Experimental Effects of Acutely Increased Intracranial Pressure on Respiration and Blood Gases*

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Over the last 10 years there has been a growing number of clinical observations concerning the alteration of respiratory function with disease of the central nervous system.14,28,34-37,50 Within the specific field of head injury and increased intracranial pressure, improved monitoring techniques have called attention to the relationship of intracranial pressure waves and respiratory patterns.28 Equally important, and intimately related, have been the observations concerning blood gas and acid-base alterations in head injury, and their prognostic significance.5,18,33,50,55 These changes in the blood gases and acid-base balance alter the caliber of the cerebral vessels and the rate of cerebral blood flow. These in turn further increase the intracranial pressure.

Clinical observations of our own have led us to believe that alterations in respiratory pattern may well be one of the earliest signs of increased intracranial pressure, far preceding the classical symptoms of clinically observed changes in pulse and blood pressure. This paper reports an investigation of the interrelationship of intracranial pressure, respiration, and blood gases under a controlled situation, using an extradural balloon to simulate compression of an epidural hematoma.19,20 Such a model, while subject to the criticism that relatively slow compression by a balloon simulates only one form of acute injury, is nevertheless controllable and repeatable. The absence of other factors in acute injury, such as acceleration, deceleration, contusion, hemorrhage, and laceration, limits the injurious effects studied primarily to those caused by compression and its resultant distortion; the method also provides means for careful monitoring of these effects.

Materials and Methods

Thirty-three adult mongrel dogs were used, varying from 20 to 40 lbs. An extradural hematoma was simulated by a rubber balloon, placed in the epidural space through a trephine hole in the fronto-central region over the convexity, and connected to a syringe by a small polyethylene tube. The intracranial pressure was monitored by a similar but smaller balloon placed in the opposite epidural space, and connected by polyethylene catheter to a Statham strain gauge. The systems were filled with saline. Arterial blood pressure was monitored by a femoral artery catheter attached to a Statham strain gauge. Respirations were monitored by a Harvard pneumograph connected to a Statham gauge. The latter system was air-filled. Two silver wires were used to monitor a biparietal electroencephalograph (EEG). These were placed through small bony openings and rested on the dura. All skull defects were closed and the various devices fixed by applying acrylic dental cement. Recordings were made on the Beckman Dynograph.

All animals were anesthetized intravenously with Diabutal and maintained throughout the procedure on small amounts of the intravenous fluid. No additional anesthesia was needed or given during the course of compression. All animals were breathing spontaneously, but had endotracheal tubes in place. Secretions were cleared by suction periodically as needed.

Increments of fluid were added to the compressing balloon in amounts starting at 0.4 to 0.5 cc, and toward the end of compression tapering to 0.1 cc. The compression was carried out over a 2-hour period, until the animals showed evidence of cerebral vasoparesis, defined as "that state in which there is apparent loss of vasomotor tone of the cerebral vessels, as evidenced by the spontaneous rise of intracranial pressure in direct relation to, and paralleling, the sys-

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Acute elevations of intracranial pressure. This state was almost always accompanied by a flat EEG and fixed, dilated pupils.

Blood samples were taken and analyzed for PaCO₂, PaO₂ and pH at baseline, at the clear onset of slowing and deepening of the respiratory patterns, and at vasoparesis. In addition, in 14 animals, 100% O₂ was administered for periods of 3 minutes at the same stages of compression; blood gas samples were obtained before and during the administration of the O₂, which was always spontaneously inspired. Thus, each of these 14 animals served as his own control for blood gases.

Results

The initial alteration noted after compression was begun was a slowing and deepening in respiration, which would persist even with compensation of intracranial pressure to nearly baseline levels. If the animal was under relatively light anesthesia, a brief period of hyperventilation sometimes preceded this change (Figs. 1 and 2).

As compression continued, the respirations became deeper and less frequent (Fig. 3). By the time vasoparesis was clearly evident, they were occurring at intervals of 1 minute, either as single great gasps or bursts of two or three respirations in "decrescendo" order (Fig. 4). The intracranial pressure and blood pressure at this stage clearly showed waves correlated with the respirations. At the same time, the EEG would become flat bilaterally, and both pupils fixed and dilated. If at this point the pressure was not promptly relieved, the respirations would cease quite abruptly; the blood pressure and intracranial pressure would then fall and the animal would die.

The PaO₂, PaCO₂, and pH samples obtained from the femoral artery during compression are shown in Table 1. Since each dog serves as his own control, the variation in baseline values from dog to dog can be disregarded. In 76% of the animals there was a drop in PaO₂, and in 78% a rise in PaCO₂, by the time the respirations were clearly slower and deeper. The pH at this

![Fig. 1. Baseline and initial compression. Top line indicates minutes at slow speed (25 mm/min) and seconds at fast speed (25 mm/sec). Pressure calibrations are in mm Hg. Arrows above ICP line indicate increments of 0.2 cc fluid in compressing balloon. Arrows above respiration line indicate start and stop of administration of 100% O₂. No change is seen in respiration. Note speeding of respirations with compression.](image-url)