Therapeutic Value of Oxygen at Normal and Hyperbaric Pressure in Experimental Head Injury

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Previous work reported from this laboratory with a model that simulates an expanding extradural hematoma has shown a profound alteration in blood gases and respiration during head injury in dogs. It seemed reasonable to investigate the therapeutic benefits associated with improving the blood oxygen in the post-compression period.

Material and Method

Group 1 (Controls). The previously designed model of head injury was established in 20 dogs used as controls. After Nembutal anesthesia, bilateral fluid-filled extradural balloons were placed, a large one for compression by adding increments of fluid, and a smaller one connected to a Statham strain gauge for pressure measurements. Biparietal dural EEG leads were also placed and referred to the vertex. The balloons and EEG leads were fitted in place, and bone openings closed with acrylic cement. Arterial blood pressure was monitored by a femoral catheter attached to a Statham strain gauge, and respirations by a pneumotachygraph attached to a strain gauge. Fluid was added to the compressing balloon in small increments over a 1½ to 2½-hour period until we obtained both a flat EEG bilaterally for 3 minutes and a spontaneously rising intracranial pressure that corresponded to the blood pressure, reflecting vasoparesis. The balloon was then deflated. When these criteria were met, a 95% mortality was obtained, usually within 24 hours post-decompression, and at the longest, within 30 hours. Figure 1 shows the record of an animal at vasoparesis with rising intracranial pressure and flat EEG's.

Three additional groups of 10 dogs each were compared with the control group.

Group 2. These 10 dogs had respirations supported by a Bird respirator adjusted to deliver 100% oxygen at a rate and depth comparable to that recorded in the anesthetized dog prior to compression; the respirator was maintained for 4 hours following balloon decompression. Arterial PaCO₂ was monitored to be sure that a level above 25 mm Hg was maintained; we have found that this is the lower limit of normal range in the spontaneously respiring dog.

Group 3. These 10 dogs were allowed to respire 100% O₂ spontaneously for 4 hours at ambient pressure following balloon decompression.

Group 4. These 10 dogs spontaneously respired 100% O₂ at 2 atmospheres absolute pressure for 4 hours following balloon decompression in a small animal hyperbaric chamber.

Results

The results as compared to the controls in Group 1 are summarized in Table 1. The animals whose respirations were artificially supported (Group 2) showed a reduction in mortality of 70%. The quality of survival was very poor in five animals, however, and only two (20%) were able to feed themselves or ambulate.

The dogs in the groups spontaneously resiping 100% oxygen (Group 3) were progressively improved in quality of survival. With 100% O₂ at ambient pressure there was a 30% survival, and all dogs could walk, eat, and drink. Although the mortality rate was high, the length of time until death was increased to 3 to 5 days.

The group receiving 100% O₂ at 2 atmospheres (Group 4) showed still more reduction in the mortality to 50%, and the quality of survival was good in all. Even several of
the dogs that later died looked much better initially than the control dogs on first being removed from the chamber. Death occurred at 3 to 5 days, as it did in the animals receiving 100% \( \text{O}_2 \) at ambient pressure.

**Discussion**

In a separate study\(^9\) we have reported the effect of the balloon compression injury on cerebral blood flow and arterial sagittal sinus oxygen difference. In that study it was shown that, following balloon decompression, the arteriovenous oxygen difference progressively widens and the cerebral blood flow falls. Oxygen consumption, as the product of these functions, remains constant for a time until the increased oxygen extraction, as measured by the widening arteriovenous oxygen difference, can no longer compensate for the reduced flow. When this stage is reached, the oxygen consumption falls and shortly after the animal dies.

The primary problem in this form of injury, as we see it, is a metabolic one, with lack of sufficient available tissue oxygen. It is clear that the situation could be improved either by increasing the flow to the tissue, or by leaving the flow unchanged but increasing the amount of oxygen available per unit of blood flow. The effect of improving the flow reported in our previous paper\(^6\) resulted in a reduction in mortality of 50%, the survivors being of good quality. The present study makes no attempt to change blood flow or composition directly but, rather, to increase the amount of available oxygen carried in the blood.

It has been shown\(^1\) that increasing the inspired \( \text{O}_2 \) to 100% at ambient pressure, as in our Group 3, adds 2.28 cc of \( \text{O}_2 \) to the

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**Fig. 1.** Record of an animal at the point of vasoparesis. Time marked at top indicates minutes at slow speed and seconds at fast speed. Calibration of intracranial pressure is in mm of mercury. Note spontaneously rising intracranial pressure and blood pressure with concomitant flattening of EEG.