Vascular insufficiency and differential distortion of brain and cord caused by cervicomedullary football injuries

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The problem of serious and fatal football injuries involving the cervicomedullary junction is presented. The mechanisms of such injuries ("stick blocking," "spearing") have been analyzed with the help of motion pictures of the action. The second cervical segment of the spinal cord was the commonest site of pathological change. A poor collateral vascular supply to the vertebrobasilar region, compression of the venous sinus, and the great disparity between the distortion of the freely mobile brain and the relatively fixed cervical spinal cord were implicated as the most likely background for the damage, which in turn may lead to acute cerebral edema and tentorial or tonsillar herniation.

TRAUMATIC lesions of the central nervous system are usually discussed under the separate headings of cranial or spinal injuries, with few reports appearing in the literature pertaining to the region of the cervicomedullary junction. This paper is concerned with serious or fatal football injuries due to vascular insufficiency to the brain stem and spinal cord and/or to the dynamic sequelae related to the differential distortion between the brain and spinal cord at the time of direct vertex impact.

The football field is one of the few places where the actual mechanisms of trauma to the central nervous system may be witnessed and later studied on the television recordings or the movie film. Several surgeons have collaborated to present the case histories of football players who have sustained cervicomedullary injuries of varying degrees of severity. In some cases documentation was possible of how the injury was incurred; in others, the evolution of the neurological pattern and the findings at postmortem examination were recorded.

No single neurosurgeon is likely to encounter these cases frequently, but by pooling experiences this group has provided a more complete understanding of the problems involved in such injuries.

Vertebrobasilar Vascular Insufficiency

Hyperextension Injuries Without Cervical Fracture Dislocation

After the vertebral arteries emerge from the foramina transversaria at the first cervical level, they cross the laminae of the atlas to enter the foramen magnum. At this site they are particularly vulnerable and may be
contused by the occipital condyles of the skull. This may lead to spasm or an ascending thrombosis extending as high as the basilar artery (Fig. 1). Such an injury with transient vasospasm might cause only a few minutes of neurological deficit.

The first two cases represent severe types of depletion of vertebral blood flow.

Case 1 (Acute Hyperextension Injury Without Cervical Fracture Dislocation). In a college game the tackler's face mask became caught against the ball carrier's buttocks, throwing his head and neck into acute hyperextension (Fig. 2). He was not unconscious, but except for his feet, had an immediate complete motor loss in all four extremities with preservation of all sensory modalities. A diagnosis of acute central cervical spinal cord injury secondary to vertebral, anterior spinal, and anterior sulcal (or central) artery insufficiency was made. His recovery, which was complete, followed the typical pattern of patients exhibiting this syndrome, with the legs regaining movement first, followed by the trunk and arms, with hand motion returning last. There was no fracture dislocation of the cervical spine. Vascular insufficiency in the upper cervical spinal cord was suspected.

Case 2 (Acute Hyperextension Injury Without Cervical Fracture Dislocation). This college football player was participating in light contact drill without full equipment; he was blocking using his right shoulder against the midsection of the opponent's body. The play had ended and he was relaxed when two other men struck him from the opposite side, throwing his head into full extension. His respirations ceased but he was fully conscious when he struck the ground. Mouth to mouth resuscitation was immediately instituted, and he was taken to a local hospital where a tracheostomy was performed. At that time he was found to have a complete tetraplegia with a sensory level at the C-2 dermatome, a condition which remained unchanged until he died 5 weeks later from an overwhelming pneumonia. In spite of intensive x-ray examinations there was no evidence of bone disruption of the cervical spine.

At autopsy a neurosurgeon performed a complete examination that included the skull, brain, and cord; on laminectomy of

![Fig. 1. Left: The vertebral arteries thread through the foramina transversaria, passing over the flattened lamina of the atlas beneath the occipital condyles, and cephalad into the foramen magnum to form the basilar artery. Prebasilar branches are given off which unite to form the anterior spinal artery. Right: With hyperextension of the cervical spine, and without fracture or dislocation, there is compression of the vertebral arteries between the occipital condyles and the flattened first cervical vertebral laminae.](image)
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Fig. 2. Case 1. An excerpt from the motion pictures shows the tackler (arrow A) has spun around, his head thrown into hypertension by the face guard caught against one of the opposing players. His arms are dropped at his sides and his legs are extended limply. Note approximation of the white stripes on the helmet to the white numerals on the tackler's back indicating the degree of cervical hyperextension. The ball (arrow X) lies free after the fumble. (Courtesy of J. Neurosurg., 1961, 18:348-360. Schneider, R. C., and Schemm, G. W. Vertebral artery insufficiency in acute and chronic spinal trauma.)

the cervical spine he found no instability. Although it had been over a month since his injury there was no evidence of any tear of the ligamentous structures or evidence of any resolving hematoma. The odontoid process was in normal relationship to the foramen magnum. The vertebral arteries were dissected at the C-1 and C-2 vertebral level, and both appeared grossly and histologically normal. A well-defined loculated cystic cavity was demonstrated at the C-2 level of the spinal cord (Fig. 3) where it was associated with a complete interruption of the cord.

This patient was one of two football players who sustained a serious cervicospinal injury at the same college during the same season. The second player lived but was totally tetraplegic.

Fig. 3. Case 2. This autopsy specimen of the cerebellum, midbrain, and cervical spinal cord shows the cavitating lesion at the C-2 spinal cord segment (arrows). There was no other gross visible lesion.
Case 3 (Subacute or Delayed Hyperextension Injury Without Fracture Dislocation).
A 14-year-old boy was injured in a football accident on October 5, 1968. Although he was unconscious for a short time, he walked home following his recovery. He consulted an orthopedic surgeon because of a left clavicular fracture. That evening at dinner he had two vomiting episodes. On the following day the patient was found unresponsive and tetraplegic. He was taken to the hospital where his temperature rose to 107°F. A tracheotomy was performed because of an apneic episode. Subsequently he was transferred to a second hospital where a lumbar puncture, bilateral carotid angiography, and spine and skull x-rays were found to be within normal limits.

When examined the patient was mute but had an alert and very apprehensive appearance. He was unable to follow commands. Some spontaneous weak and nonpurposeful movements were noted in his arms. He had a left Horners' syndrome, and could not protrude the tongue. There was an incomplete tetraparesis, with the legs spastic and the arms flaccid. He had a complete sensory loss of all modalities below the C-6 dermatome bilaterally. There were hyperactive patellar, Achilles, and biceps reflexes with absent triceps reflexes bilaterally. Extensor plantar reflexes were noted on both sides, but no pyramidal tract signs in the arms were found. The abdominal reflexes were absent. The patient was placed on a Stryker frame with indwelling catheter drainage and given prophylactic antibiotic therapy. Skeletal cervical traction was instituted. The state of consciousness gradually improved from complete lethargy to a more alert condition but he remained mute throughout the entire period of hospitalization. Myelography failed to reveal an obstruction of the subarachnoid space throughout the cervical spine or at the level of the foramen magnum. On October 18, 1968, a right retrograde brachial cerebral angiogram demonstrated obstruction of the right vertebral artery near the foramen magnum (Fig. 4 left). On October 22, 1968, a left retrograde brachial arteriogram showed a thrombosis of the left vertebral artery at the same level (Fig. 4 right). On October 25, 1968, a left carotid arteriogram demonstrated good filling bilaterally of the posterior cerebral arteries but no flow was seen in the basilar artery. Throughout his hospital stay the patient had intensive physical therapy. A gastrostomy was performed.

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Fig. 4. Case 3. Left: A right retrograde brachial arteriogram revealed obstruction of the right vertebral artery at the foramen magnum (arrow) on October 18, 1968. The right carotid artery filled intracranially. Right: Four days later, on October 22, 1968, a left retrograde brachial arteriogram demonstrated thrombosis of the left vertebral artery (arrow) at the level of the foramen magnum on the lateral view.
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When the patient was transferred to a rehabilitation institute 3 months later he was still mute; he had however weak grasp with the right hand, flexion of the forearm, and weak extension of both legs. Gradually he developed control of his tongue and a gag reflex. The patient's grasp with the right hand improved slowly so that he could work an electric typewriter, but his left hand was not really functional. The generalized spasticity in his extremities seemed to be increasing when last examined.

Hyperextension With Atlanto-Axial Dislocation

Because of their confined course in the foramina of the cervical spine, the vertebral arteries may be acutely or gradually compressed when dislocation or slippage of the vertebral bodies occurs, causing inadequate blood supply to the brain stem.

Case 4 (Acute Hypertension Injury With Atlanto-Axial Dislocation).* A professional football player while making a tackle sustained two successive blows from the knees of a hard-charging ball carrier, which struck the tackler's head forcing the neck into hyperextension (Fig. 5). He walked to the locker room where he became belligerent, disoriented, and developed a decerebrate pattern with dilated fixed pupils. He succumbed within 1½ hours. At autopsy he had marked cerebral hyperemia and edema with an atlanto-axial dislocation; the cord was almost completely severed at the C1-C2 level. The dislocation had carried the vertebral arteries forward and compressed them (Fig. 6).29,50

Case 5 (Chronic Hyperextension Injury With Atlanto-Axial Dislocation). While leaping to catch a pass this professional football player was tackled from the rear, landing on his back and striking his occiput. Although dazed and dizzy he did not lose consciousness. He had pain in the back of the head, neck, and between the scapulae. The unremitting headache finally subsided after 2 weeks. Approximately 2 months later he sustained an identical type of injury. There-


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after his symptoms progressed so that he missed two games but was able to complete the season. Whenever he bent over or flexed his neck he experienced dizziness and blurred vision. At no time did he lose consciousness. When lying down with both hands behind his head he noted tingling in his arms which disappeared when he placed his arms beside his body. After starting football practice again during the following summer his symptoms recurred with greater severity and led to medical consultation.

On examination he had a fairly full range of motion of his neck except for pain and partial limitation on cervical flexion. The cervical spine x-ray films showed an 8 mm atlanto-axial dislocation on forward flexion of the neck (Fig. 7 left), which was corrected by hyperextension (Fig. 7 right). A fracture of the odontoid process had been excluded and it was believed that the dislocation was secondary to a disruption of the transverse portion of the cruciate ligament. A stabilization procedure was performed with wiring of the laminae of C-1 and C-2 so that the spine was maintained in correct position.

Acute Massive Cerebral Displacement

When a football player runs head on into an opponent who is charging directly at him the impact may cause instantaneous respiratory or cardiac arrest. The mechanism for such an injury, commonly called "spearing," may involve impairment of the vascular supply to the brain stem. There may be difficulty in certain cases in differentiating an expanding supratentorial lesion with an uncal herniation from an acute brain stem contusion, infarction, or hemorrhage. Cases 6 and 7 illustrate this problem.

Case 6 (Acute Brain Stem Contusion). This 16-year-old high school player, charging forward with the ball, dove directly into the line, striking the abdomen of an opposing player. He was immediately unconscious and required artificial respiration to initiate breathing. Oxygen was administered as he was rushed to the hospital where he was found to be semicomatose, vomiting, and with right hemiplegia. The blood pressure was 110/70, pulse 72, and the labored respirations 24 per min. The left pupil was 1 to 2 mm. larger than the right. The fundi
were normal. The right extremities exhibited spastic extension with hyperreflexia and a Babinski response. The x-ray films of the skull and cervical spine, including special odontoid views, appeared to be normal. The patient was given mercuhydrin and intramuscular phenergan. A lumbar puncture was performed without difficulty; the spinal fluid was pink and under a pressure of 120 mm H₂O.

With a diagnosis of brain stem contusion, he was placed on a hypothermic regime. Bilateral frontal burr holes were made 36 hours after injury to exclude a supratentorial lesion, and clear colorless fluid under increased pressure was obtained. During the procedure his blood pressure fell, and he progressed to a decerebrate state. Hypotension continued in spite of the administration of vasopressor agents, a careful balancing of his electrolytes, and the maintenance of a hypothermic regime with his temperature at 90°. The patient died a week after the injury. Autopsy showed a basilar artery thrombosis with infarction of thepons, plus terminal pneumonia. There was no evidence of any expanding supratentorial lesion other than massive cerebral edema.

Case 7 (Disparity in Movement of Brain and Cord). A college football linebacker was injured in a scrimmage when he made a head-on tackle leaving the ground and striking a hard-driving ball carrier directly in the abdomen. He had an immediate cardiac and respiratory arrest with complete apnea for 20 min, but with the aid of cardiopulmonary resuscitation in the form of intracardiac adrenalin and external cardiac massage he regained spontaneous respirations and a cardiac rhythm within an hour at the local hospital. The patient was then transferred...
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**Fig. 6.** Case 4. Lateral view of dislocated atlas on axis showing points of compression of vertebral artery at 1st cervical intervertebral foramen and point where occipital condyle slides over groove in 1st cervical lamina. (Courtesy of *Neurology*, 1959, 9:643-656. Schneider, R. C., and Crosby, E. C. Vascular insufficiency of the brain stem and spinal cord in spinal trauma.)

**Fig. 7.** Case 5. Lateral x-ray views of the cervical spine demonstrate the degree of instability between the atlas and the axis. *Left:* In flexion there is a 7 mm dislocation (*arrows*). *Right:* In hyperextension the anterior ring of the atlas fits firmly against the odontoid process.
to another hospital where an examination revealed an areflexic tetraplegia with no response to painful stimuli. His pupils were equal, and he opened his eyes occasionally. There was frequent irregular twitching of the face and neck muscles, and he had only diaphragmatic respirations. The patient's skull and cervical spine x-ray films revealed no lesion. Although there was an equivocal cardiac enlargement in the chest film, the electrocardiogram was normal. A tracheostomy was performed and a frothy material aspirated from the intratracheal tube. A respirator was used, but he died 17 ½ hours after injury.

At postmortem examination there was no evidence of any expanding intracranial hemorrhage. There was bilateral uncal herniation with midbrain compression due to cerebral edema. Pathological sections of the cerebellum showed the type of necrosis in the Purkinje cell layer of the cerebellum characteristic of hypoxia. Sections of the cervical spinal cord demonstrated recent traumatic contusion with hemorrhage and necrosis at the C1-C2 level approximately 3 cm below the medulla (Fig. 8). These bilateral lesions were in the portion of the ventrolateral reticulospinal tract which contains respiratory neurons.

**Discussion**

In the majority of these cases, attention has been directed to the cervicomedullary damage that may result from the impairment of vertebral or basilar artery blood flow. The manifestations of nausea, dizziness, unsteadiness, nystagmus, dysarthria, blurring of vision, and unconsciousness have been regarded as the sequelae of vascular insufficiency of these vessels. Many of the reports in the literature concerning this topic are related to chiropractic manipulations or forced torsion of the neck, which have been major etiologic factors. The latter mechanism has been demonstrated angiographically. In some instances the neurological sequelae may be attributed to a direct compression of the high cervical spinal cord near the cervicomedullary junction or may be secondary to a compromise of the blood flow through the vertebral arteries due to spasm or subsequent propagation of a thrombus from the proximal site of injury distally into the basilar artery.

Injuries to the vertebral artery have been
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reported in association with fractures or fracture dislocations of the cervical spine. Symptoms of posterior inferior cerebellar artery insufficiency have been described by Suechting and French in fracture dislocation of the cervical spine and by Schwarz, et al., after chiropractic manipulation. The direct blow with the radial border of the extended hand to the lateral cervical spine at the C1-C2 level is an accepted karate technique which is very effective in causing probable transient vertebral insufficiency with unconsciousness. The contusion to the vertebral artery occurs at the atlas or atlanto-occipital membrane. Forcible cervical hyperextension in football players may result in transient vertebral artery spasm when the occipital condyle compresses the vertebral artery as it traverses the C-1 lamina. This mechanism is illustrated by Case 1 in which the patient exhibited the syndrome of acute central cervical cord injury syndrome followed by complete recovery. In some instances the hyperextension of the cervical spine due to a blow on the face mask of a football player may result in the posterior part of the rigid helmet, which is firmly fixed on the head by the chin strap, guillotining the neck to produce a fracture dislocation of the cervical spine with delayed partial or immediate complete transection of the cervical spinal cord.

In our Case 4 there was delayed flexion of the cervical spine with forward displacement of the vertebral arteries, already captive in the foramina transversaria, that resulted in impaired blood flow to the brain stem, a decerebrate convulsive seizure, and secondary high cervical cord transection. In Case 5 a similar atlanto-axial dislocation occurred but in that case there was a forced cervical flexion injury when the player received a blow to the occiput as he fell, forcing the cervical spine into flexion, causing disruption of the transverse portion of the cruciate or check ligament and thus permitting the characteristic dislocation. The symptoms of this type of injury may occur immediately after the injury.

The normal blood supply of the cervical spinal cord is currently receiving considerable attention. In general, the major portion of this supply is through the vertebral arteries, the anterior spinal artery, and the anterior sulcal (or central) artery to supply the center of the spinal cord. There is a pial mesh circulation (the arteriae coronae of the older literature) about the circumference of the cord, which evolves from the subclavian and radicular arteries in the cervical region, and supplies the periphery of the cervical spinal cord.

There appear to be zones of poor collateral circulation at the T-4 and T-12 levels of the spinal cord due to diminished flow through the anterior spinal artery at these levels. Turnbull, et al., have stressed the importance of the radicular arteries and noted that the anterior radicular vessels divide into ascending and descending branches which are then designated as the anterior spinal artery. Their excellent microangiographic studies of the spinal cords of 43 cadavers demonstrate the paucity of both anterior and posterior radicular vessels in the upper three cervical levels.

Liss reported the case of a swimmer who, without striking his head, completed a turn in a race, and promptly developed deterioration of movement and died as he reached the end of the pool. Autopsy showed centrally located hemorrhages near the ventrolateral reticulospinal tract which probably compromised the respiratory pathways at the C-2 level and resulted in death. Could this have been caused by sudden anterior and posterior radicular artery or vertebral artery insufficiency as he threw his head about suddenly in flexion to make a turn? This is not an unusual movement in swimming. In Liss's case there were petechial hemorrhages at the C-2 level similar to the acute lesions in our Case 7. The area of chronic cavitation which was found at the autopsy of the football player in Case 2 was also at the C-2 level. The injury in the latter instance presumably occurred in a hyperextension position although there is no photographic documentation. The description of the play at the time of impact did not suggest the application of the same tremendous force which may be generated in a direct vertex impact.

From their experimental work in dogs, Shimomura, et al., believe that obstruction at the origin of either the vertebral or radicular vessels has little relationship to cervical ischemic myelopathy; this is contrary to
some of the human case reports. However, Ducker and Assenmacher using microphotographic techniques in monkeys have clearly demonstrated transient blanching of the anterior surface of the spinal cord when forced hyperextension of the neck causes local washing out or spasm of the anterior spinal arteries and the radicular vessels. There can be no doubt that both direct and indirect compromise of blood flow to the cervical and upper thoracic spinal cord may be an important cause of intramedullary hemorrhages or cavitation in animals and in man.

Another significant kinematic factor in these football injuries is the tremendous transmission of force from the brain to the medulla and upper cervical spinal cord during severe direct vertex impact, as documented in Cases 6 and 7. There is a direct transmission of force through the unyielding or non-resilient football helmet and the intact skull to the underlying brain.

At least three isolated or concurrent factors may be present in such cases. Initially the brain moves en masse in a cephalad direction but then rebounds (Fig. 8) forcing the cerebral hemispheres caudalward so that the uncus is acutely thrust through the tentorial notch (Fig. 9, see 1). This mechanism will cause compression of the posterior cerebral arteries (and possibly even the more caudal superior cerebellar arteries) and result in hypoxia of the cerebral hemispheres and brain stem. The massive caudal shift of the cerebral hemispheres may also transiently occlude the readily compressible subarachnoid space, straight and lateral venous sinuses (Fig. 9, see 2), confluent, and thin-walled bridging veins such as the vein of Labbé. The combination of cerebral hypoxia and simultaneous impairment of venous drainage may cause sudden massive cerebral edema and result in the rapid development of a tentorial pressure cone and instant death.

Superimposed upon this supratentorial problem is a second one, namely, the massive shift of the cerebellum and the lower brain stem during direct vertex impact. The caudal displacement of the cerebellar tonsils through the foramen magnum simultaneously creates a cerebellar pressure cone with direct compression of the cardiac and respiratory centers in the medulla (Fig. 9, see 3), and vascular spasm or compression of the vertebral arteries at the foramen magnum (Fig. 9, see 4). The impact may also cause compression of the vertebral arteries by the occipital condyles against the C-1 laminae (Fig. 9, see 5) resulting in further vascular insufficiency as in Case 6. Lindenberg has noted entrapment of the basilar artery in a fracture of the occipital region, and Loop, et al. have shown a similar ensnaring of the basilar artery in a clival fracture. However, blunt trauma to these vessels on impact may be sufficient to cause the damage.

A third factor must be specifically considered. With the massive thrust developed on vertex impact there is a disparity between the marked degree of distortion of the freely movable brain in the intracranial cavity and the relatively fixed upper spinal cord which is rigidly held by the dentate ligaments in the narrow bone canal (Fig. 9, see 6). It was postulated that there is a differential gradient of pressures and the interface of such forces would be at the C1-C2 level directly at the site where the petechial hemorrhages were found in this case (Fig. 9, see 7). Brie has shown in cadavers that the dentate ligaments anchoring the cervical spinal cord to some degree prevent movement in the cephalad and caudal directions as contrasted to mobility in the anterior and posterior planes of the spinal canal. In the preliminary experimental work reported by Gosch, et al., monkeys subjected to vertex or "head-on" impacts had petechial hemorrhages only at the C-2 level. Another monkey of the same weight, with the upper dentate ligaments sectioned, was subjected to the same impact and survived without hemorrhages in the upper cervical spinal cord.

The patterns of experimental brain distortion and ischemia, which have been described by Weinstein, et al., using balloons inflated to various sizes at different speeds with compression of the intracranial contents, cannot be correlated with the high-speed direct vertex impact of the football player.

Summary

Seven cases of serious cervicomедullary football trauma have been presented and two different mechanisms of injury discussed: 1)
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Fig. 9. Case 7. The dynamic mechanisms involved in vertex impact injuries are shown as follows. Acute tentorial pressure cone with uncal herniation (1) compressing the posterior cerebral artery, superior cerebellar artery, and brain stem; (2) obliteration of the subarachnoid space, bridging veins, and lateral sinuses. Acute cerebellar pressure cone (3) with compression of the medullary centers and the vertebral artery at the foramen magnum (4). There may be concomitant occlusion of the vertebral artery between the occipital condyle and C-1 lamina (5). The gradient of forces between the thrust of the freely moving brain caudalward and the fixation of the cervical spinal cord by the dentate ligaments (6) result in distortion of the cord and petechial hemorrhages (7).

vascular insufficiency of the vertebrobasilar system intensified at a probable normal zone of poor collateral blood supply at the C-2 level, and 2) a disparate distortion between the freely moving brain and the relatively fixed upper cervical spinal cord, resulting in a wave-like motion that produces petechial hemorrhages at the C-2 level of the spinal cord. “Stick-blocking” and “spearing” techniques, namely, the direct use of the head in blocking and tackling the charging opponent, are frequently responsible for these disabling injuries and deaths.

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