Dysfonction diastolique VG: la REVOLUTION !

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Disclosures

Honoraria for lectures:

Roche diagnostics, Bayer, MSD

Consultancy:

4TEEN4 Parmaceuticals, Adrenomed, S-Form Pharma, Sphingotec, Corteria Parmaceuticals, Implicity, Fire-1 foundry

Grants:

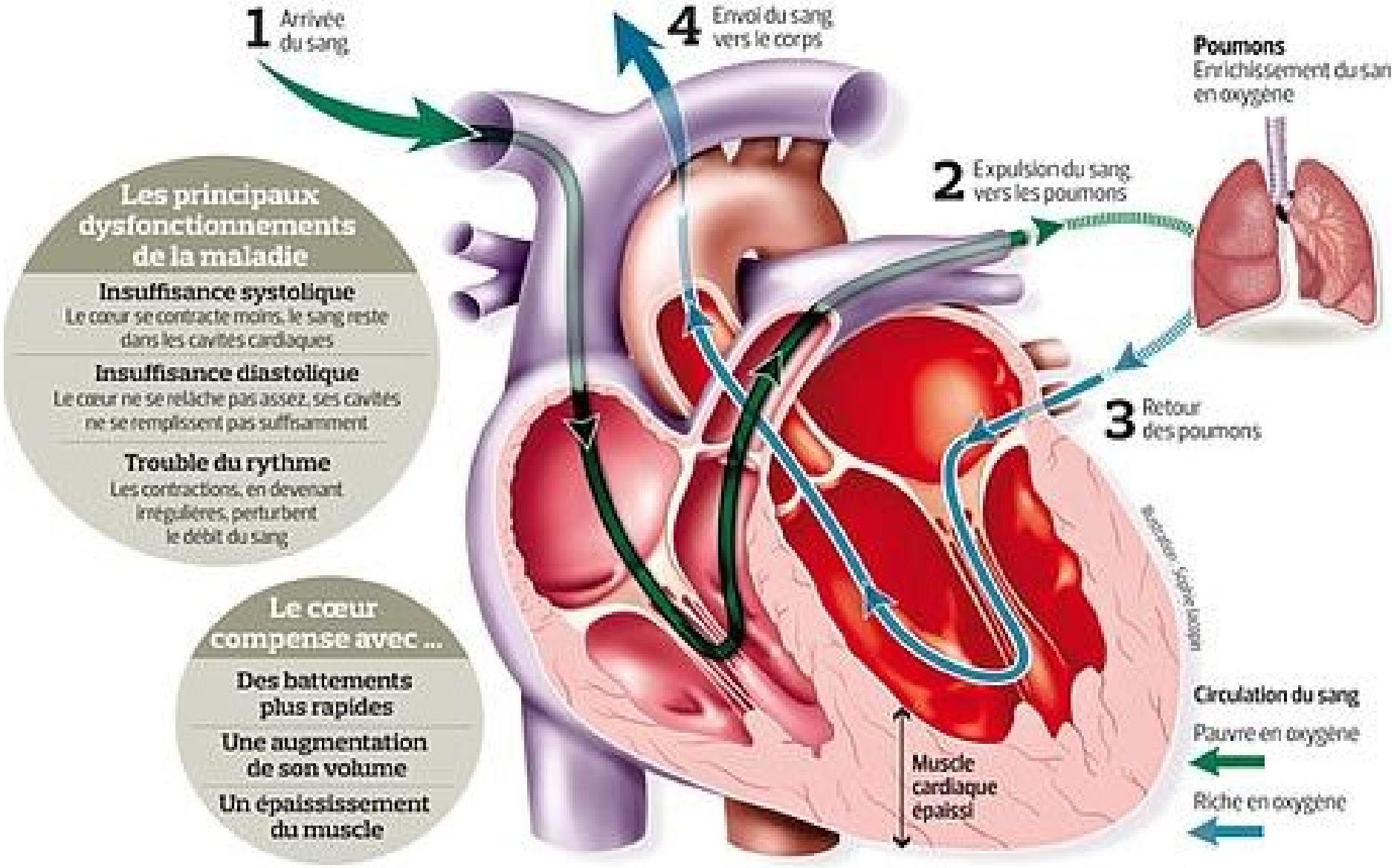
Windtree therapeutics, Roche diagnostics, Abbott Diagnostics, 4TEEN4 Pharmaceuticals

Patents:

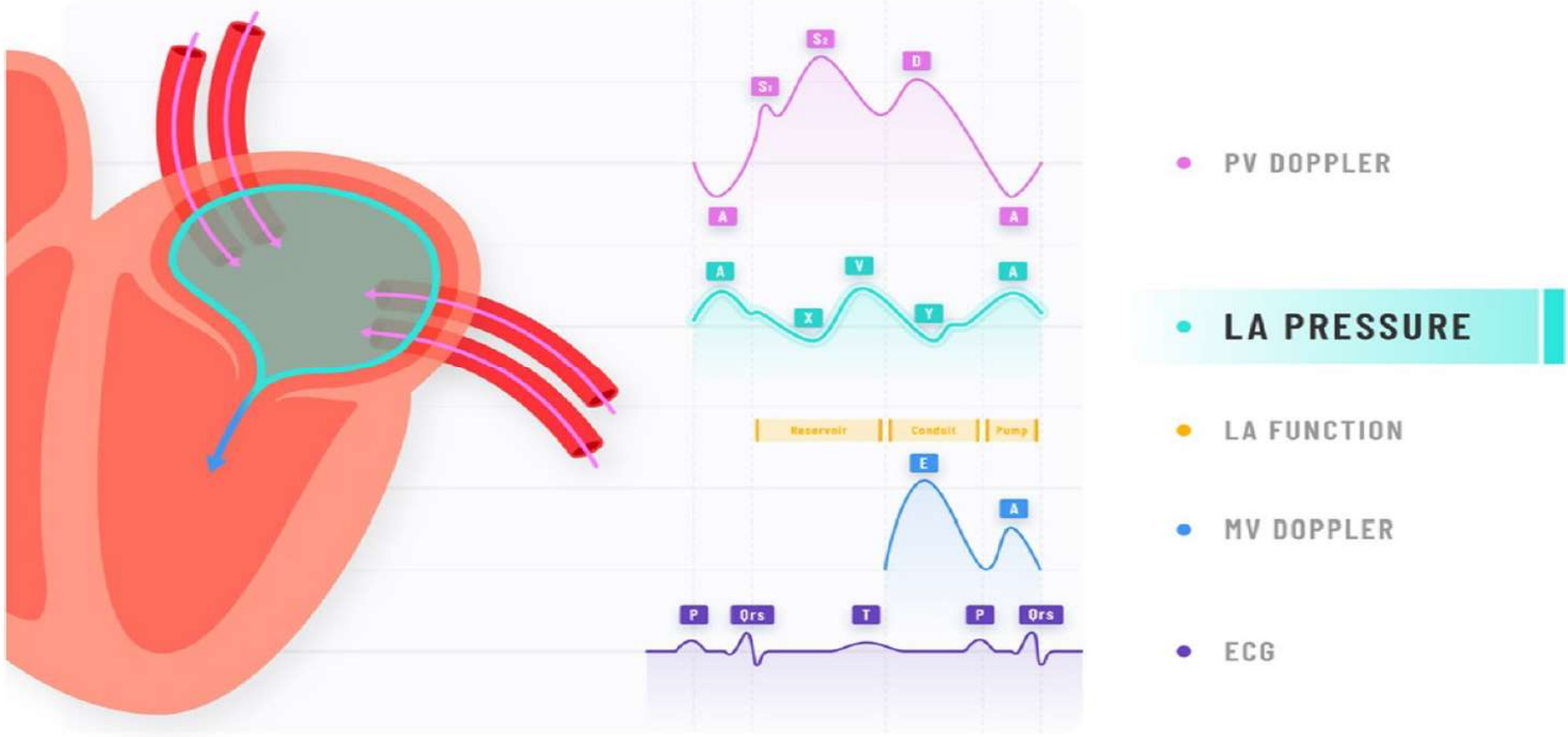
coinventor of a patent on combination therapy for patients having acute or persistent dyspnoea and another on rapid optimization of oral heart failure therapies helped by NT-proBNP testing

Principaux messages sur la fonction diastolique VG

- Elle protège le poumon et le VD
- Extrêmement fréquente : âge, obésité
- Toujours associée à une congestion
- Chronique ? : OG élargi? Insuffisance mitrale ?
- Si chronique : glifozine


1 Arrivée du sang
4 Envoi du sang vers le corps
Poumons
Enrichissement du sang en oxygène
2 Expulsion du sang vers les poumons
3 Retour des poumons
Les principaux dysfonctionnements de la maladie
Insuffisance systolique
Le cœur se contracte moins, le sang reste dans les cavités cardiaques
Insuffisance diastolique
Le cœur ne se relâche pas assez, ses cavités ne se remplissent pas suffisamment
Trouble du rythme
Les contractions, en devenant irrégulières, perturbent le débit du sang
Le cœur compense avec ...
Des battements plus rapides
Une augmentation de son volume
Un épaississement du muscle
Muscle cardiaque épaissi
Circulation du sang
Pauvre en oxygène
Riche en oxygène

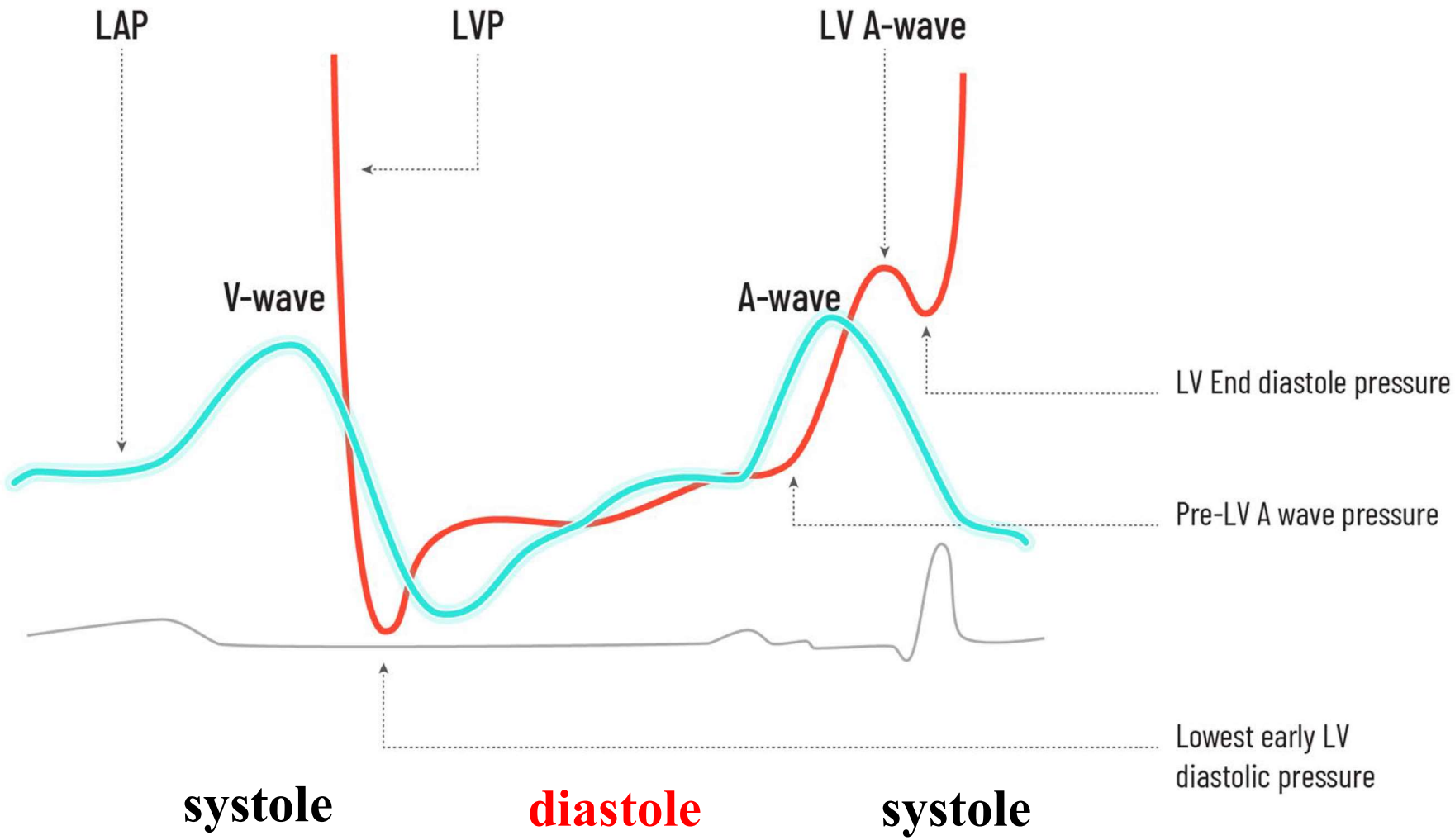
Physiology of the left atria



systole **diastole**

EM Bowcock & A Mc Lean Crit Care 2022

Physiology of the left atria

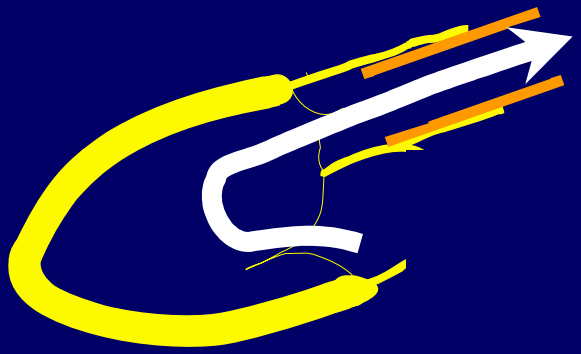


EM Bowcock & A Mc Lean Crit Care 2022

La fonction diastolique VG

- « aspire le sang » des OG et VP vers le VG
- Elle protège les poumons
- Elle consomme de l'O2
- Ischémie est associée à un OAP

LV Suction?



Rat Heart

Courtesy of:
Drs. Frits Meijler

WC Little

DETERMINANTS

LV diastolic properties
(relaxation, compliance)

LV loading conditions
(preload, afterload)

Diastolic LV filling

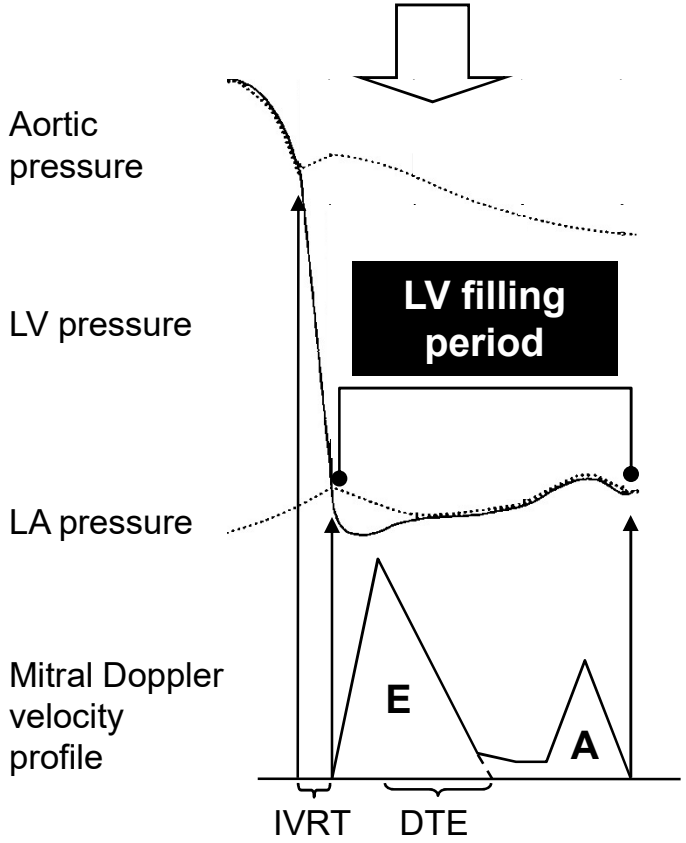
Heart rate

LV interactions
(RV, pericardium, lungs)

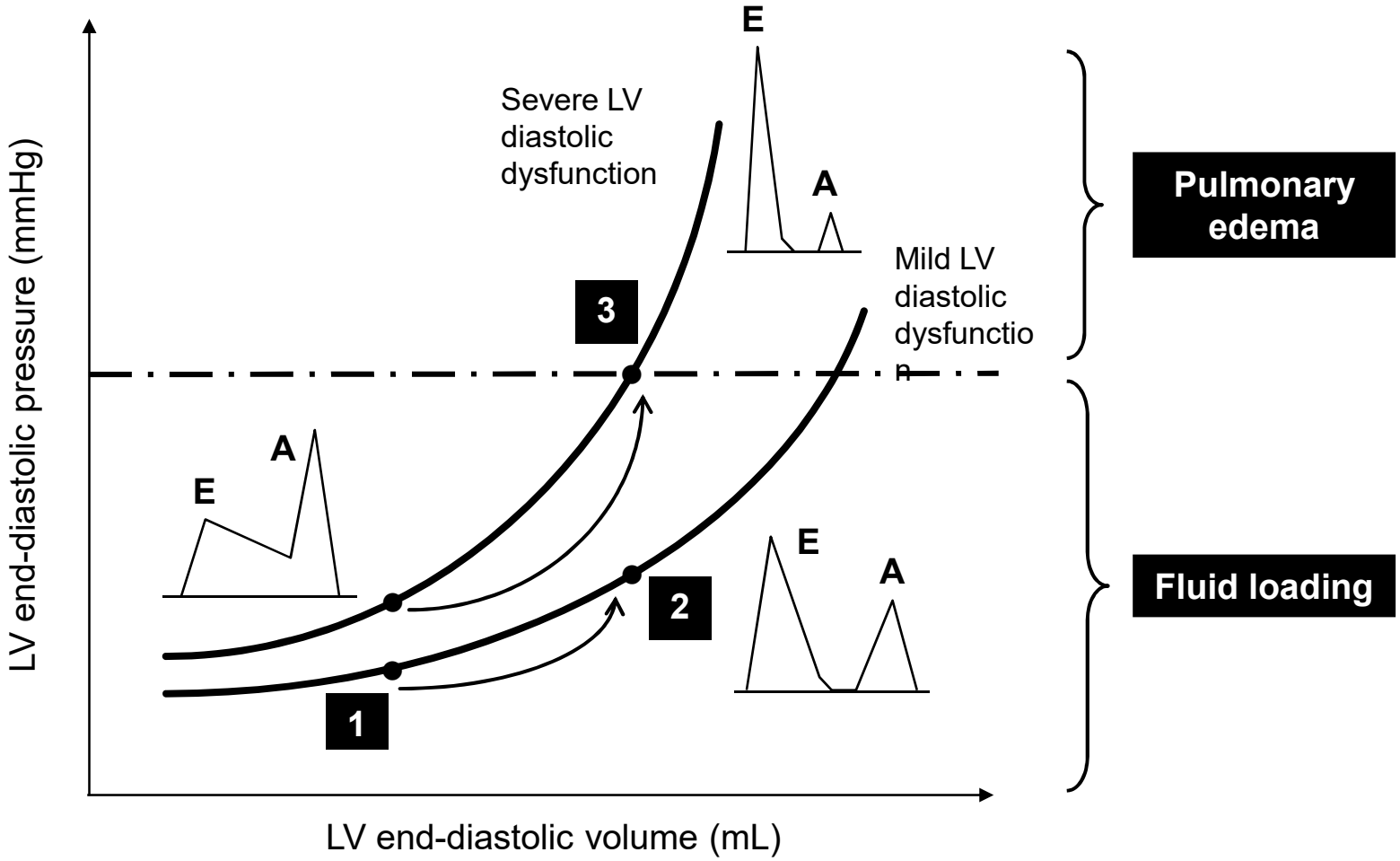
Age

LV filling pressure

CONSEQUENCES

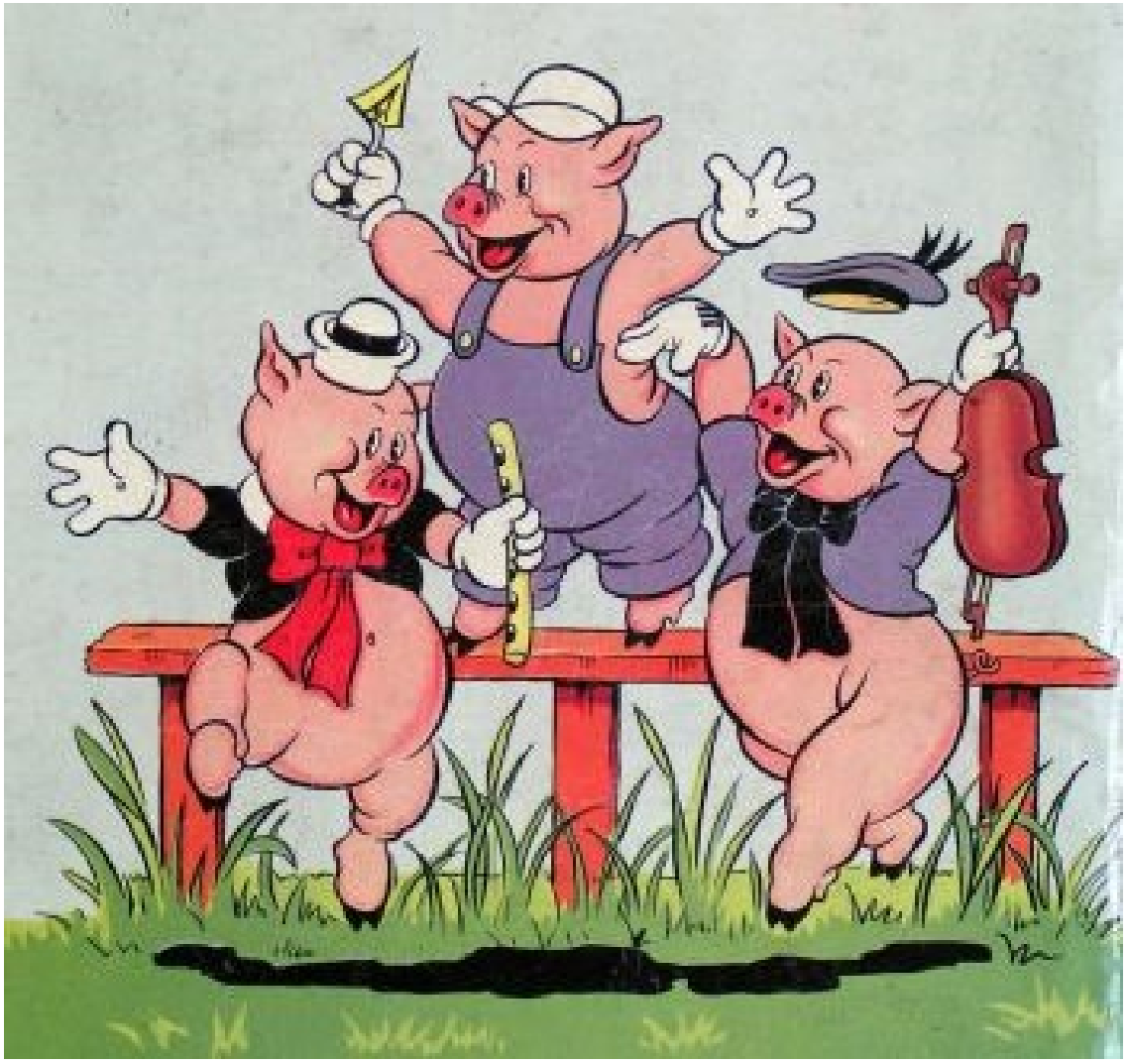


Ph Vignon, in Acute Heart Failure, Mebazaa et al, Springer.



Ph Vignon, in Acute Heart Failure, Mebazaa et al, Springer.

Les 3 scénarios cliniques de l'ICA



Les 3 scénarios cliniques de l'ICA

Clinical Scenario	Characteristics
CS1	SBP >140 mm Hg
	Symptoms develop abruptly
	Predominantly diffuse pulmonary edema
	Minimal systemic edema (patient may be euvolemic or hypovolemic)
	Acute elevation of filling pressure often with preserved LVEF
	Vascular pathophysiology
CS2	SBP 100–140 mm Hg
	Symptoms develop gradually, together with a gradual increase in body weight
	Predominantly systemic edema
	Minimal pulmonary edema
	Chronic elevation of filling pressure, including increased venous pressure and elevated pulmonary arterial pressure
	Manifestations of organ dysfunction (renal impairment, liver dysfunction, anemia, hypoalbuminemia)
CS3	SBP <100 mm Hg
	Rapid or gradual onset of symptoms
	Predominantly signs of hypoperfusion
	Minimal systemic and pulmonary edema
	Elevation of filling pressure

Mebazaa et al, Crit Care Medicine 2008

CS1: ED: Dyspnea and/or Other Signs of Congestion + Elevated SBP (> 150 mmHg)

Acute pulmonary edema

+

- Dyspnea develops abruptly
- Diffuse pulmonary edema
- Minimal systemic edema

It is a vascular illness

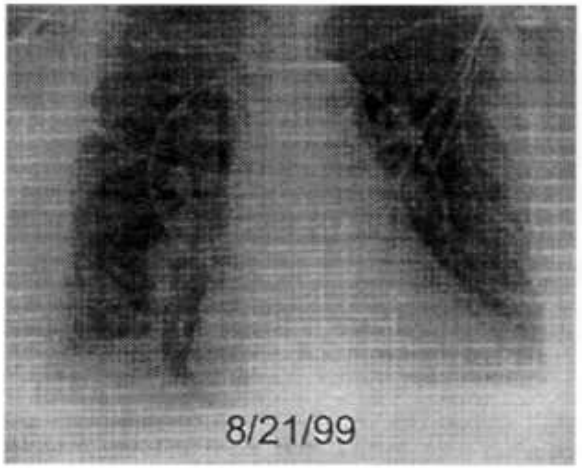
always

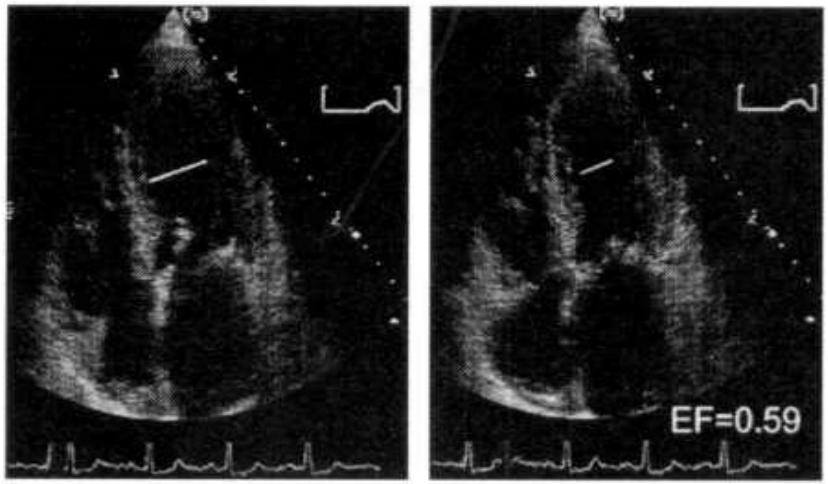
During Acute Pulmonary Edema

Blood pressure, 240/144 mm Hg

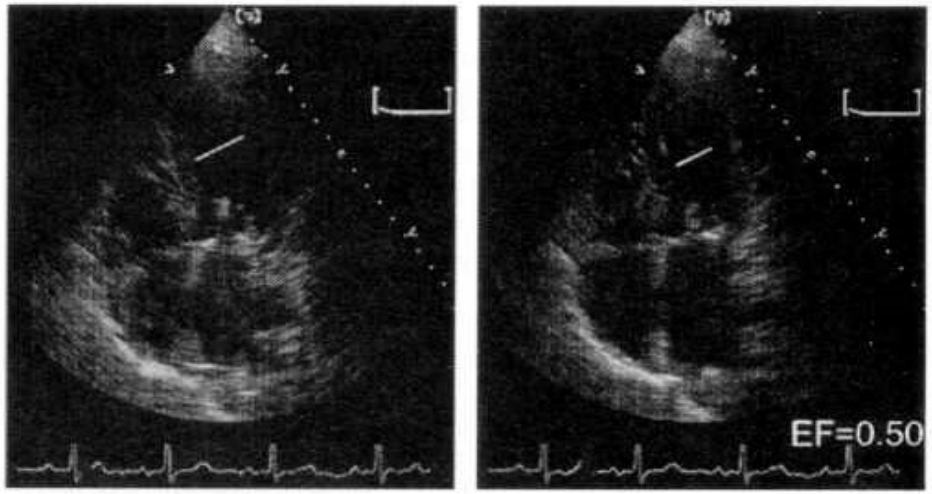
After Treatment

Blood pressure, 149/75 mm Hg





End Diastole End Systole



End Diastole End Systole

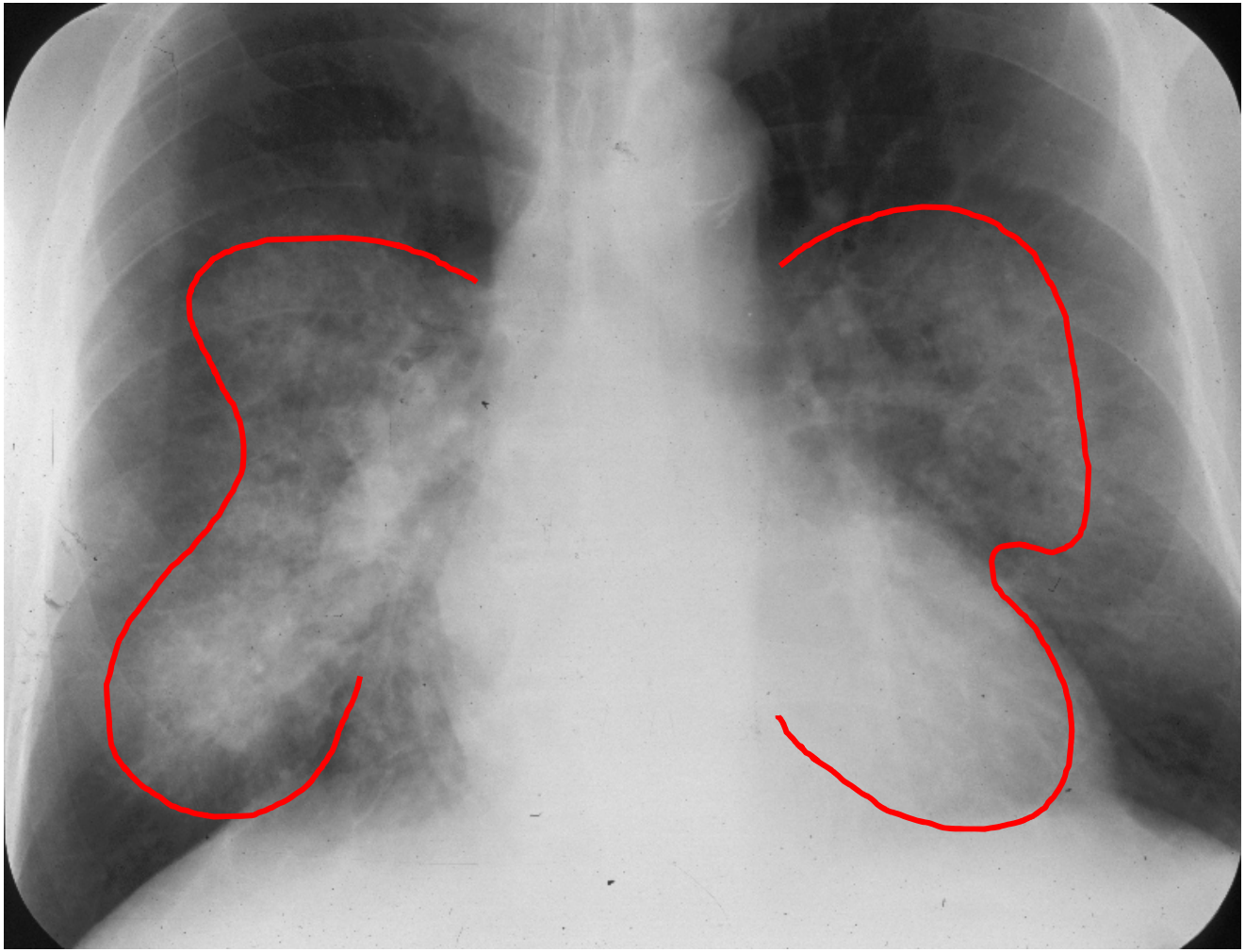
SK Ghandi, NEJM 2001, 344: 17-22

Variable	During Acute Pulmonary Edema	After Treatment
	mean ±SD	
Blood pressure (mm Hg)		
Systolic	200±26	139±17*
Diastolic	100±25	64±15*
Heart rate (beats/min)	83±14	72±12*
Mitral flow velocity (cm/sec)		
E wave	98±33	98±28
A wave	88±33	78±26*
E wave:A wave	1.31±0.80	1.51±0.97*
E-wave deceleration time (msec)	174±62	194±62*
Isovolumic relaxation time (msec)	78±19	75±25
Left ventricular volume (ml)		
End diastolic	109±43	117±50
End systolic	58±32	61±37
Left ventricular ejection fraction	0.50±0.15	0.50±0.13
Left ventricular wall thickness (mm)		
Posterior	12.8±2.9	12.8±3.1
Septal	12.5±3.7	12.9±3.6
Left ventricular dimension (mm)		
End diastolic	49.7±9.5	49.4±9.8
End systolic	38.3±10.1	38.3±10.7

*P<0.05 for the comparison with the value during the acute episode.

SK Ghandi, NEJM 2001, 344: 17-22

“Butterfly” pattern on chest X-ray



Monnier-Cholley L *Chest X-ray in Acute Heart Failure* in Mebazaa A et al (Eds) *Acute Heart Failure* (2009) Springer Science & Business Media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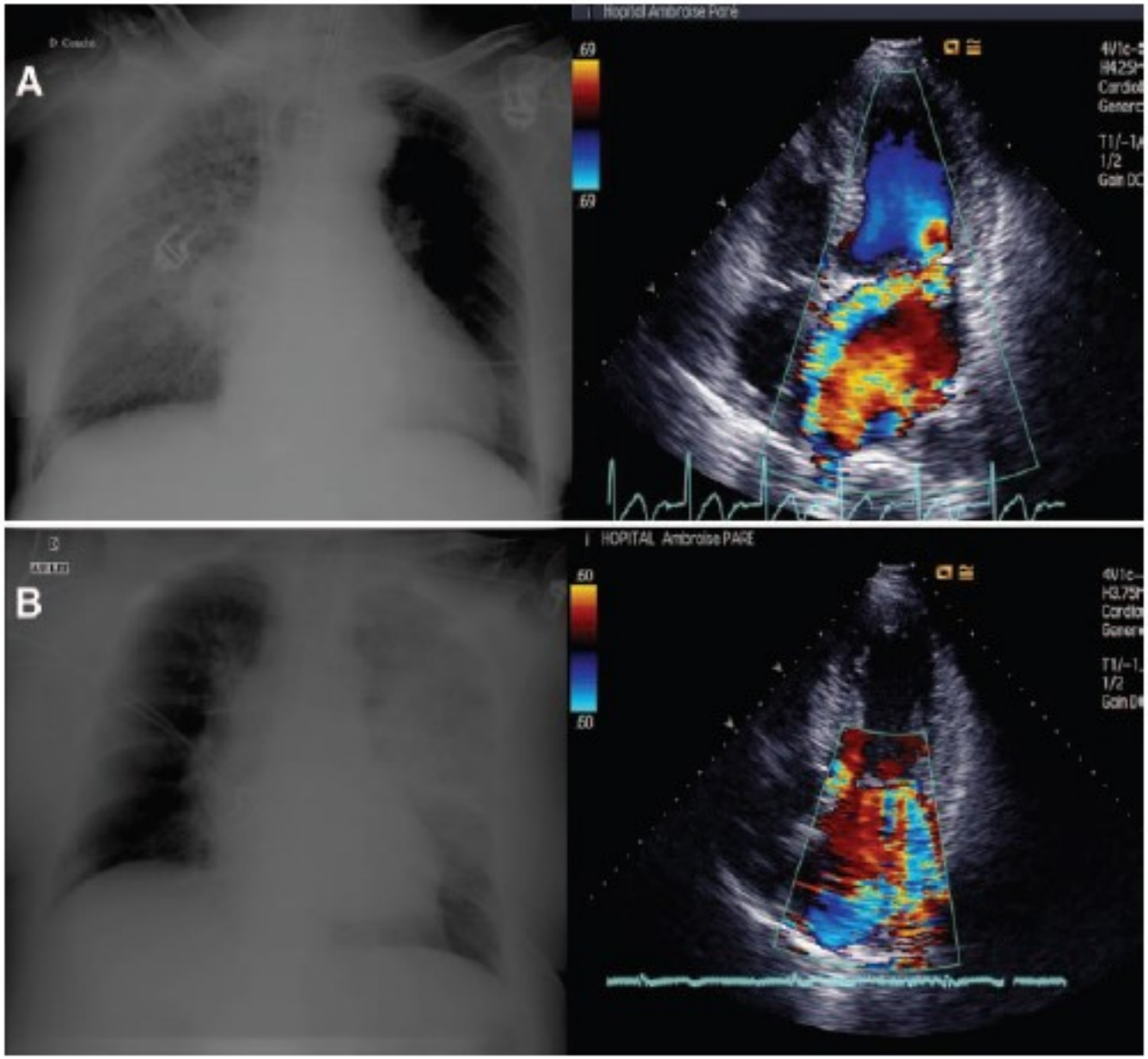


Right-sided pulmonary oedema

Left-sided pulmonary oedema

Attias D et al. *Circulation* 2010;122:1109-1115

浅草寺

CS1: ED: Dyspnea and/or Other Signs of Congestion + Elevated SBP (> 150 mmHg)

Acute pulmonary edema

+

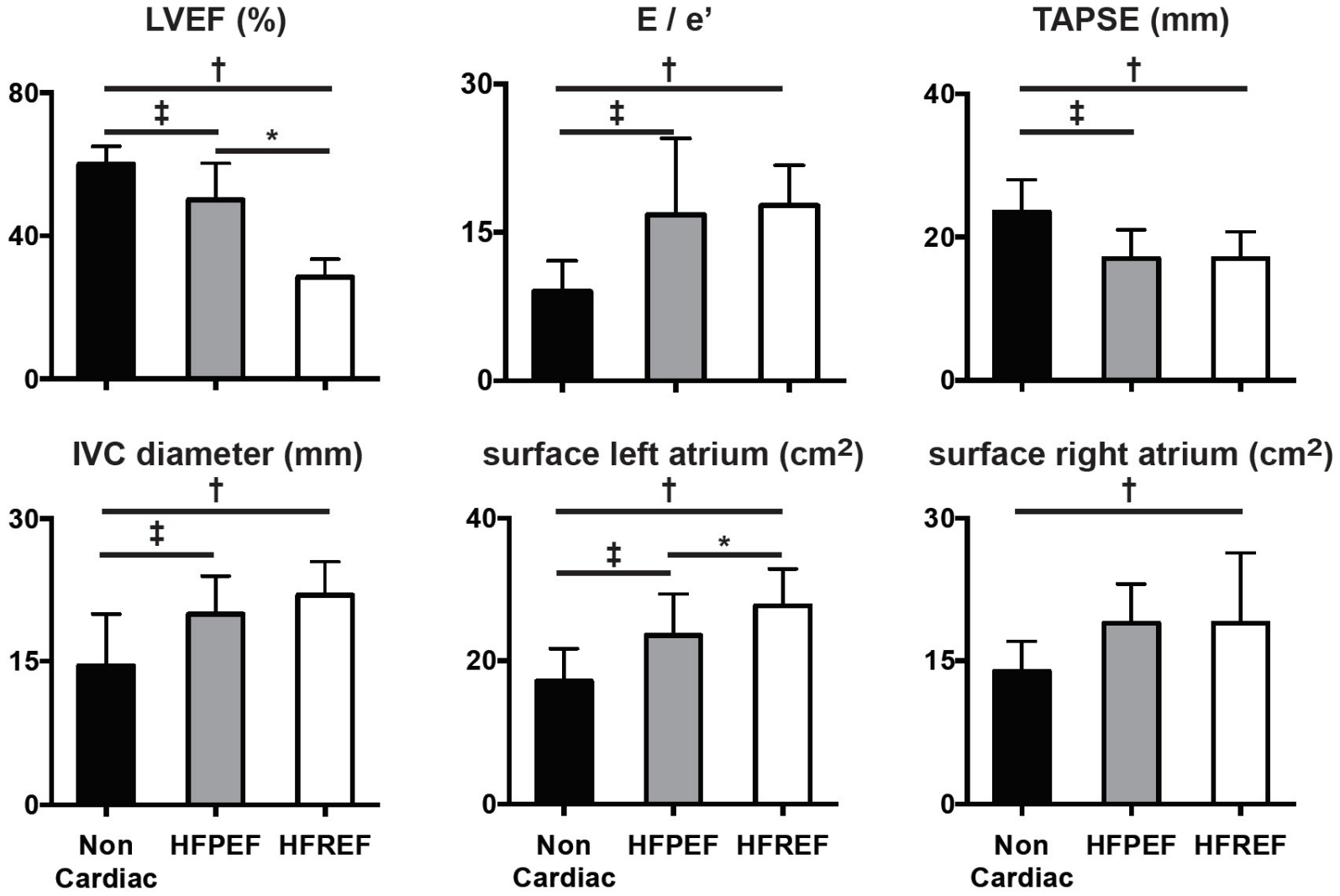
- Dyspnea develops abruptly
- Diffuse pulmonary edema
- Minimal systemic edema

It is a vascular illness

always

QUID du VD?

En urgence, la congestion VD et VG sont similaires entre HFrEF et HFpEF



L Van Aelst et al, EJHF 2018

Beijing …….

Salem *et al. Critical Care* 2014, **18**:558
http://ccforum.com/content/18/6/558

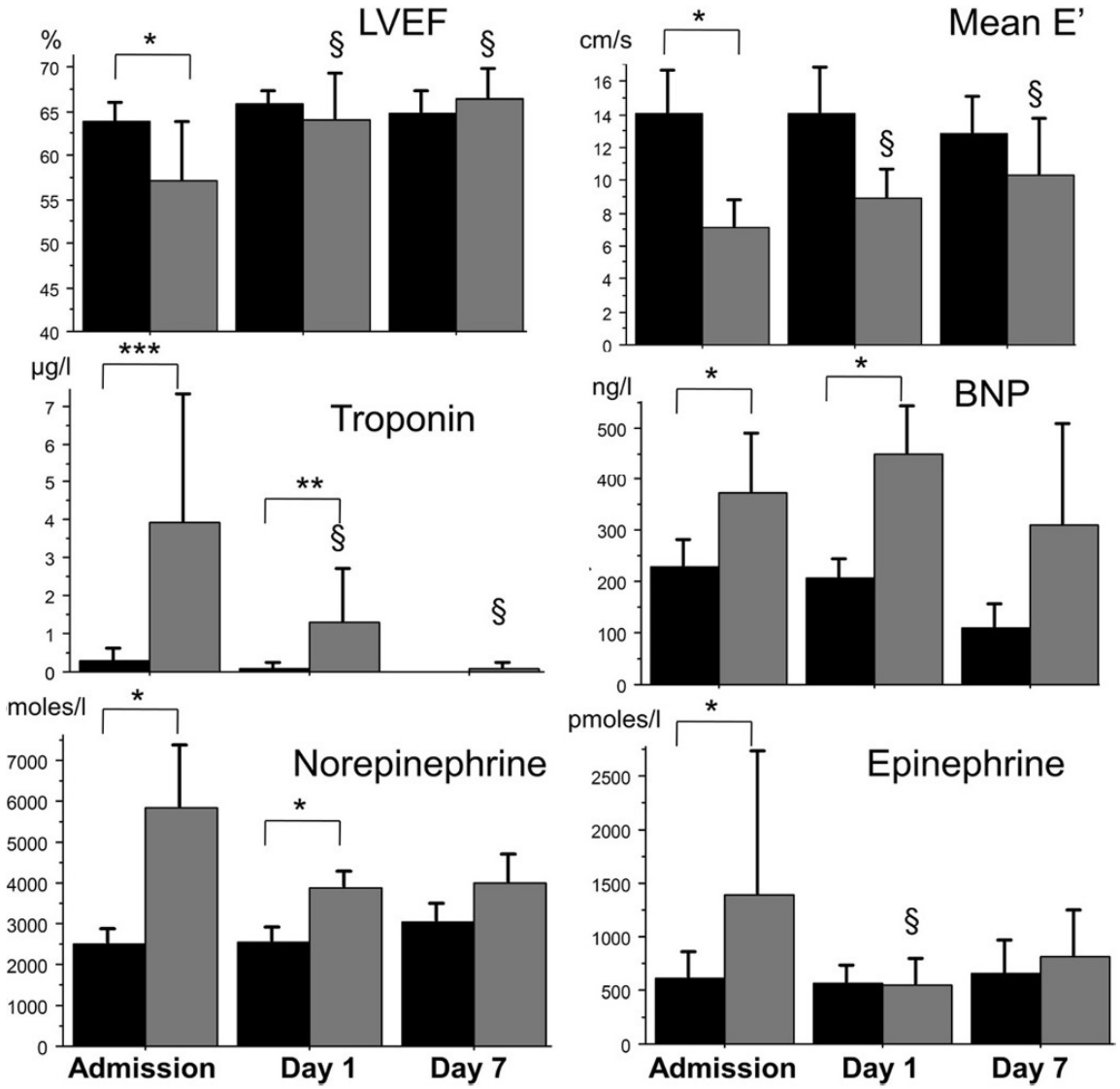
CRITICAL CARE

RESEARCH Open Access

Subarachnoid hemorrhage induces an early and reversible cardiac injury associated with catecholamine release: one-week follow-up study

Reda Salem[1,9†], Fabrice Vallée[1,5†], François Dépret[1,5†], Jacques Callebert[2], Jean Pierre Saint Maurice[3,5], Philippe Marty[1], Joaquim Matéo[1,5], Catherine Madadaki[1,5], Emmanuel Houdart[3,5], Damien Bresson[4,5], Sebastien Froelich[4,5], Christian Stapf[5,6], Didier Payen[1,5] and Alexandre Mebazaa[1,5,7,8*]

Time course of echo signs and biological parameters



Salem R et al Crit Care 2014

Est-ce que mon malade à une IC à FEVG préservé ?

ESC European Society of Cardiology

European Heart Journal (2018) **39**, 2780–2792
doi:10.1093/eurheartj/ehy301

CLINICAL REVIEW
Novel therapeutic concepts

Heart failure with preserved ejection fraction: from mechanisms to therapies

Carolyn S. P. Lam[1,2,3,4]*, Adriaan A. Voors[2], Rudolf A. de Boer[2], Scott D. Solomon[5,6], and Dirk J. van Veldhuisen[2]

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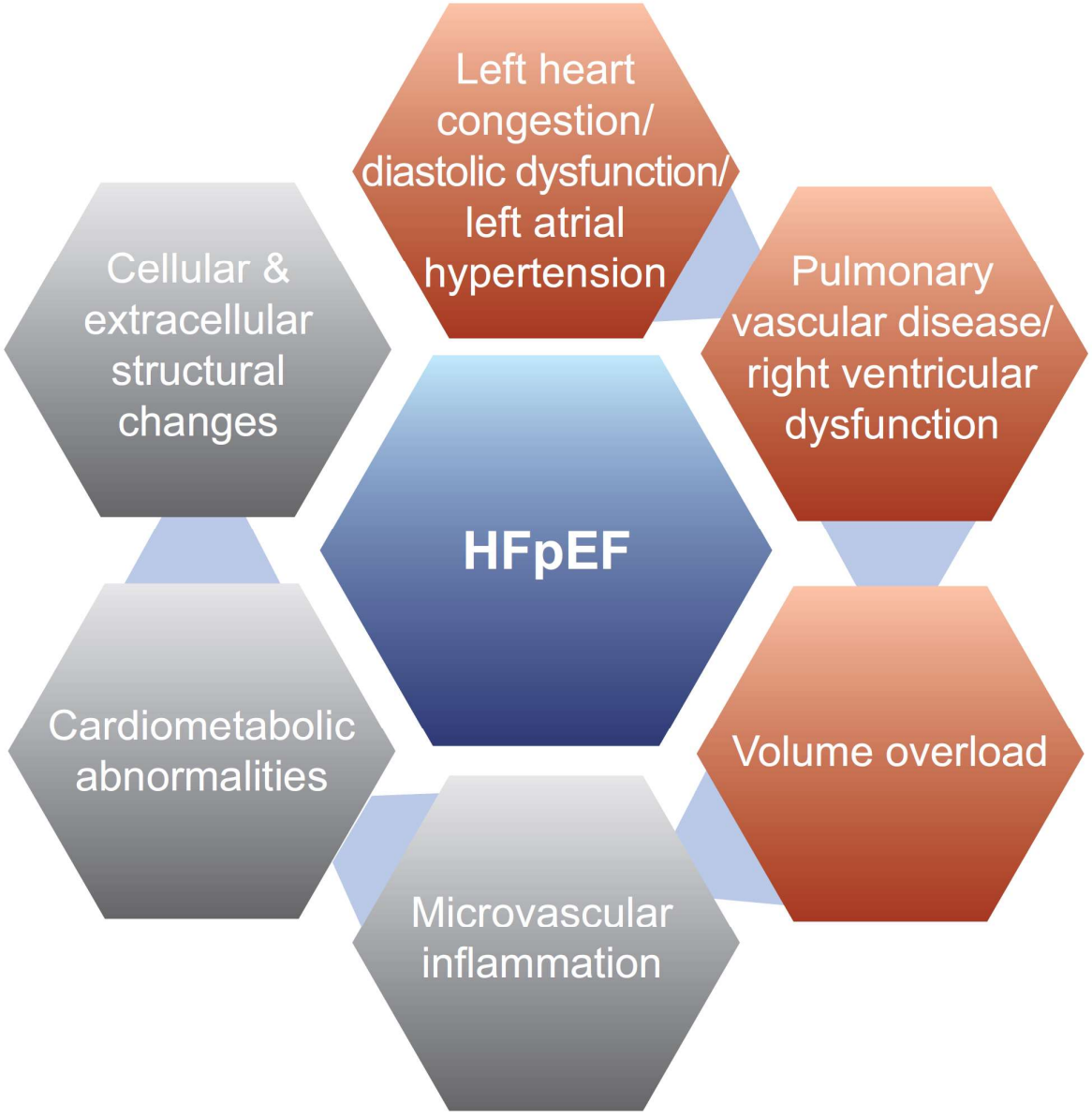
Received 18 February 2018; revised 13 April 2018; editorial decision 8 May 2018; accepted 8 May 2018; online publish-ahead-of-print 13 June 2018

Lam CSP et al, Eur Heart J, 2018

HFpEF: metabolic disease



Obesity !!!

Lam CSP et al, Eur Heart J, 2018

Ce qu'il faut savoir

- Dysfonction diastolique chronique: HFpEF (LVEF > 40%)
- L'**OG** est **élargie**
- Il a un traitement : **GLYFOZINE** ++++, spironolactone, ARNi

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