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BY

GEORGE J. PRESTON, M.D.,

PROFESSOR OF PHYSIOLOGY AND DISEASES OF THE NERVOUS SYSTEM, COLLEGE  
OF PHYSICIANS AND SURGEONS, BALTIMORE.

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**TRAUMATIC LESIONS OF THE SPINAL CORD.<sup>1</sup>**

BY GEORGE J. PRESTON, M.D.,  
PROFESSOR OF PHYSIOLOGY AND DISEASES OF THE NERVOUS SYSTEM,  
COLLEGE OF PHYSICIANS AND SURGEONS, BALTIMORE.

INJURY to the cord, resulting from external violence, is of rather more frequent occurrence than would be expected if we consider how well protected the cord is. The peculiar construction of the vertebral canal, the strength of the individual vertebræ, the very perfect and extensive articulations with each other, the elastic intervertebral discs separating the segments, and the various ligaments binding the parts together, require a high degree of violence to produce either dislocation or fracture. Added to this the spinal canal is much larger than its contained cord, and the latter is consequently not in direct contact with the bony walls, but is suspended from above, and steadied laterally by ligaments and emerging nerves.

The most common form of injury to the cord is that which results from fracture of the vertebræ. This accident occurs as the result of a general crush, as by a fall of coal in a mine, or of earth or stone in a tunnel or quarry; also by external violence applied at

<sup>1</sup> Read before the Clinical Society of Maryland, December 16, 1892.



one point, as when a man is caught between cars. Not an uncommon cause is a fall, which may cause fracture of the vertebræ either directly, as when an individual falls flat upon the back, or indirectly from striking upon the head or feet, the force of the fall being felt most strongly at certain points along the vertebral column. Extreme flexion of the body, or twists, may produce dislocation or even fracture. Gunshot wounds involving the vertebræ, while not very common in civil, are often met with in military practice.

As a result of these and similar injuries to the bony structures, the cord may be compressed, more or less lacerated, or entirely destroyed. Compression may be occasioned either by the broken bone impinging directly upon the cord in the shape of a fragment or spicule, or by a narrowing of the canal. The points at which fracture is most likely to occur are the cervical and lower dorsal regions, unfortunately the very points where the cord can least afford interference with its function. Compression of the cord may result from hemorrhage within the spinal canal, due to rupture of the spinal veins. Finally, when the cord itself is not injured, the meninges may be torn and the cord suffer secondarily.

In addition to the class of cases already referred to must be added those cases in which, without any fracture or dislocation of the vertebræ, the cord suffers more or less severely. Concussion of the *spine*, or *railway spine*, as this condition is wrongly called, has occasioned a good deal of rather acrimonious debate, largely due to the

fact that the subject has been so often in the courts. While concussion of the cord is infinitely less common than concussion of the brain, it undoubtedly does occur, and we may have all grades of injury, from a mere temporary suspension of function up to a considerable degree of laceration, without fracture of the vertebræ.

The symptomatology of injury of the cord is co-extensive with the functions of the cord, as any part of the latter may be involved. As far as location goes, we need consider only the cervical enlargement, the mid-dorsal region, and the lumbar enlargement.

In the cervical and lumbar regions are situated the important centers for the muscles of the upper and lower extremities and for the thoracic and abdominal viscera, and entering these portions of the cord are the sensory nerves from the extremities. Injury may therefore involve the nerve-centers or cells, or the white matter alone. In the dorsal region the gray matter sinks into insignificance, and we are concerned mainly with the extent of the involvement of the white matter or fiber-tracts in their passage to and from the brain.

The symptoms of injury to the cord may be divided into primary and secondary. By primary symptoms are meant those that appear suddenly, as the immediate result of the injury—such, for example, as paraplegia. By secondary symptoms are meant those that come on at a later period in the form of degeneration of fiber and cell; this class is illustrated by the various muscular atrophies and trophic lesions generally. In discussing briefly the

primary symptoms of injury to the cord, it is necessary first to note those symptoms of a general nature and not dependent upon the location of the injury, and then the special symptoms that depend upon the region involved and the extent of such involvement.

The general symptoms of injury to the cord are few. Shock is present to a greater or less degree, sometimes being marked after slight injuries, and at other times present only to a slight degree, though the injury may have been severe. Consciousness is rarely lost, even in cases proving rapidly fatal. Of particular interest is the rise of temperature, noted especially in injury to the cervical cord, and, in lesser degree, in injuries in the lower dorsal and lumbar regions. No very satisfactory explanation of this phenomenon can be given. If it were met with only after injury to the cervical cord it might be explained on the ground of interference with the heat centers, supposed by some physiologists to be located in that region; but as injury at any level may produce rise of temperature, we must conclude that this is due to interference with the fiber-tracts connected with the heat-regulating centers. Varying degrees of pain may be present, depending upon the location and extent of the injury and the involvement of nerve-roots. Priapism is a common symptom, especially in injuries to the cervical cord. Rosenthal mentions a case of injury to the cervical cord in which the priapism lasted for seven days during life and for thirty-six hours after death.

In discussing the special symptoms, it is important

to bear in mind the fact that we may have all grades of injury, and consequently the intensity of the motor and sensory symptoms will vary accordingly. Again, the cord may be involved to a greater degree on one side than on the other, causing a difference in the symptoms of the two sides. When the middle of the cervical enlargement is involved, somewhere about the sixth or seventh cervical vertebra, there is loss of motion in all parts below the level of the lesion, and of sensation over the inner half of the arm and forearm and all below this level. The temperature is always notably raised, the pupils are contracted, and the breathing is diaphragmatic. Lesions high up in the cervical region cause loss of motion and sensation from the neck down, interfere seriously with respiration, and are usually rapidly fatal. The two following cases illustrate very well the symptoms of injury to the cervical cord: the first case is one of injury to the upper part of the cervical region; the second, of injury about the mid-cervical region.

CASE I.—A male, aged twenty-four, gave a history of having plunged head first into shallow water. When admitted into the hospital, some ten or twelve hours after the accident, examination showed no signs of injury to the head. The head was somewhat retracted and any movement of it caused intense pain. All four extremities were paralyzed, the lower completely, the upper being capable of some slight movement. Sensation was lost from the neck down. Breathing was purely diaphragmatic. The patellar reflex was present, and the pupils were contracted to pin-points. The temperature was  $104^{\circ}$  F. The mind was perfectly clear. There was priapism and loss of control over the bladder and

rectum. Death occurred on the evening of the day he was admitted into the hospital, which was the day following the reception of the injury. The temperature, taken in the rectum four hours after death, was  $110^{\circ}$  F. The autopsy revealed a fracture of the fifth cervical vertebra. The cord showed laceration at the point of fracture, with extravasation of blood extending about an inch vertically.

CASE II.—A male, aged thirty-seven, fell about forty feet upon a rough, irregular surface. Examination shortly after the accident showed a lacerated wound of the scalp, but no fracture of the skull. He was perfectly conscious. There was complete paralysis of the lower extremities and partial paralysis of the upper. Sensation was entirely lost from the level of the third rib down, and impaired over the chest and arms. Respiration was diaphragmatic. Priapism was marked. There was paralysis of the bladder and rectum. The pupils were finely contracted. The temperature,  $103^{\circ}$  F. an hour after admission, reached  $109^{\circ}$  F. just before death, which took place the day following the reception of the injury.

At the autopsy it was seen that the laminæ of the fourth and fifth cervical vertebræ were fractured on both sides; the dura was slit longitudinally and there was an extra-dural hemorrhage posteriorly. The cord opposite the fracture showed hemorrhage and disintegration of its substance.

Lesions in the mid-dorsal region, if complete, cause loss of motion and sensation below the seat of injury. If the lesion be high enough, the arms may be slightly affected both as to motion and sensation, thus approximating the symptoms detailed. When the lesion is only partial, the differences between dorsal lesions on the one hand and cervical

or lumbar on the other, become much more apparent. This is very obvious if we consider that in the dorsal cord the gray matter and consequently the centers are very insignificant, the white matter or fibers composing almost the entire cord between the cervical and lumbar enlargements.

A lesion in the lumbar region, involving the lumbar enlargement, causes loss of motion and sensation in all parts below, loss of control over the bladder and rectum, and loss of the superficial and deep reflexes. It must be understood, of course, that the symptoms here detailed are only the immediate ones; the secondary symptoms, such as atrophy and the like, will be mentioned under the head of secondary symptoms. The following cases may be mentioned as illustrative:

CASE III.—A male, aged twenty-three, was crushed by a bank of earth falling upon him. There was no external injury of note. There was entire loss of motion and sensation below the level of the first lumbar vertebra, and loss of control over the bladder and rectum. There was no loss of consciousness at any time. The superficial and deep reflexes were abolished. The temperature on the day of admission to hospital, which was the day of the injury, was 102° F.; it remained at this point for five days, then sank to normal and did not rise again until just prior to death. Bedsores of a trophic nature developed in the gluteal region, and cystitis was also a later complication. The patient was admitted March 26th, and died rather unexpectedly May 15th. The autopsy showed that the last dorsal and first lumbar vertebræ were crushed and dislocated forward, compressing the cord and reducing it to an almost empty sheath.

CASE IV.—A male, aged fifty-two, fell nine or ten feet into the hold of a vessel, striking upon his back and side. There was no loss of consciousness and very little pain, but there was instant paralysis of the lower extremities. Examination showed a scalp-wound, but no fracture of the skull. There was a marked projection of the tenth dorsal vertebra. There was total loss of power in the lower extremities and loss of sensation below the umbilicus.

Bladder and rectum were both paralyzed. Cystitis, together with bedsores of a trophic nature, developed, and the patient died September 10th, having been admitted August 25th. The autopsy revealed a fracture of the tenth dorsal vertebra, greatly compressing and crushing the cord.

Injury to the *conus medullaris* causes paralysis of the bladder and rectum and anesthesia in the gluteal region. Such cases are not very common. The symptoms detailed and cases related are applicable only to total or nearly total transverse lesions, conditions in which the functions of the cord at the level of the injury are entirely abolished. It often happens, however, that the injury does not involve the entire thickness of the cord. In this latter case the symptoms present wide variations. Sensation may be greatly or very slightly affected, and all grades of paralysis may exist. A partial lesion in the mid-dorsal region may not involve the sensory conducting-fibers to any appreciable extent, and the lower centers, as those for the bladder and rectum, may be intact. Motion in such a case as this would be interfered with to a moderate degree only, or more or less entirely lost.

We may have one lateral half of the cord inter-

ferred with, giving Brown-Séquad paralysis: motion lost on the side of the lesion, sensation on the opposite side. In the cases in which the lesion does not involve the entire thickness of the cord, the patellar reflex is as a rule greatly exaggerated, unless the lesion be in the lumbar enlargement, when, of course, the knee-jerk is lost, because the center is involved and the reflex arc is broken. In those cases in which the cord is entirely severed, even though the lesion is in the cervical or dorsal region, the patellar reflex, as Bastian has pointed out, is lost and we have a flaccid paralysis. A case recently under my care showed the curious combination of entire loss of motion and sensation in the lower extremities, with greatly exaggerated reflexes and ankle clonus. The only explanation of such symptoms that can be given is that the cord was involved in almost its entire thickness, having intact only a few white fiber-tracts, probably the direct cerebellar columns. It must be borne in mind that occasionally the nerve-roots only are involved, without any injury to the cord: the local nature of the symptoms renders the diagnosis easy, as a rule.

In considering the cases in which only moderate injury is done to the cord, a question of great interest arises, namely: To what extent may the cord be injured by blows, falls, and the like, which do not cause fracture or dislocation of the vertebræ? We may, I think, have three varieties of cord-injury resulting from external violence which has not been sufficiently great to cause any rupture of the bony parts: laceration to a moderate degree, compression from hemorrhage due to rupture of the spinal veins, and

finally, a general disturbance of nutrition, without any immediate alteration of structure or marked symptoms, the condition known as concussion proper. The following case will illustrate the first variety, or moderate laceration without any fracture or dislocation of the vertebræ:

CASE V.—A female, aged twenty-two, on July 25th fell through a skylight to the floor beneath, a distance of fourteen feet. There was no loss of consciousness and but little shock. Examination showed a bruised and somewhat tender spot in the lumbar region, but no evidence of fracture or dislocation.

The temperature was normal, as also were the pupillary reflexes. There was complete paralysis of the lower extremities and of the bladder and rectum. The deep and superficial reflexes, from the waist down, were abolished. There was anesthesia of the gluteal region and of nearly all the surface of the lower extremities. The patient has slowly improved, and at present, nearly six months after the injury, has recovered to some extent the use of her right leg. The left leg is still nearly powerless, and shows marked foot-drop, and only partial return of sensibility. The reflexes are very feeble.

The second variety of compression by intra-spinal hemorrhage without fracture is interesting, and has been confirmed by surgical operations for the relief of the pressure occasioned by the hemorrhage; the bleeding generally is found to come from the rupture of the spinal veins. The following case might perhaps be included in this category.

CASE VI.—A male, aged sixty-two, was crushed in an elevator. Several ribs were broken, but no

distinct fracture of the vertebræ could be made out. There was no displacement of the vertebræ. There was almost total loss of motion in the lower extremities, with anesthesia extending to the crests of the ilia, and above this a very sharply-defined band of hyperesthesia. There was no elevation of temperature. The patellar reflex was abolished; the bladder was not paralyzed. Examination on the following day showed that the hyperesthesia had disappeared, and in a few days motion and sensation began to return and the patient was discharged cured at the expiration of two weeks. The deep reflexes had not returned when the patient left the hospital.

The third variety of injury to the cord, without fracture of the vertebræ, or any evidence of gross lesion, is that condition known as concussion of the spine. The limits of this paper do not permit any discussion of this interesting part of the general subject of injury to the cord. Ever since the appearance of Erichsen's notable paper, there have been bitter discussions in regard to the existence of such a condition. Unfortunately, the battle-grounds have been oftener in the courts than in the medical societies.

While a certain proportion of the cases of "railway spine" are fraudulent, or at least imaginary, and the symptoms stimulated by prospective "damages," there can be no doubt of the genuineness of many cases. Nor is the probable pathology hard to conceive. As we have seen, the cord may be injured severely by a blow or a fall that does not cause fracture or displacement of the vertebræ. Now, it would seem not improbable that a severe fall might seriously disturb the nutrition of certain parts of

the cord, causing injury much less in degree than laceration or hemorrhage, which have already been considered.

As the result of a concussion, there might be sufficient injury done to the gray matter to initiate a degenerative process in the cellular structure, and, as a consequence, muscular weakness and atrophy in the parts depending for their innervation and trophic influence upon the cells in question.

Again, if the fiber tracts be even slightly injured, a myelitis may result and cause a more or less widespread secondary degeneration. Suppose an injury, to the posterior part of the cord, by a fall, blow, or twist; the immediate effects of such an injury may not be at all marked, yet if there has been decided damage done to the fiber-tracts there will be an upward degeneration of the posterior columns, or the pathologic lesion of *tabes dorsalis*. The specimen shown of Case II. very beautifully illustrates this point. The injury to the cord was in the lumbar region, and the cervical cord shows most marked degeneration of the posterior columns, as can be seen from the section exhibited. This very marked degeneration has taken place in two months. In like manner we may have degeneration of the lateral, the direct cerebellar columns, etc.

It is, I think, quite possible that we have underestimated the importance of traumatism, in searching for some satisfactory etiology of the chronic system-degenerations or scleroses of the spinal cord. As a matter of fact, we have reports of cases pointing clearly to traumatism as their starting-point, and it is a common observation that an apparently trivial

accident will often hasten and develop a case of incipient sclerosis.

The foregoing remarks sufficiently illustrate the nature of the changes of a secondary character that take place in the cord. As a result of these secondary degenerations, we have muscular atrophy, contractures, bedsores, cystitis, and like symptoms. Cystitis is especially troublesome and very commonly leads to secondary kidney-changes.

The diagnosis of these cases is not generally difficult, but, as Gray has suggested, it is always well to write out carefully all the symptoms, so that we may compare them with the tables given in the books and express the limits of the involvement of the cord in terms of the vertebræ.

The question of treatment in these cases of traumatic injury of the cord is an exceedingly difficult one to determine. As we know, we can expect little from medicine. Rest, counter-irritation, electricity, gold and silver salts, mercury, arsenic, and many other therapeutic agents are employed, but it is very questionable whether any of them influence in any degree the morbid processes. Undoubtedly, some cases do recover, but everything probably depends upon the degree of injury and the subsidence of the compression. The prognosis in those cases in which the cord is crushed and its whole substance destroyed is, if treated medically, absolutely hopeless. Such cases either die at once, or drag out a miserable existence through a few months and die of exhaustion from bedsores or secondary kidney-changes set up by the almost inevitable cystitis.

All we can do is to nourish the patient, to keep

the bladder washed out, and to minimize as far as possible the evil effect of the bedsores, which nearly always occur, in spite of the most scrupulous care, and which, in most cases, must be regarded as trophic lesions. Such being the gloomy outlook in the medical treatment of these cases, we naturally turn to surgery.

It is out of place in this paper to discuss this part of the subject, but a few considerations as to the class of cases suitable for operation may be permitted. There is hardly any question about the propriety of operating when we have cases in which there are marked signs of compression, without evidence of total destruction of the cord. The main points to be regarded in making the differential diagnosis between partial and total destruction of the cord have already been considered. A considerable number of cases successfully operated upon attest the correctness of this position. If the compressing agency be removed early, the cord may recover in great part, but if operation is long delayed, secondary degeneration is likely to set in and interference to be rendered useless.

In those cases in which there is total destruction of the cord, most neurologists advise strongly against any operation as useless. It has always seemed to me that, in view of the fact that these cases practically always prove fatal under medical treatment, we should not advise against what would seem to be a natural surgical procedure. We have broken bone, crushing and compressing the cord, and the most natural thing would be to clear away the débris and give a chance to whatever bit of cord there

might be left uninjured. Observations in a number of autopsies of cases that presented all the symptoms of complete crush of the cord have convinced me that it is rare to see the cord entirely destroyed by the traumatism, and in a considerable proportion of these cases there was distinct evidence that part of the damage was due to secondary inflammatory processes. Operative procedure offers not merely the best chance, but in nearly all of these cases the *only* chance, and if properly carried out does not materially add to the gravity of the prognosis, even if it accomplishes nothing. Abbe's brilliant work upon the cord, while as yet rather barren of results, is full of suggestion. As has been already noted, an operation, to be of any avail, must be done early. We should allow only time enough to make it certain that the symptoms are not due to shock or temporary interference with function. I know of no class of cases that more strongly appeal to me than these cases of injury to the cord, for I always feel instinctively that something ought to be done for them, and I am confident that the surgery of the future will make this possible.





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