Sinus Node Dysfunction

Dr Qurban Hussain Khan

Assistant Professor of Cardiac Electrophysiology

Rawalpindi Institute of Cardiology

Content

- Definitions
- ECG Diagnoses
- Evaluation
- Management

What is Sinus Node Dysfunction (SND)?

- SND is often related to **age dependent progressive fibrosis** of the sinus nodal tissue and surrounding atrial myocardium.
- This leads to **abnormalities of sinus node and atrial impulse formation and propagation**.
- Manifests as **bradycardic or pause-related syndromes**.

What is the basis of management?

- In sinus node dysfunction, there is **no established minimum heart rate or pause duration where permanent pacing is recommended**.
- Establishing **temporal correlation between symptoms and bradycardia** is important when determining whether permanent pacing is needed.

Epidemiology of bradycardias

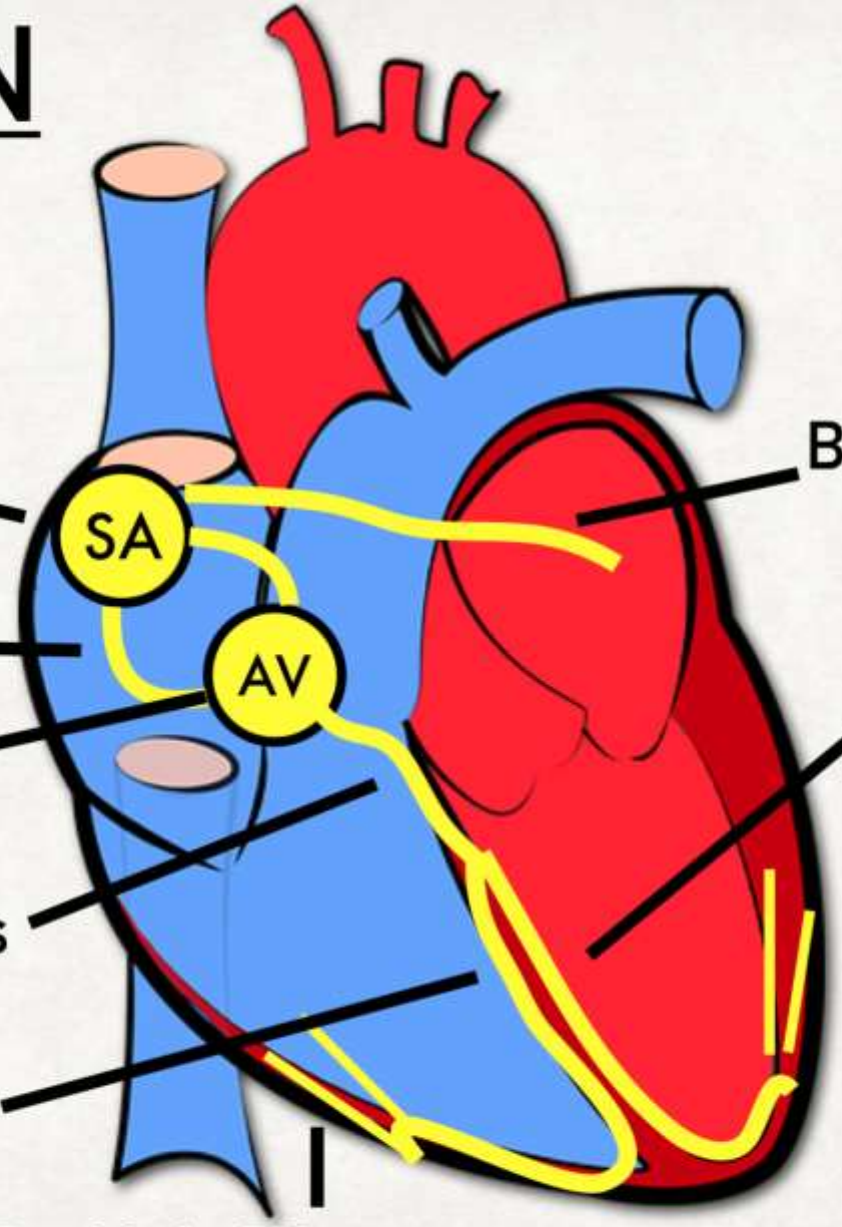
- Because slower heart rates and changes in intercellular conduction can be observed as both part of normal aging and disease progression, **bradycardia and conduction abnormalities are more commonly identified in the elderly**.
- Most common in individuals in 70s and 80s.

Types of bradycardias

- Abnormalities of the sinus node, atrial tissue, atrioventricular nodal tissue, and the specialized conduction system can all contribute to bradycardia, discordant timing of atrial and ventricular depolarization (AV block), and abnormal ventricular depolarization.

CONDUCTION

1. SA Node
2. AV Node
3. Bundle of His
4. R/L Bundle Branches
5. Purkinje Fibers



SA Node

Internodal Pathway

AV Node

Bundle of His

Right Bundle Branch

Purkinje Fibers

Bachmann's Bundle

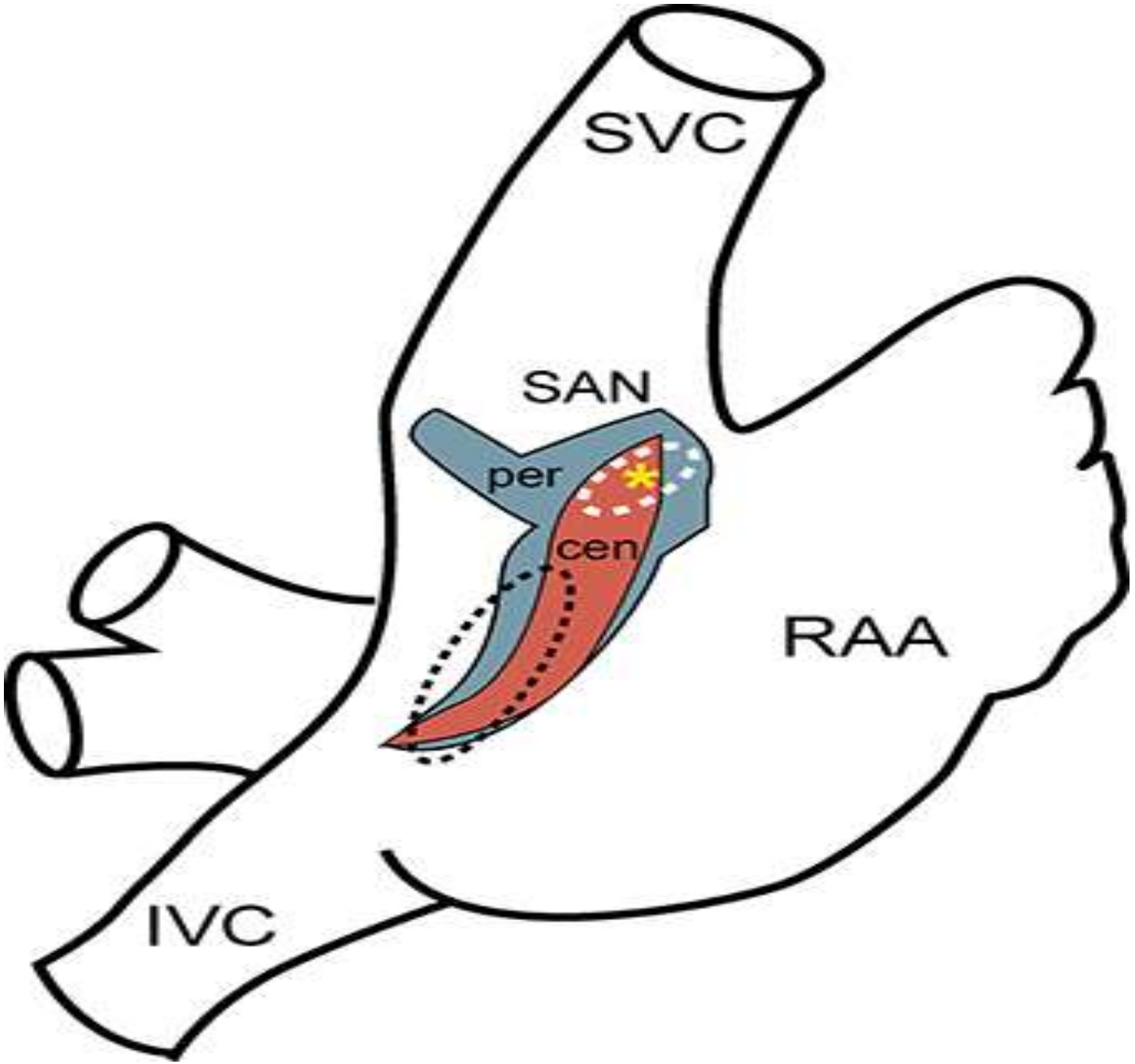
Left Bundle Branch

Purkinje Fibers

The Structure of the Sinus Node

It is an oval shaped region of special cardiac muscle in the upper back wall of the right atrium made up of cells known as **pacemaker cells**.

The sinus node is approximately 15 mm long, 3 mm wide, and 1 mm thick, located directly below and to the side of the superior vena cava.



SND = “Sick Sinus Syndrome”

- Sinus node dysfunction (SND), historically referred to as sick sinus syndrome, is most often related to **age-dependent, progressive, degenerative fibrosis of the sinus nodal tissue and surrounding atrial myocardium**.

Atrial tachyarrhythmias can co-exist

- In addition, the same milieu of degenerative fibrosis is also responsible for the development of atrial tachyarrhythmias, which **can coexist with sinus node disease and the combination often called “tachybrady syndrome.”**

Intrinsic causes of SND

- Idiopathic Degenerative Fibrosis (commonest).
- Ischemia.
- Cardiomyopathies.
- Infiltrative Diseases e.g. sarcoidosis, haemochromatosis.
- Congenital abnormalities.

Extrinsic Causes

- Drugs e.g. digoxin, beta-blockers, calcium channel blockers.
- Autonomic dysfunction.
- Hypothyroidism.
- Electrolyte abnormalities — e.g. hyperkalaemia.

ECG Diagnoses of SND

TABLE 3 Table of Definitions

Term	Definition or Description
Sinus node dysfunction (with accompanying symptoms)	■ Sinus bradycardia: Sinus rate <50 bpm ■ Ectopic atrial bradycardia: Atrial depolarization attributable to an atrial pacemaker other than the sinus node with a rate <50 bpm ■ Sinoatrial exit block: Evidence that blocked conduction between the sinus node and adjacent atrial tissue is present. Multiple electrocardiographic manifestations including "group beating" of atrial depolarization and sinus pauses. ■ Sinus pause: Sinus node depolarizes >3 s after the last atrial depolarization ■ Sinus node arrest: No evidence of sinus node depolarization ■ Tachycardia-bradycardia ("tachy-brady") syndrome: Sinus bradycardia, ectopic atrial bradycardia, or sinus pause alternating with periods of abnormal atrial tachycardia, atrial flutter, or AF (S2.2-6). The tachycardia may be associated with suppression of sinus node automaticity and a sinus pause of variable duration when the tachycardia terminates. ■ Chronotropic incompetence: Broadly defined as the inability of the heart to increase its rate commensurate with increased activity or demand, in many studies translates to failure to attain 80% of expected heart rate reserve during exercise. ■ Isorhythmic dissociation: Atrial depolarization (from either the sinus node or ectopic atrial site) is slower than ventricular depolarization (from an atrioventricular nodal, His bundle, or ventricular site).

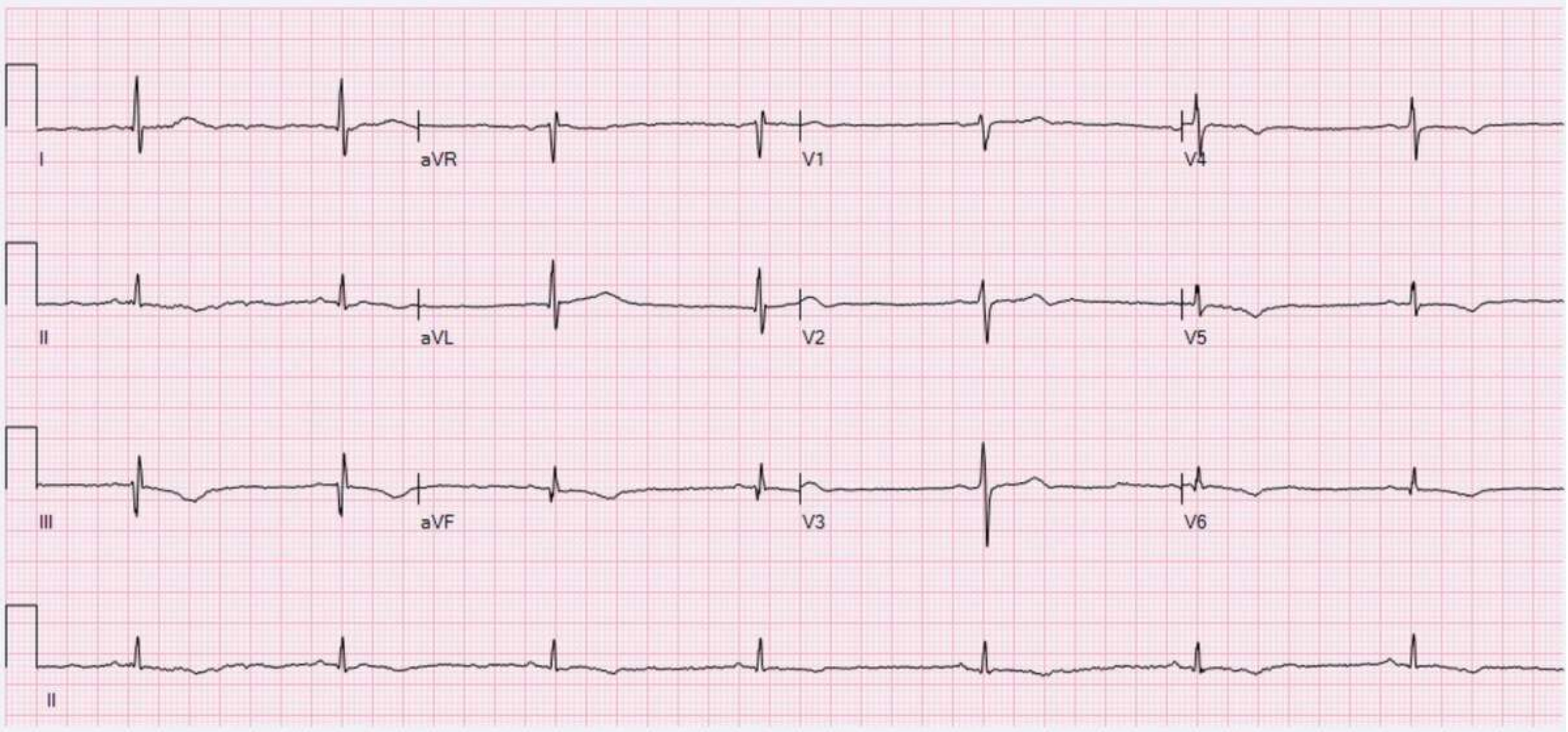
What does a Sinus Bradycardia ECG show?

- A normal P wave vector
- A normal P wave axis
- A P wave before every QRS complex
- A 1:1 conduction ratio between the atria and ventricles

Sinus Bradycardia ECG



Sinus Bradycardia ECG



Ectopic Atrial Bradycardia

- **Irregular P wave morphology:**

The P waves will look different from a normal sinus P wave, with variations in shape and axis depending on the location of the ectopic focus within the atrium.

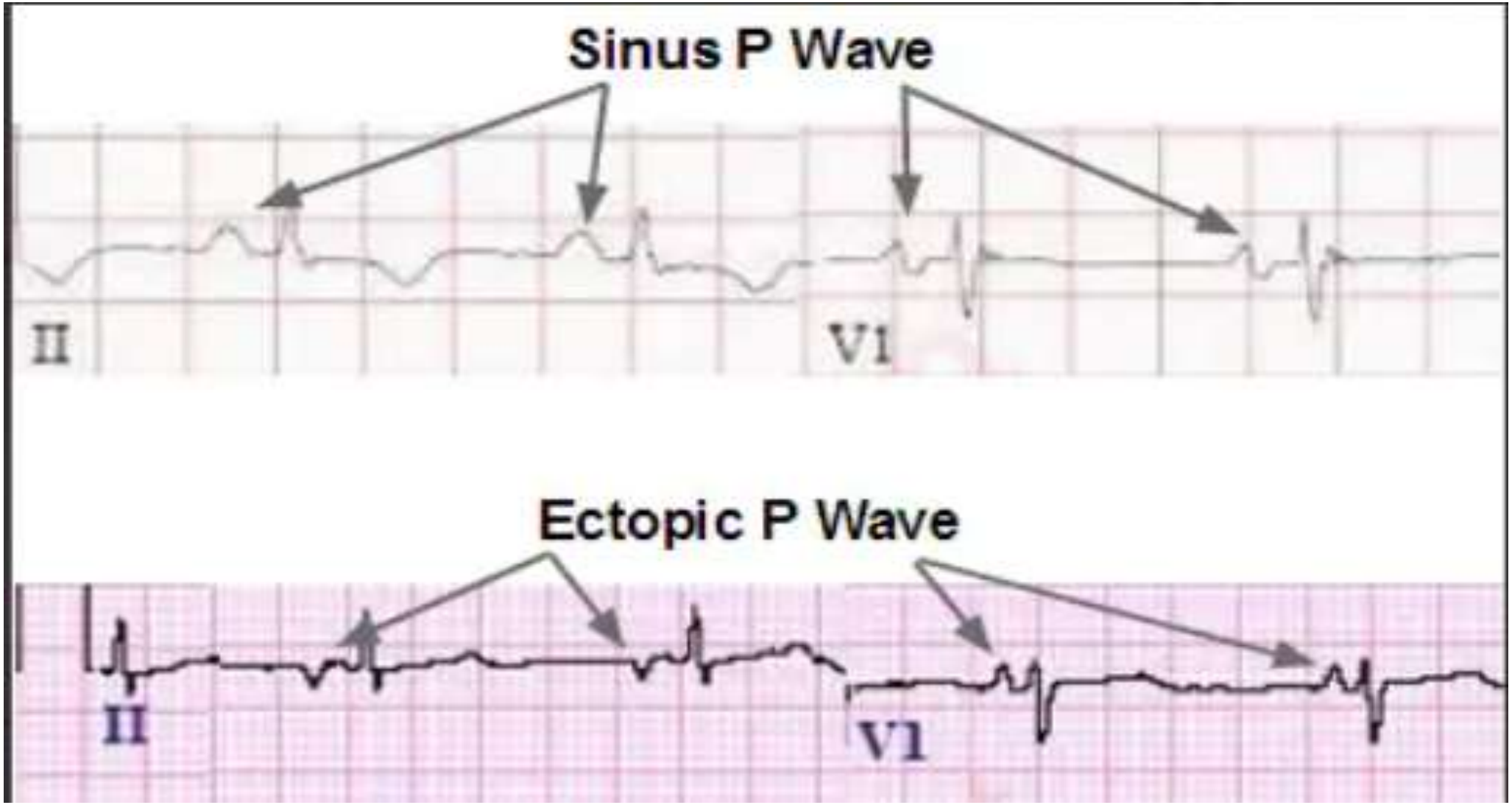
- **Regular rhythm:**

Although the P waves may be different, the rhythm itself will be regular, meaning the time between each beat is consistent.

- **Slow heart rate:**

The overall heart rate will be below 50 beats per minute, classifying it as bradycardia.

Ectopic Atrial Beats



Sinoatrial Exit Block

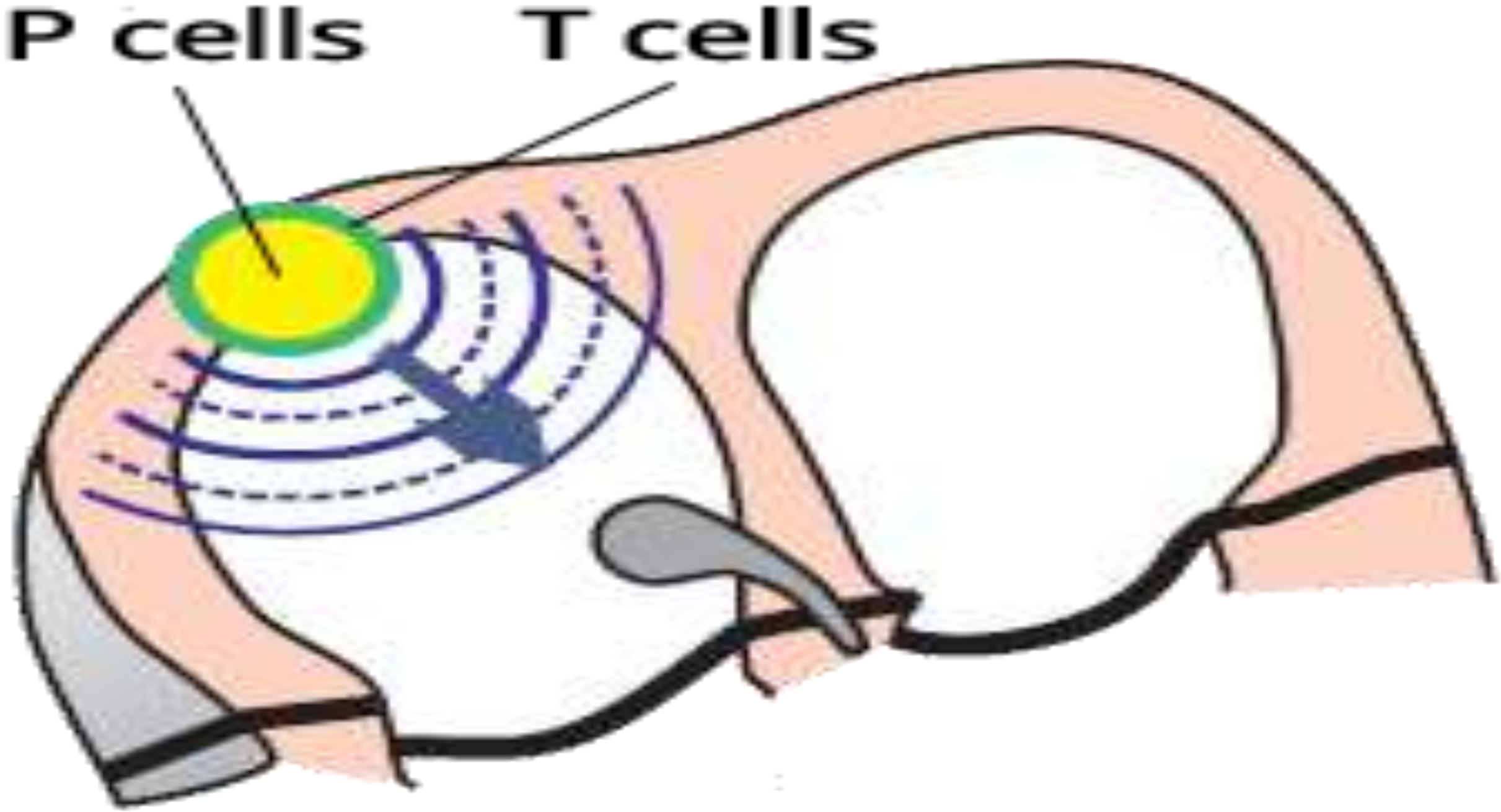
- Sinoatrial exit block is due to failed propagation of pacemaker impulses beyond the SA node.
- The sinoatrial node continues to depolarise normally.
- However, some of the **sinus impulses are "blocked" before they can leave the SA node**, leading to intermittent failure of atrial depolarisation (**dropped P waves**).

Anatomical basis of sinoatrial exit block

- The SA node consists of two main groups of cells.
- A central core of *pacemaking cells* (**P cells**) that produce the sinus impulses.
- An outer layer of *transitional cells* (**T cells**) that transmit the sinus impulses out into the right atrium.

What is the mechanism of SA exit block?

- Sinus node dysfunction can result from one of the following:
- 1. **Failure of the P cells to produce an impulse**. This leads to sinus pauses and sinus arrest.
- 2. **Failure of the T cells to transmit the impulse**. This leads to sino-atrial exit block.


P cells
T cells

Patterns of Conduction

- The patterns of conduction in SA exit block are identical to the different types of AV block.
- However, as the initial sinus impulse is not visible on the ECG, the relationship between impulse generation and transmission must be **inferred from the P waves alone** (analogous to examining only the R waves in AV block).
- **Only second degree SA block (types I and II) can be diagnosed from the 12-lead ECG**.

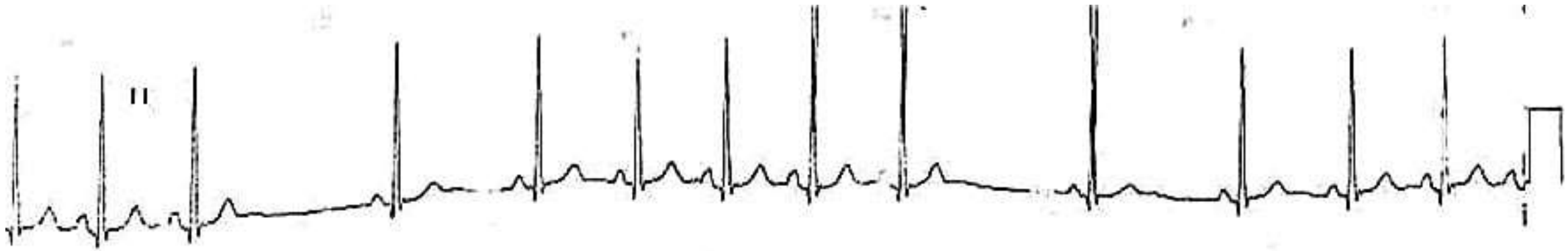
First Degree SA Block

- *= Delay between impulse generation and transmission to the atrium.*
- This abnormality is **not detectable on the surface ECG**.

2nd Degree SA Block, Type I (Wenkebach)

- *= Progressive lengthening of the interval between impulse generation and transmission, culminating in failure of transmission.*
- The gradually lengthening transmission interval pushes successive P waves closer together.
- This results in grouping of the P-QRS complexes.
- Pauses due to dropped P waves occur at the end of each group.
- The **P-P interval progressively *shortens* prior to the dropped P wave**.
- This pattern is easily mistaken for sinus arrhythmia.

2nd Degree SA Block, Type I (Wenkebach)



2nd Degree SA Block, Type II

- *= Intermittent dropped P waves with a constant interval between impulse generation and atrial depolarisation.*
- This pattern is the equivalent of Mobitz II.
- There is no clustering of P-QRS complexes.
- Intermittent P waves “drop out” of the rhythm, while subsequent P waves arrive “on time”.
- The **pause surrounding the dropped P wave is an *exact multiple* of the preceding P-P interval**.

2nd Degree SA Block, Type II



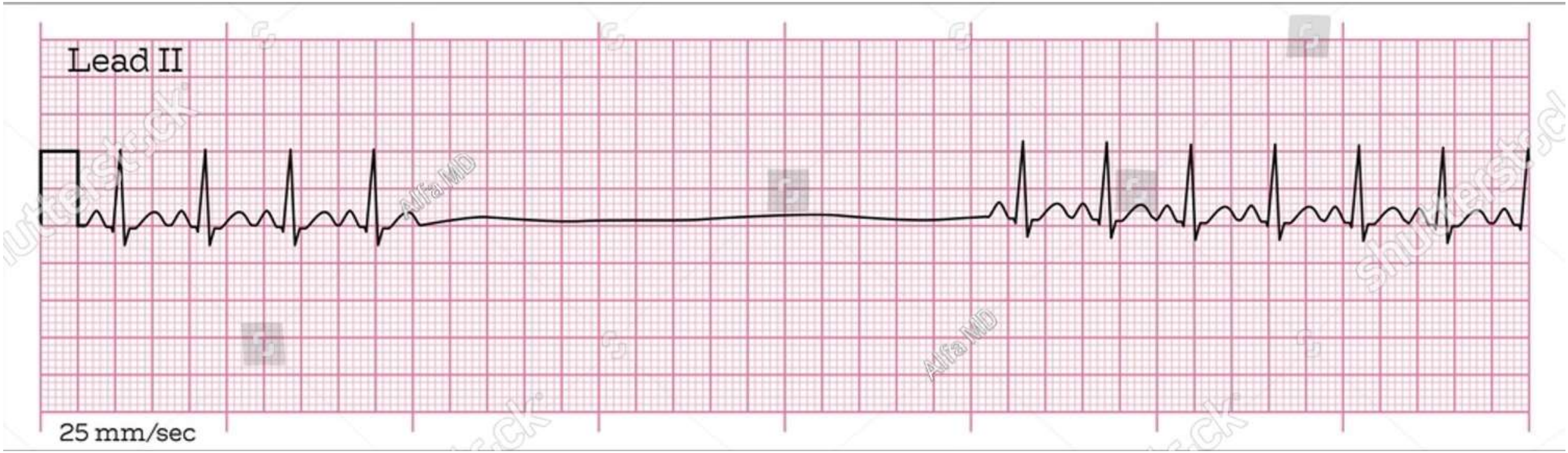
•Arrows indicate the presumed timing of each sinus impulse.
•The blue arrows represent normally transmitted impulses, i.e. resulting in P waves.
•The black arrows represent blocked sinus impulses (dropped P waves).
•The pauses around the dropped P waves (2.1 seconds) are **exactly double the preceding P-P interval** (1.05 seconds)

•The 4th QRS complex is a junctional escape beat followed by a non-conducted P wave (occurring just prior to the T wave).
•The 8th QRS complex is a junctional escape beat. The following P wave *is* conducted to the ventricles, albeit with an extremely long PR interval (400ms).

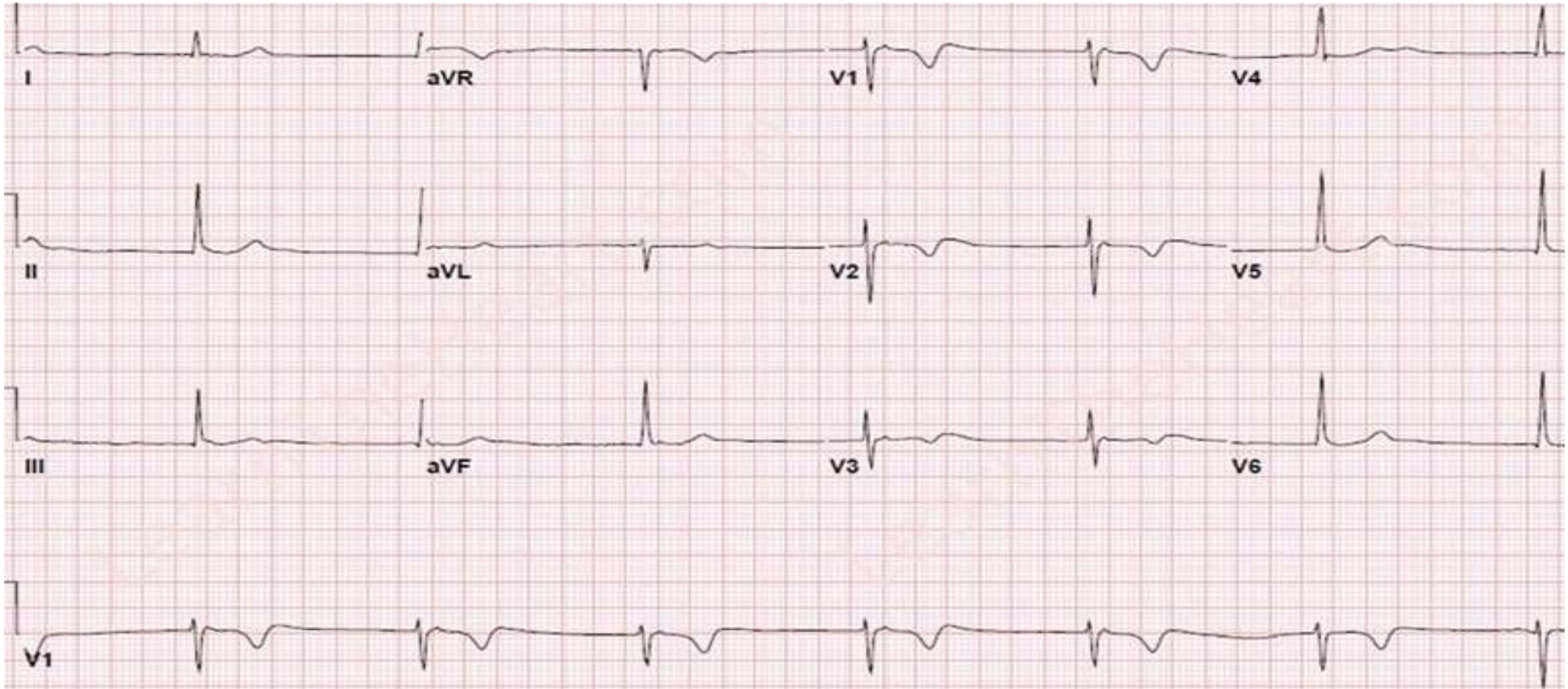
3rd Degree SA Block

- *= None of the sinus impulses are conducted to the right atrium.*
- There is a complete absence of P waves.
- The onset of 3rd degree SA block may produce long sinus pauses or sinus arrest (may lead to fatal asystole).
- Rhythm may be maintained by a junctional escape rhythm.
- **Third degree SA exit block is indistinguishable from sinus arrest due to pacemaker cell failure**. It can only be diagnosed with a sinus node electrode during electrophysiological evaluation.

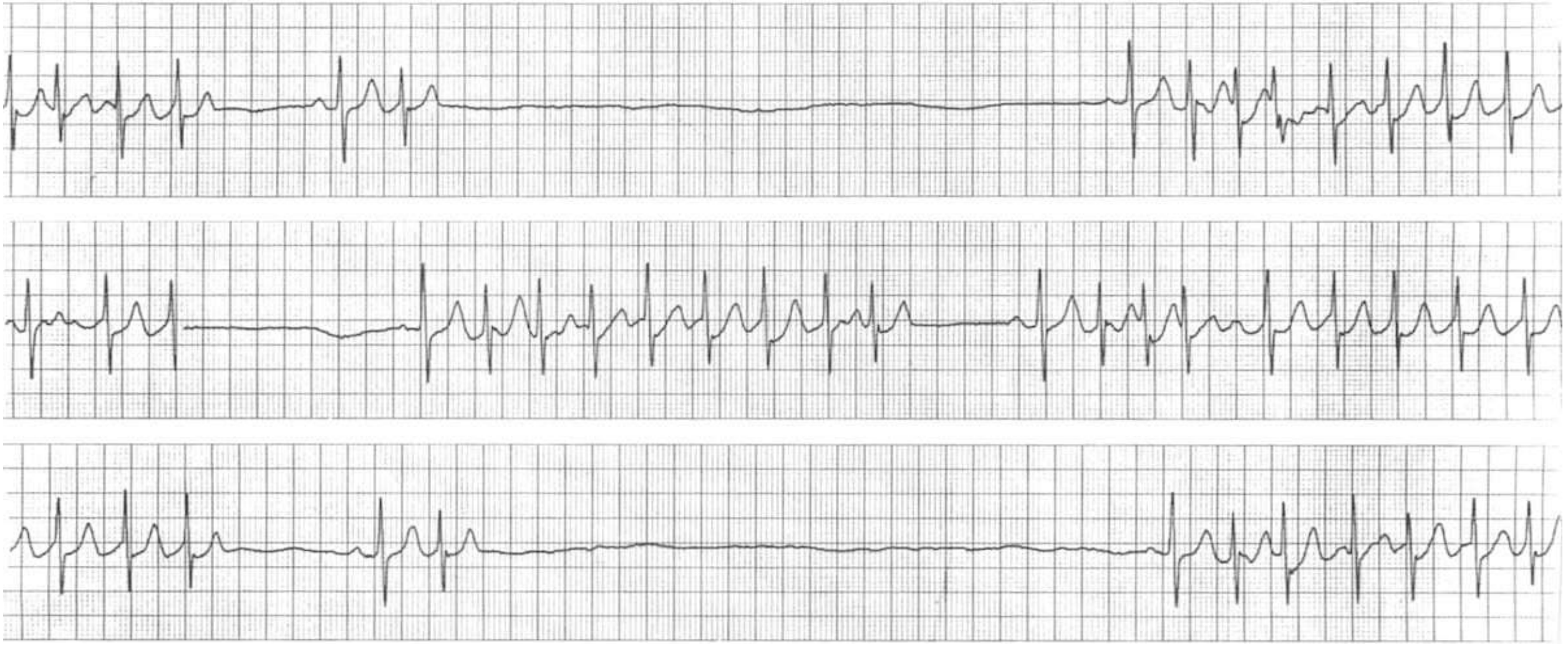
Sinus Pause (SND when >3 seconds)



Sinus Arrest = no evidence of sinus node activity/depolarisation



Tachy-brady Syndrome



Chronotropic Incompetence

- Chronotropic incompetence (CI) is a condition where the heart rate does not increase enough during exercise.
- Common in people with cardiovascular disease and can lead to exercise intolerance and a higher risk of death.
- Symptoms: Difficulty exercising, Reduced functional capacity, and Poor survival.
- Causes: sinus node dysfunction, conduction abnormalities, chronic atrial fibrillation, aging, and medication.

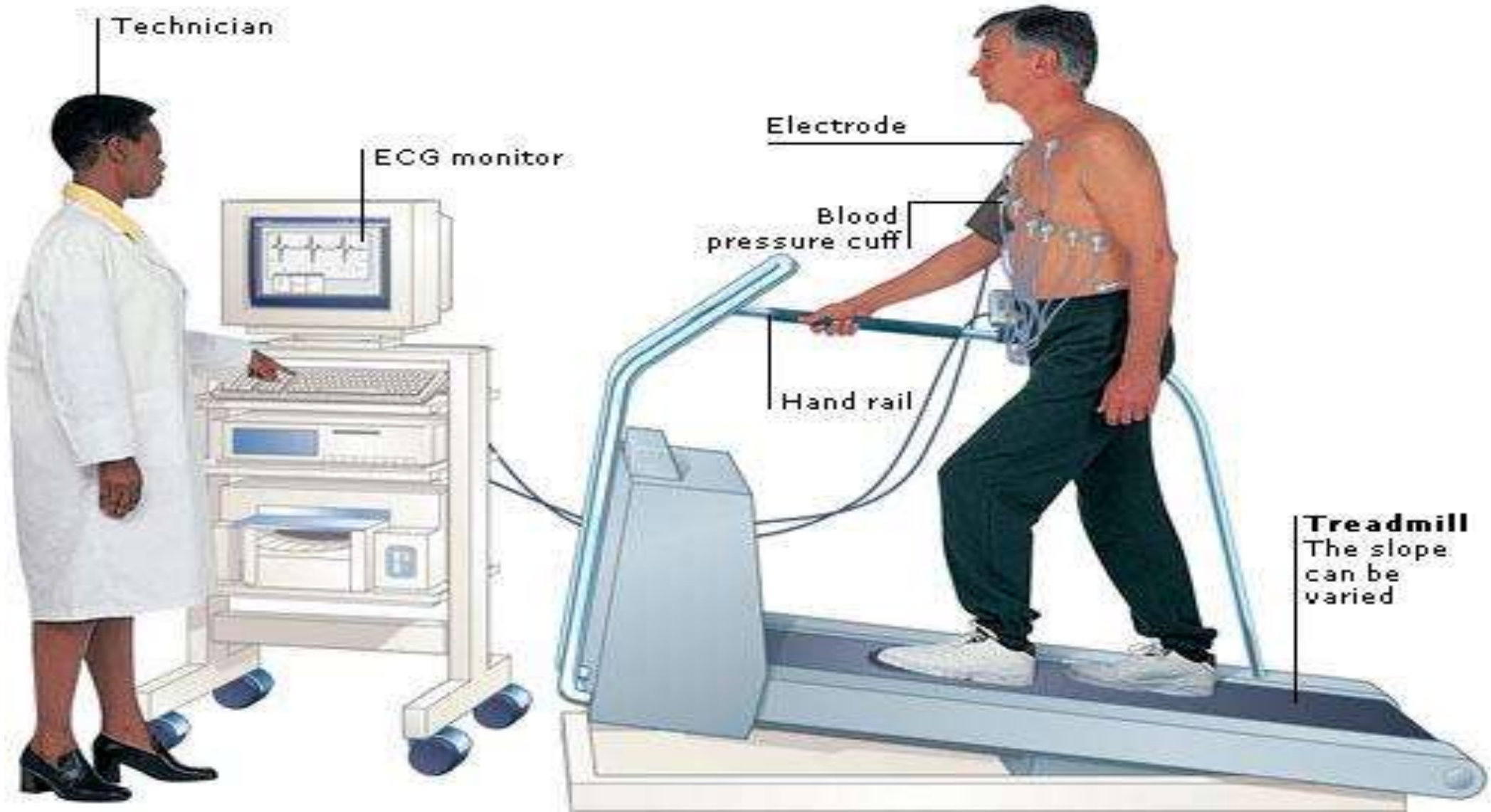
Chronotropic Incompetence (CI)

- Diagnosis: Formal exercise testing is required for a definitive diagnosis.
- CI can be defined as a **peak exercise heart rate that's less than 80–85% of the maximum age predicted heart rate** (Max-PPHR)
- Treatment: CI is potentially treatable and can lead to significant improvements in exercise tolerance and quality-of-life
- Risk: CI is an independent predictor of major adverse cardiovascular events and overall mortality

Age-Predicted Maximal Heart Rate (APMHR) Equations

Authors	APMHR Equation
Fox et al. (1971)	APMHR = 220 – Age
Tanaka et al. (2001)	APMHR = 208 – (0.7 x Age)

Stress Test


Technician
ECG monitor
Electrode
Blood pressure cuff
Hand rail
Treadmill
The slope can be varied

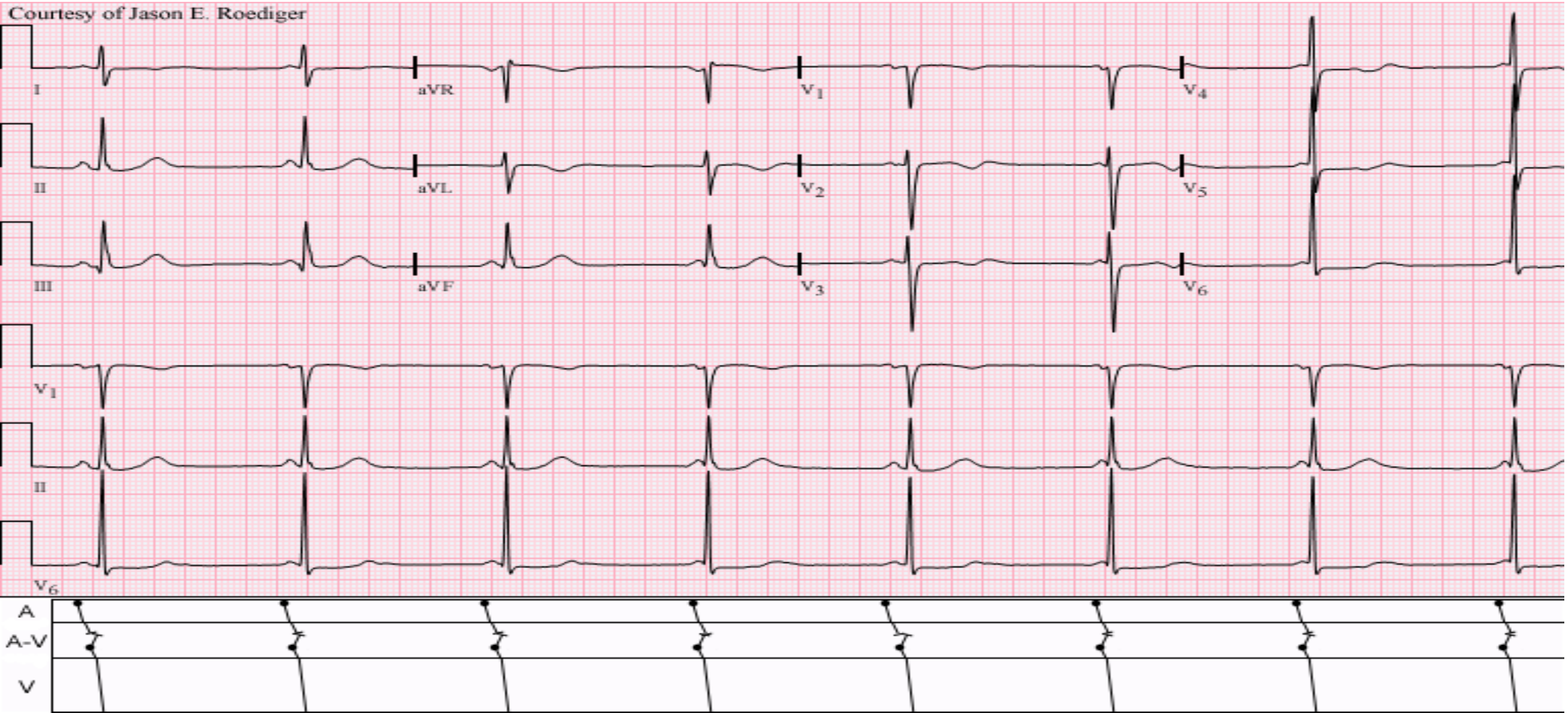
Isorhythmic Dissociation

- The dominant pacemaker slows down
- The latent pacemaker speeds up
- Retrograde ventricular impulses cause a **functional block** at the AV node

Isorhythmic Dissociation

- The P wave can appear before, after or under the QRS complex
- The P wave's position may vary cyclically
- The P wave may have a slightly constant position relative to the QRS

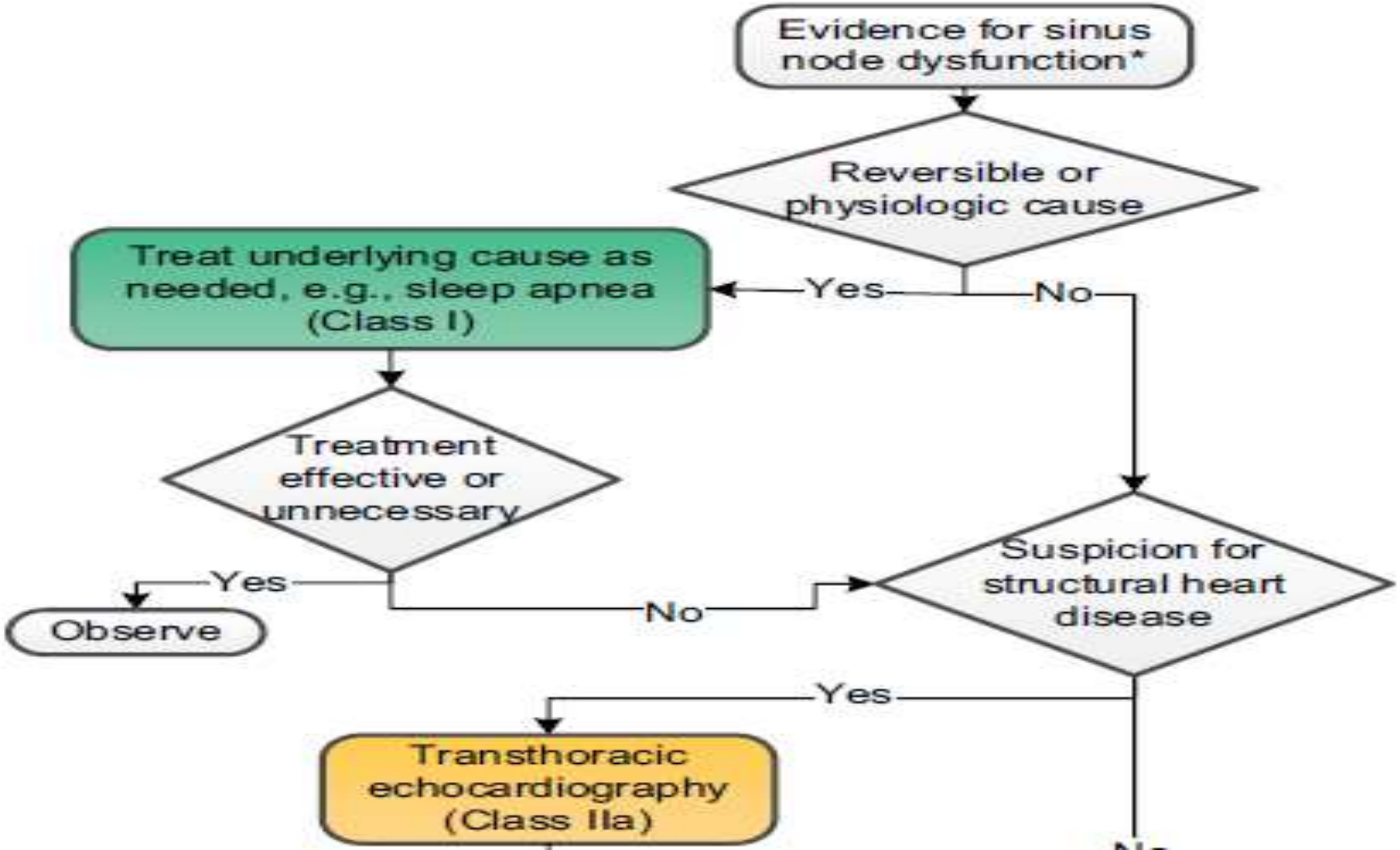
Isorhythmic Dissociation

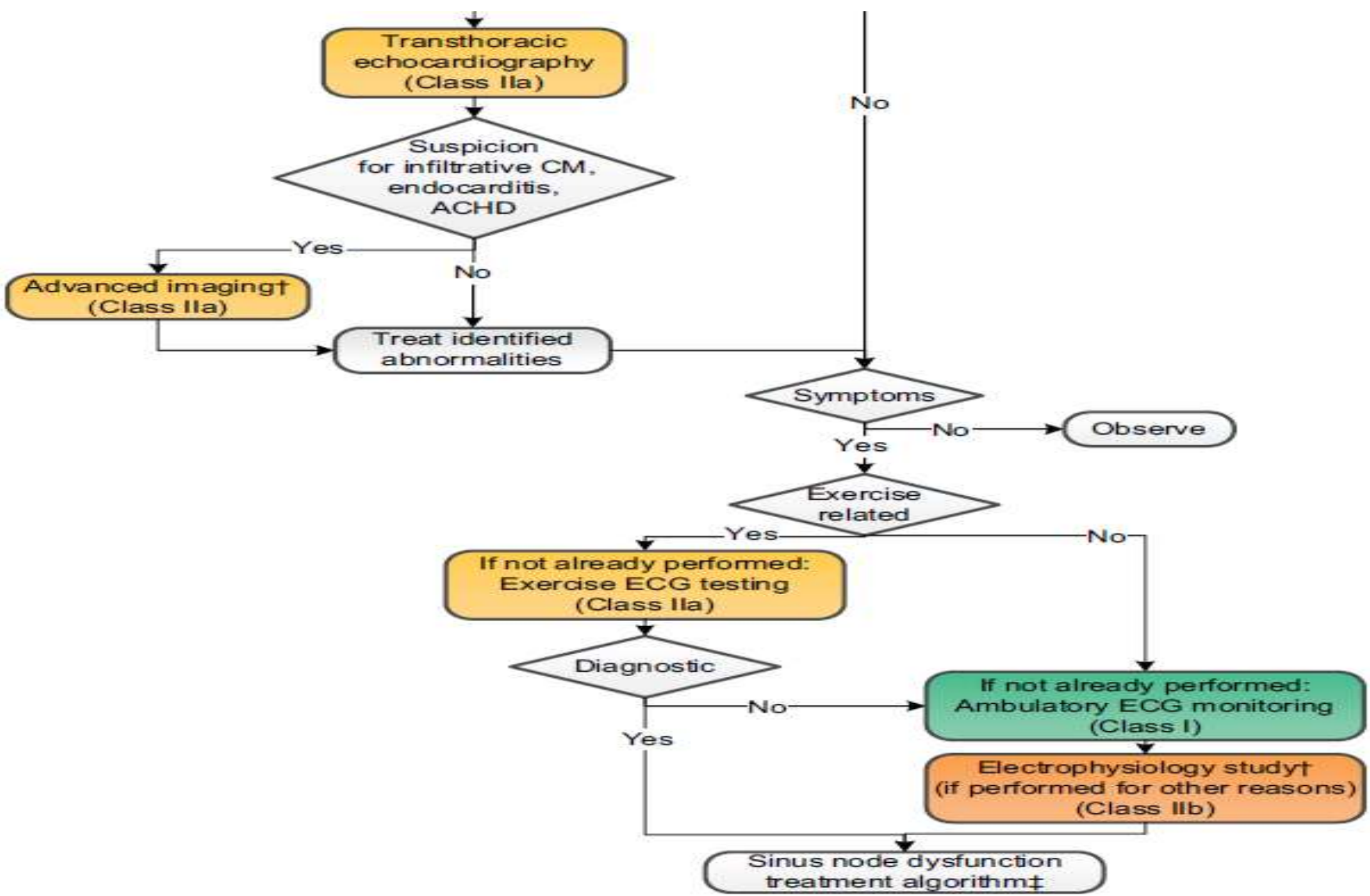


Evaluation and Management

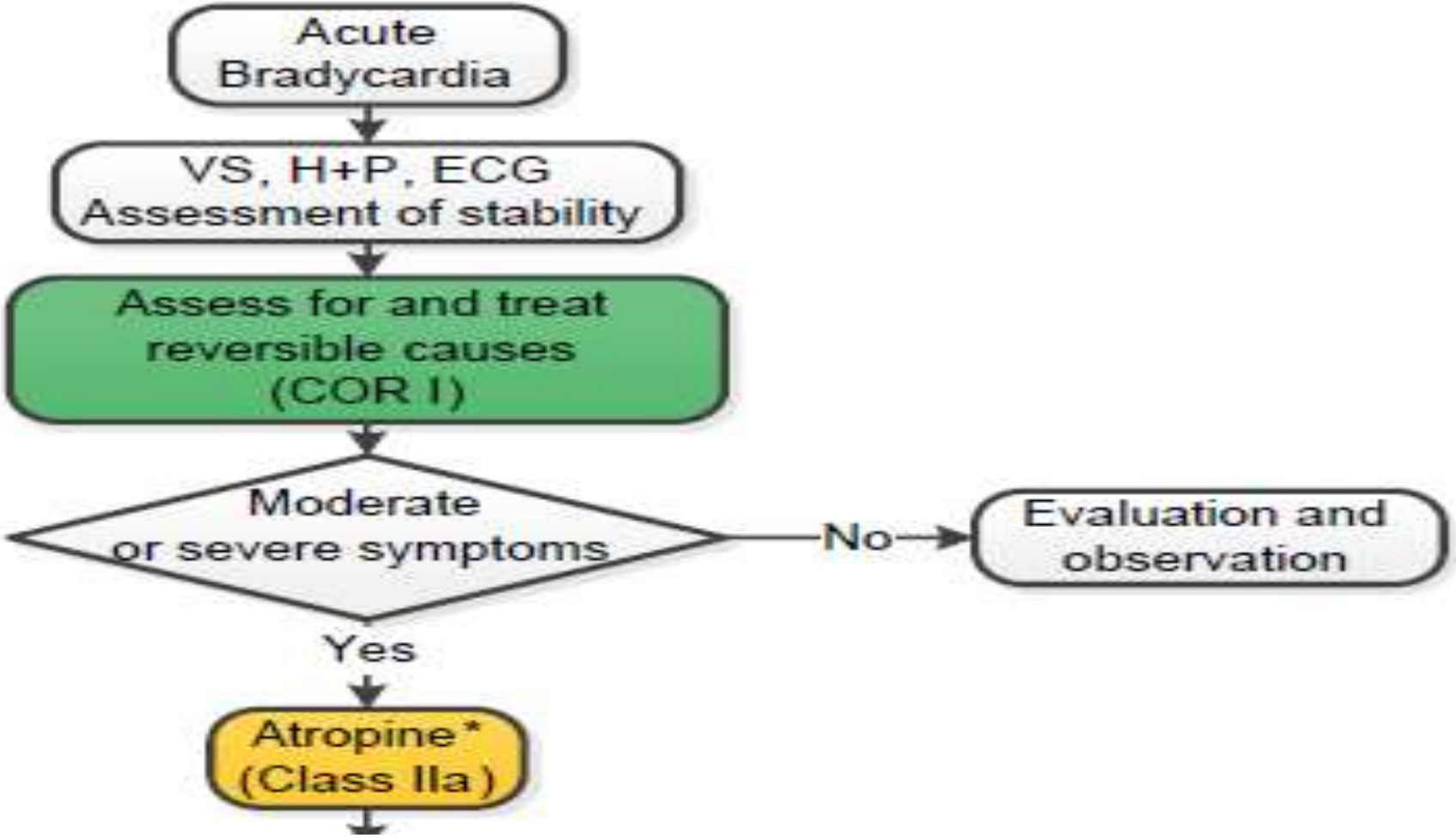
Symptoms are what really matter

SND – <u>FIRST find & treat reversible causes</u>

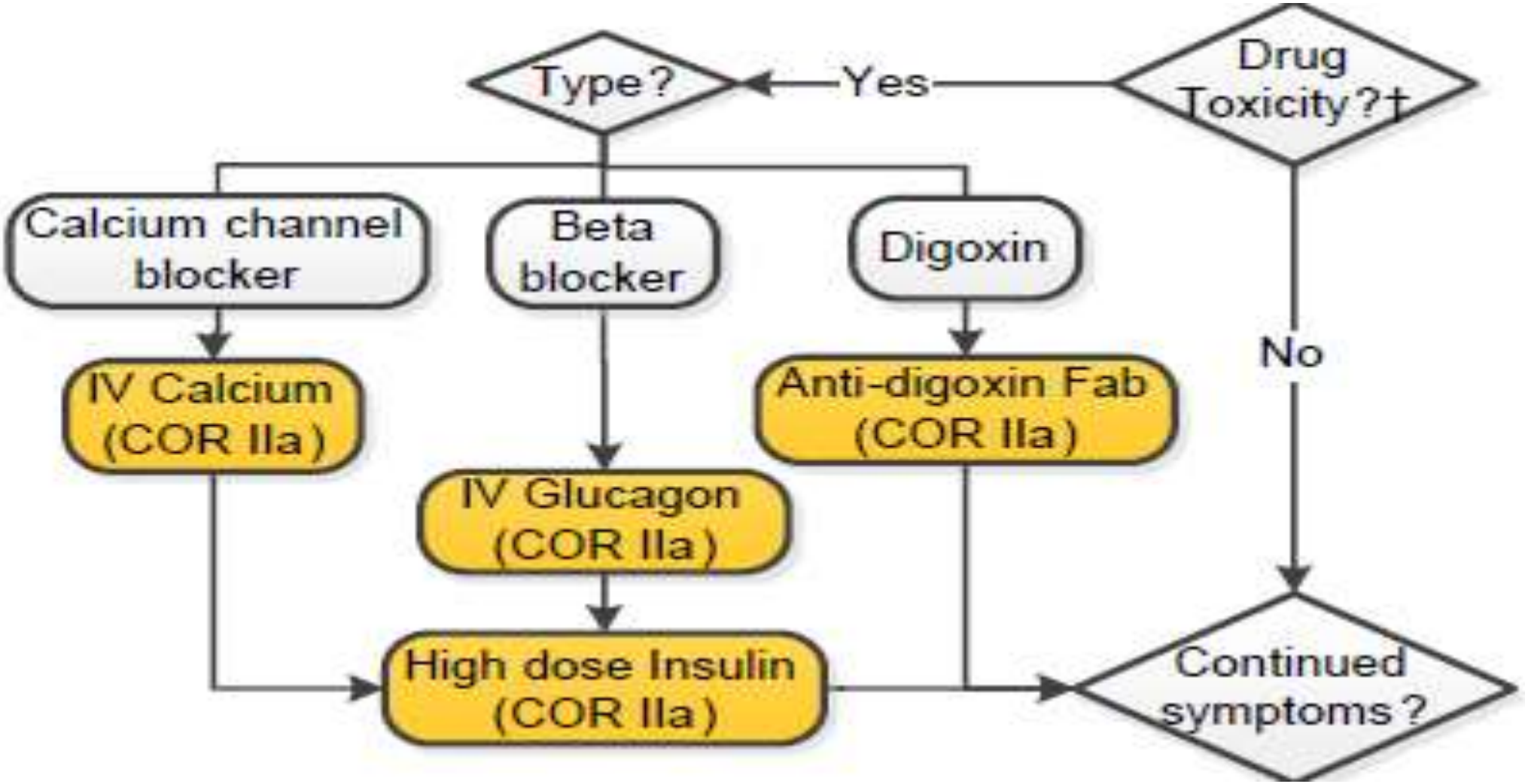



Transthoracic echocardiography (Class IIa)
Suspicion for infiltrative CM, endocarditis, ACHD
Yes
No
Advanced imaging† (Class IIa)
Treat identified abnormalities
No
Symptoms
No
Observe
Yes
Exercise related
Yes
No
If not already performed: Exercise ECG testing (Class IIa)
Diagnostic
No
Yes
If not already performed: Ambulatory ECG monitoring (Class I)
Electrophysiology study† (if performed for other reasons) (Class IIb)
Sinus node dysfunction treatment algorithm‡

Acute Management of SND



Next: Assess for Drug Causes



If severe symptoms then PACE

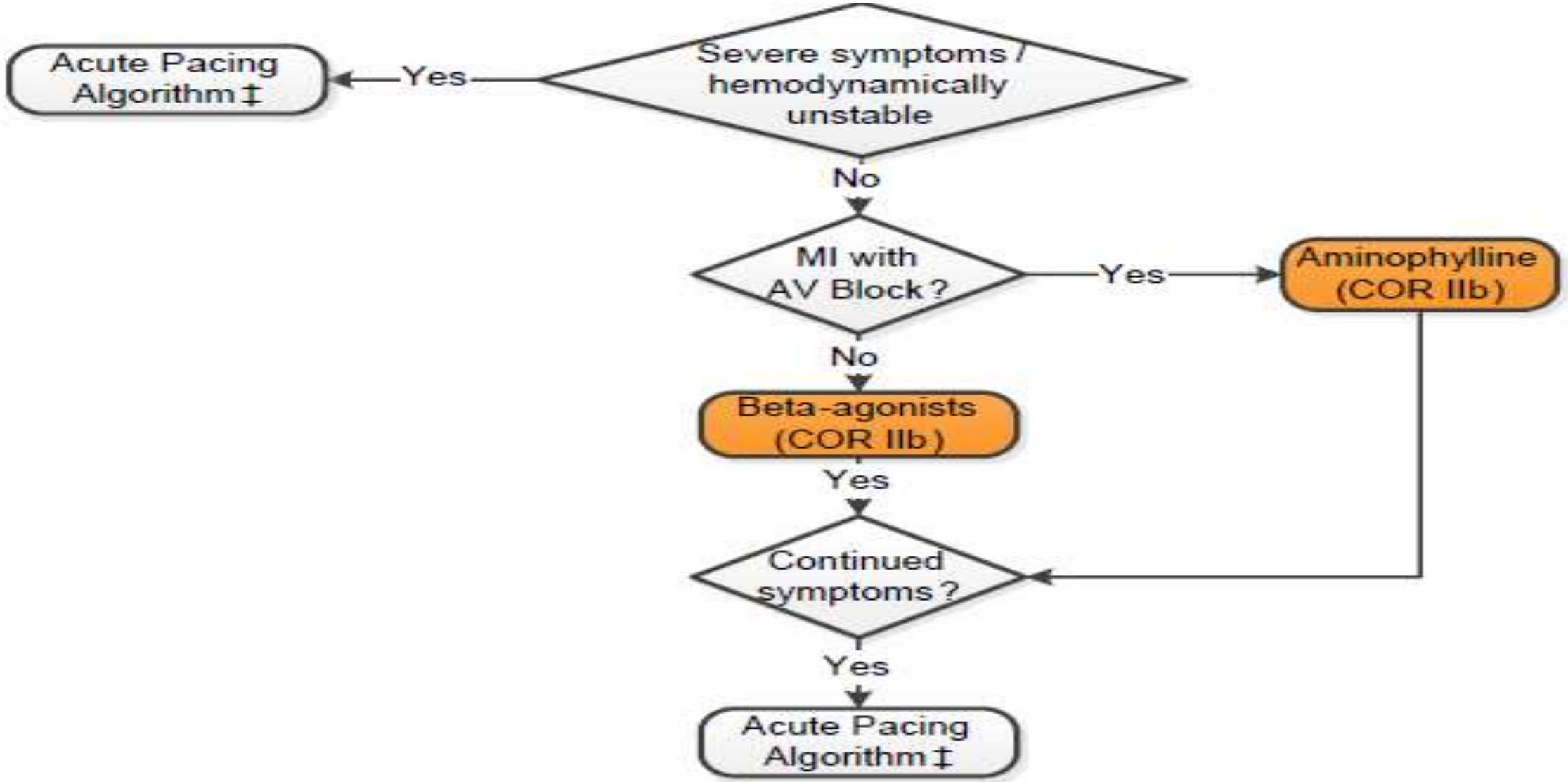
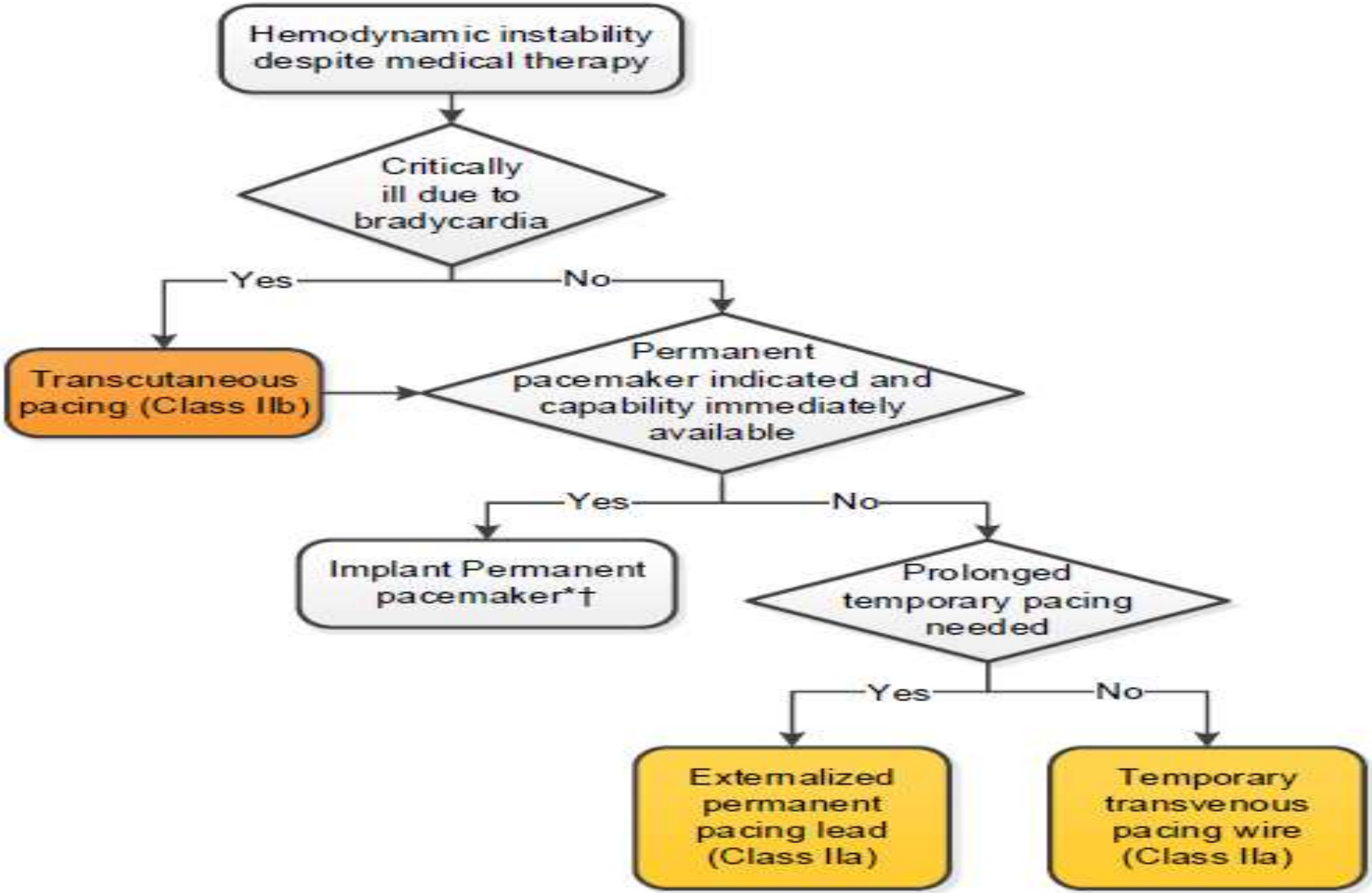
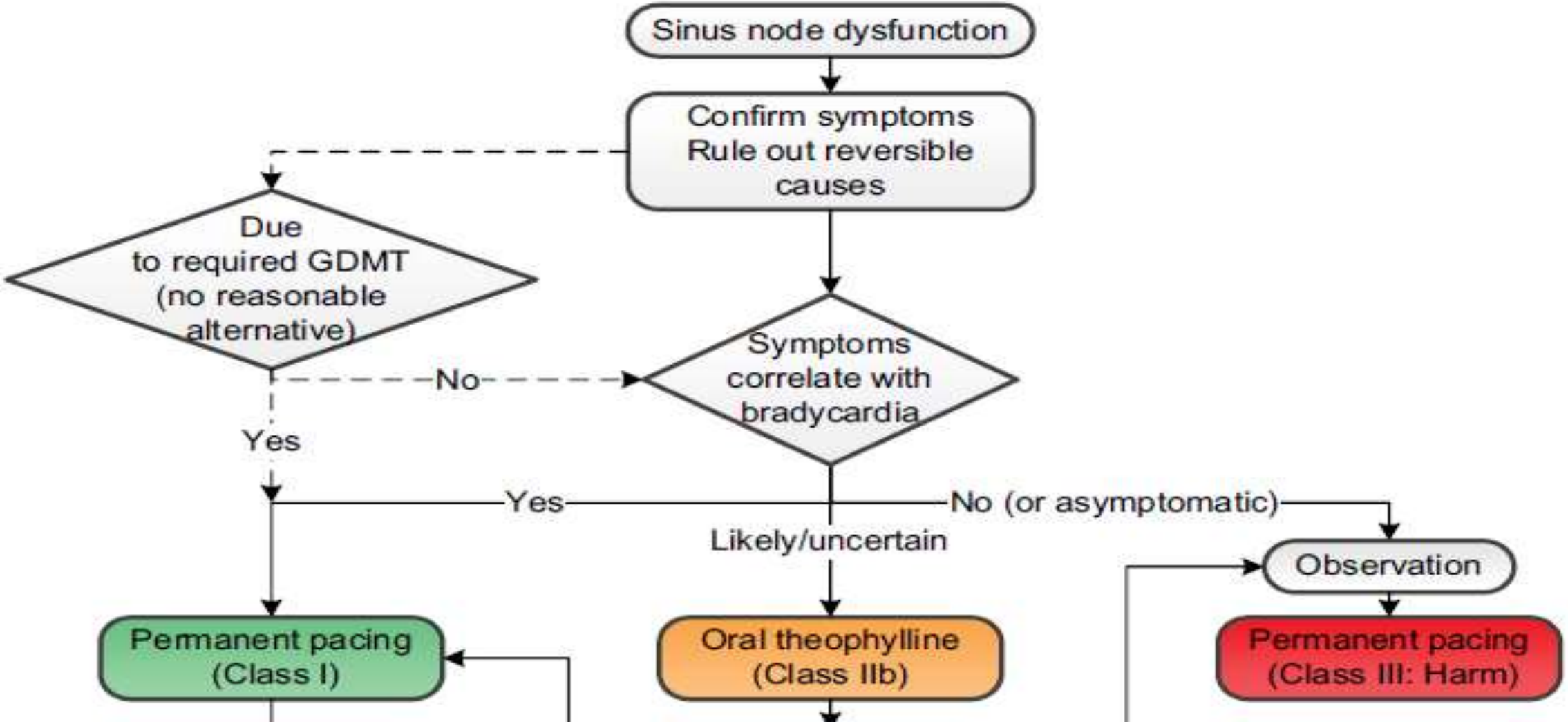


TABLE 8 Acute Medical Management of Bradycardia Attributable to SND or Atrioventricular Block

Medication	Dosage	Comments
Symptomatic sinus bradycardia or atrioventricular block		
Atropine	0.5-1 mg IV (may be repeated every 3-5 min to a maximum dose of 3 mg) (S5.3.2.4-20–S5.3.2.4-24)	
Dopamine	5 to 20 mcg/kg/min IV, starting at 5 mcg/kg/min and increasing by 5 mcg/kg/min every 2 min (S5.3.2.4-25)	Dosages of >20 mcg/kg/min may result in vasoconstriction or arrhythmias
Isoproterenol	20-60 mcg IV bolus followed doses of 10-20 mcg, or infusion of 1-20 mcg/min based on heart rate response (S5.3.2.4-26–S5.3.2.4-32)	Monitor for potential development of ischemic chest pain
Epinephrine	2-10 mcg/min IV or 0.1-0.5 mcg/kg/min IV titrated to desired effect (S5.3.2.4-17, S5.3.2.4-31, S5.3.2.4-33)	
Second- or third-degree atrioventricular block associated with acute inferior MI		
Aminophylline	250-mg IV bolus	
Calcium channel blocker overdose		
10% calcium chloride	1-2 g IV every 10-20 min or an infusion of 0.2-0.4 mL/kg/h (S5.3.2.4-34–S5.3.2.4-36)	
10% calcium gluconate	3-6 g IV every 10-20 min or an infusion at 0.6-1.2 mL/kg/h (S5.3.2.4-34–S5.3.2.4-36)	
Beta-blocker or calcium channel blocker overdose		
Glucagon	3-10 mg IV with infusion of 3-5 mg/h (S5.3.2.4-37, S5.3.2.4-38)	
High dose insulin therapy	IV bolus of 1 unit/kg followed by an infusion of 0.5 units/kg/h (S5.3.2.4-36, S5.3.2.4-39, S5.3.2.4-40).	Follow glucose and potassium levels
Digoxin overdose		
Digoxin antibody fragment	Dosage is dependent on amount ingested or known digoxin concentration (S5.3.2.4-41–S5.3.2.4-48)	■ One vial binds approximately 0.5 mg of digoxin ■ Administer over at least 30 min ■ May be repeated
Post-heart transplant		
Aminophylline	6 mg/kg in 100-200 mL of IV fluid over 20-30 min	
Theophylline	300 mg IV, followed by oral dose of 5-10 mg/kg/d titrated to effect	■ Therapeutic serum levels range from 10-20 mcg/mL ■ Usual posttransplant dosages average 450 mg±100 mg/d
Spinal cord injury		
Aminophylline	6 mg/kg in 100-200 mL of IV fluid over 20-30 min (S5.3.2.4-7)	
Theophylline	Oral dose of 5-10 mg/kg/d titrated to effect (S5.3.2.4-6)	Effective dosages often result in serum levels below the usual effective range of 10-20 mcg/mL


Hemodynamic instability despite medical therapy
Critically ill due to bradycardia
Yes
No
Transcutaneous pacing (Class IIb)
Permanent pacemaker indicated and capability immediately available
Yes
No
Implant Permanent pacemaker*†
Prolonged temporary pacing needed
Yes
No
Externalized permanent pacing lead (Class IIa)
Temporary transvenous pacing wire (Class IIa)

Final Disposal of SND



Thank You for listening