

Tricyclic Antidepressant (TCA) Overdose

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THE STORY IN ONE SENTENCE

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“Tricyclic antidepressants jam sodium channels in the heart, block acetylcholine receptors all over the body, block alpha receptors on blood vessels, and turn down GABA in the brain. This creates a four-way crisis: the heart's electrical system slows and gets chaotic, blood pressure drops, the body swings into a hot-dry-mad pattern, and the brain loses its brake pedal and seizes. Sodium bicarbonate is the first-line medicine that helps restart the heart's sodium channels.”

Normal anatomy — the body's job

The heart beats because sodium channels in heart muscle cells open and close in a precise rhythm, letting electrical signals zip from cell to cell so the whole heart squeezes in the right order. Acetylcholine (the body's main 'rest and digest' messenger) keeps pupils small, the mouth and skin moist, the bladder emptying, the gut moving, and the heart rate calm. Alpha-1 receptors on blood vessel walls respond to norepinephrine (the body's 'squeeze' signal) and keep vessels tight enough to hold blood pressure steady. GABA (gamma-aminobutyric acid, the brain's main calming neurotransmitter) acts like a brake pedal on nerve cells, keeping them from firing too fast and preventing seizures. All four systems run at the same time, each doing its own job to keep the body steady.

BODY'S JOB

Sodium channels control the heart's electrical rhythm. Acetylcholine keeps the 'rest and digest' jobs running smoothly—small pupils, wet mouth, normal gut and bladder function, steady thinking. Alpha-1 receptors keep blood vessels tight enough to hold blood pressure up. GABA keeps the brain's nerve cells calm and prevents runaway electrical storms (seizures). If you don't know these four normal jobs first, the weird findings in tricyclic overdose seem like a random mess instead of a predictable pattern.

1. Sodium channels in the heart open and close in rhythm → electrical signal zips from cell to cell → the whole heart squeezes in the right order → blood pumps out smoothly.
2. Acetylcholine lands on receptors all over the body → pupils stay small, mouth stays wet, bladder empties, gut moves, heart rate stays calm → the 'rest and digest' system works.
3. Norepinephrine hits alpha-1 receptors on blood vessel walls → vessels squeeze just tight enough → blood pressure stays steady.
4. GABA lands on receptors in the brain → nerve cells slow their firing → the brain stays calm and seizures don't happen.

What breaks

Too much tricyclic antidepressant blocks all four systems at once: it jams the sodium channels in the heart (so the electrical signal slows and the rhythm gets wide and chaotic), shuts off acetylcholine receptors everywhere (so the body swings into a hot-dry-mad anticholinergic pattern), blocks alpha-1 receptors on blood vessels (so vessels relax and blood pressure drops), and turns down GABA in the brain (so the brake pedal lifts and seizures can start). One drug, four breaks, and every weird finding flows from these upstream blockades.

CLINICAL TERM

Tricyclic antidepressant toxicity causes four simultaneous receptor blockades: (1) fast sodium channel blockade in the myocardium slows phase 0 depolarization, widens the QRS (especially >100 ms), prolongs the QT interval, and can trigger ventricular a fast heart rate (tachycardia), ventricular fibrillation, or torsades de pointes; (2) muscarinic acetylcholine receptor antagonism produces the classic anticholinergic toxidrome—dilated pupils (mydriasis) (big pupils), dry mucous membranes, hyperthermia, urinary retention, decreased bowel sounds, altered mental status, and a fast heart rate; (3) peripheral alpha-1 adrenergic receptor blockade causes vasodilation and low blood pressure (hypotension); (4) GABA-A receptor antagonism in the CNS lowers the seizure threshold and can cause generalized tonic-clonic seizures. The downstream clinical picture you see at the bedside—QRS >100 ms, tall R wave in aVR (>3 mm), low blood pressure, anticholinergic signs, and seizures—all point backward to these four upstream receptor blocks.

How the body compensates

The body tries to keep blood pressure, heart rhythm, and brain function steady for a while by squeezing vessels harder, pumping adrenaline, and tolerating mild confusion. Vital signs can look almost normal during this compensation phase, which is the most dangerous window because it hides how close the patient is to tipping into ventricular arrhythmias, refractory shock, or seizure-driven cardiac arrest. The job is to spot the trajectory—widening QRS, falling blood pressure, rising confusion, or first seizure—before the crash point hits, because once the heart flips into v-tach or v-fib or seizures drive pH down, the window to reverse it closes fast.

1. The body releases norepinephrine and adrenaline to squeeze any vessels that still respond, trying to hold blood pressure up even though alpha-1 receptors are blocked.
2. The heart tries to keep pumping, but the sodium channel blockade slows conduction more and more—QRS starts around 100 ms and can climb past 120 or 160 ms as the drug level peaks.
3. The brain tolerates mild anticholinergic confusion and agitation for a while, but as more acetylcholine receptors shut off, delirium deepens and body temperature climbs.
4. This compensation phase is the dangerous middle: the patient may look stable or just 'a little off,' so the team can miss that the QRS is creeping wider or blood pressure is drifting lower.
5. The crash point to watch for: QRS >100 ms (especially >120 ms) signals high risk of ventricular a fast heart rate (tachycardia) or ventricular fibrillation. A tall R wave in aVR >3 mm is another red flag for severe sodium channel blockade.

6. Low blood pressure (Hypotension) that doesn't respond to IV fluids alone means alpha blockade is winning and vasopressors will be needed.
7. Any seizure is a crisis because it drops blood pH (from lactic acidosis (acid from oxygen-starved tissues)), which makes the sodium channel blockade even worse and can tip the heart into arrest.
8. Watch the trajectory, not just one snapshot: a patient with QRS 110 ms who was 100 ms an hour ago is on a bad slope, even if vitals look 'stable' right now.
9. Once ventricular arrhythmias start or seizures drop pH and trigger pulseless arrest, the window to reverse the toxicity shrinks fast—sodium bicarbonate and advanced airway/pressors must move in immediately.

Domino effect

1. QRS widens past 100 milliseconds on the ECG. — Tricyclic antidepressants block fast sodium channels in the heart muscle, so the electrical signal that normally zips from cell to cell slows way down. The QRS complex—the spike on the monitor that shows the ventricles firing—gets wider and slower. A QRS over 100 ms, and especially over 120 ms, means the sodium channels are seriously jammed and the heart is at high risk for dangerous rhythms like ventricular a fast heart rate (tachycardia) or ventricular fibrillation.
2. A tall R wave appears in lead aVR (>3 mm). — When tricyclics block sodium channels, the electrical current in the heart's right ventricle can't travel the normal path, so it fires backward in a weird direction. Lead aVR—a monitor lead that normally shows a tiny downward blip—suddenly shows a tall upward spike (R wave). A tall R in aVR is a red flag that the heart's conduction system is severely poisoned and life-threatening arrhythmias are coming.
3. Blood pressure drops and doesn't respond well to IV fluids. — Tricyclics block alpha-1 receptors on the walls of blood vessels. Normally norepinephrine hits those receptors and keeps vessels squeezed tight enough to hold blood pressure steady. When the receptors are blocked, the vessels relax, blood pools in the legs and gut, and blood pressure falls. Fluids help a little, but they can't squeeze the vessels back—vasopressors (medicines that bypass the blocked receptors) are often needed.
4. Pupils blow wide open, mouth goes bone-dry, skin gets hot and flushed, and the patient can't urinate. — Tricyclics shut off muscarinic acetylcholine receptors all over the body. Acetylcholine normally keeps pupils small, keeps the mouth and skin moist, keeps the gut moving, and keeps the bladder emptying. When those receptors are blocked, the opposite happens: pupils dilate wide (because the muscle that squeezes them shut can't work), saliva stops flowing (dry mouth), sweat glands stop (hot dry skin), the bladder muscle goes limp (urinary retention), and gut sounds disappear. This is the classic anticholinergic toxidrome —'hot as a hare, dry as a bone, red as a beet, mad as a hatter, blind as a bat.'

5. The patient gets confused, agitated, or delirious. — Acetylcholine receptors in the brain help keep thinking clear and organized. When tricyclics block those receptors, the brain's normal 'rest and digest' brake comes off, and the patient swings into confusion, picking at the air, talking nonsense, or getting combative. This is part of the anticholinergic storm happening in the brain.
6. Seizures explode—usually generalized tonic-clonic (the whole body shaking). — Tricyclics turn down GABA-A receptors in the brain. GABA is the brain's main calming neurotransmitter—it acts like a brake pedal to keep nerve cells from firing too fast. When GABA gets turned down, the brake pedal lifts, nerve cells fire out of control, and a seizure starts. Seizures are especially dangerous in tricyclic overdose because they create lactic acid, which drops blood pH. A lower pH makes the sodium channel blockade in the heart even worse, which can tip the patient into cardiac arrest.
7. Ventricular a fast heart rate (tachycardia), ventricular fibrillation, or torsades de pointes starts. — Once the QRS is wide and the QT is long from sodium channel blockade, the heart's electrical system is unstable. Any extra trigger—a seizure, low potassium, low magnesium, or just the drug level peaking—can flip the rhythm into fast chaotic beats (v-tach, v-fib, or the twisting pattern called torsades). These rhythms don't pump blood, so the patient loses pulses and crashes into cardiac arrest if the team doesn't act immediately.
8. Sodium bicarbonate is pushed IV, and the QRS starts to narrow. — Sodium bicarbonate raises blood pH (makes it more alkaline) and floods the bloodstream with extra sodium. The higher pH and extra sodium help unblock the jammed sodium channels in the heart, so the electrical signal can travel faster again. The QRS narrows back toward normal, the risk of deadly arrhythmias drops, and the heart stabilizes. Sodium bicarbonate is the key first-line antidote in severe tricyclic overdose—it's given as soon as QRS is over 100 ms or the patient has low blood pressure (hypotension), arrhythmias, or seizures.

Symptoms · what the patient feels, and why

- You find a patient who is drowsy
- QRS is wide
- Blood pressure is low
- Anticholinergic poisoning signs are there
- The electrocardiogram shows QRS over 100 or tall R in aVR
- This means arrhythmia risk

Focused exam

- QRS interval wider than 100 milliseconds on the electrocardiogram: Tricyclic drugs jam the sodium gates in heart muscle cells so the electrical wave crawls instead of races. A QRS wider than 100 milliseconds (normally under 100) is the clearest warning that the heart's wiring is poisoned and dangerous rhythms can start any second.
- Tall R wave in lead aVR on the electrocardiogram: This tall spike in lead aVR is like a fingerprint left by tricyclic drugs—no other overdose leaves exactly this mark. When you see it, tricyclic overdose jumps to the top of your list.
- Blood pressure below 90 systolic: Tricyclics block alpha receptors (the clamps on blood vessel walls), so the vessels relax too much and blood pressure drops. Low pressure means organs—including the heart and brain—are not getting enough oxygen.
- Large pupils that stay wide even in bright light, dry mouth, hot dry skin, and no sweating: Tricyclics block acetylcholine (the messenger that tells glands to make moisture and muscles to squeeze). This creates anticholinergic poisoning: your pupils blow wide, your mouth dries out, and your skin turns hot because you cannot sweat. These signs almost always travel together in tricyclic overdose.
- Confusion, extreme sleepiness, or complete loss of consciousness: The drug floods into the brain and slows down the central nervous system. At the same time, low blood pressure means less oxygen reaches the brain. Together, both break the brain's ability to stay awake and think clearly.
- Seizures or sudden jerking movements: Tricyclics block GABA (the brain's main brake pedal). Without GABA holding neurons back, electrical storms (seizures) fire. Seizures also make the blood turn acidic, which makes the heart rhythm even more dangerous.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) → Is the heart's electrical system poisoned? How wide is the QRS, and is there a tall R wave in aVR?
- Arterial blood gas (ABG) → Is the blood turning acidic, and is the body getting enough oxygen?
- Basic metabolic panel (sodium, potassium, bicarbonate, creatinine) → Are the kidneys still working, and are the blood salts normal?
- Serum lactate → Are the tissues starving for oxygen because blood pressure is too low?
- Urine drug screen and specific tricyclic level (if available) → What drug caused the overdose, and how much is in the bloodstream?
- Creatine kinase (CK) → Did seizures or prolonged immobility break down muscle?

Treatment logic

- Sodium bicarbonate 1–2 mEq/kg IV bolus, repeat to keep arterial pH between 7.45 and 7.55 → Extra sodium floods the heart muscle and competes with the tricyclic drug, forcing the sodium gates back open. Higher pH also weakens the drug's grip. Together, the QRS narrows and dangerous rhythms are stopped before they start.
- IV normal saline bolus (500–1,000 mL) and norepinephrine infusion (starting 0.05–0.1 mcg/kg/min) to raise blood pressure → Fluid refills the tank; norepinephrine squeezes blood vessels tight again. Together they push blood pressure back up so the brain, heart, and kidneys get oxygen.
- Benzodiazepine (lorazepam 2–4 mg IV or diazepam 5–10 mg IV) for seizures → Benzos turn up GABA (the brain's main brake signal). Tricyclics turned GABA down, so neurons fired out of control; the benzo puts the brakes back on and stops the seizure.
- Activated charcoal 1 g/kg by mouth or nasogastric tube if the patient took the pills within 1–2 hours and can protect their airway → Charcoal sticks to tricyclic molecules still sitting in the stomach so they cannot cross into the bloodstream.
- Continuous cardiac monitor and repeat 12-lead ECG every 2–4 hours until QRS stays normal for 24 hours → You catch QRS widening or new tall R waves early, so you can push more bicarbonate before the heart collapses into ventricular fibrillation.
- 20% lipid emulsion IV (1.5 mL/kg bolus, then 0.25 mL/kg/min infusion) if the patient goes into cardiac arrest that does not respond to standard resuscitation → The fatty emulsion acts like a sponge, soaking up tricyclic molecules and pulling them out of heart cells. This frees the heart muscle to squeeze again.

Medications

Sodium bicarbonate · Alkalinizing agent (a medicine that makes the blood less acidic)

Tricyclic antidepressants (TCAs) block sodium channels in the heart, which slows the electrical signals that make the heart beat in rhythm. When the blood becomes more alkaline (less acidic), the TCA molecule loosens its grip on those sodium channels, freeing them up so the heart can conduct signals normally again. The extra sodium ions also flood the channels and help push the electrical current through despite some remaining blockade. This double effect—higher pH and more sodium—reverses the life-threatening heart rhythm problems and low blood pressure caused by TCA overdose.

1. You give sodium bicarbonate as a fast IV push (1–2 mEq per kilogram of body weight).
2. The bicarbonate raises the blood pH toward 7.45–7.55, making it more alkaline.
3. The higher pH changes the shape of the TCA molecule so it falls off the sodium channels.

4. The flood of sodium ions also competes with the TCA blockade and pushes current through the heart.
5. The heart's electrical system starts conducting normally again: the QRS interval narrows, dangerous rhythms (like ventricular a fast heart rate (tachycardia)) stop, and blood pressure comes up.

SAFETY WATCH

Watch for over-alkalinization: if pH goes much above 7.55, you can trigger muscle twitching, confusion, or low calcium and potassium. · Repeat boluses as needed if the QRS widens again or arrhythmias return; some patients need several doses. · Use continuous cardiac monitoring and check blood gases and electrolytes frequently. · Do NOT use sodium bicarbonate as a substitute for airway management or seizure control—treat those problems separately.

Normal saline (0.9 % sodium chloride IV fluids) · Isotonic crystalloid fluid

TCA overdose causes blood vessels to relax and the heart to pump weakly, which drops blood pressure. Giving IV fluids fills up the blood vessels (like topping off a half-empty tank) so there is enough volume for the heart to pump forward. The extra sodium in normal saline also helps reverse the sodium-channel blockade in the heart, just like sodium bicarbonate does.

1. You hang a bag of normal saline and run it quickly through an IV line.
2. The fluid spreads into the bloodstream and fills the blood vessels.
3. Blood pressure rises because there is now more volume for the heart to push.
4. The sodium in the fluid also floods the heart's sodium channels, helping electrical signals travel even when some channels are still blocked by the TCA.

SAFETY WATCH

Give fluids carefully if the patient already has fluid overload or heart failure (listen for crackles in the lungs). · Watch for signs of too much fluid: swelling, trouble breathing, or jugular veins that bulge. · If blood pressure does not improve after 1–2 liters of saline, you will need to add a vasopressor medication.

Norepinephrine · Vasopressor (a medicine that tightens blood vessels and raises blood pressure)

TCA overdose blocks alpha receptors on blood vessels (the switches that normally keep vessels snug) and weakens the heart, causing blood pressure to fall. Norepinephrine is a powerful signal that turns on both alpha receptors (to squeeze blood vessels tight) and beta receptors in

the heart (to make it pump harder). This two-pronged push brings blood pressure back up and keeps blood flowing to the brain and kidneys.

1. You start a norepinephrine drip through a central IV line (or a large peripheral line if no central line is in yet).
2. Norepinephrine binds to alpha-1 receptors on blood vessels, making them constrict and raising resistance.
3. It also binds to beta-1 receptors in the heart, increasing how hard and how fast the heart beats.
4. Blood pressure climbs, and vital organs get the blood flow they need.

SAFETY WATCH

Give through a central line whenever possible; if it leaks into the skin (extravasation), it can kill tissue and cause severe injury. · Watch fingers and toes for paleness or coldness—very high doses can reduce blood flow to the limbs. · Monitor blood pressure every few minutes and titrate the drip to keep systolic BP at least 90–100 mm Hg. · Never stop norepinephrine suddenly; taper it down slowly as the patient stabilizes.

Lorazepam or diazepam · Benzodiazepine (a medicine that calms the brain by boosting its main 'brake' signal)

TCA overdose lowers the seizure threshold by blocking brain receptors that normally keep electrical activity in check. Benzodiazepines turn up GABA (the brain's chief calming messenger) by making GABA receptors more sensitive, which puts the brakes on runaway electrical storms and stops the seizure.

1. You give lorazepam 2–4 mg IV (or diazepam 5–10 mg IV) as a slow push.
2. The benzodiazepine locks onto GABA-A receptors and makes them work harder when GABA lands on them.
3. Brain cells become much less excitable, and the seizure activity dies down.
4. Muscle jerking stops, and consciousness gradually returns once the seizure ends.

SAFETY WATCH

Watch breathing closely—benzodiazepines can slow or stop respirations, especially in someone already drowsy from TCA. · Have airway equipment and a bag-valve mask ready; be prepared to intubate if the patient cannot protect their airway. · Repeat the dose every 3–5 minutes if the seizure continues, up to a total of about 0.1 mg/kg lorazepam. · Do NOT give flumazenil (a benzodiazepine blocker) to wake the patient up—it can trigger more seizures in TCA overdose.

Activated charcoal · Gastrointestinal adsorbent (a powder that binds drugs in the stomach and intestines)

Activated charcoal is a fine black powder with a huge surface area, like a sponge full of tiny pores. TCA molecules stick to the charcoal particles and are trapped, so they cannot be absorbed into the bloodstream. The charcoal and the bound drug then pass out in the stool. This works only if there is still TCA sitting in the gut—usually within the first hour or two after ingestion.

1. You mix activated charcoal (usually 50 g for an adult) with water to make a thick black slurry.
2. The patient drinks it, or you give it through a nasogastric tube if they cannot swallow safely.
3. TCA molecules in the gut stick to the charcoal surface.
4. The charcoal, now loaded with TCA, moves through the intestines and is eliminated in stool before the drug can enter the blood.

SAFETY WATCH

Never give charcoal to someone who is vomiting, drowsy, or unable to protect their airway—they can breathe it into the lungs (aspiration), which causes severe pneumonia. · Protect the airway first: intubate if the patient has a depressed level of consciousness or is seizing. · Charcoal turns stool jet black; warn the patient or family so they do not panic. · Do NOT give charcoal if the patient drank a corrosive substance or has a hole (perforation) in the gut.

Intravenous lipid emulsion (Intralipid 20 %) · Lipid rescue therapy (an IV fat solution that pulls fat-loving drugs out of tissue)

TCAs are lipophilic (they love fat). When you infuse a concentrated fat emulsion into the bloodstream, it creates a 'lipid sink'—a fatty compartment that the TCA molecules are drawn into, pulling them away from the heart and brain where they are causing toxicity. This can restart the heart or stabilize dangerous rhythms when nothing else works. The exact mechanism is still debated, but clinical experience shows it can be life-saving in refractory cardiac arrest from TCA overdose.

1. You give a bolus of 20 % lipid emulsion: 1.5 mL per kilogram IV over 1 minute (about 100 mL for a 70-kg adult).
2. Immediately start an infusion at 0.25 mL/kg/min.
3. The lipid droplets circulate and TCA molecules partition into them, reducing the free drug level at the heart and brain.
4. Cardiac function may improve within minutes; continue the infusion and repeat boluses (up to 3 mL/kg total) if arrest persists.

SAFETY WATCH

Lipid emulsion can interfere with lab tests (makes blood samples look milky and blocks some analyzers). · Watch for fat overload syndrome (rare): back pain, trouble breathing, confusion, or very high triglycerides. · Do NOT delay CPR, sodium bicarbonate, or epinephrine to mix lipid emulsion—give lipid as an add-on during ongoing resuscitation. · Use a separate IV line if possible; lipid can clog filters and interact with some drugs.

Red flags

- QRS complex wider than 100 milliseconds on the EKG — the single most important danger sign in TCA overdose. It predicts seizures and deadly heart rhythms, and it is the trigger to give IV sodium bicarbonate right away.
- A tall R wave in lead aVR (taller than 3 mm) — an EKG fingerprint of the sodium-channel poisoning TCAs cause in the heart's wiring.
- Rapid crash in the first 1 to 2 hours — a TCA patient can go from awake and talking to seizing, with low blood pressure (hypotensive), and unconscious (comatose) in minutes. Never trust an early 'looks fine.'
- Seizures — each seizure makes the blood more acidic, and too much acid in the blood (acidosis) makes the TCA grip the heart's sodium channels even harder (a vicious cycle: treat aggressively and re-check the EKG).
- Blood pressure that stays low after IV fluids — both the heart pump and the blood vessels are poisoned; norepinephrine is the vasopressor of choice.
- Falling level of consciousness — plan for early airway protection; slow, shallow breathing raises CO₂, worsens too much acid in the blood (acidosis), and feeds the cardiotoxicity loop.

Nursing priorities

1. Place patient on continuous cardiac monitor and obtain 12-lead ECG immediately, checking QRS width and looking for tall R wave in lead aVR — Sodium channel blockade (TCA stopping normal electrical flow in heart cells) widens the QRS complex over 100 milliseconds and creates tall R wave in aVR, which predicts life-threatening ventricular arrhythmias and need for sodium bicarbonate treatment
2. Establish large-bore IV access and prepare sodium bicarbonate 1 to 2 milliequivalents per kilogram for immediate bolus if QRS is over 100 milliseconds or patient has low blood pressure or arrhythmia — Sodium bicarbonate increases blood sodium levels and raises pH (makes blood less acidic), which overcomes sodium channel blockade by flooding channels with sodium ions and reducing TCA binding to heart tissue, narrowing the QRS and preventing cardiac arrest

3. Check blood pressure every 5 to 15 minutes and start IV fluid bolus of normal saline 500 to 1000 milliliters; prepare norepinephrine infusion if systolic pressure stays below 90 despite fluids — Tricyclics cause vasodilation (blood vessel widening from alpha receptor blockade) and direct heart muscle depression, dropping blood pressure; norepinephrine (a vasopressor that tightens blood vessels) reverses this better than dopamine or epinephrine
4. Assess and protect airway, position patient on side if drowsy, and have suction and bag-valve-mask at bedside; monitor respiratory rate and oxygen saturation continuously — Central nervous system depression (brain sedation from anticholinergic effects and sodium channel blockade in brain tissue) causes decreased level of consciousness and loss of airway reflexes, risking aspiration pneumonia and respiratory failure
5. Keep benzodiazepines (lorazepam 2 to 4 milligrams IV or diazepam 5 to 10 milligrams IV) immediately available and give at first sign of muscle twitching or seizure activity — Seizures happen from TCA effects on brain sodium channels and increase too much acid in the blood (acidosis) (too much acid in blood), which makes cardiotoxicity worse by increasing TCA binding to heart tissue; benzodiazepines stop seizures by calming overactive brain cells
6. Monitor core temperature every 30 minutes and look for signs of anticholinergic toxidrome: flushed dry skin, dilated pupils, urinary retention, decreased bowel sounds, confusion — Anticholinergic effects (blocking muscarinic receptors that control sweating and cooling) prevent heat loss and cause hyperthermia (dangerously high body temperature); these signs confirm TCA poisoning versus other causes of altered mental status and wide QRS
7. Recheck 12-lead ECG every 15 to 30 minutes after each sodium bicarbonate dose, measuring QRS width and monitoring for ventricular arrhythmias or heart blocks — QRS narrowing below 100 milliseconds shows that sodium bicarbonate is working to reverse cardiac sodium channel blockade; continued widening or new arrhythmias mean more bicarbonate is needed or lipid emulsion rescue therapy should start
8. Prepare 20 percent lipid emulsion (Intralipid) 1.5 milliliters per kilogram bolus with infusion ready if patient develops ventricular a fast heart rate (tachycardia), ventricular fibrillation, or pulseless electrical activity unresponsive to sodium bicarbonate and defibrillation — Lipid emulsion (IV fat solution) creates a lipid sink (fat layer in blood) that pulls lipophilic (fat-loving) TCA molecules away from heart tissue, reversing cardiotoxicity when standard treatments fail in cardiac arrest situations

Sources

- ACMT tricyclic antidepressant toxicity; ToxNet

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