Increased Intracranial Pressure (ICP)

The cranium is a rigid bony structure that contains three main components: brain tissue, cerebrospinal fluid (CSF), and blood. Increased intracranial pressure (ICP) occurs when there is an increase in the volume of one or more components which cannot be offset by a volume reduction or displacement in some other component.

Typically, ICP in adults is less than 15 mm Hg and anything above 20 mm Hg is considered elevated ICP. In practical applications, the goal ICP is less than 20 mmHg. Slow increases in ICP, such as with the growth of a meningioma, tend to be associated with more subtle symptoms, while rapid intracranial hypertension, such as seen with an acute subdural hematoma, is associated with abrupt and obvious symptoms (Smith & Amin-Hanjani, 2024).

Conditions Associated with Increased ICP

The development of increased ICP may be acute or chronic. It is a common clinical problem in neurology or neurosurgical units. Many diseases or insults can result in increased ICP including:

- Increase in brain volume
    - Cerebral edema (trauma, ischemia, hyperammonemia, encephalitis, high altitude)
    - Hematoma (Epidural, subdural, subarachnoid, or intracranial hemorrhage)
    - Tumor
    - Abscess
    - Blood clots
- CSF dysregulation
    - Increase in cerebrospinal fluid (infection, choroid plexus tumor)
    - Decreased re-absorption of CSF (obstructive hydrocephalus, meningitis)
- Increase in blood volume
    - Increased cerebral blood flow (hypercarbia, aneurysms)
    - Venous stasis from venous sinus thromboses
    - Elevated central venous pressures (severe heart failure)
- Other causes
    - Idiopathic intracranial hypertension
    - Skull deformities such as craniosynostosis
    - Vitamin A intoxication
    - Tetracycline use

Acute intracranial hypertension (AIH) is a clinical syndrome in which homeostatic mechanisms are overwhelmed causing a rapid increase in intracranial pressure (ICP). AIH is a medical emergency requiring immediate recognition and treatment to prevent irreversible neurologic damage or death. Patients identified at increased risk for AIH should be monitored closely in a critical care setting.

Signs and Symptoms of Increased ICP

Conditions associated with chronically increased ICP may first present insidiously. Headaches may be the only symptom of chronic intracranial hypertension. Chronic intracranial hypertension can cause vision loss due to pressure on the optic nerve. Intracranial volume may increase steadily over months with minimal symptoms and no change in the level of consciousness, and yet present dramatically with an acute deterioration of consciousness when compensatory mechanisms are exceeded.

There should be a high clinical suspicion of increased ICP for patients presenting with acute/severe headache, papilledema, and vomiting. The patient may describe the headache as throbbing pain which worsens with actions that further increase ICP such as coughing, sneezing, recumbency or exertion. Other initial signs and symptoms of increased ICP include nausea, blurred vision, restlessness, irritability, and confusion. The clinical presentation of AIH can be mistaken for other problems, such as drug or alcohol intoxication, migraine headache, infection, or post-ictal state.

Level of consciousness will decrease progressively as ICP gets worse. The Glasgow Coma Scale (GCS) is the most common scoring system used to objectively describe the patient's level of consciousness. The GCS is composed of three objective tests: eye, verbal, and motor responses. The lowest possible total GCS is 3, indicative of deep coma, while the highest is 15. GCS scores help facilitate communication among healthcare providers and provide guidance for diagnostic workup and therapeutic intervention.

Glasgow Coma Score (Teasdale & Jennett, 1974)		
Feature	**Response**	**Score**
Best eye response If local injury, edema, or otherwise unable to be assessed, mark "Not testable (NT)"	Spontaneously To verbal command To pain No eye opening Not testable	4 3 2 1 NT
Best verbal response If intubated or otherwise unable to be assessed, mark "Not testable (NT)"	Oriented Confused Inappropriate words Incomprehensible sounds No verbal response Not testable	5 4 3 2 1 NT
Best motor response If on sedation/paralysis or unable to be assessed, mark "Not testable (NT)"	Obeys commands Localizes pain Withdrawal from pain Flexion to pain Extension to pain No motor response Not testable	6 5 4 3 2 1 NT

Increased ICP may cause protrusion or herniation of brain tissue through one of the rigid intracranial barriers. Signs of brain herniation include pupillary dilatation, hemiplegia, impaired oculocephalic movements, increased motor tone, flexion or extension to pain (posturing), and respirations containing sighs, deep yawns, or pauses. The Cushing reflex, also known as Cushing's triad, consists of hypertension, bradycardia, and diminished respiratory effort. The presence of Cushing's triad is a poor diagnostic sign and indicates impending brainstem herniation. Prompt emergency treatment is warranted.

Managing Increased ICP

Nursing measures are targeted to assessing for changes in the neurologic exam, preserving cerebral blood flow through optimizing cerebral perfusion pressure (CPP), and protecting the brain from secondary injury. Interventions, such as patient movement, suctioning, and ventilator asynchrony, can elevate ICP and should be brief with a goal of preventing sustained ICP levels greater than 20 mm Hg for less than 5-10 minutes (Smith & Amin-Hanjani, 2024).

The ABCs of Managing Increased Intracranial Pressure (Hussein et al, 2017)

	Strategy	Intervention
A	Airway	• Monitor airway patency. • Intubation, if needed, to prevent hypoxemia and hypercapnia.
B	Blood pressure	• Monitor cerebral perfusion pressure (CPP). • Maintain adequate blood pressure to optimize CPP. • Avoid hypotension.
C	Calm	• Decrease stimulation by providing a calm, quiet environment. • Minimize nursing interventions and space them out as able. • Provide intravenous (IV) sedatives as needed. Propofol is a preferred drug choice due to easy titration and short half-life that assists with neurologic assessments.
D	Decompression	• Insert a nasal (*) or oral gastric tube to decompress the stomach to decrease vomiting. **Note: Nasogastric tubes are contraindicated with basilar skull fractures or intranasal surgery, such as transsphenoidal pituitary resection.*
E	Edema	• Monitor intracranial pressure if a catheter has been placed. • Monitor for changes in the neurologic exam. • Administer osmotic diuretics or hypertonic saline as ordered.
	Eyes	• Monitor pupillary responses to light and corneal reflexes.
	Elevate the head	• Raise the head of the bed to 30 degrees to facilitate jugular venous outflow. • Maintain neck in neutral position.

F	Fluids and electrolytes	• Monitor for fluid and electrolyte imbalances that might indicate abnormal anti-diuretic hormone (ADH) levels. • Administer isotonic IV fluids and electrolytes as ordered to keep patient euvolemic and normo- to hyperosmolar.
	Food	• Provide enteral nutrition either through a feeding tube or orally if cleared by a speech pathologist.
	Family	• Provide family education and support. Participate in advanced care planning.
G	Glasgow Coma Scale (GCS)	• Monitor GCS hourly and report changes to a provider.
H	Hip flexion	• Avoid hip flexion which increases intra-abdominal and intrathoracic pressure.
	Hyperventilation	• Hyperventilation is used to lower carbon dioxide levels if other measures to lower ICP are ineffective.
	Hyperthermia	• Prevent hyperthermia to lower the metabolic requirements of the brain.
	Herniation	• Monitor for impending herniation: unilateral or bilateral pupil dilation, coma, posturing, Cushing's reflex (hypertension, bradycardia, bradypnea).
I	ICP monitoring	• Monitor ICP and inform provider of sustained ICP>20. • Monitor the effect of nursing care on ICP and minimize treatments or stimuli which increase it.
	Infection	• Strict aseptic technique when applying or changing dressings to ICP monitoring devices or ventricular drainage systems.

The prompt recognition and management of patients with increased ICP requires knowledge of at-risk patient populations and the signs and symptoms of elevated ICP. Acute intracranial hypertension resulting from rapid elevation of intracranial pressure is a medical emergency requiring immediate stabilization of airway, breathing and circulation followed by immediate brain imaging for confirmation and diagnosis of the underlying etiology. ICP monitoring, and in certain cases CSF drainage, is a cornerstone of management. Astute and high-quality nursing care includes providing a calm, quiet environment, vigilant monitoring, and interventions to optimize cerebral blood flow and prevent complications.

References:

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