Effects of positive end-expiratory pressure on intracranial pressure and compliance in brain-injured patients

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Positive end-expiratory pressure (PEEP) is established as a useful modification of controlled mechanical ventilation to improve oxygenation. The sustained pressure on the expiratory limb of the ventilator system increases the PaO₂ and decreases the alveolar-arterial oxygen tension gradient (A-a DO₂), without increasing the inspired oxygen concentration (FiO₂). In most cases it is then
possible to avoid a high, potentially toxic FiO2 while still maintaining an adequate PaO2.

It has also been suggested that PEEP, by increasing central venous pressure (CVP) and impeding cerebral venous return, must increase intracranial pressure (ICP). Increases in intrathoracic pressure may also decrease venous return and cardiac output, particularly in the neurosurgical patient who may be dehydrated following diuretic administration, which would result in decrease of cerebral perfusion pressure. Were these changes to occur, they would clearly be detrimental to the already compromised brain.

Initial laboratory studies done in cats demonstrated no significant increase in ICP with increasing PEEP. The following study examines the effect of incremental changes in PEEP on ICP at different values for intracranial compliance in man.

**Clinical Material and Method**

In seven comatose patients, ranging in age from 20 to 70 years, ICP was recorded by means of a Scott cannula* inserted into the lateral ventricle through a twist-drill hole. The cannula was connected by sterile intravenous tubing to a Statham transducer,† fixed to the level of the patient's occiput, for constant display and trend recording. All therapeutic maneuvers and measurements were carried out with the patients in a 30° head-up position. Arterial blood gas sampling and continuous blood pressure recording were obtained by direct cannulation of the radial artery using a No. 22 gauge catheter. Central venous pressure was obtained by cannulation of the anterior cubital vein. The position of the catheter was verified by chest films.

Swan-Ganz catheters‡ were placed in two patients to record pulmonary artery wedge pressure (PAWP) and cardiac output by the dye dilution technique. Pulmonary artery wedge or pulmonary capillary pressure was measured when the catheter tip was advanced into a small branch of the pulmonary vascular tree and a balloon inflated. In one patient, a catheter was inserted into the internal jugular vein and advanced to the jugular bulb to record changes in cerebral venous pressure.

All the patients were intubated and ventilated without the use of muscle relaxants. Arterial CO2 pressure (PaCO2) was maintained constant for each patient between 27 and 33 torr. Respiratory patterns were adjusted by the Bennett, Gill, or Emerson ventilators.§ Computerized circuitry controlled the application of PEEP in the first two machines (the Gill ventilator can, in fact, exert an end-expiratory pressure of +80 cm H2O but it is ordinarily inadvisable to exceed +40 cm H2O unless chest tubes have been inserted). The expiratory limb of the Emerson ventilator was positioned under water for the desired level.

All patients were established on conventional therapy for control of ICP with diuretics and steroids. Surgery was performed as indicated. No medications were given for 1 hour before recording measurements. The duration of studies was from 3 to 20 hours.

Initial intracranial compliance was calculated by the pressure volume index (PVI) according to the equation:

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PVI = \frac{\Delta V}{\log_{10} \frac{P_e}{P_0}},
\]

where \(V\) = volume injected to lateral ventricle, \(P_o = \) initial ICP, and \(P_p = \) peak ICP. A high PVI (about 20 to 25) signifies a high compliance; a low figure (< 10) indicates a severely compromised brain.

Five patients had significant pulmonary shunting (PaO2 70 torr at FiO2 0.4, representing at least 27% shunt). Two patients had normal arterial blood gases (PaO2 > 100 torr at FiO2 0.25).

**Results**

The use of PEEP from 5 to 20 cm H2O did not increase ICP (Fig. 1). At 12 cm H2O

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*Scott cannula manufactured by Codman-Shurtleff, Inc., Randolph, Massachusetts.
†Statham transducer manufactured by Statham Instruments, Inc., 2230 Statham Boulevard, Oxford, California.
‡Swan-Ganz catheters manufactured by Edwards Laboratories, 17221 Red Hill Avenue, Santa Ana, California.
§Bennett ventilator made by Bennett Respiration Products, Inc., 1639 Eleventh Street, Santa Monica, California. Gill ventilator made by Chemetron Medical Products, Division of Chemetron Corporation, 1801 Lilly Avenue, St. Louis, Missouri. Emerson ventilator made by J. H. Emerson Company, 22 Cottage Park Avenue, Cambridge, Massachusetts.
Positive end-expiratory pressure and ICP

PEEP did not cause significant change in ICP after 20 hours of continuous use in one patient (Case 5).

In two patients, PEEP at 40 cm H₂O for 15 minutes caused a slight (12%) but statistically insignificant increase in mean ICP with only a 15% increase in PaO₂ and <1% improvement in oxygen saturation over levels obtained at 20 cm H₂O PEEP. It was observed that the ICP pulse waves became higher and sharper when PEEP was increased above 35 cm H₂O (Fig. 2). Intracranial compliance was unaffected by PEEP used within the therapeutic range (pressures to 20 cm H₂O).

Cases 4 and 7, who had initial calculated PVI's of 8 and 7 (that is, low compliance systems), had similar values (9 and 8) at the conclusion of 5 hours of study. In the other five patients with moderate compromise there were similarly no statistical changes in the calculated PVI from the beginning to the conclusion of the study (<20%). Three of these patients who had all had more than 30% pulmonary shunting showed significant improvement in the PVI (increase of 50%) by the following day.

The CVP increased in all patients from a resting level off the ventilator of 1 to 4 cm H₂O to 20 to 24 cm H₂O, with PEEP up to 20 cm H₂O (Fig. 3). Similar increases in PAWP were seen in the two patients in whom Swan-Ganz catheters were inserted (Fig. 4). Cardiac output varied by 12% in the two patients studied over ranges of PEEP from 5 to 20 cm H₂O (not statistically significant).

Jugular bulb pressure was measured in only one patient on three occasions over a 4-hour period. This patient had severe respiratory distress syndrome (PaO₂ 35 torr at FiO₂ 0.6) and very low brain compliance. Increase in CVP on each occasion was less than 1 cm H₂O between zero end-expiratory pressure and PEEP of 12 cm H₂O.

Abrupt discontinuation of PEEP did not result in an increase in ICP except for a transient rise on two occasions when respiratory secretions became copious and the patients were inadequately ventilated. There were no significant changes in blood pressure or in pulse measurements in any of the patients.

All the patients had marked improvement in arterial oxygenation after 1 hour of ventila-
FIG. 2. Wave patterns with changing positive end-expiratory pressure (PEEP). Effect on intracranial pressure (ICP) wave patterns of incremental changes in airway pressure from zero end-expiratory pressure (ZEEP) to 40 cm H$_2$O PEEP. PVI = pressure volume index.

FIG. 3. Response of central venous pressure (CVP) to changing positive end-expiratory pressure (PEEP). Average increase in CVP with stepwise increase in PEEP in seven patients.

FIG. 4. Pulmonary arterial wedge pressure (PAWP) versus increasing positive end-expiratory pressure (PEEP). Average increase in PAWP in two patients as end-expiratory pressure is increased from 0 to 25 cm H$_2$O.
tion with 15 cm H₂O PEEP (average 75% increase over the ventilated state without the use of PEEP). In two patients this was concomitant with considerable improvement in neurological status.

**Discussion**

How is the increased intrapulmonary pressure dissipated? That there is not a simple transfer of pressure from the intrathoracic cavity to the intracranial compartment through the venous system has been demonstrated by the failure of the G-suit to prevent air embolism and its inability to maintain an elevated CVP. It has also been shown that elevation of CVP in cats leads only to a transient increase of brain tissue pressure. One must conclude that there is sufficient venous system interposed between the two compartments to ensure that at least moderate pressures (up to about 60 cm H₂O, which represents the pressure generated by the ventilator to deliver the pre-set tidal volume plus added PEEP) are not directly transmitted. Most probably the enormously distensible splanchnic system absorbs most of the increased pressure. Since cardiac output and blood pressure were maintained, it may be assumed that cerebral perfusion pressure was also maintained.

All the patients in this series had normal cardiac function and no adverse effects from raised CVP. It is possible that prolonged elevation of CVP in patients with incipient heart failure may have a deleterious effect greater than the beneficial effect seen by improved oxygenation. It is more probable, however, that even small amounts of PEEP (such as 5 cm H₂O) would quickly result in hypotension in such patients, necessitating the reduction of PEEP to 1 to 2 cm H₂O, or even to its abandonment and institution of other therapeutic modalities to improve cardiac action.

The change in intracranial pulse form at high levels of PEEP (40 cm H₂O) suggests a reduced compliance, but this was not reflected in increased ICP. This end-expiratory pressure level carries a high incidence of pneumothorax, and unless chest tubes are inserted it is not recommended. In addition, the extremely small improvement seen in arterial oxygenation at levels about 20 cm H₂O PEEP does not offset the increased risk involved.

The improvement in arterial oxygenation may act more quickly than was previously supposed to improve intracranial dynamics; this would account for the improvement in compliance seen in three previously hypoxic patients.

An earlier supposition that normal lung compliance allows easier transmission of increased end-expiratory pressure to the brain and resultant increases in ICP was not confirmed by these studies.

It is concluded that PEEP improves arterial oxygenation and within a safe therapeutic range does not increase ICP even in low compliance systems. The beneficial effects of this modality appear to be greater than the adverse effects and therefore, when indicated, with careful monitoring and cerebrospinal fluid drainage if necessary, it can be used safely in head-trauma victims.

**Acknowledgments**

The cooperation of the residents and staff of the Department of Neurological Surgery of the Albert Einstein College of Medicine in these studies is gratefully acknowledged.

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