INTRODUCTION. Increases in arterial carbon dioxide tension (PaCO₂) are often associated with increases in intracranial pressure (ICP). Mechanical hyperventilation is used to lower PaCO₂ and ICP in head trauma patients. Recently it has been shown that positive end-expiratory pressure may increase ICP in such patients. 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 PaCO₂.

METHODS. Eight comatose adult patients ages 20-51 years were studied. They were paralyzed with either metocurine or a combination of pancuronium and d-tubocurarine 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 CO₂ to increase PaCO₂ and in the second condition, slowing ventilation to similarly increase PaCO₂. Each patient was initially ventilated to a PaCO₂ of 26±3 torr. Compressed CO₂ was then added to the inspiratory limb to increase PaCO₂ in a stepwise fashion, keeping ventilation constant. After steady state PaCO₂ had been achieved, ICP and PaCO₂ were recorded. PaCO₂ was increased until a PaCO₂ of 35-40 torr was achieved, or the ICP exceeded 20 torr. The inspired CO₂ was then discontinued and PaCO₂ was allowed to return to its initial level. The ventilatory rate was then decreased in a stepwise fashion to allow the PaCO₂ to rise, and ICP was recorded at each level of PaCO₂. 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 PaCO₂ with the addition of CO₂ in the presence of constant high minute volume. When the PaCO₂ was increased by decreasing ventilatory rate, much smaller increases in ICP were seen. The other patients had little ICP response to either CO₂ stimulus. Figure 2 is a plot of the linear slope of the ICP response to increase in PaCO₂ in the two conditions which shows that in five patients the slope was greater when CO₂ was added than when ventilation was reduced.

DISCUSSION. In addition to the effects of PaCO₂ on ICP, increasing ventilatory rate may increase cerebral venous pressure by decreasing cere-