Anesthesiology
68:53–58, 1988

Effect of PEEP and Jugular Venous Compression on Canine Cerebral Blood Flow and Oxygen Consumption in the Head Elevated Position

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Cerebral blood flow (CBF) (radiolabelled microspheres) and oxygen consumption ($CMRO_2$) were studied in nine dogs during 30 min of either neck vein compression or application of positive end-expiratory pressure (PEEP) ventilation. With the animal in the prone position, elevation of the head from horizontal to 30 cm above the heart markedly decreased cisterna magna (P_{CSF}) and dorsal sagittal sinus pressure (P_{CV}). With the head elevated, compression of neck veins using neck tourniquet (pressure 40 mmHg) increased P_{CSF} and P_{CV} from 3.6 ± 2.2 to 6.8 ± 4.8 and −2.5 ± 2.7 to 2.3 ± 2.3 mmHg (mean ± SE, $P < 0.05$), respectively, while total or regional CBF and $CMRO_2$ remained unchanged. Application of PEEP (15 cm H_2O) increased right atrial pressure (−4.7 ± 1.7 to −0.1 ± 3.4 mmHg, $P < 0.05$), but did not affect P_{CSF} or P_{CV} (3.4 ± 3.3 to 3.3 ± 3.7 and −3.5 ± 2.6 to −4.1 ± 2.4 mmHg, respectively, $P > 0.05$). Total or regional CBF and $CMRO_2$ were also unaffected. These data demonstrate that, although neither maneuver affects CBF or $CMRO_2$, neck vein compression elevates P_{CV} above atmospheric pressure, but PEEP does not. In patients at risk for cerebral venous embolism, intermittent neck vein compression should be used as a prophylactic measure to prevent air embolism. (Key words: Brain: cerebral blood flow; cerebral O_2 consumption; jugular venous compression. Ventilation: positive end-expiratory pressure.)

WHEN PATIENTS ARE placed in the sitting position for a neurosurgical procedure, risk of venous air embolism is greatly increased. In this position, hydrostatic effects on cerebral hemodynamics become the major influencing factor in determining cerebral venous pressure.[1] The greater the gradient between cerebral veins and the right atrium, the greater the tendency for air to enter venous openings at the craniotomy site.[2] Clinically, there are three maneuvers available to reduce the risk of venous air embolism: employment of positive end-expiratory pressure (PEEP) ventilation; use of antigravity suit; and compression of the jugular veins. Because PEEP and antigravity suits raise right atrial pressure, it is presumed that cerebral venous pressure is also raised. In a previous study,[3] we demonstrated in dogs that jugular venous valves located at the thoracic inlet mechanically prevented the transmission of the increased right atrial pressure (P_{RA}) to upstream cerebral venous channels during PEEP. On the other hand, supravalvular compression of jugular veins effectively raised cerebral venous pressure. Since we did not examine cerebral hemodynamic and metabolic effects of the two maneuvers, the relative safety of clinical application of these two maneuvers is unclear. The purpose of the present study was, therefore, to assess total and regional cerebral blood flow (CBF) and cerebral O_2 consumption ($CMRO_2$) during PEEP and jugular venous compression in a canine model similar to that used in the previous study.[3]

Methods and Materials

General Procedures

The protocol for these studies has been approved by the Animal Care & Use committee of The Johns Hopkins Medical Institutions. A total of nine dogs weighing between 20 and 30 kg were studied. Following sodium pentobarbital anesthesia (30 mg/kg iv), the animals were paralyzed with pancuronium (0.1 mg/kg), their tracheas were intubated, and they were mechanically ventilated. Anesthesia was maintained with additional doses of sodium pentobarbital as required. Tidal volume and respiratory rate were adjusted to maintain a constant alveolar end-expiratory carbon dioxide tension of 4% as measured by a Beckman CO_2 gas analyzer. The dogs were then placed in the prone position with the head supported in a stereotaxic frame. The dorsal sagittal sinus was cannulated for measurement of cerebral venous pressure (P_{CV}) and blood gas analysis. Intracranial pressure (P_{CSF}) was recorded with a #16 angio catheter placed into the cisterna magna. The temperature of the animal was maintained at 38 ± 1° C with the use of a heating pad. Both femoral arteries were cannulated. In one, a catheter was advanced into the left cardiac ventricle for injection of radiolabelled microspheres, and a catheter in the other was used for reference blood sampling for the microsphere technique. Both femoral veins were cannulated, one for insertion

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Received from the Department of Anesthesiology/Critical Care Medicine, Johns Hopkins Medical Institutions, Baltimore, Maryland. Accepted for publication August 11, 1987. Supported in part by USPHS NIH grant #NS 20020.

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TABLE 1. Cerebral and Systemic Hemodynamic Changes of Head Elevation

	SBP (mmHg)	P_{RA} (mmHg)	P_{CV} (mmHg)	P_{CSF} (mmHg)
Head horizontal	120 ± 14	−3.1 ± 0.9	4.9 ± 1.1	11.2 ± 1.2
Head elevated	115 ± 12	−5.1 ± 0.6*	−3.6 ± 0.7*	2.2 ± 0.9*

SBP = mean systemic arterial blood pressure; P_{RA} = mean right atrial pressure; P_{CV} = mean sagital sinsus pressure; P_{CSF} = mean cisterna magna pressure. All values are mean ± SE (N = 9).

* $P < 0.05$. *vs.* head horizontal. Measurements were performed after 30 min of stabilization following the change of positions.

of a pulmonary artery catheter for cardiac output (CO), right atrial pressure (P_{RA}), pulmonary artery pressure (P_{PA}), and pulmonary artery wedge pressure (P_{PAW}), and one for fluid administration. The left brachial artery was cannulated for measurement of systemic arterial blood pressure (SBP).

Cerebral Blood Flow Measurement

CBF was measured with radiolabelled microspheres (15 ± 1.5 μm diameter; Dupont-NEN® products). Six radiolabels (^{153}Gd, ^{114}In, ^{113}Sn, ^{103}Ru, ^{95}Nb, and ^{46}Sc) were injected in a random sequence in each animal. Prior to each injection, the vial containing the spheres was shaken vigorously and sonicated for 20 min. Approximately 2.4×10^6 spheres were injected for each measurement of flow. The microspheres were injected into the left ventricle over a 30-s period, followed by a 20-s flush of 10 ml of saline. The reference blood sample was withdrawn from the aorta (*via* femoral artery) using a Harvard® withdrawal syringe pump set at 4.94 ml/min, beginning 1 min prior to the injection, and continuing for 2 min after the flush. At the end of the experiment, the animal was killed with KCl, and the brain was removed and fixed in 10% buffered formalin for 3–5 days.

The brain was then cut into the following areas: spinal cord, medulla, pons, midbrain, diencephalon, cerebellum, caudate nucleus, hippocampus, and posterior, middle, and anterior cerebral artery distribution. All tissue samples were weighed and placed in 15 ml poly Q vials and counted in a Packard® multi-channel autogamma scintillation spectrometer (model 5200) with a 3-inch through hole NaI crystal. The energy levels of the window settings for the six isotopes were as follows: ^{153}Gd, 70–174; ^{114}In, 74–230; ^{113}Sn, 370–430; ^{103}Ru, 460–550; ^{95}Nb, 710–820; ^{46}Sc, 840–1200 Kev, respectively. The overlap of activity among isotopes was subtracted to obtain corrected counts for each isotope by solving simultaneous equations using overlap coefficients from pure isotope standards.[4] Blood flow was calculated from the equation $\dot{Q}t = Ct \times \dot{Q}r/Cr \times Wt$, where $\dot{Q}r$ is the reference withdrawal rate, Cr is the reference sample counts, Ct is the tissue sample counts, and Wt is tissue weight in grams.

Blood Gas and *p*H Analysis and Determination of $CMRO_2$, and Cerebral Oxygen Extraction

Arterial and cerebral venous blood samples were taken directly from the femoral artery and dorsal sagittal sinus cannulae, respectively. O_2 tension, CO_2 tension, and *p*H were measured at 37° C immediately after the samples were obtained by use of Radiometer BMS3 electrodes and analyzer. Oxygen saturation and hemoglobin were measured immediately after samples were obtained with an Instrumentation Laboratories CO-oximeter (model 282). Arterial and venous O_2 contents were calculated from the measured O_2 saturation and hemoglobin concentration. $CMRO_2$ was calculated as the product of CBF and the cerebral arterial-venous oxygen content difference. Cerebral fractional oxygen extraction (E) was calculated as follows:

$$E = \frac{\text{Cerebral Arterial} - \text{Venous } O_2 \text{ Content Difference}}{\text{Cerebral Arterial } O_2 \text{ content}}$$

Experimental Protocols

Following preparation, the head of the dog was lowered to the level of the heart. After stabilization (30 min) in this position, the first set of control measurements (P_{CSF}, P_{CV}, and blood gases) were obtained. SBP, P_{RA}, P_{PA}, P_{PAW}, and CO were also obtained. The head was then elevated with the dorsum of the cranial vault 30 cm above the level of the right heart. The P_{CSF} and P_{CV} transducers were placed and zeroed at the level of the external auditory meatus, and the P_{PA}, P_{RA}, and SBP transducers were placed and zeroed at the level of the right heart. After stabilization (30 min) in the head elevated position, a second set of control measurements were obtained. The effects of application of jugular venous compression and PEEP were studied in all dogs, and each animal served as its own control for elevation of each pressure. Each maneuver was applied alterna-

TABLE 2. Cardiopulmonary Hemodynamic Changes during PEEP and Neck Compression

	PEEP 15 cm H_2O			Neck Vein Compression 40 mmHg		
	0	15 min	30 min	0	15 min	30 min
SBP (mmHg)	122 ± 14	115 ± 17	121 ± 13	119 ± 11	120 ± 9	122 ± 7
CO (L/m.)	3.8 ± 0.9	2.6 ± 0.7*	2.6 ± 0.6*	3.9 ± 0.7	3.4 ± 0.8	3.6 ± 1.0
P_{RA} (mmHg)	−4.7 ± 1.7	−0.4 ± 3.4*	−0.1 ± 3.4*	−2.5 ± 3.9	−2.8 ± 5.0	−2.8 ± 4.8
P_{PA} (mmHg)	5.3 ± 2.6	14.5 ± 6.0*	14.6 ± 5*	6.3 ± 2.8	5.6 ± 3.9	6.4 ± 4.1
HR (Beat/m.)	129 ± 40	122 ± 45	122 ± 43	133 ± 48	135 ± 39	134 ± 39
P_{PAW} (mmHg)	0.3 ± 3.1	9.1 ± 3.8*	10.3 ± 3.8*	1.8 ± 2.6	0.4 ± 2.9	0.9 ± 2.7

SBP = mean systemic arterial blood pressure; CO = cardiac output; P_{RA} = mean right atrial pressure; P_{PA} = pulmonary artery diastolic pressure; HR = heart rate; P_{PAW} = pulmonary artery wedge pressure.

All values are mean ± SE (n = 9). Measurements were made at control 0, 15, and 30 min after application of each manueuver.

* $P < 0.05$ *vs.* neck vein compression.

tively with a 30-min time period for stabilization between each study.

JUGULAR VENOUS COMPRESSION

The jugular veins were compressed using a modified scalp vein tourniquet (W.A. Brown Co., NY) placed around the lower neck at a tourniquet pressure of 40 mmHg. This pressure was previously found to be the lowest pressure that would consistently raise cerebral venous pressure (P_{CV}) above atmospheric pressure.[3]

APPLICATION OF PEEP

An end-expiratory pressure of 15 cm H_2O was applied by submerging the expiratory limb of the ventilator into a water filled reservoir. Ventilation was adjusted to maintain end-expiratory alveolar CO_2 tension constant throughout the experiment. Each maneuver (jugular venous compression and PEEP) was studied for 30 min. During the study period, all measurements were made at 0, 15, and 30 min.

Statistical Analysis

All data were compared statistically with a two-factor (maneuver and time) analysis of variance (ANOVA). When significant ($P < 0.05$) variance ratios were obtained, least significant differences were calculated by multiple comparison with Tukey's test.[5]

Results

Blood gas and *p*H values were not significantly different in all animals pre-, during, and post-manipulations with jugular venous compression or PEEP.

When the head of the dog was elevated 30 cm above the heart from a control heart level, P_{CV} and P_{CSF} decreased sharply. There were no other significant hemodynamic changes which occurred in response to elevation of the head (table 1).

Table 2 shows the cardiopulmonary hemodynamic changes after application of either PEEP or neck tourniquet with the dog in the head elevated position. With PEEP, cardiac output decreased, whereas mean right atrial, pulmonary artery, and pulmonary capillary wedge pressures increased. There were no significant changes in these variables during application of neck tourniquet. Figure 1 shows cerebral hemodynamic and metabolic changes during the application of PEEP and neck tourniquet. As tourniquet compression pressure increased to 40 mmHg, P_{CSF} increased from control (3.6 ± 2.2) to 6.8 ± 4.1 mmHg (mean ± SE), and remained elevated throughout the entire 30-min time period. P_{CV} increased from control (−2.5 ± 2.7) to 2.2 ± 2.5 mmHg (mean ± SE). CBF, $CMRO_2$, cerebral O_2E, and P_{RA} remained unchanged. With PEEP, P_{RA} increased, but all other measurements were unchanged from control values. All measurements returned to control values when PEEP or neck compression was released. Regional CBF in all brain areas was unchanged during either application of PEEP or neck tourniquet (table 3).

Discussion

When the head is elevated, cerebral venous and intracranial pressures decrease.[6] Cerebral venous pressure varies with the degree of head elevation and, in an upright sitting position, cerebral venous pressure can be as low as −10 mmHg.[1] The present study confirms our previous study, which showed that PEEP does not raise cerebral venous pressure, whereas neck vein compression does so effectively.[3] We attributed the failure of PEEP to raise cerebral venous pressure to the presence of jugular vein valves located at the thoracic inlet. Similar valves are also present in humans at the same location. The clinical significance of these valves has been studied in detail by Fisher *et al.*[7]

In an anesthetized neurosurgical patient, the change from a supine to a sitting position may result in a de-

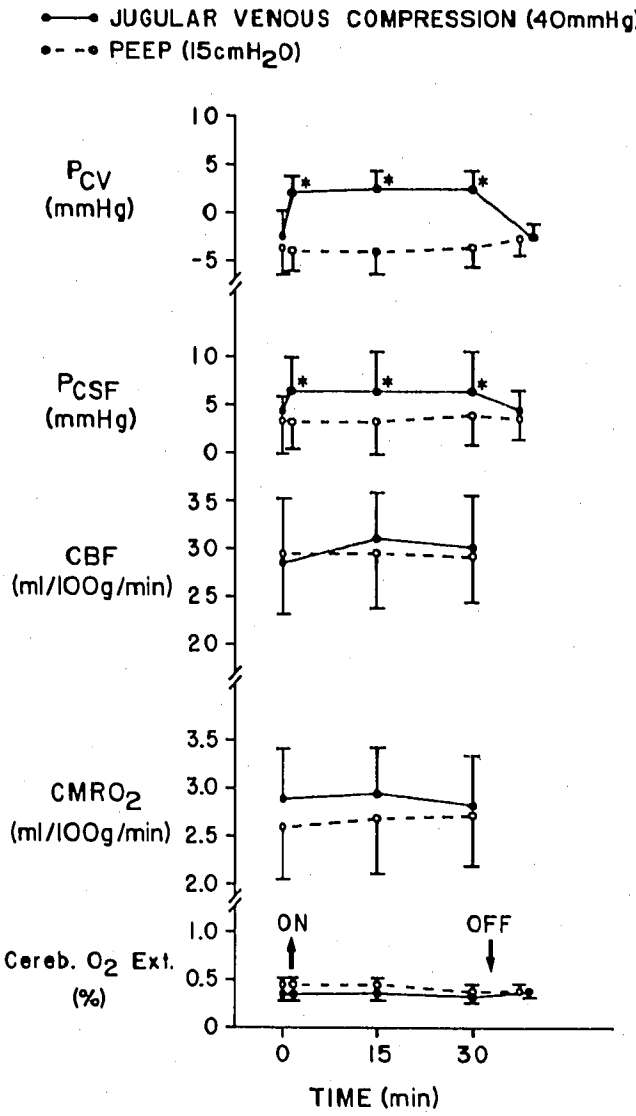

FIG. 1. Effects of application of PEEP and jugular venous compression with tourniquet on cerebral hemodynamics, oxygen consumption, and oxygen extraction. P_{CV} = mean cerebral venous pressure; P_{CSF} = mean cerebral spinal fluid pressure; CBF = cerebral blood flow; $CMRO_2$ = cerebral oxygen consumption; Cereb. O_2 Ext. = cerebral oxygen extraction. All values are the mean ± SE (n = 9). Measurements were made at control 0, 15, and 30 min after application of each maneuver. *$P < 0.05$ *versus* PEEP.

crease in right atrial pressure due to venous pooling.[8] A greater decrease is seen in hypovolemic patients; the greater the decrease in right atrial pressure, the greater the pressure gradient between the cerebral veins and the heart. It is commonly believed that the application of PEEP can offset the hydrostatic gradient and, if the level of PEEP is high enough, cerebral venous pressure may actually increase to above atmospheric pressure. However, such high levels of PEEP are not recommended for prevention of venous air embolism with the patient in the sitting position. PEEP at this high level causes not only a severe decrease in cardiac output and reduction of cerebral perfusion pressure, but also reverses the left to right atrial pressure gradient and increases the risk of paradoxical air embolism.[9] More importantly, jugular venous valves located at the thoracic inlet prevent the transmission of increased right atrial pressure induced by PEEP to the upstream cerebral venous system. Indeed, our previous animal study demonstrated that the high level of PEEP (20 cm H_2O) not only did not increase cerebral venous pressure, but actually decreased it. If PEEP is to be effective in reducing venous embolism, P_{CV} has to be raised above atmospheric pressure. This maneuver, therefore, is not effective as a prophylactic measure for prevention of venous air embolism. A similar response of cerebral sinuses pressure to PEEP in seated patients has recently been demonstrated.[1]

In contrast to the application of PEEP, compression of the jugular veins at the lower neck raises cerebral venous pressure effectively above atmospheric pressure, and the rise is linear with pressure used for compression.[3] This suggests that a tourniquet placed around the neck can act as a Starling type resistor to regulate cerebral venous pressure. In our previous study, a tourniquet pressure of only 40 mmHg was needed to raise cerebral venous pressure above atmospheric pressure, and this pressure does not affect cerebral perfusion pressure (defined as arterial pressure minus P_{CSF}). This is because, in this situation, P_{CSF} is higher than cerebral venous pressure, and P_{CSF} is the back pressure for cerebral perfusion. This is an analogous situation to the vascular waterfall model of the pulmonary circulation,[10] in which surrounding pressure—extravascular or alveolar pressure—represents the downstream pressure for pulmonary perfusion as long as left atrial pressure is less than alveolar pressure. Under such conditions, blood flow through the vascular segment is proportional to inflow pressure minus the surrounding pressure. In the present study, we extended our previous observations, and demonstrated that the compression at this cuff pressure does not impair cerebral oxygen consumption, or total or regional cerebral blood flow. These results are consistent with those following with direct elevation of cerebral venous pressure.[11-14]

There are three clinical concerns about the technique of jugular venous compression. First, the compression may cause cerebral venous outflow obstructions, resulting in an increased intracranial pressure, and thus reduce CBF. Effects of elevation of jugular venous pressure on CBF have been extensively investigated. Wagner and Traystman[11-13] have demonstrated that CBF remained unchanged as jugular venous pressure

TABLE 3. Regional Cerebral Blood Flow ($ml \cdot min^{-1} \cdot 100g^{-1}$) after Application of PEEP and Neck Tourniquet Compression (SD) (n = 9)

	PEEP			Neck Tourniquet		
		Minutes After Application			Minutes After Application	
Region	Pre-PEEP	15	30	Pre-tourniquet	15	30
Spinal Cord	15.8 ± 6.3	15.5 ± 4.5	14.5 ± 3.9	15.0 ± 6.5	15.4 ± 4.0	15.2 ± 4.5
Medulla	26.9 ± 6.8	26.1 ± 6.3	27.9 ± 6.6	27.2 ± 6.8	27.2 ± 6.8	26.0 ± 6.9
Pons	19.8 ± 4.5	19.8 ± 5.1	20.3 ± 4.0	19.7 ± 3.2	19.9 ± 4.6	19.2 ± 4.2
Midbrain	36.7 ± 8.3	40.1 ± 13.3	40.6 ± 8.3	37.6 ± 10.4	41.0 ± 11.9	36.8 ± 11.9
Diencephallon	27.7 ± 7.4	28.4 ± 0.9	29.9 ± 7.7	27.6 ± 7.4	31.4 ± 12.5	28.6 ± 10.7
Caudate Nucleus	50.0 ± 7.9	53.9 ± 27.3	47.9 ± 14.9	48.8 ± 21.2	50.9 ± 18.3	47.4 ± 18.2
Cerebellum	33.6 ± 6.0	33.6 ± 7.5	34.8 ± 8.4	32.0 ± 5.8	33.8 ± 6.6	32.2 ± 6.8
Hippocampus	24.5 ± 7.0	25 ± 6.8	25.6 ± 4.9	24.8 ± 4.8	26.7 ± 7.7	24.2 ± 6.6
PCA Regions	38.9 ± 8.9	39.0 ± 11.5	38.4 ± 5.1	39.3 ± 11.4	40.6 ± 9.3	39.0 ± 9.3
MCA Regions	29.9 ± 8.4	31.1 ± 7.1	30.4 ± 7.9	29.5 ± 7.2	32.1 ± 9.6	30.1 ± 9.5
ACA Regions	29.5 ± 8.6	29.5 ± 9.0	30.9 ± 9.3	30.4 ± 10.6	34.3 ± 14.3	29.2 ± 12.6

PCA = posterior cerebral artery; MCA = middle cerebral artery; ACA = anterior cerebral artery.

was elevated to pressures which allowed cerebral perfusion pressure to be maintained above 60 mmHg. As jugular venous pressure was further elevated (30 mmHg) and cerebral perfusion pressure was reduced to below 60 mmHg, CBF was reduced 35%. In these situations, P_{CSF} was maintained at atmospheric pressure, so that cerebral perfusion pressure was defined as mean arterial blood pressure minus jugular venous pressure, and no vascular waterfall was present. Several other studies, which have focused on the cerebral vascular response to decreases in cerebral perfusion pressure through increases in cerebral venous pressure, have provided conflicting results. Moyer *et al.*[14] showed no change in CBF with jugular venous pressure elevations to 25 mmHg. Ekstrom-Jodal[15] showed small reductions in CBF (15%) with increases in cerebral venous pressure to 25 mmHg, whereas Emerson and Parker[16] reported marked decreases in CBF with similar venous pressure elevations. Raisis *et al.*,[17] on the other hand, demonstrated a decreased CBF with venous pressure elevation, but only when pressure was elevated to levels above 75 mmHg. In most of these previous studies, mean arterial blood pressure was maintained in the range of 100–130 mmHg. If cerebral perfusion pressure is defined as mean arterial pressure minus cerebral venous pressure, only in the study of Raisis *et al.*[17] was perfusion pressure reduced to values below 60 mmHg. If one considers the generally accepted value of the lower limit of cerebral autoregulation to be around 60 mmHg, then only in the Raisis study should CBF have fallen. In any event, with a tourniquet pressure of 40 mmHg, the technique used in the present study increased cerebral venous pressure only slightly above atmospheric pressure (<6 mmHg), and thus allowed a wide margin of safety to maintain an adequate cerebral perfusion pressure.

The second possible concern is that this technique may potentially compress carotid arteries and thus decrease cerebral perfusion pressure. However, our previous study showed that carotid arterial pressure distal to compression was not decreased, and the present study shows that CBF was not reduced. The compression pressure of 40 mmHg is far less than the pressure of 1500 mmHg that was found necessary to produce global ischemia in primates.[18]

The third concern is that jugular venous compression by this technique could lead to venous engorgement, brain swelling, and, possibly, brain edema. Cerebral venous occlusion due to prolonged and excessive jugular venous compression certainly could lead to brain edema. Prompt return of elevated P_{CV} and P_{CSF} to precompression levels upon tourniquet release, as had been shown repeatedly in the present study, indicates that the complications do not occur during such a low pressure and a short duration of compression.

The use of controlled neck compression has been suggested recently for surgery performed with the patient in the sitting position to prevent venous embolism.[19,20] The amount of tourniquet cuff pressure recommended varies, however, from 15 to 90 mmHg. Structural differences in the tourniquet certainly will affect greatly the amount of pressure that is transmitted to the venous channels. It is the change in cerebral venous pressure, not tourniquet pressure, which is the important determinant in terms of preventing air entrainment. Regardless of the method used for compression, the amount of pressure applied should be the least that will sufficiently raise cerebral venous pressure above atmospheric pressure.

The results of the present study do not entirely agree with the results of Hibino *et al.*,[21] who evaluated the effects of PEEP, abdominal compression, and neck

compression on cerebral dural sinus pressure in dogs in the seated position. They found each method increased venous sinus pressure, but that the levels reached were variable and depended on the initial dural sinus pressure prior to the application of each maneuver. They attributed the variability of the effects to differences in cerebral venous drainage, whether blood drains predominantly through the collapsible jugular venous system or non-collapsible vertebral venous system. However, several important factors which may have profoundly affected their results were not discussed. These factors include: the presence of jugular venous valves which mechanically prevent the transmission of increased right atrial pressure induced by PEEP or abdominal compression; the amounts of pressure used for abdominal and neck compression; and the effects of respiratory changes (Pa_{CO_2}) induced by application of PEEP and abdominal compression on cerebral hemodynamics.

In the present study, a 30-min interval of neck vein compression was chosen to study cerebral hemodynamic and metabolic effects. Thirty minutes of neck vein compression is thought long enough to enable the surgeon to search for venous openings, which are the potential source of air entry. Once the source is identified, and adequate hemostasis has been obtained, neck compression would then be released. With the use of intermittent jugular venous compression, one hopes that a better hemostasis can be obtained, and this should decrease the incidence of venous air embolism.

The authors thank the technical staff of the Department of Anesthesiology/Critical Care Medicine for their excellent assistance. They also thank Renee Craig and Candace Berryman for their typing of this manuscript.

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