Cervical spinal cord hemorrhages in experimental head injuries

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Petechial hemorrhages at the cervicomедullary junction have been noted in football players who sustained direct "head-on" or vertex impacts when they struck an opponent. Head and cervical spinal cord injuries were produced in experimental animals on an impact track simulating this mechanism. Severe cervical spinal cord destruction was obtained in the absence of cervical flexion and extension. Cord movement was enhanced by sectioning the dentate ligaments, which prevented these lesions. It is postulated that the transmission of shear strains along the axis of acceleration is responsible for the hemorrhages when the elastic deformation of the cervical spinal cord is exceeded.

KEY WORDS:  head injury  cervical spinal cord hemorrhage

The development of petechial hemorrhages in the upper cervical spinal cord segments in experimental cerebral trauma has been observed by several investigators.2,8,9,11,20 Jakob11 found hemorrhages only after a considerable interval from the time of injury. Denny-Brown and Russell,2 contrary to previous observations, regarded these as an immediate and direct result of acceleration concussion. The distance of these lesions from the site of injury has raised questions, not only of the mechanism of their formation, but also of the relation these findings may have to experimental cerebral concussion.2,3

Schneider, et al.,17 reported two cases of football players who sustained a direct vertex impact to the head, with a fatal outcome. The only pathological findings were restricted to the second cervical spinal cord segment. In one case, petechial hemorrhages were found clustered centrally in the cord at this level, particularly in the area of the ventrolateral reticulospinal tracts, the vital pathways for respiration, so that death was practically instantaneous. In the second case there was coalescence of hemorrhages resulting in cavitation at the C-2 level with immediate tetraplegia and death.

These clinical cases and our observations of marked deformation and displacement of the cerebral hemispheres during experimental impact injuries17 suggested the mechanism of the direct transmission of impact forces from the skull and brain along the spinal axis with their distribution to the cord. An experiment was designed to reproduce direct vertex impact injuries by accelerating animals with fixed heads against a rigid barrier containing a transducer load cell.

Materials and Method

Three groups of animals were used. The animals in the first group of eight adult Macaca mulatta monkeys with an average weight of 6.5 kg were anesthetized with 20 mg of intramuscular phencyclidine hydrochloride (Sernylan). Each animal was secured on a carriage with the cervical canal at an approximate 90° angle to the site of im-
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Impact. The head was restrained to prevent flexion and extension movements during the impact. Compressed air accelerated the carriage with the animal along a double track against the vertically fixed metal barrier. The energy delivered to the vertex of the skull was recorded by a quartz transducer load cell* mounted in the impact block. The electrical signal was amplified and monitored simultaneously on a two-channel oscilloscope and a Honeywell 7600 tape recorder. Periodic calibrations of the load cell were made by static weight loading and recording the voltage output on the oscilloscope. The impact charge at the time of injury was stored on the tape recorder and then was printed out on a Grass polygraph. The voltage amplitude indicating the magnitude of impact at the vertex of the skull of each animal was converted to foot pounds.

A total cervical laminectomy was performed on a second group of six Macaca monkeys. Intramuscular sodium pentobarbital (50 mg) was administered for anesthesia. Three animals in this group in which the dura was merely opened and reapproximated served as controls. The remainder of these animals were treated similarly but had the dentate ligaments sectioned bilaterally from the foramen magnum to the C-5 segment. The dura was closed, and the paraspinal musculature was reapproximated. Impact injuries were produced to the vertex of the skull in all animals as described for the initial group.

For the cinematographic documentation of the cervical cord at the time of injury, an extensive cervical laminectomy was made in the third group of monkeys. The neck muscles were retracted laterally to provide adequate exposure of the spinal cord. This was done in six anesthetized and two dead animals. In the latter, better visualization of the cervical cord dynamics could be obtained, because of the bloodless field. A Hycam high-speed camera was mounted directly over the impact site recording the events at the time of impact at 3000 frames/sec.

All animals died within 1 hour from the time of injury or were sacrificed no longer than 2 hours after impact. A total laminectomy from C-1 through T-2 was performed on the animals not operated on prior to the impact. The brain was exposed through a craniectomy and removed with the cervical spinal cord in all animals. The specimen was examined for gross abnormalities and fixed in 10% formalin. Representative sections of the brain stem and spinal cord were embedded in paraffin, stained with hematoxylin and eosin, and examined microscopically for pathological changes.

Results

A single direct vertex impact of only 25 ft lb on a 5 kg Macaca monkey caused hemorrhages in the upper cervical segments without cervical spine fracture in the nonoperated group of animals. A higher force was required in the larger animal to produce a similar result. The average impact to the vertex of all monkeys was 29 ft lb. It should be emphasized that the impact site and the vertebral canal were placed in a straight line. In this manner it appears that a direct transmission of the impact occurs to the brain stem and the cervical spinal cord.

The immediate post-injury period of the animals was usually marked by a transient extensor spasm and irregular respiratory excursions while the animal was under light Sernylan anesthesia. Some animals showed gradual respiratory recovery. The signs commonly cited as indicative of experimental cerebral concussion, paralysis of the corneal and pinna reflexes, were observed in our experiment. The animals, however, usually died or were sacrificed before reversal of the concussion could occur.

Hemorrhages in the upper cervical cord, varying from small discrete punctate lesions to gross destruction, were found in all animals subjected to an impact of more than 25 ft lb to the vertex of the head under our experimental conditions. The extent and amount of cord destruction varied with the magnitude of impact and probably also with the degree of muscular and bony absorption of the impact energy in each individual animal.

High-speed photography at the time of injury showed marked dissipation of energy in the neck region. As the vertex of the skull struck the impact plate, the upper trunk, particularly the shoulder region, continued to move in the direction of acceleration with

* From Kistler Instrument Corporation.
compression and telescoping of the neck and head. Flexion or extension of the cervical spine was not visualized before the animal recoiled from the impact plate.

Figure 1 (top) shows a cross section of the spinal cord at the C2–3 level from an adult monkey that had received a vertex impact of 29 ft lb. The central gray matter suffered marked hemorrhagic destruction, extending into the posterior funiculi. The external surface of the cervical cord showed no contusion and the surface vessels appeared intact. The only gross abnormality was a generalized mild hemorrhage in the cerebral and spinal subarachnoid spaces. No gross intracerebral lesions were found, and even microscopic sections of the hemispheres, pons, and medulla revealed no structural changes. The zone of greatest cord destruction was in the third cervical segment with extension rostral to the C-2 and caudal to the C-4 levels. This relationship is demonstrated in a sagittal section of a specimen taken from another animal (Fig. 1 bottom).

A total cervical laminectomy of the C1–6 laminae and extensive posterior decompression, did not alter the spinal damage. Sectioning the dentate ligaments bilaterally throughout the extent of the laminectomy did not produce hemorrhages with a vertex impact of 25 to 30 ft lb. In one animal only were there smaller central lesions at the cervicothoracic junction below the level of the cut dentate ligaments.

High-speed cinematography at the time of injury aided in the study of the mechanical factors involved at the cervicomedullary junction. The cervical spinal cord showed minimal motion in the cephalocaudal direction (Fig. 2), in contrast to the marked movement of the cerebral hemispheres previously reported (Fig. 3). At the time of impact the vertebral spaces were compressed and the foramen magnum telescoped over the upper cervical cord segments, while the cord itself remained relatively stationary. There seemed to be a direct transmission of energy from the vertex of the skull and cerebrum caudally to the cervicomedullary junction. However, when the dentate ligaments had been sectioned, the cord would not rigidly oppose the cervical distortion and movement in the cephalocaudal direction was enhanced.

Discussion

A number of hypotheses for the mechanism of head injuries and concussion have been proposed. Analyses of the various factors responsible for injury have differed among the authors, but the significance of shear stresses as a cause of central nervous system damage has in general been accepted. Holbourn stressed the significance of the relative incompressibility of brain tissue in contrast to its ability to change shape. The high ratio of these two properties, namely, compression and elasticity are then the cause for shear strains at predictable locations. The importance of rotational acceleration or motion about the center of gravity of the brain during impact in causing elastic flow has been pointed out by some investigators. Thomas, et al., and Lindgren, however, emphasized the formation of a

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**Fig. 2.** The cervical cord at the C1-4 level without dentate ligaments sectioned is visualized at .0015-sec intervals just before and after a vertex impact, by two frames from a high-speed overhead film. Hemostatic clips are placed on the arachnoid (A) and dura (D). The white pin (P1) has been inserted in an intervertebral space and (P2) into the cord directly. The large arrow indicates the direction of movement. Following impact, the point of insertion between P1 and P2 has not changed. The head of the pin continues to move in the direction of acceleration.

pressure gradient along the axis of acceleration. The presence of shear stresses, as a result of this pressure gradient, were subsequently demonstrated by implanting lead particles in the brain and studying the deformation by flash x-ray techniques. Also by direct observation through a transparent Lucite calvarium, Pudenz and Sheldon demonstrated brain deformation at the time of craniocerebral injury. This work has been confirmed in our laboratory where the Lexan calvarium has been used since 1967. This technique has permitted acute and chronic observations to be made for as long a period as 19 months without significantly altering the intracranial dynamics. If the transmission of energy from the site of impact is directed along the brain stem axis with the formation of shear stresses, then the greatest damage to the central nervous system should be produced at areas where elastic flow or deformation is hindered. The frequent contusion of the temporal and frontal lobes found in severe human head injuries provide clinical evidence in support of this hypothesis. The undersurface of the brain moves across the sharp bone ridges at the base of the skull causing contusions. The formation of subdural hematomas is believed to be the result of hemorrhage from bridging cerebral veins in the parietal region where the brain movement stretches these vessels to the point of rupture. The cervical cord, which is firmly anchored to its dural covering by the dentate ligaments, will be affected by stresses if these are of sufficient magnitude to be transmitted to this area. The importance of the dentate ligaments in rigidly

**Fig. 3.** The right cerebral cortex of a Macaca monkey visualized through a Lexan calvarium during a vertex impact of 25 ft lb. The large arrow indicates the direction of movement of the animal; the head is slightly rotated. The solid arrows point to the occipital poles moving in the direction of acceleration and separating from the occipital line.

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restraining the spinal cord and preventing its displacement posteriorly in the spinal canal in chronic compression was first stressed by Kahn. However, present evidence suggests that only motion of the cord in the cephalocaudal direction is restricted. In a comprehensive study of the biomechanics of the central nervous system, Breig demonstrated the plastic deformation of the spinal cord under various stresses; ventral flexion resulted in slight elongation and dorsal extension in compression of the cord. These changes in shape, however, cause no damage as long as the elastic property of the tissue has not been exceeded.

We propose, therefore, that the transmission of energy along the brain stem and spinal cord is the cause of the cervical cord hemorrhages. These lesions are concentrated in the upper cervical segments where the initial absorption of shear strains occurs before dissipation of the energy of impact. Relatively minimal cord movement during impact occurs because this region of the central nervous system is fixed by its lateral attachments. When plastic deformation is exceeded, tissues are subject to damage. This view is strengthened by the fact that sectioning the dentate ligaments prevents hemorrhage under similar conditions when movement and deformation is permitted. It might be suggested that flexion or extension at the cervicomедullary junction could have caused these lesions. We found, however, no evidence by high-speed cinematography that this mechanism of injury was involved, which is in agreement with the observations of Denny-Brown and Russell.

These additional data tend to confirm that shear strains in the central nervous system have a damaging effect. These strains may cause only a temporary neuronal dysfunction or have a permanent or fatal effect with destruction of tissue in vital areas of the nervous system.

Summary

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