EXPERIMENTAL CEREBRAL CONCUSSION*

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SINCE the incidence of closed head injury seems to be assuming increasing proportions in modern high-speed life, a better understanding of the phenomenon of cerebral concussion is needed. Unfortunately, relatively little basic information is yet available regarding the detailed pathophysiology of cerebral concussion.2

The mechanics of brain trauma have been investigated rather extensively and Denny-Brown and Russell1 have pointed out that both compression and acceleration concussion can occur although the latter is responsible for the majority of cases of concussion seen in man.

The appearance of large amounts of free acetylcholine in the spinal fluid of experimental animals following concussion has been described by Bornstein. He pointed out that while free acetylcholine is never normally present in cerebrospinal fluid, it is found in relatively large quantities shortly after head trauma. He also felt there was a positive correlation between the concentration of acetylcholine in the cerebrospinal fluid, the clinical signs of concussion and the postconcussion EEG changes. Tower and McEachern10 have studied these factors following closed head injury in the human and demonstrated low cholinesterase activity of the CSF as well as free acetylcholine which might be present in large amounts. Recovery was associated with reversal of these changes. These findings suggest a rationale of therapy specifically directed at the reversal of one of the sequelae of trauma of the brain. In experimental animals, Bornstein found that both the EEG pattern and the stuporous condition could be abolished by appropriate doses of atropine sulfate (0.5–1.0 mg./kg.). The extension of this anticholinergic therapy to man has been carried out by Ward,12 who reported the use of large daily injections of atropine sulfate in doses of 0.1 mg./kg. (gr. 1/10 in adult man) in an initial series of patients who had sustained very severe closed head injuries. He described dramatic and consistently reproducible clinical improvement in selected instances.

If this formulation of the role of acetylcholine in closed head injury is valid, experimental verification of two sets of data should be possible.

The first of these is the manner in which acetylcholine is released follow-

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ing cerebral concussion. Walker, Kollros and Case have presented experimental evidence that would indicate that the clinical manifestations of experimental concussion are the result of intense neuronal excitation of the central nervous system at the moment of the blow to the head. Bornstein has postulated that the appearance of the acetylcholine may be the result of this intense neuronal excitation. To gain further insight, detailed studies of cortical and subcortical electrical activity during and following concussion were therefore carried out.

The second set of data deals with the effects of circulating acetylcholine so produced, and specifically with its possible relation to so-called cerebral swelling. White et al. reported an increase in volume of the concussed cat brain associated with a moderate rise in cisternal pressure within 4 hours of concussion. Acetylcholine is a powerful vasodilator and may alter vascular permeability. It is conceivable that its presence in high concentration may be a factor in this increase in brain volume. Since a marked change in brain volume should cause a change in spinal fluid pressure, continuous recordings of spinal fluid pressures were made during and following concussion.

As a part of a broad investigation of cerebral concussion, this preliminary report deals with these two factors.

**MATERIAL AND METHODS**

Acceleration concussion was produced in 20 animals (19 cats and 1 Macacus rhesus monkey). The electrical activity of the cortex and subcortical structures, respirations, EKG, blood pressures, and spinal fluid pressures were recorded during and following acceleration concussion.

*Production of Concussion.* A gas pressure gun was designed and constructed to produce acceleration concussion (Fig. 1). It has a main chamber loaded with compressed nitrogen gas up to 100 lbs./square in. A piston-like plunger is contained in a barrel leading off from this chamber, and is held in position by a trigger mechanism. On release, piston velocities up to 45 ft./sec. are obtained. Acceleration up to 2500 ft./sec. is possible with interchangeable piston units. A built-in compression head on the barrel stops the piston within 3½ cm. after contact with the animal's head. This achieves an almost instantaneous blow, the minimum impact duration being 9 msec. with a piston velocity of 18 ft./sec. In all experiments, the weight of the piston was greater than the head of the animal so that factors of inertia may be disregarded. Cats of identical body weight (3 kg.) were used with a piston velocity of 18 ft./sec. The blow.

![Fig. 1. Concussion gun.](image-url)