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INTRACRANIAL PRESSURE MONITORING

A HANDBOOK FOR THE NURSING PROFESSIONAL

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Why do we care about measuring intracranial pressure (ICP)?

The intracranial compartment is very different from other regions of the body. A boxer who is punched near his eye may experience bruising and swelling in that area. If the blow knocks the boxer unconscious, a similar swelling may occur within the brain. Unlike the soft tissue surrounding the eye, the adult brain is enclosed within a rigid compartment, the skull, that does not allow for outward displacement of swollen tissue. Instead, swelling manifests as an increase in pressure within the intracranial compartment. If the brain swelling is localized, the brain contents can shift towards the unswollen side. This shift can cause distortion and dysfunction of brain nerve pathways, and if severe enough, herniation (a pathological displacement of brain tissue). If the ICP level approaches the mean arterial blood pressure, an impairment of cerebral blood flow will occur—a scenario nearly always associated with death of the brain and thus the patient. Most major studies of patients suffering traumatic brain injury have arrived at the same conclusion: a sustained increase in ICP above 20 mm Hg is a strong predictor of poor outcome. Since the importance of elevated ICP was recognized about three decades ago, a change in management of head-injured patients aimed at keeping ICP below 20 mm Hg has improved the chances of many.

How do we know if ICP is elevated? Excluding infants with an open fontanelle, there is no reliable and continuous method of estimating ICP without actually measuring it invasively (inserting a monitor beneath the skull or into the spinal canal). When, why, and how to measure ICP is still a matter of some debate. However, if one understands the fundamentals of ICP physiology and measuring technology, the caregivers of brain-injured patients can manage a variety of conditions in a logical and straightforward manner.

The fundamentals of ICP physiology

For the sake of simplicity, it is useful to think of the intracranial compartment as containing only three components: brain, blood, and cerebrospinal fluid (CSF). Since each of these components is made up of primarily water, none are compressible (although each is displaceable). Due to the rigid skull enclosure, an increase in volume of any one of these components must be accompanied by a reduction in the volume of one of the others if the ICP is to remain constant. For example, an expanding blood clot within the brain will primarily displace CSF out of the intracranial compartment. Once the CSF volume buffering mechanism has been exhausted, further increases in the size of the blood clot will typically be associated with an increase in ICP. (See Fig. 1 for Pressure Volume Curve).

Hypothetical pressure-volume relationship given a "normal" state of compliance. "A" indicates additional volume has minimal impact on ICP. "B" indicates small additional volume increases ICP dramatically.

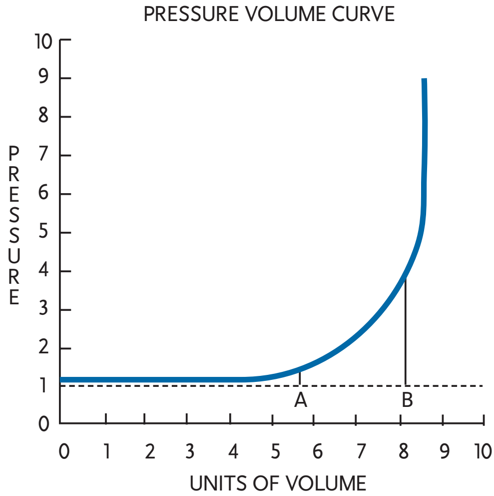

Fig. 1

In order to discuss the mechanics and management of elevated ICP, it is important to have some idea what a “normal” ICP is. Three of the main determinants are age, body weight, and body position. When lying flat, the ICP of an infant is 1-2 mm Hg; an average adult 5-7 mm Hg, and an obese adult 12-18 mm Hg. In adults, rising to the upright position results in a drop of about 7–10 mm Hg; or in other words, the ICP becomes subatmospheric (-3 to -5 mm Hg for an average adult). ICP can transiently increase to very high levels (60-80 mm Hg) with coughing, bowel movements, and during sexual intercourse. Therefore, “elevated” ICP must be interpreted in light of these factors. In general, only sustained (>5 minute) elevations in ICP levels more than 15 mm Hg above the expected baseline are considered abnormal. In practical terms, this translates as ICP elevations above 20 mm Hg in most cases.

What conditions are associated with elevated ICP?

Any condition that causes brain swelling, is associated with an intracranial mass lesion, or obstructs CSF movement can cause an elevation in ICP. In the intensive care unit (ICU), the most common diagnosis requiring ICP monitoring is traumatic brain injury. In the acute stage, traumatic brain injury is frequently complicated by expanding hematomas, progressive brain swelling, and sometimes hydrocephalus. Because these patients are often in a coma and have a limited neurological exam to base treatment decisions upon, ICP monitoring can provide critical information for patient management. Other acute conditions that can result in an elevated ICP include subarachnoid hemorrhage, spontaneous intracranial hematomas, and intraventricular hemorrhage.

In some institutions, ICP monitoring is also used to aid in the management of disorders causing massive brain swelling and coma. Some patients with fulminant hepatic failure have extremely elevated ICP values, and therefore are not candidates for transplantation. Because there is no other reliable method, including CT scans, to determine the severity of brain swelling in these patients, ICP monitoring is utilized.

What ICP monitoring tells the caregiver

ICP monitoring can provide valuable information for a more complete assessment of the patient's neurological status and allows for prompt intervention with life-saving treatment. In some situations, ICP monitoring provides data when clinical examination is impossible, such as when the patient is in barbiturate coma or chemically paralyzed. When basic assessment parameters are limited or absent, an elevation in ICP can alert the caregiver to possible changes in cerebral blood flow, expansion of hematomas, or worsening brain swelling.

In some situations, the state of cerebral blood flow (cerebral perfusion) can be implied by the interpretation of ICP measurements. This is based on the observation that ICP and intracranial venous blood pressure are nearly the same. From basic fluid mechanics, we know that blood flows from high pressure (arterial) to low pressure (venous). If the resistance to blood flow is kept constant, a larger difference between the arterial and venous blood pressure will result in more cerebral blood flow. Since ICP can be substituted for venous pressure, the cerebral perfusion pressure (CPP) can be estimated by subtracting the ICP from the mean arterial pressure (MAP): CPP = MAP-ICP. It is important to realize that CPP **cannot** be used to predict the level of cerebral blood flow in many situations. We do know that if CPP is very low (< 50 mm Hg), then it is very likely that there is an inadequate level of cerebral blood flow. In general, a CPP greater than 60-70 mm Hg is desired. The adequacy of cerebral blood flow in many situations must be measured by a different method, such as by jugular bulb venous oximetry.

An acute elevation in ICP can alert the caregiver to an increase in the size of an intracranial mass lesion, or to the onset of acute hydrocephalus. In many cases, an urgent CT scan is obtained in order to determine whether there is a situation that requires surgical intervention. As noted previously, ICP monitoring can be used prognostically in cases of fulminant hepatic failure to determine whether the patient is a viable recipient. In these cases, a low CPP is diagnostic.

In specialized cases, ICP monitoring can be used to aid in the management of complex hydrocephalus cases. The shunt used to treat this condition creates ICP values that are nonphysiologic. For hydrocephalus patients with chronic headaches, ICP monitoring can be the only way to distinguish under- and over-shunting conditions.

ICP Waveforms

The normal ICP waveform shape has three peaks within the cardiac cycle. The first peak (P1) is called the percussion wave, P2 is the tidal wave and P3 is the dicrotic wave. (Fig. 2). The intracranial waveform resembles that of the arterial line.

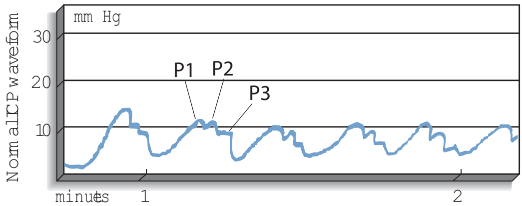

Fig. 2

In addition to the shape of the ICP waveform, longer term trends of ICP may have clinical significance. Three ICP waveforms are commonly referred to clinically.

Lundberg "A" waves (Fig. 3), also called plateau waves, are sustained elevations in ICP (often 20–50 mm Hg) lasting from 5 to 20 minutes. If the waves are prolonged or frequently recur, this signifies an intracranial compartment with a critically low compliance. There may be an association between the presence of "A" waves and a deteriorating clinical condition in come cases.

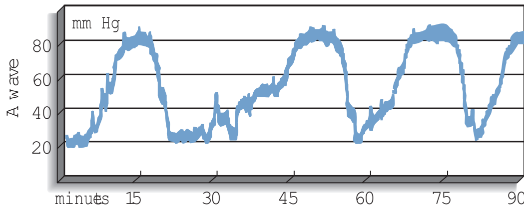

Fig. 3

"B" waves (Fig. 4) occur with a period of 1-1/2 to 2 minutes and are of lower magnitude (although they can reach elevations of 50 mm Hg). "B" waves appear to be related to respirations and may precede "A" waves. They may be an early indication of intracranial decompensation and their frequency may increase as compliance decreases.

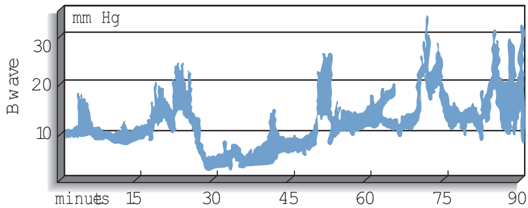

Fig. 4

“C” waves (Fig. 5) are related to systemic pulse fluctuations and appear to be rapid and rhythmic. “C” waves are not thought to be clinically significant.

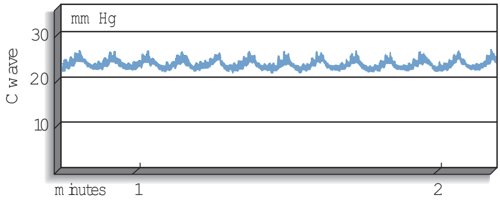

Fig. 5

Types of ICP monitors

In general, there are two types of ICP monitors. The traditional and oldest involves inserting a catheter into the lateral ventricle of the brain (commonly known as a ventriculostomy). A continuous fluid conduit is established between the ventricle and a pressure transducer (a device that converts pressure into electrical current) placed at the bedside for the measurement of the fluid pressure. Continuity of this fluid couple is mandatory in order to derive an accurate measurement of ICP. In a fashion similar to the measurement of blood pressure from an arterial catheter, the level of the transducer relative to the patient must be known in order to interpret the pressure measurement. By convention, ventriculostomy transducers are placed at a point level with the center of the ventricle system (foramen of Monro). From a nursing perspective, the tragus of the ear is a good estimation of this point.

The advantages of a ventriculostomy catheter is that it is conceptually simple, the transducer can be “re-zeroed” at any time, the catheter is relatively inexpensive, and elevations in ICP can be treated by releasing CSF. The primary disadvantages are that access to the ventricular system is not possible in all cases, the technique requires a puncture through the brain (a problem in coagulopathic patients), and occlusion of the catheter can prevent the accurate measurement of ICP.

The second basic type of ICP monitor has the transducer located at the tip of the catheter. The miniaturised transducer mechanism responds directly to pressure changes in the tissue it touches. As a consequence, this type of catheter does not require a fluid-coupled interface. There are various technologies that have been utilised for these transducers, some even utilising laser beams and fiberoptic cables. Several manufacturers offer small electronic circuit boards that are sensitive to pressure, thereby providing a much more durable system (wire not being as breakable as fiberoptics). In general, the advantages of distal transducer systems address the disadvantages of the fluid-coupled ventriculostomy system. The transducer tip can be placed anywhere beneath the skull, including the epidural, subdural, intraparenchymal, and ventricular locations. Most of the transducers are small and fairly flexible, therefore only minimally traumatising the brain. The main disadvantage is calibration drift since this type of transducer cannot be “re-zeroed.” Some systems have more drift than others, and the caregiver should be aware of the manufacturer's specifications because errors of more than 5 mm Hg can occur is some cases. If one understands the basic principles of ICP measurement, then it is usually easy to adapt to any given ICP monitor used at your institution. Recently, hybrid ICP monitors have become available that allow both drainage of CSF and measurement of ICP at the distal tip of the catheter.

Ventricular Drainage CSF Collection Systems

For ventriculostomy catheters, CSF drainage must be done in a controlled manner. The elevation at which CSF drains, however, is relative to the level of the transducer. Most external ventricular drainage (EVD) systems incorporate a graduated CSF collection cylinder (see example in Fig. 6). Beginning at the ventricle, the top of this cylinder is the terminus of the fluid couple. If the cylinder is raised above the transducer level, drainage of CSF will only occur if the ICP is greater than the fluid pressure achieved by the vertical column of CSF. Many physicians will write an order for CSF drainage "at 15 cm." This translates as placing the top of the cylinder 15 cm higher than the transducer level. All EVD systems incorporate a ruler or tape measure in order to take this measurement.

Ventricular Drainage

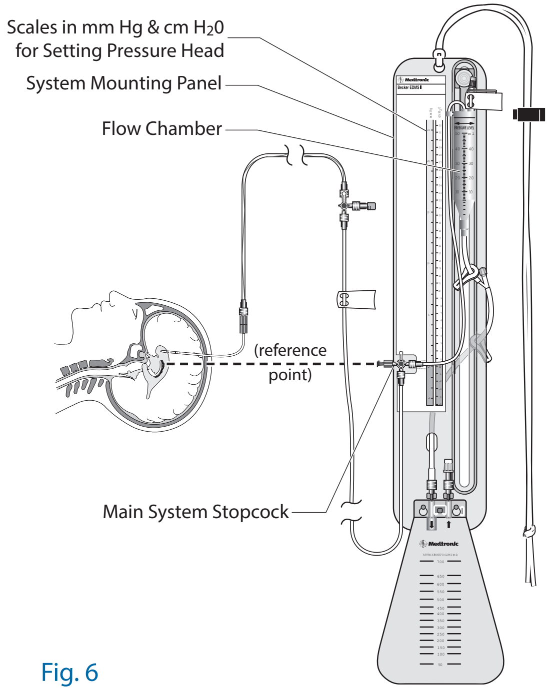

Fig. 6

Preparation of the EVD requires strict aseptic technique. This preparation should occur immediately prior to the insertion of the ventricular catheter. The system mounting panel can be hung on an IV pole using the adjustable slide clamp and cord, or fixed to the IV pole using the pole clamp. The system is then pre-filled with preservative-free sterile normal saline. A 50 mL IV bag of normal saline is the best source of fluid. The graduated flow chamber is adjusted to the proper pressure setting based upon the physician's orders. All connections should be checked to assure that the fittings are tight and leak-free.

When using an external transducer, the system must be cleared of all air bubbles since they will alter pressure readings. A dampened waveform is suggestive of an air bubble or occlusion of the catheter tip. Air is best eliminated by clearing the system with preservative-free normal saline or by withdrawing air from the monitoring line. Prior to performing either of these maneuvers, care must be taken to assure that the system is closed to the patient in order to prevent instillation of normal saline into the ventricle or to prevent any suction on the brain tissue. If the catheter is occluded with blood or debris or by position against the ventricular wall, it may be irrigated with 0.1 mL of preservative-free normal saline. This is usually done by the physician but may also be performed by a health care provider who has been credentialled to do so.

A pressure transducer may be attached to the patient line or to the main system stopcock (see Fig. 7). The main system stopcock should be leveled at a predetermined anatomical location. (Landmarks used include the top of the ear, the tragus of the ear, external auditory canal, or outer canthus of the eye.) Whichever location is used, it should be consistent for each patient. A bubble level or laser leveling device can be used to ensure accuracy of the leveling.

Ventricular Drainage with External Transducer

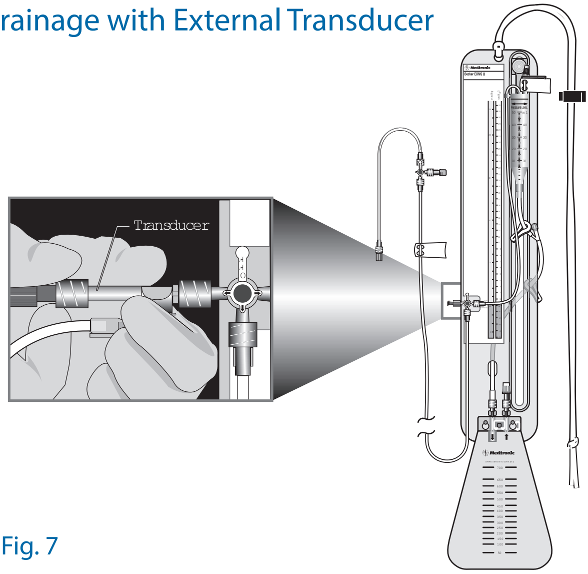

Fig. 7

The system can be used for both monitoring and therapeutic drainage of CSF. ICP measurements are most accurate when the system is closed to drainage. When the patient line is open, CSF flows into the graduated chamber when ICP exceeds a predetermined pressure setting. This preset pressure level prevents a further rise in ICP. Intermittent CSF drainage may be instituted upon physician orders. The order should specify how high the ICP should be allowed to rise and how long the elevation should be sustained before CSF is drained. The order should also specify the desired decrease in ICP and the volume of CSF to be drained.

Read the EDM system instructions for a complete understanding of the operation, warnings, and precautions.

Risks of ICP Monitoring

All invasive means of ICP monitoring carry potential risks such as infection, hemorrhage, increase in cerebral edema, and occlusion of the catheter/ sensing device. To reduce the risk of infection, many neurosurgeons prescribe prophylactic antibiotics for the duration of the catheter use. Others may limit the length of time that the catheter remains in place. Additionally, a catheter tunneled away from the incision site may help decrease infection. Strict attention to sterile technique at the time of insertion also prevents infection.

All patients should be monitored for signs of infection or meningeal irritation. Temperature should be monitored as per unit standards of care. Laboratory analysis of blood and CSF is also useful; analyses should include blood count. In addition, if a CSF infection is suspected, CSF samples should be sent for cell count, cultures, protein, and glucose. CSF analysis is not done routinely at most hospitals and should only be performed by the physician or other certified provider, as the risk of infection with sampling is high. If a CSF infection occurs, the ICP monitor may be removed and the patient should be started on appropriate antibiotics. If the patient requires continued ICP monitoring, a new monitor should be placed.

Treating Increased ICP

The American Association of Neurological Surgeons in conjunction with the Brain Trauma Foundation, reviewed the literature and developed guidelines for the management of increased ICP in severe head injury cases. Many of these guidelines can be implemented for increased ICP from causes other than head injury. The following is a brief summary of many of the commonly employed interventions aimed at lowering ICP, and is not intended to serve as management guidelines.

Hyperventilation has been used to reduce intracranial pressure by decreasing the paCO2. Decreasing the paCO2 causes blood vessels to constrict resulting in a reduction of blood flow through the brain. Prophylactic hyperventilation has been shown to worsen patient outcome, and the efficacy and safety of short term hyperventilation has not been determined. Some neurosurgeons hyperventilate to a paCO2 of 30-35 mm Hg in response to acute rises in ICP, or if hyperemia (excessive blood flow) is determined by other means.

Drug Therapy primarily includes osmotic diuretics. Osmotic diuretics, such as mannitol, cause water to be extracted from edematous brain tissue due to its high osmotic concentration, and can change the flow characteristics of blood. A disadvantage of using osmotic diuretics is a rebound effect that can occur causing an increase in ICP rather than a decrease. Barbiturate coma and chemical paralysis decrease the metabolic rate and activity that may cause increased ICP. Barbiturates cause cerebral vasoconstriction and decreased cerebral metabolism. Vasoconstriction reduces cerebral blood volume and a decrease in metabolism reduces the brain's demand for glucose and oxygen. Chemical paralysis decreases the activity of the rest of the body, which also assists in decreasing the ICP due to lack of bodily movement.

Glasgow Outcome Scale

Eyes Open	Spontaneously	4
	To Speech	3
	To Pain	2
	None	1
Best Verbal Response	Oriented	5
	Confused	4
	Inappropriate Words	3
	Incomprehensible Sounds	2
	None	1
Best Motor Response	Obeys Commands	6
	Localizes To Pain	5
	Flexor Withdrawal	4
	Abnormal Flexion	3
	Extension	2
	Flaccid	1

This scale is divided into three areas of focus. In each area the best response is recorded and the numerical values added together. This is an objective assessment tool. A score of 8 or less reflects coma and a need for vigorous intervention.

Fig. 8

Fluid Volume is usually maintained at a normovolemic state. Accurate intake and output can assist in determining volume status, but invasive monitoring such as a pulmonary artery catheter or a central venous pressure monitor provides a more accurate assessment of overall volume status. In addition, sodium should be monitored on a regular basis, as it can also be an indicator of fluid status and the presence of the syndrome of inappropriate ADH secretion, diabetes insipidus, or cerebral salt wasting.

Surgical Intervention can be the most effective method for dealing with increased ICP in specific cases, especially those caused by an expanding lesion such as an epidural, subdural, or intraparenchymal hematoma. If increased ICP is caused by cerebral edema, the skull flap may be left off to accommodate expansion of the brain tissue. Occasionally, a lobectomy may have to be performed due to uncontrolled ICP. Lobectomy is usually reserved for those patients who do not respond to other treatment modalities.

General Measures: The head of the bed is usually elevated to 30 degrees to promote venous drainage. If the CPP is low with the head of the bed elevated, the head may be lowered to promote a higher CPP. The neck should be kept in a neutral position. Hyperflexion, hyperextension, or severe rotation may impede venous drainage causing increased intracranial blood volume and possibly increasing ICP. Valsalva maneuvers may cause an increase in ICP and should be avoided if possible. The Valsalva maneuver causes an increase in intrathoracic pressure and a decrease in venous return, causing an increase in the blood volume in the brain.

The patient should be well oxygenated because hypoxemia causes cerebral vasodilatation. Hyperoxygenation should be performed prior to suctioning. Patients are at risk for pulmonary complications and good pulmonary toilet should not be neglected. Suctioning should be limited to 10-15 seconds and the patient should be monitored for effects on ICP.

Abnormal posturing may lead to increases in ICP due to the isometric nature of abnormal motor responses.

Increases in ICP may occur with Valsalva maneuvers, coughing, suctioning, inappropriate positioning, or other nursing interventions. Assessment of the patient’s response to nursing activities will dictate the frequency of interventions in order to minimize increases in ICP. Patient and family education should include the rationale for ICP monitoring, the position of the head of bed, possible replacement of the catheter, and potential need for the insertion of a shunt. It is important to involve the family in decreasing the amount of stimulation that the patient receives from visitors.

Ventricular Catheter and EVD Management

Management of the patient with a ventricular catheter requires constant assessment and intervention. All connections of the monitoring system must be tight to maintain the integrity of the closed system. The ICP monitor is an adjunct to careful patient assessment. While some patients may be awake and alert, most neurosurgical patients vary in their level of consciousness (LOC). This does not mean that all neurosurgical patients have an altered LOC, but suggests that this group has a greater predisposition to decreased levels and requires vigilant assessment.

The system should be dated. Changing of the catheter is not done on a routine basis but is determined by patient condition, continued requirement for ICP monitoring, and the neurosurgeon's preference. If a new catheter is inserted, a new drainage and monitoring system should be connected. Institutional policy should be followed in determining the frequency of changing the monitoring system and catheter. The nurse should record ICP, CPP, and the amount and characteristics of CSF drainage at least every hour, or more frequently if needed. Dressings should be observed for drainage and changed per institutional policy.

Regular neurological checks should be carried out in order to detect early changes in mental status or LOC. In addition to assessment of LOC, pupil checks and motor and sensory examinations should be carried out. Other protective reflexes (cough, gag, corneal) may also be assessed along with the vital signs. Most patients with a marked increase in ICP will have an altered LOC. A decrease in LOC is usually the first indicator of impending or actual neurological injury. Additional indicators of increased ICP are: lack of pupillary response on one or both sides due to compression of the oculomotor nerve; weakness or paralysis of the extremities on one side of the body due to compression of motor tracks from the cortex to the midbrain; changes in vital signs which are usually late indicators of increased ICP and include a widening pulse pressure and bradycardia; restlessness; respiratory distress; and respiratory arrest.

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