Prevention of venous air embolism in paediatric neurosurgical procedures performed in the sitting position by combined use of MAST suit and PEEP

P.-G. MEYER, H. CUTTAREE, B. CHARRON, M.-M. JARREAU, A.-C. PERIE AND C. SAINTE-ROSE

Summary
We studied 60 children undergoing neurosurgical procedures in the sitting position. Routine monitoring included ECG, pulse oximetry, invasive arterial pressure, in particular mean arterial pressure (MAP), and right atrial pressure (RAP). Children were allocated to two groups. In group B lower body positive pressure and positive end-expiratory pressure (PEEP) were used for preventing venous air embolism (VAE). In this group, antishock trousers (MAST suit) were adjusted in supine children. After induction of anaesthesia, different positions were studied: supine and sitting before MAST suit inflation, sitting with MAST suit inflated up to a pressure of 40 mm Hg in the lower compartments and 30 mm Hg in the abdominal compartment, and finally a combination of lower body positive pressure and PEEP of 8–10 cm H$_2$O. In group A no MAST suit or PEEP was used. Continuous monitoring of end-tidal carbon dioxide pressure throughout ($P$E$_{CO_2}$) was used to detect VAE. In order to evaluate the transmission of pressures from the right atrium to the veins at the base of the skull, jugular bulb venous pressure (JBVP) was measured in 20 patients by retrograde catheterization. The incidence of VAE was compared in the two groups. On placing children into the sitting position, a significant decrease in RAP and JBVP was noted without significant changes in MAP in the two groups. Inflation of the MAST suit induced a dramatic increase in RAP and JBVP, reinforced by addition of PEEP. There was a strong positive relationship between RAP and JBVP. There were no deleterious side effects or differences between the two groups in peroperative blood product requirements or surgical general conditions. VAE was not noted in group B, compared with a 26% incidence of peroperative VAE detected by a decrease in $P$E$_{CO_2}$ of at least 0.4 kPa resulting in significant cardiovascular changes in group A. The combination of PEEP and lower body positive pressure could be a satisfactory method of preventing VAE. (Br. J. Anaesth. 1994; 73: 795–800)

Key words

The use of the sitting position for neurosurgical procedures has been the subject of controversy [1, 2]. The occurrence of venous air embolism (VAE) and its related complications has received considerable attention in patients undergoing neurosurgical procedures in the sitting position. The overall incidence of VAE in children, as in adults, is 30–45% in most reports [3, 4]. Cardiovascular complications related to VAE are much more common in children than in adults [5, 6]. In small children significant VAE results most commonly in severe hypotension and cardiac arrhythmias. The aim of this study was to evaluate the safety and efficiency of a method of preventing VAE using military antishock trousers (MAST suit) and PEEP, in a paediatric population undergoing neurosurgical procedures in the sitting position.

Patients and methods
After obtaining institutional Ethics Committee approval and informed parental consent, we studied ASA II or III children undergoing elective surgery in the sitting position. None had acute unstable intracranial hypertension requiring emergency surgery. Patients with a body weight of 10–35 kg were selected. Preoperative echocardiography for detection of patent foramen ovale was performed in only 40 of the 60 patients, because of lack of availability of echocardiographic equipment. Patients were selected for inclusion into the two groups during the preoperative evaluation by an anaesthetist who was not involved in further management of the patients (patients seen on even dates were allocated to group B and those seen on uneven dates to group A). After a 6-h fast, patients were premedicated with lorazepam 2 mg kg$^{-1}$ orally. In group B patients, a MAST suit (Jobst paediatric antishock trousers) was carefully adjusted but not inflated in awake patients in the supine position. Anaesthesia was induced with fentanyl 15 μg kg$^{-1}$ i.v. and either propofol 2.5–3 mg kg$^{-1}$ or thiopentone 5 mg kg$^{-1}$ i.v.

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Nasotracheal intubation was performed after inducing neuromuscular block with vecuronium 0.1 mg kg⁻¹. Controlled intermittent positive pressure ventilation was then commenced (Servo 900D, Siemens) and the ventilator settings were adjusted to maintain a stable end-tidal carbon dioxide pressure $P_{\text{ET}}^{\text{CO}_2}$ at 3.7–4.2 kPa. Anaesthesia was maintained with a continuous i.v. infusion of fentanyl 5–7 μg kg⁻¹ h⁻¹, and isoflurane (1 MAC) and 50% nitrous oxide in oxygen. Continuous ECG, body temperature, hourly urine output via an indwelling bladder catheter, $P_{\text{ET}}^{\text{CO}_2}$ (Normocap, Datex) and pulse oximetry (RGM 5250, Ohmeda) were recorded. A radial artery cannula and central venous catheter (inserted percutaneously via the right internal jugular vein with its tip positioned in the right atrium under x-ray control) were used in all patients. A jugular vein bulb catheter was inserted after retrograde catheterization of the internal jugular vein in patients in group B. Because of a highly reproducible transmission of venous pressure, only the first 20 patients in the study were monitored for jugular bulb venous pressure (JVBVP). Catheterization was performed as described previously with a 1-mm outer diameter, 15-cm length polyethylene catheter [7] and correct positioning at the base of the skull was controlled by x-ray. All catheters were connected to disposable pressure transducers (Biosec) and an electronic amplifier with a multichannel recorder (66 S monitor, Hewlett-Packard). Transducers were positioned at the level of the mid-axillary line when patients were supine and at the level of the fourth intercostal space in the sitting position.

Right atrial pressure (RAP), mean arterial pressure (MAP) and JVBVP (with zero level adjusted at the external auditory meatus monitored in patients with a retrograde catheter) were recorded after a 5-min steady-state period at each step of the experimental procedure and then every 30 min during the entire surgical procedure. The study included two baseline haemodynamic measurements for patients in both groups: control in the supine position (T1), patients were then placed in the sitting position with the head anteroflexed and fastened in a pin holder (T2). In group B patients, two additional situations were analysed. The MAST suit was inflated to a pressure of 40 mm Hg beginning with the lower limb compartments. The abdominal compartment was then inflated and special attention was paid to careful adjustment of the device and possible changes in tidal volume or maximum inspiratory peak pressure recorded by the ventilator monitoring unit, up to a pressure of 25–30 mm Hg (T3). MAST suit pressures were controlled continuously by the manometers integrated in the device. After a 10-min period of stabilization, a PEEP of 8–10 cm H₂O was introduced (T4). Serial arterial blood-gas measurements were performed at each step and then at least hourly or as clinically indicated.

Diagnosis of VAE was based on capnography. The capnogram was analysed continuously using an inline, two-channel, low speed (10 cm h⁻¹) writer connected to the capnograph. The first channel displayed mean $P_{\text{ET}}^{\text{CO}_2}$ continuously and the second displayed instant $P_{\text{CO}_2}$ variations during the ventilatory cycle. Criteria used for VAE detection were instability in the alveolar plateau of the capnogram lasting at least three ventilatory cycles and abrupt decreases of at least 0.4 kPa in mean $P_{\text{ET}}^{\text{CO}_2}$ without evidence of preceding acute events such as abrupt blood loss or recent changes in ventilatory settings. Jugular venous compressions were performed in all patients when requested by the surgeon. The opinion of the surgical team on surgical exposure, "brain tightness" and dural venous tension was noted. Estimated blood loss and blood product requirements based on serial determinations of packed cell volume were registered. At the end of the surgical procedure, while the patient was still in the sitting position and after discontinuation of PEEP, the MAST suit was deflated progressively (beginning with the abdominal compartment) over 10 min. Blood samples were obtained for measurement of serum creatine phosphokinase (CPK) and arterial blood-gas concentrations and chest x-rays were performed. Mechanical ventilation was maintained and the patients observed in the ICU with special attention to possible compression injuries of the extremities. Another serum CPK measurement was performed 12 h after completion of surgery.

Haemodynamic values were compared by multivariate analysis with ANOVA and unpaired $t$ test for comparison of means when appropriate. The relationship between JVBVP and RAP was analysed by simple linear regression and correlation analysis. Data are expressed as mean (SD). Statistical significance was considered for $P < 0.05$.

Results

We studied 60 patients aged 2–13 yr (mean 7 yr) with a body weight of 10–35 kg (mean 23.6 (SD 6.8) kg), referred for posterior fossa tumour excision (46 patients), cervical laminectomy and suboccipital craniotomy (four patients), pineal tumour (six patients) or posterior fossa vascular malformation excision (four patients). The characteristics of the patients were comparable in the two groups (table 1). After placing patients in the sitting position, RAP decreased significantly in all patients from a mean of 3.44 (SD 1.9) to 0.68 (1.6) mm Hg ($P < 0.0001$), with 16 of 60 patients (26%) having a sub-atmospheric RAP level in the sitting position. No further significant changes in RAP were noted in group A. In group B, when the MAST suit was inflated (T3), a dramatic increase in RAP was noted within 5 min.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Patient data (mean (SD) [range] or number)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>7.7</td>
</tr>
<tr>
<td>[2.5–15]</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
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<tr>
<td>Surgical disease</td>
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<td>Pineal tumour</td>
<td>4</td>
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<tr>
<td>Vascular disease</td>
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<tr>
<td>Cervical disease</td>
<td>4</td>
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</table>
Prevention of venous air embolism

Mean (SD) changes in right atrial pressure (RAP) in groups A (---) and B (---) in the supine and sitting positions and with the combined use of a MAST suit and PEEP.

Figure 1

Correlation between jugular bulb venous pressure (JBVP) and right atrial pressure (RAP) (simple linear regression: JBVP = 0.973 RAP - 0.661; \( r^2 = 0.89 \)).

Figure 2

Mean (SD) changes in mean arterial pressure (MAP) in groups A (---) and B (---) in the supine and sitting positions and with the combined use of a MAST suit and PEEP.

Figure 3

Table 2 Peroperative blood products transfused in groups A and B

<table>
<thead>
<tr>
<th>Transfused (n/total (%))</th>
<th>Group A</th>
<th>Group B</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transfused (n/total (%))</td>
<td>16/30 (53)</td>
<td>24/30 (80)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Mean (SD) volume transfused (ml)</td>
<td>14.4 (5.6)</td>
<td>13.4 (4.03)</td>
<td>ns</td>
</tr>
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</table>

of full inflation. Mean RAP at this point was 9.5 (1.8) mm Hg. An increase was noted in all patients whatever their initial RAP value in the sitting position (T2). PEEP amplified the increase in RAP significantly (fig. 1). This elevated RAP was sustained with little change as long as the MAST suit was kept inflated. A strong linear relationship was found between RAP and JBVP (fig. 2). Except for a slight insignificant decrease in MAP after placing patients in the sitting position, there were no significant variations in MAP during the study period (fig. 3). Inflation of the MAST suit and PEEP increased peak inspiratory pressure to mean 26 (4) cm H\(_2\)O.

There were no side effects (reduction in tidal volume delivered by ventilator, acute changes in arterial blood–gas tensions or barotrauma) related to this increased airway pressure. VAE was detected in 26 % of children in group A. These VAE resulted in an abrupt decrease in mean \( P\dot{E}_\text{CO}_2 \) of 0.68 (0.95) kPa. Instability of the alveolar plateau of the instant capnogram was noted during episodes of VAE and \( P\dot{E}_\text{CO}_2 \) remained decreased for at least 10 ventilatory cycles in all patients. Two patients sustained more than two episodes of VAE during the same procedure. After treatment, mean \( P\dot{E}_\text{CO}_2 \) increased gradually and reached its initial value in several minutes. Severe hypotension (decrease in MAP of at least 25 mm Hg) immediately after the beginning of the VAE occurred in all patients with VAE. This hypotension was sustained as long as capnographic evidence of VAE persisted. Cardiac arrhythmias were noted in three patients and severe bronchospasm in two. All of these signs resolved after treatment of VAE including bilateral jugular venous compression, with 100 % oxygen ventilation attempts to aspirate venous air through the right atrial catheter and prompt surgical haemostasis. No additional therapeutic measures such as vascular loading or vasopressor infusions were used to restore haemodynamic status. In this group, one patient with severe VAE had unexpected delayed recovery with coma and severe residual disability, possibly related to systemic air embolism. There was no echographic evidence of a patent foramen ovale in this patient. In group B, no patient had unexpected arterial pressure instability or capnographic evidence of VAE. There was no difference in transfused blood fluids between the two groups (13.4 (4.03) in group B vs 14.4 (5.6) ml kg\(^{-1}\) in group A) (table 2). No surgical side effects related to the use of the MAST suit and PEEP were noted. Peroperative urine output was not affected by the use of PEEP and always exceeded 1 ml kg\(^{-1}\) h\(^{-1}\).

Serum CPK concentrations increased uniformly 12 h after operation to a peak of 540 (120) iu litre\(^{-1}\) (normal range 15–110 iu litre\(^{-1}\)). No significant variation in CPK concentrations was noted between the two groups. No compression injuries of the extremities were noted during the postoperative period.

Discussion

Peroperative VAE is at least as frequent in children as in adults. A 66 % incidence of VAE, detected by echocardiography, has been reported in small chil-
dren referred for craniosynostosis repair in the prone position [6]. Possible explanations for this high incidence include the high sensitivity of peroperative echocardiography used for detection of VAE and the high incidence of air aspiration through opened vessels in the skull of small children with a large head in proportion to body size. Using precordial Doppler for diagnosis in sitting children, a 30% incidence of VAE was reported with a 69% incidence of related cardiovascular complications [12]. This incidence is approximately the same as that observed in our study. The Doppler seems to be one of the most sensitive techniques for detection of VAE, but electrocautery frequently affects its function and correct positioning may be difficult in small sitting children [6]. Transoesophageal echocardiography with coloured flow Doppler is the most efficient way to detect minor VAE, but its use in children weighing less than 25 kg is very limited. Rapid response capnography has fewer sources of interference or operator malfunction than echography and precordial Doppler. Its sensitivity (0.4 ml kg⁻¹ min⁻¹) is adequate to detect significant VAE [8] that may endanger the patient. This is particularly important in children in whom bubbles in a relatively small right atrium may result in more frequent severe cardiovascular reactions than in adults.

There are potential problems associated with methods of preventing VAE in the sitting position, including effects on surgical conditions. The main reasons for using the sitting rather than the supine position are: improved surgical exposure, improved venous blood and CSF drainage; reduced incidence of "brain tightness", reduced blood loss and a lower incidence of peroperative transfusion [3, 4]. Methods that increase dural sinus pressure (DSP) might be expected to modify surgical conditions, reducing the theoretical advantages of the sitting position. The other potential problem is that increasing central venous pressure may be a disadvantage. With only slight negative pressure, venous blood from the head flows mainly through collapsible vessels that are not affected by increased central venous pressure [9].

Unpredictable effects on DSP in these circumstances have been noted and monitoring of JBVP has been proposed as an argument for the choice of the best suitable methods in individual patients [10]. The most efficient method of increasing DSP in sitting children is bilateral jugular venous compression [9]. It is considered the only consistent method of increasing DSP above atmospheric pressure whatever the pre-compression pressure [10]. However this method should be considered as a therapeutic measure rather than prophylaxis against VAE. Fluid loading has been used, but its efficiency in increasing RAP appears to be limited and transient [11] and the consequences of fluid loading on postoperative cerebral oedema in neurosurgical practice may be harmful.

Lower body positive pressure increases blood venous flow, right atrial dimension, RAP and central venous pressure when used with low inflation pressures in healthy normovolaemic patients [12, 13]. Its effect on the distribution of blood volume seems to be small [14] and the main physiological effect is an increase in after load by increased sympathetic activity and concentration of plasma atrial natriuretic factor [13].

MAST suit inflation, when used in sitting adults, induces an immediate increase in RAP and central venous pressure that is transmitted to the dural sinus via the non-collapsed veins of the neck [15]. Its effects on DSP have been questioned in patients with a slightly negative initial DSP in the sitting position and the increase in RAP induced by MAST suit inflation in adults has been reported as transient [15]. In our paediatric experience, a dramatic increase in RAP was noted immediately after MAST suit inflation in all patients and the increased RAP was always sustained as long as the MAST suit was kept inflated. In patients with a retrograde catheter, there was a positive JBVP regardless of the initial level in the sitting position in all patients after MAST suit inflation. There was a strong linear relationship between JBVP and RAP. This may be explained, in part, by the small number of patients in our study. Another possible explanation is that, despite the linear positive correlation with DSP, especially in children, JBVP is an extracranial pressure that is not strictly identical to DSP. In children, the distance between the jugular bulb and the lateral dural sinus is about 2 cm. The distance between the right atrium and jugular bulb does not exceed 15 cm. It would be surprising therefore if sub-atmospheric DSP values occurred after inflation of the MAST suit in spite of highly positive values of JBVP. The MAST suit has been shown to cause a moderate increase in intracranial pressure (ICP) without deleterious effects on cerebral perfusion pressure [16, 17]. This effect on ICP could be considered as a contraindication to the method in the presence of preoperative, unstable ICP. In sitting, normovolaemic children, MAST suit inflation reduces the degree of instability of MAP related to the position, thereby maintaining cerebral perfusion pressure. Because of the effects of MAST suit on CSF drainage, its use should probably also be limited in non-shunted acute hydrocephalus.

Because of the prolonged pressure exerted on the lower body, the MAST suit has been implicated in compartment syndromes and muscular ischaemia [18]. However, we did not observe any evidence of compression injuries and the mild increase in serum CPK concentration noted in the two groups of patients was small. This increase in muscle enzymes has been noted by others after paediatric surgical procedures without compression of the lower body [19].

PEEP has been proposed as a means of increasing DSP [20]. It has been clearly demonstrated that clinical levels of PEEP, when used alone, cannot increase DSP sufficiently to prevent VAE and it is ineffective as prophylaxis against VAE when the initial central venous pressure measured in the sitting position is relatively high [10, 11, 21]. Moreover, it may be deleterious decreasing venous blood return and cardiac output [22], especially in the presence of acute blood loss. The MAST suit prevents such deleterious effects of PEEP as peripheral venous pooling, decreased cardiac output, hypotension and
decreased cerebral perfusion pressure, especially in the sitting position [23]. The combination of increased surrounding atrial pressure induced by PEEP and increased intraluminal atrial pressure (induced by compression of the lower body) may be the best method of preventing air entry through the skull and dural venous vessels in sitting children. In this study, elevated RAP and JBV induced by the combination of these two methods prevented significant VAE, as detected by capnography, in all patients. There was no difference in surgical conditions between the two groups and bilateral jugular venous compression was requested in the same way by surgeons for haemostatic control. Most importantly, there was no significant difference in the transfused blood volume between the two groups.

Paradoxical systemic air embolism (PAE) is a dramatic, although rare, complication of air embolism. It is caused usually by massive air entry from the right to the left atrium via a patent foramen ovale. As there is a 34% incidence of asymptomatic patent foramen ovale in the general population [24], with a 30–45% incidence of peroperative VAE in the sitting position, the theoretical risk of peroperative PAE has been estimated as 10% [25].

The normal positive pressure difference from the left to the right atrium has been reported to reverse after 1 h in the sitting position [26], but in the majority of patients with a positive right to left atrial pressure difference, the pressure is not great enough to force air bubbles through a patent foramen ovale. PEEP has been implicated in reversal of the normal difference between left and right atrial pressures predisposing to PAE if VAE occurs [27]; an observation used as another argument for avoiding PEEP alone as prophylaxis. However, recent studies have suggested that PEEP does not reverse the normal inter-atrial gradient, but the sudden increase in venous return when PEEP is discontinued may be responsible for the abrupt increase in right (relative to left) atrial pressure [25,28]. The MAST suit increases right and left atrial pressures to a similar extent [13,19] and therefore the combination of lower body compression and PEEP may be expected to preserve the normal inter-atrial pressure gradient. It has been suggested that echocardiography be used to detect patent foramen ovale before surgical procedures are performed in the sitting position and the presence of a patent foramen ovale should be considered as a contraindication to this position [29]. However, the best way of preventing PAE is to avoid the occurrence of VAE and we suggest that this is achieved by the combination of a MAST suit and PEEP.

References
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