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Recovery from Anesthesia and Postoperative Extubation of Neurosurgical Patients: A Review

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Summary: The most feared complications after intracranial surgery are development of an intracranial hematoma and major cerebral edema. Both may result in cerebral hypoperfusion and brain injury. Arterial hypertension via catecholamine release or sympathetic stimulation and hypercapnia may be predisposing factors. Other systemic secondary insults to the brain such as hypoxia and hypotension may exacerbate neuronal injury in hypoperfused areas of the brain. Thus, the anesthetic emergence of a neurosurgical patient should include maintenance of stable respiratory and cardiovascular parameters. Minimal reaction to the endotracheal tube prevents sympathetic stimulation and increases in venous pressure. On one hand, a delayed emergence and later extubation in the intensive care unit (ICU) might be recommended to achieve better thermal and cardiovascular stability after major intracranial procedures. On the other hand, the timely diagnosis of neurosurgical complications is required to limit brain damage; the diagnosis of complications relies on rapid neurological examination after early awakening. After uncomplicated surgery, normothermic and normovolemic patients generally recover from anesthesia with minimal metabolic and hemodynamic changes. Thus, early recovery and extubation in the operating room is the preferred method when the preoperative state of consciousness is relatively normal and surgery does not involve critical brain areas or extensive manipulation. In the complicated or unstable patient, the risks of early extubation may outweigh the benefits. It is, however, often possible to perform a brief awakening of the patient without extubation to allow early neurological evaluation, followed by delayed emergence and extubation. Close hemodynamic and respiratory monitoring are mandatory in all cases. The availability of ultrashort intravenous anesthetic agents and adrenergic blocking agents has added to the flexibility in the immediate emergence period after intracranial surgery. **Key Words:** Recovery, awakening—Neuroanesthesia, neurointensive care unit—Hemodynamics—Metabolic response, catecholamines.

Postoperative neurosurgical complications may have devastating effects leading to death or severe disability. Anesthesiologists play a key role in the prevention of these complications, because many factors that influence cerebral blood flow (CBF), cerebral metabolism ($CMRO_2$), and intracranial pressure (ICP) are under their control. The anesthesiologist also has a major role in the early diagnosis of complications, because the reliability of the postoperative evaluation depends, among other factors, on the anesthetic technique used during the procedure. The authors review the physiological changes of anesthetic recovery after intracranial surgery and give recommendations for the management of postoperative recovery and extubation while underlining the importance of patients being awakened and extubated rapidly after a craniotomy.

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PHYSIOLOGICAL CHANGES DURING RECOVERY AFTER NEUROSURGICAL ANESTHESIA

Recovery from general anesthesia and extubation is a period of intense physiological stress for patients. Physiological stress is generally defined as the nonspecific response of an organism to a stimulus, which may apply to a wide range of clinical situations. In this review, postoperative stress is defined as the response to pain, hypothermia, discomfort caused by the presence of the endotracheal tube or catheters, and external stimulation during awakening. There are several physiological responses to postoperative stress, including increases in oxygen consumption (VO_2), catecholamine blood levels, blood pressure, and heart rate. In most cases, blood pressure and heart rate increase gradually towards preoperative values or higher as the patient awakens (Fig. 1). Tracheal extubation causes an additional transient increase in these parameters via tracheal and laryngeal stimulation, although *in fine* it relieves the stimulation of the endotracheal tube itself (1). Shivering and pain are also among the main causes of these metabolic, hemodynamic, CBF, and ICP changes.

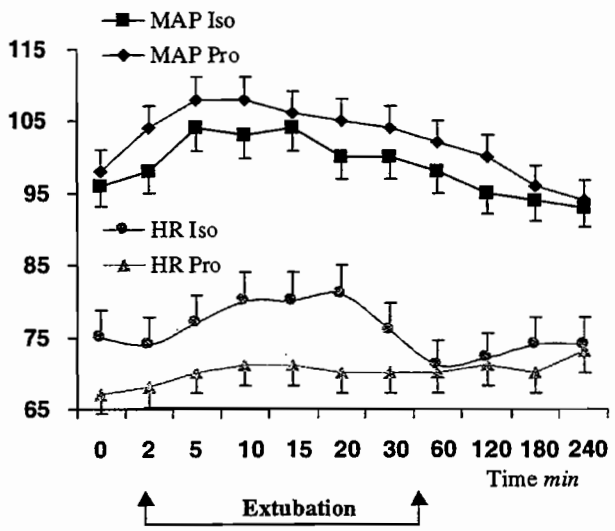

FIG. 1. Changes in mean arterial pressure (MAP) and heart rate (HR) during recovery after isoflurane (Iso) or propofol (Pro) anesthesia. (Reprinted, with permission, from Ravussin P, Tempelhoff R, Modica PA, et al. Propofol vs. thiopental-isoflurane for neurosurgical anesthesia: comparison of hemodynamics, CSF pressure, and recovery. *J Neurosurg Anesthesiol* 1991;3:85–95.)

Systemic Metabolic Changes

Increases in VO_2, sympathetic activation, and catecholamine release are the most relevant metabolic changes during recovery. As a result of the intraoperative use of inhalation anesthetic agents, intraoperative hypothermia, or both, shivering may induce large increases in VO_2. It occurs in approximately 40% of patients recovering from general anesthesia with a body temperature of less than 36.5°C (2). Although systemic and cerebral metabolic changes have been extensively studied in head trauma patients, there is, to our knowledge, no specific study on these parameters in neurological patients. In a study by Ciofolo et al (3), all patients shivered after isoflurane anesthesia for minor orthopedic procedures at a mean rectal temperature of 35.3 ± 0.2°C. VO_2 and mean arterial pressure (MAP) increased from 173 ± 26 ml/min and 76 ± 3.8 mm Hg at the end of anesthesia to 457 ± 88 ml/min and 105 ± 4.6 mm Hg during awakening (3). Other studies also showed that shivering is associated with a 200 to 400% increase in VO_2 (4–7). Furthermore, compared with patients who are maintained in a normothermic state, patients developing mild hypothermia during surgery (core temperature between 35°C and 36°C) experience a much greater increase in norepinephrine concentration, more significant vasoconstriction, and an increased arterial blood pressure in the early postoperative period (8). Normothermic nonshivering patients usually experience less increase in VO_2 (6,8–10). Forced air skin-surface warming decreases the incidence and intensity of shivering, which may explain a lack of shivering, despite the mild hypothermia still present after surgery (11,12).

Although VO_2 and hemodynamic changes might be predicted to closely covary, experimentally, there is only a weak relationship between the two (9) (Fig. 2). In a recent study, the difference in VO_2 between shivering and nonshivering patients was only 38%. This was explained by the advanced age of the patients (mean age, 70 years), which may have impaired the thermoregulatory response to hypothermia (8). Normothermia may not be obtained in all patients at the end of surgery, especially when moderate hypothermia is used intraoperatively for the purpose of brain protection. When body temperature is decreased to 34.3°C, the patients are still hypothermic at the end of surgery and shiver on admission to the intensive care unit (ICU), despite active warming during surgical closure (13).

Pain is another key stress factor which increases postoperative VO_2 and induces the release of catecholamines (6,14,15). Analgesia blunts the increase in plasma catecholamines both during and after surgery with a close

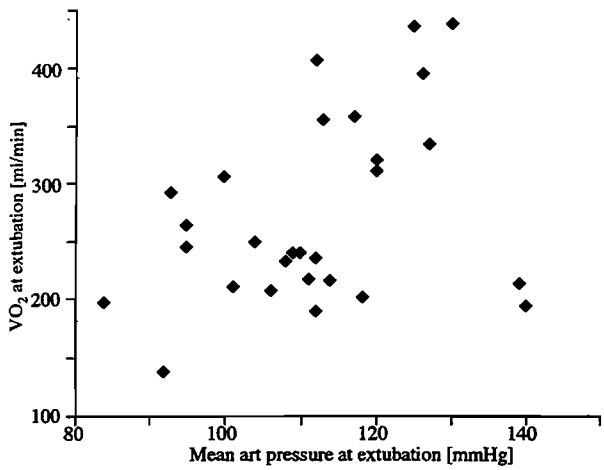

FIG. 2. Relationship between mean arterial pressure (MAP) and oxygen consumption (VO_2) at extubation in neurosurgical patients ($P < .01$; $r = 0.31$). *(Data from* Bruder N, Pellissier D, Ravussin P, et al. Evolution of oxygen consumption in neurosurgical patients during recovery from propofol anesthesia (abstract). *Br J Anaesth* 1995:74(suppl):A234; with permission.*)*

relationship between pain score and noradrenaline concentrations (16,17). It is difficult to separate the analgesic effects from the sedative and antishivering effects of the narcotics, however. Furthermore, comparison of pain scales among different studies makes it difficult to measure accurately the effect of analgesia on VO_2. It is usually accepted that morphine analgesia in critically ill patients reduces VO2 by 20% (14), but the decrease may be larger in restless patients. The study by Combes and Lavagne (18) underlines the importance of peroperative analgesia on VO_2 in the immediate postoperative period. Compared with abdominal or thoracic surgery, intracranial surgery is not considered to be extremely painful postoperatively. Thus, significant postoperative analgesia is usually not needed. Postoperative pain may depend on the anesthetic technique used. The intraoperative infusion of remifentanil does not provide any residual analgesia during recovery. In a trial comparing fentanyl and remifentanil for use in craniotomies, analgesics were required earlier in patients receiving remifentanil (19).

Recovery of spontaneous ventilation may also contribute to the postoperative increase in VO_2 and to the release in catecholamines (20,21). Large increases in VO_2 during weaning from controlled ventilation were measured in patients with cardiorespiratory disease (22). This was a result of the increase in the work of breathing as well as of that of a decrease in the efficiency of the respiratory muscles. With the exception of multitrauma patients, neurosurgical patients are usually free of cardiorespiratory disease, however. In healthy individuals, the oxygen cost of breathing accounts for less than 5% of total body VO_2, and weaning from controlled ventilation after neurosurgery should not increase VO_2 by more than 10% (23).

Rapid recovery from anesthesia to full awareness is also part of the increase in VO_2 whether it is a simple return from hypometabolism or is accompanied by anxiety and fear linked to the diagnosis, a hostile environment, or both.

In a recent study, immediate versus delayed recovery was compared after neurosurgery in 30 patients (24). All patients were free of cardiac or respiratory disease. They were actively warmed during anesthesia and were normothermic at the end of surgery. Emergence from anesthesia was associated with modest increases in catecholamine levels, VO_2, and MAP. Early recovery and extubation were associated with fewer metabolic and cardiovascular changes than a 2-hour delayed recovery (24) (Figs. 3 and 4). This may be the result of a higher residual opioid or propofol plasma level immediately after surgery, but other

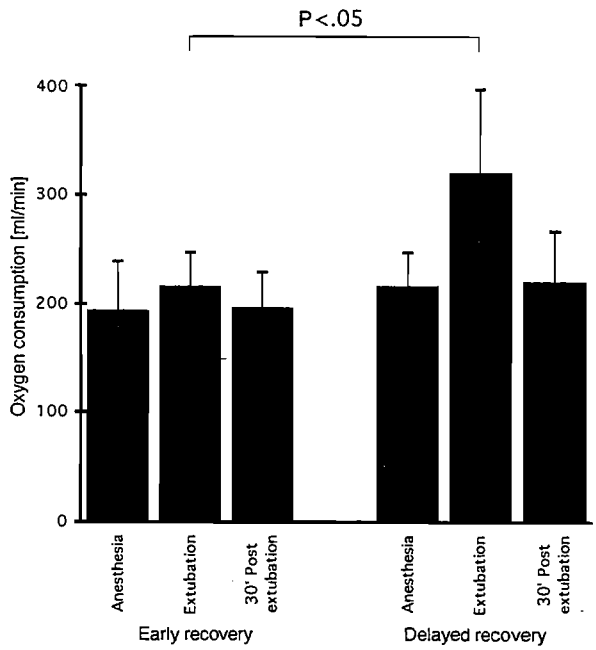

FIG. 3. Changes in oxygen consumption (V_O2) in two groups of 15 patients extubated immediately (early recovery) or 2 hours (delayed recovery) after the end of surgery. *(Adapted from* Bruder N, Stordeur JM, Ravussin P, et al. Metabolic and hemodynamic changes during recovery and extubation in neurosurgical patients: immediate versus delayed recovery. *Anesth Analg* 1999, (in press); and Bracco D, Ravussin P, Stordeur YM et al. Early awakening or long term sedation after neuroanesthesia. *Eur J Anaesth* 1998;15(suppl):28–32; with permission.*)*

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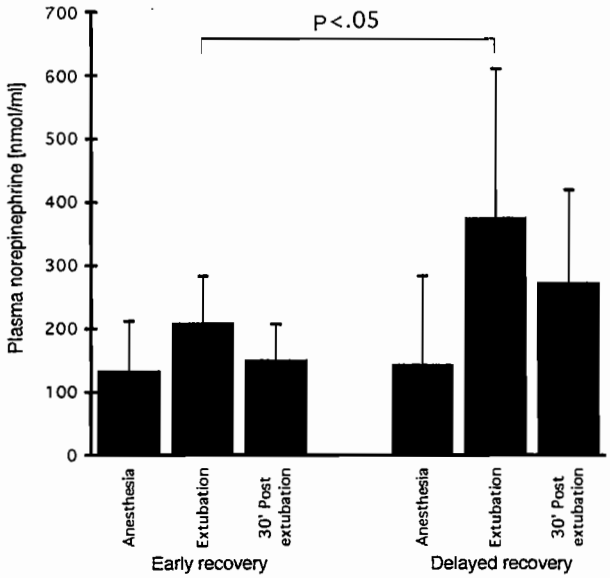

FIG. 4. Changes in norepinephrine concentrations in two groups of 15 patients extubated immediately (early recovery) or 2 hours (delayed recovery) after the end of surgery. (Adapted, with permission, from Bruder N, Stordeur JM, Ravussin P, et al. Metabolic and hemodynamic changes during recovery and extubation in neurosurgical patients: immediate versus delayed recovery. *Anesth Analg* 1999 (in press); and Bracco D, Ravussin P, Stordeur YM et al. Early awakening or long term sedation after neuroanesthesia. *Eur J Anaesth* 1998;15(suppl):28–32.

factors may have also played a role. After cardiac surgery, the hemodynamic responses and ST-segment changes were markedly reduced in sedated compared with awake patients (25). Both studies tend to show that a nearly "stress-free" recovery is possible immediately after surgery, which may be more difficult to achieve in the ICU several hours later when sedation as well as the opioid plasma level may be lower.

Hemodynamic changes

Although the definition of hypertension and the threshold values to treat it during emergence vary among anesthesiologists, hypertension is frequent after neurosurgery. If a greater than 20% increase in blood pressure is considered for treatment, 70 to 90% of patients require antihypertensive therapy during emergence (26,27). This did not depend on the anesthetics used during surgery in one study (27). In another study, however, the use of remifentanil, without analgesia before extubation, was associated with a higher systolic blood pressure during recovery, which was probably related to a higher level of pain during the early postoperative period (19). The increase in blood pressure during emergence and extubation is probably reduced by analgesia, partially through a decrease in catecholamine release (28,29).

Thus, the metabolic and hemodynamic changes during recovery from general anesthesia are highly variable and depend on multiple factors. Although some of these factors are not under the control of the anesthesiologist (e.g., preoperative patient status or the type of surgical procedure), most of the deleterious conditions seen during the recovery period are, at least in part, preventable (e.g., postoperative hypothermia, severe pain, restlessness, coughing and fighting against the tube and the ventilator).

Changes in CBF, $CMRO_2$, and ICP

Effect of Perioperative Physiological Stress on CBF

Compared with the large number of studies on cerebral circulation during anesthesia, data are scarce on the immediate postoperative period. Nevertheless, it has been well established that many stressful events are accompanied by increases in CBF and $CMRO_2$. In animals, conditions of physiological stress such as pharmacological paralysis and hemorrhagic hypotension increase both CBF and $CMRO_2$ (30). In man, stress-related conditions such as shock, ethanol withdrawal, anxiety, and hypoglycemia are associated with increases in CBF and $CMRO_2$. The mechanisms involved in these changes are complex and not fully understood, but a catecholaminergic mechanism acting through β adrenoreceptors plays a role (30). In animals, blocking the β receptors or removal of the adrenal medulla can block or attenuate the stress-induced increase in CBF and $CMRO_2$. Elevated plasma catecholamines alone are not sufficient to increase CBF and $CMRO_2$ (31–33), however, because they poorly penetrate the blood-brain barrier. Adrenergic stimulation may increase CBF and $CMRO_2$ in the case of central nervous system lesions or an increase in the permeability of the blood-brain barrier, either of which may occur in neurosurgical patients.

Another hypothesis is the release of catecholamines within the brain. In rats, emergence from halothane anesthesia induces catecholamine activation in the vasomotor center with tachycardia and hypertension at the same time. This activation is blunted by the administration of α_2 agonists (34). Changes in CBF may be evaluated by transcranial Doppler ultrasonography. In a study of 30 neurosurgical patients, CBF velocities increased significantly during emergence from anesthesia. The maximum increase was recorded on extubation (+60% over preoperative value), and the changes were not related to the anesthetic technique used (intravenous versus inhalation), $PaCO_2$, or MAP changes (35). The increase in CBF ve-

locity lasted 30 minutes after extubation and returned towards the preoperative value 60 minutes after extubation. The significance of the stress-induced increase in CBF is not clear. In several situations such as hypovolemic shock or hypoglycemia, it could be a homeostatic mechanism, which would increase or restore substrate (glucose and oxygen) supply to the brain. Catecholamine release or sympathetic activation may also worsen neuronal injury in experimental models of incomplete cerebral ischemia (36), however.

Thus, it appears that important changes in CBF and $CMRO_2$ may occur during emergence from anesthesia. The clinical relevance of these changes is unknown, except in rare neurosurgical cases, particularly after brain arteriovenous malformation (AVM) extirpation or removal of large tumors. Indeed, severe cerebral edema or hemorrhage constitutes the major source of postoperative morbidity and mortality after AVM resection (37). The pathophysiology of these complications is controversial, but one mechanism for swelling or hemorrhage is cerebral hyperemia (38). Although deranged pathophysiology may account for catastrophic hemorrhage or brain swelling after AVM resection, the most common cause is probably related to technical considerations such as a residual AVM which ruptures in the immediate postoperative period. In this case, postoperative hypertension may be harmful. The development of noninvasive and nonradioactive methods for the evaluation of CBF might improve our understanding of the cerebral physiology during recovery from anesthesia.

The effect of increased sympathetic tone on CBF in altered physiological states is well recognized. Neurogenic influences may not be *necessary* for regulatory responses, but autonomic activity can modify autoregulation in important ways. For example, reflex sympathetic constriction of larger proximal conductance arteries in response to systemic hypotension is prevented by acute surgical sympathectomy or α-receptor blockade (39). In man, upper thoracic sympathectomy increases internal carotid artery diameter and CBF velocity (40). As a result, CBF is better maintained, as autoregulation is preserved at a lower MAP. This explains why drug-induced hypotension during anesthesia is better tolerated than hypotension as a result of hemorrhagic shock. This autoregulation-sparing effect of autonomic blockade may be an important component of anesthesia-mediated "cerebral protection" (41). Low parasympathetic tone by means of parasympathetic extracranial ganglionic sectioning appears to adversely effect the outcome from cerebral ischemia in a manner analogous to high sympathetic tone (42).

Effect of Opiate and Benzodiazepine Antagonists on CBF

Naloxone may be required if extubation is planned soon after the end of surgery and if an excessive dose of opioids has been given during anesthesia. For example, in one study, 7 of 31 patients who received a mean intraoperative dose of fentanyl of 34 g/kg needed an infusion of naloxone (19). Naloxone used after anesthesia does not seem to have any effect on CBF or $CMRO_2$ by itself, but it may induce a withdrawal reaction in narcotic-treated patients, resulting in a severe sympathetic stimulation (43,44), or create a too rapid passage from sleep (and/or analgesia) to full awareness (and/or pain). Severe hypertension and pulmonary edema have been described following naloxone administration (45,46). The risk of adverse cardiac events (especially myocardial ischemia) is usually low in neurosurgical patients, but the risk of cerebral hemorrhage caused by hypertension is real. It is, however, rather unusual to use large doses of opioids in neurosurgical patients, and naloxone should not be necessary.

Flumazenil is a benzodiazepine-receptor antagonist. It may be indicated in patients premedicated with benzodiazepine or receiving midazolam for the induction or maintenance of anesthesia (47). In patients undergoing craniotomy for supratentorial tumors, flumazenil given after midazolam anesthesia has no effect on CBF or $CMRO_2$ (48), but in animal studies and cases of head injury, flumazenil may induce major changes in CBF or ICP (49,50). Moreover, flumazenil antagonizes the antiepileptic effect of benzodiazepines. Thus, flumazenil should be used cautiously when reversing benzodiazepine-induced sedation in patients with impaired intracranial compliance or seizures.

ICP Changes

The effects of emergence and tracheal extubation on ICP have not been investigated, but it is clear that intracranial hypertension is a major complication after neurosurgery. In a retrospective study of 514 patients whose ICP was monitored after elective intracranial surgery, 89 (17%) had a sustained postoperative increase in ICP (51). Of the 89 patients with an elevated ICP, 47 (53%) had an associated clinical deterioration. The most common findings on computed tomography (CT) scanning were cerebral edema and cerebral hemorrhage. It has also been well documented that tracheal stimulation increases ICP (52). In several studies, the mean ICP increase caused by tracheal suctioning was around 15 mm Hg, with a large interindividual variability (52–55). This increase in ICP is related to arousal, coughing, and transient cerebral hyper-

emia (56). Its duration is variable, depending on brain compliance, but is usually less than 5 minutes. Topical or intravenous lidocaine as well as short-acting opioids are effective for limiting the coughing and intracranial hypertension caused by tracheal stimulation (54,57,58). On extubation, tracheal stimulation caused by the removal of the endotracheal tube is often associated with hypercapnia as a result of 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.

NEUROLOGICAL RECOVERY AND NEUROSURGICAL COMPLICATIONS

Recovery of Psychomotor Function

With the use of the new hypnotic agents (desflurane, sevoflurane, propofol), nearly all patients can respond appropriately to commands within 15 minutes after the end of anesthesia; this period is somewhat longer than after nonneurological surgery (59–61). All components of neurological function do not recover at the same time. After neurosurgery, focal neurological deficits that resolve rapidly in the postoperative period are common. The central nervous system of patients who have recovered from a focal neurological deficit is particularly susceptible to the effect of anesthetics. It has been demonstrated that low doses of midazolam or fentanyl (2.8 ± 1.3 mg and 170 ± 60 μg, respectively) can exacerbate or unmask focal neurological deficits in more than 60% of patients with prior compensated neurological dysfunction (62). This is probably a nonspecific effect of anesthetics and has also been described with the use of sufentanil (63). It probably explains some reports of the complete reversal of postoperative neurological deficits with naloxone (64). It is postulated that in patients with limited neuronal reserve, minimal impairment of the remaining functioning neurons may produce an exaggerated response (62). In clinical practice, the difference between a deficit revealing a neurosurgical complication and a deficit exacerbated by anesthetics is determined by means of its evolution with time. This is another good reason to use the most short-acting anesthetics possible in neurosurgery and thus to promote rapid recovery.

Factors other than anesthetics may explain a delayed recovery. Obviously, the nature of the intracranial lesion plays a role. When compared with noncranial surgery, craniotomy for tumor excision is associated with a prolonged return to preoperative mental status (65). For the same reason, the emergence of patients with large intracranial mass lesions is slower than that of patients with small tumors or after spinal surgery (65).

Although the clinical benefit of pharmacological or hypothermic intraoperative brain protection has not been demonstrated, these methods are commonly used during aneurysm clipping and other high-risk neurosurgical cases. Intravenous anesthetic agents (thiopental, etomidate, or propofol) are commonly titrated to achieve electroencephalographic burst suppression. Burst suppression does not correspond to some unique protective dose but is rather a convenient clinical end point that can be easily monitored by simple electroencephalographic methods. If barbiturates are used (which have the best evidence supporting a protective effect), doses resulting in burst suppression may impair postoperative neurological assessment. If early emergence after surgery is considered essential, propofol is probably the best choice. In one study, it was demonstrated that 32 of 42 patients (76%) could be extubated at the end of surgery (66). The 10 patients extubated later included 8 with a decreased level of consciousness preoperatively and 2 with a surgical complication.

Mild hypothermia decreases the metabolism of most drugs. This is consistent with the observation that mild intraoperative hypothermia delays extubation (67) and prolongs the time to reach fitness for discharge from the postanesthetic care unit (PACU) even if return to normothermia is not a criterion (68). Although there is a great deal of discussion and enthusiasm for the application of intraoperative modest hypothermia to ensure brain protection (67), it has not been shown to have added benefit to the well-established safety margins of current neuroanesthetic techniques. If mild hypothermia becomes the standard of practice rather than a conversion to delayed recovery from anesthesia, modification of the anesthetic technique may be required to take advantage of the MAC lowering property of hypothermia as well as the use of even lower doses of anesthetic agents. This leaves the preoperative neurological examination or intraoperative catastrophe as a major reason for delaying emergence of anesthesia and extubation of the trachea.

Postoperative Neurosurgical Complications

The most feared postoperative complication after neurosurgery is development of an intracranial hematoma either supra- or infratentorially. In three studies comprising 2305, 4992, and 6668 procedures, the incidence of this complication was 2.2%, 0.8% and 1.1%, respectively (69–71). The outcome was poor (patients with severe neurological deficits, vegetative, or dead) in 36 to 55% of these

cases. The risk factors for the development of this complication were disturbances of coagulation, emergency surgery, and postoperative hypertension. Most bleeding occurred within 6 hours of surgery (71), which suggests that hemodynamic control during the early postoperative period is essential. Arterial hypertension may also be secondary to intracranial hypertension. In this case, excessive reduction of blood pressure leads to severe decreases in cerebral perfusion pressure and thus to cerebral ischemia. The best monitoring to detect early intracranial bleeding is repeated neurological examination. A progressive decrease in the value of the Glasgow Coma Scale score should lead to emergency CT scanning and hematoma removal if necessary.

Seizures may precipitate serious complications like cerebral edema or hemorrhage, hypoxia, and aspiration. Rapid control of seizures is mandatory and may be achieved initially by the rapid injection of a benzodiazepine or other intravenous induction agent, followed by phenytoin loading. Phenytoin may also be given prophylactically in case of supratentorial surgery. Early postoperative seizures often require sedation, endotracheal intubation, and emergency CT scanning.

After posterior fossa surgery, the cranial nerves may be injured. The integrity of the airway depends on the proper function of cranial nerves V, VII, IX, X, and XII. Swallowing dysfunction is related to injuries of cranial nerves IX, X, and XII. Inattention to this potential complication can lead to silent aspiration and pneumonia. Patients should be evaluated prior to extubation of the trachea to ensure that they possess an intact gag reflex, have an adequate respiratory rate, and are responsive to verbal commands.

MANAGEMENT OF RECOVERY AFTER NEUROANESTHESIA

Before the end of surgery, it must be decided whether recovery and even extubation will be performed immediately after the end of the procedure or will be delayed until the patient is admitted to the ICU or PACU. In difficult situations, another possibility is to allow the patient to recover sufficiently so as to enable a neurological examination and then to sedate the still intubated patient for transport to the ICU or PACU. This may give anesthesiologists time to perform extubation in safer conditions if problems are anticipated.

Early Recovery

In the majority of patients, the benefits of early recovery outweigh the potential risks (Table 1). Recovery and extubation should be planned as early as possible after the end of surgery in patients with good preoperative condition who undergo an uncomplicated surgical procedure and are normothermic at the end of surgery (Tables 2 and 3). In these patients, the metabolic and hemodynamic changes of recovery are usually minor, and evaluation of neurological condition in an awake patient is the best and least expensive method of neuromonitoring available. Attention should be paid to postoperative analgesia if remifentanil is used for anesthesia.

In every case, an intravenous antihypertensive agent

TABLE 1. *Risks and benefits of an early versus delayed recovery*

Early awakening	Delayed awakening
Advantages	Advantages
— Earlier neurological examination and re-intervention if necessary (e.g., surgical clip replacement)	— Less risk of hypoxemia and/or hypercarbia
— Earlier establishment of baseline for further clinical assessment	— Better respiratory and hemodynamic control
— Less hypertension, less catecholamine burst	— Easier transfer to the ICU
— Done by the anesthetist familiar with specific patient: brain tightness, bleeding, course of surgery, etc.	— Better late hemostasis
— Surgery and recovery period separated	— Stabilization period in same position as during surgery
— Lower costs	— Normothermia more easily achieved
Disadvantages	Disadvantages
— Increased risk of hypoxemia, hypercarbia	— Interference with neurologic examination
— Difficult respiratory monitoring during transfer to the ICU (long corridors, elevators etc.)	— If patient reacts to endotracheal tube, potential for hemodynamic changes, catecholamine release, sympathetic stimulation
— Residual hypothermia	

ICU: intensive care unit.

TABLE 2. *Suggested conditions for early emergence*

Systemic homeostasis	Brain homeostasis
— Near-normothermia (T° > 36°C)	— Normal $CMRO_2$ and CBF
— Normovolemia, normotension (70 mmHg < MAP < 120 mmHg)	— Normal ICP at the end of surgery
— Spontaneous ventilation at relative normocarbia	— Antiepileptic prophylaxis
— Normoglycemia (glucose 4–8 mmol/l)	— Adequate head-up position
— No hypoosmolality (285 ± 5 mOsm/kg)	— Intact cranial nerves for airway protection
— Hematocrit >25%	
— No coagulation disorder	

CBF: cerebral blood flow; ICP: intracranial pressure.

TABLE 3. *Procedure for early emergence after intracranial surgery*

Checklist before attempting early extubation	Suggested awakening sequence
— Adequate preoperative state of consciousness — Limited brain surgery, no major brain trauma — No extensive posterior fossa surgery involving cranial nerves IX–XII — No major arterio-venous malformation resection: low risk of malignant postoperative edema — Near-normal body temperature and adequate oxygenation; cardiovascular stability	1. Prepare awakening (aim: avoid postoperative respiratory depression) — Discontinue middle or long acting opioids (bolus or infusion) approx. 60 min. before planned emergence — Stop anesthetic administration during skin closure (syringe of intravenous agent ready or hand on vaporizer . . .) — Let neuromuscular block decrease to 2/4. Antagonize muscle relaxants before extubation if necessary — Progressive rise of $PaCO_2$ to normoventilation 2. Avoid unnecessary painful stimuli — Remove head pins as early as possible — Remove packing, perform adequate suctioning before the patient is fully awake 3. Treat blood pressure bursts — Treat hypertension due to nocioception by boluses of intravenous agents or high concentration volatile bursts-consider sympatholytics — Treat hypertension symptomatically as necessary (goal: MAP < 120 mmHg; MAP > 120 mmHg–consider beta-blockers, possibly lidocaine) 4. Evaluate the patient — Perform brief, targeted neurologic examination 5. Transfer to PACU or ICU — Supplemental O_2 (avoid hypoxemia) — Continuous monitoring (ECG, BP, SpO_2) from the OR to the ICU or PACU

ICU: intensive care unit; OR: operating room; BP: blood pressure; ECG: electrocardiogram; PACU: postanesthetic care unit.

should be available immediately. In patients susceptible to the development of postoperative hypertension, the prophylactic infusion of an antihypertensive agent may be considered. Because sympathetic stimulation is responsible for the increase in blood pressure, the infusion of β-blocking agents seems logical. Esmolol and labetalol have no significant effect on ICP and have been used successfully in neurosurgical patients (72,73). Labetalol is given in small incremental doses of 0.25 to 1 mg/kg to a maximum dose of 2.5 mg/kg. Esmolol is administered with a loading dose of 500 mg/kg, followed by a continuous infusion rate of 50 to 300 mg/kg/min. Labetalol has a much longer duration of action, which may cause hypotension when stimulation is at a lower level following extubation. In a comparative study, there was no difference in the frequency or severity of hypotension between esmolol and labetalol (73). Other antihypertensive agents may also be used, but they have a less favorable pharmacological profile for patients with intracranial pathology. Calcium channel antagonists, nitroglycerin, and sodium nitroprusside are cerebral vasodilators, or at least venodilators. These agents may increase cerebral blood volume and ICP (74). α_2-Adrenergic agonists decrease sympathetic outflow and do not seem to have adverse effects on CBF or ICP. Because these agents are sedatives, they may be used if carefully titrated.

Transport of the patient from the operating room to the ICU or PACU should be undertaken only when hemodynamic, respiratory, and neurological parameters are stable. Oxygen is mandatory during transportation, and monitoring should ideally include electrocardiography, blood pressure, and SpO_2.

Delayed Recovery

If systemic or brain homeostasis is impaired, an early recovery may not be the best choice (Table 2). This can be found after lengthy (>6 hours) surgery, major intraoperative bleeding, large or complicated AVM resection, large

tumor resection with preoperative midline shift, and intraoperative brain swelling. In these cases, recovery is planned in the ICU or PACU, and sedation is usually slowly withdrawn after a stabilization period with close monitoring of the patient's neurological and hemodynamic responses. The rationale for this approach is that an altered state of consciousness impairs respiratory and hemodynamic efficiency, which, in turn, worsens brain function (Fig. 5).

After stopping sedation, the patient should be rapidly awake and alert, which justifies the use of rapidly metabolized hypnotic agents. Ideally, it should be possible to distinguish between a residual effect of the drug and neurological worsening a few minutes after discontinuation of sedation. In all studies comparing midazolam and propofol for short-term sedation, the time from discontinuation of the infusion to emergence or extubation was similar or shorter and less variable with propofol (75–79). It was also easier to obtain a stable level of sedation with propofol (79). The sedation maintenance dose of propofol is between 2 and 6 mg/kg/h. Bolus injection of propofol should be avoided or given at doses less than 0.5 to 1 mg/kg to limit the decrease in blood pressure (79). Usually, it is not necessary to infuse narcotics in sedated patients after neurosurgery. Opioids used for analgesia in the ICU are morphine, fentanyl, or sufentanil. These agents are not rapidly eliminated after infusion for a few hours and are potent respiratory depressants. Their use should be limited to avoid the risk of hypoventilation, hypercapnia, or intracranial hypertension. Remifentanil is rapidly metabolized but has not yet been evaluated in the ICU setting. The use of a fentanyl infusion may be applicable to hemodynamically unstable patients who do not tolerate propofol, however.

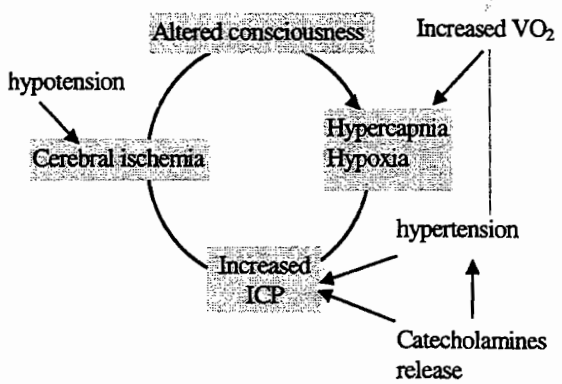

FIG. 5. Worsening of neurological deficits secondary to respiratory and hemodynamic disturbances.

Patients with injured cranial nerves IX, X, and XII should be carefully watched for 1 to 2 hours postoperatively to give them time to fully recover from anesthesia. The considerations for evaluation of the posterior fossa surgical patient are described above. Some patients may require the infusion of antihypertensive agents before extubation. In the minutes following extubation, an assessment of airway and swallowing functions may be performed using fiber-optic endoscopy.

Swelling of airway tissues has been described as a complication related to positioning in the sitting position after neurosurgery (80), but it can also occur in the prone position. This complication appears to be related to venous or lymphatic obstruction during excessive flexion of the head and compression by an oral airway. The edema usually regresses within 24 hours but may require a tracheotomy. This complication precludes early extubation, but the patient may be awakened for neurological assessment.

Difficult Situations

In some situations, the decision between an early or delayed recovery may be difficult. A trial of early recovery may always be attempted. Delayed recovery should be advised when several conditions listed in Table 2 are not present or if one physiological value is largely abnormal (e.g., temperature < 35°C). After a stabilization period of 1 to 2 hours, the situation may be analyzed again. An assessment of the neurological condition of the patient is nearly always necessary under close monitoring of the hemodynamic and respiratory parameters in the ICU or PACU. It may be performed soon after surgery while the patient awakens spontaneously. Once the neurological status has been evaluated, the patient is sedated until the extubation criteria are met. If the patient cannot be extubated, the sedation level should be adequately titrated with propofol to allow repeated clinical assessments but avoid restlessness, hemodynamic instability, hypoxia, and hypercapnia (Ramsay score II–III) (81).

CONCLUSION

Early recovery from neuroanesthesia and extubation is desirable in most cases because it allows clinical monitoring of the patient, which is essential to detect postoperative complications. Although there is no evidence that the choice of anesthetic agent influences outcome, time to awakening is more reliable after long surgical procedures when rapidly eliminated drugs (propofol, sevoflurane, desflurane, remifentanil) are used. Balanced anesthesia with the different drugs mentioned above may allow the

anesthesiologist the luxury of separating emergence from anesthesia and extubation. The flexibility offered by the balanced anesthetic technique allows low doses of various types of short-acting anesthetic agents and simplifies recovery and its titration. Pain and hypothermia should be avoided at emergence because they interfere with awakening, increase VO_2, and promote shivering, catecholamine release, and arterial hypertension. Extubation of the trachea must be carefully considered in terms of risk and benefit, however. Too rapid an emergence from anesthesia may lead to worsening of cerebral edema or cerebral hemorrhage as a result of severe hypertension. Furthermore, extubation of a patient who is not fully conscious may promote hypercapnia and aspiration. If there is doubt as to whether the patient should be extubated, a 1- to 2-hour sedation period allows time to correct hemodynamic and metabolic disturbances. In addition, a gradual emergence in the ICU makes it possible to decide whether or not extubation can be performed safely. In all cases, careful control of blood pressure, body temperature, and pain is necessary in the early postoperative period.

The availability of ultrashort-acting intravenous anesthetic agents and adrenergic blocking agents has added to the flexibility in the immediate emergence period after intracranial surgery. Pain and hemodynamic control then become much more feasible in the short term without committing the patient to prolonged intubation. Residual hypothermia and either preexisting or new neurological damage remain key issues for the timing of anesthetic emergence and extubation of the trachea.

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