Effect of positive end-expiratory pressure on the incidence of venous air embolism and on the cardiovascular response to the sitting position during neurosurgery

R. Giebler, B. Kollenberg, G. Pohlen and J. Peters

Summary

We have studied prospectively the effect of 10 cm H2O of PEEP on the incidence of venous air embolism and on the cardiovascular response to change from the supine to the seated position in a large neurosurgical population. Patients were allocated randomly to receive either PEEP (10 cm H2O, n = 45) or conventional (control, n = 44) ventilation. Cardiovascular and respiratory variables were measured in the supine and sitting positions, and monitoring included precordial Doppler probe, pulmonary artery pressure and expiratory carbon dioxide concentration. Venous air embolism was assumed if changes in precordial Doppler sounds occurred, end-tidal carbon dioxide concentration decreased or air could be retrieved from a central venous multi-orifice catheter. The incidence of venous air embolism (26%) did not differ between patients undergoing conventional ventilation and those undergoing ventilation with 10 cm H2O of PEEP. Venous air embolism was always detected first by alterations in Doppler sounds. Cardiac output was significantly higher in patients undergoing conventional ventilation than in those undergoing ventilation with PEEP in the supine but not in the sitting position. Furthermore, pulmonary vascular resistance increased significantly only in the upright position in those undergoing ventilation with PEEP. The pulmonary artery wedge pressure to central venous pressure gradient did not attain negative values with PEEP or with upright positioning. We conclude that the use of PEEP during neurosurgical procedures performed in the sitting position should be abandoned as it does not decrease the incidence of venous air embolism but is associated with significant adverse cardiovascular effects. (Br. J. Anaesth. 1998; 80: 30–35)

Keywords: embolism, air; position, effects; ventilation, positive end-expiratory pressure; ventilation, mechanical; surgery, neurological

Venous air embolism (VAE) may occur whenever venous pressure at the site of surgery is below atmospheric pressure and it may have devastating effects in patients undergoing neurosurgical procedures in the sitting position. The reported incidence of VAE varies widely, depending on the methods used for its detection, and incidences from 15% to 35% have been reported. Positive end-expiratory pressure (PEEP) has been recommended for many years for the prevention and mitigation of the effects of further VAE, despite the experimental observation that PEEP may fail to increase intracranial venous pressure. However, PEEP may decrease mean arterial pressure and reverse the left-to-right inter-atrial pressure gradient which might predispose patients with a probe-patient foramen ovale to the risk of paradoxical air embolism. Accordingly, some authors do not advocate routine use of PEEP during craniotomy in the sitting position. Nevertheless, 78% of all hospitals described the use of PEEP in a questionnaire on perioperative management during operations performed in the sitting position. However, because either only few patients were examined in prospective studies or data were collected retrospectively, differences in patient management and study design may not allow evaluation of the risk-benefit ratio of PEEP during neurosurgical procedures in the sitting position. Therefore, we studied prospectively in a large number of patients the effect of PEEP on the incidence of VAE in seated neurosurgical patients. Haemodynamic variables during ventilation with PEEP in both the supine and sitting positions were also examined, hypothesizing that PEEP decreases the incidence of VAE, and alters the pressure gradient between pulmonary capillary wedge and central venous pressures.

Patients and methods

After obtaining local Ethics Committee approval and written informed consent, we studied prospectively 89 consecutive adult patients (31 men, 57 women; mean age 52.3 (range 18–75) yr) undergoing elective neurosurgical procedures in the sitting position (85 suboccipital craniotomies, four cervical laminectomies) during a 1-yr period. Patients were allocated randomly to either PEEP (10 cm H2O, n = 45) ventilation or conventional ventilation (control, n = 44). There were no exclusion criteria.

Rainer Giebler*, MD, Barbara Kollenberg, MD, Gottfried Pohlen, MD, Jürgen Peters, MD, Abteilung für Anästhesiologie und Intensivmedizin, Universitätsklinikum der Universität - GH Essen, Germany.

*Address for correspondence: Abteilung für Anästhesiologie und Intensivmedizin Universitätsklinikum Essen, Hufelandstrasse 55, D-45122 Essen, Germany. Accepted for publication: August 21, 1997.
All patients were given flunitrazepam 1 mg orally, 1 h before operation. Anaesthesia was induced with propofol 1.5 mg kg\(^{-1}\) i.v., alfentanil 50 μg kg\(^{-1}\) i.v. and atracurium 0.5 mg kg\(^{-1}\) i.v. After tracheal intubation, the lungs were ventilated mechanically with an oxygen–air mixture (\(F_{\text{O}_2} = 0.5\)) either conventionally with a PEEP of 3 cm H\(_2\)O (control) or a PEEP of 10 cm H\(_2\)O. End-tidal carbon dioxide partial pressure was adjusted to 4.0–4.3 kPa. Anaesthesia was maintained by continuous infusion of propofol 5 mg kg\(^{-1}\) h\(^{-1}\), alfentanil 125 μg kg\(^{-1}\) h\(^{-1}\) and atracurium 0.3 mg kg\(^{-1}\) h\(^{-1}\). Before application of the skull clamp and before skin incision, a bolus dose of alfentanil 50 μg kg\(^{-1}\) was given. Patients in both groups received Ringler’s solution 4 ml kg\(^{-1}\) h\(^{-1}\) and hydroxyethyl starch solution (10% HES 500 ml) during induction. To record arterial pressure, a 20-gauge polyethylene catheter (Viggo-Spectramed, Montigny le Bretonneaux, France) was inserted into the left radial artery. For measurement of pulmonary arterial, central venous and pulmonary capillary wedge pressures, a four-lumen balloon-tipped pulmonary artery catheter (Thermocatheter Catheter 7.5 F, Viggo-Spectramed, Montigny le Bretonneaux, France) was inserted via the right internal jugular vein. In addition, a multi-orifice (2 side orifices; Arrow, Cen- tral Venous Catheterization Set with Blue Flex Tip; Reading, PA, USA) single-lumen central venous catheter allowing air retrieval in case of VAE was inserted via the right internal jugular vein and positioned near the sinus node using an ECG recorded via the catheter.\(^{12}\) Vascular pressures, ECG and respiratory carbon dioxide partial pressure were displayed on a monitor and recorded continuously on a strip chart recorder.

With the patient in the seated position a precordial Doppler probe (Dual Frequency Doppler, Model 915-AI, Parks Medical Electronics Inc., Aloha, OR, USA) was placed along the right parasternal border. Proper Doppler placement with detection of intravascular air was verified by rapid injection of 5 ml of agitated saline.\(^{13}\)

After operation, all patients were transferred to the intensive care unit and the lungs ventilated mechanically.

MEASUREMENTS

Heart rate was recorded from the ECG and cardiac output was assessed by thermodilution (Sirecust 961, Siemens, Erlangen, Germany); the average of three measurements was obtained using 10 ml of iced saline. Systemic and pulmonary vascular resistances were calculated from standard formulæ. Arterial, pulmonary artery, central venous and pulmonary capillary wedge pressures in addition to airway pressure (air filled system) were measured by electromanometer relative to barometric pressure. Pressures were referenced to the mid-axillary line in the supine position and to the anterior fifth intercostal space in the seated position. All pressure measurements were recorded continuously on a multi-channel strip-chart recorder (Siredoc 220, Siemens, Erlangen, Germany) at a paper speed of 200 mm min\(^{-1}\).

Respiratory variables (respiratory carbon dioxide partial pressure) were measured proximal to the tracheal tube by in-line infrared radiation absorption (Sirecust 961, Siemens, Erlangen, Germany), and tidal volume and ventilatory frequency by a hot wire anemometer (Anemone, Dräger, Lübeck, Germany). Arterial oxygen saturation was measured by pulse oximetry (PM 8050, Dräger, Lübeck, Germany).

For measurement of blood-gas tensions and pH, and concentrations of electrolytes and haemoglobin, arterial blood was obtained from the radial artery and pulmonary artery and analysed with electrodes at 37 °C (Ciba Corning 288 Blood Gas System, Ciba Corning, Fernwald, Germany).

EXPERIMENTAL DESIGN

Patients were allocated randomly to either ventilation with PEEP or conventional ventilation. Cardiovascular and respiratory variables were measured 15 min after induction of anaesthesia in the supine position (baseline) and 15 min after patients had been positioned in the sitting position. There was no surgical stimulation during these measurements. In patients with VAE, simultaneous measurements of central venous and pulmonary artery pressures were obtained and compared with values of these variables during the 15-min period immediately before air embolism in each patient.

During operation, the presence of VAE was assumed if either (1) changes in precordial Doppler sounds occurred,\(^{13}\) (2) end-tidal carbon dioxide partial pressure decreased suddenly by more than 0.3 kPa in a step manner\(^{14}\) or (3) air could be retrieved via the central venous catheter. Furthermore, during VAE, changes in mean arterial, pulmonary arterial and central venous pressures, and changes in arterial oxygen saturation (detected by pulse oximetry) were recorded.

STATISTICAL ANALYSIS

Results are reported as mean (SEM) unless otherwise indicated. Haemodynamic data were analysed by two-way analysis of variance for repeated measurements followed by post hoc Newman–Keuls test. A chi-square test was used to compare incidences of events between groups during different study conditions. The following a priori null hypotheses were tested: ventilation with PEEP (1) does not change the incidence of VAE and (2) does not alter values of cardiovascular variables. Results were considered to be statistically significant with an α error of \(P < 0.05\).

Results

INCIDENCE OF VENOUS AIR EMBOLISM

VAE was detected by a Doppler signal suggestive of air entry in 23 patients (26%), 11 undergoing PEEP ventilation and 12 undergoing conventional ventilation. The incidence of VAE did not differ significantly \((P = 0.95)\) between patients undergoing PEEP ventilation (24%) and those undergoing conventional ventilation (27%). Aspiration of air in those patients showing signs of air embolism was possible (recovered air volume 4–25 ml) in six patients (55%) in the PEEP group and in five (42%) in the conventional ventilation group. There was no significant
difference in the amount of air aspirated or the incidence of successful air aspiration between groups.

INCIDENCE AND SEVERITY OF HAEOMDYNAMIC ALTERATIONS AND CHANGES IN END-EXPIRATORY CARBON DIOXIDE TENSION WITH VENOUS AIR EMBOLISM

Approximately 60% of all detected venous air entries were followed by haemodynamic alterations. Most frequently, an increase in pulmonary artery pressure ($n = 13; 57\%$) was observed (PEEP group, six (55%) patients; control group, seven (58%) patients), whereas a decrease in mean arterial pressure (PEEP group, two (18%) patients; control group, three (25%) patients) or an increase in central venous pressure (PEEP group, two (18%) patients; control group, two (17%) patients) was seen less often.

VAE was accompanied by a similar incidence of a decrease in end-expiratory carbon dioxide tension (in seven (64%) patients in the PEEP group and in 10 (83%) patients in the control group). In fact, the mode of ventilation did not alter the sensitivity of different monitoring modalities to detect VAE with alterations in Doppler sounds considered the gold standard (fig. 1).

Neither the significant increase in pulmonary artery pressure (PEEP $+8.5 (2.4) \text{ mm Hg}$; control $+8.2 (1.9) \text{ mm Hg}$) nor the significant decrease in mean arterial pressure (PEEP $-14.1 (1.9) \text{ mm Hg}$; control $-15.3 (2.1) \text{ mm Hg}$), central venous pressure (PEEP $+3.5 (1.4) \text{ mm Hg}$; control $+3.0 (0.9) \text{ mm Hg}$) or carbon dioxide tension (PEEP $-0.6 (0.2) \text{ kPa}$; control $-0.8 (0.2) \text{ kPa}$) associated with air embolism were significantly affected by the amount of end-expiratory airway pressure. Therefore, there was no significant difference in the incidence or severity of cardiorespiratory alterations after VAE between patients in the PEEP and those in the conventional ventilation group (fig. 2).

EFFECT OF PEEP AND POSITIONING ON CARDIOVASCULAR VARIABLES

Moderately but significantly higher pulmonary artery pressure and pulmonary capillary wedge pressure values were observed in the PEEP group compared with the conventional ventilation group both in the recumbent and in the seated position. Central venous pressures was greater with PEEP in the sitting position but not in the supine position. Of note, pulmonary artery wedge pressure was higher than central venous pressures in the supine position in all patients.

When patients were placed in the seated position, pulmonary artery pressure, pulmonary capillary wedge pressure and central venous pressure decreased significantly (table 1). As pulmonary

![Figure 1](image1.png) Ability of different monitoring devices to detect venous air embolism in comparison with Doppler in 11 patients undergoing ventilation with 10 cm H$_2$O of PEEP and in 12 patients undergoing conventional ventilation, demonstrating signs of venous air embolism during neurosurgical operations in the seated position.

![Figure 2](image2.png) Cardiovascular alterations and changes in end-expiratory carbon dioxide tension associated with venous air embolism in neurological patients undergoing ventilation with 10 cm H$_2$O of PEEP and in patients undergoing conventional ventilation.
### Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SEM)</th>
<th>PEEP</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Supine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean pulmonary artery pressure (mm Hg)</td>
<td>23.7 (0.7)</td>
<td>21.2 (0.7)</td>
<td>19.8 (0.7)†</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure (mm Hg)</td>
<td>16.8 (0.5)†</td>
<td>14.7 (0.5)</td>
<td>13.0 (1.2)†</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>14.8 (0.1)†</td>
<td>12.9 (0.7)</td>
<td>11.2 (0.1)†</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure to central venous pressure gradient (mm Hg)</td>
<td>2.5 (0.3)</td>
<td>2.7 (0.3)</td>
<td>2.1 (0.3)</td>
</tr>
<tr>
<td>Cardiac index (litre min⁻¹ m⁻²)</td>
<td>2.3 (0.1)†</td>
<td>2.7 (0.1)</td>
<td>2.1 (0.1)*</td>
</tr>
<tr>
<td>Pulmonary vascular resistance index (dyn s⁻¹ cm⁻² m⁻²)</td>
<td>222 (15)</td>
<td>226 (22)</td>
<td>318 (20)†</td>
</tr>
<tr>
<td>Systemic vascular resistance index (dyn s⁻¹ cm⁻² m⁻²)</td>
<td>2740 (125)</td>
<td>2552 (115)</td>
<td>3167 (159)</td>
</tr>
<tr>
<td><strong>Seated</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean pulmonary artery pressure (mm Hg)</td>
<td>21.2 (0.7)</td>
<td>19.8 (0.7)†</td>
<td>16.1 (0.5)*</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure (mm Hg)</td>
<td>14.7 (0.5)</td>
<td>9.3 (0.3)*</td>
<td>13.0 (1.2)†</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>12.9 (0.7)</td>
<td>8.3 (0.4)*</td>
<td>11.2 (0.1)†</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure to central venous pressure gradient (mm Hg)</td>
<td>2.7 (0.1)†</td>
<td>2.5 (0.3)</td>
<td>2.3 (0.1)*</td>
</tr>
<tr>
<td>Cardiac index (litre min⁻¹ m⁻²)</td>
<td>2.1 (0.1)*</td>
<td>2.7 (0.1)</td>
<td>2.3 (0.1)*</td>
</tr>
<tr>
<td>Pulmonary vascular resistance index (dyn s⁻¹ cm⁻² m⁻²)</td>
<td>318 (20)†</td>
<td>240 (16)</td>
<td>3167 (159)</td>
</tr>
<tr>
<td>Systemic vascular resistance index (dyn s⁻¹ cm⁻² m⁻²)</td>
<td>3167 (159)</td>
<td>2845 (106)</td>
<td>3167 (159)</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>91.6 (3.0)</td>
<td>93.1 (2.8)</td>
<td>86.9 (2.4)</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>57 (1)</td>
<td>59 (2)</td>
<td>56 (1)</td>
</tr>
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</table>

**Discussion**

This study was the first to assess prospectively and in a randomized manner the impact of PEEP on the incidence of VAE and on cardiovascular alterations in a large number of patients operated on in the sitting position. We found that ventilation with a PEEP of 10 cm H₂O during neurosurgical procedures in the seated position offered no benefit but rather may be harmful.

Because reliable measurements and interpretation of results depend on central venous and pulmonary artery wedge pressures measured accurately in the supine and seated positions, we referenced all pressures to the mid-axillary line in the supine position and to the anterior fifth intercostal space when patients were in the seated position.¹⁰¹¹ There is consensus that pulmonary artery wedge pressure reasonably reflects mean left atrial pressure in the absence of cardiac valve abnormalities and during some unphysiological states (i.e. PEEP values up to 10 cm H₂O or more)¹⁵⁻¹⁷. As the pressure gradient between central venous pressure and pulmonary capillary wedge pressure is normally small, minor differences in the technique of measurement could alter the results obtained. Thus all variables were recorded continuously with each transducer referenced to zero repeatedly and compared between groups at similar times. Another possible source of error is that the pulmonary artery catheter tip might be wedged in regions where pulmonary capillary wedge pressure reflects airway pressure rather than left atrial pressure. However, Benumof and colleagues found important peripheral catheter locations in only 6.9% of patients in whom a pulmonary artery catheter had been inserted.¹⁸ Therefore, from a statistical point of view there was no significant difference between groups in pulmonary vascular resistance while in the supine position. Furthermore, pulmonary vascular resistance increased significantly with PEEP only in the upright position (table 1). Although mean arterial pressure tended to decrease and systemic vascular resistance to increase after attaining the seated position, these differences were not significant with regard to mode of ventilation or body position (table 1).

In conclusion, our study strongly suggests that ventilation with a PEEP of 10 cm H₂O during neurosurgical procedures in the seated position is not recommended because of the high incidence of VAE and on cardiovascular alterations.
view, false measurements caused by catheter misplacement are unlikely to have influenced the results in our 89 patients. Furthermore, to avoid possible changes in pulmonary catheter position (e.g. by use of PEEP itself), we ventilated the lungs of our patients with either PEEP or conventionally without changing airway pressure within a study group. Thus pressures measured and comparison between groups should have yielded reliable data.

The overall incidence of VAE (26%) observed in this study is consistent with that reported in several other series. With no difference in the incidence of VAE with and without PEEP, however, our data do not confirm the results of Lee, Lichtman and Weintraub who reported a decrease in VAE from 55% in patients whose lungs were ventilated without PEEP (n = 9) to 26% in patients who underwent ventilation with 10 cm H2O of PEEP (n = 19). However, as a PEEP value of up to 15 cm H2O has no detectable effect on sagittal or transverse sinus pressure in humans, our results demonstrating PEEP to be ineffective in preventing VAE were not unexpected.

We used a precordial Doppler probe to detect VAE. With 0.25 ml of air injected i.v. already altering Doppler sounds, precordial Doppler monitoring is more sensitive in detecting VAE than measurements of end-expiratory carbon dioxide partial pressure or pulmonary artery pressure. However, these latter two methods have the advantage of providing quantitative physiological information on the sequelae of venous air emboli and return of pulmonary artery pressure towards baseline can serve as a guide for the effectiveness of therapy. Our data are in accordance with these reports as changes in Doppler sounds always preceded a decrease in end-expiratory carbon dioxide tension and cardiovascular changes in both the conventional ventilation and PEEP groups. In addition, we could not demonstrate a significant impact of PEEP on the cardiovascular responses associated with VAE.

Historically, the incidence of VAE was associated with procedures on the head, neck, chest and in the post-partum period. Recently, VAE has been detected, for example in orthopaedic hip replacement and abdominal procedures, in addition to neurosurgical procedures in the prone, lateral or supine position. Therefore, VAE is not restricted to the sitting position and it may be speculated if our findings can be applied to these situations.

Pulmonary artery pressure, pulmonary artery wedge pressure and central venous pressure were higher during PEEP ventilation. This is not surprising as PEEP exerts its major cardiovascular effects by increasing intrathoracic pressure. When patients were moved from the supine to the sitting position, vascular pressures decreased regardless of whether or not PEEP was applied, mirroring the decrease in venous blood return to the heart and diminished filling of the pulmonary circulation. These data are consistent with previous results demonstrating in spontaneously breathing supine humans assuming the sitting position that intrathoracic blood volume decreased by approximately 300–500 ml with a decrease in both pulmonary artery wedge and central venous pressures. Techniques which are used to maintain filling pressures in the sitting position such as anti-gravity suits or high volume management were not used in this study. However, filling pressures measured in our patients did not differ from those reported in studies using anti-gravity suits. As the increase in filling pressures induced by anti-gravity suits has been reported to be only transient in adults we rarely use anti-gravity suits in our department. Whether PEEP may be effective in decreasing the incidence of VAE in patients with filling pressures higher than reported so far is beyond the scope of this investigation.

Depletion of the central circulation was also the likely reason for the decrease in cardiac output observed in both groups after patients were seated. However, in the seated position cardiac output in patients who underwent conventional ventilation was not significantly different compared with that during ventilation with PEEP. Presumably, this reflects the pronounced effect of positioning compared with PEEP ventilation or, conversely, the small additional circulatory depressant effect of PEEP in the seated position. Possibly, a much greater increase in PEEP may change this pattern but this has not been evaluated.

While PEEP increased both pulmonary artery wedge pressure and central venous pressure, it failed to affect the pulmonary capillary wedge pressure: central venous pressure gradient. In contrast, Perkins and Bedford reported that 10 cm H2O of PEEP caused reversal of this pressure gradient in the seated position. However, their results were from only 11 patients, with seven (64%) apparently showing a higher central venous than pulmonary artery wedge pressure during application of PEEP. In contrast, Zasslow and colleagues observed a higher central venous than pulmonary artery wedge pressure in only one of 12 patients whose lungs were ventilated with 20 cm H2O of PEEP in the seated position. Therefore, with only 11, 12 or six patients per study group, the results of these studies are inconsistent and may have led to misrepresentation of the risk of developing a negative pulmonary capillary wedge pressure:central venous pressure gradient.

Pulmonary vascular resistance increased significantly only in seated patients in the PEEP group but was not affected by PEEP application in the supine position or by positioning in those ventilated conventionally. Similar to the unchanged pulmonary vascular resistance observed in our conventional ventilation group when seated, pulmonary vascular resistance did not increase significantly in anaesthetized humans whose lungs were ventilated without PEEP when placed in the upright position. Most likely, pulmonary vascular resistance increased in our seated patients who underwent ventilation with 10 cm H2O of PEEP because of the additional impairment of pulmonary vascular filling by positioning, possibly causing smaller pulmonary vascular dimensions and hence increased flow resistance.

The data of this prospective, randomized study in a large number of patients demonstrated that ventilation with 10 cm H2O of PEEP failed to decrease the incidence of VAE and did not alter the sensitivity of monitoring devices to detect VAE. Furthermore, both PEEP and seated positioning impaired cardiovascular performance, with a small cardiac output measured in seated patients whose lungs
were ventilated with PEEP. However, positioning per se appears to have had a much greater effect on cardiac output and pulmonary vascular resistance than PEEP. We suggest therefore that routine use of 10 cm H2O of PEEP during neurosurgery in the seated position has no apparent benefit and cannot be recommended.

References


