Syncope: A Practical Approach

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Disclosures

No relevant disclosures

Objectives

1. Understand the varying presentation of the three different types of syncope
2. Appreciate high and low risk features
3. Determine the type of investigations based on presenting features
4. Understand the role of different testing modalities

Epidemiology

Syncope is common: 1-1.5% of emergency department visits

Lifetime incidence of 32-35% of the general population (Canada, Netherlands)

12-15% of patients are admitted to hospital (an overall decrease): 0.9/1000 population in New Brunswick, to 0.3/1000 in Alberta and Manitoba. Overall lower rate than other countries.

In-hospital mortality 0.7% (0.4-1.1%) across the provinces

Economic burden: including hospitalization, outpatient visit, and physician and drug cost: >90$ million/year (Alberta between 2009-2014)

Syncope and TLOC: Transient Loss of Consciousness

Syncope : TLOC due to cerebral hypoperfusion, characterized by a rapid onset, short duration, and spontaneous complete recovery.

A sudden cessation of cerebral blood flow for as short as 6-8 seconds can cause complete loss of consciousness.

A systolic blood pressure of 50-60mmHg (30-45mmHg at the brain level) in the upright position, can similarly cause a loss of consciousness

Presyncope: symptoms and signs that occur before unsconsciousness in syncope

Syncope

Non-Cardiac Syncope

Reflex Syncope

Vasovagal: orthostatic and emotional types

Situational:
Micturition, GI stim (swallowing), coughing, sneezing, post-exercise

Carotid sinus syndrome

Orthostatic intolerance

Exacerbated by venous pooling during exercise, post-prandial, and prolonged bedrest

Dehydration
Medication: vasodilators, diuretics, anti-depressants, phenothiazines
Neurogenic:
- -primary autonomic failure
  - -Parkinsons, MSA
- -secondary autonomic f.
  - -DM, amyloid, CKD

POTS: postural orthostatic tachycardia
Orthostatic hypotension

Cardiac Syncope

Arrhythmic: brady, tachy
Structural: Mi, aortic stenosis, HCM, cardiac masses, tamponade, congenital…
Cardiopulmonary: PE, aortic dissection, pulm htn

Conditions Incorrectly Diagnosed as Syncope

Seizures
Falls without transient loss of consciousness: no unresponsiveness or amnesia present
Intracerebral or subarachnoid hemorrhage
TIA:
Subclavian steal syndrome: focal neurological signs
Metabolic disorders: hypoglycemia, hypoxia, hyperventilation: much longer duration than true TLOC; consciousness may be impaired and not lost
Intoxication: duration longer than TLOC
Cardiac arrest: no spontaneous recovery
Coma: much longer duration

Neurologic causes of syncope, such as TIA, stroke, or seizure are almost always associated with features suggestive of the underlying cause: e.g. deficits in speech/motor weakness
-These account for <5% of all causes of syncope

Red Flags

History or sign of cardiovascular disease – aortic stenosis, outflow tract obstruction, heart failure, myocardial infarction
Syncope during exertion
Lack of prodrome
Palpitations at time of syncope
Family history of early sudden cardiac death
Risk factors on ECG: e.g. bifascicular block
Mobitz 1 second degree or complete heart block
Ischemic changes: ST depressions, T wave inversions
Brugada syndrome
Prolonged QT
New neurologic deficits
Seizure

Initial Evaluation

Initial Evaluation: Detailed History Most Important

Ensure that there was actually transient loss of consciousness (TLOC): Short duration

a) Abnormal motor control
b) Loss of responsiveness
c) Amnesia for the period of LOC

1. Initial history should include present and previous syncopal attacks, and witness accounts

2. Physical exam: supine and standing BP measurements, and auscultation for murmurs

3. Electrocardiogram; Monitoring if there is suggestion of arrhythmia (almost everyone)

4.Echocardiogram: previous known heart disease, data suggestive of structural heart disease, or syncope secondary to cardiovascular cause

5. Carotid sinus massage (in pts > age 40)
6. Bloodwork: Hb, troponin, blood gas, D-dimer

Echocardiography

Recommendations	Class[a]	Level[b]
Indications		
Echocardiography is indicated for diagnosis and risk stratification in patients with suspected structural heart disease.[235,236]	I	B
Two-dimensional and Doppler echocardiography *during exercise* in the standing, sitting, or semi-supine position to detect provocable left ventricular outflow tract obstruction is indicated in patients with HCM, a history of syncope, and a resting or provoked peak instantaneous left ventricular outflow tract gradient <50 mmHg.[245–249]	I	B
Diagnostic criteria		
Aortic stenosis, obstructive cardiac tumours or thrombi, pericardial tamponade, and aortic dissection are the most probable causes of syncope when the electrocardiogram shows the typical features of these conditions.[237–244]	I	C

Exercise testing

Recommendations	Class[a]	Level[b]
Indications		
Exercise testing is indicated in patients who experience syncope during or shortly after exertion.	I	C
Diagnostic criteria		
Syncope due to second- or third-degree AV block is confirmed when the AV block develops during exercise, even without syncope.[253–257]	I	C
Reflex syncope is confirmed when syncope is reproduced immediately after exercise in the presence of severe hypotension.[250–252]	I	C
Additional advice and clinical perspectives There are no data supporting routine exercise testing in patients with syncope.		

Coronary angiography

Recommendations	Class[a]	Level[b]
Indications		
In patients with syncope, the same indications for coronary angiography should be considered as in patients without syncope.[258]	IIa	C
Additional advice and clinical perspectives Angiography alone is not diagnostic of the cause of syncope.		

[a]Class of recommendation.
[b]Level of evidence.

Clinical Features that can Suggest a Diagnosis on the Initial Evaluation

1. Syncope due to Orthostatic Hypotension

-While standing, or after standing

-Prolonged standing

-Standing after exertion

-Post-prandial hypotension

-Temporal relationship with changes in doses of vaso-depressive drugs/diuretics

-Presence of an autonomic neuropathy, or Parkinsons

Measuring Orthostatic Blood Pressure

1. Have the patient lie down for 5 minutes.
2. Measure blood pressure and pulse rate.
3. Have the patient stand.
4. Repeat blood pressure and pulse rate measurements after standing 1 and 3 minutes.

Diagnostic response: At 3 minutes: Progressive or sustained fall in systolic BP from a baseline value ≥ 20mmHg or diastolic BP ≥ 10mmHg, or a decrease in systolic BP to <90mmHg, with symptoms consistent with orthostatic hypotension

POTS (postural orthostatic tachycardia syndrome):

Can be diagnosed when there is an orthostatic HR increase (>30 bpm or to >120 bpm within 10 minutes of active standing).

Can also consider 24 hour Blood pressure monitoring to evaluate for orthostatic hypotension

2. Reflex Syncope: Situational or Vasovagal Syncope

-Long history of recurrent syncope, particularly occurring < age 40

-After unpleasant sight, sound, smell or pain

Vasovagal: Class I

-Prolonged standing

-During meal

-Being in crowded or hot place

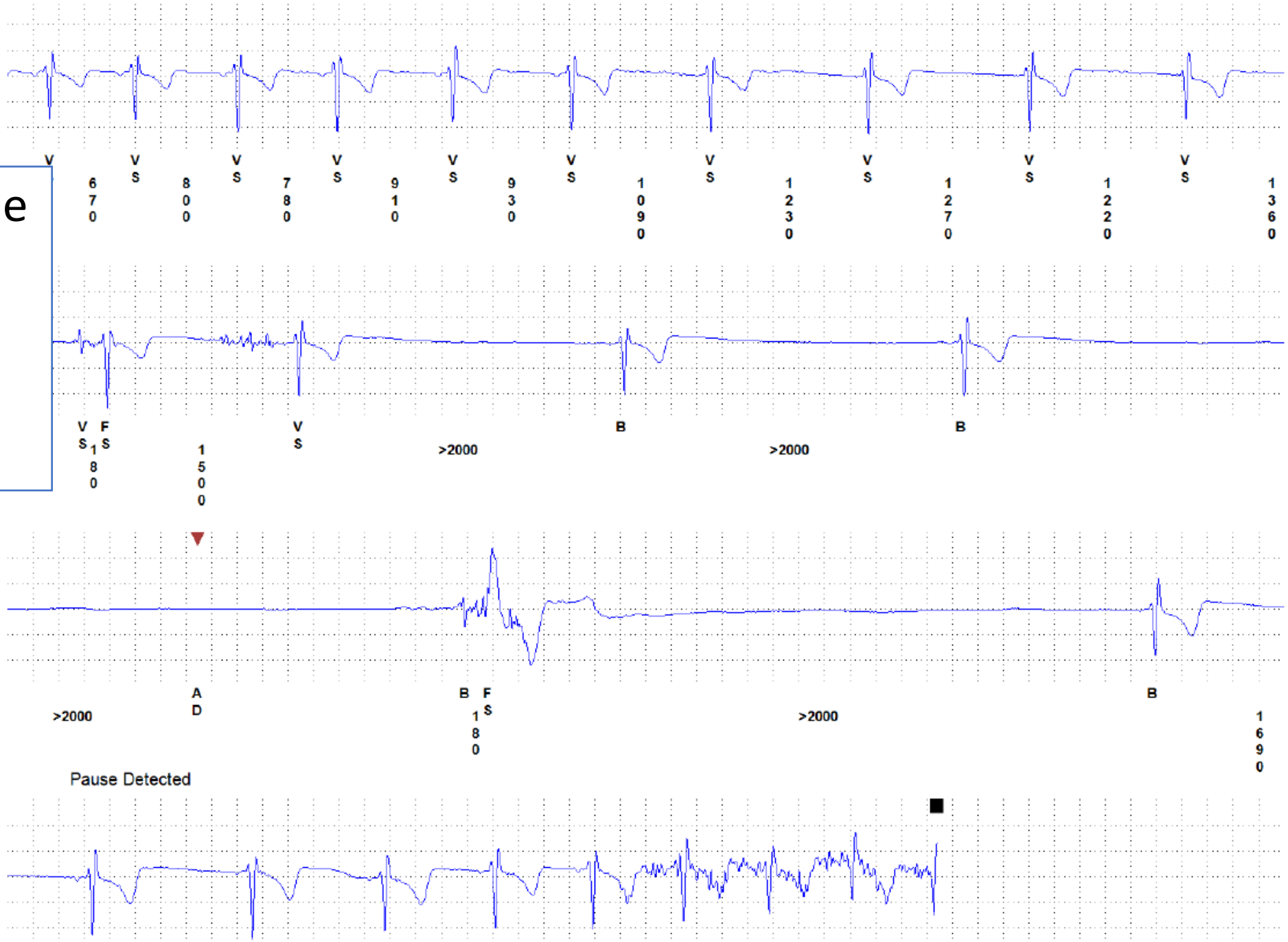
Vasovagal: Class I

-Autonomic activation before syncope: ie. pallor, sweating and/or nausea+vomiting

-With head rotation or pressure on carotid sinus (tumors, shaving, tight collar)

-Absence of heart disease

28 year old male
4 episodes of syncope, typically after a hot shower



Treatment for Vasovagal episodes?

- Avoidance of triggers
- Increased salt and fluid intake: no clinical trials
- Counterpressure maneuvers: open-label randomized studies
- Fludrocortisone: benefit shown in secondary analyses
- ?selective serotonin reuptake inhibitors
- Dual-chamber pacing: syncope and asystole during positive tilt test
- (Cardioneural ablation)

Annals of Internal Medicine

ORIGINAL RESEARCH

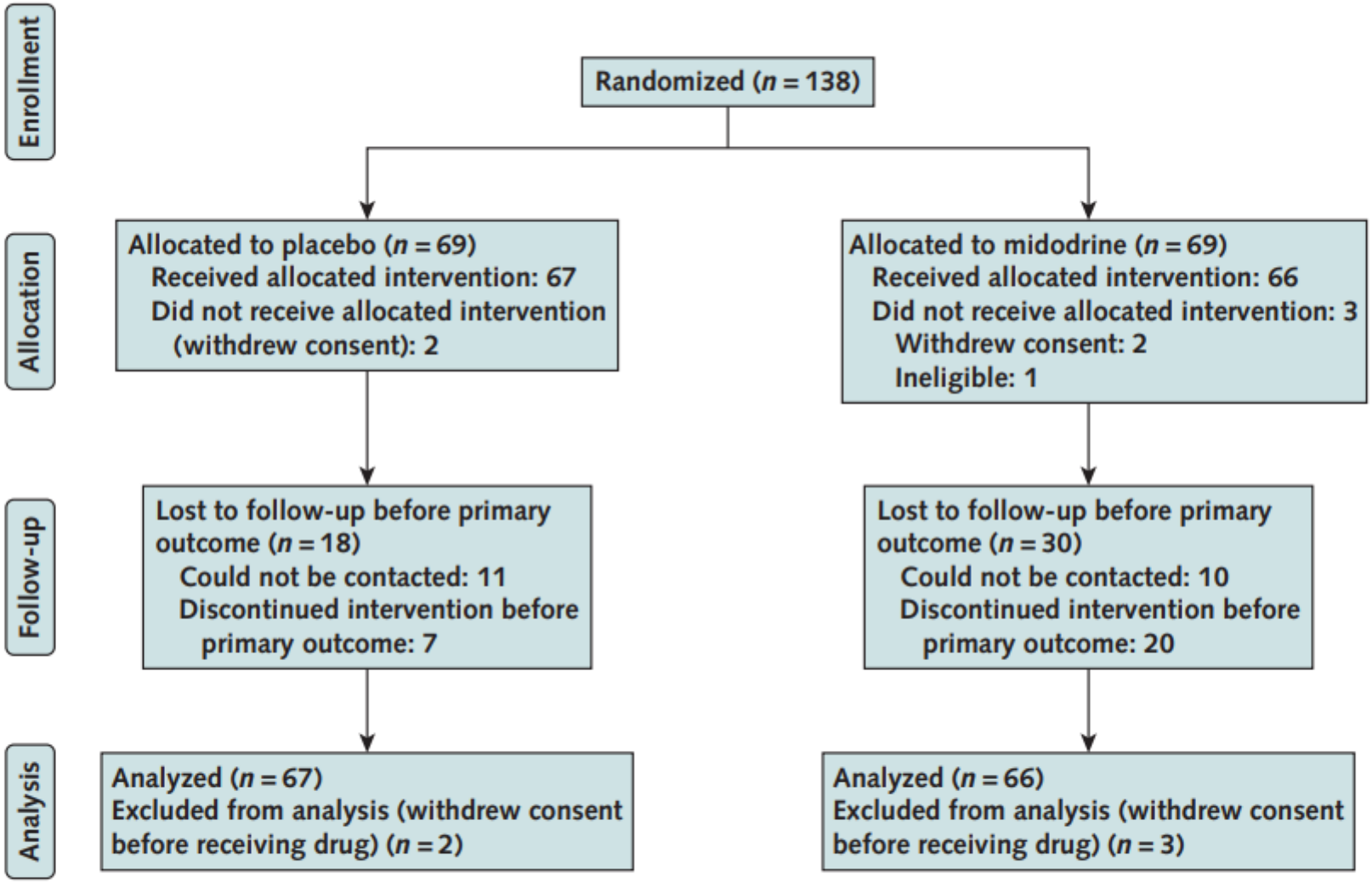
Midodrine for the Prevention of Vasovagal Syncope

A Randomized Clinical Trial

Robert Sheldon, MD, PhD; Peter Faris, PhD; Anthony Tang, MD; Felix Ayala-Paredes, MD; Juan Guzman, MD, MSc; Manlio Marquez, MD; Carlos A. Morillo, MD; Andrew D. Krahn, MD; Teresa Kus, MD, PhD; Debbie Ritchie, MN; Shahana Safdar, PhD; Connor Maxey, BSc; and Satish R. Raj, MD, MSCI; for the POST 4 investigators*

2021-Oct, Annals int med

Figure 1. Study flow diagram.



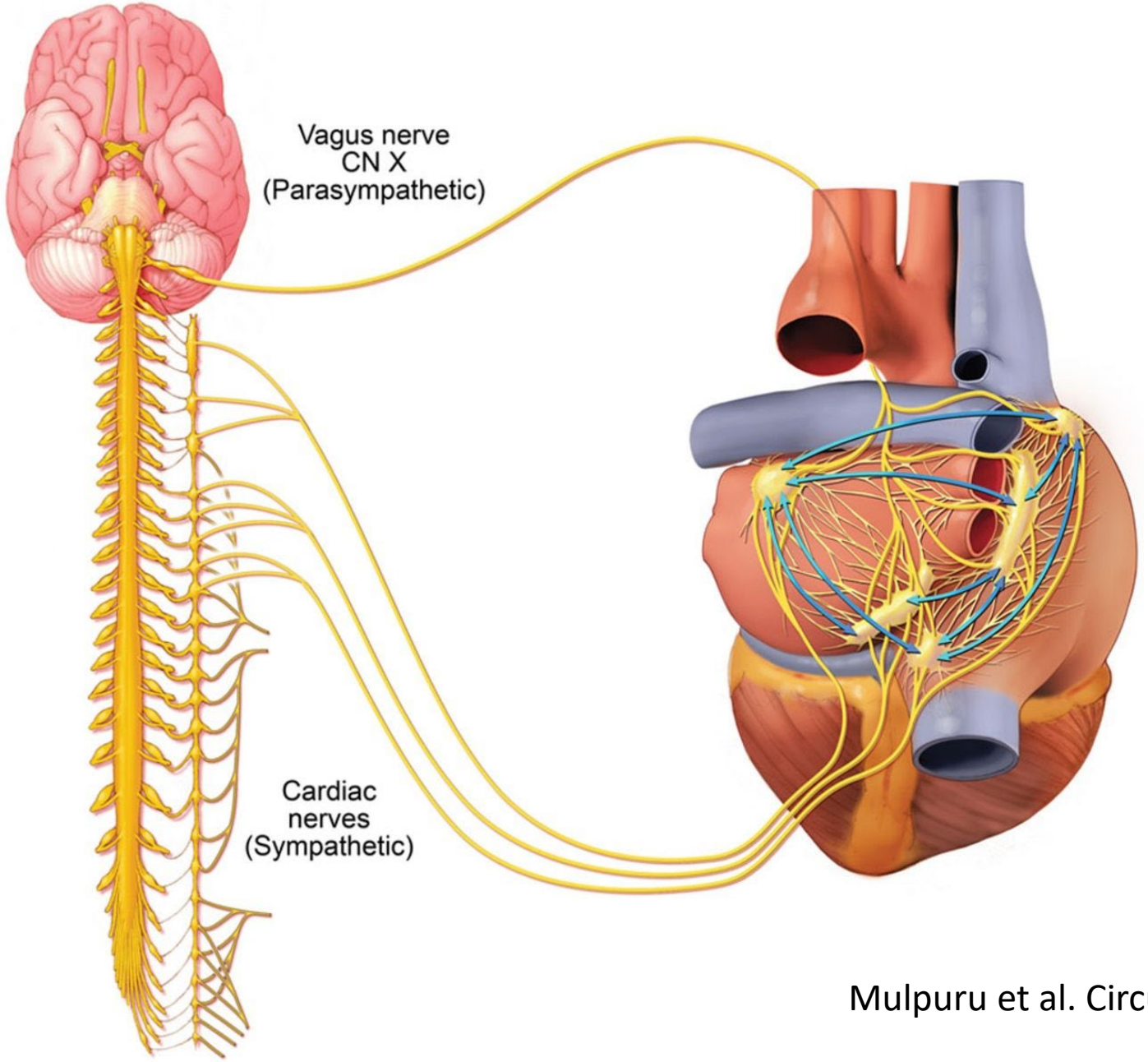
The reasons for withdrawal of the study drug in the placebo group were adverse effects ($n = 2$), stopped fainting ($n = 1$), continued fainting ($n = 2$), study fatigue ($n = 1$), and lost contact ($n = 1$). The reasons for withdrawal of the study drug in the midodrine group were adverse effects ($n = 2$), stopped fainting ($n = 1$), continued fainting ($n = 5$), study fatigue ($n = 1$), family doctor preference ($n = 1$), other ($n = 3$), and lost contact ($n = 6$).

Table 1. Baseline Characteristics of Study Treatment Groups

Characteristic	Placebo (*n* = 67)	Midodrine (*n* = 66)
Median age (IQR), *y*	35 (27–47)	31 (25–43)
Female sex, *n (%)*	50 (75)	48 (72)
Syncope history		
Median age of onset (IQR), *y*	18 (14–27)	17 (14–25)
Median lifetime syncope episodes (IQR), *n*	23 (11–250)	21 (10–100)
Median symptom duration (IQR), *y*	14 (4–25)	14 (3–26)
Median syncope frequency (IQR), *episodes/y*	5 (1–20)	4 (1–9)
Median Calgary Syncope Symptom Score (IQR)	3 (1–5)	3 (1–4)
Median syncope episodes in previous year (IQR), *n*	7 (4–25)	5 (3–12)
Previous medical therapy for syncope, *n*		
Salt supplements	21	25
Increased fluid	33	34
Fludrocortisone	8	11
β-Blocker	12	9
Disopyramide	1	1
SSRI	5	5
Median supine systolic BP (IQR), *mm Hg*	118 (110–127)	116 (108–124)
Median supine heart rate (IQR), *beats/min*	68 (62–81)	72 (62–82)

BP = blood pressure; IQR = interquartile range; SSRI = selective serotonin reuptake inhibitor.

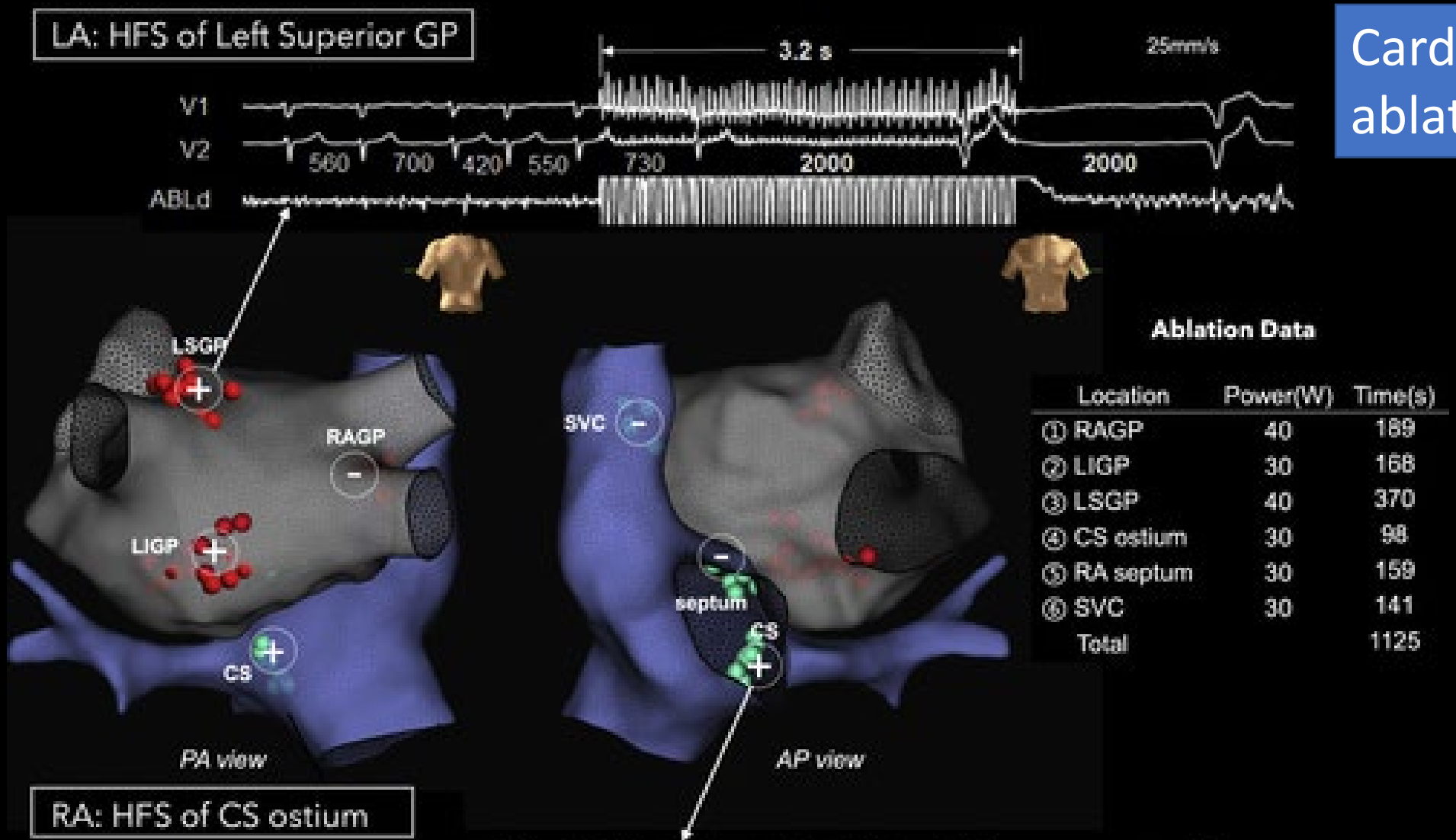
Alternative Treatment for Vasovagal episodes?



Intrinsic cardiac nervous system: clusters of nerve fibers in the epicardial fat tissue are referred to as ganglia.

Mulpuru et al. Circulation; 2017 vol 10 issue 2

Cardioneural ablation



Ablation Data

Location	Power(W)	Time(s)
① RAGP	40	189
② LIGP	30	168
③ LSGP	40	370
④ CS ostium	30	98
⑤ RA septum	30	159
⑥ SVC	30	141
Total		1125

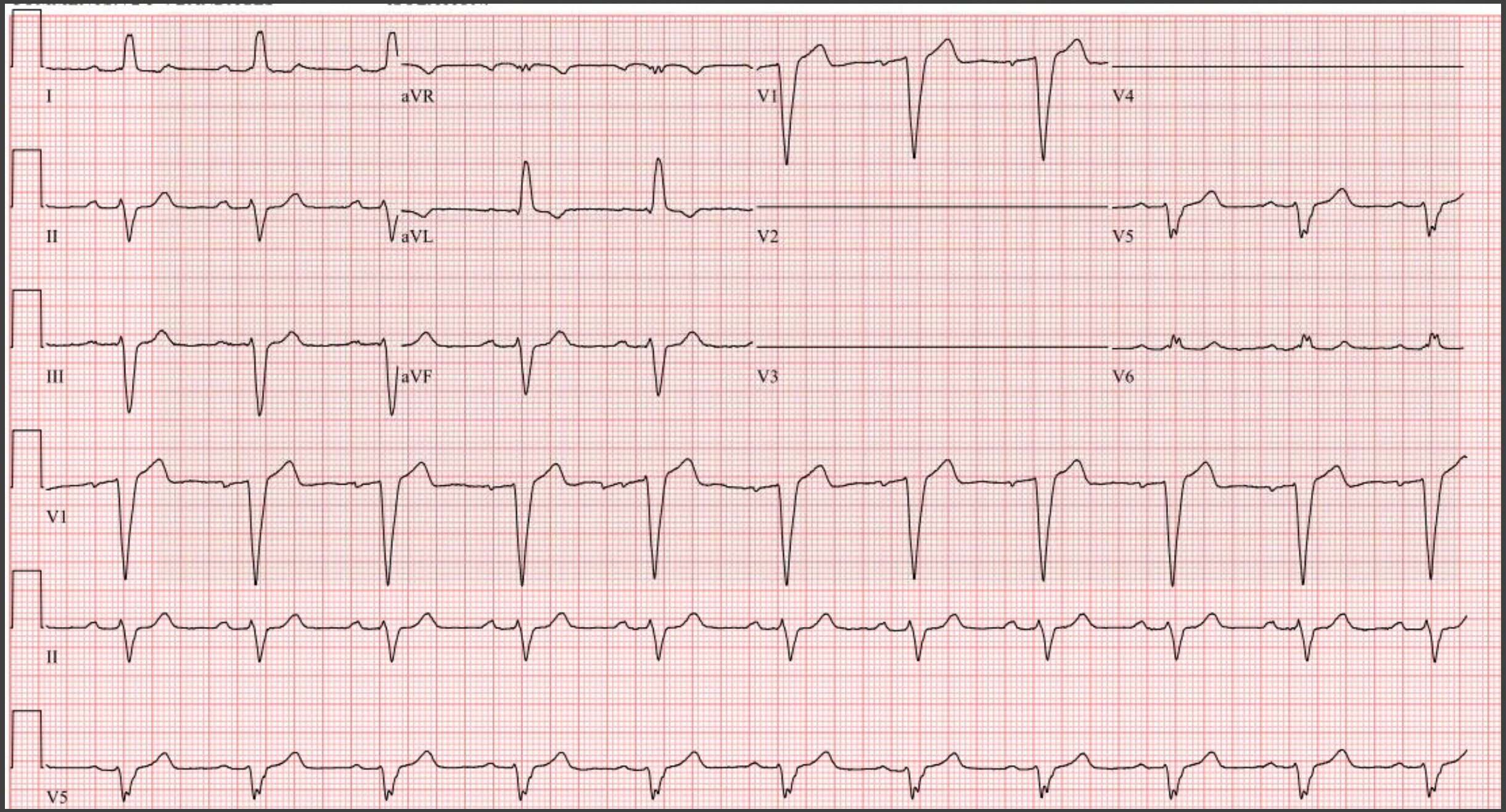
Lu, Tung JACC 2020 Jul, 2(8) 1161-

Case.
53 year-old man with no past medical history, no family history, no medications

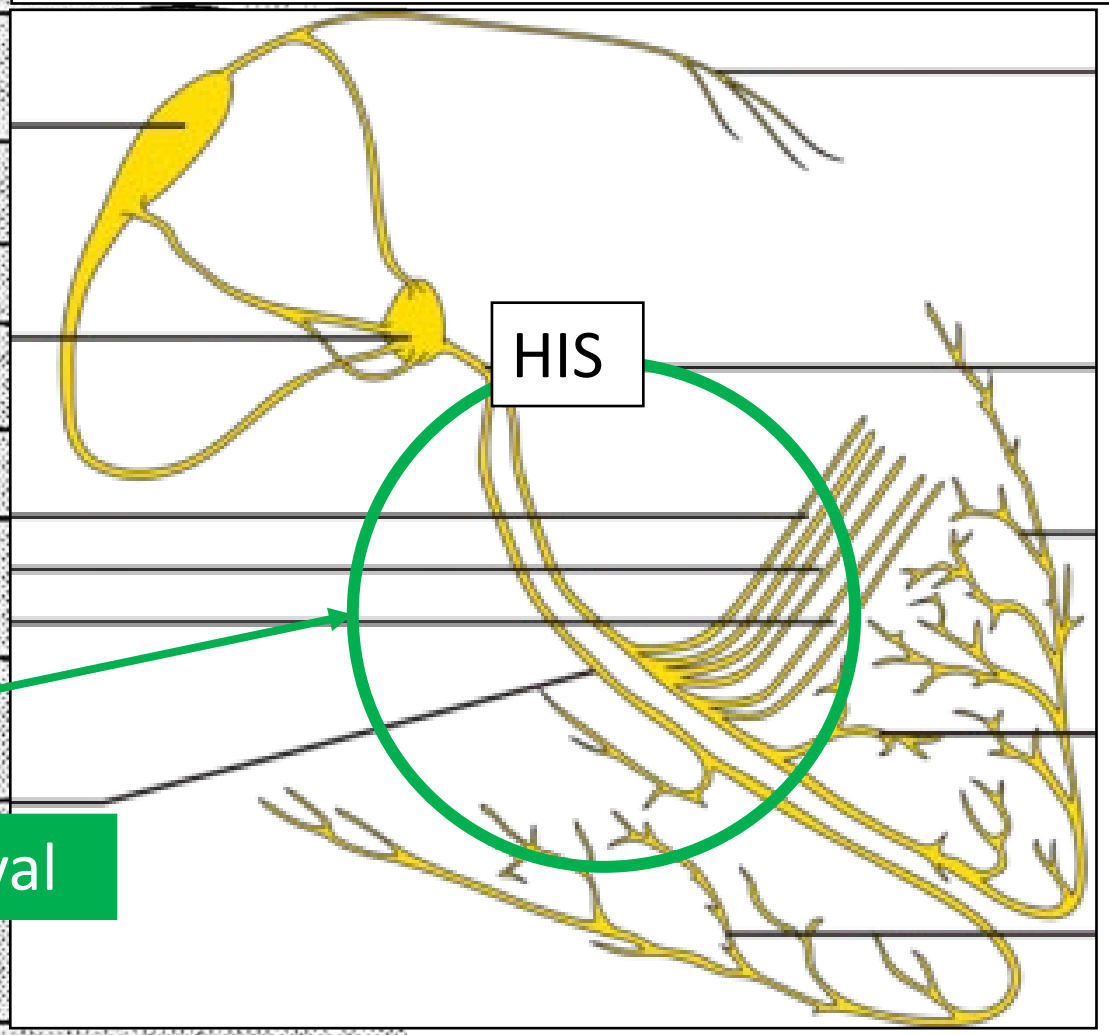
Presented for work-up of syncope.

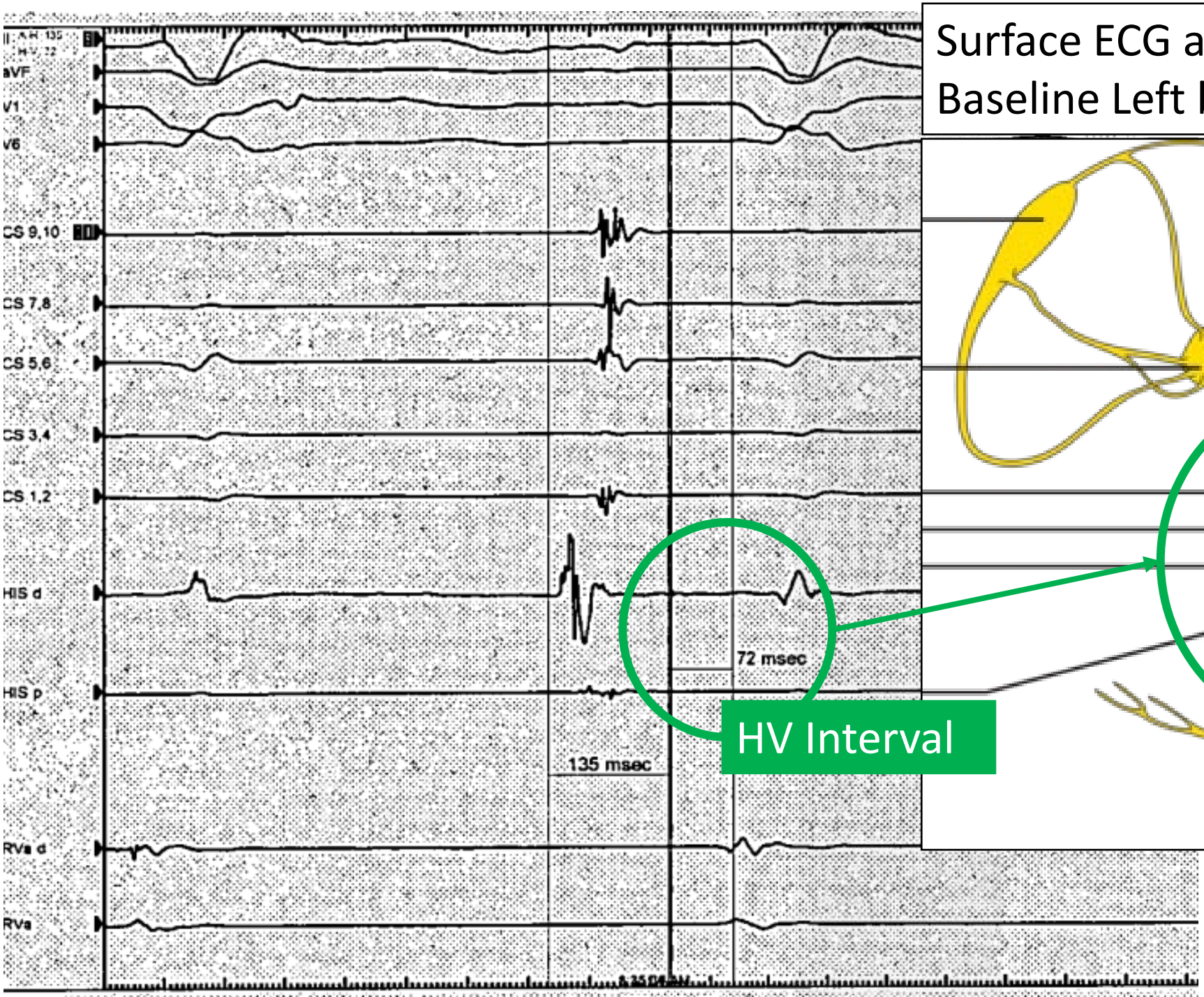
1st episode: He was playing badminton and then sudden loss of consciousness. No warning. Within 3 seconds of syncope, was back to his usual state. Initial investigations normal.

2nd episode: 6 months later. Returned to play, and had a similar episode with a "head rush" . Transthoracic echo demonstrated a mildly reduced LVEF at 50%. Coronary angiogram negative for any significant coronary disease.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Surface ECG at 200mm/s paper speed
Baseline Left bundle branch block





1 year later, sitting at his desk, felt a heat wave through him and put his head down on his desk. Unsure if he passed out, but extreme weakness

3. Cardiac Syncope

- **<u>During exertion***</u>**
- Sudden palpitations immediately before syncope
- Family history of unexplained sudden death at age <50
- Presence of structural heart disease (valvulopathy – aortic stenosis) or coronary artery disease
- ECG findings
- Head or facial trauma

ECG[a]	
Low-risk	
• Normal ECG[26, 35, 36, 55]	
High-risk	
Major	**Minor** (high-risk only if history consistent with arrhythmic syncope)
• ECG changes consistent with acute ischaemia • Mobitz II second- and third-degree AV block • Slow AF (<40 b.p.m.) • Persistent sinus bradycardia (<40 b.p.m.), or repetitive sinoatrial block or sinus pauses >3 seconds in awake state and in absence of physical training • Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy, or Q waves consistent with ischaemic heart disease or cardiomyopathy[44, 56] • Sustained and non-sustained VT • Dysfunction of an implantable cardiac device (pacemaker or ICD) • Type 1 Brugada pattern • ST-segment elevation with type 1 morphology in leads V1–V3 (Brugada pattern) • QTc >460 ms in repeated 12-lead ECGs indicating LQTS[46]	• Mobitz I second-degree AV block and 1°degree AV block with markedly prolonged PR interval • Asymptomatic inappropriate mild sinus bradycardia (40–50 b.p.m.), or slow AF (40–50 b.p.m.)[56] • Paroxysmal SVT or atrial fibrillation[50] • Pre-excited QRS complex • Short QTc interval (≤340 ms)[46] • Atypical Brugada patterns[46] • Negative T waves in right precordial leads, epsilon waves suggestive of ARVC[46]

07-APR-1943 (77 yr)
Male

Room:
Loc:17

Vent. rate	59	BPM
PR interval	364	ms
QRS duration	150	ms
QT/QTc	530/524	ms
P-R-T axes	33 -25	130

Sinus bradycardia with sinus arrhythmia with 1st degree AV block
Possible Left atrial enlargement
Left bundle branch block
Abnormal ECG

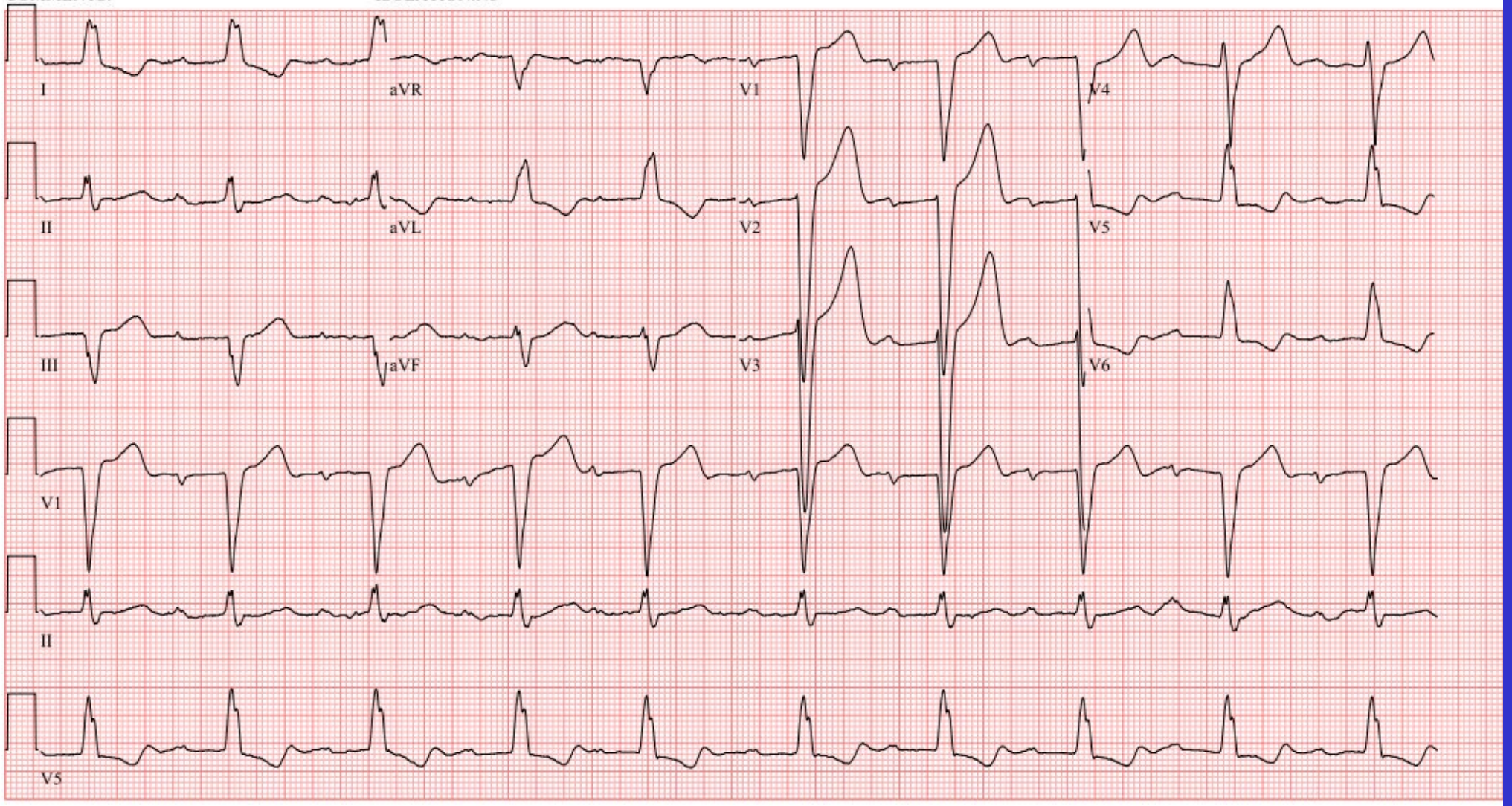
Technician:Raphaël Cafaro TEPM
Test ind:

Referred by: C7

Confirmed By: James Brophy MD

COMMENTS:

ISOLATION:No



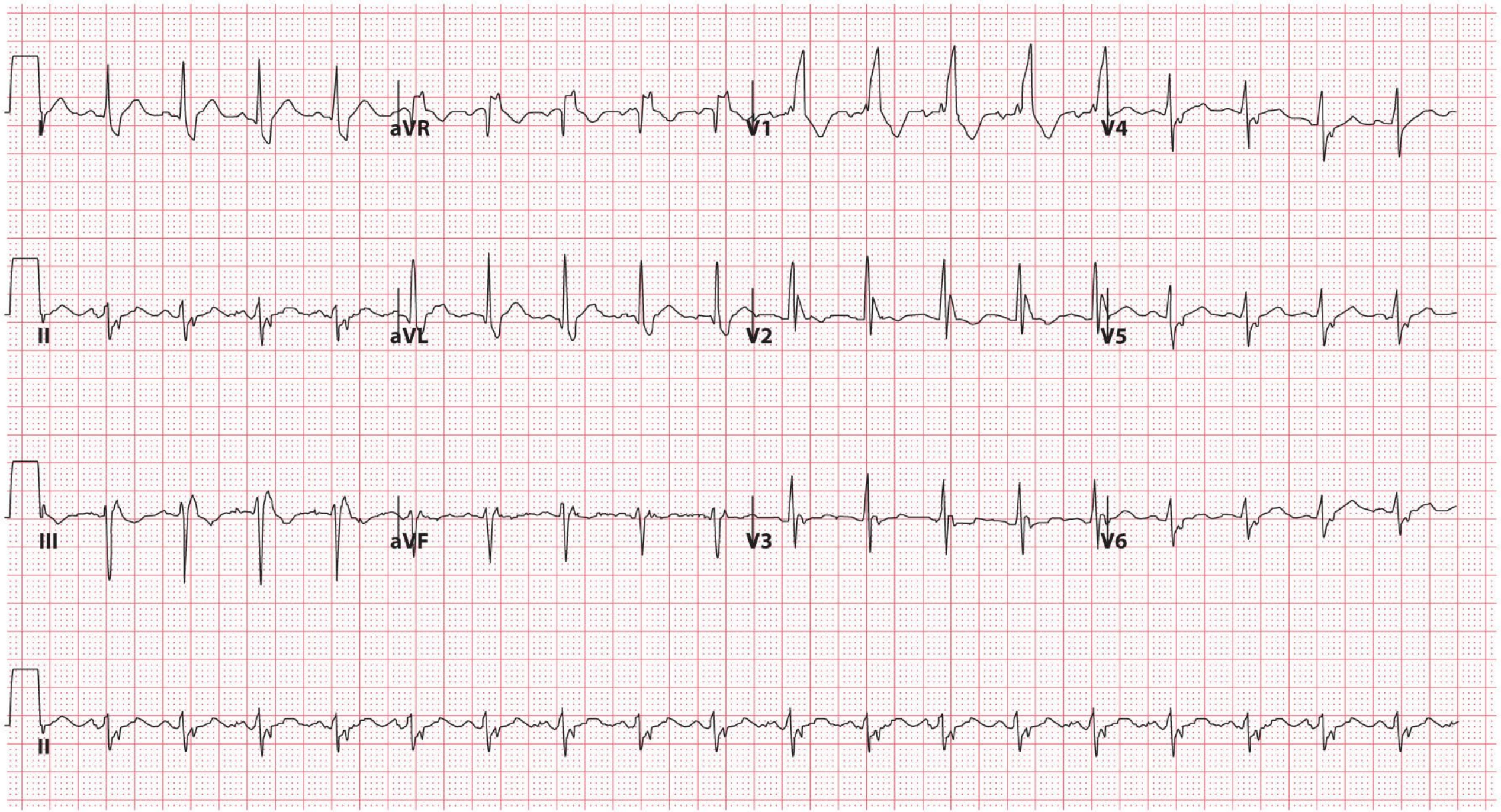

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Table 2. High-risk electrocardiogram features

Feature	Description
Bradyarrhythmia	
Sinus node dysfunction	Asymptomatic inappropriate sinus rate < 50 bpm or slow AF (40-50 bpm), sinus block, sinus pause > 3 seconds in the absence of negatively chronotropic medications
Conduction disease	Bifascicular block
	Intraventricular conduction delay (QRS 120 ms)
	Second-degree AV block type 1 with prolonged PR interval
	Second-degree AV block type 2
	Third-degree AV block
Tachyarrhythmia	
Supraventricular	Ventricular pre-excitation
	Supraventricular tachycardia or AF
Ventricular tachycardia	Nonsustained ventricular tachycardia
	Evidence of acute ischemia or previous myocardial infarction
	Long (> 460 ms) QT on repetitive ECGs or short (< 340 ms) QT interval
	Type 1 Brugada
	Brugada pattern (RBBB with ST elevation V_1-V_3)
	Arrhythmogenic right ventricular cardiomyopathy features (negative T waves in right precordial leads, epsilon wave, ventricular late potentials)
	Ventricular hypertrophy

AF, atrial fibrillation; AV, atrioventricular; bpm, beats per minute; ECG, electrocardiogram; RBBB, right bundle branch block.

Empiric pacemaker compared with a monitoring strategy in patients with syncope and bifascicular conduction block-rationale and design of the Syncope: Pacing or Recording in ThE Later Years (SPRITELY) study

Bifascicular block:
RBBB+LAFB
RBBB+1st degree
True LBBB
Etc..

Syncope + bifascicular block on ECG
Age > 50

Pacemaker

Implantable Monitor

Endpoint: composite of syncope, symptomatic bradycardia, symptomatic complete heart block, acute and chronic complications of pacemaker and monitor, and death. 2 years follow-up

Krahn, Sheldon et al.. unpublished

Late Breakers – Heart Rhythm Society Meeting

BOSTON -- For older syncope patients found to have bifascicular block, heading straight to pacing without watching for actionable findings with an implantable loop recorder appears to give better outcomes, the pragmatic SPRITELY (POST 3) trial showed.

An empiric pacemaker-first strategy lowered the combined rate of syncope, symptomatic or asymptomatic bradycardia, acute or chronic device complications, or cardiovascular death compared with an implantable cardiac monitoring strategy (19 of 57 versus 44 of 58, $P<0.001$).

The higher rate in the loop recorder group was driven by bradycardia events (28 vs 0, p<0.001), and not by syncope (loop- 14 vs 13 in pacemaker p=0.87).

Risk Stratification and Disposition from the Emergency Department

SYNCOPAL EVENT
Low-risk
• Associated with prodrome typical of reflex syncope (e.g. light-headedness, feeling of warmth, sweating, nausea, vomiting)[36,49] • After sudden unexpected unpleasant sight, sound, smell, or pain[36,49,50] • After prolonged standing or crowded, hot places[36] • During a meal or postprandial[51] • Triggered by cough, defaecation, or micturition[52] • With head rotation or pressure on carotid sinus (e.g. tumour, shaving, tight collars)[53] • Standing from supine/sitting position[54]
High-risk
Major
• New onset of chest discomfort, breathlessness, abdominal pain, or headache[26, 44, 55] • Syncope during exertion or when supine[36] • Sudden onset palpitation immediately followed by syncope[36]
Minor (high-risk only if associated with structural heart disease or abnormal ECG):
• No warning symptoms or short (<10 s) prodrome[36, 38, 49, 56] • Family history of SCD at young age[57] • Syncope in the sitting position[54]

PAST MEDICAL HISTORY
Low-risk
• Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode[58] • Absence of structural heart disease[27, 58]
High-risk
Major
• Severe structural or coronary artery disease (heart failure, low LVEF or previous myocardial infarction)[26, 27, 35, 55, 59]

Category	Points
Clinical evaluation	
Predisposition to vasovagal symptoms*	–1
History of heart disease†	1
Any systolic pressure reading < 90 or > 180 mm Hg‡	2
Investigations	
Elevated troponin level (> 99th percentile of normal population)	2
Abnormal QRS axis (< –30° or > 100°)	1
QRS duration > 130 ms	1
Corrected QT interval > 480 ms	2
Diagnosis in emergency department	
Vasovagal syncope	–2
Cardiac syncope	2
Total score (–3 to 11)	____

*Triggered by being in a warm crowded place, prolonged standing, fear, emotion or pain
†Includes coronary or valvular heart disease, cardiomyopathy, congestive heart failure and non-sinus rhythm (electrocardiogram evidence during index visit or documented history of ventricular or atrial arrhythmias, or device implantation)
‡Includes blood pressure values from triage until disposition from the emergency department

Total score	Estimated risk of serious adverse event,§ %	Risk category
–3	0.4	Very Low
–2	0.7	Very Low
–1	1.2	Low
0	1.9	Low
1	3.1	Medium
2	5.1	Medium
3	8.1	Medium
4	12.9	High
5	19.7	High
6	28.9	Very High
7	40.3	Very High
8	52.8	Very High
9	65.0	Very High
10	75.5	Very High
11	83.6	Very High

§ Shrinkage-adjusted expected risk

Figure 2. Canadian Syncope Risk Score.

Investigations

Tailoring Cardiac Monitor Selection to Symptom Frequency

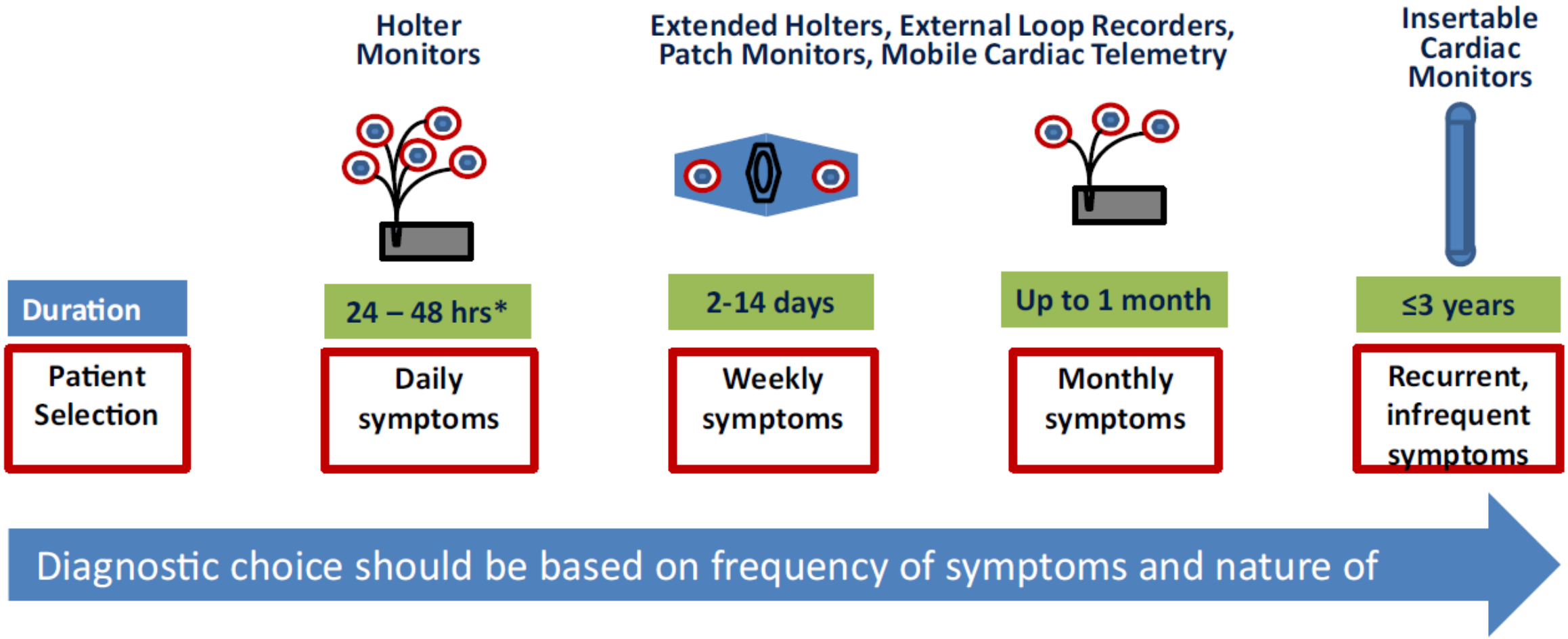
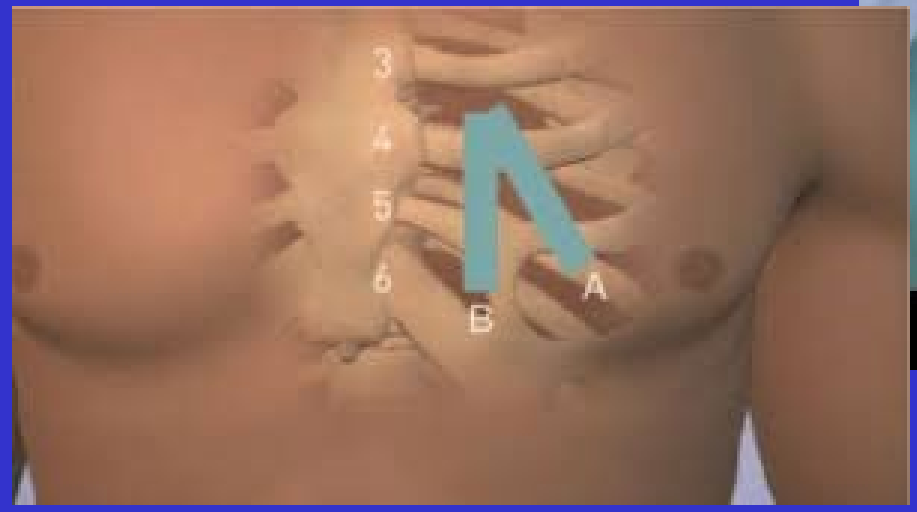


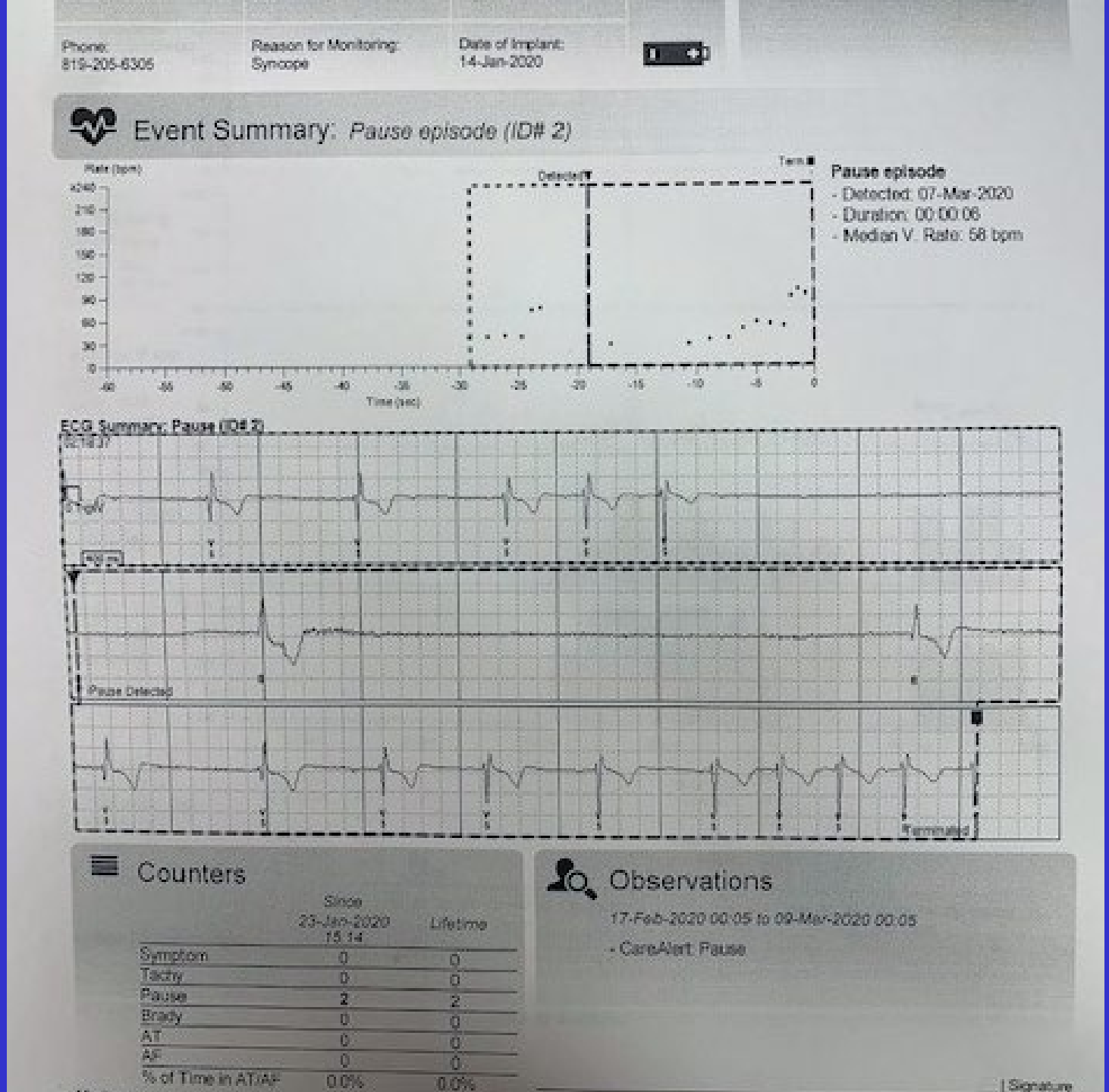
Figure 4. Selection of cardiac monitors for evaluation of suspected arrhythmic syncope.


3
4
5
6
B
A

70M presents after having repeated episodes of brief syncope

Echo normal

ECG normal



Arrhythmia Episodes

SYMPTOM | Tachy | Pause | Brady | AT | AF | Sorted by Date/Time

#	Type	Date	Time hh:mm	Duration hh:mm:ss	Max V. Rate	Median V. Rate	Detai
2	Pause	07-Mar-2020	02:18	:06		58 bpm (1030 ms)	ECG
1	Pause	20-Feb-2020	11:59	:05		52 bpm (1160 ms)	ECG

---------- Last Medtronic CareLink Monitor Session 23-Jan-2020 ----------

---------- Last Programmer Session 20-Jan-2020 ----------

(Data prior to last session has not been interrogated.)

#1: Plot ECG Text Previous Next

ECG Reveal

(0.1 mV)

Markers

Interval (ms)

>2000 2080 1170 2200 2100 4400 1700 2100 1700 2300 1800 7600

Pause Detected Terminated

Print... Close

Interrogate... End Session...

Tilt Table Testing

Should only be considered if there is diagnostic uncertainty:

-Older patients with few clues in the history
-Distinguishing convulsive syncope from epilepsy
-Nonhemodynamic collapse investigation

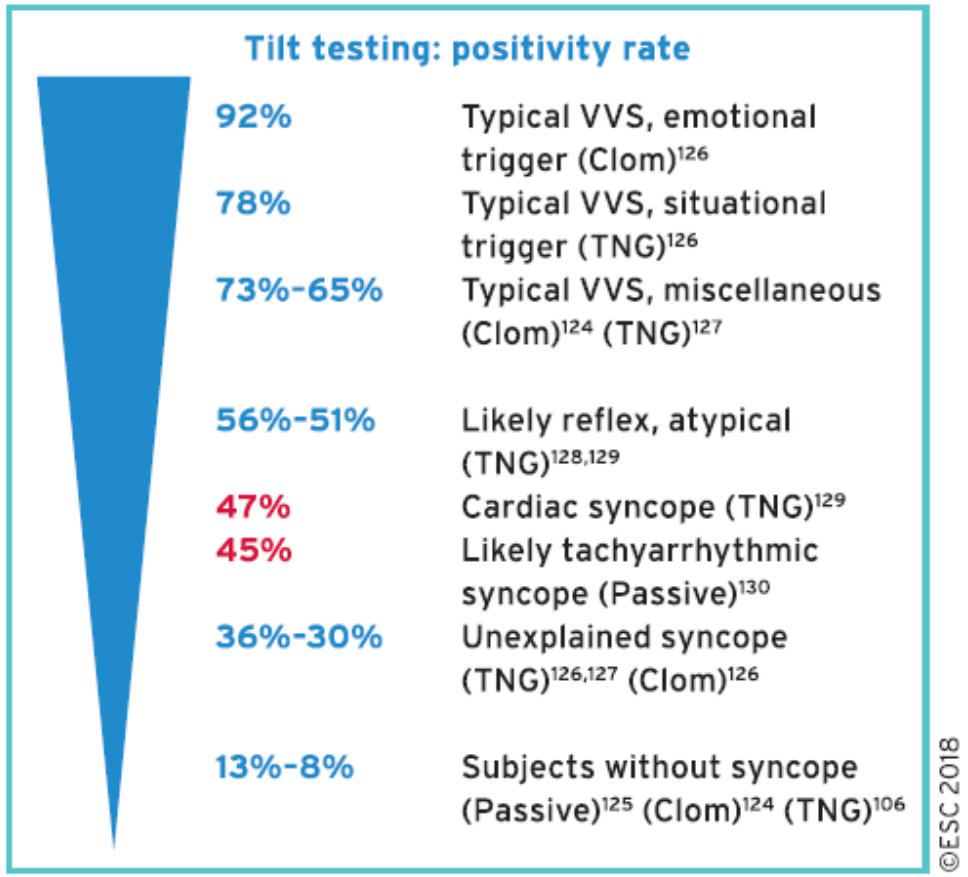
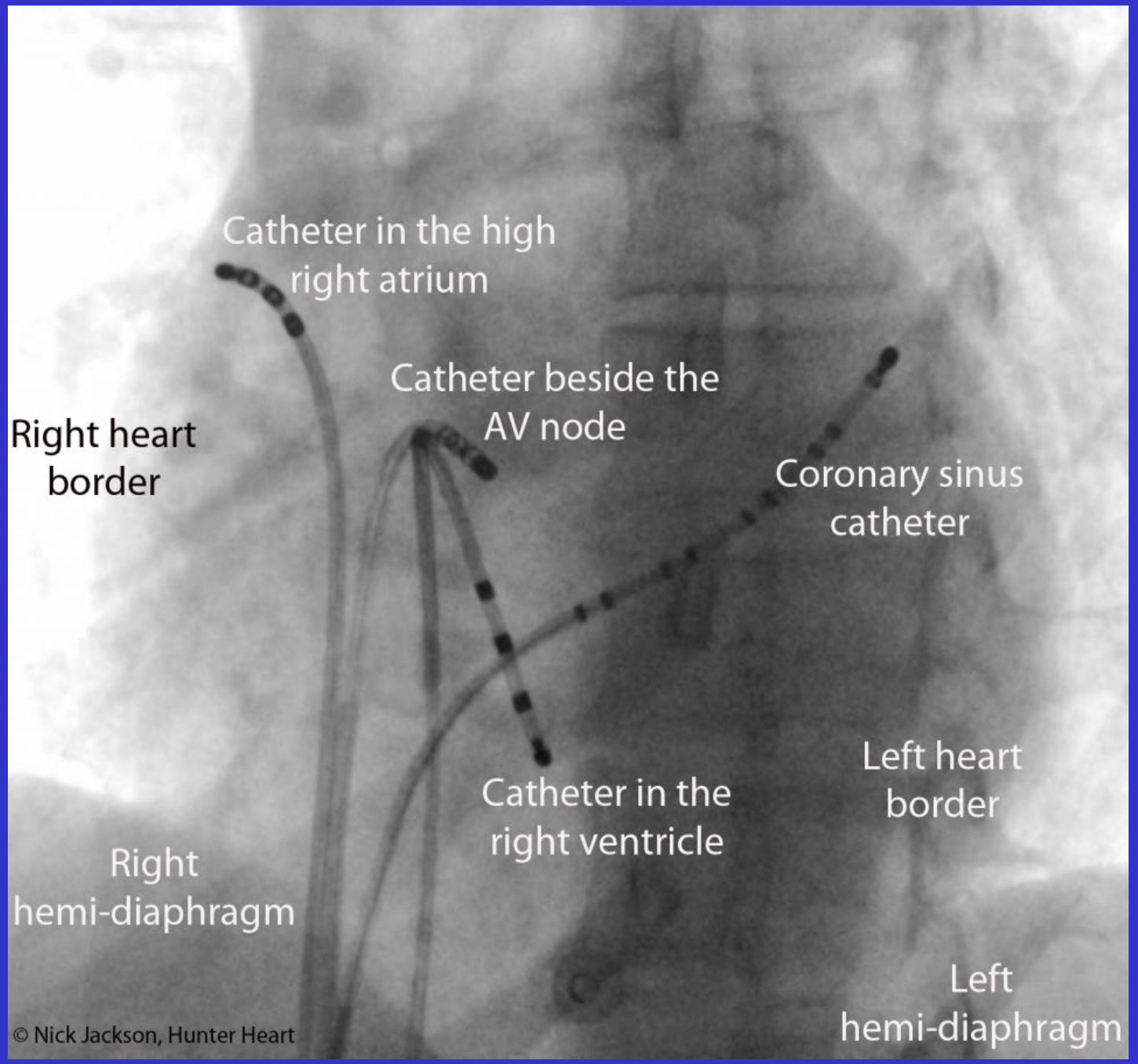


Figure 7 Rates of tilt testing positivity in different clinical conditions. These studies used the Westminster protocol for passive tilt,[125] the Italian protocol for trinitroglycerin tilt,[106] and the clomipramine protocol,[124] for a total of 1453 syncope patients and 407 controls without syncope. Studies using other tilt protocols, e.g. isoproterenol challenge, were not included. Clom = clomipramine; TNG = trinitroglycerin; VVS = vasovagal syncope.

Electrophysiology Study

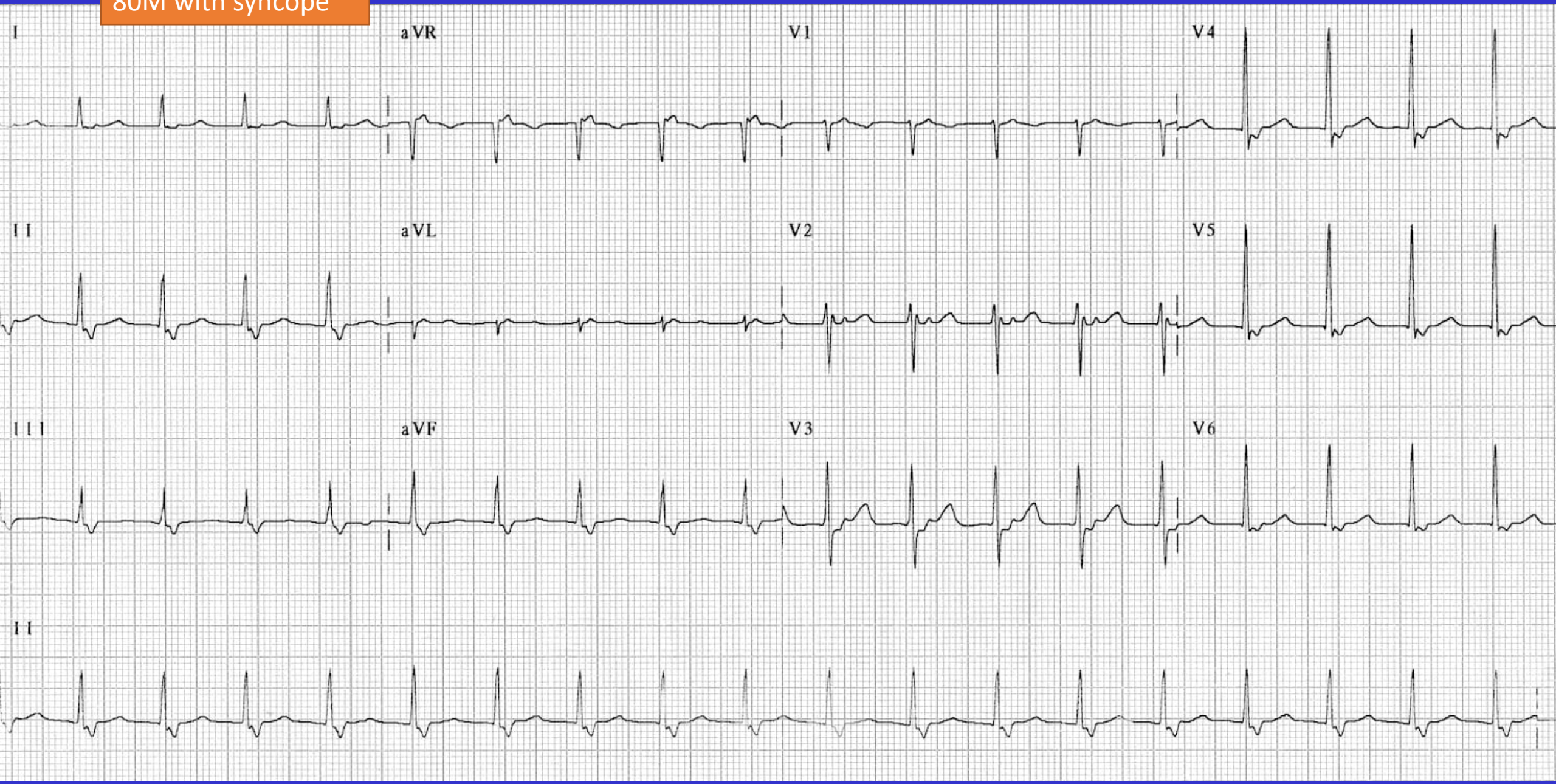
Should be limited to those with a suspected arrhythmic cause or abnormal ECG or structural heart disease after noninvasive testing.

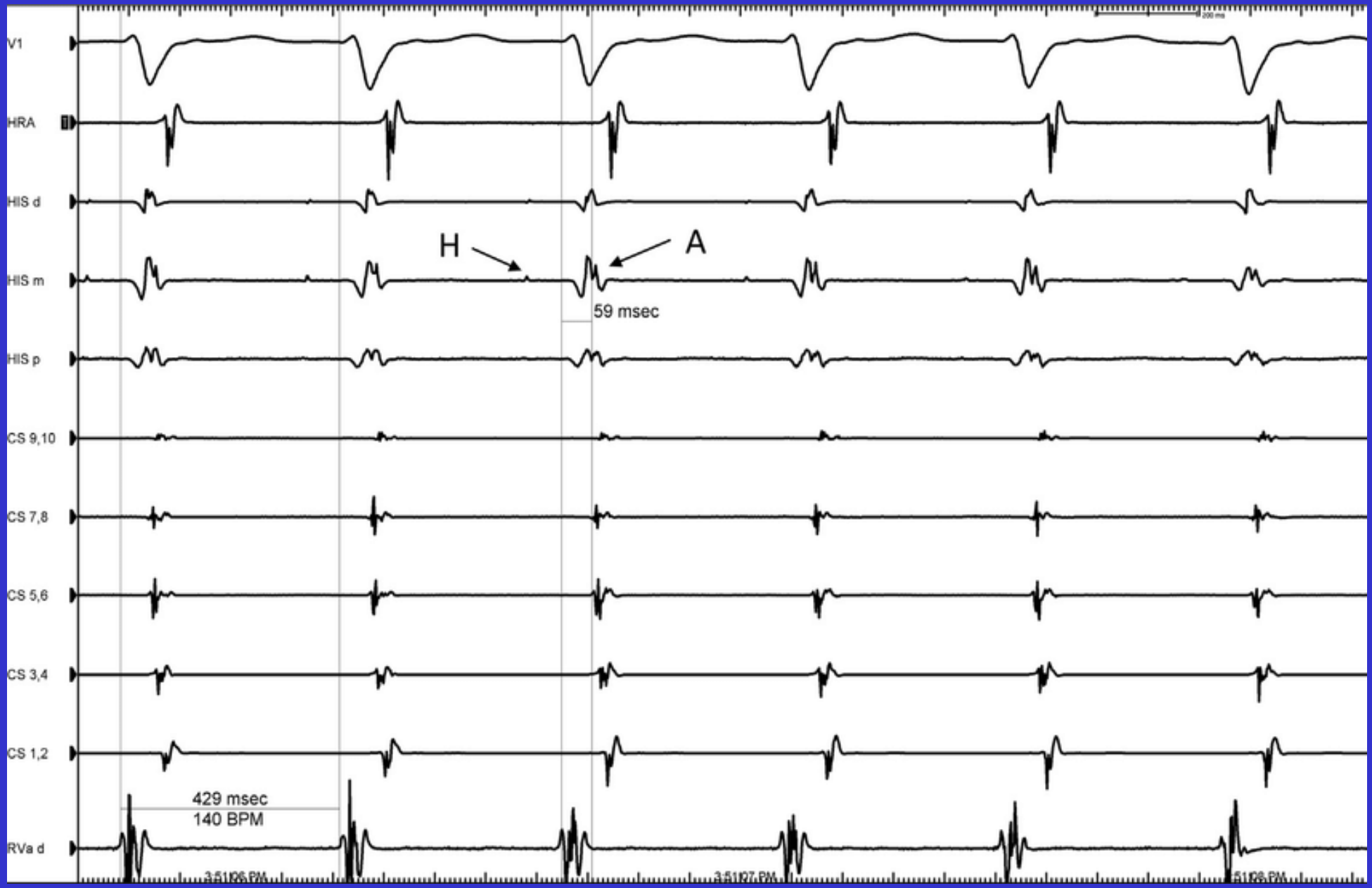


Clinical presentation	EP study	Implications
Sinus bradycardia/ Pauses	Corrected sinus node recovery time (CSNRT) >526 msec	Pacemaker
Bundle branch block with prolonged PR interval	HV interval 100 msec or more	Pacemaker
Ventricular ectopy with LV dysfunction	Inducible sustained monomorphic VT	ICD
Palpitations preceding syncope	Inducible SVT/ RVOT-VT/ Fascicular VT	Radiofrequency ablation

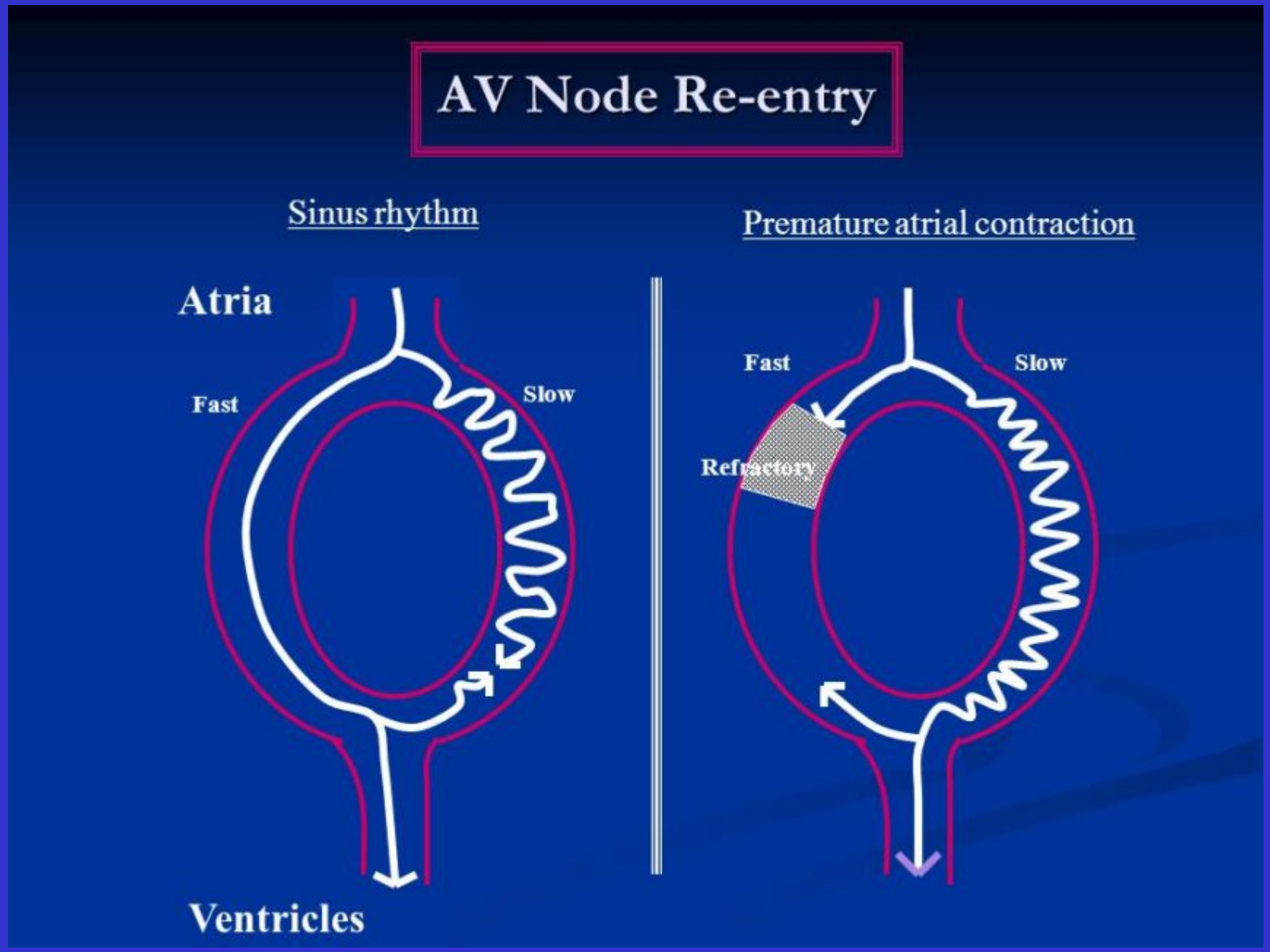
Abbreviations: EP, electrophysiological; ICD, implantable cardioverter defibrillator; LV, left ventricle; RVOT, right ventricular outflow tract; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

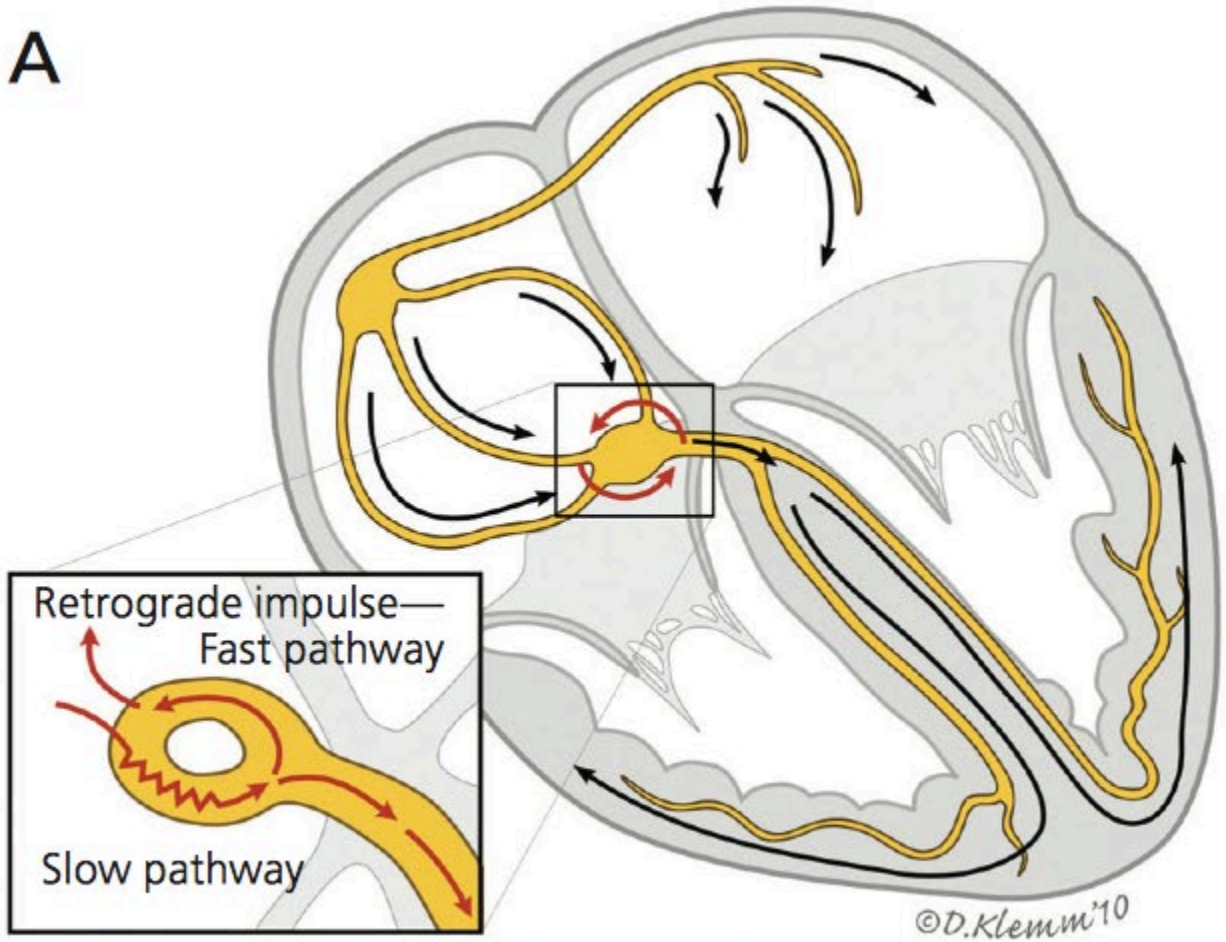
80M with syncope

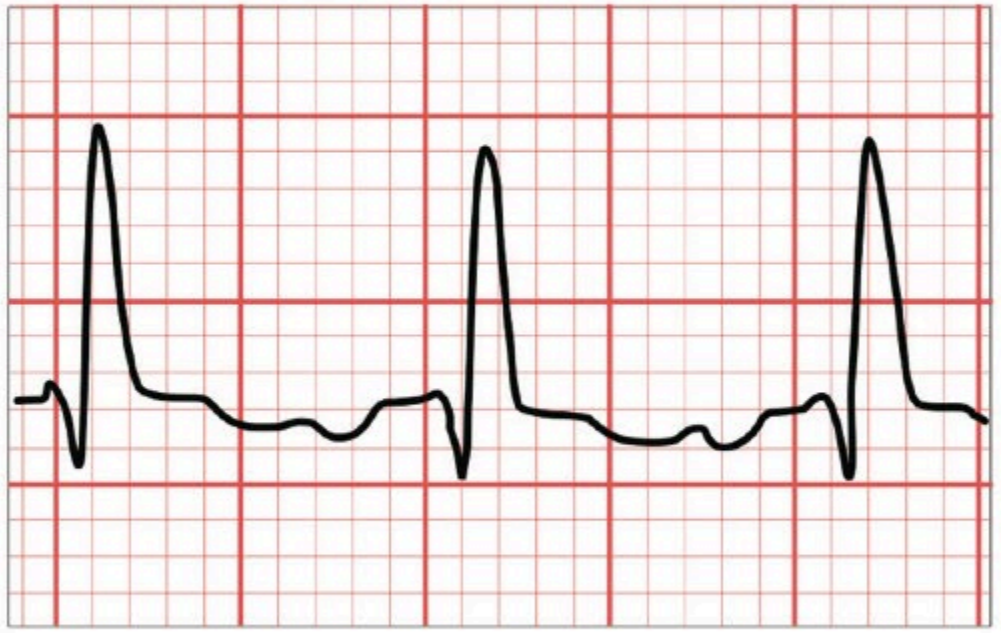



V1
HRA
HIS d
H
A
HIS m
59 msec
HIS p
CS 9,10
CS 7,8
CS 5,6
CS 3,4
CS 1,2
429 msec
140 BPM
RVa d

AV Node Re-entry



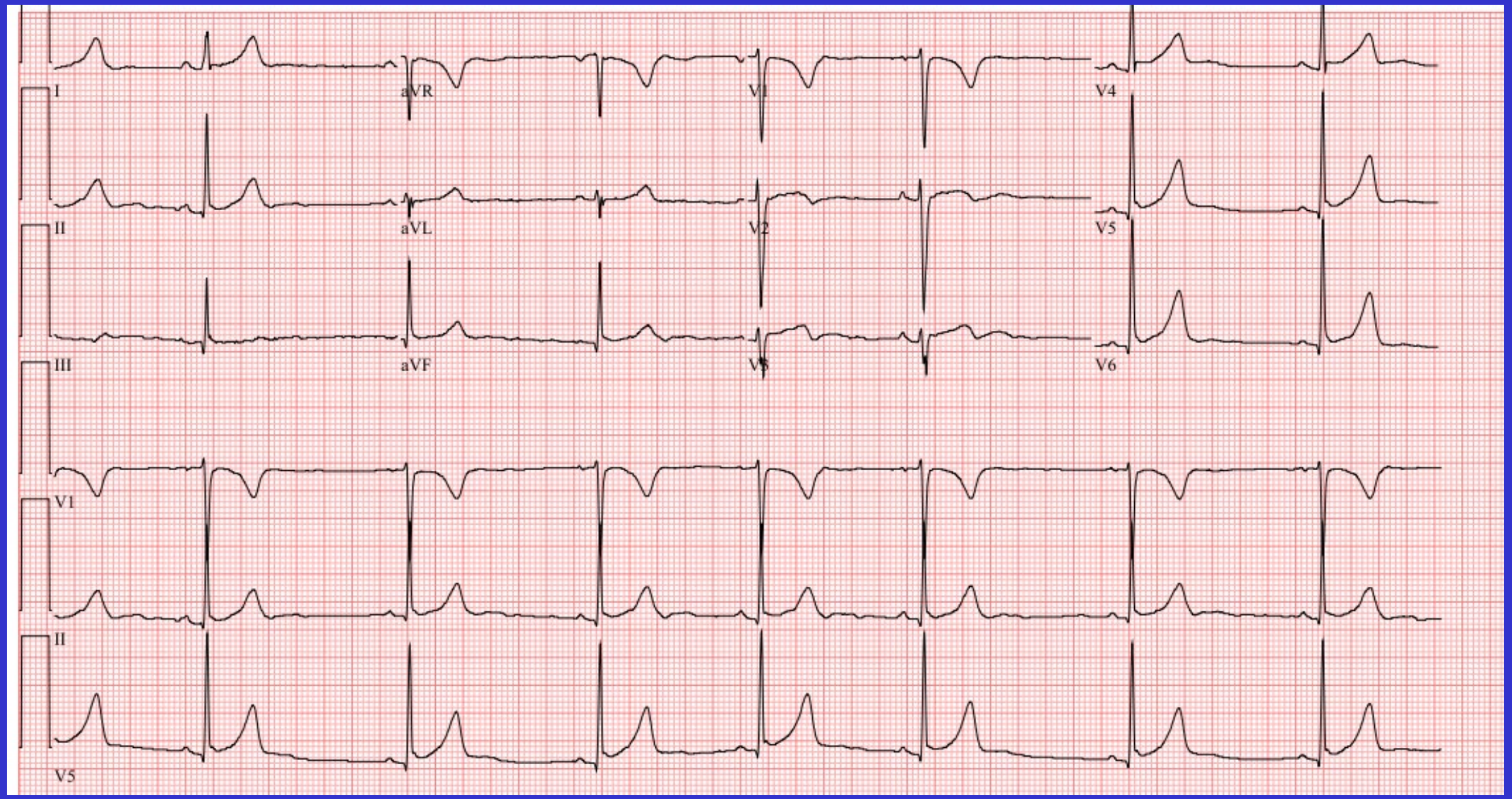




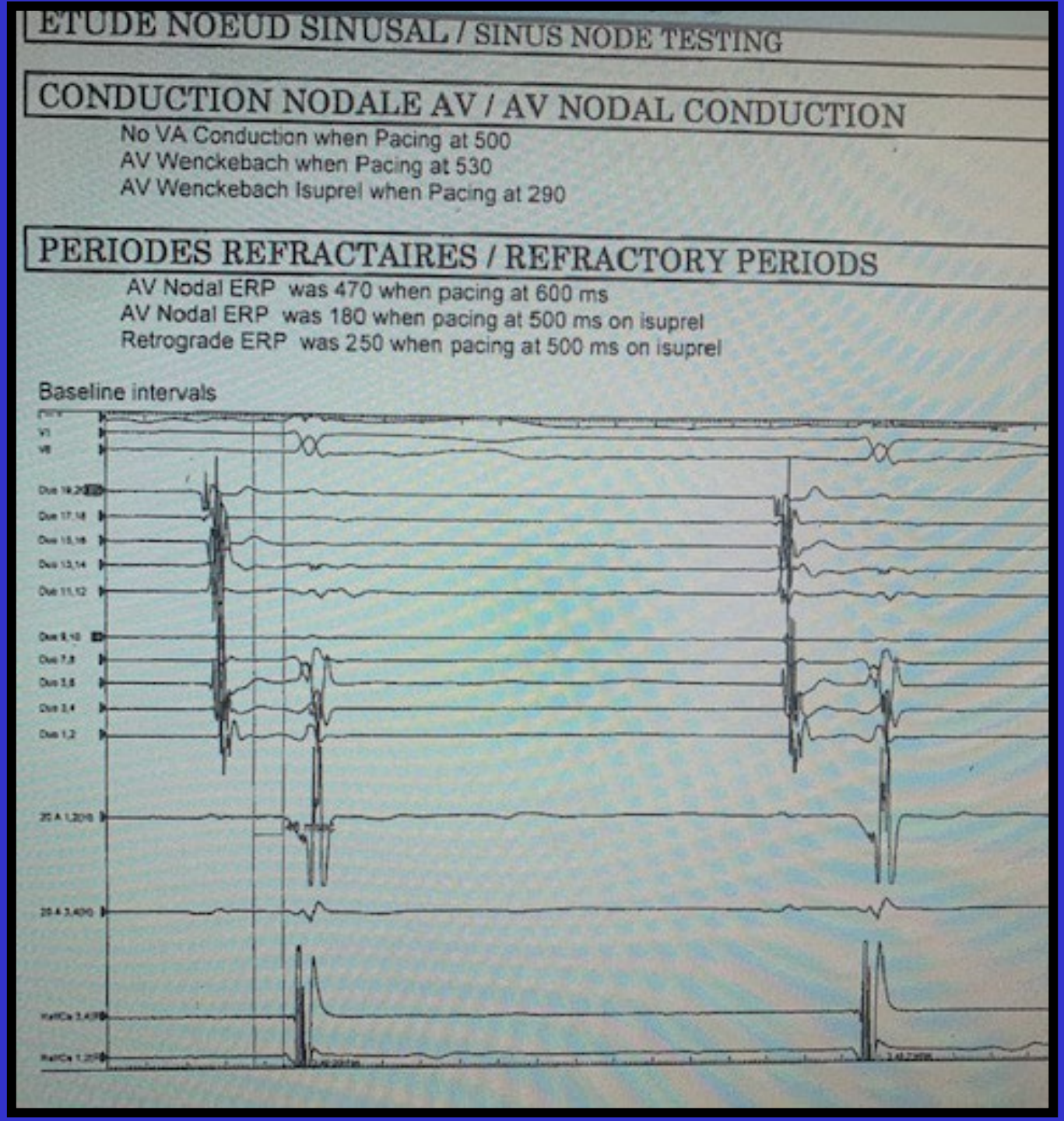
Atrioventricular nodal reentry


when not used.
418120
EP-LAB
9
IntelliVue
Not Admitted
23 Oct 2018 12:15
Profiles
Tachy 245 > 180
245
1mV
Pleth

21 year-old F presenting to hospital with an episode of syncope 15 minutes after exercise. + troponin. Normal coronary arteries. Echo: normal. MRI: normal.



Referred for: EP study +-



ETUDE NOEUD SINUSAL / SINUS NODE TESTING

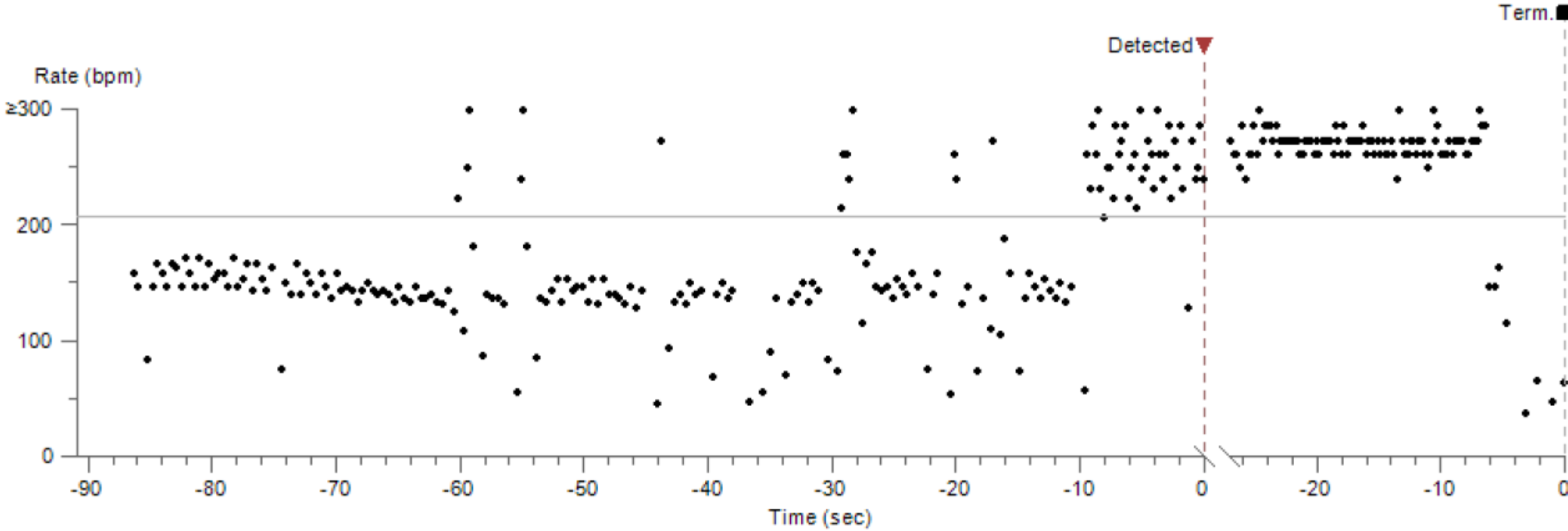
CONDUCTION NODALE AV / AV NODAL CONDUCTION

No VA Conduction when Pacing at 500
AV Wenckebach when Pacing at 530
AV Wenckebach Isuprel when Pacing at 290

PERIODES REFRACTAIRES / REFRACTORY PERIODS

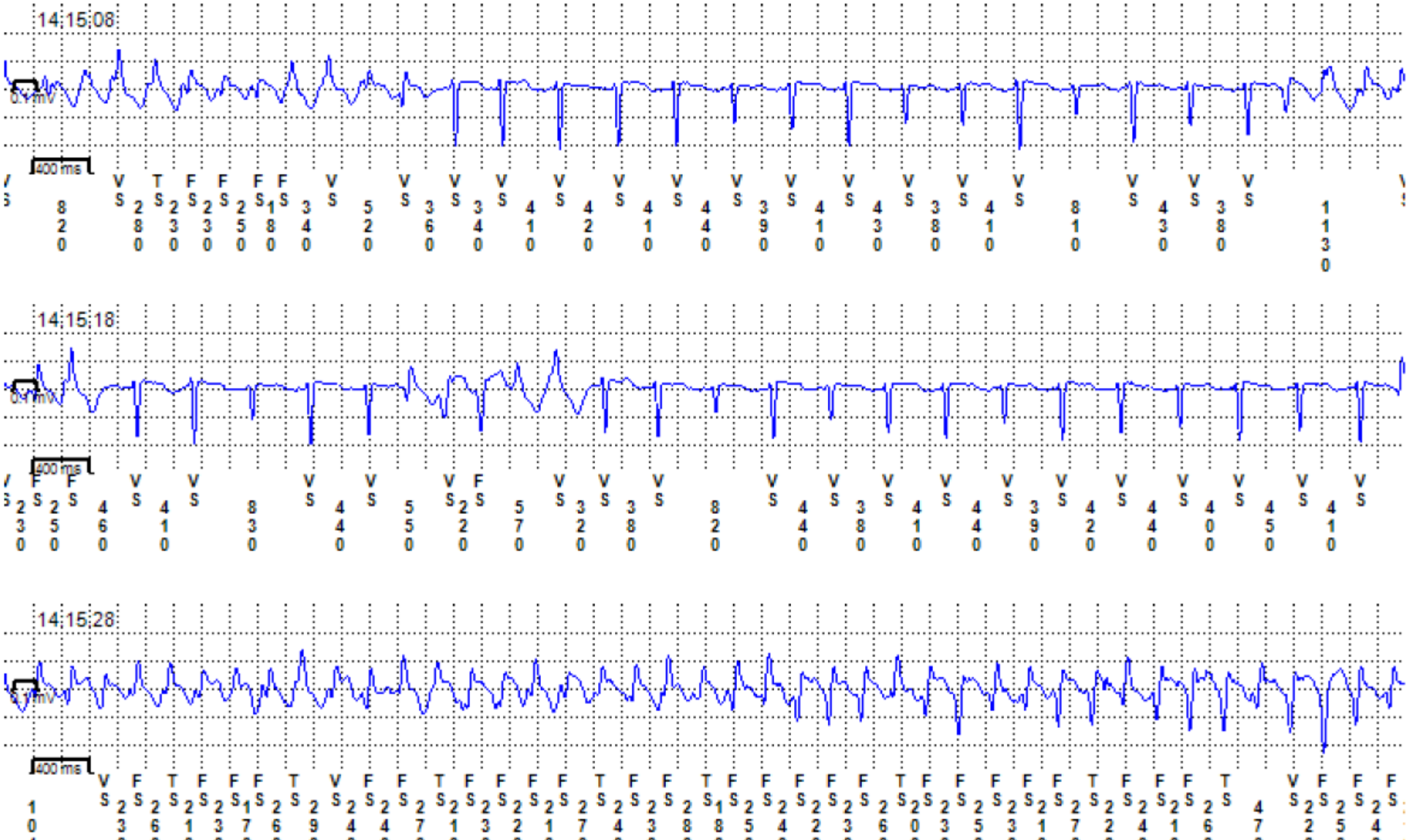
AV Nodal ERP was 470 when pacing at 600 ms
AV Nodal ERP was 180 when pacing at 500 ms on isuprel
Retrograde ERP was 250 when pacing at 500 ms on isuprel

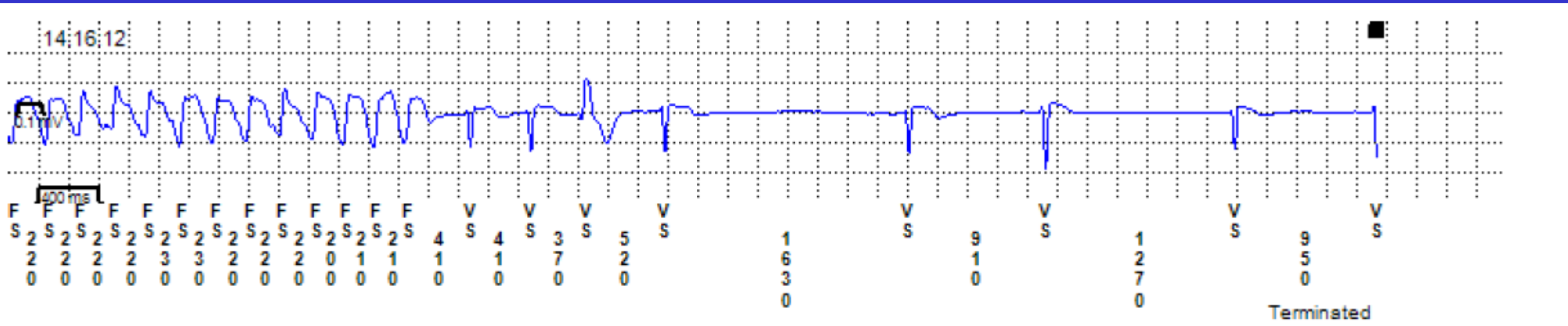
Baseline intervals



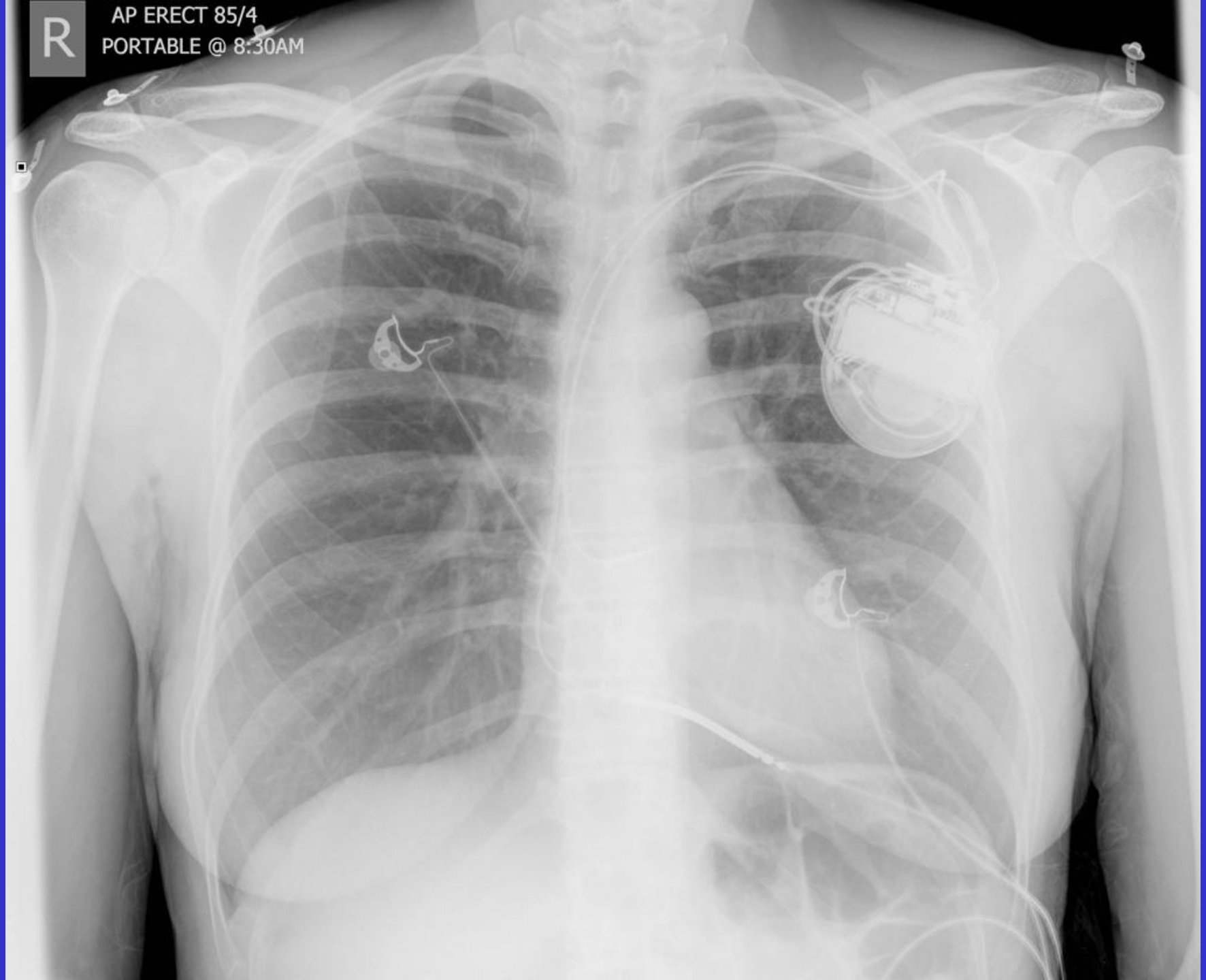
Assessment Legend: Appropriate Indeterminate Inappropriate

ID#	Assessment	Type	Date	Detected hh:mm	Duration hh:mm:ss	Max V. Rate	Median V. Rate
63		Tachy	10-Jul-2020	14:15	00:01:06	273 bpm	250 bpm


14:15:08
0.1 mV
400 ms
14:15:18
0.1 mV
400 ms
14:15:28
0.1 mV
400 ms



Plan: ICD implant, medication (beta blocker)


R
AP ERECT 85/4
PORTABLE @ 8:30AM

Treatment

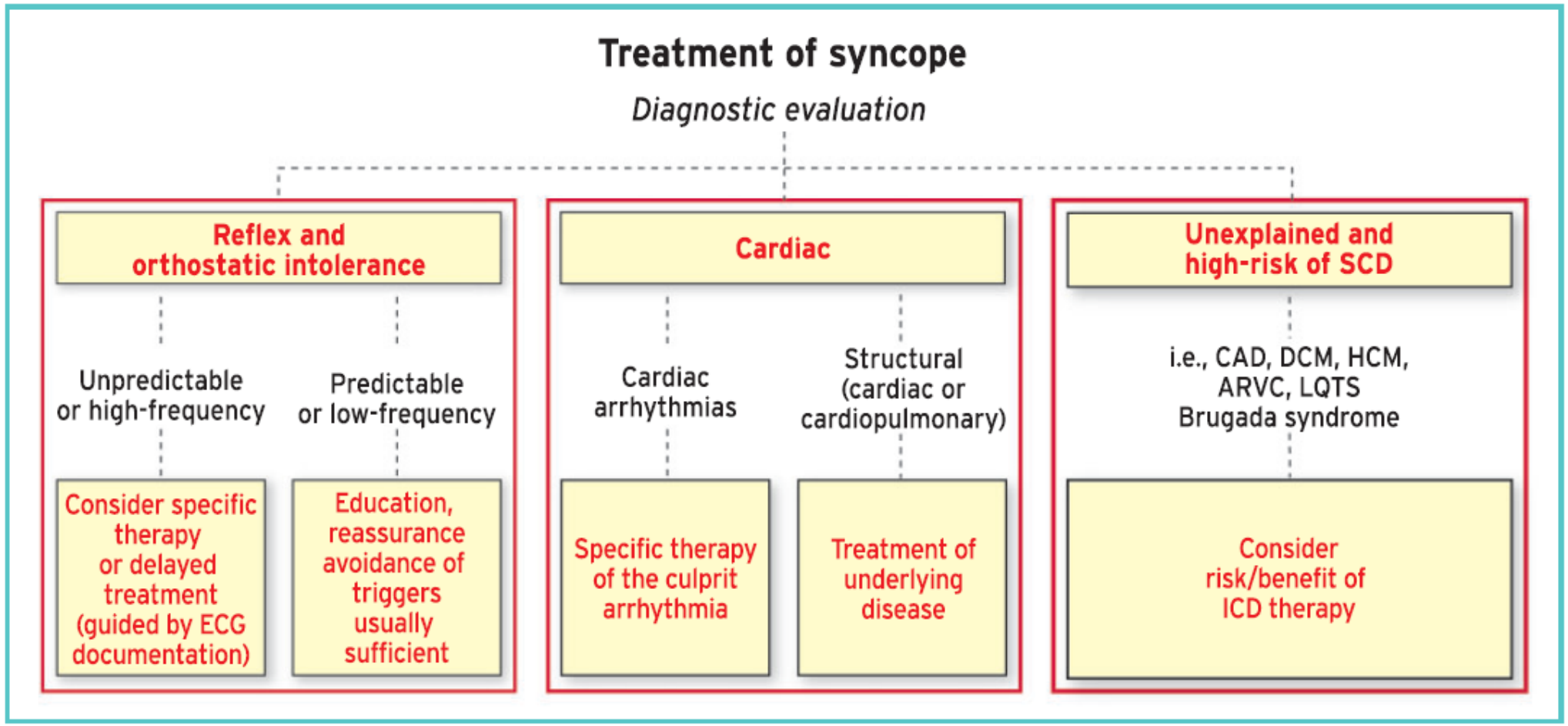
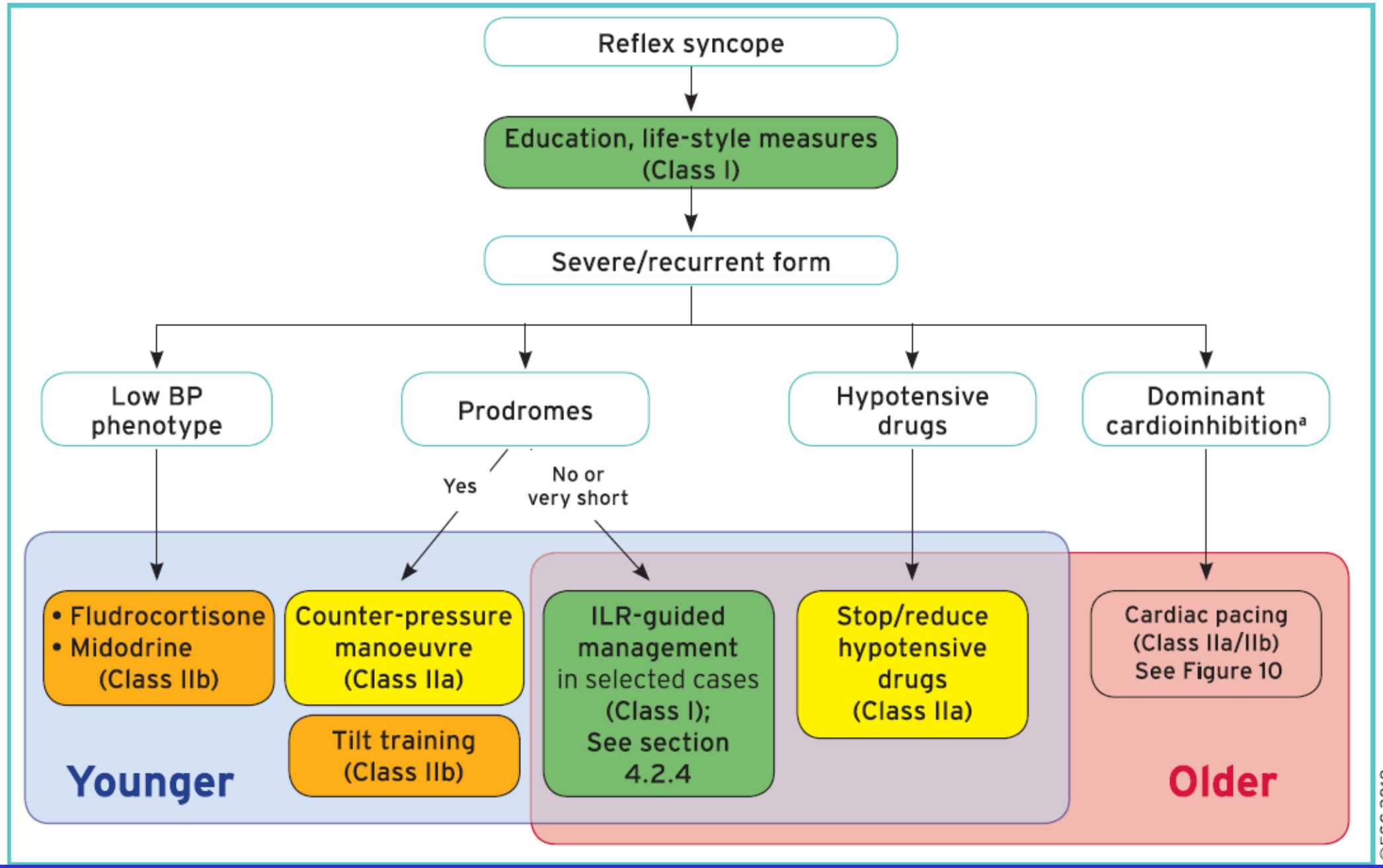
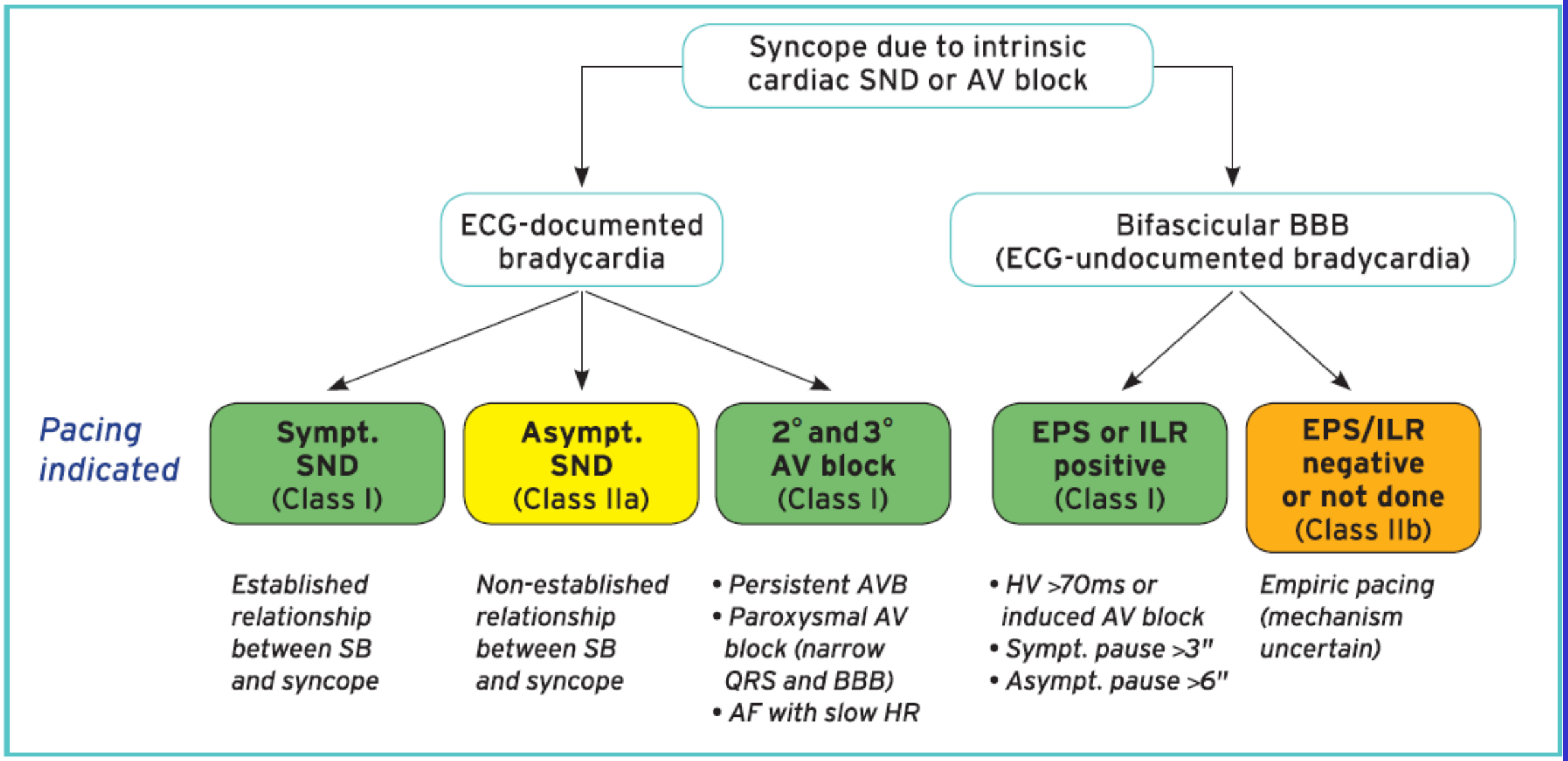


Figure 8 General framework of treatment is based on risk stratification and the identification of specific mechanisms when possible. ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; DCM = dilated cardiomyopathy; ECG = electrocardiographic; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter defibrillator; LQTS = long QT syndrome; SCD = sudden cardiac death.


Reflex syncope
Education, life-style measures (Class I)
Severe/recurrent form
Low BP phenotype
Prodromes
Hypotensive drugs
Dominant cardioinhibition[a]
Yes
No or very short
• Fludrocortisone
• Midodrine
(Class IIb)
Counter-pressure manoeuvre (Class IIa)
Tilt training (Class IIb)
ILR-guided management in selected cases (Class I); See section 4.2.4
Stop/reduce hypotensive drugs (Class IIa)
Cardiac pacing (Class IIa/IIb) See Figure 10
Younger
Older


Syncope due to intrinsic cardiac SND or AV block
ECG-documented bradycardia
Bifascicular BBB (ECG-undocumented bradycardia)
Pacing indicated
Sympt. SND (Class I)
Asympt. SND (Class IIa)
2° and 3° AV block (Class I)
EPS or ILR positive (Class I)
EPS/ILR negative or not done (Class IIb)
Established relationship between SB and syncope
Non-established relationship between SB and syncope
• Persistent AVB
• Paroxysmal AV block (narrow QRS and BBB)
• AF with slow HR
• HV >70ms or induced AV block
• Sympt. pause >3"
• Asympt. pause >6"
Empiric pacing (mechanism uncertain)

Conclusion

1. Three different type of syncope include: reflex syncope, orthostatic hypotension, and true cardiac syncope

2. Use the high and low risk features based on history, physical exam, and ECG to determine need to act quickly

3. Apply therapies based on the most likely diagnosis

References

1. European Heart Journal (2018) 39, 1883–1948
2. Canadian Journal of Cardiology 36 (2020) 1167e117
3. Krahn et al. Europace 2012
4. Sheldon et al. Annals Internal Medicine Oct 2021