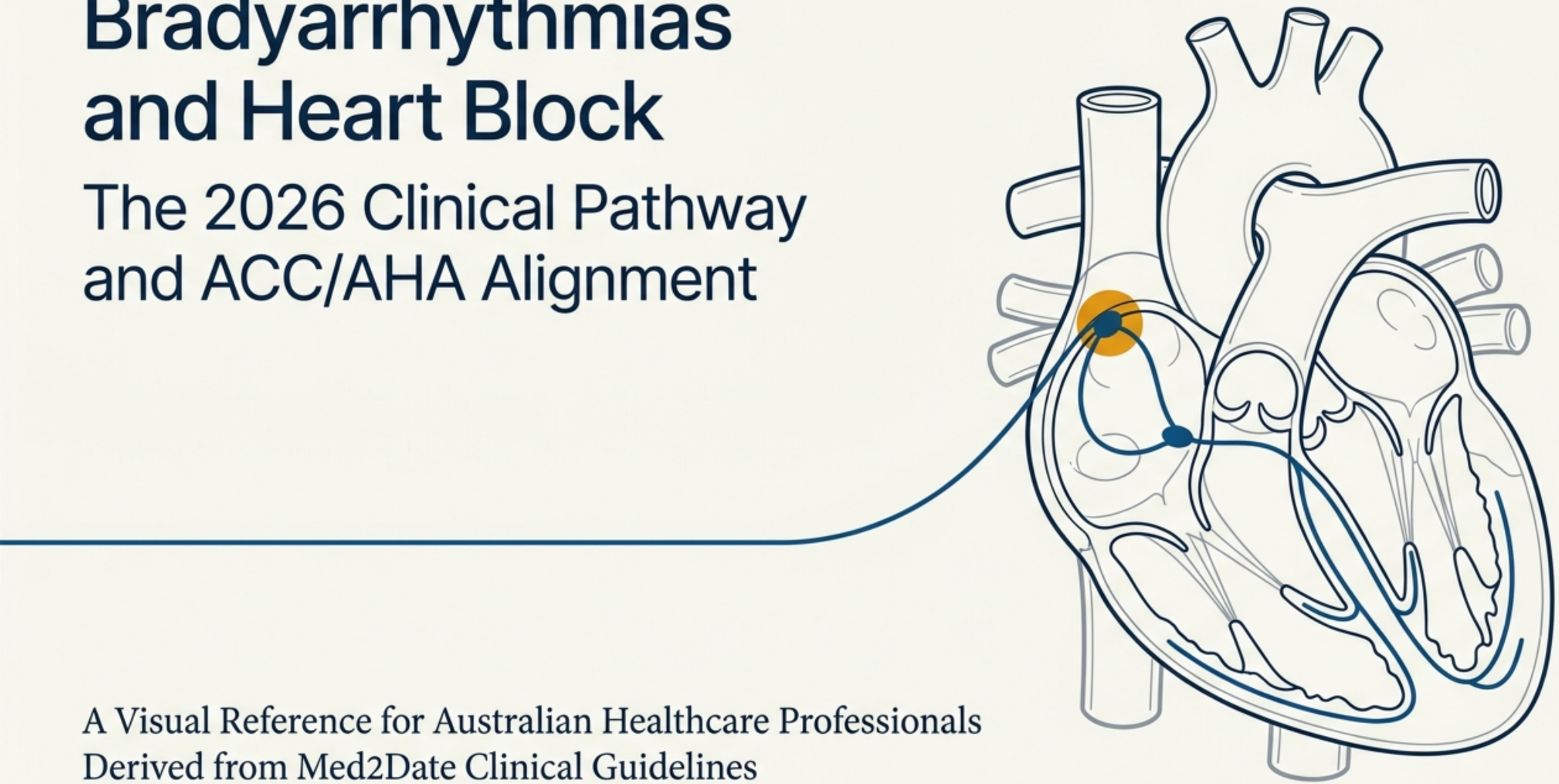
Bradyarrhythmias and Heart Block

The 2026 Clinical Pathway and ACC/AHA Alignment



A Visual Reference for Australian Healthcare Professionals
Derived from Med2Date Clinical Guidelines

The Australian Bradyarrhythmia Landscape

100,000+

Annual hospitalisations for cardiac arrhythmias in Australia (AIHW, 2023).

20,000

New pacemaker implantations per year (ANZCPR data).

77 Years

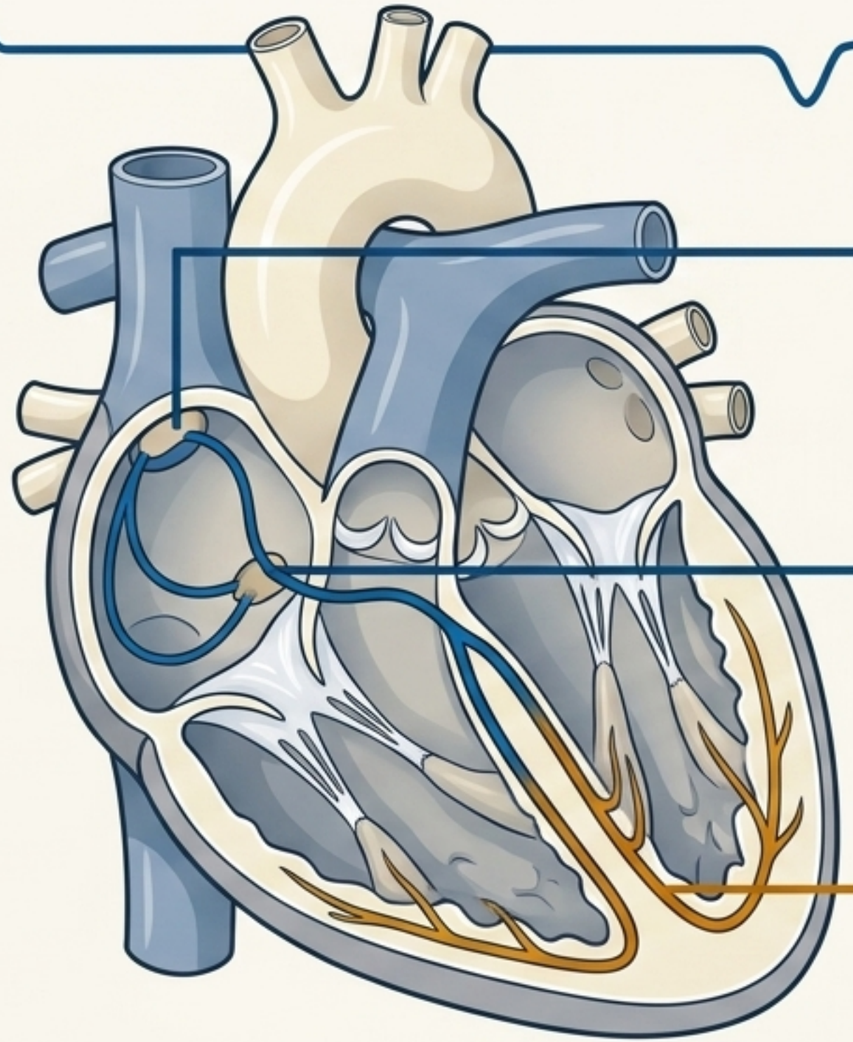
Median age at first pacemaker implantation, reflecting degenerative conduction system disease.

10–20x
Risk Multiplier

Indigenous Australians face drastically higher hospitalisation rates for rheumatic heart disease (RHD) complications, including high-degree AV block.

The Anatomical-Clinical Pathway

Understanding bradyarrhythmias requires mapping the defect to its exact anatomical source. The origin determines the clinical syndrome, the escape rhythm reliability, and the ultimate pacing indication.



1. The Source (Sinoatrial Node)

Impulse generation and sinus node dysfunction (SND).

2. The AV Node (Supra-Hisian)

The physiological bottleneck; often benign blocks responding to autonomic tone.

3. The His-Purkinje System (Infra-Hisian)

The high-risk distal wires; blocks here risk sudden cardiac death and mandate pacing.

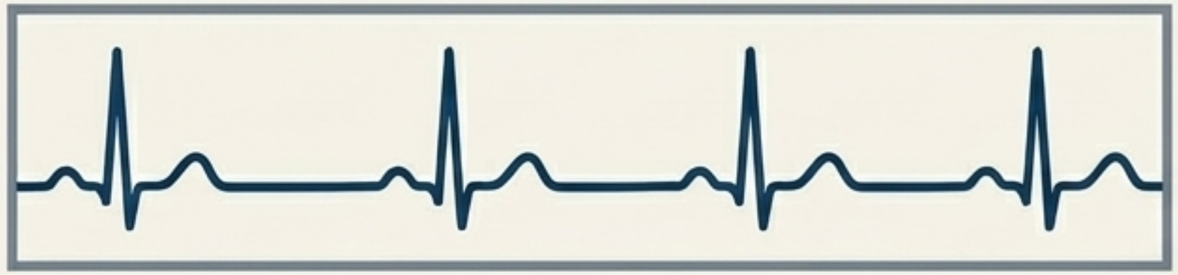
Sinus Node Dysfunction (SND): Pathology and Reversible Causes

SND (or Sick Sinus Syndrome) results from degenerative fibrosis and fatty infiltration of the SA node and perinodal tissue. Before diagnosing intrinsic SND, reversible causes—accounting for up to 30% of bradycardia presentations in elderly patients on polypharmacy—must be excluded.

The Reversible Exclusions Checklist:

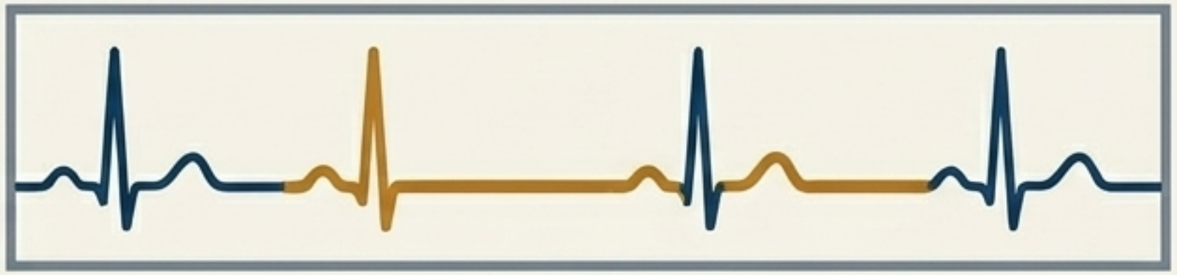
- ☑ **Medications:** Beta-blockers, non-dihydropyridine calcium channel blockers (verapamil, diltiazem), digoxin, amiodarone, ivabradine.
- ☑ **Endocrine:** Hypothyroidism (Check TSH, fT4).
- ☑ **Metabolic:** Hyperkalaemia or hypermagnesaemia suppressing SA automaticity.
- ☑ **Autonomic:** Increased vagal tone.

Clinical Syndromes of the SA Node



Sinus Bradycardia

Rate <60 bpm. Normal P waves and PR interval. Pathological only if symptomatic or rate <40 bpm at rest.

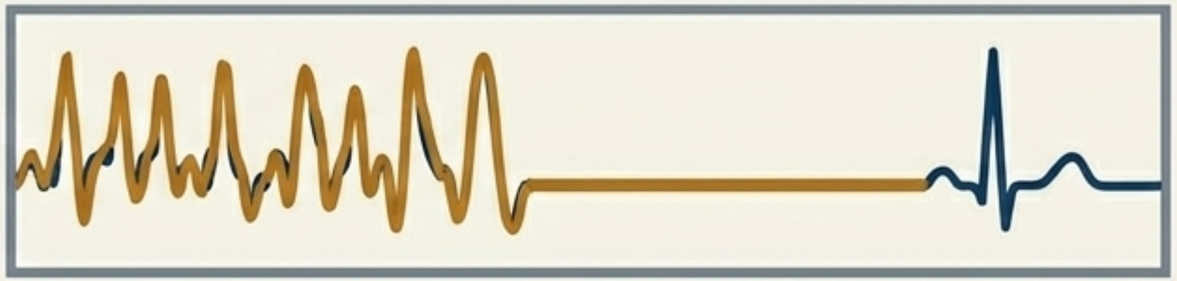


Sinoatrial Exit Block

Type II features a sudden dropped P wave where the pause is a multiple of the basic PP interval. Clinically significant.

Sinus Arrest

Absence of P waves. Pauses >5 seconds during waking hours are a Class I pacing indication.

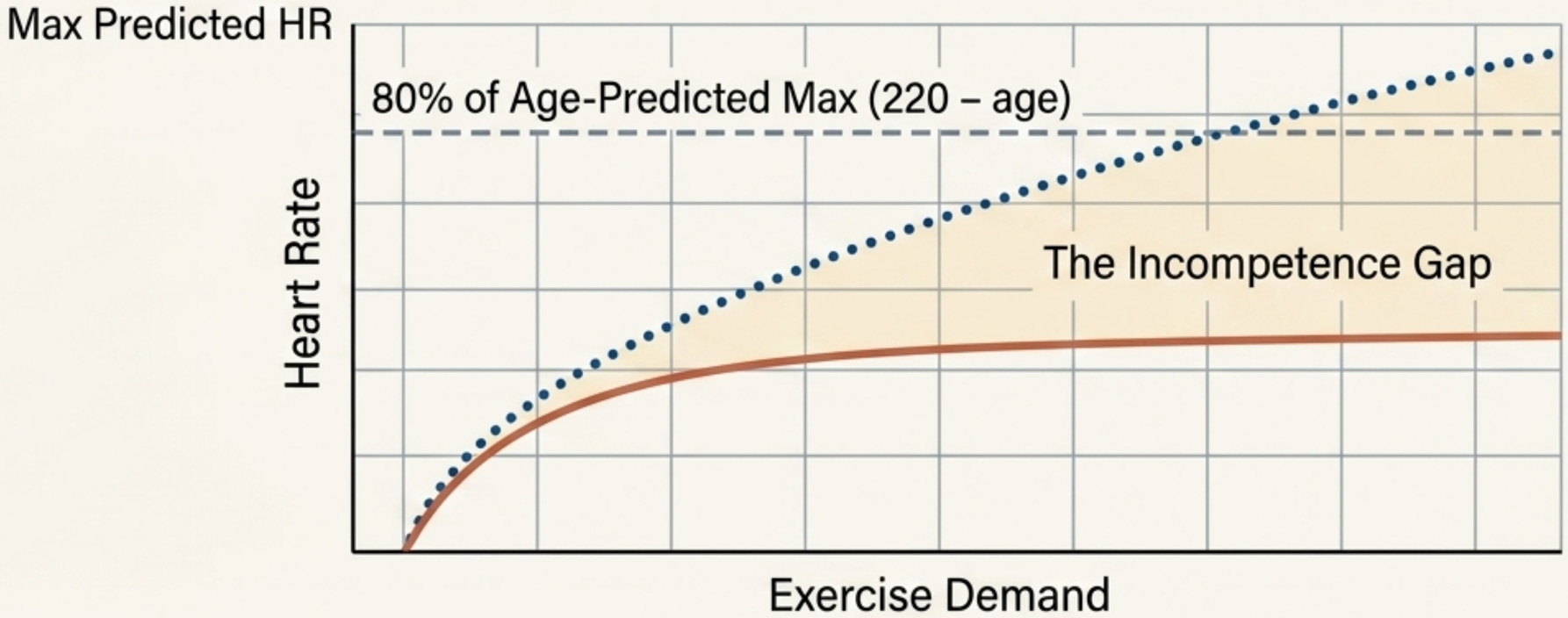


Tachy-Brady Syndrome

Alternating atrial tachyarrhythmias with bradycardia upon termination. Occurs in 40–50% of SND patients; requires pacing for safe antiarrhythmic use.

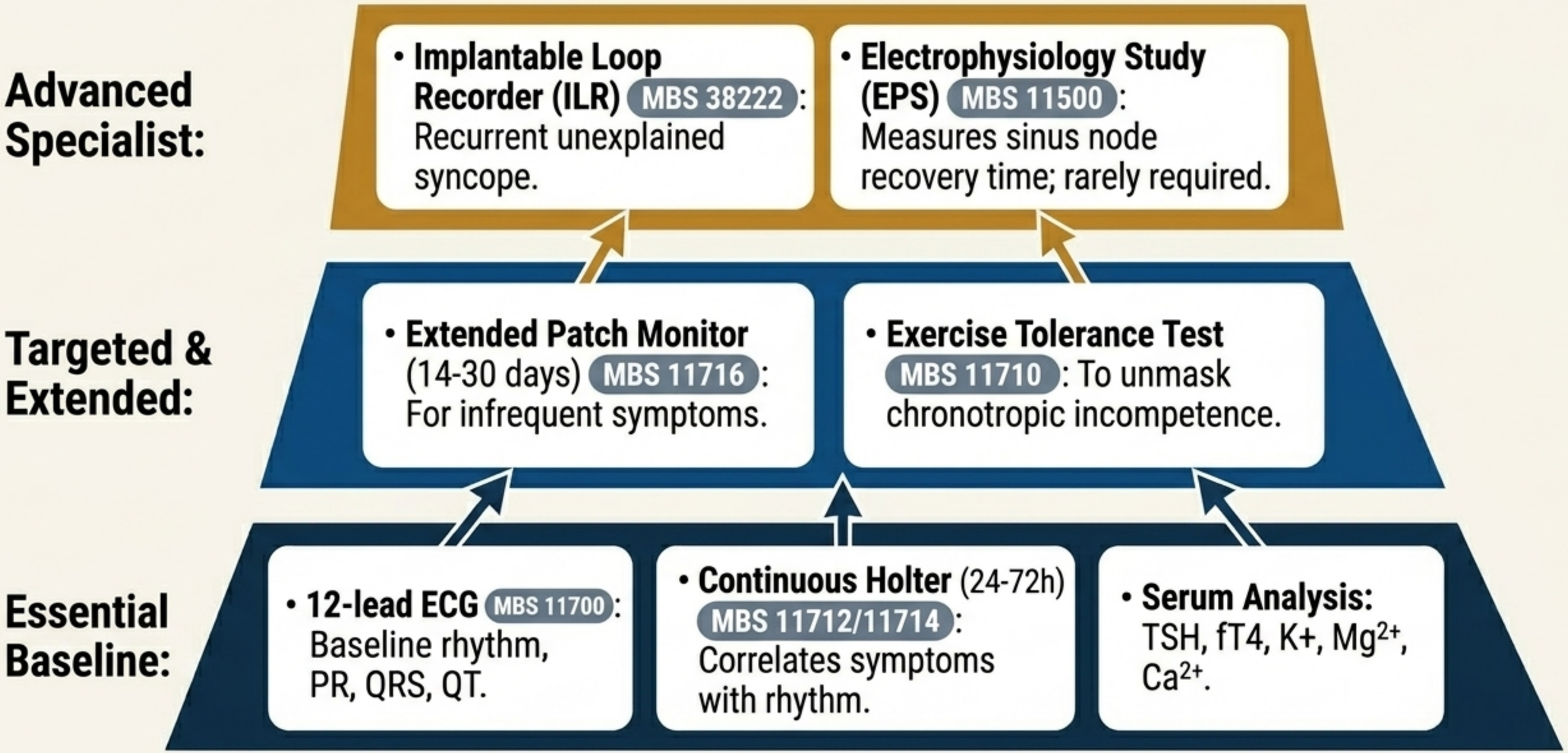
The Chronotropic Incompetence Gap

Chronotropic incompetence is the inability of the heart to increase its rate proportionally to metabolic demand. It may be the sole manifestation of SND and requires rate-adaptive pacing.



Diagnostic Threshold: Failure to achieve ≥80% of age-predicted maximal heart rate during an Exercise Tolerance Test (ETT) or Cardiopulmonary Exercise Testing (CPET).

Diagnostic Pathway for Suspected SND



The Escape Rhythm Spectrum

The danger of an AV block is defined by the escape rhythm that takes over when the primary signal fails. The lower the block, the slower and more unreliable the backup generator.

SA Node (Normal)

60–100 bpm. Reliable, narrow QRS.

Junctional Escape (AV Node/Supra-Hisian)

40–60 bpm. Narrow QRS. Usually stable enough to prevent cardiogenic shock; pacing only if persistent >7 days.

Ventricular Escape (Infra-Hisian)

25–40 bpm. Wide QRS. Highly unreliable. Frequently results in syncope or cardiogenic shock. Requires urgent pacing.

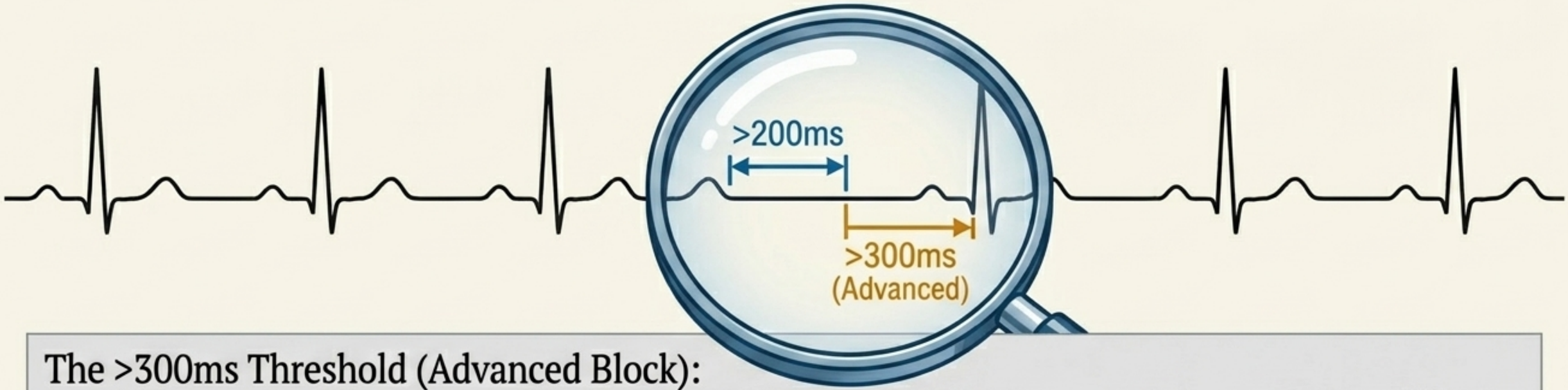
The Site of Block Diagnostic Matrix

Localizing the block precisely via surface ECG clues dictates immediate management and prognosis.

Diagnostic Dimension	Supra-Hisian (AV Node Level)	Infra-Hisian (His-Purkinje Level)
ECG Clues	Narrow QRS, Wenckebach pattern	Wide QRS (Bundle Branch Block), Mobitz II
Response to Atropine	Improves conduction	Atropine-resistant (may worsen block)
Common Aetiology	High vagal tone, inferior MI, AV nodal blocking drugs, RHD	Lenègre-Lev disease (fibrosis), anterior MI, structural/infiltrative disease
Progression Risk	Rarely progresses to complete block	High risk of complete block & sudden death
Pacing Strategy	Only if symptomatic (with reversible causes excluded)	**Class I Indication: Permanent pacing required even if asymptomatic**

First-Degree AV Block: When Benign Becomes Symptomatic

First-degree AV block (1:1 conduction with prolonged PR) occurs in up to 5% of the population and is primarily a benign delay through the AV node.



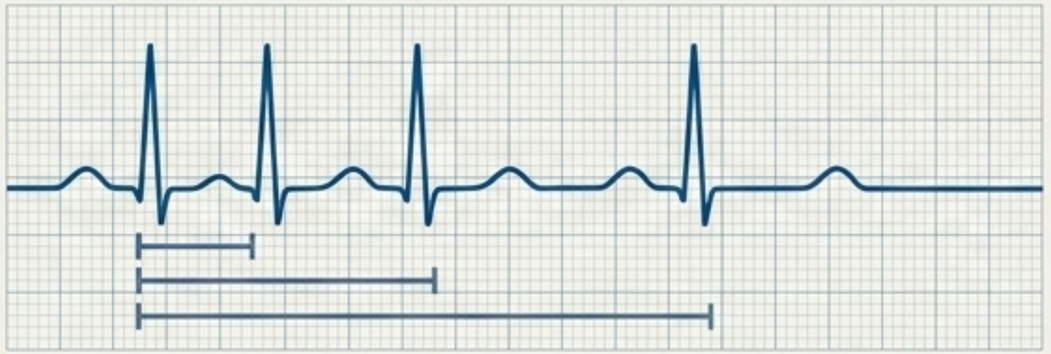
The >300ms Threshold (Advanced Block):

- While standard first-degree block (PR >200ms) requires no intervention, an advanced block (PR >300ms) in elderly patients can cause “PR interval–related symptoms”.
- The extreme delay causes loss of AV synchrony, resulting in fatigue, exercise intolerance, and cannon A waves—closely mimicking pacemaker syndrome.
- Action: This is a Class IIa indication for permanent dual-chamber (DDD) pacing.

The Mobitz Divide

The ECG pattern reveals the anatomical site of the block, fundamentally altering the pacing indication.

Mobitz Type I (Wenckebach)



- Anatomy: Supra-Hisian (AV Node).
- ECG: Progressive PR prolongation before a dropped QRS. Pause is shorter than two basic PP intervals. Usually a narrow QRS.
- Action: Benign. Pacing (Class IIa) only if strictly symptomatic.

Mobitz Type II

- Anatomy: Infra-Hisian (His-Purkinje).
- ECG: Sudden dropped QRS with absolutely constant PR intervals for conducted beats. Usually a wide QRS (≥120ms).
- Action: Lethal. High risk of progression. Class I indication for permanent pacing regardless of symptoms.

Third-Degree (Complete) AV Block in Acute MI

Complete dissociation of atrial and ventricular activity. In the setting of an acute STEMI, the **infarct location** predicts the block's behavior and reversibility.

Inferior STEMI (AV Node Block)

- Mechanism: Ischemia of the AV node (Supra-Hisian). Narrow QRS escape.
- Prognosis: Usually transient (resolves in days with reperfusion).
- Management: Temporary pacing only if symptomatic/hypotensive. Permanent pacing only if block persists >14 days.

Anterior STEMI (His-Purkinje Block)

- Mechanism: Massive infarct destroying distal conduction (Infra-Hisian). Wide QRS escape.
- Prognosis: High mortality. Unreliable escape <40 bpm. May not recover.
- Management: Cardiovascular emergency. Immediate temporary pacing (transcutaneous → transvenous). Permanent pacing prior to discharge.

Acute Symptomatic Bradycardia: Management Flowchart

1. First-Line Pharmacotherapy

Atropine: 0.5–1 mg IV bolus (repeat every 3–5 min). Maximum total 3 mg.

Note: Ineffective for infra-Hisian blocks.

2. Pharmacological Bridge

Isoprenaline (Isoproterenol): 1–4 µg/min IV infusion. Titrate to heart rate.

Use with continuous ECG monitoring (may precipitate VT).

3. Electrical Intervention

Transcutaneous Pacing: For immediate haemodynamic instability (systolic BP <90 mmHg, cardiogenic shock).

Urgent Intervention Protocol

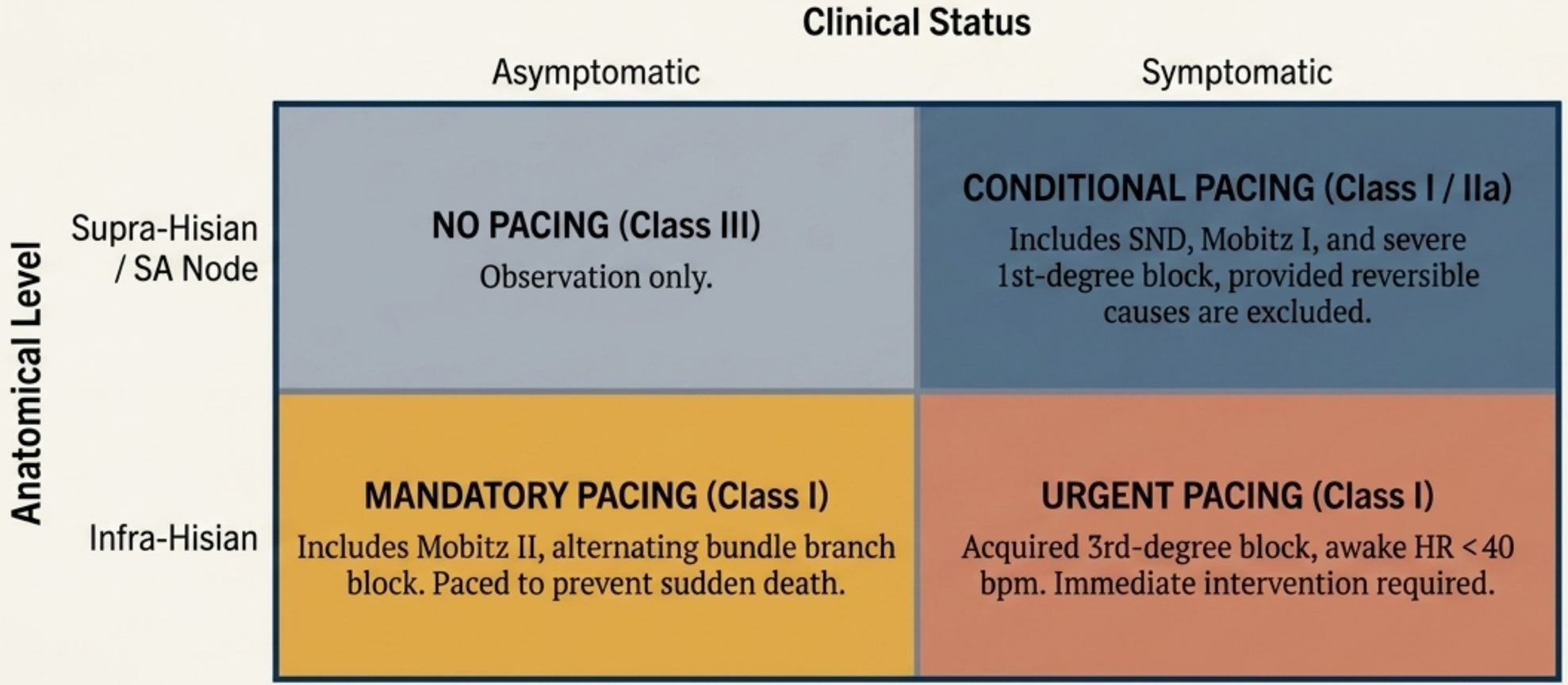
Settings: Synchronised mode, start at 60–70 bpm, current 40–80 mA.

Bridge to urgent transvenous pacing.

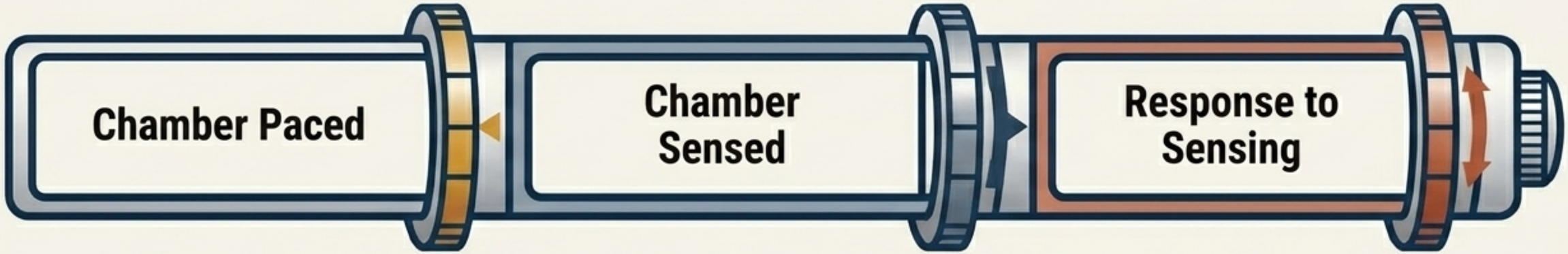
Do not delay for drugs if the patient is unstable.

The Master Pacing Decision Matrix

The 2023 ACC/AHA/HRS guidelines follow a strict underlying logic: distal blocks are inherently lethal, while proximal blocks require proven symptoms.



The Pacemaker Decoder Ring (NBG Codes)



DDD (Dual-Chamber)

Paces Atrium & Ventricle. Senses both. Maintains normal AV synchrony.

Best for: SND with intact AV conduction, or AV block with normal sinus function.

VVI (Single-Chamber Ventricular)

Paces & Senses Ventricle only.

Best for: Permanent Atrial Fibrillation with slow ventricular response (prevents tracking of fibrillatory waves). Causes loss of AV synchrony in sinus rhythm.

Conduction System Pacing (His-Bundle / LBBAP)

Directly paces the native conduction tissue.

Best for: Patients with reduced LVEF or expected ventricular pacing burden >40% to avoid pacing-induced cardiomyopathy.

Programming Parameters & Device Longevity

Rate-Adaptive Programming:

Utilizes activity (accelerometer) or minute ventilation sensors for chronotropic incompetence.

- Lower Rate Limit (LRL): Typically set to 60 bpm (50 bpm for young, active patients).
- Upper Tracking/Sensor Rate (UTR/USR): 120–130 bpm for elderly; 150–170 bpm for active younger patients.

Expected Generator Longevity:

Device replacement is a day-procedure confirmed when battery hits Elective Replacement Indicator (ERI). ERI to End of Service (EOS) is typically 3–6 months.

Device Type	Expected Lifespan
VVIR (Single-Chamber)	10–15 years
DDDR (Dual-Chamber)	8–12 years
His-Bundle / LBBAP	7–10 years (higher capture thresholds required)

Remote Monitoring (Standard of Care):

Trials (IN-TIME, CONNECT, TRUST) demonstrate remote telemonitoring reduces in-clinic visits by 40-50%, drastically decreasing time-to-clinical-action for arrhythmias and lead failures.

Routine schedule: In-clinic at 2-6 weeks, monthly remote transmissions, in-clinic every 6-12 months.

MRI and EMI Navigation:

- MR Conditional: Most modern devices are safe (≤1.5 Tesla, whole-body SAR ≤2 W/kg) but MUST be programmed to asynchronous mode prior to the scan by cardiac staff.
- Safe Daily Use: Mobile phones (contralateral ear), household appliances, induction cooktops, security arches (walk through, don’t linger).
- Avoid: Monopolar electrocautery, diathermy, industrial welding.

Special Populations: Clinical Adjustments

Pregnancy

- Fluoroscopy shielding reduces foetal dose to <1 mGy (safe).
- DDD preferred to maintain cardiac output.
- Screen for congenital AV block if anti-Ro/La antibodies present.

Paediatrics (<15 kg)

- Epicardial pacing preferred over transvenous.
- DDDR to maintain growth/activity response.
- Frequent follow-up (3-6 months) for somatic growth lead adjustments.

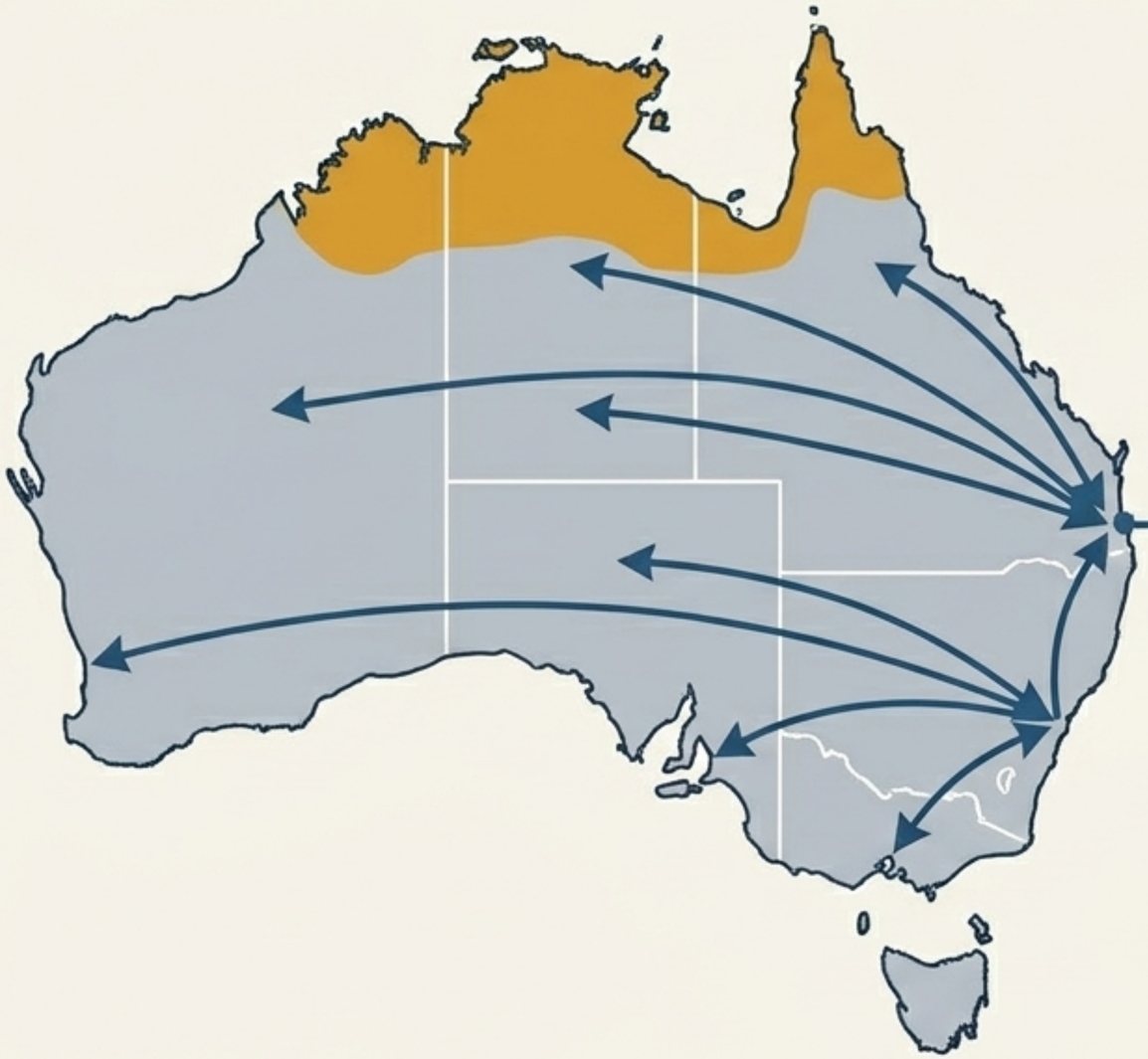
The Elderly (≥75)

- DDD preferred to reduce syncope-related falls.
- VVI acceptable for frail patients with limited mobility.
- Ensure MRI-compatible devices for future cognitive assessments.

Renal & Immunocompromised

- Dialysis: preserve AV fistulas (avoid ipsilateral subclavian).
- Adjust digoxin clearance.
- Immunocompromised: Atypical infection presentations; consider antibacterial envelopes (TYRX™) at implant.

Aboriginal & Torres Strait Islander Health Considerations



The Rheumatic Heart Disease (RHD) Burden:

Indigenous Australians experience RHD at 10-20x the rate of non-Indigenous populations. RHD-related valvular and myocardial disease frequently causes young-onset high-degree AV block. Screen all presenting patients with echocardiography.

Access and Telemetry:

Pacing services are concentrated in metropolitan hubs. Remote monitoring is absolutely critical to bridge the gap. Primary care in remote clinics must be equipped with standardized transcutaneous pacing protocols and aeromedical retrieval pathways.

Cultural Safety:

Involve Aboriginal health practitioners in pre-procedure counseling. Respect cultural obligations affecting appointment attendance. Ensure same-sex practitioners are available for wound checks where requested.

The 2026 Bradyarrhythmia Rapid Reference

1. Exclude Reversible Causes: Meds (Beta-blockers, CCBs, Digoxin), Ischaemia, TSH, K+.

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2. Identify the Anatomical Level:

- **SA Node:** Sinus Brady, Arrest (>5s = pace), Chronotropic incompetence.
- **AV Node (Narrow QRS):** Mobitz I. Atropine responsive. Pace only if symptomatic.
- **His-Purkinje (Wide QRS):** Mobitz II, Complete Heart Block. High mortality. Permanent pacing mandated.

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3. Acute Stabilisation (Unstable): Transcutaneous pacing (60-70 bpm, 40-80 mA). Do not delay for drugs.

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4. Programming: DDD for AV synchrony. VVI for permanent AF. Rate-response enabled for chronotropic incompetence.

Reference: *2026 Med2Date Guidelines & 2023 ACC/AHA/HRS Guidelines for Cardiac Pacing*