

Intracerebral Hemorrhage

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THE STORY IN ONE SENTENCE

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“Intracerebral bleeding (hemorrhage) means a burst blood vessel dumps blood into the brain tissue. Lower blood pressure fast. Stop blood thinners. Watch pressure inside the skull. Surgery if the clot is big and the brain is shifting.”

Normal anatomy — the body's job

The brain sits inside a hard skull and is fed by a network of arteries that branch smaller and smaller, bringing oxygen and sugar to every brain cell. These vessels have strong, elastic walls that handle the pulse of blood pressure without leaking.

BODY'S JOB

Brain arteries deliver oxygen-rich blood under steady pressure. The vessel walls flex with each heartbeat, and the blood stays inside, never touching brain tissue. Deep small arteries (called penetrating arterioles) dive into the white matter and basal ganglia to feed the brain's core. Larger arteries near the surface feed the cortex (the brain's outer layer). All of this happens in a closed space—the skull—so total volume (brain + blood + fluid) stays constant.

1. Heart pumps blood up through the carotid and vertebral arteries into the brain.
2. Large arteries at the brain's base (the Circle of Willis) split into smaller branches that spread across the surface and dive deep inside.
3. Tiny arterioles deliver oxygen and glucose to neurons and then drain into veins.
4. The skull is a rigid box, so brain volume, blood volume, and cerebrospinal fluid volume must stay balanced—any extra pushes pressure up.

What breaks

A small artery deep in the brain or near the surface bursts, and blood leaks out into the brain tissue. Instead of feeding cells, the blood becomes a growing clot that squashes and kills the brain around it.

CLINICAL TERM

Intracerebral bleeding (hemorrhage) (ICH). A ruptured cerebral artery causes bleeding directly into the brain parenchyma (the working brain tissue). The a pooled collection of blood (hematoma) (blood clot) expands, destroys neurons, and raises intracranial pressure (ICP).

How the body compensates

The brain cannot escape the skull, so when a blood clot grows inside, the only options are to squeeze out cerebrospinal fluid, compress veins, and then push the brain itself. Early on, vital signs may look normal because the body is buying time. Once that buffer is used up, pressure spikes and the brain herniates (shifts and squeezes through openings in the skull), cutting off blood flow and causing death.

1. {'what': "Cerebrospinal fluid (CSF) is pushed out of the brain's fluid spaces (ventricles and subarachnoid space) into the spinal sac to make room for the growing clot.", 'limit': 'Only about 100–150 mL of CSF can be displaced before this system is exhausted.'}
2. {'what': 'Veins in the brain are squeezed shut, reducing blood volume inside the skull.', 'limit': 'Once veins collapse, there is no more give—intracranial pressure (ICP) starts to climb.'}
3. {'what': 'The brain tissue itself is compressed. If the clot is in one hemisphere, the opposite side and the brainstem start to shift toward openings in the skull (herniation).', 'limit': 'Herniation crushes the brainstem (which controls breathing, heart rate, and consciousness), causing coma and death within minutes to hours.'}
4. {'what': 'Blood pressure rises (Cushing reflex) to try to push blood through the squashed brain.', 'limit': 'This is a late, ominous sign (high BP, slow heart rate, irregular breathing) that means herniation is starting.'}
5. {'what': "The patient becomes less awake as the reticular activating system (the brainstem's awake-switch) is squeezed.", 'limit': 'Drowsiness → stupor → coma is the progression; once coma sets in, the window to reverse damage is nearly closed.'}

Domino effect

1. A small artery deep in the brain or on the surface ruptures. — Years of high blood pressure, amyloid buildup, an AVM, a tumor, or blood thinners weaken the vessel wall until it tears, and blood pours out under arterial pressure.
2. Blood forms a pooled collection of blood (hematoma) (clot) that grows and destroys brain tissue. — The leaked blood does not stay liquid—it clots and expands like a bruise, crushing and killing neurons directly. The clot can grow for hours, especially if blood pressure stays high or clotting is impaired.
3. Sudden, severe headache (often called 'the worst headache of my life'). — Blood irritates the meninges (the brain's protective layers) and stretches pain-sensitive structures, triggering intense head pain.
4. Nausea and vomiting. — Rising pressure inside the skull pushes on the area postrema and vomiting center in the medulla (lower brainstem), triggering the throw-up reflex.
5. Weakness, numbness, or paralysis on one side of the body. — If the bleed hits the internal capsule, basal ganglia, or motor cortex (areas that control movement and sensation), signals to the opposite side of the body are cut off, causing weakness on one side (hemiparesis) (one-sided weakness) or paralysis on one side (hemiplegia) (one-sided paralysis).
6. Trouble speaking or understanding (aphasia). — If the bleed is in the left hemisphere language areas (Broca's or Wernicke's area), the patient cannot produce or understand words.
7. Worsening drowsiness, confusion, or loss of consciousness. — The growing clot raises intracranial pressure (ICP), squeezing the reticular activating system in the brainstem (the brain's on-switch), and reducing blood flow to the cortex, so the patient gets sleepier and less responsive.
8. Brain herniation: the brain shifts and squeezes through openings in the skull. — When ICP is too high, the brain is pushed downward or sideways. The uncus (part of the temporal lobe) pushes through the tentorium and compresses the third cranial nerve (causing a blown pupil—one pupil huge and unresponsive to light) and the brainstem, or the cerebellar tonsils push down through the foramen magnum and crush the medulla, stopping breathing and heart function. Herniation is the final, irreversible step that causes death.

Symptoms · what the patient feels, and why

- Sudden weakness or numbness on one side of the body: Blood leaking into the brain tissue crushes and injures the nerve pathways that control movement and feeling. When the motor strip (the brain's movement command center) or its wiring is damaged, signals to muscles and skin on the opposite side of the body stop getting through.

- Severe headache that comes on suddenly: Blood pooling inside the skull stretches pain-sensitive membranes (the meninges that wrap the brain) and pushes on brain tissue. Blood itself is irritating to these tissues, triggering sharp, intense pain — like inflating a balloon inside a sealed box.
- Trouble speaking or understanding words (aphasia): Bleeding in the left side of the brain (in most people) damages language centers such as Broca's area (which helps form speech) or Wernicke's area (which helps understand words). Without these working, the brain cannot put thoughts into words or decode what others say.
- Sudden vision problems (blurred vision, double vision, or loss of part of the visual field): Blood pressing on the visual pathways (the optic tracts or occipital cortex, the brain's seeing center at the back) blocks the signals from the eyes. The brain cannot build a clear picture, so vision blurs, splits, or goes dark in patches.
- Loss of balance or trouble walking: Bleeding in the cerebellum (the brain's balance control center at the back-bottom) or in the pathways that coordinate movement disrupts the signals that keep you steady. The brain loses its 'gyroscope', making every step wobbly or impossible.
- Nausea and vomiting: Rising pressure inside the skull (from pooling blood and swelling) squeezes the brainstem, which houses the vomiting center. The brain interprets this pressure as a poisoning signal and triggers nausea and vomiting to try to protect itself.
- Confusion or difficulty staying awake (altered level of consciousness): Blood and swelling press on the brainstem's reticular activating system (the brain's wakefulness switch) and disrupt the networks that keep you alert and thinking clearly. Without these firing normally, consciousness dims like a phone battery draining fast.
- Seizures: Blood touching the surface of the brain (the cortex) is like throwing sand into electrical circuits. It irritates neurons and causes them to fire wildly all at once, producing a seizure — uncontrolled shaking or stiffening, sometimes with loss of awareness.
- High blood pressure (hypertension) at the time of the bleed: Chronic high blood pressure weakens the walls of tiny arteries deep in the brain over years (like a garden hose cracking under constant high pressure). When the wall finally ruptures, blood bursts into brain tissue. High BP at the moment of the bleed can also make the bleeding worse.

Focused exam

- Blood pressure over 160/100 mmHg: High blood pressure is the most common reason blood vessels inside the brain burst. When pressure is too high for too long, the walls of small, deep brain vessels crack and bleed.
- Sudden weakness on one side of the body (arm, leg, or face): Blood spilling into the brain is crushing the motor strip (the part of the brain that controls movement). The side that is weak tells you which side of the brain is bleeding.

- Severe headache that started suddenly: Blood leaking into brain tissue stretches the skull and irritates pain sensors. A sudden, worst-ever headache means the bleed happened minutes or hours ago, not days.
- Eyes looking toward the side of the bleed: The frontal eye field (a spot on the brain's surface that controls where your eyes point) is damaged or pushed by swelling. The eyes drift toward the injured side, like they are staring at the problem.
- Getting drowsy, confused, or hard to wake up: Blood and swelling are taking up space inside the skull and squeezing the reticular activating system (the brain's wake-up center in the brainstem). When pressure climbs, the brain shuts down to save energy.
- Vomiting without feeling sick to the stomach first: Rising pressure inside the skull pushes on the area postrema (the brain's vomit trigger zone at the base of the skull). This kind of vomiting is a red flag that pressure is climbing fast.

Diagnostics · every test answers a question

- Non-contrast head CT → Is there blood inside the brain tissue, and if so, where and how big is the pool?
- Complete blood count (CBC) with platelet count → Does the patient have enough platelets to form clots and stop bleeding?
- PT/INR and aPTT → Is the patient on a blood thinner that is blocking clotting, and how strong is the effect?
- Basic metabolic panel (BMP) → Are the kidneys working well enough to clear medicines and handle fluids?
- CT angiography (CTA) of the head → Is there a vascular abnormality like an aneurysm or AVM causing the bleed, especially in younger patients without high blood pressure (hypertension)?
- Electrocardiogram (ECG) → Is the heart rhythm stable, and is there evidence of strain from high catecholamine surge?

Treatment logic

- Lower systolic blood pressure to under 140 mmHg using IV nicardipine or clevidipine → High pressure keeps forcing blood out through the crack in the vessel wall. Lowering the pressure slows bleeding and stops the pool of blood from growing bigger.
- Reverse blood thinners: give vitamin K and prothrombin complex concentrate (PCC) for warfarin, idarucizumab for dabigatran, or andexanet alfa for rivaroxaban or apixaban → Blood thinners block clotting proteins or platelets. Reversal agents turn clotting back on so the body can plug the hole in the vessel and stop active bleeding.

- Manage intracranial pressure: elevate head of bed to 30 degrees, give mannitol (0.25–1 g/kg IV) or 3% hypertonic saline, sedate if agitated, and consider an ICP monitor if GCS ≤ 8 → Blood takes up space in the skull and squeezes healthy brain tissue, cutting off its oxygen. Osmotic agents pull water out of swollen brain cells, lowering pressure and protecting tissue around the bleed.
- Surgical evacuation if cerebellar bleed is larger than 3 cm, or lobar bleed larger than 30 mL that is getting worse; insert external ventricular drain (EVD) if hydrocephalus develops → Surgery physically removes the blood clot, relieving skull pressure instantly and restoring blood flow to squashed brain tissue. For cerebellar bleeds, it prevents the brainstem from being crushed, which would stop breathing.
- Give levetiracetam 500–1000 mg IV twice daily for lobar hemorrhages to prevent seizures → Injured brain tissue near the surface fires wild, uncontrolled electrical bursts. Levetiracetam (a seizure medicine) calms these signals, preventing a seizure that would burn oxygen and spike intracranial pressure.
- Avoid platelet transfusion unless platelet count is below 50,000 or urgent surgery is planned; avoid preventive (prophylactic) antiepileptics if no seizure occurred → Giving platelets when they are not critically low can worsen outcomes by promoting clot expansion. Antiepileptics without a seizure do not help and add side effects.

Medications

Nicardipine · Calcium channel blocker (intravenous, fast-acting)

Nicardipine relaxes the smooth muscle cells wrapped around blood vessels so the vessels widen and pressure drops quickly. After a brain bleed, high blood pressure pushes harder on the torn vessel and can make the bleeding worse. Nicardipine is given through an IV and works fast enough to bring systolic pressure down to less than 140 within minutes, which helps stop the bleed from getting bigger.

1. Blocks calcium channels on the surface of smooth muscle cells in artery walls.
2. Without calcium flowing in, the muscle cells cannot squeeze as hard.
3. The arteries relax and widen (dilate).
4. Blood pressure falls because there is less resistance pushing back against the heart's pumping.
5. Lower pressure means less force driving more blood out of the broken vessel in the brain.

SAFETY WATCH

Can drop blood pressure too low if the dose is not watched closely, which can reduce blood flow to the brain and other organs. · May cause a faster heart rate (reflex a fast heart rate (tachycardia)) as the body tries to make up for lower pressure. · Requires continuous IV infusion and close monitoring in an intensive care unit. · Dose must be adjusted every few minutes based on blood pressure readings.

Clevidipine · Calcium channel blocker (intravenous, ultra-short-acting)

Clevidipine works just like nicardipine—it blocks calcium from entering the smooth muscle cells in artery walls, so the arteries relax and blood pressure drops. The difference is that clevidipine breaks down in the blood within minutes, which means you can dial the dose up or down and see the change almost instantly. This ultra-short action makes it easier to hit the exact target pressure (systolic less than 140) without overshooting.

1. Blocks calcium channels on arterial smooth muscle cells.
2. Smooth muscle relaxes because it cannot squeeze without calcium.
3. Arteries widen and resistance drops.
4. Blood pressure falls quickly.
5. The drug is broken down by enzymes in the blood and tissues within 2–4 minutes, so its effect disappears fast when you stop or lower the infusion.
6. Lower pressure reduces the force pushing more blood out of the broken vessel.

SAFETY WATCH

Can cause blood pressure to drop too low if the infusion rate is too high. · May trigger a reflex increase in heart rate. · Comes dissolved in a lipid (fat) emulsion, so prolonged high-dose infusion can add extra triglycerides (fat in the blood)—watch triglyceride levels if used for many hours. · Requires continuous IV infusion and close monitoring in an ICU.

Labetalol · Combined alpha and beta blocker (intravenous)

Labetalol blocks two kinds of receptors—beta receptors (which normally speed up the heart and make it pump harder) and alpha receptors (which normally tighten blood vessels). By blocking both, labetalol slows the heart a bit and relaxes arteries, so blood pressure drops. It is given by IV push or infusion and brings pressure down within minutes, helping keep systolic pressure below 140 to reduce the risk of re-bleeding.

1. Blocks beta-1 receptors in the heart, so the heart beats a little slower and with less force.
2. Blocks alpha-1 receptors in artery walls, so vessels relax and widen.

3. Together, these effects lower the amount of blood pumped per minute and reduce resistance in the arteries.
4. Blood pressure falls.
5. Lower pressure means less stress on the torn vessel and less chance of pushing more blood out.

SAFETY WATCH

Can cause the heart to beat too slowly (bradycardia) or drop blood pressure too low. · Should NOT be used in people with asthma or severe chronic obstructive pulmonary disease (COPD), because beta blockers can tighten airways. · Avoid in people with certain heart rhythm problems (second- or third-degree heart block) or severe heart failure. · Requires close monitoring of heart rate and blood pressure.

Vitamin K (phytonadione) · Vitamin, clotting-factor cofactor

Vitamin K is a helper molecule the liver needs to finish building four clotting proteins (factors II, VII, IX, and X). Warfarin works by blocking vitamin K, so those clotting factors run low and blood becomes thin. Giving vitamin K by IV lets the liver start making fresh, working clotting factors again. It takes several hours to see the full effect because the liver has to build new proteins, but vitamin K reverses warfarin's blood-thinning over 12–24 hours.

1. Vitamin K enters liver cells.
2. An enzyme called gamma-glutamyl carboxylase uses vitamin K to add a chemical tag (carboxyl group) onto clotting factors II, VII, IX, and X.
3. Without that tag, the factors cannot grab calcium and work properly on blood vessel walls.
4. With vitamin K available again, the liver finishes the factors and releases them into the blood.
5. As new, working factors build up over hours, the blood's ability to clot returns to normal.

SAFETY WATCH

Rarely causes an allergic reaction (flushing, chest tightness, or anaphylaxis) if given too fast by IV—infuse slowly over 20–30 minutes. · High doses can make it hard to restart warfarin later if the patient needs long-term anticoagulation. · Does not help if the liver is too damaged to make new clotting factors.

Prothrombin complex concentrate (PCC, four-factor) · Clotting-factor replacement (pooled from donated plasma)

PCC is a concentrated mix of the four vitamin-K-dependent clotting factors (II, VII, IX, X) that warfarin depletes. When you give PCC by IV, it instantly replaces those missing factors so the

blood can clot normally again within minutes. This emergency reversal is critical after a brain bleed in someone on warfarin, because waiting hours for vitamin K to work means more bleeding and more brain damage.

1. PCC is infused into a vein and the clotting factors immediately enter the bloodstream.
2. Factors II, VII, IX, and X are now present at near-normal levels.
3. When a blood vessel is broken, these factors form a chain reaction (the coagulation cascade) that builds a mesh of fibrin threads to plug the hole.
4. Clotting returns to normal within 10–30 minutes.
5. The bleeding inside the brain can stop or slow, limiting further damage.

SAFETY WATCH

Carries a small risk of clotting complications (deep vein clot formation (thrombosis), pulmonary embolism (a clot lodging in a lung artery), heart attack, or stroke) because you are suddenly restoring full clotting ability. · Risk of allergic reaction or infection is very low (the product is screened and heat-treated), but not zero. · Expensive and must be stored frozen or refrigerated, so not always immediately available in small hospitals. · Must be given with vitamin K because PCC factors are used up within hours; vitamin K keeps new factors coming from the liver.

Idarucizumab · Monoclonal antibody fragment (specific reversal agent for dabigatran)

Idarucizumab is a tiny protein designed to grab onto dabigatran molecules in the blood and lock them up so they cannot block thrombin (the enzyme that turns fibrinogen into fibrin clots). Within minutes of giving idarucizumab by IV, dabigatran is pulled out of circulation and the blood can clot normally again. This instant reversal is life-saving for a brain bleed in someone taking dabigatran.

1. Idarucizumab is infused into a vein.
2. Each idarucizumab molecule finds and tightly binds a molecule of dabigatran.
3. The dabigatran is now locked up and cannot block thrombin anymore.
4. Free thrombin can work again, turning fibrinogen into fibrin to form clots.
5. Normal clotting is restored within minutes.

SAFETY WATCH

Generally very safe with few side effects. · After reversal, the patient has no anticoagulation, so there is a risk of clotting (stroke, a clot in a deep leg vein (deep vein thrombosis), pulmonary embolism (a clot lodging in a lung artery))—especially important because many of these patients were on dabigatran for atrial fibrillation (an irregular, quivering heartbeat) to prevent stroke in the first place. · Expensive and not stocked everywhere; call a stroke center or pharmacy early. · Rarely, hypersensitivity reactions (rash, itching) can occur.

Andexanet alfa · Recombinant modified factor Xa decoy (specific reversal agent for factor Xa inhibitors)

Andexanet alfa is a fake version of factor Xa (one of the clotting proteins). Drugs like rivaroxaban, apixaban, and edoxaban work by blocking real factor Xa. Andexanet acts as a decoy—it soaks up all those Xa-inhibitor molecules so they leave the real factor Xa alone. Once the inhibitors are mopped up, real factor Xa can work again and the blood clots normally within minutes. This reversal is critical for brain bleeds in patients on rivaroxaban or apixaban.

1. Andexanet alfa is given by IV bolus then continuous infusion.
2. Rivaroxaban, apixaban, or edoxaban molecules in the blood bind tightly to andexanet instead of real factor Xa.
3. Real factor Xa is now free to participate in the clotting cascade.
4. Factor Xa activates prothrombin to thrombin, and thrombin builds fibrin clots.
5. Normal clotting is restored within minutes and lasts for the duration of the infusion (about 2 hours).

SAFETY WATCH

Risk of serious clotting events (stroke, heart attack, a clot in a deep leg vein (deep vein thrombosis), pulmonary embolism (a clot lodging in a lung artery)) because coagulation is suddenly restored in someone who needed anticoagulation. · Infusion reactions (flushing, feeling hot, cough) can occur. · Very expensive and not available at all hospitals—early transfer to a comprehensive stroke center may be needed. · The antidote effect wears off over a few hours; monitor closely for re-bleeding.

Mannitol · Osmotic diuretic

Mannitol is a sugar molecule that stays in the bloodstream and pulls water out of brain tissue by osmosis (water flows toward higher concentration of particles). After a brain bleed, the tissue around the clot swells, and that swelling raises pressure inside the skull. Mannitol draws

the extra water into blood vessels, shrinking the swollen brain tissue and lowering intracranial pressure within 15–30 minutes. This buys time to get to surgery or for the swelling to settle.

1. Mannitol is given by rapid IV infusion.
2. It stays in the blood vessels and raises the osmolarity (particle concentration) of the blood.
3. Water in swollen brain cells flows across cell membranes into the blood vessels to balance the concentration (osmosis).
4. The brain tissue shrinks slightly, and pressure inside the skull drops.
5. Mannitol is filtered by the kidneys and pulled into the urine, taking the extra water with it, so you urinate more (diuresis).

SAFETY WATCH

Can cause dehydration and low blood pressure if too much fluid is pulled out. · May worsen kidney function, especially with repeated doses or in people who already have kidney disease. · Can cause electrolyte imbalances (low sodium, low potassium) that need monitoring. · Repeated use can lead to rebound increased intracranial pressure when the drug wears off. · Avoid if the patient has severe heart failure, because the sudden fluid shift can overload the heart.

Hypertonic saline (3% or 23.4% sodium chloride) · Osmotic agent (concentrated salt solution)

Hypertonic saline works just like mannitol—by raising the salt concentration in the blood, it pulls water out of swollen brain tissue through osmosis. The salty blood draws fluid from brain cells into blood vessels, shrinking the tissue and lowering pressure inside the skull. Hypertonic saline can be given as a continuous infusion (3%) or a fast bolus (23.4%) and works within minutes. It is often preferred over mannitol because it does not cause diuresis (extra urination) or dehydration as quickly.

1. Hypertonic saline is infused into a vein, raising blood sodium and osmolarity.
2. Water inside swollen brain cells flows across membranes into the bloodstream to balance the salt concentration.
3. Brain tissue shrinks and intracranial pressure drops.
4. The kidneys eventually clear some of the extra sodium and water, but the effect on brain swelling happens before significant diuresis.

SAFETY WATCH

Can cause blood sodium to rise too high (hypernatremia), which can lead to confusion, seizures, or dangerous heart rhythms—check sodium levels every few hours. · Rapid infusion of 23.4% saline through a small vein can cause vein irritation or damage—use a central line if possible. · Overuse can cause cells throughout the body to shrink and malfunction. · May worsen heart failure by increasing blood volume.

Levetiracetam · Antiepileptic drug (broad-spectrum)

Levetiracetam calms overexcited brain cells by binding to a protein called SV2A on the surface of neurons. SV2A helps control the release of neurotransmitters (chemical messengers between nerve cells). When levetiracetam binds SV2A, it slows down the burst of signals that can turn into a seizure. After a lobar (surface) brain bleed, blood and breakdown products irritate the cortex (the brain's outer thinking and movement layer), making seizures more likely. Levetiracetam helps prevent that runaway electrical activity.

1. Levetiracetam binds to SV2A, a protein on synaptic vesicles (tiny sacs that store neurotransmitters inside nerve endings).
2. This binding reduces the release of excitatory neurotransmitters like glutamate.
3. With less glutamate released, the next neuron is less likely to fire repeatedly.
4. Abnormal, synchronized firing (the hallmark of a seizure) is less likely to start or spread.
5. The overall electrical activity in the cortex stays more stable.

SAFETY WATCH

Can cause drowsiness, dizziness, or fatigue. · Some people experience mood changes, irritability, or even aggression. · Generally safe for the kidneys and liver, but dose must be lowered if kidney function is poor. · Lower risk of drug interactions compared to older antiepileptic drugs like phenytoin.

Phenytoin · Antiepileptic drug (sodium channel blocker)

Phenytoin stops seizures by blocking voltage-gated sodium channels on the surface of nerve cells. These channels open when a neuron fires, letting sodium rush in and triggering the next electrical signal. Phenytoin keeps the channels closed longer after each signal, so neurons cannot fire over and over in the rapid, uncontrolled way that causes a seizure. It is sometimes used to prevent seizures after a lobar brain bleed, though levetiracetam is now more common because it has fewer side effects.

1. Phenytoin binds to sodium channels that have just opened and closed.
2. It holds the channel in an inactive state for a longer time.

3. This means the neuron cannot fire again right away—there is a mandatory rest period.
4. Rapid, repetitive firing (the electrical pattern of a seizure) is blocked.
5. Normal, controlled signaling can still happen, but runaway excitation is prevented.

SAFETY WATCH

Can cause gum overgrowth (gingival hyperplasia), especially with long-term use. · May cause a serious skin rash (Stevens-Johnson syndrome) in rare cases—stop immediately if a rash appears. · Interacts with many other drugs because it affects liver enzymes. · Can lower blood counts (anemia, low white cells) with long-term use. · IV phenytoin must be given slowly (no faster than 50 mg/min) to avoid dangerous drops in blood pressure or heart rhythm problems; fosphenytoin is a safer IV alternative.

Red flags

- Sudden, severe headache described as 'the worst headache of my life'
- Rapid worsening of confusion, weakness, or loss of consciousness
- Seizure in someone with no prior seizure history
- Signs of brainstem injury: trouble breathing, very slow or irregular heartbeat, or pupils that do not respond to light
- Symptoms appearing suddenly in someone taking blood thinners (anticoagulants or antiplatelet drugs)

Nursing priorities

1. Check airway, breathing, and oxygen saturation every 15 minutes; keep oxygen saturation above 94% and position patient on their side if vomiting or unconscious. — Intracerebral bleeding (hemorrhage) can cause decreased level of consciousness and loss of protective airway reflexes (ability to cough and protect the airway), so aspiration (breathing in vomit or saliva) can cause pneumonia and low oxygen worsens brain swelling by causing vasodilation (widening of blood vessels that increases intracranial pressure).
2. Measure blood pressure every 15 minutes and notify the provider immediately if systolic pressure is above 140 mmHg; prepare IV nicardipine or clevidipine infusion as ordered. — High blood pressure makes the bleeding worse by forcing more blood out of the ruptured vessel, so acute blood pressure lowering to systolic under 140 mmHg within the first hour reduces a pooled collection of blood (hematoma) expansion (growth of the blood clot) and improves survival without increasing brain starved blood flow (ischemia) (lack of blood flow).

3. Perform full neurologic assessment using Glasgow Coma Scale (eye opening, verbal response, and motor response scores) and NIH Stroke Scale every hour and immediately report any worsening weakness, confusion, pupil changes, or decreased consciousness. — Rapid neurologic decline indicates a pooled collection of blood (hematoma) expansion or rising intracranial pressure from brain swelling, and early detection allows time for repeat CT scan, blood pressure adjustment, osmotic therapy (mannitol or hypertonic saline to pull fluid from brain tissue), or emergency neurosurgery before irreversible brain herniation occurs.
4. Elevate the head of the bed to 30 degrees with the neck in neutral midline position and avoid hip flexion above 90 degrees. — Head elevation promotes venous drainage (blood flow out of the brain through veins) which lowers intracranial pressure, while neck twisting or extreme hip flexion increases intra-abdominal pressure that backs up into the venous system and raises pressure inside the skull.
5. Hold all anticoagulants and antiplatelet drugs and prepare reversal agents: vitamin K 10 mg IV and four-factor prothrombin complex concentrate for warfarin patients, idarucizumab for dabigatran, or andexanet alfa for apixaban or rivaroxaban as ordered. — Impaired blood clotting (Coagulopathy) (abnormal blood clotting) from blood thinners allows ongoing bleeding and a pooled collection of blood (hematoma) expansion, so immediate reversal restores normal clotting factors (proteins that stop bleeding) and significantly reduces mortality, with reversal ideally completed within 4 hours of symptom onset.
6. Keep the patient calm and quiet; avoid painful procedures without sedation; give sedation as ordered if the patient is agitated; prevent straining with bowel movements by giving stool softeners. — Agitation, pain, and Valsalva maneuvers (straining that increases chest and abdominal pressure) cause sudden spikes in blood pressure and intracranial pressure that can enlarge the a pooled collection of blood (hematoma) or cause rebleeding from the damaged vessel.
7. Monitor for signs of increased intracranial pressure: worsening headache, repeated vomiting, new confusion, decreased consciousness, or Cushing triad (high blood pressure with wide pulse pressure, slow heart rate, and irregular breathing); prepare mannitol 0.25 to 1 gram per kilogram IV or 3% hypertonic saline as ordered. — The growing a pooled collection of blood (hematoma) and surrounding swelling (edema) (brain swelling) compress brain tissue and raise pressure inside the rigid skull; osmotic agents pull excess water out of brain tissue into the bloodstream through osmosis (movement of water toward higher concentration of particles) and can temporarily reduce pressure until definitive neurosurgical treatment.

- 8.** Maintain normoglycemia (normal blood sugar) by checking blood glucose every 4 hours and treating levels above 180 mg/dL with insulin; avoid low blood sugar (hypoglycemia) below 70 mg/dL. — High blood sugar (Hyperglycemia) (high blood sugar) worsens brain injury through oxidative stress (cell damage from toxic byproducts) and inflammation that increases perihematomal swelling (edema) (swelling around the blood clot), while low blood sugar (hypoglycemia) (low blood sugar) deprives injured brain tissue of glucose needed for cellular energy and repair.

Sources

- AHA/ASA Guideline for the Management of Spontaneous Intracerebral Hemorrhage 2022
- Cochrane Systematic Reviews (acute stroke intervention)
- NEJM clinical review: Intracerebral Hemorrhage
- CDC Stroke Facts and Guidelines

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