



Bradycardia & Heart Block

AV conduction disease · Is it symptomatic? Is it reversible? Is it infranodal?

SITE DECIDES FATE
nodal vs infranodal

CLASSIFY *where the block sits predicts whether it will progress*

Degree	ECG	Site & behavior
First degree	PR > 200 ms, every P conducts	Usually nodal . Benign in isolation. Marked delay (> 300 ms) can cause pseudo-pacemaker symptoms.
Mobitz I (Wenckebach)	Progressive PR lengthening, then a dropped beat; grouped beating	Nodal (AV node) . Often vagal or drug-related, frequently benign, rarely progresses. Improves with exercise or atropine.
Mobitz II	Constant PR , then a sudden dropped beat; often wide QRS	Infranodal (His-Purkinje) . Unstable, progresses to complete block without warning. Escape rhythm is slow and unreliable .
2:1 block	Every other P conducts — cannot classify by PR pattern alone	Ambiguous. Narrow QRS + PR long favors nodal; wide QRS favors infranodal. Exercise or atropine improves nodal, worsens infranodal .
Third degree (complete)	AV dissociation, atrial rate > ventricular, regular P-P and R-R	Escape site sets the risk: junctional (narrow, 40–60) is more stable than ventricular (wide, 20–40), which is unreliable.

ACUTE MANAGEMENT *treat the patient, not the number*

- 1 **Is the patient unstable?** Hypotension, altered mentation, ischemic chest pain, or acute heart failure. A rate of 38 in a well athlete needs nothing; a rate of 48 with shock needs everything.
- 2 **Atropine 1 mg IV**, repeat every 3–5 min to a maximum of 3 mg. Works on the **AV node** — so it helps nodal block but is **often ineffective in Mobitz II or complete block**, and by speeding the atria it can **worsen** infranodal block.
- 3 **Transcutaneous pacing** if atropine fails — sedate, set rate 60–80, increase output until **electrical and mechanical capture** (confirm with a pulse, not the monitor).
- 4 **Chronotropic infusion** as a bridge: dopamine 5–20 mcg/kg/min or epinephrine 2–10 mcg/min.
- 5 **Transvenous pacing** for persistent unstable bradycardia, then decide on a permanent device once reversible causes are excluded.

FIND THE REVERSIBLE CAUSE *before anyone implants a device*

DRUGS — check the chart first
Beta-blockers, non-dihydropyridine **calcium channel blockers**, digoxin, amiodarone, clonidine, acetylcholinesterase inhibitors. **Glucagon** for beta-blocker toxicity, **IV calcium** for CCB toxicity, **digoxin Fab** for digoxin.

METABOLIC & ISCHEMIC
Hyperkalemia (widening QRS, peaked T) · **hypothyroidism** · hypothermia · **inferior MI** (RCA supplies the AV node; often transient and atropine-responsive) · **anterior MI** with block, which signals extensive infarction and is ominous.

INFILTRATIVE & INFECTIOUS
Lyme carditis — young patient, endemic exposure, high-grade block that usually **resolves with antibiotics**, so do not implant early. Also sarcoidosis, amyloidosis, endocarditis with **root abscess**, myocarditis, post-TAVR or post-surgical.

PERMANENT PACING *symptoms that match, or a block that will not recover*

Indication	Detail
Symptomatic bradycardia	Pace when symptoms correlate temporally with the bradycardia and no reversible cause is found. Correlation is the whole game.
Mobitz II, high-grade, or complete block	Permanent pacing when not attributable to a reversible cause — regardless of symptoms, because progression is unpredictable.
Do not pace	Sleep-related sinus bradycardia or transient sinus pauses during sleep should not be paced unless another indication exists. Asymptomatic Mobitz I generally needs no device.
Choose the right pacing mode	If LVEF 36–50% with AV block and > 40% ventricular pacing expected, use more physiologic activation (CRT or His-bundle pacing) rather than RV pacing, to prevent pacing-induced heart failure.

BLOCK IN ACUTE MI *the territory tells you the prognosis*

INFERIOR MI — usually benign
The **RCA supplies the AV node** in most people. Block is **nodal**, with a narrow escape, often **atropine-responsive**, and typically **resolves within days** of reperfusion. Temporary pacing may bridge; permanent devices are usually unnecessary.

ANTERIOR MI — ominous
Block reflects **extensive septal infarction** destroying the His-Purkinje system. **Infranodal**, wide escape, **atropine will not help**, and it signals a large infarct with high mortality. Prepare for pacing early and expect a permanent device.

AFTER THE DEVICE *what to check and what can go wrong*

Issue	Recognize it
Failure to capture	Pacing spike with no following complex . Causes: lead displacement, rising threshold, hyperkalemia, ischemia, or metabolic derangement.
Failure to sense	Spikes falling where they should not, risking R-on-T . Undersensing means the device is blind to intrinsic beats.
Oversensing	The device inhibits inappropriately (myopotentials, electromagnetic interference) and the patient becomes symptomatically bradycardic.
Early complications	Pneumothorax , hematoma, lead perforation with tamponade , and infection. Get a post-procedure chest X-ray; fever with a device demands blood cultures and an echo.
Pacing-induced cardiomyopathy	Falling EF with a high RV pacing burden — the reason physiologic pacing is preferred when heavy pacing is anticipated.

PEARLS & PITFALLS

- **Atropine can worsen infranodal block**. It speeds the atria while the diseased His-Purkinje system cannot follow, dropping the ventricular rate. Have pads on before you push it.
- **Wide QRS escape = infranodal = unstable**. Narrow junctional escape is comparatively reassuring.
- Always check **potassium and TSH**, and review every rate-slowing drug, before calling it primary conduction disease.
- **Lyme carditis usually recovers** — ask about travel and rash in the young patient with high-grade block before implanting.
- Confirm pacing **capture by palpating a pulse**. Electrical spikes on the monitor without mechanical capture is a trap.
- Block with an **anterior MI** implies a large infarct and carries far worse prognosis than block with an inferior MI.

