Anesthesia For Craniotomy

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Anesthesia for craniotomy presents special considerations. The brain is enclosed in a rigid skull and the majority of craniotomies are performed for the treatment of space occupying lesions. At the same time, the brain is a highly vascular organ presenting potential for massive perioperative hemorrhage. Tolerance of the brain to interruption of substrate delivery is minimal.

Anesthetics and physiologic factors controlled by the anesthesiologist have profound effects on the brain. Interactions between anesthesia and surgical outcome can be expected. This article is a practical review of the anesthetic management of patients with intracranial pathology requiring surgery.

Preoperative Evaluation

The initial approach to the patient requiring craniotomy is similar to that of all other patients. There are additional considerations. It is important to obtain an appropriate baseline neurologic evaluation. At emergence from anesthesia, failure to recover baseline neurologic function can be attributed to surgery, anesthesia, or an interaction between the two. It is incumbent on the anesthesiologist to recognize changes from baseline so as to participate in defining further action. It is also important to gain insight into the magnitude and acuity of intracranial hypertension and possible interactions with anesthetic agents. Patients with acute changes in intracranial pressure (ICP) (e.g., epidural hematoma) are more likely to be sensitive to anesthetic effects on ICP. The anesthesiologist also can benefit from appreciating characteristics of the lesion when considering the potential for major hemorrhage.

For aneurysms, it is valuable to know the time interval since subarachnoid hemorrhage. In most centers, clipping/coiling is performed urgently to reduce the risk of spontaneous re-bleeding. Patients receiving nimodipine/nicardipine may exhibit exaggerated hemodynamic responses to volatile anesthetics. For arteriovenous malformations (AVM), history of preoperative neuroradiologic intervention can assist the anesthesiologist in anticipating magnitude of blood loss and potential for perioperative malignant brain swelling. Resection of a radiologically obliterated AVM typically poses little risk for these complications, whereas previously untreated lesions raise concern.

Preoperative treatment with anxiolytics or opioids should be performed with caution and under the direct supervision of the anesthesiologist. Most patients with intracranial tumors have received dexamethasone, often with substantial symptomatic improvement. As a result, compassionate use of these agents is well tolerated. However, patients with acute neurologic decompensation resulting from intracranial hypertension gain little from preoperative medication but assume risk of increased ICP from respiratory depression. More important, spontaneous neurologic deterioration may be masked by use of preoperative medication in the absence of continuous patient observation.

Monitoring

For most craniotomies, monitoring consists of standard monitors in addition to an intra-arterial catheter. The arterial catheter is valuable in providing strict control of blood pressure, particularly during emergence. Central venous pressure (CVP) monitoring is usually not required for management of tumors unless surgery is expected to be exceedingly long or if major hemorrhage is expected (e.g., vascular meningioma, tumor encasement of major vessels). Otherwise, indications for CVP and pulmonary artery pressure monitoring remain the same as for other patient populations dictated principally by cardiac, renal, and pulmonary status.

One exception to this rule is intracranial aneurysm surgery. CVP monitors have considerable value for this procedure. Use of mannitol essentially voids urine output as a monitor of intravascular volume status. The brain receives approximately 20% of cardiac output when the body is at rest. If cardiac output is approximately 5 L/min, it is easy to appreciate that uncontrolled aneurysmal hemorrhage can result in rapid exsanguination. Resuscitation will be aided by monitoring CVP. Another important reason for placement of a central venous catheter is delivery of vasoactive medications. It is difficult to predict whether the

surgeon will request blood pressure to be increased (e.g., during temporary occlusion of the parent vessel) or decreased (e.g., to facilitate clipping or reduce rate of hemorrhage). Delivery of drugs directly into the central circulation provides the fastest possible onset of action and shortens the feedback loop for dose titration facilitating exquisite control of blood pressure within the desired range.

Routine use of intraoperative electrophysiologic monitoring to detect ischemia remains controversial. Although monitoring of evoked potentials make sense, there are numerous reports of false positive and false negative readings. Some advocate use of electroencephalographic (EEG) monitoring for the purpose of pharmacologically inducing burst suppression for neuroprotection. Scientific evidence supporting this practice is weak. Monitoring of cranial nerve function is often used during posterior fossa procedures. Implications for anesthesia largely pertain to limitation in use of muscle relaxants. Although there is no contraindication to neuromuscular blockade during induction and positioning, it is important to assure recovery of neuromuscular function before surgical stimulation of the cranial nerves. Surgery for excision of epileptic foci often requires intraoperative EEG mapping. It is important to appreciate the anticonvulsant effects of the different anesthetics. Most IV and volatile anesthetics suppress EEG activity and may degrade mapping. Conversion to a nitrous narcotic technique before mapping with sufficient time to eliminate agents that suppress EEG activity is commonly practiced. Conversely, small doses of proepileptic agents (e.g., methohexital) may be requested to aid in identification of the focus.

Anesthesia Induction

Concerns unique to induction of anesthesia for craniotomy are related to ICP in the case of mass lesions and prevention of hemorrhage in the case of vascular lesions. The history of effects of anesthetics on ICP began in the 1960s when the earliest measurements were made in patients anesthetized for tumor surgery (1). Major increases in ICP were observed with anesthetic induction. In the subsequent zeal to provide optimal care, it was advocated that any increase in ICP could only be adverse to the patient and thus use of anesthetics known to increase ICP was discouraged. Although logical, this came at some cost. Something must be used to provide anesthesia and those drugs known to reduce ICP (e.g., thiopental, propofol) typically have prolonged durations of action or produce hemodynamic instability. There is little data regarding any relationship between anesthetic effects on ICP and outcome from craniotomy. The few human studies that have been performed have used crude outcome assays preventing a definitive assessment of the relevance of this problem. As a result, the use of various anesthetics during craniotomy has been broadened to allow all facets of a successful anesthetic to be considered (2).

We cannot measure ICP in each patient. As a result, we rely on information derived from limited human data and extrapolate information from animal studies. This seems to work. Case reports in the literature showing a causal relationship between anesthetics and brain herniation on induction are almost nonexistent. The one exception to this is the patient with an occult lesion undergoing surgery for non-neurosurgical procedures. The vast majority of patients anesthetized for craniotomy emerge from anesthesia either with neurologic status unchanged or with changes directly attributable to the site of surgery. As a result, it is difficult to advocate a specific anesthetic for the purpose of induction. We do know that ICP effects of volatile anesthetics can be blunted by simultaneous moderate hyperventilation (3). We also know that high concentrations of volatile anesthetics perturb cerebral autoregulation (4). Cumulatively these concerns must be weighed when inducing anesthesia for craniotomy, particularly in the context of co-existing disease.

For cerebral aneurysms, ICP is of less concern than is prevention of an abrupt increase in mean arterial pressure (MAP) that may cause aneurysmal rupture. In this case, there is evidence that a poorly controlled hemodynamic state during induction is contributory. The mortality rate associated with aneurysmal rupture during induction is substantial (5). The goal is avoidance of hypertension. If an error is to be made it should be towards hypotension. Some advocate purposeful reduction of MAP during induction with vasodilatory agents to prevent hypertensive intubation responses. This is usually unnecessary. Controlled and progressive increases in depth of anesthesia sufficient to blunt responses to intubation are sufficient to prevent hemorrhage. The induction procedure should not be rushed.

Anesthesia Maintenance

Maintenance of anesthesia is usually uncomplicated and generic in many respects. There are two special considerations. In patients with intracranial mass lesions, brain bulk can be a problem, particularly when the dura is being opened. A swollen brain can herniate through the dural defect prohibiting further dural incision. In this circumstance the anesthesiologist is frequently requested to "relax" the brain. There are multiple methods by which this can be achieved. Usually several changes are made simultaneously that cumulatively result in improved operating conditions. The

anesthesiologist can often prevent this problem during patient positioning by assuring the head is sufficiently elevated above the heart to promote venous drainage. Further head elevation intraoperatively can often cause dramatic reduction in brain bulk. This must be weighed against the risk of air embolism in which case transcardiac Doppler monitoring might be considered. This is rarely essential. Placement of the head 10°–15° above the heart is usually sufficient to promote venous drainage without risk of air embolism or hemodynamic instability (6).

Reduction in brain bulk can also be achieved by discontinuation of inhalation anesthetics that are known vasodilators. The first drug to discontinue is nitrous oxide. It is rapidly eliminated and a greater vasodilator than isoflurane because it does not have compensatory reduction in metabolic rate (7). Temporary discontinuation of the volatile anesthetic may also be of benefit.

There is no evidence that opioids increase brain bulk. There is evidence that opioids increase ICP (8). This effect is modest and transient. Human study has provided convincing evidence that opioids increase ICP as a result of effects on MAP (9). When autoregulation is intact, reduced MAP causes vasodilation resulting in increased cerebral blood volume (CBV) and ICP. Opioid induced increases in ICP can largely be avoided simply by controlling MAP during opioid administration. As a result, opioids are unlikely to be an issue during maintenance with respect to brain bulk.

Mannitol reduces brain bulk by creating an osmotic gradient across the blood brain barrier causing water to flux from the extracellular extravascular to intravascular compartments. Mannitol also improves deformability of red blood cells, thereby reducing viscosity promoting increased blood flow. As a result, autoregulation causes vasoconstriction reducing CBV. Mannitol is best given around the time of skin incision (typically 0.5 g/kg) so that benefit occurs coincident with dural opening. Additional mannitol may be of value if the brain is still "tight."

If hypercapnia is present it should be corrected. The response of the cerebral vasculature to changes in $Paco_2$ is rapid. There has been a major shift in attitude regarding the value of hypocapnia with current opinion being that major reduction in $Paco_2$ poses risk of secondary ischemic injury. Stabilizing the $Paco_2$ in the range of 30–35 mm Hg is usually adequate to offset the vasodilatory effects of volatile anesthetics.

If the above actions are not successful, benefit may be obtained from thiopental. Moderate doses cause major reduction in metabolic rate and a coupled reduction in CBF. This can be effective but will likely prohibit adequate neurologic evaluation on emergence and may commit the patient to postoperative mechanical ventilation. Consultation should be made with the surgeon before taking this step.

Lumbar cerebrospinal fluid (CSF) drains are often placed for aneurysm surgery. The CSF volume drained often exceeds 100 mL, making this technique perhaps the most effective of all options in reducing brain bulk. The goal in aneurysm surgery, however, is different from that of tumor surgery. In most aneurysm cases, reduction in brain bulk is performed to reduce the magnitude of retractor pressure required to expose the aneurysm at the base of the brain. CSF drainage is usually not used until after the dura is opened. This is because rapid and profound reduction in brain bulk can tear veins draining into sinuses. An acute subdural hematoma can be formed if the drain is opened before surgical exposure of the brain with little hope of prompt hemostasis. A practical approach to this is to ensure patency of the drain after positioning by observing progression of the air/fluid level through the connected catheter (usually <1 mL of CSF drainage is required to confirm this) and then leave the drain closed until surgical requirements dictate that it be opened. CSF drains are rarely used for most types of tumor surgery because of fear of transtentorial herniation. Occasionally CSF drains are placed for transphenoidal pituitary surgery, not to drain CSF, but to allow injection of saline or air to force the tumor towards the sphenoid sinus to facilitate surgical excision.

Management of Ventilation

The classic reflex when confronting a patient with intracranial hypertension is the use of hyperventilation. This is derived from knowledge that alteration of $Paco_2$, within the range of approximately 20–80 mm Hg, causes parallel changes in CBF. CBF, in fact, is only a surrogate for the true determinant of ICP, that being CBV. CBF is easy to measure, whereas CBV is not (particularly in humans). It is logical, however, that when given a constant MAP, $Paco_2$ induced changes in CBF should correlate with cross-sectional diameter of the cerebral arterial vasculature. Decreasing $Paco_2$ results in decreased CBF and it is presumed that this causes decreased CBV. Indeed, there is abundant clinical evidence in patients with ICP monitors in place, that reduction of $Paco_2$ results in at least transient reduction in ICP. Neuroanesthetic practice, therefore, had been to cause large reductions in $Paco_2$. Data from head injury patients has caused a major change in this perspective. Use of retrograde jugular venous hemoglobin-oxygen saturation measurement techniques has repeatedly shown that reduction in $Paco_2$, in fact, can exacerbate cerebral hypoperfusion (10). This makes common sense. If the problem with intracranial hypertension is decreased bloodflow, it

does not seem logical to treat the disorder with vasoconstriction. As a result, major reductions in $Paco_2$ are no longer advocated in patients with intracranial hypertension. Modest reductions in $Paco_2$ remain valuable to counteract vasodilatory effects of volatile anesthetics.

It is also important to recognize the value of end-tidal CO_2 monitoring during craniotomy. This advance has reduced the need for repeated arterial blood gas sampling. However, analysis of arterial to end-tidal CO_2 gradients in neurosurgical patients has shown that the gradient is not predictable and should be measured for the individual patient when management of intracranial hypoperfusion is a concern.

Muscle Relaxants

Several muscle relaxants have received special consideration in the context of craniotomy. The most interesting is succinylcholine. There is clear evidence from both experimental animals and humans that succinylcholine can increase ICP under conditions of intracranial hypertension. The magnitude of increase is typically small and transient. The mechanism was originally thought to be attributable, not to succinylcholine, but to preservatives used in its formulation. This argument has been dispelled. It has been shown in humans that ICP changes caused by succinylcholine can be blocked by preadministration of a defasciculating dose of a nondepolarizing relaxant (11). This suggests that fasciculations resulting from succinylcholine play a role in the ICP effects of this drug. Animal evidence supports this. A probable mechanism is the massive fasciculation-induced afferent barrage from muscle spindles to the brain that cause transient increases in metabolic rate and coupled increases in CBF (12). Common sense plays a major role in the decision whether to use succinylcholine in patients with intracranial hypertension. Pretreatment with a small dose of a nondepolarizing agent most likely makes the argument moot. At the same time, emergency airway management in the likely context of a full stomach and the clear desire to minimize hypercapnia and hypoxemia in patients with traumatic brain injury (TBI) dictate that succinylcholine is an appropriate adjunct for tracheal intubation until a relaxant with similar speed of onset and duration of action is introduced to clinical practice.

There is clear evidence that the duration of action of nondepolarizing muscle relaxants is reduced by a variety of anticonvulsant medications (13). Even short durations of exposure to anticonvulsants can elicit this change. The mechanism remains unclear. Most patients requiring craniotomy are being treated with anticonvulsants. As a result, the non-depolarizing relaxant dosing regimen most likely will require alteration. Atracurium and cis-atracurium seem to be largely resistant to these effects, most likely because metabolism is achieved by Hoffman elimination.

Posterior Fossa Considerations

Because of the primacy of the brainstem in maintaining vital function, posterior fossa procedures present special concerns. This is derived from three factors. First, the volume of the infratentorial compartment is small. Thus smaller volumes of hematoma formation may be sufficient to compromise neural function. Further, brainstem edema developing after conclusion of surgery, may insidiously impair vital function. For this reason, it is appropriate to consult with the surgeon before extubation to determine if sufficient brainstem manipulation occurred to warrant overnight tracheal intubation. In this circumstance, a transient emergence from anesthesia in the operating room is performed to allow definition of postoperative baseline motor function before beginning sedation to allow intubation to be tolerated. Second, retraction on the brainstem can cause ischemic loss of function of nuclei that regulate hemodynamics and ventilation. In earlier days of neurosurgery, infratentorial procedures were often performed with spontaneous ventilation to continuously monitor function of respiratory drive centers. This has largely been abandoned in favor of mechanical ventilation allowing control of brain bulk. A surrogate marker is heart rate. Precipitous decreases in heart rate are considered to be a signal of brainstem ischemia. This requires prompt notification of the surgeon. Most often, this spontaneously clears with adjustment of retractor placement. Occasionally resuscitation with atropine is required. Third, cranial nerve function may be compromised. This is especially critical for the ninth and tenth nerves that regulate gag reflex and laryngeal function. Again, consultation with the surgeon is helpful in defining the potential for cranial nerve dysfunction. In such cases, it is appropriate to plan emergence such that integrity of the gag reflex can be tested before extubation. Absence of the gag reflex provides sufficient reason for continued intubation to prevent aspiration. In such cases, major sedation is usually unnecessary simply because the patient does not perceive the stimulation normally caused by the tube.

Management of Temperature

There has been abundant enthusiasm over the past decade for routine use of intraoperative hypothermia in patients requiring brain surgery. This is based on overwhelming laboratory evidence that reduction of body temperature by even 2°–3°C can cause major neuroprotection (14). Until recently, there was no evidence of mild hypothermia efficacy in humans. An

appropriately powered trial in patients with TBI failed to demonstrate benefit and in fact identified worsened outcomes in the elderly (15). In contrast, recent data has definitively shown efficacy of mild hypothermia for brain resuscitation in selected individuals remaining comatose after restoration of spontaneous circulation after out-of-hospital cardiac arrest (16). Although this data supports the concept that intraoperative mild hypothermia might be protective, it does not offer direct proof. Held in the context of increasing evidence that intraoperative hypothermia increases morbidity in the general surgical population, routine use of intraoperative hypothermia for craniotomy cannot currently be advocated. We will soon have efficacy data specific to the neurosurgical population. The Intraoperative Hypothermia Aneurysm Surgery trial is currently being conducted with 1000 patients being randomized to normothermia (36.0°–37.0°C) or hypothermia (32.5°–33.5°C) during intracranial aneurysm surgery (17). The results of this study will likely be available in late 2003.

In contrast, there is near universal agreement that hyperthermia should be avoided. Not only is there absence of theoretical benefit, but also substantial circumstantial evidence in humans that hyperthermia increases brain damage. Most studies supporting this contention are correlative and the data has not been obtained from a randomized trial. Such a trial is unlikely to be performed. It is also clear that hyperthermia is a frequent sequel to brain injury (18). Patients suffering from subarachnoid hemorrhage, TBI, or cardiac arrest are likely to have multiple spontaneous episodes of hyperthermia during acute convalescence. Vigilance and prompt treatment of hyperthermia is recommended.

Pharmacologic Neuroprotection

A Holy Grail of academic neuroanesthesia has been a definition of neuroprotective efficacy from anesthetics and other purported neuroprotective compounds. The simple interpretation of four decades of research is that we still lack definitive proof (particularly in humans) that pharmacologic neuroprotection is a reality. Because most anesthetics reduce brain metabolic requirements, it certainly makes sense that in the context of diminished substrate supply anesthetics will increase tolerance to ischemia. However, the issue is now recognized to be far more complex than this logic describes. More important, other than a few studies examining hypnotics in cardiac surgical patients, there are no human studies that have prospectively examined presence or absence of neuroprotection from anesthetics.

Laboratory studies have irrefutably shown that anesthetics increase tolerance of brain to ischemia in rodents. This is independent of the type of ischemia (global versus focal). The mechanisms are likely related to inhibition of glutamatergic (excitatory) neurotransmission and potentiation of GABAergic (inhibitory) neurotransmission. Many anesthetics meet these criteria (i.e., volatile agents, propofol, barbiturates). Other compounds such as nitrous oxide and opioids appear to be inert. It is clear that animals sustaining stroke while awake develop larger lesions than if anesthetized. However, the question of which anesthetic is superior remains controversial. Many clinicians persist in the use of thiopental as a first line agent. The logic for this is that thiopental has the longest track record of experimental efficacy and there is one human cardiac surgical trial that found benefit (albeit small). It is difficult, therefore, to recommend one agent as being superior to the others. There is one exception to this conclusion and that is etomidate. Although outcome evidence is not available, human studies have provided reasonable evidence that etomidate may exacerbate injury (19).

Another problem is that if one selects an anesthetic to provide intraoperative neuroprotection the maximally efficacious dose remains undefined. Most studies have compared different anesthetics without completion of dose-response curves for the respective anesthetics. Many would say that, for barbiturates, maximal effect is obtained coincident with the dose required to provide EEG burst suppression. This is an anachronistic practice based on theoretical information. Recent studies in rodents indicate that substantially lower doses of barbiturates provide a similar magnitude of protection (20).

As a result of incomplete science regarding anesthetic neuroprotective efficacy in humans, and in the absence of any other drugs approved for the purpose of neuroprotection in humans, the anesthesiologist is left with a speculative practice when providing pharmacologic neuroprotection. Perhaps the best we can do for the patient, with certainty, is to provide oxygenated blood at a sufficient perfusion pressure with simultaneous control of temperature and glucose concentration.

Management of Emergence

Planning for emergence begins with anesthetic induction. The goals of emergence are a predictable recovery to allow testing of motor function in the context of controlled hemodynamics and airway. A unique concern is that failure to emerge may be attributable to either anesthesia or surgery. The treatment is highly dependent on the etiology. If failure to emerge is attributable to surgery, a computerized tomographic scan is usually performed to rule out hematoma formation. In contrast, if the problem is anesthesia based,

patience in allowing elimination of anesthetic agents (or use of opioid antagonists or reversal of neuromuscular blockade) is the solution and the surgeon should be counseled that that anesthesia is a likely explanation. Therefore, when planning the anesthetic, it is helpful to restrict use of agents to those that can be monitored for end-tidal concentration or those for which sufficient knowledge of pharmacokinetics allows highly predictable clearance by the time emergence is desired. As an example, induction doses of thiopental or propofol are unlikely to relate to failure to emerge from a 3–4 h procedure. In contrast, persistent blood propofol concentrations sufficient to prevent awakening after a prolonged infusion may be difficult to diagnose. It is best to keep the anesthetic simple so that each compound can be independently ruled out as an etiology for failure to emerge.

Emergence from anesthesia for craniotomy presents unique management concerns. It should be remembered that craniotomy is generally well tolerated in awake patients. Thus, during most phases of surgery, the magnitude of surgical stimulation is minimal. As a result, one of the strongest stimuli is application of the head dressing, which causes motion of the endotracheal tube in the trachea. This combined with lightening of anesthesia for the anticipated emergence can result in loss of hemodynamic control and difficulty in airway management, particularly if neuromuscular blockade is insufficiently reversed to allow extubation. A practical approach to this event is to assume that the anesthesiologist has 5–10 min after completion of the head dressing to allow a controlled emergence. Thus, neuromuscular blockade is maintained until completion of the head dressing. Elimination of volatile anesthetics can be initiated at the time of bone flap replacement. Anesthesia is maintained by either residual concentration of opioid (i.e., fentanyl or sufentanil) or continued infusion of remifentanil. Supplementation with nitrous oxide is likely superior to the use of IV agents because its concentration can be defined by end-tidal gas analysis, which aids in defining failure to emerge. Rapidly cleared IV agents such as lidocaine can be of value in sustaining anesthesia for a few additional minutes. If remifentanil is used, the rate of infusion can remain unchanged until the dressing is complete (21). This supports anesthesia during placement of the dressing but still allows a prompt and reliable emergence. It is important, however, to provide transitional analgesia before discontinuation of remifentanil. Administration of 10 mg morphine or 100–150 μg fentanyl (in adults) is usually sufficient to provide analgesia without altering predictability of emergence.

For reasons not yet understood, patients undergoing craniotomy frequently exhibit hypertension during emergence that is sustained through the early phases of recovery. Because of the risk of intracranial hemorrhage, it is imperative to plan for treatment of hypertension before it becomes manifest. Prophylactic doses of labetolol are helpful, usually requiring 40–60 mg to be effective. It has not been proven that emergence hypertension contributes to hematoma formation. It has been shown that many patients who develop postoperative hematomas have had episodes of hypertension during emergence or early recovery (22). The brain, however, has been shown to be hyperemic in the early postoperative period independent of MAP (23) leaving the mechanism of hemorrhage unclear. The source of hemorrhage is almost always within the surgical field and thus quality of hemostasis undoubtedly is important. Because the mortality associated with postoperative hematoma formation requiring emergent evacuation is high, it seems incumbent on the anesthesiologist to aggressively manage hemodynamics during emergence.

Postoperative nausea and vomiting (PONV) is a frequent and persistent problem after craniotomy (24). Several studies have shown that greater than 50% of patients suffer this complication. The incidence of PONV appears to be independent of whether the craniotomy was performed awake or under anesthesia, and independent of opioid dose. This suggests that brain surgery itself is contributory. Females, younger patients, and those undergoing infratentorial craniotomy are at greater risk. PONV is not only an early emergence problem but can be sustained for hours or days after surgery. Ample evidence is now available that prophylactic antiemetic therapy administered shortly before emergence markedly reduces the magnitude of this problem (25). A single dose is likely to be transiently beneficial, however, and will require repeated supplementation if PONV is to continue to be suppressed.

Emergency Procedures

It is important to know if the patient is dying of intracranial hypertension. This is particularly true of expanding hematomas. These cases are unquestionable surgical emergencies. The cure is surgical and the most important thing the anesthesiologist can do is make the patient ready for incision as rapidly as possible. Regardless of academic discussion on the importance of anesthetic effects on ICP in scheduled craniotomies, most agree that all effort should be made to minimize increases in ICP in patients requiring emergency craniotomy. Patients who are herniating or near herniating are not likely to be conscious. Prevention of recall is of minor importance relative to maintenance of hemodynamic stability. Effort should be made to secure the airway as rapidly as possible and turn the patient over to the surgeon. Small doses of barbiturate (so as to not cause hypotension) to reduce metabolic

rate and coupled blood flow or opioid may be appropriate until surgical decompression is achieved. Profound hyperventilation should be avoided. Inhalational anesthetics have little or no role in the early stage of these procedures. Enhanced venous and arterial access can be made by the anesthesia team simultaneous with onset of surgery.

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