Monitoring lung compliance and end-tidal oxygen content for the detection of venous air embolism

J. KYTTÄ, T. RANDELL, P. TANSKANEN, Y. KAJIMOTO AND P. H. ROSENBERG

Summary
Venous air embolism (VAE) is a recognized complication of surgery performed with the patient in the sitting position, but it occurs also during other operations. We report two cases of VAE, associated with a notable decrease in dynamic lung compliance, detected by side-stream spirometry. Based on these cases, an experiment with 10 pigs was designed to evaluate the usefulness of side-stream spirometry in the diagnosis of VAE. Three doses of air (0.5, 1.0 and 2.0 ml kg\(^{-1}\)) were injected via the proximal part of a 5-French gauge pulmonary artery catheter. Only the largest dose was followed by haemodynamic deterioration. Significant increases in end-tidal oxygen content and decreases in dynamic lung compliance were detected with all doses of air together with conventional signs of VAE, that is increases in pulmonary artery pressures and arterial carbon dioxide tensions, and decreases in end-tidal concentration of carbon dioxide. We conclude that continuous monitoring of end-tidal oxygen concentration and side-stream spirometry offers valuable supplements to other monitoring techniques in the detection of VAE. (Br. J. Anaesth. 1995; 75: 447–451)

Key words

Patients
PATIENT NO. 1
A 31-yr-old male, weighing 75 kg, was undergoing extirpation of an intracranial arteriovenous malformation after incomplete embolization of the anomaly. At the preoperative visit, the patient was found to be slightly disoriented but otherwise in good physical condition. The preoperative chest x-ray and ECG were normal.

After induction of anaesthesia with fentanyl and thiopentone, tracheal intubation was performed, facilitated with a non-depolarizing neuromuscular blocker. Mechanical ventilation was commenced with a Servo 900 C ventilator (Siemens-Elema, Sweden). Minute ventilation was set to 8 litre min\(^{-1}\) with a ventilatory frequency of 13 b.p.m. The duration of the inspiratory phase was 25 % and the inspiratory pause was 10 % of the respiratory cycle. A positive end-expiratory pressure (PEEP) of 6 cm H\(_2\)O was used. These settings resulted in an end-tidal carbon dioxide concentration \(\text{[ECO}]\) of 4.1 %. Anaesthesia was maintained with 0.7 % isoflurane in 100 % oxygen, and pancuronium was administered as needed.
After induction of general anaesthesia and tracheal intubation, mechanical ventilation was started with a Servo 900 C ventilator. Minute ventilation was 7.5 litre min$^{-1}$ and ventilatory frequency 12 b.p.m. PEEP was not applied. Monitoring of the patient was similar to that in patient No. 1. Anaesthesia was maintained with 0.5 % isoflurane in 100 % oxygen.

There were no marked cardiovascular changes associated with positioning of the patient from the supine to the sitting position. A pressure of 40 mm Hg was applied to the pneumatic anti-shock trousers.

Baseline blood-gas analysis revealed $P_{\text{ACO}_2}$ of 3.6 kPa and $P_{\text{ECO}_2}$ 3.7 % (table 1). Fifteen minutes after the start of surgery, air embolization was suspected because $P_{\text{ECO}_2}$ decreased suddenly to 1.7 % and carbon dioxide partial pressure in arterial blood was 4.1 kPa. Doppler sounds were disturbed frequently by electrocautery. Lung compliance was noted to change from 41 to 33 ml cm H$_2$O$^{-1}$ (19.5 % decrease). A PEEP of 4 cm H$_2$O was immediately applied.

The values returned close to baseline levels within 1 h.

### Materials and methods

The study was approved by the Animal Use and Care Committee of the hospital. We studied 10 domestic pigs, weighing 15–20 kg. The animals were unpremedicated. A vein of one ear was cannulated for obtaining samples for blood-gas analysis. A pulmonary artery catheter (size 5-French gauge) was introduced through the right jugular vein. Pulmonary artery pressure and central venous pressure were monitored continuously.

<table>
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<th>Patient No. 1</th>
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<th>Immediately after VAE</th>
<th>1 h after VAE</th>
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<td>$S_{\text{O}_2}$ (%)</td>
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<td>98</td>
<td>99</td>
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<td>69</td>
<td>78</td>
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### Table 1

Respiratory variables during anaesthesia before and after venous air embolism (VAE) in two patients undergoing craniotomy in the sitting position. $S_{\text{O}_2}$ = Haemoglobin saturation of oxygen, $P_{\text{ACO}_2}$ = end-tidal carbon dioxide concentration, $P_{\text{ECO}_2}$ = peak arterial carbon dioxide tension, $P_{\text{Peak}}$ = peak airway pressure, $C$ = dynamic lung compliance,$V_{E}$ = expired minute volume measured with the side-stream spirometry, PEEP = positive end-expiratory pressure. *New episode of VAE occurred before a sample for blood-gas analysis was obtained.

After induction of anaesthesia, a radial artery was cannulated for continuous arterial pressure monitoring and for obtaining blood samples for frequent analysis of blood-gas tensions. A precordial Doppler ultrasound device (Meda Sonics, USA) was used for detection of VAE. A side-stream spirometer (Datex Ultima, Instrumentarium, Finland) was connected to the ventilator tubing, giving continuous information on lung compliance, tidal volume and minute volume. The ventilator used was the Servo 900 C (Siemens Elema, Sweden). The inspiration–expiration ratio was set to 25 % and the pause time to 10 %. Ventilatory frequency was set to 20 b.p.m. and tidal volume was adjusted to maintain $P_{\text{ECO}_2}$ between 4.0 and 4.5 %.

A side-stream spirometer (Datex Ultima, Instrumentarium, Finland) was connected to the ventilator tubing, giving continuous information on lung compliance, tidal volume and minute volume. The difference in concentration of oxygen in the inspiratory and expiratory gas ($O_2(1-E)$), the inspiratory and end-tidal isoflurane concentration and $P_{\text{ECO}_2}$ were analysed continuously.

One of the femoral arteries was cannulated for continuous monitoring of arterial pressure and for obtaining samples for blood-gas analysis. A pulmonary artery catheter (size 5-French gauge) was introduced through the right jugular vein. Pulmonary artery pressure and central venous pressure were monitored continuously.

A 44-yr-old woman with lymphatic leukaemia was undergoing extirpation of a cerebral tumour. The physical condition of the patient was good.

### Materials and methods

The study was approved by the Animal Use and Care Committee of the hospital. We studied 10 domestic pigs, weighing 15–20 kg. The animals were unpremedicated. A vein of one ear was cannulated and an infusion of Ringer's acetate was started. Glycopyrronium 10 µg kg$^{-1}$ was administered, and anaesthesia was induced with a slow dose of thiopentone. Pancuronium 0.2 mg kg$^{-1}$ was used to facilitate tracheal intubation. Before instrumentation, fentanyl 5 µg kg$^{-1}$ was given i.v. Anaesthesia was maintained with isoflurane (end-tidal concentration 1.0 %) in oxygen and air 1 : 1 during cannulation, and thereafter with 0.5 % isoflurane in 100 % oxygen. Pancuronium in 1-mg increments was given as needed. The anaesthetic technique was similar to that used in the patients operated on in the sitting position.

The ventilator used was the Servo 900 C (Siemens Elema, Sweden). The inspiration–expiration ratio was set to 25 % and the pause time to 10 %. Ventilatory frequency was set to 20 b.p.m. and tidal volume was adjusted to maintain $P_{\text{ECO}_2}$ between 4.0 and 4.5 %.
Lung compliance and $F_{\text{O}_2}$ in air embolism

Haemoglobin oxygen saturation ($S_{\text{O}_2}$) and ECG were monitored continuously. A precordial Doppler ultrasound device (Meda Sonics, USA) was used for detection of VAE.

Baseline values of arterial, pulmonary artery and central venous pressures, cardiac rhythm and rate, $S_{\text{O}_2}$ and respiratory variables were recorded and a sample for analysis of blood-gas tensions was obtained. Then air was injected via the proximal port of the pulmonary artery catheter at a speed of 1 ml s$^{-1}$. Haemodynamic and respiratory variables were recorded at the end of and 30 s after, injection, then at 1-min intervals for 10 min and every 2 min for another 10 min. Blood-gas tensions were analysed 2 min after injection of air.

Each animal was tested with three injections of air. The volumes and order of the injections were 0.5, 1.0 and 2.0 ml kg$^{-1}$, and they were given at least 30 min apart. Our pilot study showed that these volumes did not cause irreversible haemodynamic collapse, and that the changes observed in the haemodynamic and respiratory variables, and also in the Doppler ultrasound, subsided within 20–30 min.

After the experiment, the animals were killed with a toxic dose of barbiturate and i.v. injection of air 10 ml kg$^{-1}$.

Data were analysed with ANOVA or paired Student’s $t$ test. If significant differences were observed, Fisher PLSD test was used for post hoc analysis. The results are presented as mean (SEM). $P$ less than 0.05 was considered statistically significant.

Results

The animals survived all injected doses of air. No significant changes were observed in heart rate with any of the doses. After the lowest dose, mean arterial pressure remained stable, whereas with doses of 1.0 and 2.0 ml kg$^{-1}$, significant decreases in pressures were noted immediately and 30 s after the end of injection, respectively (fig. 1). Significant increases in pulmonary artery pressures and central venous pressure were observed immediately after injection with all doses of air (fig. 2a, b, c).

Arterial carbon dioxide partial pressure increased significantly in all groups (table 2). End-tidal carbon dioxide concentration decreased significantly immediately after the end of air injection (fig. 3). Decreases were observed in the inspiratory to expiratory oxygen concentration difference, but the changes did not appear until 1 min after the end of injection of air (fig. 4).

Arterial oxygen partial pressure decreased significantly after injection of all volumes of air (table 2).
There were no significant changes in $2Oa$. The lowest values ranged from 95% to 99% with 0.5 ml kg$^{-1}$ of air, from 95% to 100% with 1.0 ml kg$^{-1}$ of air and from 80% with 2.0 ml kg$^{-1}$ of air.

Changes in lung compliance were moderate, but reached statistical significance (fig. 5). With a dose of air above the baseline value for 10 min. after injection (14.3 (2.6) to 15.4 (3.0) cm H$_2$O at 30 s after the end of the injection, from 13.2 (2.5) cm H$_2$O at 3 min after the end of injection, from 12.5 (2.5) to 1.0 ml kg$^{-1}$ are neither specific nor quantitative [2, 3, 6]. Trans-

Discussion
The methods commonly used for diagnosis of VAE are neither specific nor quantitative [2, 3, 6]. Trans-

oesophageal echocardiography (TEE) and precordial ultrasonic Doppler have been found to be the most sensitive indicators of VAE [6, 8]. In our reported cases, Doppler ultrasound was unreliable because of the noise caused by the frequent use of the electrocautery. TEE requires expertise and is not feasible in our institution. In our patients, the diagnosis of VAE was confirmed by the marked discrepancy between $P_{aCO_2}$ and $P_{aCO_2}$. However, measuring the difference between arterial and end-tidal carbon dioxide concentration cannot be continuous and there is an inevitable time delay.

In our study, the largest dose of air was followed by marked depression of the circulation, which is seldom seen in the clinical setting. However, none of the measured variables showed a clear correlation with the amount of air injected. The differences in the baseline values of compliance and pulmonary pressures suggest that some air had remained in the pulmonary circulation. As this was anticipated, the order of injecting the different volumes of air was not
randomized. The methods used in this study do not allow quantitative analyses but our results reflect the effect of cumulative VAE.

It has been reported that VAE can result in an increase in airway pressure [9], but in the study of Dash and colleagues, this method was found to be inferior to monitoring of \( F_{\text{\text{CO}}_2} \) in the early detection of VAE [3]. We observed consistent increases in airway pressure in the pigs. The discrepancy between our results and those of Dash and colleagues [3] may be explained by the method of monitoring. The changes in lung compliance occurred concomitantly with increases in airway pressures.

In an earlier study, static lung compliance was found to be a late sign of VAE, becoming apparent only occasionally with large doses of air [6]. We observed statistically significant decreases in lung compliance in the pigs as early as 30 s after the end of injection of air, with even the smallest doses of air. However, the change was gradual and reached its peak several minutes after injection. Compliance was measured continuously in our study, and the side-stream method excludes the possible error caused by compliance of the respirator and tubing. This may explain the somewhat different results obtained by English and co-workers [6]. Other possible factors that can cause error in side-stream spirometry are humidity of the expired air, and the ratio of tidal volume to volume of the sample taken by the monitor. The former may have contributed to the differences in baseline values of compliance. The latter cause of error may be ignored, because the trend during each period was analysed, and the ratio of the volumes remained constant throughout.

The amount of injected air was small compared with the volume of the thoracic cavity, and therefore does not explain the decrease in lung compliance. However, VAE caused significant increases in pulmonary arterial pressures, indicating increased pulmonary vascular resistance. This may have resulted in pooling of blood in the pulmonary circulation and decrease in lung compliance.

Pulse oximetry cannot be considered an indicator of VAE. Oxygen extraction in the lungs, however, decreased markedly after air embolism, as indicated by the small difference in the concentration of inspired and expired oxygen. This change occurred concomitantly with the decrease in end-tidal concentration of carbon dioxide. Linko and Paloheimo have reported significant increases in \( F_{\text{\text{CO}}_2} \) and \( Pa_{\text{\text{CO}}_2} \) with a concomitant decrease in \( Pa_{\text{\text{CO}}_2} \) after alveolar hypoventilation in pigs [10]. Thus these variables considered together offer a method for differentiating between some important clinical problems during anaesthesia.

Regardless of the use of several monitoring techniques, the diagnosis of VAE remains unreliable and difficult. Episodes of air embolism have been reported during some orthopaedic operations performed in some positions other than sitting [4]. On such occasions, where routine monitoring is not focused on detection of VAE, the complications may not be diagnosed promptly. Therefore, monitoring of inspiratory to expiratory oxygen concentration, and dynamic lung compliance are valuable supplements to other monitoring techniques for the detection of pulmonary VAE.

References