

Unstable tachyarrhythmia

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THE STORY IN ONE SENTENCE

The heart is beating so fast that it cannot fill with blood between beats, so the amount of blood pumped out drops and the body goes into shock.

“Unstable tachyarrhythmia — the broken rhythm is now the reason the patient is dying, and you must fix it in minutes.”

Normal anatomy — the body's job

The sinus node (a cluster of pacemaker cells in the right atrium, the top-right chamber) sets how fast the heart beats based on signals from the brain and body—faster when you run, slower when you sleep. Each heartbeat has two phases: systole (when the heart squeezes and pumps blood out) and diastole (when the heart relaxes and the ventricles, the bottom chambers, fill with blood). During diastole, the atria (top chambers) squeeze last to give the ventricles a final 20 percent push of blood—that is called the atrial kick. Then the electrical signal travels down the AV node (the connection between top and bottom chambers) into the bundle of His and then spreads through the Purkinje fibers so both ventricles squeeze together in an organized, powerful wave that pushes blood out to the lungs and body. The coronary arteries deliver oxygen-rich blood to the heart muscle itself, but only during diastole when the heart is relaxed and the vessels are not squeezed shut. Keep that picture in your head, because every weird finding in unstable tachyarrhythmia is a change from this normal flow.

BODY'S JOB

The sinus node's job is to keep the heart beating at exactly the rate the body needs to deliver oxygen and nutrients everywhere. Diastole's job is to let the ventricles fill completely so they have enough blood to pump out with the next squeeze. The atrial kick's job is to top off the ventricles right before they contract. The conduction system's job is to make sure the signal spreads in the right order—top chambers first, then bottom chambers together—so the heart pumps efficiently. The coronary arteries' job is to feed the heart muscle itself with oxygen during rest time. If any of these jobs break, the heart cannot deliver blood to the body.

1. The sinus node fires an electrical signal.
2. The signal spreads across both atria, and they squeeze, filling the ventricles below.
3. The signal reaches the AV node, pauses briefly, then travels down the bundle of His.
4. The signal shoots through the Purkinje fibers and spreads across both ventricles, which squeeze together and pump blood out.
5. The heart relaxes (diastole), the ventricles fill with blood, and the coronary arteries feed the heart muscle with oxygen.
6. The cycle repeats at the rate the body needs.

What breaks

In unstable tachyarrhythmia, the normal pacemaker loses control. Instead, either a different spot in the heart starts firing electrical signals faster than the sinus node (called an ectopic focus), or electricity starts spinning around in a loop (reentry circuit) that fires over and over, or the AV node lets signals through too fast without the normal delay. Whatever the cause, the ventricles are now being told to squeeze 150, 200, or even 250 times per minute. They squeeze before they have time to fill with blood, so even though the heart is beating fast, almost no blood is pumped out with each beat. The amount of blood the body gets per minute (cardiac output) drops, and blood pressure falls. That is the core break.

CLINICAL TERM

The primary defect is loss of sinus rhythm with takeover by a rapid ectopic focus (atrial a fast heart rate (tachycardia), atrial flutter, atrial fibrillation (an irregular, quivering heartbeat) with rapid ventricular response, junctional a fast heart rate) or a ventricular reentry circuit (ventricular a fast heart rate, supraventricular a fast heart rate via an accessory pathway such as in WPW). In all cases, ventricular diastolic filling time is severely shortened, stroke volume falls, and cardiac output drops below the level needed to perfuse vital organs. The patient is hemodynamically unstable—meaning low blood pressure, altered mental status, chest pain, pulmonary edema (fluid flooding the lungs), or signs of shock—because the rhythm itself is causing the collapse.

How the body compensates

The body tries hard to keep blood pressure and oxygen delivery steady even as the heart is failing, so vital signs can look okay for a while. That is the dangerous middle—the patient looks stable but is actually on the edge of collapse. Your job is to recognize the hidden crash point and act before the patient tips over into cardiac arrest.

1. {'step': 'The sympathetic nervous system releases catecholamines (adrenaline and noradrenaline) to try to squeeze the heart harder and faster.', 'why': 'The body senses low blood pressure and tries to boost output, but making the heart beat even faster only shortens diastole more and worsens the filling problem.'}
2. {'step': 'Blood vessels clamp down (vasoconstriction) to try to keep blood pressure up.', 'why': 'Squeezing the arteries raises resistance and props up the blood pressure number, but it does not fix the fact that almost no blood is being pumped, so organs are still starving.'}
3. {'step': 'The kidneys hold onto salt and water to try to keep blood volume up.', 'why': 'More volume in the pipes can raise pressure, but the heart cannot pump that extra fluid forward, so it backs up into the lungs (pulmonary swelling (edema)) and liver.'}
4. {'step': 'The patient may still have a pulse and a measurable blood pressure for minutes to hours.', 'why': 'Compensation can prop things up temporarily, which is why the team may not realize how sick the patient is—but this is borrowed time, not stability.'}
5. {'step': 'Watch for the crash point: confusion or loss of consciousness, chest pain, systolic blood pressure below 90, pulmonary edema (fluid flooding the lungs) (shortness of breath, crackles in the lungs), or signs of shock (cold skin, weak pulse, low urine output).', 'why': 'Any of these means the rhythm is now immediately life-threatening and you must move from medicines to immediate synchronized cardioversion (a controlled electrical shock) or, if the patient has no pulse, unsynchronized defibrillation.'}

6. {'step': 'Do not wait for full cardiac arrest. Recognize instability early and act.', 'why': 'Once the patient loses pulses or drops into ventricular fibrillation, the window to get the heart back shrinks fast, and brain damage begins within minutes.'}
7. {'step': 'Look at the trend, not just one moment. A patient with a blood pressure of 95 that was 110 five minutes ago and 120 ten minutes ago is crashing, not stable.', 'why': 'Compensation is failing in real time, and the next step is collapse.'}

Domino effect

1. An ectopic focus or reentry circuit takes over and fires electrical signals faster than the sinus node. — The sinus node normally controls the rate, but when a different spot in the atria, AV node, or ventricles starts firing faster—or when electricity spins in a loop that triggers itself over and over—that abnormal rhythm hijacks control and the heart rate shoots up to 150, 200, or more beats per minute.
2. Diastole (the heart's relaxation and filling time) almost disappears. — At 200 beats per minute, the heart is squeezing three times per second, so there is almost no time between beats for the ventricles to relax and fill with blood from the atria.
3. The atrial kick is lost or out of sync. — In many fast rhythms, the atria are either fibrillating (quivering uselessly) or contracting at the wrong time, so the final 20 percent push of blood into the ventricles is gone, making the filling problem even worse.
4. Stroke volume (the amount of blood pumped with each beat) drops sharply. — The ventricles are squeezing when they are nearly empty, so even though the heart is beating fast, almost no blood is ejected with each contraction.
5. Cardiac output (total blood pumped per minute) falls below what the body needs, and blood pressure drops. — Cardiac output equals stroke volume times heart rate. Even though the rate is very high, the stroke volume is so low that total output falls, and organs begin to starve for oxygen.
6. The body releases catecholamines (adrenaline and noradrenaline) to try to boost output. — The brain senses low blood pressure and triggers the sympathetic nervous system to make the heart beat harder and faster, but this only makes the rate go higher and shortens diastole even more, worsening the spiral.
7. The heart muscle itself becomes blood-starved (ischemic) (starved of oxygen). — The coronary arteries deliver blood to the heart muscle only during diastole when the heart is relaxed and the vessels are not squeezed shut. A heart beating 200 times per minute with almost no rest time cannot get enough oxygen, so the muscle weakens and can start to die—even if the coronary arteries are wide open.

8. The patient develops confusion, chest pain, shortness of breath, and signs of shock. — The brain, heart, lungs, and kidneys are not getting enough blood, so the patient becomes confused (low brain oxygen), has chest pain (heart starved blood flow (ischemia)), breathes fast and has crackling lungs (fluid backs up into the lungs), has cold clammy skin and weak pulses (shock), and makes little urine (kidney failure).
9. Without treatment, the rhythm degenerates into ventricular fibrillation or pulseless ventricular a fast heart rate (tachycardia), or the heart muscle has a full heart attack and stops pumping entirely. — The blood-starved (ischemic), overstressed heart muscle becomes electrically unstable and breaks into chaotic, fatal rhythms, or it simply runs out of energy and stops. Either way, the patient has no pulse and will die within minutes unless you defibrillate and do CPR.

Symptoms · what the patient feels, and why

- Low blood pressure (systolic below 90): When the heart beats too fast (usually over 150), the chambers don't have enough time to fill with blood between beats, so less blood gets pumped out with each squeeze — like trying to fill a water balloon while someone keeps squeezing it before it's full. Less blood out means lower pressure in the arteries.
- Confusion, trouble speaking, or acting strangely: The brain needs a steady stream of oxygen-rich blood to work right. When the heart is racing and pumping poorly, not enough blood reaches the brain — like dimming the lights when the power drops — so thinking gets fuzzy and the person may seem 'out of it.'
- Chest pain or pressure: The heart muscle itself needs oxygen from the coronary arteries (the blood vessels that feed the heart). When the heart is beating extremely fast, it burns through oxygen faster than the blood can deliver it — starved blood flow (ischemia) (not enough blood reaching the tissue to feed it) — and the muscle starts to hurt, like a cramp.
- Shortness of breath or rapid breathing: The left side of the heart can't keep up with the fast rate, so blood backs up into the lungs — pulmonary edema (fluid flooding the lungs) (fluid leaking into the tiny air sacs). This crowds out space for oxygen, making every breath feel like sucking air through a wet sponge.
- Very fast pulse (usually over 150 beats per minute at rest): An abnormal electrical circuit or focus in the heart fires signals much faster than the normal sinus node (the heart's natural pacemaker). The heart obeys these rapid signals and beats way too fast — like a drummer playing double-time without stopping.
- Feeling dizzy or about to faint (presyncope): Less blood pumped per minute means less reaches the brain. The brain's blood flow drops just enough to make you feel lightheaded — like standing up too fast — warning that you might pass out if it gets worse.

- Patient may look pale, sweaty, and anxious: Low blood pressure and lack of oxygen trigger the body's stress system — the sympathetic nervous system (the 'fight or flight' branch) — which dumps adrenaline (epinephrine, a hormone that amps up the body). Adrenaline shunts blood away from the skin (making you pale), cranks up sweat glands, and makes you feel jittery and scared.

Focused exam

- Heart rate above 150 beats per minute with a pulse you can feel: The speed itself is the problem — when the heart beats this fast, it does not have enough time to fill with blood between beats (during diastole, the relaxing phase). You need to measure the rate and ask: Is the patient sick because of this speed?
- Blood pressure less than 90 systolic or dropping from the patient's normal baseline: Low blood pressure means the fast heartbeat has broken the pump — the heart is squeezing many times but barely pushing any blood out because each beat is nearly empty. The body is not getting enough blood to stay stable.
- Confusion, altered mental status, or the patient acting strange or not responding normally: The brain is not getting enough oxygen-rich blood because the fast rhythm stole the filling time. This is the clear sign that the arrhythmia has crossed from a rhythm issue to a body-wide emergency.
- Chest pain or pressure while the fast rhythm is happening: The heart muscle itself gets fed during diastole (when it relaxes), but diastole is now too short. The heart is working hard but starving itself — this is called demand starved blood flow (ischemia) (not enough blood reaching the heart muscle to keep up with its own work).
- Crackles (wet popping sounds) in the lungs when you listen, or the patient is short of breath: Fluid is backing up into the lungs because the weak pump cannot push blood forward fast enough. The body has tried to force more fluid into the system with stress hormones like epinephrine (adrenaline), but the pump cannot handle it.
- Irregular or regular fast rhythm on the monitor — study the pattern of the heartbeats: The pattern tells you the source: chaotic and irregular means atrial fibrillation (an irregular, quivering heartbeat) (the top chambers quivering), regular and narrow usually means a reentry loop (a signal spinning in a circle), regular and wide often means the problem is in the bottom chambers (ventricles). Each one needs a different treatment.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) → Is the rhythm wide or narrow? Regular or irregular? Which chamber is driving the fast rate?

- Serum potassium → Is the potassium level abnormal enough to trigger or worsen a dangerous rhythm?
- Serum magnesium → Is magnesium low enough to make the heart electrically unstable?
- Serum troponin I or T → Is the heart muscle being damaged right now by lack of blood flow?
- Thyroid-stimulating hormone (TSH) → Is an overactive thyroid gland causing or contributing to the fast rhythm?
- Serum lactate → Is the body switching to backup energy production because it is not getting enough oxygen?
- Chest X-ray → Is there fluid backing up into the lungs (pulmonary swelling (edema)) or another structural problem?

Treatment logic

- Synchronized cardioversion (an electric shock timed to the R wave on the ECG) → Stops the chaotic or too-fast rhythm by resetting every heart cell at the same moment, giving the sinus node (the heart's natural pacemaker in the right atrium) a chance to take control again. Blood output jumps back up because the heart can now fill and squeeze in a normal rhythm.
- Adenosine IV push 6 mg (followed by 12 mg if no response) for stable narrow regular a fast heart rate (tachycardia) → Blocks the AV node (the electrical gate between the top and bottom chambers) for about one second, which breaks a reentry loop (a signal spinning in a circle) if one exists. The sinus node then restarts normal rhythm, fill time returns, and blood output climbs.
- Diltiazem IV or metoprolol IV (beta-blocker) for stable atrial fibrillation (an irregular, quivering heartbeat) with rapid ventricular response → Slows how many signals get through the AV node per minute, so the ventricles (bottom chambers) beat slower even though the atria (top chambers) are still quivering. Longer fill time means more blood per beat, so output and blood pressure improve even though the rhythm stays irregular.
- Amiodarone IV 150 mg over 10 minutes or procainamide IV 20–50 mg/min for stable wide regular a fast heart rate (tachycardia) → Slows down the fast-firing cells or breaks a reentry circuit by making the heart muscle slower to conduct electricity. This gives the sinus node a chance to regain control and restore a normal rhythm.
- Immediate unsynchronized defibrillation (shock without timing) for pulseless ventricular a fast heart rate (tachycardia) or ventricular fibrillation → Stops the chaotic quivering or disorganized firing in the ventricles so the heart can restart organized electrical activity. Without this, the brain and organs get zero blood and die within minutes.

- Vagal maneuvers (carotid massage, bearing down as if having a bowel movement, or ice water to the face) for stable narrow regular a fast heart rate (tachycardia) → Triggers the vagus nerve (the main nerve of the parasympathetic 'rest-and-digest' system) to release acetylcholine (a calming neurotransmitter), which slows the AV node and can break a reentry loop. The sinus node then takes over, fill time returns, and output improves. This is free, safe in most patients, and works in seconds.
- Correct serum potassium and magnesium to normal range → Stabilizes the electrical charge across every heart cell membrane, making arrhythmias less likely to start or continue. Normal potassium (3.5–5.0 mEq/L) and magnesium (≥ 2.0 mg/dL) reduce the risk of life-threatening rhythms like torsades de pointes.

Medications

Synchronized cardioversion (electrical procedure, not a drug) · Emergency electrical therapy

A controlled shock is delivered through pads on the chest, timed to land during the heart's safe moment (the thick part of the heartbeat wave on the monitor, called the R wave). This timing prevents the shock from landing during the heart's vulnerable window (the T wave) when a jolt could flip a fast rhythm into ventricular fibrillation (a chaotic, deadly quiver). The shock briefly stops all electrical activity in the heart, giving the sinoatrial node (the heart's natural pacemaker in the upper-right chamber) a chance to take charge again and restore a normal, organized beat.

1. The monitor watches the heart's electrical wave and finds the tallest spike (the R wave, when the lower chambers squeeze).
2. The machine sends the shock exactly on that spike, never on the T wave (the resting phase when the heart recharges).
3. The shock stops all rogue electrical signals firing from extra spots or spinning in loops.
4. The sinoatrial node, the heart's natural boss, can now restart a steady rhythm from the top.
5. Blood flow improves immediately as the chambers squeeze in an organized pattern again.

SAFETY WATCH

The patient must be sedated (put briefly to sleep with medicine) because the shock is painful. · If the rhythm is atrial fibrillation (an irregular, quivering heartbeat) and has lasted more than 48 hours, or if the time is unknown, an ultrasound of the heart (transesophageal echocardiogram) should check for clots in the upper chambers, or the patient needs blood thinners for 3 weeks before the procedure to prevent stroke. · Skin burns can occur where the pads touch; conductive gel or special pads reduce this risk. · The shock can trigger dangerous rhythms if it accidentally lands on the T wave (which is why synchronization is critical). · Low potassium or low magnesium in the blood makes the heart more likely to fall back into a bad rhythm after cardioversion, so these must be corrected first.

Adenosine · Antiarrhythmic (purine nucleoside)

Adenosine is a natural chemical that the heart uses to slow down electrical traffic. When given as a medicine, it floods adenosine receptors on cells in the atrioventricular (AV) node (the electrical gate between the upper and lower heart chambers). This makes the gate slam shut for a few seconds, blocking extra signals from getting through. If the fast rhythm is caused by a spinning electrical loop that depends on the AV node, breaking the loop stops the a fast heart rate (tachycardia) instantly. If the rhythm is coming from a different source (like atrial flutter or atrial fibrillation (an irregular, quivering heartbeat)), adenosine will slow the heart just long enough to unmask the underlying pattern on the monitor, which helps the team figure out the diagnosis.

1. Adenosine enters the bloodstream and reaches the heart in seconds (it only lasts 10 seconds before the body breaks it down).
2. It locks onto adenosine A1 receptors on the surface of AV node cells.
3. This opens potassium channels inside those cells, letting potassium rush out.
4. The cells become hyperpolarized (electrically quieter), making it much harder for an electrical signal to pass through.
5. The AV node briefly stops conducting any signals from the atria to the ventricles.
6. If the a fast heart rate (tachycardia) was a reentry loop using the AV node (such as AV nodal reentrant a fast heart rate or AV reentrant a fast heart rate with an accessory pathway going backward), the loop breaks and the heart resets to normal rhythm.
7. If the a fast heart rate (tachycardia) was atrial flutter or atrial fibrillation (an irregular, quivering heartbeat), the ventricles pause for a moment and the true atrial rhythm becomes visible on the ECG, then the fast rate resumes.

SAFETY WATCH

The patient will feel a frightening sensation for a few seconds: chest tightness, shortness of breath, or a feeling that the heart has stopped (it has, briefly). Warn them this is normal and will pass quickly. · Adenosine must be pushed very fast through a large IV close to the heart (in the arm or above), followed immediately by a flush of saline, because it is destroyed in seconds. · It can trigger bronchospasm (airway tightening) in people with asthma; use with caution or choose a different drug. · Caffeine and theophylline (an asthma medicine) block adenosine's effects; higher doses may be needed. · Dipyridamole (a blood thinner) and carbamazepine (a seizure medicine) make adenosine much stronger; use a lower dose. · A brief pause or slow rhythm may appear right after the dose; this is expected and resolves in seconds, but have a defibrillator ready. · Do NOT use adenosine if the ECG shows a wide, irregular a fast heart rate (tachycardia) that could be pre-excited atrial fibrillation (an irregular, quivering heartbeat) (accessory pathway conducting rapidly)—this can cause cardiac arrest.

Diltiazem · Calcium channel blocker (non-dihydropyridine)

Diltiazem blocks calcium channels on cells in the AV node (the gate between the upper and lower heart chambers) and in the sinoatrial node (the heart's natural pacemaker). Calcium normally rushes into these cells to help them fire electrical signals. When diltiazem blocks the calcium gates, the cells fire more slowly and conduct impulses less eagerly. In atrial fibrillation (an irregular, quivering heartbeat) or atrial flutter with a rapid ventricular response, the AV node is letting too many chaotic signals through from the atria to the ventricles, making the heart race. Diltiazem slows the AV node down so fewer impulses get through, which drops the heart rate and gives the ventricles time to fill with blood, improving circulation and symptoms.

1. Diltiazem enters the bloodstream and reaches the heart.
2. It plugs L-type calcium channels on the surface of AV node and SA node cells.
3. Calcium can no longer flow into the cells as easily.
4. Without enough calcium, the cells take longer to charge up and fire an electrical signal.
5. The AV node conducts impulses from the atria to the ventricles more slowly.
6. In atrial fibrillation (an irregular, quivering heartbeat) or flutter, this means fewer of the rapid atrial beats make it through to the ventricles.
7. The ventricular rate drops (often from 150–160 beats per minute down to 80–110), the heart fills better between beats, and blood pressure and symptoms improve.

SAFETY WATCH

Diltiazem can drop blood pressure because it also relaxes blood vessels slightly; monitor closely and give fluids or slow the infusion if pressure falls. · It can worsen heart failure (when the heart muscle is already weak) because it reduces the force of contraction. · Do NOT use diltiazem with a beta-blocker at the same time unless absolutely necessary and closely monitored—both slow the heart and together can cause dangerous a slow heart rate (bradycardia) (heart rate too slow) or heart block. · Avoid in patients with severe heart failure, very low blood pressure, or second- or third-degree AV block (when the AV node is already barely conducting). · Watch for dizziness, fatigue, and swelling in the legs (from the blood-vessel-relaxing effect). · Do NOT use in wide-complex a fast heart rate (tachycardia) of uncertain origin—if it turns out to be ventricular a fast heart rate, calcium channel blockers can cause cardiovascular collapse.

Metoprolol (or other beta-blocker such as esmolol) · Beta-adrenergic blocker (beta-1 selective)

Metoprolol blocks beta-1 receptors on cells in the AV node (the electrical gate between the upper and lower chambers) and the sinoatrial node (the heart's natural pacemaker). Normally, adrenaline and noradrenaline (the body's stress hormones) lock onto these receptors and rev up the heart—making it beat faster and conduct signals more eagerly. Metoprolol sits in those receptor parking spots so adrenaline cannot, which slows down the AV node's willingness to pass signals from the atria to the ventricles. In atrial fibrillation (an irregular, quivering heartbeat) or atrial flutter with rapid ventricular response, this means fewer of the chaotic atrial impulses get through, the ventricles slow down, and the heart has time to fill with blood and pump effectively again.

1. Metoprolol enters the bloodstream and reaches the heart.
2. It binds to beta-1 receptors on the surface of AV node and SA node cells, blocking adrenaline and noradrenaline.
3. Without adrenaline's push, the cells fire electrical signals more slowly.
4. The AV node takes longer to conduct each impulse from the atria to the ventricles.
5. In atrial fibrillation (an irregular, quivering heartbeat) or flutter, fewer of the rapid atrial beats make it through the gate.
6. The ventricular rate drops (often from 140–160 down to 70–100 beats per minute).
7. The heart fills better, stroke volume (blood pumped per beat) improves, and blood pressure stabilizes.

SAFETY WATCH

Beta-blockers can drop blood pressure and heart rate too low, especially in patients who are already with low blood pressure (hypotensive) or bradycardic; monitor closely. · Use with extreme caution or avoid in acute decompensated heart failure (when the heart is failing right now), because beta-blockers reduce the force of contraction and can worsen pump function in the short term. · Contraindicated in severe asthma or chronic obstructive pulmonary disease (COPD) exacerbations, because even beta-1 selective blockers can narrow airways at higher doses. · Do NOT combine with diltiazem or verapamil (calcium channel blockers) without close monitoring—together they can cause severe a slow heart rate (bradycardia) or complete heart block. · Can mask signs of low blood sugar (hypoglycemia) in diabetics, especially the rapid heartbeat and shakiness. · Avoid in second- or third-degree AV block unless a pacemaker is in place. · Esmolol is an ultra-short-acting beta-blocker given by continuous IV infusion; it is useful when you need to test the effect or turn it off quickly if blood pressure drops.

Amiodarone · Antiarrhythmic (class III, but has mixed actions)

Amiodarone works on multiple electrical switches in heart muscle cells, making it a broad-spectrum rhythm stabilizer. Its main job is to block potassium channels that help cells reset after each beat. When potassium cannot leave the cell as easily, the recharging phase (repolarization) takes longer. This lengthens the time before the cell can fire again (the refractory period), which breaks up fast spinning loops of electricity (reentry circuits) and quiets cells that are firing on their own. Amiodarone also gently blocks sodium channels (slowing the upstroke of the electrical signal), calcium channels (calming the AV node), and beta-receptors (reducing adrenaline's push). In stable wide-complex a fast heart rate (tachycardia) that is likely ventricular a fast heart rate, amiodarone slows and stabilizes the chaotic firing in the ventricles, often restoring normal rhythm. It can also be used in atrial fibrillation (an irregular, quivering heartbeat) to help convert the rhythm or prevent it from returning.

1. Amiodarone enters heart muscle cells and blocks potassium channels that let potassium flow out during repolarization.
2. The cell takes longer to reset to its resting state, so the refractory period (the 'do not disturb' window) stretches out.
3. Reentry circuits (electrical loops spinning too fast) cannot keep going because the tissue stays unresponsive longer, breaking the loop.
4. Cells that are firing extra beats on their own (automaticity) are suppressed because amiodarone also blocks some sodium and calcium flow.
5. The AV node conducts more slowly because of calcium-channel and beta-receptor blockade, which helps control rate in atrial arrhythmias.

6. In ventricular a fast heart rate (tachycardia), the ventricles slow down and often flip back into normal sinus rhythm.
7. In atrial fibrillation (an irregular, quivering heartbeat), the drug reduces the number of chaotic wavelets in the atria and may restore sinus rhythm over hours to days.

SAFETY WATCH

Amiodarone can drop blood pressure, especially when given IV; slow the infusion or give fluids if this happens. · It can cause severe a slow heart rate (bradycardia) (slow heart rate) or heart block, especially in patients already on beta-blockers or calcium channel blockers. · Long-term use can damage the lungs (pulmonary fibrosis, which causes scarring and shortness of breath), the thyroid (either overactive or underactive), the liver, and the eyes (corneal deposits that blur vision). · Skin can turn blue-gray with chronic use, and patients become very sensitive to sunlight (wear sunscreen). · Amiodarone interacts with many drugs: it boosts the effect of warfarin (blood thinner), digoxin, and other heart medicines, so doses must be adjusted. · Do NOT use in patients with severe sinus node disease or second- or third-degree AV block unless they have a pacemaker. · Can prolong the QT interval on the ECG, which raises the risk of torsades de pointes (a dangerous twisting ventricular rhythm); check electrolytes (potassium, magnesium) before giving it.

Procainamide · Antiarrhythmic (class Ia sodium-channel blocker)

Procainamide blocks sodium channels in heart muscle cells, which slows down the speed at which an electrical signal travels through the tissue (conduction velocity). It also blocks potassium channels, which lengthens the time cells need to recharge before they can fire again (the refractory period). Together, these actions help break up reentry circuits (electrical loops spinning too fast) and quiet down cells that are firing extra beats on their own. In stable wide-complex a fast heart rate (tachycardia) that is likely ventricular a fast heart rate, procainamide slows the chaotic electrical activity in the ventricles and often restores normal rhythm. It can also be used in atrial fibrillation (an irregular, quivering heartbeat), especially when there is an accessory pathway (pre-excitation), because it slows conduction through both the normal pathway (AV node) and the extra pathway.

1. Procainamide enters heart muscle cells and plugs sodium channels.
2. The upstroke of each electrical signal (phase 0 of the action potential) becomes slower and less steep.
3. Electrical impulses travel more slowly through the atria, ventricles, and any accessory pathways.
4. Procainamide also blocks potassium channels, so the cell takes longer to repolarize (reset).
5. The refractory period (the 'cooling-off' time before the cell can fire again) gets longer.

6. Reentry circuits cannot sustain themselves because the tissue stays unresponsive longer, breaking the loop.
7. In ventricular a fast heart rate (tachycardia), the ventricles slow and often convert back to normal sinus rhythm.
8. In pre-excited atrial fibrillation (an irregular, quivering heartbeat), procainamide slows conduction through the accessory pathway, preventing dangerously fast ventricular rates.

SAFETY WATCH

Procainamide can drop blood pressure during infusion; slow or stop the infusion if this happens, and give IV fluids. · Stop the infusion if the QRS complex on the ECG widens by more than 50% of its starting width, or if the QT interval becomes dangerously long—these are signs of toxicity. · It can cause severe a slow heart rate (bradycardia) or heart block, especially in patients with weak hearts or conduction system disease. · Long-term use can trigger a lupus-like syndrome (joint pain, rash, fever) in up to 30% of patients, or blood cell problems (low white cells, low platelets). · Procainamide is broken down partly by the kidneys, so the dose must be reduced in kidney disease. · Can prolong the QT interval and trigger torsades de pointes (a life-threatening twisting ventricular rhythm); check and correct potassium and magnesium before giving it. · Avoid in patients with severe heart failure or very low blood pressure.

Immediate unsynchronized defibrillation (for pulseless ventricular a fast heart rate (tachycardia) or ventricular fibrillation) · Emergency electrical therapy

When the heart is in ventricular fibrillation (a chaotic quiver with no organized beat) or pulseless ventricular a fast heart rate (tachycardia) (a very fast rhythm in the lower chambers that produces no blood flow), there is no time to synchronize the shock—the patient has no pulse and is clinically dead. An unsynchronized shock delivers maximum energy immediately, without waiting for any particular part of the heartbeat. The goal is to jolt every heart cell at once, briefly stopping all electrical activity (chaotic and organized alike), so the sinoatrial node (the heart's natural pacemaker) has a chance to take over and restore a normal, life-sustaining rhythm. Defibrillation is the single most important treatment for ventricular fibrillation and pulseless ventricular a fast heart rate; every minute of delay drops survival by about 10%.

1. Paddles or adhesive pads are placed on the chest (one below the right collarbone, one on the left side below the nipple).
2. The defibrillator charges to 120–200 joules (biphasic) or 360 joules (monophasic).
3. The shock is delivered immediately, without waiting for any specific point in the cardiac cycle.
4. A massive electrical current passes through the heart, depolarizing (firing) every muscle cell at once.

5. All chaotic electrical activity stops for a brief moment.
6. The sinoatrial node, if it can, begins firing again and takes charge, producing organized beats.
7. If a normal rhythm returns, the heart begins pumping blood again; CPR continues if no pulse returns, and the shock is repeated every 2 minutes.

SAFETY WATCH

Everyone must stand clear of the patient and the stretcher before the shock is delivered—touching the patient during the shock can electrocute the rescuer. · Remove any oxygen mask or nasal cannula from directly over the chest pads (though it is safe to leave oxygen running in the room). · Wipe off wet skin or large amounts of sweat before applying pads, to ensure good contact and reduce the risk of skin burns. · Do NOT delay defibrillation to place an advanced airway or get IV access—shocking the heart is the priority. · High-quality chest compressions must resume immediately after each shock (do not stop to check a pulse right away); check rhythm and pulse only after 2 minutes of CPR. · If the patient has an implanted pacemaker or defibrillator, place pads at least 8 cm (3 inches) away from the device. · Shocks can cause skin burns where the pads contact the chest; this is unavoidable and not a reason to delay or withhold defibrillation.

Red flags

- Systolic blood pressure below 90 mmHg
- Altered mental status (confused, not responding normally, or unresponsive)
- Chest pain or signs of heart attack (acute heart attack (myocardial infarction))
- Signs of heart failure (fluid in the lungs causing extreme shortness of breath, inability to lie flat)
- Loss of consciousness or about to pass out

Nursing priorities

1. Place the patient on continuous cardiac monitoring and pulse oximetry, and obtain a 12-lead ECG within 1 minute. — The ECG shows whether the QRS complex (the electrical signal through the ventricles) is narrow or wide and whether the rhythm is regular, which determines the exact treatment protocol and whether the patient needs immediate cardioversion.
2. Establish large-bore IV access in two sites immediately. — Unstable a fast heart rate (tachycardia) may require rapid IV medication pushes like adenosine or amiodarone, and if the patient crashes we need reliable access for emergency drugs and fluids without delay.

3. Obtain a manual blood pressure every 2 to 5 minutes and assess mental status continuously.
— Blood pressure under 90 systolic or new confusion means the a fast heart rate (tachycardia) has become unstable and the patient needs synchronized cardioversion right now because the brain and organs are not getting enough blood flow.
4. Prepare the defibrillator in synchronized mode with pads on the chest and sedation medication drawn up at the bedside. — Synchronized cardioversion must happen within minutes if the patient shows instability signs, and having equipment ready prevents dangerous delays when seconds count for brain blood flow (perfusion) (blood flow).
5. Administer high-flow oxygen by non-rebreather mask to keep oxygen saturation above 94 percent. — The fast heart rate reduces cardiac output (blood pumped per minute) so tissues are already oxygen-starved, and maximizing blood oxygen content helps prevent starved blood flow (ischemia) (tissue damage from low oxygen) to the heart and brain.
6. Draw blood for troponin, electrolytes including potassium and magnesium, and thyroid studies before giving any medications. — Low potassium or magnesium can cause or worsen arrhythmias and must be replaced, elevated troponin (a heart enzyme) shows the heart muscle is dying, and hyperthyroidism (overactive thyroid) can trigger atrial fibrillation (an irregular, quivering heartbeat) requiring different treatment.
7. Attempt vagal maneuvers like bearing down or ice to the face only if the patient is stable with narrow-complex regular a fast heart rate (tachycardia). — Vagal maneuvers increase parasympathetic nervous system activity which can slow conduction through the AV node (the electrical gateway between upper and lower heart chambers) and break SVT, but must never delay cardioversion in unstable patients.
8. Document the exact time symptoms started, heart rate trends, and any interventions with the patient response every 2 to 5 minutes. — Rapid deterioration is common in unstable a fast heart rate (tachycardia), and precise documentation shows whether treatments are working or if the patient is progressing toward cardiac arrest requiring CPR and defibrillation.

Sources

- AHA/ACC 2023 ACLS guideline
- ESC 2019 guideline for the management of patients with supraventricular tachycardia
- AHA/ACC/HRS 2015 guideline for the management of adult patients with supraventricular tachycardia
- Cochrane review on emergency treatment of tachyarrhythmias

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