



Sociedad Médica de Santiago

Sociedad Chilena de Medicina Interna

150 años al Servicio de la Medicina

IX CURSO MEDICINA INTERNA HOSPITALARIA 2019

Bradicardia y Marcapasos Aspectos prácticos para el Hospitalista

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Complejo Asistencial Dr. Sótero del Río



Puntos a tratar

- Generación
- Propagación
- Manejo Médico
- Terapia con dispositivos
 - Indicaciones
 - Nociones generales
 - Complicaciones
- Preguntas



Visible Spectrum



Red

orange

Yellow

Green

Blue

Indiga

Violet



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Generación del impulso

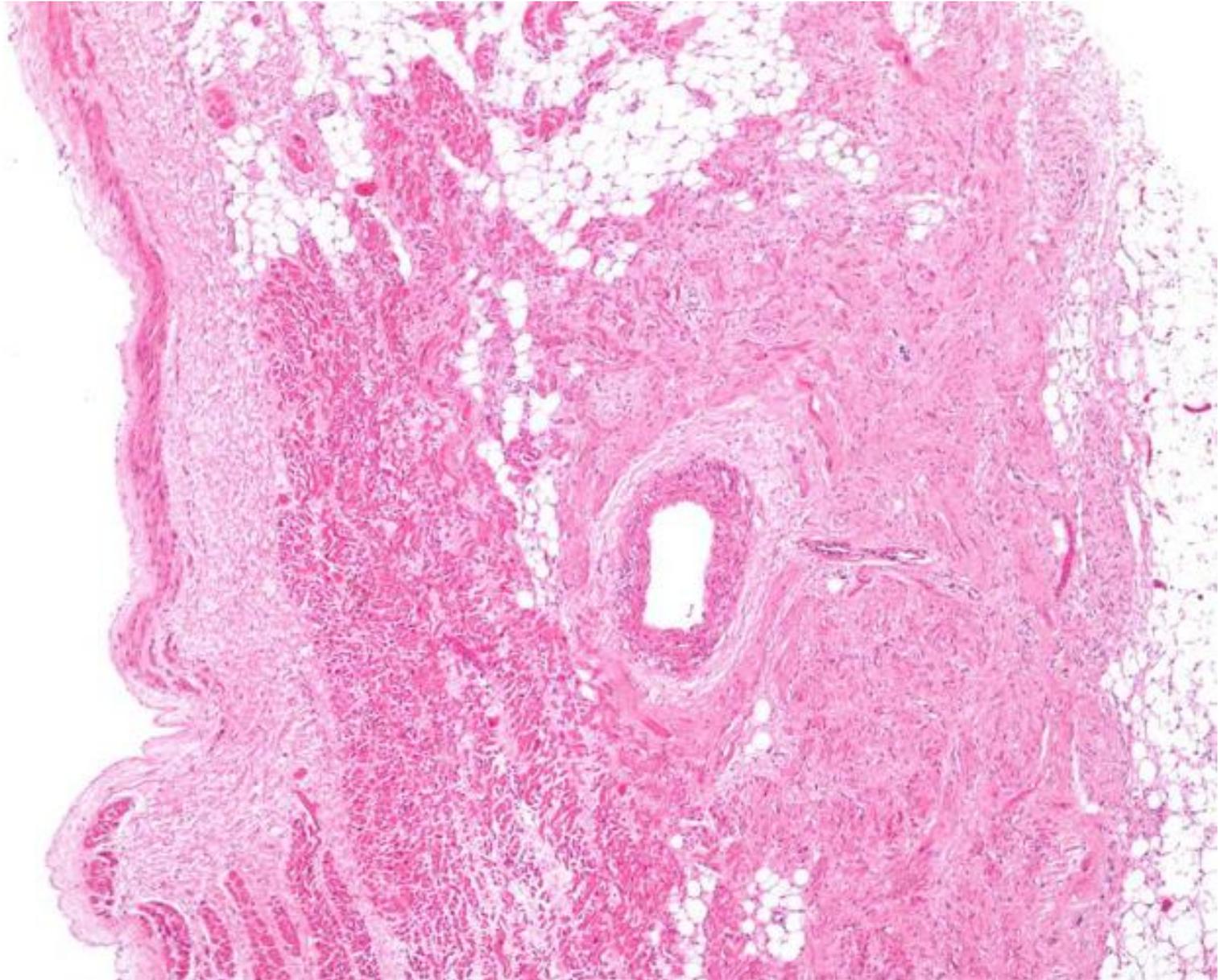




Figure 2: 3D Computer Reconstruction of the Human Heart Sinoatrial Node from Histological and Immunohistochemical Data Demonstrating the Extent of the Sinoatrial Node and Peripheral Pacemaking Tissue

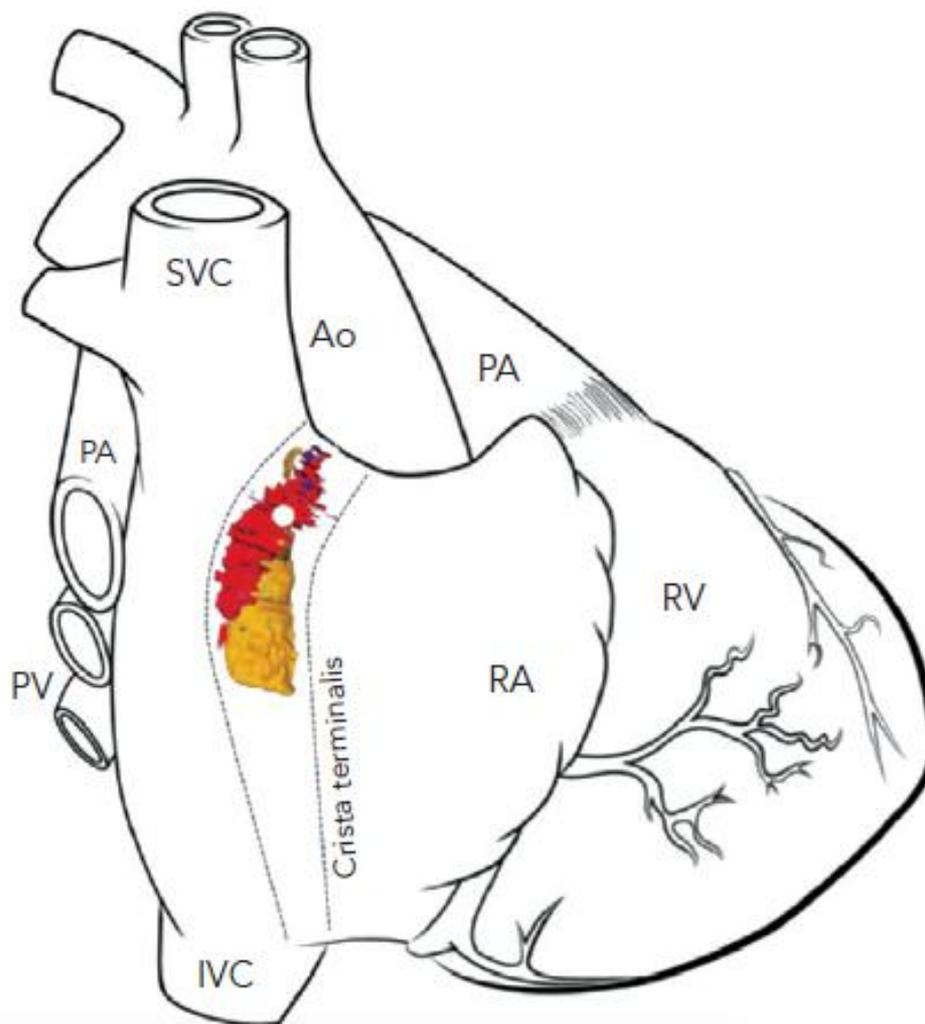
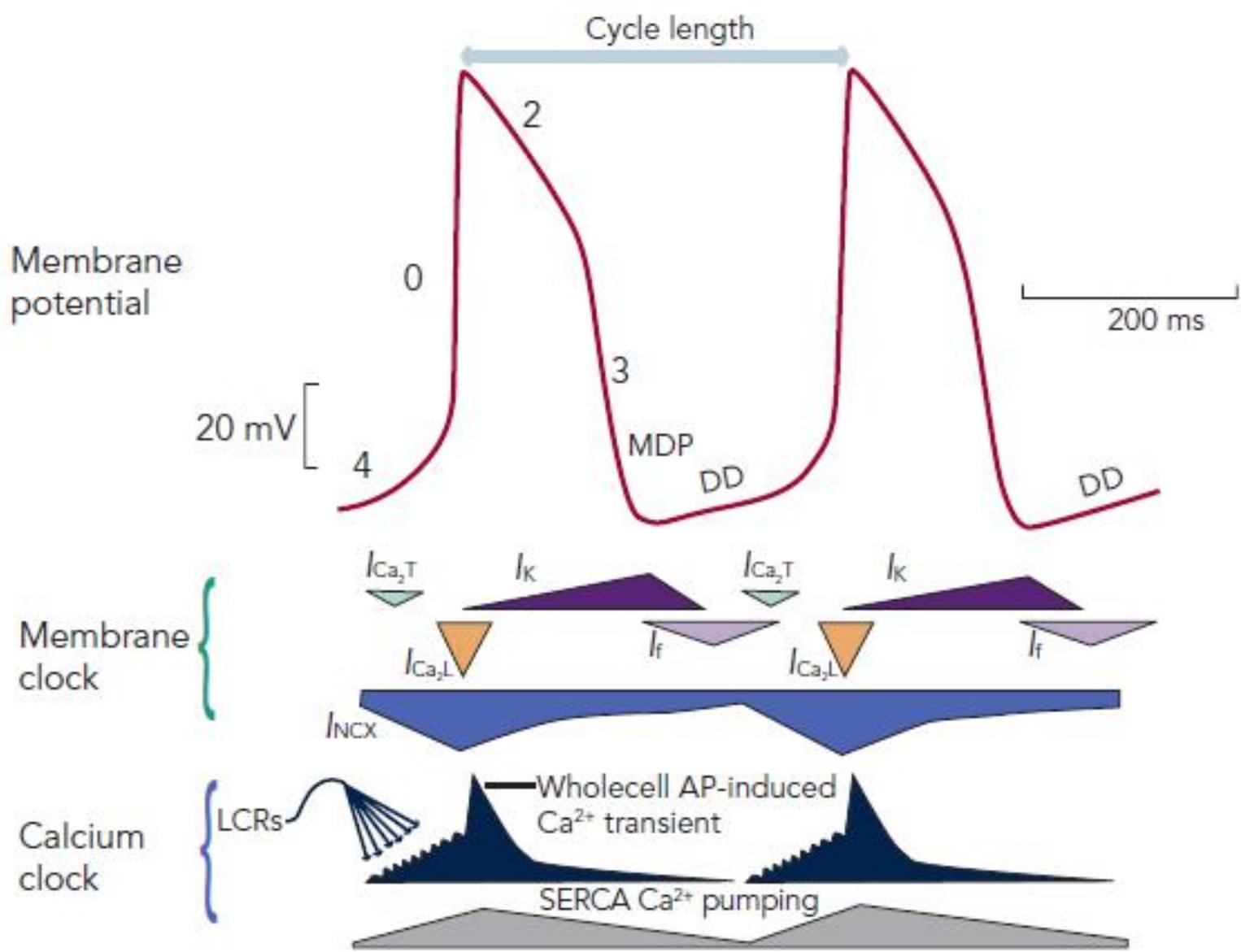
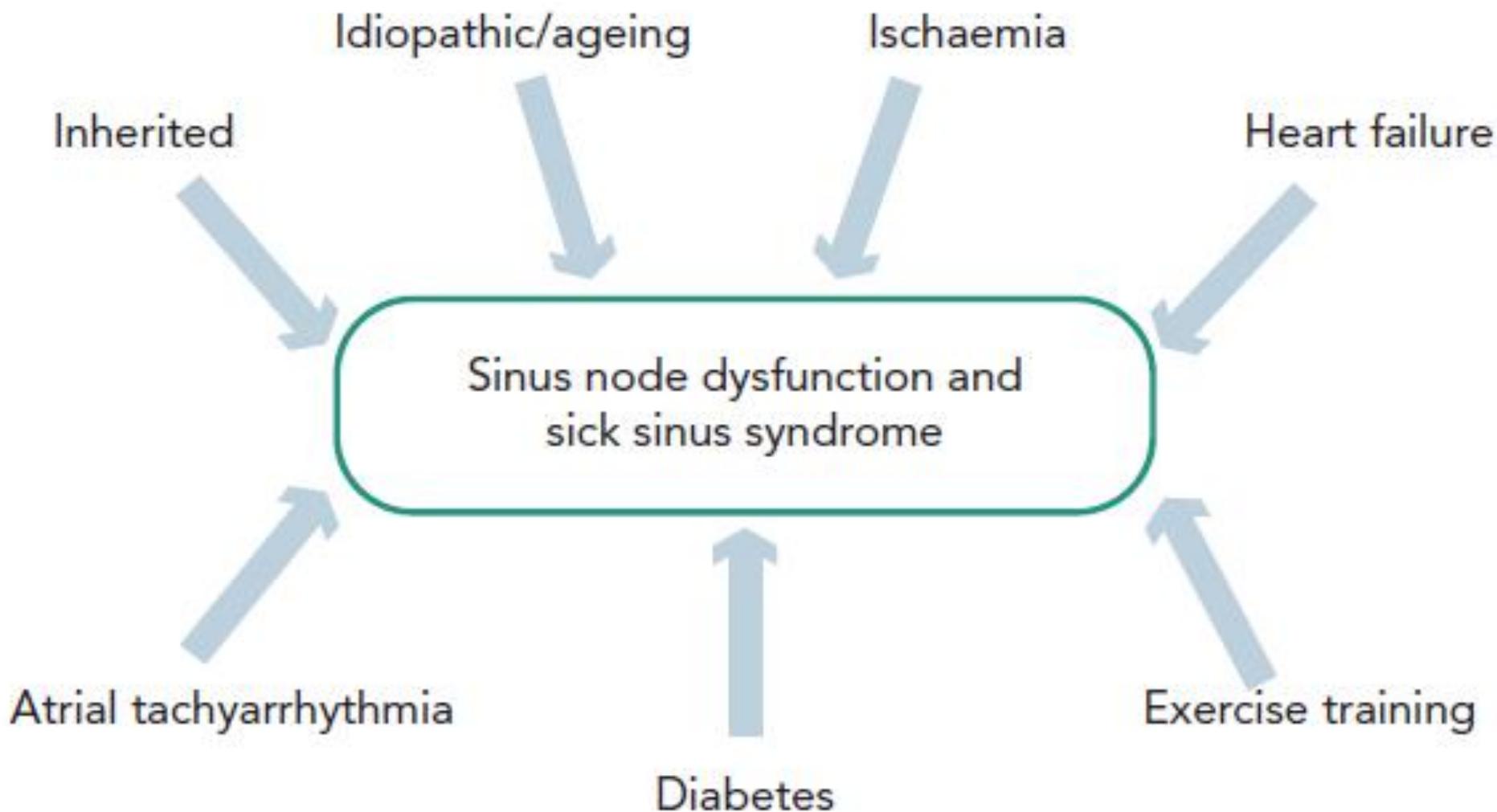




Figure 4: Typical Sinoatrial Node Membrane Action Potentials (Red Trace) and the Timing of Membrane Clock and Ca^{2+} Clock Components





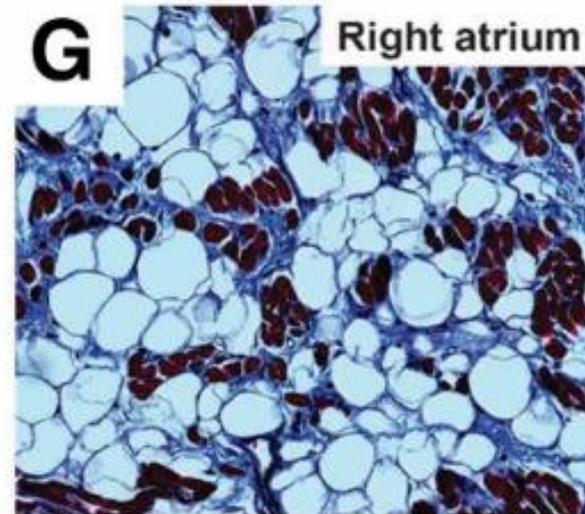
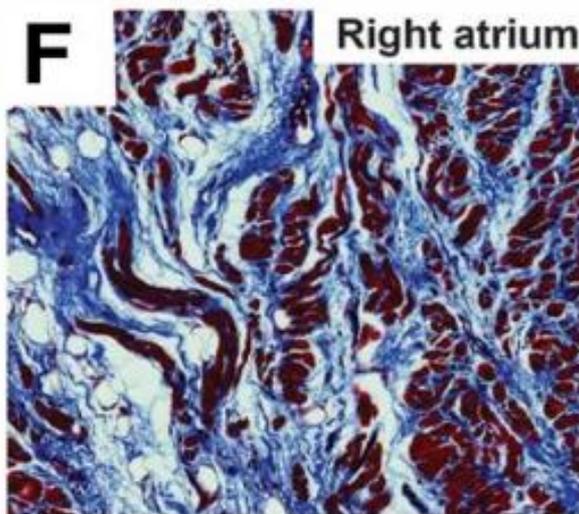
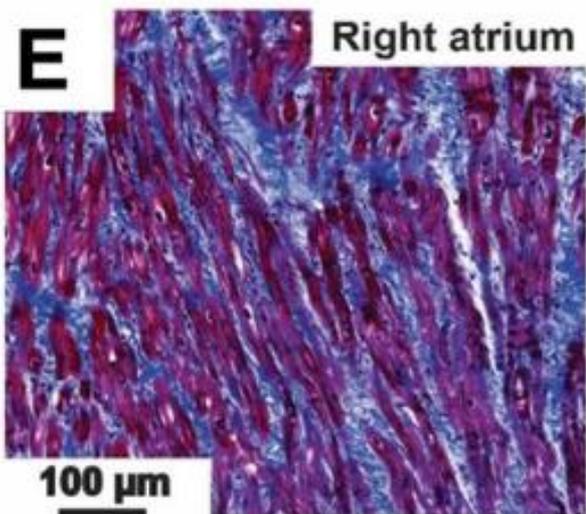
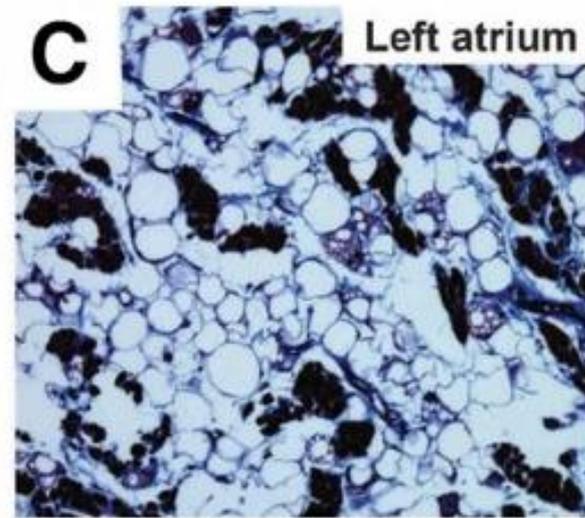
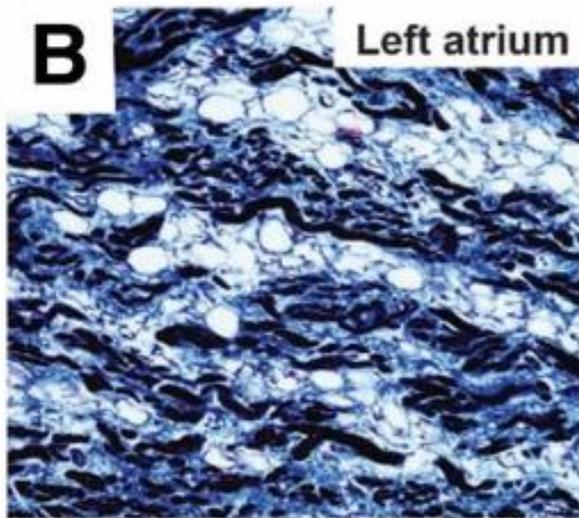
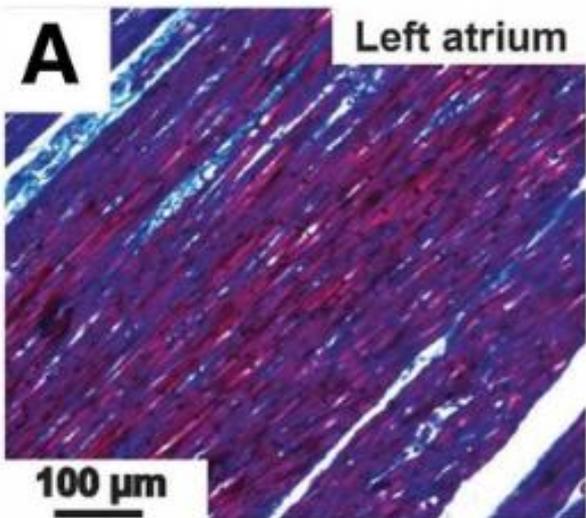




Sinus Rhythm Non-Failing Heart

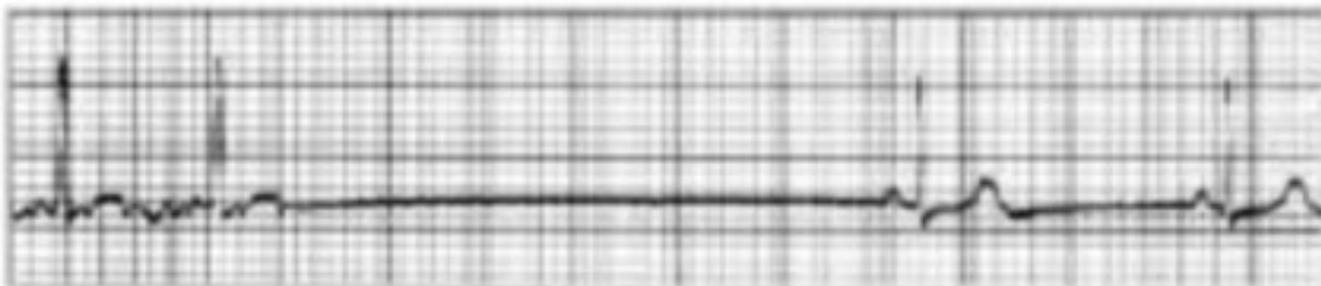
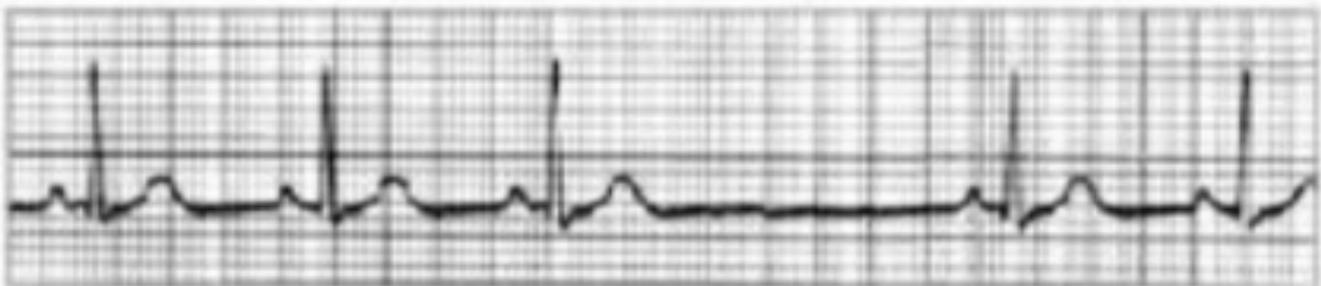
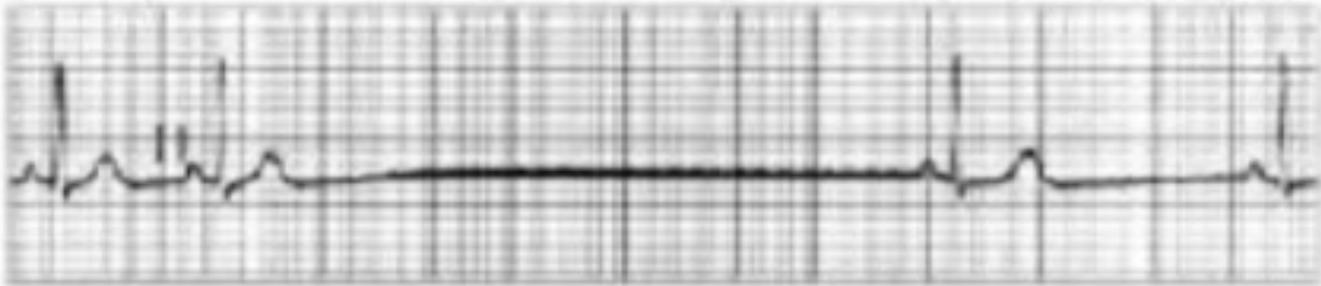
Sinus Rhythm End-stage Heart Failure

Atrial Fibrillation End-stage Heart Failure



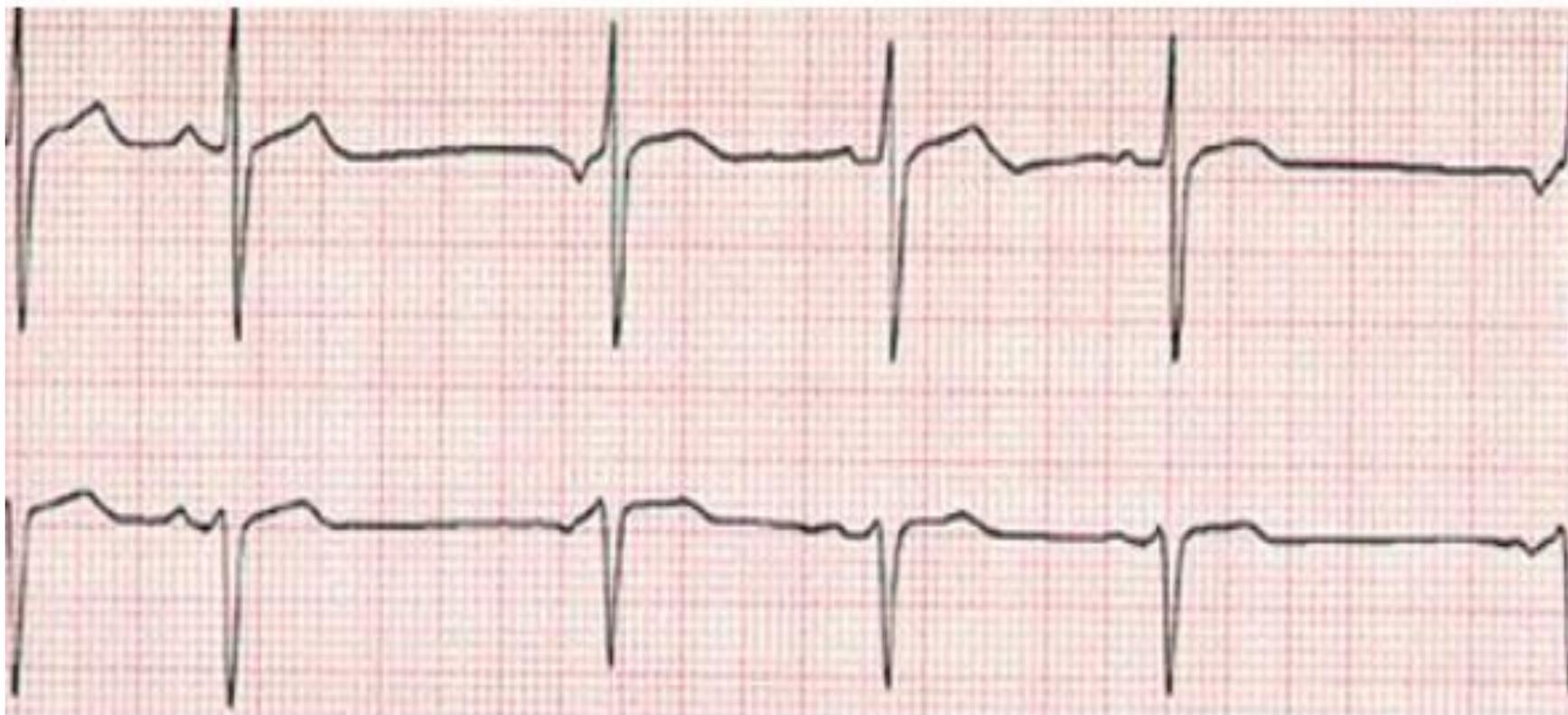


Société
Médicale
des Officiers
de l'Armée





Caso clínico





4. GENERAL EVALUATION OF PATIENTS WITH DOCUMENTED OR SUSPECTED BRADYCARDIA OR CONDUCTION DISORDERS

4.1. History and Physical Examination of Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for History and Physical Examination in Patients With Documented or Suspected Bradycardia or Conduction Disorders

COR	LOE	Recommendation
I	C-EO	1. In patients with suspected bradycardia or conduction disorders a comprehensive history and physical examination should be performed.



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Table 7. Common Potentially Reversible or Treatable Causes of SND^{SS.3.1-1}

Acute myocardial ischemia or infarction ^{SS.3.1-2-SS.3.1-4}
Athletic training ^{SS.3.1-5}
Atrial fibrillation ^{SS.3.1-6}
Cardiac surgery
Valve replacement, ^{SS.3.1-7,SS.3.1-8} maze procedure, ^{SS.3.1-7} coronary artery bypass graft ^{SS.3.1-9,SS.3.1-10}
Drugs or toxins*
Toluene, organophosphates, tetrodotoxin, cocaine ^{SS.3.1-11}
Electrolyte abnormality
Hyperkalemia, ^{SS.3.1-12} hypokalemia, ^{SS.3.1-13} hypoglycemia ^{SS.3.1-14}
Heart transplant: ^{SS.3.1-15} Acute rejection, chronic rejection, remodeling ^{SS.3.1-16,SS.3.1-17}
Hypervagotonia ^{SS.3.1-18,SS.3.1-19}
Hypothermia
Therapeutic (post-cardiac arrest cooling ^{SS.3.1-20}) or environmental exposure ^{SS.3.1-21}
Hypothyroidism ^{SS.3.1-22}
Hypovolemic shock ^{SS.3.1-23}
Hypoxemia, hypercarbia, acidosis ^{SS.3.1-24}
Sleep apnea, respiratory insufficiency (suffocation, drowning, ^{SS.3.1-25} stroke, ^{SS.3.1-26} drug overdose)
Infection ^{SS.3.1-27}
Lyme disease, ^{SS.3.1-28} legionella, psittacosis, typhoid fever, typhus, listeria, ^{SS.3.1-29} malaria, leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre ^{SS.3.1-30}
Medications*
Beta blockers, non-dihydropyridine calcium channel blockers, digoxin, ^{SS.3.1-31} antiarrhythmic drugs, lithium, ^{SS.3.1-32} methylodopa, risperidone, cisplatin, interferon

- 1.- Entren. atlético
- 2.- Fibrilación auricular
- 3.- Hipotiroidismo
- 4.- SAHOS
- 5.- Drogas
Litio
Mdopa, Cloni, Dex
Risperidona



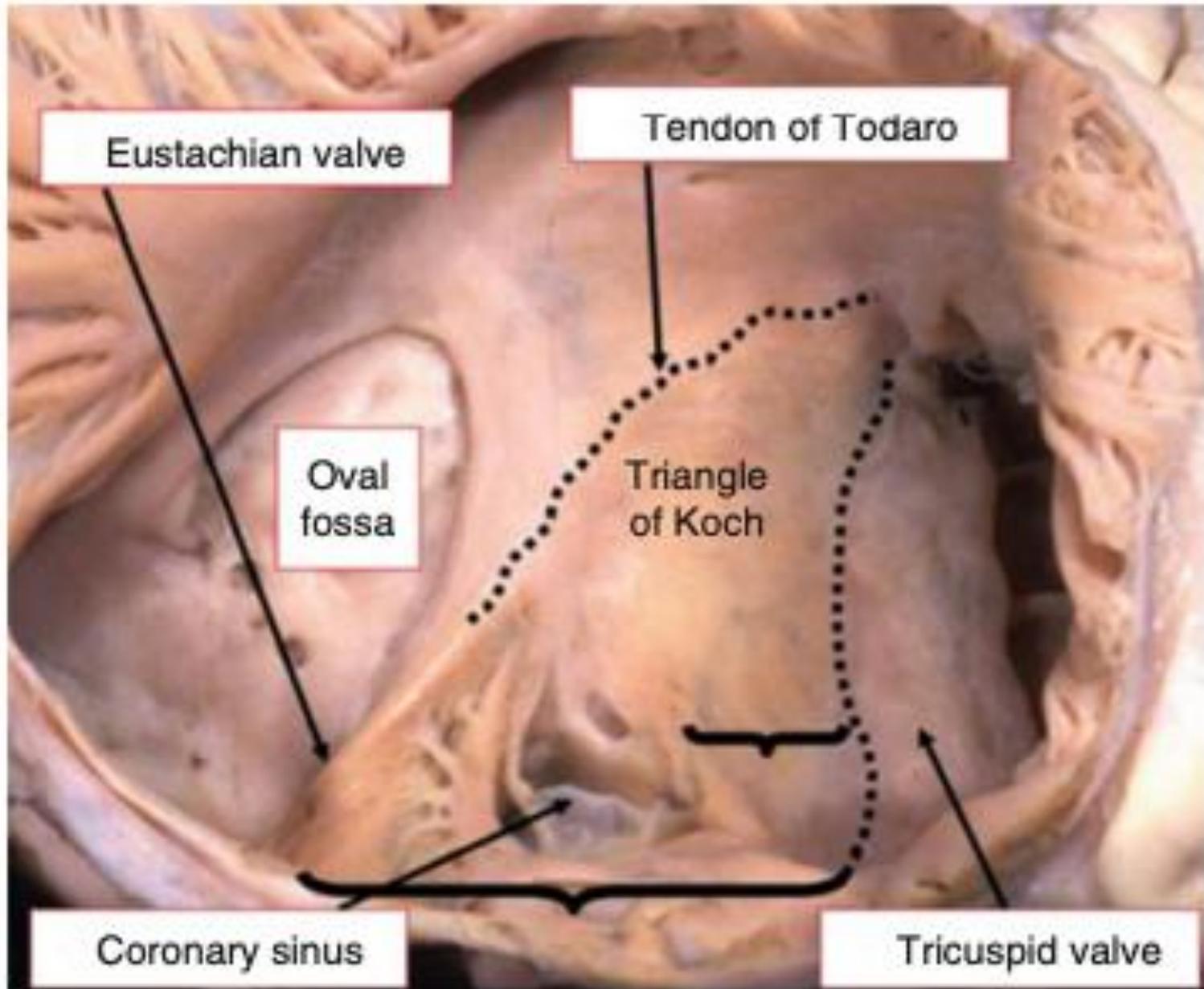
Extrinsic
Autonomic perturbation
Carotid sinus hypersensitivity
Neurally-mediated syncope/presyncope
Physical conditioning
Situational syncope
Cough
Defecation
Glottic stimulation
Medical procedures
Micturition
Vomiting
Sleep (with or without sleep apnea)
Metabolic
Acidosis
Hyperkalemia
Hypokalemia
Hypothermia
Hypothyroidism
Hypoxia

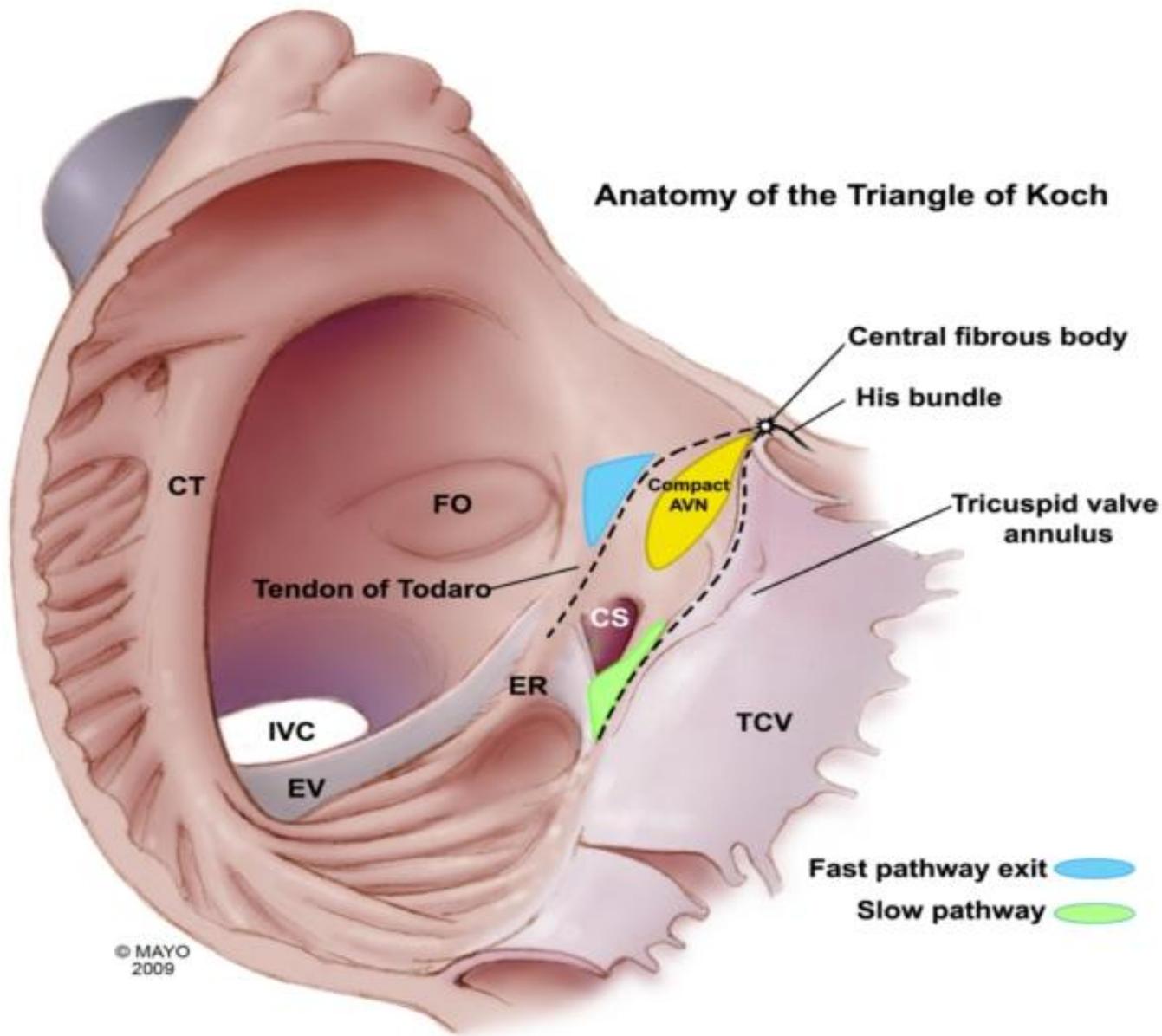
- 1.- Acidosis
- 2.- Hiper – Hipokalemia
- 3.- Hipotermia
- 4.- Hipoxia
- 5.- Infarto

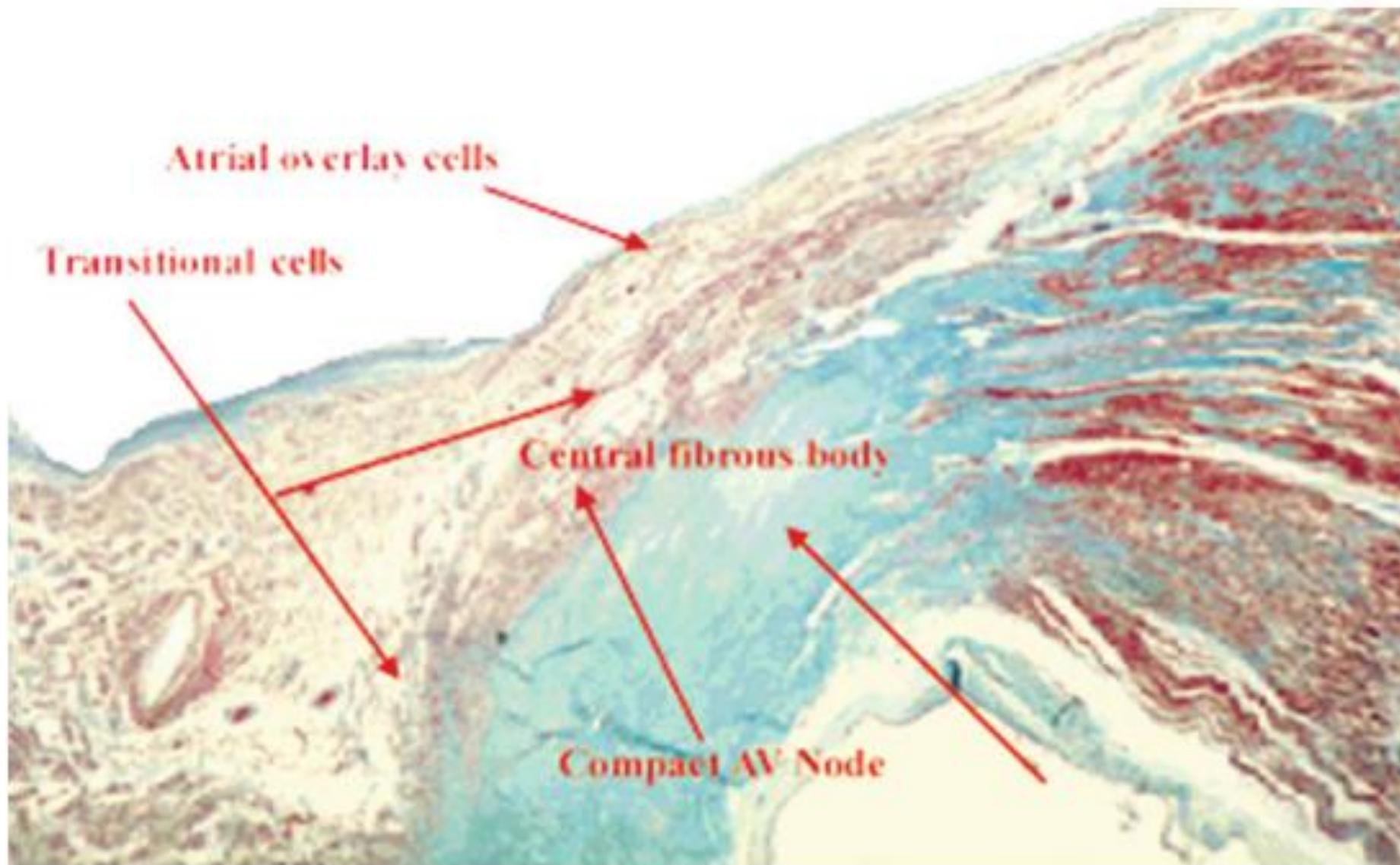


Propagación del impulso



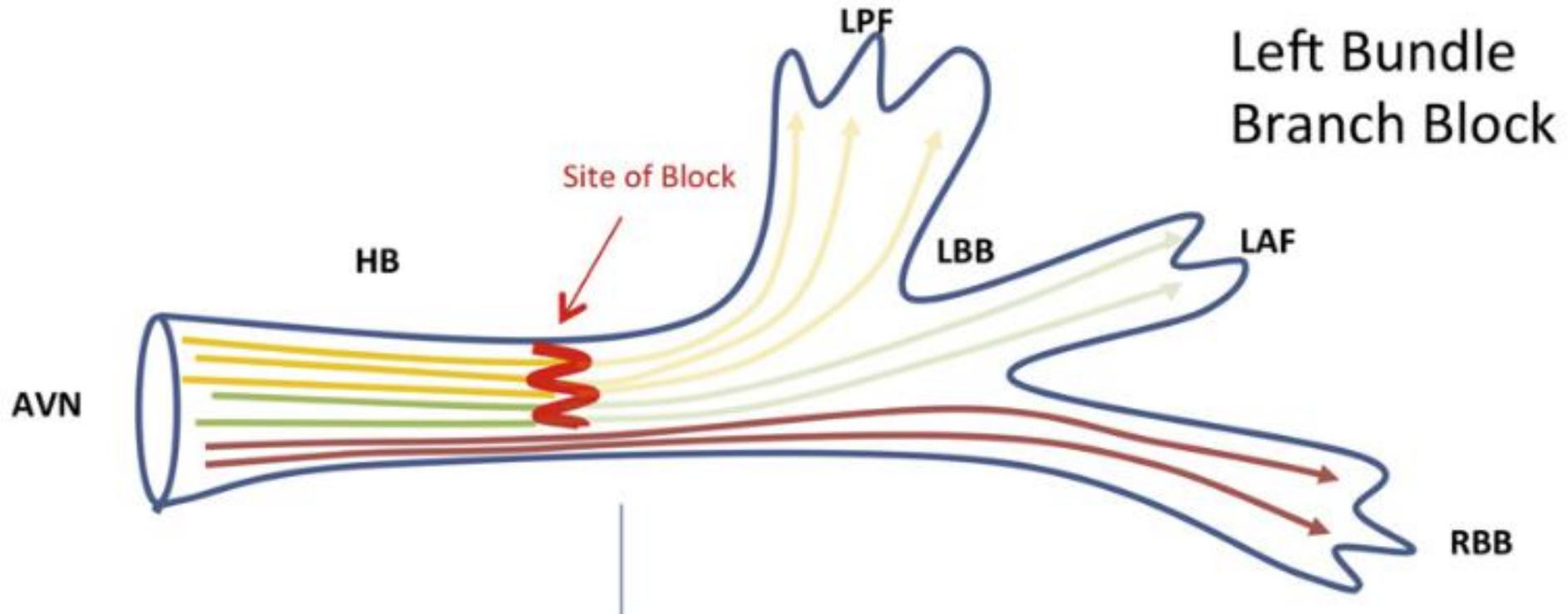


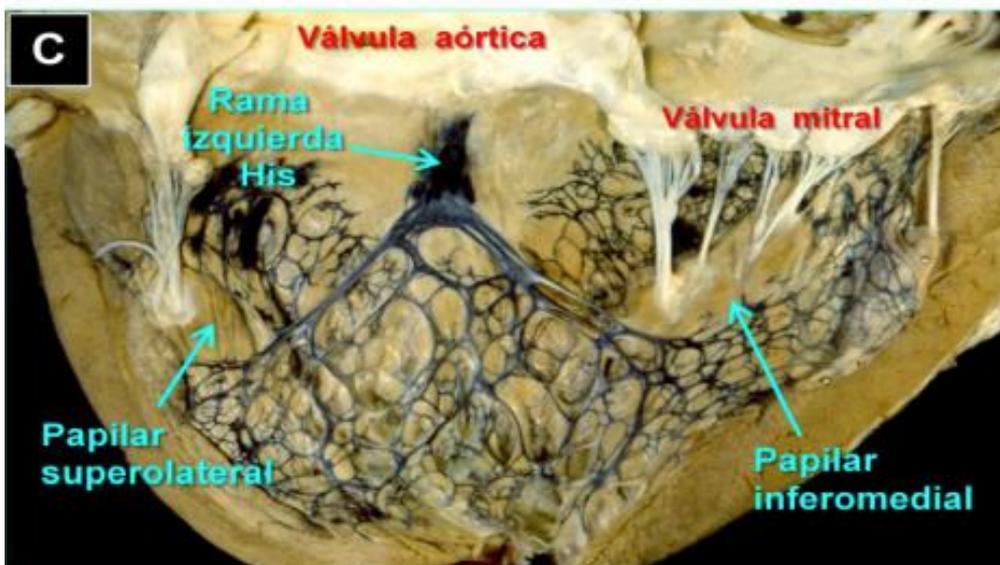
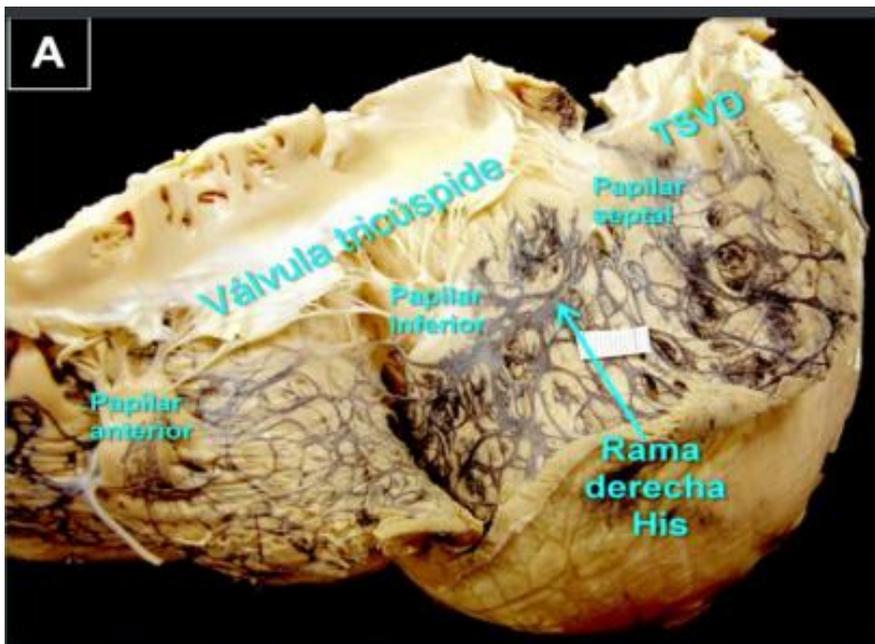


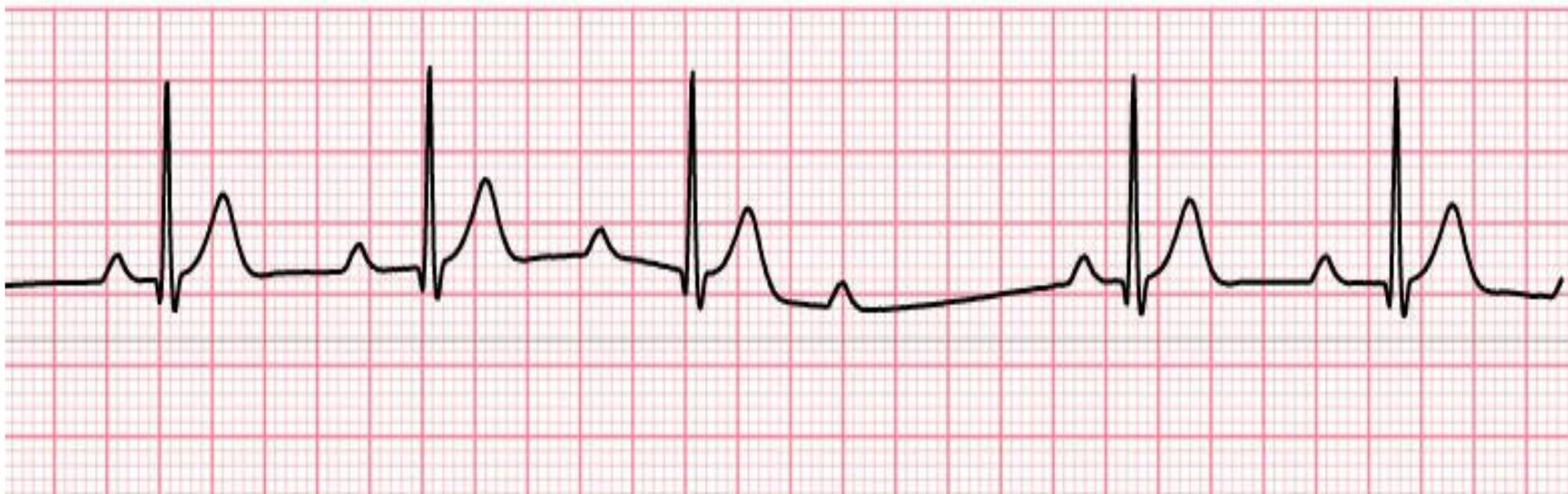


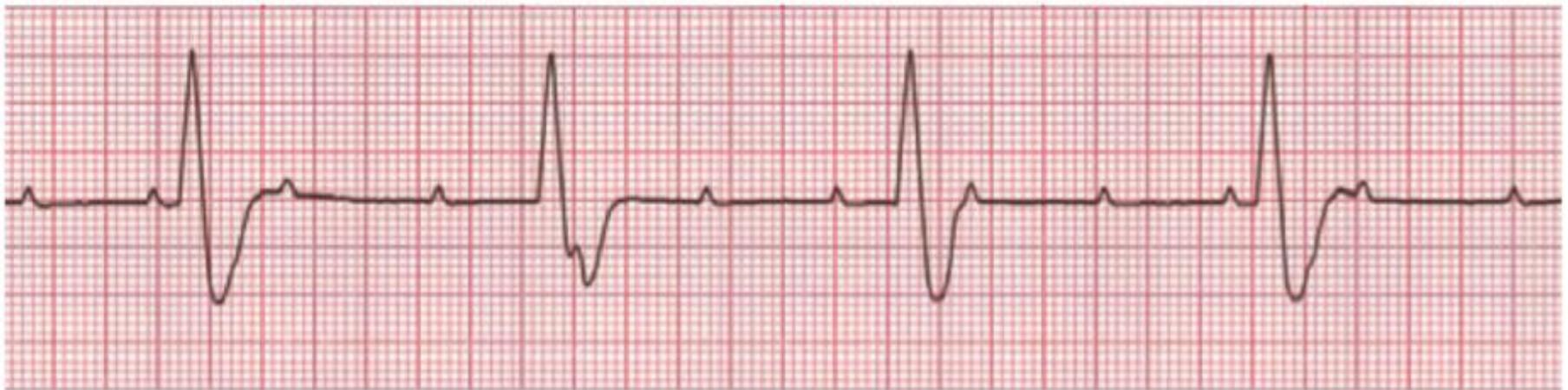
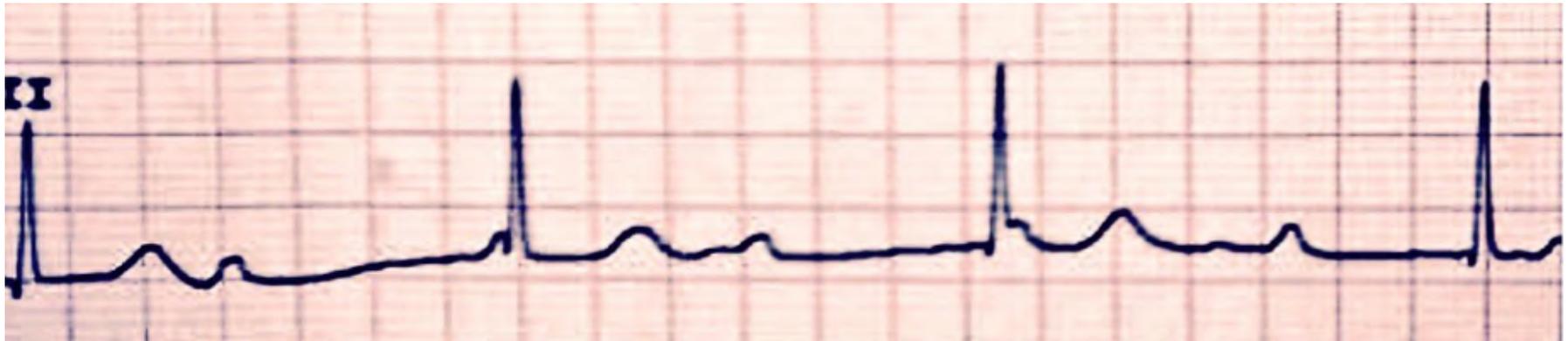


A.E. Teng et al. / Journal of Electrocardiology 49 (2016) 644–648









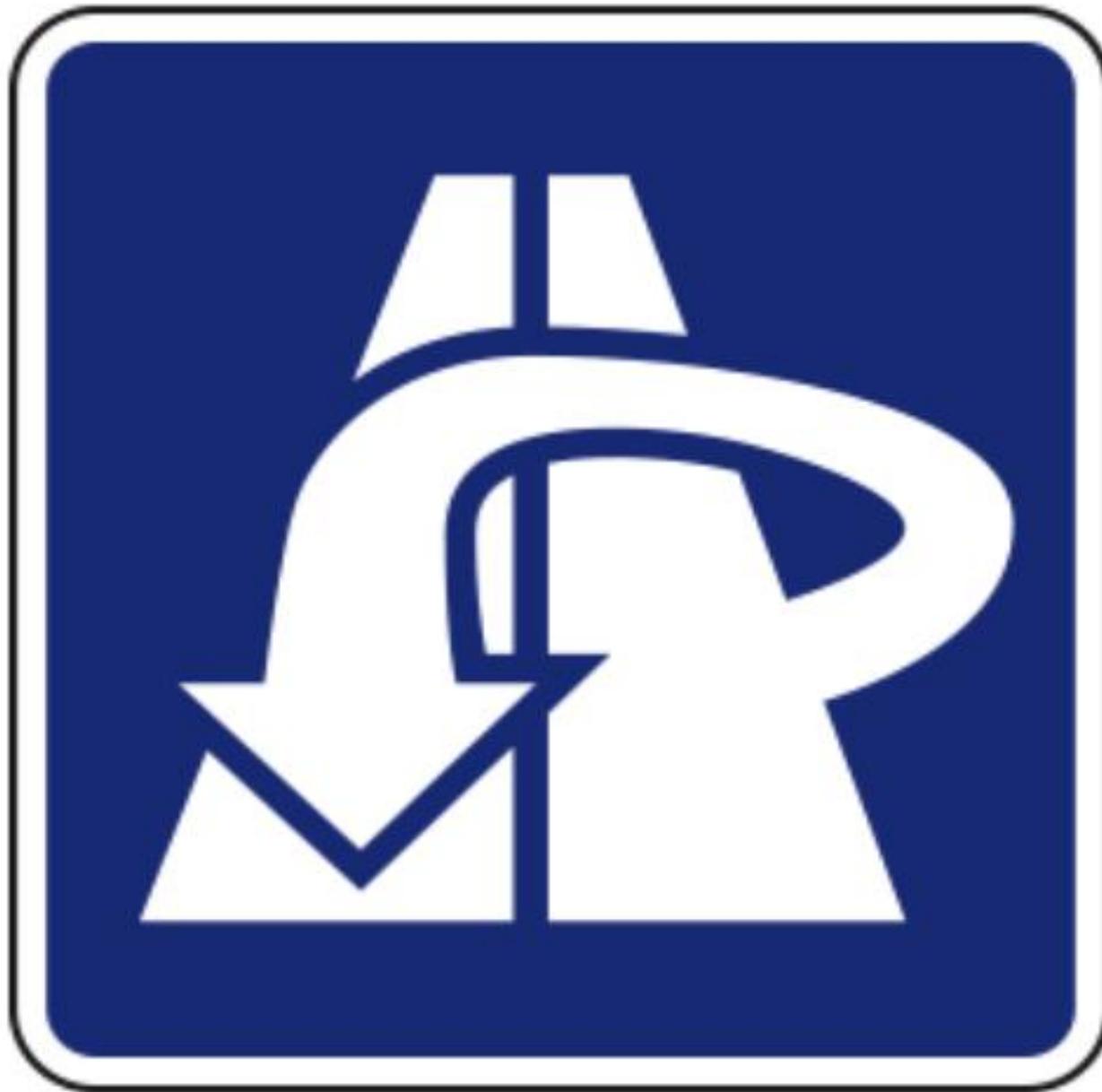




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Causas

Table 2: Various causes of atrioventricular block

Type of disorder	Causes
Autonomic	Carotid sinus hypersensitivity, vasovagal
Metabolic and endocrine	Hyperkalemia, hypermagnesemia, hypothyroidism, adrenal insufficiency
Drug induced	Beta blockers, CCBs, adenosine, lithium, digitalis, antiarrhythmics
Infections	Endocarditis, Lyme disease, Chagas disease, TB, toxoplasmosis, syphilis
Heritable and congenital	Congenital heart disease, maternal SLE, myotonic dystrophy, progressive familial heart block
Inflammatory and infiltrative	SLE, scleroderma, mixed connective tissue disorder, rheumatoid arthritis, amyloidosis, sarcoid, hemochromatosis
Neoplasms	Lymphoma, mesothelioma, radiation, catheter ablation
Degenerative	Lev disease, Lenegre's disease
Coronary artery disease	Acute myocardial infarction ⁽¹⁾

CCBs: Calcium channel blockers, TB: Tuberculosis, SLE: Systemic lupus erythematosus



Causas

Infarto Agudo al miocardio

Hiperkalemia

Hipotiroidismo

Endocarditis

Intoxicación y drogas

Type of disorder

- Autonomic
- Metabolic and endocrin
- Drug induced
- Infections
- Heritable and congenita
- Inflammatory and infiltra
- Neoplasms
- Degenerative
- Coronary artery diseas
- CCBs: Calcium channel blo

ck
coid, hemochromatosis



Circulation

ACC/AHA/HRS GUIDELINE

2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay

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



Tratamiento médico

Medication	Dosage	Comments
Symptomatic sinus bradycardia or atrioventricular block		
Atropine	0.5–1 mg IV (may be repeated every 3–5 min to a maximum dose of 3 mg) ^{55.3.2.4-20-55.3.2.4-24}	
Dopamine	5 to 20 mcg/kg/min IV, starting at 5 mcg/kg/min and increasing by 5 mcg/kg/min every 2 min ^{55.3.2.4-25}	Dosages of >20 mcg/kg/min may result in vasoconstriction or arrhythmias
Isoproterenol	20–60 mcg IV bolus followed doses of 10–20 mcg, or infusion of 1–20 mcg/min based on heart rate response ^{55.3.2.4-26-55.3.2.4-32}	Monitor for potential development of ischemic chest pain
Epinephrine	2–10 mcg/min IV or 0.1–0.5 mcg/kg/min IV titrated to desired effect ^{55.3.2.4-17,55.3.2.4-31,55.3.2.4-33}	
Second- or third-degree atrioventricular block associated with acute inferior MI		
Aminophylline	250-mg IV bolus	



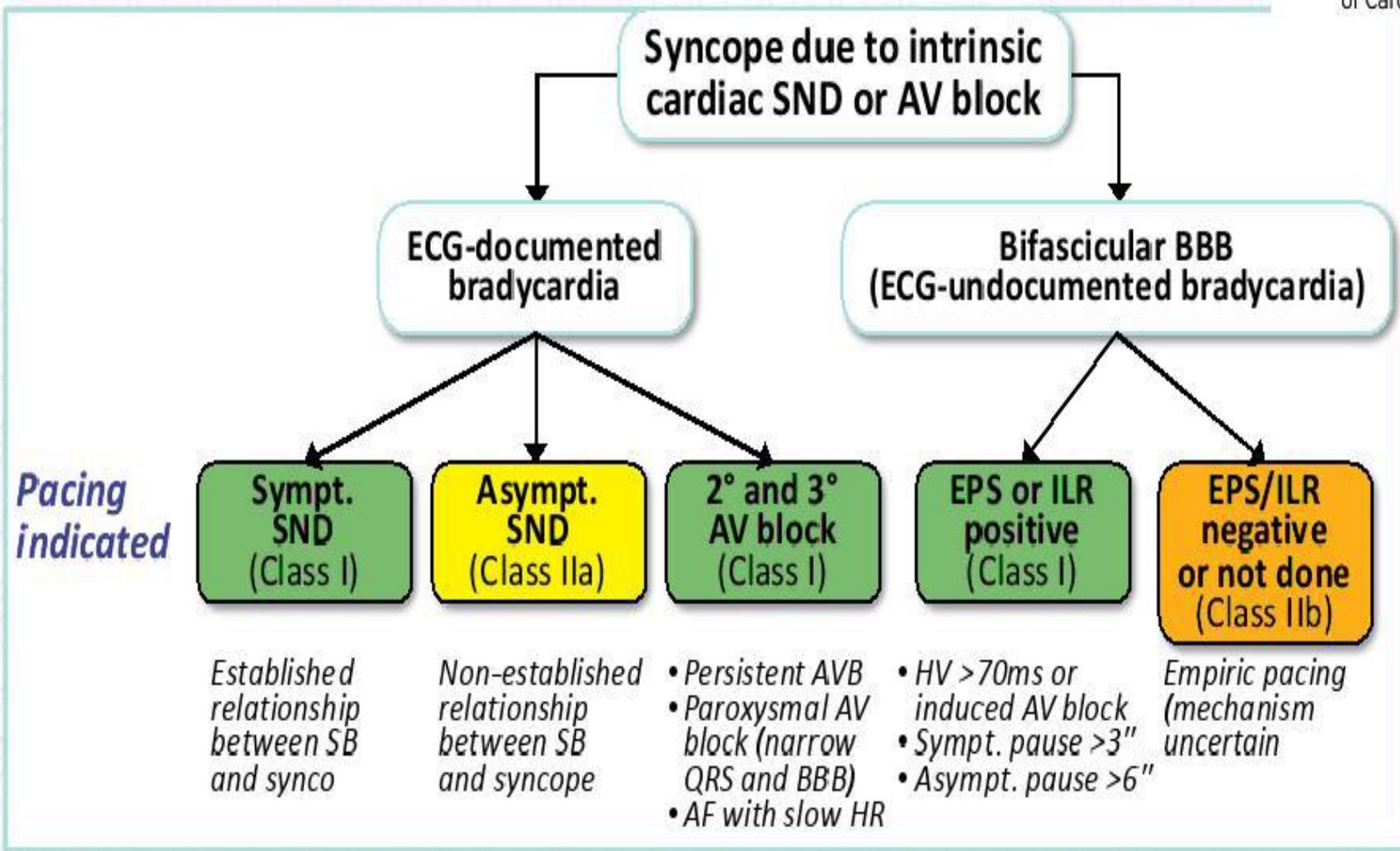
Tratamiento médico

Calcium channel blocker overdose		
10% calcium chloride	1–2 g IV every 10–20 min or an infusion of 0.2–0.4 mL/kg/h ^{55.3.2.4-34-55.3.2.4-36}	
10% calcium gluconate	3–6 g IV every 10–20 min or an infusion at 0.6–1.2 mL/kg/h ^{55.3.2.4-34-55.3.2.4-36}	
Beta-blocker or calcium channel blocker overdose		
Glucagon	3–10 mg IV with infusion of 3–5 mg/h ^{55.3.2.4-37,55.3.2.4-38}	
High dose insulin therapy	IV bolus of 1 unit/kg followed by an infusion of 0.5 units/kg/h. ^{55.3.2.4-36,55.3.2.4-39,55.3.2.4-40}	Follow glucose and potassium levels
Digoxin overdose		
Digoxin antibody fragment	Dosage is dependent on amount ingested or known digoxin concentration ^{55.3.2.4-41-55.3.2.4-48}	One vial binds approximately 0.5 mg of digoxin
		Administer over at least 30 min
		May be repeated



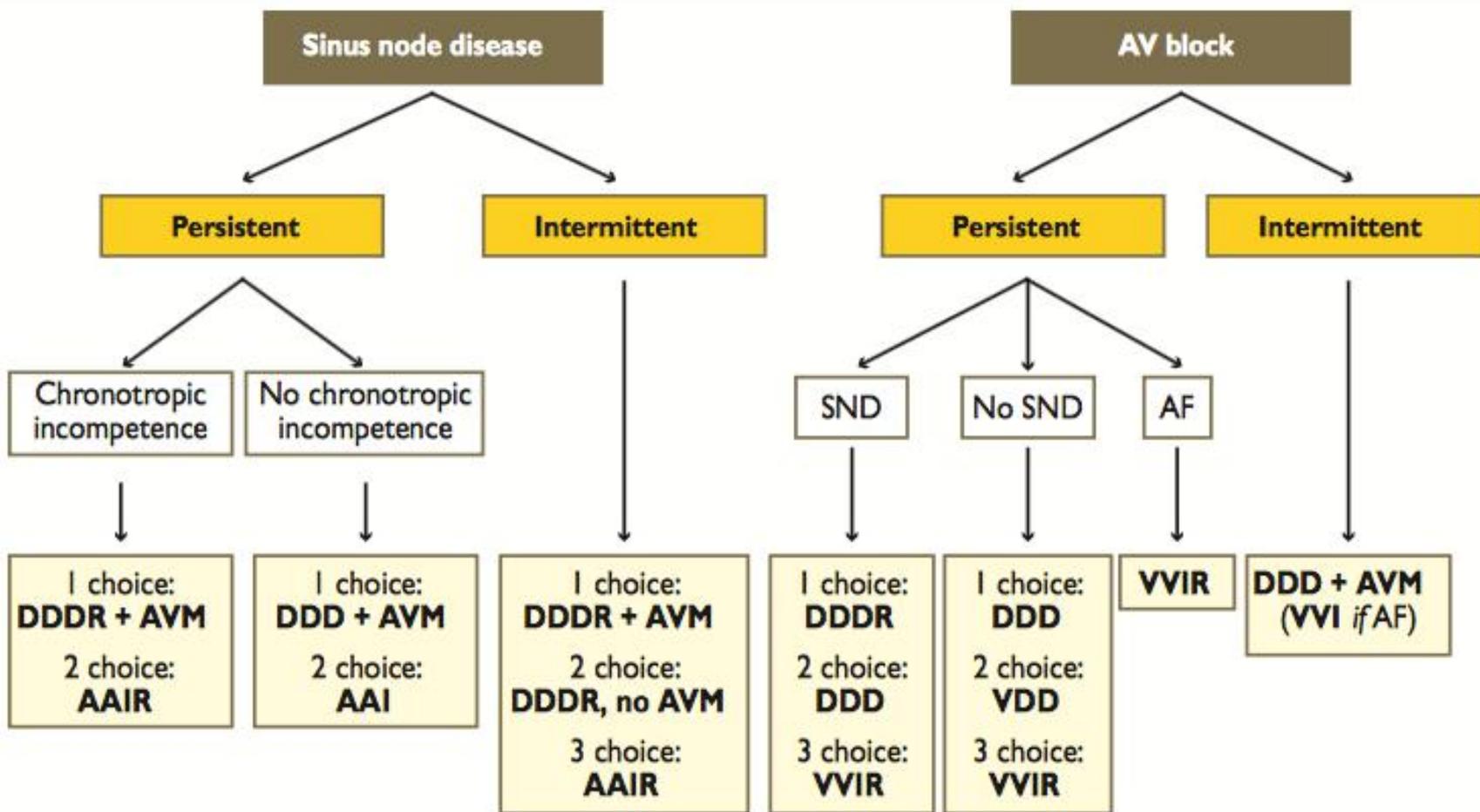
Terapia con dispositivos

Treatment of syncope: Cardiac arrhythmias





Indicaciones

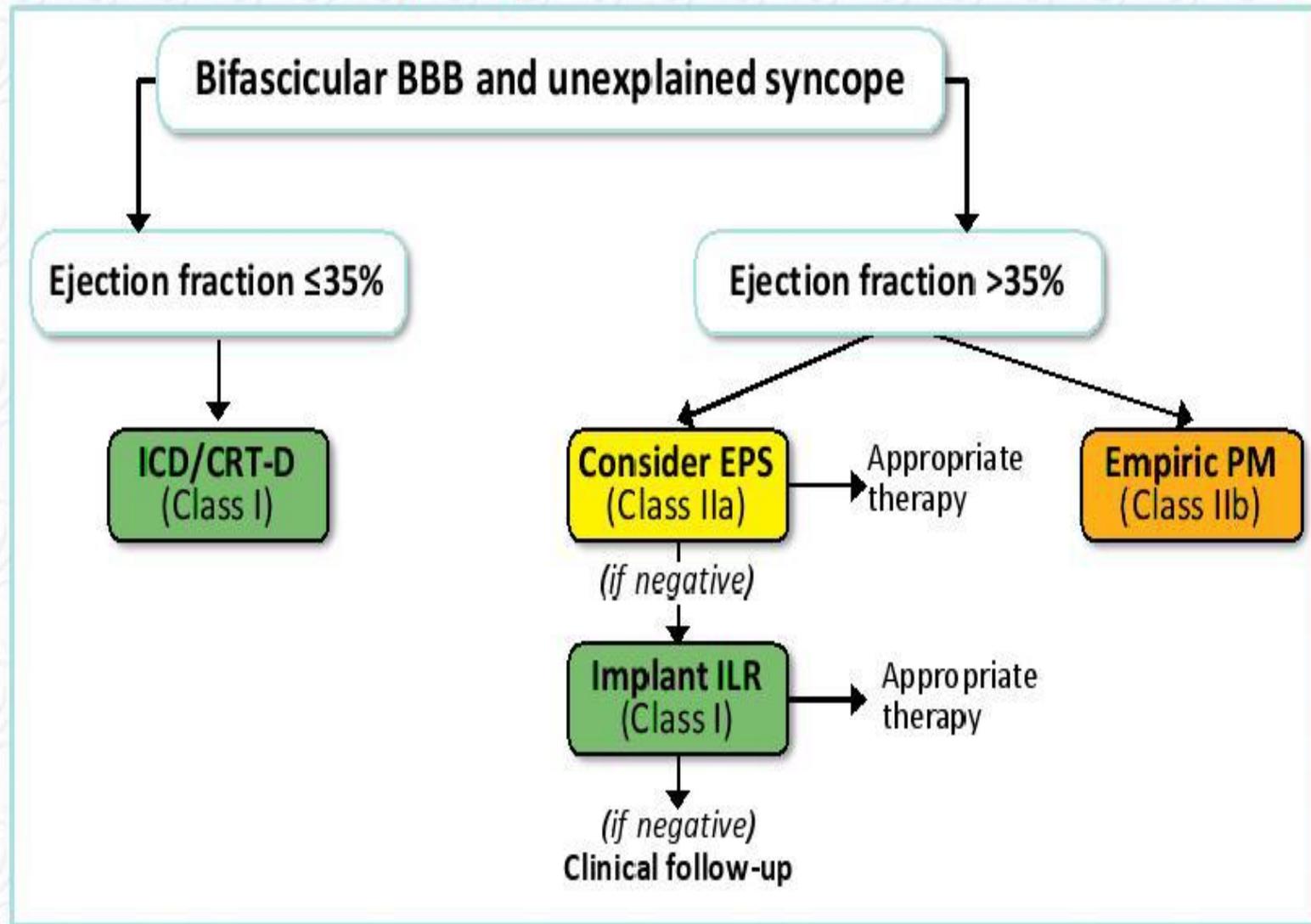


Consider CRT if low EF/HF

Dual-chamber versus ventricular pacing

Outcome	Dual-chamber benefit over ventricular pacing
All-cause deaths	No benefit
Stroke, embolism	Benefit (in meta-analysis only, not in single trial)
Atrial fibrillation	Benefit
HF, hospitalization for HF	No benefit
Exercise capacity	Benefit
Pacemaker syndrome	Benefit
Functional status	No benefit
Quality of life	Variable
Complications	More complications with dual-chamber

Treatment of syncope: Bundle Branch Block

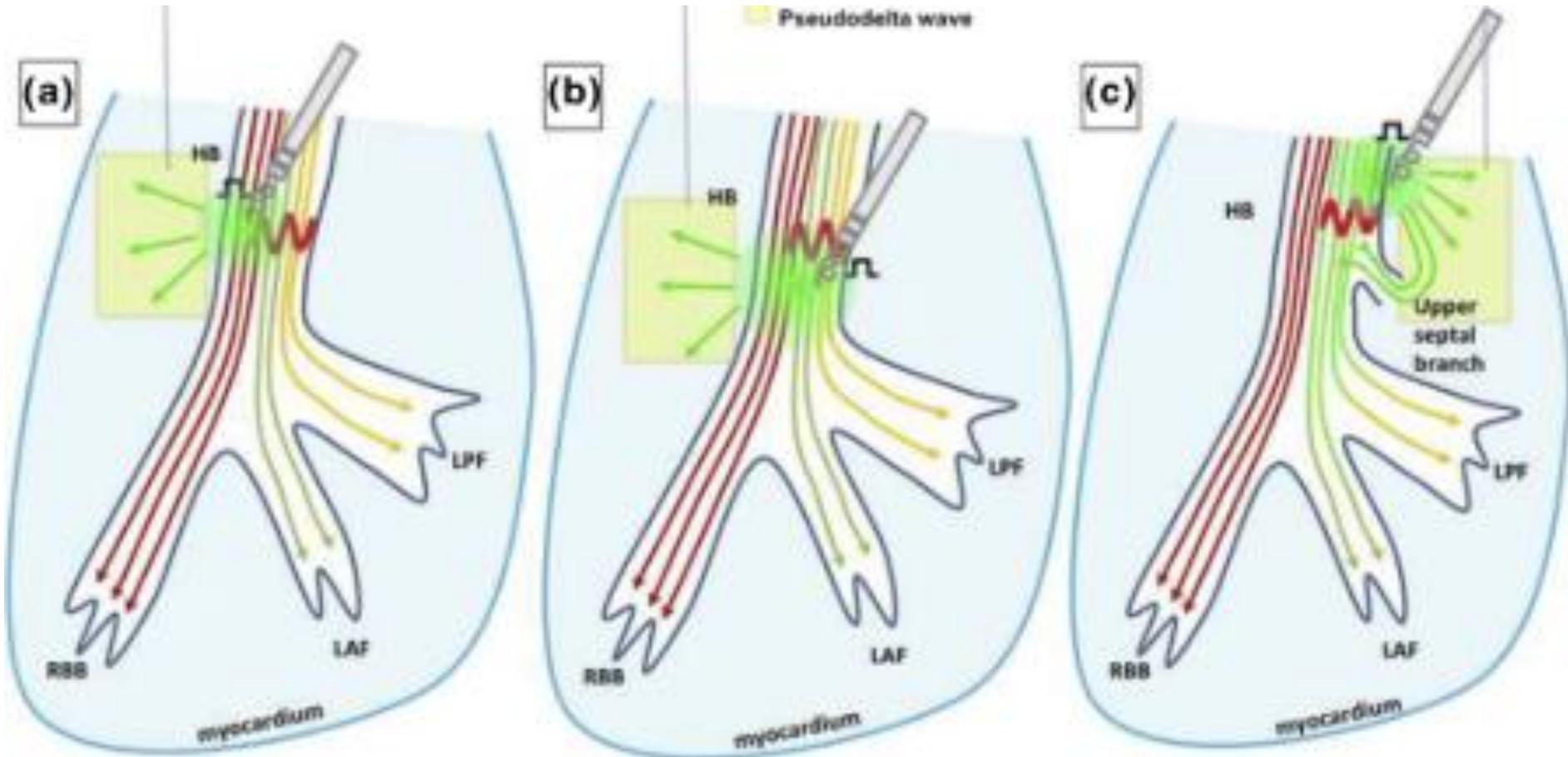




ACC/AHA/HRS GUIDELINE

2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay

- In patients with a left ventricular ejection fraction between 36% to 50% and atrioventricular block, who have an indication for permanent pacing and are expected to require ventricular pacing >40% of the time, techniques that provide more physiologic ventricular activation (eg, cardiac resynchronization therapy, His bundle pacing) are preferred to right ventricular pacing to prevent heart failure.





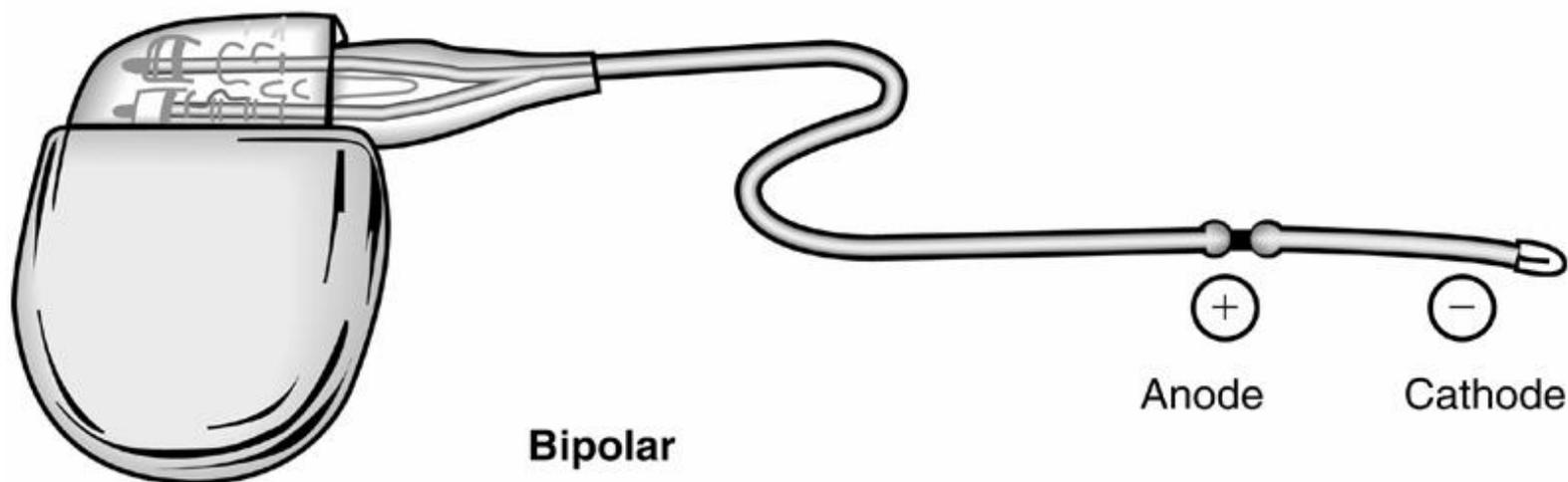
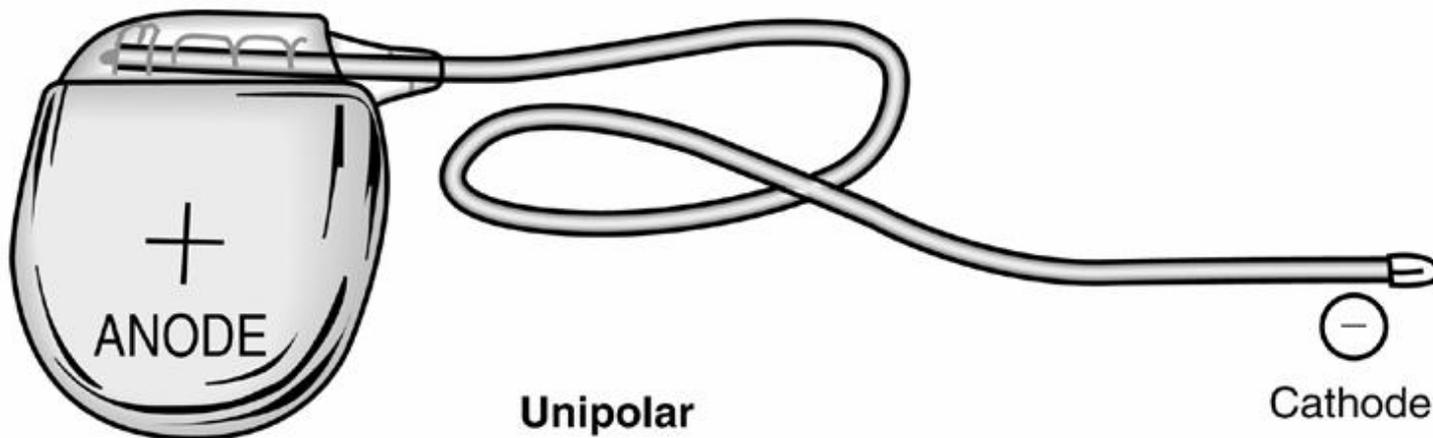
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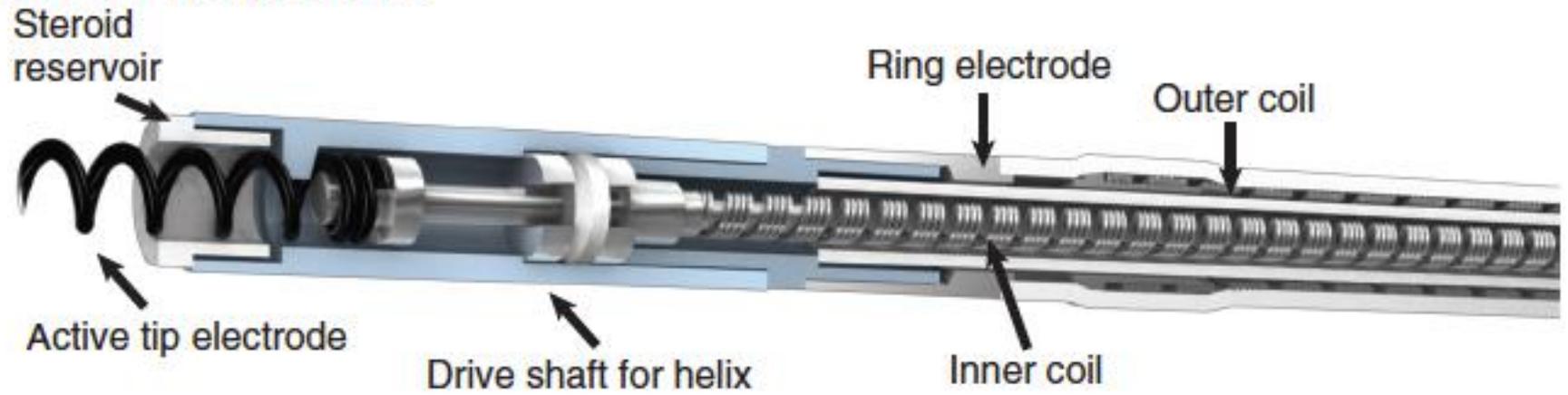
150 años al Servicio de la Medicina



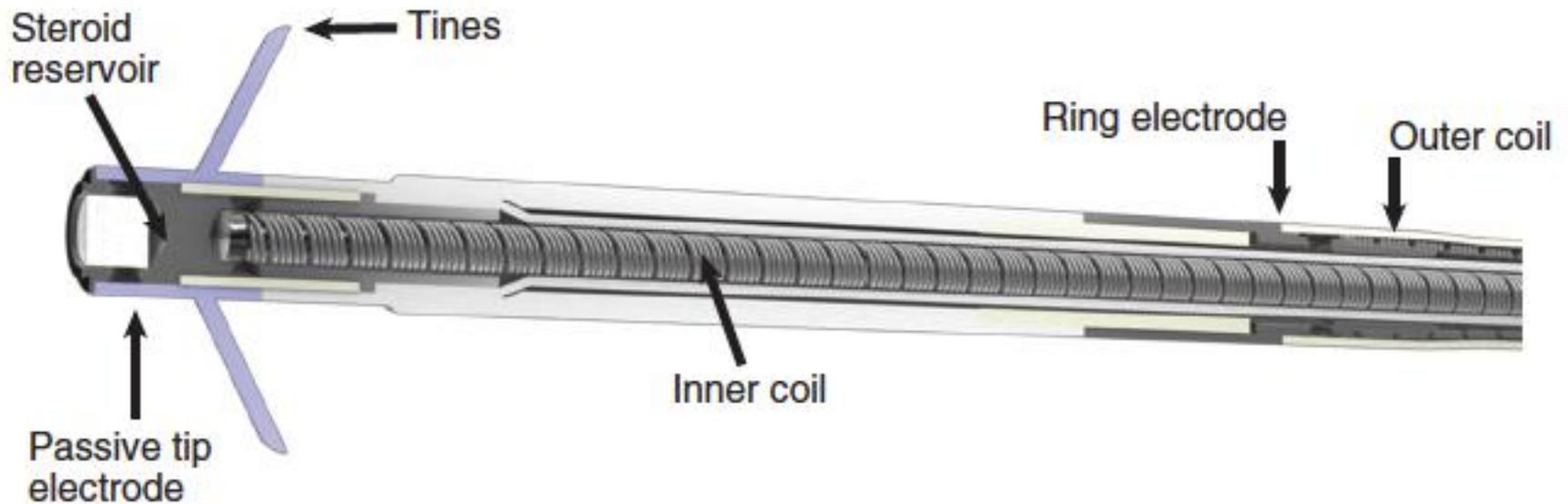
Marcapasos



ACTIVE PACING LEAD



PASSIVE PACING LEAD





Captura





	<i>Unipolar</i>	<i>Bipolar</i>
VENTAJAS	<ul style="list-style-type: none">— Umbrales de estimulación más bajos— Menor resistencia en el electrodo indiferente— Mejor análisis de la onda detectada— Cables más delgados— Mejor identificación ECG	<ul style="list-style-type: none">— Menos interferencias— Menos inhibición por miopotenciales— Mejor detección de la onda auricular— Posibilidad de conversión en unipolar
INCONVENIENTES	<ul style="list-style-type: none">— Mayor posibilidad de detección de interferencias— Mayor posibilidad de inhibición por miopotenciales— Posible estimulación muscular	<ul style="list-style-type: none">— Cables más gruesos y menos flexibles— Implantación más difícil— Fabricación más costosa— Identificación del ECG más difícil



Unipolar

Bipolar

— Umbrales de estimulación más bajos

— Menos interferencias

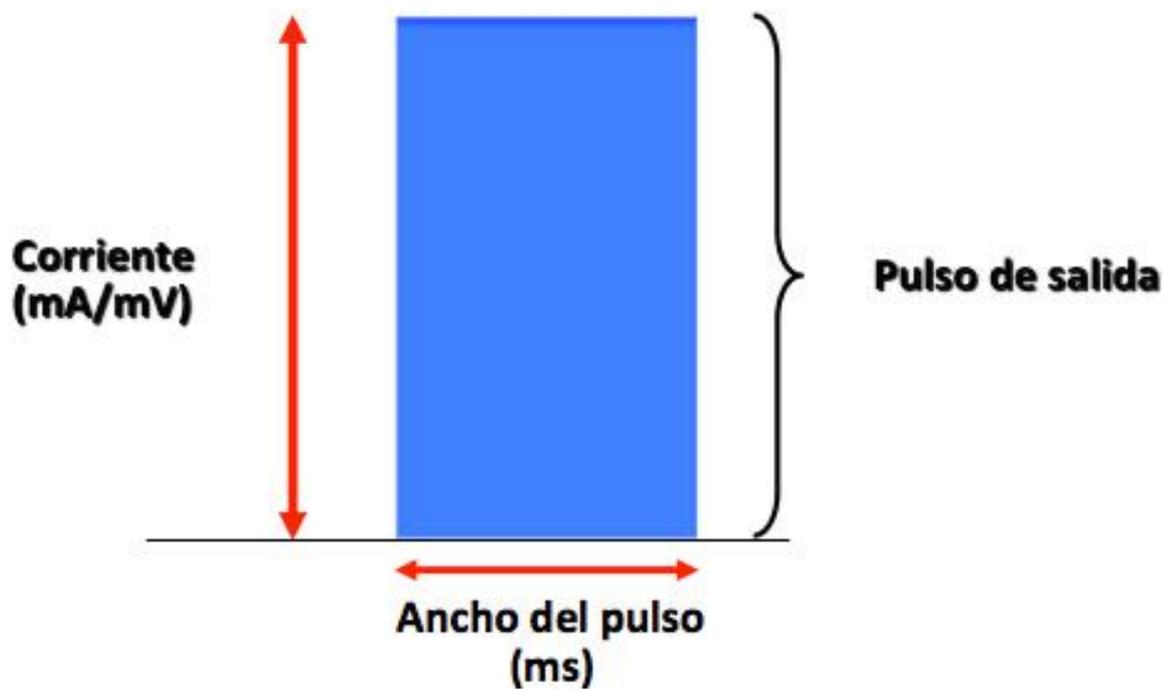
TABLA 1. Características de las dos configuraciones de estimulación

	Espícula	Umbral (V)	Impedancia	Energía
Unipolar	Mayor	Menor	Mayor	Igual
Bipolar	Menor	Mayor	Menor	Igual



Umbral

OUTPUT O SALIDA





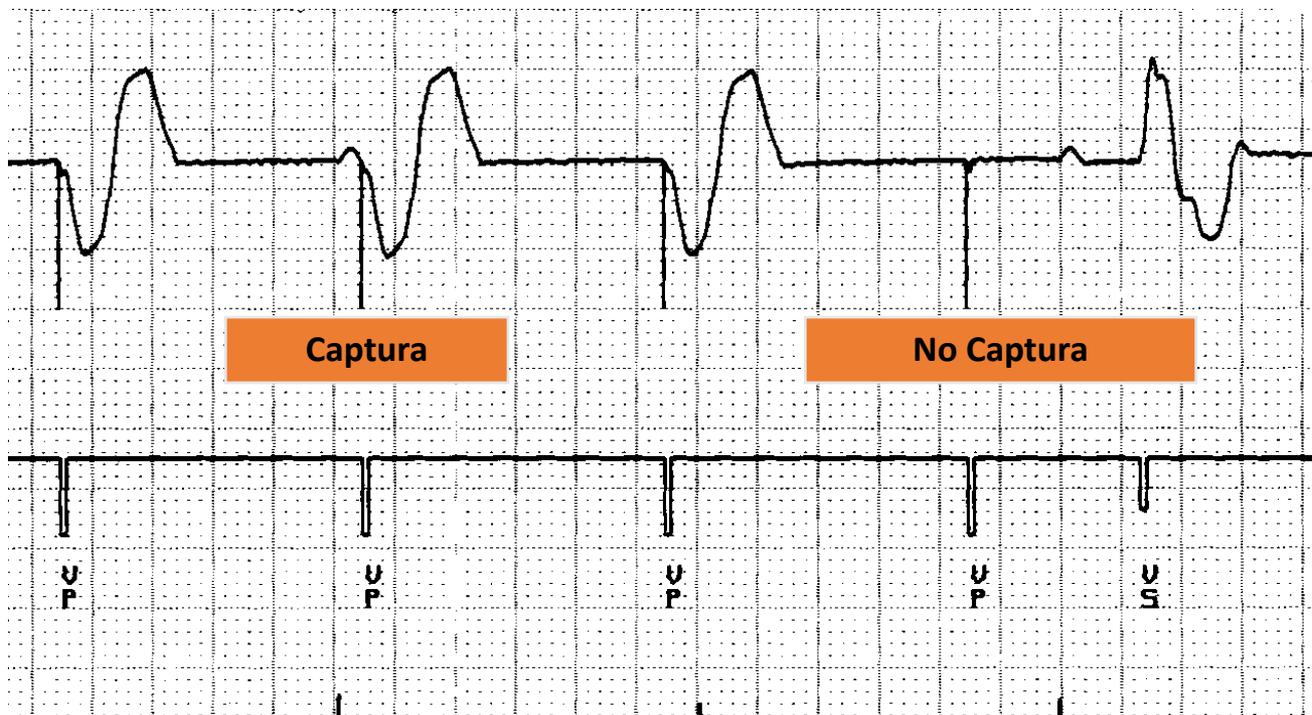
3 mV



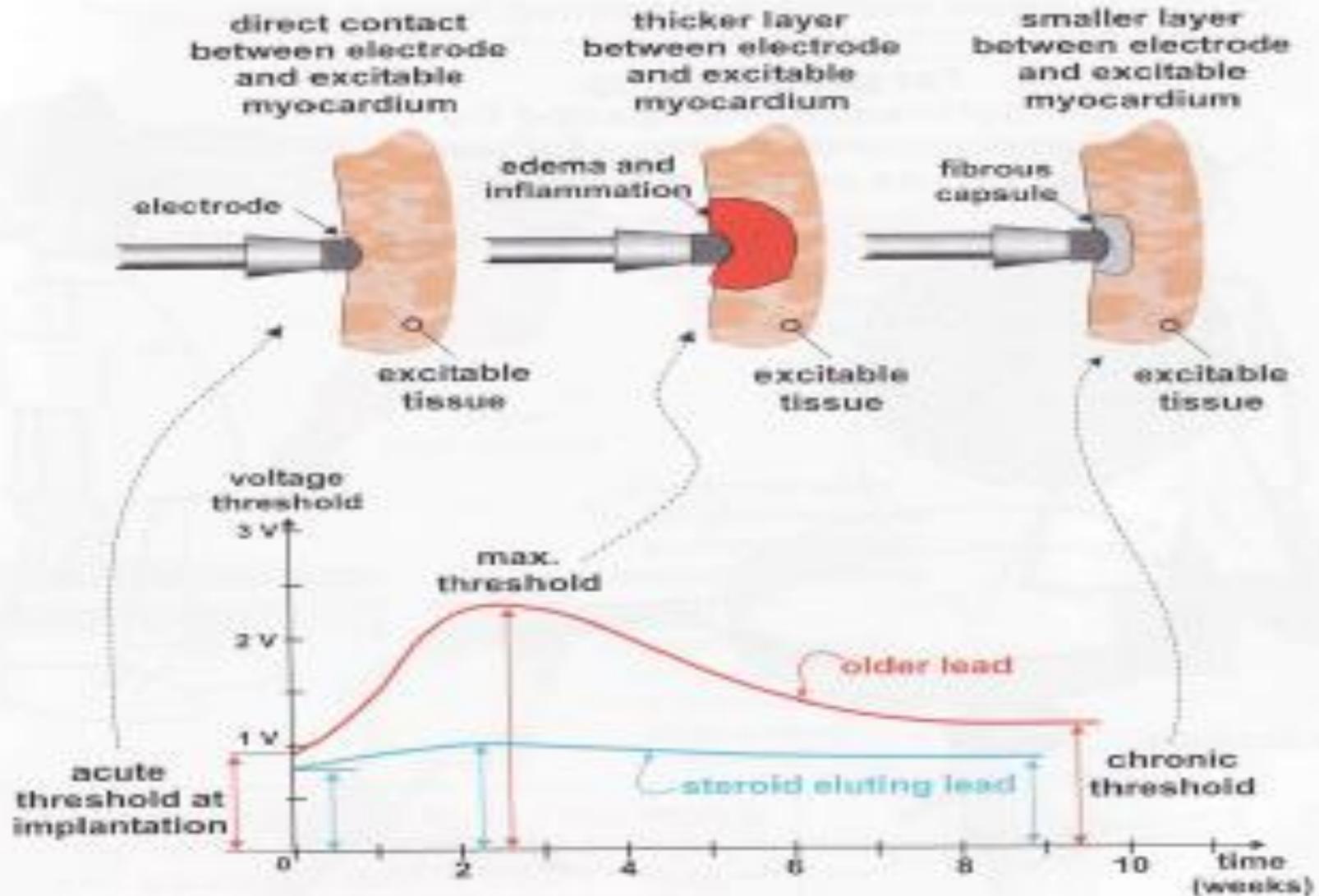
2 mV

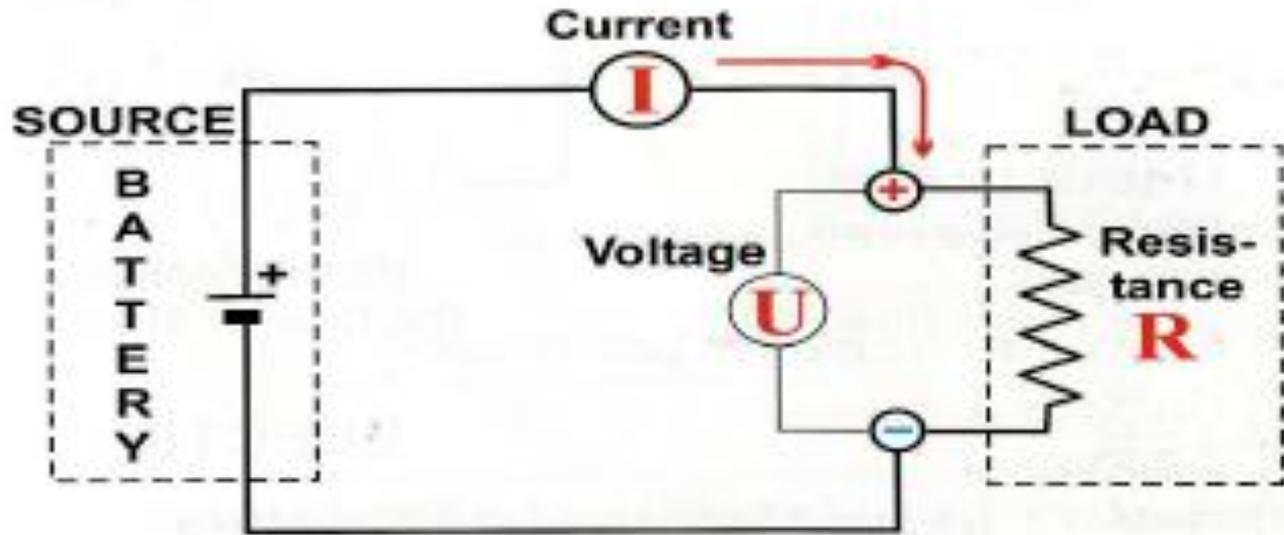


1 mV



EVOLUTION OF PACING THRESHOLD





U = voltage (in volt V)

I = current (in ampere A)

R = resistance (in ohm Ω)





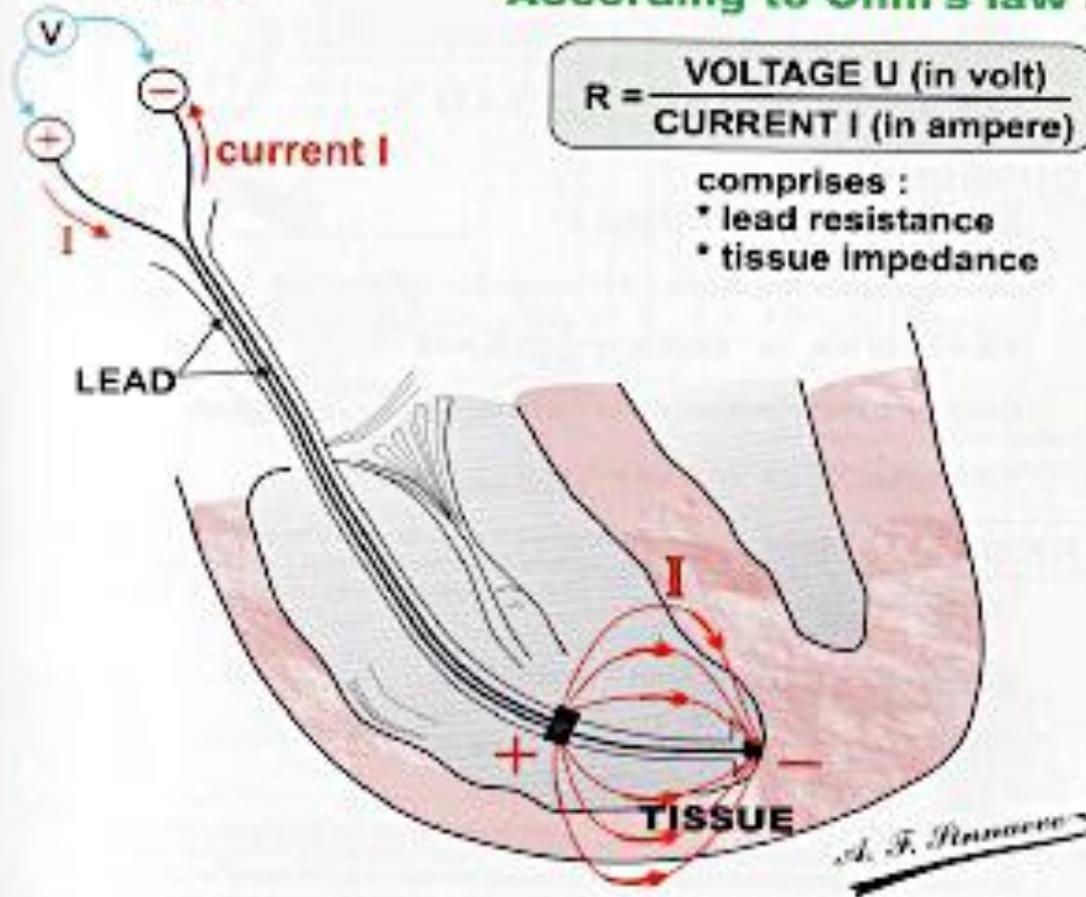
voltage U

According to Ohm's law :

$$R = \frac{\text{VOLTAGE } U \text{ (in volt)}}{\text{CURRENT } I \text{ (in ampere)}}$$

comprises :

- * lead resistance
- * tissue impedance

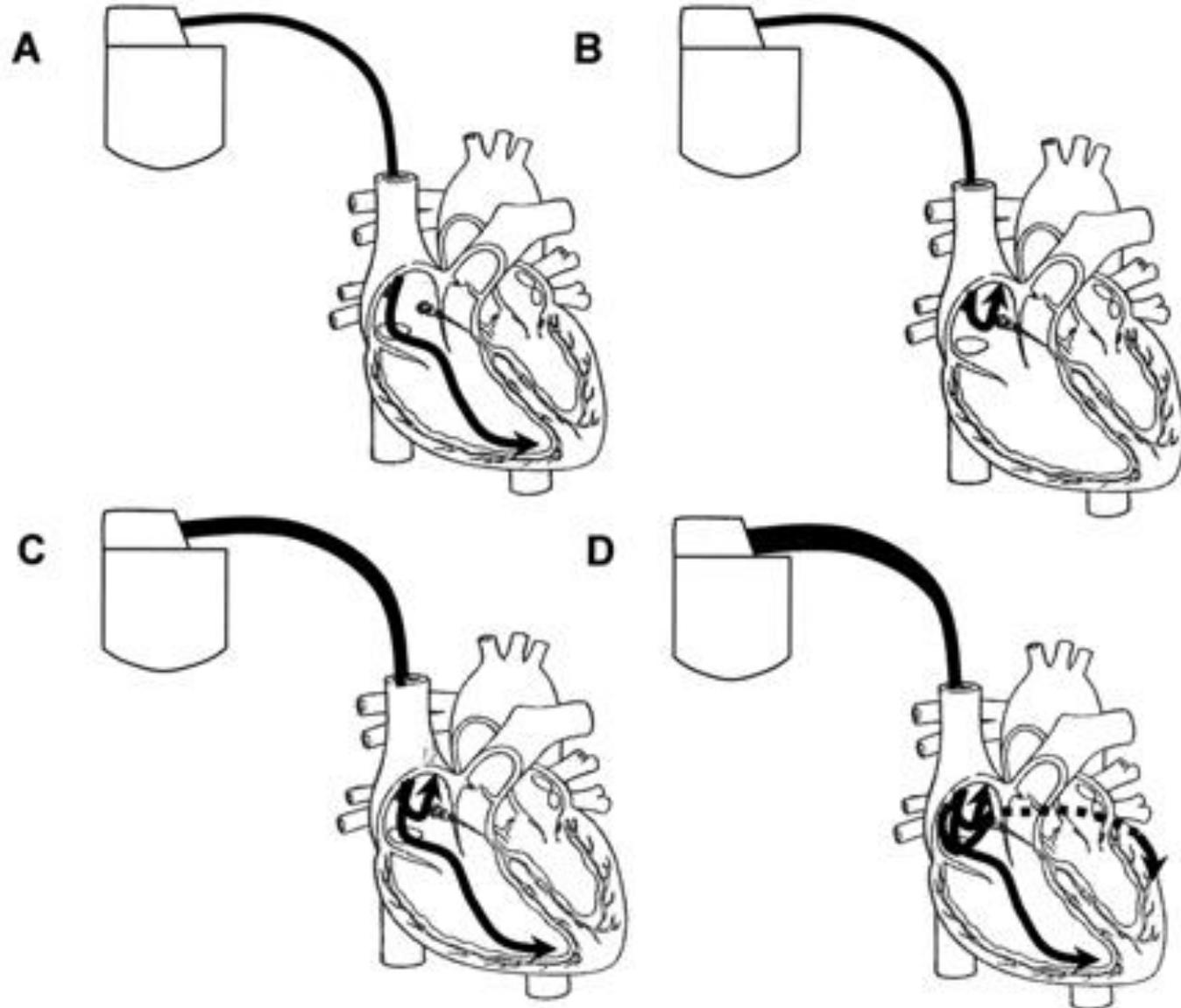


INSULATION
DEFECT
< 250Ω

NORMAL
PACING
IMPEDANCE
ca. 500Ω

LEAD
FRACTURE
> 1000Ω







Asincrónico

A00



V00

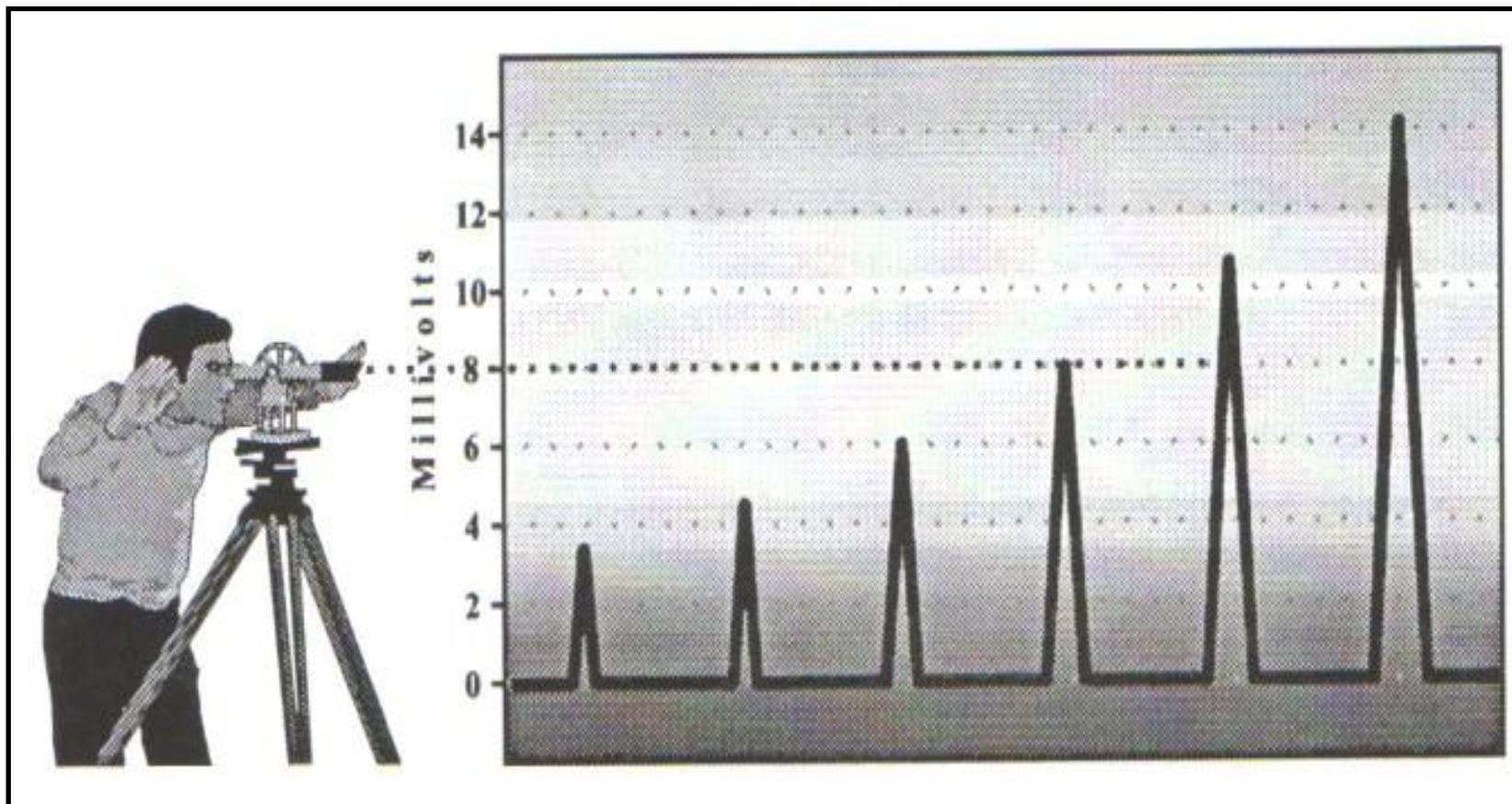


D00



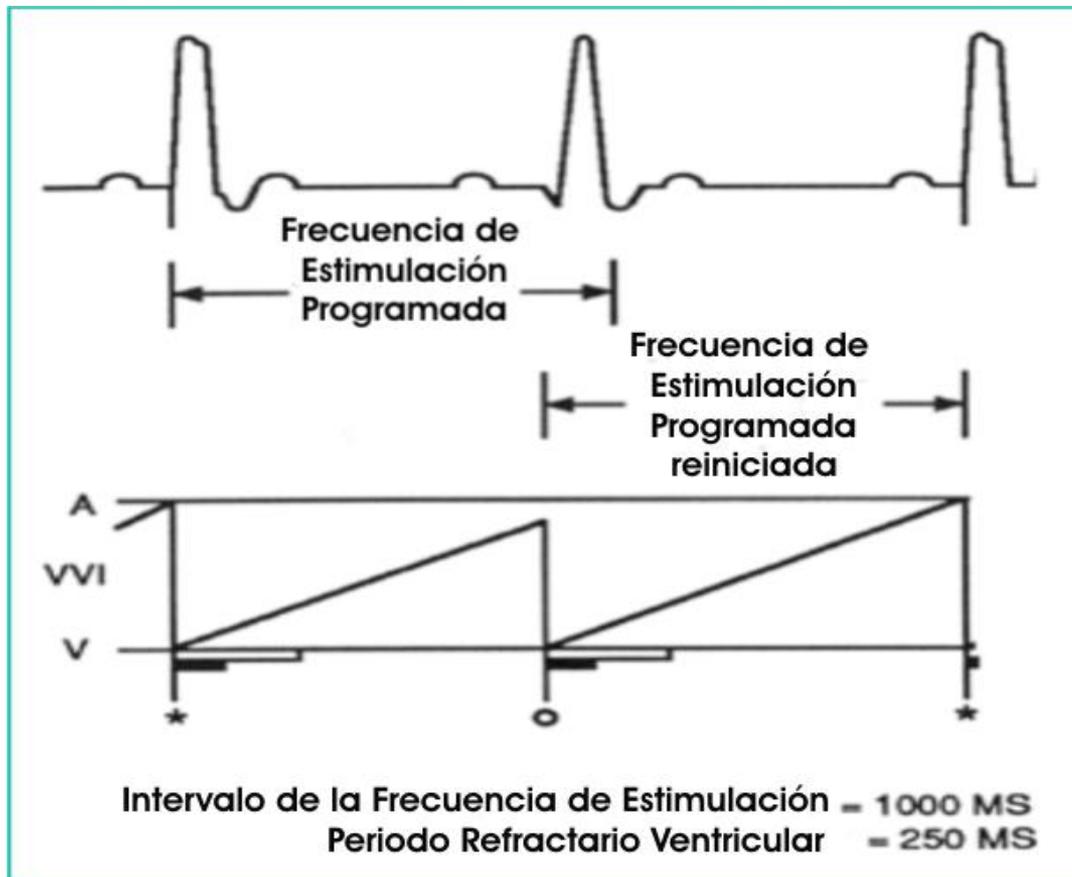


Sensibilidad





Demanda





Código genérico para estimulación antibradicardia

I	II	III	IV	V
Cámara estimulada	Cámara sensada	Respuesta al sentido	Modulación de frecuencia	Estimulación multisitio
O = Ninguna	O = Ninguna	O = No	O = No	O = No
A = Aurícula	A = Aurícula	T = Estimula	R = Modulación de frecuencia	A = Aurícula
V = Ventrículo	V = Ventrículo	I = Inhibe		V = Ventrículo
D = Ambas (A+V)	D = Ambas (A+V)	D = Ambas (T+I)		D = En ambas (A+V)

Modificado de Bernstein AD. et al. The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. Pacing Clin Electrophysiol 2002 ¹.



VOO en MCP
Apaga terapia en DAI



Problemas asociados a MCP



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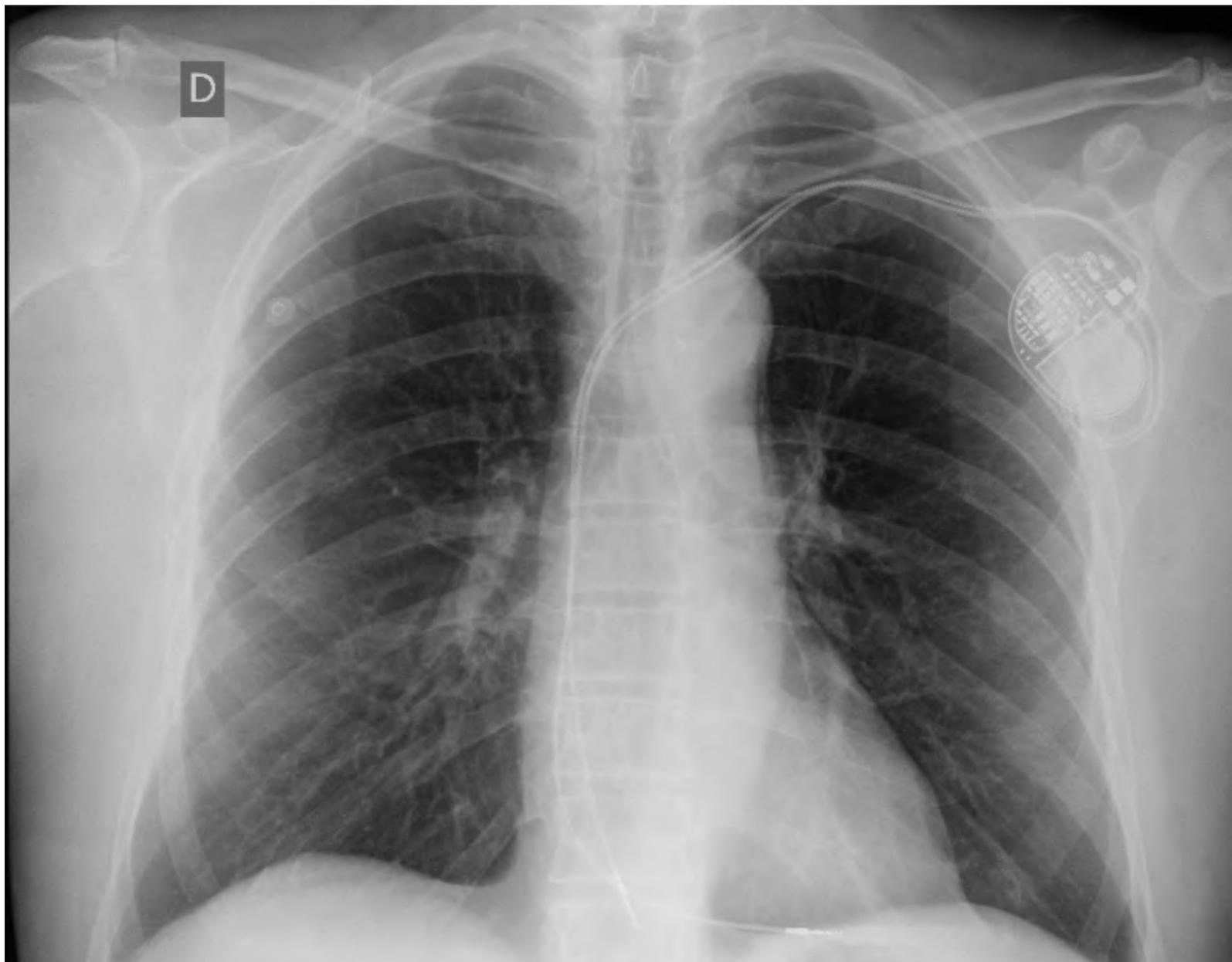


Funcionamiento



Falla de captura





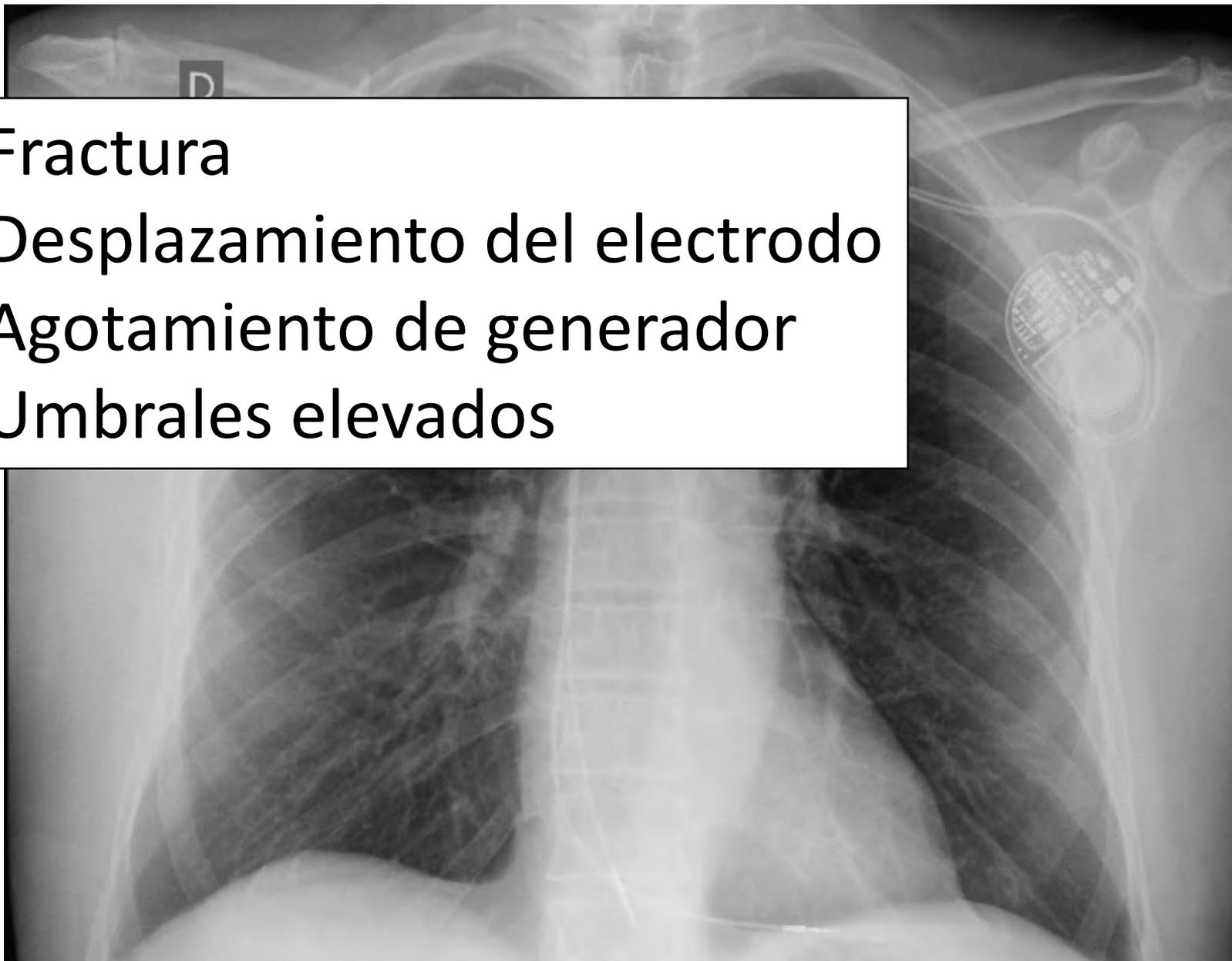


Fractura

Desplazamiento del electrodo

Agotamiento de generador

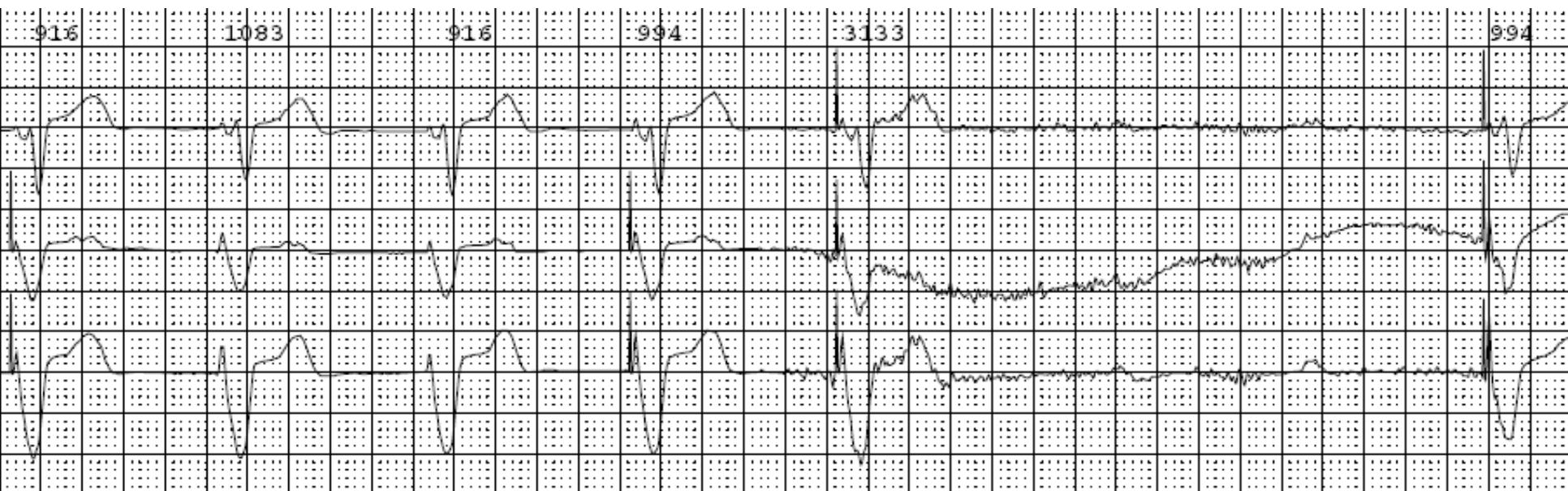
Umbrales elevados





Falla de salida





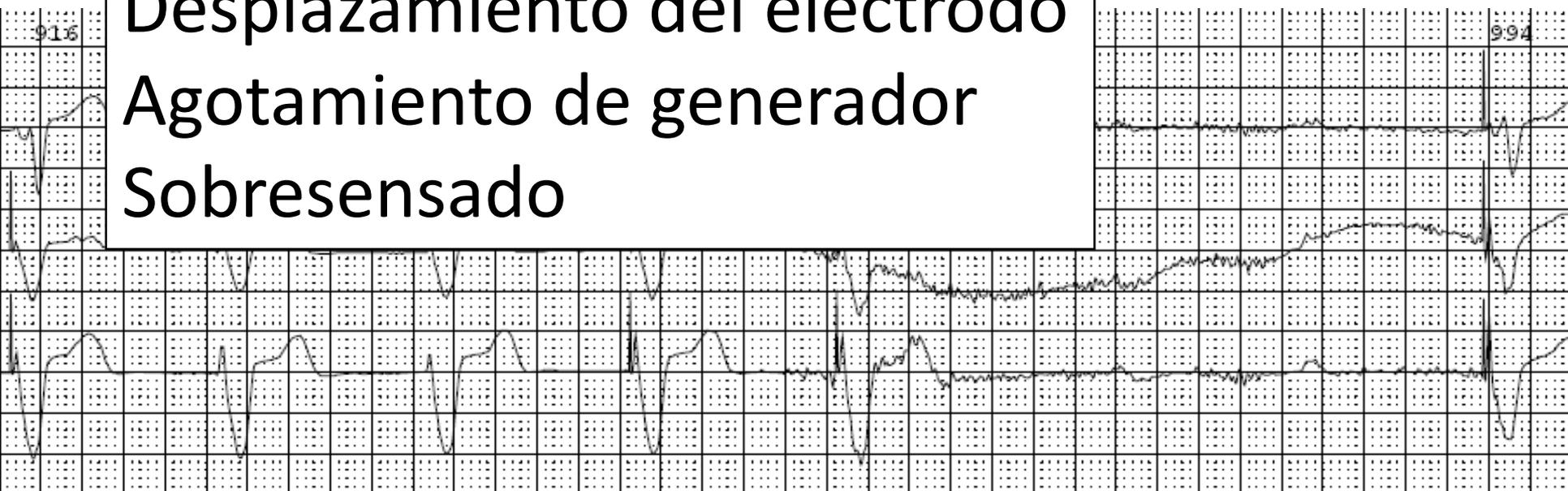


Fractura

Desplazamiento del electrodo

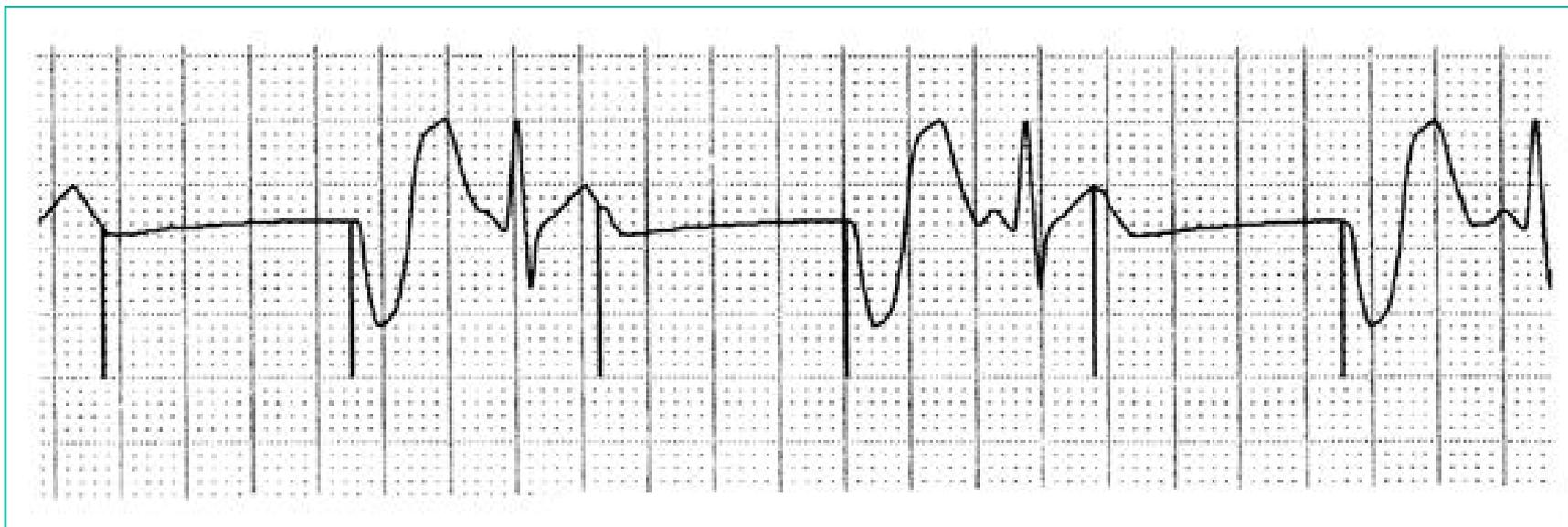
Agotamiento de generador

Sobresensado





Subsensibilidad





Subsensibilidad

Fractura

Desplazamiento del electrodo

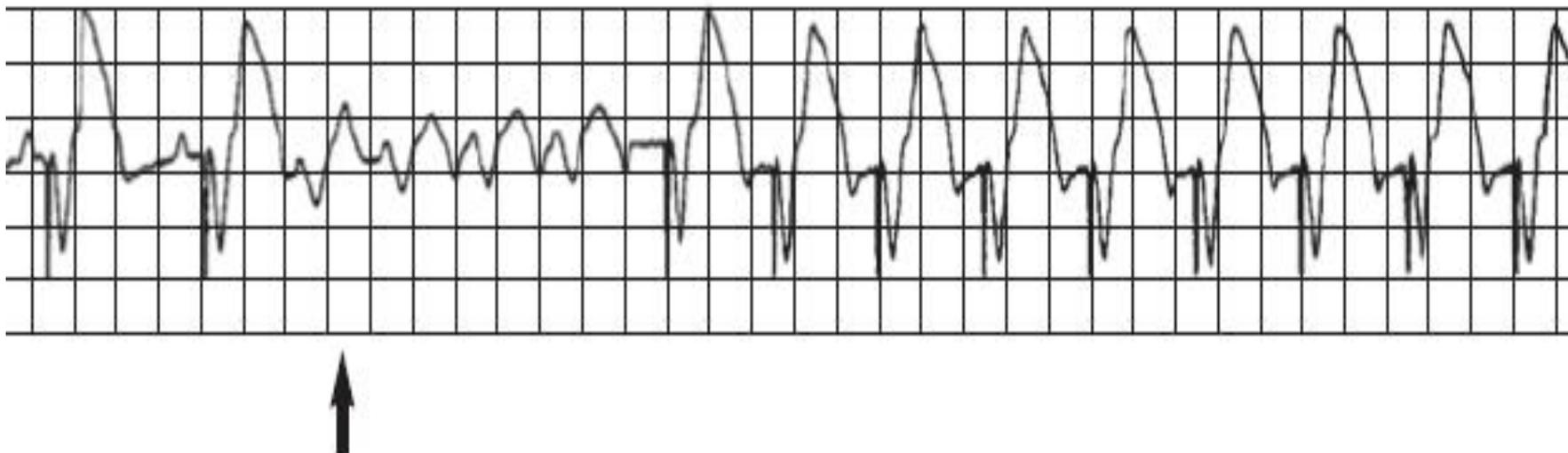
Agotamiento de generador

Programación inadecuada





TMP



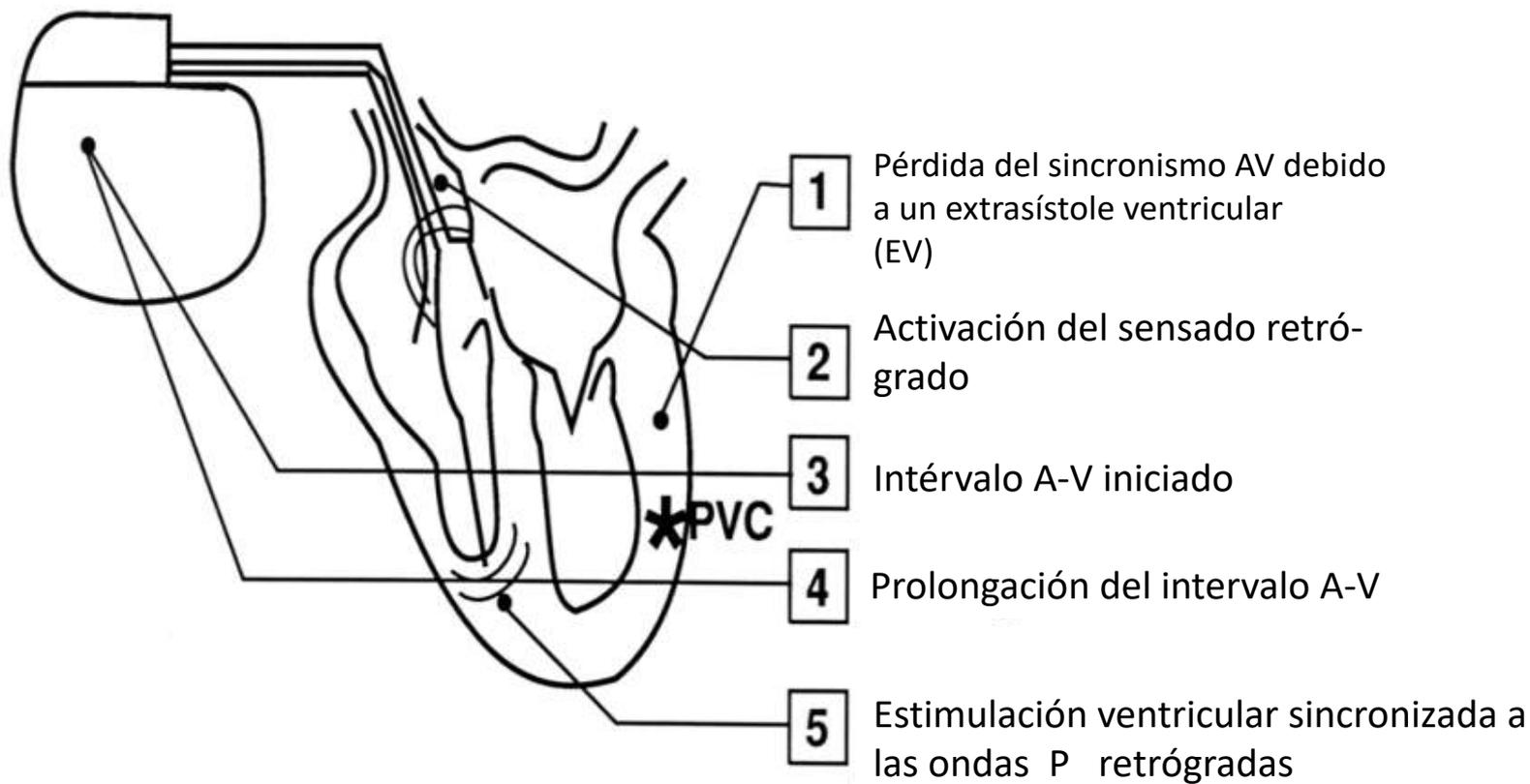


TPM

- Ocorre en MP bicamerales :
 - Tracking de una taquiarritmia auricular, FA o flutter atrial.
 - Conducción AV retrógrada (endless loop tachycardia).

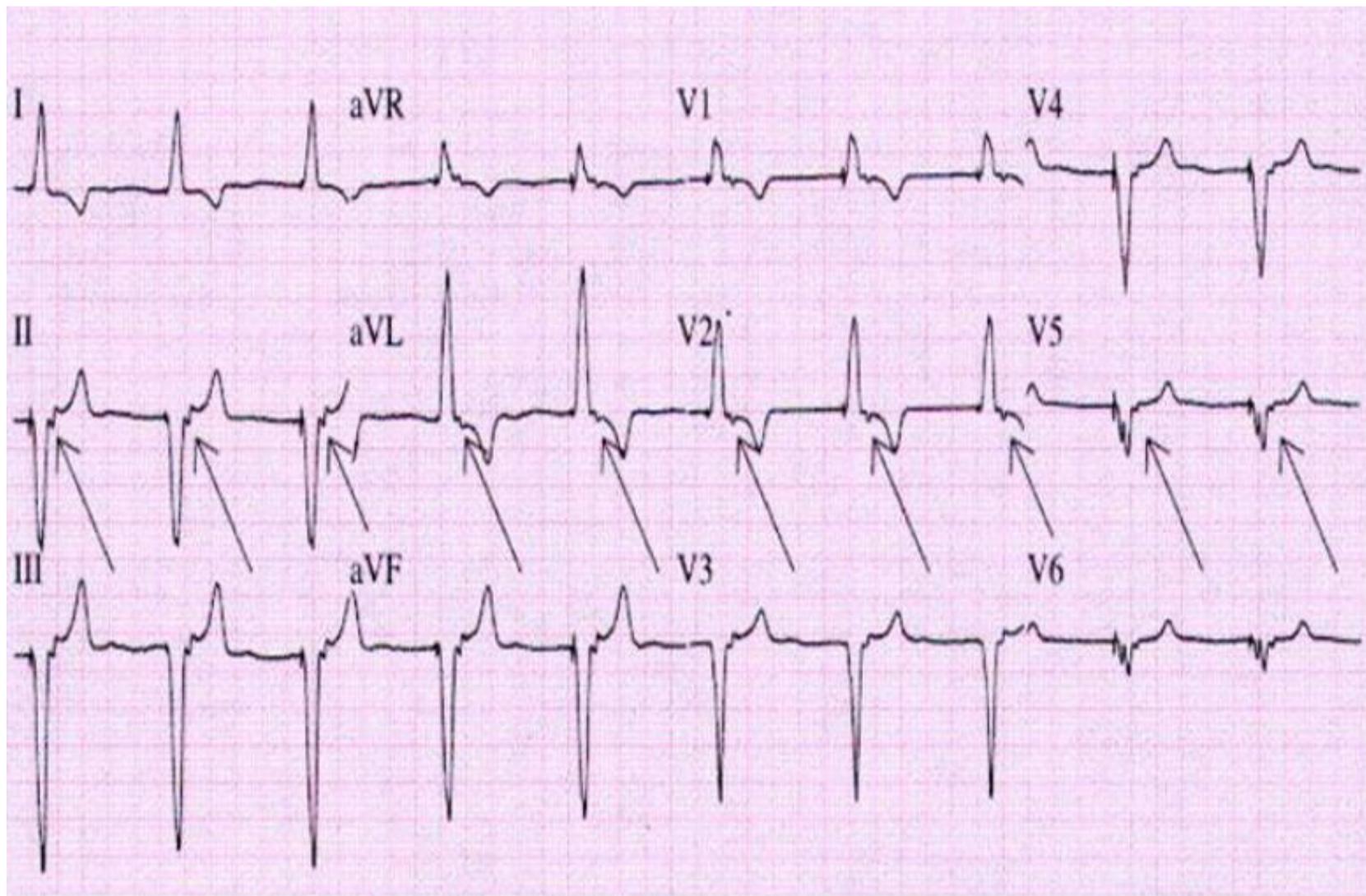


Conducción Retrógrada



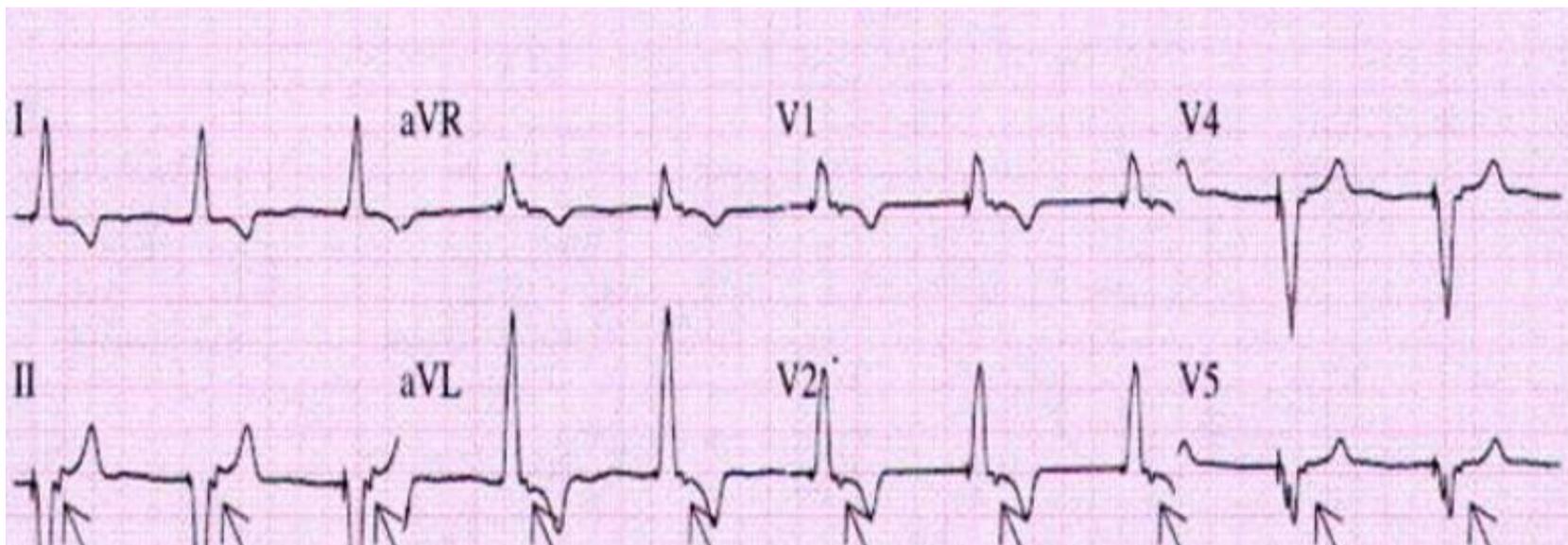


Sd. MCP

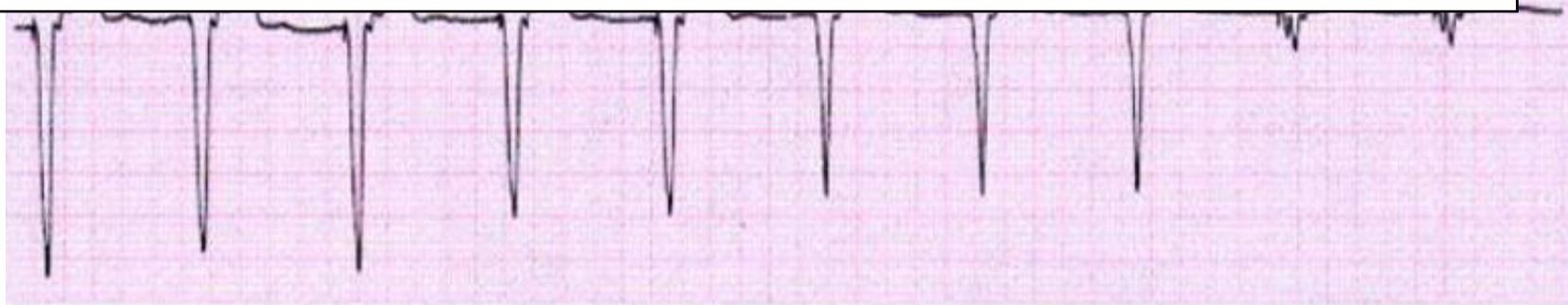




Sd. MCP

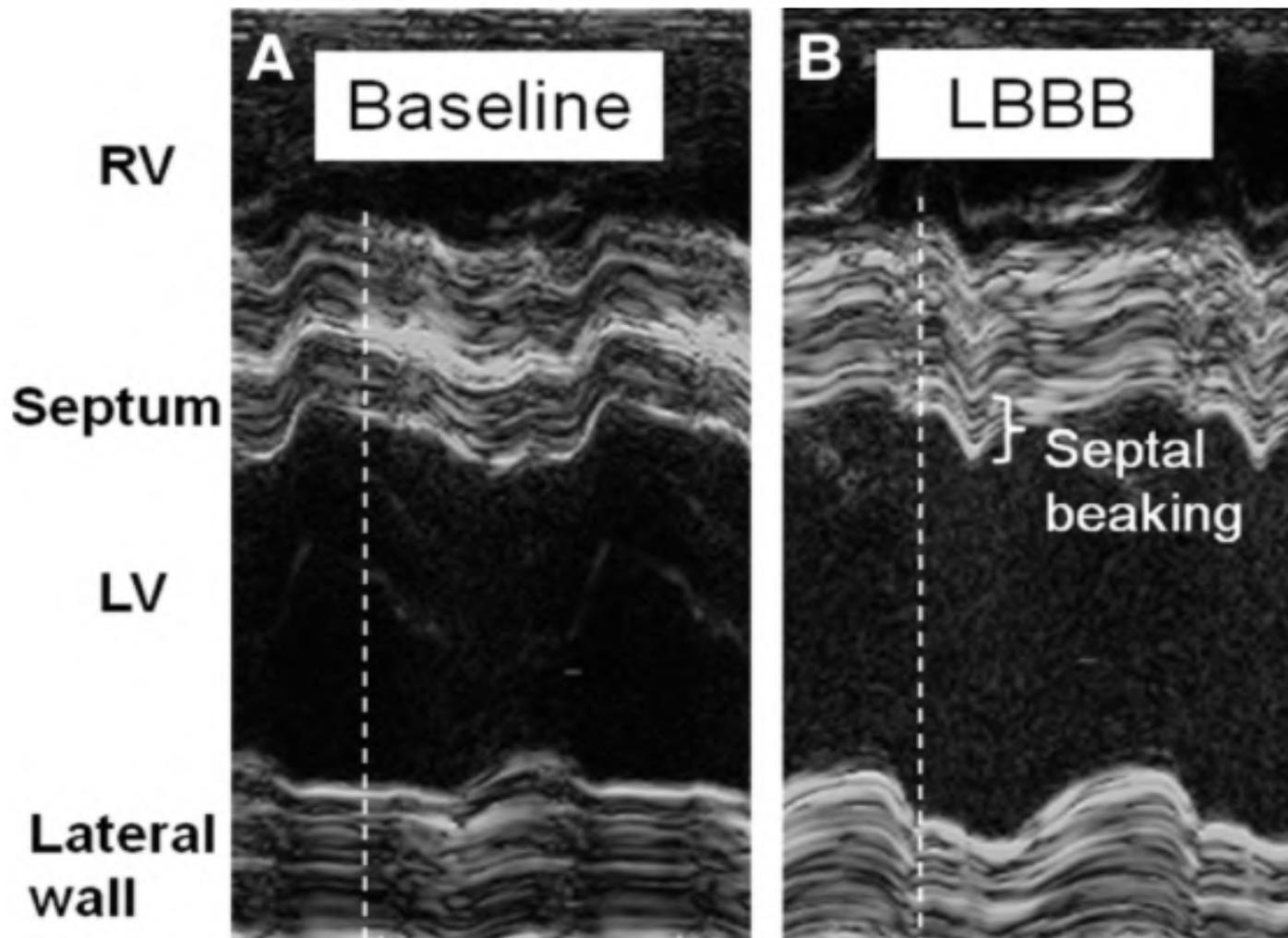


Fatiga, disnea, mareos, presíncope, síncope y pulsaciones desagradables en el cuello





Miocardiopatía por MCP





Miocardopatía por MCP

- RCTs con estimulación crónica aVD (independientes de la sincronía AV):
 - Aumento de incidencia FA
 - Aumento de incidencia de ICC/Hospitalización por ICC
 - Aumento incidencia de **Disfunción Sistólica VI**

Nielsen JC, Circulation 1998; 97:987-995

Nielsen JC, JACC 2003;42:614-623

Sweeney MO, Circulation 2003; 23:2932-2937

- Estimulación aVD durante terapia con ICD:
 - Tendencia a mayor riesgo de arritmias ventriculares y M°CV

Steinberg JS, J Cardiovasc Electrophysiol.2005;16:359-365

DAVID Trial. JAMA 2002;288:3115-3123



Sociedad Médica de Santiago

Sociedad Chilena de Medicina Interna

150 años al Servicio de la Medicina



FIN

Muchas gracias!