POSTOPERATIVE NEUROSURGERY

Peter LeRoux MD, FACS
Department of Neurosurgery
University of Pennsylvania
Philadelphia, PA

Overview and the goal of postoperative neurosurgery

The practice of neurointensive care first developed to manage postoperative neurosurgical patients and now has evolved to provide comprehensive medical and neurological care to patients with life-threatening neurological disorders. Neurointensive care strives to prevent secondary neuronal injury and the neurointensivist integrates all aspects of neurological and medical management into a single care plan. All patients who undergo surgery under regional or general anesthesia, even a well-performed operation, are in a potentially unstable cardiorespiratory state and at risk for secondary neuronal injury. This is valid for “minimally invasive” interventions as well. The object of postoperative neurosurgical care is to prevent or minimize secondary neuronal damage and to improve functional central nervous system (CNS) recovery and patient quality of life. This effect may be the greatest on patients who are in better preoperative condition.

Evidence-based approaches to specific management after neurosurgical procedures are limited and concepts for individualized, patient-tailored therapeutic strategies are only beginning to be elucidated. After major general and cardiac surgery optimized care and use of goal-directed therapy improves patient outcomes when started in the immediate postoperative phase (1). There is substantial evidence that ICU care improves neurosurgical outcome (2). However there is little research to define the state of the CNS after neurosurgical procedures and to provide insight about the CNS consequences of this dynamic post-procedural and post-anesthetic phase. Therapy during this early phase of care may have a major impact on patient outcome since “time is brain”, and the just-operated-on CNS is very vulnerable to secondary insults. The well being of whole-body systems is a priority in care, but systemic, CNS-directed therapy also is essential. The basic goals therefore of postoperative neurosurgical care are:

- Provide smooth emergence from anesthesia
- Optimize post-operative hemodynamic, volume, and electrolyte status,
- Optimize airway and respiratory status
- Treat homoeostatic disorders
- Optimize management of complications and postoperative dysregulations
- Have reliable and appropriate systemic and neuromonitoring tools
- Use subtle and reproducible neurological examination methods
These goals depend on many variables; important questions to ask and answer include:

- What was status (medical and neurological) of the patient before surgery?
- What neurological disease is being treated?
- What other neurological disorders does the patient have?
- What position was the patient in during surgery?
- What procedure was performed (procedure specific and expected complications)?
- What happened during surgery, e.g. blood loss, vascular injury?
- What anesthetic technique was used?

The topic “postoperative neurosurgery” is vast and encompasses all facets of neurointensive care many of which will be discussed elsewhere in this review course. To manage a postoperative neurosurgical patient the neurointensivist requires knowledge of how the CNS reacts to stress and anesthesia and of potential complications associated with each specific procedure. Furthermore postoperative neurosurgery is a continuum and a dynamic process that begins with preoperative planning and intraoperative management. This section will focus on the following topics: 1) who goes to the NICU, 2) the effects of anesthetic agents on the CNS, 3) basic complications after neurosurgery, 4) effects and management of emergence from anesthesia, 5) extubation, 6) post-operative pain, 7) postoperative nausea and vomiting, 8) basic care, and 9) postoperative monitoring.

Who goes to and who stays in the NICU?

Postoperative neurosurgical cases account for many NICU admissions and in many institutions there is a flexible relationship between the NICU and PACU. Traditionally, patients who had neurosurgical procedures, and in particular craniotomies and other more complex surgeries, are nursed postoperatively and overnight in the ICU, before transfer to a neurosurgical ward. This may limit access to the ICUs for other patients and so it is helpful for each institution to have a written set of criteria based on patient diagnosis, preoperative condition, comorbidities (overall health), the surgical procedure, intraoperative complications, potential postoperative complications and postoperative status to identify which postoperative patients should come directly to the NICU. A patient's risk of prolonged stay (>1 day) can be predicted by preoperative radiologic findings (e.g. tumor location, mass effect), large intraoperative blood loss, fluid requirements, and the decision to keep the patient intubated at the end of surgery (3). Among those patients admitted primarily for observation (anticipatory admission) very few “patient days” are created and it is estimated that about 15% actually require and receive active treatment. Furthermore, when a patient stay in the NICU is <1 day about 50% require no interventions beyond postanesthetic care and frequent neurologic exams and in two-thirds of these patients no further interventions occur after the first 4 hrs.
Effect of anesthetic agents on neurosurgical patients

An important aspect of postoperative neurosurgical care is to distinguish residual effects of anesthetic agents (e.g. drowsiness or confusion) from signs that indicate intracranial pathology. While it often is believed that patients with neurological disease are prone to anesthetic effects this probably is not true particularly in those patients who are fully awake preoperatively. Confusion or dementia maybe exacerbated by anesthetic agents but it is rare for focal deficits to be aggravated by anesthesia. However, the effects of anesthetic agents are complex and depend in part on what is used. For example, increased reflexes and extensor plantar responses may be observed in 50-60% of patients who receive enflurane or ethrane, <30% who receive halothane and almost never with a nitrous – narcotic mix (4). Anesthetic associated abnormalities will return to normal when the patient is fully awake (follows commands). Nondepolarizing neuromuscular blockers may be associated with persistent weakness or ophthalmoplegia but usually the rest of the exam is benign. However opthalmoplegia should never be attributed to drugs alone. Occasionally anticonvulsant toxicity may cloud recovery. As a general rule, however, any progressive or fluctuating deterioration should be assumed to be a complication from the operative procedure rather than an anesthetic effect. When available SSEPs may help distinguish an anatomic from an anesthetic effect since drugs usually do not affect SSEPs.

It is beyond the scope of this review to address all anesthetic agents. In addition the “best” anesthetic regimen for neurosurgery still is debated. Anesthetics with a short context-sensitive half-time (i.e. the time required for the effect-site concentration of an IV drug to decrease by 50% at steady state), such as the opioids, remifentanil and sufentanil, are suitable for anesthesia when early neurologic assessment is preferred. Several studies have compared in a randomized fashion different drug combinations, balanced anesthesia e.g. sevoflurane-fentanyl, total intravenous anesthesia (TIVA) or inhalational anesthesia to examine impact on recovery (5, 6). The results are varied and rather than the specific agent used the effects may depend more on the way in which the agent is used.

How often and what complications occur?

Between 20 and 50% of neurosurgical patients may develop early postoperative complications and about 25% will have more than one complication (7, 8). Postoperative complications may be classified as: 1) general, 2) respiratory, 3) cardiovascular, 4) disease or procedure specific and 5) unexpected. Many of these complications are “minor”, the commonest being nausea and vomiting (30%), or shivering (18%). Younger patients who have elective spine surgery may be at most risk for minor complications. The incidence of other complications is difficult to determine and in part depends on the procedure and how the complications are classified but include: respiratory (3%), airway trauma (4%), cardiovascular (7%), and neurological (6%). Respiratory impairment (\(P_{O_2}\)<90 mm Hg or \(P_{CO_2}>45 \text{ mmHg}\)) may occur in about 25% of patients usually within the first 30 to 60 minutes. About 1% of patients require re-intubation. Overall serious complications may occur in 10% of patients.
In patients who undergo emergency surgery (90%) or have a depressed preoperative level of consciousness (Glasgow Coma Scale ≤8), this risk is greater (>40%).

**Postoperative hematomas:** About 2% of patients who undergo a cranial procedure will develop a postoperative hematoma (PICH). Overall about 0.8% patients develop a PICH that requires surgical evacuation. A decreased level of consciousness (60% of patients) is the commonest presentation. PICH also should be considered in those who do not recover or improve in the expected manner after surgery. A third of patients develop focal findings and when ICP is monitored about 90% will have elevated ICP. By contrast in the absence of a PICH, ICP is elevated in about 10% of patients. In most patients (50%) clinical deterioration associated with a postoperative hematoma occurs within 6 hours of surgery (9, 10). About 20% of PICH however may develop after one day and in particular patients who undergo posterior fossa surgery or emergency craniotomy often require longer periods of ICU observation. Risk factors for a PICH, particularly one that requires surgery include: meningioma surgery; intraoperative or immediate (12 hour) postoperative hypertension (11), intraoperative blood loss >500ml, age >70 years, hypoxia, coughing and hiccoughs, lower post-operative prothrombin time, fibrinogen and platelets. Two thirds of patients have a favorable outcome.

**Other complications:** It is important to recognize what may happen during surgery to best manage the patient after surgery. Complications depend in part on position or the procedure e.g.:

1) **Ocular:** Periorbital or conjunctival edema or chemosis may occur in the prone position or during pterional or orbitozygomatic craniotomies. Posterior ischemic optic neuropathy or central retinal artery occlusion also may occur. A third nerve palsy or blindness may result from posterior communicating artery or carotid ophthalmic artery surgery.

2) **Transphenoidal surgery** can be complicated by CSF leak, alterations in visual function (acuity, fields, ocular movement) and diabetes insipidus (DI).

3) **Use of a lumbar drain** may cause intracranial hypotension or hemorrhage distant to the surgical site

4) **Anterior cervical surgery:** Soft tissue swelling can cause airway obstruction or swallowing abnormalities

**The effects of recovery from anesthesia on neurosurgical patients**

Recovery from anesthesia and surgery is a period of intense stress for patients. There are several physiological responses including: an increase in oxygen consumption (VO2), sympathetic activation with catecholamine release, increase in blood pressure or heart rate, changes in arterial blood gases, or hyperglycemia, all of which affect the CNS. Extubation causes further sympathetic discharge via tracheal and laryngeal stimulation, although it relieves the endotracheal tube stimulation itself. Shivering, pain, and regaining awareness are additional stress factors in recovery.
Systemic effects

1) Shivering occurs in approximately 40\% of patients recovering from general anesthesia with a body temperature of \(<36.5{\degree}C\) and is associated with a 200–400 \% increase in \(VO_2\). Mild intraoperative hypothermia can increase norepinephrine or epinephrine release, which may extend into the early postoperative period. Forced-air skin-surface warming may reduce the incidence and intensity of shivering.

2) Pain is another stress factor, and appropriate analgesic therapy may blunt the increase in plasma catecholamines both during and after surgery. Most patients will experience moderate or severe pain in the first two days following major intracranial surgery (12).

3) Hypertension is frequent in the early postoperative period after neurosurgery. If a >20\% blood pressure increase is considered for treatment, 40 to 90\% of patients require antihypertensive therapy during emergence. Analgesics, and particularly narcotics, reduce the sympathetic and catecholamine response to pain and extubation. Patients with PICH are 3.6 times more likely to be hypertensive than their matched controls. In particular there is a very strong association between intracranial hemorrhage and patients being normotensive intraoperatively, but hypertensive postoperatively (11). Postoperative blood pressure generally is managed in the range of 120–150 mmHg systolic.

Cerebral effects: Stressful events, including surgery and emergence from anesthesia can alter CBF and CMRO\(_2\). Sympathetic stimulation acting through beta-adrenoreceptors may play a role.

1) CBF: TCD studies suggest that CBF velocities increase significantly during emergence from anesthesia. The maximum increase is at extubation (+60\% over preoperative value) and return to normal in about 60 minutes (13). The CBF increase is independent of anesthetic technique, \(PaCO_2\) or arterial pressure changes but can cause cerebral edema or hemorrhage, and postoperative confusion. Changes in CBF are associated with \(VO_2\) and so prevention of agitation, shivering and coughing is important.

2) ICP: There are few data on the effects of emergence and extubation on ICP. Endotracheal suctioning increases ICP (14). Similarly, extubation particularly when associated with coughing can increase ICP. The ICP increase usually lasts 2 or 3 minutes, but is longer when intracranial compliance is reduced. Up to 20\% of patients who undergo intracranial surgery may develop increased ICP and when it occurs half will develop clinical deterioration in large part from edema or hemorrhage (15).

3) Hyperemia and NPPB: The cerebral arterio-venous oxygen content difference (AVDO\(_2\)) often is depressed immediately after craniotomy, suggestive of transient cerebral hyperemia (13). This may result in hemorrhage or severe edema in 3–12.5\% of cases. Patients with large and deep AVMs, with low feeding artery pressure, with multiple arterial inflows but only a single venous draining vessel and with intense steal around the AVM nidus are at high risk for postoperative hyperemic complications including “normal perfusion pressure breakthrough”.

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These at risk patients may be best managed with a staged therapy for their AVM or a barbiturate based anesthetic continued into the postoperative period with tight blood pressure control and invasive cardiovascular monitoring to optimize filling pressure and cardiac performance.

Management of neurosurgical recovery and extubation

Waking-up patients and the emergence from general anesthesia after neurosurgery still have considerable variation in clinical practice. The anticipated speed of recovery of consciousness should account for preoperative, surgical and anesthetic factors. However, anesthetic emergence must maintain stable respiratory and cardiovascular parameters. The goal is to prevent adverse CNS effects. On one hand, a delayed emergence and later extubation in the ICU may achieve better thermal and cardiovascular stability after major neurosurgical procedures and so limit secondary insults. On the other hand, the timely diagnosis of neurosurgical complications is required to limit CNS damage; the diagnosis of complications relies on rapid neurological examination after early awakening and an awake patient is the best and the cheapest neuromonitoring available. If, after surgery, a patient does not rapidly recover consciousness, or a focal neurological deficit becomes apparent, imaging should be performed as soon as possible to exclude a surgical complication. On average, emergence time and extubation time should be 10-15 minutes and 18 minutes respectively. However many factors may contribute to delayed emergence including: 1) perioperative opiate analgesia and anxiolytics, 2) metabolic disturbances (electrolyte or acid-base), 3) comorbidity especially hepatorenal that affect drug clearance, 4) stroke, 5) pneumocephalus or CSF hypotension and 6) seizures.

Rapid awakening and recovery: The rationale for "rapid-awakening" after craniotomy with general anesthesia is that an early diagnosis of postoperative neurological complications can limit potentially devastating consequences. 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.

Delayed recovery: In the complicated or unstable patient, the risks of early extubation may outweigh the benefits. Delayed recovery is appropriate after: long (> 6 hours) surgery, surgery for large tumors or AVM resection, major intraoperative bleeding, complicated surgery, preoperative altered consciousness, severe cardiac or respiratory impairment, posterior fossa surgery where there is possible injury to lower cranial nerves, select cervical procedures where re-intubation may be difficult. 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. Alternatively an immediate postoperative CT may be obtained or ICP monitor placed. Before extubation, airway and swallowing functions should be carefully evaluated, and everything should be ready for a possible reintubation.
Extubation: The key question of respiratory management in the immediate postoperative period is: “to extubate, or not to extubate?” This is a particularly vexing question in neurosurgery since early postoperative clinical neurological assessment dictates early postoperative extubation but this must be balanced by the patient’s perioperative neurological status and prognosis, surgical concerns, and respiratory status. For successful extubation, the patient should be 1) awake, 2) fully reversed from neuromuscular relaxation and spontaneously breathing, 3) hemodynamically stable, and 4) normothermic (Table 1 and 2).

Weaning strategies and extubation failure: Weaning from mechanical ventilation means liberation from ventilator support and from the endotracheal tube. Standard weaning criteria includes normal mental status, and so these criteria are not always appropriate for neurosurgery patients. To be ready for extubation, the neurosurgical patient should successfully complete a spontaneous breathing trial. The initial trial should last 30 minutes and consist of either T-tube breathing or low levels of pressure support (≤8cmH₂O). A simple “leak test” with cuff deflation may help to identify laryngeal edema. Further details are provided in the recent report of the Task Force on Weaning from Mechanical Ventilation by the 6th International Consensus Conference on Intensive Care Medicine (Table 3). The GCS and partial pressure of arterial oxygen/fraction of inspired oxygen ratio are factors that may predict extubation. For example the success of extubation is >75% when the GCS is ≥8 but ~33% when GCS is <8 (16). Extubation failure is diagnosed after extubation if the patient develops one or more of the following: tachypnea (respiratory rate >25/min for 2 hours), clinical signs of muscle fatigue or increased work of breathing, oxygen desaturation (SaO₂ < 90%, PaO₂ < 80 on FIO₂ >0.5), hypercapnia (PaCO₂ >45 mmHg, or an increase by >20%), and acidosis (pH <7.33).

Postoperative pain

Surgery and the associated tissue injury and inflammation are almost always associated with postoperative pain. Pain is unpleasant and also associated with worse physical outcomes. Recent evidence indicates that post-craniotomy pain is reported as moderate to severe in up to 80% of patients and may persist for several days postoperatively (12). Pain however often is underestimated and undertreated in neurosurgery patients. There are several reasons for this: 1) the patient may not be able to communicate because of aphasia, altered mental status, or cognitive impairment, 2) the side-effects of analgesic drugs are feared, 3) there is ongoing debate about the choice of the “best” anesthetic regimen for intracranial surgery, 4) there is a lack of standardized, proactive protocols to assess and evaluate post-craniotomy pain and pain therapy, 5) few studies have examined this question.

Management of post neurosurgery pain: Analgesia needs will depend in part on the procedure. For example where there is extensive muscle dissection e.g. a suboccipital approach, pterional craniotomy where the temporalis muscle was divided, or thoracolumbar approach more analgesia will be required than procedures where there is little muscle dissection e.g. an ACFD or frontal craniotomy. Basic postoperative analgesia consists of opiates, non-steroidal inflammatory medicines, and paracetamol/acetominophen based preparations.
Postoperatively, these can be titrated depending on the patient's response or through patient controlled analgesia. When performed at the end of surgery, local anesthetic infiltration in the wound or nerve blocks (e.g. bupivacaine or marcaine) can provide short-term postoperative pain relief and so may stabilize the hemodynamic profile by blunting sympathetic responses to intraoperative stimulation.

Opioids are the mainstay of analgesia. All opioids blunt the respiratory response to hypercarbia and so there are concerns that opioid-induced carbon dioxide retention will trigger increases in cerebral blood volume and so aggravate cerebral edema with associated ICP increases. In addition there are fears that opioids may cause sedation, miosis, or interfere with recovery from anesthesia and postoperative neurological assessment. For this reason codeine phosphate is the most commonly used analgesic post-craniotomy (17). However when properly titrated, morphine does not increase side effects (respiratory depression, sedation, pupil size, or nausea) compared with codeine and is more effective (18, 19). Furthermore, codeine is an unpredictable pro-drug and most of its analgesic efficacy is derived from the 5–15% that is metabolized to morphine by hepatic CYP2D6. Inter-individual and ethnic differences in CYP2D6 can influence codeine’s efficacy e.g., because of genetic variation 15% of Caucasians do not experience any effect from codeine (20). Consequently morphine or fentanyl is recommended after surgery. The benefits (e.g. blood pressure control) outweigh risks. The non-narcotics ketoprofen, tramadol, and paracetamol (acetaminophen) or gabapentin may be useful as supplemental, opioid-sparing drugs or add-on agents. There is a need for larger trials to delineate safety and efficacy of post-neurosurgery analgesia with a focus on short- and long-term outcomes. Pain control is reviewed by Nemergut et al. (21).

**Post-operative nausea and vomiting (PONV)**

Postoperative nausea and vomiting are less common now that propofol is an induction agent. However PONV remains a common complication after neurosurgical procedures. The incidence is uncertain since study design may influence the outcome and few studies have looked specifically at neurosurgery PONV. Nevertheless PONV may complicate between 30 and 70% of neurosurgical procedures (22). While nausea is a source of patient discomfort PONV also can cause major complications, and post-craniotomy patients may be at an increased risk. Vomiting can lead to aspiration (particularly with a compromised swallowing reflex and impaired consciousness), electrolyte disturbances, ICP increases, and intracranial bleeding. During the pre-ejection phase of the vomiting reflex, there is sympathetic stimulation. This can complicate control of blood pressure postoperatively. Furthermore, increased intra-abdominal (>100 mmHg) and intra-thoracic pressure during the ejection phase directly translates into elevated ICP (23).

General risk factors for PONV include: 1) female gender, 2) previous PONV or motion sickness, 3) non-smoker, 4) duration of surgery >60 mins, 5) postop-opiods (24). Specific neurosurgical risk factors include: 1) surgery location (i.e., infratentorial, surgery near the area postrema at the floor of the fourth ventricle), 2) CSF space opened (chemical meningitis), 3) awake versus general anesthesia, 4) intraoperative CSF leak and subsequent pneumocephalus,
5) use of a fat graft for a CSF leak, 6) a lumbar intrathecal catheter and intracranial hypotension.

**PONV management:** Various pharmaceutical agents can be used to manage PONV. Serotonin (5HT₃) antagonists, such as ondansetron, are effective but expensive. Cyclizine is a cheap antihistamine commonly prescribed whenever opiates are given. Alternatives include dopamine antagonists, e.g. metoclopramide or droperidol. Steroids also work but there may be a ceiling effect (5 – 8mg). There are synergistic effects of dexamethasone and ondansetron. Intravenous ondansetron administration (4mg) at the time of dural closure can help reduce the incidence of PONV and the use of “rescue” antiemetics. However it seems that (5HT₃) antagonists have different effects on nausea and vomiting. Neufeld et al (22) preformed a recent meta-analysis of 7 prospective, randomized, placebo-controlled trials that together included 448 patients and found that ondansetron only had a significant impact on vomiting.

**Basic postoperative neurosurgical care**

Basic postoperative neurosurgical management is centered on the ABCs of care: 1) Maintain a secure airway, 2) Adequate respiration to maintain oxygen saturation, 3) Hemodynamic stability and fluid management. Other management goals are an awake patient, normothermia, normoglycemia and pain free (or has sufficient analgesia). “Normo – homeostasis” may be regarded as neuroprotective (25). Other aspects of postoperative neurosurgical care (seizure control, prevention and management of infection, venous thromboembolism, ventriculostomy care) are beyond the scope of this review. The typical postoperative patient probably does not require gastrointestinal prophylaxis.

**Respiratory care:** Adequate oxygenation and ventilation are required to balance oxygen delivery to the brain, cerebral blood flow, cerebral perfusion pressure, and ICP. The Brain Trauma Foundation recommends maintaining PaO₂ >60 mmHg and arterial oxygen saturation > 90% for oxygenation and ventilation of traumatic brain injury (TBI) patients (BTF). These are reasonable goals during postoperative neurosurgical care. The following complications may be observed:

**Airways obstruction:** This may be caused by many factors e.g. laryngospasm, soft tissue swelling around the pharynx (especially children) or laryngeal or glottic edema (e.g. anterior cervical surgery, carotid endarterectomy), foreign bodies (loose teeth), hypotonia of pharyngeal muscles from the remaining anesthetic, and viscous fluids (blood, e.g. transphenoidal surgery). Other risk factors for airway obstruction especially post-extubation include female gender, intubation > 3 days, and low height to endotracheal tube size ratio. In all patients who develop airway obstruction, a patent airway must be achieved immediately (head tilt chin lift, airway adjuncts, or intubation). The signs of airway obstruction include:

- Stridor
- Tachypnea (sometimes with tachycardia)
- Tracheal tug (downward displacement of the trachea during inspiration)
- Use of accessory muscles
• Intercostal and supraclavicular muscle recession
• Reduced oxygen saturations and hypoxemia (late signs)

_Hypoventilation:_ A reduced ventilatory capacity can be caused by a depressed neurogenic respiratory drive including opioid drugs, hypothermia and a metabolic alkalosis secondary to intermittent positive pressure ventilation or by mechanical difficulty in breathing. Impaired chest expansion may result from parenchymal lung disease (e.g. obstructive airways disease secondary to smoking), muscle weakness (e.g. electrolyte derangement), hindered diaphragm movement (pain, obesity), and the residual effect of paralyzing agents on the chest wall musculature.

_Hypoxemia:_ This is a PaO$_2$ that is lower than that expected in a healthy patient breathing the same oxygen concentration. The principal causes of hypoxemia include: 1) a reduced FiO$_2$, 2) hypoventilation associated with a depressed consciousness or airway obstruction and 3) ventilation or perfusion mismatch (e.g. lung collapse, atelectasis, bronchospasm, pulmonary edema, pneumothorax, pulmonary embolism). Thoracic and abdominal surgery often may alter the chest expansibility, and contribute to decreased oxygen saturation. This cause is less frequent after neurosurgery (unless a thoracotomy was performed [e.g. thoracic disc or anterior decompression]).

_Neurogenic pulmonary edema (NPE)_ is a potential complication of CNS insults such as intracranial hemorrhage including SAH, uncontrolled generalized seizures, TBI, tumors, and neurosurgical procedures. The postulated cause is sympathetic discharge. The treatment is mainly supportive (mechanical ventilation and alpha-adrenergic blocking agents while managing ICP).

_Who should be ventilated?_ Intubation and mechanical ventilation is indicated in neurosurgical patients in the following conditions: inability to protect the airway or clear secretions; need to reduce ICP by ventilation control; PaO$_2$ <60 mmHg despite supplemental O$_2$; PaCO$_2$ >50 mmHg, or pH <7.2; respiratory rate >40/minute or <10/minute; muscle fatigue; airway compromise; and hemodynamic instability. Orotracheal intubation with rapid sequence induction is the preferred technique. Nasotracheal intubation should be avoided particularly when there is a basilar skull fracture or skull base surgery. Maintenance of normocapnia is the major goal of ventilation therapy in neurosurgical patients. Whereas hypocapnia, achieved through short periods of hyperventilation is a potent cerebral vasoconstrictor, and can reduce ICP, it can exacerbate brain ischemia in patients with brain injury. Hyperventilation also decreases venous return, cardiac output, and PVO$_2$ and may increase V/Q mismatch.

Mechanical ventilation goals have changed from achieving normal blood gases to reducing the risks of ventilator-induced lung injury i.e. lung-protective ventilation strategy (26). Positive-pressure ventilation with a tidal volume of 6 ml/kg (or less) of an ideal body weight is used to maintain a plateau pressure <30 cmH$_2$O. Positive end-expiratory pressure (PEEP) is adjusted to keep FiO$_2$ <0.6 to prevent oxygen toxicity, with an oxygenation goal of PaO$_2$ >60 mmHg or SaO$_2$ >90%. The risks and benefits of lung-protective ventilation in neurosurgical patients are unclear.
Lung-protective ventilation may cause hypercapnia that can worsen brain conditions in patients with elevated or borderline ICP. In addition, variable clinical responses to PEEP may occur in neurosurgical patients secondary to PEEP’s effect on hemodynamic and respiratory variables. High levels of PEEP may decrease CPP due to decreases in cardiac output and increases in ICP (27). The influence of PEEP on ICP is less prominent in patients with stiff lungs (e.g., acute lung injury/ARDS), who may be the patients who most need PEEP. PEEP should be applied carefully in patients with increased ICP, and ICP should be monitored simultaneously.

**Cardiovascular management:** Cardiovascular disturbances (e.g. hypotension, hypertension, dysrhythmias and myocardial failure) are common in patients who undergo neurosurgery. They occur as consequences of medical or surgical therapy, central neurogenic effects on the heart and the autonomic system, or from concurrently associated medical conditions that interact with CNS pathology. The management goal is to prevent and correct the underlying cause. Potential iatrogenic induced cardiovascular problems include: diuretic and steroid-induced hypovolemia and hypokalemia-induced ventricular irritation, bradycardia and low cardiac output caused by surgical stimulation of the vagal nucleus in the brainstem, and a Cushing’s-like response with poor venting of ventricular perfusate during endoscopic third ventriculostomy. Prone and seated positions are associated with low cardiac output, venous return and blood pressure.

**Blood pressure control:** Changes in blood pressure are common postoperative complications. 

**Hypotension:** Fluid loss from the intravascular space (bleeding) and extravascular space (e.g. vomiting, diarrhea, and sweating) can contribute to hypovolemia ([Table 5](#)). This may exacerbate cerebral ischemia. Fluid therapy is discussed below. Circulation support to influence CBF is achieved best by increasing blood pressure, as cardiac output appears not to vary with CBF. The drug of choice to increase blood pressure is phenylephrine. With active baroreflexes, bradycardia may occur. Careful anticholinergic administration then is necessary to augment the sympathomimetic’s hypertensive action. Patients with low myocardial reserve may require an inotrope, such as dopamine or epinephrine.

**Hypertension:** Increased blood pressure that may be associated with pain, emergence and the underlying disease can lead to postoperative hemorrhage and exacerbate edema. Acute hypertension is associated with increased mortality in the NICU (11). The precise level that represents a risk varies and depends on patient, disease and procedure, lesion size, traumatic disruption of vessels, and premorbid BP. Management of hypertension is important. This includes prevention and timely treatment of bladder distention, pain and shivering. Since sympathetic stimulation is responsible for the blood pressure increase, beta-blocker infusions are largely used. Esmolol and labetalol are effective agents since they have no significant effect on ICP. Calcium channel antagonists, nitroglycerin, and sodium nitroprusside are cerebral vasodilators; these agents may increase cerebral blood volume and ICP. In patients who are on chronic anti-hypertensives their medications should be restarted at one half to two-thirds the dose. In patients with severe hypertension post operatively, the elevated blood pressure also should considered to be a sign of intracranial pathology. This is particularly important after posterior fossa surgery.
**Fluid status**: Osmolality is the primary determinant of water movement across the intact blood–brain barrier. Reduced serum osmolality can increase cerebral edema and ICP. The goals of fluid management after neurosurgery are: 1) maintain intravascular volume, 2) preserve CPP, and 3) minimize cerebral edema. In neurosurgical patients, and often in the postoperative period, intravascular volume is depleted (e.g. diuretic use or osmotherapy). Systemic hypotension (MAP <70 mmHg) and negative fluid balance (<594 ml) independently aggravate outcome in TBI patients (28). Basic fluid and electrolyte requirements must be considered in the postoperative period. In clinical practice, fluid management requires circulating blood volume assessment (Table 5). A patient generally is asymptomatic until the circulating volume has decreased by at least 10%. A persistently low urine output (<0.5 ml/kg/hour) may indicate inadequate fluid replacement and thirst often is the first sign of reduced intravascular volume even though other vital signs are in the normal range. However when diuretics or mannitol are given, urinary output can be misleading, and thirst is not present if the patient is drowsy or sedated.

**Fluid therapy**: Complications can result from inadequate volume replacement. However, excess fluid therapy (particularly in patients with comorbidities) can induce or exacerbate heart failure, pulmonary function or cerebral edema. There are few human data about the impact of fluids on the brain that can guide rational fluid management in neurosurgical patients. The optimal fluid to prevent secondary brain damage after neurological insult also is unknown. A formula often used to estimate the quantity of fluid needed for replacement is:

- **Replacement fluid** = Resuscitation + Maintenance + Losses (hidden, third space).

**Maintenance fluid requirements** are 1.5-2 ml/kg/hour (2.5 - 3.5l/24 hours for a 70 kg adult), and these generally are independent of the type of surgical procedure. Daily sodium maintenance is 1-2 mmol/kg/day (140 mmol a day) and potassium 1 mmol/kg/day (70 mmol a day).

To calculate losses, patient weight and the suspected percentage loss in blood volume (Table 5) are needed. Because 60% of body weight is water, in a 70 kg patient, 70 × 0.6 = 42 liters is the intravascular volume. With an estimated 10% circulation volume loss, volume loss is 42 × 0.1 = 4.2l. This and maintenance requirements of about 3l and any immediate resuscitation are needed. A fluid challenge (IV bolus of a fixed volume of crystalloid or colloid) can be used to assess and treat volume depletion, particularly in acutely ill patients. The central venous pressure should be monitored during and can guide further fluid administration. For example, if the CVP increases and then decreases after a fluid challenge, more fluid is needed. When blood has been lost, the volume of crystalloid replacement required is three times that of the estimated blood loss (only a third of the volume remains intravascular). If a large volume of blood is lost, replacement with blood is optimal.

**Which fluid?** Fluid administration that reduces osmolality should be avoided. Serum osmolality can be calculated from the following formula:

- **Calculated serum osmolality** = 2 x (Na) + urea/2.8 + glucose/18
Small volumes of lactated Ringer’s (1–3L) are unlikely to be detrimental and may be used. When larger volumes are needed a more isotonic fluid e.g. normal saline (0.9% NS) is preferred. Rapid NS infusion may cause a dose-dependent hyperchloremic metabolic acidosis (normal anion gap). When large volumes are needed, a combination of isotonic crystalloids and colloids should be considered. However Hetastarch and Dextran must be used with caution since coagulation disorders and platelet dysfunction may occur. Hypertonic saline now is more popular and it can move fluid from the body’s own extravascular space into the circulation across a sodium gradient (29). This is useful when there is hypotension and increased ICP. Hyperglycemia is an independent predictor of poor outcomes, and so it is reasonable to avoid glucose-containing fluids.

**Sodium balance:** Sodium homeostasis is critical in neurosurgical patients. Disorders of sodium metabolism including diabetes insipidus, syndrome of inappropriate ADH secretion (SIADH) and cerebral salt wasting syndrome need to be diagnosed and treated.

**Postoperative monitoring**

Systemic and neuro-monitoring are essential after neurosurgery to identify patients who deteriorate. However relatively few studies describe how postoperative monitoring influences outcome. Therefore, in most patients decisions about monitoring should be based on the patient’s presentation, the surgical procedure and clinical judgment. The most important monitor after elective neurosurgical procedures is the repeated clinical examination.

**Neurological evaluation:** Postoperative neurological evaluation is focused on two characteristics - consciousness and focal neurologic findings. The procedure may determine the focal finding to evaluate. For example, foot drop after an L4/5 lumbar discectomy, severe neck pain after a cervical laminectomy that suggests a postoperative hematoma, respiration after craniocervical surgery or dysphasia after left temporal surgery to name a few. Objective scoring instruments are useful since they can limit inter- and intra-observer variability and objectify subtle changes in function. Common instruments include: the Glasgow Coma Score, Full Outline of UnResponsiveness (FOUR score), Reaction Level Score, Mini Mental State Examination, and NIH Stroke Scale.

**Systemic:** Hypoxia and hypotension are the two most important systemic secondary insults in TBI patients, and it is likely that this also is true for postoperative neurosurgical patients. Therefore, oxygen saturation by pulse oximetry, and blood pressure should be continuously monitored. Continuous EKG also should be considered (e.g. severe arrhythmias may occur after SAH). Other cardiovascular monitors (e.g. pulmonary artery catheter, other cardiac output monitors) may be necessary for patients with pre-existing cardiac disease or neurogenic pulmonary edema, or in SAH patients. The arterial partial pressure of CO₂ (PaCO₂) is an important determinant of CBF. A PaCO₂ change can be determined by blood gas analysis or estimated from end-tidal CO₂ (ETCO₂). There, however, is debate about ETCO₂ reliability in neurosurgical patients.
Intracranial Pressure: The incidence of elevated ICP after neurosurgical procedures has had little study and most likely is underestimated. In addition, the impact of elevated ICP on outcome after neurosurgery has not been examined despite management of cerebral edema and elevated ICP being critical components of perioperative craniotomy care. Postoperatively elevated ICP can be expected in about 15% of patients. An ICP monitor should be considered in the following circumstances: large vascular tumors, severe edema, trauma surgery, exam cannot be followed, complication during surgery e.g. aneurysm rupture, known vessel occlusion, failure to wake up, multisystem trauma, large fluid shifts expected.

Other monitors: For most patients the extent of specialized neuromonitoring should be based on the clinical presentation and the experience of the responsible physician. This includes 1) bedside CBF assessment (e.g. jugular bulb oximetry, Transcranial Doppler sonography [TCD] Thermal diffusion flowmetry, Near infrared spectroscopy [NIRS]), 2) Microdialysis and brain tissue oxygen tension (PbtO₂) and 3) Electroencephalography (cEEG).

Imaging: Imaging is a snap-shot in time. CT investigations in critically ill neurosurgical patients are useful to monitor the course of the illness and for the early detection of complications and should be considered when neurological deterioration occurs or the expected postoperative improvement does not occur. When early detection of ischemia is necessary MRI is superior to CT since diffusion weighted imaging (DWI) can recognize ischemic injury within 30-60 minutes of onset. There is a well documented risk of transporting patients to scanners.

Reoperation

Reoperation is necessary in some patients. Removal of various types of hematomas is the most common surgical procedure at reoperation. Outcome is favorable in about half the patients indicating the importance of prevention. Factors associated with poor outcome include: histological type of the tumor, clinical state at admission, GCS score before urgent reoperation, time interval between primary surgery and urgent reoperation, and patient age (30).

Conclusion

Following a neurosurgical procedure, the patient remains vulnerable to secondary CNS injury because of the pathological changes associated with the disease, the procedure and the physiological changes associated with management. The level of care after surgery should be no less than that given during the procedure. Whereas the surgeon may influence patient's anatomy, it is the neurointensivist's role in collaboration with the neurosurgeon to ensure the patient's physiological stability and to navigate the transition from pre- and intra-operative care through recovery and return to the ward.
CITED AND RECOMMENDED LITERATURE


Table 1. Systemic and brain conditions necessary for rapid postoperative awakening and extubation after a neurosurgical procedure

<table>
<thead>
<tr>
<th>Brain Conditions</th>
<th>Systemic Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal pre-operative level of consciousness</td>
<td>Normothermia (~36°C)</td>
</tr>
<tr>
<td>Surgery &lt; 6 hours</td>
<td>Normovolemia, normotension (70 mmHg &lt; MAP&lt; 120 mmHg)</td>
</tr>
<tr>
<td>No major CNS or vascular injury</td>
<td>Spontaneous ventilation and PaCO₂ &lt;50 mmHg</td>
</tr>
<tr>
<td>No brain swelling</td>
<td>Normoglycemia (glucose 4-8 mmol/L)</td>
</tr>
<tr>
<td>Antiepileptic prophylaxis when indicated</td>
<td>Normosmolality (&gt;280 mOsm/kg)</td>
</tr>
<tr>
<td>Intact lower cranial nerves (IX,X,XII) - airway protection</td>
<td>Hemoglobin ~9g/dl</td>
</tr>
<tr>
<td></td>
<td>Normal coagulation status</td>
</tr>
<tr>
<td></td>
<td>No major swelling of face and tongue</td>
</tr>
</tbody>
</table>
Table 2. Checklist before extubation of a neurosurgical patient.
- Discuss expected postoperative course and potential complications and agree with the neurosurgeon about postoperative management
- Check antiepileptic prophylaxis
- Infuse analgesics before the end of anesthesia
- Check respiratory and cardiovascular parameters
- Check adequate recovery of muscle strength if muscle relaxants were used
- Check pupil size and awareness
- Prepare IV antihypertensive agents for blood pressure control
- Check adequate spontaneous ventilation with end-tidal CO$_2$ < 50 mmHg
- Check the vacuum system
- Be prepared to give supplemental oxygen after extubation
- Assess adequate recovery of neurologic function

Table 3. Factors associated with readiness to wean ventilator support in neurosurgical patients.

Clinical assessment
- Adequate cough
- Absence of excessive tracheobronchial secretions
- Resolution of disease acute phase for which the patient was intubated;
- Normal intracranial pressure (ICP)

Objective measurements
- Clinical stability
  - Stable cardiovascular status (i.e. heart rate $\leq$140 beats/min; systolic BP 90-160 mmHg, no or minimal vasopressors)
  - Stable metabolic status
- Adequate oxygenation
  - SaO$_2$ > 90% on FIO$_2$ $\leq$0.4 (or PaO$_2$/FIO$_2$ $\geq$150 mmHg)
  - PEEP $\leq$8 mmHg
- Adequate pulmonary function
  - Respiratory rate $\leq$35 breathes/minute
  - Maximal inspiratory pressure $\leq$ -20 to -25 cm H$_2$O
  - VT >5 mL/kg
  - VC >10 mL/kg
  - Rapid shallow breathing index <105 breaths/min/L
  - No significant respiratory acidosis

Abbreviations: BP = blood pressure; FIO$_2$ = inspiratory oxygen fraction; PaO$_2$ = arterial oxygen tension; PEEP = positive end-expiratory pressure; SaO$_2$ = arterial oxygen saturation; Rapid shallow breathing index = respiratory rate/VT; VT = tidal volume; VC = vital capacity.
Table 4. Spontaneous breathing trials in neurosurgical patients: failure criteria

Clinical assessment and subjective indices
- Agitation and anxiety
- Deterioration in mental status
- Diaphoresis
- Cyanosis
- Evidence of increasing effort
  - Increased accessory muscle activity
  - Dyspnea

Objective measurements
- Agitation and anxiety
- \( \text{PaO}_2 \leq 50-60 \text{ mm Hg on FIO}_2 \geq 0.5 \) or \( \text{SaO}_2 < 90\% \)
- \( \text{PaCO}_2 > 70 \text{ mmHg} \) or an increase in \( \text{PaCO}_2 \) by \( > 8 \text{ mmHg} \)
- \( \text{pH} < 7.32 \) or a decrease in \( \text{pH} \geq 0.07 \text{ pH units} \)
- Respiratory frequency \( > 35 \text{ breath/min} \) or increased by \( > 50\% \)
- Rapid shallow breathing index \( > 105 \text{ breath/min/L} \)
- HR \( > 140 \text{ breath/min} \) or increased by \( > 20\% \)
- Systolic BP \( > 180 \text{ mmHg} \) or increased by \( > 20\% \)
- Systolic BP \( < 90 \text{ mmHg} \)
- Cardiac arrhythmias

Abbreviations: BP = blood pressure; FIO\(_2\) = inspiratory oxygen fraction; HR = heart rate; PaCO\(_2\) = arterial carbon dioxide tension; PaO\(_2\) = arterial oxygen tension; SaO\(_2\) = arterial oxygen saturation.


Table 5: Clinical assessment of volume status in a 70 kg patient

<table>
<thead>
<tr>
<th>Blood loss (ml)</th>
<th>( \leq 750 )</th>
<th>750-1500</th>
<th>1500-2000</th>
<th>( \geq 2000 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (% of blood volume)</td>
<td>\leq 15</td>
<td>15-30</td>
<td>30-40</td>
<td>\geq 40</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>( &lt; 100 )</td>
<td>( &gt; 100 )</td>
<td>( &gt; 120 )</td>
<td>( \geq 140 )</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal</td>
<td>Reduced</td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td>Capillary return</td>
<td>Normal (( &lt; 2 ) s)</td>
<td>Prolonged</td>
<td>Prolonged</td>
<td>Prolonged</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min)</td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>( \geq 35 )</td>
</tr>
<tr>
<td>Urine output (ml/hour)</td>
<td>( &gt; 30 )</td>
<td>20-30</td>
<td>5-15</td>
<td>Negligible</td>
</tr>
<tr>
<td>Mental status</td>
<td>Normal</td>
<td>Anxious</td>
<td>Confused</td>
<td>Drowsy</td>
</tr>
</tbody>
</table>