PHOTOELASTIC CONFIRMATION OF THE PRESENCE OF SHEAR STRAINS AT THE CRANIOSPINAL JUNCTI0N IN CLOSED HEAD INJURY*

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In work reported previously it was shown that in closed head injury the concussive effects are caused by acceleration or deceleration of the head and/or deformation of the skull. These mechanisms result in sudden increase in intracranial pressure at the time of impact and mass movements of the contents of the skull. The levels of acceleration and intracranial pressure have been recorded and reported upon. Both the barium titanate and the Statham accelerometers have been used in the dog to record accelerations, and the strain gage pressure pick-ups have been utilized for recording increases in pressure at the time of impact. It has been found that the level of peak acceleration did not correlate with the degree of the concussive effect in the dog. In 1 animal, a peak acceleration of 750 g produced no concussive effect, while in others, fatal concussion accompanied much lower levels of acceleration. However, the level of the intracranial pressure and its duration correlated well with the physiological effects produced.

Since increase in intracranial pressure at the time of the impact appeared to be important in the production of the pathophysiological responses, another group of experiments were performed in which an external source of air pressure was connected to an opening in the skull through a valve which consisted of a spring-driven piston in a cylinder. By varying the external air pressure and the spring force in the valve, both the magnitude and the duration of the pressure could be controlled. The actual pressure to which the brain was subjected was measured with a strain gage pressure pick-up, screwed into a tapped hole in the skull on the side opposite to that where the pressure was introduced. The respirations, pulse and blood pressure were recorded during the tests. The microscopic study of the tissues of the brain showed that there was evidence of nerve-cell damage in the brain stem with no evidence of damage of nerve cells in the cortex, except an occasional instance in the deep portions of the temporal lobes and the posterior regions of the thalamus. These changes were found not only in severe and moderate injuries, but also in minimal and subconcussive effects. The damage in the craniospinal junction could be explained on the basis of shear strains caused by the anatomical features of the skull and spinal canal. The skull, a solid structure containing the brain, cerebrospinal fluid and blood (a semiliquid mixture), communicates through the foramen magnum with the spinal canal which is surrounded by a series of bones, the vertebrae, held together with intervening membranous tissue. The intervertebral foramina and the ligaments holding the vertebrae together may deform under the influence of sudden increase in pressure with rapid dissipation of the latter. With a sudden increase in pressure in the cranial cavity, a pressure gradient is produced at the foramen magnum because of the rapid dissipation of the intracranial pressure in the spinal canal. This pressure gradient causes shear strains resulting in neural damage. The magnitude of the shear strain produced is a function of
both the magnitude of the pressure within the cranial cavity, and its time duration.  

**TECHNIQUE**

The present study utilizes the photoelastic method. A 1-inch thick sagittal section containing the foramen magnum was reproduced in a plastic model. The opening at the base represented the foramen magnum and the beginning of the spinal canal (Fig. 1). This model was filled with a 1.5 per cent solution by weight of milling yellow (N.G.S.), an aniline dye which is doubly refracting when subjected to shear stress. The model was inverted with the simulated spinal canal superiorly, and it was suspended in a polariscope to study the stresses produced by impact.

**RESULTS**

When pressure was applied to the walls of the model, shear stresses were obtained in the region of the opening at the base, representing the region of the brain stem. Hammer blows delivered to the circumferential border and to the side walls of the model also resulted in shear stresses, predominantly in the simulated craniospinal junction. These changes were photographed by a movie camera at 50 to 500 frames per sec. The milling-yellow solution in the model shown in Fig. 2 has become doubly refracting in the craniospinal junction because of the shear stress in this area. The alternate dark and light bands are produced by the doubly refracting property of the milling-yellow structure because of the shear strain resulting from the blow. The magnitude of the shear strain is a function of the number of bands produced. It should be pointed out that the blows upon the border of the model resulted not only in deformation, but also in acceleration of the model. It is felt that increase in pressure is caused by both acceleration and deformation of the model.

**SUMMARY**

1. The photoelastic method has been used to investigate shear strains in a simulated head model representing a sagittal section of the skull and contents through the craniospinal junction.

2. The studies carried out with rapid cinephotography show a concentration of shear strains at the craniospinal junction of the model so tested.

3. It is felt that these shear strains are caused by increase in pressure in the model because of impact, with pressure gradients at the simulated craniospinal junction, re-
resulting in neural damage mainly in this region.

4. This confirms the previously reported studies utilizing the microscopic method. With known quantity and duration of pressure pulse applied to the dural sac of the dog’s head, damaged nerve cells were found in the brain stem, the posterior portion of the thalamus, and occasionally in the medial portions of the temporal lobes.

REFERENCES


