Comparison of the Effects of Positive End-expiratory Pressure and Jugular Venous Compression on Canine Cerebral Venous Pressure

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The confluent sinus pressure was measured in eight mongrel dogs in the head-up position to compare the effectiveness of positive end-expiratory pressure (PEEP) and jugular venous compression in increasing cerebral venous pressure. When the head was elevated 30 cm above the heart, confluent sinus pressure decreased from 9.6 ± 1.8 (mean ± SEM) to -5.3 ± 0.5 mmHg. At constant arterial carbon dioxide tension (PaCO₂ = 28 ± 2 mmHg), PEEP (20 cmH₂O) did not increase cerebral venous pressure. However, when the jugular veins were compressed with a neck tourniquet with pressures of 20-140 mmHg, cerebral venous pressure increased rapidly. When neck tourniquet pressure was maintained at 40 mmHg, confluent sinus pressure in all dogs was increased and sustained at 2.4 ± 0.8 mmHg. Carotid artery pressure measured distal to the tourniquet was not altered. The efficacy of extrathoracic venous pressure elevation (neck tourniquet) is greater than intrathoracic (PEEP), and this may relate to the Starling resistor effects of neck veins and the presence of jugular venous valves. We conclude that prophylactic use of PEEP in the prevention of air embolism during the sitting position may not be as effective as jugular venous compression. (Key words: Anesthesia; neurosurgical. Embolism: air. Position: sitting. Veins: jugular. Ventilation: PEEP.)

NEUROSURGICAL PROCEDURES that are performed with the patient in the sitting position present the risk of venous air embolism with an incidence reported as high as 50%.1 Subatmospheric venous blood pressure at the level of the surgical wound is the underlying cause of venous air embolism in this position. There are two clinical maneuvers designed to artificially increase venous pressure in the head in this setting: 1) indirectly by the use of either positive end-expiratory pressure (PEEP)2 or the anti-gravity suit (G-suit or MAST)3,4 and 2) directly by jugular venous compression.2 Increase in central venous pressure with PEEP has led to the belief that venous pressure in the head also will increase. Direct measurement of cerebral venous pressure in humans during the sitting position with PEEP has not been reported. In the present study, confluent sinus pressure was measured in mongrel dogs maintained in a head-up position during the application of both PEEP and neck tourniquet in order to evaluate the effectiveness of these two maneuvers in increasing cerebral venous pressure.

Methods

GENERAL PROCEDURES

Eight mongrel dogs (16-21 kg) were anesthetized with sodium pentobarbital (30 mg/kg, iv), paralyzed with pancuronium (0.1 mg/kg), endotracheally intubated, and mechanically ventilated (Harvard® respirator model 607). Tidal volume and respiratory rate were adjusted to maintain a constant arterial carbon dioxide tension (PaCO₂) of 28 ± 2 mmHg (mean ± SEM). Both femoral veins were cannulated, one for right atrial pressure (Pra) monitoring and one for fluid infusion. A femoral artery catheter was placed for mean systemic arterial pressure (Psm) measurement. Mean common carotid artery pressure (Pcar) was measured via a #22 angio catheter at C7 level placed cephalad in the right common carotid artery. Pleural pressure (Ppl) was monitored with a metal-tip catheter with multiple holes inserted at the right midchest through the sixth intercostal space. Airway pressure (Paw) was measured at the proximal end of the endotracheal tube. Cerebral venous sinus pressure (Pcv) was measured at the confluence of the sinuses with a large bore cannula. Intracranial pressure was recorded via a #16 angio catheter placed into the cisterna magna (Pcm). Expiratory CO₂ concentration continuously was measured with a Beckman® Infrared CO₂ Analyzer.

The cerebral physiologic effects of elevating the head above the heart was evaluated in each dog. Control measurements were obtained with the dog in the prone position and head lowered to the level of the heart. Following this baseline measurement with positive-pressure ventilation and zero expiratory pressure, the head was positioned in the head holder with the dorsum of the cranial vault 30 cm above the level of the right heart. Transducers for Pcm, Pcv, and Pcv were placed and zeroed at the level of external auditory meatus; Pra, Ppl, Paw, and Pcm were at the right heart level and zeroed.
was sufficient for physiologic changes to occur and to adjust the ventilatory rate to maintain end-tidal CO\textsubscript{2} constant. Blood gases were analyzed at 5 and 10 min during each PEEP level. P\textsubscript{a} was maintained at least 70 mmHg during the application of PEEP by infusion of 5% glucose in lactated Ringer's solution to ensure that cerebral perfusion pressure was maintained within the normal cerebral autoregulatory range.

**Neck Tourniquet:** The tourniquet was inflated to 20, 30, 40 mmHg and then in increments of 20 mmHg up to 140 mmHg. Each increment was maintained for 2 min. The cerebral physiologic effects of prolonged jugular vein compression was evaluated in four additional dogs. Following the completion of the tourniquet study, tourniquet pressure of 40 mmHg was applied and maintained for 20 min. The tourniquet was placed at the lower neck (C5) level. Arterial blood gases were determined periodically to maintain normal acid–base status and constant P\textsubscript{a}CO\textsubscript{2} as in the PEEP study.

All results were analyzed by ANOVA with contrast comparisons and the control values were compared with each subsequent experimental value.

### Results

#### EFFECT OF ELEVATING THE HEAD

30 cm Above the Heart

Upon raising the head 30 cm above the heart, P\textsubscript{a} decreased sharply from a mean of 4.5 ± 0.8 (mean ± SEM) to −5.3 ± 0.5 mmHg. P\textsubscript{a} decreased from 9.6 ± 1.8 to −0.3 ± 0.4 mmHg. Within 5 min, both P\textsubscript{a} and P\textsubscript{a} stabilized at 1.6 ± 0.4 and −4.3 ± 0.4 mmHg, respectively. There was no significant change in P\textsubscript{a} or P\textsubscript{a}, which was similar to P\textsubscript{a} during the prone position, became an average of 20 mmHg lower than P\textsubscript{a} after the elevation of the head, accurately reflecting the hydrostatic gradient between the transducers.

### EXPERIMENTAL PROTOCOLS

**PEEP:** PEEP was applied sequentially from control (0 cmH\textsubscript{2}O) in 5-cm increments up to 20 cmH\textsubscript{2}O and maintained for 10 min at each level. This 10-min period

#### PEEP Study

P\textsubscript{a}CO\textsubscript{2} was constant before and after application of PEEP, and there was no significant change in P\textsubscript{a}CO\textsubscript{2}.

Figure 1 is a polygraph recording of all variables from one animal during the application of PEEP from 0 to 5, 10, 15, and 20 cmH\textsubscript{2}O. The recording demonstrates that, despite the marked increase of P\textsubscript{a} and P\textsubscript{a} with PEEP, P\textsubscript{a} and P\textsubscript{a} do not change. Also, no significant transient alterations in P\textsubscript{a} or P\textsubscript{a} are seen with PEEP.

Table 1 shows the mean hemodynamic responses for all animals. P\textsubscript{a} and P\textsubscript{a} increased sharply with PEEP, and the increase directly was related to the amount of PEEP applied. P\textsubscript{a} decreased to 88 ± 6 and 77 ± 7 mmHg with 15 and 20 cm PEEP, respectively. P\textsubscript{a} was unchanged as PEEP was elevated.

Figure 2 shows the mean P\textsubscript{a} and P\textsubscript{a} responses with PEEP for all animals. P\textsubscript{a} was unchanged at any level of

<table>
<thead>
<tr>
<th>PEEP (cmH\textsubscript{2}O)</th>
<th>P\textsubscript{a} (mmHg)</th>
<th>P\textsubscript{a} (mmHg)</th>
<th>P\textsubscript{a} (mmHg)</th>
<th>P\textsubscript{a} (mmHg)</th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>0.4 ± 0.3</td>
<td>−5.0 ± 0.6</td>
<td>100 ± 6</td>
<td>−2.1 ± 0.3</td>
</tr>
<tr>
<td>5</td>
<td>1.0 ± 0.6</td>
<td>−4.2 ± 0.4</td>
<td>97 ± 5</td>
<td>0.8 ± 0.7*</td>
</tr>
<tr>
<td>10</td>
<td>5.0 ± 0.6*</td>
<td>−5.1 ± 0.6</td>
<td>96 ± 6</td>
<td>3.3 ± 0.7*</td>
</tr>
<tr>
<td>15</td>
<td>5.0 ± 0.7*</td>
<td>−5.6 ± 0.7</td>
<td>88 ± 6*</td>
<td>6.4 ± 0.7*</td>
</tr>
<tr>
<td>20</td>
<td>5.6 ± 0.4*</td>
<td>−5.5 ± 0.9</td>
<td>77 ± 7*</td>
<td>8.5 ± 0.4*</td>
</tr>
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Each value represents the mean ± SEM of eight animals. P\textsubscript{a} was measured at the level of the external auditory meatus, 30 cm above the level of the right atrium.

* = P < 0.05.
PEEP, whereas $P_{cv}$ was decreased slightly at 15 and 20 cmH$_2$O PEEP.

**Tourniquet Study**

There were no significant changes in blood gases, $P_{aw}$, $P_{raf}$, $P_{ra}$, or $P_{en}$ with neck tourniquet pressures up to 140 mmHg. Figure 3 shows the effects of neck tourniquet pressure increases on $P_{cv}$ and $P_{raf}$. Application of neck tourniquet rapidly increased $P_{raf}$ as tourniquet pressure increased. $P_{raf}$ returned towards the control value when neck tourniquet pressure was released. $P_{cv}$ also increased significantly as tourniquet pressure increased and returned to control when pressure was released.

With a sustained application of tourniquet pressure to 40 mmHg for 20 min, $P_{cv}$ was maintained above atmospheric pressure and remained stable throughout the entire period (Figure 4). After the initial increase, $P_{raf}$ was similarly stable. When the tourniquet pressure was released, both $P_{cv}$ and $P_{raf}$ immediately returned to control levels. Hemodynamic measurements and blood gas values did not change significantly during the period of neck tourniquet pressure.

**Discussion**

The suctioning effect created by the subatmospheric venous pressure at the operative site causes air to move into the open vessels. Subatmospheric venous pressure can occur whenever the operative site is higher than the heart. To avoid venous air embolism, the venous pressure at the surgical site must be increased to at least atmospheric pressure. It generally is believed that this can be accomplished by the application of PEEP. While PEEP of 10 to 15 cmH$_2$O has been recommended during the sitting posterior fossa craniotomy for the prevention of venous air embolism, $^{6}$ emboli still have been noticed despite use of this level of PEEP. $^{6,7}$ Thus, it appears that PEEP does not prevent air from entering the open vessels. In addition, PEEP may increase right atrial pressure and thereby increase the risk of a paradoxical embolus via a functionally patent foramen ovale.

The present study demonstrated that PEEP had little or no effect on $P_{cv}$, despite marked elevation of $P_{ra}$ and $P_{pl}$. This finding was unexpected because it has been thought that the increase in $P_{ra}$ would increase venous pressure throughout all parts of the body, including the cranial venous sinuses. This is based on the assumption that increased $P_{ra}$ or $P_{pl}$ readily is transmitted to the cranium through venous channels. According to hydrodynamic principles, $^{8}$ this phenomenon would occur in a rigid pipe system. However, this is not necessarily applicable to the cranial and cervical venous systems for two reasons. First, since the collecting jugular veins are flexible, the veins would collapse when the head is elevated above the heart and the veins thus would act as a Starling resistor. $^{9}$ A Starling resistor may be described as a thin-walled collapsible tube that traverses a closed chamber in which the pressure in that chamber may be higher, lower, or equal to the pressure within the tube. This is probably why the decrease in $P_{cv}$ is smaller than the hydrostatic gradient when the head is elevated 30 cm above the heart. Second, the presence of valves in the jugular veins disrupts the pressure transmission mechanism. The clinical significance of jugular venous valves in humans and dogs recently was discussed by Fisher et al. $^{10}$ They found that valves located at the thoracic inlet prevent retrograde flow of venous blood during a sudden and/or sustained increases in intrathoracic pressure, such as coughing and the Valsalva maneuver.

![](image1)

**Fig. 2.** Effects of PEEP on cerebral venous ($P_{cv}$) and cerebral spinal fluid ($P_{csf}$) pressures in the head elevated position (30 cm above the heart).

![](image2)

**Fig. 3.** Effects of neck vein compression with tourniquet on cerebral venous ($P_{cv}$) and cerebral spinal fluid ($P_{csf}$) pressures in the head elevated position (30 cm above the heart). Note both $P_{cv}$ and $P_{csf}$ increased significantly ($P < 0.05$) with the application of tourniquet pressure. $^* = P < 0.05$. 

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they demonstrated a static transvalvular pressure gradient of as high as 80 mmHg in a competent valve. When the head is elevated above the heart, the combination of two effects, i.e., the Starling resistor effect of the neck veins and the obstructing effect of jugular venous valves, make transmission of pressure unlikely to be simple and direct.

It can be argued that when the increase in $P_{SA}$ is sustained, as might occur with PEEP, it should limit or impede cerebral venous outflow and therefore PEEP eventually should increase cerebral venous pressure. This may occur under three conditions: first, if the head is maintained at the same level with the right atrium (Starling resistor effect does not exist)\(^\text{11}\), second, if the increase in $P_{SA}$ exceeds the hydrostatic gradient and the venous valves cannot function properly, and three, if the heart fails and no output ensues. However, in the sitting position, using a clinically applicable range of PEEP and in an otherwise healthy subject, none of these conditions exist. Furthermore, if the impedance to cerebral venous outflow does occur, the increase in cerebral blood volume will occur slowly over time and this will be reflected by a gradual increase in intracranial pressure. In our study, no change in $P_{sf}$ and $P_{cv}$ were observed with over 10-min application of various levels of PEEP.

Increasing cerebral venous pressure by compression of the jugular veins above the valves appears to be more logical, producing a more direct effect. Control of the pressure by a lightly inflated collar or a thin rubber tourniquet around the neck has been suggested by Hewer and Logue.\(^2\) Digital compression of the jugular veins has since become one of the first steps recommended for management of air embolism in an emergency situation. In this study, using a neck tourniquet, $P_{cv}$ in each dog increased rapidly above atmospheric pressure as the cuff was inflated to 40 mmHg. Continuous compression of the jugular veins at this pressure for 20 min did not produce any apparent irreversible physiologic effects as evidenced by an immediate return of $P_{cv}$ and $P_{sf}$ to pre-inflation levels following the release of compression. During compression, the increase in $P_{cv}$ was sustained and stable. Cerebral inflow pressure measured as common carotid arterial pressure distal to the tourniquet was not altered, and the systemic artery pressure remained unchanged. Because the increase in $P_{cv}$ correlated well with the applied neck tourniquet pressure (up to 60 mmHg, r = 0.979), a lower tourniquet pressure could be used if $P_{cv}$ is less subatmospheric, i.e., when the head is less elevated. Further studies on the correlation between the pressure gradient ($P_{cv}$ to $P_{RA}$) and head position are needed to determine the proper amount of tourniquet pressure required to increase $P_{cv}$ to above atmospheric levels.

The results of this study indicate that in the experimental setting, PEEP up to 20 cmH\(_2\)O does not increase cerebral venous pressure above atmospheric pressure, but compression of the jugular veins is effective in increasing cerebral venous pressure. These data suggest that studies should be conducted comparing the safety and efficacy of jugular compression and PEEP in the prevention of venous air embolism in patients undergoing craniotomies or laminectomies in the sitting position.

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References