DEVELOPING A SAFE ANESTHESIA PLAN FOR A TRAUMATIC BRAIN INJURY PATIENT
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Key points
- Normal physiology
- Management factors
- Anesthesia agents

What might be my priorities for safely providing anesthesia for the patient with a traumatic head injury? I will already assume that I have some degree of brain insult.

I will try to maintain normal physiology by ensuring:
- Maintenance of cerebral perfusion
- Maintenance of Autoregulation
- Maintenance of carbon dioxide reactivity
- Provide for the reduction of intracranial pressure

Cerebral blood volume (CBV) is the intracranial component that is capable of rapidly increasing or decreasing the circulating volume within the cranium. Small increases/decreases in either CBV or cerebral blood flow (CBF) can significantly alter intracranial pressures (ICP). To control CBF and CBV under anesthesia, monitoring PaCO2 is vital. Hypercapnia (PaCO2 > 45mmHg) will lead to vasodilation, causing increased CBF and increased ICP. Excessive hypocapnia (<25mmHg) will lead to vasoconstriction, decreasing CBF and causing ischemia. Controlling ventilation is of the utmost importance. Patients already exhibiting signs of increased ICP may benefit from PaCO2 between 30-25mmHg (mild to modest hyperventilation).

Hypoxia (PaO2 < 50mmHg) increases CBF which may cause increased ICP. Supplemental oxygen therapy in the perianesthetic arena may include an oxygen hood, oxygen cage, and flow by oxygen via a mask. Nasal cannulas should be avoided as restraint/placement may involve inadvertent pressure on the jugular veins inhibiting venous return from the brain.

Autoregulation may be blunted by the amount of trauma received by the patient. Autoregulation of CBF and ICP in the normal brain occurs when the PaCO2 is maintained between 30-40mmHg and PaO2 is > 50mmHg, and the mean arterial pressure (MAP) ranging between 50-150mmHg. Autoregulation may be blunted or impaired either focally or globally.

Cushing’s reflex, the occurrence of hypertension, bradycardia and alterations of respiratory rates/patterns, is seen following intracranial hypertension. The patient may initially show tachycardia with hypertension before bradycardia is seen. This is a hypothalamic response to brain ischemia which causes an increase in the peripheral vascular resistance. The baroreceptor response to hypertension causes bradycardia. This triad of activity, hypertension, bradycardia and respiratory alterations signals the possibility of brain herniation. Treatment may include the administration of mannitol, hyperventilating the patient and/or elevating the head of the patient from a 10-30 degree angle.
Anesthesia goals are to provide adequate tissue perfusion and oxygenation, and maintain ICP.

Premedication goals are to decrease anxiety, amount of induction agent and provide analgesia. Pre-oxygenation is a must and if the patient resents or fights a tight fitting mask, remove the rubber diaphragm to make it more acceptable.

Anticholinergics must be administered on a case to case, as needed basis.

Agents to avoid –
Anticholinergics must be administered on a case to case, as needed basis.

Acepromazine is controversial as it has been purported to reduce the seizure threshold.

Ketamine and tiletamine increase sympathetic tone, ICP, intraocular pressure (IOP). However; studies are being funded to explore the possibility in humans with hydrocephalus that ketamine decreases ICP.

Nitrous oxide may increase the volume or pressure within a closed space by equilibrating with closed gas spaces and its use should be avoided.

The use of alpha2 (α2) agonists in patients with neurological disease/dysfunctions are being studied in human trials.

Acceptable protocols:

Premedication with either low dose pure mu (µ) opioids +/- a benzodiazepine, +/- an anticholinergic. µ Opioids may produce vomiting in the pre-operative area so a low dose is recommended to avoid emesis. Respiratory depression may be seen with some patients receiving pure µ opioids which should be avoided.

Mixed agonists/antagonists have been shown to have little or no respiratory depressive effects. These agents may be safe to administer but will have less impact on the amount of induction agent and have little MAC sparing abilities if inhalant anesthetics are used. My personal preference would be butorphanol with the dose ranging from 0.2-0.4 mg/kg. Buprenorphine may be used but should intra-operative analgesics be required it will be harder to achieve due to the longer duration as well as the high affinity of binding with the Mu (µ) receptors.

Induction may include:

Administration of propofol (4-6 mg/kg calculated) IV to effect. Remember that a rapid bolus will cause apnea and the calculated dose should be titrated to effect. Propofol is a hypnotic with no analgesic properties. The patient may be maintained on a propofol CRI with adjunctive analsgesics being administered and mechanical ventilation being provided to control PaCO2. The usual dose for a propofol CRI is 0.4mg/kg/minute and may be adjusted up or down according to patient response provided the patient is adequately analgesed.

Fentanyl, hydromorphone or oxymorphone in combination with either diazepam or midazolam administered IV. Depending on the premedicant and maintenance plan, you may administer fentanyl (5mcg/kg), hydromorphone (0.1mg/kg to 0.2mg/kg) or oxymorphone (0.1mg/kg to 0.2mg/kg) followed by your benzodiazepine of choice. I use the 0.2 mg/kg dose for either benzodiazepine. Rapid capture of the airway is desired.
Etomidate, another hypnotic agent, has not been incriminated in an increase in ICP, IBF or IBV and should be safe to use in combination with a benzodiazepine. Induction with the etomidate/benzodiazepine combination is not necessarily rapid and the patient may have untoward side effects of myoclonic activity as well as nausea and vomiting.

Patient maintenance may be achieved by either incorporating the use of isoflurane, sevoflurane or desflurane at 1.1 MAC or less. These inhalant anesthetic agents have been shown to increase CBF and ICP at concentrations higher than 1.1 MAC.

Total intravenous anesthesia (TIVA) may be utilized as well, patient needs and facility capabilities dependant.

Balanced anesthesia/analgesia should be obtained with the use of multiple agents to control systemic blood pressure, heart rate, depth of anesthesia and appropriate analgesia.