

ECG Monitoring and Arrhythmia Analysis

Every rhythm is a statement about where an impulse started and how it traveled — read in the same five steps, every time

No matter the etiology or type of arrhythmia — treat the patient and the symptoms, not the arrhythmia.
Aligned to Region SOP

Six questions, asked in the same order, every time

1 Rate

Bradycardia under 60, tachycardia over 100. Count it the same way every time.

2 Rhythm

Regular, occasionally irregular, regularly irregular, or irregularly irregular.

3 P waves

Present? Regular? One per QRS? Upright or inverted? Do they all look alike?

4 PR interval

Normal is 0.12–0.20 s. Any deviation is an abnormal finding.

5 QRS complex

Do they all look alike? What is the duration? Normal is 0.04–0.12 s.

6 And then: the patient

Stable or unstable? The rhythm name never decides treatment on its own.

Narrow or wide? That question sorts most strips before you have named anything

An impulse starting at or above the AV junction runs down the normal His–Purkinje system — fast, coordinated, NARROW QRS. An impulse starting below it spreads cell to cell through muscle — slow, disorganized, WIDE and bizarre QRS. Site of origin is written in the width.

The chapter sorts arrhythmias by site of origin — and so does this deck

SA node

Bradycardia, tachycardia, arrhythmia, arrest, block, pause, sick sinus.

Atria

Wandering pacemaker, MAT, PACs, SVT, flutter, fibrillation.

AV junction

The blocks, PJs, junctional escape and accelerated rhythms.

Ventricles

Escape, AIVR, PVCs, VT, torsades, VF, asystole.

Conduction

Aberrancy, bundle branch block, WPW — plus electrolytes and hypothermia.

CASE STUDY

Read now. We solve it at the end.

Dispatch

Weakness and dizziness, 1420 hours.

On arrival

A 58-year-old woman, two days of feeling faint, worse today. She nearly passed out walking to the kitchen. No chest pain. She takes lisinopril and nothing else new.

She is pale and cool. HR 38, BP 82/50, RR 18, SpO₂ 96%. Lungs clear. Alert but says she feels “far away.”

The monitor shows a regular atrial rate near 75 with upright P waves. The PR interval on every conducted beat is identical at 0.18 s. Every second or third P wave has no QRS after it. The QRS complexes that do appear are 0.14 s wide.

Your partner draws up atropine.

Hold these questions:

- 1 Walk the five steps. What is the rhythm?
- 2 The PR intervals are all identical. Which block does that rule out?
- 3 The QRS is 0.14 s. What does that tell you about where the block sits?
- 4 Should your partner push the atropine? Why or why not?

SECTION 1

ECG Monitoring

What the machine records, what the paper means — and what the ECG cannot tell you.

What it is · Leads · Paper and rate · Waves and intervals

What the ECG records — and what it cannot tell you

1 It records electrical activity only

“It tells you NOTHING about the heart’s pumping ability.” That clause is the whole basis of pulseless electrical activity — and the reason you check a pulse, not a screen.

2 Up, down, and flat

Positive impulses deflect UPWARD, negative DOWNWARD, and the absence of impulse gives the ISOELECTRIC line — the baseline everything is measured from.

3 Toward the positive electrode is upward

Which is why the same beat looks different in different leads. Lead placement changes the picture without changing the patient.

4 Artifact is anything not from the heart

Movement, shivering, muscle tremor, loose or dry electrodes, a disconnected cable, electrical interference, CPR. Mostly a preparation problem, not a machine problem.

When the trace looks impossible, look at the patient before you treat the strip

A patient who is talking to you is not in ventricular fibrillation. Artifact that mimics a lethal rhythm is common enough to have caused real harm — and the fix is almost always dry skin, electrodes over bone rather than muscle, and secured cables.

Dry the skin

Sweat breaks contact. Wipe and, if needed, abrade lightly.

Clip, do not shave

Hair lifts the electrode off the skin.

Bone, not muscle

Place over bony prominences — muscle adds tremor.

Secure the cables

A swinging lead wire writes its own rhythm.

REGION SOP Every cardiac protocol opens the same way: “Obtain ECG and 12 lead, transmit to receiving facility, and monitor capnography.” The strip is data — the branch point is the patient.

Leads — a pair of electrodes and a point of view

Lead group	What it is	Which leads	Plane
Bipolar limb	One negative and one positive electrode	I, II, III — Einthoven's triangle	Frontal
Augmented (unipolar)	Two negative bipolar leads combined to create an axis	aVR, aVL, aVF — “a” augmented, “V” unipolar, then the extremity	Frontal
Precordial (unipolar)	Electrodes across the chest surface	V1–V6	HORIZONTAL

1 Lead II is the monitoring lead

Positive electrode at the apex, negative below the right clavicle, ground on the left. Depolarization travels from the SA node down and to the LEFT — toward that positive electrode — so P waves and QRS complexes stand up clearly.

2 MCL1 is the other one you will see

It approximates V1, and is better for telling right from left bundle branch patterns, and ventricular beats from supraventricular beats conducted aberrantly.

3 A monitoring lead reads RHYTHM, not infarction

One viewpoint, and ischemia is regional. That limitation is the entire argument for the 12-lead in Part 5.

A lead is a point of view, not a wire

It is created by the relationship between a positive and a negative pole — which is why the same beat looks different in every lead, and why “change the lead” is a legitimate troubleshooting step when a rhythm is unclear.

The paper — and three ways to count a rate

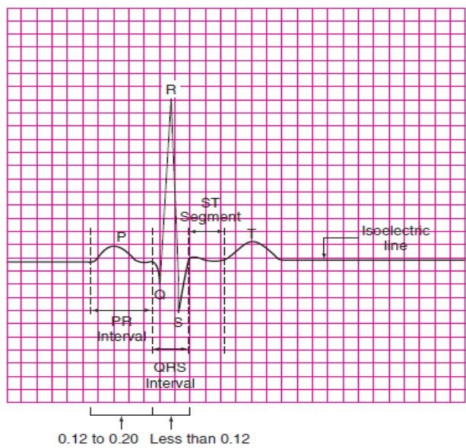


Figure 33-28 The ECG waveform on graph paper.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 25 mm per second

The fixed paper speed is what converts horizontal distance into time.

2 Small box = 0.04 s

Light lines 1 mm apart. Five small boxes make one large box.

3 Large box = 0.20 s

Heavy lines 5 mm apart, enclosing 25 small squares. Five large boxes = 1 second.

4 Two large boxes = 1 mV

The vertical axis is voltage. Marks at the top sit every 3 seconds — 15 large boxes.

Method	How	Use it when
Six-second	Count QRS complexes between two 3-second marks and multiply by 10	ANY rhythm — and the only honest method for an irregular one
300 method	$300 \div$ the number of large boxes between two R waves. Memorize 300, 150, 100, 75, 60, 50	Regular rhythms — fast and accurate
1500 method	$1500 \div$ the number of small boxes between two R waves	Regular rhythms, when you need precision

When P waves and QRS complexes are not one-to-one, count the ATRIAL and VENTRICULAR rates separately. In complete heart block the atria may run at 80 and the ventricles at 35 — a single number hides the diagnosis.

Waves and intervals — the measurements that name the rhythm

P wave

Atrial depolarization.

QRS complex

Ventricular depolarization.

T wave

Ventricular repolarization.

U wave

Occasional — may signal electrolyte abnormality, classically hypokalemia, or be normal.

Interval	What it measures	Normal	What abnormal tells you
PR	Atria to ventricles — the AV node delay	0.12–0.20 s	LONG: delayed AV conduction — first-degree block, or Type I lengthening. SHORT: junctional origin, or an accessory pathway bypassing the node (WPW)
QRS	Time for ventricular depolarization	0.04–0.12 s	WIDE: the impulse did not use the normal system — ventricular origin, bundle branch block, or aberrant conduction
ST segment	Between depolarization and repolarization	Isoelectric	Depression or T inversion with ischemia; elevation with injury; a Q wave can indicate infarction — all of Part 5
QT	Total ventricular depolarization and repolarization	0.33–0.42 s	Inverse to rate, so it is corrected (QTc). PROLONGED QT carries an increased risk of ventricular arrhythmia and sudden death — torsades

Measure — do not eyeball. Estimating intervals misclassifies first-degree block, and misses a wide QRS at exactly the moment width matters most.

Which leads look at which surface

Surface	Leads	Artery, usually	What else that artery feeds — so what to expect
ANTERIOR	V1–V4	Left anterior descending	The largest territory and most of the septum — expect pump failure and cardiogenic shock
LATERAL	I and aVL	Circumflex	Often extends from an anterior or inferior pattern
INFERIOR	II, III and aVF	Right coronary artery	The right ventricle, and in most hearts the SA and AV nodes — expect bradycardia, AV block, and the right ventricular question

Say the surface, then the artery, then what else that artery feeds

That three-step habit converts a pattern into a prediction. Inferior means RCA, and RCA means the nodes and the right ventricle — which is why an inferior infarct arrives bradycardic and why a venodilator can be the wrong drug.

A correction worth making out loud

Some printed material lists the inferior leads as “II, III and IV.” There is no lead IV. It is II, III and aVF.

Rhythm versus territory

A monitoring lead answers what the impulse is doing. Lead-to-surface mapping answers where the muscle is hurt. Same strip, two different questions.

REGION SOP ACS: “If positive ST elevation then contact STEMI receiving center and call Alert at earliest possible time.” Territory drives destination.

SECTION 2

The Method

One procedure, applied to every strip — including the ones you already recognize.

Five steps · Normal sinus rhythm · How arrhythmias arise

The five-step analysis — the standard in EMS

1

Rate

Under 60 is bradycardia; over 100 is tachycardia. Count atrial and ventricular rates SEPARATELY when they differ.

2

Rhythm

OCCASIONALLY irregular — a few intervals. REGULARLY irregular — a repeating pattern. IRREGULARLY irregular — no relationship at all.

3

P waves

Present? Regular? One per QRS? Upright or inverted? All alike? Each answer locates the pacemaker.

4

PR interval

0.12–0.20 s is normal. Constant, progressively lengthening, or variable — that answer separates the AV blocks.

5

QRS

All alike? What duration? Narrow means it used the normal system. Wide means it did not.

What you see	What it means
No P waves at all	The impulse did not start in the atria — junctional or ventricular, or the atria are fibrillating
INVERTED P waves	Retrograde atrial depolarization — a junctional origin firing backward into the atria
P waves that vary in shape	The pacemaker is wandering between sites
More P waves than QRS complexes	A block — and the PR pattern tells you which one
IRREGULARLY irregular, no P waves	Atrial fibrillation until proven otherwise

Then the sixth question, which no strip can answer: is the patient tolerating it? “Treat the patient and the symptoms, not the arrhythmia.”

Normal sinus rhythm, and the three ways a rhythm goes wrong

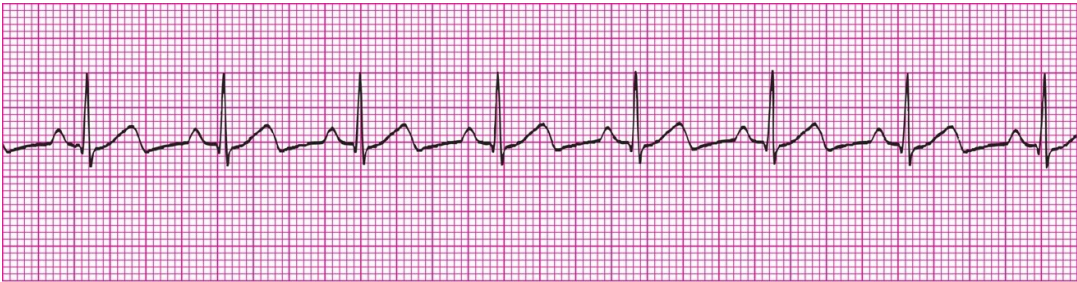


Figure 33-30 Normal sinus rhythm.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

Normal sinus rhythm	Value
Rate	60–100
Rhythm	Regular
P waves	Normal and upright, one per QRS
PR interval	0.12–0.20 s
QRS	Less than 0.12 s

1 Enhanced automaticity

A patch of myocardium becomes irritable enough to fire before the sinus node gets there. Driven by ischemia, catecholamines, electrolytes, hypoxia, stimulants — the mechanism behind most premature beats.

2 Reentry

Conduction slows in one path and blocks in another, so an impulse that should have died finds a way back into tissue that has already repolarized — and circles. Atrial flutter, most SVT, and the circuits in VF.

3 Escape

The opposite of the other two. A higher pacemaker fails or slows, and a lower one takes over at its own intrinsic rate. The fail-safe hierarchy from Part 1.

Ectopic beats come EARLY. Escape beats come LATE, after a pause. Ectopy is irritability and may need suppressing; escape is rescue and must never be — the difference is timing, and it is a clinical decision, not a classification exercise.

SECTION 3

The Rhythms

Sorted by where the impulse starts — because that is what the five steps actually determine.

SA node · Atria · AV junction · Ventricles · Conduction

Sinus bradycardia and sinus tachycardia

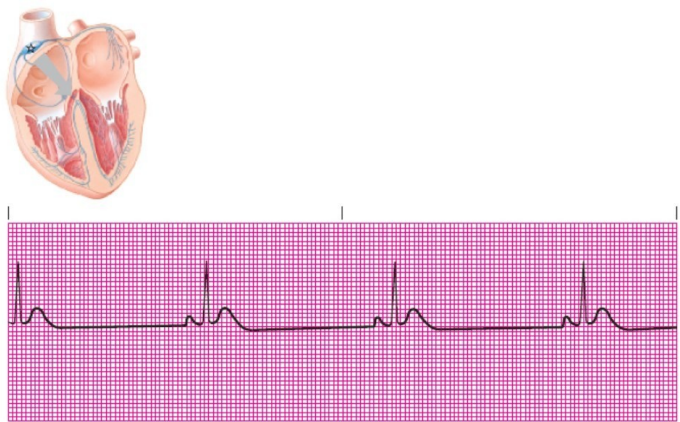


Figure 33-31 Sinus bradycardia.

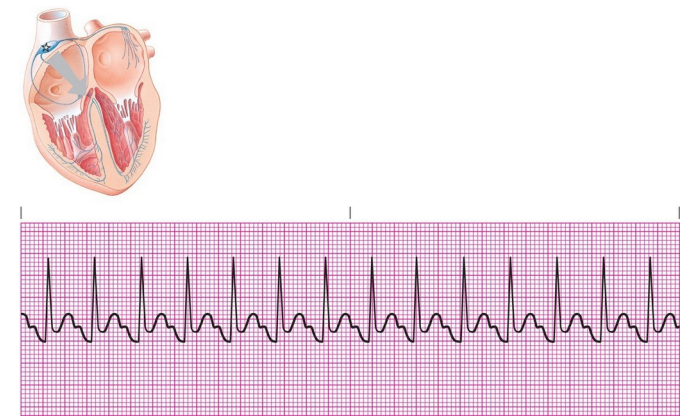


Figure 33-32 Sinus tachycardia.

	SINUS BRADYCARDIA	SINUS TACHYCARDIA
Rate	Less than 60	Greater than 100
Rhythm	Regular	Regular
P waves	Upright, normal morphology	Upright, normal morphology
PR / QRS	Both normal	Both normal
Causes	Vagal tone · inferior MI · beta and calcium channel blockers · hypothermia · raised ICP · suctioning	Hypovolemia · hypoxia · fever · pain · anxiety · stimulants · PE · shock of any cause
The point	May be NORMAL in athletes — a rate in the 40s with a normal pressure is a finding, not a problem	Often a COMPENSATORY mechanism for decreased stroke volume. Treat the cause, not the number

Sinus tachycardia in an adult rarely exceeds about 150–160 — a regular narrow tachycardia at 180 with no visible P waves is far more likely SVT.

When the sinus node hesitates — arrhythmia, arrest, block, pause

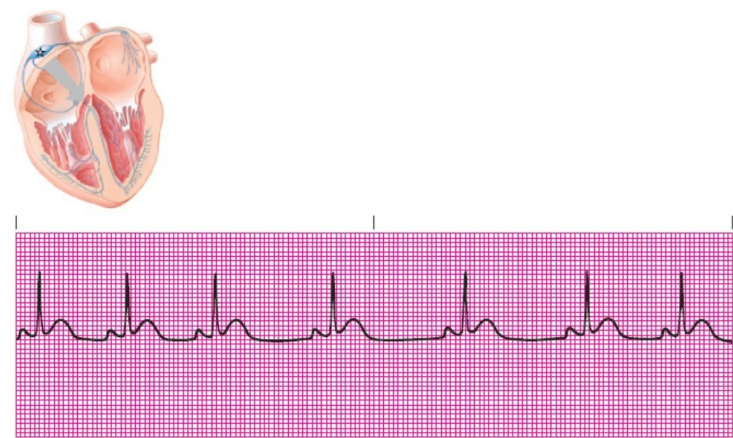


Figure 33-33 Sinus arrhythmia.

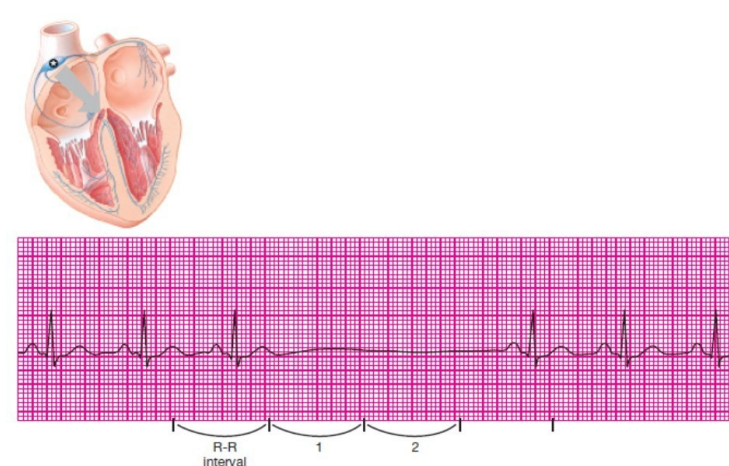


Figure 33-34 Sinus arrest.

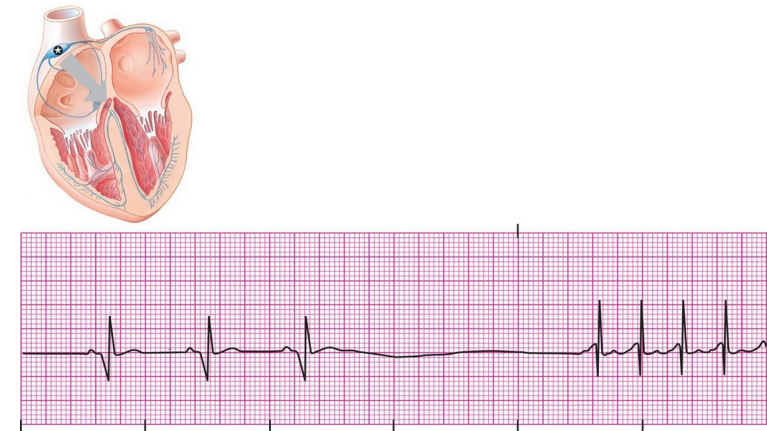


Figure 33-37 Sick sinus syndrome.

Rhythm	What the node did	Rhythm regularity	Significance
Sinus arrhythmia	Fired normally, at a rate varying with respiration	Irregular	A NORMAL variant with no clinical significance — common in the young
Sinus arrest	Failed to discharge, giving a period of standstill	Irregular	Prolonged or frequent episodes reduce output — but an escape rhythm typically develops
Sinus block	Fired on time; the impulse was blocked before leaving the node	Regular — the underlying timing is preserved, so the pause is a MULTIPLE of the R-R	Same as arrest: watch the pause length and the escape
Sinus pause	Failed to discharge, missing a complex	Irregular	Same — the question is how long, and how the patient responds
Sick sinus syndrome	A diseased node doing all of the above	Varies	Alternating bradycardia and tachycardia, pauses and arrest — often why the patient has a pacemaker

Arrest, block and pause are commonly confused, and prehospital management does not differ. What matters is how long the pause is, whether an escape rhythm appears, and whether the patient is symptomatic.

Wandering atrial pacemaker and multifocal atrial tachycardia

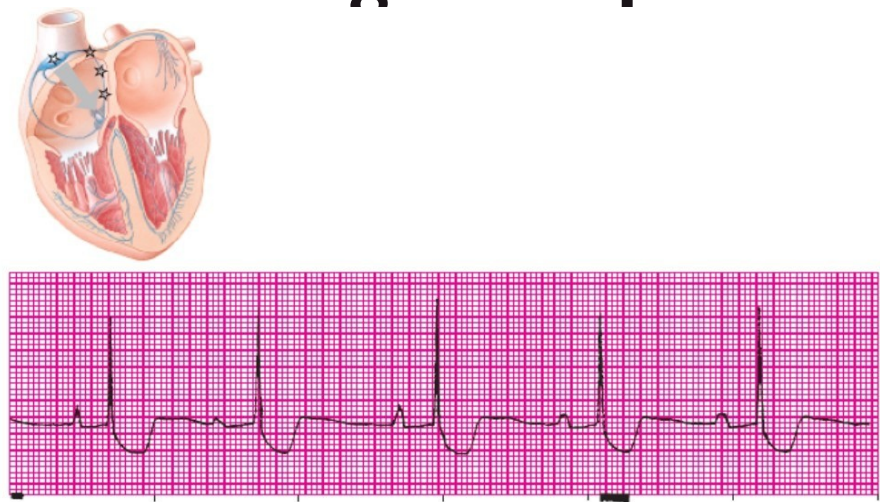


Figure 33-38 Wandering atrial pacemaker.

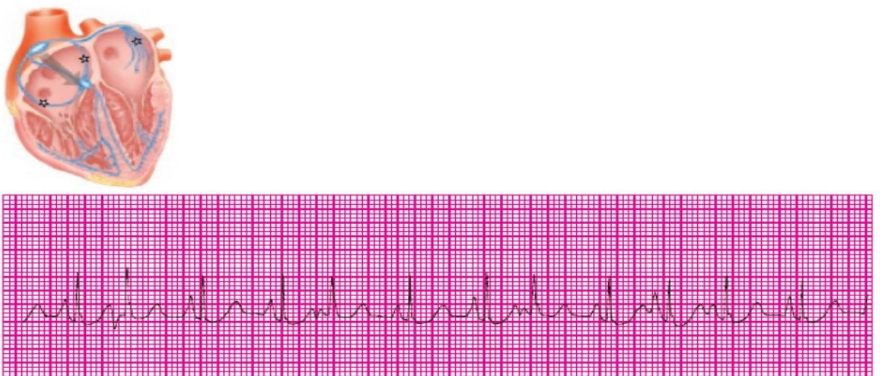


Figure 33-39 Multifocal atrial tachycardia.

	WANDERING ATRIAL PACEMAKER	MULTIFOCAL ATRIAL TACHYCARDIA
Mechanism	Passive transfer of the pacemaker site from the SA node to latent sites in the atria and AV junction	The same wandering pacemaker — at a rate greater than 100
Rate	Usually normal	Greater than 100
P waves	At least three different shapes in the same lead — because the site keeps moving	The same varying morphologies, faster
PR	Varies with the site	Varies
Significance	“Has no detrimental effects” — seen in the young, in athletes and in the elderly	“Usually seen in ACUTELY ILL patients” — severe COPD, PE, sepsis, theophylline toxicity, electrolyte derangement

MAT is routinely mistaken for atrial fibrillation — both are irregular and fast. MAT has VISIBLE P waves of three or more shapes; AF has none.

Premature atrial contractions and supraventricular tachycardia

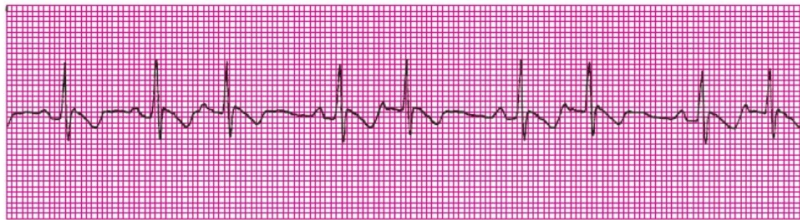


Figure 33-40 Premature atrial contraction.

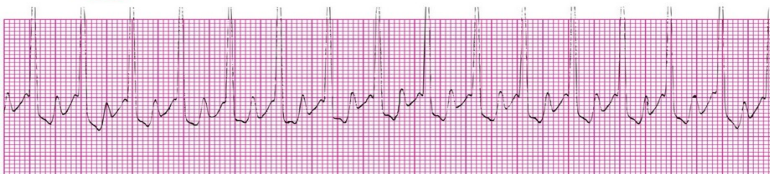
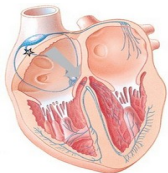


Figure 33-42 Supraventricular tachycardia.

1 PAC — one early beat from outside the SA node

The P wave of the early beat is a DIFFERENT SHAPE, because it started somewhere else. QRS usually normal — the impulse still uses the normal system.

2 Frequent PACs mean atrial irritability

Caffeine, stress, hypoxia, ischemia, electrolytes, stimulants. In a susceptible patient they can precede flutter or fibrillation.

3 SVT — rapid depolarization overriding the SA node

Usually a reentry circuit at the AV node. 150–250, relentlessly regular, P waves buried in the preceding T wave, QRS narrow.

4 Sinus tach or SVT? Get this right

Sinus: visible upright P waves, rarely over 150–160, varies, has a cause. SVT: faster, unvarying, no visible P waves, starts and stops abruptly like a switch.

REGION SOP SVT — STABLE: vagal maneuver, then ADENOSINE 6 mg, then 12 mg at 2 min. UNSTABLE: MIDAZOLAM, then cardioversion. Pearl: risk of sustained asystole after adenosine.

Atrial flutter and atrial fibrillation



Figure 33-43 Atrial flutter.

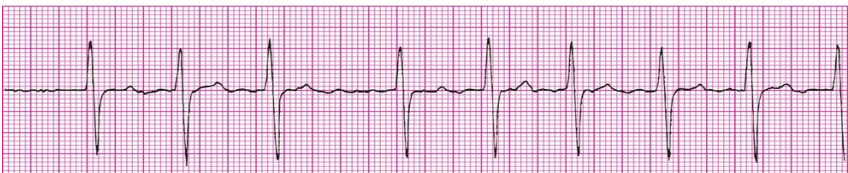


Figure 33-44 Atrial fibrillation.

A regular narrow tachycardia at almost exactly 150 is 2:1 flutter until you have looked for sawtooth waves.

	ATRIAL FLUTTER	ATRIAL FIBRILLATION
Mechanism	A rapid atrial reentry CIRCUIT, with an AV node that cannot conduct every impulse	MULTIPLE areas of reentry, or multiple ectopic foci bombarding the AV node
Atrial rate	250–350	350–750 — cannot be counted
Ventricular rate	Varies with the conduction ratio — 2:1, 3:1, 4:1	Varies greatly
Rhythm	Often regular	IRREGULARLY IRREGULAR
Baseline	Sawtooth flutter waves	No P waves — a fibrillating baseline

REGION SOP Rapid A-flutter/fib — UNSTABLE: MIDAZOLAM, then cardioversion. STABLE: vagal maneuver, then VERAPAMIL 5 mg over 5 min, repeat at 15 min if SBP ≥ 90, max 10 mg. Pearl: STROKE RISK before cardioversion.

First-degree block and second-degree Type I

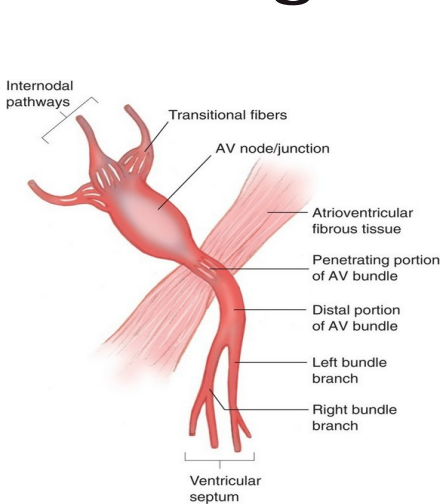


Figure 33-45 The AV node.
Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

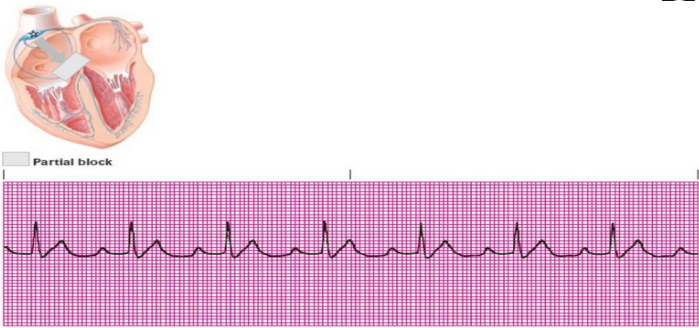


Figure 33-46 First-degree AV block.



Figure 33-47 Type I second-degree block.

	FIRST DEGREE	SECOND DEGREE TYPE I
What it is	A DELAY at the AV node, not an actual block — nothing is dropped	The node fatigues progressively until it fails to conduct one beat
PR interval	Greater than 0.20 s and CONSTANT	Lengthens progressively, then a P wave drops — and the cycle resets
Rhythm	Follows the underlying rhythm	Atrial regular, VENTRICULAR IRREGULAR — grouped beating
QRS	Normal	Usually narrow — the problem is above the bundle branches
Significance	"Usually no danger in itself"	Often transient, drug-related, and responsive to atropine

March out the P waves first — then look at what the PR interval is doing

Regular P waves with a CHANGING PR interval is Type I. A CONSTANT PR interval with dropped beats is Type II — a different problem, sitting lower in the conduction system.

Type II and third-degree block



Figure 33-48 Second-degree AV block, Type II.

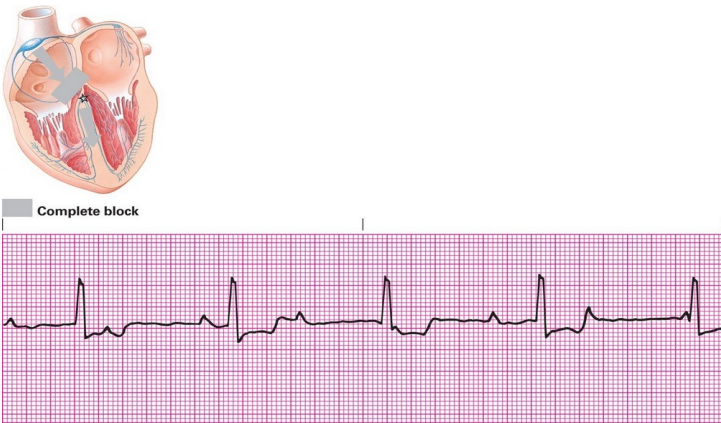


Figure 33-50 Third-degree AV block.

	TYPE II	THIRD DEGREE
What it is	Beats dropped without warning — usually BELOW the node, in the bundle branches	NO conduction between atria and ventricles at all
PR interval	CONSTANT on conducted beats	No relationship — P waves and QRS march independently
QRS	Often wide — the block is low	Narrow if the escape is junctional; wide if it is ventricular
Rate	Ventricular usually bradycardic	40–60 junctional escape; UNDER 40 if the pacemaker is lower
Significance	Structural, not vagal. Progresses to complete block without warning — this is the pacemaker one	Severely compromises output through rate AND loss of the atrial kick

2:1 conduction cannot be classified from the strip

Never two consecutive conducted beats, so you cannot see whether the PR was lengthening. Treat it as the more dangerous one.

REGION SOP Bradycardia NOTE: “Do not administer ATROPINE for 2nd Degree Type 2 or 3rd Degree Heart Block — skip straight to Transcutaneous pacing.” Atropine blocks vagal input at the nodes; a block below the node is not vagal, and speeding the atria can worsen the ratio.

Junctional beats and rhythms — the backup pacemaker at work

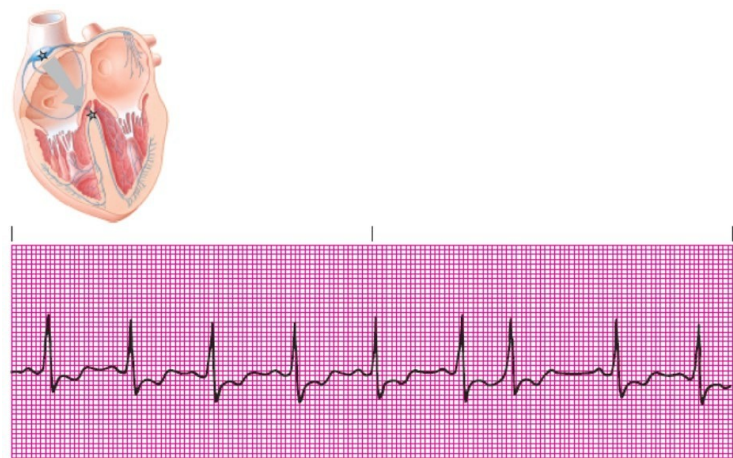


Figure 33-51 Premature junctional contraction.

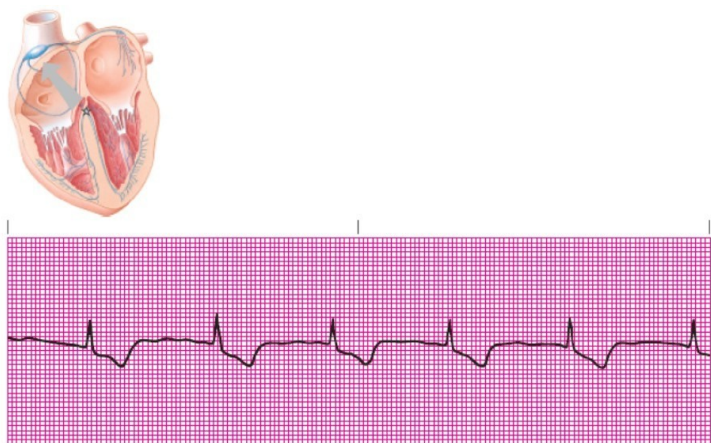


Figure 33-52 Junctional escape rhythm.

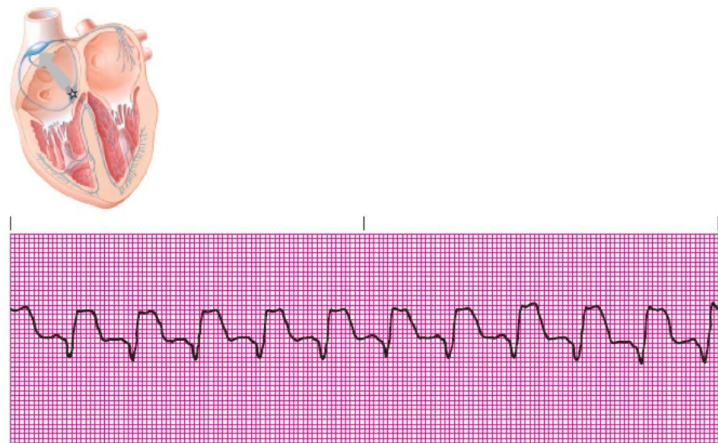


Figure 33-54 Accelerated junctional.

Rhythm	Rate	Mechanism	What to do about it
Premature junctional contraction	Underlying rhythm	A single junctional impulse arriving EARLY	Usually benign; frequent PJC's suggest junctional irritability
Junctional escape rhythm	40–60	The sinus node has slowed or failed, and the junction takes over at its intrinsic rate	It is RESCUE. Do not suppress it — treat symptoms and find out why the sinus node stopped
Junctional bradycardia	Under 40	The backup pacemaker is itself failing	More worrying — manage as a symptomatic bradycardia
Accelerated junctional	60–100 (junctional tachycardia above 100)	Enhanced automaticity, NOT escape — classically digitalis toxicity, ischemia, electrolytes	Look for the cause. Something is driving the junction above its own rate

Narrow QRS with inverted or absent P waves is the junctional signature — the impulse went down the normal path and BACKWARD into the atria at the same time.

Escape rhythms and accelerated idioventricular rhythm

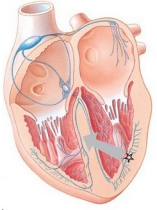


Figure 33-56 Idioventricular rhythm.

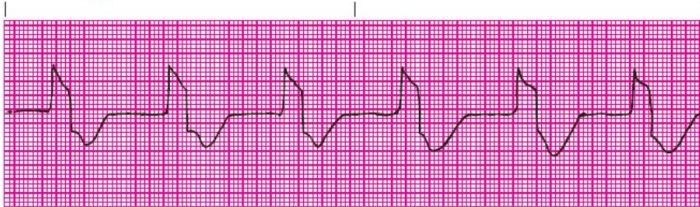
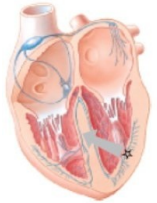


Figure 33-57 Accelerated idioventricular rhythm.

1 Wide, bizarre, no P waves

A ventricular focus spreads cell to cell through muscle instead of down the His-Purkinje system — QRS greater than 0.12 s. No atrial involvement, so no P wave and no PR interval.

2 Escape: 15–40, and it is keeping them alive

Higher pacemakers failed or fell below the ventricular rate. “A safety mechanism that you should NOT suppress.” Manage it as a profound bradycardia — pace, and treat the cause.

3 AIVR: 60–110, during an acute MI

Often a reperfusion rhythm — it appears as an occluded artery opens. No treatment unless hemodynamically unstable; then atropine or overdrive pacing.

4 One focus, three names, decided by rate

15–40 escape • 60–110 accelerated • over 100 in runs of three or more is ventricular tachycardia. The morphology never changes.

“A safety mechanism that you should not suppress.” It is the last pacemaker the patient has. In AIVR, the principal action is aggressive treatment of the infarction.

Premature ventricular contractions

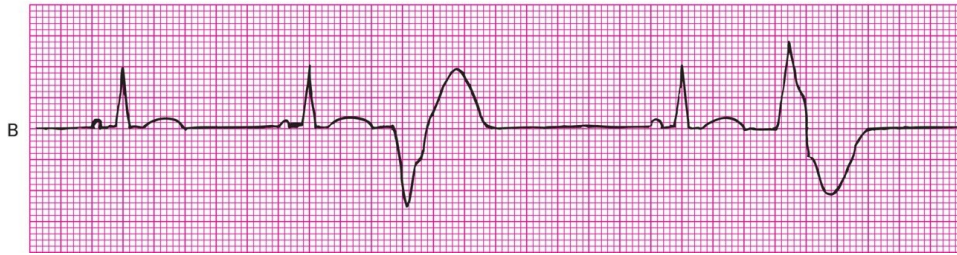
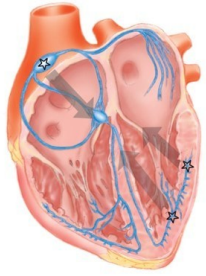


Figure 33-59b Premature ventricular contractions.

R on T

A PVC landing on the preceding T wave falls in the relative refractory period — the vulnerable window from Part 1 — and can precipitate ventricular tachycardia or fibrillation. It is a warning sign, not a curiosity.

1 Early, wide, no P wave

An irritable ventricular focus firing before the next expected beat. QRS over 0.12 s and bizarre; PR absent.

2 Unifocal or multifocal

All alike means one focus. Differing shapes mean several — more ominous, because it suggests widespread irritability rather than one grumpy patch.

3 Bigeminy, trigeminy, quadrigeminy

Every second, third or fourth beat. Two in a row is a couplet; three or more over 100 is ventricular tachycardia by definition.

4 Why it feels like a skipped beat

The early beat has not filled, so it makes little stroke volume and often no pulse. The compensatory pause lets the next beat overfill — the patient feels the gap and the thump.

Ask why the myocardium is irritable

Hypoxia · ischemia · hypokalemia and hypomagnesemia · acidosis · stimulants and caffeine · digitalis toxicity · mechanical irritation from a catheter or wire. Most are correctable, which is the better question to ask.

“PVCs may be insignificant if small in number, but frequency indicates myocardial irritability and may trigger lethal ventricular arrhythmias.”

Ventricular tachycardia and torsades de pointes

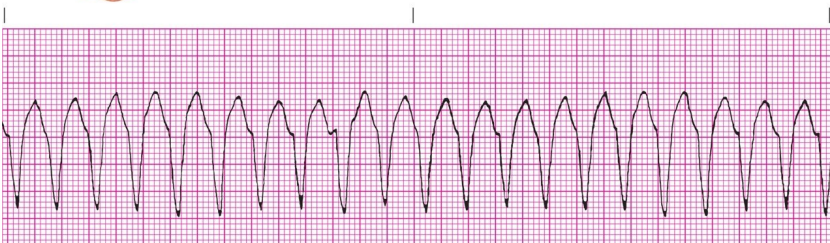
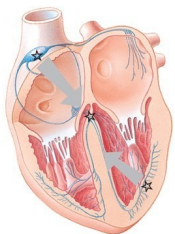


Figure 33-60 Ventricular tachycardia.



Figure 33-61 Torsades de pointes.

Pulse or no pulse decides everything

Pulseless VT is a cardiac arrest rhythm and is DEFIBRILLATED. VT with a pulse follows the wide-complex protocol, and within it, the stable or unstable branch.

	VENTRICULAR TACHYCARDIA	TORSADES DE POINTES
Definition	Three or more ventricular complexes in succession at 100 or more	Polymorphic VT — the complexes twist around the isoelectric line
Rate	About 100–250	Varies
Morphology	Uniform, wide, bizarre	Changing amplitude and shape — which is why it cannot be synchronized
P waves	If present, NOT associated with the QRS	Not discernible
Associated with	Ischemia, infarction, electrolytes, cardiomyopathy	A PROLONGED QT — low magnesium, low potassium, QT-prolonging drugs

REGION SOP Wide complex, pulse present — UNSTABLE: MIDAZOLAM, cardioversion, then AMIODARONE 150 mg over 10 min (max 300). STABLE: AMIODARONE 150 mg. TORSADES: OLMC for MAGNESIUM 2 g over 5 min — and DEFIBRILLATE, since it cannot be synchronized.

Ventricular fibrillation, agonal rhythm and asystole

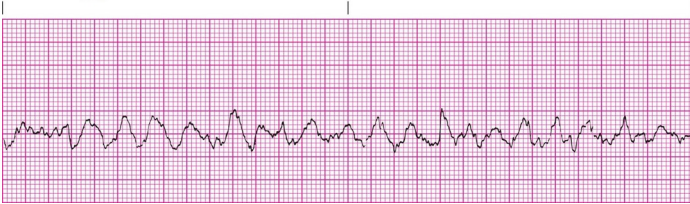
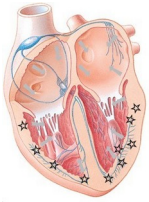


Figure 33-62 Ventricular fibrillation.

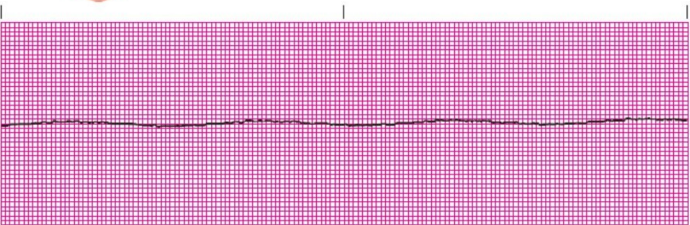
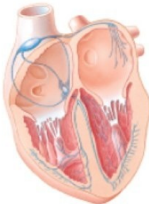


Figure 33-65 Asystole.

1 VF — many reentry circuits at once

No depolarization, no contraction, no output. “A lethal arrhythmia.” Treatment is early electricity with high-quality compressions around it.

2 Coarse and fine describe amplitude

Amplitude falls with time and hypoxia, and fine VF can be mistaken for asystole. Confirm in more than one lead before calling a flat line.

3 Asystole — no electrical activity at all

Occasionally only P waves are seen with no QRS — the atria still firing while the ventricles have stopped. “The prognosis for resuscitation is very poor.”

4 A flat line has four causes

A lead off, the gain down, the wrong mode, or true asystole. Check all four — and asystole is never shocked, because there is nothing to depolarize.

Treat asystole as for all cardiac arrest — look for and treat the underlying cause. The rhythm is the end of a process, and the process is what you are reversing.

Artificial pacemaker rhythms

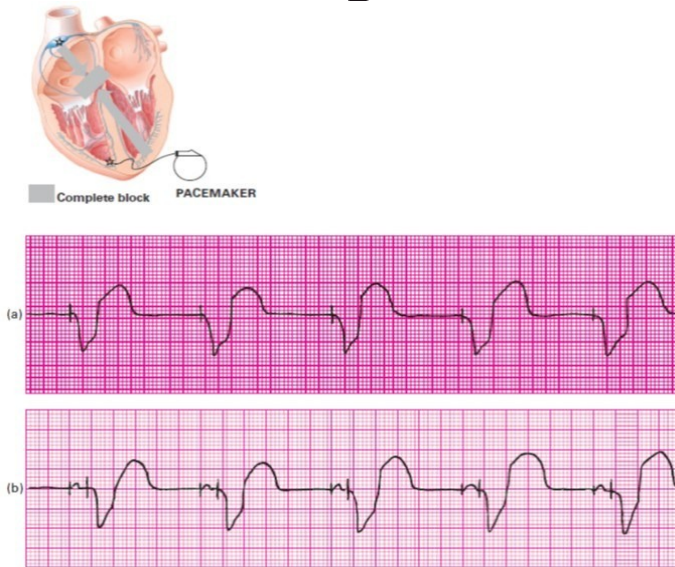


Figure 33-67 Artificial pacemaker rhythm.

Pads and implanted devices

Ask about the device and look for a card or a bulge below the clavicle. Keep defibrillation pads at least 8–10 cm from an implanted generator, and prefer anterior–posterior placement. Defibrillation is NOT contraindicated — the device is built to survive it.

1 Read the spike, then what follows it

Spike then a WIDE QRS is ventricular pacing. Spike then a P wave is atrial pacing. Two spikes is dual chamber.

2 Most units are DEMAND units

They fire only when the intrinsic rate falls below a threshold, so a mixture of paced and native beats is normal. Absent spikes with an adequate own rate is correct function.

3 Three failure modes

FAILURE TO CAPTURE — a spike with no complex after it. FAILURE TO SENSE — spikes at inappropriate times, including on the patient's own complexes. FAILURE TO PACE — no spikes at all.

4 Treat the patient, not the device

A failing pacemaker presents as the bradycardia it was implanted to prevent — manage it as one. Transcutaneous pacing works over an implanted unit.

The one that surprises people

A patient with an implanted defibrillator may be shocked internally while you are touching them. It is startling, and it is not harmful to you.

REGION SOP Bradycardia pearl: “Patients who have undergone cardiac transplant will not respond to ATROPINE.” Ask what hardware and what history the patient has.

Aberrancy, bundle branch block and preexcitation

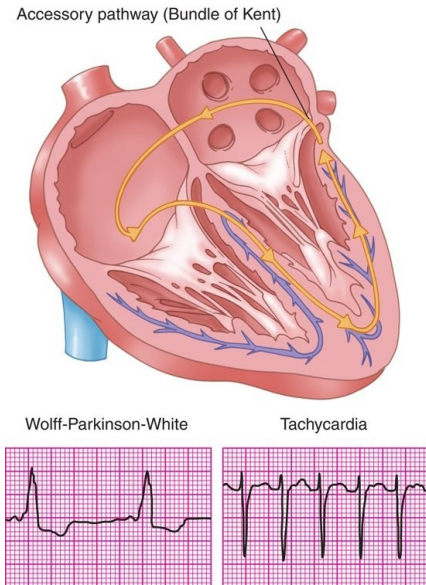


Figure 33-68 Wolff-Parkinson-White.



Figure 33-69 Delta wave.

1 Aberrancy — one beat, delayed

A single supraventricular beat arrives while a bundle branch is still refractory, so it goes down the other branch and then cell to cell. ONE wide complex in a narrow rhythm.

2 Bundle branch block — every beat

ALL supraventricular beats conducted in a delayed manner. Incomplete has a normal QRS; COMPLETE has a wide one.

3 So a wide QRS is not automatically ventricular

Your wide-complex protocol lists the differential: WPW, SVT with aberrancy, paced rhythm, hyperkalemia, hypoglycemia, toxins, MI, hyperthyroidism.

4 WPW — a hole in the gate

An accessory pathway bypasses the AV node. SHORT PR under 0.12 s, LONG QRS over 0.12 s, and a slurred upstroke — the DELTA WAVE. Mostly asymptomatic, but a high incidence of tachyarrhythmias.

In the field, treat a wide complex tachycardia as ventricular tachycardia until proven otherwise

The assumption is safe if you are wrong. The reverse is not — treating ventricular tachycardia as SVT has killed patients.

ECG changes from electrolytes and hypothermia

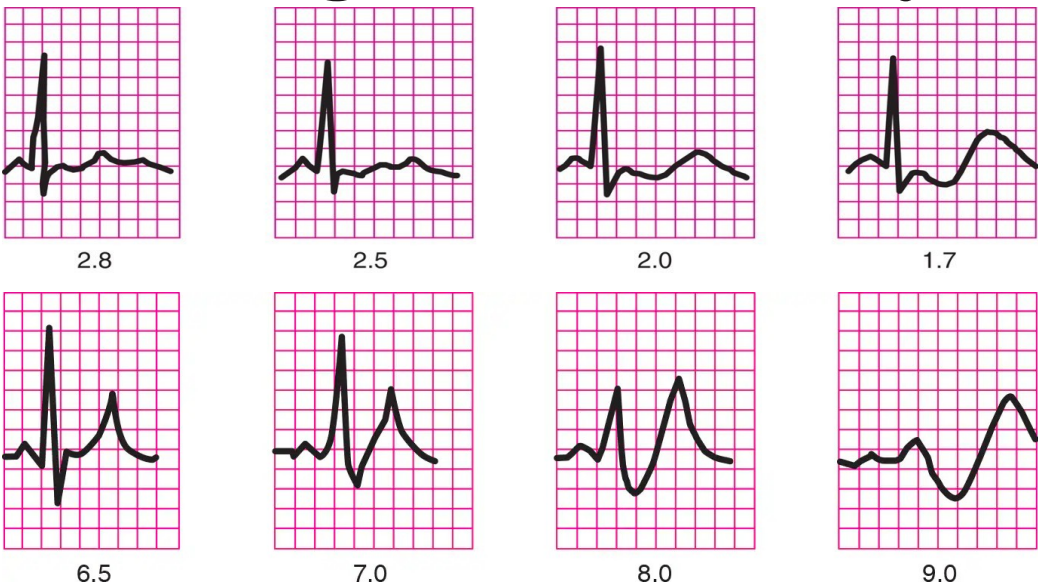


Figure 33-70 ECG changes with potassium.

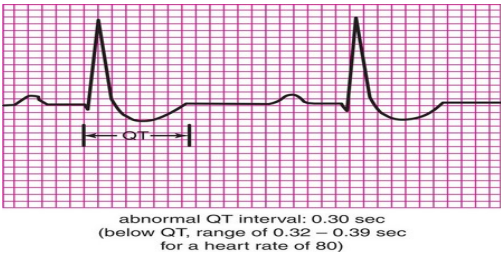


Figure 33-74 ECG changes with calcium.

Derangement	What appears on the ECG
HYPOkalemia (under 3.5)	A prominent U WAVE and flattening of the T wave — plus a long QT and a tendency to ectopy
HYPERkalemia (6.0–6.5 and up)	T waves TALLER and PEAKED toward 7.0 · at 8.0 the T merges with the QRS into a very wide complex · near 9.0 the waveform becomes almost SINUSOIDAL
HYPOnormalcalcemia	Prolonged repolarization — a long ST segment, and therefore a long QT
HYPERnormalcalcemia	Shortened repolarization — the ST shortens until the T wave sits almost on the QRS
HYPOTHERMIA	A J (OSBORN) wave — a slow positive deflection at the end of the QRS. Also T inversion, PR/QRS/QT prolongation, bradycardia, AF or flutter, AV block, PVCs, VF, asystole

A wide, slow, bizarre rhythm with peaked T waves in a dialysis patient is HYPERKALEMIA until proven otherwise

It will not answer an antiarrhythmic, because the problem is not irritable myocardium — it is the membrane potential itself. And a cold heart responds poorly to drugs and electricity until it is warmed: handle gently, treat the temperature.

From the strip to the protocol — and the question that comes first

Ask this before you name anything: stable, or unstable?

UNSTABLE: altered mental status, MAP under 65, or SBP under 90. STABLE: GCS 15, MAP above 65, SBP 90 or greater, skin warm and dry. The branch comes first, and it decides whether you reach for electricity or a drug.

The five steps said...	Rhythm family	Protocol — stable	Protocol — unstable
Rate under 60	Sinus bradycardia, junctional escape, AV blocks, idioventricular	Monitor and find the cause	ATROPINE 1 mg, repeat q3 min to 3 mg, and pace — BUT skip atropine entirely for Type II and third-degree block
Fast, regular, narrow, no visible P waves	SVT	Vagal maneuver, then ADENOSINE 6 mg then 12 mg	MIDAZOLAM, then synchronized cardioversion
Fast, irregularly irregular, no P waves	Atrial fibrillation · flutter with variable conduction	VERAPAMIL 5 mg over 5 min, may repeat to 10 mg	MIDAZOLAM, then synchronized cardioversion — weighing stroke risk
Fast and WIDE	Ventricular tachycardia until proven otherwise	AMIODARONE 150 mg over 10 min, max 300 mg	MIDAZOLAM, cardioversion, then amiodarone. Torsades: magnesium, and DEFIBRILLATE — it cannot be synchronized
No pulse	VF · pulseless VT · asystole · PEA	—	Arrest algorithms. VF and pulseless VT are SHOCKED; asystole and PEA are not

The rhythm name buys you three things: which protocol to open, whether electricity is likely to help, and which reversible cause to hunt. It does not, by itself, tell you to treat.

What Part 2 gives you for the rest of the chapter

1 The ECG records electricity, not pumping

"It tells you nothing about the heart's pumping ability." Check a pulse, not a screen — and check the patient before treating an impossible-looking strip.

2 Five steps, every strip, out loud

Rate, rhythm, P waves, PR interval, QRS. Then the sixth question no strip can answer: is the patient tolerating it?

3 Narrow or wide sorts the field

At or above the junction uses the normal conduction system — narrow. Below it spreads cell to cell — wide and bizarre. Site of origin is written in the width.

4 Three mechanisms make arrhythmias

Enhanced automaticity, reentry, and escape. Ectopic beats come EARLY; escape beats come LATE — and escape must never be suppressed.

5 The blocks are told apart by the PR

Long and constant is first degree. Progressively lengthening is Type I. Constant with dropped beats is Type II. No relationship at all is third degree.

6 Wide does not always mean ventricular

Aberrancy, bundle branch block, pacing, WPW and hyperkalemia all widen the QRS — but in the field, treat a wide tachycardia as VT until proven otherwise.

7 Some rhythms are corrections, not problems

Sinus arrhythmia is normal. Wandering atrial pacemaker has no detrimental effects. Ventricular escape is the last pacemaker they have.

8 Look-alikes you can fix

Peaked T waves widening into a sinusoidal complex is hyperkalemia. A J wave is hypothermia. Neither answers an antiarrhythmic.

Next: Part 3 — assessment and management of the cardiovascular patient

Forty-five minutes: the focused history and physical, and the management foundations — including defibrillation, cardioversion and pacing, which are the interventions this deck kept pointing at.

The 58-year-old — and why the atropine stays in the box

Work the five steps

Atrial rate about 75, ventricular about 38 — count them separately. Ventricular rhythm irregular. P waves present, regular, upright, and MORE of them than QRS complexes. PR constant at 0.18 s. QRS 0.14 s — wide.

The constant PR is the answer

Type I lengthens progressively before dropping a beat. A PR that never varies, with beats dropped without warning, is SECOND-DEGREE TYPE II. The wide QRS says the block sits low — below the node, in the bundle branches.

Why the atropine stays in the box

Atropine blocks vagal input at the SA and AV nodes. A block in the His–Purkinje system is structural, not vagal — so it does nothing, and by speeding the atria it can worsen the conduction ratio and slow the ventricles further.

What she actually needs

She is unstable — MAP about 61, pale, cool, “far away.” Transcutaneous pacing, with MIDAZOLAM 2 mg for sedation and fentanyl available. OLMC for push-dose epinephrine if the MAP stays under 65. Transport for a transvenous pacer.

The rhythm name did not decide the treatment — the LOCATION of the block did. And that came from two measurements: the PR pattern and the QRS width.

Check your understanding

- 1 What does the ECG record — and what does it explicitly not tell you?
- 2 Which way do positive and negative impulses deflect, and what is the isoelectric line?
- 3 Name the three lead groups and the plane each views.
- 4 Why is lead II the standard monitoring lead?
- 5 Give the paper speed, the value of a small box, a large box, and 1 mV.
- 6 Which rate method works for an irregular rhythm, and why do the others fail?
- 7 State the normal PR, QRS and QT intervals.
- 8 What does a long PR mean? A short PR? A wide QRS?
- 9 Map the lead groups to the three surfaces, and name the artery for each.
- 10 List the five steps of rhythm analysis, and the sixth question that follows them.
- 11 Name the three categories of irregularity. Which one suggests atrial fibrillation?
- 12 Give the five criteria for normal sinus rhythm.
- 13 Name the three mechanisms of arrhythmia. How do you tell an ectopic beat from an escape beat?
- 14 When is sinus bradycardia normal? When is sinus tachycardia a compensation?
- 15 Contrast wandering atrial pacemaker with multifocal atrial tachycardia.
- 16 Give the atrial rates for flutter and fibrillation, and the two things the patient loses in AF.
- 17 Distinguish the four AV blocks by what the PR interval is doing.
- 18 Why does your protocol forbid atropine in Type II and third-degree block?
- 19 What is the ECG signature of a junctional rhythm, and why does it look that way?
- 20 Give the rates for ventricular escape, AIVR and VT. Which must never be suppressed?
- 21 Define unifocal, multifocal, bigeminy — and explain why R on T matters.
- 22 Define ventricular tachycardia. What makes torsades different, and why can it not be synchronized?
- 23 What do coarse and fine describe in VF? What are the four causes of a flat line?
- 24 How do you recognize ventricular pacing, and name the three pacemaker failure modes.
- 25 Contrast aberrant conduction with bundle branch block.
- 26 Give the three ECG findings of Wolff-Parkinson-White and the anatomy behind them.
- 27 Describe the hyperkalemia progression. What ECG finding suggests hypothermia?
- 28 Define unstable — and say when that question gets asked.

Answer these before the session and again after — that gap is the measure of what you learned.