XVI curso

Gestión Integral del Medicamento en los servicios de URgencias

GIMUR

INSUFICIENCIA CARDIACA AGUDA

JAVIER JACOB
HECTOR ALONSO

ORGANIZA:

sefh Sociedad Española de Farmacia Hospitalaria

fefh Fundación Española de Farmacia Hospitalaria

redfaster Grupo de trabajo de Atención Farmacéutica en Urgencias de la sefh

Insuficiencia Cardiaca Aguda

Impacto en la salud

Time trends in characteristics, clinical course, and outcomes of 13,791 patients with acute heart failure

Pere Llorens[1] · Patricia Javaloyes[1] · Francisco Javier Martín-Sánchez[2,3] · Javier Jacob[4] · Pablo Herrero-Puente[5] · Víctor Gil[6] · José Manuel Garrido[7] · Eva Salvo[8] · Marta Fuentes[9] · Héctor Alonso[10] · Fernando Richard[11] · Francisco Javier Lucas[12] · Héctor Bueno[13,14] · John Parissis[15] · Christian E. Müller[16] · Òscar Miró[3,6,17] on behalf of the ICA-SEMES Research Group

Clinical Research in Cardiology 2018;107:897-913

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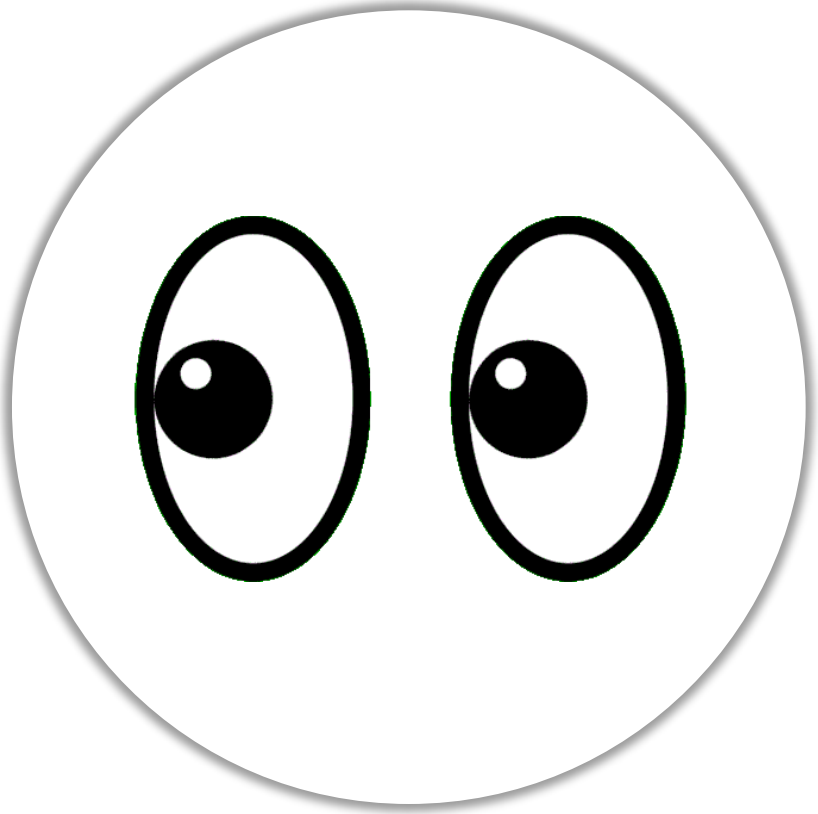
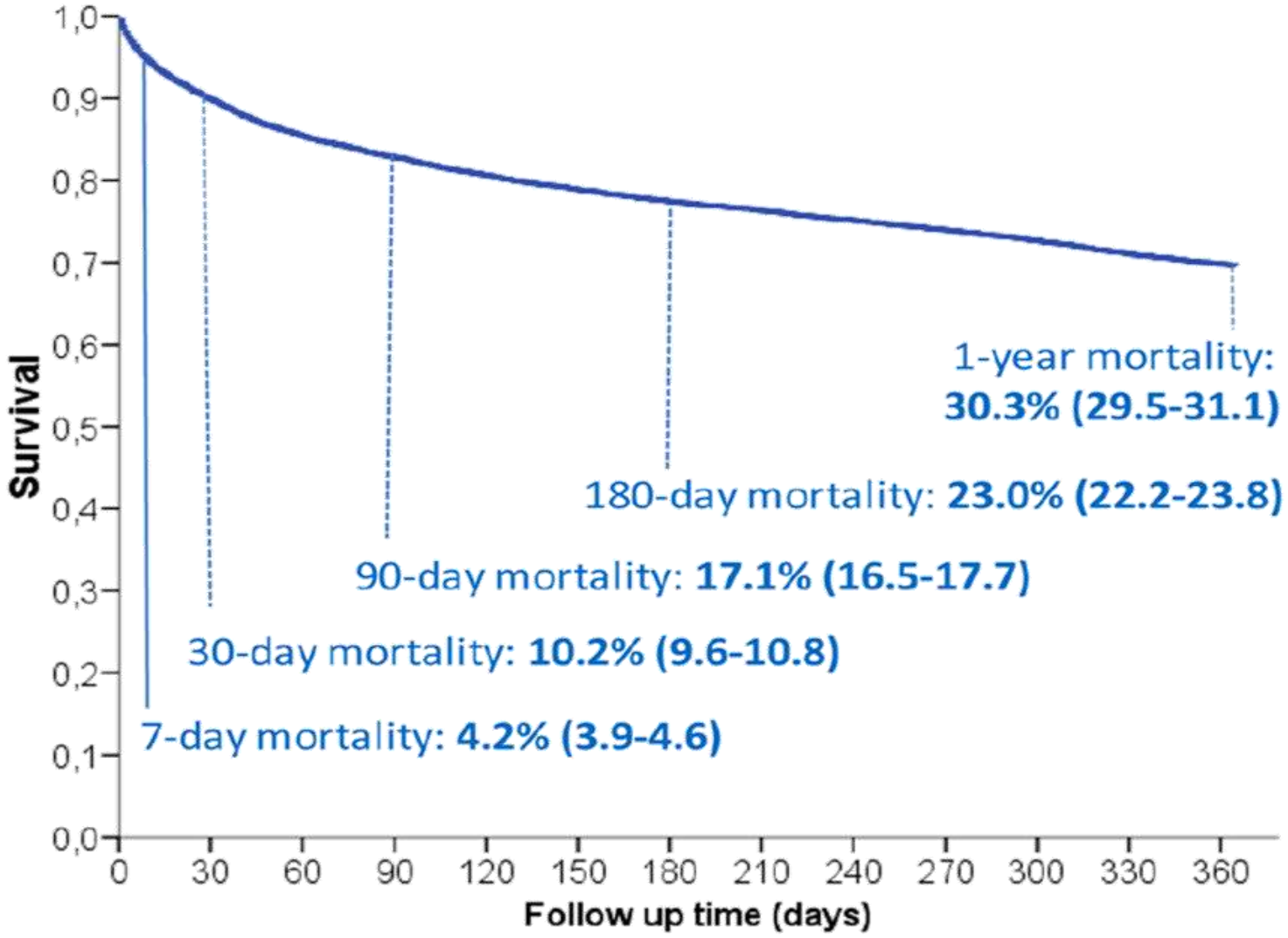
Clinical Research in Cardiology 2018;107:897-913



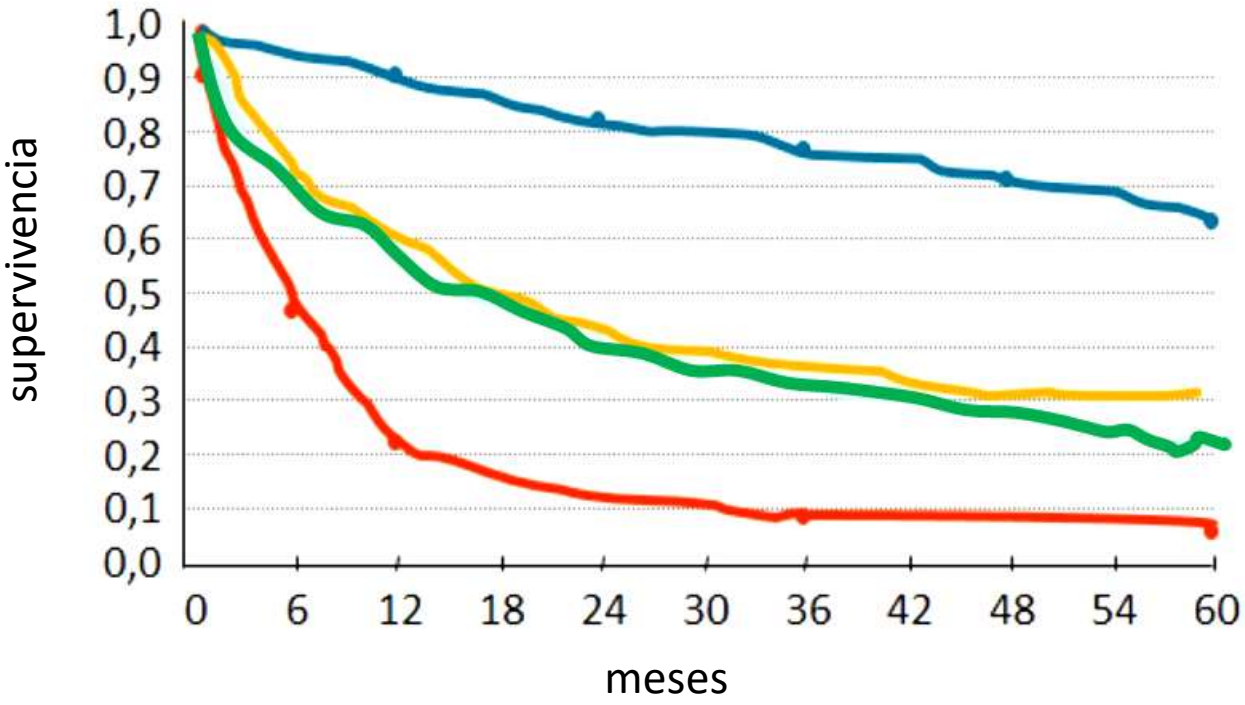
MORTALIDAD POR TODAS LAS CAUSAS

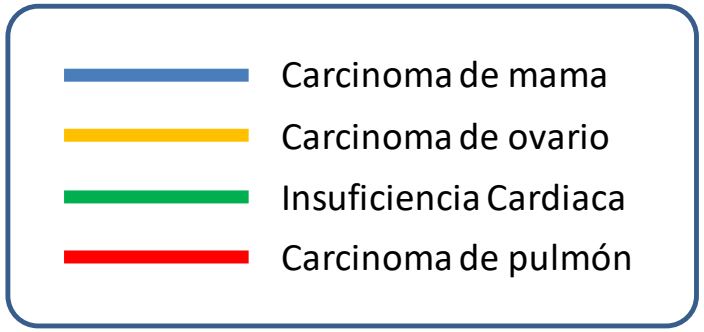


European Journal of Heart Failure 2001;3:315-322

More 'malignant' than cancer? Five-year survival following a first admission for heart failure

Simon Stewart[a,b], Kate MacIntyre[b], David J. Hole[c], Simon Capewell[b], John J.V. McMurray[a,*]





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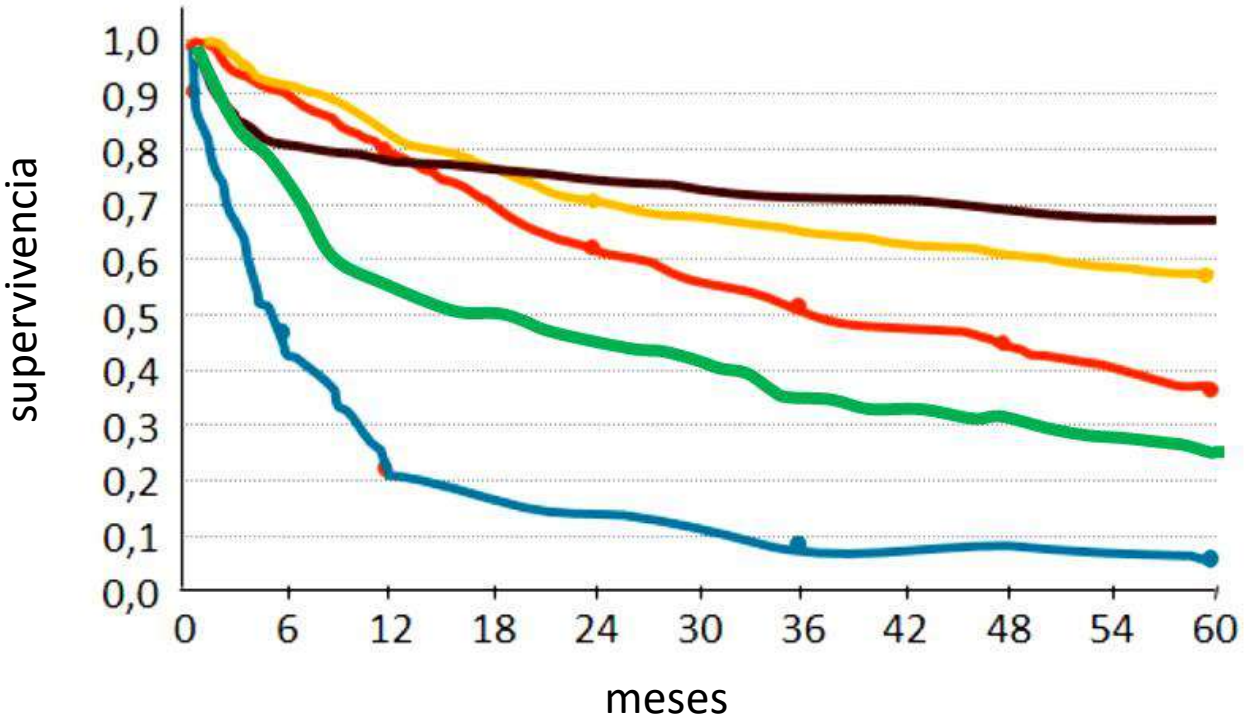
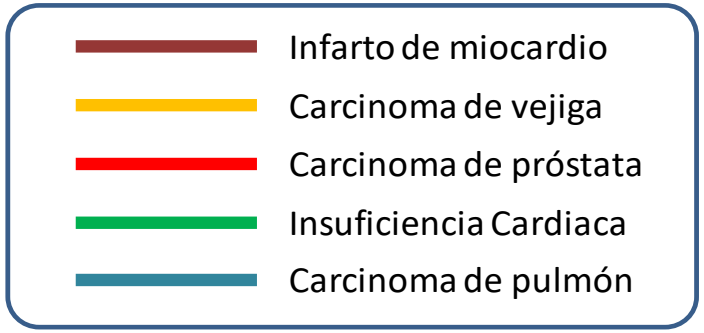
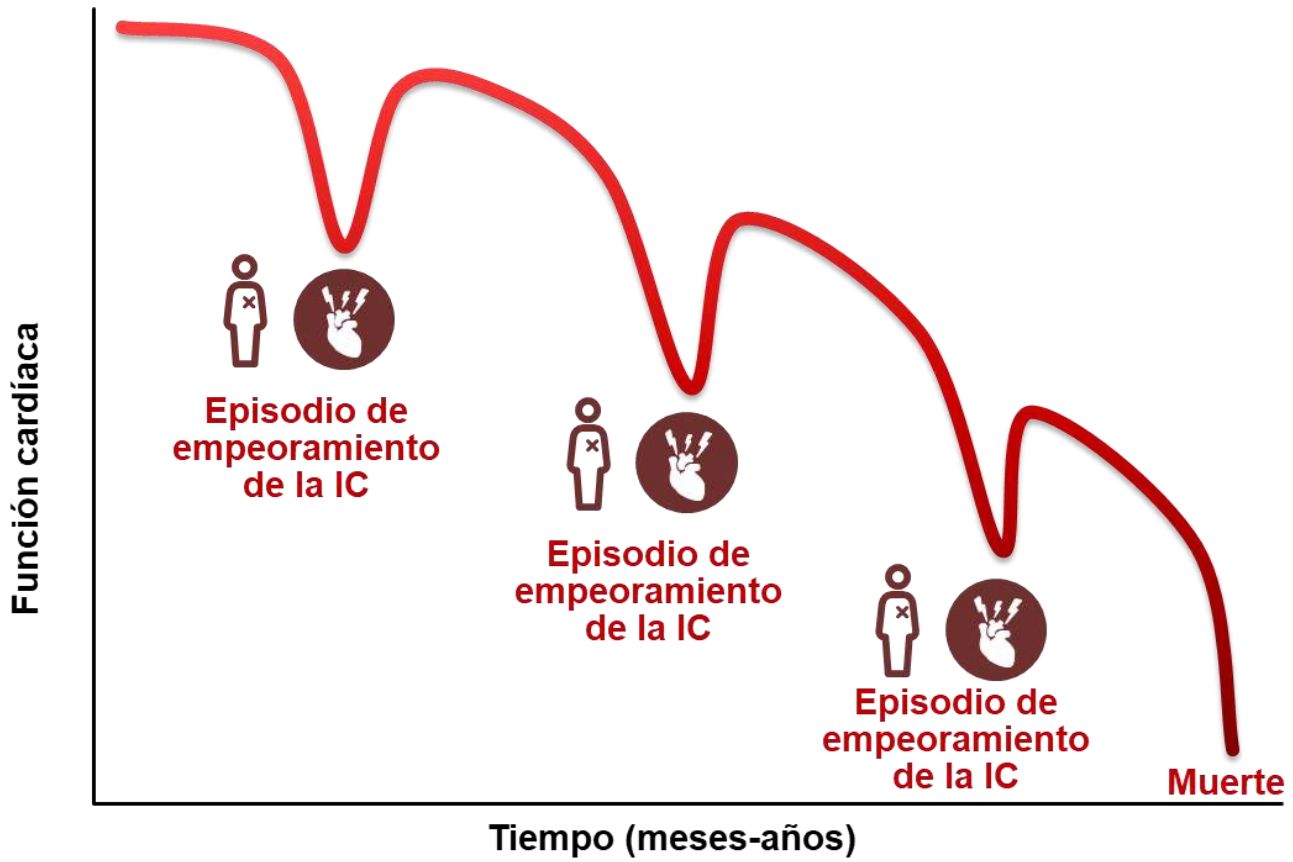
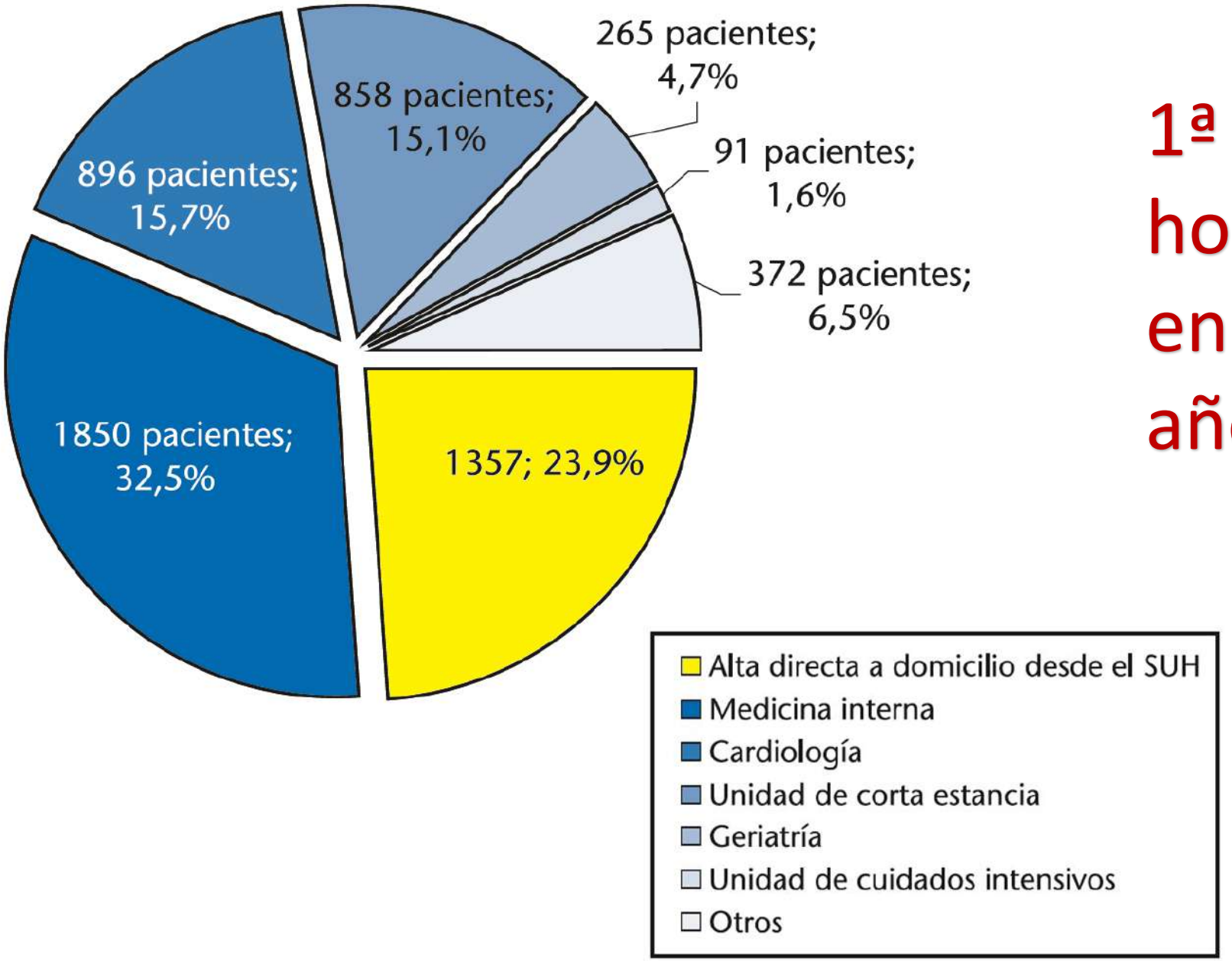




Am J Cardiol 2005;96(suppl):11-17

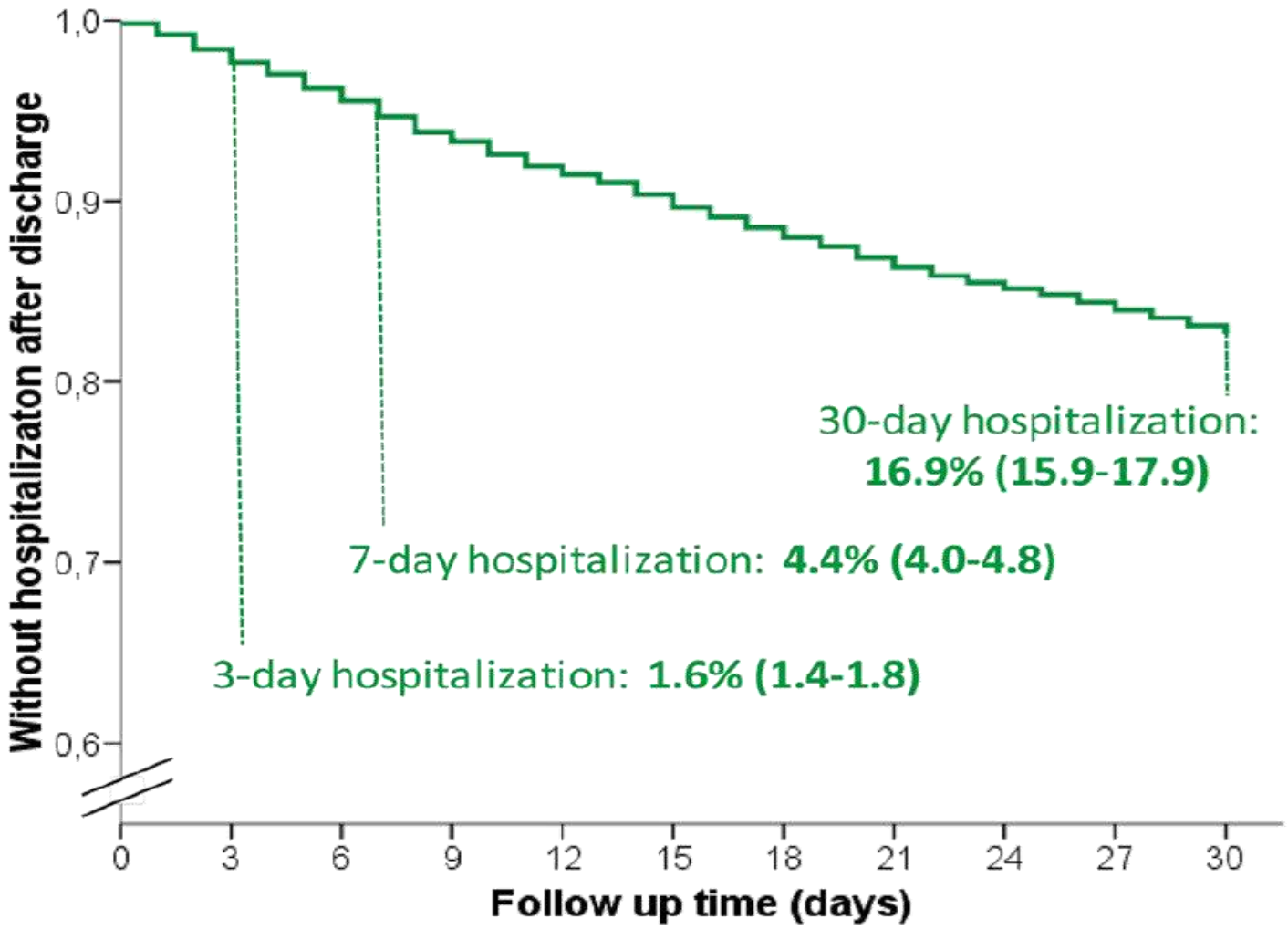


Impacto económico



1ª causa hospitalización en mayores de 65 años

NECESIDAD DE HOSPITALIZACIÓN TRAS EL ALTA



Management of patients with HFrEF

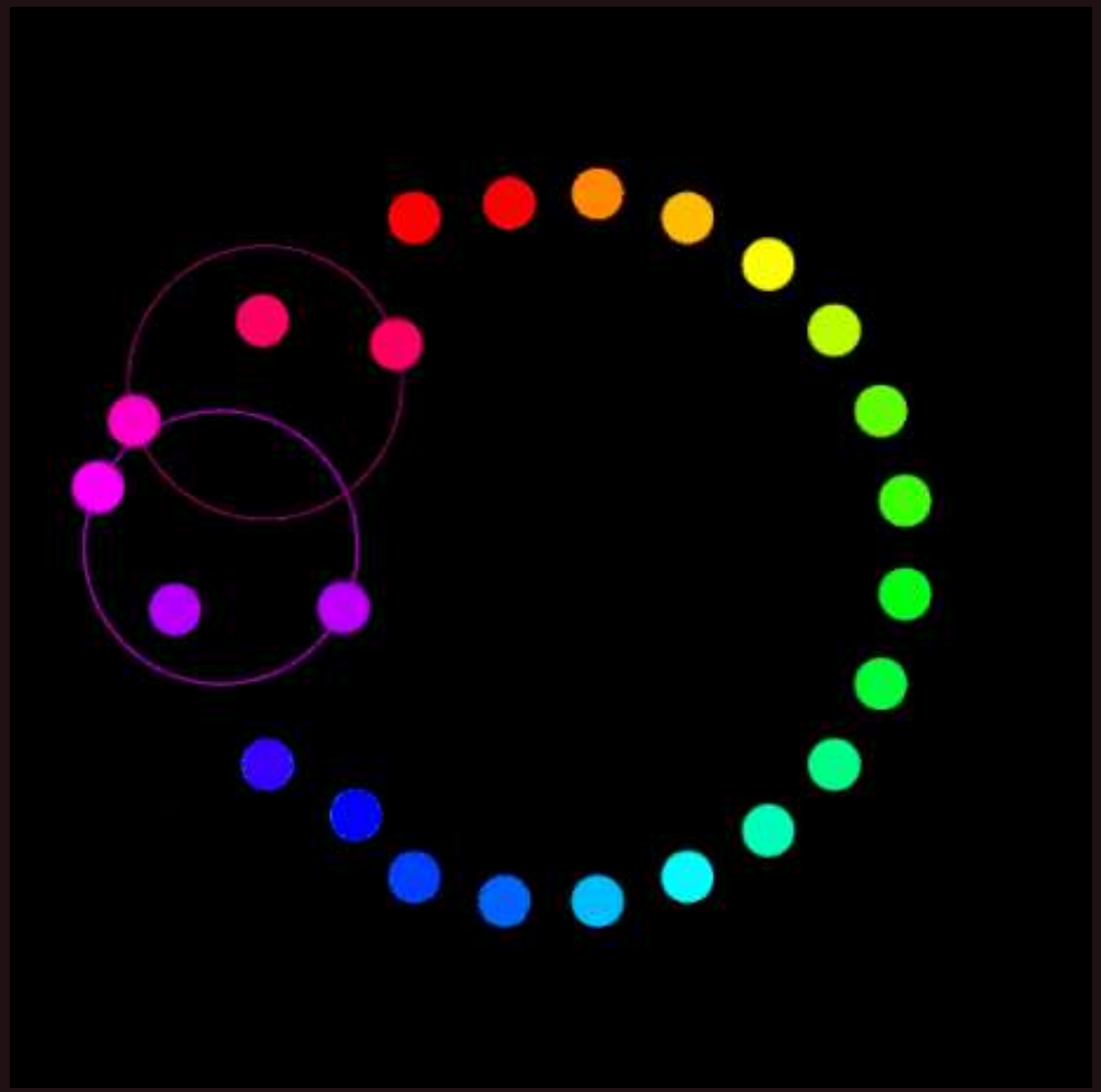
- ACE-I/ARNI[a]
- Beta-blocker
- MRA
- Dapagliflozin/Empagliflozin
- Loop diuretic for fluid retention

(Class I)

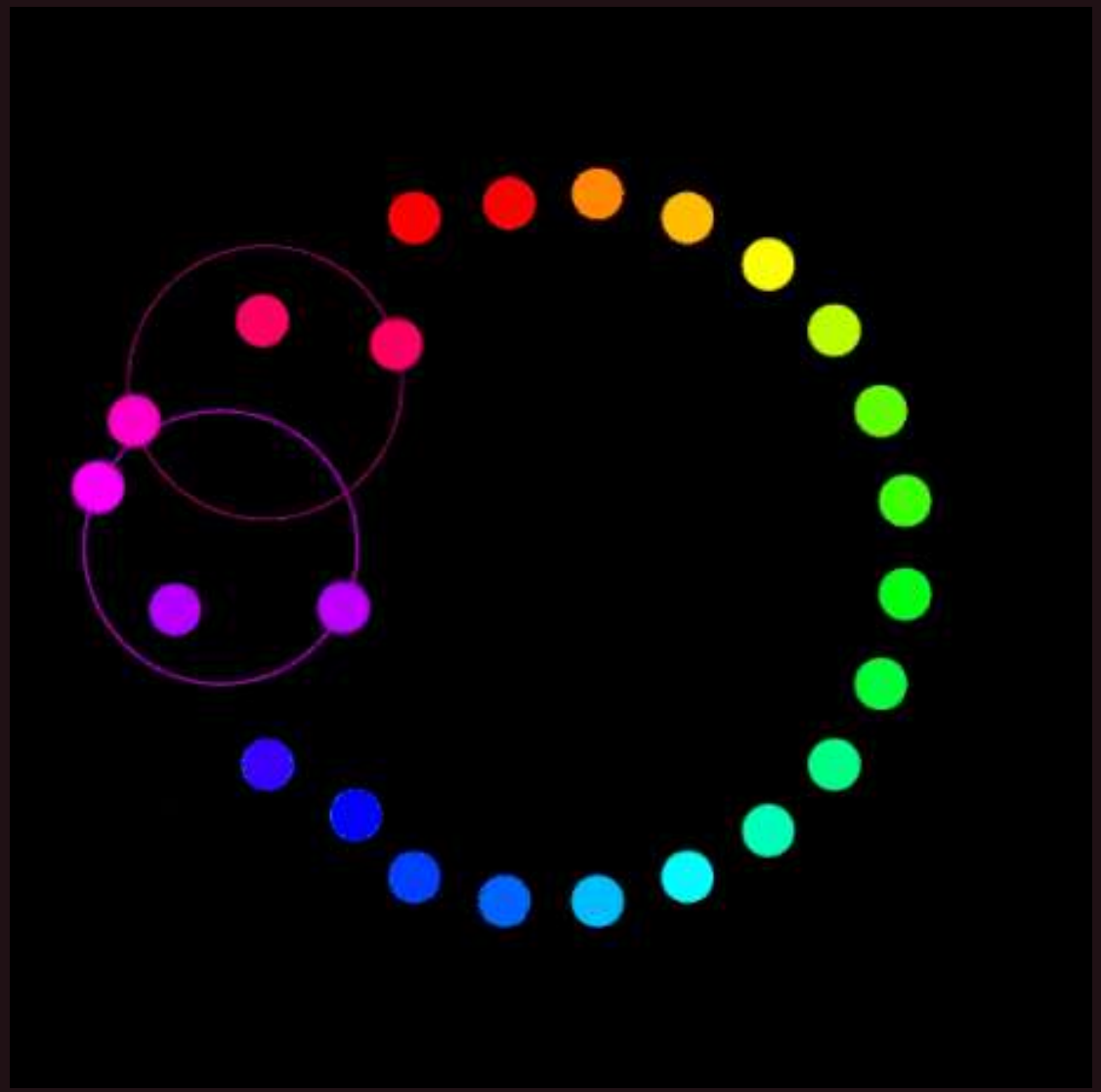
ESC

En este pronóstico actúan muchos actores

Toma de decisión adecuada



- Edad
- Función renal
- NTproBNP
- Hiperpotasemia
- Hiponatremia
- Natriuresis
- Dependencia
- NYHA
- Factores desencadenantes
- Nivel socioeconómico
- Presión arterial
- Saturación oxigeno



Ann Intern Med. 2017; doi:10.7326/M16-2726

Predicting 30-Day Mortality for Patients With Acute Heart Failure Who Are in the Emergency Department

A Cohort Study

Òscar Miró, PhD; Xavier Rossello, MD; Víctor Gil, PhD; Francisco Javier Martín-Sánchez, PhD; Pere Llorens, PhD; Pablo Herrero-Puente, PhD; Javier Jacob, PhD; Héctor Bueno, PhD; and Stuart J. Pocock, PhD*; on behalf of the ICA-SEMES (Acute Heart Failure of the Spanish Society of Emergency Medicine) Research Group

ROC

0,836 (95%CI 0,818-0,853)

Ann Intern Med. 2017; doi:10.7326/M16-2726

Sociedad Española de Medicina de Urgencias y Emergencias

MEESSI-AHF RISK MODEL

Barthel index at admission
- ≥75
- 50-74
- 25-49
- <25
- Unknown

Barthel Index

Systolic BP (mm Hg)
- ≥155
- 140-154
- 125-139
- 110-124
- 95-109
- <95

Age (years)
- <75
- 75-79
- 80-84
- 85-89
- ≥90

NT-proBNP (pg/mL)
- <8000
- 8000-15999
- 16000-23999
- ≥24000
- Unknown

Potassium (mEq/L)
- <3.5
- 3.5-4.9
- 5-5.5
- >5.5

NYHA class IV at admission
- Yes
- No

Positive troponine level
- Normal
- Positive
- Unknown

Respiratory rate(breaths per min)
- <25
- 25-29
- ≥30

Low output symptoms
- Yes
- No

Oxygen saturation (%)
- 95-100
- 90-94
- 84-89
- <84

Episode associated with ACS
- Yes
- No

Hypertrophy at ECG
- Yes
- No

Creatinine (mg/dL)
- <1.5
- 1.5-2.4
- ≥2.5

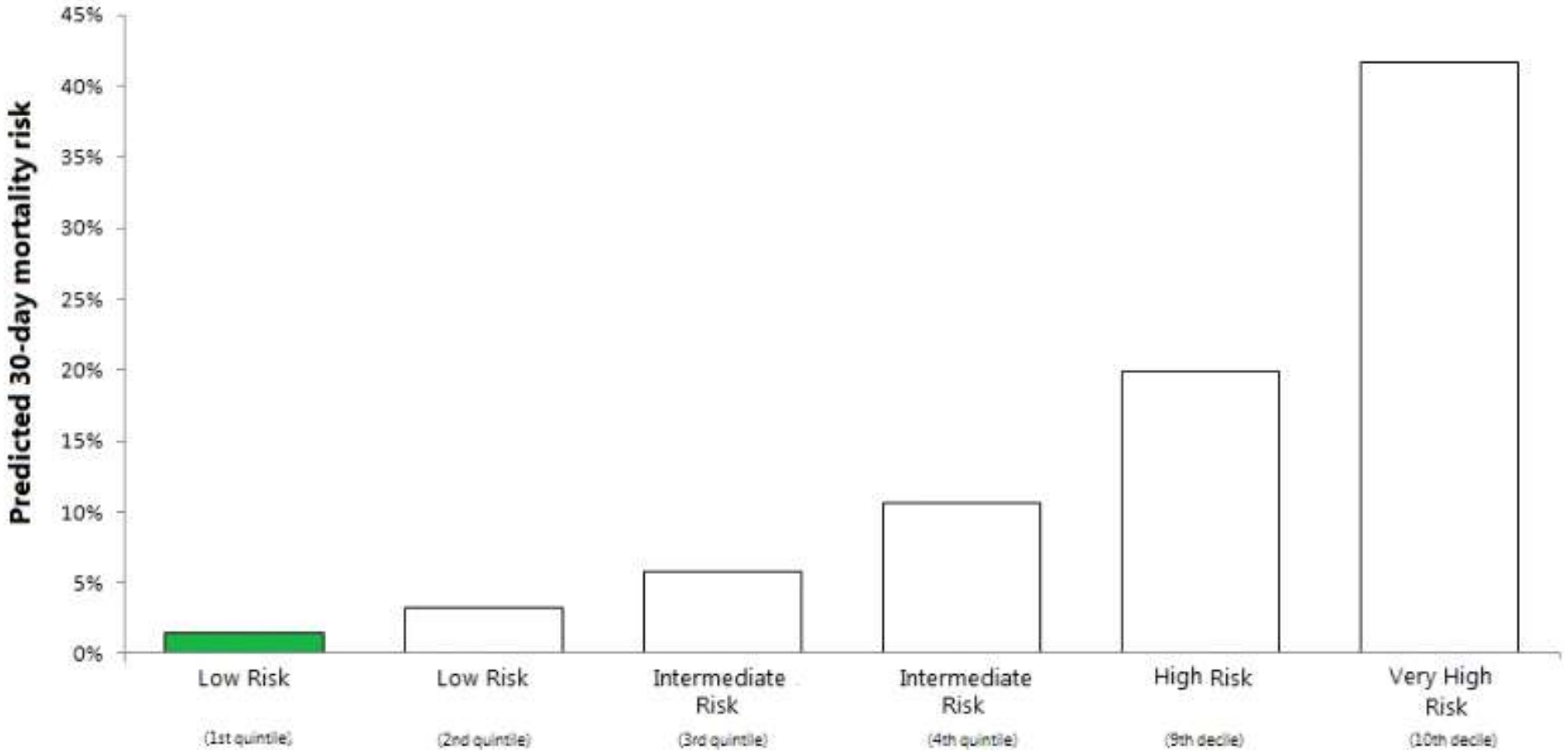
CALCULATE

https://meessi-ahf.gruposemes.org/calc.html

Score

This patient's predicted 30-day mortality risk is 2.084%

This patient is in **LOW** risk group



Cuál es el problema de manejo de los pacientes con ICA

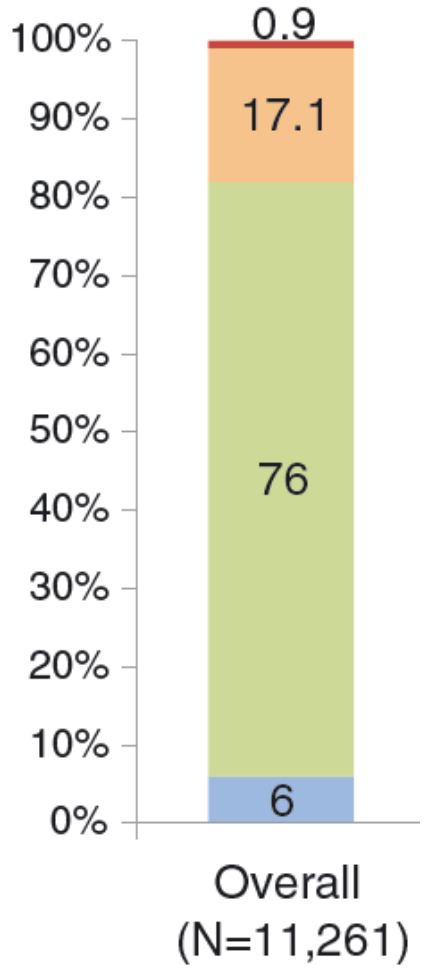
La congestión

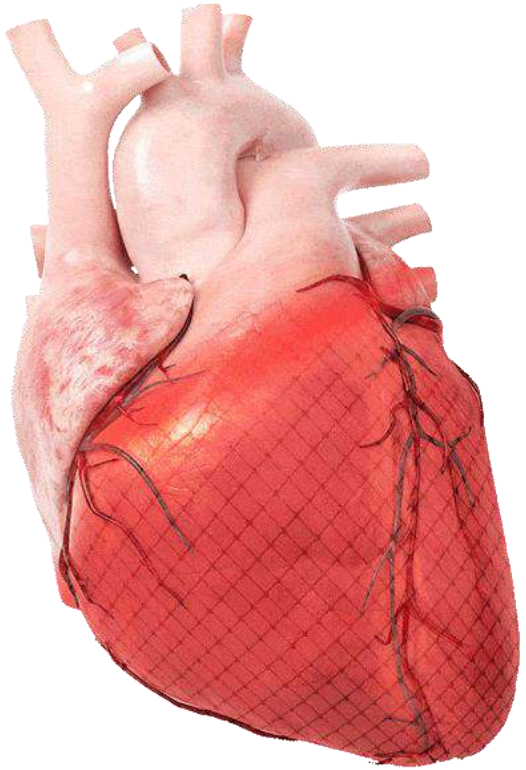
European Journal of Heart Failure 2019;21:1353-1365

Clinical phenotypes of acute heart failure based on signs and symptoms of perfusion and congestion at emergency department presentation and their relationship with patient management and outcomes

Patricia Javaloyes[1†], Òscar Miró[2†*], Víctor Gil[2], Francisco Javier Martín-Sánchez[3], Javier Jacob[4], Pablo Herrero[5], Koji Takagi[6,7], Aitor Alquézar-Arbé[8] and Pere Llorens[1], on behalf of the ICA-SEMES Research Group[‡]

>90% tienen congestión





sistema nervioso simpático

sistema renina-angiotensina-aldosterona

sistema arginina-vasopresina

SNS

Epinefrina
Norepinefrina → Receptores α_1, β_1, β_2

Vasoconstricción

- Actividad SRAA ↑
- Vasopresina ↑
- Frecuencia cardiaca ↑
- Contractilidad ↑

Signos y síntomas de la IC

Sistema PN

RPN ← PN

Vasodilatación

- ↓ Presión arterial
- ↓ Tono simpático
- ↑ Natriuresis/diuresis
- ↓ Vasopresina
- ↓ Aldosterona
- ↓ Fibrosis
- ↓ Hipertrofia

Neprilisina → Fragmentos inactivos

SRAA

Ang II → RAT_1

Vasoconstricción

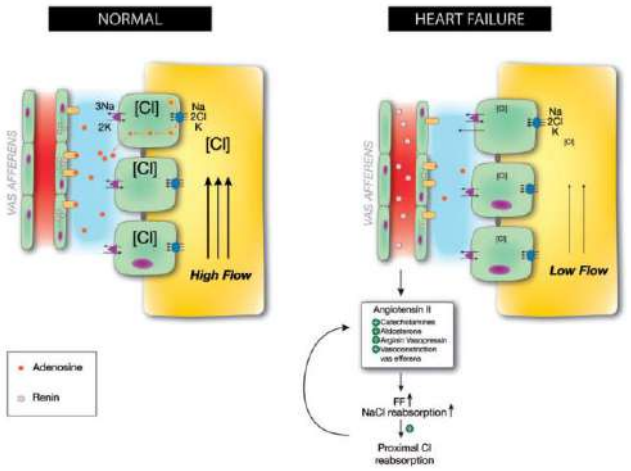
- Presión arterial ↑
- Tono simpático ↑
- Aldosterona ↑
- Hipertrofia ↑
- Fibrosis ↑

European Heart Journal (2017) **38**, 1872–1882
doi:10.1093/eurheartj/ehx035

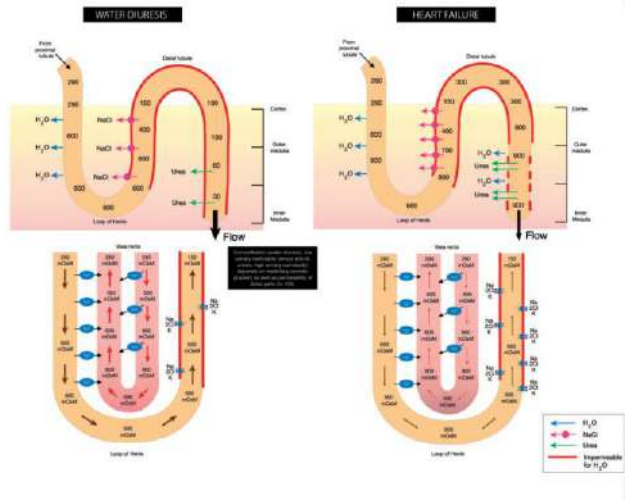
REVIEW

Renal sodium avidity in heart failure: from pathophysiology to treatment strategies

Wilfried Mullens[1,2]*, Frederik Hendrik Verbrugge[1], Petra Nijst[1,3], and Wai Hong Wilson Tang[4]

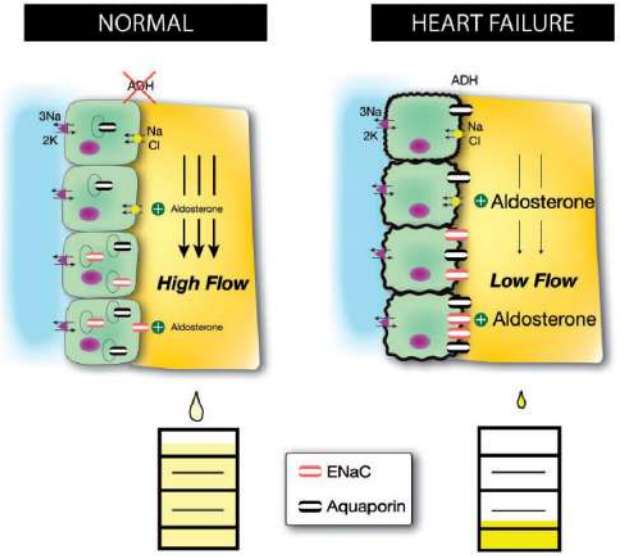


The macula densa

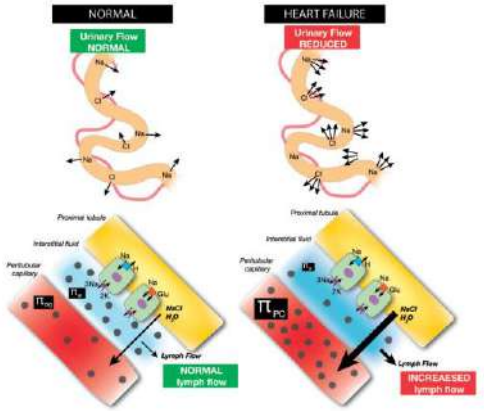


Loop of Henle and vasa recta

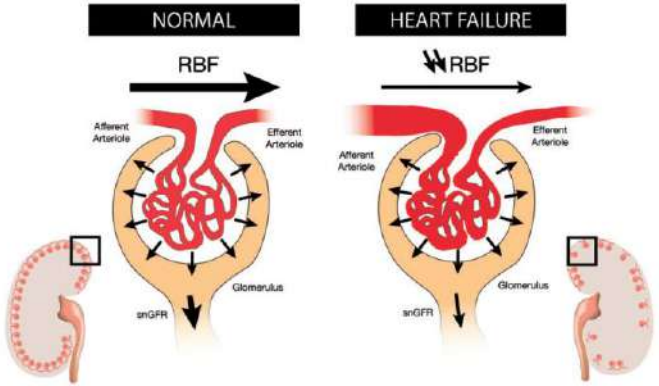
Avidez global por retener el sodio



Distal convoluted tubules and collecting ducts



Proximal tubules

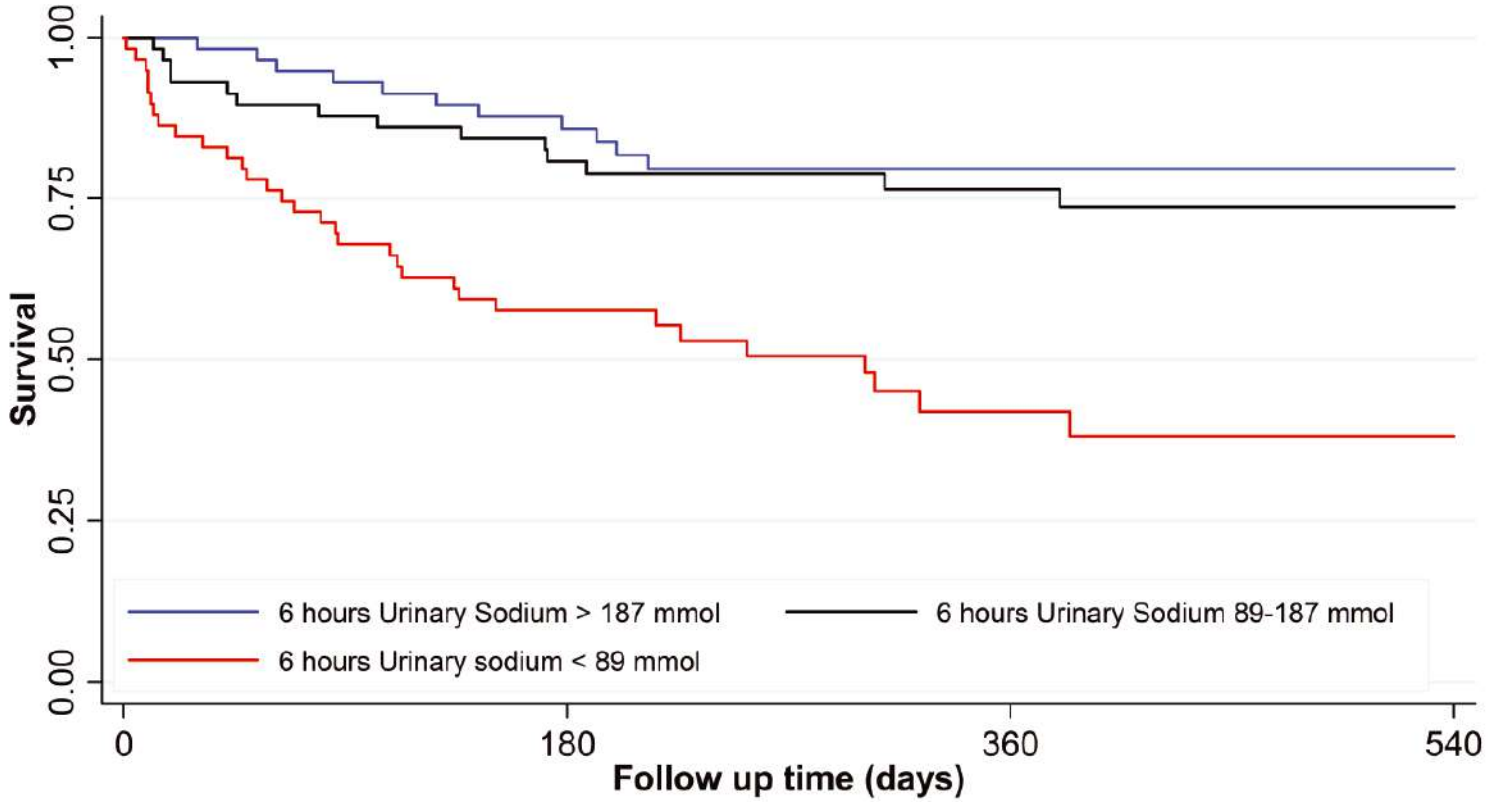


The glomerulus

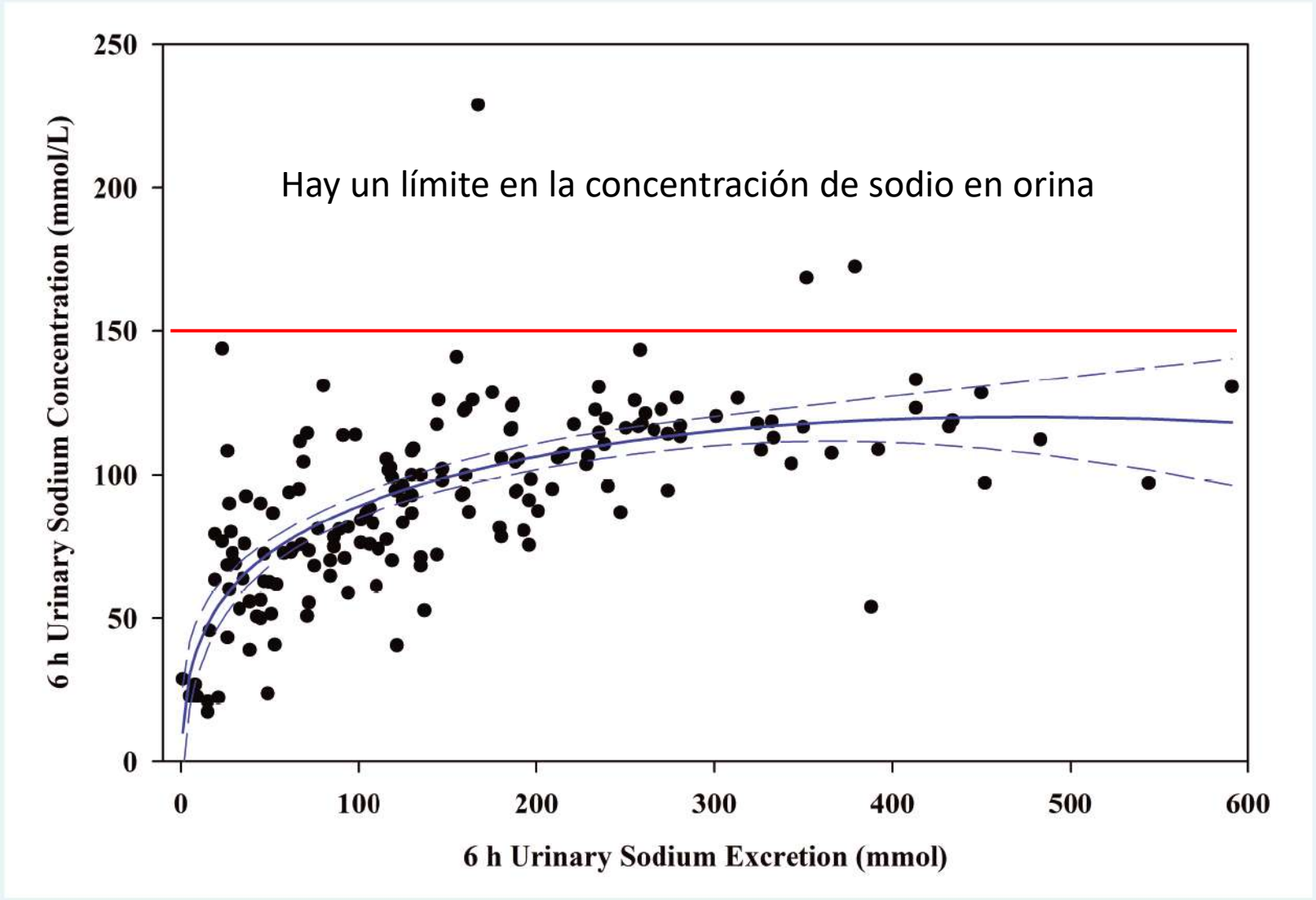
European Journal of Heart Failure 2020;22:1438-1447

Clinical importance of urinary sodium excretion in acute heart failure

Sodio en orina bajo mayor mortalidad



excreción de sodio en orina/6 h y concentración de sodio



En los rangos más bajos tanto de excreción total como de concentración hubo una asociación lineal, que se aplanó con una mayor excreción urinaria total de sodio, casi ningún paciente tuvo una concentración urinaria de sodio >150 mmol/L

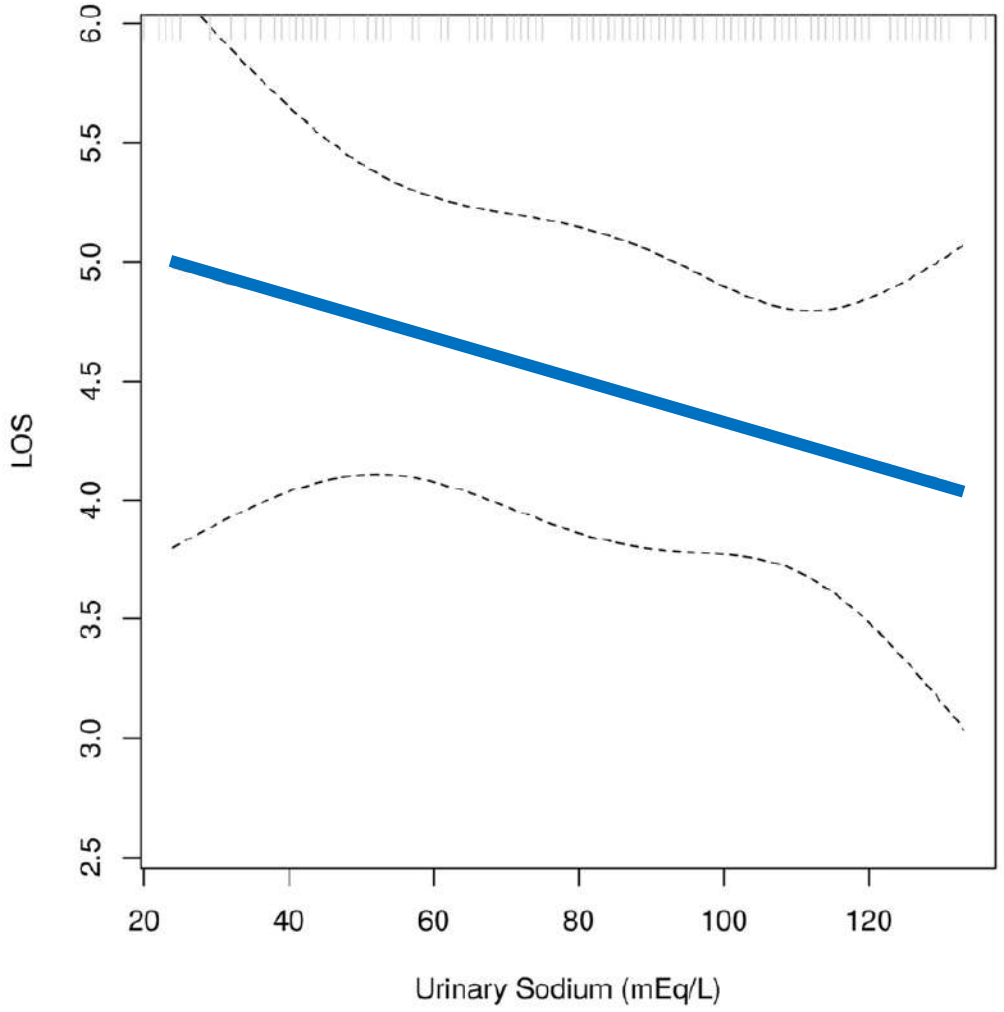
ORIGINAL RESEARCH Open Access

Markers of diuretic resistance in emergency department patients with acute heart failure

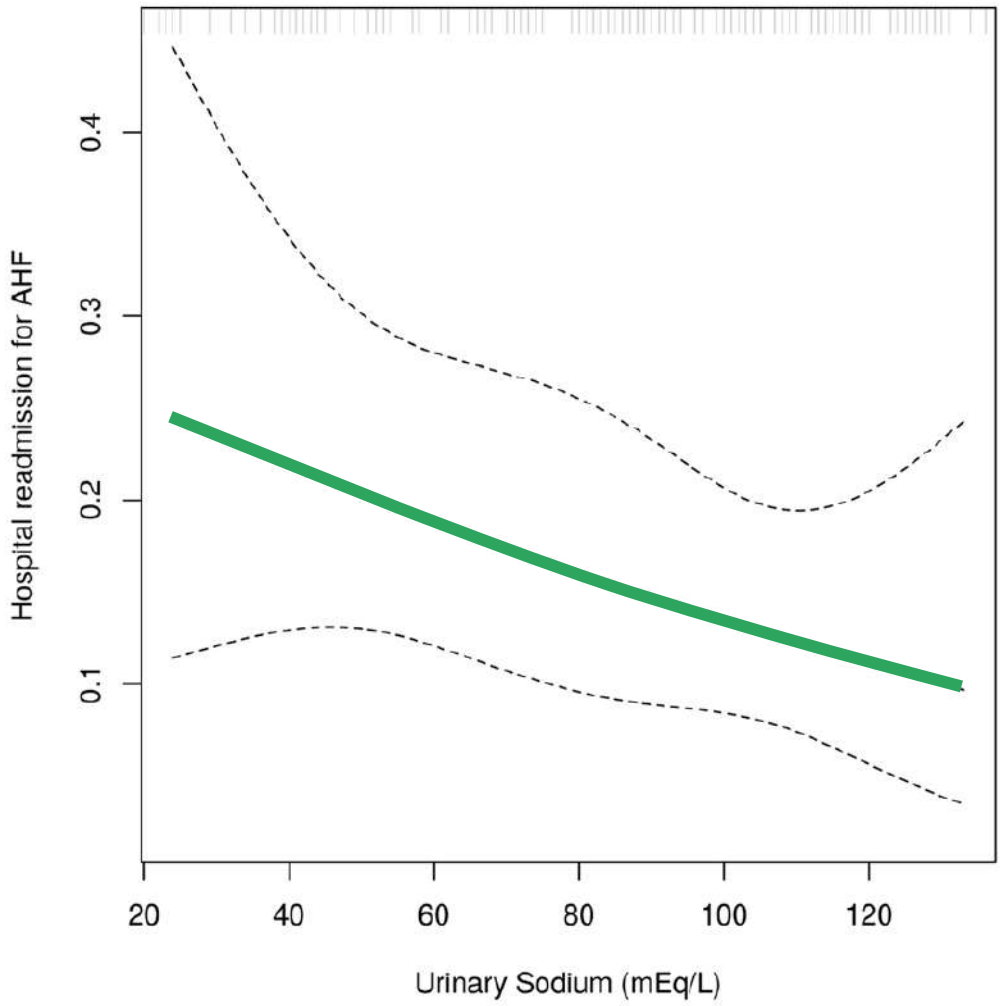
Andrew Doering[1], Cathy A. Jenkins[2], Alan B. Storrow[1], JoAnn Lindenfeld[3], Gregory J. Fermann[4], Karen F. Miller[1], Matthew Sperling[4] and Sean P. Collins[1*]

Patients who were diuretic resistant based on a spot urinary Na <50 meq/L had a higher rate of hospital readmission for AHF compared to those who were not (28 vs 13%, p = 0.03).

Estancia hospitalaria



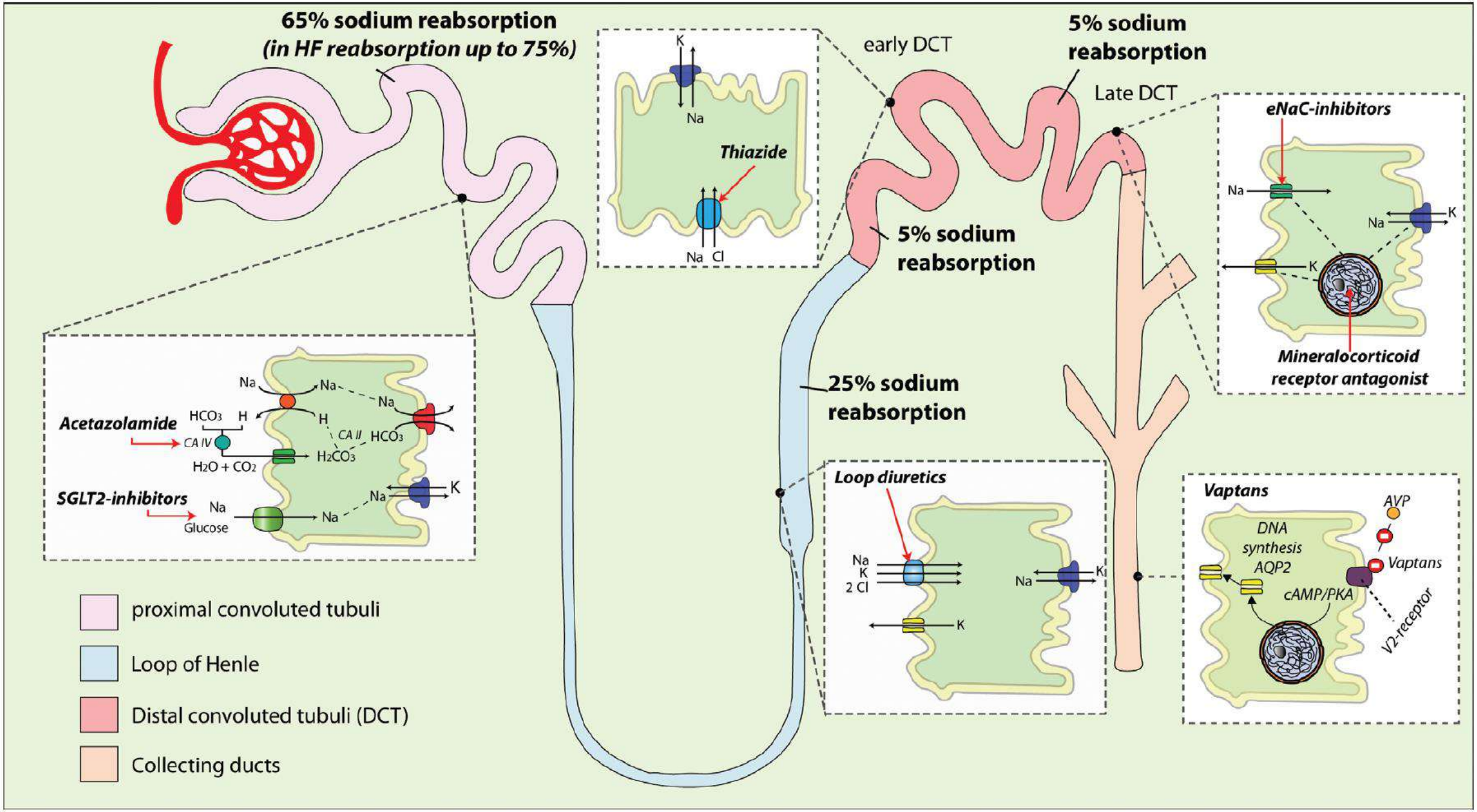
Reingreso hospitalario



Sodio en orina

Terapia personalizada

Tratamiento de la congestión hipervolémica

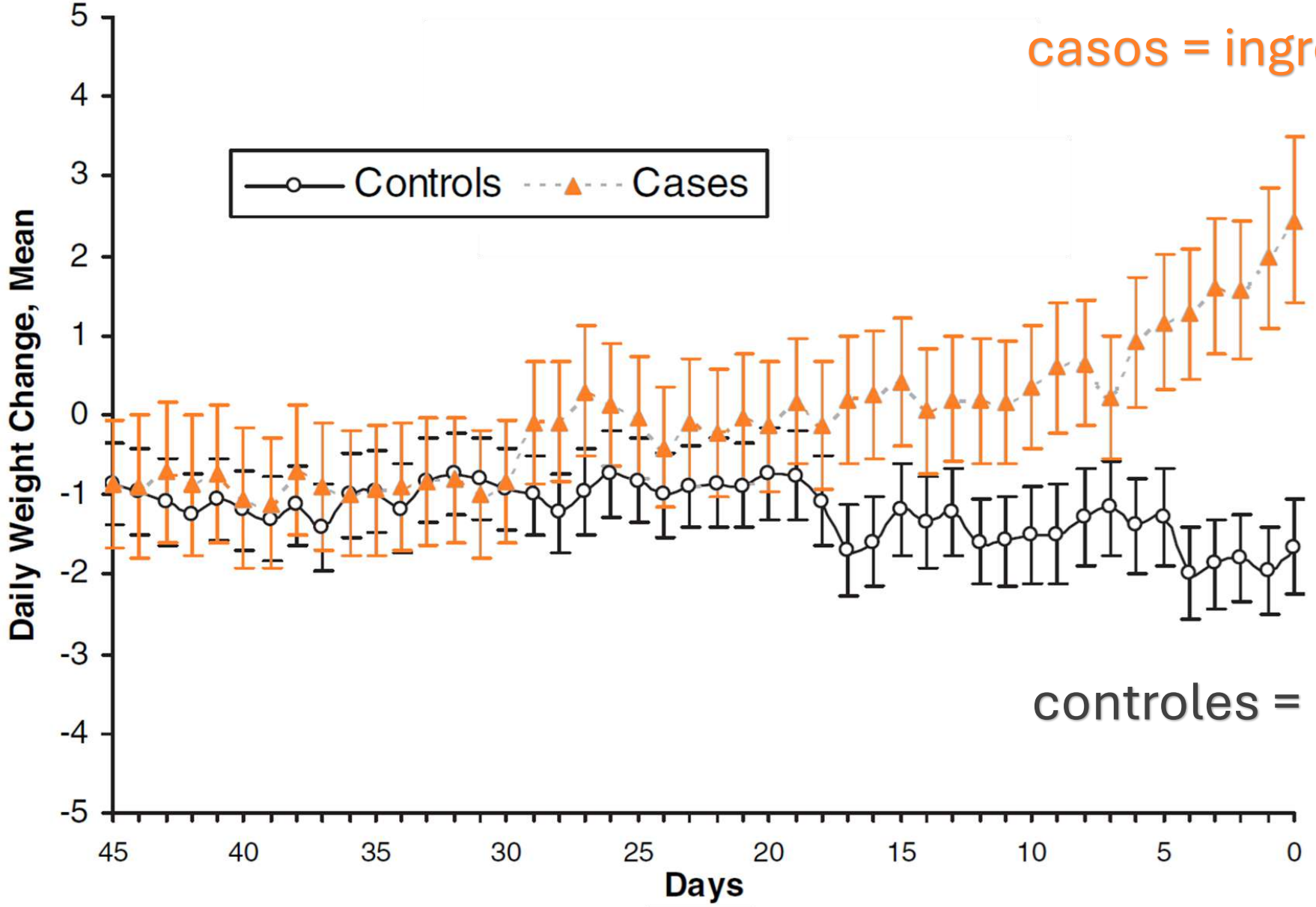

65% sodium reabsorption
(in HF reabsorption up to 75%)
early DCT
5% sodium reabsorption
Late DCT
5% sodium reabsorption
25% sodium reabsorption
Thiazide
eNaC-inhibitors
Mineralocorticoid receptor antagonist
Acetazolamide
CA IV
CA II
SGLT2-inhibitors
Glucose
Loop diuretics
Vaptans
AVP
DNA synthesis AQP2
cAMP/PKA
V2-receptor
proximal convoluted tubuli
Loop of Henle
Distal convoluted tubuli (DCT)
Collecting ducts

De entrada… todos entendemos bien la congestión como consecuencia de la hipervolemia

Circulation. 2007;116:1549-1554

Patterns of Weight Change Preceding Hospitalization for Heart Failure

Sarwat I. Chaudhry, MD; Yongfei Wang, MS; John Concato, MD, MPH;
Thomas M. Gill, MD; Harlan M. Krumholz, MD, SM



casos = ingresan por ICA

controles = no ingresan por ICA

Necesidad de ingreso hospitalario por ICA

2 libras son 0,9 Kg

Conditional Logistic Regression Models of Heart Failure Hospitalization (n=240)

Weight Gain, lbs	Case Patients, n (%)	Control Patients, n (%)	Matched Unadjusted OR (95% CI)	Matched Adjusted OR (95% CI)	Adjusted *P*
≤2	65 (54)	92 (77)	Reference group	. . .	. . .
>2 up to 5	21 (18)	16 (13)	2.40 (1.05–5.45)	2.77 (1.13–6.80)	0.026
>5 up to 10	17 (14)	8 (7)	3.81 (1.35–10.77)	4.46 (1.45–13.75)	0.009
>10	17 (14)	4 (3)	5.65 (1.81–17.65)	7.65 (2.22–26.39)	0.001

Weight gain is during 1 week preceding hospitalization of case patients. Results were adjusted for comorbid conditions and the medications shown in Table 1.

Necesidad de ingreso hospitalario por ICA

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46%

Weight gain is during 1 week preceding hospitalization of case patients. Results were adjusted for comorbid conditions and the medications shown in Table 1.

Necesidad de ingreso hospitalario por ICA

2 libras son 0,9 Kg

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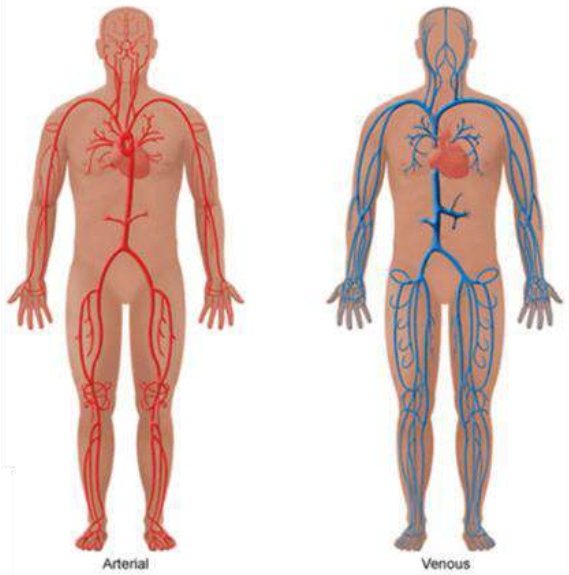
46%

Weight gain is during 1 week preceding hospitalization of case patients. Results were adjusted for comorbid conditions and the medications shown in Table 1.

Hay un perfil de pacientes congestivos sin hipervolemia

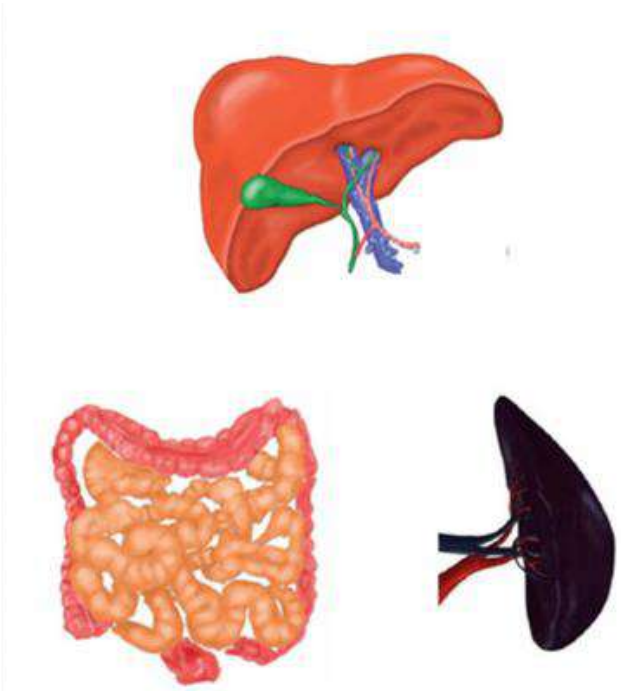
Central Compartment: Effective Circulatory Volume/Stressed Volume

30% **40%**



Splanchnic Compartment: Venous Reservoir/Unstressed Volume

20-30%



compartimento vascular esplácnico

baja resistencia y alta capacidad

Anesthesiology 2008; 108:735–48

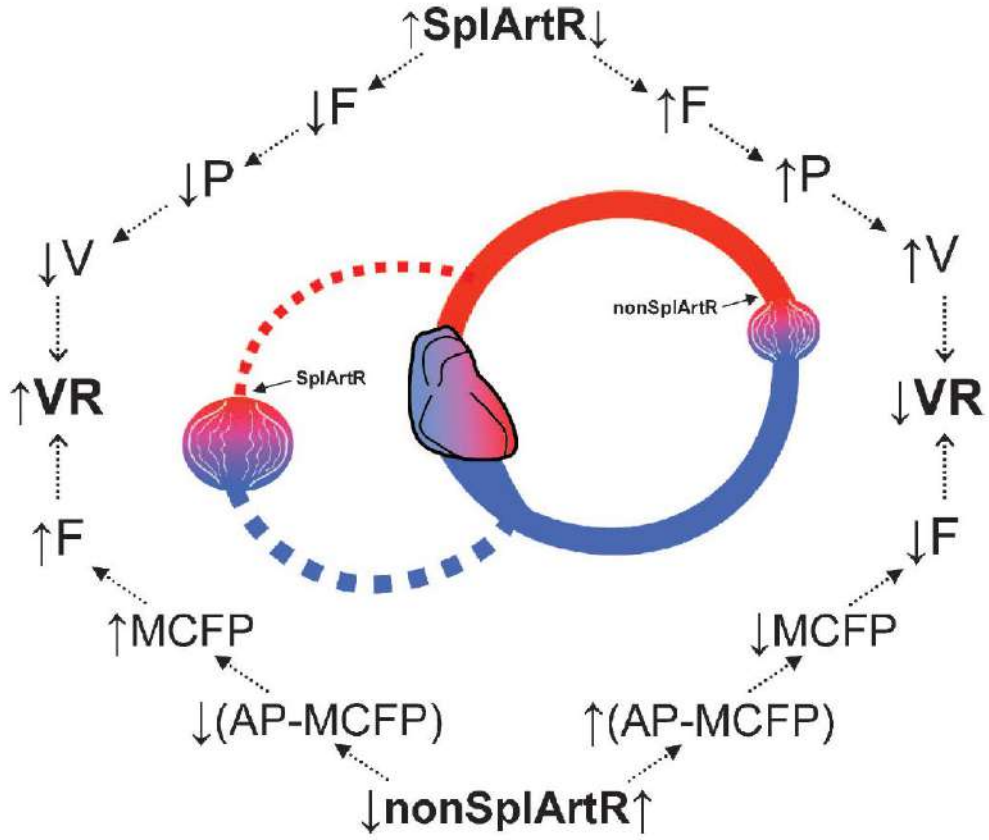
Venous Function and Central Venous Pressure

A Physiologic Story

Simon Gelman, M.D., Ph.D.*

receptores α1 y α2
receptores β2

deterioro de la capacidad de almacenamiento

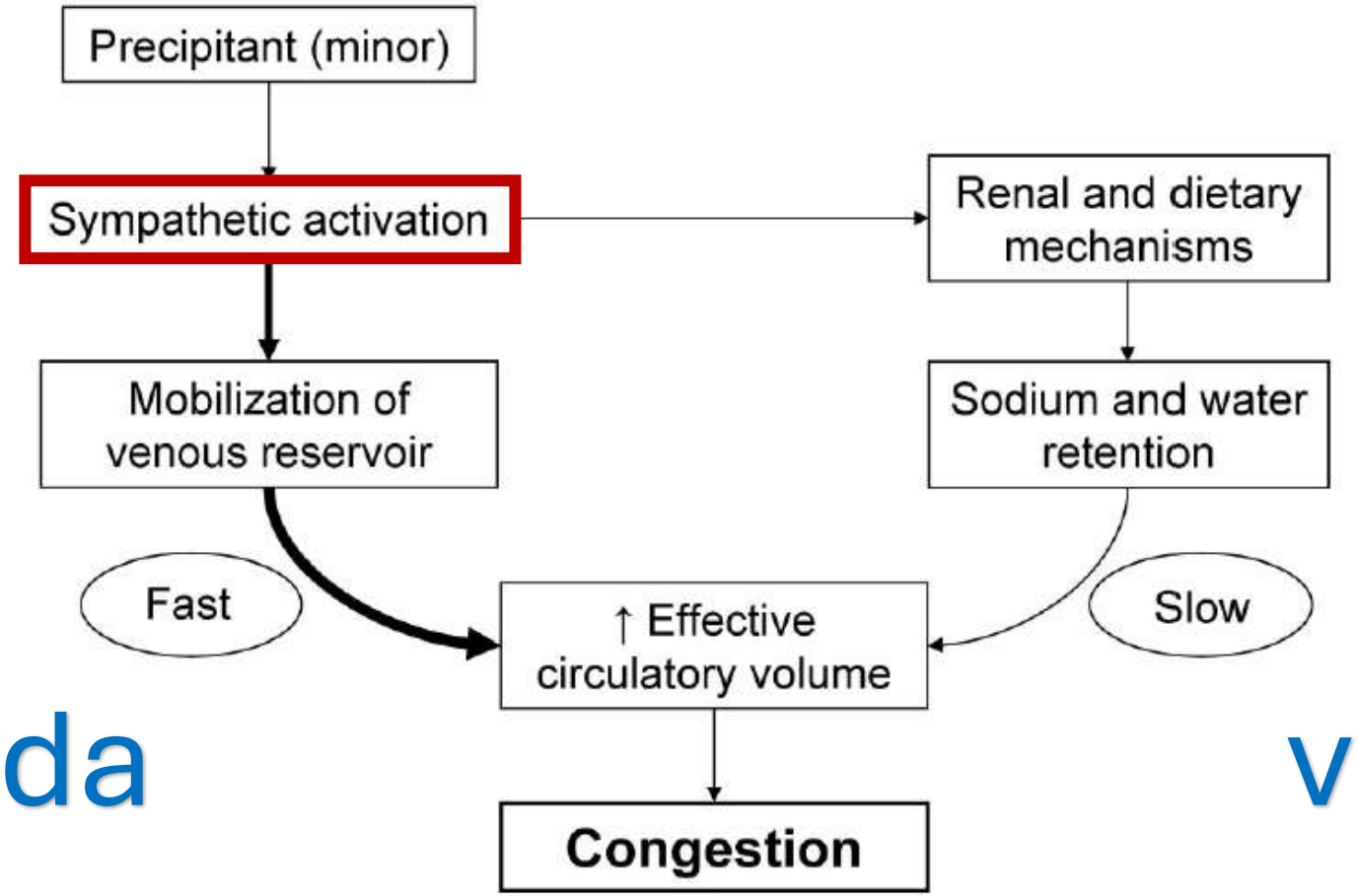


Circ Heart Fail. 2011;4:669-675

Sympathetically Mediated Changes in Capacitance

Redistribution of the Venous Reservoir as a Cause of Decompensation

Catherine Fallick, MD, FACC; Paul A. Sobotka, MD, FACP, FACC; Mark E. Dunlap, MD, FACC, FAHA



vía rápida

vía lenta

Congestión pulmonar

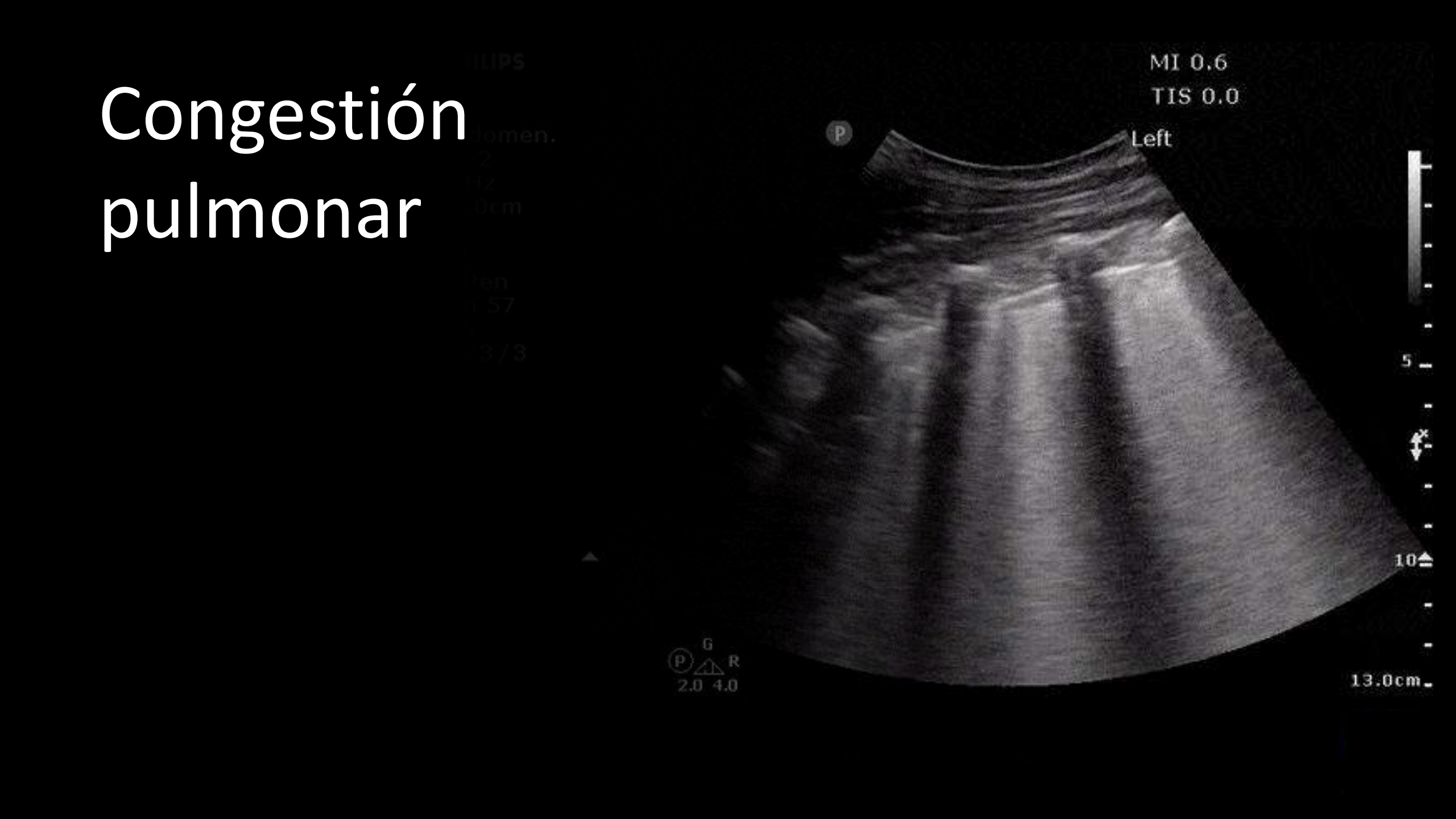
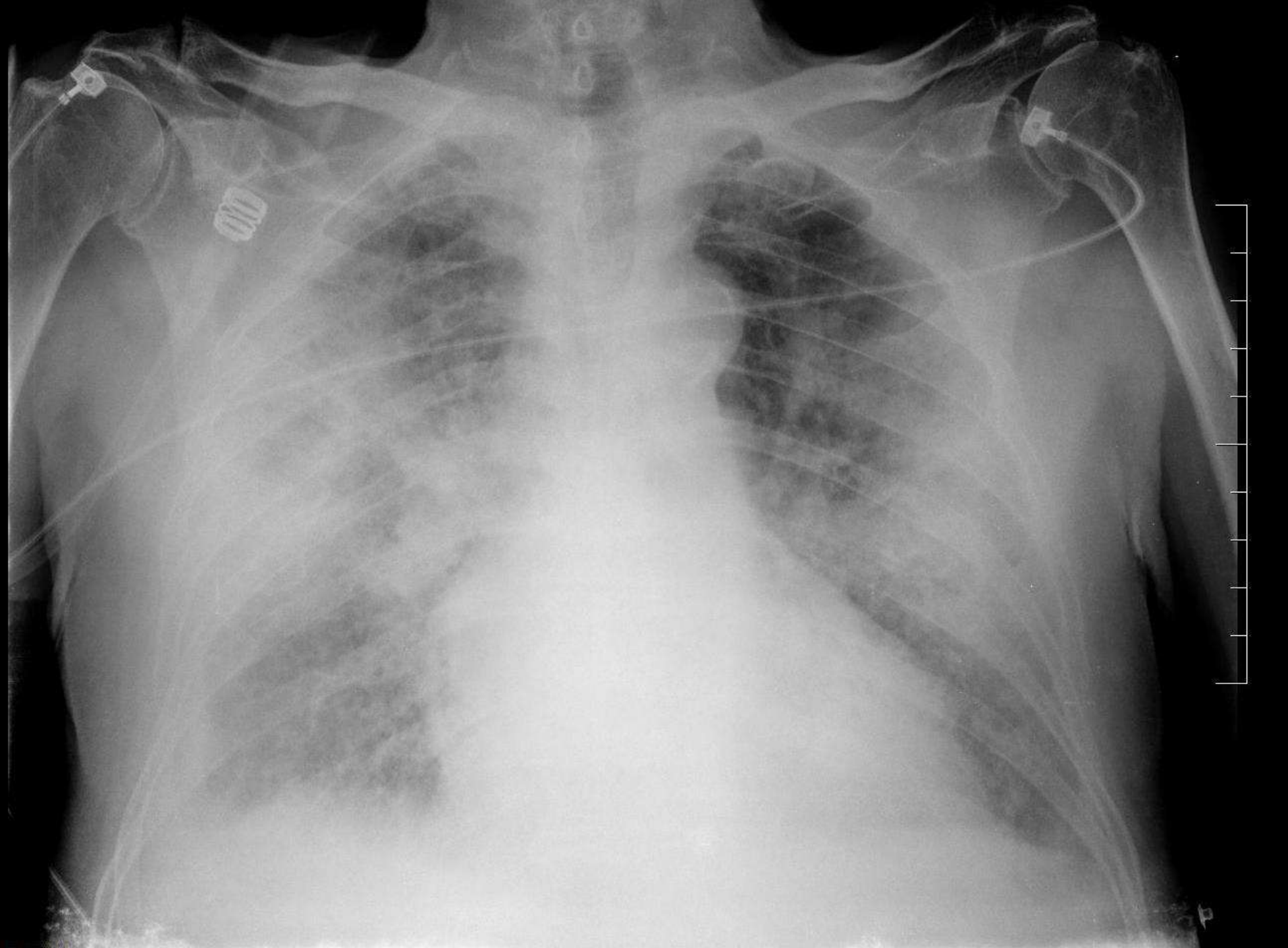
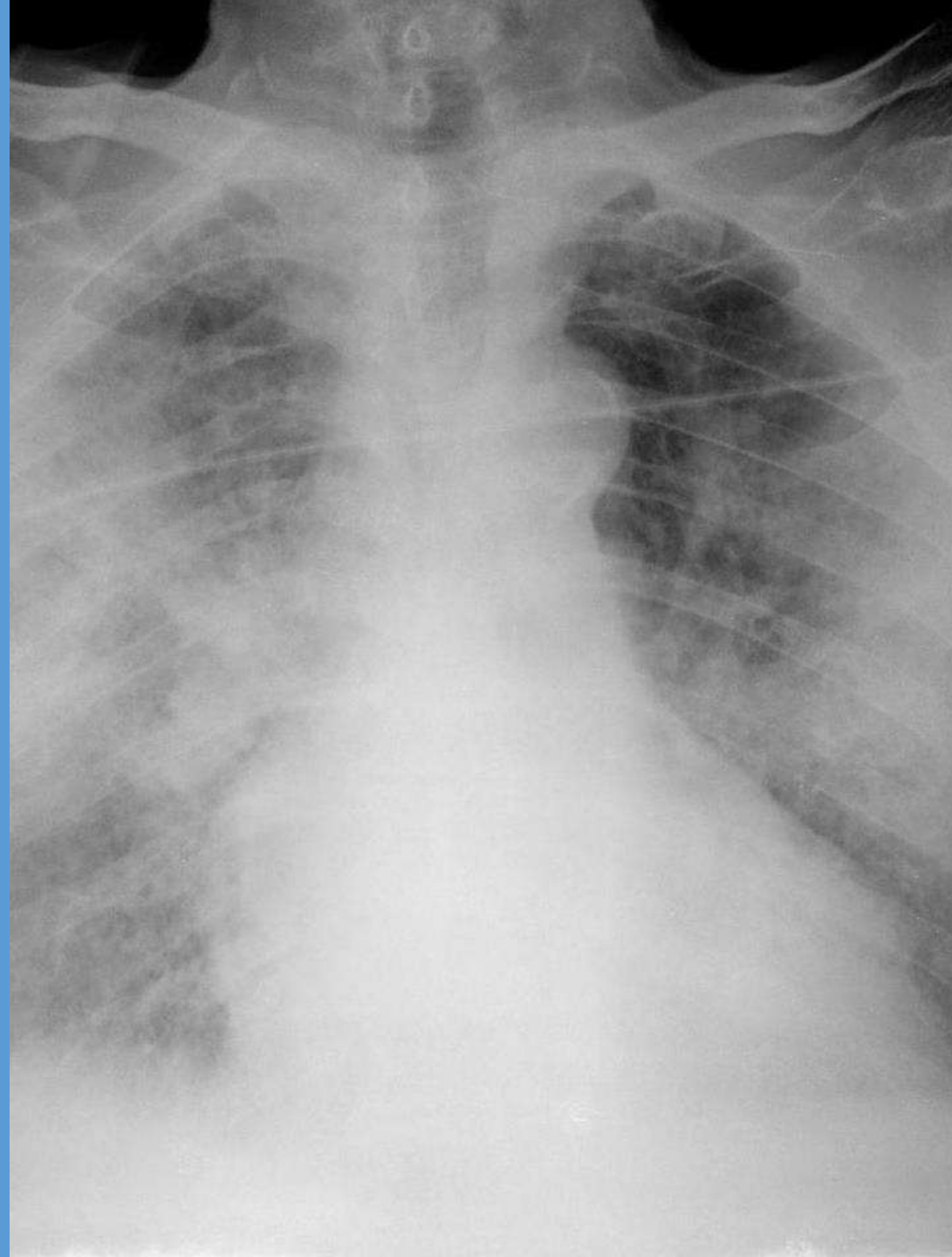


Imagen propiedad del autor con consentimiento del paciente

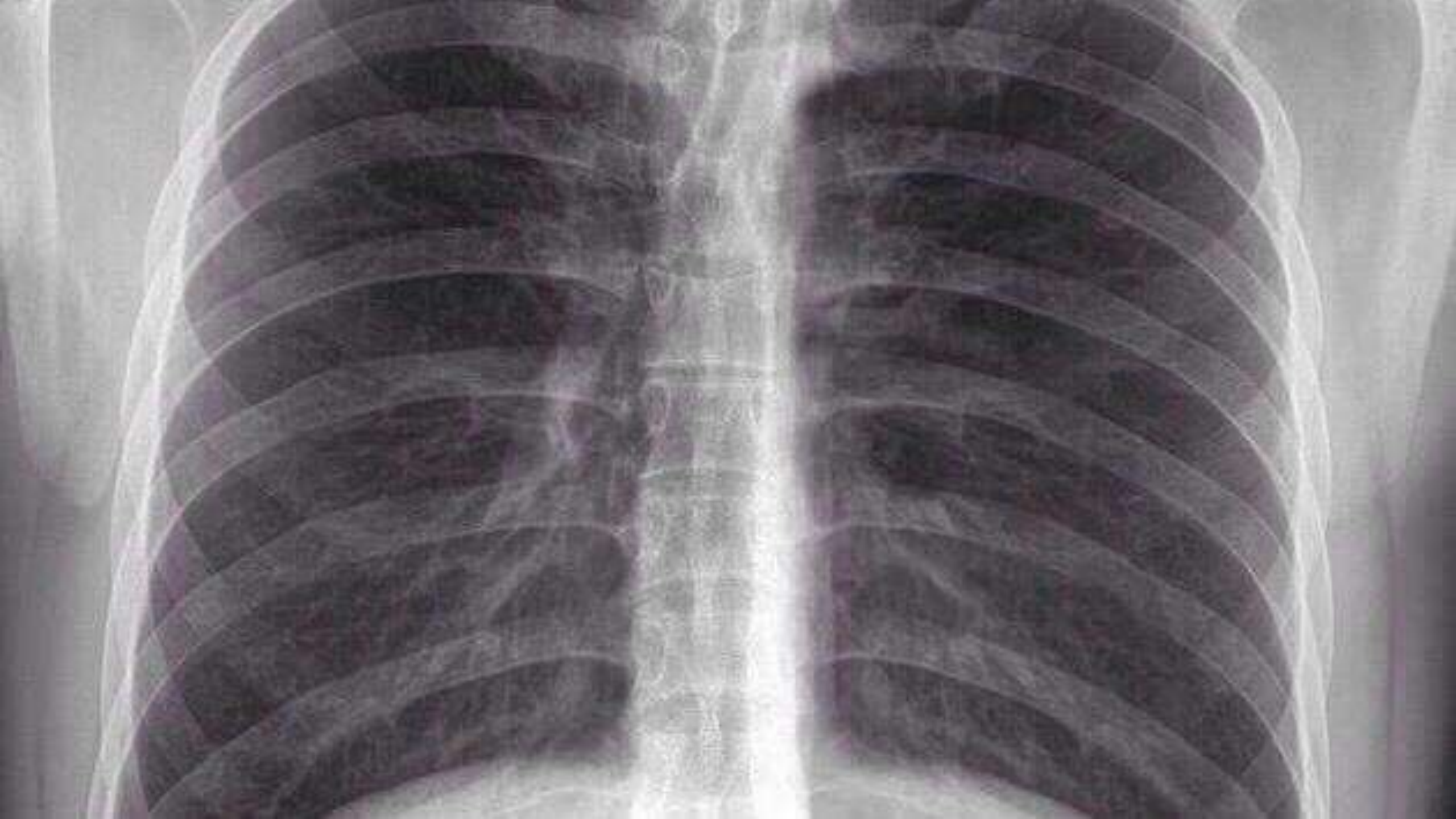


Tratamiento de la congestión NO hipervolémica




2
1

Xoc cardiogénico



ESC HF Guidelines[15]

SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion

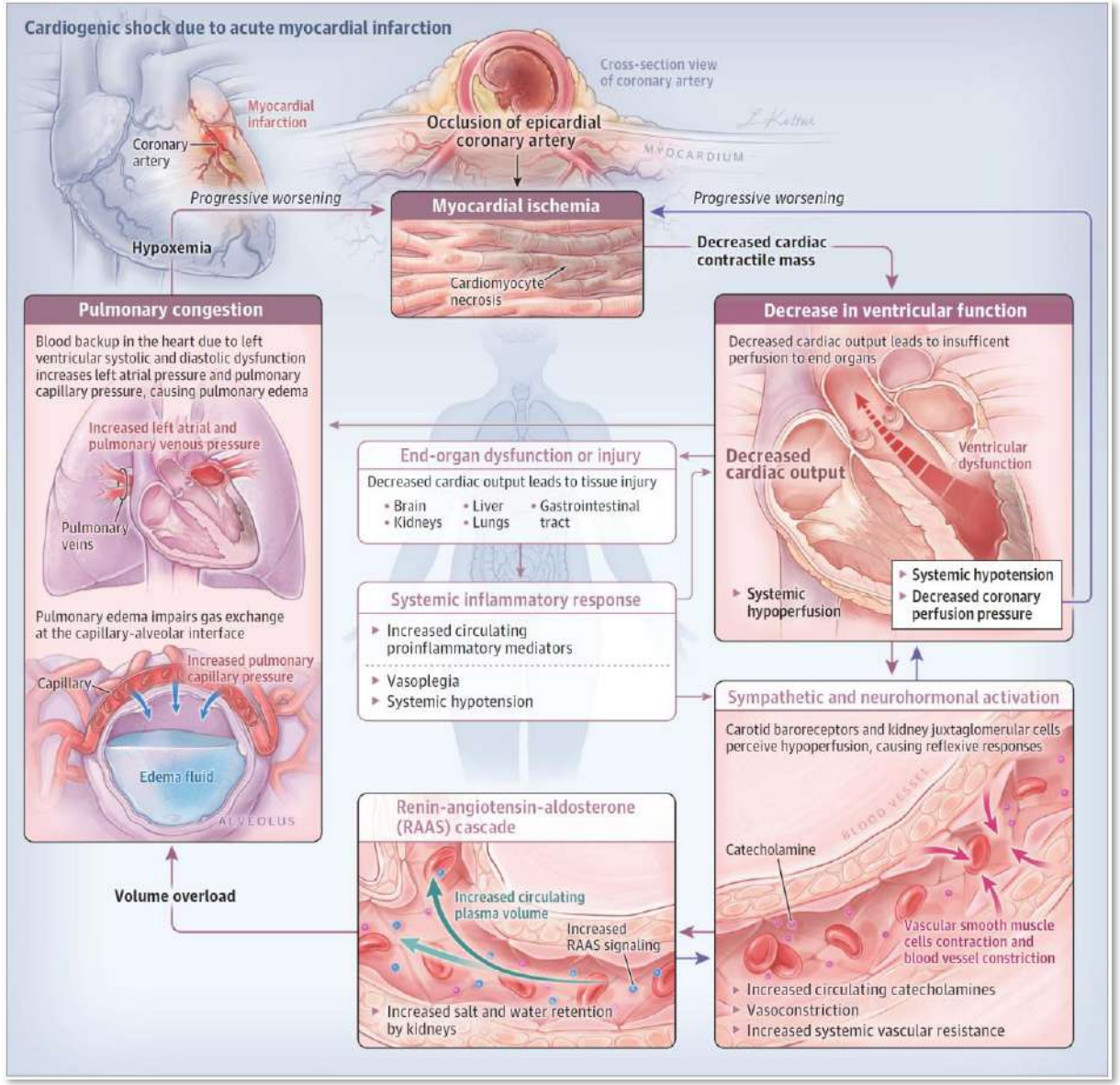
Clinical hypoperfusion:
Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure

Laboratory hypoperfusion:
Metabolic acidosis, elevated serum lactate, elevated serum creatinine

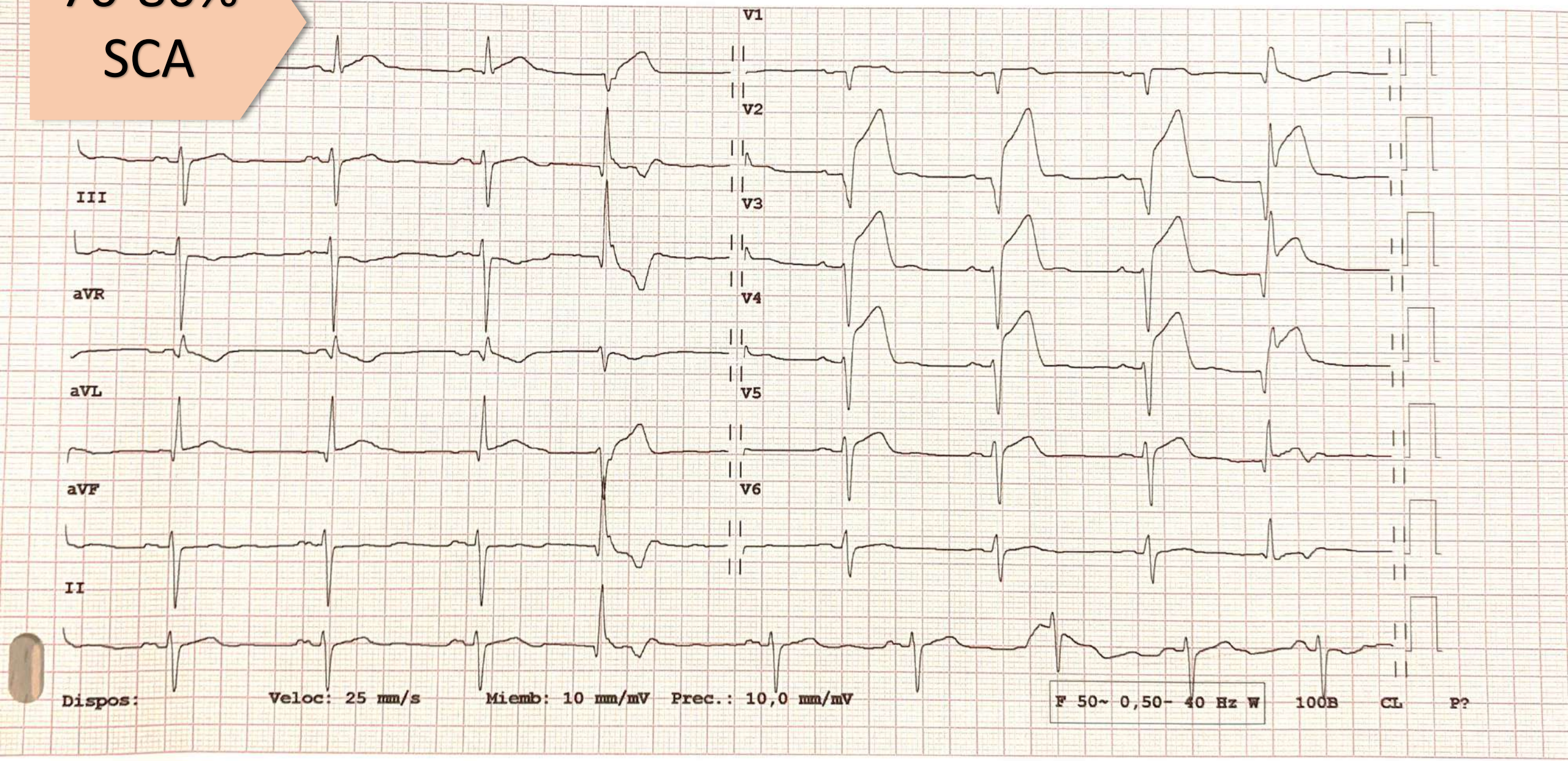
Hipotensión con hipoperfusión

Órganos diana


infarto
↓gasto cardiaco
Activación simpática
Activación RAA
Congestión pulmonar
↓ oxigenación

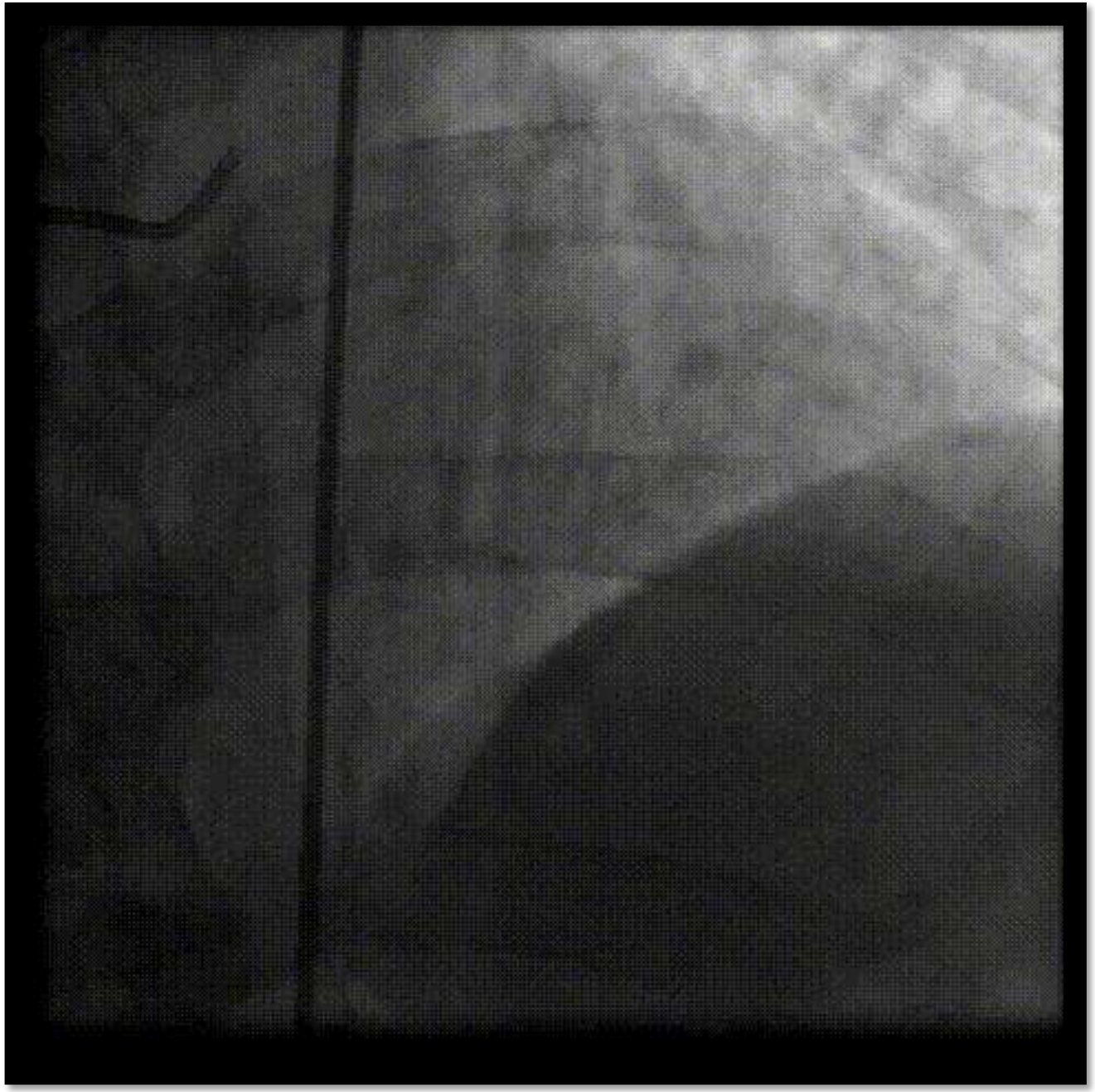

Cardiogenic shock due to acute myocardial infarction
Cross-section view of coronary artery
Myocardial infarction
Coronary artery
Occlusion of epicardial coronary artery
MYOCARDIUM
Progressive worsening
Myocardial ischemia
Progressive worsening
Hypoxemia
Decreased cardiac contractile mass
Cardiomyocyte necrosis
Pulmonary congestion
Blood backup in the heart due to left ventricular systolic and diastolic dysfunction increases left atrial pressure and pulmonary capillary pressure, causing pulmonary edema
Increased left atrial and pulmonary venous pressure
Pulmonary veins
Pulmonary edema impairs gas exchange at the capillary-alveolar interface
Capillary
Increased pulmonary capillary pressure
Edema fluid
ALVEOLUS
Volume overload
Decrease in ventricular function
Decreased cardiac output leads to insufficent perfusion to end organs
Decreased cardiac output
Ventricular dysfunction
Systemic hypoperfusion
Systemic hypotension
Decreased coronary perfusion pressure
End-organ dysfunction or injury
Decreased cardiac output leads to tissue injury
Brain
Kidneys
Liver
Lungs
Gastrointestinal tract
Systemic inflammatory response
Increased circulating proinflammatory mediators
Vasoplegia
Systemic hypotension
Sympathetic and neurohormonal activation
Carotid baroreceptors and kidney juxtaglomerular cells perceive hypoperfusion, causing reflexive responses
Catecholamine
BLOOD VESSEL
Vascular smooth muscle cells contraction and blood vessel constriction
Increased circulating catecholamines
Vasoconstriction
Increased systemic vascular resistance
Renin-angiotensin-aldosterone (RAAS) cascade
Increased circulating plasma volume
Increased RAAS signaling
Increased salt and water retention by kidneys

70-80%
SCA


V1
V2
III
V3
aVR
V4
aVL
V5
aVF
V6
II
Dispos:
Veloc: 25 mm/s
Miemb: 10 mm/mV
Prec.: 10,0 mm/mV
F 50~ 0,50- 40 Hz W
100B
CL
P?

Código IAM

emergències mèdiques



Manejo del SC

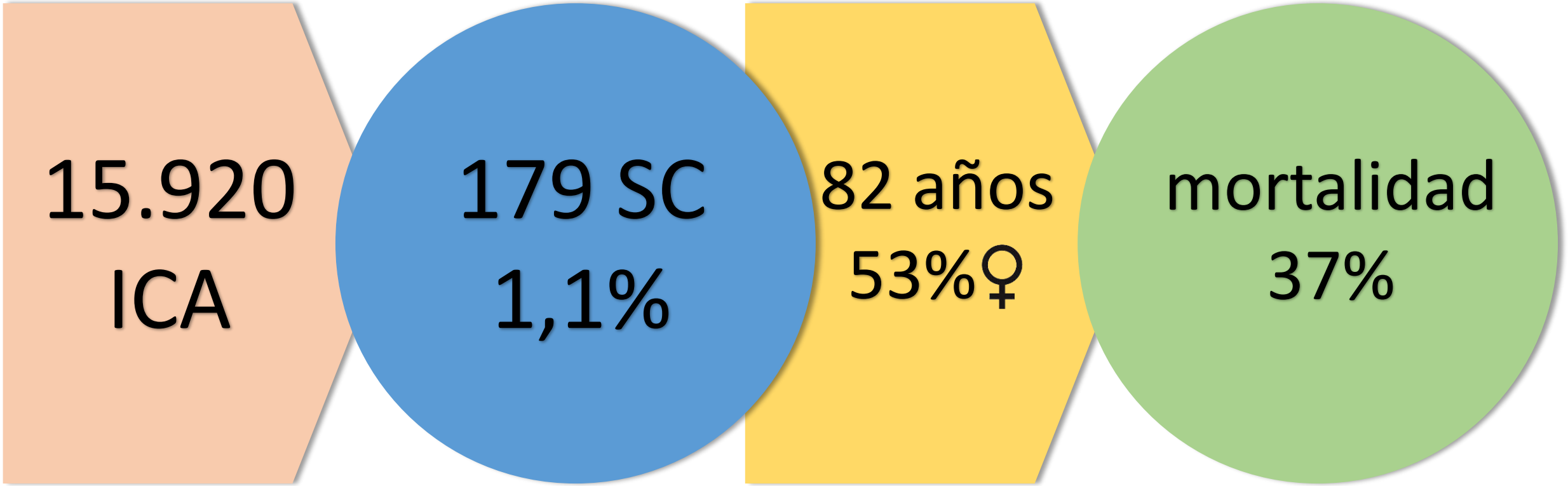
- Manejo del IAM
- Soporte de la ventilación
- Soporte farmacológico
- Soporte renal
- Soporte mecánico

Soporte mecánico continuo (SMC)

Emergencias 2025. Ahead of print

Características epidemiológicas, clínicas y evolutivas de los pacientes con insuficiencia cardiaca aguda y shock cardiogénico diagnosticados en urgencias

Begoña Espinosa[1*], Pere Llorens[1*], Javier Jacob[2], Víctor Gil[3], Aitor Alquézar[4], Elena Dieste Ballarín[5], María Pilar López-Díez[6], José Manuel Garrido[7], Sonia del Amo[8], Josep Tost[9], Pilar Paz Arias[10], Lluís Llauger[11], Judith Gorlicki[12], Josep Masip[13], Òscar Miró[3] (en representación del grupo de investigación ICA-SEMES)



Insuficiencia cardiaca terminal
Manejo paliativo

Tratamiento del Shock cardiogénico

shock center