

# Cerebral Venous Sinus Pressure in Seated Dogs: Impact of PEEP, Cervical Venous Compression, and Abdominal Compression

Haruo Hibino, M.D.,\* and Masaji Matsuura, M.D.†

The authors studied the effects of positive end-expiratory pressure (10 cmH<sub>2</sub>O PEEP), abdominal compression, and neck compression on dural venous sinus pressure (VSP) in seated dogs. Abdominal compression increased the central venous pressure (CVP) as well as both the systemic arterial pressure and the cardiac output and thus may offer a useful substitute for an antigravity suit. Except when CVP was greater than 8 mmHg, there was little or no correlation between CVP and VSP. Moreover, each method increased VSP, but this effect was closely related to VSP prior to application of the method (pre-VSP). On comparing the VSP changes in relation to the pre-VSP levels when they were either above or below -1.0 mmHg, significant differences were noted in VSP increases, *i.e.*,  $-0.4 \pm 1.3$  (mean  $\pm$  SEM) and  $4.3 \pm 1.2$  mmHg by PEEP,  $1.9 \pm 0.3$  and  $6.4 \pm 0.4$  mmHg by abdominal compression, and  $10.2 \pm 1.3$  and  $1.5 \pm 0.5$  mmHg by neck compression, respectively. This indicates that PEEP and abdominal compression were more effective in increasing relatively highly negative pre-VSP ( $< -1.0$  mmHg), while neck compression greatly increased pre-VSP when it was at or above a slightly negative pressure ( $-1.0$  mmHg). The authors conclude that a single application of any one of these three methods during sitting-position surgery may not be effective in increasing cerebral dural sinus pressure. (Key words: Anesthesia; neurosurgical. Embolism: air. Position: sitting. Veins: dural sinus; venous pressure.)

VENOUS AIR EMBOLISM occurring during neurosurgery with the patient in a sitting position can result in serious complications.<sup>1</sup> For the diagnosis of air embolism, an air infusion of as little as  $0.02 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  can be detected in dogs with the use of a precordial Doppler ultrasound monitor.<sup>2</sup> However, for the prevention of complications due to air embolism, in addition to early detection of air inflow, the abolition of further inflow is necessary.

Air embolism occurs when the pressure within non-collapsible veins, such as the diploic veins and dural venous sinuses, is decreased below atmospheric pressure. Positive end-expiratory pressure (PEEP),<sup>3</sup> an antigravity suit (G suit),<sup>4</sup> and neck compression<sup>5</sup> are among three methods proposed as prophylaxis.<sup>6</sup> PEEP and the G suit increase the intracranial venous pressure by increasing the central venous pressure. In the case of neck compression this is achieved by collapsing the cervical

veins. In dogs, the effect of a G suit, *i.e.*, increase in the central venous pressure without reducing the arterial pressure and cardiac output, can be achieved by abdominal compression. The purpose of this study was to compare the effects of PEEP, neck compression, and abdominal compression (as a substitute for the G suit) on the dural venous sinus pressure, using dogs.

## Materials and Methods

Twenty adult mongrel dogs of either sex, weighing 7.5-12.0 kg were used. Under anesthesia with ketamine (100 mg, im), the femoral vein was cannulated and lactated Ringer's solution containing 0.1% ketamine was infused at a rate of  $8 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ . After endotracheal intubation, the dogs were paralyzed with pancuronium (an initial dose of 1.0 mg followed by 0.5 mg at intervals of 45 min) and ventilated with air with the use of an animal ventilator (R50, AIKA) at a tidal volume of 20 ml/kg and a respiratory rate of 18/min. The femoral artery was cannulated for measurement of the mean systemic arterial pressure (MAP) and heart rate (HR) from their waves. A 5 Fr Swan-Ganz® catheter (Edwards Laboratories) was passed into the pulmonary artery from the femoral vein for measurement of the central venous pressure (CVP), mean pulmonary arterial pressure (MPAP), pulmonary capillary wedge pressure (PCWP), and cardiac output (CO). A small hole was opened in the parietal bone at the top of the head, through which a catheter was inserted into the superior sagittal sinus for measurement of the venous sinus pressure (VSP). All the pressures were monitored continuously with the use of pressure transducers (P231D, Gould) and an eight-channel recorder (RM6000, Nihon Koden). CO was measured by the thermodilution method (EW-90, Erma Optical Works) and expressed as the mean of three repeated measurements.

The operated dogs were seated upright at 90 degrees and their head fastened with a pin holder. After confirmation of a near steady state in hemodynamics, measurements were made of HR, MAP, CVP, MPAP, PCWP, VSP, CO, and oxygen and carbon dioxide tensions of arterial and dural venous sinus blood ( $\text{PaO}_2$ ,  $\text{PaCO}_2$ ,  $\text{PvsO}_2$  and  $\text{PvsCO}_2$ ) (prevalues). PEEP, abdominal compression, or neck compression were then applied randomly, and the above variables were measured at 20

\* Instructor in Anesthesiology.

† Assistant Professor of Anesthesiology.

Received from the Department of Anesthesiology, Nagoya University School of Medicine, 65 Tsuruma-cho, Showa-ku, Nagoya, Aichi 466, Japan. Accepted for publication March 26, 1985.

Address reprint requests to Dr. Hibino.

TABLE 1. Effects of Positive End-expiratory Pressure (PEEP), Abdominal Compression, and Neck Compression on Hemodynamics and Blood Gases (mean  $\pm$  SEM)

	PEEP, 10 cmH <sub>2</sub> O (n = 12)		Abdominal Compression (n = 20)		Neck Compression (n = 14)	
	Pre-	Post-	Pre-	Post-	Pre-	Post-
HR (beats/min)	196 $\pm$ 9	193 $\pm$ 9	188 $\pm$ 6	169 $\pm$ 6*	184 $\pm$ 8	177 $\pm$ 9*
MAP (mmHg)	113 $\pm$ 6	88 $\pm$ 10*	124 $\pm$ 6	151 $\pm$ 6*	125 $\pm$ 8	133 $\pm$ 7*
CVP (mmHg)	-4.7 $\pm$ 1.0	-0.4 $\pm$ 0.8*	-4.8 $\pm$ 0.7	1.6 $\pm$ 1.1*	-2.9 $\pm$ 1.3	-2.8 $\pm$ 1.4
MPAP (mmHg)	7.3 $\pm$ 1.0	14.7 $\pm$ 0.9*	7.5 $\pm$ 0.6	13.5 $\pm$ 1.2*	9.3 $\pm$ 1.0	10.1 $\pm$ 1.2
PCWP (mmHg)	-1.3 $\pm$ 0.6	3.2 $\pm$ 0.7*	-0.6 $\pm$ 0.6	7.2 $\pm$ 0.8*	1.2 $\pm$ 1.0	1.0 $\pm$ 0.9
VSP (mmHg)	-4.9 $\pm$ 2.3	-2.6 $\pm$ 1.8	-5.8 $\pm$ 1.4	-0.7 $\pm$ 1.0*	-2.8 $\pm$ 2.1	2.9 $\pm$ 3.2*
CO (ml $\cdot$ kg <sup>-1</sup> $\cdot$ min <sup>-1</sup> )	79.1 $\pm$ 6.1	51.1 $\pm$ 4.6*	54.2 $\pm$ 4.2	68.4 $\pm$ 4.2*	66.4 $\pm$ 4.3	58.4 $\pm$ 3.2*
Pa <sub>O<sub>2</sub></sub> (mmHg)	113 $\pm$ 3	100 $\pm$ 3*	106 $\pm$ 2	96 $\pm$ 2*	106 $\pm$ 4	104 $\pm$ 3
Pa <sub>CO<sub>2</sub></sub> (mmHg)	34 $\pm$ 1	38 $\pm$ 2*	34 $\pm$ 1	39 $\pm$ 1*	37 $\pm$ 2	36 $\pm$ 2*
Pvs <sub>O<sub>2</sub></sub> (mmHg)	38 $\pm$ 2	37 $\pm$ 2	35 $\pm$ 1	40 $\pm$ 2*	39 $\pm$ 2	37 $\pm$ 2*
Pvs <sub>CO<sub>2</sub></sub> (mmHg)	47 $\pm$ 2	53 $\pm$ 2*	49 $\pm$ 1	52 $\pm$ 1*	51 $\pm$ 2	51 $\pm$ 1

Abbreviations: HR = heart rate; MAP = mean systemic arterial pressure; CVP = central venous pressure; MPAP = mean pulmonary arterial pressure; PCWP = pulmonary capillary wedge pressure; VSP = venous sinus pressure; CO = cardiac output; Pa<sub>O<sub>2</sub></sub> and Pa<sub>CO<sub>2</sub></sub> = ar-

terial oxygen and carbon dioxide tensions; Pvs<sub>O<sub>2</sub></sub> and Pvs<sub>CO<sub>2</sub></sub> = oxygen and carbon dioxide tensions of venous sinus blood.

\* *P* < 0.05 compared with prevalue.

min after the pressure application (postvalues). When the vital signs were stable after release of the pressure, the two remaining methods were studied in random sequence. As PCWP and/or CO were not measurable in some dogs with PEEP or abdominal compression, these dogs were excluded from the cases of PEEP or abdominal compression. The midpoint of the long axis of the sternum was used as the zero point for MAP, CVP, MPAP, and PCWP. As the zero point for VSP, the point of puncture site was used. The value for each pressure at end-expiration was chosen from electrically measured values. Blood gases were analyzed with a blood gas analyzer (ABL2, Radiometer). PEEP was applied at 10 cmH<sub>2</sub>O. For abdominal compression, the abdomen was compressed with a small cushion strapped to the animal at the pressure at which MAP was not reduced and CVP was increased to its maximum. For neck compression, compression pads were placed in the anterior and lateral regions of the neck bilaterally, and the neck was compressed manually so as to obtain the maximum increase in VSP. After the end of the above experiment, using nine of the 20 animals, CVP was varied by adjusting PEEP or abdominal compression in order to examine the relationship between CVP and VSP.

The data are expressed as mean  $\pm$  SEM. Student's paired *t* test was used to compare prevalues and postvalues. The comparison of the VSP changes in relation to pre-VSP was tested by the unpaired *t* test. The correlation coefficient and its 95% confidence interval were obtained for the relationship between CVP and VSP. Linear regression curves between varied CVP and VSP with nine dogs were determined with the use of the least-squares method. Differences were considered significant for *P* < 0.05.

## Results

Table 1 summarizes the results with the three prophylactic methods. Abdominal and neck compression demonstrated almost equal effects on VSP. PEEP tended to increase VSP but not significantly. CVP, MPAP, and PCWP were increased by PEEP and abdominal compression. MAP and CO were decreased by PEEP but increased by abdominal compression. Neck compression was associated with significant changes in HR, MAP, and CO. PEEP and abdominal compression produced a slight decrease in Pa<sub>O<sub>2</sub></sub> and an increase in Pa<sub>CO<sub>2</sub></sub>. With PEEP and abdominal compression, Pvs<sub>CO<sub>2</sub></sub> increased with a concurrent increase in Pa<sub>CO<sub>2</sub></sub>. Pvs<sub>O<sub>2</sub></sub> was significantly increased by abdominal compression and reduced by neck compression.

CVP and VSP were positively correlated, though very weakly, with a correlation coefficient of just 0.266 (the 95% confidence interval was 0.120 to 0.400). Since the VSP values were very scattered by CVP values less than 8 mmHg, it was impossible to estimate VSP from CVP (fig. 1).

The effects of the three methods were related to the pre-VSP levels (fig. 2). Neck compression increased VSP markedly when pre-VSP  $\geq$  -1.0 mmHg but much less so when pre-VSP < -5.0 mmHg. Abdominal compression increased VSP markedly when pre-VSP < -5.0 mmHg but less so when pre-VSP > -1.0 mmHg. With PEEP, VSP was slightly increased when pre-VSP < 0.0 mmHg but was frequently reduced with pre-VSP > 0.0 mmHg. On comparing the VSP changes in relation to pre-VSP values greater or less than -1.0 mmHg, there were significant differences in the effects on VSP increases among the three methods (table 2).

At highly negative initial VSP values, the increases in

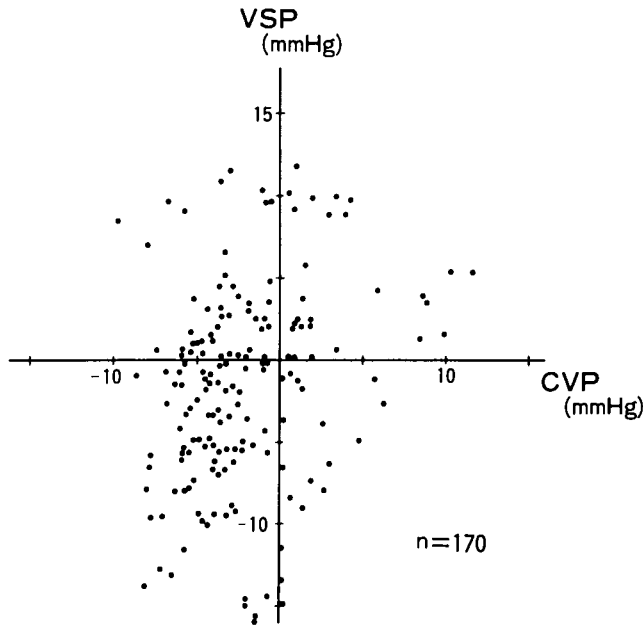


FIG. 1. Relationship between central venous pressure (CVP) and venous sinus pressure (VSP) in seated dogs. Values by neck compression are not plotted.

VSP almost corresponded to those in CVP. However, when initial VSP was greater than atmospheric pressure, VSP underwent little change, despite CVP increases (fig. 3). Thus, VSP did not, in many cases, respond to the changes in CVP.

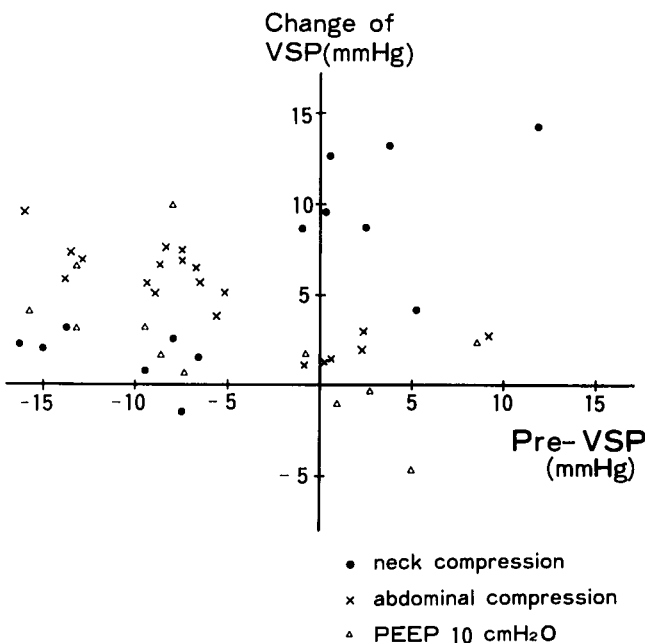


FIG. 2. Relationship between venous sinus pressures (VSP) before application of each prophylactic method and the degree of change during application.

TABLE 2. Changes in Venous Sinus Pressure (VSP) in Relation to Pre-VSP (mean  $\pm$  SEM and numbers of dogs)

	Changes in VSP	
	Pre-VSP $\geq$ -1.0 mmHg	Pre-VSP < -1.0 mmHg
PEEP, 10 cmH <sub>2</sub> O	-0.4 $\pm$ 1.3 (5)	4.3 $\pm$ 1.2 (7)*
Abdominal compression	1.9 $\pm$ 0.3 (6)	6.4 $\pm$ 0.4 (14)*
Neck compression	10.2 $\pm$ 1.3 (7)	1.5 $\pm$ 0.5 (7)*

Abbreviation: PEEP = positive end-expiratory pressure.

\*  $P < 0.05$  compared with the value at pre-VSP  $\geq$  -1.0 mmHg.

**Discussion**

The results of the present study indicate that VSP cannot be estimated from CVP. Furthermore, VSP can be increased effectively by methods that increase CVP (PEEP or abdominal compression) when the initial VSP is highly negative and by collapsing the cervical veins (neck compression) when the initial VSP is only slightly negative or positive. These results suggest that venous blood flow from the head flows in two hemodynamically different venous channels, *i.e.*, collapsed and noncollapsed vessels.

In dogs, venous blood from the brain and cranial bone flows into the right atrium via the external jugular

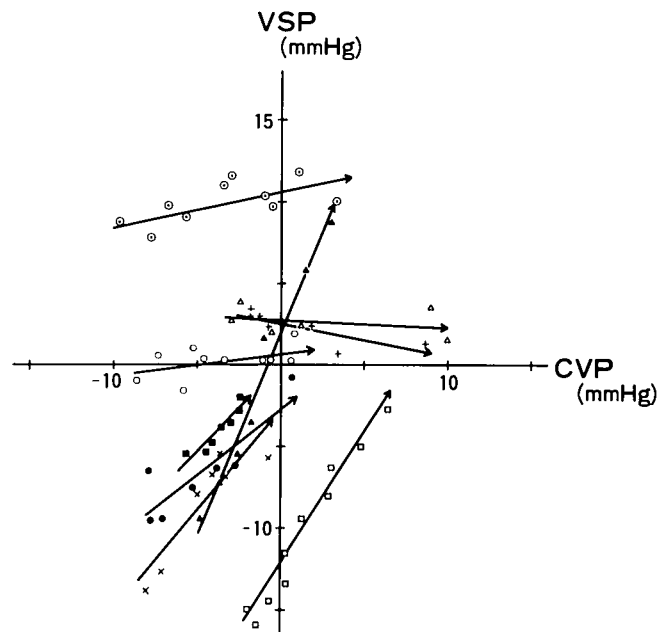
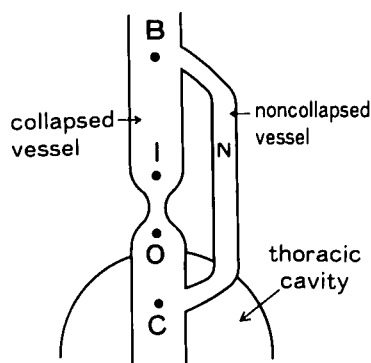


FIG. 3. Relationship between central venous pressure (CVP) and venous sinus pressure (VSP) in nine dogs. VSP in response to varied CVP by PEEP or abdominal compression are plotted and shown in linear regression curves. The left end of the curve shows the approximate initial value prior to the CVP change. VSP in response to varied CVP changed from the left end of the curve in the direction indicated by an arrow.

FIG. 4. Flow model of venous blood from the head via collapsed (BIOC) and noncollapsed (BNC) vessels. These vessels are arranged vertically.



pressure (cmH<sub>2</sub>O) at C : P<sub>C</sub>  
 at O : P<sub>O</sub>  
 at I : P<sub>I</sub>  
 at B : P<sub>B</sub>  
 flow resistance (cmH<sub>2</sub>O·min·ml<sup>-1</sup>) between  
 B and I : R<sub>C</sub>  
 B and C via N : R<sub>N</sub>  
 flow (ml/min) at C : Q  
 distance (cm) between  
 B and I : h<sub>1</sub>  
 B and C : h<sub>2</sub>  
 specific gravity of venous blood : ρ

vein, internal jugular vein, vertebral vein, and vertebral venous sinuses.<sup>7</sup> Veins in the relatively superficial layers of the neck collapse when the surrounding pressure exceeds the venous pressure, that is, when the transmural pressure is negative. However, veins in the deep layers such as the vertebral venous sinuses are not able to collapse, even at a negative transmural pressure.<sup>8</sup> When the dog is in the sitting position the venous pressure in the neck is usually less than atmospheric pressure because of hydrostatic pressure. Therefore, since the veins in the superficial layers of the neck collapse, the venous blood from the head flows via two types of venous channels, collapsed and noncollapsed. This also may apply to humans in the sitting position.

Figure 4 shows a simple model of the venous blood flow through collapsed and noncollapsed vessels. The blood flow in a collapsed vessel was called a "vascular waterfall" and was hemodynamically analyzed by Permutt *et al.*<sup>9,10</sup> According to their theory, the inflow pressure P<sub>I</sub> at the collapsed site equals the surrounding pressure, and the flow that streams in a collapsed vessel corresponds to the value obtained by dividing the pressure difference between points B and I by the flow resistance between these points and is thus unrelated to the outflow pressure P<sub>O</sub>. Therefore, the flow-pressure relationship in this model is expressed as follows:

$$Q = \frac{P_B + \rho h_1 - P_I}{R_C} + \frac{P_B + \rho h_2 - P_C}{R_N} \quad (1)$$

Hence,

$$P_B = \frac{1}{\frac{1}{R_C} + \frac{1}{R_N}} Q - \frac{1}{1 + \frac{R_C}{R_N}} \rho h_1 - \frac{1}{1 + \frac{R_N}{R_C}} \rho h_2 + \frac{1}{1 + \frac{R_C}{R_N}} P_I + \frac{1}{1 + \frac{R_N}{R_C}} P_C \quad (2)$$

Assuming that R<sub>C</sub> in equation (2) is infinite,

$$P_B = R_N Q - \rho h_2 + P_C \quad (3)$$

Assuming that R<sub>N</sub> in equation (2) is infinite,

$$P_B = R_C Q - \rho h_1 + P_I \quad (4)$$

When the entire blood flows in a noncollapsed vessel, the flow-pressure relationship is expressed by equation (3) where P<sub>B</sub> is unrelated to P<sub>I</sub> and increases only by the amount of P<sub>C</sub> increase. When the entire blood flows in a collapsed vessel, the flow-pressure relationship is expressed by equation (4), where P<sub>B</sub> is unrelated to P<sub>C</sub> and increases corresponding to increases in P<sub>I</sub>. Guyton and Adkins<sup>11</sup> demonstrated that increasing the intraabdominal pressure causes progressively increasing pressure in the femoral vein. When venous blood flows in both collapsed and noncollapsed vessels, the P<sub>B</sub> changes due to the changes in P<sub>I</sub> or P<sub>C</sub> are determined by the ratio of R<sub>C</sub> and R<sub>N</sub>. P<sub>B</sub> is more likely to increase in response on increase in P<sub>C</sub> with an increase in R<sub>C</sub>/R<sub>N</sub>, namely, an increase in the blood flow in noncollapsed vessels.

The ρh term in equations (3) and (4) expresses the hydrostatic pressure. In venous hemodynamics, RQ as a dynamic pressure component of flow and flow resistance is usually low<sup>8,12</sup> and the role of hydrostatic pressure in determining P<sub>B</sub> is large. A comparison of equations (3) with (4) shows that -ρh<sub>2</sub> < -ρh<sub>1</sub> and also P<sub>C</sub> < P<sub>I</sub>, because the reference point of the hydrostatic pressure in a collapsed vessel corresponds to the site of collapse.<sup>8</sup> It is expected, therefore, that P<sub>B</sub> of equation (3) is less than P<sub>B</sub> of equation (4). Actually, -ρh + P approaches closer to -ρh<sub>2</sub> + P<sub>C</sub> as R<sub>C</sub>/R<sub>N</sub> increases, and, conversely, it approaches to -ρh<sub>1</sub> + P<sub>I</sub> as R<sub>N</sub>/R<sub>C</sub> increases. Thus, P<sub>B</sub> is likely to have a greater value as the blood flow in collapsed vessels increases.

The above discussion is based on the assumption that the flow, the height of the hydrostatic column, and the flow resistance stay uniform, despite changes in the pressure. Therefore, the model does not precisely reflect actual *in vivo* hemodynamics but is useful for explaining our results. P<sub>B</sub> loses its relationship with P<sub>C</sub> when venous blood flows mostly in collapsed vessels, thus CVP and VSP are not necessarily interrelated.

Neck compression increases P<sub>B</sub> by increasing the

surrounding pressure of the collapsed vessel. It would be expected, therefore, to produce a marked effect when venous blood flows dominantly in collapsed vessels, that is, when the initial VSP is relatively high. In contrast, when the initial VSP is low and the venous blood flow is primarily in noncollapsed vessels, neck compression may not produce a significant increase in VSP. A marked CO decrease such as occurred with PEEP may reduce venous blood flow from the head. A decrease in flow decreases VSP, as seen in equation (2). The effect of increasing CVP by PEEP on VSP may be enhanced when the initial VSP is low and the blood flow in noncollapsed vessels increases. Therefore, at a highly negative initial VSP, PEEP increases VSP but it may even reduce VSP due to a decrease in the venous blood flow with greater initial VSP. Abdominal compression results in approximately the same hemodynamic effects as those produced by a G suit (table 1)<sup>4,13,14</sup> and may therefore substitute for it. The purpose of abdominal compression is to increase VSP by way of a CVP increase in the same way as PEEP and its effect was marked when pre-VSP values were highly negative and there was therefore a large venous blood flow in noncollapsed vessels. However, it differs from PEEP in increasing CO and the venous blood flow from the brain as reflected by the increase of  $P_{vsO_2}$ . The increase in the venous blood flow may be one of the reasons for a greater effect of abdominal compression than PEEP on the VSP increase. The G suit has been reported to be an inadequate prophylaxis, because the CVP increase produced by the G suit returns to its original level within 5 min.<sup>14,15</sup> Abdominal compression seems to be preferable for this purpose, as CVP remains increased even at 20 min after its application.

Massive air embolism may occur when the venous pressure in noncollapsible vessels is highly negative.<sup>2,16</sup> In the present study, at highly negative initial VSP, the increase in CVP produced by either PEEP or abdominal compression was especially effective in increasing VSP. However, an increase in CVP does not always increase VSP above atmospheric pressure. Caution is needed especially for PEEP, which may conversely reduce VSP because of reduced CO. Voorhies *et al.*<sup>3</sup> recommended the use of PEEP but did not note abnormal Doppler sounds in 41 of 81 cases, despite application of PEEP up to 20 cmH<sub>2</sub>O; clinically significant air embolism occurred in one of these 41. Ideally, noncollapsible venous pressure should be maintained at as close to atmospheric pressure as possible. The combined application of all three prophylactic methods may be desirable to assure a stable increase in the noncollapsible venous pressure of the head and neck above atmospheric pressure. The combination of PEEP and the abdominal compression has been found to prevent PEEP-induced CO decreases and should thus enhance the effect of PEEP on the VSP

increase.<sup>17</sup> Neck compression had a maximum effect on VSP when the initial VSP was only slightly negative or positive. Because it readily increased VSP above atmospheric pressure, air embolism would be completely prevented in this circumstance. Neck compression has been reported as effective therapy or prophylaxis for air embolism.<sup>5,18</sup> However, Millar<sup>19</sup> reported one case of probable air embolism and two cases of entry of air into occipital veins in 110 patients undergoing sitting-position operations with the use of frequent intermittent neck compression. The results of the present study suggest that neck compression is incapable of preventing air embolism in all circumstances, since its effect on VSP is slight at highly negative initial VSP levels.

The intracranial noncollapsible venous pressure in the sitting position is not always below atmospheric pressure in the dog (fig. 2) and presumably in humans. To further increase an already positive venous pressure detracts from the advantages of the sitting position; therefore, routine application of prophylaxis may not be desirable in all patients. As shown in this study, the venous pressure in the head responded significantly either to CVP increase or to neck compression but not to both. It is, therefore, impossible to predictably obtain meaningful increases in the venous pressure by application of any one of the three methods. For selection of the most suitable prophylaxis and for avoidance of unnecessary increases in venous pressure, it would be necessary to measure or estimate intracranial noncollapsible venous pressure. Catheterization of the internal jugular vein and measurement of the venous pressure in the vicinity of the superior bulb may be one means of achieving this. Prophylaxis would then be introduced only when the venous pressure measured at the level of the operation site is below atmospheric pressure.

In conclusion, in the seated dog, prophylaxis for air embolism by increase in CVP as achieved by PEEP or abdominal compression would be effective when the intracranial noncollapsible venous pressure is relatively highly negative. When the noncollapsible venous pressure is only slightly negative or positive, neck compression is more effective than increase in CVP. Therefore, if these observations apply to humans, a single application of any one of these methods in all procedures performed in the sitting position may result in unpredictable prevention of air embolism. The method best suited for individual cases could be determined only by knowing the noncollapsible venous pressure in the head.

## References

1. Gronert GA, Messick JM Jr, Cucchiara RF, Michenfelder JD: Paradoxical air embolism from a patent foramen ovale. *ANESTHESIOLOGY* 50:548-549, 1979
2. Gildenberg PL, O'Brien RP, Britt WJ, Frost EAM: The efficacy of Doppler monitoring for the detection of venous air embolism. *J Neurosurg* 54:75-78, 1981

3. Voorhies RM, Fraser RAR, Poznak AV: Prevention of air embolism with positive end expiratory pressure. *Neurosurgery* 12:503-506, 1983
4. Martin JT: Neuroanesthetic adjuncts for surgery in the sitting position. II. The antigravity suit. *Anesth Analg* 49:588-593, 1970
5. Kano T, Araki Y, Awata M, Ohtsu T, Ohmori K, Morioka T: A neck compression apparatus devised for a prophylaxis of air embolism. *Masui* 29:166-172, 1980
6. Michenfelder JD, Gronert GA, Rehder K: Neuroanesthesia. *ANESTHESIOLOGY* 30:65-100, 1969
7. Reinhard KR, Miller ME, Evans HE: The craniovertebral veins and sinuses of the dogs. *Am J Anat* 111:67-87, 1962
8. Gauer OH, Thron HL: Postural changes in the circulation, *Handbook of Physiology, Section 2, Circulation, vol 3*. Edited by Hamilton WF, Dow P. Washington, D.C., American Physiological Society, 1965, pp 2409-2439
9. Permutt S, Bromberger-Barnea, Bane HN: Alveolar pressure, pulmonary venous pressure, and the vascular waterfall. *Med Thorac* 19:239-260, 1962
10. Permutt S, Riley RL: Hemodynamics of collapsible vessels with tone: The vascular waterfall. *J Appl Physiol* 18:924-932, 1963
11. Guyton AC, Adkins LH: Quantitative aspects of the collapse factor in relation to venous return. *Am J Physiol* 177:523-527, 1954
12. Clark JH, Hooker DR, Weed LH: The hydrostatic factor in venous pressure measurements. *Am J Physiol* 109:166-177, 1934
13. Bondurant S, Hickam JB, Isley JK: Pulmonary and circulatory effects of acute pulmonary vascular engorgement in normal subjects. *J Clin Invest* 36:59-66, 1956
14. Gray S, Shaver JA, Kroetz FW, Leonard JJ: Acute and prolonged effects of G suit inflation on cardiovascular dynamics. *Aerospace Med* 40:40-43, 1969
15. Tinker JH, Vandam LD: How effective is the G suit in neurosurgical operations? *ANESTHESIOLOGY* 36:609-611, 1972
16. Emery ERJ: Air embolism. A report of two cases, one treated successfully. *Anaesthesia* 17:455-466, 1962
17. Hewer AJH, Logue V: Methods of increasing the safety of neuroanaesthesia in the sitting position. *Anaesthesia* 17:476-481, 1962
18. Leivers D, Spilsbury RA, Young JVI: Air embolism during neurosurgery in the sitting position. Two case reports. *Br J Anaesth* 43:84-90, 1971
19. Millar RA: Neurosurgical anaesthesia in the sitting position. A report of experience with 110 patients using controlled or spontaneous ventilation. *Br J Anaesth* 44:495-505, 1972