

XXXV

Congreso Nacional de la Sociedad Española de Medicina Interna (SEMI)

IV Congreso Ibérico de Medicina Interna

II Congreso de la Sociedad de Medicina Interna de la Región de Murcia

19-21 de Noviembre de 2014
Auditorio y Centro de Congresos
Víctor Villegas. Murcia





1542

Universidad
Zaragoza



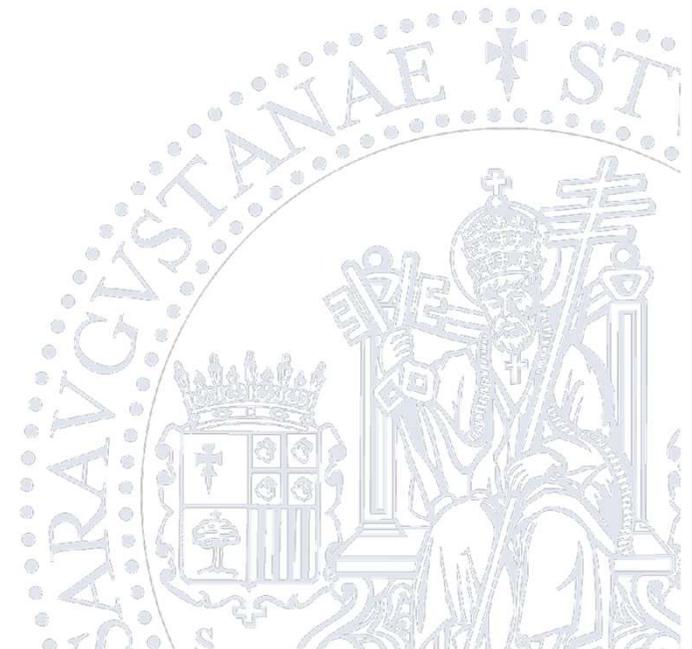
Hospital Clínico Universitario
"Lozano Blesa" de Zaragoza



IIS Aragón

Instituto de Investigación
Sanitaria Aragón

Juan Ignacio Pérez Calvo



Mesa redonda 25: “Avances en el conocimiento de la IC aguda: de la concepción hemodinámica a la protección tisular”

1. Fisiopatología de la ICA que explica su mal pronóstico
2. ¿Qué hay que mejorar en el abordaje clínico del paciente con IC aguda?
3. Retos terapéuticos de los perfiles de pacientes más vulnerables
4. Nuevas evidencias terapéuticas en IC crónica

Moderadores:

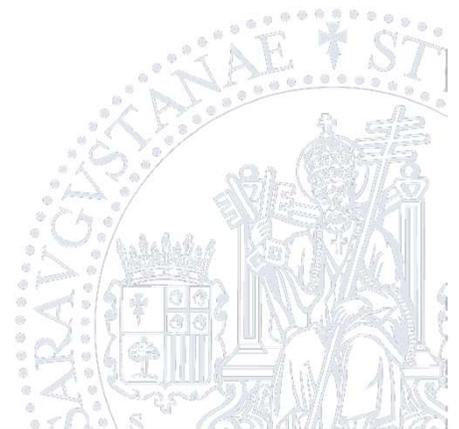
F. Formiga
P. Bettencourt



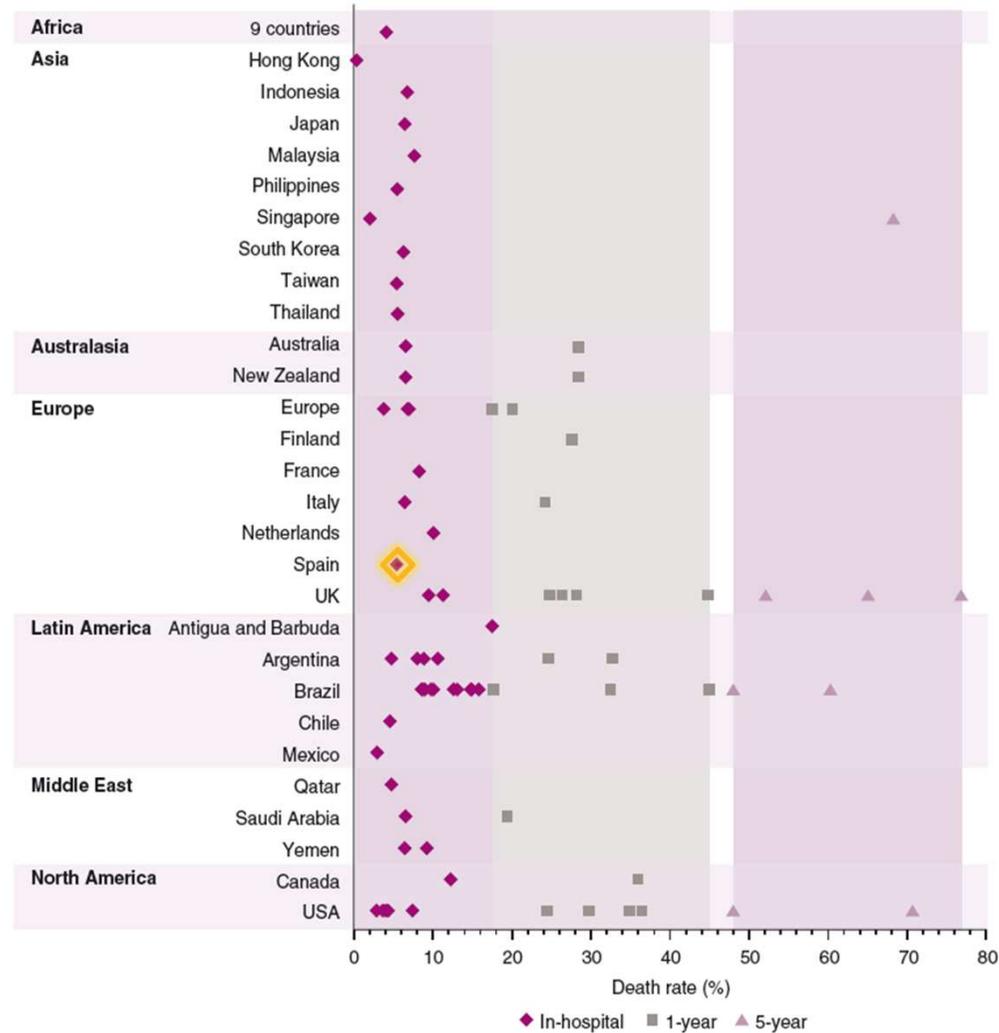
PROBLEMA



*Fisiopatología de la ICA que
explica su mal pronóstico*

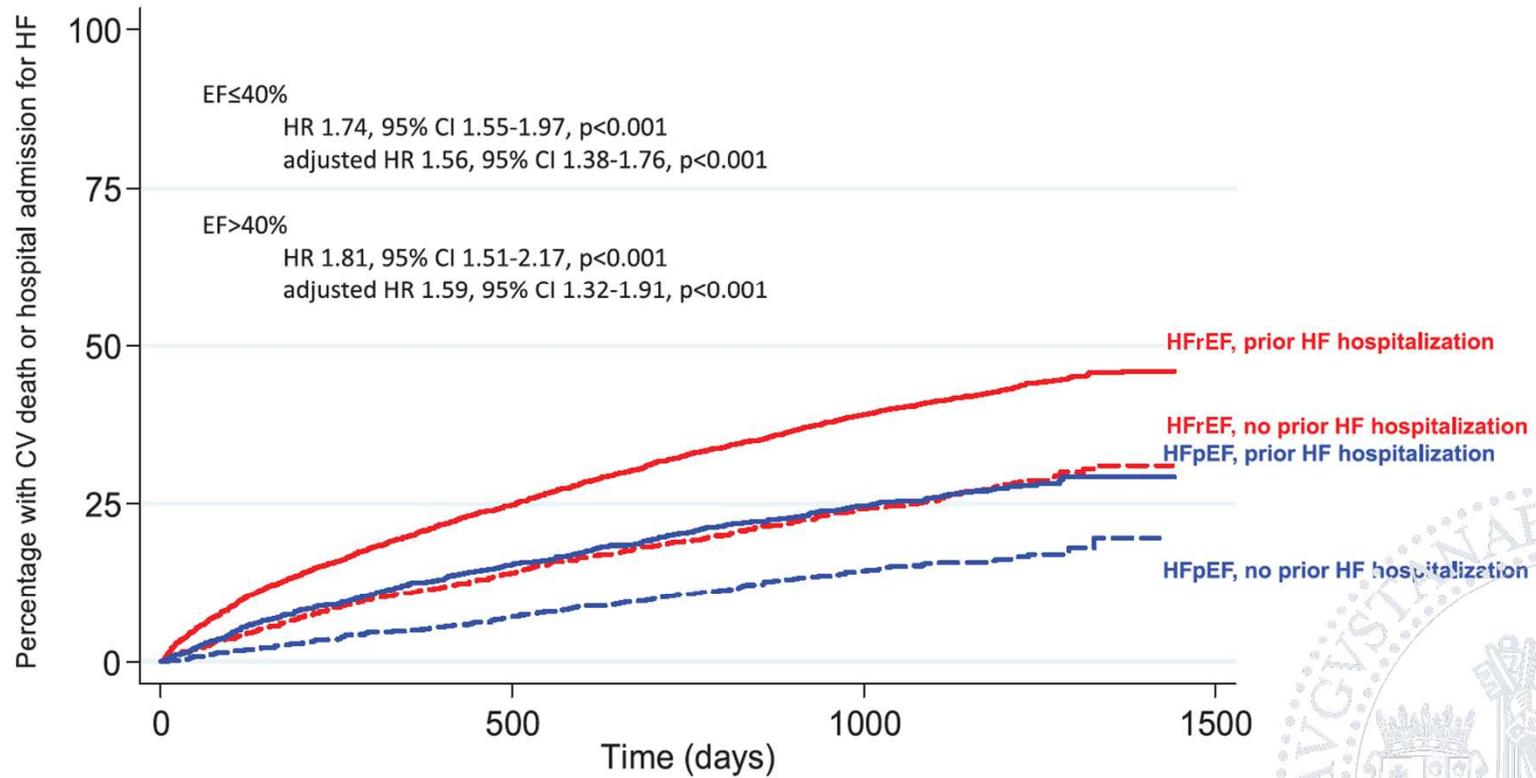


PRONÓSTICO

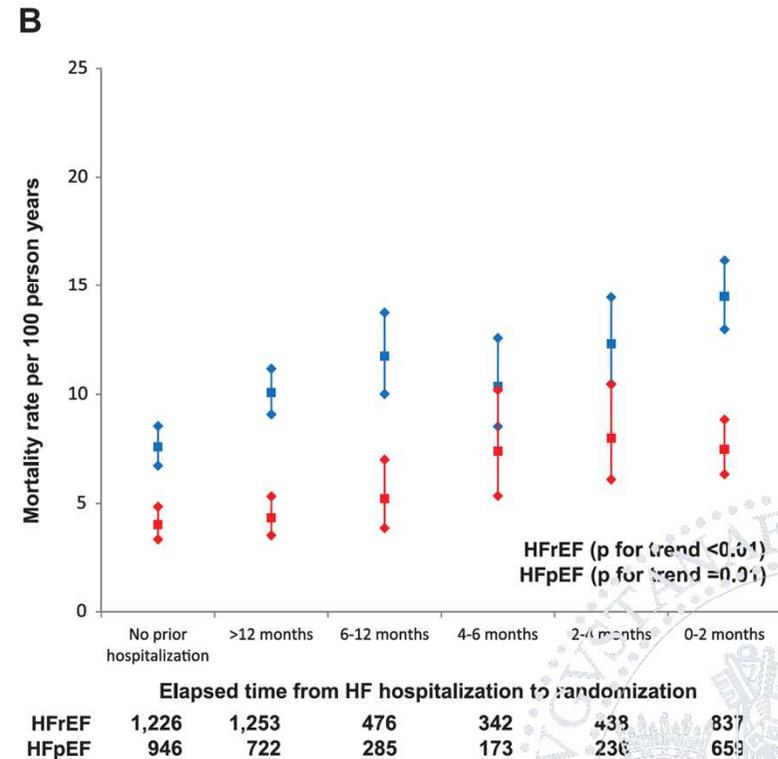
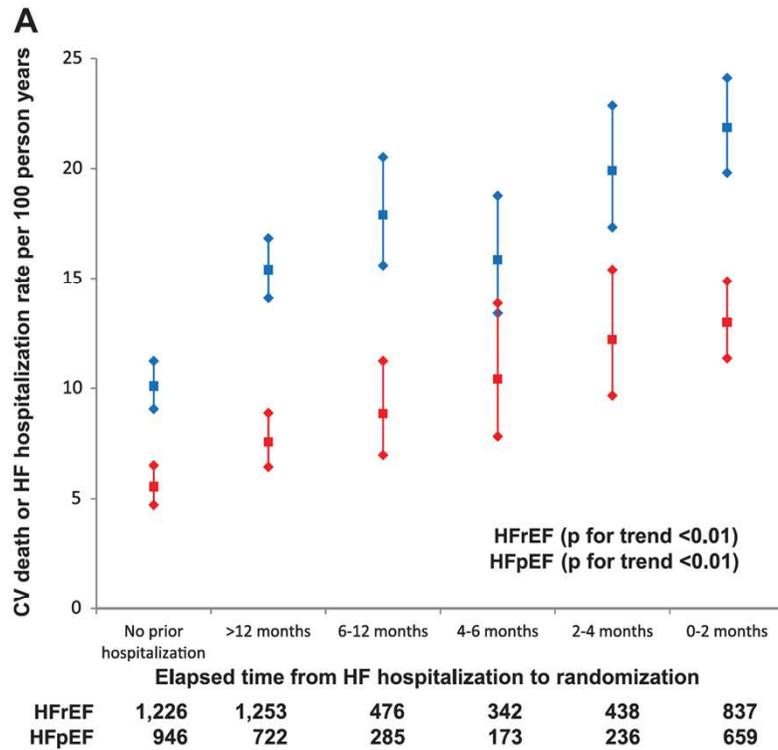


PRONÓSTICO

7599 pacientes CHARM



PRONÓSTICO



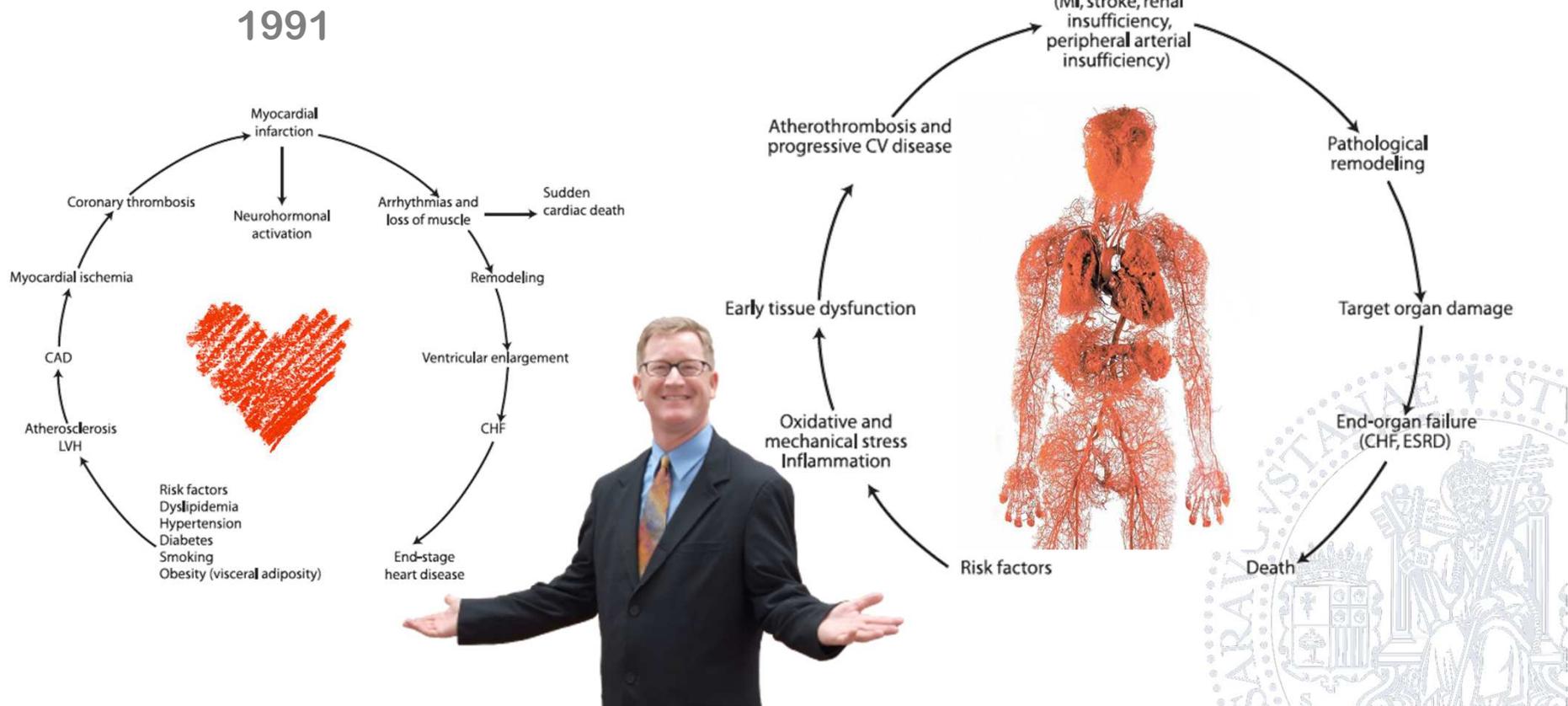
FISIOPATOLOGÍA DE LA ICA

- El “continuum” cardiovascular
- Órganos diana de la IC aguda
- Mecanismos de lesión

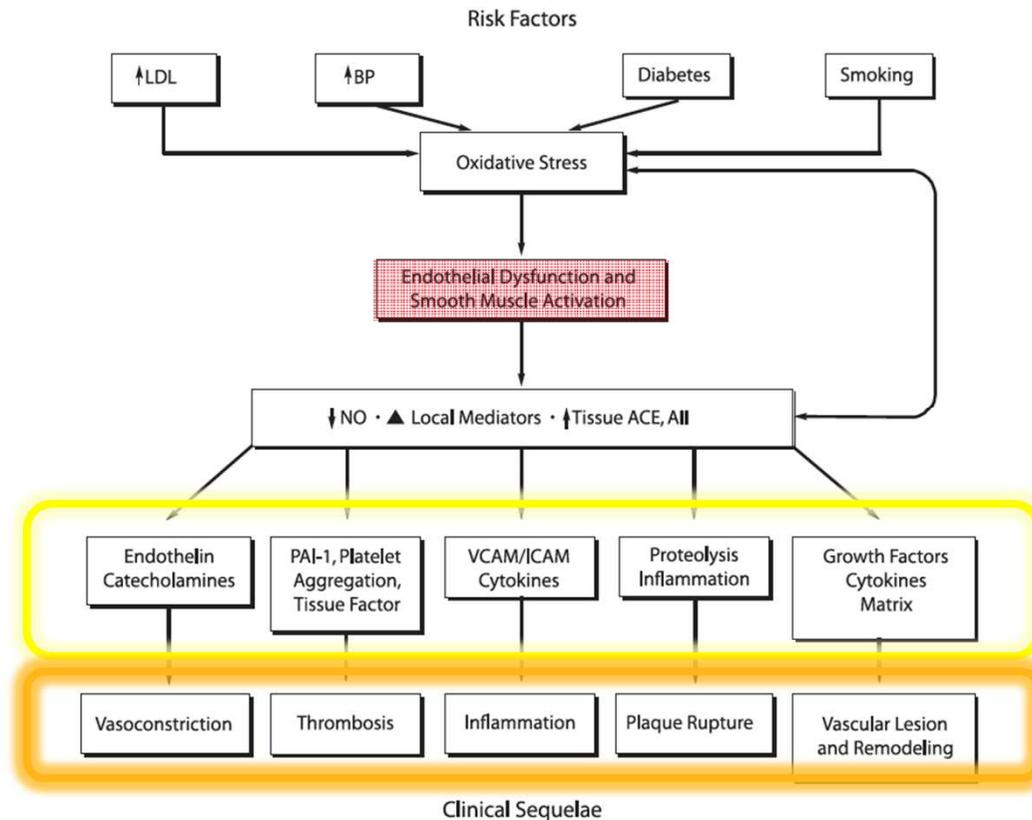


“CONTINUUM” CARDIOVASCULAR

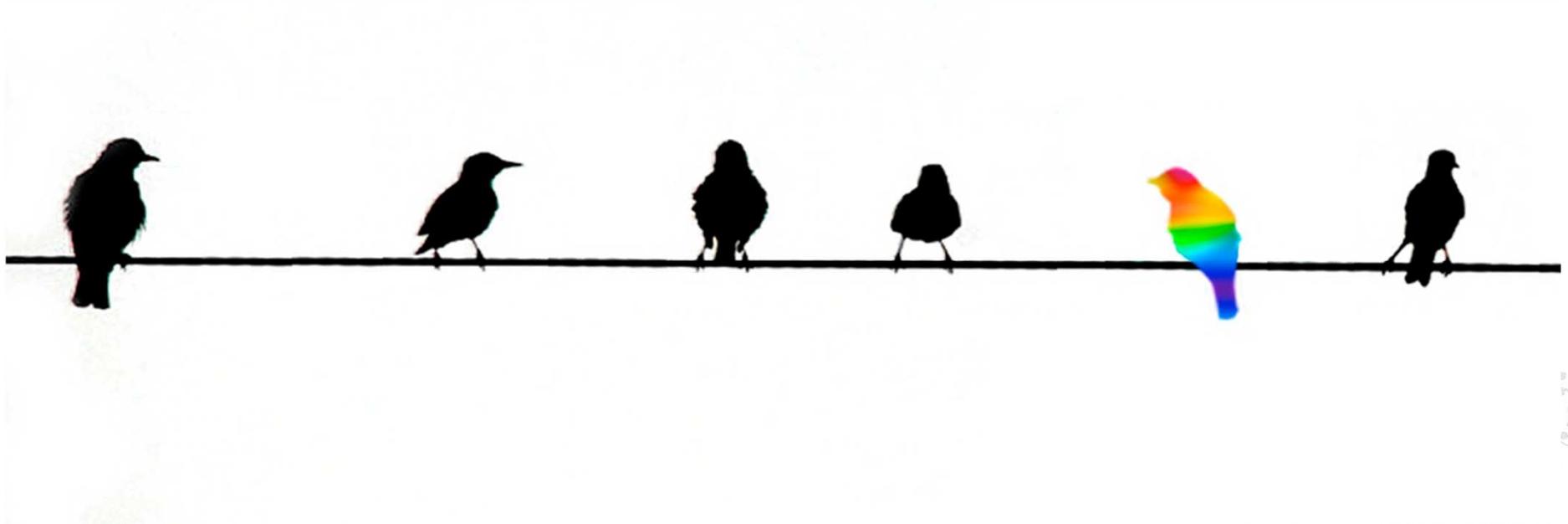
2006



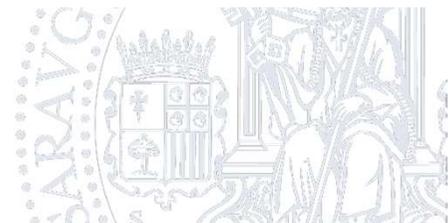
“CONTINUUM” CARDIOVASCULAR



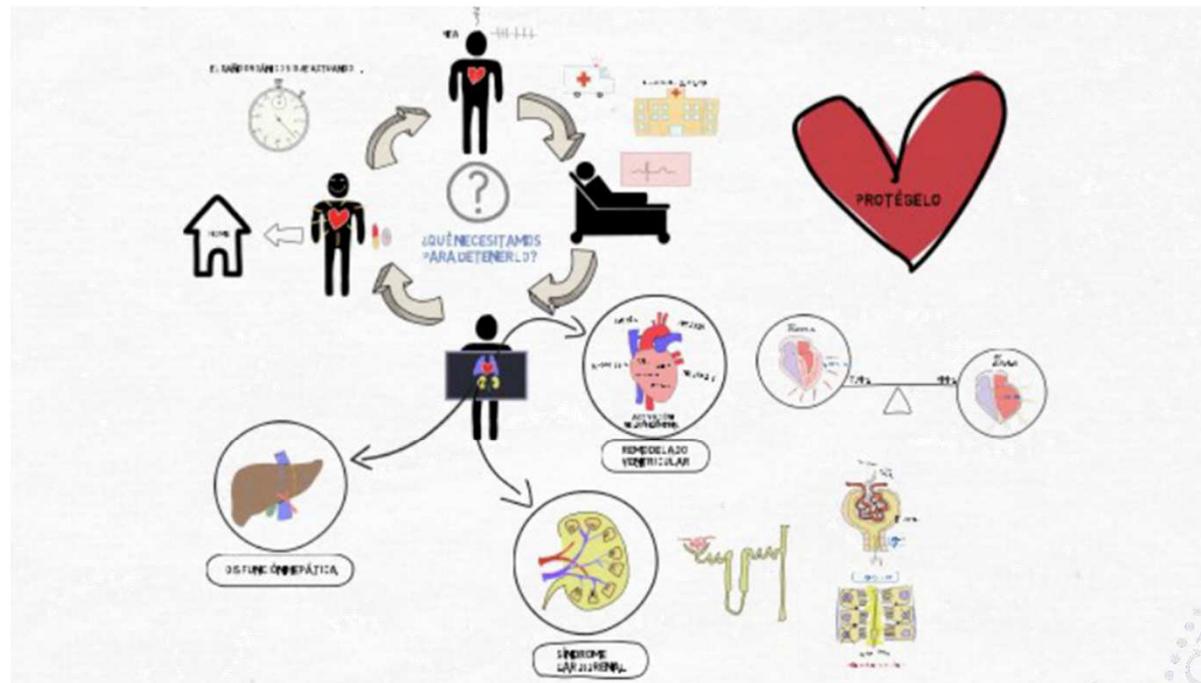
ÓRGANOS DIANA



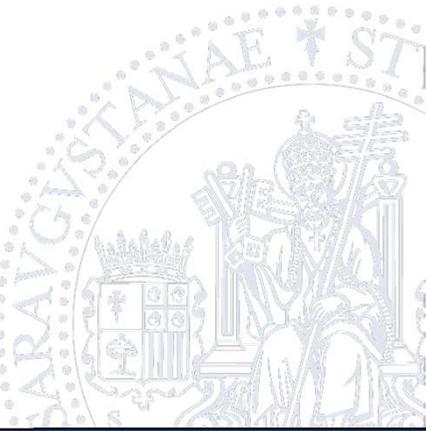
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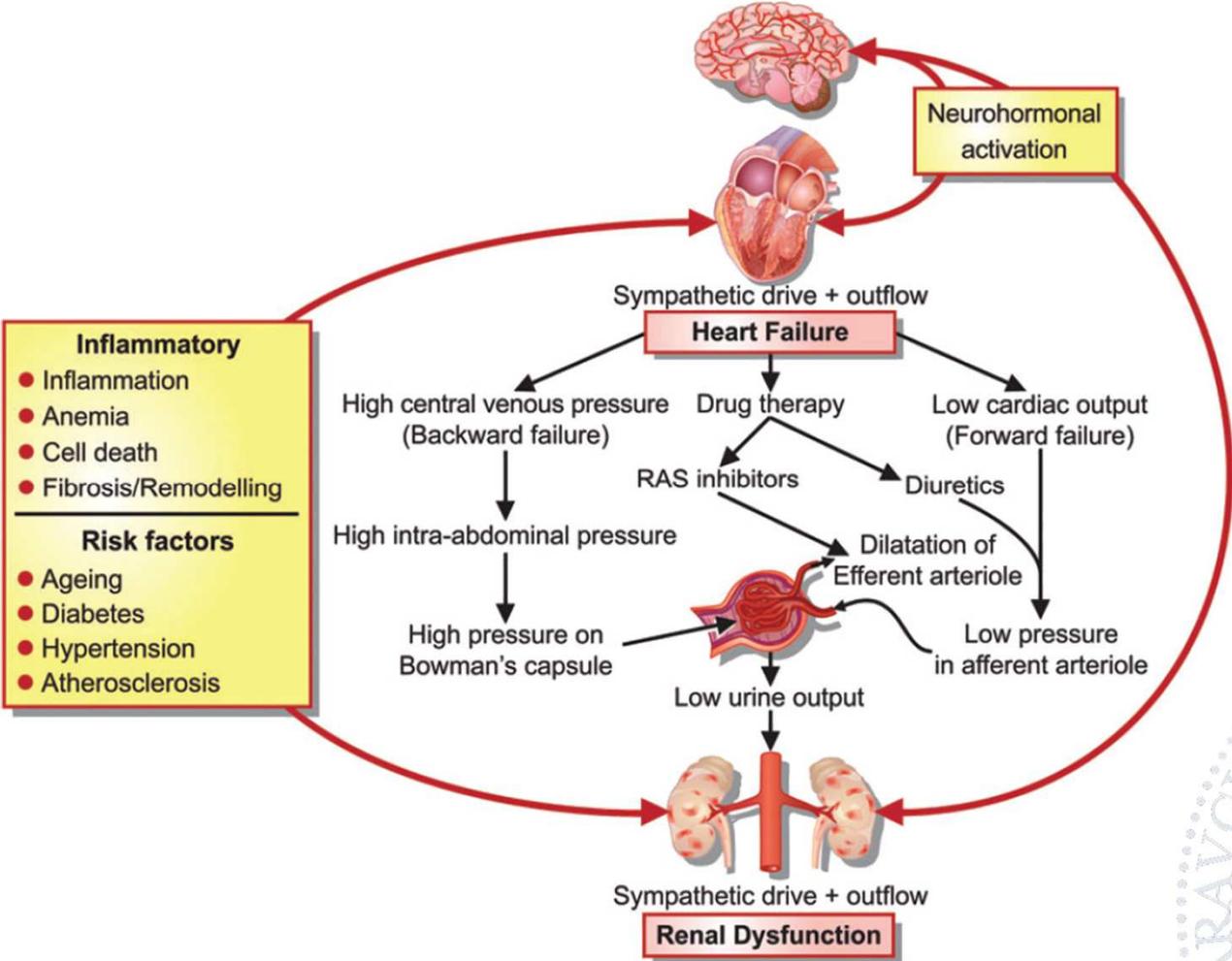
VISIÓN INTEGRAL



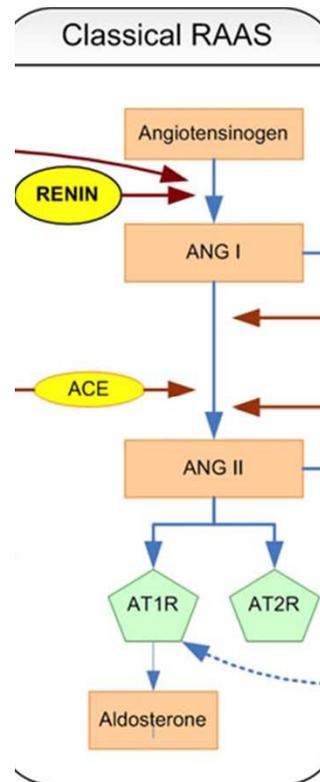
MECANISMOS DE LESIÓN Y COMPENSACIÓN



MECANISMOS DE LESIÓN Y COMPENSACIÓN

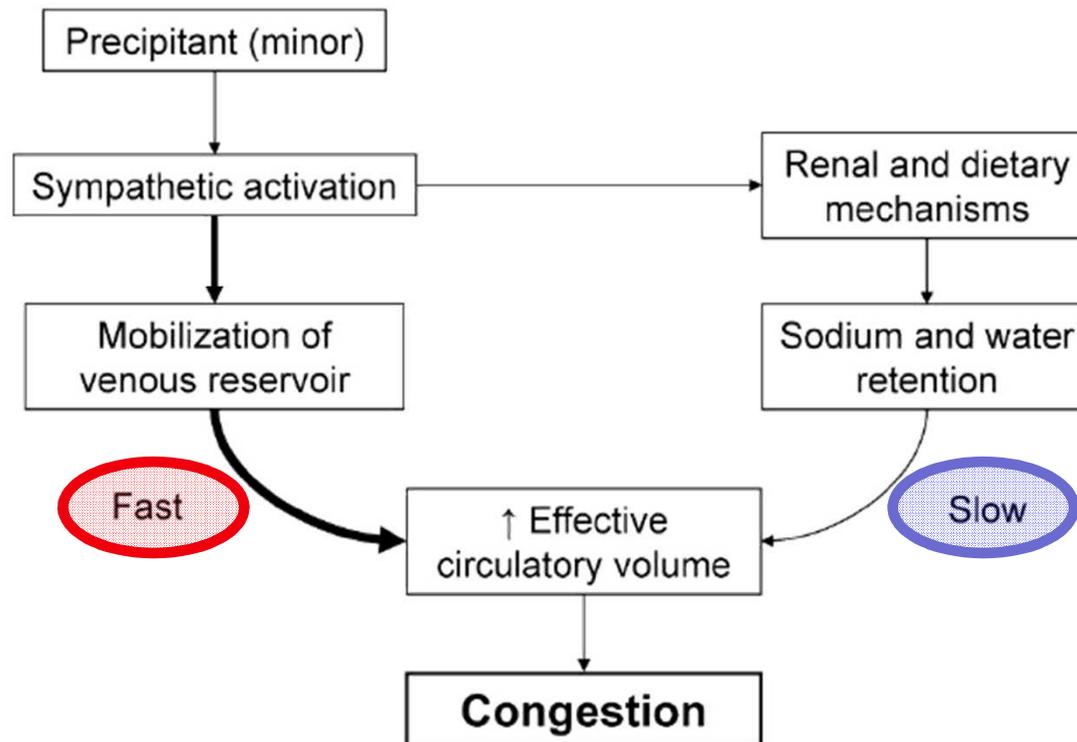


VASOCONSTRICCIÓN



CONGESTIÓN

Fast and slow mechanisms of circulatory congestion



CONGESTIÓN

145 pacientes ICA FE $20 \pm 8\%$

Tx médico guiado por cateterismo dcho.

*Deterioro función renal *:*

Si: 40%

No: 60%

Poco frecuente si PVC < 8 mm Hg

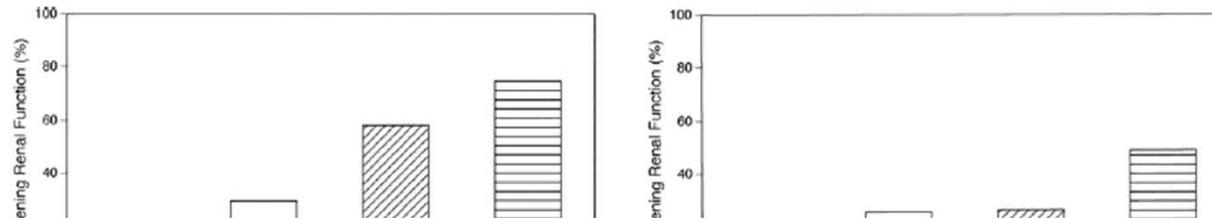
	Basal mm Hg	Post Tx mm Hg
SI	18 ± 7	11 ± 8
NO	12 ± 6	8 ± 75



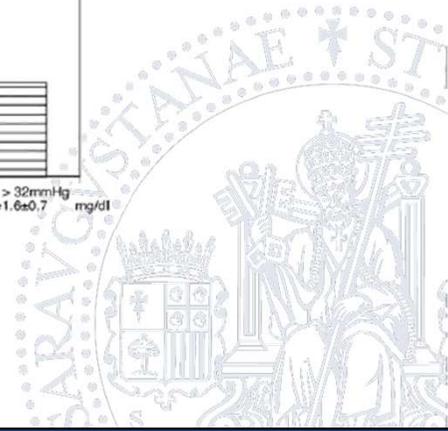
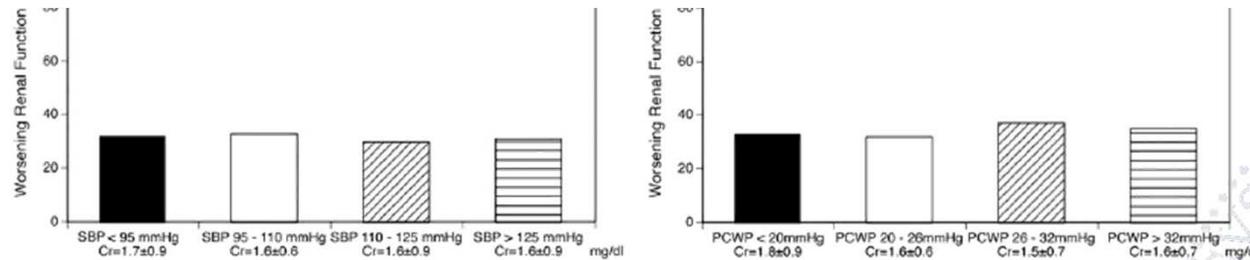
* DFR: Δ creatinina $\geq 0,3$ mg/dl



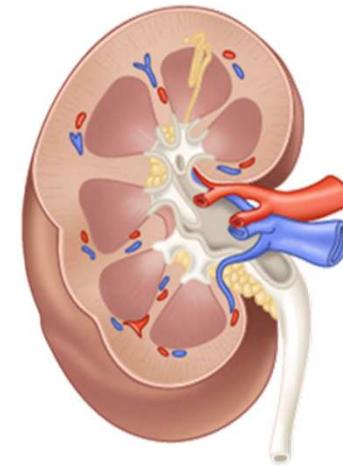
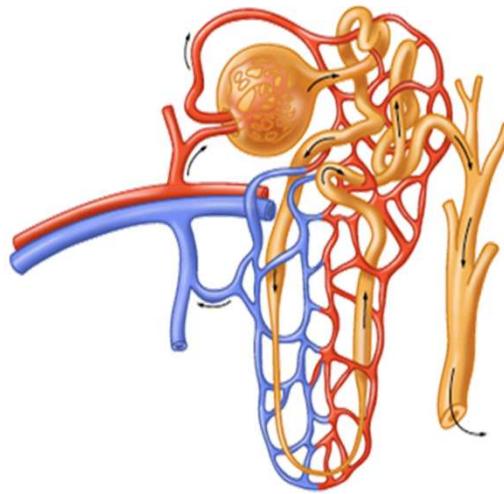
CONGESTIÓN



FALLO RENAL CONGESTIVO



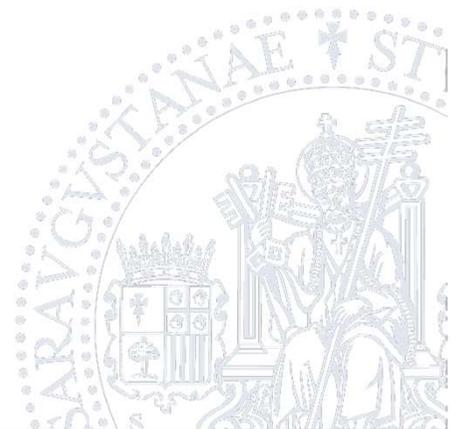
CONGESTIÓN



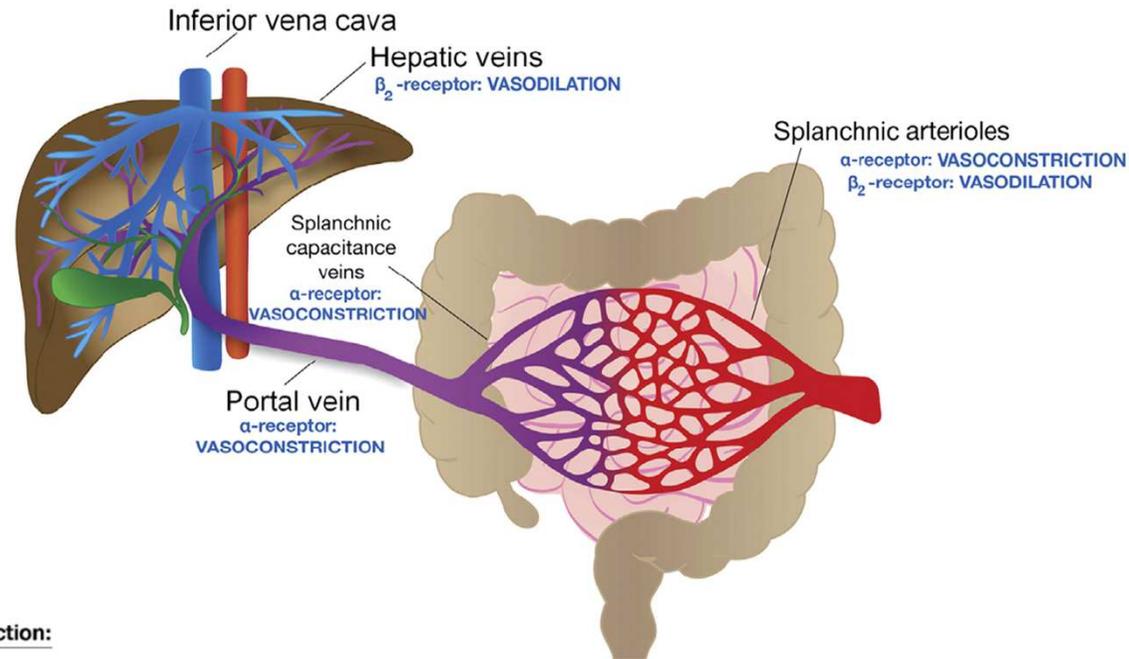
Circulación esplácnica

Presión intraabdominal

“Membrana” Intersticial



CIRCULACIÓN ESPLÁCNICA



Regulation of capacitance function:

1. PASSIVE:

Arteriolar perfusion $\downarrow \rightarrow P_{\text{capacitance veins}} \downarrow \leftrightarrow$ elastic recoil

2. ACTIVE:

Sympathetic stimulation

Capacitance veins vasoconstriction

Capacitance volume \downarrow

Hepatic veins vasodilation

Venous impedance \downarrow

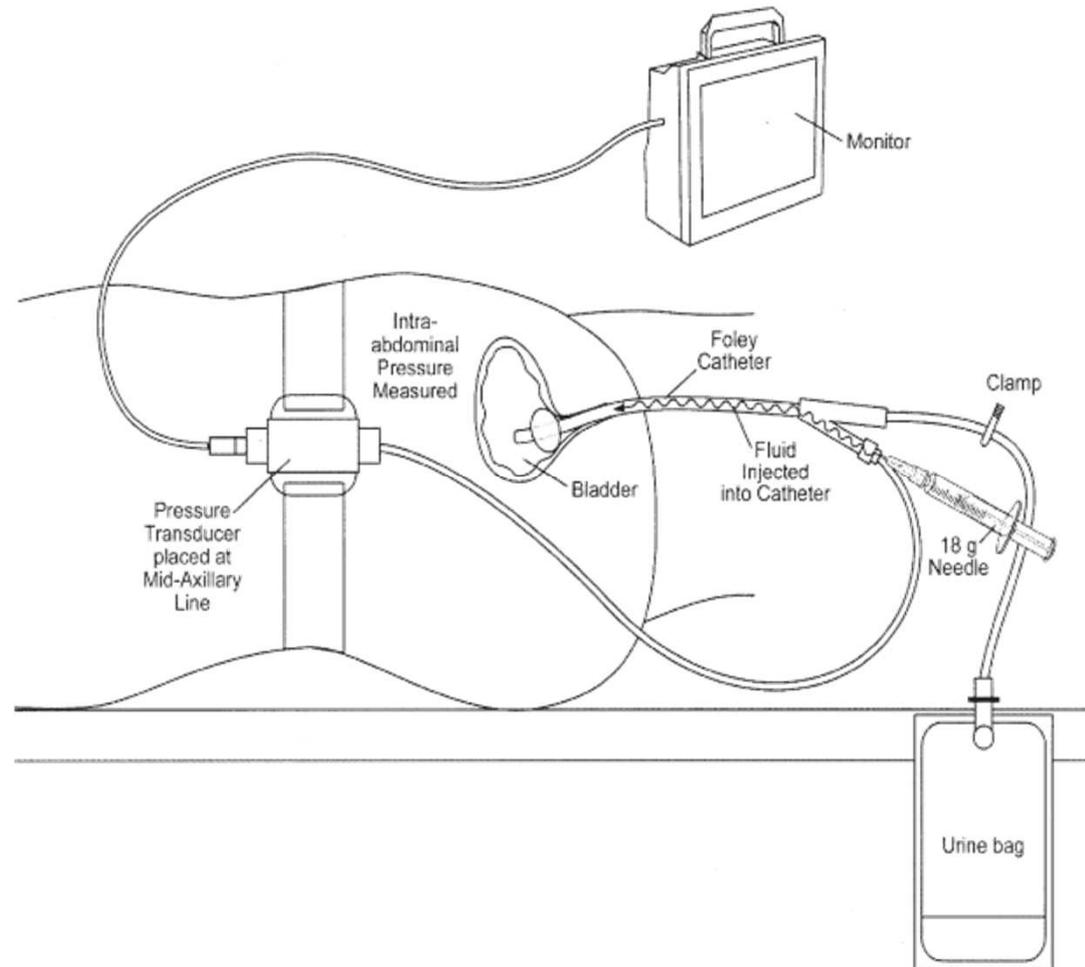
Result:
Effective Circulatory Volume \uparrow



CIRCULACIÓN ESPLÁCNICA



PRESIÓN INTRAABDOMINAL

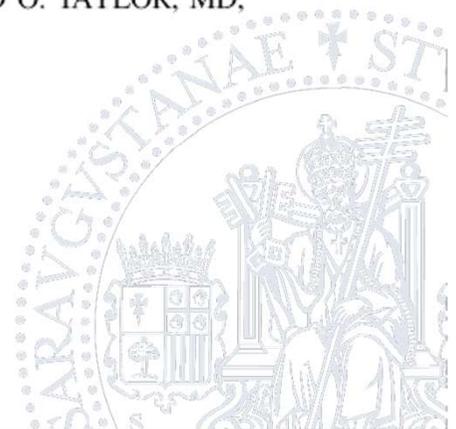


PRESIÓN INTRAABDOMINAL

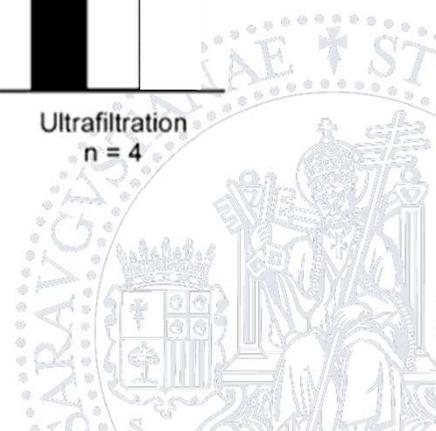
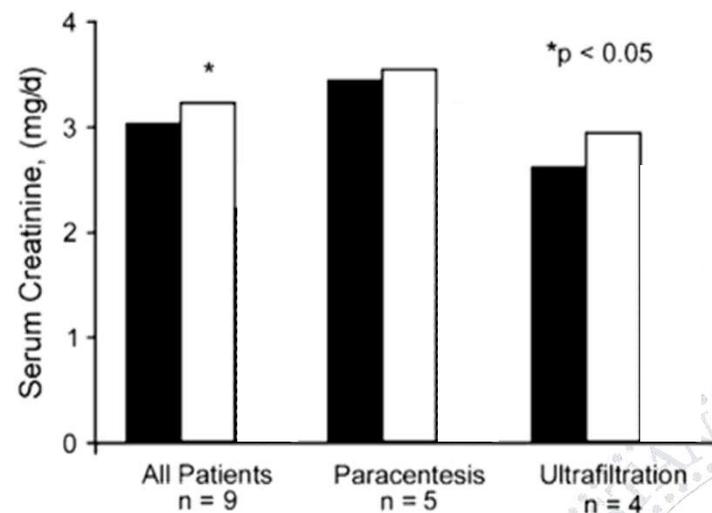
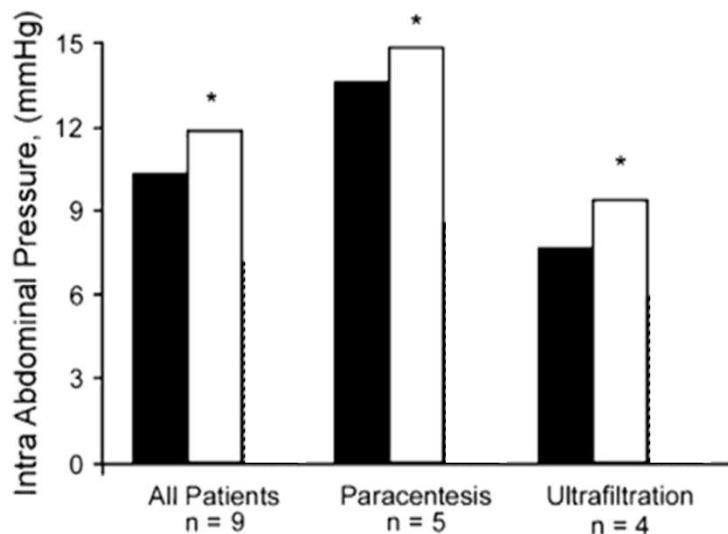
Prompt Reduction in Intra-Abdominal Pressure Following Large-Volume Mechanical Fluid Removal Improves Renal Insufficiency in Refractory Decompensated Heart Failure

WILFRIED MULLENS, MD, ZUHEIR ABRAHAMS, MD, PhD, GARY S. FRANCIS, MD, DAVID O. TAYLOR, MD,
RANDALL C. STARLING, MD, MPH, AND W.H. WILSON TANG, MD

Cleveland, Ohio



PRESIÓN INTRAABDOMINAL



INTERSTICIO

475 pacientes ICA FE $28 \pm 14\%$

Cohorte del estudio VMAC

238 pacientes con cateterismo dcho.

Presión AD basal y tras 24 h.

Pérdida neta de líquidos en 24 h.

DFR: Δ creatinina $\geq 0,3$ mg/dl

35,3%

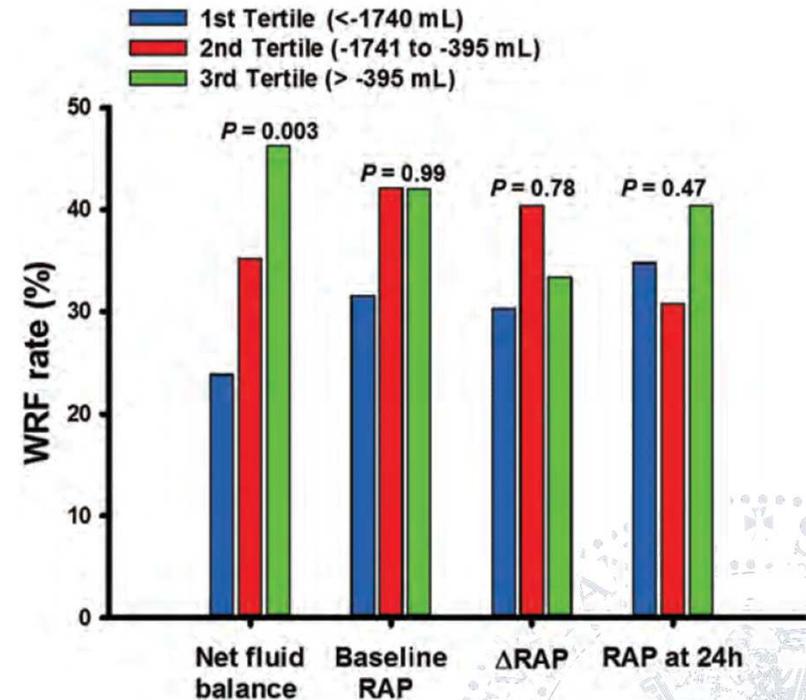
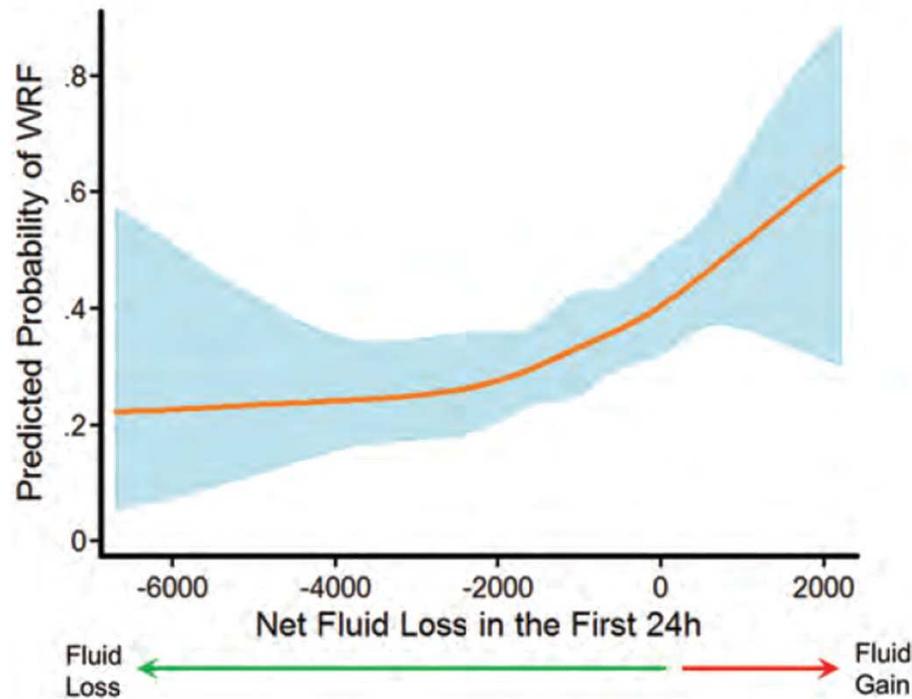
*incidencia
disfunción renal*



RAMBAM
MEDICAL CENTER



INTERSTICIO





INFLAMACIÓN ¿EL NEXO?



European Heart Journal (2014) 35, 448–454
doi:10.1093/eurheartj/eh456

CLINICAL RESEARCH

Heart failure/cardiomyopathy

Peripheral venous congestion causes inflammation, neurohormonal, and endothelial cell activation

Paolo C. Colombo^{1*}, Duygu Onat¹, Ante Harxhi¹, Ryan T. Demmer², Yacki Hayashi¹, Sanja Jelic³, Thierry H. Lejemtel⁴, Loredana Bucciarelli⁵, Moritz Kepschull⁶, Panos Papananou⁷, Nir Uriel¹, Ann Marie Schmidt⁸, Hani N. Sabbah⁹, and Ulrich P. Jorde¹

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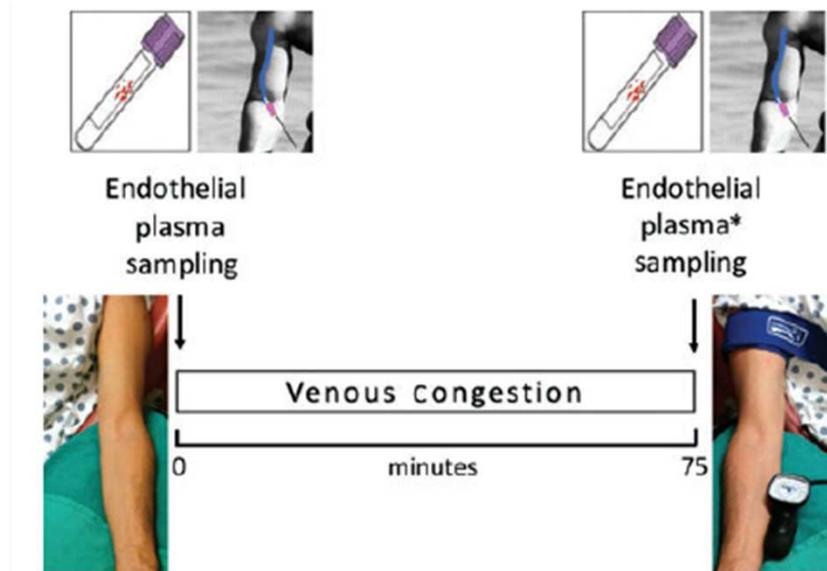


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INFLAMACIÓN ¿EL NEXO?



“Test de stress venoso”

24 sujetos sanos

Extracción sanguínea y de células endoteliales

Brazo congestivo y normal (control)

Antes y 75 minutos después de inflado

IL-6, ET-1, AT II, VCAM-1, Chemokine ligand 2

mRNA



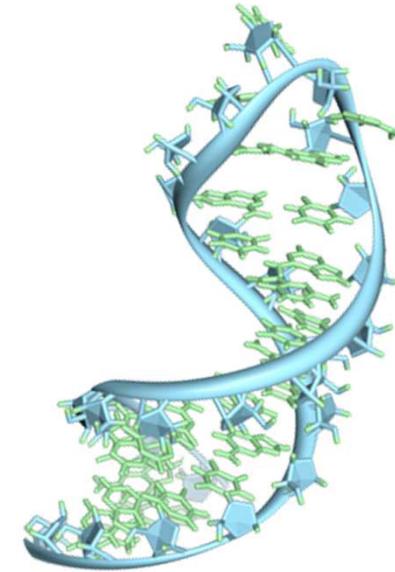
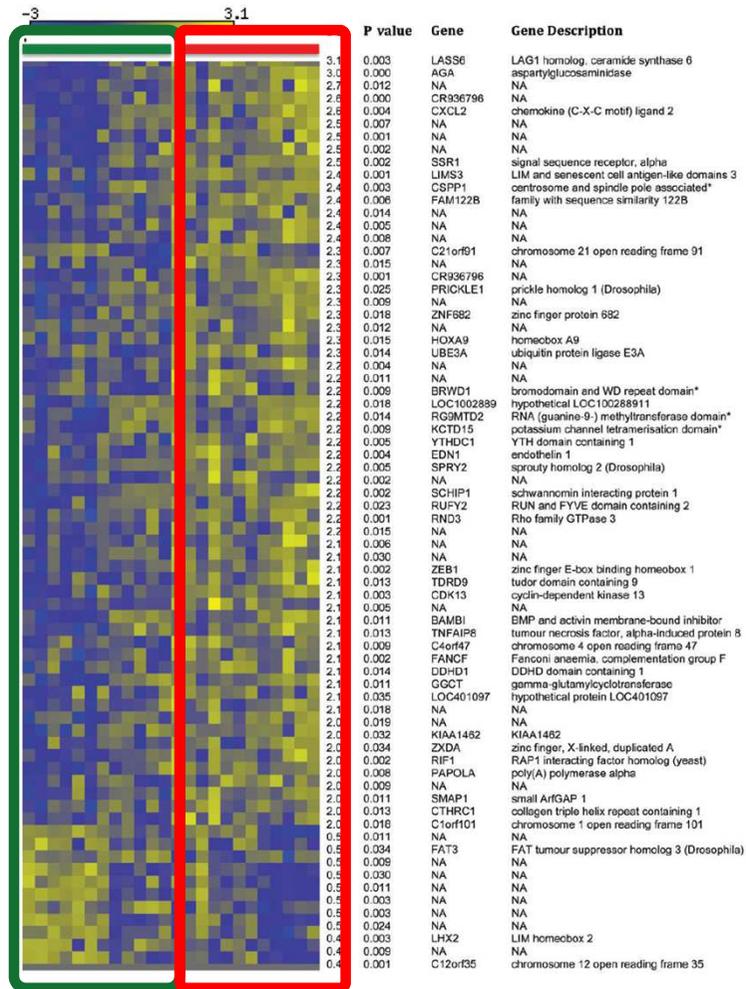
INFLAMACIÓN ¿EL NEXO?

Table 2 Plasma measurements (means \pm SEM) before and after 75 min of venous congestion

Variable	Baseline (B) (0 min)	Control arm (C) (75 min)	Test arm (T) (75 min)	P-value (T vs. B)	P-value (T vs. C)
IL-6 (pg/mL)	1.37 \pm 0.44	1.79 \pm 0.53	2.26 \pm 0.58	<0.01	<0.01
TNF- α (pg/mL)	1.35 \pm 0.08	1.27 \pm 0.08	1.35 \pm 0.11	0.75	0.22
ET-1 (pg/mL)	1.46 \pm 0.19	1.26 \pm 0.13	2.43 \pm 0.27	<0.0001	<0.0001
All (pg/mL)	27 \pm 3	25 \pm 3	32 \pm 4	0.01	<0.01
VCAM-1 (ng/mL)	557 \pm 26	544 \pm 24	589 \pm 25	<0.01	<0.01
ICAM-1 (ng/mL)	158 \pm 9	158 \pm 7	167 \pm 9	0.09	0.06
vWF:Ag (%)	105 \pm 9	100 \pm 6	113 \pm 9	0.37	0.15

IL-6, interleukin-6; TNF- α , tumor necrosis factor- α ; ET-1, endothelin-1; All, Angiotensin II; VCAM-1, vascular adhesion molecule-1; ICAM-1, intercellular adhesion molecule-1; vWF:Ag, vonWillebrand factor antigen.

INFLAMACIÓN ¿EL NEXO?



¿Y EN EL DÍA A DÍA QUÉ?

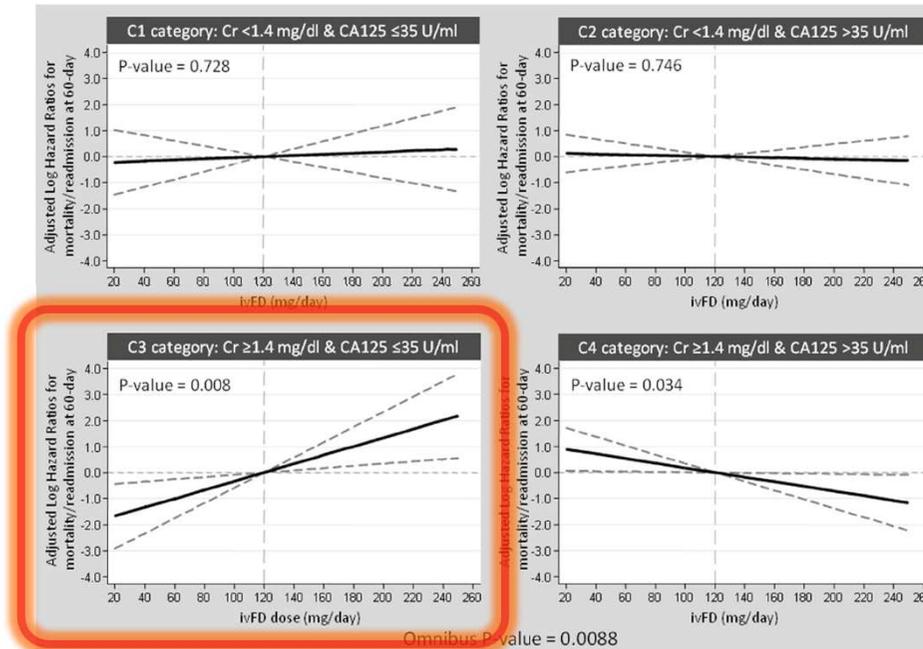


526 pacientes con ICA

Administración de Furosemida i.v. durante las primeras 48 h.

Objetivo: cambio en concentración de creatinina según grupos (grado de congestión y de función renal)

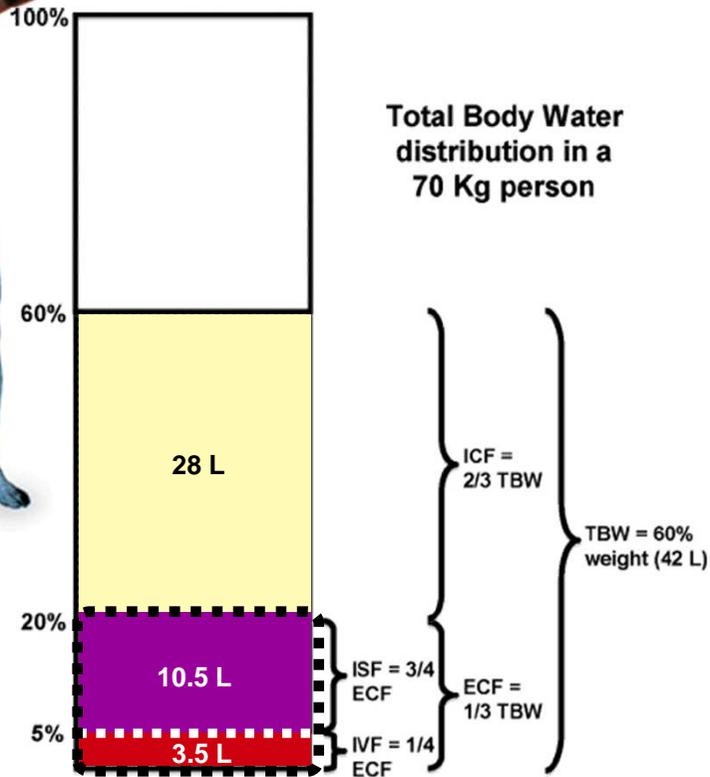
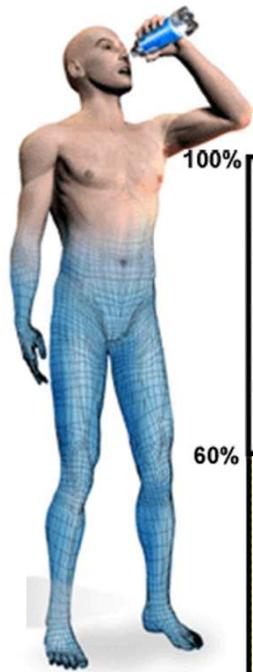
Función renal preservada



Congestión



RESUMEN Y CONCLUSIONES

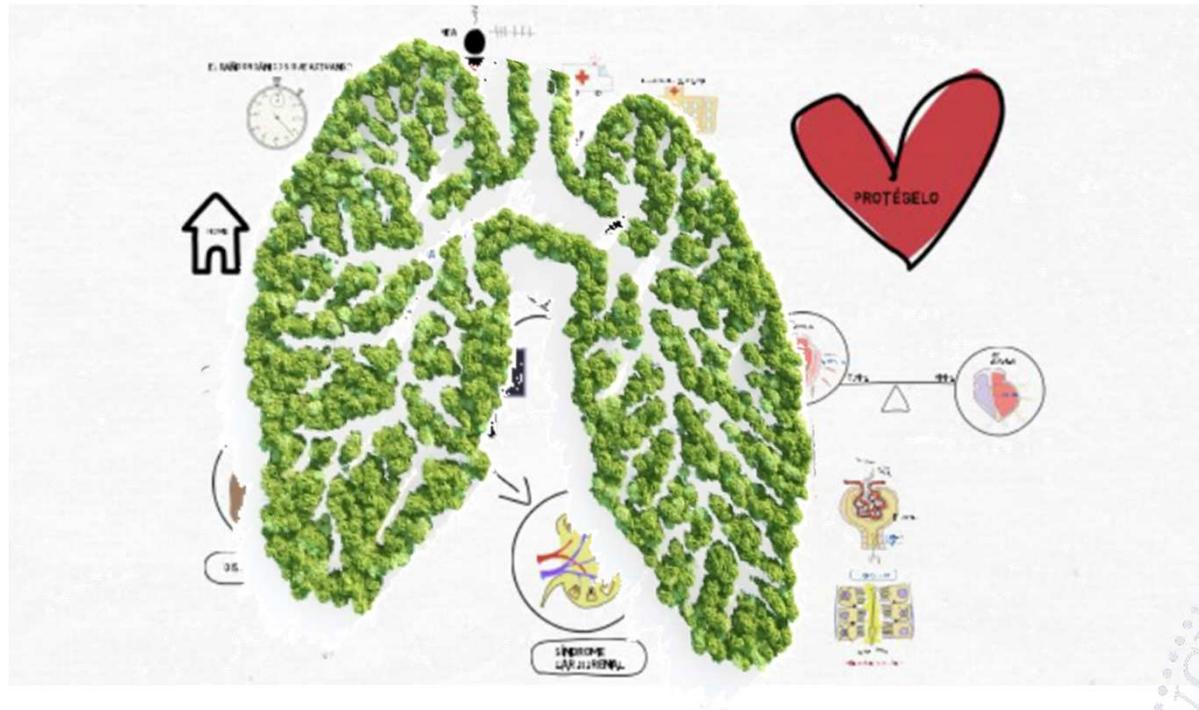


Congestión sistémica e ICA

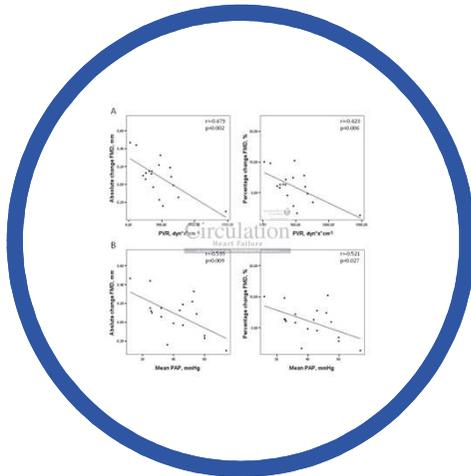
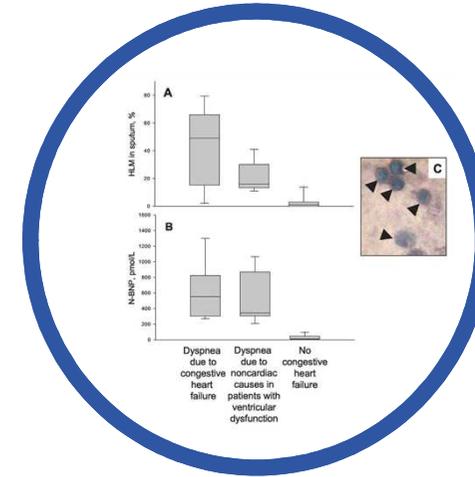
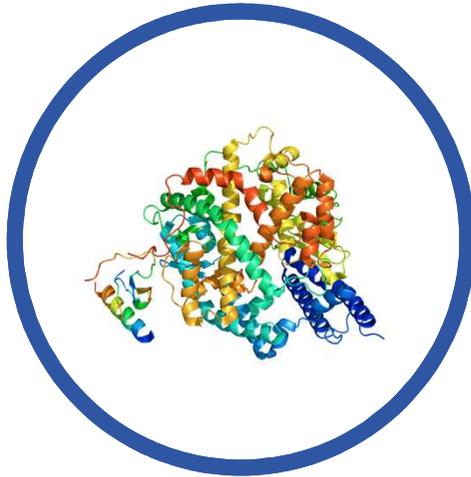
- 1. Circulación esplácnica*
- 2. Presión intraabdominal*
- 3. Tasa de relleno vascular*



VISIÓN INTEGRAL



VISIÓN INTEGRAL



Simplicity does not precede complexity, but follows it.

(Alan Perlis)



Muchas gracias por vuestra atención

