

Chapter 6: Ventricular Rhythms

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After this chapter you should be able to recognize premature ventricular complexes and their patterns; identify idioventricular and accelerated idioventricular rhythm; distinguish monomorphic from polymorphic ventricular tachycardia; and recognize ventricular fibrillation, asystole, and pulseless electrical activity. **Reading:** Aehlert, *ECGs Made Easy*, 7th ed., Chapter 6 (p. 151 to 179).

Note to students: Rates and cutoffs in this guide follow **Aehlert, 7th edition**, because that is the edition this guide follows. Other references disagree on some of these numbers, and that is normal rather than a sign that someone is wrong. Accelerated idioventricular rhythm is the clearest example: this guide and your textbook use 41 to 100, while published ranges elsewhere run from 40 to 120. When a practice question online conflicts with this guide, answer from the textbook. **What the last chapter gives you:** junctional rhythms taught you the escape hierarchy and the habit of asking one question about QRS width. This chapter is that same pattern one level further down, so most of it is recognition rather than new material.

At a Glance: this chapter in one box

Numbers you must know

- Wide QRS is **0.12 s or more** (3 small boxes or more).
- Idioventricular (ventricular escape) **20 to 40**; accelerated idioventricular **41 to 100**; ventricular tachycardia **over 100**.
- Ventricular fibrillation: **coarse** is 3 mm or taller, **fine** is under 3 mm.
- Roughly **4 out of 5** wide-complex tachycardias are ventricular tachycardia.

The one rule this chapter is built on

- A rhythm starting **above** the bundle branch bifurcation rides the fast bundles, so the QRS is **narrow**. Starting **below** it, the impulse is too late to get on, so it crawls muscle cell to muscle cell and the QRS is **wide**.

Top 3 traps

- Assuming wide always means ventricular, and that ventricular is always wide. Neither holds.
- Believing a stable, talking patient cannot be in ventricular tachycardia.
- Suppressing an escape rhythm. It may be the only thing perfusing the patient.

You Already Know This: The Last Chapter, One Level Down

This chapter is the junctional chapter moved one level further down the conduction system. Same escape hierarchy, same three-speed ladder, same single question about QRS width. Learn the parallel and you already know half of what follows.

	Junctional (last chapter)	Ventricular (this chapter)
Escape	Junctional escape, 40 to 60	Idioventricular, 20 to 40
Accelerated	Accelerated junctional, 61 to 100	AIVR, 41 to 100
Tachycardia	Junctional tachycardia, 101 to 180	Ventricular tachycardia, over 100
QRS	Narrow , it used the fast track	Wide , it took the detour
P waves	Inverted, buried, or retrograde	Absent or dissociated

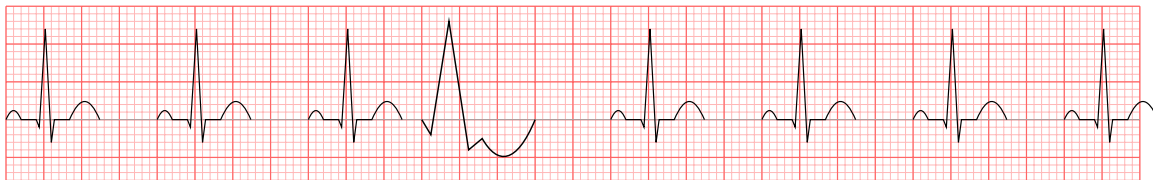
Why Wide Means Ventricular

The His-Purkinje system is a highway. It carries the impulse across both ventricles quickly and evenly, and that is why a normal QRS is narrow. The on-ramp to that highway is the bundle branch bifurcation.

A beat that starts **above** the bifurcation gets on the highway, so it is narrow. That is why atrial rhythms from Chapter 4 and junctional rhythms from Chapter 5 are both narrow, despite starting in completely different places. A beat that starts **below** the bifurcation has missed the on-ramp. It has to travel muscle cell to muscle cell on the surface streets instead. Two things follow, and between them they explain this entire chapter: the trip takes **longer**, which is the wide QRS, and it travels in a **different direction** than usual, which is the bizarre shape and the T wave pointing opposite the QRS.

Premature Ventricular Complex (PVC)

★ sinus rhythm at 75 with one PVC



Regularity	Underlying rhythm is regular; the PVC interrupts it
Rate	Underlying rate is unaffected
P waves	None before the PVC ; the sinus P wave may be hidden inside it
PR	Not measurable on the PVC
QRS	0.12 s or more , wide and bizarre, T wave opposite

Causes Ischemia, electrolyte disturbance (low potassium, low magnesium), stimulants including caffeine and nicotine, hypoxia, heart failure, and structurally normal hearts.

Patient A PVC arrives early, so the ventricle had less filling time and ejects less blood. That beat may produce a weak pulse or none at all, which is why a monitor rate and a radial pulse can disagree. Count an apical rate.

Memory hook The highway and the surface streets. The impulse missed the on-ramp, so it crawls through muscle instead of riding the bundles. Longer trip, wider complex.

Fast tell The T wave points the *opposite* way from the QRS. That discordance is a free second confirmation on the same beat.

Where students go wrong Naming the pattern instead of the rhythm. Say “sinus rhythm with PVCs in bigeminy,” never just “bigeminy.” Bigeminy is a pattern layered on a rhythm, not a rhythm.

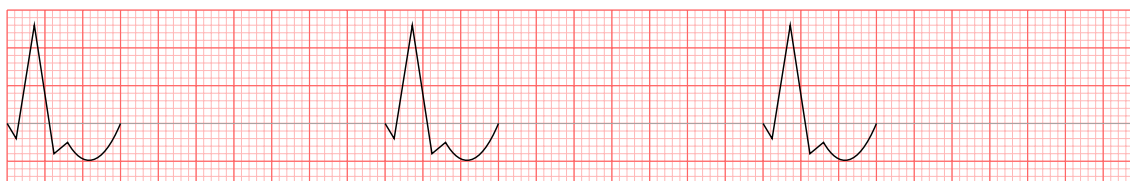
Patterns worth naming. The Latin prefix counts the beats in the repeating group: **bigeminy** is every second beat, **trigeminy** every third, **quadrigeminy** every fourth. Two PVCs in a row is a **couplet**, three or more is a **run**. Those sit on one continuous scale rather than in separate boxes, which is why a couplet is worth reporting: it signals increased irritability in a heart that could progress to a run of ventricular tachycardia.

The Three-Speed Ladder

The ventricles have their own intrinsic rate, exactly the way the junction does, and the same three speeds apply. The only difference is that everything below the bifurcation is wide.

Idioventricular Rhythm (Ventricular Escape)

★ the last pacemaker standing, 20 to 40



Wide, slow, regular, and no P waves anywhere. Rate 30. Each complex crawls muscle cell to muscle cell, which is why it takes 0.12 s or more and looks nothing like the narrow complexes above the bifurcation.

Regularity	Regular
Rate	20 to 40
P waves	None
PR	Not measurable
QRS	0.12 s or more, wide and bizarre

Causes Failure of every pacemaker above the ventricles. The SA node and the AV junction have both stopped or slowed below 20, and the His-Purkinje fibers are the last tissue left that can fire. Seen in complete heart block with a ventricular escape, in terminal rhythms, and briefly during reperfusion.

Patient Profoundly symptomatic. At 20 to 40 the cardiac output is marginal. Expect lightheadedness, hypotension, altered mental status, possibly syncope. This patient needs pacing (transcutaneous, then transvenous), not drugs. Atropine is unlikely to help because the problem is below the AV node.

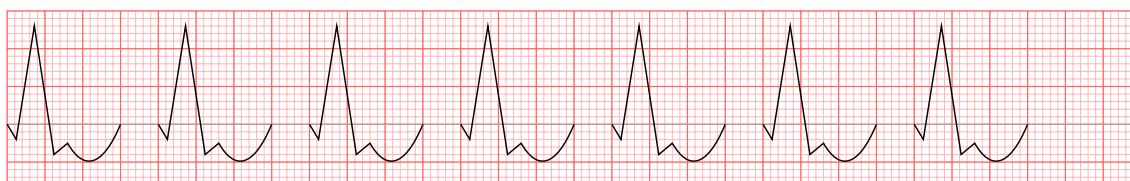
Memory hook The flashlight. The SA node is the power company, the junction is the building's backup generator, and the ventricles are the flashlight. It runs, but it was never meant to carry the load, and the output shows it.

Fast tell Wide, slow, regular, no P waves. If the rate is under 40 and every QRS is wide, you are looking at the bottom of the ladder.

Where students go wrong Suppressing it. This escape rhythm may be the only thing keeping the patient alive. Antiarrhythmics here can remove the last pacemaker and produce asystole. **Support the rate; do not abolish the rhythm.**

Accelerated Idioventricular Rhythm (AIVR)

★ the ventricle, accelerated but not yet tachycardic



Wide complexes at 75, regular, no P waves. The rate looks normal on the monitor, but the tissue producing it is firing at nearly double its own baseline. This is the ventricular parallel of accelerated junctional rhythm from the last chapter.

Regularity	Regular
Rate	41 to 100
P waves	None, or AV dissociation if the sinus node is still firing independently
PR	Not measurable
QRS	0.12 s or more, wide and bizarre

Causes Reperfusion after acute myocardial infarction is the signature cause and is worth naming every time this rhythm appears. Also digoxin toxicity, cocaine, and the post-resuscitation period.

Patient Usually stable and often transient. The rate is in the normal range, so the patient may feel nothing at all. The danger is not the rhythm; it is missing what the rhythm tells you. In the right clinical setting, AIVR is a sign that the occluded artery just opened.

Memory hook Same ladder, one level down. Idioventricular 20 to 40, AIVR 41 to 100, VT over 100. "Accelerated" describes the tissue, not the patient, exactly as it did in the junction.

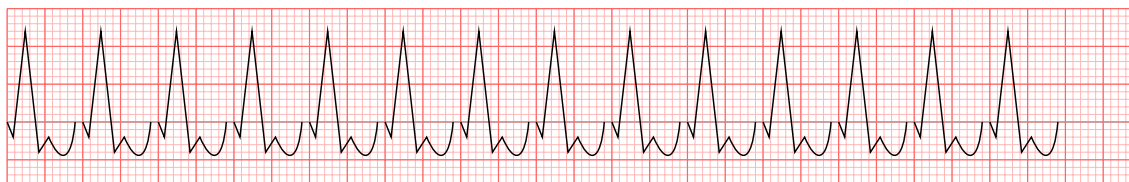
Fast tell Wide complexes at a normal-looking rate, regular, no P waves. If the patient just had a coronary artery reopened,

this is the rhythm you expect to see.

Where students go wrong Calling it VT because the QRS is wide. The rate separates them: under 100 is AIVR, over 100 is VT. And do not treat AIVR with antiarrhythmics. It is usually a sign that reperfusion worked, and suppressing it can remove a rhythm the heart is relying on.

Monomorphic Ventricular Tachycardia

★ one focus, one shape, every beat alike



Wide, fast, regular, and every complex is a copy of the last. Rate 150. No P waves are visible; the sinus node may still be firing independently somewhere underneath, but you cannot see it. "Monomorphic" means one shape, and that is the whole strip in two words.

Regularity Regular

Rate Over 100, typically 150 to 250

P waves Usually absent; AV dissociation may be present but is often invisible

PR Not measurable

QRS 0.12 s or more, wide and bizarre, **all the same shape**

Causes Prior myocardial infarction with scar tissue (the most common cause), acute ischemia, electrolyte disturbance (low potassium, low magnesium), antiarrhythmic drugs, and structurally normal hearts (idiopathic VT, which carries a better prognosis).

Patient Ranges from stable and talking to pulseless. A young patient with good ventricular function can hold a blood pressure in VT for a surprisingly long time. Assess the patient, but do not let a good blood pressure talk you out of the diagnosis. About **4 in 5** wide-complex tachycardias are VT.

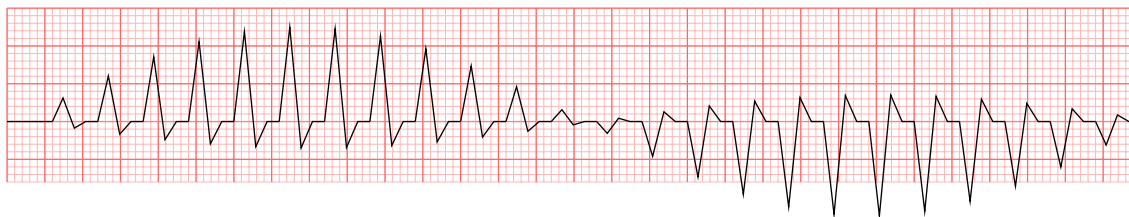
Memory hook "Monomorphic" means one shape. Every QRS looks like its neighbor because they all come from the same ventricular focus. That uniformity is the difference from polymorphic VT and from torsades, where the shape keeps changing.

Fast tell Wide, fast, regular, and every complex matches the last. If you can lay one QRS on top of the next and they overlap, it is monomorphic.

Where students go wrong Believing a stable, talking patient cannot be in VT. Clinical stability does not distinguish VT from supraventricular tachycardia with aberrancy. **If in doubt, treat it as VT.**

Torsades de Pointes

★ polymorphic VT with a prolonged QT



Regularity Irregular

Rate Typically 200 to 250 bpm

P waves None visible

PR Not measurable

QRS Wide, and **continuously changing** in amplitude and direction

Causes Anything that prolongs the QT interval: congenital long QT syndromes, drugs, and electrolyte disturbance (low potassium, low magnesium, low calcium).

Drug classes that prolong the QT Every one of them starts with "anti," so the whole list is a single alphabet run: Antiarrhythmics, Antibiotics, Antipsychotics, Antidepressants, Antiemetics, Antifungals.

Memory hook The name is the instruction. *Torsades de pointes* means "twisting of the points." On the strip it narrows to a waist and then flares out again, like a party streamer taped at both ends.

Fast tell Look for the short-long-short beat sequence just before it starts, and for a prolonged QT on the strip that came before. Torsades is a rhythm you can often see coming.

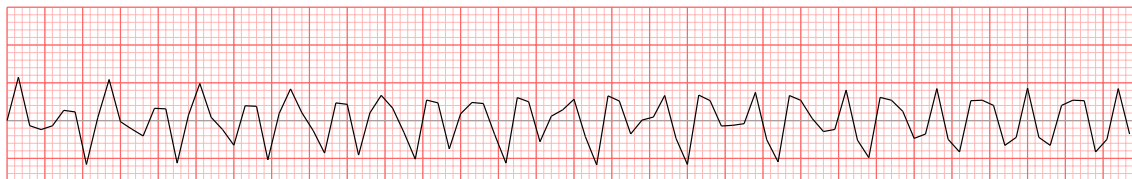
Where students go wrong Calling every twisting rhythm torsades. **All torsades is polymorphic VT, but not all polymorphic VT is torsades.** Torsades additionally requires a prolonged QT. The most common cause of non-torsades polymorphic VT is ischemia.

The Three Arrest Rhythms

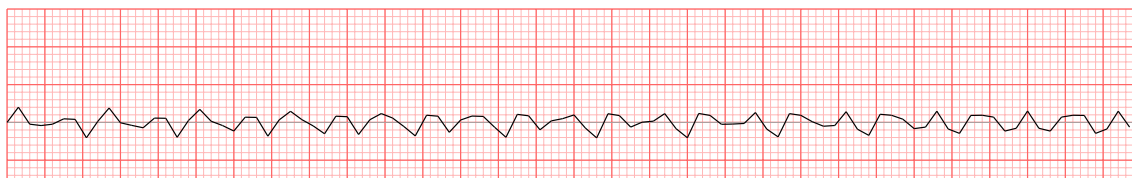
Ventricular fibrillation, asystole, and pulseless electrical activity are the three rhythms of cardiac arrest. They share one clinical fact: **no pulse, no output, the patient is in arrest**. They differ in what the monitor shows, and that difference drives the treatment.

Ventricular Fibrillation

★ the terminal rhythm, no output, no pulse



Coarse VF. Grossly irregular waves, 3 mm or taller, with no identifiable QRS complex and no flat baseline. The ventricles are quivering, not contracting.



Fine VF. Low-amplitude waves under 3 mm. Easily mistaken for asystole, which is why you check the gain and the leads before calling a flat line.

Regularity	Chaotic , no measurable regularity
Rate	Not measurable ; no identifiable QRS
P waves	None
PR	Not measurable
QRS	None identifiable ; coarse waves 3 mm or taller, fine waves under 3 mm

Causes VT degenerating into VF (the most common pathway), acute myocardial infarction, severe electrolyte disturbance, hypoxia, electrocution. VF is the most frequent initial rhythm in witnessed sudden cardiac arrest.

Patient No cardiac output, no pulse, unconscious. This is cardiac arrest. CPR immediately, defibrillation as soon as the pads are on. Every minute of untreated VF costs roughly 7 to 10 percent survival.

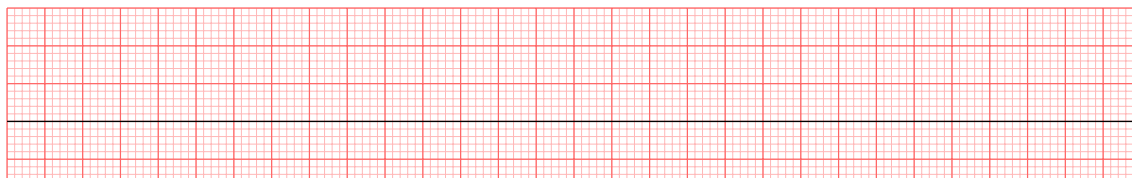
Memory hook The ventricles are quivering, not pumping. Think of a bag of worms. No organized contraction means no output, regardless of how much electrical activity the monitor shows.

Fast tell Coarse VF has big, grossly irregular waves. Fine VF is low amplitude and can look almost flat. Neither has identifiable QRS complexes, and neither has a flat baseline (that would be asystole).

Where students go wrong Treating coarse and fine differently. **Both get CPR and defibrillation.** Coarse versus fine is prognostic vocabulary (coarse is more likely to defibrillate successfully), not a treatment fork.

Asystole

★ the flat line



No waves, no complexes, no electrical activity. A flat tracing in two leads. Before calling it, confirm the leads are connected and the gain is up, because fine VF and a disconnected cable can both look like this.

Regularity	None
Rate	None
P waves	None
PR	Not measurable
QRS	None

Causes End-stage cardiac disease, prolonged arrest (VF that has exhausted the myocardium), severe hypoxia, massive pulmonary embolism, profound hyperkalemia. Asystole is often what is left after VF has run its course without treatment.

Patient No pulse, no output. Confirm in **two leads** before calling it. If it is truly asystole, the treatment is CPR, epinephrine, and finding the reversible cause. **Asystole is not a shockable rhythm.**

Memory hook Flat line. No electricity, no contraction, no pulse. The heart has stopped.

Fast tell A flat tracing in two leads. Check lead connections and gain before you call it, because fine VF can mimic asystole and you do not want to miss a shockable rhythm.

Where students go wrong Shocking asystole. You cannot defibrillate a flat line; there is no organized activity to reset. The treatment is CPR and epinephrine, not electricity. **If in doubt between fine VF and asystole, treat it as VF and shock.**

Pulseless Electrical Activity (PEA)

★ the strip lies; the patient has no pulse

*No strip is shown, by design. PEA cannot be identified from a monitor tracing. The strip shows organized electrical activity that **should** produce a pulse. The patient has no pulse. The diagnosis is the gap between those two facts.*

Regularity Whatever the underlying rhythm shows

Rate Whatever the underlying rhythm shows

P waves Present or absent, depending on the rhythm

PR Depends on the rhythm

QRS Can be narrow or wide

Causes The **H's and T's**: Hypovolemia, Hypoxia, Hydrogen ion (acidosis), Hypo-/Hyperkalemia, Hypothermia; Tension pneumothorax, Tamponade (cardiac), Toxins, Thrombosis (pulmonary), Thrombosis (coronary).

Patient No pulse despite organized electrical activity. The monitor shows a rhythm that looks like it should be perfusing, but the heart is not pumping. This is cardiac arrest. CPR, epinephrine, and **run the H's and T's** to find the reversible cause.

Memory hook PEA is not a rhythm; it is a clinical diagnosis. The strip shows organized electricity. The patient has no pulse. The two facts together are the diagnosis, and you cannot make it from the strip alone.

Fast tell Any organized rhythm on the monitor in a patient without a pulse. The strip is a red herring by design; it looks like it should be working.

Where students go wrong Looking for PEA on the strip. You will not find it there. A student who says "the rhythm is PEA" based on the monitor alone has not checked a pulse. **PEA is what you diagnose when the monitor says one thing and the patient says another.**

Common Trap

A wide QRS always means the beat started in the ventricle. True or false?

False, and it fails in both directions. Bundle branch block, supraventricular tachycardia with aberrancy, paced rhythms, and hyperkalemia all widen the QRS without a ventricular origin. Going the other way, roughly one in five ventricular tachycardias can be narrow, when the focus sits close enough to the conduction system to get on the highway anyway.

The honest version of the rule: **a wide QRS tells you the impulse did not use the normal fast pathway. It does not by itself tell you why.**

Common Trap

Your patient is in a wide-complex tachycardia at 170, alert, talking to you, blood pressure 118/70. Does being stable rule out ventricular tachycardia?

No, and this is the most dangerous belief in this chapter. Clinical stability does not distinguish ventricular tachycardia from supraventricular tachycardia with aberrancy. A young patient with good ventricular function can hold a blood pressure in VT for a surprisingly long time.

About four out of five wide-complex tachycardias are VT, and the published criteria can rule VT *in* but never *out*. **If in doubt, treat it as VT.**

Common Trap

Your patient's idioventricular rhythm is running at 30. The resident orders lidocaine to "suppress the ectopy." What is wrong with this picture?

The rhythm is not ectopy. It is an **escape**. The SA node and the AV junction have both failed, and the ventricles are the last pacemaker standing. Lidocaine suppresses ventricular automaticity, which means it will suppress the only rhythm keeping this patient alive.

The rule from the junctional chapter carries straight across: **an escape rhythm is the answer to a problem happening higher up, not the problem itself.** You support the rate (pacing, atropine for the junction), you correct the cause, and you leave the escape alone.

Before You Move On

You should now be able to:

- apply the bifurcation rule to explain why every rhythm in this chapter is wide
- state the ventricular ladder from idioventricular through accelerated idioventricular to ventricular tachycardia
- separate monomorphic VT from polymorphic VT, and torsades from other polymorphic VT by the QT interval
- explain why a stable, talking patient does not rule out ventricular tachycardia
- distinguish coarse from fine ventricular fibrillation and say why the distinction does not change treatment
- explain why asystole is not a shockable rhythm
- recognize that pulseless electrical activity cannot be identified from a strip at all

The next chapter changes the question. Until now you have asked where a beat started. From here you ask whether it got through.