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THE PRESSURE PARALYSIS OF POTT'S DISEASE.*

By GEORGE R. ELLIOTT, M. D.

The object of this paper is to determine the mechanism of the lesion which gives rise to pressure paralysis of the spinal cord, limited to that form of paralysis which complicates Pott's disease.

Anatomical Considerations.—It may not be out of place to call attention to the following anatomical points:

1. The spinal cord reaches only to the lower border of the first lumbar vertebra.

2. The cord only partially fills the vertebral canal.

3. The cord swings in the spinal canal, insulated by means of the arachnoid fluid which surrounds it.

4. The cord is much nearer the anterior wall of the canal than the posterior, held in place by means of the anterior nerve-roots.

5. The tracts conducting motor impulses are more superficial than those transmitting sensory impulses.

It further may not be out of place to state that paralysis in Pott's disease bears no relation to deformity. A spinal column may be bent at nearly a right angle without giving

* Read before the Section in Pathology of the Ninth International Congress, September, 1887.

rise to any paralytic symptoms, while in another case, manifesting no deformity, *complete paraplegia* may be present.

Pathology.—In order to arrive at an intelligent understanding of the pathology of the lesion to be considered in this paper, I cite briefly the carious process which is the exciting ætiological factor. It matters little whether we accept the tubercular theory of caries or not. The process is one insidious in its onset and slow in its progress. Cornil and Ranvier, in comparatively recent investigations, seem to show rather conclusively that the disease begins not as an inflammatory process, but a fatty degeneration of the bone cells. The result—a destruction of the bone cells and consequent inability on their part to perform their function as agents of nutrition. The osseous trabeculæ, killed by the death of their cellular elements, form so many small foreign bodies which determine suppurative inflammation about themselves. The medulla becomes vascular, adipose cells disappear and are replaced by embryonic cells, and suppuration is established. The bone cells which have escaped fatty degeneration become active, the osseous substance surrounding them is dissolved, the necrosed trabeculæ become free, and granulations or fungosities are formed from embryonic medulla. Islets of osseous tissue become necrosed oftentimes, and are dislodged by granulations and carried away by suppuration. In their place are left irregular cavities. Here, then, we have a suppurative process in progress which is most frequently confined to the anterior portions of the bodies of the vertebræ. These diseased portions, giving way, permit the vertebræ to fall together. The disease creeps dorsad and destroys, to a greater or less extent, the posterior common ligament. The latter, dissociated, allows the pus to pass from the vertebræ. The dura mater and the peridural tissue are aroused to inflammatory

activity. The external surface of the dura mater becomes covered with granulations of a fungoid character, and infiltrated with caseous pus. This condition passes under the name of *pachymeningitis externa caseosa.* This process shows no tendency to extend beyond the site of the vertebral disease. It is limited. In a cross-section we can see how the diseased condition abruptly gives way to the healthy membrane.

Up to this point the pathology stands, I can safely say, undisputed. In the large majority of cases of caries of the spine the pathological chapter closes here. The products of inflammation remain imprisoned, ultimately to be absorbed and replaced by reparative material. Frequently paralytic symptoms manifest themselves, and here the pathology is by no means settled.

Ollivier * was among the first to describe at all carefully the changes which take place in the cord as a result of slow compression. He cites numerous cases where the membranes were found thickened and the cord softened without evidence of further disorganization. His findings were verified and described later by Louis.† These writers, and many of their followers who have described this form of paralysis, have dwelt upon the mechanical lesion alone as the cause of the paralysis. From time to time cases were reported where the cord seemed harder than normal, a condition not dwelt upon by these writers, and consequently no explanation offered. That the cord should become injured as a result of crumbling and broken vertebral bodies sufficient to lead to the production of marked angular curvatures, was considered but a natural sequence. This idea

* "Ueber das Rückenmark und seine Krankheiten," uebersetzt von Dr. J. Radius, Leipsic, 1824, p. 202.

† "Mémoire sur l'état de la moelle dans la carie vertébrale," Paris, 1826.

became so thoroughly fixed that many books, even to this day, refer only to bone as the pressure cause of the paralysis. It was noticed, however, that very frequently no paralysis followed in cases where the deformity was very marked. The spinal column might even be bent to a right angle as a result of the disease, and still no paralytic symptoms become manifest. That marked deformity can occur without the cord becoming compressed is also observed in lateral curvature, where sometimes the deformity assumes a frightful aspect. In the words of Fagge,* "every pathological museum contains specimens which show that the spinal canal generally retains its full width, however much its direction may be altered."

Further, paralysis was seen to complicate caries when careful examination of the spinal column gave no evidence of deformity. These observations naturally called for some explanation other than the current view.

The middle of this century, which inaugurated an active interest in the physiology and pathology of the great nerve-centers, produced men who sought a solution of the problem. Among others, Charcot † and his pupils made investigations and published to the medical world their conclusions. To Michaud, ‡ I think, can be credited the pioneer article upon this subject. These authors and investigators contended, and apparently demonstrated, that the cause of the paralysis in these cases is not directly due to a mechanical lesion, but to a myelitis the result of the mechanical lesion. Michaud held that myelitis was invariably the lesion, and even went so far as to claim its existence in cases giving no evidence of paralysis.

* Fagge, vol. i, p. 410.

† "De la compress. lente de la moelle ép.," "Leçons sur les mal. du syst. nerv.," 2me sér., 2me fasc., 1873.

‡ "Sur la méningite et la myélite dans le mal vertebral," Paris, 1871.

This explanation of the cause of this form of paralysis, first described by Michaud and Charcot, and subsequently supported by Bouchard * and Courjon,† was accepted and apparently verified by Leyden ‡ in his microscopical examinations of compressed cords.

Great controversy has now long been in vogue regarding the exact nature of inflammatory processes of the cord. So loosely has the term myelitis been employed that various affections of the cord have been called myelitis—polio-myelitis, ascending and descending degeneration, various kinds of softening; all these have been described by observers under the head of inflammatory lesions.

Recent pathologists have come to look upon the disease myelitis as one giving rise to certain unmistakable and ever-present findings. Stricker, Leyden, Joffroy, Schultze,# Strümpell, ‖ and others have found the following pathological findings which they regard as pathognomonic of the inflammatory lesion:

1. Increased size of nerve-elements, axis-cylinders enlarged, or without the sheath of Schwann.

2. Swelling of the interstitial tissue and increase of same, according to age of process.

3. Increased number of round cells in the connective tissue and around the blood-vessels.

4. Enlargement of Deiter's cells.

The later stages characterized by hyperplasia of the connective tissue.

* "Compress. lente de la moelle," "Diction. ency. des sc. médic.," 2me sér., vol. viii, p. 664, 1874.

† "Étude sur la paraplégie dans le mal de Pott," Paris, 1875.

‡ "Klinik der Rückenmarks-Krankheiten," vol. ii, first part, p. 149.

"Arch. f. klin. Med.," vol. xxv, p. 297; Virch. "Archiv," vol. lxviii, p. 111.

‖ "Archiv f. klin. Med.," vol. xxx, p. 526.

Erb practically coincides with the view of these authors, and their observations have been verified by numerous competent and careful observers both here and abroad.

The progress of the lesion in cases of slow compression is, as a rule, insidious, and, to reconcile the clinical symptoms with the lesion, the interstitial form of myelitis is believed to predominate. This is characterized by an increase of the neuroglia or connective tissue, similar to that occurring in other organs, the parenchyma becoming secondarily implicated. The interstitial tissue becomes increased, together with proliferation of the nuclei, great thickening of the walls of the blood-vessels, and distinct atrophy of the nerve-fibers. A later stage is characterized almost exclusively by the presence of connective tissue.

In support of the inflammatory theory are usually found post-mortem findings, showing the cord at the site of compression converted largely into connective tissue with thickened blood-vessels and sparsely distributed changed nerve-fibers and granular bodies.

Further, in one case examined by Michaud, when the compression had not as yet produced paralysis, but where sensory disturbances in the form of fulgurating pains were complained of by the patient, the microscope revealed increase of the connective tissue throughout the posterior root zone.

This explanation, then, of this form of pressure paralysis, which was so ably treated by Michaud and Charcot, supplemented by the studies of Courjon and Echéverria,* became as thoroughly implanted and accepted as had the explanation of Ollivier and Louis a quarter of a century before.

Let us proceed now to a closer study of the subject in

* "Sur la nature des affections dites tuberculeuses des vertébres," Thèse, Paris, 1860.

hand, and to that end we ask, *Can a mechanical lesion satisfy the demands of pathology and symptomatology?* Let us see.

A mechanical cause is present. We have seen how the bodies of the vertebræ become destroyed by the carious process going on in the bone, leading to the formation of an abscess the walls of which are composed of bone and of the thickened surrounding soft tissues. We have further seen how the dura mater becomes involved, the process which goes on being called pachymeningitis externa caseosa. The abscess-cavity is not a simple one, but in it are developed fungoid granulations, and the abscess products tend to accumulate. Nature tends to fortify the walls as far as possible. It is not common for the pus to perforate the walls and escape into the surrounding tissues. The site of least resistance is toward the spinal cord, which swings insulated in the spinal canal. The membrane bulges into the canal and the cord recedes. The canal offers escape for the cord up to a certain limit, when, if bulging still goes on, the medullary substance is destined to suffer. Here, then, is practically a fluid sac, its pressure force commensurate with its fluid tension, mechanically an elastic ball.

Clinically, the appearance of a psoas or gluteal abscess in a case of paralysis is often attended with the disappearance of paralytic symptoms. No one who has seen many of these cases has failed to note this clinical symptom. It is unnecessary to dwell upon the mechanism of this relief further than this allusion to it. I may add that bone is sometimes found pressing upon the spinal cord post mortem in cases which have had paralytic symptoms.

What does the gross lesion tend to show? At the compressed site the spinal cord is often reduced in size to such an extent as to appear simply like a band of connective

tissue. It is a striking fact that the diseased dura mater is limited to the site of the diseased vertebræ.

The change from "pachymeningite externe" to normal dura mater is abrupt. This has been noted by numerous observers. Cornil and Ranvier* say: "Inflammation of the dura mater is limited exactly to the parts of the vertebræ diseased." This change in the dura mater is invariably in the anterior part of the membrane, and only in grave cases of caries does it encircle more than half of the cord. Does not this limitation of the diseased dura to the exact site of vertebral disease argue the non-tendency of the inflammatory process to extend in the membrane beyond what is necessary to protect the cord from the abscess cavity itself—beyond nature's necessary protective area?

Medullary Surface of the Dura Mater.—This is usually intact and perfectly normal in appearance, the inflammatory process seeming to have spent its force in the external layers. Microscopical examination of the diseased membrane rarely shows even infiltration of the internal layers. Can we not draw from this a further inference of the non-tendency of the process to extend? Here, then, is a slow form of inflammation which does not seem active enough to extend beyond the limits of the diseased vertebræ, not active enough to involve, as a rule, the internal layers of the membrane in which it originates. Is it possible, then, *a priori*, that it will extend to the cord? It is proved, then, I think, that the *irritation force* is simply that of the pressure of a bland mechanical body.

Is there anything in the pathological findings to militate against the lesion being a mechanical one? Examination of compressed cords in the recent state show:

1. Granule cells, many or few, according to the mass of destroyed nerve-fibers.

* Ed. 1882, vol. i, p. 606.

2. *Débris* of broken-down nerve-tissue.

3. Swollen axis-cylinders, or absence of the same.

If examined at a later stage:

1. Nothing indicating changes in the blood-vessels.

2. No increase of cells about the vessels.

3. Occasionally traumatic hæmorrhage.

4. Nerve-fibers in course of destruction and others which have been destroyed.

5. Swollen axis-cylinders or empty spaces, the former site of nerve-fibers.

Then follows, as in all other analogous conditions, a secondary increase of the neuroglia.

Experimental Physiology.—It is a settled fact that pressure upon nerve-trunks interferes with nerve conductivity. It has been demonstrated over and over again that a very moderate constrictive force about a nerve separates the myeline within the sheath, and, if severe enough, leads to secondary degeneration, accompanied by all its pathological findings, rendering the nerve incapable of transmitting nerve impulses—*e. g.*, the injury to the musculo-spiral nerve from pressure. Has it been demonstrated in these cases that the pressure invariably leads to a neuritis in the sense of an inflammatory lesion?

Vulpian has shown experimentally that paralysis follows the introduction of a wooden match so placed as to press upon the spinal cord of a guinea-pig. The pressure was continued a quarter of an hour, and the paralysis disappeared one hour after the pressure was removed.

Attention is directed to what occurs after a nerve has been tied:

1. Segmentation of the myeline and disintegration of the same.

2. Disintegration of the axis-cylinders.

3. Complete absorption of the mass, leaving the nerve-sheath empty or containing only *débris* and nuclei.

The nuclei of the perineurium and endoneurium aid in the transformation of the nerve into a band of connective tissue. Here, then, is a process the result of which is practically identical with that following compression of the cord —changes, degenerative in character, the result of the mechanical interference, followed by increase of the connective-tissue elements. Pathological views characterizing degenerative changes as inflammatory are by no means established.

In the endeavor to settle this point, Dr. O. Kahler * made certain experiments upon dogs. He injected bee's-wax about the spinal cord; motor and sensory paralytic symptoms followed in the lower extremities. At varying periods the cords were carefully examined. After the compression had lasted from *six to thirteen hours* he found slight nodular foci located in the posterior and lateral columns. Microscopical examination of the same revealed marked swelling of the axis-cylinders, disappearance of the myeline sheath—*no interstitial changes.*

Two to Ten Days.—Microscopical examination showed greater swelling of the axis-cylinders and destruction of the same, granular cells, slight increase of Deiter's cells.

Later, five weeks to six months, there were found nodules of sclerosis with marked thickening of the connective tissue. He claims to have found none of the characteristics considered by Leyden, Joffroy, Stricker, and Schultze as pathognomonic of myelitis, and concluded that the results were purely those of a mechanical lesion.

Kahler states, as in peripheral nerves so in the spinal cord a moderate pressure is enough to excite a break in con-

* "Zeitschrift für Heilkunde," vol. iii, 1882, p. 187.

duction. A few fibers are destroyed in such cords, and we find lacunar changes distributed in the form of nodules.

Do the connective-tissue-growth changes which are usually found at the site of the compression lesion necessarily signify the inflammatory character of the lesion?

It is not denied that we usually find a sclerotic condition of the cord at the compression site when paralysis has existed for any length of time. A glance at the observations will make this conclusive. That such is the outcome of the lesion is beyond dispute. We contend, however, that the presence of sclerotic tissue in the cord is no proof that the initial lesion was inflammatory. We have seen that this follows various processes.

Ziegler says the increase of connective tissue is a result of every process of softening. The connective-tissue increase characterizes every process of degeneration. Throughout the nerve-centers destruction of nerve-tissue by processes degenerative or otherwise *is invariably followed by increase of the neuroglia.* The examination in the great majority of these cases is made long after the onset of the lesion. We find only the result of the process which has produced destruction. Leyden,* who came to look upon the lesion as identical with transverse myelitis, drew his conclusions almost exclusively from old findings, and these are identical with those following myelitis. Charcot's † case—where the disease was cured after one year's standing, and where, two years subsequently, death occurred, the cord at the compressed site being found smaller and presenting a sclerotic condition—does not seem to offer any great support to the theory that the difficulty had been inflammatory. Michaud himself offers but one case as the strong pillar of his argu-

* "Klinik der Rückenmarks-Krankheiten," vol. ii, part 1st, p. 149.

† "Communication de M. Charcot à la Société de biologie," Sept., 1871.

ment, where interstitial changes were found at an early stage, and this case had not as yet given evidence of paralysis.

Erb * says the fact that in not a few cases of compression of the cord the microscope reveals in the softened mass no granule cells, no hyperplasia of the connective tissue, no proliferation of the nuclei, but only swollen and disintegrated nerve-elements, speaks unmistakably in favor of the view that the process is sometimes one of simple softening.

Strümpell † contends that in repeated examination of cords compressed through spinal caries he has never found signs of inflammatory change. He refuses it upon pathological grounds, and believes that there is nothing found histologically that can not be accounted for as a result of a mechanical lesion. He says: "We must maintain, against the theory generally received at present, on the ground of many of our own investigations, that we have not the slightest reason to refer the occurrence of paralysis in spondylitis to a secondary myelitis. Such a 'compression myelitis'—that is, an inflammation of the spinal cord arising from the pressure as such—is to be rejected from general pathological reasons, and the microscopical examination of the cord also shows nothing which points to an inflammation or what may not be entirely the result of mechanical compression. If we make stained cross-sections of the hardened cord, we see under the microscope no signs of vascular changes, of hyperæmia, of accumulation of cells about the vessels, and only exceptionally a little traumatic hæmorrhage; but we do find, in addition to many still preserved nerve-fibers, other fibers which are involved in the disintegration, and evidence of those already destroyed. If the destruction of nervous

* "Ziemssen," vol. xiii, p. 469.

† "Lehrbuch der sp. Pathologie und Therapie der inneren Krankheiten," vol. ii, part 1st, p. 167.

tissue has advanced to a certain degree, there is in the later stages, as in all analogous processes, *a secondary implication of the neuroglia.* Now follows an increase of the interstitial connective tissue. Its proliferations, which take the place of the destroyed nervous tissue, seem diffuse at first, but later firm and fibrillary. Thus it happens that in old cases we find nothing at the point of compression but a loss of nerve-fibers in the cord, and in their place a *firm fibrous tissue.*"

What inferences can be drawn from a study of post-mortem findings? In the light of the present well-recognized value of post-mortem findings in settling pathological questions it would be a superficial study, indeed, in the present instance that neglected to consider them. From the literature of the subject those cases only are selected which have been reported with some attention to detail, for from such cases only is it possible to draw any reliable inferences. The cases are first cited and then analyzed.

Observation I.—Male, aged six years, with marked angular spinal curvature of eighteen months' duration, complicated with paraplegia which had existed for four months. Examination showed complete loss of voluntary power of both lower extremities; very little muscular atrophy; exaggerated reflexes even to the exhibition of spinal epilepsy. Died, seventeen months after the beginning of the paraplegia, from pulmonary complications.

Autopsy.—Partial carious destruction of the sixth, seventh, and ninth dorsal vertebræ, and complete destruction of the eighth. An abscess, the contents of which had degenerated into a white, cheesy mass, occupied the site. Pultaceous matter occupied the body of the eighth dorsal vertebra, and pressed upon the spinal cord.

Microscopical examination by Dr. E. C. Seguin showed *cord converted into connective tissue at the site of compression*, also above and below, the usual lesion of secondary degeneration.—Gibney, "The Illustrated Quarterly of Medicine and Surgery," April, 1882, p. 43.

Observation II.—Male, aged twenty-three years, complained for a short period of lancinating pains in the lower extremities, preceded by malaise and fever, and soon followed by complete paraplegia. The legs were flexed upon the thighs, and the latter upon the abdomen. Reflexes markedly exaggerated. Death occurred five weeks after the onset of the paralysis.

Autopsy.—Body of fifth dorsal vertebra completely destroyed by caries. The spinal cord was found compressed at the level of the fifth dorsal vertebra by a purulent mass which existed in the vertebral canal. Spinal cord showed *sclerotic changes at site of compression;* inflammation of pia mater involving spinal portion.—Mathieu, "Le progrès médical," No. 35, September 2, 1882.

Observation III.—Male, aged thirty-one years, complained for three months of a fixed pain in the region of the inferior angle of the left scapula. This was soon followed by a painful stiffness of the neck, together with marked atrophy of the infra-spinatus and deltoid muscles of the right side; later by atrophy and paralysis of muscles of both upper extremities. Died, five months after the onset of the lesion, from asphyxia.

Autopsy.—Cheesy osteitis, third, fourth, and fifth cervical vertebræ; "pachy. externe" limited to site of lesion, compressing same at height of fourth pair of cervical nerves through a cheesy proliferation which had broken through the dura mater.—Chantemesse, "Le progrès médical," No. 8, 1883.

Observation IV.—Male, aged forty-eight years, complained of occasional sharp pains in left arm, followed by weakness of both upper extremities and contraction of same. Lower extremities subsequently became weak, accompanied by a spastic condition of same. After ten weeks the lower extremities became completely paralyzed, with marked contracture. The upper extremities remained only partially paralyzed, with a high grade of atrophy of the thenar eminence of both hands; partial degenerative reaction of the atrophied muscles; otherwise normal electrical excitability, no fibrillary contractions, tendon reflexes increased only in lower extremities. The only disturb-

ances of sensibility were slight pains and paræsthesia in the arms. Death occurred, seven months after the beginning of the disease, from sepsis.

Autopsy.—Beginning caries, fourth, fifth, and seventh cervical vertebræ, without deformity of same. External surface of dura mater presented the condition of "pachy. externe" limited to the site of the bony lesion, the medullary surface of the dura mater remaining smooth and intact.

The spinal cord at the site of compression was found *softened* in a marked degree and unfit for microscopical examination.—Kahler, "Prager medicinische Wochenschr.," No. 49, p. 480, 1883.

Observation V.—Female, aged six years, with a history of paraplegia of four years' duration; reflexes markedly exaggerated; no sensory disturbances. Under treatment the paralysis disappeared to a considerable extent, so that the patient could bear considerable weight upon the feet. Death occurred from embolic pneumonia.

Autopsy.—Caries of the seventh cervical and first five dorsal vertebræ, the bodies of same being hollowed out and filled with cheesy pus. The spinal cord included between the second and seventh dorsal segments, measuring about 7 ctm., is transversely flattened, and has an increased consistency. In this portion of the cord there is destruction of both the gray and white matter. The walls of the blood-vessels are thickened, and the anterior portion of the dura mater is thickened and contains calcareous deposits. The cord above and below the compressed portion shows the usual lesion of secondary degeneration.—Ridlon, "N. Y. Path. Soc. Rep.," "Medical Record," March 26, 1887, p. 361, and July 16, 1887, p. 84.

Observation VI.—The patient, a male, complained for some months of pain in the back, followed by paresis of the lower extremities, which soon became completely paralyzed; reflex movements exaggerated; no marked sensory disturbances in same.

Autopsy.—Partial destruction of the four upper dorsal vertebræ. The spinal cord was found compressed at the point opposite the second dorsal vertebra. At this point careful ex-

amination failed to detect any inflammatory evidence.—Kadner, "Archiv d. Heilk.," xvii, 6, p. 481, 1876.

Observation VII.—Male, aged forty-four years, November 10, 1875, seized with pain in chest, neck, and shoulders, accompanied by cough and dyspnœa. These symptoms were soon followed by paralysis of the lower extremities, rectum, and bladder; sensibility of lower extremities diminished and reflex movements increased. Died February 17, 1876.

Autopsy.—Caries of the seventh cervical and four upper dorsal vertebræ. Examination of the spinal cord *negative.* There was a marked purulent infiltration beneath the periosteum, some pus having found its way into the spinal cord. This was the only lesion to account for the paralytic symptoms.—Kadner, *loc. cit.*

Observation VIII.—Patient gave a history of stiffness of the neck, followed soon by a numb feeling in the arms, which extended up the arms over the trunk. Head, neck, and legs remained unaffected. Examination upon admission into the hospital at Stockholm elicited partial anæsthesia, most marked upon the right side in hands and fingers. There was also paresis of the lower extremities. The right side of the body became completely paralyzed. Patient died from failure of respiration.

Autopsy.—Caries second, third, and fourth cervical vertebræ, with subperiosteal blood effusion. At a point opposite the second cervical vertebra the spinal cord was found compressed by a portion of bone. The pia mater at point opposite second and fourth cervical vertebræ was injected, but there was no exudation. The spinal cord at same height was softened exactly where the piece of bone pressed. Small hæmorrhagic foci were found in the cord substance at the site of compression. —Malmsten, Schmidt's "Jahrb.," Bd. 171, S. 65.

Observation IX.—Patient, aged sixteen years, upon admission into hospital was found to have complete paralysis below the upper extremities, with marked anæsthesia of same; reflexes markedly increased. Died four months after admission.

Autopsy.—Body of sixth cervical vertebra almost totally destroyed by caries. A rough protuberance projected into the anterior part of the spinal canal at this point, narrowing it con-

siderably. Dura mater shows the lesion "pachy. externe." Spinal cord at this point was found compressed and flattened. Microscopical examination not given.—Savory, "St. Barthol. Hosp. Reports," v, p. 45, 1869.

Observation X.—Male, aged sixteen years; voluntary movements of lower extremities completely abolished; no anæsthesia; electrical contractility normal; reflexes markedly exaggerated; absence of pain in the beginning of the disease, but later some pains of a lancinating character; pupils dilated, but they react slowly to light; function of bladder and rectum normal.

Autopsy.—Caries of the first four dorsal vertebræ. The *anterior columns* of the spinal cord were compressed at the site of the lesion, and found *softened* and *markedly anæmic;* the remaining portions of the cord are normal.—E. Rallett, "Wien. med. Wochenschr.," iv, p. 24, 1864.

Observation XI.—Patient, aged five years and a half, brought to the hospital with measles, complicated with capillary bronchitis. Examination of the spinal column showed an angular curvature at point of ninth and tenth dorsal vertebræ; no paralysis. Death occurred from the pulmonary disease.

Autopsy.—At the posterior surface of the diseased vertebræ a small pocket was found formed by the prevertebral cellular tissue, and filled with cheesy pus; the posterior ligament was found perforated at this point, and the contents were in contact with the anterior surface of the dura mater. The membranes appeared as though sprinkled with vegetations, forming plaques 4 to 5 ctm. square. Hardened sections of the cord showed *beginning myelitis.*—Michaud, "Sur la méningite et la myélite dans le mal vertébral," Paris, 1871, p. 66.

Observation XII.—Patient, aged two years, admitted to hospital November 9, 1869, with left hemiplegia, with rigidity. Later, the right inferior limb became paralyzed. The thighs were flexed upon the abdomen, and the legs upon the thighs. Nothing was observed abnormal with the right superior extremity, which alone could execute movements. Died April 2, 1870.

Autopsy.—Spinal cord at the site of the compression lesion

due to caries showed marked evidence of sclerosis.—Michaud, *ibid.*, p. 68.

Observation XIII.—Female, aged thirty-four years, noticed, after birth of first child, at the age of twenty-five years, slight deformity of the spinal column, followed three months later by numbness in the lower extremities. Entered the Hôtel Dieu with complete paraplegia of the lower extremities, accompanied by contracture of same. The paraplegia continued for more than four years. Patient died from a complicating morbus coxarius, September, 1869.

Autopsy.—Upon opening the rhachidian canal, the dura mater was found covered with a grayish exudation, which caused it to adhere to the posterior face of the changed vertebral bodies. An osteoform projection compressed the spinal cord at the level of the third dorsal vertebra. The spinal cord at this point was found extremely *diminished in size and of about the size of a goose-quill.* Above and below, the cord regained gradually its normal dimensions. Microscopical examination showed sclerosis of cord, with ascending and descending degeneration.—Michaud, *ibid.*, p. 70.

Observation XIV.—Male, aged thirteen years, entered hospital Sainte-Eugénie, September 16, 1869, with dorsal angular curvature and complete paraplegia; moderate contracture of legs and thighs; sensibility to touch, temperature, and pain completely abolished; reflex movements exaggerated; function of bladder and rectum normal. Died January 29, 1870.

Autopsy.—Caries of the five lower dorsal vertebræ, the apex of the angular deformity being found by the tenth. Two of the vertebræ are completely destroyed; the osseous tissue is infiltrated with tubercle. The *dura mater* is thickened at the site of the disease, showing the lesion "*pachy. externe.*" The spinal cord at the site of the lesion shows well-marked evidence of sclerosis, with dislocation of one of the anterior horns.—Michaud, *ibid.*, p. 73.

Observation XV.—Male, aged forty-two years, admitted into the service of M. Vulpian at hospital La Pitié, March 31, 1870, complaining of severe neuralgic pains in the lumbar and epigastric regions. Examination revealed angular curvature of

the spinal column at the junction of the upper and middle thirds of the dorsal region; analgesia of the lower extremities most marked upon the left side; sensibility to temperature very well preserved. Died January 18, 1870.

Autopsy.—The dura mater is considerably thickened at a point corresponding to the eighth and ninth dorsal vertebræ, and the latter present a condition of extreme friability as a result of caries; the posterior ligament is destroyed opposite the diseased vertebræ. The spinal cord at the site of the lesion does not offer a uniform alteration. Toward the superior part of the compressed segment are found tracts of sclerosis in the posterior columns following the course of the internal radical filaments. The corresponding nerve-roots present evidence of neuritis, with granulo-fatty change. At the lower part of the compressed segment is found a peripheral sclerosis, presenting a tendency to assume the annular form, most marked upon the lateral columns; the left lateral half of the gray substance is deeply changed. Above the site of the lesion is found sclerosis, which continues in the lateral columns up to the cervical region. —Michaud, *ibid.*, p. 74.

Observation XVI. — Infant, aged eight months, began gradually to lose power of the right arm, and subsequently of the left. After two months and a half, partial paralysis of the lower extremities. Died at the end of seven months after the onset of the disease.

Autopsy.—Caries of the sixth and seventh cervical vertebræ, opposite which point the cord seemed enlarged. This enlargement was due to tubercular infiltration, which at this point had caused complete absorption of the proper tissue of the cord. The tubercular mass seemed to have had its origin in the right postero and postero-lateral columns; then extended until the cord was gradually destroyed, only slight traces of the anterior columns remaining.—Gull, "Guy's Hosp. Reports," 1858, 3d series, vol. iv, p. 206.

Observation XVII.— Male, aged fifty-eight years, complained of backache for thirteen years, the pain being most severe in the lumbar region, thence radiating toward both sides; loss of power slowly developed in the lower extremities, which

soon became completely paralyzed; reflex movements markedly exaggerated; severe muscular cramps.

Autopsy.—Caries of the sixth, seventh, and eighth dorsal vertebræ. At a point opposite the posterior surface of the body of the eighth dorsal vertebra the dura mater is thickened and adherent to the bone; the vertebral canal is narrowed at this point; the intercostal nerves in the vicinity of the diseased vertebræ are imbedded in thickened callous tissue. Above and below the site of the lesion the dura mater is normal. The *spinal cord* at the site of the lesion was compressed, and smaller than that above and below. *Microscopical* examination of the same revealed absence of a portion of the nerve-fibers; medullary sheaths destroyed in places; connective tissue increased; the nuclei were but slightly increased, in no place more than two or three being found together; little fat droplets and amyloid bodies also found.—Frommann, Virchow's "Archiv," Bd. 54, S. 42.

Observation XVIII.—Male, aged thirty-seven years, fell on the 10th of October, 1885, spraining his right hand; three weeks later complained of some pain in his right shoulder, and gradually his right arm became paralyzed. Examination showed paralysis of deltoid, brachialis anticus, and biceps muscles; sensibility of arm slightly diminished; hyperæsthesia over the region of the superior cervical nerve; pupils dilated. One year after beginning of the disease general emaciation, with very pronounced atrophy of the muscles of the right arm; less of the left. Finally, complete paralysis of both upper extremities and of the muscles of the neck, with moderate anæsthesia; paresis of legs, with increased tendon reflexes. Died November 14, 1886.

Autopsy.—Bodies of the third and fourth cervical vertebræ carious; intevertebral substance destroyed; the posterior ligament is bowed with the convexity toward the spinal cord, compressing same. *Spinal cord:* 4 to 5 ctm. of the cervical enlargement softened into a reddish pulp; the *meninges* are intact; fourth cervical nerve surrounded by pus.—Grasset and Estor, "Revue de méd.," viii, 2, p. 113, 1887.

Observation XIX.—Female, aged sixty years, admitted

into Guy's Hospital, November 6, 1867, with complete paraplegia, which had been progressive from time of onset, three months previous; partial anæsthesia, but no pain. Died suddenly, ten days after admission, with symptoms of heart-failure.

Autopsy.—Third and fourth dorsal vertebræ partially destroyed by caries, with complete destruction of the intervertebral substance. At the site of the lesion a mass of softened material projected into the spinal canal, causing pressure upon the cord. Spinal membranes intact. Spinal cord diminished in size at site of compression and somewhat softened.—Ogle, "Trans. of the Path. Soc. of London," vol. xix, p. 16, 1868.

Observation XX.—Male, aged forty-one years, afflicted with caries of the middle dorsal region of the spine, complicated with paresis of both lower extremities; reflex movements markedly exaggerated. Died from phthisis complicating the carious disease.

Autopsy.—Caries of the fifth and sixth dorsal vertebræ and complete destruction of the intervertebral substance. At the site of lesion an abscess pressed upon the spinal cord. The spinal membranes were found intact, and the spinal cord presented no evidence of inflammatory change.—Ogle, *loc. cit.*

Observation XXI. — Male, aged forty-five years, complained, during year 1878, of stiffness of his neck which rendered rotation of same impossible. During the year a retro-pharyngeal abscess formed, which broke externally. Examination, January 1, 1879, showed rigidity of the upper part of the vertebral column, but no paralysis of upper or lower extremities. The only marked symptom aside from the rigidity of the muscles of the neck was profound atrophy of the *thenar eminence* and *interossei* of both hands.

Autopsy.—Posterior ligament in great part destroyed from the third dorsal to the superior cervical vertebræ; body of first dorsal and all the cervical vertebræ carious. Dura mater opposite diseased site shows the condition of "pachy. externe," the internal surface, however, remaining intact. The condition of "pachy. externe" extends laterally in the dura mater far enough

to include the anterior and posterior spinal nerve-roots traversing same.

Microscopical Examination. — The anterior nerve-roots which form the brachial plexus show the degenerative lesion—proliferation of the nuclei, atrophy, and crowding together of the myeline and axis-cylinders. This same lesion is found in the nerves which supply the atrophied muscles. The posterior spinal roots appear normal.

Spinal Cord.—Several ctm. of the middle and inferior cervical segments show: *a.* Slight sclerosis of the direct cerebellar tract. *b.* The internal root-zones of the posterior columns show a sclerotic lesion similar to that of locomotor ataxia, which lesion is prolonged into the gray substance. The motor cells of the anterior horns are also somewhat atrophied, and the cells fewer than normal. No evidence of ascending or descending degeneration in the white columns usually found consequent to lesions of the cord.—Proust and Ballet, "Revue mensuelle," vol. iv, p. 425, 1880.

Analysis of the Findings. — In examining the cases which have just been cited, we are struck with the superficial manner in which some have been reported—so imperfectly reported in some instances, even by competent observers, as to render them almost worthless beyond giving a rough description of the gross findings. Yet, inasmuch as these are valuable to us, they have been included with those where the microscopical findings are also given.

Dura Mater.—The medullary surface is found intact in all observations excepting III and VII, which showed perforation of the dura.

Pia Mater.—In Observation VIII found injected, but unaccompanied by exudation; in the remaining cases *normal.*

Spinal Cord.—Observations I, II, V, IX, XII, XIII, XIV, and XVII show the spinal cord *converted* into *con-*

nective tissue at the *site of compression.* This gives no clew to the pathology of the original lesion, since we have seen that connective-tissue growth is a result of a variety of lesions affecting nerve-tissue.

Softening of the cord at the compression site is found in Observations IV, VIII, X, XVIII, and XIX. In Observation VIII the cord was softened exactly where it was pressed upon by bone, and small hæmorrhagic foci were found in the cord substance—changes non-inflammatory, similar to changes already described, which experimental physiology tends to show take place in the cord as a result of compression. In Observation X the lesion was limited exactly to the anterior columns of the cord, and these were found *softened* and *anæmic*, the presence of the latter condition, as held by the observer, pointing to a non-inflammatory lesion. In Observation XIX, Ogle adds, with some emphasis, that the lesion at the site of compression was purely a mechanical one, as nothing indicative of inflammation was found. In Observations IV and XVIII the lesion may or may not have been inflammatory. A compression cause existed in the form of abscess products which compressed the cord. In both cases it is to be noted that the *meninges* were found *normal.* Observations VI, VII, and XX give no evidence of inflammatory destruction reported by such observers as Kadner and Ogle. In Observation XVI the cord tissue proper was found absorbed by the tubercular neoplasm. In Observation XI Michaud sums up the histological findings under the head of *beginning myelitis*, without detailing the formr.

If we carefully compare the findings in Observations XV and XXI, we will notice a marked resemblance and at once see that they differ materially from those histological changes usually met with at the site of compression. While no paralysis existed in either case, it is deemed best to refer

to them here, inasmuch as they have been thought by some authors to indicate the early lesion of so-called "compression myelitis." In Observation XXI, especially, we notice that the lesion is confined to definite tracts in the spinal cord—viz.: the posterior root-zones or columns of Burdach and gray matter adjoining, the direct cerebellar tracts, the anterior horns and nerves originating from the latter. How is such a lesion to be explained? Proust and Ballet were inclined to accept the following hypothesis: Irritation of the posterior nerve-roots excited inflammation of the posterior root-zones, which inflammation extended from thence to the parts of the cord found implicated—in other words, an *ascending neuritis*. Kahler,* in commenting on this case, calls attention to a fact which we have also observed, that usually in cases of lower cervical caries giving rise to symptoms similar to those enumerated, the reflexes of the upper extremities are preserved even when the atrophy is quite profound, and correctly infers that such phenomena are quite inconsistent with a neuritic disease of the cervical nerve-roots.

The distribution of the pathological findings to rather definite tracts of the cord argues a lesion having a definite cause and progressing in a definite order. The following hypothesis would seem to offer a satisfactory explanation: The lesion is twofold in character: 1. Alteration of the posterior root-zones and direct cerebellar tracts due to a degenerative lesion, the result of implication of the sensory roots at the site of *pachy. externe*. 2. The changes in the anterior horns are believed to be the direct result of moderate compression of the cord, and the degeneration of certain of the anterior nerve-roots necessarily secondary. The preservation of the reflexes in these cases renders it conclusive that the spinal-cord substance itself is primarily in-

* Kahler, "Prag. med. Woch.," viii, No. 49, p. 480.

volved. Even here, then, there seems to be nothing which a mechanical lesion will not readily explain.

We may add, Is there, even in those cases where the sclerosis is said to have an irregular distribution at the compressed site, anything especially incompatible with the theory that such changes are but later stages of non-inflammatory foci, induced by moderate compression of the cord? That such is the case is rather corroborated by Kahler's experiments, already dwelt upon in an earlier part of this paper.

To briefly summarize, then, we have shown:

1. That we have present a simple mechanical pressure in the form of *abscess products*, thickened *dura*, or *bone*.

2. That the inflammatory process is invariably a limited one, the inflammation of the dura mater being limited exactly to the site of the diseased vertebræ, exhibiting no tendency to extend in the membrane—nature's medullary protection.

3. That the *medullary* surface of the dura mater is almost invariably normal.

4. It follows, then, that the pressure lesion is simply mechanical, possessing no tendency to involve the spinal cord through any inherent specific characters of the carious process—a bland mechanical lesion—and the damage it inflicts commensurate with the pressure exerted.

5. That examinations of the pathological findings have, in the vast majority of cases, necessarily been confined to old cases where the lesion had existed for a very considerable period, and that such examinations usually revealed the cord largely converted into connective tissue at the site of compression.

6. The presence of sclerotic tissue at the site of compression is no evidence that the original lesion was of an

inflammatory character, since it is a well-established pathological law, to which we have alluded, that throughout the nerve-centers destruction of nerve-tissue, by processes degenerative or otherwise, is *invariably followed by increase of the connective tissue.*

7. Experimental physiology gives no evidence of an inflammatory lesion following experimental compression of the cord, microscopical examination of the same showing a few granular cells, swollen axis-cylinders and evidence of destruction of the same, foci of traumatic hæmorrhage—changes identical with those observed by Strümpell, Kahler, and others, at the site of recent compression of the human spinal cord. We do not find signs of vascular changes, of hyperæmia, of accumulation of cells about the vessels, and in the connective tissue; in other words, no evidence of those pathological changes which are considered by Leyden, Joffroy, Stricker, Schultze, and others as pathognomonic of inflammatory change.

8. A careful examination of the pathological findings of reported cases reveals to us, in the light of recent pathological knowledge, but few instances where we have reason to believe the original lesion to have been inflammatory. These were cases where the process was virulent, leading to a perforation of the dura mater, thus allowing the purulent products to come in direct contact with the cord, giving rise to a lepto-meningitis.

Finally, then, our researches have led us to conclude that the original lesion is, as a rule, non-inflammatory, and we have endeavored to demonstrate this from a pathological standpoint, support it by experimental physiology, and corroborate it by clinical manifestations.

We must await further researches in neuro-pathology to clear away certain obstacles which we recognize exist. When the pathology of the so-called combined sclerosis is thor-

oughly understood, together with all the facts pertaining to the physiological and pathological rôle played by the posterior root ganglia and the vascular mechanism of the cord, it will doubtless be possible to complete the pathological picture of this peculiar form of paralysis, and establish as certainties what at present exist but as probable hypotheses.

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