Intracranial hypertension can lead to potentially catastrophic consequences, including permanent neurological damage and disability. Intracranial hypertension may be due to central nervous system infection, brain edema, cerebral hemorrhage or ischemic injury, or brain trauma. Traumatic brain injury is a major cause of morbidity and mortality; it accounted for 50,000 deaths in 1998. Fatality may not result from the immediate traumatic or hemorrhagic injury; rather, progressive damage to brain tissue may develop over time. Brain damage becomes even more progressive if intracranial hypertension is a consequence of injury.

Astute nursing assessment and early, aggressive resuscitation of critically ill patients may prolong life. With brain trauma, the initial injury can be avoided only by using primary prevention strategies. Rapid control of fluctuations and elevations in intracranial pressure (ICP) may increase the potential for optimal functional recovery. In this article, I provide an overview of intracranial physiology and assessment findings specific to intracranial hypertension. Assessment findings reflect underlying pathophysiology in response to injury. These features are typically the first ones nurses consider in evaluating patients. I also review ICP monitoring and waveform analysis as central in caring for patients with intracranial hypertension.

Background

Monroe-Kellie Doctrine

The Monroe-Kellie doctrine provides the framework for reviewing physiology and treatment for elevations in ICP. This concept holds that the skull is a rigid compartment that contains 3 components: brain tissue, arterial and venous blood, and cerebrospinal fluid (CSF). Under normal circumstances, these components are balanced in a state of dynamic equilibrium. If an increase occurs in the relative volume of one component, such as brain tissue, the volume of one or more of the other components...
must decrease or an elevation in ICP will result. In a brain with disease or injury, small increases in volume may result in large increases in ICP. In adults, normal ICP is less than 10 to 15 mm Hg. Intracranial hypertension is defined as ICP greater than 20 mm Hg. Sustained intracranial hypertension is defined as an ICP greater than 20 mm Hg that persists for 5 minutes or longer.

**Pathophysiology**

Elevations in ICP are critical because they can decrease cerebral perfusion and blood flow. The brain requires 50 to 55 mL of blood per 100 g of brain tissue to maintain a normal metabolic state. Cerebral perfusion pressure (CPP) is an approximation of global cerebral blood flow. Patients with traumatic brain injury or cerebral infection, edema, or ischemia are at risk for marked, sustained elevations in ICP that if untreated may have devastating consequences.

First, sustained elevations in ICP compromise CPP, which is determined by subtracting mean ICP from mean arterial blood pressure. Decreased CPP, whether from hemodynamic compromise (decreased mean arterial blood pressure) or elevated ICP, can lead to global cerebral ischemic injury and marked neurological changes. Adequate CPP is vital for supporting cerebral perfusion. Many clinicians advocate that CPP in adults should be maintained at no lower than 70 mm Hg. Robertson suggests that a CPP of 60 to 70 mm Hg is adequate after traumatic brain injury. Controlled clinical studies will be necessary to firmly determine optimal CPP after traumatic brain injury.

Second, sustained ICP elevations can also lead to herniation syndromes, which are due to a shifting of brain tissue from an area of high pressure, such as occurs with a unilateral lesion within one cerebral hemisphere, to an area of lower pressure.

**Assessment Findings**

Assessment findings associated with intracranial hypertension (Table 1) and herniation syndromes (Table 2) are classified as early and late. Sustained elevations in ICP dramatically increase mortality and morbidity. Herniation syndromes can be further classified into supratentorial and infratentorial (Figure 1). Supratentorial herniations occur above the meningeal layer of the tentorium cerebelli and include central, uncal, and subfalcine herniation. Central herniation, caused by brain swelling or an expanding space-occupying lesion, causes compression and lengthening of the diencephalon. The medial temporal lobes are displaced along the brain stem through the tentorial notch. Subfalcine herniation, caused by an expanding mass lesion, for example, in a temporal lobe, results in displacement of the uncus of the temporal lobe through the tentorial notch. Subfalcine herniation, caused by a unilateral mass lesion such as an expanding hematoma results in displacement of the cingulate gyrus under the falx cerebri. Infratentorial herniation includes brain stem herniation and cerebellar tonsillar herniation syndromes.

**Table 1**  Assessment findings associated with intracranial hypertension

<table>
<thead>
<tr>
<th>Early findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased level of consciousness</td>
</tr>
<tr>
<td>Changes in mental status</td>
</tr>
<tr>
<td>Confusion</td>
</tr>
<tr>
<td>Lethargy</td>
</tr>
<tr>
<td>Abnormal findings on eye examination</td>
</tr>
<tr>
<td>Unequal and/or sluggish pupillary responses</td>
</tr>
<tr>
<td>Papilledema: swelling of optic disc because of venous congestion</td>
</tr>
<tr>
<td>Cranial nerve deficit: ptosis</td>
</tr>
<tr>
<td>Seizure activity</td>
</tr>
<tr>
<td>Sensory changes</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Later findings</td>
</tr>
<tr>
<td>Continued decrease in level of consciousness</td>
</tr>
<tr>
<td>Stupor</td>
</tr>
<tr>
<td>Coma states</td>
</tr>
<tr>
<td>Headache</td>
</tr>
<tr>
<td>Hemiplegia</td>
</tr>
<tr>
<td>Decorticate (flexion) posturing</td>
</tr>
<tr>
<td>Decerebrate (extensor) posturing</td>
</tr>
<tr>
<td>Vomiting (without nausea)</td>
</tr>
<tr>
<td>Brain stem pressure</td>
</tr>
<tr>
<td>Decrease or loss of protective reflexes: cough, gag, corneal reflexes</td>
</tr>
<tr>
<td>Changes in vital signs</td>
</tr>
<tr>
<td>Bradycardia, hypertension (initially)</td>
</tr>
<tr>
<td>Respiratory changes (rate, depth, pattern)</td>
</tr>
<tr>
<td>Cheyne-Stokes respirations, ataxic respirations.</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Terminal findings</td>
</tr>
<tr>
<td>Unstable blood pressure, heart rate</td>
</tr>
<tr>
<td>Profound hypotension</td>
</tr>
</tbody>
</table>
Depending on location of the lesion, upward displacement of structures in the posterior fossa may also occur.\textsuperscript{13-15}

**Etiology**

The causes of intracranial hypertension are classified as acute or chronic. Acute causes include brain trauma, ischemic injury, and intracerebral hemorrhage. Infections such as encephalitis or meningitis may also lead to intracranial hypertension. Chronic causes include many intracranial tumors, such as ependymomas, that may gradually impinge on CSF pathways and interfere with CSF outflow and circulation. Chronic subdural hematomas also may cause intracranial hypertension. Intracerebral hemorrhage is the result of arterial bleeding directly into brain tissue. Localized injury occurs at the source of the bleeding, and the hemorrhage may directly damage the brain tissue as well as cause global injury due to brain edema and elevations in ICP. Intracerebral hemorrhage may also lead to obstructive hydrocephalus due to compression or occlusion of the CSF pathways. As the ICP continues to increase, the brain tissue becomes distorted, leading to herniation and additional vascular injury.\textsuperscript{10}

In severe head trauma, in addition to the localized, direct tissue damage and hemorrhage\textsuperscript{5} associated with the injury, edema within brain tissue commonly occurs, resulting in ICP elevation and further brain injury.\textsuperscript{8} Meningitis is an inflammation of the meninges. It can lead to localized or global cerebral ischemia, cerebral edema, and hydrocephalus due to decreased CSF reabsorption and increased CSF production.\textsuperscript{16-18}

These acute and chronic causes of intracranial hypertension can, if not managed aggressively, lead to devastating consequences. Figure 2 shows a catastrophic elevation in ICP with associated early hemodynamic consequences of a hyperdynamic state. Figure 3 shows hemodynamic changes that occur in response to catastrophic intracranial hypertension and brain stem herniation.

**Table 2  Assessment findings associated with supratentorial herniation syndromes\textsuperscript{13-15}**

<table>
<thead>
<tr>
<th>Herniation Syndrome</th>
<th>Early Findings</th>
<th>Late Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cingulate/subfalcine herniation</td>
<td>Detects by using neuroimaging such as head computed tomography or magnetic resonance imaging. Often an indication of brain decompensation and poor intracranial compliance. This herniation syndrome does not have a specific set of assessment findings but may lead to further injury related to localized compromise in blood flow and tissue distortion.</td>
<td>Pupils unequal and nonreactive progressing to pupils fixed and dilated. Decreased responses to stimulation (tactile, auditory). Decreased blood pressure.</td>
</tr>
</tbody>
</table>
initial brain injury. Secondary injury is progressive and often begins shortly after brain injury. Contributing factors to secondary brain injury include hypotension, cerebral edema, hypoxemia, and intracranial hypertension.9,19 Primary and secondary injury may then begin a “vicious cycle” leading to multiple stages of progressive neuronal damage. From the point of injury, disruption of cell membranes leads to loss of electrical sta-

Figure 1 Coronal views of supratentorial and infratentorial compartments with associated changes caused by specific herniation syndromes. A, Normal relationship of the compartments. B, Central transtentorial herniation. The brain is swollen, the diencephalon is compressed and elongated, and the medial temporal lobes are forced along the brain stem through the tentorial notch. C, Uncal and transtentorial herniation. The cingulate gyrus is herniated under the falx cerebri, the uncus herniates through the tentorial notch, and the brain stem is compressed and shifted laterally and downward.

Adapted from Stubgen and Caronna,13 with permission from Elsevier.

Figure 2 ICP waveforms show catastrophic elevation in ICP and associated early hemodynamic consequences of a hyperdynamic state. Devastating ICP elevation with hemodynamic surges leads to arterialization of ICP waveform and lethal cerebral perfusion pressure.

Abbreviations: ABP, arterial blood pressure; CPP, cerebral perfusion pressure; HR, heart rate; ICP, intracranial pressure; PAWP, pulmonary artery wedge pressure; RESP, respirations; SaO2, oxygen saturation; VENT TACHY, ventricular tachycardia.
bility and depolarization. Through the effects of the inflammatory response, generation of free radicals and calcium-mediated damage, membrane integrity is further degraded, leading to increased tissue edema and ICP elevation. The initial brain injury plus secondary events results in a cumulative process that is life threatening. Ultimately, physiological stability becomes disturbed and compromising tissue damage occurs, advancing the cycle of secondary injury. The resulting change in cerebral physiology may produce dramatic, potentially catastrophic ICP elevations in excess of 20 mm Hg. If significant ICP elevations are not aggressively treated, severe, permanent neurological deficits or death will occur.

Intracranial hypertension can cause additional ischemic injury and highlights the need for early ICP monitoring and optimal hemodynamic management to maintain adequate global cerebral perfusion. Cerebral ischemia is an important secondary event after brain injury. Cerebral ischemia may occur for 2 reasons: (1) an already compromised cerebral blood flow near mass lesions caused by compression of localized blood vessels or (2) the presence of vasospasm after traumatic injury or subarachnoid hemorrhage. Loss of autoregulation, the brain’s ability to maintain consistent cerebral blood flow despite variations in systemic arterial pressure, may also occur after traumatic brain injury. Loss of autoregulation increases the risk for secondary brain injuries such as cerebral ischemia and intracranial hypertension.

**Rationale for ICP Monitoring**

The Monroe-Kellie doctrine describes pressure-volume relationships within the intracranial cavity. Secondary brain injury may be a
direct consequence of intracranial hypertension. Therefore, monitoring ICP and CPP should be an immediate objective in the care of patients with severe traumatic or hemorrhagic brain injury. Vigilant assessment and monitoring of intracranial hypertension is the standard of care. ICP monitoring is appropriate in patients with severe head injury if 2 or more of the following are present: age greater than 40 years, unilateral or bilateral motor posturing (decortication or decerebration), or systolic blood pressure less than 90 mm Hg. Evidence of distortion, swelling, or hydrocephalus on imaging studies such as head computed tomography is an additional indication for ICP monitoring. Before ICP monitoring is started, the initial evaluation of patients with potential neurological injury includes a physical examination and an imaging study such as head computed tomography. This workup yields vital information on the state of the brain before invasive monitoring is started. Continuous monitoring of ICP and CPP, a determining factor for clinical outcome, provides effective guidance in managing ICP elevations and hemodynamic status. Further interventions can be based on these clinical findings. The goal of ICP and hemodynamic monitoring is to augment and maintain stability of CPP and cerebral blood flow to prevent further cerebral ischemic injury.

**Significance of ICP Pulse Waveforms**

ICP pulse waveforms are generated from a pressure wave transmitted through the cardiovascular system into the tissues within the intracranial cavity, including the choroid plexus, where CSF is produced. The pulse waveform consists of 3 components: P-1, P-2, and P-3 (Figure 4). The percussion wave, P-1, reflects pulsations of the choroid plexus as transmitted from the cardiovascular system at systole. Under normal conditions, P-1 is the highest of the 3 waveform components. The tidal wave, P-2, has a more variable shape and reflects relative brain volume. The P-2 component is often elevated in response to a rapidly expanding mass lesion such as a hematoma or an intracranial tumor. The dicrotic wave, P-3, follows the dicrotic notch on the downslope of the individual ICP pulse waveform. Additional waves that may represent pulsations within the cerebral venous system may occur before the next P-1. Normally, the segments of the ICP pulse waveform progress in a descending staircase pattern; P-1 is the highest waveform segment, and P-3 is the lowest.

**Intracranial Compliance**

Intracranial compliance refers to the ability of the brain to tolerate stimulation such as endotracheal suctioning or increases in volume of 1 or more of the brain’s 3 components (brain tissue, blood, and CSF) without a corresponding increase in ICP. Several methods are used to assess intracranial compliance. In one method, a limited volume of fluid such as preservative-free saline (in 1-mL increments) is instilled into the intracranial space and the ICP response is measured (volume-pressure curve). An increase in ICP greater than 2 mm Hg for each milliliter of fluid added to the system indicates compromised intracranial compliance. In another method, ICP is measured before and after a stimulus such as endotracheal suctioning. An ICP that returns to normal or baseline values quickly (within 5 minutes) indicates adequate intracranial compliance. An ICP that takes 20 to 25 minutes to return to normal or baseline values may indicate poor intracranial compliance. In a third method, intracranial compliance is assessed by directly examining the ICP pulse waveform. A higher than normal or baseline amplitude of the P-2 component of the pulse waveform is associated with compromised intracranial compliance, particularly if P-2 is the highest-amplitude component of the pulse waveform. Figure 5 shows ICP pulse waveforms associated with normal and compromised intracranial compliance.
ICP Monitoring Techniques

Several methods are used to monitor ICP. These methods include insertion of an intraventricular catheter, a subarachnoid bolt or screw, or a subdural or epidural catheter and intraparenchymal insertion of a fiberoptic transducer-tipped catheter (Figure 6). Each method has specific advantages, disadvantages, and nursing considerations (Table 3).

Ventriculostomy/Intraventricular Catheter

Currently, ventriculostomy is the most accurate, cost-effective, and reliable method of monitoring ICP. It is the standard for ICP measurement. The ventriculostomy catheter is part of a system that includes an external drainage system and a transducer. The drainage system and transducer are primed on insertion with preservative-free saline. The transducer can easily be calibrated or zeroed against a known pressure. This calibration ensures the consistency and accuracy of the pressure measurements obtained.
Table 3 Advantages, disadvantages, and nursing considerations of ICP monitoring techniques

<table>
<thead>
<tr>
<th>Monitoring device</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraventricular catheter</td>
<td>Allows accurate ICP measurement</td>
<td>Provides an additional site for infection</td>
</tr>
<tr>
<td></td>
<td>Provides access to CSF for drainage or sampling</td>
<td>Is most invasive ICP monitoring technique</td>
</tr>
<tr>
<td></td>
<td>Provides access for instillation of contrast media</td>
<td>Requires frequent transducer balancing or recalibration</td>
</tr>
<tr>
<td></td>
<td>Allows reliable evaluation of intracranial compliance (volume-pressure relationships)</td>
<td>Catheter may become occluded by blood clot or tissue debris</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Insertion is difficult if ventricles are small, compressed, or displaced</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Is associated with risk for CSF leakage around insertion site</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Is associated with increased risk for infection</td>
</tr>
<tr>
<td>Subarachnoid bolt or screw</td>
<td>Is associated with lower infection rates than is ventriculostomy</td>
<td>Has potential for dampened waveform (cerebral edema, blood or tissue debris)</td>
</tr>
<tr>
<td></td>
<td>Is quickly and easily placed</td>
<td>Is less accurate at high ICP elevations</td>
</tr>
<tr>
<td></td>
<td>Can be used with small or collapsed ventricles</td>
<td>Requires frequent balancing/recalibration (and with position changes)</td>
</tr>
<tr>
<td></td>
<td>Requires no penetration of brain tissue</td>
<td>Provides no access for CSF sampling</td>
</tr>
<tr>
<td>Subdural or epidural catheter or sensor</td>
<td>Is least invasive</td>
<td>Increase in baseline drift over time means possible loss of reliability or accuracy</td>
</tr>
<tr>
<td></td>
<td>Is associated with decreased risk of infection</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Is easily and quickly placed</td>
<td></td>
</tr>
<tr>
<td>Fiberoptic transducer-tipped catheter</td>
<td>Can be placed in subdural or subarachnoid space, in a ventricle, or directly within brain tissue</td>
<td>Provides no access for CSF sampling/drainage</td>
</tr>
<tr>
<td></td>
<td>Requires zeroing only once (during insertion)</td>
<td>Cannot be recalibrated after placement</td>
</tr>
<tr>
<td></td>
<td>Has baseline drift of up to 1 mm Hg per day</td>
<td>Requires periodic replacement of probe</td>
</tr>
<tr>
<td></td>
<td>Is associated with decreased risk for infection when brain tissue is not penetrated</td>
<td>Is easily damaged</td>
</tr>
<tr>
<td></td>
<td>Provides good quality ICP waveforms (less artifact than with other devices)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Requires no adjustment in level of transducer with patient's change of position</td>
<td></td>
</tr>
</tbody>
</table>

*Fluid-filled ICP monitoring systems not maintained with continuous flush or pressure bag.

Abbreviations: CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; ICP, intracranial pressure.

Adapted from Shpritz, with permission from Elsevier. Added material based on information in March, Hickey, Kerr and Crago, and American Association of Neuroscience Nurses.
A ventriculostomy is most commonly placed in the nondominant lateral ventricle via a twist drill hole in the skull. The dura is incised or punctured, and the catheter is passed through the cerebral tissue into the ventricle. The catheter is then typically tunneled under the scalp and sutured to the skin surface a few centimeters from the initial insertion site.

Advantages of using an indwelling ventricular catheter include allowing CSF drainage to effectively decrease ICP and using the catheter as a means to instill medications. Access to CSF drainage allows serial laboratory tests of CSF and determination of volume-pressure relationships. Disadvantages of ventriculostomy include risk of infection, which is higher than that associated with other ICP monitoring techniques. In addition, the catheter may become occluded with blood or tissue debris, interfering with CSF drainage or ICP monitoring. Also, if significant cerebral edema is present, locating the lateral ventricle for insertion of the ventriculostomy catheter may be difficult. Last, bleeding or ventricular collapse may occur if CSF is drained too rapidly. For this last reason, many clinicians set the ventriculostomy drainage system to drain CSF when the ICP is greater than 15 to 20 mm Hg by adjusting the height of the drip chamber. In addition, a limit of ventricular drainage such as 5 to 10 mL/h has been used to avoid excessively rapid CSF drainage. Using a ventriculostomy may allow lifesaving CSF drainage and control of intracranial hypertension.

**Subarachnoid Bolt or Screw**

The subarachnoid bolt or screw is inserted via a twist drill hole to the
level of the subarachnoid space over the nondominant cerebral hemisphere. The dura is then opened, and the device (filled with preservative-free saline) is placed in contact with the subarachnoid space. The bolt is then secured to pressure tubing and a transducer system also primed with preservative-free saline.14,15,34,35

This technique of ICP monitoring is easier to initiate than is a ventriculostomy, a characteristic that is an advantage. Locating the ventricles is not necessary, facilitating ease of insertion and ICP monitoring even in patients who have mass effect or brain edema. Because the cerebral tissue is not violated, the infection rate for the procedure is lower than the rates for other ICP monitoring methods that penetrate cerebral tissue.7,11,14,15,34

Disadvantages of a subarachnoid bolt or screw include the following. The bolt can be occluded by debris, blood clots, or herniation of brain tissue into the device. This blockage may dampen the ICP waveform and make the pressure measurement inaccurate. As with any invasive monitoring technique, leakage of CSF and infection may still occur. Last, bleeding and intracranial hematoma formation may occur, possibly causing further brain injury.7,11,14,15

Subdural or Epidural Catheter

Placement of a subdural or epidural fiberoptic transducer-tipped catheter is accomplished via a small twist drill hole through the skull. This monitoring technique does not require penetration of the cerebral tissue, and the risk of infection is less than that associated with other ICP monitoring techniques that penetrate cerebral tissue. Catheter insertion and initiation of monitoring are also easier, and recalibration of the system is not necessary.

Disadvantages of this technique are multiple. First, access to CSF is not possible. Second, a wedge effect may occur from pressure between the catheter tip and the adjacent dura. This effect may compromise the accuracy of the ICP measurements and waveform quality. Third, volume-pressure relationships cannot be determined as an indication of intracranial compliance. Last, ICP waveforms may be of poor quality, limiting the amount of useful information obtained with this monitoring technique.7,11,14,15,34,35 Because epidural or subdural monitoring tends to be less accurate than other monitoring techniques, it is used far less frequently.

Intraparenchymal Sensor

Intraparenchymal sensors are typically placed via a small twist drill hole and passed through a bolt device placed in contact with the subarachnoid space. The fiberoptic transducer-tipped catheter is placed through the bolt into the brain tissue. This ICP monitoring device is easy to insert and maintains excellent quality waveforms even in patients with cerebral edema.

An advantage to using this technique is the minimal risk of tissue herniation and the accuracy of ICP measurements despite variations in head positioning. Because head positioning has minimal influence on the accuracy of ICP measurements, ICP monitoring can also be maintained during patients’ transport.7,11,14,15

Disadvantages of this technique include lack of access to CSF for drainage or laboratory tests and inability to assess intracranial compliance by determining volume-pressure relationships. In addition, the fiberoptic cable may be fragile, and manipulation of the catheter may damage optical fibers, affecting ICP readings.7,11,14,15,34,35 Last, although ICP measurements obtained at the time of insertion are accurate, baseline drift can occur, which may result in an error in ICP measurements after several days of use.7

Nursing Considerations

The Monroe-Kellie doctrine dictates that the 3 components of the skull (blood, brain tissue, and CSF) interact with one another for stability of ICP and intracranial physiology. Treatment of elevations in ICP involves selectively manipulating or decreasing the relative volume of one or more of these components. Interventions for ICP elevation must do more than address the specific derangement in intracranial physiology such as elevated cerebral blood flow, edema, or hydrocephalus; they must also be tailored to the specific physiological changes at a given moment.

The interactions between the 3 components of the skull are a dynamic process. How the brain responds to injury over time is similarly dynamic. For example, an intervention that provides effective management of intracranial hypertension the first day after injury, such as using osmotic diuresis to treat cerebral edema, may not be as effective for intracranial hypertension related to an increase in cerebral blood flow that occurs 3 days after injury.12 This change in the effectiveness of interventions underscores the need for continual neurological assessment and ICP
measurement and the need to determine the cause of elevated ICP in order to tailor therapy specifically to the physiological disturbance at a given time.4,12

The need for vigilance in ICP monitoring and patients’ assessment is further underscored by the ICP response to medical and nursing interventions. Cervical spine immobilization by means of rigid cervical collars may also result in ICP elevations by compressing the jugular vein.38 Prone positioning of patients who have acute respiratory distress syndrome after subarachnoid hemorrhage can also result in increased ICP.39 In these instances, clearly, measures to immobilize the cervical spine and optimize oxygenation are appropriate; however, the potential effects of these interventions on ICP necessitate vigilance and appropriate attention.

Optimal assessment of intracranial compliance is tremendously important for favorable outcomes. Recognition of even subtle changes in intracranial compliance can have far-reaching implications in overall management, including nursing considerations. One example is pulmonary care and endotracheal suctioning. A patient with compromised intracranial compliance is at higher risk than a patient without compromise for potentially dangerous, acute elevations in ICP and further neurological injury related to the cough reflex, noxious stimulation, and dramatic increases in intrathoracic pressure.5,7,24 Under these circumstances, measures such as administration of analgesics or intratracheal lidocaine to attenuate the cough reflex and the response to noxious stimuli may be appropriate considerations in high-risk patients. An arousal response to endotracheal suctioning can be detected by clinical assessment and can be documented by using electroencephalography-based monitoring.25,26 The arousal response can be blunted by giving patients intravenous opioids40 or intratracheal lidocaine41 before the airway is suctioned.

Patients with poor intracranial compliance are more sensitive to stimuli that can increase ICP. These stimuli include nursing care activities, peripheral venipuncture, and repositioning, among others. As clinically appropriate, interventions should be spaced over time to limit a cumulative effect on increasing ICP.4

Patients experiencing intracranial hypertension are among the most challenging in critical care practice. Rapid initiation of treatment to protect them from a devastating outcome depends on aggressive and thorough clinical assessment.

Changes in the level of consciousness are the most important measure of cerebral stability. Consciousness reflects physiological stability and coordination between and among multiple brain areas. Consequently, any change in level of consciousness, cognition, or responsiveness, even if subtle, can have great significance. Decreases in the level of consciousness can be caused by brain compression due to hemorrhage, edema, hematoma formation, and expanding solid tumors. Monitoring of consciousness and arousal by using the Glasgow Coma Scale14,42 (Table 4) is vital.

Monitoring changes in pupil size and reactivity and alterations in vital signs is also imperative. ICP elevations and localized space-occupying lesions may result in compression or displacement of the optic or oculomotor nerves, causing changes in pupil size and reactivity. Changes in vital signs may be an early or late indication of intracranial hypertension. Nurses should also be alert for changes in respiratory rate, depth, or pattern, which may indicate brain stem compression. Clinical assessment, particularly of unconscious patients or

<table>
<thead>
<tr>
<th>Response</th>
<th>Score</th>
</tr>
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<tbody>
<tr>
<td>Eye opening</td>
<td></td>
</tr>
<tr>
<td>Opens eyes spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>Opens eyes in response to speech</td>
<td>3</td>
</tr>
<tr>
<td>Open eyes in response to painful stimulation (eg, endotracheal suctioning)</td>
<td>2</td>
</tr>
<tr>
<td>Does not open eyes in response to any stimulation</td>
<td>1</td>
</tr>
<tr>
<td>Motor response</td>
<td></td>
</tr>
<tr>
<td>Follows commands</td>
<td>6</td>
</tr>
<tr>
<td>Makes localized movement in response to painful stimulation</td>
<td>5</td>
</tr>
<tr>
<td>Makes nonpurposeful movement in response to noxious stimulation</td>
<td>4</td>
</tr>
<tr>
<td>Flexes upper extremities/extends lower extremities in response to pain</td>
<td>3</td>
</tr>
<tr>
<td>Extends all extremities in response to pain</td>
<td>2</td>
</tr>
<tr>
<td>Makes no response to noxious stimuli</td>
<td>1</td>
</tr>
<tr>
<td>Verbal response</td>
<td></td>
</tr>
<tr>
<td>Is oriented to person, place, and time</td>
<td>5</td>
</tr>
<tr>
<td>Converses, may be confused</td>
<td>4</td>
</tr>
<tr>
<td>Replies with inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Makes incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>Makes no response</td>
<td>1</td>
</tr>
</tbody>
</table>
patients receiving sedatives, analgesics, or neuromuscular blocking agents, may not be optimal for detecting intracranial hypertension. For this reason, ICP monitoring will remain essential in patients’ management. Intracranial pressure elevations and diminished compliance can be quickly detected, facilitating optimal treatment.

Acknowledgments
The author thanks Cathy McDeed-Breault, RN, MSN, CANP, at the Veterans Administration Medical Center, San Diego, Calif, for her editorial assistance as a publishing partner in the preparation of this manuscript.

References
CE Test Form

Intracranial Hypertension: Monitoring and Nursing Assessment

Objectives:
1. Identify the acute and chronic causes of intracranial hypertension
2. Describe the 4 methods of intracranial pressure monitoring
3. Discuss nursing assessment and monitoring of intracranial hypertension

Mark your answers clearly in the appropriate box. There is only 1 correct answer. You may photocopy this form.

1. a 2. a 3. a 4. a 5. a 6. a 7. a 8. a 9. a 10. a 11. a 12. a
   b b b b b b b b b b b b b
   c c c c c c c c c c c c c
   d d d d d d d d d d d d d

Program evaluation
Objective 1 was met
Objective 2 was met
Objective 3 was met
The content was appropriate
My expectations were met
This method of CE is effective for this content

The level of difficulty of this test was:
☐ easy  ☐ medium  ☐ difficult
To complete this program, it took me
____________________ hours / minutes.

Name ________________________________
Address ________________________________
City ____________________ State____ ZIP________
Phone ( ) _____________________________
E-mail address __________________________
RN license number (required) ______________
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CE Test Questions
Intracranial Hypertension: Monitoring and Nursing Assessment

1. According to the Monroe-Kellie doctrine, which 3 components contained in the skull are balanced in a state of dynamic equilibrium?
   a. Bone, brain tissue, and cerebral blood
   b. Brain tissue, cerebral blood, and cerebrospinal fluid
   c. Cerebral blood, cerebrospinal fluid, and bone
   d. Cerebrospinal fluid, bone, and brain tissue

2. Which intracranial pressure (ICP) is indicative of intracranial hypertension?
   a. 12 mm Hg
   b. 15 mm Hg
   c. 18 mm Hg
   d. 21 mm Hg

3. Which of the following conditions is not an acute cause of intracranial hypertension?
   a. Cerebral trauma
   b. Ischemic injury
   c. Intracranial tumor
   d. Cerebral hemorrhage

4. Which of the following factors does not contribute to secondary brain injury?
   a. Hypertension
   b. Cerebral edema
   c. Hypoxemia
   d. Intracranial hypertension

5. Before ICP monitoring is started, the initial evaluation of patients should include a physical examination and which of the following studies?
   a. Electroencephalogram
   b. Carotid ultrasound
   c. Computed tomography of the head
   d. Carotid arteriogram

6. Which component of the ICP pulse waveform is elevated in response to a rapidly expanding lesion such as a hematoma?
   a. P-1
   b. P-2
   c. P-3
   d. P-4

7. Which of the following is the most accurate, cost-effective, and reliable method of monitoring ICP?
   a. Subarachnoid bolt or screw
   b. Subdural or epidural catheter
   c. Intraparenchymal sensor
   d. Ventriculostomy catheter

8. Which complication of ventriculostomy is higher than that associated with other ICP monitoring techniques?
   a. Cerebral bleeding
   b. Cerebral ischemia
   c. Cerebral infection
   d. Cerebral edema

9. Which of the following interventions does not result in ICP elevation?
   a. Supine position
   b. Rigid cervical collar
   c. Suctioning
   d. Venipuncture

10. Acute elevations in ICP can be prevented by blunting the arousal response to endotracheal suctioning through administration of which medication?
    a. Intratracheal lavage with saline
    b. Intratracheal lidocaine
    c. Intratracheal opioids
    d. Intratracheal bronchodilators

11. Which is the most important measure of cerebral stability?
    a. Motor function
    b. Sensory function
    c. Pupil size
    d. Level of consciousness

12. Which of the following scales is vital in monitoring arousal and consciousness?
    a. Glasgow Coma Scale
    b. Ramsay Sedation Scale
    c. Motor Activity Assessment Scale
    d. Glasgow Outcome Scale
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