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## The occasional neuroanesthesiologist—the questions to ask to assure success

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The preoperative work up of the neurosurgical patient obviously involves the «routine» history, physical and appropriate laboratory tests. However there are a few additional questions which will make planning the intraoperative care easier. These are:

What's the diagnosis and what operation will you do?  
What position will the patient be in?  
How much bleeding will there be?  
Do you anticipate any ischemia?  
Will there be any neuromonitoring?  
Is the ICP elevated?  
Where will the patient go afterwards?

### WHAT'S THE DIAGNOSIS AND WHAT OPERATION WILL YOU DO?

#### Acute subdural

These are usually associated with acute head trauma so that the underlying brain is injured as well. Management is focused on the associated elevated intracranial pressure (ICP) and the other injuries. Surgery is usually a craniotomy.

#### Chronic subdural

These usually occur in older patients who fell in the recent past and then developed slowly progressive neurological deterioration. Many are on anticoagulants for cardiovascular disease. The neurological deterioration is slow because cortical atrophy results in a lot of space for the hematoma to accumulate in before ICP starts increasing. The underlying brain is usually not injured. Initial anesthetic management may involve managing the elevated ICP but once the hematoma has been removed, the brain should be allowed to

fill the space i.e. PaCO<sub>2</sub> should be normal or slightly elevated. Surgery may be burr holes or a craniotomy.

### Intracerebral Hemorrhage (ICH)

Intracerebral hemorrhage is usually associated with trauma or hypertension. An aneurysm or AVM may also be the cause of the bleed and if not diagnosed preoperatively may result in torrential intraoperative bleeding.

### Tumor

These usually present with features of elevated intracranial pressure and/or seizures. Anesthetic management is focused on preventing increases in ICP and preferably lowering it.

### WHAT POSITION WILL THE PATIENT BE IN?

#### Supine

#### Lateral

*Modified lateral (Park Bench).* The patient is placed lateral and then leaned forward with the head turned towards the floor. It is used by some for posterior fossa and cervical procedures.

*Prone*—used for posterior fossa and spinal procedures.

*Sitting*—infrequently used these days because of concerns about air embolism.

### HOW MUCH BLEEDING WILL THERE BE?

Performing a craniotomy i.e. «the opening» should result in < 250 mL blood loss. Most intraparenchymal tumors are not very vascular and should not result in significant hemorrhage. Conversely meningiomas can be very vascular and

adherent. Preoperative angiography and embolization can often substantially reduce blood loss.

Cerebral aneurysms have the potential to bleed significantly although this is uncommon with experienced, competent aneurysm surgeons.

### **DO YOU ANTICIPATE ANY ISCHEMIA?**

The potential for neural ischemia may be an indication for neuromonitoring and the surgeon may request some (purported) neuroprotective drugs. There is abundant experimental evidence that currently used anesthetics e.g. sevoflurane, propofol, thiopentone, produce cerebral protection as assessed by multiple surrogate endpoints. However, in the context of neurosurgery there are no prospective randomized clinical trials showing a benefit to any of the commonly used techniques including drugs, shunts and physiological manipulation.

### **WILL THERE BE ANY NEUROMONITORING?**

Sensory and/or Motor Evoked potential monitoring are frequently used during spinal procedures and also intracranial, neurovascular operations. The purpose of the monitoring is to prevent ischemic injury. Prospective randomized trials of any of the neuromonitoring modalities are lacking and the best available studies are cohort studies and historical controls.

#### **Somatosensory evoked potentials (SSEP)**

Most commonly the median and/or the posterior tibial nerves are stimulated and the responses collected at the cervical and cortical levels. The SSEP indicates the integrity of the specific sensory neural pathway stimulated and injury to areas of the nervous system outside these tracts may not be detected. SSEPs are sensitive to inhalational anesthetics and become progressively suppressed as concentration increases. SSEPs are very much less influenced by intravenous agents such as propofol, opioids, thiopental. Thus suitable anesthetic choices are a TIVA anesthetic or low dose inhalational agent (<0.75 MAC) with an opioid.

#### **Motor evoked potentials (MEP)**

Clinical use of MEPs utilizes multiple transcranial electrical stimulations to stimulate a motor response in the upper and lower limbs. MEPs are very much more sensitive to anesthetic suppression than SSEP. Ketamine and etomidate

may actually increase the amplitude making them useful adjuvants when good quality signals are not being obtained.

### **IS THE ICP ELEVATED?**

Elevated ICP is most easily determined when it is directly measured although the majority of patients will not have intracranial monitors in place and a clinical estimate should be made from clinical signs e.g. headache, drowsiness, pupillary dilation, hemiparesis, and from CT/MRI e.g. midline shift and ventricular compression. It is also important to determine if the increase in ICP is sudden e.g. acute subdural or more gradual e.g. tumor. Patients with acute coma producing elevated ICP or very large lesions will have exhausted endogenous compensatory mechanisms and will be less tolerant of anesthetic techniques that may increase ICP. In such patients a prudent option may be a propofol-opioid infusion or sevoflurane-opioid at least until the dura is opened and the mass decompressed. In patients with well compensated masses the actual choice is likely less important. No prospective randomized trials have examined anesthetic impact on post operative patient outcome.

Propofol and thiopental have been shown to reduce elevated ICP. Of the vapors, sevoflurane is the least vasodilatory and does not seem to increase ICP until well above 1 MAC. No inhalational agent actually decreases ICP.

Hyperventilation has been a «tradition» in neuroanesthesia but has fallen into disfavor as there is evidence, at least with prolonged use in head trauma, that it produces ischemia and potentially a worse neurological outcome. The current recommendation is to keep the PaCO<sub>2</sub> in the mid 30's and to reduce it further only if needed and preferably for short periods. Our recent multicenter randomized blinded trial in elective cases found that hyperventilation (PaCO<sub>2</sub> 28) improved operating conditions and reduced ICP in patients with supratentorial tumors.

Other techniques to reduce ICP or a bulging brain include a head-up position, avoidance of venous outflow obstruction and mannitol. One should also eliminate or reduce the amount of cerebral vasodilators being used including (high dose) inhaled anesthetics and vasoactive drugs such as nitroprusside & nitroglycerine.

### **WHERE WILL THE PATIENT GO AFTERWARDS?**

The disposition of the patient to the ICU or the PACU may influence the anesthetic choice and may also be reflective of the severity of the neurologic impairment or the extent of the planned surgery.

## REFERENCES

1. Banoub M. Pharmacologic and physiologic influences affecting sensory evoked potentials: implications for perioperative monitoring. *Anesthesiology* 2003;99:716.
2. Bekker A. Dexmedetomidine for neurological surgery. *Neurosurgery* 2005;57:1-10.
3. Bekker A. Dexmedetomidine does not increase the incidence of intracarotid shunting in patients undergoing awake carotid endarterectomy. *Anesth Analg* 2006;103:955.
4. Bekker A. The effect of dexmedetomidine on perioperative hemodynamics in patients undergoing craniotomy. *Anesth Analg* 2008;107:1340.
5. Bloom M. Dexmedetomidine infusion and somatosensory evoked potentials. *J Neurosurg Anesthesiol* 2001;13:320.
6. Boisseau N. Comparison of the effects of sevoflurane and propofol on cortical somatosensory evoked potentials. *Br J Anaesth* 2002;88:785.
7. Cenic A. Cerebral blood volume and blood flow responses to hyperventilation in brain tumors during isoflurane or propofol anesthesia. *Anesth Analg* 2002;94:661.
8. Gelb AW. Remifentanyl with morphine for transitional analgesia results in more rapid neurological recovery than fentanyl in patients undergoing elective supratentorial craniotomy. *Can J Anaesth* 2003;50:946.
9. Gelb AW. Anesthetics and cerebral ischemia—should we continue to dream the impossible dream? *Can J Anaesth* 2001;48:727.
10. Gelb AW. Does hyperventilation improve operating condition during supratentorial craniotomy? A multicenter randomized crossover trial. *Anesth Analg* 2008;106:585.
11. Gelb AW. Hyperventilation – an ill wind that sometimes blows good. *Can J Anesth* 2008;55:735.
12. Haure P. The ICP-lowering effect of 10 degrees reverse Trendelenburg position during craniotomy is stable during a 10-minute period. *J Neurosurg Anesthesiol* 2003;15:297.
13. Himmelsehr S. Revising a dogma: ketamine for patients with neurological injury? *Anesth Analg* 2005;101:524.
14. <http://www.braintrauma.org> [provides guidelines for the anesthetic and surgical management of the patient with traumatic brain injury].
15. <http://www.trauma.org/neurotrauma> [outlines management of elevated intracranial pressure in trauma patients].
16. Kaisti KK. Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral blood flow, oxygen consumption, and blood volume in humans. *Anesthesiology* 2003;99:603-13.
17. Kalkman CJ. Effects of propofol, etomidate, midazolam, and fentanyl on motor evoked responses to transcranial electrical or magnetic stimulation in humans. *Anesthesiology* 1992;76:502-9.
18. Kaye A. The comparative effects of desflurane and isoflurane on lumbar cerebrospinal fluid pressure in patients undergoing craniotomy for supratentorial tumors. *Anesth Analg* 2004;98:1127.
19. Lips J. The role of transcranial motor evoked potentials in predicting neurologic and histopathologic outcome after experimental spinal cord ischemia. *Anesthesiology* 2002;97:183.
20. Matta BF. Direct cerebral vasodilatory effects of sevoflurane and isoflurane. *Anesthesiology* 1999;91:677.
21. Nathan N. Influence of propofol concentrations on multipulse transcranial motor evoked potentials. *Br J Anaesth* 2003;91:493-7.
22. Patel P. No magic bullets: the ephemeral nature of anesthetic-mediated neuroprotection. *Anesthesiology* 2004;100:1049-51.
23. Petersen KD. Intracranial pressure and cerebral hemodynamic in patients with cerebral tumors: a randomized prospective study of patients subjected to craniotomy in propofol-fentanyl, isoflurane-fentanyl, or sevoflurane-fentanyl anesthesia. *Anesthesiology* 2003;98:329-36.
24. Pinaud M. Effects of propofol on cerebral hemodynamics and metabolism in patients with brain trauma. *Anesthesiology* 1990;73:404-9.
25. Sloan T, Janik D, Jameson L. Multimodality monitoring of the central nervous system using motor-evoked potentials. *Curr Opin Anaesthesiol* 2008;21:560-4.
26. Talke P. A comparison of three anesthetic techniques in patients undergoing craniotomy for supratentorial intracranial surgery. *Anesth Analg* 2002;95:430-5.
27. Ubags LH. The use of ketamine or etomidate to supplement sufentanil/N<sub>2</sub>O anesthesia does not disrupt monitoring of myogenic transcranial motor evoked responses. *J Neurosurg Anesthesiol* 1997;9:228-33.
28. Zornow MH. Intracranial pressure effects of dexmedetomidine in rabbits. *Anesth and Analg* 1992;75:232-7.