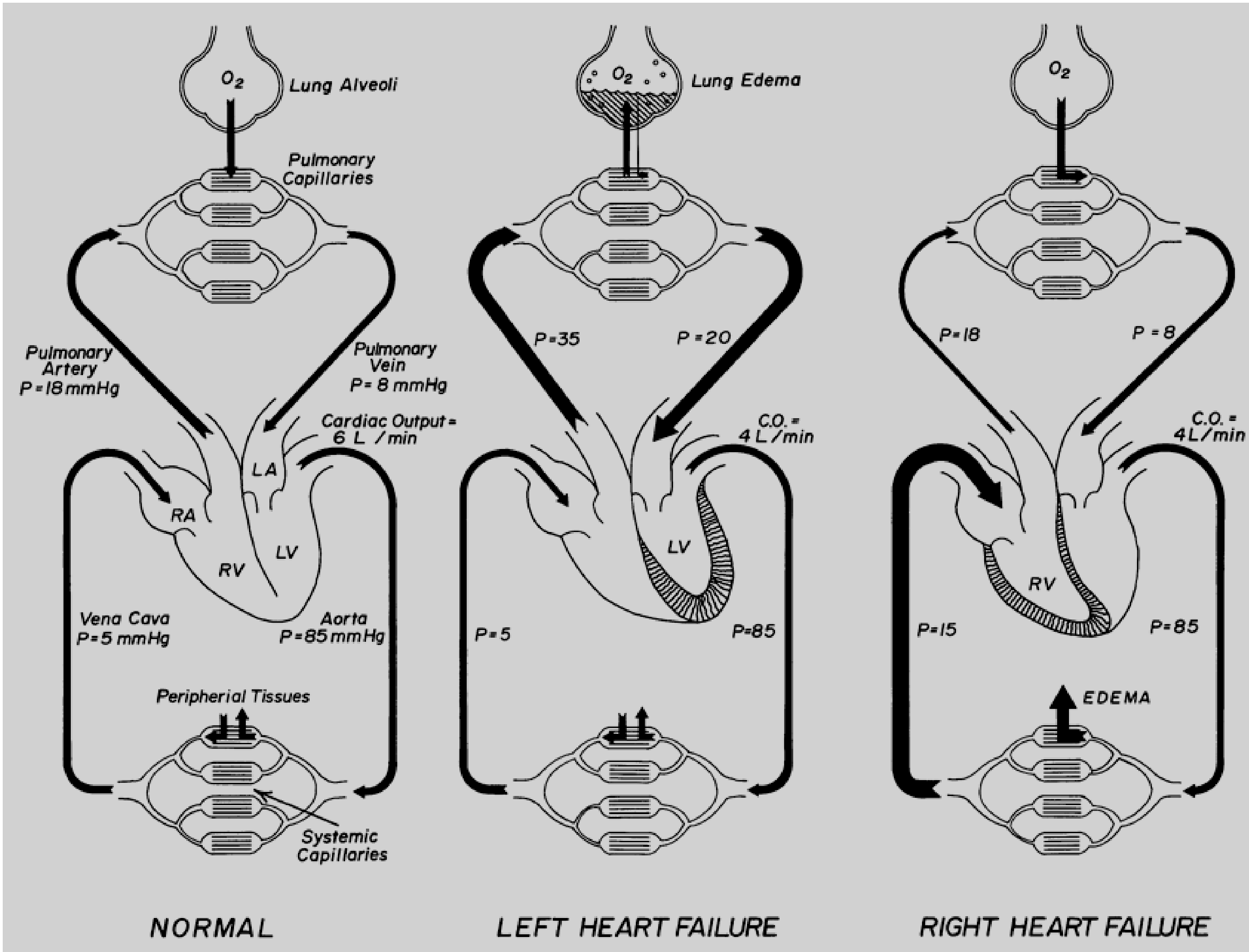
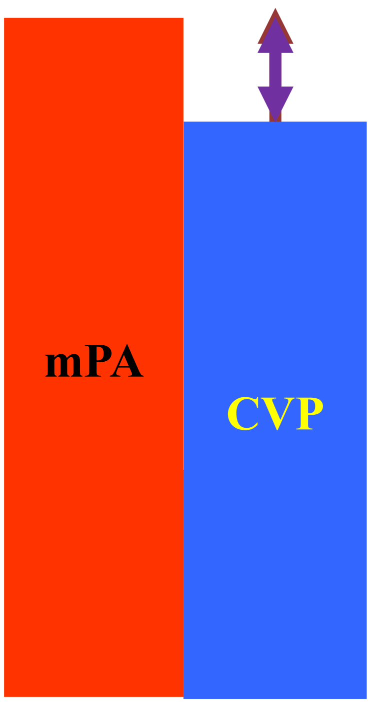
L’insuffisance cardiaque, une maladie pulmonaire ?

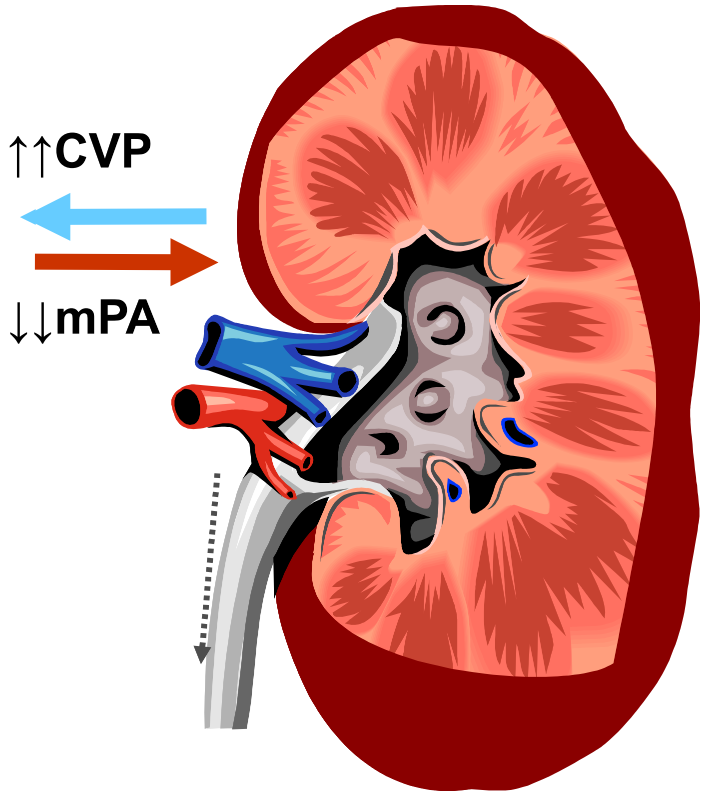
Guillaume JONDEAU
Hopital Bichat
Paris

Insuffisance cardiaque G vs Dte



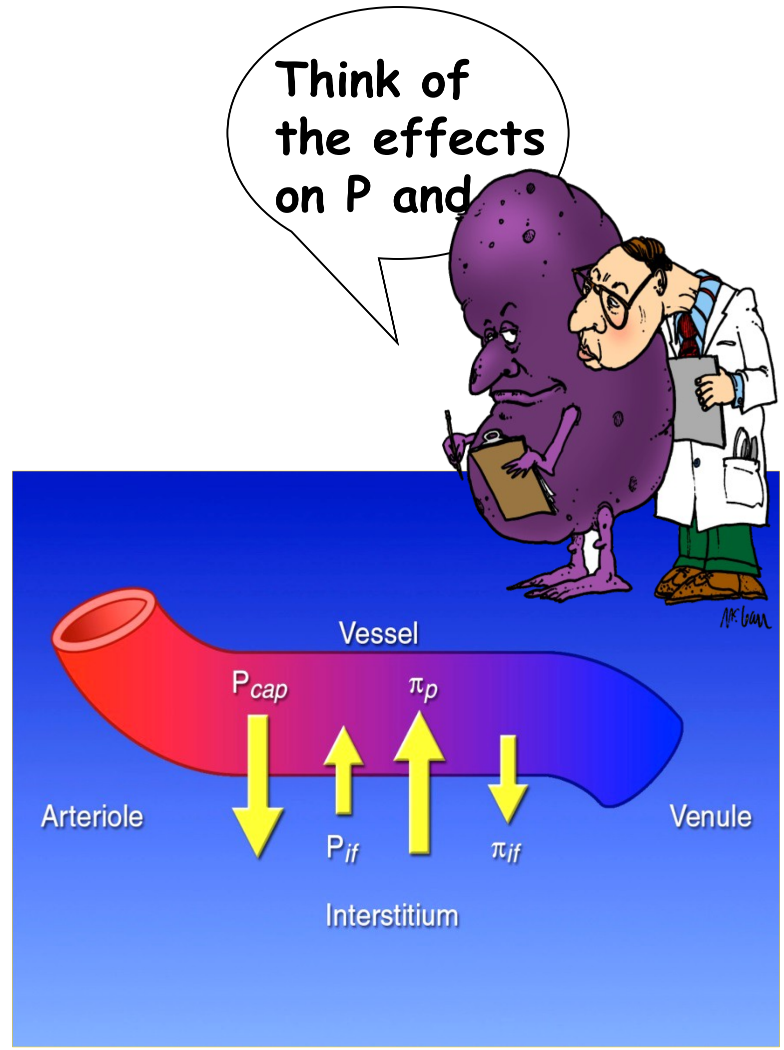
Hypervolémie



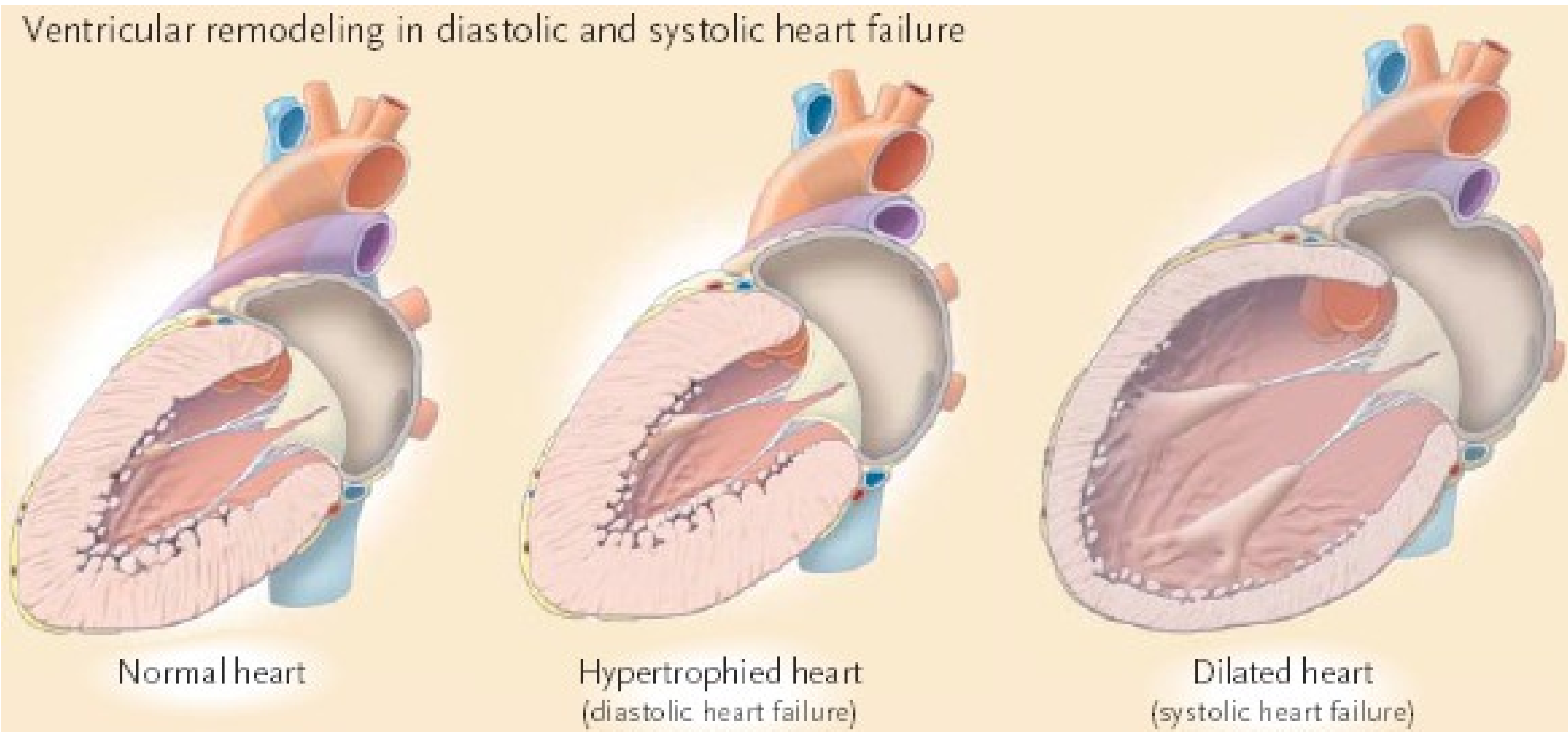


Mécanismes de la dyspnée

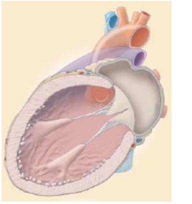
- Œdème pulmonaire
  - Hypoxie
  - Compliance pulmonaire
- Fatigue muscles respiratoires
- Ergorécepteurs périphériques
- …



Insuffisances cardiaques



progression de l’insuffisance cardiaque



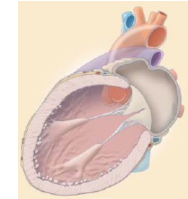
↓φονχτιον σψστολιθυε

Vasodilatateur

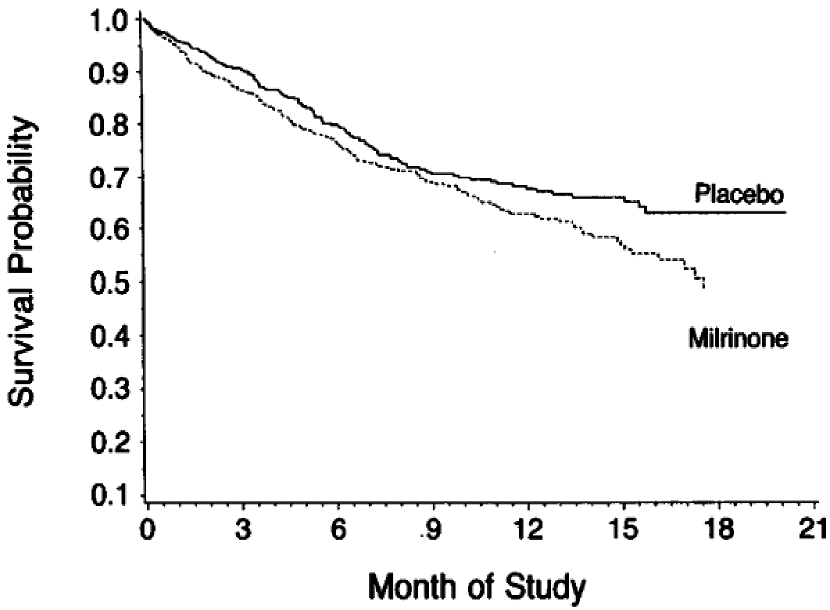
inotrope positif

↑ postcharge

Survie avec inotropes+



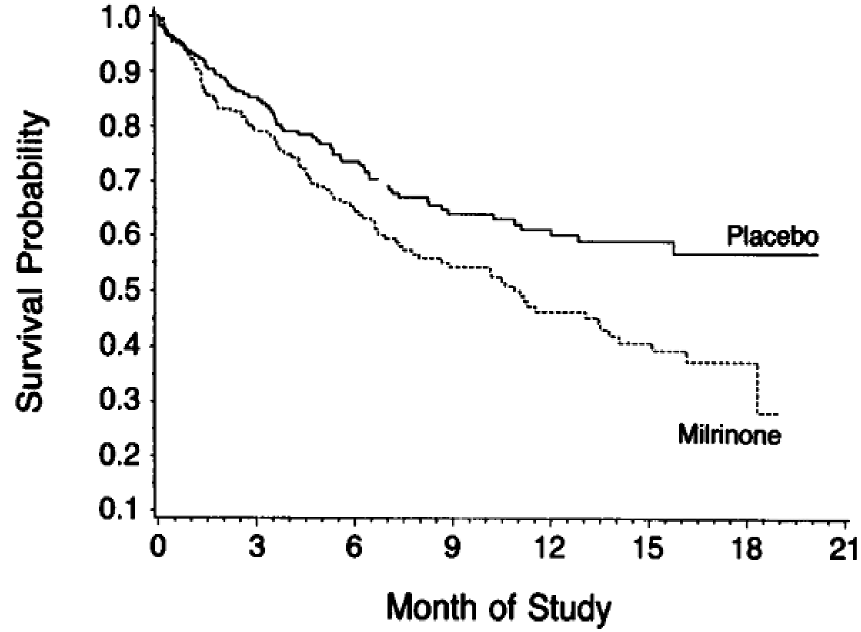
PROMISE NEJM 1991;325:1468



Placebo	527	375	270	185	137	77	21
Milrinone	561	395	284	184	132	74	14

Figure 1. Kaplan–Meier Analysis Showing Cumulative Rates of Survival in Patients with Chronic Heart Failure Treated with Milrinone or Placebo.

Mortality was 28 percent higher in the milrinone group than in the placebo group (P = 0.038). The numbers of patients at risk are shown at the bottom of the figure.

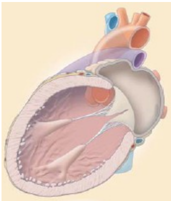


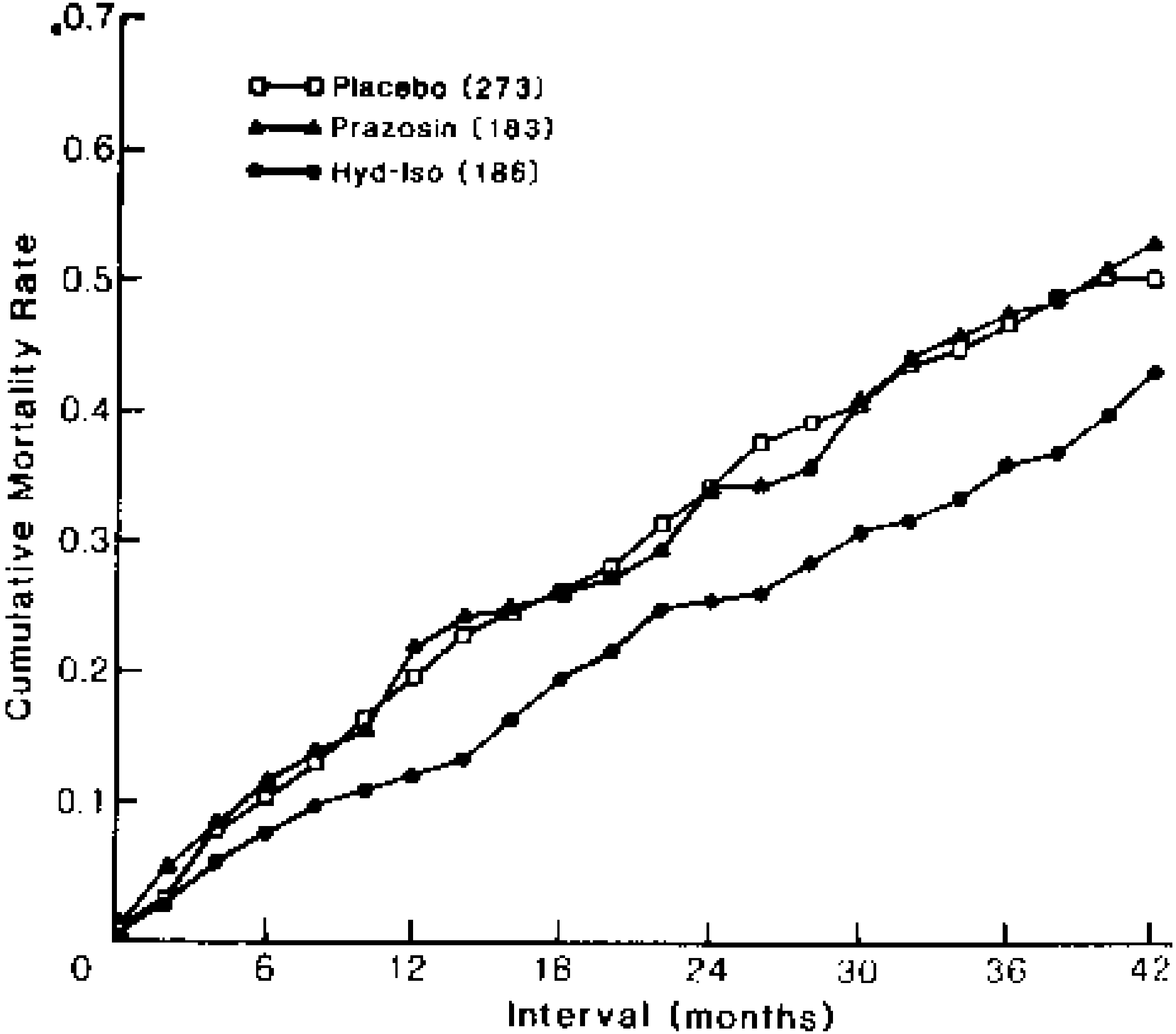
Placebo	224	159	116	78	59	35	12
Milrinone	233	155	109	69	49	30	6

Figure 2. Kaplan–Meier Analysis Showing Cumulative Rates of Survival in Patients with Class IV Heart Failure, According to Treatment Group.

Mortality was 53 percent higher in the milrinone group (P = 0.006).

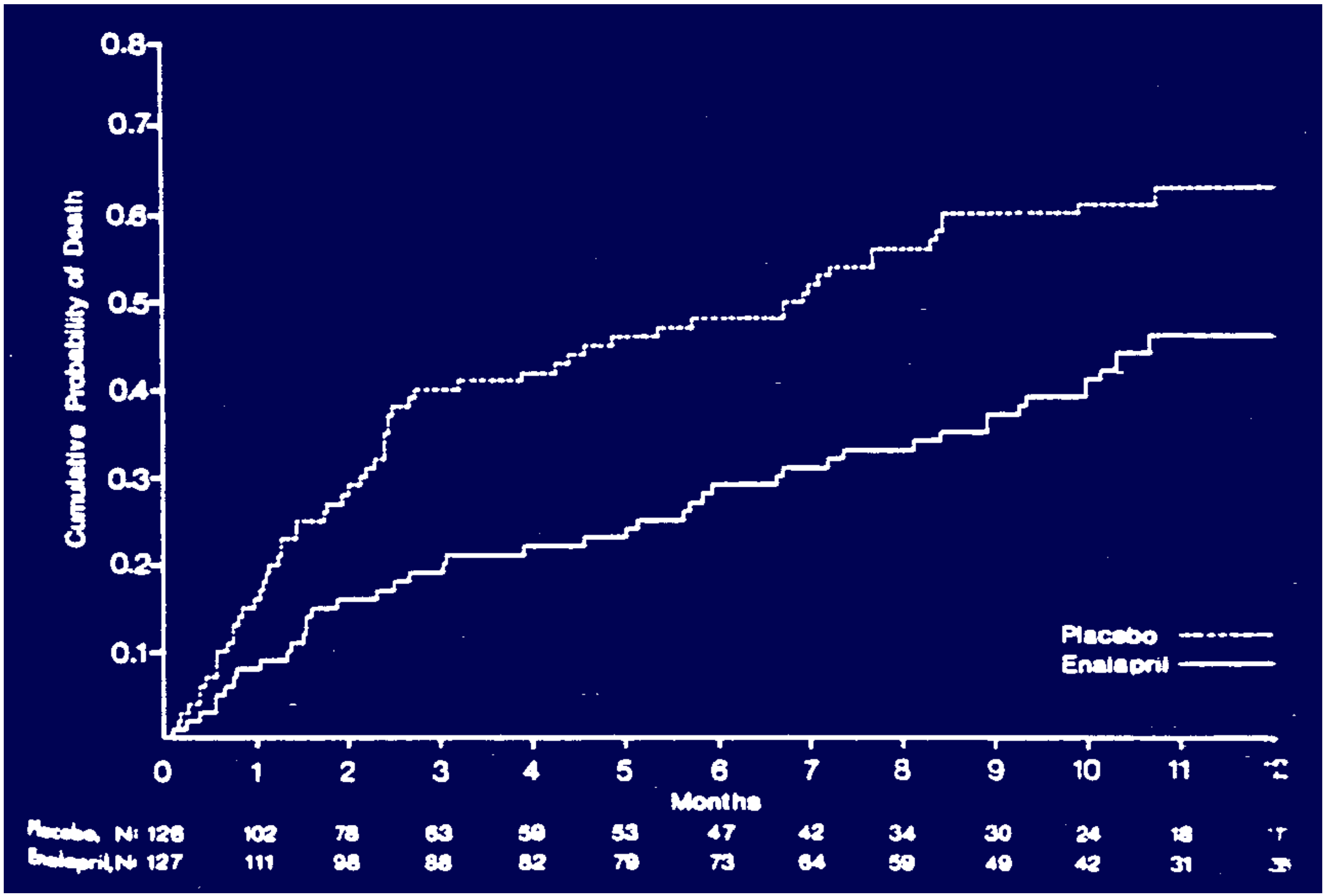
Survie avec vasodilatateur





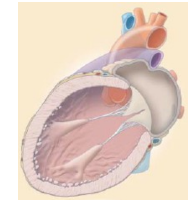
NEJM 1986;314:15

CONSENSUS I: âge 70 ans



NEJM 1987;316:1429-35

Progression de l’insuffisance cardiaque

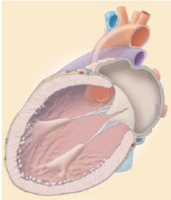


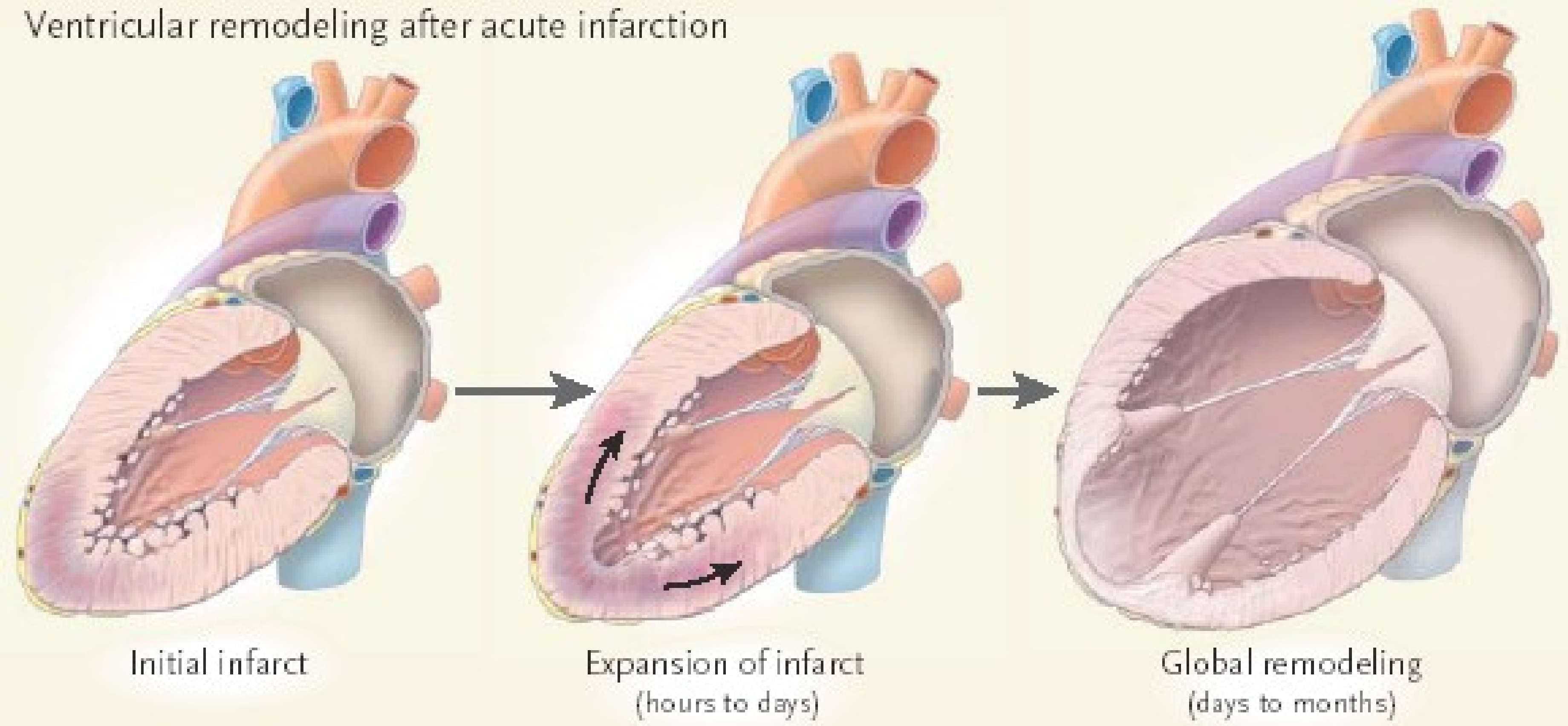
Dilatation VG
« remodeling »

↓φονχτιον σψστολιθυε

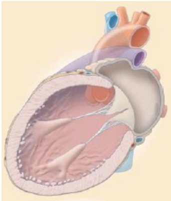
↑ postcharge

Remodelage après IDM



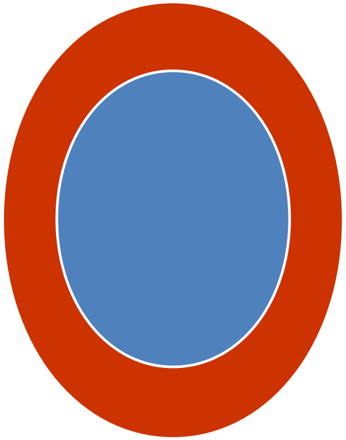


Conséquences à distance

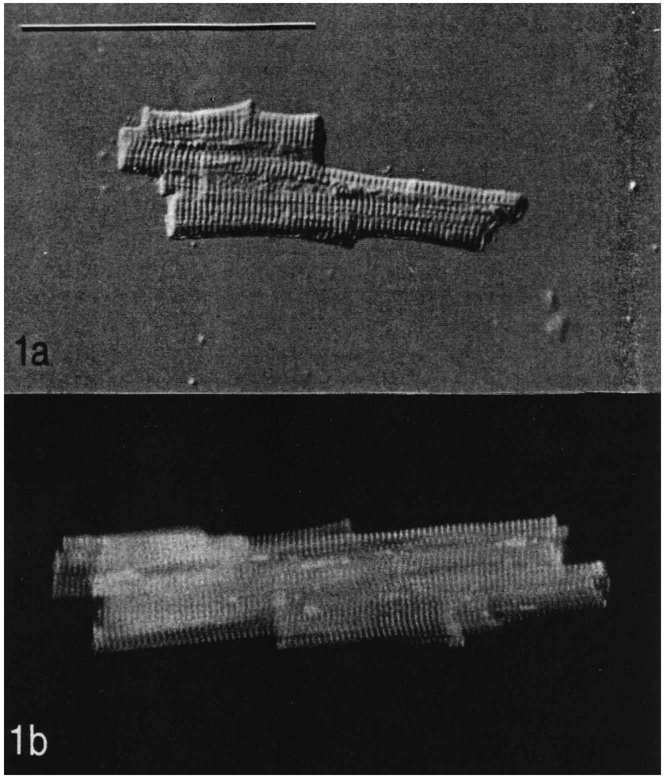


Loi de Laplace :

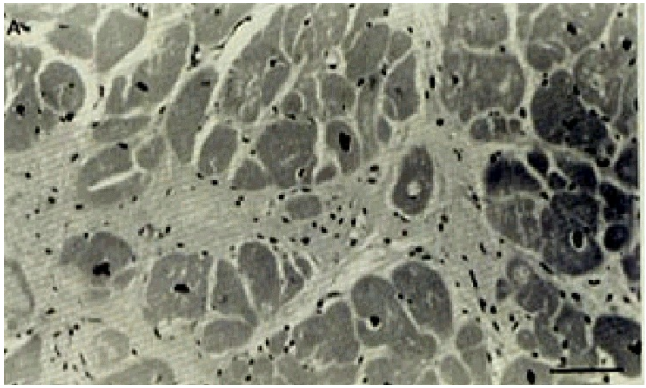
contrainte = P x r/e



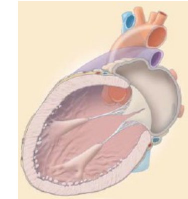
Hypertrophie des myocytes



Fibrose interstitielle à distance d'ur



progression de l’insuffisance cardiaque

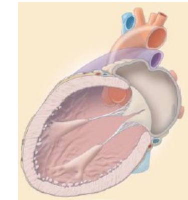


Dilatation VG
« remodeling »

↓φονχτιον σψστολιθυε

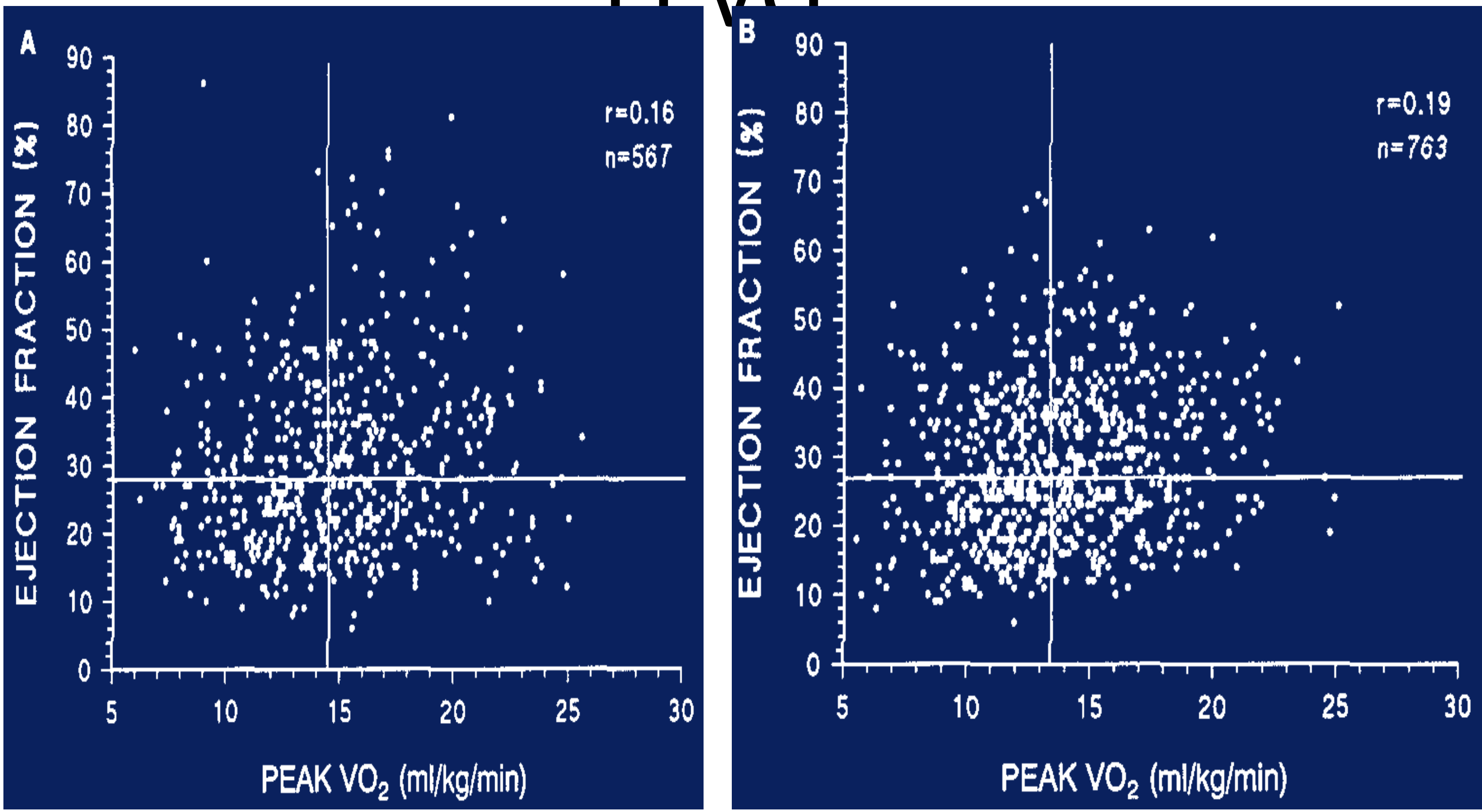
↑ postcharge

Muscle squelettique
Dysfonction endothéliale

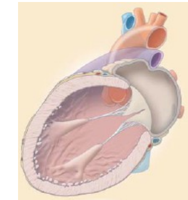


Indépendance de VO2 et FEVG

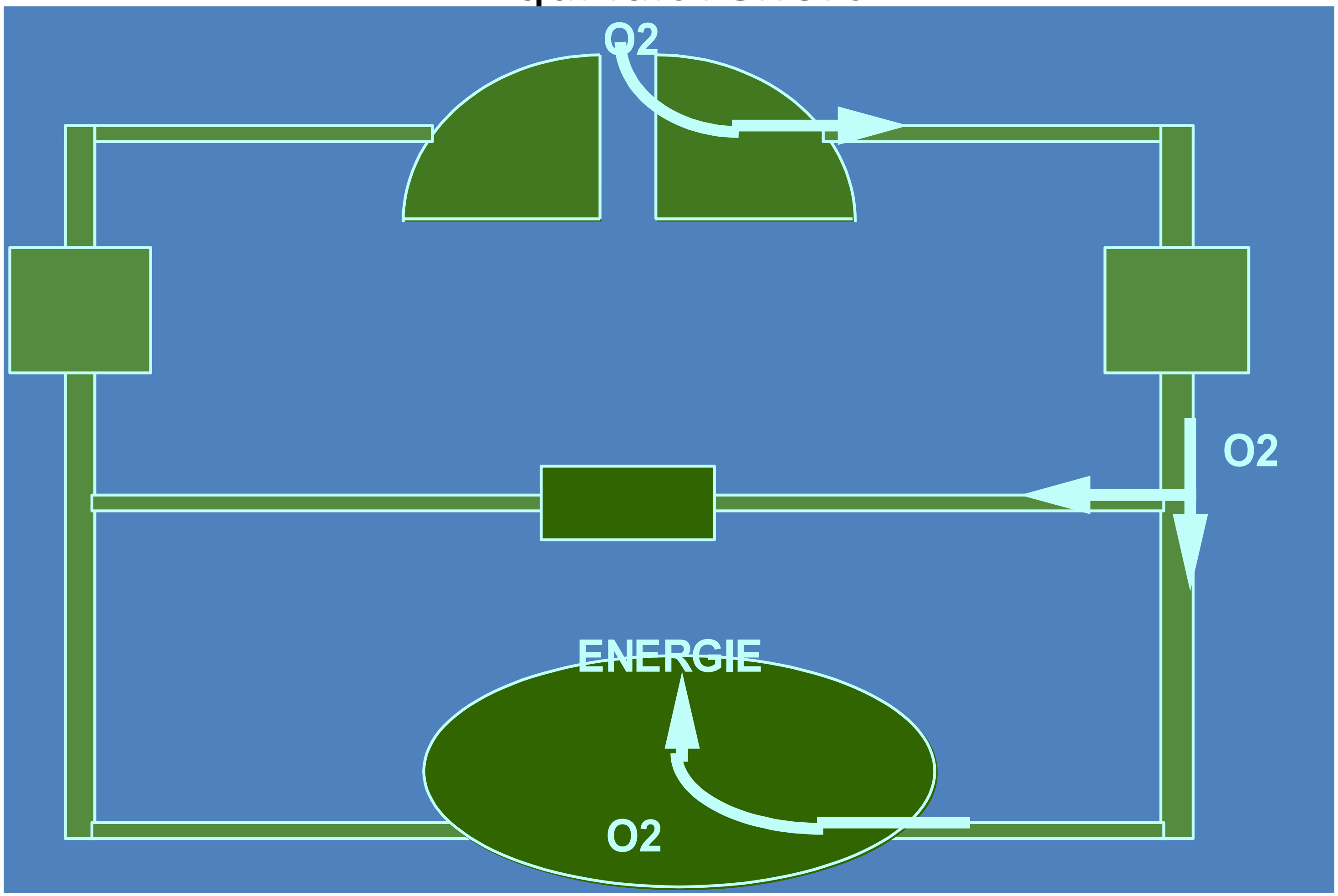
VeHF 1 **VeHF 2**



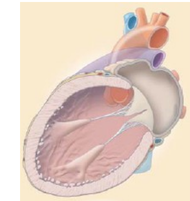
Circulation, 1993;87:VI-5

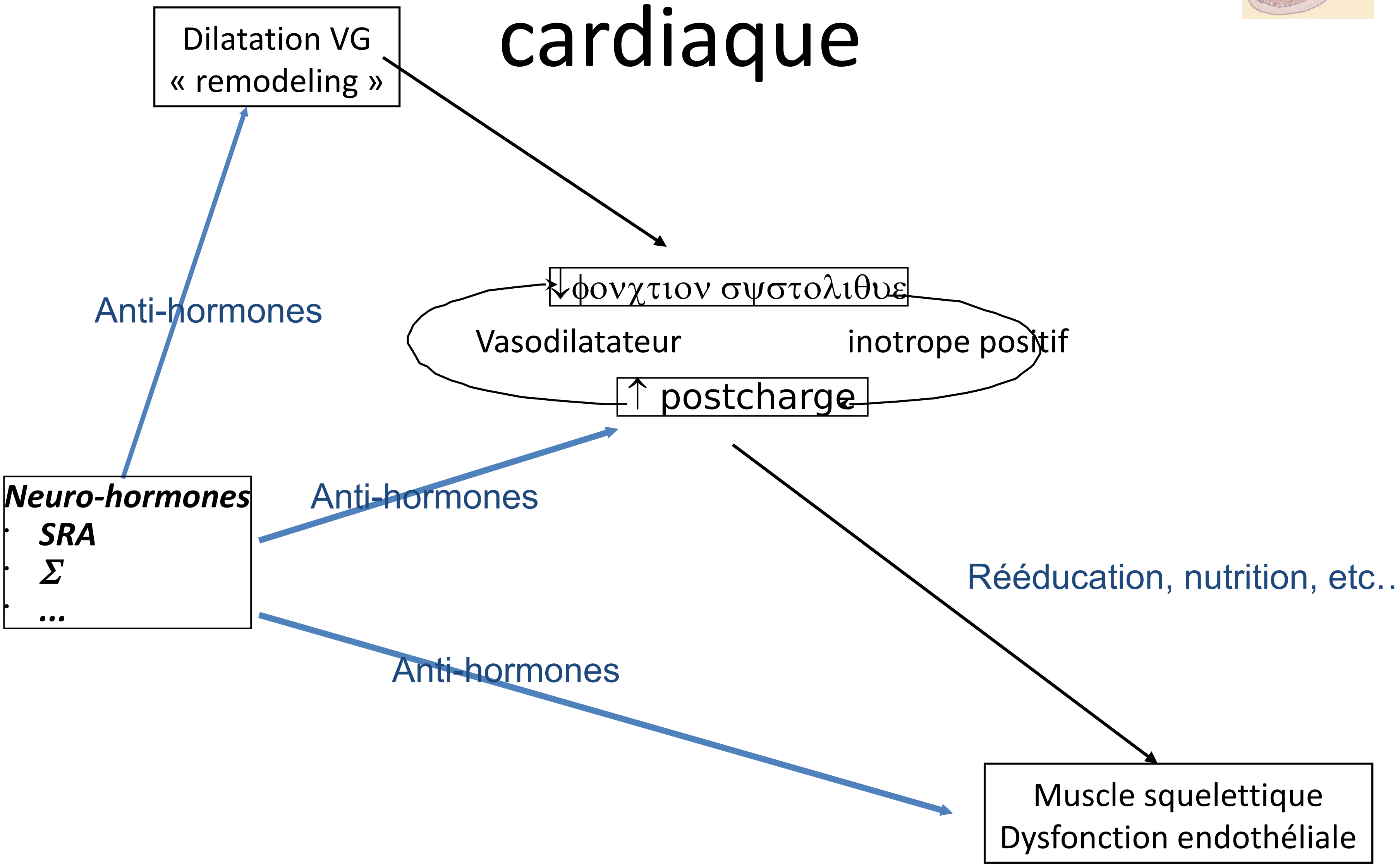


Le trajet de l'oxygène jusqu'au muscle squelettique qui fait l'effort

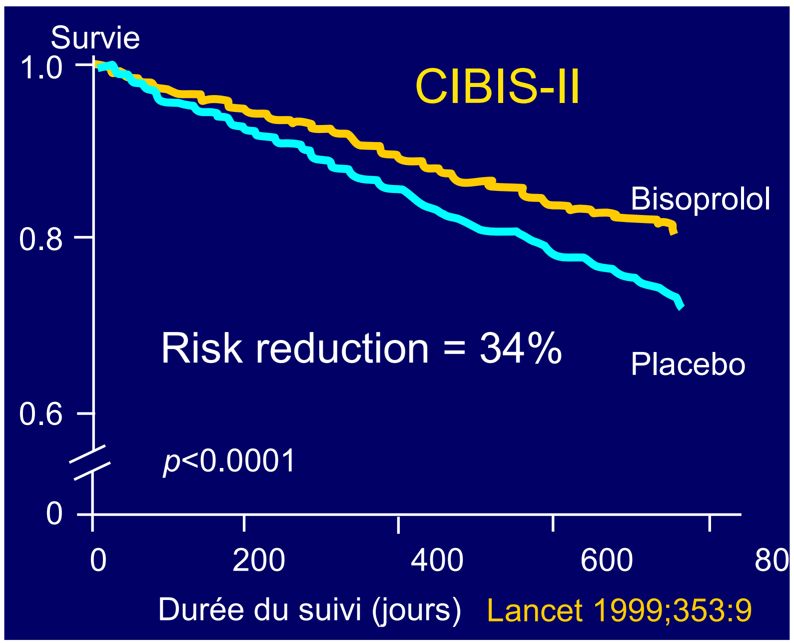


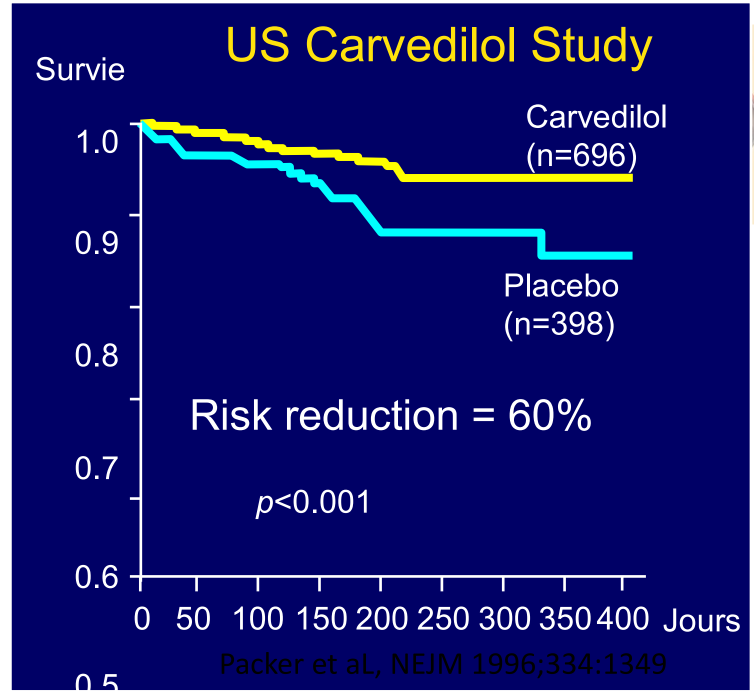
progression de l’insuffisance cardiaque

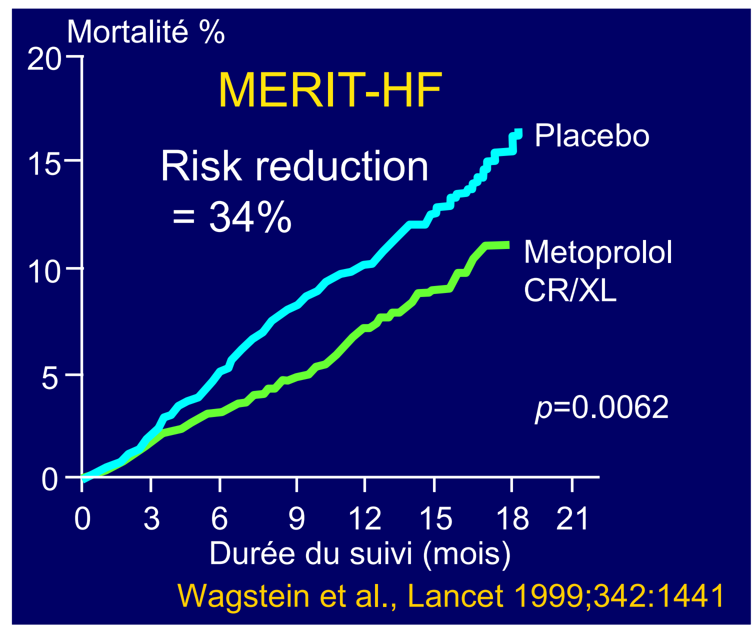




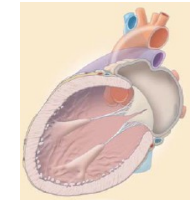
Années 1995-2000: Béta-Bloquants

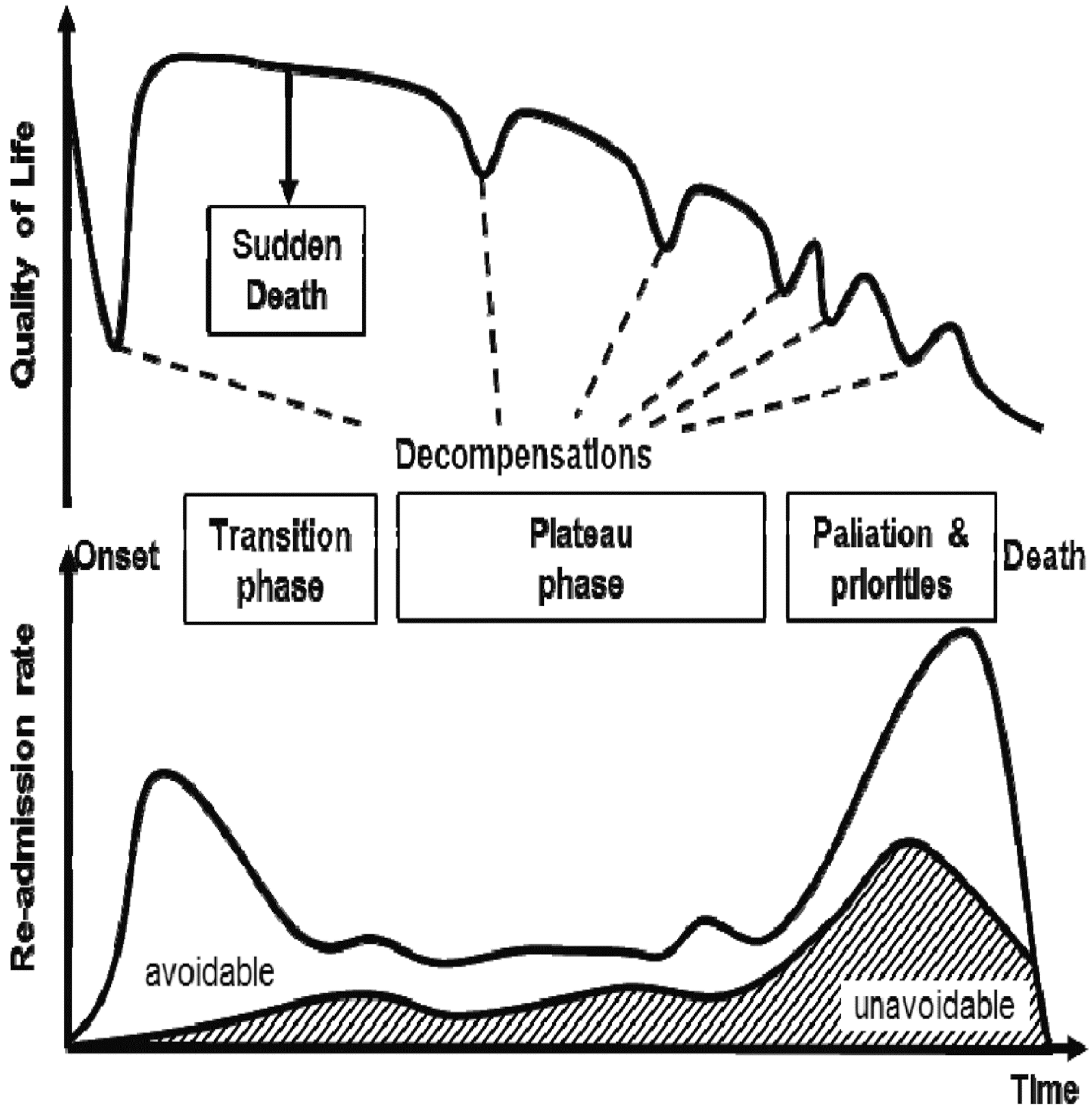






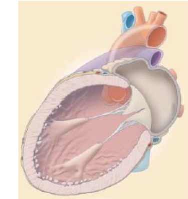
Métaanalyse : RR entre 30-35% à 1 an





Quality of Life
Sudden Death
Decompensations
Onset
Transition phase
Plateau phase
Paliation & priorities
Death
Re-admission rate
avoidable
unavoidable
Time

insuffisance cardiaque aigue



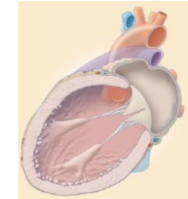
↓φονχτιον σψστολιθυε

Vasodilatateur

inotrope positif

↑ postcharge

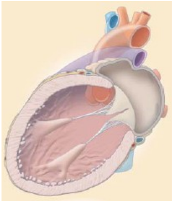
Philosophie du traitement

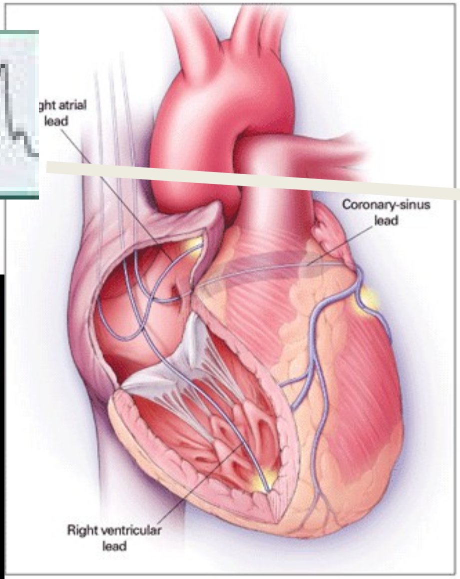


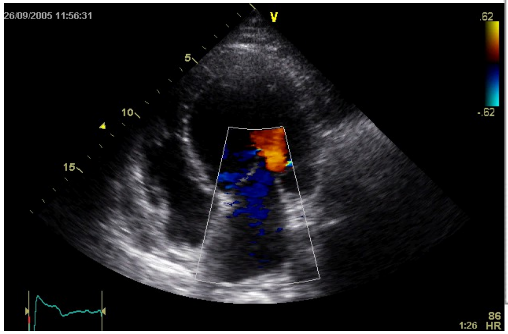
- Insuffisance cardiaque aiguë
  - hémodynamique
- Insuffisance cardiaque chronique
  - limiter la congestion
    - diurétiques
    - critères de suivi
  - limiter l ’évolution de la maladie (↓ mortalité)
    - anti-hormones
      - IEC, ARAII
      - béta-bloquants
      - aldactone
    - doses maximales tolérées
  - Economie d’énergie
    - Éviter tachycardie

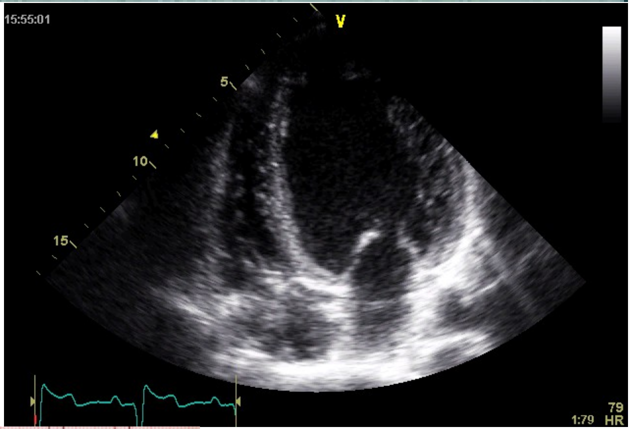
Chronique		Aigue
NON	Vasodilatateurs	OUI
NON	Inotropes	±
OUI	Blocage hormonal	NON
OUI	Diurétiques Education	OUI

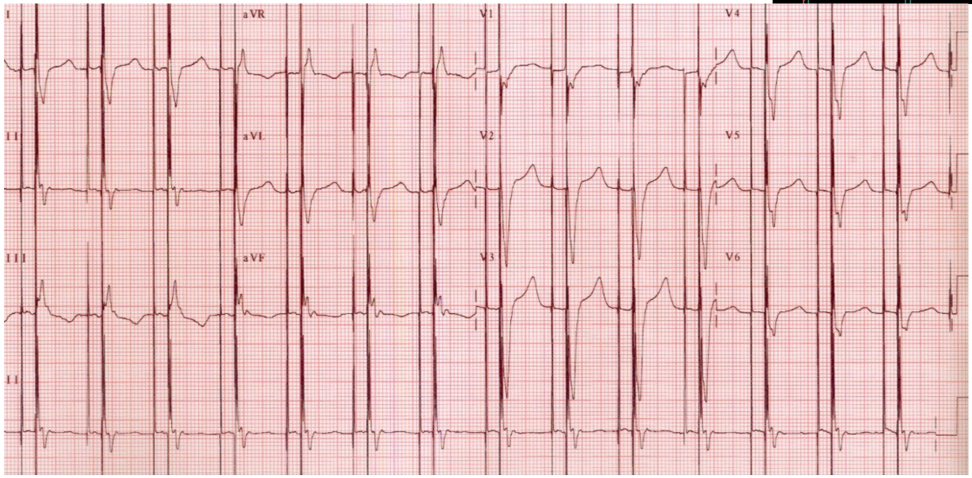
Resynchronisation cardiaque



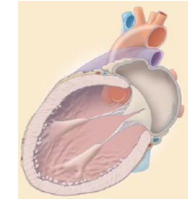






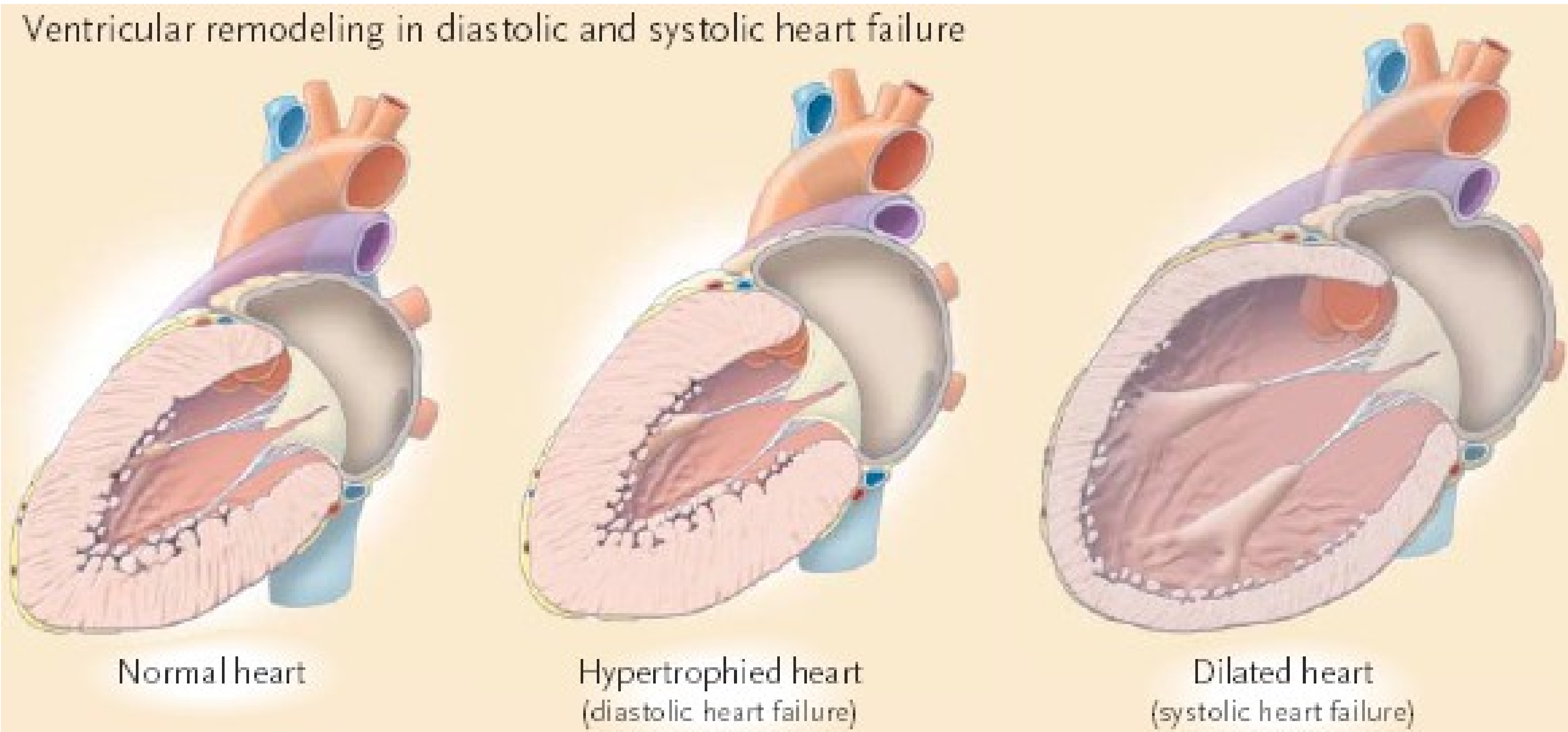


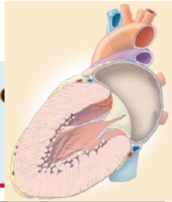
Take home messages



- Insuffisance cardiaque par dysfonction systolique
  - Aigue: maladie hémodynamique
  - Chronique:
    - Maladie hormonale auto-évolutive
    - Évolue par poussées

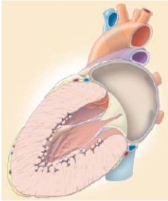
Insuffisances cardiaques





Web Table 9.1 **Phase II and III clinical trials performed in patients with heart failure with mid-range ejection fraction and heart failure with preserved ejection fraction**

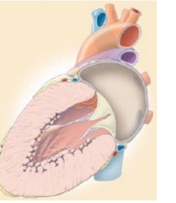
Trial	Intervention	Major inclusion criteria	Mean follow-up	Primary endpoints
PEP-CHF[320]	Perindopril vs placebo.	LV wall motion index ≥1.4 (corresponding to LVEF ≥40%), symptomatic HF treated with diuretic, diastolic dysfunction in echocardiography, age ≥70 y.	2.1 y	No difference in combined all-cause mortality or cardiovascular hospitalization (36% vs 37%, P =0.35).
I-PRESERVE [318]	Irbesartan vs placebo.	LVEF ≥45%, NYHA III–IV with corroborative evidence, or NYHA II with HF hospitalization in recent 6 months, age ≥60 y.	4.1 y	No difference in combined all-cause mortality or HF hospitalization (24% vs 25%, P =0.54).
CHARM-Preserved[319]	Candesartan vs placebo.	LVEF >40%, NYHA II–IV, history of cardiac hospitalization.	3.0 y	Trend towards a reduction in combined cardiovascular mortality or HF hospitalization by 11% (22% vs 24%, unadjusted P =0.12, adjusted P =0.051).
Aldo-DHF[330]	Spironolactone vs placebo.	LVEF ≥50%, NYHA II–III, peak VO_2 ≤25 mL/min/kg, diastolic dysfunction on echocardiography or atrial fibrillation, age ≥50 y.	1.0 y	Reduction in E/e' by – 1.5 (P < 0.001) No change in peak VO_2 (P =0.81).
TOPCAT[310]	Spironolactone vs placebo.	LVEF ≥45%, ≥1 HF sign, ≥1 HF symptom, HF hospitalization within recent 12 months, or BNP ≥100 pg/mL or NT-proBNP ≥360 pg/mL, age ≥50 y.	3.3 y	No difference in combined cardiovascular death, aborted cardiac arrest, or HF hospitalization (19% vs 20%, P =0.14).
SENIORS[173]	Nebivolol vs placebo.	HF confirmed as HF hospitalization in recent 12 months and/or LVEF ≤35% in recent 6 months, age ≥70 y, 36% with LVEF >35%.	1.8 y	Reduction in combined all-cause mortality or cardiovascular hospitalization by 14% (31% vs 35%, P =0.04).
DIG-PEF[323]	Digoxin vs placebo.	HF with LVEF >45%, sinus rhythm.	3.1 y	No difference in combined HF mortality or HF hospitalization (21% vs 24%, P =0.14)
PARAMOUNT[309]	Sacubitril/ valsartan vs valsartan.	HF with LVEF ≥45%, NYHA II-III, NT-proBNP >400 pg/mL.	12 w	Reduction in NT-proBNP: ratio of change sacubitril/ valsartan 0.77, 95% CI 0.64–0.92 (P =0.005).
RELAX[311]	Sildenafil vs placebo.	HF with LVEF ≥45%, NYHA II-IV, peak VO_2 <60% of reference values, NT-proBNP >400 pg/mL or high LV filling pressures.	24 w	No change in peak VO_2 (P =0.90).



Augmentation de la PA avec l’age

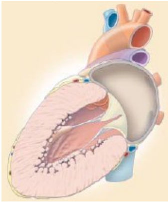
Burt VL and al. Hypertension 1995;26:305-313

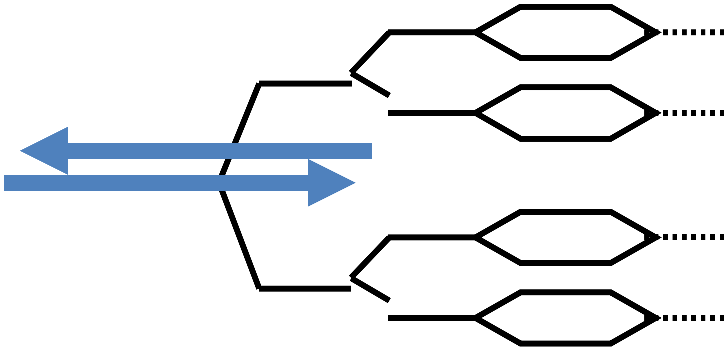
Augmentation de la pression pulsée



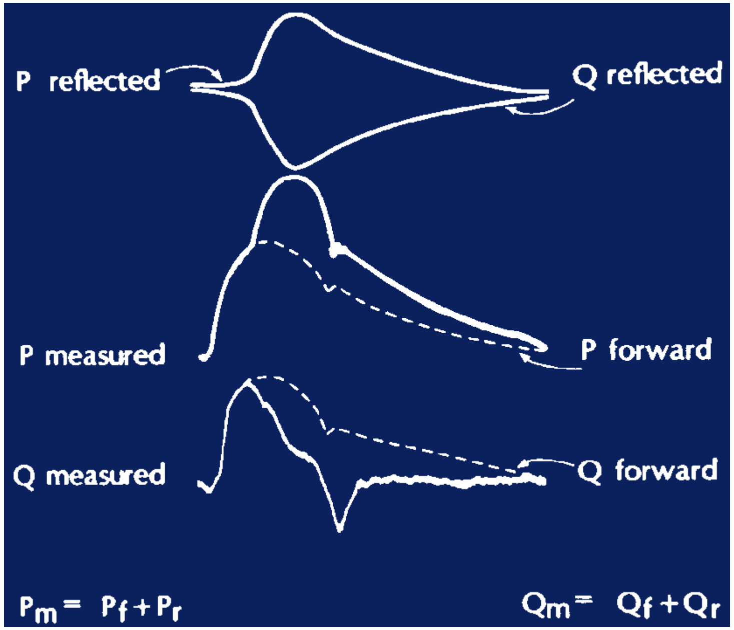
- Diminution de la compliance des vaisseaux
  - Plus d’augmentation de pression pour un meme volume

- Diminution de la compliance des vaisseaux
  - Augmentation de la vitesse de l’onde de pouls

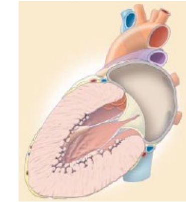


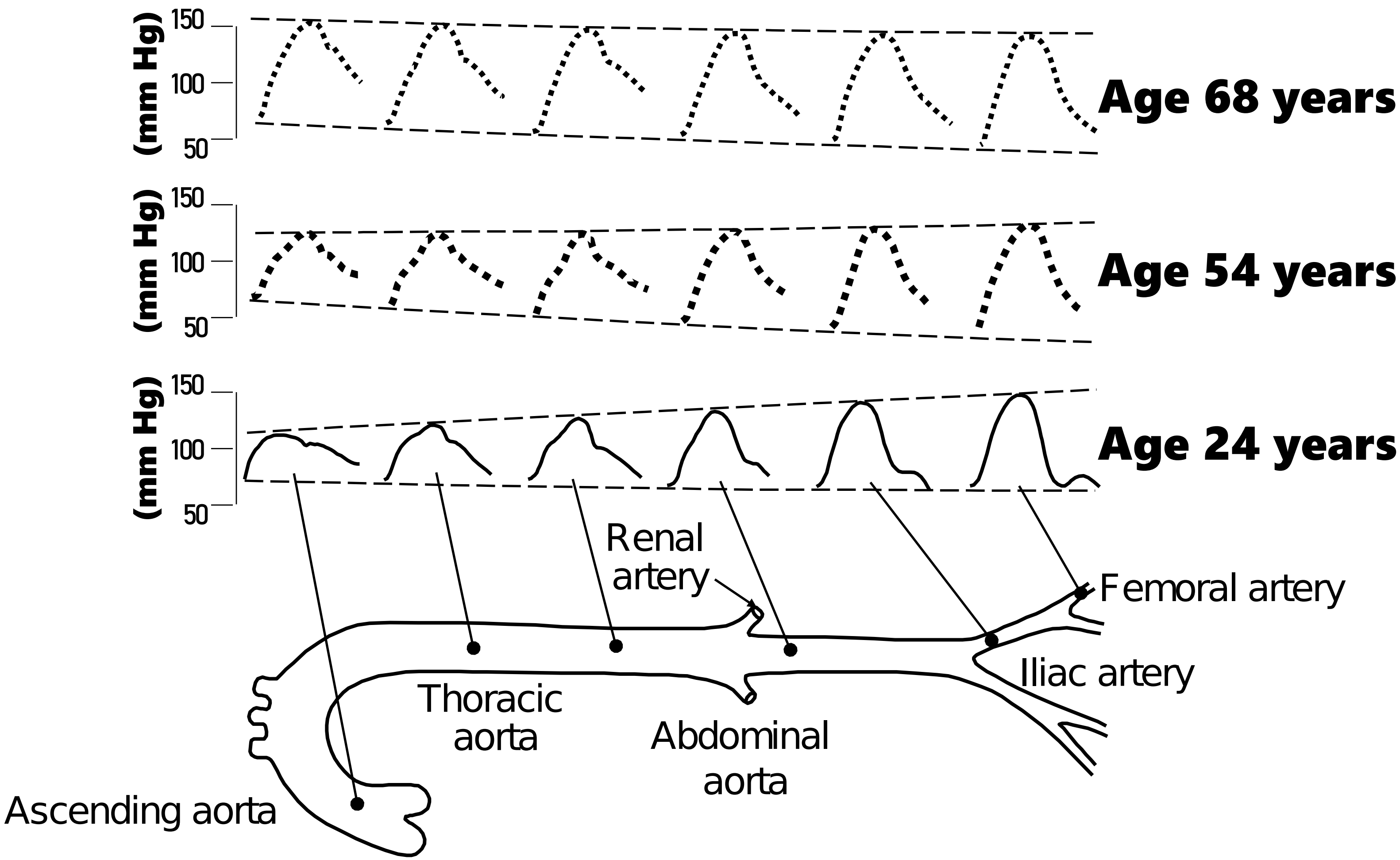


Reflexion sites

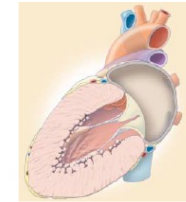


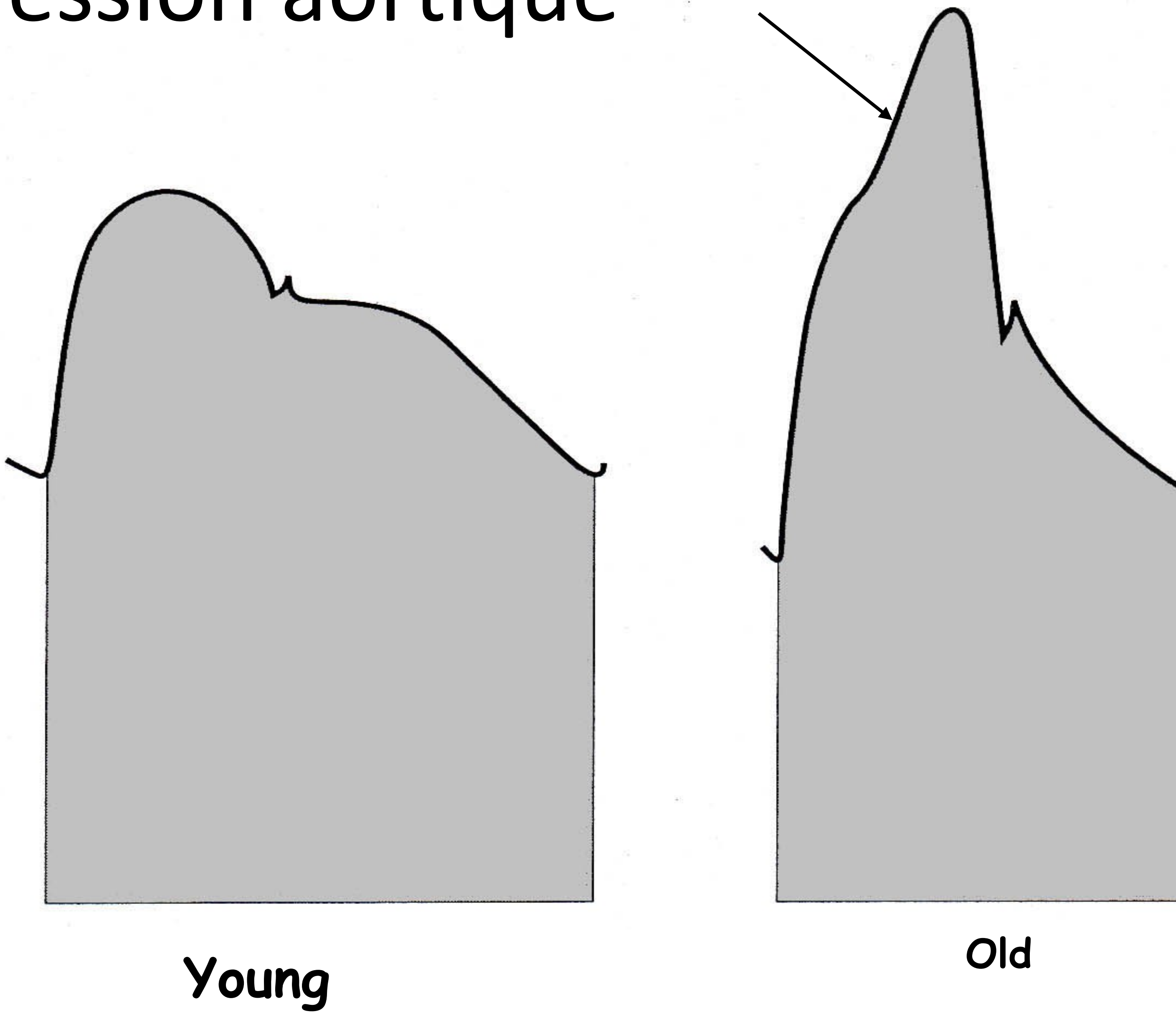
Nichols et al. 1987



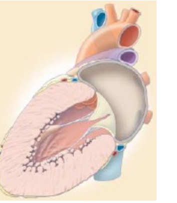

150
100
50
(mm Hg)
Age 68 years
Age 54 years
Age 24 years
Renal artery
Femoral artery
Thoracic aorta
Abdominal aorta
Iliac artery
Ascending aorta

Pression aortique



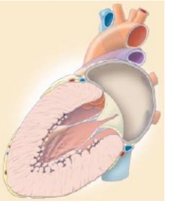


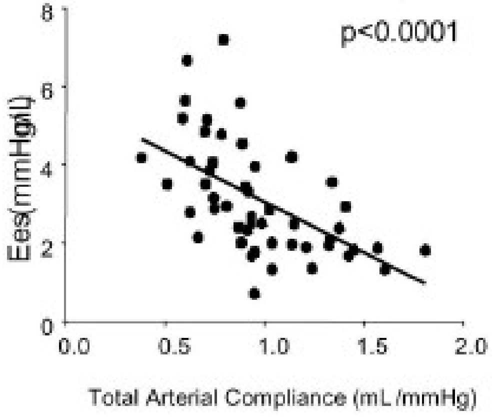
Rigidification des artères



- Age
- Diabetes
- Maladie rénale
- Hypertension
- …

Relation cœur / vaisseaux



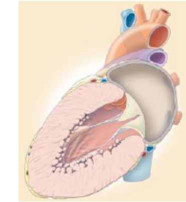


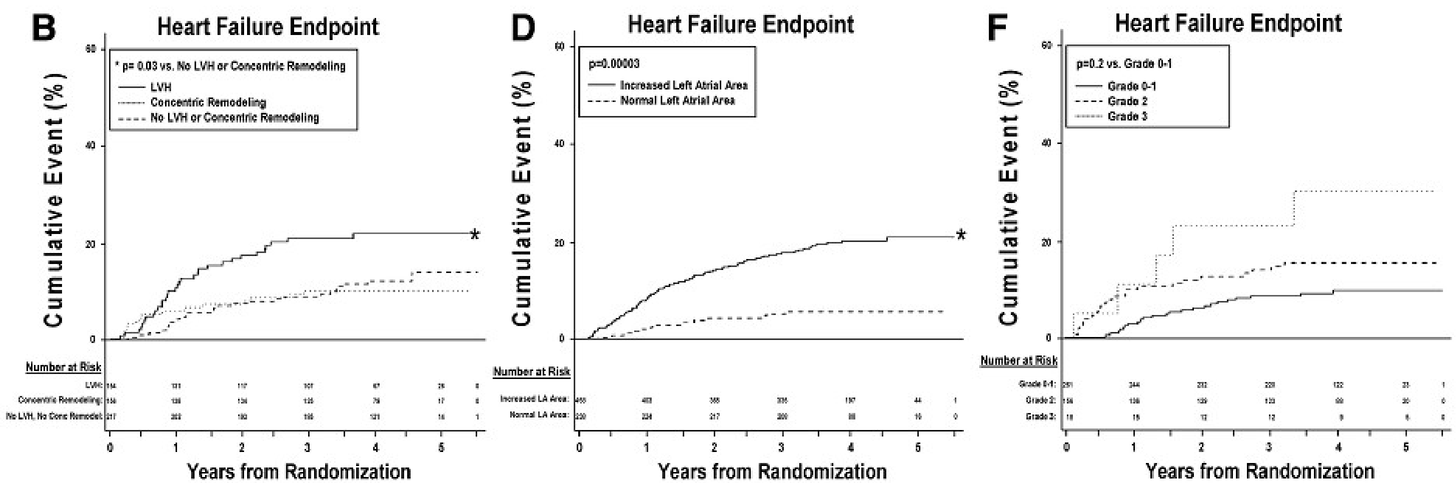
Kawagushi M, Circulation 2003;107:714

- Les mêmes facteurs diminuent la compliance VG et des vaisseaux
  - diabète
  - age…
- Vaisseaux rigides entrainent un cœur rigide

Diagnostic ?

I PRESERVE paramètres associés avec hospitalisation pour insuffisance cardiaque
MR Zile, Circulation 2011;124:2491



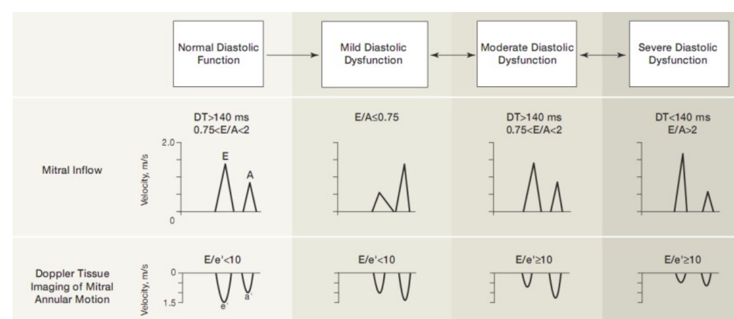


HVG

Taille OG

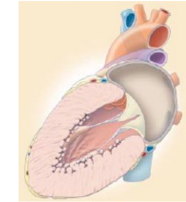
Pression OG

Concentric remodeling
WT/DD≥0.42 without hypertrophy



F Bursi, JAMA.2006;296:2209-2216

Échographie pleuro-pulmonaire
Réanimation 12 (2003) 19–29; D.A. Lichtenstein



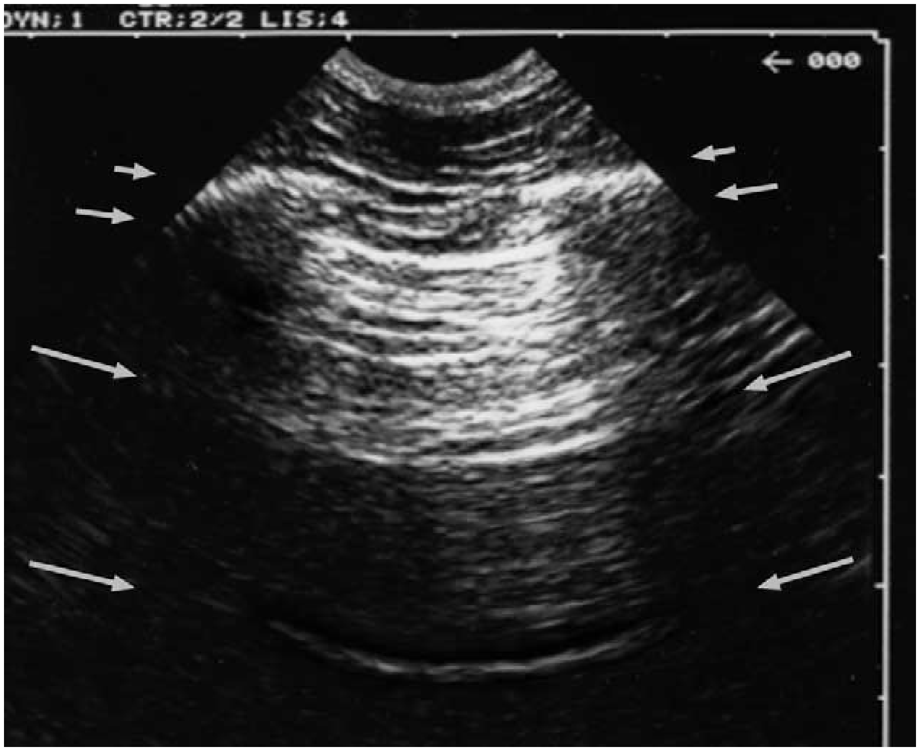
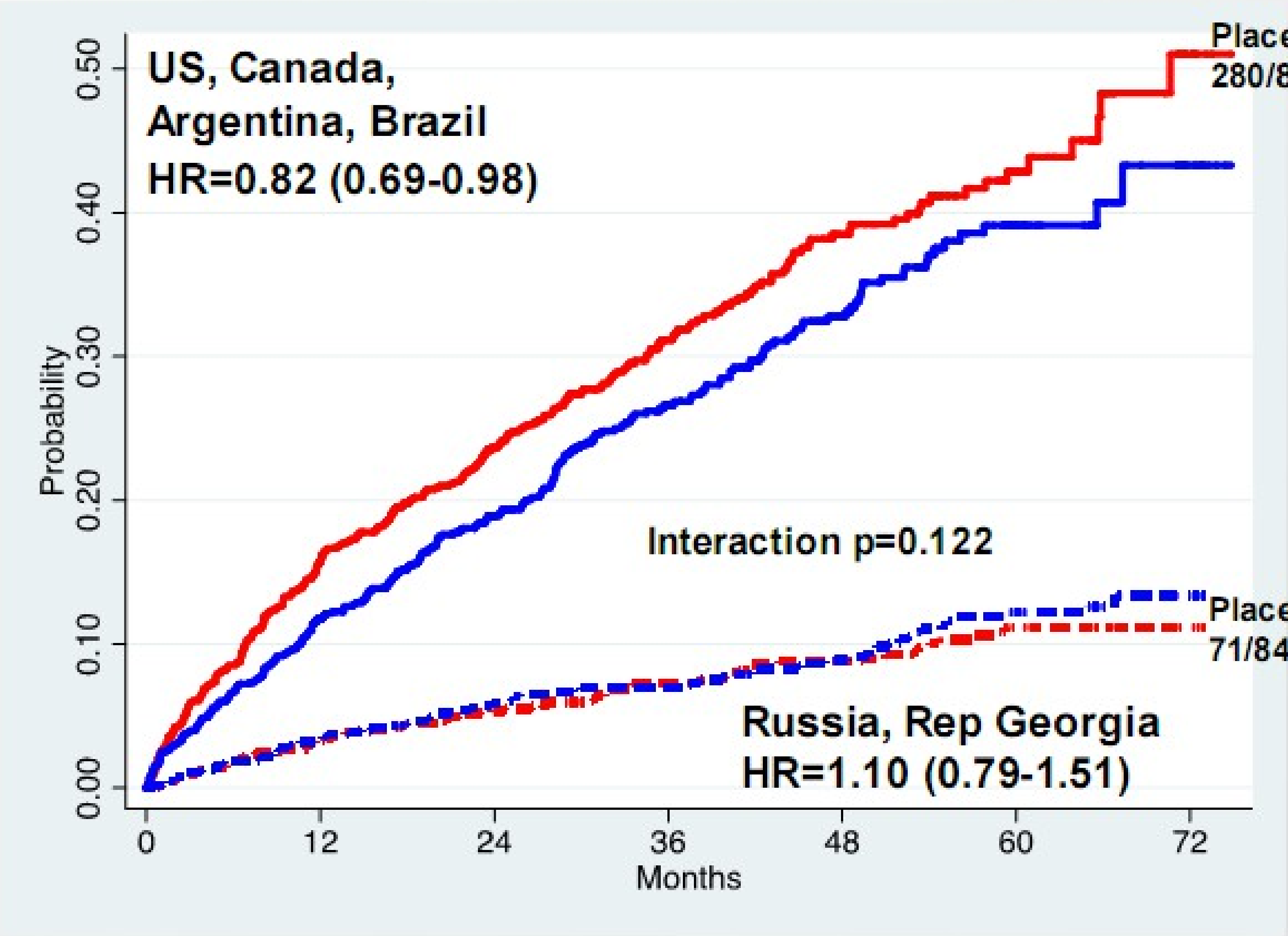


Fig. 1. (Poumon normal, lignes A). Coupe longitudinale d'un espace intercostal. A priori, tout n'est qu'artefact, puisque entre les artefacts osseux se profilent des artefacts aériques. Toutefois, le dôme des côtes (flèches courtes) et la ligne pleurale (flèche intermédiaires) dessinent un profil caractéristique, évoquant une chauve-souris vue de face et dont le dos est matérialisé par la ligne pleurale. Noter des lignes horizontales de répétition de la ligne pleurale (flèches longues) ou lignes « A », un élément qui fait partie intégrante de la sémiologie pulmonaire.

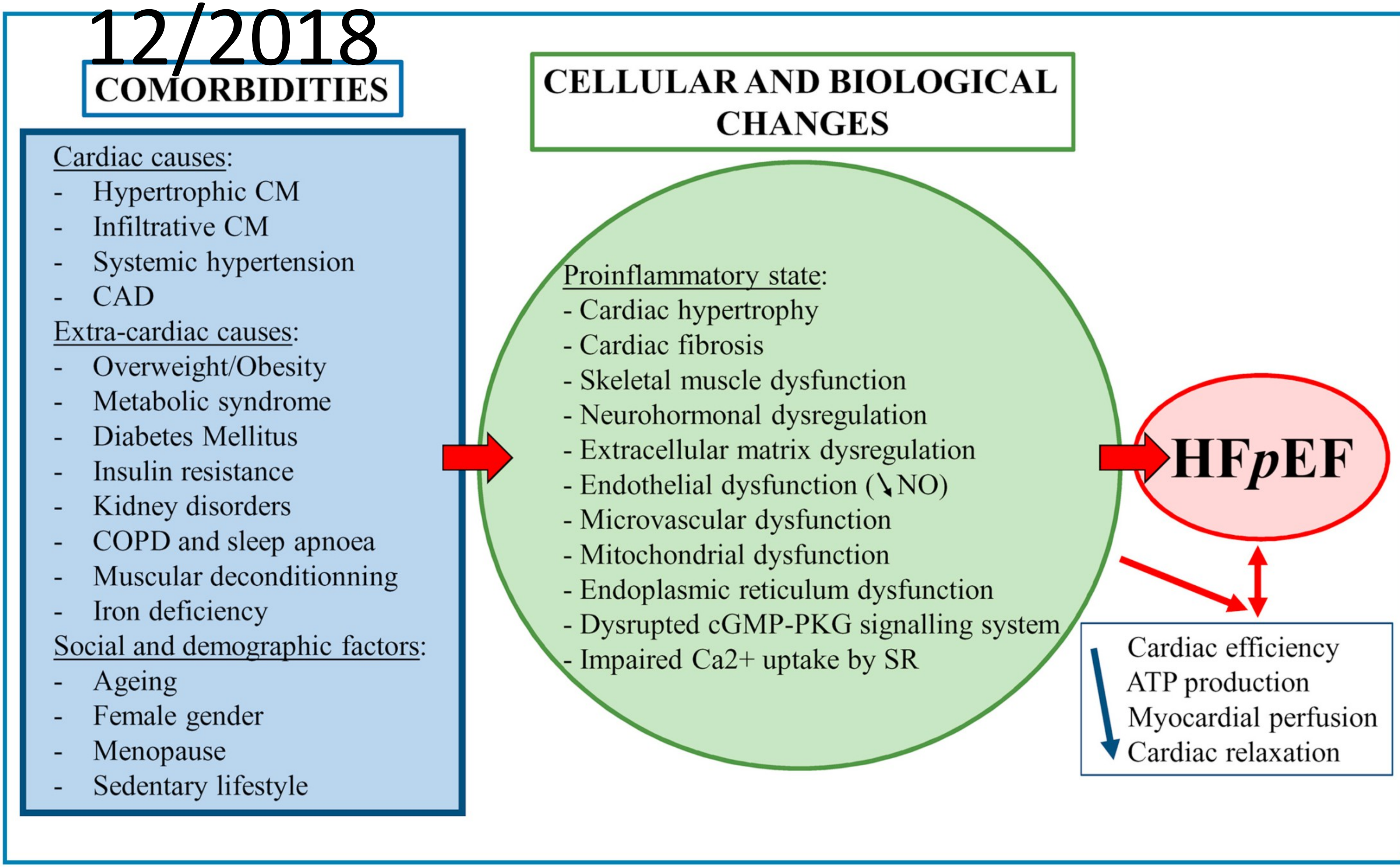
Fig. 2. (Fusées pleurales). Ces artefacts aériques en queue de comète ont la particularité d'être bien définis tels des rayons laser (on en dénombre quatre ou cinq), à disposition verticale, de naître strictement de la ligne pleurale et de s'étendre jusqu'à la limite inférieure de l'écran sans épuisement. Cet ensemble définit les « lignes B ». Plusieurs « lignes B » simultanément visibles définissent des « fusées pleurales ». Des « fusées », qui peuvent se voir chez le sujet normal si confinées aux bases, signent le syndrome interstitiel aigu quand elles sont diffuses. Patient présentant un oedème aigu pulmonaire.

Exploratory (post-hoc): Placebo vs. Spiro by region

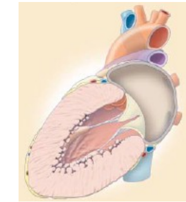
TOPCAT
Funded by t... I



Y Juilliere, Arch Cardiovasc Dis 12/2018



Patient en insuffisance cardiaque à FE préservée



Bon contrôle tensionnel ?
(MAPA si doute)

oui

Non : Adapter traitement médicamenteux (IEC, diurétiques, Ical, BB)
Évoquer sténose artère rénale

FA bien ralentie ?
(Holter si doute)

oui

Non: BB, Ical ralentisseur, voire digoxine
Si maladie oreillette, considérer PM

Altération de la fonction rénale ?

Attention AINS

Nephroangiosclérose: Lasilix 40 mg/j

Hb normale ?

oui

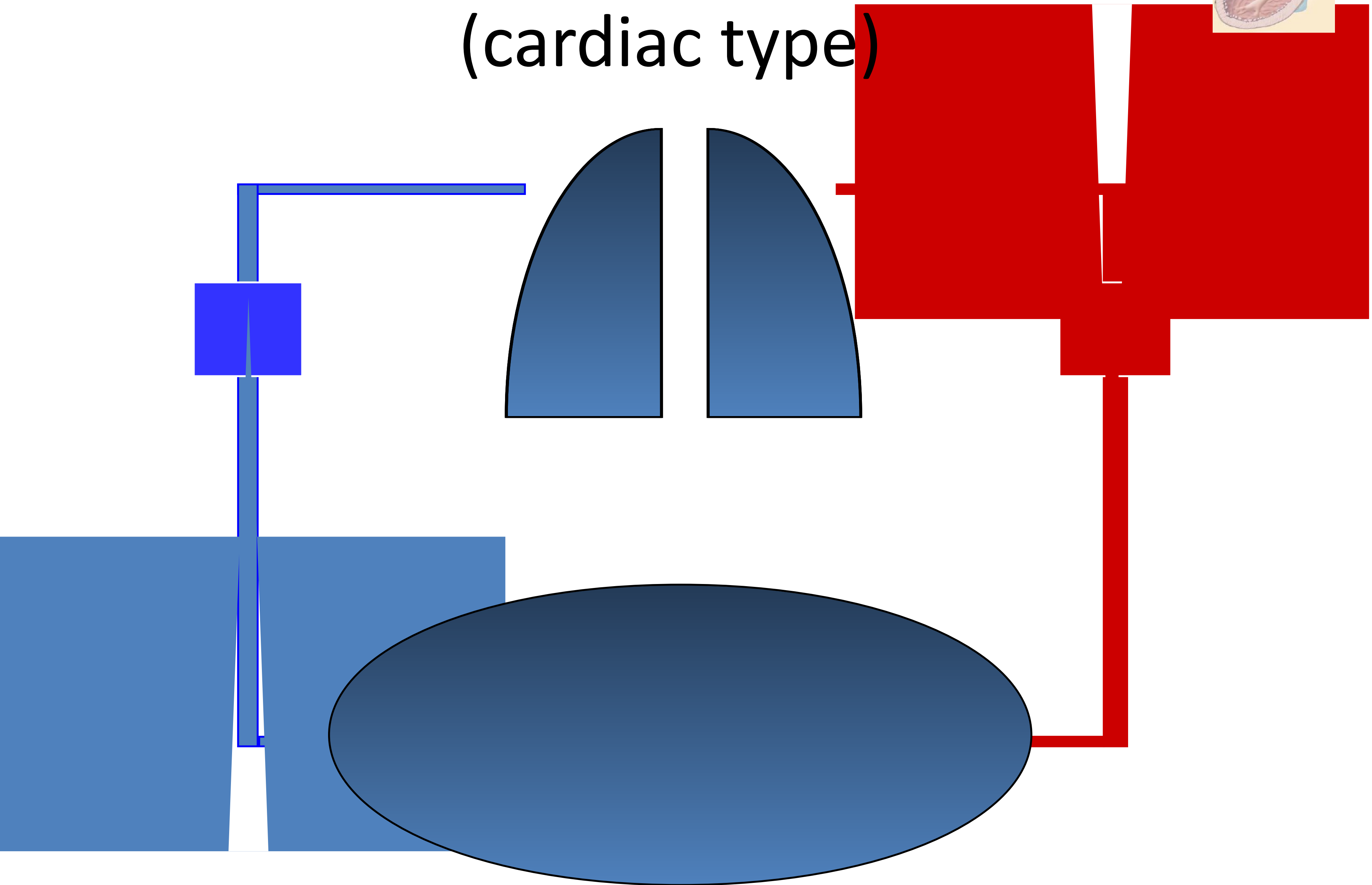
Non: Bilan martial, supplémentation au besoin

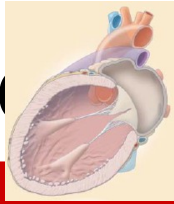
Evaluation hydratation, adapter la dose de diurétiques,
aldactone si possible

Education thérapeutique: attention deshydratation, attention surcharge sel

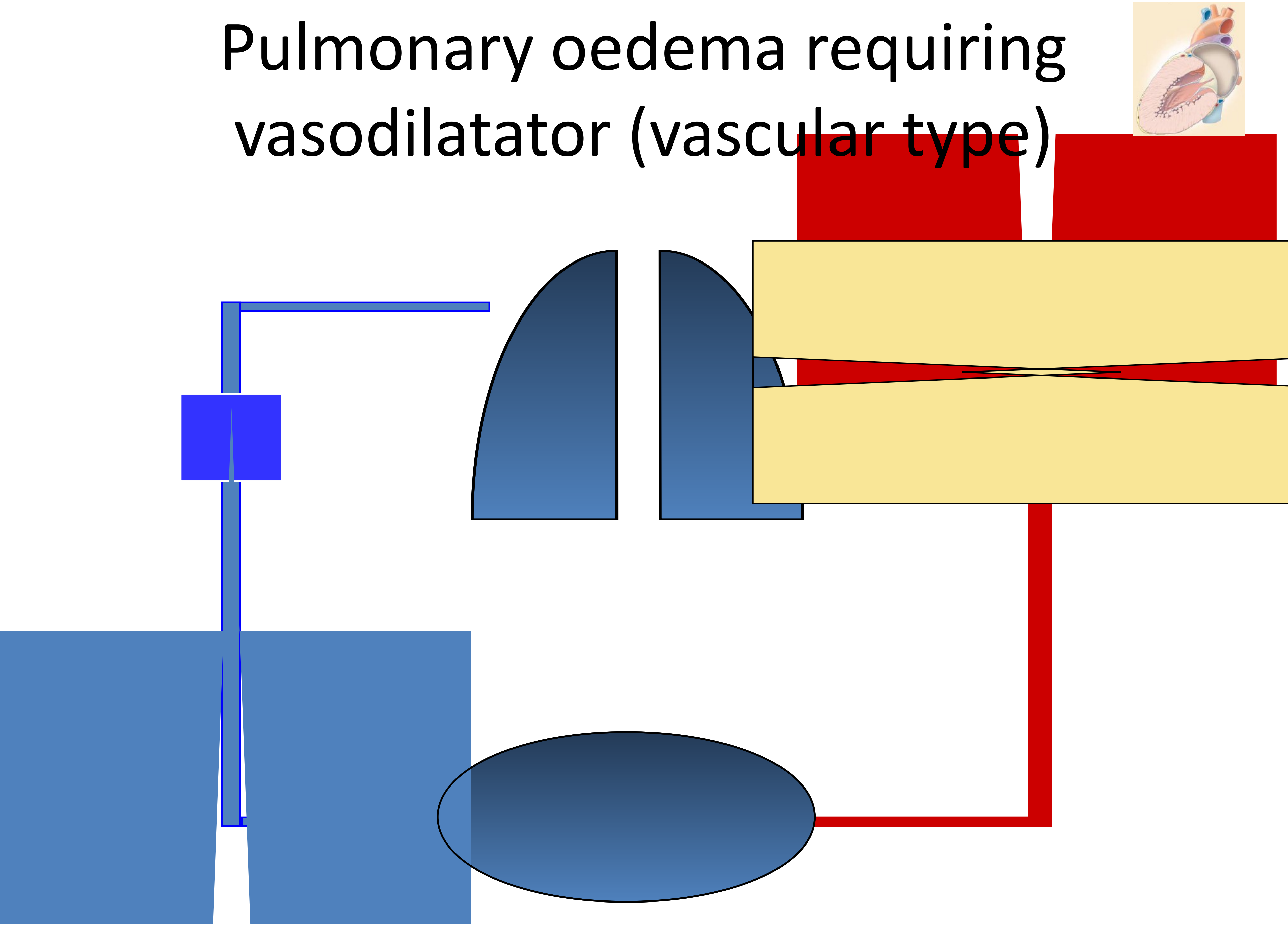
Suivi régulier avec évaluation TA, volémie, iono, urée créatinine (BNP ne sert pas); adaptation des diurétiques

pulmonary oedema requiring diuretic (cardiac type)





Pulmonary oedema requiring vasodilatator (vascular type)

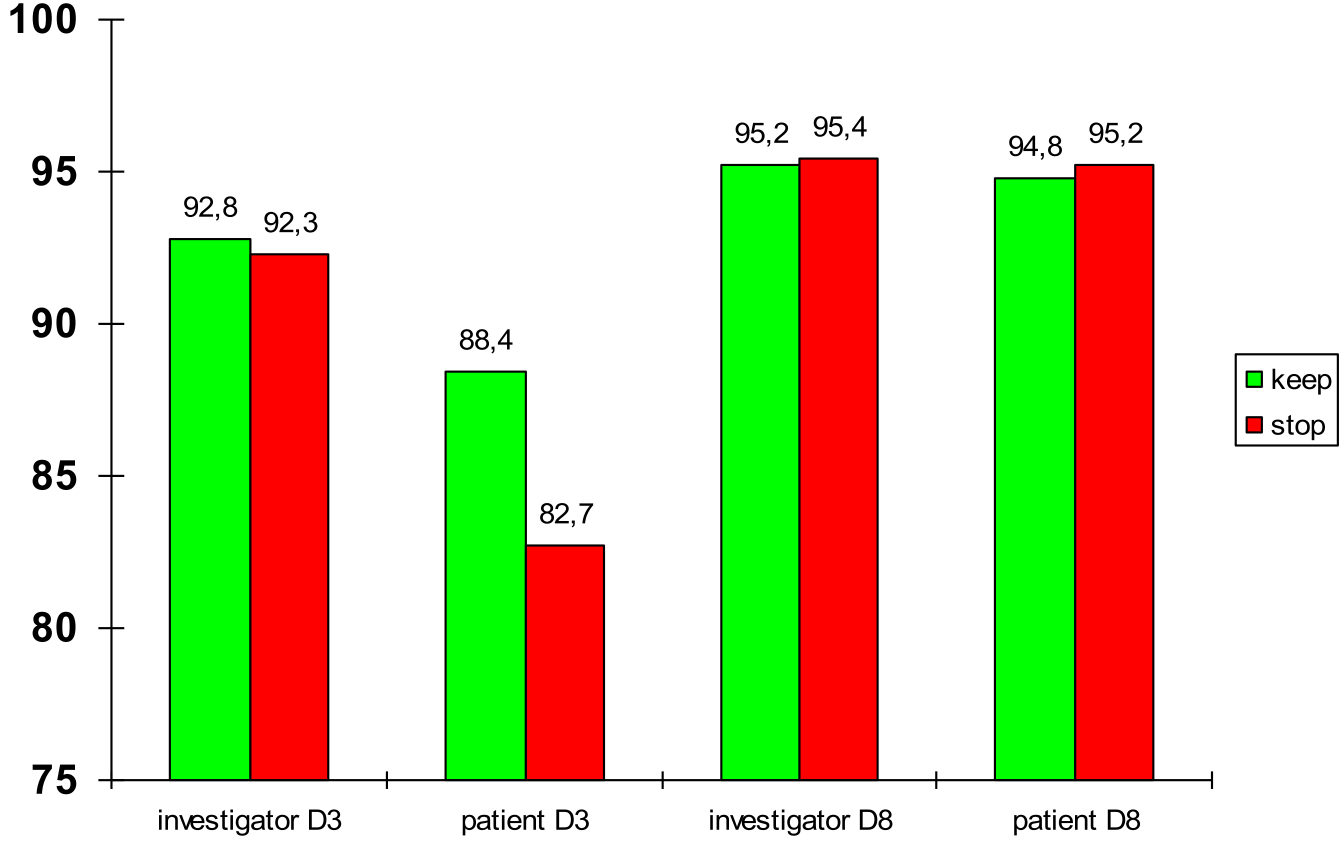


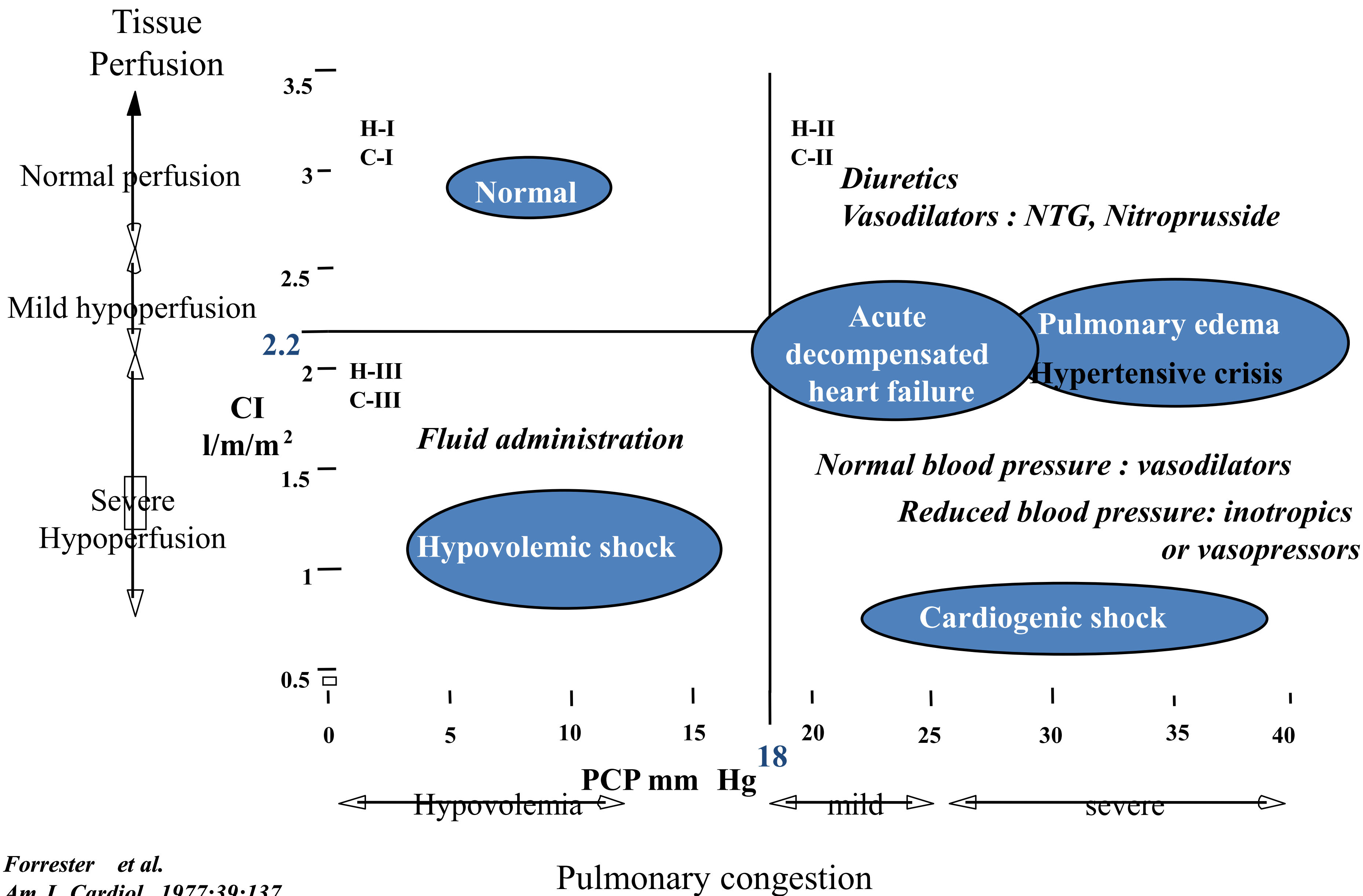
Causes d'élévation du BNP (ESC 2016)

Cardiac	Heart failure Acute coronary syndromes Pulmonary embolism Myocarditis Left ventricular hypertrophy Hypertrophic or restrictive cardiomyopathy Valvular heart disease Congenital heart disease Atrial and ventricular tachyarrhythmias Heart contusion Cardioversion, ICD shock Surgical procedures involving the heart Pulmonary hypertension
Non-cardiac	Advanced age Ischaemic stroke Subarachnoid haemorrhage Renal dysfunction Liver dysfunction (mainly liver cirrhosis with ascites) Paraneoplastic syndrome Chronic obstructive pulmonary disease Severe infections (including pneumonia and sepsis) Severe burns Anaemia Severe metabolic and hormone abnormalities (e.g. thyrotoxicosis, diabetic ketosis)

B- CONVINCED

% improvement for both dyspnea and general well being





Forrester et al.
Am J Cardiol 1977;39:137

ESC 2016

CONGESTION (-)

CONGESTION (+)
- Pulmonary congestion
- Orthopnoea/paroxysmal nocturnal dyspnoea
- Peripheral (bilateral) oedema
- Jugular venous dilatation
- Congested hepatomegaly
- Gut congestion, ascites
- Hepatojugular reflux

HYPOPERFUSION (-)

HYPOPERFUSION (+)
- Cold sweated extremities
- Oliguria
- Mental confusion
- Dizziness
- Narrow pulse pressure

Hypoperfusion <u>is not synonymous</u> with hypotension, but often hypoperfusion is accompanied by hypotension.

Mortalité intra-hospitalière – Données OFICA

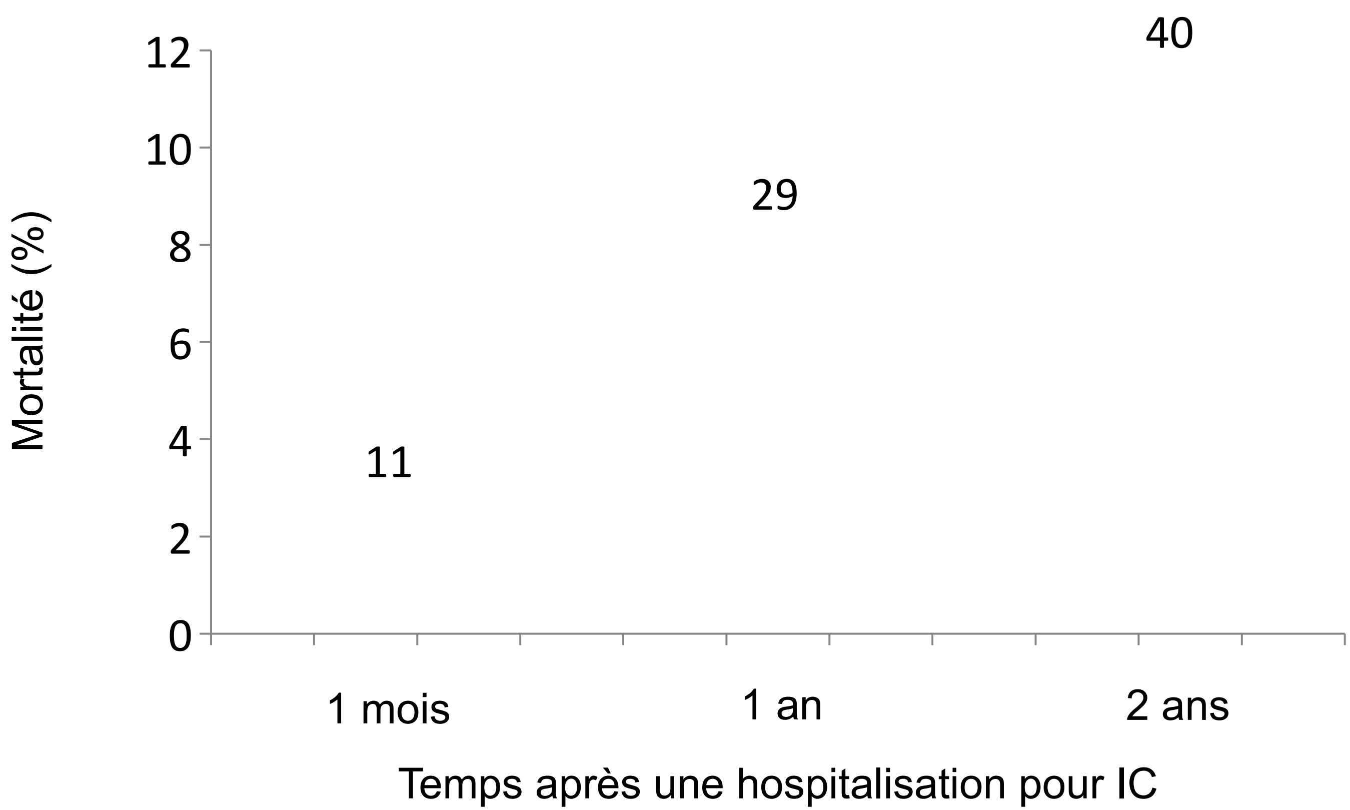
Mortalité intra-hospitalière	8,2%

Mortalité intra-hospitalière fonction de la FEVG		
FEVG altérée	8,3%	p=0,039
FEVG ≥ 50%	5,2%	

Logeart D *et al.* Current aspects of the spectrum of acute heart failure syndromes in a real-life setting: the OFICA study. Eur J Heart Fail. 2013;15:465-76.

Evolution de la mortalité après hospitalisation

<u>En France</u>



Tuppin *et al*. Arch Cardiovasc Dis. 2014;107:158-168.