

Increased Icp and Herniation

High brain pressure? Raise the head of the bed 30 degrees. Give hypertonic saline or mannitol to pull fluid out of the brain. Keep the patient calm and quiet. Watch pupils and breathing every hour. Brain surgery may be needed fast to remove the mass or drain fluid.

THE ONE-LINE TRUTH

When the brain swells or a mass grows inside the hard skull, pressure climbs and squeezes the brainstem—the part that keeps you breathing and awake.

The chain — how it unfolds

1. A mass (tumor, a pooled collection of blood (hematoma), abscess), cerebral swelling (edema) (from trauma, stroke, infection), blocked CSF drainage (hydrocephalus), or venous clot adds volume inside the rigid skull. — why: The skull is a closed box that cannot stretch. Extra volume has nowhere to go, so intracranial pressure (ICP) begins to climb. At first the body compensates by shifting CSF and blood out, keeping pressure normal and symptoms mild—maybe just a dull headache or mild confusion.
2. As the mass or swelling grows, compensation runs out. Pressure spikes. Blood vessels get squeezed, cutting oxygen delivery to brain cells (ischemia). — why: Cerebral blood flow (perfusion) pressure (CPP) equals mean arterial pressure minus ICP. When ICP rises above 20–30 mm Hg, CPP falls below the 50–60 mm Hg needed for cells to survive. Starved of oxygen, cells swell further (secondary swelling (edema)), creating a vicious cycle: more swelling raises pressure, which cuts flow, which causes more swelling.

3. You see classic signs: severe headache (worse lying flat or in the morning), projectile vomiting (from pressure on the brainstem's vomiting center, the area postrema), swelling of the optic nerve (papilledema) (swollen optic disc from blocked venous drainage), and progressive drowsiness (from starved blood flow (ischemia) in the reticular activating system, the brainstem network that keeps you awake). — why: High ICP stretches pain-sensitive structures (dura, blood vessels), so the headache is deep and throbbing. Lying flat or bending over raises venous pressure, making it worse. Vomiting happens because pressure irritates the medulla. Swelling of the optic nerve (Papilledema) develops over hours to days as CSF pressure transmits along the optic nerve sheath, stalling venous return and swelling the disc. Drowsiness progresses from confusion to stupor to coma as blood flow (perfusion) falls and the reticular activating system shuts down.
4. Rising pressure pushes the brain downward. In uncal herniation the medial temporal lobe (uncus) squeezes through the tentorial notch, compressing cranial nerve III (oculomotor nerve) and the midbrain. You see a fixed, dilated pupil on the same side as the mass (the nerve's parasympathetic fibers stop working, so the pupil cannot constrict) and then weakness on the opposite side of the body (the cerebral peduncle carrying motor signals gets crushed). — why: The uncus herniates because it sits right at the edge of the tentorium, the dura fold separating brain and cerebellum. Nerve III runs alongside, so it gets pinched first—parasympathetic fibers on the outside of the nerve are most vulnerable, causing pupil dilation (mydriasis). Continued herniation crushes the midbrain's motor tracts (corticospinal tract in the cerebral peduncle), causing contralateral weakness on one side (hemiparesis) or posturing.
5. The brainstem tries to save itself by triggering Cushing's reflex: blood pressure shoots up (hypertension) to force blood through the squeezed vessels, and the heart rate slows (bradycardia) because the vagus nerve (cranial nerve X in the medulla) fires in response to high pressure. Breathing becomes slow and irregular (Cheyne-Stokes or uncoordinated (ataxic) pattern) as the medulla's respiratory centers fail. — why: The Cushing reflex is a last-ditch effort by the medulla to maintain cerebral blood flow (perfusion). The medulla detects starved blood flow (ischemia) and sends a massive sympathetic surge, raising systemic blood pressure. The high pressure stretches baroreceptors in the carotid sinus and aortic arch, triggering a vagal reflex that slows the heart. This triad—high blood pressure (hypertension), a slow heart rate (bradycardia), irregular respirations—is a late and ominous sign that herniation is imminent or already happening.

6. In central transtentorial herniation both cerebral hemispheres push down, compressing the diencephalon (thalamus and hypothalamus) and then the midbrain. Pupils become small and reactive at first, then mid-position and fixed. The patient develops decorticate posturing (arms flexed, legs extended—damage above the midbrain) and then decerebrate posturing (arms and legs extended—damage at or below the midbrain). — why: Central herniation happens when pressure rises evenly across both sides—often from diffuse swelling (edema). As the diencephalon gets squeezed, you lose higher control: small reactive pupils (intact midbrain), then Cheyne-Stokes breathing (damage to cortex and diencephalon). When the midbrain herniates, pupils fix in mid-position (parasympathetic and sympathetic pathways both fail), and posturing worsens from decorticate (red nucleus intact) to decerebrate (red nucleus lost, only vestibular reflexes remain).
7. In tonsillar herniation the cerebellar tonsils push down through the foramen magnum, crushing the medulla. Breathing stops suddenly (apnea) because the medulla's respiratory center shuts off. Blood pressure crashes, and the heart stops soon after. — why: The foramen magnum is the only exit at the skull base, where the brainstem becomes the spinal cord. The medulla sits here and controls automatic breathing, heart rate, and blood pressure. When the tonsils herniate and compress it, you lose respiratory drive—the patient stops breathing and becomes bradycardic, progressing to cardiac arrest within minutes. This is the final common pathway of death from increased ICP.
8. If you trace every finding backward—headache, vomiting, swelling of the optic nerve (papilledema), dilated pupil, slow heart, high blood pressure, abnormal posturing, irregular breathing—they all point upstream to one broken thing: rising intracranial pressure from added volume in a closed space. The fix is not to chase each symptom but to lower ICP fast (elevate head, give hyperosmolar therapy, hyperventilate briefly, drain CSF, or remove the mass surgically) and restore blood flow (perfusion) before herniation becomes irreversible. — why: Every sign you see is the body's attempt to cope with too much pressure—pushing fluid out, raising blood pressure, shutting down consciousness to lower metabolic demand. But compensation eventually fails. The only way to save the patient is to remove the cause: shrink the brain (mannitol or hypertonic saline pulls water out), drain CSF (ventriculostomy), remove the mass (craniotomy for a pooled collection of blood (hematoma) or tumor), or treat the underlying problem (antibiotics for abscess, antiedema measures for stroke). Once herniation is complete and the medulla is crushed, brain death follows, so early recognition and aggressive ICP management are life-saving.

Spot it — what you see, and why

- Headache that gets worse and worse: The rising pressure inside the skull pushes on pain-sensitive tissue around the brain (like the dura, which is the tough outer cover). The brain itself has no pain nerves, but the structures around it do, so they signal pain as they stretch.

- Throwing up without feeling sick to the stomach first: High pressure presses on the area postrema (the vomit-trigger zone in the brainstem at the base of the brain). This center fires off a vomit reflex even though there is no stomach problem — the signal comes from inside the skull.
- One pupil gets bigger than the other, or both pupils stop reacting to light: When part of the brain (often the temporal lobe) herniates (gets squeezed downward), it traps the oculomotor nerve (cranial nerve III, which controls pupil size). The nerve stops working, so the pupil stays wide open and will not shrink when you shine a light in the eye.
- Vision gets blurry or dims, sometimes seeing double: Pressure inside the skull squeezes the optic nerves (the cables that carry vision from the eye to the brain) and can also press on the abducens nerve (cranial nerve VI, which moves the eye outward). Squeezing the optic nerve clouds vision; pinching nerve VI causes double vision because the eyes do not line up.
- Confusion, trouble staying awake, or falling into a coma: Rising pressure squeezes the reticular activating system (a network in the brainstem that keeps you awake and alert). When it gets compressed, the brain's wake-up signal fades and the person becomes drowsy, confused, and eventually unconscious.
- Body suddenly gets stiff with arms bent inward or stretched out straight (posturing): Herniation cuts off signals from the cerebral cortex (the outer thinking layer) or damages the brainstem (the control tower for movement and posture). Without the cortex keeping the brainstem in check, primitive reflex circuits fire and lock the muscles into abnormal postures — decorticate (arms bent in) or decerebrate (arms straight out).

Red flags — act now

- One pupil suddenly much bigger than the other or not reacting to light
- Stiff posturing (arms bent in tight or stretched out straight)
- Glasgow Coma Scale dropping (person less awake, less responsive)
- Cushing's triad: high blood pressure, slow heart rate, and abnormal breathing
- Sudden severe headache with projectile vomiting

Treat it — in this order

1. Elevate the head of the bed to 30 degrees and keep the neck midline (straight, not turned) → Gravity helps venous blood and CSF drain out of the skull through the jugular veins (the big veins in the neck), lowering the volume inside and dropping ICP by several mmHg.
2. Hypertonic saline IV—3% continuous infusion or 23.4% bolus (30 mL over 10–20 minutes) → The high salt concentration in the blood pulls water out of swollen brain tissue (osmosis) and into the bloodstream, shrinking the brain and dropping ICP within minutes. Target serum sodium is 145–155 mEq/L. Effect lasts 4–6 hours.

3. Mannitol IV—0.25–1 g/kg bolus over 15 minutes → Mannitol (an osmotic diuretic, a drug that pulls water into the blood and makes you pee) pulls water out of the brain into the blood, then the kidneys remove it. ICP drops in 10–30 minutes. Effect lasts 2–6 hours.
4. Controlled hyperventilation—target PaCO₂ 30–35 mmHg → Lowering CO₂ makes brain arteries constrict (squeeze tight), which shrinks the volume of blood inside the skull and drops ICP within minutes. This is the fastest emergency measure.
5. External ventricular drain (EVD)—catheter placed into the lateral ventricle to drain CSF → Removes CSF directly from the brain's fluid chambers, instantly creating space and dropping ICP. Also allows real-time ICP monitoring. Can drain 5–20 mL per hour depending on pressure.

Your top priorities at the bedside

1. Keep the head of the bed at 30 degrees and the neck straight in midline position without flexion or rotation — why: This position lets blood drain out of the brain through the jugular veins (neck veins) and keeps cerebrospinal fluid flowing; a bent or twisted neck blocks these veins and traps blood and fluid inside the skull
2. Check pupils and Glasgow Coma Scale score every 15 to 60 minutes and report any change immediately — why: A dilating pupil or dropping Glasgow Coma Scale means the brain is herniating (pushing down through the skull opening onto the brainstem) and the patient may die within minutes without emergency treatment
3. Give 3% hypertonic saline continuous IV infusion or 23.4% saline bolus as ordered and monitor serum sodium every 4 to 6 hours — why: Hypertonic saline (extra-salty IV fluid) pulls water out of swollen brain tissue into the blood vessels through osmosis (water moving toward salt); this shrinks the brain and lowers pressure