VERTEBRAL ARTERY INSUFFICIENCY IN ACUTE AND CHRONIC SPINAL TRAUMA

WITH SPECIAL REFERENCE TO THE SYNDROME OF ACUTE CENTRAL CERVICAL SPINAL CORD INJURY*

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(Received for publication August 5, 1960)

For many years surgeons have been puzzled by the fact that some patients with injuries of the cervical spine have sustained severe neurological deficit without alteration in the alignment of the bony spine. This neurological disability frequently has followed a definite pattern which was described several years ago as the “acute central cervical spinal cord injury syndrome.” It was thought that this syndrome resulted from a certain type of cervical cord “contusion.” However, careful neurological evaluation of subsequent cases has suggested that this syndrome also may be caused by a second mechanism, namely, one of “partial” or “relative” insufficiency of the vertebral artery to the cervical spinal cord.

In this paper 4 cases are presented to demonstrate the mechanisms involved and to support the evolution of these concepts.

CONTUSION

In 1948 Taylor and Blackwood discussed paraplegia in patients with hyperextension injuries of the cervical spine with normal radiographic appearances. Three years later Taylor demonstrated conclusively by myelography in cadavers with cervical spondylolisthesis that there was simultaneous encroachment by the hypertrophic spur anteriorly on the cord as well as impingement posteriorly by the wrinkled ligamentum flavum in hyperextension. This resulted in severe compression or “squeezing” of the cervical spinal cord. Schneider et al. reported that when this occurred the center of the cervical cord received the major damage resulting in a group of symptoms designated as the “acute central cervical spinal cord injury syndrome.”

The syndrome “is characterized by disproportionately more motor impairment of the upper than of the lower extremities, bladder dysfunction, usually urinary retention, and varying degrees of sensory loss below the level of the lesion. If the findings are caused by central cord destruction with bleeding, hematomyelia, there may be caudal or cephalad extension of the lesion with further progression of symptoms, perhaps culminating in complete tetraplegia or death. But if the symptoms are caused by concussion or contusion, with an edematous type of central cord involvement, there may be gradual return of function in a definite sequence. The amount of recovery depends upon the degree of edema present compared to the extent of hematomyelia (Fig. 1). The lower extremities tend to recover motor power first, bladder function returns next, and finally strength in the upper extremities reappears, with the finer finger movements coming back last.† The varying degrees of sensory impairment do not follow any set recovery pattern.”

This syndrome was first described in 1954 with a presentation of 9 cases. Four years later another report of an additional 12 cases

† Italics added.
was published.\textsuperscript{20} One of these (Case 9\textsuperscript{17}) presented the first pathological proof in support of the proposed syndrome. Fig. 2 shows the cervical segment of the spinal cord of a 72-year-old man with cervical hypertrophic arthritis who had sustained an hyperextension injury with the pattern of acute central cord injury and had followed the classical pattern but with only partial recovery. He died of atelectasis and pneumonia 1 month after injury.\textsuperscript{20}

The explanation of the clinical pattern still seemed satisfactory from a motor standpoint, but puzzling facts remained unexplained in connection with the sensory findings. \textit{In some of the patients showing the syndrome there was an immediate complete loss of all sensation, while in others there was little or no alteration of the sensory pattern.}

There are some patients with cervical hypertrophic arthritis who sustain “deceleration hyperextension cervical spine injuries” (the authors have discarded the term “whiplash”) with some degree of transient neurological disability. This is caused by oscillation of the head forward and backward without any apparent fracture or dislocation of the hypertrophic cervical spine. It may be associated with marked transient motor impairment and only a minimal degree of sensory loss, which is confined primarily to impairment of the cervical sensory root. This is illustrated by the following case.

\textit{Case 1.} J.S., a 58-year-old man, was admitted to the hospital on Aug. 16, 1939, following an accident in which his slowly moving car was struck from the rear by another vehicle. The patient did not strike his head nor lose consciousness, remembered the sound of the crash, and could describe accurately the following course of events. Immediately after the crash, he had “numbness” of
the entire trunk and extremities and was unable to move either his arms or legs. His car continued to roll forward toward a tree. He called to his wife who was sitting next to him to apply the brake, thus bringing the vehicle to a halt. While sitting in the car and awaiting the ambulance, he eventually was able to move his toes of the left lower extremity, and after a few moments, regained movement in his right ones. A short time later he was able to move the upper arm bilaterally and finally could use the forearm. It was not until he was on his way to the hospital room that he was able to extend and flex his fingers.

On admission to the Emergency Room, there was diffuse cervical tenderness at C5 and C6 spinous processes and gentle downward thrust upon the head caused pain in that region. There was limitation in movement of the head and neck. The patient’s biceps and triceps reflexes were equal and active. His grip was weak bilaterally, but strength was good in all other movements. There was hypalgesia in the right C8 dermatome and equivocal hypalgesia in the C6 dermatome bilaterally.

The remainder of his sensory findings were normal in all modalities. He had slight ataxia on the left heel-to-knee test.

Roentgenogram of the cervical spine showed a narrowing of all the interspaces between the level of C4 and T1 vertebrae with degenerative hypertrophic arthritic spurs on all the bodies. There was no evidence of a fracture or dislocation.

A diagnosis was made of severe hyperextension of the arthritic cervical spine with acute central cervical spinal cord contusion, C6 spinal nerve root contusion, and cervical muscular sprain.

The prognosis for recovery was stated to be excellent. This proved to be the case, since on examination 4 months after his injury his neurological status was normal and he complained only of slight “heaviness” in the left arm.

Comment. This patient exhibited the syndrome of acute central cervical cord injury, which at that time was believed to be caused by contusion of the spinal cord in a patient with cervical spondylosis by the mechanism described by Taylor. It should be emphasized that the only sensory change was confined to isolated dermatomes of the cervical root and there was no abnormality in the long-tract sensory modalities of the spinal cord. These observations suggested that there must be some other mechanism responsible for this syndrome rather than a contusion to or a “squeezing” of the cervical spinal cord. Otherwise one would anticipate much more severe injury to the sensory fibers since they lie nearer the periphery of the spinal cord than the more extensively involved motor fibers. A study of the vascular supply of the spinal cord and some clinical material now has provided what appears to be an explanation for this variability in the sensory findings.

VASCULAR INSUFFICIENCY

The literature treating the problems of vascular insufficiency to the brain is increasing rapidly, but that related to the spinal cord has received scant attention. Schneider and Crosby reviewed the latter topic recently and presented 5 cases explaining some of the neurological symptoms and signs that are observed in vascular insufficiency to the spinal cord in trauma. A case from a previous publication by the senior author had aroused an interest in this topic. A 49-year-old man who had sustained an injury with severe hyperextension of the cervical spine and immediate complete paralysis, died 3 days later.

Autopsy disclosed a severe crushing destruction of the cervical cord at the site of the fracture-dislocation. There were areas of grossly appearing normal cord several segments above and below the site of injury (Fig. 3). The lowermost point of destruction was fully 6 neural segments below the site of impact and major damage to the cord (Fig. 4).

In the report by Schneider and Crosby (Case 4) it was stated: “It had been impossible to explain the peculiar phenomenon of a point of destruction at C4-C5 interspace followed by relatively less damaged caudal sections of the cord, only to find a much more marked area of central destruction some 6 neural segments distal to the site of injury.”

A brief review of the normal circulation of the cervical segments of the spinal cord is essential for an understanding of this pattern. Adamkiewicz, Bolton, Frazier, Herren and Alexander, Kadyi, Suh and Alexander, Yoss, and Zülch, have contributed greatly to the study of the anatomy of this circulation. In their textbooks, Bing and Mettler have ably presented a condensation of these authors’ works.
Normally the vertebral arteries ascend in the neck posterior to the common carotid arteries entering the 6th intervertebral foramina. They curve about the massa lateralis atlantis, perforating the dura mater and entering the skull at the lateral margins of the foramen magnum (Fig. 5). After a short course intracranially, they fuse to form the basilar artery. A branch is given off from each vertebral artery prebasilarly and these vessels extend a short distance caudally prior to forming the anterior spinal artery on the anterior surface of the spinal cord, the vessel narrowing somewhat near the upper thoracic segments (T4). In a comprehensive piece of work, Zülch has demonstrated the general vascular supply to the spinal cord, emphasized the direction of blood flow, and indicated that the two zones of poor, or almost compromised, collateral circulation are in the anterior portion of the cord at the T4 and L1 segments, respectively (Fig. 6). It is this zone of impaired collateral circulation that is demonstrated in the pathological specimen.

When the circulation of the spinal cord is examined in the sagittal plane, there is an arterial branch extending upward into the sulcus from the anterior spinal artery which supplies the anterior and lateral columns of the cord. This vessel has been designated as the anterior sulcal artery by Herren and Alexander. At any segment, the vessel

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**Fig. 4.** Multiple transverse sections of spinal cord with the uppermost one at the top of the specimen and the lowermost fully 6 neural segments below the site of constriction. Destruction of central cord may be seen throughout entire specimen. (Reproduced with modification from Schneider et al.)

**Fig. 5.** Lateral view of dislocated atlas on axis showing points of compression of the vertebral artery at (1) the C1 intervertebral foramen and (2) the point where the occipital condyle slides over the groove in the C1 lamina. (Reproduced from Schneider and Crosby.)
Fig. 6. Diagram showing origins of anterior spinal artery from vertebral artery bilaterally. The anterior spinal artery supplies the anterior two-thirds of the cervical spinal cord and the uppermost thoracic segments to the T4 level. The direction of flow in this vessel is caudally. The region of the T4 neural segment is one of poor collateral supply, so that hypoxia will present the possibility of infarction here in the distribution of the anterior sulcal arteries with central cord destruction.

The remainder of the arterial supply for the thoracic cord from T4 to L1 is by intercostal branches from the aorta. The major one, the great ventral radicular artery, enters at T9 segment in this diagram. There is another zone of poor collateral supply at L1 dermatome. (Reproduced from Bing. Courtesy of W. Haymaker.)

supplies only one-half of the cord. Bolton has indicated that the posterior spinal arteries supply the posterior columns and the more posterior portions of the gray columns. “Segmentally, arteries are given off from the intercostal vessels or lateral spinal arteries which accompany the posterior and anterior nerve roots, respectively. These unite with the posterior and anterior spinal arteries directly and then are joined together segmentally along the periphery of the spinal cord as the arteriae coronae. . . . From these arteriae coronae, branches extend inward along the periphery of the cord supplying the posterolateral and anterolateral portions of the cord.” Upon this sagittal section of the spinal cord, the authors have superimposed various fiber tracts which are pictured in Fig. 1.

It may then be seen that the anterior sulcal artery supplies the anteromedial gray matter, the rubrosegmentospinal tract, and the more medial portion of the lateral corticospinal tract including the hand and arm areas (Fig. 7). The leg area of the lateral corticospinal tract receives its blood through the posterior funicular branch from the posterior spinal artery or arteriae coronae. The region of the lateral spinothalamic tract on the outer portion of the spinal cord is supplied by the small branches of the arteriae coronae from the periphery of the spinal cord. These are the factors that are so important in the anatomical, pathological, and clinical relationships.

The studies of Ford, Ford and Clark, Hutchinson and Yates, and Radner have done much to emphasize the importance of the vertebral artery in trauma and disease.

It is the authors’ belief that a transient or permanent impairment of the vertebral blood supply to the brain stem or cervical cord may be the cause of some fatal cases of acute cranioencephalic injury which, at autopsy, present no apparent pathological cause for death.

Schneider and Crosby indicated that the vertebral arteries might be compressed simultaneously at three main sites: a) Any fracture-dislocation of the cervical spine above the C6 intervertebral foramen may cause compression of the vessels at the displaced intervertebral foramina. b) Atlantoaxial dislocation will cause compression at the C1 intervertebral foramina. c) Atlantoaxial dislocation may cause compression at the point where the occipital condyle slides forward over the well indented groove in the lamina of the first cervical vertebra. With bilateral spasm or compression of the vertebral artery, a relative insufficiency may occur intracranially in the basilar artery and its branches as well as cervically in the anterior spinal artery.

In 1933 de Kleyn and Versteegh, using autopsy material, showed that when the head
was twisted backward and then in a contralateral direction, the circulation could be occluded in one vertebral artery. They indicated that normal people were usually asymptomatic because of adequate collateral circulation from the contralateral side. The variability in the size of the vertebral vessels is suggested by the injections of the aortic arch in a cadaver so that both arteries are filled simultaneously with the same degree of pressure. If the major vessel became occluded or was thrown into spasm, a relative or complete vascular insufficiency could occur to the cerebral area supplied by the impaired vessel. Tatlow and Bammer injected radio-opaque material into cadavers and determined that the site of partial obstruction or narrowing of flow in the vertebral artery was at the level of the atlas and axis when the head was rotated toward the contralateral side. They noted coincidental occlusion of the contralateral vertebral vessel at the C5-C6 intervertebral foramen by a hypertrophic spur. (This finding is probably of major significance and will be discussed later with regard to Case 4.)

The following case report focuses attention on this problem.

Case 2. On Nov. 22, 1959, at 1:30 p.m., J.H., a sturdy 19-year-old football player, raced down the field on the opening kick-off and charged into the opposing ball carrier striking him with great force, causing his opponent to lose the ball. The mechanism of injury is shown in a print from the documentary motion pictures of the game (Fig. 8). The patient was not rendered unconscious. He recalled that he struck the other player hard and felt his head and shoulders were thrown backward forcibly as he fell to the ground, “numb all over” and paralyzed in all four extremities. He remembered that he could first move his toes of the left foot and a few minutes later, those of the right foot. The game was interrupted for 10 minutes while he was being examined on the field. He was then carried to the locker room where the team physician noted at 1:50 p.m. that the man was able to move his lower extremities well, but he had some weakness in the arms and profound weakness in the grip of both hands. The patient recalled that he had no numbness over his body, but rather a severe burning pain over the outer aspect of both shoulders and along the inner border of his left upper arm.

At the hospital roentgenograms of the skull and cervical spine revealed no fracture or dislocation. At 4:30 p.m. one of the authors examined the patient and found no abnormality of the cranial nerves, with hyperesthesia over the C5 dermatome bilaterally and the left T1 dermatome. Otherwise, sensation was normal in all modalities. The grip definitely was weak bilaterally. The biceps reflexes were depressed symmetrically, but the remainder of the deep reflexes were equal and active. He had no pathological reflexes. There was
little or no tenderness over the cervical spine.

By 8 a.m. on the following morning, the strength of grip in both hands had returned almost completely and, with the exception of the hyperesthesia described above, his neurological status was normal. He had begun to have increasing pain and stiffness in his neck. Lumbar puncture showed normal pressure, cell count, and protein determinations. He was discharged from the hospital on the 5th day after injury with some slight residual stiffness of the neck. His recovery was complete within 5 days of injury.

Comment. Roentgenograms of the patient's cervical spine showed neither hypertrophic arthritis nor a fracture-dislocation. He exhibited the syndrome of an acute central cervical spinal cord injury without signs of long-tract sensory impairment, probably on the basis of vascular insufficiency. This might have been caused by direct trauma to the anterior spinal artery with spasm, or, as was more than likely, by primary compression of the vertebral arteries at the point where the occipital condyle slides forward over the well indented groove in the lamina of the 1st cervical vertebra. Indirectly this would result in a relative insufficiency in the blood flow through the anterior spinal artery.

The mechanism in this case is one of acute severe hyperextension of the cervical spine as shown in Fig. 8. The patient's headgear was held firmly in place by the chin strap and his solid plastic face guard was caught on the buttocks of one of the opposing players so that the patient's head was thrown backward forcibly.\(^\text{19}\)

The minimal sensory findings consisting of hyperesthesia exhibited by this patient were caused by compression or "squeezing" of the nerve roots at the C5-C6 and C7-T1 interspaces. These roots were at either end of the arch formed when the cervical spine was forced into hyperextension. There were no sensory signs indicative of direct contusion to any of the long sensory tracts of the cervical spinal cord. The patient has had a complete neurological recovery.

Case 3. R.S., a 15-year-old school boy, was admitted to the hospital on July 18, 1959, after having sustained an injury of the neck when he dove from a dock into two feet of water at 7 p.m. on the previous evening. He did not recall striking his head on the bottom, but he remembered being unable to move all four extremities. A friend pulled him from the water and rushed him to the local community hospital.
He had pain in both shoulders. There was some numbness and tingling in both arms and legs and he was unable to void. There was slight tenderness over the cervical spine. He had fair strength in both lower extremities with considerably less power in his upper ones. His sensations of vibration, position and light touch were preserved in the lower extremities with bilateral diminution of pain from L1 to L5 dermatomes and a light sensory level at T4 and T5 bilaterally. The intercostal muscles were functioning well. The biceps reflex was absent bilaterally, but the remaining deep reflexes in the upper and lower extremities were symmetrically hypoactive with no pathological reflexes. His temperature was slightly elevated but his vital signs were normal.

Roentgenograms of the cervical spine exhibited a dislocation of the C3 vertebral body anteriorly on a compression fracture of the C4 vertebral body. There were also a complete fracture of the base of the C2 spinous process, incomplete fractures of the C3 spinous process, and a fracture through the superior articular process of C4. A diagnosis of acute central cervical spinal cord injury was made and the prognosis was stated to be good for recovery.

Right vertebral arteriography was performed prior to skeletal traction being applied. It demonstrated some degree of spasm or compression of the vessel (Fig. 9). Re-expansion of the vessel occurred after the application of 20 pounds of traction (Fig. 10). Following the third injection of approximately 5 cc. of 35 per cent Hypaque solution, the patient lost consciousness, sustained a period of apnea and had fixed dilated pupils for a few minutes. After artificial respirations, the pa-
tient began to breathe and within another 5 minutes responded to his name. His pupils began to react. His neurological status remained unchanged for about 3 or 4 days. Re-check roentgenograms, 48 hours after application of traction, showed reduction of the fracture-dislocation and the weight was decreased to 10 pounds. Lumbar puncture 6 days after admission to the hospital showed no evidence of block on the jugular compression test and normal cerebrospinal fluid test.

Examination on Aug. 6, 1959 revealed a level of hypalgesia from C6 through T4 dermatomes and L1 through S5 dermatomes on the left side. The motor power was good in all movements of the extremities except for bilateral weakness in the grip of both hands. On Aug. 20, 1959, Dr. Robert Bailey of the Bone and Joint Department performed an anterior cervical vertebral body fusion of the C2, C3, and C4 vertebral bodies using an autogenous iliac bone graft and his recovery was uneventful. When seen on Nov. 24, 1959 he had normal neurological findings except for minimal weakness in the grip of the right hand, which was showing progressive improvement.

Comment. The diagnosis was made of an acute central cervical spinal cord injury associated with C3-C4 fracture-dislocation of the spine, and the prognosis for recovery was regarded as good. The patient was the first one in our series who had sustained cervical trauma and upon whom vertebral arteriography was performed. He was selected for vertebral arteriography because he had sustained an incomplete lesion suggesting the syndrome of an acute central cervical spinal cord injury, had intact intercostal muscles and diaphragms (in spite of a C3-C4 lesion), and seemed young enough to be a good risk for such a study. In spite of his excellent physical condition and the fact that only small increments of dye were used for the direct vertebral arteriography, he sustained some degree of vascular insufficiency with hypoxia to the brain stem resulting in apnea and fixed dilated pupils. This occurred during the third vertebral injection when the traction was applied. Actually one would have anticipated that this individual would have had improved vertebral blood supply to the cord and brain stem with traction as demonstrated by the arteriogram. One must assume that repeated manipulation of the vessels threw them into spasm or a small embolus was accidentally injected into the circulation. Suekhting and French21 have reported an interesting case of fracture-dislocation of the cervical spine which demonstrated a posterior inferior cerebellar artery syndrome produced by cervical traction. Following removal of the traction, the patient’s abnormal neurological pattern began to recede within 3 days.

It is doubtful that an older and less vigorous individual with complete neurological deficit would have survived such an episode. This experience may serve as a warning of the dangers in attempting such a procedure in this type of patient. Fortunately, there was no residual neurological deficit attributable to this episode. If any further arteriographic studies are undertaken in such patients the indirect technique of catheterization of the brachial artery to demonstrate the vertebral artery should be employed as a less hazardous approach.

This study suggested the importance of the early institution of traction for reduction of cervical fracture-dislocations, thereby possibly increasing the vascular supply of the vertebral artery to the spinal cord and brain stem. It has also raised the question of the desirability of improving the blood flow to the brain and spinal cord by the use of therapeutic agents such as the vasodilator group.

Thus far the discussion has dealt primarily with acute injuries of the cervical spinal cord. The last case to be presented is one that represents the chronic sequelae of acute trauma of the cervical spine.

Case 4. In May, 1956, C.D., a 62-year-old man, fell 25 feet from a ladder, striking the ground on his chest, forehead, and chin. He was unconscious for 6 hours. Upon regaining consciousness in the local hospital he found his left arm was flexed and lying on his chest. Roentgenograms of his skull and cervical spine showed no evidence of fracture or fracture-dislocation. His neurological status at that period is unknown but he was in good enough condition so that he could get up and about the ward within a few days.

Since he was suffering from moderately severe pain in the shoulders, he was placed in a cervical collar for about 6 weeks. After removal of this support, he began to have spells which were preceded by a burning sensation radiating up the
Fig. 11 and 12. Case 4. (Left) Right vertebral arteriogram shows compression of vertebral artery laterally at C5-C6 level. (Right) Left vertebral artery shows a normal course through the intervertebral foramina.

back of his neck into the head. This would then be followed by complete loss of vision, difficulty in hearing, inability to talk, vertigo and marked weakness of the left arm and leg with less involvement of the right upper and lower extremities. He also was unable to chew and swallow during these episodes. The attack lasted from 10 to 30 minutes. It was precipitated by hyperextending the neck and was relieved by flexion. When he fainted or lost consciousness during the spells, he volunteered that on recovery his legs began to regain motor power first, and that the arms would have return of strength next. He did not lose control of bladder or bowel during the attack.

After multiple admissions to various hospitals, he was seen at the U. S. Veterans Hospital, Ann Arbor, Michigan on Feb. 29, 1960, because he had fallen during one of his spells and had a laceration of the cervical region.

On examination his vital signs were normal. He had hypesthesia in the distribution of the left 5th nerve and diminished hearing bilaterally. The remainder of his cranial nerves and sensory and reflex findings were normal. There was minimal weakness in his left upper extremity in all movements. He had no pathological reflexes.

Roentgenograms of the skull were normal. Films of the cervical spine showed a hypertrophic arthritic spur at C5-C6 interspace. His electroencephalogram revealed a border line arrhythmia with no evidence of a focal lesion. His serology and cerebrospinal fluid were normal. Roentgenograms of the chest demonstrated moderately severe bilateral pulmonary emphysema with some chronic fibrotic changes at the right base. One could see on the lateral myelographic view minimal anterior defects in the column at C5 and C6 with some posterior defects caused by the wrinkled ligamentum flavum. On the anterior myelographic view there was good filling of all the interspaces, showing no nerve-root obliteration.

Carotid arteriograms were normal. The right vertebral arteriogram visualized by the technique of indirect catheterization showed marked displacement laterally by a bony spur at C5-C6 interspace with some degree of attenuation of this vessel distal to this point (Figs. 11 and 12). The left vertebral arteriogram was normal. These vessels showed no abnormality both at the site of origin from the aortic arch and in their intracranial distribution to suggest any intrinsic vascular disease either in the chest or in the intracranial distribution.

Comment. There is no doubt that this man had a severe acute hyperextension injury to his hypertrophic arthritic cervical spine 3½ years prior to his present hospital admission. He had sustained lacerations of his forehead and chin as well as a contusion to his chest causing severe pain in the chest. The hypertrophic cervical spurs may have been of
traumatic origin with fractures and hyperostotic healing or perhaps have been caused by the accentuation of the old arthritic process. The symptoms of insufficiency of the vertebral artery were clear-cut and suggested that the vertebral arterial compression as demonstrated by vertebral arteriography may have been of some importance. It is possible that in this patient the pulmonary emphysema with decreased vital capacity causing some degree of hypoxia may have contributed to his problem of cerebral vascular insufficiency. The patient was still in the hospital for further studies and treatment. Surgical decompression of the vertebral artery by removal of the hypertrophic spur had been suggested but the patient refused. Cervical spinal fusion had been considered to eliminate movement and compression of the vertebral artery by the spur at the involved interspace.

DISCUSSION

In traumatic injuries of the cervical spine the syndrome of acute central cervical spinal cord injury is characterized by the presence of better strength in the lower than the upper extremities with varying degrees of associated sensory loss.

This syndrome may be caused by a "squeezing" or contusion of the spinal cord with marked sensory loss as well as motor impairment. The same neurological pattern with little or no long-tract sensory impairment suggests the possibility of insufficiency of the vertebral artery (Cases 1, 2 and 4). In some cases it will be impossible to determine whether the lesion is caused by contusion or vascular insufficiency. In Case 3 the latter was probably the important factor.

In the past authors have thought too frequently in terms of complete occlusion of the vessel rather than in terms of degrees of vascular insufficiency. Because of the location of the vertebral artery and the peculiarity of the distribution of its branches, particularly to the cervical spinal cord, more consideration must be given to the condition of a "relative" rather than "absolute" pattern of vascular insufficiency. It is a thing that may not be demonstrated readily by a simple test such as vertebral arteriography but the recognition of the clinical signs will have to suffice at this time in making the diagnosis.

Perhaps more vigorous efforts should be directed toward the early reduction of the fracture-dislocation of the cervical spine when it is present, not so much to overcome compression of the cord alone but also in an attempt to increase its vascular supply.

Finally, it must be emphasized that this paper is really only a preliminary report. From the above studies numerous implications may be drawn. Lewis and Coburn11 have alluded to the interference with the circulation of the vertebral artery in the reduction of pain in the head. Grinker and Guy9 have called attention to a case of cervical sprain causing thrombosis of the anterior spinal artery. Brain et al.5 have noted the occurrence of arthropathy with syringomyelia. It becomes obvious that more clinical and pathological studies will have to be done to understand the clinical pattern of lesions in this area. If one reviews the symptoms and pathological changes presented by the patient with the syndrome of acute central cervical spinal cord injury, one wonders whether the presence of a compression of the vertebral artery by a hypertrophic arthritic spur (as noted in Case 4) may not be responsible for some cases of syringomyelia. If compression of the vertebral artery causes very gradual decrease of the blood flow, there may be merely gradual destruction of the spinal cord from atrophy. If it is more acute with infarction then liquefaction and formation of syrinx might occur. Obviously not all cases of syringomyelia will occur in this way, but it may be that a few may have this etiology. Further studies are being performed along these lines.

SUMMARY

In traumatic cervical injuries the acute central cervical spinal cord injury is characterized by more motor power in the lower extremities than in the upper ones with a varying degree of associated sensory loss.
The lesion may be caused by: 1) direct contusion to the cervical cord with permanent or transient damage to the posterior columns and the lateral spinothalamic tract with some degree of sensory loss or 2) vascular insufficiency because of indirect damage secondary to compression of the vertebral artery resulting in a relative hypoxia to the central motor pathways in the cervical spinal cord with little or no sensory impairment. It should be emphasized that a relative hypoxia may result physiologically, a thing which may not be demonstrable grossly in life or at autopsy.

The main sites of compression of the vertebral artery may be: 1) in the intervertebral foramina when cervical dislocation occurs above the C5-C6 interspace; 2) in the atlanto-axial dislocation at the C1 intervertebral foramina; 3) at the occipital-atlantal junction at the point where the occipital condyle slides forward over the groove in the lamina of the first vertebra.

A certain amount of vertebral vascular insufficiency may be present without any evidence of blockage of the spinal canal on the jugular vein compression test. Therefore, early reduction of the fracture-dislocation may be desirable to correct any impairment of vertebral arterial supply to the injured cord even though there is no definite compression of the cervical spinal cord demonstrated.

Vertebral arteriography in cases of severe injuries of the cervical cord with, and probably without, fracture-dislocation is not without considerable risk. If it is undertaken, the surgeon must be prepared to accept the possibility of further neurological disability or possibly even death.

The possibility of compression of the vertebral artery by spurs in hypertrophic cervical arthritis has been suggested as an infrequent cause of syringomyelia. Further studies are being undertaken to support or disprove this thesis.

Cervical spinal fusion has been considered to eliminate movement and compression of the vertebral artery by the spur at the involved interspace.

ADDENDUM

Since forwarding this paper to the Editors the authors have seen 2 more patients who have demonstrated the syndrome of acute central cervical spinal cord injury without long-tract sensory impairment which is believed to be on the basis of insufficiency of the vertebral artery.

The authors wish to express their appreciation to Dr. James C. White for discussing this paper and to Dr. Lyle A. French for preparing his comments on this material, presented at the meeting of the Harvey Cushing Society.

REFERENCES


19. Schneider, R. C., Reifel, E., Chisler, H. O., Oosterbaan, B. G., and Elliott, C. W. Serious and fatal football injuries involving the head and spinal cord. With special reference to the mechanisms of injury and alteration of equipment for their prevention. (To be published)


**DISCUSSION**

Dr. James C. White: We are deeply indebted to Dr. Schneider for calling attention to this syndrome of acute central necrosis of the spinal cord. His original description in 1944 with Drs. Cherry and Pantek was such a classic, vivid description that we have been on the lookout for confirmatory cases ever since. It just so happens we have two that are striking examples of this at the Massachusetts General Hospital at this minute. Both patients are in their sixties, with advanced arthritic spurring at cervical vertebrae. Each had hyperextension injury without fracture or demonstrable dislocation.

The first had complete anesthesia below C6 when she came in, plus paralysis, but retained deep tendon reflexes in her legs. Posterior-column sensation and voluntary movement in legs are now recovering, together with sensibility of pain and temperature.

The second had paralysis to the arms through C6 on one side (biceps) and C7 (triceps) on the opposite side. Sensations of touch and position were present throughout with a mild Brown-Séquard loss of pain-temperature sense on the side of the stronger leg.

Now, at 5 months and 24 months after injury, both show the predicted type of recovery—motor return is nearly complete in the legs; bladder and rectal recovery came next. They have good return of strength in one arm each, the fingers recovering last, and upper-arm recovery on the opposite side.

The sensory examination hasn’t been emphasized too much. The posterior columns show minimum residual injury. One had none to start with. The other’s ventrolateral columns have a mild Brown-Séquard impairment of pain and temperature on the side in which motor recovery is most nearly complete.

[A lantern slide was shown illustrating the topographical arrangement of axons in the spinothalamic tracts in the cervical region.] With central necrosis of the lower cervical cord, sensibility to pain and temperature also should be impaired primarily in the arms, but this has not been the case in these two individuals. This is the only discrepancy that I have been able to find in connection with Dr. Schneider’s beautiful observations.