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Medical management of increased intracranial pressure after spontaneous intracerebral hemorrhage

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The clinician is frequently faced with the patient who has suffered a spontaneous intracerebral hemorrhage. In this article, we discuss the role of differing modes of intracranial pressure (ICP) management. ICP management can serve as either a primary therapeutic modality or an adjunct to other surgical efforts to control ICP (eg, direct hematoma evacuation). At the heart of all these management strategies is the notion that sustained increased ICP leads to further permanent neurologic injury. Because increased ICP is correlated with an increased volume of intracranial contents, all ICP therapies attempt to reduce the volume occupied by the intracranial contents while preserving maximum tissue perfusion and oxygenation.

Medical management issues

Most patients with intracerebral hemorrhage should be admitted to an intensive care unit for the first 24 to 48 hours for close neurologic observation. All patients with intracerebral hemorrhage should be documented to be receiving adequate blood oxygenation. For many patients, this means placement of an arterial line. This allows for direct measurement of blood oxygenation as well as real-time monitoring of mean arterial pressure. Endotracheal intubation is required for patients who are too somnolent to protect their airways or who demonstrate marginal blood oxygenation through noninvasive means.

It is our practice to elevate the head to 30° and to maintain a neutral position. This is done to maximize venous outflow and to prevent jugular venous compression. This should in turn prevent secondary increases in ICP [1].

Patient comfort should be paramount. Agitation is often associated with unacceptable increases in ICP. Continuous sedative infusion with short-acting agents, such as propofol, may be required to prevent agitation. These agents can be turned off intermittently to examine the patient. Patient/ventilator noncompliance is often the source of the agitation. An intensive search for an appropriate ventilator mode is required.

Hyperventilation can provide rapid control of ICP through cerebral vasoconstriction and decreased cerebral blood flow. Its effect is short lived, however, and should be viewed as a bridging strategy to a more definitive surgical or medical therapy. This is due to homeostatic mechanisms, which adjust to the lowered pH rapidly.

We do not recommend the prophylactic use of hyperventilation. In a randomized trial of hyperventilation in head injury patients, subjects given 5 days of hyperventilation had far worse 6-month outcomes than those patients who did not receive this therapy [2].

Because seizures can be associated with increases in ICP, it is our practice to load all patients with marginal ICP with intravenous phenytoin (20 mg/kg). Adequate levels are maintained until ICP concerns have passed with intravenous phenytoin (3–5/mg/kg/d in divided doses).

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Patient intracranial pressure monitoring strategies

Clinical presentation and volume of hematoma predict outcome in spontaneous intracerebral hemorrhage [3,4]. Further neurologic deterioration is based on a variety of factors, including progressive hematoma growth [3], progression of cell death in perihematoma ischemic regions [5], and secondary hydrocephalus [6,7]. These factors serve to heighten the need for clinical vigilance.

ICP monitoring becomes necessary in those cases in which there are insufficient clinical cues to guide or forewarn of a devastating increase in ICP. It is recognized that large gradients of ICP may exist within or surrounding the hematoma, thus further complicating the technical question of where ICP monitoring should be directed [8]. Additional factors, such as uncorrected coagulopathies, may influence the timing of ICP monitor placement.

In general, patients in whom the Glasgow Outcome Scale score is less than 9 are candidates for invasive ICP monitoring. Of methods currently in use, the two most popular remain direct ventricular catheterization and fiberoptic intraparenchymal monitors. Ventriculostomy affords the opportunity for therapeutic cerebrospinal fluid drainage and is preferred at our institution in patients with intracerebral hemorrhage.

Raised ICP refractory to ventricular drainage may be amenable to additional pharmacologic manipulation as discussed below. Cerebral perfusion pressure (CPP) is maintained between 70 and 110 mm Hg with intravenous phenylephrine delivered through a central venous line.

Hyperosmolar therapies

Hyperosmolar therapies are directed at decreasing brain water. Several agents are available, including mannitol, furosemide, albumin, and hypertonic saline.

As the first step in osmotherapy, we use a 20% mannitol solution intravenously when CPP cannot be met and the ICP is greater than 20 mm Hg [9]. Mannitol is typically administered at a dose of 1 g/kg. Reductions in ICP are usually realized within 30 minutes. Repeat doses of mannitol (0.25–0.5 g/kg) can be infused every 6 to 8 hours to maintain a serum osmolarity greater than 308 mOsm/L. Because of the significant diuresis that is associated with mannitol, adequate hydration with hyperosmolar fluids (ie, normal saline or normal saline with 5% dextrose) is important. We target a central venous pressure of approximately 10 mm Hg. Dehydration can lead to hypotension and subsequent difficulty maintaining an adequate CPP.

It is our practice to wean mannitol over a period of several days after initiation of such therapy to guard against the theoretic possibility of rebound increases of ICP after its abrupt discontinuation [10].

Because of issues of mannitol failure and rebound increases in ICP, investigators have also considered new osmolar agents, including hypertonic saline [11–23]. We have begun to use intermittent 30-mL bolus doses of 23.4% saline for refractory ICP [23]. Such treatments can decrease increased ICP to acceptable limits for up to 15 hours in our hands.

Barbiturate coma

Proceeding up the ladder of available therapies, barbiturates can be employed in cases of refractory ICP. These medications suppress neuronal metabolic activity in a rapid fashion. We begin this therapy with an intravenous bolus of pentobarbital of 3 to 7 mg/kg. This can then be continued with intermittent boluses of 0.5 to 2 mg/kg every 15 to 30 minutes or by a constant infusion of 1 to 5 mg/kg/h. Barbiturates are associated with ileus and hypotension and respiratory depression; as such, they require mandatory ventilation and pressor support. Barbiturate therapy can also mask fever and increase the risk of nosocomial infections escaping clinical detection. Heightened surveillance for infections is warranted under these conditions.

Emerging therapies

The most recent step on the ladder of therapies for ICP control is moderate hypothermia. Because fever is associated with unacceptable increases in ICP, standard management of intracerebral hemorrhage dictates that normothermia be maintained with acetaminophen and cooling blankets.

Possible benefits of achieving more favorable ICP when temperatures are kept below normothermia (32°C–33°C) with cooling blankets date back to the early part of the last decade [24]. This study made note of problems that continue to make such treatments difficult, including cardiac irritability and rebound increases in ICP during the rewarming period.

Although there remains good evidence that hypothermia can indeed lower ICP in some situations [25], it is less clear whether such a strategy

leads to improved outcome. In 2001, a randomized multicenter trial reported no effect on clinical outcome in the subject population randomized to 48 hours of moderate hypothermia within 6 hours of injury [26].

Research in this field continues. When a cohort of 50 patients with middle cerebral artery infarction and ICP monitoring was studied for ischemic stroke, it was shown that cooling reduced mean ICP by 7 mm Hg [27] Rewarming was associated with an increase in ICP in all treated patients, however, and in the patients who died, the mechanism of death was usually herniation during the rewarming period.

Summary

There are several medical therapies available to lower unacceptable ICP. We advocate the stepwise institution of these therapies to maintain adequate CPP. At every step in the process, consideration of definitive surgical intervention (eg, hemicraniectomy, clot evacuation) should be entertained.

At this time, we cannot recommend hypothermia as a routine last step of therapy given the complications and lack of clinical effect described previously. Research into this therapy continues, however. The next several years may show us when, how, and in what situations this strategy can be applied.

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