be produced by a light application of the Paquelin cautery; blisters are no longer in favor. The same may be said for mercury, formerly so much employed. Iodide of potassium is still recommended by many writers. Landon Carter Gray advises combining it with ergot and quinine. Persistent vomiting is best treated by hypodermic injections of morphine, in cases which cannot be controlled by simpler remedies, such as iced champagne, sinapisms, etc. Surgical treatment (the removal of the arch of a vertebra, incision into the dura, and drainage) has not given encouraging results. The nutrition is of the greatest importance, especially in protracted cases. While there is still much fever, milk and broths are indicated; in the later stages and in the intervals between the exacerbations any light nutritious food may be given. Unfortunately there is often great difficulty in inducing the patient to take nourishment, and forced feeding may have to be used. As general tonics quinine in large doses, iron, the iodides, and belladonna have been recommended.

II. *Acute Secondary Leptomeningitis.*—This is distinguished from the form just described by the fact that it is secondary, not primary; according to many observers it is distinguished by the fact that it is caused by micro-organisms other than the diplococcus intracellularis Weichselbaum. These cases are very hard to distinguish from sporadic cases of cerebro-spinal fever proper, cases occurring in the absence of an epidemic. Councilman makes a sharp distinction between those sporadic cases which are proven to be due to infection by Weichselbaum's meningococcus, and the secondary cases which are caused by the pneumococcus, streptococcus, etc. Osler divides the meningitides into four classes: (1) Those due to the diplococcus intracellularis; (2) those due to the pneumococcus, which are usually secondary to pneumonia; (3) those due to the tubercle bacillus; and (4) those due to the streptococcus. Under this last head would come most of the cases which are secondary to otitis media, to mastoiditis, to erysipelas, to periostitis following trauma, to septicæmia, to ulcerative endocarditis, to extension from neighboring foci of suppuration, as pelvic cellulitis, suppurative pleuritis, etc., also the cases of meningitis complicating the acute infectious diseases. Exceptionally other micro-organisms are found in these secondary cases, as the typhoid bacillus, the colon bacillus, the staphylococcus pyogenes, and the gonococcus.

The extent of the disease is very variable. The suppuration may be limited to small foci or may form a general infiltration. The pathology is that of purulent inflammation, and does not need recapitulation. An extension of the process to the dura seldom occurs; the inflammation remains limited to the pia-arachnoid.

The symptoms are those of acute cerebro-spinal meningitis, complicated by the pre-existing disease. Headache, delirium, rigidity of the neck with or without retraction, high fever, vomiting, and convulsions, when developing in the course of an acute infectious disease, as a sequence of trauma or in a general septicæmia, would indicate an extension of the infection to the cerebro-spinal membranes. There may be trismus, epileptiform attacks, hyperæsthesia of the muscles and skin, twitchings or muscular spasms. As in the epidemic form, herpes is common. The temperature varies, but is rarely as high as in the epidemic form, and the pulse is apt to be slow. The pupils are at first contracted or unequal, later they are widely dilated.

The treatment is properly directed to the primary disease. For the cerebro-spinal symptoms the methods described above are to be tried, but the prognosis is very bad, such cases being almost invariably fatal.

III. *Acute External Meningitis, Acute Pachymeningitis, Acute Peripachymeningitis* (*"Perimeningite Aiguë"*).—By some authors these are considered two different affections, the latter involving the peridural connective tissue only, the former the dura itself.

"Perimeningitis" was first described and named by Albers in 1833 as a primary acute affection of the connective tissue surrounding the dura mater. He reported two cases. In 1879 Leyden proposed the name pachymeningitis, a name which has been generally accepted by the Germans, while the French have adhered to the original name. The disease is a rare one. Deléarde in 1900 reviewed the literature and collected sixteen cases, adding one of his own. Of these, fourteen were men, the larger number being between the ages of twenty and thirty, the exciting cause apparently exposure and overexertion. Autopsy revealed the presence of inflammation in the peridural tissue only, which sometimes extended to the tissues around the vertebral column, but was essentially primary in the connective tissue surrounding the dura.

The port of entry for the infection is unknown. The organisms isolated are the staphylococcus aureus in two cases (Antony and Netter), the streptococcus in one (Deléarde). As the dura is free from the vertebral column laterally and posteriorly, the inflammatory exudate tends to collect on these surfaces and may travel out through the intervertebral foramina along the spinal nerves, as in Meslier's case. The effusion may be serous and abundant, or fibrinous, forming a false membrane over the outer surface of the dura; or, especially in the very rapid cases, it may consist of a small amount of bloody pus. The extent of the exudate varies much. Usually the cord is softened at the point of greatest inflammation, but is normal above and below.

There are no pathognomonic symptoms. The onset is usually sudden, with pain in the limbs and paraplegia, but without spasm and with normal or diminished reflexes. Anæsthesia appears early, preceded by lightning pains, and usually there is a zone of hyperæsthesia just above the anæsthetic area. Pressure on the spine over the inflamed region is very painful. The general symptoms are fever with morning remissions and a typhoid condition. Death is preceded by a fall of temperature (89.6° F. in the rectum was noted in Lemoine and Lannon's case). Symptoms of internal meningitis, muscular spasm, rigidity of the neck, etc., appear late, if at all, and are evidences of an extension of the process to the pia-arachnoid. The rapid paralysis and anæsthesia without spasm or contraction are the chief diagnostic symptoms.

The prognosis is extremely bad. One case, that of Antony, operated on by Chipault, who performed laminectomy of the seventh to the eleventh dorsal vertebræ, recovered temporarily, but soon succumbed to suppurative endocarditis. Usually death occurs early. Meslier's case, lasting eight days, is the longest in Deléarde's series. Asphyxia from paralysis of the muscles of the thorax is the most common cause of death.

Pachymeningitis or peripachymeningitis secondary to some inflammatory process in the body is, according to the authors quoted above, to be strictly distinguished from the primary form, but the usual text-books make no such distinction, nor do they lay much stress on the limitation of the process to the dural or to the peridural tissue. Caries of the vertebræ is, naturally, the commonest cause of this condition; then follow suppurative processes near the vertebral canal, as psoas abscess, abscess of the muscles of the back, retropharyngeal abscess, deep sacral bedsores. Gowers believes that most of these cases of so-called secondary pachymeningitis are in reality cases of primary meningitis with secondary abscess formation. The inflammation almost never passes through the dura to the pia-arachnoid. Usually the inflammatory exudate is semipurulent and forms a layer over the outer surface of the dura, especially in the space between the posterior surface of this membrane and the arches of the vertebræ. In those cases which follow caries of the spine the vertical extent of the process is limited to the extent of bone disease. In any case gravity seems to affect the distribution of the exudate, for it rarely rises above the upper cervical region. In acute cases the symptoms are similar to those of the primary form, but not so rapid in development, and they are apt to be somewhat masked by the accompanying disease. Bedsores develop rapidly if the disease lasts long enough.

The only form which affords opportunity for treatment

is that in which there is an accessible focus of inflammation which can be treated surgically. Trephining and free drainage of the vertebral canal is indicated. The general treatment follows that outlined for cerebro-spinal fever.

IV. *Tuberculous Meningitis.*—This form properly occupies a place between acute and chronic meningitis, for it may be acute, subacute, or chronic. It is divided by some authors into primary and secondary, but the primary cases rest on a very infirm base. Councilman has never seen a primary case, and Osler considers such cases very doubtful. A careful search will in almost all cases reveal an old tuberculous focus, if not in the lungs then in lymphatic glands, bone, or even in the middle ear. Jacobäus attributed a case of tuberculous meningitis of the cauda equina to extension from a tuberculous endometritis.

The process is more marked in the cerebral than in the spinal membranes, and the symptoms of spinal meningitis may be masked entirely, yet it is probable that in all cases the spinal meninges are also involved. Lichtheim found tubercle bacilli in the fluid obtained by lumbar puncture in all the cases of tuberculous meningitis examined in his clinic (figures not given); Fürbringer in twenty-seven out of thirty-seven cases. Oppenheim, Heubner, and Leyden and Goldscheider consider extension to the spinal meninges almost invariable. Osler reports a case of pure spinal meningitis (tuberculous). The localization of the process in the membranes at the base of the brain has led to the name "basilar meningitis," the large amount of fluid exudate to the name "acute hydrocephalus" or "water on the brain." It is much more common in children than in adults, but is rare during the first year of life; more common from the second to the fifth years.

The membranes at the base of the brain are cloudy, and covered with an exudate which is serous or gelatinous, or semipurulent, and causes matting of the membranes. There may be numerous tiny white tubercles scattered throughout, or they may be very few and revealed only after a careful search. They tend to form along the small pial arteries, and examination of the choroid plexus may reveal them, or it may be necessary to make a careful dissection of the branches of the middle cerebral arteries, when they will be found along the sheaths of the smaller vessels. Though more fully developed in the basilar region, the tuberculous process spreads to the vertex and to the vertebral canal. The abundant exudate—which is no measure of the extent of the tuberculous process—may cause flattening of the convolutions of the brain and enormous distention of the ventricles. Lichtheim says that the fluid is generally clear, but a delicate veil of fibrin often appears on standing, and is apt to contain in its meshes tubercle bacilli.

The tubercles are miliary, but have a tendency to early caseation, which is found even in the rapid cases—those of nine or ten days. In the slower cases there is an extensive confluence of the tubercles with formation of large caseous masses, in which the bacilli are found in great numbers. Giant cells are not common. Hektoen has described a peculiar tuberculous endarteritis which is not an extension from without, but due to an implantation of bacilli from the blood. The dura may escape entirely, or may show simply congestion. Ophüls describes tubercles of the ependyma either deep, from infection through the lymphatics, or superficial, from infection through the cerebro-spinal fluid.

The prodromal stage is protracted as compared with that of purulent meningitis, and the disease follows well-marked stages. The symptoms of the prodromal stage are headache, listlessness, irritability, constipation, loss of appetite, more or less insomnia. These may pass gradually or suddenly into the stage of irritation: severe headache, vomiting, fever, often convulsions. Headache is apt to be intense and agonizing, requiring powerful sedatives and being sometimes uncontrollable. The temperature rises rather gradually and is not high, 100°–103° F., the pulse increasingly irregular. In this stage the pupils are contracted, and there are muscular twitchings or spasms. The next stage is marked by a subsidence of the irritative symptoms, the headache is less severe, the head retracted, the patient is dull and stupid, the eyes half closed, the eyeballs moving slowly from side to side, or there may be strabismus, the pupils dilated. Sudden flushes may appear over limited areas and redness follows rubbing or passing the nail quickly over the skin (the tache cérébrale). Convulsions may occur in this stage, which passes over into the stage of paralysis, characterized by a low delirium or coma, with dilated pupils, a weak, rapid pulse, a subnormal temperature, and sometimes paralysis of face or limbs. Death occurs in two to three weeks, sometimes preceded by a sudden rise of temperature.

The type described is that usually seen in childhood. In adults the process is extremely variable. Chantemesse, Boix, and Jaccoud all emphasize the great difference between the form in childhood and that in adult life, Jaccoud, indeed, asserting that the fatal outcome is the only common symptom. Jacobäus, however, explains this partly on the ground that it is the unusual cases only which find their way into the literature. The onset may be very sudden and the disease fatal within a few days. A case of Heubner's—adult—was at work up to thirty hours before death. He was seized with convulsions, delirium, and finally lethal coma. In other cases the disease may apparently begin in the spinal membranes and affect the cerebral secondarily. In a case of Jacobäus', a pulmonary consumptive, the symptoms began in the legs and travelled up. Boix reports a case which simulated tetanus, being ushered in by trismus.

The diagnosis of tuberculous meningitis depends upon the discovery of a tuberculous focus elsewhere in the body, the long prodromal stage, the comparatively low temperature, the marked irregularity and slowness of the pulse, and the more gradual development of the distinctive symptoms of meningitis than occurs in the purulent form. Ophthalmoscopic examination may reveal tubercles of the choroid. The value of Quincke's lumbar puncture has already been emphasized.

The disease is almost invariably fatal. Von Leube reports one recovery. A young phthisical adult had symptoms of tuberculous meningitis, recovered, suffered an exacerbation of the symptoms in the lungs, and succumbed to a second acute attack of meningitis. Examination of the spinal membranes showed evidence of healing tubercles. Reinhold had a case which followed a similar course. In the fluid obtained from a case of supposed epidemic meningitis Freyhan found tubercle bacilli, but the patient recovered. Osler has never seen a case diagnosed as tuberculous recover, nor has he found evidence of past disease at autopsy. *Alice Hamilton.*

SPINAL-CORD DISEASES: CERVICAL HYPERTROPHIC PACHYMENINGITIS.—The first adequate description of this rare disease was given by Charcot in a communication to the Société de Biologie in 1871. Two years later it was made the subject of a thesis by Joffroy, and since that time all writers on nervous diseases have associated the names of these two observers with classical descriptions of this form of meningitis.

Pathologically the disease is characterized by a chronic inflammation beginning on the inner aspect of the dura, and resulting in an exuberant stratiform overgrowth of fibrous tissue. As a result the dura may attain to a thickness five to ten times greater than the normal. The spinal roots traversing the thickened meninges are at first irritated and finally more or less completely strangled by the fibrous overgrowth. Later on, the cord itself shares in the morbid process, either by mechanical compression or by actual invasion, in the latter case the inflammation creeping in along the pial septa and blood-vessels. The ultimate result is a welding together of membranes, roots, and cord in a dense, stratified mass of fibrous tissue which may undergo partial ossification. Vacuolation of the cord has been said to occur as an end result in certain

cases, and is probably due to the retraction of the fibrous tissue.

The disease usually begins in the dura surrounding the lower portion of the cervical cord, and produces a fairly constant clinical expression of its presence; it may, however, invade the pons and medulla or involve the upper dorsal region.

From an etiological standpoint nothing definite is known about the disease. Charcot speaks of it as accidental, having nothing to do with the family or hereditary types of nervous affections. Exposure to cold and damp seems to be the chief causative factor, although traumatism is accorded its usual place in the list, probably for the sake of uniformity. Oppenheim thinks that syphilis is a very common if not a prime cause; in the opinion of the writer the disease is much too rare to warrant this view.

The symptomatology has been somewhat arbitrarily divided by Charcot into three periods corresponding to the three stages of pathologic development. In the first—neuralgic or pseudo-neuralgic period—there is very severe pain in the neck, in the back of the head and between the shoulders, and sometimes in the top of the chest. With this there is a feeling of tension or stiffness, and the cervical spine may be tender to percussion. In addition, paræsthesiæ and neuralgic pains appear in the regions supplied by the ulnar and median nerves—the radial practically escapes—and are accompanied or followed by tremor and a certain degree of muscular tension. This stage of the disease may last for from four to six months, and is usually followed by the second or paralytic period.

At the beginning of this second period the pain has practically subsided. Paralysis of neuritic origin has, however, succeeded. This paralysis corresponds chiefly to the distribution of the ulnar and median nerves, the radial practically escaping. The muscles mainly involved are the small muscles of the hand and the flexors of the hand and fingers. As a result the unantagonized action of the extensor group brings about a peculiar attitude of the hands—hyperextension at the wrist-joint, with extension of the basal and flexion of the middle and end phalanges. This is known as the preacher hand (*main de prédicateur; Predigerhand*) and is practically pathognomonic. The reason for the exemption of the radial nerve from participation in the morbid process is not obvious, since its roots certainly lie within the diseased dural territory. It is curious also that mention is not oftener made of an implication of the cilio-spinal fibres of Klumpke-Dejerine arising from the first dorsal segment of the cord, with consequent changes in the pupil and in the palpebral fissure.

The third symptomatologic period is not always clearly to be separated from the others. In it appear the signs of a break in the conductivity of the cord, the result of strangulation or infiltration. This is evidenced by spastic paraplegia, anæsthesia, and loss of control of the vesical and rectal sphincters. These symptoms are produced in part by the local compression or asphyxiation of the cord, as has been stated, and in part by the secondary degenerations which naturally result therefrom.

As a rule, the disease extends over a period of years. It may, however, come to a standstill at any stage, leaving permanent damage which corresponds in degree with the amount of cord or nerve destruction that has taken place, or allowing complete restitution to the normal. Fatal cases are recorded.

When the disease process occurs in the classical situation, the lower portion of the cervical enlargement, its clinical features are sufficiently characteristic to determine the diagnosis. Obscurity comes when there is bulbar or pontine involvement or when the upper dorsal cord and roots are mainly implicated. An early cervical Pott's disease may be mistaken for cervical hypertrophic pachymeningitis; in such a case the obvious implication of the vertebræ themselves, which occurs sooner or later in the course of Pott's disease, should serve to differentiate. From tumors starting in the meninges or from the cord itself and growing vertically, differentiation is very difficult if not impossible; here the rarity of the disease under consideration should influence the decision.

The therapeutic measures most indicated are hot baths, the local application of the actual cautery over the lower cervical vertebræ, and the internal administration of potassium iodide and mercury, the last two being of value even in cases which are not specific in origin. In a case of Remak's galvanism rendered good service. Surgical measures are to be avoided. *Joseph W. Courtney.*

SPINAL-CORD DISEASES: CHRONIC SPINAL MENINGITIS.—Localized areas of chronic meningitis accompany chronic disease of the bones of the spinal column, tumors, chronic inflammatory diseases of the cord, etc. It is practically impossible to separate chronic pachymeningitis from chronic leptomeningitis. The process may predominate in one membrane and the symptoms may vary accordingly, but the other is invariably involved to a greater or less extent. Chronic productive inflammation of the pia-arachnoid and dura mater is very common, especially in old age; it is associated with trauma, atrophy of the cord and brain, chronic alcoholism, and general paralysis. It is usually accompanied by hemorrhage into the newly formed tissue and is therefore denominated pachymeningitis interna hæmorrhagica. In the early stages the inner surface of the dura mater is covered with a fine, easily removable, veil-like membrane consisting of a fibrinous network with spindle-shaped connective-tissue cells and leucocytes. Gradual organization of this membrane takes place by the formation of connective-tissue fibres, and the outgrowth of vascular buds which form a rich vascular network. These thin-walled vessels are very prone to rupture, so that the membrane becomes studded, at first with hemorrhages, later with masses of blood pigment from the disintegrated corpuscles. The outer part, next the dura, grows sclerotic, and is inseparable from the dura itself. Large hemorrhages may occur—hæmatoma of the dura—with symptoms of compression. The early stages may be found at autopsy, in acute infectious diseases, without having caused symptoms during life.

Undoubtedly many cases of so-called "chronic meningitis," especially those of the earlier literature, were really diseases of the cord, for chronic spinal meningitis is rare as a primary malady, and our knowledge of it is as yet slight and defective. The clinical picture differs widely from that of the acute, for the symptoms of irritation are replaced by those of pressure. The nerve roots seldom escape in this form. Usually they are red and swollen or compressed and atrophied. The cord suffers to a variable degree, most extensively where the process is pronounced in the pia-arachnoid, and the newly formed tissue not only compresses the nerve roots, but passes in along the sheaths of the vessels, causing a marginal sclerosis and a general infiltration of round cells and epithelioid cells. Degenerations following this sclerosis cause the symptoms of a mixed lesion, a meningomyelitis. Usually the changes are localized, especially in the cervical cord; very rarely they are general, as in a case reported by Mitchell Clarke, in which the pathological changes and new formation of connective tissue rich in blood-vessels involved the inner surface of the dura and the pia-arachnoid throughout the whole extent of the cord.

The symptoms of chronic meningitis are due, as in the acute form, to irritation and compression, but in the former there are not such severe paroxysms or spasms, and the symptoms increase gradually, at times with long remissions. Pain or stiffness in the back and neck, with radiating pains in the limbs, simulate rheumatism. This is succeeded after a long period by anæsthesia, with hyperæsthetic patches. The muscular symptoms are tremors, gradual loss of power, and wasting. The reflexes are lost. The symptoms vary naturally with the localization of the lesions. Those of the cervical cord are muscular atrophy and paresis of the upper extremities with

spastic laming of the lower, and are very similar to those of amyotrophic lateral sclerosis or syringomyelia, except for the more pronounced pain and stiffness.

The form known as pachymeningitis cervicalis hypertrophica was first differentiated by Charcot and Joffroy, who claimed for it a special symptomatology. The thickening of the dura is most pronounced in the cervical cord, where it forms a ring of dense fibrous tissue, with adhesions to the bony canal and to the pia-arachnoid. The outgoing nerve roots are compressed and show degeneration with proliferation of interstitial tissue. Pressure on the periphery of the cord causes a marginal sclerosis which is followed by ascending and descending degenerations. The proliferation of new tissue may be so extensive as to flatten the cord antero-posteriorly.

Charcot divides the symptoms into a first period lasting two or three months, with severe pains in the back of the neck passing up to the occiput and down to the arms, persistent, with exacerbations. There are rigidity of the neck and back, simulating Pott's disease, paræsthesia in the arms, perhaps paresis. This corresponds to the stage of compression of the nerve roots, while in the second stage there are the symptoms of degeneration, the pains cease, there are laming and atrophy, especially of the muscles supplied by the ulnar and median nerves. There are laming and contraction of the muscles of the legs, also general cutaneous anæsthesia, decubitus, and there may be paralysis of the sphincters.

The prognosis is grave, for the disease is usually regularly progressive, and a recovery probably speaks for a wrong diagnosis. Rest is essential; warm baths and counter-irritation, by stimulating liniments or sinapisms, may be employed. Joffroy recommends the use of the thermo-cautery for the hypertrophic form. Mercury and iodide of potassium are recommended, the former by inunction over the spine, and combined with cantharides for counter-irritation. In using anodynes it is important to bear in mind the long course of the disease and the importance of frequently changing the drug and of trying to reduce the dose occasionally. Galvanism, massage, and passive movements are indicated for the muscular wasting.

Syphilitic Meningitis.—It is very probable that many of the cases which are diagnosed as chronic meningitis are syphilitic in origin. The most common change found in the spinal cord in syphilis is meningomyelitis, which generally affects the membranes of the brain as well. Williamson states that in thirty-two cases of spinal syphilis examined by him, sixteen were meningomyelitis, three were chronic meningitis. The syphilitic process shows itself in the vessel walls especially, although the weight of opinion is against a specific syphilitic arteritis. A general arteritis, however, in young individuals is apt to be syphilitic. The dura is diffusely infiltrated and thickened, there may be typical gummata with caseous centres, but more frequently there are only local accumulations of new connective tissue. The pia may in some places exhibit five to ten times its normal thickness, being gelatinous or densely fibrous, and adherent to the dura. The changes in the membranes extend to the cord in the form of proliferation of the connective tissue along the sheaths of the vessels and of the neuroglia. The changes in the vessels are proliferation, sometimes most marked in the intima, sometimes in the adventitia, with thrombosis and obliteration of the smaller vessels. Japha, Schwarz, and Wullenweber report cases of syphilitic meningitis accompanied by cavity-formation in the cord, the cavities being caused probably by necrosis of anæmic infarcts from the blocking of the sclerotic vessels. This condition is not to be confused with syringomyelia, for there is no trace of gliosis around the cavities.

Chronic syphilitic meningitis can cause any of the symptoms outlined under chronic meningitis in general, depending on the localization of the fibrous proliferation. Typically, it is focal in character and associated with focal lesions in the cord itself, so that the pains are usually irregular, unsymmetrical, followed soon by impairment of sensibility and muscular weakness. The pain is said to be worse at night. The rise of temperature is not great—100°–101° F.—and may be subnormal at the end. In Shoyer's case the symptoms began in the left foot and moved gradually upward, the disease lasting over four years.

The prognosis is more favorable than that of any other form of chronic meningitis; the treatment follows the same lines as that for syphilis in general.

Alice Hamilton.

SPINAL-CORD DISEASES: COMBINED DEGENERATIONS OF THE CORD.—Introduction.—The attitude toward the so-called combined scleroses of the spinal cord has changed with increasing post-mortem study and with the general acceptance of the neurone doctrine as applied to pathological processes. The term "system or systemic disease," as formerly applied by Charcot and others, should be modified to include only degenerations of neurones, "systemic neurone disease," whereas the various degenerations, involving chiefly the white matter of the cord, irrespective of neurones, should be differently classified. A failure to recognize this fact, even on the part of recent writers, has led to a very considerable degree of confusion in the nomenclature, especially since lesions of varying extent and character may give rise to very similar clinical symptoms. With the more accurate study which the new conception of the anatomy of the nerve cell has rendered possible, and with a large number of post-mortem investigations, it is apparent that the "combined scleroses" are more frequently diffuse in distribution than limited to fibre tracts of functional identity. If, then, the term "combined sclerosis," or "combined disease of the dorsal and lateral tracts," be used, it should be understood that there is no implication that the neurones as such are primarily involved, although the lesions may lie, in great part, in the lateral and dorsal portions of the cord. Doubt has been thrown upon the existence of a true systemic degeneration of the peripheral sensory neurones (dorsal tracts), and the central motor neurones (pyramidal tracts) in the form of a combined disease (Goldscheider), except in certain hereditary affections (Friedreich's ataxia, cerebellar ataxia) and in dementia paralytica. It is certainly far less frequent than formerly believed. In the following discussion, therefore, I shall place chief stress upon diffuse lesions of the cord, predominantly located in the dorsal and lateral tracts, and giving rise to the symptoms of ataxic paraplegia. This in no way denies the possibility of the existence of combined disease which is actually systemic in character.

The following provisional groups may be made:

I. Diffuse (combined) degeneration of the cord; combined sclerosis.

II. True combined dorsal and lateral systemic (neurone) disease.

III. Combined systemic disease. (*a*) Result of congenital defect (Friedreich's ataxia, cerebellar ataxia). (*b*) In dementia paralytica.

I. *Diffuse (Combined) Degeneration of the Cord; Combined Sclerosis; Subacute Spinal Ataxia; Subacute Combined Degeneration of the Spinal Cord; Combined System Disease.*—Under these varied titles has been included a type of affection which is sufficiently characteristic to have a definite place among the diseases of the spinal cord. Attention has been drawn to the combination of sclerosis of the dorsal and lateral tracts by Kahler and Pick, Westphal, Strümpell, and Oppenheim; the association of such alterations with pernicious anæmia and cachectic states has become familiar through the work of Lichtheim, Putnam, Dana, Minnich, Nonne, Burr, Bastianelli, Russell, Batten, Collier, very recently by Billings, and many others. The identity of the lesions in these various conditions is still in dispute; that they vary in detail is of course natural and inevitable; that they represent a general group of alterations which may later be subdivided is, however, at present a supposition, justified by the facts at our disposal. No attempt at subdivision—as, for example, lesions due to anæmia or lesions

dependent upon general cachexia or toxæmia—will be attempted in what follows. Anæmia has undoubtedly been given a place of undue importance in relation to these lesions. The insistence upon other and more general causes is due primarily to Putnam's work, published in 1891.

Pathological Anatomy.—The alterations in the cord in this type of disease consist macroscopically in degenerations of the white matter, chiefly in the dorsal and lateral tracts, irregular in distribution, and either not involving whole groups of fibres or extending beyond the confines of such tracts. The dorsal tracts, for example, are affected irregularly, groups of fibres in the immediate neighborhood of much degenerated areas are spared, and the root zones are rarely involved, in striking contrast to the systemic degeneration of tabes. The region of the pyramidal tracts is often much involved, but the alterations always extend beyond the confines of these tracts, chiefly along the peripheral portions of the cord, and on either side of the ventral fissure. The alterations are least marked in the lumbar region, and increase markedly in extent in the upper thoracic and cervical regions. Even to the naked eye the stained section often presents a peculiar, vacuolated appearance. Microscopic examination of the lesions shows the alterations to be essentially limited to the cord, with exceedingly slight alterations of the oblongata or hemispheres. The lesions are characterized by disintegration of myelinated fibres, overgrowth of neuroglia, in part sufficient to form a dense sclerosis and in part slight, vacuolation being due apparently to dilating and degenerating myeline sheaths, many fat granule cells, irregularity of distribution, often with very small foci of degeneration, scattered irregularly through the white matter, and at times focal softening. The gray matter is very slightly involved, and frequently not at all; the nerve roots are not involved; there is no evidence of meningeal thickening, and the blood-vessel walls rarely show changes. The pathological features common to the general group may, therefore, be summarized as follows:

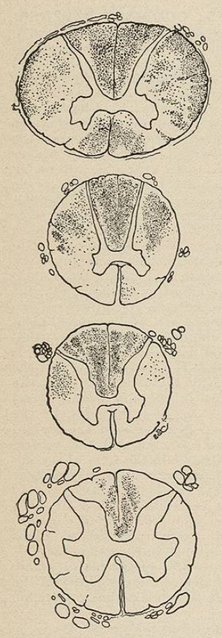

Fig. 4411.—Diffuse (Combined) Degeneration of the Spinal Cord.

1. A diffuse degeneration for the most part limited to the cord, often in more or less discrete patches.
2. A constant involvement of the dorsal and lateral columns, without strict regard to neurone systems.
3. A predominance of the lesion in the cervical and thoracic regions.
4. The common freedom from degeneration of nerve roots, both motor and sensory, and peripheral nerves.
5. The essential non-involvement of gray matter.
6. Insignificant vessel changes.

The reason for the peculiarly constant location of the lesions is probably to be sought in the distribution of the blood supply to the cord, a theory first advanced by Marie. Those who cling to the systemic character of the affection are inclined to search for a more subtle explanation, but the involvement of the cord alone, the tendency toward a peripheral distribution of the lesions, the non-involvement of gray matter, and the frequent occurrence of the disease in impoverished blood states, all indicate the rôle of the blood-vessels and their contained blood. Certain writers have maintained a double lesion, one of diffuse character and the other systemic, with possible secondary degenerations.

Etiology.—The persons in whom the disease occurs in typical form are usually of middle age, weak constitution, often with neurotic personal or family antecedents, of poor nutrition, cachectic in appearance, and of reduced bodily vigor. It is considerably more frequent in women than in men, according to Putnam's statistics. Syphilis may be disregarded as an etiological factor. A frequent though by no means universal accompaniment or possible cause of the disease is anæmia of the pernicious type. This association has led to the popular but wholly erroneous assumption that the alterations of the spinal cord described above are peculiar to anæmia. Lead poisoning, malaria, grave secondary anæmia, and other debilitating influences have also been given as causes, and the disease has at times occurred in otherwise healthy individuals. Overwork, excessive anxiety, gastro-intestinal disorders, may have a predisposing influence. Further than this our accurate knowledge does not go. Whether or not a subtle toxæmia, of a character as yet not understood, may ultimately be found responsible is possible, but far from being proved. It may be said with assurance at the present time that a well-characterized affection of the cord occurs in cachectic individuals toward middle life, leading to diffuse, quasi-systemic degenerations, dependent, in part at least, upon the distribution of the vascular supply.

Symptomatology.—In general, during the early stages, the symptoms are somewhat vague in character and may simulate functional disorders. The most conspicuous early symptom is paræsthesia of the extremities, noticed earlier in the feet. Disordered sensations may occur in other portions of the body—genitals, distributions of cranial nerves—but are much less usual. This paræsthesia is frequently very distressing. Slight disorders of micturition and rarely of defecation may occur. Objective disorders of sensation are not conspicuous, though they certainly occur, and in a well-reported group of cases (Russell, Batten, Collier) take an important place in the later stages of the clinical picture. Disorders of motion are of the character of a slight spastic paraplegia, with exaggerated deep reflexes, and the Babinski phenomenon. As the disease progresses, the symptom-complex assumes the general character of an ataxic paraplegia, with a predominance of ataxia or paraplegia depending upon the greater involvement of the dorsal or lateral columns. In later stages, as the lumbar portion of the cord is invaded, the knee-jerk may be abolished, and the patient be reduced to a helpless paraplegic condition, with loss of sphincter control. The mind is rarely affected, and the cranial nerves show no noteworthy involvement. Pigmentation of the skin, epileptic attacks, excessive diarrhœa or constipation, atrophy of the optic nerve, mental instability, dissociation of sensation, pain in the back and limbs, transient œdema, muscular atrophy, altered electrical reactions, have all been described as occasional symptoms.

Certain writers, notably Bastianelli and Russell, Batten and Collier, have attempted to classify cases either by etiology (Bastianelli) or by symptomatic course (Russell, etc.). The latter divide the disease sharply into three stages: first, slight ataxic paraplegia, constituting one-half to three-quarters of the duration of the illness; second, sudden loss of sensation, with increasing motor paresis, leading to inability to walk or stand, marked spastic paraplegia; third, stage of flaccidity, flaccid paraplegia, complete flaccid motor paralysis, complete anæsthesia, with loss of deep reflexes, incontinence, wasting

of muscles, and loss of faradic reaction. In the experience of others these sharp divisions cannot be maintained; such a course evidently marks a variety of the general affection, which is highly important, but cannot be regarded as characteristic of the entire group. In general, it must be said that on the symptomatic side attempts to deduce a perfectly characteristic clinical picture have as yet failed, although the grouping of the symptoms of primary paræsthesia, ataxic paraplegia increasing rather rapidly in intensity, followed by a stage of more or less complete motor and sensory paralysis, in the absence of lancinating pains and Argyll-Robertson pupil, point strongly toward combined lesions of the character under consideration.

Diagnosis, Course, and Prognosis.—The diagnosis in the later stages should not be difficult, and in the early stages should be assumed as probable in cases in which an otherwise unexplained and persistent paræsthesia, especially of the legs, is associated with slight ataxia and a tendency toward spasticity with exaggerated deep reflexes, particularly if the patient be of middle age, with evidences of cachexia. Multiple sclerosis may simulate this affection at various stages, but in general it may be distinguished by the youth of its victims, the characteristic speech defect and tremor, with a marked preponderance of motor over sensory involvements. Myelitis should also receive consideration in the differential diagnosis. Uncomplicated tabes should give rise to no confusion.

The course of the disease is from six months to two years, with occasional instances of much longer duration. As a rule, the patients are already in a debilitated condition when the symptoms manifest themselves, a fact which evidently must have a more or less direct bearing upon the outcome.

The prognosis is always bad, both from the point of view of the general condition of the patient and also because of the usually rapidly advancing cord degenerations.

Treatment.—Attention should first be directed toward the discovery and amelioration of the underlying condition causative of or associated with the alterations in the cord. Anæmia, whether of the so-called pernicious variety or secondary, general debility, and disordered bodily functions in general, should be treated by the recognized means at our disposal. It is possible that much might be done in the earliest stages to avoid or modify the serious and ultimately irremediable cord degenerations. If they have developed, however, and the patient is ataxic, or ataxic paraplegic, recourse should certainly be had to the Frenkel exercises, modified to meet the indications in individual cases. The usual drugs—iron, strychnine, arsenic, the iodides, etc.—may be given, but with small hope of permanent benefit.

II. *Combined Dorsal and Lateral Systemic (Neurone) Disease.*—In spite of the doubt which has been thrown upon the existence of a true neurone degeneration involving dorsal and lateral tracts, cases have been reported clinically, and in a few instances anatomically verified, of the existence of such a combined lesion (Oppenheim). Degenerations of the columns of Goll and Burdach, the pyramidal tract, the direct cerebellar tract, and less frequently of Gowers, and the uncrossed pyramidal tracts, have been described, with the assumption that these tracts were degenerated as systems and not as a part of a more general process.

Such cases of primary degeneration of lateral and dorsal neurone systems give rise to the symptoms of tabes and lateral sclerosis, the one or other type of symptom predominating in direct relation to the extent of the dorsal or lateral pathological change. Symptoms of tabes, with lancinating pains and Argyll-Robertson pupil, but with exaggerated knee-jerk, should always give rise to a strong suspicion of a combined systemic degeneration, giving the general clinical picture of ataxic paraplegia. Should the definite signs of tabes, including loss of knee-jerk, be associated with muscular weakness, a similar diagnosis of combined lesion is justified. The signs of tabes, on the other hand, may be in great measure subordinated to those of lateral degeneration—spastic paraplegia. In this case slight disorders of sensation, sharp pains, condition of the pupils, bladder weakness, should be carefully investigated to determine the possible involvement of sensory neurones. Ataxic paraplegia, as commonly observed, is no doubt usually due to the more diffuse process described above. The important differential point is the existence of a true tabes, which does not occur in the diffuse combined lesions.

The treatment is as indicated for the foregoing disease, and for Friedreich's ataxia.

III. *Combined Systemic Disease.*—(*a*) *Result of congenital defect (Friedreich's ataxia); cerebellar ataxia; hereditary cerebellar ataxia;* (*b*) *in dementia paralytica.*

(*a*) *Friedreich's Ataxia.*—See article on this subject.

Cerebellar Ataxia.—Hereditary cerebellar ataxia, type Nonne-Marie, bears a close resemblance to Friedreich's ataxia, except for the facts that the knee-jerks are increased, the pupils are immobile, there is atrophy of the optic nerve, there are marked disorders of sensibility, and club-foot and kyphosis do not develop. Lesions of the cerebellum and other portions of the brain have been found, and in one case (Menzel) a combined system disease of the cord.

(*b*) *Combined Systemic Disease in Dementia Paralytica.*—Alterations of the spinal cord (Westphal), in the form of degeneration of the pyramidal tracts or of the dorsal, sensory tracts, or of both, are frequent in dementia paralytica. Whether the alterations in the pyramidal tracts are to be regarded as primary neurone degenerations or as secondary to changes in the cortex remains in doubt.

Gowers' Ataxic Paraplegia.—In 1886 Gowers drew attention to a condition presenting the symptoms of degeneration in the dorsal and lateral tracts, to which the clinical name ataxic paraplegia was given. The classification of this condition is difficult, but from the description of the pathological alterations it should rather be included among the diffuse combined degenerations than among the true systemic diseases. After what has been said it does not require separate description.

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SPINAL-CORD DISEASES: CONGESTION OF THE CORD.—Our actual knowledge regarding spinal congestion is very limited, and hypothetical statements that it is the basis of many nervous symptoms are unwarranted. The diseases in which it is uniformly found after death are those in which the patient has died in convulsions complicated by asphyxia, or in the early stages of myelitis. The only positive evidence that a spinal congestion has existed during life is the discovery of distended capillaries, accompanied by small capillary hemorrhages. Without the latter the congestion found may have been a post-mortem occurrence, due to the position of the body on the back.

The *causes of active congestion* are excessive muscular exertion, violent sexual excesses, poisoning by strychnine, alcohol, and carbonic oxide, the sudden arrest of menstruation or the stoppage of hemorrhage from piles, and possibly exposure to cold. Traumatism of the vertebræ, especially general concussion of the spine, such as