

What our colleagues says.... Basic anesthetic considerations in neurosurgery

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ABSTRACT

Rapid development in neurosurgical practice poses new challenges for anaesthesiologists requiring search for newer and safer anaesthetic agents and modification in basic anaesthetic techniques. The present article describes the basic neuro-anaesthetic considerations and some of the newer developments in anaesthetic drugs and techniques which are important during neurosurgical practice.

Key words: Anaesthetic agents, anaesthetic techniques, neurosurgery

INTRODUCTION

Evolution of neurosurgery is accompanied by new challenges for the anesthesiologist. Increasingly, we must think not only as an anesthesiologist but also as a neurosurgeon and neurologist. With the recent advancements and focus on functional and minimally invasive procedures, there is an increased emphasis on the provision of optimal operative conditions, preservation of neurocognitive function, minimizing interference with electrophysiological monitoring, and a rapid, high-quality recovery to facilitate early neurological examination.

Patients who undergo neurosurgery have various types of intracranial pathology and systemic diseases and their responses to anesthetics may be different from those of normal subjects. Brain tissue hypoxia, acidosis, and edema are the main pathologic consequences of most of the brain disorders. Cerebral perfusion pressure (CPP) and carbon dioxide tension in the arterial blood (PaCO_2) are the important variables that influence cerebral blood flow (CBF). Autoregulation is the physiologic maintenance of constant CBF over a wide range of CPP values.

Anesthetic drugs and techniques influence cerebral circulation, metabolism and ICP. Some anesthetic drugs may have a potential for neuroprotective effects. Major goals in neurosurgical anesthesia are to provide adequate tissue perfusion to the brain so that the regional metabolic demand is met and to provide adequate surgical conditions (a “relaxed brain”). If anesthetic drugs or anesthetic techniques are improperly used, they can worsen the existing intracranial pathology and may produce new damage. Some anesthetics or anesthetic techniques may help to protect the brain subjected to metabolic stress or even ameliorate damage from such an insult.

EFFECTS OF ANESTHETIC DRUGS

Inhalational Anesthetics

In general intravenous anesthetics decrease cerebral metabolic rate (CMR) and CBF, whereas most inhalational anesthetics decrease CMR with an increase in CBF. All inhalational anesthetics are cerebral vasodilators and possess the capability of increasing ICP. Inhalational anesthetics with the possible exception of nitrous oxide (N_2O), usually depress metabolism. Despite the disassociation of CBF and CMRO_2 , changes in the magnitude of cerebral vasodilation appear to be related to the level of tissue metabolism.

Nitrous oxide

Nitrous oxide continues to generate debate in neuroanesthesia in terms of its effect on cerebral dynamics (CBF, CMR and ICP). Classic studies in humans demonstrated that N_2O did not significantly

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affect CBF, although it decreased the cerebral metabolic rate for oxygen (CMRO_2). However the effects of N_2O on CBFV are preserved in children during propofol anesthesia.^[11] It is now generally agreed that N_2O increases CBF, CMRO_2 and ICP. The most dramatic increase in CBF and ICP occurred when N_2O was administered alone or with other minimal background anesthetics. The increase in CBF and CMRO_2 with N_2O do not appear to be related solely to sympathetic hyperactivity. N_2O appears to be related to have no direct vasodilating effect.^[12] Potential benefits of induced hypocapnia in patients with intracranial pathology may be offset by the use of N_2O .^[13]

Halothane

In most animal experiments, it was found that halothane produces an increase in CBF with a decrease in cerebrovascular resistance (CVR). In humans also, most studies demonstrated that halothane induces cerebral vasodilation and increases CBF. The increase in cortical CBF appears greater with halothane than with enflurane or isoflurane at equal minimal alveolar concentration (MAC). The mechanism of the cerebral vasodilation produced by halothane has not been thoroughly understood. A direct effect on vascular smooth muscles or an increase in cyclic adenosine monophosphate (cAMP) in the brain has been postulated, but the evidence is inconclusive. Nitric oxide (NO) might be an important mediator of cerebral vasodilation produced by volatile agents.^[4-6] In both animals and humans, halothane raises ICP in a dose related fashion. The rise in ICP with halothane is maximum among the commonly used volatile anesthetics. However, at 0.5 MAC or less, the effect on ICP is minimal.

Isoflurane

Most animal studies show that isoflurane produces an increase in CBF accompanied by a decrease in CVR and CMRO_2 except at low concentration. It has been found that isoflurane is safe to use in patients with small supratentorial tumors in whom only a small midline shift has occurred.^[17] In a PET study in humans, isoflurane (0.2 to 1.0%) was reported to produce no change in global CBF.^[18] The increase in ICP caused by isoflurane, is mild and can be prevented by induced hypocapnia.

Enflurane

The effect of enflurane, a stereoisomer of isoflurane, are intermediate between those of isoflurane and halothane. Because of enflurane's potential effects on ICP and possible epileptogenic properties, high concentrations of the agent should be avoided in patients at risk. Low dose of enflurane can be used safely, but this agent does not seem to be useful in neurosurgical anesthesia.

Sevoflurane

PET demonstrated that either a decrease^[9] or no change^[10] in global CBF occurred with use of sevoflurane. It can be the result of reduction in CBF caused by CMR suppression.^[11] Middle cerebral artery velocity (Vmca) has been reported to be either decreased,^[12] unchanged^[13] or increased with sevoflurane (at less magnitude than with isoflurane).^[14] Sevoflurane produces small increase in ICP in both animals and humans and this can be blocked by hyperventilation. The extent of the increase in ICP is in the order: Desflurane > isoflurane > sevoflurane.^[15]

Desflurane

Desflurane has been reported to produce a dose dependent increase in CBF and a decrease in CMRO_2 .^[16] Because of its low blood/gas partition coefficient (0.42) relative to other clinically used volatile inhalational anesthetics, desflurane can provide rapid onset and offset of anesthesia, which facilitates early neurological evaluation. Desflurane at 1 MAC decreases CMRO_2 by 50% and CBF by 22% and preserves cerebrovascular CO_2 reactivity.^[17]

Intravenous Anesthetic Drugs

Barbiturates

Barbiturates were the first anesthetics to be examined for their cerebral vascular effects. Thiopental decreases CBF and cerebral metabolic rate for oxygen consumption (CMRO_2) in a parallel fashion up to the point of isoelectricity on the electroencephalogram (EEG). The changes in CBF are thought to be secondary to the changes in CMRO_2 (a coupled decrease in flow and metabolism). At the point of isoelectric EEG after the administration of thiopental, an approximately 50% decrease in CMRO_2 occurs without any toxicity. If high dose of barbiturates is used for cerebral protection, the endpoint of EEG burst suppression is often used to provide near-maximal metabolic suppression. However, the associated hypotension may require concomitant use of a vasopressor to maintain cerebral perfusion pressure (CPP).

Thiopental, even in high doses, does not appear to abolish cerebral autoregulation or CO_2 reactivity. As a result of the reduction in both CBF and cerebral blood volume (CBV), barbiturates lower ICP. Barbiturates are used clinically for this purpose and may even be effective when other methods for reducing ICP have failed.

Etomidate

Etomidate, like the barbiturates, reduces CBF and CMRO_2 . An isoelectric EEG can be induced with etomidate, and, as with thiopental, there is no evidence of cerebral toxicity as reflected by normal brain metabolites. In addition, no further reduction in CMRO_2 occurs when

additional doses are given after EEG burst suppression is achieved. A maximal decrease in CBF was achieved before the maximal decrease in CMRO₂.^[18] Myoclonus produced by the drug has the disadvantage of being misinterpreted as seizure activity in neurosurgical patients. Prolonged use of etomidate may suppress the adrenocortical response to stress. Less cardiovascular depression with etomidate as compared to thiopental makes this drug advantageous for the induction of anesthesia in trauma and elderly patients. Etomidate has also been shown to reduce ICP without decreasing CPP.

Propofol

Propofol produces dose-related reductions in both CBF and CMRO₂. In neurosurgical patients who are hypovolemic, the reduction in MAP might be substantial when they receive large bolus doses of propofol. A continuous infusion of propofol may be used intraoperatively as part of a total intravenous anesthesia (TIVA). The combination of an infusion of propofol and a narcotic (such as remifentanyl) is particularly useful when the monitoring of evoked potentials precludes the use of other than low concentrations of inhalational drugs. Propofol is also useful for sedation during awake craniotomies and as a substitute for an inhalational drug at the end of a general anesthetic to shorten the wake-up time.

Autoregulation and CO₂ response are preserved during the administration of propofol. Because it also reduces MAP, its effect on CPP must be carefully monitored. Nonetheless, propofol's ICP-lowering effect makes it useful in the intensive care unit (ICU) for the sedation of patients in whom elevated ICP is a concern. Propofol has the advantage of allowing prompt awakening which is advantageous in patients whose neurologic status needs to be evaluated serially. In the operating room, moderately deep sedation with propofol does not increase ICP in comparison to no sedation in patients undergoing stereotactic biopsy for brain tumors. During craniotomy for resection of brain tumors, ICP has been shown to be lower in patients who receive propofol-fentanyl in comparison to patients anesthetized with isoflurane-fentanyl or sevoflurane-fentanyl. The vasoconstrictive activity of propofol may be suitable for carotid endarterectomy^[19] and revascularization surgery for moyamoya disease,^[20] because the cerebral steal phenomenon can be avoided with propofol.

The antiemetic effect of propofol is also advantageous in neurosurgical patients because many of them receive large doses of narcotics, which are associated with a high incidence of nausea and vomiting leading to rise in ICP. Careful attention to sterile technique is essential when using propofol as an infusion because the solubilizing

agent in which propofol is prepared provides an excellent medium for bacterial growth.

Narcotics (opioids)

The effects of narcotics on CBF are difficult to characterize due to conflicting experimental reports. It appears, however, that low doses of narcotics have little effect on CBF and CMRO₂ whereas higher doses progressively decrease both CBF and CMRO₂. The reductions in CBF and CMRO₂ parallel progressive slowing of the EEG. However, burst suppression and an isoelectric EEG are never achieved. High doses of narcotics have been shown to produce seizures in laboratory animals but rarely in humans. Cerebral autoregulation and CO₂ reactivity are maintained with narcotics.

Under most conditions, narcotics produce either no change or a slight decrease in ICP. Narcotics can, however, increase ICP under certain study conditions. For example, the bolus administration of sufentanil has been shown to produce transient increase in ICP in patients who have severe head injury.^[21,22] Thus, when narcotics are administered to the neurosurgical patient, they should be given in a manner that does not cause a sudden reduction in MAP. The narcotic antagonist naloxone, when carefully titrated, has little effect on CBF and ICP. When used in large doses to reverse narcotic effects, use of naloxone may be associated with hypertension, cardiac arrhythmias, and intracranial hemorrhage.

Ketamine

Ketamine produces an increase in CBF and CMRO₂. The mechanism of the increase may be manifold in CBF. Respiratory depression with mild hypercapnia in spontaneously ventilating subjects, regional neuroexcitation with a concomitant increase in cerebral metabolism and direct cerebral vasodilatation. Although seizures have been reported in epilepsy patients receiving ketamine, generally no epileptiform activity is seen on EEG analysis. Cerebral autoregulation and CO₂ reactivity are maintained with ketamine.

During spontaneous ventilation, ketamine produces an increase in PaCO₂ and ICP, in both the presence and absence of pre-existing intracranial hypertension. Thus, it is usually avoided in most neurosurgical patients. Interestingly, ketamine is a noncompetitive N-methyl-daspartate antagonist. In an animal model of incomplete cerebral ischemia, so ketamine was shown to reduce cerebral infarct size.

Benzodiazepines

Benzodiazepines, including diazepam, midazolam and lorazepam, produce small decrease in CBF and CMRO₂

with both small and large doses. Contrary to the assumption that ICP would be reduced because of a lower CBF, diazepam (0.25 mg/kg) did not change ICP.^[23] As with the barbiturates, some of the CBF-lowering effect of benzodiazepines is thought to be secondary to a reduction in CMRO₂. Benzodiazepines are known anticonvulsants and are used clinically for this purpose.

CBF autoregulation and CO₂ reactivity are maintained with benzodiazepines. ICP effects are small with benzodiazepines, which cause either no change or a slight reduction in ICP. Midazolam is commonly used as a premedication in neuroanesthesia, with small intravenous doses titrated to the patient's response and as an anesthetic adjuvant.

Muscle Relaxants

Muscle relaxants do not cross the blood-brain barrier. Any cerebral effects are thus secondary to histamine release, systemic hemodynamic changes, actions of metabolites and altered cerebral afferent input.

Nondepolarizing Muscle Relaxants

Short-acting drug like Mivacurium metabolized by plasma cholinesterase and undergoes ester hydrolysis in the liver. It is commonly given by infusion because of its rapid metabolism. When large doses of mivacurium are given rapidly, histamine release can occur which can cause rise in CBF and ICP.

Intermediate-acting drugs like Atracurium can also release histamine when given in large bolus doses. It is metabolized by ester hydrolysis and Hoffmann elimination, so in patients with renal or liver dysfunction, its metabolism is not affected. Laudanosine, a metabolite of the Hoffmann elimination of atracurium, has been shown to cause seizures in laboratory animals. The newer analog of atracurium, cis-atracurium, does not cause histamine release and formation of toxic metabolites.

Vecuronium has the advantage of maintaining stable hemodynamics even when given in large doses. One possible exception is that bradycardia may occur when vecuronium is combined with large doses of narcotics for induction of anesthesia, leaving the vagotonic effect of the narcotic unopposed. Its stable hemodynamics and lack of cerebral effects have made vecuronium a popular choice in neuroanesthesia.

Rocuronium has a relatively stable hemodynamic profile (weakly vagolytic) and is excreted unchanged by the biliary system and the kidneys. Unlike vecuronium, it is not associated with the production of active metabolites. The rapid onset of rocuronium makes it an excellent

choice for intubation in the neurosurgical patient who is at risk for succinylcholine side effects.

Long-acting drug like Pancuronium causes hypertension and tachycardia leading to rise in CBF and ICP and which is rarely used now-a-days.

Depolarizing muscle relaxants Succinylcholine can cause an increase in CBF and ICP. This is secondary to muscle fasciculation, which increase cerebral afferent input.^[24] The changes in ICP are modest and transient. The other greater concern is the large release of potassium that occurs with certain neurologic injuries such as closed head trauma, cerebrovascular accidents, hemiparesis, spinal cord trauma, and neuromuscular disorders.

Positioning

Patient positioning for optimal surgical access during neurosurgery may have major implications for the neuroanesthetist and neurosurgeon.

Supine position

Patients with cerebellopontine angle tumors (e.g. acoustic neuromas) are often operated on in this position. However, access to the posterior fossa often requires substantial lateral rotation of the head, resulting in stretching of the jugular veins and brachial plexus. In patients undergoing craniotomy for cerebral aneurysm, a 10 degree reverse Trendelenburg tilt decrease ICP, although the CPP remain unchanged.^[25]

Prone position

The prone position offers good access to midline structures but bleeding can obscure the surgical field. Head-up tilt is used to reduce hemorrhage but this increases the risk of air embolism. The chest and iliac crests should be well supported to ensure free movement of the abdomen during respiration. Obese patients are at particular risk because of restricted diaphragmatic movement. The head is fixed in clamps in preference to a horseshoe to minimize pressure on the face and eyes. Pressure points should be well padded and the tracheal tube kept as secure as possible. In children with brain tumors, brain swelling and ICP rise is higher in the prone position than in supine.^[26]

Lateral position

The lateral position is suitable for approaches to lesions not in the midline, particularly the cerebellopontine angle. This position facilitates gravitational retraction of the dependent cerebellum and facilitates CSF and venous drainage. A variation called the 'park bench' position is often used because of its resemblance to the way a tramp sleeps on a park bench. A pad should be placed under the body in the axilla to minimize weight

on the lower arm and shoulder. The head is fixed in pins. Excessive flexion of the neck can obstruct the internal jugular veins. This can be avoided by ensuring a two or three fingers-breadth gap between chin and sternal notch.

Sitting position

The sitting position though rarely used, provides good surgical access to midline structures, improves surgical orientation and allows good drainage of blood and CSF. Functional residual capacity and vital capacity are improved and access to the airway is facilitated.

However, there are increased risks of cord compression, pneumocephalus and venous air embolism. Macroglossia and peripheral nerve injuries are also more common in the sitting position. Hypotensive techniques increase the risks of ischemic damage. Many authorities now contend that with modern anesthetic techniques there is no place for the sitting position in neuroanesthesia. Excessive head-up tilt in other positions, however, exposes the patient to similar risks. Despite the well-recognized complications of the sitting position, several case series have established its relative safety in carefully selected patients.^[27,28] A magnetic resonance imaging (MRI) study in healthy volunteers supports the clinical findings of an improvement in cerebrovascular and intracranial compliance in sitting position.^[29]

Although one of the oldest subspecialties of surgery, modern neurosurgery is where some of the greatest changes are happening and neuroanesthesia practice must keep pace. Although the basic principles remain the same, new challenges are faced as indications for functional and minimally invasive neurosurgery are increasing. Anesthesia provision for these new procedures may be very different, require new management strategies, and pose new risks. We may never reach a consensus on the ideal drug or the perfect recipe for neuroanesthesia and continue to choose drugs and techniques which accommodate specific surgical requirements and personal preferences. However, as technological advances and new anesthetic techniques push the boundaries of what or who is treatable more than ever; close cooperation among the neurosurgeon, neuroanesthetist, and patient is vital.

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Announcement

INTERNATIONAL CONFERENCES, 2012

No.	Conference	Date	Location	Contact information
1.	8 th International Congress on Meningiomas and Cerebral Venous System	May 1-6, 2012	Mumbai, India	http://www.neurosurgery2012.org/
2.	World Federation of Skull Base Societies 6 th International Congress	May 16-19, 2012	Brighton, UK	http://www2.kenes.com/skullbase/Pages/Home.aspx
3.	32 nd Meeting of the Japanese Congress of Neurological Surgeons (JCNS)	May 11-13, 2012	Yokohama, Japan	http://wwwsoc.nii.ac.jp/jcns/english/meetings.html
4.	The Society of Neurological Surgeons Annual Meeting	May 19-22, 2012	Atlanta, USA	http://www.societyofneurosurgeons.org/meeting_info.html
5.	9 th Annual World Congress for Brain, Spinal Cord Mapping & Image Guided Therapy	June 2-4, 2012	Toronto, Canada	http://www.worldbrainmapping.org/
6.	Neurosurgical Society of America Annual Meeting (NSA 2012)	June 10-13, 2012	Park City, Utah, USA	http://www.neurosurgicalsociety.com/cal/event_list.asp?ID=151
7.	Biennial Meeting - American Society for Stereotactic and Functional Neurosurgery	June 3-6, 2012	San Francisco, California, USA	http://www.assfn.org/#
8.	11 th Biennial Congress of the International Stereotactic Radiosurgery Society	June 16-20, 2012	Toronto, Canada	http://www.isrsy.org/view.php?id_27
9.	3 rd World Congress of Minimally Invasive Spine Surgery and Techniques	August 16-18, 2012	Bahia, Brazil	http://www.wcmisst2012.com.br/
10.	5 th International Cerebrovascular and Skull Base Workshop	September 6-8, 2012	Kyiv, Ukraine	http://www.skullbase2012.org.ua
11.	9 th Asian Congress of Neurological Surgeons (ACNS 2012)	September 2-5, 2012	Istanbul, Turkey	http://www.acns2012.com/
12.	The XX Congress of the European Society for Stereotactic and Functional Neurosurgery	September 26-29, 2012	Lisbon, Portugal	http://www.essfn2012.org/
13.	International Spinal Cord Society (ISCOS 2012)	September 3-5, 2012	London, UK	http://www2.kenes.com/iscos2012/Pages/Home.aspx
14.	62 nd Annual Meeting of the Congress of Neurological Surgeons	October 6-11, 2012	Chicago, Illinois, USA	http://w3.cns.org/meetings/2012/index.asp
15.	American Academy of Neurological Surgery 74 th Annual Meeting	October 17-20, 2012	Cape Cod, USA	http://americanacademyns.org/content.aspx?id=34
16.	EANS Annual Meeting 2012	October 24-27, 2012	Bratislava, Slovakia	http://www.eans.org/events/event-59/
17.	2012 Japan India Neurosurgical Conference	October 19-20, 2012	Osaka, Japan	kohata@med.osaka-cu.ac.jp
18.	Cervical Spine Research Society Meeting	December 6-8, 2012	Chicago, USA	http://www.csrso.org/web/index.html

NATIONAL CONFERENCES, 2012

No.	Conference	Date	Location	Contact information
1.	21 st Annual Conference of the National Neurotrauma Society Of India, Neurotrauma 2012	August 17-19, 2012	Kochi, Kerala	www.neurotrauma2012.com
2.	14 th Annual Conference of the Skull Base Society of India SKULLBASE 2012	September 23-24, 2012	Mumbai	skullbase2012@gmail.com
3.	Annual Conference of Indian Society of Pediatric Neurosurgery, NEUROPEDICON 2012	November 1-2, 2012	New Delhi	www.neuropedicon2012.com
4.	61 st Annual Conference of Neurological Society of India	December 19-22, 2012	New Delhi	www.neurocon2012.com