

Title: Help! He was just awake, now he's not! Acute Cerebral Herniation during pre-operative evaluation.

Moderators:

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Goals: Participants will:

1. Discuss cerebrovascular physiology in children.
2. Prepare a preoperative plan, monitoring techniques, anesthetic agents and management strategies to prevent secondary brain injury.
3. Discuss the signs and symptoms, differential diagnosis, and immediate treatment of acute cerebral herniation.
4. Discuss hyperosmolar therapies and hyperventilation strategies and their impact in the pediatric population with intracranial pathology.
5. Contrast different IV fluid therapies as well as establish blood transfusion thresholds in the pediatric neurosurgical patient.

Case History

A 6 year old presents to the ER with mild altered mental status after bumping heads with his brother while on a trampoline. The patient was mildly somnolent but otherwise neurologically intact. CT of his brain reveals a very large cystic tumor in the right frontal temporal region with moderate mass effect. He then undergoes MRI with sedation. Intermittent bradycardia was noted. The child recovers uneventfully, transfers to PICU, and is scheduled for resection via craniotomy the following morning.

Questions: Preoperative assessment and planning

What are the different types and locations of brain malignancies in children? What are the most common types and locations? What is the age distribution of tumor types in children? What are your concerns with intracranial space occupying lesions? How would the patient's history and physical help with your assessment of ICP? When looking at the imaging results, what information is relevant to planning your anesthetic? What laboratory studies would you obtain? How would positioning affect your plans? What monitoring modalities and lines would you employ? Role for premedication?

Case History

You review all pertinent images and note a large L FRONTAL TUMOR with significant mass effect, midline shift and surrounding edema. Laboratory results are all normal except for a sodium of 132 mEq/L. The

nurse reports that the patient has been neurologically appropriate other than a mild headache, and has had a light meal since returning from the radiology suite. In addition the nurse reports that the patient's blood pressure has been elevated ranging from 115 to 133 systolic and 70 to 85 diastolic, ECG monitoring has been mostly NSR with occasional PVCs. You enter the room and begin examining the patient and notice that the child now is unresponsive has abnormal posturing to noxious stimulus to his fingernail bed and his right pupil is fixed and dilated.

Questions: Crisis Management

How does intracranial compliance differ between infants, children and adults? What are the components to ICP? What are the different types of neurologic herniation? How would you treat acute cerebral herniation and what are the mechanisms responsible for their effect? What drugs would you administer to secure the airway?

Case History

The head of the bed is immediately elevated and you begin to hyperventilate the patient by bag mask with 100% oxygen. This improves the patient's neurological exam though he remains somnolent? A modified rapid sequence intubation with IV lidocaine, propofol and rocuronium is performed in the PICU. His vital signs remain stable. A dose of Mannitol is administered (0.25g/kg). After successfully intubating the patient, you continue to hyperventilate the patient and rush him to the OR for emergent BIFRONTAL craniotomy.

Questions: Perioperative management

What is your anesthetic plan? What lines do you obtain and how do you monitor this patient? How do you maintain anesthesia? Is one technique for maintenance of anesthesia superior to the rest? What are the effects of anesthetics on CMRO₂, CBV, CBF and Flow – Metabolism coupling, Auto-regulation and CO₂ responsiveness? How do you choose a hyperosmolar agent and how does it work? What type of fluids do you choose?

Case History

Upon entering the OR you continue to hyperventilate to target ETCO₂ of 30mmHg, administer a repeat dose mannitol dose? and the patient is positioned and prepped for surgery while you obtain arterial access and an additional i.v. line. Blood gas analysis shows : pH 7.46, PaO₂ 412, PCO₂ 31. The surgeon performs a craniotomy and notices the dura appears very tight.

Questions: Perioperative management

What are the controversies surrounding hyperventilation? How does your anesthetic choice affect CO₂ responsiveness and how low of a PaCO₂ do you consider safe? What immediate options do you have to lower ICP prior to durotomy? What is your target MAP? If you choose to augment MAP, how will you do so and what are the effects on cerebral vascular resistance? Is there a role for mild or moderate hypothermia?

Case History

Durotomy is performed, the tumor is identified and resection commences. While the tumor is being resected, the patient becomes hypotensive. You begin to transfuse PRBCs and notice a lack of blood

pressure response to volume challenges. ETCO₂ declines and hypotension worsens. You inform the surgeon to stop while you stabilize the patient for a suspected venous air embolism.

Questions: Crisis Management

What are your immediate treatment goals VAE? What else is in your differential? How would you evaluate? What is your goal HCT?

Case History

After a one-half blood volume resuscitation, hemodynamic stabilization, and full resection of the tumor, the surgeon begins to close.

Questions: Perioperative and postoperative management

How and when would you choose to normalize PaCO₂? What would be your criteria for extubation for this patient? If you choose to extubate the patient, how will you do so? What problems would you anticipate in the post-operative course?

Discussion:

Acute herniation in a child with a frontal tumor presents with various challenges. Given the limited compliance of the cranial vault in children, small changes in intracranial volume lead to large changes in ICP in patients who already have considerable mass effect from malignancies with significant edema. Immediate attention to decreasing ICP must take precedence while awaiting definitive treatment. During acute herniation, understanding the impact of anesthetic agents, hyperosmolar therapies, as well as ventilation strategies on the components of intracranial pressure (CSF, Cerebral blood volume, interstitial fluid and cellular mass) help guide treatment with the goal of decreasing the extent of secondary injury.

Hyperosmolar therapies are cornerstone to decreasing ICP in acute herniation. The immediate effect following administration is due to an expansion of the intravascular compartment with a concomitant rise in CPP, as well as a decrease blood viscosity, both leading to increases cerebral oxygenation thereby causing reflex cerebral vasoconstriction via autoregulatory mechanisms (decreasing CBV). The more delayed effects are secondary to raising plasma osmolarity beyond the normal value 285-295 milli-osmoles per kg with shifting of parenchymal fluid to the intravascular plasma compartment. Overall, both mechanisms lead to a decrease in ICP. Of the available hyperosmolar therapies, Mannitol is the most used pharmacological agent in the perioperative setting, though hypertonic saline is increasingly being used as first or second line therapy. With regards to improving surgical conditions in elective craniotomies, recent studies have demonstrated equal efficacy between both therapies.

Hyperventilation leading to a rapid decline in PaCO₂ decreases ICP via cerebral vasoconstriction. As a “bridging” measure in acute herniation, decreasing PaCO₂ is clearly advantageous. Routine hyperventilation in elective intracranial surgery is controversial given the potential of decreasing cerebral blood flow below ischemic thresholds despite clearly improving surgical conditions both in patients anesthetized with either propofol or volatile based techniques. Mechanisms for cerebral vasoconstriction in the setting of acute hypocapnea are not clearly elucidated though there is experimental evidence that increases in CSF pH lead to increases in intracellular calcium concentration in cerebral smooth muscles resulting in increase vascular tone.

Anesthetic technique employed needs to be carefully considered given that the anesthetic agents used may have deleterious consequences and possibly worsen outcome. The anesthetic effects on CBV and ICP differ in patients with altered cerebral physiology. This is particularly evident in those with critical elevations in ICP. Modern volatile agents are proven to be safe when used in patients with normal to mildly decreased intracranial compliance, though when used in settings of critically low intracranial compliance, their vasodilatory effects may increase ICP, reducing CPP and raising likelihood of secondary injury. Though Intravenous agents and Volatile agents both reduce CMRO₂, a propofol-based anesthetic has the advantage of consistently decreasing CBV and thus ICP.

Intracranial tumor resections present various challenges for the anesthesiologist. Though this discussion is very limited, other relevant topics is the use of therapeutic hypothermia both intraoperative and the postoperative settings, the effects of various vasoactive substances, the use of steroids, antiepileptic's and fluid management strategies. Refer to some of the references below for further reading.

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