

Stroke Window

In an acute blood-starved (ischemic) stroke, every minute that passes kills more brain cells — the clock decides whether clot-busting medicine or a catheter rescue is still safe to use.

THE ONE-LINE TRUTH

A clot is blocking an artery in the brain, killing brain tissue minute by minute.

The chain — how it unfolds

1. A clot blocks a cerebral artery — why: The clot usually forms on a ruptured cholesterol plaque in the artery wall (atherothrombotic stroke) or flies in from the heart during atrial fibrillation (an irregular, quivering heartbeat) (cardioembolic stroke), lodging where the vessel narrows — commonly the middle cerebral artery (MCA) or its branches.
2. Neurons downstream run out of oxygen and ATP within minutes — why: Brain cells burn glucose and oxygen to make ATP (the energy currency). With no blood flow, ATP is gone in 5 minutes, ion pumps fail, sodium floods in, the cell swells and bursts — this is the a patch of dead tissue (infarct) core, and it is dead.
3. The penumbra enters electrical silence but stays alive on collateral trickle — why: The tissue just beyond the core is getting 10–20 mL/100 g/min from pial collaterals (normal is 50) — enough to keep cells alive but not enough to fire, so the region goes quiet (which is why the patient loses function) but can wake up if you restore flow fast.
4. Clinical deficit maps to the blocked artery — why: Each artery feeds a specific brain territory: MCA → lateral cortex (face droop, arm > leg weakness, loss of language ability (aphasia) if left side, neglect if right side); ACA → medial cortex (leg > arm weakness, personality blunting); PCA → occipital lobe (homonymous hemianopsia, cortical blindness); basilar → brainstem and cerebellum (locked-in syndrome, quadriplegia, coma, a spinning sensation (vertigo), loss of coordination (ataxia), cranial-nerve palsies). The pattern of what is lost tells you which vessel is plugged.

5. Blood pressure spikes (permissive high blood pressure (hypertension)) — why: The brainstem's vasomotor center (in the rostral ventrolateral medulla) detects low cerebral blood flow (perfusion) pressure and fires sympathetic nerves to squeeze peripheral arteries and jack up systemic pressure, trying to drive collateral flow. Guidelines say **do not** lower BP in the first 24 hours unless it is $>220/120$ (or $>185/110$ if giving alteplase), because dropping pressure can kill the penumbra.
6. The time window closes as penumbra converts to core — why: Every minute, roughly 1.9 million neurons die. IV alteplase (clot-buster) is proven effective up to 4.5 hours in most patients; endovascular thrombectomy (catheter clot removal) is proven up to 24 hours **if** imaging (CT blood flow (perfusion) or MRI diffusion/FLAIR mismatch) shows a large penumbra still alive. Outside the window, treatment risk (bleeding, no salvageable tissue) outweighs benefit.
7. Cerebral swelling (edema) peaks at 24–72 hours in large strokes — why: Dead neurons swell with water; in a massive MCA stroke (malignant MCA syndrome), swelling pushes the brain sideways (midline shift on CT), compresses the opposite hemisphere, and herniates the uncus or cerebellar tonsils, crushing the brainstem and causing sudden death. Decompressive hemicraniectomy (removing a skull flap) within 48 hours cuts mortality by half in patients under 60.
8. Reperfusion may cause bleeding-driven (hemorrhagic) transformation — why: Starved blood flow (Ischemia) damages the blood-brain barrier; when blood flow returns (spontaneously or after treatment), red cells leak through the broken barrier into dead tissue. Petechial (tiny-dot) bleeding (hemorrhage) is common and harmless; symptomatic parenchymal a pooled collection of blood (hematoma) (big blood clot in the brain) happens in ~6% after alteplase and worsens outcome.

Spot it — what you see, and why

- Sudden weakness or numbness on one side of the face, arm, or leg: A blocked or burst blood vessel in the brain stops oxygen from reaching the cells in one area, so the brain can't send signals to the muscles on the opposite side of the body—that side droops or goes weak.
- Sudden trouble speaking or slurred speech: When the language centers in the brain (usually on the left side, in areas called Broca's area and Wernick's area) lose their blood supply, the person can't form words clearly or can't understand what others are saying.
- Sudden vision loss or blurred vision in one or both eyes: The occipital cortex (the back of the brain that processes what you see) or the pathways that carry vision signals are starved of oxygen, so the picture the brain builds goes dark or fuzzy.

- Sudden severe headache with no clear cause: When a blood vessel bursts inside the skull (a bleeding-driven (hemorrhagic) stroke), blood pushes on the brain and irritates pain-sensing nerves in the meninges (the covering layers of the brain), causing intense, sudden head pain.
- Sudden dizziness, loss of balance, or trouble walking: The cerebellum (the back-bottom part of the brain that keeps you balanced) or the brainstem (which coordinates movement signals) runs out of oxygen, so the body can't keep steady or walk in a straight line.
- Sudden confusion or trouble understanding what is happening: Parts of the cerebral cortex (the outer thinking layer of the brain, especially the frontal and parietal lobes) lose blood flow, so the brain can't process thoughts, make sense of surroundings, or remember the moment clearly.

Red flags — act now

- Symptoms started less than 24 hours ago (the treatment window is still open—this is a medical emergency)
- Sudden severe headache, vomiting, or decreased alertness (may signal bleeding in the brain)
- Blood pressure over 185/110 mm Hg (high pressure raises the risk of bleeding if clot-busting medicine is given)
- Blood sugar below 50 mg/dL or above 400 mg/dL (very low or very high sugar can mimic stroke and must be fixed first)
- Patient is on blood thinners or has a bleeding disorder (raises the risk of dangerous bleeding during treatment)

Treat it — in this order

1. IV alteplase (0.9 mg/kg, max 90 mg) or tenecteplase (0.25 mg/kg, single bolus) within 4.5 hours of last known well time → The clot-busting drug dissolves the blood clot blocking the artery, letting blood flow back to the penumbra (struggling brain tissue) before it dies. The sooner you give it, the more brain you save. Time is brain.
2. Mechanical thrombectomy (catheter to pull out the clot) for large vessel blockage (occlusion), up to 24 hours from last known well in selected patients with favorable imaging → The catheter physically removes the clot from the blocked artery, restoring blood flow directly without waiting for medicine to dissolve it. Works even when clot-busting drugs alone are not strong enough. Can extend treatment window to 24 hours if imaging shows large penumbra and small core (DAWN/DEFUSE-3 criteria).

3. Blood pressure control: keep $< 185/110$ mmHg before thrombolysis, $< 180/105$ mmHg for 24 hours after thrombolysis → High blood pressure in the first hours of stroke can cause bleeding into the dead tissue, especially after clot-busting medicine. Controlled pressure lets the medicine work without that bleeding risk.
4. Aspirin 325 mg within 24–48 hours after stroke onset (but NOT in the first 24 hours after thrombolysis) → Aspirin stops platelets from clumping and forming new clots in the same artery or elsewhere, reducing the risk of another stroke by about 13 per 1,000 patients treated (ISIS-2, CAST, IST trials).
5. High-intensity statin therapy (atorvastatin 80 mg or rosuvastatin 20 mg daily) started in hospital → Statins lower LDL cholesterol (the bad kind that clogs arteries) and stabilize plaques so they do not rupture and throw clots. Reduces future stroke and heart attack risk. Start in hospital regardless of baseline cholesterol (SPARCL trial).

Your top priorities at the bedside

1. Establish exact last known well time and notify the stroke team immediately so the physician can evaluate for thrombolysis within 4.5 hours. — why: Every 15 minutes of delay causes more starved blood flow (ischemia) (lack of blood flow), killing approximately 120 million neurons. Time is brain. Thrombolysis eligibility depends on the clock from last known well, and mechanical thrombectomy decisions require rapid imaging to identify large vessel occlusions (blockages in major brain arteries).
2. Check blood pressure every 15 minutes and keep it under 185/110 mmHg before thrombolysis and under 180/105 mmHg for 24 hours after thrombolysis, using IV labetalol or nicardipine as ordered. — why: High blood pressure during thrombolysis increases the risk of bleeding-driven (hemorrhagic) transformation (the stroke turning into a bleed inside the brain). The clot-busting drug breaks down not only the clot but also normal clotting, so fragile blood-starved (ischemic) blood vessels can rupture under high pressure, causing fatal brain bleeding (hemorrhage).
3. Maintain the head of the bed flat or at 30 degrees or less unless the patient has airway concerns, and avoid flexing the neck. — why: Flat positioning uses gravity to maximize cerebral blood flow (perfusion) pressure (blood flow to the brain). The penumbra (the border zone of dying but salvageable brain tissue around the a patch of dead tissue (infarct) core) survives on collateral flow (backup blood supply), and even a 10 mmHg pressure increase from lying flat may save tissue during the acute window.