

# Conduction Disease and Implantable Cardiac Devices

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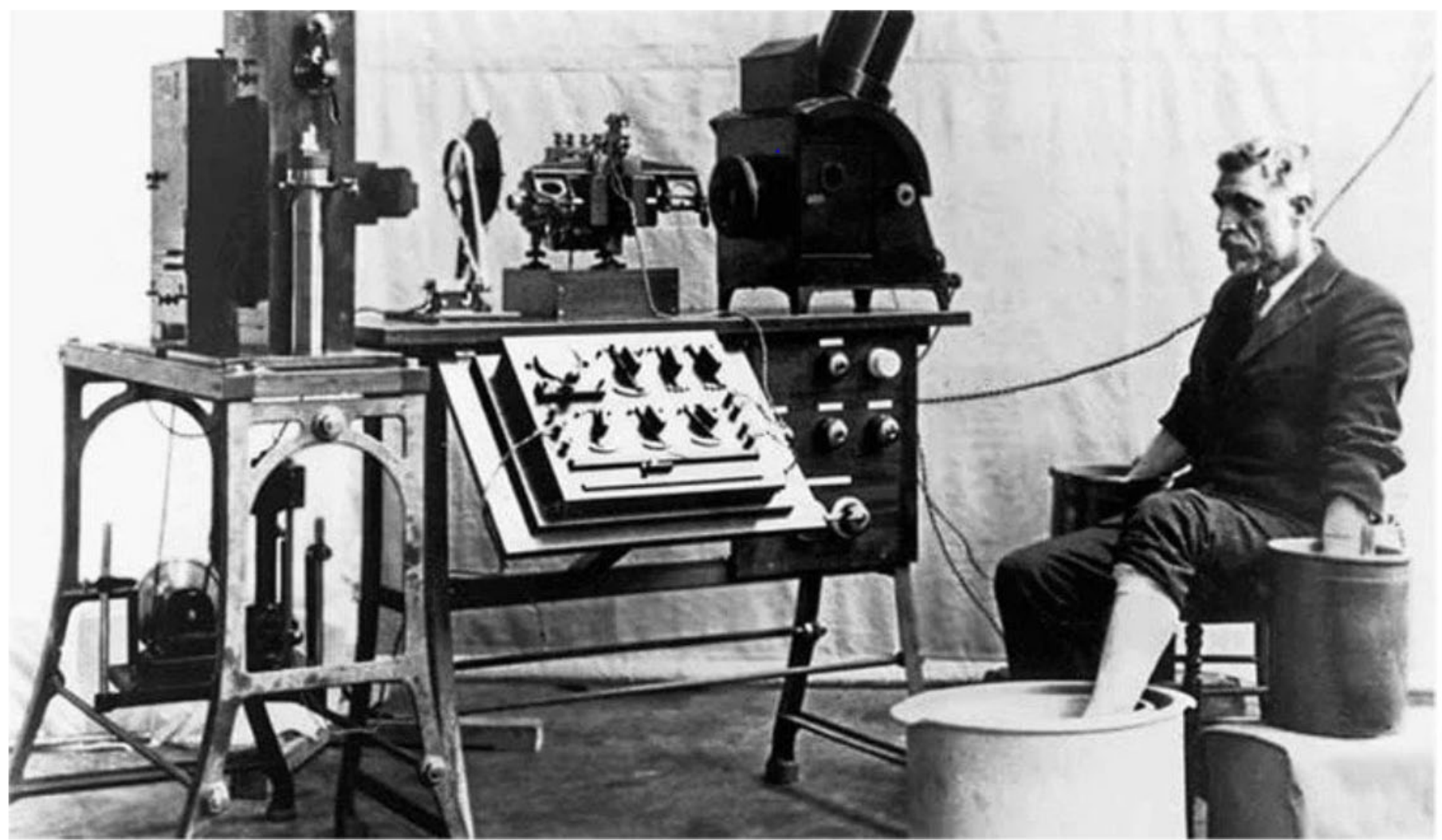
# Disclosures:

- none

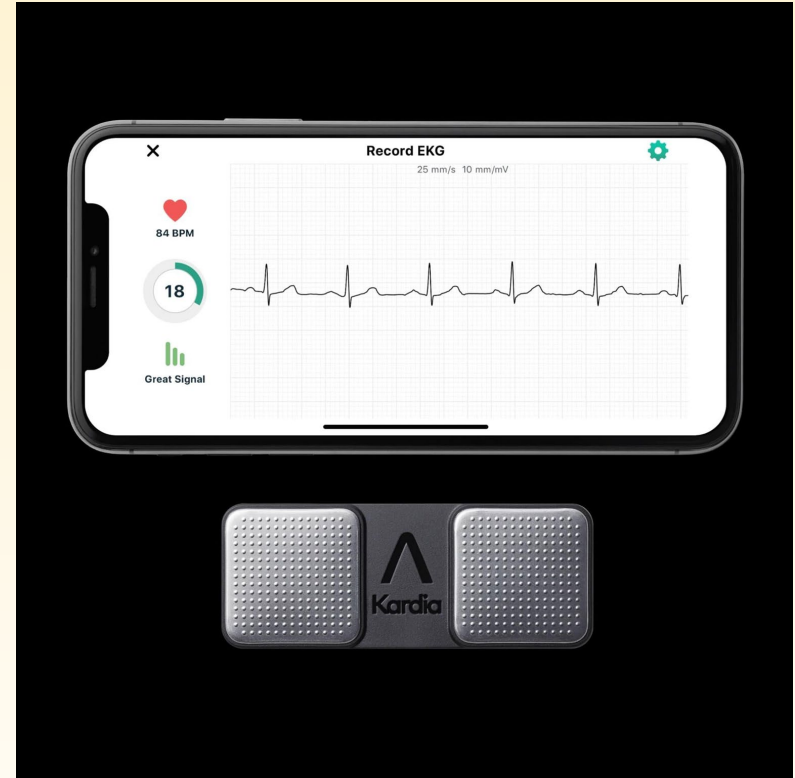
# Objectives

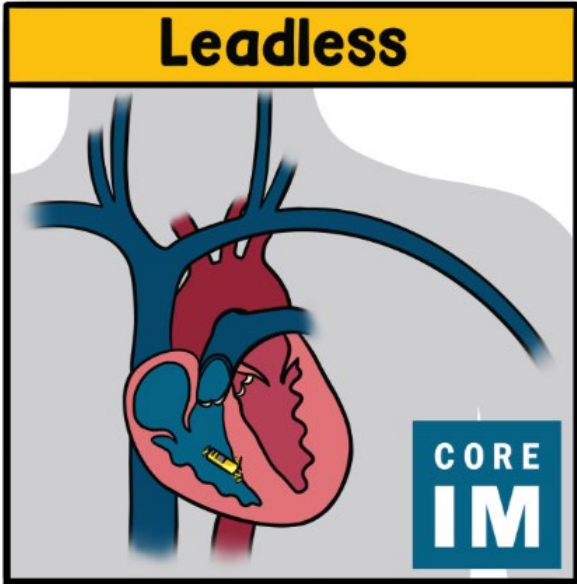
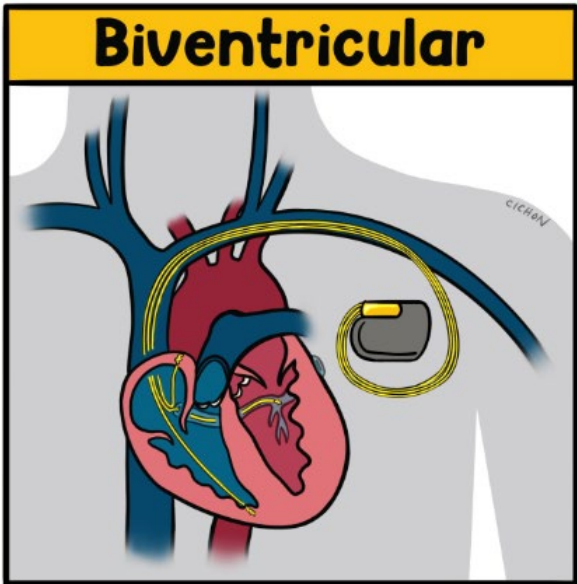
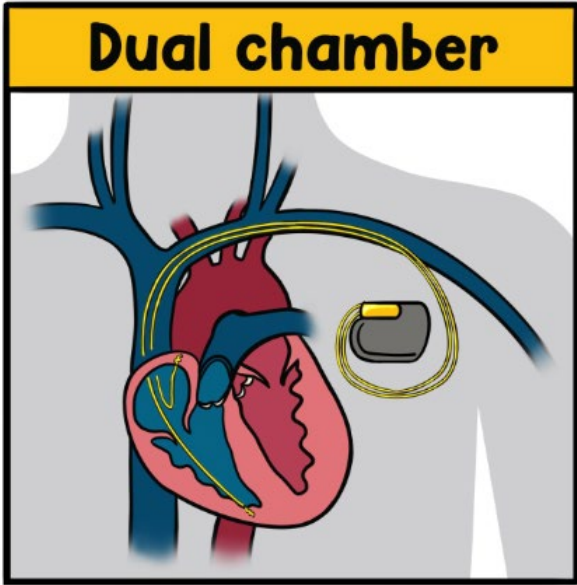
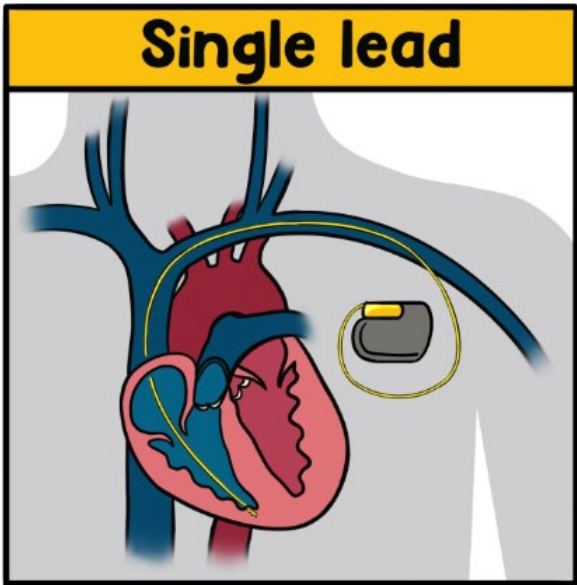
Upon completion of this presentation, attendees will be able to:

- 1) order the appropriate cardiac monitor for the indicated clinical presentation
- 2) interpret cardiac monitor device reports for data relevant to the primary care physician
- 3) increase their pre-consult efficacy and appropriateness for utilization of cardiology consult services



MAINE ACADEMY OF  
FAMILY PHYSICIANS  
STRONG MEDICINE FOR MAINE





## Understanding pacemaker modes: PACERS mnemonic

CORE  
IM

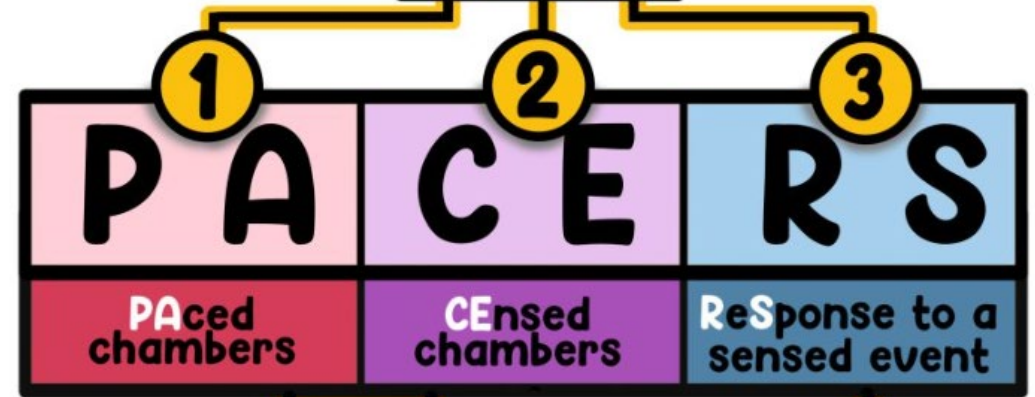
CICHOH

Ex 1: 

D	D	D
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 Ex 2: 

V	V	I
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A = Atrium  
 V = Ventricle  
 D = Dual (A + V)

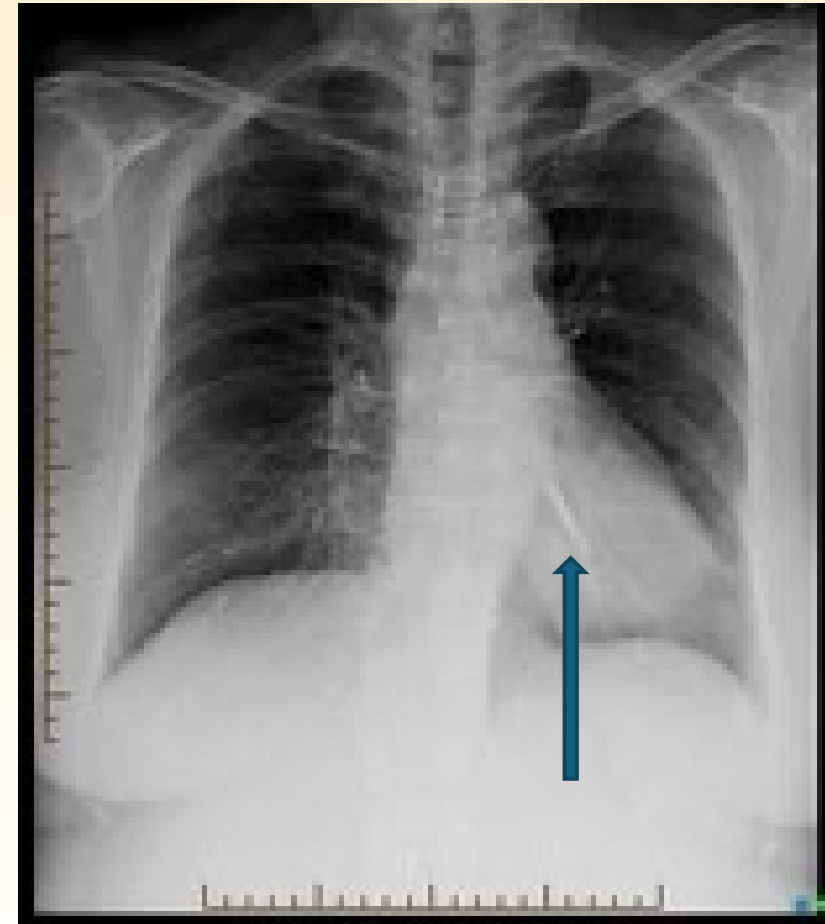
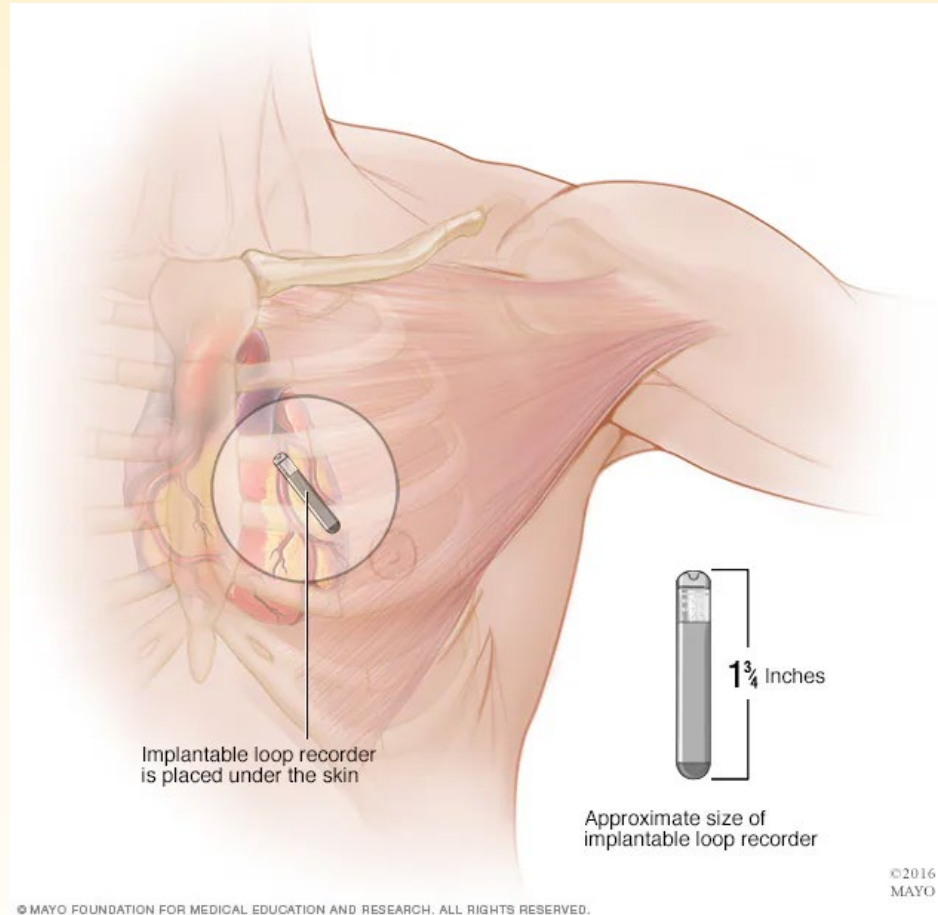
O = None  
 I = Inhibited  
 T = Triggered  
 D = Dual (I + T)

### Putting it together:

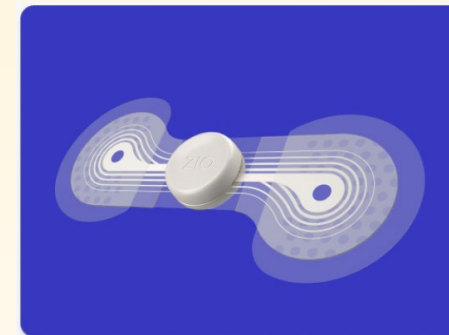
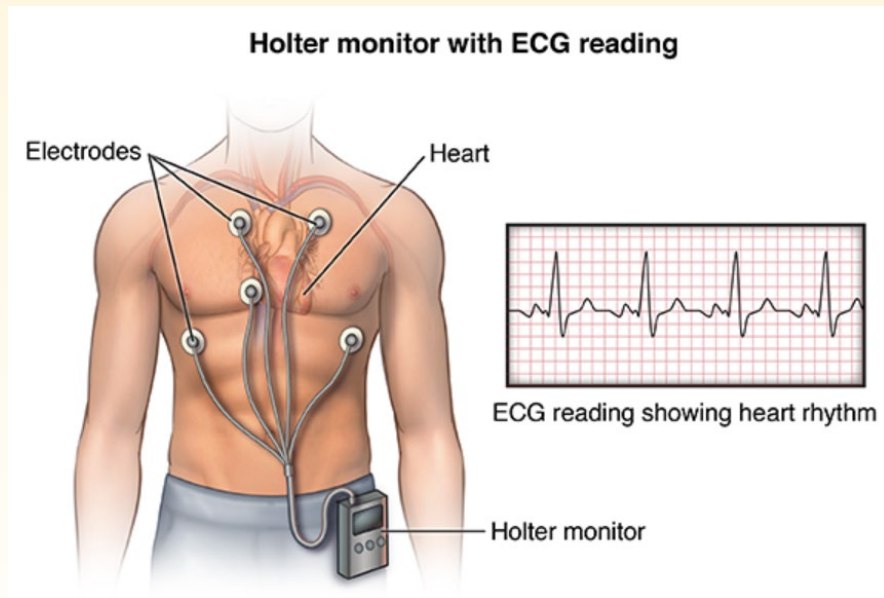
**Ex 1: DDD**  
 D = paces both atrium and ventricle  
 D = senses both atrium and ventricle  
 D = can either inhibit or trigger pacing

**Ex 2: VVI**  
 V = paces ventricle  
 V = senses ventricle  
 I = inhibits pacing if ventricular impulse is sensed

# Implantable Loop Recorders (ILR)



# “Holter” vs. Mobile Cardiac Telemetry (MCT) vs. “Event Monitor” vs. ZioPatch vs. long-term continuous monitor



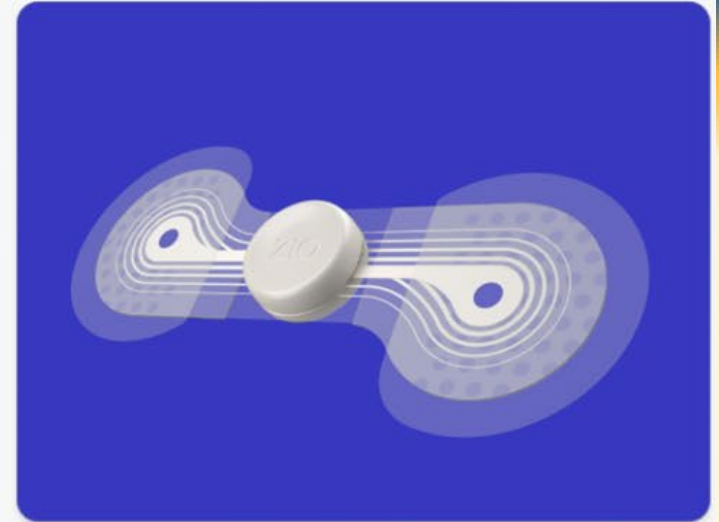
## Zio<sup>®</sup> monitor

= Zio XT = "Holter" = LTCM = event monitor

### Long-term continuous monitoring

The thinnest and lightest LTCM monitor<sup>19</sup> delivers an improved patient experience that results in 99% patient compliance with prescribed wear times.<sup>17</sup>

[Learn more](#)



## Zio AT<sup>®</sup>

### Mobile cardiac telemetry (MCT) monitoring

MCT monitoring service provides continuous, uninterrupted recording with wear-time transmissions, actionable wear-time reports, and a comprehensive end-of-wear report.<sup>20-23</sup>

[Learn more](#)



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# Order the right test:

## “Holter”/LTCM/Zio XT

- All other indications

## MCT/Zio AT

- Stroke with high suspicion for AFib
- Cardiogenic Syncope

# Dx: Palpitations

FINDINGS: The predominant underlying rhythm was sinus rhythm with a minimum sinus rate of 48 beats per minute, maximum sinus rate of 106 beats per minute, for an average sinus rate of 62 beats per minute. There were periods of bundle branch block. The patient overall had frequent supraventricular ectopy in the form of PACs with a 6.1% burden, as well as rare couplets and rare triplets. The patient did have 89 individual runs of nonsustained supraventricular tachycardia, the fastest 5 complexes with a rate of 176 beats per minute, and the longest 16 complexes with a rate of 106 beats per minute. In regard to ventricular ectopy, the patient overall had frequent ventricular ectopy in the form of PVCs with a 5.4% burden, rare couplets and rare triplets. Longest episode of bigeminy was 10.2 seconds. Longest episode of trigeminy was 11.4 seconds. The patient also did have 2 runs of nonsustained ventricular tachycardia, the fastest 11 complexes with a rate of 174 beats per minute, as well as the longest episode of 9 complexes with a slightly lower rate of 122 beats per minute. The patient triggered the device a total of 3 times and had 3 total diary entries. The diary entries were recorded by the patient as a sensation of irregular beats as well as lightheadedness and fluttering or racing of the chest. These seemed to be associated with periods of underlying sinus rhythm with periods of more frequent both supraventricular ectopy in the form of PACs as well as supraventricular couplets, as well as ventricular ectopy in the form of PVCs with ventricular couplets. Triggered events were also consistent with sinus rhythm with more frequent supraventricular or ventricular ectopy.



# Couplets, Triplets, Bigeminy, Trigeminy, oh my...

- FREQUENT supraventricular ectopy (6.1% PACs)
- FREQUENT ventricular ectopy (5.4% PVCs)
- 16 complexes of SVT
- 11 complexes of non-sustained monomorphic VTach

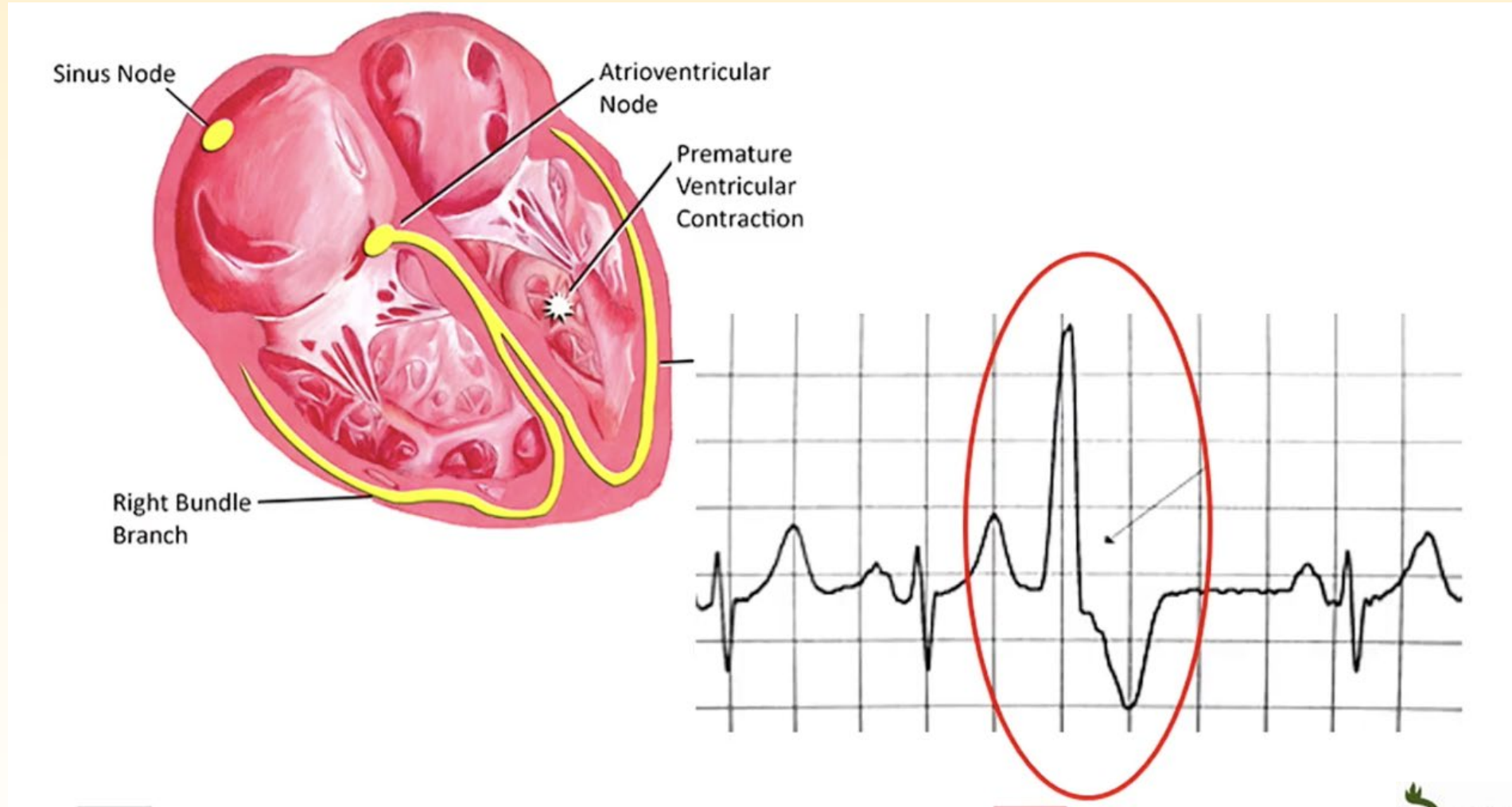
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# What is a PVC?



# PVCs

- 70% patients in 24h monitoring
- “normal” if <500 PVCs in 24 hours
  
- a/w non-cardiac issues:  
stimulants, EtOH, lytes, endocrinopathies

\*In the absence of SHD or ion channelopathies, PVCs are referred to idiopathic, and considered harmless\*

# PVCs – who to worry about:

- Fam hx SCD, h/o abrupt syncope, murmur
- High burden: >10%
- Highly symptomatic, attributable to PVCs
  
- Heart failure due to:

## **PVC-induced cardiomyopathy**

# PVCs - Treatment

- High burden
- Symptomatic

- Structural Heart Disease
- PVCi-CMP

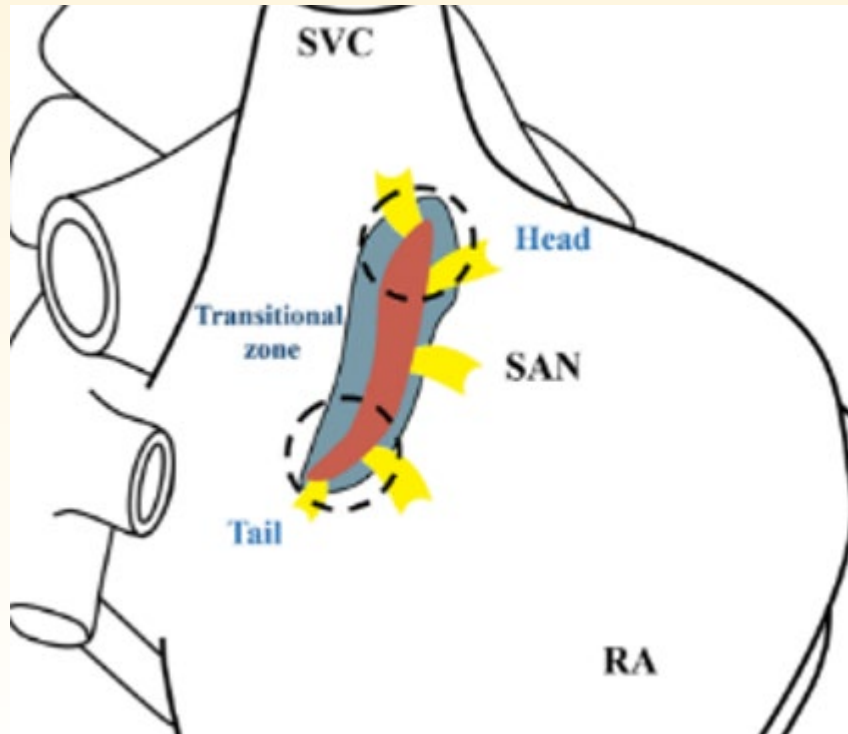
Beta-Blocker  
NDHP CCB

→ Ablation

# BRADYCARDIA

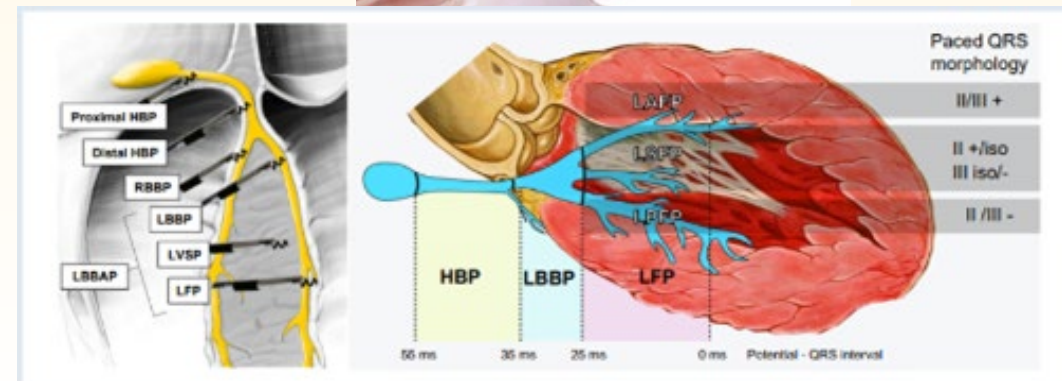
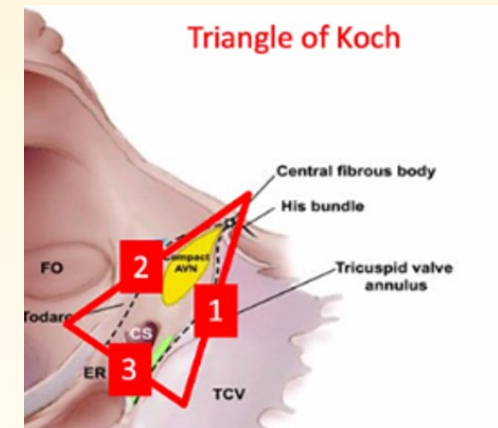
- SINUS NODE DYSFUNCTION

- Upper nodal dysfunction



- AV NODE DYSFUNCTION

- Lower nodal dysfunction



# 2018 ACC Bradycardia Guidelines

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).

A 56-year-old man is referred for pacemaker placement. He has a history of hypertension, obesity, and alcohol use disorder. He was evaluated by his primary care provider for fatigue. Physical examination findings, resting electrocardiogram (ECG), blood work results, and echocardiogram are unrevealing. A Holter monitor shows nocturnal sinus bradycardia and complete heart block with an 8-sec pause that occurred at 2 a.m. Daytime rates show normal heart rates and variability.

Which one of the following is the best next step?

**A.** Referral for sleep apnea evaluation.

**B.** Pacemaker implantation.

**C.** Implantable loop recorder.

**D.** Exercise tolerance test.

# 2018 ACC Bradycardia Guidelines

III: Harm

C-LD

1. In asymptomatic individuals with sinus bradycardia or sinus pauses that are secondary to physiologically elevated parasympathetic tone, permanent pacing should not be performed (S5.4.1-1–S5.4.1-7).

III: Harm

C-LD

2. 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 (S5.4.1-1–S5.4.1-7).

III: Harm

C-LD

3. 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 (S5.4.1-5–S5.4.1-7).



**TABLE 7 Common Potentially Reversible or Treatable Causes of SND (S5.3.1-1)**

Acute myocardial ischemia or infarction (S5.3.1-2–S5.3.1-4)

Athletic training (S5.3.1-5)

Atrial fibrillation (S5.3.1-6)

Cardiac surgery

- Valve replacement (S5.3.1-7, S5.3.1-8), maze procedure (S5.3.1-7), coronary artery bypass graft (S5.3.1-9, S5.3.1-10)

Drugs or toxins\*

- Toluene, organophosphates, tetrodotoxin, cocaine (S5.3.1-11)

Electrolyte abnormality

- Hyperkalemia (S5.3.1-12), hypokalemia (S5.3.1-13), hypoglycemia (S5.3.1-14)

Heart transplant (S5.3.1-15): Acute rejection, chronic rejection, remodeling (S5.3.1-16, S5.3.1-17)

→ Hypervagotonia (S5.3.1-18, S5.3.1-19)

Hypothermia

- Therapeutic (post-cardiac arrest cooling (S5.3.1-20)) or environmental exposure (S5.3.1-21)

→ Hypothyroidism (S5.3.1-22)

Hypovolemic shock (S5.3.1-23)

→ Hypoxemia, hypercarbia, acidosis (S5.3.1-24)

- Sleep apnea, respiratory insufficiency (suffocation, drowning (S5.3.1-25), stroke (S5.3.1-26), drug overdose)

Infection (S5.3.1-27)

- ■ Lyme disease (S5.3.1-28), legionella, psittacosis, typhoid fever, typhus, listeria (S5.3.1-29), malaria, leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre (S5.3.1-30)

Medications\*

- Beta blockers, non-dihydropyridine calcium channel blockers, digoxin (S5.3.1-31), antiarrhythmic drugs, lithium (S5.3.1-32), methyldopa, risperidone, cisplatin, interferon



# Vagal triggers

- Pain
- Nausea/suctioning/intubation
- Prolonged standing
- Heat
- Emotional (stress/fear)

# Dx: Fatigue

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# Cc: fatigue

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70yo F, during monitor, patient went for a 30 minute brisk power walk that was limited d/t exercise intolerance

# Chronotropic Incompetence

- Sinus node dysfunction
- Failure to reach optimal HR (220 minus age)
  
- Correlate monitor with activity
  - Or
- Exercise stress test

# 2018 ACC Bradycardia Guidelines

## Atrioventricular block (S2.2-7)

- **First-degree atrioventricular block:** P waves associated with 1:1 atrioventricular conduction and a PR interval  $>200$  ms (this is more accurately defined as atrioventricular delay because no P waves are blocked)
- **Second-degree atrioventricular block:** P waves with a constant rate ( $<100$  bpm) where atrioventricular conduction is present but not 1:1
  - Mobitz type I: P waves with a constant rate ( $<100$  bpm) with a periodic single nonconducted P wave associated with P waves before and after the nonconducted P wave with inconstant PR intervals
  - Mobitz type II: P waves with a constant rate ( $<100$  bpm) with a periodic single nonconducted P wave associated with other P waves before and after the nonconducted P wave with constant PR intervals (excluding 2:1 atrioventricular block)
  - 2:1 atrioventricular block: P waves with a constant rate (or near constant rate because of ventriculophasic sinus arrhythmia) rate ( $<100$  bpm) where every other P wave conducts to the ventricles
  - Advanced, high-grade or high-degree atrioventricular block:  $\geq 2$  consecutive P waves at a constant physiologic rate that do not conduct to the ventricles with evidence for some atrioventricular conduction
- **Third-degree atrioventricular block (complete heart block):** No evidence of atrioventricular conduction
- **Vagally mediated atrioventricular block:** Any type of atrioventricular block mediated by heightened parasympathetic tone
- **Infranodal block:** Atrioventricular conduction block where clinical evidence or electrophysiologic evidence suggests that the conduction block occurs distal to the atrioventricular node

# Not Every Pause is the Problem: Clinical Reasoning in Conduction System Disease

**A 68-year-old man with Bipolar d/o, HTN, Essential Tremor is referred for evaluation of recurrent presyncope and fatigue over the past several weeks after reduced PO intake d/t viral illness.**

- Episodic **lightheadedness**
- Two **near-syncope events while standing**
- No clear exertional component
- No chest pain or dyspnea

Current medications:

- Lithium, Propranolol, Lisinopril, Sertraline

Findings:

- HR: **44 bpm** BP: **112/64**; regular bradycardia without m/r/g, neg pulm + neuro exam
- Creatinine mildly elevated from baseline
- **Lithium level: 1.2 mEq/L (upper therapeutic range)**

**TABLE 4 Medications That Can Induce/Exacerbate Bradycardia or Conduction Disorders****Antihypertensive**

- Beta-adrenergic receptor blockers (including beta-adrenergic blocking eye drops used for glaucoma)
- Clonidine
- Methyldopa
- Non-dihydropyridine calcium channel blockers
- Reserpine

**Antiarrhythmic**

- Adenosine
- Amiodarone
- Dronedarone
- Flecainide
- Procainamide
- Propafenone
- Quinidine
- Sotalol

**Psychoactive**

- Donepezil
- Lithium
- Opioid analgesics
- Phenothiazine antiemetics and antipsychotics
- Phenytoin
- Selective serotonin reuptake inhibitors
- Tricyclic antidepressants

**Other**

- Anesthetic drugs (propofol)
- Cannabis
- Digoxin
- Ivabradine
- Muscle relaxants (e.g., succinylcholine)



**A 34-year-old woman presents with two episodes of syncope occurring shortly after standing from a seated position with**

- Lightheadedness ,flushing/warmth
- Dimming vision prior to loss of consciousness
- Rapid recovery without confusion
- No structural heart disease, No family history of sudden death
- **Orthostatic vitals positive in clinic**

### **Ambulatory Rhythm strip finding**

- During sleep at **3:12 AM**
- **Sinus pause lasting 5.2 seconds**
- No associated symptoms

A **72-year-old man** presents for evaluation of **two unexplained syncopal episodes**.

Episode characteristics:

- One event occurred **suddenly while walking with no prodrome**
- Another event was preceded by **lightheadedness**

Baseline ECG shows:

- **Left Anterior Fascicular Block**
- **First-degree AV delay (PR 260 ms)**

Because of concern for conduction disease, he undergoes **ambulatory rhythm monitoring**.

**Rhythm strip findings**

- Episodes of **Mobitz I (Wenckebach) AV block**
- **No prolonged pauses**



An **81-year-old woman** is referred for outpatient evaluation after **two sudden syncopal episodes** over the past month.

Episode characteristics:

- **Abrupt loss of consciousness without prodrome**
- Witnessed collapse while standing
- Rapid recovery within seconds

Baseline clinic ECG shows:

- **Sinus rhythm, PR interval 210 ms**

Because of the concerning history, she undergoes **ambulatory rhythm monitoring**.

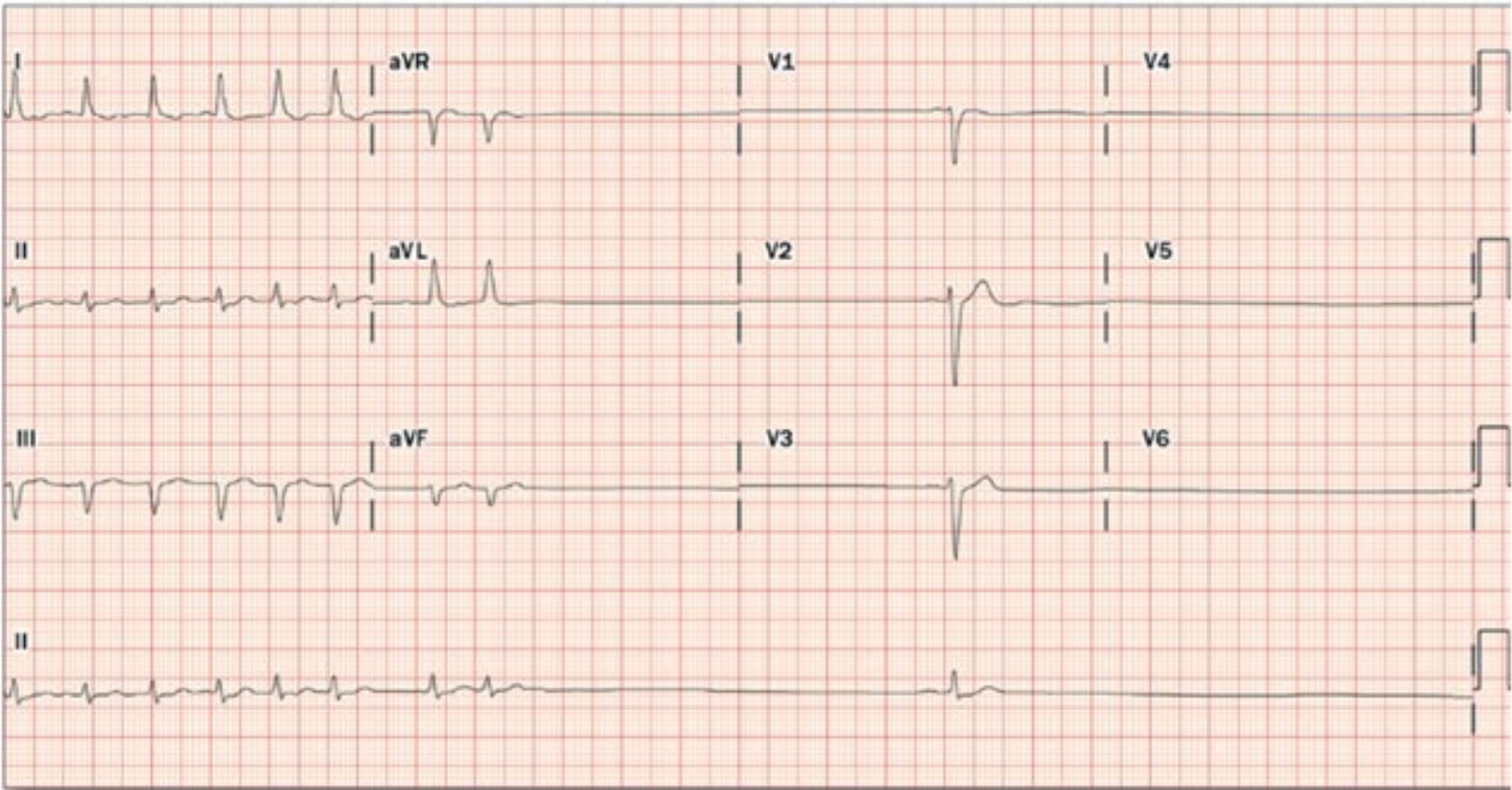
At **2:14 PM while awake**, the monitor captures:

- **Abrupt complete AV block**
- Ongoing atrial activity
- **No ventricular escape for ~7 seconds**
- Followed by slow ventricular escape rhythm



The patient reported **sudden dizziness during the episode**.

An 83-year-old woman with hypertension, hyperlipidemia, and paroxysmal atrial fibrillation presents to the emergency department with syncope and scalp laceration. Besides a headache, she has no other symptoms. She reports that her sister has atrial fibrillation and recently had a pacemaker implanted. Head computed tomography is negative for intracranial hemorrhage. Echocardiography reveals an ejection fraction of 60%. Her medications are atorvastatin 10 mg daily, metoprolol succinate 25 mg daily, and apixaban 2.5 mg twice daily and have been unchanged for 2 years. Her 12-lead electrocardiogram (ECG) is shown (Figure 1)



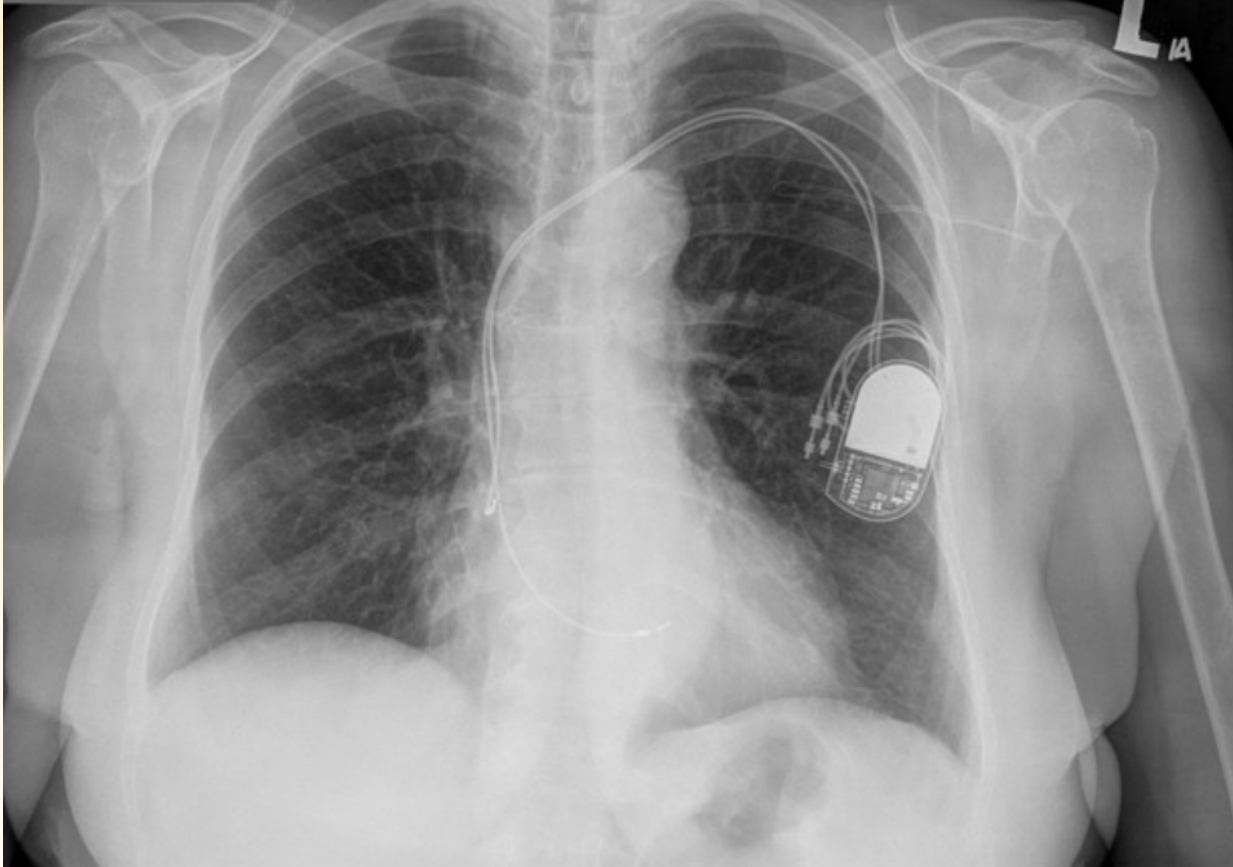
# Chronic PPM mgmt

- Lead failure, dislodgment, battery life
- Infectious (especially staph bacteremia)
- Dyssynchrony and cardiomyopathies

# Pacemaker vs. Defibrillator

- **Permanent Pacemaker (PPM)**
  - Record data
  - Pace chamber
- **Internal Cardioverter-Defibrillator (ICD)**
  - Record data
  - Pace chamber
  - Disrupt focal arrhythmia (Anti-tachycardia pacing)
  - Defibrillate

# Does my patient have an ICD?



# End of Life Care

- Pacemakers do NOT need to be shut off at end of life
  - Low pH, severe electrolyte disturbances, cardiac hypoxia will lead to electrical impulses without captured contraction response
  - Pacemaker may need to be considered in discussion of life-sustaining treatment (eg. In cases of brain death but ongoing cardiopulmonary function)
- ICD's SHOULD be turned off if withdrawing life-sustaining care
  - Strong magnet over device will deactivate it
  - Blue donut magnet on code carts

THANK YOU!