Chapter 11
Intracranial Pressure Management

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11.1 Introduction

Because the skull is a closed, non-compressible vault, constant equilibrium must be maintained between the skull contents, including brain tissue, cerebrospinal fluid (CSF) and blood. The Monro-Kellie hypothesis states that an increase in one of these components must be accompanied by a relative volume adjustment. The brain has a dynamic capacity for self-protection and compensation in the early stages of an injury including autoregulation, restriction of blood flow, decreased production and shunting of CSF. However, when a critical volume change has occurred, compensation is lost and an elevated intracranial pressure (ICP) is the result. Sustained intracranial hypertension is defined as ICP >20 mmHg for >5 min [1–3]. Cerebral

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perfusion pressure (CPP) is an estimate of the cerebral blood flow and is calculated by subtracting ICP from the mean arterial pressure (MAP) or CPP=MAP − ICP. The normal adult value is a range of 70–100 mm Hg. Data from several studies support a CPP goal of >50–60 mm Hg to prevent cerebral ischemia in traumatic brain injury patients [4]. Interventions to modulate blood pressure may be used to increase or decrease CPP as necessary.

Compliance is a measure of the adaptive capacity of the brain to maintain intracranial equilibrium in response to physiological and external challenges to the system. Compliance represents the ratio of change in volume to change in pressure ($C = \Delta V/\Delta P$) [5]. When compensatory mechanisms are intact, there is adequate volume shift intracranially leading to normal ICP measurements or adequate compliance. When compensatory mechanisms are overwhelmed, ICP may rise quickly because of poor compliance. Compliance can be lost with even the smallest volume shifts depending on the patient's underlying pathology (See Fig. 11.1).

Cerebral edema is a common cause of elevated ICP and is classified in two categories: vasogenic and cytotoxic. Vasogenic cerebral edema is characterized by an increase of fluid in the extracellular space and occurs when there is a breakdown in the blood brain barrier. Trauma, brain tumors, infection, hemorrhage and surgical procedures are common causes. Cytotoxic cerebral edema is characterized by an excess of fluid within the intracellular space. Sodium and water shift into the cell, causing swelling and, eventually, cell death [1, 2, 5]. This type of cerebral edema is typically seen in acute hypoxic injury, cerebral ischemia, and hypo-osmolality, but can also occur in trauma, inflammation, and hemorrhage. Both types of cerebral edema, if not managed effectively, can lead to herniation syndromes (see Table 11.1).

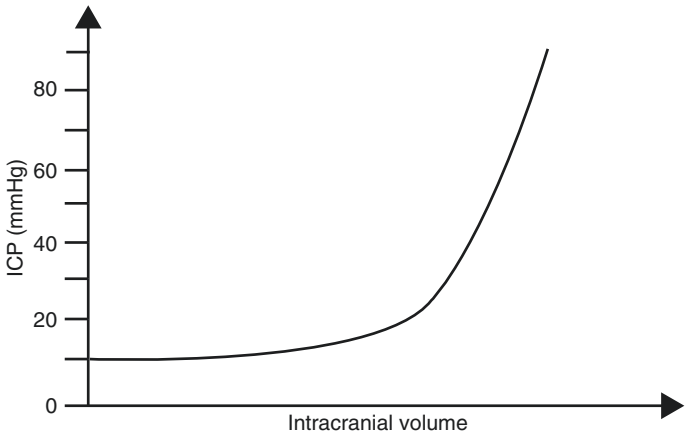

Fig. 11.1 Intracranial compliance curve. Initial increases in intracranial volume are well tolerated. Continued increase in intracranial volume results in loss of compliance and rapid elevations in ICP. Sustained increases can lead to secondary brain injury and herniation syndromes (No re-print premission needed)

Table 11.1 Signs of increased ICP

Changes in level of consciousness:
Confusion
Lethargy
Agitation
Changes in vital signs:
Hypertension
Bradycardia
Widening pulse pressure
Irregular respiratory pattern
Headache
Visual disturbances
Nausea and vomiting
Pupillary changes
Hemiplegia/hemiparesis

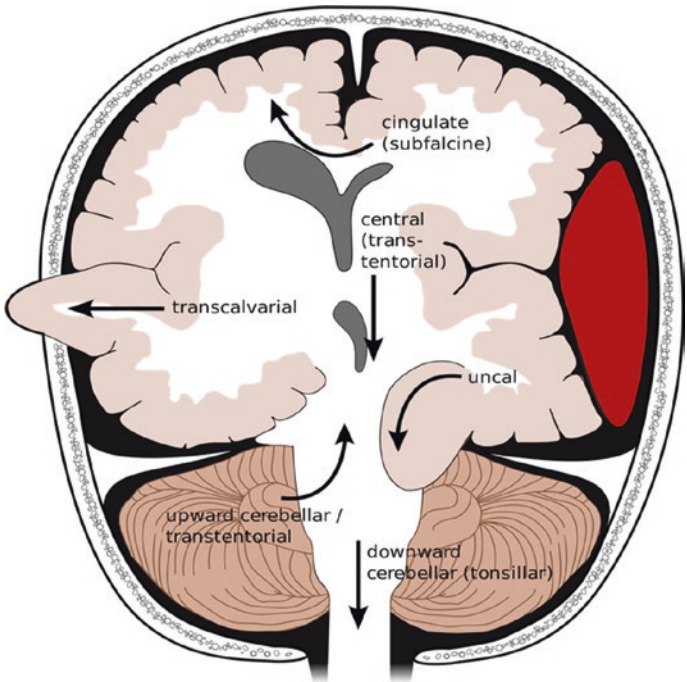

Fig. 11.2 Herniation Syndromes. The brain tissue herniates from an area of increased pressure to an area of lesser pressure causing clinical exam changes leading to poor outcomes if not treated immediately. The different types of herniation syndromes are illustrated below (Retrieved from Wikimedia Commons, a free medical repository. No re-print permission needed)

Herniation syndromes include uncal, transtentorial, subfalcine, and cerebellar [5] (See Fig. 11.2). Each is related to mass effect and compartmental pressure changes because of the rigid intracranial boundaries. The brain tissue herniates from an area of increased pressure to an area of lesser pressure causing

clinical exam changes leading to poor outcomes if not treated immediately. Patients with herniation syndrome will present with progressive somnolence, decreased respirations and Cushing's Triad of symptoms: sudden hypertension, bradycardia, and respiratory irregularities.

11.2 Case Presentation

A 23-year-old male with no past medical history was brought to the emergency department following a motor vehicle collision (MVC). He was an unrestrained driver T-boned by another vehicle at approximately 50 mph. Extrication from the vehicle was required. He was unresponsive in the field and immediately intubated upon arrival of EMS in the field for airway protection. Trauma scans revealed a right tentorial subdural hematoma with mild mass effect, a right pontine/midbrain intraparenchymal hemorrhage, fracture of the right occipital condyle, and fracture of the eighth tooth. No other injuries were identified. An external ventricular drain (EVD) and intraparenchymal ICP monitor, temperature, and brain tissue oxygen probes via a triple lumen intracranial bolt were placed. He was admitted to the Neuro ICU in hemodynamically stable condition. Proper positioning, sedation, analgesia, and seizure prophylaxis were immediately implemented. Because of ICP elevations to 55 mmHg sedation was increased; a paralytic administered, and 3% hypertonic saline was started. The patient subsequently had a temperature spike and surface cooling with a goal of normothermia was initiated. Acetaminophen was given and empiric antibiotics began to prevent further fever. The paralytic was gradually weaned off. Over the course of a week, his ICP was maintained <20 mmHg and the intracranial bolt was removed. EVD was removed on hospital day 13.

11.3 Initial Evaluation

It is the role of the neurocritical care clinician to recognize physiologic signs of elevated ICP and take any necessary steps to prevent secondary brain injury and herniation. Initial evaluation should include baseline vital signs and a complete neurologic exam. Symptoms to be evaluated include changes in vital signs, mental status, and pupil size; in addition to headache, visual disturbances, vomiting and motor function abnormalities (see Table 11.2). If there is sudden or unexplained ICP elevation, the patient must be examined immediately. Interventions to lower the ICP must be initiated urgently prior to any diagnostic studies. Head CT should be repeated to rule out new mass lesion that may require surgical evaluation such as new subdural hematoma, epidural hematoma, or hemorrhagic transformation of stroke.

Most ICP lowering therapies are effective for variable amounts of time and early management goals should include placement of an ICP monitoring device. The purpose of ICP monitoring is to improve the clinician's ability to maintain

Table 11.2 Common causes of increased ICP

Primary causes
Edema
Trauma
Brain tumors
Infection/inflammation
Hemorrhage
Ischemic injury/infarction
Hypoxic injury
Hydrocephalus
Secondary causes
Agitation
Hypertension
Seizures
Fever

adequate cerebral perfusion pressure and oxygenation to the brain. The diagnosis of elevated ICP is made from clinical findings based on the patient's exam, imaging, and past medical history.

Any patient that is suspected to be at risk for elevated ICP should be considered for placement of an ICP monitoring device. There are four common sites used for ICP measurements; intraventricular, intraparenchymal, subarachnoid, and epidural. Intraventricular monitors are the gold standard of ICP monitoring catheters. They are placed through the skull into the ventricular system and attached to a pressure transducer with collection bag (See Fig. 11.3 for example). The major advantage of an intraventricular system is that CSF can be drained. Disadvantages are infection and potential hemorrhage during placement. Intraparenchymal devices are built around a thin cable with a fiber optic transducer at the tip. These devices are inserted through the skull directly into the brain parenchyma. Their main disadvantage is the lack of ability to drain CSF; advantages include ease of placement and lower risk of infection. Subarachnoid bolts are fluid filled systems within a hollow screw that are placed through the skull adjacent to the dura. The dura is punctured and the CSF communicates with the fluid column and the transducer. Advantages of subarachnoid monitors are low rates of infection and hemorrhage. The major disadvantage is frequent clogging of the system, which renders the measurements unreliable. Epidural ICP monitors contain and optical transducer that rests against the dura once passed through the skull. They are often inaccurate and have limited use clinically.

Once ICP monitors are in place, the waveforms can be easily accessed to help the clinician evaluate intracranial compliance. There are three peaks to the waves that are referenced for clinical significance. P1 is the percussion wave, which reflects arterial pulsation. P2 (tidal wave) represents intracranial compliance and P3 is the dicrotic wave signaling closure of the aortic valve

Fig. 11.3 Extraventricular drain (EVD). This is a temporary method to reduce ICP that can be regulated manually. The drain is attached to a bag that is transduced to atmospheric pressure and leveled to the midbrain. The drain can then be raised or lowered to different anatomical reference points to facilitate drainage (Photo courtesy of University of Pittsburgh Medical Center)

[1, 2]. Normal intracranial compliance is depicted by a sharp, high P1, followed by a P2 that is lower than P1, followed by an even lower P3 wave. Poor intracranial compliance is seen as a P2 wave that is equal to or higher than that of P1 (See Fig. 11.4).

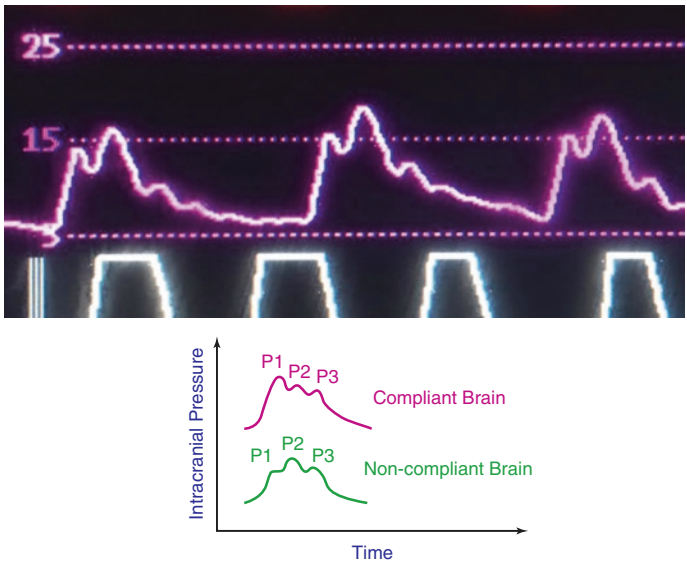

Fig. 11.4 ICP Waveforms. Normal intracranial waveforms depicted by a high P1, followed by a P2 that is lower than P1 followed by an even lower P3 wave. Poor intracranial compliance is seen as a P2 wave that is equal to or higher than that of P1 (Photo courtesy of University of Pittsburgh Medical Center)

11.4 Management and Interventions

There are several ICP management options used in neurocritical care. A step-wise approach to treatment is suggested and consists of some simple techniques such as positioning maneuvers to more complicated treatments for refractory patients (see Table 11.3). Many of the treatments currently in use are outlined in the following pages.

Table 11.3 Stepwise treatment of increased ICP

Positioning
Sedation/analgesia
Normothermia
CSF diversion
Hyperventilation
Osmotherapy (may repeat treatments)
Hypothermia
Pentobarbital Induced coma
Decompressive hemicraniectomy

11.4.1 Head Elevation

One of the easiest interventions to help manage ICP is proper positioning of the patient. The head should be positioned forward and straight without the neck being flexed backwards or forwards. This promotes adequate perfusion and drainage of the cerebral vessels. Head of bed at a maximum of 30° is acceptable to maintain adequate CPP. Special care should also be taken if a cervical collar is in place. The collar should be properly positioned and should not be so tight as to restrict venous outflow.

Flat positioning is acceptable for patients who do not have a large abdominal girth, but because of the risk of aspiration or gastric reflux, reverse Trendelenburg could be considered. For those with a large abdomen, head of bed should be raised slightly to decrease intra-abdominal pressure. Trendelenburg is always contraindicated because of obstruction of venous return, which increases cerebral blood volume and in turn increases ICP. In general, proning and extreme hip flexion are also discouraged, as they will also increase abdominal pressure and raise ICP. However, in a patient with ARDS where proning may be a life saving measure, it can be done if necessary. There should be close teamwork and communication with neurosurgical colleagues in this situation.

11.4.2 Sedation and Analgesia

Adequate sedation and pain control are important early interventions for ICP management. Sedation can decrease cerebral oxygen demand in addition to controlling a multitude of other factors that elevate ICP. Restlessness, agitation, coughing, and ventilator asynchrony can all be prevented or minimized with adequate pharmacological support. This is best achieved with short acting narcotics for analgesia and propofol or similar short acting agents for sedation [6]. These drugs are ideal for interruption for neurologic exams because of their short half-lives (See Chap. 22 for information on commonly used sedation and analgesia agents).

11.4.3 Temperature Control

Fever increases cerebral oxygen demand and may contribute to cerebral ischemia and elevated ICP. Cerebral blood flow and cerebral metabolic rate increase about 5% for every 1 °C temperature increase [5]. Cooling blankets and acetaminophen can be instituted quickly and easily. The goal is for normothermia (37.0 °C) to be maintained throughout the patient's course.

Hypothermia is reserved for refractory cases of elevated ICP. If core temperature remains uncontrolled, surface cooling and cooling catheters may be necessary. Hypothermia lowers ICP but there is no evidence that it improves outcomes. Moderate hypothermia with a target core temperature of 32–34 °C is associated with a predictable reduction in ICP [4].

11.4.4 Cerebrospinal Fluid Diversion

Insertion of extra ventricular drains (EVD) allows CSF to be displaced and can facilitate treatment of elevated ICP related to

hydrocephalus. The EVD catheter itself gives the clinician direct ICP measurements. As the fluid is removed, ICP decreases and creates more room inside the cranial vault. This is a temporary method to reduce ICP that can be regulated manually. The drain is attached to a bag that is transduced to atmospheric pressure and leveled to the midbrain. The drain can then be raised or lowered to different anatomical reference points to facilitate drainage (see Fig. 11.3).

11.4.5 Osmotherapy

Osmotherapy decreases the amount of cerebral edema by creating an osmotic gradient across the blood-brain barrier and facilitating the movement of free water out of the cerebral tissue. There are a few different osmotherapy options available. Mannitol is an osmotic diuretic that pulls free water from the brain, decreasing brain volume and lowering ICP. Mannitol works quickly and lowers ICP within minutes. It should be given as a single bolus dose of mannitol 20% 1 gm/kg rapid IV infusion. There is large volume diuresis seen with mannitol administration; consequently, accurate documentation of intake and output is essential. Measures should be taken to avoid systemic hypovolemia when this agent is used. Additionally, impaired renal function reduces the utility of this medication and patient can develop acute kidney injury with prolonged use.

Hypertonic saline (HTS) is also a well-recognized form of osmotherapy to help control ICP. HTS has minimal side effects and has been shown to produce a significant reduction in ICP when compared to mannitol [7]. However, there is a lack of consensus over optimal concentration and length of use. Current available concentrations are 1.5%, and 3%, given as a bolus dose followed by continuous infusion, or 7.5% and 23.4% given as bolus doses up to every 6 h. Serum sodium, electrolytes and osmolality should be checked routinely while the patient is

receiving osmotherapy [8–12]. See Chap. 22 for osmotherapy administration and monitoring information.

11.4.6 Hyperventilation

Carbon dioxide is a known cerebral vasodilator. Lowering CO_2 can cause a rapid vasoconstriction of cerebral blood vessels causing a decreased cerebral blood flow and leading to a decrease in ICP. The goal is a $PaCO_2$ of 32–36 mm Hg and can be achieved by increasing the ventilator rate for an intubated patient. An ambu bag can be used to hyperventilate the non-intubated patient. While this works in acute episodes of intracranial hypertension, the effectiveness is transient in nature. Prolonged hypocarbia may reduce cerebral blood flow to the point of ischemia so careful monitoring with frequent blood gas sampling or end tidal CO_2 detector is necessary and hyperventilation should only be used as a bridging measure toward alternative therapies.

11.4.7 Pentobarbital

Continuous pentobarbital infusion should be considered in patients with ICP elevations refractory to treatment with osmotherapy, hyperventilation and sedation. The mechanism of action of pentobarbital is a profound reduction of cerebral metabolic rate. Pentobarbital should be given as a bolus dose of 10 mg/kg. If this initial dose results in an ICP reduction, then a continuous infusion can be started. The initial rate is usually at 1 mg/kg per hour, but can be titrated upward as needed [13]. Continuous EEG should be applied to these patients to monitor brain activity and watch for burst suppression. The goal of pentobarbital infusions is a burst-suppression pattern on EEG consisting of a 1–2 s burst of activity followed by 8–10 s of suppression. Titration of pentobarbital above the level to produce this pattern should be undertaken with

extreme caution, and only if a decrease in the ICP occurs in response to upward titrations. Hemodynamic support may also be required as the most common side effect is hypotension.

11.4.8 Decompressive Hemicraniectomy (DHC)

DHC can be performed when other methods of ICP reduction fail, or the area of injury is expected to swell and herniation syndromes are anticipated. Part of the skull is removed, the dura is opened, and the skin is closed to allow the brain additional room outside of the cranial vault. Cranial skull flaps are typically replaced weeks to months later. Management after DHC consists of aggressive medical care to optimize patient outcomes and avoid complications such as respiratory failure leading to tracheostomy and DVT. Some institutions have incorporated DHC into ICP treatment algorithms as an early intervention although there is no strong evidence supporting this practice [14].

11.4.9 Additional Therapies

11.4.9.1 Paralysis

Paralytics, or neuromuscular blockade, can be used in addition to sedation to help control elevated ICP. Vecuronium and cisatracurium are common agents. All patients with neuromuscular blockade on board should have adequate sedation and must be monitored for appropriate level of blockade to be achieved. Side effects can be minimized by carefully monitoring the dosage and degree of blockade attained with a train-of-four peripheral nerve stimulator applied to the ulnar or facial nerve. Because of the neuromuscular blockade, seizure activity would be undetectable; therefore, it is helpful to have continuous EEG monitoring in place during paralysis.

11.4.9.2 Steroids

Patients with vasogenic edema in the non-traumatic population are often started on high dose corticosteroid therapy, such as dexamethasone, to help decrease intracranial pressure. Steroids have *not* been shown to be effective for cytotoxic cerebral edema, and are contraindicated in traumatic brain injury [15]. Consideration must be made for blood glucose levels and subsequent initiation of insulin therapy to maintain normoglycemia.

11.4.9.3 Seizure Control

Seizures cause elevated ICP by increasing the cerebral metabolic rate and oxygen consumption. Patients with elevated ICP from brain injury are prone to seizures, which may further elevate ICP. There should be a low threshold for either routine or continuous EEG monitoring. Any seizure activity should be treated aggressively with antiseizure medications [16].

Summary Points

- The skull is a closed, non-compressible vault. The Monroe-Kelli Hypothesis states that an increase in one of the three internal components of the skull (brain, blood or CSF) should be followed by a relative decrease in one of the other components. Compliance is the adaptability of the brain. When compliance is lost an increase in ICP may result.
- Frequent neurological and vital sign assessments can help clinicians detect early changes in ICP and lead to earlier treatment.
- Treatments of ICP can consist of simple techniques such as positioning maneuvers to more complicated treatments like pentobarbital for refractory patients. Management should be done in an organized step-wise approach.

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