THE SYNDROME OF ACUTE ANTERIOR SPINAL CORD INJURY*

RICHARD C. SCHNEIDER, M.D.

Department of Surgery, Section of Neurosurgery, University of Michigan Medical School,
Ann Arbor, Michigan

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Immediately following trauma to the spine a syndrome of acute anterior spinal cord injury may sometimes be observed. This is characterized by an immediate complete paralysis with hypesthesia and hypalgesia to the level of the lesion together with the preservation of touch, motion, position and some of vibration sense. This syndrome may be the result of acute anterior spinal cord compression by a dislocated bone fragment or herniated disc or actual destruction of the anterior portion of the cord. Since a surgical lesion causing spinal cord compression is indistinguishable from a nonsurgical destructive one, the author feels that all of these patients should have an exploratory laminectomy and many of them should have a spinal fusion secondarily 2 or 3 weeks later. Emphasis should be placed upon the fact that these patients have attained their maximal neurological deficit and they do not present the usual criteria for surgery, namely, progression of neurological signs or evidence of blockage of the cerebrospinal fluid on the jugular vein compression test.

Thirteen cases are presented in detail to illustrate the pathological conditions producing this neurological pattern and the problems involved in their treatment.

In 1947 Kahn published a report which first described the rôle of the dentate ligaments in chronic anterior spinal cord compression by a posteriorly protruded cervical nucleus pulposus. Spasticity, hyperreflexia, disturbance of gait, weakness, and subjective sensory disturbances suggested the diagnosis of lateral sclerosis. Previously it had been thought that these symptoms were on the basis of compression of the anterior spinal artery, but this paper suggested that the spinal cord usually was firmly fixed over a posteriorly displaced disc by strong dentate ligaments, and that there was exertion of stress just above the dentate ligaments, resulting in pyramidal tract involvement. Kahn stated:

"In anterior spinal cord compression I believe that, with pressure over a period of time, the pyramidal tracts, because of the greater stress on them and the large size of their fibers, have more disturbance of conductivity than the pain fibers of the spinothalamic tracts, even though the latter are closer to the compressing mass, be it midline herniated nucleus pulposus or tumor. Touch is preserved because, even

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though the touch fibers of the ventral spinothalamic tracts may fail to conduct, there is still sufficient sensation carried in the more protected posterior columns to prevent the clinical detection of touch disturbance. Postural sense is preserved because the attachment of the dentate ligaments prevents the posterior columns, which are farthest from the compressing mass, from being pressed against the unyielding laminae" (Fig. 1).

**HERNIATED DISK**
*(Diagram of Stress Analysis)*

Fig. 1. Case 1. "Showing lines of stress in anterior spinal cord compression. Greatest stress is anterior on tracts disturbance of which would not be demonstrable by clinical tests. Secondary stress is directly on pyramidal tracts. The leg area is most lateral in pyramidal tracts, while the hand area is most medial, explaining usual sparing of the hands." (Courtesy of Dr. Edgar A. Kahn)

Bucy et al.\(^1\) regarded this mechanism as a possible explanation of the gradual development of neurological symptoms in these chronic cases.

In 1951 the author reported the first 2 cases\(^3\) of what was regarded as an acute phase of Kahn’s chronic cervical cord compression mechanism. This topic will be further developed in this paper. The photographs and a brief summary of the original 2 cases are reprinted here to aid in portraying the entire problem.

**CASES**

Case 1. J.S., a 36-year-old male, was pinned under his truck in an accident on Aug. 21, 1948. At St. Luke’s Hospital, Cleveland, he was found to have had immediate complete paralysis with a level of hypesthesis and hypealgesia to the C7 dermatome with preservation of touch. Cervical roentgenograms showed no fracture-dislocation; a lumbar puncture with jugular vein compression test showed no block. After no change in neurological status for 11 days a preoperative diagnosis of pos-
sible herniated cervical nucleus pulposus was made. Laminectomy was performed at C5, C6 and C7. The cord appeared normal. Transdural exploration revealed a totally extruded disc at C6-C7 interspace (Fig. 2). Within 11 days the patient moved his legs and a week later had some return of bladder control.

Two years after injury the patient had a residual Brown-Séquard pattern. He required one cane but worked as a nurseryman and could supervise the care of his sixty-four acres of trees.

![Image of spine and nerve roots]

**Fig. 2.** Case 1. "The 7th, 6th and 5th cervical nerve roots are exposed, and an extruded nucleus pulposus (arrow) is seen protruding through an incision in the anterior surface of the dura between the 6th and 7th nerve roots." (Courtesy of *Journal of Neurosurgery*)

**Case 2.** R.C., a 21-year-old college football player, sustained a cervical injury on Nov. 13, 1948. At Crile Veterans Hospital, Parma, Ohio, he was found to have had immediate complete paralysis with hypesthesia and hypalgesia to C6 dermatome bilaterally but touch and part of vibration sense were preserved. On lumbar puncture there was no block on the Queckenstedt test. Cervical roentgenograms showed a severe compression fracture-dislocation of the C5 vertebral body with some displacement of the inferior margin of the body posteriorly into the spinal canal (Fig. 3). It was suggested that the slight posterior displacement of the vertebral body could cause the symptoms, but that a herniated disc at C5-C6 interspace could not be excluded as a possibility. Laminectomy of C4, C5 and C6 was performed. A depressed fracture of C5 lamina was noted bilaterally. The normal-appearing spinal cord was displaced posteriorly. Transdural exploration revealed ex-
truded disc tissue in the spinal canal with several pieces of bone attached to it. There was also a slightly elevated bony ridge.

Three weeks later there was movement in the lower extremities. Twenty-one months after injury the patient drove fifty miles alone. He had some spasticity in his lower extremities but could walk on flat ground without aid. The patient was continuing his physical education career.

As a result of the study of the 2 cases presented in the original article, it was believed that one should add the acute anterior cervical cord injury syndrome to the list of indications for early operation in cases of acute spine injuries. Since that report 11 more cases of this syndrome have been added.

Case 3. At 3:45 P.M. on July 8, 1951, J.W., 19-year-old white male, dove into two feet of water, striking the lake bottom and had to be carried out by companions because of the immediate paralysis of all four extremities.

Examination. Upon admission to University Hospital, Ann Arbor, 3 hours later, he had a level of hypesthesia and hypalgesia to the C5 dermatome on the right and C6 on the left. Touch, motion and position senses were intact. Vibration sense was diminished below the level of the lesion. Deep pain was absent below the epigastrium. Supination of both forearms was possible but otherwise there was an areflexic motor tetraplegia. Priapism was present.

Roentgenograms of the cervical spine showed a compression fracture-dislocation of C5 vertebral body with a posterior fragment projecting into the spinal canal (Fig. 4). A lumbar puncture revealed normal spinal fluid dynamics.

Operation. At 9 P.M. on July 8, Crutchfield tongs were inserted and a laminectomy of C4, C5 and C6 was performed. The arch and spinous process of C5 vertebra were found disrupted and lying free. The dura mater was opened in the midline leaving the arachnoid intact, and the cord did not pulsate until the dentate ligaments had been cut bilaterally at two levels. There was no contusion or softening noted on the posterior and lateral aspects of the cord. Palpation and inspection revealed no disc, but only the bony ridge of the posteriorly dislocated C5 vertebra protruding anterior to the cord. The dura mater was left open to prevent any acute spinal cord constriction.

Course. Postoperative roentgenograms on July 24, 1951 demonstrated only partial realignment of the cervical spine (Fig. 5). Unfortunately the Crutchfield tongs
were removed inadvertently on Aug. 7, 1951, and roentgenograms demonstrated a marked redislocation of the cervical spine (Fig. 6). The tongs were reinserted and a more satisfactory realignment was accomplished with traction (Fig. 7). Spinal fusion from C2 to T2 was performed on Sept. 11, 1951, using a tibial graft. Traction was removed Oct. 23, 1951 and a Forrester collar was applied for support.

Fig. 4-7. Case 3. (4) Initial injury. Typical acute flexion or “tear-drop” fracture-dislocation. (5) Partial reduction after traction. (6) Redislocation after inadvertent removal of traction. (7) After re-application of traction.
On discharge the patient had a level to pin-prick at the T3 dermatome. Below this level all other sensory modalities were intact. There was good movement in adductor and fair power in abductor muscles of the shoulders. Flexors of the elbows and pronators and supinators of the forearm showed good strength. With the exception of very minimal voluntary movement of the toes of the right foot, there was no motion in the lower extremities. Deep reflexes of both upper and lower extremities were equal and hyperactive, with a positive pyramidal tract sign in the upper extremity bilaterally, and a positive right extensor plantar reflex.

Approximately a year after his injury the patient was readmitted for relief of spasms in his lower extremities. Marked contractures were present in the joints of all extremities. There was moderately good voluntary dorsiflexion of the right foot and toes and some flexion of the right knee, but poor extension of the leg. Little or no voluntary movement was found in the left lower extremity. Marked adduction spasm was present, with intermittent flexion spasms of the left lower extremity. There had actually been only a very slight degree of recovery, for superficial sensation had been intact at the time of discharge from the hospital. The patient refused anterior rhizotomy for spasms in his right leg and was discharged.

Comment. This patient had the typical acute anterior cervical cord injury syndrome with no evidence of a block on manometric testing of the cerebrospinal fluid. Early operation confirmed the presence of a posterior bony ridge pressing on the anterior surface of the cord at C5–C6 interspace, with no evidence of contusion or laceration. Postoperatively there was some degree of recovery but it was minimal. The neurological residual could have resulted from the severe initial contusion of the cord with irreparable damage or it could have been the result of further injury by redislocation of the spine, or perhaps have been related to the final angulated fusion of the involved vertebra, so that there had been an incomplete correction of the deformity, causing signs of chronic anterior cord compression. A typical “tear-drop” fracture of the C5 vertebral body was noted with the anterior inferior border of the vertebra dropping forward and the posterior inferior border protruding posteriorly into the spinal canal. This was similar to the roentgenographic findings in Case 2.

Case 4. H.P., 16-year-old school boy, sustained a severe cervical spine injury while tumbling in a school gymnasium at 1 p.m. on May 14, 1948. He stated that he had suffered an immediate complete paralysis of all four extremities with a loss of sensation below the neck. A local physician took cervical roentgenograms which revealed a fracture-dislocation of C4 on C5 vertebral body (Fig. 8). He was transferred to University Hospital, arriving at 2 a.m. on May 15, 1948.

Examination. There were minimal movements of the biceps and deltoid muscles with involuntary twitching of the left thumb. No movement was observed in the pectoralis, triceps, or muscles of the forearms or lower extremities, and there was complete areflexia. A lumbar puncture demonstrated a spinal fluid pressure of 150 mm. of water which rose rapidly to 250 mm. upon jugular vein compression bilaterally and then fell slowly, suggesting an incomplete block. The latter was ascribed by the resident to “a faulty mechanism.”

2nd Admission, May 1952. The patient’s presenting complaint was severe involuntary spasms with contractures of the fingers.
Examination. There was a level at T6 dermatome below which superficial pain and temperature sensations were absent. Hyposthesia was present to C6 dermatome. Motion and position senses were preserved throughout the body. Vibration sense was present except over the right external malleolus. Touch was good except in the left lower extremity where responses were only spottily correct. Generalized spasm was found in all extremities, but the fingers were contracted down into fists and the legs were frequently extended rigidly. There was sufficient spasm occasionally to precipitate the patient out of the bed onto the floor. There was bilateral wrist and left patellar clonus. Reflexes were hyperactive throughout. Extensor plantar reflexes could not be elicited, probably because of extreme extensor spasm.

Cervical roentgenograms at this time demonstrated a loss of the intervertebral space at C3-C4, and C4-C5 with apparent fusion and slight anterior dislocation of C3 on C4 vertebral body and posterior displacement of C4 on C5 body (Fig. 9). Lumbar puncture showed a pressure of 150 mm. of water with a prompt rise to 300 mm. on jugular compression and a rapid fall to the original level within 10 seconds. Cervical myelogram (Fig. 10) revealed midline defects in the contrast medium at C3-C4 and C4-C5. Motion, position and vibration senses were further impaired in the lower extremities after myelography.

Operation, May 19, 1952. Cervical laminectomy of C3 through C6, in spite of the apparent fusion on the roentgenogram, revealed marked instability of both the 3rd and 4th cervical vertebra but no evidence of a fracture. There were marked adhesions between the arachnoid and the overlying dura mater laterally at the C4-C5 interspace and after cutting the dentate ligaments at four levels bilaterally, gentle
retraction of the cord showed the bony kyphosis demonstrated in the roentgenogram. No herniated disc could be demonstrated. There was a definite variation in the level of insertion of the dentate ligaments with a more posterior displacement of the spinal cord at C3-C4 and C4-C5 than at the other two exposed interspaces. The dura mater was closed.

Course. Postoperatively the patient lost the wrist clonus, and tonus in the upper extremities was decreased about 50 per cent, but the mass reflexes in the lower extremities were slightly accentuated. Finally anterior rhizotomy was performed in the latter part of June 1952. This procedure adequately controlled the spasms of the lower extremities. Marked spasticity and clonus remained in both upper extremities.

Comment. When this patient was admitted to the hospital at the time of his injury, he had an acute anterior cervical cord injury syndrome. There was no progression of signs and only partial block on jugular compression test, consequently he was not operated upon. No regression of symptoms had occurred over a period of 4 years. The cervical exploration should have been performed as soon after injury as the patient's condition permitted. The pronounced instability of C3 and C4 vertebral bodies even 4 years after injury suggests the importance of early fusion of cervical spines associated with these incomplete lesions, and demonstrates how misleading the roentgenograms may be in interpreting stability of the cervical spine. This is a case of the acute anterior cervical cord injury syndrome without a complete block, which did not show recovery after 4 years. Another "tear-drop" fracture is demonstrated with associated prolonged spinal instability.

Case 5. M.S., a 17-year-old tumbler, slipped and struck upon the vertex of his head at 8:45 A.M. on March 29, 1951. He was not rendered unconscious, but had complete paralysis of all four extremities except for questionable movement of his right great toe. There was headache, with pain in the neck and numbness from the neck downward over the trunk and extremities.

Examination. There were hypesthesia and hypalgesia to the level of the C4 dermatome, but touch and position senses were preserved bilaterally throughout the entire body. Both lower extremities were completely paralyzed. The patient could extend the wrists but had no motion in the fingers. There were no extensor plantar reflexes. Cervical spine roentgenograms revealed a compression fracture-dislocation of the C4 vertebral body with a 3 to 4 mm. posterior displacement (Fig. 11).

Course. Crutchfield tongs were applied. On March 31, 1951, lumbar puncture was done and on jugular compression test there was a rise in pressure to 300 mm. of water and a slow drop suggesting the possibility of partial subarachnoid block. In this spinal fluid specimen with only 1 blood cell/high power field the Pandy test was reported as ±1 and total protein was 63 mg./100 cc. On April 14, 1951, lumbar puncture showed a rise of pressure to 240 mm. of water and a slow fall to normal. This specimen contained no blood cells and total protein was 110 mg./100 cc.

On April 23, 1951, the sensory level was gradually receding, and there was onset of return of function in the lower extremities with an ability to flex the fingers of both hands. A plaster body jacket was applied on April 27, 1951 at the time of removal of the Crutchfield tongs. A back brace was substituted for this appliance on June 14, and the patient was able to ambulate moderately well. There was a residual urinary volume of 75 cc. On August 3, the patient was up in a walker. It was dis-
covered that his sensory level, which had receded to T12-L1, had now risen again to the T8 dermatome. There was weakness in the extensors of the left wrist with wrist drop. Right foot drop was also present. Bilateral extensor plantar reflexes and ankle clonus were elicited. Bladder control and bowel function were good.

Recheck stability roentgenograms of the cervical spine on May 21, 1952 exhibited motion at C3-C4, while there was none apparent at C4-C5 and at C5-C6 (Fig. 12). By August, 1953 the patient began to have some increasing spasticity in

the lower extremities and was readmitted for further studies. A cervical myelogram on Sept. 1, 1953 demonstrated a marked narrowing of the cervical spinal canal (Figs. 13 and 14).

Operation, Sept. 2, 1953. At decompressive laminectomy, bilateral healed fractured laminae of C4 and C5 were removed. These laminae were thinned and osteoporotic. Parts of the C3 laminae were removed. The bony spine was extremely unstable. The dura mater was opened in the midline and the dentate ligaments, which were very thin, were sectioned at two levels. At the C4-C5 interspace there was a 3–4 mm. central bony protrusion. The dura mater was left open, protected by the intact arachnoid in the center of the dural aperture. An inverted V-shaped bone graft was inserted bilaterally from C3 to C6 spinous processes.

Course. Postoperatively the patient was able to walk with a cane, using a spring
kick-up brace. Hypesthesia and hypalgesia were spotty to a level of T10 dermatome on the right side. Temperature sense was not clearly defined over the lower extremities, particularly on the right side. Motion and position senses were intact bilaterally. There were bilateral pyramidal tract signs with increased tonus in the upper and lower extremities, ankle clonus and extensor plantar reflexes bilaterally.

The patient has continued to improve, so that he is now back to his preoperative status.

Comment. This patient had a typical acute anterior cervical cord injury syndrome, combined with the frequently observed acute flexion or “tear-drop” vertebral body fracture. It is particularly interesting to note that he did not have a complete block on jugular compression test on two days, 5 days apart, and yet he had a definitely progressively elevated protein without any blood being present in the specimen. This would suggest irritation of the cord, possibly caused by bony pressure upon its edematous anterior surface, and shows that one does not need a complete block or hemorrhage to have an elevated protein. Perhaps it merely indicates contusion of the cord, but in that case the protein should not have become progressively elevated. Cervical myelography may be extremely dangerous because of the degree of narrowing of the canal. The patient apparently seemed to be making a very nice recovery.
from his injury initially but then began to have regression with increasing spasticity. In retrospect, the patient should have been operated upon as soon after his injury as his condition permitted and then either primarily or secondarily had a spinal fusion.

Case 6. L.C., an 18-year-old boy, dove into shallow water at 4 P.M. on July 11, 1953. He sustained immediate paralysis of both legs with marked weakness of his arms and hands, and had to be dragged from the water by his companions. On admission to the hospital the patient complained of pain in the neck.

Examination. There was marked weakness of all movements of the hand, particularly extension and flexion of the wrist. There was complete paralysis of the lower extremities. Hypalgesia and hypesthesia were demonstrated to the level of C7 dermatome. Position sense and motion sense were impaired but not absent, while vibration sense was lost below the level of C7 dermatome. The biceps and triceps jerks were hypotonic, abdominal reflexes were absent, and the knee and ankle jerks were absent. There were no definite extensor plantar reflexes.

Roentgenograms of the cervical spine revealed a fracture of the anterior inferior lip of the 6th cervical vertebra (Fig. 15). Lumbar puncture demonstrated a partial block upon jugular vein compression.

Operation. The laminae of C6 and C7 vertebrae were found to be fractured bilaterally and a central fragment was depressed downward on the cord. Decompression was carried out by sharp dissection. The dura mater was opened in the midline, and the cord appeared normal. Upon sectioning the dentate ligaments, two on the right side, and one on the left, the cord pulsated normally. Transdural exploration anteriorly revealed a bony elevation at the C6-C7 intervertebral space. The dura mater was closed tightly.

Course. Postoperatively the patient had some difficulty with atelectasis and mental confusion. On the 4th day after operation while secretions were being removed by suction from the nasopharynx, he suddenly ceased breathing. All attempts to revive him were of no avail.

Necropsy. There was evidence of postoperative extradural hemorrhage at the site of the wound, but the dura mater had been closed tightly. Upon opening the dura mater the cord now appeared to be swollen and purple on its posterior surface.
and at the upper margin of the dural incision at C4 lamina there was a definite constriction (Fig. 16). Multiple incisions were made across the cord at the levels of C3 through T2 (Fig. 17). There was definite dissection or cavitation of the lesion as high as the C3 level. At the site of the fracture-dislocation at C5-C6 there was marked destruction of the cord and it is scarcely conceivable that the patient could have had a normal-appearing posterior cord at the time of operation. The damage which involved the central portion of the cord could be visualized to T2 level.

Comment. This patient presented the anterior cord injury syndrome, and is the only one who succumbed to the spinal cord injury following operation. There were several errors. First, the extent of laminectomy was not sufficiently large. From this experience the author is inclined to feel that at least three laminae should be removed so that the dura mater may be incised in the midline and the dentate ligaments cut at least at three levels. If this
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degree of exposure is not obtained, there is too much of a tendency to press upon the cord in an attempt to reach the dentate ligaments and section them. The orthopedic surgeons are not anxious to have the neurosurgeon remove this amount of bone, but adequate exposure is necessary. The second error was the failure to obtain a completely dry wound and prevent any degree of extradural hemorrhage. Thirdly, it is doubtful whether the dura mater should have been closed over the cord even though the latter structure

![Image](image_url)


appeared to be quite normal and free of edema. One should probably have allowed for more postoperative swelling. There was definite evidence of a constriction at the upper margin of the incision at the C4 level. It was probably a combination of compression by the hemorrhage and the constricting dura mater which accentuated anoxia to the already edematous cord and was responsible for progressive edema to the C4 segment, resulting in a respiratory death. A typical “tear-drop” or flexion fracture was present.

*Case 7.* C.A., a 14-year-old boy, dove into a shallow pool striking his head on the bottom. He could not move his four extremities upon his removal from the pool. At the local hospital the intern confirmed the fact that there was an areflexic paraplegia,
but merely made the note "sensory examination grossly normal." Priapism developed shortly after the injury. Cervical roentgenograms demonstrated a compression fracture of the C5 vertebra with minimal fracture-dislocation. Traction was applied. Examination 48 hours after admission noted no change in motor power, but a level of hypalgesia above the nipple line with motion and position sense preserved.

On April 16, 1952, 5½ weeks after injury, he was transferred to University Hospital for intensive physiotherapy.

Examination. Vibration, position and touch sensations were intact throughout but there was a definite level of hypalgesia to the T2-T3 dermatome. There was inability to flex the fingers bilaterally with extension occurring at the wrist on attempted flexion. Impairment of extension of the arms at the elbows was present bilaterally with complete paralysis of the lower extremities. Deep reflexes were symmetrically hyperactive with bilateral extensor plantar reflexes and sustained ankle clonus.

Recheck cervical roentgenograms showed a vertical splitting fracture of the C5 vertebral body (Fig. 18) with minimal anterior dislocation of C5 vertebral body on C6 (Fig. 19), suggesting that a bony ridge was pressing anteriorly on the spinal cord. On lumbar puncture the spinal fluid manometrics were normal. A diagnosis of anterior cord compression was made.

Operation, April 18, 1952. A C5, C6 and C7 laminectomy was performed, the dentate ligaments being cut at these interspaces, and thereafter the normal-appearing cord was noted to pulsate freely. No protruded disc could be palpated but the ridge of the slightly displaced C5 vertebral body was noted. The wound was closed in layers.

Course. Within 36 hours the patient had movement of his toes bilaterally and some flexion of the fingers of his right hand. By April 27, 1952 there was some improvement in strength in the lower extremities and marked return of motor function in the upper extremities, including the intrinsic hand muscles. His indwelling catheter could be removed. Because of a tendency toward moderate kyphosis (Fig. 20) on removal of the traction apparatus 9 weeks after injury a spinal fusion was performed on June 2, 1952 (Fig. 21).

Comment. This patient had the typical anterior cervical cord injury syndrome. Since there was no block on manometric studies, critics will observe that this patient might have recovered spontaneously. However, after 5½ weeks with no improvement in neurologic status, there was a recovery of motor power in the fingers and the toes within 36 hours postoperatively. This quick response to decompression appears to be more than a mere coincidence. The minimal degree of dislocation of the anterior floor of the spinal canal was probably sufficient to cause pressure on the anterior cervical spinal cord, which was restrained by the dentate ligaments. The force of the injury was not severe enough to pinch off the anterior inferior margin of the vertebral body to form the typical flexion fracture.

Case 8. S.S., a 31-year-old man, was involved in an automobile accident at 1:30 a.m. on June 29, 1952. He was immediately paralyzed.

Examination. Upon admission to the University Hospital at 6 a.m. a 1 cm. right frontal scalp laceration was noted. There was a complete flaccid paralysis of the lower extremities with some weakness of the biceps muscles bilaterally. There was
a level of hypalgesia and hypesthesia to C7 dermatome bilaterally. Touch, motion and position senses were intact. Areflexia of the lower extremities was present with an absence of the right biceps reflex. The biceps jerk on the left was present and there were bilateral hypoactive triceps reflexes.

Cervical spine roentgenograms revealed a fracture-dislocation of C6 vertebral body anteriorly on C7 with comminution of the C7 vertebral body (Fig. 22). There was displacement posteriorly of the superior rim of the C7 vertebral body into the spinal canal.

1st Operation. The right frontal scalp laceration was debrided by Dr. Lloyd Lemmen at 8:30 A.M. and a linear fracture was demonstrated which extended into the right temporal area. Two burr holes over the site of the fracture showed a small extradural hematoma. Crutchfield tongs were applied.

A laminectomy of part of C4, C5, C6 and part of C7 laminae was performed. It was found that the laminae of C5 and C6 were fractured bilaterally and were impinging upon the dura mater. There was a moderate amount of extradural hemorrhage. The dura mater was opened in the midline, and the spinal cord appeared normal. The dentate ligaments were cut at three levels. The dura mater at the point of exit of the C6 nerve root was torn and had to be sutured. The remainder of the dura mater in the midline could be readily closed.

Course. Because of respiratory distress, a tracheotomy was performed at 12:30

P.M. The patient’s arms showed an increase in strength shortly after operation. On July 11, 1952 he exhibited the first voluntary movement in his lower extremities.

2nd Operation. On Aug. 6, 1952, a cervical spinal fusion was performed, using a left tibial bone graft (Fig. 23). This was fitted in place as a clothes-pin graft between the spinous processes of C2 and C7 and was fastened with #16 steel wire.

Course. On examination on Aug. 10, 1952 there were good movements in all four extremities, including the toes. There was a vague level of hypalgesia to the C6 dermatome, but touch, motion, and position senses were intact. Sensation was now noted on irrigation of the bladder, but true bladder control had not been established.

On follow-up examination on July 15, 1954 good motor power with increased tone was elicited in all four extremities and there was almost complete recovery of sensation. Bladder control still was poor, requiring the use of a leg urinal during working hours. He is now working full time selling electrical appliances.

Comment. This case presented a typical pattern of an anterior cervical spinal cord compression. The tracheotomy was necessary to remove tracheobronchial secretions adequately. Surgical intervention seemed to be of definite value to this patient.

Case 9. K.L., a 41-year-old farmer, while driving his truck ran off the road and hit a tree on Dec. 29, 1952. There was an immediate paralysis of all four extremities, except for minimal movement in the fingers of the right hand. He was numb from the chest down but could perceive touch. There was no interval of unconsciousness. About the 2nd day the patient began to have some return of movement in his arms, but the legs remained paralyzed. There was no bladder and bowel control, but the
patient could distinguish bladder distention during irrigation. He was having some respiratory difficulty and exploration at this time was deemed inadvisable.

Examination revealed about 25 per cent impairment of the biceps muscle function bilaterally. There was complete flaccid paralysis below this level. Light touch, deep touch, and position senses were preserved. Partial vibration sense was noted in the ankles and at the knees. There was loss of pain and temperature sensations to level of T4. Biceps reflexes were faint bilaterally; triceps reflexes were absent. There was a complete lower extremity areflexia.

Cervical roentgenograms showed an acute flexion fracture of C6 vertebral body with dropping forward of anterior inferior margin and posterior margin backward into the spinal canal (Fig. 24). Lumbar puncture revealed clear colorless CSF. Queckenstedt test was negative.

Operation, Jan. 15, 1953. Laminectomy was done with removal of the laminae of C4, C5 and C6. All of these laminae were fractured, but not depressed. The dura mater was intact, and upon opening it in the midline the spinal cord appeared to be normal. Exploration anterior to the cord showed no evidence of any disc or anterior spinal cord compression. The dura mater was closed in the midline since the cord did not appear swollen.

Course. Six months postoperatively the patient had slight movement in the toes of the right foot and was able to flex the right leg slightly at the knee. He was able to close the fingers of the right hand fairly well, but the grip of the left hand was markedly impaired.

Comment. This patient was not operated upon early because of rather marked respiratory embarrassment at the time of admission. There was some posterior displacement of the body of the C6 vertebra into the spinal canal. The typical “tear-drop” fracture of the anterior portion of the body was found. Unfortunately, this patient probably had more anterior cord destruction than compression.

Case 10. M.V., a 40-year-old white female, was riding in a truck with her husband when it overturned at 3 a.m. on May 8, 1953. She was not rendered unconscious and noted immediate complete paralysis with numbness and tingling in her
arms and legs. She was admitted to University Hospital a few hours after her injury.

Examination. There was no movement of the arms or legs. Position, deep pain, and touch sensations were present. Vibration sense was impaired over the malleoli and iliac crest bilaterally. Hypalgesia and thermanesthesia were present to the level of C6 dermatome. There were pyramidal tract signs in the upper extremities and there was withdrawal on attempting to elicit extensor plantar reflexes. The biceps, triceps, knee, and ankle jerks were all mildly hypoactive. Upon lumbar puncture no block was found on performing the Queckenstedt test. The CSF was bloody.

Roentgenograms of the cervical spine revealed a unilateral dislocation of C3 on C4 vertebra with disruption of the unco-vertebral joint on the right side (Fig. 25).

Course. On May 16, 1953 the patient recovered the first faint movements of the toes on both feet. She was transferred to her local hospital for a period of 5 weeks and then readmitted to University Hospital.

She had improvement in flexion of the hands with greater power on the left side. There was fair strength in both triceps muscles. Movement was now present in all muscle groups in the lower extremities, being more marked on the left side. No definite sensory deficit could be established. There was hyperactivity particularly of the lower extremity reflexes, with bilateral ankle clonus. There were bilateral extensor plantar reflexes.

Check cervical spine roentgenograms demonstrated a very minimal dislocation at the C3-C4 intervertebral space.

Operation. Complete laminectomy was done of C3 and C4 vertebrae and the dura mater was opened in the midline, preserving the arachnoid. The arachnoid was then incised laterally so that the dentate ligaments could be cut at two levels. Palpation anterior to the cord demonstrated a slight dorsal protuberance at the C3-C4 in-

tervertebral space. The dura mater was closed in the midline, and a firm iliac bone graft was inserted between the spinous processes of C2 and C5. This graft consisted of an imbricated notched single piece of bone inserted between the spinous processes on either side. Bone fragments were then placed along the margins of the roughened facets at the lateral border of the wound. A piece of wire was used to maintain position of the graft, being anchored to the C2 and C5 spinous processes (Fig. 26).

**Course.** Follow-up examination on June 28, 1954 showed that the patient had some spasticity in all four extremities but she could walk with crutches. She was able to use her hands well enough to carry out 60 per cent of all functional tests. With more intensive physiotherapy she should have an excellent prognosis.

**Comment.** It would have been dangerous to operate upon this patient at the time of admission in view of the possibility of producing phrenic paralysis because of the high site of the lesion. In retrospect it would have been much better to have applied traction, reduced the fracture-dislocation with tongs, and performed a laminectomy with section of dentate ligaments 2 or 3 weeks after injury. Spinal fusion could probably have been performed at the same time as the laminectomy, thereby saving 5 or 6 additional weeks in skeletal traction.

**Case 11.** R.S., a 19-year-old male, was involved in an automobile accident on Aug. 20, 1950. He was unconscious for 15 minutes and had immediate paralysis of arms and legs, but it was reported that “some sensation was intact.” Motor power was never regained in any of his extremities. The patient was placed on a Stryker frame in October 1950, and for a period of 3 months prior to transfer he had marked flexion spasms of the upper and lower extremities. On March 2, 1951 he was admitted to University Hospital.

**Examination.** There was a good range of motion present in the neck, but there was spastic paralysis of all four extremities with slightly more active movement in the right shoulder than the left. Touch sense was preserved over the entire body. Motion and position senses were absent in lower extremities but present in the upper. Impairment of vibration sense was noted to the T7 dermatome level. Hypalgesia was present to C4 dermatome. There were normal knee and ankle jerks but the biceps and triceps were symmetrically hyperactive. Mass reflexes occurred bilaterally upon the slightest stimulation. A definite left extensor plantar reflex was observed with an equivocal one on the right.

Cervical spine roentgenograms revealed a compression fracture of the superior
aspect of C5 with minimal anterior dislocation of the C4 vertebral body on C5 (Fig. 27). Lumbar puncture revealed no block on manometric study and a total protein of 31 mg./100 cc.

A diagnosis was made of a posteriorly protruded disc or pressure on the anterior surface of the cord because of the bony ridge of the dislocated vertebral body.

Operation. March 6, 1951. Laminectomy was performed at C4, C5 and C6. Marked instability of C5 and C6 vertebrae was demonstrated. Little epidural fat was found at this level and the ligamentum flavum at the C4-C5 interspace was torn. The dura mater was opened in the midline. Adhesions bound the arachnoid to the spinal cord posteriorly at the posterior columns. The dentate ligaments were sectioned at three levels to permit good exposure and anterior exploration revealed a slight bony posterior projection of the C5 vertebral body. Firm adhesions had to be dissected away gently anteriorly and at this point in the procedure the patient ceased breathing on several occasions, but respirations started after manipulation of the cord was halted. No protruded disc was found. At the level of the adhesions there was also firm scarring of the cord. The dura mater was closed, and the wound was sutured in layers.

Course. The patient was discharged on April 22, 1951 with no change in his neurological status except that there was some adduction and abduction of the right thumb, but unfortunately his spasms were slightly more severe. Anterior rhizotomy was rejected by the patient and relatives.

Comment. This patient was seen 7 months after his injury and presented the picture of anterior cervical cord injury without a block on spinal fluid manometric studies. There could have been an immediate complete tetraplegia with a blockage of the spinal fluid on jugular compression test immediately after injury. The symptoms might, on the other hand, have been caused by prolonged spinal cord edema which left the patient with the syndrome of anterior cord compression, or possibly by traumatic thrombosis of the anterior spinal artery. The latter, however, may cause segmental changes. If there had been a spinal fluid block immediately after injury, according to the usual criteria the patient should have been operated upon. We feel that even if there was no obstruction on the Queckenstedt test the neurologic pattern of anterior cervical cord injury would have suggested the possibility of a surgical lesion requiring decompression. Operation was performed in this case with little hope of accomplishing anything. It was urgently requested by the patient and his family.

Case 12. A.C., a 53-year-old man, was involved in an automobile accident on March 14, 1951. He was able to walk away from the site of the accident, but about 5 minutes later there was incomplete paralysis of his arms and legs. He was taken to a local hospital, and 5 days later, when his condition became progressively worse, he was transferred to University Hospital, on March 19, 1951, at 6 P.M.

Examination. The patient was able to move his toes very slightly, but otherwise there was little movement in the lower extremities. At 7:30 P.M., it was observed that there was complete paralysis of the lower extremities with no movement of his fingers and there was weakness of the triceps and biceps, bilaterally. Motion, position, vibration and superficial touch sensations were intact. There was a vague level of hypalgesia below T9 bilaterally. Deep reflexes were equal and active bilaterally,
and there was an equivocal extensor plantar reflex bilaterally. The patient was incontinent of urine and his abdomen was markedly distended.

Cervical roentgenograms revealed a fracture-dislocation of C5 vertebral body on C6 (Fig. 28). Crutchfield tongs were inserted. The patient was then rotated carefully on his side maintaining the head in the same plane of the body, and a lumbar puncture was done. There was a complete block on the Queckenstedt test. The spinal fluid procured at this time was reported as xanthochromic but pale, and contained 250 mg. of protein. Shortly after lumbar puncture the sensory level for motion, position, and hypalgesia was at T4 dermatome, indicating a rise of approximately 5 segments.

A diagnosis of anterior spinal cord compression was made.

Operation. Cervical laminectomy was performed under intratracheal anesthesia with the removal of laminae C4, C5 and C6. The ligamentum flavum was completely avulsed between C5 and C6, and C5 vertebral body was displaced anteriorly so that the tip of the spinous process of C5 came just to the level of the lamina of C6. After laminectomy the dura mater began to pulsate and bulge posteriorly. The dura mater was opened in the midline, and the arachnoid maintained intact. The arachnoid was incised laterally and the dentate ligaments were cut at three levels. At C5 the cord was markedly elevated and then dipped down anteriorly at the C6 vertebral level. The vessels and the cord itself appeared to be normal after sectioning the dentate ligaments and exploring anterior to the cord. There was no evidence of any herniated
disc. It was observed that after the dentate ligaments were cut the cord itself rose upward into the wound. The dura mater was left open. Cervical traction was maintained, but complete reduction of the fracture-dislocation was not accomplished because the facets were still locked (Fig. 29).

Course. A week after operation the patient's blood pressure dropped to 70/40, the pulse rate was 98, and his temperature was 102°. At 9:30 that morning his respirations became irregular, the blood pressure continued to fall and he was placed in a respirator. Transfusions were given to the patient. He complained of no pain but had a progressively downward course. His respirations ceased at 5 a.m. on March 27.

Necropsy. There was a subtotal extrusion of a disc into the spinal canal at C5-C6 interspace (Fig. 30). There were a few minute areas of contusion on the spinal cord at that level (Fig. 31). Acute purulent confluent lobular pneumonia was found and there were acute multiple abscesses in the prostate. Multiple acute gastric ulcers were discovered. The cause of death was listed as severe hemorrhage into the stomach from a bleeding ulcer.

Figs. 30–31. Case 12. (30) Postmortem appearance of anterior surface of spinal canal. Arrow indicates partially extruded cervical nucleus pulposus. (31) Cervical cord shown at necropsy. Arrows indicate the level of the cord that lay immediately above the extruded disc, demonstrating that there is little gross anatomical damage to the cord.

Microscopic sections of a portion of the cord that appeared normal grossly revealed extensive degenerative changes within the cord itself. Perivascular edema and extensive myelin degeneration of the cord were present.

Comment. This patient exhibited a progression of neurological signs from the time of accident until he entered the first hospital. On the basis of this,
operation was indicated. By the time he arrived at University Hospital after a trip of 60 miles without the benefit of traction, a true anterior cord compression syndrome had developed. The edema was very pronounced so that when lumbar puncture was performed, there was evidence of complete block, which was another indication for operation. If traction had been instituted during the trip to University Hospital, the block might not have been present. The case demonstrates how rapidly progression of neurological signs can occur in the face of complete block on lumbar puncture.

It should be noted that ulcerations of the stomach and duodenum have been reported in conjunction with injuries to the cervical area. The author is unfamiliar with any definite mechanism that might be the cause of such lesions.

Case 13. H.H., a 33-year-old housewife, was involved in an automobile accident on the afternoon of July 13, 1953. She was thrown from the car and struck directly upon her back. She regained consciousness at a local hospital, but was suffering from traumatic shock.

Examination. By the morning of July 14, 1953 the neurosurgical consultant found a complete areflexic paraplegia except for an equivocal flexion of the right
great toe. There were hypalgesia and hypesthesia to the T4 dermatome but vibration, motion, and position sensations were retained over the entire body. Roentgenograms showed a fracture-dislocation of T3 on T4 vertebral body (Figs. 32 and 33), and T10 on T11 vertebral body (Figs. 34 and 35). Fractures of the right clavicle and scapula and the right transverse processes of L1, L2, and L3 were also demonstrated.

*Operations.* On July 17, 1953, the lamina were removed from T2 to T5, bilaterally. There was marked anterior compression and left lateral displacement of the spinal cord by the T4 vertebral body. The T3 nerve root was sacrificed and a portion of the T4 vertebral body was chiseled away in order to accomplish decompression of the cord anteriorly and laterally.

On July 22, 1953, a complete laminectomy of T9 through T11 was performed. A portion of the fractured T11 vertebral body had to be removed anterior to the cord, and in order to accomplish this the right T10 nerve root had to be sacrificed. The dura mater was opened in the midline and the dentate ligaments were cut at two levels. The cord did not pulsate, but appeared to be normal. A catheter was passed upward with no evidence of blockage of the subarachnoid space. The dura mater was left open.

*Course.* By August 10, 1953 there was a drop in the level of hypalgesia from the T4 dermatome to T10, bilaterally. There was no movement of the lower extremities, but the Achilles reflexes could be obtained. On August 30, 1953, the patient could
move her left ankle, and within a week or two all movements had returned in the left lower extremity, but there was only minimal strength on the right side.

By July 1, 1954 the patient had improved markedly and was able to stand for a short time without the use of braces. Spasticity in her lower extremities had gradually diminished and her bladder control had almost entirely returned. However, about July 15, 1954, the left leg became numb around the region of the knee, and the left quadriceps muscle became weak. Strong burning sensations developed from the hips downward and it was difficult to determine whether there was some increasing spasticity that was masking the strength in the left leg, or a true loss of motor power.

Comment. Originally the syndrome of anterior spinal cord injury was described merely in association with acute cervical spine trauma. It was thought that it would not be found in thoracic spine injuries because the force required to dislocate the thoracic spine necessarily must be very great to overcome not only the stability of the spine itself but also the added fixation effect of the thoracic cage. Because of the narrowness of the spinal canal, any such force causing disruption almost certainly would completely transect the spinal cord. In this case the patient's cord was apparently preserved because she struck very hard and flat on her back upon the ground. The facets were fractured at the two sites without acute hyperextension or acute flexion of the spine, thus permitting the laminae to be displaced dorsally, accomplishing decompression with preservation of the spinal cord. This patient unquestionably was aided by surgical intervention. If movement in the acute phase of convalescence had not irreparably injured the tightly compressed spinal cord, then chronic spinal cord compression still might have caused neurological deficit as more bone was laid down at the fracture sites (see Case 5).

CONCLUSIONS

1. The syndrome of anterior cervical cord injury is characterized by an immediate complete paralysis below the site of the lesion associated with a level of hypalgesia and hypesthesia consistent with the lesion, with preservation of motion, position and touch, and some vibration sense. In only 1 case was the syndrome not present immediately but evolved rapidly within a few hours (Case 12).

2. The anterior spinal cord injury syndrome may be associated with an isolated herniated cervical nucleus pulposus (Case 1), a herniated cervical nucleus pulposus in conjunction with a fracture-dislocation of the cervical spine (Cases 2 and 12) or fracture-dislocation alone (Cases 3, 4, 5, 8, 9, 10), and compression fracture of the cervical vertebra (Case 7). In 2 of the cases anterior spinal cord destruction was visualized at necropsy or at operation (Cases 6 and 11). The syndrome has been observed in only 1 case of thoracic injury and that was associated with a double fracture-dislocation (Case 13).

3. There was complete blockage on the Queckenstedt test in only 1 patient (Case 12). This patient was the only one who did not show the pure syndrome, for there was a marked progression of neurological signs immedi-
ately after injury and secondarily a block developed immediately after lumbar puncture. With the exception of this case none of these patients was operable under the usual criteria of the presence of a complete subarachnoid-space block or progression of neurological signs. It is impossible to differentiate those patients who are suffering from anterior cord compression by a lesion, with the restriction of posterior cord displacement by the dentate ligaments, from those who have anterior cord destruction. Therefore, early operation is advisable, with section of the dentate ligaments and inspection of the interspaces for a surgical lesion.

4. In general, myelography is contraindicated in this group of patients for fear of causing further damage to the already injured spinal cord.

5. These patients have been operated upon at varying intervals, ranging from 18 hours to 4 years after injury with varying degrees of recovery. The most striking result was observed in Case 7. This patient had not moved his extremities for a period of 5½ weeks after injury, and yet 36 hours after cervical laminectomy with section of the dentate ligaments, he began to move his fingers and toes. This suggests that early operation is indicated to prevent prolonged anoxia caused by direct anterior spinal cord compression or possible compression of the anterior spinal artery.

6. When fracture-dislocation has occurred the "tear-drop fracture" (the name suggested by the anterior inferior surface of the vertebral body "dripping" anteriorly), "diver fracture," or "acute cervical flexion fracture" is common (Cases 3, 4, 5, 6 and 9). The posterior inferior margin of the same vertebral body in each of these cases had become displaced posteriorly into the spinal canal, causing anterior spinal cord compression or destruction. These patients should be placed immediately in skeletal tong traction upon an anterior Stryker or Foster frame. If the lesion is at the level of the phrenic segments C3-C4 (Case 10), or if there is respiratory difficulty, exploration should be deferred until this situation is stabilized. If the injury has occurred below C3-C4 level, decompressive laminectomy should be performed early with the patient in traction on the frame. A carefully inserted intratracheal tube should be used for anesthetization, and laminectomy should include three laminae. The dura mater should be opened in the midline and the arachnoid cut laterally so that the dentate ligaments may be sectioned at two or three levels. Transdural exploration may then be carefully made to exclude a herniated disc. If spinal cord swelling is marked, the dura mater should be left open, and the arachnoid, which has been left intact in the midline, will seal off the cerebrospinal fluid. After a period of 3 weeks in traction, cervical spinal fusion should be performed by wiring together the proximal and distal spinous processes, inserting a carefully shaped, well fitting roof bone graft bilaterally to cover the surface of the cord. Bone chips may then be placed laterally over the roughened facets. After 8 weeks the tongs should be replaced by a Forrester collar with a back brace.

Decompression with removal of three laminae is necessary to permit
sufficient space for section of dentate ligaments at two or three levels and exploration anterior to the cord. A limitation of the laminectomy to two laminae in order to pacify orthopedic colleagues so they need perform a less extensive fusion could readily have cost the life of the patient in Case 6. The posterior surface of the cervical spinal cord appeared grossly normal in this case at operation, but a postmortem examination 5 days later revealed a constriction at the level where the C4 lamina had compressed the posterior surface of the spinal cord. Swelling of the cord from traumatic handling may have resulted in compression, hypoxia and further central cephalad destruction, thus destroying the phrenic segments at the C4 level and causing the patient’s death.

7. Spinal fusion is a necessity in the cases in which there is “tear-drop” or “acute flexion fracture-dislocation” with a partially damaged cord, for even after keeping patients in traction 12 weeks (Case 7) there may be slippage with the possibility of destroying or severely injuring the cord (Cases 3, 4, and 6). Case 4 demonstrated that although the cervical roentgenograms suggest a solid fusion, only a fibrous union with pronounced spinal instability was found at the time of operation 4 years after injury.

8. Some patients may recover some degree of function spontaneously from anterior cervical spinal cord compression (Case 5). However, there is always danger of chronic arthritic spurring at the level of the fracture-dislocation, causing chronic anterior cord compression and resulting in spasticity (Case 5) requiring operation at a later date. The author firmly believes that it is advisable to operate upon these patients early in order to decompress the cord and permit it to ride freely over any bony defect. Also if there is an associated herniated disc present at the time of the acute injury, it is more readily removed and does not become calcified to cause chronic anterior cord compression. Stabilization of the spine by spinal fusion prevents movement and continued irritation to the bone, preventing calcium from being laid down anterior to the cord.

**SUMMARY**

In summary, in acute trauma to the spine there is a syndrome of acute anterior spinal cord injury which is characterized by an immediate complete paralysis with hypesthesia and hypalgesia to the level of the lesion, with preservation of touch, motion, position and part of vibration sense. This syndrome may be caused by acute anterior spinal cord compression or destruction of the anterior portion of the cord. Since a surgical lesion causing spinal cord compression is indistinguishable from a nonsurgical destructive one, these patients should have an exploratory laminectomy and the majority of them should have a spinal fusion secondarily 2 or 3 weeks later. Emphasis should be placed upon the fact that these patients have attained their maximum neurological deficit and they do not present the usual criteria for surgery, namely, progression of neurological signs or evidence of blockage
of the cerebrospinal fluid on the Queckenstedt test. Thirteen cases are presented in detail to illustrate the pathological conditions producing this neurological pattern.

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