

Intracranial Hypertension in the Perioperative Period

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Control of intracranial hypertension (ICHT) is crucial when treating patients with head trauma and treatment in these cases has already been extensively described in the literature.¹⁻³ Although few authors have focused on this aspect, ICHT is a particular problem during the perioperative period when factors such as cerebral retraction pressure and the ability to provide optimal surgical conditions come into play. These aspects of ICHT treatment will be reviewed in this issue of *Anesthesiology Rounds*.

PHYSIOPATHOLOGY OF INTRACRANIAL HYPERTENSION

The adult brain is contained in a hard cranial shell with a fixed volume. Its contents can be divided into three parts: the cerebral parenchyma (which accounts for 90% of the contents and includes the intra- and extracellular fluids and cellular membrane), and the arterial and venous blood, and cerebrospinal fluid (CSF), (that together make up the remaining 10%). The Monroe-Kellie hypothesis as modified by Cushing at the turn of the last century states that, "if the cranium is intact, the sum of the volumes occupied by the cerebral parenchyma, CSF, and blood compartment is constant." Thus, any increase in the volume of one of the components must be compensated by a reduction in the volume of either or both of the others (compensatory mechanisms) for the intracranial pressure (ICP) to remain constant.⁴

ICHT can therefore develop when 1 of the 3 components increases in volume, or an expansive lesion forms and depletes the compensatory mechanisms. Expansive lesions, whether or not associated with perilesional edema, are the primary cause of ICHT in the perioperative period. Non-communicating hydrocephalus and an increase in cerebral blood volume due to vasodilatation or a blocked vein are less common causes.

The relationship between variations in intracranial volume and ICP is non-linear (Figure 1). Initially, a slow climb in intracranial volume will not cause the ICP to rise because the CSF is translocated to the medullary compartment, CSF reabsorption may increase, and the venous portion of the cerebral blood volume may decline. In this case, intracranial compliance is normal (segment 1 of the curve in Figure 1). Once these compensatory mechanisms are exhausted, however, a tiny increase in volume will lead to a sharp rise in ICP and intracranial compliance is reduced (segments 2 and 3 of the curve in Figure 1).

Normal ICP ranges between 10 and 15 mm Hg. A value in excess of 18 to 20 mmHg is abnormal and must be treated. Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial pressure (MAP) and ICP if the latter is known and higher than the central venous pressure. Cerebral arterial circulation is normally autoregulated to maintain a constant cerebral blood flow (CBF) for a CPP between 50 and 150 mm Hg (Figure 2). Outside these limits, CBF varies linearly with MAP. Below 50 mm Hg CPP, the vasodilatory capacity of the cerebral vessels is exhausted, CBF drops, and there is a risk of cerebral ischemia. Above 150 mm Hg, the diameter of the vessels is reduced to the minimum and the increased CBF may lead to cerebral edema. When autoregulation is normal, CBF will return to its base value within 5 seconds of an abrupt change in MAP. Autoregulation can be generally or regionally disrupted by cranial trauma, subarachnoid hemor-

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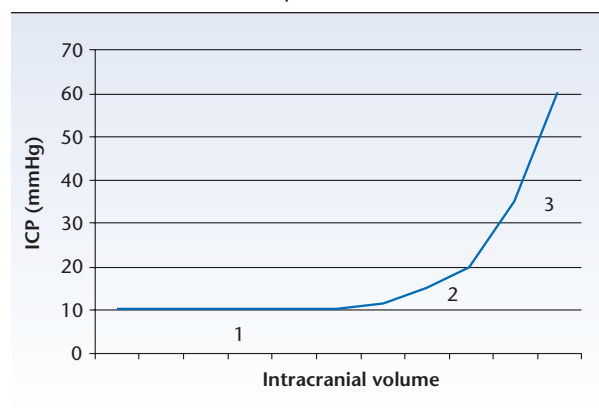
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FIGURE 1: Intracranial compliance curve



rhage, some anesthetic agents, the presence of a brain tumour or an arteriovenous malformation, hypoxemia, and excessive hypercarbia. With a chronically hypertensive patient, the autoregulation curve shifts to the right (Figure 2).⁵ It is believed that the autoregulation curve quickly returns to its normal position once the hypertensive patient has been treated.

There is still some controversy surrounding the existence of the ideal CPP for head-trauma patients.⁶ Nonetheless, for patients with reduced intracerebral compliance or ICHT in the perioperative period, maintaining the CPP above the estimated lower threshold of autoregulation is generally recommended.

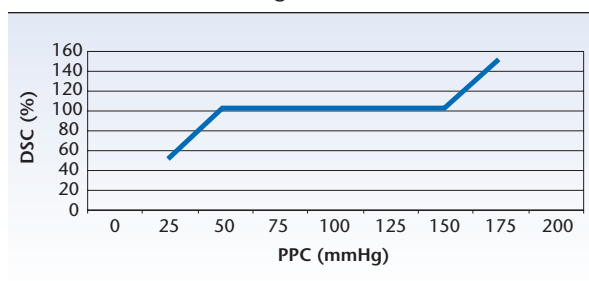
SECONDARY CEREBRAL DAMAGE

The complications associated with ICHT are serious, ranging from cerebral ischemia to rapidly fatal transtentorial herniation. In traumatology literature, these complications are classified under the heading “secondary cerebral damage” and are considered avoidable. Secondary cerebral damage may be caused by ICHT as well as the factors that aggravate it, particularly hypercarbia. Hypoxemia, hyperglycemia, hyperthermia, and arterial hypertension are also important contributing factors. In the intraoperative period, in addition to these conditions, herniation through the craniotomy phase, ischemia by cerebral retraction, and poor surgical conditions are complications that contribute to secondary cerebral damage. Anesthesiologists, through a solid understanding of the mechanisms at play, are in a good position to prevent these complications.

INTRACRANIAL HYPERTENSION IN THE PERIOPERATIVE PERIOD

The position of the patient is an important factor in treating ICHT; this also applies during the perioperative period. The head must be raised (15° to 30°) and in a neutral position (if the surgery permits) to allow good jugular venous drainage.^{7,8} Coughing must be prevented and the patient must not be

FIGURE 2: Cerebral autoregulation curve



allowed to fight the respirator, two conditions that can cause a sharp rise in ICP. Pain, as well as agitation and convulsions, must also be treated, preferably with short-acting agents, to allow a neurological examination if necessary. The procedures for treating ICHT are meant to prevent the components of the intracranial space from increasing in volume, or aimed at decreasing them.

Induction of anesthesia

Ideally, anesthesia is induced in patients with ICHT by means of an intravenous agent such as thiopental, propofol, or etomidate. All reduce ICP and, to varying degrees, are also likely to lower CPP. Thiopental and etomidate reduce ICP more than MAP, thereby maintaining or increasing CPP. Propofol may have stronger effects on the MAP than on the ICP, thereby maintaining or decreasing CPP. Etomidate is associated with a stable hemodynamic profile and can therefore be used in hypotensive patients who cannot be satisfactorily stabilized before anesthesia induction. A synthetic opiate with no histamine-releasing effect (fentanyl and derivatives) and/or lidocaine (1.5 mg/kg) are also used for better hemodynamic control during intubation. There are no data indicating that one opiate is preferable over another for patients with ICHT, as long as the CPP is kept within the autoregulation limits. The chosen opiate should be administered immediately before or after the induction agent to keep the patient from hypoventilating before the anesthesiologist can control the ventilation. If the patient with ICHT presents with a full stomach, rapid sequence induction should be used; however, ICHT as such is not an indication for rapid sequence induction. With rapid sequence induction in patients with ICHT, it is pointless to ask them to hyperventilate voluntarily before induction because, not only is it anxiogenic, it also precludes obtaining a lower postintubation PaCO₂ level than during conventional rapid sequence induction.⁹ A non-depolarizing rapid-onset muscular relaxant such as rocuronium, or succinylcholine preceded by a defasciculating dose of a non-depolarizing agent, are good choices for facilitating intubation in patients with ICHT. It is important to

monitor the level of the neuromuscular block and to ensure that the patient is completely curarized before intubating the airway.

Mannitol, furosemide, hypertonic saline

Agents with high osmotic pressure such as mannitol and hypertonic saline cause water in the cerebral parenchyma to cross the hematoencephalic barrier (HEB) into the intravascular space. Osmotic pressure is the main force regulating the flow of fluid through the intact HEB and its maintenance is an essential factor in preventing peritumoural edema.¹⁰ Oncotic pressure plays only a modest role.

Mannitol: A 20% mannitol solution at the dose of 0.5 to 1.0 g/kg administered 30 to 45 minutes before the dura mater is opened is the most commonly used osmotic agent (1280 mOsm/kg). Mannitol dehydrates the brain tissue and subsequently reduces cerebral blood volume and CSF formation.¹¹ Mannitol administration requires an intact HEB and it may be less effective if cerebral damage is extensive. Mannitol acts in two phases: there is an initial brief increase in ICP (due to the increase in the circulating cerebral volume), followed by a sharp and prolonged drop in ICP (about 4 hours). The initial rise has no clinical effects during general anesthesia and would be more pronounced if the base ICP is normal.¹² Mannitol is frequently combined with a small dose of furosemide (0.1 to 0.2 mg/kg) for a synergic, longer-lasting reduction in ICP.

Furosemide: The administration of furosemide alone does not automatically reduce the ICP.^{13,14} The mechanism underlying its action is not clear. Furosemide does not increase plasmatic osmolarity and therefore does not reduce the brain's water content.¹⁵ Only a very strong dose of furosemide can reduce CSF production. Combined with mannitol, furosemide potentializes the increase in plasmatic osmolarity and inhibits active ionic cellular regulatory mechanisms, thereby preventing the repletion of the cerebral intracellular volume.¹⁶ The use of furosemide as the sole agent can be considered in cases of severe heart failure that contraindicate the use of mannitol. In this case, initiating substantial diuresis with furosemide might also precede the use of mannitol.

Hypertonic saline: Hypertonic saline, although used less frequently than mannitol intraoperatively, produces similar surgical conditions.¹⁷ Few studies in humans have proposed hypertonic saline as treatment for cerebral edema or ICHT. The mechanism of action of hypertonic saline is similar to mannitol and based on the same premises, notably the presence of an intact HEB. In the literature, the concentrations studied have varied considerably (3%, 7.5%, and 23.4%, respective osmolarity: 1026 mOsm/L,

2565 mOsm/L, and 8008 mOsm/L) and there is no clearly defined dose-response curve. The safety of hypertonic saline has not been accurately assessed in humans; however, aside from hypernatremia and its potential consequences, few side effects have been reported. During the intraoperative period, a 3% hypertonic saline is administered at the dose of 250 to 500 cc approximately 45 minutes before the dura mater is opened. The resultant reduction in ICP is of shorter duration than with mannitol (about 2 hours). For the same indication, 2.5 ml/kg of 7.5% saline can also be used.¹⁷ Natriuresis and the secondary increase in diuresis caused by the hypertonic saline is less than that induced by mannitol. Hypertonic saline may therefore be a better choice than mannitol for patients in a precarious volemic state, although no randomized prospective study has assessed hypertonic saline in this clinical context.¹⁸

Furosemide may theoretically augment the effect of hypertonic saline. The possibility of hypernatremia, as well as mannitol's safety record and known antioxidant effect¹⁹ make hypertonic saline an agent of second choice for treating ICHT in the perioperative period. Anecdotally, 30 to 60 ml of 23.4% saline was able to reduce the ICP of patients resistant to the usual treatment for ICHT, including mannitol.²⁰

Volemic repletion

The use of an intravenous hypo-osmolar solution (5% glucose, Ringer's lactate) should be discouraged in neurosurgery patients during the perioperative period. It has been demonstrated on a number of occasions and in different animal models that hypo-osmolar solutions increase the ICP, as well as the quantity of water in both healthy and injured brain tissue.^{21,22} Use of colloids do not offer an advantage over an isotonic salt solution (0.9%).²³ The clinician should aim to maintain normovolemia. Substantial dehydration, in addition to compromising the integrity of other organs/systems, does not produce a greater reduction in cerebral water content than the judicious use of a hyperosmolar solution.²⁴

Maintaining anesthesia

Although several anesthetic techniques give satisfactory results,²⁵ special attention must be given to the choice of anesthetic agents and their use for ICHT patients.

Normal CBF is 50 to 55 ml/100 g of brain tissue/minute (ml/100 gr/min). There is a close relationship between cerebral metabolism and the CBF. An increase in cerebral metabolism causes an increase in CBF through vasodilatation, while conversely, a decrease in cerebral metabolism leads to arteriolar vasoconstriction and reduced CBF. This effect on the cerebral vessels' tone is considered to be indirect

because it is mediated by changes in cerebral metabolism, rather than the result of a direct action on the vessels themselves. This metabolic coupling can be disrupted by head trauma and the use of halogenated anesthetic agents.

Halogenated agents have mixed effects on cerebral circulation. They reduce cerebral metabolism and have an indirect vasoconstrictive effect (preservation of metabolic coupling). Concomitantly, halogenated agents have a direct vasodilatory effect on cerebral vascularization and can therefore increase the CBF. The net effect on cerebral circulation depends both on the agent and dosage used. Sevoflurane is the least vasodilatory agent, followed by isoflurane and lastly, desflurane, the most powerful direct vasodilatory agent.^{26,27} Sevoflurane has a net vasoconstrictive effect on the brain and also preserves cerebral autoregulation up to a concentration of 2 MAC.²⁸ Isoflurane has a minimal effect on CBF up to a concentration of 1 MAC, but autoregulation is completely abolished at 1.5 MAC. Desflurane disrupts autoregulation at concentrations as low as 0.5 MAC and abolishes it completely at 1.5 MAC.²⁹ Desflurane is therefore not an agent of choice for patients with ICHT or reduced intracerebral compliance.

The return to a normal neurological state after extended anesthesia for a craniotomy is faster with sevoflurane than with isoflurane, because of their respective solubility.³⁰ However, the choice of anesthetic agent is not the only factor that influences how fast patients with intracranial pathology will emerge from anesthesia. It has been demonstrated that patients with a large brain tumour (>30 mm in diameter with >3-mm displacement of structures from the median line) take a long time to awaken compared to patients with a small intracranial lesion or those who have undergone a laminectomy.³¹

Nitrous oxide (N₂O) increases cerebral metabolism and therefore should not be used. As well, with growing use of agents with very low solubility (eg, sevoflurane), its use should become obsolete.

There is a threshold below which the CBF is insufficient to meet the brain's metabolic needs. This is known as the "critical CBF," below this level the brain becomes ischemic. In an awake patient, the critical CBF is 25 ml/100 gr/min. This threshold drops substantially with the use of certain anesthetic agents. Thus, the critical CBF is 10 ml/100 gr/min with isoflurane, 11.5 ml/100 gr/min with sevoflurane, and 20 ml/100 gr/min with halothane.^{32,33} Sevoflu-

rane and isoflurane therefore have an advantage if the clinician anticipates a substantial drop in CBF.

Propofol is also an excellent choice for maintaining anesthesia in patients with ICHT. It is an indirect cerebral vasoconstrictor with no direct vasodilatory properties and clinically, its effect on ICP and cerebral relaxation is either equivalent³⁴ or superior to that of isoflurane.³⁵

Cerebral blood vessels are also highly sensitive to fluctuations in PaCO₂. The relationship between the CBF and PaCO₂ is directly proportional and linear for PaCO₂ values between 20 and 80 mm Hg. The CBF varies by 4% for each 1 mm Hg change in PaCO₂. Reactivity to CO₂ is not influenced by halogenated anesthetic agents at the doses usually used in neurosurgery;³⁶ however, it is slightly reduced by propofol.³⁵ In the perioperative period, it is recommended to maintain the PaCO between 30 and 35 mm Hg in the patient with ICHT. Hyperventilation lowers the CBF without reducing cerebral metabolism. This can therefore be harmful for certain areas of the brain where substrate delivery is already compromised. Hyperventilation, as an adjuvant technique for the treatment of ICHT, should therefore only be considered as a last resort in the perioperative period, ie, when all other methods have proven ineffective.

THE INTRAOPERATORY THERAPEUTIC APPROACH TO BRAIN PRESSURE

The most common cause of ICHT during the intraoperative period is undoubtedly the presence of a brain tumour or some other expansive mass (hematoma). When the bone segment is removed, the dura mater will be very tense, making cerebral herniation inevitable once it is opened. A brain under pressure generally indicates a high ICP.³⁷ This can make the surgeon's job very difficult and limit exposure to the surgical site. For the patient, there are direct repercussions: more time on the operating table and the need to apply greater pressure to the cerebral retractors, which gives rise to the possibility of secondary cerebral ischemia. Hydrocephalus, frequently associated with high-grade subarachnoid hemorrhage, is a less common cause of brain pressure. CSF drainage, either at the lumbar or ventricular level, is then a very effective procedure to decompress the brain. A brain that suddenly becomes tense should alert the clinician to the possibility of a cerebral or intratumoural hemorrhage during the operation. Inappropriate curarization can also cause the

brain to protrude through the craniotomy incision. Patients treated with anticonvulsants recover quickly from a neuromuscular block and the degree of curarization must therefore be very closely monitored in these patients.

Anesthesiologists can use various techniques to reduce the level of pressure in the brain. It should be recognized that in some cases, however, removing the tumour may be the only effective way of creating intracranial space. Logically, sequential adjustments of the various anesthetic parameters should make it possible to determine, to a certain extent, the cause of ICHT or its contributing factors. Still, it is preferable and faster to make a number of adjustments simultaneously.

When dealing with a brain under pressure, the anesthesiologist must first ensure that the anesthetic technique complies with the fundamental principles discussed in the section (Maintaining anesthesia) above. Notably, the patient's position, degree of curarization, saturation, and level of exhaled CO₂ must be quickly checked. The FiO₂ should be increased to 1.0 until the situation is corrected. The patient's blood pressure must be controlled, especially if a serious threat to autoregulation is suspected. In such cases, any significant rise in MAP can lead to an increase in CBF and a tense, hyperemic brain. To keep the CPP from falling too low, care must be taken not to reduce the MAP below 60 to 70 mm Hg.

Nitrous oxide, if used, must be stopped and the flow of fresh gas increased to purge the anesthetic circuit as quickly as possible. The concentration of halogenated agents should be reduced, especially if the dose is 1 MAC or more.³⁷ Alternately, the clinician can stop administering any halogenated agent and use boluses of thiopental or propofol followed by a perfusion. It should be noted, however, that if only a small concentration (0.3 to 0.5 MAC) of isoflurane or sevoflurane was used, this procedure is generally not very helpful. An additional dose of 0.5 to 1.0 g/kg of mannitol can also be administered. This extra dose, while effective, is relatively slow-acting, ie, taking 15 to 30 minutes following administration. If the fluid balance is positive, a dose of 0.1 to 0.2 mg/kg of furosemide can be combined with the mannitol. Finally, the patient's ventilation should be increased to attain a PaCO₂ level of 25 to 30 mm Hg. This should reduce CBF substantially within a few minutes. Steroids are not indicated in the acute phase of ICHT and, moreover, are only effective for treating peritumoural vasogenic edema.

Emergence

The main goal during the neurosurgical patient's emergence from anesthesia is a rapid awakening to enable neurological testing immediately post-surgery, while ensuring proper control of hemodynamics and the airway. No connection between postoperative arterial hypertension and intracranial hematoma formation has ever been formally proven, but since cerebral hemorrhage is a serious postoperative complication, ensuring sound control of blood pressure during this period is recommended. This makes labetalol, at a dose of 0.5 to 1.0 mg/kg, an excellent choice. For the same reason, nausea and vomiting must be prevented. Their reported prevalence exceeds 50% in some sets, especially for posterior fossa craniotomies.³⁸

CONCLUSION

ICHT in the perioperative period is a problem with potentially serious consequences for the patient's prognosis. It can contribute to serious complications such as new neurological abnormalities or even death. It can also be associated with more subtle and insidious problems such as memory loss, difficulty concentrating, and changes in the thought process. To prevent the occurrence of new cerebral damage, the anesthesiologist must be familiar with the physiopathology of ICHT and the impact of anesthetic drugs, high osmotic pressure agents, and hemodynamic interventions on ICP. Although no study on the outcome of patients following intracranial surgery has yet explored the subject, it is reasonable to believe that a careful choice of anesthetic agents will not only help achieve optimal surgical conditions, but also reduce the risk of secondary cerebral damage.

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