

Subarachnoid Hemorrhage

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THE STORY IN ONE SENTENCE

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“Subarachnoid Bleeding (Hemorrhage) — worst headache ever (like being hit with a bat), get a CT scan right away, do a spinal tap if CT is clean, seal the leak with coils or a clip, and give nimodipine to stop blood vessels from squeezing shut.”

Normal anatomy — the body's job

Your brain floats in a thin layer of clear fluid called cerebrospinal fluid (CSF), held in a space called the subarachnoid space between two protective layers (the meninges). Blood vessels run through this space but stay sealed tight. No blood should ever leak into this cushion.

BODY'S JOB

The subarachnoid space cushions your brain from bumps and delivers nutrients. The blood vessels bring oxygen and sugar to feed every brain cell. The meninges wrap everything like a protective shell. When all of this works, your brain stays safe, fed, and at the right pressure.

1. Blood flows through brain arteries under pressure, like water through a garden hose.
2. The artery walls are strong and stretchy, holding the pressure without leaking.
3. Clear CSF bathes the brain in the subarachnoid space, absorbing shocks and washing away waste.
4. The meninges (brain's protective wrapping) stay smooth and quiet, not irritated.
5. Pressure inside the skull stays steady and low, so the brain has room to work.

What breaks

A weak balloon on a brain artery (aneurysm) bursts, or a tangle of abnormal vessels (AVM) bleeds, or a head injury tears a vessel. Blood floods the space around the brain where only clear fluid belongs.

CLINICAL TERM

Ruptured saccular (berry) aneurysm (80–85% of cases), arteriovenous malformation (AVM), traumatic vessel tear, or rarely perimesencephalic bleed (unknown cause, better outcome). The break dumps blood into the subarachnoid space, irritating the meninges and spiking intracranial pressure.

How the body compensates

The body tries to protect the brain by raising blood pressure to force oxygen through squeezed vessels, but this same response can make the weak spot bleed again. Early on, vital signs may look okay even as disaster brews inside the skull.

1. {'what': 'Blood pressure spikes (hypertensive response)', 'why': 'The brain senses rising pressure inside the skull and commands the heart to pump harder (Cushing reflex) to keep oxygen flowing. This buys time but risks rebleeding before the aneurysm is sealed.'}
2. {'what': 'Meninges inflame and stiffen the neck', 'why': 'Blood irritates the meninges (the brain's protective layers), triggering neck muscle spasm (nuchal rigidity) to splint and guard the head.'}
3. {'what': 'CSF tries to drain but may get blocked', 'why': 'Blood clots can clog the narrow drainage paths (aqueduct, fourth ventricle outlets), leading to hydrocephalus (fluid backup) and even higher pressure.'}
4. {'what': 'Vessels may go into spasm days later (vasospasm)', 'why': 'Breakdown products from the pooled blood (oxyhemoglobin) irritate artery walls, causing them to squeeze down (peak risk days 4–14). This chokes off oxygen and can cause stroke.'}
5. {'what': 'Brain may swell and herniate', 'why': 'If pressure climbs too high, the brain has nowhere to go and can push down through the skull opening (herniation), crushing the brainstem and stopping breathing.'}

Domino effect

1. Aneurysm or vessel ruptures, blood floods the subarachnoid space — The weak balloon or abnormal vessel bursts under pressure, spraying blood into the cushion space around the brain where only clear fluid belongs. This is the upstream break that starts everything.

2. Sudden thunderclap headache, worst of your life — Blood irritates pain-sensing nerves in the meninges (brain's protective wrapping) and spikes pressure inside the skull. Your brain's alarm goes off all at once — like being struck by lightning or hit with a bat.
3. Stiff neck, sensitivity to light, nausea, vomiting — Irritated meninges trigger neck muscle spasm (nuchal rigidity) to guard the head. The same nerve irritation makes bright light painful (photophobia). Rising pressure squeezes the vomiting center in the brainstem.
4. Confusion, sleepiness, or loss of consciousness — High pressure inside the skull squeezes brain tissue and cuts blood flow. The reticular activating system (the brain's wake-up switch in the brainstem) gets compressed, making you drowsy or unresponsive.
5. Blood pressure shoots up (hypertensive response) — The brainstem senses dangerously high skull pressure and orders the heart to pump harder (Cushing reflex) to force oxygen through. This keeps the brain alive short-term but risks rebleeding or stroke from the high pressure.
6. Risk of rebleeding before aneurysm is secured (peak in first 24 hours) — The torn vessel has only a fragile clot plugging it. Any pressure spike, straining, or time delay can blow the clot out and cause a second, often fatal bleed.
7. Vasospasm (vessel squeezing) days 4–14 after the bleed — Blood breakdown products (especially oxyhemoglobin) sitting in the subarachnoid space chemically irritate artery walls, causing smooth muscle to contract and narrow the vessel. This chokes oxygen delivery and can cause delayed stroke (delayed cerebral starved blood flow (ischemia)).
8. Hydrocephalus (fluid backup) or herniation (brain pushes down) — Blood clots block the narrow drainage channels for CSF, so fluid backs up and pressure climbs even higher. If unchecked, the swollen brain herniates through the skull base, crushing the brainstem and stopping breathing — the final, irreversible crash.

Symptoms · what the patient feels, and why

- Sudden, severe headache — often called the worst headache of your life: Blood bursts into the subarachnoid space (the thin layer between the brain and its protective covering). The blood irritates the meninges (the sensitive membranes that wrap the brain), triggering intense pain signals. The sudden spike in pressure inside the skull also stretches pain-sensing nerves.
- Stiff neck (can't easily bend your chin down toward your chest): The blood in the subarachnoid space irritates the meninges that cover the brain and spinal cord. Your neck muscles tighten up as a protective reflex to guard against moving the inflamed tissue.
- Sudden nausea and vomiting: The sudden rise in pressure inside the skull triggers the vomiting center in the medulla (the lower brainstem that controls automatic body functions like breathing and heart rate). Blood irritating the meninges also sends alarm signals that can make you feel sick to your stomach.

- Sensitivity to light (photophobia — bright light hurts your eyes or makes your headache worse): The irritated meninges connect to the nerve pathways that sense light — the optic nerves (which carry vision signals) and the trigeminal nerve (which senses pain in the face and head). When these pathways are already inflamed, bright light triggers more pain signals.
- Confusion or trouble staying awake (altered level of consciousness): Rising pressure inside the skull squeezes brain tissue and cuts down the blood flow that feeds the brain. The brain needs a steady supply of oxygen and sugar to stay alert. When that supply drops, thinking gets foggy and you may become drowsy or hard to wake up.
- Sudden loss of consciousness or fainting (syncope): The sudden bleed causes a sharp spike in intracranial pressure (pressure inside the skull), which can briefly stop normal blood flow to the brain. Without enough oxygen for a few seconds, you pass out. Some people also have a brief seizure from the irritation to the brain surface.
- Seizure (sudden jerking movements or blanking out): Blood in the subarachnoid space irritates the cortex (the surface layer of the brain where most thinking and movement control happens). This disrupts the normal electrical activity of brain cells and can cause abnormal, synchronized electrical bursts that show up as a seizure.
- Weakness or numbness on one side of the body, trouble speaking, or vision loss (focal neurologic deficits): The bleeding often comes from a ruptured aneurysm (a weak, bulging spot in a brain artery). Blood or pressure from the aneurysm can damage nearby brain tissue or squeeze arteries shut, cutting off oxygen to specific brain regions — for example, the motor cortex (which controls movement), Broca's area (which produces speech), or the visual cortex (which processes sight).
- Blurred or double vision: Rising pressure or bleeding near the brainstem can squeeze the cranial nerves that move the eyes — especially the oculomotor nerve (cranial nerve III), trochlear nerve (IV), and abducens nerve (VI). When these nerves are compressed, the muscles that aim your eyes don't work together, so you see double or things look blurry.

Focused exam

- Thunderclap headache — sudden, worst headache of life, reaches peak intensity within seconds to minutes: This is the alarm bell for bleeding around the brain. Blood rushes into the subarachnoid space (the cushion between the brain and its outer wrapping) and triggers instant, explosive pain. The headache does not build slowly — it screams 'bleed happening right now.' About 97% of patients with subarachnoid bleeding (hemorrhage) from a ruptured aneurysm describe this sudden, peak-intensity pattern.

- Neck stiffness (meningismus) — patient cannot bend neck forward to touch chin to chest: Blood in the subarachnoid space irritates the meninges (the three protective layers that wrap the brain and spinal cord). The body locks the neck muscles to guard against movement. This stiffness comes from chemical irritation by the blood itself — not infection like in meningitis.
- Light sensitivity (Photophobia) — bright light hurts the eyes, patient wants darkness: The irritated meninges make light signals painful as they travel through inflamed nerves. Turning off the lights does not fix the bleed, but the reaction tells you the brain's protective layers are soaked in blood and angry.
- Glasgow Coma Scale drop or altered mental status — confusion, drowsiness, or unresponsiveness: Blood and swelling press on brain tissue. As pressure climbs inside the skull, the brain shuts down. A low GCS (below 13) means the bleed is winning and the patient is in immediate danger of herniation (the brain getting squeezed down through the hole at the skull base). This can stop breathing and cause death within minutes.
- Focal neurologic deficit — one-sided weakness, slurred speech, vision loss, or facial droop: The bleed or vasospasm (blood vessels squeezing shut in reaction to the blood) is hitting a specific brain region. This tells you where the aneurysm (weak balloon bulge in the vessel wall) burst or where blood is pooling. The middle cerebral artery territory is most common for both bleeding and vasospasm-related stroke.
- Fundoscopy — retinal hemorrhages or subhyaloid bleeding (hemorrhage) (blood trapped between the retina and its inner surface layer): The blood pressure spike from the bleed can blow small vessels in the back of the eye. These eye bleeds (Terson syndrome) happen in about 10-15% of subarachnoid hemorrhages and are nearly certain proof of bleeding around the brain when present.

Diagnostics · every test answers a question

- Non-contrast head CT (computed tomography scan) → Is there blood in the space around the brain right now?
- Lumbar puncture with cerebrospinal fluid (CSF) analysis — red blood cell count and xanthochromia (visual inspection or spectrophotometry) → If the CT is negative but suspicion is high, is there blood breakdown in the spinal fluid?
- CT angiography (CTA) of the head and neck → Where is the ruptured aneurysm or vessel abnormality?
- Electrocardiogram (ECG) → Is the heart rhythm stable, or is the brain bleed triggering dangerous heart changes?
- Troponin I or T → Is the heart muscle injured from the stress of the brain bleed?
- Serum sodium → Is the patient developing low sodium from brain injury?

- Transcranial Doppler ultrasound (TCD) → Are the brain arteries squeezing shut (vasospasm) and threatening stroke?
- CT or MRI head (follow-up imaging) → Is hydrocephalus (fluid backup in the brain) or delayed starved blood flow (ischemia) (stroke from vasospasm) developing?

Treatment logic

- Secure the airway and stabilize blood pressure — target systolic BP 140-160 mmHg (or MAP 70-110 mmHg) until the aneurysm is repaired → Prevents rebleeding (which happens in about 4% of patients in the first 24 hours and jumps to 40% if untreated over 4 weeks) while keeping enough pressure to feed the injured brain. Too high and the aneurysm can burst again; too low and the brain starves because autoregulation (the brain's ability to control its own blood flow) is broken after the bleed. Intubation protects the airway if GCS < 8.
- Aneurysm repair — endovascular coiling or surgical clipping within 24 hours (earlier if feasible) → Seals the aneurysm sac so no more blood leaks into the subarachnoid space. This stops the upstream break that caused everything. Coiling threads platinum coils through a catheter (thin tube) inserted in the groin and guided into the aneurysm sac; clipping places a metal clip across the aneurysm neck during open brain surgery. Current guidelines (AHA/ASA 2012, updated 2023) favor early repair (< 24 hours) to prevent rebleeding. Choice between coiling and clipping depends on aneurysm location, shape, patient age, and neurosurgeon/interventionalist skill.
- Nimodipine 60 mg by mouth or nasogastric tube every 4 hours for 21 days → Prevents delayed cerebral starved blood flow (ischemia) (stroke from vasospasm, blood vessels squeezing shut) that peaks on days 4-14 after the bleed. Nimodipine (a calcium channel blocker that relaxes smooth muscle in vessel walls) is the ONLY medication proven in randomized trials to reduce death and severe disability (relative risk reduction about 34%). It works even if vasospasm still occurs on imaging, possibly by protecting neurons directly.
- External ventricular drain (EVD) placement if hydrocephalus develops (ventricular enlargement with rising intracranial pressure or declining mental status) → Drains excess cerebrospinal fluid when blood clogs the arachnoid granulations or the fourth ventricle outlet and fluid backs up in the brain ventricles (fluid-filled chambers). Without drainage, rising intracranial pressure causes coma, herniation (brain squeezing down through the skull base), and death. About 20-30% of patients need an EVD. Target intracranial pressure < 20 mmHg.

- Seizure prevention (prophylaxis) — levetiracetam 500-1000 mg intravenously or by mouth twice daily for 3-7 days → Prevents early seizures (within the first 72 hours) from blood irritating the cerebral cortex (the brain's outer processing layer). Seizures raise intracranial pressure, increase oxygen demand, and worsen brain injury. Routine long-term prevention (prophylaxis) (beyond 1 week) is NOT recommended unless a clinical or electrographic seizure actually occurs, because antiseizure drugs do not improve outcomes and may harm recovery.
- Transcranial Doppler ultrasound monitoring every 1-2 days from day 3 to day 14 → Detects vasospasm before stroke happens by measuring blood flow speed in the large brain arteries (especially the middle cerebral artery). Mean velocities > 120 cm/sec suggest vasospasm is starting; > 200 cm/sec means severe spasm with high stroke risk. Early detection allows targeted treatment (blood pressure augmentation, sometimes intra-arterial vasodilators like verapamil or nicardipine) before permanent damage occurs.
- Induced high blood pressure (hypertension) and euvolemia if delayed cerebral starved blood flow (ischemia) (vasospasm-related stroke symptoms) develops despite nimodipine → Raises cerebral blood flow (perfusion) pressure (the driving force pushing blood through narrowed, spastic vessels) by increasing mean arterial pressure to 100-120 mmHg (or higher, titrated to symptom reversal). Euvolemia (normal fluid balance, not volume overload) maintains cardiac output. This can reverse new neurologic deficits in 50-70% of cases if caught early. Older 'triple-H therapy' (hypertension, hypervolemia, hemodilution) is no longer standard because hypervolemia causes pulmonary edema (fluid flooding the lungs) and does not improve outcomes.

Medications

Nimodipine · Calcium channel blocker (dihydropyridine)

Nimodipine relaxes the smooth muscle in the walls of brain arteries. After a bleed in the space around the brain, these arteries can squeeze down tight (vasospasm) days later, starving brain tissue of oxygen. Nimodipine keeps calcium—the signal that tells muscle to squeeze—from flooding into the muscle cells in the artery wall, so the vessels stay open and blood flows.

1. Blood leaks into the subarachnoid space (the cushion layer around the brain) and breaks down, releasing chemicals that irritate artery walls.
2. These irritating chemicals cause calcium to rush into smooth muscle cells in the artery wall, telling the muscle to squeeze tight.
3. Nimodipine blocks L-type calcium channels—the gates that let calcium into these muscle cells.
4. With less calcium inside, the muscle cannot contract as hard, so the artery stays wider.

5. Wider arteries mean more blood flow, which lowers the risk of stroke from vasospasm during the danger window (days 4 to 14 after the bleed).

SAFETY WATCH

Blood pressure can drop, especially in the first few hours. Monitor closely and adjust other blood pressure medicines if needed. · Never give nimodipine intravenously (into a vein)—it can cause dangerous drops in blood pressure and heart problems. Always give by mouth or feeding tube. · Watch for dizziness, headache, or nausea—common but usually mild. · Use carefully in patients with low blood pressure or severe heart failure. · Check liver tests if using long-term, because the liver breaks down nimodipine.

Labetalol or nicardipine (for blood pressure control before the aneurysm is secured) · Labetalol: combined alpha and beta blocker; Nicardipine: calcium channel blocker

High blood pressure after a subarachnoid bleeding (hemorrhage) can push on the weakened aneurysm wall and cause it to bleed again before it is clipped or coiled. Labetalol blocks receptors (docking sites) on blood vessels and the heart that respond to adrenaline, slowing the heart and relaxing vessels. Nicardipine blocks calcium channels in artery walls, causing them to relax. Both lower the pressure inside the aneurysm, buying time until it can be repaired.

1. After a subarachnoid bleeding (hemorrhage), the unsecured aneurysm is like a weak balloon. High pressure can make it burst again.
2. Guidelines say keep systolic blood pressure below 140 to 160 mm Hg until the aneurysm is clipped (surgery) or coiled (catheter repair).
3. Labetalol or nicardipine is given into a vein so it acts within minutes and can be adjusted easily.
4. Labetalol blocks adrenaline's push on the heart and vessels. Nicardipine blocks calcium entry into vessel muscle.
5. Both smooth out the pressure wave hitting the aneurysm wall with each heartbeat, lowering the chance of re-bleeding.

SAFETY WATCH

Labetalol can hide signs of low blood sugar in diabetics. Use carefully in patients with asthma or severe heart block. · Nicardipine can cause reflex fast heart rate in some patients. · Monitor blood pressure continuously. Both drugs can overshoot and cause dangerously low pressure, which can cut blood flow to the brain. · Adjust dose carefully—small changes can have big effects. · Switch to pills once the aneurysm is secured and the patient is stable.

Levetiracetam or phenytoin (short-term seizure prevention) · Levetiracetam: synaptic vesicle protein modulator; Phenytoin: sodium channel blocker

Blood in the subarachnoid space and the injury from the bleed can irritate the brain's surface, making neurons (brain cells) fire in wild bursts—a seizure. Levetiracetam binds to a protein called SV2A on the storage bubbles that hold neurotransmitters (chemical messengers), making it harder for the cells to dump their signals all at once. Phenytoin blocks sodium channels on neuron membranes, stopping the rapid-fire electrical waves that spread a seizure.

1. Subarachnoid blood and increased pressure irritate the cortex (the brain's outer layer), lowering the seizure threshold.
2. Levetiracetam or phenytoin is often started in the first few days, especially if there is a lot of blood, brain swelling, or a large aneurysm.
3. Levetiracetam makes it harder for neurons to release their chemical messengers in a synchronized burst.
4. Phenytoin stabilizes the neuron membrane so it cannot fire over and over in the rapid pattern that spreads a seizure.
5. Most guidelines say stop after 7 days if no seizures occur, because long-term use does not improve outcomes and may slow recovery.

SAFETY WATCH

Levetiracetam can cause drowsiness, dizziness, and rarely mood changes (irritability or depression). Watch for personality shifts. · Phenytoin can cause gum overgrowth, rash (including serious allergic reactions like Stevens-Johnson syndrome), low blood counts, and confusion at high levels. Check drug levels and adjust dose. · Both can interact with other medicines broken down by the liver. · Stop after the high-risk period (usually 7 days) unless seizures occur. Prolonged use is not recommended. · Monitor for signs of seizure (jerking, staring, loss of awareness) and call the team right away if one happens.

Hypertonic saline (3%) or fludrocortisone (for cerebral salt wasting or symptomatic low sodium (hyponatremia)) · Hypertonic saline: concentrated salt solution; Fludrocortisone: synthetic mineralocorticoid

After subarachnoid bleeding (hemorrhage), the brain can send signals that make the kidneys dump too much sodium into the urine (cerebral salt wasting), or release too much ADH—antidiuretic hormone—which makes the body hold water and dilute the blood (SIADH). Both drop the sodium level in the blood (hyponatremia). Low sodium causes brain cells to swell and can lead to confusion, seizures, or coma. Hypertonic saline delivers concentrated sodium to raise blood sodium quickly. Fludrocortisone tells the kidneys to hold onto sodium and water, fixing the loss in cerebral salt wasting.

1. Subarachnoid bleeding (hemorrhage) triggers brain injury signals that disrupt the hypothalamus (the brain's hormone control center) and pituitary gland.
2. In cerebral salt wasting, the kidneys lose sodium. In SIADH, ADH makes the body hold water and dilute sodium.
3. Low blood sodium (less than 135 mEq per liter, often dropping below 125) is common in the first week and can worsen brain swelling.
4. Hypertonic saline is given into a vein to raise sodium quickly if the patient is symptomatic (confused, sleepy, seizing) or if sodium is dangerously low.
5. Fludrocortisone is given by mouth or feeding tube to fix cerebral salt wasting by making the kidneys hold onto sodium.
6. Treatment is guided by measuring blood sodium, urine sodium, and fluid balance. Raise sodium slowly—no more than 6 to 8 mEq per liter per day—to avoid osmotic demyelination syndrome (brain injury from correcting too fast).

SAFETY WATCH

Hypertonic saline: monitor sodium levels every 4 to 6 hours. Correcting too fast can cause osmotic demyelination syndrome (damage to the brain's white matter). Watch for fluid overload, especially in patients with heart or kidney disease. Requires central line placement for safe delivery. · Fludrocortisone: can raise blood pressure and cause fluid buildup, swelling, and low potassium (hypokalemia). Check potassium and blood pressure regularly. Do not use if SIADH is the cause—it will worsen water retention. · Tell cerebral salt wasting apart from SIADH by checking urine sodium, blood volume, and response to fluids. Treatment is opposite for the two.

Red flags

- Sudden, worst headache of your life — especially if it peaks within seconds to minutes (thunderclap headache)

- Loss of consciousness, seizure, or trouble staying awake after the headache starts
- Sudden weakness, numbness, trouble speaking, or vision loss on one side
- Stiff neck with severe headache and sensitivity to light — may signal bleeding around the brain
- Headache after head injury, especially if you passed out or vomited

Nursing priorities

1. Check airway patency and oxygen saturation every 15 minutes and prepare for intubation if Glasgow Coma Scale drops below 8 — A dropping level of consciousness means brain swelling or rebleeding and the patient cannot protect their airway from vomit or secretions which causes death from aspiration
2. Keep systolic blood pressure between 140 and 160 mmHg using IV labetalol or nicardipine until the aneurysm is secured — High blood pressure above 160 can blow open the aneurysm clot and cause rebleeding but too-low pressure under 140 starves the injured brain of oxygen and blood flow
3. Perform hourly neurological checks including Glasgow Coma Scale score and pupil size and reaction to light — Sudden changes in Glasgow Coma Scale or a blown pupil (dilated and not reactive) signal rebleeding or herniation where the brain pushes through the skull opening and death follows in minutes
4. Give nimodipine 60 mg by mouth or feeding tube every 4 hours for 21 days starting as soon as the diagnosis is confirmed — Nimodipine (a calcium channel blocker) prevents vasospasm (blood vessel squeezing) that peaks 7 to 10 days after the bleed and causes stroke in 30 percent of patients if untreated
5. Monitor EVD (external ventricular drain) output hourly and keep the drain at the ordered height above the tragus of the ear — The EVD drains excess cerebrospinal fluid to prevent hydrocephalus (fluid backup) that increases intracranial pressure and wrong drain height either over-drains and collapses the ventricles or under-drains and allows dangerous pressure
6. Keep the head of bed elevated to 30 degrees and maintain head in midline neutral position — Elevation helps cerebrospinal fluid drain and lowers intracranial pressure while twisting the neck kinks the jugular veins and traps blood in the skull raising pressure
7. Maintain a quiet dark room and limit visitors to prevent stimulation and straining — Straining with bowel movements and agitation from noise or visitors spike blood pressure suddenly and can rupture the aneurysm again before it is clipped or coiled

8. Hold all aspirin and anticoagulants and check PT INR and platelet count immediately — Blood thinners make the bleeding (hemorrhage) expand and any clotting disorder must be reversed with vitamin K fresh frozen plasma or platelets before surgery or the patient bleeds uncontrollably

Sources

- AHA/ASA Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage (2012, updated 2023)
- Neurocritical Care Society
- Cochrane Database of Systematic Reviews
- UpToDate Clinical Decision Support

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