Inside the rigid vault of the bony skull, there are three components: brain tissue (cells and water), blood and cerebrospinal fluid (CSF).

An increase in any one of the three can cause increased pressure inside the brain, compressing brain tissue and restricting blood flow. A focal collection of blood such as a subdural hematoma or localized swelling (such as with ischemic or hemorrhagic stroke) will create a mass effect, pushing the rest of the brain to the side and/or downward. Or diffuse cerebral edema from a severe closed head injury or metabolic insult may cause downward herniation. Or a clot from a subarachnoid or intraventricular hemorrhage can obstruct the third or fourth ventricles, causing acute hydrocephalus.

The brain can compensate a little by displacing CSF into the spinal cord and collapsing the sulci and ventricles (except in obstructive hydrocephalus), but eventually the mass effect may become severe, creating shifting of brain structures and potentially severe neurologic injury or even death. This is called secondary injury, and efforts to prevent it are the focus of neurocritical care in these patients.

Serial neurologic examinations may detect progressive dysfunction associated with cerebral edema, but in critically ill patients who are on ventilators and sedated, the neurologic exam is less sensitive until serious dysfunction or damage has occurred. In these cases, monitoring of intracranial pressure may detect intracranial hypertension (ICH) so that immediate measures can be taken to reduce it.

Considerable debate exists as to the benefit of intracranial pressure (ICP) monitoring. A Cochrane database review found no randomized controlled studies comparing outcomes in patients who had ICP monitoring vs. those who didn’t, and so concluded there are no data to clarify the use of the monitoring in acute coma. However, ICP monitoring in the setting of Glasgow Coma Scores of less than 9 and in intubated, sedated ICU patients with known neurologic injury is common.

There are four approaches to ICP monitoring: intraparenchymal sensor, placed directly into brain tissue; epidural bolt, placed just below the skull in the epidural space; subarachnoid screw, placed into the subarachnoid space just above the cerebral cortex; and ventriculostomy, placed into one of the lateral ventricles. The ventriculostomy allows CSF to be drained or sampled. However, if the ventricles are distorted or collapsed, the sensor, screw or bolt may be necessary.

These monitors can be placed at bedside or in the operating room, usually by a neurosurgeon. They are then attached to transducers to allow projection of the CSF waveform, similar in appearance to that of the right or left atrium, onto the monitor screen.

Normal ICP is 1 to 15 mm Hg (to convert to cm H2O, multiply by 1.3). ICH is defined as ICP >20 mm Hg (26 cm H2O). Sustained ICH is ICP >20 mm Hg for >5 minutes. The true impact of ICH is currently thought to be on cerebral perfusion pressure (CPP). As ICP rises, blood volume inside the cranium falls, presumably decreasing blood flow. In the injured brain, autoregulation may be disrupted, and cerebral perfusion becomes dependent upon cerebral blood flow. CPP is thought to be an adequate representation of blood flow and brain perfusion. It is calculated as mean arterial pressure (MAP) − ICP = CPP. CPP-targeted therapy is still a topic of debate, but popular protocols have demonstrated improved neurologic outcomes with CPP of 60 to 70 mm Hg.

Clinically detectable, ventriculostomy-induced intracranial hematoma occurs in less than 2% of patients, although routine head CT scans after placement find clinical silent hematomas. Ventriculostomies and parenchymal monitors carry higher risks of infection than the less invasive bolts and screws. Any ICP monitoring device is subject to clotting, accidental malpositioning, collapse, and inaccurate readings due to abnormal respiratory pattern, excessively high PEEP or transducer or system failure. Observer error can be problematic, if care is not taken regarding patient and transducer positioning. If a patient’s ventriculostomy is open to drainage, and the patient’s head is raised or lowered, excessive CSF drainage or return may create catastrophic neurologic decompensation.

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