Elevated intracranial pressure management

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ABSTRACT

The treatment strategy for increased intracranial pressure (ICP) management includes decisions on head and body position, hypothermia, ventilation, anesthetics, osmotic drugs and surgical procedures. We can treat sudden increases in ICP using osmotic agents, some anesthetics and short episodes of mild hyperventilation. Propofol seems to be suitable for sedation of the increased ICP patients. Surgical decompression of the cranium seems to improve the outcome of the younger patients (below 50 years old), especially children.

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The tissue of the brain accounts for approximately 88% of the intracranial volume, the cerebrospinal fluid (CSF) contributes 9%, and the vascular system of the brain's intravascular blood is the smallest component, with 2-3% of the volume. Most of the cerebral blood volume (CBV) resides in the venous system, whereas only 15% of the CBV is in the arteries and 15% in the sinus system. Normal intracranial pressure (ICP) is less than 15 mm Hg. Elevated ICP can result from changes in the vascular compartment, including hyperemia or hematoma caused by vascular rupture. Factors affecting ICP include tumors, vasogenic, cytotoxic and interstitial edema secondary to hypoxia, infection, and trauma. The obstruction of CSF pathways and an alteration in CSF production or reabsorption may also increase ICP. Space-occupying lesions elevate ICP, and this can be compensated for especially if the process develops slowly. The ability to compensate for increases in compartmental volume is termed intracranial compliance, and is defined as the change in ICP to a change in intracranial volume. Any increase in the volume of one compartment will be compensated by volume reduction in another compartment to maintain normal ICP. The CSF system has the best buffering capacity of the intracranial compartments. Expansion of a non CSF intracranial component results in the displacement of CSF from the cranium through the foramen magnum into the spinal subarachnoid space. Further volume compensation may be provided by increased absorption of CSF by arachnoid villi. This mechanism is pressure dependent to an upper ICP limit of approximately 30 mm Hg. Reduction of CBV provides additional space for an expanding lesion. The CBV reduction occurs first by compression of the low pressure venous system, followed by capillary collapse, leading to cerebral edema and ischemia. The cerebral perfusion pressure (CPP) is equal to mean arterial blood pressure (MAP) minus ICP. An adequate CPP is probably more important than the ICP per se. Elevated ICP treatment should emphasize the immediate reduction of ICP to less than 20 mm Hg. The most powerful predictor of poor neurological outcome in the patients with intracranial lesions is the presence of an ICP higher than 20 mm Hg.[1] The CPP should be kept between 60-70 mm Hg. In head trauma patients, a CPP below 60 mm Hg is associated with poor outcome. Episodes of hypotension with a lower CPP as well as hypoxemia in severe traumatic brain injury (TBI) patients.[2]

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Position of head and body. As a result of flexion, extension, or torsion of the neck ICP increases because the cerebral venous outflow is obstructed.[2] Giving a reverse Trendelenburg position in prone or supine positioned patients reduces ICP without affecting CPP.[3,4] The beneficial effect of the prone position on cerebral tissue oxygenation by increasing arterial oxygenation appears to outweigh the expected adverse effect of prone positioning on cerebral tissue oxygenation by decreasing cerebral perfusion pressure in acute respiratory distress syndrome patients.[5]

Intracranial pressure moniterization. The main goal of detecting ICP is to prevent and treat herniation, and brain shifts when ICP increases. The gold standard for ICP measurement is ventricular catheterization, because of its accuracy and the possibility of the therapeutic CSF drainage for control of increased ICP. The ICP can be monitored in the intraventricular, the intraparenchymal, the subarachnoid, and the epidural space.[6]

Hypothermia. Hypothermia can be used to control increased ICP, but no conclusion has been drawn on whether this improves the outcome.[7] Prolonged hypothermia may be beneficial, particularly in patients with increased ICP refractory to conventional manipulations.[8] The indication for hypothermia is not clear, obviously hyperthermia should be avoided.[9]

Anesthetic agents. Sedation using narcotic and hypnotic agents is performed to reduce stress and to control ICP in patients with intracranial lesions. The choice of anesthetic agents is based on the consideration of their effects on cerebral blood flow (CBF), the cerebral metabolic rate for oxygen ($CMRo_2$), CBV, cerebrovascular autoregulation, and carbon dioxide reactivity. When you use higher concentrations of volatile anesthetics, you begin to depress cerebral metabolism, and if you increased the dose of the anesthetics, depression level increases. With this depression, the cerebral vasodilation increases and as a result ICP increaes. Desflurane produces more cerebral vasodilatation than isoflurane or sevoflurane,[10] whereas isoflurane is more vasoactive than sevoflurane.[11] Autoregulation and the carbon dioxide response remain intact with 1.0 minimal alveolar concentration (MAC) sevoflurane. At this concentration, sevo-flurane decreases cerebral metabolism and CBF is comparable to propofol,[12] 1.0 MAC sevoflurane is suitable for neurosurgical patients without hypertension. In contrast to propofol, sevoflurane does not reduce CBV. Intravenous anesthesia has received much attention in neuroanesthesia as a means to avoid the vasodilating effects of volatile anesthetics and nitrous oxide. Propofol maintains flow metabolism coupling intact even at higher doses,[13] induces cerebral vasoconstriction and decreases $CMRo_2$,[14] which results in decreased CBF, CBV and ICP. High doses of propofol (6-8 mg/kg per hour) can alter cerebrovascular autoregulation in TBI patients.[15] Using barbiturates results in a reduction in CBF and CBV secondary to suppression of cerebral metabolism.[16] In hemodynamically stable patients, barbiturates can control ICP when other treatments have failed, but there is no evidence that it improves the outcome of the acute severe head injury patients. In severe TBI patients, the infusion of ketamine/midazolam has a similar effect on mean daily values of ICP and CPP as sulfentanyl/midazolam.[17] Patients receiving ketamine had a lower requirement for catecholamine infusions, were hemodynamically more stable, and enteral food intake was earlier. Furthermore, ketamine has a bronchodilatory effect, and in experimental studies, a neuroprotective effect of ketamine was demonstrated.[18] Therefore, ketamine can be used for analgesia in TBI patients. Sufentanil and remifentanil seem to have no influence on middle cerebral artery blood flow velocity and ICP as long as MAP is stable.[19,20] Transient increases in ICP without changes in middle cerebral artery blood flow velocity occurred concomitant with decreases in MAP. Remifentanil can be used for analgesia in TBI patients without adverse effects on cerebrovascular hemodynamics, CPP or ICP.[19]

Osmotic drugs. Mannitol administration is the first choice for ICP reduction and is recommended as a guideline.[21] Mannitol has an immediate plasma-expanding effect that reduces hematocrit and blood viscosity and increases CBF and cerebral oxygen delivery. This effect might explain the early decrease in ICP. Hyperosmotic agents remove more water from the brain than from the other organs because the blood-brain barrier impedes the penetration of the osmotic agent into the brain, maintaining an osmotic diffusion gradient. This osmotic effect of mannitol is delayed for 15-30 minutes. Mannitol decreases ICP for 1-6 hours. In TBI patients, mannitol is superior to pentobarbital, improving CPP, ICP and outcome.[22] Mannitol seems to be most effective in reducing ICP when compared with ventriculostomy drainage or hyperventilation.[23] A single short administration of high dose mannitol (1.4 g/kg) in the emergency room significantly improves the 6-month clinical outcome after TBI.[24] To reduce ICP, several studies have shown that hypertonic saline is equal or even superior to mannitol. Vialet et al[25] suggested that hypertonic saline (2 ml/kg, 7.5%) is an effective and safe initial treatment for intracranial hypertension episodes in TBI patients when osmotherapy is indicated. Even very high concentrated hypertonic saline solutions (23.5%)

can be used and can reduce ICP in poor grade patients with subarachnoid hemorrhage.[26] The most important risk of administration of hyperosmotic agents is the rebound effect, which might increase ICP. To reduce this rebound effect risk, it is recommended that mannitol should be used as repeated boluses rather than continuously, only in patients with elevated ICP and no longer than 3-4 days.[27] Mannitol is entirely excreted in the urine, and there is a risk of acute tubular necrosis, particularly if serum osmolarity exceeds 320 mOsmol/l.[28] As an antidiuretic drug, furosemide itself has only minimal effect on ICP, in combination with mannitol on plasma osmolality, resulting in greater reduction of brain water content.[29] Therefore, furosemide can be recommended as a supplemental treatment.

Mechanical ventilation. Mechanical ventilation is crucial in all TBI patients with high ICP. The control of arterial oxygen tension and arterial carbon dioxide tension ($Paco_2$) is mandatory and will affect cerebral hemodynamics and ICP. Patients with isolated TBI can be managed with traditional ventilatory strategies, but those with chest trauma, pulmonary aspiration, or massive resuscitation after shock are at a high risk of developing acute lung injury. In those patients, to improve oxygenation and to avoid atelectasis the use of positive end expiratory pressure (PEEP) has to be used.[30] The PEEP may increase intrathoracic pressure and potentially increase ICP by impeding venous drainage or reducing CPP.[6] The clinical relevance of these small changes in ICP caused by a PEEP of 10-15 cmH_2O is questionable because it does not affect CPP.[31,32] It seems to be justified to use PEEP levels up to 10-15 cmH_2O in patients with intracranial hypertension and severe chest trauma. Hyperventilation increases pH in the extracellular space, constricts cerebral blood vessels, decreases CBV and CBF, and thereby rapidly reduces ICP, whereas it has no effect on cerebral metabolism. In patients with elevated ICP, CBF might already be reduced to a critical threshold of 18-20 ml/100g brain tissue per minute, whereas metabolism is maintained. Further reduced CBF by hyperventilation aggravates the flow metabolism of imbalance, leading to cerebral ischemia. The effect of hyperventilation on ICP is only transient, because the extracellular space of the brain rapidly accommodates to the pH change induced by hyperventilation.[33] Hyperventilation can be life saving and can temporize until more definitive treatment for intracranial hypertension can be undertaken.

Decompressive craniectomy. After TBI or stroke, decompressive craniectomy is a surgical procedure used to control severely increased ICP and to prevent herniation. The beneficial effect of decompressive craniectomy in the treatment of patients with intracranial hypertension is controversial.[34] The most common cause of brain death in patients with TBI is uncontrollable intracranial hypertension. As a result, direct monitoring and treatment of intracranial hypertension has long been considered a cornerstone of TBI management. Early decompressive craniectomy is obviously not aimed directly at preventing secondary brain injury but, rather, at alleviating elevated ICP. Numerous anecdotal or cohort reports suggest the benefits of this procedure. However, to date no well designed, prospective, randomized trial has demonstrated the efficacy of decompressive craniectomy for improvement of outcomes following TBI. In a pilot study, Ruf et al[35] found that surgical decompression using craniectomy is largely seen as a last resort therapeutic option. Restoration of cerebral perfusion by surgical enlargement of the intracranial space is the primary goal of decompression.[36] This may necessitate a large craniotomy with duraplasty. Taylor et al[37] demonstrated an improved neurological outcome of patients who were treated with an early decompressive craniectomy in a cohort of 27 children compared with historical controls. For surgical decompression, the heterogeneous approach is used, including unilateral or bilateral fronto-temporo-parietal, bifrontal or suboccipital or hemicraniectomies.[35,38] Sometimes the removed bone flap is stored by cryopreservation or a subcutaneous part of the abdominal wall is used to keep, until secondary cranioplasty. Craniectomy is planned with duraplasty or not. If duraplasty is planned then an autologous galeal flap or any synthetic dura flaps are used. The results of surgical treatment in patients less than 50 years of age undergoing decompressive craniectomy are even more encouraging.[39] After severe TBI with severe brain edema, decompressive craniectomy with duraplasty improved good outcome for all patients, especially in children.[40-42] The craniectomy should be performed in the interval up to 48 hours after the trauma, before irreversible brainstem damage or generalized brain damage has occurred.[42]

In conclusion, as an intravenous anesthetic agent, propofol seems to be suitable for the patients with ICP. It decreases ICP but at the same time reduces CPP. In hemodynamically stable patients, high dose barbiturates may be considered. In immediate and refractory intracranial hypertension, use of mild hyperventilation may be preferred. The use of prophylactic hyperventilation therapy should be avoided because it can compromise cerebral blood perfusion during a time when CBF is reduced. Serum osmolarity should be kept below 320 mOsmol/l and hypovolemia should be avoided when mannitol

or hypertonic saline is administered. In the case of sustained increase in ICP (>20 mm Hg) under intensified conservative therapy conditions and early decompressive craniectomy, including duraplasty has to be considered.

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