PHYSIOLOGICAL BASIS OF CONCUSSION*

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(Received for publication September 10, 1957)

Based upon the studies of Gurdjian and associates\(^*\) rather discrete information is now available regarding certain of the physical forces involved in concussion as well as the changes in energy in skull and brain that occur at the moment of impact.

However, we must ultimately know the details of events that transpire at the membranes of nerve cells if we are to understand the phenomenon of concussion. Since obvious changes in the function of the central nervous system occur following head injury, it is certain that changes in the properties of neuronal membranes have been induced by this transfer of energy. However, we currently have no direct information on the mechanism by which this transfer of energy acts on membranes. The rise in intracranial pressure demonstrated by Gurdjian and Webster certainly does not produce concussion by simple obliteration of the blood supply to the nerve cells since concussion is immediate in onset whereas the potential of the neuronal membrane can be maintained for appreciable periods of time in the absence of blood flow. Unfortunately it is difficult to conceive of any other mechanism by which pressure alone, within reasonable limits, can alter the function of the membrane. However, it is possible to construct hypotheses by which shear forces can deform membranes if actual movement occurs. Such shear forces can induce depolarization of the membrane with profound alterations of function of the central nervous system. This proposal, in fact, has been made by Gurdjian et al. However, even the determination of relatively gross changes in energy of the type they describe is no mean achievement and investigation of the relationships of energy at submicroscopic levels is currently not feasible.

For this reason, we have elected to study the changes in the properties of neuronal membranes by observing the alterations in electrical activity of the cells at various levels of the central nervous system. Changes in spontaneous electrical activity at various levels of the brain should accompany the changes in function that are known to be present following head injury.

These studies, carried out over the past 6 years, have utilized acceleration as the method of producing the concussion since it is the common mechanism by which concussion is produced clinically, occurring in head injuries as the

\(^*\) This work was supported by a contract Nonr-942(00) between the Office of Naval Research and the University of Washington.

Presented at the meeting of the Harvey Cushing Society, Detroit, Michigan, April 35, 1957.
result of falls and automobile accidents. The criteria used to determine the presence of concussion were essentially the same as those proposed by Denny-Brown and Russell. The criteria were not altered by the presence or absence of anesthesia. Acceleration concussion was produced in cats and monkeys by means of a pneumatic gun in which a piston, driven by compressed nitrogen, strikes the freely suspended head of the animal, imparting to it a critical acceleration of the order of magnitude of 75 G's. The electrical activity of the cerebral cortex was monitored through dural electrodes implanted in the skull and the activity of subcortical structures was recorded from stereotactically implanted electrodes also rigidly fixed to the skull. These variables were monitored before, during and after the blow. Obviously the technical problems of recording the electrical activity at various levels of the central nervous system were not simplified by having the head moving through space with a velocity of 30 ft./sec. at the time critical measurements are to be made, but the artifact of movement was minimized to the point at which adequate recording could be obtained within one second of the blow.

At the moment of impact (Fig. 1) there is an immediate artifact from the blow. Less than a second later, when the amplifiers become unblocked, the changes in cortical activity are surprisingly minimal in view of the massive change in physiological function consisting of coma, apnea and loss of corneal reflexes. In certain cases, no definite changes in cortical activity could be observed although, in the majority of instances, a generalized flattening of the electroencephalogram appeared immediately after the blow and persisted for 10 minutes up to several hours. In no instance was any increase in cortical activity seen during the first 10 seconds following the blow and nothing resembling seizure discharges was observed at any time regardless of the presence or absence of anesthesia.

The electrical activity of certain subcortical structures was also moni-