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Cerebral oxygen saturation and cardiac output during anaesthesia in sitting position for neurosurgical procedures: a prospective observational study

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Abstract

Background: Neurosurgical operations in the dorsal cranium often require the patient to be positioned in a sitting position. This can be associated with decreased cardiac output and cerebral hypoperfusion, and possibly, inadequate cerebral oxygenation. In the present study, cerebral oxygen saturation was measured during neurosurgery in the sitting position and correlated with cardiac output.

Methods: Perioperative cerebral oxygen saturation was measured continuously with two different monitors, INVOS® and FORE-SIGHT®. Cardiac output was measured at eight predefined time points using transoesophageal echocardiography.

Results: Forty patients were enrolled, but only 35 (20 female) were eventually operated on in the sitting position. At the first time point, the regional cerebral oxygen saturation measured with INVOS® was 70 (SD 9)%; thereafter, it increased by 0.0187% min^{-1} ($P<0.01$). The cerebral tissue oxygen saturation measured with FORE-SIGHT® started at 68 (SD 13)% and increased by 0.0142% min^{-1} ($P<0.01$). The mean arterial blood pressure did not change. Cardiac output was between 6.3 (SD 1.3) and 7.2 (1.8) litre min^{-1} at the predefined time points. Cardiac output, but not mean arterial blood pressure, showed a positive and significant correlation with cerebral oxygen saturation.

Conclusions: During neurosurgery in the sitting position, the cerebral oxygen saturation slowly increases and, therefore, this position seems to be safe with regard to cerebral oxygen saturation. Cerebral oxygen saturation is stable because of constant CO and MAP, while the influence of CO on cerebral oxygen saturation seems to be more relevant.

Clinical trial registration: NCT01275898.

Key words: anaesthesia; cardiac output; heart; neurosurgery; position, sitting

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Editor's key points

- The sitting position has some advantages, and is still used, for some neurosurgical operations.
- This position during anaesthesia can have profound haemodynamic consequences, with potential cerebral hypoperfusion.
- The authors measured cerebral oxygenation and cardiac output in a cohort of sitting patients.
- Cerebral oxygenation increased from baseline during the procedure and was correlated with cardiac output.

For neurosurgical procedures in the dorsal cranium, the sitting position is often required, which necessitates a careful anaesthetic approach. The sitting position in anaesthetized patients might result in a deterioration of the cardiac output (CO) with a consecutive decrease in mean arterial blood pressure (MAP).[1–3] Changes in haemodynamics may influence cerebral blood flow and, thereby, cerebral oxygen saturation, which can be measured easily using near-infrared spectroscopy (NIRS).[4] This method is well established and can detect cerebral oxygen desaturation caused by cerebral hypoperfusion during carotid and cardiovascular operations.[5–7] As cerebral oxygen saturation seems to be independent of MAP,[5 8] it is possible that cerebral oxygen saturation might correlate with CO and, therefore, is flow dependent rather than pressure dependent. Cardiac output can be measured by transoesophageal echocardiography (TEE), which is used anyway during neurosurgical procedures in the sitting position to detect potential air embolism.[9]

The hypothesis tested in the present study is that the sitting position impairs cerebral oxygen saturation, measured with two different monitors, in anaesthetized patients undergoing neurosurgery in the dorsal cranium. We also investigated whether MAP, CO, or both determine cerebral oxygenation.

Methods

The study was approved by the local ethics committee of Rhineland-Palatinate, Germany [approval number: 837.009.10 (7018)], and written informed consent was obtained from all patients. The presented data are a subset from the previously registered study at clinicaltrials.gov with the registration number NCT01275898. All patients were recruited and anaesthetized at the Department of Anaesthesiology at the University Medical Centre Mainz in Germany. Patients of the cohort study were screened according to the operation schedule pending for neurosurgical treatment in the sitting position and were screened by one of the authors. Exclusion criteria were symptomatic cerebrovascular diseases, symptomatic coronary diseases, haemoglobin (Hb) concentration <10 mg dl^{-1}, and pregnancy.

In all patients, general anaesthesia was administered. Thirty minutes before induction of general anaesthesia, all patients received oral lorazepam 1 mg or midazolam 7.5 mg for premedication. The ECG, non-invasive blood pressure, and peripheral oxygen saturation (Sp_{O_2}) were monitored after arrival of the patient. General anaesthesia was induced using sufentanil 0.3–0.5 μg kg^{-1} i.v. followed by propofol 2 mg kg^{-1} i.v. and atracurium 0.5 mg kg^{-1} i.v. Anaesthesia was maintained with continuous infusion of propofol and remifentanil (Table 1), and repeated injections of atracurium as clinically indicated. The MAP was measured using an arterial cannula in a radial artery and maintained >60 mm Hg using continuous norepinephrine infusion (Table 1). Pressure-controlled mechanical ventilation

Table 1 Cardiac output, cerebral oxygen saturation, and physiological parameters at predefined time points. The time points represent the predefined measurements, as follows: (1) after induction of anaesthesia and attainment of a steady state in the supine position; (2) after infusion of 500 ml of hydroxyethyl starch 130/0.4/6%; (3) before positioning in the sitting position; (4) 5–10 min after reaching the sitting position; (5) before the start of surgery; (6) 5–10 min after the start of surgery; (7) at the end of the surgical procedure in the sitting position; and (8) 10 min after repositioning the patient in the supine position. CO, cardiac output; Hb, haemoglobin; HR, heart rate; MAP, mean arterial blood pressure; Pa_{CO_2}, arterial partial pressure of carbon dioxide; Pa_{O_2}, arterial partial pressure of oxygen; rS_{O_2}, regional cerebral oxygen saturation measured with INVOS®; St_{O_2}, cerebral tissue oxygen saturation measured with FORE-SIGHT®. Measurements are presented as the mean (SD), and for norepinephrine as median [IQR]

Parameter	Time points							
	1	2	3	4	5	6	7	8
CO (litre min^{-1})	6.7 (1.6)	7.0 (1.8)	7.2 (1.8)	6.3 (1.3)	6.8 (1.8)	6.6 (2.0)	7.0 (1.9)	7.0 (2.3)
MAP (mm Hg)	82 (14)	84 (12)	84 (12)	76 (10)	77 (10)	76 (10)	72 (12)	83 (12)
HR (beats min^{-1})	60 (9)	61 (9)	62 (8)	59 (9)	60 (10)	61 (10)	62 (11)	65 (14)
rS_{O_2} (%)	70 (9)	68 (11)	69 (10)	68 (10)	69 (10)	69 (10)	72 (11)	74 (10)
St_{O_2} (%)	68 (13)	68 (5)	69 (5)	68 (5)	69 (4)	69 (4)	71 (5)	77 (7)
Pa_{O_2} (kPa)	24.3 (8.4)	23.7 (7.0)	23.5 (6.4)	24.9 (6.7)	24.6 (5.8)	24.2 (5.8)	24.7 (5.2)	22.3 (6.7)
pH	7.42 (0.03)	7.42 (0.04)	7.42 (0.04)	7.41 (0.03)	7.40 (0.04)	7.40 (0.03)	7.39 (0.04)	7.39 (0.04)
Pa_{CO_2} (kPa)	5.2 (0.6)	5.4 (0.5)	5.2 (0.6)	5.2 (0.4)	5.3 (0.4)	5.2 (0.4)	5.0 (0.5)	4.9 (0.5)
Hb (g dl^{-1})	11.5 (1.4)	10.4 (1.3)	10.6 (1.4)	11.4 (1.4)	11.7 (1.3)	11.8 (1.3)	11.9 (1.3)	11.8 (1.6)
Propofol (mg kg^{-1} h^{-1})	4.6 (1.3)	5.0 (1.4)	5.4 (1.5)	5.8 (1.4)	6.4 (1.4)	6.7 (1.2)	6.5 (1.3)	6.4 (1.5)
Remifentanil (μg kg^{-1} min^{-1})	0.1 (0.0)	0.1 (0.1)	0.2 (0.1)	0.2 (0.1)	0.2 (0.1)	0.3 (0.1)	0.2 (0.1)	0.2 (0.1)
Norepinephrine (μg kg^{-1} min^{-1})	0.000 [0.000–0.000]	0.000 [0.000–0.003]	0.000 [0.000–0.019]	0.022 [0.016–0.038]	0.025 [0.011–0.043]	0.021 [0.011–0.043]	0.027 [0.012–0.055]	0.022 [0.009–0.036]