Effects of the sitting position on the distribution of blood volume in patients undergoing neurosurgical procedures

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The use of the sitting position in neurosurgery is often associated with decreased arterial pressure (MAP) and stroke volume index (SVI). A shift in blood from the intra- to the extrathoracic compartment may be responsible for this cardiovascular response. However, little is known of the amount of shift in blood volume after transfer from the supine to the sitting position. Therefore, we measured simultaneously changes in intrathoracic blood volume (ITBV) caused by a change in body position in anaesthetized patients. Measurements of cardiac index (CI), ITBV, pulmonary (PBV) and total circulating (TBV_{circ}) blood volumes were performed in the supine and sitting position. CI, ITBV, PBV and TBV_{circ} were measured using a thermo–dye dilution technique. Fluid input was restricted to 14 ml kg^{-1} before induction of anaesthesia. Change in body position caused a significant decrease in ITBV and was accompanied by a significant decrease in CI, SVI and MAP. Changes in ITBV correlated (r=0.78) with changes in SVI. Thus a change in blood volume distribution between the intra- and extrathoracic compartment occurred after a change from the supine to the sitting position. Indicator dilution enables quantification of this shift and may be helpful in guiding fluid therapy in selected patients.

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Although there is much debate about the advantages of the sitting position in neurosurgery, this position is used routinely in many institutions for patients undergoing surgery in the posterior fossa.[1 2] In addition to the increased risk of air embolism, the sitting position is often associated with haemodynamic instability, in particular a decrease in mean arterial blood pressure, stroke volume index and cardiac index.[3–7] It can be assumed that the cardiovascular effects of the sitting position are caused mainly by a reduction in cardiac preload.[7] However, no data are available on the quantity of blood volume redistribution from the intra- to the extrathoracic compartment in anaesthetized patients undergoing neurosurgical procedures in the sitting position. Thus in this prospective, controlled clinical study, we have investigated the effects of change from the supine to the sitting body position on the distribution of blood volume using an indicator dilution technique.

Patients and methods

We studied 10 patients (seven females, three males, ASA II) undergoing elective neurosurgical procedures performed in the sitting position. The study was approved by the Institutional Review Board Committee and patients gave written informed consent. None of the patients had a history of cardiopulmonary, renal or liver disease and none was receiving cardiac medications before operation.

Premedication comprised flunitrazepam 1–2 mg orally on the evening before surgery and 1 h before induction of anaesthesia. After arrival, ECG leads II and V5 were attached to the patient (Sirecust 961, Siemens, Germany). Before induction of anaesthesia, infusions of crystalloid 7 ml kg^{-1} (Eufusol, Braun Melsungen, Germany) and colloid 7 ml kg^{-1} (Gelafundin, Braun Melsungen, Germany) were administered via a peripheral venous line over a period of 30 min. No more fluids were given during the study, except for 200 ml of ice-cooled solution for thermodilution and dye dilution measurements. Under local anaesthesia a subclavian venous line (12 F, Arrow Int., Reading, PA, USA) was inserted and its tip advanced to lie in the right atrium. The correct position of the right atrial catheter was verified by transoesophageal echocardiography when anaesthesia was instituted. For measurements of cardiac output (CO), intrathoracic blood volume (ITBV), pulmonary blood volume (PBV) and total circulating blood volume (TBV_{circ}), a combined 4-French gauge fibreoptic-thermistor catheter

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(Pulsiocath PV 2024, Pulsion Medical Systems, Germany) was inserted 40 cm up into the descending aorta via a 5-French gauge introducer (Arrow Int., Reading, USA) in the left femoral artery. The fibreoptic catheter was connected to a commercially available opto-electronic device (COLD-System, Pulsion Medical Systems, Germany) which allows simultaneous detection of thermodilution and dye dilution curves.[8 9]

General anaesthesia was induced with fentanyl 6 μg kg^{-1} and midazolam 0.1 mg kg^{-1}. Pancuronium 0.1 mg kg^{-1} was administered to facilitate tracheal intubation. Anaesthesia was maintained with fentanyl 10 μg kg^{-1} h^{-1} and midazolam 150 μg kg^{-1} h^{-1}. Vasoactive drugs or inhalation anaesthetics were not given. After tracheal intubation, patients' lungs were ventilated mechanically in a volume-controlled mode with a positive end-expiratory pressure (PEEP) of 5 cm H_2O. Respiratory minute volume was adjusted to maintain normocapnia (Pa_{CO_2} 4.8–6.0 kPa). Inspired oxygen fraction (FI_{O_2}) was 0.5. No further ventilatory adjustments were made during the study.

Standard haemodynamic variables, including heart rate (HR), mean arterial (MAP) and right atrial (RAP) pressures were obtained and indicator dilution measurements were performed under stable haemodynamic conditions after induction of anaesthesia (supine position) and after raising the patient to a 45° sitting position (sitting position). Pressure transducers were positioned at the midaxillary level when patients were supine and at the level of the fifth intercostal space anteriorly when they were sitting. Arterial and venous blood samples were obtained at each measurement to determine haemoglobin content, oxygen saturation (OSM-3 Hemoximeter, Radiometer, Denmark) and blood-gas analysis (ABL 500 Blood Gas Analyser, Radiometer, Denmark).

Thermodilution and dye dilution curves were obtained by triple bolus injection of 20 ml of ice-cooled saline solution and two additional bolus injections of 20 ml of ice-cooled indocyanine green dye (ICG) 1.25 mg ml^{-1} into the right atrium. Injections were spread randomly over the respiratory cycle. The resulting thermodilution and dye dilution curves obtained in the aorta were digitized and stored on a microcomputer (COLD-System, Pulsion Medical Systems, Germany). CO was assessed using the thermodilution technique. Intrathoracic blood volume (ITBV) was calculated from CO and the mean transit time of the indicator (mtt_{dye}) through the thoracic compartment of the circulation.[9]

$$ITBV = CO \times mtt_{dye} \text{ (ml)} \quad (1)$$

PBV was calculated from the product of the exponential downslope time ($t_{dt\ dye}$) of the dye curve obtained in the aorta and CO.[9] The downslope time is defined as the reciprocal value of the time constant characterizing the downslope of the indicator dilution curve.[9]

$$PBV = CO \times t_{dt\ dye} \quad (2)$$

Table 1 Data on blood gases and airway pressure during the study (mean (SD)). Hb=Haemoglobin, HCT=haematocrit, Sa_{O_2}=arterial oxygen saturation, Pa_{O_2}=arterial oxygen partial pressure, Pa_{CO_2}=arterial carbon dioxide partial pressure, *P*aw-peak=peak airway pressure, *P*aw-mean=mean airway pressure

	Supine position	Sitting position
Hb (g dl^{-1})	11.7 (1.5)	11.8 (1.7)
HCT (%)	35.8 (4.5)	35.8 (4.9)
Sa_{O_2} (%)	99.1 (0.5)	98.8 (0.9)
Pa_{O_2} (kPa)	34.9 (10.3)	28.3 (7.6)
Pa_{CO_2} (kPa)	5.7 (0.5)	5.4 (0.3)
*P*aw-peak (mbar)	24.8 (5.1)	24.4 (4.9)
*P*aw-mean (mbar)	10 (1.0)	9.9 (0.9)

Table 2 Haemodynamic variables (mean (SD)). HR=Heart rate, MAP=mean arterial pressure, RAP=right atrial pressure, CI=cardiac index, SVI=stroke volume index, SVRI=systemic vascular resistance index, ITBV=intrathoracic blood volume, PBV=pulmonary blood volume, TBV_{circ}=total circulating blood volume. *Significant influence of the sitting position ($P \leq 0.05$)

	Supine position	Sitting position
HR (beat min^{-1})	68 (5)	63 (4)*
MAP (mm Hg)	92 (14)	83 (18)*
RAP (mm Hg)	6 (2.6)	3 (3.2)*
CI (litre min^{-1} m^{-2})	2.4 (0.4)	1.8 (0.3)*
SVI (ml m^{-2})	36 (6)	29 (5)*
SVRI (dyn s cm^{-5} m^2)	2853 (359)	3509 (480)*
ITBV (ml m^{-2})	626 (97)	534 (89)*
PBV (ml m^{-2})	151 (17)	141 (15)
TBV (ml kg^{-1})	58 (10)	60 (13)

Total circulating blood volume (TBV_{circ}) was calculated simultaneously from the aortic dye dilution curves.[10] Complete mixing of the indicator in blood was assumed to occur after 80 to 150 s. The concentration of ICG at injection time ($C_{0(ICG)}$) was determined by monoexponential extrapolation of the dye dilution curve after complete mixing of indicator.[10] According to the principle of mass conservation, TBV_{circ} was calculated by the following equation[11]:

$$TBV_{circ} = m_{ICG}/C_{0(ICG)} \text{ (ml)} \quad (3)$$

m_{ICG}=amount of injected ICG

All volume- and flow-related variables were normalized to body surface area or body weight.

Statistical analysis

Results are expressed as mean (SD). Paired Student's *t* tests were used to compare data obtained before and after transfer from the supine to the sitting position. $P \leq 0.05$ was considered statistically significant. All statistical procedures were performed on a microcomputer using the SPSS/PC+ statistical software package.

Results

Mean age of patients was 47 (range 27–68) yr. Mean body weight and height were 69 (SD 10) kg and 168 (5) cm, respectively. Cardiopulmonary data and data on blood gases and haemoglobin content are presented in Tables 1 and 2 and Figures 1 and 2, respectively.

Haemoglobin content and blood gases remained constant

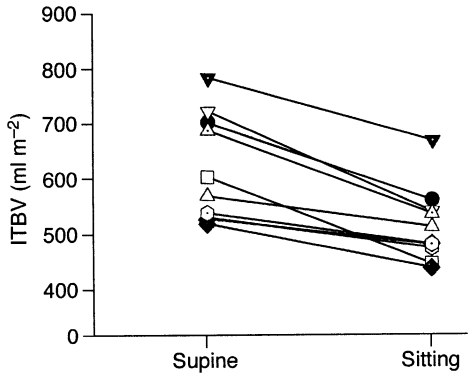

Fig 1 Relative changes in intrathoracic blood volume (ITBV) for individual patients. Each patient is depicted by a different symbol. In all patients a decrease in ITBV was observed. Mean difference of ITBV between supine and sitting positions was 14%.

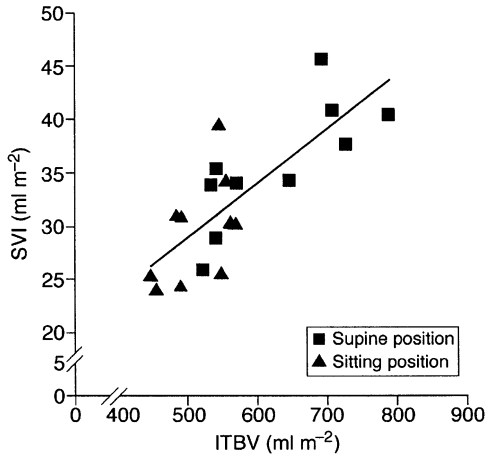

Fig 2 Correlation between relative changes in stroke volume index (SVI) and intrathoracic blood volume (ITBV) associated with transfer from the supine to the sitting body position in anaesthetized patients (r=0.78).

during the study (Table 1). MAP and RAP decreased significantly when the patient position changed from the supine to the sitting. Figure 1 summarizes our findings with regard to ITBV. Indicator dilution measurements demonstrated that the transfer to the sitting position resulted in a significant decrease in ITBV by 14% (Fig. 1) in CI and SVI by 24% and 17%, respectively (Table 2). In contrast, PBV remained almost unchanged. Systemic vascular resistance index (SVRI) increased by 14% (Table 2). Correlation analysis revealed a linear relationship between relative changes in SVI and ITBV (r=0.78) (Fig. 2). In contrast, there was no correlation between changes in RAP and changes in SVI (r=0.45). Circulating blood volume (TBV_{circ}) did not change significantly throughout the study. Mean and peak airway pressures did not differ between the supine and sitting positions (Table 1). No catheter-related complications or allergic reactions were observed during the study.

Discussion

In our study, we found that a shift in blood volume of 14% from the intra- to the extrathoracic space occurred after change from the supine to the sitting position, resulting in a significant decrease in CI by 24 %. Also, volume loading with a total of 14 ml kg^{-1} of crystalloid and colloid fluids could not entirely prevent the haemodynamic instability of this procedure.

The sitting position in neurosurgery is associated with specific risk factors.[1] Haemodynamic instability is often observed, in particular in patients with limited cardiac reserve.[7] Several studies have investigated the haemodynamic effects associated with transfer from the supine to the sitting position in healthy subjects or patients undergoing neurosurgical procedures.[5–7] In most studies, arterial hypotension, defined as a decrease in mean arterial pressure of >10% or a decrease in systolic arterial pressure of >20% was observed with an incidence of 5–32%.[3–5]

The choice of anaesthetic technique may have a significant influence on haemodynamic stability in anaesthetized patients in the sitting position.[7] Dalrymple, MacGowan and MacLeod observed a 37% decrease in CI when anaesthesia was maintained with nitrous oxide and fentanyl after raising into the sitting position.[6] The decrease in CI in our patients was less pronounced than in the study of Dalrymple, MacGowan and MacLeod. This is most likely because different fluid regimens were used.[6] More recently, Marshall, Bedford and Miller studied the cardiovascular response to the sitting position.[7] Patients received either enflurane–nitrous oxide, halothane–nitrous oxide, fentanyl–droperidol–nitrous oxide or morphine–nitrous oxide for maintenance of anaesthesia. A significant decrease in CI was observed in all groups after change from the supine to the sitting position. The combination of morphine–nitrous oxide caused the least change.[7] However, haemodynamic stability in this study may have been related to high plasma concentrations of catecholamines because of inadequate depth of anaesthesia. In our study, maintenance of anaesthesia was performed with fentanyl combined with continuous infusion of midazolam.

Our investigation differed from previous studies in two ways. First, we measured simultaneously direct volumetric variables of intra- and extrathoracic blood volume compartments to determine the net effects of the sitting position on blood volume distribution in humans. Second, fluid input was standardized and restricted to 7 ml kg^{-1} of a crystalloid and 7 ml kg^{-1} of a colloid solution before the first set of measurements.

Indicator dilution has gained acceptance for bedside measurement of intrathoracic, pulmonary and total circulating blood volume.[9 10 12 13] As ITBV primarily represents the blood volume of the heart and pulmonary circulation, it seems a better indicator of cardiac preload than filling pressures.[8 12 14 15] In our patients, a decrease in ITBV after repositioning was consistently accompanied by a decrease

in SVI whereas no linear correlation between RAP and SVI was observed. Thus measurement of ITBV before and after change in body position enables titration of volume therapy to optimize blood volume and SVI. This may be of clinical value in other situations in which blood volume shifts occur, such as inversed ratio ventilation and sepsis-induced vasodilatation.

Perkins-Pearson, Marshall and Bedford studied the effects of patient position and anaesthesia on right and left heart filling pressures using pulmonary artery catheters.[16] They found a decrease in PCWP in the sitting position whereas RAP remained unchanged, and they attributed this reduction to a decrease in pulmonary blood volume.[16] By directly measuring PBV, we could not verify the observation of Perkins-Pearson, Marshall and Bedford as PBV remained almost unchanged in our patients, whereas RAP decreased significantly. However, it is possible that the tip of the pulmonary artery catheters in their study may have become lodged in a part of the lung where PCWP reflects airway pressure rather than left atrial pressure.

In our study, fluid input was restricted to a total of 14 ml kg^{-1} of crystalloid and colloid solutions. As indicated by the decrease in ITBV, this amount of fluid was not able to compensate for the shift of blood from the intra- to the extrathoracic compartment. However, without any attempt to volume load the patients, the changes may have been more pronounced.

Because of the short half-life of the crystalloid solution, significant amounts of fluid may have been lost in the extravascular space, possibly influencing measurements of ITBV and PBV. However, measurements of TBV_{circ} did not differ significantly between values obtained before and after transfer from supine to sitting, suggesting that no significant loss of fluid from the intravascular compartment occurred during the study. The decrease in ITBV in our patients thus primarily quantified the amount of blood shifted from the intra- to the extrathoracic compartment.

In summary, we have demonstrated that intrathoracic blood volume decreased by 14% after transfer from the supine to the sitting position, whereas total circulating blood volume remained constant. This indicates a volume shift from the intra- to the extrathoracic compartment. Concomitant changes in arterial pressure and stroke volume index are thus caused primarily by a reduction in cardiac preload, which can be monitored by serial indicator dilution measurements of intrathoracic blood volume.

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