THE RÔLE OF THE DENTATE LIGAMENTS IN SPINAL CORD COMPRESSION AND THE SYNDROME OF LATERAL SCLEROSIS*

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The importance of the dentate ligaments in the pathological physiology of spinal cord compression has not been sufficiently stressed. I believe there are cases of anterior spinal cord compression that have been mistakenly diagnosed as primary lateral sclerosis chiefly because of the rôle played by the dentate ligaments.

Wechsler and Brody in a recent article state as their opinion that “selectivity” on the pyramidal tracts “by a noxious agent, whatever its nature,” accounts for a disease entity, primary lateral sclerosis. Of their 61 cases recorded in the past ten years with the tentative diagnosis of lateral sclerosis at least 34 later evolved as other syndromes, including spinal cord compression. It is not the purpose of this article to attempt solution of the problem as to whether there is a disease entity of primary lateral sclerosis, but rather to show how a condition similar clinically can have a mechanical origin.

Case 1. A man aged 50 showed the typical clinical picture of lateral sclerosis. There was no subjective history of pain or involvement of the upper extremities. Spastic paralysis of the lower extremities with numbness and tingling† had progressed gradually over a period of 14 months until the patient was no longer able to walk. There recently had been noted difficulty in voiding while standing but none while lying down.

Examination. There was marked spastic paralysis of both lower extremities with bilaterally positive Babinski’s sign. Tactile and pain sense were entirely normal including the sensation of heat and cold in the bladder. Vibratory sense was diminished bilaterally, more marked on the right leg. Both sense of motion and position of the toes were normal. The upper extremities were normal. Blood studies, including Kahn test, were negative. X-rays of the cervico-dorsal spine, although not very satisfactory, were thought to be negative except for arthritic changes. Review at a later date when the diagnosis was established showed evidence of calcification in the intervertebral disc between the 6th and 7th cervical bodies.

Lumbar puncture revealed no block but a total protein of 118 mg. per 100 cc. Spinal fluid Kahn test was negative. A myelogram disclosed a suggestive defect between the 6th and 7th cervical bodies. Operation was advised with the diagnosis of a midline herniation of the nucleus pulposus.

Operation. On Dec. 19, 1945, the 5th through the 7th cervical spinous processes and laminae were removed. An extradural approach revealed a marked posterior protrusion of the intervertebral disc in the midline between C-6 and C-7. The dura was opened. The cord was found to be firmly anchored to the anterior mass by the dural attachments of the dentate ligaments. These attachments were divided, mobilizing the cord. The cord was retracted laterally and an incision made through the anterior dura and posterior longitudinal ligament into

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† Subjective numbness may be complained of if either tactile or pain and temperature, or even articular or vibratory sense, has been diminished or lost (Elsberg).†
the herniated disc. Only a small amount of calcified nucleus pulposus was removed, and this with difficulty. It was thought that the cutting of the dentate attachments alone, thus decompressing the cord backward, would relieve the patient's symptoms.

Course. Immediately postoperatively the patient was very much worse and both hands were extremely weak. The strength of the hands improved rapidly but improvement of the legs was slow. A perineal prostatectomy was performed on Feb. 22, 1946, for benign prostatic hypertrophy. Examination 14 months after operation revealed improvement in that he could stand alone and walk with crutches, neither of which was he able to do preoperatively.

Discussion. Nothing was accomplished in the removal of this small amount of calcified disc substance, and the cord was traumatized during the procedure. Had the mass been the usual soft midline herniated nucleus pulposus, its excision would have been indicated. Excision of a calcified nucleus pulposus from in front of the cord is fraught with danger. Some years ago the author removed such a disc from the dorsal region of a man, who promptly developed a permanent incontinence with increase in his spastic paralysis. Freeing the dentate attachments and allowing the cord to escape backward might have resulted in cure, as the calcified mass would never have expanded farther.

Case 2. A woman aged 36 entered the University Hospital Aug. 20, 1946, with a chief complaint of weakness of the hands and legs. The patient had been well until October 1945, when she noted numbness and tingling in the hands. In November 1945, weakness of the hands developed. In March 1946, weakness and numbness of the legs were noted, with difficulty in walking. There was never any pain or urinary disturbance. Examination elsewhere at that time revealed a hemoglobin of 65 per cent and lumbar puncture was said to be normal. She was treated with liver extract with some improvement.

Past history revealed that 8 years previously she had struck her head in diving. A month later severe stiff neck developed and was said to have been relieved by a chiropractor. Shortly after, stiff neck again developed but disappeared after a series of chiropractic manipulations, never to occur again.

Examination. She was an obese woman walking with a markedly spastic gait. There was weakness in both hands with a suggestion of atrophy. Hoffmann's sign was bilaterally present. Biceps and triceps jerks were active. The abdominal reflexes were obtained. The knee and ankle jerks were hyperactive. Babinski's sign was present bilaterally and there was marked spasticity of the legs.

Sensory examination revealed normal pain and temperature sense. Touch and sense of motion and position of the toes were normal. Vibratory sense was diminished bilaterally. There was astereognosis of the hands, though sense of motion and position of the fingers was normal. No definite sensory level could be established.

X-rays of the cervical spine were essentially negative. Lumbar puncture revealed no evidence of block. The spinal fluid protein was 67 mg. per 100 cc. A myelogram was carried out using 2 cc. of lipiodol. A block was present between the 4th and 5th cervical vertebral bodies. Lateral x-ray studies with the patient in the head-down, prone position suggested that the compression arose anteriorly.

Operation. On Sept. 23, 1946, under intratracheal anaesthesia, the laminae of the 3rd, 4th and 5th cervical vertebrae were removed. There was no abnormality seen on exposure or palpation of the dura. The dura was opened leaving the arachnoid intact. There was the usual amount of fluid visible in the subarachnoid space, beneath which the cord could be seen. The dentate ligament was identified on the right and two attachments cut. No abnormality was seen on the right, lateral or anterior to the spinal cord. On the left the dentate ligament was firmly attached to the dura, and fixed to a soft extradural swelling. The dentate attachment was divided, allowing the cord to be easily mobilized posteriorly and to the right. This
had been impossible before this dentate attachment was divided. A smooth bulging mass was seen lateral and anterior to the spinal cord. An incision was made through the anterior dura over the swelling, which extended to the midline. Herniated disc substance immediately appeared. Four free and separate pieces were removed with slight traction. The entire material when assembled as one piece was about the size of a date pit. The cord now lay perfectly free, another dentate attachment above on the left being divided. The cord itself seemed somewhat flattened but there appeared to be no pressure on the cervical nerve root as the apex of the swelling appeared between the nerve roots, and directly over a dentate attachment. The dura was closed with a continuous silk suture and muscles were closed in the usual manner with silk.

Course. Less than 6 hours postoperatively patient stated that the strength in the hands had increased and this seemed to be borne out by examination. The following day the spasticity in the legs was less marked. She was discharged the 14th postoperative day, greatly improved. Examination 5 months postoperative revealed complete recovery.

Discussion. It is my opinion that similar cases are not rare and the following is an attempt to explain how pressure exerted anterior to the cord could result in a syndrome of lateral sclerosis.*

Gasser and Erlanger, in studying action potential waves in peripheral nerves, classified nerve fibers into three groups, A, B, and C, according to strength of stimulus necessary to produce the wave, and the speed of conduction. The A fibers were the largest in diameter, were the most irritable, and conducted at the greatest speed. The B fibers were smaller, conducted at less speed, and probably carry rapidly conducted or "first pain" of Lewis and Po chin. The C fibers were extremely small, probably unmedullated, conducted very slowly, and many of them presumably correspond to the "second or delayed pain fibers" of Lewis and Po chin.

Gasser and Erlanger found that on compression of a peripheral nerve the fibers were affected in the order of their size, the large fibers being the first to lose their conductivity. Thus, motor, vibratory sense, and touch fibers lose their conductivity before pain. Wortis et al., studying loss of function of a lower limb subjected to acute anoxia, found that touch and vibratory sense, and depression of ankle jerk were lost in the human before sense of position and pain. "First pain," carried by larger fibers than "second pain," was always affected first. Zotterman, distending the blood pressure cuff above the systolic pressure and leaving it on the upper arm for 40 minutes, was able to produce a differential anaesthesia of the hand and fingers. At a certain stage only pain sensation remained, and its rate of conduction was very slow, corresponding to the C fibers of Gasser and Erlanger.

Gasser and Erlanger accounted for the greater effect on fibers of large diameter, namely, motor, vibratory, position sense and touch, by analogy

* Dr. I. S. Wechsler has kindly sent me abstracts of 4 of his cases in which a surgical spinal cord lesion in the cervical region mimicked degenerative spinal cord disease. Two of the patients were men with a syndrome suggestive of amyotrophic lateral sclerosis without bulbar signs. It was atypical in that both patients had pain in the shoulders and increased spinal fluid protein with arrest of the contrast medium at the 5th cervical vertebra. One had a herniated intervertebral disc between the 5th and 6th cervical bodies, and the other a meningioma anterolateral to the cord. Both showed postoperative improvement. These cases will be reported in detail from the Mount Sinai Hospital in New York.
with the effect of pressure on thin-walled tubes. It is a well known law of mechanics that under pressure the large tubes would collapse before the small ones. It is not certain, however, that nerve fibers with their fat-like myelin sheaths act as tubes. If they act as solid cylinders instead of tubes, the effect is known to be mechanically opposite, the smaller fibers suffering the greater damage. It is possible, however, that the smaller fibers might be protected in the interstices of the larger fibers which have so much greater a surface area. A fiber with a diameter of 10μ covers a surface area one hundred times as great as does a fiber of 1μ.

Another explanation seems more plausible. Gerard observed that when nerves are subjected to anoxia in a closed chamber, fibers are not blocked in a homogeneous or continuous fashion but fall out in groups, probably determined by fiber diameter. In this type of experiment the block occurs first in groups that have a higher rate of metabolism. Replacement of the fluid in which the fibers are bathed has no effect in preventing or delaying block. This indicates that block is due to exhaustion of an oxidizing reserve and militates against the supposition that it is due to the accumulation of metabolites. Fulton also states that fibers of larger diameter are more subject to lack of oxygen than the smaller fibers. The principle is of considerable importance in the interpretation of in vivo asphyxia experiments, according to Wortis et al. The work of Wortis et al. suggests that those portions of the peripheral nerves that conduct impulses at higher rates of speed and that are most irritable have the highest metabolic requirements and are most susceptible to metabolic disturbances.

It seems only logical, and there is good evidence to show, that the fibers that make up the long ascending and descending tracts of the spinal cord are roughly proportional in size to their corresponding peripheral nerve fibers. Häggqvist, after careful study, believes that finely medullated fibers with diameter of less than 2μ are the conductors of pain sensation in the spinal cord. He found the spinothalamic tracts to be made up of relatively fine fibers and the posterior funiculi of coarser fibers. Häggqvist also concluded that the fibers of the pyramidal tract have a diameter varying from 5 to 21μ. Sjöqvist states: “Axis cylinders which are large outside of the central nervous system will remain large also in the medulla.”

If, then, fiber size in the spinal cord is proportional to corresponding fibers in peripheral nerves, compression applied to the spinal cord should affect conductivity of fibers of large size first, whether it acts as if the fibers were a series of tubes, which is doubtful, or whether the pressure results in the anoxia of ischemia. Thus, motor, vibratory sense, and touch fibers would be affected before the minute pain fibers, were the pressure transmitted equally in all directions. The strong attachment of the dentate ligaments in anterior spinal cord compression prevents this equalized transmission of pressure for a time at least. Stress analysis by Mr. Alfred Zweig of Albert Kahn, Associated Architects and Engineers, shows that on anterior spinal cord compression, the cord being firmly fixed by the dentate liga-
ments, the first pressure wave is farthest anterior. There is, however, a secondary wave of pressure lateral in the cord and running posterior to the dentate ligaments which would act directly on the pyramidal tracts. Least stress of all is placed on the posterior columns (Fig. 1).

In anterior spinal cord compression I believe that, with pressure over a period of time, the pyramidal tracts, because of the greater stress on them and the large size of their fibers, have more disturbance of conductivity than the pain fibers of the spinothalamic tracts, even though the latter are closer to the compressing mass, be it midline herniated nucleus pulposus or tumor. Touch is preserved because, even though the touch fibers of the ventral spinothalamic tracts may fail to conduct, there is still sufficient sensation carried in the more protected posterior columns to prevent the clinical detection of touch disturbance. Postural sense is preserved because the attachment of the dentate ligaments prevents the posterior columns, which are farthest from the compressing mass, from being pressed against the unyielding laminae.* Thus may be produced a clinical, though not necessarily a pathological

* Further evidence that the dentate ligaments prevent the posterior columns from being pushed backwards is the presence of normal epidural fat over the lesion.
picture of lateral sclerosis with pyramidal tract disturbance and little or no sensory change.

I should like to discuss the subject of early versus late spinal cord compression. In the ischemia of early compression the factor of anoxia with disturbance in conductivity in the larger fibers must come into play before true physical damage to these same fibers; this means physiological or functional before anatomical disturbance and is a reversible reaction. Thus is explained the rapid return in motor power sometimes seen after relief of spinal cord compression (Case 2) a few hours postoperatively, with return to normal a few days later. This can only mean change in conductivity in otherwise normal fibers. With long-continued pressure, however, pathological change takes place in nerve fibers because of direct mechanical effect on "tubes or cylinders" and a true sclerosis with gradual disappearance of myelin sheaths and axis cylinders and their replacement by glial tissue is seen. This is an irreversible process and explains the poor results seen in operations for long-continued spinal cord compression.

The Brown-Séquard syndrome in spinal cord compression is, I believe, usually caused by a laterally placed, extramedullary, intradural tumor anterior to the dentate ligament. The picture at operation is familiar to all neurosurgeons. The cord is seen to be firmly anchored to the dura on the side of the tumor by the dentate ligament, which forms an arc over the tumor, with its convexity posterior. The lateral attachment of the dentate ligament must be freed before the tumor can be approached.

Dr. Rollo McCotter, Professor of Anatomy at the University of Michigan, has suggested to me that the dentate ligaments and their attachments on being subjected to a long period of abnormal stress may increase in strength and size. This type of ligamentous change is commonly seen in orthopedic conditions.

It is obvious that pressure transmitted to the cord in this way is not equally distributed. The greatest pressure is applied directly to the spinothalamic tract and next to the pyramidal tract of the same side. The effect on the posterior columns is unimportant (Fig. 2).

A reverse Brown-Séquard syndrome is sometimes seen. This has been attributed to lateral pressure on the spinal cord by tumor, with impingement of the spinal cord on the unyielding bony wall of the opposite side. In other words, the tumor being softer than the vertebral wall, more damage is caused to the cord by the latter. Newton's Law of Action and Reaction, which states that if a force is exerted it is resisted by an equal and opposite force, shows that this conception is wrong. However, if a force were exerted over a wide surface area and were resisted by a smaller surface area, the greater force would be exerted over the smaller surface area. I believe that this is what happens in the reverse Brown-Séquard syndrome: There is lateral pressure exerted extradurally by tumor over the area between dural prolongations which carry the nerve roots. These prolongations might resist lateral displacement at their point of attachment to the dura but the cord could be
displaced laterally between them. Here the dentate ligaments would play no rôle, as the dura with its enclosed cord, membranes and ligaments would be bodily moved across the vertebral canal. The surface area of the tumor exerting its force on the spinal cord would be broader than the area of impingement on the bony canal, and a greater local force would be exerted on the cord at that point (Fig. 3). The reverse Brown-Séquard syndrome is more

likely to occur in the dorsal region where the canal is so much narrower than in the cervical region. Elsberg\(^1\) mentions the reverse Brown-Séquard syndrome and the one case he describes in his book *Tumors of the Spinal Cord* is that of an extradural tumor in the dorsal region.

The argument might be used that the herniated intervertebral disc would act as an anterior extradural tumor and move the dura with its contents backwards *en masse* so that the dentate ligaments would play no part. Anatomical studies, however, show that the dural prolongations strongly resist posterior displacement of the dura and provide a fixed point from which the dentate attachments can act. The intervertebral disc lies at exactly the level of the dural prolongations.

In closing, I should like to digress and consider a spinal cord condition

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*Fig. 2. Author's conception of usual Brown-Séquard syndrome. The stress on the pyramidal tract is almost as great as on the lateral spinothalamic.*
that does not have a mechanical basis, the posterolateral sclerosis of pernicious anemia. There is unanimity of opinion today that this degenerative condition is caused by a metabolic disturbance. Greenfield and Carmichael showed that there was an effect on the peripheral nerves here as well as on the cord. They showed that fibers in the peripheral nerves of large size were more affected than those of smaller size. This also holds true in the spinal cord, as the posterior columns and pyramidal tracts are more affected than the spinothalamic tracts. Moreover, the dorsal spinocerebellar tracts, which Ranson explicitly states are made up of fibers of uniformly large size, are also involved. Is it then possible that this metabolic disturbance acts chiefly on the large fibers of the spinal cord with their greater metabolic requirement and thus ultimately produces the pathological picture seen in posterolateral sclerosis? An explanation would have to be found for the fact that the direct pyramidal tracts do not seem to be involved, though this might be due to a difference in blood supply.

SUMMARY

1. Gasser and Erlanger, studying action potential waves, found that on compression of a peripheral nerve, the fibers were affected in order of their size, the larger fibers being the first to lose their conductivity.

2. The larger fibers with their greater irritability and their greater rate of conduction have higher metabolic requirements than the smaller fibers.
They, therefore, more quickly reflect the metabolic disturbance caused by the ischemia of compression with its resultant anoxia.

3. Fiber size in the tracts of the spinal cord is grossly proportional to corresponding fibers in peripheral nerves.

4. The changes in conductivity found on compression of a peripheral nerve should hold for the spinal cord were the pressure equally distributed. In anterior spinal cord compression, however, the strong attachments of the dentate ligaments may prevent this equal distribution of pressure and produce the syndrome of lateral sclerosis.

5. The suggestion is put forth that the disturbance in metabolism which produces degeneration in the posterolateral sclerosis of pernicious anemia, does so by acting on the large fibers of the posterior columns and pyramidal tracts, with their higher metabolic requirements.

REFERENCES
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15. Zweig, A. Personal communication.