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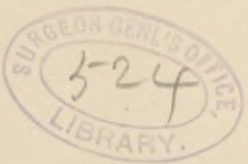

CARIES OF THE SPINE
FOLLOWED BY COMPRESSION OF THE CORD.*

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LECTURE I.

The patient before us is afflicted with bone and nervous lesions, which, unfortunately, are too common in every community, owing to the lack of thoroughness in the examination of patients suffering from the incipient stage of caries of the spine, and the failure to comprehend the gravity of the disease when recognized, and to insist upon prompt and effective measures for its arrest before irreparable damage has been done to the spinal cord and nerves.

The history of the present case is typical of the vast majority of this class, and is as follows:

Mrs. M. Mc., twenty-five years of age, born in Austria, accustomed to service, been in Colorado four years, during which time she has been married, was admitted into the nervous wards

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of the Arapahoe County Hospital, April 12, 1893. Owing to her very limited knowledge of any language except the dialect of her own native province, it was impossible to get a connected history of her case through the aid of any of the foreign-born patients in the hospital.

So far as I was able to learn, her family history presented nothing of importance except the probability of tuberculosis in some of the remote and immediate relatives. During her childhood she enjoyed good health. During the latter part of 1892 and early in 1893 she complained of pain in the lumbar region of her spine, and the pain radiated around the abdomen, and sometimes the pains were felt in the loins and legs. In January, 1893, she became pregnant, and soon after this the spinal pains increased. By April 12th, the time of her first admission to the hospital, her legs were so weak that she was unable to stand. At that time a complete record of her condition was not made, as she remained in the hospital only a few days and left the morning of the day set for a thorough examination. From some notes made at the time I first saw her—the day after she was admitted to the hospital—I find that the legs were almost completely paralyzed; she had imperfect control of the vesical and anal sphincters; the knee jerks, ankle clonus, and lower abdominal reflexes were absent; the plantar reflexes were exaggerated, with hyperæsthesia of the plantar surfaces of the feet; the epigastric reflexes were present; the dorso-lumbar region of the spine was deformed, sensitive, and the seat of constant pain, which was increased by movements of the trunk, and the sensory phenomena in the legs and lower portion of the body were perverted with areas of hyperæsthesia and anæsthesia, but the degree and extent of the sensory disturbance I did not ascertain. She left the hospital presumably because absolute rest in the recumbent posture until the bone trouble should be arrested was insisted upon. Nothing further was heard from her until she was readmitted into the nervous wards of the hospital, October 1, 1893. During this time her spinal trouble had gradually increased, and in the early part of August, two months before her readmission to the hospital, she had given birth to a child. Examination, October 3d, showed

absolute paralysis of all the muscles of the legs and hips. There was double foot-drop, and the leg muscles showed some flaccidity, more on the right than on the left side. There was partial paralysis of the vesical sphincter. At times, the water would flow from the bladder as fast as it found its way into it, and at others the water would accumulate in the bladder until several ounces were retained, when there would occur an involuntary evacuation of the bladder without her knowledge. The anal sphincter was completely paralyzed. The abdominal muscles were partially paralyzed and the abdomen distended with gas.

Knee jerks: Right, a slight reflex could be obtained only when the leg was flexed at right angles to the thigh. Left, present and slightly exaggerated. Plantar reflexes absent. No ankle clonus. Lower abdominal reflexes absent; epigastric, present.

All sensory phenomena were absent from a point in front about two inches above the umbilicus, and posteriorly from a point opposite the spine of the first lumbar vertebra downward. She could not tell when the bladder or bowels were evacuated, nor feel a catheter when passed. Well-marked deformity existed in the dorso-lumbar region of the spine, with arching of the spinal column forward and projection of the spines backward. The deformity included the spines of the eleventh and twelfth dorsal and the first lumbar vertebræ, the greatest deformity existing over the twelfth dorsal spine. The whole region was tender to pressure and the seat of pain.

Soon after her second admission to the hospital she had a severe chill, followed by a rise of temperature lasting three or four days, and ranging from 102° to 104° F., during which time she complained of great pain just to the right of the spine in the lower thoracic region. The part was sensitive to manipulation and the formation of an abscess was suspected. Similar attacks were repeated about once in two weeks for a period of two months. During this time she became very weak and emaciated considerably. No positive evidence of an abscess was manifest, and with free stimulation and the administration of strychnine, iron, cod-liver oil, and nutritious food she eventu-

ally began to improve. Abrasions of the skin took place over the sacral region on several occasions, but by means of careful dressing a bedsore was prevented. About January, 1894, her general condition was fairly good, and improvement in bone and cord symptoms became manifest. By March she had regained control of the vesical and anal sphincters and the anæsthetic area was very much lessened. By June, no area of complete anæsthesia could be found, she was able to sit up for a short time each day, could flex and extend the feet, and move the legs at the hips and knee joints. All pain and tenderness in the spine had disappeared. Since then there has been a slow and gradual improvement.

To-day we find her nutrition excellent. The muscles of the legs and feet are fairly strong and not much wasted. There is cutaneous hyperæsthesia over most of the area of former anæsthesia. There is no loss of tactile or any other form of sensation in any portion of the body. Knee jerks are present, the left being slightly exaggerated. Plantar reflexes are very much increased. No ankle clonus. Lower abdominal reflexes absent; epigastric, present. She can support her weight on her legs, but the effort causes painful contraction of the extensor muscles of the legs and thighs. At times, when lying quietly in bed, she experiences painful contractions of these muscles. She also suffers from radiating pains which extend from the dorso-lumbar region of the spine along the lower portion of the chest, around the abdomen, into the groins and down the legs.

A discussion on the diagnosis of this woman's trouble is unnecessary, as the symptoms of bone and cord lesion are so obtrusive that caries of the spine and compression of the cord, with resulting myelitis and subsequent degeneration, are evident. On account of the importance of an early diagnosis and prompt and efficient treatment in these cases, I desire to consider with you somewhat in detail the two principal morbid processes (caries of the spine and compression of the cord) from which this patient is suffering.

Caries of the Spine.—Caries of the spine, in proportion to the number of inhabitants, seems to be much more frequent in Colorado than in the eastern or southern portions of this country. There are two reasons for this: One is that a larger percentage of our population follow dangerous occupations, especially mining, in which they are peculiarly subject to injuries of the spine, and the other is that a goodly number of those who reside in Colorado are suffering from chronic tuberculosis. Tuberculosis and traumatism to the spine are the two chief causes that lead to caries of the bones of the spinal column. In regard to sex, the cases which I have observed in Colorado show that males suffer about four times as frequently as females. This seems to be due almost entirely to the character of the injuries to which our male population is exposed. In most countries males suffer more frequently than females, but the difference between the two sexes is not nearly so great as we find it in mining countries. Caries is usually most common in childhood after the third year, and next in young adults; but in Colorado we find an exception to this rule, as adults from the twentieth to the forty-fifth year are by far the most frequent sufferers. If I may judge from my own inexperience, it is a comparatively infrequent malady in childhood in Colorado. I wish to remind you that the disease may occur at any time of life. I have met with a case in a woman sixty-five years old. Gowers has observed it at fifty, and says it has been met with as late as the seventieth year. The author just referred to thinks that it is the most common form of the manifestation of scrofula during the second half of life. Caries may follow injuries to the back in which the bones have not been directly injured, but inflammation may extend to the periosteum and bone substance from the torn and inflamed ligaments. Caries of the spine following traumatism that

has not fractured the bones may come on several months after the receipt of the injury, and in some instances after all symptoms of the injury have passed away. It is probable that fracture of the bones of the spine in which there is no displacement may terminate in caries several months after the accident. Syphilis plays an uncertain rôle in the causation of caries. Adjacent inflammation, abscess, and blood poisoning may give rise to disease of the vertebræ.

The inflammation resulting in caries affects the intervertebral substance, the periosteum and the bodies of the vertebræ, and at times their processes. The inflamed structures are swollen and vascular, and later the bodies of the vertebræ soften and break down under the superincumbent weight, resulting in deformity of the spinal column. The spines occasionally become thickened from the results of periostitis.

The soft structures in the spinal canal external to the dura and those immediately surrounding the external surface of the inflamed bones are often the seat of inflammation. The areolar tissue between the dura and the bone is absorbed; the inflammation extends to the external surface of the dura, which becomes thickened by deposits of inflammatory material, and often adheres to the surface of the bone. The accumulation of pus, cheesy material, and other inflammatory products between the dura and bone, and the great thickening of the external surface of the dura, are capable of exerting considerable pressure on the cord, and directly interfering with the function and life of the spinal nerve roots.

The most common displacement is arching forward of the spinal column from giving way of the bodies of one or two vertebræ, and an angular projection backward of one or more spines. Sometimes the curvature is less acute, as it may extend over four or five vertebræ. At times there

is a lateral displacement, or the posterior portion of the body of a vertebra may be crushed by the superincumbent weight, when an arching backward of the spinal column results.

The symptoms of caries may be divided into bone, nerve root, and cord symptoms. The symptoms of bone disease should be as well understood by the physician as by the surgeon, because a failure to examine for these, and to appreciate their importance in an early stage of the disease, may result in irreparable damage before the surgeon is consulted. Gowers is the only author of a work on nervous diseases who devotes any space to the subject of caries of the spine. Admitting, as he does, that the pathological anatomy, symptoms, and treatment of the bone diseases are purely surgical subjects, yet it seems to me that the great importance of the nervous lesions which so commonly result from caries demands for it a place in every work devoted to the diseases of the nervous system, because the vast majority of persons suffering from the early stage of spinal caries consult either the neurologist or the general practitioner before the surgeon's advice is sought. Judging from my own experience, the earliest and most important symptoms of caries of the spine in the adult are pain and tenderness. I am aware that many orthopædic surgeons assert that these symptoms are frequently absent, and of inferior importance when present. This may be true of the disease in early childhood, before the sufferer has learned to give definite expression to its symptoms, but in the adult the statement I have made holds good. I suspect that one reason for orthopædic surgeons laying so little stress upon pain and tenderness is because most of the cases of spinal caries coming under their observation are first seen after the incipient stage has passed.

The pain in the spine is felt at the affected part, and is

increased by jarring or rotating the spinal column, by rapid bending of the spine backward or forward or from side to side, and especially by firm pressure over the diseased bones. Gowers considers the local tenderness of very great importance. It is among the earliest symptoms, and is probably the most constant. It is elicited by direct pressure on the spine of the affected vertebræ and also by pressing the spine from side to side. When the disease is situated in the most mobile portions of the spine, as in the cervical region, movements are the most painful. When it is situated in the latter region movements of the head are greatly restricted and painful. The patient instinctively fixes the head in an abnormal position. Sometimes it may incline to one side so persistently as to simulate torticollis, but the sterno-cleido-mastoid muscle is tense on the side toward which the head is inclined, just the opposite of what we find in the latter disease. With the pain and tenderness, rigidity of the muscles that fix the affected portion of the spine is usually found, especially during the examination. Thickening of the spines with induration of the soft tissues adjacent to them is sometimes found, especially in the cervical region. I have seen a few cases with considerable thickening of the spines in the dorso-lumbar region following injuries to this region. Caries of the spine should be detected before deformity is apparent if the patient has sought advice for the early symptoms. In only a few cases is the deformity one of the earliest symptoms. Marked deformity is often absent when the morbid process occurs in the cervical region until considerable progress has been made in the disease. In the dorsal and lumbar regions slight lateral or backward displacement of a spine may often be detected as soon as the body of one of the vertebræ begins to break down. It is unnecessary for me to call your attention to the formation of abscess, which

sometimes occurs as a result of spinal caries, further than to remind you that when you find evidences of accumulation of pus in the groin or in parts adjacent to the spinal column you should carefully examine for bone disease.

Nerve-root Symptoms.—The spinal nerves are most frequently damaged when the bone disease is situated in the cervical region. Nerve-root symptoms may be divided into four classes: sensory, motor, reflex, and trophic. The earliest of these, as a rule, are the sensory, but even these are not often sufficiently early for diagnosticating the disease in its incipiency, except when external pachymeningitis is associated with caries. In a number of cases radiating pains along the course of the nerves are quite prominent. When the disease is high up in the cervical region, the pain extends over the posterior portion of the head; when in the cervico-dorsal, the arms suffer; and when in the lumbo-sacral, especially if the disease is unilateral, the pain is felt along the course of one sciatic nerve, and is not infrequently mistaken for sciatica. When the pains are first felt, the skin over the area to which the affected nerves are distributed is hyperæsthetic, but later irregular areas of partial or complete anæsthesia are present. It is only after the disease has made considerable progress in the dorsal and lower portions of the spinal column that muscular weakness is discoverable. In disease of the cervical vertebræ any muscular weakness that may occur in the arms is easily detected. Muscular rigidity is not an early symptom from irritation of the nerve roots, and it is rarely a very prominent one from this cause. It is, however, as we found, a symptom of some importance early in the disease, when it occurs from voluntary or involuntary efforts of the patient to fix the spine in order to lessen the pain caused by movements of the spinal column.

The reflexes are usually abolished over the area supplied

by the affected nerves, and often increased plantar reflex is an early symptom of spinal caries. The mechanism by which the plantar reflexes are increased under such circumstances is not easily explained. It is not entirely, if in part, due to irritation of the nerve roots.

Trophic disturbances are seen only occasionally early in the disease, but more commonly they occur later. When the lower cervical nerve roots are irritated, some derangement of the sympathetic nerve or ganglia may take place, resulting in irregularities of the pupils and vaso-motor disturbances, with sweating, etc. At times muscular wasting of the arms and hands is prominent in caries, and occasionally it is seen in one or both legs in affections of the lumbar and sacral bones. Herpes zoster, occurring along the course of the irritated spinal nerves, is a very infrequent affection from bone disease.

Symptoms of impaired functions of the cord are very frequent as a result of caries of the vertebræ. In the majority of instances they occur after the bone disease has made considerable progress, and are then due to compression of the cord and the resulting myelitis. Occasionally, however, the cord is involved before bone disease is suspected, and, under such circumstances, the spinal marrow probably suffers from inflammation rather than compression, except in cases where the dura has become sufficiently thickened to exert pressure on the cord. As I propose, in connection with the case which we are now considering, to devote some time to the consideration of compression of the cord and the resulting inflammation and degeneration, I shall postpone what I have to say further concerning the cord symptoms of caries until the next lecture.

The diagnosis of caries of the spine in the majority of instances is not difficult, if the chief symptoms are borne in mind and repeated careful examinations are made. Pain

in a limited region of the spinal column, increased by extreme lateral, forward, and backward flexions of the spine; deep tenderness, limited to one or two spines, often associated with slight irregularities of the spines at the seat of greatest tenderness; the presence of tuberculosis or the history of an injury to the spine, or of tuberculosis in the family; nerve-root pains, with excess of cutaneous reflex action of the soles of the feet early in the disease; areas of hyperæsthesia, anæsthesia, muscular rigidity or wasting; the youth of the subject, in a majority of instances, and the cord symptoms are points of more or less importance, and should form a guide for a systematic investigation. When evidences of bone disease precede cord symptoms, a diagnosis of caries, with sequential lesions, will rarely be incorrect. When caries and cord symptoms develop at the same time, the former may be overlooked unless care is exercised in examining for it. When cord or nerve symptoms precede distinct evidence of bone disease, there is danger of mistaking the nerve lesion for the primary one; but, even in these cases, an error in diagnosis may be prevented by carefully examining for the early symptoms of bone disease, such as pain, tenderness, and slight irregularity of the spines. It is exceedingly rare for all these symptoms to be absent, but they may be slight and unobtrusive, and must be carefully searched for. Primary pachymeningitis gives rise to nerve-root and cord symptoms similar to those following caries. The absence of evidence of bone symptoms would be in favor of the former. Pachymeningitis in childhood, or young adults, or in tuberculous subjects, is much more likely to be secondary to bone disease than to be primary. The absence of other causes of pachymeningitis, such as syphilis, alcoholism, and repeated exposures to cold, is in favor of caries. When caries occurs in the cervical region, there may be little or no deformity, and the muscu-

lar wasting in the arms may simulate progressive muscular atrophy, but the irregular distribution of the muscular atrophy, the severe pains along the course of the spinal nerves, and the presence of areas of anæsthesia would be sufficient to distinguish this from the progressive muscular atrophy in which the only sensory symptoms are vague rheumatoid pains in the affected muscles. The disease may be mistaken for a primary transverse myelitis when the cord symptoms are early and prominent. Under such circumstances, the presence of the symptoms of the incipient stage of bone disease should be sufficient to put one on his guard. I have met with a number of cases of caries of the spine in which the diagnosis of spinal irritation had been made by physicians of no mean ability. In the latter trouble there are usually two or more points of spinal tenderness, or the parts over the entire spinal column may be sensitive to pressure, and deep pressure does not increase pain over that produced by slight pressure, and in some cases it causes less pain. The absence of irregularity of the spine and areas of anæsthesia is in favor of spinal irritation in doubtful cases, especially in nervous and anæmic females. A diagnosis of hysterical paraplegia with spinal tenderness has been made in young women suffering from caries of the spine resulting in myelitis. The presence of distinct evidence of bone disease and of organic lesion of the cord, which will be found if carefully searched for, will prevent such a mistake. The danger of confounding hysteria when paraplegia is present with caries of the spine is much less than the opposite error, and will not occur if care is used in the examination.

When both the sciatic nerves are the seats of pain in caries of the lumbar or sacral vertebræ, the danger of mistaking the disease for sciatica is not great if it is borne in mind that double sciatica is exceedingly rare, and that

every such case should arouse suspicion of the pain being symptomatic of other and usually graver trouble than inflammation of the sciatic nerves. When, however, only one sciatic nerve is involved in caries beginning unilaterally, an error in diagnosis may occur, but it may, as a rule, be prevented if it is remembered that in neuritis due to pressure the nerve below the seat of pressure, while it may be the seat of pain, is not tender until sufficient time has elapsed for descending neuritis to affect the peripheral portion of the nerve. Further, in pressure neuritis the greatest pain is usually experienced at the distal portion of the affected nerves. After we have satisfied ourselves of the presence of bone disease we must go further, and determine whether it is due to caries, tumor, or an eroding aneurysm. Gowers states: "In the first half of life the presence of bone disease is practically tantamount to the recognition of caries." In the second half of life evidence of disease of the spinal vertebræ should lead us to examine carefully for the symptoms of aneurysm or tumor. The presence of a tumor in another portion of the body, or the history of the removal of a tumor from a person suffering from disease of the vertebræ, should lead us to suspect tumor. The symptoms of bone disease due to aneurysm or tumor are more likely to be unilateral in their early stage than those from caries, and the pain produced by aneurysm or tumor involving the bones of the spinal column, especially on movement, is usually intense to a degree out of all proportion to the pain of caries. I will leave the consideration of the prospects of recovery and the management of caries for the next lecture, when we shall study compression of the cord, especially from disease of the bones.

LECTURE II.

Compression of the spinal cord may result from any morbid process that gives rise to narrowing of the spinal canal to a sufficient extent to encroach upon the space actually occupied by the cord. The chief cause is an affection of the bones of the spinal column, especially from caries and traumatism. Growths of the bones or of the membranes, aneurysms eroding the bones and extending into the spinal canal, and thickening of the dura, may compress the cord. Most of the causes mentioned directly affect the cord only one or two inches in its vertical extent, but sometimes we find an exception when the compression is due to pachymeningitis with considerable thickening of the dura. Under such circumstances the cord space may be encroached upon several inches in its longitudinal axis. The character of the symptoms depends largely upon the degree of the compression and the rapidity with which it is developed, regardless of what may be the morbid process by which the compression is exerted; but my remarks to-day will be limited mainly to compression of the spinal cord from caries of the spine, and its usual concomitant, thickening of the dura.

The functions of the cord are disturbed by compression and the resulting inflammation.

From a knowledge of the pathological changes that take place in compression of the cord, the symptoms are readily appreciated. These consist in narrowing of the cord at the seat of compression, change in color and consistence of the cord substance, thickening of the walls of the blood-vessels, increase of interstitial tissue and de-

struction of nerve fibers. The narrowing includes one or two inches of the vertical extent of the cord. Sometimes the cord at the seat of compression is cylindrical, some times flattened, and at others it is irregularly indented, thus accounting for the irregularity of the symptoms, as one system of fibers may be greatly damaged while another may escape. The size of the cord is often reduced to one half or even one third of the normal. In some cases, however, although the function of the cord is greatly impaired, the size at the seat of compression is but little less than that of normal. The affected portion is grayish, and the distinct difference in color between the white and gray matter is lost. In recent acute cases the consistence of the cord is lessened, but in those where the compression is very gradual, and in the chronic stage of all varieties, the consistence is increased by the development of interstitial tissue and the degeneration of nerve fibers. The change in color and consistence of the cord is due to inflammation, and not directly to the compression. The inflammation is not limited to the seat of compression, but it extends some distance above and below it. From the diseased portion of the cord ascending and descending degeneration takes place. In all cases great compression is attended with considerable inflammation, but slight compression, which usually gives rise to a mild form of inflamma tion, may set up severe myelitis. The rapidity with which the inflammation of the cord is developed is usually modified by the slow or rapid increase of pressure. Suddenly developed pressure always causes acute myelitis, and gradual increase of pressure, as a rule, gives rise to chronic inflammation, but in some instances subacute, or even acute, myelitis may result from gradually developed pressure. The microscopic appearances are those of myelitis, with increase of interstitial tissue and degeneration of nerve elements. In

the early stage various cell formations take place, but later these may in part, if not entirely, be replaced by a reticulum of interstitial tissue. The nerve elements become inflamed and undergo degeneration, and "masses of myelin, granule corpuscles, and corpora amylacea" are seen. In a few instances all the nerve fibers at the seat of greatest compression are destroyed beyond any possibility of recovery; but fortunately such cases are comparatively rare. In the majority of instances, a large proportion of these fibers persist with narrowed medullary substance, and are capable of regaining, imperfectly it may be, their power of conduction, although surrounded by a great increase of interstitial tissue. The walls of the blood-vessels are thickened, and in some portions the caliber of a vessel may be found obliterated. The intensity of the inflammation gradually lessens the farther we go from the seat of compression, but ascending and descending degeneration of the destroyed nerve fibers takes place throughout their course in the cord. The cornua of the cord suffer, the ganglion cells degenerate more or less completely, and functionless interstitial tissue takes their place. The nerve roots that pass through the affected part are crippled by the pressure, and are further influenced by the inflammatory process. They become inflamed, interstitial tissue increases, and the nerve elements degenerate.

The symptoms of compression of the cord are modified by numerous and varying conditions, and those occurring on a level with the morbid process in the cord are so obscured by the nerve-root symptoms that it will be necessary to refer to these briefly, although we discussed them at some length in our former lecture in considering them in relation to the symptoms and diagnosis of caries of the spine. The involvement of the nerve roots abolishes the reflexes on a level with the morbid process. The spinal

nerves whose roots are directly affected are the seat of pain which radiates throughout their distribution. The location of the painful nerves depends upon the position of the caries. The pain may be dull or sharp and neuralgic in character, encircling the trunk or extending down the limbs. When in the limbs the joints are often the principal seats of pain, and become extremely sensitive and the skin over them hyperæsthetic. The pain may be intermittent or constant, with paroxysms of severe suffering. Sometimes tender points may be found along the course of the nerves. After the nerves begin to degenerate, anæsthetic areas develop, but these areas are still the seat of pain. Contractures of the muscles supplied by the affected nerves are rare, but more common when the morbid process is situated in the cervical region than when the dorsal or lumbar region is affected. Weakening and wasting of the affected muscles with electrical changes of degeneration are more frequent than contractures resulting from nerve-root involvement. Symptoms of irritation of the nerve roots usually precede the cord symptoms.

The cord symptoms, as a rule, are gradual in their development, although they may be acute or even sudden in their onset, and consist generally in impairment or abolition of the conducting functions of the cord at and below the seat of the lesion. As the dorsal region is the most common seat of compression of the cord from caries, paralysis of the legs is the usual form of paralysis. There is considerable variation in regard to the time of the occurrence of caries of the spine and the development of symptoms of compression of the cord. Bone disease may occur in childhood and compression of the cord not till adult life. More frequently the symptoms of caries antedate those of compression of the cord one or two years or a few months. In some instances the symptoms of each may ap-

pear about the same time, or compression of the cord may take place before disease of the bones has been suspected. In many cases there is no apparent exciting cause to precipitate compression of the cord, while in others a slight strain of the back, a blow to the spine, a fall, or exposure to cold precedes the development of compression of the cord in persons suffering from caries of the spine. The cord symptoms usually increase slowly, although sudden giving way of the bodies of the affected vertebræ may result in rapid compression. In some instances months or years may elapse after cord symptoms have manifested themselves before compression has reached the paralytic stage, but cases are recorded in which only a few weeks, days, or hours have been required after the first appearance of the cord symptoms before paralysis was complete in the parts below the spinal lesion. The cord symptoms are usually bilateral, although it is not uncommon for them to be more marked on one side than on the other, and in some instances the symptoms are well developed for months in one leg, while they are scarcely appreciable in the other.

The symptoms may be divided into motor, sensory, reflex, and trophic. In the majority of cases of compression of the cord the motor symptoms are more marked than the sensory. When the posterior surface of the body of a vertebra gives way before the anterior the spinal column arches backward, resulting in narrowing of the spinal canal by the processes of the vertebra projecting forward and impinging against the posterior surface of the cord; the sensory symptoms are usually more pronounced than the motor. It will be convenient for us to study the general symptoms of compression of the cord regardless of the position of the morbid process, then consider special symp-

toms resulting from compression in the cervical, dorsal, and dorso lumbar regions.

Motor Symptoms.—The motor symptoms usually consist of gradually developed weakness of the muscles in the parts below the seat of the lesion, which may increase to more or less complete paralysis. In some instances, as we have seen, the paralysis is developed suddenly. When the paralysis is very gradual in its development, there may be no apparent disturbance of the bladder if the lesion is sufficiently removed from the lumbar enlargement so as not to involve this portion of the cord by the resulting inflammation. On the other hand, in suddenly developed paralysis from compression of any portion of the cord the functions of the bladder are always impaired. If the lesion is in the dorsal or cervical portion there will be "incontinence of retention" or an inability voluntarily to evacuate the bladder. This is often only temporary, unless the destruction to the cord is very great, when it may become permanent. Contractures of the affected muscles take place in lesions above the lumbar enlargement. The legs may be paralyzed before the arms when the paralysis results from compression of the cord in the cervical region.

Sensory Symptoms.—The sensory symptoms from compression of the cord are more variable than the motor. In some cases, with almost absolute paralysis of motion, the sensory phenomena may be fairly well preserved. In very severe compression of the cord at the stage of the disease when the morbid process most impairs its function, anæsthesia may be as complete as the motor paralysis. During the periods of the progress and decline of cord changes the sensory function is usually less involved than the motor. Pain is often a prominent symptom, and it may be felt in the legs when the morbid process is in the dorsal region.

Its seat of intensity depends largely upon the position of the compression. When the latter is in the dorso-cervical region, the arms are the seat of pain. Late in the disease, when ascending and descending degeneration has taken place, pains may radiate around the trunk and down the limbs from nearly the entire length of the cord. In this stage of the disease, when a fair amount of motion has been regained, there are often decided ataxic symptoms, especially in the muscles of the legs. Painful impressions are often delayed several seconds.

Reflexes.—The reflexes are always abolished on a level with the cord lesion. Increase of the superficial reflexes, especially of the soles of the feet, is often a prominent symptom early in the disease. So commonly are the plantar reflexes exaggerated in compression of the cord that they possess some diagnostic significance in obscure cases of cord disease. When the morbid process is situated above the lumbar enlargement, and the latter is not involved by inflammation or degeneration, the deep reflexes below are exaggerated, and ankle clonus is usually present.

Trophic Disturbances.—Muscular atrophy is slight or pronounced in proportion to the severity of the cord affection. Bedsores rarely form except when the cord at the seat of compression is rendered almost functionless, or when the lumbar enlargement is affected. Cystitis is not troublesome if the bladder receives proper attention, unless the lumbar portion of the cord is seriously damaged.

When the morbid process is situated in the cervical enlargement, the arms are affected as well as the legs. In the cases in which the paralysis and muscular wasting in the arms are due to pressure on the nerve roots, the arms are paralyzed before the legs; but if the paralysis is due to pressure on the cord, the legs may be paralyzed before the

arms. The cervical muscles may be weakened so that it becomes difficult for the patient to support the head erect. All the superficial reflexes below are exaggerated early in the disease, and myotatic irritability in the legs is excessive and ankle clonus is pronounced. In severe lesions the patient loses control of the bladder. Anæsthesia, more or less marked, is present in the parts below the cord lesion. The arms, upper portion of the chest, and sometimes the cervical and occipital regions, are the seats of pain. Cardiac and respiratory involvement take place when the lesion is high up in the cervical region. The intercostal muscles are paralyzed, and if the diaphragm is seriously affected death takes place from respiratory paralysis. Any pulmonary affection is grave in compression in the cervical region of the cord—even a chronic bronchitis may prove fatal. The pupils and the vaso-motor apparatus do not become affected unless the sympathetic nerves or ganglia are affected. As a rule, there is no tendency to bedsores. Contractures of the leg muscles occur. If the dorsal region is the seat of compression, the arms, the heart, and respiration escape except in lesion of the upper portion, when the intercostal muscles may be paralyzed and the arms become secondarily affected from the extension of inflammation. The disturbances in motion, sensation, and in the reflexes in the parts below are the same as in lesions of the cervical region, with the possible exception that pains are often felt in the legs in compression of the dorsal portion of the cord. When the lumbar enlargement is compressed or becomes involved by extension of inflammation, paralysis of the bladder and bowel, with the tendency to the formation of bedsores, abolition of the knee jerks, flaccidity and great wasting of the paralyzed muscles take place.

Diagnosis.—The diagnosis of compression of the cord

due to caries of the vertebræ depends upon the evidence of bone disease, nerve-root and cord symptoms. In each case other causes of compression than caries have to be carefully excluded. The first point to determine in a case presenting cord symptoms is whether the symptoms are due to compression of the cord or to other causes. After determining that the cord is compressed, we should endeavor to find the source of compression. The chief diagnostic symptoms of compression of the cord are the indications of irritations of the nerve roots, such as pains radiating from the spine, in the arms, round the trunk, into the groins, or down the legs, depending upon the position of the morbid process; the evidence of bone disease, such as localized pain and limited tenderness of the spine; and disturbances in the functions of the cord, as motor impairment, sensory changes and alterations of the reflexes, especially increased plantar reflexes, in the parts below the seat of the spinal lesion. The affections with which compression of the cord is most likely to be confounded are transverse myelitis, medullary tumor, hæmorrhage in the substance of the cord, and syringomyelia. In primary transverse myelitis there are neither nerve-root nor bone symptoms, and in those cases of chronic myelitis attended with nerve-root symptoms the latter are secondary to disturbance in the cord. A medullary growth may give rise to prominent nerve root symptoms, which are often unilateral at first, but no bone symptoms are present until late. Besides, trophic disturbance is usually much more prominent in medullary growths than in compression, and evidences of growths in other parts of the body are frequently present in tumor of the cord. The history of sudden onset and the absence of nerve-root and bone symptoms are sufficient to prevent mistaking hæmorrhage in the cord substance for compression. Syringomyelia is a condition in which the cord substance is compressed from

within outward instead of from without inward, and presents some symptoms in common with the ordinary form of compression of the cord; but the absence of temperature and pain senses, while tactile sense remains fairly good, the absence of bone and definite nerve-root symptoms, with the prolonged duration of the disease, make the symptoms of syringomyelia quite distinct from those of the condition under consideration.

Having satisfied ourselves that the cord is compressed, our next duty is to ascertain the nature of the cause of compression. Is it due to meningitis, especially chronic internal pachymeningitis, to tumor in the spinal canal or bone, to an eroding aneurysm, or to caries? In myelitis secondary to pachymeningitis the nerve-root symptoms extend over a much greater vertical area of the body than in compression due to bone disease. Bone symptoms are absent, and a cause of the meningitis can usually be traced. A tumor in the spinal canal gives rise to no bone symptoms until late; unilateral nerve-root symptoms are common and do not become bilateral until cord symptoms develop; therefore, compression of the cord manifesting itself after bilateral root symptoms would in all probability not be due to a tumor in the spinal canal. A tumor in the bone, like a growth in the canal, is usually found secondary to growths in other portions of the body, except in cases of injury to the spine. Compression of the cord in children and young adults would be against tumor in favor of caries. Nerve-root pain, especially on movements of the spinal column, is greater in cases of tumor than in caries. The presence of an eroding aneurysm could be determined by a careful examination. Points in favor of caries as the cause of the compression are the usual nerve-root and bone symptoms, the youth of the patient most commonly (although caries may occur at any age), tuberculosis, and the ab-

sence of symptoms significant of other causes of compression.

The course and prognosis of compression of the cord from caries of the spine are modified by the curability of the bone disease, the extent of the compression, both from displaced bone and thickening of the dura, the rapidity of the development and the extent of the cord lesion, and the amount of degeneration in the cord. While the progress of compression of the cord may, to some extent, be more or less independent of the progress of the bone disease, yet the two when they are present, the former as a sequential lesion of the latter, are conveniently studied together in relation to the course, prognosis, and treatment. Caries of the spine, in its early stage, before the disease has made much progress, under prompt and efficient measures for its relief, is exceedingly amenable to treatment. After the disease has advanced sufficiently for the bone substance to begin to break down and pus to form, with not infrequently an undermined condition of the health, the probable duration and termination of the disease become uncertain. In a few cases pus continues to form for years; in time the downward progress is arrested, and the patient makes an incomplete recovery. On the other hand, death may be caused by prolonged exhausting discharges and other depressing influences. It is rarely that caries is the direct cause of death, but such a termination is usually the result of myelitis secondary to the bone disease. Carious bone may heal and union occur between the affected parts. In some instances caries becomes quiescent for a time, to be followed by periods of increased activity. In some cases the myelitis is out of proportion to the apparent degree of pressure, so that as inflammatory action subsides in the cord, the paralytic and other cord symptoms may improve, although the condition of the bone remains practically the same.

It is impossible for us to determine with certainty the extent of the pathological changes that have taken place in cases of paraplegia, therefore anything like definiteness in prognosis is equally impossible. Paralysis, motor and sensory, may persist, but more commonly the sensory symptoms, except the pain, lessen or pass away and motor paralysis remains with contractures of the leg muscles when the morbid process is above the lumbar enlargement, and flaccid paralysis of the legs when the lumbar enlargement of the cord is involved. Under such conditions life may be prolonged for several years, but often cystitis, bedsores, kidney disease, or tuberculous deposits in other parts of the body put a termination to life. Children give the best prognosis in caries and compression of the cord, young adults stand next, and the degenerative period of life furnishes the gravest. But we must remember in adults that paralysis which has been complete with extensor contractures for more than a year may entirely pass away. Flexor contractures lend gravity to the prognosis. After recovery has taken place it is, as a rule, permanent, but relapses are readily produced by injuries to the spine or exposure to cold or damp, and even in some cases re lapses occur without apparent cause. Gowers states that persons who have suffered from caries during childhood without apparent injury to the cord may develop symptoms of degeneration of the lateral columns later in life. Caries and damage to the cord situated high up in the cervical region become especially dangerous on account of the tendency to respiratory paralysis. In the lumbar region, cystitis, bedsores, and other profound trophic disturbances usually result and add gravity to the prognosis. When the morbid process is situated in the dorsal region the chances of recovery are the greatest. In those cases in which absolute paralysis develops rapidly after symptoms

of myelitis begin, the prognosis is more favorable than where it takes place more gradually, because in the former instance inflammation of the cord, which may subsequently subside, has had more to do with the paralysis than the pressure from bone displacement. When sensation is preserved, the chances of recovery are greater than when it is abolished, although motor paralysis may be complete.

In the light of prognostic indications of caries of the spinal vertebræ followed by compression of the cord, what will be the probable result in the case we were engaged in studying at our last clinic? A year ago the prognosis seemed hopelessly grave, but to-day the anæsthesia has passed away almost entirely; she is able to use both legs and to bear her weight on them; the contractures of the leg muscles, which were extensor in character, are slight, and the progress of the bone trouble has been arrested with union between the inflamed surfaces, so that we can now predict that she will be able to walk in the course of a few months, and in time may have fair use of her legs. The case shows that the most desperate conditions in caries of the spine and compression of the cord may be more or less completely recovered from.

Treatment.—Although the treatment of caries of the spine comes under the province of the surgeon, yet every physician should be familiar with the general management of these cases, especially as timely and proper treatment is of so great importance. In the early stage of the disease, before symptoms of breaking down of the bone have become manifest, the mistake of allowing the patient to be on his feet is often made. Since the plaster jacket properly adjusted affords such excellent support, many trust to this to the exclusion of rest and local treatment. It seems to me that this may be another error. It is a safer method to put the patient at rest in the recumbent posture until all

signs of irritation in the bones and surrounding structures have subsided, then apply the plaster jacket and allow a moderate amount of exercise on the feet. While the patient is confined to bed local treatment is very efficient in lessening the inflammation and shortening the duration of the active process. The most effectual is with the actual cautery, at white heat, applied over the diseased bone. My experience is that a rather large or deep eschar produced by the cautery is more effectual than repeated light burnings of the skin. The jacket should not be applied until all tenderness and pain in the back have subsided. In the meantime everything should be done to maintain the patient's nutrition by the administration of cod-liver oil and iron, together with an abundance of the most nutritious food. In cases further advanced, so that the inflamed bone is beginning to break down and pus to form, or in which cord symptoms have gone on to paralysis, rest in the recumbent posture will be necessary for months, and the nutrition must be most zealously looked after and kept up; but the same active measures in the way of counter-irritation by means of the actual cautery will rarely be necessary. Some advocate the hot iron in all stages of caries of the spine, but my experience has been that it is most potent for good before much destruction of bone has taken place or the functions of the cord have been seriously damaged. In the suppurative stage of caries, or when absolute motor paralysis, with anæsthesia, has occurred, the cautery, if used at all, must be employed lightly, and care should be taken not to form an open sore in the anæsthetic area, lest a troublesome and exhaustive bedsore be the untoward result. While the plaster cast is an excellent aid in the treatment of caries of the spine, it does not seem safe to allow a person to trust to this for support so long as decided inflammation exists in the bones. The same care and vigilance necessary in the

treatment of myelitis in guarding against bedsores, cystitis, and kidney complications are demanded in compression of the cord. After the bone disease has been arrested, which is the first object to be achieved in cord disease from caries, the remainder of the treatment consists in endeavoring to prevent a relapse of the bone trouble, and in dealing with the nervous affection as one of subacute or chronic myelitis. The former object is attained by means of the plaster jacket for a year or more, maintaining the best possible health of the patient, and preventing injuries to the spine from various causes. The latter is aided by preventing contractures, employing massage and electricity, and guarding against complications.

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