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Early postoperative changes of cerebrovenous oximetry after propofol/remifentanil anesthesia in patients after elective supratentorial craniotomy

The effect of different awakening strategies

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Summary

Backround and Objectives. The monitoring of cerebrovenous oxygen saturation ($S_{cv}O_2$) is used as a measure of the cerebral oxygenation of a patient during elective neurosurgical craniotomies. The aim of the study was to determine how the parameters of cerebral oxygenation are influenced directly by different awakening strategies. ***Methods.*** 30 elective neurosurgical patients were monitored using cerebrovenous oximetry from the time of anesthetic induction until 20 minutes after extubation. Arterial and cerebrovenous blood gas analyses were taken at six defined timepoints and compared with the hemodynamic and homeostatic parameters. Regarding the extubation time patients were divided into an early extubation group (EEX) (up to one hour following surgery) and a late extubation group (LEX) (> one hour following surgery). ***Results.*** Following induction the $S_{cv}O_2$ was lowest compared with the other measurement times, then rising over the further perioperative course. Depending on the extubation time, significantly lower cerebrovenous oxygen partial pressures were found five minutes after extubation in the patients of the early extubation group; just 20 minutes after extubation there was no longer any significant difference between the two groups. As to be expected, the arterial carbon dioxide tension had a significant influence on $S_{cv}O_2$ (R^2=0.608). ***Conclusion.*** When fulfilling the generally accepted extubation criteria (normothermia, normotonia, normocapnia and normoxia, sufficient vigilance), early extubation also presents no risk to patients during elective neurosurgery. The results of the given clinical study in patients following elective intracranial surgery show p_aCO_2 to be an underlying determinant of cerebral oxygenation. *Anestezjologia i Ratownictwo 2008; 2: 378-386.*

Keywords: neuroanaesthesia, cerebrovenous oximetry, awakening, remifentanil

Introduction

New surgical techniques which facilitate a minimally invasive neurosurgical approach and an image-guided removal of pathological tissue, as well as modern neurophysiological monitoring techniques, are today a standard in elective intracranial neurosurgery.

In parallel to such neurosurgical developments, substances with favorable pharmacokinetic and pharmacodynamic profiles have been approved in

recent years for use in general anesthesia. Supportive, non-medicinal intraoperative approaches have also improved considerably. Hence the use of modern thermosystems with forced-air warming generators and flexible body blankets, for example, facilitates targeted intraoperative, mild hypothermia as well as rapid and gentle postoperative normothermia [1].

As a result of such developments, the sole prevention and treatment of perioperative cerebral edema is no longer the priority in anesthesiological and postoperative intensive care. On the contrary, anesthesia is designed in such a way that intraoperatively adequate cerebral oxygenation is permanently guaranteed, potential neuroprotective anesthetic effects are exploited, regulation mechanisms of the cerebral circulation are not impaired and postoperative complications can be detected early on.

The limited evaluability of the central nervous functions during general anesthesia dictates, however, that appropriate monitoring be carried out. Aside from the standard, continuous hemodynamic monitoring , the anesthetist can obtain important metabolic information on the perfusion and oxygenation of the brain as the target organ, by monitoring cerebrovenous hemoglobin oxygen saturation in the cerebrovenous blood. Measurement of the cerebrovenous oxygen saturation ($S_{cv}O_2$) has become an accepted method for monitoring cerebral metabolism intraoperatively and above all in intensive care, e.g. after severe neurotrauma. The prognostic significance of such jugular venous desaturation events ($S_{cv}O_2$ <55%) in neurosurgical patients could be proven both intraoperatively and over the course of intensive care [2-5].

Within this discussion it seems to be taken almost for granted that early extubation following elective supratentorial neurosurgery is a standard in the perioperative management of neurosurgical patients. Himmelseher and Pfenninger were able to demonstrate, however, that postoperatively only approximately 61% of such patients are extubated immediately [6]. Multifactorial effects during the early postoperative phase (cerebral hyperemia, arterial hypertension, respiratory insufficiency) have so far resulted in an increased rate of complications and are responsible for restraint when deciding on a "fast-track concept" following elective supratentorial surgery [7].

Our study investigated the early perioperative changes in the cerebral oxygenation parameters of neurosurgical patients undergoing elective intracranial surgery and after remifentanil/propofol anesthesia, depending on the extubation time. Hemoglobin oxygen saturation ($S_{cv}O_2$), as well as the partial pressures of oxygen and carbon dioxide ($p_{cv}O_2$, $p_{cv}CO_2$) in the cerebrovenous blood, served as surrogate parameters for cerebral perfusion.

Patients and Methods

Once the study protocol had been approved by the local institutional review board, a total of 30 patients (>18 years) having undergone elective craniotomy were recruited to the prospective study.

Anesthesia

About one hour before surgery the patients were premedicated with oral midazolam (3.75 - 7.5 mg). Anesthesia was induced using etomidate 0.15 to 0.3 mg/kg, remifentanil (Ultiva®, GlaxoSmithKline, Munich, Germany) 0.5 µg/kg/min for three minutes and cisatracurium (Nimbex®, GlaxoSmithKline, Munich, Germany) 0.15 mg/kg. General anesthesia was maintained continually with intravenous propofol (Disoprivan 2%, AstraZeneca, Wedel, Germany) 4 to 8 mg/kg/h and remifentanil 0.1 to 0.3 µg/kg/min.

The patients underwent pressure-controlled ventilation during surgery with an inspiratory oxygen concentration (F_iO_2) of 0.4. Sympathomimetics were required in individual cases to maintain adequate perfusion.

Irrespective of further anesthesiological management, analgosedation was continued postoperatively with propofol (2-3 mg/kg/h) and remifentanil (0.1-0.15 µg/kg/min) until respirator withdrawal was initiated. The patients were given intravenous doses of piritramide (0.1 mg/kg) combined with paracetamol (Perfalgan®, Bristol-Myers Squibb GmbH & Co. KgaA; Munich, Germany) or metamizole (Novaminsulfon Ratiopharm, Ratiopharm GmbH, Ulm, Germany) as pre-emptive analgesia 20 minutes before the end of sedation, as well as continuous oxygen insufflation of 5 l/min via face mask following extubation, in order to guarantee peripheral oxygen saturation of > 96%.

Awekening management

At the end of surgery, the individual patients were assigned to groups in terms of the desired awakening management in agreement with the operating surgeon. Extubation and postoperative monitoring of

these patients ensued in the surgical recovery room, provided:

- the duration of surgery (incision-suture time) was < 4 hrs
- impaired consciousness was not to be expected primarily
- cardiopulmonary state was stable (FiO_2 0.4, MAP > 60 mmHg) without catecholamines
- body temperature was measured as > 36.0 °C
- intraoperative blood loss was less than 1.0 litres or hemoglobin at least 5.7 mmol/l

Patients who could not primarily be assigned to such management (duration of surgery > 4 hours, hypothermia, cardiopulmonary instability, impaired consciousness known or anticipated) were moved to intensive care for scheduled respirator withdrawal.

Monitoring

All patients recieved a central venous, multi-lumen catheter and arterial blood pressure monitor in one of the radial arteries. The patients were also equipped with a permanent bladder catheter with temperature probe for fluid regulation.

This standard monitoring included the following parameters: Electrocardiogram (ECG), peripheral oxygen saturation (S_pO_2), arterial blood pressure (ABP), central venous pressure (CVP), end-expiratory carbon dioxide concentration ($etCO_2$), inspiratory F_iO_2 and urinary bladder temperature as a core body temperature (Tc) equivalent.

Retrograde percutaneous cannulation ensued for insertion of the access portal (5F introducer) for cerebrovenous oximetry in the internal jugular vein, at the level of the lateral cricoid of the palpated carotid artery. The side of the neck to be punctated was selected according to the localization of the predominant lesion which would undergo surgery. The right internal jugular vein was always punctured if the lesion was not clearly located on one side [8]. Once the vein had been punctured successfully, a 5 F introducer was first inserted in a retrograde direction using the Seldinger technique, ending approx. 2.5 cm below the cranial base. This served as the inlet for the Oximetrix catheter (U440, Abbott); the correct positioning of the access portal - in the jugular venous bulb - was controlled by frontal or lateral X-ray.

Postoperatively the access portal remained in situ in all patients and was removed prior to transferral to the neurosurgical ward, or on the third postoperative day in the intensive care unit at the latest.

Bloodgas analyses

To compare the intraoperative and postoperative blood gas analyses with the preoperative baseline values, the first blood gas analysis was done immediately after inserting the introducer for cerebrovenous oximetry (IN). Intraoperatively, cerebrovenous blood was taken as a standard after opening of the dura (IO). The first postoperative blood gas analysis ensued immediately after ending surgery (PO), immediately before extubation (BE) and five minutes (PE5) and 20 minutes post-extubation (PE20).

In parallel to all the recorded cerebrovenous blood gas analyses from the jugular venous bulb, arterial blood samples were taken intraoperatively (ABL 510, Radiometer Copenhagen, Germany) and postoperatively (I-STAT Corporation, East Windsor USA) for blood gas analysis. Measurements were taken for arterial oxygen partial pressure (p_aO_2), arterial carbon dioxide partial pressure (p_aCO_2), pH (pH_a) and arterial hemoglobin oxygen saturation (Sat_a).

Statistics

The data obtained from the blood gas analyses and the spatially distributed vital parameters were documented in a table using table calculation software (MS Excel 5.0, Microsoft, USA). Statistical software was used for the analysis (StatView; Abacus Concepts, Berkeley, CA, USA).

In addition to the standard deviation, the arithmetic means were calculated and provided for the statistical analysis and presentation of the mean arterial blood pressure (MAP), p_aO_2, p_aCO_2, cerebrovenous oxygen partial pressure ($p_{cv}O_2$), cerebrovenous carbon dioxide partial pressure ($p_{cv}CO_2$), cerebrovenous hemoglobin oxygen saturation ($S_{cv}O_2$) at the individual observation times. In order to compare the group data for early and late extubation, an analysis was performed using purely descriptive statistical methods as well as an ANOVA group analysis with a view to statistically relevant differences in the mean values.

The statistical dependency of two parameters was checked using linear regression (R^2 and adjusted r^2). The statistical significance was defined at $p<0.05$.

Results

The data of all 30 patients were used in the analysis. The mean age of the 14 male and 16 female patients was 55 ± 14 years. 22 patients presented with glioblastoma,

Table 1. Mean values for ± SD of the mean arterial pressure (MAP), arterial oxygen (p_aO_2) and arterial carbon dioxide partial pressure (p_aCO_2) as well as the core body temperature (Tc) over time for all 32 patients; induction [IN], intraoperative [IO], postoperative [PO], before extubation [BE], 5 min post-extubation [PE5] and 20 min post-extubation [PE20]

	IN		IO		PO		BE		PE_5		PE_{20}	
Parameter	EEX	LEX	EEX	LEX	EEX	LEX	EEX	LEX	EEX	LEX	EEX	LEX
MAP [mmHg]	80 ±16	76 ±16 *, +, ●	74 ±12 +	76 ±10 ●●	82 ±20	84 ±14 *, +, ●	96 ±21	97 ±17	100 ±15	96 ±14	99 ±12	96 ±15
P_aO_2 [mmHg]	236 ±130 *, +	203 ±80	220 ±67 *, +	196 ±73 ***, ++	228 ±94 *, +	197 ±68	202 ±85 *, +	178 ±84	132 ±40	186 ±78 †	132 ±34 †	189 ±74
P_aCO_2 [mmHg]	36 ±4.4	34 ±2.7 ***, +	38 ±2.8	36 ±3.2 *	37 ±2.1	35 ±4.1	39 ±6.4	36 ±4.5	39 ±5.0	41 ±6.9 ●	37 ±4.1	38 ±6.4
T_c [°C]	36.5 ±0.6 *	36.4 ±0.8 *, +, ●	36.0 ±0.5 +	36.0 ±0.6 **, ++, ●●	36.2 ±0.7	36.4 ±0.9 *, +, ●	36.4 ±0.7	37.0 ±0.5	36.4 ±0.6	37.1 ±0.5	36.8 ±0.7	37.3 ±0.5

*p<0.05 vs. PE5; ** p<0.001 vs. PE5 ; *** p<0.0001 vs.. PE5
+ p<0.05 vs. PE20; ++ p<0,0001 vs. PE20
● p<0,05 vs. BE; ●● p<0,0005 vs. BE
† p<0.05 vs. EEX;

six with intracerebral meningeoma and two with intracerebral metastasis.

There were no reports of inaccurate arterial punctures, thromboses or bleeding from the puncture site. The most important physiological parameters at each of the measuring timepoints are presented in Table 1.

Intraoperative cerebrovenous oximetry

The mean arterial blood pressure was elevated in both groups at the onset of general anesthesia as compared with the intraoperative values. There was no statistical significance between the two study groups at each of the measuring timepoints (Table 1).

Cerebrovenous hemoglobin oxygen saturation $S_{cv}O_2$

The arterial oxygen saturation remained constant over the entire study period in all patients, at between 98-100%.

The mean values for cerebrovenous oxygen saturation at each of the measurement times are presented in Figure 1. The $S_{cv}O_2$ was lowest at the time of anesthetic induction (IN) as compared with all other measurement times, with a statistical significance being found only in the LEX group upon intraoperative measurement (IN: 59 ± 9% vs. 67 ± 8%, p<0.05; Figure 2). There was no direct statistical dependency on the mean arterial pressure or arterial pCO_2.

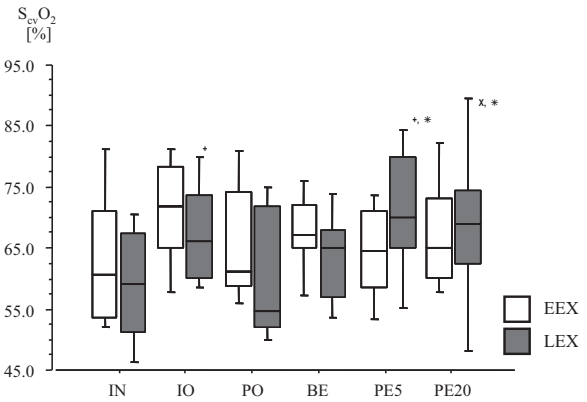

Figure 1. Mean values for cerebrovenous hemoglobin oxygen saturation ($ScvO_2$) for each patient population (EEX: early extubation group; LEX: late extubation group) at the timepoints induction [IN], intraoperative [IO], postoperative [PO], before extubation [BE], 5 min post-extubation [PE5] and 20 min post-extubation [PE20]
+ p<0.05 vs. IN, ✳p<0.05 vs. PO

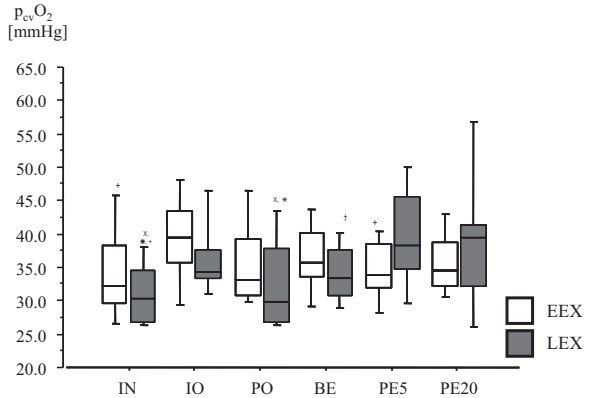

Figure 2. Mean values for cerebrovenous oxygen partial pressure ($p_{cv}O_2$) at the respective measurement timepoints; induction [IN], intraoperative [IO], postoperative [PO], before extubation [BE], 5 min post-extubation [PE5] and 20 min post-extubation [PE20]
+ p<0.05 vs. IO, x p<0.05 vs. PE20;
* p < 0.05 vs. PE5; † p<0.05 vs. PE5;
‡ p<0.05 EEX vs. LEX

Oxygen partial pressure (pO_2)

The correlation between arterial and cerebrovenous oxygen partial pressure revealed no statistical significance (R^2 = 0.007). The oxygenation in the arterial blood was in fact clearly reduced, however, in comparison with the other measurement times following extubation (p<0.05, Table 1).

The lowest $p_{cv}O_2$ values were found during induction of anesthesia, immediately after insertion of the cerebrovenous catheter. The highest cerebrovenous oxygen partial pressures were found intraoperatively (p<0.05) in the EEX group. There was a renewed increase in cerebrovenous oxygen tension following extubation in patients who were extubated in the intensive care unit (LEX group, Figure 2). There was a statistical significance in the $p_{cv}O_2$ between the two groups only five minutes after extubation (EEX: 34.4 ± 4.7 mmHg, LEX: 40.3 ± 8.5 mmHg, p<0.05). At no point during the investigations was there a significant correlation between the changes in systemic blood pressure and a change in the cerebrovenous oxygen partial pressure (R^2 = 0.008).

Carbon dioxide partial pressure (pCO_2)

The mean values of arterial pCO_2 at the different measurement points are presented in Table 1. The relationship of the cerebrovenous carbon dioxide pressure $p_{cv}CO_2$ to arterial p_aCO_2 can be deduced from Figure 3 (R^2 = 0.608). Analysis of the statistical correlation between cerebrovenous oxygen partial pressure $p_{cv}O_2$ and arterial carbon dioxide tension p_aCO_2 showed only a tendential dependency (R^2 = 0.273).

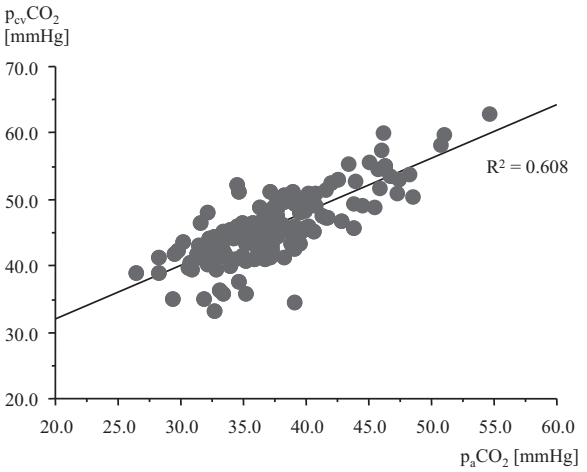

Figure 3. Relationship between arterial (p_aCO_2) and cerebrovenous ($p_{cv}CO_2$) carbon dioxide partial pressure during the entire study period

The arterio-cerebrovenous CO_2 difference showed a maximum immediately after anesthetic induction in both groups (EEX: -9.4 ± 4.0 mmHg; LEX: -10.6 ± 2.6 mmHg, n.s.) and a minimum at timepoint PE5 (EEX: -7.8 ± 2.5 mmHg; LEX: 7.0 ± 3.2 mmHg, n.s.). There was no direct statistical correlation between the arterio-cerebrovenous CO_2 difference and the mean arterial pressure (R^2=0.018).

Influence of extubation time

At the end of surgery 15 patients were assigned to an early extubation group (EEX) and 14 patients to the late extubation group (LEX). One patient could not be included in the analysis due to a lack of cerebrovenous blood gas analysis after extubation.

The mean duration of surgery was 195 ± 40 min in the patients of the EEX group and 345 ± 105 min in the late extubation group (p<0.001).

The extubation in the EEX group was performed up to one hour after the end of surgery (31 ± 18 min). In 13 patients of the LEX group, the awakening time

was 249 ± 126 min (p<0.0001).

The mean arterial blood pressure following extubation was in fact significantly increased compared to the intraoperative course in both groups, but the differences between the two goups at each of the measurement times was not statistically significant (Table 1).

Three patients presented with cerebrovenous oxygen saturation > 80% postoperatively, and in three patients $S_{cv}O_2$ values < 55% were found following extubation (EEX=2; LEX=1). The cause for desaturation below 55% was found to be an epidural hematoma, the onset of cerebral edema and a stress reaction due to persistent pain, respectively.

Postoperative shivering also occurred in three patients (EEX=1; LEX=2). Aside from elevated blood pressure and heart rate, no pathological changes in cerebral oxygenation could be found in these patients.

There were significant differences in the oxygenation of the arterial blood depending on the extubation time (PE5: EEX 131.8 ± 38.8 mmHg vs. LEX 185.8 ± 77.9 mmHg, p<0.05; PE20: EEX 131.5 ± 34.2 mmHg vs. LEX 188.8 ± 74.2 mmHg, p<0.05)

The mean cerebrovenous oxygen partial pressure five minutes post-extubation was significantly lower in the patients of the early extubation group (EEX 34.4 ± 4.7 mmHg vs. LEX 40.3 ± 8.5 mmHg, p<0.05). There was no further significant difference between both groups 20 minutes after extubation (EEX 35.8 ± 4.5 mmHg vs. LEX 39.2 ± 10.7 mmHg, n.s., Figure 2).

The difference in mean cerebrovenous oxygen saturation $S_{cv}O_2$ was only significant in either group at the two measurement times after extubation compared to the direct postoperative measurement (PO). However, there was a tendency after extubation for $S_{cv}O_2$ to increase after 20 minutes.

There was a rise in arterial carbon dioxide tension in all patients post-extubation. After an initial mean p_aCO_2 of 39.0 ± 5.0 mmHg in the EEX group and 41.0 ± 7.0 mmHg in the LEX group at timepoint NE5, the value dropped after 20 minutes to 36.6 ± 5.0 mmHg (EEX) and 41.3 ± 6.9 mmHg (LEX) (n.s.). The p_aCO_2 value at timepoint NE20 tended to be lower in the patients on late extubation than in those on early extubation (36.8 ± 4.5 mmHg vs. 39.0 ± 5.3 mmHg) (n.s).

The cerebrovenous carbon dioxide partial pressures were almost identical over the course and revealed no statistical significance between the measuring timepoints or in terms of the extubation time (Figure 4). Following extubation, the arteriocerebrovenous CO_2 difference was 7.8 ± 2.5 mmHg (EEX) and 7.0 ± 3.3 mmHg (LEX) after five minutes and 8.6 ± 3.4 mmHg (EEX) and 8.4 ± 3.2 mmHg (LEX) after 20 minutes.

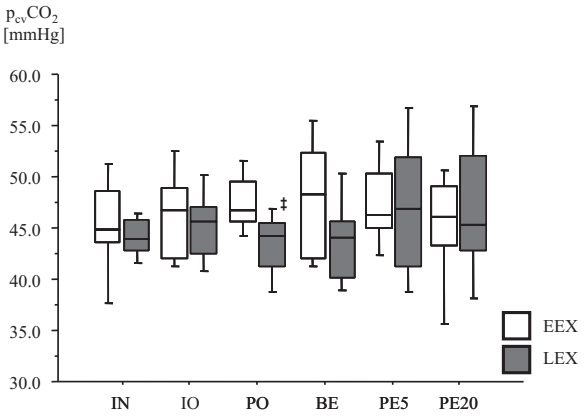

Figure 4. Mean values for the cerebrovenous carbon dioxide partial pressure ($p_{cv}CO_2$) at the respective measurement timepoints; induction [IN], intraoperative [IO], postoperative [PO], before extubation [BE], 5 min post-extubation [PE5] and 20 min post-extubation [PE20]
+ p<0.05 vs. IO, x p<0.05 vs. PE20;
* p < 0.05 vs. PE5; † p<0.05 vs. PE5;
‡ p<0.05 EEX vs. LEX

Discussion

The concept of early extubation after a complex surgical intervention - known as fast-track anesthesia - serves to improve the perioperative treatment process. To patients having undergone elective neurosurgery in particular it signifies on the one hand the possibility of rapid neurological assessment, but bears on the other hand the risk of cardiovascular or respiratory complications during the early period [7,9].

The use of cerebrovenous oximetry provides the anesthetist with the opportunity to monitor the ratio of oxygen supply to demand during neurosurgery, draw conclusions indirectly from the cerebral perfusion and, where applicable, decide on the postoperative approach. The normal value range could be determined in numerous clinical studies. If $S_{cv}O_2$ is lower than 54-50%, the global ratio between the cerebral oxygen supply and oxygen demand will be pathological [3,4].

Various mechanisms which have an influence on hemodynamics and cerebral autoregulation, and thus on cerebral perfusion, play their part during induction and at the end of anesthesia.

Our findings show a clear rise in $S_{cv}O_2$ initially following induction, which over the further perioperative course decreases significantly in terms of cerebrovenous oxygenation, but nevertheless remains above the critical limit of 60%. The mean arterial pressure in all patients was > 70 mmHg, thus the arterial carbon dioxide partial pressure primarily assumes the causal role and dictates cerebral oxygen fluctuations.

The substances remifentanil and propofol, which have been proven as safe in several studies of intracranial interventions in puncto for sustained autoregulation, were used in this study [10,11]. The μ-receptor agonist remifentanil demonstrated sustained CO_2 reactivity of the cerebral vessels. Using an induction dose of 5 μg/kg/min remifentanil under isocapnia and constant hemodynamic conditions, Paris et al. recorded a drop in the cerebral blood flow rate by 31%, for example, which could indicate a central hemodynamic effect. Induction with 2 and 3 μg/kg/min remifentanil, on the other hand, had no significant effect on cerebral blood flow [11]. Our patients were given an induction dose of 1.5 μg/kg/min remifentanil and were not at risk of a drop in CBF.

As a result of the proven reduction in CBF and cerebral metabolism, the lipid-soluble hypnotic agent propofol is ascribed a neuroprotective effect with a simultaneously sustained physiological reaction of the CBF to CO_2 and blood pressure [12,13]. However, Jansen et al. discovered in a study of cerebral tumor patients that during anesthesia under propofol combined with the opioid fentanyl, half the patients on normoventilation had signs of cerebral underperfusion because there was a drop in the recorded $S_{cv}O_2$ to below 50% and the $p_{cv}O_2$ to a mean of 27 mmHg [14]. In contrast, our study produced the highest mean cerebrovenous oxygen partial pressure of 38 mmHg intraoperatively at a mean p_aCO_2 of 40 mmHg.

Postoperative Period – "Fast track" or late extubation?

The reason for conducting this study was, among others, the assumption that the cerebral oxygen supply is endangered by a general postoperative stress reaction, with increased oxygen consumption and reduced supply. The generally applicable extubation criteria demand adequate spontaneous respiration, but a lower arterial oxygenation level is to be expected post-extubation. A current study by Magni et al. supports our hypothesis and analyses the occurrence of early postoperative complications within the first six hours following intracranial surgery. Complications occurred in 57% of the 162 analysed patients, 28% of which were of a respiratory nature (hypercarbia with p_aCO_2 > 45 mmHg, hypoxia with p_aO_2 < 90 mmHg). The given causes are intraoperative atelectasis, hypoventilation from a residual narcotic effect and reduced vigilance as a result of the underlying disease [15].

The respiratory depression normally caused by opiates could be prevented in the study patients by the use of remifentanil, since the substance loses its efficacy after about five minutes.

Shivering, which often occurs postoperatively at a rate of between 18-30%, presents a further problem. Shivering leads to an increased cardiac and systemic need for energy, oxygen consumption and increased carbon dioxide production. Inadequate respiration and cardiac function will result in hypoxemia, lactate acidosis and certainly an increase in intracranial pressure. A study by Bilotta et al. showed the oxygen consumption to be almost doubled as opposed to neurosurgical patients with shivering treated with prophylactic medication [16]. Postoperative shivering occurred in three of our patients. Apart from elevated blood pressure and heart rate, no effect on cerebral oxygenation with normal p_aCO_2, p_aO_2 and $p_{cv}O_2$ could be proven.

The changes to which cerebral circulation is subjected during the waking period following neurosurgery was investigated by Bruder et al. using transcranial Doppler. Cerebral blood flow thereby underwent a 60% increase during extubation. This significant increase could be observed until at least 30 minutes after extubation. During the entire period there was no correlation between the rate of cerebral blood flow and MAP, and the p_aCO_2, respectively. Cerebrovenous oxygen saturation was also found to increase significantly until extubation. This is consistent with the reported cerebral hyperemia, regardless of the anesthetic technique, hemodynamics and ventilation. Cerebral hyperemia is considered to be a non-specific response to the stress of waking, and could be a factor for cerebral complications during the early postoperative period [17].

A further clinical study by Bruder et al. used perioperative measurements of the oxygen consumption and

plasma catecholamine concentration to investigate the hypothesis of metabolic fluctuations following intracranial interventions being reduced by late extubation. Thereby, significantly higher oxygen consumption rates and noradrenaline levels were found during extubation in the late extubation group following two-hour propofol sedation. In the patients woken immediately post-op, the mean oxygen consumption was significantly increased during the entire postoperative monitoring period until 30 minutes post-extubation. There was no difference between the groups in terms of the hemodynamic parameters MAP and HR, nor the plasma adrenaline concentration. As a consequence, early extubation following neurosurgical interventions can be advocated with a view to stress reduction [18].

The positive results from the investigations in patients in whom a combination of the short-acting and easily controllable medications propofol and remifentanil was used as part of general anesthesia over several hours of intracranial surgery, resulted ultimately in a change to the existing concept [19,20]. Thees et al. report, for example, that 74% of the analysed 450 patients could be extubated without complication within 12 minutes of ending the remifentanil infusion and were oriented after 15 minutes, respectively [21].

The results obtained for $S_{cv}O_2$ in our study reached a maximum shortly after extubation, with lower values returning in most cases 20 minutes later. There are in fact significant differences depending on the extubation time, although they are of no clinical relevance (Figure 1). They are suggestive of the same mechanism, since the elevated postoperative oxygen consumption at least would have suggested a decrease. However, no significant increase to the intraoperative period could be proven. Changes in the arterial pCO_2 may possibly play a part here.

Limitations

Three patients in our study had postoperative cerebrovenous oxygen saturation levels > 80% and thus were in a clear hyperemic cerebral state. Admittedly, due to a lack of neurological clinical findings and controlled catheter position the extent to which the wrong positioning of a catheter was decisive remains to be discussed. Roughly 95% of the mixed venous blood in the upper third of the bulb is purely cerebrovenous, the remaining 5% is extracranial with venous outflow regions, for example the skin and musculature from the eye, temporal and mastoid area [20]. It is certainly conceivable that extracranial blood is mixed into the aspiration sample, triggered by the spontaneous movements of the alert patient. Furthermore, the potential influence of our unilateral monitoring of the cerebrovenous blood must be considered when interpreting the results. Studies have been able to demonstrate that the validity of the method is enhanced by bilateral monitoring with inclusion of the individual variability of the cerebrovenous outflow [8,22].

Conclusion

The findings of the given clinical study in patients having undergone elective intracranial surgery show p_aCO_2 to be an underlying determinant of cerebral oxygenation. In the case of the immediate postoperative period, which carries a greater risk, the question of the correct extubation time when using short-acting and easily controllable anesthetics is not important. Patients may benefit rather from prompt neurological monitoring without any major interference from the anesthetics. The determination of cerebrovenous oxygen saturation is to be regarded as a helpful means of monitoring in the detection of periods of critical perfusion, as well as an additional indication of potential intracranial complications.

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