

# Acute Intracranial Bleed

Acute intracranial bleed—carefully lower blood pressure to stop the clot from growing, reverse any blood thinners, watch every hour for signs the brain is shifting (herniation), and call neurosurgery early.

## THE ONE-LINE TRUTH

Blood leaks into the skull where it does not belong, raising pressure in a rigid box that cannot stretch to make room.

## The chain — how it unfolds

1. A blood vessel ruptures, leaking blood into the skull. — why: The upstream break is a ruptured artery or vein. In intracerebral bleeding (hemorrhage) (ICH), a small deep artery bursts, usually from chronic high blood pressure weakening the vessel wall. In subarachnoid bleeding (SAH), an aneurysm (balloon-like weak spot) on a large artery ruptures. In subdural a pooled collection of blood (hematoma) (SDH), bridging veins tear, often from trauma or brain atrophy. In epidural a pooled collection of blood (EDH), the middle meningeal artery is torn by a skull fracture. The blood leaks into the brain tissue, subarachnoid space, subdural space, or epidural space. Unlike a cut on your skin where blood clots and stops, bleeding inside the skull continues until pressure builds enough to compress the vessel or until the vessel is surgically sealed or the body's clotting system catches up (if not impaired by blood thinners).
2. Blood accumulates, forming a pooled collection of blood (hematoma) (clot) that takes up space in a rigid box that cannot expand. — why: The skull is a closed, fixed-volume container. The Monroe-Kellie doctrine says the total volume inside the skull is constant: brain tissue (about 80 percent), blood (about 10 percent), and cerebrospinal fluid (about 10 percent). When a pooled collection of blood (hematoma) forms, it adds volume. At first, the body compensates by pushing CSF out of the skull and compressing veins. But once those buffers are exhausted (usually after 50 to 100 mL of added volume), any further increase in volume directly raises intracranial pressure (ICP). Normal ICP is 5 to 15 mmHg. When ICP rises above 20 to 25 mmHg, the brain is in danger.

3. Rising intracranial pressure (ICP) reduces cerebral blood flow (perfusion) pressure (CPP).  
— why: Cerebral blood flow (perfusion) pressure (CPP) is the pressure pushing blood into the brain. CPP equals mean arterial pressure (MAP, the average blood pressure in your arteries) minus ICP. For example, if MAP is 90 mmHg and ICP is 10 mmHg, CPP is 80 mmHg (normal). If ICP rises to 40 mmHg and MAP stays 90, CPP drops to 50 mmHg (dangerously low). The brain needs CPP of at least 50 to 60 mmHg to get enough blood flow. Below that, brain cells start to starve (ischemia). Autoregulation (the brain's ability to widen or narrow vessels to keep blood flow steady) tries to compensate by dilating arteries, but this only works if CPP stays above about 50 mmHg and autoregulation is intact (it often fails after brain injury).
4. The brainstem detects falling blood flow (perfusion) pressure and activates the Cushing reflex: blood pressure rises, heart rate slows, and breathing becomes irregular. — why: When CPP drops dangerously low, the brainstem (specifically, the medulla) detects the threat to brain blood flow. It activates the sympathetic nervous system to raise systemic blood pressure—often to very high levels (systolic 180 to 220 mmHg or more)—to force blood through the compressed vessels. This is the body's last-ditch effort to save the brain. At the same time, the high blood pressure triggers baroreceptors (pressure sensors) in the carotid arteries and aorta, which activate the parasympathetic nervous system (vagus nerve) to slow the heart rate (bradycardia, often 40 to 60 beats per minute). Rising ICP also compresses the brainstem respiratory centers, causing irregular breathing (Cheyne-Stokes pattern: cycles of deep breaths followed by shallow breaths or pauses; or uncoordinated (ataxic) breathing: completely chaotic, irregular breaths). The combination of high blood pressure, slow heart rate, and irregular breathing is called the Cushing triad. It is a late, ominous sign that the brain is about to herniate. But here is the trap: high blood pressure from the Cushing reflex can worsen the bleed by raising pressure in the ruptured vessel, causing a pooled collection of blood (hematoma) expansion. This is why blood pressure control is critical but must be done carefully—too low and you drop CPP, too high and you expand the clot.

5. Mass effect (pressure from the a pooled collection of blood (hematoma) and surrounding swelling (edema)) shifts brain tissue toward areas of lower pressure, causing herniation. — why: When ICP exceeds the brain's compensatory capacity, the brain is squeezed and pushed toward the only exits: the natural openings in the skull and the membranes dividing brain compartments. Think of squeezing a water balloon—the water has to go somewhere. The brain herniates (shifts) through these openings, compressing and stretching vital structures. Subfalcine herniation: the cingulate gyrus (front part of the brain) is pushed under the falx cerebri (the membrane between the left and right sides), compressing the anterior cerebral artery (causing leg weakness on the opposite side). Uncal herniation: the uncus (the inner part of the temporal lobe) is pushed through the tentorial notch (the opening in the tentorium, the membrane separating the upper and lower brain), compressing the third cranial nerve and the brainstem. Central transtentorial herniation: both sides of the brain are pushed down through the tentorial notch, compressing the entire brainstem from above. Tonsillar herniation: the cerebellar tonsils (the lowest part of the cerebellum at the back of the brain) are pushed down through the foramen magnum (the opening at the base of the skull where the spinal cord exits), compressing the medulla (the lowest part of the brainstem). Each type of herniation has specific clinical signs because it compresses specific structures.
6. Herniation compresses cranial nerves and brainstem structures, producing focal signs: dilated pupil, abnormal posturing, and loss of consciousness. — why: Uncal herniation compresses the third cranial nerve (oculomotor nerve), which carries parasympathetic fibers that constrict the pupil. When these fibers are squeezed, the pupil dilates (gets big) and does not react to light. This happens on the same side as the a pooled collection of blood (hematoma) (ipsilateral dilated pupils (mydriasis)). The third nerve also controls most eye movements, so the eye may point down and out. Uncal herniation also compresses the brainstem motor tracts (corticospinal tracts), which carry movement signals from the brain to the body. Compression of the contralateral (opposite side) motor tract causes weakness or paralysis on the opposite side of the body (contralateral weakness on one side (hemiparesis)). Sometimes the brainstem is pushed against the opposite edge of the tentorium (a false localizing sign called Kernohan's notch), causing weakness on the same side as the a pooled collection of blood—this can confuse the clinical picture. As herniation worsens, the brainstem is compressed further, damaging the reticular activating system (the network that keeps you awake). This causes loss of consciousness, progressing from drowsiness to stupor (arousable only to painful stimuli) to coma (not arousable). Damage to motor pathways in the brainstem causes abnormal posturing: decerebrate posturing (arms and legs extended and stiff, indicating damage to the midbrain) or decorticate posturing (arms flexed and legs extended, indicating damage above the midbrain). Central transtentorial herniation compresses the upper brainstem, causing small, poorly reactive pupils (both sides), then loss of all brainstem reflexes (no pupil response, no corneal reflex, no gag reflex, no response to pain).

7. Tonsillar herniation compresses the medulla, causing sudden respiratory arrest and cardiovascular collapse. — why: The medulla (the lowest part of the brainstem) contains the respiratory center (which controls breathing) and cardiovascular centers (which control heart rate and blood vessel tone). When the cerebellar tonsils herniate down through the foramen magnum, they compress the medulla. The respiratory center fails, causing pauses in breathing (apnea) (no breathing). The cardiovascular centers fail, causing loss of sympathetic tone (blood pressure drops) and severe a slow heart rate (bradycardia) (heart rate slows or stops), leading to cardiac arrest. This is brain death if not immediately reversed. Tonsillar herniation is often the final event in progressive ICP elevation. It can also occur suddenly after lumbar puncture (spinal tap) in a patient with high ICP, because removing CSF from the spinal canal creates a pressure gradient that sucks the brain downward—this is why lumbar puncture is contraindicated (should not be done) if there is concern for elevated ICP or mass effect.
8. Without intervention, progressive brainstem compression leads to death. — why: Once herniation begins, the process is self-reinforcing. Brainstem compression impairs consciousness and breathing, causing low oxygen (hypoxia) (low oxygen) and hypercapnia (high carbon dioxide). Hypercapnia dilates cerebral vessels, increasing cerebral blood volume and raising ICP further. Low oxygen causes more swelling (edema). ICP rises higher, worsening herniation. The cycle accelerates until the medulla is crushed and the patient dies. This is why early recognition and aggressive treatment are critical. Reverse anticoagulation immediately (if the patient is on blood thinners). Control blood pressure to prevent a pooled collection of blood (hematoma) expansion (target systolic 140 mmHg for most ICH, higher for SAH). Lower ICP with osmotic therapy (mannitol or hypertonic saline), head elevation, sedation, and sometimes hyperventilation. Call neurosurgery early—large hematomas, cerebellar bleeds, and epidural hematomas often need surgical evacuation. External ventricular drain (EVD) placement can relieve hydrocephalus and monitor ICP. Decompressive craniectomy (removing part of the skull to give the brain room to swell) is a last-resort option for refractory (uncontrollable) ICP. The goal is to stop the bleed, lower ICP, maintain CPP above 60 mmHg, and prevent herniation.

## Spot it — what you see, and why

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- Sudden severe headache: Blood leaks out and irritates the meninges (the brain's tough protective wraps). The skull pressure shoots up and stretches pain nerves in the dura (the outermost layer) and blood vessel walls. When a weak bubble on a brain artery pops (an aneurysm rupture causing subarachnoid bleeding (hemorrhage)), people feel the worst headache of their life—like being hit by lightning. Large bleeds deep inside brain tissue also trigger sudden, fierce head pain because the expanding pool of blood squeezes surrounding tissue.

- Nausea and vomiting: Rising pressure inside the skull pushes down on the brainstem (the stalk that connects brain to spinal cord and runs breathing, heart rate, and vomiting reflexes). This squeezing activates the chemoreceptor trigger zone (a spot in the brainstem that senses toxins and signals the stomach to empty). When pressure climbs very high, vomiting shoots out with no warning—no belly ache or queasiness first—like a fountain.
- Altered mental status or loss of consciousness: The blob of blood and swelling squash the cerebral cortex (the brain's outer thinking layer) and the reticular activating system (a network in the brainstem that keeps you awake). When these parts are squeezed or torn, signals scramble. The person becomes confused, drowsy, hard to wake, or completely out. Bigger bleeds and higher skull pressure cause deeper coma faster.
- Weakness, numbness, vision loss, or trouble speaking: The clot of blood and swelling crush or tear specific brain zones. If the motor cortex (the strip that sends movement commands) is damaged, the opposite side of the body goes weak or paralyzed. If the sensory cortex is hit, that side feels numb or tingly. If the occipital lobe (the back of the brain that processes sight) is injured, you lose part or all of your vision. If Broca's area (front left brain, for forming words) or Wernicke's area (back left brain, for understanding language) is crushed, you can't speak clearly or understand speech.
- Seizure: Blood and breakdown products when red blood cells split open—iron and hemoglobin—irritate neurons (the brain's signal-sending cells) on the cortex surface. This lowers the electrical firing threshold, so neurons explode in wild, synchronized bursts that cause shaking, stiffening, or confusion. Seizures happen more often when the bleed sits on the outer brain or spills around the brain than when it is buried deep.
- Unequal pupils: When pressure builds, the inner edge of the temporal lobe (a hook-shaped part called the uncus) can be pushed down through a small gap in the skull's inner shelf. This squeezes the oculomotor nerve (cranial nerve III), which normally keeps the pupil small. When that nerve is crushed, the pupil on that side gets big and stops shrinking in bright light. This is an emergency sign that brain tissue is shifting and the brainstem is being compressed.

## Red flags — act now

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- Sudden thunderclap headache—worst headache of your life, peaks in seconds (classic for burst aneurysm and subarachnoid bleeding (hemorrhage))
- Rapidly declining level of consciousness—person goes from awake to drowsy to unresponsive in minutes to hours
- Unequal or non-reactive pupils—one pupil bigger than the other and doesn't shrink in bright light (sign that brain is shifting and squeezing cranial nerve III)
- Cushing triad: very high blood pressure, slow heart rate, and irregular breathing (brain is about to herniate)

- New seizure or continuous seizure activity that won't stop with first medicine

## Treat it — in this order

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1. Acute blood pressure lowering using IV nicardipine infusion (start 5 milligrams per hour, titrate by 2.5 milligrams per hour every 5 to 15 minutes, max 15 milligrams per hour) or clevidipine infusion (start 1 milligram per hour, double every 90 seconds up to 21 milligrams per hour), targeting systolic blood pressure less than 140 millimeters of mercury within 1 hour for spontaneous intracerebral bleeding (hemorrhage) without known secondary cause (per American Heart Association / American Stroke Association 2022 guidelines) → Blood pressure is the driving force pushing blood through the torn hole in the vessel wall. Think of it like water spraying from a burst pipe — the higher the pressure, the harder the water sprays out. Lowering blood pressure reduces hydrostatic pressure (the force of blood pushing outward against vessel walls), which slows active bleeding and decreases a pooled collection of blood (hematoma) expansion (the blood clot getting bigger). A pooled collection of blood expansion in the first 24 hours is the strongest predictor of neurologic deterioration and death. The landmark INTERACT-2 and ATACH-2 trials showed that rapidly lowering systolic BP to less than 140 millimeters of mercury is safe and reduces a pooled collection of blood growth, which saves lives and preserves brain function.

2. Immediate reversal of anticoagulation with agent-specific therapy: for warfarin give IV vitamin K 10 milligrams (takes 6 to 24 hours to work) PLUS 4-factor prothrombin complex concentrate (PCC) 25 to 50 units per kilogram IV bolus (works in minutes) to target INR less than 1.4; for dabigatran (Pradaxa) give idarucizumab (Praxbind) 5 grams IV as two 2.5-gram boluses; for factor Xa inhibitors (rivaroxaban, apixaban, edoxaban) give andexanet alfa (Andexxa) IV per weight-based and timing-based dosing protocol; for unfractionated heparin stop the infusion and consider protamine sulfate 1 milligram per 100 units of heparin given in the last 2 to 3 hours; platelet transfusion (1 apheresis unit or 6 units pooled platelets) is considered for platelet count less than 50,000 per microliter or documented recent antiplatelet drug use (aspirin, clopidogrel, ticagrelor) ONLY if there is active a pooled collection of blood (hematoma) expansion on repeat imaging or urgent surgery is planned, per 2016 Neurocritical Care Society guidelines (routine platelet transfusion for antiplatelet therapy does NOT improve outcomes and may worsen them) → Anticoagulants and antiplatelet drugs disable parts of the clotting cascade (the step-by-step chemical chain reaction that forms a blood clot). Reversal agents restore the blood's ability to clot by replacing missing clotting factors (PCC contains factors II, VII, IX, X — the proteins that trigger clot formation), directly binding and inactivating the anticoagulant drug molecule (idarucizumab wraps around dabigatran like a sponge soaking up a spill; andexanet alfa is a decoy molecule that binds factor Xa inhibitors), or neutralizing heparin by binding it (protamine, a positively charged protein that sticks to negatively charged heparin). Restoring normal coagulation allows the body's natural hemostasis (clot formation and vessel sealing) to plug the ruptured vessel and stop a pooled collection of blood (hematoma) expansion.
3. Hyperosmolar therapy with IV mannitol 20 percent solution (0.25 to 1 gram per kilogram, typical dose 50 to 100 grams for an adult, given as bolus over 15 to 20 minutes) or 3 percent hypertonic saline bolus (250 milliliters over 15 to 20 minutes, or 5 milliliters per kilogram up to 250 milliliters) for clinical signs of transtentorial herniation (blown pupil, decerebrate or decorticate posturing, Cushing reflex with high blood pressure and slow heart rate, rapidly declining Glasgow Coma Scale score) → The blood-brain barrier (the layer of tightly sealed cells lining brain capillaries) is intact in most of the brain even during acute bleeding (hemorrhage), except right at the injury site. Hyperosmolar agents (mannitol and hypertonic saline) are concentrated solutions that cannot cross this intact barrier. When you inject them into the bloodstream, they raise the osmolality (the concentration of dissolved particles) in the blood compared to brain tissue. This creates an osmotic gradient (a concentration difference) that pulls water out of brain tissue into the bloodstream, like a sponge drying out. This rapidly shrinks brain volume, decreases cerebral swelling (edema), and lowers intracranial pressure within 15 to 30 minutes. The drop in pressure temporarily reverses brainstem compression, buying critical time (hours) for definitive treatment like surgical evacuation or transferring the patient to a neurosurgical center.

4. Neurosurgical intervention tailored to bleed type and location: (1) Craniotomy with a pooled collection of blood (hematoma) evacuation for supratentorial intracerebral bleeding (hemorrhage) (bleeding above the tentorium, the brain's horizontal shelf) typically greater than 30 milliliters volume with mass effect causing neurologic decline, or any posterior fossa (cerebellum or brainstem area) bleeding greater than 3 centimeters diameter or with brainstem compression or hydrocephalus; (2) External ventricular drain (EVD) placement for acute hydrocephalus (cerebrospinal fluid trapped and backing up because blood blocks the drainage pathways, causing pressure to spike); (3) Endovascular coiling (threading a microcatheter through arteries to the aneurysm and packing it with platinum coils) or open surgical clipping (craniotomy to place a titanium clip across the aneurysm neck) for ruptured saccular aneurysm in subarachnoid bleeding, typically within 24 hours of presentation to prevent rebleeding; (4) Decompressive craniectomy (removing a section of skull bone to give the swelling brain room to expand outward instead of herniating downward) for malignant cerebral swelling (edema) not controlled by medical therapy, per American Heart Association / American Stroke Association and Neurocritical Care Society guidelines → Surgical a pooled collection of blood (hematoma) evacuation physically removes the blood clot, immediately eliminating the space-occupying mass causing intracranial pressure elevation and relieving compression of adjacent brain tissue and blood vessels. EVD directly drains trapped cerebrospinal fluid, lowering pressure when blood in the ventricles (the brain's fluid-filled chambers) or subarachnoid space blocks normal flow through the aqueduct of Sylvius or fourth ventricle outlets. Securing a ruptured aneurysm (coiling or clipping) mechanically seals the hole in the artery wall, preventing catastrophic rebleeding, which occurs in 4 to 14 percent in the first 24 hours and carries 70 to 80 percent mortality. Decompressive craniectomy gives the brain physical room to swell outward (like opening a pressure relief valve), preventing downward herniation. Surgery directly removes the cause of mass effect and prevents mechanical progression to brain death.

5. Antiseizure prevention (prophylaxis) with levetiracetam (Keppra) 500 to 1000 milligrams IV every 12 hours OR fosphenytoin 15 to 20 milligrams phenytoin equivalents per kilogram IV loading dose followed by 5 milligrams phenytoin equivalents per kilogram per day maintenance, typically for lobar intracerebral bleeding (hemorrhage) (bleeding in the cortex, the outer surface of the brain) or subarachnoid bleeding, guided by institution protocol and Neurocritical Care Society 2012 guidelines (universal prevention is NOT recommended for all hemorrhages, only high-risk subtypes; continuous EEG monitoring is preferred over blanket prevention when available) → Seizures dramatically increase the brain's metabolic demand (oxygen and glucose consumption can double or triple) at a time when the injured brain cannot meet that demand because blood supply is disrupted. Seizure activity also raises cerebral blood flow (through neurovascular coupling — active neurons signal blood vessels to dilate and deliver more blood), which increases intracranial pressure. Both visible convulsive seizures and nonconvulsive seizures (electrical seizures on EEG without visible shaking, occurring in 10 to 30 percent of patients with intracranial bleeding (hemorrhage)) worsen secondary brain injury through excitotoxicity (excess glutamate release damaging neurons), free radical generation (toxic oxygen molecules), and spreading depolarization (waves of electrical shutdown spreading across the cortex). Seizures also cause blood pressure spikes that can trigger a pooled collection of blood (hematoma) expansion. Preventive (Prophylactic) antiseizure medication stabilizes neuronal membranes (levetiracetam binds synaptic vesicle protein SV2A and modulates neurotransmitter release; phenytoin blocks voltage-gated sodium channels, preventing repetitive firing), reducing seizure risk and preventing this cascade of injury.

## Your top priorities at the bedside

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1. Elevate head of bed to 30 degrees and keep head in midline neutral position without neck flexion or rotation. — why: Elevating the head uses gravity to help venous blood (blood in veins) drain from the brain, lowering intracranial pressure (ICP, the pressure inside the skull), while neck twisting can kink jugular veins and trap blood in the head.
2. Monitor neurologic status every 15 minutes using Glasgow Coma Scale (GCS, a scoring system for alertness and responses) and check pupil size, symmetry, and reaction to light. — why: Rapid decline in GCS or new unequal pupils (anisocoria) (unequal pupils) means the bleeding is expanding or causing herniation (brain tissue shifting and squeezing through openings in the skull), which needs immediate surgical intervention.
3. Maintain strict blood pressure control with continuous IV nicardipine or clevidipine infusion to keep systolic BP below 140 mmHg, checking BP every 5 minutes during titration. — why: High blood pressure forces more bleeding from ruptured vessels through a process called a pooled collection of blood (hematoma) expansion (the blood clot getting bigger), and AHA guidelines show keeping systolic under 140 within the first hour improves outcomes.