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2 W. R. Gowers: Clinical Lecture; National Hospital for the Paralyzed and Epileptic, London, 1895 (unpublished).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-

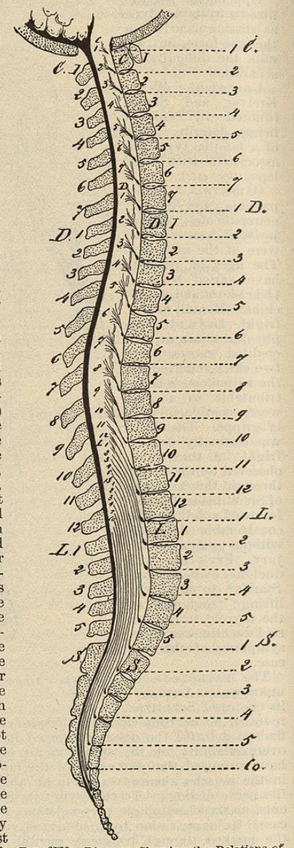

FIG. 3739.—Diagram Showing the Relations of the Spinous Processes to the Bodies of the Vertebræ, and of these to the Points of Origin of the Spinal Nerves. (From Gowers.)

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.

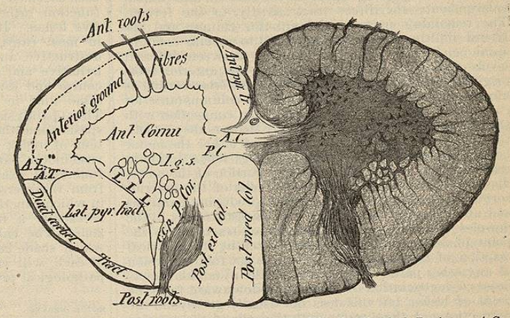

FIG. 3740.—Diagram of a Section of the Spinal Cord in the Cervical Region. *A.C.*, Anterior commissure; *P.C.*, posterior commissure; *I.g.s.*, intermediate gray substance; *P.cor.*, posterior cornu; *c.c.p.*, caput cornu posterioris; *L. L. L.*, lateral limiting layer; *A.-L. A. T.*, antero-lateral 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

level of a destructive lesion. The descending tracts are the direct and crossed pyramidal tracts, concerned in the transmission of cerebral impressions downward to the motor centres in the anterior horns. They diminish in size downward, going chiefly to the cervical and lumbar enlargement; the direct tract mostly to the former. The remaining parts of the anterior column and the lateral limiting tract are supposed to contain commissural conductors of motor impulses between different levels of the cord on the same side. The anterior roots originate in the gray matter of the anterior cornua as already described, traverse the anterior columns (anterior root zones), and go to the muscles. In connection with the sensory roots through the gray matter, they constitute the "reflex arcs," interruption of either the motor or sensory division of which abolishes reflex action in that segment. Destruction of any portion of the motor division from the muscles to the cells of the anterior cornua, including the latter, besides producing paralysis and abolition of reflex action, also causes atrophy in the muscles and the corresponding motor tracts up to the point of lesion, which is characterized by the electrical reaction of degeneration. This does not follow a lesion of any other part. Destruction of the pyramidal tracts causes a degeneration of the column downward from the point of lesion, but this does not usually go beyond the connections of these tracts with the cells of the anterior horns. Atrophy, and the reaction of degeneration are absent. The reflex arc is not only preserved, but reflex excitability is increased. Impairment of voluntary power, and exaggerated motor reflexes frequently amounting to clonic or tonic spasm, characterize lesions of these tracts. Lesions of the cervical region may be accompanied by disturbances of respiration, of the cardiac functions, of the cilio-spinal centre (radiating fibres of iris), and by vaso-motor phenomena, showing either increased or diminished functional activity, as the lesion is irritative or destructive. Lesions in the lower segments of the cord interfere with certain functions of the bladder, the rectum, the sexual, and the vaso-motor apparatus in a similar manner, serving as aids to localization.

As disturbances of the bladder and rectum are usually important features in the paraplegic state, their complex functions should be referred to. Each of these organs has two sets of muscles, which are opposed to each other in action—the detrusors, which expel the excretions, and the sphincters, which oppose expulsion. Besides the local nervous apparatus which are found in the walls of all hollow muscular organs, and which probably constitute a reflex apparatus between the mucous membrane and the subjacent muscular, vascular, and glandular apparatuses, in a manner not fully known, these organs have their opposed muscular movements represented in the cord by motor centres situated somewhere between the origins of the second and fifth sacral nerves, and a reflex arc is established through sensory fibres from their mucous membrane and muscles. In addition to this there are sensory connections (posterior columns) with the brain, and voluntary paths from the brain (pyramidal tracts), by which a certain amount of control is obtained over the sphincters and detrusors, the mechanism of which is but imperfectly understood. The important facts for pathology are that, as in the involvement of other motor organs, lesions above the motor centres in the cord simply cut off the sensory impressions from these organs to the brain when the ascending tracts are destroyed, and interrupt voluntary impressions to them when the pyramidal tracts are destroyed. In the former case, there is no consciousness of the necessity of micturition or evacuation, and therefore no attempt to restrain it. In the latter, there is consciousness of the necessity, but inability to resist the expulsion. When both paths are cut off there is neither desire nor power to expel the excretions—involuntary and unconscious evacuations take place through the reflex mechanism in the cord; but when the lesion destroys the motor centres of the bladder and rectum, expulsive power and the power of the sphincters are lost, and the retention of excretions results, except evacuation due to the mechanical expulsion of liquid fæces and the dribbling of urine.

In most of the examples given to illustrate the question of localization of the lesion, destructive lesions have been assumed—that is to say, abolition or diminution of function, rather than the intensified activity of an irritative lesion. It should be borne in mind, however, that in most cases, before a pathological process becomes destructive, an irritative stage has existed; and that the irritative and destructive stages may so vary in their course and duration that both processes go on at the same time in different areas involved by the lesion. Thus paralysis may be preceded by clonic or tonic spasm; anæsthesia, by pain and by paræsthesia; abolition of reflex action, by an exaltation of the reflexes; vaso-motor paresis and failure in nutritive processes, by functional exaltation. Or, as instanced in paraplegia from transverse myelitis, between the focus of disease, in which function is lost, and the healthy portion, there is usually a region of increased activity—an irritated zone. The balance between these opposite conditions, which shade into each other, and the degree and extent of each, will vary with the nature and rapidity of the pathological process producing it.

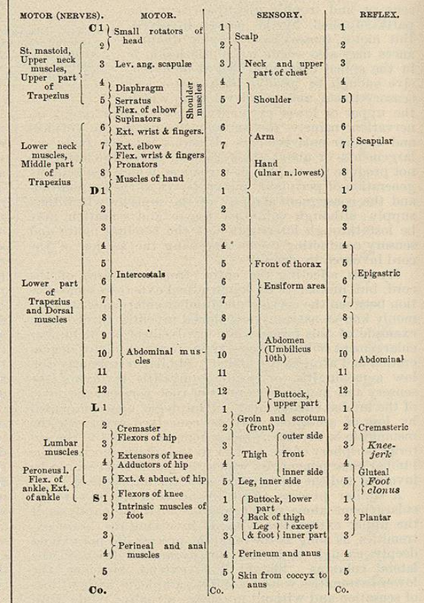

FIG. 3741.—Table Showing the Approximate Relation of the Various Motor, Sensory, and Reflex Functions of the Spinal Cord to the Spinal Nerves.

Concerning the nature of the pathological process, the common division into structural and functional will serve our purpose; meaning, by the latter, that in which no perceptible change exists—abnormal variations in those molecular movements which constitute functional activity, and which are, therefore, dynamic rather than static in character. This class comprises many of the defects due to unstable molecular conditions, partly

inherited, partly acquired, and frequently dependent upon imperfect nutritive processes in the neural tissues, either assimilative or excretory. From this extreme, we have a gradation into coarser forms of molecular derangement, and, finally, into those in which the microscope, or the eye alone, distinguishes derangement in structure. Taking the latter first, as being more tangible, we find that nearly all structural neural disease begins in non-neural tissues. True, we may have direct traumatisms, as cutting, crushing, and compression of neural tissues; and there may be degenerative processes originating primarily in nerve fibres and cells, and direct toxic effects from organic or inorganic poisons conveyed by the nutrient fluids; but there is a larger group of disorders which are secondary to inflammation, acute or chronic, in the connective-tissue structure which forms the framework or support of the neural elements, or to vascular disease, such as hemorrhages, thrombosis, embolism, or proliferative occlusion.

Leaving the consideration of these elementary propositions, we pass on to consider the actual clinical forms of disease which result in producing paraplegia.

Taking the disease which most frequently produces paraplegia, myelitis, as a type, let us study its most common form—namely, transverse dorsal myelitis. This name implies that the entire transverse area of the section is more or less involved in the inflammatory process, and does not include the more limited centres of myelitis, termed *focal* lesions, which may be located in one-half of the cord, and rarely produce paraplegia; though this may result when independent foci involve motor tracts of the anterior horns or root zones, at the same level, or the pyramidal tracts even at different levels. Where several myelitic foci exist, the term *disseminated focal myelitis* is used. While the term "transverse" implies that the longitudinal extent of the lesion is limited, this is not construed in a narrow sense, and may be properly applied when the lesion invades both the lumbar enlargement and the dorsal segments. When the entire length of the cord is involved, the term *diffuse myelitis* is applied; this may be general—*i.e.*, involving both white and gray matter—or may be limited to the latter, when the term *diffuse poliomyelitis* (myelitis of gray matter) is used. The term poliomyelitis anterior indicates that the anterior cornua alone are invaded; while the term *central myelitis*, or *diffuse central myelitis* (according as it is local or general), is applied to poliomyelitis originating about the central canal and invading any portion of the remaining gray matter. Other adjectives are in use—such as *hemorrhagic myelitis*, when secondary to a hemorrhage within the cord; *compression myelitis*, when secondary to compression of the cord, as from tumors, inflammatory exudations, and fractures or dislocations of the vertebræ; *meningomyelitis*, when the inflammatory process invades the membranes and cord. The terms acute, subacute, and chronic prefixed to these various terms, specify whether the onset has been sudden, gradual, or slow.

1. Acute Transverse Dorsal Myelitis (involvement of any segment of the cord between the cervical and lumbar enlargements).

Symptoms.—Paraplegia resulting from this disease is usually characterized by the rapid onset of motor weakness of the lower extremities, preceded by numbness and painful tingling in the extremities; formication and other paræsthesiæ, moderate or severe; pain in the back and limbs, of a boring, tearing character; irregular twitching of muscular fasciculi; painful tonic spasm (cramps), and tremor of some of the muscles of the extremities; besides, in many cases, general febrile symptoms, usually of a moderate grade, and in rare cases eclampsia, particularly in young subjects. The motor weakness may be hours or days in reaching its highest degree, and this may be gradual, or by a succession of sudden accessions with intervals of partial recovery. Before it becomes complete the gait is similar to that of double hemiplegia of cerebral origin, for it is due to involvement of the pyramidal tracts. The superficial (skin) and deep (tendon) reflexes of the extremities are preserved, and the paralyzed muscles respond to such reflex excitation and also to electrical stimulation; the reaction of degeneration is absent, and the muscles of the extremities do not undergo atrophy except from disuse. Later, the reflexes become increased, often to a high degree. Spastic conditions and contractures may follow, and frequently become permanent. The lower trunk muscles may be involved and flaccid, showing diminished response to faradic excitation; there is retention of urine at first, followed by incontinence from reflex action. The sphincter ani is also cut off from cerebral control.

The pulse is usually rapid. Sensation is rapidly lost; anæsthesia takes the places of the painful paræsthesia, though frequently a condition of hyperæsthesia exists, particularly in the distribution of the nerve coming from the limit of the lesion, where it may form a zone producing the sensation of constriction about the trunk. The dorsal spines may also be sensitive to pressure at this level, but the pain and hyperæsthesia are not increased by active or passive movement. All sensation in the parts supplied from below the upper margin of the lesion may be cut off. If recovery takes place, sensation is usually regained before motion. Death may result from general exhaustion, though myelitis limited to the dorsal region is the most favorable form for recovery. If it extends to the cervical region, respiratory failure may follow; or if it invades the lumbar enlargement, bedsores, cystitis, and nephritis may hasten death. The course and duration, as well the acuteness of onset, are very variable. Months may elapse before recovery takes place; or sensation may be partially recovered, and there may be some return of power, but with spastic conditions which may persist for years. But some patients recover so completely and rapidly that after a few months no trace remains, except an increased patellar tendon reflex.

When the myelitic process involves the lumbar enlargement, either primarily or by the extension from the dorsal region, we have—

2. Acute Transverse Dorso-Lumbar Myelitis.—Here additional phenomena are present, due to the destruction of the motor and reflex connections for the muscles of the lower extremities, and for the bladder, rectum, and sexual apparatus. In place of increased reflexes we have both skin and tendon reflexes abolished. The reflex functions of the bladder and rectum are lost, as already described. Alkalinity of the urine, cystitis, and suppurative nephritis may follow. The muscles of the extremities are flaccid, undergo atrophy, and after a time the reaction of degeneration is found (frequently not before a week or two after paralysis).

Trophic disturbances in the skin and subcutaneous tissues, in the form of bedsores, frequently occur over the sacrum and the buttocks, and sometimes suppuration in the pelvic cellular tissue. The skin of the extremities is often œdematous, livid, and, in late stages, subnormal in temperature, although at the onset it may be supernormal—it may be dry or moist.

3. Acute Transverse Cervical Myelitis.—In this form the lower extremities and pelvic viscera are affected as in the dorsal variety; and, in addition, changes occur in the upper extremities similar to those described in the dorso-lumbar variety for the lower extremities—namely, trophic changes in the muscles and nerves, and in the electrical reaction and the reflexes; besides, vaso-motor, pupillary, cardiac, and respiratory disturbances occur; painful rigidity of the cervical muscles, pallor or flushing of the face and neck, contraction or dilatation of the pupils, and slowness or rapidity of the pulse, according to whether the irritative or destructive stage prevails. Optic neuritis has been found associated. Priapism is a more frequent condition in this than in the dorso-lumbar variety. The upper extremities are paralyzed first. If the process remains strictly a central myelitis, the lateral column will not be invaded and the lower extremities will not be involved. Should the section involved lie

above the cervical enlargement, the upper extremities would be affected like the lower ones—namely, preserved and exalted reflexes, and absence of trophic changes in the muscles. Respiration, deglutition, articulation, and the diaphragmatic functions (origin of phrenic nerve) may exhibit great disturbance, death usually resulting from respiratory failure.

The morbid anatomical changes which constitute the basis of these symptoms are the result of irritative and destructive inflammatory processes in the cord; the irritative stage being represented by hyperæmia, exudation from the vessels, proliferation of lymphoid elements, minute capillary extravasation, and frequently by hemorrhage from small arteries. Later, the myelin of the medullated fibres undergoes swelling, granular degeneration, and disorganization. The axis cylinders and nerve cells may become swollen, opaque, or granular, some undergoing complete disorganization—this representing the stage of destruction. The latter process may continue until complete softening occurs, showing, on section, a softened or liquefied state, and, under the microscope, the débris of nerve cells, broken cell processes, myelin drops, lymphoid bodies, red blood cells, and the so-called granule cells—bodies many times the size of lymphoid elements, filled with highly refractive granules. When the process stops short of complete disorganization and a more chronic stage is reached, and also in the earlier stages of chronic myelitis, the neuroglia proliferates, forming more numerous and thicker bands; the walls of the vessels are thickened; lymph spaces may be obliterated or choked with lymphoid cells; so-called spider cells (Deiter's cells), larger connective-tissue cells, with larger and numerous processes, are scattered through the tissues, and the so-called corpora amylacea are numerous.

This condition is known as sclerosis. It presents a grayer appearance in unstained sections; deeper red with carmine, and a paler color with the Weigert process, than the normal tissue. The naked-eye appearance, where the cord has undergone softening, has been divided into red, yellow, and white softening, representing successive stages during which the extravasated red blood cells are being absorbed. The more vascular parts of the cord are usually the first to undergo softening. These are on either side of the central canal in the gray matter, the process usually extending outward to the white columns, frequently along blood-vessels or connective-tissue trabeculæ. The appearances of softening or sclerosis on section are often quite irregular, particularly in the white matter.

Simple Softening of the Cord is met with, probably due to occlusion of the vessels from arteritis, embolism, or thrombosis. There are fewer signs of an active inflammatory process, white or yellow softening being found. It is characterized by the absence of marked irritative symptoms, which precede the destructive stage of the inflammatory variety.

Hemorrhagic Myelitis is characterized by a more sudden onset of the paraplegia than occurs in the inflammatory form, frequently becoming complete in a few minutes. The occurrence of traumatic conditions, as concussion, injuries to the vertebræ, etc., will frequently lend support to this diagnosis. The later changes are similar to the forms already described. A hemorrhage may be so small and so located as to produce only unilateral paralysis at first, as in the anterior cornua, paraplegia recurring later from a more general myelitis, secondary to the hemorrhage.

Compression Myelitis presents the usual features of a transverse myelitis, except that paraplegia may be preceded for a longer time by irritative symptoms than in the common inflammatory form, as compression is usually slow, being due to tumors in the membranes, or to vertebral disease. In its common form it is known as *the paraplegia of Pott's disease*. The paraplegic symptoms are those due to irritation of the sensory and motor nerves at their exit from the membranes, which may present pachymeningitis, or from the vertebral foramina, giving rise, chiefly, to bilateral pains in the trunk or upper extremities, in the course of the sensory nerves involved, and being frequently accompanied by hyperæsthetic or anæsthetic areas. Herpes zoster sometimes occurs. The reflexes and the functions of the sympathetic may be disturbed (pupillary, vaso-motor, sweating), and even muscular paresis and atrophy of the upper extremities follow, from this cause. The chief diagnostic features are evidence of localized bone disease, curvature, spinal tenderness on movement, and reflex muscular spasm at the region involved on flexure of the spinal column. Paraplegia, when it does develop, may appear suddenly. The distribution of the paralysis is usually that of the dorsal or cervical varieties, and presents the variations in localization of lesion described for these conditions.

It is considered one of the most favorable forms of myelitis as regards a partial recovery.

In Compression by Tumors pain is such a prominent feature that Cruveilhier was led to characterize the paralysis which it produces as *paraplegia dolorosa*. The pain is lancinating, at first intermittent, but finally becoming constant, and increased by movement. It is of a more severe character than the pain of vertebral caries. All varieties of localization may be presented, and all possible variations in mode of onset, and in slowness or rapidity of course, depending on the location, size, and rapidity of growth of the neoplasm, various symptoms becoming prominent according as different functional tracts are chiefly involved. Sarcomata (including gliomata) are the most frequent; gummata and tubercle are about equally common, while carcinomata and hydatids are among the neoplasms occasionally found.

Meningo-myelitis may result from an extension of the myelitic process outward to the membranes, or of a meningitic process into the cord.

Meningitis, both acute and chronic, may produce paraplegia by involving the anterior roots. When the entire circumference of the cord is involved, the sensory roots being also affected, we have a condition similar to involvement of a mixed peripheral nerve.

The lancinating pains, constrictive bands, muscular rigidity, and convulsive twitching are greater than in simple myelitis, and usually serve to distinguish one from the other. In cervical meningitis the paraplegia will be confined to the upper extremities, as the pyramidal tracts will be free unless there develop a meningo-myelitis, when these tracts may be involved.

As numerous examples have already been given, it will be unnecessary to review all the possibilities that may follow, or to consider all the varieties of meningitis. With a clear appreciation of the anatomical relations and functions, the extent and location of the lesion may be concluded from the symptoms, or *vice versa*. *Traumatisms* may also be left out of consideration, after what has been said on compression myelitis.

Poliomyelitis Anterior (infantile and adult spinal paralysis) may produce a paraplegia; usually, however, the lesion in this case being limited to the anterior cornua and their neighborhood in the anterior columns, certain cell groups succumb, while others survive, leaving an irregular distribution, not symmetrically bilateral, and therefore not paraplegic. The anterior group of one leg is most frequently involved. Nearly one-half the cases are monoplegic, and when bilateral the paralysis is frequently not symmetrical. It should be stated, however, that often during the first hours, and sometimes days, of this sudden paralysis, there is complete paraplegia, the trunk and all four extremities being involved; but it is distinguished from transverse myelitis by the absence of marked sensory or vesical symptoms, and by abolished reflexes. The statement concerning the unsymmetrical distribution of the paralysis also applies to the subacute form of this disease, and in fact to chronic degeneration of the anterior cornual cells, which produces progressive muscular atrophy.

The condition known as Landry's acute ascending paralysis, and also the lateral amyotrophic paralysis of Charcot, which may produce paraplegia, are so rare as to warrant no more than their mention.

The paraplegiform affection due to the ataxia of tabes dorsalis may give place to a true paraplegia by an extension of the degenerative process to the anterior cornua, producing muscular atrophy, or, in the form combined with sclerosis, by involving the pyramidal tracts, it may develop spastic and ataxic paraplegia.

Spastic Paraplegia results, as we have seen, from the symmetrical involvement of the pyramidal tracts in any part of their course, and is usually secondary to a transverse myelitis, or to a transverse lesion of the pyramidal tracts in their intracranial portion, or, particularly in infants, to arrested development of the cortical motor areas, in both hemispheres. It is sometimes termed double hemiplegia. It may affect chiefly the arms, or the legs, and has the usual characteristics, namely, spastic movements, exaggerated reflexes, and absence of sensory and trophic changes. Primary sclerosis of the pyramidal tracts in the cord is a rare condition, if it occurs at all.

Intracranial tumors, by pressure upon the crura and pyramidal tracts in the pons and medulla, or in the cerebellum, may produce paraplegia or paraplegiform symptoms, in some cases being ataxic rather than paretic, or a combination of both.

Paraplegia from Multiple Neuritis is characterized by both motor and sensory impairment involving all the extremities, and ascending the members from periphery to trunk, also by pain, hyperæsthesia, tenderness of the nerve trunks to pressure, trophic changes in the muscles, the reaction of degeneration, œdema of the extremities, the absence of visceral disturbances (bladder and rectum), and the absence of a constriction band. It is most common in those addicted to alcohol, but is also a manifestation of certain endemic diseases, such as beriberi or kakki.

Pseudo-hypertrophic Paralysis produces a form of paraplegia somewhat similar to poliomyelitis anterior, and is considered by some to be a form of that affection, by others to be a connective-tissue disease of the muscles. The increase in size of the muscle (usually the calves) and its hardness, serve to distinguish it from ordinary poliomyelitis anterior.

There are several forms of paraplegia usually classified under functional disorders of the nervous system, namely, hysterical paraplegia, paraplegia depending upon idea, reflex paraplegia, malarial paraplegia, anæmic paraplegia, alcoholic paraplegia, and toxic paraplegia.

Hysterical Paraplegia is a less frequent form of hysterical paralysis than that of hemiplegic or monoplegic distribution. It is less apt to be confounded with paralysis of organic origin than the other varieties, for the reason that hysterical paralysis resembles in its symptoms a lesion of cerebral motor tracts in the brain and cord, rather than one in the remaining portion of the motor tract in the cord and peripheral nerves. The reaction of degeneration is absent in hysterical paralysis. Atrophy may be present, but it is that form which is dependent upon disuse. The volume of the muscles may be reduced in such cases, and may give a feeble reaction to electrical excitation; but, what is of the utmost importance, *faradic excitability is preserved, and the contractions are quick*. The skin and muscles may be cold, livid, and flabby, resembling paralysis from peripheral or cornual disease, and contractures may form as in degeneration of the pyramidal tracts, though in many cases the muscles and skin appear normal, except that voluntary control is lost. The tendon reflexes are rarely lost; usually they are increased, sometimes excessively so. The bladder and rectum are not usually affected, though voluntary control over these organs may be lost. Thus, paraplegia from transverse myelitis of the lumbar enlargement would not be confounded with hysterical paraplegia on account of the absence in the latter of trophic and electrical changes in the muscles and nerves, bedsores, and atrophic changes in the bladder; but the latter might closely resemble paraplegia from transverse dorsal myelitis in which these signs are absent. The presence of a constriction band at the level of the segment involved, and the sensory, motor, or reflex disturbances in this zone would exclude hysterical paraplegia. Hysterical paralysis of all the extremities might simulate cervical paraplegia of myelitic origin, but would be differentiated from it by the presence of trophic changes in the muscles of the upper extremities, and by the vaso-motor and visceral symptoms which accompany organic lesions of this region. Again, the onset and course of the two classes of disease are usually sufficient to distinguish one from the other. The irritative stage of most acute or subacute organic diseases of the cord, in which pain, hyperæsthesia, and slight motor irritation precede the paretic and anæsthetic period, is not usually present in hysterical paraplegia. In the latter, sudden development of the paraplegia, and sudden variations in its distribution and intensity, often serve to indicate it. Anæsthesia and analgesia may have a distribution inconsistent with the lesions producing paraplegia of structural origin.

It must not be forgotten that hysteria may accompany organic lesions, and should not, therefore, be taken as proof of the hysterical nature of the paralysis, unless organic lesions can be excluded.

Paraplegia Dependent upon Idea is a form described by Dr. J. Russell Reynolds. Though closely allied to hysterical paralysis, it may be independent of hysteria, hypochondriasis, and simulation, though frequently associated with functional debility, anxiety, and a morbid imagination. "Many cases of paraplegia following railroad accidents," says Reynolds, "may be classed under this head; the attention of the victim being influenced in the most unfortunate manner by the stories of friends, inquiries of his physician, the talk of his attorney, and the sober face of the company's physician." Pain, distributed in a manner inconsistent with the anatomical relations, on the supposition of an organic lesion; spasm, which, however, is sometimes relaxed in a remarkable way when the patient's attention is directed elsewhere; and paralysis, which is rarely complete, and almost identical with a voluntary attempt not to move the parts, or to move them with care, as in simulation, are the chief features of the affection. The removal of the morbid idea, *i.e.*, that the patient is paralyzed, or has a severe disease, results in improvement or cure. An award for damages has also frequently proven a valuable therapeutic agent in such cases.

Reflex Paraplegia, termed by older writers urinary paraplegia, was shown by Brown-Séquard to follow irritation not only of the genito-urinary tract, but also of the intestines and other viscera in animals. He attributed it to an anæmia of the cord, due to contraction of the blood-vessels, while Charcot considered the motor weakness due to inhibitory action of the sensory irritation. While we must admit the possibility of this form of paraplegia, it should not be forgotten that organic lesions may have been lost sight of, or might be sufficiently slight to be transitory. The positive evidence of some form of peripheral irritation, the removal of which has been followed by recovery, is the only basis on which it should be admitted, and then only in the absence of indications of organic disease.

Malarial or Intermittent Paraplegia is a curious form of poliomyelitis anterior, which recurs with the periodicity of intermittent fever. Alcoholic paraplegia, when not due to multiple neuritis, is a temporary affair, following an alcoholic debauch.

Anæmic Paraplegia follows ischæmia of the cord, from pressure on the abdominal aorta, and from pressure or occlusion of the iliac arteries within the pelvis, or ischæmia of muscles; rare conditions.

The indications for *treatment*, where paraplegia exists, are those adapted to the correction of the various pathological processes concerned in the diseases which we have considered. More than a brief résumé would carry us beyond the proper limits of this work. In the irritative stage of acute meningitic and myelitic processes, rest is the first essential. The reduction of hyperæmia, by means of agents supposed to cause vaso-motor contraction, such as ergot and belladonna, and the relief of pain by means of cutaneous irritation (the actual cautery, blis-

ters, sinapisms, cupping, etc.), and also by morphine, may be attempted. The use of cold to reduce inflammation in spinal-cord disease is an uncertain procedure, concerning the real effects of which we know but little.

In the destructive stage of these conditions, when paralytic and anæsthetic phenomena are present, rest may still be an important factor. The use of mercury, and of potassium iodide, may be of service in this period to promote the absorption of exudations.

In later stages and in chronic cases the use of tonics, of iron, strychnine, arsenic, etc., is called for. Nitrate of silver may be indicated. Electricity is usually to be avoided in the irritative stage of an acute affection; or, if used, as for the relief of pain, it should be in the form of a gradually increased galvanic current, avoiding interruption. For the paralysis, the interrupted galvanic and the faradic currents, to produce muscular contractions, and spinal applications of the uninterrupted galvanic current, are of value, as are also massage and passive movements. For anæsthesia, the faradic brush is often serviceable.

Extreme care and cleanliness are essential in all cases of paraplegia, and the avoidance of pressure and irritation over the buttocks and sacrum, on account of the danger and frequency of bedsores. Attention must be paid to the bladder and rectum. Catheterization, conducted with extreme cleanliness, may be necessary, and also antiseptic irrigation of the bladder. Constipation must be prevented by cathartics, enemas, etc. Compression myelitis from caries, dislocations, fractures, and other traumatisms requires appropriate surgical treatment.

W. R. Birdsall.

PARASITES.—A parasite is an organism which lives, temporarily or permanently, within the body or on the surface of some other living thing upon which it feeds. Evidently, then, not only may there be both phytoparasites and zoöparasites, but also that form which is parasitized upon and is known as the host may be equally either plant or animal. Among forms which find in man at some time or in some region a subject for attack, the phytoparasites include the prominent group of bacteria which have received attention elsewhere, and a few fungi of etiological importance, in dermal affections chiefly, which have also been discussed. Here will be given a brief discussion of the animal parasites of man, with especial reference to their biological and etiological relations.

It is important to notice first the wide range in degree of parasitism exhibited and the manner in which the various grades merge into one another, producing a scale of dependence in which almost every stage is represented. Most independent of all are the temporary parasites, like the mosquito, bedbug, or leech, which stay by the individual host only long enough to secure a single meal, and which present clearly the structure and habits of free living organisms. Some leeches suggest most plainly the close relation between the carnivorous and the parasitic habit since they often devour bodily small aquatic forms, but when favored by opportunity extract the blood of larger animals. More dependent are such forms as the fleas which can change their host and often do so, and yet their structure has been highly modified in the loss of wings which are generally characteristic of insects and by the development of powerful leaping and grasping organs. Somewhat further modified in the direction of parasitism are the lice, which, moreover, lack special means for effecting a change of host, and may be included among the list of stationary parasites—*i.e.*, those that remain with a single host constantly, or at least for considerable periods of time.

All of the forms thus far noted are parasitic upon the exterior of the host, and consequently are denominated Epizoa or ectoparasites. All human ectoparasites belong to the group of Arthropoda, and include both mites (*cf. Arachnida*) and true insects (*cf. Insects*). Among the water-living animals, however, soft-bodied forms, such as flat worms (Trematoda) and unicellular animals (Protozoa) occur as Epizoa. With the gradual assumption of an aërial or terrestrial existence on the part of the host such parasites were necessitated, if they had not already sought more sheltered regions, now at least to abandon the external surface and to colonize internal organs where thin mucous membranes afforded facilities for extracting nourishment similar to those which existed on the thin outer skin of the aquicolous animal. The choanæ, pharynx, gills, lungs, alimentary canal, and even the bladder were thus invaded by forms whose kinship to the ectoparasitic species on these lower animals is too plain to fail of recognition.

The Entozoa or endoparasites of man, however, do not even belong to the same branch of the animal kingdom as the forms ectoparasitic upon him, with the single exception of the rare and aberrant Linguatulids, now usually regarded as highly degenerate arachnids (*q.v.*), though formerly classed with the Cestoda. The human Entozoa include Protozoa, Trematoda, Cestoda, and Nematoda, and many of them are highly modified in adaptation to the parasitic mode of existence, as compared to the related free living forms which, however, are entirely wanting in the second and third groups listed.

The term helminthology has been used as synonymous with animal parasitology, and yet this is a considerable extension of its original meaning. The Helminthes or intestinal worms included the pre-eminently parasitic groups, such as Trematoda, Cestoda, and Nematoda, while the Protozoa, Arthropoda, and even the few parasitic Terbellaria, which are in fact closely related to Trematoda, were omitted. The term became thus one of convenience rather than of scientific accuracy.

It is necessary to emphasize the fact that neither Helminthes nor parasites constitute a group of systematic value. At most the forms are related in a biologic sense and not structurally, for they are comprehended in several distinct branches of the animal kingdom, and a given form is often more closely related to free living species than to other parasitic forms. Even the narrower term Helminthes embraced forms of little similarity to each other and rightly to be distributed with their related free living species into several distinct groups, namely, the Linguatulida to the Arachnida, the Trematoda and Cestoda to the Plathelminthes, and the Nematoda to the Nemathelminthes.

Location.—While the majority of endoparasites inhabit the alimentary canal and its adnexa, there is no organ which is immune to them. The following list of human parasites arranged according to the organ inhabited will serve to indicate the extent of the parasitic habit, and will assist in the identification of a given form. The records given apply only to the human host. Parasites are entered under the normal location of the species, and in the most frequent erratic location only; a few forms of doubtful standing, as human parasites or of uncertain location in this host, are omitted.

Parasite.	Stage.	Type of parasitism.	Normal habitat.	Recorded in U. S. A.
Skin and subdermal tissue.				
Leptodera Niellyi	Larva	Accidental.	Europe	No.
Gnathostoma siamense	Adult.	Occasional.	Siam	No.
Filaria medinensis	Adult.	Normal	Africa	Yes.
Uncinaria duodenale	Larva.	(?)	Cosmopolitan.	Yes.
Eye.				
Filaria loa	Adult.	Normal	Africa	Yes.
Filaria lentis	Adult.	(?)	(?)	No.
Filaria conjunctivæ	Adult.	Occasional.	Europe	No.
Cysticercus cellulosæ	Larva.	Erratic	Europe	No.
Echinococcus polymorphus.	Larva.	Erratic	Europe	Yes.
Brain and membranes.				
Cysticercus racemosus=cellulosæ.	Larva.	Erratic	Europe	Yes.
Cysticercus acanthotrias.	Larva.	Erratic (?).	U. S. A.	Yes.
Echinococcus polymorphus.	Larva.	Erratic	Europe	Yes.

Parasite.	Stage.	Type of parasitism.	Normal habitat.	Recorded in U. S. A.
Brain and membranes.				
Paragonimus Westermanni.	Adult.	Erratic	Asia	*
Connective tissue.				
Fasciola hepatica	Adult.	Erratic	Europe	*
Bothriocephalus Mansoni	Larva.	Occasional.	China	No.
Cysticercus cellulosæ	Larva.	Normal	Europe	Yes.
Cysticercus acanthotrias.	Larva.	Normal	U. S. A.	Yes.
Echinococcus polymorphus.	Larva.	Normal	Europe	Yes.
Filaria loa	Adult.	Normal	Africa	Yes.
Paragonimus Westermanni.	Adult.	Erratic	Asia	*
Muscles.				
Cysticercus cellulosæ	Larva.	Normal	Europe	No.
Cysticercus acanthotrias.	Larva.	Normal	U. S. A.	Yes.
Trichinella spiralis	Larva.	Normal	Cosmopolitan.	Yes.
Heart.				
Filaria Magalhaesi	Adult.	(?)	So. Amer.	No.
Cysticercus cellulosæ	Larva.	Erratic	Europe	No.
Echinococcus polymorphus.	Larva.	Erratic	Europe	Yes.
Blood-vessels.				
Fasciola hepatica	Adult.	Erratic	Europe	*
Schistosoma hæmatobium.	Adult.	Normal	Africa	Yes.
Echinoccus polymorphus.	Larva.	Normal	Europe	Yes.
Filaria immitis (?)	Adult.	Occasional.	Europe	*
Filaria embryos (see key under Nematoda).		Normal		Yes.
Lymph vessels.				
Filaria Bancrofti	Adult.	Normal	Tropics	Yes.
Filaria volvulus	Adult.	Normal	Africa	No.
Filaria lymphatica	Adult.	Occasional.	Europe	No.
Lungs.				
Fasciola angusta	Adult.	Erratic	Africa	No.
Paragonimus Westermanni.	Adult.	Normal	Asia	*
Cysticercus cellulosæ	Larva.	Normal	Europe	No.
Echinococcus polymorphus.	Larva.	Normal	Europe	Yes.
Strongylus apri	Adult.	Occasional.	Europe	No.
Liver.				
Fasciola hepatica	Adult.	Occasional.	Europe	*
Opisthorchis felineus	Adult.	Normal	Russia	No.
Opisthorchis sinensis	Adult.	Normal	Asia	Yes.
Opisthorchis noverca	Adult.	Normal	Asia	No.
Distoma Rathouisi	Adult.	Occasional?	Asia	No.
Dicrocœlium lanceatum.	Adult.	Normal	Europe	*
Cysticercus cellulosæ	Larva.	Normal	Europe	No.
Echinococcus polymorphus.	Larva.	Normal	Europe	Yes.
Paragonimus Westermanni.	Adult.	Erratic	Asia	*
Small intestines.				
Fasciolopsis Buski	Adult.	Normal	Asia	No.
Opisthorchis felineus	Adult.	Erratic	Russia	No.
Opisthorchis sinensis	Adult.	Erratic	Asia	No.
Heterophyes heterophyes	Adult.	Normal	Africa	No.
Dibothriocephalus latus.	Adult.	Normal	Europe	Yes.
Dibothriocephalus cordatus.	Adult.	Occasional.	Greenland.	No.
Diplogonoporus grandis	Adult.	Occasional.	Japan	No.
Dipylidium caninum	Adult.	Occasional.	Europe	Yes.
Hymenolepis nana	Adult.	Normal (?).	Europe	Yes.
Hymenolepis diminuta	Adult.	Occasional.	Europe	Yes.
Hymenolepis lanceolata	Adult.	Occasional.	Europe	No.
Davainea madagascarensis.	Adult.	Occasional (?).	Africa	No.
Tænia solium	Adult.	Normal	Cosmopolitan.	Yes.
Tænia saginata	Adult.	Normal	Cosmopolitan.	Yes.
Tænia africana	Adult.	Normal	Africa	No.
Tænia confusa	Adult.	Normal (?).	U. S. A.	Yes.
Strongyloides stercoralis.	Adult.	Normal	Asia	Yes.
Trichinella spiralis	Adult.	Normal	Cosmopolitan.	Yes.
Strongylus subtilis	Adult.	Normal	Africa	No.
Uncinaria duodenalis	Adult.	Normal	Cosmopolitan.	Yes.
Uncinaria americana	Adult.	Normal	America	Yes.
Physaloptera caucasica	Adult.	(?)	Caucasus	No.
Ascaris lumbricoides	Adult.	Normal	Cosmopolitan.	Yes.
Ascaris canis	Adult.	Occasional.	Europe	*
Ascaris maritima	Adult.	Occasional.	Greenland.	No.
Oxyuris vermicularis	Adult.	Normal	Cosmopolitan.	Yes.
Gigantorhynchus gigas	Adult.	Occasional.	Cosmopolitan.	No.
Gigantorhynchus moniliformis.	Adult.	Occasional.	Cosmopolitan.	No.

* Present in the United States of America in some other host, hence easily possible in man, although no record of its occurrence in the human host was found.

Parasite.	Stage.	Type of parasitism.	Normal habitat.	Recorded in U. S. A.
Large intestine.				
Gastrodiscus hominis	Adult.	Occasional (?).	India	Yes.
Trichocephalus trichiurus	Adult.	Normal	Cosmopolitan.	No.
Oxyuris vermicularis	Female.	Normal	Cosmopolitan.	Yes.
Kidney.				
Echinococcus polymorphus.	Larva.	Normal	Europe	Yes.
Dioctophyme renale	Adult.	Occasional.	Europe	*
Bladder.				
Leptodera pellio	Adult.	Accidental.	Europe	No.
Anguillula aceti	Adult.	Accidental.	U. S. A.	Yes.

SPUTUM—EGGS.

Parasite.	Frequence.	Size in microns.	Plate E.
Fasciola angusta	Recorded once.	143–151 × 82–88.	
Fasciola hepatica, Fasciola magna, etc.	Not observed, but possible.	Given below under Fæces.	
Paragonimus Westermanni.	Frequent	88–103 × 53–68.	Fig. *a*.
Strongylus apri	Few cases	50–100 × 39–72.	

SPUTUM—EMBRYOS.

Filaria, many species possible (see key under *Nematoda*).

URINE—EGGS.

Parasite.	Frequence.	Size in microns.	Plate E.
Fasciola hepatica, Fasciola magna, etc.	Not observed, but possible.	Given below under Fæces.	
Schistosoma hæmatobium	Frequent	135–150 × 55–66.	Fig. *c*.
Filaria Bancrofti	Recorded once.	25–28 × 15 (or 35 ?).	
Dioctophyme renale	Few cases	64–68 × 40–49.	Fig. *b*, *b'*.
Oxyuris vermicularis†	Common	50–54 × 20–27.	Fig. *d*, *d'*, *d''*.

URINE—EMBRYOS.

Filaria, many species possible (see key under *Nematoda*).

The eggs of the other kidney parasites will not hatch as long as kept in urine.

Adult forms, like accidental parasites of the Nematode type, are so small as to be easily taken for embryos (see Anguillula aceti, etc., under *Nematoda*).

FÆCES—EGGS.

Parasite.	Frequence.	Size in microns.	Plate E.
Gastrodiscus hominis	Recorded once.	150 × 72.	
Fasciola hepatica	Several cases	130–172 × 72–80.	Fig. *e*.
Fasciola magna	Not recorded	109–168 × 75–96.	Fig. *aa*.
Fasciola angusta	Recorded once.	143–151 × 82–88.	
Distoma Rathouisi	Recorded once.	150 × 80.	
Fasciolopsis Buski	Several cases	120–126 × 77.	
Opisthorchis felineus	Several cases	26–30 × 11–15.	Fig. *f*.
Opisthorchis noverca	Recorded once.	34 × 21.	Fig. *ff*.
Opisthorchis sinensis	Several cases	27–30 × 15–17.	Fig. *g*.
Dicrocœlium lanceatum.	Several cases	40–45 × 22–30.	Figs. *h*, *h'*.
Heterophyes heterophyes.	Frequent	26–30 × 15–17.	Fig. *i*.
Paragonimus Westermanni.	Frequent	88–103 × 53–68.	Fig. *a*.
Dibothriocephalus latus.	Frequent	68–71 × 45	Figs. *k*, *k'*.
Dibothriocephalus cordatus.	Reported once.	75–80 × 50.	
Diplogonoporus grandis	Few cases	63 × 48–50.	Fig. *l*.
Dipylidium caninum	Few cases	43–50, embryo 32–36.	Fig. *m*.
Hymenolepis nana	Frequent	39, or 43 × 31.	Fig. *n*.
Hymenolepis diminuta	Several cases	70–86, embryo 36 × 28.	Figs. *o*, *o'*.
Hymenolepis lanceolata	Recorded once.	50 × 35.	
Tænia solium	Frequent	30–35, embryo 20	Figs. *p*, *p'*.
Tænia saginata	Common	(Yolk membrane 70) 30–40 × 20–33.	Figs. *q*, *q'*.
Tænia africana	Recorded once.	31–34 round, or 34 × 39.	Fig. *r*.
Tænia confusa	Two cases	30 × 39.	Fig. *s*.
Strongyloides stercoralis.	Frequent	67 × 37.	Fig. *t*.
Trichocephalus trichiurus.	Common	50–54 × 21–23.	Figs. *u*, *u'*.

* Present in the United States of America in some other host, hence easily possible in man, although no record of its occurrence in the human host was found.

† Only in female through infection of vagina from rectum.