Title: MECHANICAL HYPERVENTILATION IN PATIENTS WITH HEAD TRAUMA POTENTIATES THE ICP RESPONSE TO PCO_2

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Introduction. Increases in arterial carbon dioxide tension (P_aCO_2) are often associated with increases in intracranial pressure (ICP).[1] Mechanical hyperventilation is used to lower P_aCO_2 and ICP in head trauma patients. Recently it has been shown that positive end-expiratory pressure may increase ICP in such patients.[2] The effect of mechanical ventilation (MV) on ICP has not been examined. We studied the effects of MV on ICP in head trauma patients independent of the effects of P_aCO_2.

Methods. Eight comatose adult patients ages 20-51 years were studied. They were paralyzed with either metocurine or a combination of pancuronium and d-tubocurare while monitored with a peripheral nerve stimulator. No sedatives or diuretics were given during the study or for several hours earlier, and patient stimulation was minimized during each study. Because we required stable patients with initial ICP values less than 20 torr, all subjects had been hyperventilated for several days. All patients were ventilated with a Siemens 900B ventilator using tidal volumes of 15cc/kg body weight. Blood gases were obtained from an indwelling arterial catheter. Arterial oxygen tension was always maintained at 90 torr or greater. ICP was continuously monitored using either a subdural or intraventricular catheter. Each patient was examined under two conditions; first keeping ventilation at a constant high rate while using inspired CO_2 to increase P_aCO_2 and in the second condition, slowing ventilation to similarly increase P_aCO_2. Each patient was initially ventilated to a P_aCO_2 of 26±3 torr. Compressed CO_2 was then added to the inspiratory limb to increase P_aCO_2 in a stepwise fashion, keeping ventilation constant. After steady state P_aCO_2 had been achieved, ICP and P_aCO_2 were recorded. P_aCO_2 was increased until a P_aCO_2 of 36-40 torr was achieved, or the ICP exceeded 20 torr. The inspired CO_2 was then discontinued and the P_aCO_2 was allowed to return to its initial level. The ventilatory rate was then decreased in a stepwise fashion to allow the P_aCO_2 to rise, and ICP was recorded at each level of P_aCO_2. The protocol was approved by our institution's human research committee.

Results. As shown in Figure 1, 5 of 8 patients had a marked ICP response to increases in P_aCO_2 with the addition of CO2 in the presence of constant high minute volume. When the P_aCO_2 was increased by decreasing ventilatory rate, much smaller increases in ICP were seen. The other patients had little ICP response to either CO_2 stimulus. Figure 2 is a plot of the linear slope of the ICP response to increase in P_aCO_2 in the two conditions which shows that in five patients the slope was greater when CO_2 was added than when ventilation was reduced.

Discussion. In addition to the effects of P_aCO_2 on ICP, increasing ventilatory rate may increase cerebral venous pressure by decreasing cerebral venous return. The beneficial effect of hypocapnea in head trauma patients may be opposed by the deleterious effects of mechanical hyperventilation. The febrile patient on controlled ventilation has increased CO_2 production without increased ventilation and mimics the experimental situation. Chronic hyperventilation may not only make it difficult to control the further ICP rises seen with such fevers, but may place the patient at additional risk because of the exaggerated response to CO_2. The degree of hyperventilation may be an important variable in the treatment of head trauma patients.

References.

1. Lundberg N:Continuous recording and control of ventricular fluid pressure in neurosurgical practice. Acta Psychiat Neurol Scand 36:1-193 (Suppl 149), 1960.

2. Shapiro HM, Marshall LF:Intracranial pressure responses to PEEP in head- injured patients. J. Trauma 18:254-256, 1978.

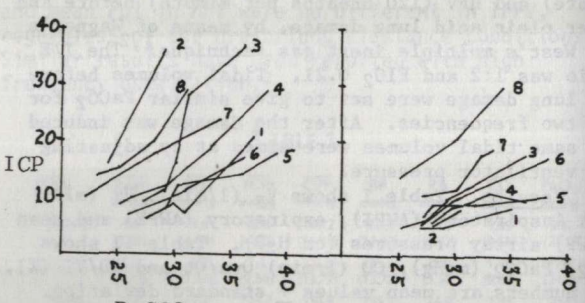

Fig. 1

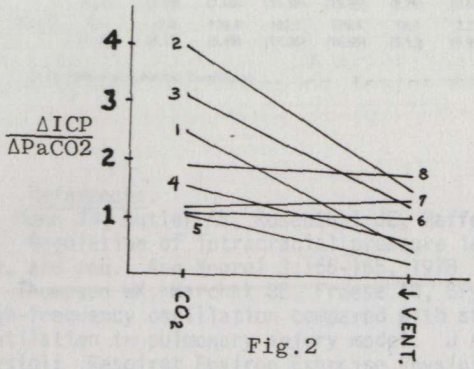

Fig.2