

Stroke Activation

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THE STORY IN ONE SENTENCE

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“A stroke alert starts the clock: find out when the person was last acting normal, score their stroke severity with the NIH Stroke Scale, get a CT scan right away to rule out bleeding, give clot-busting medicine (tPA or TNK) within 4.5 hours if it's safe, and pull out large clots with a wire retriever (thrombectomy) within 24 hours if a big vessel is blocked.”

Normal anatomy — the body's job

The brain floats in a pool of blood delivered by four main highways: two carotid arteries in the front and two vertebral arteries in the back, which join together at the base of the skull to form a safety ring called the Circle of Willis, then branch out into smaller arteries that deliver oxygen-rich blood to every neighborhood of brain tissue.

BODY'S JOB

Brain cells burn through oxygen and sugar nonstop to think, move, sense, and keep your body running, so they need a constant, heavy flow of fresh blood every second — about 15% of all the blood your heart pumps goes straight to the brain, even though the brain is only 2% of your body weight.

1. Blood leaves the heart through the aorta (the body's main pipeline).
2. The carotid arteries split off and climb up each side of the neck, carrying blood to the front and middle of the brain.
3. The vertebral arteries run up the back of the neck through holes in the spine bones, then join to form the basilar artery, feeding the back of the brain and brainstem.
4. At the base of the brain, these arteries link into the Circle of Willis — a backup loop that lets blood reroute if one pipe gets blocked.

5. Smaller arteries branch off and dive into brain tissue, splitting into tiny capillaries that sit right next to brain cells and hand off oxygen and glucose.
6. Veins collect the used blood and drain it back toward the heart, completing the loop.

What breaks

A stroke happens when one of the brain's arteries either gets clogged by a clot (ischemic stroke, about 87% of all strokes) or bursts and bleeds into the brain (hemorrhagic stroke, about 13%), cutting off blood flow so that brain cells downstream start dying within minutes.

CLINICAL TERM

Acute blood-starved (ischemic) stroke occurs when a blood clot (thrombus) (clot born right there in a brain artery, often on top of a cracked cholesterol plaque) or a traveling clot (embolus) (clot that traveled from the heart or a neck artery) blocks a cerebral artery, creating an blood-starved core (dead tissue) surrounded by a penumbra (stunned, salvageable tissue). Bleeding-driven (Hemorrhagic) stroke occurs when a vessel ruptures — either from high blood pressure blowing out a small deep artery (intracerebral bleeding (hemorrhage)) or from a brain aneurysm bursting (subarachnoid bleeding) — flooding the brain with blood that crushes and poisons nearby cells.

How the body compensates

The brain has almost no backup oxygen reserve, so once blood stops, the blood-starved (ischemic) core dies in minutes — but the penumbra (the stunned border zone) can hang on for hours if some trickle of blood arrives through collateral vessels (tiny side-street arteries that try to detour around the blockage), which is the narrow time window when clot-busting drugs or thrombectomy can still save tissue.

1. Blood pressure often shoots up automatically (the brain's attempt to force more flow past the blockage), so do NOT lower it aggressively in acute blood-starved (ischemic) stroke unless it is dangerously high ($>185/110$ if giving tPA, $>220/120$ otherwise) — the high pressure is helping perfuse the penumbra.
2. Collateral vessels (small connecting arteries) open up and try to reroute blood around the blockage, buying time for the penumbra — patients with good collaterals on imaging have better outcomes and longer treatment windows.
3. The body cannot dissolve a large clot on its own in time to save brain tissue, which is why we give tPA (tissue plasminogen activator) or TNK (tenecteplase) — enzymes that chew up the clot's fibrin mesh — or physically pull the clot out with a wire retriever during thrombectomy.

4. After the first few hours, the blood-brain barrier (the tight seal around brain capillaries) breaks down in the dying tissue, so if you give tPA too late or the clot breaks apart on its own, blood can leak into the dead zone and cause bleeding-driven (hemorrhagic) transformation (bleeding into the stroke), which is why the 4.5-hour tPA window exists.
5. In bleeding-driven (hemorrhagic) stroke, the body has no good compensation — the bleeding raises intracranial pressure, squeezes the brain, and can cause herniation (brain tissue sliding into the wrong compartment and crushing the brainstem), so the priority is to stop the bleeding, reverse blood thinners, and sometimes surgically drain the clot.
6. Watch for neurological worsening (expanding stroke, swelling, rebleeding, or seizure), dropping level of consciousness, or new headache — these mean the compensation is failing and the brain is running out of room or oxygen.
7. A patient who looks stable but has a large-vessel blockage (occlusion) on imaging is NOT stable — the penumbra is dying minute by minute, and the clock is ticking toward permanent disability.

Domino effect

1. A clot blocks a brain artery (or a vessel bursts), cutting off blood flow to downstream tissue. — Brain cells need oxygen delivered by blood every second to make energy (ATP) and stay alive — when the blood stops, the cells run out of fuel in about 3–5 minutes and start to die.
2. The blood-starved (ischemic) core (center of the blocked zone) dies fast, but the penumbra (border zone getting a trickle of blood from side streets) stays stunned and salvageable for a few hours. — Cells in the penumbra are hurt and not working, but they haven't died yet — if you restore blood flow quickly (with tPA or thrombectomy), those cells wake back up and function returns.
3. Sudden one-sided weakness, face droop, slurred speech, vision loss, dizziness, or trouble walking appear within seconds to minutes. — Each part of the brain controls specific jobs (the motor strip moves your muscles, Broca's area makes speech, the visual cortex processes sight, the cerebellum runs balance), so whichever artery is blocked determines which function fails — it's a map, not random.
4. The blood-brain barrier starts breaking down after a few hours, and swelling (cerebral swelling (edema)) builds up in the dead tissue, raising pressure inside the skull. — Dead cells leak fluid and inflammatory chemicals, the tissue swells like a bruise, and in large strokes (especially cerebellar or malignant MCA strokes), the brain can run out of room in the skull and herniate, crushing the brainstem and causing coma or death.

5. If tPA is given too late, or if the clot breaks apart on its own after the blood-brain barrier is leaky, blood spills into the stroke zone (hemorrhagic transformation), turning an blood-starved (ischemic) stroke into a bleed. — The broken barrier can't hold back blood anymore, so restoring flow after the tissue is already dead just floods the area — this is why the tPA window is strict (4.5 hours) and why we do a CT scan first to make sure there's no bleeding already.
6. In bleeding-driven (hemorrhagic) stroke, blood pools in or around the brain, acting like a growing mass that squeezes brain tissue, raises intracranial pressure, and can cause herniation or rebleeding. — Blood outside a vessel is toxic to brain cells (it releases iron and inflammatory signals), and the physical pressure crushes tissue and blood vessels, cutting off flow to areas that weren't even hit by the original bleed.
7. Without fast treatment, dead brain tissue is permanent — the neurons don't grow back, so the person is left with lasting weakness, speech trouble, vision loss, or paralysis matching the artery that was blocked. — Brain cells are specialized and can't regenerate like skin or liver — once they die, that function is lost forever, unless nearby healthy tissue can slowly learn to take over some of the job through rehab and neuroplasticity.
8. Restoring blood flow early (with tPA, TNK, or thrombectomy) saves the penumbra, reverses symptoms, and dramatically improves outcomes — 'time is brain,' so every minute of delay costs about 1.9 million brain cells. — If you break up or pull out the clot while the penumbra is still alive, oxygen and glucose flood back in, the stunned cells recover, and the person can walk out of the hospital with little or no disability — but the window is short, so stroke is always a medical emergency.

Symptoms · what the patient feels, and why

- Sudden weakness or numbness on one side of the face, arm, or leg: A clot or bleed in the brain blocks blood flow to the strip of brain tissue that controls movement and feeling on the opposite side of the body. Without oxygen, those brain cells stop working within minutes, so the muscles they control go weak or numb right away.
- Sudden trouble speaking or understanding words (slurred speech or confused answers): The left side of the brain (in most people) holds the language centers—Broca's area (speech production) and Wernicke's area (understanding words). When a stroke cuts off their blood supply, the person cannot shape words clearly or make sense of what others are saying.
- Sudden vision loss in one or both eyes, or double vision: The back of the brain (occipital cortex) processes what the eyes see, and the brainstem controls eye movements. A stroke there scrambles the signals, so vision goes dark in part of the field, or the eyes cannot line up and the person sees two of everything.

- Sudden severe headache (often called the worst headache of my life): When a blood vessel in the brain tears open (hemorrhagic stroke), blood spills into the space around the brain and irritates the meninges (the protective covering), which have many pain nerves. The sudden pressure spike inside the skull triggers intense pain.
- Sudden trouble walking, loss of balance, or dizziness: The cerebellum (the brain's balance center at the back) and parts of the brainstem coordinate posture and smooth movement. When stroke damages these areas, the person cannot steady themselves, feels the room spinning, or veers to one side when trying to walk.
- Sudden confusion or trouble understanding what is happening around them: Large strokes—especially in the frontal or temporal lobes—disrupt the networks that keep a person alert and aware. Without enough oxygen, these brain cells misfire, so thoughts become jumbled and the person cannot follow conversations or know where they are.
- High or low blood pressure reading at the time of symptom onset: High blood pressure can mean the brain is trying to push blood past a blockage (the body's emergency response), or it may be the cause of a burst vessel. Very low pressure can mean the heart is not pumping hard enough to deliver blood to the brain. Either way, checking the number helps doctors decide how to treat the stroke safely.
- High or low blood sugar reading: Very low blood sugar (hypoglycemia) starves brain cells of fuel and can mimic stroke symptoms (weakness, confusion, slurred speech). Very high sugar (hyperglycemia) can also worsen brain injury during a real stroke. Checking glucose right away rules out a sugar problem that is fixable with a snack or insulin instead of clot-busting drugs.

Focused exam

- Ask exactly what time they were last acting normal — this is the single most important number in stroke care: The clock starts ticking from the last moment the person was completely fine, not when someone noticed something wrong. This number decides whether we can give clot-busting medicine. Clot medicine only works if given within 4.5 hours of symptom start — after that, the dead brain tissue releases chemicals that make bleeding more likely than recovery.
- Check the National Institutes of Health Stroke Scale (NIHSS) — test face movement, arm strength, speech clarity, and ability to understand: This 15-point scale measures how much brain is not working. A score of 0 means no stroke signs. A score of 1–4 is mild (small area blocked). A score above 15 means a large artery is blocked and the person may need the catheter procedure to pull the clot out. The number helps predict what will happen and picks the right treatment.

- Test for one-sided weakness — ask the person to hold both arms straight out in front of them or squeeze both your hands at the same time: Weakness on one side of the body is the clearest sign of stroke because each half of the brain controls the opposite side of the body. If a blood clot blocks flow to the right side of the brain, the left arm and leg cannot get movement signals. The muscles are fine — the problem is the control wire is cut.
- Check their ability to speak and understand — can they repeat a sentence? Do they understand simple commands?: Slurred speech or trouble finding words tells us the stroke hit Broca's area or Wernicke's area, the two language centers in the left side of the brain (in most people). If the person cannot speak but can move their mouth and tongue, the damage is in the brain's language wiring, not the mouth itself.
- Check blood pressure and finger-stick blood sugar immediately: Blood sugar below 60 mg/dL causes confusion, slurred speech, and weakness that look exactly like stroke but reverse in minutes with glucose. Blood pressure above 185/110 mmHg makes brain bleeding more likely if we give clot medicine. These two quick checks catch stroke mimics and prevent treatment disasters.
- Look at their eyes — do both eyes point to one side? Can they see objects in all four corners of their vision?: Eyes that turn toward the side of the stroke (both looking right if the right brain is damaged) mean a large clot in the middle cerebral artery. The frontal eye field, the brain area that aims the eyes, lost its blood supply. Loss of half the vision (hemianopia) means the stroke hit the back of the brain, where the visual cortex lives.

Diagnostics · every test answers a question

- Non-contrast CT of the head → Is this stroke caused by bleeding in the brain (hemorrhagic) or a clot blocking an artery (ischemic)?
- CT angiography (CTA) of the head and neck → Is there a large clot in a major brain artery that we can pull out with a catheter?
- Finger-stick blood glucose → Is low blood sugar causing stroke-like symptoms instead of a real stroke?
- Electrocardiogram (ECG) → Is the heart in atrial fibrillation (an irregular, quivering heartbeat), which forms clots that travel to the brain?
- Complete blood count (CBC) and platelet count → Does the person have enough platelets and red blood cells to tolerate clot medicine or blood thinners?
- PT/INR (prothrombin time / international normalized ratio) → Is the person already on a blood thinner that blocks clotting, making tPA unsafe?
- Troponin and serial ECGs if chest pain or heart history → Is the stroke happening because the heart just had a heart attack or threw a clot?

Treatment logic

- Activate the stroke team and document exact time last known well → Knowing the precise minute the person was last acting normal unlocks the treatment window. IV thrombolytics (tPA or tenecteplase) work best within 3 hours and can be given up to 4.5 hours. Mechanical thrombectomy works up to 6 hours for most strokes and up to 24 hours if imaging shows salvageable brain tissue. Without this time, we cannot safely give either treatment.
- Non-contrast CT of the head immediately (door-to-CT goal under 20 minutes) → This scan separates bleeding-driven (hemorrhagic) stroke (bleeding) from blood-starved (ischemic) stroke (clot). If we see blood, clot-busting medicine is absolutely forbidden because it will turn a small bleed into a deadly one. If we see no blood, we can move forward with tPA or thrombectomy. This one image decides the entire treatment path.
- Check and correct blood glucose if below 60 mg/dL → Giving IV dextrose or oral glucose raises blood sugar within minutes and reverses stroke-like symptoms if low blood sugar (hypoglycemia) was the real problem. This avoids giving dangerous clot medicine to someone who never had a stroke. If symptoms persist after glucose correction, we know the problem is a true clot.
- Give IV alteplase (tPA) 0.9 mg/kg (max 90 mg) or IV tenecteplase (TNK) 0.25 mg/kg within 4.5 hours of symptom onset → Thrombolytics break apart the fibrin mesh holding the clot together, dissolving it and restoring blood flow to the downstream brain tissue. The sooner the clot dissolves, the more brain cells survive. For every 15-minute delay, the chance of good recovery drops measurably. This medicine improves odds of independent living by about 30% if given early.
- Mechanical thrombectomy (catheter-based clot retrieval) within 6 hours for large-vessel blockage (occlusion), up to 24 hours if imaging shows salvageable tissue → A neurointerventionalist threads a catheter up through the groin artery into the blocked brain artery, then pulls the clot out with a stent retriever or suction device. This restores blood flow in minutes and works even for clots too large for medicine to dissolve. Thrombectomy improves the chance of walking and living independently by about 50% in large-vessel strokes.
- Control blood pressure carefully — keep below 185/110 mmHg if giving tPA, below 220/120 mmHg if not → High blood pressure during acute stroke pushes blood through weak vessel walls and increases the risk of brain bleeding, especially after tPA. Lowering pressure gently with IV labetalol or nicardipine protects fragile tissue. Too-low pressure starves the penumbra (the struggling tissue around the dead core), so we aim for controlled reduction, not aggressive drops.

- Start aspirin 325 mg within 24 to 48 hours (but NOT within 24 hours of tPA) → Aspirin blocks platelets from clumping together, reducing the chance of a new clot forming in the same spot or elsewhere. Starting aspirin early (after the tPA bleed-risk window) lowers the risk of another stroke in the next two weeks by about 20%. This is secondary prevention — stopping the second hit.

Medications

Alteplase (tPA, tissue plasminogen activator) · Thrombolytic (clot-buster)

Alteplase is a protein that acts like molecular scissors. It cuts apart fibrin (the sticky net that holds a blood clot together), turning a solid clot into pieces small enough for the blood to wash away. This reopens the blocked artery in the brain so oxygen-rich blood can flow again and rescue dying brain cells. It works best when given within 4.5 hours of symptom start—after that window, the brain tissue is often already dead and bleeding risk outweighs benefit.

1. Alteplase is injected into a vein and travels through the bloodstream to the brain.
2. It binds to fibrin strands in the clot (the sticky net holding the clot together).
3. It activates plasminogen (a natural protein in blood) and converts it into plasmin (an enzyme that cuts fibrin).
4. Plasmin chops the fibrin net into fragments, breaking the clot apart.
5. Blood flow resumes through the artery, delivering oxygen to starved brain tissue and stopping further cell death.

SAFETY WATCH

Bleeding anywhere in the body (most dangerous: bleeding in the brain). Watch for sudden severe headache, confusion, vomiting, or neurologic worsening. · Do NOT give if blood pressure is above 185/110 mmHg (must lower it first). · Do NOT give if CT scan shows bleeding, very large stroke, or signs the stroke started more than 4.5 hours ago. · Do NOT give if the patient had recent surgery, head trauma, or active internal bleeding. · Monitor neuro checks every 15 minutes during infusion and for 2 hours after, then hourly for 24 hours.

Tenecteplase (TNK-tPA) · Thrombolytic (clot-buster)

Tenecteplase works the same way as alteplase—it is a modified version of the same clot-busting protein. It cuts fibrin (the net holding the clot) and dissolves the blockage in the brain artery. The difference is that TNK is easier to give: one quick push into the vein instead of a long drip. Dose is weight-based (0.25 mg per kilogram, maximum 25 mg) and it can be given in the

ambulance. Current evidence shows it works as well as alteplase for most strokes and may be safer with large-vessel occlusions treated with thrombectomy.

1. TNK is pushed as a single bolus into a vein and circulates to the brain.
2. It locks onto fibrin in the clot.
3. It converts plasminogen (a blood protein) into plasmin (the cutting enzyme).
4. Plasmin breaks down the fibrin mesh, dissolving the clot.
5. Blood flow restarts, oxygen reaches brain cells, and the stroke stops growing.

SAFETY WATCH

Bleeding (especially brain bleeding (hemorrhage)). Watch for new headache, sudden worsening, or drop in consciousness. · Blood pressure must be below 185/110 mmHg before giving TNK. · Do NOT give if CT shows bleeding, very large a patch of dead tissue (infarct), or time of stroke onset is unknown or beyond 4.5 hours. · Contraindicated if recent surgery, trauma, or bleeding disorder. · Monitor neuro exam closely for the first 24 hours.

Aspirin · Antiplatelet

Aspirin blocks an enzyme inside platelets (the sticky cell fragments in blood) called cyclooxygenase-1 (COX-1). Without COX-1, platelets cannot make thromboxane A2 (a chemical signal that tells platelets to clump together). This makes the blood less sticky and reduces the chance of new clots forming. In stroke, aspirin is started 24 hours after tPA (or right away if tPA was not given) to prevent another stroke in the next few days and weeks.

1. Aspirin is absorbed in the stomach and small intestine and enters the blood.
2. It permanently blocks COX-1 inside each platelet.
3. Platelets can no longer make thromboxane A2 (the 'stick together' signal).
4. Platelets become less sticky and less likely to pile onto each other and form a clot.
5. The risk of a second stroke (or heart attack) drops significantly over the next 3 months.

SAFETY WATCH

Stomach or intestinal bleeding (black tarry stools, vomiting blood, severe belly pain). · Do NOT give within 24 hours of tPA or TNK (increases bleeding risk). · Use with caution if history of ulcers or bleeding. · Watch for allergic reaction (rash, wheezing—rare but serious).

Clopidogrel (Plavix) · Antiplatelet (P2Y12 inhibitor)

Clopidogrel blocks a receptor on platelets called P2Y12. This receptor normally responds to ADP (adenosine diphosphate, a chemical released when blood vessels are injured) by making platelets stick to each other. By blocking P2Y12, clopidogrel prevents platelets from receiving that 'clump now' signal. It is often used with aspirin for 21 days after a minor stroke or high-risk TIA (dual antiplatelet therapy, or DAPT) to prevent early recurrent stroke, then continued alone for long-term prevention.

1. Clopidogrel is swallowed and absorbed in the gut.
2. The liver converts it into its active form.
3. The active drug permanently blocks P2Y12 receptors on platelets.
4. Platelets cannot respond to ADP and do not stick together as easily.
5. The chance of forming a new clot in the brain or heart arteries falls.

SAFETY WATCH

Bleeding (bruising, nosebleeds, blood in urine or stool). · Very rare: TTP (thrombotic thrombocytopenic purple bleeding blotches (purpura))—watch for fever, confusion, and purple skin spots; this is a medical emergency. · Do NOT combine long-term with aspirin unless specifically indicated (such as the first 21 days after minor stroke). · Check for drug interactions—proton pump inhibitors (like omeprazole) can reduce clopidogrel effectiveness.

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

Statins block an enzyme in the liver called HMG-CoA reductase, which is the main factory for making cholesterol. When cholesterol production drops, the liver pulls LDL cholesterol (the 'bad' cholesterol) out of the blood to use for making cell membranes and hormones. This lowers LDL in the bloodstream, shrinks cholesterol plaques in artery walls, and stabilizes plaques so they are less likely to rupture and cause another stroke. Statins also reduce inflammation in blood vessel walls.

1. The statin is absorbed in the gut and travels to the liver.
2. It blocks HMG-CoA reductase, the enzyme that builds cholesterol from smaller molecules.
3. Liver cells run low on cholesterol and respond by making more LDL receptors on their surface.
4. These receptors grab LDL cholesterol out of the blood and pull it into liver cells.
5. Blood LDL levels drop, existing plaques shrink and stabilize, and the risk of a second stroke falls by about 20–25%.

SAFETY WATCH

Muscle pain or weakness (common and usually mild; very rare serious form is rhabdomyolysis with dark urine and kidney damage). · Liver enzyme elevation (check ALT at baseline and if symptoms appear). · Slightly increased risk of new-onset diabetes (benefit outweighs risk in stroke patients). · Drug interactions (especially with gemfibrozil, certain antifungals, and grapefruit juice in large amounts).

Apixaban, Rivaroxaban, Edoxaban, or Dabigatran (if atrial fibrillation (an irregular, quivering heartbeat) is found) · Direct oral anticoagulant (DOAC)

These drugs block specific clotting factors in the blood. Apixaban, rivaroxaban, and edoxaban directly inhibit factor Xa (the enzyme that converts prothrombin into thrombin, the main clot-builder). Dabigatran directly blocks thrombin itself. By stopping these steps in the clotting cascade, DOACs prevent the blood from forming large, organized clots—especially in the left atrium of the heart during atrial fibrillation (an irregular, quivering heartbeat). This stops clots from traveling to the brain and causing embolic stroke. DOACs are first-line for stroke prevention in atrial fibrillation.

1. The DOAC is absorbed from the gut into the blood.
2. It binds to and blocks factor Xa or thrombin (depending on the drug).
3. The clotting cascade stops before fibrin can form.
4. Blood remains fluid and large clots cannot organize in the heart.
5. The risk of clot a traveling clot lodging in a vessel (embolism) to the brain drops by about 65% compared to no treatment.

SAFETY WATCH

Bleeding (any site—brain, gut, urinary tract). Watch for blood in stool, urine, severe headache, or unexplained bruising. · Do NOT give if active bleeding or very high bleeding risk (recent brain bleed, severe liver disease). · Adjust dose for kidney function (especially dabigatran and edoxaban). · Reversal agents available: idarucizumab for dabigatran, andexanet alfa for factor Xa inhibitors (expensive and not always stocked). · Do NOT combine with other anticoagulants or long-term dual antiplatelet therapy without specialist guidance.

Labetalol or Nicardipine IV (for acute blood pressure control) · Antihypertensive (labetalol: alpha + beta blocker; nicardipine: calcium channel blocker)

In acute blood-starved (ischemic) stroke, very high blood pressure (above 220/120 mmHg, or above 185/110 if tPA is planned) can increase the risk of bleeding into the brain. Labetalol

blocks beta receptors (which slow the heart and reduce how hard it pumps) and alpha receptors (which relax blood vessel walls). Nicardipine blocks calcium channels in smooth muscle cells of artery walls, causing the vessels to relax and widen. Both drugs lower blood pressure in a controlled, gradual way. After the acute phase, blood pressure is allowed to run higher for a few days (permissive high blood pressure (hypertension)) to support blood flow to the injured brain, then slowly brought down over weeks.

1. The drug is given by IV infusion and acts within minutes.
2. Labetalol slows the heart rate and reduces the force of each heartbeat, and relaxes arteries. Nicardipine blocks calcium entry into artery muscle cells, preventing contraction.
3. Blood vessels widen (dilate) and blood pressure falls gradually.
4. The target is below 185/110 for tPA eligibility, or below 220/120 to reduce bleeding (hemorrhage) risk if no tPA is given.
5. Once blood pressure is controlled, the infusion is tapered and oral agents are started for long-term management.

SAFETY WATCH

Drop in blood pressure that is too fast or too low—this can worsen brain starved blood flow (ischemia) (the brain needs adequate blood flow (perfusion) pressure after a stroke). · Labetalol: avoid in severe asthma or heart block; can mask signs of low blood sugar in diabetics. · Nicardipine: monitor for reflex fast heart rate; use cautiously in heart failure. · Frequent blood pressure monitoring (every 5–15 minutes) during IV infusion.

Red flags

- Symptoms started suddenly—within seconds or minutes—rather than building slowly over hours or days.
- The person cannot lift both arms to the same height, one side of the face droops when they smile, or their speech is slurred or makes no sense (positive FAST screen).
- Worst headache of my life, especially with stiff neck, vomiting, or sudden collapse—suggests bleeding in the brain (hemorrhagic stroke) rather than a clot.
- Symptoms are still present when emergency help arrives—do NOT wait to see if they go away, because every minute of lost blood flow kills about two million brain cells.
- History of atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat), recent heart attack, or use of blood thinners—raises the chance of a stroke caused by a clot traveling from the heart or a dangerous bleed.

Nursing priorities

1. Activate the stroke team immediately and document the exact time the patient was last known well (last seen normal). — Brain cells die at a rate of 1.9 million per minute during stroke. The last known well time determines if the patient qualifies for tPA (within 4.5 hours) or thrombectomy (within 6 to 24 hours). Time is brain.
2. Keep the patient NPO (nothing by mouth) until a swallow screen is completed—no food, liquid, or pills by mouth. — Stroke can damage the swallowing muscles and nerves, causing trouble swallowing (dysphagia) (difficulty swallowing). Giving food or liquid before a swallow test can cause aspiration (food or liquid entering the lungs), leading to pneumonia or choking.
3. Check blood glucose immediately with a fingerstick and give IV dextrose if glucose is below 60 mg/dL. — Low blood sugar (Hypoglycemia) (low blood sugar) mimics stroke symptoms like confusion, weakness, and slurred speech. Correcting low glucose can reverse symptoms immediately and prevents unnecessary tPA administration. Low glucose also worsens brain injury during stroke.
4. Establish large-bore IV access (18-gauge or larger) in both arms if possible and send stat labs including PT/INR, aPTT, CBC, BMP, and troponin. — Two IVs allow simultaneous tPA infusion and other medications. PT/INR (clotting time) and aPTT (clotting pathway time) must be checked before tPA to detect bleeding risk. Troponin (heart enzyme) checks for heart attack, which can cause or happen with stroke.
5. Perform and document a baseline NIH Stroke Scale (NIHSS) score and perform the FAST screen (Face droop, Arm weakness, Speech difficulty, Time to call 911). — The NIHSS (a 0-to-42-point scale) measures stroke severity and helps determine if the patient needs thrombectomy. A baseline score allows the team to detect improvement or worsening. The FAST screen quickly identifies stroke and documents deficits.
6. Monitor blood pressure every 15 minutes but do NOT lower it unless systolic is above 220 mmHg or diastolic is above 120 mmHg before tPA. — The brain needs high blood pressure during acute stroke to push blood through narrowed or blocked vessels (called autoregulation). Lowering BP too much can worsen brain injury. After tPA, BP must stay below 180/105 mmHg to prevent bleeding in the brain.
7. Position the patient flat or with head of bed elevated no more than 30 degrees, and keep the patient on continuous cardiac and pulse oximetry monitoring. — Lying flat increases blood flow to the blood-starved (ischemic) (starved) brain tissue by using gravity. Elevating the head too high can worsen brain starved blood flow (ischemia). Cardiac monitoring detects atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat) or arrhythmias that can cause or complicate stroke. Oxygen saturation should stay above 94 percent.

- 8.** Prepare for immediate non-contrast CT scan of the head within 20 minutes of arrival (door-to-CT goal) and stay with the patient during transport. — Non-contrast CT (scan without dye) is the fastest way to rule out bleeding-driven (hemorrhagic) stroke (bleeding). We cannot give tPA if there is bleeding. CT also detects large clots and helps decide if thrombectomy is needed. Patients can deteriorate quickly during transport.

Sources

- AHA/ASA Guideline for the Early Management of Patients with Acute Ischemic Stroke (2019, updated 2023)
- AHA/ASA Guideline for the Management of Spontaneous Intracerebral Hemorrhage (2022)
- National Institute of Neurological Disorders and Stroke (NINDS)
- American Academy of Neurology stroke practice guidelines

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