An Evaluation of Decompression in Experimental Head Injury*

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The therapy for acute brain swelling continues to be unsatisfactory in severe cases. Associated cerebral laceration and contusion account for much of the deficit noted, but there can be little doubt that the swelling which occurs can augment the degree of injury, and may possibly change a reversible lesion into an irreversible one. It is important to recognize that acute brain swelling has been shown to represent primarily an increase in the intravascular component of the intracranial volume, that is, it is a sudden distention of the vascular bed.³⁴ Later subacute brain swelling, or cerebral edema, may develop. This represents primarily an increase in the extravascular component of the intracranial volume, whether intra- or extracellular. This paper is primarily concerned with the reduction of morbidity and mortality in the acute swelling phase. We should recognize, however, that by reducing morbidity in the acute phase we may be ameliorating some of the factors that set the stage for the later development of subacute swelling.

One of the problems in assessing the various possible therapeutic methods in head injury is the establishment of an adequate model. If the model is a free head which is given a sudden, violent blow, the types and degree of injury cannot be well controlled. There may be varying degrees of superimposed brain laceration and contusion, as well as the added problems of acceleration, deceleration, and hemorrhage. Thus, the mortality and morbidity of such a model would include many more factors than swelling alone, and the results would be unpredictable. We have chosen extradural compression by a balloon because the method is simple, because the duration and intensity of the injury can be well controlled, and because a repeatable end point may be obtained. In terms of mortality one must decide whether to use a model yielding 100% mortality, 50% mortality, or some figure in between. A 100% mortality is convenient since any therapeutic alteration is significant. On the other hand, it necessarily represents a severe injury, may over-stress the animal, and may yield less suitable information than the 50% mortality figure. If a 50% mortality is used, far more animals must be used, both for control and experimental data. For simplicity we have chosen the 100% mortality figure.

On our model we used gradual inflation of an extradural balloon to simulate the conditions present in an acute epidural hematoma. If compression is stopped and the balloon deflated as soon as certain predetermined alterations in the intracranial pressure and electroencephalogram are reached, a variable number of animals will survive. If compression is continued, all will die almost immediately, before there is any opportunity to institute therapeutic methods. We tried to create an injury that would produce death slowly enough to permit time for therapeutic intervention. We also wished our injury to be carried to the point of irreversibility despite removal of our compression balloon. In order to achieve these factors, a period of negative phase hyperventilation was incorporated in the experiment. While we are aware of the objections to hyperventilation per se, due to its potential for increasing local ischemia, the technique was constant in both treated and untreated animals. Its effects, therefore, should not have altered the comparative observations, although possibly they contributed to the severity of the injury.

Materials and Method

Twenty mongrel dogs ranging from 25 to 40 lbs were used. Anesthesia consisted of intravenous Diabutal, 1 cc per 5 lbs weight. Following induction, the animals were intubated endotracheally and maintained on in-
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Travenous fluids. A Beckman dynograph was used for recording. Arterial blood pressure was monitored via a femoral catheter connected to a Statham strain gauge. Intracranial pressure was monitored by a saline-filled, small, extradural rubber balloon, connected by a catheter to a similar gauge. Respirations were monitored by a Harvard pneumograph bellows connected to a Statham strain gauge. Intracranial pressure was monitored by a saline-filled, small, extradural rubber balloon, connected by a catheter to a similar gauge. Respiations were monitored by a Harvard pneumograph bellows connected to a Statham strain gauge. Biparietal electroencephalogram recordings were made from silver wires placed against the dura and fixed with dental acrylic cement. Bilateral fronto-central burr holes were made, one for the recording balloon and one for the compressing balloon, a moderately large rubber balloon attached to a polyethylene catheter. Acrylic cement was used to close these defects also.

Following a baseline run, the compressing balloon was inflated in increments of 0.2 to 0.3 cc of fluid, tapering to 0.1 toward the end of the inflation period as one approached vasoparesis. We should state that we have taken vasoparesis here to mean the self-perpetuating increase in intracranial pressure, without additional increments of volume added to the balloon. This state has been shown by Langfitt, et al., and Ishii to be most likely related to the increase in the intravascular component of the intracranial volume, due to loss of vasomotor tone. Inflation was carried out until evidence of vasoparesis and bilaterally flat electroencephalograms were obtained. These usually occurred simultaneously, but on occasion an animal would develop incomplete evidence of vasoparesis at the time the EEG was bilaterally flat. Under such circumstances the EEG was observed for at least 3 min to be sure it was consistently flat (Fig. 1). It generally took about 2 hours to bring the animal to this point.

![ EEG tracing at onset of vasoparesis. Time markers at slow speed indicate minutes, and at fast speed, seconds. Note spontaneously rising intracranial pressure and blood pressure, with progressive flattening of EEG as described in text.](image-url)

Fig. 1. EEG tracing at onset of vasoparesis. Time markers at slow speed indicate minutes, and at fast speed, seconds. Note spontaneously rising intracranial pressure and blood pressure, with progressive flattening of EEG as described in text.