died, this being 13.7 per cent. of all deaths from diphtheritic paralysis in his series.

Cardiac paralysis is even more fatal. Statistics are of little value, as different observers vary so greatly in the criteria upon which they base their diagnoses. All 32 cases collected by the American Pediatric Society[7] were fatal. Hibbard[11] reports 47 per cent. of deaths in cases with irregular pulse. Vomiting during convalescence from diphtheria generally means heart failure, is an unfavorable sign, and occurred in over half of Hibbard's fatal cases. According to Burrows[12] it is especially to be feared when it occurs in a patient whose heart is irregular, or who presents other evidences of nerve degeneration. A very slow pulse is also unfavorable, especially in children. The patients may die within twenty-four hours of the onset of cardiac symptoms, or later during an exacerbation. Sudden death from heart failure without previous symptoms may occur in the acute stage, or it may suddenly and unexpectedly terminate a case after convalescence has appeared to be completed.

Treatment.—The varying results of statistical studies as to the effect of antitoxin have already been mentioned in connection with the subject of etiology. It seems probable that the early and vigorous use of antitoxin in any given case will decrease the likelihood that this complication will occur, but only in so far as it lessens the severity and duration of the causative disease. That this view is correct is proven on the experimental side by the work of Ransom,[1] who shows that doses of the toxin capable of producing paralysis in animals are neutralized in this respect by antitoxin injected simultaneously and modified, though not prevented, by large enough doses given from fifteen to twenty-two hours after those of the toxin. On the clinical side the report of the London Clinical Society[21] shows that the frequency of paralysis as a complication of diphtheria is less when antitoxin is used on the first two days of the disease than when its use is delayed. After the injury to the nerves or muscles has been done, it is not probable that antitoxin will have any effect in restoring them to the normal condition. The same conclusions probably hold in regard to the heart manifestations. In other words, antitoxin, given early and in large doses, has some value as a prophylactic in preventing the paralytic complications of diphtheria.

On the peripheral neuritis no form of treatment has much effect. The case should be managed like a neuritis from any other cause. Rest, careful nursing, liberal diet, tonics, massage, and electricity are of some value. Strychnine is much used, but its influence in restoring the degenerated nerves and muscles to their normal condition is at least questionable. In palatal, and especially in pharyngeal and laryngeal cases, great care should be taken in feeding the patient to prevent the entrance of food into the larynx. The œsophageal or nasal tube may be used if needed, great care being taken to avoid introducing it into the larynx and to get the end well below the glottis. In some cases it will be better to feed by the rectum. Forced feeding had better not be delayed in hope that the child will begin to eat, especially in cases in which the patient is much debilitated by a severe attack of diphtheria. Proper and sufficient nourishment is of importance both in the treatment of the general depressed condition and in that of the paralysis.

One precaution should be insisted upon. Every case of diphtheria should be watched closely for the occurrence of cardiac or respiratory symptoms, especially when evidences of palatal paralysis are present. Thomas and Hibbard[10] advise that every case of diphtheria, however mild, should be kept in bed till the throat is clear, and, if there has been any prostration, for at least two weeks more. After four weeks with no heart symptoms there is little danger. If heart symptoms arise, the greatest care must be taken to keep the patient quiet, morphine being used for this purpose if needed. Careful feeding is of importance. Medicines are of value only in meeting special symptoms. Alcohol, digitalis, and strychnine may be of service. In the respiratory cases strychnine is the most valuable remedy. Electricity may also be used. *Ralph C. Larrabee.*

References.

1 Ransom: Journal of Pathology and Bacteriology, 1900, vol. vi., p. 435.
2 Welch and Flexner: Bulletin of the Johns Hopkins Hospital, 1891, vol. ii.
3 Gowers: Diseases of the Nervous System, second edition, vol. ii., p. 903.
4 Thomas: Boston City Hospital Medical and Surgical Reports, ninth series, 1898, p. 52.
5 Baginsky: Nothnagel's Specielle Pathologie und Therapie, Bd. ii.
6 Councilman, Mallory, and Pearce: "Diphtheria," 1901.
7 American Pediatric Society, Collective Investigation: Transactions, 1896.
8 Goodall: Brain, 1895.
9 Northrup: Nothnagel's System of Practical Medicine, American edition.
10 Thomas and Hibbard: Boston City Hospital Medical and Surgical Reports, eleventh series, 1900.
11 Hibbard: Boston City Hospital Medical and Surgical Reports, ninth series, 1898.
12 Burrows: American Journal of the Medical Sciences, February, 1901.
13 Henoch: Lectures on Children's Diseases, vol. ii., 1889, p. 300.
14 Ross: Medical Chronicle, 1890.
15 Woodhead: British Medical Journal, September 3d, 1898.
16 Trevelyan: Lancet, 1900, ii., p. 1482.
17 Meyers: Lancet, 1900, ii., p. 889.
18 Hesse: Jahrbuch für Kinderheilkunde, 1893, Bd. xxxvi., S. 19.
19 Brodie: British Medical Journal, November 4th, 1899.
20 Biernacki: British Medical Journal, December 30th, 1899.
21 London Clinical Society: Report on Antitoxin, 1898.

(For further bibliography consult the articles of Northrup, Ross, and Thomas.)

PARALYSIS, FACIAL. See *Facial Paralysis.*

PARAMUCIN.—Mitjukoff has obtained from the mucoid contents of ovarian cysts a mucin-like substance, which differs from *pseudomucin* and *mucin*, chiefly in the fact that without previous boiling with dilute mineral acids it will reduce an alkaline solution of copper. ("Ueber das Paramucin," *Arch. f. Gyn.*, Bd. 49, 1895.)
Aldred Scott Warthin.

PARAMYOCLONUS MULTIPLEX.—(Synonyms: Myoclonus multiplex, Myoclonia, Polyclonia.) Originally described by Friedreich[1] in 1881, this disorder has been recognized by clinicians in Germany, France, Italy, England, and America. Its existence as a disease has been questioned, some asserting it to be a variety of hysteria, others of chorea. Still others cut the Gordian knot by claiming that both a true form and a hysterical form of the disease exist. However, the preponderance of authority as well as of evidence at the present day appears to favor its validity as a clinical entity.

It is a rare disease. Gowers[2] states that he was able to collect but fifty-two cases in the literature up to July, 1895, of which only thirteen were considered by him true examples.

One of the best accounts of the disease accessible to American readers is that of Starr,[3] which is also accompanied by a bibliography.

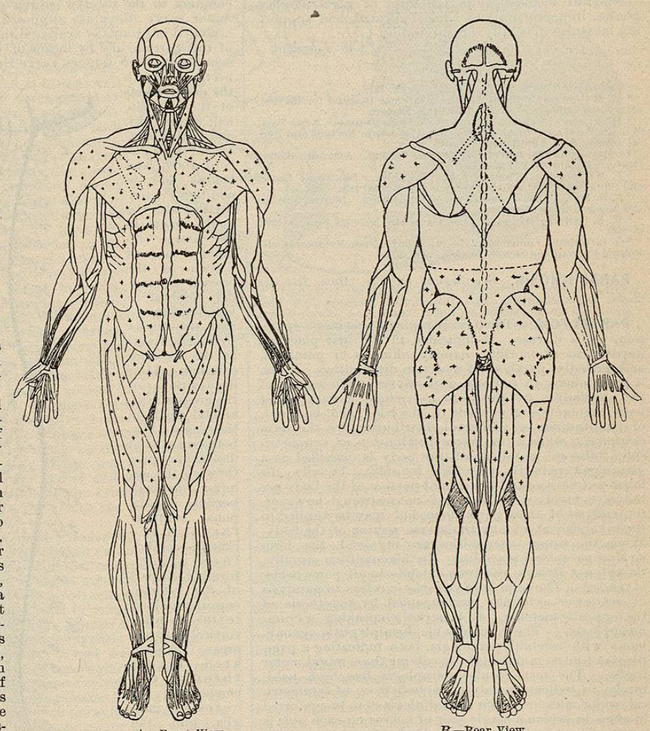
The disease is characterized by paroxysms of clonic muscular contractions, shock-like, bilateral, and symmetrical as regards the two sides of the body. The individual contractions are frequent, varying in rate from thirty to one hundred or more per minute. The duration of the paroxysms may vary between five or ten minutes and some hours. Likewise the frequency of paroxysms in a day may vary from one or two to twenty or more. The muscles affected in typical cases are the intrinsic truncal muscles and those connecting the trunk and extremities; those moving the face, hands, and feet being seldom, if ever, affected (see Fig. 3738). In one case observed by the writer,[5] the diaphragm and laryngeal muscles were involved at times, causing short, sharp, involuntary exclamatory sounds.

Negative characteristics are: consciousness is not affected; mental defects are absent as a rule, but, if present, are transient; voluntary movements of the face, hands, and feet are not abolished, even during a paroxysm, though the muscular power is much reduced; electrical changes of degenerative significance are absent, as is fibrillary twitching. Sensation is not diminished or lost, but a decided hypersensibility to sound and touch has been observed by the writer in one case.[5] A profound sense of fatigue was also noted in that case. The convulsive movements cease during sleep, and are brought on, or, if present, increased in violence, by emotional excitement, by irritation of the skin, by cold, etc., and by manipulative procedures generally.

Oppenheim[4] recognized a hysterical type as distinguished from the true form, but admits the difficulty of separation.

Unverricht, Weiss, Kreiver, and Sepilli (quoted by Oppenheim) describe cases of a familial type and associated with epilepsy (*loc. cit.*).

Causation. — The patients are usually neurasthenic. A majority of cases reported were of the male sex. The ages of patients have varied between thirteen and forty-eight. Mental worry, fright, injury, and physical strain are accredited causes. Of two cases reported by the writer one was attributable to mental worry combined with la grippe. The other was distinctly due to fright. A third case, observed by the writer through the kindness of Sir William Gowers, followed a fall from a considerable height without palpable injury. Fry's case[6] was due to overexertion, Starr's[3] to strain in lifting. Removal of the thyroid in dogs is said sometimes to cause symptoms of this disease.



A.—Front View. *B.*—Rear View.

Fig. 3738.—*A* and *B*, Paramyoclonus Multiplex. Distribution of myoclonic spasm indicated by plus marks.

The pathology is unknown. Autopsy in one case (Schultze) revealed no nervous changes. Friedreich, who was the first to describe the disease, believed it to be based on overexcitability of the spinal motor elements. Some have surmised that the cause of the faulty action lies in the muscles themselves or in an abnormal state of the nerve endings. To the writer its psychic antecedents, marked hemiplegic preponderance at times (in one case); its aggravation by mental and emotional states; the marked, though transient, mental changes in two cases; and the heightened muscle reflexes, would all suggest that the disease is to be viewed as the visible expression of a state of "*inhibitory insufficiency*," probably cortical in seat.

Diagnosis.—This is to be based on "the sudden shock-like character of the muscular contractions, their bilateral symmetry, and the comparative freedom of the extremities." (Gowers.[1]) This view is also concurred in by Walton[8] in a recent paper on the myospasms in general. Hysteria is ruled out by the absence of the stigmata and of the characteristic emotional state. There appears to be no tendency to simulation or desire for sympathy in paramyoclonus. Chorea is excluded by the non-involvement of the face and hands. Dubini's "electrical chorea," a disease endemic in a certain locality in Italy, is to be separated by its unilateral beginning, nerve and muscle degeneration, and a fatal termination in a few months.

The prognosis is variable, according to different authors. Friedreich reported that some of his patients recovered. Oppenheim considers the prognosis grave. In all the American cases reported to date the patients have recovered. Relapse may occur, but does not preclude ultimate recovery.

The duration may vary from three or four months to a year or more. In one of the writer's cases the convulsions ceased on the one hundred and second day, but recurred in twenty-four hours, to disappear again in eight days. There has been no recurrence to date (four months). In the case reported by Starr (*loc. cit.*) the patient recovered in about a year, as did also the patient in the first case reported by the writer.[5]

Treatment.—The drug treatment followed has been

so diverse as to suggest that it has had little to do with the recovery. Chloral, bromides, hyoscine and other sedatives, arsenic, quinine, thyroids, and galvanism have all been followed by improvement and recovery. The factors of rest, feeding, and time would appear to be the important ones. Sedatives may be used to mitigate the severity of the spasms and promote the patient's comfort.

Nutrient medication, in the form of glycerinophosphates, iron preparations when indicated, and supporting measures generally, are advisable.

F. W. Langdon.

REFERENCES.

1 Friedreich: Virchow's Archiv, Bd. lxxvi., 421.
2 W. R. Gowers: Clinical Lecture; National Hospital for the Paralyzed and Epileptic, London, 1895 (unpublished).
3 M. A. Starr: Familiar Forms of Nervous Disease. New York, William Wood and Company, 1891, 244; also Journ. Nervous and Mental Disease, 1887, 416 (the same case).
4 Oppenheim: Diseases of the Nervous System. American edition (Mayer). Lippincott, Philadelphia, 1900.
5 F. W. Langdon: Paramyoclonus Multiplex. Cincinnati Lancet and Clinic, 1896, 145; Journal Nervous and Mental Dis., 1902, 541.
6 F. R. Fry: A Case of Paramyoclonus Multiplex. Journal Nervous and Mental Disease, 1888, 397.
7 W. R. Gowers: Manual of Diseases of the Nervous System, 1899, ii., 630.
8 G. L. Walton: Contribution to the Study of the Myospasms, etc. Journal Nervous and Mental Disease, 1902, 403.

PARAPHIMOSIS. See *Sexual Organs, Male, Diseases of.*

PARAPLEGIA.—The term paraplegia specifies a symptom, not a disease. It indicates, in the first place, an impairment of motility, namely, akinesis or paralysis, and secondly, paralysis of a certain distribution. Thus, it is customary to indicate a paralysis involving one of the extremities by the term *monoplegia*, a paralysis of both extremities on one side of the body, that is to say, of longitudinal or unilateral distribution, by the term *hemiplegia*, while a transverse distribution or symmetrically bilateral paralysis of the body is classified as a *paraplegia* (para, Gr. παρα, side by side). Usually, the lower extremities and the caudal portion of the body are the parts involved. The term is not restricted, however, to paralysis of these parts alone, but may be applied to the involvement of any transverse section of the body. When the upper extremities are involved, the term *cervical paraplegia* is applied. In this condition, usually, though not always, paralysis extends to all parts below —caudad. The term *hemi-paraplegia* refers to paralysis of one lower extremity, accompanied by anæsthesia of the opposite member. It is, strictly speaking, a crural monoplegia. The term double hemiplegia is synonymous with cerebral paraplegia, both indicating a paraplegia of intracranial origin, involving the cerebral motor tracts. The term ocular paraplegia has been used, rarely, to indicate paralysis, in both eyes, of symmetrical ocular muscles. The term diplegia is to be preferred in cases in which a single pair of nerves on each side is paralyzed; as facial diplegia, instead of facial paraplegia. Jaccoud introduced the adjective *paraplegiform* to indicate bilateral disturbances of motility other than true paresis or paralysis, but which resemble the latter in disturbance of locomotion; as, for example, that produced by incoordination, and spastic conditions.

In paraplegia (it being defined as paralysis of a certain distribution) we have an important symptom of various pathological conditions, forming by its association with other symptoms some of the most striking clinical pictures of disease. Its proper study involves the consideration of the localization of function, the impairment of which produces paraplegia; the nature of the pathological process causing such impairment; the variations in disturbance of function due to involvement of different segments of the body; the other symptoms which may accompany it; and the diagnostic and prognostic significance of the grouping of such symptoms, together with indications for remedial measures.

From this standpoint paraplegia forms a convenient centre from which may be analyzed a great number of diseases of the nervous system, mostly of spinal origin, but also including some cerebral and peripheral nervous affections. As we have a motor impairment to consider, the motor tracts of the nervous system must be called to mind. From their periphery in the motor end-plates they pass through the mixed peripheral nerves, the anterior roots of the spinal cord, the root zones in the anterior columns, to the anterior cornua of the cord, where the motor nerve fibres are supposed to terminate in cells arranged in groups or scattered through the gray matter of this portion, and by means of which they form reflex connections with sensory nerve tracts, commissural connections with the motor tracts of the opposite half of the cord, and with different levels of the cord above and below; and, in addition, connection with the motor tracts in the lateral columns of the cord known as the crossed pyramidal, cerebral, or voluntary tracts, and the direct pyramidal tracts in the anterior columns. After decussation of the crossed pyramidal tracts in the medulla, both crossed and direct tracts pass through the anterior (ventral) portion of the pons, continue through the crura cerebri, then upward, forming a part of the internal capsule, and on to the so-called cortical motor areas. Associated tracts through the cerebellum, the cerebral ganglia, and the nuclei of the cranial motor nerves complete the system. In order to produce paraplegia, not only must some part of this motor system be affected, but the lesion must be symmetrically bilateral, must involve both halves of this duplex system. The general division may be made, therefore, into peripheral, spinal, and intracranial (cerebral) paraplegia. The divergence of the right and left motor tracts in their peripheral and cerebral por-

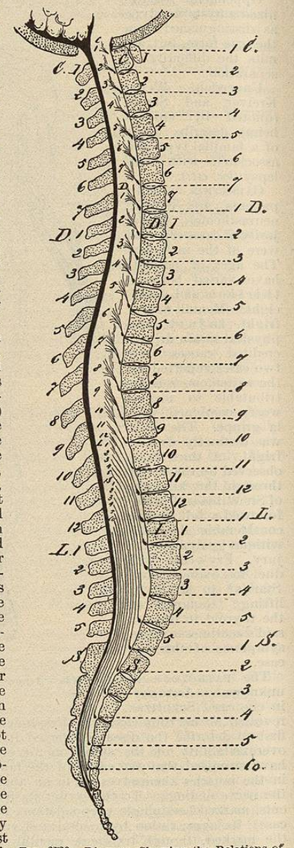


FIG. 3739.—Diagram Showing the Relations of the Spinous Processes to the Bodies of the Vertebræ, and of these to the Points of Origin of the Spinal Nerves. (From Gowers.)

tions, and their approximation in the spinal cord, medulla, and pons, admit of their frequent bilateral involvement from lesions in the latter regions, and but rarely in the first-named divisions. Consequently, the lesion producing paraplegia is usually a spinal-cord lesion. Yet it is possible to have a tumor develop in the longitudinal fissure between the hemispheres, which will involve the motor areas for the legs in each hemisphere, thereby producing a symmetrical and bilateral paralysis of the lower extremities, namely, a cerebral paraplegia. On the other hand, it is now well known that a peripheral paraplegia may be produced by a multiple neuritis involving the peripheral nerves of both lower extremities in such a symmetrical manner as closely to resemble spinal-cord lesions. Compression within the spinal cord of the bundle of peripheral nerves known as the cauda equina may also give rise to paraplegia. In the latter case, and also in multiple neuritis, we have the phenomena which attend irritation or destruction of a mixed nerve, namely, motor, sensory, and trophic disturbances in the parts supplied by the nerves involved. But motor, sensory, and trophic disturbances may also occur when the lesion is in the spinal cord, provided it be extensive enough to involve both motor and sensory tracts, and at a level from which the upper or lower extremities receive their motor innervation, namely, the anterior cornua in the cervical and lumbar enlargements. Lesions between these enlargements, or above the former or below the latter, do not produce the trophic disturbances which result in degeneration of peripheral motor nerves of the extremities, and the consequent atrophy of the muscles which they supply; although voluntary power and sensation may be lost through interruption of the cerebral motor and sensory conducting tracts traversing the section of the cord involved by disease.

Myelitis affecting the entire transverse area of the cord, but limited in its longitudinal extent to some portion between the cervical and lumbar enlargements, commonly known as transverse dorsal myelitis, furnishes an example of this form; while involvement of the lumbar enlargement will serve as a type in which motor degeneration and atrophy are added. Similar results may follow a meningitis, or meningo-myelitis involving the sensory and motor nerve roots or root zones at the level of the lumbar enlargement. Paraplegia, unaccompanied by loss of sensation, may be conceived of in case the meningitic or myelitic process remains limited to the anterior periphery of the cord, or to the anterior horns of the lumbar enlargement, by which the motor tract would be involved and the sensory tracts escape implication.

A similar process affecting the cord at its cervical enlargement alone might produce bilateral paralysis of the upper extremities without involving the lower extremities, as long as the myelitic process did not extend deeply enough to invade the pyramidal tracts in the lateral columns. Should it so extend, however, the lower extremities would exhibit paraplegia without loss of sensation and without muscular atrophy; while with a complete transverse lesion at the cervical enlargement loss of sensation in all parts below the upper extremities would be added, but still without degenerative atrophy in the lower part. The meningitic process might be extensive enough to involve both cervical and lumbar enlargements, affecting chiefly the anterior periphery of the cord, producing a paraplegia involving both upper and lower extremities with muscular atrophy in both, and even a transitory loss of sensation; or the gray matter of the anterior horns may be involved throughout the cord on both sides, as in poliomyelitis anterior, with similar results. Finally, we may have involvement of the cerebral (pyramidal) motor tracts in the lateral columns of the cord at any height, cutting off voluntary innervation to all parts supplied below the lesion, but without producing trophic disturbance.

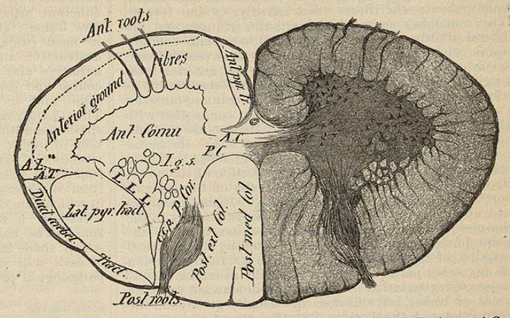


FIG. 3740.—Diagram of a Section of the Spinal Cord in the Cervical Region. *A.C.*, Anterior commissure; *P.C.*, posterior commissure; *I.g.s.*, intermediate gray substance; *P.cor.*, posterior cornu; *c.c.p.*, caput cornu posterioris; *L. L. L.*, lateral limiting layer; *A.-L. A. T.*, antero-lateral ascending tract, which extends along the periphery of the cord. (From Gowers.)

These examples show the necessity of keeping before us a mental picture of the topographical anatomy of the nervous system, and a recollection of the functions of the different tracts to the extent known, in all our attempts to localize a lesion from the symptoms found. The disturbances which accompany the bilateral paralysis constituting paraplegia will vary (1) *according to the level of the lesion* through interference with the visceral, vaso-motor, sensory, and reflex functions of the segment involved; (2) *according to the extent of the lesion transversely in the cord*, through involvement of different functional tracts and centres; and (3) *according to the predominance of an irritative, or of a destructive pathological process* constituting the lesion, producing increase, diminution, or perversion of function. The first and second factors concern the localization of the lesion, the third its nature and course. To aid in the consideration of the former, diagrams and tables are given, and a brief résumé of certain anatomical and physiological data.

The spinal canal is longer than the cord, the latter terminating in man at the upper border of the second lumbar vertebra. The exits of the several spinal nerves do not, therefore, correspond to their levels of origin in the cord, nor do the bodies of the vertebræ correspond to their spines. Gowers' diagram (Fig. 3739), showing the relations of the segments, nerves, and bodies of the vertebræ to the spinous processes, together with the table (Fig. 3741) showing the functions of the different segments, are valuable aids to diagnosis. The cervical enlargement corresponds to the lower five cervical spines; the lumbar enlargement to the tenth, eleventh, and twelfth dorsal, and first lumbar spines.

The ascending tracts of the spinal cord are (1) the antero-lateral ascending tract of Gowers, supposed by him to conduct painful sensations; (2) the direct cerebellar tract, whose function is unknown; and (3) the posterior columns, which conduct tactile and muscular sensations. Fibres serving the latter function, it is thought, occupy part of the median division of the posterior columns (columns of Goll), and do not decussate, while other sensory fibres do. The external columns (columns of Burdach) include the posterior root zones and fibres having a short course up and down the cord, probably decussating at higher levels, or connecting different levels of the cord. The ascending tracts degenerate upward from the