UR MEDICINE AFFILIATE

Cardiac Dysrhythmia Recognition & Response

Bedside Quick Reference

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Table of Contents

Section 1

Conduction System & ECG Basics

Cardiac Conduction Pathway

The heart's electrical system triggers each heartbeat in an orderly sequence. Disruption at any level produces a recognizable dysrhythmia pattern.

> **NORMAL CONDUCTION SEQUENCE**
>
> **SA Node** (right atrium) → atrial depolarization → **AV Node** (brief delay — allows ventricular filling) → **Bundle of His** → **Right & Left Bundle Branches** → **Purkinje Fibers** → ventricular depolarization

Key Points at Each Level

- **SA Node:** Primary pacemaker; located in the right atrium near the SVC. Fires spontaneously at 60–100 bpm. Autonomic nervous system modulates rate.
- **AV Node:** The only normal electrical bridge between atria and ventricles. Introduces a 0.12–0.20 s delay (the PR interval) to allow ventricular filling before systole.
- **Bundle of His:** Carries impulse from AV node into the interventricular septum, then divides into right and left bundle branches.
- **Purkinje Fibers:** Rapid distribution of impulse throughout ventricular myocardium, producing near-simultaneous depolarization and a narrow QRS (<0.12 s).

Pacemaker Hierarchy

If a higher-level pacemaker fails, a lower-level *escape pacemaker* takes over — slower but potentially life-saving.

Pacemaker Site	Intrinsic Rate	QRS Appearance	Clinical Significance
SA Node	60 – 100 bpm	Narrow (<0.12 s)	Normal primary pacemaker
AV Junction	40 – 60 bpm	Narrow (<0.12 s)	Escape when SA fails; inverted/absent P waves
Ventricles	20 – 40 bpm	Wide (≥0.12 s), bizarre	Last-resort escape; inadequate CO; prepare to pace

ECG Waveform Reference

Waveform / Interval	What It Represents	Normal Value	Abnormal → Think
P Wave	Atrial depolarization (SA node fires → atria contract)	Smooth, upright in II; <0.12 s wide, <2.5 mm tall	Absent → junctional/AF; inverted → junctional/retrograde
PR Interval	Atrial depol + AV nodal delay (P onset → QRS onset)	0.12 – 0.20 s (3–5 small boxes)	>0.20 s → AV block; <0.12 s → WPW / junctional
QRS Complex	Ventricular depolarization (ventricular contraction follows)	<0.12 s (3 small boxes)	≥0.12 s → bundle branch block, ventricular origin, aberrant
ST Segment	Early ventricular repolarization (should be isoelectric)	At baseline	Elevation → ischemia/injury; Depression → ischemia
T Wave	Ventricular repolarization	Same direction as QRS; asymmetric	Peaked (hyperK), inverted (ischemia), tall (hyperK)
QT Interval	Total ventricular depol + repol (Q onset → T end)	QTc ≤ 0.44 s (varies with rate — use corrected QTc)	Prolonged QTc → Torsades risk (drugs, electrolytes)

Small box = 0.04 s · Large box = 0.20 s · 25 mm/sec standard paper speed

📏 **ECG PAPER QUICK FACTS**

Horizontal (time): 1 small box = 0.04 s · 1 large box = 0.20 s · 5 large boxes = 1 second
Vertical (voltage): 1 small box = 0.1 mV · 10 small boxes (1 large) = 1.0 mV

Section 2

5-Step ECG Analysis Method

Use this systematic approach for *every* rhythm strip. Consistency prevents missed findings.

1

Rate

Regular rhythm — 300 Method: Count the number of large boxes between two consecutive R waves, then divide 300 by that number.

Example: 4 large boxes → 300 ÷ 4 = 75 bpm

Irregular rhythm — 6-Second Count Method: Count the number of QRS complexes in a 6-second strip (30 large boxes) and multiply by 10.

Example: 8 complexes in 6 s → 80 bpm

Memorize the sequence: 300 → 150 → 100 → 75 → 60 → 50 (1, 2, 3, 4, 5, 6 large boxes)

2

Rhythm

Compare **R-R intervals** across the entire strip using calipers or a pen mark.

- **Regular:** R-R intervals vary by <0.04 s (less than 1 small box)
- **Regularly irregular:** Pattern to the irregularity (e.g., grouped beats in Wenckebach)
- **Irregularly irregular:** No pattern — classic for atrial fibrillation

3

P Waves

Ask these questions for every strip:

- **Present?** If absent → AF, VF, or junctional rhythm
- **Upright in Lead II?** Upright = SA node origin; inverted = retrograde (junctional)
- **One P for every QRS?** More P's than QRS = AV block; no relationship = 3rd-degree block
- **All P's look the same?** Variable morphology = wandering pacemaker, PACs, or multifocal atrial

4

PR Interval

Measure from the *beginning of the P wave* to the *beginning of the QRS complex*.

- **Normal:** 0.12 – 0.20 s (3 – 5 small boxes)
- **Short (<0.12 s):** Pre-excitation (WPW), junctional rhythm
- **Long (>0.20 s):** First-degree AV block
- **Progressive lengthening → dropped QRS:** Second-degree Type I (Wenckebach)
- **Fixed PR with sudden drop:** Second-degree Type II (Mobitz II)

5 QRS Width

Measure the widest QRS from the beginning of the Q wave (or R if no Q) to the end of the S wave.

- **Narrow (<0.12 s / <3 small boxes):** Supraventricular origin (SA, atrial, or AV junctional) — normal conduction through the ventricles
- **Wide (≥0.12 s / ≥3 small boxes):** Ventricular origin, bundle branch block, aberrant conduction, or paced rhythm

Wide + fast + no pulse = VF/VT until proven otherwise → ACLS

⚡ NORMAL VALUES QUICK REFERENCE

Heart Rate	60 – 100 bpm
PR Interval	0.12 – 0.20 s (3 – 5 small boxes)
QRS Duration	<0.12 s (<3 small boxes)
QTc Interval	≤0.44 s (rate-corrected)
Rhythm	Regular (R-R varies <0.04 s)

Section 3

Sinus Rhythms

All sinus rhythms originate in the **SA node**. P waves are upright in Lead II, one-to-one P:QRS relationship, and PR interval is normal. Differences relate to rate and regularity.

RATE	RHYTHM	P WAVES	PR INTERVAL
60 – 100 bpm	Regular	Upright, uniform, 1:1	0.12 – 0.20 s

QRS	SIGNIFICANCE
Narrow <0.12 s	Normal cardiac rhythm

Nursing Action: Continue routine monitoring. No intervention required.

RATE	RHYTHM	P WAVES	PR INTERVAL
<60 bpm	Regular	Upright, uniform, 1:1	0.12 – 0.20 s

QRS	SIGNIFICANCE
Narrow <0.12 s	May be normal or pathologic

Common Causes

- Normal variant: athletes, during sleep, vasovagal response
- Medications: beta-blockers, calcium channel blockers, digoxin, opioids
- Inferior MI (RCA occlusion → SA node ischemia)
- Hypothyroidism, hypothermia, increased ICP (Cushing's reflex)
- Sick sinus syndrome

Nursing Action:

- Assess for symptoms: hypotension, chest pain, altered mental status, syncope, diaphoresis
- **Symptomatic:** Notify provider immediately → Atropine 0.5 mg IV (per order); transcutaneous pacing if refractory
- **Asymptomatic:** Increase monitoring frequency; review and report contributing medications; document
- Obtain 12-lead ECG; check electrolytes if not recent

RATE	RHYTHM	P WAVES	PR INTERVAL
>100 bpm (usually <180)	Regular	Upright, may merge with T at fast rates	0.12 – 0.20 s

QRS	SIGNIFICANCE
Narrow <0.12 s	Almost always a *secondary* response

Common Causes (Treat the Cause!)

- Pain, anxiety, fever, sepsis (most common on the floor)
- Hypovolemia / hemorrhage / dehydration
- Hypoxia, pulmonary embolism, heart failure
- Medications: atropine, epinephrine, dopamine, albuterol, thyroid supplements
- Anemia, hyperthyroidism, stimulants, caffeine

Nursing Action: Do NOT slow sinus tach without first identifying the cause.

- Assess: vital signs, O_2 saturation, pain scale, temperature, fluid status, recent medications
- Treat underlying cause (fluids, analgesia, antipyretics, anxiolytics as ordered)
- Notify provider if rate >130 without clear cause, if hemodynamically unstable, or if rate is not responding to treatment
- Obtain 12-lead ECG to rule out SVT

RATE	RHYTHM	P WAVES	PR INTERVAL
60 – 100 bpm	*Irregular* — cycles with breathing	Upright, uniform	0.12 – 0.20 s

QRS	SIGNIFICANCE
Narrow <0.12 s	Benign; normal in young adults

Rate speeds up with inspiration (vagal tone decreases) and slows with expiration (vagal tone increases). Normal variant; common in children and young athletes.

Nursing Action: No treatment required. Document as benign finding. Confirm it correlates with respiratory cycle if uncertain.

Section 4

Atrial Rhythms

Atrial rhythms arise from ectopic foci in the atria (not the SA node). P waves are present but may appear early, abnormal in shape, or buried. QRS is usually narrow (normal ventricular conduction).

RATE	RHYTHM	P WAVES	PR INTERVAL
Underlying rate + early beat	Irregular at PAC	Early, different morphology (ectopic atrial focus)	May be short or variable

QRS	PAUSE
Narrow (usually); wide if aberrant conduction	Incomplete compensatory pause (SA node resets)

PACs occur when an irritable atrial focus fires before the SA node. Usually benign. Triggers: caffeine, alcohol, stimulants, electrolyte disturbances, emotional stress, hypoxia.

Nursing Action: Usually benign — monitor and document frequency.

- Assess for triggers: caffeine, medications, electrolytes (K^+, Mg^{2+})
- Notify provider if PACs are frequent (>6/min), occur in runs, or patient is symptomatic (palpitations, presyncope)
- Frequent PACs may precede AF — monitor for rhythm changes

RATE	RHYTHM	P WAVES	PR INTERVAL
150 – 250 bpm	Regular (abrupt onset/termination)	Often buried in T wave or retrograde; may not be visible	Not measurable if P buried

QRS	SIGNIFICANCE
Narrow (<0.12 s) unless aberrant	Symptomatic; may reduce CO

Classic mechanism: **re-entry circuit** within or near the AV node (AVNRT most common). Sudden onset of rapid palpitations, light-headedness, chest tightness. Distinguishing SVT from sinus tach: SVT rate usually >150 with abrupt onset; sinus tach gradual.

Nursing Action:

1. Assess hemodynamic stability (BP, LOC, chest pain, diaphoresis)
2. **Stable:** Vagal maneuvers (Valsalva, carotid sinus massage — provider order); IV access; 12-lead ECG
3. **Adenosine 6 mg rapid IV push** (followed by 20 mL saline flush) — per order; may repeat with 12 mg ×2 if no response
4. **Unstable (hypotension, chest pain, altered LOC):** Prepare for synchronized cardioversion — notify provider/activate response team
5. Continuous monitoring; post-conversion rhythm documentation

ATRIAL RATE
250 – 350 bpm

VENTRICULAR RATE
Depends on AV conduction (2:1 → ~150; 4:1 → ~75)

RHYTHM
Regular (if fixed block ratio) or regularly irregular

P WAVES
Sawtooth flutter waves (F waves) — best in II, III, aVF

PR/FR INTERVAL
Fixed block ratio (2:1, 3:1, 4:1)

QRS
Narrow unless aberrant

Key identifier: Characteristic sawtooth (flutter) waves at ~300 bpm. At 2:1 conduction, ventricular rate ≈ 150 bpm — suspect flutter if rate is exactly 150. AV node protects ventricles from very rapid rates.

> ⚠️ **STROKE RISK**
>
> Atrial flutter carries similar stroke risk to AF. Anticoagulation should be addressed per provider. If flutter has been present >48 hours, cardioversion requires TEE or adequate anticoagulation first.

Nursing Action:

- Assess for symptoms and hemodynamic stability
- Rate control medications per order (beta-blockers, calcium channel blockers, digoxin)
- Monitor for variable block and abrupt rate changes
- Anticoagulation assessment with provider
- Stable with persistent flutter: cardioversion (electrical or pharmacologic) per provider plan

ATRIAL RATE	VENTRICULAR RATE	RHYTHM	P WAVES
350 – 600 bpm (chaotic)	Irregularly irregular; 60 – 170+ bpm	**Irregularly irregular** (hallmark)	None — chaotic fibrillatory (f) waves; undulating baseline

PR INTERVAL	QRS
Not measurable	Narrow (unless aberrant or WPW)

> 🫀 **ATRIAL KICK — CLINICAL PEARL**
>
> **Atrial fibrillation eliminates the atrial kick** — the active filling contribution of atrial contraction to ventricular preload. This reduces cardiac output by **15–20%**, which may be poorly tolerated in patients with CHF, hypertrophic cardiomyopathy, or significant diastolic dysfunction. Assess for signs of decompensation.

Key Clinical Concerns

- **Stroke Risk:** Blood pools in the left atrial appendage → thrombus formation → embolic stroke. Use CHA_2DS_2-VASc score to guide anticoagulation.
- **Rate Control:** Goal ventricular rate 60–100 bpm at rest. Medications: beta-blockers, calcium channel blockers (diltiazem IV for rapid AF), digoxin.
- **New-onset vs. chronic:** New-onset AF within 48 h may be cardioverted safely; >48 h requires anticoagulation or TEE to exclude LA thrombus before cardioversion.

Nursing Action:

- Assess HR, BP, symptoms (palpitations, dyspnea, chest pain, neurological changes)
- IV access; 12-lead ECG; cardiac monitoring
- Rate control medications per order; monitor for response
- Anticoagulation as ordered (heparin, LMWH, DOACs)
- **New neuro deficits → Stroke protocol immediately**
- **Unstable (hypotension, pulmonary edema, chest pain):** Prepare for synchronized cardioversion — notify provider stat

Section 5

Junctional Rhythms

Junctional rhythms arise from the **AV node/junction** when the SA node fails to fire or is suppressed. Key features: inverted or absent P waves, narrow QRS (unless aberrant conduction exists).

KEY CONCEPT: RETROGRADE P WAVES

The AV junction depolarizes the atria *retrograde* (backward). P waves appear:

- **Before QRS** — if atria depolarize just before ventricles (short PR <0.12 s)
- **Buried in QRS** — if simultaneous depolarization (P waves absent/hidden)
- **After QRS** — if atria depolarize after ventricles (P in ST segment, inverted)

In Lead II: retrograde P waves are **inverted (negative)**.

RATE	RHYTHM	P WAVES	PR INTERVAL
40 – 60 bpm	Regular	Inverted, absent, or after QRS	<0.12 s or not measurable

QRS	SIGNIFICANCE
Narrow <0.12 s	Backup pacemaker — SA node failed

This is a *protective escape mechanism* — the AV junction stepping in because the SA node is not functioning. May be seen with increased vagal tone, medication effects (dig toxicity, beta-blockers), inferior MI, or sick sinus syndrome.

Nursing Action:

- Assess for symptoms: dizziness, hypotension, syncope (rate may be too slow for adequate CO)
- Notify provider — identify and treat underlying cause
- Review medications (digoxin level, beta-blockers)
- Symptomatic: Atropine 0.5 mg IV per order; prepare for transcutaneous pacing
- Do NOT suppress this rhythm — it is keeping the patient alive

RATE
60 – 100 bpm

RHYTHM
Regular

P WAVES
Inverted, absent, or after QRS

PR INTERVAL
<0.12 s or not measurable

QRS
Narrow <0.12 s

SIGNIFICANCE
Enhanced automaticity of AV junction

Enhanced automaticity of the junction fires at a rate matching the SA node range. The rate is "accelerated" beyond the junction's normal intrinsic rate (40–60). Causes: digoxin toxicity, myocardial ischemia, post-cardiac surgery, electrolyte imbalance.

Nursing Action:

- Assess for symptoms; hemodynamic stability
- Monitor for digoxin toxicity if on digoxin
- Identify and address underlying cause
- Notify provider; continue monitoring

RATE
>100 bpm (usually 100 – 130)

RHYTHM
Regular

P WAVES
Inverted, absent, or after QRS

PR INTERVAL
<0.12 s or not measurable

QRS
Narrow <0.12 s

SIGNIFICANCE
Less common; may impair CO

Less common than AVNRT (SVT). May be seen with digitalis toxicity, myocarditis, or cardiac surgery. Distinguished from AVNRT by slightly lower rate and clinical context.

Nursing Action: Assess symptoms; notify provider. Manage underlying cause. Hemodynamically unstable → ACLS protocol/cardioversion per provider.

💡 **JUNCTIONAL RHYTHM SUMMARY**

All junctional rhythms share: **inverted/absent P waves** + **narrow QRS** (unless aberrant). The rate classifies them: <40 = junctional escape with poor SA & AV function · 40–60 = junctional escape · 60–100 = accelerated junctional · >100 = junctional tachycardia

Section 6

Ventricular Rhythms

Ventricular rhythms originate below the Bundle of His. **Wide, bizarre QRS complexes (≥0.12 s)** are the hallmark because impulses travel through ventricular muscle rather than the fast His-Purkinje system. These rhythms range from benign ectopy to immediately life-threatening.

RATE
Underlying rate + early PVC

RHYTHM
Irregular at PVC

P WAVES
No P before PVC; may see retrograde P after

PR INTERVAL
None for PVC

QRS
Wide ≥0.12 s, bizarre, opposite T wave direction

PAUSE
Full compensatory pause (SA node NOT reset)

PVC Terminology

Term	Definition	Significance
Unifocal	All PVCs look identical — same ectopic focus	Less concerning than multifocal
Multifocal	PVCs with different morphologies — multiple foci	More concerning; may indicate significant irritability
Bigeminy	Every other beat is a PVC	Assess hemodynamics; frequent ectopy
Trigeminy	Every third beat is a PVC	Assess hemodynamics; frequent ectopy
Couplet	Two consecutive PVCs	Increased concern; may precede VT
Run / Salvo	3+ consecutive PVCs (<30 s = nonsustained VT)	High concern — notify provider
R-on-T	PVC falls on T wave (vulnerable period)	Can precipitate VT/VF — **high risk**

Nursing Action:

- Assess symptoms: palpitations, dizziness, decreased BP with PVCs
- Identify and treat causes: hypoxia (most common), electrolyte imbalances (K^+, Mg^{2+}), ischemia, medications (digoxin toxicity), stimulants, anxiety
- Obtain 12-lead ECG; check labs (K^+, Mg^{2+}, troponin if new)
- Notify provider for: >6 PVCs/min, multifocal PVCs, couplets, runs, R-on-T pattern
- Continuous monitoring; document frequency and morphology

RATE	RHYTHM	P WAVES	PR INTERVAL
20 – 40 bpm	Regular (ventricular escape)	Absent or AV dissociation	None (AV dissociation)

QRS	SIGNIFICANCE
Very wide >0.12 s, bizarre	**Last-resort pacemaker — critical**

The ventricles are functioning as the only pacemaker — SA node and AV junction have both failed. Cardiac output at this rate is severely compromised. Immediate intervention required.

Nursing Action:

1. **Check pulse immediately** — if pulseless, this is PEA → start CPR and activate code blue
2. If pulse present: notify provider STAT; prepare for transcutaneous pacing
3. IV access; atropine per order (may not be effective as block is below AV node)
4. Do NOT suppress IVR — it is the only rhythm present
5. Cardiac consult for transvenous pacing

RATE	RHYTHM	P WAVES	QRS
40 – 100 bpm	Regular	Absent or AV dissociation	Wide ≥0.12 s, bizarre

SIGNIFICANCE
Often benign — reperfusion marker

Reperfusion rhythm: Commonly seen after successful thrombolysis or PCI for STEMI. Enhanced automaticity of ventricular cells recently restored from ischemia. Rate is between IVR and VT. Usually self-limiting and benign.

Nursing Action:

- Assess pulse and hemodynamics — if stable, this is typically benign and self-limited
- Notify provider (especially if occurring post-thrombolysis or post-PCI — document as possible reperfusion rhythm)
- Do not treat unless hemodynamically compromised — suppressing may cause worse rhythm
- Monitor closely; document onset and duration

RATE
>100 bpm (usually 150 – 250)

RHYTHM
Regular (monomorphic) or irregular (polymorphic)

P WAVES
AV dissociation — P waves may be seen but unrelated to QRS

QRS
Wide ≥0.12 s, bizarre, uniform (mono) or varying (poly)

SUBTYPES
Monomorphic, Polymorphic, Torsades de Pointes

DURATION
Nonsustained <30 s; Sustained ≥30 s

> 🚨 **PRIORITY ACTION: CHECK FOR PULSE FIRST**
>
> VT with a pulse is treated differently from pulseless VT. **Always check for pulse before initiating treatment.** Pulseless VT → CPR + Defibrillation (unsynchronized). Pulse present → see algorithm below.

VT with Pulse — Stable vs. Unstable

Status	Signs	Treatment
Unstable	Hypotension, chest pain, altered LOC, signs of shock	Synchronized cardioversion (200 J biphasic) → ACLS
Stable	Normotensive, alert, no chest pain	Antiarrhythmic therapy per order: Amiodarone 150 mg IV over 10 min; Lidocaine 1–1.5 mg/kg IV; consider elective cardioversion

Nursing Action:

1. **Check pulse immediately**
2. Call for help; activate response team / code blue if pulseless
3. Pulseless VT → CPR; defibrillate 200 J unsynchronized; ACLS epinephrine 1 mg IV q3-5 min
4. Pulse present, unstable → synchronized cardioversion; O_2; IV access; antiarrhythmics per order
5. Pulse present, stable → 12-lead ECG; notify provider; antiarrhythmics per order
6. Prepare crash cart, defibrillator, suction

RATE
No identifiable rate — chaotic

RHYTHM
Completely chaotic, no pattern

P WAVES
None

QRS
No identifiable QRS complexes — coarse or fine fibrillatory waves

PULSE
ABSENT — cardiac arrest

CO
Zero — no effective cardiac output

> 🚨 **CARDIAC ARREST — CODE BLUE**
>
> VF produces **no cardiac output**. Every minute without defibrillation decreases survival by 7–10%. Start CPR immediately and defibrillate as soon as possible. Early defibrillation is the single most important intervention.

Nursing Action — IMMEDIATE:

1. **Confirm unresponsive, no pulse, no normal breathing**
2. **Call code blue / activate rapid response**
3. **Start high-quality CPR** (100–120/min, 2 in. depth, allow full recoil)
4. Apply defibrillator pads; **charge to 200 J (biphasic)**
5. **DEFIBRILLATE** — clear all personnel, deliver shock
6. Resume CPR immediately for 2 minutes
7. IV/IO access; Epinephrine 1 mg IV q3-5 min; Amiodarone 300 mg IV after 3rd shock
8. Continue 2-minute CPR cycles; check rhythm/pulse every 2 min
9. Search for reversible causes: H's and T's

Section 7

AV Blocks

AV blocks represent impaired or absent conduction through the AV node or infranodal conduction system. Classified by *degree* (how complete the block is) and *location* (AV node vs. bundle branches). Location matters: nodal blocks respond to atropine; infranodal (His-Purkinje) blocks do not.

RATE
Normal (underlying rate)

RHYTHM
Regular

P WAVES
Normal, 1:1 P:QRS

PR INTERVAL
>0.20 s (prolonged, constant)

QRS
Narrow <0.12 s

DROPPED BEATS
None — all P waves conduct

Every P wave conducts to the ventricles, but with a delay longer than normal. The AV node conducts slowly but completely. Causes: inferior MI, medications (beta-blockers, digoxin, CCBs), myocarditis, increased vagal tone, fibrosis.

Nursing Action: Monitor only. No treatment required for isolated first-degree block. Document and notify provider. Watch for progression to higher-degree block, especially with new inferior MI.

RATE	RHYTHM	P WAVES	PR INTERVAL
Atrial: normal · Ventricular: slightly slower	Regularly irregular (grouped beats)	Regular; more P waves than QRS	**Progressive lengthening → suddenly dropped QRS**
QRS	**PATTERN**		
Narrow (AV node level)	"Grouped beating" — repeating cycles		

The AV node becomes progressively fatigued with each impulse until it fails to conduct once — then the cycle resets. **Location: AV node.** Usually benign, especially inferior MI (transient, often resolves). Atropine generally effective.

Nursing Action:

- Assess for symptoms — usually asymptomatic; may cause mild bradycardia
- Notify provider; document PR pattern (show progressive lengthening on strip)
- Monitor for progression to higher-degree block
- Symptomatic: Atropine 0.5 mg IV per order (usually effective)
- Hold QT-prolonging or AV-nodal blocking medications if implicated (per provider)

RATE
Atrial: normal · Ventricular: bradycardic

RHYTHM
Irregular (dropped beats unpredictable)

P WAVES
Regular; more P waves than QRS

PR INTERVAL
FIXED and constant → suddenly dropped QRS (no warning)

QRS
Often wide (bundle branch involvement)

LOCATION
Bundle of His or bundle branches (infranodal)

> ⚠️ **HIGH-RISK BLOCK — PACING REQUIRED**
>
> Mobitz II indicates disease in the His-Purkinje system. It can progress suddenly and unpredictably to **complete (third-degree) block** or asystole. **Transcutaneous pacing should be prepared.** Atropine is often *ineffective* (block is infranodal).

Nursing Action:

1. Notify provider **IMMEDIATELY**
2. Prepare transcutaneous pacemaker at bedside and ensure it is functional
3. IV access; continuous monitoring; 12-lead ECG
4. Atropine 0.5 mg IV per order (may not respond — do not delay pacing for atropine)
5. Anticipate transvenous pacing placement (cardiology/EP consult)
6. Do not leave patient unattended

ATRIAL RATE
Normal (SA node firing independently)

VENTRICULAR RATE
Depends on escape pacemaker: junctional 40–60; ventricular 20–40

RHYTHM
Both regular, but **completely independent**

P WAVES
Present and regular but **no relationship to QRS**

PR INTERVAL
Completely variable — no constant PR

QRS
Narrow if junctional escape; Wide if ventricular escape

> 🚨 **EMERGENCY — TRANSCUTANEOUS PACING NOW**
>
> No impulses conduct from atria to ventricles. The ventricles rely on an escape pacemaker — slow and unreliable. Patient will be hemodynamically compromised. Cardiac output is severely reduced. **This is a medical emergency requiring immediate pacing.**

Nursing Action:

1. Activate rapid response / call provider **STAT**
2. Apply transcutaneous pacemaker pads if not already placed
3. Initiate transcutaneous pacing (see Section 8 — Pacemakers for technique)
4. Atropine 0.5 mg IV (unlikely to work but may buy time in nodal block)
5. Dopamine or epinephrine drip may be needed to support BP during pacing setup
6. Prepare patient for transvenous pacemaker insertion (EP/cardiology)
7. If pulseless → CPR, code blue, ACLS

AV Block Comparison: Type I vs. Type II

Feature	2° Type I (Wenckebach)	2° Type II (Mobitz II)
PR Pattern	Progressive lengthening before dropped QRS	Constant/fixed PR — sudden dropped QRS (no warning)
QRS Width	Typically narrow (<0.12 s)	Often wide (≥0.12 s) — bundle branch involvement
Block Location	AV Node (supranodal)	Bundle of His / Bundle Branches (infranodal)
Atropine Response	Usually effective	Usually **NOT** effective
Risk of Progression	Low (usually transient)	**HIGH** — may progress suddenly to complete block
Pacing Urgency	If symptomatic, prepare pacer	**Transcutaneous pacer at bedside ALWAYS**
Common Causes	Inferior MI, medications, vagal tone	Anterior MI, fibrosis/sclerosis of conduction system

Section 8

Pacemakers

Pacemakers deliver electrical stimuli to maintain an adequate heart rate when the native conduction system fails. Understanding normal pacemaker function is essential to recognizing malfunction.

Pacemaker Nomenclature (NBG Code — First 3 Positions)

Position	I — Chamber Paced	II — Chamber Sensed	III — Response to Sensing
A	Atrium	Atrium	Triggered
V	Ventricle	Ventricle	Inhibited
D	Dual (A + V)	Dual (A + V)	Dual (T + I)
O	None	None	None

Example: VVI = Pace ventricle, Sense ventricle, Inhibit pacing when native beat sensed. DDD = Dual chamber pacing/sensing with dual response.

Common Pacemaker Modes

- **Paces:** Ventricle only
- **Senses:** Ventricle — detects native QRS complexes
- **Response:** Inhibited — pacing is suppressed when native beat sensed at or above programmed rate
- **Use:** Chronic AF with slow ventricular response; backup for bradycardia
- **Limitation:** No AV synchrony — loses atrial kick; "pacemaker syndrome" possible

On strip: Pacing spike immediately before QRS (wide, paced QRS). No atrial pacing spike.

- **Paces:** Both atria and ventricles
- **Senses:** Both atria and ventricles
- **Response:** Dual — triggered (atrial sensing triggers ventricular pace) and inhibited (native beats inhibit pacing)
- **Use:** AV block with intact sinus node; sick sinus syndrome; most physiologic mode
- **Advantage:** Maintains AV synchrony and atrial kick

On strip: Atrial spike before P wave + ventricular spike before QRS (when both are paced). May show only atrial pacing if AV conduction intact.

Normal Pacemaker Function

What You See	What It Means	Normal?
Spike → P wave	Atrial pacing — stimulates atrial depolarization	✓ **Normal atrial capture**
Spike → Wide QRS	Ventricular pacing — stimulus travels through muscle (slow), produces wide QRS	✓ **Normal ventricular capture**
No spike when native beat occurs at/above rate	Pacemaker sensed native beat and inhibited (demand mode working)	✓ **Normal sensing and inhibition**

Pacemaker Malfunction

Definition: Pacemaker spike is present but *not followed* by a P wave (atrial) or QRS (ventricular). The stimulus fails to depolarize the myocardium.

Causes: Lead displacement, fibrosis at lead tip, output too low, metabolic derangement (hyperkalemia, acidosis), battery depletion, myocardial infarction at lead site

Nursing Action: Notify provider. Increase output if programmable. Check lead connections. Prepare transcutaneous pacing as backup. Check electrolytes. Position patient left lateral if capture improves (repositions lead).

Definition: Pacemaker *does not detect* native cardiac activity and fires when it shouldn't — competes with native rhythm. Pacing spikes fall at random points in the cycle.

Danger: A pacing spike during the **T wave (relative refractory period)** can trigger VT or VF (R-on-T equivalent).

Causes: Lead displacement, low sensitivity setting, electromagnetic interference, lead fracture, fibrosis

> **Nursing Action:** Notify provider immediately. Increase sensitivity setting if temporary pacer. Avoid triggers (electromagnetic sources). If spike falls on T wave → high risk — prepare for VT/VF intervention.

Definition: No pacemaker spike is generated when one should be — the pacemaker fails to fire when the rate falls below the programmed lower rate limit.

Causes: Battery depletion, lead fracture/disconnection, oversensing (misinterpreting noise as native beats), electromagnetic interference

> **Nursing Action:** Check lead connections and cable connections. Check battery. Notify provider. Assess patient for symptoms. Prepare transcutaneous pacing. Identify and remove sources of electromagnetic interference.

Transcutaneous Pacing (TCP)

WHEN TO USE TRANSCUTANEOUS PACING

- Symptomatic bradycardia unresponsive to atropine
- Second-degree Type II or third-degree AV block
- Hemodynamically unstable bradycardia of any cause
- Bridge to transvenous pacing

TCP Procedure (Quick Reference)

1. **Explain to patient** — TCP is painful; sedation/analgesia required (midazolam + fentanyl per order)
2. **Apply pads:** Anterior-posterior placement preferred (anterior: left precordium / posterior: left subscapular). Ensure good pad contact; shave if needed.

3. **Set pacing rate:** Start at 60–80 bpm (or as ordered)
4. **Set output (mA):** Start low (begin at 20 mA); increase in 10 mA increments until capture
5. **Confirm capture:**
    - Electrical capture: pacing spike followed by wide QRS on monitor
    - Mechanical capture: palpable pulse with each QRS (femoral or carotid — do NOT use radial); BP improves
6. **Set output 10 mA above threshold** (safety margin)
7. Reassess patient; document settings; arrange transvenous pacing

> ⚠️ **TCP NOTES**
>
> - Pacing artifact can look like electrical capture — always confirm mechanical capture by palpating a pulse
> - Patient will feel muscle twitching — ensure adequate analgesia
> - Do not delay pacing while titrating medications
> - TCP is a bridge — arrange definitive transvenous pacing promptly

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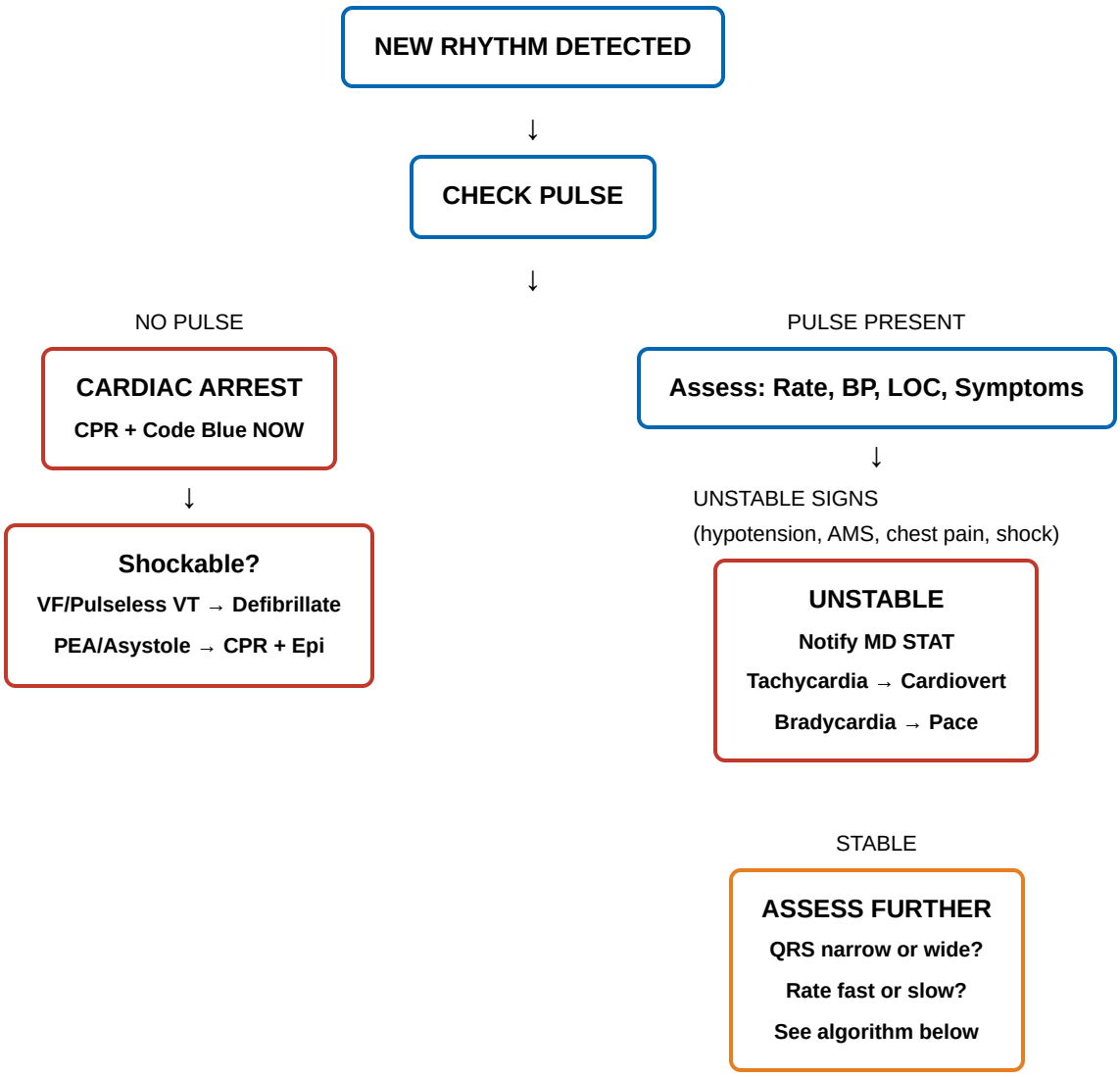
Section 9

Quick Reference & Clinical Decision Aids

Is This Rhythm Dangerous? Decision Tree



Bradycardia Action Guide (HR < 60 bpm)

BRADYCARDIA ALGORITHM (SIMPLIFIED — PER ACLS)

1. **Is the patient symptomatic?**
   Signs of poor perfusion: hypotension, altered mental status, ischemic chest discomfort, acute heart failure, shock
2. **If YES → Immediate treatment:**
   - O_2 if SpO_2 <94%; IV access; 12-lead ECG; monitor
   - **Atropine 0.5 mg IV** — may repeat every 3–5 min to max **3 mg total**
   - If atropine ineffective: **Transcutaneous pacing** (preferred over drugs if available)
   - Dopamine 2–10 mcg/kg/min IV or Epinephrine 2–10 mcg/min IV while awaiting pacing
3. **If NO (asymptomatic):**
   - Identify and treat underlying cause (medications, electrolytes, ischemia)
   - Observe; increase monitoring; notify provider
4. **If Mobitz II or 3rd-degree block:** Prepare pacemaker regardless of symptoms — these can deteriorate without warning

Tachycardia Action Guide (HR > 100 bpm)

TACHYCARDIA ALGORITHM (SIMPLIFIED — PER ACLS)

1. **Is the patient unstable?** (hypotension, chest pain, altered LOC, signs of shock)
2. **UNSTABLE → Immediate synchronized cardioversion**
   - Regular narrow-complex: 50–100 J biphasic
   - Regular wide-complex: 100 J biphasic
   - Irregular (AF with RVR): 120–200 J biphasic
   - Sedate if time permits; notify provider; apply pads
3. **STABLE → Identify rhythm type:**
   - **Narrow QRS, regular:** Vagal maneuvers → Adenosine 6 mg rapid IV push → 12 mg if no response
   - **Narrow QRS, irregular (AF/Flutter):** Rate control (diltiazem, metoprolol, digoxin) per order; anticoagulation assessment
   - **Wide QRS, regular:** Presume VT; Amiodarone 150 mg IV over 10 min; prepare for cardioversion
   - **Wide QRS, irregular:** May be pre-excited AF, polymorphic VT, Torsades; avoid AV nodal blockers in WPW; Torsades → Mg^{2+} 1–2 g IV, defibrillate if unstable

Emergency Drug Quick Reference

Drug	Indication	Adult Dose	Notes
ATROPINE	Symptomatic bradycardia; AV block (nodal level)	0.5 mg IV bolus; repeat q3–5 min; max **3 mg**	Anticholinergic — accelerates SA node rate. NOT effective for Mobitz II or 3rd-degree block (infranodal). <0.5 mg may worsen bradycardia.
ADENOSINE	Stable SVT (narrow QRS, regular); diagnostic for wide-complex tachycardia	**6 mg** rapid IV push with 20 mL saline flush; may repeat **12 mg ×2** if no response	Very short half-life (~10 s) — must give via large antecubital or central IV rapidly. Causes transient asystole (warn patient). Contraindicated in WPW + AF/Flutter, sick sinus without pacemaker, 2°/3° AV block.
AMIODARONE	VT with pulse (stable); refractory VF/pulseless VT (cardiac arrest); rate control AF	**Stable VT:** 150 mg IV over 10 min, then 1 mg/min × 6 h, then 0.5 mg/min × 18 h **Cardiac arrest:** 300 mg IV/IO push; second dose 150 mg	Prolongs QT interval — monitor QTc. Multiple drug interactions. Hypotension with rapid infusion. Phlebitis with peripheral line — use central if possible. Long half-life (weeks).
LIDOCAINE	VT with pulse; refractory VF/pulseless VT (alternative to amiodarone)	**1–1.5 mg/kg** IV bolus (max 3 mg/kg loading); maintenance **1–4 mg/min** infusion	Class Ib antiarrhythmic. CNS toxicity: tinnitus, tremor, seizures at toxic levels. Reduce dose in liver failure and elderly. Less effective than amiodarone for refractory VF in most studies.

When to Activate Rapid Response vs. Code Blue

Response	Activate When	Examples
Rapid Response Team	Patient has pulse; acutely deteriorating; you are concerned but not in arrest	New hemodynamically significant dysrhythmia; Mobitz II or 3rd-degree block; SVT/VT with BP drop; acute mental status change; respiratory distress
Code Blue	Unresponsive; pulseless; no normal breathing	VF; pulseless VT; PEA; asystole; sudden collapse; monitor shows VF and patient unresponsive

🚨 **TRUST YOUR ASSESSMENT**

If the patient looks bad — call for help. Don't wait for a "perfect" rhythm diagnosis before activating a rapid response. An early call is always better than a late code. Your assessment of clinical deterioration is valid even if you're uncertain of the exact rhythm.

✅ **THE H'S AND T'S — REVERSIBLE CAUSES OF CARDIAC ARREST**

H's:

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo/Hyperkalemia
- Hypothermia

T's:

- Tension pneumothorax
- Tamponade (cardiac)
- Toxins
- Thrombosis — pulmonary
- Thrombosis — coronary (MI)

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made in the context of the individual patient. Thompson Health Nursing Education Department is not responsible for outcomes resulting from the use of this reference guide in isolation from professional clinical judgment.