

Stroke Window

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The brain is fed by a network of arteries — the internal carotid arteries split into the anterior cerebral artery (ACA, which feeds the leg strip on top of the brain) and the middle cerebral artery (MCA, which feeds the face, arm, and language centers on the side). The vertebral arteries join to form the basilar artery, which splits into the posterior cerebral arteries (PCA, feeding the vision centers in the back). Autoregulation keeps blood flow steady: when blood pressure drops, the arteries widen; when pressure rises, they tighten. Collateral vessels (tiny backup loops, like the circle of Willis at the base of the brain and pial collaterals on the surface) can sometimes reroute blood around a blockage and keep tissue alive downstream.

BODY'S JOB

These arteries deliver oxygen and glucose to brain cells, which cannot store energy and die within minutes if the blood supply is cut. Each artery feeds a specific slice of brain real estate, so the job that fails (speech, vision, leg movement) tells you exactly which artery is blocked.

1. Oxygenated blood leaves the heart and travels up the carotid and vertebral arteries in the neck.
2. The internal carotid arteries enter the skull and split into the ACA (feeding the top middle strip, where leg movement lives) and the MCA (feeding the side strip, where face, arm, and speech live).
3. The two vertebral arteries join at the base of the brainstem to form the basilar artery, which then splits into the right and left PCA (feeding the occipital lobes, where vision is processed).

4. At the base of the brain, these arteries connect in a ring called the circle of Willis, which acts as a safety valve: if one artery clogs, blood can sometimes loop around from another.
5. Smaller pial collaterals on the brain surface link neighboring artery territories, offering another backup route.
6. Autoregulation keeps cerebral blood flow around 50 mL per 100 g of brain per minute, even when systemic blood pressure swings between roughly 60 and 150 mmHg mean arterial pressure.

What breaks

A clot suddenly blocks one of the brain's arteries, cutting off oxygen to everything downstream.

CLINICAL TERM

Acute blood-starved (ischemic) stroke: a a blood clot (thrombus) (clot that forms on the spot, usually on top of a ruptured cholesterol plaque) or a traveling clot (embolus) (clot that travels from the heart or a vessel upstream — common sources are atrial fibrillation (an irregular, quivering heartbeat), a recent heart attack, or carotid plaque) lodges in a cerebral artery and stops blood flow.

How the body compensates

The body tries to save the penumbra by raising blood pressure to force more blood through collaterals. Vital signs may look stable for hours, but the brain is silently dying. The danger is missing the tipping point — when swelling crushes the brainstem, when reperfusion causes bleeding (hemorrhage), or when the airway is lost — because once it tips, recovery may be impossible.

1. {'step': 'Blood pressure rises (often to 160–220 systolic)', 'why': 'The brainstem's pressure centers (in the medulla) sense low oxygen and low flow in the blood-starved (ischemic) territory and crank up sympathetic tone, raising heart rate and constricting arteries everywhere else to drive more blood toward the dying brain. This is called autoregulatory or blood-starved high blood pressure (hypertension).'} }
2. {'step': 'Heart rate and respiratory rate increase', 'why': 'The body shifts into fight-or-flight mode to maximize oxygen delivery; a fast heart rate (tachycardia) and fast breathing (tachypnea) are normal acute-stress responses.'} }
3. {'step': 'Collateral vessels dilate maximally', 'why': 'Arteries downstream of the clot sense low oxygen and release local signals (adenosine, nitric oxide) that relax vessel walls to their widest diameter, trying to pull in every available drop of blood from backup routes.'} }

4. {'step': 'Glucose is shunted preferentially to the brain', 'why': 'Stress hormones (cortisol, epinephrine) raise blood sugar, and the brain — normally first in line for glucose — takes what it can to keep neurons firing as long as possible.'}
5. {'step': 'The danger zone: these defenses mask how bad things are', 'why': 'A patient may have a normal or high blood pressure, normal oxygen saturation, and look 'stable' on the monitor while millions of neurons are dying. The clock is ticking silently.'}
6. {'step': 'Watch for cerebral swelling (edema) (brain swelling)', 'why': 'Dead neurons swell with water (cytotoxic swelling (edema)) within hours; by 24–72 hours a large stroke can swell so much it shifts the brain sideways (midline shift) and herniates the brainstem downward through the skull opening, causing coma and death. Younger patients with massive MCA infarcts may need decompressive hemicraniectomy (removing part of the skull) within 48 hours to prevent herniation.'}
7. {'step': 'Watch for bleeding-driven (hemorrhagic) transformation', 'why': 'After clot-busting medicine (IV alteplase) or thrombectomy, reperfusion can flood damaged, leaky vessels and cause bleeding into the dead tissue (hemorrhagic transformation) — symptomatic bleeding happens in roughly 6% and can be fatal.'}
8. {'step': 'Watch for aspiration', 'why': 'Strokes that damage the brainstem or cortex often knock out the swallowing reflex, so food, spit, or vomit silently slide into the lungs (aspiration), causing pneumonia or sudden airway obstruction.'}
9. {'step': '"Trend, don't snapshot"', 'why': 'A stable exam at hour 2 is meaningless if the NIHSS (stroke severity score) is climbing from 8 to 15 to 22 — the patient is crashing in slow motion. Always compare the current state to the last check.'}

Domino effect

1. A clot blocks a cerebral artery — 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 — 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 — 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 — 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)) — 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 — 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 — 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 — 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.

Symptoms · what the patient feels, 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.
- Face droop on one side (one side of the smile sags): The motor cortex (the strip of brain that sends movement commands) or the motor pathways that control facial muscles are cut off from oxygen, so the muscles on one side of the face go limp.
- Arm drift (one arm drifts downward when both arms are held out): Damage to the motor cortex or the descending pathways (the nerve highways that carry 'move' signals down the spinal cord) means one arm no longer gets the signal to stay lifted, so it sinks down without the person noticing.

Focused exam

- Last known well time documented precisely: In acute blood-starved (ischemic) stroke, this time — not when someone found the patient — starts the clock for which treatments can work. The stroke window counts from when the brain was last definitely normal.
- Sudden focal neurologic deficit matching one arterial territory: The body part that stops working tells you which artery is blocked. One blocked artery starves one region. MCA (middle cerebral artery) stroke loses face and arm and speech. ACA (anterior cerebral artery) stroke loses the leg. PCA (posterior cerebral artery) stroke loses vision. This match proves the diagnosis.
- National Institutes of Health Stroke Scale (NIHSS) score: This number measures how bad the stroke injury is. A high score (typically ≥ 6) means big clot burden and higher chance the patient needs mechanical thrombectomy (a catheter to pull the clot out), not just medicine.

- Blood pressure before and after any clot-busting medicine: In acute blood-starved (ischemic) stroke, the body raises blood pressure on purpose to push blood past the clot to save the penumbra (brain tissue that is struggling but not dead yet). If you drop the blood pressure too fast, you kill the tissue you were trying to save. Must be under 185/110 mmHg before thrombolysis (clot-busting medicine), under 180/105 mmHg after.
- Ability to swallow before feeding: A stroke can paralyze the swallowing muscles. Food going into the lungs instead of the stomach causes aspiration pneumonia (lung infection from food or spit). Swallow screen first, feeding tube if needed — food only when safe.

Diagnostics · every test answers a question

- Non-contrast head CT → Is there bleeding in the brain, and is there visible early blood-starved (ischemic) change (darkened tissue from the stroke)?
- CT angiography (CTA) of head and neck → Is there a large vessel blockage (occlusion) (big artery blocked), and where exactly is the clot?
- CT blood flow (perfusion) or MRI diffusion/blood flow imaging → How much brain tissue is already dead (core), and how much is struggling but salvageable (penumbra)?
- Serum glucose → Is the glucose very low, mimicking stroke symptoms, or very high, worsening brain injury?
- Complete blood count (CBC) with platelet count → Are platelets too low to clot safely, raising bleeding risk with thrombolysis?
- PT/INR and aPTT → Is the blood too thin from anticoagulants (blood thinners), raising bleeding risk?
- Electrocardiogram (ECG) and telemetry monitoring → Is atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat) present, suggesting the clot came from the heart?
- Troponin and BNP (if cardiac source suspected) → Is there heart damage or heart failure that could throw clots to the brain?

Treatment logic

- 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.

- 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).
- 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.
- 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).
- 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).
- Anticoagulation (apixaban, rivaroxaban, dabigatran, or warfarin) for atrial fibrillation (an irregular, quivering heartbeat) or other cardioembolic source, started after brain imaging rules out bleeding-driven (hemorrhagic) transformation (typically 4–14 days post-stroke depending on size) → Anticoagulation stops the blood from clotting in the heart, preventing new clots from traveling to the brain. Reduces stroke recurrence by about 60% in atrial fibrillation (an irregular, quivering heartbeat) (SPAF, ARISTOTLE, RE-LY trials).
- Decompressive hemicraniectomy (surgical removal of part of the skull) for malignant MCA tissue death from blocked blood flow (infarction) with brain swelling in patients ≤ 60 years old, within 48 hours of symptom onset → Removing part of the skull gives the swollen brain room to expand instead of crushing the brainstem. Saves life in young patients with massive stroke and brain herniation. Reduces mortality from 80% to 30% (DESTINY, DECIMAL, HAMLET trials).

Medications

Alteplase (tPA) or tenecteplase (TNK) · Thrombolytic (clot-buster)

Breaks apart the blood clot blocking the artery in the brain so oxygen-rich blood can flow again and save dying brain cells.

1. The drug is injected into a vein and travels through the bloodstream to the brain.

2. It activates plasminogen (a natural protein in the blood) and turns it into plasmin (a protein-cutting enzyme).
3. Plasmin cuts apart fibrin (the tough threads that form the clot's structure), breaking the clot into tiny pieces.
4. Blood flow restarts through the artery, bringing oxygen and sugar to starving brain tissue.
5. This rescue must happen within 3 to 4.5 hours of symptom start (the blood-starved (ischemic) stroke window) to save the most brain cells.

SAFETY WATCH

Blood pressure **MUST** be under 185/110 **BEFORE** giving the drug and stay under 180/105 **AFTER**, or bleeding risk shoots up. · Risk of brain bleeding (intracranial bleeding (hemorrhage)) in about 6% of patients—watch for sudden severe headache, vomiting, sudden confusion, or worsening weakness. · Cannot be given if the patient had recent surgery, active bleeding, or stroke within the past 3 months. · No antiplatelet drugs (aspirin, clopidogrel) or blood thinners for 24 hours after tPA unless no bleeding is seen on a brain scan.

Aspirin · Antiplatelet

Stops platelets (tiny blood cells that start clots) from sticking together, making clots less likely to form or grow in brain arteries.

1. Aspirin permanently turns off COX-1 (an enzyme that makes thromboxane A2, a chemical signal that tells platelets to clump).
2. Without thromboxane A2, platelets lose their main 'stick together' signal.
3. This keeps small clots from forming in narrowed or damaged brain arteries.
4. The effect lasts the platelet's whole life (about 7–10 days), so one dose affects platelets until new ones replace them.

SAFETY WATCH

Can cause stomach upset or stomach bleeding—take with food if needed. · Do **NOT** give within 24 hours of thrombolytic (tPA) because bleeding risk multiplies. · Watch for easy bruising, blood in stool (black or red), or nosebleeds that won't stop. · Allergy is rare but real—watch for hives or trouble breathing.

Clopidogrel (Plavix) or ticagrelor (Brilinta) · Antiplatelet (P2Y12 inhibitor)

Blocks a different platelet signal (the P2Y12 receptor) so platelets can't clump even when other 'stick' signals are present. Used alone or with aspirin for higher-risk minor strokes.

1. The drug binds to the P2Y12 receptor on each platelet.

2. When ADP (a signal released by injured vessel walls) tries to dock, the receptor is already occupied and can't send the 'clump now' message.
3. Platelets stay separate and flowing, lowering the chance of a new clot forming in a narrowed brain artery.
4. Clopidogrel's block lasts the platelet's lifetime; ticagrelor's block is reversible and wears off in 1–2 days if the drug is stopped.

SAFETY WATCH

Dual antiplatelet therapy (aspirin + clopidogrel or ticagrelor) is used for minor strokes or TIAs with high recurrence risk, usually for 21 to 90 days, then switched to single antiplatelet to lower bleeding risk. · Watch for unusual bruising, bleeding gums, blood in urine or stool. · Ticagrelor can cause shortness of breath in some people—usually mild and fades over time. · Do NOT use clopidogrel if the patient has a known poor metabolizer gene variant (CYP2C19) without pharmacy guidance—it may not activate properly.

Atorvastatin (Lipitor) or rosuvastatin (Crestor) · Statin (HMG-CoA reductase inhibitor)

Lowers cholesterol, especially LDL ('bad' cholesterol), and stabilizes the fatty plaques in arteries so they are less likely to crack open and trigger a clot.

1. The drug jams HMG-CoA reductase, the liver enzyme that runs the cholesterol assembly line.
2. With less cholesterol being made, the liver pulls LDL cholesterol out of the blood to recycle it.
3. Blood LDL levels drop, meaning less cholesterol is available to deposit in artery walls.
4. Existing plaques in brain and neck arteries become more stable and less inflamed, lowering the risk they will rupture and cause another stroke.

SAFETY WATCH

Most common side effect is muscle aches (myalgia)—tell the doctor if legs or shoulders hurt. · Rare risk of severe muscle breakdown (rhabdomyolysis)—stop the drug and get help immediately if urine turns dark brown or cola-colored. · Liver enzymes should be checked before starting and if symptoms (yellowing skin, severe fatigue, belly pain) appear. · Avoid grapefruit juice with atorvastatin—it raises drug levels and increases side-effect risk.

Warfarin (Coumadin) or apixaban (Eliquis), rivaroxaban (Xarelto), dabigatran (Pradaxa), edoxaban (Savaysa) · Anticoagulant (blood thinner)

Slows the blood's clotting cascade so clots are less likely to form in the heart (especially the left atrial appendage in atrial fibrillation (an irregular, quivering heartbeat)) and travel to the brain.

1. Warfarin stops the liver from recycling vitamin K, so several clotting proteins can't be activated.
2. DOACs directly jam a single step in the clotting cascade—either factor Xa (the enzyme that makes thrombin) or thrombin itself (the enzyme that weaves fibrin).
3. Either way, the blood takes much longer to clot, so clots are less likely to form in a quivering atrium (atrial fibrillation) or damaged heart valve.
4. This sharply lowers the risk that a clot will break off, travel through the carotid arteries, and block a brain vessel.

SAFETY WATCH

All anticoagulants raise bleeding risk—watch for blood in stool or urine, severe nosebleeds, vomiting blood, or sudden severe headache. · Warfarin requires regular INR blood tests (goal usually 2–3) and dose adjustments; diet (vitamin K in greens) and other drugs affect levels. · DOACs do NOT require routine blood tests but MUST be dose-adjusted for kidney function—too much if kidneys are weak leads to dangerous bleeding. · Do NOT stop suddenly without doctor guidance—stroke risk rebounds quickly. · Antidotes exist: vitamin K and plasma for warfarin; idarucizumab for dabigatran; andexanet alfa for factor Xa inhibitors (apixaban, rivaroxaban, edoxaban).

Antihypertensives (e.g. lisinopril, amlodipine, losartan, hydrochlorothiazide) · ACE inhibitor, calcium channel blocker, ARB, or thiazide diuretic

Lowers blood pressure to reduce the chronic strain on artery walls, slowing plaque buildup and lowering the chance of another stroke.

1. Each class works a different lever: less squeezing signal (ACE inhibitor, ARB), less muscle contraction (calcium blocker), or less fluid volume (diuretic).
2. Arteries stay wider, the heart pumps against less resistance, and peak pressure drops.
3. Lower pressure means less damage to the endothelium (inner lining of arteries), slower plaque formation, and lower risk of plaque rupture.
4. Target blood pressure after stroke is generally under 130/80 (per AHA/ASA 2021 guidelines), though individual goals vary.

SAFETY WATCH

ACE inhibitors can cause a dry cough (switch to ARB if bothersome) or, rarely, angioedema (severe face/throat swelling—stop immediately). · Calcium blockers may cause ankle swelling or constipation. · Diuretics increase urination and can drop potassium—watch for muscle cramps or weakness; check potassium levels. · Stand up slowly to avoid dizziness, especially when starting or increasing dose. · Do NOT stop abruptly—rebound high blood pressure can occur.

Red flags

- 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)

Nursing priorities

1. Establish exact last known well time and notify the stroke team immediately so the physician can evaluate for thrombolysis within 4.5 hours. — 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. — 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. — 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.
4. Keep the patient NPO (nothing by mouth) until a swallow screen or formal swallow evaluation confirms safe swallowing. — Acute stroke damages the brainstem or cortical areas controlling the pharynx (throat), causing trouble swallowing (dysphagia) (difficulty swallowing) in up to 50 percent of patients. Aspiration (food or liquid entering the lungs instead of the stomach) leads to aspiration pneumonia, the leading cause of death after stroke.
5. Perform hourly neurologic checks using the NIHSS or a facility-approved stroke scale, and report any worsening immediately. — Deterioration signals bleeding-driven (hemorrhagic) transformation, growing a patch of dead tissue (infarct), cerebral swelling (edema) (brain swelling), or reocclusion (the clot blocking the artery again). Early detection allows for repeat imaging and interventions like hyperosmolar therapy with mannitol (a diuretic that pulls fluid from swollen brain tissue) or repeat thrombectomy.
6. Check fingerstick blood glucose immediately and treat if under 60 mg/dL, then recheck every 4 hours for the first 24 hours. — Low blood sugar (Hypoglycemia) mimics stroke and must be ruled out before thrombolysis. High blood sugar (Hyperglycemia) (high blood sugar, over 180 mg/dL) worsens blood-starved (ischemic) injury by increasing lactate (an acid) production in oxygen-starved neurons, expanding a patch of dead tissue (infarct) size and worsening outcomes.
7. Establish two large-bore IV lines (18-gauge or larger) before thrombolysis, and avoid arterial punctures, central lines, Foley catheters, or NG tubes until at least 24 hours after thrombolysis. — Thrombolysis dissolves clots everywhere in the body for 24 hours. Invasive procedures create bleeding sites that cannot clot normally. Even a femoral artery stick for blood gas can cause retroperitoneal a pooled collection of blood (hematoma) (hidden bleeding in the abdomen behind the intestines) requiring transfusion or surgery.
8. Administer aspirin 325 mg orally or rectally 24 hours after thrombolysis, or immediately if the patient did not receive thrombolysis, unless contraindicated. — Aspirin irreversibly blocks platelet aggregation (clumping of clotting cells), reducing early recurrent stroke risk by 30 percent. Giving it sooner than 24 hours after thrombolysis increases bleeding risk. If thrombolysis was not given, aspirin becomes the primary acute antiplatelet therapy to limit clot propagation (growth).

Sources

- AHA/ASA 2019 Guidelines for the Early Management of Acute Ischemic Stroke
- European Stroke Organisation 2021 guidelines
- CDC Stroke Facts
- The Lancet Neurology 2020 acute stroke review

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