PATHOPHYSIOLOGY

In adults, the rigid cranial cavity created by the skull is normally filled with three essentially *noncompressible* elements: the brain (85%), CSF (5%), and blood (10%).

A state of dynamic equilibrium exists.

If the volume of any of these components ↑, the volume of the others must ↓ to maintain normal pressures in the cranial cavity.

Monro-Kellie hypothesis: If volume ↑ in any of brain, CSF, or blood → volume of others must ↓

ETIOLOGY

Brain requires constant supply of oxygen and glucose If blood flow interrupted → ischemia, disruption of cerebral metabolism.

Compensatory mechanisms to maintain blood flow when ICP increases

- Pressure autoregulation
- Chemical autoregulation
- Displacement of some CSF to spinal subarachnoid space
- Increased CSF absorption

Autoregulatory mechanisms have limited ability to maintain cerebral blood flow

CAUSES

Head injury	Cerebral edema
Hydrocephalus	Imbalance between production/absorption of CSF
Excess CSF	Congenital or acquired
Brain tumor or abscess	Head trauma
Intracranial hemorrhage	Infection
	Tumor

COLLABORATION

Identify and treat underlying cause

Control ICP to prevent herniation syndrome

ICP >40 mmHg = life-threatening medical emergency	Diagnosis made on basis of observation, neurologic assessment
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CLINICAL MANIFESTATIONS

Loss of autoregulation	• ICP continues to rise, cerebral perfusion falls
	• Causes cerebral tissue ischemia, manifestations of cellular hypoxia
Changes in cortical function	Earliest manifestations may be delayed by compensatory measures
	• If slow onset of IICP, decrease in level of consciousness (LOC) might not be presenting symptoms Instead visual disturbances, vomiting, or headache
	• Lumbar puncture could cause brain herniation
	• **Cushing triad**
	• Behavior, personality changes
	• Impaired memory, judgment
	• Changes in speech pattern
	• LOC decreases to coma, death

DX TESTS

Diagnosis made on basis of observation, neurologic assessment

Lumbar puncture not performed when IICP suspected	Release of pressure could cause herniation

Serum osmolality, arterial blood gases (ABGs)

Electroencephalogram (EEG) may be used to monitor depth of coma or to diagnose brain death

Transcranial Doppler (TCD) to measure cerebral blood flow velocity	Especially for patients who have vasospasms related to cerebral hemorrhage

ASESSMENT

Observation and patient interview	- LOC using GCS
	- Any loss of motor control
	- Primary complaints
	- Events leading up to current condition
	- Basic medical hx
Physical examination	- Assessment of neurologic status
	- Pupillary size, reaction to light
	- V/S, incl. temp.
Ongoing monitoring	- Assess for, report manifestations of IICP every 15–60 minutes
	- Look for trends
	- Sudden changes may indicate deterioration
	- Subtle change may be early sign of declining neurologic condition

Monitor pulse oximetry, ABGs

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ASESSMENT (cont)

If device to monitor IICP is in place	- Recording readings
	- Assess patency of catheter
	- Monitor insertion site for s/s of infection

NSG DX

PLANNING

IMPLEMENTATION

Ensure adequate oxygenation
Reduce intracranial pressure
Reduce environmental stimulation
Reduce environmental stimulation
Prepare patient and family for discharge

EVALUATION

Expected outcomes may include	- Patient's ICP returns to acceptable limits following treatment
	- Patient's LOC improves with reduction of ICP
	- Patient experiences no infection as result of ICP monitoring
	- Family describes appropriate outcome expectations
	- Patient and family institute, maintain adequate safety measures after discharge

EVALUATION (cont)

Some patients require days, weeks, or months of monitoring for ICP changes	- Reassess plan of care to be relevant to patient's current condition