Commemorative Article

The physiological basis of concussion: 50 years later

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ALTHOUGH the brain has been thought to undergo physical changes when the head is struck a blow producing a loss of consciousness, the nature of the alterations has not been resolved. Fifty years ago, the explanations were related to a temporary cerebral anemia, a sudden increase in intracranial pressure, or traumatic paralysis of reflex activity of the brain. Based upon electroencephalographic records and clinical observations made at the time of a concussive blow, Walker, et al., concluded that the concussive phenomena were the result of traumatic rupture of the polarized cell membrane of many neurons, thus stimulating nervous tissue. This was akin to the report by von Haller in 1753 that a nerve, when irritated with a scalpel, caused a convulsion of the muscles.

A great deal of exquisite experimental work on concussion in the past half century has indicated the complexity of the problem. The old definition of concussion as the physical shaking of the nervous system has been replaced by a more specific concept involving not the entire brain (to a greater or lesser degree) but neuronal systems and circuits. When Magoun demonstrated that a multisynaptic "reticular activating system" regulated the state of consciousness, concussive blows were shown to depress its activity and to impair or abolish its responsibility. Acetylcholine, not normally present in the cerebrospinal fluid, was found in considerable amounts following a traumatic insult to animals and humans. The presence of acetylcholine was assumed to indicate a breakdown or depolarization of the cell membrane, and the amount of acetylcholine to correlate with the degree and length of coma. The fact that the direct current potentials of the cerebral cortex became 5 to 9 mV negative to white matter was further evidence of membrane depolarization.

The reported electroencephalographic concomitants have varied. Some investigators have reported no epileptic-like discharge at the moment of concussion. In the monkey, Ommaya described an immediate high-amplitude, slow-activity recording, particularly in the parietal areas. However, in animals and probably in humans, a long-lasting discharge followed trauma to the medial temporal structures (amygdala and hippocampus) as a result of the insertion of a needle electrode or electrical stimulation. The lack of agreement in results is possibly due to the variety of animals used, the dissimilar anesthetic agents, the diverse electronic recording equipment, and the different techniques employed to produce concussion.

Although by definition concussion is not associated with gross cerebral lesions, modern radiological techniques demonstrate edematous lesions or small hemorrhages, especially at points where dural septa impinge upon the cortex of persons who are comatose for more than a few minutes.

The clinical manifestations of concussion, in addition to the comatose state, have included graded unawareness usually manifested by a loss of recent memory. In man, numerous press reports have described athletes who, after sustaining a head injury without obvious loss of consciousness, have played the remainder of the game but later were unable to remember anything that happened after their mishap. Eschun, et al., produced concussion in rats, causing coma for 1 to 2 seconds but a memory loss for 30 minutes. Such a dissociation may be modified by pharmacological agents. After a severe injury of the somatosensory cor-
text in rats, Weisend and Feeney reported that the loss of ipsilateral hippocampal pyramidal cells of CA3 is markedly decreased by ketamine therapy. Because the recovery of beam-walking capability that correlates with the degree of thalamic cell loss and gliosis was the same in both treated and untreated rats, they suggested that the hippocampal alterations may result from excitotoxins responding to specific pharmaceutical agents.

The transition from the concepts of 1944 to those of 1994 has had a slow and irregular course, which is not to say that the beliefs of the 1940's were entirely erroneous. In some animals, a concussive blow did modify the cerebral blood flow, did cause a sudden rise in intracranial pressure, did produce paralysis of some reflex activity of the brain, and did mechanically stimulate some parts of the encephalon. However, these reactions were only fragments of the cerebral response to trauma. A great deal of well-planned and painstaking research in the past half century has given some insight into the complex concussive state but, "tho' much is taken, much abides" (Ulysses — Lord Tennyson).

References

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