Effects of positive end-expiratory pressure ventilation on cerebral venous pressure with head elevation in dogs

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Toung, Thomas J. K., H. Aizawa, and Richard J. Traystman. Effects of positive end-expiratory pressure ventilation on cerebral venous pressure with head elevation in dogs. J. Appl. Physiol. 88: 655–661, 2000.—Mechanical ventilation with positive end-expiratory pressure (PEEP) may prevent venous air embolism in the sitting position because cerebral venous pressure (Pcev) could be increased by the PEEP-induced increase in right atrial pressure (Pra). Whereas it is clear that there is a linear transmission of the PEEP-induced increase in Pra to Pcev while the dog is in the prone position, the mechanism of the transmission with the dog in the head-elevated position is unclear. We tested the hypothesis that a Starling resistor-type mechanism exists in the jugular veins when the head is elevated. In one group of dogs, increasing PEEP linearly increased Pcev with the dog in the prone position (head at heart level, slope = 0.851) but did not increase Pcev when the head was elevated. In another group of dogs, an external chest binder was used to produce a larger PEEP-induced increase in Pra. Further increasing Pra increased Pcev only after Pra exceeded a pressure of 19 mmHg (break pressure). This sharp inflection in the upstream (Pcev)-downstream (Pra) relationship suggests that this may be caused by a Starling resistor-type mechanism. We conclude that jugular venous collapse serves as a significant resistance in the transmission of the Pra to Pcev in the head-elevated position.

right atrial pressure; jugular venous pressure; intracranial pressure; Starling resistor; chest binder

MECHANICAL VENTILATION with positive end-expiratory pressure (PEEP) has been advocated as a means of preventing venous air embolism in the sitting position (7, 17). It is hypothesized that PEEP may decrease the incidence of venous air embolism by increasing right atrial pressure (Pra), which, in turn, increases cerebral venous pressure (Pcev) to a level greater than atmospheric pressure. Whereas PEEP may increase Pcev in prone dogs positioned with the head at heart level (14), PEEP up to 20 cmH2O has little effect on Pcev with the head in an elevated position (16). This attenuated effect of PEEP on Pcev in subjects in the head-elevated position could be due to the presence of jugular venous valves, distensible veins in the neck that disperse the effect of PEEP, or collapsible venous channels (Starling resistor effects) that offset the increase in Pra. Previously, our laboratory (16) hypothesized that PEEP may increase Pcev if PEEP increased to a level that caused Pra to exceed the hydrostatic gradient between the right atrium and cerebral veins created by head elevation.

This study was undertaken to determine the Starling resistor effect in the transmission of increased Pra to Pcev in the head-elevated position. We utilized PEEP alone and in combination with external chest compression to further increase PEEP-induced Pra to characterize the relationship between Pra and Pcev in the head-elevated position.

MATERIALS AND METHODS

The study was conducted in accordance with the National Institutes of Health guidelines for the use of experimental animals, and the protocols were approved by the Animal Care and Use Committee of The Johns Hopkins Medical Institutions.

Preparation

Twenty-four dogs were studied. Adult male beagle dogs (9–12 kg) were anesthetized with pentobarbital sodium (30 mg/kg iv) and maintained with intermittent intravenous administration of fentanyl (1–2 µg·kg⁻¹·h⁻¹). All dogs were paralyzed with pancuronium bromide (0.2 mg/kg iv), and their lungs were mechanically ventilated via an endotracheal tube (tidal volume 10 ml/kg). All animals were given supplemental anesthesia (pentobarbital and fentanyl) in response to hypertension and tachycardia. End-tidal CO2 was maintained between 30 and 35 mmHg throughout the experiment by adjusting respiratory rate. Arterial blood gases were measured with a self-calibrating Radiometer electrode system (ABL 3; Copenhagen, Denmark). Supplemental oxygen was administered to maintain arterial PO2 between 100 and 150 Torr. Esophageal temperature was maintained at 38 ± 1°C with a heat lamp and heating pad.

With the dog in the supine position, catheters were placed in the femoral arteries for monitoring mean arterial blood pressure (MABP) and obtaining blood-gas samples. A 7-Fr catheter was advanced through the right femoral vein and positioned at the right atrium for measurement of Pra. A catheter was placed via the other femoral vein for fluid administration. In one group of 12 dogs, jugular venous pressure was measured at two different locations (upper jugular (Puj) and lower jugular (Plj)). In these dogs two 4.0-Fr (18-gauge) catheters were inserted nonocclusively into the midportion of the left jugular vein. One catheter was directed 5 cm cephalad for measurement of Puj (tip at angle of mandible, −20 cm from the right atrium), and the other catheter was directed 5 cm caudally for measurement of Plj (tip at the level of omocervical vein, −10 cm from the right atrium) (Fig. 1).

The dog was then turned prone, and the head was stabilized in a head holder with the external auditory meatus positioned at right atrial level. The superior sagittal sinus was exposed at the level of the coronal suture and cannulated in the direction of the confluence of sinuses. This catheter was
and 80 mmHg, and then after addition of 17 and 34 cmH2O
chest binder uninflated, with the chest binder inflated to 40
Pcev, CSFP, and Pra were first recorded at PEEP
prone position with the head at heart level, measurements of
explanation.

49x172]. Measurements are vertical distances
506.172] II

49x322] auditory meatus to 5th intercostal space at the lower one-
49x372] head elevated 25 cm above the right atrium (from external
49x392] PEPP on external Puj and Plj and Pcev in the prone position

49x606] Part I. All transducers were zeroed at the
49x615] jugular venous pressure (Plj) venous
49x624] jugular venous pressure (Puj) and upper
49x651] cerebrospinal fluid pressure (CSFP). In
49x678] position, with its head elevated 25 cm 

49x87] Experimental Protocol

Part I. We investigated the effects of increasing Pra with
PEEP on external Puj and Plj and Pcev in the prone position
with the head at the level of the right atrium and with the
head elevated 25 cm above the right atrium (from external
auditory meatus to 5th intercostal space at the lower one-
third of dorsoventral distance). With the dog in the prone
position, with the head at heart level or in the head-elevated
position, all transducers were zeroed at the level of the right
atrium. Measurements of Pcev, Puj, Plj, and Pra were
recorded at PEEP = 0 and then after the addition of 5, 10, 15,
20, 25, 30, and 35 cmH2O by placing the expiratory tubing
from the endotracheal tube under varying depths of water.
Each PEEP increment lasted for 1–2 min; PEEP = 0 was
established for 1–2 min between each increment to determine
the change between PEEP = 0 and each increment of PEEP.
After obtaining all data, we repeated the protocol with the dog
positioned with its head elevated 25 cm above the level of the
right atrium with the head supported by a head holder.

Part II. We investigated the effects of further increasing
Pra during PEEP on Pcev. Only two levels of PEEP, 17 and 34
cmH2O (12.5 or 25 mmHg), were studied. With the dog in the
prone position with the head at heart level, measurements of
Pcev, CSFP, and Pra were first recorded at PEEP = 0, with
chest binder uninflated, with the chest binder inflated to 40
and 80 mmHg, and then after addition of 17 and 34 cmH2O
PEEP with the chest binder unbound and bound. Similar
studies were repeated with the head elevated 25 cm above the
heart level as in part I. All transducers were zeroed at the
level of the right atrium.

RESULTS

Part I

Throughout each protocol, blood-gas values were
maintained within physiological limits. At baseline,
arterial Pco2 was 35 ± 4 Torr, arterial Po2 was 120 ± 15
Torr, and pH was 7.395 ± 0.093, and these values were
unchanged throughout the protocol.

At baseline, MABP was similar between the head at
heart level and the elevated positions (135 ± 6 vs.
136 ± 5 mmHg). With the head at heart level, increasing
PEEP to 20 cmH2O decreased MABP. With the head
elevated, the first significant decrease in MABP occurred
at PEEP of 25 cmH2O (Table 1). MABP continued
to fall as PEEP was elevated to 30 and 35 mmHg
with the head at heart level or when the head was in
the elevated position (Table 1).

At baseline, Pcev was higher in the head at the
heart-level position compared with the head-elevated
position, 7.6 ± 0.5 vs. 2.3 ± 0.8 mmHg (transmural).
With the head at the heart-level position, Pcev
increased progressively with the increase in PEEP, and
the rise was greatest at a PEEP of 35 cmH2O. In this
position, an increase in PEEP to only 10 cmH2O increased
Pcev (Table 1). In contrast, Pcev was not
altered by increasing PEEP, despite a similar magni-
tude in the increase of Pra by PEEP (Table 1). For each
individual animal, a linear regression was performed
between Pra and Pcev. The average slope of this
relationship in the head at the heart-level position
(slope of 0.851 with an intercept at 2.1 mmHg, r = 0.820)
was significantly greater than the slope in the
head-elevated position (slope of 0.254 with an intercept
at 21.2 mmHg, r = 0.310) (Fig. 2).

In the head at the heart-level position, there was no
difference among Pcev, Puj, Plj, and Pra. As a result of
increasing PEEP, there was a linear increase in Pcev,
Puj, and Plj in concert with the increase in Pra (Table 1; Fig.
3A). However, in the head-elevated position, the
PEEP-induced increased Pra was transmitted upward
in a decreasing order. With the sagittal sinus located at
the highest level from the heart, the pressure (Pcev)
was not significantly increased (Fig. 2B). Puj measured
at the second highest level did not significantly
increase until PEEP increased to 25 cmH2O (Pra = 11.7 ±
0.9 mmHg) (Table 1; Fig. 3B), whereas Plj, measured at
Table 1. Cerebral venous and external jugular venous pressures measured in part I

<table>
<thead>
<tr>
<th>PEEP, cmH\textsubscript{2}O</th>
<th>Head Position</th>
<th>MABP</th>
<th>Pra</th>
<th>Plj</th>
<th>Puj</th>
<th>Pcev</th>
</tr>
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<tr>
<td>0 H</td>
<td>135 ± 6</td>
<td>6.5 ± 0.5</td>
<td>6.6 ± 0.6</td>
<td>6.5 ± 0.6</td>
<td>7.6 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TM</td>
<td>136 ± 5</td>
<td>4.6 ± 0.5</td>
<td>0.6 ± 0.7</td>
<td>-0.1 ± 0.7</td>
<td>2.3 ± 0.8</td>
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</tr>
<tr>
<td>+</td>
<td>140 ± 4</td>
<td>7.3 ± 0.5</td>
<td>7.7 ± 0.7</td>
<td>7.7 ± 0.6</td>
<td>8.3 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>5 H</td>
<td>134 ± 6</td>
<td>6.0 ± 0.7</td>
<td>1.4 ± 0.5</td>
<td>0.5 ± 0.7</td>
<td>2.7 ± 0.8</td>
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</tr>
<tr>
<td>E</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TM</td>
<td>132 ± 6</td>
<td>7.5 ± 0.1</td>
<td>2.3 ± 0.7</td>
<td>0.6 ± 0.7</td>
<td>2.8 ± 0.7</td>
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<tr>
<td>+</td>
<td>9.5 ± 0.4</td>
<td>15.1 ± 0.8</td>
<td>23.7 ± 0.9</td>
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<td></td>
<td></td>
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<tr>
<td>10 H</td>
<td>134 ± 6</td>
<td>9.6 ± 0.4</td>
<td>9.4 ± 0.6</td>
<td>9.5 ± 0.5</td>
<td>9.9 ± 0.7</td>
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<tr>
<td>E</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TM</td>
<td>129 ± 6</td>
<td>9.1 ± 0.8</td>
<td>3.8 ± 0.6</td>
<td>1.0 ± 0.8</td>
<td>3.1 ± 0.8</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>11.0 ± 0.6</td>
<td>15.5 ± 0.8</td>
<td>24.0 ± 1.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 H</td>
<td>128 ± 6</td>
<td>11.1 ± 0.5</td>
<td>10.7 ± 0.7</td>
<td>11.2 ± 0.6</td>
<td>11.1 ± 0.7</td>
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<tr>
<td>E</td>
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<td></td>
</tr>
<tr>
<td>TM</td>
<td>130 ± 4</td>
<td>10.2 ± 0.9</td>
<td>5.3 ± 0.7</td>
<td>1.7 ± 0.9</td>
<td>3.1 ± 0.8</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>12.5 ± 0.5</td>
<td>16.2 ± 0.8</td>
<td>24.0 ± 1.0</td>
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<td></td>
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<tr>
<td>25 H</td>
<td>122 ± 6</td>
<td>12.2 ± 0.7</td>
<td>11.8 ± 0.8</td>
<td>11.7 ± 0.6</td>
<td>12.2 ± 0.7</td>
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<tr>
<td>E</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>TM</td>
<td>124 ± 5</td>
<td>11.7 ± 0.9</td>
<td>6.3 ± 0.8</td>
<td>2.4 ± 0.9</td>
<td>3.3 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>13.5 ± 0.6</td>
<td>16.9 ± 0.7</td>
<td>24.2 ± 1.0</td>
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<td></td>
<td></td>
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<tr>
<td>30 H</td>
<td>120 ± 6</td>
<td>12.9 ± 0.7</td>
<td>12.8 ± 0.7</td>
<td>12.7 ± 0.7</td>
<td>13.2 ± 0.8</td>
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<td>E</td>
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</tr>
<tr>
<td>TM</td>
<td>119 ± 6</td>
<td>12.5 ± 1.0</td>
<td>7.2 ± 0.9</td>
<td>2.1 ± 0.7</td>
<td>3.0 ± 0.6</td>
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<tr>
<td>+</td>
<td>14.4 ± 0.7</td>
<td>16.6 ± 0.7</td>
<td>23.9 ± 0.9</td>
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<tr>
<td>35 H</td>
<td>114 ± 5</td>
<td>13.8 ± 0.8</td>
<td>13.5 ± 0.8</td>
<td>13.4 ± 0.8</td>
<td>14.2 ± 1.0</td>
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<tr>
<td>E</td>
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<td></td>
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</tr>
<tr>
<td>TM</td>
<td>114 ± 6</td>
<td>13.2 ± 0.9</td>
<td>8.1 ± 0.8</td>
<td>2.7 ± 0.6</td>
<td>2.9 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>15.3 ± 0.8</td>
<td>17.2 ± 0.7</td>
<td>23.8 ± 1.0</td>
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</tr>
</tbody>
</table>

Values are means ± SE in mmHg, measured in 12 dogs on positive end-expiratory pressure (PEEP) in the head at heart (H) and elevated (E) position, TM, transmural pressure; +, transducers zeroed at right atrium and measured. MABP, mean systemic arterial blood pressure; Pra, right atrial pressure; Pcev, cerebral venous pressure; Puj, upper jugular venous pressure; Plj, lower jugular venous pressure. Vertical distance = Pcev – RA = 20.86 ± 0.3 mmHg. Puj – Pra = 14.46 ± 0.2; Plj – Pra = 7.19 ± 0.2.

An increase in both Pcev and CSFP was not noted until Pra was increased to 20 mmHg at a PEEP of 17 and 34 cmH\textsubscript{2}O and when combined with a chest-binder pressure of 40 and 80 mmHg. The relationship between Pcev and Pra is shown in Fig. 4D with a slope of 0.73 and \( r = 0.68 \), which was significantly different compared with Fig. 4C. In the head at the heart-level position, the relationship was not different from that of PEEP alone without the chest binder.

**DISCUSSION**

In the head at the heart-level position, application of PEEP produced a relatively linear increase in Pra and Pcev; however, in the head-elevated position, even a very high level of PEEP (35 cmH\textsubscript{2}O) did not increase Pcev, despite a similar increase in Pra. In the head-elevated position, the transmission of PEEP-induced increased Pra was in a decreasing order along the jugular vein according to the vertical distance from the right atrium. In both positions, chest-binder inflation further increased Pra, regardless of PEEP level. In the head-elevated position, only when Pra was increased to >20 mmHg by combination of PEEP and chest-binder inflation did Pcev increase.

The mechanism by which PEEP increases CSFP with the head at heart level has been well studied in animal models. With normal lung and chest wall compliances, approximately one-half of the applied airway pressure is transmitted intrathoracically (2). Huseby et al. (11) showed that PEEP increased CSFP by increasing Pra and pleural pressure. Luce et al. (14) showed that the lowest level, increased significantly at a PEEP of only 10 cmH\textsubscript{2}O (Pra = 7.5 ± 0.1 mmHg). The average vertical distances from the levels at which Pcev, Puj, and Plj were measured to the level of the right atrium were 28.1 ± 0.5, 19.7 ± 0.3, and 9.8 ± 0.9 cm, respectively.

Part II

Application of the chest binder is more effective in raising Pra than that of PEEP alone in both positions (Table 2). In the head at the heart-level position, application of the chest binder during PEEP ventilation caused a further increase in Pcev and CSFP. In the head-elevated position, with application of a chest-binder pressure at 80 mmHg alone without PEEP, Pra increased to 18.0 ± 2.0 mmHg; with this pressure, both Pcev and CSFP increased significantly. With PEEP alone, either at 17 or 34 cmH\textsubscript{2}O, Pcev was not changed (Table 2). When the relationship between Pcev and Pra was analyzed, it was similar to that obtained in the part I study, with a slope of 0.22 and \( r = 0.31 \) (Fig. 4C).
PEEP increased CSFP primarily by increasing superior vena caval pressure and decreasing cerebral venous outflow. Because there is a close correlation between CSFP and pleural pressure, they (14) concluded that the acute changes in CSFP caused by PEEP were mediated by pleural pressure. These studies indicate that pressure transmission occurs primarily through venous channels.

With the animal in the head-elevated position, our previous studies in dogs (16) and human subjects (12) demonstrated that PEEP had no effect on Pcev. The reason for this failure of PEEP to increase Pcev is unclear; however, it has been hypothesized that the presence of jugular venous valves located at the thoracic inlet may mechanically prevent transmission of the increased Pra via the jugular veins. Another possibility is that the PEEP-induced increased Pra may not be high enough to overcome the closing pressure of the collapsed jugular veins.

The evidence that transmission of intrathoracic pressure to the jugular veins is blocked by valves at the thoracic inlet comes from the studies of Fisher et al. (5) and Guerci et al. (6) in both humans and animals. Although a retrograde flow of blood was prevented by the competent jugular venous valves during sudden forceful coughing in humans (transvalvular pressure gradients of 52 mmHg), the closure of the valves should only be momentary because the outflow from the brain must continue, and these valves will remain open. Furthermore, the animal studies were carried out either in vivo static conditions or during cardiopulmonary resuscitation in fibrillated dogs. There was no flow in each condition. These conditions contrast sharply with studies of PEEP in living animals and earlier human studies with regular coughing. In the study of Huseby et al. (11) on the effects of PEEP on intracranial pressure in dogs, they found the magnitude of intracranial pressure changes depended on the changes in the PEEP-induced increase in venous pressure. No mention was made concerning the presence of jugular venous valves. In earlier human studies by Myerson et al. (15), a two- to fourfold increase in jugular venous pressure was found during regular coughing, straining, and even during loud talking. Luce et al. (14), studying the mechanism by which PEEP increased CSFP in dogs, found that CSFP could be increased immediately and parallels the rise in superior vena cava pressure. In the present study with the animal in the head-elevated position, we have further shown that Plj, measured beyond the jugular venous valves, could be elevated in parallel with PEEP-induced increases in Pra. As these studies have demonstrated that the jugular venous valves do not prevent the pressure transmission, it is likely that these jugular venous valves are inconsequential.

With the assumption that the entire increase in Pra is transmitted through the jugular venous channel, collapse of jugular veins, when the head is elevated, could be the main resistance to pressure transmission upward. One mechanism by which the surrounding pressure of a vascular system can affect blood flow is based on the vascular waterfall model of the pulmonary circulation (13). This model suggests that, if extravascular pressure exceeds intravascular pressure within a collapsible segment, there must be a point at which the surrounding pressure will equal the intravascular pressure. Under these conditions, blood flow through the vascular segment proximal to that point will be proportional to the arterial inflow pressure minus the surrounding pressure. As long as the outflow pressure remains less than surrounding pressure, the outflow pressure will have no effect on altering flow through the vascular segment. However, when outflow pressure is equal to or higher than surrounding pressure, it (outflow pressure) becomes the back pressure for flow. Similarly, in the brain, it has been demonstrated that the vein collapses when it is elevated above the heart because the surrounding pressure is greater than the pressure inside the vein, thus creating a Starling resistor effect. In a Starling resistor, defined as an easily collapsible vascular segment submitted to an external pressure, blood flow will start when upstream pressure is large enough to cause the pressure inside the collapsed segment (intravascular pressure) to exceed the external pressure (extravascular pressure) and thus open the vessels. The Starling resistor upstream pressure at which the vessels collapse is often referred to as the closing pressure. With relatively constant flow, such as in a living subject, the upstream pressure remains almost constant at this level, despite small changes in downstream pressure. Holt (9) demonstrated in dogs that, when a vein was held above heart
level, atrial pressure had to be increased by several centimeters of water before there was any rise in peripheral venous pressure. Holt (10) further showed in humans that, with the subject in the sitting position, the elevated cephalic vein partially collapsed at the point at which it entered the upper end of the thorax, and the venous pressure was not a function of Pra. Jugular veins collapse when elevated above the heart, such as in the sitting position. These veins will then behave as a Starling resistor. In part I of this study, with PEEP elevated to 35 cmH₂O, sagittal sinus pressure (Pcev), which could be referred to as the upstream pressure (as explained in the Starling resistor model), was not affected by the increase in Pra (the down-

Table 2. Cerebrospinal fluid and vascular pressure measured in part II

<table>
<thead>
<tr>
<th>PEEP, cmH₂O</th>
<th>Binder Pressure, mmHg</th>
<th>Head Position</th>
<th>MABP</th>
<th>Pra</th>
<th>Pcev</th>
<th>CSFP</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>H</td>
<td>122±5</td>
<td>3.2±0.6</td>
<td>9.6±0.8</td>
<td>10.7±0.9</td>
</tr>
<tr>
<td>(0)</td>
<td>0</td>
<td>E</td>
<td>121±5</td>
<td>3.2±1.0</td>
<td>26.6±0.6</td>
<td>26.9±1.5</td>
</tr>
<tr>
<td>0</td>
<td>40</td>
<td>H</td>
<td>130±6</td>
<td>10.0±1.4</td>
<td>12.5±1.2</td>
<td>13.9±1.2</td>
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<td>(0)</td>
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<td>15.8±1.4</td>
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<tr>
<td>(0)</td>
<td>80</td>
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<td>117±9.0</td>
<td>18±2.0</td>
<td>29.7±2.2</td>
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<td>(12.5)</td>
<td>0</td>
<td>E</td>
<td>103±6.0</td>
<td>8.7±1.0</td>
<td>25.4±0.7</td>
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<td>H</td>
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<td>(25.0)</td>
<td>0</td>
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<td>83±6.0</td>
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<td>23.9±0.6</td>
<td>29.2±1.5</td>
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<td>H</td>
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<td>13.8±1.1</td>
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<td>17.0±1.1</td>
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<tr>
<td>(12.5)</td>
<td>40</td>
<td>E</td>
<td>113±8</td>
<td>16.4±1.5</td>
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<td>32.3±2.0</td>
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<tr>
<td>80</td>
<td>E (TM) (25.0)</td>
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Values are means ± SE in mmHg, measured in 12 dogs on PEEP in the H and E positions. CSFP, cerebrospinal fluid pressure. Binder pressure is chest binder inflation pressure. Values in parentheses are TMs. All transducers were zeroed at right atrium level and measured at right atrium level.

Fig. 4. Effect of chest binding on Pcev. Data are from all animals used in part II of study. In the head at heart position, with PEEP alone (A), Pcev increased significantly with a PEEP-induced increase in Pra. With application of chest-binder pressure (B) and a further increase in Pra, there was a greater increase in Pcev. In the head-elevated position, when data were analyzed between Pcev and Pra <20 mmHg, the correlation was not significant (C; P > 0.05; r = 0.31), but when Pra was >20 mmHg (chest bound), the correlation became significant (D; P < 0.03; r = 0.68).
stream pressure) up to 13 mmHg. Jugular venous pressure, however, measured at the level of the mandible (20 cm above the right atrium and ~8 cm below the level of the sagittal sinus) increased when Pra was increased to 10 mmHg. Similarly, jugular venous pressure measured at an even lower level (Pij; 10 cm above the right atrium) increased when Pra increased to only 7.5 mmHg. These data suggest that the jugular veins collapsed at the thoracic inlet. From part I of our study, it is clear that Pcev could be elevated when Pra was increased to ≥20 mmHg by the addition of a pressurized chest binder to augment the Pra increase. These results suggest that the back pressure to overcome the closing pressure is ~20 mmHg.

In the sitting position, or head-elevated position, in humans, cerebral venous blood exits through the internal jugular veins, emissary veins, and vertebral venous plexus. Epstein et al. (4) demonstrated in monkeys in the erect position that cerebral venous blood drains primarily from the vertebral venous system with little from the jugular veins and that the proportion of outflow depended on the airway pressure. Eckenhoff (3) explained that cerebral venous outflow is a dynamic state: during inspiration, high-intrathoracic pressure causes a major proportion of blood to drain via the vertebral plexus; during inspiration, low-intrathoracic pressure causes blood to drain freely from both the jugular veins and vertebral plexus. During forced expiration, such as during heavy lifting, or when the chest is splinted, high-intrathoracic pressure stops the return of blood via the jugular veins. All cerebral venous blood then flows into the vertebral plexus and out into other venous beds. The vertebral venous plexus has been described as an enormous, valveless, thin-walled vascular bed contained within the spinal canal that courses between and parallel to the bodies of the vertebrae (1). Blood is free to flow in and out of these veins as directed by pressure gradients. Because these venous plexus do not directly connect to the superior vena cava, they are not subjected to immediate intrathoracic pressure variations. These two unique characteristics, its large capacitance and its indirect connection with the superior vena cava, may also partially explain why pressure transmission is less effective in the head-elevated position. Because the proportion of cerebral venous blood draining through the vertebral plexus was not investigated in this study, it is difficult to determine how much of the increased Pra was transmitted via this route.

Hibino and Matsmura (8), studying the effect of PEEP on Pcev in the seated dog, noted that the effect is variable. The effects of PEEP depend largely on the route of cerebral venous blood outflow: PEEP increases Pcev only when blood flows in the noncollapsed vertebral vessels, whereas, when blood flows primarily in the collapsed jugular veins, Pcev frequently decreases further. Contrary to their finding, our laboratory’s previous study (16) in dogs in the head-elevated position demonstrated that, despite the low initial Pcev (less than ~4.0 mmHg), PEEP up to 20 cmH2O (Pra = 6 mmHg) did not elevate Pcev, but compression of the jugular veins (20 mmHg) did so rapidly and significantly. This indicates that the jugular veins may have collapsed but that blood flow did not cease.

In the present study, we investigated the Starling resistor effect in determining the effect of PEEP on Pcev in dog in the head-elevated position. We demonstrated that PEEP up to 35 cmH2O had no effect on Pcev but effectively elevated jugular venous pressure (depending on the level at which the pressure was measured). When the PEEP-induced increase in Pra was further increased with a chest binder, and when it exceeded the closing pressure of the collapsed jugular veins, PEEP then increased Pcev. These findings can be interpreted, in clinical settings, to mean that PEEP may be beneficial in preventing venous air embolism for cervical spine surgery (where the site of surgery is not as highly located as the head from the heart) in the sitting position.

The results of this and previous studies from our laboratory indicate that PEEP is ineffective in raising Pcev for prophylaxis of venous air embolism in the head-elevated position. Even a very high level of PEEP (35 cmH2O) does not raise Pcev, and this level of PEEP may result in a marked reduction in systemic arterial blood pressure and cardiac output such that cerebral blood flow may be reduced. Acute elevation of Pcev may be best achieved in the clinical setting with manual compression of the jugular veins or with a carefully regulated neck-cuff occluder.

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REFERENCES