Respiration and the Cerebrospinal Fluid in Experimental Cerebral Concussion

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In recent years there has been increasing interest in the respiratory complications of cerebral trauma as well as the role of the cerebrospinal fluid (CSF) in respiration. Published reports have concentrated on the respiratory, acid-base, and blood gas alterations seen in more severe forms of cranial trauma where the comparability of the level of injury was questionable, and multiple injuries were frequently involved. The question then arises: What part of the devastating effects of respiratory disturbance after cranial trauma is precipitated by primary neurogenic factors and what part follows as secondary effects of airway obstruction and pulmonary insufficiency? Ducker and associates have helped to elucidate part of this problem in their study of the role of raised intracranial pressure and the Cushing reflex in the genesis of pulmonary edema after head injury. In an attempt to separate the primary central nervous system derangements from those caused by secondary airway and pulmonary factors, we have begun a study of respiratory function, blood, and CSF pH and gas tensions after experimental cerebral concussion (ECC). This model of head injury represents our baseline of minimal and reversible brain trauma wherein the physiopathology of blunt head injuries may be analyzed, and on which studies of more severe injury may be built. The criteria for concussion and the biomechanical aspects of the trauma were developed for the Rhesus monkey, as previously described by Ommaya, et al.

Materials and Methods

Thirty Rhesus monkeys (Macaca mulatta) weighing between 1.6 and 5.1 kg were used in this study. Under phencyclidine HCl, 2 mg/kg body weight, atropine 0.1 mg, and local 1% procaine, animals were prepared with central arterial and venous catheters, a right jugular bulb catheter, and a cisternal catheter introduced via a mid-dorsal laminectomy. Controlled ventilation was maintained with a Harvard Respirator and a succinylcholine drip or repeated doses of gallium triethiodide. End-tidal CO₂ was measured by a Godhart Capnograph and recorded on a Honeywell Electronic 19 Recorder. The respirator was adjusted according to serial blood gas measurements to provide optimum ventilation. A bilateral cervical vagectomy was performed in four monkeys. All wounds were closed, and the succinylcholine drip was stopped in 14 animals which resumed spontaneous respirations. Finally, each animal was given 10 cc of Evans Blue intravenously to help demonstrate lesions in the blood-brain barrier. During the surgery, small amounts of Ringer's lactate were given intravenously, and any acidosis resulting from the surgery was corrected with small amounts of NaHCO₃.

The following physiological data were recorded on two Gilson polygraphs (Fig. 1): EEG (scalp electrodes), EKG (standard limb leads), respiration, central arterial and venous pressure, and cisternal CSF pressure via suitable strain gauge manometers. Rectal temperature was recorded continuously with a Yellow Springs Tele-thermometer.

Blood and CSF pH and gas tensions were determined with the Instrumentation Laboratory Model 113 pH/Gas Analyzer. Arterial blood was sampled during surgical preparation at intervals of 45 min. After the animals were breathing spontaneously or in a steady state on the respirator, 1 cc samples of heparinized ascending aortic, inferior vena cava, and right jugular venous blood were analyzed for pH, pCO₂, and pO₂. A 1
Fig. 1. Experimental arrangement for analysis of respiratory and CSF changes after head injury. A. Top view of monkey with schematic connections to recording apparatus. B. Posterior view of monkey to show mode of suspension of head. C. Diagram of concussive gun used in these experiments.

c sample of cisternal CSF was also analyzed for pH, pCO₂, and pO₂ at approximately the same time. After these baseline samples were obtained, the animal was concussed and similar samples of blood and cisternal CSF were analyzed for pH, pCO₂, and pO₂, at intervals of 1 min, 20 mins, 40 mins, 1 hr, 2 hrs, 3 hrs and 4 hrs post-concussion. All samples were run immediately after being obtained. The same procedure and intervals for blood and CSF analysis was also used in control animals.

Concussion was produced by striking the animals on the occipital protuberance with a modified Remington Arms Company Humane Stunner Model 412-A, as previously described. During impact the animals were in the right lateral position with body and shoulders fixed, and head and neck suspended to provide uninhibited movement after the blow (Fig. 1 B). The criteria for concussion consisted of apnea over 3 sec in duration followed by irregular, slow respirations, a definite bradycardia as seen on the electrocardiographic tracing, loss of response to ear pinch, loss of corneal reflexes, and loss of voluntary movement. Confirmatory evidence of concussion consisted of an immediate onset of high amplitude slow activity in the electroencephalogram, lowering of the diastolic blood pressure and an increased pulse pressure, and electrocardiographic changes consisting of elevated T waves, prolonged Q-T interval, QRS complex amplitude changes, and PVC's.

After the 4-hr post-concussion samples were analyzed, each animal was killed by perfusion with a glutaraldehyde formaldehyde mixture. The brain and cervical spinal cord were removed en bloc, and careful note was made of any fractures, hematomas, hemorrhages, or contusions present. Color photographs were made of any surface lesions present, and the brains were then sectioned and photographed. The sections were then replaced in the fixative mixture for fu-