

108 Reunión de la Sociedad Castellana de Cardiología

FLASHLIGHTS en



INSUFICIENCIA CARDIACA

PROGRAMA DE ACTUALIZACIÓN CARDIOVASCULAR:
MENSAJES CONCRETOS SOBRE NOVEDADES EN ÁREAS TEMÁTICAS



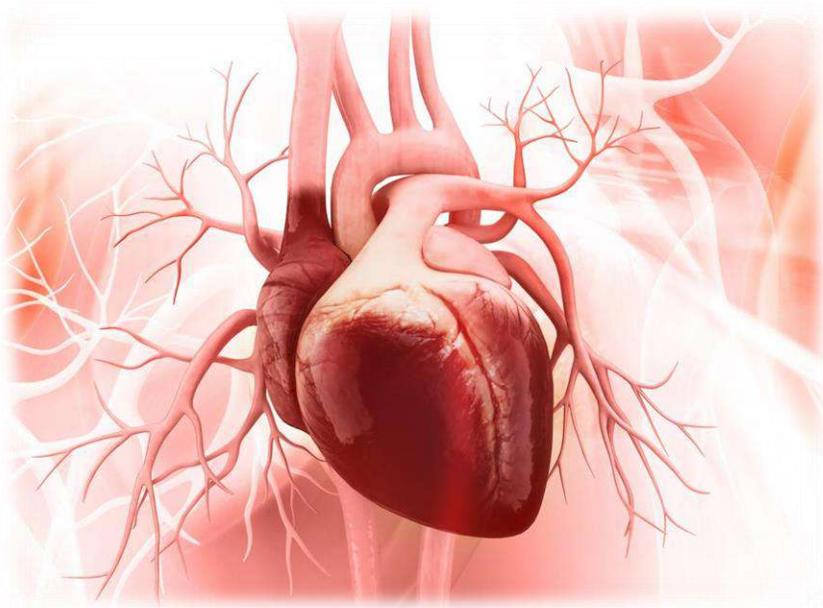
PRESENTACIONES

Jueves 20 Febrero 2020

Colegio de Médicos de Madrid (aula Jiménez Díaz)

Co-Directores: Dra. Marta Cobo, Dr. Alberto Esteban y
Dr. Juan Górriz





Novedades en Insuficiencia Cardíaca en 2019

Dr. Ramón Bover - Programa de Insuficiencia Cardíaca
Hospital Universitario Clínico San Carlos, Madrid

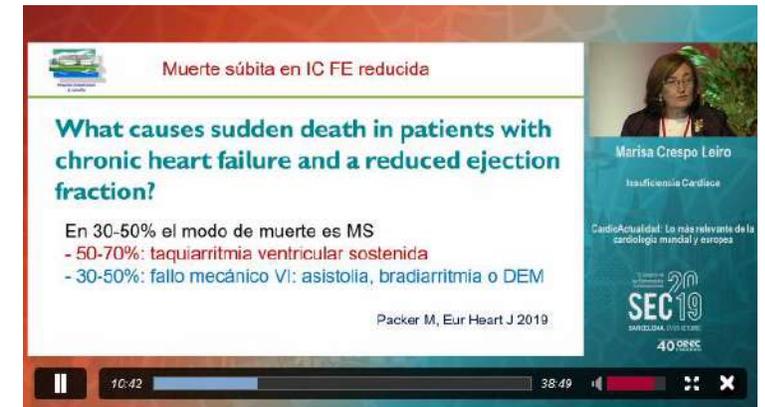


Lo mejor del 2019 en IC crónica...

Visión personal

No está todo, pero lo que está merece
la pena

No solo publicaciones, también
trending topics



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SOCIEDAD
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CARDIOLOGÍA

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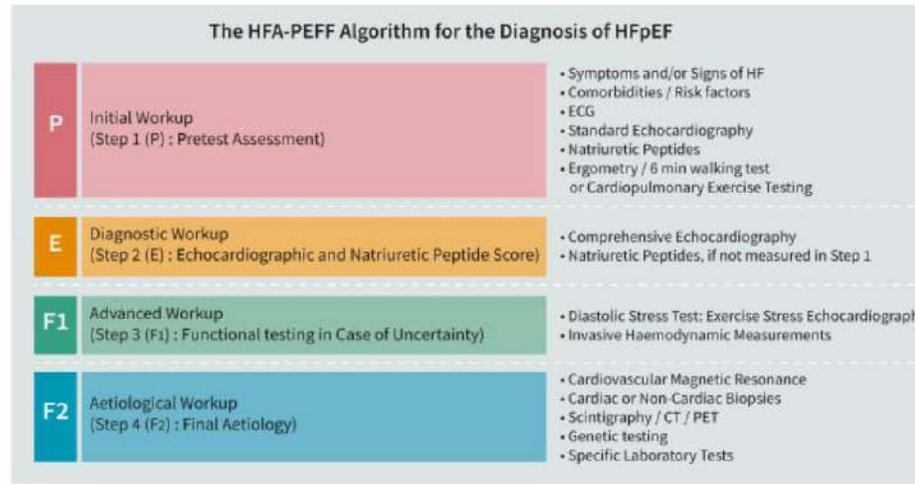
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Clinico - Leganés - Alcalá - Fuenlabrada

How to diagnose heart failure with preserved ejection fraction: the HFA-PEFF diagnostic algorithm: a consensus recommendation from the Heart Failure Association (HFA) of the European Society of Cardiology (ESC)

European Heart Journal (2019) 40, 3297–3317



	Functional	Morphological	Biomarker (SR)	Biomarker (AF)
Major	septal $e' < 7$ cm/s or lateral $e' < 10$ cm/s or Average $E/e' \geq 15$ or TR velocity > 2.8 m/s (PASP > 35 mmHg)	LAVI > 34 ml/m ² or LVMi $\geq 149/122$ g/m ² (m/w) and RWT > 0.42 #	NT-proBNP > 220 pg/ml or BNP > 80 pg/ml	NT-proBNP > 660 pg/ml or BNP > 240 pg/ml
Minor	Average $E/e' < 9-14$ or GLS $< 16\%$	LAVI 29-34 ml/m ² or LVMi $> 115/95$ g/m ² (m/w) or RWT > 0.42 or LV wall thickness ≥ 12 mm	NT-proBNP 125-220 pg/ml or BNP 35-80 pg/ml	NT-proBNP 365-660 pg/ml or BNP 105-240 pg/ml
	Major Criteria: 2 points	≥ 5 points: HFpEF		
	Minor Criteria: 1 point	2-4 points: Diastolic Stress Test or Invasive Haemodynamic Measurements		

Figure 3 Step 2 (E): Echocardiographic and natriuretic peptide heart failure with preserved ejection fraction workup and scoring system (diagnostic workup).

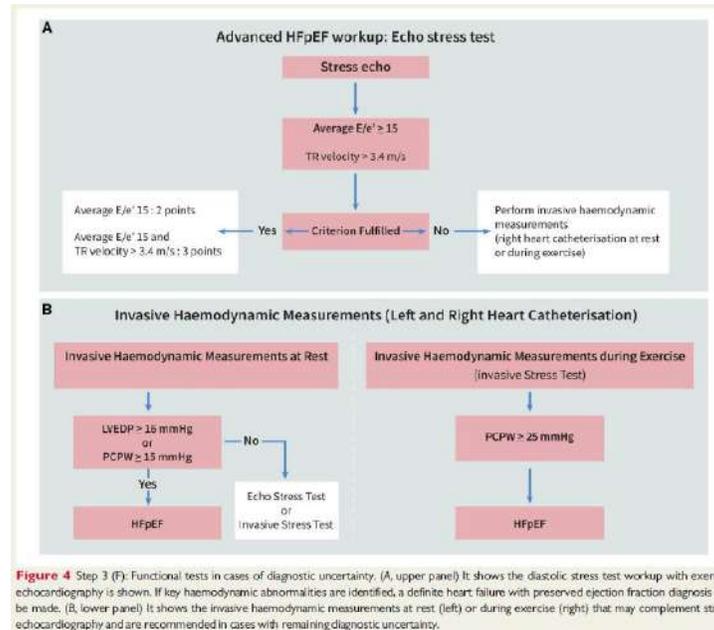


Figure 4 Step 3 (F): Functional tests in cases of diagnostic uncertainty. (A, upper panel) It shows the diastolic stress test workup with exercise echocardiography is shown. If key haemodynamic abnormalities are identified, a definite heart failure with preserved ejection fraction diagnosis can be made. (B, lower panel) It shows the invasive haemodynamic measurements at rest (left) or during exercise (right) that may complement stress echocardiography and are recommended in cases with remaining diagnostic uncertainty.

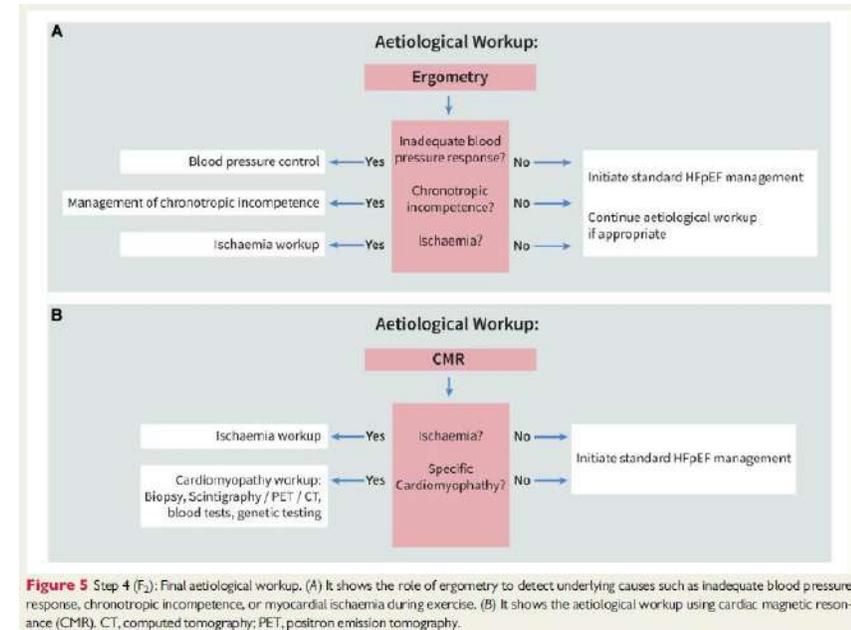


Figure 5 Step 4 (F₂): Final aetiological workup. (A) It shows the role of ergometry to detect underlying causes such as inadequate blood pressure response, chronotropic incompetence, or myocardial ischaemia during exercise. (B) It shows the aetiological workup using cardiac magnetic resonance (CMR). CT, computed tomography; PET, positron emission tomography.



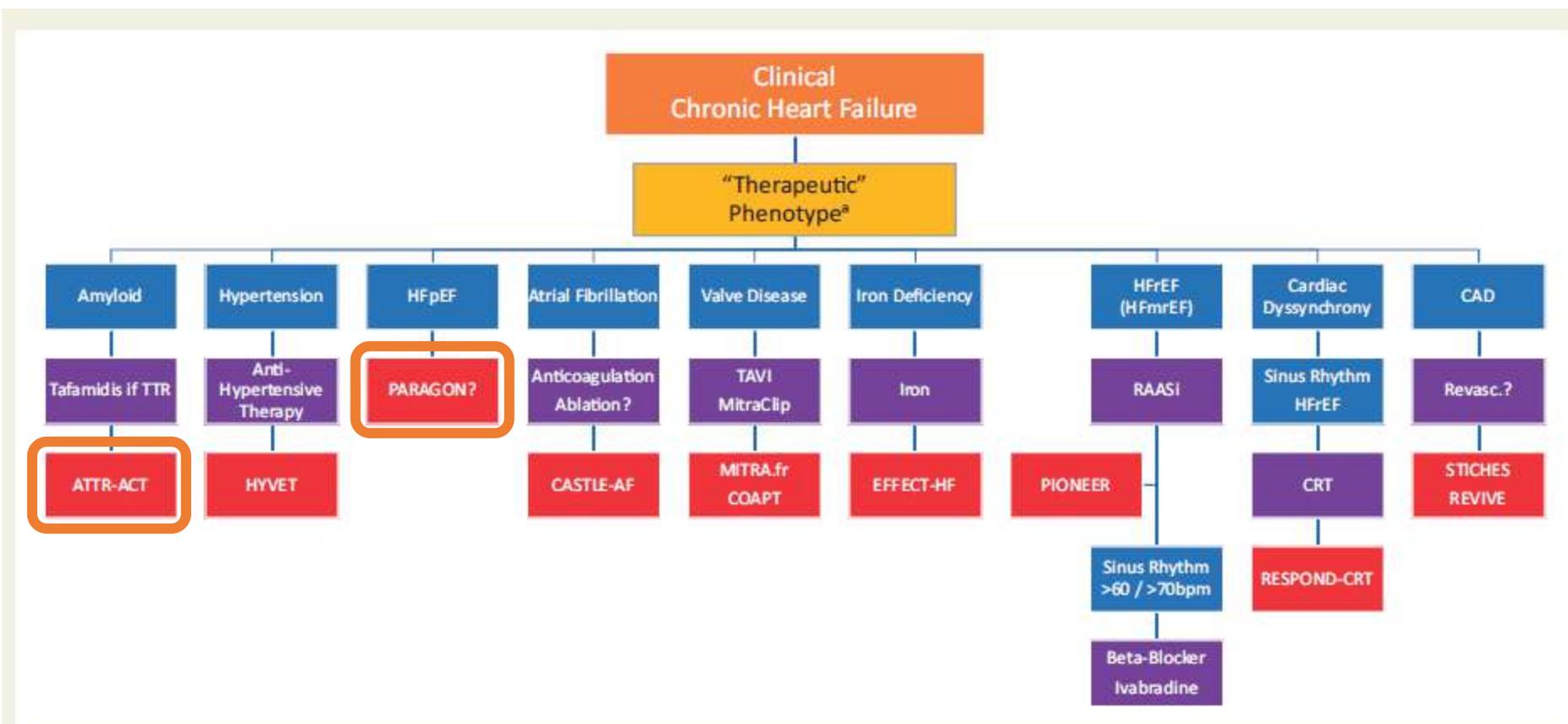
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IC-FEp: Fenotipos



Take home figure Heart failure, classified by 'therapeutic' phenotypes (highlighted in blue) with their relevant treatment (highlighted in purple) and most recent or relevant randomized trial (highlighted in red). ^aA patient may have more than one phenotype.

European Heart Journal (2019) 40, 651–662



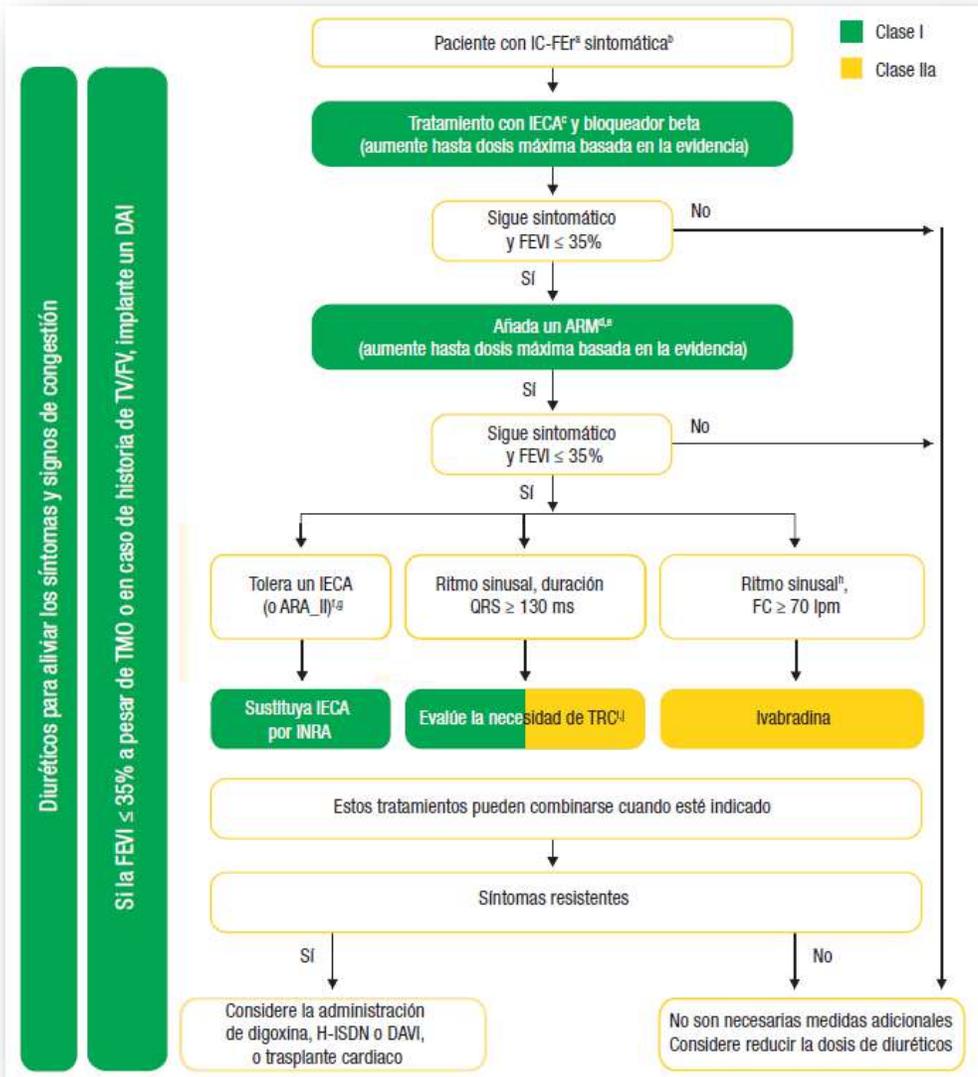
@Cardio_Red1



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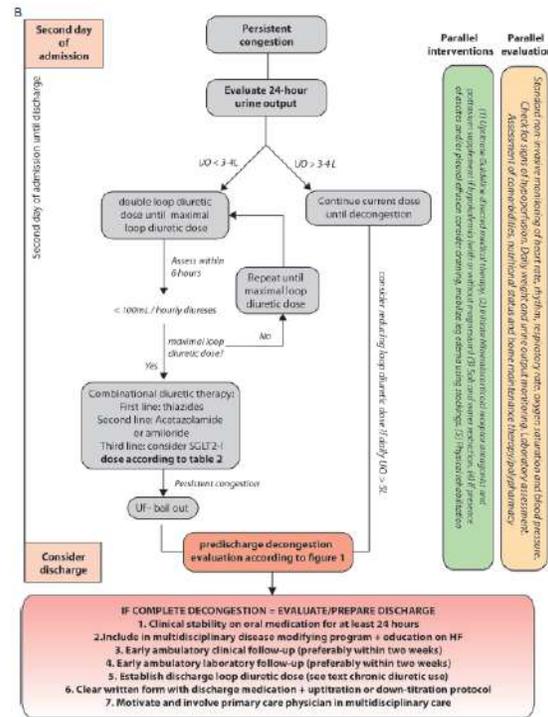
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The use of diuretics in heart failure with congestion — a position statement from the Heart Failure Association of the European Society of Cardiology

Wilfried Mullens^{1,2*}, Kevin Damman³, Veli-Pekka Harjola⁴, Alexandre Mebazaa⁵, Hans-Peter Brunner-La Rocca⁶, Pieter Martens^{1,2}, Jeffrey M. Testani⁷, W.H. Wilson Tang⁸, Francesco Orso⁹, Patrick Rossignol¹⁰, Marco Metra¹¹, Gerasimos Filippatos^{12,13}, Petar M. Seferovic¹⁴, Frank Ruschitzka¹⁵, and Andrew J. Coats¹⁶



European Journal of Heart Failure (2019) 21, 137-155

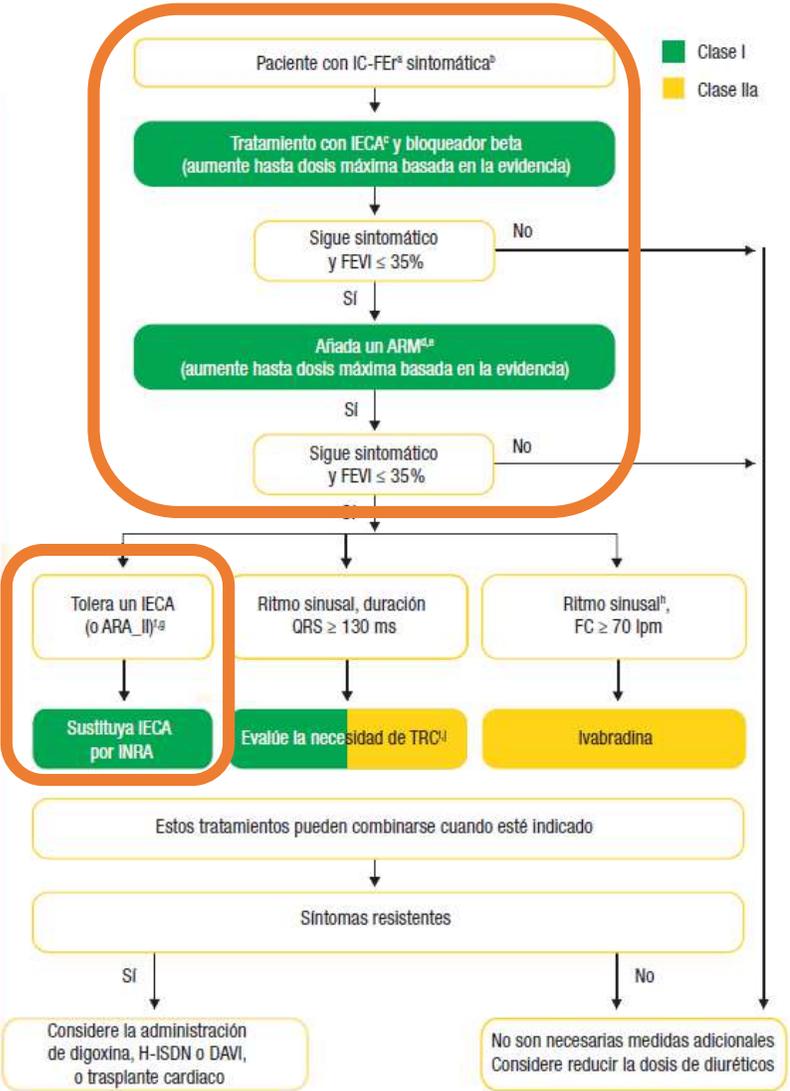
Debate

¿Damos los 3 desde el inicio?

TRED-HF: Withdrawal of pharmacological treatment for heart failure in patients with recovered dilated cardiomyopathy (TRED-HF): an open-label, pilot, randomised trial. Lancet. 2019 Jan 5;393(10166):61-73.

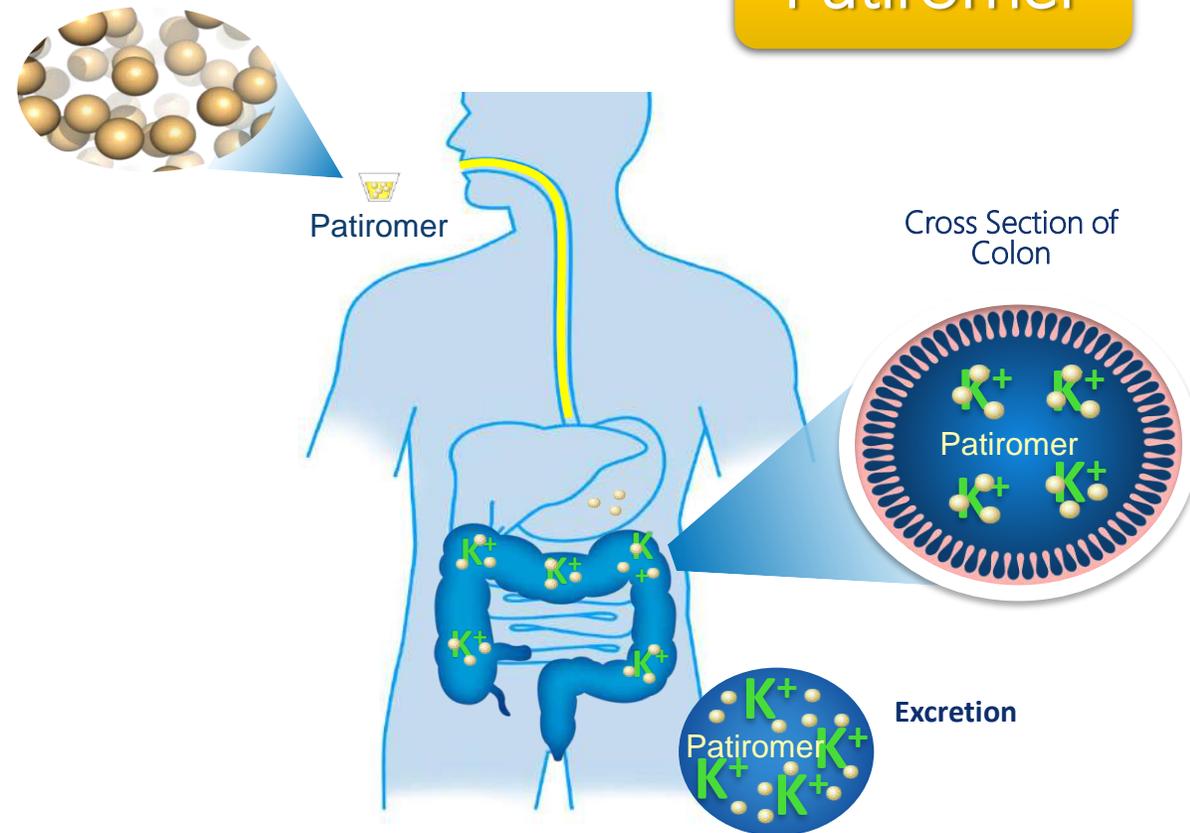
¿Pacientes de novo y naive también?
¿En el paciente hospitalizado?

Diuréticos para aliviar los síntomas y signos de congestión
 Si la FEVI ≤ 35% a pesar de TMO o en caso de historia de TV/FV, implante un DAI



Patiromer

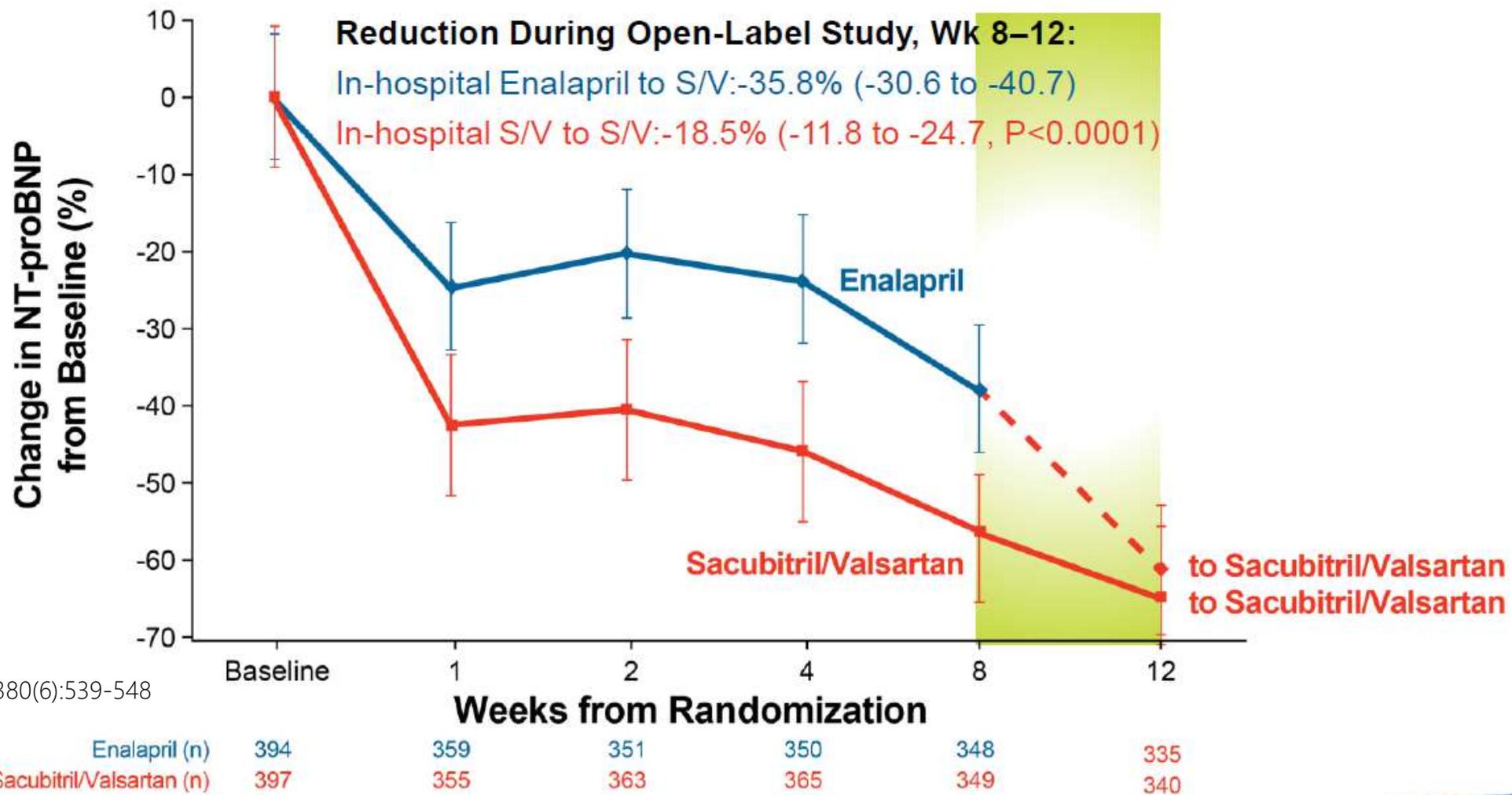
- Patiromer es un polímero de intercambio de cationes que no se absorbe, y que contiene un complejo de calcio-sorbitol como contraión.
- Se une al K^+ intercambiándolo por Ca^{2+} , predominantemente en el colon distal, donde la concentración de K^+ es mayor.
- La unión al K^+ reduce la concentración de K^+ libre en el lumen del colon, lo que produce una reducción de los niveles de K^+ sérico.



El intercambio se realiza mediante Ca^{2+} en lugar de Na^+ , previniendo los potenciales eventos adversos en pacientes con Insuficiencia cardiaca, HT severa o edema (todos, comunes en pacientes con ERC)



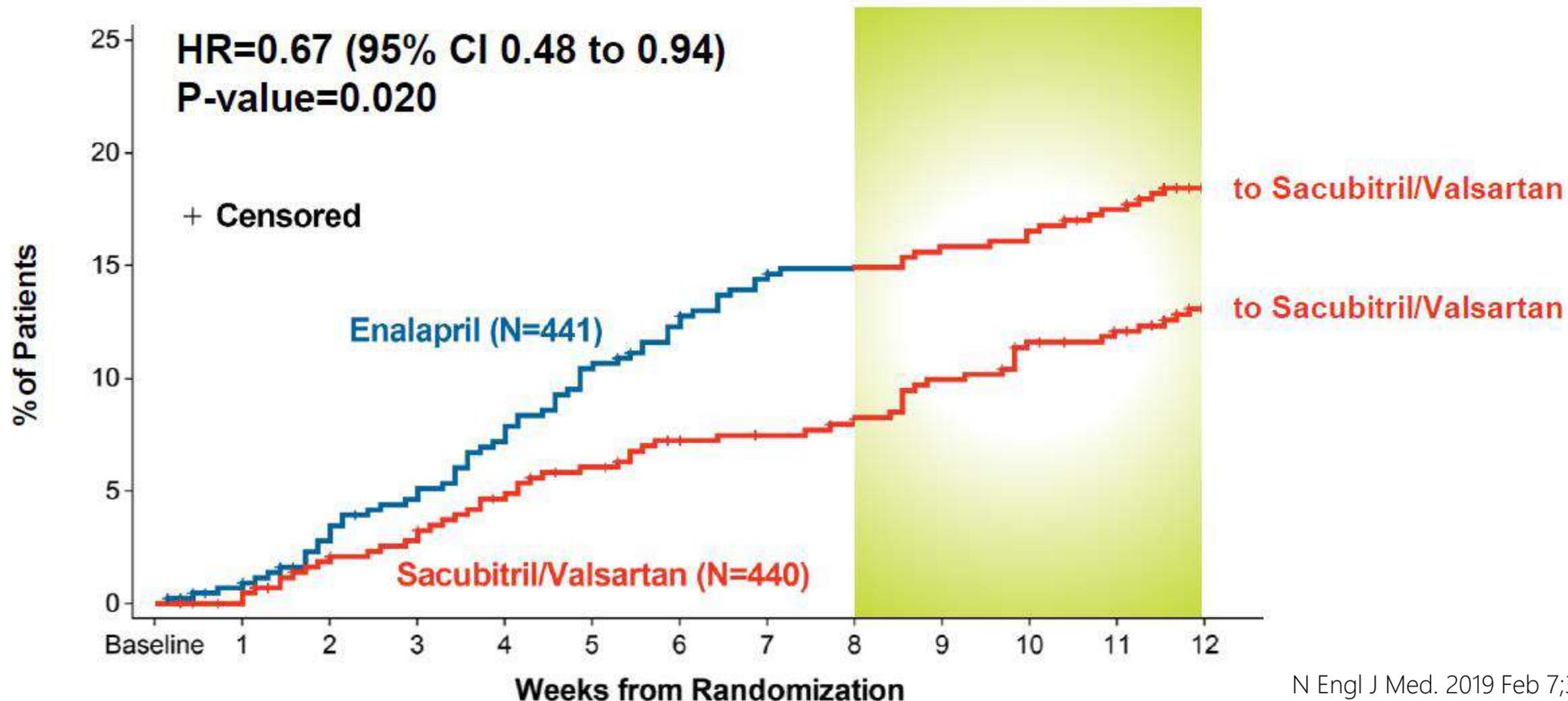
Change from Baseline in NT-proBNP



N Engl J Med. 2019 Feb 7;380(6):539-548



Death, HF Hospitalization, or LVAD Implantation



N Engl J Med. 2019 Feb 7;380(6):539-548

N at Risk

	Baseline	1	2	3	4	5	6	7	8	9	10	11	12
Enalapril (n)	441	432	420	411	400	386	376	366	363	360	358	349	302
Sacubitril/Valsartan (n)	440	435	423	418	408	400	391	388	385	376	368	364	300



These data include
adjudicated HF hospitalizations



ACC.19

Clinical practice update on heart failure 2019: pharmacotherapy, procedures, devices and patient management. An expert consensus meeting report of the Heart Failure Association of the European Society of Cardiology

Petar M. Seferovic¹, Piotr Ponikowski², Stefan D. Anker^{3*}, Johann Bauersachs⁴, Ovidiu Chioncel⁵, John G.F. Cleland⁶, Rudolf A. de Boer⁷, Heinz Drexel⁸, Tuvia Ben Gal⁹, Loreena Hill¹⁰, Tiny Jaarsma¹¹, Ewa A. Jankowska², Markus S. Anker¹², Mitja Lainscak¹³, Basil S. Lewis¹⁴, Theresa McDonagh¹⁵, Marco Metra¹⁶, Davor Milicic¹⁷, Wilfried Mullens¹⁸, Massimo F. Piepoli¹⁹, Giuseppe Rosano²⁰, Frank Ruschitzka²¹, Maurizio Volterrani²², Adriaan A. Voors⁷, Gerasimos Filippatos²³, and Andrew J.S. Coats^{24*}

Implantable cardioverter-defibrillators

Consensus recommendation

The consensus group did not identify any new evidence to alter the 2016 guideline recommendations⁸ on implantable cardioverter-defibrillator (ICD) implantation in patients with HFrEF and CAD.

The consensus view was that *one may consider not to implant* an ICD in patients with non-ischaemic HFrEF who (i) are aged >70 years, or (ii) have advanced symptoms (NYHA class III/IV), or (iii) have life-shortening co-morbidity (e.g. severe lung disease or Stage IV CKD) and hence are likely to die for reasons other than sudden arrhythmic death (SAD).



RECOMENDACIONES DE LA HFA-ESC EN EL PACIENTE **HOSPITALIZADO** CON IC-FEr

Considerar el inicio de **sacubitrilo/valsartán** antes que IECA/ARA II en los **PACIENTES HOSPITALIZADOS RECIEN DIAGNOSTICADOS O DESCOMPENSADOS** para reducir el riesgo a corto plazo de eventos adversos y para simplificar su manejo (evitando la necesidad de titular el IECA primero y luego cambiar a sacubitrilo/valsartán).

Sacubitrilo/valsartán es eficaz y seguro en pacientes naïve: 53% de los pacientes incluidos en PIONEER-HF y 24% en TRANSITION lo eran.

RECOMENDACIONES DE LA HFA-ESC EN EL PACIENTE **AMBULATORIO** CON IC-FEr

Sacubitrilo/valsartán se recomienda como **substituto de IECA/ARAII** para **reducir el riesgo de hospitalización por IC y muerte** en **PACIENTES AMBULATORIOS** con IC-FEr que permanecen sintomáticos a pesar del tratamiento médico optimizado con un IECA, betabloqueante y ARM.

Sacubitrilo/valsartán es **seguro y eficaz** en un amplio espectro de pacientes con IC-FEr, incluidos **PACIENTES NAÏVE**, por lo tanto, **su inicio puede considerarse también en estos pacientes.**

Las combinaciones terapéuticas más eficaces para IC-FEr son:

- i) **sacubitrilo/valsartán + betabloqueante + ARM**
- ii) IECA + ARM + ivabradina

Estas combinaciones reducen la mortalidad por todas las causas (vs placebo) un **62%** (i) y **59%** (ii), y las hospitalizaciones por todas las causas un **42%** en ambos casos



Actualización ESC 2019

- ▶ **iSGLT2 (Sodium–glucose co-transporter 2 inhibitors):** “The 2016 guidelines indicated that empagliflozin should be considered in patients with type 2 diabetes mellitus (T2DM) in order to prevent or delay the onset of HF or prolong life. The 2019 expert consensus was that canagliflozin and dapagliflozin should also be considered for patients with T2DM and either established cardiovascular (CV) disease or at high CV risk in order to prevent or delay the onset of and hospitalizations for HF. At this stage, no specific recommendations for the use of sodium–glucose co-transporter 2 (SGLT2) inhibitors in patients with established HF can be made.”
- ▶ **Quelantes de potasio:** “Patiromer and ZS-9 may be considered in patients with HF with or without CKD to manage hyperkalaemia. In selected patients these therapies may enable use of MRAs and other renin–angiotensin–aldosterone system inhibitors (RAASi) in more patients and at higher doses, but it is not known whether this will improve patient outcomes. Patiromer and ZS-9 may be considered in selected patients with HF with or without CKD in order to enable up-titration of MRA while avoiding hyperkalaemia.”



Hot Line Session 1

SESSION

Congress : ESC CONGRESS 2019

Topic : European Society Of Cardiology - Other

Session type : Late-Breaking Science

Date : 01 September 2019

Time : 14:30 - 16:10

DAPA HF - The Dapagliflozin And Prevention Of Adverse-outcomes In Heart Failure Trial

★★★★★ (29)

Congress : ESC Congress 2019

Session : Hot Line Session 1

Speaker : J McMurray (Glasgow,GB)

Abstract

Slides

Video

ESC TV



PARAGON-HF - Angiotensin Receptor Neprilysin Inhibition in Heart Failure with Preserved Ejection Fraction

★★★★★ (6)

Congress : ESC Congress 2019

Session : Hot Line Session 1

Speaker : S Solomon (Boston,US)

Abstract

Slides

Video

ESC TV

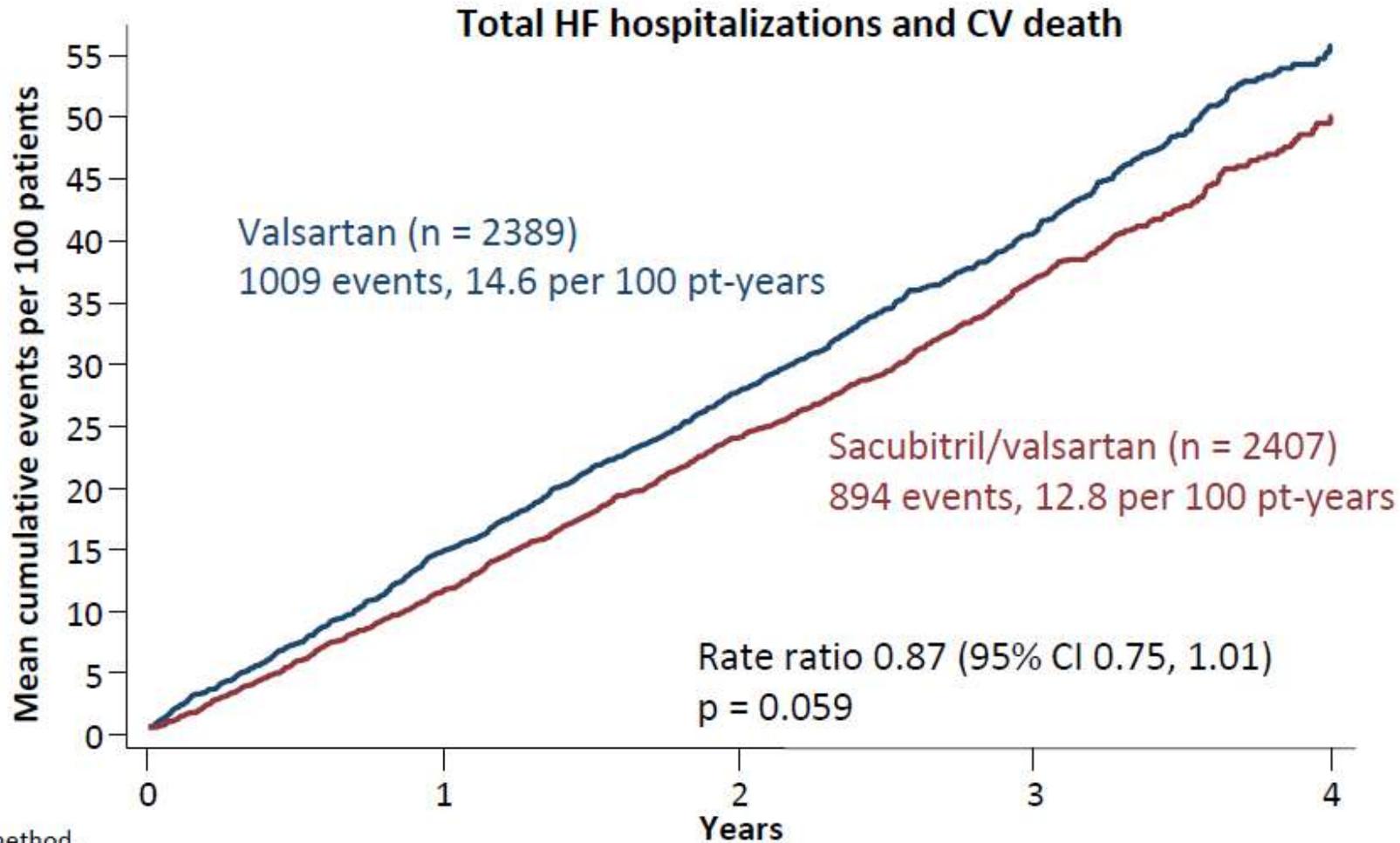


PARAGON-HF primary results

Recurrent event analysis of total HF hospitalizations and CV death*

PARAGON-HF

N Engl J Med. 2019 Oct
24;381(17):1609-1620



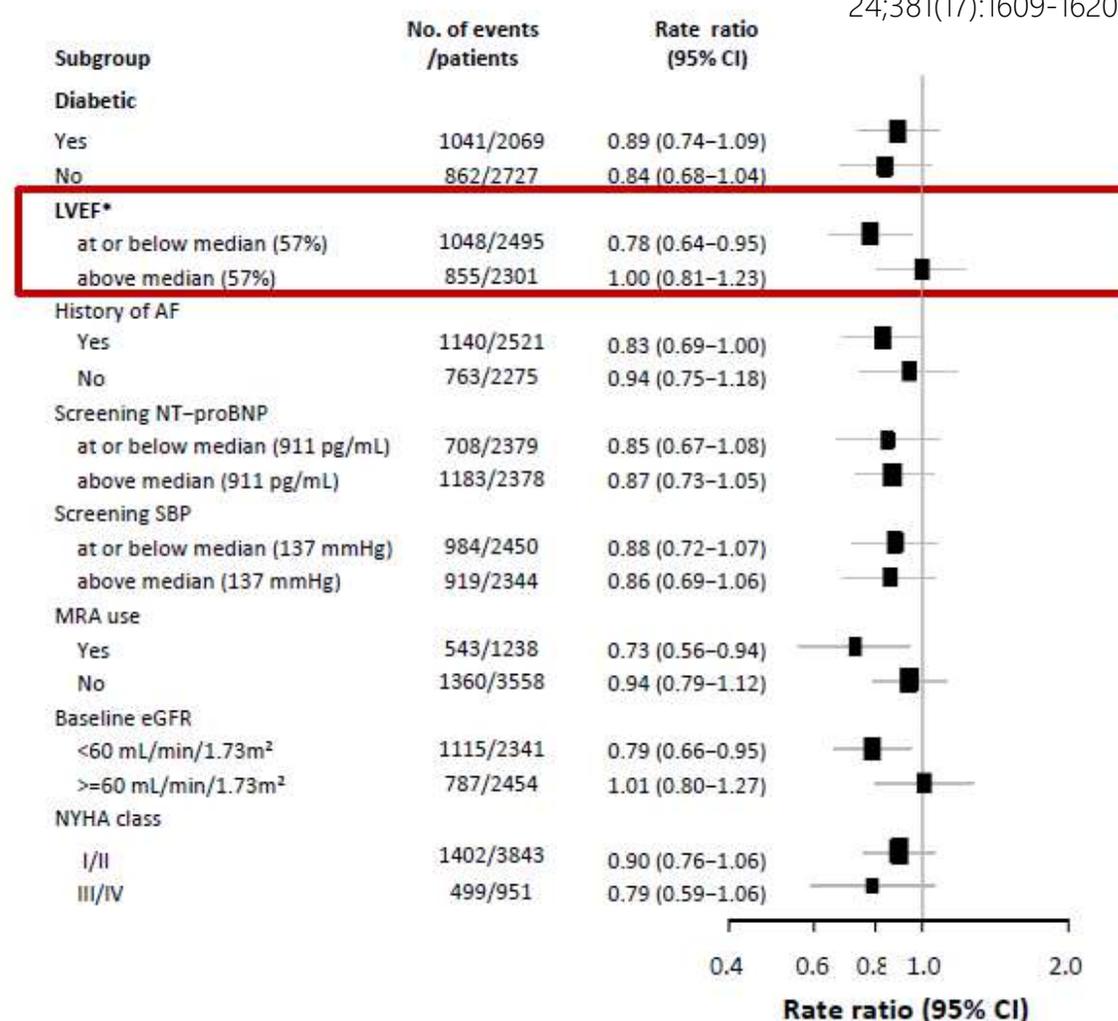
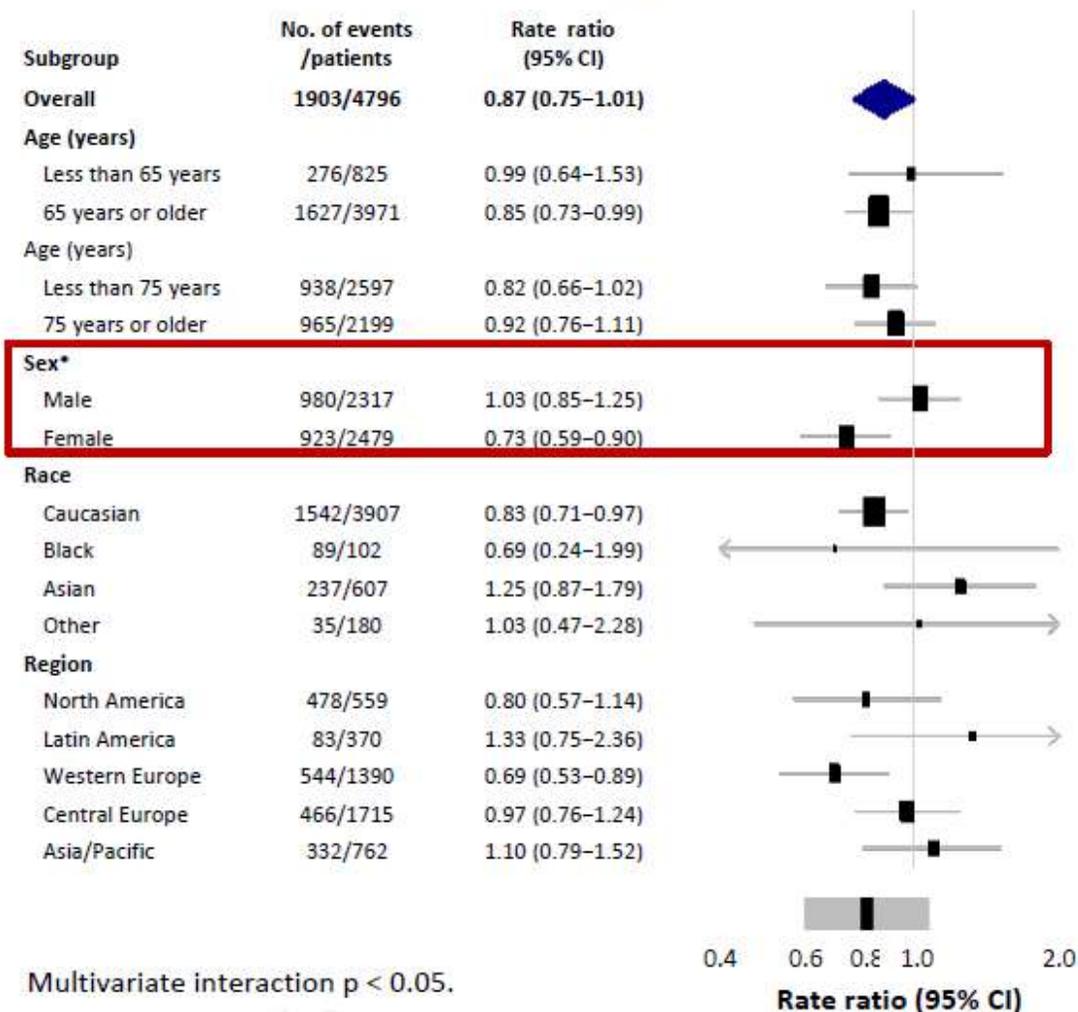
*Semiparametric LWYY method.

Pre-specified subgroups for primary endpoint

PARAGON-HF

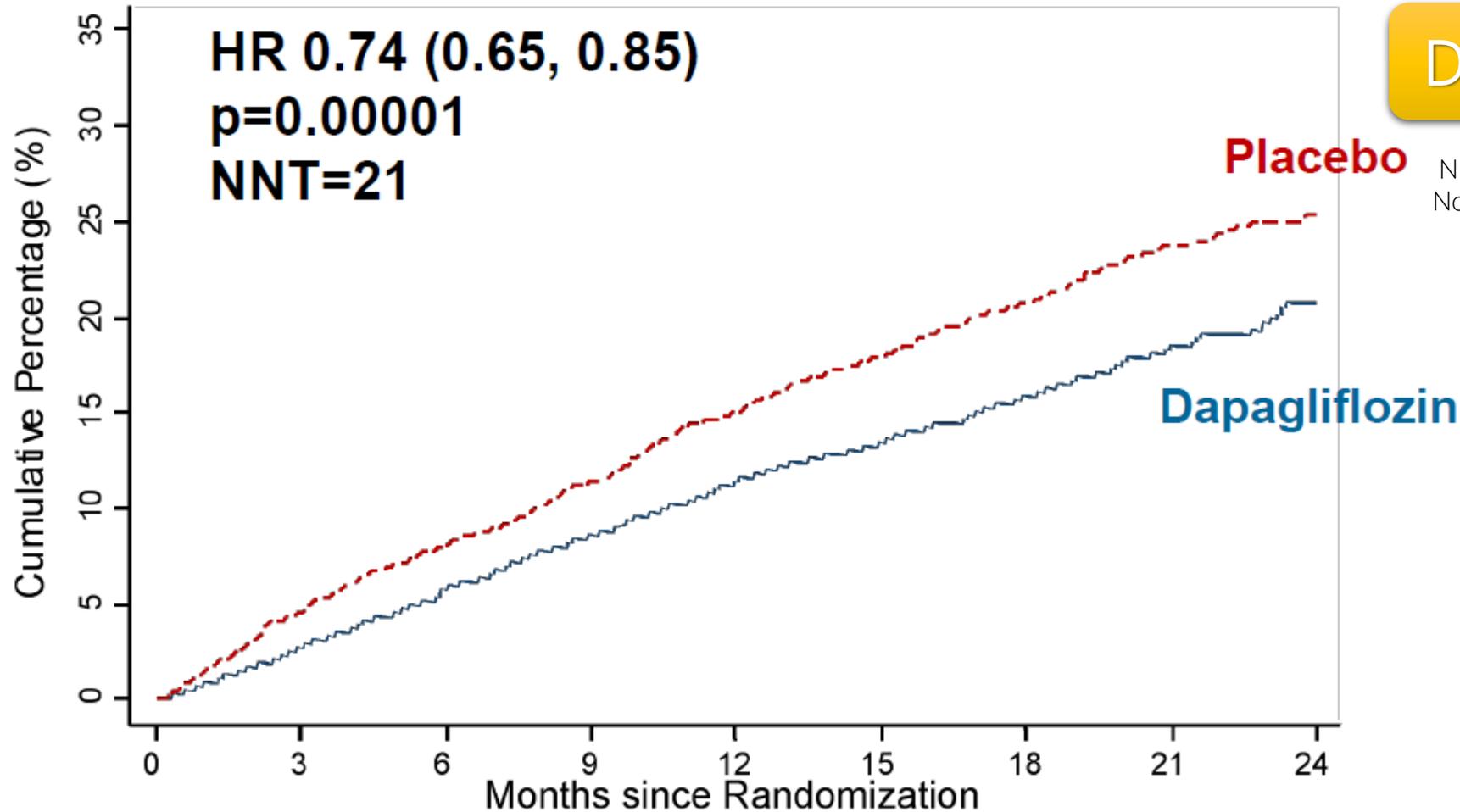
Evidence for overall heterogeneity

N Engl J Med. 2019 Oct 24;381(17):1609-1620



Primary composite outcome

CV Death/HF hospitalization/Urgent HF visit



DAPA-HF

N Engl J Med. 2019
Nov 21;381(21):1995-
2008

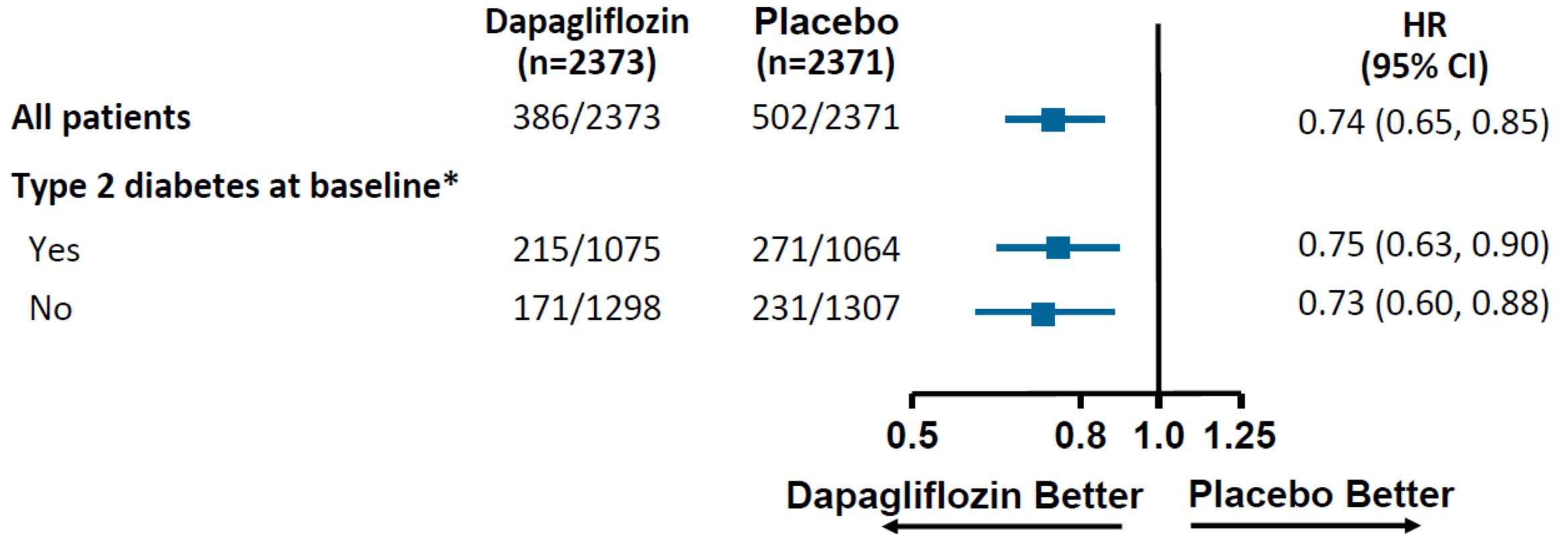
Number at Risk

Dapagliflozin	2373	2305	2221	2147	2002	1560	1146	612	210
Placebo	2371	2258	2163	2075	1917	1478	1096	593	210

No diabetes/diabetes subgroup: Primary endpoint

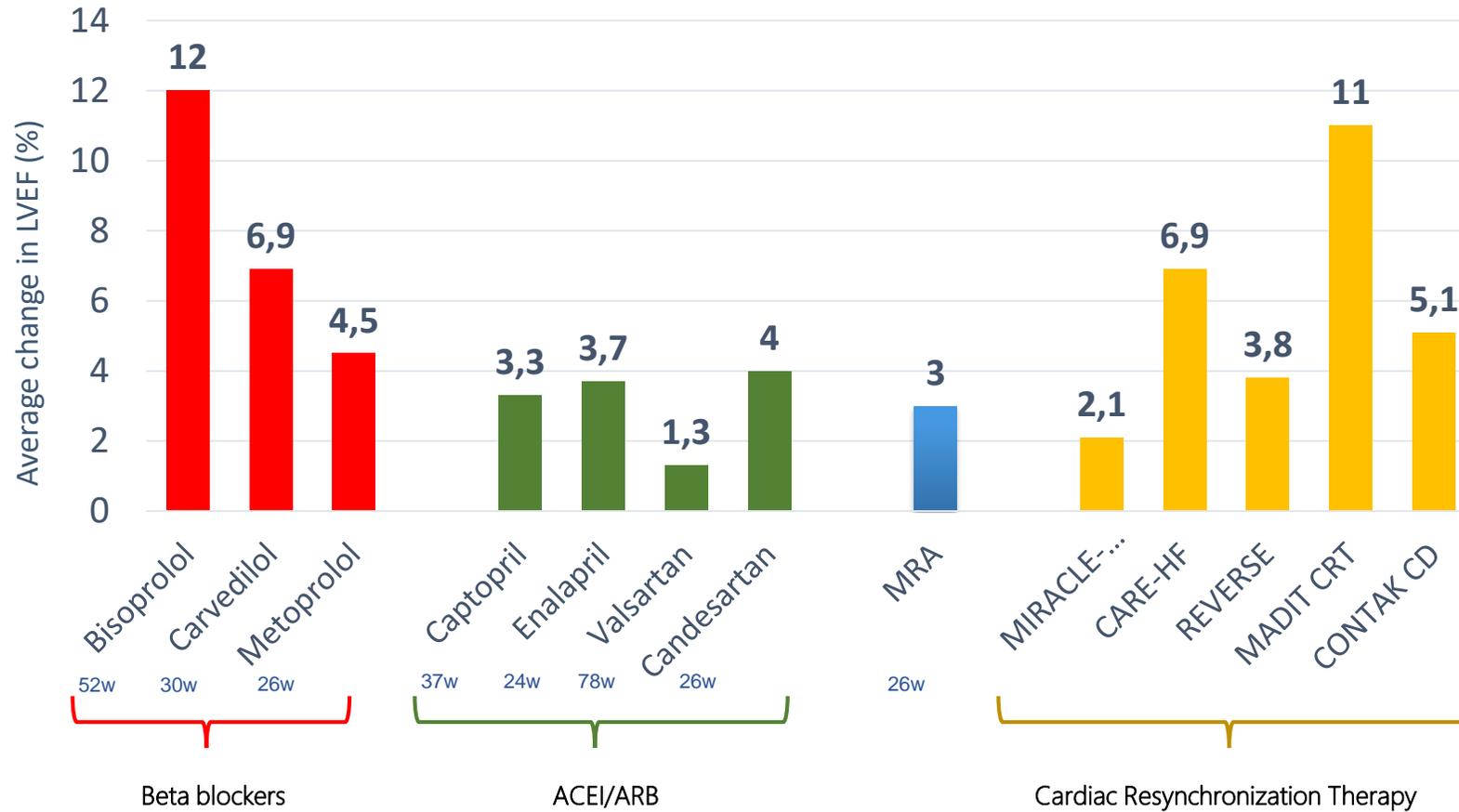
DAPA-HF

N Engl J Med. 2019
Nov 21;381(21):1995-
2008



*Defined as history of type 2 diabetes or HbA1c $\geq 6.5\%$ at both enrollment and randomization visits.

Remodelado inverso



Prog. Cardiovasc. Dis. 2017;60:226-236

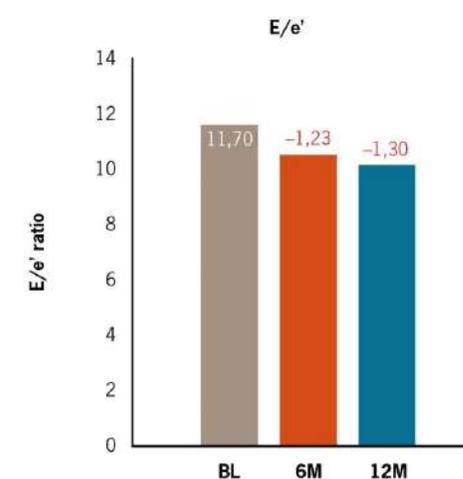
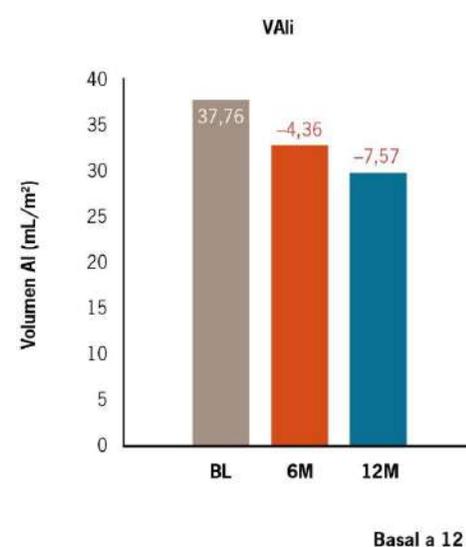
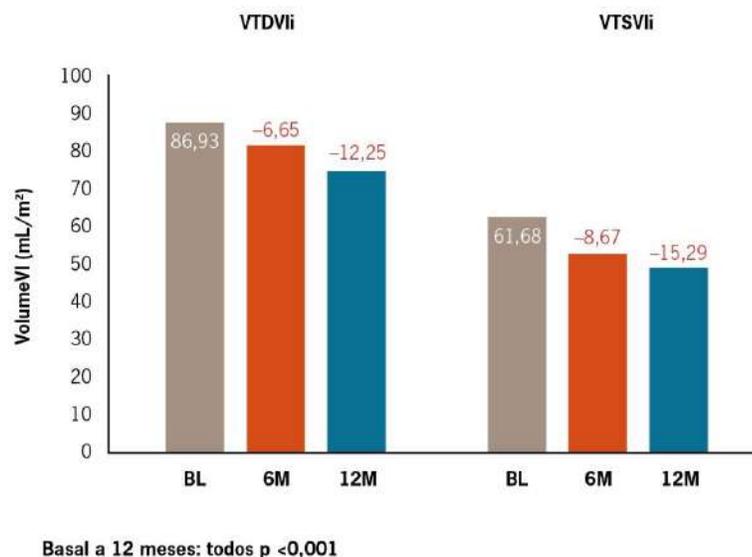
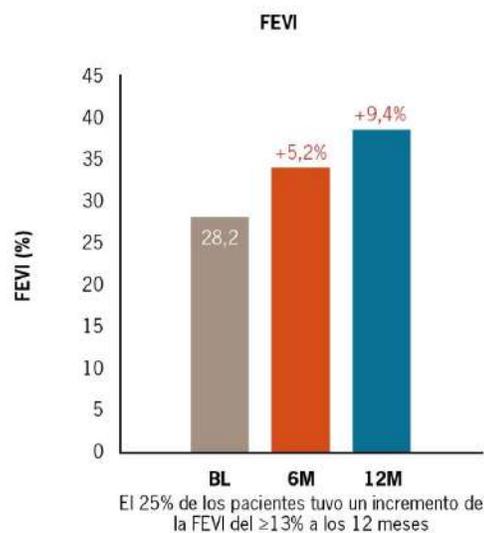


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Remodelado inverso con Sacubitrilo/valsartán

PROVE-HF



JAMA. 2019 Sep 2:1-11



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Subgrupos preespecificados de interés

- El remodelado cardiaco inverso fue comparable en cada subgrupo preespecificado de interés

Todos p <0,001, excepto donde se indique

IC de novo y/o naïve para IECA/ARA II (N= 118)		Concentración NP < criterios de inclusión PARADIGM* (N= 292)		No alcanzan dosis objetivo (N= 278)	
Parámetro	Cambio en LSM, BL a 12 meses (IC del 95%)	Parámetro	Cambio en LSM, BL a 12 meses (IC del 95%)	Parámetro	Cambio en LSM, BL a 12 meses (IC del 95%)
FEVI (%)	+12,8 (+11,05; +14,5)	FEVI (%)	+9,4 (+8,6; +10,3)	FEVI (%)	+9,4 (+8,4; +10,3)
VTDVli (mL/m ²)	-13,81 (-15,78; -11,83)	VTDVli (mL/m ²)	-11,32 (-12,24; -10,40)	VTDVli (mL/m ²)	-10,99 (-12,21; -9,77)
VTSVli (mL/m ²)	-17,88 (-20,07; -15,68)	VTSVli (mL/m ²)	-14,15 (-15,15; -13,15)	VTSVli (mL/m ²)	-14,32 (-15,67; -12,97)
VAli (mL/m ²)	-8,44 (-9,73; -7,15)	VAli (mL/m ²)	-7,06 (-7,54; -6,58)	VAli (mL/m ²)	-7,23 (-7,9; -6,50)
E/e'	-2,60 (-3,83; -1,37)	E/e'	-0,93 (-1,43; -0,43)	E/e'	-0,46 (-1,32; +0,40); P= NS

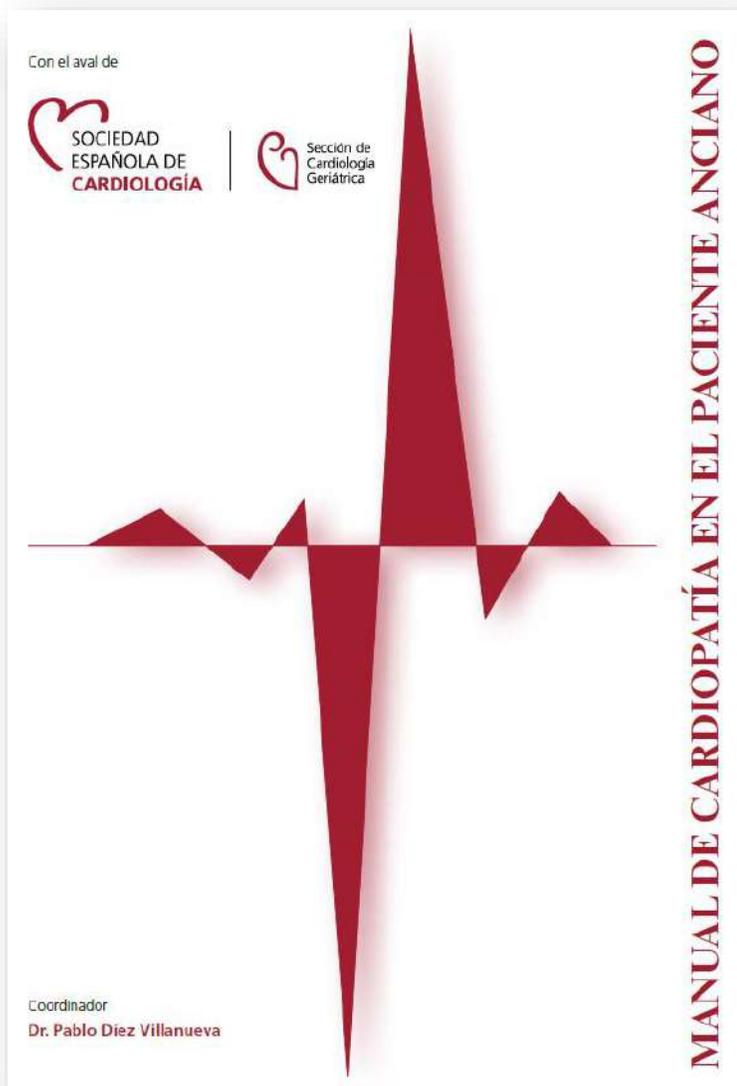
*NT-proBNP <600 pg/mL si no está hospitalizado o <400 pg/mL si fue hospitalizado en los últimos 12 meses; BNP <150 pg/mL si no está hospitalizado o <100 pg/mL si fue hospitalizado por IC en los últimos 12 meses.

JAMA. 2019 Sep 2:1-11



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Consensos

Documento de consenso y recomendaciones sobre cuidados paliativos en insuficiencia cardiaca de las Secciones de Insuficiencia Cardiaca y Cardiología Geriátrica de la Sociedad Española de Cardiología



José Manuel García Pinilla^{a,b}, Pablo Díez-Villanueva^{c,*}, Ramón Bover Freire^{b,d}, Francesc Formiga^e, Marta Cobo Marcos^{b,f}, Clara Bonanad^{b,g}, María G. Crespo Leiro^{b,h}, Juan Ruiz Garcíaⁱ, Beatriz Díaz Molina^j, Cristina Enjuanes Grau^k, Lluisa García^l, Lourdes Rexach^m, Alberto Estebanⁿ y Manuel Martínez-Sellés^{b,o}

Rev Esp Cardiol. 2020;73:69-77

Recomendaciones de la Sección de Cardiología Geriátrica de la Sociedad Española de Cardiología para la valoración de la fragilidad en el anciano con cardiopatía



Pablo Díez-Villanueva^{a,*}, Albert Arizá-Solé^b, María Teresa Vidán^c, Clara Bonanad^d, Francesc Formiga^e, Juan Sanchis^{f,g}, F. Javier Martín-Sánchez^h, Vicente Ruiz Ros^f, Marcelo Sanmartín Fernándezⁱ, Héctor Bueno^{j,k,l} y Manuel Martínez-Sellés^m

Rev Esp Cardiol. 2019;72(1):63-71

Consenso de expertos en el manejo multidisciplinar y abordaje integral de la insuficiencia cardiaca. *Desde el alta hospitalaria hasta la continuidad asistencial con primaria.*

Autores: Grupo de Trabajo de insuficiencia cardiaca y rehabilitación cardiaca (secciones de insuficiencia cardiaca y riesgo vascular y rehabilitación cardiaca de la SEC)

www.secardiologia.es - Publicaciones



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Nuevas estrategias para evaluar la efectividad de servicios de insuficiencia cardiaca

Novel strategies to evaluate the effectiveness of heart failure services

William J. McKenna^{a,b,c,*} y María Nieves Montoro-López^a

^aHeart Hospital, Hamad Medical Corporation, Doha, Qatar

^bInstitute of Cardiovascular Science, University College London, Londres, Reino Unido

^cSection of Cardiovascular Diseases, Yale School of Medicine and YNHH Heart and Vascular Center, New Haven, CT, Estados Unidos

DOI: 10.1016/j.recesp.2019.08.017



Gracias



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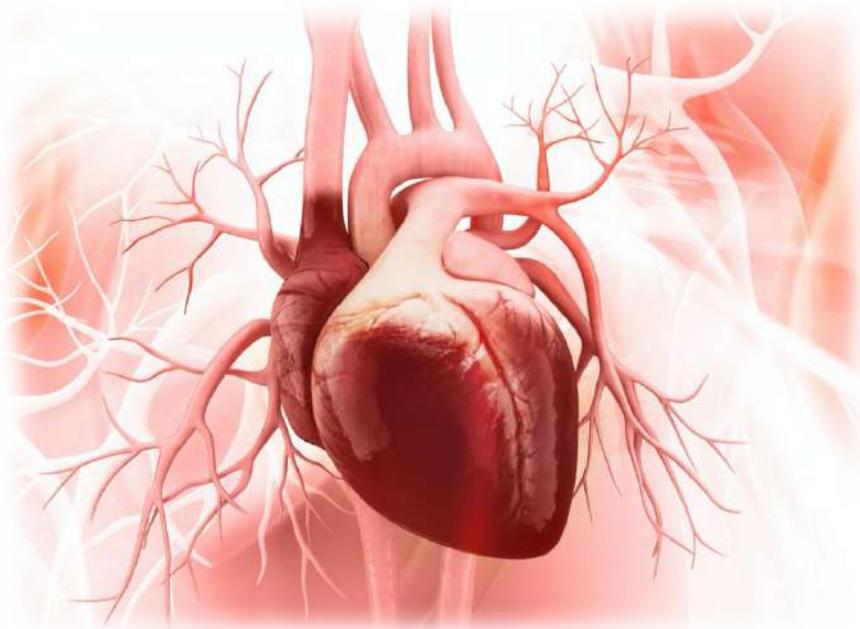


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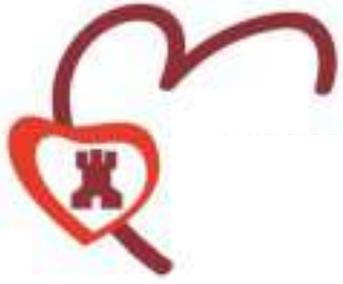




¿Cómo debo tratar al paciente con insuficiencia cardiaca aguda?



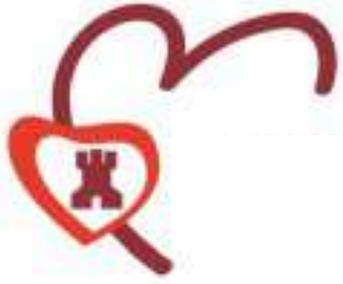
Angel Manuel Iniesta Manjavacas



¿Qué es la ICA?

- Aparición o empeoramiento, rápidos, de síntomas y/o signos de insuficiencia cardiaca
- Requiere un diagnóstico y tratamiento rápido, muchas veces con ingreso hospitalario
- Puede suponer IC “de novo” o ser una descompensación de IC crónica

Eur Heart J. 2016 Jul 14;37(27):2129-2200.

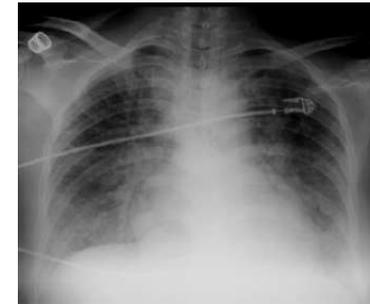


Alteraciones fisiopatológicas

Congestión sistémica



Congestión pulmonar



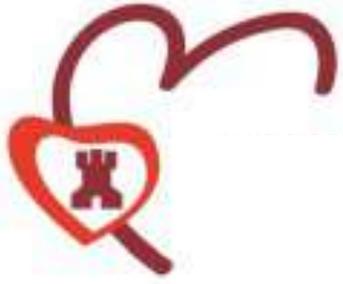
Hipoperfusión tisular



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**INSUFICIENCIA
CARDIACA**



Evaluación inicial

- ✓ **Insuficiencia respiratoria**
- ✓ **Hipoperfusión tisular (shock)**
- ✓ Causas tratables “CHAMPI”:
 - síndrome **C**oronario agudo
 - emergencia **H**ipertensiva
 - **A**rritmia
 - complicación **M**ecánica
 - embolia **P**ulmonar
 - **I**nfección





Congestión sistémica



Diuréticos



- De elección la vía intravenosa
- Bolos o perfusión continua
- Diuréticos de asa → furosemida, dosis inicial:
 - *Naive*: 20-40 mg
 - Tratamiento crónico: al menos equivalente a la dosis oral previa
- Respuesta insuficiente: combinación (tiazidas, antialdosterónicos)



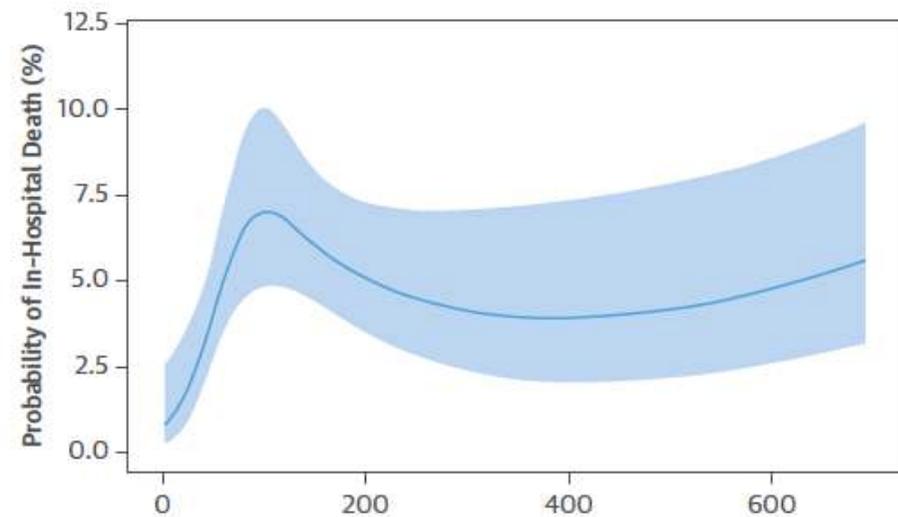
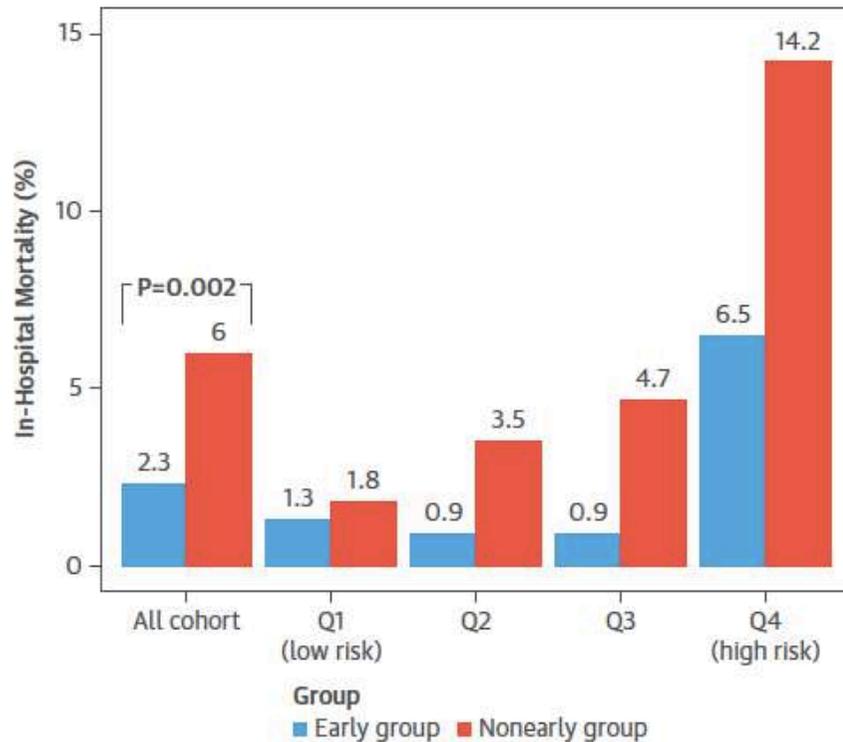


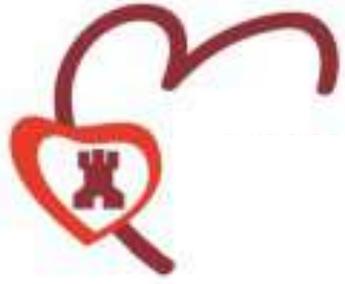
Congestión sistémica



Diuréticos

- Administración precoz (< 60 min)





Congestión sistémica



Terapia de sustitución renal

- Ultrafiltración
- Hemo(dia)filtración veno-venosa continua
- Diálisis

Indicaciones:

- Acidosis ($\text{pH} < 7.2$)
- Electrolitos ($\text{K} > 6.5$)
- Intoxicaciones
- Overload (ICA refractaria a diuréticos)
- Uremia (en situación aguda $> 120 \text{ mg/dL}$)



FLASHLIGHTS en

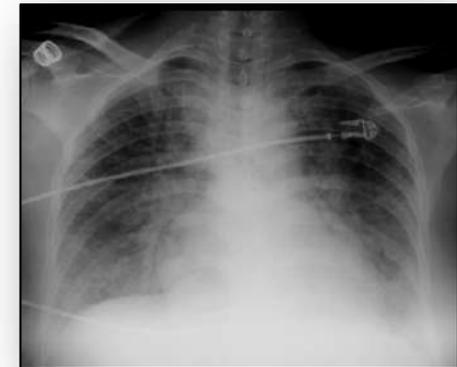


**INSUFICIENCIA
CARDIACA**



Congestión pulmonar

- Tratamiento de la **insuficiencia respiratoria** aguda:
 - Oxigenoterapia convencional
 - Ventilación no invasiva
 - Ventilación mecánica invasiva
- Vasodilatadores
- Diuréticos (y TSR)



FLASHLIGHTS en



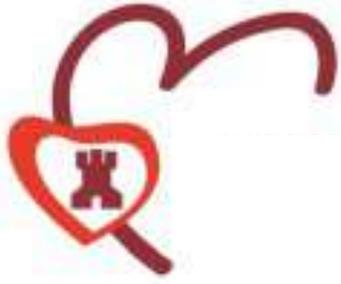
**INSUFICIENCIA
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Insuficiencia respiratoria

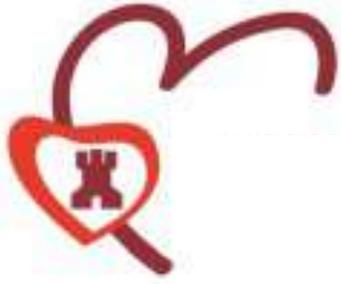
- Oxigenoterapia convencional:
 - Gafas nasales / mascarilla efecto Venturi / reservorio
 - Indicada si $\text{satO}_2 < 90\%$ ($\text{PaO}_2 < 60 \text{ mmHg}$)
 - Considerar si disnea y satO_2 90-93%
 - Objetivo satO_2 95-98% (evitar hiperoxia)





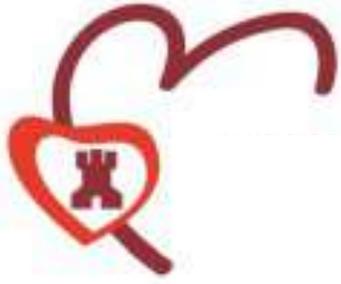
Insuficiencia respiratoria

- **Ventilación mecánica no invasiva**
 - Mejoría más rápida de la disnea, reduce necesidad de IOT, posible reducción mortalidad
 - Indicado en situación de **distress respiratorio** (FR > 25 rpm, SpO2 < 90%)
 - **Edema agudo de pulmón**
 - Podría considerarse en shock cardiogénico
 - Otras formas ICA



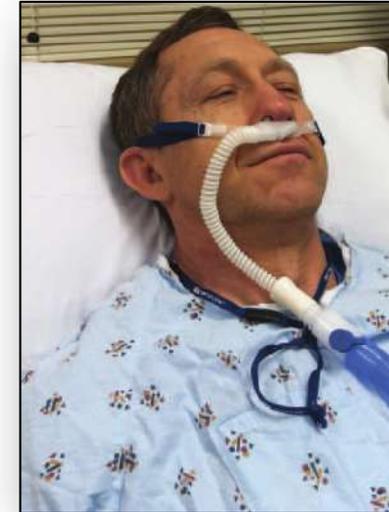
Insuficiencia respiratoria

- Ventilación mecánica no invasiva
 - **CPAP:**
 - Muy sencilla, no requiere ventilador, uso extrahospitalario
 - No asiste la inspiración
 - Indicaciones: **EAP**, atelectasias, SAOS
 - **NIPSV (BiPAP):**
 - Da soporte ventilatorio, con CPAP y ayuda inspiratoria
 - Requiere ventilador, más compleja
 - Indicaciones: EAP, **EPOC** con ICA, insuficiencia respiratoria **hipercápnica**, destete



Insuficiencia respiratoria

- Gafas nasales de alto flujo
 - Sencillas, no requieren ventilador, fácil adaptación
 - No asistencia inspiratoria
 - Indicaciones: pre-EAP, insuficiencia respiratoria hipoxémica, intolerancia VMNI, destete





Insuficiencia respiratoria

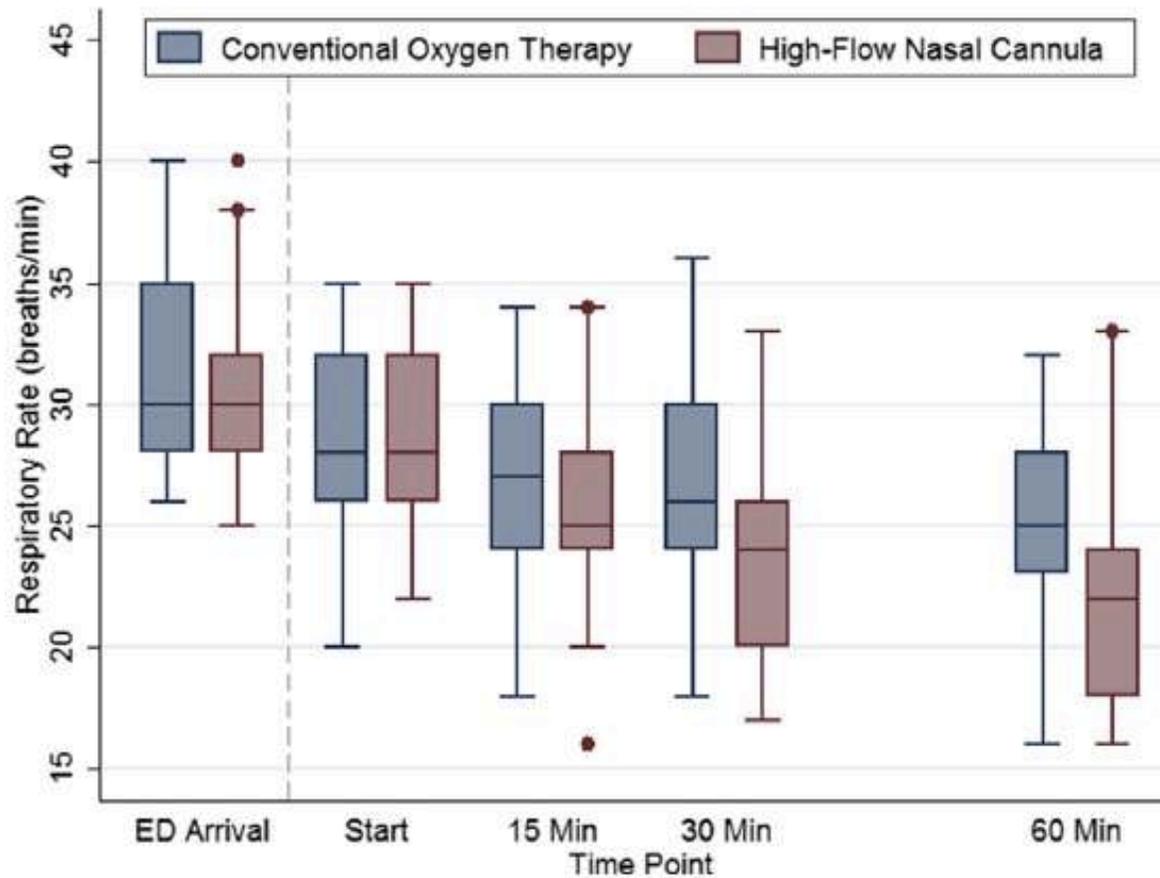
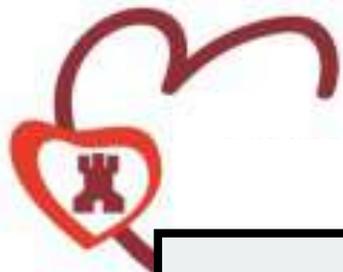
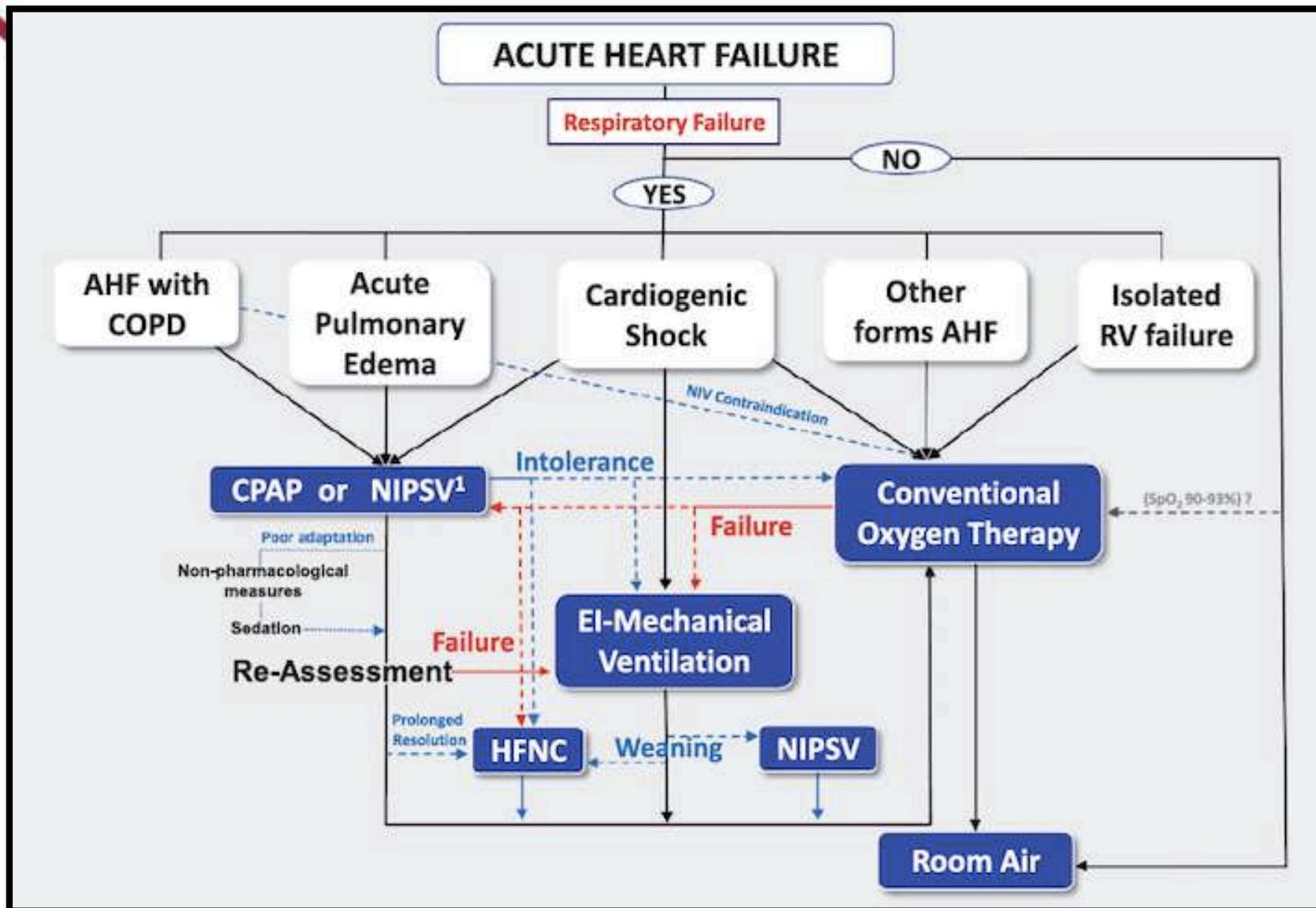
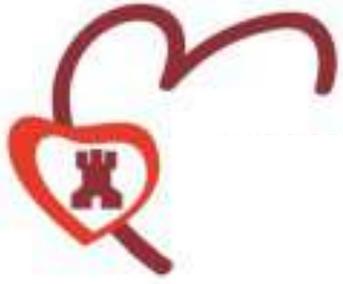


Figure 3. Respiratory rate at each point.



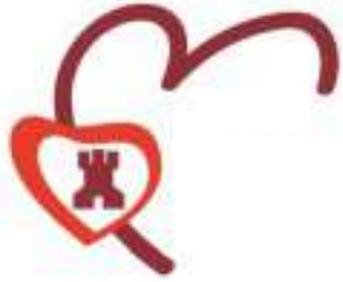
Insuficiencia respiratoria



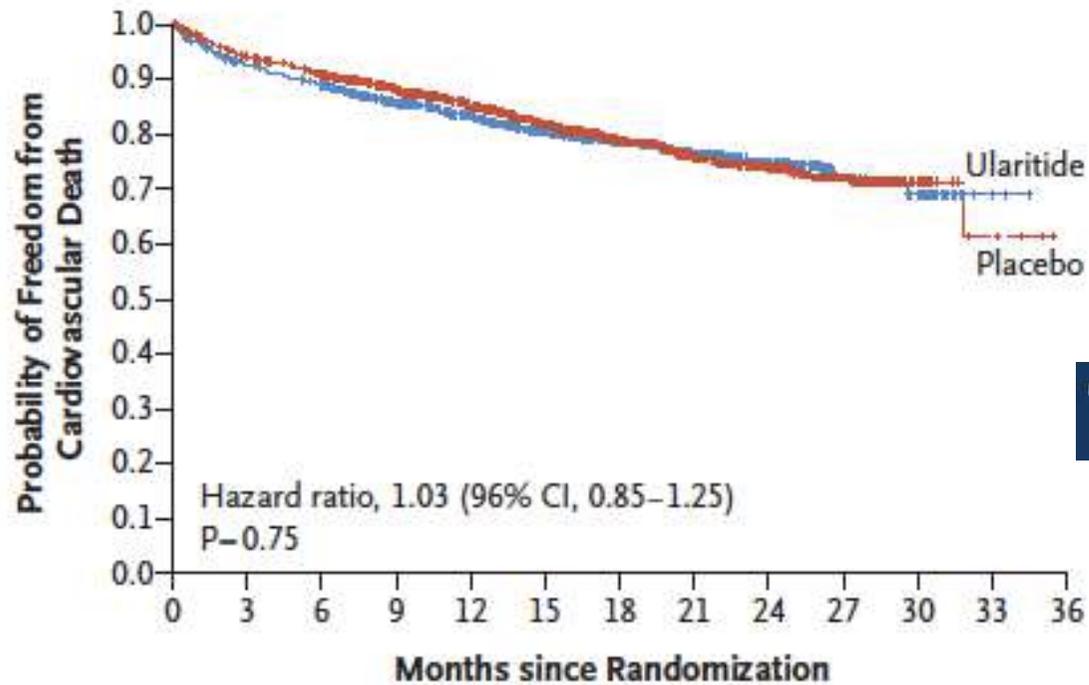


Vasodilatadores

- Vasodilatadores i.v. → indicados en el tto sintomático de los pacientes con TAS > 90 mmHg (sin hipotensión sintomática)
 - Edema agudo de pulmón
 - ICA hipertensiva
 - Síndrome coronario agudo
- **Nitroglicerina**, nitroprusiato, nesiritide, ...



Vasodilatadores



TRUE-AHF

No. at Risk

Ularitide	1088	988	942	789	669	546	456	356	234	106	26	2	0
Placebo	1069	987	934	786	668	547	444	338	219	104	19	5	0

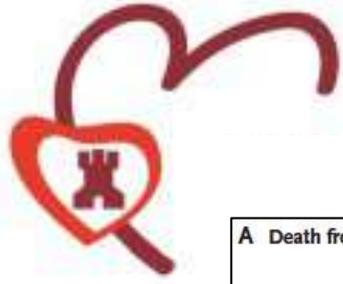
Figure 2. Kaplan–Meier Estimates of the Probability of Freedom from Death from Cardiovascular Causes.

N Engl J Med. 2017 May 18;376(20):1956-1964.

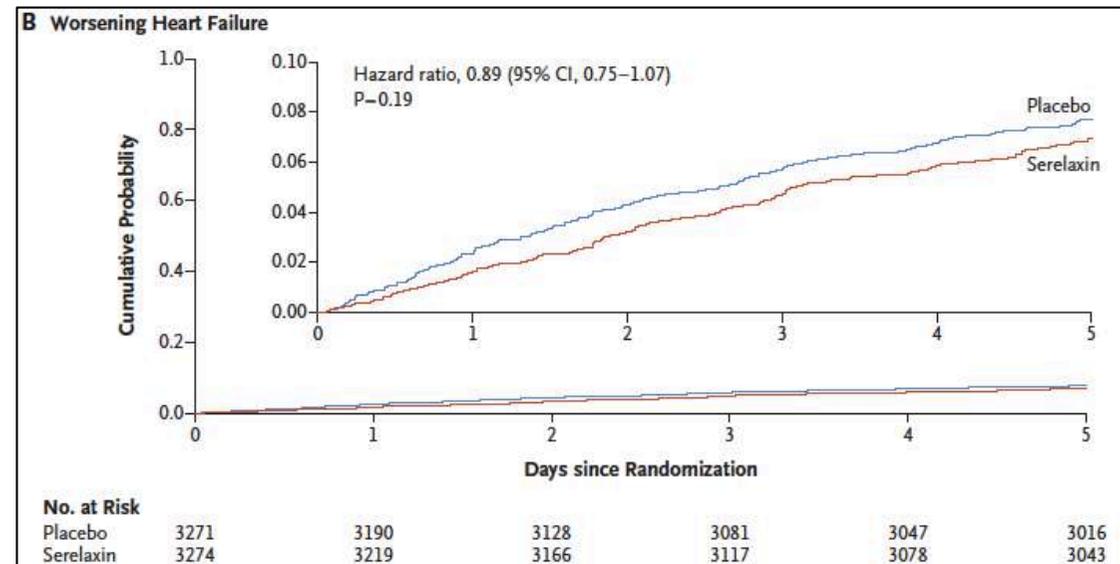
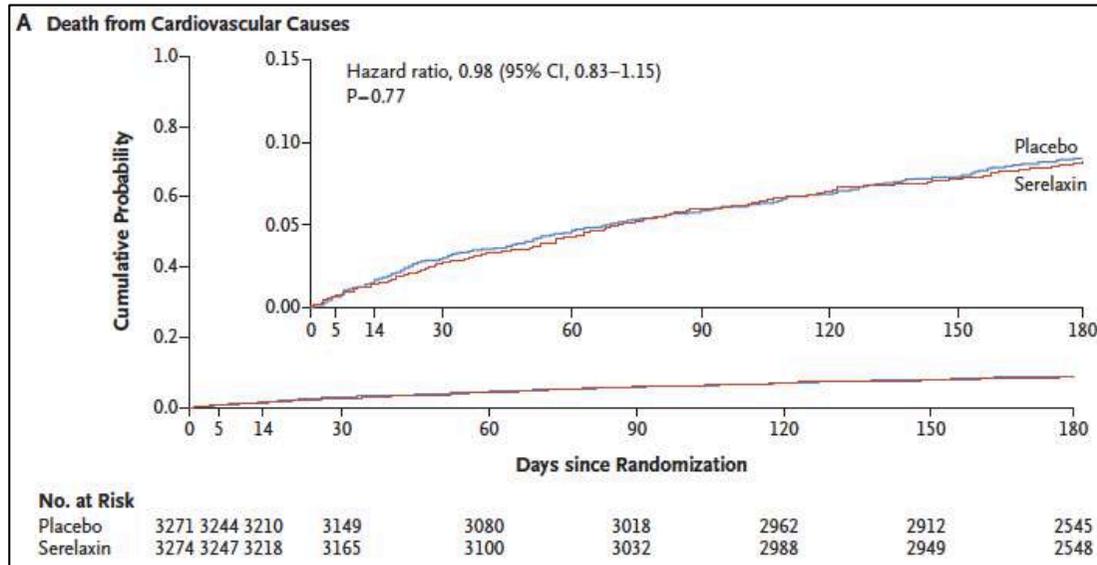
FLASHLIGHTS en



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CARDIACA**



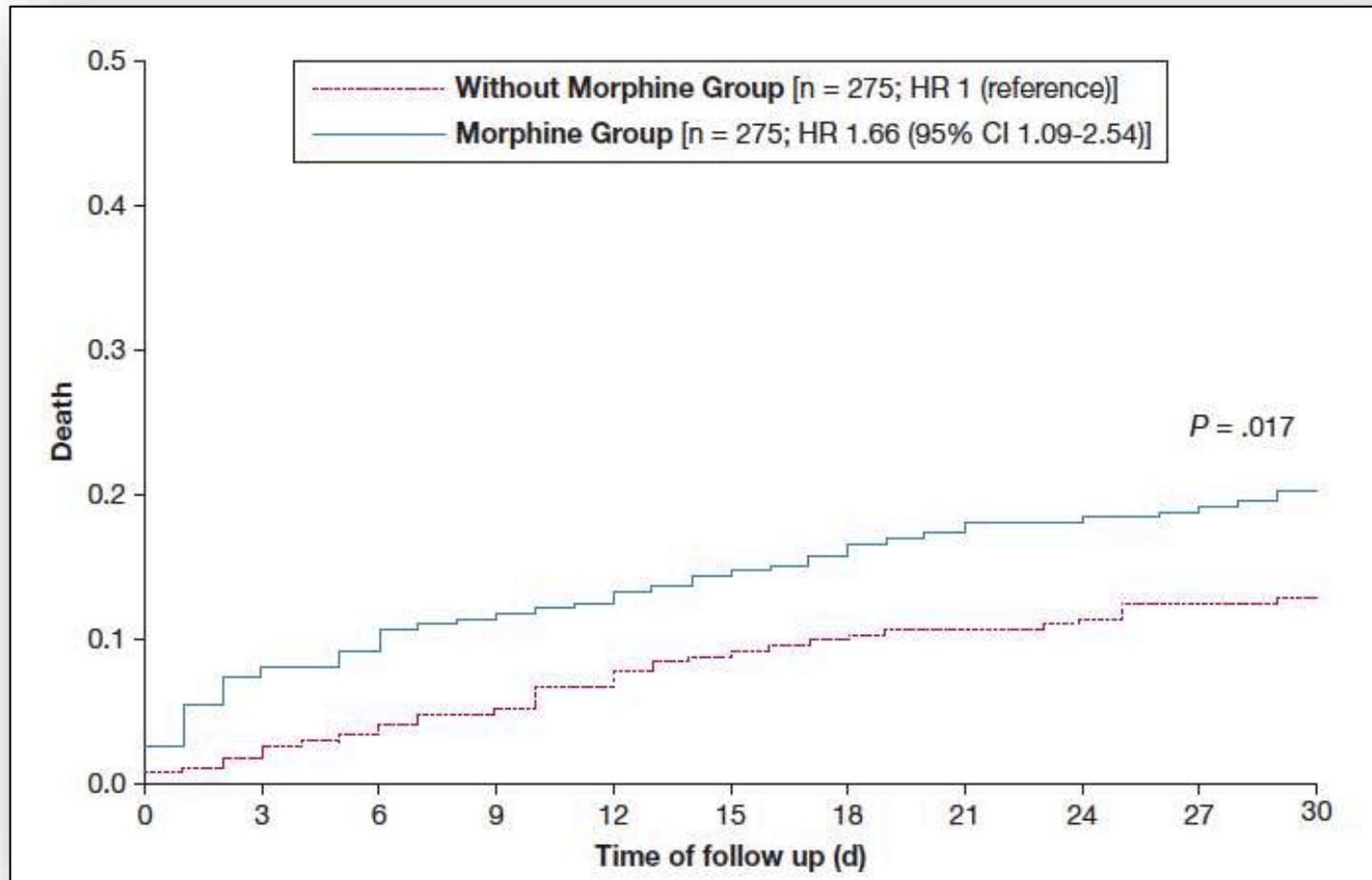
Vasodilatadores



N Engl J Med. 2019 Aug 22;381(8):716-726.



Vasodilatadores

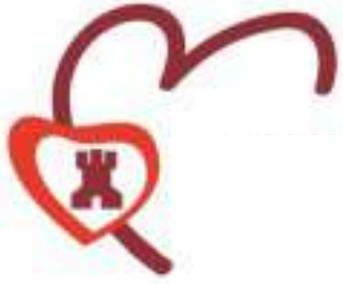


Chest. 2017 Oct;152(4):821-832.

FLASHLIGHTS en



**INSUFICIENCIA
CARDIACA**



Hipoperfusión tisular

- Inotrópicos
- Vasopresores
- Soporte circulatorio mecánico
- Trasplante cardiaco

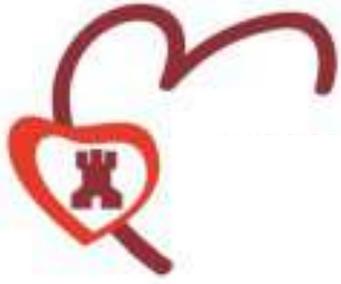


Eur Heart J. 2016 Jul 14;37(27):2129-2200.

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Inotrópicos

- Dobutamina, ~~dopamina~~, levosimendán, milrinona
- Indicados (IIb) si hipoperfusión tisular y/o hipotensión (TAS < 90 mmHg)
- Levosimendán o inhibidores PDE3 (IIb) si betabloqueantes



Vasopresores

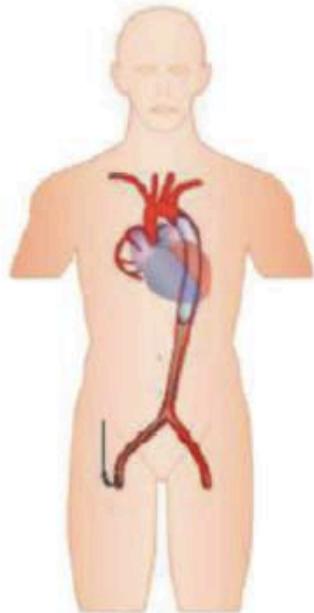
- Indicados en shock cardiogénico, especialmente si persiste hipotensión a pesar de tratamiento inotrópico
- Preferiblemente **noradrenalina** (estudio SOAP)
- ¿Adrenalina?, ¿dopamina?



Soporte circulatorio mecánico

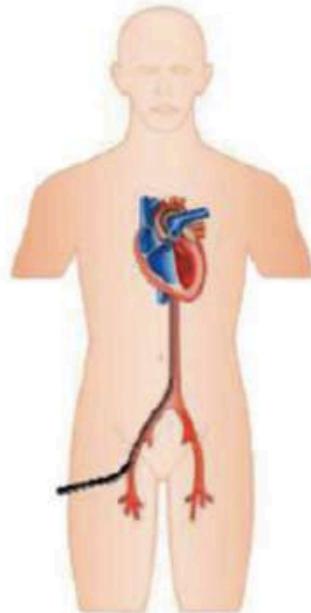
- Indicado en shock cardiogénico refractario

IABP

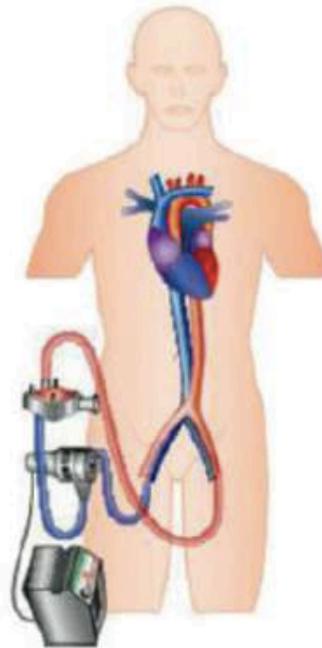


Impella

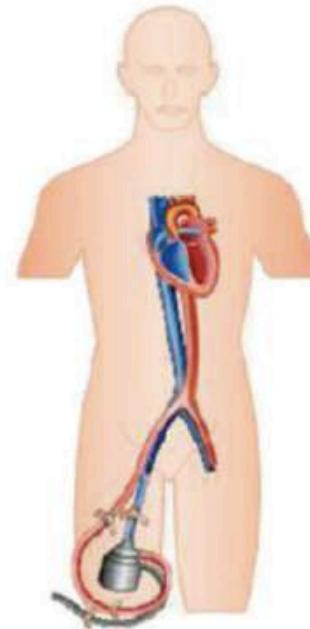
2,5
3,5
5,0



ECLS
(ECMO)



Tandem Heart



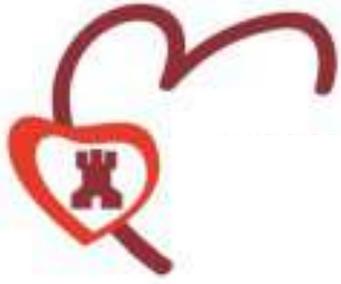
Levitronix
Centrimag



FLASHLIGHTS en

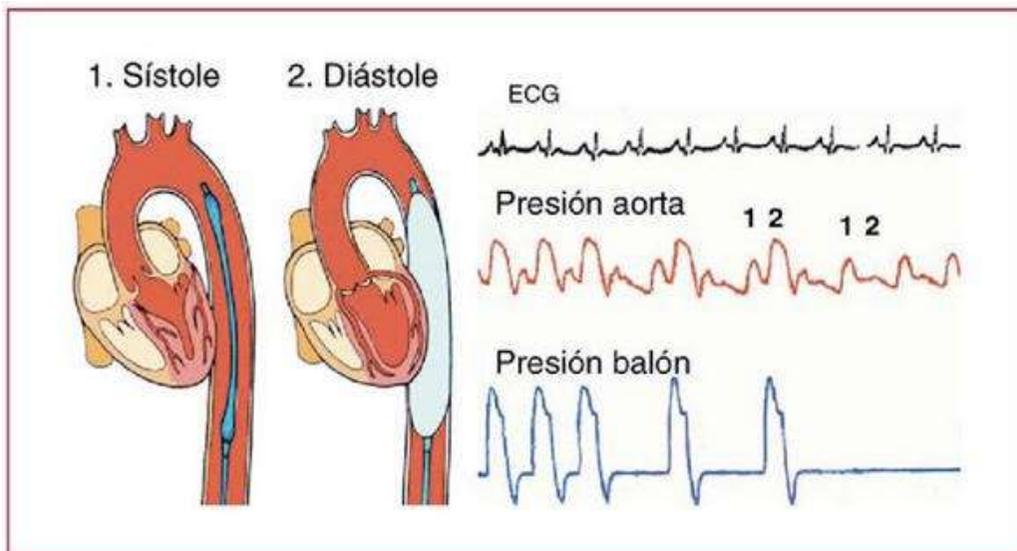


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Soporte circulatorio mecánico

- Balón de contrapulsación intraaórtico:
 - No de rutina (no beneficio en IAM, III A)
 - Medida de “estabilización” transitoria
 - Útil insuficiencia mitral aguda o CIV



FLASHLIGHTS en



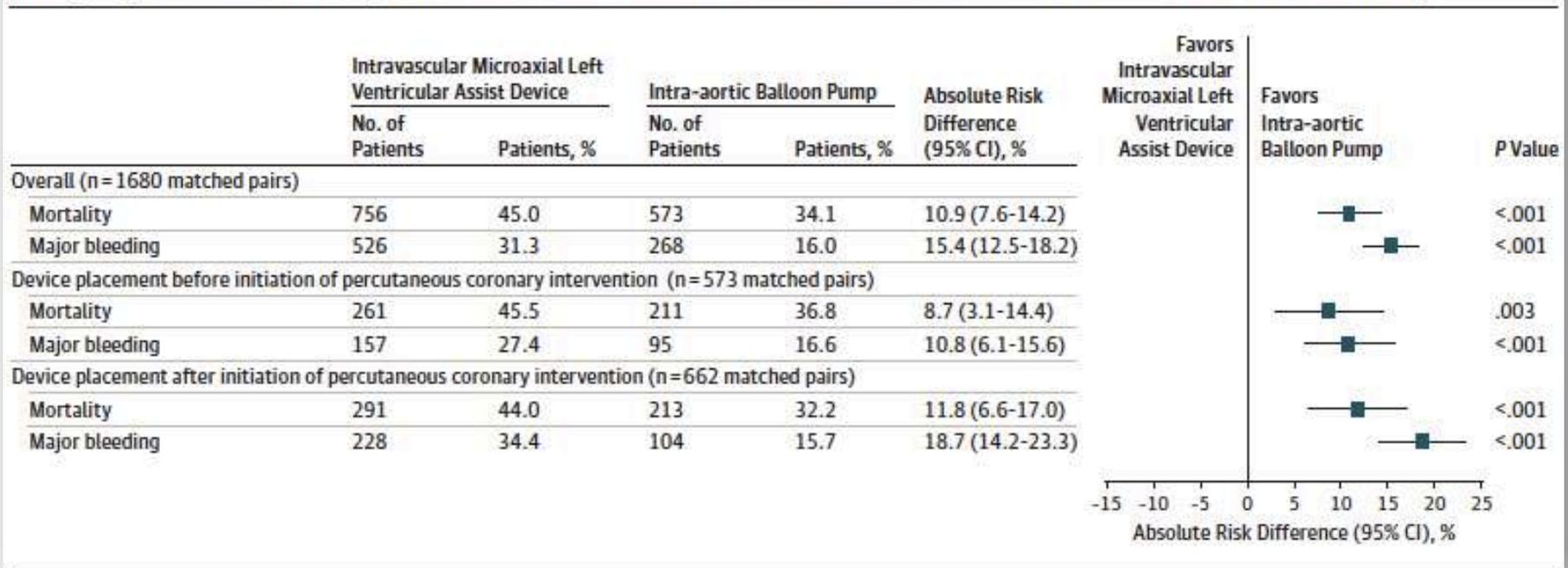
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Soporte circulatorio mecánico

- ¿Impella?

Figure 2. In-Hospital Outcomes Among Propensity-Matched Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock Undergoing Percutaneous Coronary Intervention With Intravascular Microaxial Left Ventricular Assist Device vs Intra-aortic Balloon Pump

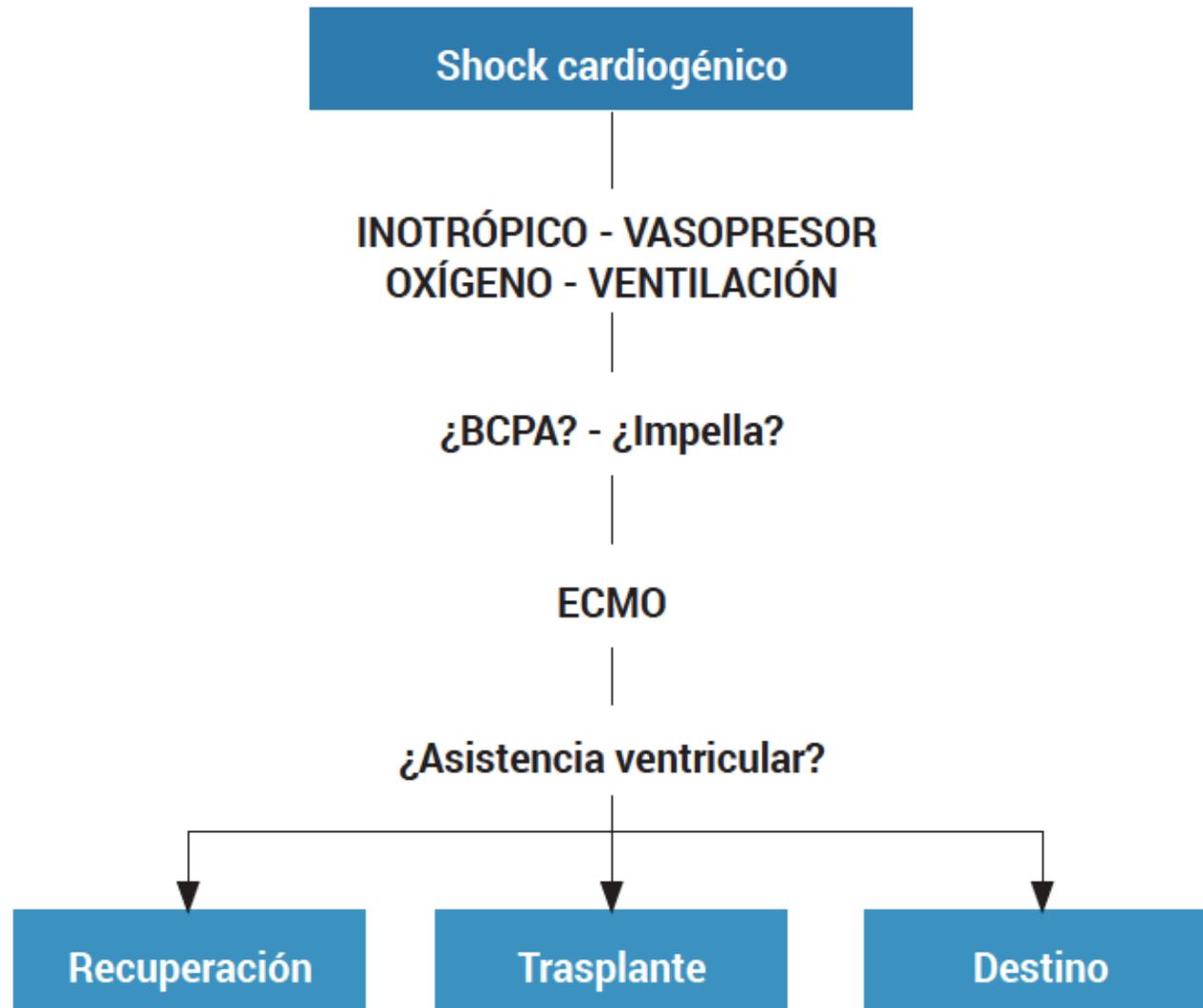


JAMA. 2020 Feb 10.

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Etiologías específicas ICA

Identification of acute aetiology:

- C** acute **C**oronary syndrome
- H** **H**ypertension emergency
- A** **A**rrhythmia
- M** acute **M**echanical cause^a
- P** **P**ulmonary embolism
- I** **I**nfection





Síndrome coronario agudo

- IAMCEST → ICP inmediata
- SCASEST → ICP < 24 h (< 2 h en casos graves)

SCA + SHOCK

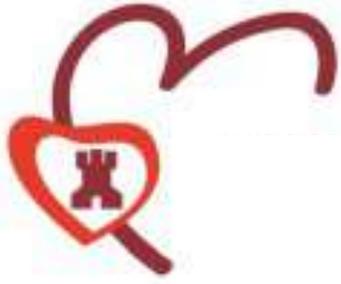


**Beneficio pronóstico de la
revascularización coronaria**

FLASHLIGHTS en

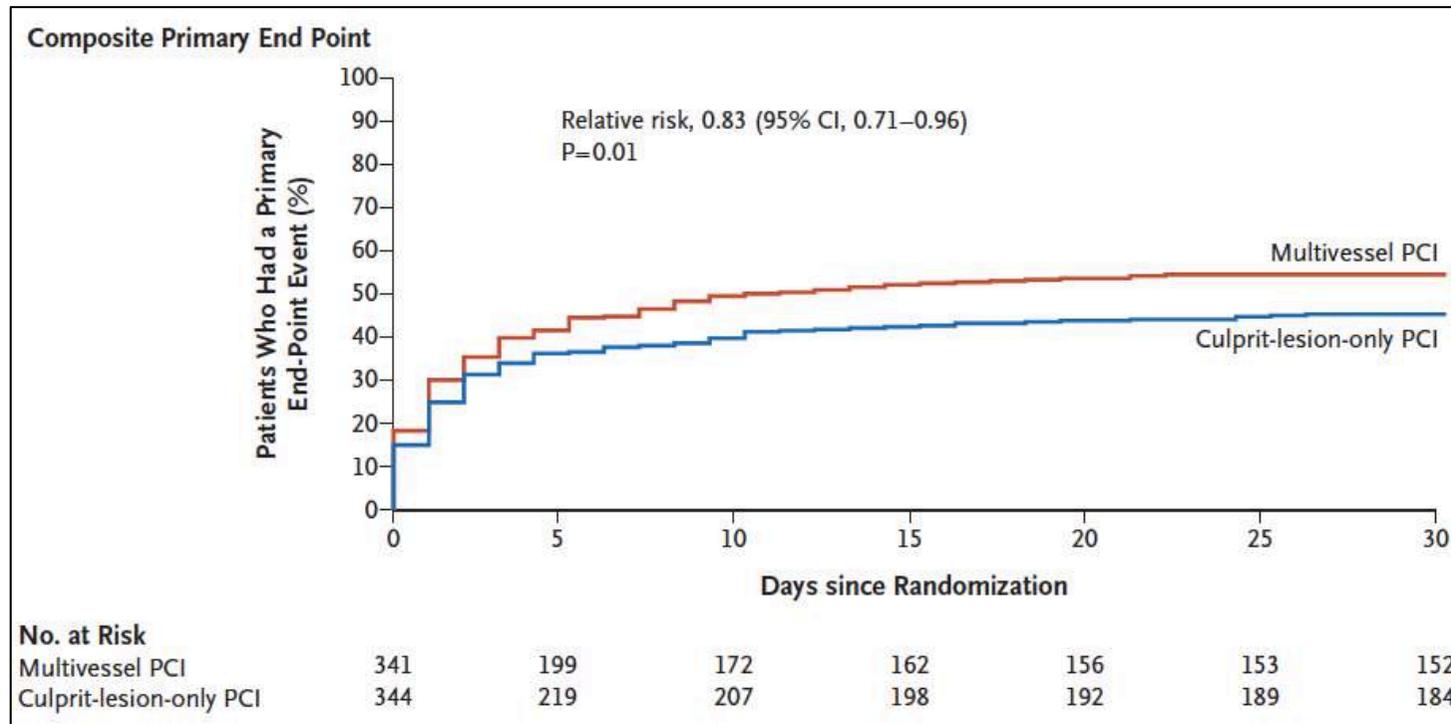


**INSUFICIENCIA
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SCA y shock cardiogénico

- Revascularización completa vs solo arteria culpable

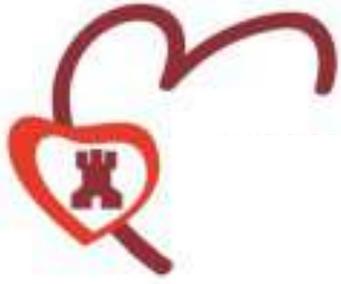


N Engl J Med. 2017 Dec 21;377(25):2419-2432.

FLASHLIGHTS en



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CARDIACA**



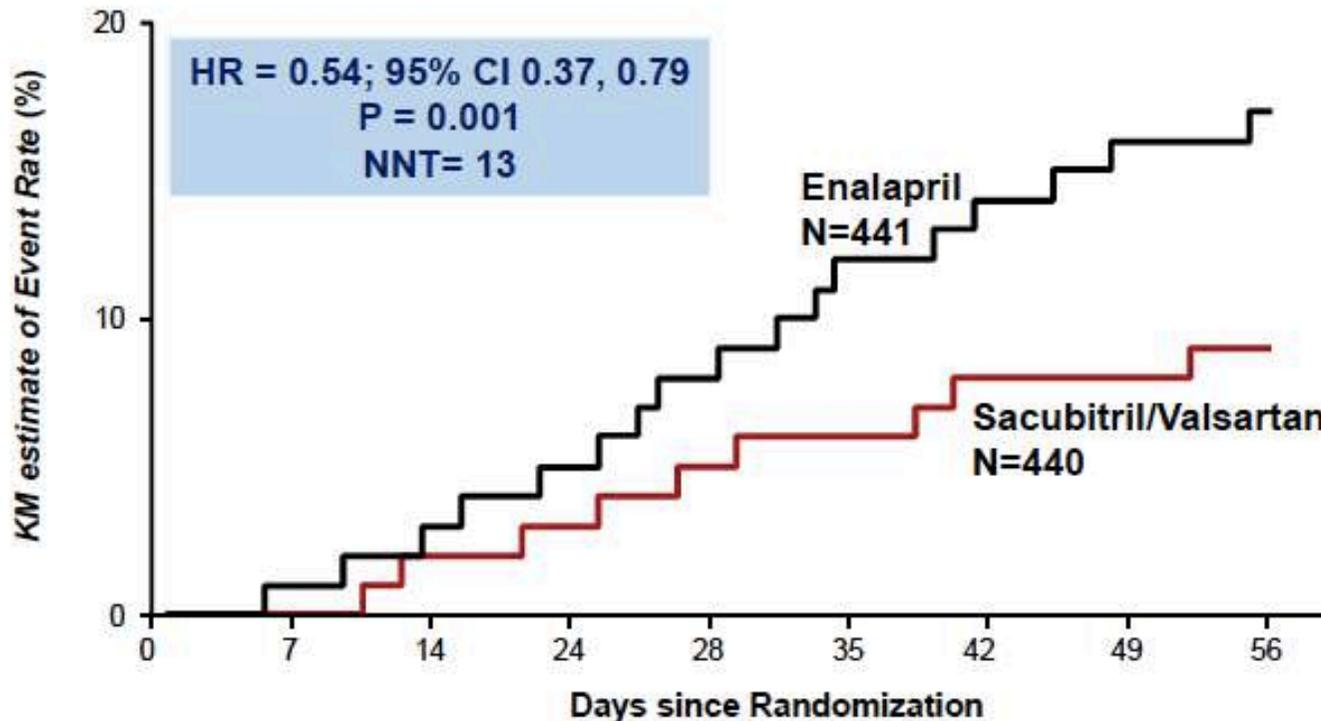
ICFE reducida: fármacos con beneficio pronóstico

- Mantener siempre que sea posible los fármacos modificadores de la enfermedad en los pacientes que ya los tomaban (incluidos β -bloquantes)
- Iniciarlos lo antes posible en IC *de novo*
 - Ivabradina en fase aguda puede ser útil y no afecta a la dosis final de β -bloqueante (ETHIC-AHF)



ICFE reducida: fármacos con beneficio pronóstico

Muerte, reingreso por IC, necesidad de asistencia ventricular izquierda o ser incluido en lista de espera de trasplante cardíaco

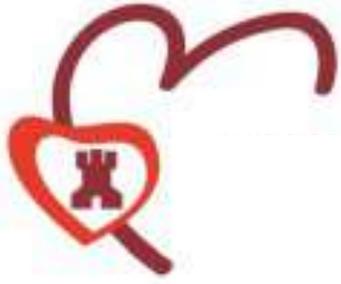


N Engl J Med. 2019 Feb 7;380(6):539-548.

FLASHLIGHTS en

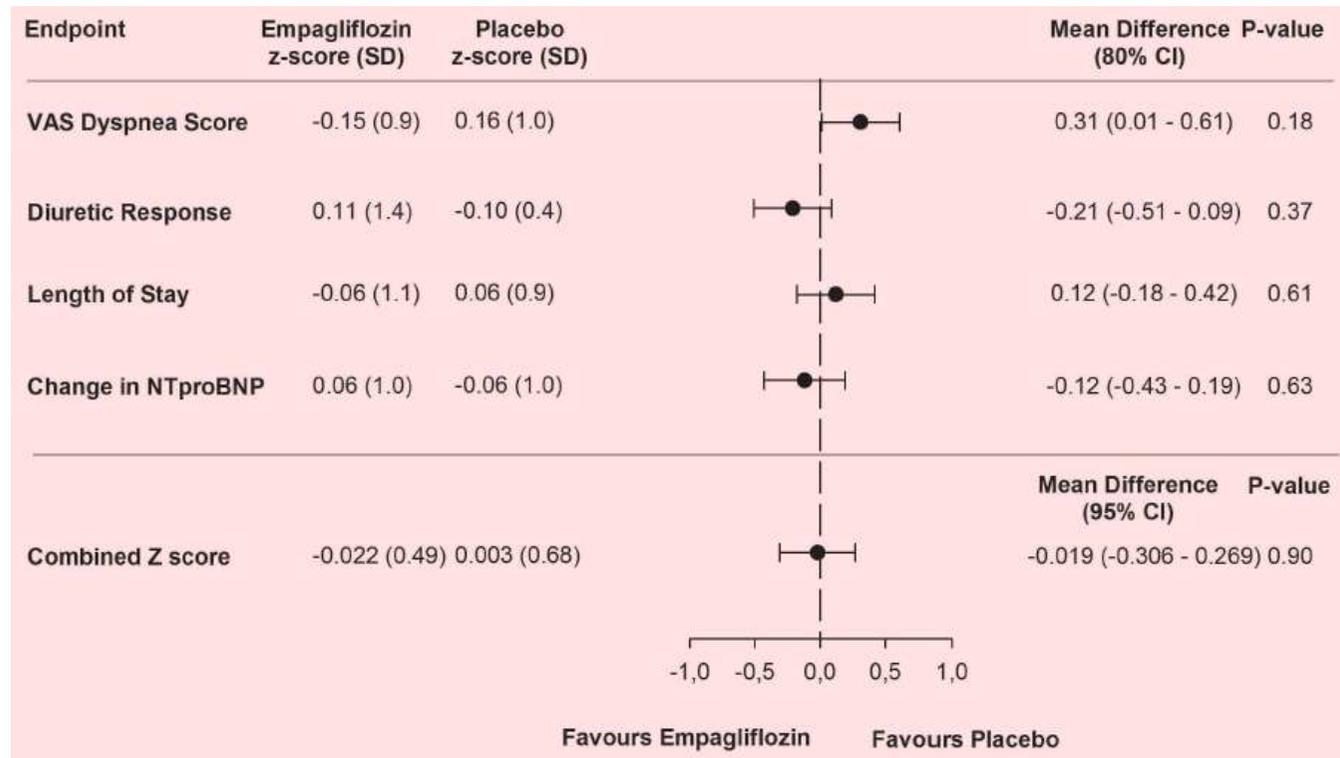


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ICFE reducida: fármacos con beneficio pronóstico

- EMPA-RESPONSE-AHF: empagliflozina vs placebo

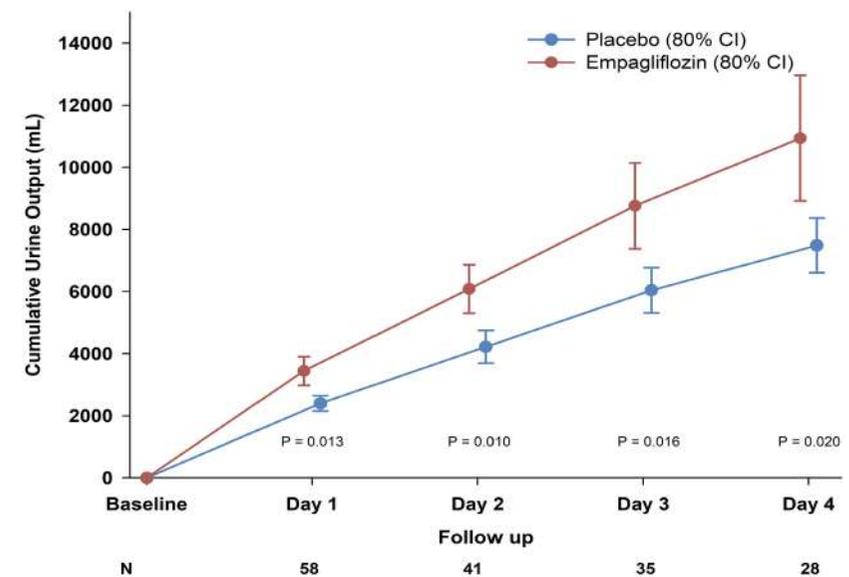
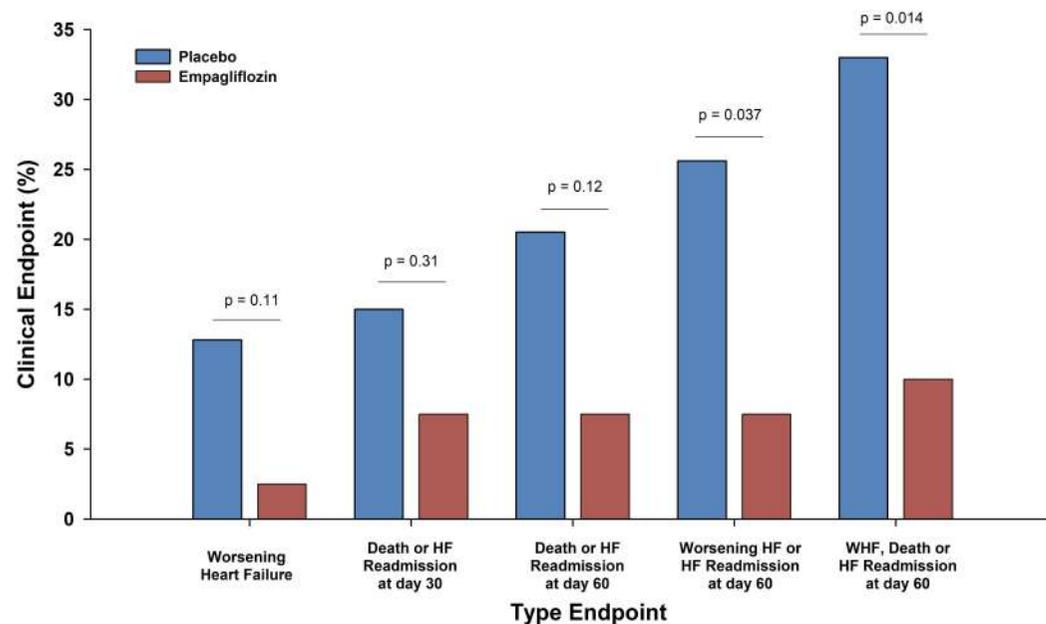


Eur J Heart Fail. 2020 Jan 7.

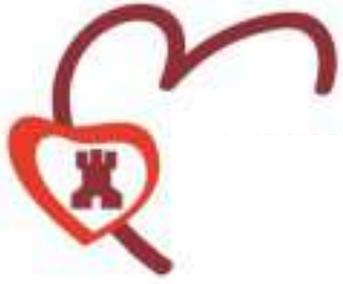


ICFE reducida: fármacos con beneficio pronóstico

- EMPA-RESPONSE-AHF: empagliflozina vs placebo



Eur J Heart Fail. 2020 Jan 7.



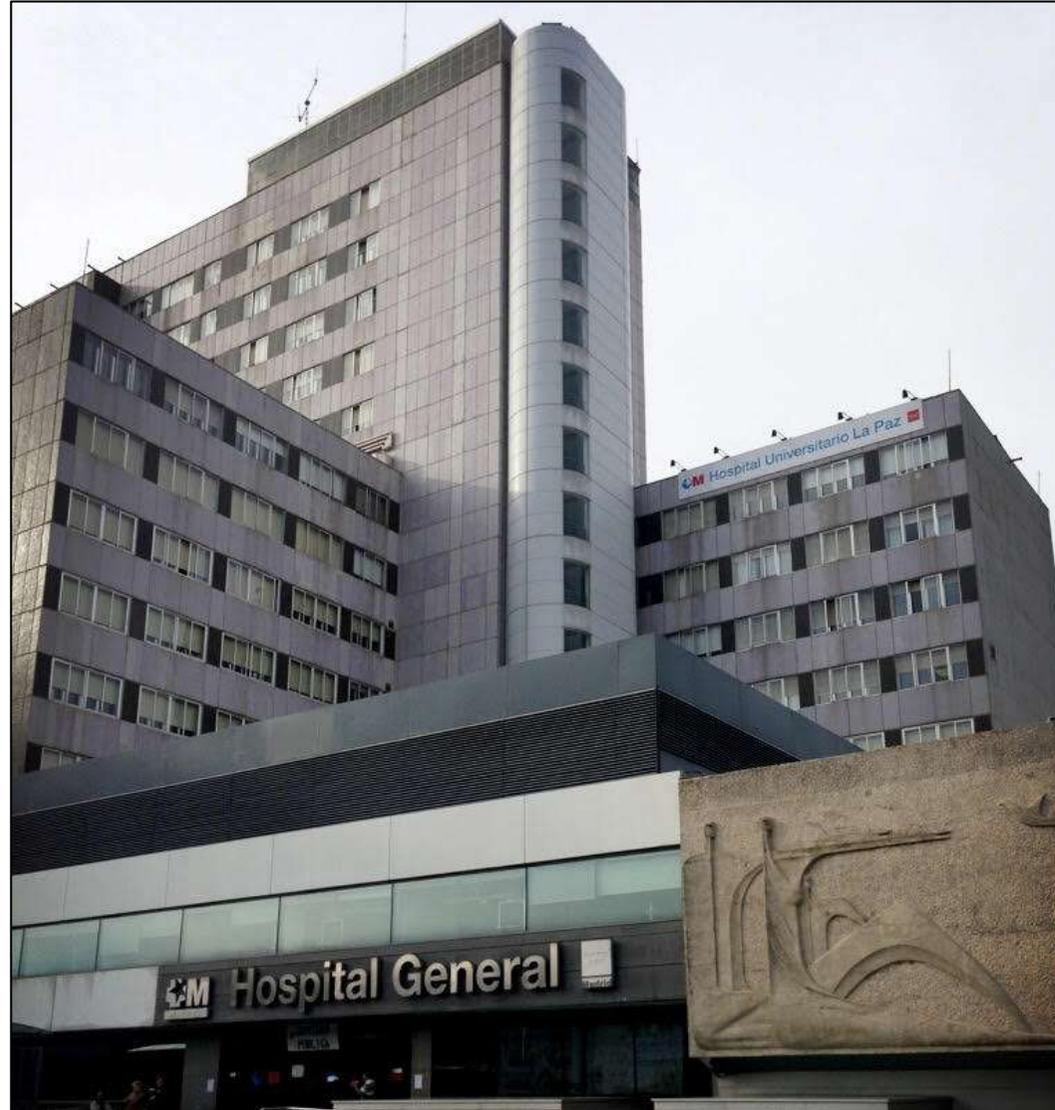
Conclusiones

- Los **objetivos** del tratamiento de la ICA son **eliminar la congestión** y **restaurar la perfusión** tisular
- Demostrar el beneficio de las intervenciones es muy difícil en ICA, por lo que estas se basan en la **experiencia** y el **consenso**
- El **tratamiento “crónico”** es una parte del tratamiento de la ICA

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MUCHAS GRACIAS

FLASHLIGHTS en



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SCA y shock cardiogénico

- 80% SCA, de estos 70% IAMEST
- ICP emergente → reduce mortalidad a 6 m

TABLE 4. MORTALITY AMONG STUDY PATIENTS.*

OUTCOME AND SUBGROUP	REVASCLARIZATION	MEDICAL THERAPY	DIFFERENCE BETWEEN GROUPS (95% CI)	RELATIVE RISK (95% CI)	P VALUE
	percent (number in subgroup)		percent		
30-day mortality					
Total	46.7 (152)	56.0 (150)	-9.3 (-20.5 to 1.9)	0.83 (0.67 to 1.04)	0.11
Age <75 yr	41.4 (128)	56.8 (118)	-15.4 (-27.8 to -3.0)	0.73 (0.56 to 0.95)	0.01†
Age ≥75 yr	75.0 (24)	53.1 (32)	+21.9 (-2.6 to 46.4)	1.41 (0.95 to 2.11)	
6-mo mortality‡					
Total	50.3 (151)	63.1 (149)	-12.8 (-23.2 to -0.9)	0.80 (0.65 to 0.98)	0.027
Age <75 yr	44.9 (127)	65.0 (117)	-20.1 (-31.6 to -7.1)	0.70 (0.56 to 0.89)	0.003†
Age ≥75 yr	79.2 (24)	56.3 (32)	+22.9 (0.7 to 46.6)	1.41 (0.97 to 2.03)	

N Engl J Med. 1999 Aug 26;341(9):625-34.

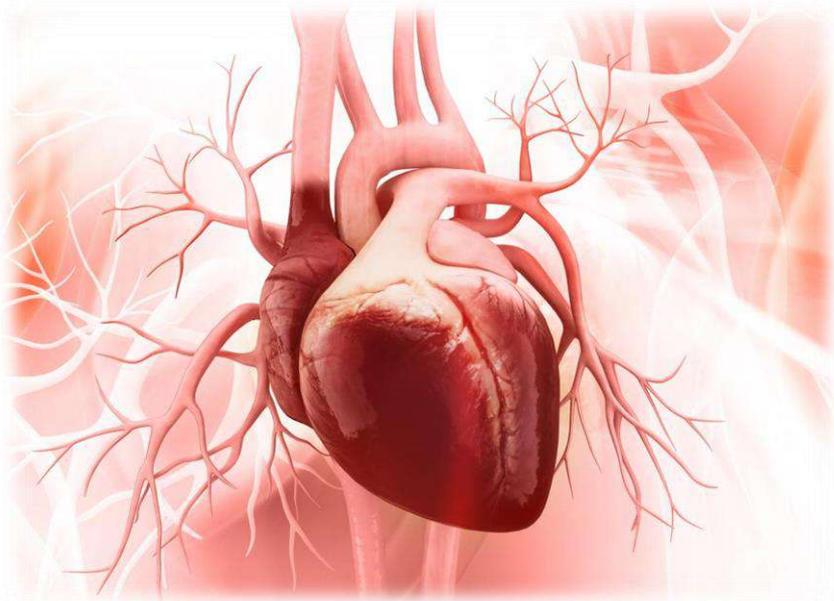
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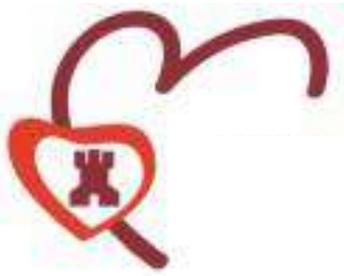
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Cuándo y cómo buscar la etiología en la miocardiopatía dilatada no isquémica



Fernando Domínguez. MD, PhD
Hospital U. Puerta de Hierro Majadahonda



MIOCARDIOPATÍA DILATADA no isquémica

Introducción

- ✓ **Prevalencia:** 35.5/100000 (Olmsted County, Minnesota 1975-1984)
 - Probablemente infraestimada: Hasta 14% de pacientes asintomáticos (Devereux 2001)
 - 1/250-500 ?¹

3^a causa más frecuente de IC tras cardiopatía isquémica e HTA

Diagnóstico más frecuente en pacientes sometidos a trasplante cardíaco²

✓ Pronóstico:

1994: 25% mortalidad anual, 50% en 5 años (*Fuster et al. NEJM*)

2020: Importante impacto de terapias actuales

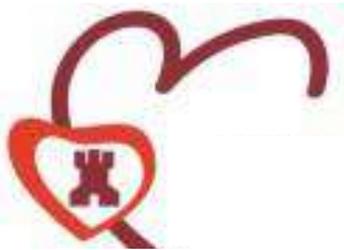
- ✓ 87% de pacientes libres de trasplante cardíaco a los 8 años³.

FLASHLIGHTS en



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CARDIACA**

1. Hershberger RE et al. *Nar Rev Cardiol.* 2013; 10: 531-547
2. Lund LH et al. *J. Heart Lung Transplant.* 2014;33:996–1008
3. Merlo M et al. *Eur J Heart Fail* 2014;16:317–324.



MIOCARDIOPATÍA DILATADA no isquémica

Introducción

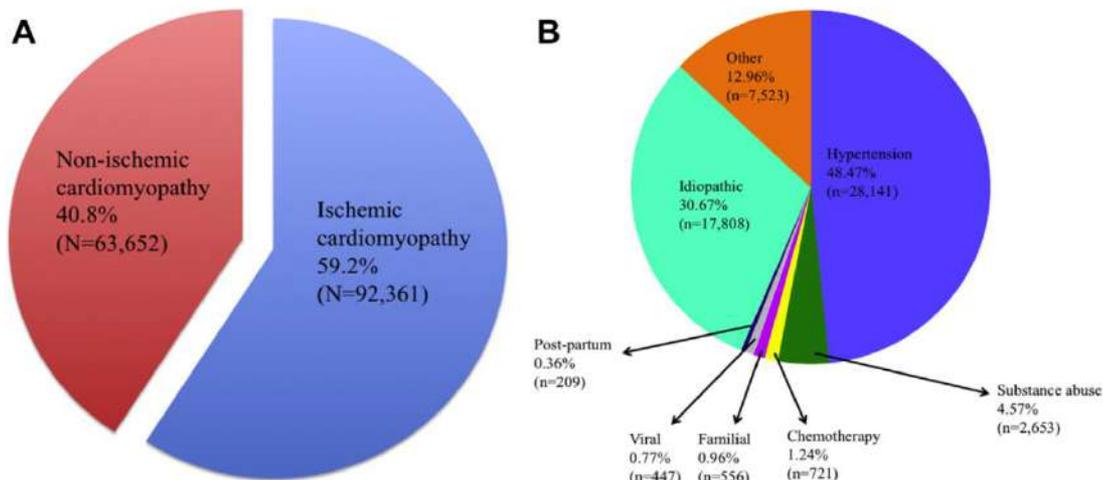
Characteristics, Treatments, and Outcomes of Hospitalized Heart Failure Patients Stratified by Etiologies of Cardiomyopathy

30.6% de casos con causa desconocida!!

Supriya Shore, MD, MSCS,* Maria V. Grau-Sepulveda, MD, MPH,† Deepak L. Bhatt, MD, MPH,‡
Paul A. Heidenreich, MD, MS,§ Zubin J. Eapen, MD, MHS,‡ Adrian F. Hernandez, MD, MHS,‡ Clyde W. Yancy, MD,‡
Gregg C. Fonarow, MD¶

156000 pacientes ingresados por IC en EEUU (2005-2013)

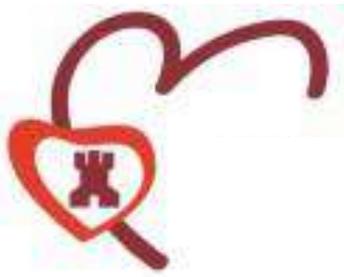
FIGURE 2 Prevalence of Ischemic and Nonischemic Cardiomyopathy and Various Nonischemic Etiologies



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MIOCARDIOPATÍA DILATADA no isquémica

ETIOLOGÍA

- ✓ **Muy heterogénea**
- ✓ **Dilatación y disfunción ventricular:**
 - Fenotipo muy frecuente
 - Estadío final de otras miocardiopatías.

40-70%¹

**FAMILIAR
GENÉTICO**



30-50% (incluyendo “idiopática”²)

**NO FAMILIAR
NO GENÉTICO**



HOUSE

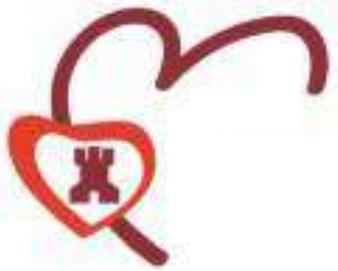
FLASHLIGHTS en



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1. Cuenca S et al. *J. Heart Lung Transplant.* 2016;35:625–635.

2. Shore S et al. *JACC Heart Fail.* 2015;3:906–916.



MIOCARDIOPATÍA DILATADA no isquémica

ETIOLOGÍA

Dilated cardiomyopathy

Genetic

Cardiac phenotype:

- titin (TTN)
- lamin A/C (LMNA)
- myosin heavy chain (MYH7)
- troponin T (TNT2)
- Myosin binding protein C (MYBPC3)
- phospholamban (PLN)

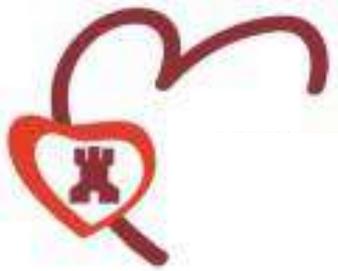
Neuromuscular disorders:

- Duchenne muscular dystrophy
- Becker muscular dystrophy

FLASHLIGHTS en

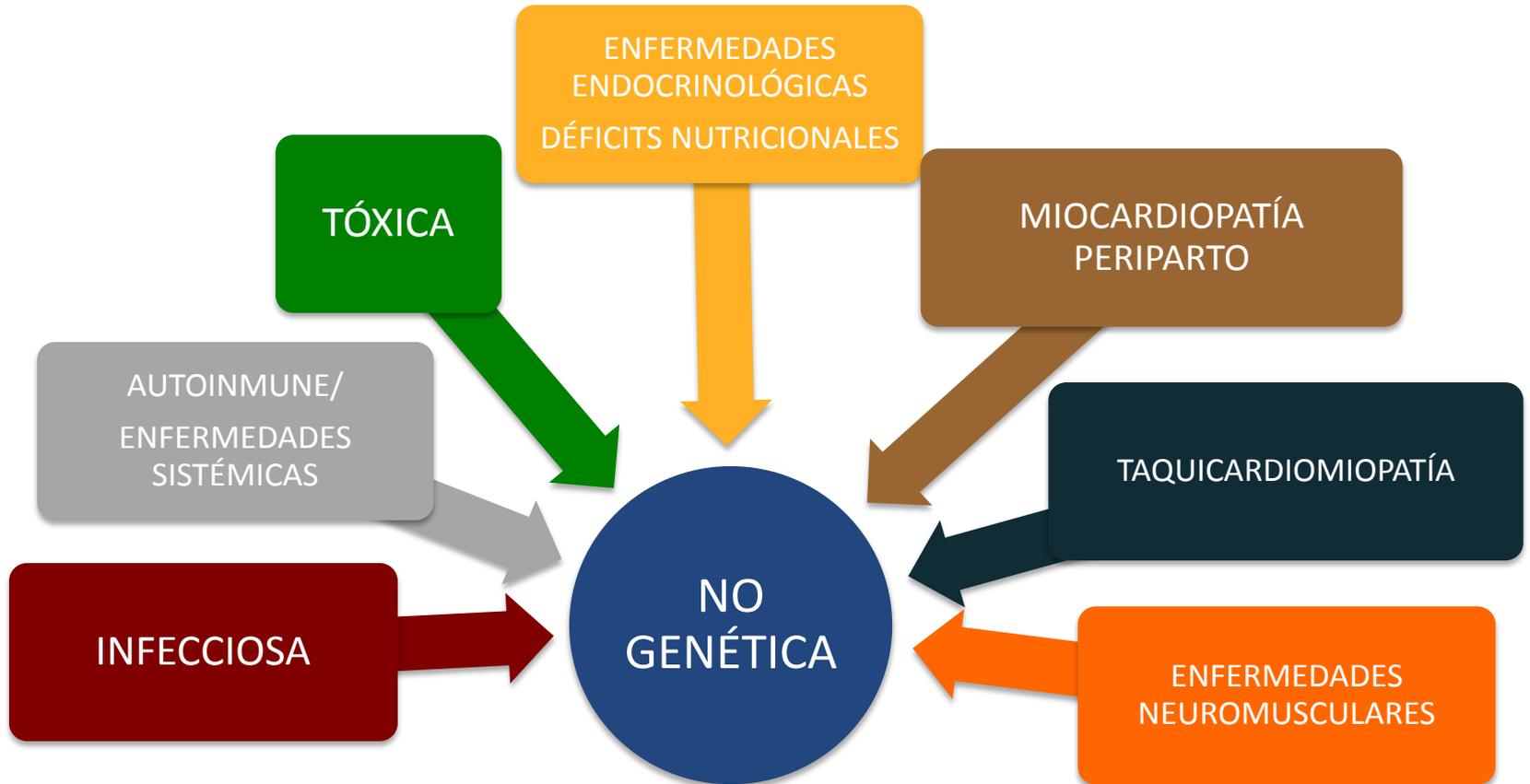


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MIOCARDIOPATÍA DILATADA no isquémica

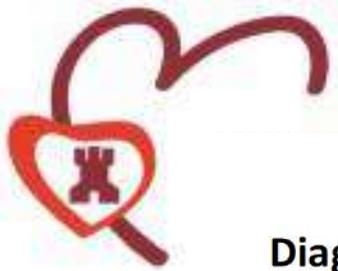
ETIOLOGÍA



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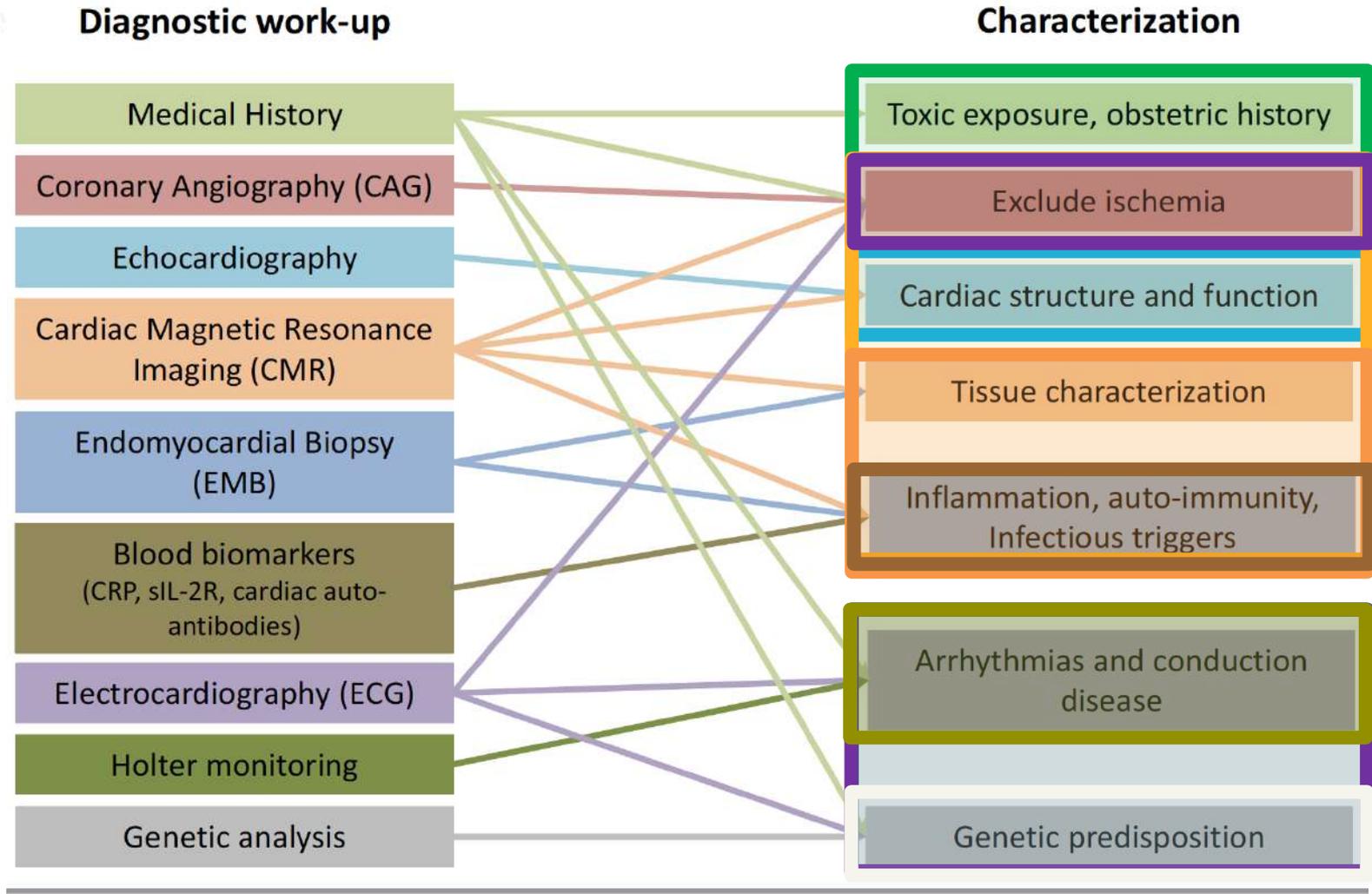


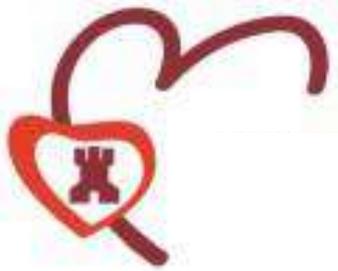
**INSUFICIENCIA
CARDIACA**



MIOCARDIOPATÍA DILATADA no isquémica

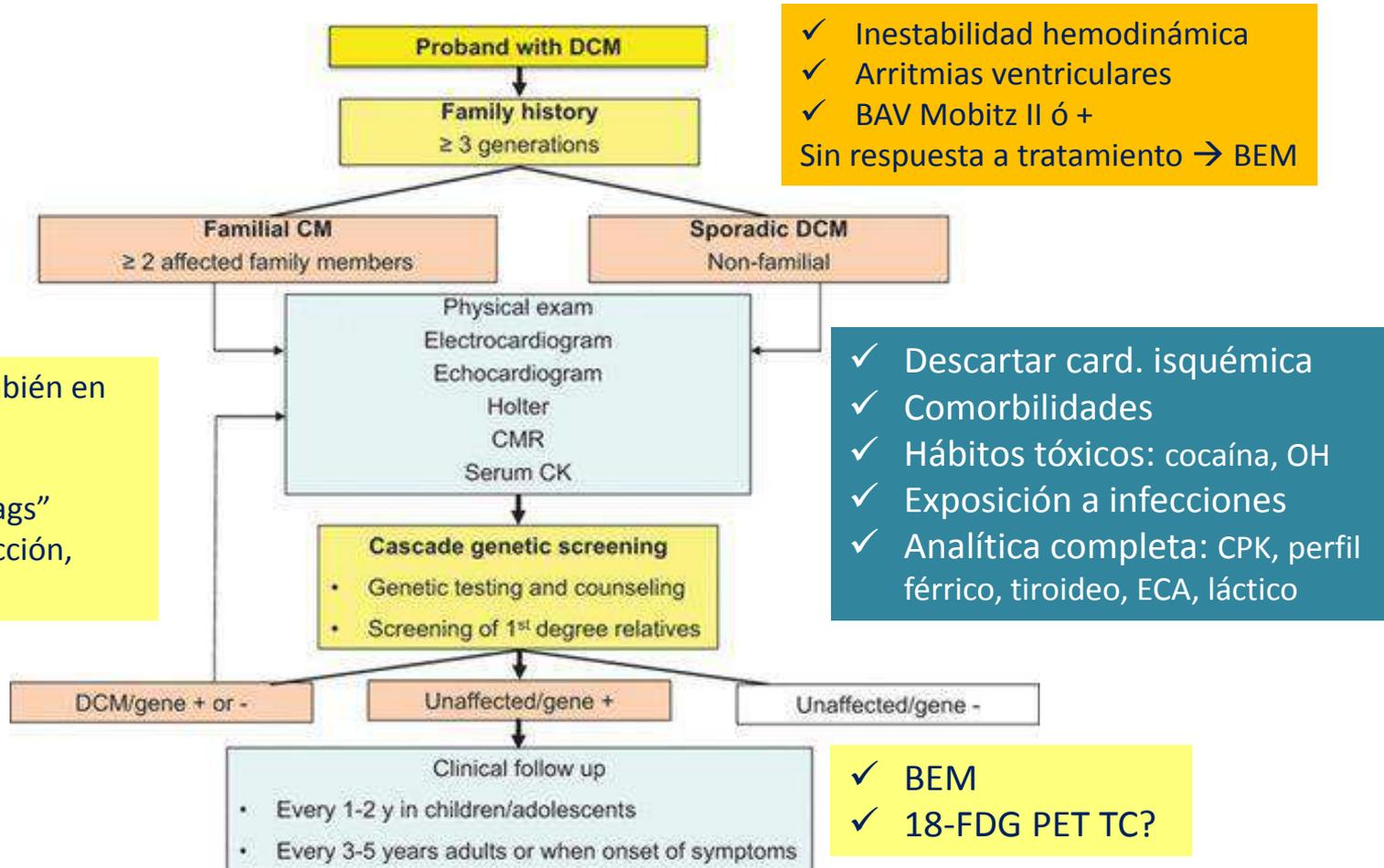
Herramientas diagnósticas

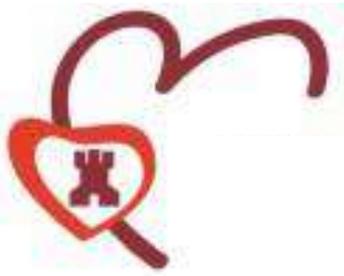




MIOCARDIOPATÍA DILATADA no isquémica

Cuándo y cómo buscar etiología



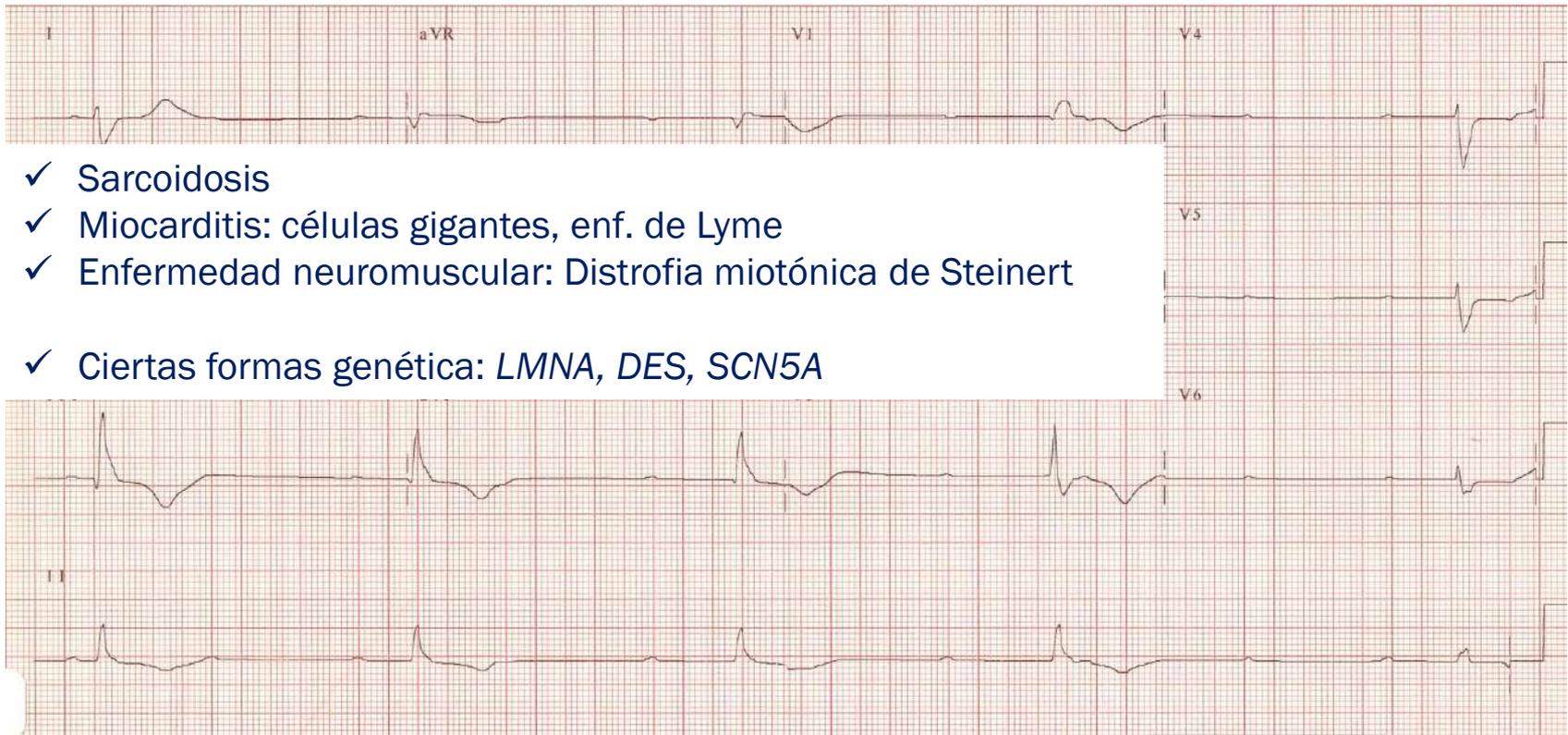


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Herramientas diagnósticas



MCD + bloqueo AV

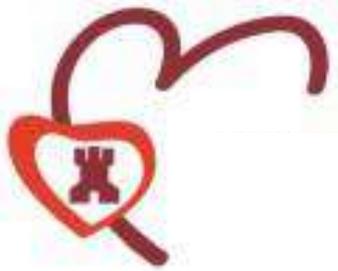


- ✓ Sarcoidosis
- ✓ Miocarditis: células gigantes, enf. de Lyme
- ✓ Enfermedad neuromuscular: Distrofia miotónica de Steinert
- ✓ Ciertas formas genética: *LMNA*, *DES*, *SCN5A*

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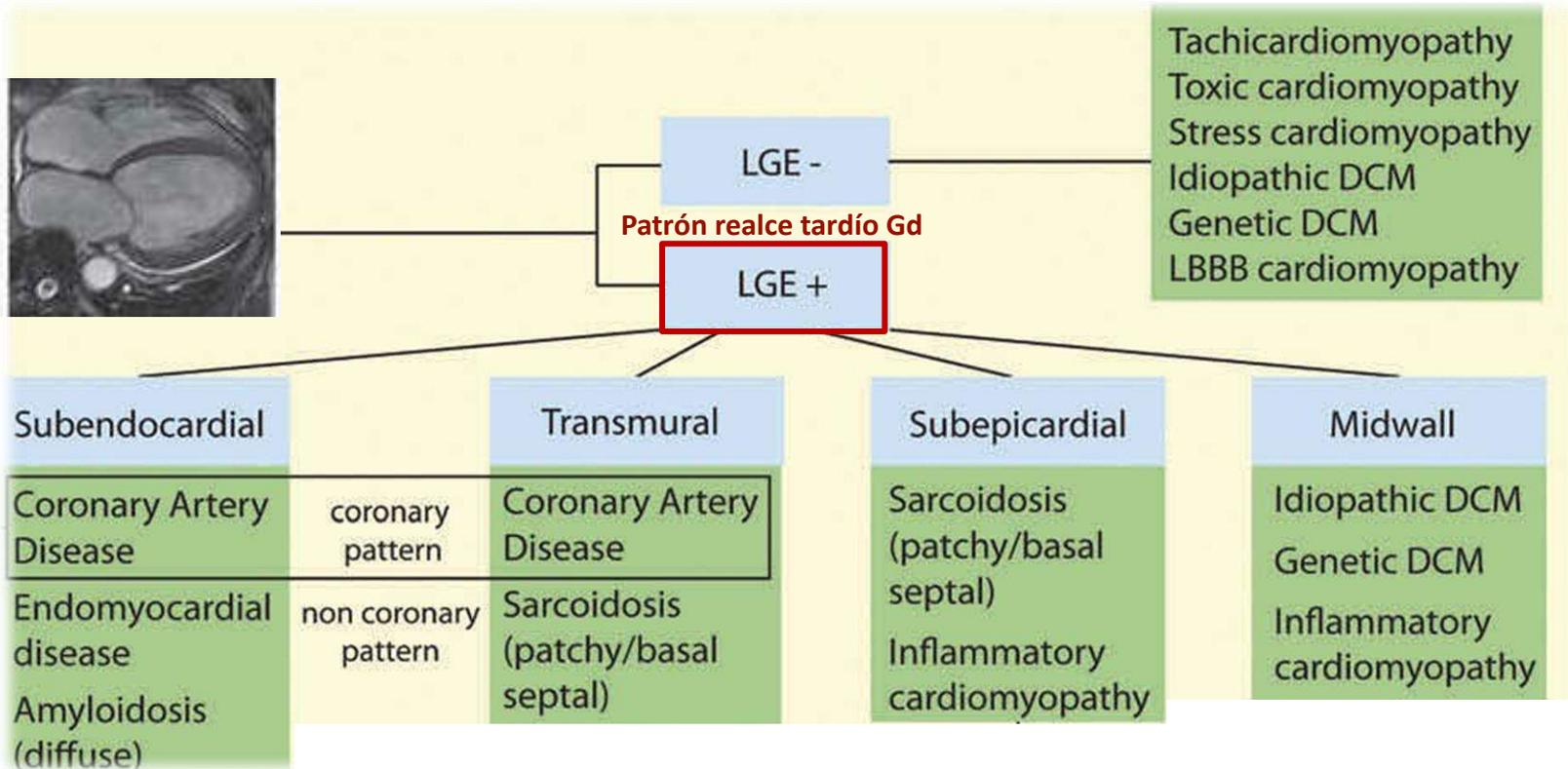


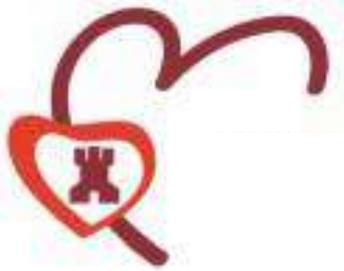
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Herramientas diagnósticas: Cardio RMN





MIOCARDIOPATÍA DILATADA no isquémica

Herramientas diagnósticas - Genética

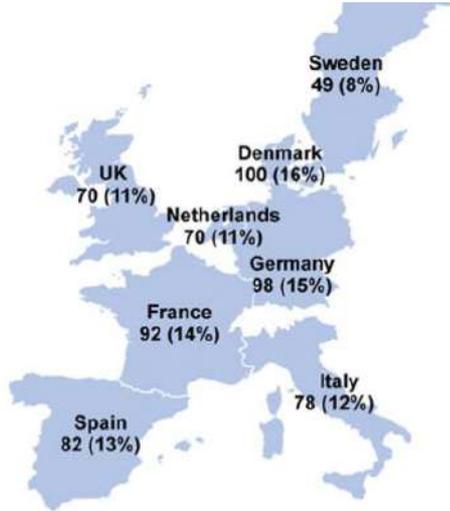
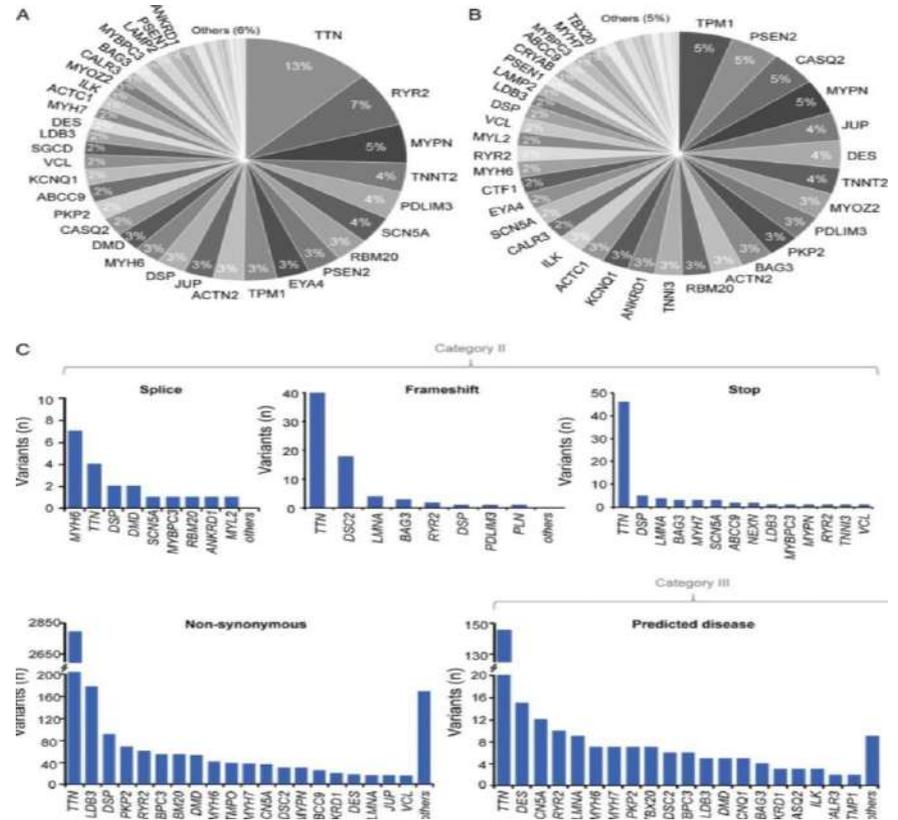


Table 2 Multiple mutations affecting single patients

Number of mutations	HGMD ^a variant pos patients (%)	Category Ib–III ^b variant pos patients (%)
0	245 (54.0)	171 (26.7)
≥ 1	294 (46.0)	468 (73.2)
≥ 2	82 (12.8)	243 (38.0)
≥ 3	14 (2.2)	82 (12.8)
≥ 4	2 (0.3)	16 (2.5)

^aCategory Ib.

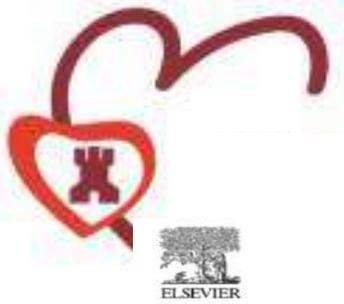
^bEither category Ib or category II or category III.



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Genética

The Journal of
Heart and Lung
Transplantation
<http://www.jhltonline.org>

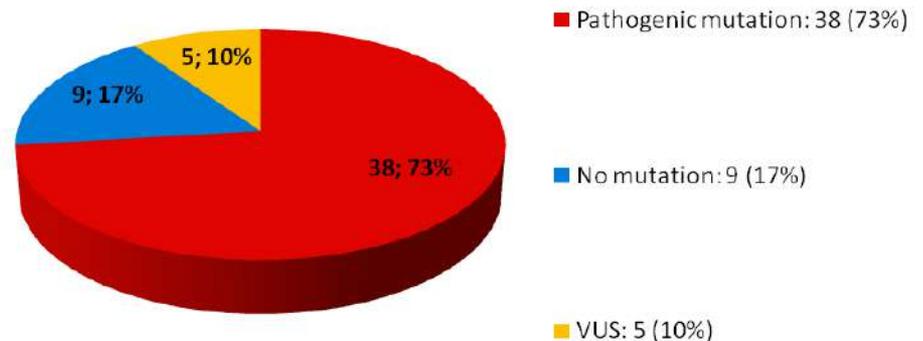


Genetic basis of familial dilated cardiomyopathy patients undergoing heart transplantation

Sofia Cuenca, MD,^a María J. Ruiz-Cano, MD, PhD,^b
Juan Ramón Gimeno-Blanes, MD, PhD,^c Alfonso Jurado, MD,^b Clara Salas, MD,^d
Iria Gomez-Diaz, BSc,^e Laura Padron-Barthe, PhD,^f Jose Javier Grillo, MD,^g
Carlos Vilches, MD, PhD,^h Javier Segovia, MD, PhD,^a
Domingo Pascual-Figal, MD, PhD,^c Enrique Lara-Pezzi, PhD,^f
Lorenzo Monserrat, MD, PhD,^e Luis Alonso-Pulpon, MD, PhD,^a
Pablo Garcia-Pavia, MD, PhD^{a,f} and for the Inherited Cardiac Diseases
Program of the Spanish Cardiovascular Research Network
(Red Investigación Cardiovascular)

- 52 pacientes con MCD y TxC
- Evaluación de 220 familiares
- ✓ N= 21 con mutaciones patogénicas
- ✓ N= 17 con VSI reclasificadas a patogénicas tras estudio familiar

Final genetic results

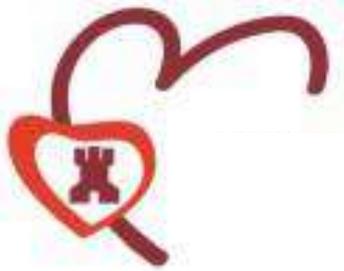


MCD genética: 73% de los pacientes sometidos a TxC

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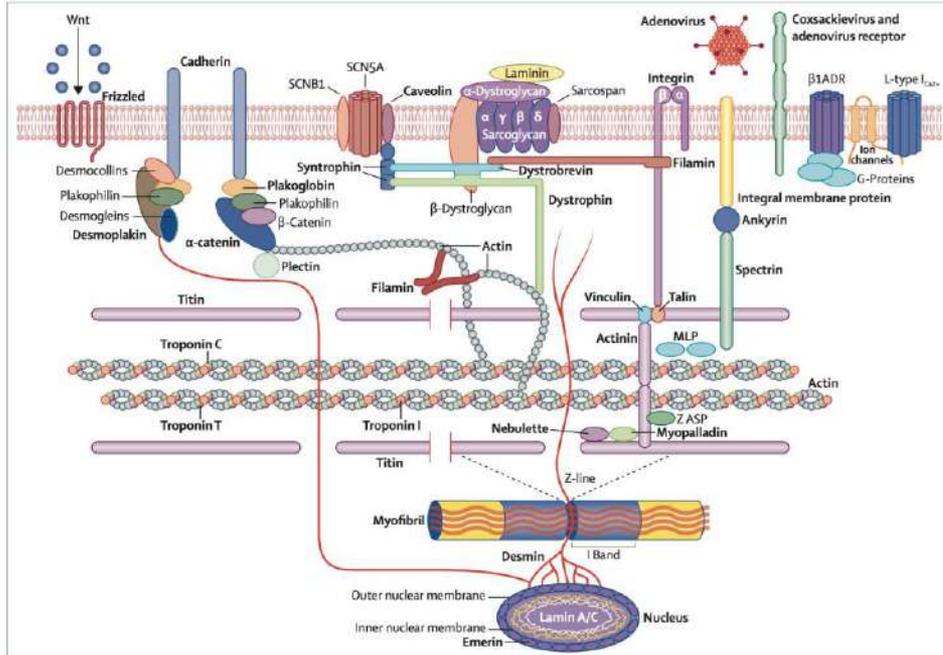


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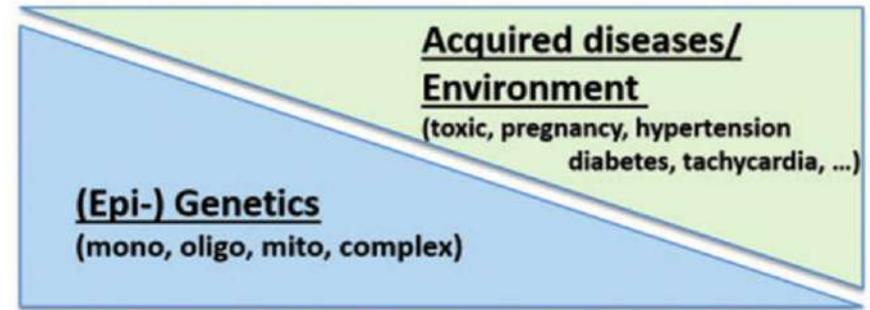


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Genética vs. ambiente



DCM phenotype: gene-environmental interaction



e.g. Lamin A/C
Phospholamban
RBM-20

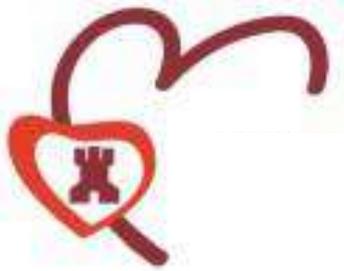
e.g. MyH7

e.g. Titin tv

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Genética- Truncamientos en TTN – Teoría del “2º hit”

- Miocardiopatía periparto(NEJM, 2016):
- 15% de TTNtv vs. 4.7% in población general

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
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VOL. 71, NO. 20, 2018

Genetic Etiology for Alcohol-Induced Cardiac Toxicity



James S. Ware, MRCP, PhD,^{a,b,c,d,e} Almudena Amor-Salamanca, MD,^{d,e} Upasana Tayal, MRCP, PhD,^{a,b,c} Risha Govind, MSc,^{a,b,c,d,e} Isabel Serrano, MD,^f Joel Salazar-Mendiguchía, MD,^{g,h} Jose Manuel Garcia-Pinilla, MD, PhD,^{i,j} Domingo A. Pascual-Figal, MD, PhD,^{k,l} Julio Nuñez, MD, PhD,^{i,j} Gonzalo Guzzo-Merello, MD, PhD,^l Emiliano Gonzalez-Vloque, PhD,^m Alfredo Bardaji, MD, PhD,^f Nicola Manito, MD, PhD,^g Miguel A. López-Garrido, MD,^h Laura Padron-Barthe, PhD,^{h,i} Elizabeth Edwards, PhD,^h Nicola Whiffin, PhD,^{h,i} Roddy Walsh, MSc, PhD,^{h,i} Rachel J. Buchan, MSc,^{h,i} William Midwinter, BSc,^{h,i} Alicja Wilk, BSc,^{h,i} Sanjay Prasad, MD,^{h,i} Antonis Pantazis, MD,^h John Baski, MRCP, PhD,^h Declan P. O'Regan, MRCP, PhD,^h Luis Alonso-Pulpon, MD, PhD,^{h,i} Stuart A. Cook, MRCP, PhD,^{h,i,j,k,l} Enrique Lara-Pezzi, PhD,^{h,i} Paul J. Barton, PhD,^{h,i} Pablo Garcia-Pavia, MD, PhD,^{h,i,j,k}

10% de pacientes con MCD enólica presenta truncamientos en TTN

MCD +TTNtv + alcohol excesivo

→ Más proclives a empeorar la FEVI que los bebedores moderados o aquellos sin variantes genéticas.

THE NEW ENGLAND JOURNAL OF MEDICINE

ORIGINAL ARTICLE

Shared Genetic Predisposition in Peripartum and Dilated Cardiomyopathies

James S. Ware, Ph.D., M.R.C.P., Jian Li, Ph.D., Erica Mazaika, M.A., Christopher M. Yasso, B.S., Tiffany DeSouza, B.S., Thomas P. Cappola, M.D., Emily J. Tsai, M.D., Denise Hilfiker-Kleiner, Ph.D., Chizuko A. Kamiya, M.D., Ph.D., Francesco Mazzarotto, M.Sc., Stuart A. Cook, M.D., Ph.D., Indrani Halder, Ph.D.,

Circulation

ORIGINAL RESEARCH ARTICLE



Genetic Variants Associated With Cancer Therapy-Induced Cardiomyopathy

BACKGROUND: Cancer therapy-induced cardiomyopathy (CCM) is associated with cumulative drug exposures and preexisting cardiovascular disorders. These parameters incompletely account for substantial interindividual susceptibility to CCM. We hypothesized that rare variants in cardiomyopathy genes contribute to CCM.

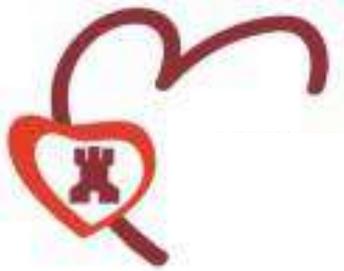
Pablo Garcia-Pavia, MD, PhD*
 Yuri Kim, MD, PhD*
 Maria Alejandra Restrepo-Cordova, MD*

7.5% of patients with MCD por QT presentan truncamientos en TTN

(vs. 1.1% en población con cáncer de referencia)

MCD por QT +TTNtv

→ Más FA, IC y no recuperación de la FEVI vs. MCD por QT sin variantes de truncamiento en TTN



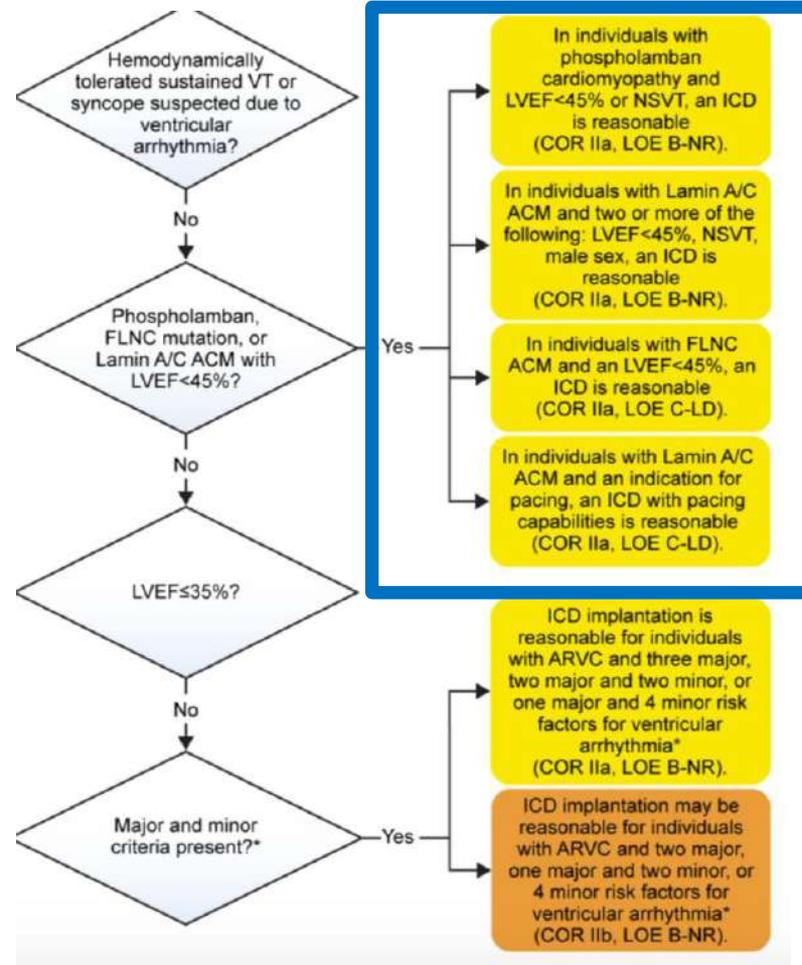
MIOCARDIOPATÍA DILATADA

Prevención de MS – La genética importa!

2019 HRS expert consensus statement on evaluation, risk stratification, and management of arrhythmogenic cardiomyopathy

Diferentes umbrales para implante de DAI (IIa B)

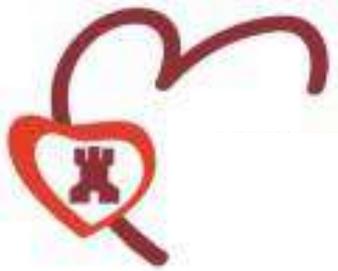
- **PLN:** FEVI <45% /TVNS
- **FLNC:** FEVI <45% /TVNS
- **LMNA:** varón, FEVI <45%, TVNS (2 ó +)



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Herramientas diagnósticas - BEM

CLASS OF RECOMMENDATION
I

Fulminant heart failure
<2 weeks duration

Dilated LV with
Ventricular arrhythmias
or AV block
or Refractory to conventional therapy
2 weeks - 3 months duration

CLASS OF RECOMMENDATION
IIA

Dilated LV with
Ventricular arrhythmias
or AV block
or Refractory to conventional therapy
> 3 months duration

Dilated LV with
Eosinophilia

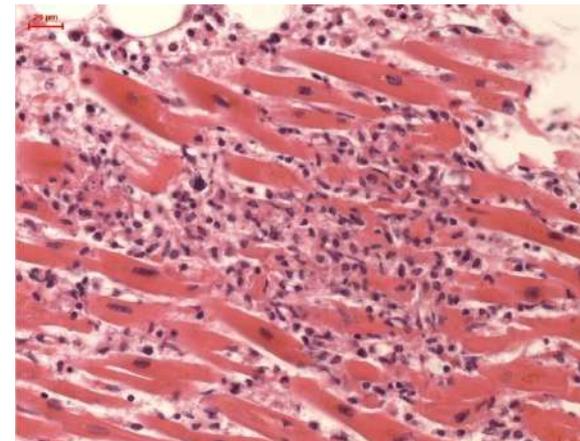
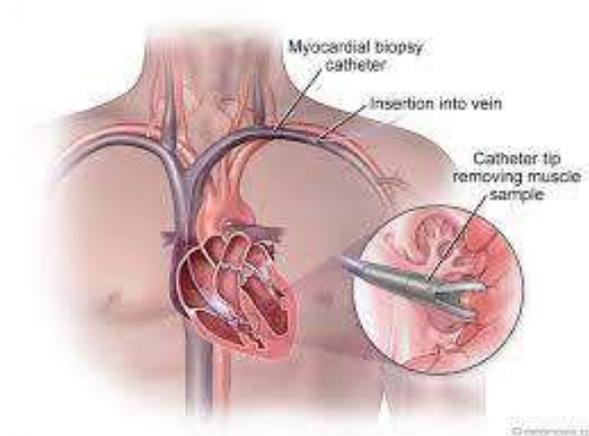
Selected cardiac masses
Not myxomas

Restrictive cardiomyopathy
or suspected anthracycline toxicity
Not diagnosed by imaging

CLASS OF RECOMMENDATION
IIB

Dilated LV *without*
Ventricular arrhythmias
or AV block
Responds to usual therapy in 1-2 weeks
> 2 weeks duration

Unexplained HCM Unexplained ARVC Unexplained ventricular arrhythmias



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MIOCARDIOPATÍA DILATADA no isquémica

BEM – Tratamientos dirigidos

Tabla 2

Opciones terapéuticas actuales para la miocarditis de células gigantes aguda y la miocardiopatía inflamatoria autoinmunitaria

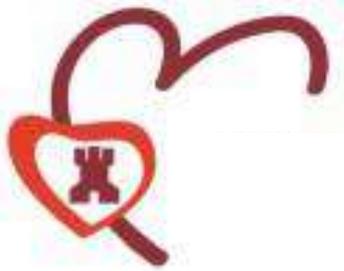
Miocarditis de células gigantes (Cooper et al^{33,34})
<i>Antitimoglobulina</i>
275 mg en 500 ml de solución salina al 0,9% durante 12/24 h
Días 1 a 5
Con monitorización cardiaca
<i>Ciclosporina</i>
Dosis inicial de 200 mg/24 h (100 mg/12 h)
Objetivo de concentración valle: 100-120 µg/ml
1 año
<i>Metilprednisolona</i>
Dosis inicial: 1 mg/kg
Después de 4 semanas: reducir la dosis 10 mg y luego otros 10 mg cada 2 semanas hasta llegar a la dosis de mantenimiento de 5-10 mg

Dominguez et al. Rev Esp Cardiol. 2016; 69:178–187

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Herramientas diagnósticas – 18 FDG PET/TC

Más allá de la sarcoidosis?

Incidence of abnormal positron emission tomography in patients with unexplained cardiomyopathy and ventricular arrhythmias: The potential role of occult inflammation in arrhythmogenesis  



Roderick Tung, MD, FHRS,* Brenton Bauer, MD,* Heinrich Schelbert, MD,†
Joseph P. Lynch III, MD,† Martin Auerbach, MD,† Pawan Gupta, MD,† Christiaan Schiepers, MD,†
Samantha Chan,* Julie Ferris, BA,* Martin Barrio, MS,† Olujimi Ajijola, MD, PhD,*
Jason Bradfield, MD, FHRS,* Kalyanam Shivkumar, MD, PhD, FHRS*†

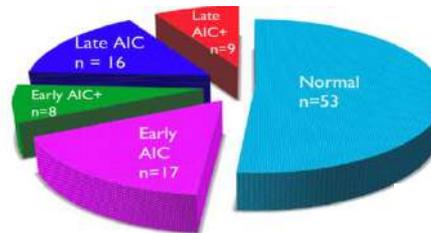
17% sarcoidosis

103 PET

FEVI media 32%

49% con captación cardiaca (n=5)

- +/- defectos de perfusión
- +/- captación ganglionar



✓ Mejoría del 20% LVEF en pacientes con inflamación sin defectos de perfusión

vs.

✓ Mejoría del 5% en pacientes con defectos de perfusión.

(p:0.045)

19 p. con mapa electroanatómico: **79%** correlacionan bajos voltajes con inflamación

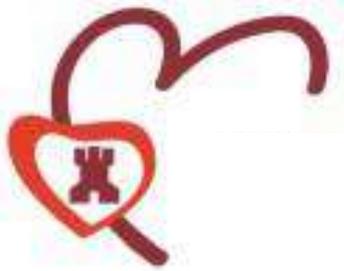
25 p. con biopsias

- 10 BEM: 30% granulomas , 60% inflamación crónica, 10% sin hallazgos.
- 15 ganglionar: 100% granulomas

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MIOCARDIOPATÍA DILATADA no isquémica

Tratamientos específicos por etiología

	Current State	Ongoing Projects	Future Directions
Upstream triggers			
Genetic	Prevention with device therapy in gene mutations susceptible to malignant arrhythmias (ie, <i>LMNA</i>)	Phase 3 clinical trial using p38 inhibition in <i>LMNA</i> DCM patients	<ul style="list-style-type: none"> Unravel molecular consequences of specific gene mutations Gene correction therapies towards a clinical application
Inflammation	No guideline-directed therapy; although there is evidence from retrospective studies showing benefit from immunosuppression	Phase 2B RCT in acute myocarditis using anakinra vs standard care	<ul style="list-style-type: none"> Multicenter RCT using immunosuppressive therapy in inflammatory DCM
Auto-immunity	No guideline-directed therapy; although there is evidence showing benefit from immunoadsorption	...	<ul style="list-style-type: none"> Multicenter RCT using immunoadsorption in DCM with cardiotoxic autoantibodies
Viral	No guideline-directed therapy; although there are retrospective studies and case reports showing benefit from IVIg	Phase 3 RCT using IVIg for chronic PVB19-related DCM	<ul style="list-style-type: none"> RCT using specific antiviral therapies (val-/ganciclovir) Multicenter RCT for IVIg if phase 3 trial is positive

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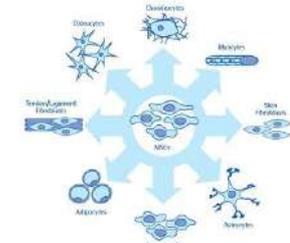


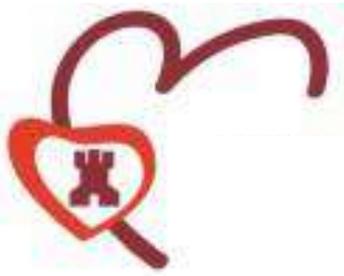
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MIOCARDIOPATÍA DILATADA

Tratamientos específicos

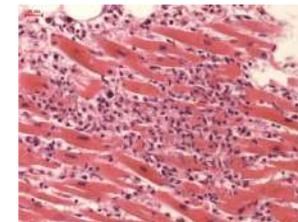
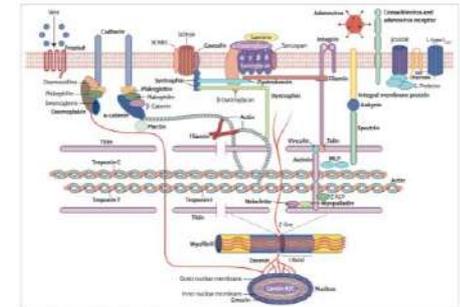
<p>Toxic</p>	<p>No guideline-directed therapy; withholding or reducing exposure has been shown to be the most effective; in some cases, cardioprotective compounds for anthracycline toxicity are advised.</p>	<p>Phase 1 RCT using MPCs in anthracycline-induced DCM</p> 	<ul style="list-style-type: none"> Unravel molecular changes in cocaine-induced DCM Phase 2 RCT using stem cell therapy in cardiotoxic chemotherapy-induced DCM Define the timing, dose, and duration of prophylactic therapy to prevent HF onset in those patients receiving cardiotoxic chemotherapy at risk
<p>Electrical</p>	<p>Early treatment of electrical disturbance (AF, ablation; left bundle branch block, CRTD)</p>	<p>...</p>	<ul style="list-style-type: none"> Better insight in the pathomechanisms of the interplay between HF and electrical disturbances to detect their causal relationships and better stratify patients who will benefit from (device) therapy
<p>Peripartum</p>	<p>No guideline-directed therapy; although 1 prospective study showed benefit from bromocriptine</p>	<p>...</p>	<ul style="list-style-type: none"> A placebo-controlled study with bromocriptine to assess safety and efficacy More data regarding long-term outcome of PPCM after bromocriptine use



MCD NO ISQUÉMICA- Etiología

CONCLUSIONES

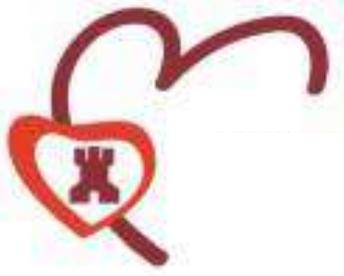
- La miocardiopatía dilatada tiene **etiologías muy diversas**, pero con mucha frecuencia la causa no se encuentra y se considera idiopática.
- Siempre es necesario excluir en primer lugar la cardiopatía isquémica, valvular o congénita.
- Los estudios familiares han revelado que entre el **40 y el 70%** de los pacientes diagnosticados con de MCD idiopática tienen un **componente genético**
→ *el manejo clínico puede cambiar por la genética!*
- En algunos casos, la **biopsia endomiocárdica** puede confirmar el diagnóstico etiológico y permite iniciar un tratamiento específico.



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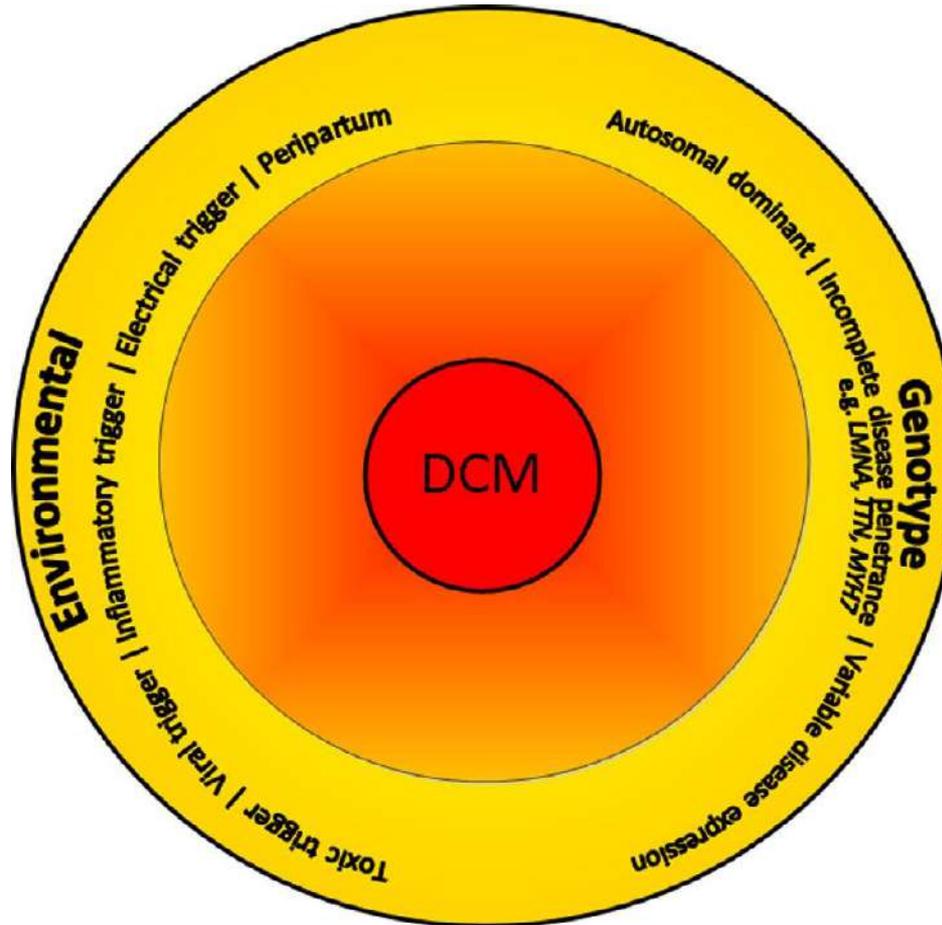
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MCD NO ISQUÉMICA- Etiología

CONCLUSIONES

Ambiente

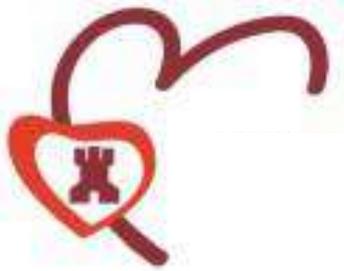


Genotipo

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Muchas gracias por vuestra atención!



www.cardiopatiasfamiliares.es

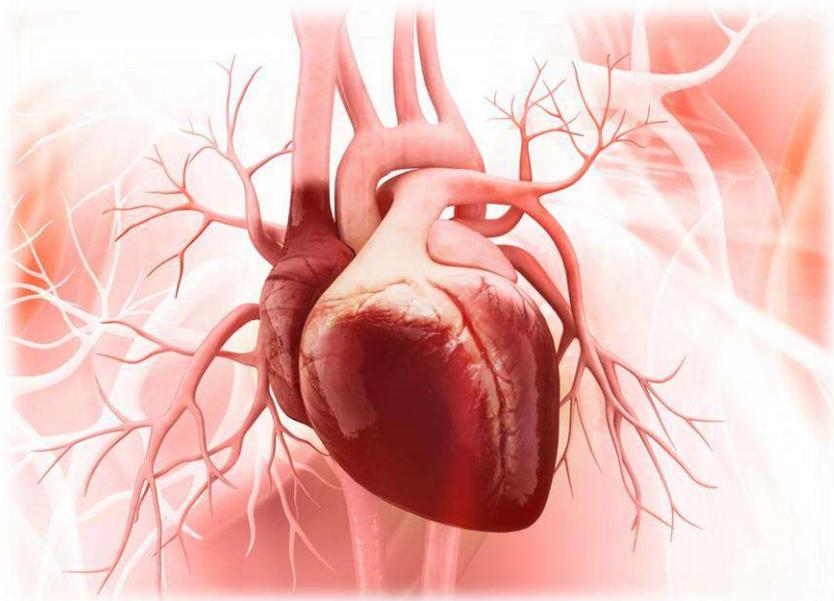
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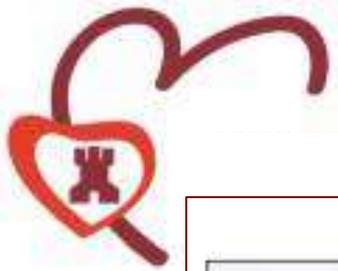
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¿Cómo hacer un buen diagnóstico de hipertensión pulmonar?

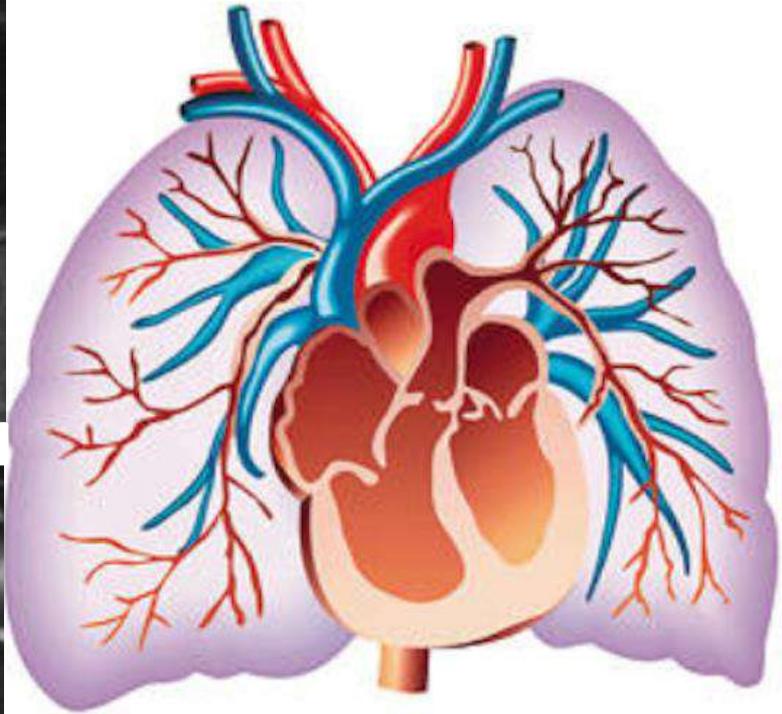
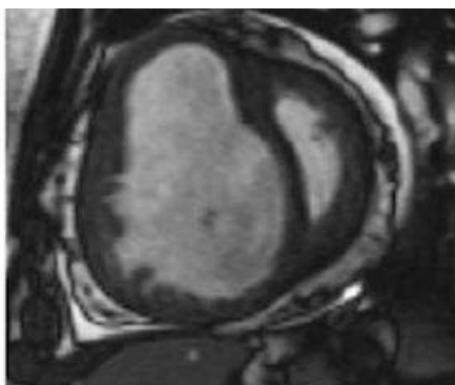
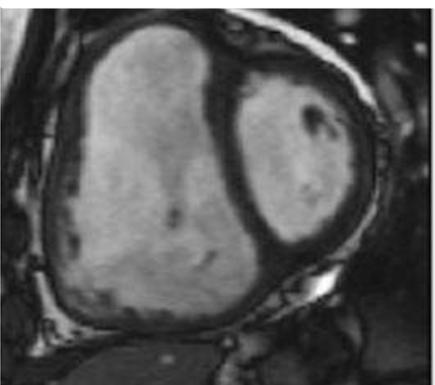
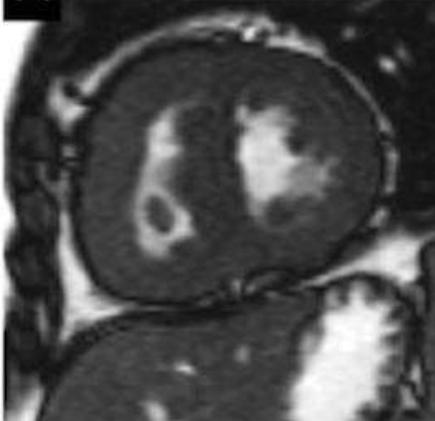
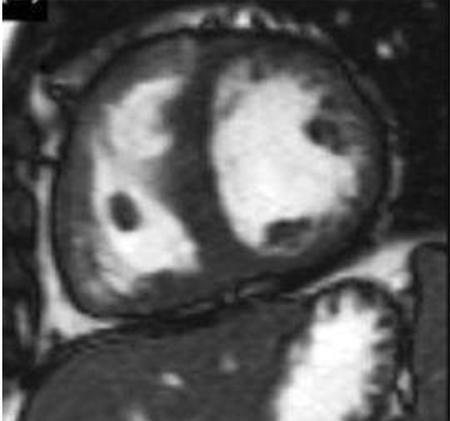


Dra. María Lázaro Salvador
Complejo Hospitalario de Toledo



¿Qué es la HAP ?

Healthy patient



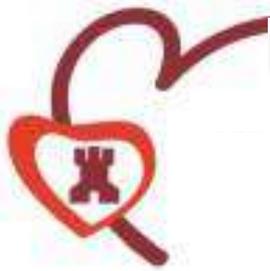
Hipertensión Pulmonar



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1 PAH

- 1.1 Idiopathic PAH
- 1.2 Heritable PAH
- 1.3 Drug- and toxin-induced PAH (table 3)
- 1.4 PAH associated with:
 - 1.4.1 Connective tissue disease
 - 1.4.2 HIV infection
 - 1.4.3 Portal hypertension
 - 1.4.4 Congenital heart disease
 - 1.4.5 Schistosomiasis
- 1.5 PAH long-term responders to calcium channel blockers (table 4)
- 1.6 PAH with overt features of venous/capillaries (PVOD/PCH) involvement (table 5)
- 1.7 Persistent PH of the newborn syndrome

2 PH due to left heart disease

- 2.1 PH due to heart failure with preserved LVEF
- 2.2 PH due to heart failure with reduced LVEF
- 2.3 Valvular heart disease
- 2.4 Congenital/acquired cardiovascular conditions leading to post-capillary PH

3 PH due to lung diseases and/or hypoxia

- 3.1 Obstructive lung disease
- 3.2 Restrictive lung disease
- 3.3 Other lung disease with mixed restrictive/obstructive pattern
- 3.4 Hypoxia without lung disease
- 3.5 Developmental lung disorders

4 PH due to pulmonary artery obstructions (table 6)

- 4.1 Chronic thromboembolic PH
- 4.2 Other pulmonary artery obstructions

5 PH with unclear and/or multifactorial mechanisms (table 7)

- 5.1 Haematological disorders
- 5.2 Systemic and metabolic disorders
- 5.3 Others
- 5.4 Complex congenital heart disease

Clasificación

Haemodynamic definitions and updated clinical classification of pulmonary hypertension.

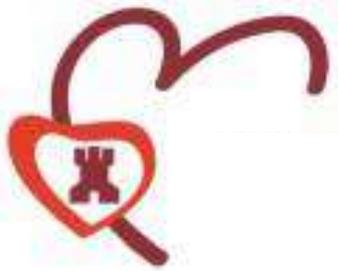
Gérald Simonneau et al. ERJ Express. December 13, 2018

doi: 10.1183/13993003.01913-2018

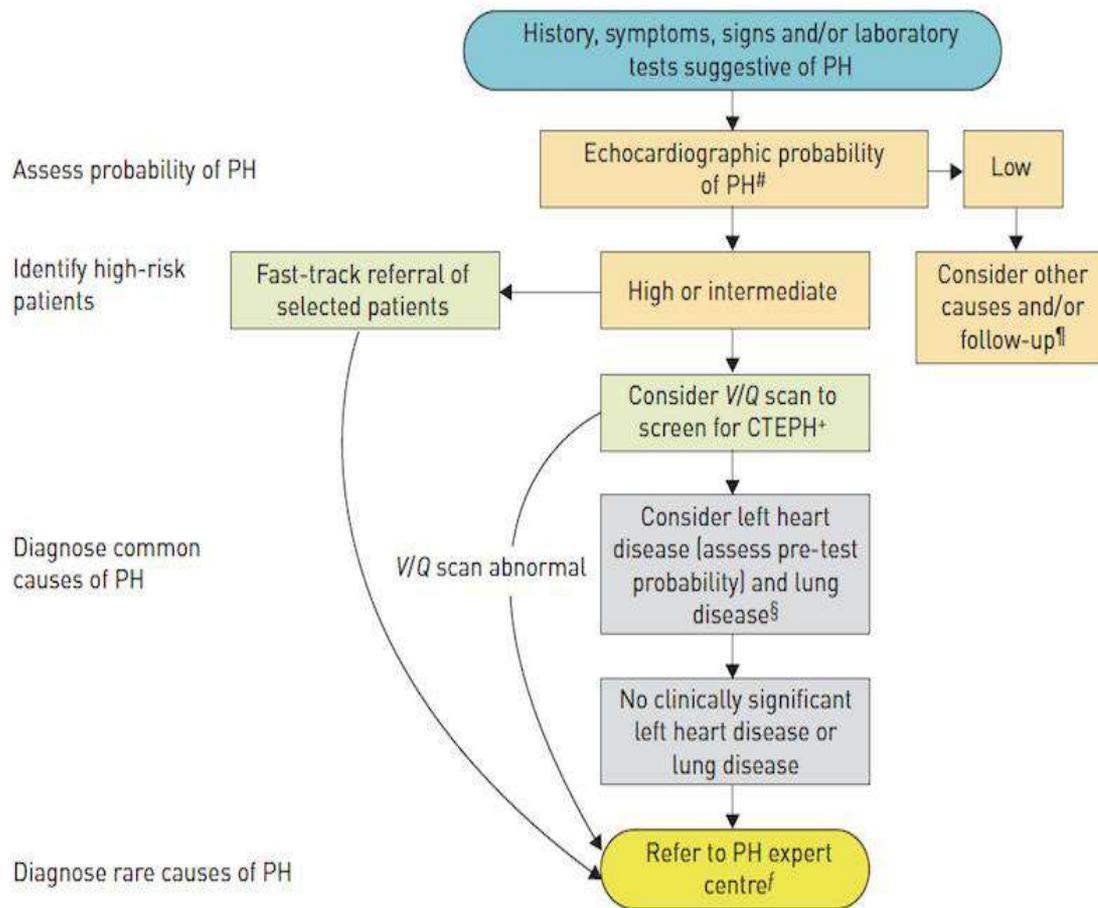
FLASHLIGHTS en



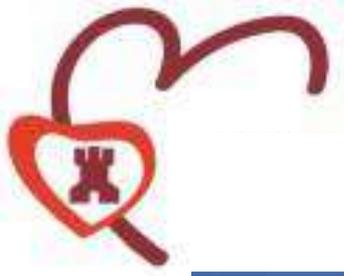
**INSUFICIENCIA
CARDIACA**



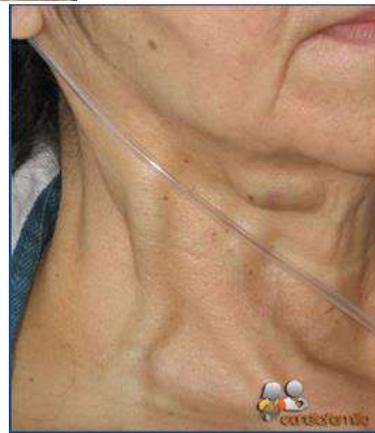
Diagnóstico



Diagnosis of pulmonary hypertension. Adaani Frost et al. ERJ Express. December 13, 2018 as doi: 10.1183/13993003.01904-2018



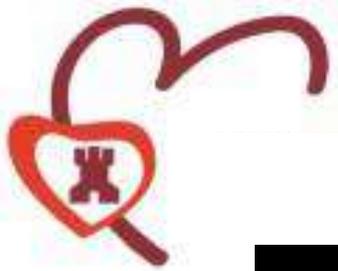
Diagnóstico: síntomas



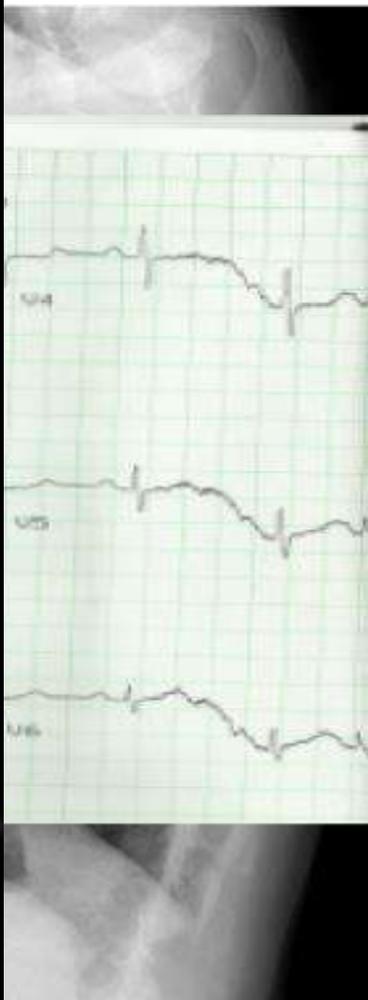
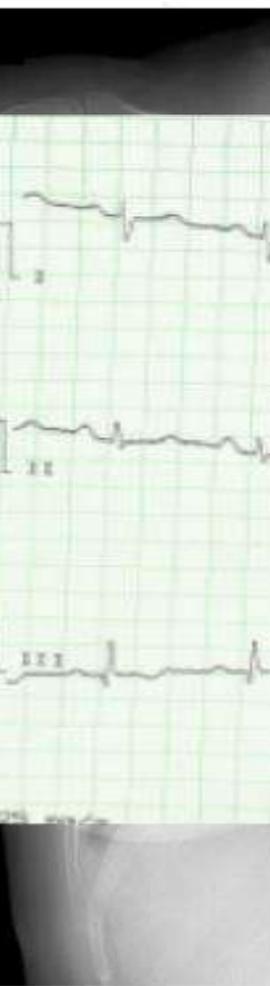
FLASHLIGHTS en



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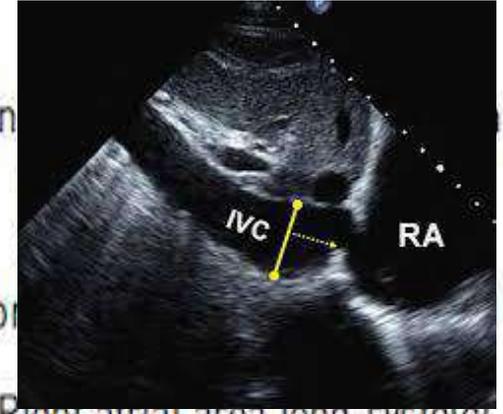
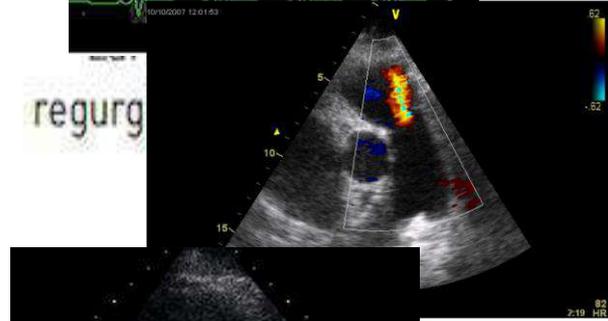
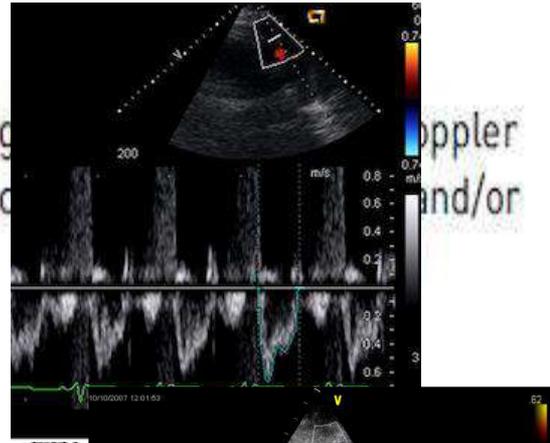
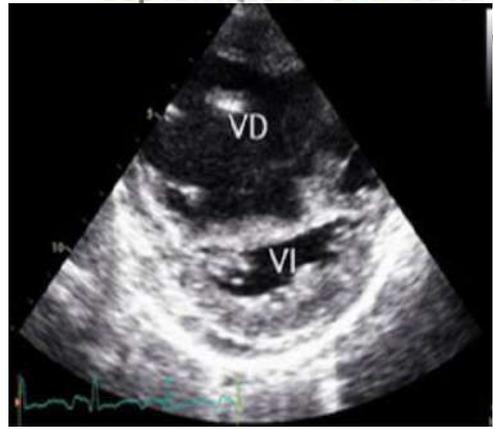
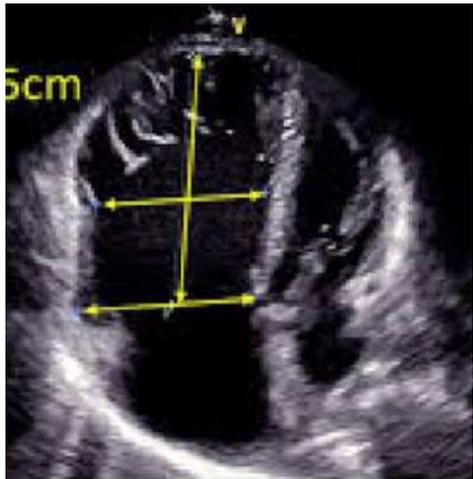
Diagnóstico: Pruebas



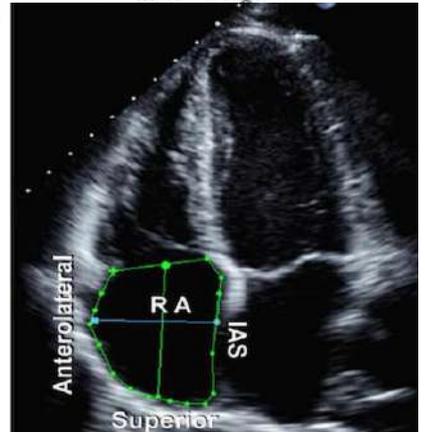
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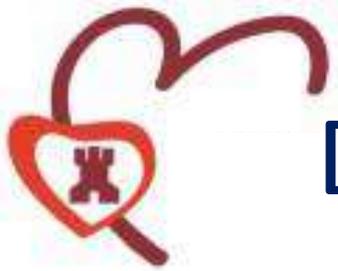
Diagnóstico: Ecocardiograma



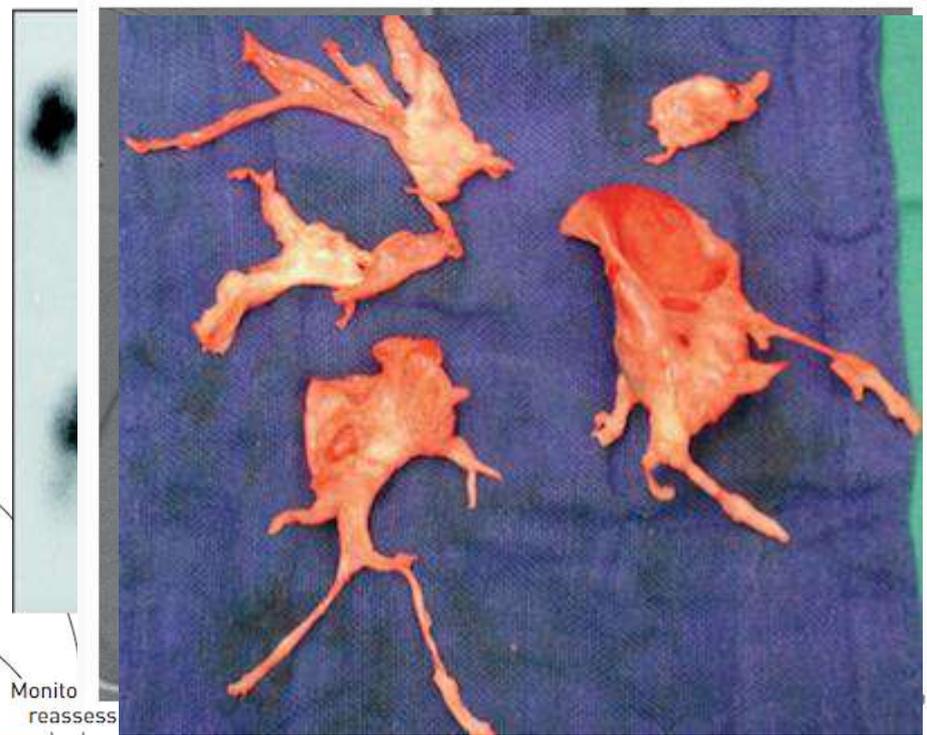
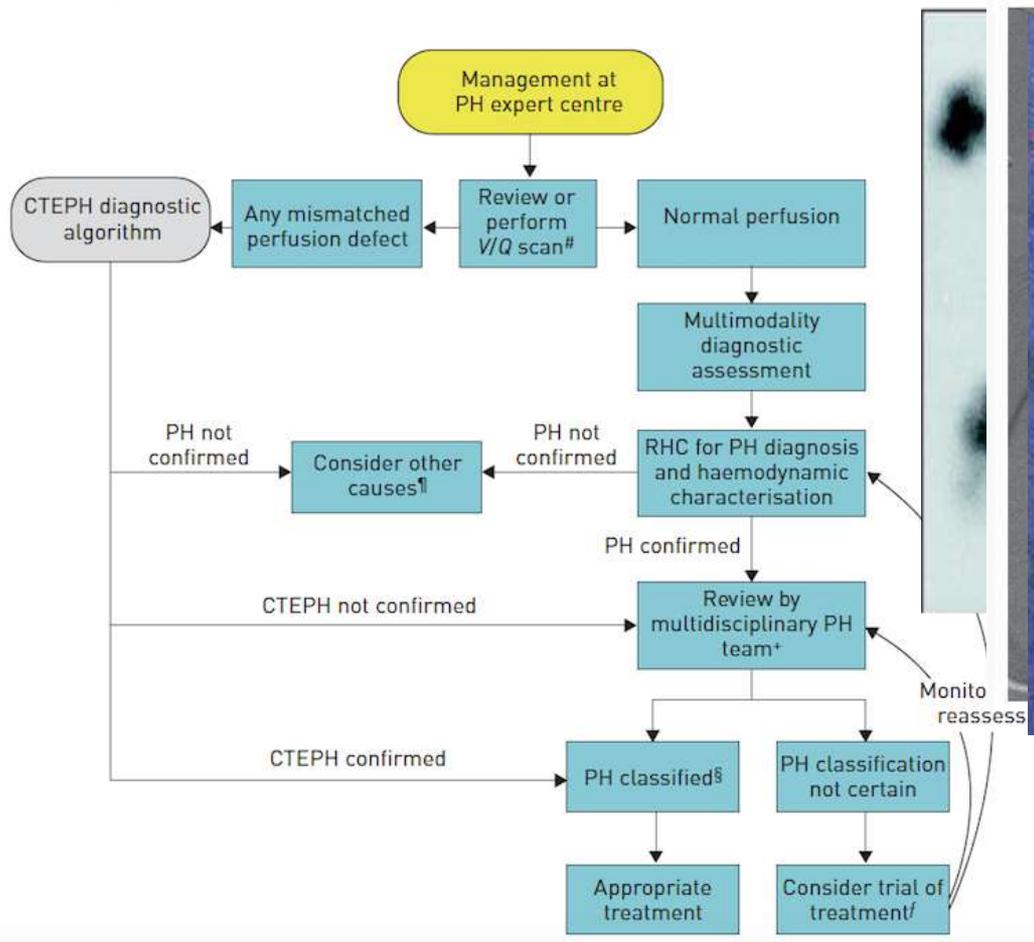
Right atrial area (end-systole)



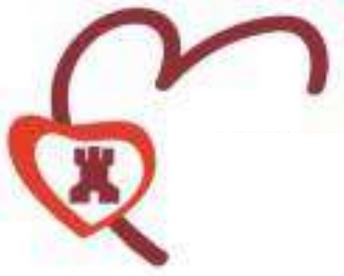
Frost A, et al. European Respiratory Journal 2019 53:1801904.



Diagnóstico: Confirmación

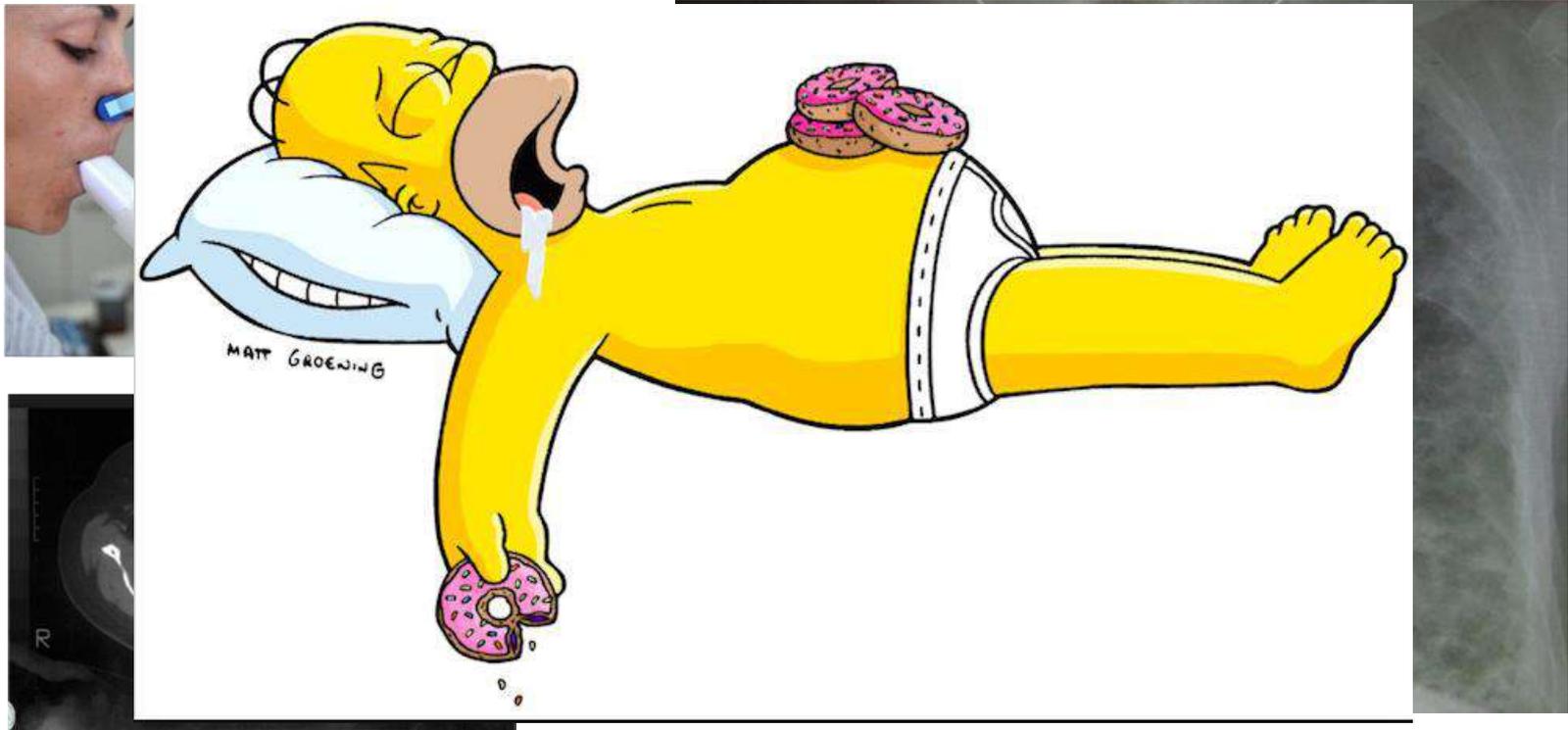


Diagnosis of pulmonary hypertension. Adaani Frost et al. ERJ Express. December 13, 2018 as doi: 10.1183/13993003.01904-2018



Diagnóstico diferencial

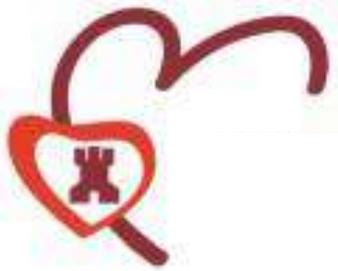
- HP Grupo 3



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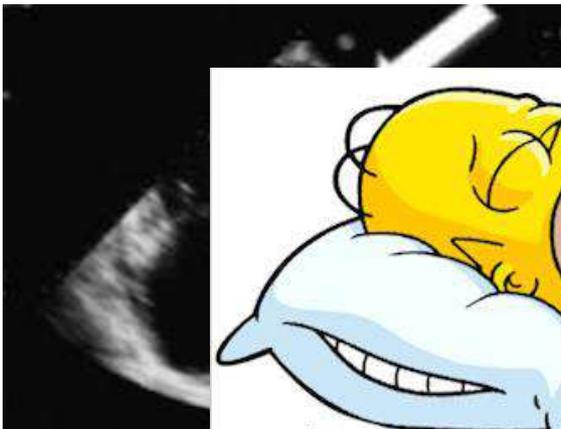


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Diagnóstico diferencial

- HP grupo 2



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Clasificación hemodinámica

H ARTERIAL P: HP PRECAPILAR

- mPAP >20 mmHg
- PAWP ≤15 mmHg
- PVR ≥3 WU



HP POSTCAPILAR

- mPAP >20 mmHg
- PAWP >15 mmHg
- PVR <3 WU

(PAPD-PAWP)

DPD ≥ 7

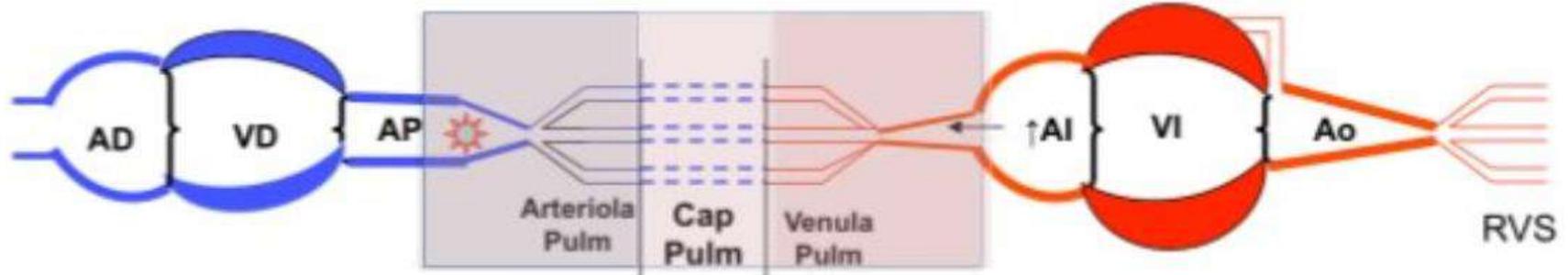
RVP >3

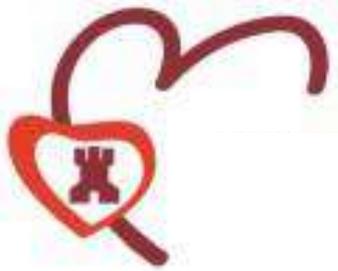
+ componente precapilar

DPD < 7

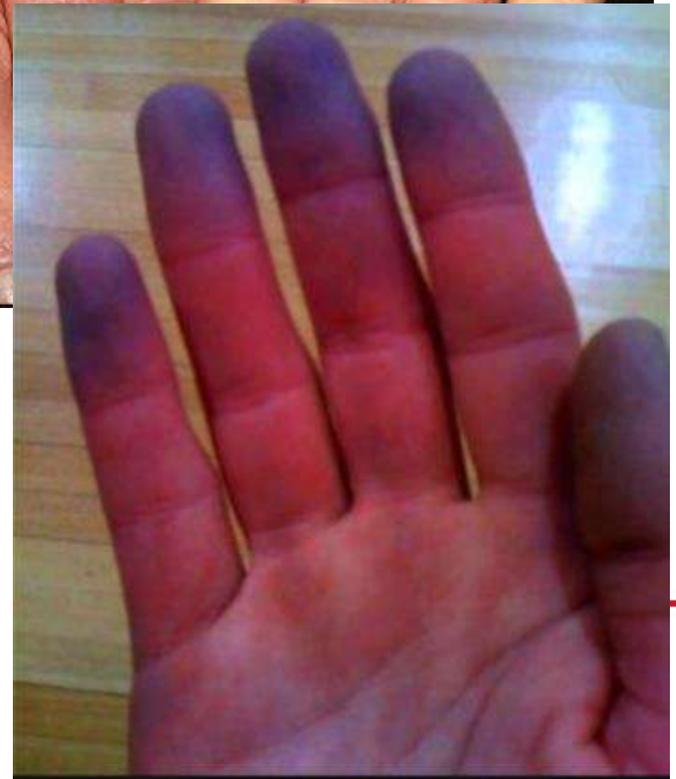
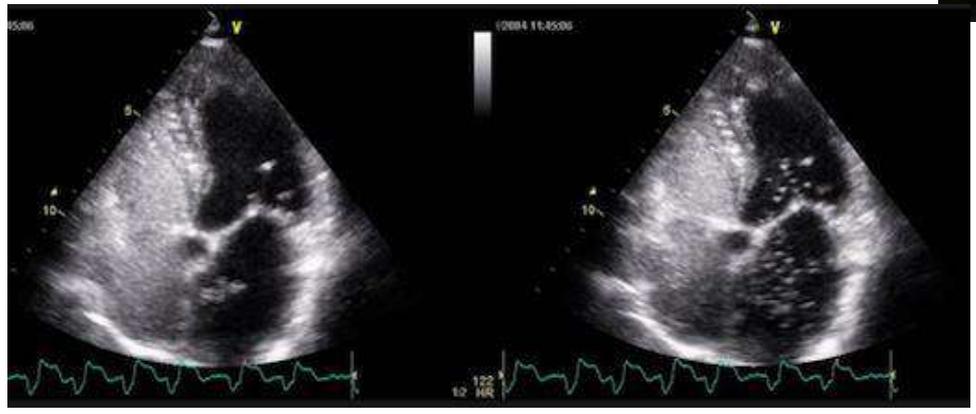
RVP ≤3

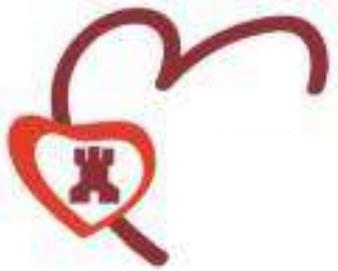
aislada





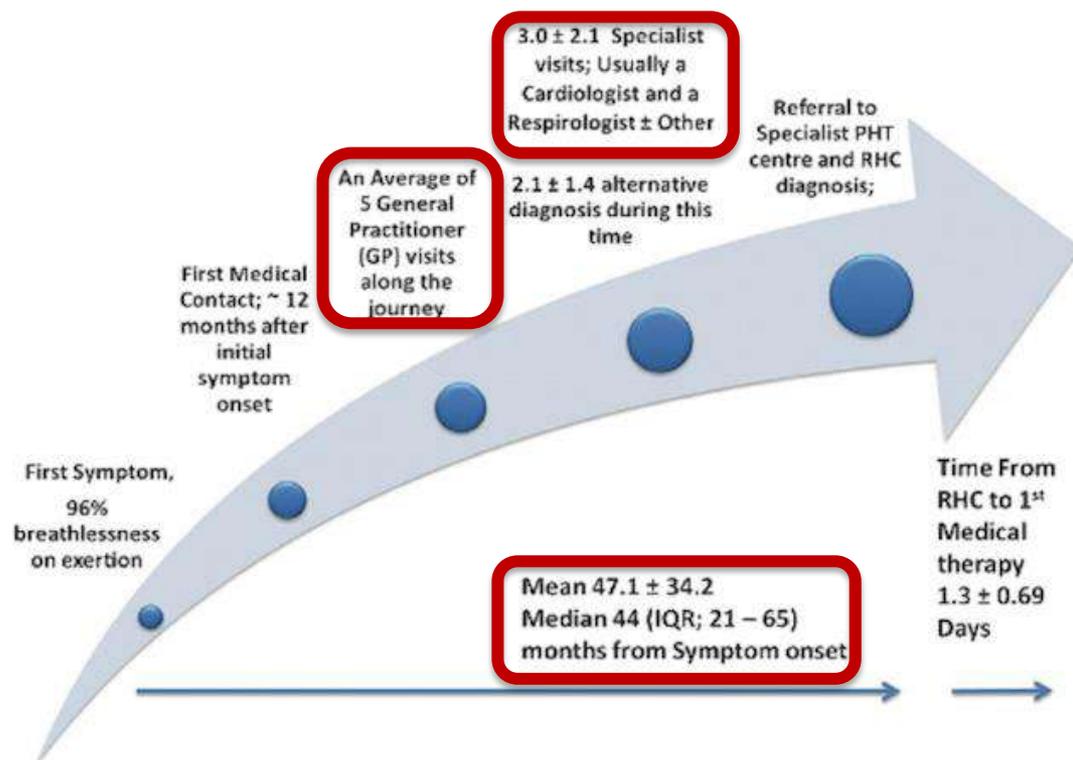
Caracterizar HAP -1





Tiempo al diagnóstico

Strange et al.: DELAY study

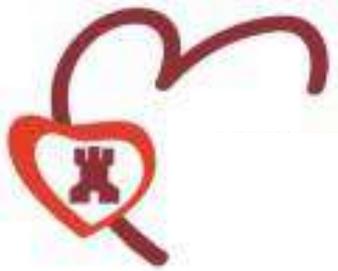


Strange G, et al. Pulm Circ 2013; 3:89-94.

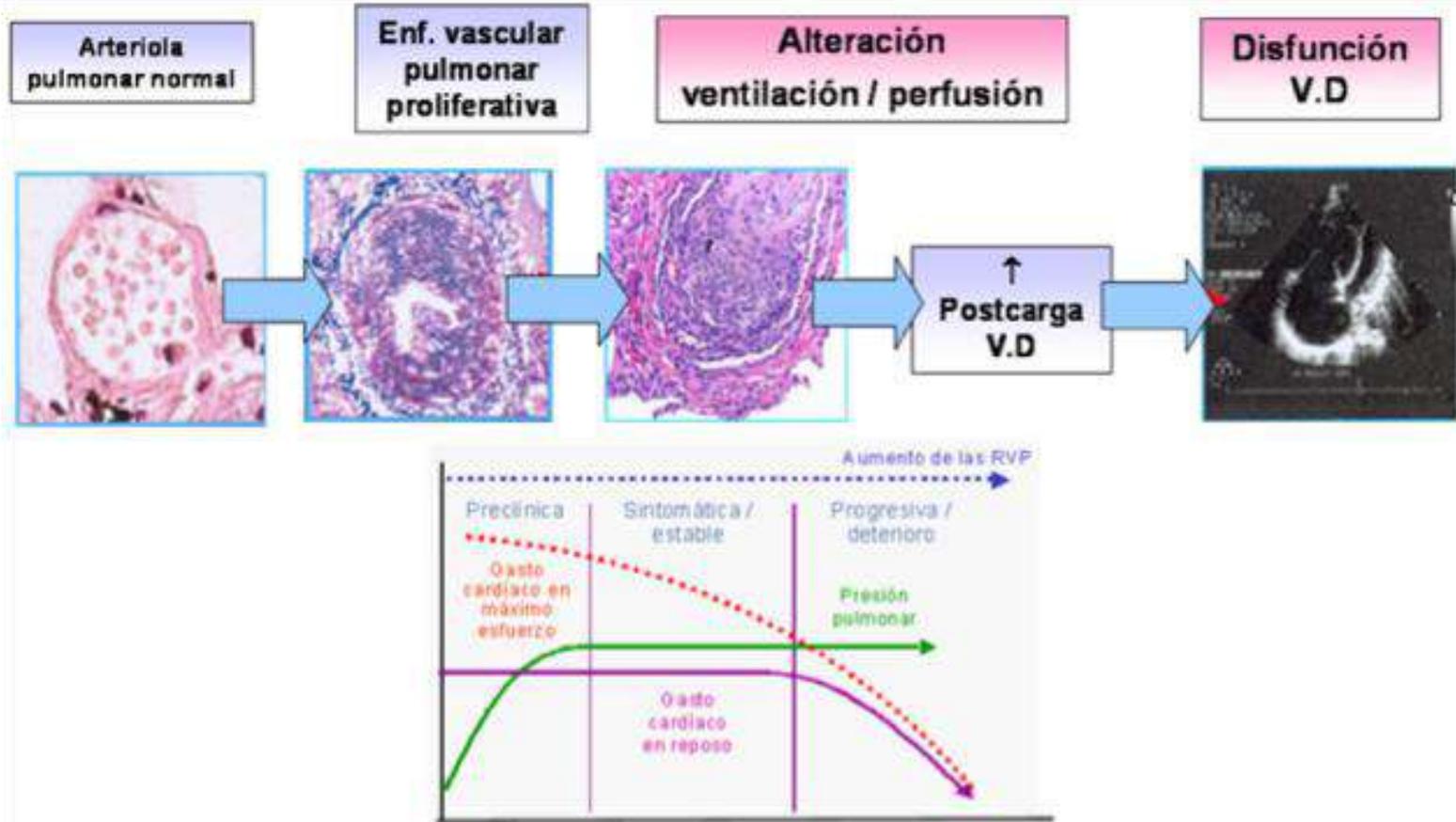
FLASHLIGHTS en



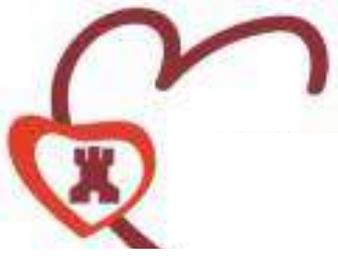
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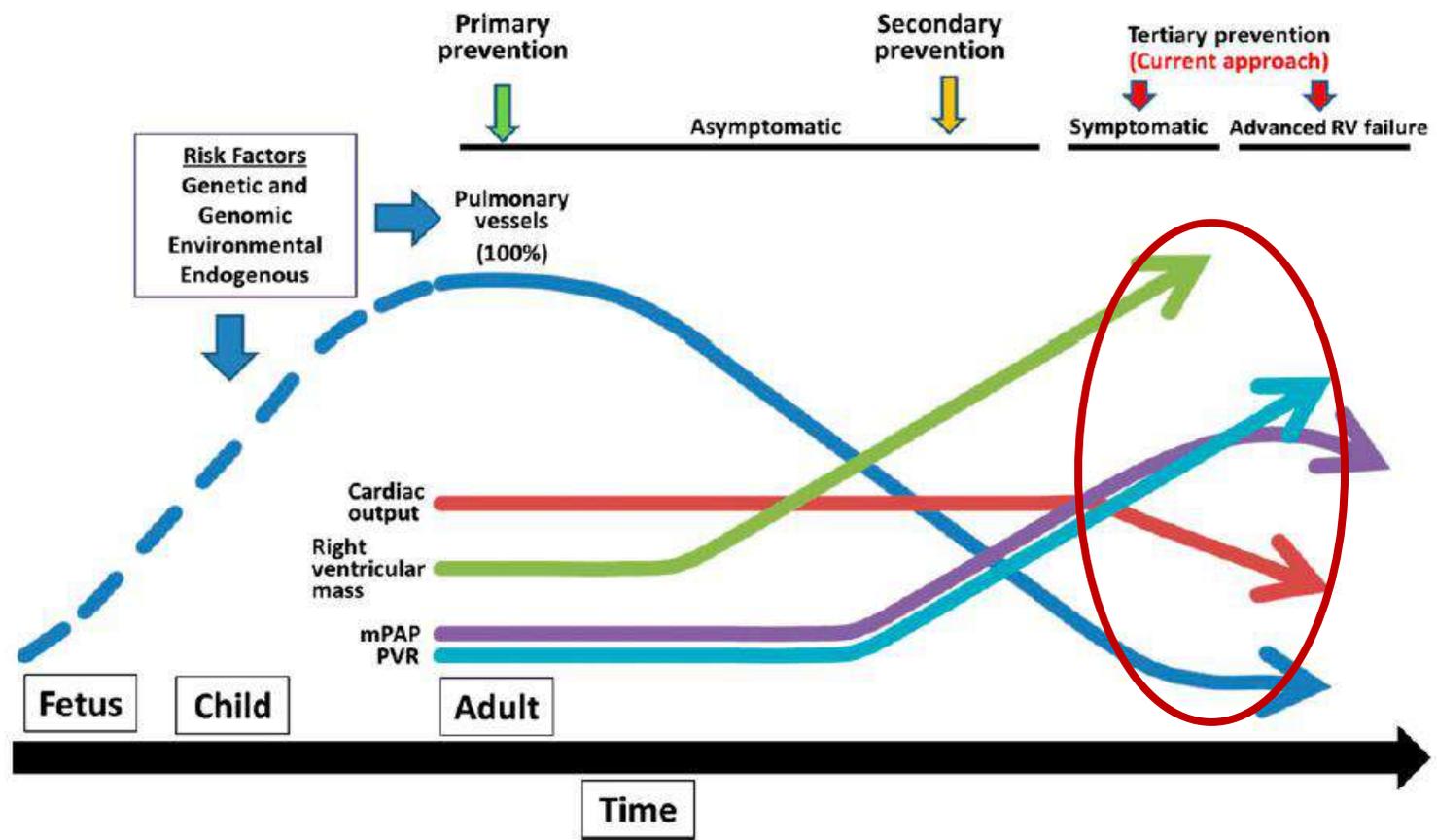
Historia natural



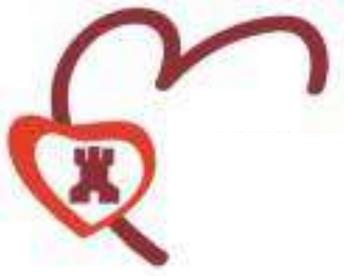
Friedman EB (2006). Classification and prognosis of pulmonary arterial hypertension. In Mandel J and Taichman D (Eds.) Pulmonary vascular disease p66-82. Philadelphia, PA: Saunders Elsevier.



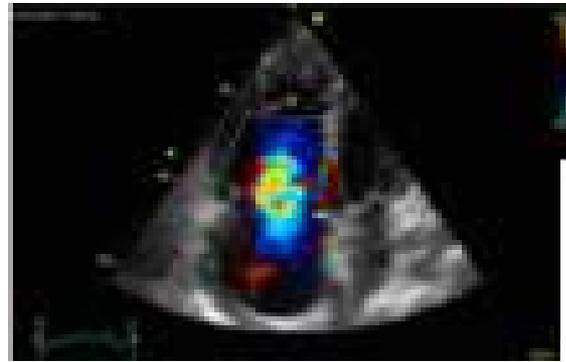
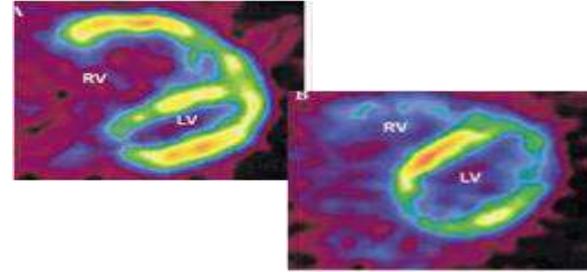
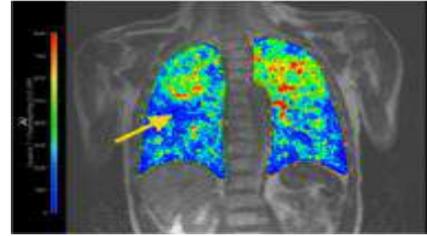
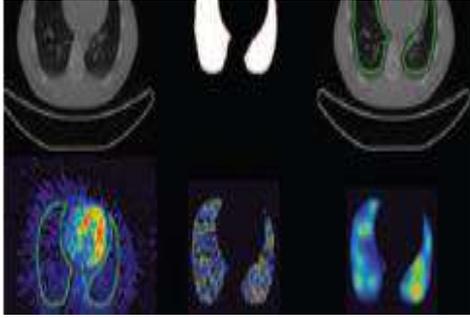
Diagnóstico tardío



Austin ED et al Ann Am Thor Soc 2014, 11: 5178-85



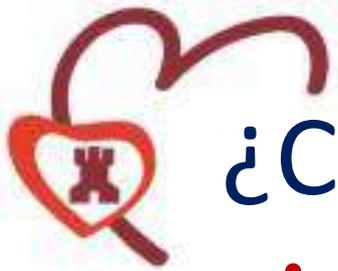
Diagnóstico precoz



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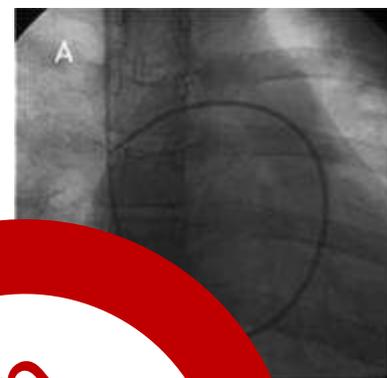
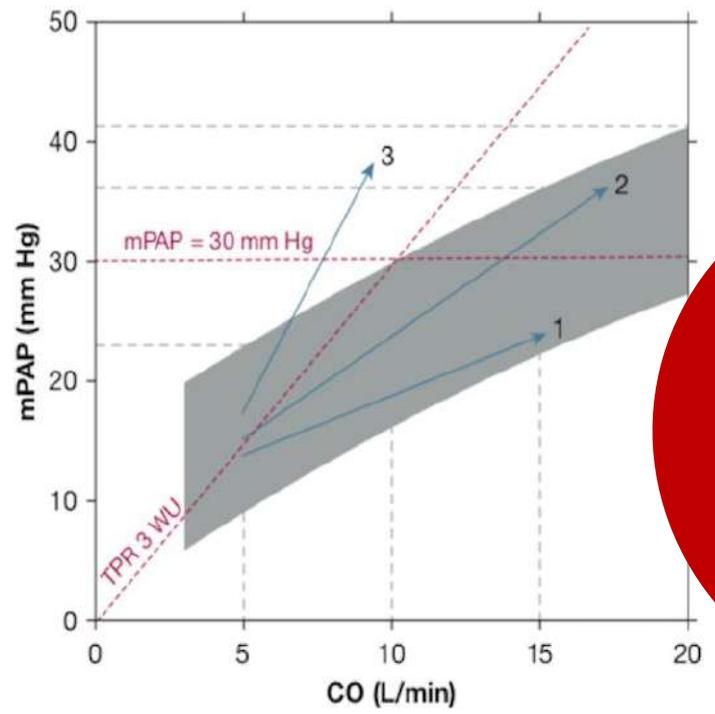


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¿Cómo adelantar diagnóstico?

- ¿¿ Desenmascarar HAP al esfuerzo ??



Aún
no

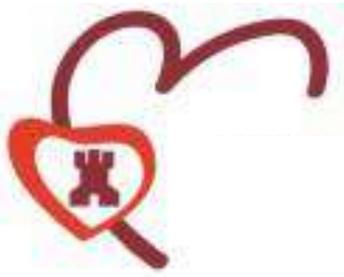


*Exercise-Induced Pulmonary Hypertension Translating Pathophysiological Concepts Into Clinical Practice. Robert Naeije et al. CHEST 2018.
<https://doi.org/10.1016/j.chest.2018.01.022>*

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Ergoespirometría

Ergoespirometría en HAP

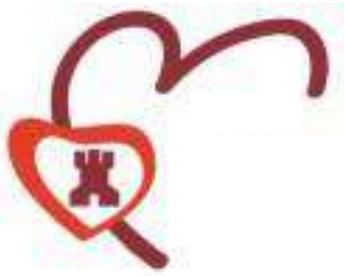
- Descenso capacidad aeróbica:
 - ↓ VO_2
 - ↓ UA
 - ↓ VO_2/FC
- Ineficiencia ventilatoria:
 - ↑ $EqCO_2$
 - ↑ EqO_2
 - ↓ PET CO_2
 - ↑ PET O_2



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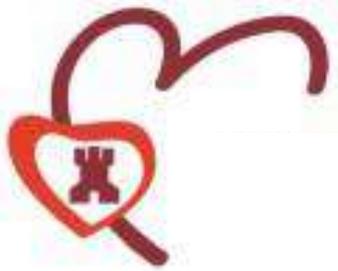
Mensajes para llevar

- La HAP es una enfermedad rara y muy grave
- Diagnóstico difícil: Diagnóstico diferencial
- Diagnóstico tardío
- El cateterismo derecho es imprescindible
- Testar el sistema con el esfuerzo:
 - Cateterismo esfuerzo
 - Ergoespirometría

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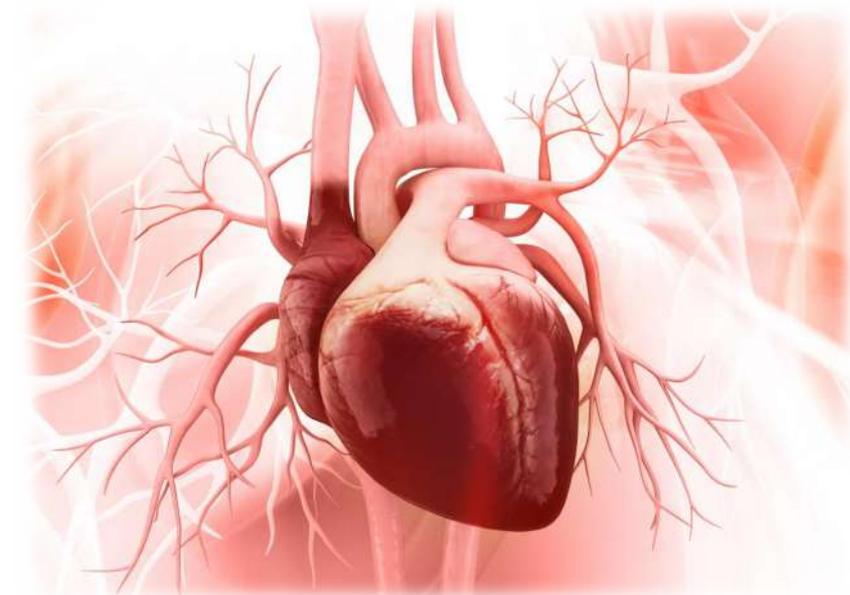


Gracias





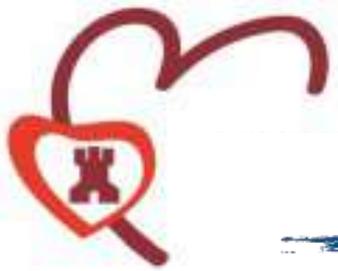
¿Trato igual al diabético que al no diabético?



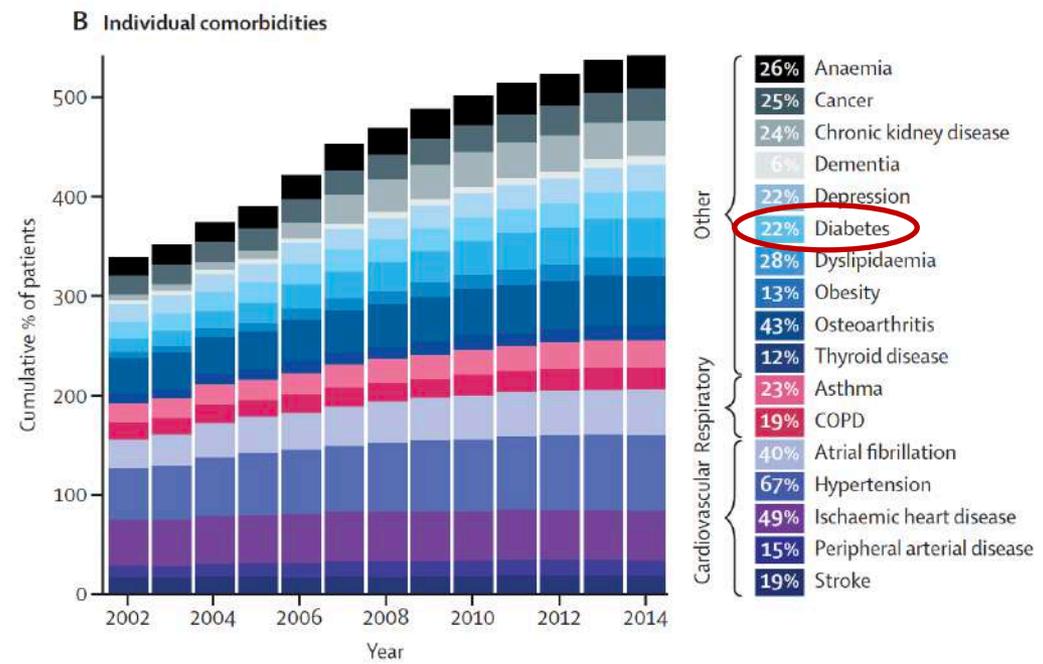
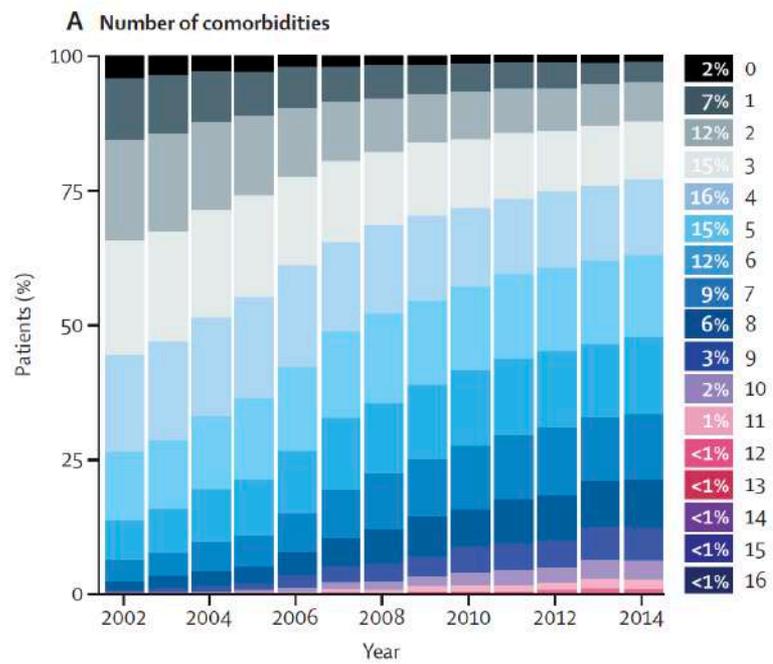
Dr. Javier de Juan Bagudá

Programa Transversal de Insuficiencia Cardíaca

Hospital Universitario 12 de Octubre

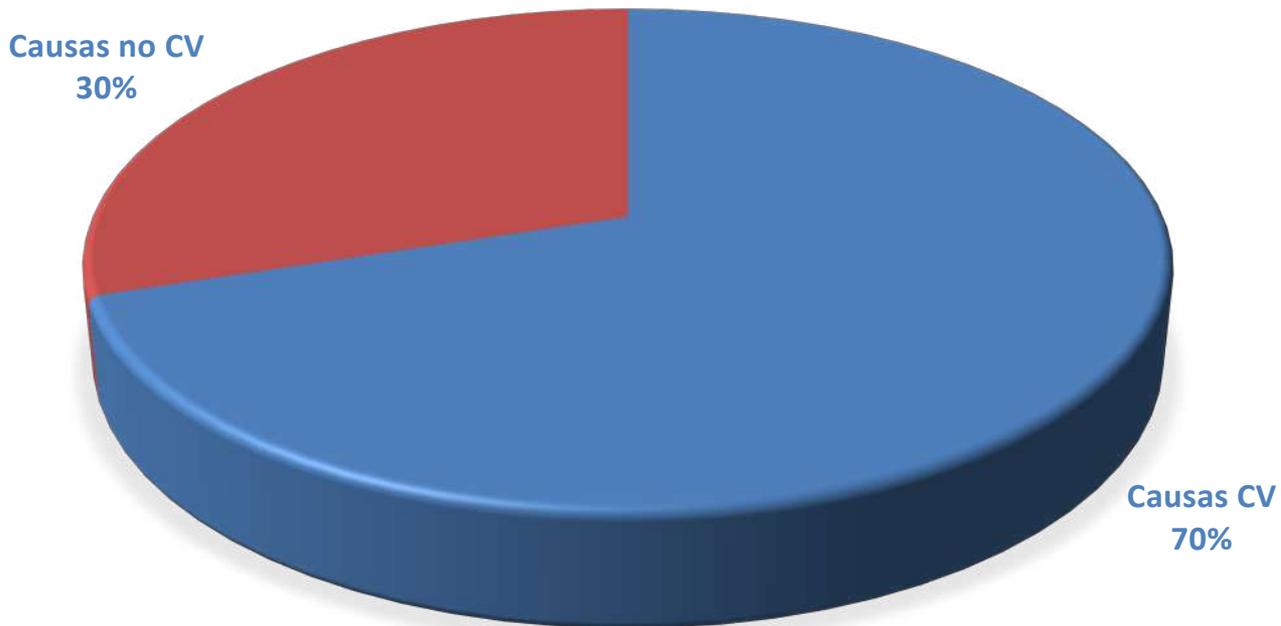


Alta prevalencia de DM en IC





1ª causa de mortalidad en DM: CV

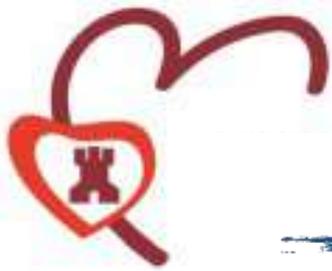


Mediavilla J. SEMERGEN. 2014; 30(Supl. 1):68-69
Lundberg V et al. J Intern Med. 1997 Jun;241(6):485-92.
Air EL et al. Diabetes Care. 2007;30(12):3131-3140.

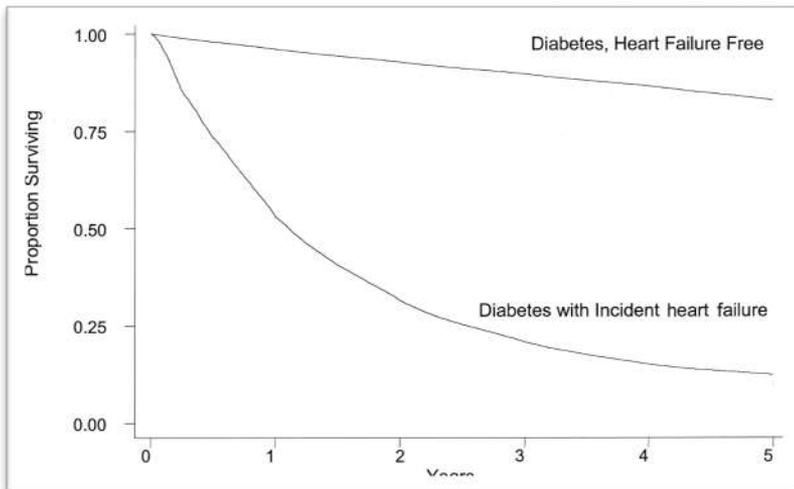
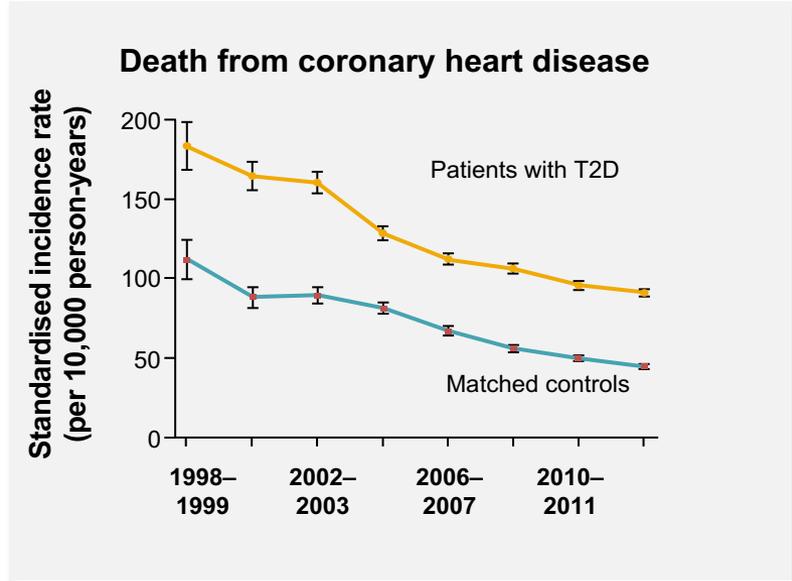
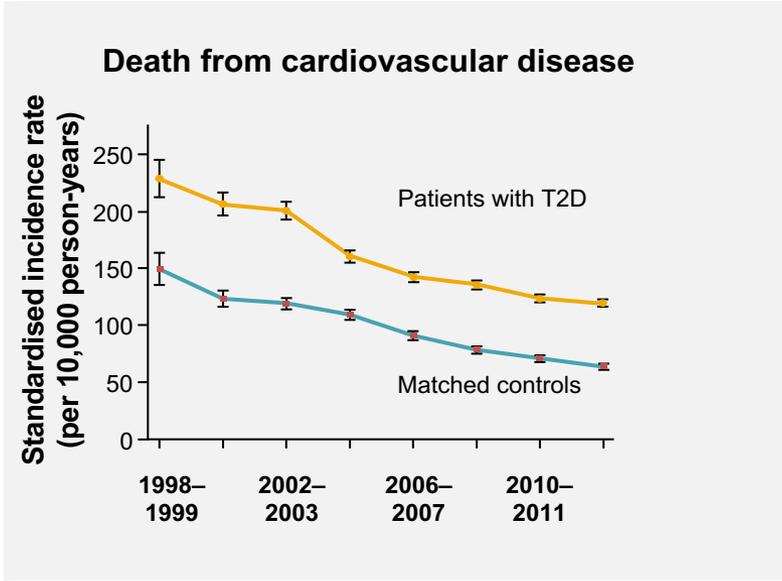
FLASHLIGHTS en



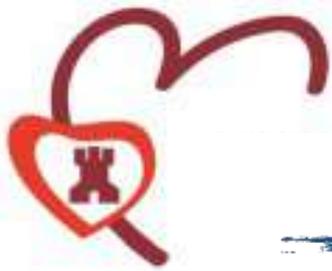
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La DM aumenta la mortalidad en pacientes CV



Rawshani A, et al. *N Engl J Med.* 2017;376:1407-1418.
Bertoni AG et al. *Diabetes Care.* 2004 Mar;27(3):699-703.



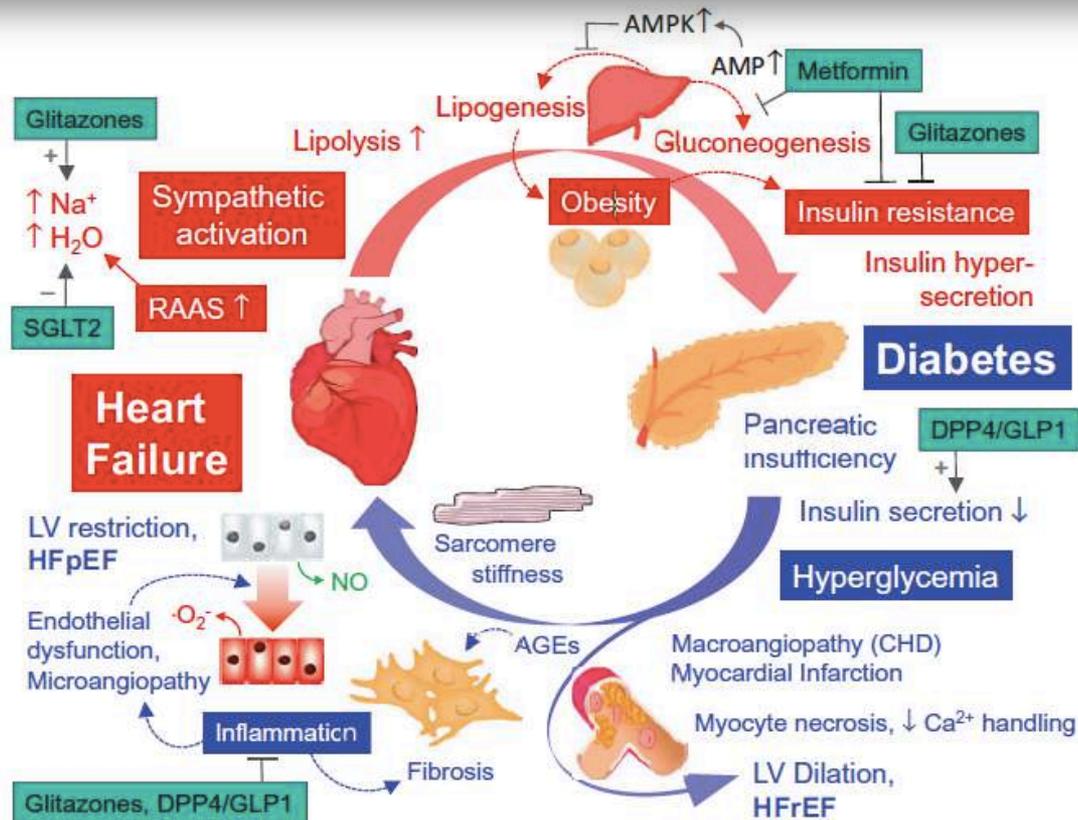
DM e IC, muy relacionadas

Diabetes

Diabetic cardiomyopathy; mitochondrial dysfunction; abnormal calcium homeostasis; oxidative stress; renin-angiotensin-aldosterone system (RAAS) activation; atherosclerosis; coronary artery disease

Incident and worsening diabetes mellitus via sympathetic and RAAS activation

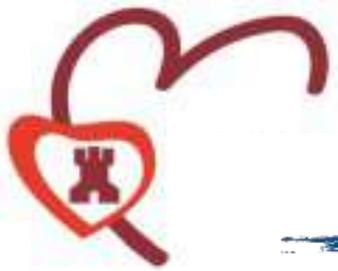
More prevalent in HFpEF
Similar increased risk for mortality in both groups



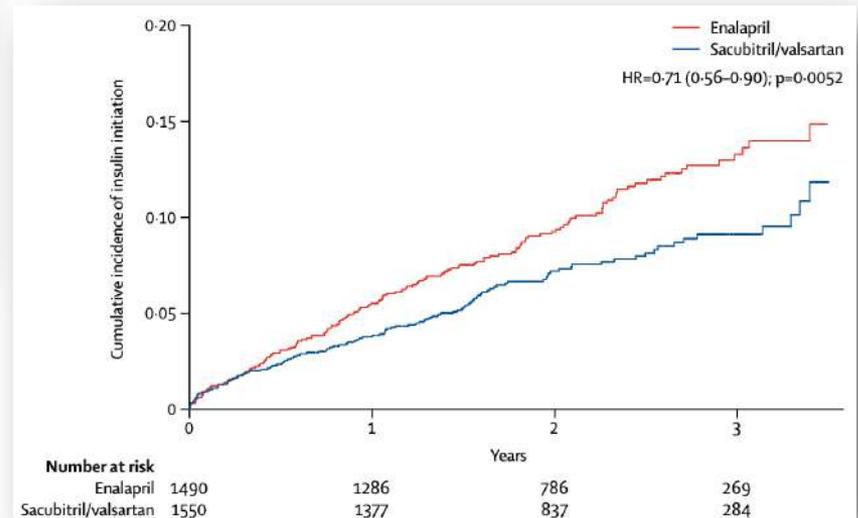
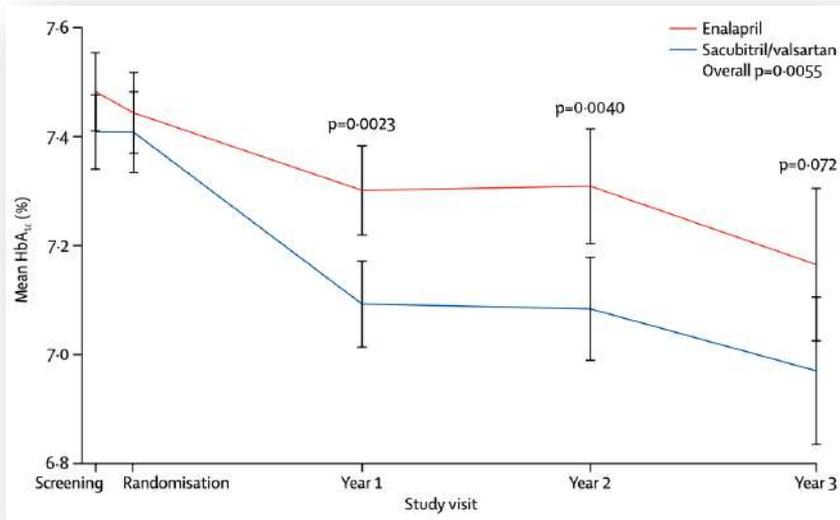
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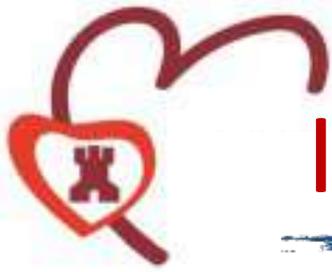
SV, mejor control de DM



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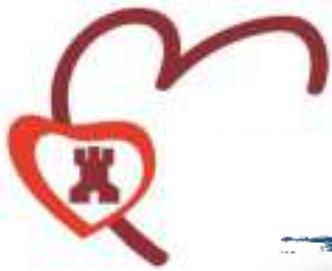
Impacto del control glucémico

Study	Microvascular		CV Disease		Mortality	
UKPDS	↓	↓	↔	↓	↔	↓
DCCT / EDIC*	↓	↓	↔	↓	↔	↔
ACCORD	↓		↔		↑	
ADVANCE	↓		↔		↔	
VADT	↓		↔		↔	

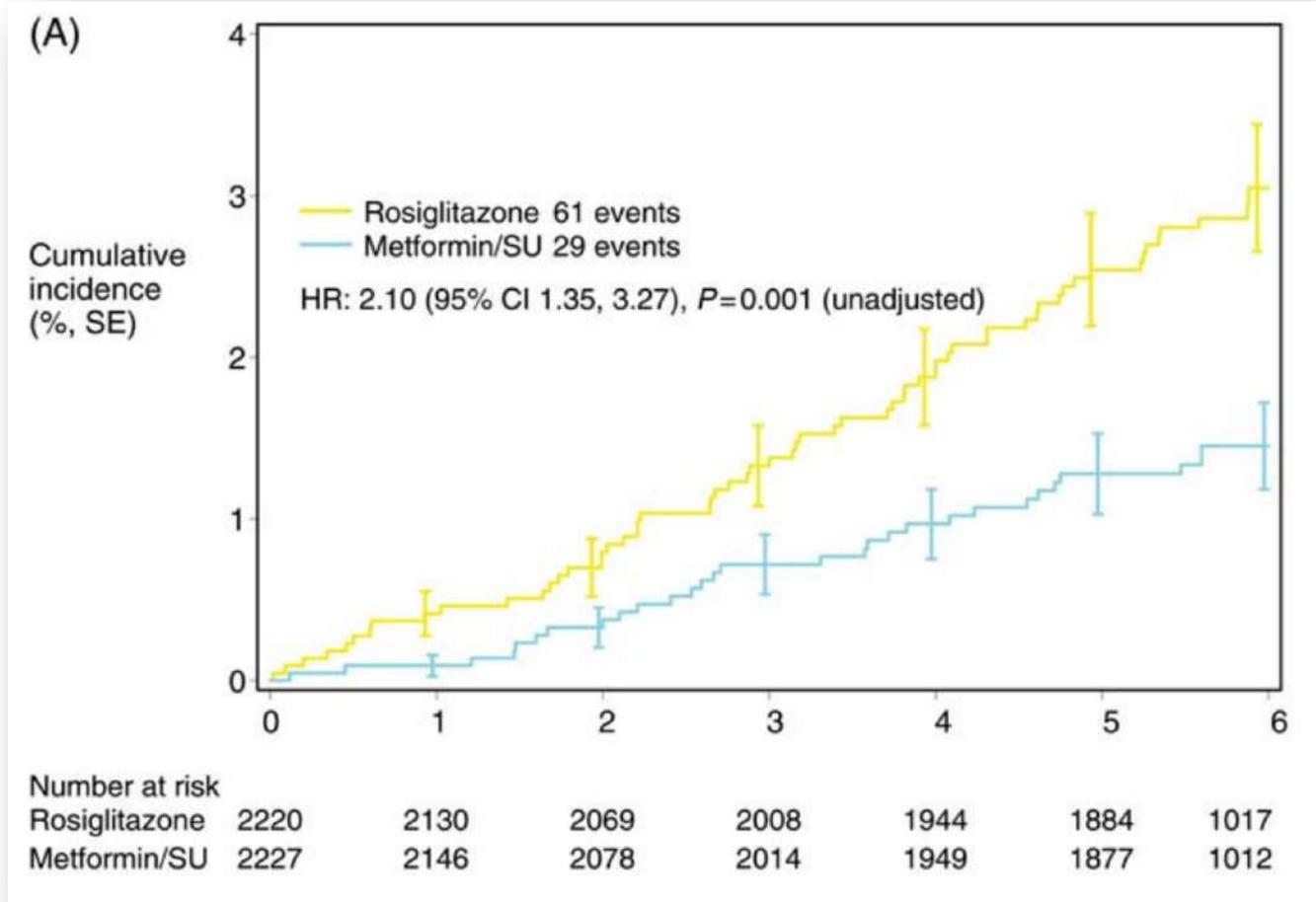
FLASHLIGHTS en



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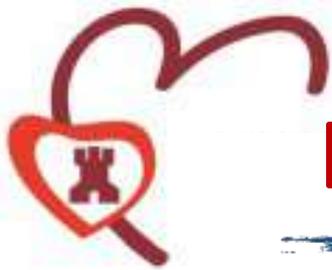
Estudio RECORD



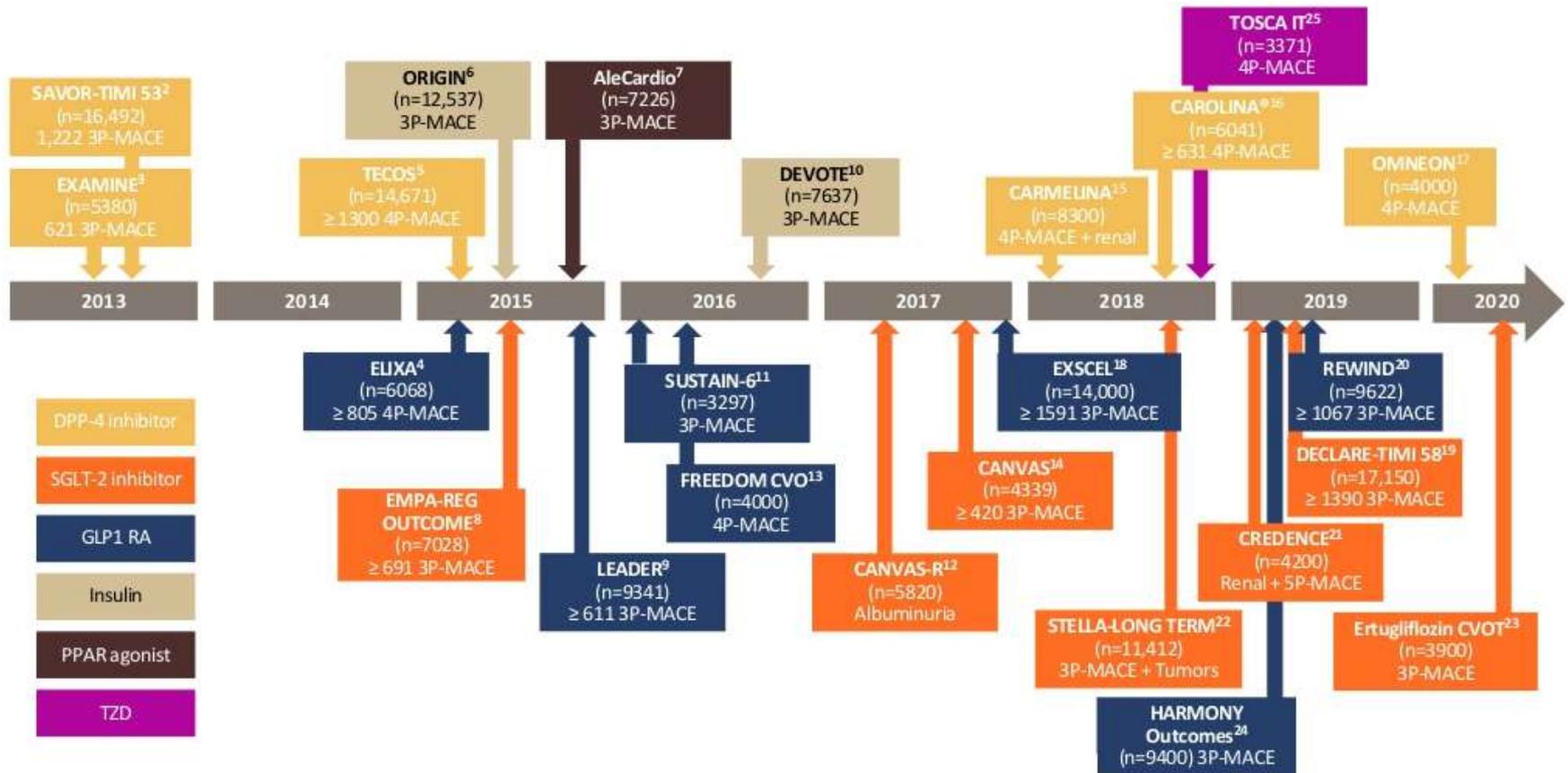
FLASHLIGHTS en



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Estudios de DM con objetivos CV



- Johansen OE. 2015
- Scirica BM et al. 2013
- White WB et al. 2013
- Pfeffer MA et al. 2015
- Green JB et al. 2015

- ORIGIN. 2012
- Lincoff AM et al. 2014
- Zinman B et al. 2015
- Marso SP et al. 2016
- NCT01959529

- NCT01720446
- NCT01989754
- NCT01455896
- NCT01032629
- NCT01897532

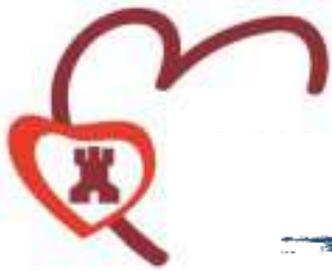
- NCT01243424
- NCT01703208
- NCT01144338
- NCT01730534
- NCT01394952

- NCT02065791
- NCT02479399
- NCT01986881
- NCT02465515
- NCT00700886

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Estudios principales iSGLT2

Empaglif
and
Bernard Zinna
David Fitch
Michaela
Odd Erik Johans
and Silvio E. In

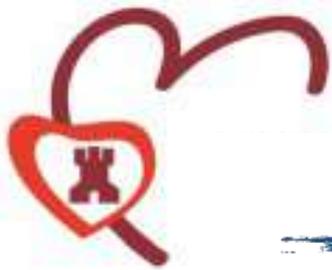


Outcomes
M.G. Silverman,
D.K. McGuire,
P.A. Johansson,
Investigators*
9;380:347-357

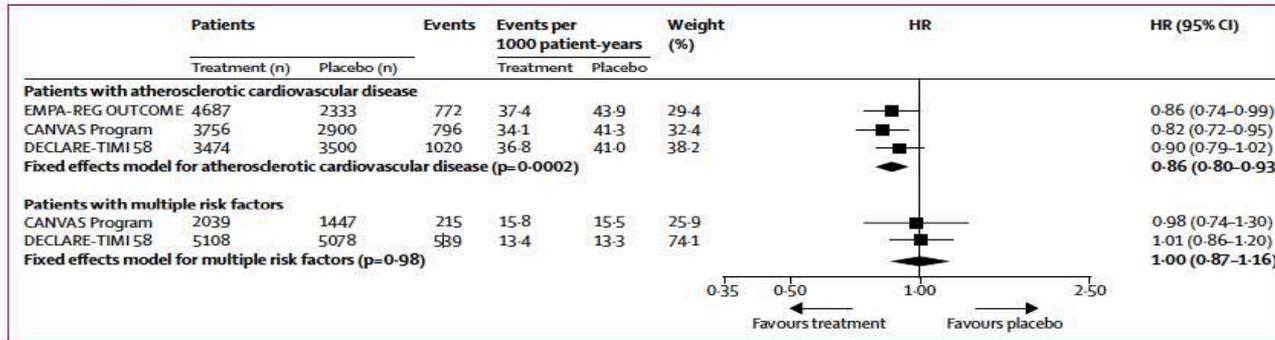
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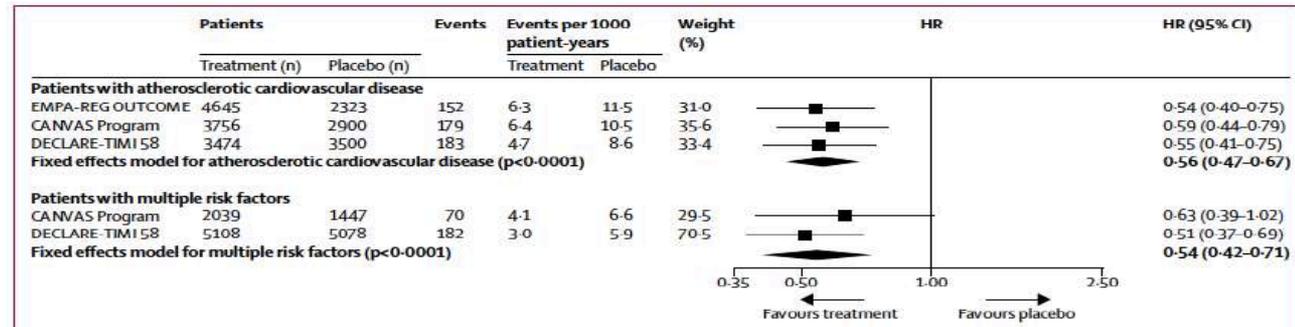


Beneficio de los iSGLT2

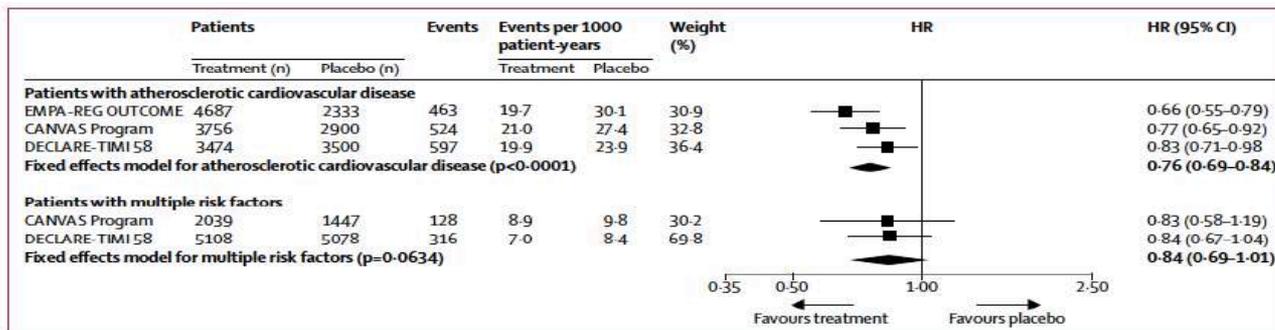


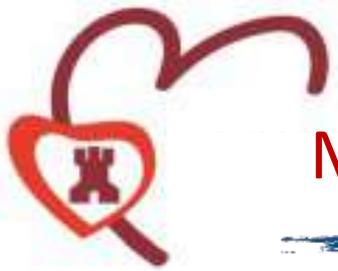
MACE

Hospitalización por IC

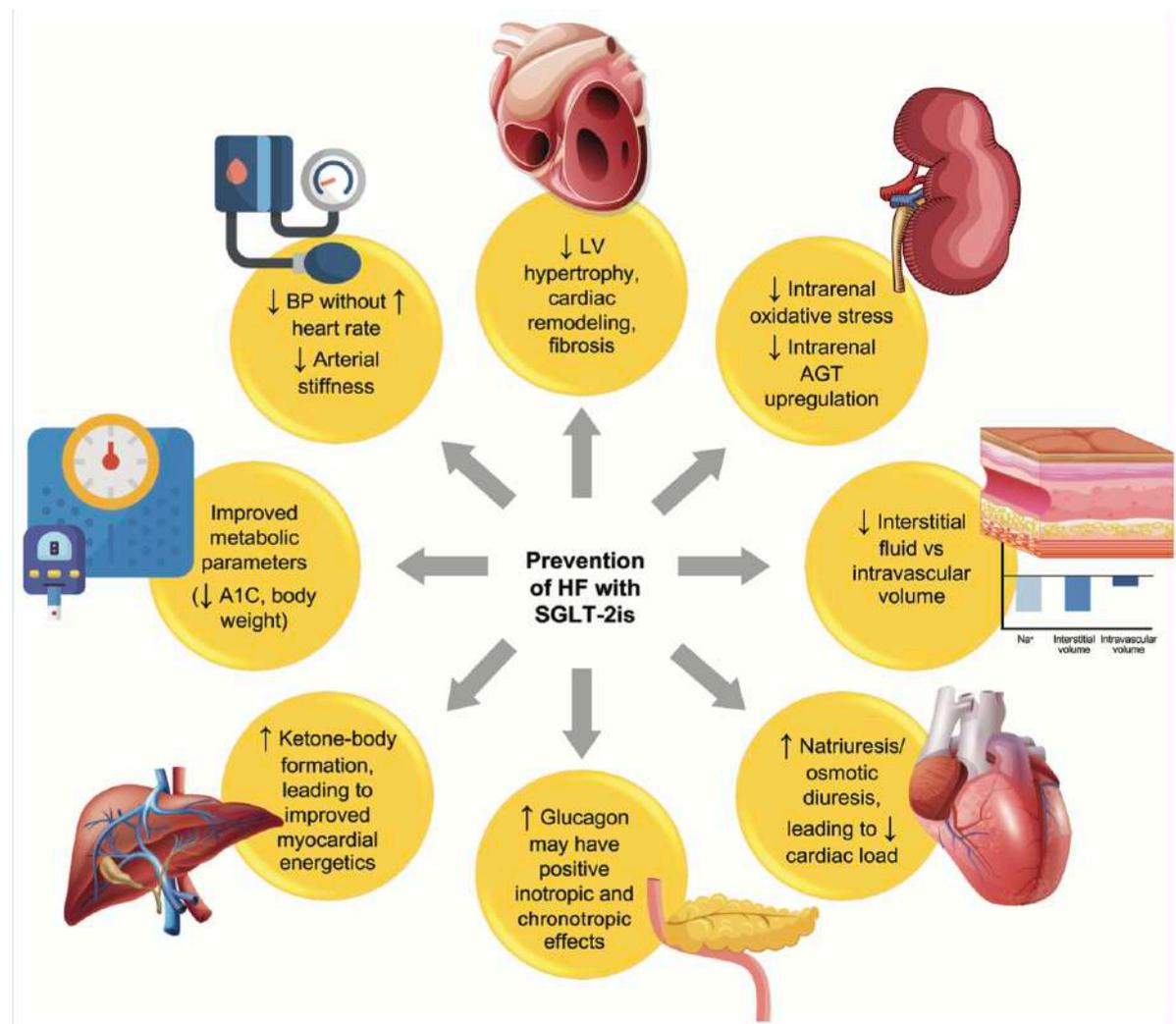


Nefroprotección





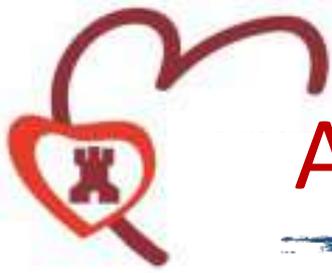
Mecanismos de los iSGLT2 para mejoría de la IC



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**INSUFICIENCIA
CARDIACA**



Actualización en IC 2019 de la ESC

Sodium–glucose co-transporter 2 inhibitors

Consensus recommendation

The 2016 guidelines indicated that empagliflozin *should be considered* in patients with type 2 diabetes mellitus (T2DM) in order to prevent or delay the onset of HF or prolong life.⁸

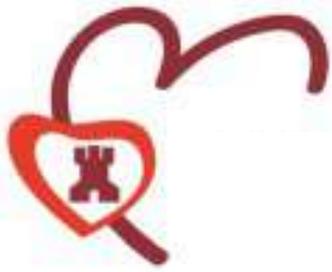
The 2019 expert consensus was that canagliflozin and dapagliflozin *should also be considered* for patients with T2DM and either established cardiovascular (CV) disease or at high CV risk in order to prevent or delay the onset of and hospitalizations for HF.

At this stage, no specific recommendations for the use of sodium–glucose co-transporter 2 (SGLT2) inhibitors in patients with established HF can be made.

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ESC

European Society
of Cardiology

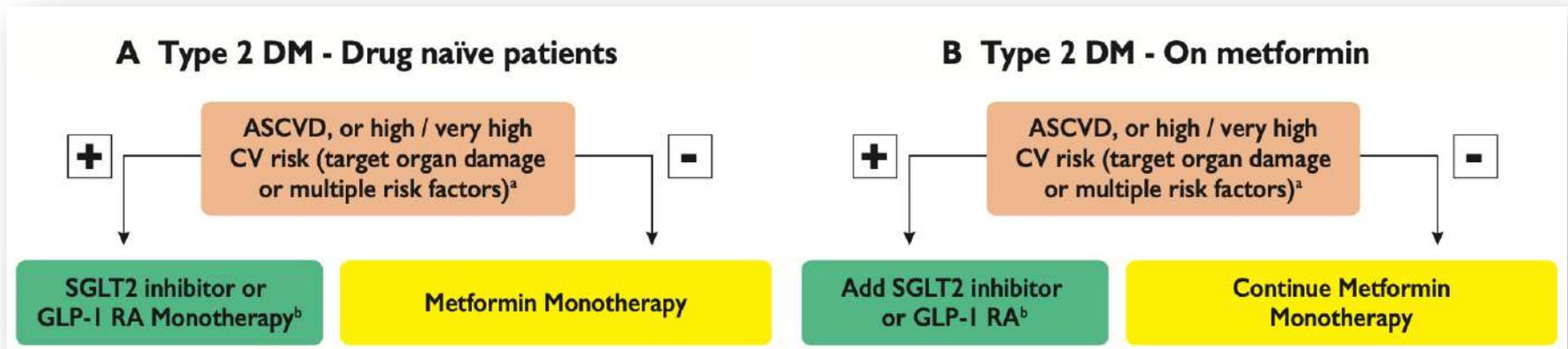
European Heart Journal (2019) 00, 1–69
doi:10.1093/eurheartj/ehz486

ESC GUIDELINES



2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD

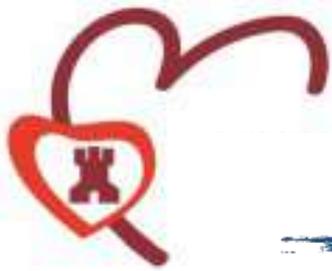
Pacientes con DM 2 y enfermedad CV aterosclerótica, o riesgo CV alto/muy alto



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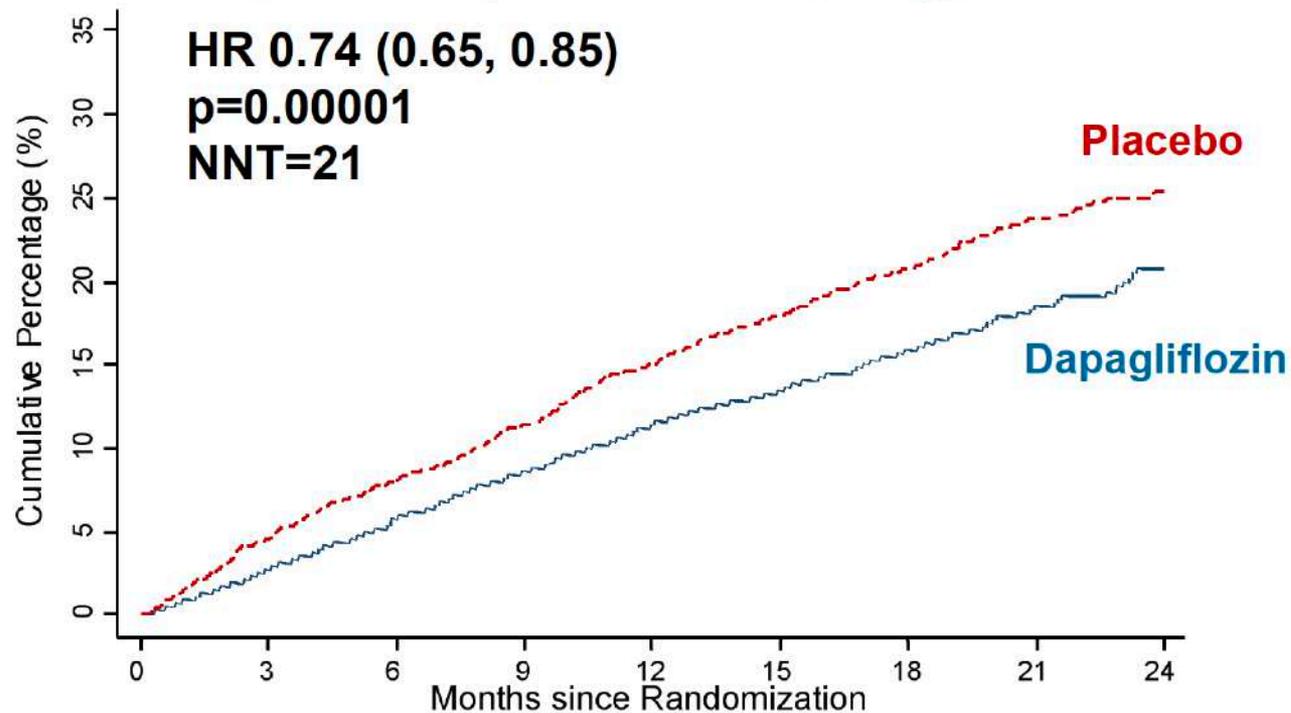


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Estudio DAPA-HF

CV Death/HF hospitalization/Urgent HF visit



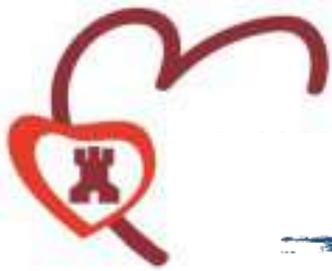
Number at Risk

Dapagliflozin	2373	2305	2221	2147	2002	1560	1146	612	210
Placebo	2371	2258	2163	2075	1917	1478	1096	593	210

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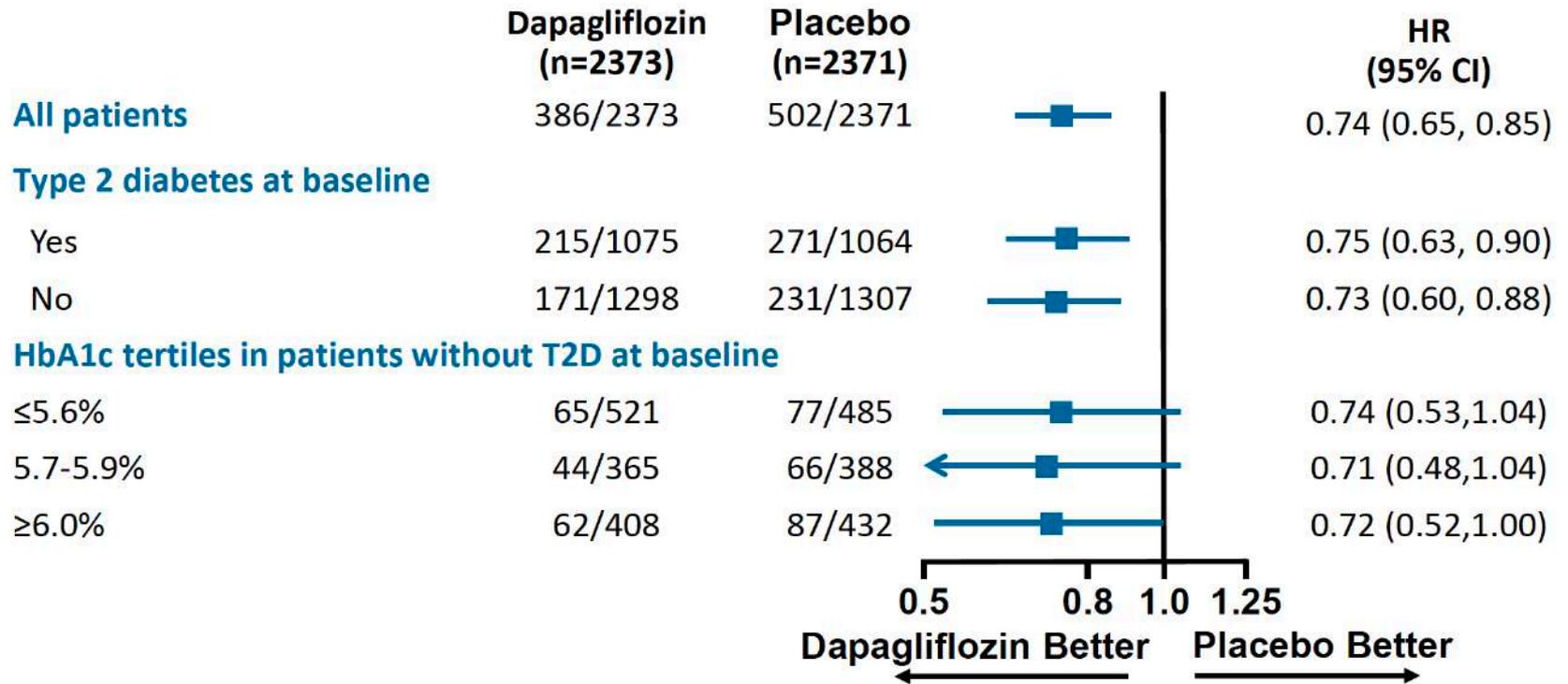


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Estudio DAPA-HF

Primary endpoint



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CCS/CHFS Heart Failure Guidelines: Clinical Trial Update on Functional Mitral Regurgitation, SGLT2 Inhibitors, ARNI in HFpEF, and Tafamidis in Amyloidosis

RECOMMENDATION

5. **Updated.** We recommend SGLT2 inhibitors, such as empagliflozin, canagliflozin or dapagliflozin, be used for treatment of patients with type 2 diabetes and atherosclerotic cardiovascular disease to reduce the risk of HF hospitalization and death (Strong Recommendation, High-Quality Evidence).
6. **New.** We recommend SGLT2 inhibitors, such as dapagliflozin be used in patients with type 2 diabetes aged > 50 years with additional risk factors for atherosclerotic cardiovascular disease to reduce the risk of HFrEF (Strong Recommendation, High-Quality Evidence).
7. **New.** We recommend SGLT2 inhibitors, such as canagliflozin, be used in patients aged > 30 years with type 2 diabetes, and macroalbuminuric renal disease, to reduce the risk of HF hospitalization and progression of renal disease (Strong Recommendation, High-Quality Evidence).
8. **New.** We recommend SGLT2 inhibitors, such as dapagliflozin be used in patients with mild to moderate HF due to reduced LVEF ($\leq 40\%$) and concomitant type 2 diabetes, to improve symptoms and quality of life and to reduce the risk of hospitalization and cardiovascular mortality (Strong Recommendation, High-Quality Evidence).
9. **New.** We recommend SGLT2 inhibitors, such as dapagliflozin be used in patients with mild to moderate HF due to reduced LVEF ($\leq 40\%$) and without concomitant diabetes, to improve symptoms and quality of life and to reduce the risk of hospitalization and cardiovascular mortality (Conditional Recommendation, High-Quality Evidence).

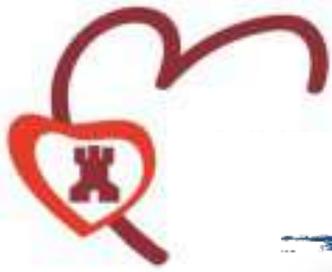
Table 3. Practical issues surrounding initiation of SGLT2 inhibitors

Issue	Concomitant diabetes	No concomitant diabetes
Glycemic control	<ul style="list-style-type: none"> • Collaboration with diabetes team if available • Concomitant insulin or sulfonurea therapy: no adjustment necessary with poorly controlled glucose, consider 25% reduction of each medication • Reinforce glucose monitoring • SGLT2 inhibitors contraindicated in type 1 diabetes • Euvolemia: optional to reduce loop diuretic by 25-50% • Volume overloaded: no need to reduce concomitant loop diuretic • Hypovolemia: do not start until volume depletion corrected 	No concerns for hypoglycemia
Volume control	<ul style="list-style-type: none"> • Safe with eGFR 30 mL/min/1.73 m². Early 20% decrease in eGFR acceptable. With larger change in eGFR, evaluate clinically, consider reduction of loop diuretic 	Same as with diabetes
Renal function	<ul style="list-style-type: none"> • Safe with eGFR 30 mL/min/1.73 m². Early 20% decrease in eGFR acceptable. With larger change in eGFR, evaluate clinically, consider reduction of loop diuretic 	Same as with diabetes
Peripheral vascular disease	Caution with history of amputation or active peripheral arterial ulcer	Caution with history of amputation or active peripheral arterial ulcer
Perineal hygiene	Careful local hygiene—single dose of fluconazole typically effective in event of fungal infection	
Urinary tract infection	<p>SGLT2 inhibitors might lead to increased urinary frequency but not directly associated with infection.</p> <p>However, urinary tract infection might occur independently of SGLT2 inhibitor use, and requires index of suspicion</p>	Same as with diabetes
Diabetic ketoacidosis	<p>As per CDA guidelines, this medication is on the "Sick Day" list. High index of suspicion for DKA required during clinical deterioration. Direct serum anion gap measurement suggested.</p> <p>In addition to volume-depleting conditions, hold for concomitant infection, trauma, surgery, or other major physiologic stressor</p>	<p>Hold during volume depleting intercurrent illness until oral intake adequate</p> <p>DKA not specifically recognized as a risk in nondiabetic patients</p>

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Estudios en marcha de iSGLT2 en IC

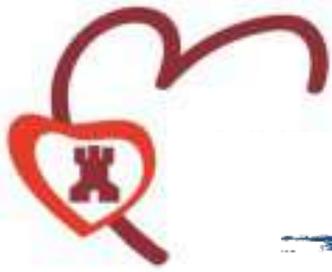
Table 3 Clinical trials of sodium–glucose co-transporter-2 inhibitors in patients with type 2 diabetes, heart failure, chronic kidney disease, and/or atherosclerotic cardiovascular disease

Study name (clinical trial identifier)	Indication	Treatment	No. patients	Primary outcome	Estimated completion date
DAPA-CKD (NCT03036150)	CKD	DAPA vs. PBO	4000 ^a	≥ 50% sustained decline in eGFR, ESRD, CV death, or renal death	November 2020
DELIVER (NCT03619213)	HFpEF	DAPA vs. PBO	4700 ^a	CV death, hospitalisation for HF, or urgent HF visit	June 2021
DETERMINE-Reduced (NCT03877237)	HFrEF	DAPA	300 ^a	Change from baseline in 6MWD at week 16	January 2020
DETERMINE-Preserved (NCT03877224)	HFpEF	DAPA	400 ^a	Change from baseline in 6MWD at week 16	February 2020
EMPEROR-Reduced (NCT03057977)	HFrEF	EMPA vs. PBO	2850 ^a	CV death or hospitalisation for HF	June 2020
EMPEROR-Preserved (NCT03057951)	HFpEF	EMPA vs. PBO	4126 ^a	CV death or hospitalisation for HF	June 2020
SOLOIST-WHF (NCT03521934)	T2D + WHF	SOTA vs. PBO	4000 ^a	CV death or hospitalisation for HF	January 2021
MUSCAT-HF (UMIN000018395)	T2D + HFpEF	LUSEO vs. AGI	190	Change from baseline BNP level at week 12	NA
EMPERIAL-Reduced (NCT03448419)	HFrEF	EMPA vs. PBO	300 ^a	Change in exercise capacity	December 2019
EMPERIAL-Preserved (NCT03448406)	HFpEF	EMPA vs. PBO	300 ^a	Change in exercise capacity	December 2019

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Conclusiones

- La IC y la DM son enfermedades altamente prevalentes e íntimamente relacionadas
- El manejo de la IC puede mejorar la DM
- El tratamiento de la DM con iSGLT2 previene a aparición de IC
- El tratamiento de la IC con FEr con iSGLT2 (al menos con dapaglifozina) mejora el pronóstico tanto de pacientes con DM como sin DM
- Mucha evidencia por llegar en relación a los iSGLT2 en relación con la IC

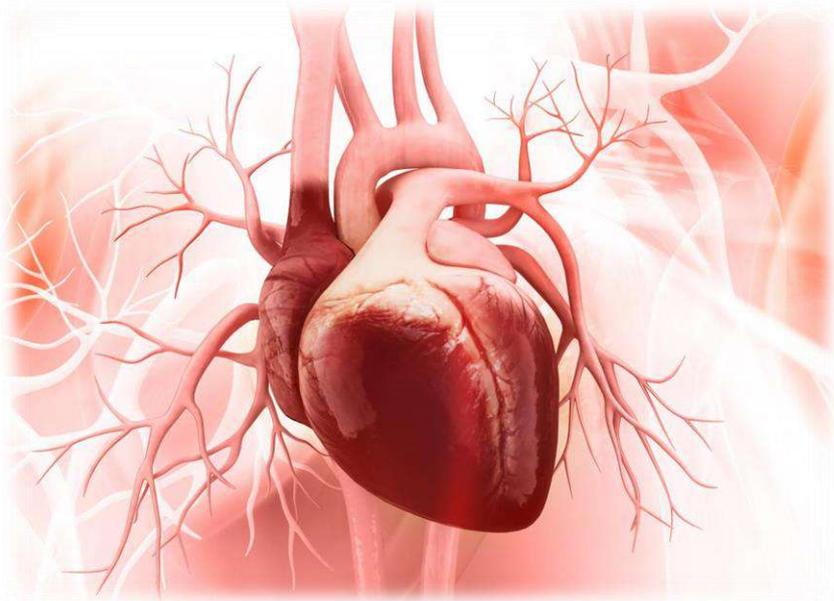
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Trucos para el tratamiento de las alteraciones hidroelectrolíticas

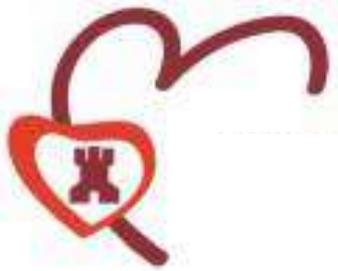


Dr. Jesús Casado

Unidad Funcional Multidisciplinar de
Insuficiencia Cardíaca

Servicio de Medicina Interna

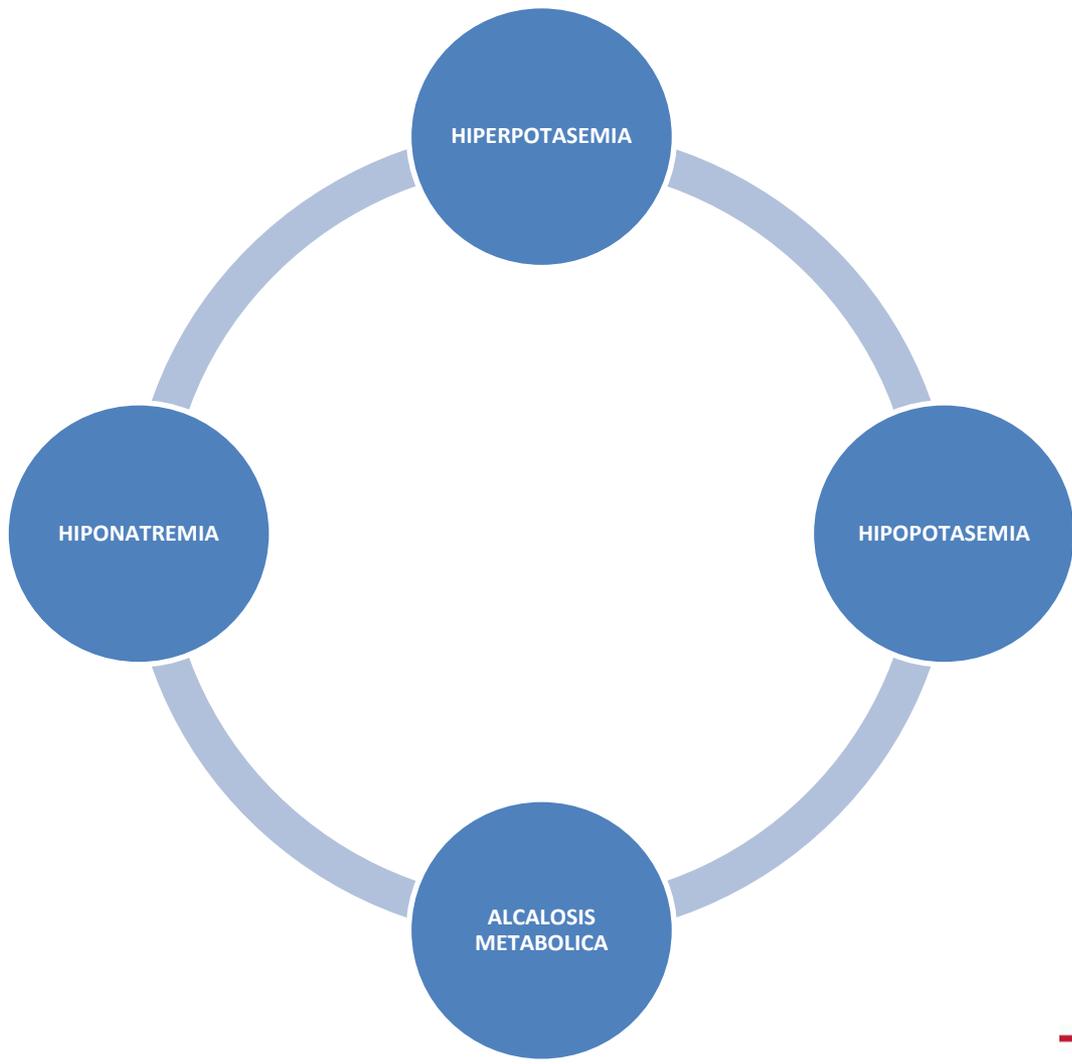
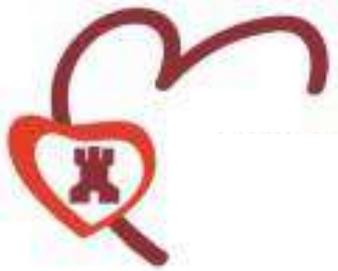
Hospital de Getafe



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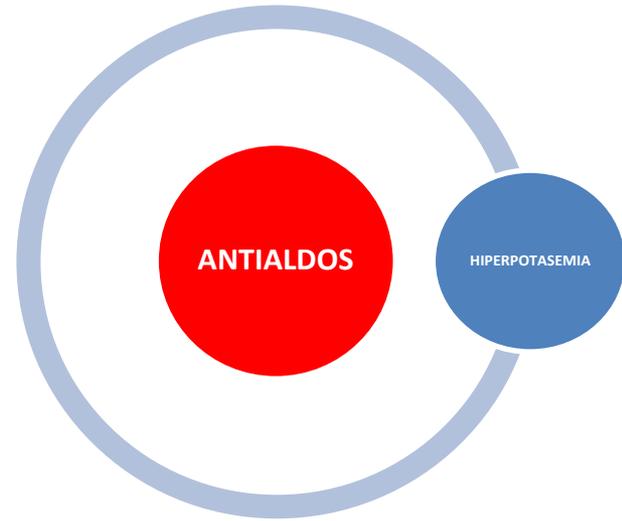
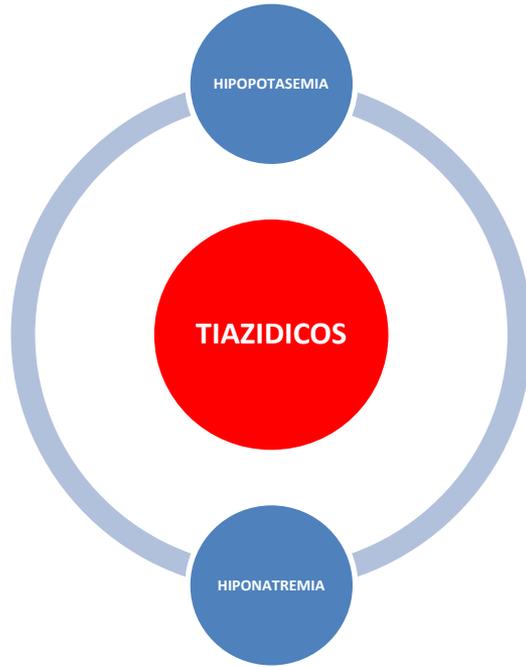
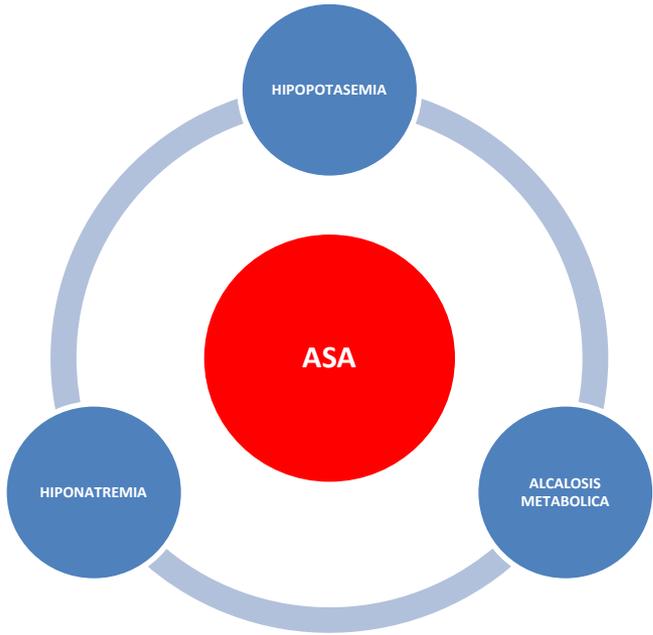
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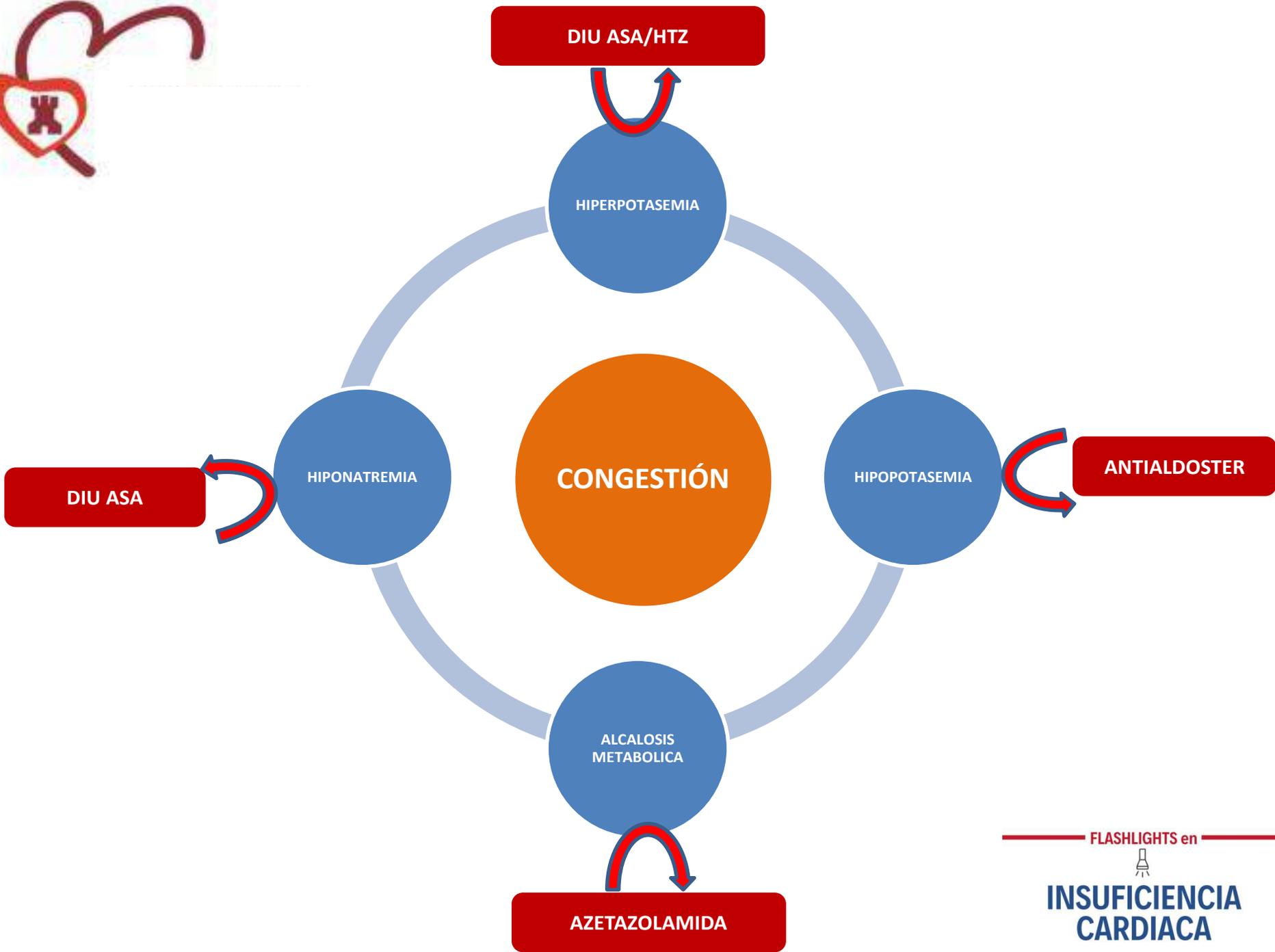
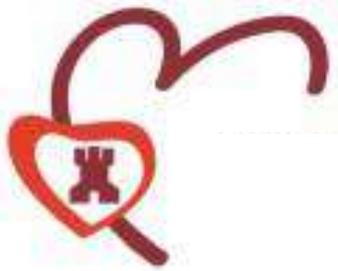
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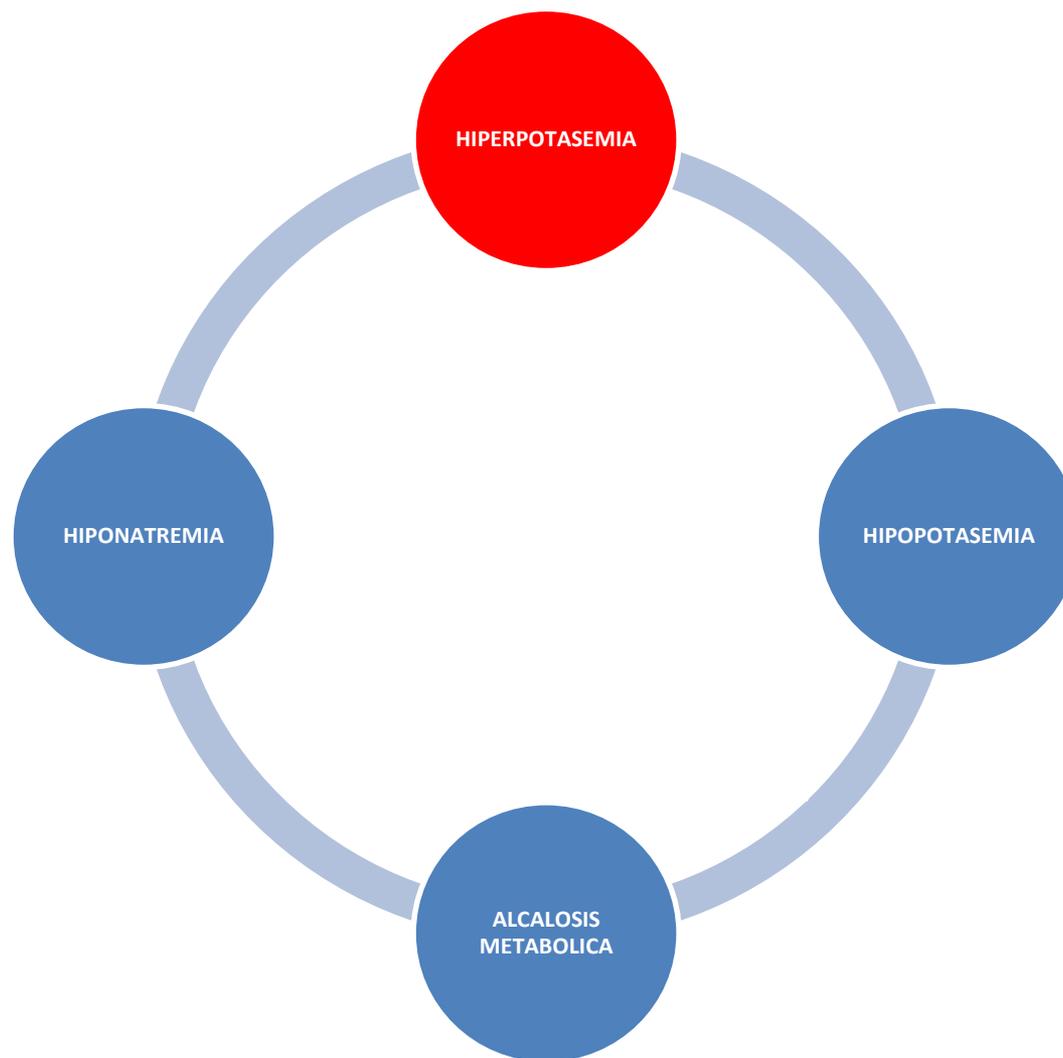
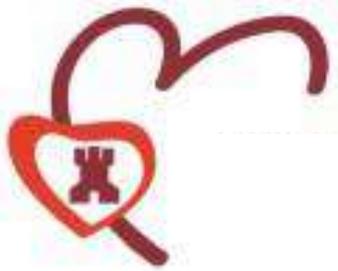


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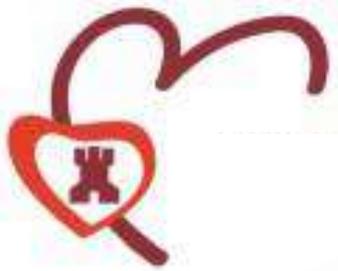




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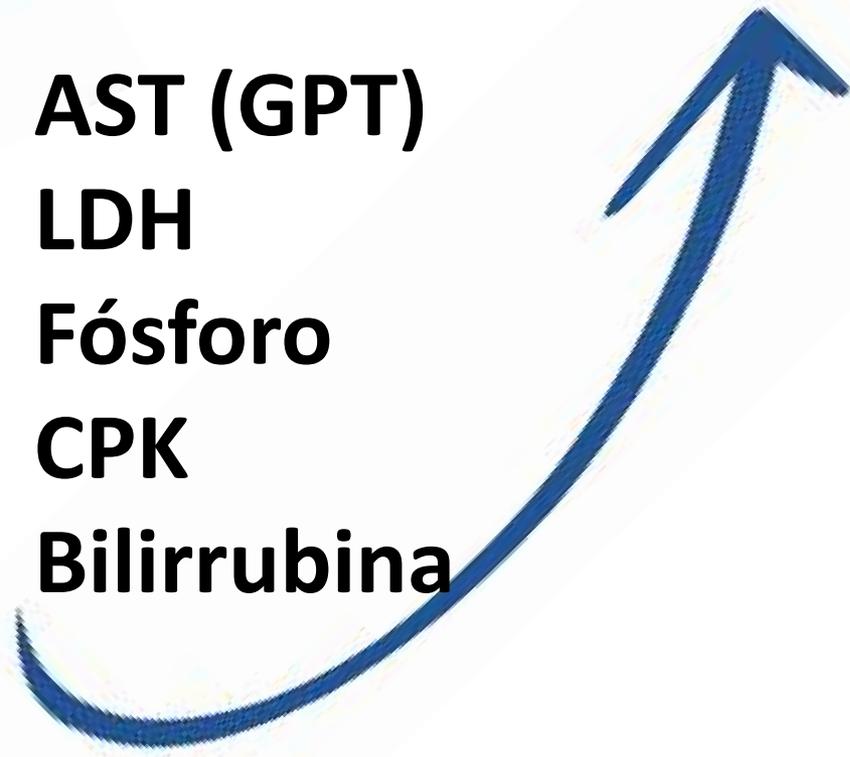


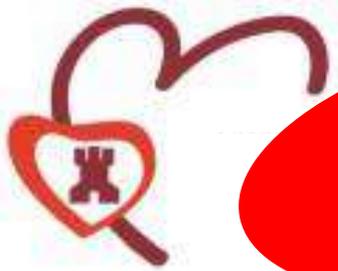
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FAKE

AST (GPT)
LDH
Fósforo
CPK
Bilirrubina

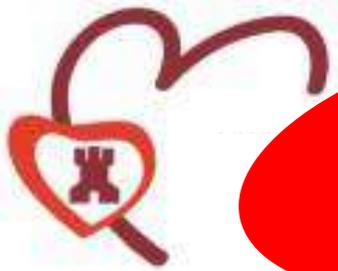




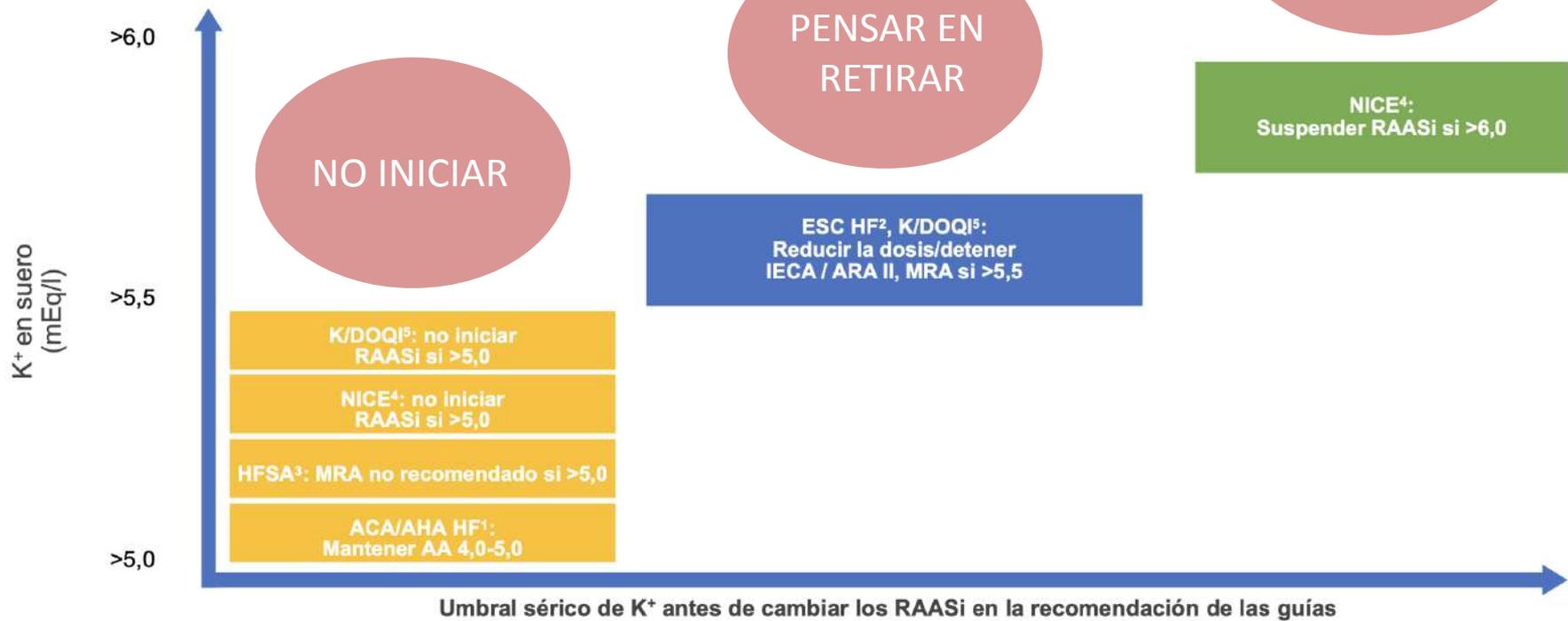
HIPERPOTASEMIA AGUDA



- Paso 1: proteger el corazón
- Paso 2: desplazar el k al interior celular
- Paso 3: eliminar el K del cuerpo
- Paso 4: monitorizar K y glucosa
- Paso 5: prevenir recurrencias



HIPERPOTASEMIA CRÓNICA





HIPERPOTASEMIA CRÓNICA

RESINCALCIO

- Gran uso, poca evidencia
- Mal tolerados
- Asociar laxantes
- Bajo cumplimiento

PATIROMER

- Separar 3h de otros F.
- Con o sin alimentos
- Polvo para suspensión oral
- No tiene sabor

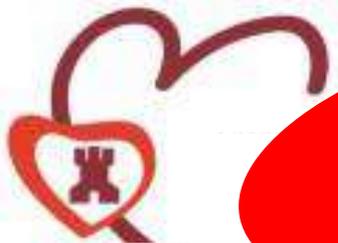
CICLOSILATO DE ZIRCONIO Y SODIO

- No disponible
- No evidencia en IC

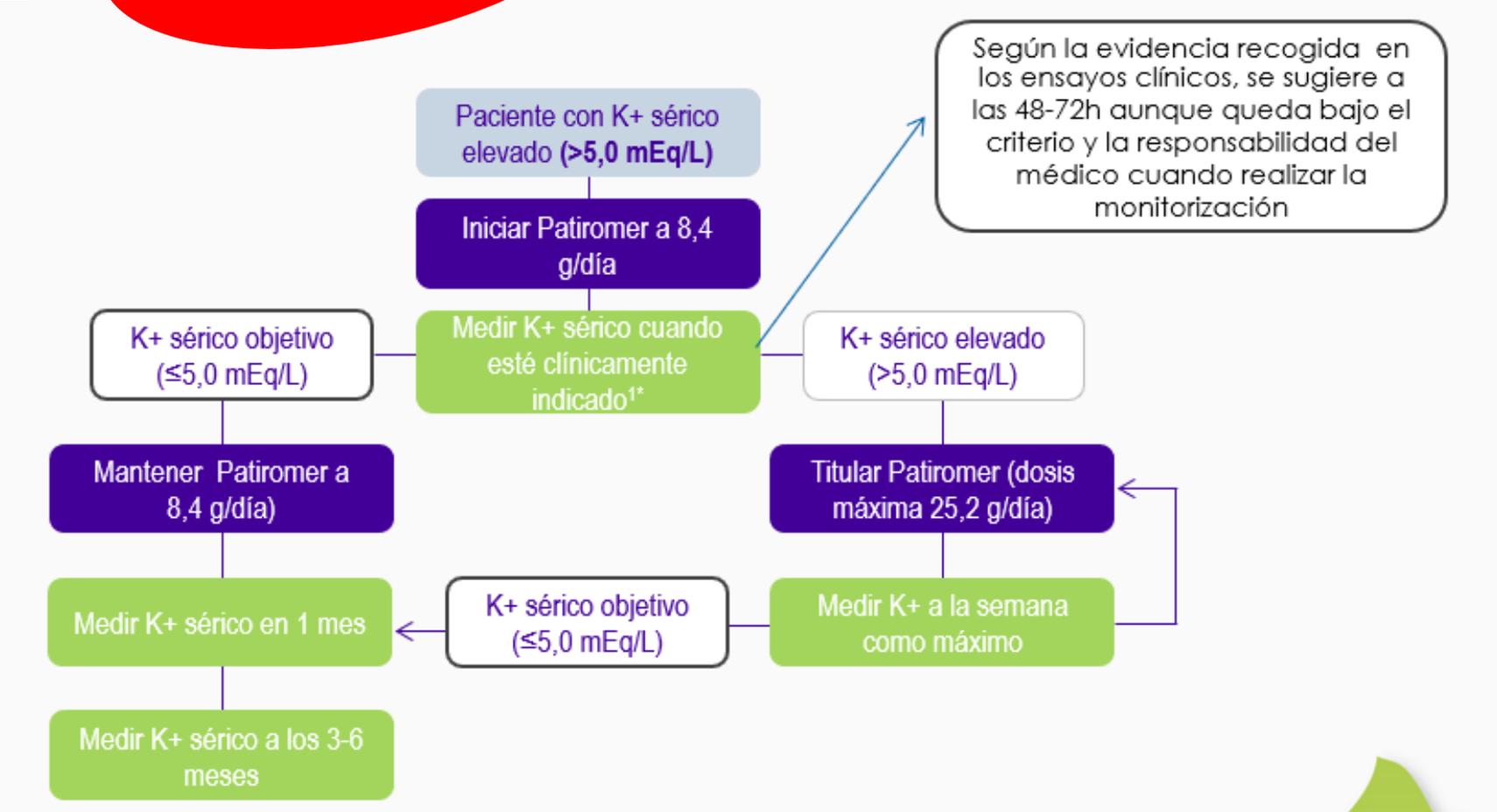
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HIPERPOTASEMIA CRÓNICA





Condiciones financiación (Patiromer, veltassa®) (IPT)

Medicamento con receta médica, condicionado a visado

➤ **Condiciones de visado:**

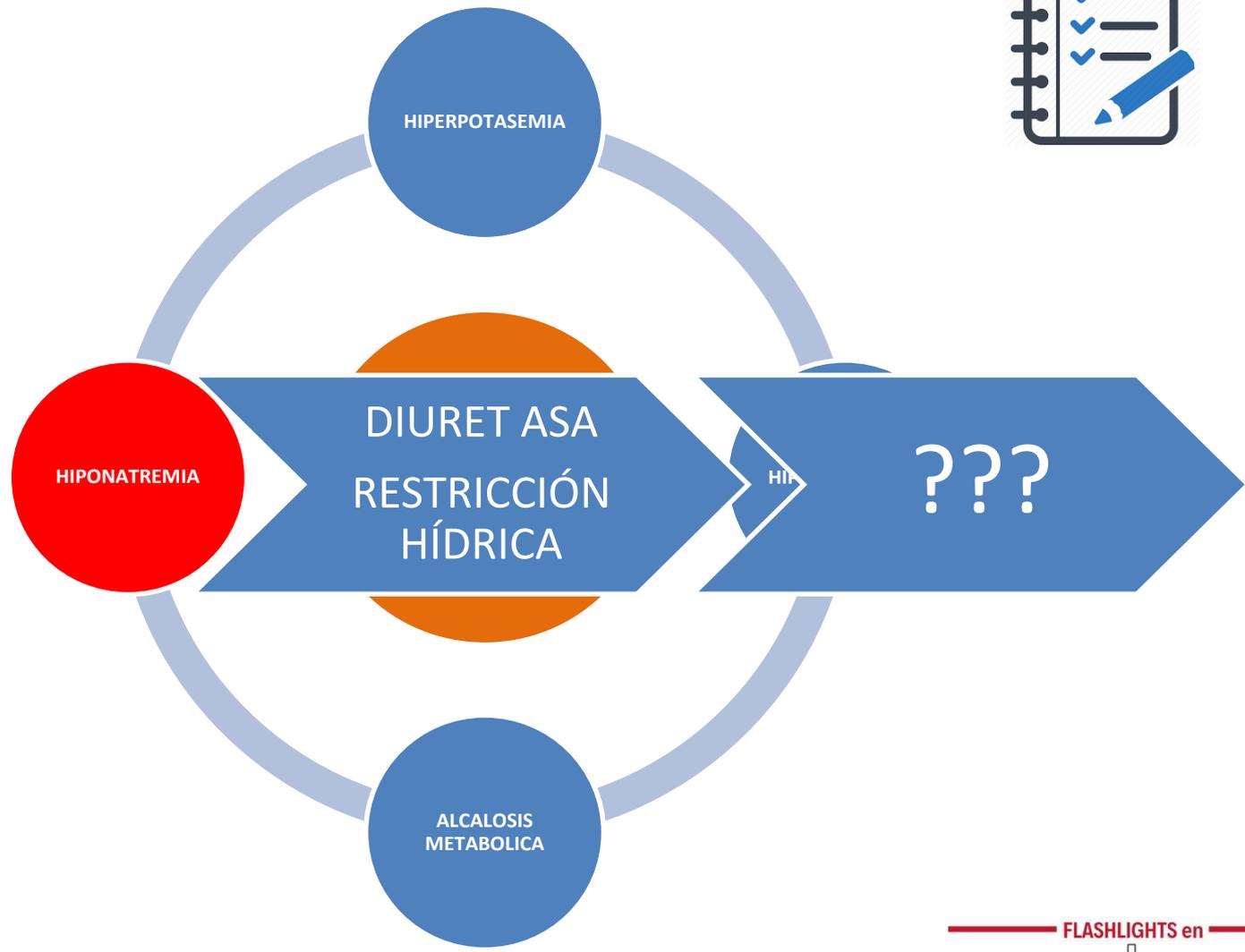
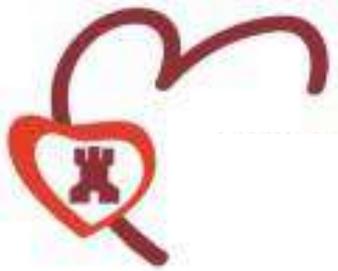
- Para ser prescrito por especialistas en Nefrología, Cardiología y Medicina Interna
- A pacientes con ERC avanzada e insuficiencia cardíaca grado III-IV y con hiperpotasemia leve a moderada (5,5-6,4 mmol/L)
- En tratamiento con inhibidores del SRAA y en los que se considere imprescindible su continuación,
- Y con fracaso o intolerancia a resinas de intercambio iónico.

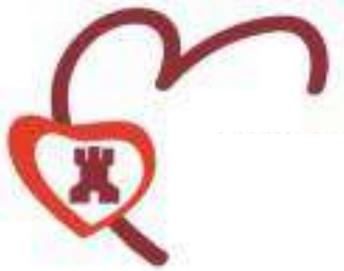
➤ **Con aportación reducida**

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◦PROTOCOLO DE SUERO SALINO HIPERTÓNICO:

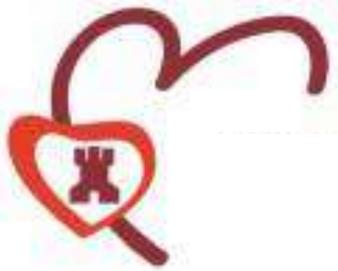
- Furosemida: 250-500 mgr/12h/iv en 30 minutos
- BoiK aspártico: 2 comprimidos cada 8 horas
- Restricción hídrica de 1.000 cc diarios
- Función renal e ionograma diario
- 150 cc SSH/12h/iv en 30 min. Concentración de Na⁺ en la infusión según natremia

Concentración de la infusión	Niveles de Na ⁺ en sangre	Forma de preparación
Suero salino hipertónico al 4,6%	<125 mEq/L	Diluir 19 ampollas de ClNa 20% en 1.000 ml de suero salino 0,9% y administrar 150 ml de la dilución en 30 minutos/12 horas
Suero salino hipertónico al 3,5%	126-135 mEq/L	Diluir 14 ampollas de ClNa 20% en 1.000 ml de suero salino 0,9% y administrar 150 ml de la dilución en 30 minutos/12 horas
Suero salino hipertónico al 2,4%	136-145 mEq/L	Diluir 8 ampollas de ClNa 20% en 1.000 ml de suero salino 0,9% y administrar 150 ml de la dilución en 30 minutos/12 horas
Suero salino hipertónico al 1,4%	>145 mEq/L	Diluir 3 ampollas de ClNa 20% en 1.000 ml de suero salino 0,9% y administrar 150 ml de la dilución en 30 minutos/12 horas

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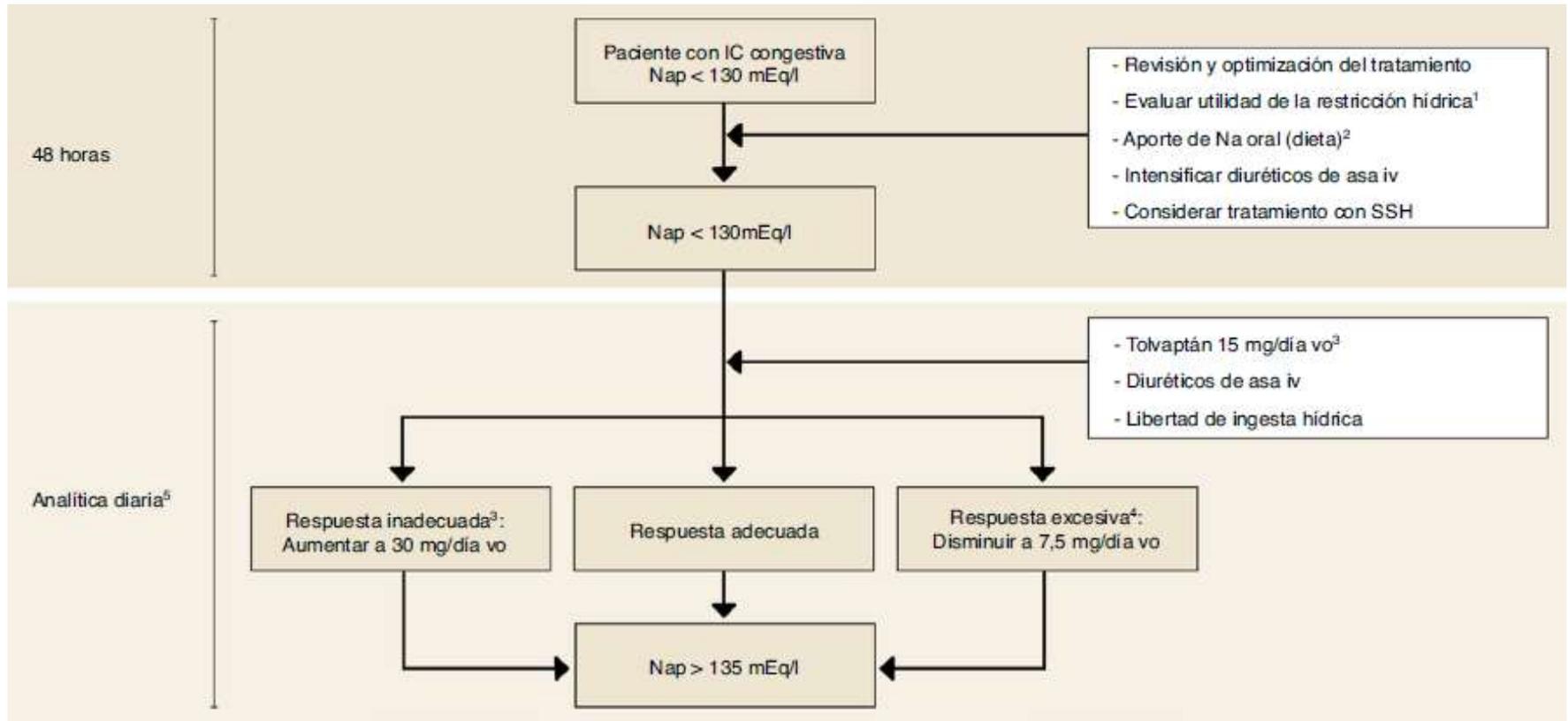


ARTÍCULO ESPECIAL

Hiponatremia e insuficiencia cardiaca congestiva refractaria a tratamiento diurético. Utilidad del tolvaptán

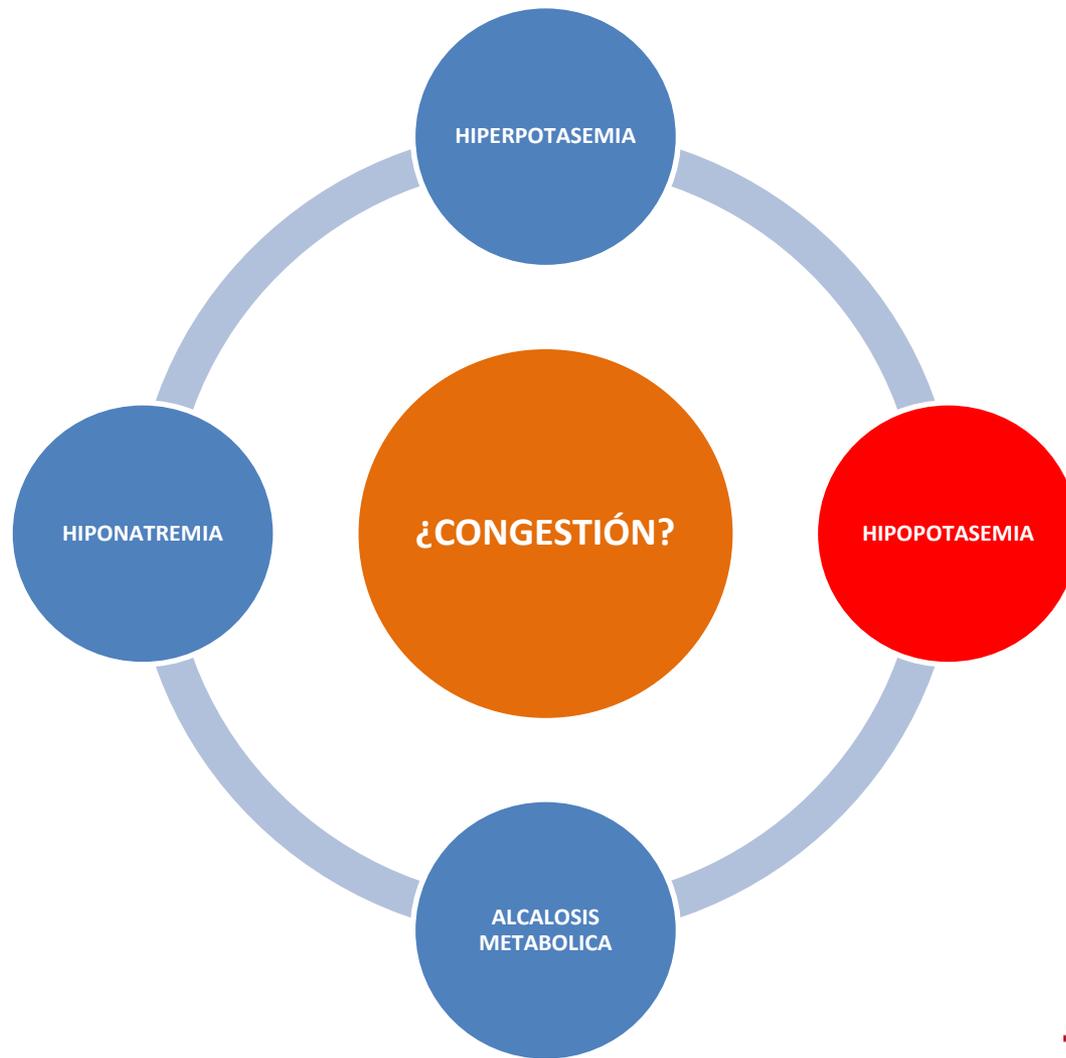
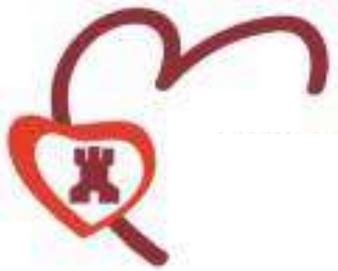


A. Pose^{a,*}, L. Almenar^b, L. Manzano^c, J.J. Gavira^d, A. López Granados^e,
J. Delgado^f, O. Aramburu^g, J.C. Arévalo^h, M. Méndezⁱ, J. Comín^j y N. Manito^k



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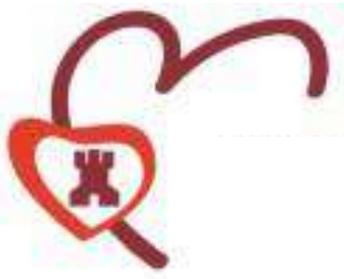




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- ✓ Grave $<2,5....$
- ✓ Vigilar Magnesio
- ✓ Alcalosis metabólica

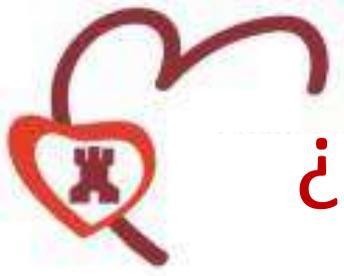


$K_u < 15 \text{ mEq/L}$



$K_u > 15 \text{ mEq/L}$

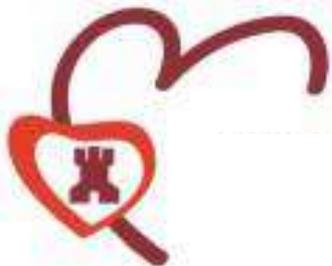




¿Administrar potasio siempre que se paute diurético?

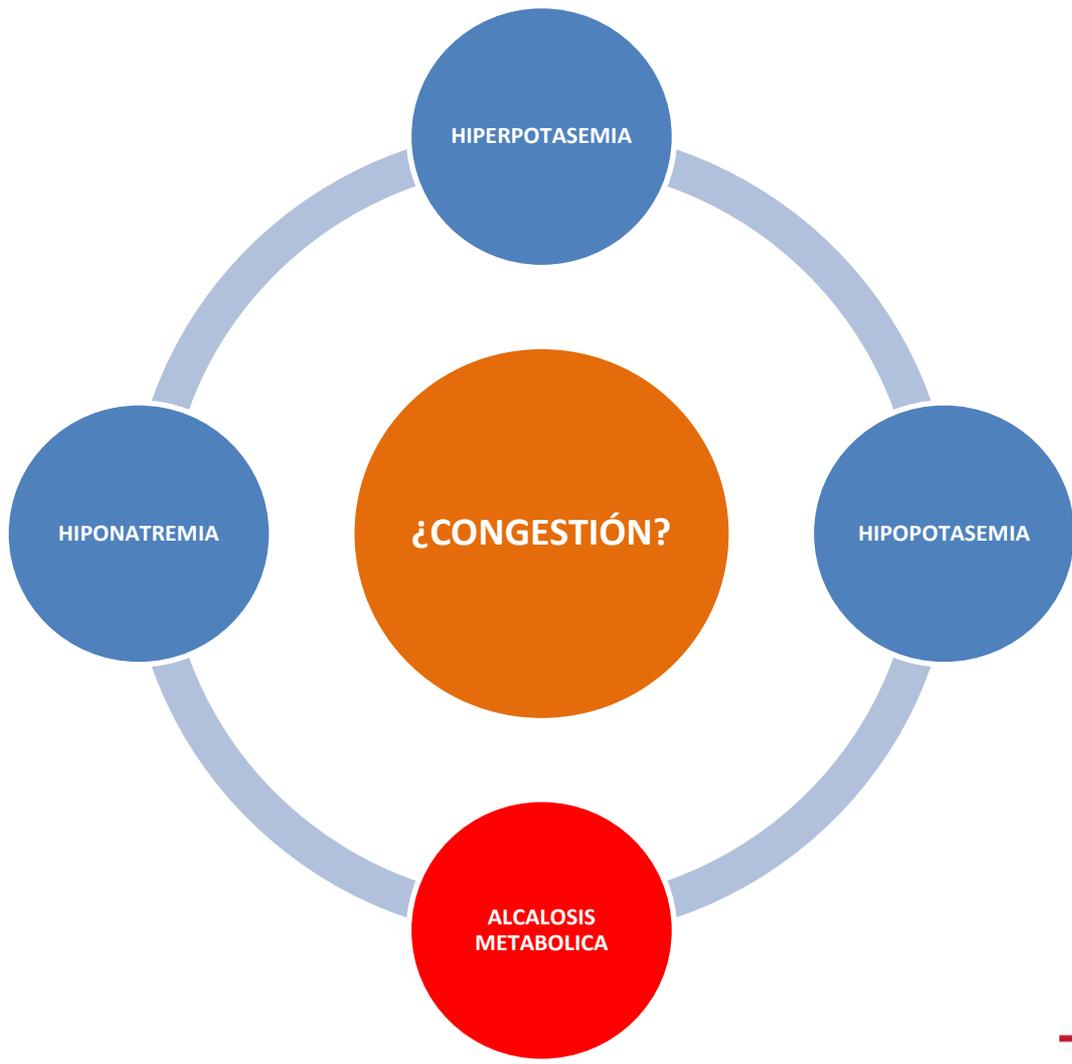
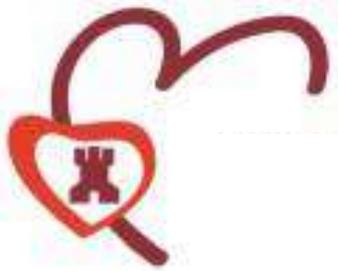
- **IR:** NO, salvo dosis elevadas de diurét. de asa
- **No IR:**
 - Tiazidas > 50 mg/d
 - Furosemida > 80 mg/d o Torasemida > 10 mg/d
 - Terapia combinada

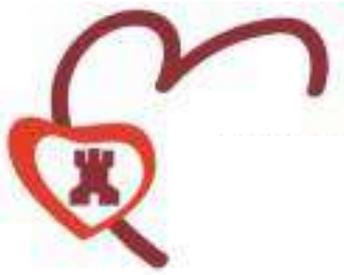




Ascorbato de potasio:
Boi-K®: 1 c contiene 10 mEq de K
Boi-K aspártico®: 1 c contiene 25 mEq de K
Gluconato potásico (No disponible en España, pero sí en EEUU y Sudamérica en varias formulaciones)
Kaon®, Boi-K gluconato ®: 1 sobre contiene 20 mEq de K
Cloruro de potasio:
Potación® 600 mg cápsulas: 1c contiene 8 mEq de K
Glucoheptonato de potasio:
Potación® solución: 1 ml contienen 1 mEq de K
Citrato potásico
Acalka®: 1 c contiene 10 mEq de K (utilizado para alcalinizar orina)
Lactato de magnesio
Magnesioboi® los c de 5 y 10 mg contienen 20 y 40 mEq
Magnesio NM® c y sobres: 8.7 y 15 mEq
Pidolato de magnesio
Actimag® 2G/5ml solución: 5 ml contienen 14,3 mEq







Article

Acetazolamide Therapy in Patients with Heart Failure: A Meta-Analysis

Acetazolamida en IC

9 estudios (3 ensayos y 6 cohortes)

N = 229

- Descenso de pH, pCO₂ y HCO₃
- Incremento de la natriuresis
- Mejora de los índices de apnea



European Journal of Heart Failure (2018) 20, 1591–1600
doi:10.1002/ehf.1307

STUDY DESIGN

Rationale and design of the ADVOR (Acetazolamide in Decompensated Heart Failure with Volume Overload) trial

Wilfried Mullens^{1,2*}, Frederik H. Verbrugge¹, Petra Nijst¹, Pieter Martens¹,
Katrien Tartaglia¹, Evi Theunissen¹, Liesbeth Bruckers², Walter Droogne³,
Pierre Troisfontaines⁴, Kevin Damman⁵, Johan Lassus⁶, Alexandre Mebazaa⁷,
Gerassimos Filippatos⁸, Frank Ruschitzka⁹, and Matthias Dupont¹

¹Ziekenhuis Oost-Limburg, Genk, Belgium; ²Hasselt University, Diepenbeek/Hasselt, Belgium; ³University Hospitals Leuven, Leuven, Belgium; ⁴CHR Citadelle Hospital, Liège, Belgium; ⁵University Medical Center Groningen, Groningen, The Netherlands; ⁶Helsinki University Central Hospital, Helsinki, Finland; ⁷University of Paris Diderot, Paris, France; ⁸National and Kapodistrian University of Athens, Athens, Greece; and ⁹UniversitätsSpital Zurich, Zurich, Switzerland

Received 2 June 2018; revised 11 July 2018; accepted 2 August 2018; online published ahead of print 21 September 2018

Methods

The ADVOR trial is set up as a multicentre, randomized, double-blind, placebo-controlled study, aiming to recruit 519 patients with AHF and clinically evident volume overload. All study participants receive high-dose intravenous loop diuretics as background therapy and are randomized towards intravenous acetazolamide at a dose of 500 mg once daily vs. placebo, stratified according to including study centre and ejection fraction (< 40% vs. ≥ 40%). The primary endpoint is successful decongestion with no more than trace oedema assessed on the third morning after hospital admission, with good diuretic efficacy defined as a urine output > 3.5 L during the first 30–48 h of decongestive treatment. Secondary endpoints include all-cause mortality or heart failure readmission after 3 months, length of hospital stay for the index admission, and longitudinal changes in the EuroQol-5 dimensions questionnaire.

Wongboonsin J, et al. Acetazolamide Therapy in Patients with Heart Failure: A Meta-Analysis. J Clin Med. 2019;8:349

Mullens W, et al. Rationale and design of the ADVOR trial. Eur J Heart Fail. 2018;20:1591–1600

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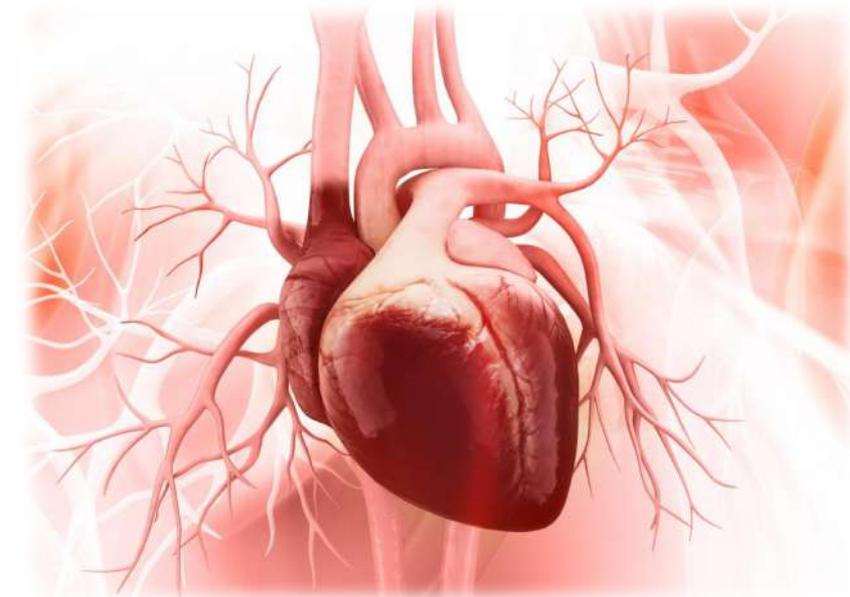


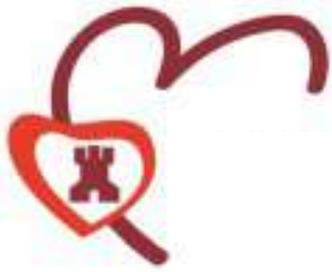
Muchas gracias



Manejo de la resistencia a diuréticos

Susana del Prado
Unidad de Insuficiencia Cardiaca
Hospital Universitario Ramón y Cajal





Manejo de la resistencia a diuréticos

Deterioro de la sensibilidad a diuréticos



natriuresis y diuresis reducida



limitando la euvolemia



Persistencia de la congestión
 pese a dosis crecientes de
 diuréticos

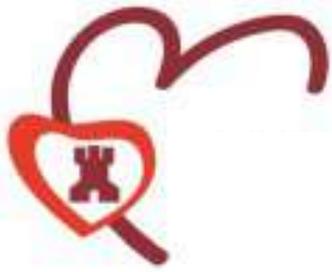
Ausencia de pérdida de peso
 con tto iv

EF Na < 0,2%

Necesidad de dosis altas de diurético
 (>furosemida 80mg)

Ausencia de balance negativo pese
 a tratamiento iv





Manejo de la resistencia a diuréticos

¿Cómo evaluar la respuesta a diuréticos? Eficacia diurética

- Datos de descongestión:
 - Persistencia de signos y síntomas de congestión pese a incrementar la dosis de diurético
- Diuresis/pérdida de peso:
 - Ausencia de pérdida de peso o balance negativo a pesar de diurético iv
 - <0.4 kg/dosis diurética (40 mg furosemida)
 - <0.5l/dosis diurética

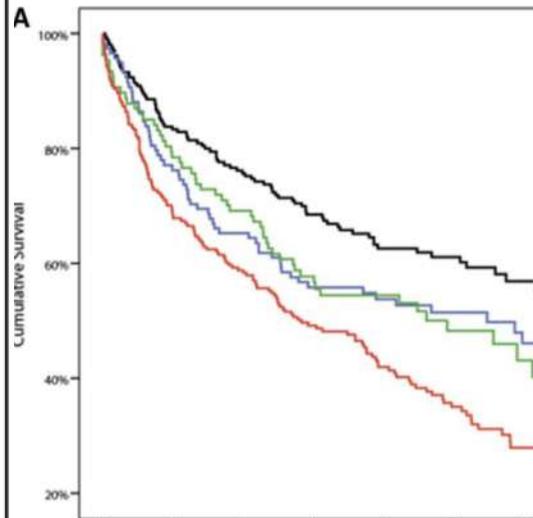
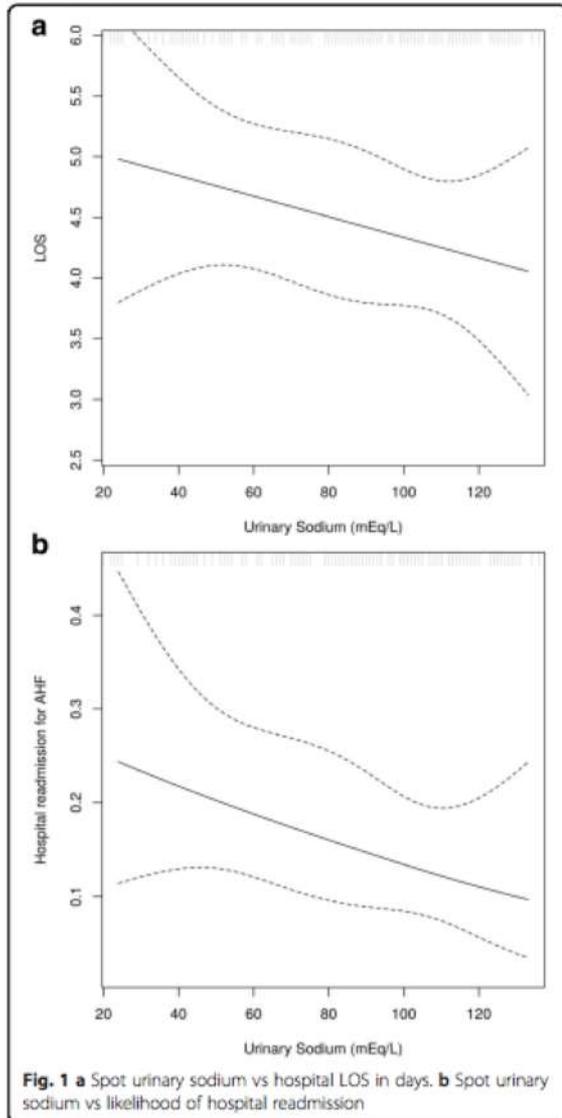
– Na orina

Table 1. Metrics of diuretic efficacy/resistance

Metric ^a	Convenience	Accuracy	Plausibility	Volume status dependency
Weight loss	+++	+++	+	+++
Net fluid loss	+	+	++	+++
Urine output	++	++	++	+
Natriuresis	+	+++	+++	++

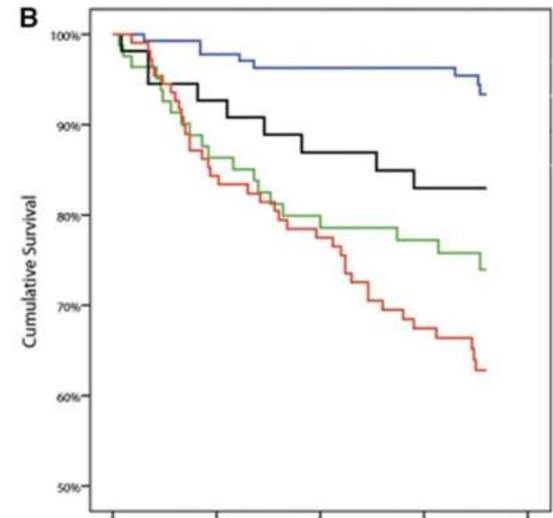
^aPer 40 mg of intravenous furosemide-equivalent dose

Manejo de la resistencia a diuréticos



Time to Death (Years)							
211	175	159	135	95	71	38	
118	90	77	62	51	35	25	
107	84	74	53	44	26	15	
221	151	130	100	71	50	25	
Number at risk							

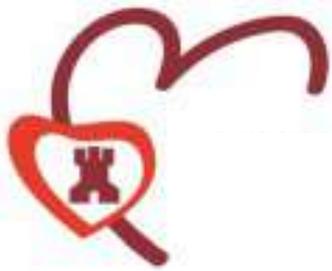
- Low Loop Dose / High Efficiency
- Low Loop Dose / Low Efficiency
- High Loop Dose / High Efficiency
- High Loop Dose / Low Efficiency



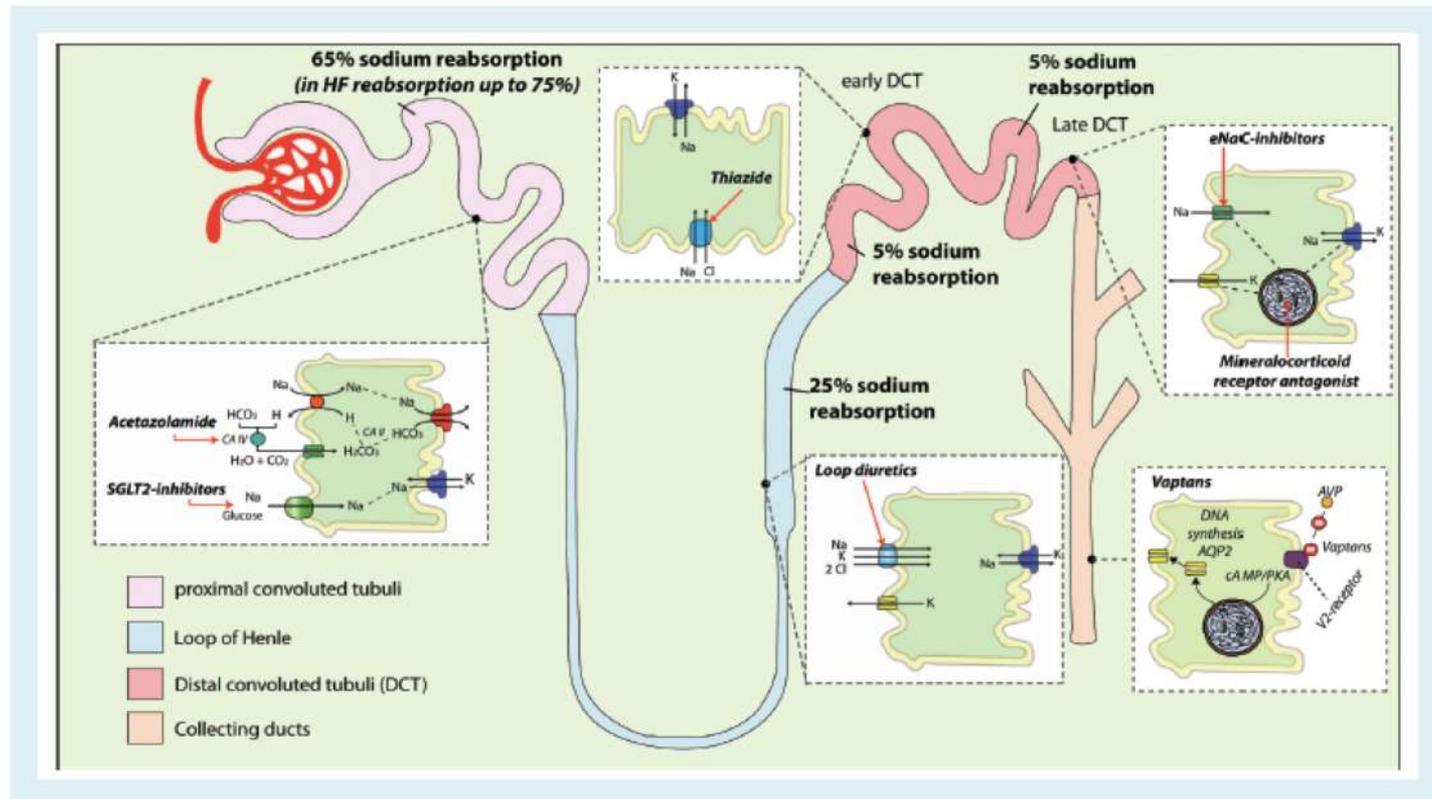
Time to Death (Days)				
140	133	126	123	
83	69	61	55	
55	50	44	42	
112	89	79	65	
Number at risk				

- Low Loop Dose / High Efficiency
- Low Loop Dose / Low Efficiency
- High Loop Dose / High Efficiency
- High Loop Dose / Low Efficiency

Doering A et al. *Int J Emerg Med.* 2017 Dec;10(1):17
 Testani JM et al. *Circ Heart Fail.* 2014;7:261-270



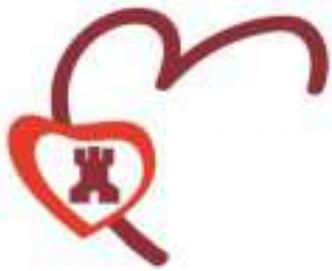
Manejo de la resistencia a diuréticos



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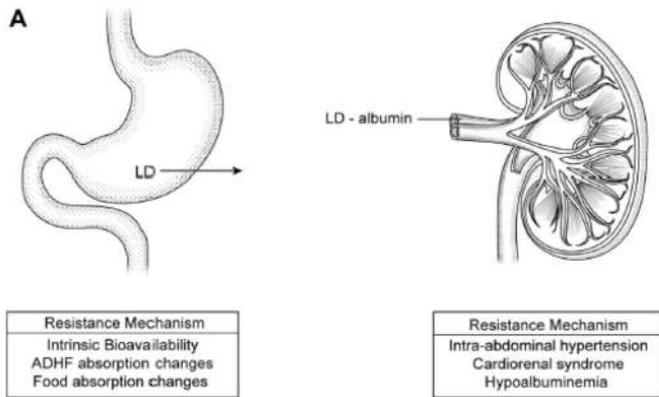


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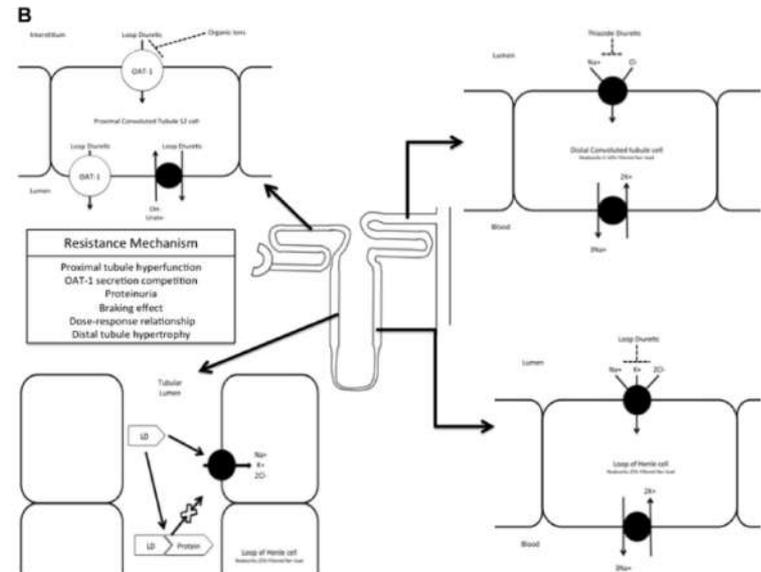


Manejo de la resistencia a diuréticos

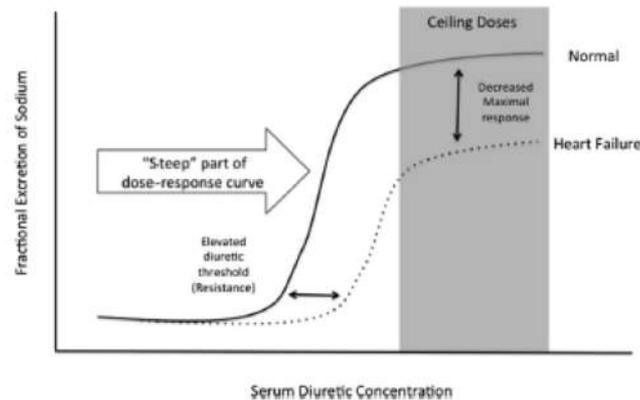
- Disminución de biodisponibilidad oral de diurético

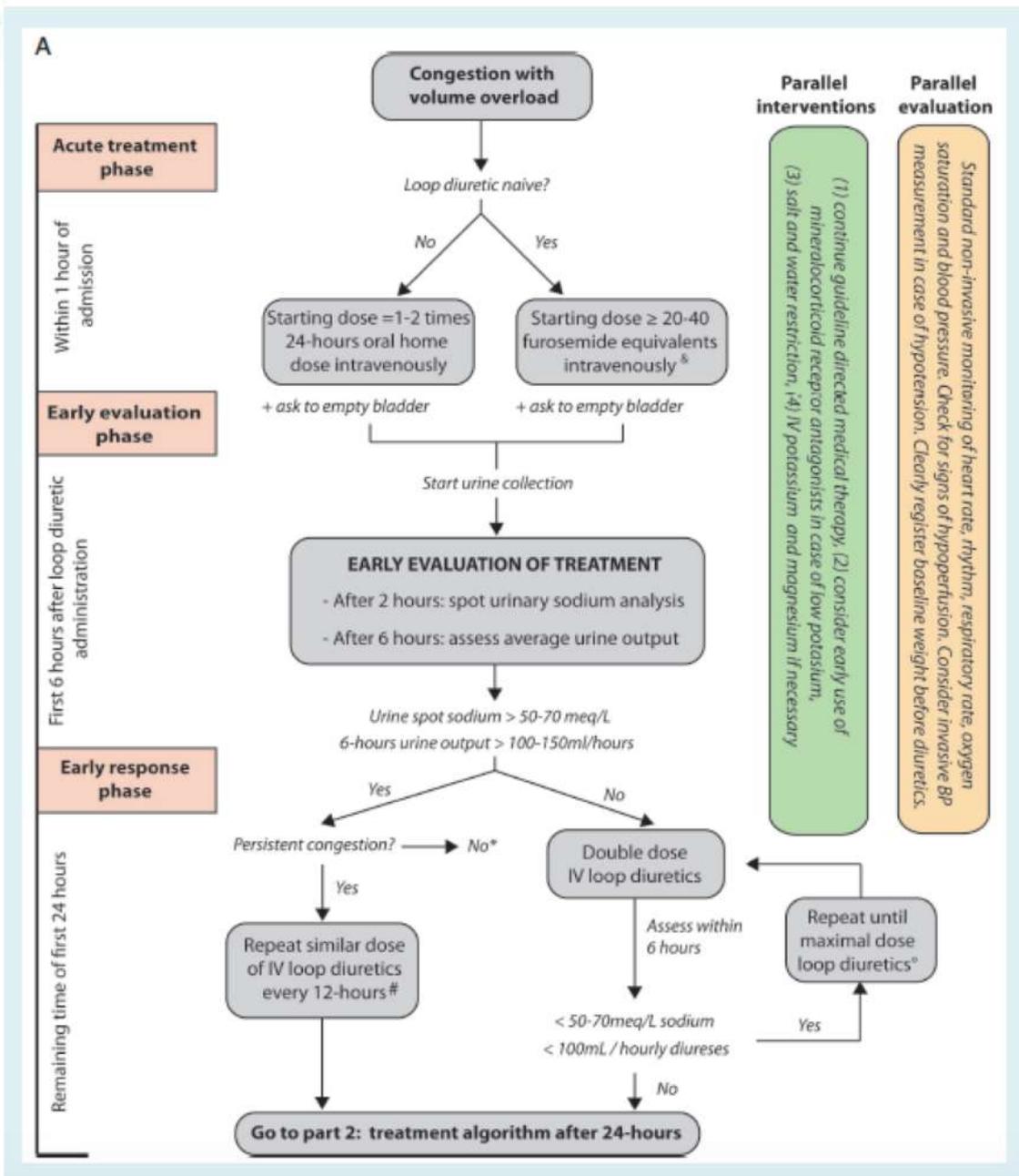
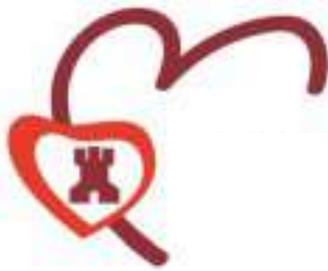


Absorption	Furosemide	Bumetanide	Torsemide
Bioavailability	10–100%	80–100%	80–100%
Affected by food	Yes	Yes	No



- Menor eficacia diurética en IC



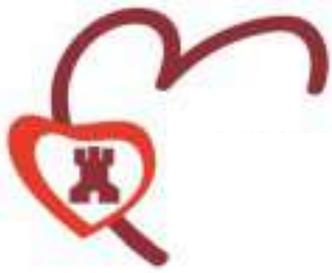


Aumentar dosis/frecuencia
Mejorar biodisponibilidad: iv
Restricción hidrosalina
Añadir un segundo diurético

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Manejo de la resistencia a diuréticos

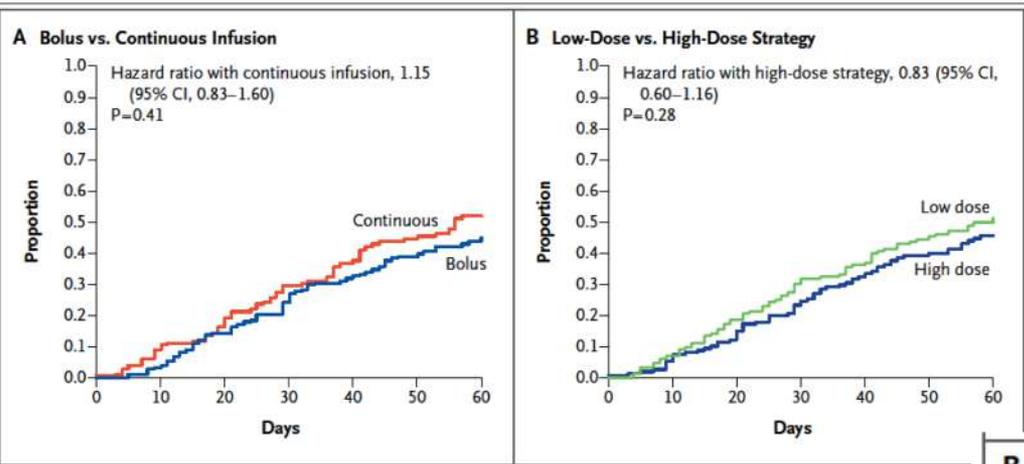
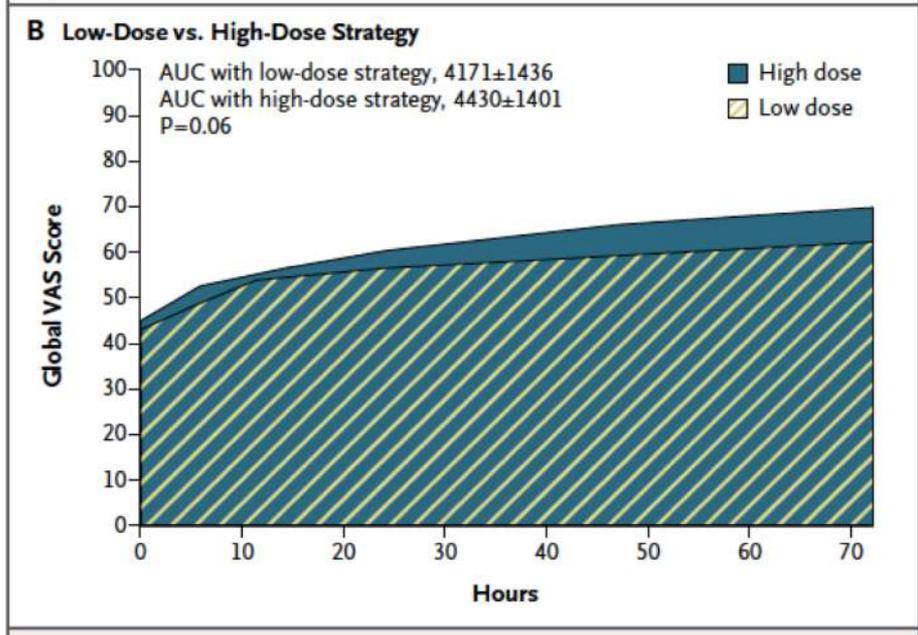
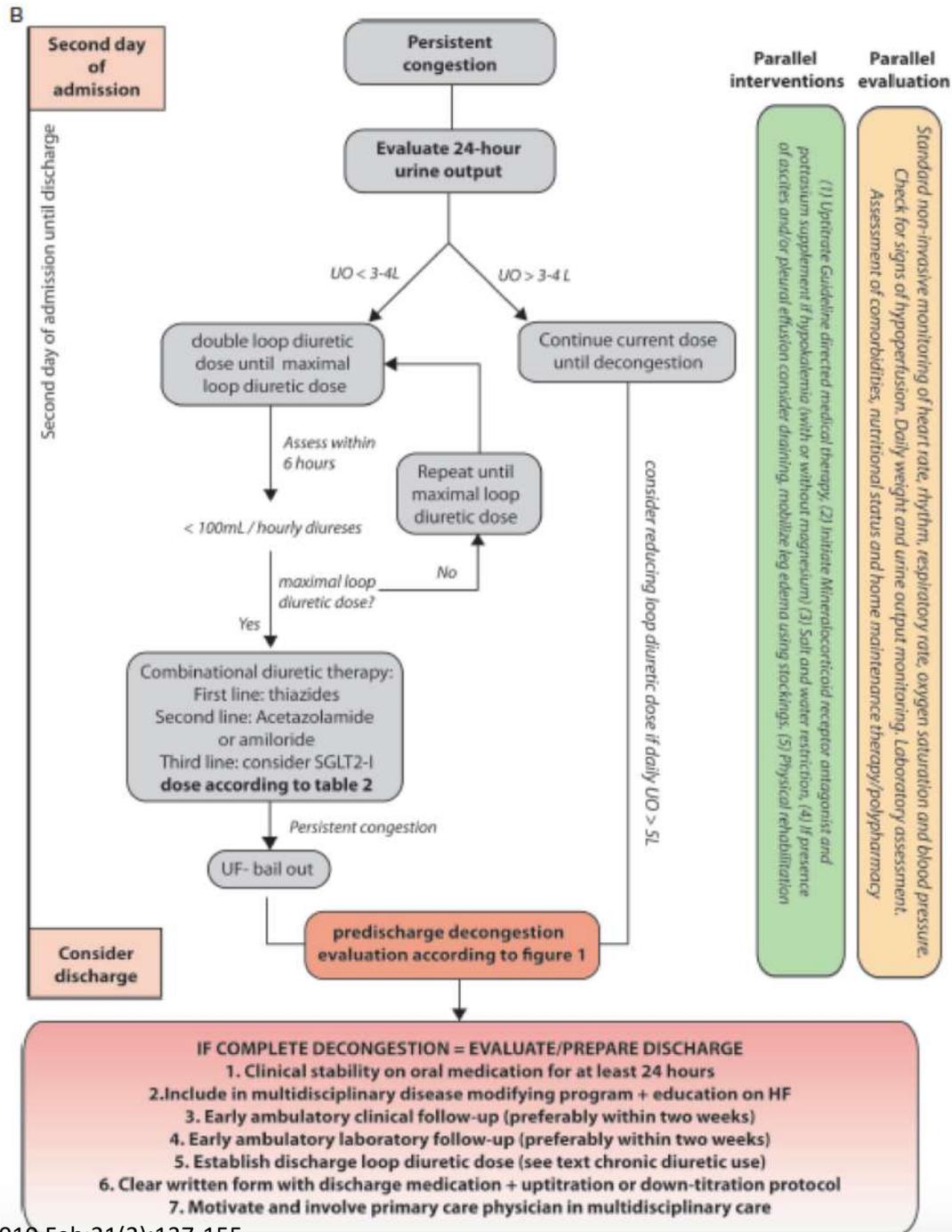
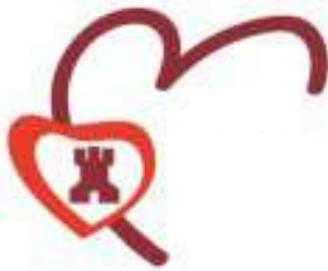
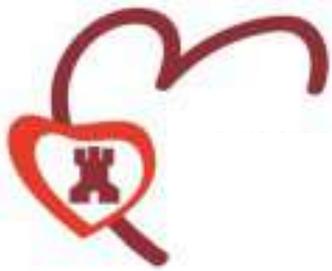


Figure 3. Kaplan–Meier Curves for the Clinical Composite End Point of Death, Rehospitalization, or Emergency Department Visit.
 Kaplan–Meier curves are shown for death, rehospitalization, or emergency department visit during the 60-day follow-up period in the group that received boluses every 12 hours as compared with the group that received a continuous infusion (Panel A) and in the group that received a low dose of the diuretic (equivalent to the patients' previous oral dose) as compared with the group that received a high dose (2.5 times the previous oral dose) (Panel B).





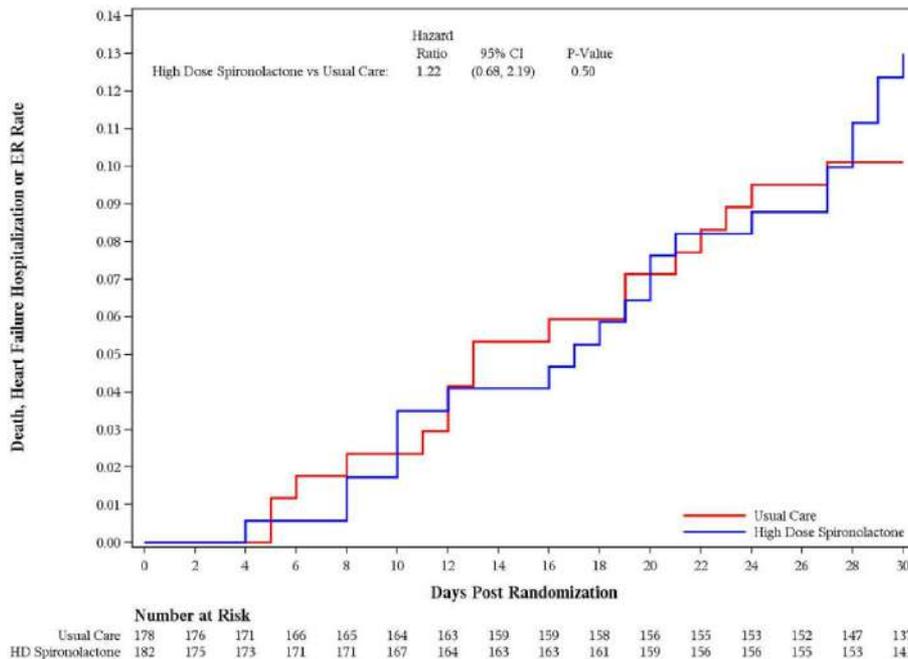


Antagonistas del receptor mineralcorticoide

September 2017

Efficacy and Safety of Spironolactone in Acute Heart Failure The ATHENA-HF Randomized Clinical Trial

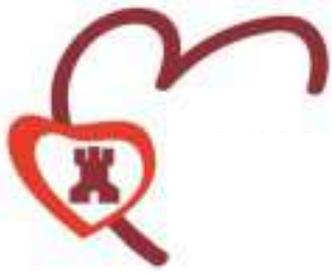
Javed Butler, MD, MPH¹; Kevin J. Anstrom, PhD²; G. Michael Felker, MD, MHS³; [et al](#)



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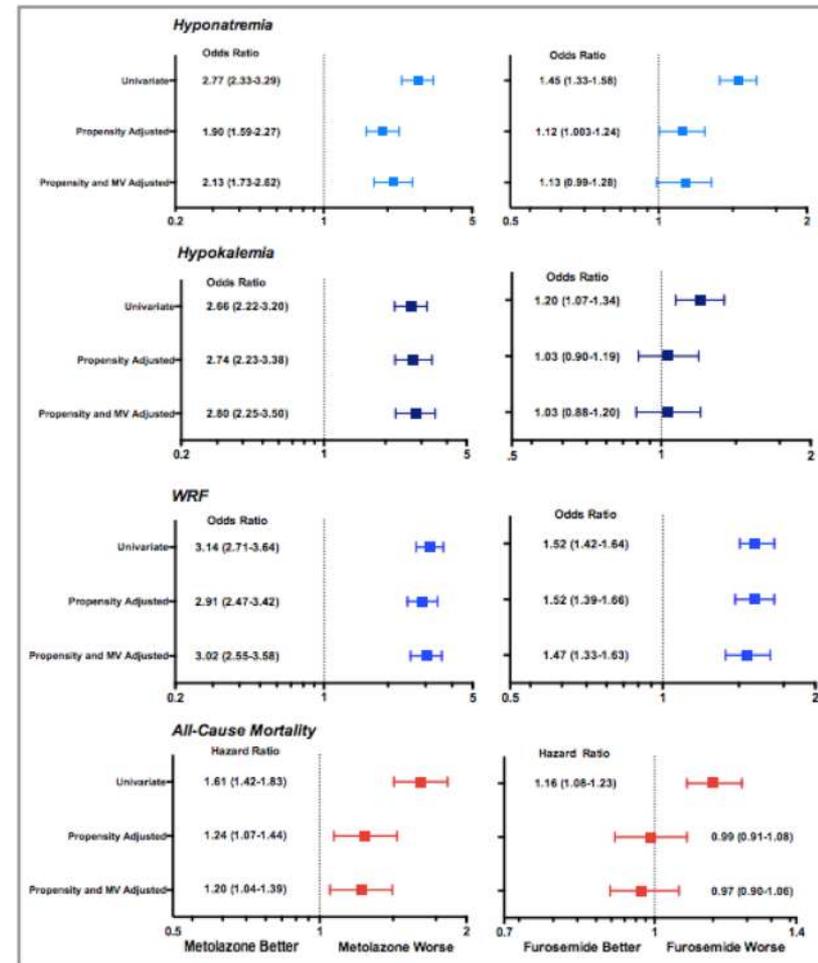
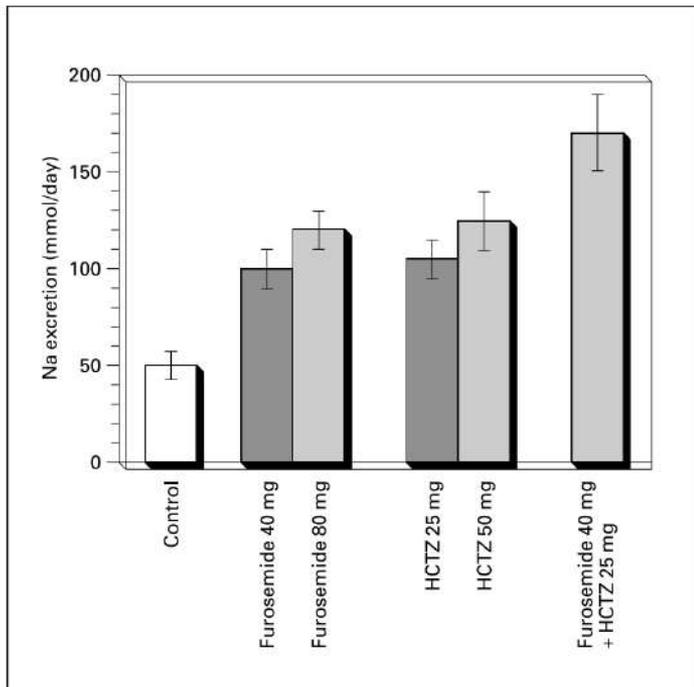


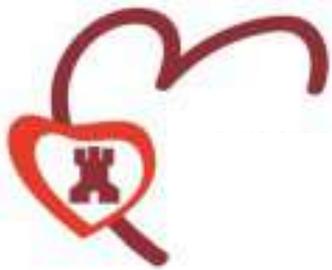
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Tiazidas

- Bloqueo túbulo contorneado distal
- Actúa sobre “fenómenos de frenada”
- Clortalidona vida media larga
- Riesgo de hiponatremia e hipopotasemia





Manejo de la resistencia a diuréticos

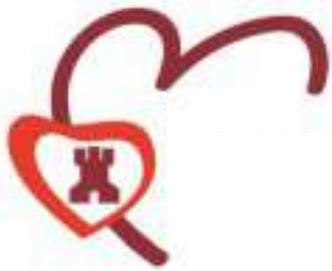
Table 2 Pharmacology of diuretics

	Acetazolamide	Loop diuretics	Thiazide-like diuretics	MRA ^a	Amiloride
Site of action	Proximal nephron	Ascending loop of Henle	Early distal convoluted tubule	Late distal tubule	Late distal tubule
Starting dose/usual chronic dose	Oral: 250–375 mg Intravenous: 500 mg	Furosemide: 20–40/40–240 mg ^b Bumetanide: 0.5–1.0/1–5 mg ^b Torsemide: 5–10/10–20 mg ^b	HCTZ: 25/12.5–100 mg ^c Metolazone: 2.5/2.5–10 mg ^c Chlorthalidone: 25/25–200 mg ^c Chlorothiazide: 500–1000 mg (IV formulation available)	Spirolactone: 25/25–50 mg Eplerenone: 25/25–50 mg Potassium canrenoate: 25–200 mg/not for chronic use	5/10 mg
Maximum recommended total daily dose	Oral: 500 mg 3x/day Intravenous: 500 mg 3x/day	Furosemide: 400–600 mg Bumetanide: 10–15 mg Torsemide: 200–300 mg	HCTZ: 200 mg Metolazone: 20 mg Chlorthalidone: 100 mg Chlorothiazide: 1000 mg	50–100 mg (doses up to 400 mg are used in hepatology)	20 mg
Half-life	2.4–5.4 h	Furosemide: 1.5–3.0 h Bumetanide: 1–1.5 h Torsemide: 3–6 h	HCTZ: 6–15 h Metolazone: 6–20 h Chlorthalidone: 45–60 h	Canrenone: 16.5 h ^d Eplerenone: 3–6 h	Normal GFR: 6–9 h GFR < 50 mL/min: 21–144 h
Onset	PO: 1 h IV: 15–60 min	PO: 0.5–1 h ^a IV: 5–10 min ^a SC: 0.5 h ^a	PO: 1–2.5 h IV: Chlorthiazide is IV available, onset action: 30 min	PO: 48–72 h ^d IV: potassium canrenoate; 2.5 h	PO: 2 h IV: not available
Oral bioavailability	Absorption is dose-dependent, dose >10 mg/kg exhibit variable uptake	Furosemide: 10–100% Bumetanide: 80–100% Torsemide: 80–100%	HCTZ: 65–75% Metolazone: 60–65% ^f Chlorthalidone: unknown Chlorothiazide: 9–56%	Spirolactone: ~90% Eplerenone: 69%	30–90%
Enteral absorption affected by food	May be taken with food. Food decreases symptoms of GI upset.	Furosemide: yes (slowed) Bumetanide: yes (slowed) Torsemide: no	HCTZ: unknown Metolazone: unknown Chlorthalidone: unknown	Spirolactone: bioavailability increase with high fat food Eplerenone: unknown	Unknown
Potency (FENa% ^g)	4%	20–25% ^a	5–8%	2%	2%

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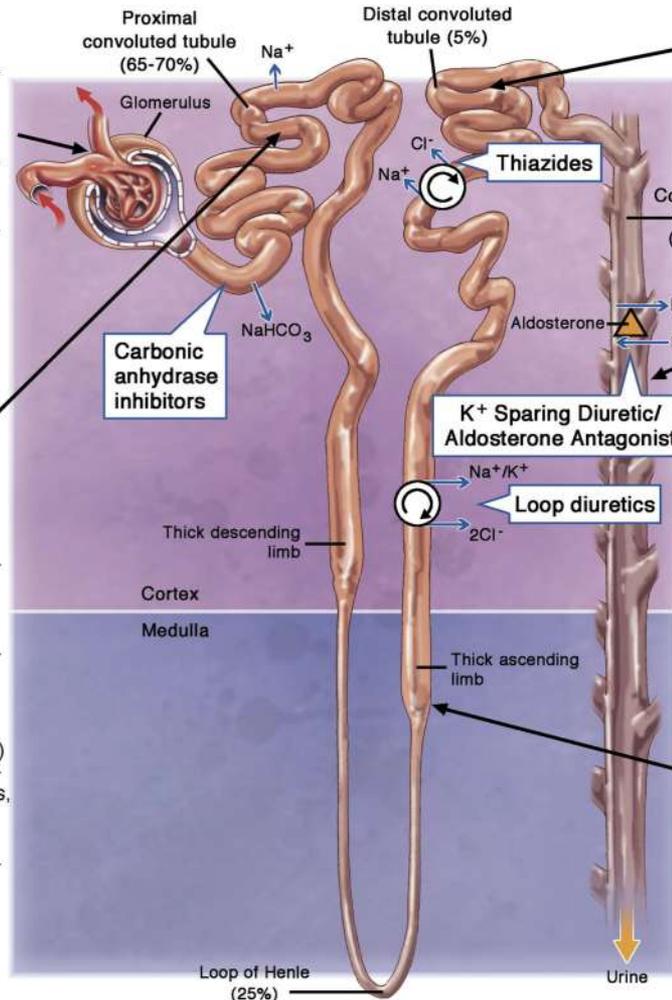
Acetazolamida

Reduced GFR:

Barriers	Potential solution
Abnormal glomerular hemodynamics	Discontinue NSAIDs, consider holding ACEI/ARB
Low cardiac output	Hemodynamic support
Chronic kidney disease or functional renal hypoperfusion	Increase LD dose

Proximal Tubule Hyperfunction:

Barriers	Potential solution
Neuro-hormonal activation	ACEI/ARB
Sodium-avid states	Increased LD doses, proximal tubule diuretics (i.e. acetazolamide)
Post-diuretic effect	Multiple daily doses, continuous LD infusion
Excessive daily sodium intake	Sodium restriction



Distal Tubule Hypertrophy:

Barriers	Potential solution
Rebound sodium retention	Sequential nephron blockade (Combination diuretic therapy)

Distal Nephron Hyperfunction:

Barriers	Potential solution
Excessive aldosterone-mediated sodium retention	Aldosterone antagonist, K ⁺ -sparing diuretic (ENaC blocker)
Excessive vasopressin-mediated water retention	Vasopressin antagonist, free water restriction

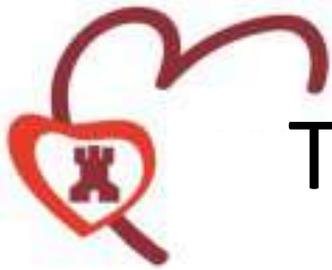
Loop of Henle Hyperfunction:

Barriers	Potential solution
Braking effect	Higher LD doses

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Tolvaptan

Effects of Oral Tolvaptan in Patients Hospitalized for Worsening Heart Failure

The EVEREST Outcome Trial

Marvin A. Konstam, MD; Mihai Gheorghiade, MD; John C. Burnett, MD; et al

- Antagonista de la vasopresina (v2)-acuarético
- VO
- Randomizado 537 ptes
- No efecto sobre mortalidad
- Mejoría disnea y datos de congestión
- Selección de pacientes: hiponatremia

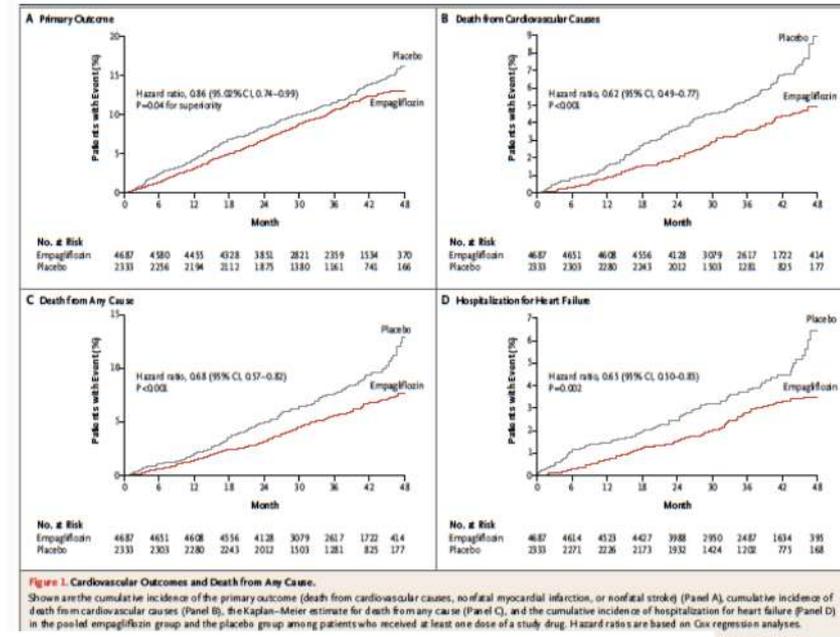
ISGLT-2

THE NEW ENGLAND JOURNAL OF MEDICINE

ORIGINAL ARTICLE

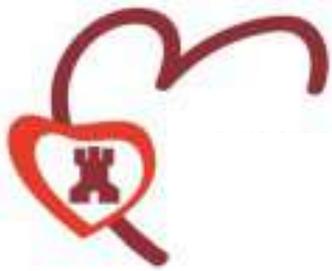
Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes

Bernard Zinman, M.D., Christoph Wanner, M.D., John M. Lachin, Sc.D., David Fitchett, M.D., Erich Bluhmki, Ph.D., Stefan Hantel, Ph.D., Michaela Mattheus, Dipl. Biomat., Theresa Devins, Dr.P.H., Odd Erik Johansen, M.D., Ph.D., Hans J. Woerle, M.D., Uli C. Broedl, M.D., and Silvio E. Inzucchi, M.D., for the EMPA-REG OUTCOME Investigators



Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction

John J.V. McMurray, M.D., Scott D. Solomon, M.D., Silvio E. Inzucchi, M.D., Lars Køber, M.D., D.M.Sc., Mikhail N. Kosiborod, M.D., Felipe A. Martinez, M.D., Piotr Ponikowski, M.D., Ph.D., Marc S. Sabatine, M.D., M.P.H., Inder S. Anand, M.D., Jan Bělohávek, M.D., Ph.D., Michael Böhm, M.D., Ph.D., Chern-En Chiang, M.D., Ph.D., et al., for the DAPA-HF Trial Committees and Investigators*



Suero Salino Hipertónico

- Disminución de todas las causas de muerte
- Disminución del riesgo de rehospitalización por ICA
- Menor estancia hospitalaria, > pérdida de peso y < deterioro de función renal



Gandhi S et al. *Int J Cardiol.* 2014 May 1;173(2):139-45

Estrategias en congestión refractaria: efectos del suero salino hipertónico en insuficiencia cardiaca aguda

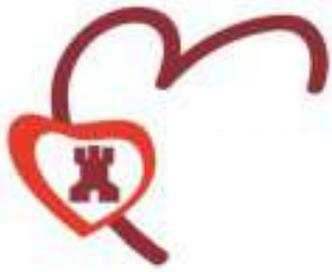
Tabla 2 – Parámetros clínicos y analíticos 24 h antes y después de la administración de suero salino hipertónico con furosemida intravenosa

	24 h antes SS + Fiv	24 h después SS + Fiv	Diferencia	Valor de p
Diuresis (ml)	1.375 ± 733	2.161 ± 966	771 ± 706	<0,001
Peso (kg)	80 ± 21	79,2 ± 20	-0,9 ± 1	<0,001
PAS (mmHg)	112 ± 16	107 ± 18	-4,5 ± 13	0,02
Cr (mg/dl)	1,7 ± 0,9	1,7 ± 0,9	0 ± 0,3	0,75
Na (mEq/l)	137 ± 4,6	138 ± 4,2	0,3 ± 2,3	0,258
K (mEq/l)	4,3 ± 0,5	4,1 ± 0,6	-0,1 ± 0,5	0,14

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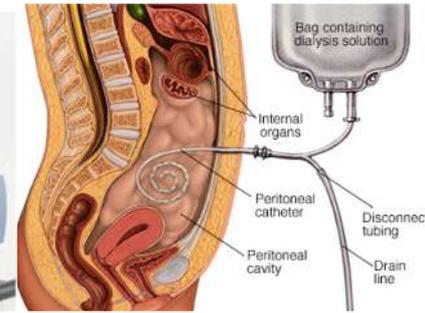


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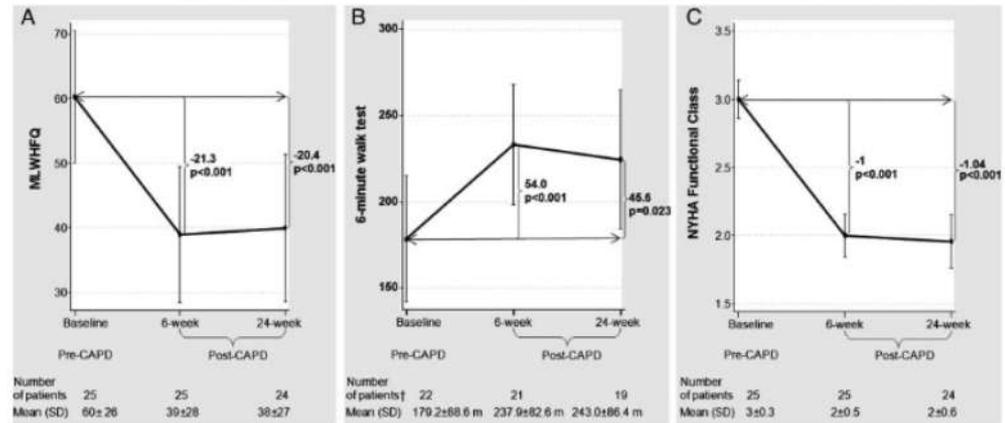
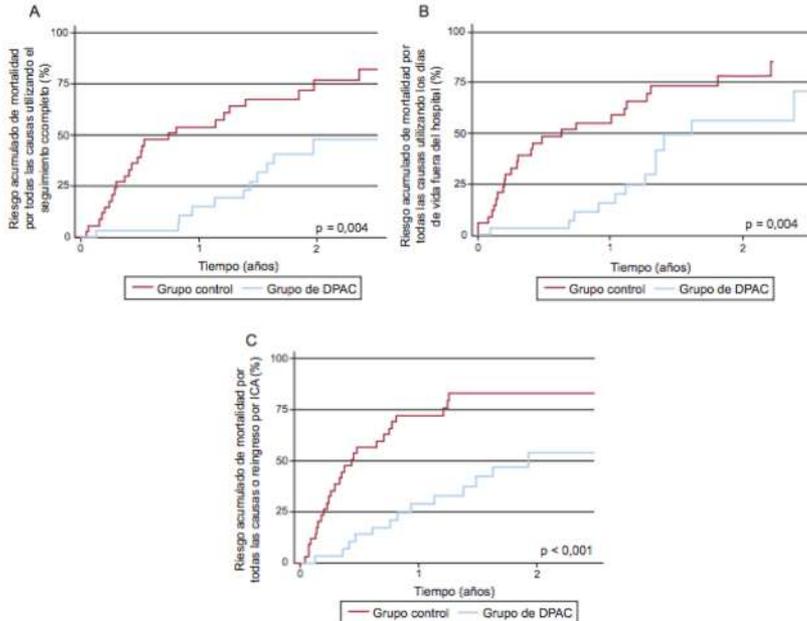


Terapia renal sustitutiva

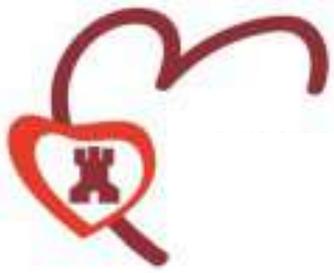
- La ultrafiltración elimina el agua de plasma a través de una membrana semipermeable
- Oliguria/anuria/acidosis/uremia. TFG <<<
- CARRESS-HF
- Diálisis peritoneal en IC refractaria e IR



J. Núñez et al/ Rev Esp Cardiol. 2012;65(11):986-995

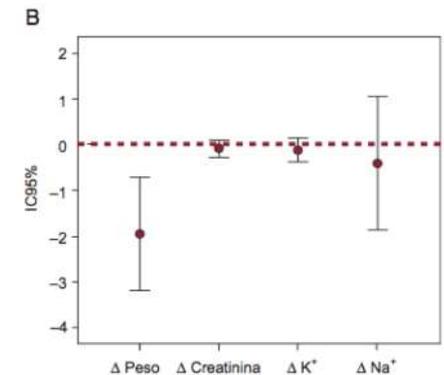
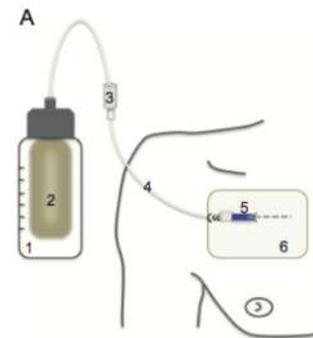


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Otros: perfusión continua domiciliaria

- Subcutánea o intravenosa
- Resistencia a diuréticos orales y falta de respuesta/imposibilidad de administración en dosis intermitentes
- Infusión a velocidad constante de 2 ml/h (300cc)
- Colaboración con C. Paliativos/MAP/ESAD
- Escasa evidencia científica
- pH furosemida/estabilidad



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Gracias por vuestra atención

