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10:30-12:00
15 min

Sinus node dysfunction

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Conflict of interest

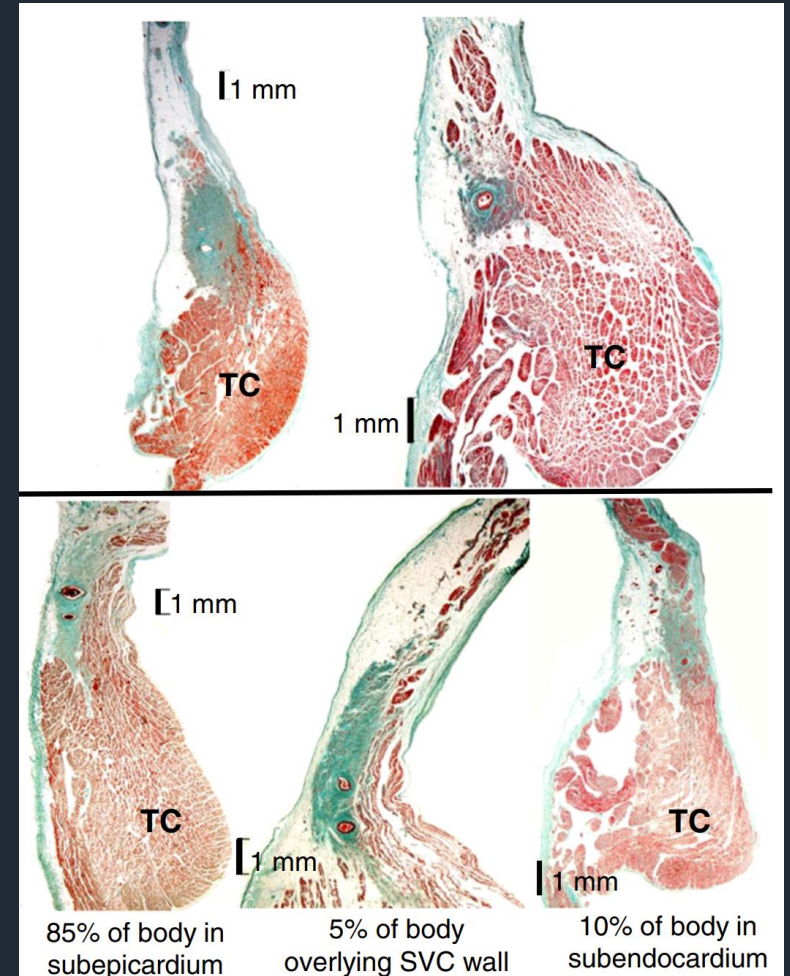
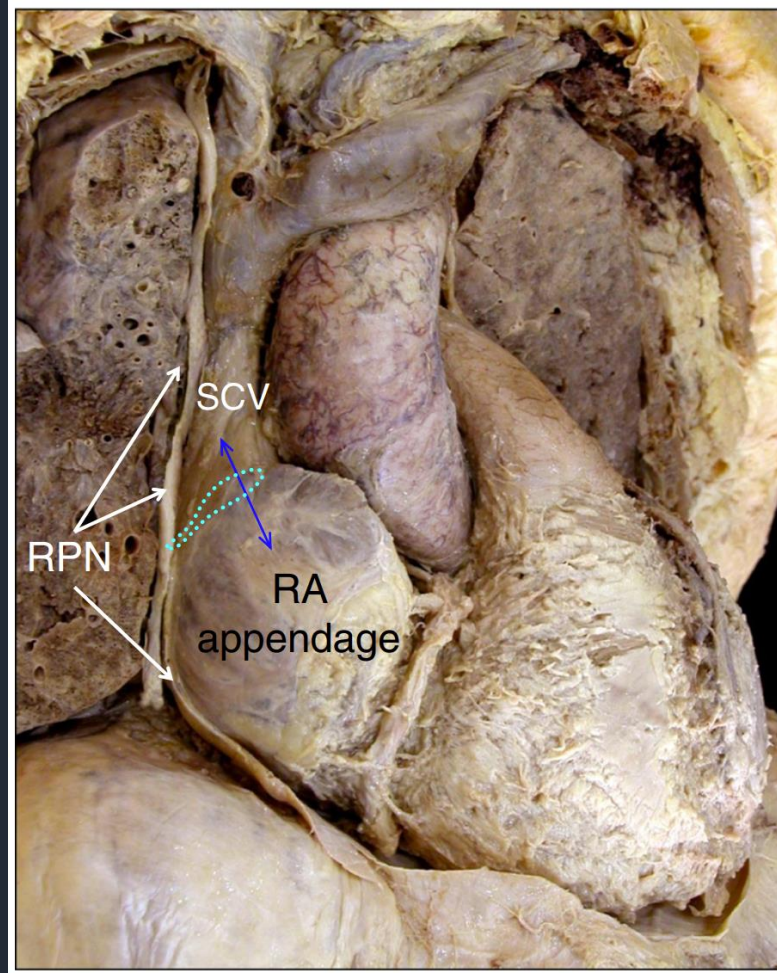
- Research grant from SkyLabs

Contents

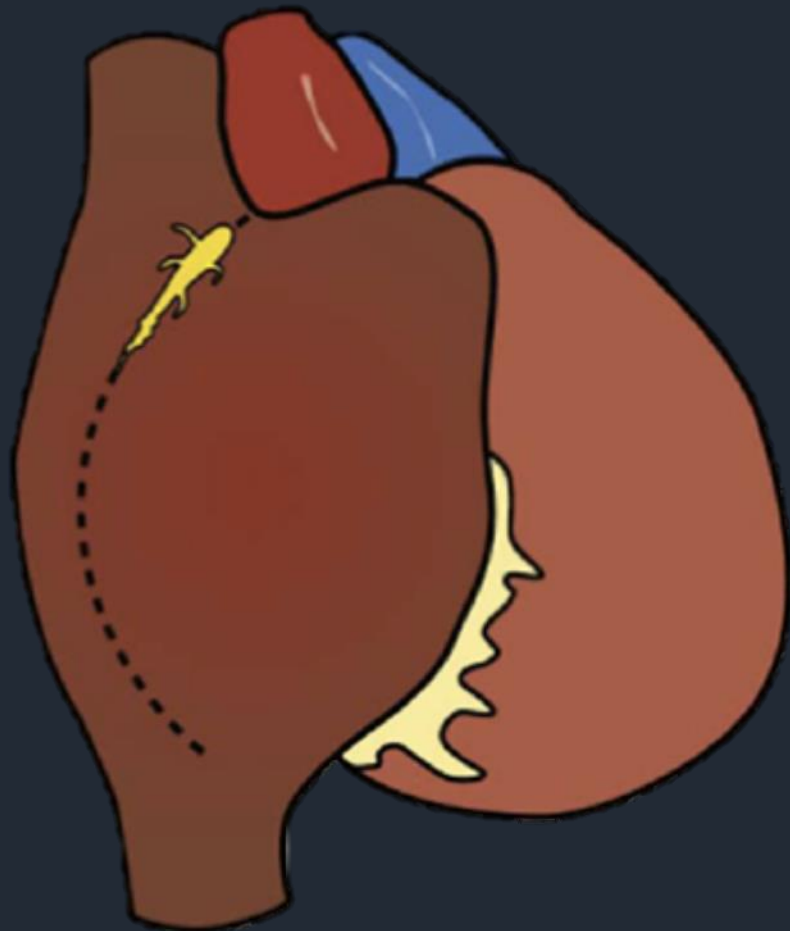
- Sinus node
- Sinus node dysfunction

Sinus node, location

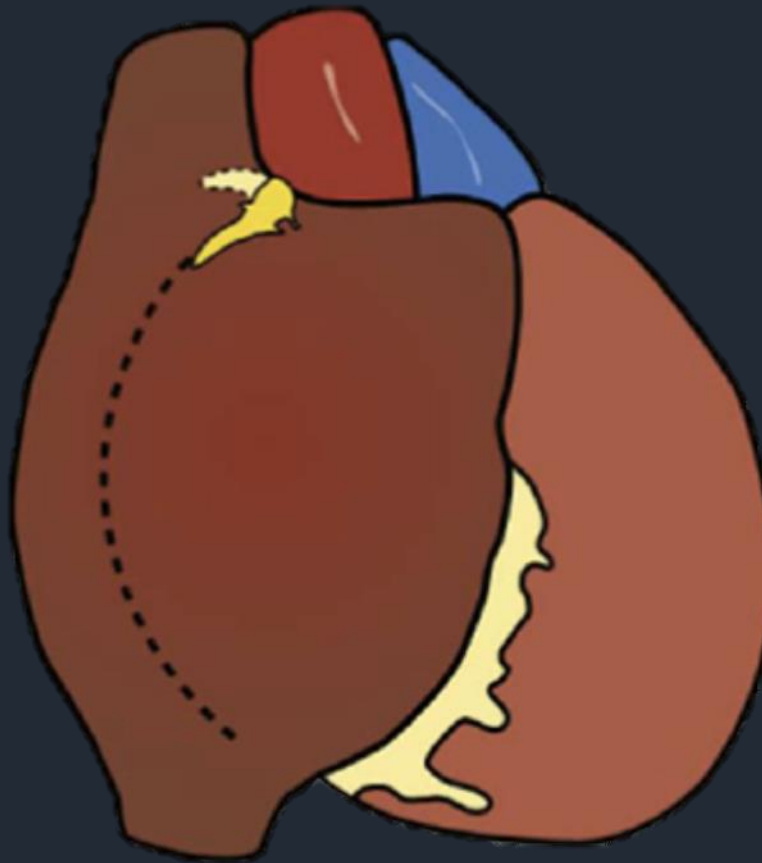
- Localized to the crest of the RAA, at the lateral SVC/RA junction.
- Elaborate structure, extending from the SVC/RA junction down the inferolateral aspect of the crista terminalis toward the IVC/RA orifice



Sinus node, location, variant



Typical location

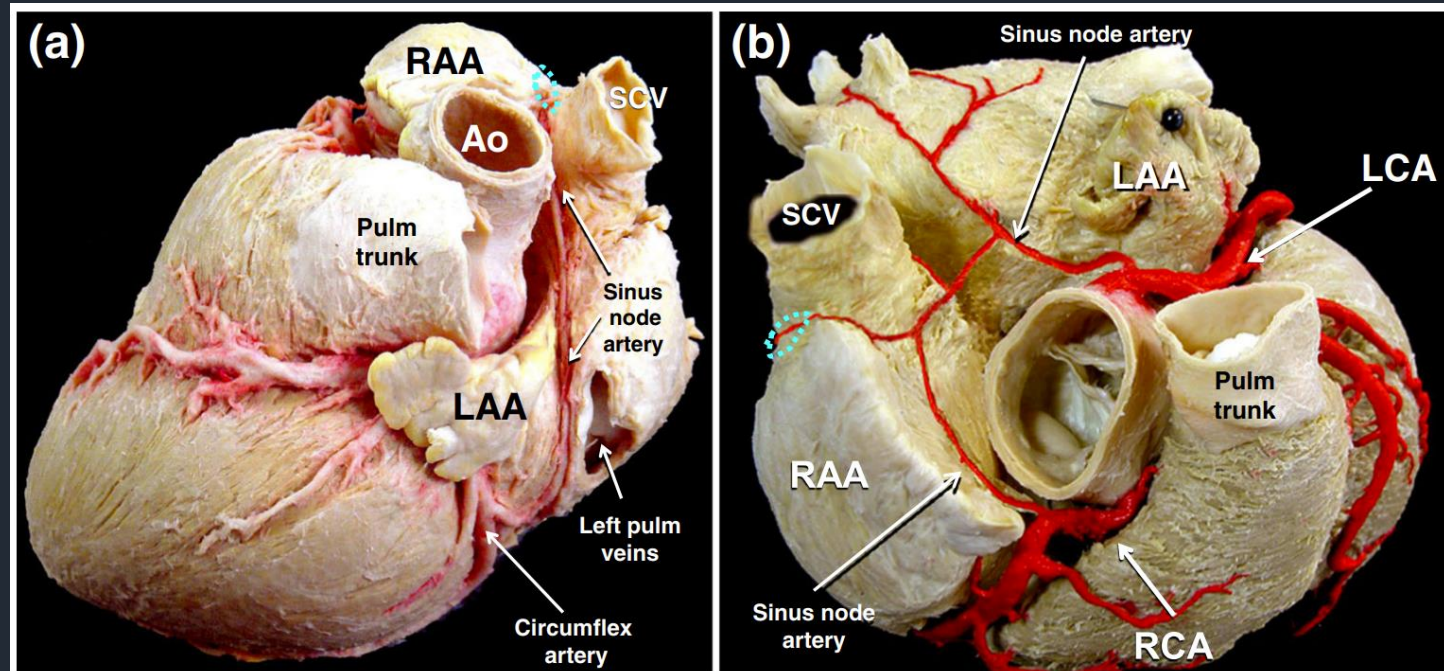
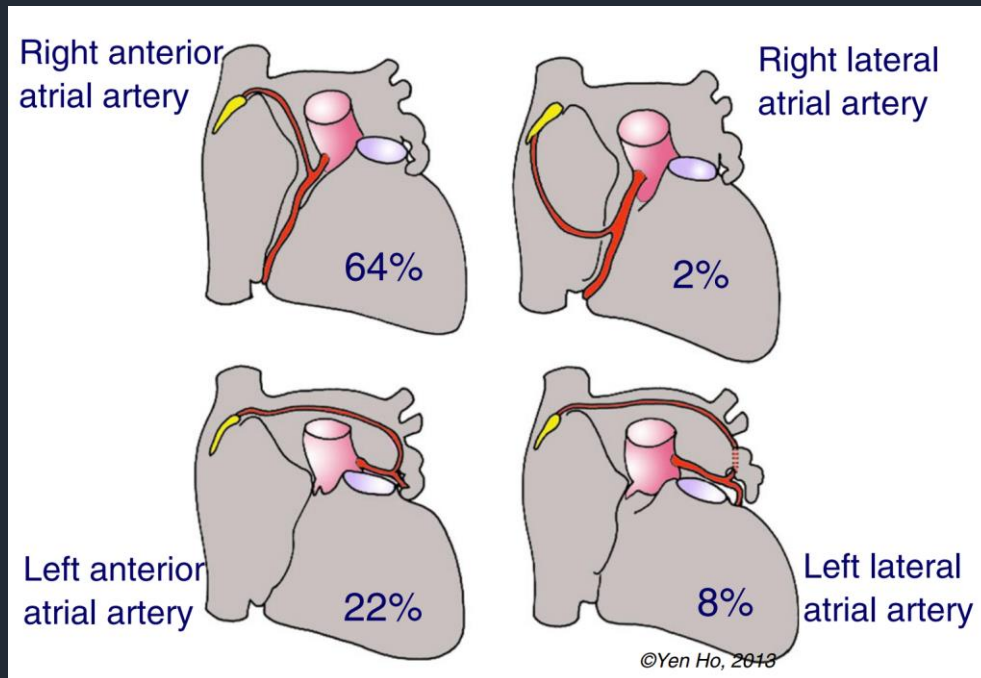


Horseshoe variant

Found in 10 % of human hearts, with the node situated anteriorly and extending across the crest of the right atrial appendage in a horseshoe shaped arrangement, within the inter-atrial groove

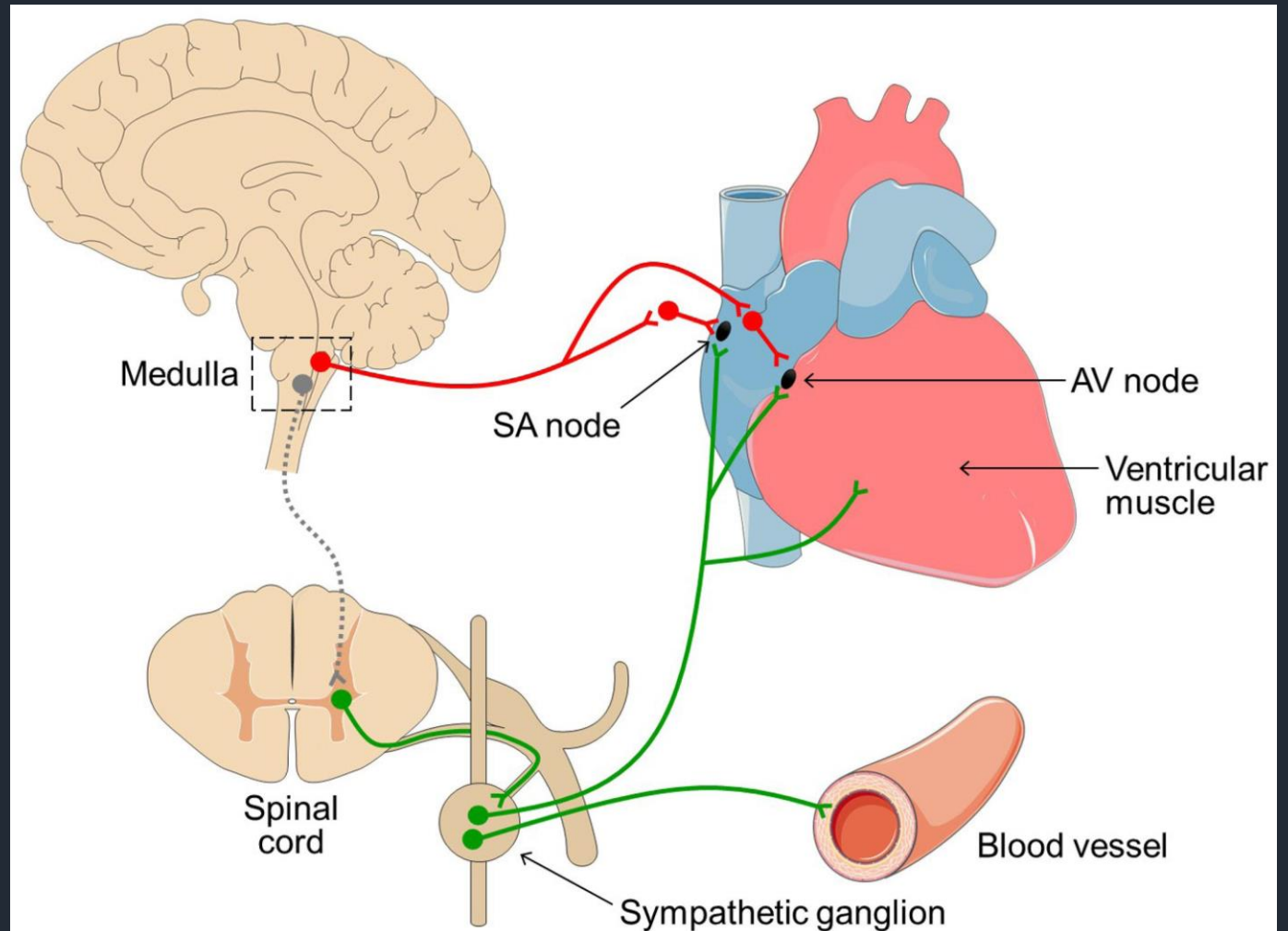
Sinus node, blood supply

- Receives its blood supply predominantly from the sinus node artery, a branch of either the right coronary artery (in about 55-60% of people) or the left circumflex artery (in about 40-45% of people).



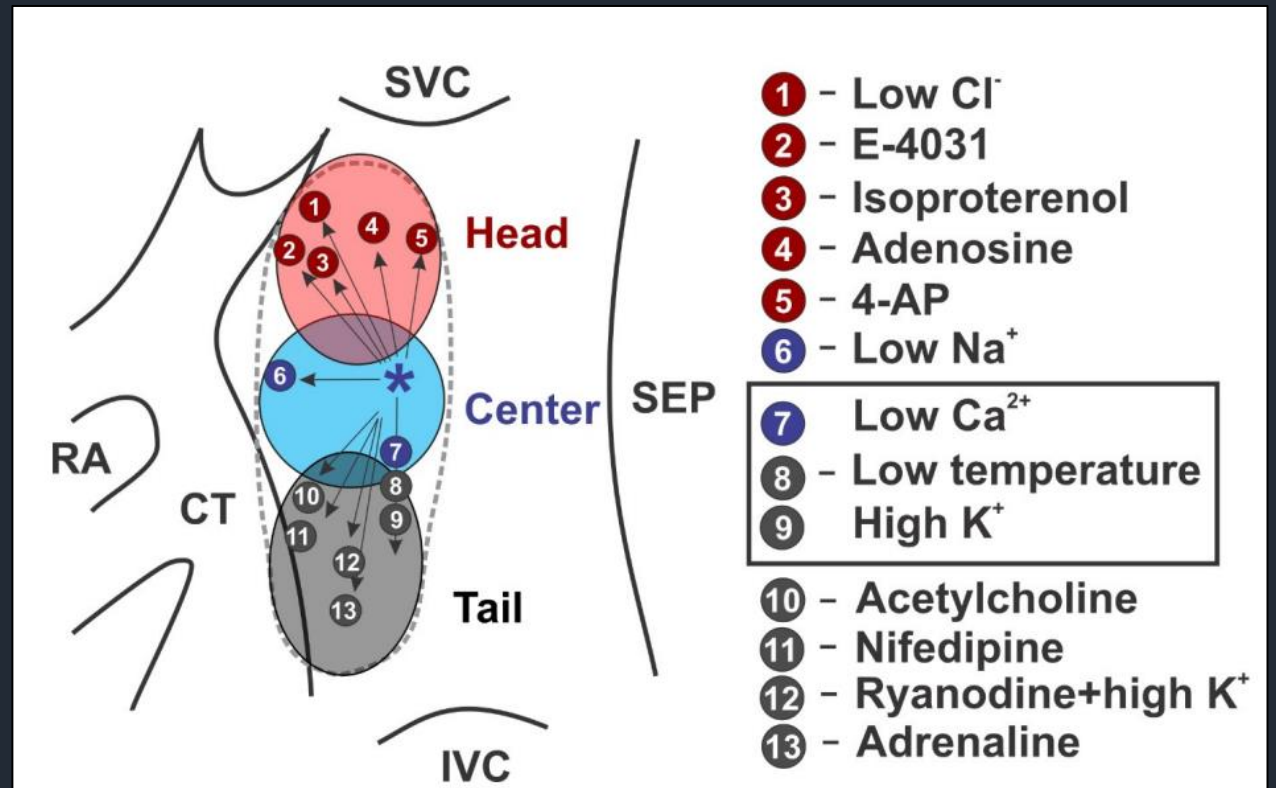
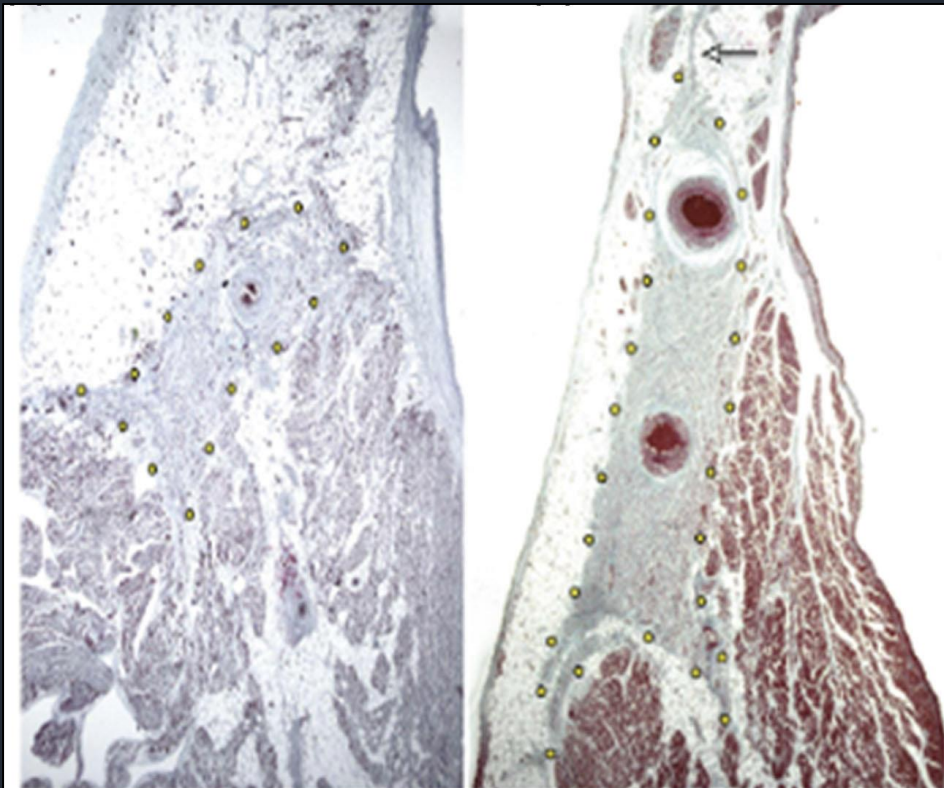
Sinus node, autonomic innervation

Richly innervated by the autonomic nervous system, which plays a crucial role in the regulation of sinus rate, by modulating ionic currents



Sinus node, histology, cell clusters

- Seen as a dense aggregation of specialized cells within a highly fibrous connective tissue matrix

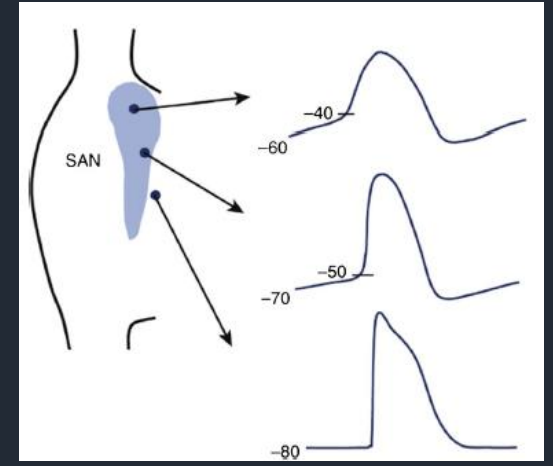


Sinus node, histology, cell clusters

- Mosaic features may help to gradually match nodal activity with that of the right atrium and promote a gradient favoring an antegrade direction of conduction.
- Functional purpose of the transitional zone may be to facilitate the exit of the action potential from the node into the atrial muscle.

Sinus impulse generation

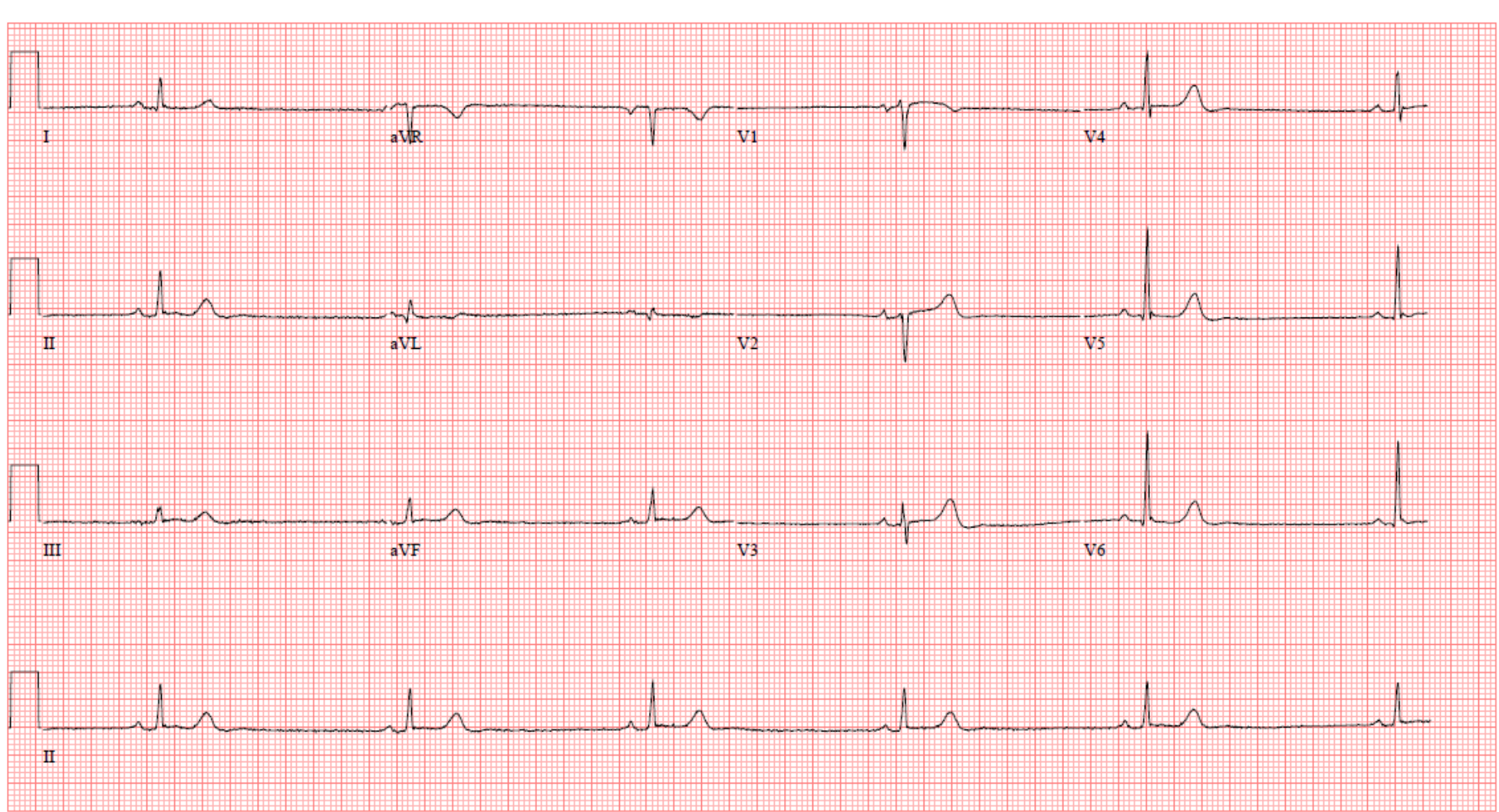
- **Leading pacemaker**
 - typically originates superiorly
 - In response to physiological stimuli, it can shift position in the node to become more inferior or even multifocal (pacemaker shift)
- **Sympathetic** stimulation shifts the leading pacemaker site superiorly resulting in an increase in heart rate
- **Parasympathetic** activity results in an inferior shift and heart rate slowing.



Sinus node dysfunction (SND)

- = Sick sinus syndrome
- Abnormal conduction and propagation of electrical impulses at the sinoatrial node
- Intrinsic / Extrinsic cause
- Sinus bradycardia, sinus pauses or arrest, sinoatrial exit block, chronotropic incompetence, or tachycardia-bradycardia syndrome

EKG 1



EKG 2

Sinus node dysfunction



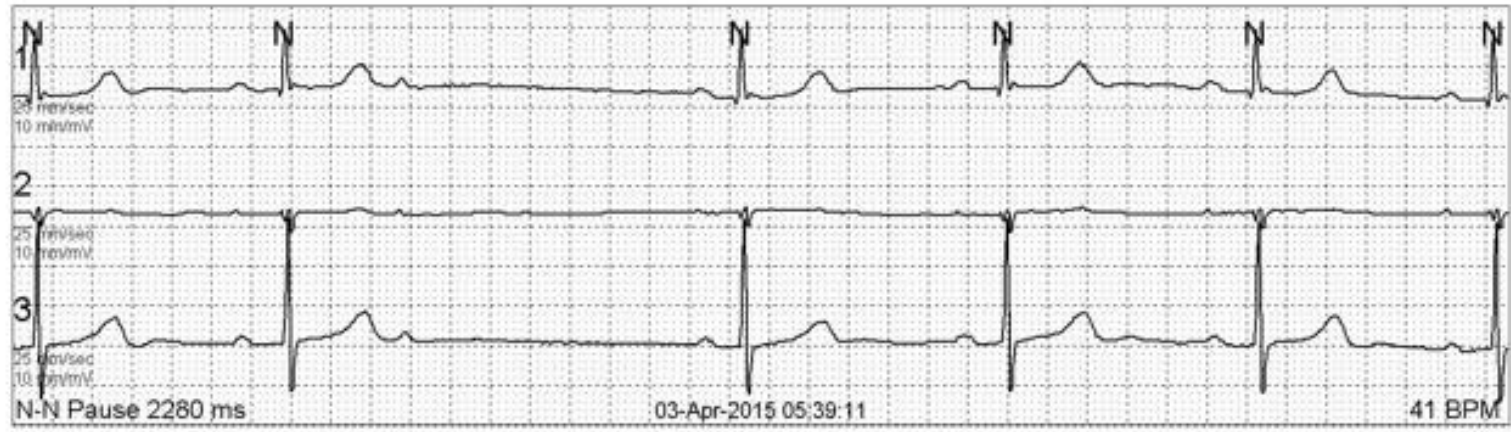
Paroxysmal complete atrioventricular block



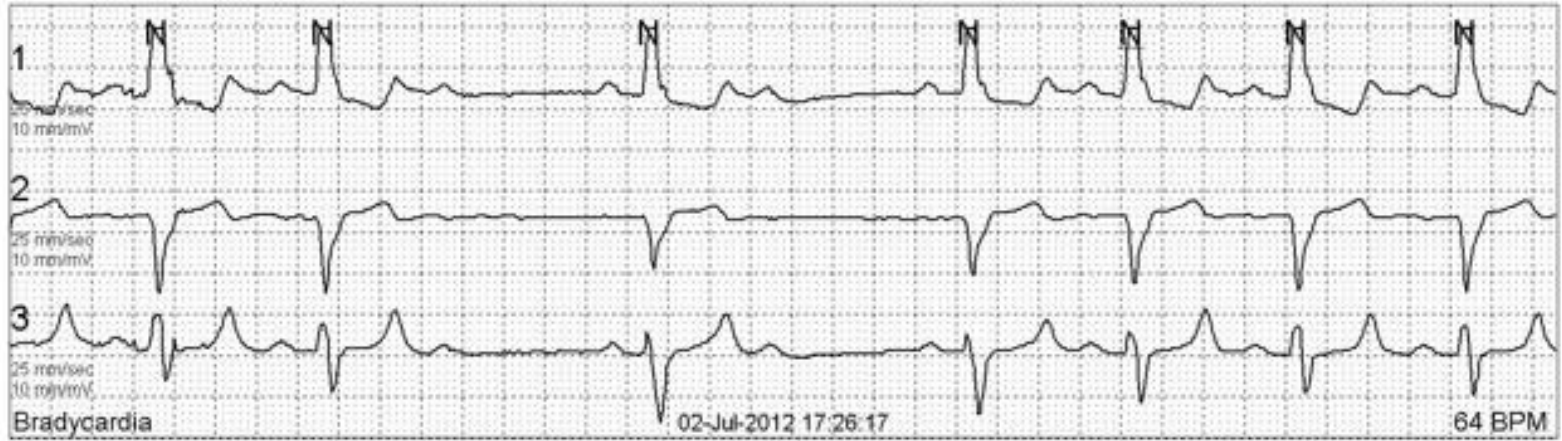
EKG 3



EKG 4

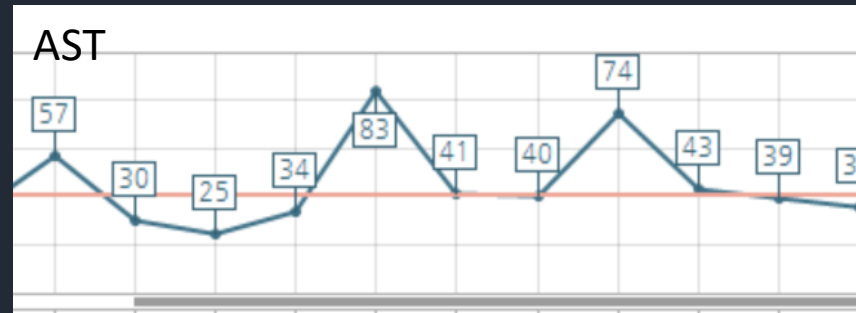
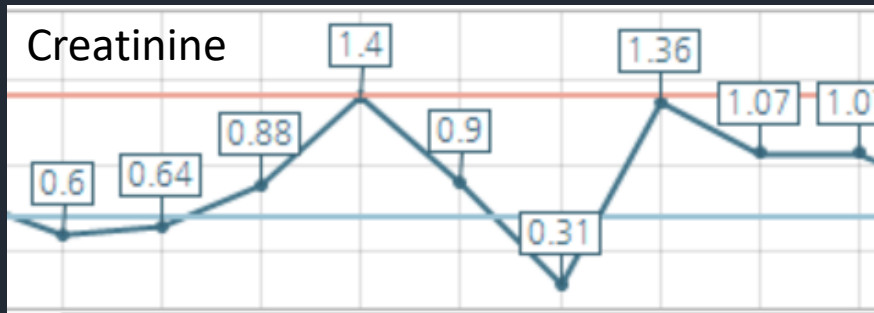


EKG 5

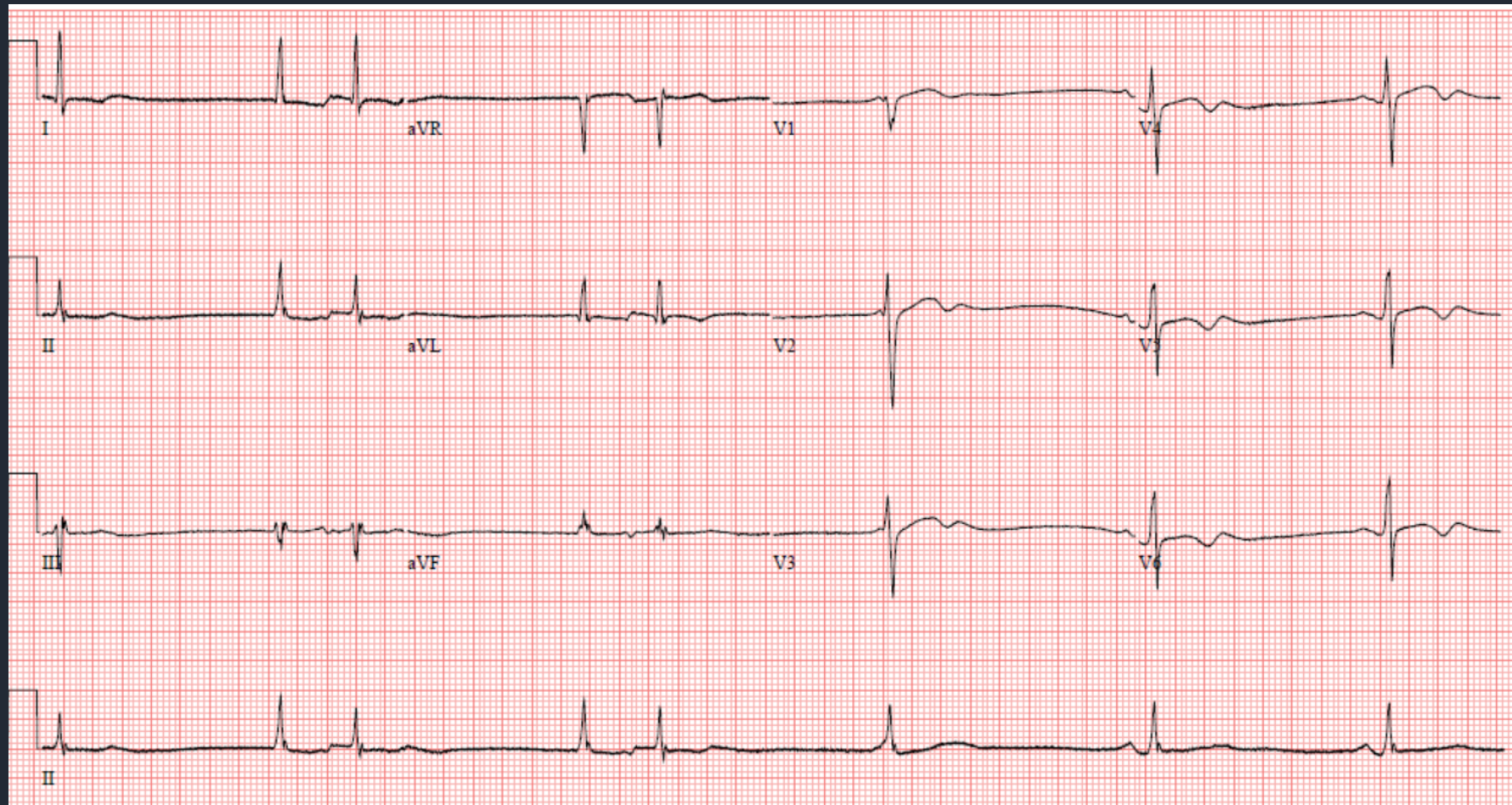


CASE 1

- Female / 48 YO
- Bipolar disorder on Lithium (600mg → 400mg)
- Unstable kidney and liver function

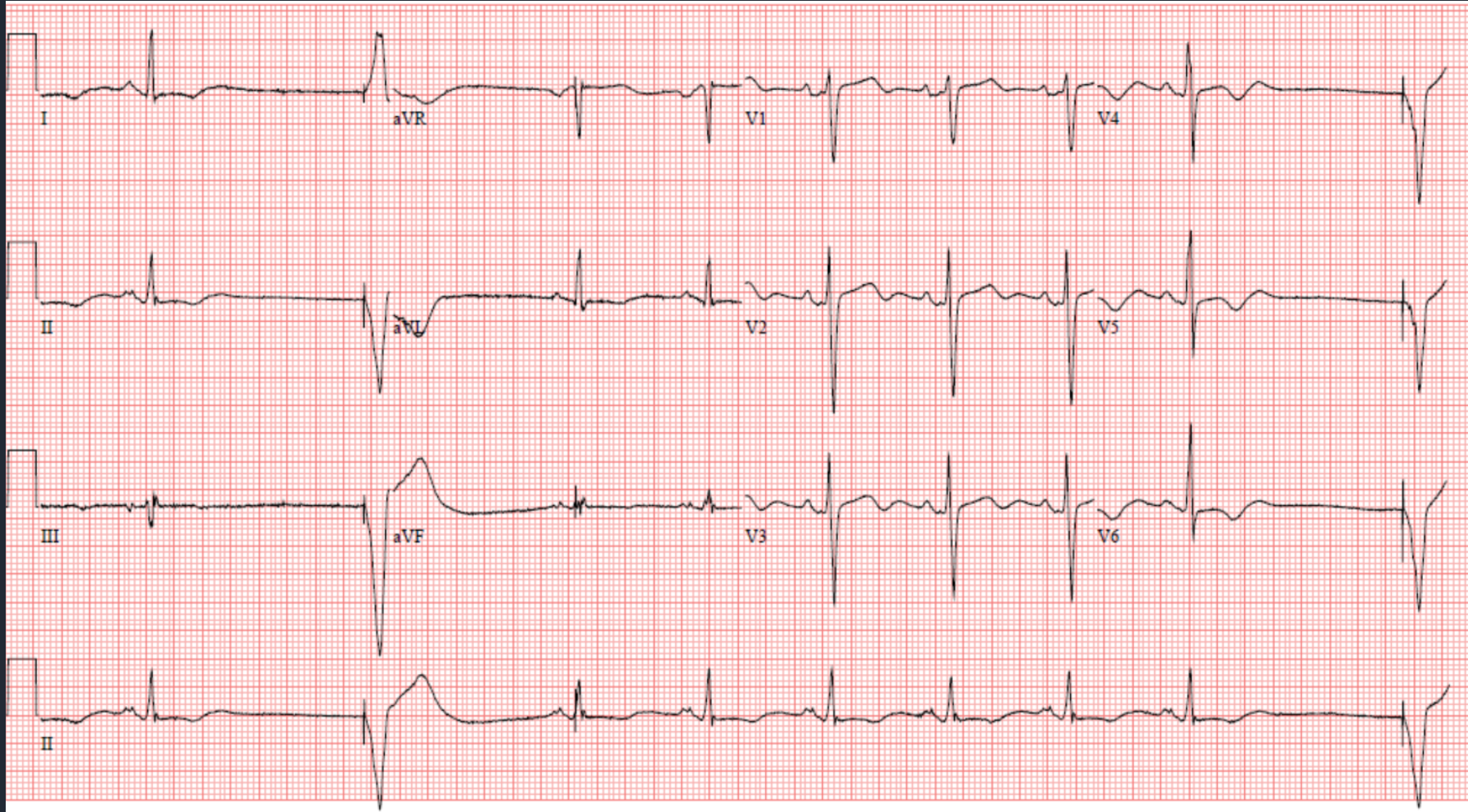


CASE 1



Lithium discontinued and medication change

CASE 1

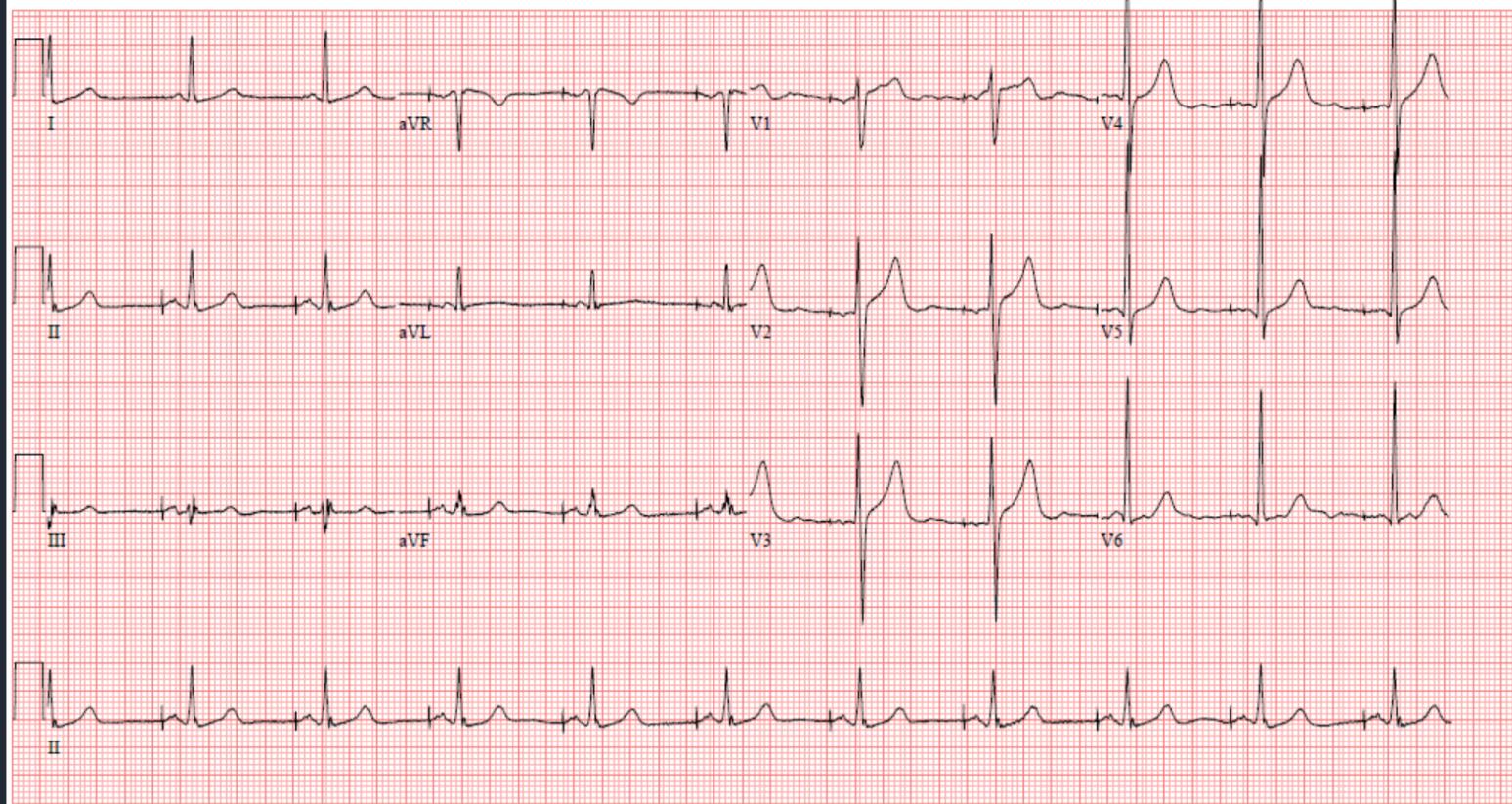


Aggravated psychotic symptom
Intermittent sinus pause with pacing back-up with temporary pacemaker

CASE 1

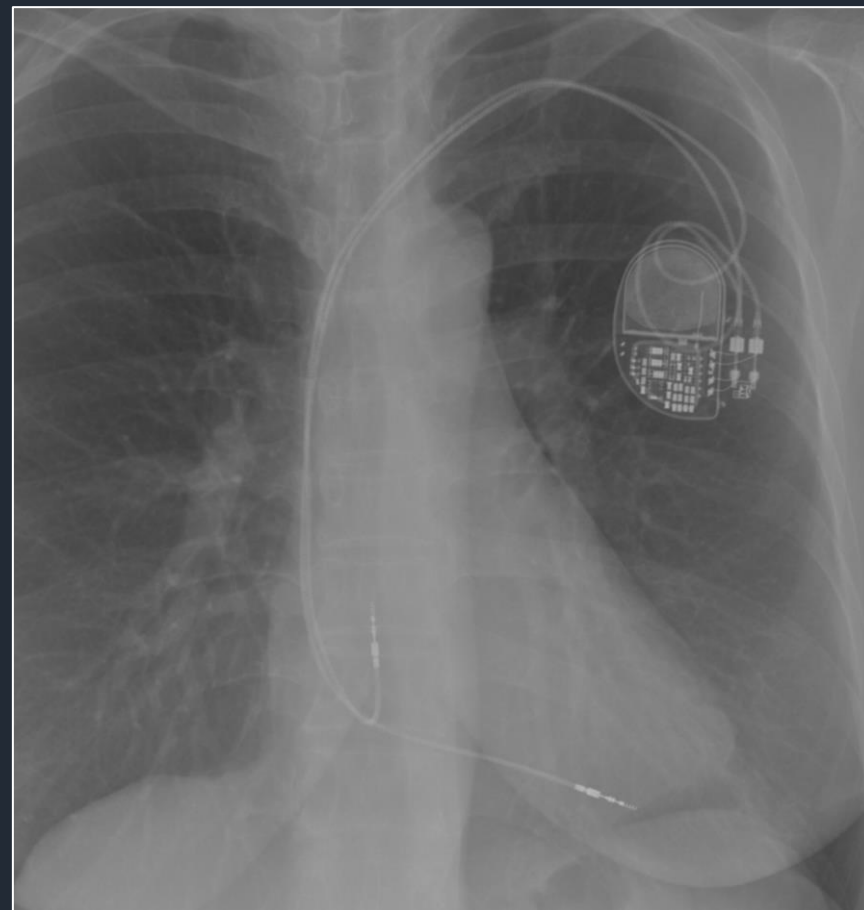
Referred by: CV, CHA MYUNG JIN

Unconfirmed



25mm/s 10mm/mV 150Hz 9.0.9 12SL 243 CID: 0

EID:13 EDT: ORDER:



Common Potentially Reversible or Treatable Causes of SND

- In symptomatic patients presenting with sinus node dysfunction (SND), evaluation and treatment of reversible causes is recommended.

Acute myocardial ischemia or infarction ^{S4.1.1-2-S4.1.1-4}
Athletic training ^{S4.1.1-5}
Atrial fibrillation ^{S4.1.1-6}
Cardiac surgery
Valve replacement, ^{S4.1.1-7,S4.1.1-8} maze procedure, ^{S4.1.1-7} coronary artery bypass graft ^{S4.1.1-9,S4.1.1-10}
Drugs or toxins*
Toluene, organophosphates, tetrodotoxin, cocaine ^{S4.1.1-11}
Electrolyte abnormality
Hyperkalemia, ^{S4.1.1-12} hypokalemia, ^{S4.1.1-13} hypoglycemia ^{S4.1.1-14}
Heart transplant: ^{S4.1.1-15} Acute rejection, chronic rejection, remodeling ^{S4.1.1-16,S4.1.1-17}
Hypervagotonia ^{S4.1.1-18,S4.1.1-19}

Hypothermia
Therapeutic (post-cardiac arrest cooling ^{S4.1.1-20}) or environmental exposure ^{S4.1.1-21}
Hypothyroidism ^{S4.1.1-22}
Hypovolemic shock ^{S4.1.1-23}
Hypoxemia, hypercarbia, acidosis ^{S4.1.1-24}
Sleep apnea, respiratory insufficiency (suffocation, drowning, ^{S4.1.1-25} stroke, ^{S4.1.1-26} drug overdose)
Infection ^{S4.1.1-27}
Lyme disease, ^{S4.1.1-28} legionella, psittacosis, typhoid fever, typhus, listeria, ^{S4.1.1-29} malaria, leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre ^{S4.1.1-30}
Medications*
Beta blockers, non-dihydropyridine calcium channel blockers, digoxin, ^{S4.1.1-31} antiarrhythmic drugs, lithium, ^{S4.1.1-32} methyldopa, risperidone, cisplatin, interferon

Can Induce/Exacerbate Bradycardia

Antihypertensive	Antiarrhythmic	Psychoactive	Other
Beta adrenergic receptor blockers (including beta adrenergic blocking eye drops used for glaucoma)	Adenosine	Donepezil	Anesthetic drugs (propofol)
Clonidine	Amiodarone	Lithium	Cannabis
Methyldopa	Dronedarone	Opioid analgesics	Digoxin
Non-dihydropyridine calcium channel blockers	Flecainide	Phenothiazine antiemetics and antipsychotics	Ivabradine
Reserpine	Procainamide	Phenytoin	Muscle relaxants (eg, succinylcholine)
	Propafenone	Selective serotonin reuptake inhibitors	
	Quinidine	Tricyclic antidepressants	
	Sotalol		

Acute Medical Therapy for Bradycardia

- In patients with SND associated with symptoms or hemodynamic compromise, atropine is reasonable to increase sinus rate (IIA)
- In patients with SND associated with symptoms or hemodynamic compromise who are at low likelihood of coronary ischemia, isoproterenol, dopamine, dobutamine, or epinephrine may be considered to increase heart rate and improve symptoms. (IIB)
- In patients who have undergone heart transplant without evidence for autonomic reinnervation, atropine should not be used to treat sinus bradycardia. (III)

Acute Medical Therapy for Bradycardia

Atropine 0.5–1 mg IV (may be repeated every 3–5 min to a maximum dose of 3 mg)

Dopamine 5 to 20 mcg/kg/min IV, starting at 5 mcg/kg/min and increasing by 5 mcg/kg/min every 2 min
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
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
2–10 mcg/min IV or 0.1–0.5 mcg/kg/min IV titrated to desired effect

General evaluation

• Treadmill test

- In patients with suspected chronotropic incompetence, exercise electrocardiographic testing is reasonable to ascertain the diagnosis and provide information on prognosis. (IIa)
- In patients with exercise-related symptoms suspicious for bradycardia or conduction disorders, or in patients with 2:1 atrioventricular block of unknown level, exercise electrocardiographic testing is reasonable. (IIa)

• Holter monitoring

- Cardiac rhythm monitoring is useful to establish correlation between heart rate or conduction abnormalities with symptoms, with the specific type of cardiac monitor chosen based on the frequency and nature of symptoms, as well as patient preferences (I)

Sleep Apnea Evaluation

- In patients with documented or suspected bradycardia or conduction disorder during sleep, screening for symptoms of sleep apnea syndrome is recommended with subsequent confirmatory testing directed by clinical suspicion.

Case 2

- Female / 66 YO
- Hypertension on telmisartan 40mg qd
- Occasional symptomatic PVCs with dilatrend 12.5mg bid

- CC> Palpitation and Dizziness

Case 2

- Holter monitoring

General

56289 QRS complexes
0 Paced beats (< 1%)
1261 Ventricular beats (2%)
475 Supraventricular beats (< 1%)
0 BB beats (< 1%)
0 Junctional beats (< 1%)
0 Aberrant beats
0 % of total time in AF/AFL
< 1 % of total time classified as noise

Ventriculars (V, F, E, I)

1241 Isolated
10 Couplets
0 Bigeminal cycles
0 Runs totaling 0 beats

Heart Rates

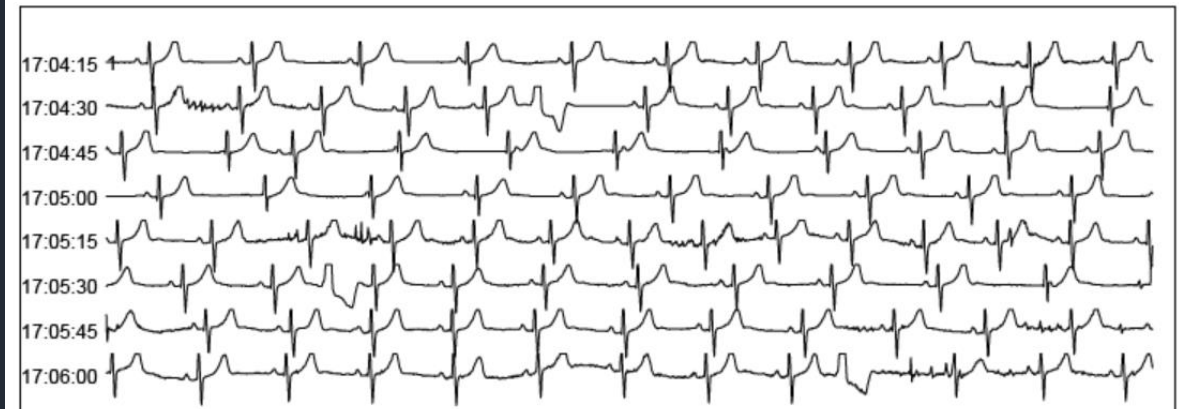
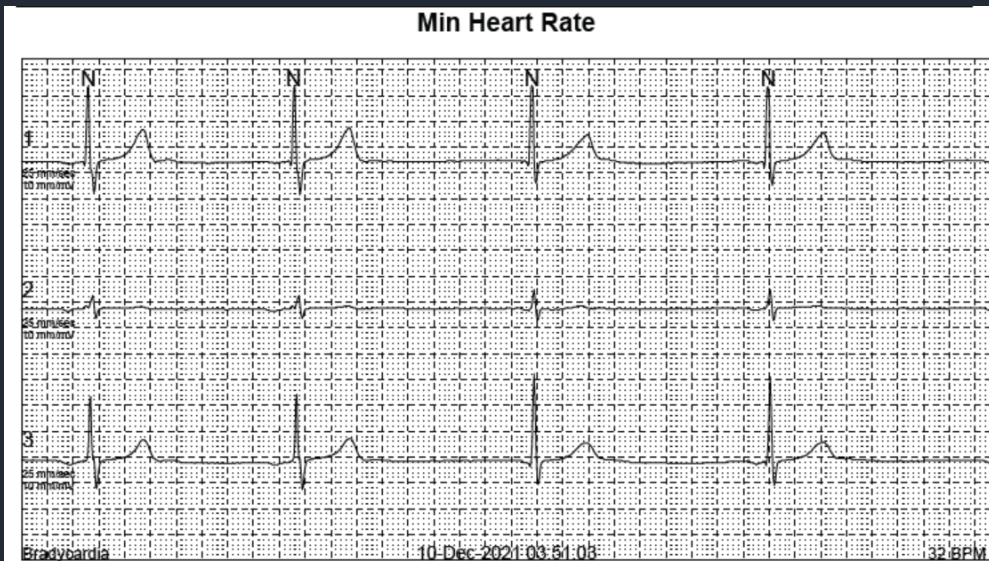
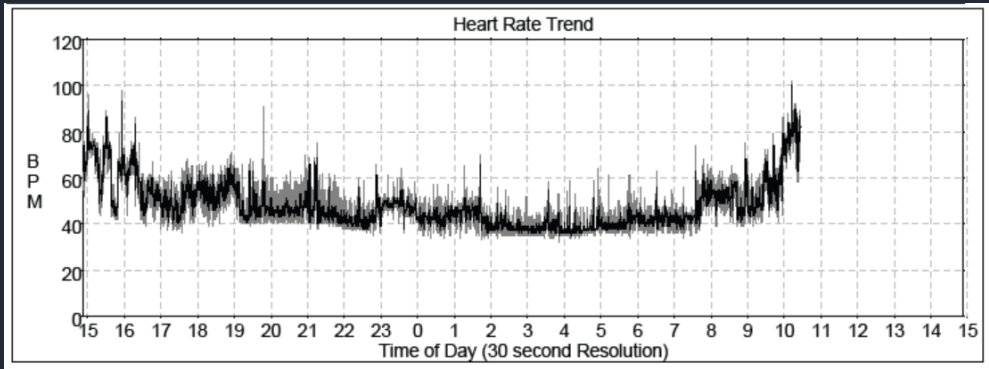
32 Minimum at 03:51:03 10-Dec
48 Average
102 Maximum at 10:12:45 10-Dec
36 Beats in tachycardia (≥ 100 bpm), < 1% total
47336 Beats in bradycardia (≤ 60 bpm), 84% total
1.97 Seconds Max R-R at 06:31:52 10-Dec

Supraventriculars (S, J, A)

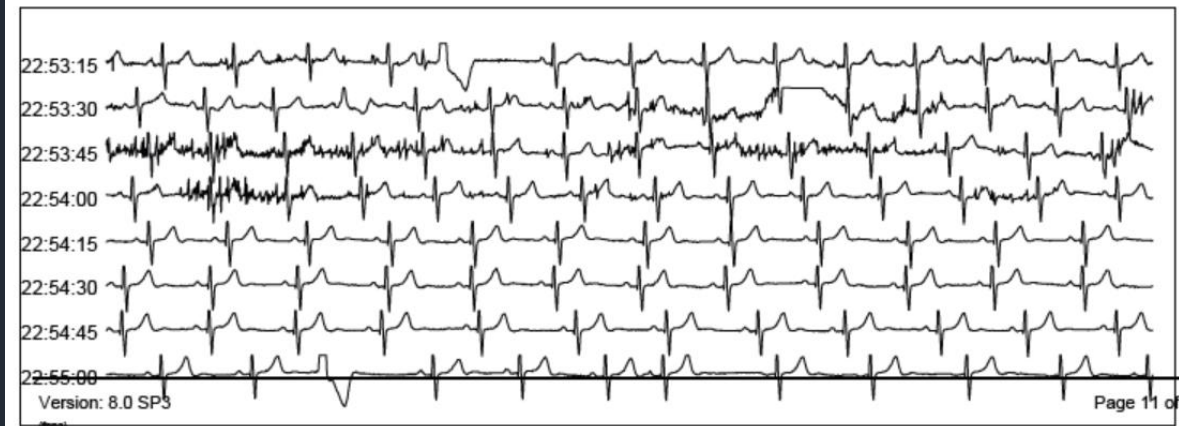
426 Isolated
11 Couplets
22 Bigeminal cycles
6 Runs totaling 27 beats
7 Beats longest run 91 bpm 19:47:32 09-Dec
3 Beats fastest run 104 bpm 18:20:11 09-Dec

Case 2

- Holter monitoring



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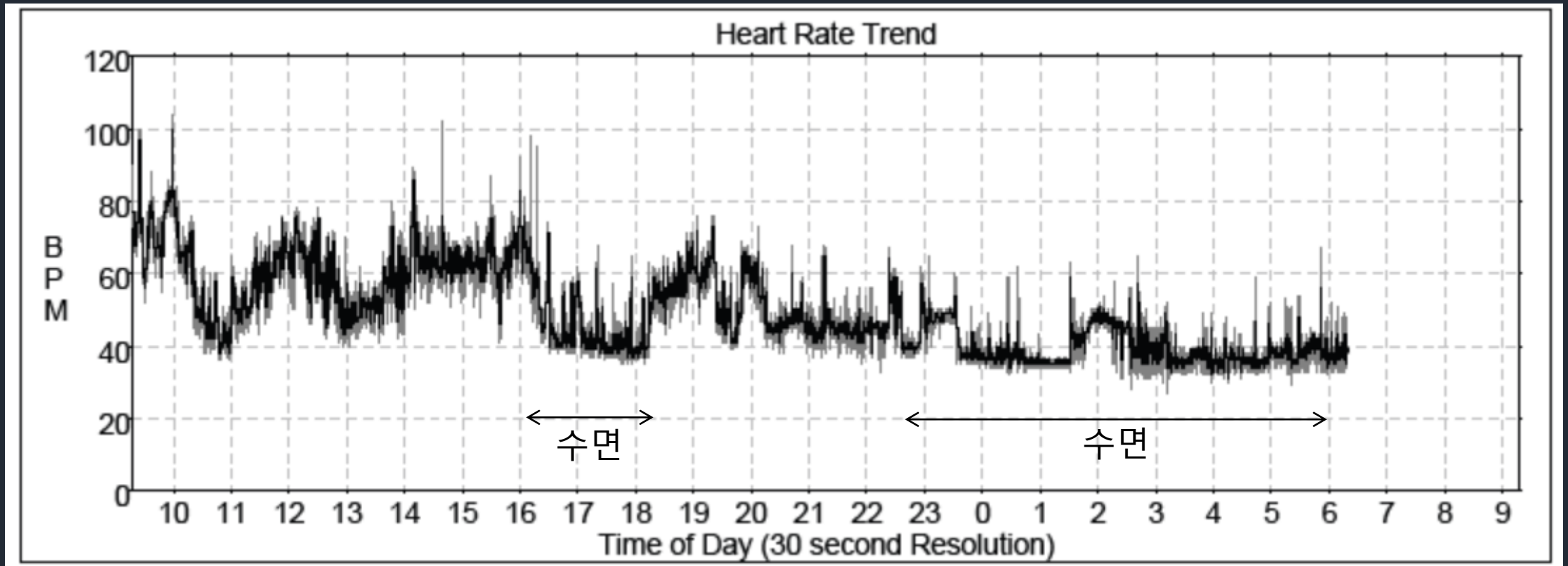
Case 2

- Treadmill test

<u>Phase</u>	<u>Stage</u>	<u>Time in Stage</u>	<u>Speed mph</u>	<u>Grade %</u>	<u>METS</u>	<u>HR bpm</u>	<u>BP mmHg</u>	<u>RPP</u>	<u>PVC VE/min</u>
PRETEST	SITTING	1:49	0.0	0.0	1.0	55	161/98	88	0
	WARM-UP	0:08	0.8	0.0	1.0	59			0
EXERCISE	STAGE 1	3:00	1.7	10.0	4.6	100	179/91	179	0
	STAGE 2	3:00	2.5	12.0	7.0	120	194/95	232	0
	STAGE 3	2:03	3.4	14.0	10.1	136			1
RECOVERY		1:00	0.0	0.0	5.7	98			1
		1:00	0.0	0.0	3.4	80	200/102	160	0
		1:00	0.0	0.0	2.3	74	172/97	127	0
		1:00	0.0	0.0	1.7	71	168/98	119	0
		1:00	0.0	0.0	1.4	64	156/97	99	0
		1:00	0.0	0.0	1.3	65	165/104	107	0
		0:10	0.0	0.0	1.3	64			0

Case 2

- Holter monitoring follow-up



=> Observation

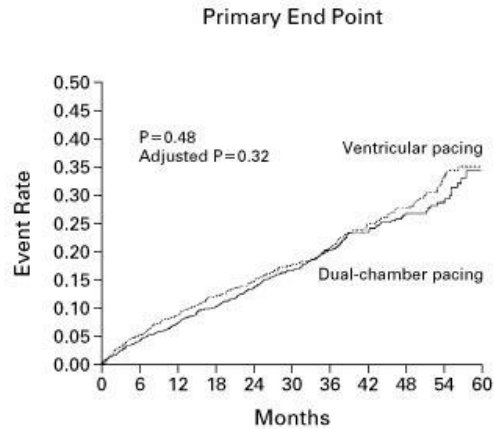
Pacing should not be performed

III: Harm	In asymptomatic individuals with sinus bradycardia or sinus pauses that are secondary to physiologically elevated parasympathetic tone , permanent pacing should not be performed.	C
III: Harm	In patients with sleep-related sinus bradycardia or transient sinus pauses occurring during sleep , permanent pacing should not be performed unless other indications for pacing are present.	C
III: Harm	In patients with asymptomatic SND , or in those in whom the symptoms have been documented to occur in the absence of bradycardia or chronotropic incompetence , permanent pacing should not be performed.	C

Pacing should not be performed

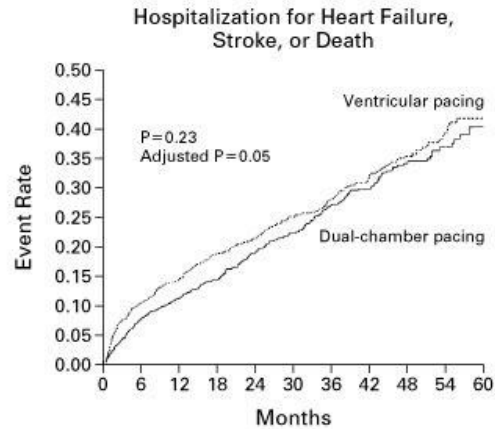
I	In symptomatic patients with SND, atrial based pacing is recommended over single chamber ventricular pacing.	B-R
I	In symptomatic patients with SND and intact atrioventricular conduction without evidence of conduction abnormalities, dual chamber or single chamber atrial pacing is recommended.	B-R

Dual versus Single chamber pacing in SND



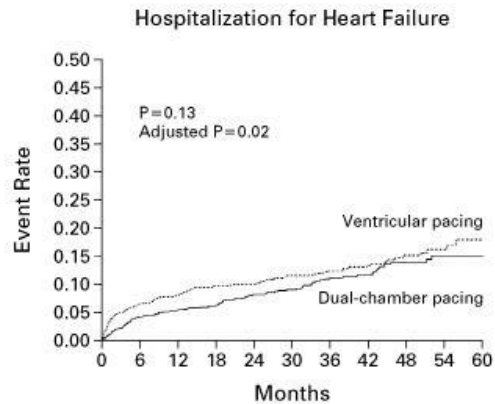
No. AT RISK

Ventricular pacing	996	934	897	813	678	557	431	320	218	125	39
Dual-chamber pacing	1014	963	930	833	693	555	431	328	214	120	28



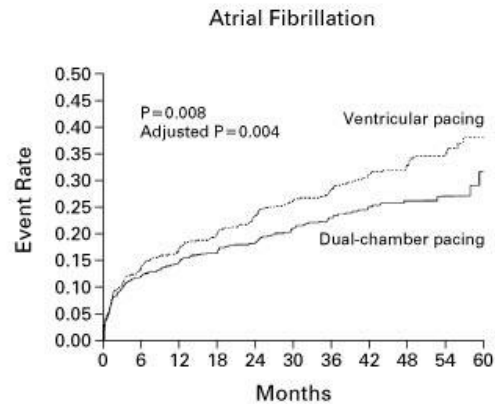
No. AT RISK

Ventricular pacing	996	880	839	752	624	504	388	287	193	110	35
Dual-chamber pacing	1014	926	889	793	649	518	394	297	188	105	26



No. AT RISK

Ventricular pacing	996	890	855	766	637	516	402	300	200	116	36
Dual-chamber pacing	1014	932	894	801	658	528	406	307	191	106	27



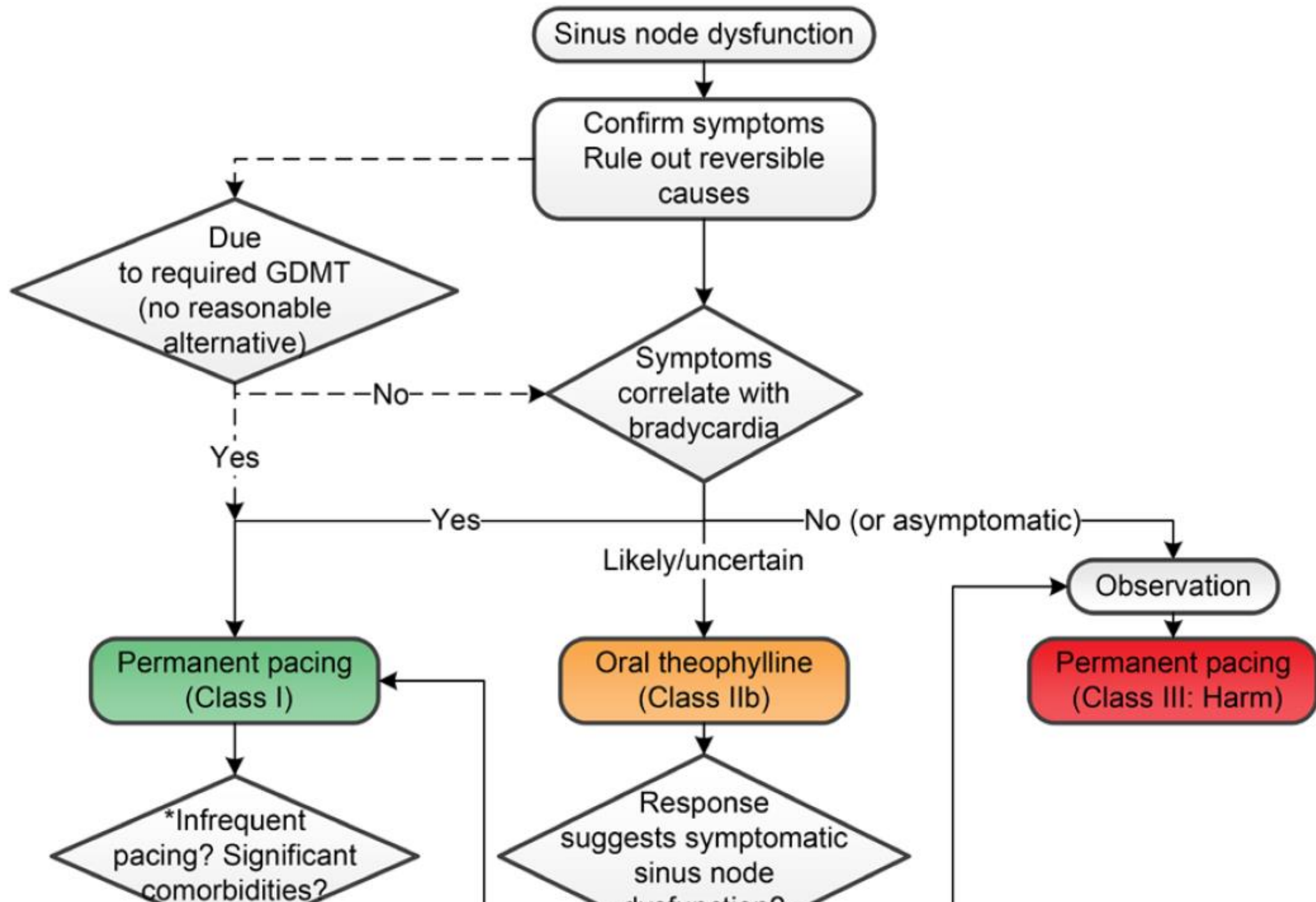
No. AT RISK

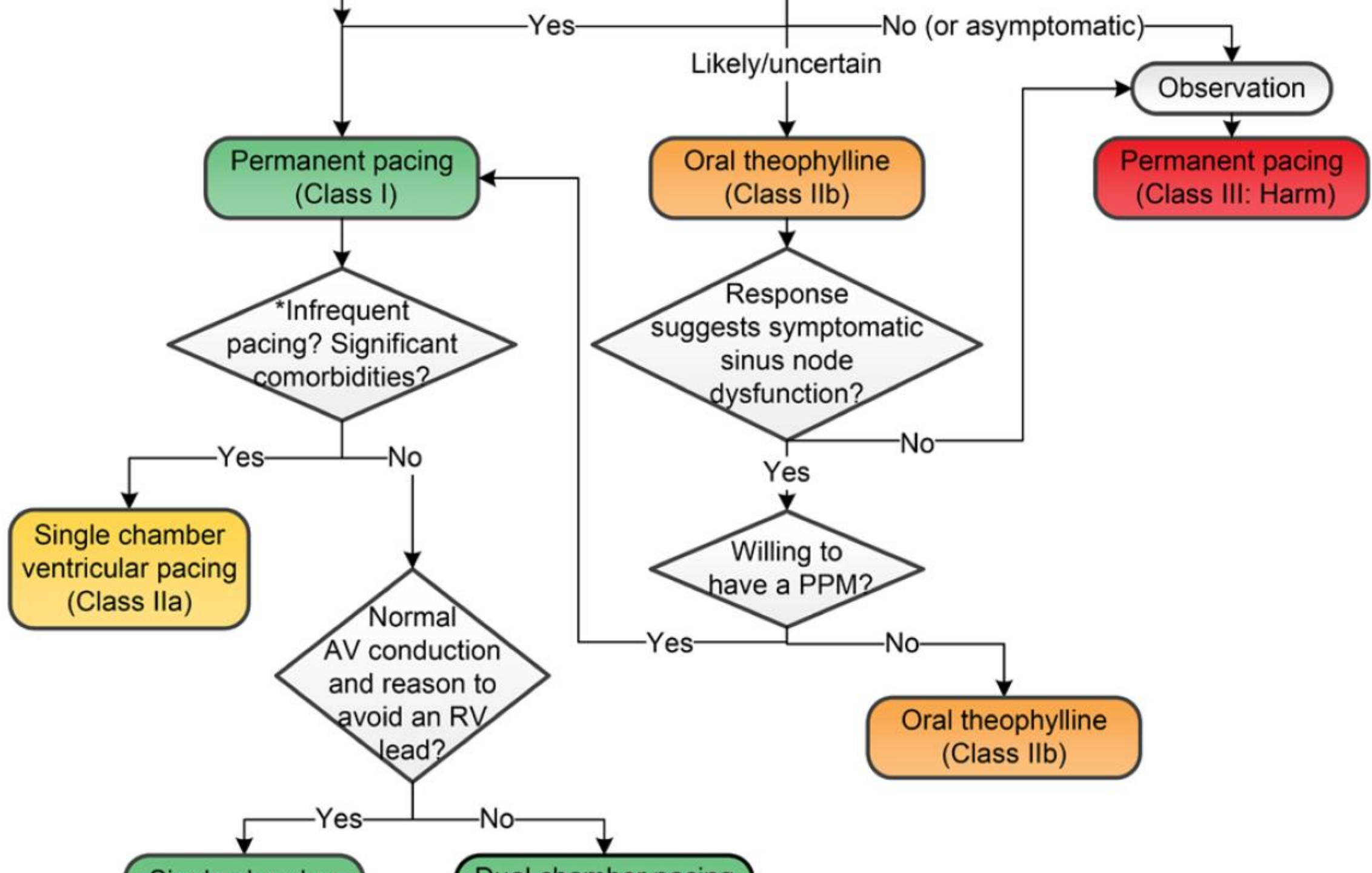
Ventricular pacing	996	815	761	668	542	432	333	242	162	92	27
Dual-chamber pacing	1014	852	795	700	572	444	341	248	148	77	20

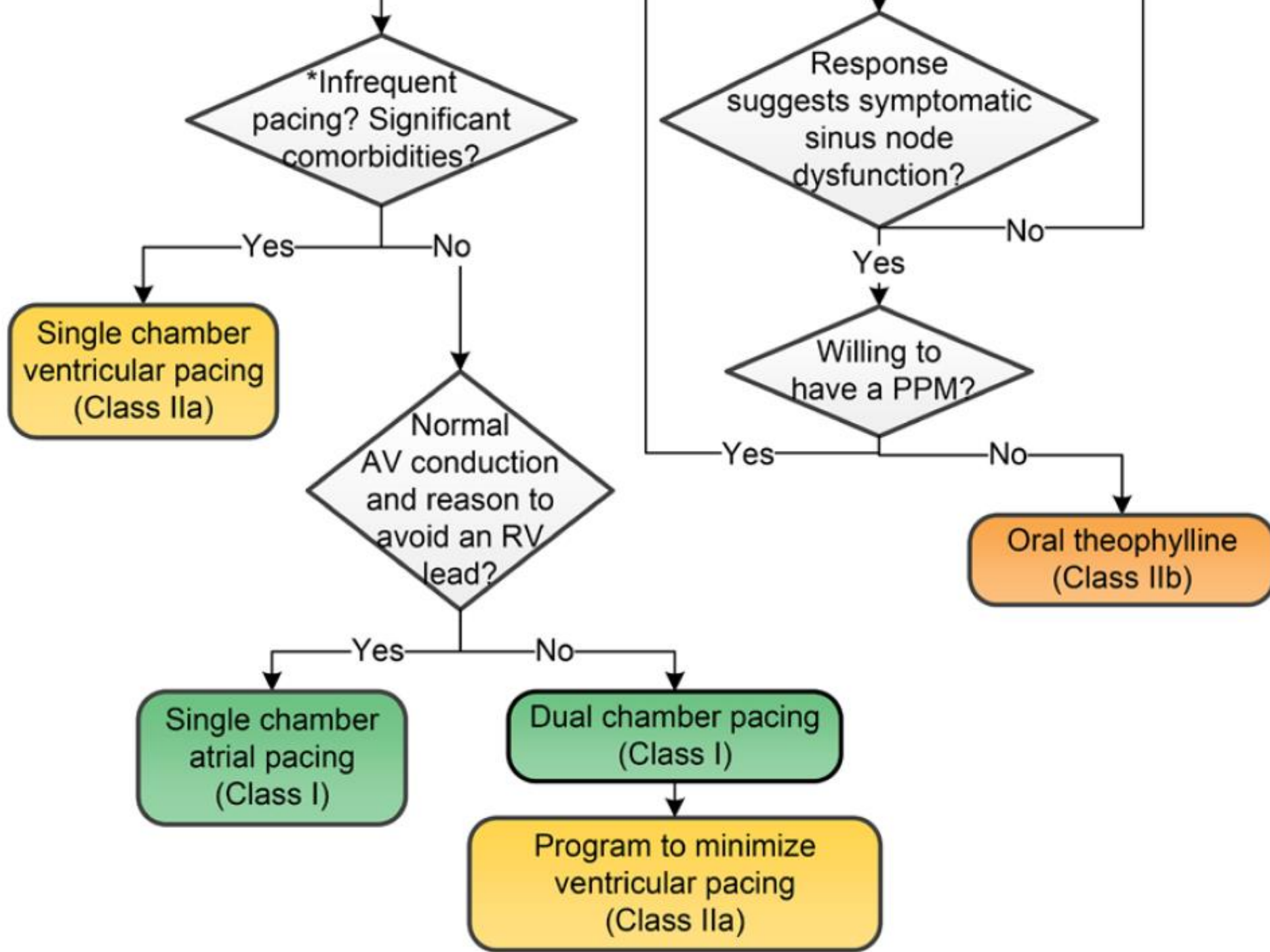
- In sinus-node dysfunction, dual-chamber pacing does not improve stroke-free survival, as compared with ventricular pacing.
- However, dual-chamber pacing reduces the risk of atrial fibrillation, reduces signs and symptoms of heart failure, and slightly improves the quality of life.

Oral theophylline

IIb	In patients with symptoms that are likely attributable to SND, a trial of oral theophylline may be considered to increase heart rate, improve symptoms, and help determine the potential effects of permanent pacing.	C- LD
IIa	In post-heart transplant patients, aminophylline or theophylline is reasonable to increase heart rate if clinically Indicated	C- LD
IIa	In patients with SND associated with symptoms or hemodynamic compromise in the setting of acute spinal cord injury , aminophylline or theophylline is reasonable to increase heart rate and improve Symptoms	C- LD







Take Home Messages

1. Sinus node dysfunction is **most often related to age-dependent** progressive fibrosis of the sinus nodal tissue and surrounding atrial myocardium leading to abnormalities of sinus node and atrial impulse formation and propagation and will therefore result in various bradycardic or pause related syndromes.

Take Home Messages

2. The presence of nocturnal bradycardias should prompt consideration for screening for sleep apnea, beginning with solicitation of suspicious symptoms. However, **nocturnal bradycardia is not in itself an indication for permanent pacing.**

Take Home Messages

3. 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.

Take Home Messages

4. In patients with bradycardia who have indications for pacemaker implantation, shared decision-making and patient-centered care are endorsed and emphasized in this guideline.

Take Home Messages

5. Identifying patient populations that will benefit the most from emerging pacing technologies (eg, His bundle pacing, transcatheter leadless pacing systems) will require further investigation as these modalities are incorporated into clinical practice.