

Ventricular Tachycardia and Ventricular Fibrillation

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THE STORY IN ONE SENTENCE

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“The ventricles beat so fast or so chaotically that they can't pump blood. If unstable, shock or pace right away. If stable, use medicine. If it happens twice, put in a defibrillator to prevent sudden death.”

Normal anatomy — the body's job

The heart's electrical system starts in the sinoatrial node (the natural pacemaker in the right atrium, one of the top chambers). That signal travels down through the atrioventricular node (the relay station between the top and bottom chambers) and then splits into the bundle of His and its branches, spreading across both ventricles so they squeeze together in one strong, coordinated push. This orderly flow keeps blood moving smoothly to the lungs and body.

BODY'S JOB

The ventricles are the heart's main pumps. The right ventricle pushes oxygen-poor blood to the lungs to pick up oxygen. The left ventricle pushes oxygen-rich blood out to the whole body. Each squeeze has to be strong and timed perfectly so the chambers fill completely between beats.

1. The sinoatrial node (SA node) in the right atrium fires an electrical spark about 60–100 times per minute.

2. The spark spreads across both atria, making them squeeze and push blood down into the ventricles.
3. The signal reaches the atrioventricular node (AV node), which acts like a speed bump, slowing the signal just enough to let the ventricles fill all the way.
4. The signal shoots down the bundle of His, splits into the left and right bundle branches, and fans out through the Purkinje fibers, which are like tiny wires woven into the ventricle walls.
5. Both ventricles squeeze at the same instant, pushing blood out to the lungs and body.
6. The ventricles relax, refill with blood from the atria, and wait for the next signal. The cycle repeats every second or so.

What breaks

A patch of ventricle muscle starts firing its own chaotic or very fast electrical signal, ignoring the normal pacemaker. The ventricles either race too fast to fill (ventricular a fast heart rate (tachycardia)) or quiver uselessly without any real squeeze (ventricular fibrillation). Either way, blood flow drops or stops completely.

CLINICAL TERM

Ventricular a fast heart rate (tachycardia) (VT) and ventricular fibrillation (VF). In VT, a reentrant circuit or irritable focus in the ventricle fires at 120–250 beats per minute or faster, producing wide QRS complexes on the monitor. In VF, multiple chaotic wavelets fragment across the ventricles, producing a jagged, disorganized line with no effective contraction and no cardiac output.

How the body compensates

The body has almost no time to compensate once VT or VF begins. If the rhythm is fast VT with a pulse, the sympathetic nervous system releases adrenaline to try to keep blood pressure up, but within seconds to minutes the heart cannot fill enough and blood pressure crashes. If the rhythm is VF, there is no cardiac output at all—the person loses consciousness in seconds and will die in minutes without CPR and defibrillation.

1. {'what': 'The carotid and aortic baroreceptors (pressure sensors in the neck and chest arteries) sense the sudden drop in blood pressure.', 'why': 'They signal the brainstem to release adrenaline (epinephrine) and norepinephrine, trying to squeeze blood vessels tighter and speed the heart even more to restore pressure.'}

2. {'what': 'The brain, heart, and kidneys—high-priority organs—get whatever little blood is still moving, while skin, gut, and muscles are starved.', 'why': 'This buys a few extra seconds of consciousness in VT with a pulse, but it cannot hold for long because the heart is not filling properly.'}
3. {'what': 'If the rhythm is VF or pulseless VT, compensation stops instantly.', 'why': 'The ventricles are not squeezing at all—just quivering. No blood moves. Brain cells start dying after four to six minutes without oxygen. CPR provides about 25–30% of normal blood flow, just enough to keep brain and heart cells alive until a defibrillator shock can reset the rhythm.'}
4. {'what': 'Even after the rhythm is shocked back to normal, the heart muscle may be stunned and pump weakly for hours.', 'why': 'The chaotic firing and lack of oxygen injure the cells. The body needs time, fluids, and sometimes medicines (like dopamine or norepinephrine) to support blood pressure while the heart recovers.'}

Domino effect

1. A patch of ventricle muscle fires rapidly on its own, or multiple patches fire chaotically, taking over from the normal sinoatrial node pacemaker. — Scar tissue from an old heart attack, low potassium, an inherited ion-channel mutation, or acute starved blood flow (ischemia) makes the ventricle electrically unstable. A single extra beat at just the wrong moment can start a reentry loop or trigger chaotic firing.
2. In ventricular a fast heart rate (tachycardia), the ventricles beat so fast—often 150 to 250 times per minute—that they cannot relax and fill with blood between beats. Cardiac output drops sharply. — The person feels their heart racing (palpitations), becomes dizzy or short of breath, has chest pain, and may faint (syncope) as blood pressure falls. If VT is very fast or lasts more than a few seconds, it often degenerates into ventricular fibrillation.
3. In ventricular fibrillation, the ventricles quiver in a completely disorganized way—no coordinated squeeze at all. Cardiac output is zero. — The person collapses instantly, loses consciousness, stops breathing normally (may gasp a few times), and has no pulse. This is sudden cardiac arrest. Without CPR and defibrillation within minutes, brain death and irreversible organ failure follow.
4. The brain, heart, kidneys, liver, and all organs are starved of oxygen and nutrients. Lactic acid builds up as cells switch to anaerobic metabolism. — Brain cells start dying after four to six minutes. Heart muscle that is already blood-starved (ischemic) suffers more damage. Kidneys shut down. The longer the arrest lasts, the lower the chance of survival and the higher the risk of permanent brain injury even if the heart is restarted.

5. If the rhythm is shocked back to normal (or if VT spontaneously stops), the heart may restart but remain weak and unstable. — The underlying cause—scar, low electrolytes, starved blood flow (ischemia), or genetic mutation—is still there. Without fixing the root problem and protecting the heart (often with an implantable cardioverter-defibrillator, or ICD), VT or VF will likely happen again, sometimes within hours or days.
6. Recurrent VT or VF leads to progressive heart failure, repeated shocks from an ICD, and eventual death if the cycle is not broken. — Each episode injures the heart a little more. Treatment must address the cause: revascularization (stents or bypass) for blocked arteries, correcting potassium and magnesium, stopping toxic drugs, ablating the scar tissue that creates reentry circuits, or placing an ICD to shock any future VF automatically and save the person's life.

Symptoms · what the patient feels, and why

- Fast, wide heartbeat pattern on the monitor (ventricular a fast heart rate (tachycardia)) or chaotic squiggly line (ventricular fibrillation): The bottom pumping chambers (ventricles) are firing signals on their own instead of waiting for the normal pacemaker up top, so the whole heart rhythm is taken over by the wrong part. In VT the ventricles beat very fast but in a steady pattern; in VFib they just quiver and can't pump at all.
- Sudden collapse or loss of consciousness: When the ventricles beat so fast or quiver uselessly, they can't fill with blood properly between beats, so almost no blood is pumped to the brain. The brain runs out of oxygen in seconds and the person passes out or collapses.
- No pulse or very weak pulse: The ventricles are either beating too fast to squeeze out blood properly (VT) or just shaking and not pumping at all (VFib), so little or no blood reaches the arteries where we feel a pulse.
- Chest pain or pressure: The heart muscle itself needs steady blood flow through the coronary arteries. When the ventricles can't pump well, blood pressure drops and the heart muscle may not get enough oxygen, causing pain. Also, VT and VFib are often triggered by a heart attack, which hurts on its own.
- Sudden shortness of breath or gasping: The lungs depend on the heart to pump oxygen-rich blood back out to the body. When the ventricles fail to pump, blood backs up in the lungs and oxygen delivery drops, so the person feels starved for air.
- Dizziness or lightheadedness: Not enough blood is reaching the brain because the ventricles are pumping poorly or not at all. The brain notices the drop in oxygen and warns you by making you feel dizzy, like you might faint.
- Pounding or fluttering feeling in the chest (palpitations): The ventricles are beating so fast (often 150–250 times per minute in VT) that the person can feel the rapid, forceful thumping inside the chest. It feels like the heart is racing or skipping.

- Pale, cold, sweaty skin: When blood pressure drops because the heart isn't pumping well, the body pulls blood away from the skin to protect the brain and other vital organs. This leaves the skin pale and cold. The nervous system also triggers sweating as a stress response.

Focused exam

- No pulse at the neck (carotid artery) or wrist (radial artery): Ventricular a fast heart rate (tachycardia) or ventricular fibrillation means the heart's bottom chambers (the ventricles) are either firing too fast or shaking wildly — either way, they are not squeezing blood out. No blood flowing means no pulse under your fingers. This tells you the heart is in a deadly rhythm.
- Patient unresponsive or confused, not following commands: The brain needs oxygen every second. When ventricular fibrillation or pulseless ventricular a fast heart rate (tachycardia) stops blood flow, the brain starves and shuts down within 10–20 seconds. Unresponsiveness means the rhythm is killing the brain right now.
- ECG shows wide QRS (greater than 0.12 seconds), very fast (over 100 beats per minute), or chaotic squiggles with no pattern: Ventricular a fast heart rate (tachycardia) is a wide, fast rhythm that starts in the ventricles instead of the normal spark at the top of the heart. Ventricular fibrillation looks like the heart is shaking — no organized beats at all, just electrical chaos. Both mean the heart is not pumping blood.
- No blood pressure reading possible on the cuff: Ventricular fibrillation and pulseless ventricular a fast heart rate (tachycardia) produce no squeeze, so no pressure wave travels down the arteries. The cuff reads nothing. This confirms the heart is not working as a pump.
- Skin pale, cold, and clammy: Blood is not reaching the skin because the chaotic rhythm in the ventricles is not pumping it forward. The body pulls blood away from the skin to try to feed the brain and heart. Cold, clammy skin means the body is in shock.
- If conscious and stable: patient feels pounding heartbeats, dizziness, or chest pain: Stable ventricular a fast heart rate (tachycardia) (with a pulse) lets the patient stay awake long enough to feel the fast, wrong rhythm. This is your warning sign before the heart loses its pulse and the patient collapses.

Diagnostics · every test answers a question

- 12-lead ECG → What rhythm is the heart in — and is it starting in the ventricles or the top chambers?
- Serum potassium (K+) → Is potassium too low or too high — either can trigger deadly rhythms?

- Serum magnesium (Mg^{2+}) → Is magnesium too low — a hidden trigger for torsades de pointes (a twisting form of ventricular a fast heart rate (tachycardia))?
- Troponin (high-sensitivity cardiac troponin) → Is the heart muscle damaged or dying right now — like in a heart attack?
- Echocardiogram (echo) → How strong is the heart's squeeze — and is there scar or structural damage?
- Toxicology screen and medication review → Is a drug or poison causing the rhythm — like cocaine, amphetamines, or a QT-prolonging medication?
- Coronary angiography (heart catheterization) → Is a blocked artery causing a heart attack right now — the trigger for the deadly rhythm?

Treatment logic

- CPR (chest compressions 100–120 per minute, at least 2 inches deep) + defibrillation (one shock at 200 J biphasic) immediately → Compressions push blood to the brain and heart to keep them alive. The shock stops the chaotic electrical signal in the ventricles and gives the heart a chance to reset to a normal rhythm. Together they can restart the heart and save the brain.
- Epinephrine 1 mg IV push every 3–5 minutes during CPR → Squeezes blood vessels (vasoconstriction) and strengthens the heart's squeeze so more blood reaches the brain and coronary arteries while CPR and shocks are happening. This keeps vital organs alive long enough for the next shock to work.
- Amiodarone 300 mg IV push after the first shock, then 150 mg IV if the rhythm returns within 3–5 minutes → Blocks chaotic electrical signals in the ventricles (blocks sodium, potassium, and calcium channels and beta receptors) and helps the shock reset the rhythm to normal. It also steadies the heart after rhythm returns so it does not break again immediately.
- Correct potassium to 4.0–4.5 mEq/L and magnesium to ≥ 2.0 mEq/L — give IV potassium chloride and magnesium sulfate 2 g over 5–10 minutes → Potassium and magnesium stabilize the heart's electrical wiring. Low levels make the ventricles fire chaotically. Raising them back to normal steadies the rhythm and prevents it from breaking again. Magnesium is the specific fix for torsades de pointes.
- Stop any drug that prolongs the QT interval (antipsychotics, certain antibiotics like azithromycin or fluoroquinolones, tricyclic antidepressants) → QT-prolonging drugs delay the heart's electrical reset and create a window where chaotic rhythms (especially torsades de pointes) can start. Stopping the drug closes that window.

- Emergency coronary angiography and stent or bypass if a heart attack (acute MI) triggered the rhythm → Opens the blocked artery, restores blood flow to the starving heart muscle, and stops the trigger for the deadly rhythm. This fixes the root cause if the rhythm came from a fresh heart attack.
- Implantable cardioverter-defibrillator (ICD) if ejection fraction $\leq 35\%$ after 40 days post-heart attack, or $\leq 35\%$ despite 3+ months of optimal heart failure medications (ACE inhibitor or ARB, beta-blocker, aldosterone antagonist) → A device placed under the skin detects ventricular a fast heart rate (tachycardia) or ventricular fibrillation and shocks the heart back to normal automatically. It prevents sudden death at home and gives the patient a second chance after a near-fatal rhythm.

Medications

Epinephrine (adrenaline) · Vasopressor and cardiac stimulant

Epinephrine tightens blood vessels all over the body so blood pressure climbs back up, and it pushes the heart to squeeze harder and beat faster. During a cardiac arrest (when the heart has stopped pumping), this helps blood reach the brain and heart muscle itself, buying time until the rhythm can be fixed.

1. You give 1 mg into a vein every 3 to 5 minutes during CPR.
2. Epinephrine locks onto alpha receptors in arteries and veins, squeezing them tight so pressure rises.
3. It also locks onto beta-1 receptors in the heart, telling the muscle to contract with more force.
4. Higher pressure means more blood flows through the coronary arteries (the heart's own fuel lines) and up to the brain.
5. This keeps critical organs alive while you work to shock the heart back into a normal rhythm.

SAFETY WATCH

Can cause the heart to become more irritable and trigger extra dangerous beats after the person wakes up. · High doses or repeat doses may harm heart muscle because the heart is already starved for oxygen. · Once circulation returns, watch for high blood pressure and fast heart rate that may need a different drug to calm.

Amiodarone · Antiarrhythmic (class III, but blocks multiple ion channels)

Amiodarone slows down and smooths out the electrical signals running through heart muscle. It does this by blocking several gates (potassium, sodium, and calcium channels) that let

charged particles in and out of heart cells, and it also lightly blocks adrenaline receptors. The result is a longer, steadier heartbeat cycle that is harder to flip into chaos.

1. During cardiac arrest with ventricular fibrillation or pulseless ventricular a fast heart rate (tachycardia), you give 300 mg IV push after the first shock and epinephrine; if the rhythm persists, you give another 150 mg.
2. Amiodarone enters heart cells and blocks potassium channels, which lengthens the time it takes for a cell to reset (repolarize) between beats.
3. It also blocks fast sodium channels, slowing the speed of the electrical wave traveling through the muscle.
4. Calcium-channel blockade and mild beta blockade calm the heart further.
5. All together, these actions make it harder for a wandering or circling electrical signal (re-entry circuit) to keep spinning and easier for the heart to lock into one steady rhythm.

SAFETY WATCH

Can drop blood pressure, especially during the loading dose; give it slowly if the person has a pulse. · Long-term use (weeks to months) can harm the thyroid, liver, lungs, eyes, and skin, but these risks matter less during a short resuscitation. · Can prolong the QT interval on an ECG, which rarely triggers a different dangerous rhythm called torsades de pointes (a twisting pattern of ventricular a fast heart rate (tachycardia)). · Interacts with many other drugs (warfarin, digoxin, statins) by slowing their breakdown in the liver.

Lidocaine · Antiarrhythmic (class Ib sodium-channel blocker)

Lidocaine blocks fast sodium channels in heart muscle, which slows down the runaway electrical signals that cause ventricular a fast heart rate (tachycardia). It works best in injured or oxygen-starved heart tissue (like after a heart attack), because damaged cells let sodium leak in and become electrically irritable. Lidocaine quiets those irritable spots without slowing the whole heart much.

1. You give 1 to 1.5 mg per kilogram IV push during cardiac arrest (alternative to amiodarone), or as a bolus followed by an infusion (1–4 mg/min) for stable ventricular a fast heart rate (tachycardia).
2. Lidocaine enters heart cells and plugs sodium channels, especially ones that are already open or recovering.
3. This shortens the action potential (the electrical wave in each cell) and raises the threshold needed to fire another beat.
4. Irritable or extra beats from damaged muscle are suppressed, while the normal heartbeat from the sinus node continues.
5. The rhythm smooths out and the risk of flipping into fibrillation drops.

SAFETY WATCH

High blood levels cause dizziness, slurred speech, seizures, or confusion (central nervous system toxicity). · Use a lower dose in people with liver disease or low blood flow (like shock or heart failure), because the liver breaks down lidocaine. · Rarely causes the heart to beat too slowly or the pumping to weaken further. · Check for numbness around the mouth or ringing in the ears as early warning signs of toxicity.

Magnesium sulfate · Electrolyte replacement and antiarrhythmic

Magnesium is a charged particle (electrolyte) that helps stabilize the heart's electrical system. It blocks calcium from rushing into heart cells, which calms overly excited signals. It is especially important for a twisted form of ventricular a fast heart rate (tachycardia) called torsades de pointes, which happens when the heart's reset time (QT interval) is too long. Magnesium can also help if the person's magnesium or potassium level is low.

1. For torsades de pointes, you give 1 to 2 grams IV push over 5 to 15 minutes, then start a continuous drip.
2. Magnesium blocks calcium channels, which slows the inward calcium current during each heartbeat.
3. This shortens the reset phase and stops the twisting, rapid ventricular rhythm.
4. Magnesium also helps potassium channels work properly, which stabilizes the cell membrane.
5. The chaotic rhythm stops, and the heart falls back into a regular beat.

SAFETY WATCH

Giving it too fast can drop blood pressure suddenly or cause flushing and warmth. · Very high levels can weaken muscles, slow breathing, and even stop the heart (cardiac arrest from hypermagnesemia). · Use cautiously in people with kidney failure, because the kidneys clear magnesium and levels can climb dangerously high. · Check deep-tendon reflexes (like the knee jerk); if they disappear, magnesium is too high.

Procainamide · Antiarrhythmic (class Ia sodium- and potassium-channel blocker)

Procainamide slows the electrical signals in the heart by blocking sodium channels (which slows the upstroke of each heartbeat wave) and potassium channels (which lengthens the reset time between beats). This double action stops re-entry circuits—abnormal loops of electricity

that spin around scar tissue or damaged muscle and cause ventricular a fast heart rate (tachycardia). It is an alternative when amiodarone is not available or did not work.

1. For stable monomorphic ventricular a fast heart rate (tachycardia) (one repeating shape on the ECG), you give 20 to 50 mg per minute IV infusion until the rhythm converts, the patient's blood pressure drops, the QRS widens by 50%, or you have given 17 mg per kilogram total.
2. Procainamide enters heart cells and blocks sodium channels, slowing the speed of electrical spread.
3. It also blocks potassium channels, lengthening the time each cell takes to recover.
4. These effects break the circular path of electricity that keeps ventricular a fast heart rate (tachycardia) going.
5. The heart slips back into normal sinus rhythm.

SAFETY WATCH

Can drop blood pressure, especially if you give it too fast; watch closely and slow or stop the infusion if pressure falls. · Prolongs the QT interval, which can trigger torsades de pointes in some patients. · Long-term use (weeks to months) can cause a lupus-like syndrome (joint pain, rash, fever) or lower blood counts, but this matters less during acute treatment. · Use cautiously in people with kidney failure; the active breakdown product (NAPA) builds up and adds to toxicity. · Avoid in people who already have a very wide QRS or severe heart failure.

Beta blockers (e.g. metoprolol, esmolol) · Beta-adrenergic receptor antagonists

Beta blockers attach to beta-1 receptors on heart cells and block adrenaline (epinephrine and norepinephrine) from speeding the heart or making it squeeze harder. This lowers heart rate, reduces oxygen demand, and calms electrical irritability. In people with ventricular a fast heart rate (tachycardia) triggered by stress or adrenaline surges (catecholamine-sensitive VT), beta blockers can prevent or stop the arrhythmia. They are also part of long-term treatment after a heart attack or in heart failure to protect the heart and lower the risk of sudden death.

1. You give a beta blocker by mouth daily for long-term prevention, or IV (e.g. metoprolol 5 mg slow push, or esmolol infusion) for acute control of a fast rhythm.
2. The drug locks onto beta-1 receptors and blocks adrenaline's 'go faster, squeeze harder' signal.
3. Heart rate slows, and each beat takes a little longer to prepare, which reduces the chance that an extra or early beat will trigger ventricular a fast heart rate (tachycardia).

4. Oxygen demand drops, so starved blood flow (ischemia) (lack of blood flow) is less likely to irritate the heart muscle.
5. Over weeks to months, beta blockers also remodel damaged heart tissue and lower the risk of life-threatening rhythms.

SAFETY WATCH

Can drop blood pressure and heart rate too much, especially in people with heart failure or low blood volume. · May worsen asthma or chronic lung disease by narrowing airways (beta-2 effect with non-selective agents). · Can hide the warning signs of low blood sugar (shakiness, fast heart rate) in people with diabetes. · Never stop suddenly after long-term use; rebound high adrenaline can trigger chest pain, heart attack, or dangerous rhythms. · Use cautiously in people with certain conduction problems (like high-grade heart block) because beta blockers slow signal travel through the AV node.

Potassium and other electrolyte replacement · Electrolyte supplement

Potassium (K^+) is a charged particle that heart cells pump in and out to create the electrical wave of each heartbeat. Low potassium (hypokalemia) makes cells fire extra beats and can trigger or worsen ventricular a fast heart rate (tachycardia) and fibrillation. Replacing potassium (and often magnesium at the same time) stabilizes the cell membrane and restores normal electrical flow, lowering the chance of chaotic rhythms. The goal is to keep potassium between 4.0 and 5.0 mEq/L in anyone at risk for dangerous rhythms.

1. You check a basic metabolic panel and find potassium is low (for example, 3.0 mEq/L).
2. You give potassium chloride (KCl) through a vein (typically 10–20 mEq per hour for serious arrhythmias, carefully monitored) or by mouth for mild cases.
3. Potassium enters the bloodstream and then moves into cells.
4. The sodium-potassium pump uses this potassium to maintain the resting charge across the cell membrane.
5. Proper potassium levels keep the repolarization phase (reset after each beat) smooth and predictable, so extra beats and re-entry circuits are less likely.
6. The heart rhythm stabilizes.

SAFETY WATCH

Giving IV potassium too fast or in too high a concentration can stop the heart (cardiac arrest from high potassium (hyperkalemia)). · Always infuse through a large vein or central line if giving concentrated doses, because it burns and can damage small veins. · Monitor potassium levels frequently; too much potassium (above 5.5–6.0 mEq/L) causes muscle weakness, dangerous heart blocks, and can lead to ventricular fibrillation. · Use extreme caution in people with kidney failure, because they cannot clear excess potassium. · Also replace magnesium if low, because low magnesium makes it hard for cells to hold onto potassium.

Red flags

- No pulse and no breathing (cardiac arrest — start CPR and call for a defibrillator immediately)
- Sudden collapse or complete loss of consciousness
- Chest pain with a very fast heart rate and low blood pressure
- Ventricular fibrillation on the monitor (the heart is just quivering and must be shocked right away)

Nursing priorities

1. Check for a pulse within 10 seconds. If no pulse, start chest compressions immediately at 100 to 120 pushes per minute, pushing down 2 inches on the center of the chest. — Without a pulse, the heart is not pumping blood to the brain and other organs. Brain cells die after 4 to 6 minutes without oxygen. Chest compressions squeeze the heart between the breastbone and spine to push blood out to the body until we can shock the heart back to normal rhythm.
2. Attach defibrillator pads to the bare chest. Charge to 200 joules (biphasic energy level) and deliver shock immediately if the rhythm is ventricular fibrillation or pulseless ventricular a fast heart rate (tachycardia). Resume compressions right after the shock without checking pulse. — The shock stops all heart electrical activity for a split second, allowing the heart's natural pacemaker to restart a normal rhythm. Every minute of delay reduces survival by 7 to 10 percent. Biphasic shocks use less energy but work better than old-style shocks.
3. Give epinephrine 1 milligram IV push (intravenous injection) every 3 to 5 minutes during CPR. Continue until the patient has a pulse and normal rhythm. — Epinephrine (also called adrenaline) squeezes blood vessels tight, which pushes more blood up to the brain during chest compressions. It also makes the heart muscle more likely to respond to the defibrillator shock. The medicine wears off quickly, so we repeat it every few minutes.

4. Give amiodarone 300 milligrams IV push after the second shock if the rhythm does not change. If the dangerous rhythm continues and we shock again, give another 150 milligrams of amiodarone IV push. — Amiodarone (an antiarrhythmic drug that calms electrical signals in heart muscle) makes the heart less irritable and helps the normal rhythm stay stable after the shock works. It blocks the abnormal electrical pathways in the lower heart chambers that cause ventricular a fast heart rate (tachycardia) and ventricular fibrillation.
5. Draw blood immediately for potassium and magnesium levels. Give magnesium sulfate 2 grams IV over 15 minutes if levels are low or if we suspect low levels. Replace potassium through IV if the blood test shows less than 4.0. — Potassium and magnesium are electrolytes (charged minerals) that control electrical signals in heart muscle cells. Low levels make the heart very irritable and cause repeated dangerous rhythms even after successful shocks. The ventricular a fast heart rate (tachycardia) or ventricular fibrillation will keep coming back until we fix these levels.
6. Prepare for advanced airway placement with an endotracheal tube (breathing tube inserted into the windpipe) if CPR continues past 2 minutes. Give 100 percent oxygen through a bag-mask until the tube is placed. — The brain and heart need oxygen to survive and recover. Chest compressions only pump about 25 to 30 percent of normal blood flow, so we must give pure oxygen to make every drop of blood carry as much oxygen as possible. The breathing tube protects the airway during continued compressions and prevents vomit from entering the lungs.
7. Check the medication list and IV pumps. Stop any QT-prolonging drugs like ciprofloxacin, ondansetron, or haloperidol that might have triggered torsades de pointes (a twisting type of ventricular a fast heart rate (tachycardia)). — Some common medicines make the heart's electrical reset time longer (called QT prolongation on the heart monitor). When the reset takes too long, the heart can develop torsades de pointes, which looks like a twisting ribbon on the monitor. Continuing these drugs will cause the dangerous rhythm to return even after successful treatment.
8. After successful rhythm conversion, start a continuous amiodarone IV drip at 1 milligram per minute for 6 hours, then 0.5 milligrams per minute for 18 hours. Keep the patient on a cardiac monitor with alarm limits set. — The heart remains very irritable for hours after ventricular a fast heart rate (tachycardia) or ventricular fibrillation. The continuous amiodarone infusion keeps medicine levels steady in the blood to prevent the dangerous rhythm from starting again. Most patients who survive have repeat episodes within the first 24 hours without prevention medicine.

Sources

- AHA/ACC 2017 Guideline for Management of Patients With Ventricular Arrhythmias

- AHA Advanced Cardiovascular Life Support (ACLS) 2020
- European Society of Cardiology 2022 Ventricular Arrhythmia Guideline
- UpToDate Clinical Overview: Ventricular Tachycardia and Fibrillation

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