

ANESTHESIA MANAGEMENT IN INFRATENTORIAL SURGERY

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Introduction

Alternate name: Infratentorial brain tumors, Brainstem glioma. Posterior fossa tumor is a type of brain tumor located in or near the bottom of the skull. The posterior fossa is a small space in the skull, found near the brain stem and cerebellum. The cerebellum is the part of the brain responsible for movement. If a tumor grows in the area of the posterior fossa, it can block the flow of spinal fluid and cause increased pressure on the brain and spinal cord. Most of tumors of the posterior fossa are primary brain cancers, which originate in the brain, rather than spreading from elsewhere in the body (Tyson J).

Approximately 50% of tumors in children older than one year of age are infratentorial. Epidemiological features associated with each of the most common subtypes, as well as intrinsic tumors of the brainstem. Infratentorial Tumor Histology as Medulloblastoma (PNET) 30-55%, CerebellarAstrocytoma 25%, Ependymoma 20% (Sandberg DI). Because 60–70% of brain tumors in children occur in the posterior fossa, the pediatric neurosurgeon must be familia with the specialized techniques necessary for successful surgery in this region. (Hudgins, 1987).

Presentation varies significantly depending upon the patient's age, the tumor's location within the posterior fossa, and histological tumor classification. The most common presenting signs and symptoms are those associated with elevated intracranial pressure from hydrocephalus: headache, nausea, vomiting, and lethargy.

Infants may present with macrocrania and a full fontanelle or with more subtle findings such as irritability, failure to thrive, and loss of developmental milestones. Infants may also present with torticollis. Additional presentations include gait abnormality, other cerebellar signs such as dysmetria, or cranial neuropathies. Progression of tumors is typically more rapid for malignant tumors, such as medulloblastoma, than benign tumors, such as pilocytic astrocytoma (Sandberg DI).

There are no known cause or risk factors associated with them. Symptoms occur very early with posterior fossa tumors and may include: Ataxia (uncoordinated gait), Drowsiness, Headache, Imbalance, Nausea, Vomiting. Symptoms from posterior fossa tumor also occur when the tumor damages local structures, such as cranial nerves. Symptoms of cranial nerve damage include: dilated pupils, eye deviations, facial muscle weakness, hearing loss, loss of sensation of part of the face, taste disturbances, unsteadiness when walking, visual field deficits. Diagnosis is based on through history and physical examination, followed by imaging test. The best way to look at the posterior fossa is with an MRI. Posterior craniotomy (open brain surgery) or stereotactic biopsy (using special instruments to get a small piece of the tumor) can be used to obtain tissue for diagnosis. Tumors of the posterior fossa usually require surgical removal, even if they are benign (noncancerous). This is because of the delicate structures in the area that can be compressed by any abnormal growth and the frequency of symptoms associated with the tumors (Tyson J).

Case report

Here we have a successful report of anaesthetic for female, 48 years old, and 60 Kg bodyweight. She was diagnosed with Suspected Macroadenoma Progressive Hypophyse Cranial Nerve II. She underwent Craniotomy tumor removal to evacuate the tumor mass. Blood pressure was 154/94 mmHg, HR 90 x/m, RR 20 x/m, core temperature was 36⁰ C, and GCS was E₄V₅M₆. From head CT scan seen that tumor mass introduce through Medulla Oblongata. After arterial line attempt, induction was started with Fentanyl 100µg, Propofol (+Lidocaine 20 mg) 100 mg, Roculax 50 mg, Lidocaine 40 mg, Propofol 40 mg. Intubation with non kinking Endotracheal Tube no.7. Maintenance with Oxygen, Sevoflurane, Air, also Propofol continuous and Vecuronium (Norcuron) continuous by Syringe Pump. After 6 hours and 30 minutes, the anaesthesia for craniotomy ended, and the patient was transferred to ICU. The next two days patient was transferred to room care.

Discussion

The confines of the posterior fossa and the myriad of neuronal and vascular structures that traverse it create a challenge for the anesthesiologist, whose intraoperative goals are to facilitate surgical access, minimize nervous tissue trauma, and maintain respiratory and cardiovascular stability. This discussion focuses on the anesthetic considerations for posterior fossa surgery in adult patients; preoperative evaluation, preparation, and premedication; general monitoring considerations; choice of position for surgery; anesthetic considerations, risks, prevention, detection, treatment, and complications of air embolism; and special monitoring issues (Smith DS, 2010).

Preoperative evaluation and preparation

Patient physical status, particularly in reference to cardiovascular and pulmonary stability and airway manageability, is a determinant of the choice of patient position for posterior fossa surgery. The efforts to obtain optimal operating conditions and maintain a stable perioperative course may sometimes be at cross-purposes. For example, patients with previous cerebrospinal fluid shunting procedures may be at greater risk for subdural pneumocephalus with surgery in the head-up position. Thus a thorough evaluation of previous operations and cardiopulmonary problems, current cardiac and respiratory status, evidence of cerebrovascular compromise, and suitability of vascular access for right atrial catheter placement are of particular importance in the patient undergoing posterior fossa surgery (Smith DS, 2010).

In patients with altered limits of cerebral autoregulation, impaired cerebral perfusion, or abnormal baroreceptor function resulting from hypertension, cardiovascular disease, cerebrovascular insufficiency, or prior carotid endarterectomy, the occurrence of hypotension during anesthesia in the head-up position may be especially detrimental. Intravascular volume depletion may result from decreased oral intake, supine diuresis, vomiting, and administration of intravenous contrast agents for diagnostic studies. Incremental administration of intravenous fluids before induction may help limit hypotension during anesthesia induction and positioning. Application of thigh-high compression stockings to the legs limits venous pooling in the lower extremities (Smith DS, 2010).

Assessment of vascular access for right atrial catheter placement helps determine the most promising route. Patients who are obese, have poor vasculature due to disease or chronic intravenous cannulation, or have short, thick necks should

be identified early so that necessary time may be allotted for catheter placement. Some authorities have advocated echocardiography to detect patent foramen ovale (PFO) in patients scheduled for surgery in the head-up position; the use of an alternative position for those who have PFO might reduce the occurrence of paradoxical air embolism (PAE). A detection rate of 10% to 30% with use of echocardiography is comparable with the 20% to 30% incidence reported in autopsy findings. The noninvasive nature of echocardiography makes it attractive for screening purposes; its specificity is reported to be 64% to 100%. However, preoperative screening echocardiography lacks sensitivity (i.e., nondetection of PFO does not guarantee its absence). Transesophageal echocardiography (TEE) is used after induction of anesthesia in some institutions, but it is not 100% sensitive for detection of PFO. More recently Kwapisz and associates described their experience in 35 patients scheduled for posterior fossa surgery in the sitting position. After induction of anesthesia, contrast-enhanced transesophageal echocardiography was performed to check for PFO. Three of the 35 patients were shown to have PFO, and the planned surgical position was altered (Smith DS, 2010).

General monitoring issues

The goals of monitoring are to ensure adequate central nervous system perfusion, maintain cardiorespiratory stability, and detect and treat air embolism. Not every "routine" monitor listed in the box is always used for every posterior fossa procedure. For surgery on the head or neck, many clinicians prefer placement of central venous catheters in the forearm or the antecubital fossa, preferably via the basilic vein after induction of anesthesia. In patients with small veins, a modified Seldinger technique can be used for specialized right atrial catheters or pulmonary

angiography catheters. Prolonged head-down position and head rotation for jugular vein catheter placement should be minimized because these maneuvers may reduce cerebral blood perfusion. A specialized Doppler ultrasound device can be used to localize the jugular or subclavian vein before needle insertion. Whenever catheters are placed via the neck or subclavian routes, the insertion sites should be sealed with bacteriostatic ointment and dressing to minimize air entrainment, especially for patients in head-up positions. Another precaution is to place and remove these central lines while the patient is flat or has the head down, never in the head-up position, because air embolism has been reported in patients in the head-up position (Smith DS, 2010).

Choice of patient position

Surgical access to the posterior fossa can be obtained through various patient positions, such as the sitting position and variants of the horizontal position, which include supine, prone, three-quarter prone, and lateral positions (Smith DS, 2010).

Sitting Position

To establish the sitting position, the patient's skull should be secured in a three-pin head holder; infiltration of the scalp and periosteum at the pin sites reduces the hypertensive response to insertion of pins into the outer table of the skull. The arterial pressure transducer is zeroed at the skull base during positioning and throughout the procedure to make maintenance of adequate cerebral perfusion pressure (CPP) easier. Bony prominences should be well padded, the legs placed in thigh-high compression stockings to limit pooling of blood, elbows supported by pillows or pads to avoid contact with the table or stretch on the brachial plexus, and

the legs freed of pressure at the level of the common peroneal nerve just distal and lateral to the head of the fibula. Efforts to prevent cervical cord stretching and obstruction of venous drainage from the face and tongue include maintenance of at least a 1-inch space between chin and chest, avoidance of large airways and bite blocks in the pharynx, and avoidance of excessive neck rotation, especially in elderly patients. Abdominal compression, lower extremity ischemia, and sciatic nerve injury are prevented by avoidance of excessive flexion of the knees toward the chest. Compression stockings should be applied carefully to avoid a tourniquet effect and ischemic injury to the leg (Smith DS, 2010).

A "lounge chair" modification of the sitting position, with the thoracic cage raised 30 to 45 degrees, may be used for lateral lesions. Access to more midline structures may be impeded by the degree of neck flexion required. Another modification, the lateral sitting position, allows rapid head lowering to the left lateral decubitus position and continuation of the operation in the event of hypotension or persistent venous air embolism (VAE). For the anesthesiologist, advantages of the sitting position include lower airway pressures and ease of diaphragmatic excursion, improved ability for hyperventilation, better access to the endotracheal tube and thorax for monitoring, access to the extremities for monitoring, fluid or blood administration and blood sampling, and visualization of the face for observation of motor responses during cranial nerve stimulation (Smith DS, 2010)..

Improved postoperative cranial nerve function has been reported in patients undergoing acoustic neuroma resection in the sitting position than in those operated on in horizontal positions. Relative contraindications to the sitting position are known intracardiac defects, known pulmonary arteriovenous malformations, severe

hypovolemia or cachexia, severe hydrocephalus, and lesion vascularity (Smith DS, 2010).

Prone Position

The prone position is associated with a lower incidence of VAE. However, the patient's head is usually elevated above the heart to decrease venous bleeding, so the risk of VAE is not eliminated. Access to superior posterior fossa structures and ease of head manipulation are not as favorable as in the sitting position; the sitting position may also offer better operating conditions for high cervical decompression, in which neck flexion and weight-bearing on the head are detrimental. When the patient is in the head-elevated position, placement of the shoulders at or above the edge of the operating table back prevents the face from becoming compressed against the cephalad edge of the table when it is inclined. Eye compression can produce blindness from retinal artery thrombosis; this risk is greater for prone and lateral patient positions, particularly when a padded facial headrest is used. Conjunctival edema is a benign consequence of the prone position that resolves quickly. Visual loss from a variety of mechanisms, usually perioperative ischemic optic neuropathy, is a rare but catastrophic outcome of operative intervention and may be of particular relevance in spinal fusion procedures (Smith DS, 2010).

Venous pooling sufficient to impair venous return can occur in the lower extremities when they lie below the right atrium. Elderly, debilitated patients may not tolerate even a brief discontinuation of monitoring during the turn to the prone position without suffering severe hypotension. In these patients, monitoring cables and transducers should be oriented to allow uninterrupted electrocardiogram (ECG) and arterial blood pressure monitoring throughout the turn to the prone position and positioning adjustments (Smith DS, 2010).

Lateral, Three-Quarter Prone, and Park-Bench Positions

The lateral position is used for unilateral neurosurgical procedures in the upper posterior fossa. The three-quarter prone position, a modification of the prone and lateral positions, and the park-bench position are used for similar procedures to permit greater head rotation and access to more axial structures (Smith DS, 2010).

Risk-Benefit Analysis of Sitting Position Compared with Other Positions

The usefulness or appropriateness of the sitting surgical position for access to the posterior fossa continues to spark debate among neurosurgeons and neuroanesthesiologists, because alternative positions can be used for posterior fossa access and the occurrence of VAE is more common and severe in posterior fossa procedures performed in the sitting position than in alternative positions. Investigators from different institutions have reported their experience with the sitting position, with particular emphasis placed on complications and outcome. Some of the reported complications might have been prevented or reduced if the sitting position had not been used (Smith DS, 2010).

Anesthetic considerations

The clinical significance of theoretic considerations regarding the choice of anesthetic drugs for patients who undergo posterior fossa exploration remains to be determined. First is the question of the effects of inhalational versus intravenous anesthetic drugs on the lungs' ability to retain air that enters the venous circulation, preventing its passage to the arterial circulation. Transpulmonary air passage occurs

in humans and is supported by reports of cerebral air emboli in the absence of an intracardiac defect, as well as detection of left-sided heart air on echocardiogram without demonstration of an intracardiac defect. The intravenous anesthetics pentobarbital, fentanyl, and ketamine maintain a higher threshold for trapping air bubbles in the pulmonary circulation than halothane. Thus such agents may decrease the risk and severity of air emboli if they occur (Smith DS, 2010).

A second consideration is the maintenance of adequate CPP. Before surgical incision, administration of intravenous anesthetic drugs has been demonstrated to have less effect on cardiovascular function than inhalational anesthetics in patients placed in the sitting position. Whether the relationship continues after the start of surgery has not been investigated. A third issue is the potential benefit of preserving cardiovascular responsiveness to surgical manipulation of brainstem structures. In such instances, the avoidance of anticholinergic drugs or long-acting β -adrenergic blockers that would mask cardiovascular response may provide useful information to the surgeon and anesthesiologist (Smith DS, 2010).

An additional consideration surrounds the use of N_2O in cases in which the risk of VAE is increased. A prospective, randomized study of patients requiring posterior fossa exploration or cervical spine surgery demonstrated that 50% N_2O had no significant effect on the incidence or severity of VAE if the N_2O was discontinued when air was detected by Doppler ultrasonography. Its analgesic effect, rapid elimination and emergence characteristics, and facilitation of the postoperative neurologic assessment continue to make it a popular adjunct. However, fentanyl-based anesthesia with supplemental isoflurane has been administered with no difference in time to emergence from anesthesia between patients who received 50% N_2O and those who did not (Smith DS, 2010).

Premedication

Administration of surgical premedication is individualized by patient physical status, evidence of increased intracranial pressure (ICP), and level of patient anxiety. Long-term antihypertensive therapy is continued; perioperative corticosteroids and antibiotics are routinely ordered by the neurosurgeon. Narcotic premedication is avoided in patients with space-occupying lesions or hydrocephalus from fourth ventricle occlusion because the resultant hypoventilation and CO₂ retention may raise ICP. Oral benzodiazepines given 60 to 90 minutes before the patient's arrival in the operating room are effective in reducing anxiety and do not have significant effects on ICP. Often, however, now that most patients come to the hospital on the day of surgery, no premedication is given until arrival in the operating room. Often it is not given at all (Smith DS, 2010).

Induction of Anesthesia

Direct arterial blood pressure monitoring established before induction of anesthesia allows tighter control of blood pressure and CPP during induction and intubation, especially in patients at risk for increased ICP. The use of a low-dose (4 to 6 µg/kg fentanyl), narcotic-based, muscle relaxant technique with 0.5 to 1.0 MAC volatile inhalational anesthetic after intravenous induction with thiopental or propofol affords adequate analgesia and amnesia, preservation of autonomic nervous system activity, and rapid awakening after discontinuation of the inhalational anesthetics; thus an early postoperative neurologic examination is facilitated if desired. Some anesthesiologists continue to use nitrous oxide in oxygen (typically 50%) unless air

embolism occurs, but with desflurane there appears to be little advantage to nitrous oxide. A propofol infusion (50-100 µg/kg/min) often provides better surgical access than inhalational anesthetic alone. β-Adrenergic blocking drugs and direct-acting vasodilators may be used alone or in combination to treat increases in blood pressure. Use of long-acting antihypertensive drugs is avoided until the patient has been placed in the operating position. The need for vasopressor administration may arise after induction of anesthesia or positioning, especially in chronically hypertensive or debilitated patients. Short-acting drugs, such as small boluses of ephedrine or phenylephrine, are usually effective. Rarely, after all correctable derangements such as hypovolemia have been ruled out, inotrope infusions may be required throughout the surgical procedure, but a cause for an underlying mechanism should be sought (Smith DS, 2010).

Verification of appropriate placement of the endotracheal tube after final positioning, but before surgical incision, is of utmost importance, regardless of the position employed. Intraoperative access to the airway is limited by virtue of the proximity of the operative site, and neck flexion or extension can produce caudad or cephalad displacement of the endotracheal tube, respectively, by as much as 2 cm. Palpation of the endotracheal tube cuff above the sternal notch is a useful maneuver to ensure that the tip of the endotracheal tube rises above the carina (Smith DS, 2010)

Maintenance of Anesthesia

Controlled positive-pressure ventilation with paralysis has the following advantages:

- Maintenance of lighter levels of anesthesia
- Hyperventilation, which diminishes $Paco_2$, thereby decreasing both sympathetic

stimulation and blood pressure at any given depth of anesthesia

- Cerebral vasoconstriction
- Less bleeding
- Lower ICP
- Less cardiovascular depression because of decreased anesthetic depth
- Less likelihood of patient movement

The MAC for desflurane (and presumably other anesthetic drugs) is not altered by the sitting position. Excessive decreases in inhaled agent concentration as a strategy to combat hypotension may allow awareness. Intraoperative hypothermia should be avoided. More liberal administration of intravenous fluids may be required during head-elevated prone procedures because of relaxation of the lower extremity capacitance vessels and resultant venous pooling. This pooling may be offset by preoperative application of compression stockings, but some loss of intravascular fluid to the extravascular space will occur over time. If large volumes of fluid are administered during surgery, a small prophylactic dose (5 to 10 mg) of furosemide will promote postoperative diuresis of excess fluids reabsorbed from the extravascular space. Glucose-containing solutions are not used because of the possible detrimental effects of hyperglycemia on areas of the brain at risk for cerebral ischemia (Smith DS, 2010).

The administration of osmotic and loop diuretics for tumor resection and vascular procedures may predispose sitting patients to electrolyte disturbances or cardiovascular instability caused by hypovolemia. Also, the size of the pneumocephalus may be increased. Simultaneous administration of intravenous

colloid is appropriate to maintain CPP and should probably have minimal effect on the cerebral dehydrating action of the diuretic (Smith DS, 2010).

Emergence from Anesthesia

The anesthetic goals during emergence from anesthesia are to prevent abrupt rises in blood pressure, effect rapid awakening, return motor strength, and minimize coughing and straining on the endotracheal tube. The feasibility of immediate postoperative extubation is determined by the nature and extent of surgery (e.g., extensive brainstem manipulation with a greater likelihood of postoperative brainstem edema or brainstem injury caused by a difficult tumor resection).^[44,45] If extensive manipulation of the medullary structures or significant edema is a factor, a secured airway should be maintained until the patient is awake, following commands, and demonstrating return of protective airway reflexes. Additional sedation may be required until this point of recovery is reached. Persistent postoperative hypertension in a previously normotensive patient should alert the anesthesiologist to possible brainstem compression, ischemia, or hematoma (Smith DS, 2010)

Electrophysiologic Monitoring

Various forms of monitoring, such as raw or processed electroencephalogram (EEG), brainstem auditory evoked potentials (BAEPs), and somatosensory and motor nerve stimulation, are being used with increasing frequency to determine the integrity of cerebral function during posterior fossa surgery. Such monitoring is used in selected intracranial, spinal, and cerebrovascular procedures and is generally

handled by experienced electrophysiologists. Bimodal or multimodal measurements of EEG, BAEPs, and SSEPs have been advocated as a more effective means of monitoring central nervous system function for posterior fossa surgery than single-modality monitoring (Smith DS, 2010).

Brainstem Auditory Evoked Potentials

BAEPs are robust signals that are minimally influenced by the type or depth of anesthesia. Cranial nerve VIII monitoring during acoustic neuroma resection or microvascular decompression has been advocated to help preserve nerve VIII function. Bilateral changes in BAEPs are indicative of brainstem compromise. Normalization of BAEPs during emergency posterior fossa decompression has been used to guide postoperative management and timing of extubation (Smith DS, 2010).

Somatosensory Evoked Potentials

SSEPs may be of use in detecting morbidity from cerebral air embolism, spinal cord ischemia caused by hypotension, or stretch of the cord due to excessive neck flexion. Monitoring of short-latency SSEPs, which monitor subcortical components of central sensory pathways, has been advocated for surgery on the cervical cord and posterior fossa. Long-latency components of SSEPs may be difficult to evaluate because of greater variability in both latency and amplitude (Smith Ds, 2010).

Electroencephalogram

EEG signals provide information regarding depth of anesthesia because they are sensitive to both inhalational and intravenous anesthetics. Intraoperative EEG

monitoring during posterior fossa surgery can detect decreased cortical responses resulting from deep anesthesia or ischemia. The information from the cortical components of SSEPs is similar to that from EEG signals (Smith DS, 2010).

Facial Nerve Monitoring

Monitoring of facial nerve (VII) function may help reduce complications of surgical dissection and manipulation for resection of acoustic neuromas and microvascular decompression. Muscle paralysis, which can interfere with the signal, should be significantly reduced or avoided when muscle stimulation is required (Smith DS, 2010).

The blood – brain barrier is also affected by intracranial pathologic conditions. Normally the blood – brain barrier is impermeable to large or polar molecules and variably permeable to ions and small hydrophilic nonelectrolytes. Thus any disruption of the blood – brain barrier permits water, electrolytes, and large hydrophilic molecules to enter perivascular brain tissues, leading to vasogenic brain edema. In this case, leakage – and the resulting brain edema – is directly proportional to the cerebral perfusion pressure (CPP). Vasogenic edema should be differentiated from osmotic edema (caused by a drop in serum osmolality) and cytotoxic edema (secondary to ischemia). Blood osmolality is a critical determinant of cerebral edema because a 19 – mmHg pressure gradient across the blood – brain barrier is generated for every milliosmole. In contrast, oncotic pressure plays a minor role. Neuroimaging shows disruption of the blood – brain barrier in many tumors. New strategies are being investigated to improve drug delivery to brain tumors. In the future, it is possible that new treatments to augment blood – brain permeability

(osmotic blood – brain barrier disruption, intra – arterial chemotherapy) will interfere with perioperative management (Bruder N, Ravussin PA, 2010).

CBF is regulated at the level of the cerebral arteriole. It depends on the pressure gradient across the vessel wall (which in turn is the result of CPP) and PaCO₂ value (which depends on ventilation). CBF autoregulation, dominant to ICP homeostasis, keeps CBF constant in the face of changes in CPP or mean arterial pressure (MAP). It does this through alterations in cerebral vasomotor tone (i.e., cerebrovascular resistance [CVR]). Autoregulation is normally functional for CPP values of 50 – 150 mmHg and is impaired by many intracranial (e.g. blood in CSF, trauma, tumors) and extracranial (e.g., chronic systemic hypertension) pathologic conditions. It is also affected by drugs used in anesthesia (Bruder N, Ravussin PA, 2010).

If CPP is inadequate, tissue perfusion will decrease when the lower limit of autoregulation is less than 50 mmHg (if autoregulation is intact). Ischemia results at levels of CBF below 20 mL/100g/min unless CPP is restored (by increasing MAP or decreasing ICP) or cerebral metabolic demand is reduced (through deepened anesthesia or hypothermia). Increased ICP resulting in reduced CPP is met by cerebral arteriolar relaxation; in parallel, MAP is increased via the systemic autonomic response. As a result, a vicious cycle can be established, particularly in the presence of impaired intracranial homeostasis, as cerebral vessel relaxation increases cerebral blood volume (CBV), thus further raising ICP. In addition, an acute reduction in CPP or MAP tends to acutely increase ICP (the so – called vasodilatory cascade). Reductions in PaCO₂ induce vasoconstriction, reducing CBF, CBV, and thus ICP. Conversely, hypercapnia increases ICP and should be prevented in the perioperative period. This makes hyperventilation is a useful tool for the acute

control of intracerebral hyperemia and elevated ICP. As summarized: The anesthetic goal: haemodynamic stability; The reason: autoregulation takes 30 to 120 second to be established; thus sharp MAP fluctuations entrain undesirable CBF, CBV, and ICP changes; The formulas: $CBF = CPP/CVR$; $CPP = MAP-ICP$; Normally, $ICP < CVP$ (Bruder N, Ravussin PA, 2010).

Seizures are treated with lorazepam, 2 mg i.v.; phenytoin, loading dose 15 mg / kg i.v. over 20 minutes followed by 5 to 7 mg / kg / day, or fosphenytoin, loading dose PE 15 to 20 mg/kg i.v., and then 4 to 6 PE mg/kg/day. The American Heart Association (AHA) recommends that seizure prophylaxis with phenytoin be given for 1 month to all patients after ICH. Euvolemia is maintained with an intravenous infusion of isotonic solution. Hypotonic fluids may exacerbate cerebral edema, and glucose – containing solutions are not used unless patients are hypoglycemic (Manoach S, Charchafliéh JG, 2007).

Multiple trials in patients above 45 years of age have failed to demonstrate benefit from craniotomy and evacuation of an intracerebral hematoma. Indications for operation that have traditionally been accepted or may be inferred from recent trials include cerebellar hematomas $> 3 \text{ cm}^2$ or accompanied by neurologic deterioration, large accessible cortical hematomas ($< 1 \text{ cm}$ from cortical surface), and neurologic deterioration. Younger patients are more likely to benefit from surgery than older patients (Manoach S, Charchafliéh JG, 2007).

Nutritional support and stress – ulcer prophylaxis are provided using H_2 antagonists such as famotidine, 20 mg i.v., every 12 hours, or proton – pump inhibitors such as pantoprazole, 40 mg i.v. daily (Manoach S, Charchafliéh JG, 2007). In this case we give ranitidine as prophylaxis before give induction agent. Anesthesia exerts major effects on intracranial environment through a variety of drug

and nondrug effects. These effects are sensitive to the state of the intracranial and extracranial environment (e.g., cerebral compliance, presence or absence of intracranial pathologic condition, general volemic state) (Bruder N, Ravussin PA, 2010).

Intravenous anesthetics include barbiturates, propofol, etomidate and ketamine. A part from anesthesia induction, propofol is being increasingly used for maintenance as a continuous intravenous infusion (often computer controlled). All the intravenous drugs mentioned are cerebral vasoconstrictors that act by depression of cerebral metabolic rate (CMR), except ketamine. Ketamine increases whole brain CBF without changing CMR in healthy volunteers. At subanesthetic doses, ketamine increases regional glucose metabolic rate and CBF. The other agents decrease CBF, CBV, and ICP while leaving autoregulation and vessel reactivity to PaCO₂ intact. CMR reduction reflects brain activity and is mediated through the electrical but not the basal metabolic activity of the neurons. Hence there is a ceiling effect for CMR reduction at electroencephalogram (EEG) burst suppression. In contrast to volatile anesthetics, propofol has been shown capable of suppressing the cerebrostimulatory effects of nitrous oxide. Etomidate directly inhibits adrenal cortisol secretion for 24 to 48 hours even after a single injection, and its use is often associated with myoclonic (not epileptic) movements (Bruder N, Ravussin PA, 2010).

All volatile anesthetics are cerebral vasodilators, but isoflurane, sevoflurane, and desflurane also reduce CMR. A flat EEG is obtained with this three agents at around 2 minimum alveolar concentrations (2 MAC), a concentration at which maximum metabolic depression is achieved. The response of cerebral metabolism to rising concentrations of volatile anesthetics is not linear. The decrease in CMR is

steep from 0 to 0.5 MAC and then more gradual up to 2 MAC. The effect of volatile anesthetics on CBF is the result of their vasodilatory properties and flow – metabolism coupling. At low concentrations (< 1 MAC), CBF is lower than in the awake person. But CBV is unchanged with isoflurane and decreased with propofol at comparable concentrations. Among the newer volatile anesthetics, sevoflurane is the least vasodilating and desflurane the most (Bruder N, Ravussin PA, 2010). In so that, for this case, we use Sevoflurane as the volatile maintenance anesthesia combine with propofol intravenous titration. The effects of xenon are more complex. This agent decrease CBF in gray matter, particularly in specific brain areas like the thalamus, the cerebellum, the cingulated gyrus, and the hippocampus, and increases CBF in white matter. It does not impair flow-metabolism coupling (Bruder N, Ravussin PA, 2010).

For the normal brain and volatile concentrations below 1 MAC, PaCO₂ reactivity remains intact, permitting control of vasodilation by hypocapnia. However, the presence of a pathologic brain condition or use of a high – MAC volatile anesthetic may impair or even abolish PaCO₂ reactivity and autoregulation (Bruder N, Ravussin PA, 2010).

Nitrous oxide is cerebrostimulatory, increasing CBF, CMR, and sometimes ICP. Its effect is not uniform throughout the brain but is limited to selected brain regions (basal ganglia, thalamus, insula), changing the regional distribution of CBF. If substituted for an equipotent concentration of a volatile anesthetic agent, nitrous oxide increases CBF. For the normal brain, the resulting cerebral vasodilation can be controlled by hypocapnia or the addition of an intravenous anesthetic. However, volatile agents have no such attenuating effect; CMR and CBF are higher during 1 MAC anesthesia produced by a nitrous oxide – volatile anesthetic combination than

that produced only by a volatile anesthetic. This effect is especially deleterious in the actual or potential presence of brain ischemia (Bruder N, Ravussin PA, 2010). In order that, for this case we must be without nitrous oxide application. But because of limited of qualification in anesthesia machine in operating theater was without room air facility, so that we give nitrous oxide in low concentration (Oxygen : Nitrous oxide equivalent was 75% : 25%). Particularly for repeat craniotomy, the potential of nitrous oxide, which is poorly soluble, to diffuse into and hence expand hollow spaces must be remembered as it could cause tension pneumocephalus in patients with intracranial air (repeat neurosurgery or head trauma) (Bruder N, Ravussin PA, 2010).

Opioids have been associated with short – term increases in ICP, particularly sufentanil or alfentanil. Reflex cerebral vasodilation after decreases in MAP and hence in CPP is the underlying mechanism for the transient increases in ICP, although a direct modest cerebral vasodilator effect has been demonstrated. This effect demonstrates the sensitivity of intracerebral drug effects to the intracranial and extracranial environment and the importance of maintaining normovolemia for ICP stability. Generally, opioids modestly reduce CMR and do not affect flow – metabolism coupling, autoregulation, or the carbon dioxide sensitivity of the cerebral vessels.¹³ In this case, we use fentanyl opioid for analgetic during surgery. Ramifentanil has been extensively studied. Its cerebral effects are comparable to those of other opioids, and its use in neuroanesthesia has been validated in clinical trials (Bruder N, Ravussin PA, 2010).

Vasodilating antihypertensive agents such as nitroglycerine, nitroprusside, and nicardipine increase ICP and should be avoided.¹³ In this case, neurologist as the first medical care, was given nicardipine as an antihypertensive drugs for the

patients is suspected hypertensive emergencies with target organs is cerebral haemorrhage. But in perioperative periode, we stopped nicardipine titrated slow down, to keep away from rebound effect. Cerebral vasodilation may result from a normal autoregulation response or direct arterial vasodilation. For axample, sodium nitroprusside does not change CBF. Conversely, verapamil decreases cerebrovascular resistance in humans by inducing direct cerebral vasodilation. Theophylline constricts cerebral vessels but increases CSF production and is a potent central nervous system (CNS) stimulant, raising the risk of convulsions. Most β – adrenergic blockers, especially esmolol, do not interfere with cerebral blood flow or metabolism (Bruder N, Ravussin PA, 2010).

The anesthesiologist posseses a number of instruments to achieve ICP reduction and brain relaxation, and thus to improve the quality of surgical exposure and to reduce retractor pressure. The effectiveness of these instruments depends on intact intracerebral homeostatic mechanisms. Strategies to prevent intracranial hypertension and brain bulging are euvolemia, sedation, analgesia, anxiolysis, no noxious stimulus aplied without sedation and local anesthesia, head – up position, no compression of the jugular veins, head straight, osmotic agents: mannitol, hypertonic saline, β – Blockers or clonidine or lidocaine, steroids, adequate hemodynamics: mean arterial blood pressure, central venous pressure, pulmonary capillary wedge pressure, hear rate, adequate ventilation: $\text{PaO}_2 > 100$ mmHg, PaCO_2 35 mmHg, Intrathoracic pressure as low as possible, hyperventilation on demand before induction, use of intravenous anesthetic agents for induction and maintenance in case of tensed brain (Bruder N, Ravussin PA, 2010). In this case, we kept the patient in euvolemia, by urinary monitoring, adequately sedation and

analgesia with inhalation sevoflurane, intravenous propofol titration, and opioid fentanyl, with slight hyperventilation.

Treatment for managing intracranial hypertension and brain bulging are cerebrospinal fluid drainage if ventricular or lumbar catheter in situ, osmotic agents, hyperventilation, augmentation of anesthesia with intravenous anesthetic agents: propofol, thiopentone, etomidate, muscle relaxants, venous drainage: head up, no positive end – expiratory pressure, reduction of inspiratory time, mild controlled hypertension if autoregulation present (Bruder N, Ravussin PA, 2010). We do in this case with inhalation sevoflurane combined with intravenous propofol titration.

Hyperventilation result in hypocapnia and subsequent cerebral vasoconstriction. In the context of intact autoregulation, CBF is roughly linearly related to PaCO₂ between 20 and 70 mmHg. However, the carbon dioxide reactivity of cerebral vessels may be impaired or abolished in the presence of head injury or other intracerebral pathologic conditions, by high inspired concentrations of volatile anesthetics, or particularly if the vessels are already dilated, by nitrous oxide. The CBF-, CBV-, and ICP- reducing effects of hypocapnia are acute and apparent for less than 24 hours. A typical value to aim for is a PaCO₂ of 30 – 35 mmHg; arterial blood gas analysis rather than end – tidal CO₂ (ETCO₂) should be used as a controlling variable because of the possibility of large arterioalveolar CO₂ gradients in neurosurgical patients. The effectiveness of hyperventilation (PaCO₂ at 25 ± 2 mmHg) for controlling brain bulk in the patient under either isoflurane or propofol anesthesia has been demonstrated (Bruder N, Ravussin PA, 2010).

The main complication associated with hyperventilation is reduction of CBF, which gives to cerebral ischemia. Thus, the anesthesiologist must balance the benefit of brain relaxation against the risk of cerebral hypoperfusion. Other side

effects are linear reduction in coronary artery flow, reduced cardiac venous return, hypokalemia, and potentiation of the brain's response to opioids (Bruder N, Ravussin PA, 2010).

Osmotic diuretics such as mannitol and hyperosmotic saline increase blood osmolality acutely, thus reducing brain water content (mainly in healthy brain tissue with an intact blood – brain barrier) and hence brain bulk and ICP. This response improves brain deformability and thereby facilitates surgical exposure. A further beneficial effect is improvement in blood rheology as a result of the reduction in edema of vascular endothelium and erythrocytes (increasing erythrocyte deformability) – the basis of mannitol's classic "antisludge" effect. A typical regimen is to give 0.5 to 1 g / kg mannitol (150 – 400 mL 20% Mannitol) intravenously, split between a more rapid pre-craniotomy dose and a slower infusion, until brain dissection is complete. The ICP effect is prompt, removes about 90 mL of brain water at peak effect, and lasts for 2 to 3 hours. Normally the aim is to keep osmolality at less than 320 mOsm/kg. Problems with the use of osmotic diuretics include hypernatremia, hypokalemia, and acute hypervolemia, which could be deleterious in patients with congestive heart failure. There is no additional benefit to using loop diuretics such as furosemide, which induced hypovolemia and does not reduce brain water content. On the contrary, serum saline should be infused to replace urinary losses in order to avoid hypovolemia and maintain blood pressure (Bruder N, Ravussin PA, 2010). In this case, we use mannitol 0,5 g/kbw because of cerebral oedema in that patient. Urinary output and fluid balance was the first priority.

Conclusion

The patient undergoing posterior fossa surgery poses challenges to the anesthesiologist in terms of preoperative evaluation, positioning, choice of anesthetic agents, and monitoring, particularly for prevention of air embolism and preservation of neurologic function. The goals of monitoring are maintenance of hemodynamic stability and early detection of air embolism. Active clinical and basic science investigations continue to improve the means by which these challenges may be met in optimal fashion.

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