

Acute Ischemic Stroke

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THE STORY IN ONE SENTENCE

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“Time is brain. Every minute a stroke goes untreated costs about 1.9 million neurons—call 911 the instant you see the signs.”

Normal anatomy — the body's job

The brain has zero fuel reserve and depends second by second on fresh blood arriving through four main pipes: two carotid arteries running up the front of the neck and two vertebral arteries climbing the back. These four pipes meet at the base of the brain and join into a backup ring called the circle of Willis, then branch out into smaller and smaller vessels that reach every patch of brain tissue.

BODY'S JOB

Deliver a nonstop stream of oxygen and glucose to every neuron so it can fire signals, think, move muscles, see, speak, and stay alive. If blood stops for even a few minutes, the cells start to shut down and die.

1. Heart — Pumps oxygen-rich blood up into the neck.
2. Carotid arteries (left and right, front of neck) and vertebral arteries (left and right, back of neck) — Four main pipes carrying blood into the head.
3. Circle of Willis (ring at the base of the brain) — Backup loop that can sometimes reroute blood if one pipe gets blocked.
4. Three big cerebral arteries on each side: anterior (front), middle (side), and posterior (back) — Each one feeds a different territory of the brain.
5. Tiny end-arteries branching off the big ones — Each little branch feeds one small patch—no overlap, no backup.

6. Neurons (brain cells) — Fire signals only as long as oxygen and glucose keep arriving every second.

What breaks

A blood clot gets stuck inside one of the brain arteries. The tissue directly downstream loses its blood supply in seconds. Around that dead center sits a border zone (called the penumbra) where cells are stunned and barely hanging on, kept alive by tiny backup vessels that reroute a trickle of blood from nearby. That border zone can still be saved if blood flow comes back fast enough.

CLINICAL TERM

Focal cerebral starved blood flow (ischemia) (not enough blood reaching the tissue) leads to failure of the sodium-potassium pump in neurons, causing cytotoxic swelling (edema) (cells swell because they cannot pump water back out). The a patch of dead tissue (infarct) core (dead center) forms within minutes. Surrounding it is the blood-starved (ischemic) penumbra (border zone with critically low but not zero flow), kept barely alive by collateral circulation (blood rerouted through small backup arteries on the brain surface and from the circle of Willis). The penumbra will convert to dead tissue unless reperfusion (restoring blood flow) happens within hours.

How the body compensates

The body tries to keep the border zone alive by opening small backup vessels (collaterals) that reroute blood around the block. Blood pressure often shoots up—the body is pushing harder to force flow through narrowed or blocked pipes. That high pressure is actually protective in the first forty-eight hours of a stroke and should not be lowered aggressively unless it is dangerously high (over 220 systolic or 120 diastolic), because dropping it too fast can kill the penumbra.

1. Collateral vessels (small backup arteries on the brain surface and connections through the circle of Willis) open wider and carry more blood around the blocked artery.
2. The small arterioles (tiny vessels) downstream from the clot dilate (widen) as far as they can to pull in whatever trickle of blood arrives, a process called autoregulation.
3. The body raises systemic blood pressure (the squeeze in every heartbeat) to push more blood through the partial block and through the collaterals.
4. Neurons in the penumbra slow down their firing and go quiet to save energy, but they are not dead yet—this is called electrical silence with preserved cell structure.
5. If blood flow is not restored within hours, the energy reserves run out and the penumbra converts to permanent a patch of dead tissue (infarct) (dead tissue that will never recover).

Domino effect

1. A clot lodges inside a brain artery. — Either a clot breaks off from the heart (cardioembolic, most often because atrial fibrillation (an irregular, quivering heartbeat) lets blood sit and clot in the left atrium) or a clot forms right on top of hard cholesterol plaque inside the brain artery itself (atherothrombotic). Less commonly, the artery wall tears (dissection) or a clot floats up from a narrowed carotid artery in the neck.
2. Blood flow to the tissue downstream stops or drops to a critical level. — Neurons have zero oxygen reserve. They depend on a constant supply arriving every second. When the pipe is blocked, oxygen delivery stops in that territory within seconds.
3. The center of the blocked territory (the core) dies within minutes; a stunned border zone (the penumbra) survives on a trickle of blood from collaterals. — The core is too far from any backup vessel. The penumbra sits close enough to collaterals to stay alive—but barely. That penumbra is what clot-busting treatment and thrombectomy are racing to save.
4. Function controlled by the dead or stunned region drops suddenly. — If the middle cerebral artery is blocked, the person loses strength and sensation on the opposite side of the body (face, arm, leg) and may lose the ability to speak or understand words (aphasia). If the posterior cerebral artery is hit, half the visual field goes dark (hemianopia). If the vertebral or basilar artery is blocked, balance, coordination, and consciousness can be lost.
5. Cytotoxic swelling (edema) (cell swelling) begins within hours. — Without oxygen, the sodium-potassium pumps inside neurons fail. Sodium and water flood into the cells, making them swell. The tissue gets puffy and presses on surrounding brain.
6. Hour by hour, more of the penumbra converts to permanent a patch of dead tissue (infarct) if flow is not restored. — Time is brain. Studies show that roughly 1.9 million neurons, 14 billion synapses, and 12 kilometers of nerve fibers are lost every minute a large-vessel stroke goes untreated.
7. Reperfusion therapy—tPA (clot-dissolving medicine) or mechanical thrombectomy (physically pulling the clot out with a wire basket)—restores blood flow. — tPA must be given within 4.5 hours of symptom onset (or within 4.5 hours of last-known-normal if the stroke was discovered on waking). Thrombectomy can be done up to 6 hours for most patients and up to 24 hours in select patients if imaging shows a large penumbra still alive. The earlier either treatment happens, the more brain is saved and the better the recovery.
8. After the acute event, secondary prevention starts to stop a second stroke. — The risk of another stroke is highest in the first 90 days. Treatment includes antiplatelet drugs (aspirin, clopidogrel) or anticoagulation (apixaban, rivaroxaban, warfarin) if atrial fibrillation (an irregular, quivering heartbeat) or another clotting source is found; a high-intensity statin (atorvastatin 80 mg or rosuvastatin 20–40 mg) to stabilize cholesterol plaques; blood pressure control (goal under 130/80 after the first few days); and smoking cessation, diabetes management, and lifestyle changes to lower all vascular risk.

Symptoms · what the patient feels, and why

- Sudden face droop on one side: A clot blocks blood flow to the motor cortex (the brain's movement control room), so the nerve signals that tell one side of the face to move stop working. The face sags because those muscles no longer get their orders.
- Sudden arm or leg weakness on one side: The corticospinal tract (the highway carrying movement commands from brain to muscles) loses its blood supply, so signals can't travel down. The arm or leg goes weak or numb because the brain can't tell it what to do.
- Slurred speech or trouble finding words: Broca's area (the speech-making zone) or Wernicke's area (the understanding-words zone) is starved of oxygen. Words get jumbled, slurred, or lost because those brain centers can't do their job without fuel.
- Loss of vision on one side in both eyes: The optic radiations (cables carrying sight signals) or the occipital cortex (the seeing center at the back of the brain) lose blood. One half of the visual field in both eyes goes dark, like a curtain dropping on one side.
- Sudden dizziness, loss of balance, or trouble walking: The cerebellum (balance control center) or brainstem (coordination highway) is cut off from blood. Without these working, the body can't coordinate movements or stay steady.
- Sudden severe headache unlike any before: This can signal a bleeding-driven (hemorrhagic) stroke (bleeding inside the skull instead of a clot). Blood leaking into the brain irritates tissues and raises pressure, causing intense pain. Must rule out bleeding before giving clot-busting medicine.
- Confusion or decreased alertness: A large clot blocks a major artery or affects the brainstem (the brain's power switch). The whole brain's oxygen supply drops, so thinking gets foggy and the person may become drowsy or unresponsive. This is dangerous because the airway can be lost.
- Double vision or trouble swallowing: A stroke in the brainstem damages cranial nerve nuclei (control centers for eye movements and throat muscles). Eyes don't line up anymore, and swallowing becomes hard or unsafe, risking choking.
- Sudden numbness on one side of the face, arm, or leg: The sensory cortex (feeling zone) or thalamus (relay station for touch and pain) loses blood. Nerve signals carrying touch, temperature, and pain can't reach the brain, so that side feels numb or tingly.
- Sudden loss of coordination in one arm or leg: The cerebellum or its connections are starved of oxygen. These parts fine-tune movements, so when they fail, the limb becomes clumsy—reaching misses the target, or the leg wobbles and stumbles.

Focused exam

- NIH Stroke Scale score 6 or higher: A score this high means the blockage is causing moderate-to-severe loss of brain function. It triggers an immediate call to the thrombectomy team—a specialized procedure that threads a tiny device through the blood vessel to pull the clot out, like fishing for a cork stuck in a pipe.
- Eyes both point toward one side, away from the weak arm and leg: The eyes look toward the side of the brain that is damaged. This tells you a major artery in the brain is blocked, not a small side branch. Major-vessel blockages often need the thrombectomy procedure to restore blood flow in time.
- One side of the face droops, but the forehead can still wrinkle: When the forehead can still move, the problem is in the brain itself (the outer layer called the cortex), not in the facial nerve outside the brain. This pattern confirms stroke rather than Bell palsy, which is a nerve problem that affects the entire half of the face including the forehead.
- When the person holds both arms straight out with palms up, one arm slowly drifts downward: This drift shows subtle weakness that the person may not feel yet. It reveals damage to the motor strip on the opposite side of the brain—the strip that sends movement commands down the spinal cord.
- Pulse is completely irregular with no repeating pattern: An irregularly irregular pulse is the hallmark of atrial fibrillation (an irregular, quivering heartbeat), a chaotic rhythm in the upper chambers of the heart. That chaotic rhythm lets blood pool and clot in the atrium; when a clot breaks free it rides the bloodstream up into the brain and plugs an artery. Finding atrial fibrillation changes your long-term plan: the person will need a blood thinner to prevent the next clot.

Diagnostics · every test answers a question

- Non-contrast CT scan of the head → Is blood leaking inside the skull, or is an artery blocked?
- CT angiography of the head and neck → Is one of the big arteries completely blocked?
- CT blood flow (perfusion) or MRI with diffusion-weighted imaging and FLAIR sequences → How much brain tissue is already dead, and how much is starving but still alive?
- Fingerstick blood glucose → Is low blood sugar copying stroke symptoms?
- 12-lead electrocardiogram and continuous heart-rhythm monitor (telemetry) → Is the heart rhythm chaotic, showing atrial fibrillation (an irregular, quivering heartbeat)?
- PT, INR, platelet count, and basic metabolic panel → Is it safe to give tPA or start a blood thinner?
- Troponin → Is the heart muscle damaged at the same time?

Treatment logic

- IV alteplase (tPA) 0.9 mg per kilogram of body weight, maximum dose 90 mg, given within 4.5 hours of last-known-well time → Alteplase dissolves the fibrin threads holding the clot together, like unraveling a tangled net. Blood flow rushes back into the starving penumbra, and brain cells that were on the edge of death start working again. Many people regain speech or movement within hours.
- IV tenecteplase 0.25 mg per kilogram (maximum 25 mg) as a single push over 5 seconds, within 4.5 hours → Tenecteplase works the same way as alteplase—it breaks up the clot—but you give the entire dose in one quick push instead of an hour-long drip. The AcT and NOR-TEST trials showed it works just as well, and many stroke centers now prefer it because it is faster and simpler.
- Mechanical thrombectomy: threading a catheter up through the groin artery into the brain, then using a tiny cage (stent retriever) or suction tube to pull the clot out → For large-vessel blockages, thrombectomy is the most powerful treatment we have. It physically yanks the clot out of the artery. The HERMES meta-analysis pooled seven trials and proved that one in three people treated with thrombectomy will walk out of the hospital independent, compared to one in five with medical therapy alone. With blood flow (perfusion) imaging, you can offer it up to 24 hours after stroke onset if there is still a large penumbra (DAWN and DEFUSE-3).
- Let blood pressure run high: systolic up to 220 mmHg and diastolic up to 120 mmHg before reperfusion → When an artery is blocked, high pressure forces blood through tiny side channels (collaterals) around the blockage, keeping the penumbra alive a little longer. Lowering pressure too soon can make the starved blood flow (ischemia) worse, like turning down the water pressure when you are already getting a trickle.
- Aspirin 325 mg by mouth, started 24 to 48 hours after tPA (or right away if no tPA was given) → Aspirin stops platelets from clumping together. The CAST and IST trials showed it lowers the chance of another stroke in the first two weeks by about 1 percent—a small but real benefit when you are treating thousands of people.
- High-intensity statin: atorvastatin 80 mg or rosuvastatin 40 mg once daily, started in the hospital → Statins glue a cap over unstable cholesterol plaques in the arteries, making them less likely to crack open and form a clot. The SPARCL trial showed that high-dose statin cuts the risk of a second stroke by roughly 20 percent over the next five years.
- Oral anticoagulation with apixaban, rivaroxaban, edoxaban, or dabigatran if atrial fibrillation (an irregular, quivering heartbeat) is confirmed → Direct oral anticoagulants block the clotting cascade—the chain reaction that turns liquid blood into solid clot. In people with atrial fibrillation (an irregular, quivering heartbeat), these drugs cut embolic stroke risk by about 65 percent compared to aspirin alone (RE-LY, ROCKET-AF, ARISTOTLE, ENGAGE AF-TIMI 48).

Medications

Alteplase (tPA) · Tissue plasminogen activator (fibrinolytic)

Turns on plasminogen, a natural protein in your blood that breaks down clots. Once activated, plasminogen becomes plasmin—an enzyme that cuts the clot into pieces. The clot dissolves and blood flow returns to brain tissue that was starving for oxygen.

1. Alteplase lands on fibrin threads inside the clot.
2. It turns plasminogen (sleeping protein) into plasmin (active enzyme).
3. Plasmin chews through the fibrin threads, breaking the clot apart.
4. Blood flows again to the penumbra—brain tissue around the dead center that can still be saved.

SAFETY WATCH

Must get a CT scan of the head (no dye) before giving tPA to make sure there is no bleeding in the brain. · Blood pressure must be under 185/110 mmHg before you give tPA, and must stay under 180/105 mmHg for 24 hours after, or bleeding risk goes up. · No blood thinners (heparin, warfarin) or antiplatelet drugs (aspirin, clopidogrel) for 24 hours after tPA. · Check how the patient moves, talks, and thinks every 15 minutes for the first 2 hours, then every 30 minutes for 6 hours, then every hour for 16 hours. This catches bleeding-driven (hemorrhagic) transformation—when the stroke zone starts bleeding. · Only give within 4.5 hours of symptom start in most patients (3 hours if over age 80, prior stroke, diabetes, or on anticoagulants). · Do not give if INR over 1.7, platelets under 100,000, blood sugar under 50 mg/dL, recent surgery in last 14 days, or history of brain bleed.

Aspirin · Antiplatelet (COX-1 inhibitor)

Blocks the enzyme COX-1 inside platelets forever. COX-1 makes thromboxane A2, a chemical that tells platelets to stick together. When COX-1 is off, platelets cannot clump and form new clots on damaged artery walls.

1. Aspirin permanently shuts off COX-1 in platelets.
2. No thromboxane A2 is made.
3. Platelets lose their sticky signal and cannot pile onto cholesterol plaques.
4. New clots do not form as easily.

SAFETY WATCH

Wait 24 hours after tPA before giving aspirin—earlier use raises bleeding risk. · Watch for stomach or gut bleeding: black tarry stool, vomit that looks like coffee grounds, or dropping hemoglobin on blood tests. · Give 160–325 mg within 24–48 hours of stroke symptom start (AHA/ASA guideline) unless tPA was given.

Clopidogrel · Antiplatelet (P2Y12 inhibitor)

Blocks the P2Y12 receptor on platelets. This receptor is a docking site for ADP, a chemical that activates platelets and makes them sticky. When the receptor is blocked, platelets cannot respond to ADP and stay calm instead of clumping.

1. Clopidogrel plugs the P2Y12 receptor on the platelet surface.
2. ADP (adenosine diphosphate) cannot land and send the 'stick together' signal.
3. Platelets do not activate or clump.
4. New clots form more slowly.

SAFETY WATCH

Do not give within 24 hours of tPA. · Watch for unusual bruising, nosebleeds, blood in urine, or prolonged bleeding from cuts. · Often used instead of aspirin if the patient cannot tolerate aspirin, or combined with aspirin for 21–90 days after high-risk minor stroke or TIA (POINT and CHANCE trial data). · Requires liver enzyme CYP2C19 to activate—some patients are poor metabolizers and get less benefit.

Dual Antiplatelet Therapy (DAPT): Aspirin + Clopidogrel · Combination antiplatelet regimen

Uses two drugs that block platelets in different ways. Aspirin stops thromboxane A2 production, and clopidogrel blocks the ADP receptor. Together they shut down two main pathways that make platelets sticky, lowering the chance of a second stroke in the next few weeks.

1. Aspirin turns off COX-1, stopping thromboxane A2.
2. Clopidogrel blocks P2Y12, stopping ADP signals.
3. Platelets lose two activation pathways at once.
4. New clot formation drops sharply for 21–90 days after a minor stroke or TIA.

SAFETY WATCH

Use only for minor stroke (NIH Stroke Scale ≤ 3) or high-risk TIA (ABCD2 score ≥ 4). · Start within 24 hours of symptom onset and continue for 21–90 days (guideline: AHA/ASA 2021). · Do not use if patient received tPA or if there is any sign of bleeding. · After 21–90 days, switch to single antiplatelet therapy to lower bleeding risk. · Watch closely for any signs of bleeding—bruising, blood in stool or urine, gum bleeding.

Apixaban, Rivaroxaban, Dabigatran, or Warfarin · Anticoagulants (oral)

Blocks clotting factors in the blood (proteins that build clots). Apixaban and rivaroxaban block Factor Xa, the enzyme that turns prothrombin into thrombin. Dabigatran blocks thrombin directly. Warfarin blocks vitamin K, which the liver needs to make several clotting factors. All four reduce clot formation from the heart in patients with atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat that lets blood pool and clot in the atrium).

1. Drug blocks a step in the clotting cascade (Factor Xa, thrombin, or vitamin K cycle).
2. Clotting factors cannot assemble into a stable clot.
3. Blood stays thinner and clots form more slowly.
4. Risk of stroke from atrial fibrillation (an irregular, quivering heartbeat) drops.

SAFETY WATCH

Start anticoagulation 4–14 days after blood-starved (ischemic) stroke, depending on stroke size and risk of bleeding-driven (hemorrhagic) conversion (AHA/ASA guideline). · Do not start within 24 hours of tPA. · Warfarin requires regular INR blood tests (goal 2.0–3.0); DOACs (apixaban, rivaroxaban, dabigatran) do not. · Watch for bleeding: black stool, blood in urine, severe headache, unusual bruising. · Use only if stroke was caused by atrial fibrillation (an irregular, quivering heartbeat) or another cardioembolic source—not for atherosclerotic strokes.

Atorvastatin or Rosuvastatin · HMG-CoA reductase inhibitor (statin)

Blocks HMG-CoA reductase, the enzyme the liver uses to make cholesterol. When cholesterol production drops, the liver pulls LDL cholesterol out of the blood to use instead. Blood LDL levels fall. Lower LDL means less cholesterol builds up in artery walls, slowing plaque growth and stabilizing plaques so they do not rupture and cause another stroke.

1. Statin blocks HMG-CoA reductase in liver cells.
2. Liver cannot make as much cholesterol.
3. Liver pulls LDL cholesterol from the blood to meet its needs.
4. LDL cholesterol levels drop; existing plaques stabilize.

SAFETY WATCH

Start high-intensity statin (atorvastatin 40–80 mg or rosuvastatin 20–40 mg) for all blood-starved (ischemic) stroke or TIA patients with atherosclerotic cause, regardless of baseline LDL (AHA/ASA 2021). · Check liver enzymes (ALT, AST) at baseline and if patient has symptoms. · Watch for muscle pain or weakness; check creatine kinase if symptoms appear. · Goal LDL <70 mg/dL, or ≥50% reduction from baseline.

Antihypertensives (ACE inhibitors, ARBs, thiazide diuretics, calcium channel blockers) · Blood pressure lowering agents

Lower blood pressure through different pathways. ACE inhibitors (lisinopril, enalapril) block angiotensin II formation, relaxing arteries. ARBs (losartan, valsartan) block angiotensin II receptors. Thiazide diuretics (hydrochlorothiazide) remove sodium and water through urine. Calcium channel blockers (amlodipine) relax artery walls. All reduce the force blood pushes on artery walls, lowering stroke risk over time.

1. Drug targets one pathway that raises blood pressure.
2. Blood vessels relax or fluid volume drops.
3. Blood pressure falls.
4. Lower pressure reduces strain on artery walls and slows plaque damage.

SAFETY WATCH

Do NOT lower blood pressure aggressively in the first 24–48 hours of acute stroke unless BP is >220/120 mmHg or patient received tPA. · Start or resume antihypertensive therapy a few days after stroke once the patient is stable (AHA/ASA guideline). · Goal long-term blood pressure <130/80 mmHg to prevent recurrent stroke. · Monitor kidney function (creatinine, eGFR) and potassium with ACE inhibitors and ARBs.

Red flags

- Sudden severe headache with the deficit—may be bleeding in the brain instead of a clot; get an emergency CT scan immediately before giving any blood thinner
- Neurologic symptoms getting rapidly worse after starting tPA—suggests bleeding inside the skull from the clot-buster; stop the infusion, repeat CT right away, and reverse the medicine with clotting factors and platelets
- Coma with tiny pinpoint pupils—basilar artery stroke cutting off blood to the brainstem; very high risk of death; secure the airway and consider emergency thrombectomy to pull the clot out

- Blood pressure above 185/110 before giving tPA—too high increases bleeding risk; must lower it carefully with IV medicine like labetalol or nicardipide before starting the clot-buster
- Seizure at the moment stroke symptoms start—raises the chance the clot will turn into bleeding later; confirm with imaging before giving reperfusion therapy

Nursing priorities

1. Confirm last-known-well time with a reliable witness — Drives every treatment decision.
2. Activate the stroke alert - CT within 25 min of arrival — Every minute of delay costs brain.
3. Hold oral intake until swallow screen is passed — Aspiration is the #1 preventable cause of post-stroke pneumonia.
4. Q15 neuro checks x2 h post-tPA, then q30 x6 h — Detects bleeding-driven (hemorrhagic) conversion early.
5. Head of bed flat unless BP or oxygenation demands 30 degrees — Maximises collateral blood flow (perfusion).
6. Fall precautions and skin protection — Paralysis on one side (Hemiplegia) + inattention = high fall and pressure-injury risk.

Sources

- AHA/ASA 2019 Guidelines for the Early Management of Acute Ischemic Stroke
- AHA/ASA 2018 Guidelines for Endovascular Treatment
- NINDS tPA Stroke Study
- DAWN and DEFUSE-3 trials

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