

Missouri Law Review

Volume 11
Issue 2 April 1946

Article 2

1946

Medico-Legal Aspects of Spinal Cord Injuries

Foster Kennedy

Peter G. Denker

Follow this and additional works at: <https://scholarship.law.missouri.edu/mlr>

 Part of the [Law Commons](#)

Recommended Citation

Foster Kennedy and Peter G. Denker, *Medico-Legal Aspects of Spinal Cord Injuries*, 11 Mo. L. REV. (1946)
Available at: <https://scholarship.law.missouri.edu/mlr/vol11/iss2/2>

This Article is brought to you for free and open access by the Law Journals at University of Missouri School of Law Scholarship Repository. It has been accepted for inclusion in Missouri Law Review by an authorized editor of University of Missouri School of Law Scholarship Repository. For more information, please contact bassettcw@missouri.edu.

MEDICO-LEGAL ASPECTS OF SPINAL CORD INJURIES†

FOSTER KENNEDY, M.D.*

and

PETER G. DENKER, M.D.**

INTRODUCTION

Properly to appraise the relationship of a spinal cord ailment to an associated injury, one needs knowledge of the anatomy and physiology of the spinal cord, as well as of the changes, both gross and microscopic, that can be produced in the spinal cord by various injuries. Experience with the normal clinical course of spinal cord diseases, their cause, when known, as well as their incidence after injury is also essential. In certain types of injury, it is evident that the spinal cord lesion was the direct result of the trauma sustained, as in fractures of the spine with compression and laceration of the spinal cord. In other cases, such an association may not be so evident. The trauma injury sustained and the spinal cord disease present may be coincidental; as has occasionally been noted, the under-

†The Journal, in this issue, is printing two articles from the national Symposium series dealing with "Scientific Proof and Relations of Law and Medicine" (2nd Series). The Symposium contains fifty or more studies prepared by legal and medical scholars on problems of joint interest to the two professions. The papers will be published in the pages of participating legal and medical journals during the Spring and Summer of 1946. The intent of the effort is to muster up legal and scientific learning relevant to various type problems which need illumination from both sources for their proper solution. The scientific writers have undertaken, under editorial direction, to prepare their studies in a basic style comprehensible to lawyers, without, however, any sacrifice of scientific authority.

The new Symposium is a continuation of the first series, published by leading law reviews and medical journals in the Spring of 1943. As before, the general Editor of the Symposium is Hubert Winston Smith, who holds an appointment under the Distinguished Professorship Fund, as Professor of Legal Medicine in the University of Illinois affiliated with the College of Law and with the College of Medicine. Readers interested in procuring a master index containing citations to the studies published in both first and second series of "Scientific Proof and Relations of Law and Medicine," may do so by sending 20c in currency or stamps to Professor Smith, College of Law, University of Illinois, Urbana, Illinois. Copies so reserved will be mailed between May 15th and June 1st.

*Professor, Clinical Neurology, Cornell University Medical College; Director, Neurological Service, Bellevue Hospital, New York City.

**Assistant Professor, Clinical Neurology, Cornell University Medical College; Associate Attending Neurologist, Bellevue Hospital, New York City. Annotations and footnotes for the present paper were prepared by Hubert Winston Smith, A.B., LL.B., M.D., Professor of Legal Medicine at the University of Illinois and General Editor of the Symposium Series.

lying disease of the spinal cord may have given rise to the injury. Such an association, for example, has been observed in early cases of multiple sclerosis, where, because of a slight weakness in the muscles of a foot, a fall results, with a blow to the spine, and the then noticed foot weakness is attributed to the fall. One should be aware of the kind of spinal cord disease which can be produced by injury, and the degree of severity necessary to produce such change. And one must know the spinal abnormalities for which injury is *not* responsible.

The last war in which occurred vast numbers of spinal injuries, of which more later, greatly helped the assessment of the part played by trauma in the production of cord disease. Too often, in our medical books, an author reports a *single* case where a back injury was followed, sooner or later, by some spinal cord disease. He yields readily to the temptation to make one cause the other, ignoring the hundreds of cases where trauma did *not* precede the development of this particular disease.¹ The publication of unscientific, undocumented trivia of this sort is at best mere advertisement, at worst, malfeasance for the production of specious arguments in the law courts.

We propose here to review briefly the structure and functions of the spinal cord, and then, having critically examined the evidence, to give an opinion on the relationship between injury and various spinal cord diseases.

These opinions are based on a 30-year experience with these cases, as seen in the Neurological Wards of Bellevue Hospital, New York City, a municipal hospital where spinal injuries and diseases are constantly encountered.

ANATOMY AND PHYSIOLOGY

The spinal cord is a solid cable of nervous tissue, about eighteen inches in length, enclosed within a bony vertebral canal, and extending down from the brain, and continuous with it. It is encased in a series of membranes, called the *pia mater*, the *arachnoid* and the *dura mater*. The transparent pia mater lies closest to the spinal cord. The dura mater lies outermost and is the toughest of the membranes. Between the two, is the delicate, weblike arachnoid. Between the pia mater and arachnoid, and surrounding

1. This involves the fallacy of so-called "post hoc ergo propter hoc" reasoning, *i.e.*: after this, therefore on account of it—the fallacy of arguing from mere temporal sequence to cause and effect relationship.

the cord substance like a water-cushion, is a normally crystal clear liquid: the *cerebrospinal fluid*. All these structures lie within the vertebral canal, a sort of hollow tube within the solid bony encasement of the vertebrae, or spinal column. These vertebrae in turn are supported by a thick band of muscle tissue and strong fibrous ligaments, giving added protection to the sensitive and important underlying nervous structure.

The main function of the spinal cord is one of transmission: to carry sensory impulses inwards to the brain, or to relay outwards motor commands from the brain to the body. It also serves as a reflex station for such functions as urination, defecation and the sexual act. It plays an important role in the nutrition of the skin and muscles. In the cervical (neck) and lumbar (lowback) regions,² the spinal cord is enlarged, since in these areas the main motor cells for the arms and legs are placed. The cord ends at the level of the first or second lumbar vertebra, where it tapers off into the nerve roots of the cauda equina. There are eight cervical, twelve thoracic, five lumbar, five sacral and one coccygeal segment in the cord, and from each of these segments, spinal nerve roots extend, on either side of the cord. These nerve roots are paired. The anterior (or ventral) roots are motor in function, and the posterior (or dorsal) roots carry various sensations to the cord upwards. Shortly after leaving the cord, the nerve roots, one motor and one sensory, join, forming the spinal nerve of that segment. In these spinal nerves therefore both motor and sensory impulses are carried. They extend outward to the various muscle or skin areas served by them.

Sensation is transmitted by the nerves from the highly specialized nerve endings found in the skin and mucous membranes of the body into the spinal cord, by the posterior (or dorsal) nerve roots, and from here upward, in the spinal cord in certain nerve *tracts*; these finally reach the brain, where sensation is received, criticized, and occasionally ignored. These tracts are arranged according to function, some conveying stimuli of touch, others pain, heat, cold, position sense, et cetera. Motor impulses come from the motor area of the brain, along the large nerve fibres comprising the *pyramidal tract* to the spinal cord. These tract fibres end around the anterior ventral horn cells in the spinal cord. From this point, a second relay of fibres, the anterior (or ventral) nerve roots carry stimuli,

2. The vertebrae are the thirty-three bones which make up the spinal column. They consist of seven *cervical*, twelve *thoracic* or *dorsal*, five *lumbar*, five *sacral*, and four *coccygeal* vertebrae.

or impulses, to the particular muscle, or group of muscles in which action is desired. In the substance of these muscles, the nerve ends in the form of a motor nerve-plate.

In this manner, the main forms of sensation *from* the body, and motor stimuli *to* the muscles, are transmitted by and through the spinal cord. There are, therefore, both sensory and motor tracts in the cord, each affecting their respective functions, and on cross-section, these various tracts can be observed. It will be noted that most of these tracts which ascend or descend in a vertical line within the spinal cord, lie in the *white matter* of the cord. The central butterfly-shaped portion is called the *grey matter*. It consists of many cells serving generally the purpose of relaying stimuli from one cell to another. The posterior wings of the grey matter are called the *posterior horns* and these contain many of the "synapses" or liaisons of the sensory nerve cells. The anterior or ventral parts of the grey matter are known as the *anterior horns*, and contain the anterior horn cells, the lower, or spinal motor units from which the anterior nerve roots arise. In the lateral part of the grey matter are additional cells whose function is to convey special impulses for nourishing the skin and mucous membranes through the sympathetic nervous system.

The principal sensory, or ascending tracts are:

(1) The *tracts of Goll and Burdach*, in the dorsal columns of the white matter, conveying the sensations of pressure, tactile discrimination, vibration and position sense of muscle and joint to the brain.

(2) The *spinothalamic tract* lies laterally in the white matter, between the anterior and posterior horns of the grey matter. This tract transmits sensations of pain and temperature to the brain.

(3) The *spinocerebellar tracts* also lie in the lateral portions of the white matter, more peripherally than the spinothalamic tracts. They convey impulses to the cerebellum³ from the muscles for the purpose of maintaining coordination.

The main *motor* or descending tracts in the spinal cord are:

(1) The *pyramidal*, or cortico-spinal tract. The fibres of this tract arise from the large pyramid-shaped cells in the motor area of the brain, passing down through the brain and into the cord. It lies in the lateral

3. *Cerebellum*: That division of the brain behind the cerebrum and above the pons and fourth ventricle. The cerebellum is concerned in the coordination of movement.

part of the white matter adjacent to the spinathalamic tract. The fibres in this tract finally end around the cells of the anterior horn. From these anterior horn cells, fibres run directly to the muscles by the anterior nerve roots. This tract conveys voluntary motor impulses from the brain to the muscles, and is the principal motor pathway for the transmission of such impulses.

(2) The *rubro-spinal tract* originates in the mid-brain and is close to the pyramidal tracts in the white matter. This tract conveys impulses from the cerebellum and other brain areas needed for proper coordinated muscle function and automatic associated control of muscles.

(3) The *vestibulo-spinal* and *tecto-spinal tracts* lie in the ventral columns of the white matter, and convey from the brain those impulses needed to maintain equilibrium and the visual and auditory reflexes.

There are very many other tracts, of course, not mentioned here. They have little bearing on the problems we discuss.

THE NEUROLOGICAL EXAMINATION AND THE SIGNIFICANCE OF VARIOUS FINDINGS

It may be helpful, at this point, to outline the steps of a neurological examination to determine the presence or absence of spinal cord disease. Lawyers frequently find it difficult properly to interpret medical descriptions. Essential in any such examination is a careful *history* of the case; often, such a history by itself can make certain the presence of severe spinal cord injury. Not only the symptoms complained of at the time of examination, but also the chronological timing and mode of onset are important. How severe was the injury? Was the patient in good health and working regularly and without complaint previous to this time? If he fell, what was the distance of the fall? Did he land on his feet or his back? What part of his body was the direct point of contact? Was the blow of sufficient force to lacerate the skin or underlying tissues? Was he unconscious? If so, how long? Was he immediately paralyzed, and if so, in which limbs? and did he temporarily recover from such symptoms? Was he able to void his urine or was paralysis of the bladder present? Exact answers to these questions may give important clues to the nature and severity of the injury. They are of the very greatest value in estimating the relationship between the injury and the disease present, as well as the future course of the illness. In our experience, practically every important spinal cord injury is accompanied with immediate evidence of injury of a motor, sensory, or reflex character.

This leads to the second part of the neurological examination: the testing of muscle power and sensation in the arms, legs and body, and of the deep and superficial reflexes.⁴ The examination of the cranial nerves⁵ will not be described here, since in spinal cord injuries, we are usually concerned with the area below the head. Suffice it to say, however, that the pupillary reflexes of the eyes⁶ may be of significance in this connection because of either of two conditions which may be present: (1) *syphilis of the central nervous system*, which may be suspected by reason of certain pupillary reflex abnormalities, or (2) the *Horner Syndrome* produced by lesions (organic damage) in the low cervical and upper thoracic spinal cord, or root, segments. Here one observes a small pupil, a narrow palpebral aperture,⁷ and a somewhat sunken appearance in the eye on the affected side. If the patient be able to walk, his gait should be observed. Spinal cord injuries, if severe, usually give rise to a spastic,⁸ stiff, gait with or without unsteadiness, depending on the nerve tracts affected. If sensation be impaired, it is important to determine the upper level on the body of such impairment, and whether the sensory loss below this level is complete for all forms of sensations, or only for certain types.⁹ Thus, in a complete transection (severance) of the spinal cord, all forms of sensation below the level of injury will be lost; whereas hemorrhage into the substance of the cord may produce a disassociated loss of sensation; that is, pain and

4. *Deep and superficial reflexes*: Any disease or injury which breaks any part of the nervous pathways involved in the reflex arc will abolish or diminish the reflex. Superficial reflexes are obtained by gently stroking or scratching the skin with a blunt object. The deep reflexes are obtained by striking the tendon of a muscle briskly with a soft rounded object, preferably a rubber reflex mallet.

5. *Cranial nerves*: The cranial nerves are arranged in twelve pairs. Should the reader have need to consult a clearly written but authoritative book concerning the requisites of a sound examination of the nervous system, he would do well to examine SPURLING, R. GLEN: PRACTICAL NEUROLOGICAL DIAGNOSIS (3d ed., 1944), Springfield, Ill., C. C. Thomas.

6. *Pupillary light reflex or retraction*: Each pupil, the other eye being covered, dilates and contracts as the eye is alternately shaded by the hand and exposed to light, and the vision is constantly fixed upon some distant object. Normally when a pupil contracts to light (direct reflex), the pupil of the other eye also contracts (consensual reflex).

7. *Palpebral Aperture*: The fissure between the eyelids.

8. *Spastic gait*: A walk in which the legs are held together and move in a stiff manner, the toes seeming to drag and catch.

9. As there is a segmental distribution of nerve supply corresponding with particular levels or segments in the spinal cord, the determination of the upper level on the body of the sensory impairment enables one to infer the level of the injury to the spinal cord.

temperature sense may be lost, and tactile and vibration sense may be retained. The deep reflexes of the arms and legs, or the tendon reflexes as they are often called, give additional information. They are usually known as the biceps, triceps and radial jerks in the arms, and the knee and ankle jerks in the legs. They may be normally active, absent, or overactive, all of which is significant as regards the total picture. They may be equal in both limbs or exaggerated on one side as compared to the other. A markedly overactive ankle-jerk is frequently known as *ankle clonus*, and its presence may be important. Lastly, the abdominal reflexes,¹⁰ cremasteric reflexes¹¹ and the *Babinski sign* are sought for. All are of importance, especially the latter. This sign of Babinski is elicited by stroking the outer border of the sole of the foot with some blunt object, such as a key, or a pencil, and the movement of the large toe observed. Normally the big toe flexes, moves downward, as a result of this stimulus. When the big toe, however, moves upward, the response is known as a "positive Babinski sign," and indicates damage to the pyramidal tract, the main motor pathway from the brain to the spinal cord.

Note should always be made on bladder and rectal function. Were these normal? Or was there retention or incontinence of urine or feces? Or difficulty in starting the flow? Likewise, ability to perform the sex

10. *Abdominal reflexes*:—*Editor*: As Spurling says: "These reflexes can best be elicited with the patient recumbent. The abdominal walls must be relaxed. The skin of the abdomen is stroked with a blunt point, preferably a match or a wooden applicator. In testing for the upper abdominal reflex, the skin of the right and left upper abdominal quadrant is stroked. This causes a contraction of the muscles of the upper abdominal wall, thus producing a deviation of the umbilicus to the side of the stimulus. The lower abdominal reflex is obtained by stroking the skin of the lower abdominal quadrants. The muscles of the lower abdominal walls contract and thus pull the umbilicus outward and downward."

"The pathway for the upper abdominal reflex is through the ninth and tenth thoracic segments of the spinal cord. For the lower abdominal reflex, the pathway is through the eleventh and twelfth thoracic segments."

"These reflexes are frequently absent in elderly people, in multiparae (women who have previously given birth to a child) and the very obese patients. Any healed lateral abdominal incision may cause them to disappear on the side of the incision, due to the disturbance by the scar of the neural (nervous) pathways. In acute peritoneal inflammation, they may disappear completely." SPURLING, *op. cit. supra* n. 5, p 137.

11. *Cremasteric reflexes*: *Editor*: As Spurling says: "Pricking or stroking the skin of the inner side of the thigh causes a contraction of the cremasteric muscle, thus lifting the testicle on that side. This reflex varies considerably in normal individuals; hence, it may be an unreliable sign unless it coincides with the other clinical findings. The arc concerned in this reflex is through the first lumbar segment." *Id.*

act may become completely lost by spinal injury or disease in the male, because of inability to obtain erection or orgasm.¹²

TYPES OF SPINAL CORD INJURY

Though the spinal cord is well protected from injury by its water-cushion or cerebrospinal fluid and its surrounding bony vertebral encasement, as well as by thick, strong muscle layers between the vertebrae and the skin, it may be damaged, directly or indirectly, in the course of the following conditions:

1. Fractures or dislocations of the vertebrae.
2. Laceration or contusion of the spinal cord, without vertebral fracture or dislocation. This occurs, for instance, in the case of penetrating wounds of the spinal cord, such as stab or gunshot wounds.
3. Hermatomyelia, or hemorrhage into the spinal cord substance.
4. Extra- or Intradural hemorrhages, with cord compression. These are rare.
5. Concussion of the Spinal cord: a concept to be discussed in fuller detail below.
6. Prolapse of the nucleus pulposus, from injury to the intervertebral disc.¹³

12. Injuries of this sort command very large jury verdicts, and rightly so. Oftentimes the injured individual will require a special attendant for the remainder of his life. *Ward v. Iroquois Gas Corp.*, 233 App. Div. 127, 251 N.Y.S. 300 (1931) (modified in 258 N. Y. 573, 180 N. E. 338 (1932) (40-year old steam fitter sustained a fracture of the tenth dorsal vertebra with severe injury to the spinal cord rendering him unable to walk without crutches and depriving him of control of bowel and bladder functions; he had been rendered impotent; medical and hospital expenses to the time of trial amounted to \$3500; held: a verdict of \$60,000 was not excessive.) In *Span v. Jackson, Walker Coal and Mining Co.*, 322 Mo. 158, 16 S. W. (2d) 190 (1929) a 36 year-old coal miner sustained a fracture of two lumbar vertebrae with coincidental injury to the spinal cord which caused him to lose control of his bowels and bladder and to become sexually impotent; a verdict of \$50,000 was held to be not excessive. Large verdicts were similarly upheld for such injuries in the following cases: *Ramey v. Missouri P. R.*, 323 Mo. 662, 21 S. W. (2d) 873 (1929); *Potter v. Shute*, 7 Tenn. App. 222 (1928); *Sterns v. Hellerich*, 130 Neb. 251, 264 N. W. 677 (1936); *Zamecnik v. Royal Transit, Inc.*, 239 Wis. 175, 300 N. W. 227 (1941) (complete severance of spinal cord among other injuries); *Duren v. City of Binghamton*, 172 Misc. 580, 15 N. Y. S. (2d) 518 (1940); *Lang v. Ingham County*, 277 Mich. 345, 269 N. W. 197 (1936) (First thoracic vertebra crushed allegedly resulting in paralysis and loss of control of bowels and urinary system); *Goldberg v. Capitol Freight Lines*, 314 Ill. App. 347, 41 N. E. (2d) 302 (1942) (Loss of control of bladder and bowels.)

13. *Nucleus pulposus*: A pulpy body in the center of the intervertebral discs which separate the bodies of the vertebrae and provide a resilient cushion between them to absorb sudden shocks or stresses. For a discussion of ruptured intervertebral disc, see *infra*.

7. As a late result of a process, primary or secondary, initiated by old injury. Here there may occur progressive changes stemming from the original injury and affecting cord function at some later date; for example, *meningitis circumscripta serosa* and *Kummel's disease*.

In the majority of cases, injuries to the spinal cord result from severe injury to the vertebral column, usually *fracture* or *dislocation* of the vertebrae. Yet, in some, no vertebral damage can be proven. In such cases, the spinal cord lesion is probably secondary to a disturbance of the spinal circulation produced by the injury, and not directly due to a crushing of the cord, as occurs when the vertebra is fractured or dislocated. Fractures of the spine on the whole are more common in the highly movable cervical (neck) and in the upper lumbar (mid-back) regions than in the more rigid thoracic (upper back) portion. In 100 cases of injury to the spinal column, Frazier found approximately 60 per cent fracture dislocations, 20 per cent fractures without dislocation, and 20 per cent dislocations alone. In the servical region, about 75 per cent of fracture-dislocations or dislocations are associated with injury to the spinal cord; in the lumbar region, the figure is approximately 25 per cent (Wortis and Sharp). When the spinal cord has been so injured, mortality is naturally greatly increased. The higher the site of the fracture, the greater the mortality. Thus, in the cervical region, where signs of cord injury are present, 75 per cent of patients die; in the thoracic region, about 50 per cent are fatal; in the lumbar region, this figure is reduced to about 15 per cent. In those that survive, most will remain totally or partially paralyzed, the maximum improvement usually being reached in 6 months to 1 year, indicating the general seriousness with which these cases must be regarded. This applies especially to those in the cervical region.

In civil life, *fracture dislocations* of the cervical spine are almost always produced by indirect violence, such as falls from a height, diving and driving accidents, a crushing blow from above, or a vertical fall in which the patient has landed jarringly on the heels. A violent flexion of the spine results from these various causes, with a sharp jack knifing of the vertebral column, producing fracture or dislocation of the vertebra. With the newer insulin or electrical methods used in the treatment of certain mental disorders, compression fracture of a vertebral body, usually in the thoracic region, has been reported, occurring particularly when technique has been faulty; it is caused by the sharp flexion spasm of the body when the con-

vulsion is produced. Fortunately, in these cases, the spinal cord itself is usually not damaged; except for local back pain for one or two weeks, there are no symptoms and an excellent recovery occurs.

It would be well to stress again at this point that the injury to a vertebra is of far less importance and seriousness than is an associated injury to the spinal cord. In frequent cases, there is no demonstrable bony injury, yet the cord certainly has been damaged. Here it is thought there may have been a sudden dislocation of a vertebra followed by an immediate return to normal alignment, producing some interference with the cord's blood supply, or even hemorrhage within the cord substance proper: *hematomyelia*. In many cord injuries, small, punctate hemorrhages¹⁴ are disseminated throughout the white and grey matter in a manner in no way as massive as in a true hematomyelia. There may be also focal areas of necrosis, *i.e.* death or destruction of nerve cells and fibres, with edema (swelling) of the cord, and in more severe cases, laceration (cutting) of the cord itself. When the spinal cord has been damaged, however, whether or not there be an associated vertebral injury, such damage is almost instantaneously obvious, and this is of great medicolegal importance. It is usually manifest at once, immediately after the infliction of injury, and most frequently is recognized by retention of urine for one or two days, by a partial or complete paralysis of the legs, or by a definite sensory impairment or reflex changes. Depending on the level of the injury, the legs, or the legs and arms, are weak or paralyzed, sensation is reduced up to the injured cord segment,¹⁵ and reflexes below this level may be absent at first. There is likely to be complete temporary paralysis of the bladder, and maybe of the rectum also. All cases therefore should be suspect, in which an injury is claimed to be the cause of a subsequent cord disease, and in which some of these symptoms did not follow the trauma immediately.

We agree with Kinnier Wilson who wrote, "In genuine trauma of the central nervous system (and this applies especially to the spinal cord) symptoms arise practically at once."

14. *Punctate hemorrhages*: Spots of blood effused into the tissues from capillary hemorrhage.

15. It has been pointed out previously that the body may be divided into belts or segments which derive their nerve supply from corresponding segments in the spinal cord so that the level of impaired sensation on the surface of the body can be correlated with the level of injury to the spinal cord.

Penetrating wounds of the spinal cord are infrequent in civil life, frequent in war. These are produced by bullet or from wounds by metal fragments, or by a knife blade passing between vertebrae into the spinal cord. In one of our cases the dagger tip remained broken off between the vertebrae for 15 years before symptoms of spinal cord pressure manifested themselves. Here the point of the dagger, having passed between vertebrae, had not pierced the cord but had reached its outer margin. The gradual accumulation of rust on the steel and the inflammatory adhesions about it, gradually caused a cord compression and paralysis of the legs. Similar instances of stab wounds of the spinal cord have been reported by Jones (with symptoms occurring 18 years after the stabbing), and by Antonelli (36 years after the original injury). The development of symptoms at a later date in these cases, however, should not be confused with the immediate onset of symptoms in cord injuries due to fracture or dislocation of the spine and, of course, the vast majority of spinal cord knife wounds produce immediate symptoms.

Gun shot wounds may injure the spinal cord by direct laceration or contusion of the cord as the bullet penetrates the substance, or indirectly, by splintering a vertebra and secondary penetration or compression of the cord by a chipped-off piece of bone. Rare cases of spinal cord injury have been reported following the bursting of large shells near a patient, though no external wound of the spine had occurred. Sudden changes in atmospheric pressure caused by explosion have produced these injuries usually by hemorrhage into cord substance. The lesions produced, however, have been confirmed by histological observation,¹⁶ as by Guillain and Barré, who found morbid changes in the brain and spinal cord, together with hemorrhagic spinal fluid; Marie and Banistry also described lesions varying from a few, often microscopic, punctate hemorrhages or patches of necrosis (death of tissue) to complete pulpification of the spinal cord. Some spinal cases have often been called "*concussion of the spinal cord*," a bad term because of its ambiguity. These may have definite spinal cord lesions demonstrable microscopic examination, and also have indubitable clinical evidence of injury to the cord. They truly in no way differ from contusions (bruises) of the spinal cord; the whole matter has always been a mere terminological quibble. The difficulty arose in 1875, when little was known of the microscopic appearance of spinal cord injuries. At that time, Ericksen speculated on

16. *Histological observation*: Study of tissue anatomy under the microscope.

a "molecular" disturbance of the spinal cord in cases lacking obvious spine fracture or dislocation. Lacking a better term, he called them "concussion," but as Frazier has pointed out, "His clinical observations were so faulty, and his neurological knowledge so inadequate, that time is poorly spent in studying his cases, or giving serious consideration to his conclusions." The crux of the matter is that as a result of the injury a lesion of the spinal cord exists and whether one calls it contusion, or concussion is academic. From a practical point of view we would like to see the term "concussion" abolished, as it is a theoretical distinction productive of endless semi-learned speculation. Were all these cases called "spinal cord injuries,"—"reversible" and "ir-reversible"—and no attempt made to resolve them primarily into further pathological subdivisions,¹⁷ it would be easier to distinguish cases due to "injury" from those due to "pre-existing disease," and injured cases of small moment from injured cases that are momentous. The purposes of justice would be more faithfully served.

Though *injuries to the intervertebral discs* with protrusion¹⁸ of the nucleus pulposus are dealt with in a separate study in this Symposium, it might be well for purposes of completeness, to speak of them here, also. Such injuries may occur at any level but are usually in the lower lumbar region of the spine; they cause there compression and irritation of the lower lumbar and upper sacral nerve roots, with, as the most prominent symptom, pain radiating down the back of the leg. In less common numbers, however, such disc injuries may occur in the cervical or thoracic spine, and we have seen a picture in some such cases of complete spinal cord "block." They *appear* if pressure be quick or slow, to be either transverse myelitis¹⁹ or spinal cord tumor. Fortunately, surgical removal of these protuberant bodies is attended with excellent results; in most cases, complete relief of symptoms occurs. For a more detailed discussion of this important subject, the reader is referred to the Symposium study by Barr and Craig entitled "Ruptured Intervertebral Disc."²⁰

Electrical injuries may produce physiological or pathological changes

17. *Pathological subdivision: i.e., finer subdivisions or analyses based upon structural and functional changes caused by injury or disease.*

18. Barr, Joseph S. and Craig, Winchell M.: *Ruptured Intervertebral Disc*, to be published in one of the Spring issues (1946) of the JOURNAL OF NERVOUS AND MENTAL DISEASE and to be submitted for legal publication.

19. *Transverse Myelitis*: Myelitis is inflammation of the spinal cord; transverse myelitis is a form which extends across the cord.

20. *Id.*, n. 18, *supra*.

in the spinal cord. This accident can occur when an electrical household appliance is improperly insulated or improperly used, or by contact with a high tension wire in industry or as the result of some disaster or accident. If strong enough, such a current through the body may kill instantly; otherwise spinal cord lesions may occur, of lesser or of severe degree. Pain and weakness in the affected limbs, if organic neural changes have occurred, occur almost immediately after the injury, and may be permanent or passing. Neurological examination in these cases usually produces the signs of a spinal cord lesion; if the man has been in good health previously and the symptoms appeared immediately, or within a few days after the electrocution, it is but fair to blame the passage of the electric current for his spinal cord ailment. In some cases signs produced immediately may be thereafter progressive.²¹

Compressed air illness (Caisson Disease) occurs in persons working in high-compression chambers, in tunnels, under rivers, et cetera. It is often called "bends" or "diver's paralysis," and comes from the setting free of nitrogen as bubbles in the body-tissues.²² In about 10 per cent of these cases, the central nervous system is involved; the spinal cord is the area most often affected, giving rise to weakness of the legs and a sensory loss below the level of the lesion. It is believed that the cause of the frequent spinal cord localization is its high fat and myelin²³ content: such tissue has a capacity for nitrogen absorption about five times as great as blood plasma. When atmospheric pressure is increased, large amounts of nitrogen are absorbed by the fat and myelin of the cord; during decompression this is released leaving free bubbles of the gas in the cord substance or occluding (stepping up) some of the smaller vessels with air blocks. The paralysis may be temporary, if treatment be instituted rapidly: in many cases it has

21. See, in this Symposium series, the following paper: Hyslop, G. H.: *The Effects of Electrical Injuries with Particular Reference to the Nervous System*, to be published in a Spring number (1946) of OCCUPATIONAL MEDICINE and to be submitted for legal publication.

22. For cases involving Caisson disease, see: *Williams v. Missouri Bridge and Iron Co.*, 212 Mich. 150, 180 N. W. 357 (1920); *Beaty v. Foundation Co.*, 245 Mich. 256, 222 N. W. 77 (1928); *Taylor v. List and Weatherby Const. Co.* 146 So. 353 (La. App. 1933); *Maryland Casualty Co. v. Gerlaske*, 68 F. (2d) 497 (C.C.A. 5, 1934); *Mo. Valley Bridge & Iron Co. v. Ballard*, 53 Tex. Civ. App. 110, 116 S. W. 93 (1909).

23. *Myelin*: Any one of a certain group of lipid (fat-like) substances found in various normal and abnormal tissues and differing from fats in being doubly refractive. It is the substance which forms a sheath around the medullated (myelinated) nerve fibers.

been permanent. As in spinal cord lesions from other types of injury, maximum recovery occurs within six months; after a year, no further improvement can reasonably be expected.

Sometimes injury to the spinal cord, or its nerve roots, occurs from therapeutic procedures, such as *lumbar puncture*, *instillation of iodized oil* for diagnostic purposes, *spinal anesthesia*, and, more recently, from *caudal anesthesia* in obstetrics.²⁴ Though, in the vast majority of cases, lumbar punctures are performed as a routine procedure in the diagnosis of nervous disorders with no complications, rarely nerve root injuries or sterile meningitis²⁵ have occurred (neither writer has ever seen this.) These are said to be transient; they lead to no important or permanent impairments. In some few cases, the intervertebral discs have been injured; the spinal needle here has penetrated too deeply, and has damaged the disc; thereafter possible collapse of the disc may occur and a resultant narrowing of the intervertebral space as seen by X-rays. Pease, Gallman, and more recently, Everett, have reported such cases. Rarely, the lumbar puncture needle has been broken in this wise, requiring surgical removal.

Of greater frequency, and of more serious prognosis, are those cases of cord or nerve root injury following spinal anesthesia with cocaine or its derivatives. Though surgeons consider this procedure to be comparatively safe, *cauda equina*, as well as more distant spinal cord, and even cranial nerve injuries occasionally have resulted from it. They have been reported by Hyslop, Brock, Bell and Davison, Kellman and Abbot and others. Ferguson and Watkins have recently described 14 such sequelae. In some, the nervous lesions are transient; in many they are permanent: in a few cases, death has resulted from transverse myelitis²⁶ produced by the spinal anesthetic. It must be borne in mind, however, that in the many thousands of cases in which this procedure has been used, such cord or nerve root complications have occurred relatively seldom; the highest figures given

24. Though such injuries have been reported in the medical literature, there seems to have been little litigation, as yet, arising from the procedures mentioned. In *Loudon v. Scott*, 58 Mont. 645, 194 Pac. 488 (1920), 12 A. L. R. 1487, the question was presented as to the physician's liability for administering anesthesia to a patient while he was intoxicated, thereby allegedly causing his death; the court held, in affirming a judgment of non-suit, that the causal connection in such a case could not be established without expert testimony.

25. *Sterile meningitis*: Meningitis is inflammation of the meninges which invest the brain and spinal cord. Sterile meningitis is meningitis in which there is no infection, *i.e.*, meningitis caused by injection of air or of serum.

26. *Transverse myelitis*: See n. 19, *supra*.

being one-half per cent or one in two hundred cases (a figure high enough to make the writers determined to refuse spinal anesthesia for themselves or families!) Gready has recently quoted Hawkin's experience of similar nerve root injury in the course of *caudal anesthesia* to relieve the pains of childbirth. This is a rare complication, since Hingson and Edwards have recently reported 1,000 successive caudal anesthetics without a case of spinal cord or nerve root injury. However, we would observe that the mortality or permanent disability of women by the pain of childbirth is nil,—this method seems only justifiable if it proves to increase the fecundity of American women. Also, rare serious spinal cord injuries have occurred following injection of alcohol in paravertebral spaces²⁷ to block the sensory nerve roots for the relief of cardiac pain (Winterstein, Molitch and Wilson, Groff and Lewy, Herschboeck and Gillespie.) The injection of 80 per cent alcohol anywhere is hazardous; blind surgery needs at least as much skill and care as does the surgery of vision.

Iodized poppyseed oil (lipiodol), used for the visualization by X-ray of compressing lesions of the spinal cord or nerve roots, has produced injuries to both cord and roots, usually as an inflammatory reaction.²⁸ Marcovich, Walker and Jessico have reported recent experiences, as have many others; they feel that short transient reactions with increase of cells and protein in the spinal fluid occur in about half the cases, but that permanent ill effects are rare. Others have not been so fortunate; recently Bucy has described a most unfortunate situation; many others have similarly reported permanent arachnoiditis²⁹ and nerve root irritation, together with persistence indefinitely of the lipiodol in the spinal canal. We, also, have seen such things. We are reluctant to advise the diagnostic use of lipiodol, unless all other means of neurologic examination have failed to produce the accurate knowledge needed for operation. The entire subject of the use of lipiodol and its dangers has been excellently reviewed in the recent articles by Garland, and Walsh and Love. We have recently seen a case with all the signs of acute encephalitis,³⁰ following injection of lipiodol for spinal

27. *Paravertebral spaces*: The spaces beside the vertebral column.

28. *Inflammatory reaction*: Inflammation is the condition into which tissues enter as a reaction to injury. Inflammation is characterized by pain, heat, redness, and swelling, and histologically by such microscopic findings as hyperemia (increased blood supply to a part), stasis, changes in the blood and walls of the small vessels, and by various exudations.

29. *Arachnoiditis*: Inflammation of the arachnoid membrane, the middle of the three membranes which invest the brain and spinal cord.

30. *Encephalitis*: Inflammation of the brain.

diagnosis. X-ray showed dense oil shadows in shadows in every cerebral ventricle:³¹ an event difficult to explain easily to the owner of the shadows.

Two clinical entities distantly related to spinal cord injuries need mention. The first is *Kummel's Disease*,³² a traumatic disease of the spinal vertebrae, which may, at some date after injury, cause spinal cord compression. The usual history is one of a blow to the back, which may have been mild or severe, followed by, for a few days, some incapacity. There is some pain in the injured area for a few months followed by increase of pain and then the gradual signs and symptoms of spinal cord involvement: leg weakness, radiation of pain along affected nerve root distribution and perhaps, kyphosis³³ of the spine. X-rays of the spine—the diagnostic test—reveal a typically affected vertebra which was probably fractured at the time of the original injury. Subsequently, rarefaction and inflammation of bone occurs; ultimately the affected vertebra collapses and compresses the cord. This condition is a rarefying osteitis;³⁴ it is generally believed to be produced by injury. The other late sequel to spinal injury is *meningitis circumscripta serosa*. Here, severe nerve root pains may occur at a late date after injury from meningeal and thickening adhesions. These come from trauma to the membranes at the time of injury. We feel that, here, proof or reasonable evidence of the relationship between the original injury and the subsequent state, should be clearly given; that the injury should have been severe enough to have produced symptoms of spinal cord or root involvement at the time of its occurrence, and that the present level of spinal cord disease should coincide with the area affected by injury.

31. *Cerebral ventricles*: The cavities within the brain, including the two lateral, the third, the fourth, and the fifth ventricles.

32. *Kummel's disease*: Also referred to as traumatic or post-traumatic spondylitis. Spondylitis means inflammation of a vertebra. The disability is similar to that following ordinary compression fracture except that in absence of proper and adequate support and treatment, severe permanent disability usually results. For a case of alleged Kummel's disease attributed to wrenching of back when ice man lost control of 400 pound block of ice, see *Easthope v. Industrial Commission of Utah*, 80 Utah 312, 15 P. (2d) 301 (1932) (Some of the medical experts testified that X-ray pictures of plaintiff's vertebrae did not show evidence of any recent injury but rather a disease which had probably existed for ten or twelve years; compensation commission entered an award denying compensation and this was affirmed on appeal.)

33. *Kyphosis*: Humpback; abnormal curvature and dorsal prominence of the vertebral column.

34. *Rarefying osteitis*: Osteitis is inflammation of a bone. Rarefying osteitis is a bone disease in which the inorganic matter is lessened and the hard bone becomes cancellated—i.e., develops a lattice-like structure.

It is also reasonable to ask for evidence of some "bridging symptoms"³⁵ as in Kümmel's Disease from the time of the accident to the date the complete clinical picture of this condition has become manifest. By insistence on this, the risk of chicanery and innocent imposition will both be minimized. It is easy for any man with a spinal cord ailment to jog his memory to a remote back injury. Before poliomyelitis was proven to be an epidemic infection, every child brought to hospital with infantile paralysis had a history of his having been "dropped by a neighbor woman who'd been let tend him."

MULTIPLE SCLEROSIS

Multiple sclerosis is a chronic disease of the nervous system involving usually both brain and spinal cord: in early stages, the spinal cord alone may be affected. It is a fairly common ailment. Its cause is unknown; it has never been proven to be due to injury of the spinal cord. It occurs in early adult life, usually between the years of twenty and forty; it is progressive in its course; frequent remissions punctuate its progress. At long last, most patients become incapacitated and bedridden; this may not occur until ten or twenty years after onset.

Numerous patches of edema (swelling) and/or sclerosis (hardening due to overgrowth of connective tissue elements) are scattered throughout the nervous system. In these patches there is degeneration of the myelin sheath, the insulating layer which surrounds individual nerve fibres. The white matter of the spinal cord is frequently affected; the pyramidal tracts are involved early, giving rise to absent abdominal reflexes, overactive knee jerks and a positive Babinski sign. However, mostly, we find evidences of the presence of similar areas in the brain as shown by defects in the field of vision, called scotomata, ocular muscle palsies,³⁶ speech disturbances, or cerebellar involvement.³⁷ These are likely to be found if sought: A history

35. *Bridging symptoms*: Symptoms filling the time interval between receipt of the traumatic stimulus and the appearance of disability allegedly due to it. Such evidence goes to proof of causal connection and is extremely important in determining whether the disability was due to the injury, as alleged, or was due to some independent cause. For an excellent discussion of this problem, see in the first Symposium series, the following paper: Brahdly, Leopold and Samuel Kahn: *Clinical Approach to Alleged Traumatic Disease* (1943) 23 B.U.L. REV. 261; (1943) 18 ANN. INT. MED. 491.

36. *Ocular muscle palsies*: Paralysis of one or more of the ocular muscles which take part in producing movements of the eye.

37. *Cerebellar involvement*: Involvement of the cerebellum, that division of the brain behind the cerebral and above the pons and fourth ventricle which is concerned in the coordination of movement.

of some loss of bladder control, or of former or present numbness in the hands or feet. These symptoms disappear for awhile in characteristic remissions: sooner or later, usually sooner, the disease again flares up in a manner both episodic and progressive.

Though the cause of this illness is unknown, it may be certainly stated that it is *not* caused by injury. No one has ever been able to produce lesions of multiple sclerosis by experimental injuries in animals.³⁸ We believe it is either toxic or degenerative in origin though neither is proven. Infectious diseases, pregnancy, or severe emotional disturbances in persons already afflicted are known to precipitate attacks, or to aggravate the condition, but they certainly do not cause it. A study of this question, undertaken by the late Dr. Llewellys Barker, for the Association for Research in Nervous and Mental Diseases, concluded that injury is not the cause of multiple sclerosis. Injury may occur because this illness had already been present. A severe injury can aggravate the disease already existing.³⁹ Kinrier Wilson, neurological consultant to the British Army in the first World War, found that of 900 consecutive neurological cases seen from 1916 to 1918, "only two were multiple sclerosis, and neither of these two patients was wounded, gassed, blown up, or otherwise injured." He therefore quite rightly feels that the cause of multiple sclerosis "cannot possibly be assigned to trauma," and that injuries "can at most have done no more than possibly accelerate the development of the disease, though it might indeed be contended, with perfect justice, that they played no part at all in its evolution." Similar experiences with veterans of the first World War have been reported by Matz, who found only two cases of multiple sclerosis out of 2750 patients admitted to the neuropsychiatric service of the U. S. A. General Hospital No. 11 before 1920. Pollock, in over 1,200 cases of peripheral nerve injuries seen 18 months after the injury, did not find one case of multiple sclerosis, or, for that matter, any complicating mental disease. Pollock summarizes his experiences on this subject as follows: "With the exception of residuals of direct injury to the brain, spinal cord, or peri-

38. For cases of multiple sclerosis allegedly caused or aggravated by trauma (injury) see the following: *Garden Farm Dairy v. Dorchak*, 102 Colo. 36, 76 P. (2d) 743 (1938) (award of compensation, finding claimant to be 25 per cent disabled as a working unit by reason of his accident, was upheld on the theory that pre-existing multiple sclerosis was aggravated by an accidental fall); *Zanski v. Yellow Cab and Baggage Co.*, 143 Neb. 340, 9 N. W. (2d) 302 (1943); *Davis v. Lotz*, 126 N.J.L. 615, 20 Atl. (2d) 602 (1941).

39. See n. 38, *supra*.

pheral nerves, I do not recall one instance of disease of the nervous system that I could attribute solely to trauma." This corroboration, by an intelligent observer with a large experience, of the previous opinions expressed by Kinnier Wilson, and Matz, is of far greater significance than the scattered examples in the medical literature of single cases of multiple sclerosis which have appeared after injury. One of us writers saw two cases of early multiple sclerosis in four years' service in France in an army of the multiple sclerotic age of a nation in whom multiple sclerosis is rife. So isolated reports are interesting as coincidental experiences, but as nothing else. As pointed out in an article by Kennedy, the influence of injury as an etiologic factor in the "precipitation" or production of organic disease of the nervous system, is chiefly stressed with regard to those nervous ailments, the cause of which is entirely unknown. "No true scientist would ever think of reaching general conclusions from so paltry a number of special instances," and "almost no body of men so easily falls a victim to 'post hoc, propter hoc' reasoning⁴⁰ as do we physicians, especially in the matter of relationship of injury to disease." To overlook the tens of thousands of cases where multiple sclerosis has developed with no history of injury, and to stress the rare case where it has been said to have followed injury, is to ignore statistical controls, and to suspend critical judgment and rigid thinking. Were injury a cause of multiple sclerosis there surely should have been between 1914-1919 a small epidemic, at least, of that disease. A similar report by Verguth to the International Neurological Conferences at Berne in 1931, states "The common trauma-causing World War brought with it no increase in general paresis,⁴¹ multiple sclerosis, Parkinson's disease,⁴² nor the muscular atrophies⁴³ nor syringomyelia."⁴⁴

40. See n. 1, *supra*.

41. *General paresis*: Also known as dementia paralytica, a chronic disease of the brain of a degenerative sort marked by progressive loss of mental and physical power, due to syphilitic infection and involvement of the central nervous system. The disease lasts from several months to several years. For cases involving alleged activation of dormant syphilis or aggravation of existing disease, or some other medicolegal aspect of general paresis, see the following cases: *Finkelday v. Henry Heide, Inc.*, 193 App. Div. 338, 183 N. Y. Supp. 912 (1920), *aff'd* 230 N. Y. 598, 130 N. E. 909 (1921); *Atchison v. Colgate & Co.*, 3 N. J. Misc. 451, 128 Atl. 598 (1925), *aff'd* 102 N.J.L. 425, 131 Atl. 921 (1926); *Barry v. Miller*, 104 Conn. 362, 133 Atl. 37 (1926); *Great Northern Life Ins. Co. v. Cazner's Committee*, 282 Ky. 515, 139 S. W. (2d) 424 (1940).

42. *Parkinson's disease*: Shaking palsy (paralysis), or Parkinson's disease: a disease usually of late life, progressive in its course, and marked by a characteristic tremor of the muscles, weakness, delay of voluntary motion, a peculiar hurried gait, and a muscular contraction, causing peculiar and characteristic positions of the

It is important here to maintain one's common sense. The opinions of capable persons possessed of honesty, large experience, and a sense of perspective, are more important than the rare case reported by some,—usually obscure,—observer, clearly lacking critical judgment. It is likely that these

limbs and head. The disease is often attended with excessive sweating and feelings of heat and cold. For cases involving alleged causation or aggravation of Parkinson's disease (paralysis agitans) by injury (trauma), see: *Rothermel v. Sunbury Converting Works*, 5 Pa. W. C. Dec. 59 (1920); *Krauss v. Kaminski*, 6 N. J. Misc. 144, 140 Atl. 277 (1928); *Hartford Accident & Indem. Co. v. Industrial Commission*, 64 Utah 176, 228 Pac. 753 (1924); *Kentucky Traction & Terminal Co. v. Bain*, 174 Ky. 679, 192 S. W. 656 (1917), L.R.A. 1917D, 813. This case illustrates the type of imposition which the authors decry. Defendant's motor car left the track and ran into and demolished a part of plaintiff's home in which she was at the time, jarring, frustrating and exciting her. Plaintiff was a 51 year old woman. It was alleged that this episode caused in her the development of paralysis agitans and five physicians testified in her favor that "a severe jar or shock such as plaintiff sustained in this accident is calculated to and may produce paralysis agitans," and they asserted that in their judgment the jarring shock did in fact produce her trouble. Defendant called a number of witnesses to prove that prior to the accident Mrs. Bain had suffered from a nervous disorder of the nature of which she now complained, though it was then less pronounced, and that in truth she was not an able-bodied person before the happening of the accident, as she claimed to be. A verdict and judgment in plaintiff's favor for \$4,000 was affirmed on appeal. *Natalini v. Riefler & Sons, Inc.*, 286 Pa. 301, 133 Atl. 547 (1926) (pre-existing paralysis agitans allegedly aggravated by injury to back sustained in fall); *Larson v. Callahan Canning Co. of Coeur D'Alene*, 53 Idaho 746, 27 P. (2d) 967 (1933); *Ginsburg v. Byers*, 219 Minn. 230, 17 N. W. (2d) 354 (1945); *Miller v. Harris*, 344 Pa. 55, 36 Atl. (2d) 309 (1944); *Wood Preserving Corp. v. McManigal*, 39 F. Supp. 177 (W.D. Ky., 1941); *Shell Petroleum Corp. v. Ind. Comm.*, 366 Ill. 642, 10 N. E. (2d) 352 (1937). (Held: Evidence did not show that Parkinson's disease was aggravated by a blow on the head.)

43. *Muscular atrophies*: There are several rare degenerative diseases of muscle which are not caused by injury and have no medicolegal aspects. Among these are Myasthenia gravis, Family periodic paralysis, Muscular paralysis associated with renal failure, Muscular dystrophies, Thomsen's disease (Myotonia congenita) and Myotonia Atrophica. There is one condition, however, involving atrophy of muscles which may well have medicolegal connections, namely, the Generalized neuromuscular exhaustion syndrome, described by Dr. J. M. Nielsen of Los Angeles, in a paper so entitled, to be submitted for publication in this Symposium series.

44. *Syringomyelia*: The existence of abnormal cavities filled with liquid in the substance of the spinal cord. These cavities are surrounded by an abnormal tissue resembling that which is found normally surrounding the central canal. The disease is due to hemorrhage and consequent softening and necrosis (death of tissue), and is believed to be dependent on some defects of development. It occurs in adults between the ages of twenty and thirty years, and is marked by muscular atrophy, loss of the sense of temperature, and pressure, and by various vasomotor (circulatory) and trophic (nutritional) disturbances.

Kessler says: "In the German jurisprudence there have been reported cases of syringomyelia, in which persons suffering from this disease have injured their fingers, which were insensitive. Compensation has been allowed for the local changes, the result of a definitely proved accident, but not for the syringomyelia. (See the decision of R.B.A. Vol. V, p. 177, No. 72.) Finger deformities from syringomyelia are sometimes confused with "Dupuytren's contracture." KESSLER, HENRY H.: ACCIDENTAL INJURIES (2d ed.) Phila., Lea & Febiger, 1921, p. 543.

rare cases, if examined before injury, would have been found owning the disease. When Harris reports that in 7 per cent of the cases of multiple sclerosis he has seen over a number of years, "trauma played a role in its etiology," he ignores the injured in the general population who never developed multiple sclerosis, and pays no attention to the 93 per cent uninjured. Nor does he explain why multiple sclerosis is not more often observed in those having hazardous occupations, or in wartime. More germane are Patrick and Levy, who insist that "To prove that trauma is an etiologic factor in a disease it would be necessary to show that the chances of getting the disease are greater in the traumatized population." These figures do not exist.

We believe that injury plays no role in the production of multiple sclerosis; that no proof, clinical or experimental, has ever been submitted that such a relationship occurs, and that the rare cases in the medical literature of such sequential association of the two conditions are within the bounds of co-incidence. In fact, we do not believe that injury can precipitate, or aggravate, an incipient multiple sclerosis,⁴⁵ since no increased incidence of this disease has ever been shown either in the stresses and strains of warfare, nor in industries which are hazardous and involve high rates of injury. In our wards at Bellevue Hospital where many cases of severe spinal injuries are encountered, we have never observed a case of multiple sclerosis following such injury.

AMYOTROPHIC LATERAL SCLEROSIS

Amyotrophic lateral sclerosis and *progressive muscular atrophy* are diseases of the spinal cord which will be considered together since they are probably types of a similar disease process. In both conditions, the anterior horn cells of the spinal cord are affected; the difference between them is that in amyotrophic lateral sclerosis, the pyramidal tracts are clinically also involved. They are diseases, therefore, limited to the motor system, of a degenerative type, and progressive in their course.

Their cause is unknown. Rarely they appear after syphilis or encephalitis. We have never seen a case following injury to the spine, and do not feel that trauma plays any role in the production of this illness. All we have said in discussing the relationship of injury to multiple sclerosis, holds true

45. *Incipient multiple sclerosis*: Multiple sclerosis just beginning to affect the nervous system and therefore present at a subclinical level only, rather than as an established or disabling disease.

for amyotrophic lateral sclerosis. We are not impressed by Jelliffe, who, in 1935, published summaries of 109 cases culled from the literature of eighty years "where trauma seemed of possible etiological significance in the production of this disease."⁴⁶ The cases are cited in abstract. In many, the accuracy of diagnosis, from the date given, is open to grave question. As said by Davis, in only 49 of the cases is there any reasonable indication that the alleged disease was true amyotrophic lateral sclerosis, and that it clearly followed injury, yet it is still unknown "whether the known trauma was actually casual or only coincidental." In more than twenty of the cases, the trauma consisted of "prolonged exposure to dampness or to gruelling exhaustion." Jelliffe gives no final opinion on his collected material; when one thinks of the pains incurred in combing a century's literature only to have found so insignificant a number of cases of doubtful diagnostic accuracy and appearing often after almost no injury at all, one remains an unbeliever. As in multiple sclerosis, no greater incidence of these conditions was noted in the Great War in which so many individuals underwent a vast variety of injury and exposure. We have not seen this disease oftener in the oftener hurt people in our population, nor is it seen frequently in those of the first half of life, when accidents are common. In fact, the opposite is true: the disease is prevalent after age 35; not before. We think injury plays no role in the production of these two conditions.⁴⁷

Acute anterior poliomyelitis (infantile paralysis), is an acute infection of the spinal cord; it appears in epidemic form. It is not due to injury; it would need no mention had not a few Germans recorded in the medical literature a few rare cases occurring shortly after injury. Here again, coincidence is confused with relationship. This illness is widespread; it is not unusual to find the infection already incurred, when injury happens. This is true of other infectious disease. There is no relationship between this illness and injury.⁴⁸

46. The scarcity of appeal court decisions involving alleged causation or aggravation of amyotrophic lateral sclerosis by trauma (injury) indicates how infrequently this subject comes into litigation. The same is true of progressive muscular atrophy.

47. See n. 46, *supra*.

48. This is true, but the disease may still have medicolegal implications. For instance, nurses or physicians may accidentally contract it during the course of their employment in a hospital where contagious cases of the disease are being treated, or, again, a workman who has gone into hospital for diagnosis or treatment of compensable injury may accidentally contract poliomyelitis during his convalescence. See the following cases: *Children's Hospital Society of Los Angeles v. Indus-*

SYPHILIS OF THE SPINAL CORD

Syphilis⁴⁹ attacks the central nervous system frequently and, in the spinal cord, may give rise to many different clinical pictures according to the tracts affected. *Tabes dorsalis*, or locomotor ataxia,⁵⁰ usually appears many years after the first syphilitic lesion, or chancre. Injury cannot cause this condition, for it possesses an established infectious etiology. There is no proof for the highly theoretical repetitions in textbooks that an injury to a luetic person⁵¹ may produce *tabes dorsalis*, or other type of spinal syphilis when, otherwise, he would not have developed it. An ancient legend, this, to be destroyed. Medical experience, in the vast laboratory of World War I, despite the frequency of venereal infection and the most

trial Accident Comm. of Calif., 22 Cal. App. (2d) 365, 71 P. (2d) 83 (1937); Los Angeles County v. Industrial Accident Comm. 14 Cal. App. (2d) 134, 57 P. (2d) 1341 (1936). (A certain hospital was filled with patients who were being treated for highly contagious poliomyelitis; claimant, a nurse, worked in the ear and nose clinic of the county hospital where these patients were treated; other nurses and resident physicians became afflicted with the disease during the course of the epidemic; held: there was ample evidence to warrant an award made by the Industrial Accident Commission based on the finding that claimant contracted her disease out of and in the course and scope of her employment.) See, also, Lumbermen's Mutual Casualty Co. v. Vaughn, 174 S. W. (2d) 1001 (Tex. Civ. App. 1943); Wold v. Industrial Accident Comm., 42 Cal. App. (2d) 512, 109 P. (2d) 398 (1941).

49. *Syphilis*: A contagious venereal disease leading to many structural and cutaneous (skin) lesions, due to a micro-organism, the *Treponema Pallidum*. It is generally propagated by direct venereal contact or by inheritance. It may affect almost any part of the body at one stage or another of the disease. The availability of penicillin as an effective therapeutic agent has changed the prognosis of the disease considerably.

50. *Locomotor ataxia (tabes dorsalis)*: A chronic disease of the nervous system characterized by degeneration of the posterior columns of the spinal cord and the centers for pupillary light reaction and of the sensory nerve trunks, and manifested clinically by the presence of lightning-like pains, abolition of the tendon and periosteal reflexes, contracted pupils that do not react to light, peculiar paroxysms of pains (so-called *tabetic crises*) in the stomach, larynx or other viscera, trophic disturbances (nutritional disturbances) of the bones and joints, impairment of sensation (vibration and sense of position of the joints), impairment of sexual power, retention or incontinence of urine and feces and progressively increasing incoordination of movement (ataxia). It is a disease of middle life, especially frequent in males and is due to syphilitic affection of the nervous system. The pains and abolition of reflexes and pupillary disturbances are usually the first symptoms, forming the pre-ataxic stage of the disease. Its course is slow, usually progressive, and may be associated with general paresis, commonly known as "softening of the brain"; in such cases the condition is generally spoken of as *taboparesis*.

For cases involving alleged causation, activation of the dormant disease, or aggravation of established locomotor ataxia by injury, see the following: Behan v. John B. Honor Co., 143 La. 348, 78 So. 589 (1917), L.R.A. 1918 F., 862; Hershiser v. Chicago, B. & Q. R. R., 102 Neb. 820, 170 N. W. 177 (1918); See, also: Culbertson v. Kieckhefer Container Co., 197 Wis. 349, 222 N. W. 249 (1928).

51. *Luetic person*: A person infected with syphilis.

severe types of spinal injuries, found no more tabes dorsalis than is normally encountered; the same lack of increase in general paresis, or syphilis of the brain, was often reported; yet there were severe head injuries in the score of thousands. In fact, as Terbruggen points out, general paresis actually decreased during the War, an experience which should definitely dispel the fanciful idea of aggravation of an already syphilitic condition by injury. We should remember that, in syphilis of the central nervous system, spontaneous waves of improvement and of exacerbation are not infrequent. In tabes, long periods of remission may occur, followed by worsening. Should an injury precede such a spontaneous "flare-up," one naturally would tend to find a relationship between the two. This is inadmissible in that it has never been proven that there is more of this than the laws of chance permit. In the past decade, despite an avalanche of motor car injuries, tabes dorsalis has become a rare ailment, due probably to better treatment of early syphilis. This is the opposite of what one would expect, were injury of importance in localizing or aggravating an already existing syphilis. Kessler's very large experience has made him state that, "aggravation, even in the face of a severe accident, should not be admitted, if the tabetic state was already far advanced because of the inevitable disability without injury." We agree with this; we feel that injury does not induce tabes, when syphilis is in the system, nor modify its course to any appreciable extent when already established.⁵²

52. It is now agreed by all the writers on the subject, that injury cannot cause any form of neurosyphilis for the reason that syphilis is due to one cause only, namely, infection with a spirochaete, *treponema pallidum*. It seems to be conceded that neurosyphilis may be aggravated by injury in very special cases, but that few cases can comply with the criteria of proof which must be satisfied to demonstrate a causal connection.

Kessler says, for instance: "The relation between injury and tabes (dorsalis or locomotor ataxia) must be considered from the standpoint of aggravation only. Such a relation may be conceded if there is a history of severe injury involving the spinal cord, with symptoms indicative of spinal cord injury, and with an immediate onset of symptoms of tabes followed by a progressive course. Foix and Lagrange call attention to waves of aggravation occurring in tabes after long remissions or stationary phases, without apparent cause."

"Most of the reported industrial cases fail to meet the rigid requirements set forth above in order to prove a relation with an alleged accident. All accidents that are reported late should be given no credence. Furthermore, in view of the existing unsteadiness and dizziness of the tabetic individual, notice should be taken of the probability that the disease caused the accident instead of resulting from it. Aggravation, even in the case of a severe accident, should not be admitted if the tabetic stage was already far advanced, because of the inevitable disability without injury. In the face of a proved compensable condition, allowance can be made for complete disability." KESSLER, *op. cit. supra*, p. 549.

SYRINGOMYELIA

Syringomyelia is a spinal cord disease due usually to a congenital defect in the cord; there is secondary gliosis⁵³ and cavity formation in the affected region. It is most often found in the cervical (neck) and lumbar (lowback) enlargements of the cord. Characteristic atrophy (wasting) of hands or legs occurs, and sensory loss of typical, disassociated pattern. This condition is not produced by injury. However, by severe trauma, sudden hemorrhage may occur in a previously symptomless pre-existing cavity of the spinal cord. Rapid paralysis may follow such injury. This is known as *hematomyelia*; when this occurs, it may be said surely that the injury increased the symptoms. Hematomyelia may arise from injury to normal individuals without spinal disease. However, the cord hemorrhage comes from injury; the signs of its presence will be found at once after the injury.⁵⁴

SPINAL CORD TUMORS

New growths affecting the spinal cord occur often. They are not caused by injury. Certainly, any history of injury at the site of tumor formation

See, also, in the Symposium series "Scientific Proof and Relations of Law and Medicine (1st Series, April, 1943)" the following article: Merritt, H. Houston and Solomon, Harry C.: *Relation of Trauma to Syphilis of the Nervous System* (1943) 23 B. U. L. REV. 261; (1943) 117 ANNALS OF SURGERY 623. Merritt and Solomon limit the possible relationship of trauma to syphilis of the nervous system to aggravation. They say, in respect to tabetic neurosyphilis, (*i.e.*, tabes dorsalis or locomotor ataxia): "The problem here as in all cases of neurosyphilis, is one of precipitation or exaggeration of symptoms. Logically it would seem necessary that the injury involved the spine to be a factor in evoking the spinal symptoms or to the head for the mid-brain symptoms of tabetic neurosyphilis. They insist, further, that "for proof of causation, it must be shown that: (1) the injury was bona fide and severe enough to damage nervous tissue, and (2) that the symptoms had their onset immediately or within a reasonable interval after the injury." In conclusion these authors admit that: "the role of trauma in the precipitation or aggravation of the symptoms of neurosyphilis has not been adequately established on a scientific basis. The one proven fact is that neurosyphilis is due to the invasion of the nervous system by the spirochaete. The deleterious effect of trauma on the pathological processes of neurosyphilis can only be deduced. There is divergence of opinion as to the severity of injury necessary, and the time interval which must elapse between the injury and the appearance of symptoms. Since there is a basis for controversy in individual cases, it is the duty of the medical expert to keep within the limits of what is reasonable and not give the dignity of probability to conclusions that may only be considered as remotely possible. Likewise, it behooves the judiciary, granting that where there is reasonable doubt, the benefit of doubt should accrue to the victim, not to go too far in relating minor injuries of the head, or spine, or injuries to other parts of the body than the nervous system to the development of neurosyphilis."

53. *Secondary gliosis*: An attempt at repair which involves the development of neuroglial tissue; the latter is the supporting structure of nervous tissue.

54. For a case involving alleged traumatic hematomyelia, see the following: *Mohr v. Desimone & Sayers* 110 Pa. Super. Ct. 44, 167 Atl. 504 (1933).

is rarely obtained. When it is one must remember the number of back injuries having no aftermath of neoplasm. We feel, as do Elsberg and Davis, that injury plays no role in the production of spinal cord neoplasms.⁵⁵

55. Claimant had suffered a jarred or strained back. It was contended that this was causally connected with metastatic carcinoma of his spine; *held*: medical testimony that the jar or strain could not cause metastatic carcinoma nor accelerate the employee's death, sustained an award denying compensation. *Thomson v. Garten*, 58 N. E. (2d) 942 (Ind. App. 1945). There have been very few claims in court that tumors of the vertebral column or spinal cord were caused or aggravated by trauma. Still, in every case of alleged disability due to traumatic injury of the vertebrae or cord proper neurological and X-ray studies should be done to eliminate the possibility that all or part of claimant's symptoms are due to pre-existing disease such as an unsuspected tumor.