

Increased ICP (pressure in brain)

The skull is a closed box — nothing can expand inside without crushing something else.

THE STORY IN ONE SENTENCE

The skull is a closed box — nothing can expand inside without crushing something else.

“Increased ICP (pressure inside the skull) — the box is full, and the brainstem gets squeezed toward the exit.”

Normal anatomy — the body's job

The brain lives inside a rigid skull that cannot stretch. Inside are three things that share the space: brain tissue, blood in arteries and veins, and cerebrospinal fluid (CSF) made by the choroid plexus in the brain's hollow chambers (ventricles). CSF flows from the ventricles through narrow channels, around the brain and spinal cord in the subarachnoid space, and drains into veins through one-way valves called arachnoid granulations. Blood flows in through arteries and out through veins. Pressure inside the skull (intracranial pressure, or ICP) normally stays between 5 and 15 mmHg — low and steady.

BODY'S JOB

The job is to keep brain blood flow (perfusion) pressure (CPP) high enough so oxygen-rich blood reaches every neuron. CPP equals mean arterial pressure (the push of blood coming in) minus intracranial pressure (the squeeze pushing back). Healthy brains auto-regulate: when blood pressure changes, small arteries inside the brain widen or tighten to keep flow steady between about 50 and 150 mmHg. CSF production and drainage stay balanced so fluid does not pile up. The Monroe-Kellie doctrine says the skull is a closed box with fixed volume, so if one part (blood, CSF, or brain tissue) gets bigger, another must shrink or pressure rises.

1. Heart pumps blood → carotid and vertebral arteries carry it into the skull.
2. Blood flows through brain capillaries, delivering oxygen and glucose to neurons.
3. Choroid plexus in the ventricles makes about 500 mL of clear CSF every day.

4. CSF flows from lateral ventricles → through the foramen of Monro → third ventricle → aqueduct of Sylvius → fourth ventricle → out through foramina of Luschka and Magendie into the subarachnoid space around the brain and spinal cord.
5. CSF cushions the brain and drains into the venous system through arachnoid granulations, keeping volume and pressure steady.
6. Veins collect used blood and drain it out through the jugular veins.
7. At every moment, $CPP = MAP - ICP$. If ICP climbs, CPP drops unless MAP rises to compensate.

What breaks

Something takes up extra space inside the closed skull, and pressure climbs. The causes fall into four groups: a mass (tumor, blood clot, abscess), brain swelling (from stroke, trauma, liver failure, or infection), blocked CSF flow (hydrocephalus), or backed-up venous blood (clot in a big vein or jugular compression). At first the body squeezes out CSF and venous blood to make room. Once that buffer runs out, ICP shoots up fast. High ICP squeezes blood vessels, starving the brain, and pushes brain tissue toward openings in the skull (herniation). The brainstem — which controls breathing, heart rate, and wakefulness — sits right above the biggest opening (foramen magnum), so it gets crushed first.

CLINICAL TERM

Intracranial high blood pressure (hypertension) ($ICP > 20\text{--}22$ mmHg sustained). Causes: mass effect (hematoma, tumor, abscess), cerebral swelling (edema) (cytotoxic from blood-starved (ischemic) stroke or trauma, vasogenic from tumor or infection, interstitial from hydrocephalus), obstructive hydrocephalus (aqueductal narrowing (stenosis), posterior fossa mass, meningitis blocking CSF drainage), communicating hydrocephalus (subarachnoid bleeding (hemorrhage), meningitis impairing arachnoid granulations), venous sinus clot formation (thrombosis), or with no known cause (idiopathic) intracranial high blood pressure. When ICP rises, CPP falls ($CPP = MAP - ICP$). Below CPP $\sim 50\text{--}60$ mmHg, starved blood flow (ischemia) begins. Autoregulation fails under sustained high ICP or MAP extremes, making flow pressure-passive. The Monroe-Kellie doctrine predicts exponential pressure rise once compensatory reserve (CSF displacement into spinal canal, venous compression) exhausts. Herniation syndromes follow pressure gradients.

How the body compensates

The body has a narrow safety buffer. At first it squeezes CSF down into the spinal canal and compresses veins, keeping ICP steady even as volume rises. Once that reserve is gone, ICP climbs steeply — the compliance curve goes vertical. Vital signs may look stable during

compensation (normal blood pressure, normal heart rate), hiding how close the patient is to the crash point. The danger zone is herniation: brain tissue squeezes through skull openings, crushing the structures that keep you alive.

1. {'what': 'CSF displacement', 'how': 'About 150 mL of CSF moves from the skull down into the stretchy spinal subarachnoid space. This buys time but exhausts quickly.'}
2. {'what': 'Venous compression', 'how': 'Veins inside the skull are thin-walled and collapse easily. Squeezing them out reduces blood volume and keeps ICP from rising — until veins are fully flattened.'}
3. {'what': 'Cushing reflex (late sign)', 'how': 'When ICP climbs high enough to squeeze the brainstem's vasomotor center (in the medulla), sympathetic neurons fire massively. Arteries all over the body clamp down, shooting MAP up to force blood past the high ICP and protect CPP. You see severe high blood pressure (hypertension) (MAP sometimes >130 mmHg). This is the body's last-ditch effort to keep the brain alive.'}
4. {'what': 'Reflex a slow heart rate (bradycardia) (Cushing triad component)', 'how': 'Baroreceptors in the aortic arch and carotid sinus sense the dangerously high MAP and trigger the vagus nerve to slow the heart, trying to bring pressure back down. You see a slow, bounding pulse. Cushing triad = high blood pressure (hypertension) + a slow heart rate (bradycardia) + irregular breathing.'}
5. {'what': 'Hyperventilation attempt (then failure)', 'how': 'Rising CO₂ and falling O₂ stimulate the respiratory centers in the medulla and pons, driving faster, deeper breaths to blow off CO₂ and lower ICP (lower CO₂ → vasoconstriction → less blood volume). As brainstem compression worsens, breathing becomes irregular (Cheyne-Stokes, uncoordinated (ataxic), or apneustic) and eventually stops.'}

Domino effect

1. Volume inside the closed skull rises. — A mass (blood, tumor, abscess), brain swelling (edema from stroke, trauma, infection, liver failure), CSF buildup (hydrocephalus), or backed-up venous blood adds volume. The skull is rigid bone and cannot stretch, so pressure inside (ICP) must rise. At first, CSF squeezes down into the spinal canal and veins collapse, keeping ICP stable — this is the flat part of the compliance curve.
2. Compensatory reserve exhausts and ICP shoots up. — Once all the movable CSF is gone and veins are fully flattened, even a tiny bit more volume makes ICP climb steeply — the compliance curve goes vertical. Now ICP can jump from 15 to 40 mmHg in minutes.
3. Cerebral blood flow (perfusion) pressure (CPP) drops. — $CPP = MAP - ICP$. If ICP climbs and MAP stays the same, CPP falls. Below about 50–60 mmHg, blood flow cannot deliver enough oxygen, and neurons start to die (ischemia). Starved blood flow (Ischemia) makes cells swell (cytotoxic swelling (edema)), which raises ICP even more — a vicious cycle.

4. Brainstem compression triggers the Cushing reflex. — High ICP squeezes the medulla (the lowest part of the brainstem), crushing the vasomotor center (a cluster of neurons that controls blood vessel tone). These neurons sense they are being strangled and fire in a panic, sending a massive sympathetic surge through the whole body. Arteries everywhere clamp down hard, shooting blood pressure way up (sometimes systolic >200 mmHg). This is the body's last attempt to force blood past the high ICP and keep CPP above the starved blood flow (ischemia) threshold.
5. Heart rate slows (reflex a slow heart rate (bradycardia)). — Baroreceptors (pressure sensors) in the aortic arch and carotid arteries detect the dangerously high blood pressure and signal the vagus nerve (cranial nerve X) to slam the brakes on the heart. The pulse drops, sometimes to 40–50 bpm, trying to protect blood vessels from rupturing. Cushing triad = high blood pressure (hypertension) + a slow heart rate (bradycardia) + irregular respirations, and it is a late, ominous sign.
6. Breathing becomes slow, irregular, or stops. — The medulla and pons contain the respiratory centers (groups of neurons that set the rhythm and depth of each breath). As ICP crushes these centers, the normal smooth breathing pattern breaks down. You see Cheyne-Stokes (cycles of deep breaths, then shallow, then pauses), uncoordinated (ataxic) breathing (completely chaotic), or apneustic breathing (long gasping inhales with pauses). Eventually breathing stops (apnea) as the medulla fails.
7. Pupils dilate and stop reacting to light. — The oculomotor nerve (cranial nerve III) runs along the edge of the tentorium (a shelf of dura inside the skull) and controls the sphincter muscle that shrinks the pupil. When rising pressure pushes the uncus (part of the temporal lobe) or the whole brain downward (herniation), CN III gets stretched and compressed against the tentorial edge or the posterior cerebral artery. Parasympathetic fibers (which constrict the pupil) run on the outside of the nerve and are crushed first, so the pupil dilates and becomes fixed (blown pupil). This often starts on one side (ipsilateral to the mass) and is a neurosurgical emergency.
8. Posturing appears: decorticate, then decerebrate. — As the brain herniates downward, it shuts off upper control centers in stages. Decorticate posturing (arms flexed tight to the chest, legs stiff and straight) means the cerebral cortex is offline but the red nucleus (in the midbrain) still has some control. Decerebrate posturing (all limbs extended, arms rotated inward, back arched) means the midbrain is now crushed and only the brainstem (pons, medulla) is firing. Posturing is not a seizure; it is the brain losing its wiring from top to bottom.

9. Herniation becomes irreversible; brain death follows. — Herniation compresses arteries (posterior cerebral, basilar), cutting off blood supply to the brainstem. Neurons die within minutes without oxygen. Even if ICP is lowered with medicine or surgery, the crushed tissue cannot recover. Tonsillar herniation (cerebellar tonsils pushed through the foramen magnum) compresses the medulla so suddenly that breathing and heartbeat stop almost immediately, causing death before treatment can help. Every second counts once herniation starts.

Symptoms · what the patient feels, and why

- Headache that gets worse when lying down or in the morning: When pressure builds inside the skull, it squeezes pain-sensing tissue around the brain. Lying flat or sleeping lets fluid pool in the head, which pushes pressure even higher.
- Throwing up without feeling sick to the stomach first (projectile vomiting): High pressure presses on the area postrema (a trigger zone at the base of the brain that controls vomiting), setting off the vomit reflex without the usual warning nausea.
- Blurry vision or brief moments when vision goes dark (visual obscurations): Pressure travels along the optic nerve (the cable that sends pictures from the eye to the brain) and squeezes it, blocking signals and sometimes making the back of the eye swell (papilledema).
- Feeling confused, sleepy, or hard to wake up: Rising pressure squeezes the reticular activating system (a network deep in the brainstem that keeps you alert and awake), slowing down the signals that keep your mind clear.
- One pupil becomes larger than the other or stops shrinking in bright light: Pressure pushes part of the brain (the uncus of the temporal lobe) down and traps the oculomotor nerve (cranial nerve III, which controls pupil size). The trapped nerve can't send the 'shrink' signal anymore.
- Seizures: High pressure irritates neurons (brain cells that fire electrical signals), and when many fire together all at once in a chaotic wave, a seizure happens.
- Cushing triad: high blood pressure, slow heart rate, and irregular breathing: This is a last-ditch response when the brainstem (the control tower for breathing, heart rate, and blood pressure) is being crushed. The body cranks up blood pressure to try to force blood into the squeezed brain, the vagus nerve (cranial nerve X) slows the heart in response, and the breathing centers in the medulla (the lowest part of the brainstem) fire unevenly, making breaths choppy or slow.
- Stiff neck or being forced into an arched-back position (opisthotonus): Pressure irritates the motor pathways in the brainstem, causing muscles along the spine and neck to lock tight and pull the body backward.

Focused exam

- Pupil size and light response — one side bigger than the other, or both huge and stuck: The third cranial nerve (the nerve that tells the pupil to squeeze smaller) runs right next to the edge of the brain. When rising pressure pushes the uncus (a hook-shaped part of the brain) downward, it pinches that nerve. A blown pupil (big and stuck, no shrinking to light) on one side means uncal herniation is starting on that side. This is the single most important sign that the brain is being squeezed hard enough to shift downward.
- Blood pressure high, heart rate slow, breathing irregular — Cushing triad: When rising pressure squeezes the brainstem (the stalk at the bottom of the brain that controls breathing, heart, and blood pressure), the body tries a last reflex to save itself. It cranks blood pressure way up to force blood into the skull. At the same time, the vagus nerve (a big nerve that slows the heart) fires, slowing the pulse. Irregular breathing means the brainstem's breathing center is already failing. This triad is a late sign — the patient is in immediate danger of dying.
- Posturing — arms bent in toward chest (decorticate) or arms and legs locked straight (decerebrate): Posturing shows where the squeeze is hitting the brain. Decorticate posturing (arms bent, legs straight) means pressure is damaging the cerebral cortex (the thinking outer layer) and upper brainstem. Decerebrate posturing (everything stiff and straight, like a board) means the damage has moved lower, closer to the brainstem. Each pattern tells you how far down the herniation has gone.
- One-sided weakness or one side of the face drooping: When uncal herniation happens, the brain bulges downward on one side. The third nerve and the nearby motor pathways (the wires that carry movement signals) get pinched first on that side. This causes weakness or paralysis on the opposite side of the body, because motor signals cross over in the brainstem.
- Change in alertness — from awake to sleepy to not waking up: The skull is a rigid box. When pressure rises, blood flow to the brain drops because the squeezing chokes off the arteries. The reticular activating system (the part of the brainstem that keeps you awake) stops working. The patient gets drowsy, then harder to wake, then finally unresponsive. This downward slide means the whole brainstem is being crushed.
- Headache that is new, severe, or different from any headache before: A sudden, terrible headache means the skull is overfilled and the meninges (the wrapping around the brain) are being stretched. But remember — no headache does not mean no high pressure. A patient who is intubated, sedated, or unconscious cannot complain. Trust the exam findings more than the words.

Diagnostics · every test answers a question

- Non-contrast head CT (computed tomography scan) → Is there a mass (tumor, blood clot, abscess), swelling, or blocked cerebrospinal fluid (CSF) flow causing the pressure to rise?
- Intracranial pressure (ICP) monitor — intraventricular catheter or intraparenchymal bolt → What is the actual pressure inside the skull right now, and is it high enough to hurt the brain?
- Cerebral blood flow (perfusion) pressure (CPP) calculation — MAP minus ICP → Is enough blood getting through the squeeze to feed the brain?
- Serum sodium and serum osmolality → Is the blood salty and thick enough to pull water out of the swollen brain?
- Arterial blood gas (ABG) — specifically PaCO₂ → Is the CO₂ level in the blood low enough to shrink brain blood vessels and buy time?
- Fundoscopic exam (looking at the back of the eye with an ophthalmoscope) → Is the optic nerve swollen from pressure backing up from the brain?

Treatment logic

- Head of bed elevated 30 degrees, neck midline and straight → Gravity helps venous blood drain out of the jugular veins in the neck, lowering blood volume inside the rigid skull. Keeping the neck straight prevents kinking the veins. This lowers ICP by a few mmHg within minutes.
- Hypertonic saline (3% continuous infusion or 23.4% bolus) or mannitol 0.25–1 g/kg IV → These hyperosmolar agents pull water out of the swollen brain tissue into the bloodstream through osmosis (water follows salt). This shrinks brain volume and lowers ICP within 15–30 minutes. Hypertonic saline is preferred in trauma because it also supports blood pressure (Neurocritical Care Society). Mannitol works but can drop blood pressure.
- Sedation and analgesia — propofol infusion, fentanyl boluses → Sedation lowers the brain's metabolic rate (how much oxygen and glucose it burns), which lowers cerebral blood flow and ICP. Pain and agitation spike ICP by raising blood pressure and causing the patient to fight the ventilator. Propofol also has direct neuroprotective effects.
- Controlled hyperventilation to PaCO₂ 30–35 mmHg → Lowering CO₂ causes cerebral vasoconstriction (brain arteries squeeze smaller), reducing blood volume in the skull and lowering ICP fast. This is a rescue intervention for acute herniation, not a sustained strategy.
- Drain cerebrospinal fluid via external ventricular drain (EVD) → Draining CSF directly removes volume from the skull box, immediately lowering ICP. This is the fastest mechanical intervention. It also allows continuous ICP monitoring.

- Decompressive craniectomy (removing part of the skull) → Removes the rigid skull bone over part of the brain, allowing the swollen brain to bulge outward instead of downward. This stops herniation and lowers ICP immediately. Used in malignant cerebral swelling (edema) (massive stroke), refractory traumatic brain injury, and some hemorrhages.
- Definitive source control — evacuate a pooled collection of blood (hematoma), resect tumor, shunt hydrocephalus → Removes the actual thing taking up space or unblocks the CSF flow. This fixes the root cause, not just the symptom. A pooled collection of blood (Hematoma) evacuation (removing a blood clot) is guideline-recommended for certain bleeds > 30 mL with midline shift (AHA/ASA stroke guidelines). VP shunt (ventriculoperitoneal shunt) fixes obstructive hydrocephalus permanently.

Medications

Hypertonic saline (3% or 23.4%) · Osmotic agent (a fluid that pulls water across membranes)

Creates a salt gradient that pulls extra water out of swollen brain tissue and into the bloodstream, where the kidneys can flush it out. Think of it like sprinkling salt on a wet sponge—the salt draws the water toward it.

1. Hypertonic saline enters the bloodstream and raises the salt (sodium) level in the blood vessels running through the brain.
2. Because the blood now has more salt than the brain tissue, water follows the salt—moving from brain tissue into the blood vessels.
3. This shrinks the swollen brain tissue and lowers the pressure inside the skull.
4. The kidneys filter the extra water and salt out as urine within hours.

SAFETY WATCH

Can cause blood sodium to climb too high (hypernatremia), which makes cells throughout the body shrivel and can trigger seizures or confusion. Check sodium level every 4 to 6 hours. · Can overload the bloodstream with fluid and strain the heart, especially in people with heart failure. Watch for lung crackles and shortness of breath. · Repeated doses can cause a rebound—when the effect wears off, pressure may spike even higher. Use only as a bridge to definitive treatment. · Must go through a central IV line (a line placed in a large vein near the heart) if using 23.4%, because it is so salty it can damage smaller veins. · Can cause low potassium and low calcium; check levels and replace as needed.

Mannitol · Osmotic diuretic (a sugar alcohol that pulls water into blood vessels and then into urine)

Works like hypertonic saline but uses a sugar molecule instead of salt. Mannitol stays in the blood vessels and pulls water out of brain tissue, then the kidneys flush it all out as urine within 30 to 60 minutes.

1. Mannitol is given as an IV bolus and stays inside the blood vessels—it does NOT cross into brain tissue.
2. Because mannitol molecules are large and concentrated, they pull water from the swollen brain into the bloodstream (osmosis).
3. This shrinks the brain tissue and drops the pressure inside the skull within 15 to 30 minutes.
4. The kidneys filter out the mannitol along with the extra water, creating a large amount of urine quickly.

SAFETY WATCH

Can cause sudden dehydration and low blood pressure because it pulls so much water into urine so fast. Monitor blood pressure and urine output closely. · Can damage the kidneys (acute tubular tissue death (necrosis)) if used repeatedly or in high doses, especially if the patient is already dry. Check creatinine. · Can cause rebound swelling—mannitol may leak into damaged brain tissue, then pull water back IN and make pressure worse when it wears off. Limit to 3 to 4 doses in 24 hours. · Do NOT give if the patient has severe kidney failure or is anuric (making no urine)—mannitol will build up and cause fluid overload and heart failure. · Can cause electrolyte imbalances (low sodium, low potassium). Check labs every 6 to 12 hours.

Sedatives (propofol, midazolam, or dexmedetomidine) · Sedative-hypnotics and anesthetics (drugs that calm the brain and lower its activity)

Slow down the brain's electrical firing and reduce how much oxygen and glucose the brain demands. When the brain works less hard, it makes less waste heat and swelling, and blood vessels can shrink a bit—all of which lowers pressure inside the skull. Think of it like dimming the lights in a busy factory so it uses less power and gives off less heat.

1. Sedatives bind to GABA-A receptors (the brain's main calming receptors) and make them stay open longer.
2. This floods neurons with chloride ions, which makes it harder for the neuron to fire an electrical signal.
3. Quieter neurons use less oxygen and glucose, so brain metabolism drops by 30 to 50 percent.

4. Lower metabolism means less heat, less carbon dioxide, and less swelling—so pressure inside the skull falls.
5. Sedation also prevents the patient from coughing, straining, or fighting the breathing tube, all of which would spike pressure.

SAFETY WATCH

Propofol can cause very low blood pressure, which is dangerous because the brain needs good blood flow. May need IV fluids or pressors (drugs to raise blood pressure) at the same time. · Propofol infusion syndrome is a rare but deadly complication (muscle breakdown, heart failure, severe too much acid in the blood (acidosis)) if propofol is used at high doses for more than 48 hours. Watch for rising creatine kinase, lactate, and triglycerides. · All sedatives make it impossible to check the neurologic exam (pupil response, movement, speech), so doctors fly blind. Use the lowest dose that controls pressure and agitation. · Midazolam can build up in the body and cause prolonged sedation, especially in kidney or liver failure. · Dexmedetomidine can cause slow heart rate (bradycardia) and low blood pressure, but it allows a lighter sedation so the exam can be checked more often.

Pain control (fentanyl, morphine, or other opioids) · Opioid analgesics (pain relievers that work in the brain and spinal cord)

Block pain signals in the spinal cord and brain so the patient does NOT thrash, strain, or tense up—all of which would spike pressure inside the skull. Pain and stress also trigger the release of adrenaline and cortisol, which raise blood pressure and brain blood flow, pushing pressure even higher. Opioids stop that cascade.

1. Opioids lock onto mu receptors in the spinal cord and brainstem.
2. This blocks the release of substance P and other pain messengers, so pain signals do NOT climb to the brain.
3. Less pain means less sympathetic surge (the fight-or-flight response), so heart rate, blood pressure, and stress hormones stay lower.
4. Lower blood pressure and less agitation mean less blood is forced into the brain, which helps keep intracranial pressure stable.

SAFETY WATCH

Can cause slow, shallow breathing (respiratory depression), which raises carbon dioxide in the blood. High CO₂ dilates brain blood vessels and RAISES intracranial pressure—the opposite of what you want. If the patient is on a ventilator, the machine controls breathing so this is less of a problem, but still monitor closely. · Can lower blood pressure, which is risky because the brain needs strong blood flow to survive high pressure (cerebral blood flow (perfusion) pressure = blood pressure minus intracranial pressure). · Can cause constipation, nausea, and confusion, especially with repeated doses. · All opioids cloud the neurologic exam, so use the lowest effective dose.

Short-term hyperventilation (lowering PaCO₂ to 30–35 mmHg) · Mechanical ventilation strategy (not a drug, but a breathing adjustment)

Breathing the patient faster blows off carbon dioxide (CO₂). Low CO₂ makes brain blood vessels squeeze tight (vasoconstriction), which shrinks the volume of blood inside the skull and drops pressure fast—within seconds. Think of it like letting air out of a balloon to make it smaller.

1. The ventilator is set to breathe the patient faster and deeper than normal.
2. Extra breaths blow CO₂ out of the lungs, so blood CO₂ (PaCO₂) drops.
3. Cerebral blood vessels sense the low CO₂ and the rising pH (less acidic blood) and respond by constricting.
4. Narrower vessels mean less blood volume in the brain, so intracranial pressure falls within 30 seconds.

SAFETY WATCH

Drops cerebral blood flow so much that it can cause starved blood flow (ischemia), especially in areas already on the edge. Monitor with transcranial Doppler or brain oxygen monitors if available. · Effect fades within 4 to 24 hours as the brain compensates. Do NOT keep doing it—aim to wean back to normal CO₂ (35–40 mmHg) as soon as possible. · Can cause too little acid in the blood (alkalosis) (blood becomes too alkaline), which shifts the oxygen-hemoglobin curve and makes it harder for tissues to grab oxygen. · Guidelines say: use hyperventilation ONLY for acute herniation (brain is being squeezed down through the skull) while racing to the operating room. NOT a long-term strategy.

Positioning (head of bed elevated 30°, neck midline) · Supportive care measure (not a drug)

Gravity helps blood and spinal fluid drain out of the brain through the jugular veins (the big veins in the neck). Keeping the neck straight prevents kinking or squashing those veins. This

lowers the total volume inside the skull and drops pressure gently. Think of it like tilting a bottle so liquid flows out more easily.

1. Elevating the head lets gravity pull blood down and out of the brain veins.
2. Keeping the neck straight prevents the jugular veins from being pinched or twisted.
3. Better drainage means less blood pooled in the skull, so intracranial pressure drops by a few mmHg.
4. This also improves venous return to the heart without compromising arterial flow into the brain.

SAFETY WATCH

Do NOT elevate the head if the patient is in shock or has a spinal cord injury that has not been cleared—keeping them flat may be necessary to maintain blood flow. · Make sure the neck is truly midline and NOT rotated, flexed, or extended—any twisting can kink the jugular veins and RAISE pressure. · Use this measure in EVERY patient with suspected high intracranial pressure unless there is a specific reason not to. It is simple, safe, and costs nothing.

Red flags

- Confusion, extreme sleepiness, or impossible to wake up
- One pupil much larger than the other or not reacting to light
- Sudden seizure
- Cushing triad: very high blood pressure, slow pulse, and weird breathing pattern
- Sudden severe headache described as 'the worst of my life'

Nursing priorities

1. Position the head of bed at 30 degrees and keep the patient's head and neck in straight midline alignment without any rotation or flexion — Elevating the head lets gravity help drain venous blood and cerebrospinal fluid away from the brain, and keeping the neck straight prevents kinking of the jugular veins (neck veins) which would trap blood in the head and raise pressure higher
2. Perform neurological assessments every 1 hour using Glasgow Coma Scale (a 15-point scoring system measuring eye opening, verbal response, and motor movement) and pupil checks with a penlight — Declining Glasgow Coma Scale score or a pupil that becomes large and fixed signals brain herniation (brain tissue squeezing downward) is starting, giving only minutes to intervene before permanent brain damage or death

3. Maintain continuous ICP monitoring via the external ventricular drain (EVD) or intraparenchymal bolt (a pressure probe inserted into brain tissue), keeping ICP below 22 mmHg and cerebral blood flow (perfusion) pressure (CPP) above 60 mmHg — Cerebral blood flow (perfusion) pressure equals mean arterial pressure minus ICP—it measures actual blood flow reaching brain tissue—and when CPP drops below 60 mmHg the brain does not get enough oxygen and cells begin dying
4. Administer hypertonic saline 3% continuous infusion or give 23.4% saline 30 mL bolus over 5 minutes through a central line as ordered, checking serum sodium (blood salt level) every 4 to 6 hours and keeping it between 145 and 155 mEq/L — Hypertonic saline pulls water out of swollen brain tissue through osmosis (water moving toward higher salt concentration), shrinking the brain and lowering pressure, but sodium above 160 mEq/L causes dangerous muscle weakness and seizures
5. Provide adequate sedation with propofol infusion (typical 20 to 50 mcg/kg/min) and analgesia with fentanyl boluses (25 to 100 mcg IV every 1 to 2 hours) to keep the patient calm and comfortable — Pain, anxiety, and agitation make the patient strain and fight the ventilator which spikes blood pressure and intrathoracic pressure (pressure in the chest), forcing more blood into the head and increasing ICP
6. Maintain mechanical ventilation with PaCO₂ (carbon dioxide level in arterial blood) at 30 to 35 mmHg if hyperventilation is ordered, monitoring with continuous end-tidal CO₂ and arterial blood gases every 4 hours — Lower CO₂ causes vasoconstriction (blood vessel narrowing) in the brain which reduces blood volume inside the skull and drops ICP quickly, but going below 30 mmHg cuts off oxygen delivery and causes starved blood flow (ischemia) (tissue oxygen starvation)
7. Drain cerebrospinal fluid in 2 to 3 mL increments through the external ventricular drain when ICP rises above 20 mmHg, recording output color and amount hourly — Removing cerebrospinal fluid directly decreases volume inside the fixed skull space and lowers pressure immediately, and cloudy or bloody drainage signals infection or new bleeding that needs urgent physician notification
8. Avoid any unnecessary stimulation including suctioning, bathing, or repositioning during the same time period, and pre-medicate with lidocaine 1.5 mg/kg IV and extra sedation before required procedures — Each stimulation causes a pressure spike—clustering care and pre-medicating blunts these spikes and prevents cumulative ICP elevations that could trigger herniation

Sources

- Neurocritical Care Society
- Brain Trauma Foundation ICP guidelines
- AAN (American Academy of Neurology)

- NEJM clinical review on intracranial hypertension

Last reviewed: 2026-07-07