

# Intracranial Hemorrhage

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## THE STORY IN ONE SENTENCE

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“Intracranial bleeding (hemorrhage) means bleeding inside the skull; the type of bleed tells you the cause, the danger, and the fix.”

## Normal anatomy — the body's job

The skull is a rigid bony box that holds the brain, blood in vessels, and cerebrospinal fluid (the clear liquid cushion around the brain). These three—brain tissue, blood, and fluid—balance perfectly inside a fixed space, like three passengers in a car with no extra seats. The Monroe-Kellie doctrine says that because the skull cannot expand, if one part grows (like a puddle of blood), the others must shrink or pressure rises and crushes the brain.

## BODY'S JOB

The brain floats in cerebrospinal fluid and gets oxygen from blood flowing through arteries. Blood vessels inside and around the brain stay sealed so blood stays inside the pipes. The dura is a tough leather-like sheet that wraps the brain; the arachnoid is a delicate web beneath it; and the pia is the thin skin stuck to the brain surface. These layers make pockets—epidural (between skull and dura), subdural (between dura and arachnoid), subarachnoid (between arachnoid and pia, where big arteries run), and intracerebral (inside the brain tissue itself). Normally, no blood leaks into any of these spaces.

1. Blood flows into the brain through carotid and vertebral arteries, branches into smaller vessels, delivers oxygen to neurons, then drains out through veins.

2. Cerebrospinal fluid cushions the brain, absorbs shocks, and drains waste; it flows in the subarachnoid space and ventricles (fluid chambers inside the brain).
3. The skull, dura, arachnoid, and pia keep everything sealed and balanced—brain tissue at about 80%, blood at 10%, cerebrospinal fluid at 10%.

## What breaks

A blood vessel inside or around the brain bursts, and blood spills into a space where it does not belong—either into brain tissue, the fluid cushion around the brain, the gap under the dura, or the thin space above the dura next to the skull.

### CLINICAL TERM

Intracranial bleeding (hemorrhage) is rupture of a cerebral blood vessel with extravasation of blood into the parenchyma, subarachnoid space, subdural space, or epidural space. The mechanism depends on the type: trauma shears bridging veins (subdural) or tears the middle meningeal artery (epidural); chronic high blood pressure (hypertension) weakens small penetrating arterioles and they burst deep in the basal ganglia, thalamus, pons, or cerebellum (intracerebral); a saccular aneurysm (weak balloon on a brain artery, usually at the Circle of Willis) ruptures and dumps blood into the subarachnoid space; arteriovenous malformations (tangles of abnormal vessels) or tumors erode vessel walls; or anticoagulation (warfarin, DOACs, heparin) or antiplatelet drugs (aspirin, clopidogrel) prevent clotting so even small tears bleed uncontrollably. The leaked blood creates a space-occupying mass, raises intracranial pressure, disrupts the Monro-Kellie balance, triggers vasogenic and cytotoxic swelling (edema), and can herniate brain tissue through skull openings, crushing the brainstem.

## How the body compensates

When blood first spills into the skull, the body squeezes out cerebrospinal fluid and compresses veins to make room and keep pressure from spiking. The sympathetic nervous system (the body's fight-or-flight system) raises blood pressure to force blood past the rising pressure and keep the brain fed with oxygen. These tricks buy minutes to hours, so vital signs can look deceptively stable even while the brain is being crushed—this is the dangerous middle where teams miss how bad things really are. Once the skull runs out of room to give, pressure skyrockets, herniation starts, and the patient crashes fast.

1. Cerebrospinal fluid is pushed out of the skull into the spinal sac to make room for the growing blood clot—intracranial pressure stays normal for a short time.
2. Veins collapse and blood flow shifts away from less critical areas to protect the brainstem and vital centers.

3. The sympathetic nervous system fires, releasing norepinephrine (the body's main adrenaline-like messenger) and epinephrine (adrenaline itself), which jack up heart rate and blood pressure to ram blood through squeezed vessels—this is Cushing's reflex when you see high blood pressure with a slow heart rate.
4. The body keeps oxygen saturation and blood pressure looking okay on the monitor, hiding how close the brain is to the edge.
5. Once compensation maxes out, even a small new bleed or a few mL of swelling tips intracranial pressure over the cliff—pupils blow (dilate and stop reacting to light), breathing gets irregular, and the patient stops responding.
6. This is why you watch the trend—pupil changes, worsening headache, new weakness, dropping Glasgow Coma Scale, rising blood pressure with slowing heart rate—not just one set of vitals.
7. A stable patient whose exam is slowly getting worse is not stable; you are watching live decompensation and the window to intervene is closing.

## Domino effect

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1. A blood vessel inside or around the brain bursts, and blood spills into the skull. — The trigger is trauma tearing a vein or artery, chronic high blood pressure popping a deep small artery, an aneurysm rupturing, tangled vessels (AVM) breaking, a tumor eating through a vessel wall, or blood-thinner medicine preventing a normal clot from sealing a tiny leak.
2. Blood pools in a space where it does not belong—either inside brain tissue (intracerebral), in the fluid cushion around the brain (subarachnoid), under the dura between the brain and skull (subdural), or between the dura and the skull bone itself (epidural). — That puddle of blood is like a growing balloon inside a locked box; the skull cannot stretch, so the blood takes up space meant for brain and fluid.
3. The blood clot presses on brain tissue, squeezes blood vessels, and blocks oxygen from reaching neurons. — Brain cells need a constant stream of oxygen and glucose; when vessels are crushed or flow is blocked, neurons start to die within minutes (ischemia), and the tissue swells like a bruise (edema).
4. Intracranial pressure rises, triggering headache (the worst of your life if subarachnoid), confusion, vomiting, weakness or numbness on one side, slurred speech, vision loss, seizures, or loss of consciousness. — These symptoms are the brain's alarm system—different parts of the brain control movement, speech, vision, and alertness, so where the bleed lands determines which functions break first.

5. If the bleed is in the subarachnoid space (around the surface arteries), blood irritates vessels and they clamp down in spasm (vasospasm), cutting off even more blood flow days later. — Breakdown products from the leaked blood—especially oxyhemoglobin—trigger smooth muscle in artery walls to contract, causing delayed blood-starved (ischemic) stroke 3–14 days after the first bleed; this is why subarachnoid bleeding (hemorrhage) needs ICU monitoring with daily transcranial Doppler ultrasound and nimodipine (a calcium channel blocker) to prevent vasospasm.
6. As pressure keeps climbing, the brain herniates—soft tissue is pushed through openings in the skull or the tentorium (the dura shelf separating brain compartments)—compressing the brainstem. — The brainstem controls breathing, heart rate, and consciousness; when it is crushed (uncal herniation squashes the third cranial nerve and midbrain, central herniation pushes the diencephalon down, tonsillar herniation shoves the cerebellum into the foramen magnum), pupils dilate and stop reacting to light, breathing becomes irregular or stops, and the patient dies.
7. If you do not diagnose the bleed with an urgent non-contrast head CT, reverse anticoagulation (vitamin K, prothrombin complex concentrate, or idarucizumab depending on the drug), lower systolic blood pressure gently to 140 mmHg (or 110–140 for intracerebral bleeding (hemorrhage) per ATACH-2/INTERACT-2), evacuate large clots surgically when needed, clip or coil a ruptured aneurysm, give nimodipine for subarachnoid bleeds, drain cerebrospinal fluid, give mannitol or hypertonic saline to pull down swelling, and monitor in the ICU, the patient herniates and dies. — Every intervention buys time and space—stopping the bleed, pulling fluid out of the brain, draining blood or cerebrospinal fluid, and fixing the broken vessel—so the brain can survive long enough to heal; delay means irreversible neuron death and brainstem compression.
8. Even if the patient survives, leaked blood and dead tissue leave behind scar, seizures, permanent weakness, speech or swallowing problems, memory loss, or need for a shunt if cerebrospinal fluid drainage is blocked. — Neurons do not grow back; the brain reorganizes around the damage, but function lost to dead tissue or stroke from vasospasm often stays gone, and irritation from old blood and gliosis (brain scar tissue) lowers the seizure threshold for life.

## Symptoms · what the patient feels, and why

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- Sudden, severe headache (often called 'the worst headache of my life'): Blood spills into the space around the brain or into brain tissue itself, which stretches the sensitive covering of the brain (the meninges) and irritates pain-sensing nerves. The pressure inside the skull rises quickly, triggering intense pain.

- Nausea and vomiting: Rising pressure inside the skull presses on the brainstem (the part of the brain that controls automatic body functions like breathing and stomach signals), triggering the vomit reflex.
- Confusion or trouble staying awake: Blood pools and presses on brain tissue, blocking normal electrical signals. The brain's network for awareness and thought (especially the cerebral cortex and reticular activating system, which keeps you alert) can't work properly under that pressure.
- Weakness or numbness on one side of the body: The bleed damages or presses on the motor cortex or nearby pathways (the strips of brain that send movement commands down the spinal cord to muscles). When one side of the brain is hurt, the opposite side of the body loses strength or feeling.
- Slurred speech or trouble understanding words: Blood disrupts the language centers in the brain—usually Broca's area (which creates speech) or Wernicke's area (which understands words), most often in the left side of the brain. Without clean signals, words get jumbled or lost.
- Seizure: Blood spilled onto brain tissue is irritating, like pouring salt on a wound. The neurons (brain cells) around the bleed fire wildly and out of sync, creating an electrical storm that spreads as a seizure.
- Stiff neck and pain when bending the head forward: In subarachnoid bleeding (hemorrhage) (bleeding into the fluid-filled space around the brain), blood mixes with cerebrospinal fluid and irritates the meninges. The neck muscles tighten in reflex to protect the inflamed covering.
- Vision changes—blurry vision, double vision, or loss of part of the visual field: Blood can press on the optic nerve (the cable that carries pictures from the eye to the brain) or damage the occipital cortex (the back of the brain where vision is processed). Pressure inside the skull can also squeeze the nerve, cutting the signal.

## Focused exam

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- One-sided weakness or numbness: The blood pooling inside the skull is pressing on or damaging the motor cortex (the brain's movement-control strip) or the internal capsule (the bundle of wiring that carries movement commands from brain to body). The side of weakness points to which side of the brain is bleeding — right-brain bleeds weaken the left body, left-brain bleeds weaken the right body.
- Sudden, severe headache — worst headache of life — that peaks in seconds: This thunderclap headache means blood just burst into the skull. Subarachnoid bleeding (hemorrhage) (bleeding into the space around the brain) is the classic cause. The blood irritates pain sensors on the meninges (the brain's protective wrapping), triggering instant, extreme pain.

- Eyes that do not move together or pupils of different sizes (anisocoria): The blood or the swelling brain is squeezing cranial nerve III (the oculomotor nerve, which controls pupil size and eye position). A blown pupil — one pupil much larger than the other — signals uncal herniation: the brain is being pushed down through the skull opening and crushing the brainstem. This is a surgical emergency.
- Confusion, drowsiness, or decreasing level of consciousness: As blood fills the skull, it steals space from the brain and raises intracranial pressure. High pressure squeezes the reticular activating system (the brainstem's wakefulness network) and the cerebral cortex (the thinking layer). The person becomes confused, then sleepy, then unresponsive as the brain runs out of room.
- Stiff neck (nuchal rigidity) or pain when the chin is bent to the chest: This sign is classic for subarachnoid bleeding (hemorrhage). Blood in the subarachnoid space irritates the meninges and triggers muscle spasm in the neck. The irritation is chemical — blood does not belong in cerebrospinal fluid, and the meninges react by tightening up.
- Sudden seizure — new-onset jerking or stiffening: Blood touching the cortex (the brain's outer layer) irritates neurons and makes them fire chaotically, like a short circuit. Seizures raise intracranial pressure even higher and can worsen bleeding. They are more common with lobar (surface) hemorrhages.

## Diagnostics · every test answers a question

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- Non-contrast head CT → Is there blood inside the skull, where is it, and how much?
- Complete blood count (CBC) → Does the person have enough red cells to carry oxygen, and are platelets high enough to stop bleeding?
- Prothrombin time (PT/INR) and activated partial thromboplastin time (aPTT) → Is the blood clotting normally, or is anticoagulation making the bleeding worse?
- Basic metabolic panel (BMP) with creatinine → Are the kidneys working well enough to handle contrast dye if CT angiography is needed?
- CT angiography (CTA) of the head → Is there an aneurysm (bulging artery), arteriovenous malformation (AVM), or other vascular source that caused the bleed?
- Lumbar puncture (LP) with opening pressure and cerebrospinal fluid (CSF) analysis → If CT is normal but thunderclap headache suggests subarachnoid bleeding (hemorrhage), is there blood or breakdown products in the spinal fluid?
- Repeat non-contrast head CT at 6-24 hours → Is the bleeding (hemorrhage) expanding, stable, or shrinking?

## Treatment logic

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- Reverse anticoagulation immediately: vitamin K 10 mg IV + prothrombin complex concentrate (PCC) 25–50 units/kg, or fresh frozen plasma (FFP) if PCC unavailable, for warfarin; idarucizumab 5 g IV for dabigatran; andexanet alfa for apixaban/rivaroxaban → Restores clotting factors and stops the blood from staying too thin, which slows or halts bleeding (hemorrhage) expansion. PCC works faster than FFP (minutes vs. hours) and is the guideline first choice (AHA/ASA, 2022).
- Lower systolic blood pressure to 130–150 mmHg within 1 hour using IV nicardipine (5–15 mg/hr) or labetalol (10–20 mg IV bolus, then infusion) → Reduces the force pushing more blood out of the ruptured vessel, limiting a pooled collection of blood (hematoma) growth. The INTERACT2 and ATACH-2 trials support this target for intracerebral bleeding (hemorrhage) (AHA/ASA).
- Neurosurgery consult within 1 hour: consider craniotomy for a pooled collection of blood (hematoma) evacuation (> 30 mL lobar, cerebellar > 3 cm with brainstem compression), external ventricular drain (EVD) for hydrocephalus, aneurysm clipping or coiling, or AVM resection → Relieves mass effect (the pressure from the blood clot squashing brain tissue), lowers intracranial pressure, secures the bleeding source to prevent re-rupture, and restores cerebrospinal fluid flow if blocked.
- Manage intracranial pressure: elevate head of bed 30°, keep head midline, osmotic therapy with mannitol 0.25–1 g/kg IV or 3% hypertonic saline 150–250 mL bolus if signs of herniation, avoid low oxygen (hypoxia) (keep  $\text{SpO}_2 \geq 94\%$ ) and hypercarbia (keep  $\text{CO}_2$  35–40 mmHg) → Pulls fluid out of swollen brain tissue (osmotic agents), improves venous drainage (positioning), and keeps brain oxygen supply steady. This lowers intracranial pressure and preserves cerebral blood flow (perfusion) pressure ( $\text{CPP} = \text{MAP} - \text{ICP}$ , goal  $\geq 60$  mmHg).
- Preventive (Prophylactic) antiseizure medication — levetiracetam 500–1000 mg IV twice daily — for lobar bleeding (hemorrhage), cortical involvement, or early seizure → Prevents seizures that would spike intracranial pressure, worsen brain injury, and complicate monitoring. Routine prevention (prophylaxis) is controversial; AHA/ASA recommends it only in high-risk cases, not for all hemorrhages.
- Transfuse packed red blood cells if hemoglobin < 7 g/dL, platelets if count < 50,000/ $\mu\text{L}$  and active bleeding or planned surgery → Restores oxygen-carrying capacity so neurons stay alive, and provides enough platelets to form clots and stop oozing. Threshold of Hb < 7 is standard in neuro-critical care (Neurocritical Care Society).
- Serial neurologic exams every 1–2 hours and repeat imaging at 6–24 hours or sooner if exam worsens → Catches a pooled collection of blood (hematoma) expansion, hydrocephalus, or herniation early so intervention can happen before irreversible brainstem damage. About 30% of hemorrhages grow in the first 24 hours.

## Medications

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### **Prothrombin Complex Concentrate (PCC) or Fresh Frozen Plasma (FFP) · Reversal agent for warfarin (a blood thinner)**

Warfarin blocks the liver from making clotting proteins. When a bleed starts in the brain, you need those clotting proteins right away to stop the bleeding. PCC or FFP gives you instant clotting factors—the proteins your blood needs to form a strong clot. Think of it like adding cement mix back into wet concrete so it can harden and seal a crack.

1. Warfarin stops the liver from recycling vitamin K, which the liver needs to build clotting proteins II, VII, IX, and X.
2. Without these proteins, your blood cannot clot well, so a bleed keeps growing.
3. PCC is a concentrated dose of these exact clotting factors; FFP is donated plasma that contains them.
4. When you give PCC or FFP through an IV, the clotting factors flood the bloodstream immediately.
5. They travel to the bleeding vessel in the brain and plug the leak, stopping the bleeding (hemorrhage) from getting worse.

#### **SAFETY WATCH**

Risk of clots forming elsewhere (heart attack, stroke in a different artery, deep vein clot in the leg) if you give too much · Allergic reaction or fluid overload, especially with FFP · Must monitor clotting labs (INR) to confirm reversal worked

### **Idarucizumab (Praxbind) · Reversal agent for dabigatran (a direct oral anticoagulant, or DOAC)**

Dabigatran blocks thrombin, the enzyme that turns fibrinogen into fibrin strands to form a clot. Idarucizumab is a special antibody that grabs onto dabigatran molecules like a magnet and locks them up, pulling them out of circulation. Once dabigatran is trapped, thrombin is free again and clotting can restart.

1. Dabigatran sits on thrombin and blocks it from making fibrin, so blood cannot clot well.
2. Idarucizumab is injected into a vein.
3. It binds to dabigatran with very high affinity—much stronger than dabigatran's grip on thrombin.
4. The dabigatran-idarucizumab complex is inactive and gets cleared by the kidneys.
5. Thrombin is now unblocked and can help stop the brain bleed.

**SAFETY WATCH**

Patient may become pro-thrombotic (at higher risk for clots) once dabigatran is reversed, so restart anticoagulation carefully after the bleed is controlled · Rare allergic reactions · Does not require dose adjustment for kidney or liver function

**Andexanet Alfa (Andexxa) · Reversal agent for factor Xa inhibitors (rivaroxaban, apixaban, edoxaban)**

Factor Xa inhibitors block factor Xa, an enzyme in the clotting cascade that makes prothrombin turn into thrombin. Andexanet alfa is a decoy—it looks just like factor Xa, so the drug sticks to andexanet instead of the real factor Xa. Once the inhibitor is soaked up by the decoy, your own factor Xa is free to work and clotting restarts.

1. Rivaroxaban, apixaban, or edoxaban latches onto factor Xa and blocks it.
2. Andexanet alfa is given by IV; it is a modified, inactive version of factor Xa.
3. The anticoagulant drug binds to andexanet with high affinity and lets go of the real factor Xa.
4. Real factor Xa is now free to convert prothrombin to thrombin, restarting the clotting cascade.
5. The andexanet-drug complex is cleared from the blood.

**SAFETY WATCH**

High risk of thrombotic events (stroke, heart attack, deep vein clot) after reversal—reported in trials · Expensive and not universally available · Infusion-related reactions · Restart anticoagulation cautiously once bleeding is controlled

**Vitamin K (phytonadione) · Vitamin supplement and reversal adjunct for warfarin**

Warfarin blocks the liver from recycling vitamin K, which it needs to make clotting factors. Giving vitamin K by IV or mouth restocks the liver's supply so it can start making clotting factors again. Think of it like refilling the ink cartridge in a printer—the liver can now print clotting proteins. Vitamin K takes hours to work, so it is given alongside PCC or FFP for immediate reversal.

1. Warfarin prevents the enzyme that recycles vitamin K, so the liver runs out of the active form.
2. Without active vitamin K, the liver cannot finish building factors II, VII, IX, and X.
3. You give vitamin K by slow IV push or by mouth.
4. The liver uses the new vitamin K to restart production of clotting factors.

5. After 6–24 hours, clotting factor levels rise and stay up, giving sustained reversal.

**SAFETY WATCH**

Anaphylaxis with rapid IV push; give slowly over at least 30 minutes · Oral vitamin K is safer but takes longer to work · Can make warfarin very hard to restart if given in high doses

**Platelet Transfusion · Blood product replacement**

Antiplatelet drugs like aspirin and clopidogrel block platelets from clumping together to form the first plug at a bleeding site. Transfusing fresh platelets gives the patient a new supply of working platelets that are not blocked by the drug. These fresh platelets can stick to the torn vessel wall and help stop the bleed.

1. Aspirin permanently blocks an enzyme (COX-1) in platelets, and clopidogrel blocks a receptor (P2Y<sub>12</sub>) that platelets need to activate.
2. The patient's own platelets cannot clump, so bleeding continues.
3. Donor platelets are transfused by IV.
4. These new platelets are fully functional and can bind to the damaged vessel wall.
5. They release signals that recruit more platelets and start forming a stable plug to seal the leak.

**SAFETY WATCH**

Transfusion reactions (fever, allergic reaction, rarely lung injury called TRALI) · Risk of infection from donor blood · Volume overload in patients with heart failure · Platelet refractoriness (patient makes antibodies against donor platelets) with repeated transfusions

**Nicardipine or Labetalol (IV) · Antihypertensive (nicardipine is a calcium channel blocker; labetalol is a combined alpha- and beta-blocker)**

After a brain bleed, very high blood pressure can make the bleeding worse by pushing more blood out of the torn vessel. Nicardipine relaxes the smooth muscle in artery walls by blocking calcium channels, widening the arteries and lowering pressure. Labetalol blocks adrenaline receptors in the heart and blood vessels, slowing the heart and relaxing vessels. Both drugs bring blood pressure down in a controlled, smooth way.

1. High blood pressure increases the force pushing blood through the torn vessel, expanding the a pooled collection of blood (hematoma).
2. Nicardipine blocks L-type calcium channels in vascular smooth muscle; without calcium influx, the muscle cannot contract, so arteries relax and widen.

3. Labetalol blocks beta-1 receptors in the heart (slowing heart rate and reducing force of contraction) and alpha-1 receptors in vessels (causing dilation).
4. Arterial pressure drops in a controlled, titratable way (you can adjust the IV drip rate minute by minute).
5. Lower pressure reduces stress on the bleeding site, helping the clot to seal without rebleeding.

**SAFETY WATCH**

Monitor blood pressure continuously; goal is typically systolic 140–160 mm Hg in intracerebral bleeding (hemorrhage) (per AHA/ASA guidelines) to balance bleeding risk and brain blood flow (perfusion) · Nicardipine: reflex a fast heart rate (tachycardia) (heart speeds up), flushing · Labetalol: bronchospasm in asthma patients, a slow heart rate (bradycardia), avoid in severe heart failure or heart block · Avoid abrupt drops in pressure—can cause starved blood flow (ischemia) (not enough blood flow) to the brain

**Mannitol or Hypertonic Saline (3% or 23.4% NaCl) · Osmotic diuretic (mannitol) and hyperosmolar agent (hypertonic saline)**

A large bleed in the brain takes up space and squeezes the brain tissue, raising pressure inside the skull (intracranial pressure, or ICP). Mannitol and hypertonic saline are very concentrated solutions that pull water out of swollen brain tissue by osmosis—water moves from the tissue into the bloodstream because the blood becomes saltier. This shrinks the swelling and lowers the pressure, protecting the brain from further damage.

1. The bleeding (hemorrhage) and surrounding swelling (edema) (swelling) increase the volume inside the fixed skull, raising intracranial pressure.
2. High ICP can crush brain tissue, reduce blood flow, and cause herniation (brain shifts and squeezes through skull openings).
3. Mannitol or hypertonic saline is given by IV, raising the osmolality (saltiness) of the blood.
4. Water in the swollen brain tissue moves across the blood-brain barrier into the more concentrated blood by osmosis.
5. Brain tissue volume shrinks, ICP drops, and blood flow improves.

**SAFETY WATCH**

Mannitol: can cause rebound swelling if used repeatedly, acute kidney injury, electrolyte imbalances (low sodium, low potassium), volume depletion · Hypertonic saline: can cause dangerously high sodium (hypernatremia), volume overload, heart failure, central pontine myelinolysis if sodium rises too fast · Monitor serum sodium and osmolality closely · Use only with ICP monitoring and neurosurgical consultation

**Levetiracetam (Keppra) · Antiseizure medication (anticonvulsant)**

Blood and clot in the brain irritate the surrounding neurons, making them fire chaotically and trigger seizures. Levetiracetam binds to a protein called SV2A on the surface of nerve terminals, which helps control the release of neurotransmitters (the chemical messengers neurons use to talk). By stabilizing this release, levetiracetam calms overexcited neurons and prevents seizure activity.

1. The a pooled collection of blood (hematoma) and surrounding swelling (edema) create a focus of irritable, hyperexcitable neurons.
2. These neurons can start firing in synchronized bursts, causing a seizure.
3. Levetiracetam binds to the SV2A protein on synaptic vesicles.
4. This modulates the release of excitatory neurotransmitters like glutamate.
5. Neurons become less likely to fire all at once, reducing seizure risk.

**SAFETY WATCH**

Generally well tolerated; fewer drug interactions than older anticonvulsants · Can cause drowsiness, dizziness, irritability, or mood changes · Rare severe behavioral or psychiatric reactions · Dose must be reduced in kidney disease · Do not stop abruptly—taper to avoid withdrawal seizures

**Nimodipine · Calcium channel blocker (specific for cerebral vasospasm)**

After a subarachnoid bleeding (hemorrhage) (bleeding around the brain from a ruptured aneurysm), blood in the subarachnoid space irritates arteries and causes them to clamp down (vasospasm), starving brain tissue of oxygen. Nimodipine blocks L-type calcium channels in the smooth muscle of these arteries, preventing them from contracting. This keeps the vessels open and preserves blood flow, reducing the risk of stroke from vasospasm.

1. Blood breakdown products in the subarachnoid space release substances that cause arterial smooth muscle to contract.
2. Calcium flows into smooth muscle cells through L-type calcium channels, triggering contraction and vasospasm.
3. Nimodipine is given by mouth or through a feeding tube every 4 hours.
4. It blocks the calcium channels, preventing calcium influx.
5. Arterial smooth muscle stays relaxed, vessels remain open, and brain tissue gets adequate blood flow, lowering the risk of delayed cerebral starved blood flow (ischemia) (a stroke that happens days after the initial bleed).

**SAFETY WATCH**

Can lower blood pressure—monitor closely; some patients need IV fluids or pressors to keep pressure up · Headache, flushing, nausea · Must NOT be given IV—IV administration has caused severe, sometimes fatal low blood pressure (hypotension); oral or enteral route only · Continue for 21 days after subarachnoid bleeding (hemorrhage) per guidelines

## Red flags

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- Sudden, thunderclap headache—the worst headache of your life, peaking in seconds
- Fainting or collapsing, especially right after the headache starts
- Rapidly worsening confusion, sleepiness, or inability to wake someone up
- New weakness, numbness, or paralysis on one side of the face or body
- Seizure in someone with no prior seizure history

## Nursing priorities

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1. Check neurologic status every 15 minutes using the Glasgow Coma Scale (a 3-to-15 point scoring system measuring eye opening, verbal response, and motor response), and immediately alert the provider if the score drops by 2 or more points. — A falling Glasgow Coma Scale score means the brain is herniating (shifting downward because pressure is too high), compressing the brainstem (the part controlling breathing and heart rate), which can cause death within minutes if not treated with emergency surgery or a burr hole (a small skull opening to drain blood).
2. Keep the head of the bed elevated to 30 degrees and the patient's head in neutral midline position, not turned to either side. — Elevating the head helps venous blood (blood in veins) drain out of the skull by gravity, lowering intracranial pressure; twisting the neck kinks the jugular veins (the large neck veins draining the brain) and traps blood inside the skull, raising pressure.
3. Measure blood pressure every 5 to 15 minutes and titrate IV nicardipine or labetalol to keep systolic pressure between 130 and 150 mmHg, never lowering it faster than 20 mmHg per hour. — High blood pressure pushes more blood into the bleeding (hemorrhage), making it bigger, but dropping pressure too fast or too low cuts off blood flow (perfusion) (blood flow) to healthy brain tissue around the bleed, causing blood-starved (ischemic) stroke (tissue death from lack of oxygen) on top of the bleeding.

4. Reverse any anticoagulation immediately: give vitamin K 10 mg IV slow push plus prothrombin complex concentrate 25 to 50 units per kilogram IV for warfarin, idarucizumab 5 grams IV for dabigatran, or andexanet alfa per dosing protocol for apixaban or rivaroxaban. — Blood thinners prevent clotting, so the bleeding (hemorrhage) keeps expanding until you restore clotting factors (proteins that form clots); prothrombin complex concentrate works in 15 minutes versus 12 to 24 hours for vitamin K alone, stopping the bleed faster and saving brain tissue.
5. Keep the patient NPO (nothing by mouth) and place at aspiration precautions with suction at the bedside, turning the head to the side if vomiting occurs. — Intracranial bleeding (hemorrhage) often damages the brainstem or cortex areas controlling swallowing, causing trouble swallowing (dysphagia) (difficulty swallowing); vomiting is common from rising intracranial pressure, and aspiration (breathing in stomach contents) causes pneumonia and respiratory failure in patients who cannot protect their airway.
6. Monitor for seizure activity and keep lorazepam 2 to 4 mg IV at the bedside; pad the side rails and keep the bed in the lowest position. — Blood irritates the cerebral cortex (the brain's outer layer) and lowers the seizure threshold (how easily abnormal electrical activity starts); seizures raise intracranial pressure even higher, increase metabolic demand (the brain's need for oxygen), and can cause the patient to fall and bleed more if the head hits a hard surface.
7. Avoid any straining, coughing, or Valsalva maneuvers by giving stool softeners like docusate 100 mg twice daily and controlling pain and nausea with scheduled acetaminophen and ondansetron, not waiting for the patient to ask. — Straining, bearing down, coughing, and vomiting all raise intrathoracic pressure (pressure in the chest), which backs up into the venous system and spikes intracranial pressure by 30 to 40 mmHg in seconds, forcing more blood out of the ruptured vessel.
8. Prepare for emergency CT scan (computed tomography, a special X-ray showing brain slices) immediately, and have large-bore IV access (18-gauge or larger) in place for possible urgent surgery or transfer to the ICU. — CT imaging in the first hour confirms the location, size, and type of bleed, guiding whether the patient needs craniotomy (surgical skull opening to remove the clot), external ventricular drain (a tube placed in the brain to drain cerebrospinal fluid and lower pressure), or medical management; large IV access allows rapid infusion of blood products, reversal agents, and anesthesia drugs if the patient crashes.

## Sources

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- American Heart Association / American Stroke Association Guidelines for the Management of Spontaneous Intracerebral Hemorrhage (2022)

- American Heart Association / American Stroke Association Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage (2023)
- Neurocritical Care Society
- CDC Stroke Facts

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