Awakening management after neurosurgery for intracranial tumours

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Purpose of review

Major complications after intracranial surgery occur in 13–27% of patients. These complications may have multiple causes, but a body of arguments suggests that the haemodynamic and metabolic changes of anaesthesia recovery may be responsible for intracranial complications. The aim of this review is to explain the rationale of this hypothesis and analyse the recent studies relevant to neuroanaesthesia recovery.

Recent findings

Rapid recovery and extubation after intracranial tumour surgery is desirable in order to make an early diagnosis of intracranial complications. Since light pharmacological sedation may worsen a neurological deficit, short-acting anaesthetics are preferable intraoperatively. Extubation in the operating room, however, may be associated with agitation, increased oxygen consumption, catecholamine secretion, hypercapnia and systemic hypertension. This may exacerbate cerebral hyperaemia observed even during an uneventful recovery, leading to cerebral oedema or haemorrhage.

Summary

Pain, hypothermia, hypercapnia, hypoxia, hypoosmolality, hypertension, and anaemia should be avoided during emergence. Early emergence is associated with minimal haemodynamic and metabolic changes. If there is any doubt as to whether the patient should be extubated in the operating room, a gradual emergence in the intensive care unit makes it possible to decide whether or not extubation can be performed safely.

Keywords

neurosurgery, recovery period, cerebral hyperemia, extubation, oxygen consumption, cerebral blood flow

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Abbreviations

CBF	cerebral blood flow
ICP	intracranial pressure
$CMRO_2$	cerebral metabolic rate in oxygen

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Introduction

Postoperative complications after intracranial tumour surgery may have devastating effects even after an uneventful neurosurgical procedure. Anaesthesiologists and neurosurgeons suppose that physiological changes during anaesthesia recovery may cause intracranial bleeding or cerebral oedema. Despite the lack of evidence, our efforts as anaesthesiologists should be to minimize the occurrence of coughing, hypertension, hypoxia and hypercapnia during emergence, which may cause intracranial complications. These complications are common. In a prospective study of 486 patients, 54.5% of the patients who could be extubated during the 4 h following surgery had at least one complication. The most common complication was nausea or vomiting (38%), but respiratory problems occurred in 2.8%, cardiovascular complications in 6.7% and neurological complications in 5.7% of patients [1]. In other retrospective studies, the overall major complication rate was between 13 and 27.5% [2–4]. Fast-tracking may be associated with a more frequent incidence of these events than a slower emergence. This is probably the reason why postoperative ventilation is common in some neurosurgical centres after intracranial surgery. In a survey on ventilation practices in neuroanaesthesia in Germany, only 61% of patients with brain tumours were managed without postoperative ventilation [5]. There was a trend toward early extubation from 1991 to 1997, however. Due to the potential risks associated with early recovery and extubation, we need to understand the physiopathology of anaesthesia recovery and to evaluate the risk versus benefit ratio of early extubation.

Rationale for a rapid emergence

The early diagnosis of a postoperative cerebral complication is essential in order to limit the consequences and improve outcome. It has been demonstrated that low doses of midazolam (2.8 ± 1.3 mg) or fentanyl (170 ± 60 μg) can exacerbate or unmask focal neurological deficits in more than 60% of patients with prior compensated neurological dysfunction. Similarly, under light sedation with midazolam, patients whose initial stroke syndrome had improved clinically showed transient reemergence of their initial focal syndrome [6•]. This finding probably explains some reports of dramatic reversal of postoperative neurological deficits with naloxone [7] and the common observation of transient neurological deficits during the recovery period following neurosurgery. This is a good reason to use short-acting agents

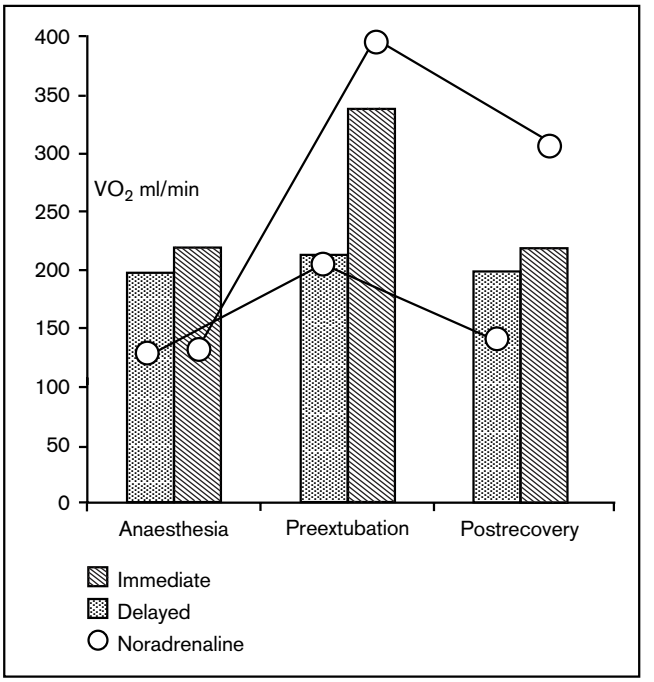
during neurosurgical anaesthesia to avoid unnecessary head computed tomography scans or emergency transfer from the intensive care unit to the operating room. It has been demonstrated that times to follow verbal commands and tracheal extubation were more rapid and more predictable with remifentanil than with fentanyl [8,9]. In patients undergoing spinal fusion under propofol anaesthesia, the time until recovery of orientation increased from 13.2 ± 4.7 min without fentanyl to 54.3 ± 14.4 min when the fentanyl concentration was maintained at 4.5 ng ml^{-1} during surgery [10]. After moderate doses of fentanyl (30 $\mu g\ kg^{-1}$), naloxone is frequently needed to allow tracheal extubation [9]. Factors other than anaesthetics, however, may explain a delayed recovery. The nature, the location (frontal or not) and the size of the intracranial lesion play a role [11]. Mild hypothermia, which may be used for intraoperative neuroprotection, also delays recovery and extubation.

Mechanism of cerebral complications after intracranial surgery

Rapid emergence from anaesthesia may be associated with systemic hypertension. Hypertension during emergence is frequent in neurosurgical patients and has been reported in 70–90% of patients [12,13]. This is the consequence of sympathetic stimulation demonstrated by an increase in circulating catecholamine and oxygen consumption [14] (Fig. 1). It is clear that severe hypertension (systemic blood pressure >200 mmHg) is a risk factor for intracranial haemorrhage in patients recovering from intracranial surgery. The risk associated with less severe hypertension, however, is not demonstrated. Basali *et al.* [15] established a link between perioperative hypertension and intracranial haemorrhage after craniotomy in a retrospective case control study. Patients with postoperative intracranial haemorrhage were 3.6 times more likely to be hypertensive than their matched controls. Of particular interest was the very strong association with intracranial haemorrhage when blood pressure remained in the normal range intraoperatively but became hypertensive postoperatively. This suggested that loose surgical haemostasis performed at a low blood pressure may bleed at a higher blood pressure.

Intracranial hypertension is common after neurosurgery. In a retrospective study of 514 patients whose intracranial pressure (ICP) was monitored after elective intracranial surgery, 76 (18.4%) of the 412 patients operated on in the supratentorial region had a postoperative sustained ICP elevation exceeding 20 mmHg [16]. Abnormally high ICP occurred after 13 (12.7%) of the 102 infratentorial operations. Of the 89 patients with elevated ICP, 47 (52.8%) had an associated clinical deterioration. The most common findings on computed tomography scans were cerebral oedema and cerebral

Figure 1. Changes in oxygen consumption (VO_2) (ml/min) and noradrenaline blood concentration (pg/ml) during early (in the operating room) or delayed (2 h) recovery in neurosurgical patients



Adapted from [14].

haemorrhage. It has also been well documented that tracheal stimulation increases ICP, although cerebral perfusion pressure is maintained in most cases [17,18•]. This ICP increase is related to arousal, coughing and transient cerebral hyperaemia [17]. Its duration is variable, depending on brain compliance, but is usually less than 5 min. On extubation, the tracheal stimulation is often associated with hypercapnia due to increased carbon dioxide production and respiratory depression. Large increases in ICP may thus be anticipated in patients with a 'tight brain' at the end of surgery. Laryngotracheal lidocaine is effective for limiting coughing due to tracheal stimulation [19•]. Short-acting opioids (remifentanil and alfentanil) decrease coughing, agitation and cardiovascular stimulation during emergence from anaesthesia [20,21], showing the importance of postoperative analgesia after craniotomy. These agents, however, may interfere with clinical assessment.

Another reason for the occurrence of cerebral complications is postoperative cerebral hyperaemia. Tracheal extubation is associated with a 60–80% increase in cerebral blood flow (CBF) velocity from preinduction baseline value, with an increase in the jugular venous bulb saturation in oxygen [22•] (Fig. 2). This may occur after tumour or aneurysm surgery and does not depend

on the anaesthetic technique used (propofol or isoflurane). This complication has also been demonstrated after surgery for chronic subdural haematoma [23–25] or carotid surgery or stenting [26••]. In elderly patients, cerebral hyperaemia was significantly associated with postoperative delirium and exacerbated by postoperative systemic hypertension [23]. The significance of postoperative hyperaemia after intracranial tumour surgery is unknown, however [27]. A provocative study suggested that deep opioid analgesia with a 1½–2 h delayed emergence after completion of surgery reduced the incidence of postoperative intracranial haemorrhage [28]. This result was explained by better postoperative haemodynamic control. The study, however, was retrospective and did not include a control group. The same results could probably be obtained with an early recovery including tight haemodynamic and metabolic control. In another study using lower dose narcotics, a 2 h delayed recovery was associated with larger metabolic and cardiovascular changes than an early recovery [14].

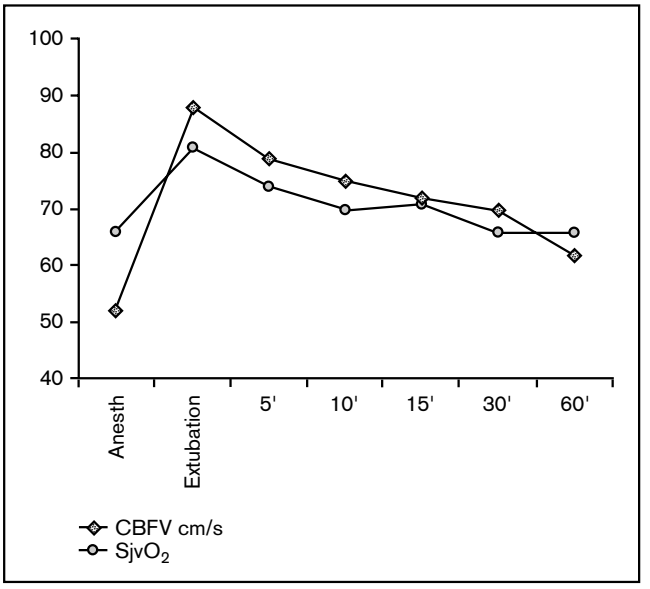
Neurological monitoring during emergence

Although clinical neurological assessment is highly specific and reliable monitoring of brain function, a neurological deficit is often a sign of a severe intracranial complication. More sensitive methods would be useful to detect earlier more subtle cerebral circulatory or pressure changes. ICP is seldom monitored in the period after surgery despite the high frequency of postoperative intracranial complications. CBF monitoring would be useful to detect cerebral hyperaemia. Transcranial Doppler is widely used for this purpose, however this technique measures velocity, not flow. The development of noninvasive and nonradioactive methods for the continuous monitoring of CBF might improve our understanding of the cerebral physiology during recovery from anaesthesia [29,30]. The validity of these methods during periods of rapid changes in CBF and $CMRO_2$, however, is not demonstrated [31,32•].

How to manage early recovery after intracranial surgery

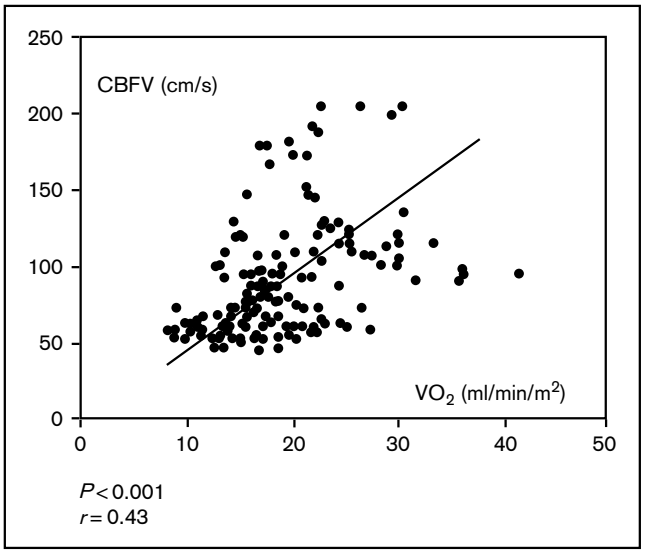
Several conditions are required for a smooth and uncomplicated emergence from neurosurgery [33]. First, hypothermia should be avoided because it promotes shivering, increases oxygen consumption and cardiac output or blood pressure. The best way to limit postoperative shivering is to maintain normothermia, which is not difficult using forced air warming during neurosurgery. In most normothermic patients, oxygen consumption and blood catecholamine increases are very small during recovery immediately after surgery [14]. In hypothermic patients, pharmacological methods can prevent postoperative shivering [34,35•]. The relationship between cardiac output or blood pressure and CBF during emergence is not clear, but there is a link between the intensity of the sympathetic discharge and cerebral hyperaemia. We found a statistical relationship between the increase in oxygen consumption during recovery and CBF velocity at extubation (Fig. 3).

Figure 2. Changes in cerebral blood flow velocity (CBFV) and jugular venous bulb saturation in oxygen ($SjvO_2$) during recovery from neurosurgical anaesthesia disclosing cerebral hyperaemia



Adapted from [22•].

Figure 3. Relationship between body oxygen consumption (VO_2) and cerebral blood flow velocity (CBFV) during recovery from neurosurgical anaesthesia using propofol as the main anaesthetic



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Postoperative analgesia

Pain should also be prevented. In a prospective randomized study comparing remifentanil and fentanyl, the remifentanil group was more frequently hypertensive postoperatively, which was probably due to the lack of postoperative analgesia [9]. Craniotomy is less painful than other surgical procedures such as facial reconstruction or lumbar laminectomy [36]. The increasing use of low-dose narcotic techniques or remifentanil, however, has renewed the interest in postoperative pain management after craniotomy. Paracetamol alone does not provide pain relief and must be associated with tramadol or nalbuphine [37••]. Tramadol is efficient, does not affect ICP or cerebral perfusion pressure [38], but there is some concern that this agent increases the frequency of nausea and vomiting and induces somnolence [39]. Morphine is effective with few side effects, but postoperative somnolence may limit its indications [40]. Scalp infiltration with bupivacaine decreases postoperative pain but its effect is limited to the first few hours after surgery [41].

Nausea and vomiting

The frequency of nausea is 50% after craniotomy and vomiting occurs in approximately 40% of patients [1,42,43]. Emesis is more frequent after infratentorial surgery and in women rather than in men [44]. A prophylaxis against postoperative nausea and vomiting is often indicated. Ondansetron is safe [45] and has few side effects but is only partially effective [42,43,46,47•]. Droperidol is more effective than ondansetron for preventing vomiting and does not induce sedation if the dose is less than 1 mg [43]. Since repeated infusions of droperidol may induce sedation, the combination of droperidol (less than 1 mg) and ondansetron is indicated. Metoclopramide is not very effective for preventing postoperative nausea and vomiting and was reported to increase ICP in one head-injured patient [48].

Haemodynamic control

Systemic hypertension is common during emergence and a prophylactic infusion of esmolol may be considered. Doses as high as 200–300 $\mu g\ kg^{-1}\ min^{-1}$ are usually needed to control haemodynamic changes [12]. Beta-blockers may blunt cerebral hyperaemia during emergence. In every case, an intravenous antihypertensive agent should be available immediately (labetalol, urapidil, nicardipine). In a study comparing labetalol and nicardipine for the control of emergence hypertension after craniotomy, the nicardipine-treated group experienced a higher incidence of hypotension and tachycardia than the labetalol group [49]. The association of diltiazem and nicardipine is effective, with the advantage of inducing less tachycardia than nicardipine alone.

Close attention should be paid to the vacuum system connected to the extradural drainage system. First, uncontrolled haemorrhage may occasionally occur leading to severe hypotension if not discovered in time. Second, the negative pressure of the drains may induce intracranial hypotension and lead to intracranial haemorrhage remote to the operating site [50,51•]. This is most probably a common and often overlooked cause of cerebral haemorrhage after craniotomy. Transient bradycardia after connecting the vacuum device is a warning sign of this complication.

Delayed recovery after intracranial surgery

Rapid recovery may fail or may not be a good choice. This is the case after lengthy surgery (>6 h), large tumour resection, injury to the cranial nerves (especially IX, X and XII), complications during surgery, hypothermia, and severe respiratory or cardiovascular complications during emergence [33] (Table 1). In a prospective study, 11% of patients remained intubated for more than 4 h after the end of surgery [1]. In most situations, an assessment of the neurological condition of the patient is possible under close haemodynamic and respiratory monitoring. Then the patient is sedated until emergence and extubation are indicated. This allows the diagnosis of swallowing disorders, the correction of coagulopathy or anaemia, adequate ventilation, return to normothermia, and control of pain. Since sedation impairs neurological evaluation, an early computed tomography scan before transfer to the intensive care unit may be indicated, especially after difficult procedures. After stopping sedation in the intensive care unit, the patient should be rapidly awake and alert. Propofol is probably the best choice to perform a reliable clinical assessment a few minutes after stopping the infusion.

Conclusion

The advent of new short-acting anaesthetic agents has made perioperative anaesthetic management in neurosurgery easier and extubation delays more predictable. These new techniques, however, have shifted some

Table 1. Systemic and cerebral conditions making delayed emergence considered to be a good choice

Systemic	Cerebral
Hypothermia ($T<35.5^{\circ}C$)	Preoperative altered consciousness
Hypertension (SBP >150 mmHg)	Large tumour resection with midline shift
Hypotension–hypovolemia	Long lasting surgery (>6 h)
Haematocrit <25%	Intraoperative brain swelling
Hypoxia or hypercapnia	Injury to cranial nerves (IX, X, XII)
Ineffective spontaneous ventilation	Convulsions during emergence
Hypoosmolality (<280 mOsmol/kg)	
Disorders of coagulation	
Residual neuromuscular blockade	

SBP: systolic blood pressure

problems from the operating room to the post anaesthesia care unit. There is some evidence that an explosive emergence and the lack of haemodynamic control during extubation may lead to cerebral complications such as worsening of cerebral oedema or haemorrhage. Further studies are needed, however, in order to understand the relationship between cerebral complications and management during emergence. Early recovery should be performed after most neurosurgical procedures because clinical assessment is the best neurological monitoring. The physiological changes during emergence and the associated complications should be anticipated to limit their effects on the cerebral circulation and metabolism. Thus, emergence and extubation should be performed with the same monitoring and the same anaesthetic care as during the surgical procedure. This is the necessary condition to perform fast-tracking neuroanaesthesia without increasing the complication rate.

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