

Syncope - *2008*

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UTHSCSA and STVHCS

I have no conflicts of interest related to this presentation.

Outline

- Epidemiology
- Etiology
- Assessment
- Management

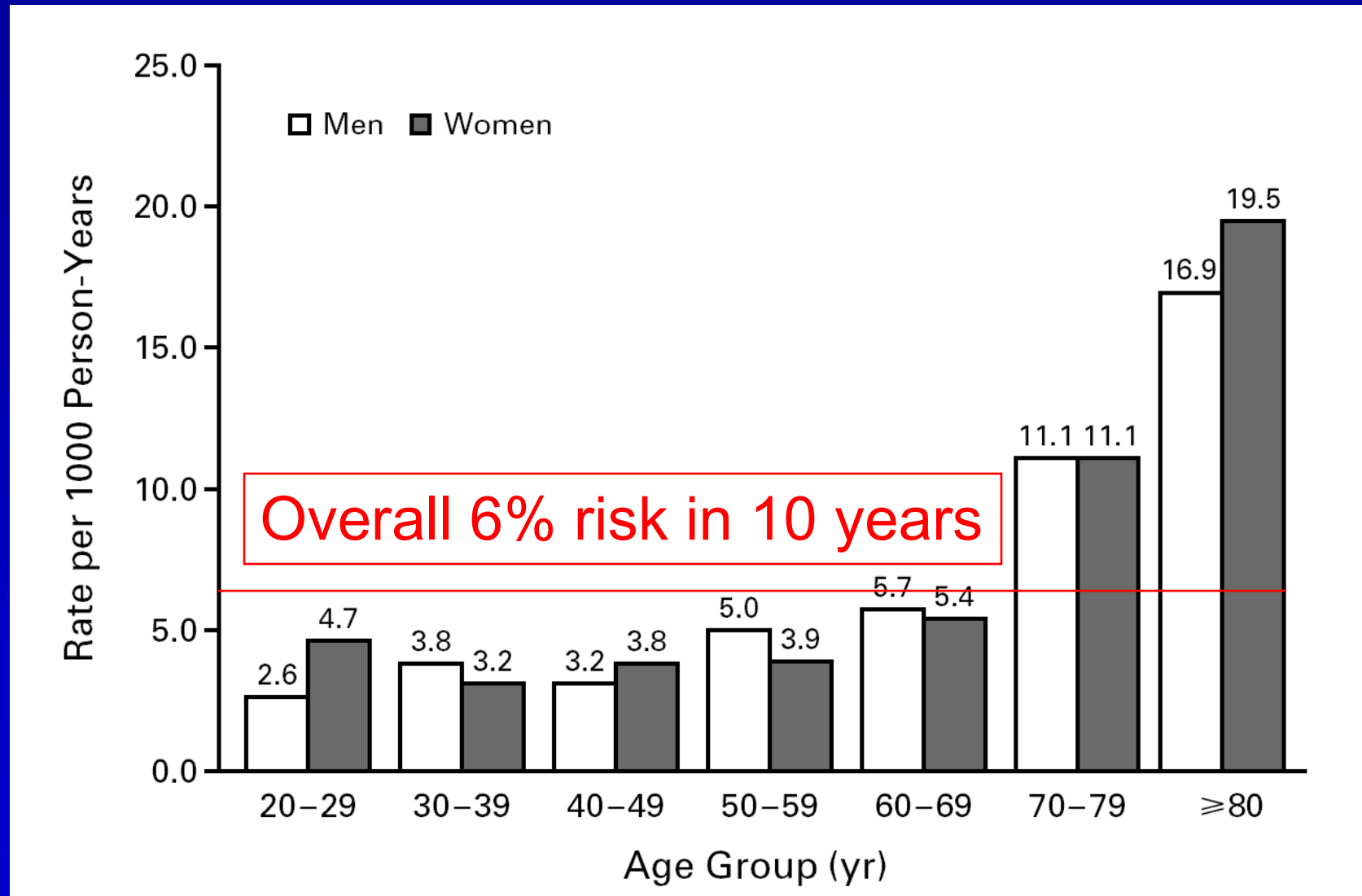
Role of Provider in Management of the Patient with Syncope

- Provide answers to the patient
 - Diagnosis – identification of cause
 - Prognosis – risk of death
- Prevent **death** or disability
- Provide relief of symptoms – improve patient's quality of life

Syncope is a Frequent Complaint

- Definition: Sudden loss of consciousness and inability to maintain postural tone followed by spontaneous recovery
- Syncope accounts for
 - 1-3% of ER visits
 - about 1% of hospital admissions
- Incidence of first episode of syncope is 6 per 1000 person/years (Framingham)
- Prevalence of syncope in persons >45 y.o. is about 15-20% (closer to 20% in women and 15% in men; Mayo Clinic)

Syncope Incidence, Framingham



Syncope: Etiology

- Vascular: orthostatic hypotension or reflex-mediated syncope
- Cardiac: arrhythmia, obstructive disease (valve, HCM, PE)
- Apparent syncope: neurologic causes (seizure, cerebrovascular insufficiency)
- Apparent syncope: metabolic causes

Syncope: Pathophysiology

- Transient (10 sec) cessation of blood flow to reticular activating system of medulla, with spontaneous return

$$BP = C.O. * SVR$$

$$BP = (HR * S.V.) * SVR$$

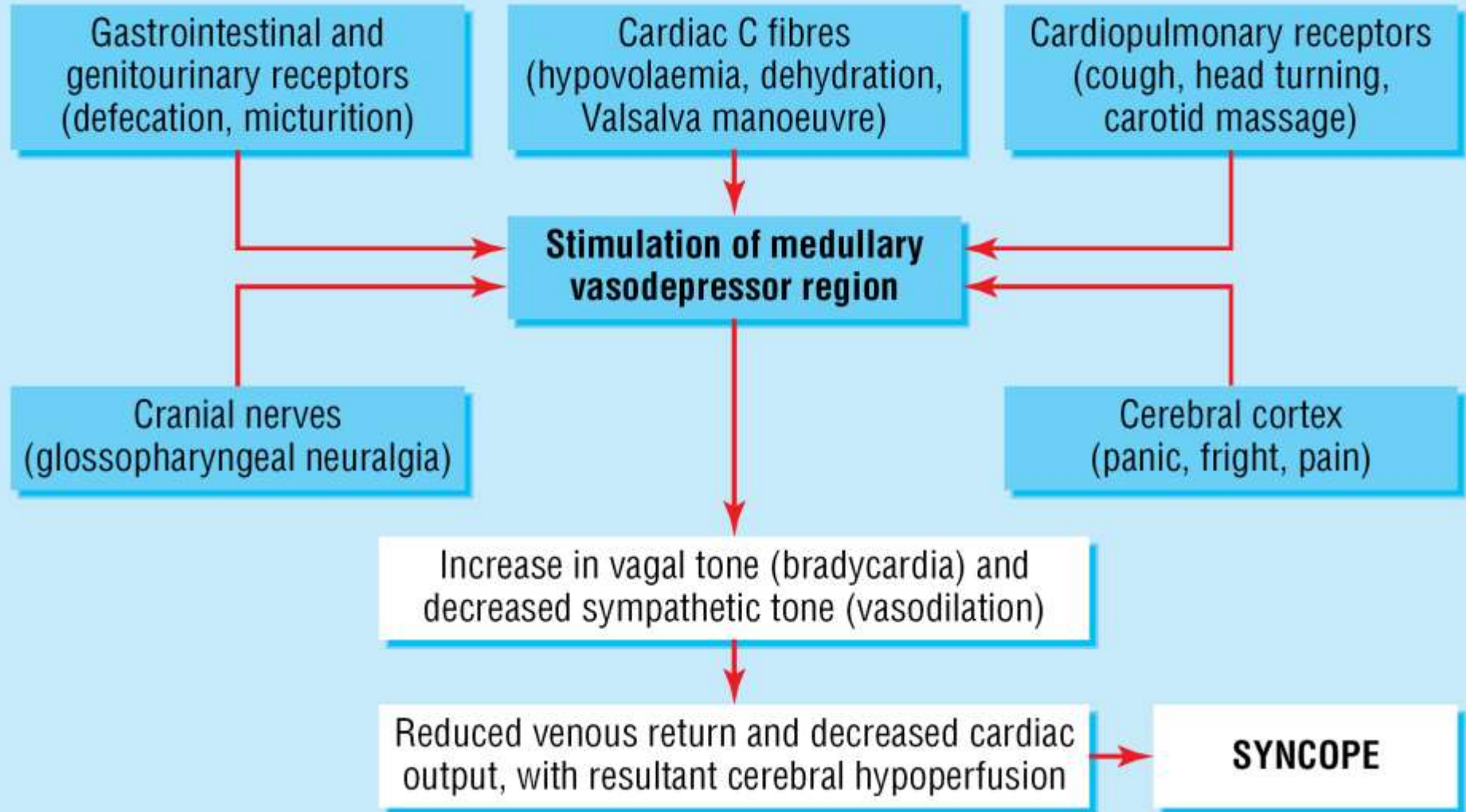
Reflex mediated
Orthostasis

Autonomic insufficiency
Medication, alcohol

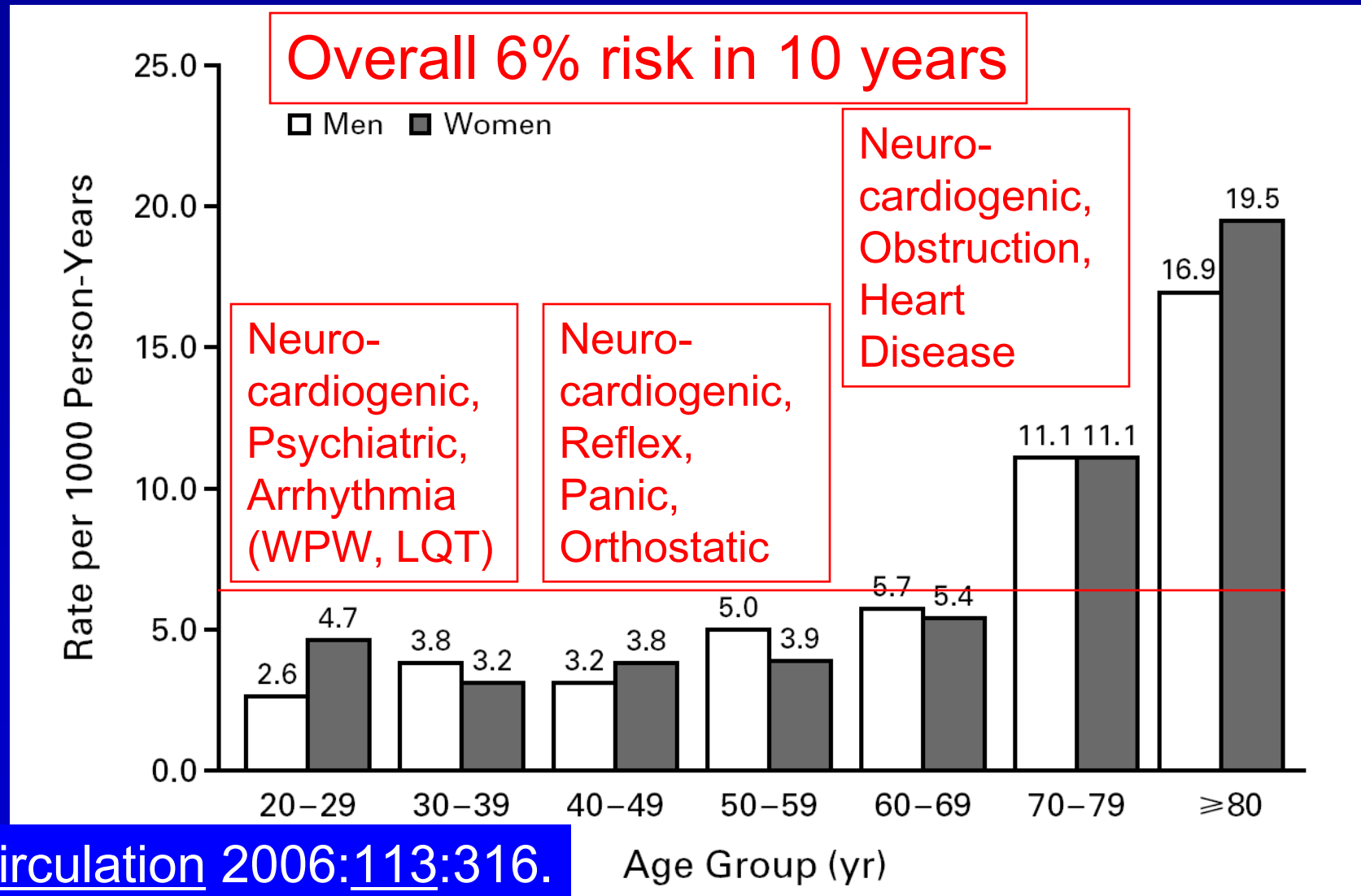
Bradycardia
(sinus or AV
block)

Diastolic volume
Diastolic filling (pulm embolus)
Diastolic filling time (tachycardia)
Systolic function (aortic stenosis, etc.)

Neurocardiogenic Syncope



Syncope Incidence, Framingham



Soteriades ES et al. N Engl J Med. 2002;347:878.

TABLE 1. CAUSES OF SYNCOPE ACCORDING TO SEX AND THE PRESENCE OR ABSENCE OF CARDIOVASCULAR DISEASE AT BASE LINE.

Framingham

Framingham

CAUSE	CARDIOVASCULAR DISEASE ABSENT (N=599)		CARDIOVASCULAR DISEASE PRESENT (N=223)		TOTAL SAMPLE (N=822)
	MEN (N=232)	WOMEN (N=367)	MEN (N=116)	WOMEN (N=107)	
	percent of subjects				
Cardiac	6.5	3.8	26.7	16.8	9.5
Unknown*	31.0	41.7	31.0	37.4	36.6
Stroke or transient ischemic attack	1.7	2.5	9.5	9.4	4.1
Seizure	7.3	3.3	6.9	2.8	4.9
Vasovagal	24.1	24.5	11.2	14.0	21.2
Orthostatic	9.5	10.9	6.9	6.5	9.4
Medication	7.3	6.5	4.3	9.4	6.8
Other†	13.0	6.8	3.5	3.7	7.5

*When a participant did not seek medical attention for syncope and the history, physical examination, and electrocardiographic findings were not consistent with any of the specific causes, the cause was considered to be unknown.

†Cough syncope, micturition syncope, and situational syncope were included in the category of other causes.

Syncopal Prognosis,

Framingham

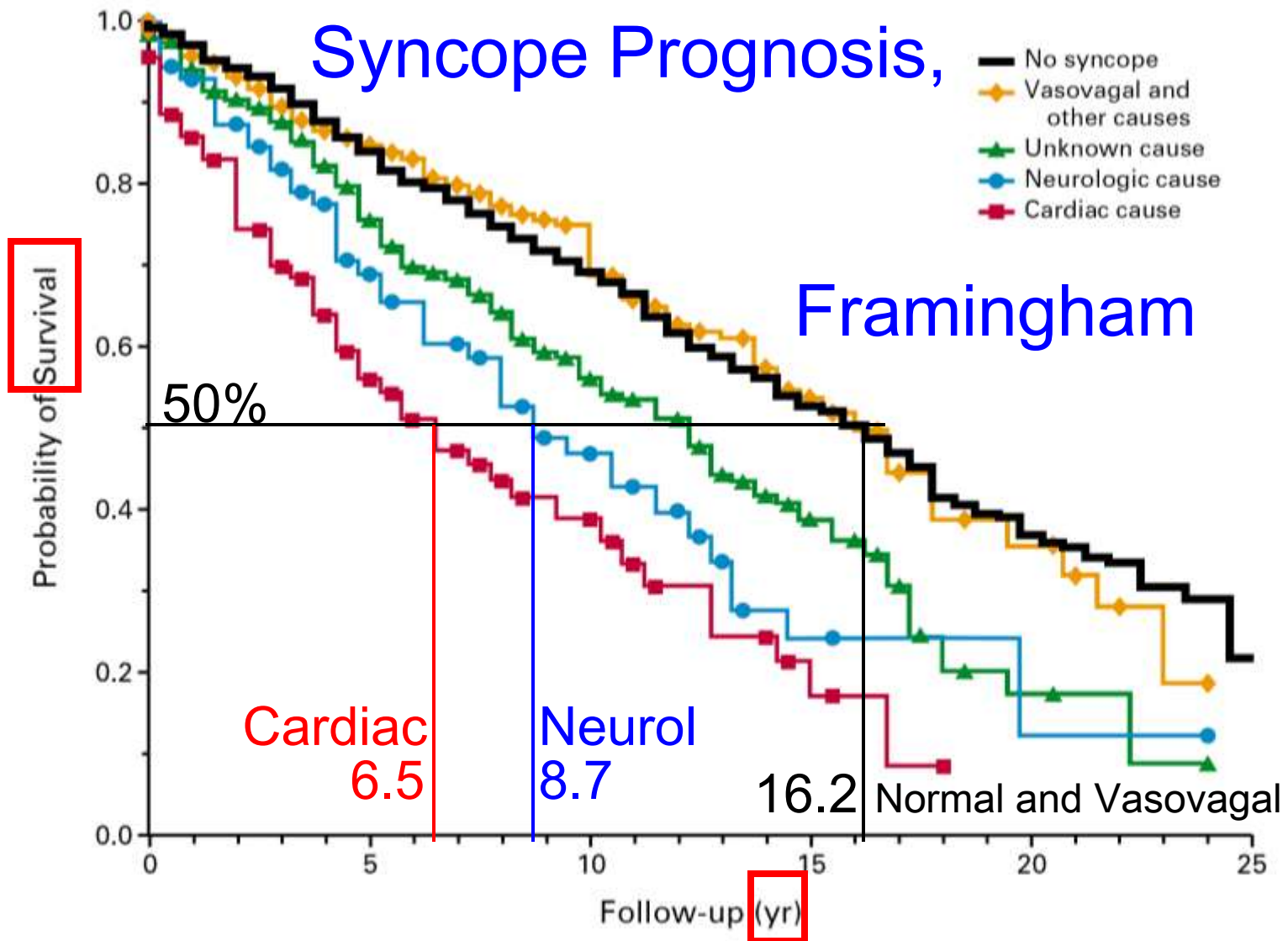


Figure 2. Overall Survival of Participants with Syncope, According to Cause, and Participants without Syncope.

$P < 0.001$ for the comparison between participants with and those without syncope. The category "Vasovagal and other causes" includes vasovagal, orthostatic, medication-induced, and other, infrequent causes of syncope.

Evaluation of the Patient: The “Five-finger” Approach of Dr. W. Proctor Harvey

History



Physical Signs



ECG

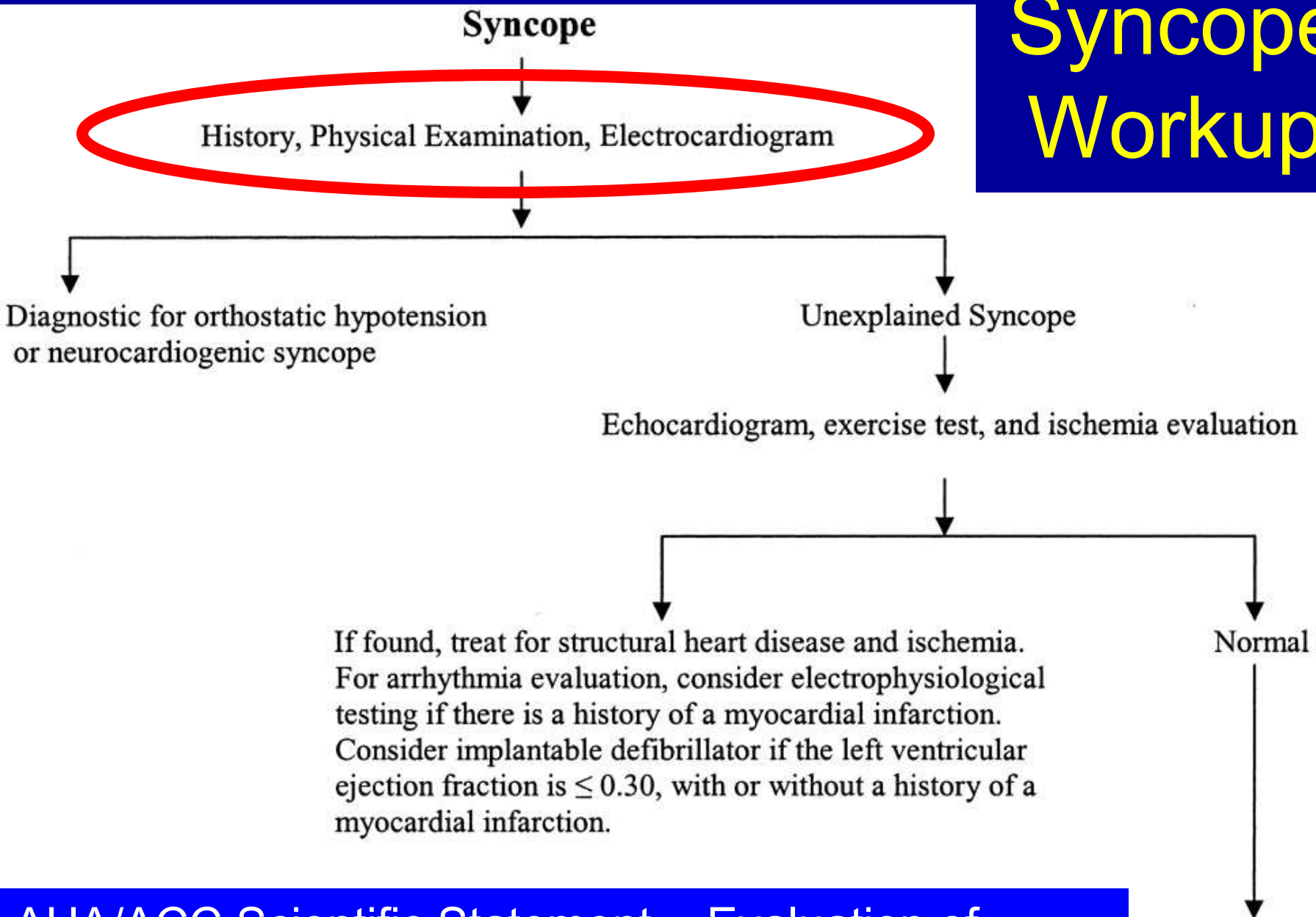
X-Ray



Diagnostic Laboratory

W. Proctor Harvey, April 19, 1918 to September 26, 2007

Syncope Workup



AHA/ACC Scientific Statement - Evaluation of Syncope. Circulation 2006;113:316.

Aspects of the History in the Patient Presenting with Syncope

- Note: The history is the most important contributor to a correct diagnosis
- The history
 - The days and hours before the event
 - The prodrome and precipitants
 - The episode and consequences (injury) and the recovery

Days and Hours Before the Event

- Recent immobilization or injury (pulmonary embolism)
- Change in medication (orthostatic hypotension, proarrhythmia)
- Recent illness or decreased fluid intake
- Heat exposure, dehydration

Prodrome in Syncope

- Feeling of heat, lightheadedness, sweating, pallor, or nausea or vomiting – neurocardiogenic
- No prodrome (or <5 sec) – arrhythmia, autonomic dysfunction with hypotension
- Aura or premonition – seizure
- Palpitations – arrhythmia, neurocardiogenic

Precipitants in Syncope

- Standing or after exercise – neurocardiogenic or postural hypotension
- During exercise – cardiac, neurocardiogenic
- Seated or reclining – cardiac
- Loud noise or extreme stress in young - cardiac
- Noxious stimulus, pain or fear – neurocardiogenic
- Turning of head, shaving or tight collars – carotid sinus hypersensitivity
- Deglutition, micturition, defecation, cough, laugh – reflex syncope

The Clinical Setting in Syncope

- Age forms an important context
- Prior head trauma might indicate neurologic cause (seizure)
- Prior cardiac history or current cardiac symptoms (coronary, valvular, myocardial, or congenital disease)
- Family history of syncope or sudden death (in family member <30 yo), especially in young patients

The Event and Sequelae

- Observations by onlookers often provide critically important information – tonic-clonic seizure activity can occur both with true seizure and with cardiac and neurocardiogenic causes of syncope
- No sequelae – arrhythmia, orthostatic hypotension
- Fatigue, nausea, weakness – neurocardiogenic
- Postictal confusion or focal neurologic symptoms – neurologic
- Injury is present in about 1/3, no diagnostic significance

History to Distinguish Syncope from LOC due to Seizure

- Waking with cut tongue after spell +2
- Prodrome of déjà vu or jamais vu +1
- LOC with emotional stress +1
- Head turn to one side during LOC +1
- Unresponsive, posturing, jerking limbs, no recollection during LOC +1
- Confusion after LOC +1
- Lightheaded spells -2
- Diaphoresis before LOC -2
- LOC with prolonged stand or sit -2
- Score ≥ 1 is Seizure with accuracy 85%
- Score < 1 is Syncope

Evaluation of the Patient: The “Five-finger” Approach

Physical Exam

History

ECG

Chest X-Ray

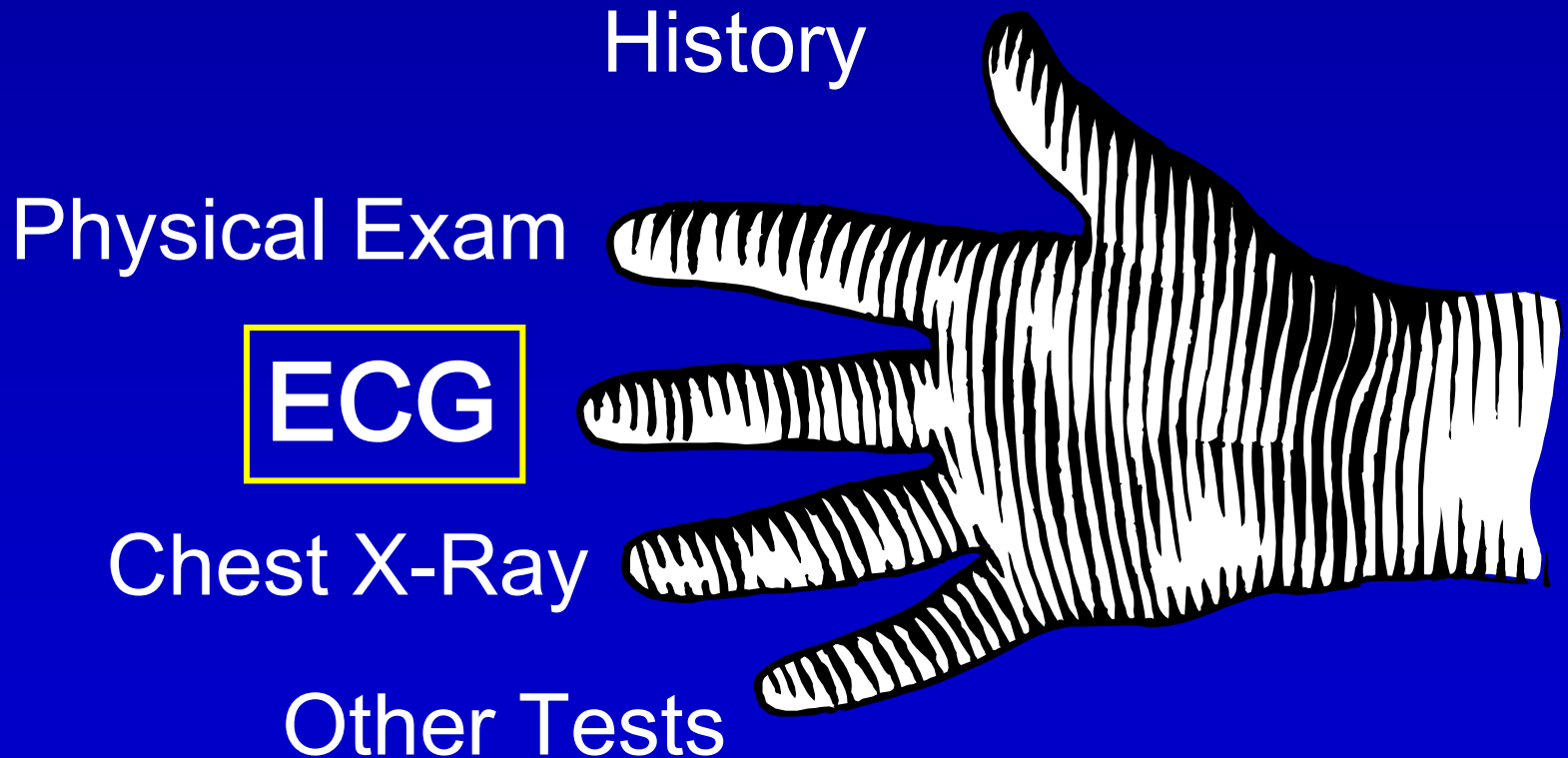
Other Tests



Physical Examination in the Patient with Syncope

- Vital signs – orthostatic BP at 3 minutes, check femoral pulses, heart rate, regularity of rhythm
- Head trauma – tongue biting, esp. unilateral
- Cardiovascular – jvp, bruits, LV heave, RV lift, heart sounds (loud P2), gallops, murmurs (AS, MS, tumor plop)
- Abdominal pain or tenderness
- Neurologic – cognition, speech, visual fields, motor strength, tremor, gait
- Bedside maneuvers – carotid sinus massage, Valsalva maneuver (autonomic function)

Evaluation of the Patient: The “Five-finger” Approach



ECG Abnormalities in Syncope

(helpful in about 5%)

- Bifascicular block or IVCD (QRS >0.12 sec)
- Mobitz I (Wenckebach) AV block (?1st AVB)
- Asymptomatic sinus bradycardia (<50) or SA exit block or pause (>3 s) in absence of negative chronotropic medications
- Delta wave – WPW and tachyarrhythmia
- Long QT interval, Brugada syndrome
- Arrhythmogenic RV cardiomyopathy
- Signs of heart disease – MI, (hypertrophy)

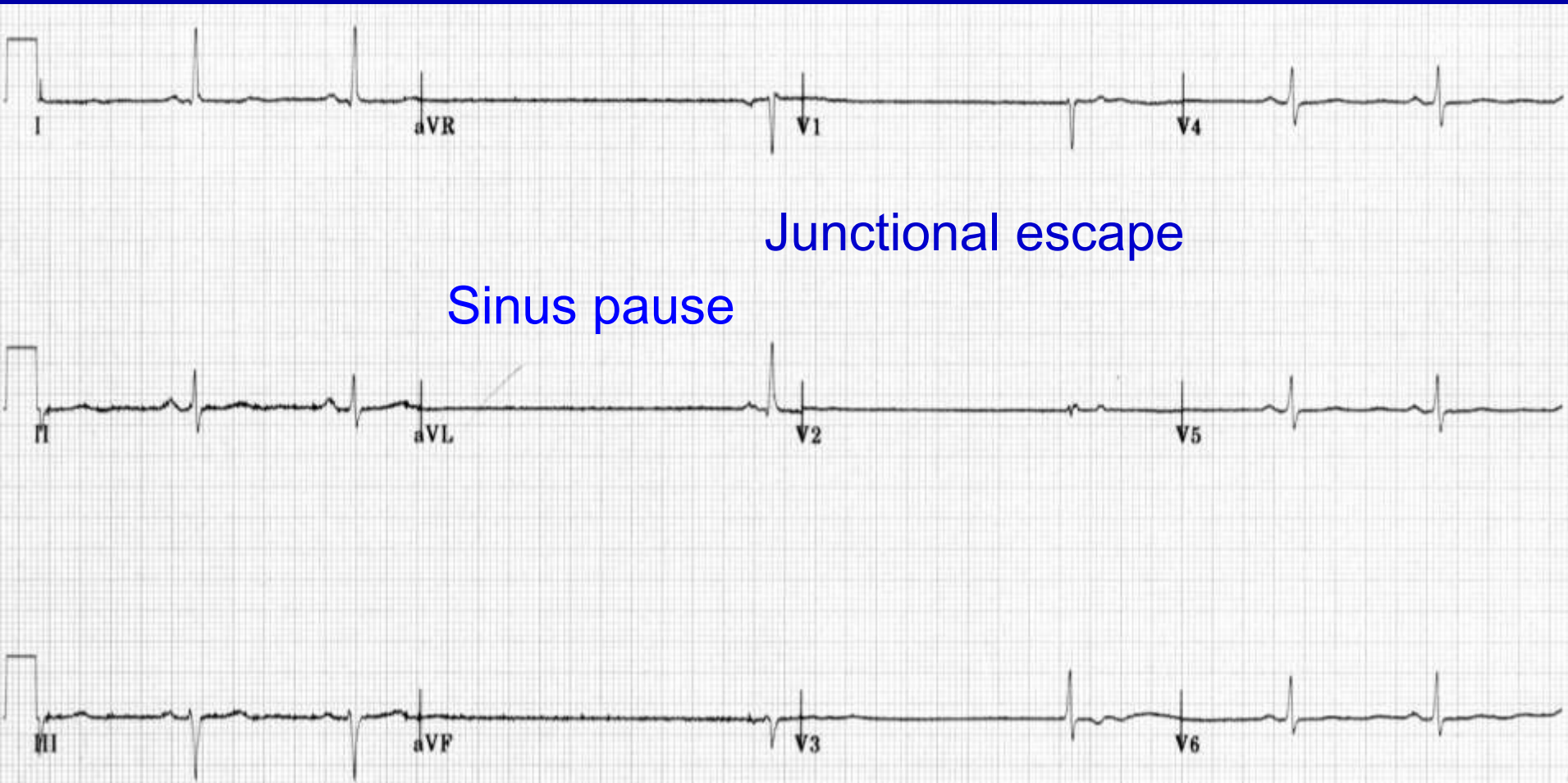
Arrhythmic Causes of Syncope

Bradyarrhythmia

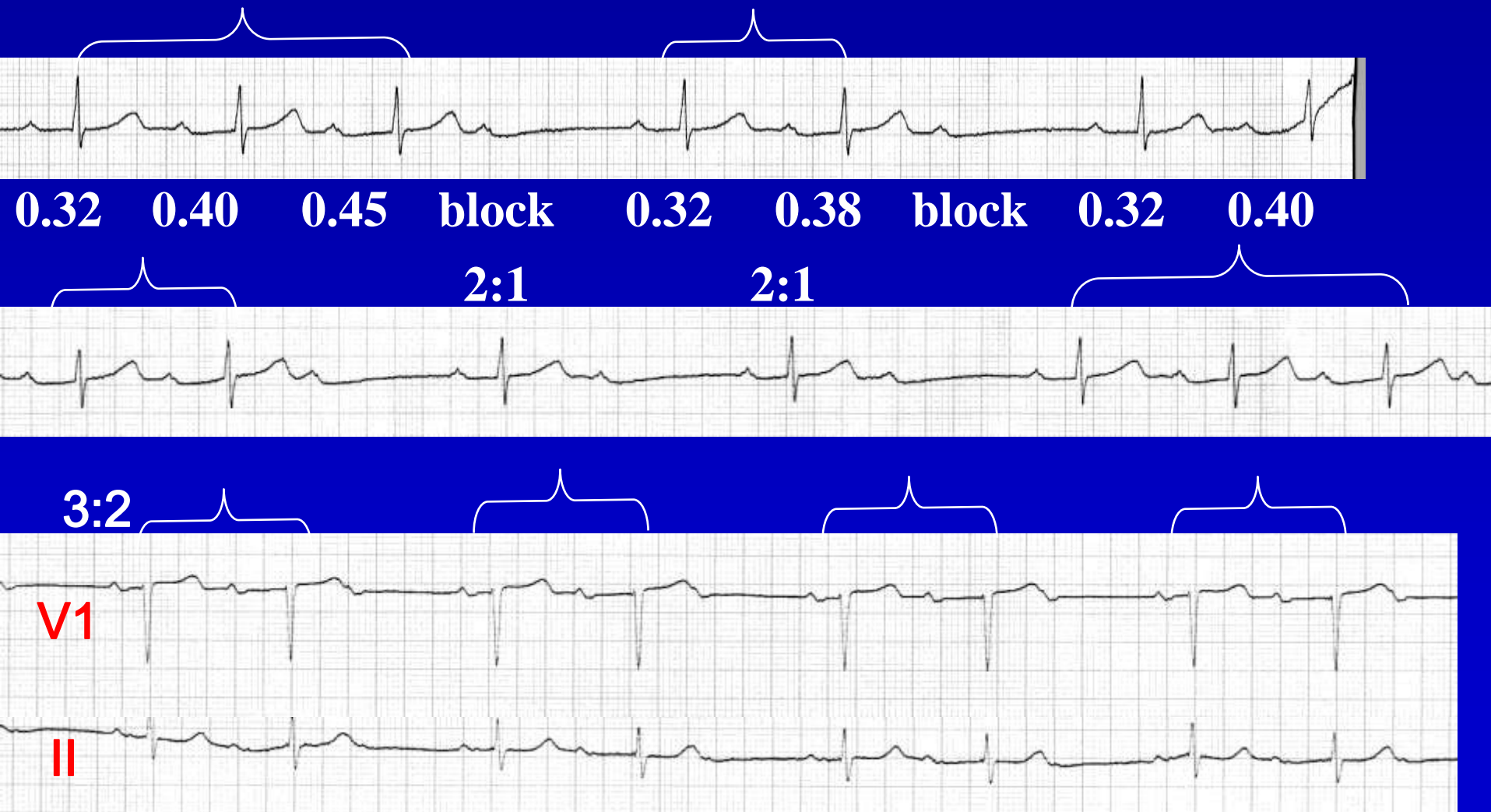
Tachyarrhythmia

Syncope and Bradyarrhythmias

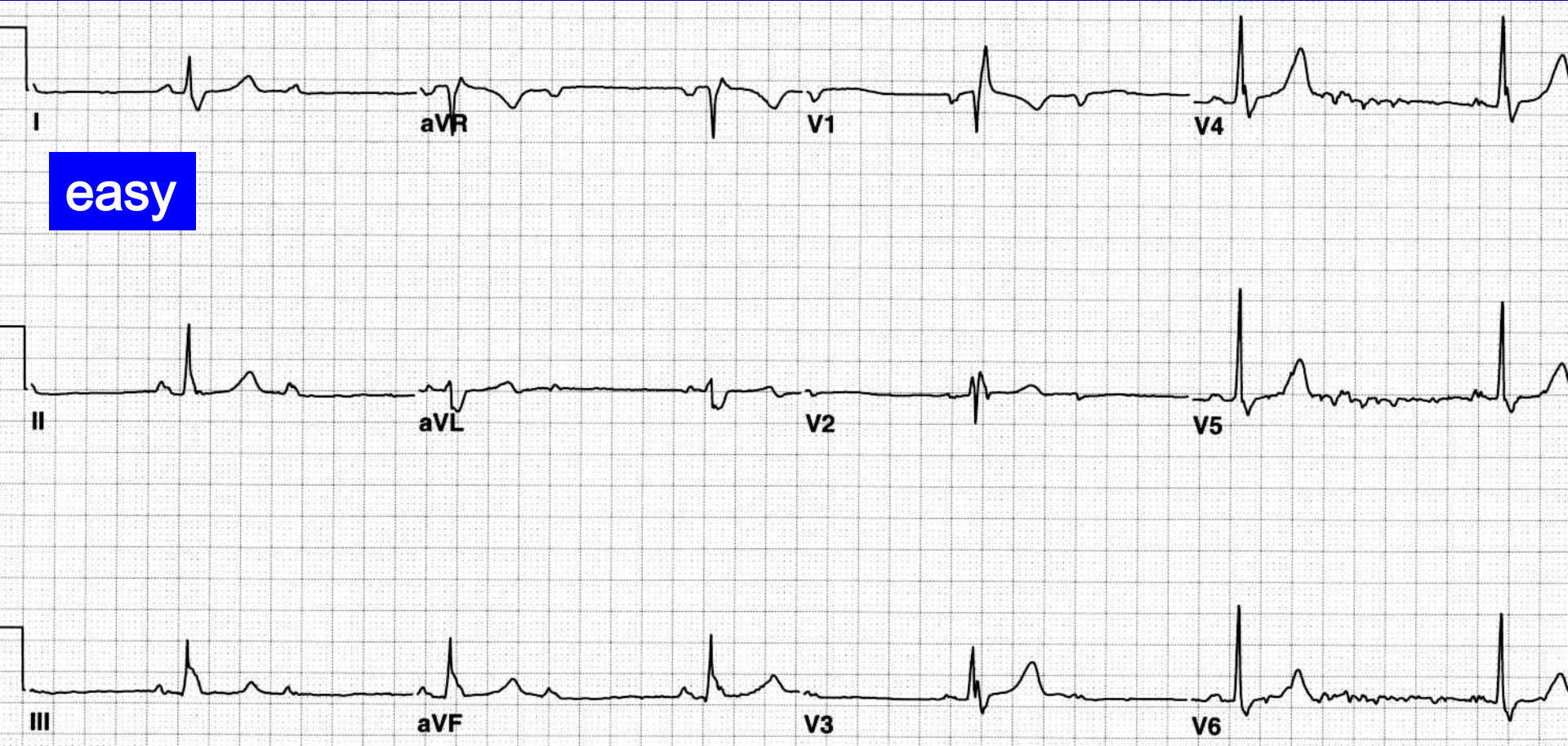
- About 2/3 of patients with syncope due to arrhythmia are due to bradyarrhythmia (AV block more than sinus node dysfunction)



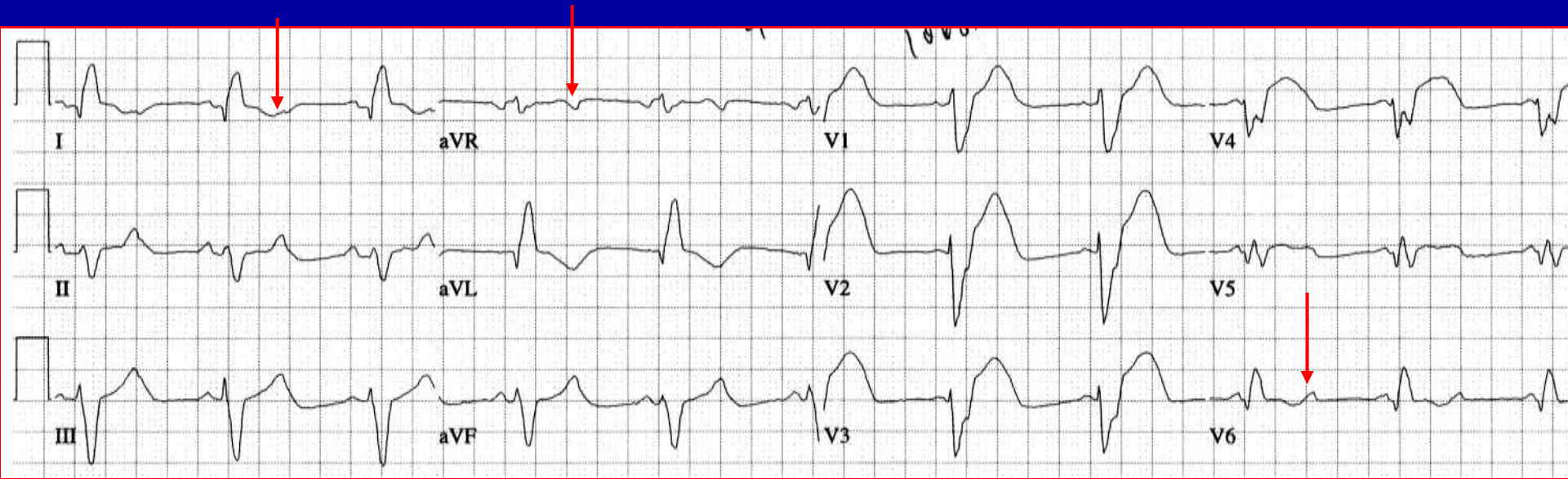
Second Degree AV Block, Wenckebach (Mobitz I)



Second Degree AV block, 2:1



Second Degree AV block, 2:1



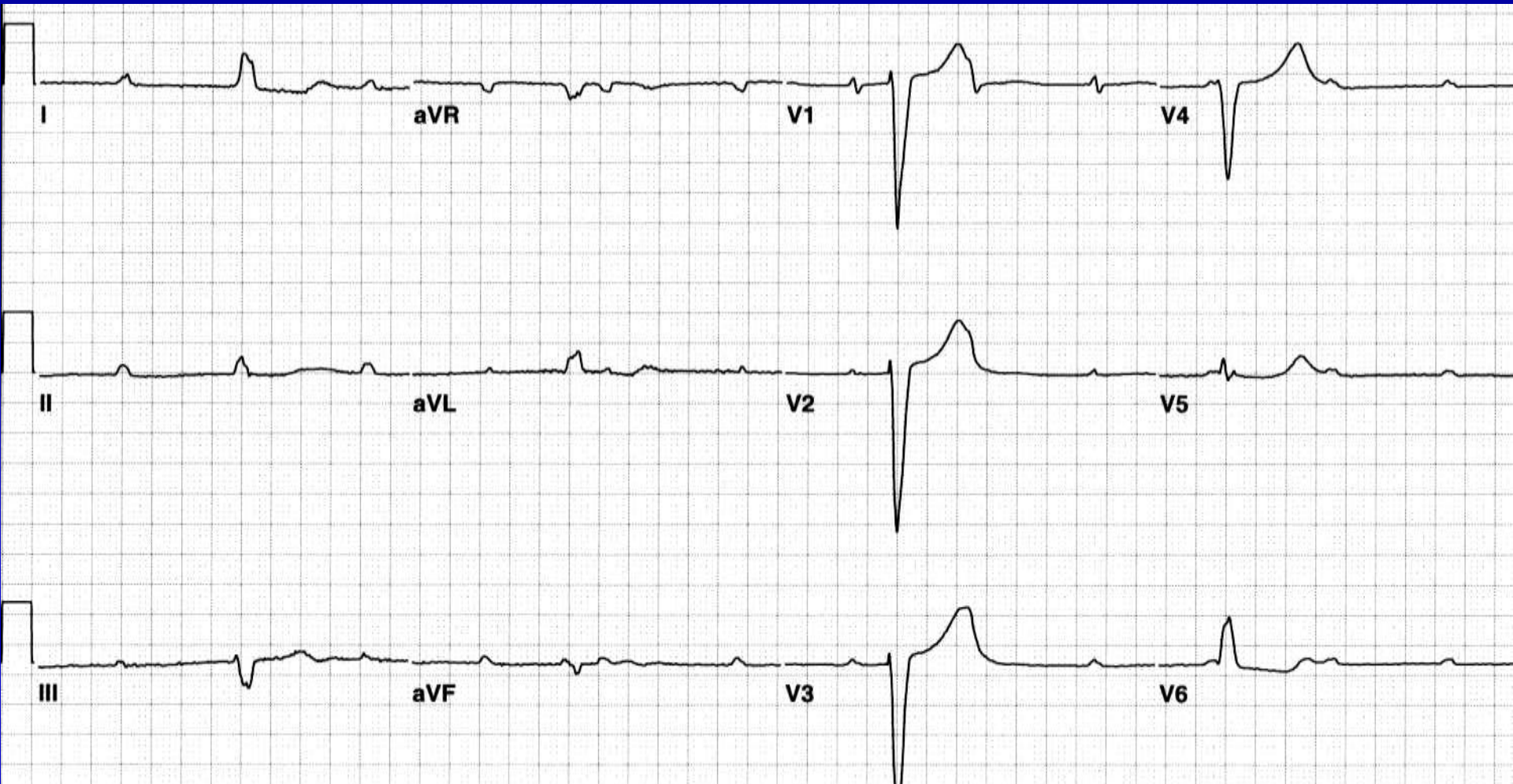
- Not so easy... could misdiagnose as NSR rate 64.
- But actually is sinus tachycardia at rate of 128 (patient is likely sick) with 2:1 block.
- The extra P waves are best seen at the 3 red arrows, and are same shape and axis as the sinus P waves.
- Wide QRS indicates disease below the bundle of His.

Second-Degree AV Block, Mobitz II

- Intermittent blocked P waves
- PR interval constant for conducted beats
- Most are associated with BBB
- About 1/3 of patients with Mobitz II have block located in the His bundle, so QRS is narrow
- Rarely Mobitz II is due to block in the AV node



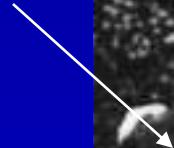
Third Degree AV block (Complete Heart Block)



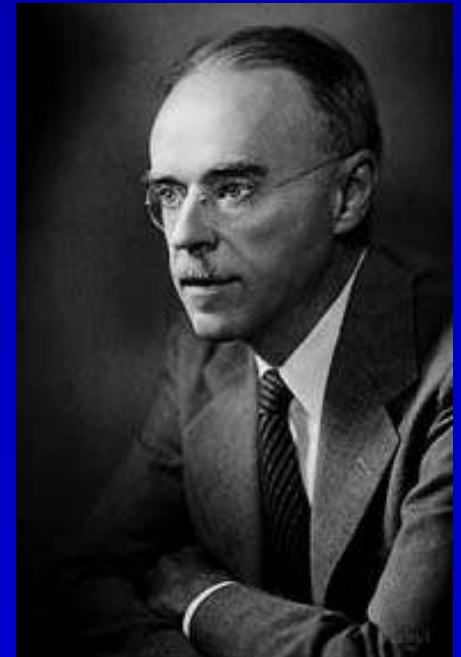
Wolff-Parkinson-White

The Three Authors

Louis Wolff



Sir John Parkinson



Paul Dudley White

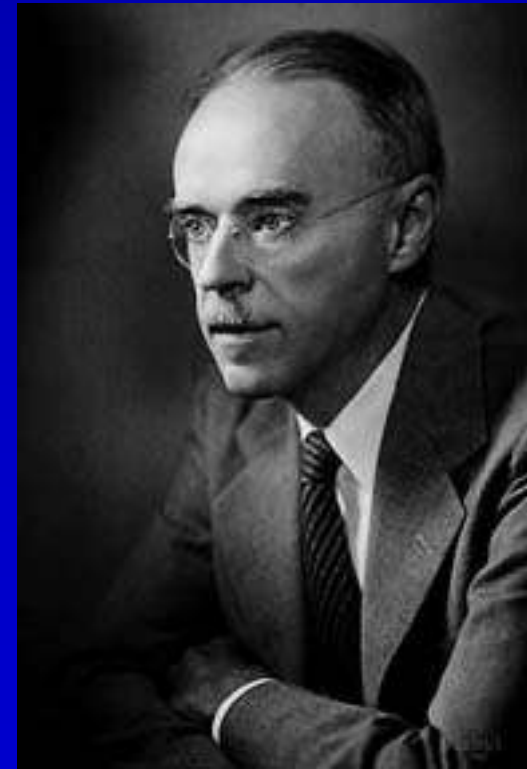
Paul Dudley White

(1886-1973)

Founder of preventive cardiology



1986



Original Article

The American Heart Journal

VOL. V

AUGUST, 1930

No. 6

Original Communications

BUNDLE-BRANCH BLOCK WITH SHORT P-R INTERVAL
IN HEALTHY YOUNG PEOPLE PRONE TO
PAROXYSMAL TACHYCARDIA

LOUIS WOLFF, M.D., BOSTON, MASS., JOHN PARKINSON, M.D., LONDON,
ENG., AND PAUL D. WHITE, M.D., BOSTON, MASS.

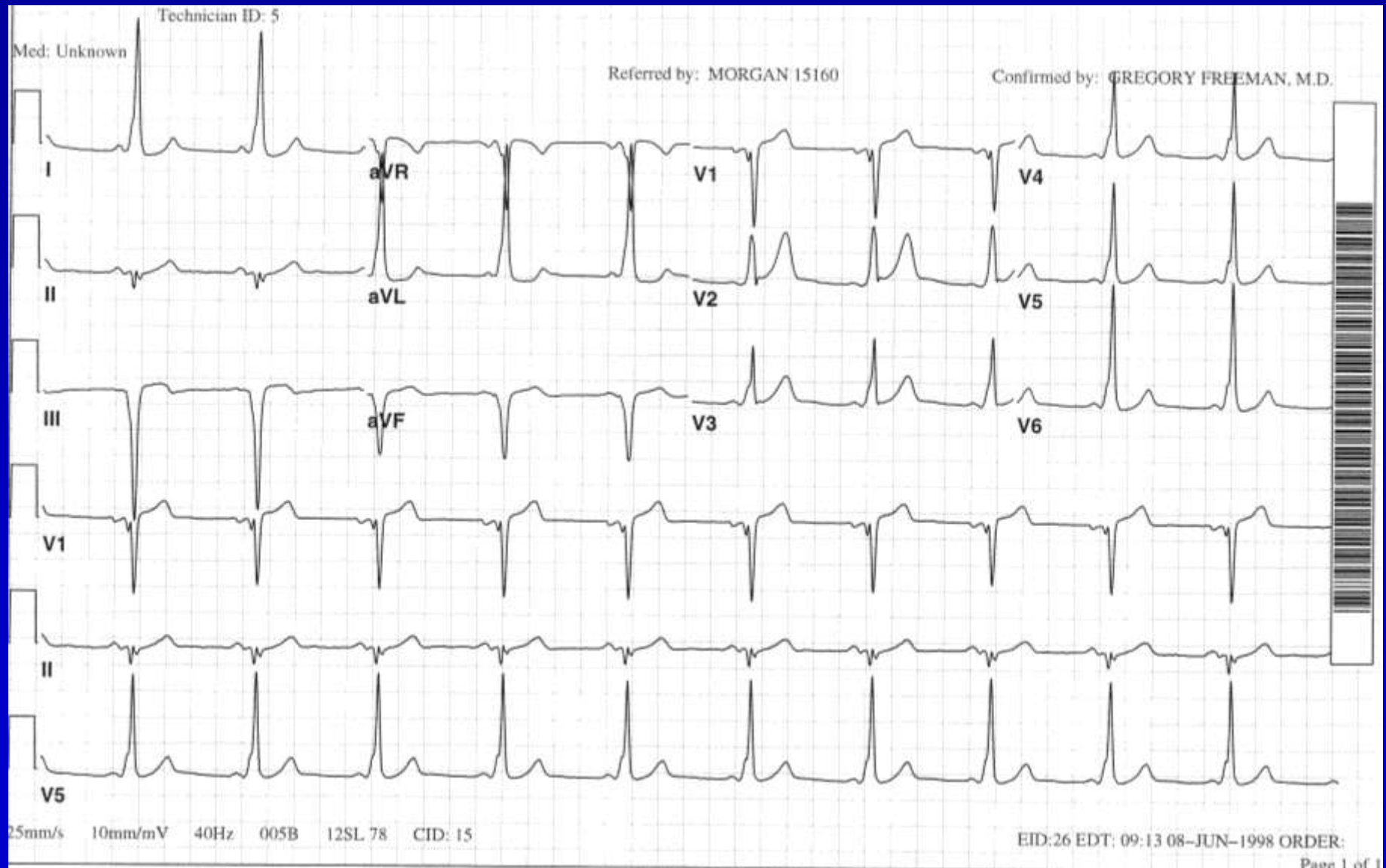
Original Article, Case III, Intermittent Pre-excitation



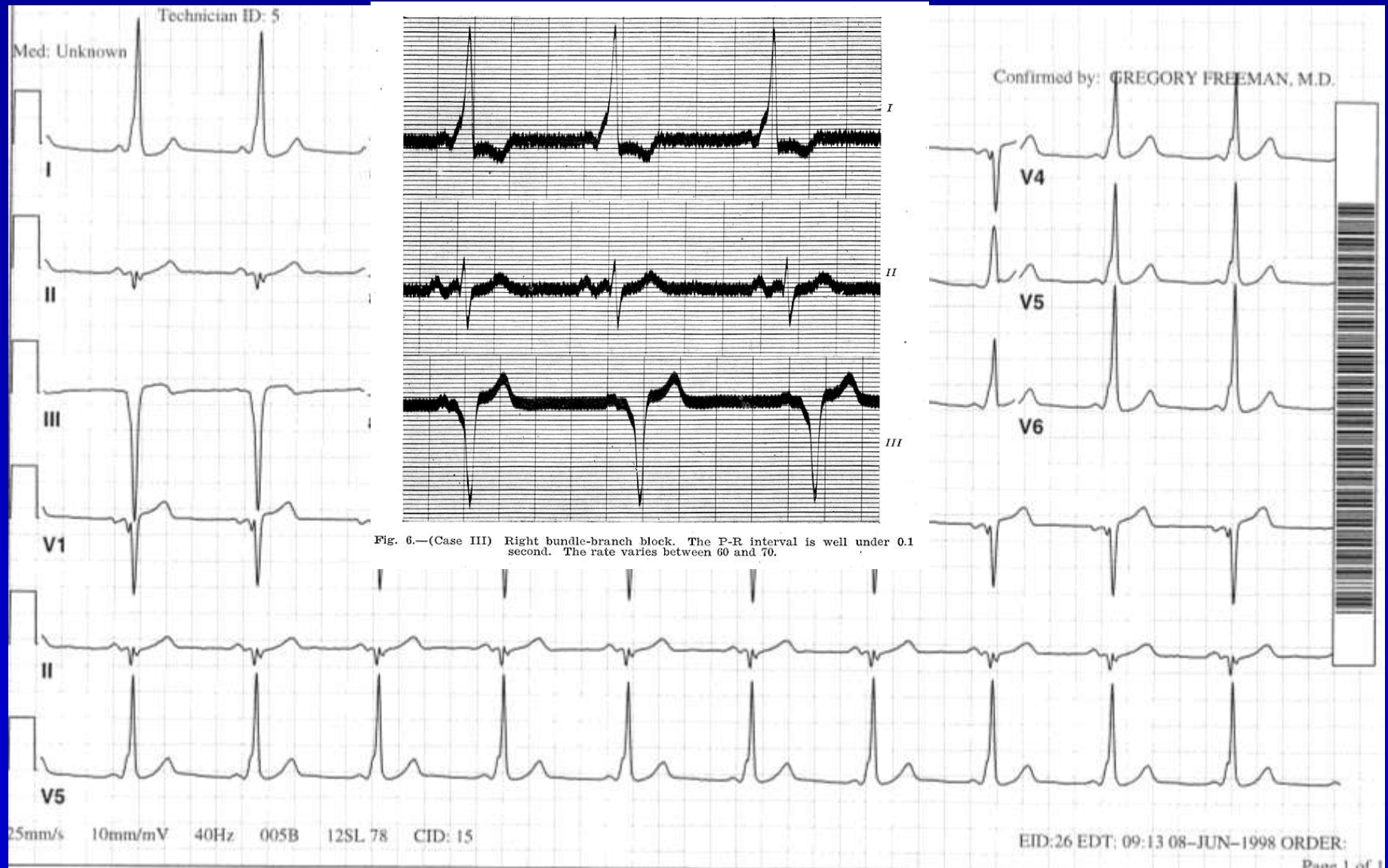
Fig. 6.—(Case III) Right bundle-branch block. The P-R interval is well under 0.1 second. The rate varies between 60 and 70.

1930

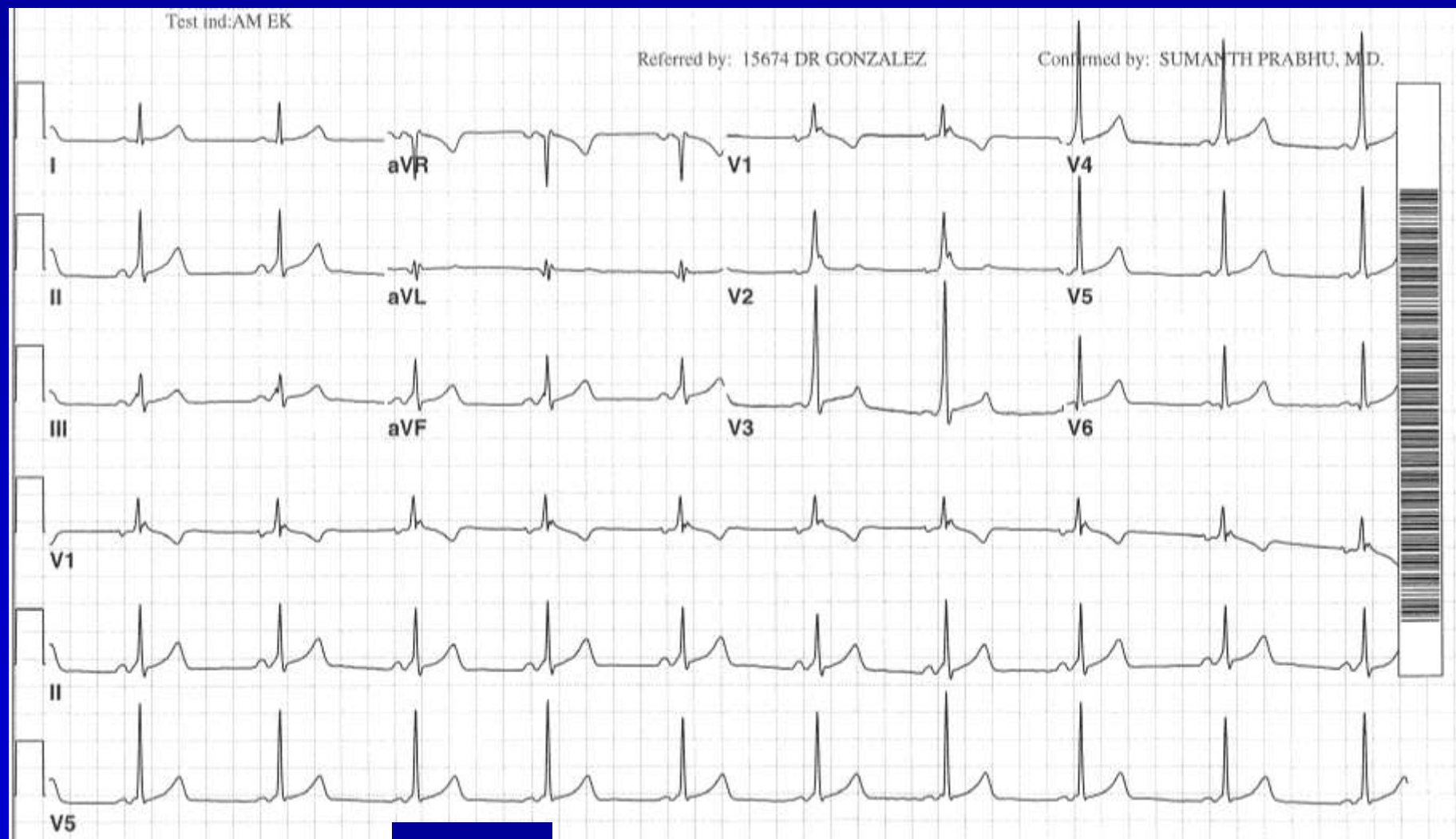
WPW



WPW

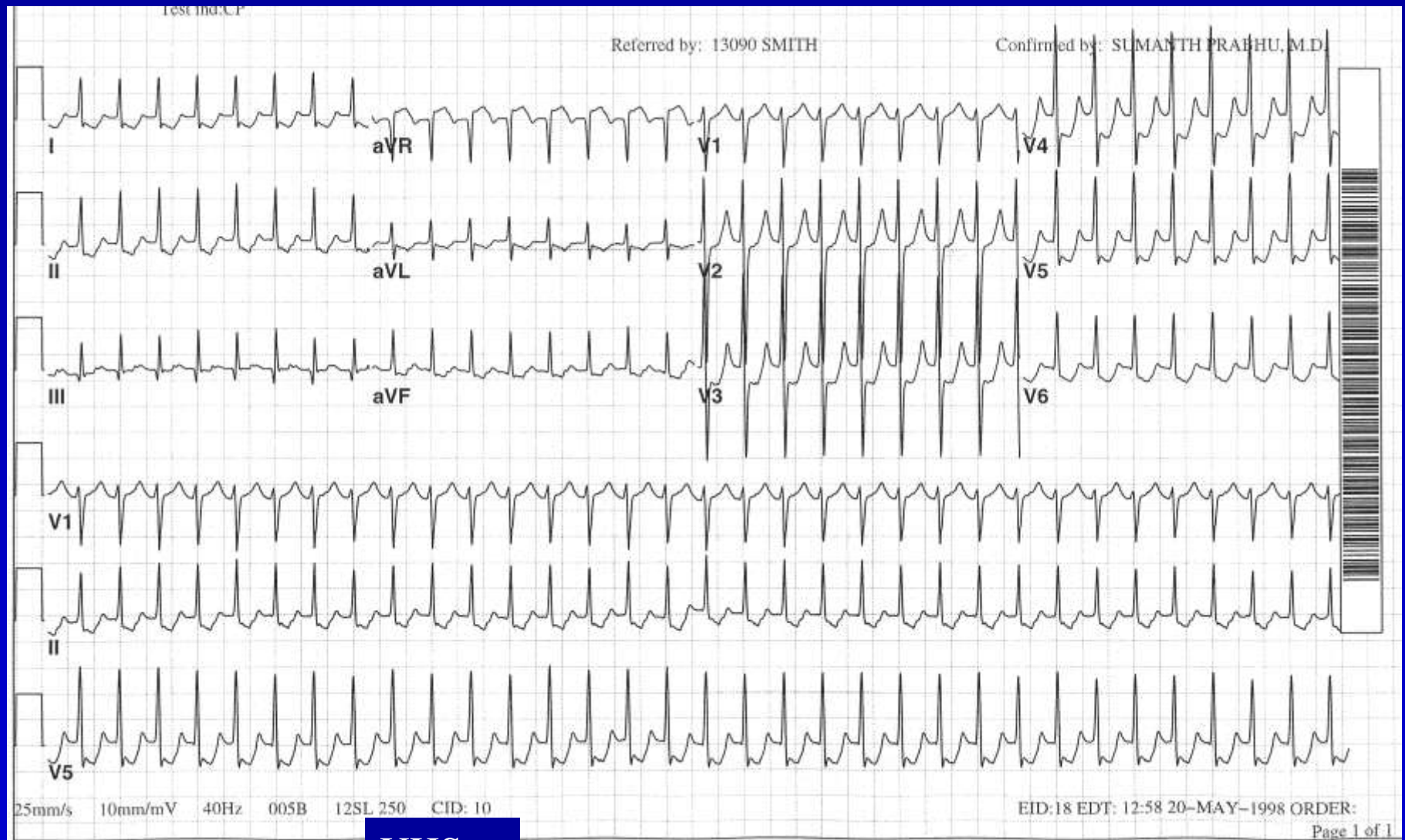


WPW



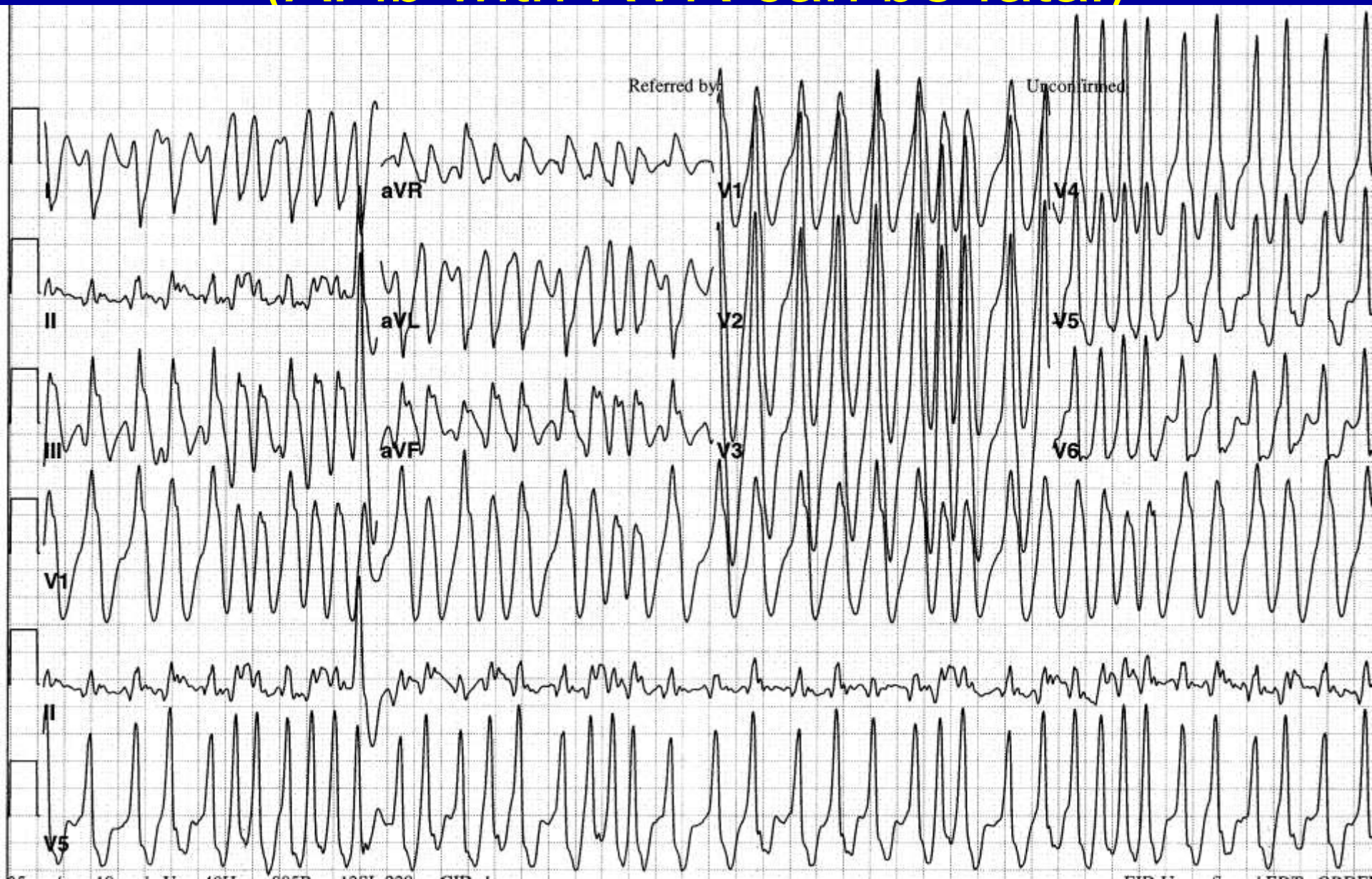
UHS pt

WPW: Orthodromic AVRT

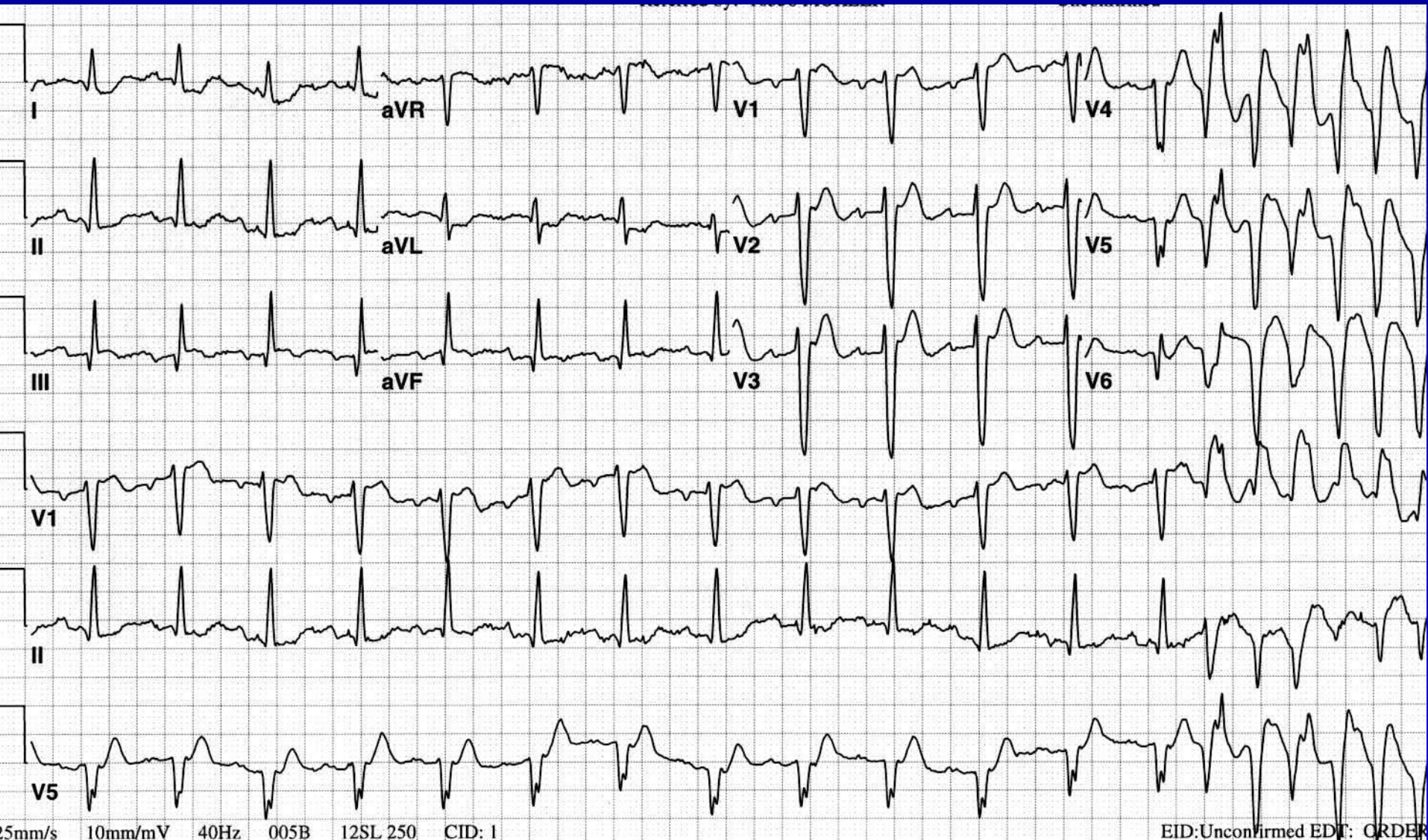


UHS pt

WPW and Atrial Fibrillation (AFib with RVR can be fatal)



Ischemia with Consequent VT



5mm/s 10mm/mV 40Hz 005B 12SL 250 CID: 1

EID:Unconfirmed EDT: ORDER

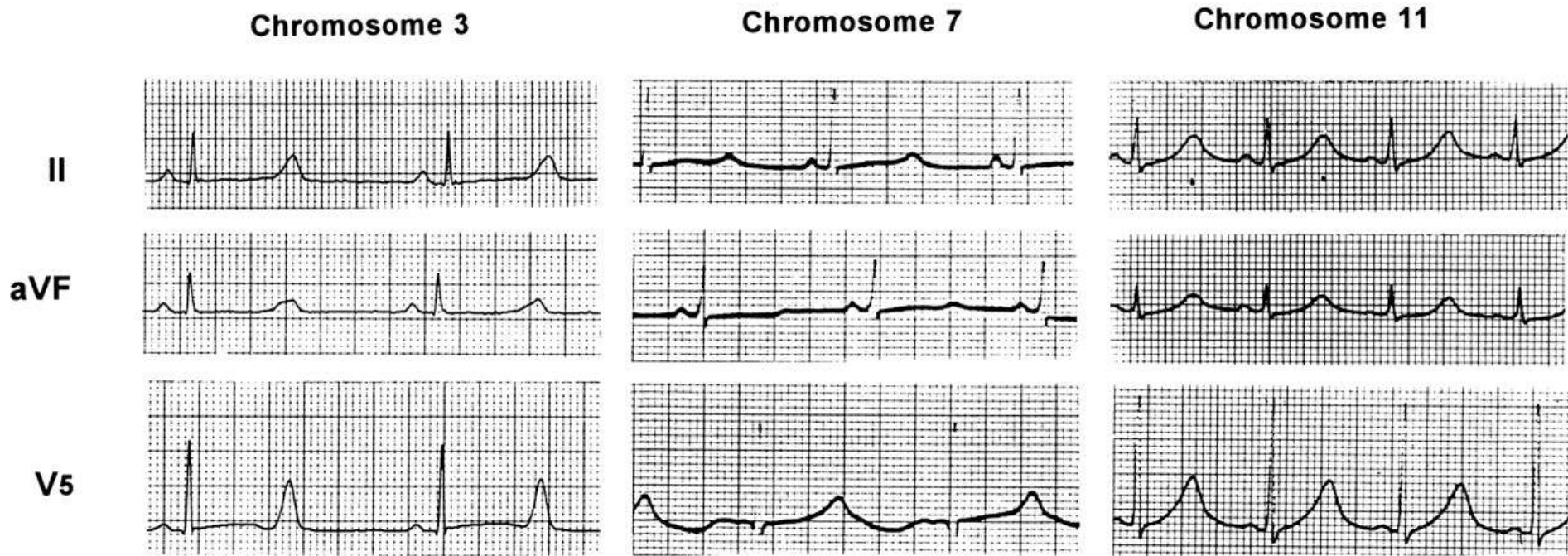
Baseline ECG shows inferior injury and then polymorphic VT

Ischemia with Consequent VT



Polymorphic VT 10 seconds later

Congenital Long QT Syndrome

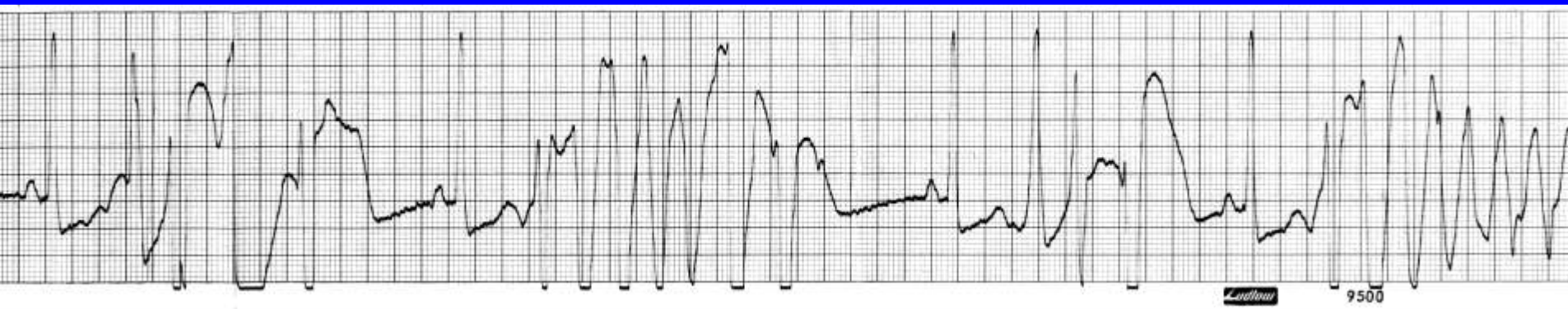
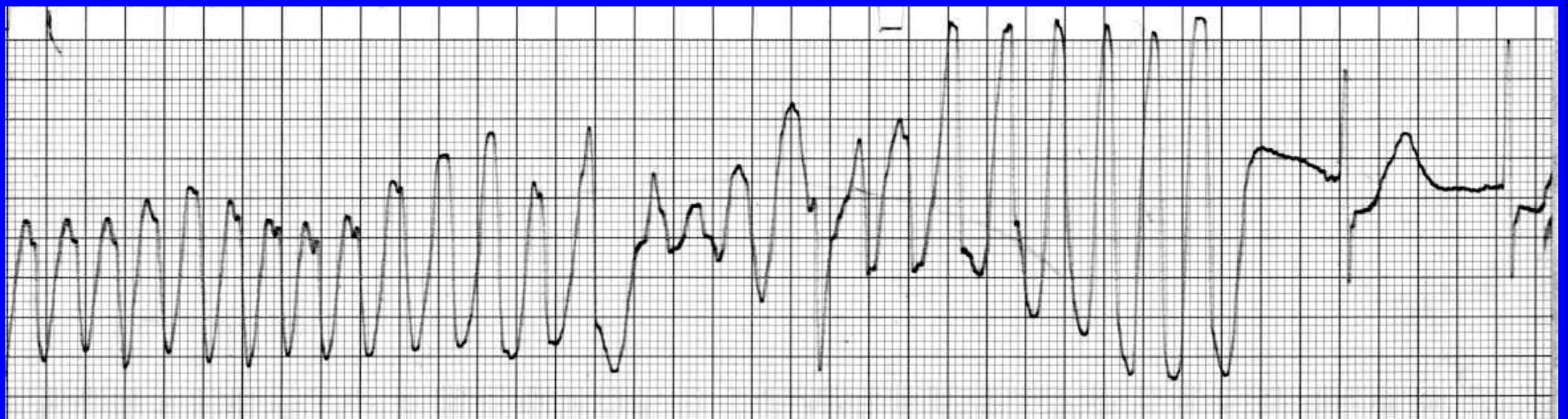
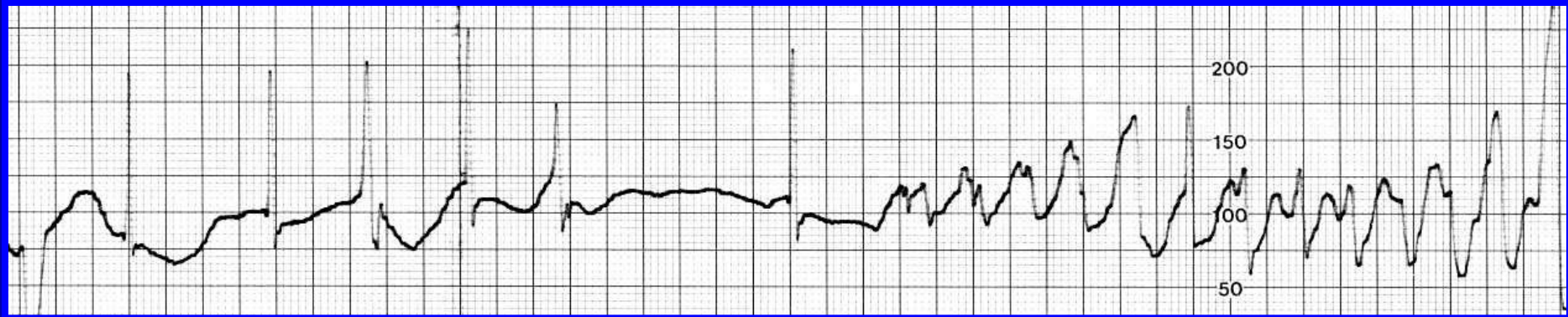


Three patients with long QT syndrome linked to genetic markers. None were receiving β -adrenergic blocking medication. Chromosome 3 mutation in the cardiac sodium channel gene *SCN5A*, the QTc in lead II is 570 ms with late-onset T waves of normal duration and amplitude. Chromosome 7, the QTc in lead II is 583 ms with low-amplitude T waves. Chromosome 11, the QTc in lead II is 573 ms with early onset of broad-based T waves.

AHA/ACC Scientific Statement - Evaluation of Syncope.

Circulation 2006;113:316.

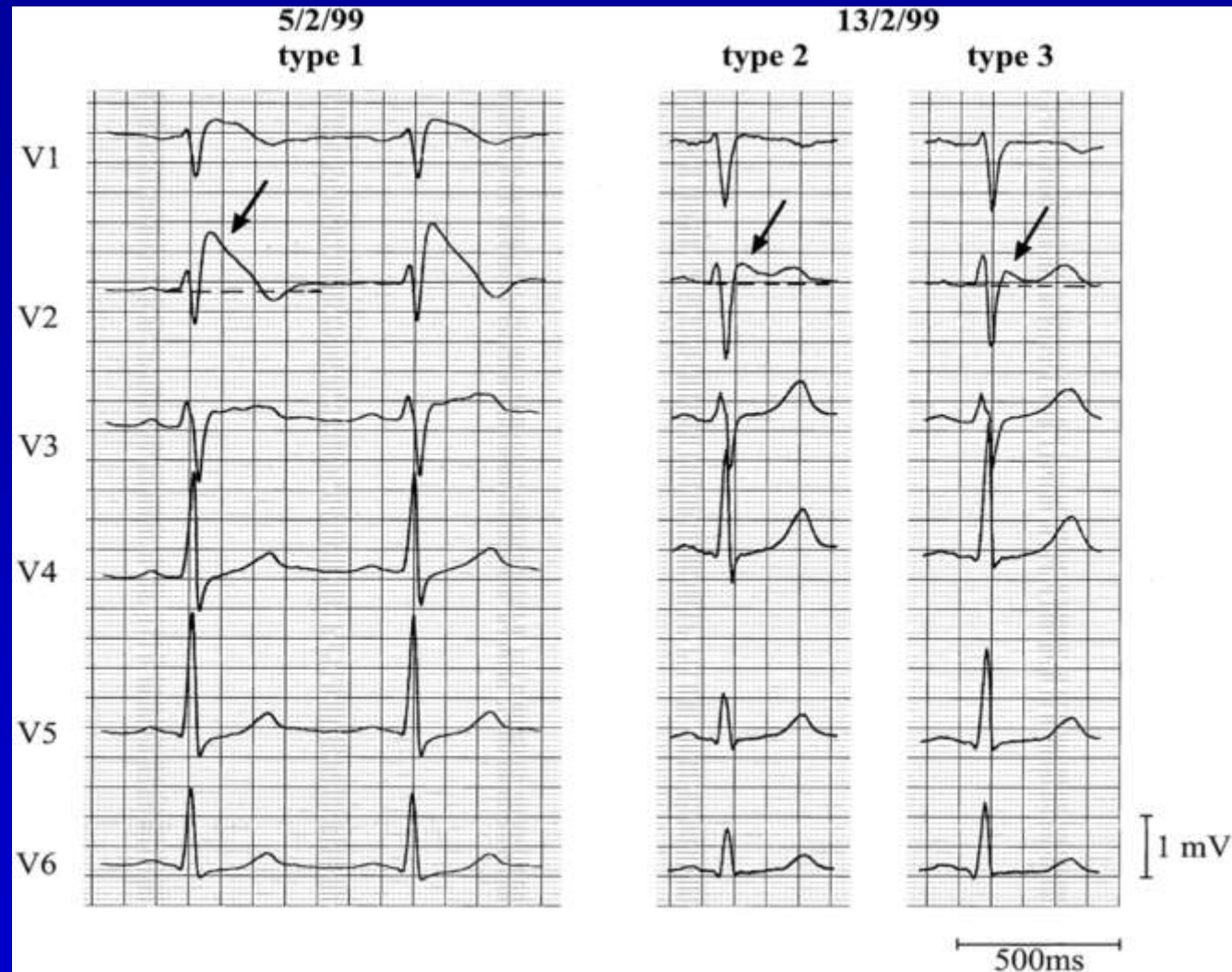
Acquired Long QT interval and Torsades



Brugada ECG Abnormality

ECG changes in the Brugada syndrome. ST elevation occurs in the anterior precordial leads, leads V1 and V2.

Type 1 (coved) ECGs with 1 mV of ST elevation have the most prognostic significance. ECG recordings may change over time, as in this example, and serial ECGs may be important.



AHA/ACC Scientific Statement - Evaluation of Syncope. Circulation 2006;113:316.

Kies P et al. Heart Rhythm 2006;3:225.

Arrhythmogenic RV Cardiomyopathy

Epsilon wave

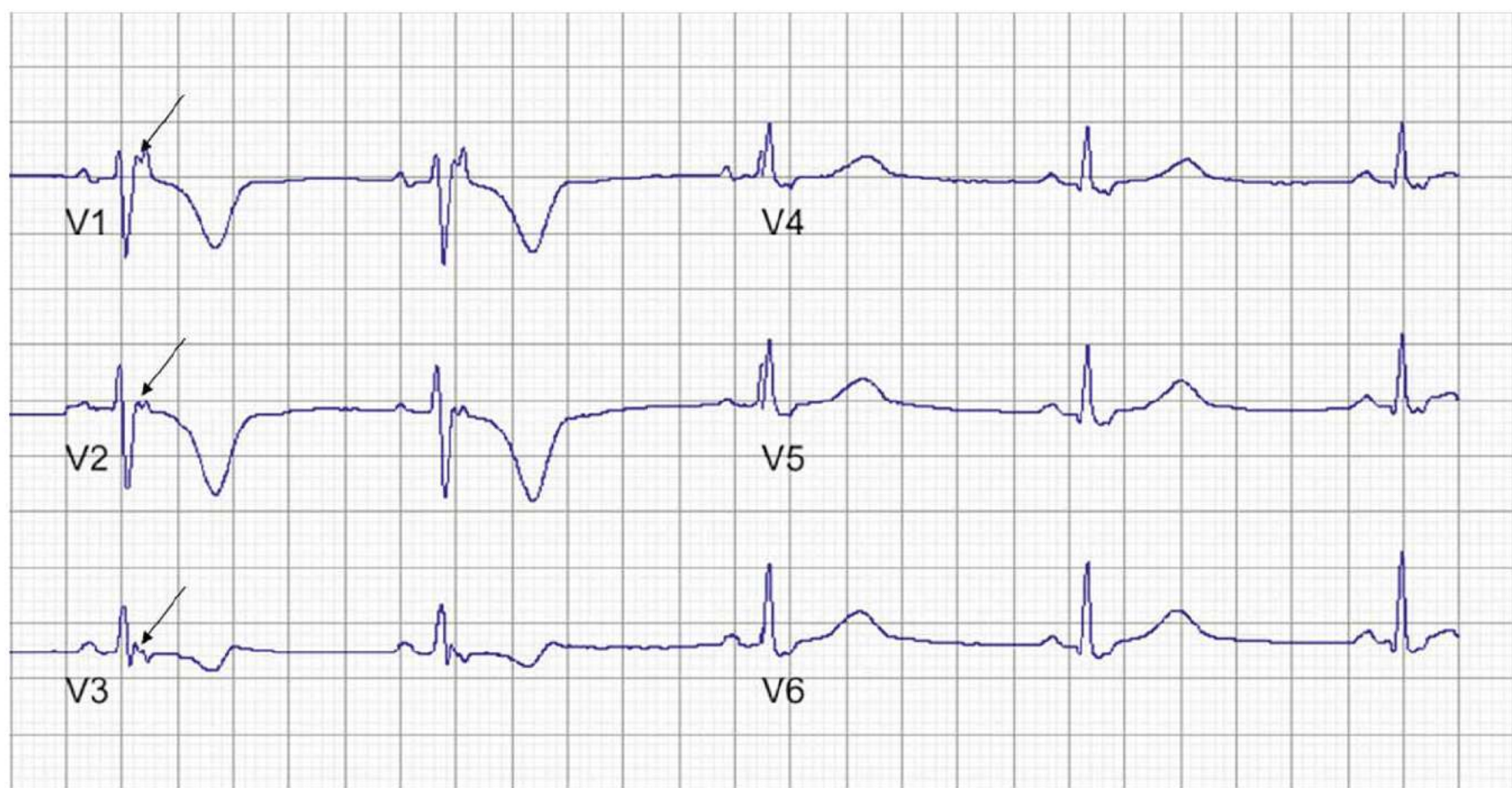
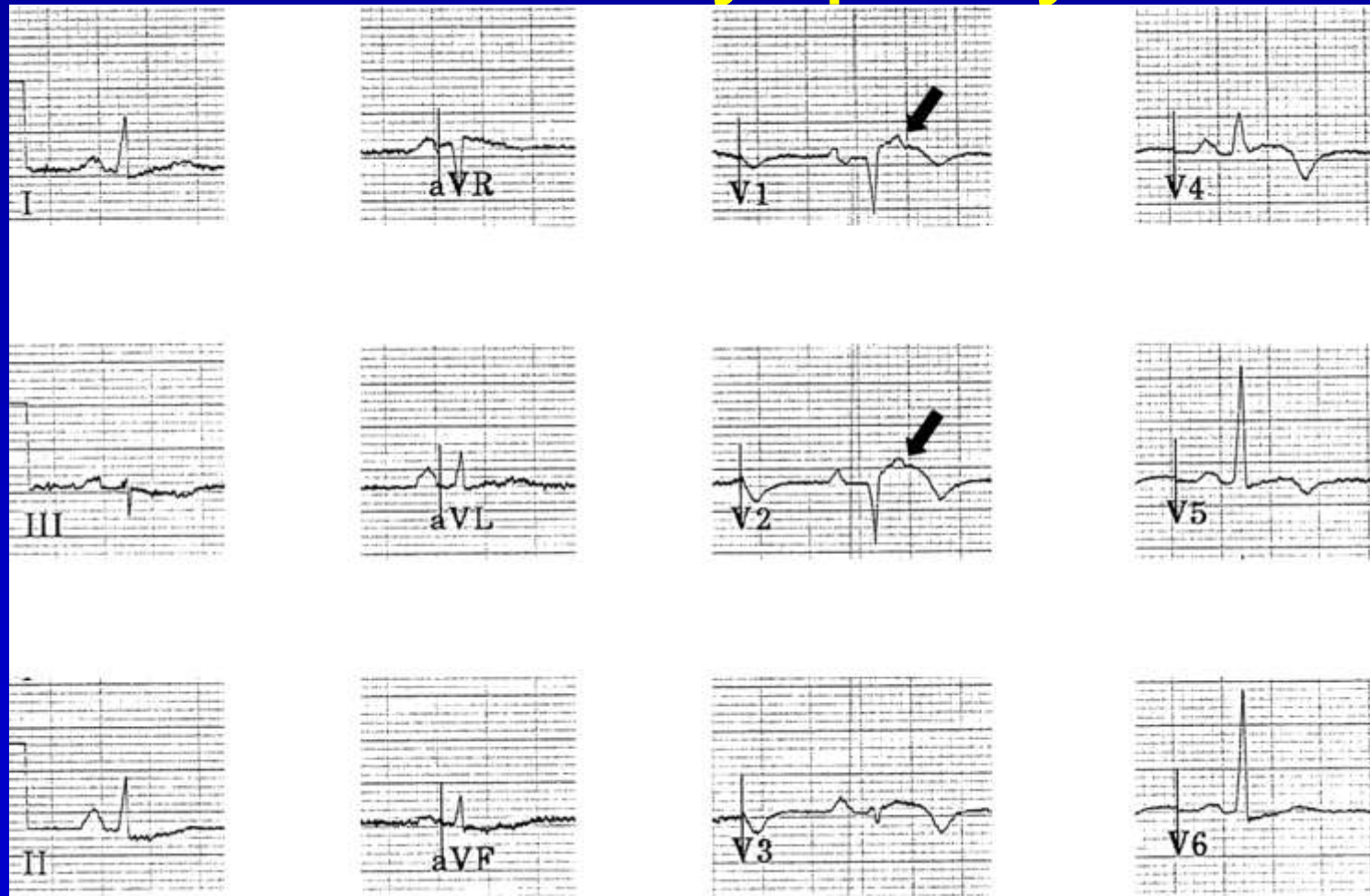


Figure 2 Precordial leads of an ECG from a 44-year-old woman recorded during regular sinus rhythm, with an epsilon wave (*arrow*) in leads V₁–V₃. The ECG shows a right bundle branch block pattern.

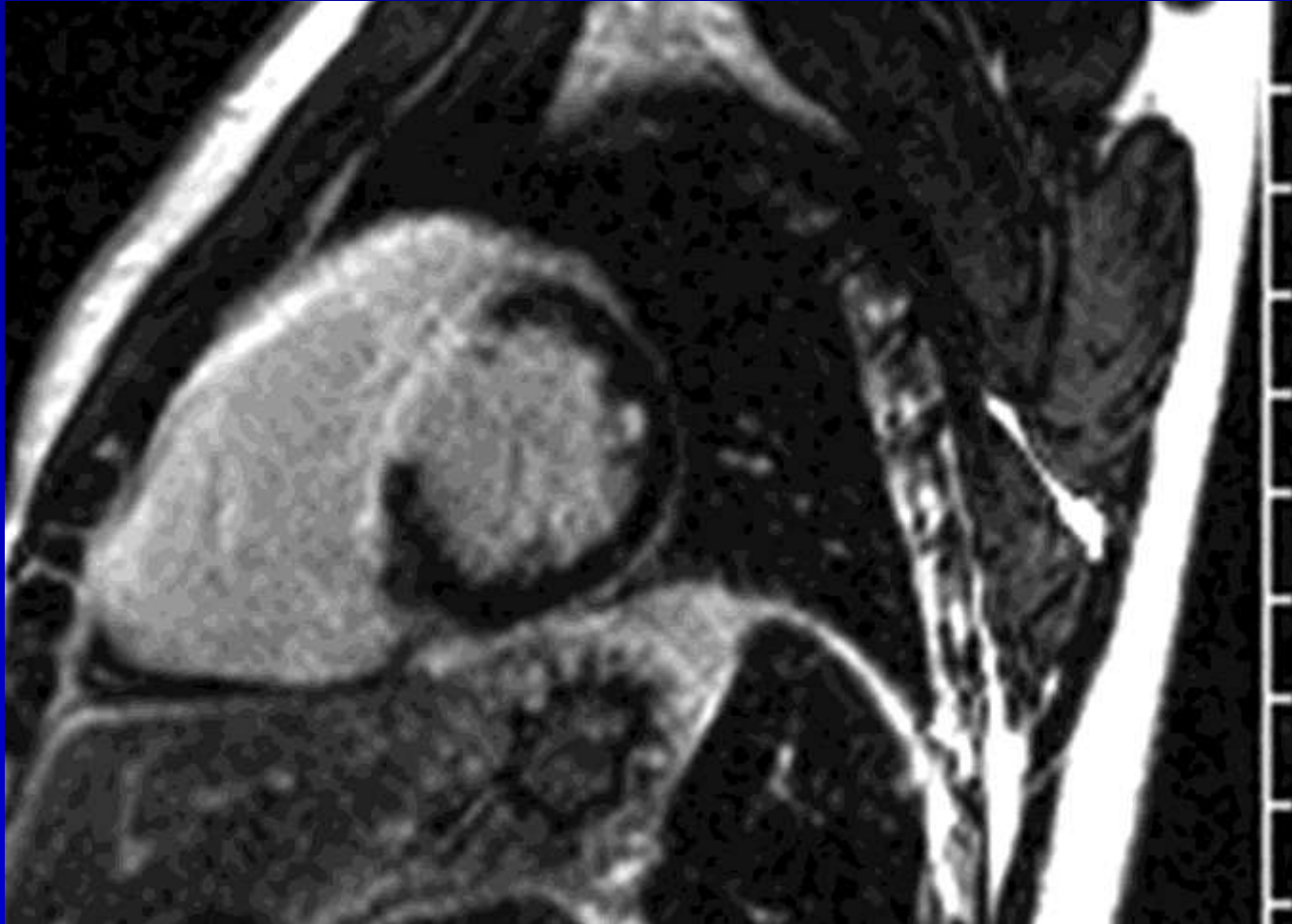
Arrhythmogenic RV Cardiomyopathy

Epsilon wave



RV enlargement and
hyperenhancement of RV
free wall and septum

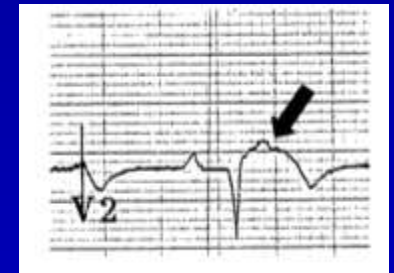
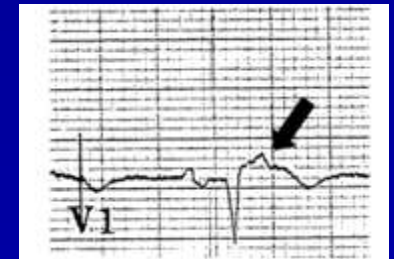
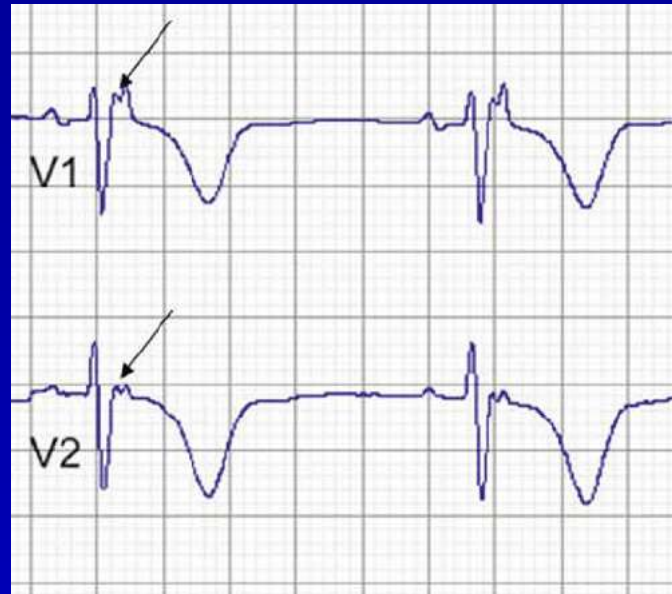
Arrhythmogenic RV Cardiomyopathy



Kenigsberg DN et al. Circulation. 2007;115:e538-e539

ECG in Brugada and RV Cardiomyopathy

RV Cardiomyopathy
Epsilon wave



5/2/99
type 1

13/2/99

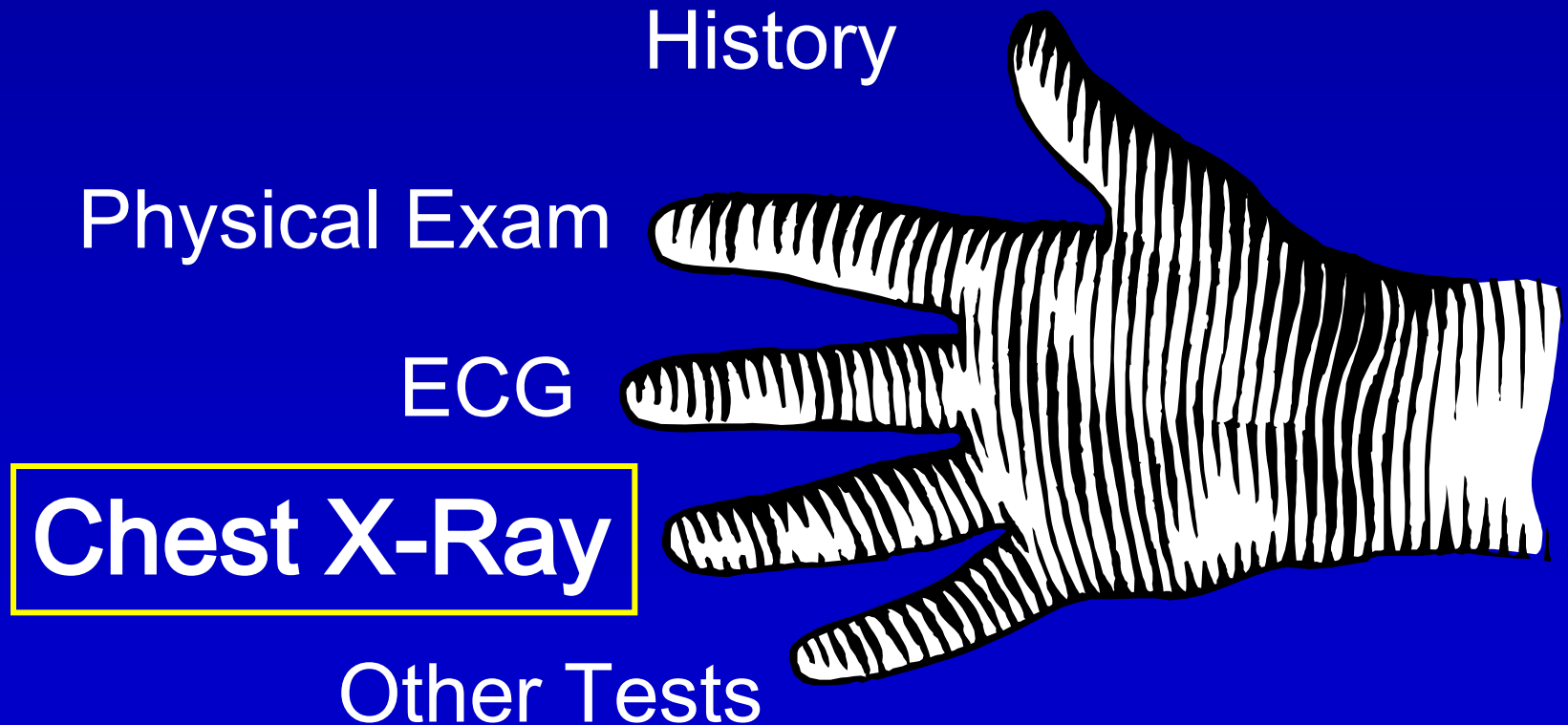
type 2

type 3



Brugada pattern
Coved ST elevation

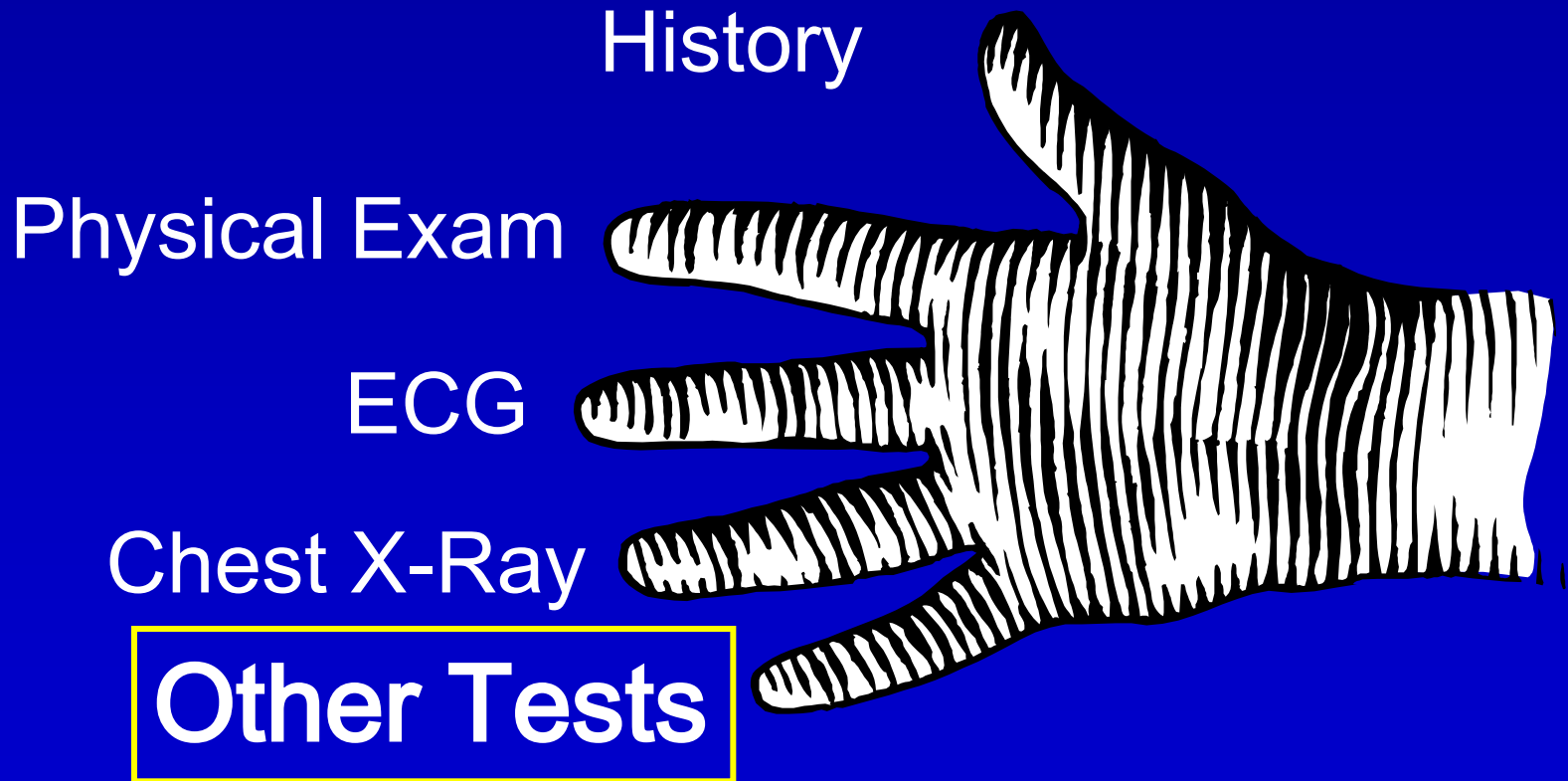
Evaluation of the Patient: The “Five-finger” Approach



Chest X-Ray in Syncope

- Often not helpful, but important if cardiac symptoms or signs are present or if ECG shows hypertrophy or signs of cardiopulmonary disease
- Cardiomegaly
- Pulmonary hypertension/embolus
- Cardiac calcification (AoV, coronary)

Evaluation of the Patient: The “Five-finger” Approach

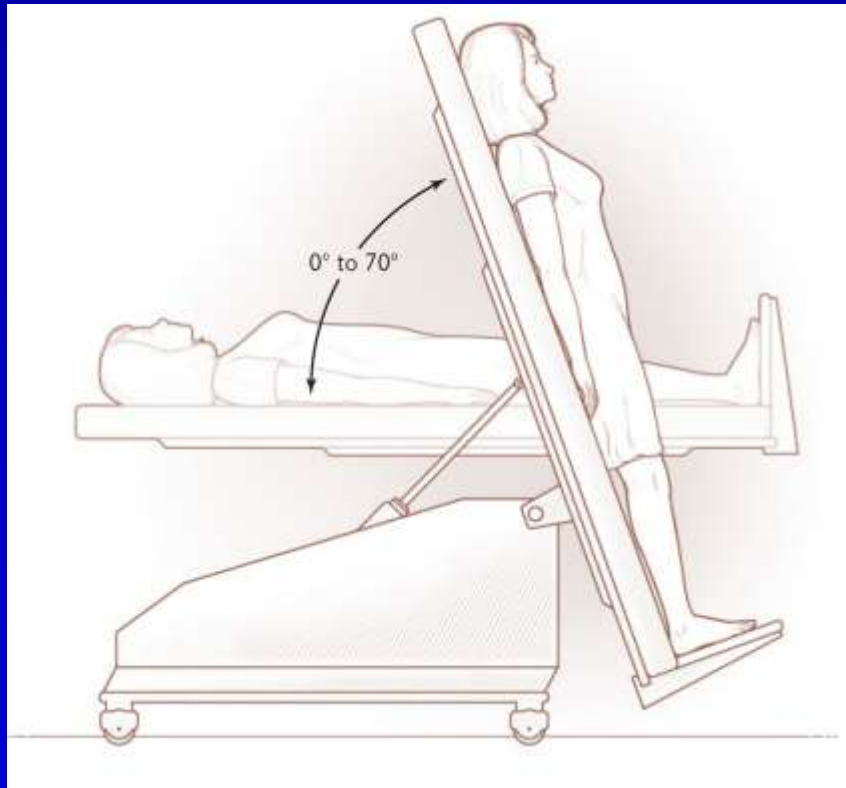


Other Tests in Syncope Evaluation

Testing should only be done when indicated by findings in the history, physical examination or ECG

- Holter Monitor
- Tilt Table Testing
- Head CT scan
- Cardiac stress test with or without imaging
- Echocardiogram
- Lab tests (hct, BUN)

Tilt Table Testing



- Specificity 90%
- Sensitivity 26% to 80%
- In patients with a negative initial evaluation and no evidence of heart disease, the pretest probability of neurocardiogenic syncope is high, so a test contributes little to the diagnosis

Tilt Table Testing

Table 1. Indications for Tilt-Table Testing.*

Definite indications

- Unexplained recurrent syncope or a single episode in the absence of organic heart disease either associated with injury or in settings that pose a high risk of injury
- Unexplained recurrent syncope or a single episode in the presence of organic heart disease after cardiac causes of syncope have been excluded
- A case in which the cause of syncope has been determined but the predisposition to neurocardiogenic syncope may alter the treatment used

Possible indications

- Differentiation of convulsive syncope from epilepsy
- Assessment of recurrent, unexplained falls
- Evaluation of recurrent, unexplained near-syncope and light-headedness
- Evaluation of recurrent syncope in the setting of autonomic failure or peripheral neuropathies
- Evaluation of postexertional syncope when an episode cannot be reproduced by exercise testing

Overview of Common Syncopal Situations and their Management

- Neurocardiogenic Syncope
- Orthostatic hypotension
- Cardiac Causes

Neurocardiogenic Syncope

- A syndrome in which “triggering of a neural reflex results in a usually self-limited episode of systemic hypotension characterized by both bradycardia (asystole or relative bradycardia) and peripheral vasodilation.”
 - “Vasodepressor” (vasodilation)
 - “Cardioinhibitory” (bradycardia)
 - “Mixed” (both vasodilation and bradycardia)
- Neurocardiogenic syncope is caused by an abnormal or exaggerated autonomic response to various stimuli, of which the most common are standing and emotion.

Neurocardiogenic Syncope

Differential Diagnosis

- Syncope after cough, micturition, and defecation suggests situational syncope
- Syncope with throat or facial pain (CN IX or VII neuralgia) suggests neurally mediated syncope with neuralgia
- Syncope with pain, fear, or noxious stimuli suggests neurocardiogenic syncope
- Syncope with rotation or turning of the head or neck pressure from shaving, tight collars or neckwear or carotid massage or tumor compression suggests carotid sinus syncope

Rational Treatment of Neurocardiogenic Syncope

- Avoid predisposing situations (dehydration, stress, alcohol consumption, warm environments, tight clothing)
- Management of anxiety
- Development of coping skills (coping with precipitating conditions)
- Reassurance that this is a benign condition
- Recognition of presyncopal symptoms

Physical Countermeasures in Neurocardiogenic Syncope

- This study was performed in 9 patients with neurogenic orthostatic hypotension, and Valsalva maneuver was avoided; maneuvers continued approximately 45 seconds; biofeedback and 45-minute training sessions were used
- Leg crossing - When standing, cross the right foot over the left and contract the leg musculature
- Toe raise, marching, squat, isometric quadriceps exercise
- Blood pressure increment approximately 20 mmHg
- Patients preferred leg crossing, thigh contraction, toe raise, and squat

Physical Countermeasures in Neurocardiogenic Syncope

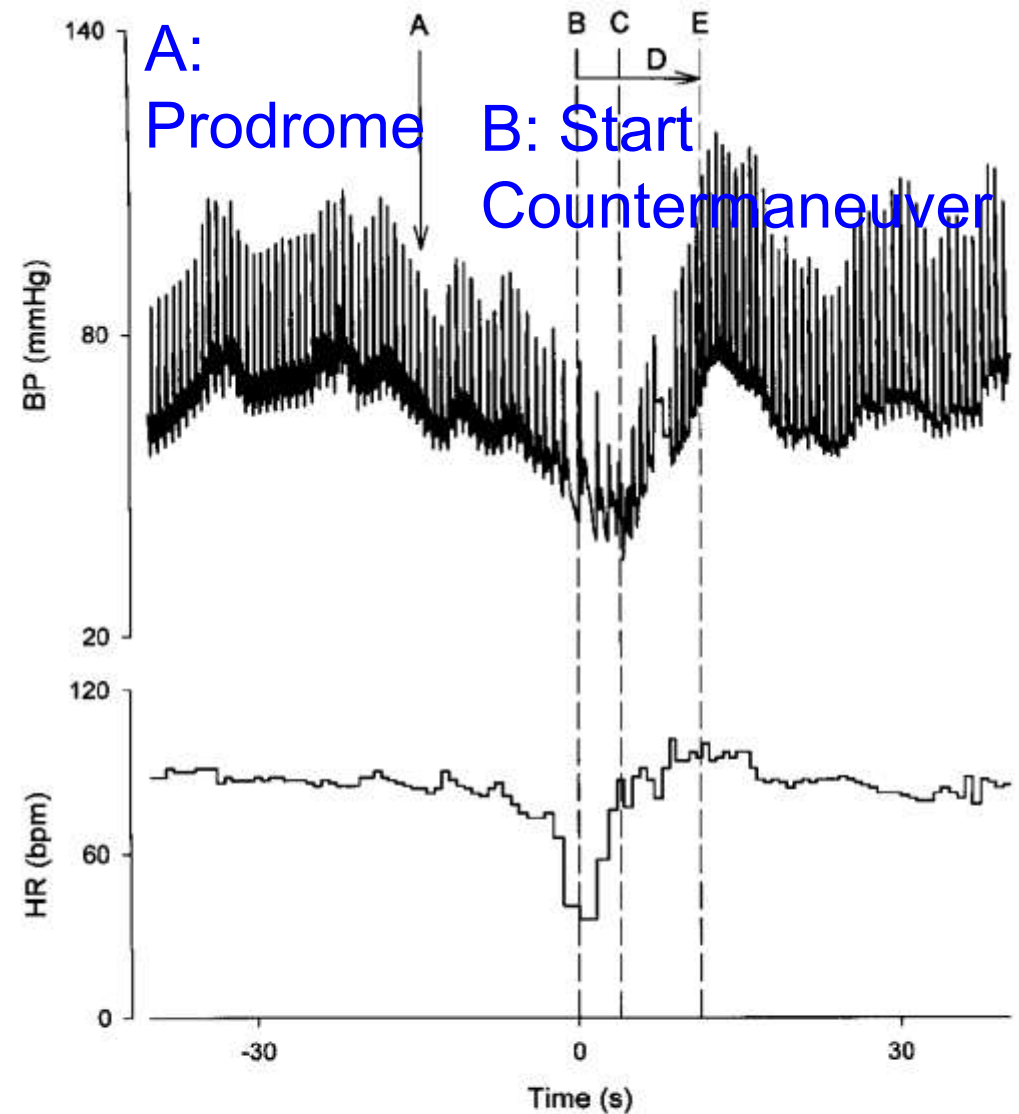
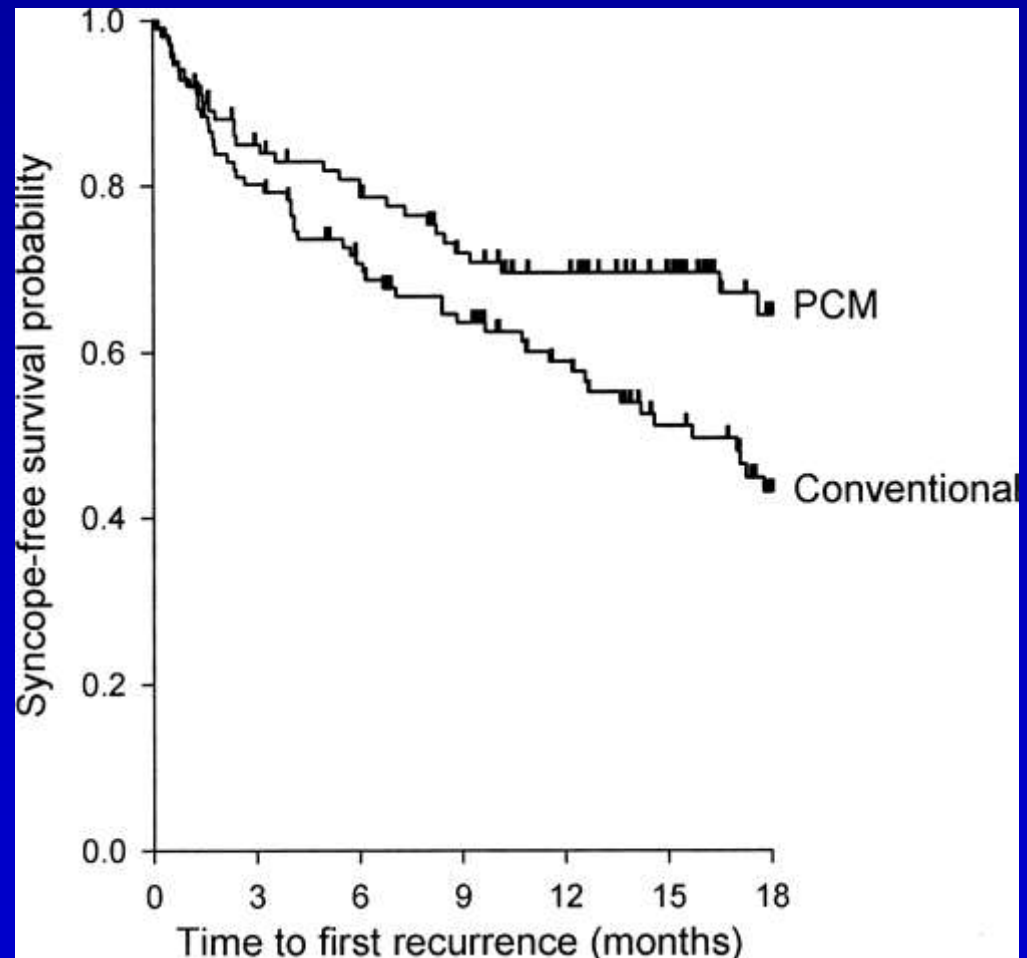


Figure 2. Original tracing in a 34-year-old male subject during a vasovagal episode while tilted head-up. A, Onset of prodromal symptoms. B, Start of physical counter-maneuver. C, Blood pressure nadir. D, Latency between start of physical counter-maneuver and disappearance of prodromal symptoms. E, Stabilization of blood pressure.

Krediet CPT et al.
Circulation 2002;106;1684-1689.

Physical Countermeasures in Neurocardiogenic Syncope

- Randomized 14-month follow-up between 110 pts with conventional therapy and 99 pts trained on physical countermeasures (included biofeedback)



Pharmacologic Treatment of Neurocardiogenic Syncope: (No Agent is Recommended)

- Beta-blockers are rational but ineffective in randomized trials
- Alpha-agonists (midodrine) have been shown to be effective
- Selective serotonin reuptake inhibitors may be effective (1 month, paroxetine in 68 pts)
- Fludrocortisone may be effective
- Disopyramide is not first choice
- Other anticholinergics possible

Orthostatic Hypotension: Effects of Aging

- Less HR acceleration (lower parasympathetic tone) and α_1 -adrenergic vasoconstriction
- Less renal responsiveness to dehydration (with aging: renin, angiotensin and aldosterone lower and natriuretic peptides higher)
- Lower myocardial chamber compliance means greater dependence on ventricular preload and lower tolerance of volume depletion

Orthostatic Hypotension

- Definition: SBP fall > 20 mmHg or DBP fall > 10 mmHg or symptoms of cerebral hypoperfusion within 1-3 min of standing
- If the HR increases by 20, it is probably volume depletion, if <10 it is probably baroreflex impairment (autonomic dysfunction)

Drugs that Cause Orthostatic Hypotension

- Alpha-blockers
- Antipsychotics
- Antihypertensives
- Beta-blockers
- Bromocriptine
- Diuretics
- Levadopa
- Marijuana
- Narcotics and sedatives
- Phosphodiesterase – 5 inhibitors
- Tricyclics
- Vasodilators

Treatment of Orthostatic Hypotension

- Adjust offending medications
- Arise slowly
- Avoid straining, coughing, prolonged standing in hot weather
- Physical countermeasures
- Raise head of bed 10-20 degrees
- Small meal and coffee in the morning
- Elastic waist high stocking
- Liberalize salt and water intake
- Exercise (swimming, recumbent bike, rowing)

Drug Treatment of Orthostatic Hypotension

Drug	Dose (mg)	Contra-indication	Side Effects
Fludro-cortisone	Initial 0.1/d Max 1/d	Hyper-sensitivity	Supine htn, hypo-kalemia, HF, HA
Mido-drine	Initial 2.5 tid Max 10 tid	Sev OHD, urin retention, ARF	Supine htn, paresth, pruritis, piloerection,
Ibuprofen	400-800 tid	Sens to NSAID, bleeding, CRI	GI intol, bleeding, HA, dizziness, CRI
Caffeine	100-250/da	Hyper-sensitivity	GI irrit, insom, agit, nervousness
E-poietin*	25-75 U/Kg	Uncontr htn	Stroke, MI, Htn

Syncope due to Bradyarrhythmias

Sinus node dysfunction

AV node dysfunction

- Secondary
 - to a reversible cause, remove the cause and observe for improvement
 - to necessary therapy (e.g., beta-blocker for brady-tachy syndrome or for heart failure or angina), pacemaker is often the preferred option
- Primary (sick sinus syndrome or AV block), pacemaker is generally the preferred option

Pacemaker Therapy in Neurocardiogenic Syncope

- Not the usual initial treatment
- Dual chamber pacemaker may relieve symptoms if there is a large cardioinhibitory (bradycardia) component

Syncope due to Tachyarrhythmias

There are many types of tachycardias and many options for therapy, cardiology is often helpful

- Supraventricular tachycardias (less likely to cause syncope) may respond to a radiofrequency ablation procedure (WPW, atrial flutter, others) and pharmacologic therapy
- Most ventricular tachycardias may require a combination of ICD and pharmacologic therapy
- Some ventricular tachycardias occur in a structurally normal heart and may respond to a beta blocker or radiofrequency ablation

Syncope due to Cardiac Obstructive Lesion

- Severe aortic stenosis – poor prognosis without surgery, recommend aortic valve replacement (PS, MS also)
- Pulmonary embolism – recommend anticoagulation, consider fibrinolytic therapy or embolectomy for massive or submassive embolism
- Pulmonary hypertension

Syncope and Neurology

- Asystole occurring in a seizure is rare but not impossible, less rare in temporal seizures
- SUDEP: sudden unexpected death in epilepsy
- Treatment/prevention – meticulous control of seizures; occasionally pacemaker

Syncope: Indications for Hospitalization

For Diagnosis

- Suspected or known significant heart disease
- Suspicious ECG abnormalities
- Syncope during exercise
- Syncope with severe injury
- Family history of SCD
- Palpitations, frequent symptoms, supine syncope
- High suspicion for cardiac syncope

For Treatment

- Cardiac arrhythmias causing syncope
- Syncope due to ischemia
- Syncope due to structural cardiac or cardiopulmonary disease
- Neurocardiogenic syncope requiring pacemaker

ESC Guideline

Hospitalization for Syncope

TABLE 1. Emergency Department Risk Stratification of Patients With Syncope of Unknown Cause

High-Risk Group	Intermediate-Risk Group	Low-Risk Group
Chest pain compatible with acute coronary syndrome	Age ≥ 50 y	Age < 50 y
Signs of congestive heart failure	With previous history of:	With no previous history of:
Moderate/severe valvular disease	Coronary artery disease	Cardiovascular disease
History of ventricular arrhythmias	Myocardial infarction	Symptoms consistent with reflex-mediated or vasovagal syncope
ECG/cardiac monitor findings of ischemia	Congestive heart failure	Normal cardiovascular examination
Prolonged QTc (> 500 ms)	Cardiomyopathy without active symptoms or signs on cardiac medications	Normal ECG findings
Trifascicular block or pauses between 2 and 3 seconds	Bundle-branch block or Q wave without acute changes on ECG	
Persistent sinus bradycardia between 40 and 60 bpm	Family history of premature (< 50 y), unexplained sudden death	
Atrial fibrillation and nonsustained ventricular tachycardia without symptoms	Symptoms not consistent with a reflex-mediated or vasovagal cause	
Cardiac devices (pacemaker or defibrillator) with dysfunction	Cardiac devices without evidence of dysfunction	
	Physician's judgment that suspicion of cardiac syncope is reasonable	

Recommended

Consideration

Not recommended

Shen WK et al. Circulation. 2004;110:3636-45.

(SEEDS)

Hospitalization for Syncope

- Evidence for heart failure
- Evidence for structural heart disease
- High risk features
 - Older age and comorbidities
 - ECG ischemia, conduction abnormalities, or dysrhythmias
 - Hematocrit <30 (if obtained)

For diagnosis

Strongly recommended

- Suspected or known significant heart disease
- Electrocardiographic abnormalities suggestive of arrhythmic syncope
- Syncope occurring during exercise
- Syncope causing severe injury
- Strong family history of sudden death

Occasionally may need to be admitted

- Patients with or without heart disease but with:
 - sudden onset of palpitations shortly before syncope
 - syncope in supine position
 - worrisome family history
 - significant physical injury
- Patients with minimal or mild heart disease when there is high suspicion for cardiac syncope
- Suspected pacemaker or implantable cardioverter-defibrillator problem

For treatment

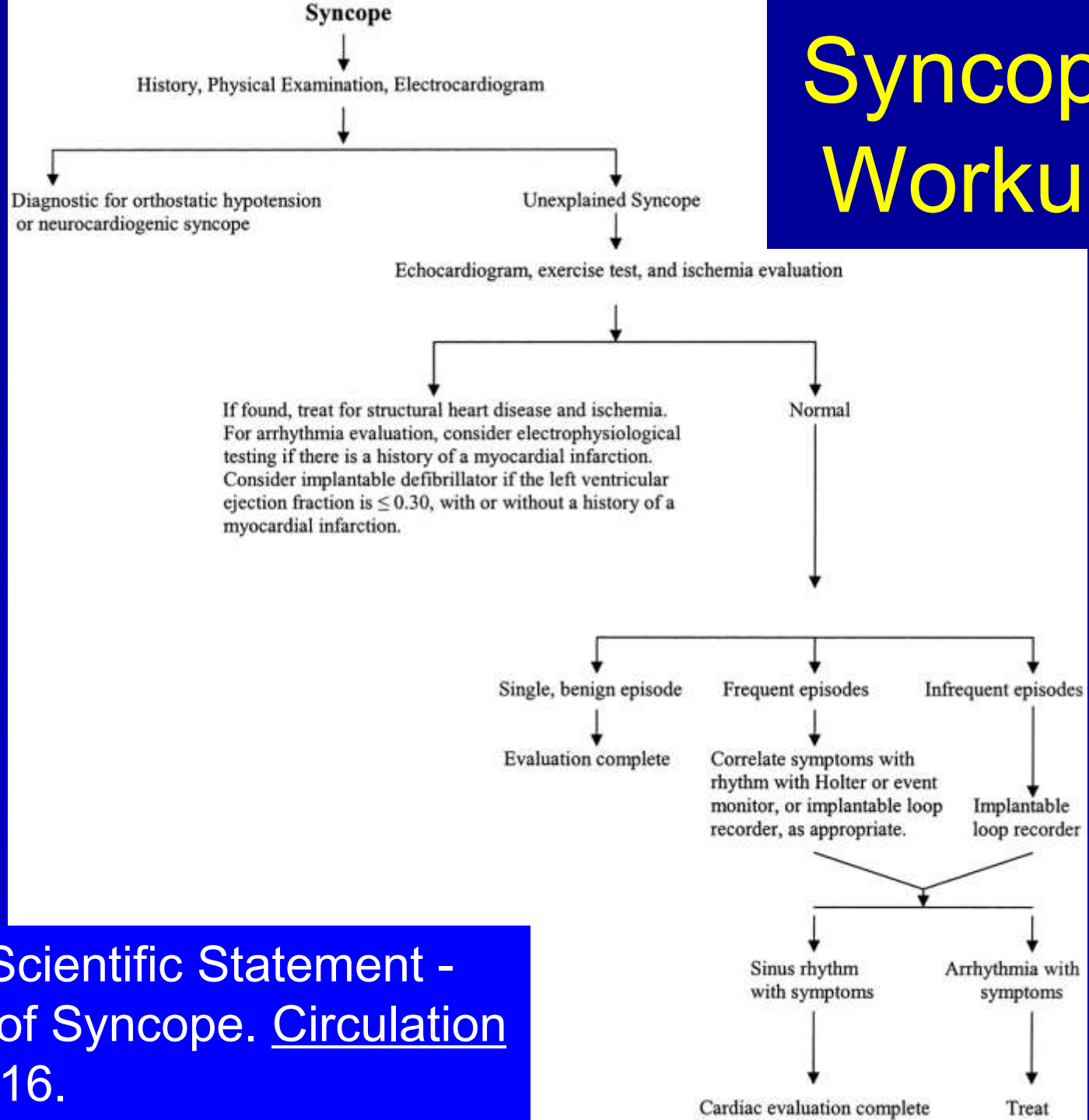
- Cardiac arrhythmias as cause of syncope
- Syncope due to cardiac ischaemia
- Syncope secondary to the structural cardiac or cardiopulmonary diseases
- Stroke or focal neurologic disorders
- Cardioinhibitory neurally mediated syncope when a pacemaker implantation is planned

Hospitalization for Syncope

From 2004
ESC Guideline

Brignole M et al.
J Am Coll
Cardiol.
2008;51:284-7.

Syncope Workup



AHA/ACC Scientific Statement -
Evaluation of Syncope. Circulation
2006;113:316.

Italian Application of Guidelines in Urgent Care Setting

- Used a highly structured computerized algorithm based on the 2004 European guidelines; applied to eleven Italian hospitals over a 31-da period (541 pts, 1% of ER visits)
- Hospitals were equipped with full evaluation capabilities including tilt-table, beat-to-beat noninvasive BP, and autonomic function testing as well as the usual facilities
- Computer software suggested diagnoses and recommended tests; an expert was required to provide recommendations to follow the diagnostic workup in 150 (32%) patients

Italian Application of Guidelines – Patient Characteristics

Total patients attending the emergency room	51 049
Total patients with LOC attending the emergency room (%)	541 (1.1)
Patients with LOC per hospital (mean \pm SD)	49 \pm 12
Time between LOC and arrival at emergency room <6 h (%)	385 (71)
Median age (interquartile range)	71 (48–81)
Male gender (%)	289 (53)
Normal electrocardiogram (%)	327 (60)
No structural heart disease (%)	341 (63)
Comorbidities	
Hypertension (%)	221 (41)
Any neurological disease (%)	110 (20)
Diabetes (%)	47 (9)
Any therapy at the time of LOC (%)	272 (50)
History of LOC: first episode (%)	313 (58)
Recurrent LOCs: median number per patient (interquartile range)	2 (2–4)
Interval between first and last episode – year (interquartile range)	3 (1–8)
History of pre-syncope (%)	109 (20)
Injuries related to fainting (%)	138 (25)
Major injuries (fractures, brain concussion) (%)	60 (11)
Minor injuries (bruises, etc.) (%)	78 (14)
No warning at the onset of the attack (%)	163 (30)
Circumstances just prior to the attack ^a	
Standing (%)	363 (67)
Rest (no activity) (%)	286 (53)
Any pre-disposing factors (%)	474 (87)

Brignole M. et al. Eur Heart J. 2006;27:76-82.

Italian Application of Guidelines – Patient Flow

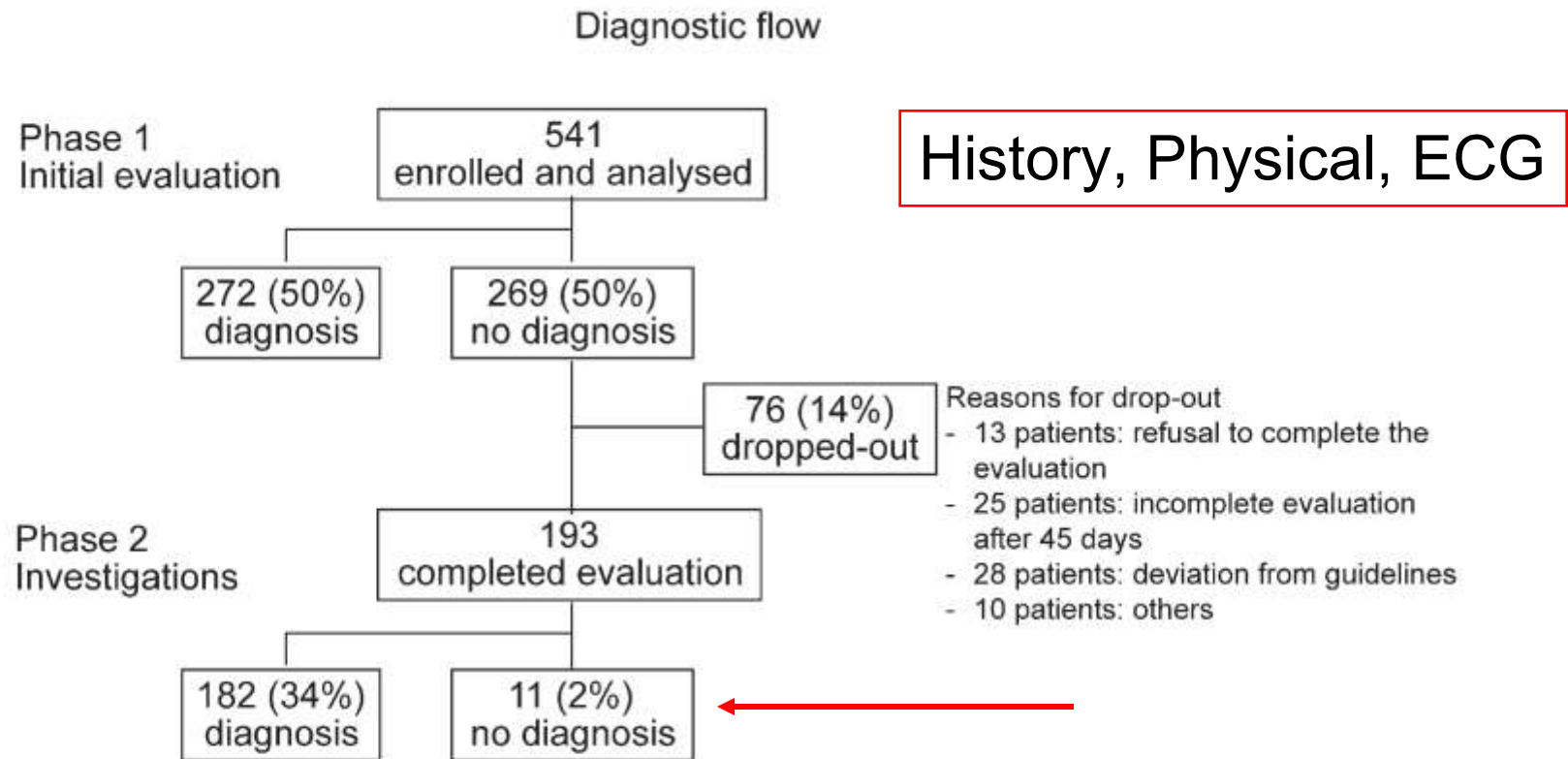


Figure 1 Diagnostic flow of 541 patients affected by transient loss of consciousness which, on initial evaluation, was attributed to a syncopal condition or because a syncopal condition could not be excluded (non-syncopal loss of consciousness). There were several reasons for drop-out in 76 patients. The most frequent were: some patients decided to leave the emergency room against the physician's intention; some patients could not complete the evaluation within 45 days because of dominant comorbidities or severe trauma secondary to syncope; in some cases, the physician in charge refused to follow the recommendations of the guidelines and/or the suggestions of the syncope expert; and finally, there were few cases of incorrect insertion in the database.

Results of Evaluation in Syncope

Cause	Initial Eval	More Tests	Total	%
Neurally mediated	202	107	309	66
Orthostatic Hypotension	36	10	46	10
Cardiac Arrhythmias	30	23	53	11
Structural Disease	4	17	21	5
Cerebrovascular Dz	0	0	0	0
Unknown			11	2
Non-Syncope		25	25	6
Total (541, 76 dropped out)	272	182	465	100

Brignole M. et al. Eur Heart J. 2006;27:76-82.

Details of Results in Syncope

Table 2 Causes of loss of consciousness in 465 patients (according to the ESC classification)

Causes of syncopes	Initial evaluation ^a	Investigations ^b	Total
Neurally mediated (reflex) (%)	202 (43)	107 (23)	309 (66)
Vasovagal syncope	101 (22)	89 (19)	190 (41)
Classical form: fear, pain, emotion, instrumentation	82 (18)	–	
Classical form: prolonged standing and typical prodromal symptoms	19 (4)	–	
Non-classical form: tilt-positive	–	38 (8)	
Likely vasovagal (non-classical form) after exclusion of other causes and absence of heart disease	–	51 (11)	
Carotid sinus syncope	0 (0)	18 (4)	18 (4)
Situational syncope ^c	71 (15)	–	71 (15)
Single/rare syncopes, no heart disease	30 (6)	–	30 (6)
Orthostatic hypotension (%)	36 (8)	10 (2)	46 (10)
Cardiac arrhythmias as primary cause (%)	30 (6)	23 (5)	53 (11)
Sinus node dysfunction (including bradycardia/tachycardia syndrome) (%)	5 (1)	7 (1)	12 (3)
Atrioventricular conduction system disease (%)	15 (3)	8 (2)	23 (5)
Paroxysmal supraventricular tachycardias (%)	8 (2)	3 (1)	11 (2)
Paroxysmal ventricular tachycardias (%)	2 (0)	5 (1)	7 (1)
Structural cardiac or cardiopulmonary disease ^d (%)	4 (1)	17 (4)	21 (5)
Cerebrovascular (%)	0 (0)	0 (0)	0 (0)
Unknown (%)	–	–	11 (2)
Causes of non-syncopal attacks (commonly misdiagnosed as syncope) (%)	–	25 (6)	25 (6)
Metabolic disorders (hypoglycaemia)	–	1 (0)	1 (0)
Epilepsy	–	8 (2)	8 (2)
Intoxication	–	2 (0)	2 (0)
Vertebro-basilar transient ischaemic attack – no. (%)	–	4 (1)	4 (1)
Falls – no. (%)	–	6 (1)	6 (1)
Psychogenic pseudo-syncope (%)	–	4 (1)	4 (1)

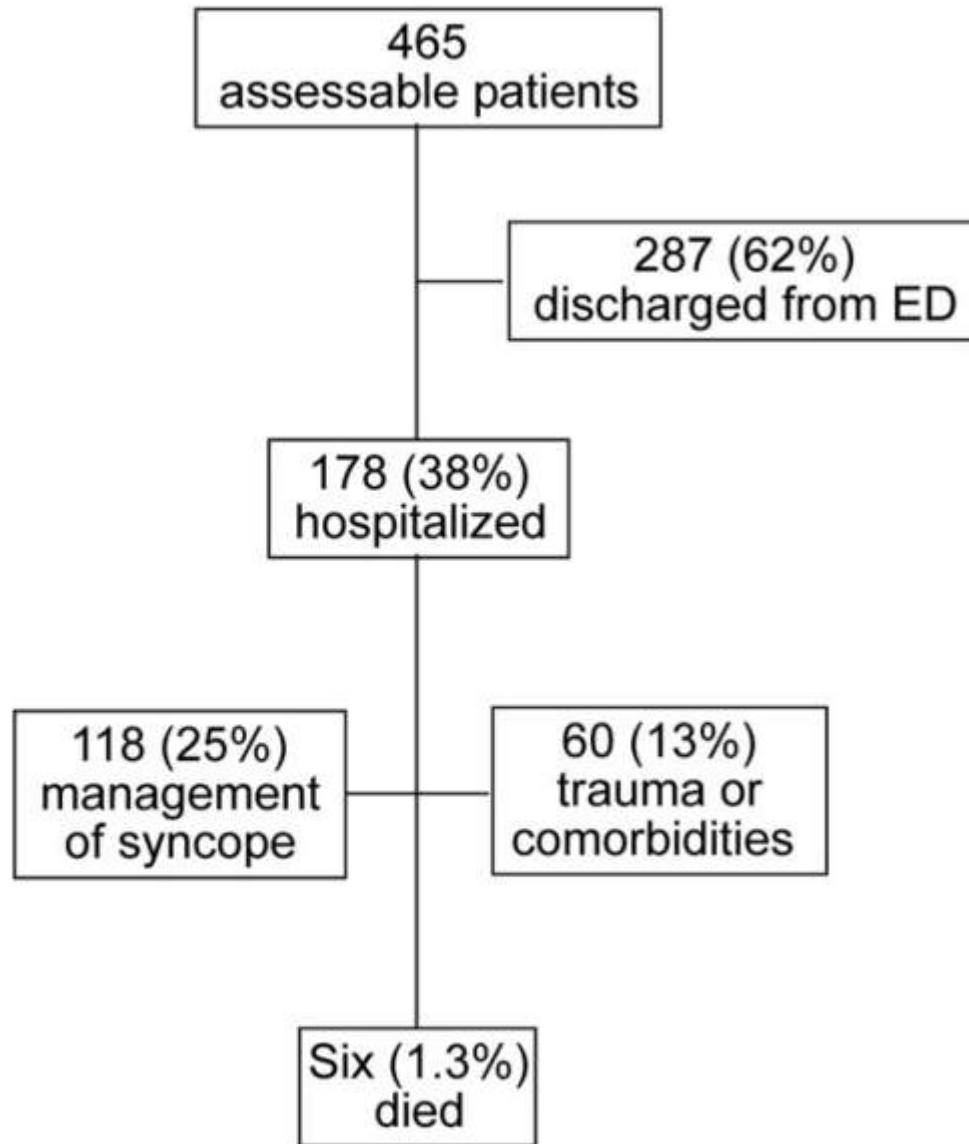
Testing Use in Syncope Evaluation

Table 3 Tests: diagnostic yield in 465 patients

	Diagnostic purposes		Trauma or comorbidities or routine	Total
	Appropriate ^a	Of which diagnostic ^a		
Electrocardiography (%)	465 (100)	34 (7)	0 (0)	465 (100)
Basic laboratory tests (%)	52 (11)	21 (40) ^b	114 (25)	166 (36)
Echocardiography (%)	49 (11)	5 (10) ^c	25 (5)	74 (16)
Tilt testing (%)	76 (16)	46 (61)	2 (0)	78 (17)
Carotid sinus massage (%)	65 (14)	18 (28)	4 (1)	69 (15)
24 h blood pressure monitoring (%)	1 (0)	1 (100)	0 (0)	1 (0)
ATP test (%)	1 (0)	0 (0)	0 (0)	1 (0)
In-hospital ECG monitoring (%)	21 (5)	13 (62)	0 (0)	21 (5)
24 h Holter monitoring (%)	12 (3)	3 (25)	8 (1)	20 (4)
External loop recorder (%)	4 (1)	2 (50)	0 (0)	4 (1)
Exercise test (%)	10 (2)	3 (30) ^d	5 (1)	15 (3)
Electrophysiological study (%)	15 (3)	5 (33) ^e	1 (0)	16 (3)
Coronary angiography (%)	8 (2)	5 (62)	1 (0)	9 (2)
Pulmonary computed tomography/Scintigraphy (%)	5 (1)	4 (80)	0 (0)	5 (1)
Electroencephalography (%)	16 (3)	5 (31)	7 (2)	23 (5)
Brain computed tomography (%)	16 (3)	3 (19)	51 (11)	67 (14)
Brain magnetic resonance imaging (%)	6 (1)	2 (33)	3 (1)	9 (2)
Carotid echo-Doppler (%)	0 (0)	0 (0)	26 (6)	26 (6)
Chest X-ray (%)	0 (0)	0 (0)	51 (11)	51 (11)
Other X-ray (%)	0 (0)	0 (0)	22 (5)	22 (5)
Abdominal ultrasound examination (%)	0 (0)	0 (0)	11 (2)	11 (2)
Gastroscopy (%)	1 (0)	1 (100) ^f	2 (1)	3 (1)
Other echographies (%)	0 (0)	0 (0)	3 (1)	3 (1)
Total number of tests	823	171	346	1156
Mean no. of tests per patient (\pm SD)	1.8 \pm 1.2			2.5 \pm 1.8

Brignole M. et al. Eur Heart J. 2006;27:76-82.

Results of Guideline-Based Management



Mechanism of discharge

- 160 patients directly from ER
- 65 patients after a short period of observation in ED
- 62 patients referred to syncope clinic

Reason for hospitalization

- 58 patients significant heart disease
- 35 patients ECG abnormalities (arrhythmic)
- Seven patients frequent recurrent episodes
- Four patients syncope occurring during exercise
- Two patients palpitations shortly before syncope
- 50 patients therapy
 - Arrhythmia (no. 31)
 - Myocardial ischaemia (no. 6)
 - Structural cardiac (no. 9)
 - Stroke (no. 4)
- 13 patients trauma
- 47 patients comorbidities

Cause of death

- Two patients heart failure
- One patient acute myocardial infarction
- One patient pulmonary embolism
- One patient rupture of aneurism
- One patient stroke

Summary

- Evaluation of syncope: History is the main diagnostic tool
- Management of syncope
 - Hospitalize the high risk patient
 - Reassure and educate the low risk patient
 - Avoidance of precipitants
 - Techniques of physical countermeasures