute cases following sudden hæmorrhage that this is true, and even in these there is subsequently deficient quality of blood. Generally changes in quality

concur in causing the symptoms. Some of the diseases in which the symptoms of cerebral anæmia may be found, caused by changes in the nutrition of the blood, are dysentery, diarrhœa, gastric catarrh, phthisis, suppurative diseases, syphilis, typhoid and other fevers, and malarial poisoning.

Pathological Anatomy.—There is simply paleness of both white and gray substance, comparative emptiness of the blood-vessels, and, excepting there be compression from some cause, moisture of the cut surface. The lymph-spaces are filled with serum or lymph to compensate for the diminished fullness of the blood-vessels. In chronic cases there is undoubtedly change of nutrition; but our means of examination do not enable us to recognize such change.

In local anæmia from obstruction of blood-vessels, changes occur which will be more properly considered elsewhere.

Symptoms.—The symptoms vary according as the anæmia is acute or has come on gradually. If the patient has been exhausted by previous disease, the symptoms will be less violent than if he has been in vigorous health. Whether the brain has been overworked and excited must also be taken into consideration.

When a rapid hæmorrhage causes cerebral anæmia, the attack is acute, there is a loss of mental power, vertigo, dimness of sight and of hearing, tinnitus, sensation becomes blunted, the pupils are contracted, then dilated, the skin is cool, consciousness is lost, and convulsions occur. Respiration is accelerated, then slow; pulse small, frequent, and of diminished tension. This combination of symptoms may be seen not only in severe hæmorrhages, as in surgical and obstetrical practice, but less completely developed where a patient greatly exhausted rises from the bed too suddenly; also in ordinary fainting from whatever cause.

The description of more chronic forms of cerebral anæmia is rendered difficult by the fact that very few

cases are uncomplicated. Nervous exhaustion, from anxiety, worry, fatigue, or overwork of brain, serves to intensify or change the symptoms; also the symptoms of anæmia and hyperæmia of the brain are very similar —so similar that it is oftentimes impossible to decide which condition is present without regard to preceding circumstances. Again, an irritable brain, anæmic as a whole, may be locally congested, or may receive temporarily an increased supply of blood, though a supply below that appropriate for health, and, owing to its abnormal irritability, be excited as if hyperæmic.

In chronic anæmia, thought is an effort, sustained mental exertion is impossible, memory is uncertain, the patient is drowsy; occasionally after rest there may be flashes of brilliancy, but they are brief. If effort is absolutely necessary, the patient may have learned that a very small amount of wine or spirit is a temporary aid. Headache is one of the most annoying symptoms, and is very persistent, and may incapacitate for the ordinary duties of life. After lying down long and being quiet, the headache may be relieved, but recurs on attempting to go about. Perhaps, in part owing to the headache, in part owing to the malnutrition of the nerve-centers, there is likely to be a change of disposition; irritability of temper, fretfulness, and peevishness are seen. Sometimes there is dizziness; more frequently there are noises in the head, tinnitus aurium, also various disturbances of vision, muscæ volitantes, dimness, even amaurosis, though the latter is rare. Respiration may not be much changed, but there may be a feeling of discomfort, as if not air enough were inspired, and so there is sighing. The pulse usually varies only slightly from the normal, unless there is present some disease to account for an increased rapidity, as phthisis. The same is true of the temperature; it is usually about normal or a little below, but may be elevated if any febrile affection exists. General weakness is almost always seen, but rarely complete paraly-

sis. Convulsions are not seen in cases of chronic anæmia.

When the anæmia is very great, there may occur delirium; if the anæmia has come on with moderate rapidity in a rather vigorous person, without previous disease, the delirium is usually active, the patient may even be maniacal, and may have hallucinations, hearing voices, and holding conversations with imaginary persons; he may not recognize his friends, or may desire to escape. This feature of cerebral anæmia has so much the appearance of excitement from hyperæmia that many refer it to such a condition, supposing that there is local congestion; but the active delirium is seen only when the affection has been developed rather rapidly, and is probably owing to the state of irritable weakness, to which reference has already been made. When the anæmia occurs during the course of an exhausting disease, either as a result of the disease or of insufficient feeding, the delirium is more likely to have a quiet character. Finally, the mental powers may be entirely lost.

Diagnosis.—In forming a diagnosis of this affection it is necessary to take into consideration the previous circumstances of the patient. The diagnosis from hyperæmia may be very difficult, and, as the treatment would be quite different, it is important to be as nearly correct as possible. If there is a history of long-continued privation, with worry and anxiety, or of hard work, physical or mental, and loss of appetite, or exhausting discharges, it is probable that the condition is anæmic, although there may be much excitement. It is sometimes more difficult to decide, where there is active delirium, whether it is insanity or anæmia. Here also the previous history will be of assistance; but, as anæmia may lead to change of structure in the nerve-cells, it may pass over into insanity, and without mania it may pass into melancholia. In insanity induced by cerebral anæmia there is not a long-continued period of

excitement; the condition is rather one of depression, with occasional attacks of irritability.

To recognize that delirium in febrile diseases is owing to cerebral anæmia is all-important. Especially in children with gastro-intestinal affections the symptoms resemble those of serious organic brain disease; the previous history must not be overlooked; it would be disastrous to treat a child with anæmia for meningitis. During typhoid fever in adults there may be a similar mistake. It would seem that care in watching the patient—not only the fever, but also the feeding of the patient—might prevent such an error. Excessively high temperatures may give rise to symptoms similar to those of anæmia; a careful use of the thermometer will guard against this mistake.

PROGNOSIS.—If there is no serious complication, as cardiac or Bright's disease, the prognosis is favorable, provided sources of exhaustion can be removed. The prognosis in the case of other diseases may be favorable for the anæmia, though unfavorable for the primary disease.

TREATMENT.—If the case is one of acute anæmia, or of extreme weakness after protracted disease or exhausting discharges, it will be very important to keep the head low; perhaps the foot of the bed should be raised; the body should be kept warm, by artificial means if necessary; stimulants may be necessary; food in a form easily digested, in small amounts, frequently repeated.

The chronic form needs methodical rest and feeding. As many of the symptoms are due to exhaustion of the nervous system, the effort should be made to withdraw the patient from all such influences as tend to exhaust him. In many cases the course of treatment recommended by Weir Mitchell in "Fat and Blood" will give excellent results. In every case the best tonic is food. The food must be easily assimilated, not in too great quantities, and should be taken at short

intervals. Milk is one of the best to begin with; not only is it easily digested and contains all the constituents of the body, but is largely composed of water. Fothergill's remarks about water in anæmia are deserving of attention.* Among drugs, arsenic, iron, and quinine are valuable.

Where there is restlessness, sleeplessness, delirium, it may be necessary to give chloral and bromide of potassium, or paraldehyde. Small doses of these are worse than useless; even if frequently repeated, they are not efficacious; less than twenty grains of each is not sure of giving rest; in many cases it may be necessary to give thirty or even forty grains at one dose. Yet chloral should be given with caution, and not administered every time a patient does not sleep; it is often given injudiciously. Spirit will sometimes aid sleep. Opium is a valuable agent, and may often be given to advantage in small doses—a tenth or twelfth of a grain—the object being rather to obtain its stimulating effect. Sleep may sometimes be produced by a grain or two or quinine at bedtime, or by a dose of phosphoric acid, or by a light lunch just before retiring. A cup of beef-tea during the night may overcome the habit of lying awake.

CEREBRAL HYPERÆMIA.

When the arteries are distended, or there is an increased flow of blood through them, there is active hyperæmia; when the veins are over-distended, it is passive. The latter condition may, in reality, be one of anæmia, so far as concerns the state of the circulation in the brain.

Some authors deny the existence of cerebral congestion.

CAUSES.—Probably a predisposition to cerebral hyperæmia is constitutional with some persons, just as

* "Handbook of Treatment," pp. 51, 52.

some blush more easily than others; the predisposition may be acquired. Whatever has a tendency to cause a fullness of the cerebral arteries, and keep the blood flowing rapidly through the brain, may give rise finally to a predisposition to cerebral hyperæmia. Excessive and often-repeated emotional disturbances, excessive and protracted brain-work, are among these agents. But it must be kept in mind that these also produce exhaustion, and so irritability, which may lead the brain to respond unhealthily to the normal amount of blood, or to be excessively excited by less than the normal amount.

Among other influences may be mentioned a low temperature; thus, most cases are said to occur in winter. A very high temperature is also said to cause congestion, and especially if the sun shines directly upon the head; but the symptoms following such exposure are rather due to elevation of temperature and change in the quality of the blood.

Increased activity of the heart is also said to be a cause of cerebral congestion; hence violent exertions may give rise to it.

Certain drugs may cause cerebral hyperæmia, as nitrite of amyl. Opium and belladonna have been thought to do so; but this is not certain. Alcohol may act as a cause, but only acutely; chronic alcoholism acts rather by producing changes in the quality of the blood, and so changes in the nerve-structures.

Malarial poison may excite congestion of the brain; indeed, probably every attack of chills and fever is attended with cerebral hyperæmia, and this may be one cause of danger in severe malarial diseases.

Passive congestion may be caused by any interference with the return of the blood from the brain.

PATHOLOGY.—In acute cases very little change may be expected; much or most of the blood drains off post-mortem. Yet even then, and with rather more frequency in cases of longer standing, the smaller vessels

in the cerebral substance show with unusual distinctness upon section. The surface of the section is thickly sprinkled with bloody points, the gray substance is darker, and the white substance may have a decided pinkish color, from the fullness of the minutest vessels.

In chronic cases the constant dilatation of the vessels may lead to changes around them; the perivascular sheaths may contain granules of blood pigment. There are seen, also, cavities in the brain containing the transverse section of a vessel. These are thought by some to be caused by dilated perivascular sheaths, by others to be dependent upon dilatation of the vessels. The latter may act as one agent in their production, another may be shrinking of the cerebral substance, a slight atrophy which causes a dilatation of the perivascular sheaths.

Constant and repeated hyperæmia must interfere with the nutrition of the nervous structures; the high blood-pressure is unfavorable for the interchange of elements. The changes thus resulting occur slowly, but finally may be very serious and may lead to insanity. These changes will also explain why it often requires so long a time for recovery from symptoms which seem insignificant. In this connection it must be remembered that not only do the proper structures of the brain suffer, but also the walls of the blood-vessels and their vaso-motor nerves undergo changes—at least functional changes, and probably slight organic changes.

Symptoms.—Many of the symptoms of cerebral hyperæmia are the same as are found in cerebral anæmia, and many which are usually ascribed to hyperæmia are quite as dependent upon exhaustion from overwork, anxiety, etc. It is impossible to entirely separate the two classes of symptoms in giving an account of the affection.

If not severe, there may be a sense of heaviness, or pain in the head, with tinnitus (though this is more common in anæmia), dizziness, sleeplessness, more or

less agitation, perhaps at times a tingling sensation in the fingers or feet, as if they were "asleep." These symptoms at first recur only occasionally, but may become more permanent.

There may be more serious symptoms: the temperament may be changed and the patient be fretful and irritable, the mental power diminished, and there may be absolute inability to apply the mind in certain directions. When an intense application of the mind to one class of questions has brought on the affection, there is inability to apply the mind in that direction. A teacher can not teach; the effort to do so may cause such confusion that he will have no command over his speech; or a lawyer may be unable to try a case before a jury, the attempt to do so being preceded by sleepless nights, and accompanied by such distress, or even semi-delirium, as to make it impossible. It would seem as though in these cases there is a local irritability of the nerve-centers relating to such pursuits, which, when an attempt is made to use these centers, gives rise to an increased flow of blood, not only to them, but to other parts of the brain also. It is interesting to note that the use of other centers has not this effect: thus, the lawyer who can not try a case in court may be able to attend to other business (though it is not by any means safe to allow it); the merchant may not be able to keep the run of his goods, but he can attend to his garden and care for his country residence without distress.

Memory is affected: there is confusion, dullness, wrong words are used in talking; there may be delirium, or the excitement may run into mania. At times there may be weakness of the limbs, twitching of muscles, especially of the face; paralyses almost never occur; convulsions belong rather to anæmia or epilepsy; and also disturbances of sight and hearing are more frequent in anæmia; vomiting is rare. Respiration is little affected; the pulse is usually full and resistant, perhaps rapid, possibly moderate. The face is usually flushed

or ruddy, is rarely pale, and the conjunctivæ may be injected.

Many authors describe a form of this affection attended with convulsions, which, however, Trousseau refers to epilepsy, and this explanation is now generally accepted.

There is a condition which I have met a few times which seems to be dependent on congestion rather than anæmia. I have only seen it in women run down nervously; so nervous exhaustion is one element in causing it. The patient, after some emotion or shock, only slightly more severe than usual, or after some exertion, as ascending stairs, feels weak, is unable to stand or sit, has distress in the head, then loses all power of motion, and speech; lies as if in a faint, but the face is flushed; the heart beats vigorously, perhaps not more so than normal; respiration is little if at all affected. If the attack is not severe, the patient may lie utterly helpless, unable even to move an eyelid, yet know all that is said and done. In severer cases, consciousness is lost. I have known such an attack to last two or three hours. There is no spasmodic action. Recovery is gradual, then respiration may be sighing; subsequently there is great distress in the head and confusion of thought for several hours.

These attacks may occur during the night, either in consequence of a dream or from the previous day's exhaustion and the recumbent position. One gentleman told me that, after learning that his wife had these, the peculiar respiration, in her case noisy, aroused him. These attacks differ from epilepsy in that a definite cause can be so frequently traced that it is reasonable to think such a cause always exists; they recur as irregularly as the causes which give rise to them.

Diagnosis.—It is not always easy to distinguish congestion from anæmia or from simple nervous exhaustion. Sometimes it is necessary to suspend judgment for a while to watch the course of the symptoms.

A superficial examination of the patient will be as likely to lead to an error in diagnosis as to give correct results.

Some physicians recognize hyperæmia in almost every obscure morbid functional state of the brain; others disbelieve in it entirely.

The previous history of the patient will assist materially in diagnosis. Has the patient been well fed, or poorly? been happily situated, or miserably? had prosperity or poverty? have there been exhausting drains upon the system, frequent small hæmorrhages, or severer hæmorrhages? Is the patient full-blooded or generally anæmic? Is the face ruddy, flushed, or pale? Did the attack come on as the result of excitement, or during the strain of some intense mental effort, or during a violent physical exertion? All these inquiries will assist in forming a correct diagnosis when the symptoms are uncertain.

A diagnosis depending upon the symptoms may be made in many cases with some degree of probability. In anæmia the symptoms are those of excitement only in rare cases, and then the excitement is not of long duration; as a rule, in the more chronic cases there is depression. In hyperæmia, excitement and exaltation predominate, and there is apparent depression only when the brain is overpowered by the severity of the attack, or the congestion is passive. The headache is more acute in anæmia; more of a feeling of painful fullness in hyperæmia. The pulse is fuller and more bounding in hyperæmia. These peculiarities, with attention to the whole group of symptoms as described above under both these affections, will in most cases lead to a correct diagnosis. It would be easy to pick out typical cases of both these conditions from actual practice and describe them, but such cases are not easily mistaken, and, unfortunately, form only a small proportion of the cases we see. As in many other diseases, a careful examination and consideration of

all the circumstances are necessary to a correct diagnosis.

Other affections which must be separated from cerebral congestion are cerebral hæmorrhage, cerebral embolism, and thrombosis. These will be better considered under those affections. Vertigo from disease of the ear and from derangement of the digestive organs also needs to be distinguished.

The most important affection, next to anæmia, to correctly recognize is epilepsy. Some forms of epilepsy are so obscure, especially in the commencement of that affection, that it is no uncommon circumstance to have it overlooked, and the patient, his friends, and perhaps the physician, consider the attack is a mere passing rush of blood to the head. This will be more intelligently considered under epilepsy.

A reasonable care in the examination of the urine will suffice to distinguish Bright's disease, which may give rise to symptoms closely resembling those caused by disturbance of the cerebral circulation. The urine should be examined more than once if the diagnosis is doubtful.

Prognosis.—The danger from an attack of cerebral congestion depends upon the severity of the attack and its suddenness. The brain may be completely overpowered by the sudden influx of blood, consciousness may be lost, and, if the medulla is also affected, life may be extinguished at once or very soon. Walter Moxon, however, finds no satisfactory post-mortem evidence that acute congestion is ever a cause of death.

But generally the patients do not die at once, and in chronic cases there seems to be no immediate danger to life. Recovery, however, is tedious. Almost always there have been various circumstances in the patient's history acting as predisposing causes, and this is one reason for the slow recovery. Where there is no complication, a recovery may be expected in time, provided the patient will submit to proper treatment.

Among other complications, the most unfavorable is organic change in the nervous elements of the brain. This may give rise to insanity.

TREATMENT.—Congestion of the brain may be accompanied with so severe symptoms, with such immediate danger to life, with unnatural fullness of the arteries, with such strong action of the heart, that general bleeding is indicated. These cases are, however, rare, and such bleeding would not be advisable in any except a robust and plethoric patient. Local bleeding by cups or leeches would be beneficial in a larger number of cases. In the severer forms, when it is not desirable to withdraw blood, means may be taken to increase the flow of blood to distant parts of the body, as by hot foot-baths or mustard foot-baths ; or to diminish the amount of blood by acting on the bowels, saline cathartics, croton-oil, etc. ; or by promoting copious sweating by hot-air baths, steam baths, hot-water baths, keeping ice on the head. Jaborandi acts especially on the skin ; but, as it frequently gives rise to violent vomiting, it would be hardly safe, lest the straining in vomiting should increase the congestion.

Cold applications to the head—ice, a rubber tube coiled up, with cold water running through it, evaporating lotions—may give relief ; but to be of much benefit, the application must be continuous.

When there is violent delirium, mania, as one of the most marked symptoms, it will be frequently found that there have been circumstances tending to render the brain excitable. The measures already mentioned may be employed : sometimes a wet cup to the back of the neck taking a few ounces of blood aids in giving relief ; sometimes a dry cup gives equal relief. Bromide of potassium and chloral, in doses sufficient to produce sleep, are especially indicated ; from thirty to sixty grains of each. Small doses of chloral repeated hourly have no effect, but rather aggravate the symptoms, and the combination of the two drugs is more

efficacious than either alone. Hyoscyamus will often procure sleep and quiet if given in large doses. In cases of mania from cerebral hyperæmia, opium should not be given, but during delirium from anæmia it may be beneficial.

Where the hyperæmia is chronic and has been produced by excessive mental application, or by constant emotional excitement, the first indication is to remove the cause. The patient should drop his studies, his business, whatever has intensely occupied him, and withdraw from all associations which give rise to emotional disturbance. A quiet, regular, systematic life, with easily digested, mild food, is the most favorable. Bromide of potassium, without chloral, fifteen or twenty grains three times a day. Bromide of sodium, lithium, calcium, or ammonium, have been used instead, and are more agreeable to some patients. Ergot, either as fluid extract, half a drachm to a drachm, or ergotin, three to five grains, three times a day. Ice to the back of the neck will sometimes relieve the discomfort in the head better than when applied to the head directly.

Most cases of chronic cerebral hyperæmia are also complicated with nervous exhaustion, and it may be desirable to give tonics to counteract the exhaustion. The vaso-motor nervous system is at fault, and requires not only a temporary stimulant to cause the arteries to contract, but it needs also to be permanently strengthened. A systematic course of hydrotherapy may be of value in this direction; also the ordinary tonics.

The chief object of these remedies is to restore the normal action of the arteries and the vaso-motor nerves. To do this, iron, strychnia, arsenic, quinine, zinc, nutritious but unstimulating food, are the most valuable agents. Spirituous liquors are to be avoided; tea and coffee taken only in moderation, if at all; tobacco should be forbidden. It is scarcely necessary to mention that the digestive and other functions should be kept in a normal condition.

Those who have had attacks, or who seem liable to attacks of cerebral congestion, should avoid public gatherings where the air is likely to become impure and heated, and where there is more or less excitement, as theatres, concerts, balls, etc. They should be quiet in all their movements, avoiding exertions which would cause an increase of blood-pressure in the brain, as running, lifting weights, straining at stool, and venereal indulgence. They should sleep in cool, well-ventilated rooms; better on a hair mattress, with head elevated. They should take exercise in the open air, but avoid being chilled in cold weather. All intellectual efforts which produce the least discomfort in the head should be avoided. As one can not stay at home surrounded by familiar objects without the mind running more or less in its old ruts, and being recalled by old associations into old trains of thought, it is especially important to break up all such associations in cases of chronic hyperæmia, and, where the patient's health permits, traveling or a sojourn away from home is very desirable. Often it is the method whereby quickest relief can be obtained.

CHAPTER V.

HÆMORRHAGE.

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MENINGEAL HÆMORRHAGE.

When a vessel of the dura mater ruptures, the blood may escape either between the dura and the skull, or into the dura itself, between the layers of its fibers, or between it and layers of false membrane. The former is more frequently the result of an injury; the latter is found in pachymeningitis hæmorrhagica. The blood may also escape into the arachnoid space. This is not common, and, when it occurs, it can not be distinguished from hæmorrhage following the rupture of one of the vessels of the pia mater.

ÆTIOLOGY.—The causes of hæmorrhage are blows and falls, bursting of aneurisms, other disease of blood-vessels, or thrombosis of sinuses. Rupture of aneurisms is the more common cause; these may be either miliary or of considerable size. The effused blood may be but little in amount, or may cover a large extent, even dipping down into the sulci and fissures, and extending to both sides or penetrating into the ventricles.

PATHOLOGICAL ANATOMY.—The blood is usually poured out in such quantity that death follows before any change can take place in the clot; occasionally death is delayed long enough to show that absorption has commenced, and in a very few instances the blood has been in such small quantity that it has been absorbed, and, subsequently, the remains found in pigmented spots on the membranes. In these cases there is a reasonable doubt whether the disease is not pachymeningitis rather than meningeal hæmorrhage.

SYMPTOMS.—The symptoms will vary according to the locality and extent of the hæmorrhage. If there has been previous disease of blood-vessels, especially an aneurism of considerable size, there will have been corresponding symptoms preceding the attack. Many times these will be merely headache, lassitude, heaviness, vertigo.

If the blood escapes suddenly in one gush, there will be a sudden and immediate loss of consciousness, and paralysis with slow pulse and stertorous respiration, and the patient may soon die. If the blood escapes less rapidly, the loss of power will come on gradually, and the patient may be able to walk across the room, or even farther, and call for assistance before sinking to the floor and losing consciousness.

According to the locality of the hæmorrhage, to the irritation excited by it, and perhaps other illy defined circumstances, there may be convulsions attending the attack. These may affect one or more limbs, may be universal, or may only affect certain cranial nerves.

Occasionally the patient recovers from the first attack of unconsciousness, and seems to be gaining until another, and, perhaps, several successive attacks terminate fatally.

The symptoms are the result of a combination of local irritation, pressure, and anæmia of the brain. If the vessel is in the pia mater, the blood may be poured out with such violence and in such a direction as to tear up a portion of the cortex immediately adjacent.

DIAGNOSIS. — The peculiarities wherein meningeal hæmorrhage differs from intra-cerebral hæmorrhage are, the more frequent occurrence of convulsions and contraction in about a quarter of the cases; the paralysis is less likely to be local, all the limbs suffer alike, and there is gradually diminishing power until total paralysis is reached, though sometimes there is hemiplegia. The temperature follows very much the same course as in intra-cerebral hæmorrhage.

PROGNOSIS.—Excepting in very rare instances, death is the invariable result.

TREATMENT.—The treatment is the same as for intra-cerebral hæmorrhage.

CEREBRAL HÆMORRHAGE.

Cerebral hæmorrhage and apoplexy are not synonymous terms. The latter is applied to all attacks wherein there is sudden loss of consciousness without convulsions, coma continuing for a longer or shorter time, ending in death, or partial or entire recovery. This may be the result of other causes than hæmorrhage, as congestion, or plugging of an artery by an embolus.

ÆTIOLOGY.—The amount of blood poured out depends upon the size of the blood-vessel; also upon its situation. The white substance is much more easily torn up than the gray substance, and, when the blood bursts into one of the ventricles, there is much less

resistance to the bleeding than when it is confined by the cerebral substance. The form of the clot and of the cavity containing it depends upon the direction of the nerve-fibers among which the blood is poured out.

The size of the clot may vary from that of a pin's head or of a pea to a clot occupying nearly the whole of a hemisphere. The largest clots are those where the hæmorrhage begins in the corpus striatum or the optic thalamus, and extends into the white substance of the centrum ovale. The relations which the size of the clots and the frequency of hæmorrhage bear to the cerebral circulation are considered by Duret. The anterior part of the caudate nucleus is supplied by nutrient branches arising from the anterior cerebral and anterior communicating arteries. These are all small; hæmorrhages here are rare, and are usually small. The lenticular nucleus, and the anterior portion of the optic thalamus, are supplied by arteries from the middle cerebral, which is a large artery and nearly in line with the carotid; the nutrient branches are comparatively large, and hæmorrhages more frequently occur from these and are more likely to be copious. There are no large arteries running through the centrum ovale; hence hæmorrhages here are rare and of small extent. The posterior lobe is supplied with larger vessels from the posterior cerebral artery; hence hæmorrhages of considerable size may be found there. These are some of the more important and interesting conclusions at which Duret arrives.

To Charcot and Bouchard belongs the credit of referring cerebral hæmorrhage to a periarteritis of the smaller arterioles, the external coats being first affected. There is an increase of nuclei of the lymphatic sheath; the adventitia is also affected. The nuclei may be so crowded together that nothing else can be seen. Sometimes, with less increase of nuclei, the adventitia may be thickened and may have longitudinal striæ. Next, the muscular elements disappear without fatty degen-

eration, the muscular markings on the artery becoming less distinct and fewer, until finally they disappear in limited regions; then the artery may dilate, bulge out locally, and a minute aneurism is formed; sometimes only a fusiform swelling of the artery is seen. These miliary aneurisms may be very numerous throughout the brain, or a few in a limited region. They sometimes seem to be quite large from the staining of the tissue immediately around them.

They are found with most frequency in the corpus striatum and the optic thalamus, then in the pons Varolii, and the gray substance of the convolutions. These minute aneurisms bear an important relation to cerebral hæmorhage, as in much the larger number of cases their rupture is the cause of the hæmorrhage.

Atheromatous degeneration of the larger arteries may favor a rupture by impairing the elasticity of the vessels. Increased strength in the action of the heart will also act as a cause of hæmorrhage by sending the blood with increased force into the diseased vessels; hence many cases occur while the patient is making violent effort; but also many occur during sleep.

While it is true that disease of the kidneys is found in many instances with cerebral hæmorrhage, it is not as yet determined how frequent this association is.

Some writers lay stress upon a vitiated state of the blood as a cause. This might affect the nutrition of the arteries, and so favor the disease.

The condition of the brain is mentioned as another element in the production of hæmorrhage. It is scarcely possible for periarteritis with many miliary aneurisms to exist without interfering with the nutrition of the brain, and doubtless many of the so-called premonitory symptoms are thus produced; but how much such change favors the occurrence of hæmorrhage we have no means of estimating. Whether such changes are of the nature of softening, or of an increase of the interstitial tissue, is not yet known. Minute hæmorrhages are sometimes

found around tumors in the brain; generally they are very small, and cause no special symptoms.

It is well to mention that cerebral hæmorrhage occurs the more frequently after the age of forty years; but it is also found in infancy; it is perhaps more common in winter than in summer, and in men than in women.

PATHOLOGICAL ANATOMY.—The changes in the brain and the blood-vessels predisposing to hæmorrhage have been already described when speaking of the ætiology.

The blood which escapes from the ruptured artery forces its way among the nerve-elements, separating some, tearing apart others, sometimes, if the clot is large, entirely isolating masses of cerebral tissue torn from their connections. At first the clot is dark red, uniform in consistency, resembling any other clot of blood. In a few hours the clot is somewhat less consistent, the watery constituents are absorbed, the color becomes lighter; finally only a pale, yellowish-colored remnant with a few blood-crystals can be found. The nervous structures which have been torn and bruised undergo fatty degeneration, there is more or less softening around the clot, the surrounding structures imbibe the coloring-matter and are yellow, and granular corpuscles and fatty degeneration increase the extent of this colored zone. Inflammation may set in which will destroy extensive tracts of brain-substance and aid in bringing about the fatal termination.

If the inflammation is slight and the patient survives, the cerebral tissue immediately around the clot undergoes a fibrous change, the interstitial elements increase, and a cyst is formed, a firm wall separating the diseased from the healthy brain-substance. If the clot is small, there may be no cyst; there is simply a small cicatrix of tough connective tissue. Sometimes there is no well-defined cyst-wall, the wall being soft and formed of a mixture of fibrous tissue and granular corpuscles in such proportions as not to have the firm-

ness above mentioned; then this character of tissue passes imperceptibly into the normal cerebral structure.

When the hæmorrhage is situated so as to implicate the deeper layers of the cortex and the white substance beneath, and especially the anterior two thirds of the posterior limb of the internal capsule, a secondary degeneration appears after some weeks or months, following the nerve-fibers, through the crus cerebri, pons, and medulla, into the cord.

According to Jaccoud, the clot remains soft and homogeneous during three to five days; then absorption continues to the tenth or twelfth day. After fifteen or twenty days the clot has contracted into a dense, solid mass of a yellowish color, quite different in appearance from coagulated blood. The new formation around the clot begins generally on the seventh or ninth day; toward the twentieth the cyst is formed, and by the thirtieth or fortieth the limiting membrane has become organized.

Symptoms.—Premonitory symptoms will be recognized less frequently with some classes of patients than with others; the less observing may give no heed to sensations or conditions which others may notice. Many patients have for a short time, or even days and weeks, preceding the hæmorrhage, symptoms which are worthy of notice. These premonitory symptoms are very important as indications for a course of treatment to ward off the threatening attack, and even if in the majority of such cases no attack occurs, yet they should never be neglected.

Among the symptoms which precede hæmorrhage for several days or even weeks, the most common are those which show disturbance of the circulation or nutrition of the brain; among these the most frequent are sensorial disturbances. Oftentimes there have been long-continued symptoms of cerebral hyperæmia, headache, or sense of pressure in the head, dizziness, disturbance of eyesight, noises in the head, pricking, and

numbness, especially in the fingers, perhaps also in the feet, sometimes on one side, sometimes on both ; mental confusion, slight forgetfulness of words, loss of memory, and change in disposition. Owing to impaired motor power or diminished sensation, there is a loss of delicacy in the touch, the character of the handwriting is changed, and there is awkwardness in using the hands.

Sometimes, if severe and persistent, especially if unilateral, these symptoms are caused by slight hæmorrhages which may precede a more severe one. If there has been a hæmorrhage with partial recovery, a recurrence of these symptoms may be the warnings of renewed danger.

Ophthalmoscopic examination may occasionally show the presence of miliary aneurisms, dilatations of the retinal arteries, or slight retinal hæmorrhages, and so be of value as indicating danger from the rupture of cerebral arteries.

The symptoms attending the rupture of a blood-vessel in the brain will vary according to the locality of the lesion, the size of the vessel, and the rapidity and force with which the blood escapes. It is easily understood, therefore, that there may be great diversity in the initial symptoms as well as in the subsequent course of the case.

The severest form of cerebral hæmorrhage is that which may be called apoplectic. The patient suddenly loses control over himself, falls, if he is either sitting or standing, and soon entirely loses consciousness. At the same time reflex action of the limbs is abolished, and, unless there are convulsions, the patient lies limp and inert, simply breathing and swallowing if the substance is put far enough back in the fauces to excite the involuntary muscles of deglutition, though occasionally even these fail to act. The respiration may become noisy from the paralysis of the soft palate or from the accumulation of mucus in the bronchi ; at first the

countenance is generally pale, but, if respiration is interfered with, it becomes dusky red. The cheeks flap back and forth with every respiration, passively following the current of air. The shortest time on record before death under these circumstances is five minutes; more frequently from half an hour to several hours elapse before the fatal termination.

The attack, as above described, is far from common; much more frequently there is a gradual development of the symptoms; the patient is conscious that something is amiss, and may try to rise from his chair, may be able to reach the bed or sofa, or, finding himself unable to hold articles in his hands, turns to speak to a friend and can not make himself understood. Soon he sinks powerless, and passes gradually into coma, which is not so extreme but that he can be aroused by a loud voice, or a powerful irritation may give rise to expressions of pain. Reflex movements generally persist. The unconsciousness may continue only a few minutes, or may persist much longer—even until death.

During the unconscious stage the physician can often recognize that one side is paralyzed. The mouth is drawn to one side, the limbs on one side are stiffer than on the other, or half voluntary movements are made only with one side; a strong irritation causes movements only on one side. But little can be learned from the condition of the pupils, as they are very variable; but many times the eyes are turned continuously, both toward the same side, and the head is rotated with the face toward that side. This phenomenon is not of long duration; it is seen in only a small proportion of patients, but, when present, it is valuable as indicating serious organic lesion, and may aid in determining its location. (See above, p. 44.)

After the return of consciousness it will be noticed that there is hemiplegia, generally affecting both the arm and the leg. This paralysis may be complete, total loss of power; cutaneous reflex action is usually

lost on the paralyzed side; after a few hours or few days power of motion returns, by degrees one act after another can be performed; usually the legs gain the most rapidly, the more complicated action of the arms and hands being recovered later. Hughlings Jackson has formulated the proposition that the most instinctive, automatic actions are the first to return.

The patient may continue to improve in his power of using his limbs for many months; perhaps to a casual observer there is finally complete recovery; but more frequently a stage is finally reached after which no further improvement can be expected; the limbs on one side are weak; certain motions can not be performed, or are executed only with difficulty and imperfectly; there is hemi-paresis.

The muscles of the face are often affected, and immediately after the attack the mouth is drawn to the opposite side, the naso-labial fold being more marked on that side. The nerve-fibers which supply the upper part of the face—the orbicularis palpebrarum, the frontal and corrugator supercilii muscles—usually are not affected. Nothnagel states that, when the tract of nerve-fibers passing along the base of the nucleus lenticularis is involved, the above muscles are paralyzed. They may be paralyzed also if the lesion is in the lower part of the pons.

Nothnagel also states that the muscles of the trunk are generally partially paralyzed.

Speech may be interfered with; the more frequently from loss of power over the organs of speech, the muscles of the throat, and mouth and tongue. Only occasionally is there aphasia when the right side is affected. This is a much more frequent symptom in embolism.

On the second to the fourth day after the attack symptoms may develop showing that there is an inflammatory process around the clot. There is headache, confusion of thought, feverishness, contraction of the

paralyzed limbs, sometimes slight convulsions. The duration of this stage is variable, from a few to several days; sometimes these symptoms recur two or three times or more.

Bourneville, considering the temperature, divides cases of cerebral hæmorrhage into three clases: 1. Fulgurant or multiple hæmorrhages, death occurring in a very few hours, with initial depression of temperature. 2. Cases ending in death in ten, fifteen, twenty hours, initial depression lasting only one to three hours or so, and subsequent rapid and considerable elevation of temperature. 3. Cases ending in death only after several days; initial depression of short duration; then a stationary period, continuing two to four days, with a primary slight elevation and subsequent oscillation about the normal; finally an ascending period. During the initial period of depression the pulse and respiration are but little changed. If the patient is to recover, the ascending period is of brief duration, and the temperature does not rise very high, or it is entirely wanting.

The above description refers to cases of severe cerebral hæmorrhage with loss of consciousness. In many cases there are no comatose symptoms. The patient more or less gradually loses power over one side, and falls with consciousness intact. The paralysis may be as complete, and the recovery of motion may occur in the same order, as in the other class of cases. After a first attack with the consciousness preserved, a second may occur soon in which that faculty is lost.

Yet lighter attacks occur in which there is only a slight impairment of motion in only one or more limbs, or the attack may be confined to the face. There are all degrees of severity, from the very lightest to the most complete.

Disturbance of sensation is not so common as that of motion. At first, indeed, sensation may be abolished, but it is more quickly recovered. Generally all varie-

ties of sensation are equally affected. Occasionally other parts than those whose motion is lost show diminution of sensation. Sometimes there is a persistent change of sensation, which is perhaps not always sought for. An object is perceived on both sides, but the impression is less acute on the affected side, or contact with the object excites also a peculiar tingling sensation besides the usual sense of touch. Sometimes there is increased sensitiveness to painful impressions.

Ollivier has found in many cases a change in the urinary secretion after cerebral hæmorrhage. There is first an increased secretion of urine, and albumen is found in it; later sugar may be found. These changes occur almost immediately after the attack and continue only twelve to twenty-four hours; they are not dependent upon the locality of the lesion.

Cutaneous reflexes may remain diminished or lost on the side affected. Tendon reflex is often exaggerated, especially after contractures have appeared.

Dr. Sanders has written upon hæmorrhages into the ventricles, separating such cases from both cerebral and meningeal hæmorrhages. He has collected ninety-four cases of such primary hæmorrhages, and considers their ætiology and pathology, which differ little from those of other cerebral hæmorrhages. The diagnostic symptoms he mentions are suddenness of the attack without premonitory symptoms; convulsions in the beginning, or later; partial or complete coma, paralysis, contracture, dilated or contracted pupils. Death usually occurs early, generally within twelve hours; a few patients recover. The above symptoms are almost exactly those found in any case of severe cerebral hæmorrhage. A positive diagnosis is in many or most cases impossible.

An *explanation of the phenomena* attending cerebral hæmorrhage will aid to a clearer understanding of the subject, and will be of value in determining treatment.

Several explanations have been given of the initial loss of consciousness, the more important of which are, that by Niemeyer, who refers it to cerebral anæmia caused by compression of capillaries; that by Trousseau and Jaccoud, and Jackson referring it to shock. The shock is direct on the side of the hæmorrhage, is transmitted or reflex on the other.

Nothnagel, after reviewing these and other theories, says: "We find ourselves, then, finally, obliged to admit that the physiological relations of hæmorrhagic apoplexy have not yet been made so clear as is commonly believed."

The most reasonable explanation seems to be that there is both shock with consequent exhaustion, and anæmia, not simply from compression of vessels, but also from reflex contraction.

The paralysis, both of motion and sensation, is the result of the direct injury, of the shock, of the local or general anæmia from compression of the smaller vessels, the tearing across of others, of the œdema, the infiltration of the surrounding cerebral substance with serum absorbed from the effused blood, this œdema also giving rise to anæmia. It is impossible to decide how large a share belongs to each of these elements in producing the patient's condition at the moment of recovery of consciousness. The influence of shock and anæmia due to compression from the size of the clot pass off soonest. The anæmia due to the œdema will slowly disappear; as the liquid parts of the clot are absorbed, the uninjured nerve-fibers surrounding the clot gradually regain their function. Another probable source of improvement is found in the possibility that functions performed by the destroyed nerve-elements may be acquired by those of other parts of the brain, so that in time there seems to be very little paralysis remaining. Also patients learn to use to the greatest advantage the power which remains. One cause of delay in recovery may be found in the functional inertia of

long disused nerve-fibers, so that even after organic restoration there may still be a period of diminished functional activity. Finally, it is not unlikely that many injured nerve-elements heal, and nerve-fibers which have been ruptured or bruised may recover their organic integrity. After these processes of repair have gone to their utmost limits, there must still be a very large number of nerve-elements destroyed beyond possibility of recovery. If, then, the hæmorrhage is so situated that these elements are necessary for perfect motion and sensation, there will be a residuum of paralysis from which it is utterly useless to expect recovery.

As a result of the imperfect healing of the torn nerve-fibers, a certain amount of paralysis remains permanently. After a few months—two to four—a stiffness of the paralyzed limbs is noticeable; there is a certain amount of contraction. The degree of the contraction varies from a scarcely perceptible stiffness of the fingers to a firm closure of the hand, with flexion at the elbow and adduction of the arm. The upper extremity is more frequently affected than the lower; the lower is rarely affected alone. The flexor muscles are almost invariably the ones affected. At first the resistance of the contracted muscles is easily overcome, and during sleep the muscles relax spontaneously. On first waking, the hand is as supple as the other; involuntary motions of stretching and yawning may be made by it in unison with the unparalyzed hand. Soon, however, as voluntary actions are performed, the contraction reappears, to persist until the patient again sleeps. Patients and their friends are often encouraged by this relaxation. It is far from being a favorable indication, and should never deceive the physician. Eventually the contraction may become persistent even during sleep.

These contractures must be distinguished from those which occur earlier, either at the time of the attack and soon disappear, or a few days after, at about the

time when inflammatory action arises around the clot. These also disappear within a short time. These latter varieties have been explained by supposing a direct irritation from the clot or from the subsequent inflammatory processes. The first variety of contraction has been explained, by Charcot and others, by the presence of secondary degeneration in the lateral columns of the spinal cord.

Associated movements, interesting to observe, are often seen in hemiplegic patients. After the partial return of voluntary motion, if the patient tries to move the paralyzed limb, the unaffected limb will involuntarily perform the same motion. It would seem that the motor impulse required to act on the partially paralyzed muscles needs to be so great to overcome the resistance offered by the injured nerve-fibers that the lower motor centers on the opposite side are also set in action, the impulse crossing by the commissures to the unaffected side.

In many cases of cerebral hæmorrhage, after a partial recovery of motion, the effort to perform an action gives rise to irregular contraction of the muscles of the paralyzed limb which may resemble chorea, or when the will is not exercised there may be slow, irregular, or more rapid movements of the partially paralyzed muscles. These post-hemiplegic movements are well described by Gowers. They vary from a very slight motion of the fingers to an almost constant motion of the whole arm, and even of the toes and leg. Among these movements is that which has been named athetosis by Hammond, which is more frequently seen after hemiplegia occurring in infancy, but may also occur in adult life.

More rarely there is sometimes seen a reflex tremor on the *healthy* side, occurring whenever the affected limb is moved.

The mental faculties are almost always impaired after cerebral hæmorrhage. In severe cases, of course,

these are at first entirely, or almost entirely, destroyed; but even in light cases it is soon noticed that patients are very different in disposition and intellectual power. They are irritable and emotional, easily angry, or easily bursting into tears. One who has been very guarded in the use of language may, on slight provocation, or with no provocation, break out into oaths. The emotional excitement is almost exclusively associated with *left* hemiplegia. Memory may be more or less defective. Even after almost perfect recovery some impairment of mental power may remain so as to render the patient unfit to carry on his business without assistance, and he may be so obstinate and suspicious as to render it advisable for him to give up all attempts to continue in business.

The paralysis is usually confined to one side of the body, the face, arm, and leg being affected on the same side, but on the side opposite the seat of the hæmorrhage. This is the common and regular form, a complete hemiplegia, and in these cases usually the upper branches of the facial nerve are not affected. There are, however, occasional irregular forms. What has been said in regard to localization of cerebral lesions will aid in diagnosticating the seat of the hæmorrhage in these cases. Occasionally all four limbs are paralyzed, either from multiple hæmorrhages or from large effusions into the pons and medulla. Such cases are rapidly fatal. In a very few cases the paralysis has been found to be on the same side with the hæmorrhage. Sometimes the arms are affected on one side, the legs on the opposite side. Occasionally only the cranial nerves are affected, or only the arm is paralyzed.

Acute bed-sores may form two to four days after the attack. They are situated on the paralyzed side, over the glutei muscles. Bed-sores may appear at a later period, being developed more gradually. They may occur on the knee or the heel, and are perhaps more frequent in elderly patients than in younger.

norance of such persons usually gives rise to inconsistencies in their account of the symptoms. The pretended contraction is not like the real. Associated movements on the healthy side do not occur when an effort is made to move the affected limb. Almost always the true hemiplegic will endeavor to aid the disabled hand with the well hand, or the body will be inclined to act as a fulcrum to help raise the arm. The pretender does not do this.

Jastrowitz* states that pressing the greater saphenous nerve about a hand's breadth above the internal condyle of the femur causes the testicle to rise on the healthy side, but has no effect on the hemiplegic side. It has been said that other reflex acts also do not take place on the hemiplegic side. When the skin is exposed there is no goose-flesh; tickling the nostril does not produce sneezing; touching the eyelashes does not cause winking. If there should be a difference between the two sides in these respects, a diagnosis as between narcotic poisoning or simulation and cerebral lesion could be made; but whether that lesion is a hæmorrhage or some other must depend on other data.

During the earlier hours or days the attempt to localize the lesion will often be useless. It is not until the effects of the shock and pressure of the clot have passed away that the more permanent symptoms can be recognized, and these must chiefly be considered in localizing the lesion.

Prognosis.—During the comatose stage, soon after the attack, it is impossible to form any opinion as to how severe the attack will prove; but the longer this stage continues the less favorable the prognosis, and if it lasts beyond forty-eight hours there is very little probability of recovery. If the attack is accompanied with severe convulsions, which are not due to epilepsy, the prognosis is the more serious, as the convulsions attend large hæmorrhages—those which burst into the

* "Berlin. kl. Wochenschrift," 1875, No. 31.

ventricles and those which are situated in the pons and medulla. The occurrence of Cheyne-Stokes respiration is of unfavorable augury.

There is a form of attack which has been called *ingravescent*, which is always fatal. With prodromic symptoms, coma gradually comes on, or there is a sudden loss of consciousness of short duration, after which intelligence is partially or entirely recovered, to be gradually lost again; the coma steadily deepens, and paralysis becomes more and more complete. The symptoms steadily increase in severity until the patient lies helpless and senseless, simply breathing, not to be aroused by any form of irritation. These cases are hopeless; the hæmorrhage occurs from one of the larger vessels between the lenticular nucleus and the external capsule, the nerve-fibers are pressed apart, not many are torn asunder; hence the earlier symptoms are slight.

After the initial depression of temperature, if the patient survives and the temperature steadily rises, the prognosis is unfavorable. If there is a slight rise and then a stationary period varying but little from 100°, after which another rise of temperature, then the prognosis is unfavorable again. If the temperature does not rise a second time, or falls to normal, the prognosis is favorable. When polyuria, albuminuria, and glycosuria exist in a very marked degree, the prognosis is grave.

During the period of inflammatory reaction the prognosis depends upon the intensity of the fever and attendant symptoms.

Acute bed-sores appearing shortly after the attack are extremely unfavorable, and are almost certain to be followed by death.

If the patient survives, a complete recovery is rarely to be expected. Trousseau considers that if the motor power returns in the leg first, before the arm, the prognosis is more favorable, at least for retention of mental

power. Recovery of motion may progress slowly for an indefinite period. If the late contraction appears, there is little or no chance of further improvement, and, in regard to disappearance of the contraction, the prognosis is absolutely unfavorable.

A second attack may occur in any one who has suffered from a cerebral hæmorrhage. If there is evident disease of the arteries, Bright's disease, or retinal hæmorrhages, this is more likely to occur. Also, if the patient be past middle life, there is more probability of another attack. After an attack, a return of prodromic symptoms would indicate renewed danger.

TREATMENT.—When the physician first sees a patient attacked with cerebral hæmorrhage, the injury has probably been done; in most cases the blood has ceased to escape from the ruptured vessel. Trousseau advocates very strongly to let the patient alone; others advise bleeding (Jaccoud, Huguenin) under certain conditions. If the patient is hearty, robust, with a strongly acting and healthy heart, and is evidently suffering from too great blood-pressure in the cranial cavity, they advocate general bleeding as the most effectual means of relieving this excessive blood-pressure. Bleeding is not indicated where the pulse is weak, if the patient is aged or feeble, or if there is heart disease, or when the coma has been of short duration and consciousness has returned. Practically, very few cases are suitable for bleeding under these conditions. In by far the larger number of cases nothing can be done except to place the patient on a bed with the head rather elevated, loosen all the clothing, and wait. As perfect quiet as possible should be maintained, the patient not moved, and fed with the simplest diet: if previously in full health and well nourished, it will be no disadvantage to feed sparingly; if in poor health and ill nourished, the feeding should be more abundant. If the heart's action is feeble and there is evident lack of vitality, stimulants, at first external, afterward, if neces-

sary, internal, should be used. After return of consciousness no special medical treatment is needed until the period of inflammatory reaction, when cold to the head, a laxative to open the bowels, and, if there is much headache, chloral, or some preparation of opium or belladonna, to relieve the pain. Occasionally, dry cups to the back of the neck or local bleeding may be called for. Ergot, by mouth or subcutaneously, may be used to diminish the danger of renewed hæmorrhage.

After the danger from inflammatory reaction is passed, many times there will be necessity for medical treatment. The patient should be kept quiet, secluded, in a well-ventilated apartment, with proper regard for all hygienic influences; should have a plain, unstimulating diet, not half starved, but should receive sufficient food. After a few weeks the nutrition of the muscles would be benefited by systematic rubbing, massage; later, after five or six weeks, electricity may be used. If proper care is exercised in not using too strong a current, and not continuing the application too long, this agent may be used without danger, and even with benefit, earlier than many authors advise it. The galvanic current, using from three or four to twelve cells, may be applied to the head—one electrode on the upper cervical vertebræ, the other over the mastoid process, or just below—or one pole on each side of the head, the positive on the same side with the hæmorrhage. Great care is to be taken not to suddenly interrupt the current, to use it only one minute, or at most two; to use a current which will not cause dizziness. Whether any benefit is ever obtained by this use of the galvanic current is extremely doubtful, and it is mentioned entirely upon the authority of several European observers.

The application of the induced or faradic current locally is attended with less risk, and is many times of positive benefit. One pole may be placed on some indifferent point, and the other passed lightly over the

different muscles, the current being graduated so as to cause the muscles to contract slightly without pain. The weakest current which will do this is strong enough. It is not necessary to move the limbs. The application should not exceed half a minute to a minute for each muscle, and this not continuously, but one muscle after another may be exercised for a few seconds, and then the limb be gone over again. This application will have the advantage of sustaining the nutrition of the muscles; also, the muscles would not fall into a state of sluggishness from simple inertia. Sometimes it will be found that electricity does harm; then, of course, it should be immediately omitted.

During the period of gradual recovery little can be done in the way of medication; absorption and restitution of structure and function advance slowly. Formerly, and even now, strychnia has been given very freely. It is not of sufficient benefit to offset the danger arising from its use in these cases. The absorbent properties of iodide of potassium render that drug acceptable, but with care not to disturb the stomach. Occasionally, especially in syphilitic cases, mercury has seemed of value. After the occurrence of late contraction, little or no improvement need be expected; though some cases are reported of benefit from electricity in these cases, I have never seen any.

As pneumonia and bronchitis are especially liable to attack the lung on the affected side, especial care should be taken after an attack to avoid exposing the patient.

After an attack the patient is anxious to guard against its recurrence. All measures necessary to sustain perfect health are in place—the avoidance of whatever will produce an increase of pressure in the cerebral blood-vessel, a quiet, composed life, with recreation and amusement sufficient for healthy action of the mind without excitement. If in active business, the activity should be moderated.

CHAPTER VI.

OCCLUSION OF CEREBRAL ARTERIES.

LANCEREAUX, E., De la thrombose et de l'embolie cérébrales considérées principalement dans leurs rapports avec le ramollissement du cerveau. Paris, 1862.—GELPKE, OTTOMAR, Vergleichende Zusammenstellung der Symptome von Hirnapoplexie und Embolie der Hirnarterien. *Archiv der Heilkunde*, 1875.—MEISSNER, Berichte über Embolien und Thrombosen. *Schmidt's Jahrb.*, 109, 117, 131.

The cerebral arteries may be suddenly plugged by the lodgment of a portion of a clot or other foreign body brought from a distance, an embolus, or gradually by the growth of a tumor, by the thickening of the walls of the artery, or by the coagulation of the blood at the point where the obstruction occurs. The sudden stopping of an artery by a clot brought from a distance is called embolism; the plugging by a clot formed on the spot is called thrombosis.

EMBOLISM.

ÆTIOLOGY.—The emboli may arise in the pulmonary veins, or the left side of the heart, or in any of the vessels between the heart and the point where they lodge. Much the more frequently they arise in the heart as the products of acute or chronic endocarditis. Warty growths form on the valves, are torn off and carried into the circulation, or a blood-clot forms in the heart and portions are broken off. Aneurisms of the aorta are sometimes the source whence the fragments of clot

arise. Disease of the lungs, as pneumonia or phthisis, cancer or embolism, or thrombosis of the pulmonary vessels, may serve as the point of origin of an embolus which may be carried by the pulmonary vein to the heart, and thence to the brain. Any diseases, then, which may give rise to endocarditis or the above pulmonary affections are remote causes of embolism.

PATHOLOGICAL ANATOMY.—It is not necessary to describe the changes which the embolus undergoes, except to say that in rare cases it is broken down and absorbed.

Immediately after the occlusion of an artery the blood from the veins flows back into the distal branches of the obstructed artery, and there is more or less stasis. In the brain it is very rare that the anastomoses are sufficient to maintain an active circulation, hence the region which depends upon these branches for its nutrition suffers from lack of healthy blood; the walls of the vessels also suffer and allow the blood to escape. The cerebral tissue is infiltrated with serum, the blood undergoes change, and its coloring-matter is diffused through the part, and gives a red or yellowish tint to the broken-down nervous tissues.

The nervous elements, being deprived of healthy blood, lose their vitality, soften, undergo fatty degeneration, and are reduced to a semi-fluid pulp. If the region affected is small, this may be absorbed, a cicatrix is formed, and the spot of softening may finally disappear, but more frequently a cyst remains filled with serum and crossed by bands of connective tissue.

The softening does not show itself immediately; it is perceptible only thirty-six to forty-eight hours after the occlusion of an artery.

Owing to the direction in which the different arteries are given off from their main trunks, emboli are much the more frequently carried into the left carotid, and are generally lodged in the middle cerebral artery or one of its branches. Duret has described the distribu-

tion of softening which belong to the various branches of the cerebral arteries.

SYMPTOMS.—Generally without warning, the patient is attacked with loss of consciousness and entire loss of power. There may be, for a few minutes, headache or vertigo, but the attack is usually sudden and complete. The loss of consciousness is of less duration than in hæmorrhage, and may be only momentary. It is frequently accompanied with general epileptiform convulsions. Vomiting and delirium are sometimes present. Occasionally paralysis is the first symptom. Consciousness is not always lost; there may be merely confusion of thought.

After recovery of consciousness and voluntary power it will be found that the patient has paralysis of one side. Usually the face and limbs of the side opposite the lesion are affected, and as the left middle cerebral artery is much the more frequently the seat of embolism, the right side is usually the one affected.

The temperature is slightly lowered immediately after an attack, but quickly rises, and, if the case proves fatal within three or four days, the rise of temperature is almost unbroken. If the patient lives from five to fifteen days, the temperature is irregular. If the attack is not fatal, the temperature falls to very nearly or quite to the normal after three or four days.

After the earlier stage of the disease the symptoms are essentially the same as in hæmorrhage. Contraction of the limbs is seen less frequently than after hæmorrhage, yet is essentially the same when it occurs. The intellectual disturbance is rather less marked.

Disturbance of the faculty of speech is not uncommon in embolism, and when the right side is paralyzed there is almost always aphasia.

Occasionally the aphasia is the only symptom present; there is no loss of consciousness, nor paralysis. Some of these cases are due to embolism of small ar-

teries, some are due to disturbance of circulation depending upon other causes.

The embolus may lodge in an artery, stop the blood-current for a few minutes, and then, by a change of its position, the blood may be able to pass it in sufficient quantity to restore the nutrition of the brain. If the embolus is then broken up and absorbed, there may be no further trouble; or if it adheres firmly to the wall of the vessel, there may be no further trouble. If, however, its position is again changed, it may plug the vessel finally, and then the symptoms become permanent.

When other than the middle cerebral artery is plugged by an embolus, the symptoms will vary according to the portion of the brain affected.

DIAGNOSIS.—The diagnosis is almost exclusively between hæmorrhage and embolism. The question as to the diagnosis of thrombosis is reserved till the latter affection has been considered.

It is frequently impossible to be certain whether there has been embolism or hæmorrhage; but, by a careful consideration of all the symptoms and other circumstances, it will generally be possible to form a satisfactory diagnosis.

Gelpke has given a valuable review of the diagnostic points between apoplexy and embolism. The first is age; apoplexy is by far the more frequent after fifty—embolism before fifty. More than sixty per cent (nearly or quite seventy per cent) of the cases of apoplexy occur after fifty; more than sixty per cent of the cases of embolism occur before fifty.

In hæmorrhage there is disease of arteries; so this is found most frequently in connection with Bright's disease or where there are atheromatous changes. Embolism occurs most where there is cardiac valvular disease. In hæmorrhage there may be premonitory symptoms; in embolism the attack occurs generally without warning. When there seem to be pre-

monitory symptoms, they are rather due to independent attacks.

The symptoms which are found during and immediately after the attack may be very much alike, yet there are slight differences which may aid in diagnosis. Both embolism and hæmorrhage are frequently accompanied with hemiplegia; but in embolism it is almost always on the right side; in hæmorrhage on either side: so left hemiplegia would rather point to hæmorrhage; right hemiplegia not necessarily to embolism. Epileptic attacks at the time of seizure rather indicate embolism. The muscular paralysis is greater in embolism than in hæmorrhage. Aphasia, agraphia, and amimia depend upon changes in or near the island of Reil, and are the more frequent in embolism. Ataxic loss of speech depends upon lesion of the corpus striatum, and is the more common in hæmorrhage. In embolism the symptoms of cerebral pressure are wanting, as diminished frequency of pulse, stertorous respiration, vomiting, contracted pupil, and strabismus.

After the attack the mental powers are more likely to be affected in hæmorrhage, especially the emotional faculties. There is also more likely to be reaction; and a return of function, if it occurs, is slower in hæmorrhage.

The discovery of an embolism in other arteries, as radial, femoral, etc., would aid in establishing a diagnosis.

The temperature may also assist. In embolism, during the first few hours, the initial depression of temperature below normal is wanting or very slight; in hæmorrhage it is much more marked. In embolism, after a temporary rise, the temperature returns to the normal with irregular exacerbations or evening elevations; in hæmorrhage it returns less quickly to the normal unless a second hæmorrhage occurs. After the stationary period the rise of temperature is slower in

embolism, and generally does not attain so high a figure as in hæmorrhage.

Exceptional cases of embolism occur where the variation of temperature more nearly resembles that found in hæmorrhage.

If there is complete recovery within a few days after an attack of complete hemiplegia, there probably was not hæmorrhage.

PROGNOSIS.—"At the outset of and during the primary attack, no prognosis can be given as to the probable course of the case, except that its severity is likely to be proportionate to the extension and severity of the primary symptoms. If the paralytic symptoms disappear after a brief period, there will be no reason to fear the presence of serious structural disease, but the chance of future attacks can not be excluded." ("Ziems. Cyclop.")

Severe cases are more serious than severe cases of hæmorrhage, and more likely to prove fatal.

When the vertebral or basilar artery is plugged, the prognosis must necessarily be very unfavorable.

TREATMENT.—Unless the patient is very feeble and requires slight stimulation, the less done during the first few days the better. Blood-letting and depressing measures are decidedly contra-indicated. If there arise indications of cerebral irritation from collateral hyperæmia, the bowels should be freely acted upon and the head kept cool.

The subsequent treatment may be the same as in cerebral hæmorrhage.

THROMBOSIS OF CEREBRAL ARTERIES.

ÆTIOLOGY.—The causes of thrombosis of cerebral arteries are to be found either in the vessels themselves, in the composition of the blood, or in the diminished action of the heart. The walls of the vessels may become roughened from disease, as in arteritis obliterans, whether syphilitic or not, and also in case of atheroma,

especially if the inner epithelial layer is broken; the vessel may be contracted through a small extent of its length, and thus the blood-current be retarded. The disease of the walls of the vessels as found in old age interferes with their normal elasticity, and so favors retardation and coagulation of the blood. Disease or weakness of the heart may also cause the blood to flow less rapidly, and so favor the formation of a thrombus; or the blood may coagulate more readily than usual, as in certain febrile diseases. Disease of arteries and feebleness of heart's action are found combined in old age; consequently thrombosis is most common in advanced life; very few cases occur below forty years of age; most patients are over sixty.

PATHOLOGICAL ANATOMY.—When the thrombus fills an artery whose branches are not connected by anastomoses with other arterial supply, the brain, deprived of blood, must necessarily undergo the same degeneration as is found in embolism. Often the smaller end arteries are stopped up, and then small spots of softening will be found throughout the brain; this is most commonly seen in the white substance. The cavities thus formed may vary in size from a sixteenth of an inch in diameter to an inch or more; if numerous, they average an eighth to a quarter of an inch; they are crossed by bands of connective tissue containing blood-vessels; there may be a new growth of vessels if inflammation has set in. The walls of these cavities usually contain granular corpuscles. If recent, the nerve-fibers around the border of the cavity may show enlargement of axis-cylinders and other inflammatory changes.

Sometimes, instead of a cavity, cicatricial tissue forms and a hard nodule is left.

SYMPTOMS.—As the arteries are usually closed gradually, there is a less sudden onset of the symptoms than is found in embolism. For a variable length of time the patient has had more or less discomfort in the head —pain or dizziness—memory may be less strong, the

patient may show signs of mental disturbance or temporary loss of consciousness, which are referred to old age, or there may be actual insanity; sometimes temporary loss of power or abnormal sensations in the limbs, which soon pass away, but again appear, show that there is serious disturbance of the cerebral circulation. If the region of the pons and cerebral peduncles are affected, individual cranial nerves may be more or less paralyzed. There may be a numbness and tingling in the limbs on one or both sides, or a slowly increasing paralysis may be the result of gradual loss of power in the motor tracts.

After these undefined, perhaps doubtful and confusing, symptoms have continued, it may be for months, there may occur an apoplectic attack; the vessel which had been only partially obliterated is suddenly entirely plugged; then the symptoms of embolism follow.

DIAGNOSIS.—Thrombosis and embolism differ in the above premonitory stage, which perhaps may be described more properly as the gradual development of the symptoms.

It may be almost impossible to decide whether there is a thrombus or a hæmorrhage when the artery is finally plugged suddenly. A number of independent attacks of paralysis, of unconsciousness, or of dizziness, from which the patient soon recovers, followed by the finally permanent attack; the advance of the symptoms by stages, as it were, with intervals when there was no advance—would point to a thrombus rather than a hæmorrhage. A preceding or accompanying acute disease, as pneumonia, or a great general feebleness, should cause a suspicion of thrombosis. Age, disease of arteries and of kidneys, would be as significant of one as the other.

After the apoplectic attack the diagnosis of thrombus would be made from the same peculiarities as are found in embolism.

When there are several attacks of unconsciousness,

or of paralysis, or of dizziness, there may be a question as to whether the patient is suffering from epilepsy. It should be kept in mind that epilepsy rarely originates in old age; a careful study of the phenomena attending the attack will show a lack of resemblance with epilepsy; bromide of potassium is more likely to aggravate the symptoms in thrombosis, but relieves them in epilepsy.

The diagnosis of locality must be made from comparison of symptoms with facts which have already been given, only remembering that the symptoms due to pressure would be absent.

PROGNOSIS.—The physician must judge of the danger to life by the course and severity of the symptoms. The more extended the signs of disturbance, the longer the unconsciousness continues, the more certain the lesion can be located in the mesencephalon, the more serious must be the prognosis.

TREATMENT.—When a diagnosis of thrombosis can be made, the treatment should be tonic and mildly stimulating; when it is impossible to certainly exclude hæmorrhage, it is not desirable to give stimulants freely; and in very doubtful cases it may be better to do nothing more than to put the patient in as favorable hygienic conditions as possible, insure quiet, rest, and absence of excitement. If the patient is young, and has had syphilis, a course of iodide and mercury would certainly be appropriate.

After an apoplectic attack the treatment would be the same as under similar circumstances arising from other causes.

CHAPTER VII.

TUMORS OF THE BRAIN.

LADAME, PAUL, Symptomatologie und Diagnostik der Hirngeschwülste. 1865.—MACABIAN, JEAN FIRMIN, Quelques considérations sur les tumeurs du cervelet. Paris, 1869.—BRAMWELL, BYRON, Clinical Lectures on Intra-cranial Tumors. *Edinburgh Med. Jour.*, 1881.—NOTHNAGEL, H., Topische Diagnostik der Gehirnkrankheiten. Berlin, 1879.—BERNHARDT, M., Beiträge zur Symptomatologie und Diagnostik der Hirngeschwülste. Berlin, 1881.—KLEBS, E., Beiträge zur Geschwülstlehre — Hirngeschwülste. *Prag. Vierteljschr.*, cxxxiii.—JACKSON, J. H., Diagnosis of Tumor of Brain. *Med. Times and Gaz.*, August 9, 1873.

PATHOLOGICAL ANATOMY. — Many of the tumors which are found within the skull are not properly tumors of the brain—that is, do not take their rise from the cerebral tissue, but arise from the meninges or the blood-vessels; some arise from the bones of the skull. As all these are revealed to us by the symptoms produced by their influence upon the brain, it is proper and most convenient to include all under the term "tumors of the brain."

It is unnecessary to give details of the histology of the different kinds of tumors which may be found in the brain or connected with its envelopes. Tubercle, cancer, gummata, sarcoma, osseous growths, myxoma, and lipoma do not differ essentially from the same growths found elsewhere. Glioma, psammoma, and cholesteatomata are among the tumors which more peculiarly belong to the brain. The first, glioma, is simply a development of the normal neuroglia with sometimes the admixture of more or less fibrous tissue.

They vary in consistency according as cells or fibrous tissue predominate.

Psammoma consist in the formation of granules of calcareous substance infiltrated into the cellular tissue. There may be also hyperplasia of the pineal gland or of the pituitary gland.

ÆTIOLOGY.—Certain tumors, as tubercle, cancer, or syphilitic gummata, must be referred to a constitutional diathesis. Blows and falls upon the head are often the direct cause of the development of abnormal growths. The cause of a large number of tumors can not be certainly discovered.

SYMPTOMS.—There are certain symptoms which may be called general, or common, which are found in almost every case of intercranial tumor without reference to its locality; other symptoms depend upon the situation of the tumor and aid in locating it. The latter symptoms are important as well in assisting to form a diagnosis of the presence of a tumor, the common or general symptoms oftentimes not being definite enough for that purpose.

Sometimes a tumor gives rise to so few symptoms, or they are so slight, that no notice is taken of them, or there may be no symptoms. This is more likely to occur where the tumor is quite small, where it is situated in certain parts (anterior or posterior) of the white substance, and where it is very slow in growth.

The symptoms depend upon the immediate effect of the tumor upon the nervous structures, destruction or irritation; upon its effect at a distance, irritation and pressure; and upon the inflammatory or other changes which it excites in the cerebral substance, more especially in its vicinity.

The general symptoms are, in most instances, due to the remoter effects or to the less direct changes excited by the new growth. The most common symptom is headache; this is also usually one of the first, and is characterized by its persistency and the severity of the

paroxysms. Remissions, sometimes even intermissions, may occur, in which the headache is replaced by a feeling of slight discomfort in the head. In some cases a slight noise or the least motion brings on an attack. When severe, remedial agents have no power to alleviate the pain. The more rapid the growth of the tumor, the more severe and persistent is the headache.

Dizziness, or vertigo, is a frequent symptom in the early stages of the disease. Nausea and vomiting are very frequently present, and may be very obstinate. These are rather the more frequent when the posterior part of the hemispheres or the cerebellum is affected.

Various mental disturbances belong among the earlier symptoms, such as change of disposition, a pleasant, good-natured patient becoming cross and irritable, or one who has been careless and unconcerned taking upon himself the opposite qualities; one who has been open and frank becoming silent, morose, and suspicious. Memory fails, power of mental application is lost, and business cares and responsibilities become a burden. None of these changes are sufficient to rank as insanity; there is simply slight mental disturbance, which may be perceived even before the headache shows itself.

When all these symptoms are found together, persisting in spite of treatment or only partially relieved thereby, the presence of cerebral tumor is almost certain. The diagnosis is yet more certain when any of the following symptoms are also present:

There may be a general failure of muscular strength, no definite paralysis of any set of muscles, but a simple and increasing enfeeblement. When the cerebral disease is secondary to disease elsewhere, as tubercular or cancerous, it may be impossible to decide how much this weakness is due to the constitutional state, or to the local disease in the brain.

Spasms and convulsions are sometimes so general or indefinite that they are to be considered as general symptoms. They are, however, probably dependent

upon an irritation of the cortical gray substance, either direct or remote, either primary or reflex. Spasms localized in one or a few groups of muscles belong to the localizing symptoms.

There is rarely pain in the limbs, but various abnormal sensations, as numbness, formication, pricking, and tingling, are not uncommon; sometimes there is great diminution of ordinary sensations.

Disturbances of special senses, excepting eyesight, are not common; deafness and anosmia are rare; taste is lost only or chiefly when the mesencephalon is affected. Diplopia, amblyopia, amaurosis, and hemianopsia are not rare; they belong more especially to localizing symptoms, and have been more or less fully considered already.

Disturbance of speech, rotatory movements, compelled movements forward or backward, conjugate deviation of the eyes and head, also belong to localizing symptoms, and have been considered in previous pages.

Optic neuritis is very frequently one of the secondary or remote symptoms of cerebral tumor, and, when present, may be of great value in forming a diagnosis. In every case of cerebral disturbance the ophthalmoscope should be used, whether there is disturbance of vision or not. The neuritis may exist unsuspected by the patient.

Other signs of ill health may be present, as emaciation, anorexia, pyrexia, constipation, retention or incontinence of urine, disturbance of circulation or of respiration.

The symptoms which enable one to localize the tumor have been mentioned already; they are also important as showing the presence of organic change in the brain, but other symptoms indicate the nature of that change.

The temperature of the body is not very often raised, but several observations of the temperature of the head show that there is an increase of surface-heat on the

side where the tumor is, especially immediately over it. This branch of inquiry has not been pursued far enough as yet to decide whether the degree of elevation has any relation to the nature of the tumor or its rapidity of growth. Great care is necessary in taking these observations to guard against errors.

The *course* of the disease varies greatly according to the rate of increase and the situation of the new growth. It is hardly necessary to say that a rapidly growing tumor will cause many more severe symptoms than one which increases in size more slowly. Inflammation or congestion around a tumor may give rise to a sudden outbreak of symptoms, and their rapid increase in severity. This may subside, and then a remission would succeed.

As a rule, with occasional remissions, or even intermissions, in the symptoms, there is a steady advance; the headache may at length diminish in intensity, perhaps because of destruction of nerve-fibers, the convulsions may cease because the motor areas are destroyed, but with the apparent improvement the mental powers will be found to have diminished, the paralysis to have increased, the patient is more helpless; while suffering less, he has evidently lost ground.

At length there is complete hemiplegia, or possibly paralysis of the entire body, the bladder and rectum are affected as in other cases of hemiplegia, and bedsores form; it becomes constantly more difficult to give the proper nourishment, and the patient dies exhausted.

Many times, however, the fatal termination is more speedily reached, the respiration or heart's action is interfered with, or pulmonary complications set in, and the patient soon dies.

The *duration* is very variable; cancer, tubercle, and syphilitic growths are rapid in their course; glioma may slowly advance for years, with many intermissions.

There are many questions which it would be inter-

esting to consider, but the object is simply to lead to a correct diagnosis, and indicate the most rational treatment.

DIAGNOSIS.—The diagnosis of tumors of the brain from other cerebral affections is by no means always easy. *Tubercular meningitis* may sometimes closely resemble tumor. It is more common in children than adults, while tumors are more frequent in adults.

Chronic thickening of the membranes, especially if occurring at the base, so as to involve the cranial nerves, may give rise to exactly the same symptoms as a tumor. Such thickening is more frequently seen as the effect of a syphilitic taint.

Abscess of the brain is usually accompanied with less headache, is less likely to have ocular symptoms, to cause vomiting and vertigo, and is more frequently the result of an affection of the ears.

Apoplexy and *embolism* may generally be distinguished from tumor by the suddenness with which the symptoms occur, and the peculiarities of the first attack. Nevertheless, in rare cases, tumors have remained comparatively latent for an indefinite period of time, and then manifested their presence by an attack closely resembling apoplexy. A careful study of the symptoms attending the onset, and the previous state of the patient's health, especially whether any of the general symptoms indicating tumor existed, also an examination of the heart and kidneys, would assist materially to a correct diagnosis.

An obstinate and persistent headache, such as sometimes occurs at puberty, may give rise to anxious forebodings, lest it should be significant of serious brain trouble; much more would such anxiety arise if the headache were attended with attacks of vomiting. A careful study of the whole history of the case, and a careful examination of the patient, will aid more than anything in forming a diagnosis; but it may be necessary to wait for time to settle the question.

PROGNOSIS.—The result is almost invariably fatal, the patient being finally worn out by bed-sores, or debilitated by insufficient nourishment from the difficulty of swallowing or the continuous vomiting. Sometimes the fatal termination occurs during an epileptic attack, or in an attack of apoplexy. It is almost impossible to judge with any degree of certainty in regard to the duration of life.

Possibly, if the growth is syphilitic and has not attained too large a size, there may be recovery; though recovery itself might throw some doubt upon the correctness of the diagnosis.

TREATMENT.—The treatment of cerebral tumors may be included under two heads—treatment designed to cure the patient, and that intended to relieve certain symptoms. Where there has been a previous syphilitic infection, it is advisable to pursue an active course of antisyphilitic treatment—iodide of potassium without hesitation in sufficient doses to produce a marked effect within a comparatively short time, and mercury. Even where syphilis is not proved, a course of iodide of potassium may be of great benefit.

Counter-irritation to the head and neck has been recommended. It is extremely doubtful if the growth of a tumor is influenced thereby, though some of the unpleasant symptoms may be mitigated.

To fulfill the second indication—if convulsions of an epileptiform character are frequent—one of the bromides in large doses may give more or less relief. As headache is one of the most distressing symptoms, patients are imperious in their demands to be relieved therefrom; if apparently not very severe, milder measures may first be tried, as counter-irritation to the neck, bromide of potassium, cannabis Indica, caffein. If the pain is severe, probably nothing will relieve it except one of the preparations of opium. There should be no hesitation in using this drug in such doses as to give relief. Often a comparatively small dose will accom-

plish the purpose; it is well occasionally to intermit its use to learn whether the headache may not have ceased; at such times a weak solution of quinine may very conveniently take the place of morphine by the mouth, or water may be used for subcutaneous injection.

The vomiting which accompanies cerebral tumor is often very obstinate and intractable. It should be treated according to the condition of the patient. Counter-irritation to the head and neck or over the stomach should not be omitted when other means fail. Ice to the head and neck may be useful. If other means do not succeed, morphia may control it.

Of course, the conditions of the patient, which require special treatment, as cystitis, bed-sores, etc., should receive the necessary care.

CHAPTER VIII.

CEREBRAL ABSCESS.

KÖHLER, A., Ein Beitrag zur Lehre von Gehirnabscess. *Schmidt's Jahrb.*, 183, 1879.—MEYER, R., Zur Pathologie des Hirnabscesses. Zurich, 1867.—NAETHER, R., Die metastatischen Hirnabscesse nach primären Lungenherden. *Deut. Arch. kl. Med.*, xxxiv, 1883.—THOMPSON, H., Case of Otitis, Cerebral Abscess, etc. *Med. Times and Gaz.*, March 29, 1878.—FENGER, CHRISTIAN, On Opening and Drainage of Abscess Cavities in the Brain. *Am. Jour. of the Med. Sci.*, July, 1884.

ABSCESS OF THE BRAIN.

ÆTIOLOGY.—The causes of cerebral abscess are almost always evident. One of the most common, perhaps the most common cause, is inflammation of the ear. When the otitis affects the bone, there is always danger that the disease may extend to the brain; either the bone is perforated, and so the dura and pia mater exposed to direct irritation, or, as is more frequent, the inflammation is transmitted through the foramina or veins. In all cases of otorrhœa in children, the danger of this complication should be kept in mind.

Injuries to the head, whether attended with fracture of the bone or not, are frequent causes of cerebral abscess.

Disease of the bone, caries, from whatever cause, may give rise to abscess. An abscess may form about a hæmorrhage, or the infarctus due to an embolism or thrombus.

Pyæmia may be a cause; erysipelas and acute diseases seem sometimes to give rise to cerebral abscess.

PATHOLOGICAL ANATOMY.—The division has been made of red and yellow inflammation: the distinction depends upon the relative amount of blood or pus contained in the diseased tissue. Abscesses may be encysted, i. e., surrounded by a close, firm layer of new-formed tissue; or they may have no containing wall, the pus, mixed with *débris* of the cerebral substance, being in direct contact with the softened, partly broken-down tissues.

The abscess may vary in size from very small to include nearly a whole hemisphere.

The histological changes which the tissues pass through consist in a cloudy swelling of the elements, a gradual breaking up into granular *débris* and transformation into granular corpuscles, and increase of connective-tissue fibers and cells around the focus, when a capsule is formed.

The nervous elements themselves are destroyed by fatty or granular degeneration, sometimes preceded by swelling, or hypertrophy of axis-cylinders and nerve-cells.

Around the abscess there may be very great œdema of the cerebral tissue, and sometimes congestion, sometimes an anæmic condition.

SYMPTOMS.—The symptoms are quite similar to those attending tumor of the brain. Headache is one of the most common. This is severe, and generally continuous. If not intense, there is at least a feeling of discomfort, of pressure, of lightness, or dizziness.

Mental confusion, disturbed memory, sometimes delirium, show how seriously the higher intellectual faculties are implicated.

Nausea and vomiting may be among the earlier symptoms, and, if very persistent soon after an injury to the head, or if they set in during an attack of otorrhœa, should give reason to suspect abscess.

Paralysis and disturbance of sensation other than headache are not very common; sometimes the motor

centers, or motor tracts, are affected, and then local paralysis or hemiplegia may be noticed.

Convulsions are rather common; they may be local only; or, as is more frequent, beginning as local, they become general; they may, from their commencement, be clearly epileptic in character. The increased irritability of the motor centers caused by the inflammation fully explains their occurrence.

When pressure increases, there may be the corresponding symptoms—retarded pulse, stupor, stertorous respiration, etc.

Sometimes meningitis is one of the results of the injury or disease causing the abscess; then the symptoms of cerebral meningitis may predominate.

Either the abscess runs a rapid course, terminating fatally within a few days or weeks, or a period of remission may set in; the abscess may remain latent for an indefinite time. When thus latent, it is probably always encysted. During this time of remission or latency, some of the symptoms may persist in a diminished degree of intensity. In other cases there is no acute initial stage; the abscess from the beginning runs a chronic or concealed course. At length an active inflammation starts up around the abscess, and all the symptoms are aggravated, the patient becomes paralyzed and comatose, and soon dies; or the œdema around the focus of disease increases suddenly and rapidly, and so the patient dies.

The abscess may rupture into the lateral ventricles, giving rise to sudden aggravation of the symptoms; general convulsions are usually excited; there may be loss of consciousness.

Very rarely a local meningitis is excited, the membranes adhere, the bone is perforated, and the abscess is discharged externally. The evacuation may be followed by recovery. Occasionally recovery results without discharge of the contents.

DIAGNOSIS.—Cerebral abscesses have many symp-

toms common to cerebral tumors: headache, vomiting, dizziness, the symptoms of pressure, are common to both affections.

The preceding otorrhœa, or the history of an injury not long before the cerebral symptoms are developed, would indicate an abscess. If a tumor follows an injury, a much longer interval must elapse before it shows its presence. Local paralysis and hemiplegia are more common with tumors, excepting toward the later stages. A rapid increase in severity of symptoms after a period of remission, amounting perhaps almost to intermission, is indicative of abscess. Local convulsions, followed sometimes by general convulsions, are met in tumors of the cortex; general convulsions, without preceding local spasms, are more common in abscess. The diagnosis between abscess and tumor may, however, frequently be impossible.

Meningitis is accompanied by higher fever, by more marked cutaneous hyperæsthesia, less frequently by convulsions. Yet the diagnosis may be very difficult or impossible, especially as meningitis may be produced by the same causes as abscess, and the two diseases may co-exist, one as the cause of the other, or both depending upon the same cause.

The diagnosis of locality is less easy than in other forms of cerebral disease. The same general principles should guide in a decision, but, as a rule, the data upon which to found an opinion are fewer and less trustworthy. After an injury the abscess may be on the opposite side, the brain having suffered there by *contre coup*.

PROGNOSIS.—Recovery is the exception—so rare that little account need be taken of such cases. A remission may excite strong hopes of cure, but there is the constant danger of a return of the symptoms in a more aggravated form.

TREATMENT.—After an encephalitis has gone so far as to give rise to an abscess, medical skill is compara-

tively powerless. The treatment must then consist in quiet, rest, and avoidance of excitement. Counter-irritation might be used if there is doubt as to the abscess having been formed ; mercury, by mouth or inunction, is strongly advised. Cold may be applied continuously.

More benefit can be hoped from the use of precautions to prevent the formation of an abscess. Otorrhœa should never be neglected, especially in children ; an effort should be made to cure the local disease.

When there has been severe injury to the head, rest and quiet should be maintained for a while, and, if there is attendant headache, the enforced quiet should be kept up till that disappears ; during this time the diet should be sufficient, but light, easily digested, and unstimulating ; cold applied continuously, and leeches may be used. Free action of the bowels by cathartics.

The question of trephining in injuries to the skull belongs to surgery.

DISEASES OF THE SPINAL CORD.

Sci., 1878.—Ott, J., and Smith, R. M., The Paths of Conduction of Sensory and Motor Impulses in the Cervical Segment of the Spinal Cord. *Am. Jour. Med. Sci.*, Oct., 1879, p. 438.—Starr, M. Allen, Localization of the Functions of the Spinal Cord. *Am. Jour. of Neurol. and Psych.*, Aug. and Nov., 1884, p. 443.—*Ibid.*, The Sensory Tract in the Central Nervous System. *Jour. of Nervous and Ment. Diseases*, July, 1884, p. 327.—See, also, Ross, Erb, etc.

ANATOMY.

The membranes of the spinal cord are usually described as three—the dura mater, arachnoid, and pia mater. The dura mater is double, the outer portion forming a periosteum for the vertebræ; the inner layer is connected with the outer by loose connective tissue, containing fat and blood-vessels. This inner layer is the portion usually referred to in speaking of the dura mater.

Opinions differ as to the arachnoid: some authors consider it as forming part of the other two membranes, while others regard it as a distinct membrane.

The pia mater is closely adherent to the cord, and through it run the nutrient vessels for the cord. It sends processes into the fissures of the cord; it surrounds the nerve-roots in their course from the cord to the dura mater. From the pia mater, on each side, arises the ligamentum denticulatum, which keeps the spinal cord in the center of the spinal canal. It runs the whole length of the cord, and stays it, by means of twenty to twenty-three teeth-like processes, to the dura mater.

The space between the pia mater and dura mater is filled with the cerebro-spinal fluid, which is contained in a very loose, wide-meshed connective tissue.

The spinal cord is then suspended in the cerebro-spinal fluid by means of many processes of the pia mater, by the nerve-roots, and the posterior septa. This suspension is so contrived that the influence of jars and shocks may be reduced as much as possible.

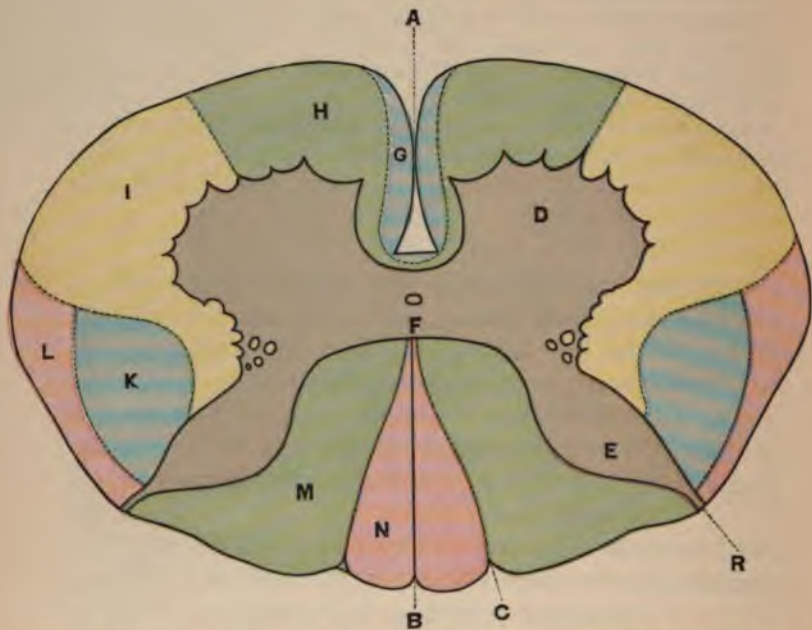

FIG. 16.—*Diagram illustrating the relations of the nerve-fiber tracts in the spinal cord.* The section is supposed to be taken transversely through the lower part of the cervical enlargement (slightly modified from Flechsig by Hammond):

A. Anterior Median Fissure.
B. Posterior Median Fissure.
C. Intermediate Fissure.
D. Anterior Gray Cornu.
E. Posterior Gray Cornu.
F. Gray Commissure, with Central Canal.
G. Uncrossed Pyramidal Tract (Flechsig), or Column of Türck.
H. Fundamental Part of the Anterior Column (Anterior Root-Zones of Charcot and his pupils).
I. Anterior Part of Lateral Column.
K. Crossed Pyramidal Tract of Lateral Column.
L. Direct Tract from Lateral Column to Cerebellum.
M. Column of Burdach, Posterior Root-Zones of Charcot and his pupils.
N. Column of Goll.

The posterior columns of descriptive anatomy include the fields M and N extending on the surface from B to R. The antero-lateral columns extend on the surface from R to A. Their anterior division includes the fields G and H; their lateral division, the fields K, L, and I.

The anastomoses between the arteries on the surface of the cord are very free, especially in the cervical and lumbar portions, least so in the dorsal region. The smaller arteries in the interior of the cord anastomose quite freely. The central gray substance and internal parts of the cornua are supplied chiefly by one set of vessels; the white substance and outer part of the cornua by another. The gray substance has a larger blood-supply than the white.

The cord extends lower in women than in men—in the former reaching the second lumbar vertebra, in the latter only the first. In men the proportion between the cord, the vertebral column, and the length of the body, is 1 : 1·62 : 3·76; in women the same proportion is 1 : 1·56 : 3·58.

The gray matter is arranged around a central cavity called the *central canal*, which extends throughout the length of the cord. Two processes project forward, one on either side, called the *anterior cornu;* two similar processes project backward, one on either side, called the *posterior cornu.* The gray matter is relatively larger, and the cornua are thicker, in the cervical and lumbar enlargements, and smaller in the dorsal region. In the anterior cornua are large nerve-cells, with many processes arranged approximatively in groups—the internal, the anterior, the antero-lateral, the postero-lateral, and central. Lockhart Clarke gave the name tractus intermedio lateralis to a group of cells corresponding to the postero-lateral group mentioned above. At the junction of the posterior cornua with the central gray matter there is found also, at certain levels, a group of cells of very nearly equal size; these cells are nearly spherical, with only one process. Scattered among these are smaller cells, usually fusiform in shape; this group is called the internal vesicular columns by Clarke; many authors name it Clarke's column.

The posterior cornua are formed of two varieties of structure, the spongy portion near the central gray sub-

stance, the gelatinous substance posterior to the former, and running forward on each side of it, having the form of an irregular crescent. A few large nerve-cells, with many processes, are found scattered through the gelatinous substance, especially along its outer border. The nerve-cells in the spongy portion are rarely of large size. In the posterior cornua are found also some of the smallest cells belonging to the spinal cord. The opposite sides are connected in front of the central canal by nerve-fibers forming the anterior or white commissure; behind the central canal is a commissure formed of gray matter—the posterior or gray commissure.

The *white substance* of the spinal cord is divided into two lateral halves by an anterior and posterior fissure. The anterior fissure is the better marked, and extends about one third through the cord to the white commissure. The posterior fissure is not quite so deep as the anterior, and is less well marked; sometimes, indeed, its position is only indicated by a small blood-vessel in a narrow band of connective tissue.

Each half of the white substance is roughly divided by the anterior and posterior cornua into three columns—the anterior, lateral, and posterior. The anterior and lateral are frequently spoken of together as the antero-lateral column. The posterior column is divided into two portions by a septum of connective tissue, usually containing blood-vessels, situated at a variable distance exterior to the posterior fissure. The column between this septum and the posterior fissure is called the internal posterior column, or more frequently the column of Goll. The nerve-fibers in this column are on an average the smallest in the cord. This division of the posterior column is seen throughout the length of the cord, but the internal portion gradually diminishes in size downward, and in the lumbar region mere traces of it are found. The portion on each side between the lateral septum and the posterior cornu is called the external posterior column, or more frequently

the external radical column or posterior root-zone, or column of Burdach. The anterior column is also divided into two portions, but with less definiteness than the posterior column. There is a narrow band along the edge of the anterior fissure, which, physiologically and pathologically, is distinct from the rest of the anterior column. This is called the direct pyramidal column, or column of Türck; this column can be traced upward to the crus cerebri of the same side without decussating. In the lateral column there is also a distinct group of fibers called the lateral pyramidal tract, which occupies about the center of the lateral column, in the cervical region, having the cerebellar fibers along its outside. In the lower dorsal and lumbar regions these cerebellar fibers gradually diminish in thickness until they disappear; hence the pyramidal fibers come to the surface. The white substance around the extremities of the anterior cornua may be called the anterior root-zones.

The pyramidal fibers in both the anterior columns and the lateral columns can be traced from the brain. Arising from the motor centers in the brain, they pass through the anterior two thirds of the posterior segment of the internal capsule, the middle of the crusta (basis) of the crus cerebri, through the pyramidal region of the pons and medulla to the anterior pyramids. In the anterior pyramids the fibers intended for the lateral columns decussate and pass down the opposite half of the cord; the direct fibers do not decussate, but pass down the anterior columns on the same side, next the anterior fissure.

It may be mentioned here that the divisions above described are differentiated physiologically, pathologically, and in some cases anatomically; and also by the fact that the nerve-fibers in the different portions acquire a medullary sheath at different periods of development, as has been demonstrated by Flechsig, Ross, Charcot, Parrot, and others.

The anterior nerve-roots enter the cord opposite the anterior cornua. Before entering the cord, the roots split up into small bundles of nerve-fibers, which are distributed laterally over a space corresponding in extent with the width of the anterior cornua. These fibers pass directly through the white substance into the gray matter, or take a longitudinal direction for a short distance, and then pass into the anterior cornua. The posterior nerve-roots enter the cord near together on a vertical line; some of the fibers pass directly into the posterior cornua, but most of them pass into the external radical column, and enter the posterior cornua at different levels. Some fibers pass directly into the vesicular columns, and some of these pass forward to one of the groups of anterior cells. Some of the fibers of the white columns run long distances before entering the gray substance; such are the fibers in the anterior and lateral pyramidal columns, the columns of Goll, and the cerebellar fibers. Other fibers run only a short distance, serving as commissural fibers for the gray substance at different levels.

PHYSIOLOGY OF THE SPINAL CORD.

The spinal cord is not a simple organ, as must be realized from the brief account of its anatomy; the functions it is intended to fulfill are many, and its physiology, consequently, is complicated. We are justified, then, in considering the cord, not as one simple organ, but as a series of organs having somewhat different functions. The cord may be divided, theoretically, into as many sections as there are pairs of nerves arising therefrom, and each section be regarded as a distinct unit connected with its fellows by commissural fibers; or it may be viewed as several different nerve-entities arranged side by side longitudinally, each individual connected with the others by transverse commissures. Sometimes one view will be most convenient for understanding phenomena, and sometimes the other.

The groups of nerve-cells in the gray substance act as centers of nervous influence, more or less independent. Those in the anterior cornua are connected with the nerve-fibers of the anterior roots, and serve as media by which motor impulses are communicated through the nerves to the muscles, whether those impulses come from the brain or from the sensory nerves of the posterior roots—that is, whether they are volitional or reflex. Attempts have been made to assign different nerve-groups to certain muscles; while this can be roughly approximated, as yet much that has been written in regard to such a division of function is theoretical.

Among these cells are to be found also centers of nutrition, trophic centers for both nerves and muscles. It is not yet settled whether distinct nerve-cells have this function, or whether it is exercised by the motor cells; as Ferrier expresses the thought, "We have as units of external function certain nerve-centers, centrifugal nerves, and peripheral organs, muscular, glandular, and their adjuncts. In union they exhibit certain vital properties and reactions which we call normal. . . . But dissolve the unity, and the tissues are left to their own powers of nutrition," and various forms of degeneration are seen.

The posterior cornua are known to belong to the sensory portion of the cord. Their cells are in some way concerned in transmitting sensory impressions to the brain, or in transferring them into reflex phenomena; perhaps they intercept some impressions and utilizing them for stimulating vital processes, do not permit such impressions to reach the brain unless they are unusually strong. The sense of pain is conveyed specially by the posterior gray substance, and a very small section of this is sufficient to transmit pain; when the posterior columns are diseased, probably some of the ordinary sensations pass through the gray substance by unusual paths; hence, perhaps, the delay sometimes noticed in conduction of sensation.

Sensations, whether of pain or the ordinary sensations, after entering the cord by the posterior nerve-roots, soon pass to the opposite side and then ascend to the brain.

The white columns are commissural; some connect the brain with different groups of cells, others connect these groups of cells with one another.

The pyramidal tracts connect the motor areas of the brain with the groups of cells in the anterior cornua—the lateral pyramidal tracts decussating in the anterior pyramids, the direct passing down without decussation. The fibers which govern the respiratory muscles seem to pass down in the lateral columns without decussation.

The anterior root-zones are commissural, and are concerned specially in reflex actions, and, perhaps, in co-ordinating the action of nerve-cells at different levels.

The posterior columns are sensory. The external radical columns, columns of Burdach, are probably chiefly commissural, and are employed for co-ordination of the sensory impressions and translation of these into reflex or semi-reflex acts. Many of the fibers of the posterior nerve-roots pass upward or downward in these before they enter the posterior cornua.

The fibers of the columns of Goll pass upward toward the brain; their mode of termination and function are not known.

The posterior columns in one or both of its divisions transmit the ordinary sensations—touch, temperature, pressure, etc.—but not the sensation of pain.

The direct cerebellar tract is composed of ascending fibers, and is said to be connected with Clarke's vesicular column at the root of the posterior cornua. Its function is not known.

GENERAL SYMPTOMATOLOGY.

It will be convenient to mention the groups of symptoms which indicate lesion of certain regions of the spinal cord.

Total *paralysis of motion*, sensation not being affected, points to lesion of the antero-lateral columns and anterior cornua. If the paralysis is unilateral, it will be on the same side with the lesion.

If, in disease of the cord, there is simply paralysis, without marked wasting or change of electrical reaction in the muscles, provided time enough has elapsed since its origin, neither the anterior cornua nor the nerve-roots at the level, whence arise the nerves supplying the paralyzed muscles, can be affected.

If besides paralysis there is spasm, contracture, and increased reflex action in the affected limbs, without pain, the lateral columns are affected. The anterior columns may be diseased too, but the symptoms would not necessarily indicate it. The spasmodic phenomena and increase of reflex action follow, whether the lesion of the lateral columns is primary or secondary.

If, in disease of the spinal cord, with the paralysis, there is also wasting of the paralyzed muscles and loss or great diminution of reaction to the faradic current, still more if there is increased reaction to the galvanic current, with reversal of qualitative reaction (degenerative reaction), the lesion is either in the anterior nerve-roots or in the anterior cornu. It is rare to have the anterior nerve-roots affected as they pass through the anterior columns, unless the lesion is traumatic in its origin. Unless the anterior cornu is diseased over a considerable length of the cord, the paralysis is local, and the muscles supplied by nerves arising below the seat of the lesion are healthy. When only a few muscles are affected, especially if they form a group which physiologically act together, the disease may be limited to a very small area.

Ferrier ("The Localization of Atrophic Paralyses," "Brain," vol. iv, 1881–'82, p. 226) gives the muscles supplied by the different nerves of the brachial and lumbar plexuses. Some of the muscles mentioned are supplied by fibers from more than one nerve; only that nerve

giving the largest proportion of fibers is mentioned. The enumeration can be considered as only approximately correct; yet it will serve as an aid in locating a lesion in the spinal cord, and perhaps will prove of most value where there is atrophy of muscles. As the skin over muscles is supplied by nerve-fibers from nearly the same region as the muscles, the distribution of anæsthesia may serve roughly as a guide to diagnosis of the level of a lesion, though in such a case much less likely to be so nearly correct as when muscular symptoms are the guide.*

The first dorsal: The intrinsic muscles of the hand, viz., muscles of the thenar and hypothenar eminences and interossei.

Eighth cervical: Long flexors, ulnar flexors of wrist, intrinsic muscles of the hand, extensors of wrist and phalanges, long head of triceps (pectoralis major?).

Seventh cervical: Teres major, latissimus dorsi, subscapularis, pectoralis major, flexors of wrist and fingers (median), triceps.

Sixth cervical: Latissimus dorsi, pectoralis major, serratus magnus, pronators (flexor of wrist?), triceps.

Fifth cervical: Deltoid (clavicular portion), biceps, brachialis anticus, serratus magnus, supinator longus, extensors of wrist and fingers.

Fourth cervical: Deltoid, rhomboid, supra- and infra-spinatus (teres minor), biceps, brachialis anticus, supinator longus, extensors of wrist and fingers, diaphragm.

In the lower extremity:

Second sacral: Intrinsic muscles of the foot, strictly parallel to the first dorsal.

First sacral: Muscles of the calf (plantar flexors), hamstrings, long flexor of big toe, intrinsic muscles of the foot.

* See, also, M. Allen Starr, "Localization of the Functions of Spinal Cord," "Amer. Jour. of Neurolog. and Psychiat.," August and November, 1884, p. 480.

Fifth lumbar: Flexors and extensors of toes, tibial muscles, sural muscles, peroneal muscles, outward rotators of thigh, hamstrings.

Fourth lumbar: Extensors of thigh, extensor cruris, peroneus longus, adductors.

Third lumbar: Ilio-psoas, sartorius, adductors, extensor cruris.

By keeping this general scheme in mind, an idea may be formed, nearly correct, of the level of the lesion, and a record may be made by which to judge whether it is extending or receding.

A study or examination of the various reflexes will also aid in the formation of a diagnosis of locality. (See page 8.)

There may not be paralysis, yet the motor conductors may be seriously affected, so that, while all motions are possible, yet there is clearly a loss of strength, and the movements are very slow; there is a retardation of the motor conduction; Burckhardt thinks this points to disease of the white columns.

Burckhardt has also studied the acceleration of motor conduction. This is not very rare, but is more difficult to recognize. Some cases of exaggerated reflex action may be due to this condition. Burckhardt refers this phenomenon to an affection of the gray substance.

There may be no paralysis, but the motions may be irregular; there is a loss of co-ordinating power, a condition known as ataxia, which Erb defines as "the disturbance of movement, produced by defective co-ordination of movement." He considers that it is of a motor nature. While all motions may be performed with power, the patient can not execute any movement with precision; the hand or foot is carried beyond or falls short of the point it is desired to touch, or it is carried to one side. In attempting to grasp an object, the strength put forth is out of proportion to the end to be gained. When extreme, this ataxia may be shown in all the motions; if the defect is but slight, it

may be necessary to ask the patient to close his eyes before the symptom can be clearly recognized. When the aid of sight is withdrawn, the patient may be unable to touch a certain part of his face, as the chin or end of the nose, with his forefinger, or in walking may be unable to propel his feet properly. Yet not every defect in walking with closed eyes is due to ataxia; weakness may cause a patient to totter and walk irregularly; vertigo may have a similar effect; anæsthesia of the soles of the feet or of the joints may cause a patient to stagger when the eyes are closed.

Erb thus describes the ataxic gait: "It is characterized by irregular hurling movements; the point of the foot is thrown forward and outward with force; the heel is brought down with a stamp, the leg stiff at the knee. The patient's eyes are continually on the ground. The gait is tottering, staggering, or even reeling from side to side; the movements are hasty, spasmodic, quite unequal; in turning about, especially, there is great uncertainty, and danger of falling. In severe cases the patient falls after a few steps." This accurate description applies to rather advanced cases; there may be only a very slight degree of disturbance early in the disease.

This ataxic condition, when dependent upon disease of the spinal cord, is the result of changes in the posterior columns, and more definitely the external radical columns.

Involuntary muscular movements are among the prominent symptoms in certain affections of the spinal cord. These are of a reflex nature, or the result of direct irritation of the motor roots or motor regions of the cord.

When the spinal cord is divided, or when destruction by disease extends across the cord, so that communication with the brain is cut off, reflex movements are exaggerated in all parts of the body below the seat of injury whose reflex nervous arc remains intact. The

reflex phenomena, as mapped out by Gowers, may assist, then, in locating the seat of the disease. (See above, p. 8.)

When there is increased irritability of the gray substance, the reflex motions in the regions supplied by nerves arising therefrom will be exaggerated. The same exaggeration is found in disease of the lateral pyramidal columns, as in secondary degenerations and sclerosis.

The pupillary reactions are reflex: if the cervical region of the cord is destroyed, the pupils may be contracted; if there is irritation, they will be dilated. The normal reactions will not be present.

Reflex action may be much diminished or abolished when any portion of the reflex arc is diseased; whether the sensory nerves or posterior nerve-roots, the gray substance, or the anterior motor regions, or motor roots, are diseased. Reflex action may be delayed under similar conditions under which sensory impressions are delayed.

In cerebral disease the reflex actions may be abolished on the paralyzed side.

Westphal first called attention to the fact that the tendon reflex is lost at an early period in locomotor ataxia; this has been confirmed by others, and it is now generally accepted as true of that disease. It has been found absent when the columns of Goll were not affected, the external radical columns alone being diseased (Westphal). It is also lost when the spinal cord is entirely disorganized; when the sensory or motor roots (or peripheral nerves) are destroyed; when the anterior cornua are diseased, so as to cause muscular atrophy; and in some other less definite conditions.

The tendon reflex may be greatly exaggerated, and may be then readily shown in connection with tendons with which it is not usually noticed, as those of the triceps humeri, of the fingers, of the sterno-mastoid.

This increase of the phenomenon is one of the symp-

toms attending sclerosis of the lateral columns; it may be present also after injuries giving rise to spinal concussion (Edes), and in hysteria; it is sometimes seen during acute febrile diseases, as typhoid fever. As a symptom of disease of the spinal cord, it is most regularly associated with disease of the lateral columns, and we are not yet able to say that, when found in apparently exceptional relations, it is not dependent upon a change thus located.

Ankle clonus is indicative of change in the lateral pyramidal columns. Gowers says that a persistent ankle clonus is always pathological; in this he is probably a little too emphatic, but its presence must be looked upon as strongly in favor of organic changes in the cord.

The spinal cord, by a reflex mechanism, exerts a control over the bladder and rectum; the will also regulates in some measure those viscera. When the contents of the bladder and rectum are sufficient to excite reflex action in their expulsory muscles, the sphincters relax, probably in consequence of an inhibitory action of the spinal centers, and an evacuation follows. The will can restrain for a while this expulsive action, or can excite it before the reflex action would arise normally.

If the spinal cord is destroyed above the lumbar enlargement, this voluntary control is lost, and then the contents of the viscera are expelled at intervals according as the reflex centers may be aroused to action by the irritation excited by the contents of the viscera. The patient then has his evacuations involuntarily, and without knowing that they occur.

If the sensory tract alone is injured, the evacuations may occur without his knowledge, but he will have power to voluntarily evacuate the viscera. If the motor tract is injured, the discharges will occur without the patient's control, but he will be conscious of the desire to evacuate the viscera, and will know when

the evacuation is accomplished. When the voluntary control is weakened, or partially lost, while the sensory tract is unimpaired, the patient is obliged to respond quickly to the calls of nature, or, the restraining influence of the will being slight, involuntary evacuation follows.

As the centers of reflex action for the sphincters and the detrusor muscles are not identical, one may be affected independently of the other; then there will be incontinence when the sphincters are paralyzed, retention when the detrusors are paralyzed. In the latter case, the sphincter acting, the urine accumulates until it may, by mere mechanical pressure, overflow, and so there may seem to be incontinence when there is really retention.

Spasm may affect the sphincters or the detrusors, and corresponding disturbances will follow.

Perversion of the sexual functions occur in many cases of spinal disease, much more marked in men than in women. There may be great increase of sexual desire, with power to gratify it; or the desire may be present without the power, or with greatly diminished power; or there may be frequent nocturnal or diurnal emissions, or spermatorrhœa. All sexual appetite may be lost, and there may be complete impotency. Priapism, complete or partial, may continue for a long period. Among women a similar disturbance of sexual desire may occur, but these symptoms have been less fully studied than among men. We can not at present draw any positive conclusions by means of the above variations from the normal condition.

Vaso-motor and nutritive changes are not uncommon in different forms of spinal-cord lesions. The simplest change is disturbance of capillary circulation; the affected parts are more or less cyanotic, or they may be unnaturally pale; in either case the temperature is below normal; sometimes there is great increase of heat, with less apparent disturbance of circulation. In one

case there is irritation of the vaso-motor centers, in the other case paralysis. It is often noticeable that paralyzed limbs are slightly œdematous, and sometimes the consequent swelling is very great.

The skin may undergo changes similar to those found in neuritis; there may be a scaly condition, due to excessive multiplication of the epidermic cells; the hair and nails may suffer in nutrition; herpetic eruptions, pustules, and urticaria may be noticed. Bed-sores, chronic or acute, are the most troublesome trophic changes in some cases, and may gradually wear out the patient. The bones, especially their articular surfaces, undergo changes of structure and form, are worn away, or become brittle and easily break.

The muscles undergo atrophy, their fibers becoming reduced to rows of fat drops or granules, and finally, these being absorbed, only the sheaths are left; sometimes a deposit of fat between the muscular fibers obscures the wasting, and the affected limbs retain their usual proportions. The electrical reaction of degeneration will show whether this atrophy has occurred and enable one to form an opinion as to how far it has advanced. If only some fibers are degenerated, these reactions may be obscured and less readily obtained, the healthy fibers giving the normal reactions. This atrophy shows that there is lesion of the motor nerves or of the anterior cornu in the cord.

The general nutrition of the body or limbs may be altered in disease of the spinal cord; there may be great emaciation, or there may be an increased deposit of fat, subcutaneous, as well as in the deeper structures. When the nerve-cells of the anterior cornu are diseased in infancy, there is not only atrophy of the muscles, but, if the change is extensive, the paralyzed limb is retarded in its subsequent growth.

The anterior part of the central gray substance is supposed to be the trophic center for the bones; the posterior part for the skin, hair, and nails; the ante-

rior cornua are the trophic centers for motor nerves and muscles.

Sensation is entirely lost only when the whole of the posterior portion of the cord, including the gray substance, is destroyed. If even a small portion of the gray substance remains, sensation is not entirely lost. It is probable also that certain parts of the lateral columns convey sensation, though the mode of distribution of sensory fibers in these columns is not known. When both sides are destroyed, a narrow band of hyperæsthesia may be found above the level of the anæsthesia.

If only one side of the cord is destroyed, there will be hyperæsthesia below the seat of injury on the same side with it, and anæsthesia on the opposite side; a narrow zone of diminished sensibility on both sides of the body will be found at the level of the injury, and above this for a short distance there may be hyperæsthesia on the same side with the injury.

When sensation is not entirely destroyed, the different kinds of sensation, as touch, temperature, pressure, pain, etc., may be affected in unequal measure.

When there is not destruction of the cord, there may be great hyperæsthesia, and the increase of sensitiveness may be so great that even a slight touch causes great suffering. This is found when there is inflammation of the meninges, or when the posterior nerve-roots are irritated, as by compression, and rarely in inflammation of the cord itself, though local hyperæsthesia is common in locomotor ataxia, especially after an attack of lancinating pain.

A sensation of a band tied around the body—a girdle sensation or pain—is frequently found in myelitis, and where there is compression of the cord. It is difficult to define its nature; it is sometimes painful, sometimes simply a slight sense of constriction. It is seated at the level of distribution of the nerves arising from the upper limit of the disease; sometimes the girdle seems to surround one or both legs instead of the body.

When the posterior nerve-roots are exposed to irritation, as from pressure or inflammatory changes, pain of different kinds will be felt at the peripheral ends of those nerves; if the nerves are suddenly compressed or bruised, a burning sensation, perhaps very painful, will be felt. The sensation referred to the periphery in disease of the spinal cord is much less likely to be pain; it rather takes the form of tingling, numbness, formication, or that peculiar sensation known as being asleep.

Backache is a very common complaint with patients; it is more common in functional than in organic diseases. Tenderness on pressure over the vertebræ is rare in organic affections; it is common in connection with certain functional disturbances.

CHAPTER X.

SPINAL MENINGITIS.

JOFFROY, A., De la pachyméningite cervicale hypertrophique. Paris, 1873.—SPENCER, W. H., Case of Idiopathic Inflammation of the Spinal Dura Mater. *Lancet*, June 14, 1879, p. 836.—LEMOINE, G., and LANNOIS, N., Périméningite spinale aiguë. *Revue de Méd.*, No. 6, 1882.—TOOTH, Dorsal pachymeningitis. *Brain*, 1884.

There are two subdivisions of spinal meningitis—one affecting the dura mater, pachymeningitis; the other the pia mater, leptomeningitis.

External pachymeningitis, inflammation of the external surface of the dura mater, is caused by changes in the adjoining parts—caries, abscess, cancer, tumor, aneurisms penetrating from without, etc. The symptoms will be so united with those caused by the primary disease that it is unnecessary to describe them.

PACHYMENINGITIS INTERNA.

Internal pachymeningitis may occur independently of other lesions. Generally, not only the internal surface of the dura mater is affected, but its whole thickness may be the seat of inflammatory hypertrophy; the pia mater may also be somewhat thickened and inflamed secondarily; it can usually be distinguished from the dura mater.

Owing to the thickening of the membranes, the cord is compressed, and undergoes inflammatory changes; sometimes cavities are formed.

The nerve-roots suffer from compression and secondary inflammation as they pass through the dura mater.

The disease is confined almost exclusively to the cervical region.

SYMPTOMS.—During the first stage of the disease the prominent symptom is pain in the posterior part of the neck and the occipital region; following the direction of the peripheral nerves, it extends frequently into the arms; it is aggravated by movements of the vertebræ, sometimes is increased by firm pressure over the spine, and at times is extremely severe. Sometimes before the pain ceases, more frequently after a period of comparative freedom from distress, paralytic symptoms make their appearance, first weakness, which gradually increases to complete paralysis. With the paralysis there is atrophy of the muscles. The distribution of the atrophy is somewhat variable; the muscles of the hand, the interossei, the lumbricales, and the muscles of the thenar and hypothenar eminences are generally greatly atrophied; in the forearm the flexors and extensors of the fingers, the flexors and pronators of the hand, are chiefly affected; the muscles of the arm generally escape, while the deltoid and the supra- and infra-spinatus suffer. Owing to the atrophy of some muscles, and the fact that others are unaffected, the hand acquires an unnatural position: it is held in a position of extreme extension, with the fingers partially flexed, the thumb extended and adducted. In some cases this position may be overcome by passive motion; in others it is noticed only when the forearm is in supination.

If the disease affects the upper part of the cervical enlargement, the position of the hand is different. Ross thus describes the position: The arm is held close to the side, the forearm is extended on the arm and strongly pronated, the hand is flexed on the forearm, the fingers are in a line with or only slightly extended on the metacarpal bones, and the phalanges are extended upon one another, while the thumb is flexed into the palm. The muscles supplied by the musculo-

spiral nerve are more affected than those supplied by the ulnar and median.

The disease has rarely been observed in the lower part of the spine.

The paralyzed muscles undergo atrophy, and the electrical reaction is changed; there is found the reaction of degeneration, or entire loss of electrical reaction.

Trophic changes in the skin, vesicular, bullous eruptions, dry and scaly condition of the skin, and a glossy skin, are occasional phenomena; sometimes bed-sores form. The temperature is frequently lower than normal. Slight convulsive shocks, and the phenomena attending lesion of the lateral columns, are sometimes met.

DIAGNOSIS.—The first stage, where there is only pain without impairment of motion, is difficult of diagnosis. The pains may be referred to a rheumatic affection, to spinal irritation or hysteria, or they may give rise to the suspicion that caries of the vertebræ is present. The pain due to pachymeningitis is said to be characterized by an increase during movement of the vertebræ, always deeply seated in the back part of the neck, on the median line; frequency of the attacks of pain, and their short duration.

When muscular atrophy sets in, the diagnosis from progressive muscular atrophy may be made from the history of the preceding attacks of pain, from the fact that the muscles are affected less regularly; in muscular atrophy the hand will assume a more or less flexed position when in the stage of contracture, but in pachymeningitis the hand is extended and supinated, or the hand and arm take the position described by Ross.

TREATMENT.—During the first stage the most pressing indication is to relieve pain, for which sedatives and anodynes may be used; hot iron applied to the neck and upper part of the dorsal region may give re-

lief to the pain, and may also act favorably upon the progress of the disease. The galvanic current along the spine has been recommended, the positive pole above, the negative pole below, the cervical region. Paralysis and atrophy of the muscles can be treated locally by the faradic or galvanic current.

Internally, iodide of potassium may be given. In judging of the value of treatment, it should be remembered that naturally the disease has periods of remission.

INFLAMMATION OF THE PIA MATER.

PATHOLOGICAL ANATOMY.—The term spinal meningitis is commonly used to designate inflammation of the pia mater, leptomeningitis. The pia mater is chiefly affected; it is found congested, thickened, œdematous; upon its surface and within its meshes there is more or less pus. The arachnoid is almost always implicated in the inflammatory process; the dura mater is also sometimes involved. The amount of pus exuded varies greatly; it may be so little as merely to give a yellowish tinge to the œdematous membrane, or the surface may be covered with a thick, creamy layer. The inflammation may extend over a very small surface, or may affect the whole cord. In about one third of the cases there is coincident cerebral meningitis.

After the exudation has been absorbed, there may be left a thickening of the pia mater from the organization of the inflammatory products; the membranes may become adherent to each other, though this is rare.

It can be easily understood that such serious disturbance of the pia mater, from which the spinal cord derives its blood-supply, must necessarily involve the spinal cord, and we almost always find some degree of myelitis associated with the meningitis.

ÆTIOLOGY.—The principal causes are exposure to cold and dampness, over-exertion of any kind, insolation, jars, concussions, falls upon the back, and inflam-

mation of neighboring parts. Occasionally, in tubercular meningitis, tubercles are found in the spinal pia mater.

SYMPTOMS.—Sometimes a short prodromal period of general discomfort with fugitive pains precedes the attack of prominent symptoms, but generally the disease commences suddenly, with pain in the back and pain radiating into the limbs; there is often a chill and the temperature rises; there may be headache and even vomiting, though this is rare, unless the disease is near the upper part of the spine or the cerebral membranes are also affected; the surface of the body becomes extremely sensitive to the touch, the muscles of the extremities are contracted, the body may be in position of opisthotonus, or there may be clonic spasms instead of tonic contraction of the limbs.

The pain, which appears early in the back, is generally very severe, is increased by the slightest motion, whether active or passive; pressure upon the vertebræ may not increase it, but percussion almost always does; extremes of temperature, either hot or cold, produce pain when applied over the region affected. The pain may radiate around the trunk in the form of a girdle, though this is perhaps less frequent than in myelitis; it may also radiate with extreme severity into the limbs. The last phenomena are due to irritation of the nerve-roots. The cutaneous hyperæsthesia is probably due to an irritation of the nerve-roots, and may be classified with other painful manifestations; it is sometimes so extreme that even the weight of the bedclothes causes torture; not only the limbs but the trunk may be thus affected, and the muscles and bones sometimes show an extreme degree of sensitiveness.

The muscular stiffness and rigidity is probably at first due to an involuntary tension of the muscles in order to avoid motion because of the extreme pain produced thereby; later the contracture is probably due to direct irritation of the anterior nerve-roots. Some-

times, especially at the beginning of the disease, clonic spasms add to the patient's suffering.

There is generally constipation and retention of urine, sometimes with a frequent desire to pass it.

Respiration is frequently interfered with, especially when the disease is seated in the cervical region, and death sometimes arises from this disturbance preceded by Cheyne-Stokes's respiration.

When the contraction diminishes sufficiently, it is found that there is partial or total paralysis, and that sensation is more or less affected; sometimes with paralysis there remains contracture of the limbs, either in extension or flexion. The electrical reaction of the muscles may be lost, or undergo the modification of degeneration.

Death frequently occurs after only a few days, or the patient may die at a much later period, apparently from exhaustion. Sometimes recovery is complete and perfect, but more frequently there remain partial paralyses and atrophies. A chronic leptomeningitis lasting for months or years is said sometimes to result from the acute disease.

DIAGNOSIS.—Jaccoud says: "The only two diseases of the spinal cord which have a febrile beginning are acute meningitis and acute myelitis; now, as a rule, these two inflammations exist together, and the difference in diagnosis is only a matter of refinement or a question of relative preponderance." The principal diagnostic symptoms of spinal meningitis are the pain in the back and limbs, the hyperæsthesia, the muscular spasm, and contracture. The opisthotonus might give rise to the suspicion that the disease is tetanus; but in that there is much less fever, less pain, except during the spasm, and little or no cutaneous hyperæsthesia; at the beginning there is trismus, the other spasms are more violent, and the reflex irritability is excessive. If there is recovery, it is, as a rule, more complete in tetanus than in spinal meningitis.

PROGNOSIS.—The prognosis is said by Erb to be "influenced for the worse by the following circumstances: A very youthful or very advanced age; bad constitution, anæmia, the previous occurrence of severe disease, etc.; by the height to which the disease ascends in the spine toward the brain; by early symptoms of paralysis, signs of general loss of strength, high fever, continually rising temperature, and increasing frequency of pulse; great difficulty in breathing, dysphagia, severe cerebral symptoms, etc."

The disease is at best serious, and even after partial recovery relapses may occur. The paralysis and atrophy that remain may disable the patient for the rest of his life.

TREATMENT.—The sensitiveness of the skin may be such as to interfere with the use of cups, but, if possible, dry or wet cups should be applied, or leeches may be used. Ice-bags should be kept constantly on the spine. After the first acute stage has passed, blisters or other counter-irritation may be applied over the spinal column. Most European authors recommend that mercury be used by inunction or internally. Ergot may be given in large doses frequently repeated. Iodide of potassium can be used after the earlier stages of the disease. The pain can be controlled by opiates, which should be given in large doses.

It is scarcely necessary to mention that quietness, rest in bed, and the ordinary hygienic measures be observed. Decubitus on the side or prone is the best position in bed.

The paralyses and contractures which remain after recovery may be treated by electricity, passive motion, friction, and baths.

CHAPTER XI.

CHANGES IN BLOOD-SUPPLY.

MAYER, SIGMUND, Zur Lehre von der Anämie des Rückenmarks. *Zeitschr. f. Heilk.*, iv, 1883, p. 26.—GULL, Paraplegia from Obstruction of the Abdominal Aorta. *Guy's Hosp. Reports*, 1858, p. 311.

SPINAL HYPERÆMIA.

Congestion, and its opposite, anæmia, of the spinal cord and its membranes have been too frequently mentioned as causes of symptoms which evidently arise from more serious lesions. Clinically, and even pathologically, it is difficult to draw the line of separation between congestion and inflammation. It is almost impossible, also, to separate these lesions of the meninges from similar lesions of the cord itself; indeed, the spinal cord is always more or less implicated when the pia mater is diseased. While it is probably true that many slight disturbances of health are ascribed to congestion when there is really inflammation, yet it is convenient to speak of congestion, and describe it as an independent disease.

ÆTIOLOGY.—One of the most frequent causes is cold, acting upon the surface of the body when the patient is heated; if dampness is combined with the cold, as when the patient's clothing is wet by a sudden shower, or when the patient, overheated, takes a cold bath, the influence of the cold is much increased.

Suppression of the menses, or hæmorrhoidal bleeding, or other habitual discharges, may act as causes of congestion.

Excessive bodily exertion, especially walking and standing, violent sexual excitement, or excess of coitus, may have the same effect.

Much of the backache, and some of the pain in the limbs found at the commencement of febrile diseases, are probably caused by spinal hyperæmia.

SYMPTOMS.—As already mentioned, it is impossible to separate the symptoms caused by congestion of spinal meninges from those produced by congestion of the spinal cord itself. The following description may serve for both conditions:

There is a heavy, dull pain in the small of the back, or higher, sometimes radiating into the legs with subjective sensations of numbness and pricking; sometimes a girdle sensation is felt; a weakness or partial paralysis of the legs is generally present; rarely slight spasms or twitches of muscles. These symptoms appear rather suddenly, and may be aggravated by lying on the back. Unless inflammatory changes are set up, they are not accompanied with febrile action, and are usually of short duration, not lasting more than a few days or, in rare cases, weeks.

DIAGNOSIS.—The diagnosis is to be made from the slightness of the symptoms and their short duration, and the absence of fever, rather than from any peculiarity of the symptoms themselves.

TREATMENT.—Active treatment, such as bleeding and purgatives, have been recommended; but, instead of general bleeding, wet cups or leeches, on both sides of the spine, are better: dry cups may be used with advantage in the same place. The actual cautery may be tried with reasonable expectation of benefit. Purgatives may be of use, but, if pushed far, would be of disadvantage, from the necessity of disturbing the patient too often. Ice-bags to the spine may be used to advantage. Internally, belladonna and ergot have been recommended.

It is better for the patient to be kept quiet, confine-

ment to the bed being preferred; the causes liable to produce congestion should be avoided, and by some a position upon the back is forbidden.

SPINAL ANÆMIA.

In animals, experiment has shown that the symptoms of anæmia may vary according as it is suddenly produced, or is brought on gradually. If all the blood is shut off at once from the cord, convulsions occur; if the cord is gradually deprived of blood, there is only loss of function, without convulsion. In man the sudden stoppage never occurs, owing to the free anastomoses. Sometimes, in aneurism of the aorta, anæmia of the cord is produced by occlusion of blood-vessels, and serious disturbance of function may result therefrom. In general anæmia and chlorosis, in cardiac disease, there may be a diminished supply of blood in the cord.

The spinal *symptoms* are not sufficiently well defined to form a positive diagnosis from them alone. There is simply disturbed function, numbness, motor weakness, and tremor, without fever. There is a condition, usually called spinal irritation, which has been referred to anæmia of the spinal cord, but without sufficient reason. The diagnosis must be made chiefly from symptoms other than those due to the spinal disturbance.

The *treatment* should be directed to the condition causing the anæmia; the patient should be kept in bed on his back if the symptoms are at all serious; hot-water bottles may be applied to the spine; strychnia has been recommended.

CHAPTER XII.

SPINAL HÆMORRHAGE.

Fox, E. L., Clinical Lecture on Spinal Hæmorrhage. *Med. Times and Gaz.*, Aug. 26, 1876, p. 219.—Goltdammer, E., Ein Beitrag zur Lehre von der Spinal-Apoplexie. *Virch. Arch.*, lxvi, p. 1.—MacMunn, C. A., Notes on a Case of Spinal Apoplexy. *Dublin Jour. of Med. Sci.*, March 1, 1880, p. 182.—Eichorst, H., Beitrag zur Lehre von der Apoplexie in der Rückenmarkssubstanz. *Charité-Annalen*, 1876, p. 192.—Hayem, G., Des hémorrhagies intrarachidiennes. Paris, 1872.

MENINGEAL HÆMORRHAGE.

Ætiology.—Spinal meningeal hæmorrhage, hæmatorrhachis, is rather a rare affection. It occurs as the result of injuries and falls, or in consequence of overtaxing the strength; secondarily as following aneurism, or during tetanus, epilepsy, or some acute diseases, yellow, typhoid, or pernicious fever.

Pathological Anatomy.—The blood may be poured outside the dura mater; it may fill the whole of the vertebral canal, but is more frequently spread over the posterior surface of the membrane; again, it may be limited to a comparatively small extent. The cervical region is rather more frequently the seat of the hæmorrhage. The condition of the blood, as found at the autopsy, depends upon the length of time that has elapsed between the attack and death.

Intra-meningeal hæmorrhage, where the blood is effused between the dura mater and arachnoid, is less frequent than the preceding. Hayem found thirty-eight cases of extra meningeal hæmorrhage, and only eleven of this variety.

The hæmorrhage under the arachnoid and into the pia mater is still more rare, Hayem finding only eight cases.

Many times the blood found in these places is poured out only during the last hours of life, and has no effect upon the primary disease; this is especially true where the hæmorrhage is merely slight or punctiform. Where there is considerable blood, however, the spinal cord may be much compressed.

Symptoms.—Many secondary hæmorrhages give rise to no special symptoms, either because they are very slight, or because the primary disease masks the special symptoms which they would cause.

The symptoms usually appear suddenly; there is first severe pain, followed almost immediately by paralysis. Occasionally the onset is more gradual. There are two classes of symptoms which must be recognized: those due to pressure upon the cord, and changes in its structure; those due to irritation of the membrane and nerve-roots by the foreign body, the clot.

The symptoms due to pressure upon the cord are primarily more or less complete paraplegia, affecting chiefly motion, but also giving rise to sensations of numbness; after the commencement of the attack there may be pain in the back, or it may be absent. Reflex action may be exaggerated. The pressure may give rise to secondary changes in the cord, myelitis may follow; the paralysis becomes more complete and permanent; sensation may be more seriously implicated, contractions may appear, and, as the myelitis advances, may disappear; the muscles may undergo atrophy.

The symptoms due to irritation by blood-clot and pressure upon the nerve-roots are so united that it is scarcely worth while to separate them. These are pains radiating in the course of the nerves and others referred to the periphery, tingling and pricking sensations, more or less anæsthesia, with possibly tenderness to touch of the parts to which the nerves are distributed; spas-

modic contractions, generally clonic, sometimes tonic; subsequently there may be atrophy and diminished electric excitability. Disturbed vaso-motor action may be found either below or at the level of the hæmorrhage.

The patient may entirely recover, but more frequently some paralysis and atrophy remain permanently, accompanied, perhaps, with contracture. The electrical reaction undergoes the usual change in atrophied muscles.

The membranes are not very prone to secondary inflammation; fever is rarely present; the pulse may be weak and slow.

The symptoms vary somewhat according to the locality of the hæmorrhage. When the upper part of the cord is affected, the pain and contraction and reflex phenomena will be most marked in the upper extremities; oculo-pupillary symptoms will be observed, and there may be disturbance of respiration. When the seat of the hæmorrhage is lower, the above symptoms will be absent, and the sensory and motor phenomena will be most marked in the back and legs; the bladder and rectum may be affected, priapism may give annoyance, or erection may be less frequent, and sexual power diminished.

DIAGNOSIS.—The chief diagnostic symptoms are the suddenness of the attack, the signs of meningeal irritation, the absence of cerebral symptoms, and the course of the disease; sometimes also the cause will aid to a diagnosis. It may not always be easy to determine at first whether the vertebræ have been fractured or the cord itself injured by the accident which has given rise to the symptoms. Extreme motor paralysis, especially if reflex action is diminished, at the commencement of the disease, and serious implication of the sphincters, would lead to an inference that the cord itself is injured.

A careful study of the symptoms would probably

be sufficient to prevent an error of diagnosis in regard to other affections of the cord and its membranes.

TREATMENT.—The most important indication which should be strongly insisted upon is absolute rest. Erb says upon the side or face; but the position is of less importance than the rest. Leeches or wet cups should be applied along the back. Strong purgation is recommended, but has the disadvantage that the patient must be disturbed too much. As in other cases of hæmorrhage, large doses of ergot may be given, if the case is one of those arising spontaneously. Pain may be relieved by opiates and anodynes. Later, iodide of potassium may be prescribed, and resulting paralyses can be treated by electricity, baths, passive motion, and massage.

HÆMORRHAGE INTO THE SPINAL CORD.—HÆMATOMYELITIS.

ÆTIOLOGY.—Hæmorrhage into the spinal cord is about four times more frequent in men than in women; it occurs chiefly in early adult life—from twenty to thirty-five. It may arise in the course of inflammatory changes in the spinal cord as a secondary complication, or be caused by influences which favor the active flow of blood to the cord; a fall, a strain in lifting heavy weights, or other excessive bodily exertion, may be a cause. These are more likely to prove efficient if the blood-vessels of the cord are diseased.

PATHOLOGICAL ANATOMY.—Of course, the primary change in the cord is its destruction and the disassociation of its fibers by the effused blood. The clot undergoes changes similar to those which follow cerebral hæmorrhage. The cord undergoes inflammatory changes and softening. It may sometimes be difficult to determine whether the softening is caused by the hæmorrhage or preceded it. Hæmorrhage is most frequent in the gray substance. The muscles and nerves undergo secondary changes, such as are found when the spinal nerve-centers are diseased.

The meninges are almost always congested, but the inflammatory changes in them are not very marked.

When the patient lives long enough, secondary degeneration, ascending and descending, will be found in the cord.

SYMPTOMS.—The symptoms due to hæmorrhage into the spinal cord may be preceded by obscure symptoms of discomfort due to disturbances of circulation or nutrition, which precede the rupture of the vessel, and perhaps depend upon the changes in the cord which give rise to the hæmorrhage. These changes of nutrition may be such as are found in myelitis, yet the symptoms caused thereby may not attract special attention, or may be the result of an acute disease, as typhoid fever.

When a blood-vessel ruptures, there may be intense pain in the back, continuing for a variable length of time, followed by paralysis of motion and sensation in the parts below. The occurrence of the hæmorrhage is not always the cause of such tumultuous symptoms; it may occur during sleep, or the symptoms may be developed gradually during a period of several hours or, in rare instances, some days. In the latter case it is more probable that a myelitis has preceded the hæmorrhage.

However it may arise, the chief symptoms are finally very similar. As the seat of the effusion is generally the central gray matter, sensation is more or less profoundly affected, and it may be entirely abolished; motion is restricted, and generally lost in the parts below the lesion. At the beginning there may be a certain amount of tetanic rigidity or spasmodic twitching, but this is of short duration, and the limbs are soon relaxed in paralysis. If the lesion is not so low as to implicate the lumbar nerves, the reflex irritability is increased, as in other cases where the lower part of the cord is severed from communication with the brain, though immediately after the shock of the hæmorrhage the reflex

functions may be temporarily suspended. Consciousness and intelligence are not affected.

At the very onset, if there has been no previous elevation of temperature due to other disease, there is no fever; soon inflammatory changes commence around the clot, and then the temperature may rise; as yet few observations of these changes have been made. The temperature of the paralyzed limbs was noticed by Levier to be 0·2° to 1·9° C. higher than the arms, the thermometer being in the fold formed by bending the knee and in the axilla.

As the secondary changes extend, the symptoms become more decided; if there was only a partial loss of sensation, the anæsthesia becomes complete; there is entire loss of motion instead of partial paralysis. The muscles which arise from the portion of the cord destroyed undergo atrophy and show the reaction of degeneration. As secondary degeneration extends below the seat of the lesion, the symptoms due to affection of the lateral columns appear.

The bladder and rectum are paralyzed, the urine may be very quickly changed in character, may contain blood, may be intensely acid, or may soon become alkaline. Some of these changes in the urine are dependent upon disturbed innervation of the kidneys and not upon cystitis, which may later cause much trouble.

Bed-sores sometimes form with amazing rapidity, and become enormous in size.

The symptoms will vary somewhat, in different cases, according to the height at which the hæmorrhage occurs and the amount of blood poured out. In view of the physiology of the cord, it will not be difficult to locate approximately the upper limit of the lesion and, somewhat roughly, its lower limit.

Diagnosis.—Hæmorrhage into the substance of the cord can be distinguished from meningeal hæmorrhage by the more complete and suddenly occurring paralysis of both motion and sensation, by the absence of

signs of great irritation, and by the rapid appearance of bed-sores. The diagnosis may be easier in cases where there has been a preceding disease of the spinal cord.

In rare cases the hæmorrhage may be confined to one side of the spinal cord; then the paralysis of motion will be hemiplegic. The fact that there is no loss of consciousness, and that sensation is affected on the side opposite the motor disturbance, will prevent such a lesion from being mistaken for cerebral hæmorrhage.

From myelitis arising spontaneously the diagnosis must be made by considering the causes, the mode of onset, and the progress of the symptoms; a careful consideration of these points will probably prevent an error of diagnosis, unless the myelitis is developed with unusual rapidity. MacMunn mentions intensely acid urine as peculiar to hæmatomyelia, distinguishing it from myelitis.

From acute anterior poliomyelitis the diagnosis may be made by the fact that in this there is no disturbance of sensation, that the bladder and rectum are not paralyzed, bed-sores do not form, and the fever, if any, is at the beginning, whereas in hæmatomyelia the fever appears later, unless the hæmorrhage is secondary. In the former also there is a tendency for some muscles to regain their function; in the latter the paralysis tends to increase.

Prognosis.—If a large amount of blood is effused, the symptoms will be correspondingly severe, and the prognosis must be serious; if only a small amount is effused, the symptoms will be proportionately light, and recovery, or partial recovery, may occur. If the hæmorrhage is in the cervical region, death is more likely to follow.

If the patient survives the first attack, he may die exhausted by cystitis or bed-sores. If he survives long enough, the paralyzed muscles may undergo atrophy,

which may persist during the rest of life, accompanied possibly with contracture.

TREATMENT.—It is quite unlikely that any measures directed to stopping the bleeding can be applied in season to be of any advantage. To prevent further damage by a renewal of the hæmorrhage or by secondary myelitis, the patient should be kept quiet, and cold applied to the back continuously. Local blood-letting may be resorted to, ergot may be given internally, pain should be relieved, the bowels and bladder should be sedulously cared for, the danger of bed-sores should be kept in mind, and subsequent paralyses and atrophies should be combated by the usual means.

CHAPTER XIII.

COMPRESSION OF THE SPINAL CORD.

KADNER, Zur Casuistik der Rückenmarkscompression. *Arch. der Heilkunde*, 1876, p. 481.—KAHLER, O., Ueber die Veränderungen welche sich im Rückenmarke in Folge einer geringgradigen Compression entwickeln. *Zeitschr. f. Heilk.*, iii, 1882, p. 187.—HUMPHREY LAURENCE, Slow Compression of the Spinal Cord. *Lancet*, Jan. 5, 1884, p. 14.—SAYRE, LEWIS A., Spinal Disease and Curvature. London, 1877.—MARSH, H., On the Diagnosis of Caries of the Spine in the Stage preceding Angular Curvature. *Brit. Med. Jour.*, June 11, 1881, p. 913.—RUSSEL, WILLIAM, The Early Diagnosis of Spinal Caries. *Brit. Med. Jour.*, Nov. 12, 1881, p. 771.

SLOW COMPRESSION.

Sudden compression, in so far as it is not surgical, has been mentioned in connection with spinal meningeal hæmorrhage.

ÆTIOLOGY.—Slow compression is caused by caries of the vertebræ, by thickening of the membranes (pachymeningitis), by cancer of the vertebræ, or by tumors within the vertebral canal.

PATHOLOGICAL ANATOMY.—The changes found in the cord are the same as those found in myelitis; sometimes the destruction is complete, the cord being softened; sometimes it is pressed out of shape, and has undergone chronic interstitial changes, which give it a consistency firmer than natural. Secondary degenerations are found above the point of compression in the posterior columns, sometimes in the cerebellar tracts, below in the anterior and lateral pyramidal columns.

The membranes are more or less inflamed, thickened,

and covered perhaps with pus; especially in caries the dura mater may be pressed inward by collections of pus so as to press upon the cord. It is rare to find the vertebral canal so narrowed by displacement of the vertebræ that the bones press upon the cord. If there is no pus formed behind the dura mater, and if myelitis is not set up, the bones may soften and fall together, so as to form a very marked curvature, with almost no symptoms referable to the cord.

If the membranes are inflamed and thickened, the nerves, as they pass out of the vertebral canal, surrounded by the diseased membrane, are also inflamed.

Symptoms.—The symptoms will vary according to the level of the disease causing the compression; but there are symptoms common to all localities.

The earlier symptoms are dependent upon irritation of the nerves or the membranes; subsequent symptoms depend also upon disease of the cord.

Pain generally first attracts attention. The pains due to irritation of the nerve-roots are of a shooting, darting character, referred to the peripheral distribution of the affected nerves. If the upper cervical nerves are thus irritated, the pain may be felt over the back of the head, the side of the face near the angle of the jaw, or over the neck and shoulders. When the cervical or lumbar nerves are affected, the pain will be felt in the limbs. If the dorsal nerves, the pain will be felt in the chest or upper part of the abdomen, usually near the median line, sometimes a little on one side. It may simulate angina pectoris, or the stomach-ache, or colic, according to location. Instead of pain, there may be only a sense of discomfort or irritation, as itching. Motions which change the relation of the vertebræ to one another, as bending, or twisting the trunk, may increase the pain very much, this is especially so when the vertebræ are diseased. Jars, as in riding, or percussion on the shoulders, will increase the pain when the vertebræ are diseased.

Hyperæsthesia may be noticed during or immediately after the attacks of pain. This hyperæsthesia may also be noticed between and independently of the attacks.

Common sensation may be very much diminished.

These disturbances of sensation depend upon lesion of the nerves, and belong to the earlier symptoms. Later, the backache may be more marked; there appear pains depending upon lesion of the cord, less lancinating in character, which resemble those found in myelitis from other causes. These pains are found in the parts supplied with nerves arising from the cord below the seat of compression; they consist in sensations of numbness, pricking or tingling, a sleepy sensation, as though the parts were asleep, or an aching. Ordinary sensation may be diminished or retarded. Finally, there may be complete anæsthesia below the lesion.

In the beginning, even before there is any pain, there may be motor symptoms, which are frequently overlooked. There is first a sense of fatigue; the patient dislikes to exert himself, and, if a child, will exchange his active plays for more quiet sedentary ones.

When carefully observed, he will be noticed to have a peculiar stiff gait, and, in stooping, the back will be kept rigid and the knees will be bent instead. This is most marked in caries and other diseases of the vertebræ. If the cervical vertebræ are affected, the patient will steady his head with his hands when lying down or rising. Passive motion will be resisted, and, if the head or body is moved forcibly, pain will be excited.

The muscular weakness gradually increases until the patient is no longer able to support himself on his legs. Finally there is entire motor paralysis.

When the disease is above the lumbar enlargement, the cutaneous reflexes are often exaggerated, so that severe contractions may follow even slight irritations. Tendon reflex may be increased, and ankle clonus may

be excited. When paralysis is complete, there is usually contraction of the legs upon the thighs, and of the thighs upon the pelvis. This may be so strong that it can not be overcome by any reasonable amount of force.

General epileptiform convulsions occasionally occur even when the disease is situated in the lower part of the cord.

The muscles may undergo atrophy. There may be herpes zoster; bed-sores may form. Disease of the joints, spinal arthritis, has been seen in vertebral caries.

In caries and cancer of the vertebræ, these symptoms may be independent of any deformity; neither is there tenderness on pressure over the spinous processes until after the earlier stages.

When the cervical or upper dorsal part of the cord is affected, the pupil may be widely dilated or contracted; generally the latter. The face and eyes may be more or less congested from paralysis of the vasomotor nerves.

The temperature of the whole body may be influenced by the disease in the cervical region. The heart's action may be slow; respiration may be disturbed.

DIAGNOSIS.—It is important to form a correct diagnosis early in vertebral caries, especially as the longer the delay the more likelihood there is of deformity.

The earliest symptoms have already been mentioned, and whenever they are met a careful examination should be made of all the circumstances attending their origin, cause, and development. The physician must disabuse himself of the idea that in caries of the vertebræ there is necessarily deformity or tenderness to pressure over the spine; there may not be tenderness even to direct percussion in the early stage, but percussion on the shoulders may give rise to pain in the diseased parts.

Acute spinal meningitis is attended with pain in the back and limbs, but it commences suddenly with fever, and is evidently a severe affection.

The pain attending spinal irritation may lead to a suspicion of compression of the cord, and it may not always be easy at once to say there is no disease of the bones. The attending symptoms will generally clear up the diagnosis. There is less of the peculiar stiffness of gait and carriage, the pain is not felt so acutely at the peripheral end of the nerves, the pain is not increased by percussion on the head or shoulders to the same degree, and in spinal irritation there is much greater tenderness on pressure over the spinous processes than is ever found in compression at so early a date. The age of the patient, the history of the origin of the affection, and the past history of the patient, may aid in diagnosis, as will also the hysterical physiognomy which is often to be noticed in the less serious affection.

The diagnosis between the different causes of compression of the spinal cord must often be made from symptoms other than those belonging to the spinal disease itself. Aneurisms of the aorta may erode the vertebræ and press on the cord; there is usually very little difficulty in recognizing the nature of this affection.

Cancer of the vertebræ may give rise to very similar symptoms with caries. When the pain, shooting along the course of the nerves, is extremely severe, without intermission, apparently independent of movement, the probability is that it is caused by cancer; yet, early in the disease, the pain may be much less severe, or may be scarcely noticeable. The spinal cord itself is less frequently implicated in cancer, and there is not the formation of pus which is seen when the bodies of the vertebræ are carious.

The age of the patient may aid in diagnosis, caries being most frequent in early childhood, an age when cancer is very rare.

The presence of cancer elsewhere, and the cancerous cachexia, would aid materially in diagnosis.

A tumor within the vertebral canal may give rise to

symptoms closely resembling those of caries. The pain, central and peripheral, may be the same; the paralysis may be similar. There is less marked stiffness in gait, less difficulty in bending the spine; percussion of the shoulders is less painful. The age of the patient, and his previous history, will aid the diagnosis. If a slight deformity is discovered, tumor would be excluded.

PROGNOSIS.—The prognosis of caries is not very unfavorable. If there is deformity, it can not be remedied, but even extreme paralysis may disappear, and the patient recover. If muscles have undergone atrophy, they may be partially restored. The nearer the disease is to the medulla, the more serious is the condition, and the greater danger of sudden death.

The prognosis in cancer of the vertebræ and tumors, or aneurisms penetrating the spinal canal, is necessarily unfavorable.

TREATMENT.—Of internal remedies, those which will restore the general health when the constitution is broken down are of most value.

In caries the only hope of recovery is to be found in ankylosis of the diseased vertebræ. As the inflammation around the diseased bones is increased by their pressure one upon the other, and by the friction of diseased surfaces against one another, it is necessary, in order to diminish that influence as much as possible, to keep the diseased parts quiet and relieve the bodies of the vertebræ of pressure. The means of accomplishing this need not be mentioned here; it belongs rather to surgery. The treatment of cold abscesses also belongs to surgery.

To relieve the paralysis in caries of the spine, the actual cautery, applied by the side of the spine, has been used with excellent results. This can not well be applied while the patient is wearing a jacket, except as that is removed for a day or two, and this is rarely advisable.

Electricity, faradic or galvanic, to stimulate paralyzed muscles, should be used.

The nutrition of the patient should be maintained as well as possible; cod-liver oil and cream are especially indicated in strumous subjects. The patient should be placed in the best hygienic conditions possible.

SPINAL TUMORS.

The more common varieties of tumors found in the vertebral canal are cancer, generally arising from the vertebræ; sarcoma and fibro-sarcoma, and osteoma; parasites, echinococcus, or cysticercus, are more frequently connected with the membranes; tubercular and syphilitic tumors may be either connected with the membranes or be seated in the substance of the cord itself; gliomata are found in the substance of the cord.

From pressure or from secondary inflammatory changes the spinal cord undergoes a degenerative process usually leading to softening; sometimes, however, there is simply atrophy of the nerve-elements, and the cord may acquire a somewhat firmer consistency than normal. When the tumor is in the substance of the cord, its center may undergo degeneration, and, by a process of softening, a cavity be formed. Many of the cavities found in the spinal cord originate in this way; gliomata are most liable to this change.

ÆTIOLOGY.—Except in cases of tubercle, syphilis, and cancer, we know very little about the causes of spinal tumors, and even in regard to these varieties we can only say that the germs are conveyed by lymphatics or blood-vessels to their new seat of growth, or that a corresponding diathesis causes their growth. Sometimes it would seem that an injury, as a fall or a blow upon the back, has served as a starting-point for the growth of tumors.

EXTRA-MEDULLARY (MENINGEAL) TUMORS.

SYMPTOMS.—The symptoms are almost the same as those found in connection with caries of the vertebræ. There are the symptoms due to irritation of nerve-roots and those depending upon compression of the cord. The symptoms may be unilateral or bilateral, according to the locality of the tumor. The growth of the tumor is usually very slow, and the development of the symptoms is correspondingly slow, the slighter early symptoms sometimes continuing for years before a definite diagnosis can be made. Pain at the seat of the tumor, of a dull, pressing nature, may be increased by motions of the body, but is felt at other times also. Percussion over the spinous processes may increase the pain or give it for a moment a more lancinating character. The nerves arising from the level of the tumor may be implicated; then the pain will be felt at the periphery, as in caries. Atrophy of the muscles to which these nerves are distributed with the reaction of degeneration will indicate the serious change which the tumor may cause in the nerve-roots. Other trophic lesions, as herpes and bed-sores, may make their appearance.

Paralysis finally sets in with increased reflex irritability, spasms, or contractures. A careful study of the nerves affected, as shown by the distribution of the paralysis or the anæsthesia, will indicate the level of the disease, and show also whether the cord is affected, or only the nerves of the chorda equina.

DIAGNOSIS.—No symptoms or combination of symptoms are sufficient for forming a positive diagnosis; it is only by a careful examination of all the circumstances that other affections can be excluded and the probability of a tumor be recognized. Caries and cancer of the vertebræ most closely resemble tumor in their symptoms.

INTRA-MEDULLARY SPINAL TUMORS.

The tumors which have been found in the substance of the cord are gliomatous, tubercular, syphilitic, or sarcomatous. They are very rare. Their growth is often slow, but they give rise to symptoms sooner than the extra-medullary growth. There is no necessity for describing these growths, as they are like others found elsewhere.

SYMPTOMS.—The symptoms are very much like those belonging to acute or chronic myelitis, including disturbance of sensation and motion, atrophy of muscles, and local trophic changes. Sometimes the symptoms much more closely resemble those due to meningeal tumor, pain, both local and peripheral, and increased reflex irritability, being prominent. The symptoms must vary with the seat, rate of growth, and consequent size of the tumor. There are no symptoms diagnostic of spinal tumors by which one can be guided to a certain conclusion.

PROGNOSIS.—The prognosis is necessarily unfavorable. A syphilitic gummata may theoretically be absorbed, but it would then be impossible to satisfy a skeptic that the diagnosis was correct.

TREATMENT.—Except the use of iodide of potassium or some equivalent preparation, there is nothing to be done further than to care for the patient's comfort and look after any complications which may arise.

CHAPTER XIV.

SYRINGOMYELIA.— FORMATION OF CAVITIES.— HYDROMYELUS.

SCHÜPPEL, O., Ueber Hydromyelus. *Archiv der Heilk.*, vi, 1865, p. 289.—WESTPHAL, Ueber einen Fall von Höhlen- und Geschwulstbildung im Rückenmarke mit Erkrankung des verlängerten Marks und einzelner Hirnnerven. *Arch. f. Psych. und Nervenkr.*, v, 1875, p. 90.—SIMON. *Ibid.*, p. 108.—SCHULTZE, F. *Ibid.*, viii, 1878, p. 367.—EICKHOLT, AUGUST. *Ibid.*, x, 1880, p. 695.—WESTPHAL, C., A Contribution to the Study of Syringomyelia (Hydromyelia). *Brain*, July, 1883, p. 145.

Occasionally cavities are found in the spinal cord, which are clearly the result of an abnormal development of the central canal; this condition may be congenital. The canal may be dilated through only a short tract, or through nearly its whole length. Sometimes the canal is double, or diverticula may be found which branch from the canal and can be followed for a few millimetres, running near the central canal. As another variety of malformation, cases are seen where the central canal has not been closed. The central canal may be secondarily dilated, when by pressure it is closed above or below the dilated portion, or it may be found dilated in connection with certain diseases, as cerebro-spinal meningitis, or occasionally in cases of chronic myelitis.

In all these instances in which the central canal is enlarged, the walls of the cavity will be lined with epithelium, and it will be situated the same as the normal canal with reference to other parts of the cord.

In a large number of cases, however, the cavity is pathological and is independent of the central canal, which may be seen just in front or to one side of the abnormal cavity; the central canal is usually distorted, and it may be so flattened as to be scarcely recognizable, only a narrow line of epithelial cells showing its location. An abnormal cavity, according to Simon, may be lined with cylindrical epithelium, as when one is formed in a glioma. He thinks position is most important for diagnosis.

The cavity is most frequently found in the posterior part of the cord, it may be formed at the expense of the gray commissure or the posterior cornua, may take part of the space occupied by the posterior columns, or it may be in the anterior cornua. The gray substance is much the more frequently affected. The cavity may be single or double, may be a few millimetres in length or may extend the whole length of the cord, and may be very small or as large as the finger.

Ætiology.—The cause of the formation of a cavity is not the same in every case. A hæmorrhage into the cord may leave a cavity after the clot has been absorbed; the plugging of blood-vessels, much more rare, may be the cause. Several cases have been reported in which it has seemed that a glioma formed in the central gray substance, and that the center of this has softened and been absorbed. Hallopeau has suggested that an inflammation about the central canal may give rise to an enlargement of that canal, or a central myelitis may lead to the formation of a canal outside the central canal.

Eichorst and Naunyn found that, after crushing the cord in young animals, a cavity was formed above the point crushed. They referred this to the dilatation of a lymph-canal which they suppose runs at the bottom of the posterior fissure. Westphal accepts this as a possible explanation of the formation of some cavities.

There are no special symptoms caused by cavities,

so far as is known. Those symptoms which have been found in cases of syringomyelia were such as were due to the disease which gave rise to the cavity.

There is nothing to be said as to treatment other than what belongs to the primary disease, if any, which causes the formation of the cavity.

CHAPTER XV.

MYELITIS.

FROMMANN, C., Untersuchungen über die normale und pathologische Anatomie des Rückenmarks. Jena, 1864, 1867.—DUJARDIN-BEAUMETZ, G., De la myélite aiguë. Paris, 1872.—ANDERSON, M'C., On a Case of Myelitis. *Edin. Med. Jour.*, Aug., 1881, p. 97.—HALLOPEAU, H., Étude sur les myélite chroniques diffuses. *Arch. gén. de méd.*, Sept., 1871.—ZUNKER, Beiträge zur Myelitis Chronica. *Charité Annalen*, v, 1880, p. 260.

Myelitis is an inflammation of the spinal cord, and may be acute or chronic; the gray or the white substance may be affected, the nervous tissues, cells, and fibers may be chiefly and primarily affected, or the principal change may be found in the interstitial tissue, the nervous structures suffering secondarily.

ACUTE MYELITIS.

ÆTIOLOGY.—Acute myelitis is most frequently caused by exposure to wet and cold; these two influences are most likely to give rise to inflammation of the spinal cord when the legs, more especially the thighs, and the back are thus exposed for a considerable length of time, as by sleeping upon the damp ground in cool weather, or riding in a carriage or on horse-back in a storm, with insufficient protection. The influence of the above causes is very much increased if there has been severe or prolonged bodily exertion at the time of the exposure or just preceding it. Excessive bodily exertions may alone be the cause of the disease.

Many acute febrile diseases are occasionally accom-

panied by a myelitis; this will be referred to again (post-febrile paralysis).

Lead-poisoning is not unfrequently the cause of symptoms closely resembling those of myelitis; indeed, it is probable that in such cases there is inflammation of the spinal cord, but generally of a chronic form.

Excess in venery, and syphilis, may give rise to myelitis; so may injuries to the back, from falls, blows, etc.

Severe emotions, as fright and anger, may occasionally give rise to inflammation of the spinal cord.

Pathological Anatomy.—The spinal cord affected with acute myelitis is generally softened, but occasionally its consistency is increased. The softening may be only slight, or the cord may be quite liquid. The color is either reddish, if there is an admixture of blood with the *débris* of the cord, or yellow, if fatty degeneration has occurred to any extent, or white. The softening may occupy a continuous stretch of the cord, or it may be scattered about in isolated spots; the gray substance is rather more easily affected than the white.

The dorsal region is more frequently the seat of softening than either the cervical or lumbar. When the cervical region is affected, it is said that the disseminated variety is the more common.

Above the portion directly affected there is found secondary ascending degeneration of the posterior columns and cerebellar tracts; below, secondary descending degeneration of the pyramidal tracts. This secondary degeneration can be best seen from the change of color after hardening in bichromate of potassa or chromic acid.

With the microscope, the minute changes of structure may be studied better upon hardened specimens. Either the nervous structures are chiefly affected, or the interstitial tissue is first altered. The nerve-fibers are first swollen, the myeline becomes granular, and the

axis cylinder is either broken up and disappears or is enlarged, even to ten times its normal diameter; these enlarged axis-cylinders may be filled with cavities—vacuoles; the enlargement is varicose or affects only a short length of the axis; it may be spherical or fusiform. These enlarged axes soon break up and disappear in the general *débris* of the softened tissue. When the cord acquires increased consistency, this hypertrophy of the nerve-fibers is either entirely wanting or is very slight.

The nerve-cells are also swollen, acquire a globular appearance, their outline may be less distinct than normal, and the nucleus may be pushed to one side, even so as to project beyond the general outline of the cell; they may be filled with vacuoles, or they may have a shining, glassy appearance—vitreous. There may be a large deposit of pigment in the cells. They finally become granular, break up, and disappear.

Changes in the neuroglia may be the starting-point in myelitis; then those in the nervous structures are secondary, and there is less likely to be hypertrophy of the nerve-fibers and cells. The nuclei of the neuroglia multiply, the fibers swell up and are thicker, and they become brittle and undergo fatty degeneration. Granular corpuscles form at the expense of the nuclei and connective tissue. As the nutrition of the nervous elements is interfered with, they also degenerate, and the cord is soon reduced to a soft, semi-liquid consistency. When the cord acquires an increased consistency, the fibers and cells of the neuroglia are multiplied somewhat as in sclerosis, though to a less degree; the nerve-fibers in these cases are destroyed, and their place is filled with granular *débris* or a liquid which becomes granular on hardening.

The walls of the blood-vessels are rarely if ever thickened in acute myelitis; they are more likely to lose consistency and rupture easily, giving rise to hæmorrhages which aid in the process of disintegration.

The walls of the vessels are often covered with granular corpuscles.

Symptoms.—Acute myelitis may begin with a chill and fever before any distinctive spinal or nervous symptoms appear. The temperature only rarely reaches 104°; the pulse may be as high as 150; with the pyrexia are the usual constitutional symptoms—anorexia, headache, and general malaise.

Very frequently the commencement of the disease is more gradual; a sense of weariness, heaviness, with backache and undefined sensations in the limbs, precede the initial fever.

Soon after the chill and fever, sometimes without any distinct pyrexia, a numbness or a pricking and tingling is noticed, usually in the toes and feet. These abnormal sensations increase in severity and gradually extend up the leg.

With these symptoms, or soon after their advent, rarely as the initial symptom, the patient is aware of a loss of strength in his legs; he is soon wearied in walking; in a very short time this increases, so that he is unable to walk, and must keep his bed. In many instances there is tremor or cramps at the beginning of the attack, but no marked convulsions nor spasms.

The disturbance of motion and sensation extends upward, affecting both limbs with increasing and nearly equal severity, until there may be entire paralysis of motion and complete loss of sensation in the legs.

The different reflexes, cutaneous and deep-seated, are first diminished, then lost, unless the myelitis is limited to a comparatively short segment of the cord above the lumbar region. The reflex actions which control the bladder and rectum are lost; there is, at first, usually retention of urine; later the urine dribbles away from over-distention of the bladder and paralysis of the sphincter. There is constipation rather than involuntary action of the bowels.

A sense of constriction, girdle sensation, is noticed

around the thighs—later around the waist. This may be very annoying to the patient.

As the inflammation extends upward in the cord, the trunk is affected, the costal respiratory muscles cease to act, the respiration becomes diaphragmatic, there is inability to expel the mucus which may accumulate in the bronchial tubes; the breathing therefore becomes noisy, the upper extremities are also affected, the patient finally ceases to breathe, and dies of apnœa.

When the inflammation extends downward rather than upward, its progress can be recognized, though less certainly, by observing the loss of reflexes in a descending order, or the gradual extinction of electrical reactions. For this, careful comparative examinations are necessary, such as it is not always desirable to make.

Pain is not a prominent symptom in acute myelitis; it is not present unless the membranes are also implicated. The tingling numbness may be so severe as to give the patient much discomfort, and there may be aching and a sense of unrest in the limbs; but it is not rare to have the disease run its course without even this amount of discomfort. Backache is said by some to be one of the symptoms of acute myelitis; if this is prominent, there is probably an accompanying meningitis. There is no tenderness over the spinous processes, and spontaneous spasms or evidences of reflex irritability are wanting. Pain or tenderness may be shown by passing a sponge wet with hot water, or a lump of ice, over the back; a severe burning sensation will be felt at the seat of the lesion. Electricity will sometimes act in the same way. Hyperæsthesia of the skin is not found in myelitis, excepting occasionally a narrow zone at the upper limit of the region affected with anæsthesia. Symptoms of motor and sensory irritation, however, are often seen in cases of myelitis, because very frequently the membranes are implicated.

A tonic contraction of the legs, a rigidity in exten-

sion, is a symptom which belongs to the later stages, when the disease is above the lumbar enlargement; there is then difficulty in abducting the legs, and passing a catheter may become difficult from the exaggeration of this contraction caused thereby.

The urine may become alkaline early in the disease, as Erb thinks, not improbably from direct nervous disturbance of the secretory functions. There is always danger, also, of this change in the urine from retention, the bladder being only imperfectly emptied. Cystitis is one of the complications to be watched for. When the lumbar enlargement is not affected, the urine may be passed involuntarily, and, if sensation is much disturbed, without the patient's knowledge. Bed-sores sometimes form with great rapidity, enormous masses of tissue sloughing away and giving rise to possible purulent infection. Even if such acute disturbance of nutrition does not occur, it is very common to have a more slowly developed bed-sore. An eruption of herpes, bullæ, or pemphigus may appear on the limbs.

The nerves arising from the part of the cord affected and the muscles supplied by them undergo destructive degeneration, and there may be wasting of the limbs, appearing more or less rapidly. The electrical reaction is affected under these circumstances, there being the reaction of degeneration.

Above and below the principal focus of disease there will be secondary degeneration if the patient lives long enough. Then there may be found the exaggerated tendon reflexes and other symptoms belonging to lesion of the lateral pyramidal tracts.

This description has been rather that of a severe case, which runs its course to a fatal termination. Frequently the symptoms are less grave: sensation is not entirely abolished in the legs; the reflexes are not absolutely lost; perhaps one side is chiefly affected, the other slightly so; after a variable length of time there is a recession of the symptoms, the disease has ceased

to advance, and the patient is recovering. The recovery is almost never complete. There generally remains some impairment of function.

As an unusual complication may be mentioned optic neuritis, occurring at the same time or just preceding subacute myelitis, as observed by Erb. He thinks that the optic nerves and the spinal cord are both easily affected by the same injurious influences, and so may together be attacked with subacute inflammation.

DIAGNOSIS.—It is necessary to distinguish acute myelitis from meningitis, hæmorrhage, and acute ascending paralysis.

It is also desirable to form an opinion as to the part of the cord affected.

In meningitis there is much more severe pain, both in the limbs and back—such pain as to cause the patient to complain of it bitterly; this pain is increased upon motion. There is often great hyperæsthesia of the limbs; fever runs higher in meningitis than in myelitis; reflex actions are much more exaggerated and the contractions are more constant, and the limbs may be flexed, or there may be opisthotonus. Paralysis is a later symptom in meningitis; trophic disturbances of the skin are rare.

Hæmorrhage is distinguished by the suddenness with which the initial symptoms arise, without fever, the injury preceding the disease, or, if spontaneous, the severe pain preceding or attending the commencement of the attack. The stationary character of the symptoms after the first attack, or their gradual extension secondarily, also when the cervical and lumbar enlargements are the seat of the hæmorrhage, the rapid wasting and loss of electrical reaction, aid in forming a diagnosis. If, however, the history of the case is imperfect, a diagnosis may be extremely difficult.

Acute ascending paralysis may be diagnosticated by the fact that sensation is little if at all affected; the bladder and rectum are not likely to be disturbed;

there is no bed-sore; the muscles do not undergo atrophy.

The diagnosis of the seat of the lesion must be made from a study of the symptoms, keeping in mind the physiology of the cord. Unilateral acute myelitis is almost never seen, excepting as the result of injuries, and need not be specially considered. In most instances the central gray substance is first affected; the disease spreads then to the white substance. If the antero-lateral columns are first affected, there will be loss of motor power, and, if the disease begins in the pyramidal tracts, the symptoms of lesion of those tracts. A much less extent of disease of the motor tracts will cause paralysis of motion than is necessary to give rise to loss of sensation; indeed, if but a small portion of the gray substance is left, sensation is not entirely destroyed.

Myelitis is much more common in the dorsal region, possibly explained by its vascular supply being less sure, as pointed out by Adamkiewicz.

An examination of the reflexes, as suggested by Gowers, will aid in fixing the upper limit, and sometimes the lower limit, of the disease; so will a careful study of the muscles paralyzed and of the region affected with anæsthesia, by which means we can recognize what nerves have lost their function.

PROGNOSIS.—When myelitis begins violently and the paralysis advances rapidly, the prognosis is unfavorable; the same is true when the disease has reached or has commenced in the cervical region, and especially if respiration is disturbed. If there is much cystitis, or if bed-sores form, the prognosis is unfavorable, even if other symptoms seem mild, and the more so if the general health suffers severely and if the patient's constitution seems undermined. It is, however, often impossible to convince either the patient or his friends that he must die, so little discomfort does he experience.

The more gradual the advance of the disease, and the more incomplete the loss of function, the more favorable is the prognosis. A slight remission of symptoms and sustained general strength are also favorable.

Even when the patient has apparently nearly regained his health, a relapse is possible, and, after one attack, a slight imprudence may cause another, so that the patient must take extra care of himself.

TREATMENT.—As soon as the disease is recognized, the patient should be put to bed and kept there. As perfect rest of mind and body as possible is absolutely necessary, even against the protest of the patient, who may be conscious of only slight numbness or weakness.

Ice-bags to the spine, applied continuously, are of benefit in meningitis, and may be used in myelitis, though their value is less certain. A mild form of counter-irritation, dry-cupping, is of value, and should be employed; two to six or eight cups can be applied daily; usually two are sufficient, changing their place each time. The cups should remain on about half an hour, and should leave the skin much congested when removed. If the attack is very severe, the danger of bed-sores may contra-indicate cupping.

Ergot, drachm doses of fluid extract, or six or eight grains of ergotin, should be given three times a day. This may be combined with one of the preparations of belladonna.

Iodide of potassium may be used even in the earlier stages with advantage, and later even more efficaciously, especially where syphilis is suspected, and in those cases mercury may be combined with it.

The greatest care should be taken to sustain the patient's nutrition, to relieve the bowels, to prevent cystitis and bed-sores.

When the patient is recovering, electricity can be used to maintain the nutrition of the muscles; this may be combined with massage, or the latter can be used alone.

Counter-irritation to the back, actual cautery, and dry-cupping may be employed during recovery, but are of doubtful efficacy then.

CHRONIC MYELITIS.

Chronic myelitis sometimes succeeds acute myelitis, or may result from injuries. It may also follow exposure to cold, fatigue, and long-continued emotional disturbances; the eruptive fevers and other acute diseases may be complicated with chronic changes in the spinal cord; syphilis is a very common cause. In many cases it will be found that lead has been received into the system. So frequently is lead one element in the ætiology, that it should be sought for in every case. Arsenic may give rise to the same symptoms.

PATHOLOGICAL ANATOMY.—After death the spinal cord is sometimes found softened; more frequently, however, it is found firmer in consistency than normal. The seat of the inflammatory changes may vary in different cases; sometimes the gray substance is chiefly affected, sometimes the white substance, and more frequently both gray and white are affected; sometimes the disease extends through the whole thickness of the cord, and sometimes only one half is affected; again, only the periphery of the cord is diseased—chronic cortical myelitis, as it has been called.

The microscopic changes vary according as the connective tissue (neuroglia) or the nerve-fibers and cells are chiefly affected. If the neuroglia is primarily diseased, we have thickening of the connective tissue with increase of its elements; secondary to these changes the nervous elements gradually disappear.

When the latter are chiefly affected, they pass through changes similar to those found in acute myelitis; the neuroglia may be somewhat thickened, or it may apparently suffer no change.

The walls of the blood-vessels are usually somewhat thickened. The tissue around the vessels may undergo

granular degeneration, and thus spots of softening may form.

Symptoms.—The symptoms of chronic myelitis will vary somewhat according to the seat of the lesion. The first symptoms may appear either in the sensory or motor function; the motor phenomena consist in a gradually increasing weakness, affecting one or more limbs, the first sign of failure being a sense of heaviness in the legs or arms, and an unusual liability to become fatigued. These symptoms slowly increase in severity, the weakness becomes more marked, and the patient may be confined to his bed many months before there is entire paralysis. There are rarely spasms; but chronic contractures are not so infrequent, the legs being held in extension and adducted, rarely flexed. Reflex actions are sometimes moderately exaggerated; this is often shown simply by an increase of the pre-existing contraction: thus, if it is desired to draw off the water, the introduction of a catheter may increase the adduction of the thighs so as to render the operation very difficult, especially in a female. The tendon reflexes are sometimes exaggerated and sometimes diminished, according to the location of the disease. When the anterior gray substance is affected, there is, of course, wasting of the muscles, in which case the electrical phenomena undergo the usual changes; otherwise the electrical reactions may even be exaggerated.

Disturbances of sensation appear very early, and are often the first symptom to attract the patient's attention. These disturbances are often simply a sense of numbness and tingling, as if the limbs had been asleep, without any disturbance of tactile sensibility; but sometimes the sense of touch is affected, and there is more or less marked anæsthesia. Pain is not very common, but is occasionally very severe. In some cases there is hyperæsthesia to touch, or the sense of touch is perverted so as to give rise to a peculiar vibrating pain. This sensory disturbance may remain limited to

one limb, to a toe or finger, during several weeks before extending, or before other symptoms appear.

The condition of the bladder and rectum varies according to the seat of the lesion. There is danger of cystitis when urine is retained, as in the acute form. The sexual function is gradually abolished, though occasionally the sexual appetite may be increased. After the patient is confined to bed, bed-sores are liable to form, especially if cleanliness is neglected.

The disease is slowly progressive toward a fatal termination, but there are occasionally periods of remission and improvement which may be so great as to encourage the hope of final recovery; but some imprudence or exposure starts up the inflammation again, and causes an aggravation of the symptoms. Death may not occur for several years after the commencement of the disease.

DIAGNOSIS.—In well-marked cases this form of myelitis is not likely to be mistaken for other diseases of the spinal cord; but in some instances it may be doubtful whether there is locomotor ataxia, multiple sclerosis, lateral sclerosis, or disease of the anterior cornua. When multiple sclerosis affects chiefly the spinal cord, it may be impossible to make a correct diagnosis.

Vulpian says: "Every time there is found in a chronic affection of the cord an irregular course of the disease—causing weakness and paralyses of different parts of the body, giving rise to combinations of symptoms belonging some to one systematic lesion, some to another, and presenting, as a whole, symptoms which, except for the rapidity of their appearance and their succession, would be more or less similar to those noticed in acute diffuse myelitis—it may be asserted that it is a case of chronic diffuse myelitis.

"Whenever, in any chronic affection of the cord, the assemblage of symptoms allows the elimination of systematic lesions, of sclerosis in patches, of chronic

myelitis of the anterior cornua, the case is one of chronic diffuse myelitis."

PROGNOSIS.—The disease is one of long duration and, as has been said, with periods of remission and improvement. Complete recovery is extremely rare, except in cases caused by lead or those occurring after fevers; it probably never occurs, there always being some impaired function remaining to show that mischief has been done to the cord.

In judging whether there is immediate danger to life, the circumstances of each case must be taken into account, and no general directions can be given.

TREATMENT.—Active measures, such as are used in acute myelitis, would be entirely out of place in chronic myelitis.

Dry-cupping, the actual cautery, the iron being heated to a white heat and drawn rapidly and lightly over the back, so as to simply char the cuticle without producing suppuration, small blisters applied in succession along the spine, and iodine, may be employed as counter-irritants; of these, the best are dry-cupping and the actual cautery. Brown-Séquard recommends a douche of hot water to the back, the application being made for two or three minutes every day.

The galvanic current may be used, one pole being placed above, the other below the probable seat of the disease; the direction of the current may be varied at different sittings, both electrodes being held stationary, or one moved slowly up and down the back; rather a weak current should be used, only for a few minutes at a time, the application being made daily, and the treatment persevered in for months. Sometimes this treatment will give rise to unpleasant symptoms; it should then be discontinued. Erb says that he has obtained benefit in fifty-two out of one hundred cases treated by galvanism.

Erb says: "The water-cure is, all things considered, one of the most important and most promising means

of treating chronic myelitis. The mistrust with which it is regarded by some authors is, as far as my own experience goes, entirely unjustifiable. It is suitable for most all cases, though, of course, the method of application must vary according to the peculiarities of the individual cases.

"Simple rubbing with wet cloths, foot-baths, sponging the back, hip-baths, half-baths, with affusions to the back, local compresses to the back, left on till they become warm, etc., seem to be the measures which are chiefly applicable. The treatment should always be begun with moderate temperatures (20° to 25° C., or 68° to 77° F.), and we should never go below 16° to 12° C. (60⅘° to 53⅗° F.). I believe, also, that excessive prolongation of the treatment is injurious."

Of internal remedies, nitrate of silver, a quarter to half a grain three times a day for four or five weeks, then omitted for a short time; ergot, half a drachm to a drachm of the fluid extract three times a day; double chloride of gold and sodium, gr. $\frac{1}{20}$ three times a day; ext. of belladonna, gr. $\frac{1}{4}$ to $\frac{1}{2}$ twice or three times a day; iodide of potassium should be used if there is lead, and anti-syphilitic treatment when it is indicated.

In chronic myelitis there is less reason to keep the patient quiet and at rest than in acute myelitis; yet over-exertion should be carefully avoided. It is much more prudent to restrain the patient's activity more than is necessary than to allow even a slight over-exertion. The same may be said of all imprudent exposures to influences which are likely to cause the disease.

ACUTE ASCENDING PARALYSIS.

This is sometimes called Landry's paralysis, because Landry first described the combination of symptoms.

It is defined by Erb as "a motor paralysis which generally begins in the lower extremities, spreads pretty rapidly over the trunk to the upper extremities, and usually also involves the medulla oblongata, which

sometimes runs its course without fever, sometimes with more or less active fever, which but slightly involves the general sensibility and the functions of the bladder and rectum, and which runs its course without notable atrophy of the muscles, and without any diminution or change in their electrical excitability."

This definition gives nearly the whole symptomatology of the disease. It is only necessary to add that prodroma, disturbed sensations, numbness, and aching in back and limbs, may precede the motor paralysis; that the disease may commence in the upper extremity; that reflex actions may be much diminished or entirely lost; the tendon reflex has not been carefully studied; and the functions of the brain do not seem disturbed until just before death.

The disease usually ends fatally when the nerves arising from the medulla are affected. The symptoms may cease to advance at almost any stage, may recede, and the patient may recover.

The symptoms recall those which are found in acute or subacute anterior poliomyelitis; the retention of electrical reaction in the muscles is the chief difference.

Many cases have been examined after death, and no lesions discovered in the cord.

When Landry described this form of paralysis in 1859, the electrical reaction of muscles was not taken into account in forming a diagnosis, and there seems no special reason why that should be added in order to form a distinct disease. Several autopsies (Eisenlohr, Fox, v. d. Velden, Peabody) have lately shown that there are changes in the cord; though these changes have seemed to be very slight, yet they have been quite diffused, and are such as may indicate an early stage of myelitis, sometimes affecting the white substance, sometimes the gray; sometimes attended with no change in electrical reaction, sometimes accompanied with such change. No case has yet been reported in

which a proper examination of the peripheral nerves has been made.

The causes of this form of paralysis are said to be the same as those of myelitis; the treatment should be the same as in acute myelitis.

It is not my purpose to enter upon a long discussion of any doubtful points. In view of the cases which have been published, I can see no satisfactory reason for retaining acute ascending paralysis as the name of a distinct disease; the cases coming under that designation can be included under one of the forms of acute or subacute myelitis.

CHAPTER XVI.

POLIOMYELITIS.—MYELITIS OF ANTERIOR CORNUA.

PETITFILS, A., Considérations sur l'atrophie aiguë des cellules matrices. Paris, 1873.—GOMBAULT, Note sur un cas de paralysie spinale de l'adulte, suivi d'autopsie. *Arch. de physiol.*, 1873.—BERNHARDT, Ueber eine der spinale Kinderlähmung ähnliche Affection Erwachsener. *Arch. f. Psych. u. Nervenkr.*, 1874, p. 370.—BENNETT, A. H., On Chronic Atrophic Spinal Paralysis in Children. *Brain*, Oct., 1883, p. 289.—SEGUIN, E. C., Myelitis of the Anterior Horns. New York, 1877.—PROUST, A., and BALLET, G., Contribution à l'anatomie pathologique de la paralysie générale spinale diffuse subaiguë de Duchenne. *Arch. de physiol.*, Oct., 1883, p. 330.

ACUTE ANTERIOR POLIOMYELITIS.

This is the name that has lately been given to a class of diseases characterized by changes in the anterior cornua. These changes are generally considered to be of an inflammatory nature.

The so-called infantile paralysis was for a long time the only recognized manifestation of this disease, but during the last ten years or so cases have been reported as occurring among adults having very nearly the same symptoms, and after death presenting similar lesions of the anterior cornua.

It ought to be mentioned that some authors consider this affection primarily a disease of the muscles. This view is not generally accepted. Leyden refers the symptoms in some cases to a diffused or general neuritis.

ÆTIOLOGY.—By far the larger number of patients

are infants from one to three years of age; among adults, the larger number are attacked between the years of twenty and forty; between the years from four to fourteen there seems to be comparative exemption from this form of myelitis. During infancy both sexes are about equally liable to the disease; among adults, males are rather more frequently attacked than females.

During dentition the nervous system of a child is in a more irritable state, and perhaps more likely to suffer from injurious influences; this may explain the frequency with which infantile paralysis occurs during the first and second years.

Cold acting upon the surface of the body may be a cause of this form of myelitis as of other forms. In a very few cases I have been able to learn that during the night preceding the occurrence of the paralysis the child has been found to have kicked off the clothing, and thus become chilled.

Falls and other injuries have sometimes seemed to be a cause. In adults, excessive exertion.

The summer months show a larger proportion of attacks than the other seasons.

PATHOLOGICAL ANATOMY.—There are reasonable grounds for the opinion that the first change is a congestion of the anterior cornua, and perhaps of other parts of the cord also. In the fœtus and in early infantile life the capillaries more closely surround the nerve-cells, each cell being inclosed in a net-work of small vessels. Adamkiewicz has shown that in the adult a system of canals, smaller than the capillaries, can be injected from the vessels so as to form a net-work, by which each nerve-cell is surrounded. After the first shock of the disturbed circulation, which causes the paralysis, the congestion diminishes, and with this the paralysis disappears, except where the injury to the nutrition of the cells has been sufficient to destroy or seriously impair their vitality.

The essential change seems to be destruction of the nerve-cells; other changes are either accidental complications or secondary. When the patient has survived many years, the affected cornua are found deformed and diminished in size; the neighboring white columns may also be misshapen.

The anterior nerve-roots arising from the affected tract are atrophied and contain degenerated nerve-fibers, or the fibers may have so entirely disappeared that simply connective tissue remains.

The muscles undergo change at a comparatively early period. The muscular fibers lose their transverse striation, are broken up into granular and fatty *débris;* sometimes the muscular nuclei are increased in number; the granular and fatty materials are absorbed, and there remains only the sarcolemma with a larger or smaller number of muscular nuclei. Sometimes, instead of undergoing atrophy, the muscles suffer from a waxy or colloid change, in which the fibers may be greatly enlarged. The interstitial tissue is usually increased in amount, and often fat is deposited between the muscular fibers.

The bones of the affected limbs grow less rapidly than the corresponding healthy limbs, when the patient is a child. In adults there is no atrophy of the bones. Observations are not yet sufficiently numerous to determine whether the bones become brittle.

Symptoms.—In children the disease begins suddenly, sometimes, though rarely, with convulsions. Most frequently the child, having been put to bed apparently in good health, is somewhat restless during the night, perhaps awakes and cries, then sleeps quietly until morning; in the morning it is discovered that one or more limbs are paralyzed; generally one or both legs are affected; sometimes the paralysis is confined to one or both arms, or an arm and a leg; very rarely are the four limbs affected. The loss of power may not be complete at first, but it reaches its height in a very few

hours. The right leg is said to be the most frequently affected. Sensation does not appear to be much disturbed; except at the very beginning, there seems to be no pain, and it is doubtful if there is pain even at the beginning. Occasionally a slight febrile attack precedes the development of paralysis; but often this is so slight, especially in very young children, and of such short duration, as to attract little or no attention, and the severity of the succeeding paralysis is not proportionate to the amount of fever.

When convulsions usher in the attack, they are usually of short duration and differ materially from the severe convulsions of cerebral origin preceding many cases of hemiplegia in infants.

Within a few days, from two or three days to one or two weeks, an improvement in the paralysis is noticed, which may, in light cases, advance to complete recovery, though more frequently the improvement ceases after a few weeks, leaving some muscles still paralyzed. Where more than one limb is affected, the muscles of one limb may entirely recover, while those of the other limb or limbs only partially recover.

Within a short time after the paralysis appears, the muscles begin to undergo the atrophic changes already mentioned, and after a few weeks the affected limb shows decided evidence of wasting. Its growth is also retarded, and after some years there may be a difference of from one to six or seven inches in the length of the legs. Erb says that, while the atrophy is progressing, the muscles are quite sensitive on pressure.

The affected muscles show a change of electrical action—reaction of degeneration—within a few days (four or five) after the attack.

The skin may be dry and scaly, and the circulation sluggish, on account of which the limb is cold and more or less cyanotic. Bed-sores do not form, nor is it likely that herpetic and allied forms of skin eruption are caused by this disease.

The reflexes, cutaneous and tendinous, are more or less disturbed according to the amount of muscular paralysis.

After months or years, sooner or later, according to the amount of wasting, deformities result. Where there is inequality of the limbs, the bones of the pelvis are tilted and the spine is curved. The affected limb suffers also from contracture; club-foot and deformity of the knee and hip joint are seen. The upper limbs are much less frequently subject to contracture and deformity than the lower limbs. Erb divides the causes for these contractures into three classes: 1. "The most frequent cause is the continued approximation of the points of attachment of the muscles, induced partly by the weight of the parts, partly by external pressure in walking, standing, etc." 2. "The antagonists of the paralyzed muscles remain effective." 3. "The proliferation of interstitial connective tissue and its subsequent retraction, which takes place with the degenerative atrophy of the muscles."

After the first febrile attack, the bladder and the rectum are not affected, and all the functions of the body, except those of the paralyzed limbs, are normally performed. Life is not shortened by this disease, and the mental powers of the child are, as a rule, not affected.

In *adults* the course of the disease is somewhat different from that noticed in children; as with other febrile affections, convulsions are absent; pain is a rather more prominent feature; febrile reaction is rather more marked, though it may be absent; the paralysis is developed rather more slowly; sensibility is more frequently disturbed at the beginning, though this usually soon disappears; occasionally vomiting and gastric disturbances are noticed. After some days or weeks, longer than with children, the paralysis amends, and, as in children, there may be complete recovery, though partial recovery and wasting are the

more frequent result. Of course, there is in adults no retarded development, and subsequent deformity is much less than in children. The electrical reaction and other symptoms are almost identical.

Diagnosis.—The disease which has been most frequently confounded with acute anterior poliomyelitis in children is hemiplegia from cerebral cause. In this, convulsions are much more frequent and severe, the paralysis is hemiplegic rather than paraplegic, the electrical reactions remain unchanged, contractions resemble the hemiplegic contractions found in adults, and there may be post-hemiplegic chorea; the growth of the paralyzed limbs is much more retarded.

Hæmorrhage into the spinal cord, though occurring suddenly without febrile reaction and followed by change in the electrical phenomena, may be recognized by the initial pain which usually attends it, by the disturbance of sensation, and the subsequent progress of the case, even partial recovery being much slower and more tedious. The history of the case ought to lead to a correct diagnosis from other diseases.

Prognosis.—The prognosis, so far as life is concerned, is favorable. Complete recovery of motion in the paralyzed limbs can be expected only in very mild cases, and even in such, more frequently than not, when the child is tired there will be a slight awkwardness in using the affected limb. Generally the recovery is imperfect. After five or six months all is gained that can be expected. If proper treatment is pursued, a much larger amount of motion can be recovered than when the child is left without treatment.

Nothing can be done to prevent retardation of growth, but deformities resulting therefrom may be at least partially prevented by mechanical appliances.

Treatment.—In the first stage the fever may be combated with the ordinary measures, and, if a diagnosis can be made out early, ergot, belladonna, and iodide of potassium may be given in rather large doses; coun-

ter-irritation is also indicated, dry cups and the actual cautery being the most desirable forms; as Seguin suggests, tincture of iodine and blisters cause too much pain and make the skin sore.

After the fever has ceased, and in cases where there is no fever, when time enough has elapsed to guard against unfavorable reaction, electricity should be used. The galvanic current should be used from the beginning, even if the muscles respond to the faradic current; the positive pole or anode should be placed on the spine near the origin of the nerves leading to the affected muscles; the negative pole, cathode, should be passed slowly over the muscles, or, the motor point for each muscle having been found, the cathode may be placed there, and the current may be slowly interrupted. Only such strength of current need be used as will cause perceptible contraction in the muscles. The application should be made every day or every other day for about one minute to each muscle. The reaction of degeneration may appear while the electricity is being used, if its use is commenced early.

In order to obtain benefit from the use of electricity, it must be continued for months; a short treatment of a few weeks, except in very mild cases, would probably be of little value. To obtain the best results, it should be used as soon as possible after all symptoms of irritation have ceased.

With children, a little caution may be necessary in order not to frighten them at the beginning by the novelty of the application; also a very mild current should be used until they are accustomed to the peculiar sensations.

Bathing the affected limbs in hot water is of advantage, allowing them to remain immersed for several minutes; then the limbs should be rubbed and kneaded for several minutes. The warmth of the limbs must be maintained by proper clothing or other means. Over-exercising of the limbs should be avoided; bath-

ing and rubbing should not follow too closely after the use of electricity.

After all hopes of further improvement have to be laid aside, much assistance can sometimes be given by orthopædic surgery and mechanical appliances.

Except in the first stage, no advantage can be gained from the use of medicines internally. Hypodermic injections of strychnia into the affected muscles have been recommended; but it is very doubtful whether more can be gained in this way than by the persevering use of electricity.

CHRONIC ANTERIOR POLIOMYELITIS.

Attention has been called within only a few years to a form of atrophic paralysis which closely resembles acute poliomyelitis, yet is, in some respects, different, the attack being less abrupt, the symptoms not exactly the same. Duchenne, in 1872, and Erb, in his volume on the spinal cord, in Ziemssen's "Cyclopædia," in 1877, gave detailed descriptions of the disease, under the name of *poliomyelitis anterior subacuta* or *chronica*. Since then many cases have been reported, some authors preferring to call it *subacuta* rather than *chronica*. As in regard to several other forms of nervous diseases, it seems that it was only necessary to have attention called to its peculiar symptoms in order that many cases might be recognized.

E. C. Seguin has given a very careful study to both the acute and subacute or chronic form of lesion of the anterior cornua.

When the cases included by different authors are compared, it will be seen that they are simply cases of chronic myelitis in which the anterior cornua, especially its nerve-cells, are affected early in the course of the disease so as to give a special physiognomy to the symptoms. Except for some peculiarities in the course of the symptoms, it would not be desirable to give these cases a separate designation.

ÆTIOLOGY.—We must confess our ignorance of the cause of the disease in many cases; in other instances chronic lead-poisoning seems to be the chief if not only cause; certainly in several patients whom I have seen with this assemblage of symptoms there was lead in the system. Injuries and chills may be the starting-point in this as in other forms of myelitis. Most of the patients are adults. I have, however, seen one child twelve years old with the disease, and an infant seemed to have at first acute poliomyelitis, but later the disease followed a chronic course. It may occur in aged persons also, though rarely.

PATHOLOGICAL ANATOMY. — Very few autopsies have been made—only five or six. In nearly all of these, other parts of the cord than the gray anterior cornua were diseased. In a case reported by Bäumler, the only change found was in the gray substance. There is atrophy and destruction of the cells of the anterior cornua; the nerves and muscles undergo corresponding degeneration. Other portions of the cord than the cornua may also be affected, and thus would be explained variations in symptoms, the chief lesion, and that which gives its name to the disease, being, however, constant; the other lesions are variable.

SYMPTOMS.—The more marked features of the disease are found among the motor functions. The patient finds a difficulty in following his usual occupation on account of weakness, which may be noticed first in the lower or upper extremities, usually the former. After a longer or shorter time this weakness increases until there is paralysis; sometimes one limb alone is attacked, or both arms or legs may be; at length the paralysis extends to those limbs not previously affected. The reflexes are diminished and lost in proportion to the severity of the paralysis. Inco-ordination is rare.

When time enough has elapsed, the affected muscles will be noticed to have wasted, unless, as occasionally happens, the increase of fat conceals the atrophy, or a

sclerotic degeneration of the muscular fibers enables them to keep their size while losing their distinctive muscular character; they will then be felt as hard resisting masses.

The electrical reactions show the changes due to degeneration of nerves and muscles. These changes will depend somewhat upon the progress of the disease and the amount of structural changes. Yet very careful observations made by Kahler and Pick show that there is no absolute relation between the electrical reactions and the loss of voluntary power. The muscles may show reaction of degeneration, yet may contract under the influence of the will. In the case recorded by Kahler and Pick there was a steady diminution of the faradic contractility, until nearly all the muscles of the limbs, body, and face were thus affected, even while the voluntary control was improving. This anomaly is the more frequently seen during restoration of the motor functions.

The progress of the disease may be arrested at any period; then, after an interval, either the symptoms may be aggravated or, more frequently, the power may slowly return; occasionally the recovery is complete, more frequently it is only partial.

The above are the constant and essential symptoms. Other phenomena depend upon what other parts of the cord are diseased.

When the pain is severe, there is probably a limited meningitis. It is more common to have soreness of the muscles, especially before any aggravation of motor symptoms. Numbness, tingling, and a sense of weariness may precede and accompany the earlier motor disturbances. Cutaneous sensibility is, as a rule, only slightly diminished, and is often not affected.

Patients are rarely seen at the very commencement of the disease, so that the symptoms at that period are only occasionally studied; fever is sometimes present, but is not very marked. The limbs which are para-

lyzed, and yet more if atrophy has set in, are generally cold and may be cyanotic.

The bladder and rectum are rarely affected. There are no bed-sores. The ordinary functions of digestion are not disturbed. Cerebral symptoms are absent, except, in a few instances, nystagmus.

DIAGNOSIS.—It is scarcely necessary to recapitulate the symptoms in acute anterior poliomyelitis for the sake of diagnosis.

It is quite probable that formerly chronic anterior poliomyelitis was confounded with progressive muscular atrophy. The latter is more slowly progressive; the paralysis or weakness appears after the atrophy or about the same time; the reaction of degeneration is wanting or is much less clearly marked; the reaction to both the faradic and the galvanic currents decreases, though the latter may persist longer than the former. In the progressive atrophy certain muscles are attacked by preference, and there is rather an irregularity in the progress of the affection; it seems to jump from one region to another, leaving intermediate muscles unaffected. There is less likely to be remissions or cures; the reflexes persist.

PROGNOSIS.—A large proportion of the patients either regain a certain amount of motor power, or, more rarely, recover. The tendency is toward remission.

The course of the disease is long and slow, and years may elapse before it can be said that all the gain possible has been made.

A few cases of death have been recorded, showing that life is not always spared. Of course, if the disease affects the medulla oblongata or the respiratory centers, the prognosis must be unfavorable.

TREATMENT.—When lead seems to be a cause, iodide of potassium should be given at once. If, after a week's use of that drug, lead can be found in the urine, it should be continued for months. Some advise large doses in order to eliminate the lead quickly. The advantage is

doubtful; it is frequently necessary to be cautious lest symptoms of acute lead-poisoning should appear. Sometimes one or two grains are as much as can be given.

Early in the disease, or when there are exacerbations, ergot and belladonna may be given as in acute myelitis.

If the pain is severe, morphia or atropia may be necessary, or the galvanic current may be found sufficient to relieve the pain.

The galvanic current should be applied to the spine even early if there is no fever; positive pole above, negative below. A current from six or eight cells, such as will not cause discomfort, can be used daily, or every other day, for five minutes at a time. Later the paralyzed muscles should be stimulated to contraction by the direct application of the electrode to them, the current being interrupted. The faradic current is less efficacious.

Counter-irritation to the back, blisters, dry cups, or, better than either, the actual cautery, may be of great benefit.

Rest in bed while the disease is advancing is desirable. If its progress is slow, it may not be necessary, however, to stay in bed all the time. As the muscles are regaining power, care should be taken not to overtax them by too prolonged or too severe use.

Massage and warm bathing, not too warm, are useful adjuncts as a means of maintaining the nutrition of the limbs.

CHAPTER XVII.

PROGRESSIVE MUSCULAR ATROPHY.

FRIEDREICH, N., Ueber progressive Muskelatrophie. Berlin, 1873.—CHARCOT et GOMBAULT, Note sur un cas d'atrophie musculaire progressive spinale protopathique. *Arch. de physiol.*, 1875, p. 736.—STURGE, ALLEN. *Lancet*, May 21, 1881, p. 828.—FOX, A. W., Case of Progressive Muscular Atrophy with Bulbar Paralysis. *Brit. Med. Jour.*, Jan. 15, 1881, p. 82.—CLARKE, J. L. *Arch. of Med.*, London, 1863, p. 1.—*Med. Chir. Trans.*, 1873, p. 103.

ZIMMERLIN, FRANZ, Ueber hereditäre (familiäre) progressive Muskelatrophie. *Zeitschr. f. kl. Med.*, vii, 1883, p. 15.—ERB, W., Ueber die juvenile Form der progressive Muskelatrophie. *Deut. Arch. f. kl. Med.*, xxxiv, 1884, p. 467.

Three theories have been advocated in regard to the nature of this disease: That it is primarily a muscular affection, the changes in the nervous system being secondary; that it is a disease of the sympathetic system; that the seat of the disease is in the spinal cord. The question as to which of these is the correct theory has not yet been settled, and I have not the data upon which to form a final opinion. I give it a place among lesions of the spinal cord because constant changes have been found in the cord, and because it seems useful to place it alongside of other affections which somewhat resemble it.

ÆTIOLOGY.—As the first symptoms appear generally in those muscles which are most used, it is probable that excessive use acts as one cause, at least as the cause for localizing the disease at the beginning.

Men are more frequently attacked, and middle adult

age is the most favorable for the development of the disease.

At least sometimes heredity seems to be an important ætiological factor.

There is a close resemblance between progressive muscular atrophy and pseudo-hypertrophic paralysis, in that brothers are frequently attacked, and the disease seems to be transmitted through the females of a family.

Pathological Anatomy.—Unfortunately, it is impossible to give a satisfactory account of the post-mortem changes connected with the nervous system, because in many cases the examination has been imperfect.

The spinal cord has been frequently found either normal or with very slight changes, not always affecting the cells. In some of the cases where there was no change, the question has been raised whether they were cases of progressive muscular atrophy.

In a large number of cases the cells of the anterior cornua have been found diseased, atrophied.

In a very few cases, comparatively, the sympathetic has been found diseased. In many more it has been examined and no disease found. In a large number of cases, even where the cord has been healthy, the sympathetic has not been examined.

The weight of evidence to the present time is in favor of the seat of the disease being in the cord; but it is necessary that more examinations should be made of thoroughly typical cases before the question can be decided.

The changes in the muscles are only occasionally of an active nature; they are rather regressive in character. In many fibers there is a tendency to split up longitudinally; much less frequently they separate transversely. Frequently there is a simple atrophy; sometimes a waxy degeneration or a fatty degeneration can be recognized.

The nuclei of the muscles may be increased quite early, and sometimes the sarcolemma sheath is filled with these nuclei after the contractile substance of the muscular fiber has disappeared. Finally the proper muscular structure disappears, and there remains only a fibrous-like substitute in place of the muscle.

An increase of the interstitial tissue with, or less commonly without, deposit of fat may proceed uniformly with the degeneration of the muscles, or may even advance so rapidly as to give the muscles an appearance of having undergone hypertrophy.

SYMPTOMS.—The atrophy from which the disease takes its name, with the attending weakness and paralysis, is the principal symptom. The wasting is almost or quite imperceptible in its beginning, progresses very slowly, and may have advanced so as to seriously interfere with the use of the limb before it is noticed. The patient is aware, perhaps, for a short time of a slight loss of skill or readiness in his motions, or that he is more quickly tired than usual; then he finds that he is unable to use his hands or arms with natural ease and strength; then notices the change in configuration, and may think the whole has occurred within a few hours; yet, on closely questioning him, it will be discovered that several weeks at least have elapsed since the first slight symptom appeared.

A large majority of patients are first attacked in the hands or arms; usually the muscles of the thenar or hypothenar eminence, the interossei and lumbricales, are the first to undergo atrophy, and the right hand or arm is much the more frequently affected first. Authors disagree as to whether the atrophy attacks the interossei or the muscles of the thenar eminence first. The position of the fingers is peculiar when the disease has made considerable progress. There is the claw-shaped hand. The thenar eminence is thinned, leaving a flattened, slightly concave surface in place of the normal convex swelling of the ball of the thumb.

Next to the small muscles of the hand, the flexors and supinators of the forearm, or the deltoid, are attacked; then other muscles connected with the scapula and those of the trunk, and finally the muscles of the lower limbs, may be affected. The progress of the disease from muscle to muscle observes no regular order, but muscles widely separated may be attacked before the intermediate muscles suffer.

Generally both sides are affected nearly at the same time, though often several days or weeks elapse before the second side is attacked. The rate of progress differs very widely; there may be long intervals when the symptoms remain stationary.

Very rarely the disease may begin with the lower limbs—either in the thighs or the legs—though Eulenburg thinks this form occurs only among children in a form allied to pseudo-hypertrophic paralysis.

As the power of motion is not lost until the muscles have almost entirely disappeared, the patient may be able to move his limbs even when there is great emaciation. When the atrophy has advanced far, so that a large number of muscles, both those of the limbs and of the trunk, are greatly wasted, he presents a pitiable appearance; especially if the facial muscles have also been attacked; he becomes a "walking skeleton," the bones apparently only covered by the integument.

Even before a muscle is seen to waste, and while that process is going on, very slight and rapid contractions of individual muscular fibers can be seen, which, occurring repeatedly in the same or adjoining bundles of fibers, produce what is called *fibrillary contractions*. These may be compared to the fitful flashing sometimes seen as the light of the aurora spreads over the sky. If these contractions do not appear spontaneously, they may be excited by giving the muscles a fillip with the finger or a pencil. By observing this phenomenon in muscles not yet wasted, it is often possible to foretell which will be next attacked.

The electrical reaction of both nerves and muscles is diminished in proportion to the amount of atrophy. The galvanic current will cause contractions longer than the faradic current. Rosenthal says that the nerve-filaments nearest the centers may react normally, while the peripheral ramifications may show diminished reaction.

Remak discovered that when the negative pole is placed over the fifth or sixth cervical vertebra, and the positive pole is placed on the side of the neck in the carotid fossa, or in the triangle between the lower jaw and the ear, there follow contractions in the atrophied muscles on the side opposite that where the positive pole is when the current is interrupted. This reaction is most easily shown with the galvanic current, but it is not constant. If both poles are placed on the side of the neck, with a weak current, the contraction may be excited on both sides. Remak called this "diplegic contraction." He referred it to a reflex contraction excited through the medium of the superior cervical ganglion. Others do not agree with this view.

Sensibility is usually not affected; even when disturbed, the change is very slight. Pain, however, in the affected muscles is not uncommon; this pain may be excited by motion or by pressure, and it is sometimes spontaneous. Cutaneous reflexes are sometimes heightened, especially in the early stages. The patellar tendon reflex has been found present in a case where the muscles of the legs and thighs were not atrophied; in another case slightly diminished. It is sometimes exaggerated, its strength depending upon the state of the muscles which contract in response to the stimulus.

The temperature of the affected limbs is sometimes at first moderately elevated, but later it is lowered. The joints may be swollen and painful, more particularly the smaller joints. There may be bed-sores toward the close, unless some intercurrent disease short-

ens life. Occasionally herpes and changes in the hair and nails are to be noticed.

The pupils may not be symmetrical, and there may be a variation in their relative size, but in very many patients there is no deviation from the normal condition.

The disease slowly advances, with occasional pauses, more muscles being invaded, until the patient is helpless. The duration may extend over two to twenty years. Finally respiratory muscles, or those of deglutition, are affected, and the patient dies.

Erb describes a special form of muscular lesion, which he calls the "juvenile form of progressive muscular atrophy," consisting in part of hypertrophy with subsequent atrophy, with greater or less formation of fat tissue and increase of interstitial connective tissue. The same sets of muscles are attacked in different cases, especially the pectorales, cucullares, latissimi, flexor group on the arm, triceps; forearm and hand are not affected; in the lower limbs, those of the thigh, the peroneal region, and the calf are attacked; also the lumbar extensors. There is no fibrillary tremor nor degenerative reaction. The disease begins in childhood or youth, and may be mistaken for progressive muscular atrophy or pseudo-hypertrophy.

Prognosis.—There is no immediate danger to life unless bulbar symptoms or disturbed respiration set in, but the prospect of cure is very slight. When seen early, it may be that the disease can be checked and even muscles restored; but this is very rarely the result. As a rule, there is before the patient only a life of gradually increasing weakness, ending in total disability, which may be prolonged through years.

Diagnosis.—The disease which has been most frequently confounded with progressive muscular atrophy is poliomyelitis—anterior, subacute, or chronic. The commencement and progress of the disease are much

more gradual in the former, the sensation is less frequently disturbed early in the disease, the electrical reactions are different, the paralysis is more proportionate to the atrophy, except when there is increase of fat, and the irregular order in which the muscles are attacked is quite characteristic. In many cases of the chronic anterior poliomyelitis, lead is found to be a factor in causing the disease; this has not yet been recognized in progressive muscular atrophy, though it was found in one of my patients.

Local injuries to both muscles and nerves may simulate progressive muscular atrophy, and wasting of muscles in consequence of joint disease may lead at first to doubt as to diagnosis, but careful study of the history and of all the circumstances will probably guard one from mistake.

The local paralysis of the hands from lead, the wrist-drop seen in that affection, can be recognized by the fact that the extensors are chiefly affected, and the interossei and the muscles of the thenar eminence escape; the fact of exposure to lead, previous lead colic, the lead cachexia, and the condition of the blood, would aid in diagnosis. Yet once in a great while a case may occur which will require great care to decide correctly.

TREATMENT.—Internal remedies are not likely to be of much value. If, however, lead can be detected in the system, an effort should be made to eliminate it by using iodide of potassium.

Electricity is of value—either the faradic or galvanic current—applied locally, so as to cause muscular contractions; that current is to be chosen which will most readily cause the muscles to contract; sometimes a very strong current will be needed. The galvanic current, of moderate strength, may be applied to the spine at the same time.

Exercise should be restricted within the limits of fatigue. Patients are mistaken in supposing that by taking much exercise they can restore the strength of

diseased muscles. It is necessary to caution them on this point. When the muscular power is much reduced, and even before, Swedish movement is of value. Massage should be used from the beginning where it is practicable.

These means for help should be used perseveringly through many months before discontinuing treatment. No benefit can be expected from a short treatment.

CHAPTER XVIII.

BULBAR PARALYSIS (LABIO-GLOSSO-LARYNGEAL PARALYSIS).

KUSSMAUL, A., Ueber die fortschreitende Bulbärparalyse und ihr Verhältniss zur progressiven Muskelatrophie. *Volkmann's kl. Vorträge*, No. 54, 1873.—STRÜMPELL, ADOLF, Zur Casuistik der apoplektische Bulbärlähmungen. *Deut. Arch. f. kl. Med.*, xxviii, 1880, p. 43.—BEEVOR, C. E., Case of Glosso-labial Paralysis with Progressive Muscular Atrophy and Lateral Sclerosis. *Brain*, Oct., 1882, p. 403.—LEYDEN, E., Zur progressiven Bulbärparalyse. *Arch. f. Psych. u. Nervenkr.*, ii, iii.—FINNY, J. M., Clinical Remarks on Cases illustrating the Essential Identity of Progressive Muscular Atrophy and Progressive Bulbar Paralysis. *Brit. Med. Jour.*, June 14, 1884, p. 1132.—BENNETT, A. H., Bulbo-spinal Atrophic Paralysis. *Brit. Med. Jour.*, March 8, 1884, p. 647.—EISENLOHR, C., Ueber acute Bulbär- und Ponsaffectionen. *Arch. f. Psych. u. Nervenkr.*, ix, p. 1, x, p. 31.—ROSS, JAMES, Labio-glosso-pharyngeal Paralysis of Cerebral Origin. *Brain*, 1882, p. 145.—KIRCHHOFF, Cerebrale Glosso-Pharyngo-Labial-Paralyse mit einseitigem Herd. *Arch. f. Psych. u. Nervenkr.*, xi, 1880, p. 132.

BULBAR PARALYSIS.

Duchenne first called attention to the combination of symptoms which are known as bulbar paralysis under the name of glosso-labio-laryngeal paralysis. There is paralysis of the muscles with atrophy of the tongue, of the soft palate, of the lips, of the pharynx and larynx; muscular atrophy may also extend to other regions, until with the symptoms of bulbar paralysis there are united those of progressive muscular atrophy.

PATHOLOGICAL ANATOMY.—Muscles undergo the changes which are found in other cases of atrophy. The nerves, especially the hypoglossal, facial, and ac-

cessory, exhibit the usual appearances of fatty degeneration, such as are found in other cases of muscular atrophy of central origin.

The medulla oblongata seems to be the primary seat of the disease. Often nothing abnormal can be recognized with the naked eye; but under the microscope changes will be discovered in the motor nuclei of the medulla. The ganglion nerve-cells undergo degeneration and atrophy either with or without disease of the surrounding tissues. The nuclei of the hypoglossal, accessory, vagus, and that part of the facial nucleus connected with its inferior branch, are most frequently affected; the nucleus of the glosso-pharyngeal nerve is less frequently affected. When the disease spreads so as to implicate the muscles of the extremities, the corresponding parts of the spinal cord will also be found affected.

ÆTIOLOGY.—Of the causes of bulbar paralysis we know almost nothing. It is confined almost entirely to advanced life, and is more frequent among males; but it has been seen as early as twelve years; Erb saw it in a girl of twenty years. Syphilis, exposure to cold, and injuries have been mentioned as causes.

SYMPTOMS.—As with most chronic diseases of the spinal cord, the earliest symptoms are so insignificant as to be often overlooked or neglected.

They vary in different cases according as the nucleus of one or another of the nerves of the medulla oblongata is chiefly or primarily affected. The hypoglossal nucleus generally suffers first, and in the majority of cases the first motor disturbance is noticed in the tongue. The patient is not able to move his tongue quite as freely as normal, and his articulation becomes imperfect. Some letters can not be readily pronounced; *ē* is first lost, then there is trouble in expressing the sounds *r*, *sh*, *s*, *l*, *k*, *g*, *t*, and later *d* and *n*; the motions of the tongue, apart from speech, are interfered with, and it can not be protruded beyond the teeth;

the affection being bilateral, it is not protruded to one side, it is not moved forward, it can not be turned in the mouth to loosen food from between the gums and cheek, and it can not be formed with a trough-like depression in the center; the tip can not be raised against the upper teeth, and the center or root of the tongue can not be arched to touch the hard and soft palate; it can not be used to press food backward in the first act of deglutition.

When the nucleus for the lower branch of the facial nerve is affected, the lips act less readily, and, if the disease begins thus, the letters in which the lips chiefly act are first pronounced indistinctly; if the lips are affected later, then the power to pronounce those letters is lost later; these letters are *o* and *u* first, later *ē* and *ā;* of the consonants, *p* and *f;* later *b*, *m*, and *v* are lost. When the lips can not be readily moved, there is much difficulty in keeping food in the mouth while eating; the saliva runs out of the mouth.

When the soft palate is paralyzed, which is only after the tongue or lips have been affected, the voice acquires a nasal tone, and the explosives can not be clearly pronounced, especially *b* and *p*. If the loss of power in the soft palate is considerable, drinks will return through the nose, and, if it is extreme, even solid food will thus return. The glottis is not properly closed, and therefore food, or more especially drink, enters and excites paroxysms of coughing. The patient gradually gives up drinking and tries to swallow only soft solids.

The patient's countenance acquires a characteristic expression; from the paralysis of the lower branches of the facial nerve the mouth and lower part of the face are motionless; the lower lip is dragged down and rolls outward from its own weight; the saliva acquires a more tenacious character, and, not being swallowed, collects and flows out at the corners of the half-open mouth. The tongue can not be protruded nor moved,

but lies on the floor of the mouth, atrophied and shrunken, constantly agitated by fine fibrillary tremors. The superior branch of the facial nerve is not paralyzed, and hence the forehead and eyelids move naturally. The ocular muscles are unaffected.

Sensation is not disturbed, nor are the special senses, taste remaining intact.

The disease advances slowly, sometimes with remissions, but as a rule steadily, until the patient at last is a pitiable spectacle, talking with extreme difficulty, perhaps unable to make himself understood; intelligent, conscious of his condition, with bright and speaking eyes, he eagerly desires the food which he can not swallow, or, if he tries laboriously to swallow, is nearly strangled by choking. He necessarily becomes weak and emaciated from lack of nutrition; but, apart from this cause of debility, it is not infrequent that the muscles of the extremities and even of the trunk suffer atrophy, as the disease of the motor cells extends to those in the anterior cornua of the cord; thus general muscular atrophy is added to the local. The arms and neck are usually first the seat of this change.

The pulse is sometimes irregular or rapid, and there may be dyspnœa, especially toward the close of life; coughing and sneezing become impossible.

Life may be cut short by disease of the lungs, excited by the entrance of food into the bronchi, or by suffocation during an attack of dyspnœa, or more slowly by starvation.

The atrophy and paralysis are attended with changes of electrical reaction in the affected muscles, such as are found in progressive muscular atrophy. The faradic reaction may seem to have suffered little, as many muscular fibers remain without atrophy, though at a late stage it will be diminished; but the galvanic current will show the reaction of degeneration when the disease has advanced somewhat.

Not only the tongue, but the other affected muscles,

may be the seat of fine fibrillary tremors, such as are seen in progressive muscular atrophy.

NATURE OF THE DISEASE.—The nature of the atrophy and paralysis in bulbar paralysis, and its relation or connection with progressive muscular atrophy, has been the subject of controversy. In both diseases there is the same combination of symptoms, making allowance, of course, for the difference of function in the different muscles. There is diminished skill or facility in the execution of movements, a slight paralysis, which gradually increases in degree; at length atrophy is noticed, but not until so much of the muscular structure has degenerated as to cause the loss of function to be prominent; fibrillary tremor; change of electrical reaction when so much of the muscle is affected that the change can be recognized; the disease progresses to finally almost entire and absolute paralysis; in both, sensation is very rarely disturbed; the destruction of the large motor cells in the nuclei of the cranial nerves and the anterior cornua in the cord is the same in nature, so far as our means of examination enable us to judge.

Besides these points of correspondence, the two diseases run into each other. Before bulbar paralysis ends in death, it is usual to see the limbs affected with muscular atrophy; and often, in progressive muscular atrophy, bulbar symptoms appear near the close of life.

The two affections may, then, be justly looked upon as pathologically one disease, as clinically distinct only because of the wide difference in the function of the parts innervated by the regions affected in the cord and medulla.

In very many cases, at the autopsy it has been found that the pyramidal tracts in the lateral and even in the anterior columns were altered. In some of these cases the exaggerated reflexes of lateral sclerosis were recognized during life. It is as yet doubtful whether this is

to be considered as a secondary degeneration and complication, or whether the few cases of this character are to be classed as amyotrophic lateral sclerosis.

DIAGNOSIS.—Especially in the early stage it will require a careful examination of all the symptoms to recognize the disease; similar symptoms may be seen in the early stages of general paralysis; then the variation in pupils, the general weakness of the limbs, the tremor, and the mental condition of the patient, may aid in diagnosis, but sometimes it will be necessary to wait for further developments. When the disease is well advanced it can hardly be mistaken, yet, if a patient is seen in an advanced stage, without having a knowledge of the history of the origin and progress of the symptoms, it may be very difficult to decide whether it is the chronic or acute bulbar paralysis.

PROGNOSIS.—There is no case on record of recovery in uncomplicated, primary bulbar paralysis. All the instances of recovery, if carefully examined, will be seen to have presented bulbar symptoms as secondary only. The prognosis is, therefore, unfavorable.

When the significance of the peculiar early symptoms is generally recognized in the profession, so that treatment can be commenced early, a more favorable result may be obtained.

Remissions of longer or shorter duration are not uncommon, as in all chronic and slowly progressive diseases. The duration of the disease is only a few years; Erb says from one to five.

TREATMENT.—It is certainly rather discouraging to consider that no treatment has as yet been successful. Electricity is the most promising, and may, for a short time at least, contribute to the comfort of the patient; it may render swallowing less difficult. The galvanic current should be used. It may be passed from one mastoid to the other, keeping the electrodes in one place; Erb also recommends galvanization of the cervical sympathetic (anode on the back of the neck, cathode

at the angle of the lower jaw). The electrodes may be applied to the sides of the neck so as to excite the muscles of deglutition, and to stimulate the recurrent laryngeal nerve, using the faradic and galvanic current alternately. As the muscles are weak, having undergone partial atrophy, they are easily tired, therefore the application should be short—three to five minutes; this may be repeated daily, or every other day. The treatment by electricity should be persevered with for several months.

Counter-irritation, by means of dry cups, blisters, or cautery, has been recommended; also hydrotherapy is advised. All these means may be employed, at least with the advantage of making the patient more contented.

Erb says that medicines taken internally have never produced the very faintest effect. Still, he recommends the trial of nitrate of silver, iodide of potassium, iodide of iron, chloride of gold and sodium, ergotin, belladonna, etc.

The most important part of treatment is the general care of the patient. The general health is to be cared for by regulating the habits; too prolonged exercise fatigues and exhausts, therefore injures the patient, especially after the muscles of the limbs are affected. If efforts to converse are continued too long, they exhaust the muscles of the throat. The food should be soft, so as to require very little mastication, and because soft solids are most readily swallowed. The patient should be fed slowly, time enough being allowed for the partially atrophied muscles to rest. It may be well to increase the number of meals during the day. Finally, it may be necessary to feed the patient through a tube introduced into the stomach.

When dyspnœa, or, as patients sometimes describe it, "attacks of asthma," occur, sedatives, narcotics, and stimulants, internally or by inhalation, or subcutaneously, must be used.

ACUTE BULBAR PARALYSIS.

Many cases have been reported in which the symptoms of bulbar paralysis have appeared suddenly, without the progressive character. Such cases may be considered as acute, and may be so called if we keep in mind that they are due to pathological changes quite different from those found in the progressive disease.

ÆTIOLOGY. — The causes of the combination of symptoms in these acute cases are: softening due to plugging of vessels by a thrombus or an embolus; hæmorrhage; acute inflammation; tumors; and, in a few cases, cerebral lesions. The causes of the acute form would then be those that would give rise to the above pathological conditions.

SYMPTOMS.—The symptoms vary considerably from those found in the chronic cases. The onset of the disease is sudden, the paralysis reaching its height in a few hours, or at most a few days. Convulsions of an epileptiform character may be among the earlier symptoms. After the first attack there may be a slight improvement or a remission, and afterward a steady progression in the disease.

Besides the bulbar symptoms due to lesion of the nerves of the medulla or their nuclei, there will probably be also a rapidly occurring paralysis of the limbs, and sensation may be affected, as it is not in the chronic form. The paralytic symptoms may extend so as to show that parts anterior to the bulb are affected; also the paralysis of the limbs may be unilateral and alternate—i. e., on the side opposite to that on which the cranial nerves are affected. In cases of acute inflammation of the medulla, such as has been reported by Leyden, the symptoms appear less suddenly than in hæmorrhage or in occlusion of vessels, but they are developed in a comparatively short time, and other than bulbar symptoms appearing with them will aid in forming a correct diagnosis.

Several times clearly marked bulbar phenomena have been seen where the lesion was entirely cerebral; this has been noticed where the lesion has been multiple, affecting both sides of the brain: but bulbar symptoms have also been found where the cerebral lesion has been unilateral.

The diagnosis of the nature of the process giving rise to acute bulbar paralysis must be made from a consideration of the circumstances attending the attack: on the same principles as will aid in forming a diagnosis in other cases of cerebral lesion. These need not be reviewed here.

The prognosis of acute bulbar paralysis is not so serious as of the progressive. Eisenlohr has reported recoveries in several cases. Strümpell reports a case of recovery. Erb reports favorable results in a case of seven months' duration.

The insidious, unpretending, progressive disease, which seems so much milder, and at first insignificant, is much the more dangerous and fatal.

The treatment must depend upon the nature of the pathological process. Iodide of potassium is indicated if there is reason to suspect an occlusion of arteries, especially if there has been syphilis; also if there is possibly a syphilitic thickening of the membranes. Erb passed a current from eight cells through the head from one mastoid process to the other; and also galvanized the cervical sympathetic.

The nerve-cells of the anterior cornua are degenerated in those cases where muscular atrophy occurs; they have also been found affected in several cases of articular and osseous lesions.

ÆTIOLOGY.—By far the larger number of patients are men, comparatively few being women; children are only rarely affected. The age at which the first symptoms are generally noticed is between twenty-five and forty-five years. Heredity is thought by some to act as a predisposing cause; excess in the use of alcoholic liquors, and acute diseases, are also predisposing causes.

Exposure to cold, especially to cold combined with wet, is one of the most frequent exciting causes. Overexertion, excessive labor, especially on the feet, are other frequent causes of the disease; hence, the larger number of patients are to be found among those whose occupation requires them to stand or walk nearly all the time, whose feet are much exposed to cold and wet.

Lately the connection of syphilis with locomotor ataxia has attracted much attention. A very large proportion of ataxics are found to have had syphilis in early life, more than half of these to have had secondary symptoms; very many have never had these. Erb assigns much importance to this relation between the two diseases. Gowers seems to count syphilis as a predisposing cause. "It seems," he says, "that one effect of constitutional syphilis may be to induce a neuropathic state in which certain degenerative diseases of the nervous system readily occur." The proportion of ataxics who have had syphilis seems to be less in America than in Europe.

Whatever connection there may be, or if there is none, it is an interesting fact that from forty-five per cent to eighty-eight per cent of ataxics are found to have had syphilis.

Venereal excesses are generally considered as one of the causes of locomotor ataxia. Many times such ex-

cesses must be reckoned among the early symptoms of the disease.

Injuries, as jar or concussion of the spine, have sometimes seemed to be the cause of ataxia. Cases in which such a relation can be traced are, however, rare.

SYMPTOMS.—The earliest and most extensive changes in the cord are found in those regions which are active in transmitting sensation; the earliest symptoms of the disease are perverted sensations; and, as the changes are found throughout the whole length of the cerebro-spinal axis, commencing at any level, it may be expected that the symptoms would show a great variety both in the beginning and during the course of the disease. This is the case.

The earliest symptom is pain, which is thought to be rheumatic or neuralgic, and no inquiry made as to its nature or cause. In most cases the pain is in the legs or feet. If the upper part of the cord is first affected, it may be first felt in the arms, or may follow the course of the occipital nerve over the back of the head; once in a while the fifth nerve is affected, and the pain is felt in the face. The pain is peculiar in character; it is of a stabbing, cutting nature, deep-seated rather than superficial, as if in the muscles or bones; each stab is of only momentary duration, and is no sooner felt than it is gone; these darts of pain succeed one another in rapid succession, or may be separated by a short interval.

The attacks may continue several minutes or hours, commencing without warning, and as suddenly ceasing. At first they are not of frequent occurrence, but as time goes on they recur at shorter intervals. Sometimes an aching or tired sensation is noticed between the throbs of pain, which gives the attack a resemblance to rheumatism; but the severe pain is quite different from a rheumatic pain: it occurs entirely independent of any movement of the limbs; the aching between the attacks is less than is usually found in

rheumatism with equally severe pain; the part affected is neither red nor swollen; the joints do not so frequently suffer as other parts of the limbs. The unexpected occurrence of the pain, and its severity, may cause the sufferer to cry out and grasp the part affected. The terms used by patients are expressive; they speak of the pain as resembling a "stroke of lightning," "a knife-thrust into the bone," "a red-hot iron suddenly buried in the flesh," though the pain does not always have this severity. The seat of the suffering varies; the pain may be in the foot, again in the calf or thigh, sometimes in the right leg, again in the left, but generally it does not give a preference for one side more than the other; also in the arms and head the different attacks affect different localities, yet, as a rule, localities supplied by the same plexus of nerves.

So little importance is given to the above symptom when first present, that medical advice is rarely sought until the attacks become more severe. Let the physician be on the watch to inquire into the particulars, and ataxia will be oftener recognized in its earliest stage; other symptoms will be found confirming the diagnosis.

During the continuance of the pain, and for a short time after the attack has ceased, there will be a tenderness of the part affected; a very light touch will be painful, yet a severer irritation, as a prick with a needle, may be scarcely felt.

The hyperæsthesia is of short duration; anæsthesia is found to be more persistent, and between the attacks there is diminution of sensation of touch as well as of pain. The anæsthesia often affects the soles of the feet; it may be found in circumscribed spots on the limbs or body, on the face or head; its distribution is independent of the seat of the pain; it is generally symmetrical. Localized anæsthesia is as constant a symptom as the pain, but is less strikingly evident to the patient, and may need to be searched for carefully

by the physician. The different forms of sensation, as touch, pain, temperature, may be affected separately, and so each should be tested.

Delay in the transmission of sensation, so that a touch or prick is not felt for several (three to ten or fifteen) seconds after contact, is less frequently an early symptom, but is often to be noticed later. Sometimes the touch is first recognized, the pain is felt later, showing a difference in the rate of transmission of the two varieties of sensation.

A sense of a girdle around the body is often noticed.

The muscular sense is not impaired till late in the disease. The patient can tell where his limbs are, even when there is much disturbance of motion; but finally he loses this power.

The special senses may be disturbed early in the disease, even before any disturbance of general sensibility. Vision is affected much more frequently than hearing; taste and smell are very rarely lost.

The sight may be diminished by reason of disturbance of accommodation, by paralysis of motor muscles, or by limitation of the field of vision.

The field of vision is limited because of atrophy of the optic nerve. The limitation, often unilateral at first, soon affects both eyes; it begins on the temporal side, and advances so that the center of the field of vision remains longest unaffected. The ophthalmoscope will show the disk with the characteristic appearance of atrophy. If the change is recent, it may be possible to exclude a preceding neuritis; if old, the neuritis can not be excluded with so much certainty. The atrophy and consequent limitation of vision are permanent.

The field of vision for colors may be limited when perception of form is still nearly or quite perfect. This is a condition which has not been very carefully studied as yet.

The atrophy of the optic nerve, and consequent

limitation or loss of vision, may be the only symptom present for several years. If with this there is absence of patella tendon reflex, the diagnosis of ataxia may be made with a fair degree of certainty.

It may be as well to mention here the motor disturbances of the eyes, as they also interfere with vision. The pupils may be extremely contracted; they may be less contracted or even dilated, and not respond to variations in the amount of light, while they will respond to accommodation. This has been called the "Argyll-Robertson symptom," from its first observer. Buzzard suggests that slight changes in the iris can be best seen by casting a strong light upon the eye with the ophthalmoscopic mirror, and watching the motions through a convex lens of about + 8 or + 10 placed behind it.

Presbyopia is one of the earlier ocular symptoms. The change is not gradual, as when the consequence of advancing years, but, when symptomatic of tabes, occurs suddenly, perhaps in one eye only; after a short time this symptom may disappear.

Diplopia from partial paralysis of some of the external ocular muscles is another form of defective vision; there may also be partial ptosis of one or both eyelids. Rarely nearly all the ocular muscles may be affected. The paralysis of these muscles may pass away and the patient may almost forget its occurrence when he applies for relief from the later symptoms of ataxia.

The hearing may suffer early—one ear may be affected or both ears may be dull of hearing. In one patient, where the hearing was practically entirely lost, partial deafness occurred suddenly, then gradually increased in degree rather rapidly.

Tinnitus aurium is mentioned as a symptom, but is so common under varying circumstances that it would be difficult to prove more than a coincidence.

Visceral disturbances, gastric, nephritic, laryngeal, and bronchial crises are phenomena of disturbed sensation occasionally met in ataxia.

The disturbance called "gastric crises" is the most important of these visceral symptoms. This may be the dominant symptom for years, and so absorb the attention of both patient and physician that other phenomena are overlooked; the patient is thought to have gastric ulcer, nervous dyspepsia, chronic catarrh of the stomach, etc. A close questioning will bring to light "rheumatic pains" in the limbs, an examination may show pupillary phenomena, and tendon reflex will be found absent. The diagnosis can not then be doubtful.

These gastric crises are irregularly periodical, recur without certain cause, and without warning. The patient is seized with severe pain in the epigastrium, as if his bowels were being tied up or twisted around; this continues for a variable time, and then vomiting sets in, at first of ingesta, then of a glairy mucus similar to that ejected in catarrh or ulcer of the stomach. The amount of vomitus may be very great, enormous, and the patient is surprised at the quantity. With the vomiting there is usually a short period of relief, only a slight soreness remaining. Soon the same scene is repeated. These attacks recur at short, sometimes long, intervals, for a few days or some weeks, and then cease for weeks or months.

During the attack the pulse is commonly greatly increased in frequency; but the temperature is not elevated. Sometimes during the vomiting the severe shooting pains in the limbs are felt in their greatest intensity, adding materially to the patient's misery.

Between the attacks the appetite and digestion are good; the former may be ravenous.

Nephritic crises have been described by Raynaud, characterized by attacks resembling renal colic with retraction of testicles, anuria or ischuria, vesical tenesmus, the attacks continuing six or eight days, recurring frequently, separated by intervals of health. There is no blood, nor pus, nor gravel found in the urine. These are much less common than gastric crises.

Laryngeal crises, where there are obstinate and causeless paroxysms of coughing, ending, perhaps, in spasm of the glottis, are even less common. Charcot and Vulpian have recorded cases of laryngeal crises in which epileptiform convulsions occurred immediately after the spasm of the glottis.

The bronchial crises, where there is great dyspnœa, sense of constriction across the chest, as if suffocation were impending, are also very rare.

The absence of patella tendon reflex (Westphal's symptom, as it has sometimes been called) is very constant in locomotor ataxia. In order that this phenomenon may be of value, it is necessary that during the examination the knee should be bare, the leg should hang free, and there should be no semi-voluntary contraction of muscles. In doubtful cases it is well to have the patient shut his eyes and to strike one knee or the other without giving him warning as to where the blow is to fall. The absence of patella tendon reflex may be found in other diseases, but in locomotor ataxia it is associated with considerable voluntary muscular power, and when the vastus internus is smartly filliped, it is seen to contract. Buzzard and Erb both insist upon the need of these conditions as aids in judging of the significance of the phenomenon.

Occasionally patella tendon reflex is present in an undoubted case of locomotor ataxia, and I have seen cases where it reappeared after having been absent. It is absent in about 1·5 per cent of healthy persons.

This is usually an early symptom, and by some authors it is claimed that it may be noticed before any other symptom. It is one of the most constant, as will be mentioned later. Sometimes, however, the tendon reflex does not disappear till after other symptoms have been long present.

Cutaneous reflexes are, as a rule, retained in normal intensity, though when there is hyperæsthesia the reflex excited by irritating such a spot may be exagger-

ated, and, when sensation is markedly delayed, cutaneous reflexes may also be delayed.

Motor disturbances do not belong to the earliest symptoms in locomotor ataxia. The first discomfort in this respect noticed by the patient is a sense of weariness; he is more quickly tired than usual. This may be laid to various causes, and frequently is thought to be one result of the rheumatism which causes the pain.

At a later period there is a loss of co-ordination in the use of the muscles. The patient is aware that he needs to use his eyes in order to walk reasonably straight; or, before he is conscious of this, if examined, he will be found to have lost the control of his limbs, and he can not walk straight with his eyes shut. If in bed, with feet wide apart, he is told to shut his eyes, raise one foot, and slowly carry it across the bed and bring it down by the side of its fellow, the foot is moved irregularly with a jerking motion and is not placed correctly in the place mentioned.

The inco-ordination may affect the arms before the legs, or after. Then the patient has difficulty in feeding himself or in writing or performing other acts requiring delicate manipulation. If, with eyes shut, he is told to touch the end of his nose with his forefinger, the finger will go wide of the mark.

The gait of an ataxic is peculiar; he walks with his feet wide apart, straddling; they come down heavily upon the floor, the heels striking first; the body sways somewhat from side to side; if the inco-ordination is great, support is needed, and then the legs move irregularly.

Inco-ordination is not as constant a symptom as many others, and is much less important than was formerly supposed.

With the loss of complete control over the limbs, and perhaps an inability to walk, there is comparatively little loss of muscular strength; while lying in bed, the patient can move freely, and resist passive flexion or

extension of the limbs with great power. Not until the closing period of the disease is there decided paralysis, except that occasionally a paralysis, which soon disappears, may follow a severe attack of pain.

Sexual desire is sometimes exaggerated, and hence venereal excess may be one of the symptoms rather than one of the causes; but it is probably rather an early symptom of the disease. Frequent emissions are also met. Sometimes sexual desire is greatly increased, but there is entire loss of power to gratify the desire, and, while suffering from it, there is not the slightest trace of erection. This impotence is not one of the earliest symptoms, though it may be noticed by the patient before he has given much thought to the other earlier symptoms.

The bladder may be affected; rarely there is pain with dysuria, and frequent calls to empty the viscus. It is more common to have difficulty in voiding the urine from partial paralysis or anæsthesia, and finally the retention may lead to cystitis.

The bowels are usually unaffected, except a slight constipation; but this may give some trouble. Rarely there are attacks of looseness. When there is great anæsthesia, the fæces may pass without the patient's knowledge.

Trophic changes are not uncommon; among these the affection of the joints is most striking and characteristic. The limb swells from effusion into the joint, which may be very great, and a large part of the limb may be enlarged; there is oftentimes some redness and slight pain, but these are only moderate. Within a very short time it will be found that the articular surfaces forming the joint have suffered loss of substance, and, when the limb is moved in certain directions, crepitus will be felt. After a while the serous effusion is absorbed, and the erosion of the articular surfaces continues if the joint is still used. The surface of the bones is roughened, and their chemical composition

altered; the earthy salts are absorbed, and a great excess of fat is deposited.

The larger joints are most commonly affected—the knee and hip, the elbow and shoulder; but the jaws and smaller joints may be attacked. The chief characteristics of this change are the sudden and rapid effusion into the joint, without fever, and with but little pain, if any; the early erosion of the articular surfaces, also without pain.

The changes in the joints give rise to dislocations, and the bones assume abnormal positions; the destruction of ligaments or their elongation allows the limbs to be hyperextended.

Occasionally osseous growths form in the vicinity of joints in the soft parts, as if an effort were made to compensate for the injury by the formation of osseous splints or supports. Sometimes the ends of the bones are not eroded, but undergo hypertrophy.

The change in the composition of bones, which facilitates erosion of articular surfaces, renders them also liable to fracture; and this accident easily happens, not only to the long bones, but to those of the pelvis as well, sometimes from simple muscular action. The callus thrown out in such cases is usually very large, and lacks in solidity.

These osseous lesions are not among the earliest symptoms, but they sometimes are the first to attract notice. Charcot places them between the pains and the inco-ordination, though sometimes they appear later.

In a few instances muscular atrophy has been seen in cases of locomotor ataxia; when an autopsy has been obtained in such cases, it has been found that the cells of the anterior cornua were secondarily affected.

Less frequent than the osseous lesions we find trophic changes in the skin, erythema, herpes, bullæ, pustular eruptions, ulcerations, as mal perforant. These changes may appear and disappear frequently, and are

found over the region supplied from the plexus, whence arises the nerve in which pain is felt, though the eruption may not occur in the tract of that nerve. They are most frequently met during the continuance of pain.

Serious mental disturbance is rare except in those cases associated with general paralysis. The symptoms of ataxia may arise before those of general paralysis appear, or the latter may be primary, and the spinal affection set in later.

Sometimes the patient becomes irritable or melancholic, but generally is in good spirits, and very patient. A very few instances of suicide have occurred in patients who dreaded a long and helpless illness.

Among rarer symptoms or complications may be mentioned apoplectic attacks with hemiplegia (Lecoq) and aphasia, usually not permanent.

A form of spinal or cerebro-spinal disease, which is probably a sclerosis, has been described by Friedreich, Carré, Rütimeyer, and others, under the title of *hereditary ataxia*. It attacks several members of a family, sometimes appearing at the age of four years, sometimes as late as the eighteenth year.

There is not the lancinating pain, the ataxic gait appears early, and soon there is inco-ordination of the upper extremities; the speech shows disturbance of co-ordination, and there is ataxic nystagmus; tendon reflex is absent; there is often a slight diminution of sensibility; muscular sense is not disturbed; at length there is paraplegia with contracture; bed-sores rarely form; there is no disturbance of the bladder; the mind is not affected.

Rütimeyer thinks the lesion affects the spinal cord primarily; the medulla oblongata and corpora quadrigemina secondarily. It is certainly not simple locomotor ataxia.

Bernhardt, Erb, and Voigt have given the percentage of cases in which the different symptoms occur in locomotor ataxia.

The following table gives the percentages according to these three observers. The number of cases of each was under sixty; the agreement between the three is noteworthy:

	Voigt.	Bernhardt.	Erb.
	Per cent.	Per cent.	Per cent.
Tendon reflex absent	96·5	95·6	98
Ataxia	93	94·1	100
Staggering, with eyes shut	....	....	93·5
Staggering, non-ataxic	93	....	....
Sense of tiredness	....	92	97·9
Loss of power in walking	95	....	....
Loss of strength	59·5	....	....
Upper extremities affected	19	....	....
Sensory disturbance	....	85·9	....
Diminished sense of touch	98	....	....
Diminished sense of place	94·5	....	....
Diminished sense of temperature	35	....	....
Diminished sense of pressure	45·5	....	....
Diminished sense of pain	68	31·6	69
Delayed sensation	72	34·37	....
Painful after impressions	41	....	....
Paræsthesia	89·5	....	....
Girdle sensation	78·5	....	....
Lancinating pains	94·5	79·5	92·5
Diminished muscular sense	66	....	....
Bladder symptoms	79	76·07	81
Constipation	51	....	....
Impotence	82	43·7	....
Gastric crises	8·5	....	....
Immobile pupil	63	48·4	....
Myosis	45·5	27·2	54
Paralysis of ocular muscles	28	....	38·7
Diplopia	....	39·6	....
Optic atrophy	17·5	10·3	12·3

A *résumé* of the clinical history may group these symptoms together more connectedly.

The earliest symptoms are apt to be mistaken or neglected; they are a temporary diplopia or blurring of vision from presbyopia, which soon passes; occasional attacks of pain in different parts of the body, these sometimes very severe, but of momentary duration; these attacks, thought to be rheumatic, recur with increasing frequency; hyperæsthesia during the

attacks and just after; circumscribed anæsthesia, often symmetrical; more serious loss of vision from atrophy of optic nerve; pupillary phenomena, Argyll-Robertson symptom; occasionally deafness or tinnitus, unilateral or bilateral; weariness and rapidly occurring tired sensation on exercising; absence of patella tendon reflex; various trophic changes; ataxic gait and inco-ordination with at most only fugitive paralysis, and rarely even that. Such are the earlier symptoms, and those of the fully developed disease.

As time elapses, the sensory and motor disturbances increase in gravity; the ataxic phenomena become more and more marked, until walking and even standing is impossible; sensation is finally entirely lost; the arms as well as the legs are affected; the patient is helpless. If to this blindness and deafness are added, his condition is pitiable; fortunately, this is rare. Death may result from some intercurrent disease, or from exhaustion.

The disease is of long duration. The earlier stage, during which the patient can keep about, suffering only during the attacks of pain, may continue for ten to twenty years. There are frequent periods of remission, or even apparent cure. The average duration of typical cases is eight or ten years.

Diagnosis.—When the history of a case is fully known, the early occurrence of lancinating pain in the legs or elsewhere, with a sense of tiredness, occurring soon after exertion; the pupillary and ocular phenomena; the absence of tendon reflex; the anæsthesia; and the ataxic disturbance of motion, occurring at a somewhat later period—are sufficient for diagnosis. At a late period of the disease, if the history is not known, the diagnosis might be less clear.

There may be anæsthesia and a resemblance to inco-ordination arising from weakness in myelitis; but in this case there will probably be little or no pain, and the weakness would show itself when the patient is

lying down. If the myelitis were at all acute, the duration would probably have been short, with some febrile action. Spinal meningitis is not likely to be mistaken for ataxia.

In disease of the cerebellum there may be lack of co-ordination, with retention of muscular strength; but the disturbance of co-ordination is rather different from that seen in ataxia. In walking, the patient has not the same gait, and there are head symptoms which are not found in ataxia.

Cerebro-spinal sclerosis generally differs from ataxia in that there is more muscular weakness, tremor on performing voluntary acts, disturbance of speech, little or no disturbance of sensation, and ataxic inco-ordination is rare. There are cases, however, where the disease has extended to the posterior columns, which it is almost or quite impossible to diagnosticate from locomotor ataxia, especially if tremor is absent, as occasionally happens.

PROGNOSIS.—In advanced cases recovery can not be expected; there may be pauses in the progress of the disease, or the patient may grow worse so very slowly that both he and his friends are encouraged in the hope of his recovery, but such hopes are almost invariably delusive. Seasons of apparent improvement, in which the pains cease and the ataxia diminishes, are not rare in the earlier stages of the disease. It is not unlikely that reported cures are cases where such improvement has occurred. If the disease is not too far advanced, its progress may be checked by treatment; individual symptoms can almost always be ameliorated. The duration is always long, extending over many years, and the knowledge of this fact is often a source of comfort to the patient and his friends.

When the disease seems to be of short duration, yet to have advanced rapidly—cases which seem to run an acute course—the prognosis is less unfavorable, though in such cases the diagnosis is not sure. Lesion of pe-

ripheral nerves may give rise to the symptoms observed under these circumstances.

TREATMENT.—If possible, patients should be restricted in regard to exercise, especially walking, and forbidden to over-exert themselves so as to cause a sense of exhaustion. They should also avoid getting their feet wet, and exposure to draughts of cold air about the feet and legs. The extremities should be warmly clothed. Mental over-exertion, excessive care and worry, are only a little less injurious than physical exertion. Much indulgence in coitus should be absolutely forbidden. Benefit is found sometimes from confining the patient to bed during several weeks, every exertion and motion being forbidden, the patient not being allowed to rise for any purpose whatever. This can be endured by the patient only when accompanied with daily frictions and massage.

Of drugs, nitrate of silver, in doses of one quarter to one half a grain three times daily, is of great benefit in many cases; many times this relieves the pain and increases the feeling of strength. Double chloride of gold and sodium is also recommended. Ergot has been much used and commended. Iodide of potassium is sometimes followed by benefit. The galvanic current is well deserving of trial, and frequently seems to be of benefit; it should be applied to the back, one pole being placed in the cervical region, either in the center of the back or on the side of the neck, the other pole being passed slowly over the dorsal and lumbar region. The duration of the application should not exceed from five to eight minutes; a comparatively weak current should be used, not so strong as to produce any discomfort. The actual cautery, passed very rapidly over the spine, may be of great service.

In Europe, hydrotherapy is much commended. *Cold* baths are not to be recommended; *cool* baths, with a temperature of from 70° to 80°, especially sponge-baths,

may often be used with advantage. *Hot* baths are to be avoided.

The pains in the legs frequently require special treatment. Sometimes external applications, as liniments, especially irritating liniments, will give relief. A lotion composed of chloroform and alcohol, in varying proportion, with a small amount of tincture of aconite-root, will often give relief; this should be put on a piece of flannel wrapped around the limb, and covered with a towel wet in water, to prevent evaporation. Sulphide of carbon may be used in the same way. Iodide of potassium internally is said to sometimes give relief. The actual cautery to the back may be used to relieve the pain in the legs. The galvanic current may be applied to the limb. If it is possible to relieve the pain without having resort to morphia, it is desirable to do so, especially in view of the long duration of the disease; sometimes, however, it is necessary to use that drug, and its effect is prompt. The frequent use of morphia endangers the development of the opium habit.

Lately nerve-stretching has been tried; many cases have seemed to be temporarily benefited, but it is rarely that any permanent benefit has been obtained. The lancinating pains have been more relieved than other symptoms by this operation, which deserves further trial.

CHAPTER XX.

SCLEROSIS.

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MULTIPLE SCLEROSIS.

Multiple sclerosis is the name given to a chronic inflammation of the interstitial tissue of the spinal cord and brain, occurring in patches of greater or less size scattered irregularly throughout the white substance, only rarely invading the gray substance. The proper nervous structures are affected secondarily, undergoing atrophy, and finally disappear.

PATHOLOGICAL ANATOMY.—The pathological process is similar to that found in interstitial inflammation in other organs. The cellular elements are increased in number and in size; the fibrous tissue is increased; the

nerve-fibers suffer in their nutrition, are secondarily affected with inflammatory changes, suffer atrophy, lose their medullary sheaths; the axis cylinders persist for an indefinite time, and finally disappear. There is left then a close net-work of fibers surrounding and inclosing nuclei and cells of the neuroglia. Granular corpuscles are found in the earlier stages. The cord acquires sometimes a firm consistency, though it may be softer than normal; it has a grayish, translucent appearance, the white substance somewhat resembling thus the gray substance.

The walls of the blood-vessels are thickened, and it is impossible to tell how soon this change sets in.

It is only exceptionally that the nerve-cells of the gray substance are altered.

The spots of disease, varying greatly in size, may be situated in any portion of the central nervous system; sometimes there are few in the cord and more in the brain, or the brain may be the less affected. They are found in all parts of the brain, in the crura, pons, and medulla, and are perhaps less frequent in the cerebellum. The nerves, especially the optic and auditory nerves, may also be affected.

ÆTIOLOGY.—The cause of sclerosis can be definitely determined in only a few cases. It is a disease of early adult life, yet it is found in childhood, and even in infancy, Seeligmüller reporting a case aged one year and nine months; several cases have been reported between four and ten years of age. It is, however, most frequent between twenty and thirty years. Charcot sets forty as the extreme limit when it appears.

Heredity seems to have little or no ætiological influence, though Seeligmüller reports four cases, all under ten years, in one family.

Charcot and some others following him have stated that the disease is more common among women than men. This may be so to some extent, but statistics are too meager to settle this point.

Accidents involving the spine, causing concussion or jar of the cord, may be exciting causes of the disease. Acute diseases, typhoid fever especially, may be followed by cerebro-spinal sclerosis. Protracted and excessive toil seems sometimes to be the cause; also mental disturbances, as worry, anxiety, or fright.

Symptoms.—After injuries, mental shocks, or when following acute diseases, cerebro-spinal sclerosis may develop rapidly, so that an early diagnosis is possible. Generally, however, the first symptoms are so insignificant that they are not considered by the patient to be of importance, and so are neglected; indeed, it is not possible to foretell whether the slight motor and sensory disturbances which first give warning of more serious trouble will continue, or may not prove mere transitory phenomena.

The earlier symptoms are generally not continuous; they are also varied in character, sometimes referable to cerebral disturbance, sometimes to spinal; the patient may be thought to be hysterical, or suffering simply from neurasthenia.

Very frequently the first complaint is in regard to motor disturbance—there is weakness, and it is difficult for the patient to ascend or descend stairs; he wearies sooner than usual in walking; he can no longer follow his usual occupation with comfort; after a while a tremor of the hand is noticed, the handwriting becomes less legible, and then it is impossible to write on account of the tremor.

The disturbances of sensation during the earlier stages are not constant; there may be distress in the head, and sometimes headache or dizziness; severe pain anywhere is rare, though it sometimes occurs; there may be backache or weariness following exertion; numbness and abnormal tingling sensations in the limbs are not uncommon. Diplopia and ambliopia are occasionally noticed. Nystagmus is a much more frequent symptom.

These earlier symptoms may be present for many weeks or months in such slight degree, or with such varying conditions of apparent good health, that no notice is taken of them. Finally they become so severe as to oblige the patient to acknowledge that he is ill; it is more frequently the tremor and weakness which cause the most annoyance. Often there is so little sensory disturbance, that the patient does not consider himself seriously sick.

The tremor is characteristic. When at rest, the limbs are quiet and motionless; when a voluntary motion is performed, the limb trembles, at first only slightly; later, the tremor is so severe as to interfere seriously with the use of the hands. It is seen most clearly when the patient tries to perform some act requiring a careful balancing of the muscular forces, as the carrying a cup or spoon to the mouth, or the use of a pen. As the disease advances, the trembling may increase until all use of the limbs is impossible, or the whole body may be tossed about, or the tremor may disappear; then there is left great weakness, partial paralysis.

The legs are affected similarly to the arms, but the tremor is less easily recognized in them; yet, by asking the patient to execute movements with the legs while lying or seated, the tremor can be recognized. There may be stiffness and weakness which interfere with walking, but differing from the inco-ordination found in ataxia. As the disease advances, the instability and weakness of the legs become so great as to oblige the patient to give up walking.

The nystagmus, which has been mentioned in connection with ocular affections, is due to the tremor of the muscles of the eyeball. The motion is a lateral one; it can sometimes be made more prominent by asking the patient to look to one side or at an object held near the eyes, so as to require an exertion of the will to adjust the axes of the eyes to distinct vision—

that is, when the eyes are at rest there is no tremor, but a voluntary effort causes it.

Allied to the other motor disturbances is a peculiar manner of speaking: at first there is merely a slight hesitation, a drawling utterance; then there is more delay in pronouncing the individual syllables, and the sentences are, as it were, scanned. Some disturbance of speech is very common.

Reflex motions are not lost, may be somewhat exaggerated, especially the patella-tendon reflex, except toward the close of the disease, or in the rare cases where the posterior columns are much affected. Ankle clonus may be present when the lateral columns are considerably diseased; it may appear, and after a while disappear, according to changes in the cord during the progress of the disease.

The electrical reaction of the muscles is not altered unless, in rare instances, the disease invades the anterior cornua, and then there is muscular atrophy.

In the later stages, sensation may be more affected, and spots of anæsthesia may be found scattered over the limbs or body. Pain is rare even in the later stages. Disagreeable sensations of tingling, formication, a sensation as if the limbs were asleep, are not uncommon. A sinking or faint feeling may give much distress.

Sometimes the cerebral symptoms are very prominent; headache, dizziness or vertigo, change of disposition, mental heaviness, are among the more frequent cerebral symptoms.

Pseudo-apoplectic attacks may occur; the patient suddenly becomes unconscious, is hemiplegic, and may have convulsions; the symptoms closely resemble those of an ordinary attack of cerebral hæmorrhage. The attack may end fatally, or gradually consciousness is regained, the motor power returns, and the patient recovers, yet is usually not quite so well as before the attack. The course of the temperature is a valuable aid in diagnosis in these attacks. According to Charcot,

the temperature rises with the commencement of the attack, and may reach 104° within twenty-four hours; unless death occurs, the temperature falls to normal by the second or third day. The pulse is also rapid at the beginning of the attack. Several of these apoplectiform attacks may occur at intervals during the course of the disease.

When the interstitial changes affect the posterior columns, we may have symptoms of locomotor ataxia complicating and masking those of the original disease. When the lateral columns are chiefly affected, symptoms corresponding with the location of the disease will be noticed.

Besides cerebro-spinal multiple sclerosis, some authors recognize a cerebral form and a spinal form of the disease. While it is true that the brain or spinal cord may be chiefly affected, it is very rare that either is exclusively the seat of the disease. Occasionally the spinal symptoms predominate, all mental phenomena, even trembling, being absent. Such a case may resemble simple chronic myelitis, except that the symptoms are more general and advance more irregularly than in that disease. It is very doubtful whether cerebral sclerosis has ever been seen without lesion of the spinal cord.

DIAGNOSIS.—Multiple sclerosis was formerly confounded with paralysis agitans on account of the tremor. If the nature of this tremor is observed, it is scarcely possible to make such a mistake; in sclerosis the tremor ceases when the limbs are at rest, or is very much less marked in the few cases where it is so severe as to be nearly constant. In paralysis agitans the tremor is at least as well marked during repose, and usually it is much diminished during voluntary exertion.

Ordinary cases of chorea and multiple sclerosis are not likely to be mistaken, except where the tremor of sclerosis is extremely severe and almost continuous.

There is much similarity between the motions in such a case and those found in severe cases of chorea. A careful study of the history and course of the disease would probably prevent a mistake.

Cerebral pachymeningitis may also resemble multiple sclerosis, but a careful study of the history of the case and examination of the symptoms will guard against error.

When the sclerosis affects the posterior columns of the spinal cord, the symptoms may correspond in many respects with those found in locomotor ataxia. If the tremor is slight, or has not shown itself, and the tendon reflex is lost, it may not be possible to make a correct diagnosis. When the characteristic tremor is present, and the tendon reflex is retained, there should not be any mistake made.

It may not always be easy to decide the diagnosis between sclerosis and general paralysis, especially if the mental symptoms are slight or entirely absent in the latter. In general paralysis there is tremor of the facial muscles and tongue during voluntary motion rather than of the muscles of the extremity ; the pupils may be unequal ; the disturbance of speech is different, the person talks with an indistinctness of utterance and a hesitation, repeating his words or sentences, sometimes pausing for a few seconds or a minute, then finishing his sentence. In sclerosis the patient is aware that his health is defective ; in general paralysis he seems to consider himself well, and does not recognize symptoms which are evident to others. If delusions of grandeur or other mental disturbances characteristic of general paralysis show themselves, the diagnosis, of course, is easy.

Prognosis.—When once multiple sclerosis has been certainly recognized, we must consider that the patient's fate is sealed, that he will not recover health, that the disease will probably steadily progress to a fatal termination unless life is shortened by some intercurrent

affection. The progress of the disease may, however, be interrupted by remissions of longer or shorter duration; during these the severity of the symptoms may diminish, and there may seem to be a flattering prospect of recovery, but the symptoms are sure to return.

It is not possible to fix the duration of the disease with any degree of certainty; from the time the nature of the affection is recognized to the fatal termination may be only twenty or thirty months, but is usually much longer, and, if the disease is confined chiefly to the spinal cord, its duration may extend to fifteen or twenty years.

TREATMENT.—The general testimony of observers is that treatment is frequently of no avail. The remedies which have proved of temporary value are, nitrate of silver in doses of from $\frac{1}{4}$ to $\frac{1}{2}$ grain three times a day; double chloride of gold and sodium, $\frac{1}{20}$ grain; ergot, iodide of potassium, and arsenic either by mouth or subcutaneously. Bathing has been recommended; the baths should be neither too cold nor too hot, but cool rather than warm. Greater advantage may be expected from a persistent use of electricity; the applications should be made daily or every other day with the galvanic current, not too strong, to the back, one pole above, the other below, over the spine; the direction of the current is a matter of indifference—it may be varied at different sittings. A much weaker current may be passed transversely through the head, the poles being placed behind the ears; great care must be taken not to use too strong a current. To obtain good results from electricity, it must be continued through many months.

Patients should not be allowed to fatigue themselves, either by attending to the ordinary occupations of life, or by taking exercise under the impression that it will strengthen them; over-fatigue of mind should also be avoided; alcoholic drinks had better be forbidden, and tea or coffee used only in moderation.

SCLEROSIS OF THE LATERAL COLUMNS.

This affection has also been called spastic spinal paralysis and spasmodic tabes dorsalis, and, though previously described by Türck, was first recognized as a distinct form of sclerosis by Charcot, and later by Erb. Though for some years there was doubt whether sclerosis of the lateral columns can properly be considered a distinct disease, the opinion seems to be gaining ground that it is distinct from other lesions of the cord.

PATHOLOGICAL ANATOMY.—The pathological changes are such as are found in locomotor ataxia, and are generally confined to the lateral pyramidal tract—that portion of the lateral columns affected by secondary descending degeneration. The pathological change may extend somewhat beyond this region. A variety of lateral sclerosis, called by Charcot amyotrophic, is characterized by destruction of the nerve-cells in the anterior cornua, in addition to the changes in the lateral columns.

The *causes* are obscure, and no satisfactory observations have been made on that point.

SYMPTOMS.—The motor symptoms are most prominent, and may commence in the upper or lower extremities; rather more frequently, perhaps, in the latter. First is noticed a slight weakness and tendency to become easily fatigued. The weakness gradually increases until the patient has great difficulty in getting about, or is confined to the bed. This symptom may first appear in one limb only; but after a while the other limb is also affected, and before death all four extremities are usually implicated. Ataxic symptoms are very seldom seen.

Besides the above paralytic phenomena, a class of symptoms are developed which are peculiar to lesions of the pyramidal tracts. These may appear even in the early stages of the disease, when the weakness is

as yet very slight; they consist in spasmodic jerkings and tremors, with other signs of irritation.

The tendon reflex is greatly exaggerated; a very slight tap just below the knee causes the leg to jerk forward with considerable force; other tendons also show an increase of reflex irritability, as in the arms just above the olecranon at the elbow, over the tendon of the triceps, or at the wrist, and sometimes over the tendons of the muscles of the shoulder and neck. Ankle clonus is generally very strong.

Owing to this increase of tendon reflex, the patient's gait is peculiar, called by Erb the spastic gait, and thus described by him: "The legs are somewhat dragged, the feet seem to cleave to the ground, the tips of the feet find an obstacle in every inequality of the ground; every step is accompanied by a peculiar hopping elevation of the whole body, dependent on a reflex contraction of the calf; the patient immediately gets upon his toes and slips forward on them, showing a tendency to fall forward. The legs are close together, held stiffly, the knees somewhat depressed forward. There is no throwing about of the feet. This gait depends on muscular tension and reflex contractions in the various groups of muscles, which are set in activity during the process of walking."

The increase of reflex irritability is shown by spontaneous jerking and twitching of the limbs occurring when the patient is lying down, but in extreme cases occurring also when he is sitting. The extensor muscles are most frequently affected; sometimes, however, the flexors draw the knees and legs spasmodically; the spasm consists simply of a strong tremor of the limbs, which is often excited by the act of stretching. A spasmodic stiffness of the limbs, at first intermittent, interferes with motion; when this stiffness becomes continuous, there is permanent contracture of the limbs, the legs and feet being extended, the toes are sometimes flexed; the adductor muscles keep the legs

closely approximated to each other. The arms are much less frequently affected with contracture.

Erb found the skin reflexes generally normal in about two thirds of his cases, increased in hardly one third. He found the faradic and galvanic excitability of the motor nerves slightly lowered, never increased.

The functions of the bladder and rectum, and the sexual function, are not interfered with.

There are no brain nor bulbar symptoms. Sensation is not disturbed when only the lateral columns are diseased.

AMYOTROPHIC LATERAL SCLEROSIS.

Charcot first described a form of disease in which sclerosis of the lateral columns is associated with atrophy and destruction of the cells of the anterior cornua. The pathological changes are the same as those in lateral sclerosis, with the addition of destruction of nerve-cells of the anterior cornua, those in the upper part of the cervical region being most affected; one group of cells is not more likely to be diseased than another; both sides are usually affected. The hypoglossal nucleus, and sometimes the other nerve-centers of the medulla oblongata, may be affected as well as the cells in the cervical region of the cord.

The nerve-roots and trunks, which take their origin from the diseased cornua, suffer secondary atrophy, and the muscles supplied by them undergo the usual atrophic changes.

The first symptom is a weakness of the hands, followed soon by wasting and spasmodic rigidity; fibrillary contractions are generally to be seen in the affected muscles. Finally, the symptoms of bulbar paralysis (labio-glosso-laryngeal paralysis) show themselves; the tongue and lips are paralyzed, swallowing is difficult or impossible, and speech is very much interfered with. The legs rarely show marked signs of wasting; the

symptoms, as already described as due to lateral sclerosis, are developed in the legs.

The electrical contractility of the muscles suffers in so far as there is atrophy. Sensation is but little disturbed, though there is sometimes a painful hyperæsthesia of the muscles affected. Sometimes the contractures disappear before death.

The prognosis is decidedly unfavorable, and the course of the disease much more rapid than that of simple lateral sclerosis, the usual duration being only two or three years.

PROGNOSIS AND DIAGNOSIS.—The progress of the disease is very slow, and, interrupted by periods of remission, it may extend through many years.

Recovery is more frequent in cases of lateral sclerosis than in many other forms of chronic disease of the spinal cord. The disease itself rarely causes death, the fatal termination usually occurring in consequence of some complication.

There is no other disease in which such a complex of symptoms is found as described above; the reflex and spasmodic phenomena are peculiar, and, so far as known, occur only when the lateral pyramidal tracts are affected. When these tracts are subject to secondary degeneration, the result of cerebral disease or disease of the mesencephalon, similar reflex phenomena are observed; but then the history of the origin and progress of the disease will render a diagnosis easy, and even without these the hemiplegic character of the symptoms would indicate their cerebral origin.

In multiple sclerosis the lateral columns may be chiefly affected, in which case the symptoms peculiar to such lesion will predominate, and, if the disease extends but little beyond those columns, an error of diagnosis is inevitable. Generally, however, there will be other symptoms pointing in the right direction. The physician's skill will be tested in unraveling the complication of symptoms so as to recognize those depend-

ing upon lateral sclerosis and those depending upon sclerosis of other parts.

TREATMENT.—Comparatively little may be said in regard to treatment; nitrate of silver is recommended by some. Erb mentions the galvanic current as affording the best results; he also favors a "reasonably conducted water treatment"; indeed, the treatment is very similar to that in other cases of chronic myelitis.

CHAPTER XXI.

PSEUDO-HYPERTROPHIC PARALYSIS.

DUCHENNE, G. B., De l'électrisation localisée, 3me édit., 1872. —ORD, W. M., Notes of a Case of Duchenne's Pseudo-hypertrophic Muscular Paralysis. *Med. Chir. Trans.*, 2d Series, vols. xxxix, xlii.—POORE, C. T. *New York Med. Jour.*, 1876.—MOORE, M. *Lancet*, June 19, 1880.—GERHARD, G. S. *Phila. Med. Times*, Oct. 16, 1875.—GOWERS, Pseudo-hypertrophic Paralysis. London, 1879.

ÆTIOLOGY.—This disease is almost confined to males, very few cases having been seen among females. It is not uncommon to find several cases in the same family, the boys being affected, and the girls as a rule escaping. When several branches of a family are affected, the disease is almost invariably found among the mother's relatives, not the father's.

The subjects of pseudo-muscular hypertrophy are children; very few adults are attacked; it usually begins before six years, and sometimes before the child learns to walk. Gowers finds that it begins later in girls than in boys.

The conditions or circumstances which cause the disease to appear are unknown.

SYMPTOMS.—The earliest noticeable symptom is diminution of motor power: the child either learns to walk late, or loses its steadiness and acquires peculiarities of gait and posture. Notwithstanding this weakness, the muscles seem to be of good size, especially those of the calves, and the parents think it is strange that, with such large, plump legs, their children find so

much trouble in getting about. This apparent hypertrophy may be noticed in other muscles, as those of the thigh, the glutæi, more rarely those of the upper extremity. Duchenne gives a representation of a patient who had enlargement, apparently, of all the muscles except the pectorals, which were atrophied.

Slowly the weakness extends and increases; the enlargement of muscles does not extend, but the loss of power is usually attended with atrophy, so that the patient finally appears reduced almost to a skeleton with enormously large legs.

The loss of power in various muscles leads to peculiarities of posture and gait which are characteristic. The patient, in standing, throws his abdomen forward, his shoulders backward, and bends his head slightly forward so as to keep his balance. Duchenne thinks this posture is caused by weakness of the muscles of the back. Gowers ascribes it to weakness of the extensors of the hip, which causes the pelvis to incline forward more than normal. When this lordosis is marked, if the patient stands, a plumb-line falling from the shoulders passes more or less in rear of the sacrum. In standing, the patient keeps his feet widely separated, and walks with a waddling or rolling gait, which depends upon the weakness of the glutæi.

When the weakness has advanced only a little, the patients need to help themselves with their arms in rising from a chair, and, if the loss of power is considerable, the patient must help himself more, and, so to speak, climbs up his own legs. Gowers describes the different ways in which patients assist themselves: Some put their hands on their knees, then on the thighs, grasping them, and the hands are moved alternately higher and higher until they are upright; others, to rise from the floor, take a position on hands and knees, then on hands and feet, or rather toes, with the feet wide apart, then, moving the hands backward on the ground till the legs are nearly perpendicular, they

put one hand on one knee, and with a slight spring rise upright.

Fibrillary twitchings are often seen in muscles undergoing atrophy, as in progressive muscular atrophy.

Reflex functions, cutaneous and tendinous, suffer, apparently, according to the amount of disease in the muscles.

Electrical reactions are diminished in proportion to the muscular atrophy and the amount of fat deposited, a large increase of fat making it necessary to use a stronger current to obtain equal reaction.

Sensibility is not disturbed, and it is very rare that there is pain.

Mental powers are only exceptionally blunted. In a few cases epileptic fits have been recorded; but, as Gowers says, they are probably the result of an associated, not of a related, cerebral disease.

The progress of the disease is slow; gradually one muscle after another is affected, sometimes one side being attacked a little before the other, but usually both nearly together. There are periods of quiescence, but the tendency is steadily onward. When the disease commences early, life is not prolonged many years; when later, the patients may live to adult years. Usually death occurs between ten and twenty-five. Death is usually caused by some intercurrent disease, often of the respiratory organs.

PATHOLOGICAL ANATOMY.—The hypertrophied muscles are found at the autopsy to be largely composed of fat; the muscular fibers are diminished in size and widely separated by masses of connective tissue filled with fat. Statements of observers do not agree as to fatty degeneration of the muscular fibers; probably it sometimes occurs; they more frequently suffer simple atrophy. Finally, the muscular tissue disappears and gives place to fibrous tissue.

Changes in the spinal cord are not constant. Many times none have been found, sometimes the cells of the

anterior cornua are more or less diseased, and sometimes the white substance near the gray is diseased. Gowers concludes that pseudo-hypertrophic paralysis of early life is not a disease of the spinal cord.

The only constant change found, in all cases, so far, has been the muscular degeneration. I am not prepared to accept Friedreich's views as to the nature of the affection. I can not form any satisfactory theory, and must leave that for the developments which may be learned in the future.

DIAGNOSIS.—There is no danger of mistaking the fully developed disease if care is taken in examination. If the legs or thighs are not much hypertrophied, there may be some doubt about the nature of the disease, and progressive muscular atrophy may be thought to be present. In the earliest stage a portion of muscle may be removed by a "harpoon," and examined under the microscope in order to settle the diagnosis.

PROGNOSIS.—The most that can be hoped is that the disease will cease advancing for a while. What has been said in regard to its course and progress will aid to an intelligent prognosis. If in any case, however, friends desire to know how long a child thus afflicted will live, we need to be cautious about giving a definite answer. We do not know.

TREATMENT.—The plan recommended for progressive muscular atrophy is that which is most rational. Gowers has obtained slight benefit from arsenic and from phosphorus.

DISEASES OF THE PERIPHERAL AND SYMPATHETIC NERVES.

CHAPTER XXII.

NEURITIS.

MITCHELL, S. WEIR, Injuries of Nerves and their Consequences. Philadelphia, 1872.—NIEDICK, W., Ueber Neuritis Migrans und ihre Folgezustände. *Arch. f. exper. Pathol.*, vii, 1876, p. 205.—MILLS, C. K., Traumatic Neuritis involving the Brachial Plexus. *Philadelphia Med. Times*, 1877, p. 564.—TREUB, HECTOR, Ueber Reflexparalyse und Neuritis Migrans. *Arch. f. exper. Pathol.*, x, 1879, p. 398.—GOMBAULT, Contribution à l'étude anatomique de la névrite parenchymateuse subaiguë et chronique. *Arch. de nevrolog.*, i, 1880, pp. 11, 127.

Multiple Neuritis.—EICHHORST, H. *Virch. Arch.*, 69, 1877, p. 265.—LEYDEN. *Charité Ann.*, v, 1880, p. 206.—STEWART. *Edinburgh Med. Jour.*, 1881, vol. xxvi, p. 865.—CASPARI. *Zeitschr. f. kl. Med.*, 1882, p. 537.—PIERSON. *Volkmann's Sammlung*, No. 224, 1883.—STRÜMPELL. *Arch. f. Psych.*, xiv, 1883, p. 339.—MÜLLER. *Ibid.*, p. 669.—VIERORDT. *Ibid.*, p. 678.—WEBBER, S. G. *Archives of Med.*, xii, Aug., 1884.—SCHEUBE, B., Die Japanische Kak-ke (Beri-beri). *Deut. Arch. f. kl. Med.*, 31, 1882, p. 141 *et seq.*

SIMPLE NEURITIS.

PATHOLOGICAL ANATOMY.—The nerve-fibers, or the sheath of the nerve, may be the seat of inflammation, which may be acute or chronic. When the nerve-fibers are affected, the nuclei in the neurilemma are multiplied, the medullary substance divides and undergoes a granular or fatty degeneration, the axis cylinder may be hypertrophied, but finally is destroyed, and there remains only a fibrous band in place of the nerve. When the sheath of the nerve, the perineurium, is chiefly affected, it becomes thickened by formation of new tissue and the infiltration of serum; the nerve-fibers are compressed, and undergo degeneration secondarily.

In acute neuritis the nerve is rather more congested, the nerve-fibers are the more frequently most affected, and pus is more likely to form; sometimes hæmorrhages occur into the sheaths of the nerve, leaving pigmentation after the blood has been absorbed.

Chronic neuritis may follow as the result of acute, or may occur spontaneously; the sheath is more likely to be the chief seat of the disease, and the nerve-trunk is thereby much thickened. The nerve-fibers degenerate and undergo atrophy.

The nerve beyond the seat of inflammation undergoes the secondary Wallerian changes when the fibers are entirely severed.

Trophic changes in the limbs may follow as results of the neuritis.

ÆTIOLOGY.—Injuries are the most common cause of neuritis. All kinds of wounds, bruises, and contusions may give rise to the disease; tumors, abscesses and inflammatory changes in the vicinity of the nerve, compression of the nerve, whether from external or internal causes, may give rise to the disturbance. Rheumatic thickening of the sheath, from exposure to cold, is a very common cause. Neuritis sometimes occurs after eruptive fevers or diphtheria, or is the result of syphilis.

SYMPTOMS.—Pain is the most prominent symptom of neuritis, at least in the patient's opinion. There may be fever with chills before the pain is felt; but this is not very common. The pain is often severe, of a burning character, sometimes more aching in nature; it is felt in the course of the peripheral distribution of the nerve; the limb is hyperæsthetic, sometimes a very slight touch causing distress; there is always tenderness over the course of the nerve, especially where it is superficial. The pain is continuous, but with seasons of exacerbation; often is most severe at night.

When the nerve is seriously affected, the sense of touch is much diminished or lost; even in rather mild cases there is a dullness of that sense, yet the anæs-

thetic part may be very tender, and a slight pressure may cause pain.

The pain may prevent motion, but subsequently the muscles to which the nerve is distributed lose their power, are paralyzed, and undergo more or less wasting according to the amount of change in the nerve. When the nerve is entirely destroyed, the motor paralysis is complete, the atrophy extreme, with the reaction of degeneration.

When the muscles are only partially paralyzed there may be tremor, very closely resembling the tremor of sclerosis; or more extensive spasm and twitching may occur.

After the acute symptoms have subsided, or from the beginning in other cases, a *chronic neuritis* may be recognized. The symptoms are the same, though perhaps less severe than in the acute, except that there is no fever, and at length other symptoms are added. The pain may have less of the burning character, but be quite as wearing; often, however, it is only moderate in degree, and in very mild cases is not present continuously; it is excited or increased by use of the limb. The numbness and pricking are the same as when the disease is acute. There is always tenderness over the affected nerve, which is often swollen.

The motor disturbance is the same as in acute neuritis, but may be more slowly developed; the reaction of degeneration is proportionate to the amount of atrophy; when the disease is very slight, the electrical irritability may be increased. Tremor, as in sclerosis, is more common than in the acute form.

Trophic changes in other than the muscular tissue are almost always noticed. These have been studied especially by Mitchell, and consist in herpetic, vesicular, and other eruptions, atrophy of the skin, "glossy skin," with a peculiar hyperæsthetic condition, "causalgia." The nails may become clubbed, brittle; their growth is less rapid. The hair is brittle, may fall

off, or may grow abnormally long, or it may become white.

Acute neuritis may be of short duration, the nerve soon recovering its normal condition. Chronic neuritis persists for many weeks or months, and, when it has apparently disappeared, the symptoms are easily excited again.

Neuritis shows a tendency to extend toward the nerve-centers, or to pass to adjoining nerves. Not infrequently the inflammation extends until it seems as if all the nerves of the limb were affected. I have seen this in the arm rather than the leg, perhaps because the patients could not or would not give the limb the needed rest. This extension is not always by continuity; the inflammation may jump over a stretch of healthy tissue. The disease may extend to the cord, and thereby death may result.

PROGNOSIS.—The patient's life is only rarely threatened when the disease extends to the nerve-centers. Perfect recovery is possible when the disease has continued comparatively long, if the changes have not become too extensive, and even serious and severe cases may do well. Many times, however, the trophic changes in muscles and other structures are so considerable that a complete recovery of function is not possible; the symptoms of neuritis disappear, but the parts remain partially helpless. In less favorable cases more or less pain may be felt at intervals, showing that the nerve-fibers are still subject to irritating influences. A relapse, or a second attack, is not uncommonly the result of comparatively slight imprudence in over-exertion or exposure.

TREATMENT.—Rest is of great importance in both acute and chronic neuritis. The limb affected should be kept quiet; if necessary, the patient should be confined to bed. If the arm is affected, it may be well to secure a splint lightly to the limb.

In acute cases, cold may be applied constantly over

the seat of the inflamed nerve. A rubber bag, so arranged that a stream of cold water will flow continuously through it, is convenient.

Galvanism has been advised, and is of benefit in some cases. It is not likely to do much good in acute cases, and in chronic is often less useful than blistering; it may even increase the pain and aggravate the symptoms. After recovery is fairly established, and the pain has ceased, electricity will be of value in restoring the use of the partially paralyzed muscles.

In subacute and chronic cases the most efficient means is blistering. A blister the size of a ten-cent piece or a quarter should be put over the tender points in the course of the nerves. If several nerve-trunks are affected, one after the other may be thus treated. It is not well to apply many at one time. Sometimes the pain is worse until a day or two after the blister is drawn, when a marked relief will be experienced; the blister seems to be of most benefit while the raw surface is healing; for this reason the healing should be favored as much as possible by not removing the cuticle, and by avoidance of irritating ointments and friction.

It may be necessary to control pain by giving morphia or other narcotics. A four-per-cent solution of carbolic acid, applied to the limb on compresses, may be useful in relieving the pain.

Salicylic acid in rheumatic cases, iodide of potassium, quinine in rather large doses, and, later, cod-liver oil and other tonics, would be of value.

During convalescence, when pain has nearly or quite ceased, electricity, massage, passive exercise, or the Swedish movement, may be used to restore function and increase the nutrition of the limb.

In syphilitic neuritis, of course the specific treatment should be used.

MULTIPLE NEURITIS (Disseminated Neuritis).

Pathological Anatomy. — The affected nerves show no special gross change; they do not seem to be enlarged or congested. Under the microscope the nerve-fibers are found to have undergone extensive changes. There is inflammation, and below this the medullary sheaths, with the axis cylinders, are segmented, then divided into smaller granular masses, and finally these are absorbed. The nuclei increase in numbers. There is only a moderate increase of the interstitial tissue.

The muscles show changes due to degeneration—a granular appearance; fat is deposited between their fibers, which may undergo simple atrophy; their nuclei are multiplied.

Ætiology.—Exposure to cold and over-exertion are considered important as causes. Certainly many patients refer their disease to "catching cold." Caspari suggests that multiple neuritis may be an infectious disease.

It has been thought by several, who have had opportunity to observe it, that *beri-beri*, or *kak-ke*, is a multiple neuritis. Scheube has carefully examined twenty cases, post-mortem, and reaches this conclusion.

Symptoms.—The disease may begin with fever, preceded or not by a chill, or the fever may be absent. The pulse is habitually rapid throughout the disease. Pain and stiffness in the limbs are usually first to attract the patient's attention—usually the legs, sometimes the arms, being first affected. Any effort to move the limbs increases the pain, which may then be most acutely felt about the joints. A hyperæsthesia of the muscles increases the resemblance to rheumatic fever.

The pain is almost constant, may be extremely severe, is attended with a sensation of tingling or pricking, or may be of a burning character, as if very hot

water were applied to the limb. Sometimes the pain seems to be confined to the course of the diseased nerve or nerves, or it is generally diffused over the region to which the nerve is distributed. Not only is there general hyperæsthesia to pressure, but the course of the nerve is tender, and, upon pressure over the nerve-trunks, the pain is increased in the limb also.

The sense of touch is diminished in the affected parts; especially is this noticed by the patient after the pain has partially or entirely disappeared.

The muscles supplied by the affected nerves are partially paralyzed early in the course of the disease, yet motion is restricted more by the pain excited than by the weakness; later there may be total paralysis of single muscles or groups of muscles.

The cutaneous reflexes are absent in about half, and the patellar tendon reflex is absent in more than nine tenths of the cases; the latter is very late in returning.

The limbs are more or less flexed, and this position may be maintained by contracture of the muscles; then passive extension is very painful.

The muscles waste and show the reaction of degeneration. This change may occur very rapidly in acute cases. Abnormal positions of the fingers and limbs may be caused by the wasting, as in other cases of muscular atrophy.

Trophic changes may be seen in the skin, occasionally there is œdema of the limbs, and sometimes excessive sweating.

The inflammation extends more or less rapidly from nerve to nerve, not following any regular order, though the disease is usually roughly symmetrical, both legs or both arms being affected at about the same time. The different muscles are not equally paralyzed on the two sides.

Mental and cerebral symptoms are usually wanting; when present, they have seemed to be due to some complication; the suffering has seemed to give rise to a

hysterical condition; occasionally a mild nocturnal delirium is noticed.

The paralysis may extend until so extensive that life is threatened, and death may result from paralysis of the respiratory nerves. In most cases, however, after a time the pain and tenderness diminish and finally disappear, leaving the anæsthesia and weakness. Recovery is slow, delayed by contractions of the limbs, and, if the nerve-structures have been seriously damaged, it may never be complete.

DIAGNOSIS.—Multiple neuritis is most likely to be confounded with anterior poliomyelitis, progressive muscular atrophy, lead paralysis, and rheumatic fever.

The sensory disturbances distinguish it from disease of the cells of the anterior cornua of the cord; these and the marked changes in electrical reactions will distinguish it from progressive muscular atrophy, where there are only very slight electrical changes, simple diminution of reaction, and rarely if ever the reaction of degeneration. Lead paralysis has many of the symptoms of multiple neuritis; but, as a rule, the sensory disturbance is less severe; the other signs of lead-poisoning and the elimination of lead by the kidneys under the use of iodide of potassium are of value in making a diagnosis.

It is only at the very beginning that neuritis and rheumatic fever resemble each other; the latter is accompanied with a higher temperature, and soon the joint affection and the course of the disease will make the diagnosis clear.

TREATMENT.—During the earlier stages of the disease, salicylic acid or the salicylate of soda, in large doses, is apparently of most value. Bags of hot water to the spine have seemed to relieve the pain; hot baths are said to be helpful. Small and frequently repeated doses of aconite are sometimes of value.

The chief indication at first is to relieve pain. Morphia should be given as freely as needed. For external

use, chloroform, or a four- to five-per-cent solution of carbolic acid, may be found serviceable. A subcutaneous injection of a two-per-cent solution of carbolic acid is recommended by Caspari. If the disease is not widespread, blisters might be of advantage.

Rest in bed is, of course, necessary; massage and passive motion should not be attempted till after the disease has come to a standstill. The same is true of electricity. These agents are, however, of great value in restoring their function to the paralyzed muscles; the galvanic current is preferable in most cases. Massage and passive motion should be used systematically to overcome the contractures which remain after the acute symptoms have disappeared.

may be absent for days or months. An extended period of freedom from all pain is rare in a patient very much affected. In severe cases remissions are more common than intermissions. The first attacks are often comparatively light, and the severity of the pain gradually increases as the attacks multiply.

The pain is always felt either at one point of a nerve or along the course of a nerve. Not infrequently a patient unacquainted with anatomy will map out the affected nerve and its branches. The locality of the pain may be different in the different attacks, shifting perhaps to the opposite side of the body. When the pain always affects the same nerve, there is a strong probability that it is due to an organic lesion of that nerve.

In the beginning of the disease, and in uncomplicated cases throughout, there is no elevation of temperature.

When one nerve has been long the seat of pain, there is usually a loss of acuteness in common sensibility of the skin, and an increase in acuteness to sensation of pain. Certain points become tender, so that a very light touch is painful. These points, *points douloureux*, were specially studied by Valleix, and are sometimes named from him. They are found where the nerve passes through bony canals, or through fasciæ, becoming thus superficial. These points may not be painful when the disease has but recently commenced; sometimes they are entirely wanting. If the pain is clearly intermittent, they may not be tender to pressure during the intermission. It is not uncommon to find the spinous processes of the vertebræ, between which the affected nerves pass, painful upon pressure, *points apophysaires.*

The pain of neuralgia is increased by all motions of the affected parts; thus, motions of the jaw in chewing, or of the face in talking and laughing, will increase the severity of facial neuralgia; so walking will render the

suffering more severe in sciatica. Necessarily, therefore, under such conditions, neuralgia will interfere with motion.

Besides the above causes of immobility, there may be actual weakness of the muscles, a partial paralysis. Twitchings, tremor, and even more severe spasms of the muscles, may attend the paroxysm as well as precede it.

There may be changes in the circulation or the nutrition of the affected parts. The arteries are at first contracted, and the skin is pale; later, relaxation of the vessels gives rise to a more congested appearance, and there may even be a tendency to cyanosis. Œdema is sometimes noticed in the limbs or face. The secretions may also be altered, the tears flow freely, and the saliva and urinary secretions are abundant.

Decided trophic changes are usual in old, obstinate cases, especially in neuralgia of the limbs; the muscles are wasted more than can be explained by their lack of exercise. This wasting has been explained by the changes in the nerve which give rise to the pain, perineuritis, or by supposing a change in the circulation of the cord, and hence disturbance of nutrition in the motor cells of the anterior cornua.

Cutaneous eruptions, herpes, erythema, pemphigus, urticaria, and psoriasis may be found among the complications of neuralgia. The skin may become thickened, and the hair may change color.

Except in severe and long-continued cases, the general health and disposition rarely suffer. Persistent and extreme pain, however, impairs the digestion, disturbs sleep, prevents exercise, taxes the endurance, and at length there is evident a disturbance of the general health; the temper becomes more irritable and peevish, mental power may be weakened, and finally there may be insanity. This is rare, as the patients usually quickly regain health when there is even temporary relief from pain.

TRIFACIAL NEURALGIA (Prosopalgia)

Is one of the most common forms of neuralgia. As the branches of the fifth nerve pass through bony canals they are much more readily compressed by a very slight swelling of their sheaths, and the pain thus produced is proportionately severe.

Exposure to cold, decayed teeth, and exostosis are likely to be among the causes; but disturbance of the stomach, intestines, generative organs, and other distant parts, may give rise to the disease.

The symptoms are such as have been already described. The pain is often *excessive;* spasm of all the muscles of the affected side of the face is excited by the agony. The painful points are the *palpebral*, at the external part of the upper eyelid; the *supra-orbital*, where the frontal nerve turns up over the edge of the brow; the *nasal*, at the upper part of the nose; the *malar;* the *infra-orbital*, at the point of emergence of the infra-orbital nerve; the *mental*, where the inferior maxillary nerve ends in the mental and passes out from the foramen. There are less important points mentioned: the *ocular;* the *labial;* the *lingual;* the *parietal*, which is common with the cervico-occipital neuralgia. The *points apophysaires* are found over the spinous processes of the first and second cervical vertebræ and the occipital protuberance.

The conjunctiva of the eye on the affected side, and sometimes of the opposite eye, may be deeply congested, tears may flow freely, and the nasal mucous membrane may secrete profusely. The pupil is often dilated.

CERVICO-OCCIPITAL NEURALGIA

Is seated in the region to which the first four cervical nerves (cervical plexus) are distributed. This includes the back and side of the head as far forward as the ear, the neck and apex of the shoulder, and posterior part

of the lower jaw. The painful points are the *occipital*, between the mastoid process and the first vertebra; the *mastoid*, over that process close to the ear near the exit of the seventh nerve; the *parietal*, in common with trifacial neuralgia, and sometimes the rim of the ear is tender. The *points apophysaires* are over the four upper cervical vertebræ.

It is important not to mistake the pain caused by caries of the upper cervical vertebræ for this form of neuralgia.

When the nerves of the brachial plexus are involved we have *cervico-brachial* neuralgia. This is more frequently traumatic in origin. The painful points are found in the axilla, over the median nerve at the elbow, the ulnar just above the elbow, the radial where it follows round the humerus, at the lower angle of the scapula, and at the lower end of the ulna. The *points apophysaires* are over the lower cervical and upper dorsal vertebræ.

Many times there is really chronic neuritis as cause of the pain.

DORSO-INTERCOSTAL NEURALGIA

Is a very frequent form. The thoracic nerves are involved; it is rather more frequent on the left side; it is one of the accompaniments of various pulmonary diseases; is common in phthisis. Herpes zoster is very common around the chest; it may be excited by disturbances of the abdominal viscera, especially of the stomach.

The painful points are found just to the side of the vertebræ; then near the center of the course of the intercostal nerves; and, anteriorly, the region of terminal expansion, as Trousseau calls it. In tracing the nerve, the curvilinear course of the ribs should not be forgotten. The *points apophysaires* are found over the vertebræ corresponding with the affected nerve.

Infra-mammary neuralgia is one variety of inter-

costal, which gives much annoyance and causes much suffering, exciting, also, fears in the patient of inflammation of the breast.

When intercostal neuralgia is severe, the respiration is disturbed, rapid, and painful; the pain radiates to the arm; there is palpitation, and angina pectoris may be closely simulated.

LUMBO-ABDOMINAL NEURALGIA

Is the name given to the disease when the crural plexus is the seat of pain. The painful points are near the spinal column; just above the crest of the ilium, near its center; above the pubis; near the lower part of the rectus muscle; there may be points in the vagina or about the scrotum; over the anterior-superior spinous process; over the crural nerve as it passes out from under Poupart's ligament; on the inside of the knee-pan; and over the saphenous nerve in front of the ankle.

Sometimes congestion and hæmorrhages from the uterus and vagina seem to depend upon this neuralgia. The pain may lead to a suspicion of uterine disease. Lumbago may be distinguished by the fact that motion causes pain, which is absent during complete quiet. The pain caused by renal calculi may be mistaken for neuralgia.

SCIATICA

Is one of the most common, most rebellious of neuralgias; it is rather more frequent in men. In the majority of cases there is a neuritis. Between the paroxysms of pain there is usually an aching or burning sensation; a heavy, bruised feeling. Exertion will often cause a relapse.

The painful points are: near the sacrum; where the nerve emerges from the pelvis; near the great trochanter of the femur (Erb considers this the most constant); at the lower border of the gluteus muscle; in the popliteal space; frequently the whole course of the nerve in the thigh is tender, and it can be felt to be enlarged;

just below the head of the fibula; behind the outer ankle.

Wasting of muscular tissue of the leg is not uncommon in cases of long standing.

ÆTIOLOGY.—Anstie considered every case of neuralgia to be one of debility. This is somewhat too sweeping a statement, yet it is true of most patients suffering from this affection that they are below par in physical or nervous strength.

Heredity is an important factor in the ætiology of neuralgia. A large proportion of the patients are born with less than the normal nervous stamina; the parents may not have neuralgia, but the children have less than normal strength and vigor. Much might be written on this division, but it is scarcely necessary.

Women are more subject to some forms than men; some authors consider that the majority of patients are women, and this is probably true, though Axenfeld says the difference is less than is usually supposed.

Children rarely have neuralgia. The most susceptible age is middle life. The more marked the hereditary tendency, the earlier the disease will be likely to appear.

It is necessary only to mention that a predisposition to neuralgia may be fostered, or even developed, by hygienic surroundings, by overwork, by anxiety, grief, etc. These influences are much the more powerful in youth, yet do not lose their power in adult years.

Acute and chronic disease may bring the system into such a state of anæmia or debility as to greatly favor an outbreak of neuralgia.

Among exciting causes, "catching cold" may be the most frequent. Exposure to wet and cold is often mentioned by patients as the cause, and probably with truth. In such cases there is many times a rheumatic thickening of the nerve-sheath, and the disease is really a neuritis. It is not always possible to recognize this by the symptoms. The nerves most exposed to this

injurious influence are those of the face, the sciatic, and less frequently those of the arms. Many patients suffer from sciatica after sitting on a stone or a metallic seat.

Injuries of nerves or in their vicinity may be a cause of the disease. A slight injury may give rise to neuralgia only after months or years. Sometimes in such cases a change in the sheath, a thickening, has been slowly taking place, until at last, either spontaneously or as the result of a forgotten exposure, the attack of pain follows.

Tumors, exostoses, caries of teeth, disease in thorax or pelvis—these and other changes near nerves, by pressure or extension of inflammation, may cause pain, and the exciting cause may not be discovered, owing to its hidden location. Such cases are not, properly speaking, cases of neuralgia, yet the diagnosis of the true cause of the pain may be impossible.

Neuralgia may be excited in a reflex way by diseases of the viscera; this is especially so in regard to the genito-urinary and digestive organs, and in regard to caries of the teeth.

Several poisons, as mercury, copper, lead, alcohol, and tobacco, are both predisposing and exciting causes; the same may be said of malarial influences. Syphilis is an active agent in many instances, but not so often a cause of neuralgia as of headache.

PATHOGENESIS.—From the definition of neuralgia, it is evident that there can be no special pathological change in the nerves. Many times, doubtless, neuritis is called neuralgia, and that name is given to other conditions where organic changes are found. It is probable that very many cases of neuralgia ought to have another name; but this is the result of ignorance or carelessness on the part of the observer.

Anstie advocated that every case of neuralgia is in reality a case of anæmia of the spinal centers, with atrophy of the posterior nerve-roots. Chapman claims

that congestion of the spinal cord is the cause of the pain.

The truth is that we do not know definitively the nature of this affection. It would be easy to quote authors to show the varying views held in regard to this subject, but it would be of doubtful advantage.

DIAGNOSIS.—Erb gives six characteristic symptoms of neuralgia: 1. The pain is limited to a definite nerve-path, or area of distribution, and is usually unilateral. 2. Without any clear reason, the pain is either intermittent or distinctly remittent. 3. The pain is very peculiar and acute. 4. Certain spots in the course of the nerve, or in the area of its distribution, are very sensitive to pressure. 5. The pain is associated with certain sensory, motor, vaso-motor, and secretory phenomena. 6. The pain is not accompanied by any inflammatory or local symptoms, or any general disturbance of health at all corresponding with the amount of subjective disorder.

These six diagnostic marks are only presumptive of neuralgia. A very large proportion of cases are probably neuritis, and it may be as well to recognize the fact in practice.

The pains of locomotor ataxia are very much the same as those of neuralgia, and it is necessary to bear this in mind.

Caries of the vertebræ may give rise to pain which can be easily mistaken for neuralgia; the same is true of other diseases of the spine or spinal membranes, of cerebral lesions, and of malignant growths in the thoracic and abdominal cavities. In all these cases a careful study of symptoms may lead to a correct diagnosis; but without this, serious mistakes must be made.

PROGNOSIS.—Unless there is a neuritis or a morbid growth pressing upon the nerve, recent cases are usually readily relieved; but there is a great probability of return. The greater the number of attacks, and the more localized the pain, the less probable is relief, be-

cause under such circumstances there is almost always a neuritis or a perineuritis. The most rebellious cases are those affecting the fifth and sciatic nerves.

TREATMENT.—In neurotic subjects the treatment of neuralgia should begin before the pain appears—that is, much can be done to prevent its development. The young child should be fed and educated, its habits formed with special regard to the possible occurrence of nervous disorders. This regimen should be all the more carefully followed after the affection has appeared. Light, air, exercise, and food are necessary in large measure. Over-feeding is sometimes of great value; frequent feeding, hourly, is one means of inducing the system to receive more than it would from the usual number of meals.

So far as possible, all causes should be avoided; warm clothing will aid much; keep the feet dry.

Any disease which may give rise to neuralgia should, of course, be treated; this is self-evident, yet easily forgotten in the presence of the pain of an attack. Teeth should be looked after. Dyspeptic or other visceral disturbance, and uterine disorders, attended to. Alcohol and tobacco should be stopped.

It is scarcely necessary to mention that metallic poisons should be eliminated if possible. In syphilitic patients a corresponding treatment must be followed.

Internally, *quinine* is often of benefit, not only in cases due to malarial poisoning, but where there is no such taint. In recent cases, given in moderately large doses, five grains every hour until the head aches or the ears are affected, it will frequently cut the attack short. It is also useful as a tonic to prevent recurrence. Anstie found it useful in affection of the ophthalmic division of the fifth nerve.

Cod-liver oil, or other form of fat, cream, or butter, is very valuable. Many patients find the oil disagreeable. Begin with half or quarter of a teaspoonful, to which a little salt may be added; continue this dose,

after meals, until there is no regurgitation of the fumes of the oil; then the dose can be rapidly increased to a tablespoonful. Phillips's emulsion is very palatable.

Iron in its various preparations is indicated, especially in anæmic and chlorotic subjects. The tincture of the chloride is one of the most valuable forms. Anstie recommends very highly a mixture of this with strychnia, ten minims of the iron tincture with $\frac{1}{40}$ grain of the strychnia. The soluble saccharated oxide of iron, in doses of half a teaspoonful, is a very pleasant preparation.

Arsenic is valuable in the same cases as the iron; it is also useful in malarial cases.

Iodide of potassium, or the syrup of hydriodic acid, is useful not only in syphilitic neuralgia, but also in rheumatic; colchicum may help in such cases, though the bowels should not be too strongly acted upon.

Phosphorus has been very highly praised, especially by many English physicians. Mr. Thompson advised it in large doses, not less than $\frac{1}{30}$ grain; he advised $\frac{1}{15}$ grain every four hours; after six doses, $\frac{1}{12}$ grain at the same interval. After forty-eight hours, if no result, he thinks some good may be effected by increasing the dose still further. This dose rarely causes trouble, yet occasionally a patient is unusually susceptible, and acute poisoning has been observed; caution is, therefore, necessary.

Gelsemium, fluid extract, in ten- to twenty-minim doses, or tincture, in half-drachm doses, is very serviceable in facial neuralgia, and perhaps in intercostal and ovarian. The preparations of this drug are sometimes inert. The dose may be repeated every half-hour, but it is prudent not to give more than three doses so near one another.

Croton chloral has been used in facial neuralgia with very good results by many; but I have never seen any special benefit from it.

Aconitia is a very valuable drug; it is especially

useful in angina pectoris, in intercostal and facial neuralgia, but is of benefit in any form of reflex or constitutional neuralgia. The pure alkaloid, made by Duquesnil, crystallized, should be used, or the effect will be uncertain. The dose varies from $\frac{1}{150}$ to $\frac{1}{120}$ grain. Dr. E. C. Seguin recommends the following formula:

℞ Aconitiæ (Duquesnil's).....gr. $\frac{1}{10}$ – $\frac{1}{6}$;
Glycerine } .....āā fl ʒ j;
Alcohol }
Aq. menth. pip .....q. s. ut ft. fl ℥ ij. M.

S. A teaspoonful two or three times a day, on an empty stomach.

This may be given even more frequently, as often as every two hours, if the effects are carefully watched; so soon as the pulse is affected, or there is tingling of the lips, tongue, or fingers, the drug must be discontinued.

Féréol found sulphate of copper, ·05 to ·10, of value in epileptiform neuralgia.

Other drugs that have been used are chloride of gold and sodium, nitrite of amyl, chloride of ammonium, strychnia, which is praised by Anstie, and phosphide or oxide of zinc.

During the attack of pain, to relieve the distress it may be necessary to use morphia or other preparation of opium. Morphia subcutaneously is the most efficacious; but care is needed lest the morphia habit should be formed. In old and obstinate cases the smallest dose which will relieve the pain, $\frac{1}{12}$ or $\frac{1}{20}$ grain, should be used only when it is necessary to give such relief. In recent cases one large dose will sometimes work a cure.

Atropia subcutaneously will frequently give as much relief as morphia; $\frac{1}{60}$ grain is usually the largest dose necessary. Anstie recommends atropia, especially for ophthalmic neuralgia.

Chloroform, in doses of five to ten minims, injected

under the skin in the vicinity of the affected nerve, often gives relief.

Schultz used carbolic acid, two to one hundred of distilled water, subcutaneously, injecting from a quarter of a drachm to a drachm of the solution.

Eulenburg injected a one-per-cent solution of osmic acid with benefit in recent cases, which were probably neuritic or perineuritic.

The subcutaneous injection of a drachm of hot water near the nerve—of course not so hot as to scald the tissues—will frequently give as much relief as small doses of morphia. Acupuncture, passing a needle into the skin until the point is near the nerve, will give relief in many cases. The needle should be worked in slowly and gradually, as near the nerve-trunk as possible; if its point can just touch the nerve, which may be known by the peculiar sensation, and then be slightly withdrawn, the result is the better. This probably is most efficacious in recent cases.

Various external applications will soothe the pain during an attack, and render the use of morphia less necessary. Hot water is useful; so, too, is spirit of turpentine. Chloroform, diluted more or less with alcohol, applied on a piece of flannel and covered with a towel wet with water, eases the pain. Generally one part of chloroform to seven of water is a good mixture, though one stronger is often better. Veratria ointment can be used, but aconite is better. The tincture of aconite may be used freely, or the ointment may be applied over the affected surface. The ointment of aconitia, if made with Duquesnil's aconitia gr. j to ʒ j lard, will be stronger than the regular officinal ointment. Care must be taken not to get any of this into the eyes, nose, or mouth, and not to rub it where the skin is abraded. The person who applies it should not use his uncovered hands. A portion half as large as a small pea is sufficient for one application. I know of no simple external application of equal value.

Blisters over the tract of the nerve, especially over the *points douloureux* and over the *points apophysaire*, not only give relief, but often effect a cure. The blisters need not be large ; an inch by an inch, or inch and a half, is sufficient. They may need to be repeated over different points, or near the same spot.

The actual cautery over the same spots may be even more valuable than blisters—may give immediate relief without so much discomfort.

Electricity is a most satisfactory agent in many cases. Sometimes the faradic, applied through a wire brush to the seat of pain, gives immediate relief. The galvanic current passed through the affected nerve or limb is usually the better. It should be used daily, with as little shock or variation of strength as possible.

Vibration communicated to the nerve at the seat of pain, by rapid percussion over the tender points, sometimes gives permanent relief. The percussion may be made by means of rubber balls attached to handles, or by mechanical contrivances.

The application of ice by means of the rubber ice-bag to the spine, either a portion or the whole, as advised by John Chapman, is soothing and grateful. If properly applied, it has a tendency to restore warmth to the feet, and will relieve pain. It should be applied from thirty to sixty minutes several times a day. Occasionally Chapman uses hot water over the upper part of the spine in facial neuralgia with hyperæmia or swelling of the face.

Surgical operations are sometimes needed to cure neuralgia ; these are excision of portions of nerves, which has been frequently done for facial neuralgia, and stretching of nerves. The latter is of comparatively recent date, and has given very good results. Patruban has tied the carotid for facial neuralgia with success in many cases.

CHAPTER XXIV.

LOCAL AND POST-FEBRILE PARALYSES.

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PERIPHERAL PARALYSIS.

By peripheral paralysis may be understood paralyses which depend upon lesions of the muscles themselves, or the nerves after they leave the spinal cord.

ÆTIOLOGY.—Among the causes may be mentioned injuries from falls or blows, or wounds ; pressure upon nerves, either by the position of the limbs or by burdens carried so as to press upon the nerves ; or by tumors, or other products of disease.

Cold is a common cause of certain forms of paralysis, so-called rheumatic paralysis. Disease of neighboring parts, even when the nerves are not directly

implicated, may give rise to loss of motion, as in hip-disease, there is loss of power in the muscles of the leg, which may be attended with atrophy.

Acute diseases are many times followed by paralysis. Certain poisons, as lead, arsenic, and some vegetable poisons, cause paralysis, apparently due to disturbance of the nerves. The same, also, may be said of syphilis, though with this there is generally a formation of new tissue around the nerves.

Over-exertion of limbs, exhaustion, may lead to temporary paralysis, or even to a more serious and more permanent loss of motion.

In some cases the paralysis is spoken of as reflex, as if it arose from disease of certain organs by reflex action through the spinal cord. It is rather doubtful whether such paralyses are really reflex, as is claimed, and do not, rather, depend upon disease either of the nerves themselves or of the spinal cord.

SYMPTOMS.—As most of the nerves are mixed nerves, there is usually loss of motion and disturbance of sensation. Sometimes pains or peculiar numb feelings and unpleasant sensations precede any loss of power; but very soon, if not at the same time with the disturbance of sensation, the patient recognizes that there is loss of power. He finds that he can not perform certain acts as readily as formerly.

The nature of the disturbance of motion will depend, of course, upon which nerve is affected. If the paralysis is not complete, the ordinary reflexes may not be seriously impaired; but if there is entire loss of either motion or sensation, both the superficial and deep reflexes will disappear.

In every case of paralysis depending upon a lesion of the nerve itself, the electrical reactions will be such as have been described under the name of the reaction of degeneration.

Frequently the paralysis is attended with a moderate degree of swelling of the affected limb, due to a

loss of tone in the blood-vessels on account of paralysis of the vaso-motor nerves, which accompany the nerves of motion and sensation. For the same reason there may be at first a rise of temperature in the limb, though subsequently the temperature is lowered, and the limb may have a cyanotic appearance.

Certain trophic changes are found after injuries and serious lesions of the nerves. These affect the nerves themselves, the muscles, and the skin. The nerves undergo a degeneration; the medullary sheath breaks up into granular material, which is absorbed, the axis cylinder also undergoing a change. The muscles lose their striated character, and after a while are changed into fatty *débris*, which is finally absorbed. Accompanying these changes, there is usually more or less multiplication of nuclei.

The skin may be covered with an erythematous eruption, or the eruption may be vesicular. Herpes and eczema are not uncommon. Sometimes the skin is thickly covered with minute scales of epithelium, which can be readily brushed off.

Mitchell has described the glossy skin which is found frequently after nerve-lesion. This is most commonly seen in the fingers, perhaps in the foot. The skin has a peculiar shiny appearance, without wrinkles, without hairs. When the fingers are affected, they taper off to their ends, and it is very common to have a severe pain as an accompaniment of this condition, causalgia. Occasionally ulcers form, mal perforant, though this is comparatively rare. The nails become brittle, rough, and deformed. The hair may fall out, or grow to an inordinate length, and sometimes loses its color, becoming gray or white.

Diagnosis. — The diagnosis between paralysis of peripheral origin and of central origin must be made in large part from the other symptoms. Electricity is the most valuable agent in forming the diagnosis. If there is the reaction of degeneration, it is certain that either

the nerves are diseased or the large cells of the anterior cornua of the spinal cord have undergone degeneration. If, then, a disease of the spinal cord can be excluded, the diagnosis is clear.

Multiple neuritis has been already considered.

PROGNOSIS.—A very large number of cases of peripheral paralysis recover completely. The more severe, however, the original injury, or the more complete the degeneration of the nerve caused by disease, the slower will be recovery, and the more likely permanent impairment of motion will result.

Among the most favorable cases are those which arise from simple pressure, from rheumatic disturbance, or from poisons and from syphilis.

Even where the reaction of degeneration is found, recovery is not to be despaired of, and treatment should be persevered in for many months. The less perfectly the reaction of degeneration is established, the more favorable is the indication. When secondary contraction has set in, or when the reaction of degeneration has evidently continued for many months, the prognosis is very unfavorable.

TREATMENT. — The treatment of these paralyses must be directed first, of course, to a removal of the cause if possible. Injuries and other diseases should receive their appropriate treatment. If tumors can be removed without destruction of a nerve, in course of time the paralysis will disappear.

In cases of exhaustion, rest is sometimes sufficient for recovery ; if not, then the same means should be used as in other cases of protracted paralysis. In cases of syphilis, the anti-syphilitic treatment should be vigorously pursued. Warmth to the limb is quite important. Electricity is of more value than any other agent. The galvanic current should be used, the current being slowly interrupted, the negative pole being placed over the motor points of the affected muscle. In cases of paralysis due to pressure or exhaustion, or after acute

diseases, this is usually sufficient. In cases of rheumatic paralysis it may be well, also, to paint over the affected nerve with tincture of iodine. Where, however, there is reason to suspect that a neuritis has occurred, small blisters placed over the nerve, as described under neuritis, will hasten the cure.

Internal remedies are of little or no value so far as the paralysis is concerned; they may be required, however, for the general health and condition of the patient.

SPECIAL FORMS OF PARALYSIS.

The nerves which move the eye are frequently subjected to pressure and injury from syphilitic disease of surrounding parts, especially of the membranes of the brain; and, as these nerves pass through the bony canals at the base of the skull, they are easily compressed, not only by such growths, but also by the influence of cold, producing a congestion and swelling and inflammation of the surrounding tissues.

When these nerves are paralyzed, vision will be more or less interfered with. If the third nerve is affected, the drooping of the eyelid and loss of power of accommodation may disturb vision, even when the motions of the eyeball seem to be perfect. When the muscles of the eyeball are paralyzed, in consequence of injury to their nerves, there is more or less immobility of the eye, and hence strabismus results. A very careful description of the various forms of strabismus may be found in Ziemssen's "Cyclopædia," vol. xi, or in Ross's work on the "Diseases of the Nervous System," or in books on diseases of the eye.

The *seventh* nerve, the facial nerve, is perhaps more frequently affected by the so-called rheumatic paralysis than any other nerve of the body. Passing through a bony canal near the ear, being very superficial where it leaves that canal, it is specially exposed to such a disturbance. Draughts of air while riding, or sitting at an open window, or other exposure of one

side of the face, may be sufficient to give rise to this paralysis.

As the seventh nerve passes through the temporal bone, separated by a very thin lamina of bone from the tympanic cavity of the ear, it is very liable to disturbance in cases of inflammation of the middle ear. New growths in the ear may also, by pressure, cause absorption of the thin layer of bone, and press upon the nerve. Blows upon the side of the head, and other injuries, may likewise result in facial paralysis.

As the facial nerve is at its origin exclusively a nerve of motion, when it is paralyzed the symptoms are chiefly those of loss of motion; in its course, however, through the Fallopian canal it receives a few branches—one of special sense, of taste, and another, near its exit from that canal, of common sensation. The auricular branch, from the vagus, passes through the temporal bone, quite near the facial nerve, and gives a small branch to it. Probably in consequence of the proximity of this nerve, many times the first symptom of facial paralysis is pain in the region of the ear, and generally there is more or less discomfort, if not actual pain, during the early part of the disease.

The most common symptom in paralysis of the seventh nerve is loss of power in all the muscles on that side of the face. Not only those of the lower part of the face, which are affected in cases of cerebral disease, but also the muscles of the forehead, and the orbicular muscles of the eyelids, are paralyzed; hence the eye remains partly open, and even in sleep is not entirely closed, although the eyeball may turn upward, so that the pupil is covered. The lids are not closely applied to the eyeball; hence, the tears do not find a ready entrance to the nasal duct, and the eye waters continually.

There may also be a loss of taste in the anterior part of the tongue, and it has been claimed that the secretion of saliva is less on that side.

Sometimes the velum palati is affected and hangs down loosely on the paralyzed side, and, when the muscles are brought into action in speaking, the action being much greater on the sound side, the palate is drawn over toward that side. Sometimes the uvula has an oblique direction. Sense of hearing may be somewhat more acute on the paralyzed than on the opposite side.

The tongue is protruded straight; but sometimes, owing to the uneven position of the lips, the tongue appears to deviate. Careful observation of its position relative to the teeth will prevent any error.

The reaction of degeneration is found in the muscles. The electrical reaction is of value as aiding in a formation of prognosis. In very mild cases the reaction of degeneration may not set in; in cases of medium severity, the extreme form of the reaction of degeneration will not appear.

It is possible, from certain peculiarities of the paralysis, to diagnosticate very closely the seat of the lesion. Erb has briefly stated the points of diagnosis, which may be summarized as follows:

1. If there is complete paralysis of all the branches, if there is no disturbance of taste or hearing, and no paralysis of the palate, and if the electrical reaction is normal, the trunk of the facial is affected external to the Fallopian canal.

2. Paralysis of all the external branches, with reaction of degeneration and absence of disturbance of taste, shows that the cause of the paralysis is within the canal and below the origin of the chorda tympani.

3. With the same symptoms and disturbance of taste, the cause is between the origin of the chorda tympani and the ganglion geniculatum. If the hearing is abnormally acute, the lesion must be above the origin of the stapedius nerve; otherwise below it.

4. If, with the above symptoms, there is paralysis of the velum palati, the lesion is in the vicinity of the ganglion geniculatum.

5. If all the above symptoms, except disturbance of taste, are present, and especially if there is also dullness of hearing and tinnitus, the lesion is at the base of the skull; and this is rendered still more certain if other cranial nerves are affected.

6. Erb states that if the same symptoms as in No. 5 are present, except simple diminution of the electrical instability instead of reaction of degeneration, and especially if unusual or crossed reflex action be present, lesion of the facial nucleus may be diagnosticated; and this is yet more certain if other cerebral nerves having their origin in this part are also paralyzed.

After facial paralysis has continued for two or three months, it is not uncommon to have secondary contraction set in, which delays recovery. The face, when at rest, may then have a more natural appearance; but, when the mouth is moved, the difference in the two sides becomes apparent. Erb explains this condition as due to the changes that occur in muscles where there is the reaction of degeneration.

Diagnosis. — The diagnosis of peripheral facial paralysis, from that caused by central lesion, is of much importance, especially for the comfort of the patient.

The reaction of degeneration is one of the most important aids; in cases due to lesions of the brain, as a rule, only the lower branches of the nerve are affected, those that go to the eye and forehead acting normally. In cases of tumor or other disease within the skull, pressing upon the nerve just before it leaves the skull, there are general symptoms of tumor as well as those relating to other nerves, especially the auditory, which will aid materially in a diagnosis.

Paralysis due to diseases of the ear must be diagnosticated by the symptoms which are more particularly referable to the ear. The large majority of cases are due to the action of cold, so-called rheumatic paralysis, and the history will not always aid in forming a

diagnosis, as patients very often are not aware that they have been exposed.

PROGNOSIS.—In the lighter forms of rheumatic paralysis the majority of cases get well. Some of the severer cases recover without special treatment; yet, generally, if there is no treatment, a certain amount of deformity remains which no subsequent treatment benefits. The best results can be obtained by the early use of appropriate measures.

The prognosis in cases arising from disease of the ear depends entirely upon the nature of that disease and the amount of mischief which has been caused to the facial nerve. The prognosis in cases of disease within the cranium must be guided by the nature of that disease.

TREATMENT.—It is necessary to say but very little in regard to the special treatment of facial paralysis. In the rheumatic form, painting over the neck just below the ear and behind the ear with tincture of iodine may be of some benefit. Electricity, the galvanic current by preference, interrupted at short intervals, is of most value. The internal use of remedies is of no value except in syphilitic cases.

PARALYSIS OF THE BRACHIAL PLEXUS.

There is nothing peculiar in the symptoms found in paralysis of the brachial plexus. A knowledge of the distribution of the nerves to the muscles will show what nerves are specially affected, and the resulting paralysis or deformity depends upon which muscles are affected.

Among the most common causes are dislocation of the humerus, the head of the bone pressing upon the nerves in the axilla; pressure of a crutch upon these same nerves; pressure upon the radial nerve as it passes around the lower part of the humerus. This is most frequently found in patients who have fallen asleep upon their arm, especially if the arm rests upon

any hard substance, and is more likely to occur when the sleep is very heavy, or from intoxication. Carrying burdens upon the arm, the hand resting upon the hip, may also be a cause.

Among infants, paralysis of these nerves is sometimes found as the result of delayed labor; the pressure upon the nerves in the neck, especially by forceps, may be a cause; or, if the arm is drawn down in breech presentations, the nerves may be injured in the operation. This is the so-called obstetric paralysis of infants.

Other forms of peripheral paralysis require no special mention.

PARALYSIS AFTER ACUTE DISEASES.

Many acute diseases are sometimes accompanied with or followed by paralysis. Apparently the nature of the lesion which caused the paralysis is different in different cases.

Among the diseases which are most frequently thus accompanied with local or more general paralysis may be mentioned small-pox, measles, scarlatina, typhoid fever, dysentery, sometimes diarrhœa, cholera, pneumonia, and diphtheria.

Generally, except in diphtheria, the paralysis occurs during the course of the disease, and may be found accompanying apparently light cases as well as the more severe.

Many times it seems as though an unusually high fever, perhaps of very short duration, were the exciting cause of the paralysis.

Several times changes have been found in the spinal cord. This is especially true of small-pox. In other cases the paralysis seems to be of peripheral origin. The legs are more frequently attacked than the arms.

The prognosis in almost all these cases is comparatively favorable unless the spinal cord is the seat of the disease; yet occasionally serious injury is done to the

nerves or nerve-centers, and recovery is imperfect, the patient remaining more or less helpless during the rest of life, with atrophy of the paralyzed muscles.

The treatment is such as has been already indicated in speaking of peripheral paralysis, or such as is required in corresponding cases where the nerve-centers are affected.

DIPHTHERITIC PARALYSIS.

Diphtheritic paralysis requires rather more attention than has been given to those arising from other acute diseases. It occurs after the primary disease has ceased. The patient is thought to have recovered health, and from eight to thirty days afterward the nervous disturbance is first noticed. The paralysis may appear after either severe or light cases of diphtheria. When it occurs soon after the primary disease, it is more gradual in its onset, and successive nerve-regions are affected one after the other.

Diphtheria is most common between the ages of two and twelve. The paralysis following diphtheria is most common between the ages of ten and eighteen. It is impossible to foretell whether or not the patient will have paralysis following diphtheria.

SYMPTOMS.—The temperature often rises for a short time before the occurrence of the paralysis. In the majority of cases there is first a slight change in the voice, which becomes nasal. The velum palati and the muscles of the larynx being paralyzed, there is regurgitation in swallowing liquids. When the attempt is made to swallow food, a portion passes down the wrong way into the larynx, causing choking and coughing.

Sometimes disturbance of sight is the first symptom, there being dimness or partial loss of vision on account of paralysis of the muscles of accommodation. Strabismus may be caused by paralysis of the motor muscles of the eyeball.

Frequently the legs lose the power of motion; the

patient is unable to walk. Next in frequency the arms and hands are affected. Occasionally there is paralysis of the diaphragm, and less frequently of the heart. It is rare to have a case in which the paralysis is general. Usually one or two limbs are most affected, the others being only slightly affected, or escaping entirely, and in the majority of the cases the paralysis is limited to the velum palati and the larynx.

The reaction of degeneration is very common in diphtheritic paralysis; indeed, in the majority of cases the loss of power is due to a lesion of the anterior roots of the spinal nerves. It is supposed, however, that the disturbance in the throat is due to a lesion of the nerve as it passes near the seat of the original disease.

Sensation is only exceptionally disturbed. Very rarely, instead of loss of motor power, severe pain is felt in the course of certain nerves.

DIAGNOSIS.—The history of a previous sore throat or attack of diphtheria is sufficient to show the nature of the subsequent nerve-lesion. Without such history it would be impossible to recognize the cause of the paralysis, though the nasal voice, the regurgitation of food, and the choking in swallowing, might lead one to suspect that there had been a diphtheria which had been overlooked.

PROGNOSIS.—The prospect is generally favorable, although occasionally patients die from an extension of the paralysis to the heart or muscles of respiration, and sometimes from inhalation's pneumonia, due to the passage of food into the bronchi. Except in such cases, the patients almost invariably recover under proper treatment.

TREATMENT.—Where deglutition is seriously interfered with, the greatest care will be necessary in the treatment of the patient to avoid the passage of food into the trachea.

Usually a soft solid can be swallowed better than

liquid food; but in many cases it is necessary to omit feeding the patient by the mouth for a while, and in that case food should be given by enemata, in the way which has already been described. If necessary, a tube may be passed down the throat into the stomach, and the patient can be thus supported by artificial feeding.

Paralysis of the limbs should be treated by keeping the limbs warm, by massage, by stimulating bathing, as with salt-water, warm rather than cold, and by the use of electricity.

Iodide of iron is one of the most valuable tonics for such patients, and cod-liver oil, if it can be taken, is of great use. Otherwise, except as indicated by the patient's general condition, no special treatment is necessary.

CHAPTER XXV.

SPASM.

NOTHNAGEL, Zur Lehre von klonische Krämpfe. *Virch. Arch.*, xlix, pp. 267, 290.—MITCHELL, S. W., On Functional Spasm. *Am. Jour. of the Med. Sci.*, Oct., 1876, p. 321.—MILLS, C. K., Spasms of the Muscles supplied by the Spinal Accessory Nerve. *Am. Jour. of the Med. Sci.*, Oct., 1877, p. 425.—REMAK, E., Zur Pathologie und Therapie localisirte Muskelkrämpfe. *Berl. kl. Wochenschr.*, May 23, 1881, p. 289.—JONES, C. H., Clinical Lecture on a Case of Spasmodic Disorders of the Lower Limbs. *Brit. Med. Jour.*, July 2, 1881, p. 41.—ROBINSON, E., Cases of Telegraphists' Cramp. *Brit. Med. Jour.*, Nov. 4, 1882, p. 880.—SINKLER, Spinal Accessory Spasm. *Med. News*, April 19, 1884, p. 453.—POORE, G. V., An Analysis of Seventy-five Cases of Writers' Cramp. *Med.-Chir. Trans.*, 61, 1878, p. 111.—*Ibid.*, Writers' Cramp. *Practitioner*, 1873.—ALTHAUS, J., On Scriveners' Palsy. London, 1870.—VIGOUROUX, R., Du traitement de la crampe des écrivains par la méthode de Wolff. *Le prog. méd.*, x, 1882, p. 37.—THOMSEN, J., Tonische Krämpfe in willkürlich beweglichen Muskeln in Folge von ererbter psychischer Disposition. *Arch. f. Psych.*, vi, 1876, p. 702. — BALLET et MARIE, Spasme musculaire au début des mouvements volontaires. *Arch. de nevrol.*, Jan., 1883, p. 1.—RINGER, SYDNEY, On the Nervous or Muscular Origin of Certain Spastic Conditions of the Voluntary Muscles. *Lancet*, Nov. 1, 1884, p. 767 *et seq.*

The convulsive actions included under the name spasm are of several varieties.

Tremor is a very fine spasm of the muscles, which produces a trembling of the limbs, sometimes scarcely perceptible. It is rather a symptom of several morbid conditions than a disease of itself.

Tonic spasm is a name given to the spasm when a muscle is contracted continuously without relaxation.

This, also, is rather a symptom than a disease, being found more especially in tetanus and spinal meningitis. It is sometimes difficult to recognize the difference between a tonic spasm and what is called contracture of the muscles; in fact, the latter may be looked upon in its earlier stages as simple tonic spasm, but later there is usually a change of structure in the muscles, and, the contracture becoming permanent and depending in large measure upon this change of structure, can no longer be called a tonic spasm.

Clonic spasm is a name given to the convulsions which are attended with a rapid contraction and relaxation of muscles. When these clonic spasms are extreme, and large groups of muscles are attacked, the name convulsions, or eclampsia, is used rather than clonic spasm, the latter name being reserved for the less severe and less extensive convulsions.

In the following descriptions, clonic spasms will be chiefly considered.

These are generally reflex in their origin, depending upon the irritation of some sensitive peripheral nerve, possibly far from the seat of the spasm. They many times, also, depend upon lesion of the central nervous system, in which case they are simply symptoms of the disease which gives rise to them. All such cases of spasm of central origin have been considered under diseases of the nerve-centers.

It is not necessary to mention in detail spasms of all the various nerves. A few have such peculiar characteristics, and occur so frequently, as to be deserving of a separate mention.

SPASM OF THE FACIAL NERVE.

Irritation of the facial nerve in its course through the temporal bone, or at the base of the skull, may give rise to a spasm of the muscles supplied by it. Slight spasm of the muscles of the face is sometimes seen after facial paralysis. Irritation of the fifth nerve may,

by reflex means, also produce spasm of the facial muscles. Very severe convulsive action of these muscles may accompany the pain in severe cases of trifacial neuralgia; or, without pain, decayed teeth, inflammation of the conjunctivæ, abscesses about the face or in the cavity of the mouth, the influence of very bright light upon the eyes, as from the molten metal in a blast-furnace, may be causes.

An irritation of distant organs, as the intestinal tract, or the uterus, seems sometimes to be the starting-point of facial spasm.

This spasm is generally unilateral. All the muscles of one side of the face may be thrown into violent convulsions, producing the most ludicrous grimaces, lasting for a few seconds, relaxation being followed soon by another attack.

The series of attacks may continue for several seconds or minutes, when there is a period of rest until the next attack occurs. Or, instead of general spasm, one or a few muscles may be affected. There may be slight twitching about the mouth or face. It may seem almost as if the patient had simply acquired a habit of which he might be readily broken. These spasms, however, are very frequently involuntary and entirely beyond the control of the will.

Sometimes the orbicularis palpebrarum is exclusively affected; then the patient closes the eye violently, or simply winks rapidly. Occasionally the muscles of the forehead are also implicated.

Blepharospasm is a tonic spasm of the eyelids, the contraction of the orbicular muscle persisting sometimes for many minutes, or even hours. A bright light, an attempt to use the eyes for near vision, especially where great care is necessary in seeing small objects, as fine print, is sufficient to bring on an attack in those who are subject to this form of spasm. Sometimes a simple mental emotion will cause an attack.

Pressure upon certain parts of the face may have

the effect of relaxing this spasm, and occasionally such pressure will cause other facial spasms to cease. These points correspond to the painful points in facial neuralgia; or such points may be found within the cavity of the mouth, or over the back of the neck, or even in regions supplied by the brachial plexus of nerves. Patients frequently learn where these points are, and are able to cut short the spasm themselves.

TORTICOLLIS, OR WRY-NECK.

Slight attacks of wry-neck may follow exposure to cold, and is spoken of as stiff-neck. This may also be the cause of more severe attacks; it is said to arise also by reflex influence from irritation of the abdominal and pelvic viscera; in very many cases the cause is unknown.

Symptoms.—The muscles affected in this form of spasm are those supplied by the spinal accessory nerve, the trapezius, and the sterno-cleido-mastoid. When the latter is contracted, the head is drawn over so that the occiput approaches the shoulder of the affected side; the chin is turned toward the opposite side, and slightly upward. When the trapezius is affected, the head is drawn backward, and inclined toward the affected side. There is no rotation. Sometimes the shoulder is raised. The spasm usually begins so quietly and mildly that the motion at first is not noticeable. Soon the action of the muscle becomes stronger, and then the head is turned and jerked in a very distressing manner.

The spasms occur in separate paroxysms, lasting for a few seconds or minutes, frequently repeated, at times, with long intervals of rest. Sometimes other muscles are also affected besides those above mentioned.

Patients learn to support the head by their hands, and forcibly to restrain the unpleasant action of the muscles. In violent cases, however, this manœuvre is only partially successful.

Sleep may be interfered with. It may be difficult for the patient to take food. The mental influence of the affliction is such as to cause depression of spirits and diminish the appetite; the patients may become thin and emaciated in consequence. Slight cases, however, have no effect upon the general health.

These muscles are occasionally affected with tonic spasm, in which case the head is firmly fixed in the positions above mentioned.

DIAGNOSIS AND PROGNOSIS.—The diagnosis of these spasms is not difficult. The principal mistake would be, in cases of tonic spasm, to consider that the antagonistic muscles were paralyzed. Spasm of other muscles of the neck may be mistaken for those already mentioned. When the splenius capitis is affected, the head is drawn backward and toward the affected side, the chin is somewhat depressed and directed toward the side of the spasm, and a hard ridge can be felt where the splenius appears beneath the anterior border of the trapezius. Spasm of the obliquus capitis inferior turns the head around its vertical axis without elevation of the chin or depression of the mastoid process. Spasm of the deep muscles of the neck draws the head strongly backward if bilateral, or toward the affected side when unilateral. (Ross.)

In both facial spasm and torticollis the prognosis is very unfavorable. Very few cases recover.

TREATMENT seems to be of very little value. In some cases electricity is successful. The galvanic current should be applied to the affected muscles, and the faradic current to their antagonists.

Apparatus to produce permanent compression over the points of arrest has been tried, in some cases with success.

Stretching of the spinal accessory, as it runs along the posterior edge of the sterno-mastoid, has been employed with success in curing the spasm of torticollis.

Division of muscles, or their tendons, has been em-

ployed in some cases with advantage, especially in the tonic form of spasm. Counter-irritation over the nerves supplying the affected muscles, by means of blisters or the actual cautery, may be of value.

Of internal remedies, the most successful have been phosphate of zinc, sulphate of zinc, bromide of potassium, arsenic, and especially subcutaneous injection of atropia.

SPASM OF THE DIAPHRAGM, if tonic, may be the cause of death, and is always a serious affection. It rarely occurs independently of other disease.

CLONIC SPASM OF THE DIAPHRAGM, OR HICCOUGH, may be a light affection, with which every one is familiar, or it may be a serious and obstinate symptom of disease of the viscera or of the nervous system.

It is frequently associated with gastric, intestinal, and hepatic diseases, and in many cases is a symptom of bad omen, indicating the approaching fatal termination. When existing independently of serious disease, it is often obstinate, resisting treatment.

The galvanic current applied along the course of the phrenic nerve, or, locally, over the insertions of the diaphragm; the faradic current applied to the epigastrium; hot applications over the epigastrium—may be of benefit. Subcutaneous injections of atropia are especially useful, and those of morphia are of benefit.

THOMSEN'S DISEASE.—A form of spasm has lately been described which is of more interest as a curiosity than practically as a disease. It consists of a stiffness and rigidity of the limbs, especially the legs, appearing only when an attempt is made to change the position, as in rising from a sitting posture, or commencing to walk after standing still. When the patient wishes to take a step, the leg is raised slowly, and with evident exertion, to an angle of about 120°. Standing very unsteadily on the other, the patient sets this down in nearly the same angle. If he then tries to raise the

other leg, he will fall, generally on the knee, rarely backward; or, if he does not fall, he will walk unsteadily, the hips and knees bent at an angle of 120°, and remaining flexed while walking. After a few steps the gait improves, and soon the patient can walk naturally. Passive motion meets with resistance which is more marked the more rapid the motion. The arms and hands, or even face, are sometimes affected, and there is a similar difficulty in executing any movement as is found in the legs.

The affected muscles are unnaturally large and hypertrophied; there is no increase of fat; no reaction of degeneration, though the electrical reaction may be less than normal. There is, as a rule, no pain, no cramp.

The disease usually commences very early in life, perhaps is congenital, and in Thomsen's case seemed to be a family trait.

Several authors locate the affection in the muscles.

PROFESSIONAL CRAMP.

Under this term may be included the difficulty which is found by writers and pianists, telegraphers, and other persons in performing the various acts required by their profession, in consequence of spasm or weakness of the muscles engaged.

ÆTIOLOGY.—The cause of this affection is usually an excessive use of the hands and fingers, long continued, in persons of a neurotic temperament, or who have been weakened by previous disease or debility. Occasionally injuries, sprains, blows upon the hand or arm, and exposure to cold, act as causes.

SYMPTOMS.—At first the difficulty experienced is very slight, consisting simply in a little awkwardness of motion or stiffness of the fingers; sometimes a mere unpleasant sensation, hardly sufficient to be called pain, indicates the approach of the trouble. The arm and hand become more easily and quickly tired. Gradually these symptoms become more marked; the hand-

writing becomes decidedly poor; spasms appear in different muscles, and sometimes the thumb and fingers are so strongly flexed that the pen is pressed against the paper and broken; at another time the extensor muscles are affected, and the fingers open, allowing the pen to drop.

There is rarely decided pain, but a sense of weariness and exhaustion. Sometimes, however, the discomfort is very great, and extends up the arm as high as the shoulder. Occasionally pain is felt along the course of the nerve-trunks.

When the disease is well advanced, the patient can write at most only one or two words—perhaps can not even sign his name.

The arm and hand can be used for the ordinary purposes in life; even laborious manual work can be performed without difficulty; but, as soon as the patient undertakes to employ the fingers for any delicate operation, as writing, playing the piano, or sewing or knitting, the symptoms immediately reappear.

The electrical reaction of the muscles is increased in the early stages of the disease, and it is only after a long time that any diminution of the reaction can be recognized.

Persons affected with this disease may learn to write with the left hand: but in so doing should be careful not to overtax that hand; if they do, the same symptoms may appear on the left side; if careful, they may be able to use the left hand without difficulty.

Views differ somewhat as to the nature of this affection. Althaus looks upon it as due to fatigue and functional irritability of the co-ordinative centers in the upper portion of the spinal axis. Ross is inclined to look upon the main lesion as situated either in the ganglion-cells of the spinal cord, or the nerves when the electrical reactions are diminished; in the cortex or conducting-path above the spinal level when the electrical reactions are increased. Others consider that the

seat of the disease is in the muscles or the terminal nerve-apparatus.

The diagnosis presents no special difficulties. The prognosis is far from favorable except in very recent cases.

TREATMENT.—Entire rest from the cause of the disease is absolutely necessary for recovery. This rest must continue for many months—six at least.

The galvanic current frequently gives good results. It is applied in various ways by different observers. One pole should be placed on the neck over the spinal column, and the other applied over the affected muscles and nerves of the arm.

Erb recommends the application of the galvanic current to the head (transversely, longitudinally, obliquely); also to the cervical sympathetic.

Showering the arm with hot or cold water sometimes gives relief.

Wolff has obtained excellent results by the combination of gymnastics and massage. He uses both active and passive motion, exercising the affected muscles until they are fatigued. The massage is applied to the fingers, hand, wrist, and arm. He uses percussion with the ulnar border of the hand over the affected muscles. His method has attracted much attention, and several articles have appeared in recent medical journals describing the process.

CHAPTER XXVI.

DISEASES OF THE SYMPATHETIC.

WRIGHT, H. G., Headaches: their Causes and their Cure. London, 1865.—SMITH, A. A., The Therapeutics of Headache. *Med. Record*, Aug. 5, 1876, p. 503.—WOAKES, E., The Ætiology and Treatment of Occipital Headache. *Practitioner*, April, 1878, p. 263.—WARNER, F., Recurrent Headache in Children. *Brit. Med. Jour.*, Dec., 6, 1879, p. 889; *Brain*, Oct., 1880, p. 309.—DAY, W. H., Headaches: their Nature, Causes, and Treatment. Philadelphia, 1883.

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CEPHALALGIA.—HEADACHE.

It is not necessary to consider headaches occurring as one of many symptoms in various constitutional and inflammatory diseases, nor as an attendant upon organic cerebral diseases. Even when not thus associated, it is frequently only one of several symptoms, but the one which gives most distress, and requiring relief.

Whatever seriously lowers the tone of the nervous system or the general health may be an efficient cause. Neurasthenic patients usually suffer from some form of headache, most frequently of a dull or heavy kind, which is almost continuous.

Defective sanitary conditions, bad drainage, or poor ventilation, may cause the disturbance; frequently a morning headache may be traced to sleeping in an ill-ventilated room.

Anæmia and hyperæmia of the brain are said to cause headache. I doubt whether the variety of pain will help to distinguish between these two conditions: the diagnosis must be made from other symptoms or conditions. It is to be kept in mind that an anæmic patient may have a sudden flow of blood to the head, giving rise to headache of the congestive variety.

Alcohol, tobacco, various deleterious gases, as sulphureted hydrogen, carbonic oxide, or the gas used for lighting, may cause headache; chronic lead-poisoning is another cause.

Headache is sometimes the only symptom of tertiary syphilis; it is frequent in Bright's disease, and may be the first sign of trouble. There is also, apparently, a connection between headache and rheumatism and gout.

A very large class of cases are reflex in origin, depending upon disease or derangement in distant organs; the digestive and urino-genital organs are most frequently the seat of such disturbance.

Differences in the refractive power of the eyes may be the exciting cause of headache, and in every doubtful case an oculist should examine the eyes.

A careful examination of all the possible derangements is necessary to form a correct diagnosis; but with care there is usually not much trouble in arriving at a reasonable conclusion: in some cases, however, it will not be possible to discover the cause or nature of the affection.

Headache is rare in early childhood, and, when present, may be of serious import; it ought to lead to a watchful care lest it should be the forerunner of some serious disease. About the period of second dentition, and until puberty, headache may be more common,

and is sometimes severe and continuous, with remissions, but few intermissions. It is not then of a sharp, piercing character, but rather heavy and dull, increased by mental exertion and confinement.

In old age it is not common, and is of more importance than in middle life.

TREATMENT.—It may be necessary to change the patient's mode of life or residence. If there is any unfavorable influence about the house, it should be remedied, or, if that is not possible, the patient should change his residence.

Too severe mental application, whether in study or in business, must be moderated, and this is not always easy to accomplish. Sedentary habits must be broken in upon, exercise out of doors must be insisted upon, and late hours, whether for business or amusement, must give place to early retiring. High heels and tight lacing, and insufficient clothing, need to be looked after, even if the contest with what is thought fashionable seems well-nigh hopeless.

The diet should be regulated; the high and generous liver may expect to suffer until he can reduce his diet. An occasional saline cathartic may give temporary relief; or, if there is a gouty tendency, colchicum may be used; but more than that is needed, and while the patient persists in indulgence not much will be gained. In gouty cases, citrate of lithia, five grains or more three or four times a day, promises well.

Alcohol and tobacco should, of course, be given up; if there is any suspicion of lead-poisoning, iodide of potassium should be given to eliminate the poison.

Dyspepsia, or other affections which may act as causes, must be treated by appropriate means if possible.

During the attack, in cases of excessive blood-supply, counter-irritation to the back of the neck, cold to the neck, or an ice-bag to the lower part of the spine, ergotin in three-grain doses, bromide of potassium in

thirty to sixty grains; if the pain is severe, wet cups to the back of the neck, or leeches behind the ear—may be tried.

In nervous headaches, or those caused by exhaustion or overwork, citrate of caffein, two to five grains, is frequently sufficient for its removal; yet the caffein sometimes causes nausea. Aromatic spirit of ammonia and sweet spirit of niter are excellent remedies; a teaspoonful of each can be given, and repeated in one or two hours if necessary. Valerianate of ammonia, spirit of lavender, camphor, or asafœtida, may serve when other remedies fail. Hot water to the head is usually more grateful than cold.

Between the attacks, ergotin, in tendency to a congestive condition, with care as to habits. In other cases, the various tonics, cod-liver oil, and good feeding. Extract cannabis Indica, in third to half-grain doses three times a day, has proved very useful, not merely in migraine, as advised by Seguin, but in more common forms. The drug should be continued several weeks.

Iodide of potassium seems to be useful in other cases than where there is a rheumatic or syphilitic taint. Dr. Haley found that it relieved a dull, heavy headache over the brows accompanied by languor, chilliness, and feeling of discomfort. He gave it in two-grain doses, in half a wine-glass of water, to be sipped slowly.

Massage to the head will often relieve the pain in a few minutes; in chronic cases, the massage should be given for a long time, and may be general.

The galvanic current, passed from the forehead to the back of the neck, or transversely, may be tried, or the faradic current from forehead to neck. It is better to use the operator's hand as the electrode on the forehead, the battery-electrode being held in his other hand. The hand fits the shape of the forehead better than the common metallic electrodes, and the operator

can thus judge more correctly as to the strength of the current, which needs to be very mild.

MEGRIM.—SICK HEADACHE.—MIGRAINE.

Megrim, or sick headache, is a paroxysmal headache, usually limited to one side, frequently attended with nausea and vomiting; the intervals between the attacks are usually free of pain.

ÆTIOLOGY.—Heredity is even more evident in this than in many neuroses. Frequently it occurs in several successive generations in the same family.

Women are slightly more liable than men; Liveing says as 5 to 4, Eulenburg says as 5 to 1.

The first attack occurs most frequently before ten, or at puberty. It rarely begins after twenty-five.

Certain influences, as imprudence in diet, exhausting exertions, excitement, late hours, noise and confusion, will give rise to an attack: and these may occur more frequently at the catamenial period; but why it is so we do not know.

SYMPTOMS.—As in other "explosive" neuroses, the patient is usually free from pain, and in the enjoyment of good health, between the attacks. Sometimes the paroxysm is preceded by a warning; perhaps an unusual buoyancy of feeling and sense of exhilaration are noticed on the preceding day, and the patient knows he is about to be sick because he feels so well. Or, again, an indisposition begins the day before, and gives notice of the coming storm.

In almost every case, pain is the most prominent symptom. The pain is felt on waking; usually it is mild at first, but increases as the day advances until it reaches its greatest intensity. Sometimes the pain begins later in the day, and occasionally it is absent throughout the attack, the other symptoms alone appearing. The pain varies much in character and severity in different attacks, even in the same person. It is usually unilateral, the side affected varying, at one

time the left, and the next time, perhaps, the right side suffering. Sometimes it is bilateral, though then one side may suffer the more severely. At the beginning of the attack the pain is limited to one region, generally the forehead or the temple; as it becomes worse, it spreads over the whole side of the head. The pain continues throughout the paroxysm, six or eight to twenty-four hours, rarely longer, though a sense of heaviness or depression may remain for a while longer. There is general tenderness of the scalp over the region affected, rather than any special tender points.

Almost from the commencement of the pain there is a loss of desire for food, or absolute loathing of it. As the headache continues, this feeling changes to nausea, and at length vomiting sets in. One severe spell of vomiting may close the attack, and the pain cease, drowsiness or sleep following or not. Generally, however, more than one fit of retching and vomiting occurs, and the prostration is correspondingly severe, as the pallor, sweating and weakness show.

The drowsiness or heavy sleep which sometimes follows the vomiting may be in part the result of the exhaustion caused by the pain and the vomiting. It is not like the quiet, natural sleep which sometimes closes the attack; it rather resembles the stupor following an epileptic fit.

Other symptoms are less common than the pain and nausea. Visual disturbances are next in frequency, and, when present, generally appear before the pain. They consist in partial or total loss of sight, and in various luminous appearances, of greater or less brilliancy, and sometimes colored. The loss of vision is sometimes central and sometimes lateral; there may be true lateral hemianopsia. The luminous phenomena may consist simply in the perception of a bright light, without definite form, or there may be zigzag lines of light, sometimes colored, resembling fortifications. A small point of light is first seen, which gradually expands,

increasing in size, assuming the above zigzag form, until it extends beyond the field of vision and disappears. Meanwhile, a new spot appears, and goes through the same changes; the lines of light have tremulous motion.

Disturbances of common sensation, anæsthesia, and sometimes of the special senses, on the same side with the headache, are less frequent. When the numbness is on the right side, there may be with it one of the forms of aphasia.

In severe cases it is impossible for the patient to apply himself mentally; but sometimes more than this mental disturbance is noticed, though not to any serious extent, even during the seizures.

PATHOGENESIS.—It is often assumed by the patient that the attacks of migraine are due to gastric disturbance; as the nausea and vomiting are so frequent, this is not strange, and too often the physician falls in with this view. While imprudence in diet may sometimes be the exciting cause of an attack, it is only incidentally so; other causes are equally as effective. The phenomena are all referable to cerebral influence; the nausea is from the encephalon, not from the stomach.

Any one interested in the different theories will find them fully discussed by Liveing.

There seem to be two conditions of the cerebral circulation during the attacks; in some the vessels seem to be in a state of spasm, contracted; in others dilated. Liveing refers the attacks to the explosive tendency to be found in the nervous system even in health.

There is usually no anatomical change possible, as the attacks are so fugitive. Occasionally certain of the sensory disturbances, as numbness or visual change, are more permanent, so that a slight structural change in the brain might be possible. A change in the circulation, and, in the above rare cases, in the structure of the posterior part of the inner capsule and adjoining portion of the optic thalamus, would explain the symp-

toms, except, perhaps, the headache and nausea. Provisionally, we may imagine this to be the seat of the change, though our actual knowledge in this regard is very slight.

There is a resemblance between the attacks of migraine and those of epilepsy, which it is not necessary to specify minutely. A few cases have been observed in which migraine in early life was later replaced by epilepsy.

PROGNOSIS.—The prospect is very slight of complete immunity from the attacks during early life; with advancing years the frequency of the paroxysms diminishes, and finally the patient is free. Yet much benefit may be derived from treatment in diminishing the severity and frequency of the attacks.

TREATMENT.—Such hygienic measures should be adopted as will most effectually remove the exciting causes; over-exertion of the brain, neglect of proper exercise and out-door life, late hours, excitement—such influences can be avoided by sufficient self-denial on the part of the patient except in the cases where the struggle for the necessaries of life demand the sacrifice. Much can be done, also, by means already alluded to, to increase the strength and vigor of the nervous system.

During the attack the patient will instinctively take the precautions as to rest and posture which are most favorable. The friends may, however, be too fussy. Quiet, i. e., freedom from noise, motion, jarring, and from conversation; exclusion of light; coolness in the atmosphere of the room, not chilliness; abstinence from food—these conditions may be obtained by the aid of friends, or officious friends may render it impossible for the patient to have them.

Drugs taken early in the attack may diminish its severity. Sometimes a large dose of quinine, ten to twenty grains, is of benefit; caffein, or citrate of caffein, in two- to five-grain doses, is more effectual; prep-

arations of guarana have an effect similar to caffein. In cases of paralysis of the vaso-motor constrictor nerves, ergot is of value; where there is spasm of those nerves, the inhalation of nitrite of amyl proves of value, but it may be necessary to repeat it several times at short intervals; one to three drops may be inhaled; when a patient's peculiarities and susceptibility to the drug are known, larger doses may be used; belladonna or atropia may be of use in the same class of cases; nitro-glycerine, or glonoin, as it is also called, has been recommended, one drop of the one-per-cent solution being used in water, but its value is doubtful. Chloride of ammonium, twenty to forty grains, may diminish the severity of the pain. Copious draughts of hot water have been of value with some patients. After the nausea has become marked, internal remedies are likely to increase it, and add to the distress by causing vomiting; though, in rare cases, the emesis relieves the pain and shortens the attack.

Several remedies have a value in diminishing the frequency of the attacks. Extract of cannabis Indica in one-third- to one-half-grain doses three times a day is very valuable, but it must be continued several weeks. Valerianate of zinc, three grains three times a day, is also useful; and in larger doses, five or six grains every three hours, it may shorten the attack.

Liveing found iodide of potassium, five grains three times a day, of advantage, and he also recommends bromide of potassium. It should be given in ten- to twenty-grain doses three times a day for several months.

The galvanic current may be used with advantage. In cases where the vessels are contracted, the negative pole should be held in the hand, or on the back of the neck, while the positive pole is placed over the cervical sympathetic without interruptions. In cases with paralysis of the constrictor vaso-motor nerves, Erb advises the cathode over the sympathetic, and that the

current should be repeatedly opened and closed, avoiding, however, too strong irritation.

The faradic current, used as advised for simple headache, sometimes gives relief.

Many times these applications can not be made during the attack, as the pain is increased by the simple manipulations necessary, and not relieved by the electricity.

Between the attacks, general faradization or galvanization may be of great service, as in other cases of diminished nervous power, in restoring the system to a normal condition.

Massage would rarely be of value during the attack, but in its incipient stage, and between the attacks, may be of great benefit.

GRAVES'S DISEASE (Exophthalmic Goître).

Eulenburg und Guttmann, Die Basedow'sche Krankheit. *Arch. f. Psych.*, i, 1868, p. 430.—Wilks, S., Exophthalmic Goître. *Guy's Hosp. Rep.*, 1870, p. 7.—Russell, J., Clinical Illustrations of Graves's Disease. *Med. Times and Gaz.*, Sept. 2, 1876 *et seq.*—Chvostek, Die Therapie der Basedow'sche Krankheit. *Zeitschr. f. Therapie*, No. 8, 1883.

Exophthalmic goître is an affection attended with three prominent symptoms—palpitation, goître, and exophthalmos.

Symptoms.—The disease usually begins by a nervous irritability and change of character, feeling of fullness in the head and eyes and neck, and palpitation. In most cases the palpitation is spoken of as the first symptom, perhaps because it first attracts attention. The heart's action rises to 100 or 150; but there are no signs of organic disease of the heart.

The projection of the eye is usually attended with a diminution in the motion of the upper lid, so that, if the eyeball is rolled upward, the lid does not move in harmony therewith.

Sight is not affected, and accommodation is not dis-

turbed. The ophthalmoscope generally shows nothing abnormal, though optic neuritis has been seen.

The thyroid gland slowly increases in size until it becomes quite prominent; even the middle lobe may be enlarged. In consequence of this enlargement, the voice may be somewhat changed in character, and respiration may be disturbed.

Various general symptoms may be associated with the above. The appetite suffers; diarrhœa sometimes sets in; there may be extreme emaciation; anæmia is not uncommon.

Among women, the catamenia may cease, or there may be dysmenorrhœa. There is sometimes unilateral sweating.

Sometimes one of the three prominent symptoms may be absent.

As a rule, the symptoms are very slightly marked at first, but gradually increase in severity, the disease being chronic in its course. Occasionally an acute case appears, in which all the symptoms are rapidly developed.

PATHOLOGY.—The pathology of this disease is by no means easy to explain. Panas is inclined to think that the disease depends upon a disturbance of the medullary oblongata, an irritation which would excite the sympathetic fibers passing to the heart, and the vaso-dilator nerves would explain the symptoms. Or, on the contrary, a paralysis of the inhibitory nerves, and of the vaso-constrictures. Panas is inclined to the belief that the latter is the correct explanation.

Anatomical changes have been found several times in the cervical sympathetic, especially in the lower ganglion.

PROGNOSIS.—Prognosis is not very favorable, though several cases of recovery have been reported.

TREATMENT.—Digitalis has but little influence in moderating the rapidity of the heart's action. Belladonna has sometimes been of use. Quinine and iron

have also proved serviceable. The best results have been obtained by Chvostek from the use of electricity. He used the ascending galvanic current to the cervical sympathetic, and on each side, stabile, one minute; to the spinal cord the positive pole on the fifth dorsal, the negative on the cervical vertebræ; he also passed a current transversely through the head from one mastoid process to the other; or in some cases applied it to the temples. The application was about one minute in each place. He used a very weak current, which caused no sense of heat, and applied it daily.

Meyer and Leube have both obtained good results from galvanism.

ANGINA PECTORIS.

See, G., De l'angine de poitrine. *France méd.*, 1876, p. 197 *et seq.*—Balfour, G. W., Upon Paroxysmal Angina Pectoris. *Edinburgh Med. Jour.*, March, 1881, p. 769.—Huchard, H. *Le concours méd.*, No. 6, 1884.

Angina pectoris is a disease characterized by painful paroxysms, the pain being situated in the neighborhood of the heart, and radiating thence to the left side of the chest and left arm. The attacks are accompanied with great anxiety and a sensation of impending dissolution.

Ætiology.—The disease attacks males by preference, and occurs most frequently after the age of forty. It is uncertain whether heredity plays any part in the ætiology of this disease. Gout, rheumatism, and alcoholism are supposed to be important as causes. Excessive tobacco-smoking is also spoken of as predisposing to the disease. Many cases of angina pectoris are found in persons who have organic diseases of the heart, and these are naturally looked upon as giving rise to the pain. Especially is this true in regard to changes in the coronary arteries, or such changes at the commencement of the aorta as are likely to interfere with

the circulation of the blood through the substance of the heart.

As directly exciting to the attacks may be mentioned exposure to cold, unusual mental emotions and bodily exertion, especially walking against a strong wind, or walking rapidly up hill, or ascending a flight of steps rapidly. Yet many cases occur in which there has been no special exciting cause, as is particularly true of those attacks which occur during sleep.

PATHOLOGICAL ANATOMY.—The anatomical changes found are such as belong to the organic diseases of the heart, which may be supposed to exert an influence as predisposing causes. Otherwise than these changes, which need no special description, there is no anatomical lesion discoverable.

SYMPTOMS.—The prominent symptom of this disease is pain, which is situated usually along the left border of the sternum, and more especially near the apex of the heart. Thence the pain may radiate over the whole chest, may descend along the left arm, rarely going below the elbow. The pain is not so sharp and acute as some other species of neuralgia, but it is attended with such anxiety and distress, such a sense of danger to life, that it is much less bearable than almost any other variety of pain.

During the attack the pulse is often increased in rapidity, but is rarely irregular unless there is organic cardiac disease. The respiration is sometimes unaffected, though very often the patient instinctively holds his breath, and remains immovable, supporting himself by his arms, as if afraid even to breathe. Each attack of severe pain is usually of but short duration; but they may succeed one another rapidly, and the duration of the whole attack is very variable, sometimes extending over days, though when so long there are generally periods of remission almost amounting to intermission.

When the attack has finally ceased, the patient is usually free from pain until the recurrence of the next,

which may not be for many months; but, as the attacks recur, their frequency becomes greater, until the intervals between are very short.

PROGNOSIS.—When the disease depends upon an organic change of the heart, death usually follows after a longer or shorter interval. Where there is no organic change of the heart, the patient may recover, and live long without a recurrence of the disease.

The nature of the disease is but imperfectly known. It is generally considered as belonging to the neuralgias. Some cases are referable to gout or rheumatism; some are probably due to fatty degeneration of the walls of the heart. Some again, perhaps, depend upon an imperfect supply of blood to the heart, in consequence of disease of its nutrient arteries. It has also been referred to a neuritis of the cardiac nerves. As has been remarked: "It is very difficult to choose an opinion out of the midst of this labyrinth of explanations, which contradict each other and destroy each other."

DIAGNOSIS.—The chief danger of mistake lies rather in considering a simple neuralgia, affecting the intercostal nerves, as angina pectoris.

The character of the pain is different, the anxiety and distress attending it are much less in intercostal neuralgia, or are entirely wanting.

Embolism of the pulmonary artery is attended with much greater dyspnœa, and the circumstances associated with it will assist to a diagnosis.

It is of primary importance to learn whether the pain is associated with organic disease of the heart. To do this, it would be necessary to examine the heart between paroxysms. And even a careful physical examination may not settle the question without doubt.

TREATMENT.—Eulenburg says: "The remedies are many, the cures few." During the paroxysm it is most important to relieve the pain and distress.

Nitrite of amyl will sometimes cut the attack short.

Five or six drops on a handkerchief, inhaled by the patient, repeated if necessary, may suffice to relieve the paroxysm.

The subcutaneous injection of morphia, with a small amount of atropia, will often give relief. With other patients, the most speedy relief is experienced from the application of ice immediately over the heart. Nitro-glycerine (glonoin), in the dose of one or two drops of the one-per-cent solution, has been recommended, and has, in some cases, proved very efficacious, not only in relieving the pain of the attack, but in warding off a recurrence.

Aconitia, by preference Duquesnil's, in dose of from $\frac{1}{150}$ to $\frac{1}{200}$ of a grain, repeated, if necessary, every hour or two hours, until there is tingling of the lips, or tongue, or fingers, will often give relief, but is less rapid in its action than the remedies previously mentioned. The benefit derived from this drug is, however, of longer duration. Tincture of aconite-root may be used instead.

Between the attacks means should be taken to diminish the danger of a recurrence. If there is any gout or rheumatic tendency, appropriate remedies should be used. In other cases the general health should be maintained, the patient cautioned against over-exertion of any kind, and, as many of these patients are run down in general health and overtaxed, a large proportion of rest is absolutely necessary. Smoking should be given up.

Among the drugs which may be used with the prospect of greatest benefit is arsenic. Besides this, preparations of iron and zinc may be used. Nitrate of silver has been recommended. Digitalis, combined with the arsenic, is also mentioned as valuable.

SYMMETRICAL GANGRENE.

RAYNAUD, M., Nouvelles recherches sur la nature et le traitement de l'asphyxie locale des extrémités. *Arch. gén. de méd.*, 1874, Jan., p. 1 ; Feb., p. 189.—WARREN, J. C., Symmetrical Gangrene of the Extremities. *Boston Med. and Surg. Jour.*, Jan. 16, 1879, p. 76.—WEISS, M., Ueber sogenannte symmetrische Gangrän. *Zeitschr. f. Heilk.*, iii, 1882, p. 233.

The causes of this peculiar affection are not known. The disease consists in a disturbance of the circulation, especially in the extremities, probably an affection of the vaso-motor nerves, by which the supply of blood is cut off, and hence the nutrition suffers ; and, when carried to an extreme, gangrene sets in, and the affected parts slough off. Fingers and toes are the most frequently affected ; but limited spots on the body, and even the face, may suffer. The disease is almost always symmetrical, and is paroxysmal.

In many patients there is a prodromic period of unrest, with change of character. The patient becomes peevish, fretful, surly, withdraws within himself, and avoids contact with those whom he formerly sought, even his own children. He sighs, and frequently sheds tears. Sleep is restless, broken by dreams. Appetite poor, digestion difficult, and the slightest excess is followed by severe gastric crises, similar to those seen in ataxia. Hearing, sight, and taste may be diminished.

A disagreeable sensation is felt in the limbs ; the circulation is sluggish ; the surface becomes cyanotic, perhaps almost black ; generally, severe pain, of a neuralgic character, sets in, which is without intermission, almost without remission. The affected parts are anæsthetic, and this may cause the gait to assume an ataxic character.

After reaching this stage, the symptoms may subside and the normal condition of the parts return. Otherwise, the disturbance of nutrition increases, and there is gangrene affecting the fingers and toes, or su-

perficial spots of the skin, and after four or five days the dried epidermis falls off, leaving a superficial ulcer, which heals slowly. If a whole phalanx of finger or toe is affected, this drops off, and the stump cicatrizes slowly.

There is no fever attending the above phenomena. The heart is unaffected; the pulse is not disturbed, even in the arteries near the cyanotic parts. The temperature is lowered in the affected limbs. Occasionally there is no pain.

Raynaud found the circulation of the fundus of the eyes affected, arteries contracted, and venous pulsation; in one case he found these changes between the attacks; in another case at the same time with the attacks.

The diagnosis is not difficult when the whole series of symptoms is before one; but at the beginning there might be some doubt as to the nature of the affection, and it would be excusable to suspect locomotor ataxia, or disease of the spinal membrane.

TREATMENT.—Warmth and rest are indicated; some of the symptoms might be relieved by gentle massage.

Raynaud obtained excellent results from the use of the galvanic current applied over the spinal column. He used from twenty-five to thirty cells, the positive pole over the fifth cervical vertebra, the negative pole over the sacrum. After a short time he slid the negative pole up to the eighth dorsal vertebra. The application was continued ten to fifteen minutes daily. The circulation became more rapid; abundant sweat appeared; the hands became moist; in some cases headache followed the application, and then it was necessary to reduce the number of cells. He also applied the positive pole over the nerves in the upper part of the limb, and the negative pole over the affected surface. He was able to use in this way from thirty to sixty cells. As improvement appeared, sensation became more acute, and it was necessary to reduce the number of cells.

UNILATERAL FACIAL ATROPHY.

GUTTMANN, P., Ueber einseitige Gesichtsatrophie. *Arch. f. Psych.*, i, 1868, p. 173.—BANNISTER, H. M., Progressive Facial Hemiatrophy. *Jour. of Nervous and Mental Diseases*, Oct., 1876, p. 539.—HAMMOND, WM. A. *Ibid.*, April, 1880, p. 250.—FLASHAR, Ein Fall von bilateraler neurotischer Gesichtsatrophie. *Berl. kl. Woch.*, Aug. 2, 1880, p. 441.—WETTE, H. *Cbl. f. d. m. Wissen.*, July 8, 1882, p. 491.—MENDEL. *Berlin. kl. Wochenschr.*, Sept. 17, 1883, p. 588.—JESSOP and BROWNE. *St. Barthol. Hosp. Rep.*, xviii, 1882.—WOLFF, Ueber doppelseitige fortschreit. Gesichtsatrophie. *Virch. Arch.*, 94, 1883, p. 393.

The cause of unilateral atrophy of the face is not certain; more cases have been noticed in women than in men; frequently injuries have preceded the atrophy.

SYMPTOMS.—Preceding the change in the tissues of the face there may be a prodromic period, during which there is pain in the face and head, with perhaps hyperæsthesia. Bannister noticed absence of perspiration on the affected side of the face, without pain.

Before the atrophy, there is usually a whitish discoloration of the skin at the point where the change is about to take place. Two or three of these spots may appear at a moderate distance from one another, and run together. The hair becomes white, and may fall out.

The atrophy affects the skin and subcutaneous tissues; the bones rarely undergo atrophy, though their growth may be retarded if the patient has not reached adult years. The muscles of the face are not subject to any fatty degeneration. Hammond found the muscular fibers diminished in size. Owing to the loss of fat tissue and change in the skin, the cheek is hollowed in; the skin, lacking in elasticity, seems to be closely adherent to the bone. Sometimes the tongue, the hard and soft palate, are affected; the eye seems to be sunken deeper into its socket, and the lids are partially closed.

There is no muscular paralysis; the electrical reactions are normal; the circulation is rarely disturbed;

there is no change of temperature on the two sides of the face; tactile sensation is not diminished.

The disease does not lead to a fatal result, and no autopsies have been made. Several theories have been advanced to explain the singular phenomena. It has been thought to be a disease of the cervical portion of the sympathetic, or of the nuclei of the facial or other cranial nerve; of the spheno-palatine ganglion, or a primitive atrophy of the adipose tissue.

No treatment has been of permanent benefit. Electricity has been thought to give a slight relief. Both the galvanic and faradic current have been applied locally to the face, but no permanent improvement followed.

CHAPTER XXVII.

VERTIGO.

RUSSELL, J., Illustrations of Stomachic Vertigo and Allied Affections. *Med. Times and Gaz.*, July 3, 1880.—JACKSON, J. H., Lecture on Auditory Vertigo. *Lancet*, Oct. 2, 1880, p. 525.—FÉRÉ, CH., et DEMARS, A., Note sur la maladie de Ménière et en particulier sur son traitement par la méthode de M. Charcot. *Revue de méd.*, No. 10, 1881.—LEVEN, Du vertige. *Gaz. des hôp.*, May 23, 1882, p. 468.—WOAKES, E., Remarks on Vertigo. *Brit. Med. Jour.*, April 28, 1883, p. 801.

Frank defines vertigo as "an illusional turning, painful and sudden, which seems to affect the person himself and external objects, whether they are in repose or moving in their ordinary manner."

Vertigo is really a symptom, not a disease. It may be reflex or sympathetic, or the cause may escape our observation, and then it is spoken of as idiopathic.

Axenfeld has said that vertigo is caused by a change in the intra-cranial circulation, either a lack of sufficient arterial blood, or hyperæmia, causing the symptom. In both these conditions the molecular interchange in nerve-cells is incomplete ; their nutrition suffers ; their functions are exalted, exhausted, or perverted.

CAUSES.—Vertigo is sometimes caused by dyspepsia. It is one of the symptoms of exhaustion, of cerebral anæmia, of intestinal or uterine disturbance. Various poisons may produce vertigo, as tobacco, alcohol, opium, oxide of carbon, lead, prussic acid, or uræmic poisoning. It is very common in certain cerebral diseases, especially those affecting the cerebellum. Heart

disease is sometimes accompanied by vertigo. It may be premonitory of various acute diseases, as the exanthemata. The *petit mal* of epilepsy is often simply vertigo attended with impaired consciousness.

A disturbance of accommodation in the eyes, especially if the eyes differ one from the other, may give rise to this disagreeable symptom. A slight weakness of some of the motor muscles of the eye, producing slight strabismus, may have the same effect. Of these, the causes are comparatively simple, and require little more than a mention. There is, however, a series of symptoms, due to disturbances of the ear, which deserve more extended mention.

Disease of the semicircular canals, or even a slight increase of pressure upon the fluid contained in these canals, is attended with vertigo. Even an accumulation of wax in the ear, or the closure of the Eustachian tube, may be sufficient to give rise to the symptoms.

Vertigo, when caused by affection of the ear, was carefully described by Ménière, and hence has been called, from him, Ménière's disease.

Besides vertigo, there is usually tinnitus, also deafness, which in severe cases is complete.

The attack is often sudden, the patient being obliged to stop, immediately seize hold of some object to support himself, or perhaps he falls, as if suffering from an attack of epilepsy. There is, however, no loss of consciousness. His face is pale, his skin is cold, he is bathed in perspiration. There is nausea, vomiting, and headache. Many times the attack is less severe—the patient simply staggers instead of falling, and suffers severely from the accompanying symptoms. As a rule, the attack is not of long duration. The patient regains his steadiness and his usual health; but, so long as the affection of the ear continues, there is danger of a renewal of the vertigo.

The *treatment* of auditory vertigo is, of course, first to remove any affection of the ear, any cause of press-

ure upon the semicircular canals. Charcot found great benefit from the use of seven to fifteen grains of quinine daily for nearly three months. If necessary, the quinine, after having been omitted, can be repeated when the symptoms recur.

Sometimes electricity may be of benefit, though very rarely.

It is important to make a correct diagnosis as to the cause of vertigo. Auditory vertigo is very likely to be mistaken for epilepsy, or for vertigo associated with dyspepsia. Unless a correct diagnosis of the cause is made, the treatment is likely to be futile.

The treatment of other forms of vertigo must be directed, according to the cause, to the primary disease.

CHAPTER XXVIII.

CHOREA.

STURGES, O., Some Statistics of Fatal Chorea. *Lancet*, July 17, 1880, p. 85.—MITCHELL, S. WEIR, Lectures on Diseases of the Nervous System. Lectures VII, VIII. Philadelphia, 1881.—STRANGE, W., Notes of 100 Cases of Chorea. *Brit. Med. Jour.*, July 16, 1881.—STURGES, O., The Heart Symptoms of Chorea. *Brain*, July, 1881, p. 164.—CHAPIN, H. D., Points of Interest in Chorea. *Med. Record*, Dec. 15, 1883, p. 648.—SANTINI, G., Sulla patogenesi della corea. *Rivista Sperimentale*, ix, 1883, p. 449.—STURGES, O., Chorea with Rheumatism. *Lancet*, Aug. 31, 1878, Nov. 29, 1879, Sept. 20, 1884.—HOUGHTON, J. H. *Brit. Med. Jour.*, Dec. 9, 1882.—RICKARDS, E. *Ibid.*, Nov. 11, 1882, p. 932.—FRASER, T. R. *Ibid.*, Dec. 9, 1882, p. 1132.

Chorea (Saint Vitus's dance) may be defined as a neurosis affecting the voluntary muscles, generally preceded and attended with slight mental disturbances, the motor phenomena consisting in irregularity of voluntary motions, or, when severe, the spontaneous development of irregular motions apart from the intervention of the will.

ÆTIOLOGY.—Certain constitutional conditions undoubtedly predispose to chorea. Heredity seems in some cases to be one of the predisposing causes; the instability of the nervous system at certain periods of life, as during second dentition, and at puberty, are also to be taken into account, many more cases occurring from six to eleven years of age than at any other equal period. The disease is extremely rare after twenty-five years of age. The debility following scarlatina, diphtheria, typhoid fever, etc., frequently seems to predispose to the disease. Rheumatism appears to be-

long rather to the exciting causes, considering the frequency with which it is followed by chorea. The relation which the two diseases bear one to the other is still an undecided question; they occur together too frequently to justify us in considering the relation purely accidental. Chorea may appear before the child recovers from the rheumatism.

The restraint and discipline of school life, especially public-school life, with its ambitions, oftentimes has an injurious influence.

Among the more immediate causes of chorea may be mentioned sorrow, care, anxiety, fright, and irritation. Occasionally chorea occurs during pregnancy, especially among primiparæ, and most frequently during the twenty-first and twenty-third years of age.

Symptoms.—The earliest symptom is a change of disposition. The child becomes restless, irritable, is thought to be getting nervous, or to have the fidgets. It loses its temper more easily, gives impertinent and saucy replies to its parents; in fact, the whole nature of the child seems to have undergone a decided change for the worse. Much too frequently the parents and teachers consider the child disobedient and naughty, whereas it is entirely irresponsible for its conduct, and, instead of taking the proper measures to stop the disease at its very beginning by curing a physical malady, they make it worse by employing reproof, punishment, and harshness to correct a moral delinquency that does not exist. This change of disposition may continue throughout the disease, and even extend beyond the time when motor disturbances have ceased. During the severest attacks there may be lack of mental power, and inability or disinclination to apply the mind vigorously; the expression of the patient may be almost idiotic.

After the above mental symptoms have existed a variable length of time, motor disturbance makes its appearance, as a rule beginning on one side and ex-

tending to the opposite side. Some authors say that the left side is the more frequently affected. Occasionally the motor disturbance is confined to one side during the entire disease. At first the irregular motion is slight, scarcely perceptible to an ordinary observer; this irregular action of the muscles becomes more marked and constant. The face and upper extremities are in constant motion; the patient is continuously making grimaces, which at first may be thought voluntary, and the child may be unjustly punished for "making faces." The fingers are flexed and extended, one or several at a time; the child picks at its clothing, pulling and perhaps tearing it. When the lower extremities are affected, the toes and legs are in constant motion, like the hands. Voluntary motion is interfered with; the child is unable to write or sew; often can not feed itself. Walking may also be difficult, or impossible. In extreme cases, not only are the limbs and face affected, but the body may also be turned and twisted and bent in various directions by the involuntary action of the muscles of the trunk. The patient is unable to be up, is tossed about in bed; the constant and violent motion causes excoriation of the skin; he has a wan and haggard, perhaps a half-idiotic look, and is a pitiable sight. The respiration in severe cases is sometimes affected, acquiring a jerky character; speech may partake of the same jerky character, and it may even be almost impossible for the patient to speak.

Except in very severe cases, sleep is not disturbed, and almost invariably the involuntary motions cease during sleep. In cases of moderate severity the patients do not complain of being tired, and there is no appearance of exhaustion. Of course, this is not true of the severest cases.

Sensation is but little if at all affected; once in a while there may be pain, and sometimes tenderness, on pressure over the nerves.

M. Rosenthal found that the reaction of the muscles to both the faradic and galvanic current was frequently increased.

It is very common to find a cardiac murmur in cases of chorea, even where there has been no rheumatism preceding. It is not very rare to find an irregular intermission of the pulse. As the patient recovers, the cardiac symptoms disappear, unless caused by organic lesion.

A so-called post-hemiplegic chorea is one of the sequelæ of cerebral disease. The motions may very closely resemble those of chorea. This has been described in connection with cerebral diseases.

When the disease is caused by fright, and occasionally under other circumstances, the motor symptoms show great intensity at the commencement, the most violent irregular action appearing in the course of a few hours. These cases, however, are exceptional.

DURATION.—The duration of chorea differs greatly in different cases. Some patients recover in three or four weeks; in others the disease is extended over several years; the average duration is said to be two or three months. It is not uncommon for a child to suffer from two or even three attacks. Between these attacks the child is considered well, and is thought to have been cured; but many times a careful observation will show that the irritability of the temper and changed disposition persist; also, if the child's hand is quietly held, a very slight spasmodic action of the fingers can be felt—too slight, perhaps, to be seen.

Many cases, therefore, spoken of as recurrence of chorea, are in reality simply cases of remission of the severer symptoms. Dr. Mitchell mentions the greater frequency of chorea in the spring, and a tendency to recur the succeeding spring.

DIAGNOSIS.—The milder cases of chorea can hardly be mistaken, especially if their history is learned; the description of the disease already given is sufficient for

diagnosis. There is a form of choreic disturbance which occurs after apoplexy, affecting the paralyzed side, which might be mistaken for genuine chorea. Charcot has well described this, and named it post-hemiplegic chorea. A history of the case showing the previous attack of paralysis, the fact that the affected limb still suffers from impaired power, and the difference between the involuntary motions in these cases and the irregular action seen in genuine chorea, together with frequent presence of contraction, would assist in forming a diagnosis between the two conditions.

In a few cases of cerebro-spinal sclerosis the irregular involuntary motions become so general, and reach such a grade of severity toward the close of the disease, that it is possible to mistake the affection and consider that the patient is suffering from chorea. In these cases the history of the disease and the general character of the motions, the fact that sensation is often affected in sclerosis, that contraction of the fingers and hand are more frequent, will aid in forming a correct diagnosis. These cases are, however, often very difficult to distinguish from chorea.

Pathological Anatomy.—The nature and seat of the lesion causing chorea are still undecided questions. Mild cases recover, leaving no disability; only severer cases result in death: and therefore it is not without reason that many authors object to ascribing the milder cases to the same lesion as is found in the severer cases. In many autopsies the smaller blood-vessels of the brain have been found plugged. In some cases minute vegetations have been found on the valves of the heart, accounting for the embolisms in the brain; but these changes are not constant. The optic thalamus and corpus striatum are the parts chiefly or exclusively affected.

It is specially interesting to recall the fact that in post-hemiplegic chorea, and in so-called athetosis, the lesions preceding these irregular motions are situated in the same portions of the brain in which these dis-

eased arteries are found in chorea. Whether this portion of the brain is affected in sclerosis with exaggerated tremor has not, to my knowledge, been a subject of investigation. It is also an interesting fact that bundles of nerve-fibers, passing through this region from the cortex, may be irritated so as to produce movements in the limbs similar to those produced by irritation of the motor centers in the cortex.

There is, therefore, a presumption in favor of a similar causation of the irregular movements in all these affections. Whether the mild cases of chorea, ending in complete recovery, can ever be referred to an organic lesion of the brain, is extremely doubtful. A simple functional disturbance is sufficient to account for all the symptoms. Considering the mental phenomena, and the frequent hemiplegic character of the disease, affecting the face as well as the limbs, it must be looked upon as primarily of cerebral origin.

PROGNOSIS.—There is very little more to be said in regard to prognosis; the milder cases always recover after a variable length of time, perhaps after two or three relapses. The older the child at the beginning of the disease, the more severe will it probably be, and the longer its duration. Death seldom occurs. In every case where I have seen such a result it has seemed to me that there was organic cerebral disease which caused the choreic symptoms and the fatal termination.

TREATMENT.—The most active and meddlesome treatment has been advocated by some, and entire abstinence from medicine by others. In reality, mild cases probably do well without drugs.

Hygienic and moral treatment is necessary in every case. The child should be noticed as little as possible; its attention not called to its infirmity, unless it can be praised for improvement. Nothing more should be demanded of the child than can be possibly helped; the desires and wants of the child should be anticipated, and gratified as far as may be without over-indulgence.

Of course, the child should be taken out of school, and, if necessary, removed from the society of playmates and the care of servants, who may irritate and annoy it by reference to its irregular movements. Common sense and knowledge of child nature will serve better than any written directions to guide this part of the treatment. Except in the severer cases, confinement to the bed and house is unnecessary.

Arsenic has proved a most successful medicine. This should be given at first in moderate doses, then the dose rapidly increased to the limit of toleration. Thus, beginning with three or four drops of Fowler's solution three times a day, the dose may be increased, by one drop every second or third day, until the child takes twelve or fifteen drops, or even more, at a dose. This remedy is to be thus continued, increasing the dose until either nausea follows or œdema below the eyes shows the approach of toxical effects. If nausea requires the drug to be discontinued, it should be resumed in the same dose as when omitted so soon as the nausea ceases. Unless given in large doses, no benefit can be expected. Many times it is of benefit to use iron also, and other tonics. Sulphate of zinc, in doses of three to five grains three times a day, has also been highly recommended. I have generally, however, found the arsenic sufficient.

In severe cases, where the spasmodic action interferes with sleep, it may be necessary to give remedies to procure the needed rest. Of these, chloral is much the best. Perhaps paraldehyde would be as serviceable. It is not desirable to give opium, or any of its preparations, if it can be avoided. Hyoscyamus, conium, and belladonna may be used as occasion requires. Calabar-bean has been used by Bouchut; he employed ·003 to ·005 grm. three or four times a day subcutaneously, with benefit to the patient. In many cases relief follows the application of ether spray to the back; or ice along the spine may be used instead of the ether.

CHAPTER XXIX.

PARALYSIS AGITANS.

LUYS J., Contribution à l'étude anatomo-pathologique de la paralysie agitante. *L'encéphale*, 1882.—BERGER, Ueber Paralysis Agitans. *Schmidt's Jahrb.*, 195, 1882, p. 246.—BUZZARD, A Clinical Lecture on Shaking Palsy. *Brain*, Jan., 1882, p. 473.—ERLENMEYER, A., Beitrag zur symptomatischen Behandlung der Paralysis Agitans. *Cbl. f. Nervenheilkunde, Psych.*, etc., May 1, 1883, p. 193.

PARALYSIS AGITANS.—SHAKING PALSY.—PARKINSON'S DISEASE.

Paralysis agitans is most common in old age, the name very well expressing its character—a tremor, more or less severe, attended with a weakness of the muscles, and a certain degree of stiffness. Violent emotions have sometimes been thought to be the cause; but it is very doubtful whether such is the case. Otherwise we know nothing as to its ætiology.

SYMPTOMS.—The tremor is peculiar and characteristic, showing itself when the limbs are at rest, diminishing or ceasing during voluntary motion, in this respect contrasting strongly with the tremor of sclerosis. The temperature is not increased, although there is such constant muscular action. Occasionally the trembling entirely ceases just before death.

In the early stages of the disease the tremor is very slight, and is usually confined to the hands and fingers, often to the thumb and index-finger, which are rapidly adducted and abducted. At first this is a slight annoyance to the patient, and does not interfere seriously with his comfort or his ordinary pursuits. As the disease advances, however, the tremor affects

other muscles, becomes more marked, and greatly annoys the patient. It does not cease entirely during voluntary motion of the parts, and so may interfere more or less seriously with the use of the hands.

Opinions differ somewhat as to whether the head is affected; it certainly is in some cases.

After a while, to the tremor is added a weakness of the muscles—partial paralysis. There is also stiffness of the joints, the hands take a peculiar position, the thumb and index-finger being approximated, as if holding a pen, and occasionally there is a slight claw-shape position of the fingers.

The electrical reaction of the muscles is normal.

In walking, the patient's head and body are bent forward, so that it seems almost as if he would fall upon his face. Instead of walking naturally, the patient trots forward with short steps, as if the position of the body produced a sensation of falling, and he found it necessary to run in order not to pitch on his face. Sometimes there is compulsory motion backward instead of forward. The tendon reflexes have been noticed to be exaggerated in both the upper and lower extremities. Sensation is variously modified in some cases, and, when the disease is advanced, the mental powers suffer. There is sleeplessness and loss of memory, sometimes melancholia.

PATHOLOGICAL ANATOMY.—No satisfactory explanation of this disease has been proposed. Many careful autopsies show no change at all of the nervous system, and the pathological changes, if any, remain to be discovered, though there is a tendency among authors to refer the disease to some disturbance of the medulla oblongata.

DIAGNOSIS.—The diagnosis must be made from sclerosis of the nerve-centers, with which this disease was formerly confounded. The difference in the character of the tremor, the positions of the hands in an advanced case, are sufficient to distinguish the two. The

progress of the disease is also different. The cerebral symptoms are similar in only a few cases. They occur earlier in sclerosis than in paralysis agitans.

The tremor of metallic poisoning, or of acholismus, may be diagnosticated by the history of the patient and by the progress of the disease.

It is scarcely possible that post-hemiplegic chorea should be mistaken for paralysis agitans if the physician is sufficiently careful.

PROGNOSIS.—Recovery is not to be expected. Intermissions may occur, but the course of the disease is generally forward.

TREATMENT.—Very little can be said in regard to treatment. Some few drugs are of value as long as they are used; but when given up, the tremor returns. Erlenmeyer found that chloral produced sleep, but had no influence upon the tremor. An infusion of valerian with bromide of potassium had a marked effect upon the tremor so long as it was used. Atropia always diminished the tremor, but he found it necessary to omit the drug on account of a slight toxic effect.

Curare, when given subcutaneously in a dose of 0·033 of a gramme, caused the tremor to diminish. This improvement continued about three days.

Eulenberg recommends very highly the subcutaneous injection of arsenic. He uses Fowler's solution diluted with two parts of distilled water, giving six to ten minims for a dose. These injections were continued daily, without unpleasant symptoms. In one case, fifteen injections, in another four, produced a very marked diminution of the tremor, lasting two months.

Hyoscyamia has been recommended, but Erlenmeyer found that it had no effect unless given in toxic doses.

The galvanic current may give relief, applying it to the head and neck; but when the treatment is discontinued, the tremor returns. Static electricity has also been used with temporary benefit.

CHAPTER XXX.

EPILEPSY.

ECHEVERRIA, M. G., On Epilepsy. New York, 1870.—BENNETT, A. H., Analysis of 100 Cases of E. *Brit. Med. Jour.*, March 22, 1879, p. 419.—JACKSON, J. H., Lectures on the Diagnosis of E. *Med. Times and Gaz.*, Jan. 11, 1879, p. 29.—MACDONALD, C. F., Feigned E. *Am. Jour. of Insanity*, July, 1880; *Boston Med. and Surg. Jour.*, Dec. 30, 1880.—WEST, J. F., On Trephining for Traumatic E. *Trans. Med.-Chir. Soc.*, 1880, p. 23.—SEGUIN, E. C., Importance of the Early Recognition of E. *Med. Record*, Aug. 6, 1881.—LUNIER, Des épileptiques ; des moyens de traitement, etc. *Annales méd psycholog.*, March, 1881, p. 217.—GOWERS, W. R., Epilepsy. London, 1881.—MARIE, P., Note sur l'état de la pupille chez les épileptiques. *Arch. de nevrol.*, iv, 1882, p. 42.—BEEVOR, C. E., On Knee-Jerk, etc., in E. *Brain*, April, 1882, p. 56.—ALEXANDER, W., The Treatment of E. by Ligature of the Vertebral Arteries. *Brain*, July, 1882, p. 170.—RUSSELL, J., The Remedies in the Treatment of E. before the Introduction of the Bromides. *Practitioner*, Feb., 1883, p. 81.—WEISS, J., Ueber E. und deren Behandlung. *Wiener Klinik*, April, 1884.—RALFE, C. H., Seventeen Cases of E. treated with Sodium Nitrite. *Brit. Med. Jour.*, Dec. 2, 1882, p. 1095.—WALSAM, W. J., On Trephining the Skull in Traumatic Epilepsy. *St. Barth. Hosp. Rep.*, 1883, p. 127.

Epilepsy is a name given to an affection whose chief characteristics are attacks, recurring with more or less regularity, in which the patient partially or entirely loses consciousness, and is generally more or less convulsed, there being no organic disease to which these convulsions can be referred as a cause.

SYMPTOMS.—In about half the patients there is a warning aura, indicating the near approach of a convulsion. This aura may consist in an involuntary mo-

tion, or a sensation in any part of the body or limbs, most frequently in the stomach; occasionally it is a sound, a sight, an odor, or it may be a confused mental action; it precedes the attack a few seconds or minutes only.

The attack may begin by a peculiar shrill cry, upon which the patient suddenly drops unconscious; or, without this, consciousness is lost, and the patient falls, perhaps injuring himself. If the aura has given sufficient warning, he may be able to sit or lie down before the attack.

In a very large proportion of cases the countenance changes, the face becomes pale, the eyes have a peculiar vacant look; tonic spasms seize the limbs and body; the patient stiffens. The attack may begin in one limb and extend to the whole body, or it may be unilateral or bilateral throughout. During the tonic stage the limbs, face, and head may be drawn into unnatural positions; there is frequently conjugate deviation of the eyes, with rotation of the head to one side.

After a few seconds or minutes clonic spasms gradually take the place of the tonic rigidity; the limbs are jerked about, the muscles of the face and mouth are affected, there is frothing at the mouth, the tongue may be bitten. The patient is not tossed about much, as occurs in hystero-epilepsy.

The countenance changes from pale to livid through venous congestion. The pupils are dilated, and do not react to light. There is conjugate deviation of the eyes, with rotation of the head to the opposite side from what it was during the tonic stage.

Cutaneous reflexes, even of the conjunctiva, are abolished during the attack.

The urine may be voided involuntarily, especially during nocturnal fits; the fæces are less commonly passed during the attack. The spasms gradually decrease in frequency; the intervals between the contractions are longer and longer, until there is quiet.

Immediately after the clonic stage, before return of consciousness, the patellar tendon reflex is increased, and ankle clonus is present in rather more than half the cases ; the tendon reflex is occasionally abolished. During this period the pupils may oscillate, the eyes roll in unison from side to side.

After lying quiet for a few minutes, the patient gradually comes to himself, being confused for a while. Often a heavy sensation in the head, or headache, succeeds the attack, continuing a few hours or days. Sometimes unilateral paralysis, or weakness, is noticed afterward, and may persist several days. Unilateral anæsthesia after the spasm is probably found only in hystero-epilepsy.

Many patients fall asleep immediately after the attack, sometimes before recovery of consciousness. This is scarcely natural sleep ; it rather resembles stupor. If the fit occurs during sleep, the patient may not awake, and in the morning be entirely unaware of what has occurred. Others will recover full possession of their faculties as suddenly as they lost them, without the least discomfort in the head or elsewhere. Sometimes the pulse is abnormally slow for several days, or even weeks, after the attack.

In the full, complete attack of epilepsy, the *loss of consciousness is absolute and entire.*

Attacks may be much less severe than those just described, consisting simply of a momentary loss of consciousness, without spasm of any kind ; or they may be reduced to the aura, consciousness not being interrupted, though mental activity and ability are undoubtedly somewhat impaired. These attacks without convulsions are called *petit mal ;* those with convulsions, *grand mal.* Every grade of severity is found, from the slightest ephemeral sensation to the severest convulsive attack.

Many authors consider loss of consciousness necessary to constitute epilepsy. Nothnagel is right, how-

ever, in saying that complete abolition of consciousness is not necessary to characterize the disease as epilepsy, "but that simple dizziness is sufficient; in fact, any alteration whatever of the mental activity occurring paroxysmally, such as hallucinations and the like."

Various unusual and exceptional manifestations of epilepsy deserve mention. Occasionally during the attack patients will perform acts which it would seem must be voluntary and conscious. In these the patient may simply perform some inoffensive act, as walking rapidly to a distance, or, as in one of my patients, a workman began to gather together pieces of wood, as if to carry home; or the acts may involve more serious consequences, as pocketing various articles not belonging to himself, or making an attack upon by-standers, and, if the impulse is strong, the patient may be thrown into a frenzy, and may commit homicide. After these acts there is no recollection of what has been done during the attack; there may be the same oppression in the head, and even the same tendency to sleep as after other attacks. Occasionally the patient seems to see objects which do not really exist; there are hallucinations of sight.

These unusual forms of epileptic seizures may coexist in the same individual with the more common, or may precede the outbreak of the latter by several months or years, or may follow after a course of remedial treatment.

The attacks vary greatly in frequency; they may be separated by an interval of months, or even years. The earlier attacks occur usually at much longer intervals than the subsequent, when the disease is left to run its course without treatment. The attacks may occur many times a day. H. Hayes Newington reports ("Journal of Mental Sciences," April, 1877, p. 89) a case in which there were six hundred and twenty-two true epileptic fits in twenty-four hours. This great number

was excessive and unusual. Often from three or four to ten or a dozen may occur in the course of the day. It is very common to have the attacks recur in groups, several within a few hours or days; then there is an interval of variable length free from attacks, and again a series of several in rapid succession.

Epileptic fits may take place either during the day or in the night; a large proportion occur in the early morning hours, just before or just after waking. It is impossible to estimate exactly the proportion of cases occurring during the night, as patients are often entirely ignorant of having had an attack. If one wakes with tongue bitten, bedclothes stained with blood and in much disorder, if the bed is wet, and there is discomfort in the head in the morning, it is more than likely that there has been an attack. Any one of the circumstances should give rise to suspicion in the case of an adult. After the attacks, small subcutaneous hæmorrhages may be seen, especially about the eyes and the rest of the face. These disappear in a few days.

The patient's limbs, and even his life, are not safe so long as he has the attacks. The fact of being in a position of iminent danger sometimes seems to prevent the occurrence of the fit; thus workmen, whose duty it is to ascend ladders or be on stagings, will sometimes be entirely free from fits in these exposed situations. It is, however, never safe to trust to such exemption. Epileptics suffer many times from bruises, and even from fractures or dislocations, as the effects of their attacks. They may also fall into the fire and receive severe burns.

Death does not often occur during the fit. Sometimes, however, cerebral hæmorrhage is the direct result of an attack. Occasionally the attacks recur with great rapidity, with scarcely a perceptible intermission; the patient is in a state of epilepsy, *status epilepticus*, as it has been called, in which the fatal termination may occur.

Between the attacks the patient may enjoy the most perfect health, and if the attacks are nocturnal, or occur in the early morning, he may be able to attend to all the duties of life—may even manage a large business without any one suspecting his infirmity. On the other hand, it is possible that the affection may lead to an impairment of mental powers, the memory at first being weakened, finally imbecility or dementia closing the scene. This is more common when the disease begins in early life. A few rarely occurring very severe attacks have generally little influence upon the mental powers; frequently recurring petit mal, giving friends much less concern, may more seriously undermine the intellect.

The paralysis, which occasionally appears after the attack as an ephemeral symptom, may become permanent, continuing from one attack to the next. In such cases it is probable there is some organic cerebral lesion.

ÆTIOLOGY.—Among those causes which give rise to a state of the nervous system predisposing to epilepsy may be mentioned heredity; not that the ancestors have necessarily had fully developed epilepsy; they may have shown only oddities of character, or mental extravagances, or they may have suffered from neuralgia or other neuroses. Parents addicted to the excessive use of alcoholic drinks may transmit to offspring a tendency to epilepsy.

The frequency with which hereditary influence can be shown to exist in this affection—eighty times in three hundred and six, according to Echeverria—should lead to caution in regard to the marriage of epileptics.

The excessive use of alcoholic drinks may develop a tendency to epilepsy in the subject; such indulgence may also be the direct cause of the attacks. Some epileptics have the attacks only after a course of rather hard drinking.

The period of puberty is that in which the disease

is most likely to be developed; much the larger number of cases begin between ten and twenty-five years of age, or even between ten and twenty.

Severe and prolonged convulsions in infancy are very frequently followed by epilepsy later in life. Apparently the cause of the earlier convulsions leads to changes in the brain, which favors their subsequent return.

Various depressing agents may be classed as predisposing causes, as worry, anxiety, grief, or excessive mental work; but it is quite likely that many times there has been in these cases a predisposition to the disease in addition to the above depressing agents.

Syphilis, especially if the bones of the skull are diseased, may be a cause of epilepsy.

Disease of the bones, induced by injuries or by other causes than syphilis, may also give rise to attacks.

The causes which may be more properly called exciting causes are blows or falls upon the head, whether giving rise to fracture of the skull or not, injuries to peripheral nerves, sunstroke, fright, anger or other violent emotion, and lead-poisoning.

Phimosis may be the cause of such an irritation as to give rise to epilepsy; it would seem to be a predisposing cause, as giving rise to such a state of the system as to favor the occurrence of epilepsy, and it also may alone excite the attacks. The same reflex influence may depend upon other sources of irritation, as that caused by teeth cutting through the gums.

Occasionally serious disorders of digestion or imprudence in diet seems to give rise to the attacks.

At the best, however, it is often impossible to discover the causes which lead to the development of the tendency, or give rise to the fits.

Brown-Séquard found that after certain lesions of the nervous system, especially in Guinea-pigs, a portion of the face suffered a change in its nutrition such

that irritation of the cheek would excite an epileptic attack; such Guinea-pigs might transmit the tendency to epilepsy to their offspring. Several times a similar epileptogenous zone has been found in epileptic patients. Otto reports a case of a patient the snapping of whose hat-elastic caused an attack.

DIAGNOSIS.—The diagnosis of epilepsy from hysteria and hystero-epilepsy will be more clear after their description in the next chapter.

The lighter attacks, the petit mal, may be recognized from common fainting or vertigo, by the regularity or frequency of their recurrence, by the brevity of the attack in epilepsy, by the occasional presence of an aura, by the occurrence of the grand mal at long intervals.

The possibility of vertigo due to Ménière's disease, or the lithemic vertigo, should be kept in mind.

Criminals and others try to simulate epilepsy to escape punishment or gain profit. A well-trained impostor is not easily discovered. The pallor of the face, the dilated pupils, reacting suddenly to light, can not be counterfeited, and it is scarcely probable that the cornea would be insensible to touch. A careful watching of all the symptoms would lead to detection of impostors, unless they were remarkably well trained. (See MacDonald's case.)

PROGNOSIS.—Most patients with epilepsy are capable of improvement; few can be expected to recover. The earlier in life the attacks begin, and the longer they have persisted, the less favorable is the prospect.

If a definite cause can be discovered, as teething, sunstroke, lead-poisoning, syphilis, etc., the chances are in favor of recovery in proportion to the readiness with which these causes can be removed, if the disease has not been of too long duration.

Mental failure is rare when the attacks begin after puberty, except in syphilitic cases; when they commence early in life, the mind is much more likely to fail.

TREATMENT.—If any source of peripheral irritation can be discovered which may act as cause of the fits, that should be removed, as cicatrices, phimosis, or irritation of gums by teeth. If fracture of skull or depression, fragments should be removed or raised.

The remedy which has been found most useful is the bromides. It is rather a matter of taste as to which bromide should be employed; the potassic, sodic, calcic, and lithic salt act very nearly alike. Sometimes an idiosyncrasy on the part of the patient will be a guide in the choice.

Gowers's method of giving the bromides is somewhat different from the ordinary way, and is of value. He gives very large doses at the beginning of treatment—two or three drachms every second or third morning, increases the dose to four drachms every fourth morning, and six drachms or an ounce every fifth morning. These large doses should be given after breakfast, in a tumblerful of water. When drowsiness and mental dullness follow during the rest of the day, he does not increase the dose. More than an ounce is rarely borne. The maximum dose should be reached in two or three weeks and repeated three or four times, and the doses then gradually reduced; the whole course lasts six or seven weeks. Unless the attacks cease entirely when a dose of four drachms is reached, he gives up the effort to cure the patient. After omitting the bromide a week or two, he gives it again regularly in smaller doses, twenty grains or more three times a day.

The usual method of giving bromide is to give a dose three times a day; usually the dose is too small. It is necessary to begin with at least fifteen or twenty grains three times a day, and the dose should be increased rapidly till some evidence of its action is obtained. Patients may take sixty to ninety grains of bromide a day for six or ten years without injury.

When large doses are given, acne appears on the face, back, etc.; three to five drops of Fowler's solu-

tion with each dose of the bromide will tend to prevent this. In very large doses patients sometimes find their legs getting weak, or lose their memory. If the dose is diminished or omitted for a few days, these symptoms disappear. In excessive doses, delirium and symptoms resembling meningitis may be produced. Of course, this ought to be avoided.

Iron, cod-liver oil, and quinine may be given to advantage with the bromides.

Occasionally the bromides seem to lose their beneficial effect, or the patient becomes disgusted with the drug. Ralfe recommends then the use of sodium nitrite, free from the nitrate.

Atropia is frequently used, either with the bromide or alone, sometimes with benefit. With it, or the extract of belladonna, may be used valerianate or oxide of zinc, and extract of hyoscyamus.

Digitalis is sometimes combined with the bromides.

Kunze* used curare subcutaneously, in 0·03 gramme doses, every fifth day for three weeks, then waited for the next attack, and repeated. The first sign of toxic action is a blurred vision.

Wildermuth† gave, with bromide, osmic acid 0·002 gramme in watery solution or pill, or 0·004 gramme potassic osmate, with benefit in an old, obstinate case when bromides alone failed.

During an attack the patient's clothes should be loosened; he should be prevented from injuring himself if the clonic convulsions are violent. A towel rolled firmly into a cone, or a piece of cork or rubber, should be put between the teeth, to prevent biting the tongue.

When an aura gives opportunity, the inhalation of nitrite of amyl may cut the attack short. Patients sometimes learn that by firmly seizing the limb in which the aura is felt, or by tightly bandaging it, they can arrest the fit.

* "Wien. med. Presse," Oct. 20, 1878.

† "Berl. kl. Wochenschr.," June 9, 1884.

In the status epilepticus, inhalation of nitrite of amyl sometimes is beneficial. Gowers finds the most good from repeated doses of chloral, morphia subcutaneously, and the application of ice to the spine.

The diet should be simple and unstimulating. It is well to restrict the use of animal food to a small amount once a day, or forbid it entirely. This diet is most valuable with young patients.

CHAPTER XXXI.

HYSTERIA AND HYSTERO-EPILEPSY.

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HYSTERIA.

Hysteria is often described as a functional disease of the nervous system; it would be more exact to speak of it as a diseased or abnormal state of the nervous system, revealing itself by peculiarities of temperament, of thought, action, and affection, with occasional outbreaks of motor or emotional disturbance.

Formerly the convulsive phenomena were considered as essential; but, while their importance is still recog-

nized, more attention has been recently given to the state of the nervous system, which renders these phenomena possible.

ÆTIOLOGY.—Briquet says half the women have a predisposition to hysteria—that is, they are very impressionable. This predisposition is also seen in a few men, and a small number of males are attacked.

The *age* at which the affection is most common lies between puberty and twenty years. After that age there is a gradual diminution in the proportion of patients. Among young girls Briquet found about a quarter or a fifth as many cases as among those who had commenced to menstruate. There are many cases reported as occurring in boys.

Heredity plays an important part in predisposing to hysteria. It will often be found that some form of nervous disturbance is prevalent among the relations of such patients. Huchard says that in hysterical women it will be found thirty times in one hundred cases that the parents have been hysterical, but in women not thus affected only four times in one hundred cases will this be found to be the case. Briquet found that a little more than half the hysterical mothers had transmitted the affection to their daughters.

It can not be doubted that the training to which young girls are subjected influences the predisposition to hysteria; their home training and school training, their food, exercise, clothing, the amount of sleep obtained, the amusements allowed, the self-restraint which they may be taught—these and many other conditions either increase their natural predisposition or diminish it.

And not only do children show the effect of their mode of life; older women show it also, and a tendency to hysteria may be created, or, if existing, may be neutralized, by the surroundings of the patient and by those indulgences which she allows herself, or by those privations to which she may of necessity be exposed.

Whatever lowers the healthy tone of the system, as a whole, may serve indirectly as a cause of hysteria: the depressing passions, care and worry, disease of any organs, but more especially of the uterus and its appendages, may give rise to the state of the nervous system which favors the development of the affection.

Accidents and bodily injuries must be counted among the predisposing causes of hysteria. Those accidents which are accompanied with nervous shock and fright, as when a horse runs away with the carriage in which the patient is riding, or railroad accidents, are most likely to give rise to this state of the nervous system.

Some of these causes which act as predisposing to hysteria may become the direct cause of an attack in patients who are otherwise predisposed thereto. Imitation may give rise directly to an attack.

SYMPTOMS.—In hysteria there is found a perversion of certain faculties and characteristics of the patients. There is a paralysis or weakness of the moral sense and of the will, an exaggeration of the emotions and the affective faculties, with irregular and perverted action of the cutaneous, visceral, and special senses. Nearly, if not quite, all the phenomena of hysteria can be explained by supposing the above changes.

The special manifestations of hysteria in different patients are almost as varied as are the diseases to which humanity is liable. Even in ordinary health these patients show a nervous mobility, a habit of exaggeration, a morbid desire for sympathy, an excessive sensitiveness and ardent imagination which is characteristic.

The disturbance of the moral sense may show itself simply by persistent exaggeration of symptoms—an exaggeration which almost amounts to simulation; or there may be deliberately planned simulation. The patient will pretend to have symptoms which do not exist; she will produce hæmorrhage from some portion

of the mouth, and pretend to have hæmoptysis; she will pretend to vomit urine instead of secreting it by the kidneys; and perhaps, in order to keep up the illusion, she will drink her urine immediately after passing it. The patient may pretend that she is paralyzed, or is so weak that she is unable to rise; yet, when she thinks herself alone, she may be seen to rise and cross the room.

A more serious and important exhibition of this propensity for lying is occasionally met in hysterical women; they will accuse a physician or dentist or priest of taking improper liberties with them, or of violating them. The records of legal medicine contain many such instances.

The foundation for some of the complaints of the hysteric may be a real discomfort; this discomfort or pain is exaggerated by the patient. A slight pain in the eyes, when open to the light, is exaggerated into severe photophobia, and the patient must remain in a dark room; yet when the oculist wishes to examine the eyes the light is born without complaint, and even the ophthalmoscope can be used without the patient's complaining. A slight discomfort in swallowing is exaggerated into entire loss of power to take food, and, if a sound is passed, a very persistent spasmodic stricture may be found. So it may be in regard to other symptoms; but it will often be very difficult to judge whether there is any foundation at all for the pretended inability.

The disturbance of the emotions and affections is sometimes very marked in hysterics. Huchard thinks it is not common to find excessive sexual excitement in hysterical patients; that sexual desire may be entirely lost. Other passions may be exaggerated; they easily acquire a repugnance for persons; they long for attention and manifestations of love from others. This peculiarity may lead to the simulation of disease; but in much the greater proportion of hysterical patients it is

shown only by a claim upon the attendants or members of the family for care and attention. Self is elevated to the highest place in these patients, and self must be served first and always. A mother may exact from a daughter constant attendance, even at the price of ruining that daughter's health; a daughter may be jealous of brothers and sisters, and become worse whenever the mother tries to give care to other members of the family.

Sometimes the patient has only one class of symptoms; probably, in such cases, there is real disease, pain, or discomfort, as a foundation for her complaints. At other times the complaints and symptoms vary from week to week, or in different attacks; even during the physician's visit she may recite a long list of ill-connected symptoms, inconsistent with one another.

"They love to carry everything to extremes, do not know how to live in simplicity; they exaggerate all their feelings, indifference as well as enthusiasm, affection as well as antipathy, love as well as hate, joy as well as despair, and they dramatize everything on the great stage of the world, where they are, and always remain, true comedians."

The intellectual condition of hysterics is generally unnatural. They are not able to turn their minds to the more serious occupations in which they may have once engaged; those who have been familiar with their mental power when in health will probably find that there has been a loss therein. Though this is true of the higher and more complicated processes of thought, yet in the ordinary round of daily life, and on a lower plane, their mental activity may be exaggerated, their conversation may be lively, animated, witty, and entertaining; they may appear even brighter than is natural, perhaps relapsing soon into silence and indifference.

The special senses, particularly hearing and smell, may be abnormally acute; occasionally hallucinations

of sight, hearing, or odor may be noticed, though it is necessary to guard against error as to the two last senses, the patient noticing sounds and odors which are not perceived by the attendants. Perversion of taste may lead to a distaste for certain articles of diet, or a desire for abnormal articles.

Ordinary sensation may be diminished or increased in acuteness; such changes are more frequent in the form called hystero-epilepsy than in simple hysteria, yet even this will show at times abnormal conditions of sensation.

Pain may be felt in different regions, and, as has been noticed, the acuteness of the pain may be much exaggerated by the patient. Headache is not uncommon, and backache is often met in these patients. The pain may be very severe just below the breast, especially on the left side. There may be severe pain and tenderness in the joints, simulating arthritis. It would, however, require much too large a space to enumerate all the changes of this nature which may be found in hysterics.

Sometimes there is a great diminution of the urinary secretion; and there may be even a temporary suppression, which has been known to extend over some days. Charcot reports a case, observed during four months, in which there was a great diminution in the amount of urine, an average of three grammes during August; on several occasions there was total suppression, once for eleven days. The patient vomited much, and the vomitus contained urea.

Among other abnormal conditions may be mentioned excessive sweating, disturbance of the gastric secretions, enormous secretion of gas in the stomach and intestines.

Paralysis affecting various parts of the motor system is sometimes seen, affecting an entire limb, or only a few muscles. Muscles thus affected give a normal reaction to the electric current. It is said by some that

normal response to the motor irritation, with loss of sensitiveness to the current, is diagnostic of hysterical paralysis. If the anæsthesia is extreme, and the reaction perfect, this may be an aid to diagnosis, but it is not alone sufficient to found a diagnosis upon. Instead of total paralysis, there may be contraction, persisting for months or years.

Aphonia may be hysterical in its origin; the patient may lose her voice entirely, and be obliged to have recourse to signs to make herself understood, or there may be an ability to whisper. This hysterical aphonia is rather intractable, and is very likely to recur.

The symptoms above noticed may appear in distinct attacks, between which the constitutional peculiarities are still to be noticed, but in less marked degree.

There are other attacks, attended with more or less violence, which are more generally known as hysterical attacks. These crises are attended with spasmodic action, or with excessive emotional disturbance; often begin with a sensation as if a ball rose from the epigastrium to the throat (*globus hystericus*), or that may be the whole of the spasmodic attack. Then, when this sensation has reached the throat, there is a general spasm, attended with screaming, with crying and sobbing, mingled with wild laughter. Sometimes this spasm commences suddenly, without the preceding aura. During the attack the patient seems to be unconscious, yet afterward may have a recollection of most that occurred. The heart may beat rapidly and strongly; the respiration is accelerated; the patient, feeling a sense of suffocation, may clutch at her throat, and try to tear her clothing. Muscular movements in these attacks are generally co-ordinated to accomplish some purpose, and are semi-voluntary, or entirely under the control of the will, though that will may be perverted. These attacks last from a few minutes to a few hours.

In the second period the loss of consciousness is not complete. The contortions are of longer duration than the epileptoid spasms, sometimes five or ten minutes; there is no foaming at the mouth, no suspension of the respiration, no inspiratory spasm, and, consequently, no turgescence of the face.

The *third period* is that of hallucinations. The patient's words and gestures are in harmony with her hallucinations. General and special sensibility are abolished.

This third period may imperceptibly merge into the fourth, or that of delirium, in which the patient seems to review the events of her past life. With this the attack may cease, the patient often passing a large amount of pale, clear urine.

Curious and important phenomena in these patients are connected with the effects of pressure over certain areas of the body. This pressure may give rise to an attack; these spots differ in different patients, and sometimes corresponding spots on both sides of the body must be pressed. These regions are just above the breasts, just below the breasts, under the axillæ, just over the crests of the ilia, between the scapulæ, and over the ovaries. Immediately after an attack, pressure over the hysterogenic zone may not induce another.

Another phenomenon is that pressure over one ovary—that which between the attacks is hyperæsthetic—will often cause the attack to cease immediately. The patient, though violently convulsed, falls on the bed entirely relaxed, and may soon recover consciousness; sometimes, however, another attack succeeds before consciousness is fully restored. This pressure must be directed so as to affect the ovary, and it may be necessary to use great force to obtain the result.

When the hysterogenic zone is not in the ovarian region, pressure over that zone may cause the attack to cease, or it may cease from pressure only when that is exerted over the ovary.

During the attack the eyes are anæsthetic, even during the period of delirium, and the cornea may be touched without exciting reflex movements of the lids; sometimes tears will flow after such a test, and sometimes the eye remains dry.

The variations of the pupils have been studied lately by Féré. During the attack the pupils are, as a rule, only slightly influenced by the light. During the first part of the first period the pupils remain contracted; immediately at the commencement of the second part of this period, when the clonic spasms set in, they are widely dilated, and remain so until the period of emotional attitudes; during that, and the period of delirium, the pupils contract and dilate according to the nature of the hallucinations, whether the objects presented to the mind seem to be near or remote.

Of course, in imperfect attacks, all these phases of the pupils may not be seen. Yet Féré noticed in one instance, where the attack consisted only of the great movements, in which the pupils are dilated, that before the attack the pupils contracted with great energy, though there was no sign of other contraction.

The condition of patients between the attacks is peculiar and interesting. As a rule, there is hemianæsthesia, affecting the side opposite the tender ovary, yet there are exceptions to this rule where the loss of sensibility is on the same side. The side which is not hemianæsthetic is in a state of hyperæsthesia.

Not only is there loss of general sensation, of touch, and pain, but the special senses are affected on the same side with the general sensibility; sight, hearing, smell, and taste may all be diminished on that side.

Féré has carefully studied the condition of the eyes in these patients. He finds the field of vision limited, almost reduced to nothing, with complete loss of perception of color. In such patients there is complete anæsthesia of the conjunctiva and cornea; the reflex action of pupil and lids is also much interfered with.

In other patients there may be only a moderate limitation of the field of vision, and partial loss of perception of color. These two kinds of visual defect generally show a direct relation and harmony between themselves. Where there is only partial loss of visual power, the anæsthesia of the eyeball varies according to the amount of visual disturbance.

DIAGNOSIS.—Sometimes the general physiognomy of the patient reveals to the physician that he has to do with a hysterical case; again, the singular combination of symptoms presented is sufficient to give a clew to the nature of the affection.

Where there are no convulsive attacks, when the patient imagines disease of some organ, the physical examination of that organ, and a comparison of the symptoms with those which ought to be present, will often be sufficient for a diagnosis. It may not be possible to arrive at a conclusion immediately, but careful observation of *all* the symptoms will generally lead to a correct diagnosis within a few days.

When the hysterical simulation takes the appearance of nervous diseases, it may not be easy to decide whether there is real disease. If the history of the patient and of the attack can be obtained, the task will be less difficult. As it would be awkward, to say the least, to treat as hysterical a patient who has a real organic lesion, great care in the first examination, and careful watching of changes, is essential. If the patient, by inheritance, age, and temperament, seems to be predisposed to hysteria, if the disease can be referred to some disturbing emotion, if its symptoms are singular and in striking contrast to those arising from any organic lesion, and if there is much variation from time to time in the symptoms, such as renders the existence of organic disease almost impossible—if these conditions exist, it may be safe to diagnosticate hysteria.

The diagnosis of the convulsive hysterical attacks from epilepsy is not always easy. The fully developed

attack of hystero-epilepsy is not to be mistaken; but the imperfect, partial attacks may not be at once recognized.

The aura is not the same; the *globus hystericus* is rarely met in true epilepsy. The manner in which the patient tells about the aura will sometimes aid in forming an opinion as to the nature of the attacks. The hysteric almost never falls suddenly, as the epileptic; there is a gradual sinking, and ability to save one's self from injury. Except in hystero-epilepsy, consciousness is not totally abolished in hysteria; the patients will remember somewhat of the events during the attack. The convulsions last longer in hysteria, and are more varied; they are also of wider excursion; the patients throw themselves about more. As the attack ends, its hysterical nature is sometimes clearly revealed by the sobbing and crying, or by the attack of laughing which follows. The state of the patient between the attacks will be an important aid to diagnosis. The epileptic, after the heaviness and dullness, or paralysis, which is sometimes seen after an attack, has passed, is as well as ever, and shows no sign of disturbance, unless the disease has begun to affect the mind. The hysteric has the psychical peculiarities between the attacks which have been previously described. If a large amount of pale urine, of low specific gravity, is passed immediately after an attack, the chances are decidedly in favor of hysteria.

PROGNOSIS.—Life is not in any special danger from hysteria. When once the predisposition has developed into the actual hysterical state, recovery is at best a distant possibility. The favorable termination will depend upon how thoroughly the patient's constitution has been undermined by early education and trials, and upon how much can be done to restore a natural vigor to the nervous system.

When the predisposition is slight, or absent, treatment is much more likely to be successful.

Relapses are very likely to occur; a patient may be well for many months, until some excitement or emotion awakens again the symptoms.

In hystero-epilepsy the prospect of recovery from the earlier attacks is the more favorable. As the frequency of the attacks increases, the prognosis becomes more grave.

TREATMENT.—Treatment should begin with children of neurotic parents about as soon as they are born, to prevent, if possible, the development of the hysterical predisposition. Care in regard to the child's surroundings, its food, exercise, sleep, study, and government, is to be continued throughout childhood and youth. Sometimes the mother is not suitable to look after the child, and the care must be given to others. In all such cases the family physician has it in his power to do much by advice and warning. Huchard formulates this line of treatment thus: "To favor the physical development at the expense of the moral and intellectual development."

After the disease has once shown itself, it will be necessary to use all the hygienic means at one's command to counteract the predisposition, and the patient's life may need to be watched and regulated for months or years. Often this can be done better away from home. The patient is self-willed, and needs to be under control, which will not be exercised at home.

As disease of one of the viscera may be a principal cause of hysteria, every organ should be examined, and, if really diseased, the disorder should be corrected if possible. The physician will, of course, bear in mind the fact that functional disturbances may be found in any part of the system. He will also do well to bear in mind that too much notice given by him to any organ may call the patient's attention to that part of the body, and so its condition be made worse. This tendency of the patient may be utilized by the physician to turn the patient's attention away from the dis-

eased organ while he really administers medicine for that.

The management of the patient between the attacks is of great importance; or, if there has been no fully developed attack, but only such an unstable nervous condition as to give rise to fear of an attack, the general management is important. This will include hygienic and dietetic agencies; but care must also be given to develop the patient's power of will, of self-restraint, and her ability to meet and resist the ordinary little disturbances which arise in daily life, as well as the more serious trials. The physician would not be wise to entirely ignore the patient's complaints; he should not set aside the symptoms mentioned as entirely imaginary, but, while letting the patient feel that she has his sympathy, and that he understands her troubles, he should also show her that, by a resolute effort of will, she can do much to help herself. He can not do this on the first visit, nor perhaps until after he has allowed time enough to elapse to win her confidence. In other cases, comparative harshness and disregard of complaints from the first will have a better effect. It will sometimes be necessary to remove the patient from home, and to seclude her in a measure, or entirely, giving her into the care of kind but firm nurses.

The boundary between hysteria and insanity is by no means well defined, and the question may arise as to the propriety of asylum treatment. Each case must be judged upon its own merits.

Of drugs which may be used, those which will act as tonic to the nervous system, and increase the vigor of the health generally, should be given, in order to remedy the natural predisposition of the patients. Besides the ordinary tonics, arsenic, zinc, and phosphorus, in their different pharmaceutical forms, may prove of benefit; valerianate of zinc, in two- or three-grain doses, is specially valuable. Cod-liver oil, or, if that can not be taken, cream, is a useful addition to the diet. If the

heart is irregular or feeble, a short course of digitalis may be of value.

Hysterical patients are very often addicted to the use of preparations of opium to relieve real or imaginary pain. Opiates should be used with extreme caution. A patient who has taken much opium loses her power of bearing pain, and when to the slight discomfort is added the craving for the drug, she can not or will not distinguish between the two sensations, and calls loudly for the opiate. If it is withheld, the need of it ceases after a while. Of course, if the patient is a confirmed opium-taker, the task of weaning her from the habit becomes the more difficult.

As a substitute for opium, recourse may be had to external applications, to atropia, hyoscyamus, conium, and cannabis Indica; subcutaneous injections of water will sometimes be of advantage.

Bromide of potassium and chloral had better be used only occasionally. If their use is habitual, the result is bad. Sleeplessness can be remedied often by massage, by exercise, by healthy occupation of the mind, by removing digestive disturbances, sometimes by a meal or a slight stimulant given at bed-time. Care should be exercised in using alcoholic stimulants, as hysterical patients easily acquire a craving for such.

During an emotional attack, various preparations of valerian, asafœtida, musk, etc., can be given; yet they are by no means always successful. Inhalations of nitrite of amyl, ammonia, and ether can be tried.

The convulsive attacks can sometimes be cut short by a command given in a sudden and authoritative manner; at other times a slap, or a glass of water thrown in the face, may stop the attack. There are some objections to this procedure, especially as the water wets both patient and bed. In hystero-epilepsy, compression of the ovary will almost always cut short the attack; the compression must be directed toward the ovary, and be quite strong.

An ice-bag placed over the hyperæsthetic ovary for half an hour, or longer, morning and evening, may have a beneficial effect in diminishing the frequency of convulsive attacks. Blisters may produce a similar effect.

Féré has caused an ovarian compressor to be made which can be worn by the patient; this causes the attacks to be postponed. Inhalation of ether may cause the attack to cease.

An indifference to attacks, in which the patient does not lose consciousness, is a useful means of diminishing their frequency and their violence, while too much curiosity and attention will cause the attacks to be more frequent and more severe.

The question of removal of the ovaries has been somewhat discussed. Several cases of recovery have been reported. The operation should be kept in mind with reference to severe intractable cases where there is evident disease of the organs.

The phenomena called metallotherapy are of considerable interest. In some cases of hystero-epilepsy, if a metal disk is bound upon an anæsthetic part, in ten to twenty minutes sensation returns; the return of sensation is preceded or accompanied by a pricking sensation. The return of sensation is not confined to the area covered by the metal, but extends beyond the disk, mostly parallel with the axis of the limb, if the metal is on a limb. Patients are not all susceptible to the same metal; one may be influenced by iron, another by copper, another by lead, and so on.

When sensation returns in a part of the anæsthetic side in consequence of the application of metal, it will be found that a corresponding spot of skin on the sound side has lost its hyperæsthesia.

There may be a similar transfer of muscular power, of vision, of hearing, taste, and smell. The circulation is affected, and the temperature may be altered.

When the metal disk is removed, the anæsthesia,

etc., oscillates for a short time between the two sides, until finally the parts recover their previous condition. Only a small proportion of the patients affected receive any permanent benefit from these applications.

Burq first, in recent times, called attention to this influence of metals. Charcot, his pupils, and many others, have followed up these investigations. Many curious and interesting facts have been learned which can not be given here.

Among agents which have been experimented with, and which may be used with some benefit, especially in hystero-epilepsy, may be mentioned the magnet, the application of which, to the skin, causes a transfer of sensation. Static electricity, in the form of direct discharges, or discharges from Leyden jars, will cause a transfer; prolonged static baths are said to have a permanent effect.

The application of both the galvanic and interrupted currents are often of value. The application may be made to the affected parts: if there is anæsthesia, the wire brush to the skin; if paralysis, the skin should be moistened, so that the electricity can pass through to the muscles. Beard and Rockwell's method of general electrization may be used; the feet of the patient are put in a basin of water (not a metallic basin), in which one pole is placed; the other pole may be passed over the patient's neck and shoulders, or may be placed in another basin of water, in which his hands are dipped.

CHAPTER XXXII.

NEURASTHENIA.

MITCHELL, S. WEIR, Fat and Blood. Philadelphia, 1884.—BEARD, G. M., A Practical Treatise on Nervous Exhaustion. New York, 1880.—MITCHELL, Lectures on the Diseases of the Nervous System. Philadelphia, 1881.—PLAYFAIR, W. S., The Systematic Treatment of Nerve-Prostration and Hysteria. London, 1883.—CLARK, F. LE GROS, Some Remarks on Nervous Exhaustion and on Vaso-Motor Action. *Jour. of Anat. and Physiol.*, April, 1884, p. 239.

Neurasthenia means simply an exhaustion, and consequent weakness, of the nervous system in general. During the last few years this condition has attracted much attention, and has been looked upon as a separate, independent affection. Beard led the attention in recent years to the many various disturbances which can be classed under this head.

ÆTIOLOGY.—The causes of neurasthenia are very numerous, the most important, perhaps, being the mode of life, habits, and customs of the present generation.

Heredity certainly plays an important part in the ætiology, many of the patients having a father or mother, or both parents, similarly affected, or suffering from some debilitating disease, as phthisis. Women are the most subject to the disease, although men are by no means exempt.

Of other causes, the training which the child received in its infancy and early years acts powerfully; the school-life and the home-life both aid in developing a weakened nervous constitution. In adult years, the wear and tear of business and of social life, the

anxieties and worries, the disappointments frequently met in the struggle for existence, aid also in the same direction. The way houses are built, the way they are warmed and ventilated, habits in regard to diet, in regard to sleeping, exercise, employment, and amusement, must be reckoned as favoring the development of this affection. To go into particulars would require an enumeration of every violation of the laws of health and hygiene which are so common at the present time.

PATHOLOGICAL ANATOMY.—There is probably no special pathological change to be discovered on inspection, or the minutest examination of the nervous system. In the vast majority of cases the disturbance is purely functional, at least in the commencement. In a few cases there is a strong suspicion or probability that the nervous exhaustion leads finally to structural changes, as sclerosis; this is, however, by no means positively proved. Most patients, however, have an unhealthy complexion. There is an expression about the eyes and mouth which is characteristic. The gait and other movements of the patient are also more or less characteristic of the languor and discomfort experienced. These peculiarities can not be well described, but can be learned by observation.

SYMPTOMS.—A patient usually comes only gradually to realize that his health is impaired.

The first symptoms are those of languor, of disinclination for exertion. The patient finds it necessary to rouse himself by an effort of the will to perform his daily duties. This languor and lassitude may be accompanied with more or less discomfort in the head, perhaps amounting to pain; or there may be a sense of weakness across the back, and pain along the spine. Sometimes there are various abnormal sensations in the limbs.

Attending the pain in the head or back, there is usually more or less tenderness on pressure over the scalp, or the spinous processes of the vertebræ. The

back of the head and upper part of the neck are very likely to be the seat of the pain. This tenderness may be excessive. This is the condition which has been called "spinal irritation." It is often attended with motor and sensory disturbance of the limbs, according to the level at which it may be. There is no need to raise this symptom to the dignity of a separate affection; it is merely the result of the general nervous exhaustion localized.

Noises in the ears, of various kinds, may also be noticed. The pupils may be widely dilated, or have an unusual mobility.

Loss of sleep is sometimes very distressing; the patient may find it difficult to get asleep, and lie awake half the night, or he may have no difficulty in dropping to sleep when he first goes to bed, but, waking up after an hour or two, lies awake for several hours. Sometimes, when the sleep seems to be sound during the whole night, the patient awakes unrefreshed, feeling as tired as when he went to bed. It is no uncommon thing to have the patient feel sleepy before going to bed, unable to do anything on account of the extreme drowsiness, and then, on retiring, he is as wide awake as possible.

When the disturbance has continued some time, and advanced considerably, there may be an irritability of temper and a change of disposition, which renders it very trying to get along with such patients. He may be unable to control his mental operations. Reading even a few sentences is fatiguing, or it is impossible to understand anything that is read, so that all intellectual work must perforce be abandoned.

Various disturbances of the secretions, either a deficiency or an increase of perspiration, or of the saliva, or of urine, show that the secretory functions are interfered with. Many patients cry very easily in a hysterical manner.

The voice may be changed and peculiar. A com-

plaining, weak, high-pitched voice is sometimes met. The vaso-motor system shows a certain amount of instability. It is common for patients to blush easily on the slightest provocation. There is also frequently a sensation of heat, flushing of the face and head, which at times is extremely disagreeable, almost painful. This sense of heat may also be experienced through the back and limbs, and alternate with chills creeping over the body.

Perhaps, owing in part to the instability of the vaso-motor system, these patients are very susceptible to changes of weather; especially, dull, cloudy, and cold weather is found to be disagreeable. Many times, also, the heat of summer is oppressive, though the patients very often desire the rooms in which they dwell during the winter to be kept at a high temperature. The strength is very easily exhausted. Having no reserve force, if there is an extra demand for exertion they find themselves unable to meet the emergency, and hence are quickly tired. They may be entirely incapacitated for the ordinary duties of life, in consequence of lack of power for sustained exertion.

The symptoms in neurasthenia change and vary from time to time even in the same patient. There are no two patients in whom the group of symptoms is the same. Also, the symptoms can not be reconciled with an organic change in any part of the nervous system. They are too variable and too contradictory to have any such sound basis. Many of these patients are more or less hysterical, and it is sufficient that the physician should mention symptoms for the patient to have them at the next examination.

Diagnosis.—The diagnosis of nervous exhaustion can be made only after a careful study of the symptoms, both subjective and objective.

The accounts already given of various organic diseases, with their symptoms, will be sufficient to enable any one, after a careful examination, to decide whether

there is any such organic change. If not, and if the symptoms are frequently changing, and if the patient has a semi-hysterical appearance, seems to be dwelling a great deal upon his own symptoms, over-anxious about himself, it will be pretty safe to decide that there is no organic change, but that the disturbance is simply nervous exhaustion.

PROGNOSIS.—The chances of recovery in these patients depends a great deal upon whether the exhaustion is excessive, and whether the patients have, through several years, gradually reached the condition in which they are found.

At the very best, it will require many months, perhaps years, for a satisfactory recovery; and, if the patient is somewhat advanced in years, he can never regain the vigor of earlier life. Death almost never results as a consequence of nervous exhaustion, though it is possible that, after several years, organic changes may be set up in the nervous system, which may then lead to a fatal termination.

Insanity, especially melancholia, is not very likely to occur as the sequel of neurasthenia. Many patients neither get well nor grow worse, but live an invalid life, suffering greatly, having very little comfort in themselves, and feeling that they are a burden and care to their friends, until some intercurrent disease ends their life.

TREATMENT.—One of the first requisites in treatment of such patients is rest, and many of the patients require bodily as well as mental rest. If the disease is but slightly advanced, it may be sufficient to send the patient away from home to get him out of the regular ruts of life, away from business and its cares, or, in the case of women, away from household duties and anxieties, or the excitement of fashionable life.

Traveling is rarely of benefit. It is better for the patient to go to some retired place where recreation and amusement can be obtained sufficient to make the time

pass pleasantly, and, settling down, he should determine to obtain the greatest amount of rest possible.

In cases where the disease is rather advanced, it is much better that the patient should be taken away from home, away from the care and the sympathy of friends, placed among comparative strangers, and subjected to the treatment which Weir Mitchell has so ably described in his two little books. This treatment consists in putting the patient to bed, feeding systematically at first with milk, and later with other easily assimilated food, supplying the place of exercise by massage and electricity.

These patients usually have much trouble in sleeping. It may at first be necessary to give various remedies in order to obtain quiet rest at night. A good dose of opium, or occasionally bromide of potassium, may be of advantage. The bromide, however, should be given in divided doses, beginning about the middle of the day, so that the patient will take three doses before night. Chloral should rarely be given; paraldehyde in doses of thirty to fifty minims is much better. Valerianate of zinc, combined with extract of hyoscyamus and extract of conium, as in the following prescription:

℞ Zinci valerianatis.....gr. ij;
Ext. hyoscyami, } ..āā gr. ½ to ⅔. M.
Ext. conii, }
Ft. pil.

given three times a day, will sometimes quiet the nervous restlessness, and favor sleep better than anything else. This combination also has the advantage of being slightly laxative. If there is instability of the vasomotor system, flushings, and chills, it may be an advantage to combine with this ergot, cod-liver oil, and other tonics; arsenic, iron, strychnia, etc., may be given as seems most desirable.

Electricity is useful, not only to obtain the passive exercise of the muscles, but, given in the form of gen-

eral faradization, as described by Beard and Rockwell, is often of very great advantage, acting as a general tonic; the static form is also useful.

It may be well to say a few words in regard to feeding. Most of these patients are underfed. In many there is nervous dyspepsia, and the stomach will bear but a small amount of food at one time. It is necessary, therefore, to feed frequently. Occasionally as often as every half-hour a few spoonfuls may be given. Milk, or some of the various preparations, as Ridge's Food, or Mellin's Food, can be given at first, but soon other things may be joined with it, as has been already mentioned in regard to the feeding of patients.

To carry out a successful treatment of these patients requires a great deal of tact and perseverance on the part of both the physician and the patient. The patient must stay in bed long enough. The mistake is more frequently made of not keeping the patients confined as long as is necessary rather than of keeping them in bed too long.

The methods of treatment while the patient is in bed must be changed and varied as circumstances require. Where there is extreme exhaustion, very little attention needs to be paid to the amusement or the recreation of the patient while thus confined. As the patient, however, gains strength, it may be well to allow some reading. After a while the patient may be allowed to read a little himself. Then other light employments may be gradually taken up; but it is necessary to remember that sewing, knitting, crocheting, etc., are really a severe tax upon the muscular system, and will often of themselves produce pain in the back and head, so that it is necessary to limit the time of such employment.

When the patient begins to get up, it will be necessary to carefully regulate the amount of exercise and exertion, in order that he may not overtax himself, and so be put back and delayed in recovery.

Many times it is necessary to confine the patient to the bed all day long. He can change from bed to lounge, or couch, and back again. At first it may be necessary to exclude friends; but, later, a friend, who has the wisdom not to stay too long and not talk too fast or much, and who does not tire the patient, may be admitted as may seem most desirable.

CHAPTER XXXIII.

TETANUS AND TETANY.

AUFRECHT, Zur pathol. Anatomie des Rückenmarks beim Tetanus. *Deut. med. Wochen.*, No. 14, 15, 1878, and *London Med. Rec.*, June 15, 1878, p. 238.—COATS, J., On the Pathology of Tetanus and Hydrophobia. *Med.-Chir. Trans.*, 61, 1878, p. 79.—WOODS, G. A., A Contribution to the Pathology of Tetanus. *Lancet*, Sept. 7, 1878, p. 326.—WOOD, H. C., Abstract of a Lecture on a Case of Idiopathic Tetanus. *Philadelphia Med. Times*, March 15, 1879, p. 273.—KNECHT, Beiträge zur Lehre vom Tetanus. *Schmidt's Jahrb.*, 181, 182, 1879.—MACDOUGALL, J. A., The Ætiology of Tetanus, etc. *Lancet*, July 19, 1884, p. 98.—BOWLBY, A. A., Five Cases of Tetanus, with some Remarks on its Pathology. *St. Barth. Hosp. Rep.*, 1883, p. 85.

Tetany.—TROUSSEAU, Lectures on Clinical Medicine. *New Syden. Soc.*, 1868, vol. i.—ERB, W., Zur Lehre von Tetanie. *Arch. f. Psych.*, iv, 1874, p. 271.—CHVOSTEK, Beitrag zur Tetanie. *Wien. med. Presse*, 1876, 1878, 1879.—WEISS, N., Ueber Tetanie. *Volkmann's Sammlung*, No. 189, 1881.—GOWERS, W. R., Clinical Lecture on Tetany. *Lancet*, July 21, 1883, p. 92.

TETANUS (LOCKED-JAW).

Tetanus is a continuous tonic spasm of the muscles, due to an increase of the reflex irritability in consequence of an injury, though sometimes apparently the result simply of a chill.

ÆTIOLOGY.—Injuries of the extremities are more frequently the cause of tetanus than those of the trunk. Both severe and slight wounds may be followed by the spasms, which may occur before the wound is healed, or only a long time afterward.

Exposure to the inclemency of the weather, and various hardships, also lack of cleanliness in the care

of the wound, are more likely to be associated as causes of the attack.

The disease is somewhat common in military surgery, and the soldiers of the vanquished party are more likely to be affected than the victors. In civil life, men are more commonly affected than women, being more exposed to injuries.

The disease is very common in certain countries in new-born children, especially during the first nine days of life.

Children are more likely to be affected in warm climates; and there are also districts in temperate zones where the disease is more likely to occur, as along the southern and eastern shore of Long Island.

It is said that the colored races are more likely to be attacked than whites.

SYMPTOMS.—The first symptom is usually a stiffness of the jaws (trismus), which renders it somewhat difficult to open the mouth. Then there is a slight spasm in the muscles of the neck, and later this spasm extends to the muscles of the back and of the trunk. The severity of the contraction steadily increases, and, the extensors being the most powerful, the body is generally drawn backward, so as to form an arch, and, when the spasm is extreme, the patient rests upon his head and heels, the body being arched above the bed (opisthotonus).

The arms and hands are often unaffected, though they may be rigid in extension.

The contraction of the muscles is attended with severe, cramp-like pains, producing extreme distress. The skin is frequently covered with perspiration during the spasm. The contraction of the respiratory muscles interferes with breathing, so that at the height of the spasm the patient has a sensation of impending suffocation. Owing to the closure of the jaws, the patient finds great difficulty in speaking, and it is almost impossible to feed him.

The muscles are continuously in a state of contraction, but are not always contracted to such an extreme degree, remissions occurring, during which the patient may resume the ordinary position in bed, be able to take food, and converse; yet the slightest irritation, even breathing upon the patient, or a slight jar of the room, or an attempt to swallow, will cause a recurrence of the extreme spasm.

The temperature is but little affected except just before death, when it may rise as high as 112° or 113°. The pulse, also, is nearly normal, except toward death it may, with the rise of temperature, become very rapid.

The mind is usually unaffected, except toward the close of life, when there may be delirium. Occasionally the patient dies in consequence of the disturbance of respiration; but this is not very common. Many times the patient dies in the interval between the spasms, apparently worn out by the disease.

PATHOLOGICAL ANATOMY.—The pathological changes found in tetanus do not explain the symptoms, nearly all being apparently the effects of the disease rather than the cause.

In many cases, however, inflammation of the nerves leading from the seat of the wound have been recognized, and occasionally there have been found changes in the spinal cord, the blood-vessels being surrounded by leucocytes. Ross mentions finding these bodies in the gray and white substance, around the vessels, though not usually aggregated in the perivascular spaces, as in hydrophobia. He also found some changes in the ganglion-cells of the anterior cornua.

DIAGNOSIS.—It is scarcely possible to mistake tetanus when well marked.

It is important to make a diagnosis as early as possible, even when there is only the first symptom of trismus.

Strychnia-poisoning resembles tetanus in some respects, but the spasms are less continuous, having pe-

tient may be entirely free, until after some months the attacks reappear. One of my patients had these attacks regularly twice a year, lasting each time three months.

Trousseau has mentioned that in the interval between the spasms a compression over the track of the nerves or the vessels will cause the attack, which continues as long as the compression is maintained, ceasing as soon as the pressure is removed.

After the attack there is for a short time loss of power in the limbs affected. Sensation is likely also to be diminished.

Trousseau's middle form of the disease combines with the spasms already mentioned other general symptoms, which he mentions as feverishness, headache, loss of appetite, slight congestions in different parts of the body; the spasms are more severe, return more frequently, and affect the muscles of the trunk and face, as well as those of the extremity. He has also enumerated a third and more grave form of the disease, which differs in nothing from the other except in being more severe.

Sometimes the patients recognize that an attack is approaching, by unpleasant sensations in the hands and feet, and slight stiffness in moving.

Several authors have recognized a very great increase of electrical irritability in both the nerves and muscles of the affected limbs during the attack.

The disease is of long duration when once it appears, extending, with the intermissions, through many years.

ÆTIOLOGY.—Very little is known as to the cause of this disease. "Catching cold" is often mentioned as the starting-point of the spasms. Various exhausting influences seem to act as causes. One patient stated that eating and drinking would bring on an attack during the time when he was liable to have them.

PATHOLOGICAL ANATOMY.—Very few autopsies have been made, and but little is known as to the changes in

the nerves or their centers. Weiss found swelling of the ganglion-cells of the anterior cornua, with a lateral position of their nuclei and vacuoles in the cells and their processes; also atrophy of the cells, with loss of their protoplasmic processes.

DIAGNOSIS.—The character of the spasm, the intermission of the attacks, and especially Trousseau's manœuvre of pressing upon the nerve or vessel of the limb, are sufficient to determine the nature of the disease.

PROGNOSIS.—The disease is rarely fatal, though a few cases of death have been reported. The patient, having passed through one attack, is not safe from subsequent attacks.

TREATMENT.—It seems as though very little could be done to cut short the spasms. Opium, belladonna, chloral, and inhalations of ether may be used as indicated.

Electricity may be tried, either by faradizing the muscles that are not affected, or the use of galvanism to the nerve-centers.

Erb saw a recovery take place from the stabile application of the anode to the vertebral column and the nerve-trunks chiefly affected. I used electricity in one case in every way I could think of, without a particle of benefit. I have obtained more benefit from the use of the fluid extract of conium, twenty drops every two hours, than from any other remedy. Between the attacks, quinine, arsenic, and valerianate of zinc, and other nerve-tonics, should be given.

CHAPTER XXXIV.

MYXŒDEMA.

ORD, On Myxœdema. *Med.-Chir. Trans.*, lxi, 1878.—CUSHIER, ELIZABETH, M., A Case of Myxœdema. *Archives of Med.*, Dec., 1882, p. 203.—OLIVER, T., Clinical Lecture on Myxœdema. *Brit. Med. Jour.*, March 17, 1883, p. 502.—EDES, R. T., Clinical Lecture on a Case of Myxœdema. *Boston Med. and Surg. Jour.*, April 24, 1884, p. 385.—WEST, E. G., A Case of Myxœdema, with Autopsy. *Boston Med. and Surg. Jour.*, July 17, 1884, p. 50.

Myxœdema consists essentially in an increase of the subcutaneous tissue, which is infiltrated with mucin, so that the general appearance is that of œdema, yet the skin does not pit on pressure.

SYMPTOMS.—The patient's appearance is very peculiar. The eyelids are thick, as if swollen with crying, or infiltrated with serum; the nose is very broad, the lips thick, the hands are large and misshapen, the fingers being club-shaped. The feet are usually affected the same as the hands; the swelling sometimes extends to other parts of the body, especially to the arms and legs, and even to the trunk itself. The tongue is usually very much swollen. The patient has many times a waxy or anæmic complexion, the red-blood corpuscles being diminished in number. The infiltrated tissue has a semi-translucent appearance.

The motions are necessarily slow and difficult. The patient manages his large fingers in a clumsy way, so that the more delicate manipulations of writing or sewing are illy performed. The gait of the patient in walking is slow, as if great exertion were required.

Ordinary sensation is diminished, and the special

sensations of taste and smell may also be diminished, probably on account of the swelling of the mucous membrane of the nose.

In some cases the temperature has been noticed to be above normal, but it is usually diminished. The pulse is generally slow.

In most of the cases observed the intelligence has seemed to be affected. The patient answers questions slowly, as if it were an effort to think or speak. She seems indifferent to her surroundings. Memory may be weakened. The simplest operations of arithmetic are performed with difficulty, or the patient is unable to give correct answers.

Sometimes the hair falls out. The nails become brittle and furrowed.

The digestion is affected only when the disease has reached an extreme degree.

Constipation is very common. Menstruation is often irregular. In some cases the thyroid gland has been very much diminished in size.

The succession of these symptoms may vary somewhat in different cases. Sometimes the mental depression, even reaching the degree of melancholia, may appear early; or, on the other hand, there may be a very great amount of swelling, with the mind almost entirely unaffected.

ÆTIOLOGY.—The causes of this disease are very obscure. In several cases worry or anxiety, or some mental shock, has preceded the attack. We, however, in fact, know almost nothing in regard to the real cause of the affection.

The *course* of the disease is very slow, most of the cases extending over several years.

PATHOGENESIS.—The nature of this disease is as yet imperfectly known. On the one hand, it is supposed that the changes in the skin are primary, and that, owing to defective sensation, the cerebral disturbance follows as a result of the cutaneous change.

Another view is that the disease is due to disturbance of the nervous system, either the sympathetic or a more general disturbance involving other than the sympathetic system, and the cutaneous changes are looked upon as secondary.

In the few autopsies which have been made, the examination has not been, as a rule, complete; in each case some important parts were overlooked, and not examined. Changes have been found in the spinal cord, in the blood-vessels, and nerve-cells.

Dr. E. M. Cushier ("Archives of Medicine," 1882, p. 216) says: "The disease in question can only be described as a nutritive disturbance, resulting in the presence, in the connective tissue, of a substance common in embryonic tissue, but not existing normally, excepting in very small amounts, in adult life."

TREATMENT is comparatively unsatisfactory; almost every means of arresting the progress of the disease has failed. A few cases have been reported in which the use of tonics, such as iron, quinine and strychnia, warm-air baths, massage, and general hygienic treatment, have seemed to be of some advantage. The double chloride of gold and sodium has been recommended. Dr. Edes refers to a curious case, in which abdominal dropsy occurred, and became so severe as to require tapping, after which operation both the ascites and myxœdema disappeared.

CHAPTER XXXV.

TOXIC NEUROSES.

Lead.—Bernhardt. *Arch. f. Psych. u. Nervenk.*, iv, 1874, p. 601.—Westphal, C. *Ibid.*, p. 776.—Riegel, F. *Deut. Arch. f. kl. Med.*, 1878, p. 175.—De Watteville, A. *Lancet*, July 10, 1880, p. 44.—Monakow. *Arch. f. Psych.*, x, 1880, p. 495.—Zunker. *Zeitschr. f. kl. Med.*, 1880, p. 496.—Birdsall, W. R. *Amer. Jour. of Neurol. and Psych.*, May, 1882, p. 176.—Webber, S. G. *Arch. of Med.*, Aug., 1882.—Putnam, J. J. *Boston Med. and Surg. Jour.*, 1883, p. 315.

Arsenic.—Seeligmüller, A. *Deut. med. Wochenschr.*, No. 14, 1881.—Popow, N. *St. Petersb. med. Wochenschr.*, 1881, p. 311.—Da Costa, J. M. *Philadelphia Med. Times*, March 26, July 2, 1881.—Mills, C. K. *Med. News*, March 3, 1883, p. 257. —Seguin, E. C. *Jour. of Nerv. and Mental Dis.*, Oct., 1882 ; *Opera Minora*, 1884.

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BROADBENT, Cases of Supposed Hydrophobia treated by Chloral, one of which recovered. *Med. Times and Gaz.*, March 17, 1883, p. 308.

CHRONIC LEAD-POISONING.

Lead may be taken into the system through the skin, by the mouth with food, or, inhaled as dust, may be swallowed with the saliva. The occupations in which lead is used are readily recognized in most cases; among those less known as dangerous may be mentioned file-cutters, brush-makers, workers in enamel, in colored papers, in lace, and in rubber-factories.

Food preserved in tin, or drink passing through lead pipes, or stored in lead-lined cisterns, not only ordinary drinking-water, but mineral-waters, ale, beer, etc., are likely to contain lead.

SYMPTOMS.—The time when the symptoms appear after exposure varies from a few days to several years.

Anæmia is one of the earliest and most common effects of lead. The red corpuscles may be reduced one third in number; their size is also slightly increased. The patient is sallow; the skin is dry and harsh, sometimes œdematous. A narrow line of bluish-purple color may be frequently noticed at the edge of the gums in patients who do not cleanse their teeth.

Lead colic is a well-known symptom. Preceding the attack, the appetite may have failed for some days, there may have been a sweetish or disagreeable metallic taste, and a general feeling of ill-ease. Constipation is usually present. The pain is light at first; gradually increases until it is of extreme severity. The spasms of pain are usually of short duration, but recur frequently, so as to be almost continuous. The patient can not keep quiet; he tosses about. Generally there is no tenderness of the abdomen; pressure may give temporary relief; nausea and vomiting may occur.

The pulse is hard; vascular tension is increased. The sphygmograph shows a slight notch at the apex of

the curve, a peculiar cupping, by which two points or teeth are produced.

Arthralgia is said to be next in frequency to colic as a symptom of lead-poisoning. The pains resemble neuralgia; the joints are somewhat swollen, and may be red; sometimes there are cramp-like pains in the muscles.

The *paralysis* of lead-poisoning usually affects the extensor muscles of the fingers and wrists; is almost always bilateral, though it may begin on one side earlier than the other. One or more attacks of colic, or arthralgia, may have preceded, and there has generally been abnormal sensation in the parts, pricking and tingling, as if the limb were asleep. The supinator longus is rarely affected; the deltoid is paralyzed rather than the biceps. The flexors of the fingers always seem weak when the extensors are paralyzed. The legs are much less frequently affected than the arms; sometimes the loss of power is noticed in all four limbs. When cosmetics are the cause of the poisoning, the muscles of the face may be paralyzed, otherwise they are usually exempt.

The onset of the paralysis is gradual; sometimes, however, a day or two is sufficient to render the hands helpless. The muscles undergo atrophy, which may be extreme. The electrical reaction is diminished, or lost; the reaction of degeneration may be recognized, unless the atrophy has progressed too far.

There may be partial loss of sensation on one or both sides; this, however, is rare.

Tremor, resembling paralysis agitans, is occasionally seen.

Instead of symptoms of peripheral paralysis, those of *myelitis* may be the only evidence of lead-poisoning. So close is the resemblance that it may be impossible to form a diagnosis from the symptoms alone. In order to form a correct opinion, it will be necessary to give iodide of potassium, and after a week or so ex-

amine the urine for lead. Every case of chronic myelitis should be thus examined for lead.

The severest form in which lead-poisoning shows itself is seen when the brain is affected—*encephalopathia saturnina*. The symptoms are headache, or simply discomfort in the head, incapacity for mental exertion, amblyopia and amaurosis, delirium, or even maniacal excitement, and epileptiform attacks. Beginning with the milder manifestations, the severe symptoms may follow within a few days, until a fatal termination is reached.

Many times albumen will be found in the urine, and hyaline casts are not uncommon.

PATHOLOGICAL ANATOMY.—Lead has been found in nearly all the tissues of the body; but its presence is not constant, and the symptoms do not seem to depend thereupon.

The nerves supplying the paralyzed muscles have been found atrophied and degenerated. Changes have been found in the nerve-cells of the anterior cornua of the spinal cord, also in the vessels of the cord. In many autopsies no such changes have been found.

The muscles undergo fatty granular degeneration.

PROGNOSIS.—It is very rare for lead colic and arthralgia to terminate fatally; relapses, or repeated attacks, are usual, if the patient continues exposed to lead. Lead paralysis is also seldom fatal unless the exposure to the poison has been very prolonged, and the symptoms have been neglected. Under proper treatment, recovery is the rule; but many months or years may be necessary for restoration of function.

The prognosis in tremor from lead is favorable.

When the cerebral symptoms are slight, chiefly headache and mental inertia, the chances of recovery are good; the same is true even in cases of delirium and mania, though then the prospect is more serious. In *eclampsia saturnina* the patient almost invariably dies.

TREATMENT.—It is scarcely necessary to say that means should be taken by all workers in lead to prevent its introduction into the system, or that, with the first symptoms of poisoning, there should be yet greater care.

Iodide of potassium should be given to remove the lead from the system. During treatment the physician must watch lest the liberation of lead from the tissues should give rise to a recurrence of acute symptoms.

Warm baths are of value, by maintaining the activity of the skin and favoring metamorphosis of tissues. Iodide of iron is of value to improve the quality of the blood.

During the attack of colic, morphia should be freely used; it will aid in relaxing spasm, and, with a cathartic, aid in opening the bowels; atropia may be combined with it to advantage.

Nitrite of amyl inhaled may often relieve the pain and shorten the attack; it also restores the normal tension of the vessels and the normal character to the sphygmographic tracing of the pulse.

To relieve the arthralgia, warm baths, sometimes cold packing, and the galvanic current applied locally to the affected joint, are of value. Sometimes tincture of iodine, though increasing the pain temporarily, may give relief subsequently.

Paralysis should be treated by massage, warm baths, and electricity; the interrupted galvanic current, if the muscles do not react to the faradic. In obstinate cases, strychnia by the mouth, or subcutaneously, is said to be of advantage; comparatively large doses are required. *The treatment must be persevered in for months or years, and not hastily abandoned.*

In cases where there seems to be myelitis, and in cerebral cases, the same course should be pursued for eliminating lead. The galvanic current to the head will sometimes relieve headache.

ARSENIC.

Arsenical poisoning is found among the various artificers in that metal; also among those who use fabrics containing arsenic in the dyes, as artificial-flower makers, seamstresses, and paper-hangers. Wall-papers, cretonnes, etc., bring others in contact with the poison.

Among some of the more common symptoms are disorders of the digestive organs, gastric catarrh, weakness of the eyes, conjunctivitis, and cutaneous eruptions. A condition of general debility, or even paralysis, anæmia, and nervous weakness, resembling neurasthenia, can sometimes be traced to wall-papers containing arsenic.

After acute poisoning by the ingestion of large doses of arsenic, when the gastro-intestinal symptoms are passing off, those pertaining to the nervous system appear. There is first pain in the back and limbs, accompanied with numbness or a sleepy sensation. With this, a weakness which increases progressively till there is total paralysis. These phenomena occur within a week or two after the poisoning. Sensation may be almost lost, or scarcely impaired.

Atrophy of the affected muscles follows, and the reaction of degeneration is found. The patellar tendon reflex has been found absent, the plantar cutaneous reflex absent, while the cremaster reflex was normal.

The paralysis begins in the legs, and is most severe in them; may also affect the arms, usually in a less degree.

Cutaneous trophic and vaso-motor changes are not infrequent. In some cases albumen has been found in the urine.

Generally, improvement begins within a month, and steadily progresses to perfect or nearly perfect recovery; a slight weakness may remain a year after the poisoning. In a few cases the paralysis may be permanent.

Seeligmüller gives several diagnostic points: 1. The acute origin of the paralysis as contrasted with the chronic nature of lead paralysis. 2. The severe sensory disturbance. 3. Arsenic affects primarily, and most frequently, the legs. 4. Atrophy and reaction of degeneration appear earlier in arsenical poisoning. 5. Other trophic changes are not seen in lead.

The evidence is sufficient to prove that the symptoms are due to a diffused myelitis affecting especially the anterior gray substance.

TREATMENT.—It does not seem to be certainly ascertained that any special medication hastens the elimination of arsenic. As it leaves the system chiefly through the kidneys, their action should be maintained, as well as that of the skin, by baths and proper clothing.

Morphia, or other anodynes, may be necessary on account of the pain.

Electricity and massage must be used to restore the use of the muscles, as in other affections with similar loss of power.

ALCOHOL.

The symptoms of acute alcoholism, as seen in simple drunkenness, need not be described here. Those of chronic poisoning vary considerably. The more common effects are a gradually undermining of the health and a change in important viscera, as liver and kidney, giving rise to diseases of those organs.

The most common nervous disturbance is found in that condition known as delirium tremens. This is the final result of a long debauch. Patients vary greatly as to the ease with which they have an attack.

It is the common belief that the sudden leaving off of drinking is the cause of delirium tremens. Sometimes this is so, but many times the patient gives up the liquor because he can not take it; the system will not longer tolerate the poison, and then the cessation

from drinking is the first symptom of the disease, not the cause of its outbreak.

Besides the influence of alcohol, it will be found that there has been abstinence from food and loss of sleep, aiding to produce the final outbreak of delirium. In severe cases, it will be found that patients have not slept for a week or more, and have eaten nothing for several days.

Preceding the attack there is a tremor of the hands, tongue, and sometimes a general tremor whenever the limbs are moved. Vomiting may appear a day or two before the delirium. At first there is only an inclination to start suddenly; there is a watchfulness in the patient's expression, and he is evidently divided in his attention to the physician and his unexpressed fears. He fingers the bedclothes with trembling hands. Perhaps he is covered with a cold perspiration. The eyes are red and watery; he has a haggard expression. After a while the delirium may become more active; various hideous and repulsive shapes present themselves to the disordered brain. The patient starts up, and struggles violently to escape from his tormentors. Hallucinations of hearing are much less common than those of sight.

It is very seldom that patients ever attempt to injure themselves or others. If attacks are made upon attendants, it is in connection with some hallucination, or in an endeavor to escape from imaginary specters. So, also, efforts to jump out of windows, etc., are made for the purpose of escape.

The temperature varies; it is often normal, is sometimes subnormal, and may be elevated. It is almost impossible to take the temperature in an excited patient.

The pulse is rapid and weak where there is much excitement, or it may be only slightly increased in frequency when the excitement is moderate; it ranges from 80 to 150, or even more.

Disgust for food, and vomiting of everything taken into the stomach, are quite common.

If the patient does not obtain sleep, the tremor, delirium, feebleness of the pulse, and general prostration increase until death. Near the close of life the temperature may rise to 106° or 107°.

A fatal termination is rare if the patient is seen early and properly treated, unless there is organic disease of one of the important organs.

Instead of delirium tremens, or after an attack, the victims of alcoholism may have serious cerebral symptoms, resembling meningitis, or there may be organic disease of the brain; sometimes there is insanity, usually mania, often general paralysis. The prognosis in such cases is unfavorable.

TREATMENT.—A quiet room is desirable, with as little disturbance from attendants as may be. It is better in some cases to have the room darkened; but many times the uncertain, fitful shadows in a dimly lighted room disturb the patient more than a bright light. His imagination transforms the shadows into grotesque or horrible shapes.

Some authors object to mechanical restraint, advising that there should be attendants enough to keep the patient quiet and prevent his injuring himself or others. Those who need restraint are excited by the opposition of other men; they will struggle against those who, they imagine, are about to injure them. If put into a strait-jacket, their struggles soon cease, or are much less violent, and there is no danger of injury from too great force applied by injudicious, tired, or excited (not angry) attendants. Of course, a patient in a strait-jacket needs to be watched lest he should work himself into a dangerous position, or roll out of bed, or otherwise harm himself. If there is vomiting, tincture of capsicum should form a part of every prescription; sometimes it is sufficient alone to produce sleep, and enable the stomach to retain food.

If the pulse is very rapid and weak, if there is much excitement and tremor, that is, in the worst cases, tincture of digitalis, with or without capsicum, is the best drug. According to the pulse, from one to four drachms should be given. If the pulse is strong and only moderately rapid, one drachm, or perhaps half a drachm, will be sufficient. In very severe cases the larger doses are needed. One dose, sufficient to strengthen the pulse, is better than to repeat small doses. Sleep may follow in half an hour. The digitalis, not in the largest doses, may be repeated after twelve or twenty-four hours if it is necessary to procure sleep again. Often only one dose is required. It must be kept in mind that the patient receives considerable alcohol with large doses of the tincture.

Chloral and bromide of potassium are valuable in the milder cases, and in the severer after digitalis has reduced the pulse. One or two large doses, thirty to forty grains of chloral, with as much bromide, is better than repeating small doses frequently. The latter may increase the excitement. If the stomach is very irritable, it is better to give only the capsicum by the mouth; chloral, if necessary, can be given by enema.

Paraldehyde, in drachm doses, may be sufficient in very mild cases, or after one or two nights' sleep obtained by other means. I have not used it in severer cases.

Formerly opium, or its preparations, were generally used. In some cases the hypodermic injection of a quarter of a grain of morphia is the best treatment.

After the patient has become quiet, the oxide of zinc, two or three grains, or sulphate of quinine, three to five grains, given three times a day, will prove most useful tonics.

It is important that the patient should be well fed; at first milk, hot or cold, as best suits the patient, or animal broths, are best; when the stomach will digest solid food, it should be given.

HYDROPHOBIA.

Hydrophobia is always caused by the bite or the inoculation with the saliva of a rabid animal. It never arises spontaneously. Domestic animals, dogs, cats, cows, etc., and wild animals, as foxes or wolves, may communicate the disease.

About half those bitten are attacked with rabies; bites on unprotected parts, hands, face, and neck, are most dangerous. Early cauterization of the wound diminishes the danger. Slight wounds, like scratches, are more likely to give trouble than severe bites which bleed freely.

There is a stage of incubation, continuing from two weeks to five years (Colin), during which the wound heals, the patient appears in usual health, though often he has a serious apprehension, a dread of the consequences, which he can not explain.

Often the first symptom of the approaching attack is a pain shooting from the seat of the wound toward the nerve-centers; the cicatrix may become livid. The patient's disposition may show a change—he may become moody or irritable. With or without the above symptoms there arises a peculiar difficulty in swallowing. All the motions can be made, but, so soon as the liquid or food touches the mucous membrane of the mouth, the irregular or spasmodic action of the throat interferes with deglutition. Generally, speech is also hindered.

At first the patient can overcome this irregular action of the muscles, but soon he loses that power, and the reflex excitability becomes so excessive that any noise suggesting food or drink excites the spasm. Other regions also acquire this abnormal excitability, so that a slight draught of air over the face, the glitter of a bright light on the eye, or a sharp sound heard, has the same effect.

The patient is unable to swallow his saliva, which

becomes thick, tenacious, and ropy. The irritation thus produced gives rise to violent paroxysms; the patient starts up, hurling violently back his attendants, clutches at his throat, tries to clear his mouth of the mucus, and spits it far from him with extreme violence. His whole effort seems to be to expel the saliva which so distresses him and interferes with reflex respiration.

Delirium, hallucinations, and delusions are sometimes noticeable during these paroxysms, and occasionally in the intervals.

After such an attack the patient lies quiet, or may sit up at his ease; can usually walk if desirable. He knows when the attack is about to be repeated, and may warn his attendants. There is almost never any effort to bite or scratch. The stories of patients barking like dogs, etc., are in a great degree the products of the imagination of attendants.

Finally, these paroxysms become more and more frequent, the patient is exhausted by his inability to take food, by sleeplessness, and by the violence of the disease. Partial paralysis may occur. Death takes place either during an attack or quietly in the interval; the power to swallow may return just before death.

The imagination, or, perhaps, it would be more correct to say the emotional conditions and the mental influences, seems to exert a great influence over the attacks. The sight of water, even a reference to drinking, will cause one; yet the same patient may immediately after urinate, both seeing the urine and hearing it fall into the vessel without any disturbance.

Diagnosis and Prognosis. — The only affection likely to be confounded with hydrophobia is hysteria, which in some of its manifestations slightly resembles the more serious disorder. A careful observation of the patient's condition during the spasm, and in the interval, will guide to a diagnosis, as will also the experiment of gently fanning his face, or the manner in which he acts when drink is offered, bracing himself for a

great effort, seizing the cup, unable to carry it to his mouth, or only succeeding in swallowing a few drops; the character of the attack is very different from hysteria.

There is no resemblance to tetanus, as has been sometimes claimed.

Hitherto death has been the uniform termination, except in a few cases, and in many of these the diagnosis is doubtful.

PATHOLOGICAL ANATOMY.—The blood-vessels of the brain and spinal cord are distended, their walls are thickened, and in many places collections of leucocytes surround the smaller vessels, and they may be found quite generally scattered through the gray substance. Sometimes the blood escapes from the vessels, forming small hæmorrhages. Ross has found the nerve-cells of the median and central groups in the anterior cornua shrunken and atrophied; the spinal accessory and pneumogastric nuclei were also altered.

TREATMENT.—As soon as one has been bitten by a dog, whether thought to be rabid or not, the wound should be sucked, either by the person himself or by some one else. If the mucous membrane of the mouth is unbroken, there is no danger in doing this. As soon as possible after this the wound should be thoroughly cauterized. Some advise nitrate of silver, others caustic potash; the latter is probably the better. The hot iron may answer the same purpose, but is less certain in its effect, as it can not so surely penetrate to every part of the cavity. Gunpowder may be poured into or upon the wound and ignited. Or the wound may be excised.

When the first symptoms of the disease have shown themselves, there is little chance of recovery. Chloral, morphia, and stimulants given by the rectum may relieve the suffering somewhat. Ether can be inhaled to give relief, and while the patient is under its influence a tube may be passed into the stomach and food intro-

duced. Curare and morphia can be injected subcutaneously. Ruxton gave six drops of tincture of cannabis Indica to a boy about six years old each time he awoke. The boy recovered. Broadbent reports a cure from enemata of twenty grains of chloral, one ounce of brandy, and two ounces of beef-jelly every three hours.

CHAPTER XXXVI.

SYPHILIS.

ZAMBACO, D. A., Des affections nerveuses syphilitiques. Paris, 1862.—HEUBNER, Die luetische Erkrankung der Hirnarterien. Leipzig, 1874.—FOURNIER, A., La syphilis du cerveau. Paris, 1879.—SAVARD, P., Étude sur les myélites syphilitiques. Paris, 1881.—WOOD, H. C. *Am. Jour. Med. Sci.*, Oct., 1880, p. 384; *Boston Med. and Surg. Jour.*, Dec. 20, 1883, Jan. 10, Feb. 28, 1884.—LANCEREAUX, E., Syphilis cérébral. *Gaz. hebd.*, 1882.—PUTZEL, L., Syphilis of the Central Nervous System. *Med. Record*, April 26, 1884, p. 450.—SEGUIN, E. C., The American Method of giving Potassium Iodide, etc. *Archives of Med.*, Oct., 1884.

Syphilis of the nervous system belongs to the tertiary period. The first symptoms may appear as early as two months after infection, or as late as thirty years; generally between three and twenty years after.

The nervous symptoms are much more frequently seen after a very mild primary attack, and in a large proportion of cases there have been no secondary symptoms. So slight has been the earlier manifestations of the disease that the patient frequently does not know he has had it.

SYPHILIS OF THE BRAIN.

PATHOLOGICAL ANATOMY.—The changes in the brain are similar to those found elsewhere. The membranes and vessels are most frequently affected. Gummatous tumors form in the membranes, varying in size from small grains to the size of a hen's egg; they are most frequent in the pia mater. They are found on the convexity, and especially at the base near the sella turcica.

Instead of distinct tumors, there may be a diffusion of the new growth over the surface of the membranes closely resembling pus, and this may contain small gummatous tumors scattered throughout its extent.

The subjacent cerebral substance is necessarily affected, partly by spread of the growth destroying the nerve-elements, in part by closure of blood-vessels, interfering with nutrition.

The syphilitic new growths may undergo degeneration, and their interior become fatty or caseous. They may excite inflammation in their vicinity, acting as any other new growth in this respect.

The arteries are also the seat of syphilitic changes. These have been described at length by Heubner. The new growth is developed between the elastic lamina of the intima and the endothelium. The lumen of the vessels is more or less encroached upon by semi-lunar segments or zones of the firm, fibrous new formation. Sometimes the artery is entirely closed by this process, or a thrombus may form at the constricted portion, and thus finally effect its closure.

The brain-substance may be primarily affected; but such cases are rather rare.

An important peculiarity of syphilitic lesions of the brain and its membranes is that they are very often multiple; even distant regions, opposite sides, may be simultaneously affected. They may be present an indeterminate period, and attain considerable size, without giving rise to any symptoms.

Secondary changes, softenings, and inflammations are found in connection with these morbid products, the same as with those of a different nature.

Symptoms.—Syphilis of the nerve-centers gives rise to no special or peculiar symptoms differing from those caused by other lesions of those parts. The diagnosis must be made rather from the grouping of the symptoms or other peculiarities in the mode in which they show themselves. As in other diseases, the symptoms

may be divided into those which are due directly to the lesion and those which are dependent upon secondary changes, which are the same as when these changes are due to other causes.

Headache is the most common and the earliest symptom, often the only one. The pain is severe, obstinate, frequently nocturnal; it occurs in paroxysms, though there may not be entire relief between the attacks. The scalp or parts of the face may be tender, and pressure upon those points may aggravate the pain. The severity of the pain is sometimes excessive—agonizing. Relief is obtained generally only from specific treatment; or, if it seems to follow other means, the pain soon returns. Sometimes external periostitis will cause a swelling over the cranial bones, which will settle the diagnosis.

Pain may also be felt in other parts of the body, in the limbs, resembling closely neuralgia from other causes.

With the pain, or independently, the patient may have a sense of pressure in the head, dizziness or vertigo, ephemeral or fugitive attacks of loss of memory, dimness of sight, numbness in the extremities, or slight impairment of motor power or of speech. These symptoms may be so insignificant that they are ignored until questions recall them to the patient's mind.

The headache, with or without the above symptoms, is of inestimable value as indicating commencing cerebral mischief at a stage when treatment can be of use. Though a similar train of phenomena may occur in cases of tumor of the brain, in uræmia, and in commencing meningitis, the possibility of syphilis should always be kept in mind, even when patients deny the primary or secondary symptoms.

The motor phenomena, *paralysis* and *spasm*, are characterized by the irregularity of the symptoms, the limitation of the paralysis or spasm to a few muscles, and their ephemeral or fugitive character.

Hemiplegia, differing in nothing from that caused by cerebral hæmorrhage, may occur without special warning in syphilis; but generally the attack is less sudden, the paralysis creeping, as it were, from one set of muscles to another, the patient not losing consciousness; the loss of power frequently does not affect the whole side; is limited to the arm or face, or spares the leg. Sensibility is rarely affected.

Attacks of paralysis of a few muscles or sets of muscles may appear and disappear several times before there is permanent loss of power; these attacks may persist a few hours or days, and be repeated at near or distant intervals.

Spasm, local or general, sometimes precedes the paralysis.

When the prodromic cephalalgia, and other slight cerebral disturbances, mentioned above, have preceded these paralytic symptoms, there is great reason to suspect syphilis, and the proper treatment should be followed.

When individual cerebral nerves are affected, as well as the limbs, if the symptoms are irregular, can not be accounted for by one lesion, the alternate paralysis being such as to show that there must be two or more centers of disease, the probability of syphilis is greatly increased.

Among the cranial nerves most likely to be paralyzed may be placed, first, the third nerve, the motor oculi; the sixth, the abducens, is next; the seventh, twelfth, the second, eighth, and fifth follow next in frequency.

Paralysis of the third nerve from syphilis is often partial; it may be combined with paralysis of the sixth or not; whether alone or in connection with disturbance of other cranial nerves, there is reason in such cases to suspect syphilis, and careful inquiry for prodromic symptoms may add to this probability.

It is unnecessary to describe minutely all the vari-

ous combinations which are frequently found in such cases.

Spasm or convulsion limited to a few muscles, as the facial, or those of one arm, of the hand—monospasm, as it is called—is an indication of irritation of the motor centers in the cortex of the brain. Very often the lesion in these cases is syphilitic. The convulsion may extend to the whole of one side, or it may begin in one region and extend to the opposite side, becoming general. Many times consciousness is preserved, or it may be only partially impaired. Sometimes severe pain in the head or in the affected limb may attend the spasm.

Convulsions, differing in no respect from true epilepsy, may be due to syphilis. If the patient is somewhat advanced in age before the attacks commence, their syphilitic origin is the more probable. If mental impairment appears early, and motor weakness follows the attacks, limited to one side or one limb, and persisting between the attacks, the probability of syphilis is the greater.

Sometimes the mental phenomena are most prominent; intellectual weakness, loss of memory, inability for consecutive thought, delirium, perhaps mania, may be present. Sometimes the mental disturbance will closely resemble general paralysis.

While the above symptoms, or groups of symptoms, individually only give rise to a suspicion of syphilis, the combination of two or more of the groups increases the certainty of the diagnosis.

The course of cerebral syphilis, if not properly treated, is steadily downward. The symptoms increase in severity, the spasms become more frequent, the paralysis extends, and the mental powers are gradually lost. Within a comparatively short time a fatal termination closes the scene.

SYPHILIS OF THE SPINAL CORD.

The membranes are most frequently the seat of the morbid changes, the cord being affected secondarily. Sometimes the medullary substance itself is attacked, the lesion being generally diffused throughout the part of the cord which is the seat of the disease. Gummatous tumors are also found. The bones are less frequently diseased. The lumbar enlargement is more generally attacked than the higher parts.

The symptoms are very similar to those produced by other lesions. When meningitis is present there are pains in the back and limbs, perhaps with spasmodic action or contraction. The symptoms are variable; there may be remissions or intermissions, which are not seen in simple meningitis.

When the cord itself is affected there is more likely to be irregularity in the course of the disease than in simple myelitis. Locomotor ataxia may be very closely simulated. The earlier symptoms may relate to the genito-urinary organs. Impotence and inability to micturate may precede the paralysis. When the latter appears, it may begin in one leg, and appear in the other later.

Sensation may be unaffected until the paralysis has become well marked; it may be only slightly disturbed during the whole course of the disease. In other cases both sensation and motion may be lost early. Sometimes the paralysis pursues an acute course very similar to the severest form of ordinary acute myelitis.

Often the course of spinal syphilis is chronic; the symptoms develop slowly; there may be periods of remission, or at least no advance is made.

The prognosis is unfavorable for complete recovery; if energetic treatment is followed, the symptoms may recede and a certain amount of benefit be received; the patient improves, but very often the cord is too seriously injured for its functions to be completely restored.

SYPHILIS OF THE NERVES.

The cranial nerves are the most frequently affected by syphilis; these have been referred to already. Any of the other nerves may be implicated in syphilitic growths. It is probable that the nerves may be directly diseased; many cases of neuralgia in syphilitic patients are probably thus caused. The symptoms seem to be very similar to those of neuritis. This department of the subject needs further investigation.

Hereditary syphilis may attack the nerve-centers; cerebral symptoms are not uncommon; the spinal cord has been the seat of syphilitic changes in very young children.

TREATMENT OF SYPHILIS OF THE NERVOUS SYSTEM.

There should be no delay in beginning an anti-syphilitic treatment. The more serious the symptoms, the more energetic this should be. As the brain and spinal cord are all-important for life, and as slight lesions produce serious permanent disability, it is necessary to begin at once with large doses of iodide of potassium, and to use mercury freely.

If the case is very urgent, from forty to sixty grains can be given at once three times a day; if less haste is needed, a dose of ten to twenty grains may be given three times the first day; the doses may be increased by ten or twenty grains each day, or every other day, until some result is obtained.

Large doses of forty-five to sixty grains are sometimes borne better by patients than small doses of four or five grains.

The dose should be increased until the disease is checked, or there is evident intolerance. What is the limit? Many patients improve when taking one hundred and twenty grains a day. I have repeatedly given two hundred and fifty grains daily, and have even gone

as high as nine hundred grains a day with no disadvantage to the patient.

The drug should be given in a *large amount* of water; slightly alkaline water is preferable. It is generally better to give it before meals, though with some patients it is better after meals, and some prefer to divide the large doses, taking part before and part after meals.

If the bowels become too loose from the iodide, the dose may be slightly reduced, more diluted, and then increased more slowly; or, if there is need for immediate effect, a few drops of laudanum can be added to each dose.

Vomiting may seriously interfere with the administration of the drug. If it does, it will be necessary to omit it for a while, then begin in smaller doses and increase more slowly. Or the plan of giving a large dose at once might be tried.

Conjunctivitis, coryza, and glossitis are rare in syphilitic patients as effects of the iodide.

Acne may be met with Fowler's solution, but is not a contra-indication to the use of the medicine.

Mercury may be given by inunction, half a drachm or two drachms being rubbed in daily. Internally, the biniodide or bichloride is preferable, and should be given in doses of from $\frac{1}{10}$ to $\frac{1}{4}$ of a grain three times a day. Many authorities prefer the inunction.

The iodide of potassium and mercury should not be stopped too soon. It is necessary to continue the treatment several weeks after the patient seems cured. The largest dose reached need not be continued.

Of other treatment, tonics, especially iron and cod-liver oil, are generally indicated. The strength should be maintained by a generous diet.

Sequelæ and complications should be treated according to the principles given elsewhere.

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