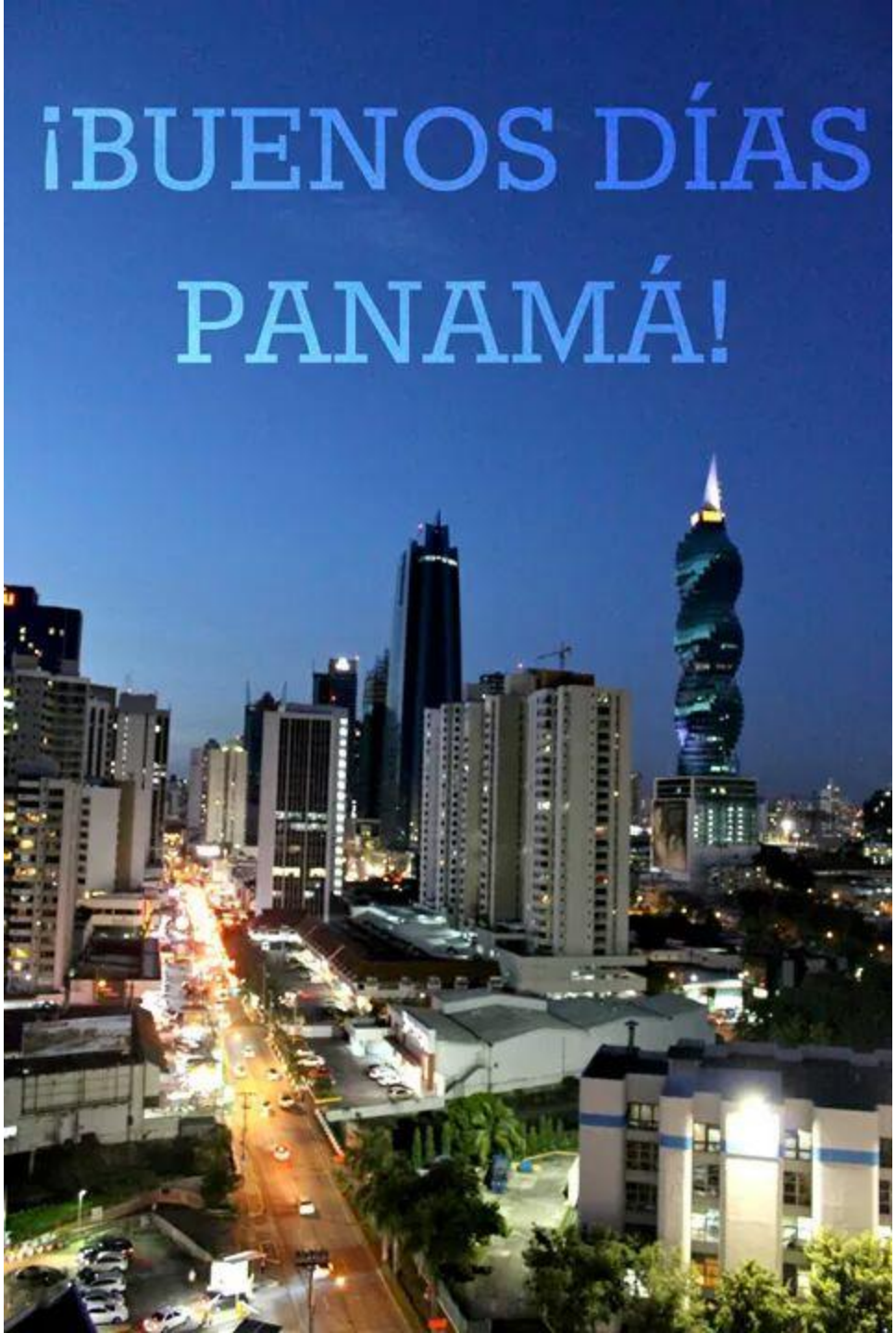
Síncope

Preparado por el Dr. Ricardo Pizarro B. para el Personal Médico y Odontológico de la Policlínica Dr. Carlos N. Brin. VII - 2017.

¡BUENOS DÍAS PANAMÁ!



Definición

Puesta al día: Arritmias (VIII)

Síncope

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RESUMEN

El síncope, definido como una pérdida transitoria de conciencia que cursa con recuperación espontánea y sin secuelas que se debe a una hipoperfusión cerebral general y transitoria, es un cuadro clínico muy prevalente. Esta definición permite diferenciar el síncope de otras entidades que cursan con pérdida de conciencia transitoria, real o aparente, en las que el mecanismo no es una hipoperfusión cerebral, como la epilepsia, las caídas accidentales o el seudosíncope psiquiátrico. Se revisa la clasificación etiológica del síncope, con especial hincapié en que el síncope reflejo es el más frecuente y tiene buen pronóstico, mientras que el síncope cardiogénico aumenta con la edad y tiene peor pronóstico. Se hace una revisión

Definición

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STATE-OF-THE-ART PAPER

New Concepts in the Assessment of Syncope

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Lavagna, Italy; and Salt Lake City, Utah

Syncope and Its Context

Definition. Transient loss of consciousness (TLOC) or faint are generic terms that encompass all disorders characterized by transient, self-limited, nontraumatic loss of consciousness. The causes of TLOC include syncope, epileptic seizures, psychogenic, and other rare miscellaneous causes. What differentiates syncope from the other forms of TLOC is its unique pathophysiology (i.e., transient global cerebral hypoperfusion due to low peripheral resistances and/or low cardiac output) (1).

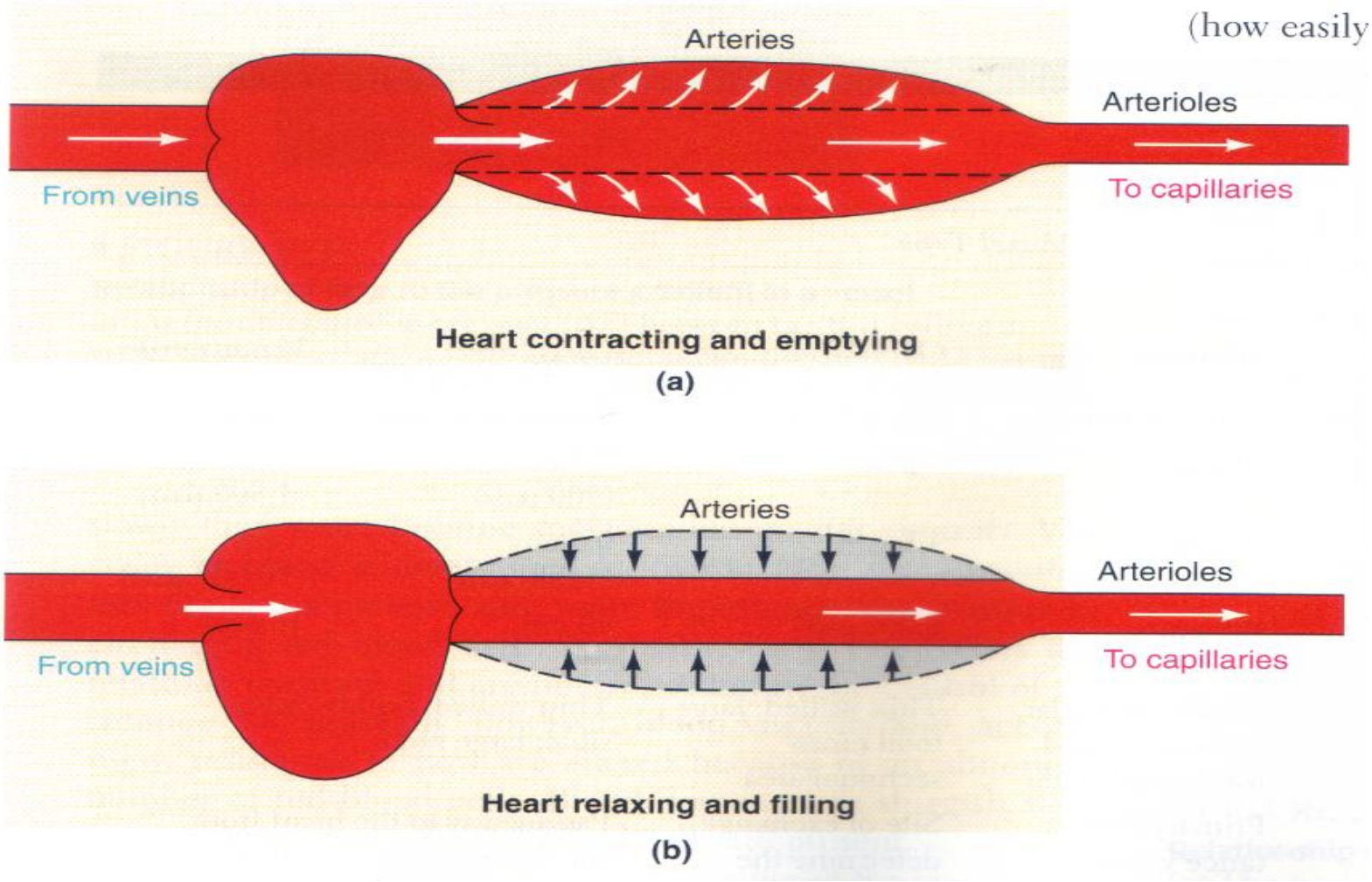
Presión Arterial

- **Presión Arterial = Gasto Cardiaco (GC) x Resistencia Periférica Total (RPT).**

- **¿Qué sucede si Gasto cardiaco aumenta o disminuye y si RPT aumenta o disminuye?**

Arterias como reservorios de energía

(how easily



Regulación de la Presión Arterial

1. **Mecanismos Nerviosos**
2. Mecanismos Renales

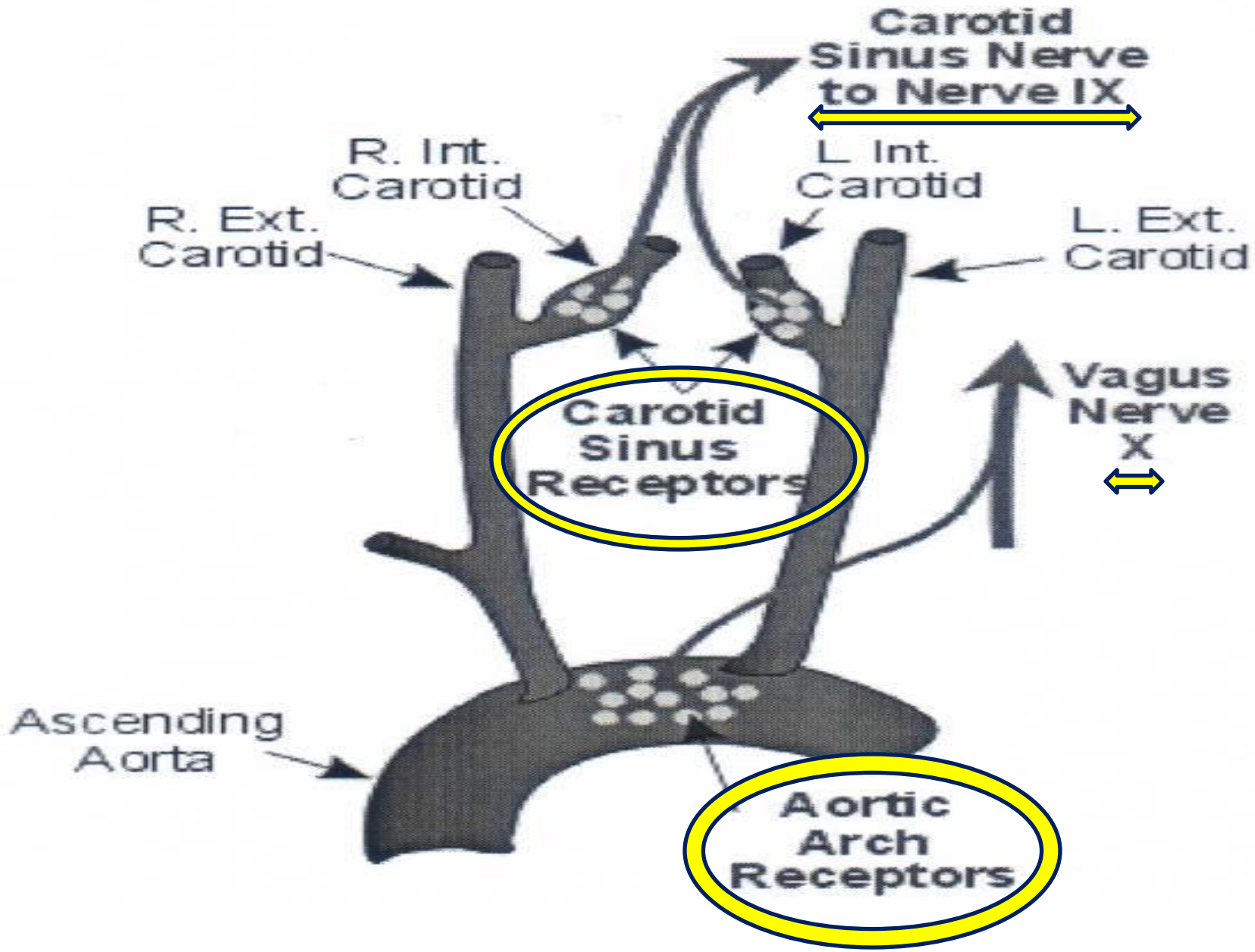
Mecanismos nerviosos de regulación de PA

1. Reflejo barororeceptor de alta presión
2. Reflejo quimioreceptor
3. Reflejo baroreceptor de baja presión
4. Respuesta Isquémica del SNC
5. Otros respuestas: sensoriales, viscerales, corticales, hipotalámicas

Reflejo baroreceptor de alta presión

Receptores:

1) Seno Carotídeo
2) Seno Aórtico
3) Mecanoreceptores: responden al estiramiento


Carotid
Sinus Nerve
to Nerve IX
R. Int.
Carotid
L. Int.
Carotid
R. Ext.
Carotid
L. Ext.
Carotid
Vagus
Nerve
X
Carotid
Sinus
Receptors
Ascending
Aorta
Aortic
Arch
Receptors

Reflejo baroreceptor de alta presión

Vía aferente:

Desde el seno carotídeo: **el IX par**

Desde el Seno Aórtico: **el X par**

Reflejo baroreceptor de alta presión

Vía eferente:

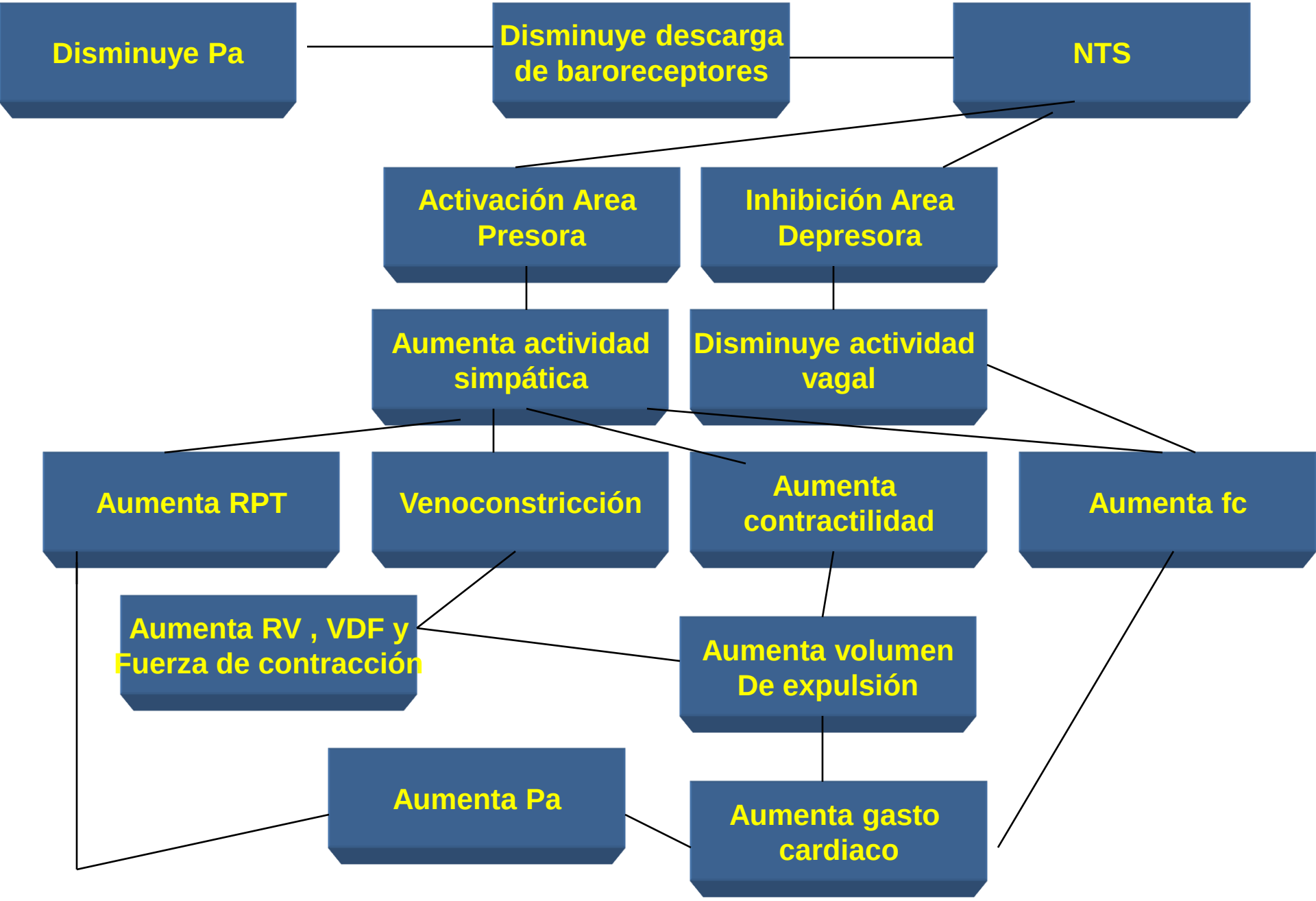
Simpático: C7 a L3

Nervio Vago

Reflejo baroreceptor de alta presión

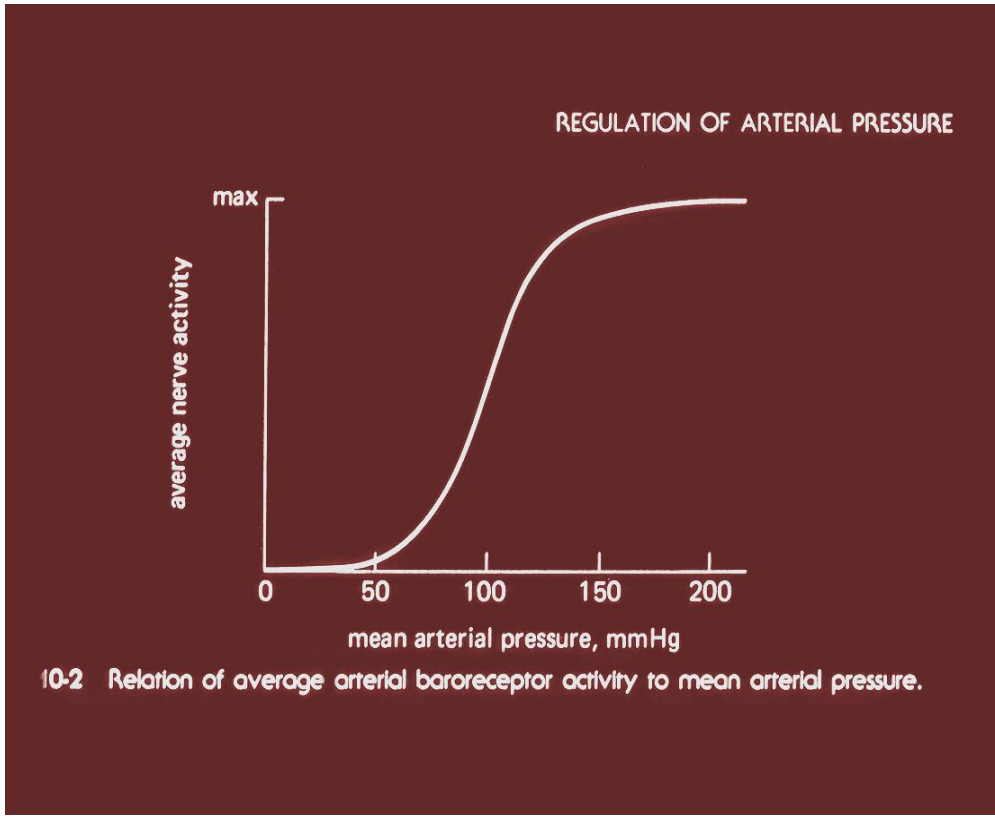
Efectores:

- **Corazón ($ß_1$)**
- **Vasos sanguíneos: arteriolas (α_1 y α_2) y venas**
- Médula suprarrenal
- Riñón


Disminuye Pa
Disminuye descarga de baroreceptores
NTS
Activación Area Presora
Inhibición Area Depresora
Aumenta actividad simpática
Disminuye actividad vagal
Aumenta RPT
Venoconstricción
Aumenta contractilidad
Aumenta fc
Aumenta RV , VDF y Fuerza de contracción
Aumenta volumen De expulsión
Aumenta Pa
Aumenta gasto cardiaco

Reflejo baroreceptor de alta presión

- Trabajan en el rango de 60 a 180 mmHg
- Se adaptan
- **Responden bien a cambios súbitos de presión**
- Su f de descarga cambia con el ciclo cardiaco



10-2 Relation of average arterial baroreceptor activity to mean arterial pressure.

Factores que determinan el valor del gasto cardiaco

2 FACTORES

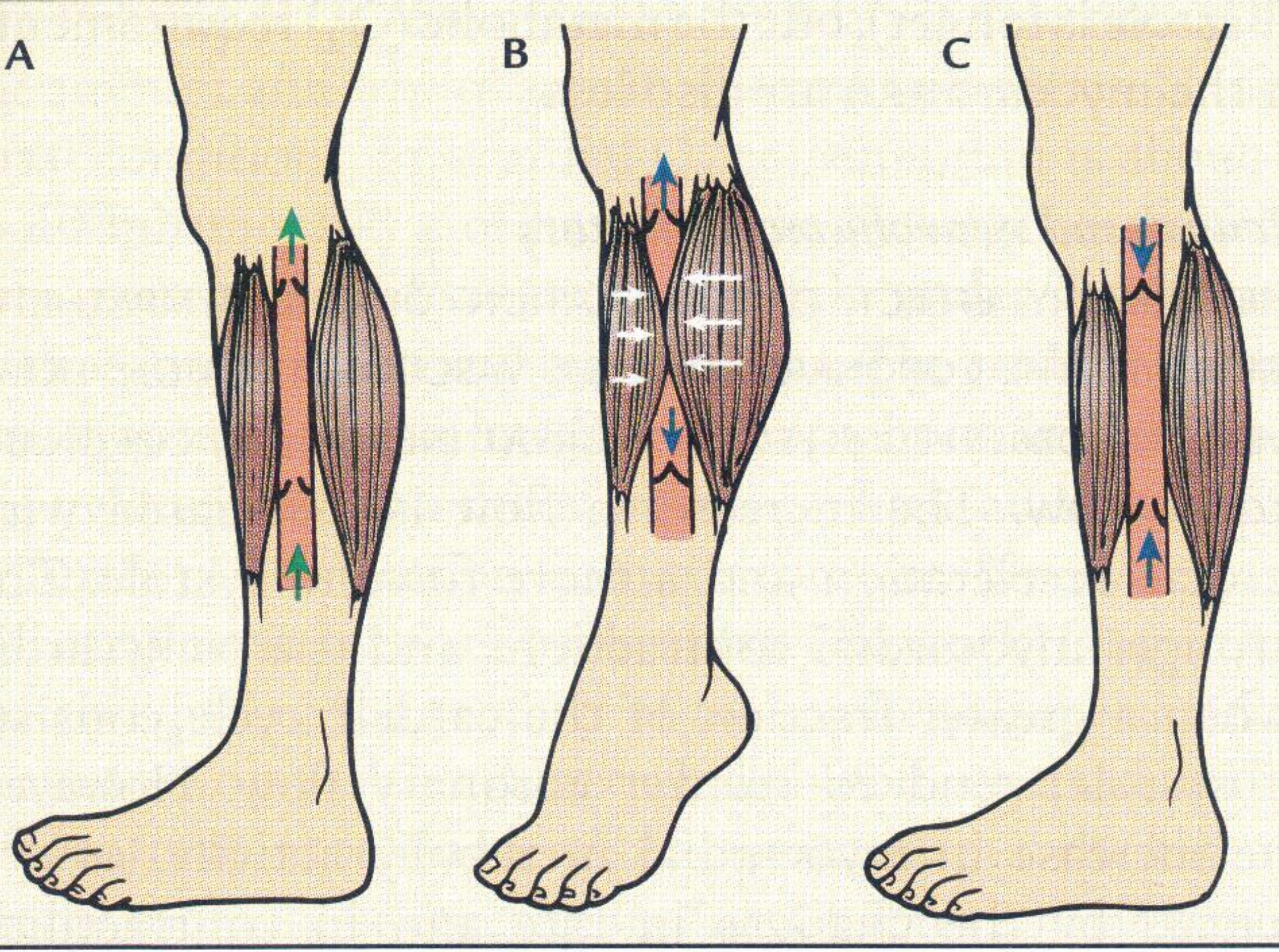
- **Volumen de expulsión**
- **Frecuencia cardiaca**

Gasto cardiaco=
Vln de expulsión x Frecuencia Cardiaca

Factores que afectan la contractilidad cardiaca

2 FACTORES

- **Descarga simpática**
- Otros factores que cambien el calcio intracelular


A
B
C

European Heart Journal (2009) **30**, 2631–2671
doi:10.1093/eurheartj/ehp298

Guidelines for the diagnosis and management of syncope (version 2009)

The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC)

Developed in collaboration with, European Heart Rhythm Association (EHRA)[1], Heart Failure Association (HFA)[2], and Heart Rhythm Society (HRS)[3]

The most relevant changes are listed here:

- An update of the classification of syncope in the larger framework of transient loss of consciousness (T-LOC).
- New data on epidemiology.
- A new diagnostic approach focusing on risk stratification of sudden cardiac death (SCD) and cardiovascular events after initial evaluation, including some recommendations for treatment in patients with unexplained syncope at high risk.
- Emphasis on the increasing role of a diagnostic strategy based on prolonged monitoring in contrast to the conventional strategy based on laboratory testing.
- An update of evidence-based therapy.

2017 ACC/AHA/HRS Guideline for the Evaluation and Management of Patients With Syncope

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society

Developed in Collaboration With the American College of Emergency Physicians and Society for Academic Emergency Medicine

Endorsed by the Pediatric and Congenital Electrophysiology Society

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CLASS (STRENGTH) OF RECOMMENDATION

CLASS I (STRONG) — **Benefit >>> Risk**

Suggested phrases for writing recommendations:
- Is recommended
- Is indicated/useful/effective/beneficial
- Should be performed/administered/other
- Comparative-Effectiveness Phrases†:
  - Treatment/strategy A is recommended/indicated in preference to treatment B
  - Treatment A should be chosen over treatment B

CLASS IIa (MODERATE) — **Benefit >> Risk**

Suggested phrases for writing recommendations:
- Is reasonable
- Can be useful/effective/beneficial
- Comparative-Effectiveness Phrases†:
  - Treatment/strategy A is probably recommended/indicated in preference to treatment B
  - It is reasonable to choose treatment A over treatment B

CLASS IIb (WEAK) — **Benefit ≥ Risk**

Suggested phrases for writing recommendations:
- May/might be reasonable
- May/might be considered
- Usefulness/effectiveness is unknown/unclear/uncertain or not well established

CLASS III: No Benefit (MODERATE) — **Benefit = Risk**
(Generally, LOE A or B use only)

Suggested phrases for writing recommendations:
- Is not recommended
- Is not indicated/useful/effective/beneficial
- Should not be performed/administered/other

CLASS III: Harm (STRONG) — **Risk > Benefit**

Suggested phrases for writing recommendations:
- Potentially harmful
- Causes harm
- Associated with excess morbidity/mortality
- Should not be performed/administered/other

LEVEL (QUALITY) OF EVIDENCE‡

LEVEL A
- High-quality evidence‡ from more than 1 RCT
- Meta-analyses of high-quality RCTs
- One or more RCTs corroborated by high-quality registry studies

LEVEL B-R (Randomized)
- Moderate-quality evidence‡ from 1 or more RCTs
- Meta-analyses of moderate-quality RCTs

LEVEL B-NR (Nonrandomized)
- Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies
- Meta-analyses of such studies

LEVEL C-LD (Limited Data)
- Randomized or nonrandomized observational or registry studies with limitations of design or execution
- Meta-analyses of such studies
- Physiological or mechanistic studies in human subjects

LEVEL C-EO (Expert Opinion)

Consensus of expert opinion based on clinical experience

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR I and IIa; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

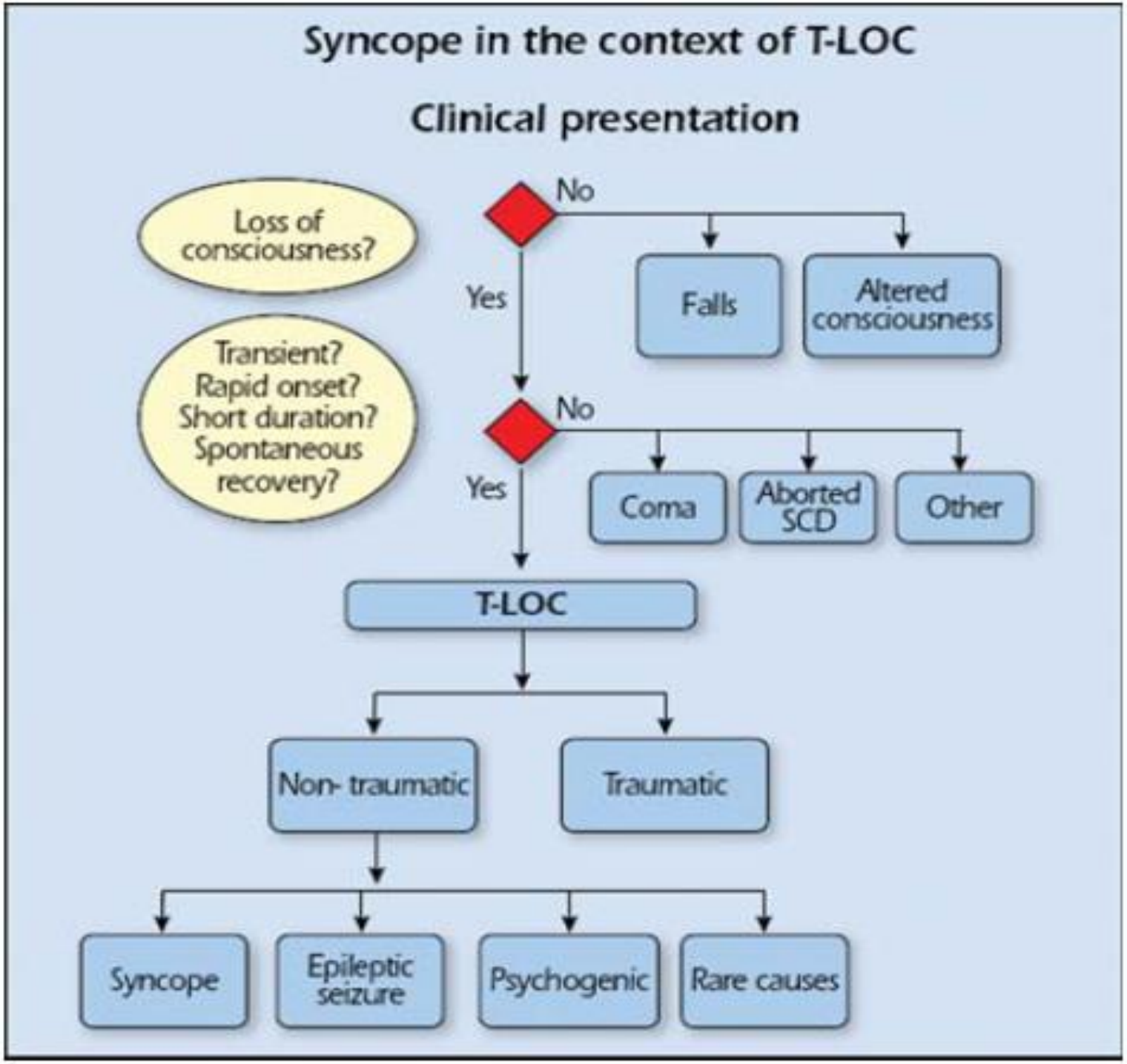

Syncope in the context of T-LOC
Clinical presentation
Loss of consciousness?
No
Falls
Altered consciousness
Yes
Transient?
Rapid onset?
Short duration?
Spontaneous recovery?
No
Coma
Aborted SCD
Other
Yes
T-LOC
Non- traumatic
Traumatic
Syncope
Epileptic seizure
Psychogenic
Rare causes

Figure 1. Syncope Initial Evaluation

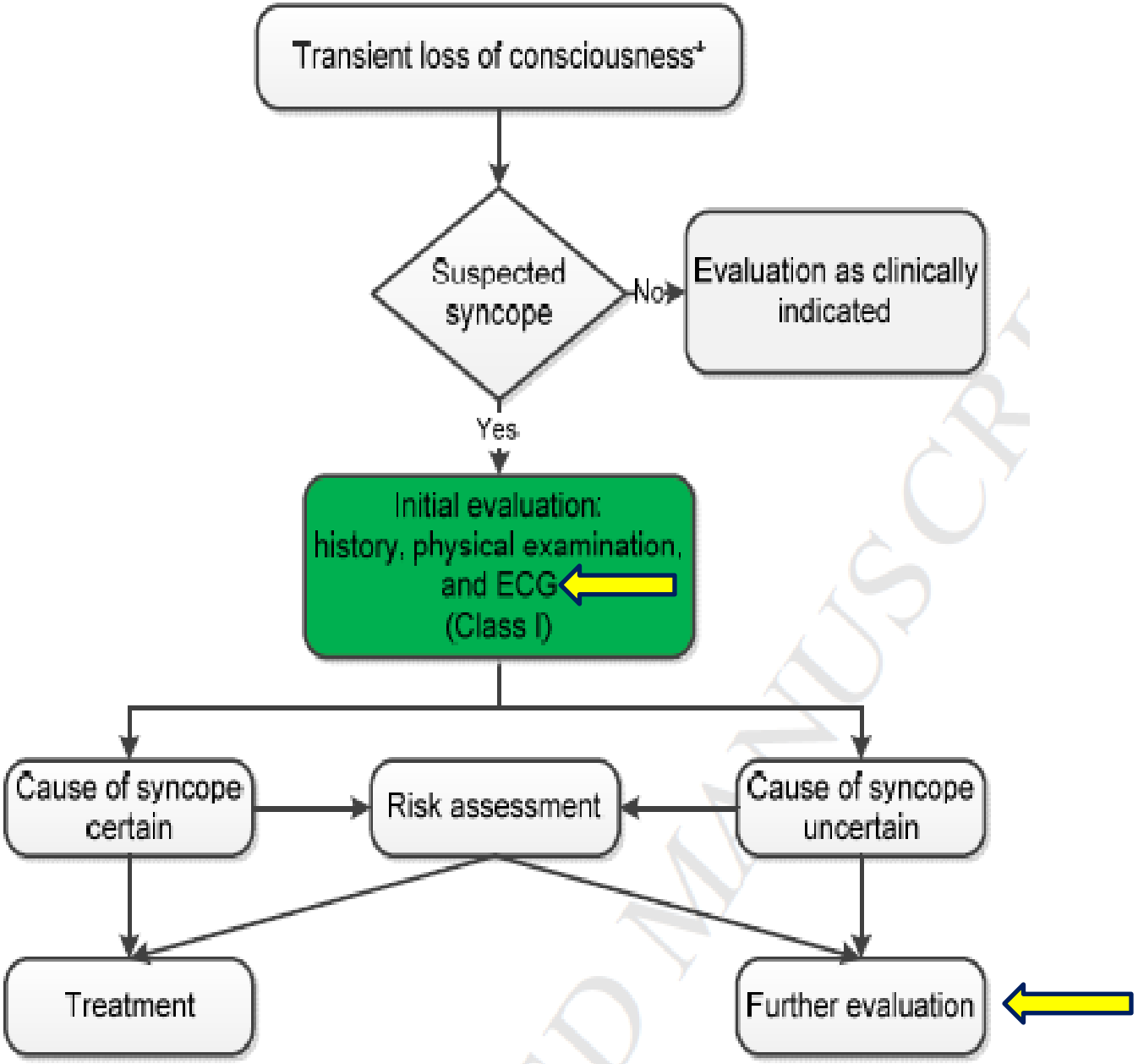


Table 3 Conditions incorrectly diagnosed as syncope

Disorders with partial or complete LOC but without global cerebral hypoperfusion
• Epilepsy
• Metabolic disorders including hypoglycaemia, hypoxia, hyperventilation with hypocapnia
• Intoxication
• Vertebrobasilar TIA
Disorders without impairment of consciousness
• Cataplexy
• Drop attacks
• Falls
• Functional (psychogenic pseudosyncope)
• TIA of carotid origin

LOC = loss of consciousness; TIA = transient ischaemic attack.

Cuadros clínicos que cursan con pérdida transitoria de conciencia (real o aparente) y pueden confundirse con síncope

Con pérdida de conciencia	Sin pérdida de conciencia
Epilepsia	Caídas inexplicadas (especialmente los ancianos)
Alteraciones metabólicas como hipoglucemia o hiperventilación con hipocapnia	*Drop attack*
Intoxicaciones	Seudosíncope psicogénico
Accidente isquémico transitorio vertebrobasilar	Cataplexia

Clasificación etiológica del síncope

Reflejo o neuromediado	
Vasovagal	Desencadenado por descarga adrenérgica Desencadenado por ortostatismo
Situacional	Relacionado con tos, estímulo gastrointestinal, micción, posprandial, tras ejercicio o risa
Síndrome de seno carotídeo	Con o sin estímulo aparente del seno carotídeo
Formas atípicas	Sin desencadenante aparente
Cardiogénico	
Bradiarritmia	Disfunción sinusal, bloqueo auriculoventricular
Taquiarritmia	Taquicardia supraventricular o ventricular
Cardiopatía estructural	Estenosis aórtica, miocardiopatía hipertrófica, mixoma auricular, taponamiento pericárdico, disección aórtica
Hipotensión ortostática	Disfunción autonómica primaria Secundaria a diabetes, amiloidosis, lesión espinal Inducido por fármacos (vasodilatadores diuréticos, antidepresivos) Hipovolemia (insuficiente ingesta de agua, hemorragia, diarrea)

Table 13 The value of history for distinguishing seizure from syncope (adapted from Hoefnagels *et al.*[5])

Clinical findings that suggest the diagnosis		
	Seizure likely	**Syncope likely**
Symptoms before the event	Aura (such as funny smell)	Nausea, vomiting, abdominal discomfort, feeling of cold sweating (neurally mediated) Lightheadedness, blurring of vision
Findings during loss of consciousness (as observed by an eyewitness)	Tonic–clonic movements are usually prolonged and their onset coincides with loss of consciousness Hemilateral clonic movement Clear automatisms such as chewing or lip smacking or frothing at the mouth (partial seizure) Tongue biting Blue face	Tonic–clonic movements are always of short duration (<15 s) and they start after the loss of consciousness
Symptoms after the event	Prolonged confusion Aching muscles	Usually of short duration Nausea, vomiting, pallor (neurally mediated)
Other clinical findings of less value for suspecting seizure (low specificity)		
Family history		
Timing of the event (night)		
'Pins and needles' before the event		
Incontinence after the event		
Injury after the event		
Headache after the event		
Sleepy after the event		
Nausea and abdominal discomfort		

Syncope due to orthostatic hypotension

Primary autonomic failure:

- *pure autonomic failure, multiple system atrophy, Parkinson's disease with autonomic failure, Lewy body dementia*

Secondary autonomic failure:

- *diabetes, amyloidosis, uraemia, spinal cord injuries*

Drug-induced orthostatic hypotension:

- *alcohol, vasodilators, diuretics, phenotiazines, antidepressants*

Volume depletion:

- *haemorrhage, diarrhoea, vomiting, etc*

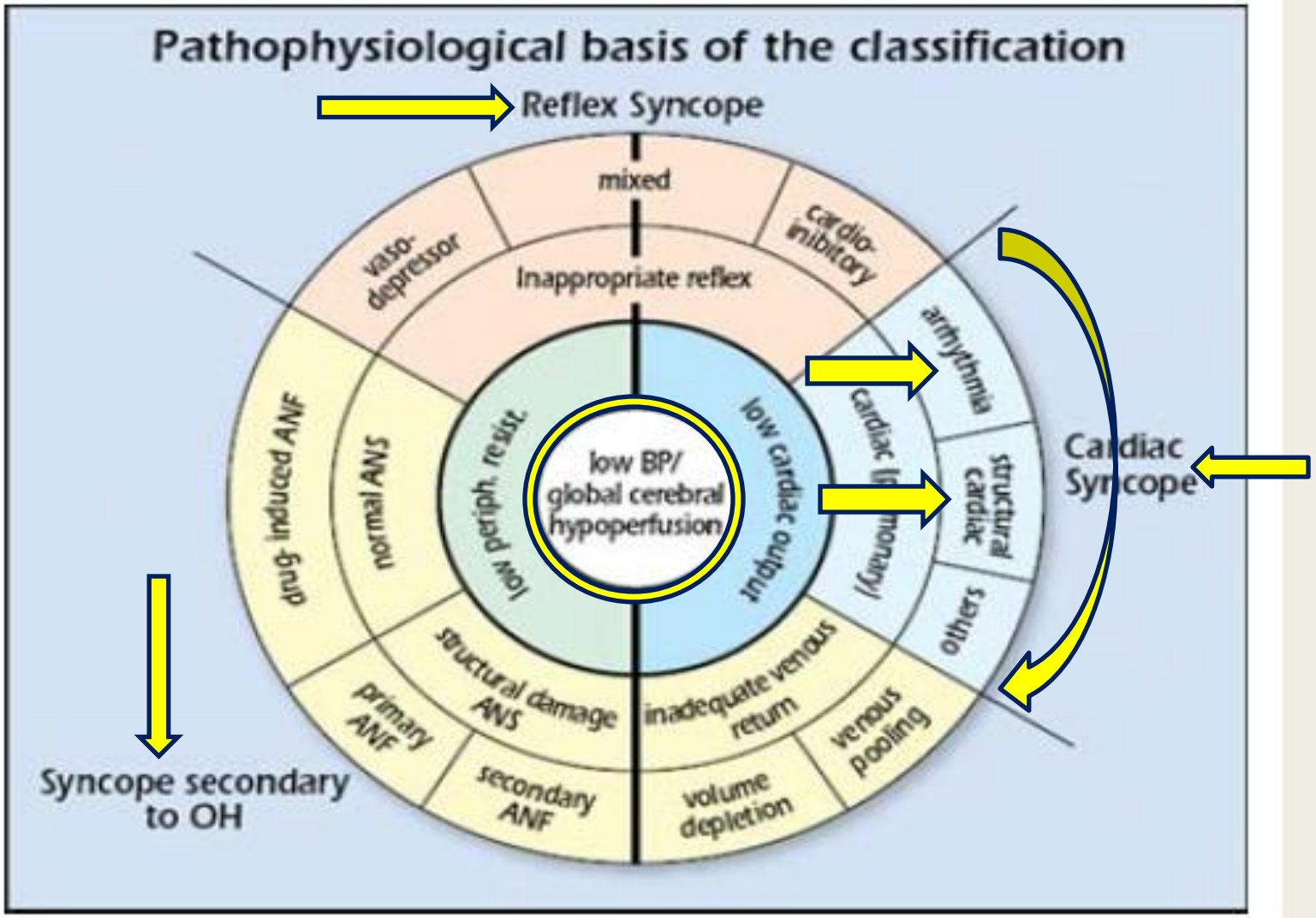


Figure 2 Pathophysiological basis of the classification (see text). ANF = autonomic nervous failure; ANS = autonomic nervous system; BP = blood pressure; low periph. resist. = low peripheral resistance; OH = orthostatic hypotension.

Table 4 Classification of syncope

Reflex (neurally-mediated) syncope

Vasovagal:

- *mediated by emotional distress: fear, pain, instrumentation, blood phobia*
- *mediated by orthostatic stress*

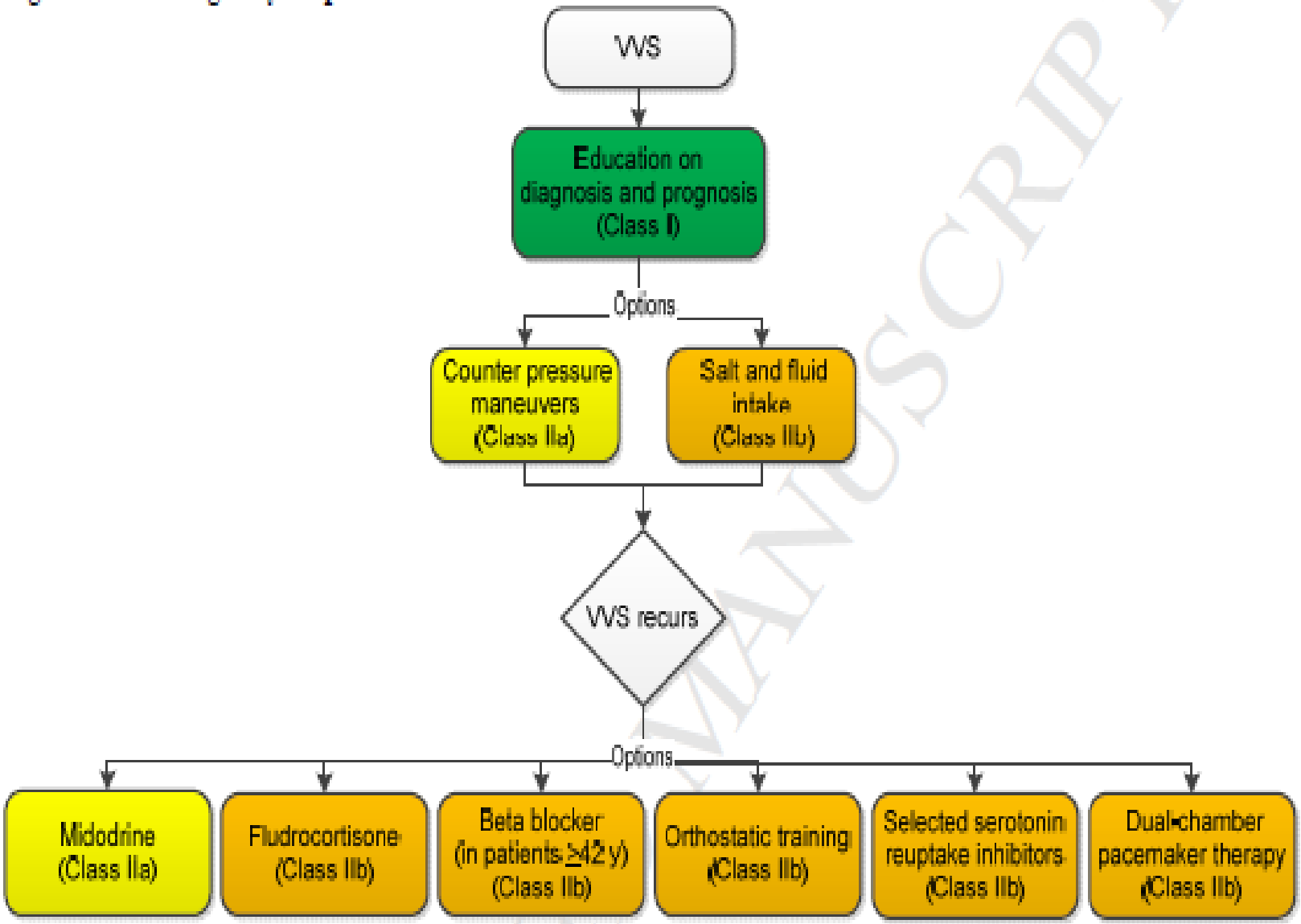
Situational:

- *cough, sneeze*
- *gastrointestinal stimulation (swallow, defaecation, visceral pain)*
- *micturition (post-micturition)*
- *post-exercise*
- *post-prandial*
- *others (e.g., laught, brass instrument playing, weightlifting)*

Carotid sinus syncope

Atypical forms (without apparent triggers and/or atypical presentation)

Figure 4. Vasovagal Syncope



Colors correspond to Class of Recommendation in Table 1.
VVS indicates vasovagal syncope.

Cardiac syncope (cardiovascular)

Arrhythmia as primary cause:

Bradycardia:

- *sinus node dysfunction (including bradycardia/tachycardia syndrome)*
- *atrioventricular conduction system disease*
- *implanted device malfunction,*

Tachycardia:

- *supraventricular*
- *ventricular (idiopathic, secondary to structural heart disease or to channelopathies)*

Drug induced bradycardia and tachyarrhythmias

Structural disease:

Cardiac: *cardiac valvular disease, acute myocardial infarction/ischaemia, hypertrophic cardiomyopathy, cardiac masses (atrial myxoma, tumors, etc), pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valves dysfunction*

Others: *pulmonary embolus, acute aortic dissection, pulmonary hypertension*

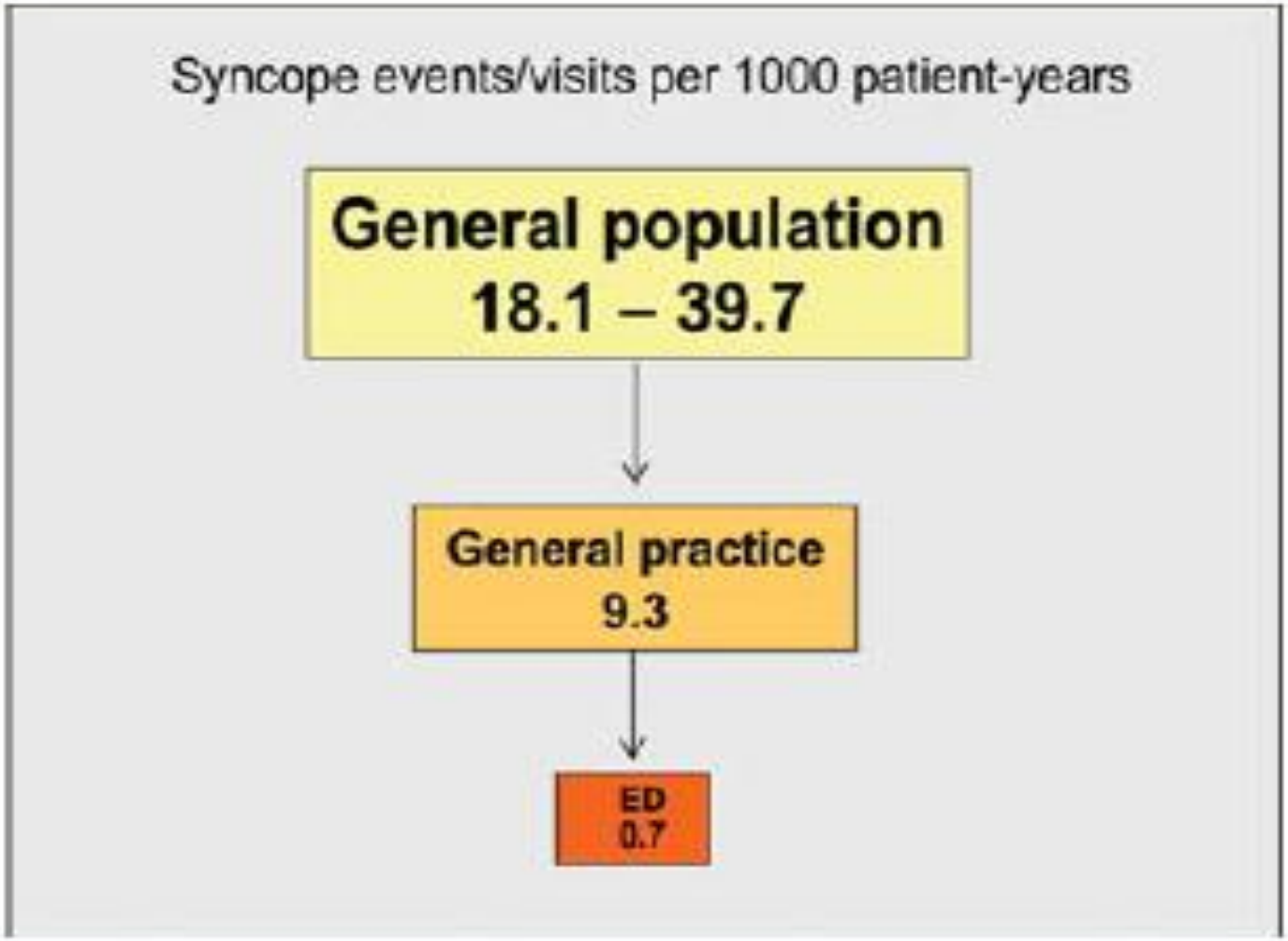


Figure 6 Syncope events/visits per 1000 patient-years in The Netherlands (from Ganzeboom *et al.*[27] with permission). ED = Emergency Department.

Part 2. Initial evaluation, diagnosis, and risk stratification

2.1 Initial evaluation

The initial evaluation of a patient presenting with T-LOC consists of careful history, physical examination, including orthostatic BP measurements, and electrocardiogram (ECG). Based on these findings, additional examinations may be performed:

2.1.1 Diagnosis of syncope

The following questions should be answered:

- Was LOC complete?
- Was LOC transient with rapid onset and short duration?
- Did the patient recover spontaneously, completely and without sequelae?
- Did the patient lose postural tone?

If the answers to these questions are positive, the episode has a high likelihood of being syncope. If the answer to one or more of these questions is negative, exclude other forms of LOC before proceeding with syncope evaluation.

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2.3.2. Electrocardiography: Recommendation

Recommendation for Electrocardiography		
COR	LOE	Recommendation
I	B-NR	In the initial evaluation of patients with syncope, a resting 12-lead electrocardiogram (ECG) is useful (76).
See Online Data Supplement 2.		ECG is widely available and inexpensive and can provide information about the potential and specific cause of the syncope episode (e.g., bradyarrhythmia with sinus pauses or high-grade conduction block; ventricular tachyarrhythmia). It may demonstrate an underlying arrhythmogenic substrate for syncope or SCD. Subsets

Dx Etiológico

La Evaluación inicial es capaz de definir la causa del Síncope en un 23 – 50% de los pacientes.

Table 9 Important historical features

Questions about circumstances just prior to the attack

- Position (supine, sitting or standing)
- Activity (rest, change in posture, during or after exercise, during or immediately after urination, defaecation, cough, or swallowing)
- Predisposing factors (e.g. crowded or warm places, prolonged standing, post-prandial period) and of precipitating events (e.g. fear, intense pain, neck movements)

Questions about onset of the attack

- Nausea, vomiting, abdominal discomfort, feeling of cold, sweating, aura, pain in neck or shoulders, blurred vision, dizziness
- Palpitations

Questions about the attack (eyewitness)

- Way of falling (slumping or kneeling over), skin colour (pallor, cyanosis, flushing), duration of loss of consciousness, breathing pattern (snoring), movements (tonic, clonic, tonic–clonic, minimal myoclonus or automatism), duration of movements, onset of movement in relation to fall, tongue biting

Table 9 Important historical features

Questions about the end of the attack

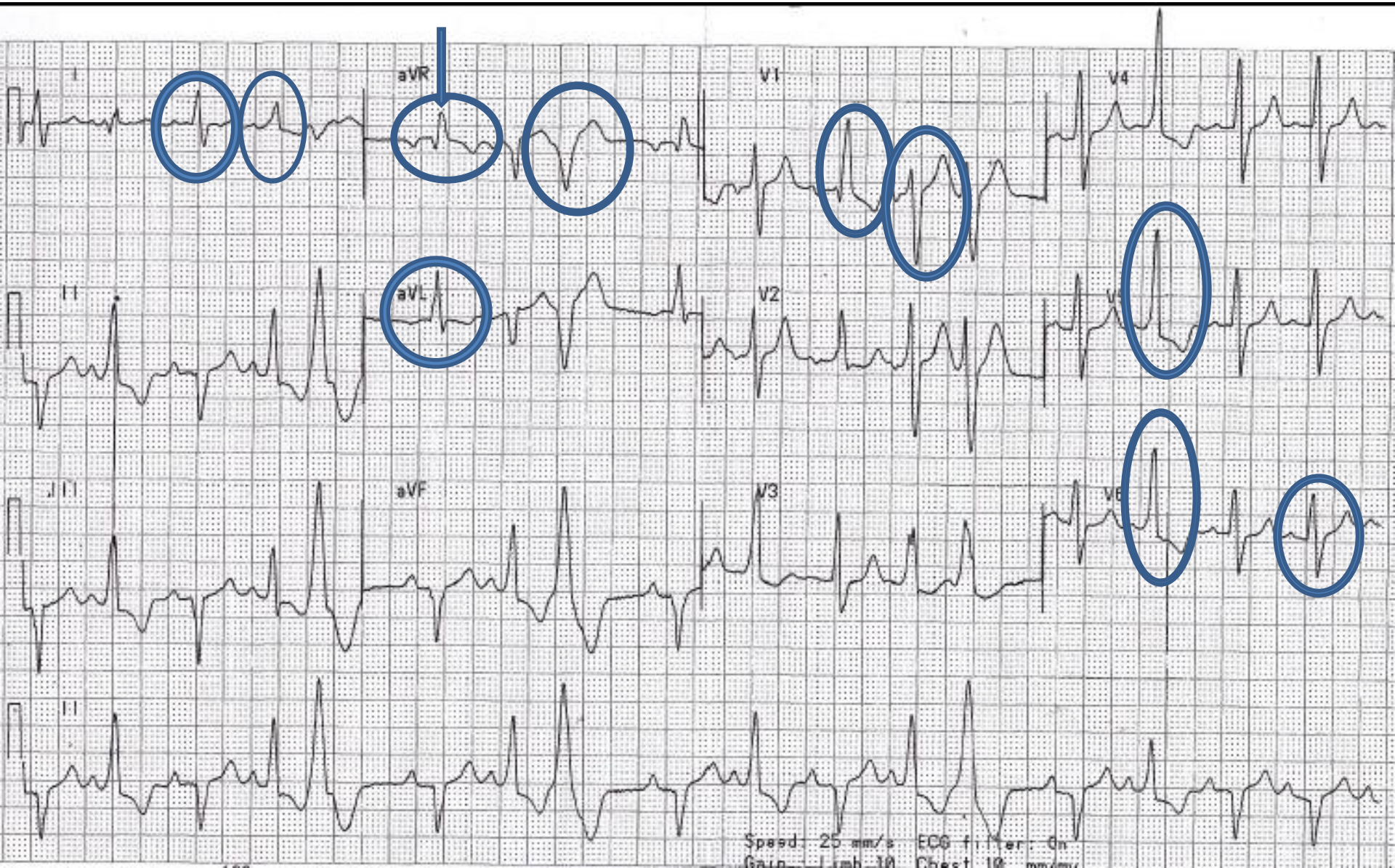
- Nausea, vomiting, sweating, feeling of cold, confusion, muscle aches, skin colour, injury, chest pain, palpitations, urinary or faecal incontinence

Questions about the background

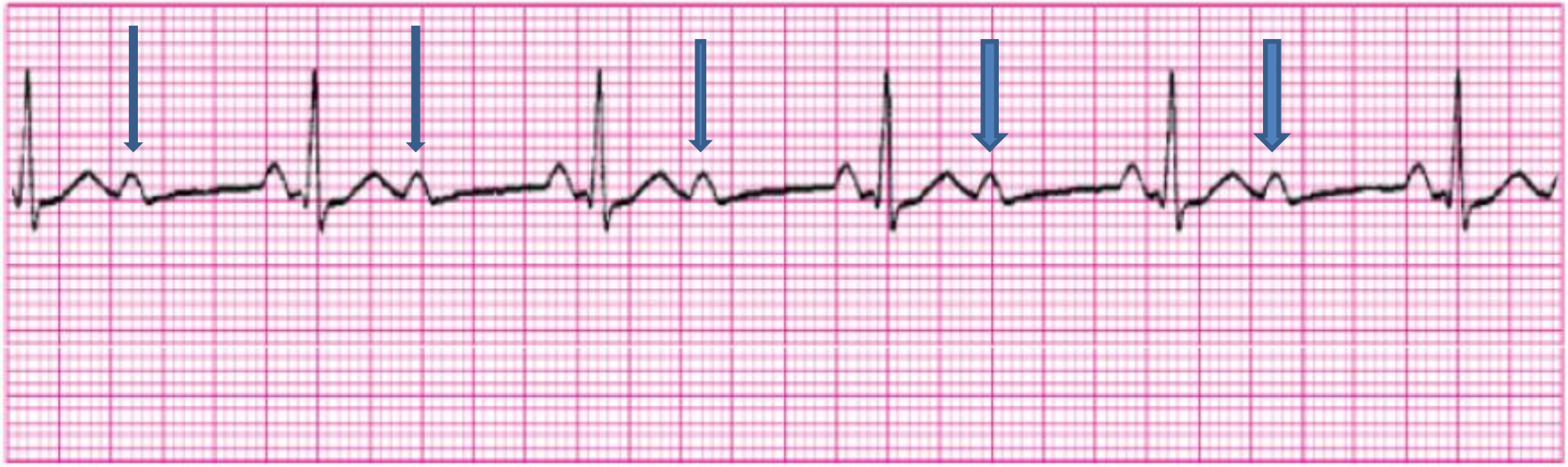
- Family history of sudden death, congenital arrhythmogenic heart disease or fainting
- Previous cardiac disease
- Neurological history (Parkinsonism, epilepsy, narcolepsy)
- Metabolic disorders (diabetes, etc.)
- Medication (antihypertensive, antianginal, antidepressant agent, antiarrhythmic, diuretics, and QT-prolonging agents) or other drugs including alcohol
- In the case of recurrent syncope, information on recurrences such as the time from the first syncopal episode and on the number of spells

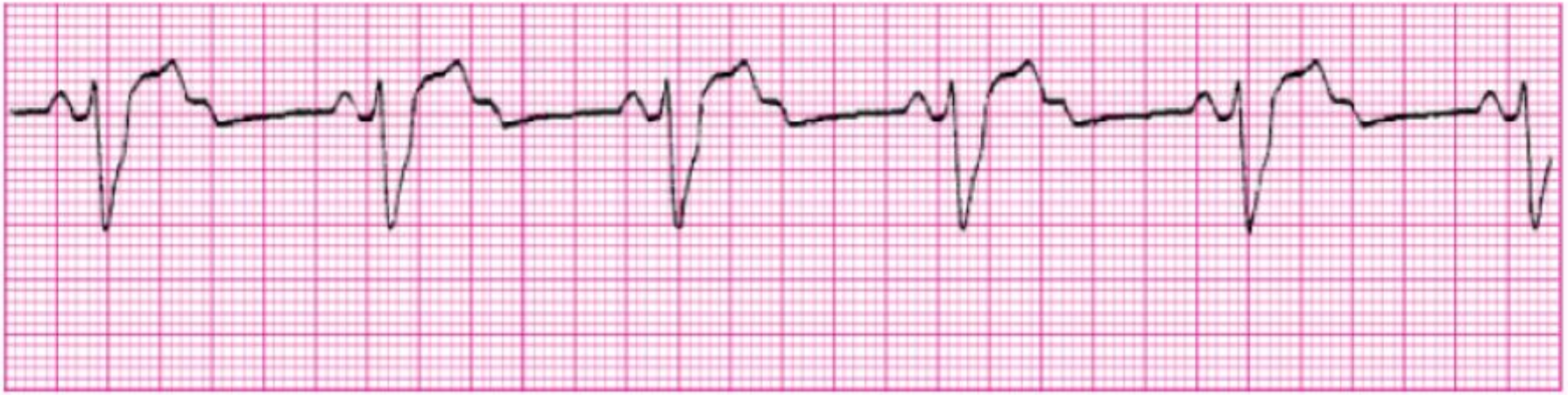
Arrhythmia-related syncope is diagnosed by ECG when there is: • Persistent sinus bradycardia <40 bpm in awake or repetitive sinoatrial block or sinus pauses ≥3 s • Mobitz II second or third degree AV block • Alternating left and right BBB • VT or rapid paroxysmal SVT • Non-sustained episodes of polymorphic VT and long or short QT interval • Pacemaker or ICD malfunction with cardiac pauses	I	C
Cardiac ischaemia-related syncope is diagnosed when syncope presents with ECG evidence of acute ischaemia with or without myocardial infarction	I	C
Cardiovascular syncope is diagnosed when syncope presents in patients with prolapsing atrial myxoma, severe aortic stenosis, pulmonary hypertension, pulmonary embolus, or acute aortic dissection	I	C

M, 69a, Asintomático. Alternancia de bloqueos R.

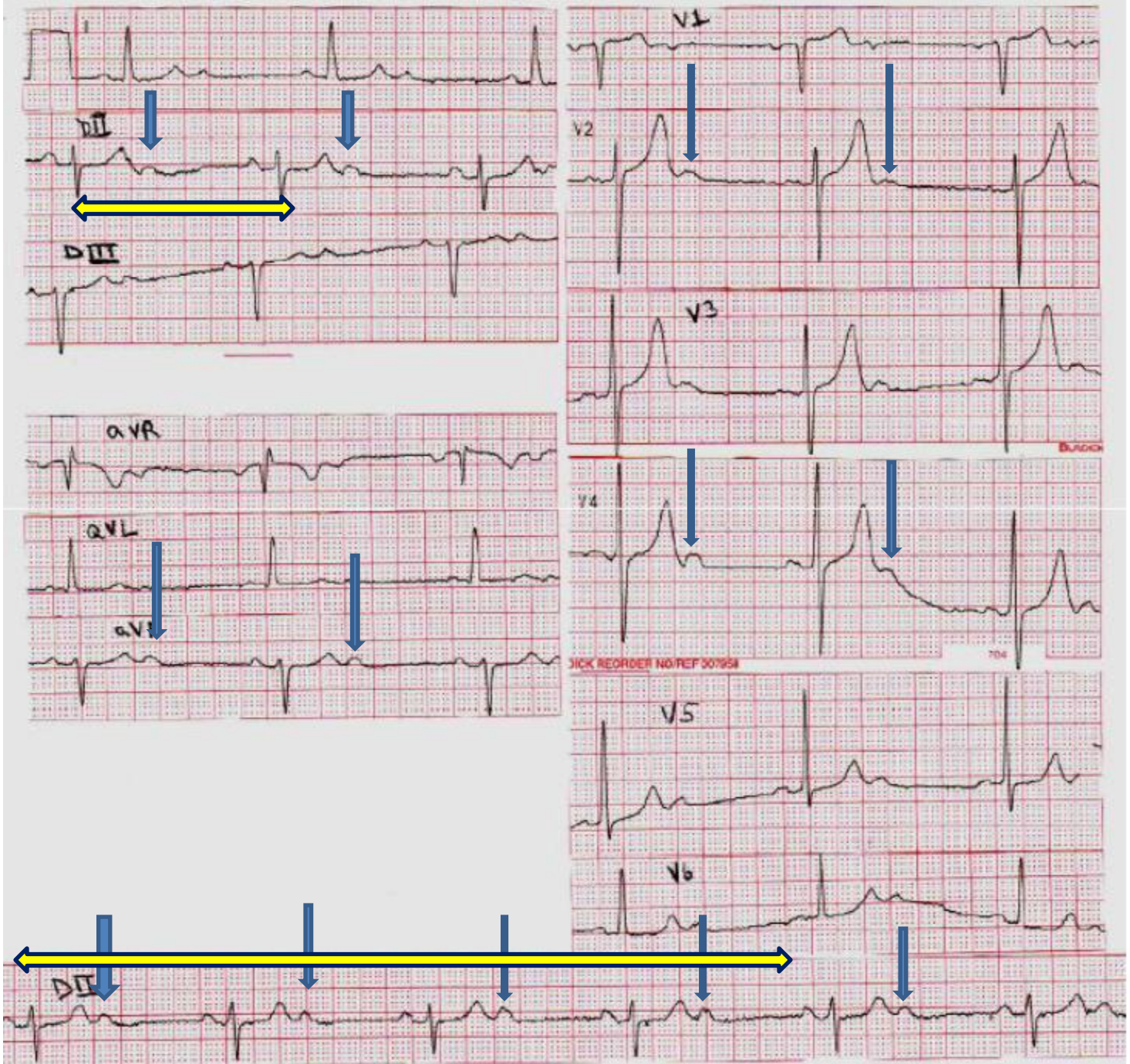


BAV 2doº Mobitz II, 2:1

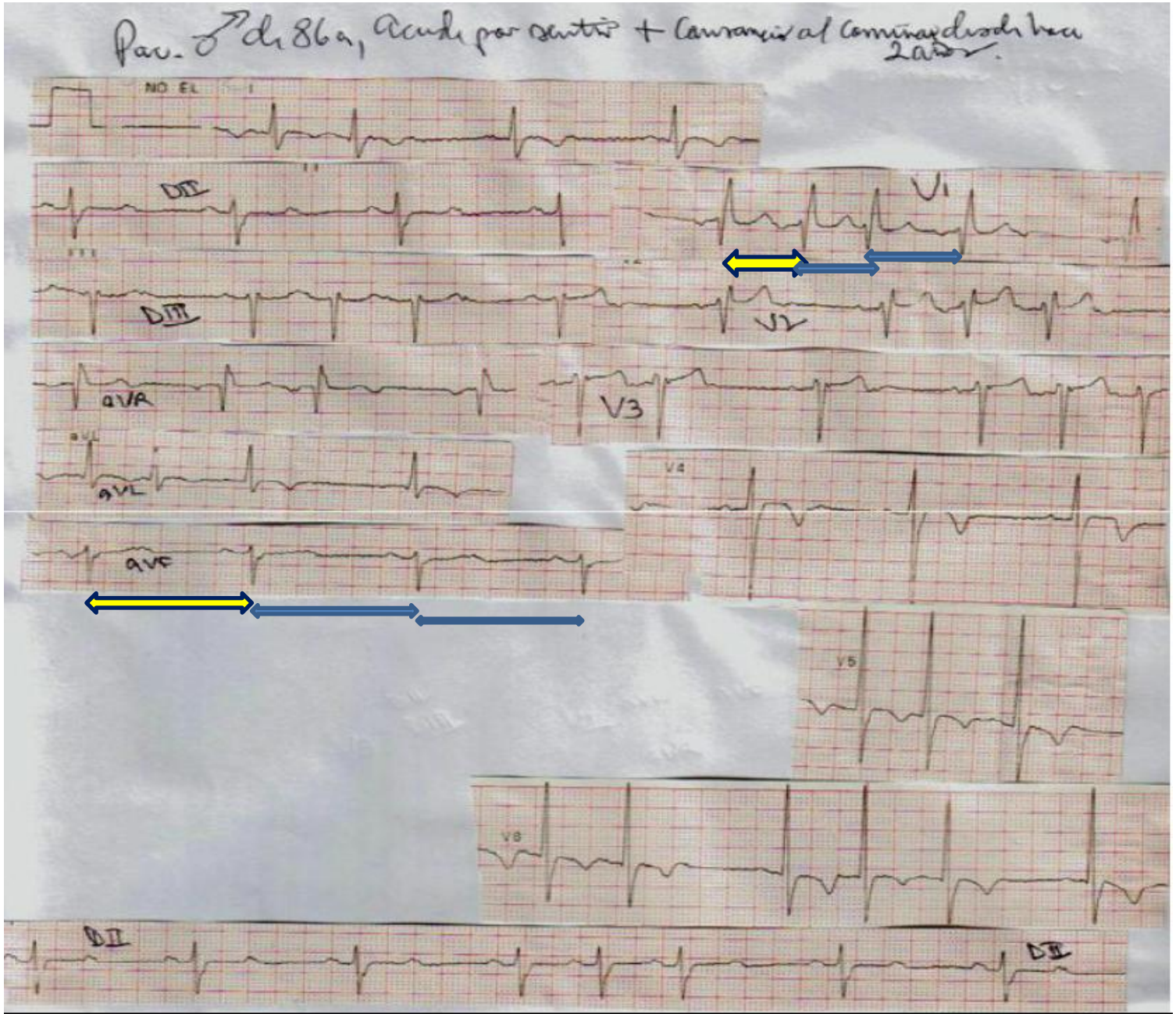




Paciente masculino de 77ª que acude con referido y con historia de síncope (hace días). Antecedente de HTA, DM y Enfermedad De Parkinson. PA: 110/70.

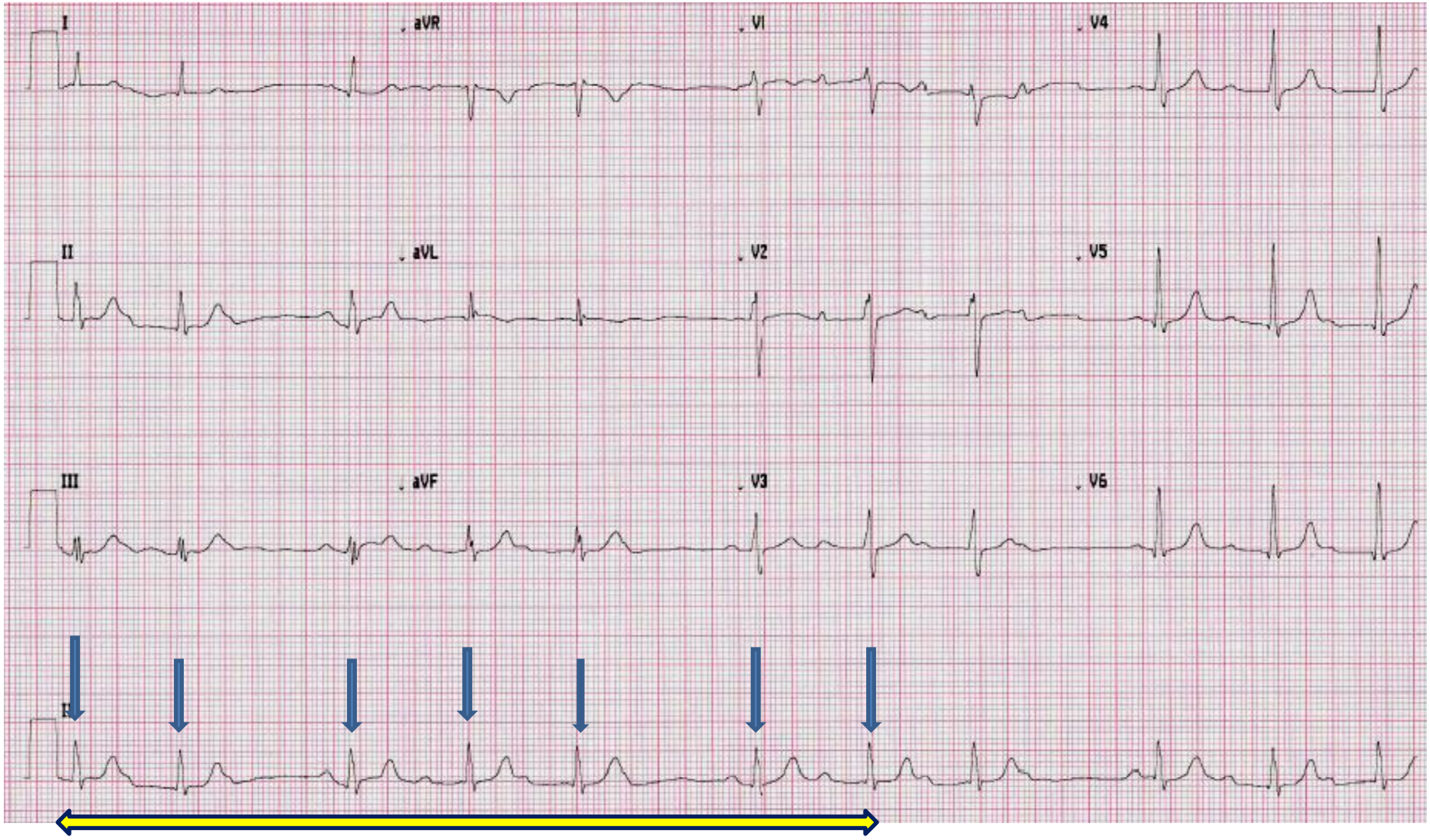


B.AV. IIº Mobitz tipo 2, Intermitente.

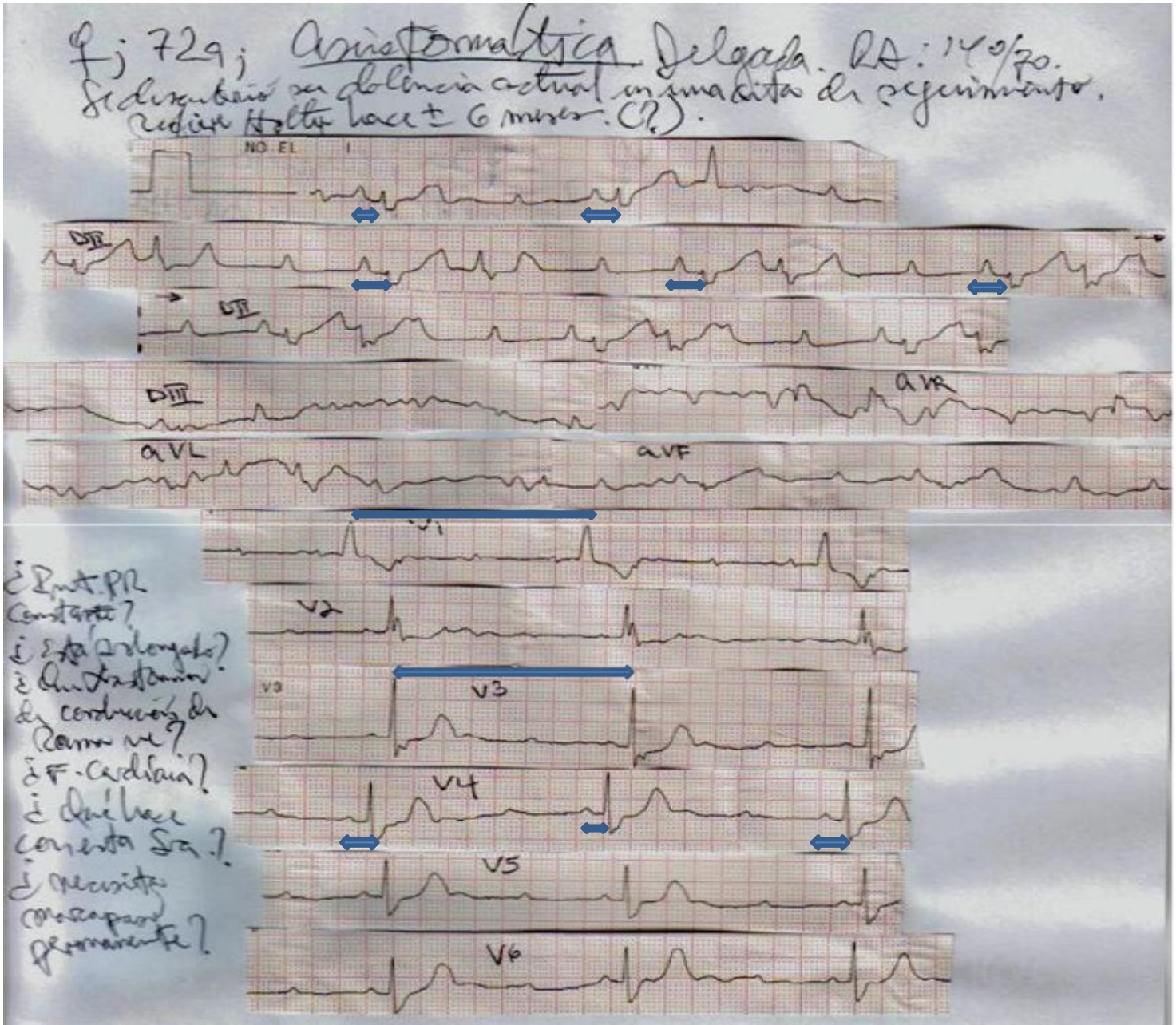


M, 54ª, Asintomático. B. AV. II° Mobitz 1.

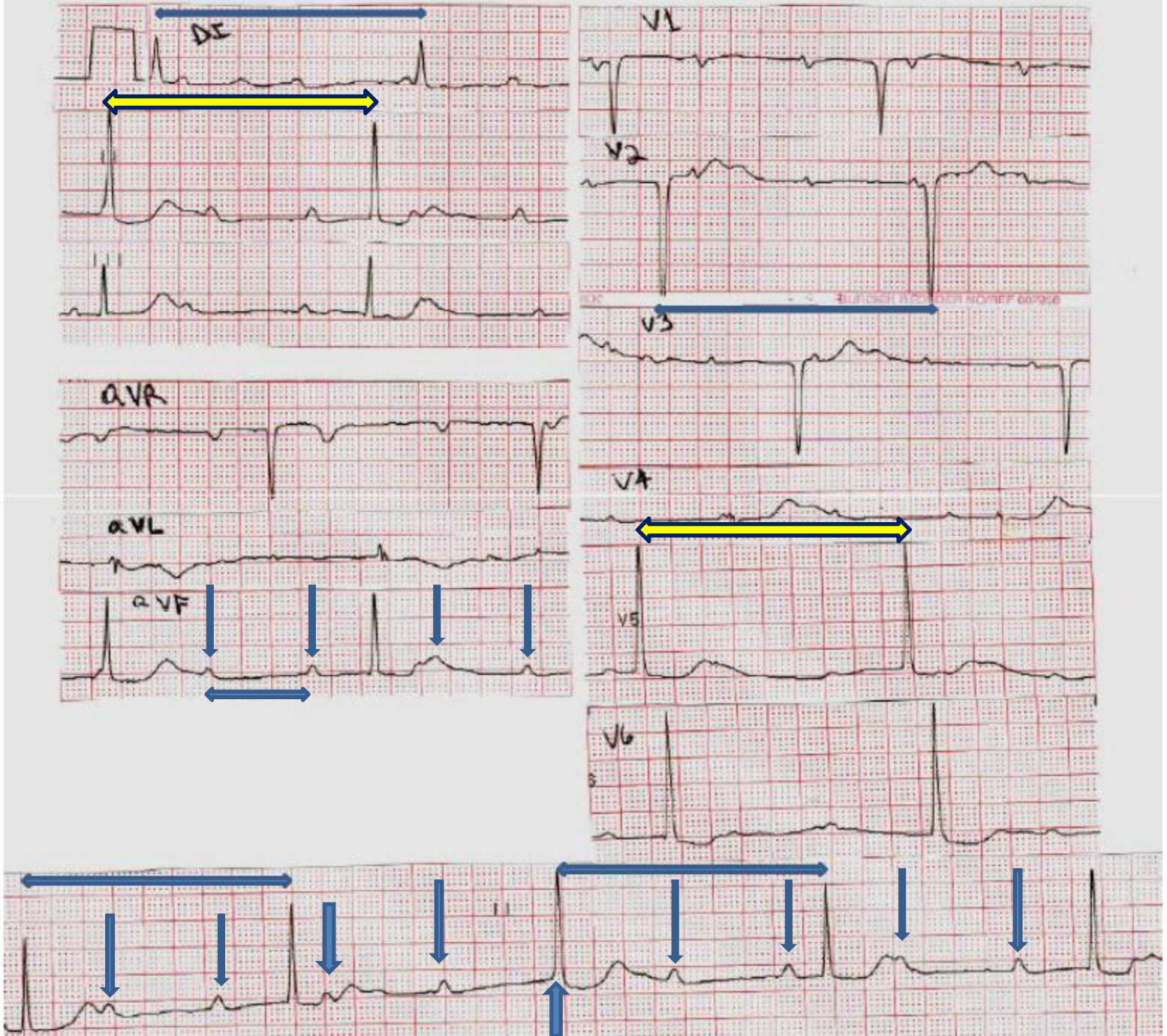
No se implica en Síncopes, ¿Por qué?



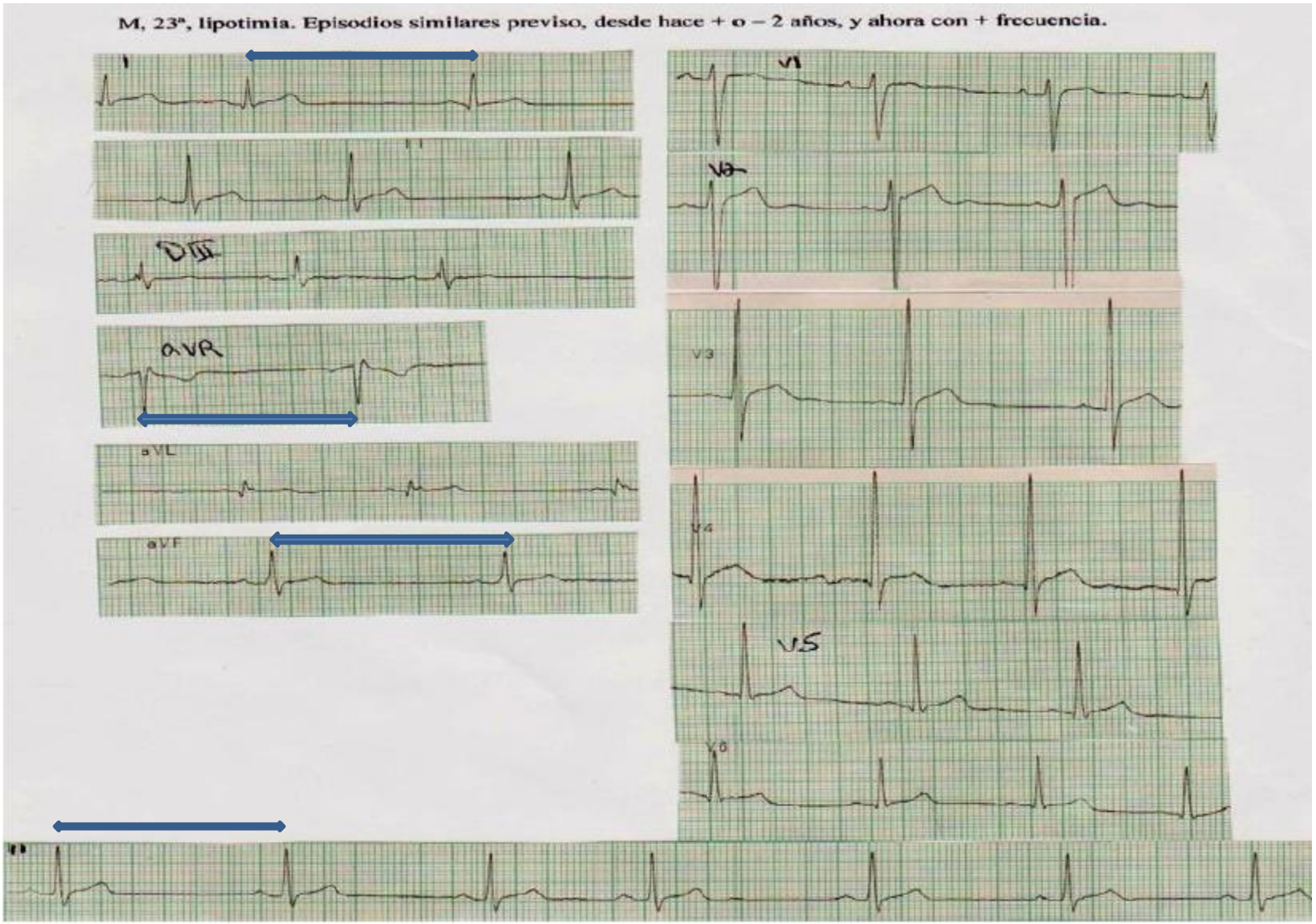
B.AV. “Avanzado”.



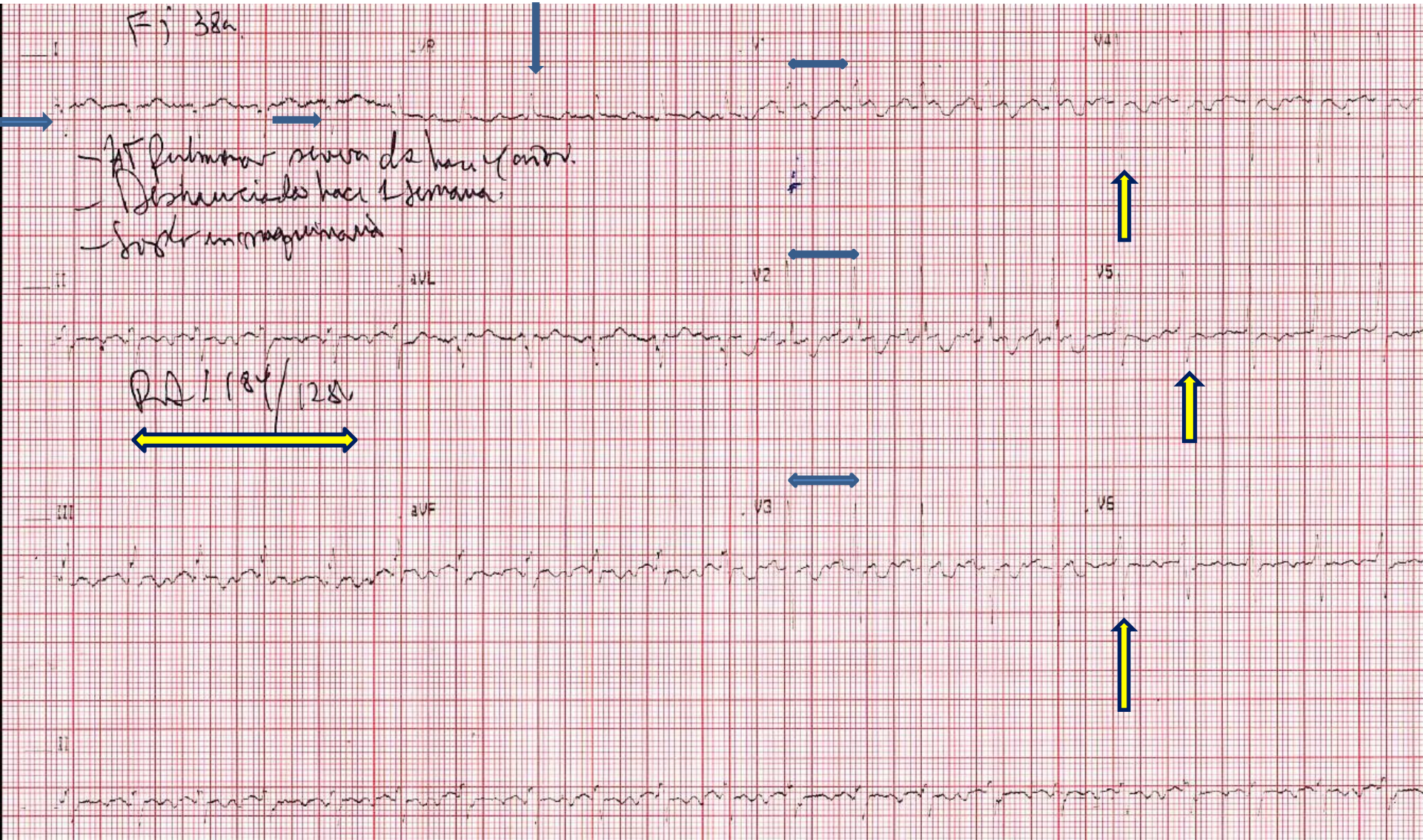
F, 58ª, Síncopes cada vez + frecuentes desde principios de este año. PA: 150/80.



El siguiente ECG de un paciente masculino de 23 años con lipotimias desde hace 2 años. Bradiarritmia sinusal. La FC va de 43 lpm (ver DI y DII largo) a 60 lpm (ver DII corto, V4 y V6).



F, 38ª, con HT Pulmonar severa.



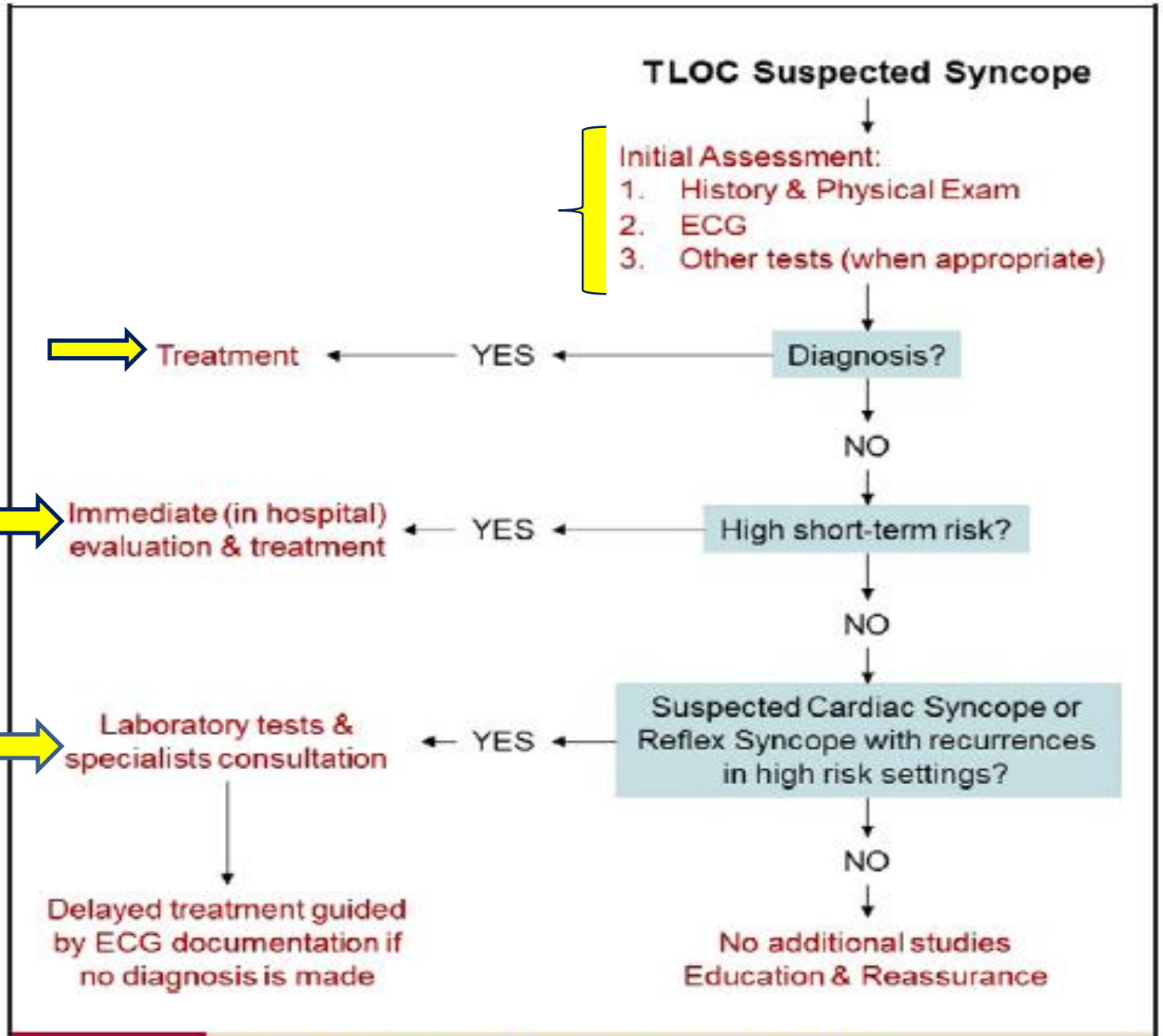
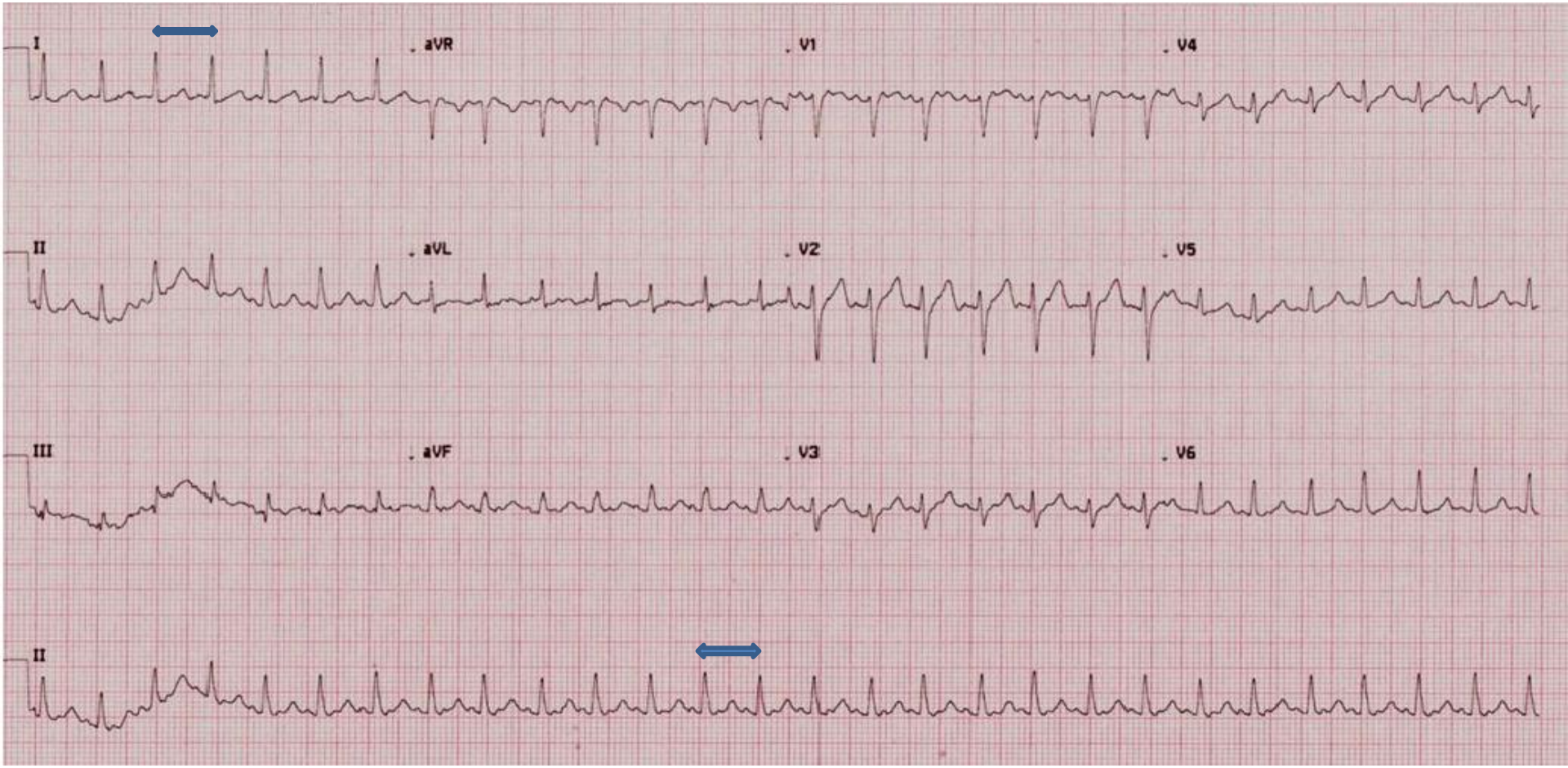
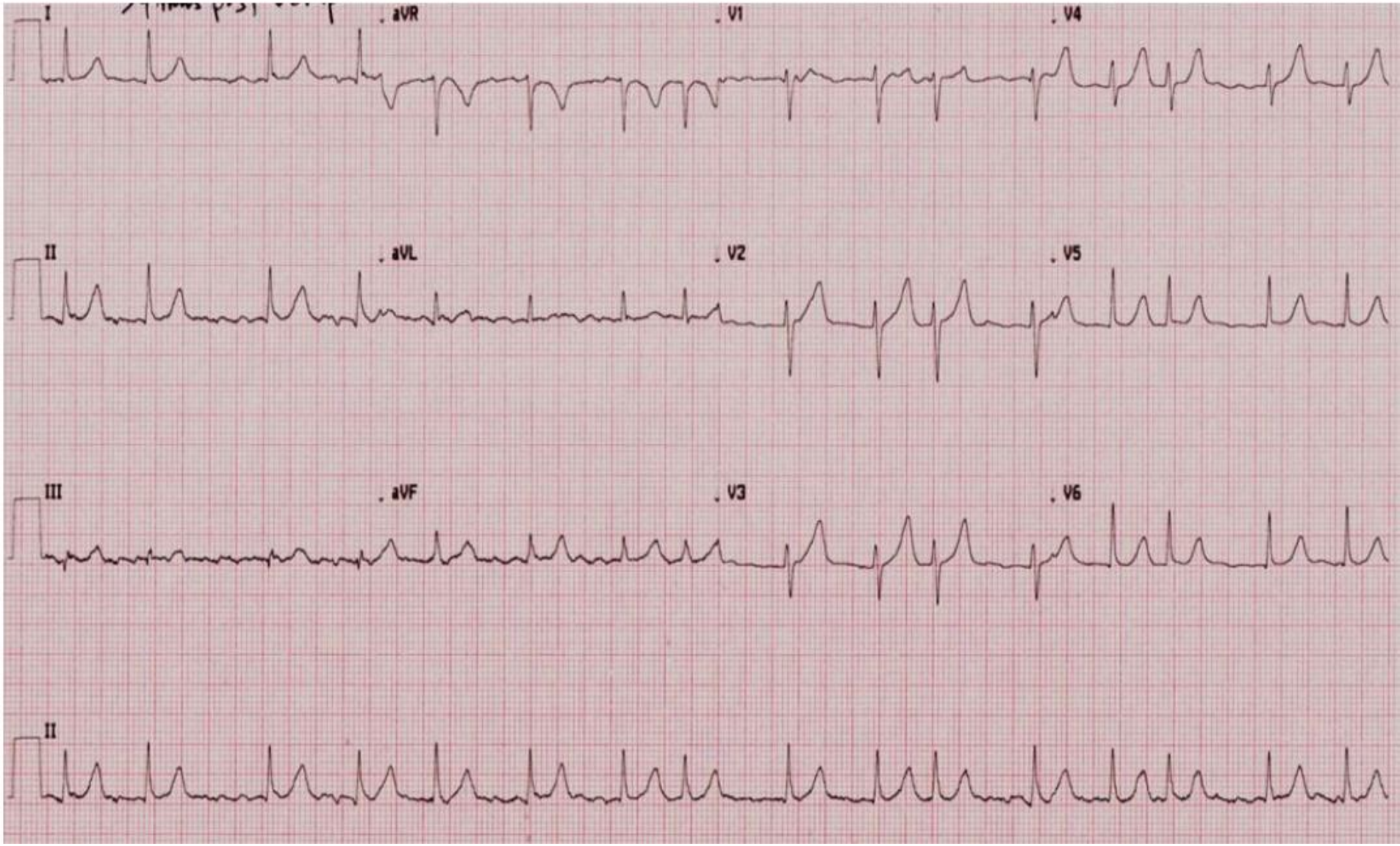


Figure 2 The Diagnostic Algorithm of a Patient Presenting With TLOC of Suspected Syncopal Nature

Paciente Femenina de 68ª con historia de síncope hace 2 horas, en su casa.



Luego de la aplicación de Verapamilo 5 mg IV, obtuvimos el siguiente ECG:



Pero todavía no está para darle salida, así que le apliqué una dosis de Amiodarona a 150 mg en 100 ml de SSN, para pasar en 30 minutos, y antes de terminar el goteo, resultó el ECG que vemos abajo:

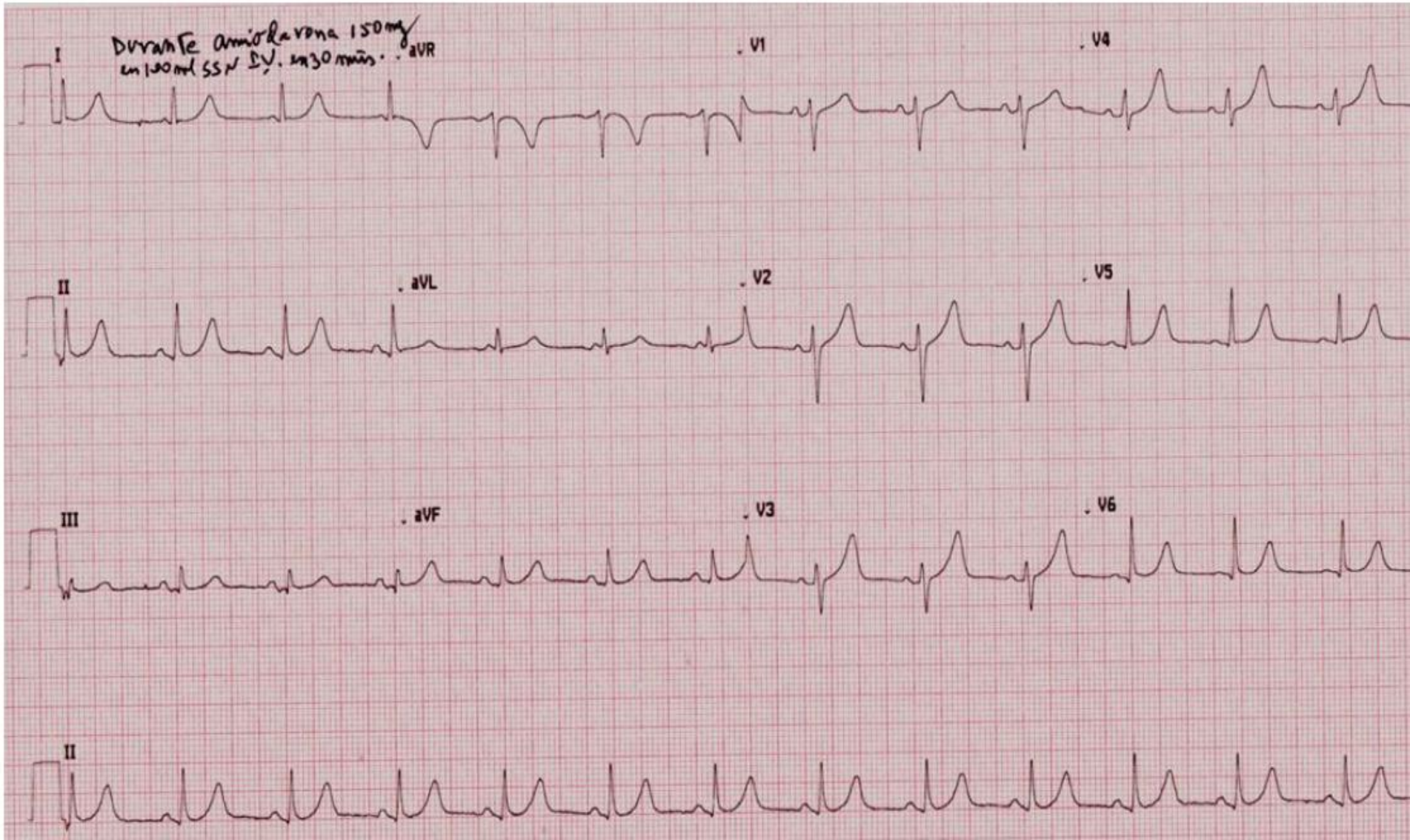
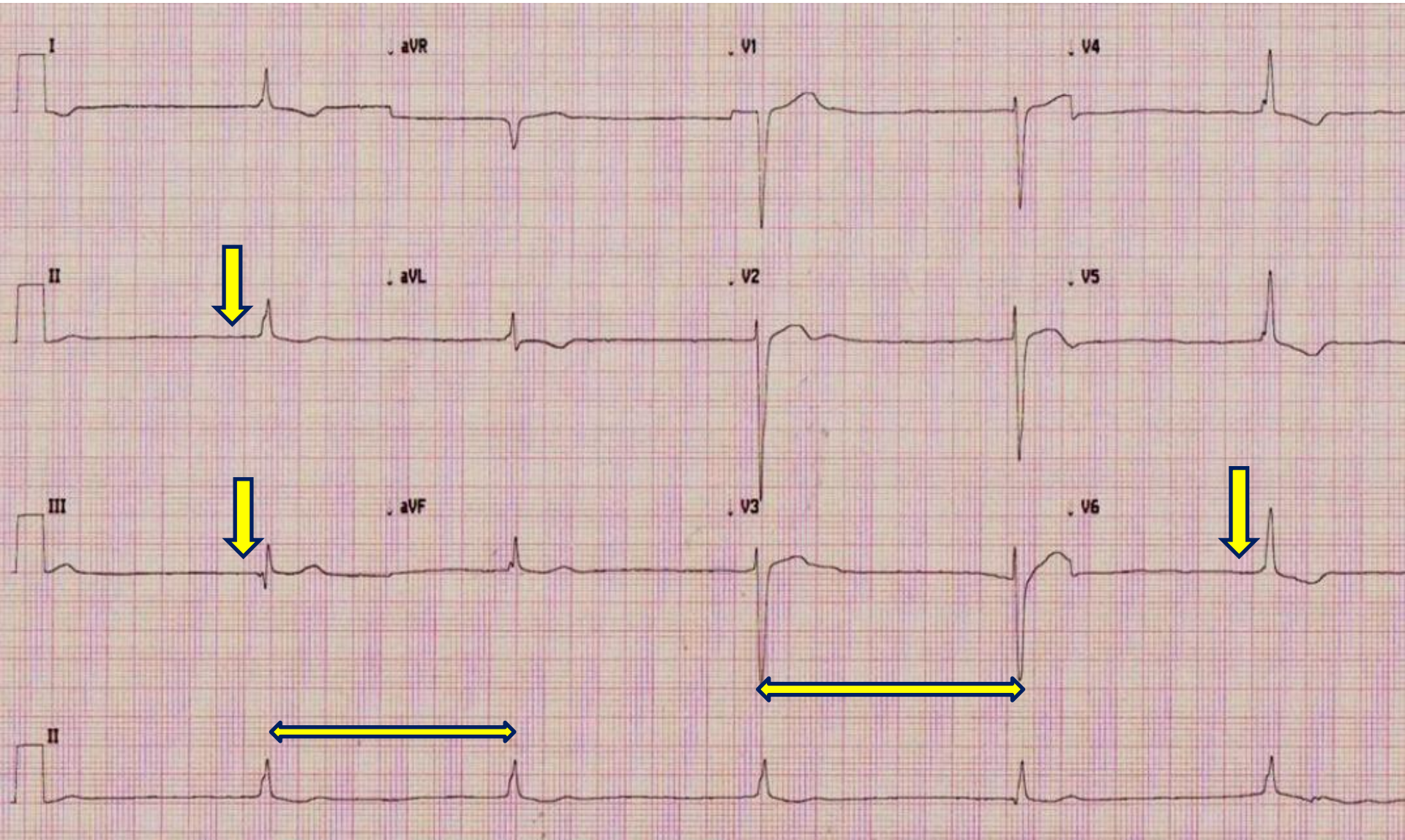


Tabla 5

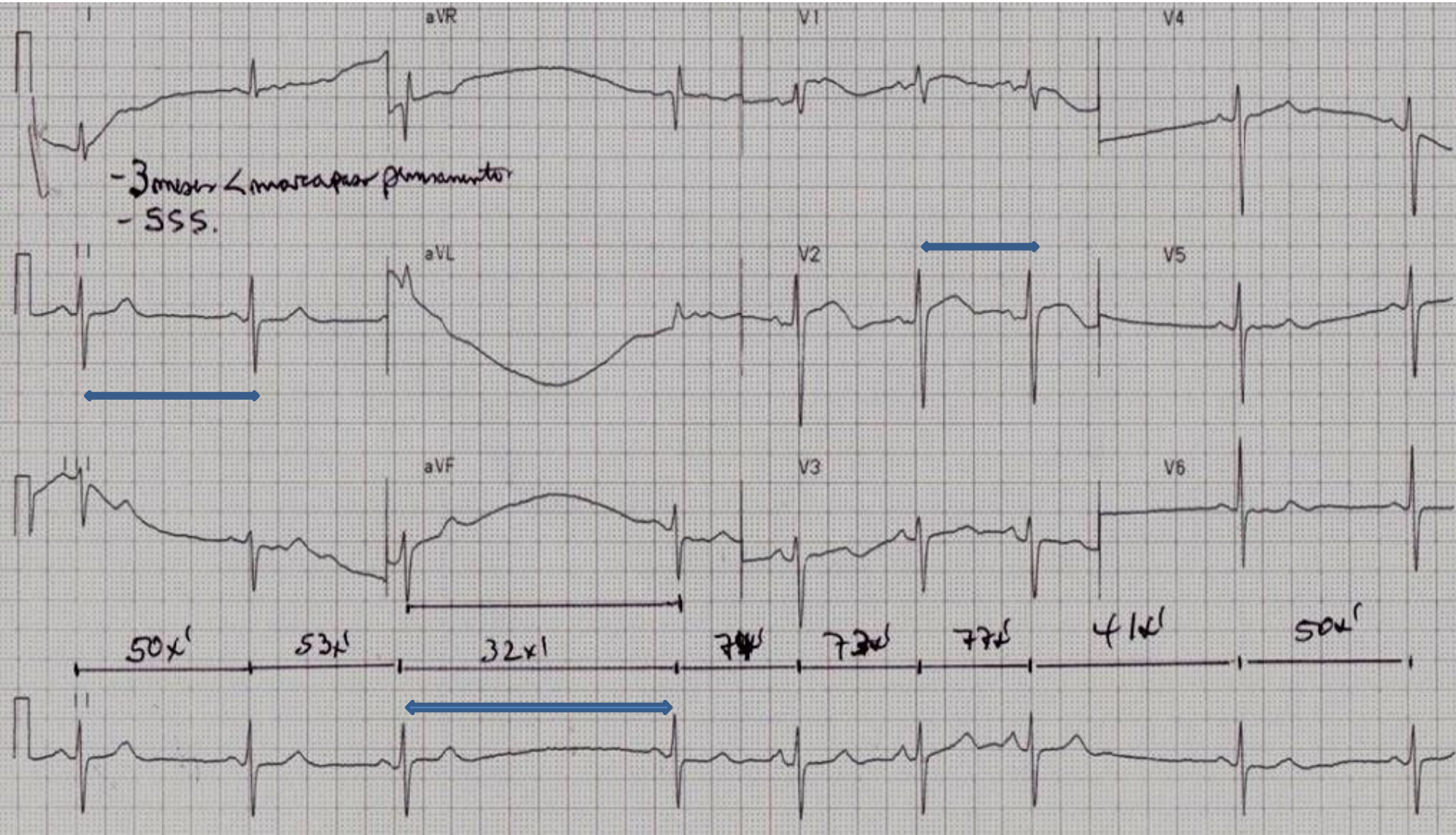
Criterios de riesgo que requieren evaluación inmediata u hospitalización

Presencia de cardiopatía isquémica o dilatada con FE < 35%
Antecedentes de necrosis miocárdica
Presencia de insuficiencia cardiaca
Episodios de TVNS
Bloqueo bifascicular (BRI o BRD más HBA o HBP) o QRS ≥ 120 ms
Preexcitación
QT largo o corto
Historia familiar de muerte súbita
ECG con patrón tipo Brugada

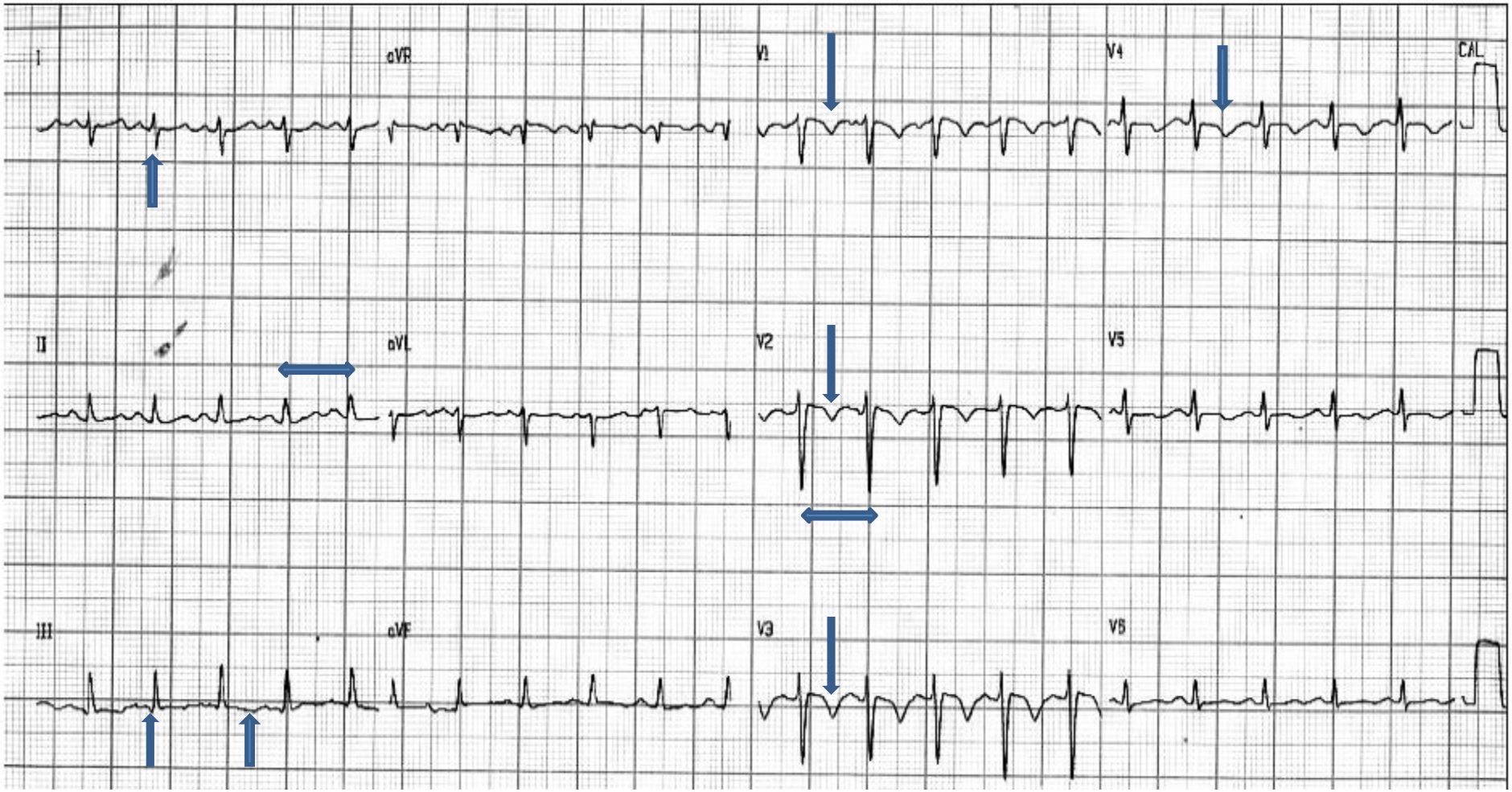
Otra posible causa de síncope y pre-síncope, ritmo nodal: con FC de 33 l/min en un paciente masculino de 73 años que ingresa por lipotimias y más cansancio.



Vemos el ECG de esta paciente de 80 años que ingresa con historia de síncope hace un par de horas, con disnea y ortopnea, y a la cual se le había implantado un marcapaso permanente hace 3 meses por Síndrome del nodo sinusal enfermo, pero que no le vemos las clásicas espigas previas a los complejos QRS:



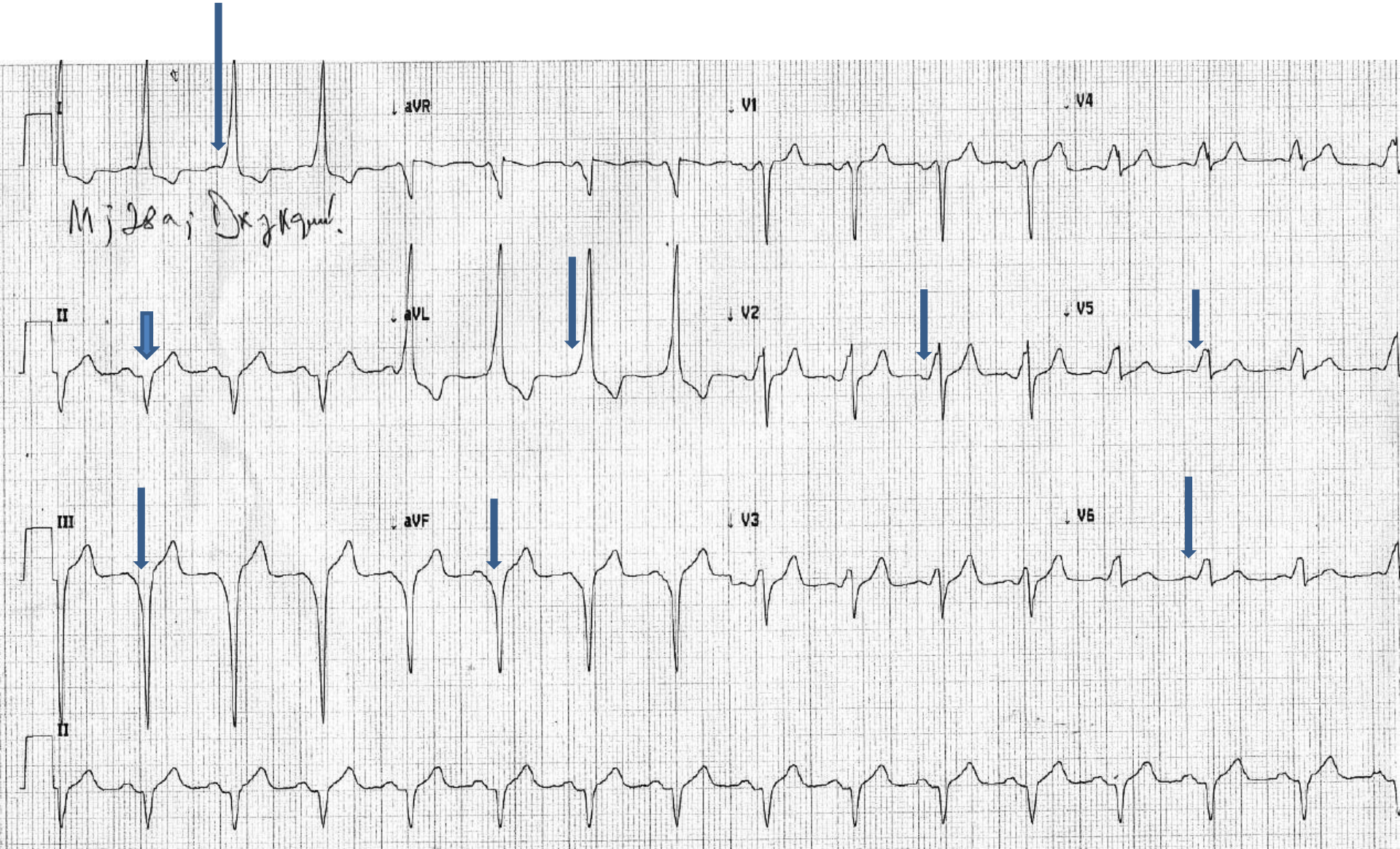
F, 21, que sufre síncope y es llevada al SU. Historia de "desmayo" hace 1 mes. Obesa. ECO: discreto crecimiento de VD sin HTPulmonar.



Claves ECGráficas en el TEP

1) **Taquicardia sinusal**: el más común.
2) "P" Pulmonale.
3) **Patrón McGinn S, White: S1Q3T3: 20%.**
4) **Desviación del Eeº del QRS a la Derecha**.
5) **BIRD o BCRD (de nueva aparición**). **Revisar expediente.**
6) Cambios difusos del ST/T**, incluyendo ondas T (-)** y depresión del ST.
7) **Deflexiones de baja amplitud** (+ en el plano frontal).
8) **Inversiones de onda T en DIII y V1-V4** (DxDif c SCA).
9) Rotación horaria (precordiales).
10) Arritmias auriculares: FA, Fl.A, TSV, Taq.Atrial y CAPs.

M, 28º, Dx y Por qué. Cómo manejaría al paciente.



Recommendations: active standing

Recommendations	Class[a]	Level[b]
Indications		
• Manual intermittent determination with sphygmomanometer of BP supine and during active standing for 3 min is indicated as initial evaluation when OH is suspected	I	B
• Continuous beat-to-beat non-invasive pressure measurement may be helpful in cases of doubt	IIb	C
Diagnostic criteria		
• The test is diagnostic when there is a symptomatic fall in systolic BP from a baseline value ≥20 mmHg or diastolic BP ≥ 10 mmHg, or a decrease in systolic BP to <90 mmHg	I	C
• The test should be considered diagnostic when there is an asymptomatic fall in systolic BP from baseline value ≥20 mmHg or diastolic BP ≥10 mmHg, or a decrease in systolic BP to <90 mmHg	IIa	C

Recommendations: neurological evaluation

Recommendations	Class[a]	Level[b]
Indications		
• Neurological evaluation is indicated in patients in whom T-LOC is suspected to be epilepsy	I	C
• Neurological evaluation is indicated when syncope is due to ANF in order to evaluate the underlying disease	I	C
• EEG, ultrasound of neck arteries, and computed tomography or magnetic resonance imaging of the brain are not indicated, unless a non-syncopal cause of T-LOC is suspected	III	B

Recommendations: treatment of reflex syncope

Recommendations	Class[a]	Level[b]
• Explanation of the diagnosis, provision of reassurance, and explanation of risk of recurrence are indicated in all patients	I	C
• Isometric PCMs are indicated in patients with prodrome	I	B
• Cardiac pacing should be considered in patients with dominant cardioinhibitory CSS	IIa	B
• Cardiac pacing should be considered in patients with frequent recurrent reflex syncope, age >40 years, and documented spontaneous cardioinhibitory response during monitoring	IIa	B
• Midodrine may be indicated in patients with VVS refractory to lifestyle measures	IIb	B
• Tilt training may be useful for education of patients but long-term benefit depends on compliance	IIb	B
• Cardiac pacing may be indicated in patients with tilt-induced cardioinhibitory response with recurrent frequent unpredictable syncope and age >40 after alternative therapy has failed	IIb	C
• Cardiac pacing is not indicated in the absence of a documented cardioinhibitory reflex	III	C
• β-Adrenergic blocking drugs are not indicated	III	A

Recommendations: treatment of orthostatic hypotension

Recommendations	Class[a]	Level[b]
• Adequate hydration and salt intake must be maintained	I	C
• Midodrine should be administered as adjunctive therapy if needed	IIa	B
• Fludrocortisone should be administered as adjunctive therapy if needed	IIa	C
• PCMs may be indicated	IIb	C
• Abdominal binders and/or support stockings to reduce venous pooling may be indicated	IIb	C
• Head-up tilt sleeping (>10°) to increase fluid volume may be indicated	IIb	C

Recommendations: treatment of syncope due to cardiac arrhythmias

Recommendations	Class[a]	Level[b]
• Syncope due to cardiac arrhythmias must receive treatment appropriate to the cause	I	B
Cardiac pacing		
• Pacing is indicated in patients with sinus node disease in whom syncope is demonstrated to be due to sinus arrest (symptom–ECG correlation) without a correctable cause	I	C
• Pacing is indicated in sinus node disease patients with syncope and abnormal CSNRT	I	C
• Pacing is indicated in sinus node disease patients with syncope and asymptomatic pauses ≥ 3 s (with the possible exceptions of young trained persons, during sleep, and in medicated patients)	I	C
• Pacing is indicated in patients with syncope and second degree Mobitz II, advanced or complete AV block	I	B
• Pacing is indicated in patients with syncope, BBB, and positive EPS	I	B
• Pacing should be considered in patients with unexplained syncope and BBB	IIa	C
• Pacing may be indicated in patients with unexplained syncope and sinus node disease with persistent sinus bradycardia itself asymptomatic	IIb	C
• Pacing is not indicated in patients with unexplained syncope without evidence of any conduction disturbance	III	C

Catheter ablation		
• Catheter ablation is indicated in patients with symptom–arrhythmia ECG correlation in both SVT and VT in the absence of structural heart disease (with the exception of atrial fibrillation)	I	C
• Catheter ablation may be indicated in patients with syncope due to the onset of rapid atrial fibrillation	IIb	C
Antiarrhythmic drug therapy		
• Antiarrhythmic drug therapy, including rate control drugs, is indicated in patients with syncope due to onset of rapid atrial fibrillation	I	C
• Drug therapy should be considered in patients with symptom–arrhythmia ECG correlation in both SVT and VT when catheter ablation cannot be undertaken or has failed	IIa	C
Implantable cardioverter defibrillator		
• ICD is indicated in patients with documented VT and structural heart disease	I	B
• ICD is indicated when sustained monomorphic VT is induced at EPS in patients with previous myocardial infarction	I	B
• ICD should be considered in patients with documented VT and inherited cardiomyopathies or channelopathies	IIa	B

5 PASOS EN EMERGENCIAS

1. ESTABLECER SI EL PACIENTE SUFRIO UN SINCOPE O NO
2. INTERROGATORIO Y EXAMEN FISICO
3. SOLICITAR EXAMENES COMPLEMENTARIOS
4. TRATAR DE ESTABLECER ETIOLOGIA
5. ESTRATIFICAR EL RIESGO DEL PACIENTE PARA INTERNARLO O NO

- Hemograma
- Glucemia
- Creatinina
- Electrolitos
- ECG
- Ecocardiograma

ES IMPORTANTE REALIZAR ECG EN LA SALA DE EMERGENCIAS PORQUE PUEDE ESTABLECER O HACER SOSPECHAR DIAGNOSTICO DE SINCOPE

DEBE HACERSE DE RUTINA

EN TODO PACIENTE CON SINCOPE

- La mayoría de los pacientes que acuden al servicio de urgencias se encuentran completamente recuperados en el momento de la consulta.
- El síncope puede ser un marcador de alto riesgo de mortalidad

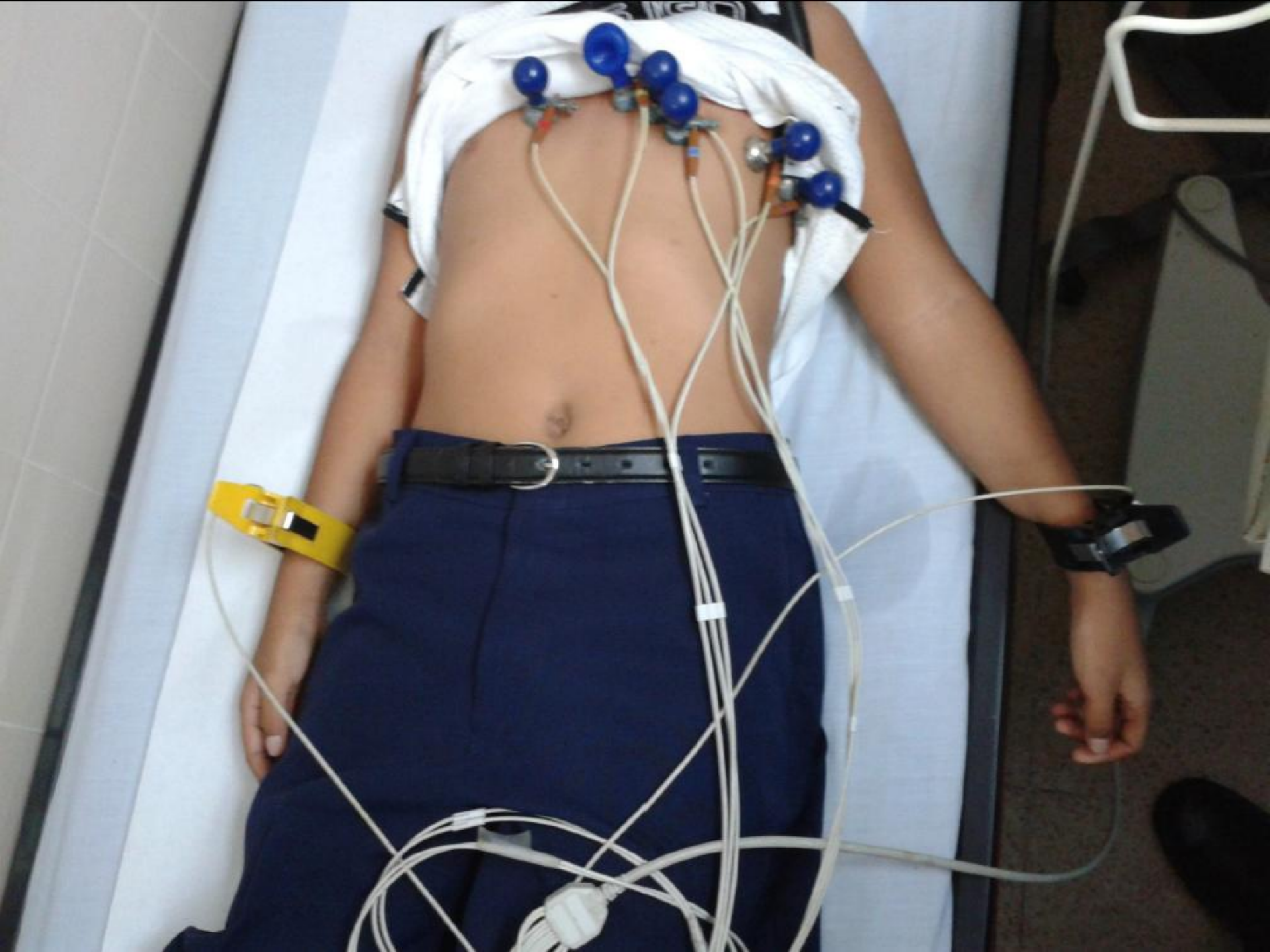
Mayo Clin Proc. 2008;83(11):1280-1293

- La mortalidad varia entre el 0 y 30 % (en el caso de pacientes con cardiopatía)
- Un 25 % de pacientes que consultan por síncope son dados de alta por urgencias sin ECG
- El 28 % de pacientes con ECG anormal y el 40 % de los mismos y cardiopatía estructural también son dados de alta

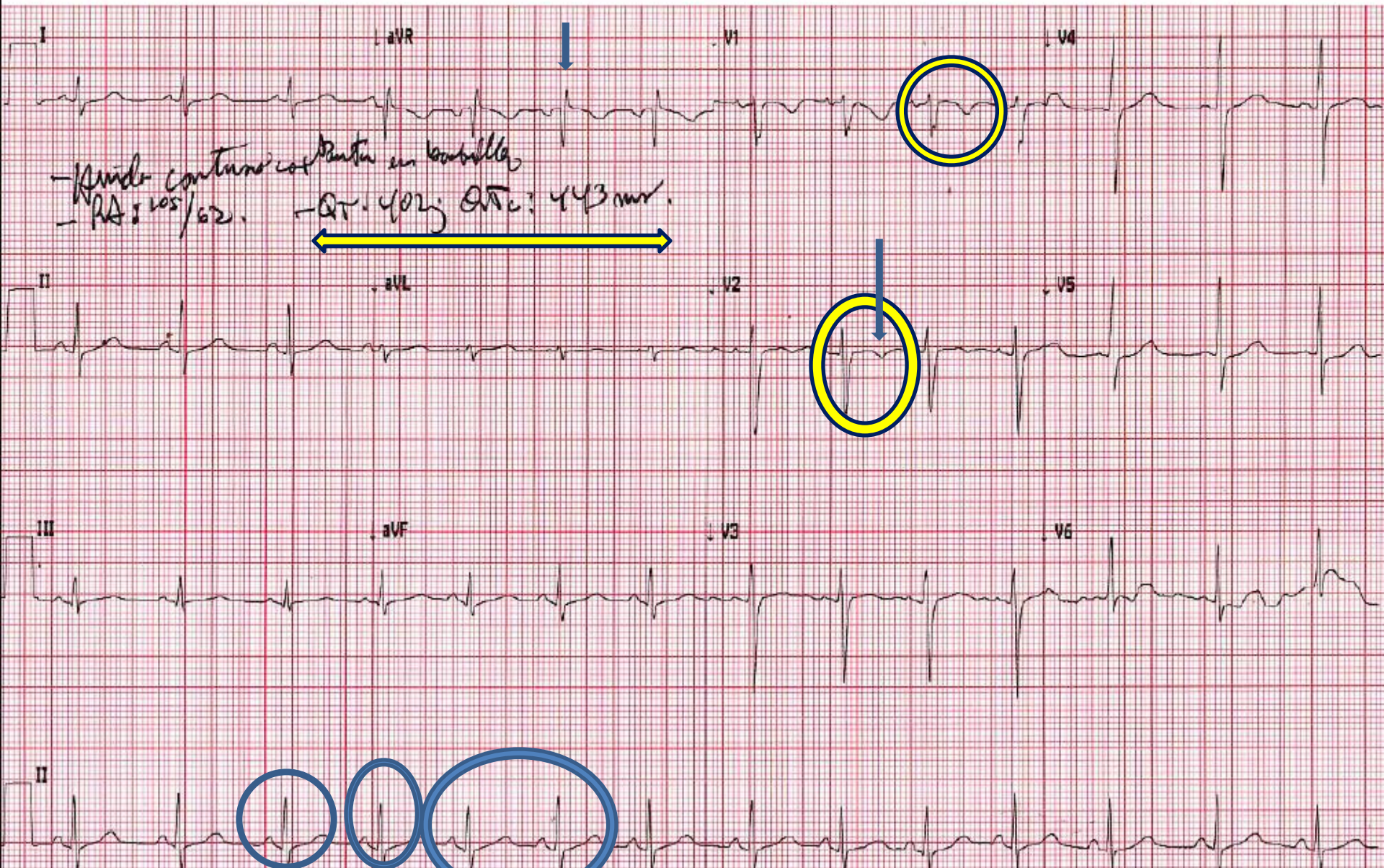
Eur Heart J.2009;30:2631
Emerg Med Clin N Am.2010;28:487
Emerg Med J. 2006;23:589-594
Scot. Med . 1999;44:155-7

CAUSAS COMUNES POR EDAD

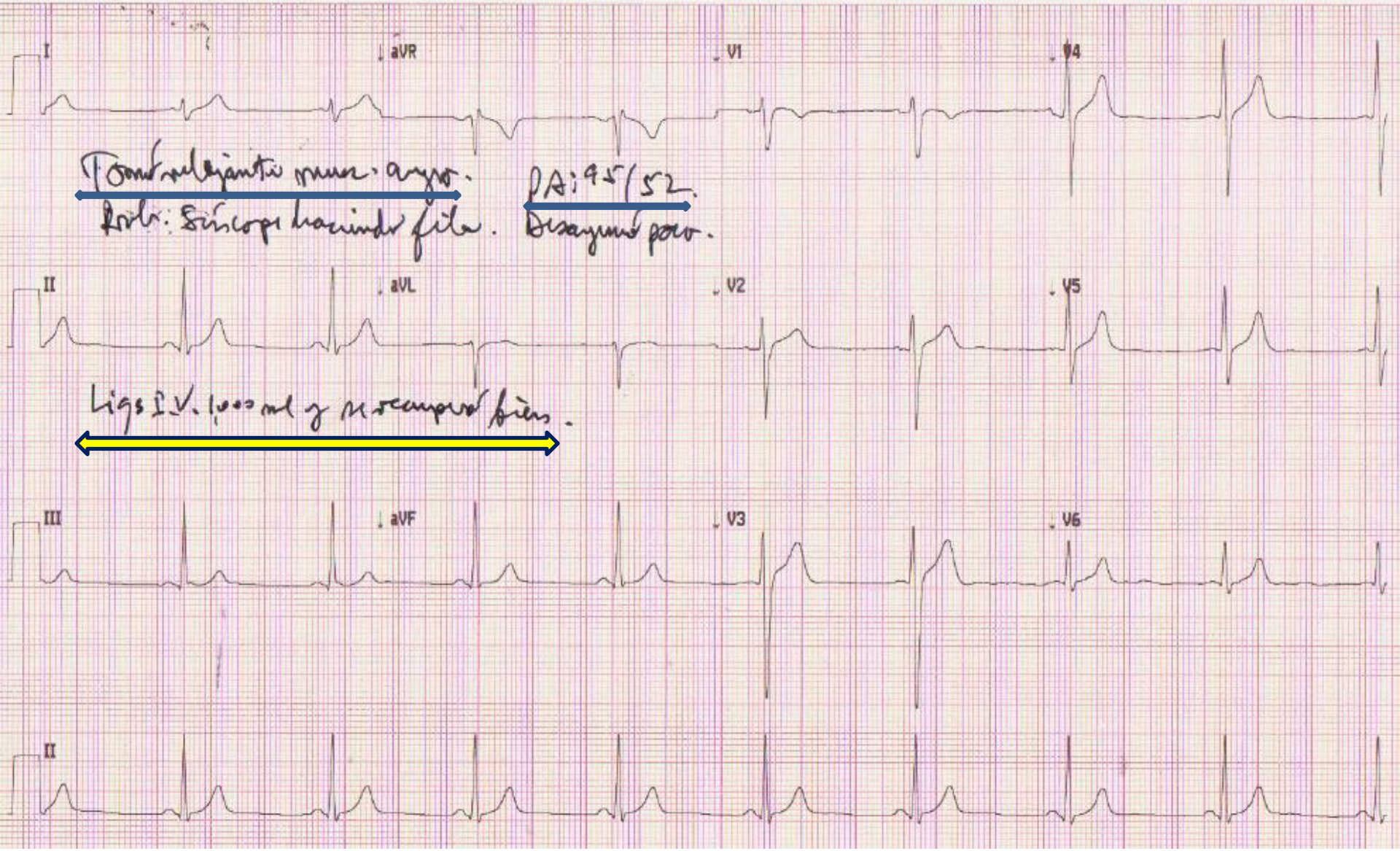
- PEDIATRICOS Y JOVENES
  - Vasovagal
  - QT largo
  - Síndrome de WPW
  - Situacional (micción, defecación, tos, etc.)
  - Crisis de hipoxia en cardiopatía congénita cianótcia



M, 9a, con síncopes frecuentes de larga data



M, 17ª, con probable síncope haciendo fila.



15 Evaluación interdisciplinaria del niño con síncope

Marcela Borghini, Cristina Domínguez, Jorge Scaglione
Unidad de Síncope Hospital General de Niños Dr. Pedro de Elizalde

Introducción

Síncope es la pérdida transitoria de la conciencia. Es un síntoma de alta prevalencia en niños y representa un motivo de consulta frecuente en los servicios de emergencias. Estos episodios resultan en una experiencia traumática para el niño y su familia y en ocasiones se producen lesiones secundarias a las caídas. El 85% de los síncopes tienen un curso benigno; sin embargo, cuando los episodios son recurrentes (30%) suele verse muy afectada la calidad de vida de estos niños.

Epidemiología

En la población pediátrica, el síncope ocurre al menos una vez en el 15% al 50% de los niños, antes de la adolescencia[3]. La edad media de presentación es a los 15 años, siendo más frecuente en el sexo femenino, relación 2/1[4]. La forma clínica que predomina es el síncope reflejo (vasovagal).

1.- Pacientes con bajo riesgo de padecer síncope de origen cardíaco

La estrategia diagnóstica en estos pacientes se plantea en función de la tasa de recidivas y la calidad de vida. No se recomiendan exámenes complementarios complejos (ej.: TAC cerebral, EEF) ya que estos raramente resultan positivos. Se sabe que estos pacientes dejarán de tener episodios sincopales en la edad adulta.

2.- Pacientes con datos compatibles de síncope cardiogénico o con factores de riesgo de eventos cardiovasculares graves a corto plazo o incluso de muerte súbita cardíaca

Este grupo incluye a pacientes a disfunción sistólica del ventrículo izquierdo o antecedentes familiares de muerte súbita por canalopatías (ej.: familias con síndrome de QT prolongado). La mayoría de estos pacientes son candidatos a CDI.

Criterios de internación

Criterios para admisión hospitalaria para diagnóstico o tratamiento.

Recomendado para diagnóstico

- Sospecha o enfermedad cardíaca conocida.
- Alteraciones del ECG que sugieran síncope arrítmico.
- Síncope durante el ejercicio.
- Síncope causante de daño físico.
- Historia familiar de muerte súbita.

Recomendado para tratamiento

Arritmias cardíacas.

Síncope secundario a enfermedad cardíaca o cardiopulmonar.Cuando estaba planeado el implante de MP o CDI.

Ocasionalmente puede ser necesario admitirlo.Sin enfermedad cardíaca pero presenta inicio de palpitaciones antes del síncope o alta sospecha de síncope cardíaco.Síncope en posición supina.

Episodios frecuentes y recurrentes.

El objetivo del tratamiento del niño con síncope es prevenir las recurrencias, evitar lesiones y prolongar la sobrevida. La importancia de estos 3 objetivos dependerá de la causa del síncope.

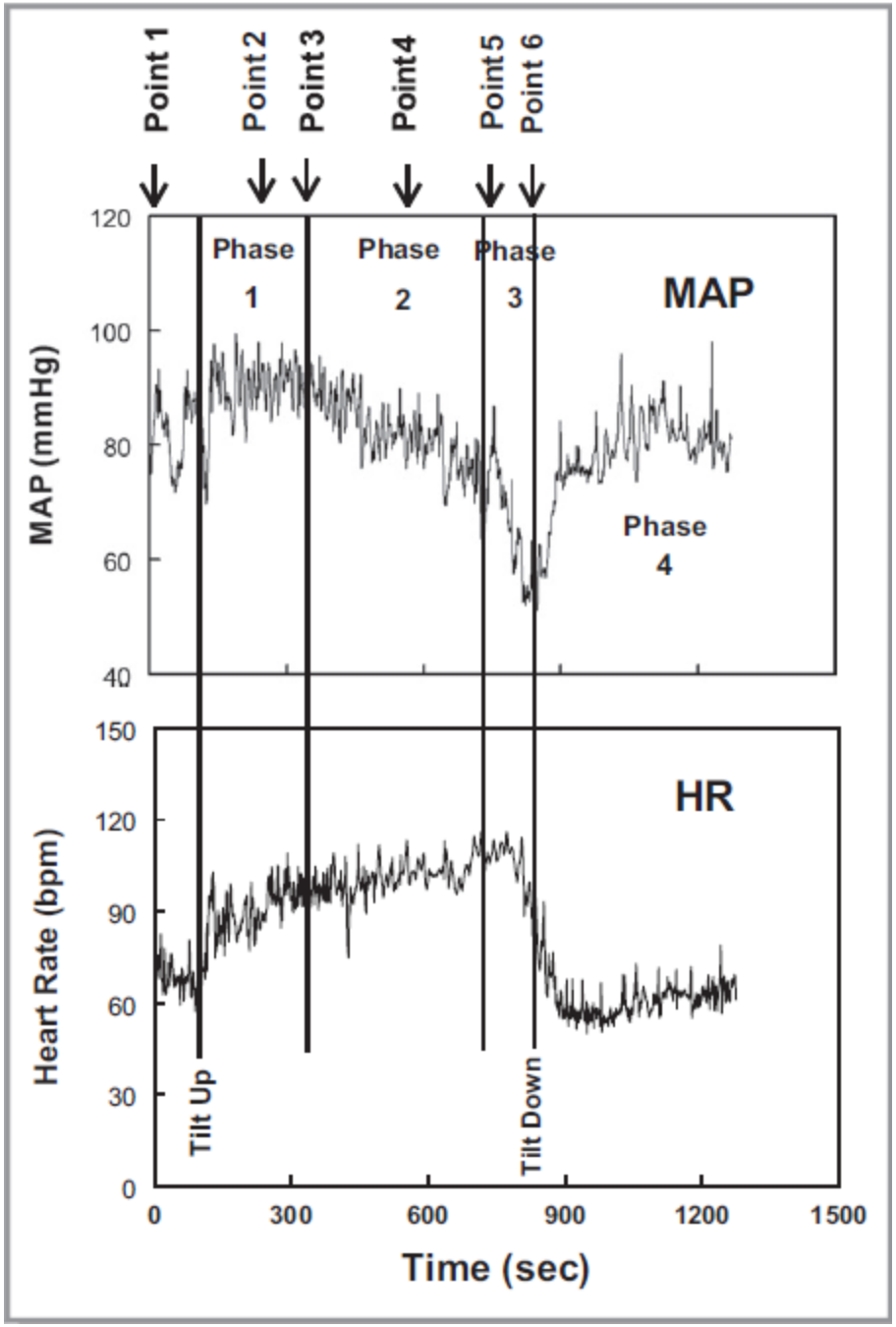
Mechanisms of Vasovagal Syncope in the Young: Reduced Systemic Vascular Resistance Versus Reduced Cardiac Output

Julian M. Stewart, MD, PhD; Marvin S. Medow, PhD; Richard Sutton, MB, BS, DSc; Paul Visintainer, PhD; David L. Jardine, FRACP, MD; Wouter Wieling, MD, PhD

Background—Syncope is a sudden transient loss of consciousness and postural tone caused by cerebral hypoperfusion. The most common form is vasovagal syncope (VVS). Presyncopal progressive early hypotension in older VVS patients is caused by reduced cardiac output (CO); younger patients have reduced systemic vascular resistance (SVR). Using a priori criteria for reduced CO (↓CO) and SVR (↓SVR), we studied 48 recurrent young fainters comparing subgroups of VVS with VVS-↓CO, VVS-↓SVR, and both VVS-↓CO&↓SVR.

Conclusions—Both ↓CO and ↓SVR occur in young VVS patients. ↓SVR is predominant in VVS and is caused by impaired splanchnic vasoconstriction. (*J Am Heart Assoc.* 2017;6:e004417. DOI: 10.1161/JAHA.116.004417.)

Upright posture causes subdiaphragmatic gravitational blood pooling, primarily within the venous system, that reduces central blood volume.[3] In the absence of skeletal muscle pump activity, this reduces venous return and cardiac output (CO). Blood pressure is maintained by baroreceptor-mediated increases in systemic vascular resistance (SVR), passive elastic recoil of venous blood, active splanchnic venoconstriction,[4] and an increase in heart rate (HR). Splanchnic vaso- and venoconstriction are integral to maintaining upright blood pressure (BP).[5] A representative time course of changes in BP and HR for a young VVS patient during 70-degree upright tilt testing is shown in Figure 1.


Point 1
Point 2
Point 3
Point 4
Point 5
Point 6
Phase 1
Phase 2
Phase 3
Phase 4
MAP
MAP (mmHg)
120
100
80
60
40
HR
Heart Rate (bpm)
150
120
90
60
30
0
Tilt Up
Tilt Down
0
300
600
900
1200
1500
Time (sec)

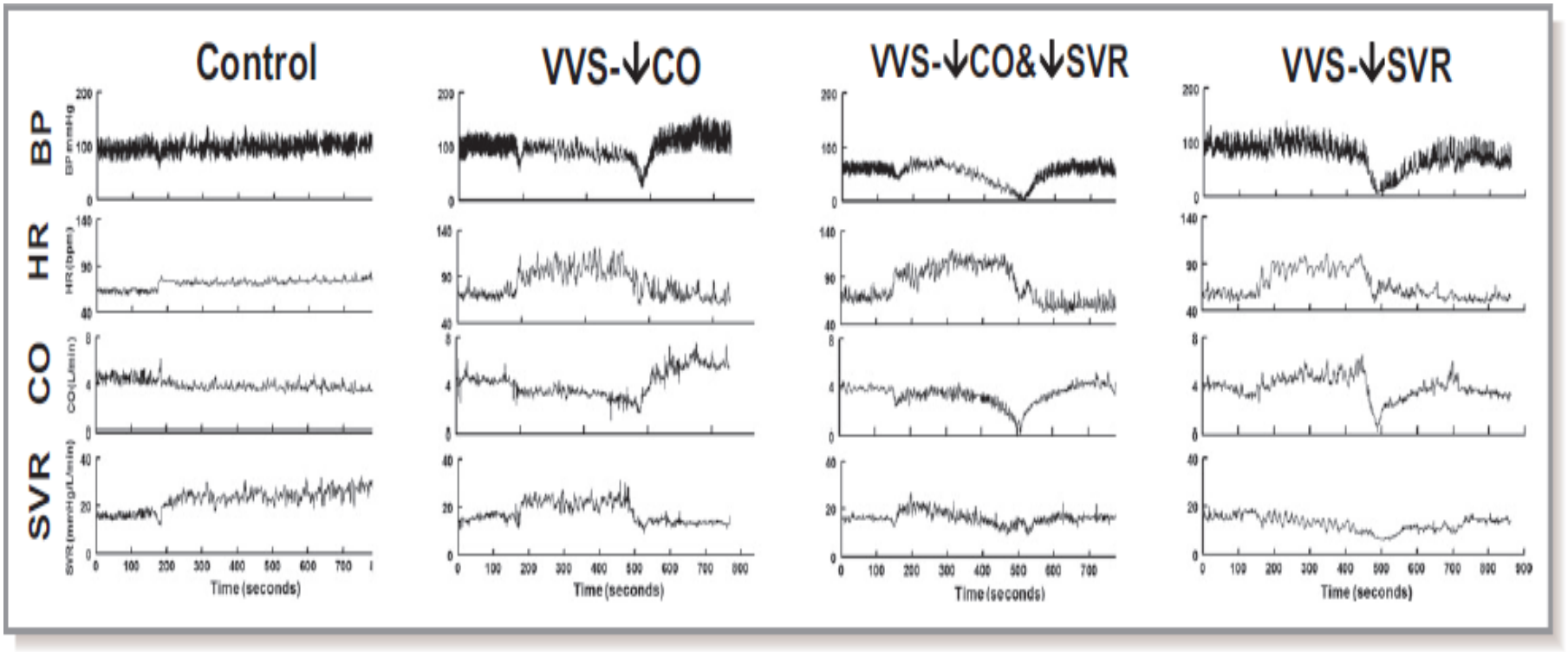


Figure 2. Blood pressure (BP), heart rate (HR), cardiac output (CO), and systemic vascular resistance (SVR) for representative subjects during head-up tilt table testing. Shown from left to right are healthy control subjects (Control), VVS syncope patients who have a decrease in CO but no decrease in SVR designated (VVS-↓CO), VVS syncope patients who have a decrease in CO and SVR during tilt designated (VVS-↓CO &↓SVR), and VVS syncope patients who have a decrease in SVR during tilt but no decrease in CO designated (VVS-↓SVR). There is an increase in HR rate with orthostasis in all subjects. BP first gradually declines, followed by rapid hypotension and bradycardia in all VVS. VVS-↓CO &↓SVR patient has a decrease in CO and initial increase in SVR, which then decreases for the remainder of tilt. CO in the VVS-↓SVR patients trends above baseline whereas SVR declines monotonically. At the time of faint, all VVS patients experienced hypotension followed rapidly by bradycardia whereas SVR and CI decreased. CI indicates cardiac index; VVS, vasovagal syncope.

VVS-↓CO&↓SVR Patients

Patients with VVS-↓CO&↓SVR had splanchnic and calf blood pooling comparable to pooling observed in VVS-↓CO patients, combined with the afterload reduction of VVS-↓SVR. In VVS-↓CO&↓SVR patients, splanchnic arterial vasodilation occurred to a lesser extent than in VVS-↓SVR and estimated venous properties appear to be similar to control. The combined effects of splanchnic pooling and reduced SVR resulted in the earliest faints among the VVS groups.

- ADULTOS EN EDAD MEDIA
  - Vasovagal
  - Situacional
  - Ortostatico
  - Cardiaco
  - Arrítmico

- ANCIANOS
  - Cardiaco
  - Arrítmico
  - Hipotensión ortostática
  - Síndrome del seno carotídeo
  - Vasovagal

CLASIFICACION DE RIESGO SEGÚN ESTUDIO SEEDS

BAJO RIESGO

- Edad menor de 50 años
- Sin historia de enfermedad cardiovascular
- Síntomas sugestivos de síncope vasovagal
- Hallazgos anormales en evaluación cardiovascular
- Hallazgos electrocardiográficos normales

RIESGO INTERMEDIO

- Edad igual o mayor a 50 años
- Historia de: enfermedad coronaria, Infarto de miocardio, insuficiencia cardiaca crónica, cardiomiopatía, historia de estar tomando medicamentos cardiológicos.
- Bloqueo de rama u ondas Q sin cambios agudos
- Historia de muerte súbita en familiares antes de los 50 años
- Síntomas no indicativos de síncope vasovagal
- Dispositivos cardiacos sin malfuncionamiento
- Sospecha medica de síncope de origen cardiaco

Alto riesgo.

- Dolor precordial compatible con síndrome coronario agudo
- Signos de falla cardiaca
- Enfermedad valvular moderada a severa
- Historia de arritmias ventriculares
- Hallazgos electrocardiográficos de isquemia aguda
- QT c prolongado mayor de 500 ms
- Bloqueo trifascicular, pausas mayores a 2,5 seg.
- Bradicardia sinusal persistente entre 40 y 60 lpm
- Fibrilación auricular o taquicardia ventricular no sostenida sin síntomas.
- Dispositivos cardiacos con disfunción.

Scores de Alto Riesgo (Estratificación en SU)

1) Score de OESIL 2 Study. Italia.

2) Score de Boston. 2007.

3) Score de San Francisco. 2006.

4) Score de ROSE. 2010. Reino Unido.

¿Qué factores tienen en común todos los scores para determinar alto riesgo ?

- Antecedente de cardiopatía o muerte súbita
- Cardiopatía estructural
- Anormalidades en el ECG
- Síncope sin prodromos, en ejercicio o acostado
- Signos vitales anormales, signos de sangrado agudo
- Dolor precordial asociado a síncope

CONCLUSIONES

- El paciente con síncope en la sala de emergencias constituye un reto diagnóstico para el médico .
- Es muy importante realizar un adecuado interrogatorio y examen físico minucioso.
- Se debe realizar siempre un ECG y es útil realizar masaje del seno carotídeo.
- Se debe tratar de establecer la etiología del sincope pero más importante es evaluar al paciente de alto riesgo
- El paciente con criterios de alto riesgo debe ser internado.

2017 ACC/AHA/HRS Guideline for the Evaluation and Management of Patients With Syncope

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society

Developed in Collaboration With the American College of Emergency Physicians and Society for Academic Emergency Medicine

Endorsed by the Pediatric and Congenital Electrophysiology Society

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Table 3. Relevant Terms and Definitions*

Term	Definition/Comments and References
Syncope	A symptom that presents with an abrupt, transient, complete loss of consciousness, associated with inability to maintain postural tone, with rapid and spontaneous recovery. The presumed mechanism is cerebral hypoperfusion (24,30). There should not be clinical features of other nonsyncope causes of loss of consciousness, such as seizure, antecedent head trauma, or apparent loss of consciousness (i.e., pseudosyncope) (24,30).
Loss of consciousness	A cognitive state in which one lacks awareness of oneself and one's situation, with an inability to respond to stimuli.
Transient loss of consciousness	Self-limited loss of consciousness (30) can be divided into syncope and nonsyncope conditions. Nonsyncope conditions include but are not limited to seizures, hypoglycemia, metabolic conditions, drug or alcohol intoxication, and concussion due to head trauma. The underlying mechanism of syncope is presumed to be cerebral hypoperfusion, whereas nonsyncope conditions are attributed to different mechanisms.
Presyncope (near-syncope)	The symptoms before syncope. These symptoms could include extreme lightheadedness; visual sensations, such as "tunnel vision" or "graying out"; and variable degrees of altered consciousness without complete loss of consciousness. Presyncope could progress to syncope, or it could abort without syncope.
Unexplained syncope (syncope of undetermined etiology)	Syncope for which a cause is undetermined after an initial evaluation that is deemed appropriate by the experienced healthcare provider. The initial evaluation includes but is not limited to a thorough history, physical examination, and ECG.

Cardiac (cardiovascular) syncope	Syncope caused by bradycardia, tachycardia, or hypotension due to low cardiac index, blood flow obstruction, vasodilatation, or acute vascular dissection (35,36).
Noncardiac syncope	Syncope due to noncardiac causes which include reflex syncope, OH, volume depletion, dehydration, and blood loss (35).
Reflex (neurally mediated) syncope	Syncope due to a reflex that causes vasodilation, bradycardia, or both (24,30,31).
• **Vasovagal syncope (VVS)**	The most common form of reflex syncope mediated by the vasovagal reflex. VVS 1) may occur with upright posture (standing or seated or with exposure to emotional stress, pain, or medical settings; 2) typically is characterized by diaphoresis, warmth, nausea, and pallor; 3) is associated with vasodepressor hypotension and/or inappropriate bradycardia; and 4) is often followed by fatigue. Typical features may be absent in older patients (24). VVS is often preceded by identifiable triggers and/or by a characteristic prodrome. The diagnosis is made primarily on the basis of a thorough history, physical examination, and eyewitness observation, if available.
• **Carotid sinus syndrome**	Reflex syncope associated with carotid sinus hypersensitivity (30). Carotid sinus hypersensitivity is present when a pause ≥3 s and/or a decrease of systolic pressure ≥50 mm Hg occurs upon stimulation of the carotid sinus. It occurs more frequently in older patients. Carotid sinus hypersensitivity can be associated with varying degrees of symptoms. Carotid sinus syndrome is defined when syncope occurs in the presence of carotid sinus hypersensitivity.
• **Situational syncope**	Reflex syncope associated with a specific action, such as coughing, laughing, swallowing, micturition, or defecation. These syncope events are closely associated with specific physical functions.

Orthostatic intolerance	A syndrome consisting of a constellation of symptoms that include frequent, recurrent, or persistent lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, exercise intolerance, and fatigue upon standing. These symptoms can occur with or without orthostatic tachycardia, OH, or syncope (24). Individuals with orthostatic intolerance have ≥1 of these symptoms associated with reduced ability to maintain upright posture.
Orthostatic tachycardia	A sustained increase in heart rate of ≥30 bpm within 10 min of moving from a recumbent to a quiet (nonexertional) standing position (or ≥40 bpm in individuals 12–19 y of age) (24,30,31).
Orthostatic hypotension (OH)	A drop in systolic BP of ≥20 mm Hg or diastolic BP of ≥10 mm Hg with assumption of an upright posture (31).
• **Initial (immediate) OH**	A transient BP decrease within 15 s after standing, with presyncope or syncope (31,32).
• **Classic OH**	A sustained reduction of systolic BP of ≥20 mm Hg or diastolic BP of ≥10 mm Hg within 3 min of assuming upright posture (31).
• **Delayed OH**	A sustained reduction of systolic BP of ≥20 mm Hg (or 30 mm Hg in patients with supine hypertension) or diastolic BP of ≥10 mm Hg that takes >3 min of upright posture to develop. The fall in BP is usually gradual until reaching the threshold (31).
• **Neurogenic OH**	A subtype of OH that is due to dysfunction of the autonomic nervous system and not solely due to environmental triggers (e.g., dehydration or drugs) (33,34). Neurogenic OH is due to lesions involving the central or peripheral autonomic nerves.

Postural (orthostatic) tachycardia syndrome (POTS)	A clinical syndrome usually characterized by all of the following: 1) frequent symptoms that occur with standing (e.g., lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, exercise intolerance, and fatigue); and 2) an increase in heart rate of ≥30 bpm during a positional change from supine to standing (or ≥40 bpm in those 12–19 y of age); and 3) the absence of OH (>20 mm Hg reduction in systolic BP). Symptoms associated with POTS include those that occur with standing (e.g., lightheadedness, palpitations); those not associated with particular postures (e.g., bloating, nausea, diarrhea, abdominal pain); and those that are systemic (e.g., fatigue, sleep disturbance, migraine headaches) (37). The standing heart rate is often >120 bpm (31,38-42).
Psychogenic pseudosyncope	A syndrome of *apparent* but not true loss of consciousness that may occur in the absence of identifiable cardiac, reflex, neurological, or metabolic causes (30).

*These definitions are derived from previously published definitions from scientific investigations, guidelines, expert consensus statements, and Webster dictionary after obtaining consensus from the WC

BP indicates blood pressure; ECG, electrocardiogram; OH, orthostatic hypotension; POTS, postural tachycardia syndrome; and VVS, vasovagal syncope.

Figure 1. Syncope Initial Evaluation

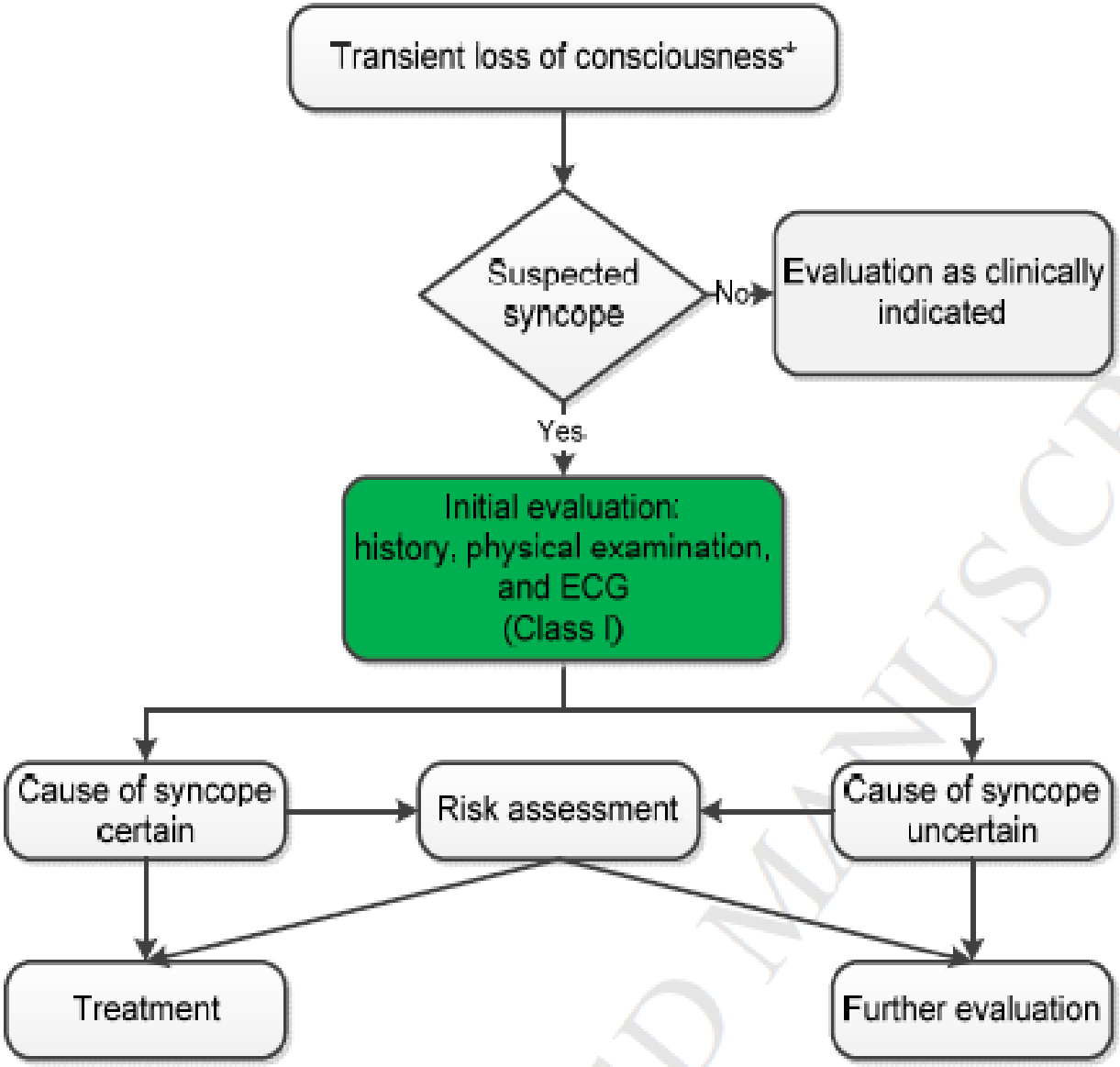
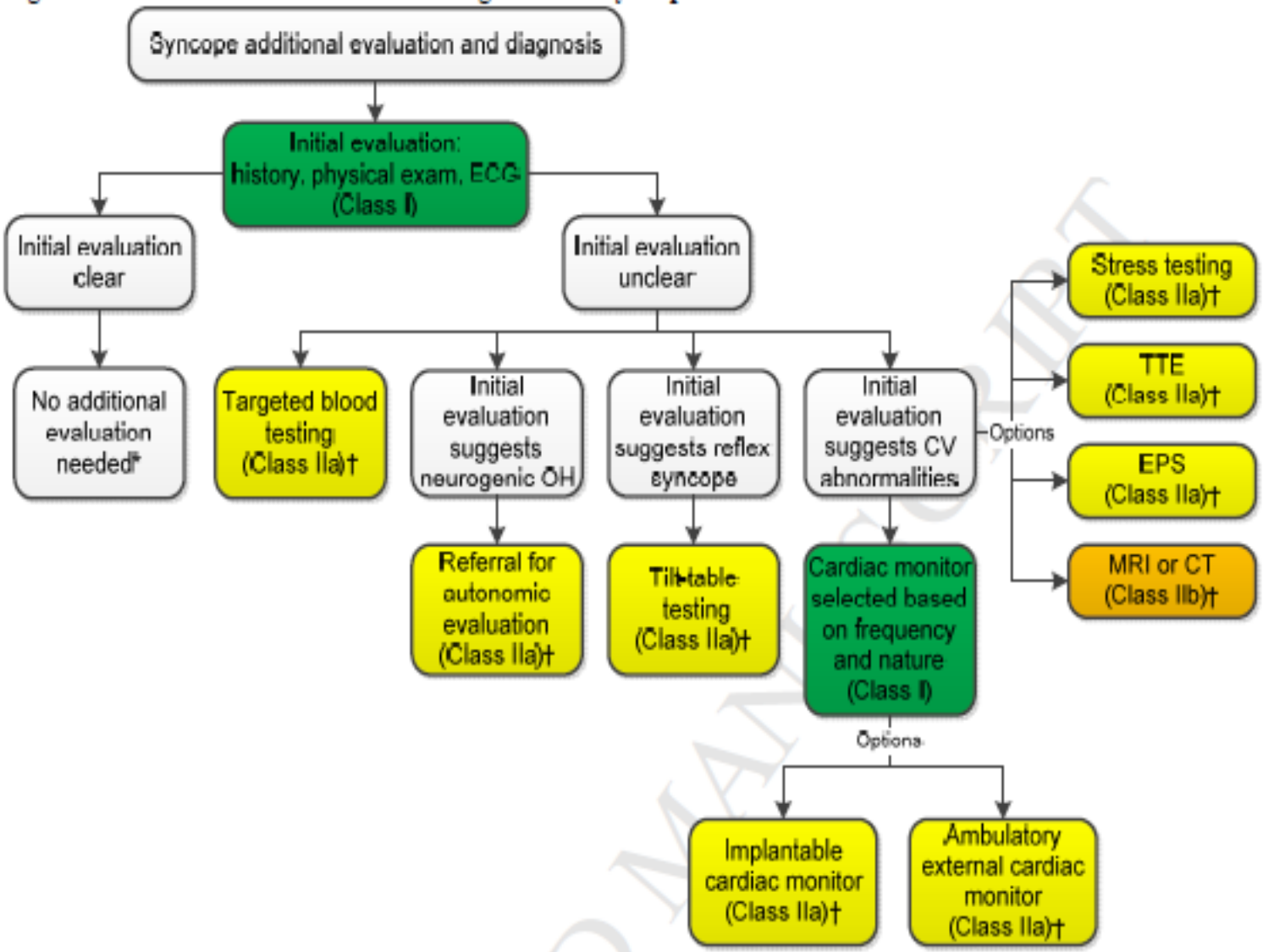


Figure 3. Additional Evaluation and Diagnosis for Syncope



CT indicates computed tomography; CV, cardiovascular; ECG, electrocardiogram; EPS, electrophysiological study; MRI, magnetic resonance imaging; OH, orthostatic hypotension; and TTE, transthoracic echocardiography.

SPAIN

- **Investigadores españoles reducen los síncopes hasta 7 veces**
- **Publicado: 20 Marzo 2017** | Actualidad - Comunicación - Notas de prensa
- http://secardiologia.es/comunicacion/notas-de-prensa/notas-de-prensa-sec/8395-investigadores-espanoles-consiguen-reducir-los-episodios-de-sincopes-hasta-siete-veces
- La celebración de la 66ª edición de la ***Annual Scientific Session del American College of Cardiology*, el congreso de cardiología más importante del continente americano**, marcará un antes y un después en la investigación cardiovascular española.

  Por primera vez, **SPAIN, un trabajo español de la Agencia de Investigación de la Sociedad Española de Cardiología,** ha sido escogido para ser presentado en las prestigiosas sesiones conocidas como Late Breaking Clinical Trials, las 30 investigaciones más aclamadas por la comunidad científica que son elegidas entre los más de 4.000 trabajos que se presentan en el congreso“.

SPAIN

"nuestro trabajo, realizado en once hospitales españoles y uno canadiense, ha probado que la ***implantación de un marcapasos*** *se posiciona como tratamiento efectivo para el síncope recurrente en los pacientes mayores de 40 años y* ***cuyos resultados en el test de tabla basculante deben ser positivos y con cardioinhibición".***

SPAIN

"***Entre el 25% y el 30% de estas personas sufre lo que se conoce como el síncope recurrente.***

Nosotros, en nuestro trabajo, nos hemos centrado en aquellos que han sufrido un mínimo de cinco desvanecimientos en toda su vida y al menos dos de ellos en el último año, lo que representa entre el 5% y el 10% de los síncopes recurrentes", explica el Dr. Barón y Esquivias.

SPAIN

"Otro de los aspectos destacados", prosigue el doctor, "*es que, mientras que el tiempo transcurrido hasta el primer síncope en los pacientes con marcapasos era de 29 meses, en los pacientes con las funciones del marcapasos anuladas era solo de nueve meses".*

En general, el síncope se produce por una bradicardia, que genera menos circulación de la sangre hacia el cerebro, lo que provoca el desmayo. *La función del marcapasos es detectar la bradicardia y estimular al corazón para que este recupere su ritmo normal y no se produzca el desmayo.*

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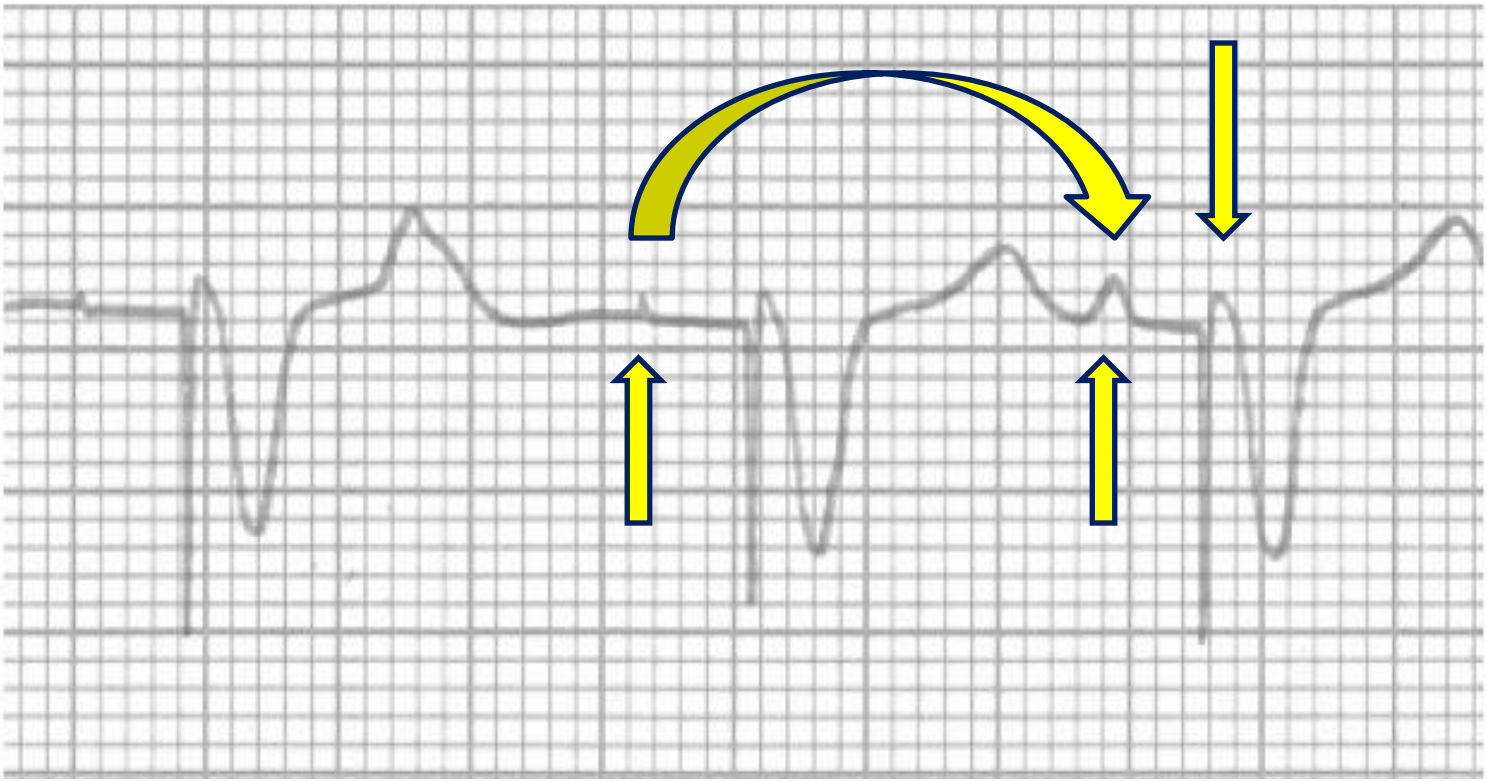
Actualmente, los investigadores están ampliando el estudio con un número mayor de pacientes. Tras este nuevo trabajo, y en el caso de que se confirmen los resultados previos, **se espera que la implantación del marcapasos DDD-CLS se incluya en las guías de práctica clínica como tratamiento en mayores de 40 años con síncopes vasovagales cardio-inhibitorios.**

Marcapasos DDD

- **DDD,R o DDDR**: **estimulación aurículo-ventricular (Bicameral)**, con inhibición del canal auricular por eventos auriculares y del canal ventricular por eventos espontáneos ventriculares, pero con respuesta de gatillo ventricular por evento auricular sensado.
- La frecuencia es determinada por el sensor.

*Marcapasos **DDD** con capturas normales, que muestra la sensibilidad normal del canal auricular, que se inhibe en presencia de un latido auricular precoz, que a su vez, desencadena el estímulo ventricular.*

Nótese cómo la espiga auricular es de magnitud pequeña en esta derivación (D2).




AY COÑO QUE
BUENO QUE LLEGASTE
Ivan Eduardo
SE DESMAYO EL ÁRBOL

COMENTARIOS,

APORTACIONES,

EXPERIENCIAS.

¡MUCHAS

GRACIAS!