Cerebral Hemodynamics and Metabolism Following Experimental Head Injury

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The pathogenesis of cerebral concussion has long been debated. The present experiments were undertaken to clarify the nature of acute cerebral disorders resulting from head injury.

Concussion was defined by Denny-Brown as a “transitory and reversible nervous reaction with immediate onset following physical stress of sufficient violence and brevity, and characterized by progressive recovery thereafter.” There are two main theories concerning the pathogenesis of concussion: the excitation theory of Walker, et al., and the paralytic theory of Denny-Brown and Russell. These two theories, which postulate opposite mechanisms, will be reviewed.

Walker and his associates observed the appearance of fast activity in the electroencephalogram (EEG) with little change in amplitude immediately after a compressive impact applied to the exposed dura and brain in experimental animals. This was followed by “extinction.” The EEG changes were frequently accompanied by tonic extension movements of the extremities. They suggested that this type of concussion resulted from excitation of the central nervous system.

An opposite view was proposed by Denny-Brown and Russell. Based on experimental observations of concussion produced by a pendulum striking the freely moving head (acceleration concussion), they concluded that this type of concussion was due to temporary paralysis of nervous function.

In man, concussion is characterized by transient loss of neural function, accompa-

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respiratory changes are important criteria for estimating the effects of concussion.

End-tidal CO₂ was monitored with a Beckman infrared gas analyzer, and if the carbon dioxide was not within the range of 30 to 42 mm Hg prior to injury, the data was discarded. Systemic blood pressure was recorded with a Statham pressure transducer connected to a catheter inserted into the abdominal aorta. Blood samples for measuring cerebral arteriovenous metabolic differences were taken from the femoral artery and a catheter inserted into the torcular.

A burr hole was made in the region of the occipital protuberance, and a small (0.9 mm in outer diameter) catheter was inserted into the torcular for sampling the cerebral venous blood. Another burr hole was made in the parietal region for inserting a small rubber balloon for recording intracranial pressure. A third burr hole was made in the opposite parietal region into which was screwed the muzzle of an airgun covered with a rubber membrane for providing the concussive blow. After adjusting the apparatus, each hole was tightly sealed with methylmethacrylate plastic cement.

Both internal jugular veins were exposed and their cervical branches ligated. The vertebral and external jugular veins were all exposed and ligated. Cerebral venous outflow was measured continuously by two electromagnetic flow meters whose probes were applied about each internal jugular vein. To calculate CBF in ml/100 gm brain/min, a correction was made for the sinus blood removed for metabolic measurements. The scalp and temporal muscles were incised and widely reflected, and six electrodes were inserted through drill holes in the frontal, parietal, and occipital regions for recording the EEG. The electrocardiogram (EKG) was also recorded.

The compressive blow was delivered by a specially constructed airgun.* The firing pressure was held constant at 40 lbs per sq inch, but the duration was varied between 10 ms and 300 ms. The longer the duration of the blow the more severe the concussion or contusion. The monkey was placed in the supine position with the neck slightly extended and the head fixed. Brain stem laceration was performed by thrusting a small scalpel into the exposed brain stem in the region of the pons, but care was taken to avoid laceration of the basilar artery and its major branches.

Cerebral arteriovenous oxygen differences (cerebral A-VO₂ differences) were monitored by the use of a Guyton A-VO₂ oxygen analyzer. Mean values for cerebral A-VO₂ difference obtained from the Guyton recorder agreed within 2% with the mean values obtained by the standard Van Slyke manometric method. The CMRO₂ was calculated from the product of CBF and cerebral A-VO₂ difference, and it was expressed as ml/100 gm brain/min.

Arterial (a) and cerebral venous (CV) values for pH were monitored with Beckman pH electrodes, values for oxygen tension (PO₂) with a Clark type electrode, and carbon dioxide tension (PCO₂) with a modified Severinghaus electrode mounted in a cuvette maintained at 37°C.* The animals were given intravenous heparin prior to these procedures.

Blood samples for lactate and pyruvate measurements were drawn from the femoral artery and from the torcular, and estimated by the colorimetric method of Hochella and Weinhouse for lactate, and by the fluorimetric method of Segal, et al., for pyruvate. Cerebral lactate and pyruvate production (CMR lactate and CMR pyruvate) were calculated from the product of CBF, and the cerebral arteriovenous lactate and pyruvate differences were expressed as mg/100 gm brain/min. Excess lactate formation by the brain was calculated from the formula of Huckabee expressed as mg/100 ml.

All recordings were graphed on a Grass Model 5 polygraph and a Grass EEG machine. Mean arterial blood pressure (MABP) was expressed in mm Hg (tor) by adding one-third of the pulse pressure to the diastolic pressure. Cerebral vascular resistance (CVR) was calculated by dividing MABP by CBF and expressed as mm Hg/ml/100 gm brain/min. At the end of each experiment, the brain was weighed and serial sections were examined.

The data were subjected to statistical

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