Section 1 Neuroanesthesia

Part I
Case
1

Craniotomy. Supratentorial craniotomy

Preoperative evaluation

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Supratentorial craniotomy is a common neurosurgical procedure, which may be emergent (e.g., evacuation of an expanding hematoma) or routine (e.g., scheduled resection of a mass lesion). Although the core preoperative evaluation is similar to that of the non-neurosurgical population, there are special considerations for patients with neurologic disease.

Case description

The patient was a 61-year-old female who presented with new-onset seizures, which were preceded by several weeks of bilateral frontal headaches described as "dull and achy." The headaches had become nearly continuous and were more recently accompanied by vertigo and nausea. The morning of admission, she had a witnessed generalized seizure. The seizure terminated and she presented to the emergency room where she was treated with benzodiazepines and supportive care; reversible physiologic and pharmacologic derangements were ruled out. Computed tomography (CT) and magnetic resonance imaging (MRI) were performed, which revealed a large left temporal fossa mass suggestive of a meningioma. She was admitted to the neurosurgical intensive care unit, where dexamethasone and a loading dose of phenytoin were administered. The patient was scheduled for tumor excision via stealth-guided craniotomy.

Prior to the headaches and seizure, the patient was otherwise healthy except for a history of asthma. She used an albuterol inhaler several times each week and had been taking ibuprofen for her headaches. On the morning of surgery, the patient was visibly anxious, but was alert and oriented to person, place, and time. Her cranial nerve examination was normal, pupils were equal and reactive to light, she demonstrated normal strength bilaterally and showed no pronator drift. Heart sounds were normal, but wheezing was appreciated on auscultation. Her blood pressure during the examination was 175/92, considerably elevated from her usual baseline. Electrocardiogram and laboratory values were normal, including a therapeutic phenytoin level.

The patient's anxiety, increased blood pressure, and bronchospasm were addressed immediately. The anesthesiologist had a reassuring conversation with the patient and midazolam was administered, followed by an albuterol breathing treatment. Oxygen was delivered through a nasal cannula and she was monitored continuously with a blood pressure cuff, pulse oximeter, and by direct visualization to assure she did not experience somnolence or respiratory depression due to the benzodiazepine.

Anesthetic concerns for this patient included: (1) vigilance for any signs or symptoms of increased intracranial pressure (ICP), (2) keeping the patient as comfortable as possible during an emotionally difficult preoperative period, (3) history of asthma, (4) recent history of seizures and consequent phenytoin use, (5) maintaining proper fluid status during a procedure that could include fluid shifts both planned (diuretics) and unplanned (bleeding), and (6), the need for a timely, hemodynamically stable, and thorough awakening.

Once in the operating room induction of anesthesia was initiated with fentanyl, sodium pentothal and vecuronium; great care was taken to maintain the patient's blood pressure within 20% of baseline. The trachea was intubated with a 7.5 mm LITA (Laryngotracheal Instillation of Topical Anesthesia) endotracheal tube. An arterial catheter and two large-bore intravenous catheters were placed. General anesthesia was maintained with isoflurane and a total of approximately 10 mcg/kg of fentanyl was administered throughout the case for analgesia. The surgeons reported swelling of the brain and so the patient was hyperventilated to a $PaCO_2$ of 30 mmHg. Mannitol was administered to reduce brain volume and dexamethasone was given for the prevention of cerebral edema. There was an estimated blood loss of 1300 mL,

and the patient was resuscitated with 2 liters of crystalloid, 500 mL of 5% albumin and one unit packed red blood cells. As surgical closure began, isoflurane was discontinued and infusions of propofol and remifentanil were initiated. After the surgical drapes were removed, the LITA tube was dosed with 4% lidocaine and albuterol was delivered via the endotracheal tube. The patient emerged within several minutes of the head dressing being applied, without bucking or coughing. She was neurologically intact.

Discussion

As with other complex cases, the patient presenting for supratentorial craniotomy is best served by an organized and systematic approach. Below is one strategy for the preoperative evaluation of these neurosurgical patients.

1. Stratify the case

It is first important to distinguish between the "elective" or scheduled resection of a supratentorial mass lesion and the patient presenting for emergent decompression due to intracranial hypertension. The former situation allows time for a full evaluation process [1] while the latter demands immediate attention to the "ABCs" of airway, breathing, and circulation, as well as management of increased intracranial pressure (ICP; discussed in the next chapter) [2].

2. Assess the patient

Even during the introductory process, the anesthesiologist should be assessing the patient's mental status and gross neurologic function. Are they alert or somnolent? Do they have any obvious facial asymmetry? Do they respond appropriately and with intact language skills? If the patient is interactive, obtain any history possible. If not, assess for ABCs.

Next, assess the vital signs – significant hypertension and bradycardia could be a Cushing's reflex revealing poor intracranial elastance. The physical examination that follows should focus on neurologic status, including a cranial nerve examination, assessment of the pupils and their reflexes, and motor strength. It is imperative to establish a preoperative neurologic baseline to which the postoperative status can be compared.

3. Review the chart

For a non-emergent case, there is often valuable information that can be derived from the surgical evaluation and that can help guide the preoperative anesthetic assessment. Any available neuroimaging studies should also be reviewed, with particular attention to the size and location of the lesion as well as signs of increased ICP such as midline shift, effacement of sulci and gyri, loss of gray–white differentiation, and herniation. Laboratory values should also be reviewed with particular attention to hematocrit, coagulation studies (especially in patients with hemorrhage or hematoma), sodium irregularities (common in neurosurgical patients), and glucose (which, when high, can exacerbate neural injury) [3]. Cardiac studies such as electrocardiogram or echocardiogram can provide important information, especially if the patient has suffered a stroke [3]. Review all medications, especially any anticonvulsants that could affect drug metabolism.

4. Develop a plan

Based on (1) your stratification of the case, and (2) your assessment of the patient, develop an anesthetic plan that addresses any medical issues that might be manifested in the perioperative course of a craniotomy.

Preparation: In general, an arterial catheter and large-bore intravenous lines are recommended for these cases, but especially in those involving vascular lesions and tumors with high potential for hemorrhage (such as meningiomas). Invasive blood pressure monitoring allows beat-to-beat readings and the ability to regularly assess pH status and $PaCO_2$ levels with arterial blood gases. Depending on the co-morbidities and venous access, a central line may be required. The subclavian approach is more appropriate for the neurosurgical patient, as an internal jugular catheter can potentially compromise venous return from the brain and thus increase ICP.

Ensure that commonly used agents such as mannitol, dexamethasone, and furosemide are available. Discuss anticonvulsant administration with the neurosurgical team. Phenytoin is often administered, but fosphenytoin is preferable because of decreased cardiovascular side effects. In either case, the drug should be administered slowly, as the goal is prophylaxis rather than acute termination of seizures. An anti-emetic strategy should also be established [4].

Premedication: Neurologic disease and neurosurgical intervention is a highly stressful event for

the patient and the family. The need for compassion and reassurance cannot be overemphasized. Pharmacologic support can be attained using any number of drugs – it must be noted, however, that any sedative can cause respiratory depression. The patient must be monitored continuously, since a decrease in ventilation can cause an increase in CO_2 and therefore an increase in ICP via cerebral vasodilation. Sedation may also unmask or exacerbate focal neurologic deficits [5].

Anesthetic regimen: The choice of anesthetics depends on the state of intracranial elastance. Induction with propofol or sodium pentothal is appropriate, with attention to cardiovascular status. Ketamine is typically avoided, as it increases cerebral metabolic rate. In terms of maintenance, inhalational anesthetics may be used if the patient has normal intracranial elastance [6]. If the patient has compromised elastance and intracranial hypertension, then these anesthetics should be avoided. Although inhalational anesthetics reduce cerebral metabolic rate, they dilate cerebral blood vessels (nitrous > desflurane > isoflurane > sevoflurane), which increases cerebral blood volume and thus ICP. Inhalational agents can also disrupt cerebral autoregulation, which normally maintains constant cerebral blood flow (50 mL/100 g tissue/min) in the face of changing mean arterial pressures. The classically taught range of autoregulation is a mean arterial pressure of 50–150 mmHg, but the lower limit may be as high as 70 mmHg. Chronic hypertension can shift the autoregulatory curve to the right, which means that higher pressures are needed to stay in the autoregulatory range. The intravenous agents propofol and pentothal decrease cerebral metabolic rate but do not dilate cerebral blood vessels – their use is therefore recommended in patients with poor intracranial elastance and they also have minimal effects on autoregulation.

Analgesia: Appropriate pain control is imperative for patient comfort and a smooth emergence [7]. Preoperative scalp block may help reduce intraoperative opioid requirements and postoperative pain (see Case 5). Major stimulating events include intubation, cranial pinning, incision, removal of bone flap, and durotomy. An early focus on analgesia during the case is important such that opioid-associated sedation and hypoventilation are not a problem during emergence.

Emergence: All physiologic parameters should be optimized prior to emergence. There are two main strategies for achieving an emergence that occurs directly after undraping and does so without the patient undergoing hemodynamic responses that might increase the chance of intracranial hemorrhage. The first is by using, or least switching to, short-acting anesthetics and analgesics. For example, isoflurane can be turned off 1–1.5 hours prior to wake-up and replaced with remifentanil, propofol, or both. With this strategy, less remifentanil or propofol is required to achieve general anesthesia since the offset of isoflurane anesthesia is relatively slow and the stimulus of closing is minor. Remifentanil, like most narcotics, tends to soothe the airway and is especially helpful. Another method of facilitating a smooth wake-up is to decrease the stimulus of the endotracheal tube, which can be accomplished with the use of a LITA tube. The LITA allows the easy administration of lidocaine both above and below the cuff, anesthetizing the airway and thereby minimizing the irritation during wake-up. If a LITA is not available, one can place lidocaine down the endotracheal tube or into the posterior pharynx. This strategy may be especially important in patients with a reactive airway, as the patient described above. Careful planning to minimize physiologic and pharmacologic confounds in the immediate postoperative period is essential for an effective neurosurgical evaluation [8].

Conclusion

In conclusion, supratentorial craniotomy is a common case in neurosurgery during which both the neurosurgeon and anesthesiologist are modulating the same organ. Thoughtful planning and clear communication between the teams is required for optimal patient care.

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Part I
Case
2

Craniotomy. Supratentorial craniotomy

Evaluation and anesthetic management of elevated intracranial pressure

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Understanding the appropriate interventions to manage increased intracranial pressure (ICP) is an essential skill of a neuroanesthesiologist. Intracranial pressure is of paramount importance because the cranial vault is nondistensible and within it are contained three noncompressible substances: brain, blood, and cerebrospinal fluid (CSF).

Case description

The patient was a 75-year-old male who presented for emergent subdural hematoma evacuation. During a witnessed slip and fall accident with no associated loss of consciousness, the patient suffered a left temporal contusion. Shortly after the accident he became confused and lethargic. Noncontrast head computed tomography (CT) revealed an acute right temporal subdural hematoma with 5 mm of horizontal shift. Past medical history was significant for hypertension and atrial fibrillation; current medications included coumadin, metoprolol, and lisinopril. Neurologically the patient was oriented to person only and moved all four extremities equally with full strength. No cranial nerve deficits were appreciated. The patient's blood pressure upon presentation was 155/85.

The immediate anesthetic goal was to minimize the rise in ICP while at the same time maintaining adequate cerebral perfusion pressure until the neurosurgeons could provide definitive treatment. In order to better monitor arterial pressure, a radial artery catheter was placed. Laboratory tests including type and screen, coagulation values, and complete blood count were drawn. After placement of standard monitoring and preoxygenation, general anesthesia was induced with propofol and care was taken to prevent precipitous increases in blood pressure during both laryngoscopy and placement of the head in a Mayfield frame. The patient was then positioned supine with the head of the bed elevated 30 degrees. A midline head alignment and unobstructed cervical venous drainage were ensured. Based on the patient's baseline blood pressure and estimated ICP, a mean arterial pressure (MAP) >80 mmHg was maintained in order to maintain cerebral perfusion pressure (CPP) >60 mmHg. Prior to the dura being opened, mannitol and furosemide were administered to decrease parenchymal volume. Definitive correction of intracranial hypertension was achieved with hematoma evacuation.

Discussion

The cranium is a nonexpandable bony structure with a fixed volume and therefore with only limited means of compensating for increased ICP. If pressure within that nonexpandable structure is allowed to rise the result is life-threatening neural injury. Normal ICP in the adult is <20 mmHg with a high degree of normal physiologic variability. A sustained ICP >20 mmHg should be considered pathologic and treatment should be initiated. Intracranial pressure along with MAP determine cerebral perfusion based on the equation CPP = MAP – ICP. Therefore, when managing patients with elevated ICP, adequate MAP must be achieved in order to maintain CPP >60 mm Hg. Below this threshold cerebral ischemia can occur.

The determinants of ICP are the contents of the cranium and include brain parenchyma (neurons, glia), blood (arterial and venous circulation), and fluid (interstitial and CSF). Increases in one of these volumes must be offset by decreases in the other constituent volumes or ICP will increase [1]. The magnitude of change in ICP is directly related to the intracranial elastance. As the volume of blood, fluid, or brain expands, physiologic mechanisms of compensation such as displacement of CSF out of the cranium or vasoconstriction result in relatively small increases in ICP. As intracranial elastance decreases, these mechanisms are eventually outstripped and further small increases in volume result in large increases in ICP

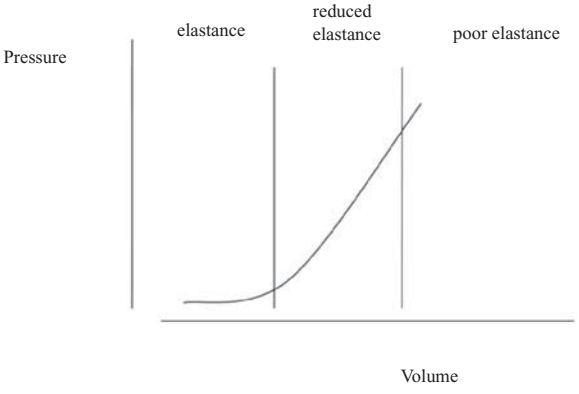

Figure 2.1. Intracranial elastance curve.

(Figure 2.1) [1]. A direct relationship exists between cerebral blood flow (CBF) and ICP, such that elevated ICP can be modified by decreases in CBF so long as cerebral perfusion is not compromised. If insufficient CBF results in brain ischemia, the associated cerebral edema will increase ICP, further compromising CBF and setting in motion a dangerous downward spiral.

Increased ICP is associated with many pathophysiologic states, each with the common mechanism of increasing the intracranial volume of CSF, blood, or brain tissue (Table 2.1). Increased tissue volume is caused by space-occupying lesions such as tumors, either primary or metastatic. Cerebral malignancies also increase ICP by obstructing absorption and outflow of CSF and producing cerebral edema. Cerebral edema is classified as cytogenic or vasogenic in origin. Cytogenic cerebral edema develops as the result of inadequate intracellular energy supplies resulting in failure of membrane integrity, increased cellular swelling and ultimately cell death. Vasogenic cerebral edema by comparison is the result of increased vascular permeability and the movement of protein-rich fluid into the cerebral interstitial space.

Increased intracranial blood volume elevates ICP and can present in varied forms. Subarachnoid hemorrhage is frequently caused by head trauma, disruption of aneurysms, and arteriovenous malformations. Blood in the subarachnoid space or in the ventricles impedes CSF outflow and results in ICP elevation. Intracranial hematomas, blood-filled space-occupying lesions, are classified by their location relative to the meningeal layers. Subdural hematomas are more frequently seen in elderly patients due to natural cerebral atrophy and the resulting fragility of cerebral bridging veins. Acute epidural hematomas are frequently the result of severe head trauma and produce a characteristic lenticular-shaped hemorrhage on CT scan.

Table 2.1. Common causes of elevated intracranial pressure.

Substrate	Pathophysiology
Parenchyma	Malignancy Primary Metastatic Cerebral edema Cytogenic Vasogenic Eclampsia Fulminant hepatic failure
Blood	Subarachnoid hemorrhage Intraparenchymal hemorrhage Subdural hematoma Epidural hematoma Vasodilatation Venous outflow obstruction
Cerebrospinal fluid	Hydrocephalus CSF outflow obstruction

Mental status can range from drowsiness to coma depending on ICP and the extent of brain tissue displacement. As ICP increases, signs and symptoms include headache, nausea, vomiting, and papilledema. Bradycardia, hypertension with widened pulse pressure, and Cheyne–Stokes breathing are referred to as Cushing's Triad, an ominous sign of decompensation. When intracranial elastance is low, further increases in ICP can result in brain tissue displacement and herniation of brain parenchyma across meningeal barriers, which heralds serious brain injury or death.

Intracranial pressure can be monitored in several ways. As described above, the clinical examination can indicate relative changes in ICP and therefore impact management. In addition, neuroimaging can be used to estimate intracranial elastance based on the degree of sulcal effacement and ventricular collapse. Invasive methods of ICP monitoring are routine; correct interpretation and management is critical. Ventricular catheters, parenchymal monitors, and subarachnoid bolts can all be placed at the bedside by trained neurosurgeons and neurointensivists. Ventricular catheters, which are considered the gold standard for ICP monitoring, can be used to drain CSF and therefore modulate ICP [1, 2]. Transduction of the ICP reveals a waveform composed of a larger P1, and smaller P2 deflections. This relationship becomes inverted as intracranial compliance decreases. The "A" wave or plateau wave is a sign of intracranial hypertension.

Numerous therapeutic maneuvers exist for lowering ICP, each with the common mechanism of decreasing the volume of one or more intracranial components. It cannot be overemphasized that all treatments of ICP carry risks and so each must be monitored closely. A rapid method of decreasing ICP is to raise the head as much as possible; this is not always possible, especially during surgery. Decreasing the size of a brain mass is not a job for the anesthesiologist; that can only be accomplished surgically or, if the mass is inoperable, perhaps by radiation or chemotherapy. Decreasing the amount of CSF can be accomplished using a ventriculostomy. Interstitial edema can be treated with corticosteroids but this is not an effective acute treatment. Reduction of cytogenic cerebral edema can be achieved with the administration of osmotic or loop diuretics. Hyperosmotic agents like mannitol increase plasma osmolarity and draw water out of tissues including across an intact blood–brain barrier. Mannitol is infused slowly at a dose of 0.25–1 g/kg and may transiently increase ICP due to increased cerebral blood volume. Maximal reduction in ICP is evident after 10–15 minutes and remains effective for two hours. Hypertonic saline, commonly used in 3% and 7.5% solution, has proved effective in decreasing ICP. However, no evidence of improved neurologic outcomes or survival has been shown [3]. The loop diuretic furosemide is used to decrease ICP by systemic diuresis and decreasing CSF production. Furosemide does not increase intravascular volume and therefore is a better choice in patients with impaired left ventricular function. Intravascular volume and electrolyte balance should be monitored closely during the administration of furosemide and diuretic therapy. Hyperventilation reduces CBF 1–2 mL/100 g/min for each 1 mmHg reduction in $PaCO_2$ and therefore reduces ICP. However, reducing end-tidal CO_2 ($ETCO_2$) below 30 mmHg is not recommended due to compromise of CBF and the development of cerebral ischemia. As discussed above, MAP, CBF, and ICP are intertwined and therefore stable hemodynamics are critical to preventing further increases in ICP. Ultimately the pathologic process underlying intracranial hypertension must be corrected.

Anesthetic management for patients with elevated ICP begins in the preoperative setting. Preoperative anxiolysis should consist of a calm environment free of unnecessary distractions and, when indicated, medication. However, administration of preoperative sedatives may cloud the neurologic examination and should be considered carefully in patients with decreased intracranial elastance as modest hypercarbia resulting from hypoventilation may dangerously increase ICP via cerebral vasodilation. Arterial and central venous catheters may need to be placed after induction of anesthesia to avoid additional patient stress.

The operating room should be a calm environment before induction, free from music, loud conversation, or undue distractions. After application of standard monitoring and thorough preoxygenation, anesthesia can be induced with a combination of agents. Succinylcholine can be used if a rapid sequence induction is required but the resulting defasciculations can produce a transient increase in ICP. Adequate depth of anesthesia should be achieved prior to tracheal intubation and a bolus of sedative hypnotic or lidocaine can be administered before laryngoscopy. After placement of the endotracheal tube and confirmation of $ETCO_2$, the patient can be modestly hyperventilated. The arterial/end-tidal CO_2 gradient should be investigated and an $ETCO_2$ of 30–35 mmHg targeted. The use of positive end-expiratory pressure to maintain oxygenation must be applied carefully. Increased intrathoracic pressures, transmitted through compliant lungs, can impede cerebral venous drainage and further increase ICP.

In order to assure a stable operative field, the patient's head may be placed in Mayfield pins. Prior to the application of pins, a bolus of sedative and or narcotic should be administered to blunt the response to this profound stimulation. Positioning for potentially lengthy neurosurgical procedures is of critical importance and should be carried out with the cooperation of members of the operative team. Patients with elevated ICP should be maintained in a neutral head position with slight reverse Trendelenburg bed tilt to ensure cerebral venous drainage [4].

Maintenance of anesthesia can be achieved with a variety of agents tailored to the patient's medical history and the presence of neurologic monitoring. All combinations should have the common traits of adequate depth, stable hemodynamics and lowered ICP. Volatile anesthetics are potent cerebral vasodilators and progressively abolish the cerebral autoregulatory curve at higher doses. However, volatile agents do decrease cerebral metabolic rate of oxygen and at modest doses do not produce significant elevations of ICP. By contrast, propofol and thiopental

both reduce cerebral metabolism and decrease cerebral blood volume by vasoconstriction [5]. This combination produces a reduction in ICP. The use of neuromuscular blockers may also facilitate the management of ICP by preventing patient bucking or straining.

Conclusion

In conclusion, increased ICP can be a life-threatening condition, the definitive treatment of which is often in the hands of the neurosurgeon. It is the job of the anesthesiologist to effectively manage increased ICP until such treatment can be accomplished.

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Part I
Case
3

Craniotomy. Supratentorial craniotomy

Traumatic brain injury

Adam Brown and Paul S. Moor

Traumatic brain injury (TBI) is a leading cause of death and permanent disability in the western world and imparts a significant social burden. According to recent US statistics, of the reported annual TBI cases, approximately 50 000 patients die, 80 000 have permanent disability, and 235 000 are admitted to hospital [1]. With respect to mortality, 50% of patients who die from TBI do so within 2 hours of injury, and approximately 30% of patients admitted to a hospital with a Glasgow Coma Scale (GCS, Table 3.1) <13 will ultimately die.

The injury spectrum in TBI encompasses not only the initial insult, but includes the cascade of systemic responses and pathophysiology that occur after the focal or diffuse brain injury. Anesthesiologists are often involved throughout the care of TBI patients. It is through our effective physiologic manipulation of and pharmacologic intervention in elevated intracranial pressure (ICP), decreased cerebral perfusion pressure (CPP), secondary ischemic brain injury, and metabolic derangements, that we can positively influence patient outcome.

Case description

The patient was a 49-year-old male motorcyclist who was evaluated in the emergency room after a motor vehicle collision with an articulated truck ("Semi"). He was found prone 100 feet from the expressway with bilateral upper limb deformities, coarse respirations, and a GCS of 3. Endotracheal intubation was performed at the scene and transportation to our facility was complicated by five episodes of ventricular tachycardia, each requiring direct current cardioversion to sinus rhythm.

On arrival at the emergency room, primary survey revealed a normothermic intubated male with a patent size 7.0 mm endotracheal tube and cervical collar in place; breath sounds and chest excursion were equal bilaterally. Pulse oximetry revealed saturations of 100% on 15 liters oxygen, via nonrebreather mask. Five-lead EKG monitoring showed sinus tachycardia of 110 beats per minute without ectopy. Neurologically the patient was moving all four limbs spontaneously and purposefully, prior to anesthesia for computed tomography (CT) scan. Standard monitoring was applied and large bore intravenous access was obtained in two limbs; radial artery cannulation afforded invasive arterial blood pressure monitoring, as well as assessment of adequate gas exchange, balanced electrolytes and blood glucose. The patient was stabilized, sedated, and transferred for CT scanning.

Ongoing monitoring of pupillary size and reactivity was also maintained prior to direct ICP measurement. In the paralyzed and sedated patient, such clinical signs were indicative of tentorial herniation.

Noncontrast head CT revealed a right-sided epidural hematoma (Figure 3.1). Preliminary evaluation of the CT revealed no fractures or malalignments of the cervical spine. The preliminary review of the CT of the chest, abdomen, and pelvis revealed evidence of numerous, but stable, pelvic fractures and a right humeral fracture.

Around the time of CT scanning, the patient's right pupil became dilated and unreactive (6 mm unreactive right vs 3 mm reactive left). He was subsequently transported to the operating room for immediate craniotomy, evacuation of the extradural hematoma, and ventriculostomy insertion. The primary concern of the anesthesia team was poor intracranial elastance, increased ICP and potentially inadequate cerebral perfusion in the face of hemorrhagic hypovolemia. The team was also focused on the prevention of secondary brain injury [2, 3]. This required vigilance for and prevention of hypotension, hypoxia, hypercarbia, hyperthermia, and hyperglycemia, as these derangements contribute to enhanced parenchymal injury. A further concern, heightened by the mechanism of injury and polytrauma, was the suspicion of an accompanying