AER
ACTUALITÉS EN RÉANIMATION

Evaluation de la fonction ventriculaire droite

Vendredi 18 Novembre 2022

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Echo–Doppler Demonstration of Acute Cor Pulmonale at the Bedside in the Medical Intensive Care Unit

Antoine Vieillard-Baron, Sebastien Prin, Karim Chergui, Olivier Dubourg, and François Jardin

Am J Respir Crit Care Med Vol 166. pp 1310–1319, 2002

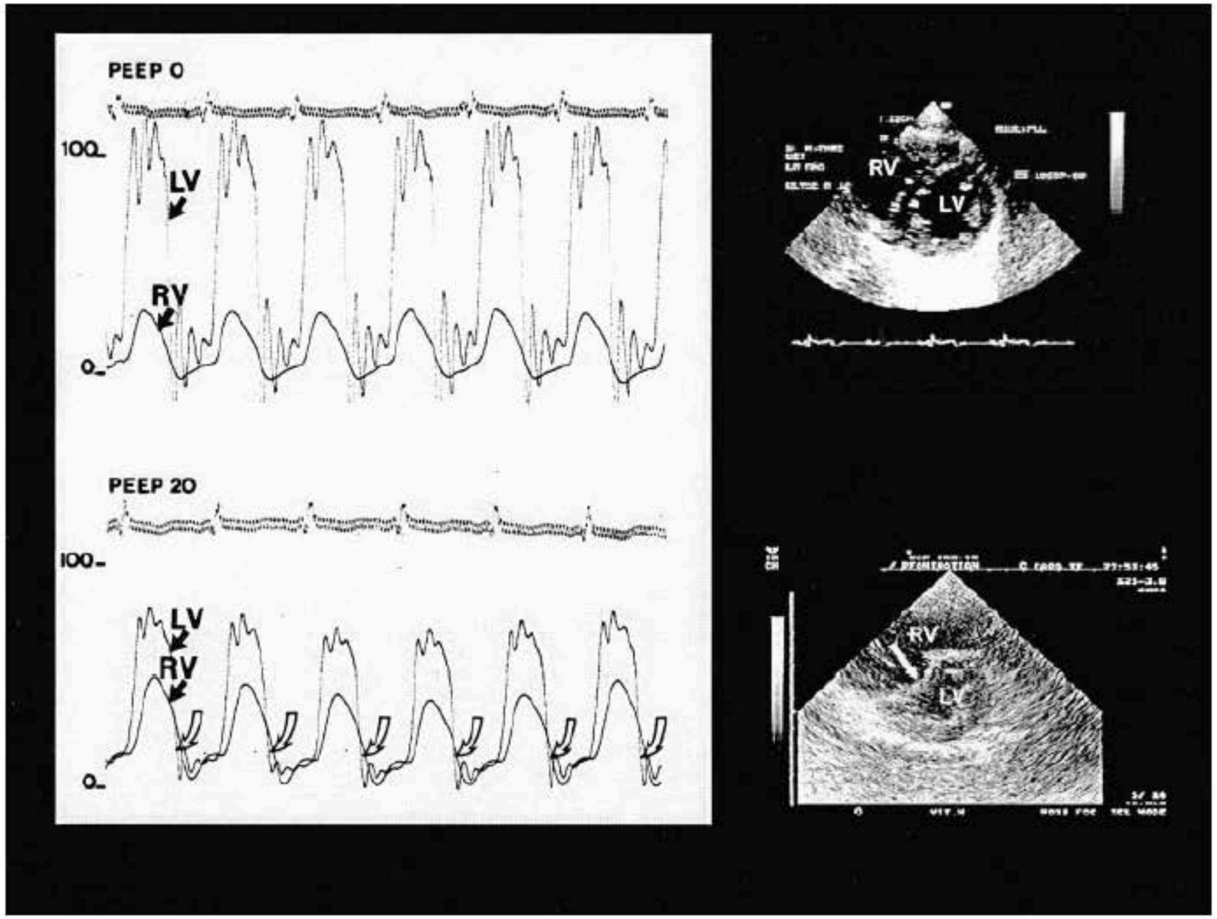
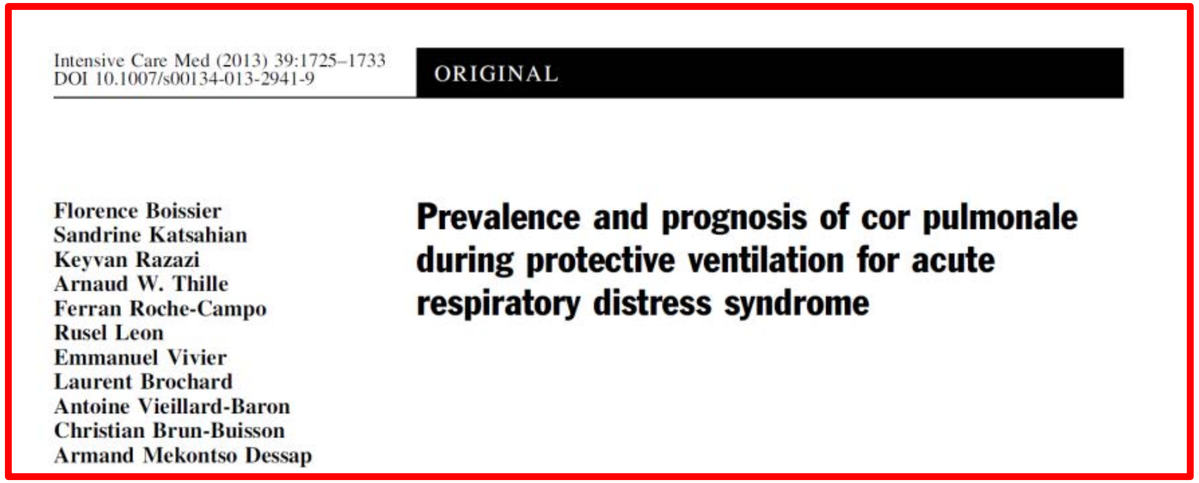


Figure 1. Effect of an acute increase in right ventricular impedance on the transseptal pressure gradient in a patient receiving mechanical ventilation for acute respiratory distress syndrome, and its consequences on septal position. *Left*: Left and right ventricular pressures were simultaneously recorded (LV = left ventricle; RV = right ventricle) at a PEEP of 0 cm H_2O (*top*) and at a PEEP of 20 cm H_2O (*bottom*). Note that right ventricular pressure becomes higher than left ventricular pressure (*open arrows*) at end systole/onset diastole during PEEP ventilation. *Right*: A simultaneous short-axis examination of the septal shape. Note septal flattening with PEEP (*white arrow*).



Intensive Care Med (2013) 39:1725–1733
DOI 10.1007/s00134-013-2941-9

ORIGINAL

Florence Boissier
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Antoine Vieillard-Baron
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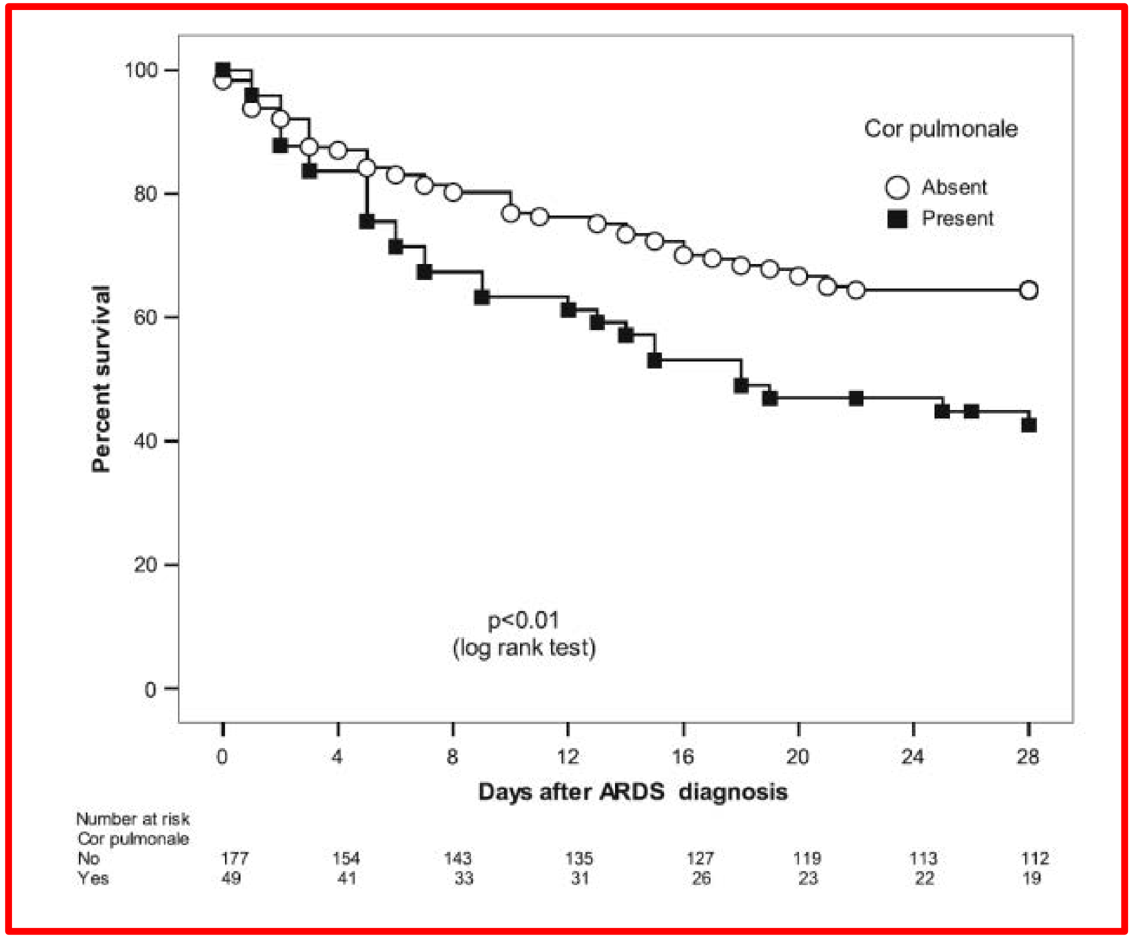
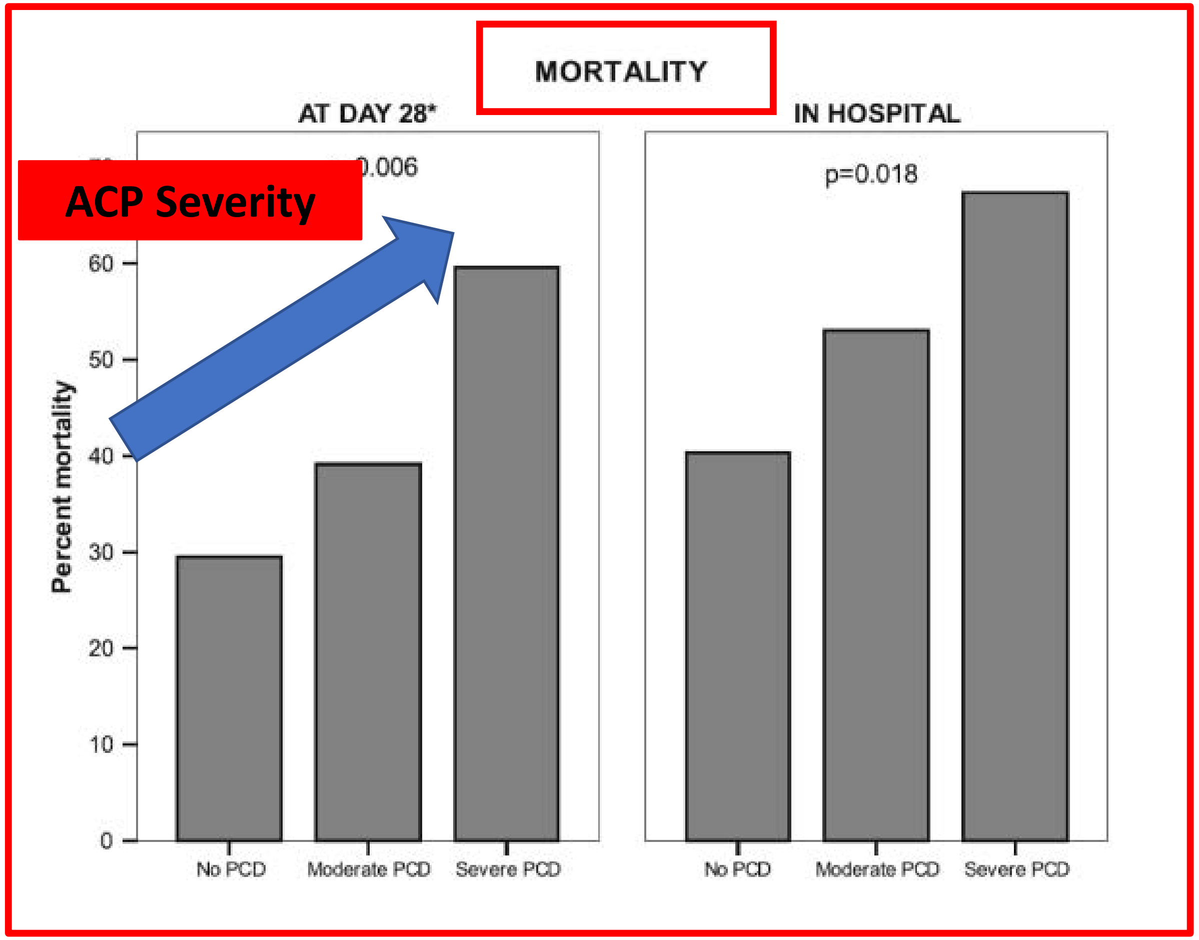
Prevalence and prognosis of cor pulmonale during protective ventilation for acute respiratory distress syndrome





MORTALITY

ACP Severity

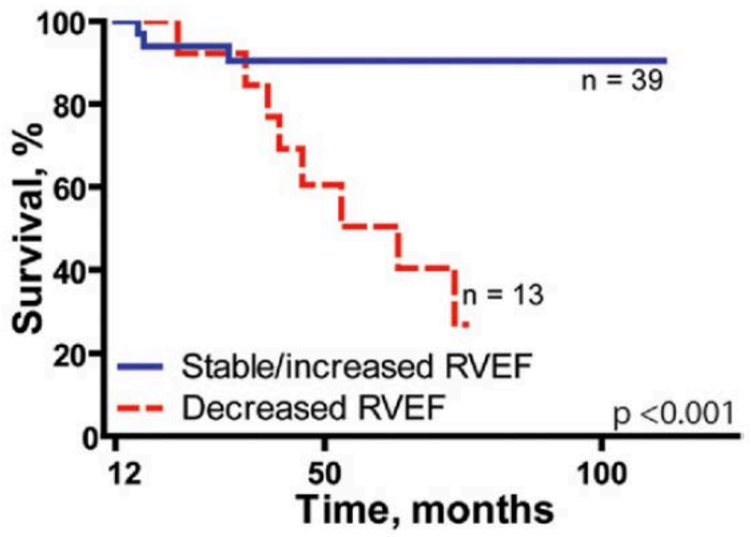


Figure 5 **Landmark Analysis**

Landmark analysis at month 12 of 52 patients with decreased PVR after therapy. Patients with stable/increased RVEF (n = 39) had better survival rates than patients with decreased RVEF (n = 13) ($p < 0.001$). Abbreviations as in Figure 2.

van de Veerdonk *et al.*
Progressive Right Heart Dysfunction After Therapy

JACC Vol. 58, No. 24, 2011
December 6, 2011:2511–9

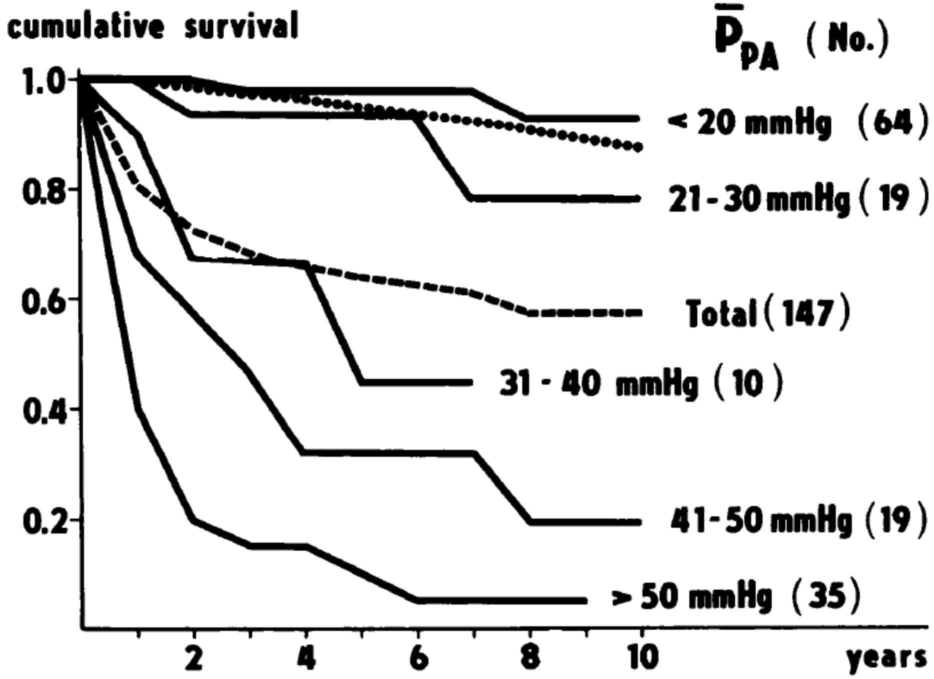
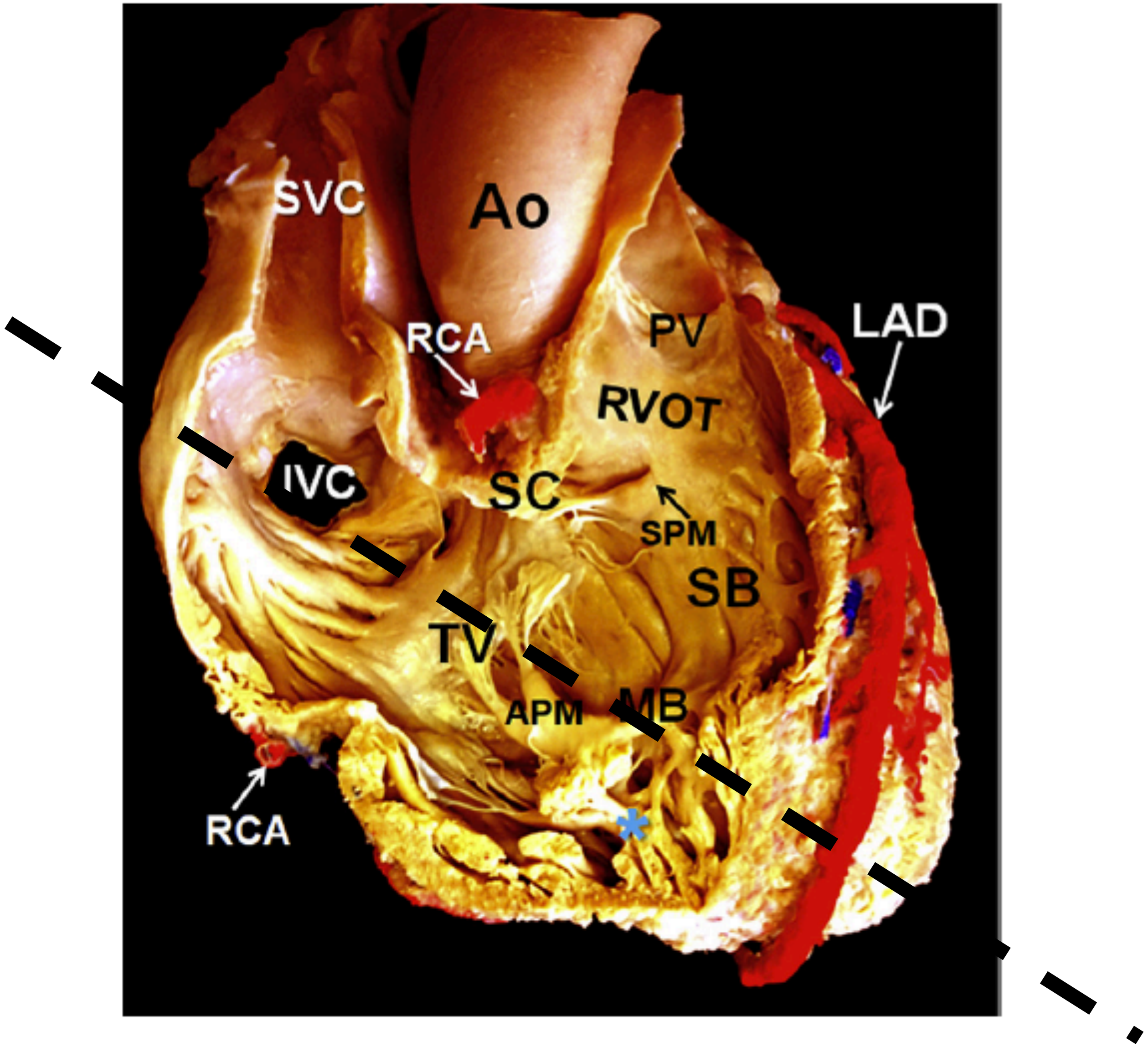


FIGURE 2. Cumulative survival curves according to the initial P_{PA}. Dotted line represents predicted survival among men 40-50 years old.

CHEST, 81: 2, FEBRUARY, 1982 LONGTERM FOLLOW-UP IN PULMONARY THROMBOEMBOLISM 155


SVC
Ao
RCA
PV
LAD
RVOT
IVC
SC
SPM
SB
TV
APM
MB
*
RCA

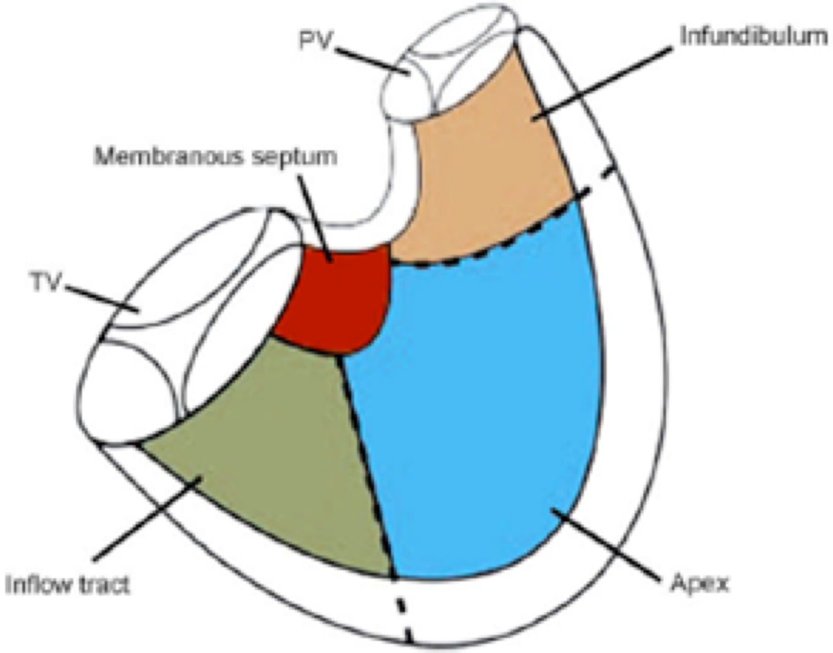
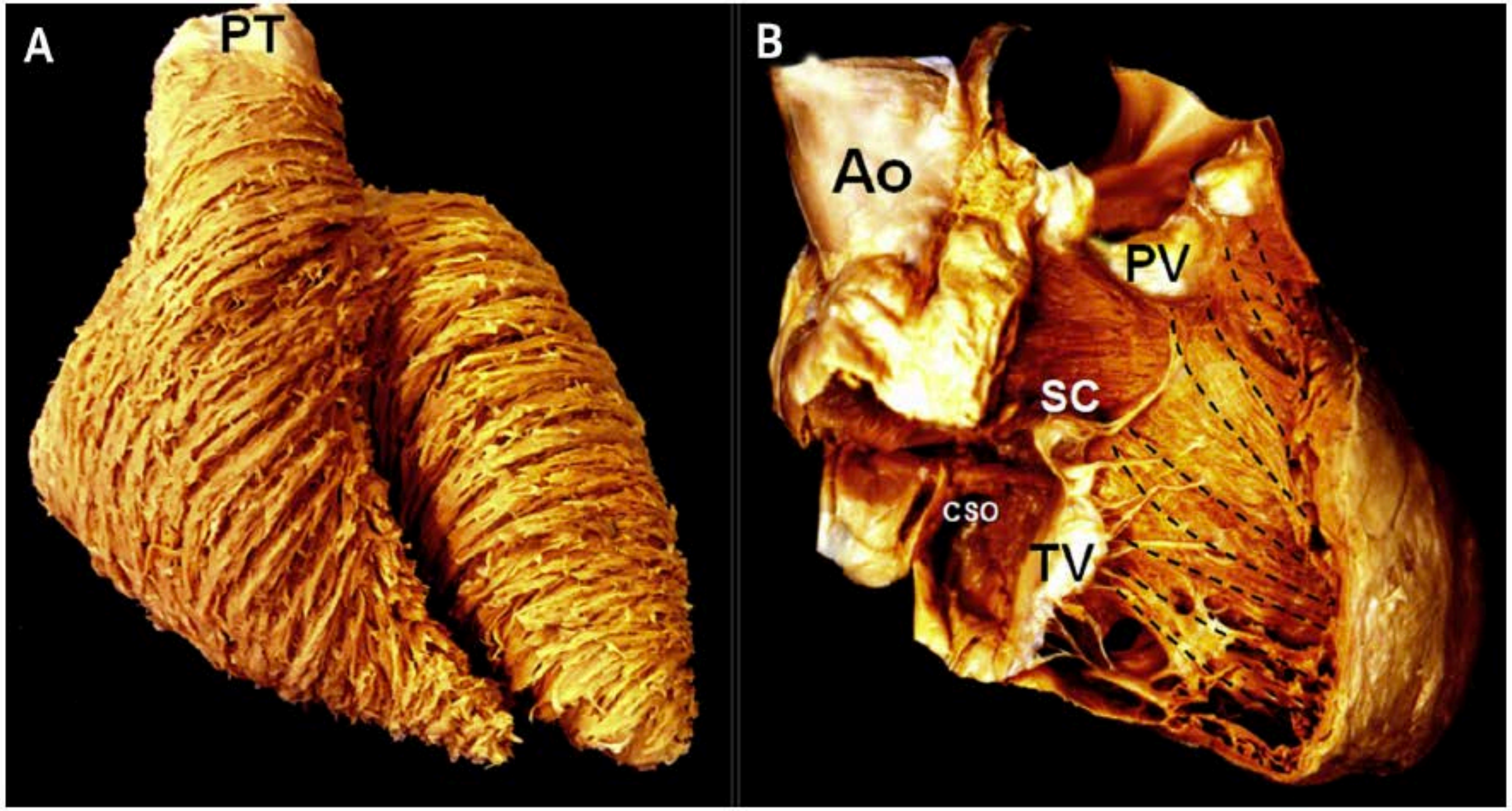
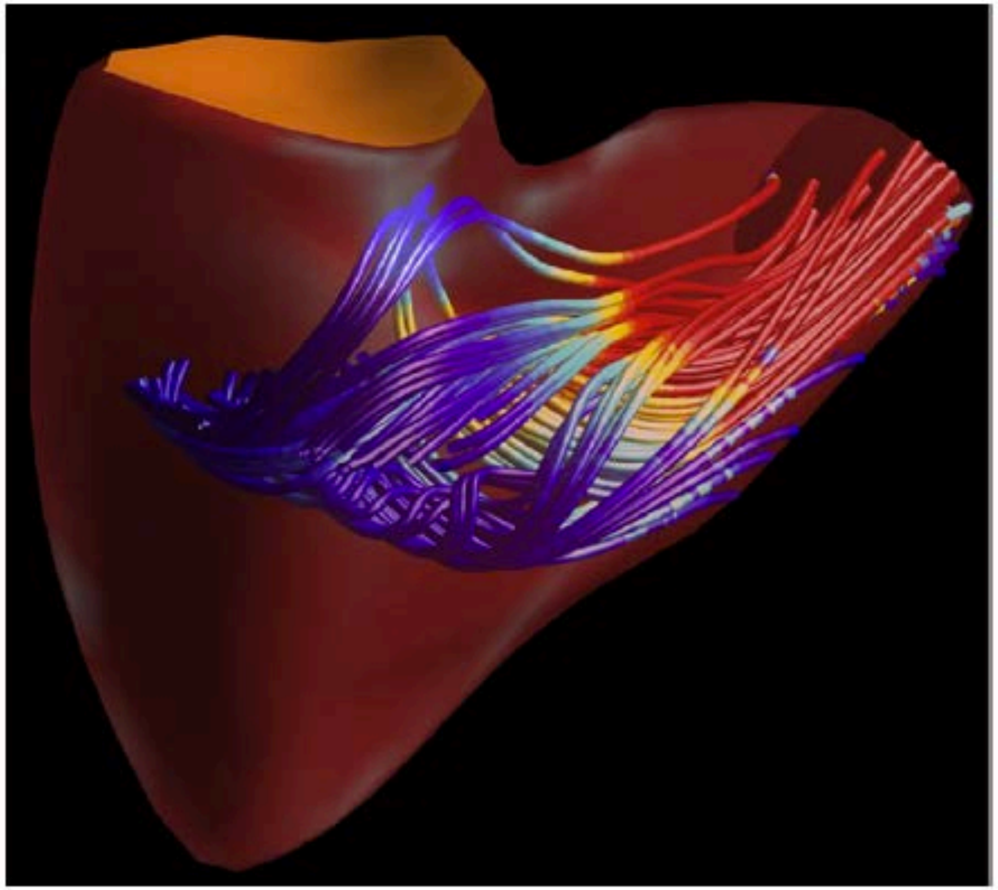

PV
Infundibulum
Membranous septum
TV
Inflow tract
Apex

FIGURE 2 RV Myoarchitecture

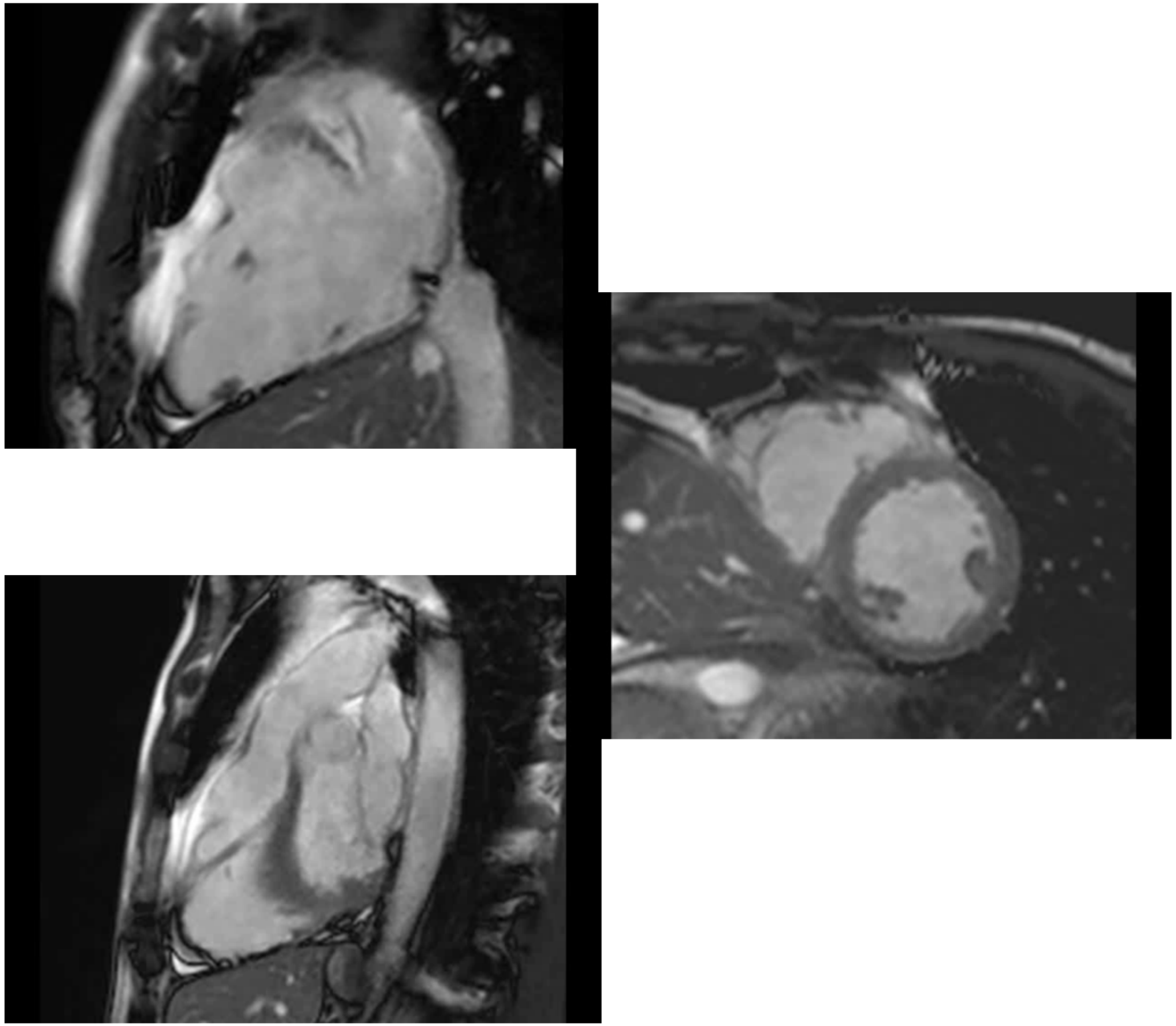


(A) Normal heart viewed from the front shows the circumferential arrangement of superficial RV and middle LV layers of aggregated cardiomyocytes. **(B)** Opened normal RV showing longitudinally arranged subendocardial myocyte aggregates **(black dashed lines).** CSO = coronary sinus ostium; LV = left ventricle; PT = pulmonary trunk; other abbreviations as in Figure 1.

FIGURE 3 3-Dimensional Representation of Helical Streamlines of Normal RV Intracavitary Flow During Systole



Reprinted with permission from Sengupta and Narula (28).



Grande capacitance, grande compliance, préservation du débit

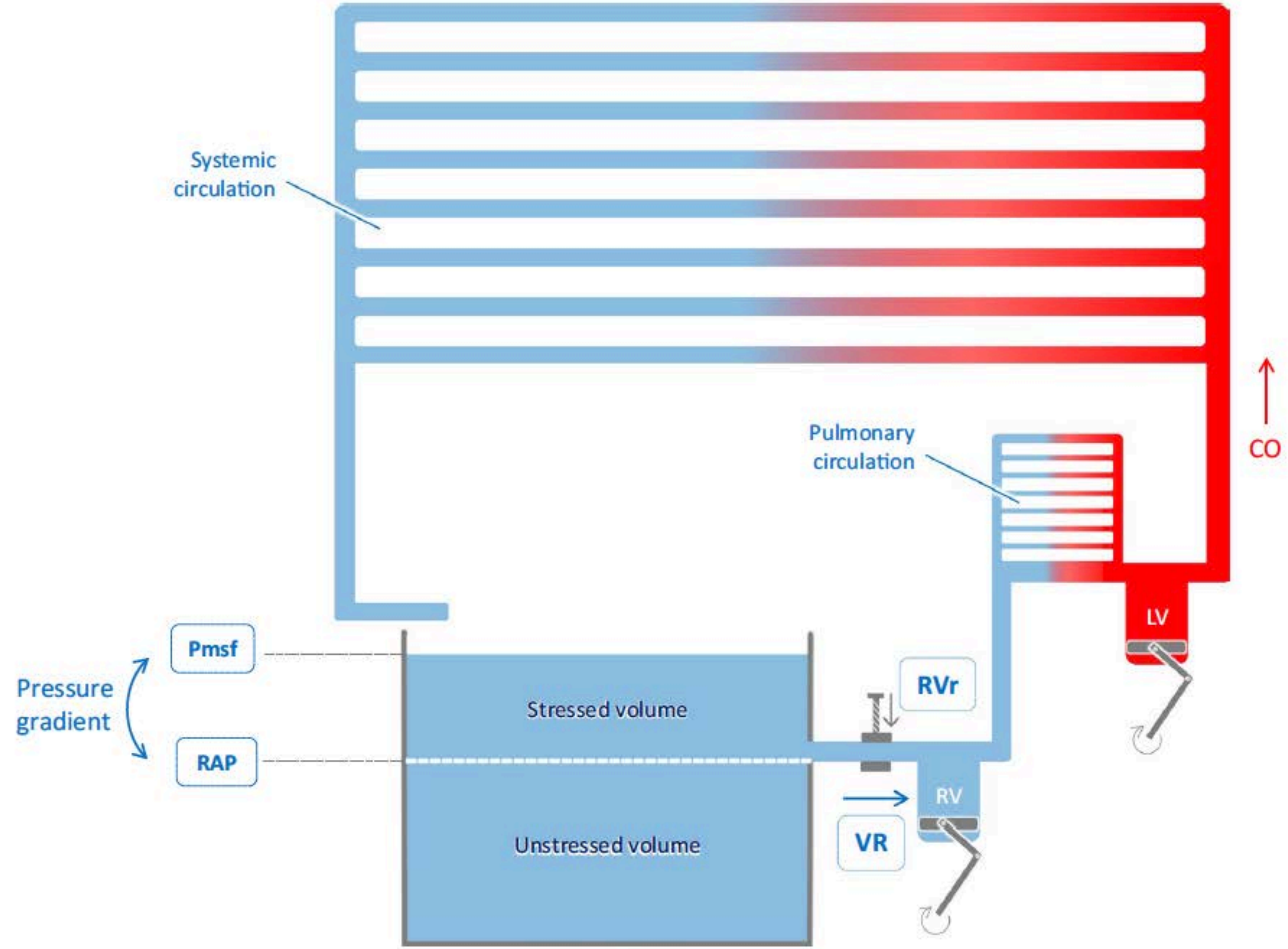

Systemic circulation
Pulmonary circulation
CO
LV
Pmsf
Pressure gradient
RAP
Stressed volume
Unstressed volume
RVr
RV
VR

Fig. 1 The three determinants of venous return. At equilibrium, cardiac output (CO) and venous return (VR) are similar. Venous return is the inflow of the right heart. Its three determinants are the mean systemic filling pressure (Pmsf), the right atrial pressure (RAP) and the resistance to venous return (RVr) according to the formula: venous return = (Pmsf–RAP)/RVr. LV: left ventricle, RV: right ventricle

Persichini Crit care 2022

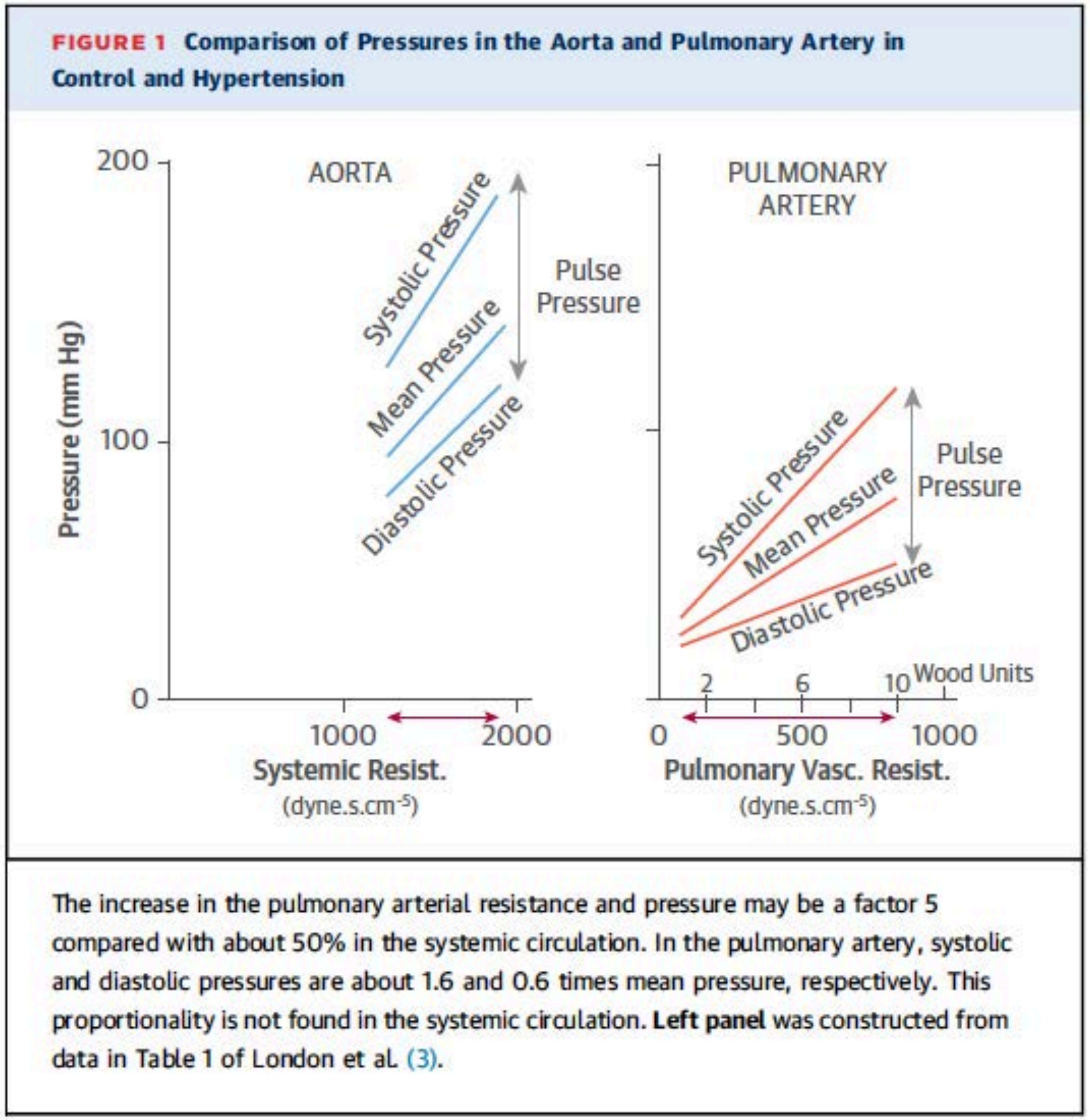
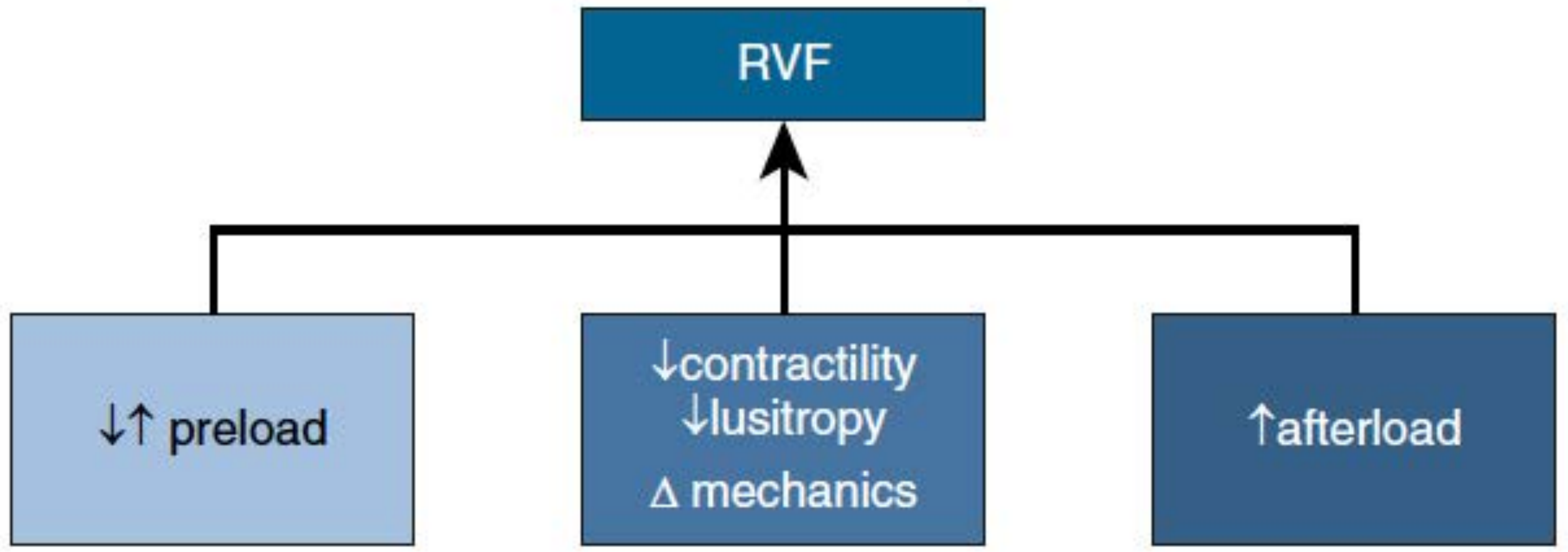


FIGURE 1 **Comparison of Pressures in the Aorta and Pulmonary Artery in Control and Hypertension**

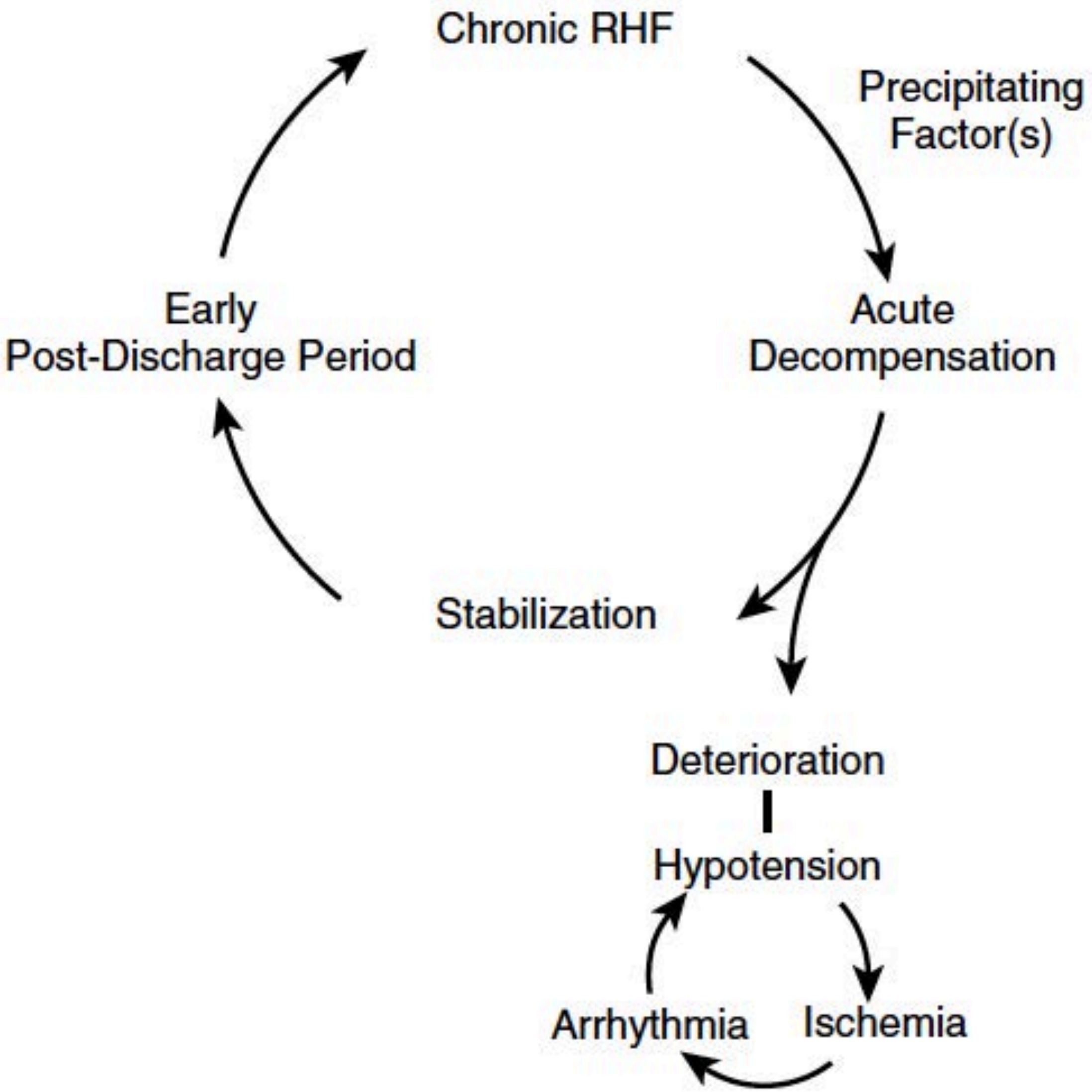
The increase in the pulmonary arterial resistance and pressure may be a factor 5 compared with about 50% in the systemic circulation. In the pulmonary artery, systolic and diastolic pressures are about 1.6 and 0.6 times mean pressure, respectively. This proportionality is not found in the systemic circulation. **Left panel** was constructed from data in Table 1 of London et al. (3).

Noordegraaf JACC 2017

The task force defined **RVF as a complex clinical syndrome characterized by insufficient delivery of blood from the RV and elevated systemic venous pressure at rest or exercise.**


RVF
↓↑ preload
↓contractility
↓lusitropy
Δ mechanics
↑afterload

Lahm AJRCCM 2018



Chronic RHF

Precipitating Factor(s)

Acute Decompensation

Stabilization

Early Post-Discharge Period

Deterioration

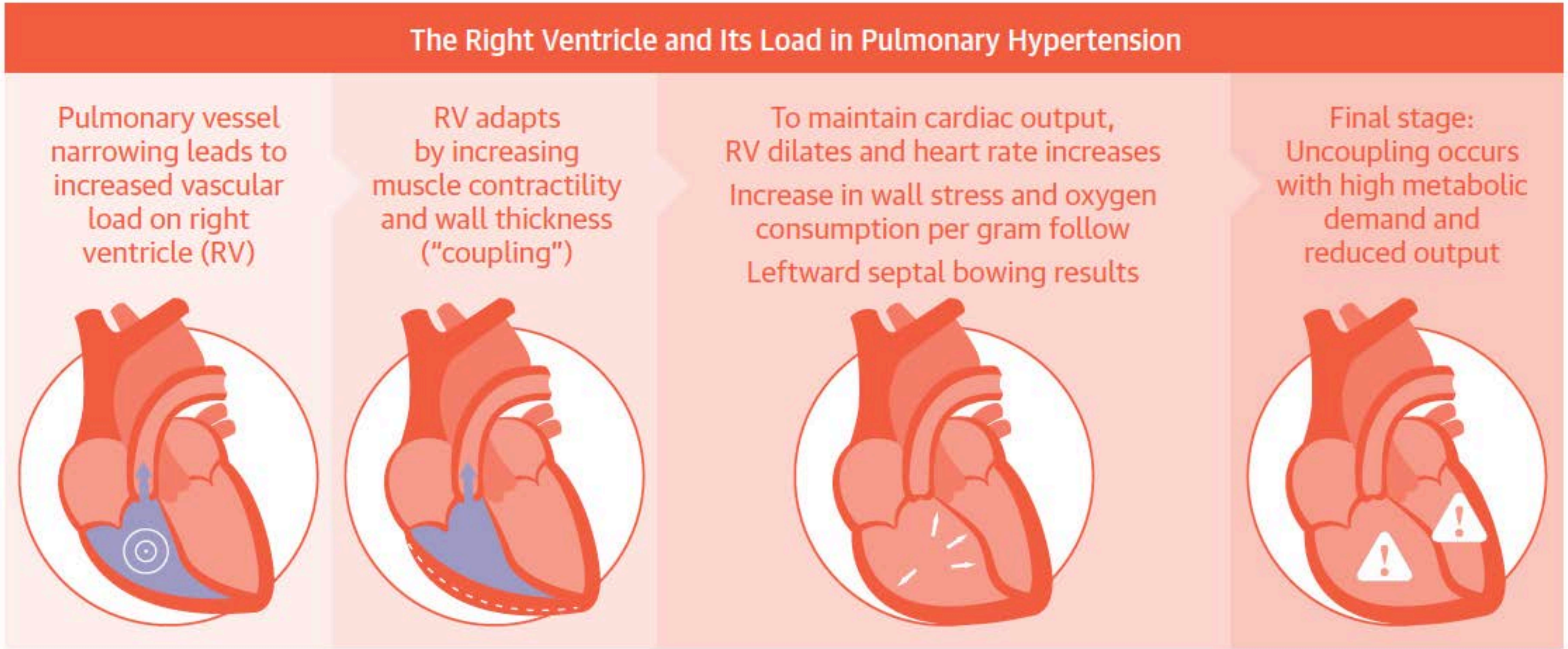
Hypotension

Ischemia

Arrhythmia

Lahm AJRCCM 2018

CENTRAL ILLUSTRATION Sequence of Events



The Right Ventricle and Its Load in Pulmonary Hypertension

Pulmonary vessel narrowing leads to increased vascular load on right ventricle (RV)

RV adapts by increasing muscle contractility and wall thickness ("coupling")

To maintain cardiac output, RV dilates and heart rate increases

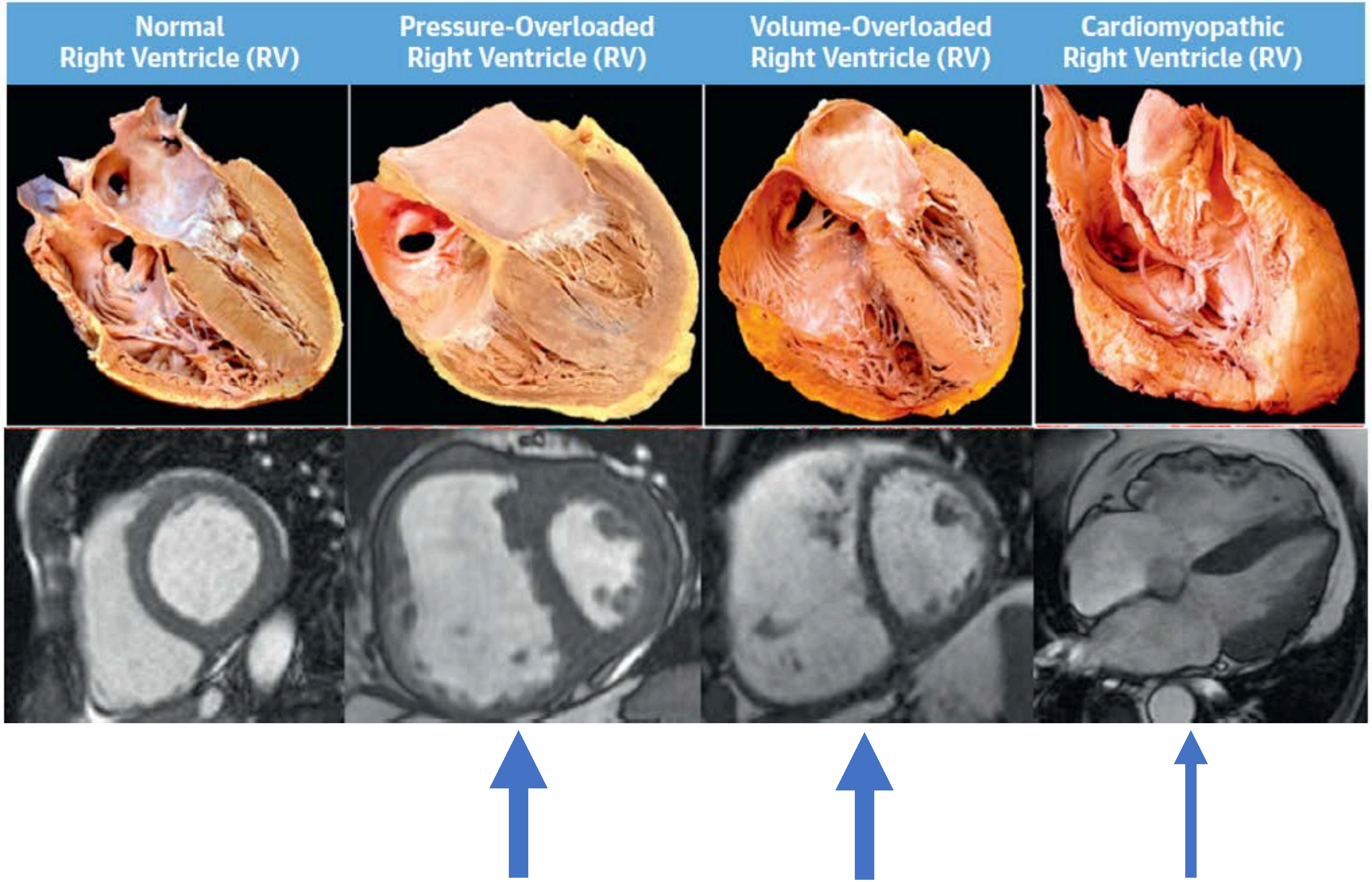
Increase in wall stress and oxygen consumption per gram follow

Leftward septal bowing results

Final stage: Uncoupling occurs with high metabolic demand and reduced output

Vonk Noordegraaf, A. et al. J Am Coll Cardiol. 2017;69(2):236–43.

The consequence of pulmonary hypertension for the right ventricle. Initially, coupling of the ventricle to the high arterial load is maintained by increasing contractility, subsequently followed by dilation and finally uncoupling. RV = right ventricle.



Normal Right Ventricle (RV)

Pressure-Overloaded Right Ventricle (RV)

Volume-Overloaded Right Ventricle (RV)

Cardiomyopathic Right Ventricle (RV)

ICU ++

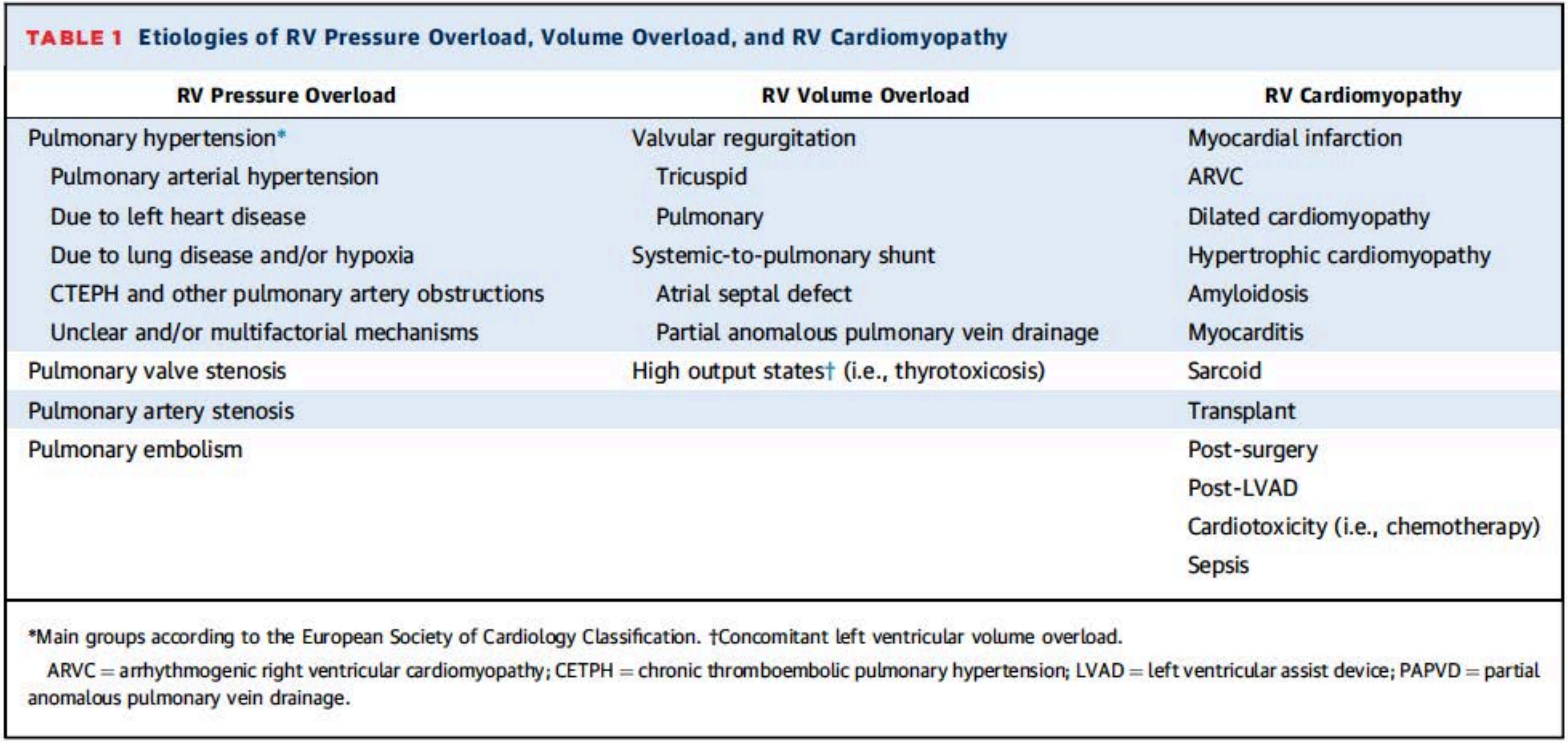


TABLE 1 Etiologies of RV Pressure Overload, Volume Overload, and RV Cardiomyopathy

RV Pressure Overload	RV Volume Overload	RV Cardiomyopathy
Pulmonary hypertension*	Valvular regurgitation	Myocardial infarction
Pulmonary arterial hypertension	Tricuspid	ARVC
Due to left heart disease	Pulmonary	Dilated cardiomyopathy
Due to lung disease and/or hypoxia	Systemic-to-pulmonary shunt	Hypertrophic cardiomyopathy
CTEPH and other pulmonary artery obstructions	Atrial septal defect	Amyloidosis
Unclear and/or multifactorial mechanisms	Partial anomalous pulmonary vein drainage	Myocarditis
Pulmonary valve stenosis	High output states† (i.e., thyrotoxicosis)	Sarcoid
Pulmonary artery stenosis		Transplant
Pulmonary embolism		Post-surgery
		Post-LVAD
		Cardiotoxicity (i.e., chemotherapy)
		Sepsis

*Main groups according to the European Society of Cardiology Classification. †Concomitant left ventricular volume overload.

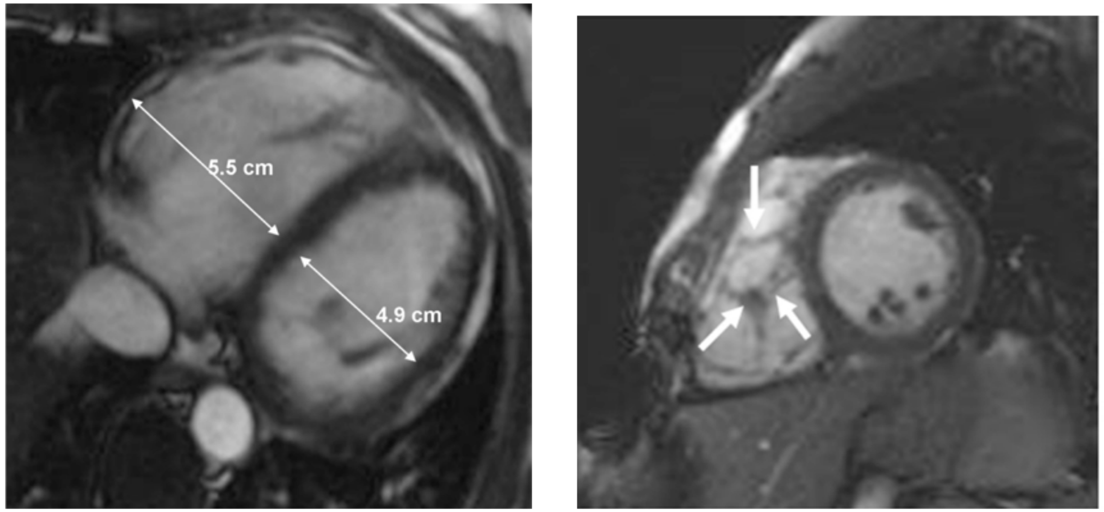
ARVC = arrhythmogenic right ventricular cardiomyopathy; CETPH = chronic thromboembolic pulmonary hypertension; LVAD = left ventricular assist device; PAPVD = partial anomalous pulmonary vein drainage.

Méthodes d’évaluation classiques mais pas très pratiques du VD

SIEMENS Healthineers
MAGNETOM Sola

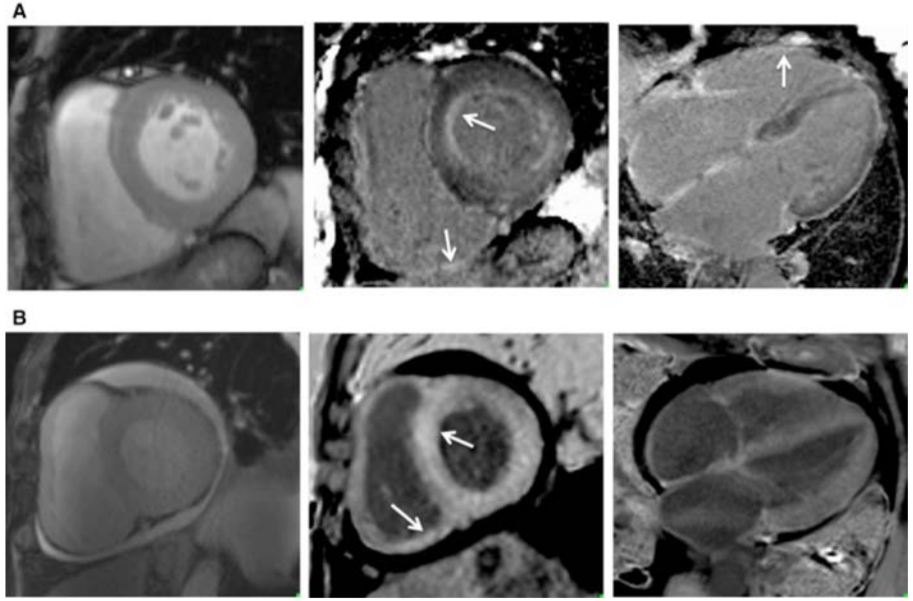
IRM cardiaque

Dysplasie arythmogène du VD

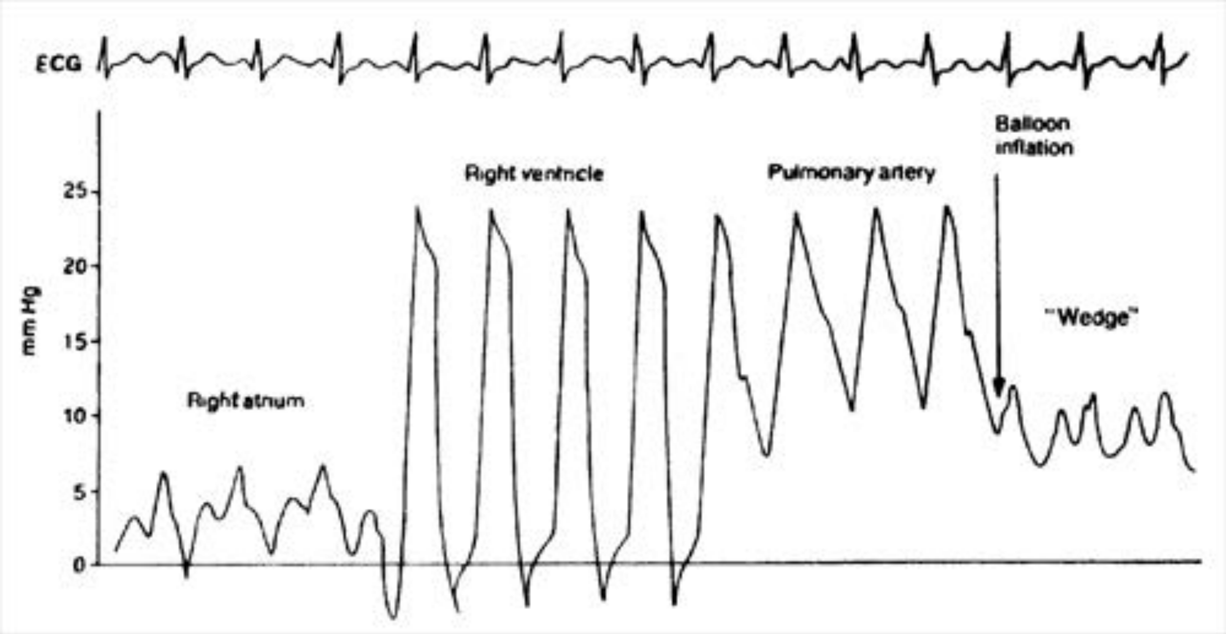


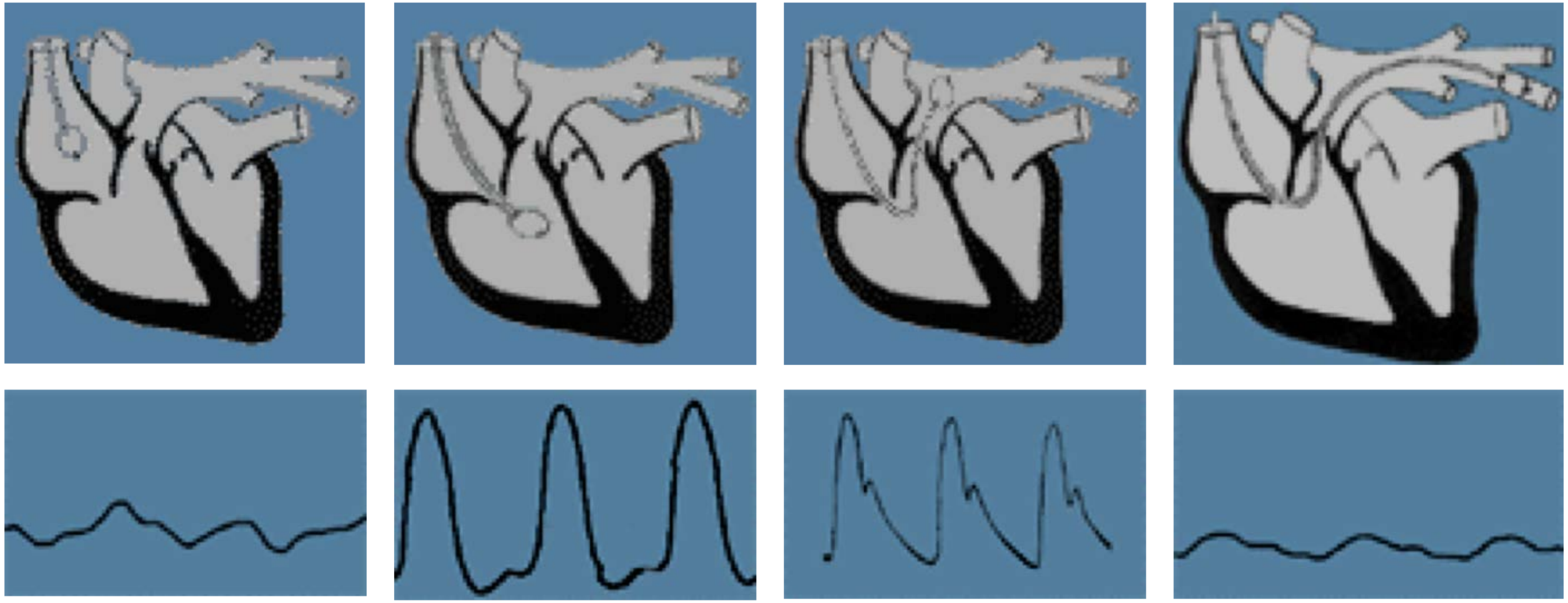
Jain, J Cardvasc Magn Reson 2008

Amylose cardiaque



Wan Heart Vessels 2018


ECG
mm Hg
25
20
15
10
5
0
Right atrium
Right ventricle
Pulmonary artery
Balloon inflation
"Wedge"



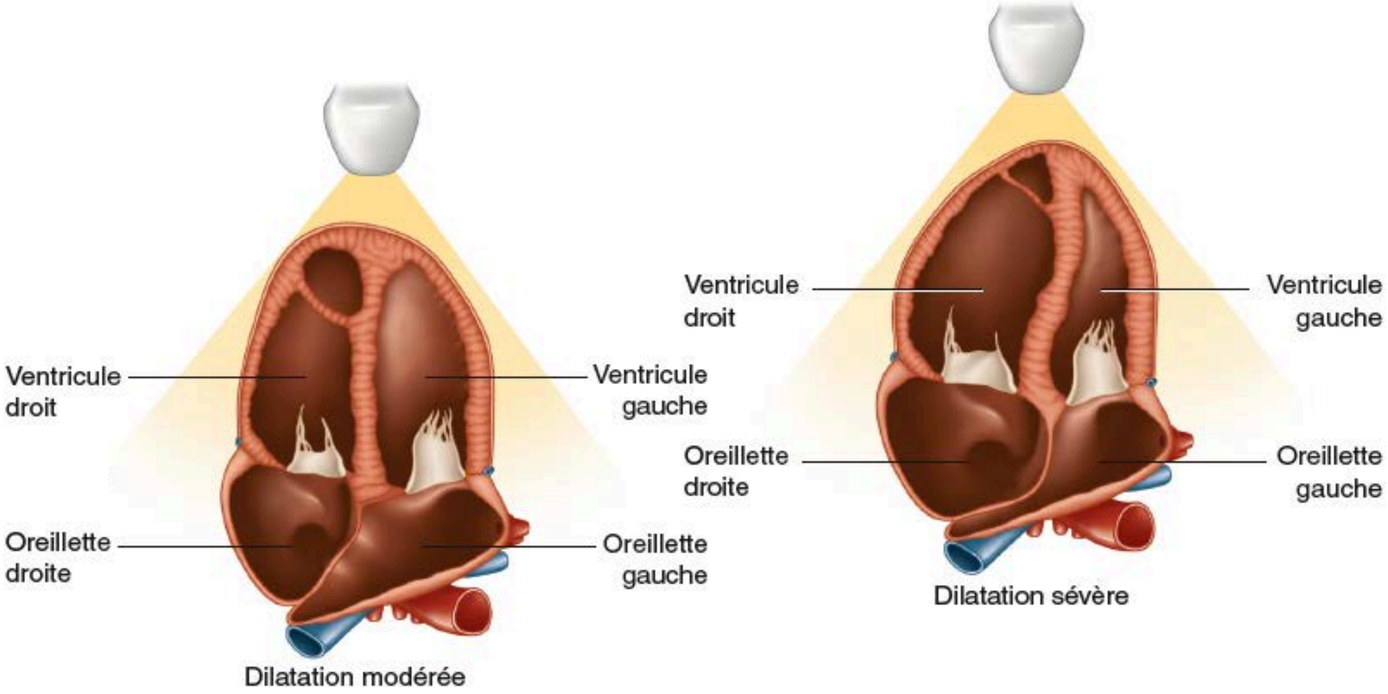
PVC : Pression Veineuse Centrale	3 à 8 cm H2O
POD : Pression Oreillette Droite	0 à 8 mmHg
PVD : Pression Ventricule Droit	systolique : 15 à 30 mmHg télédiastolique : 0 à 8 mmHg
PAP : Pression Artère Pulmonaire	systolique : 15 à 30 mmHg diastolique : 3 à 12 mmHg moyenne :9 à 16 mmHg
Pcap : Pression capillaire	1 à 10 mmHg
DC : Débit Cardiaque	5 à 6 l / mn
IC : Index Cardiaque	DC / surf. corp. (l/min/m^2) = 2,5 à 3,5
PAM : Pression Artérielle Moyenne	PAD + ((PAS - PAD) / 3)
IS : Index systolique	IC / Fc (ml / bat / m^2) = 40 à 50
RVSI : Résistance vasculaire systémique	(PAM - POD) × 80 / IC = 2000 à 2400
RVPI : Résistance pulmonaire	(PAP - Pcap) / DC = 48 à 88
IWSVG : travail du VG	(PAM - Pcap) x IS x 0,0136 = 35 à 85
IWSVD : travail du VD	
CaO2 : contenu artériel en oxygène	(SaO2 x Hb x 1,34) + (PaO2 x 0,003)
DAV : différence artério-veineuse	CaO2 - CvO2 = 4 +/- 0,5 ml / 100 ml

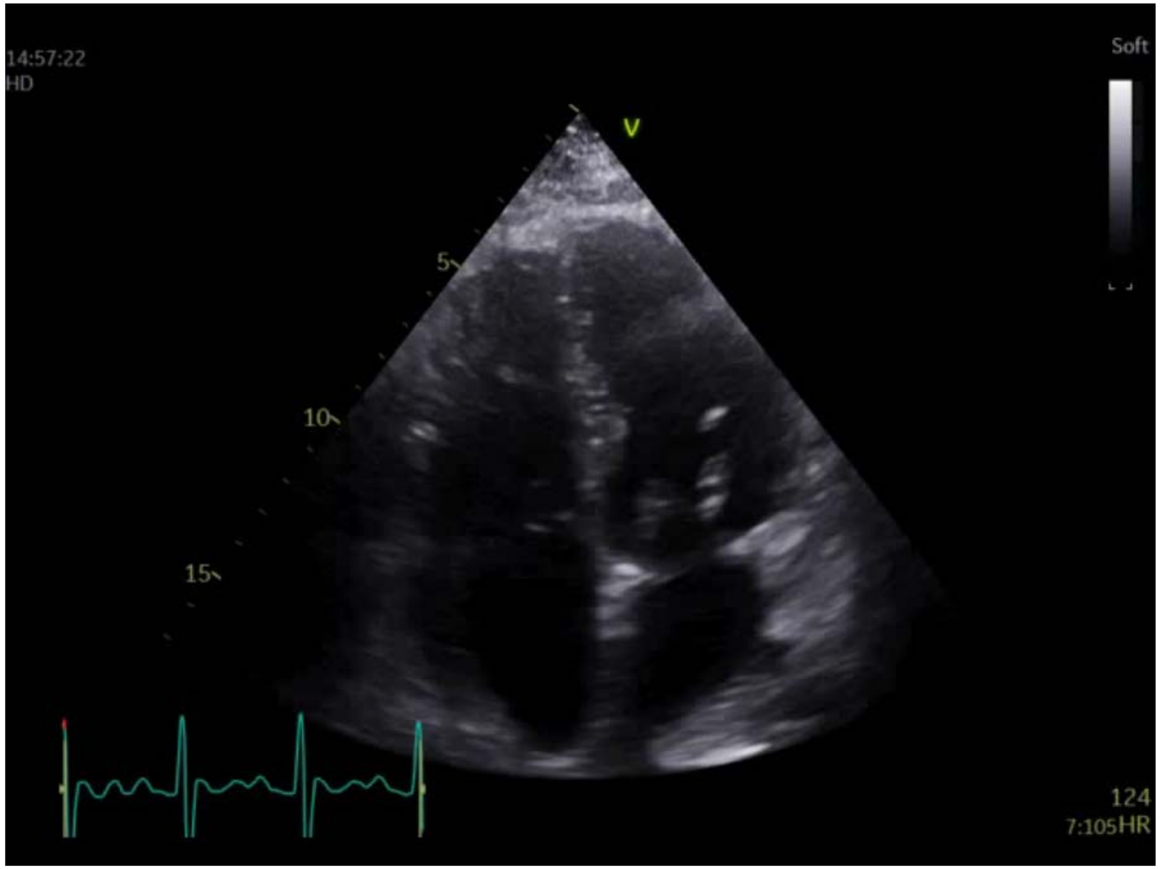
Méthodes d’évaluation classiques et simples d’utilisation du VD

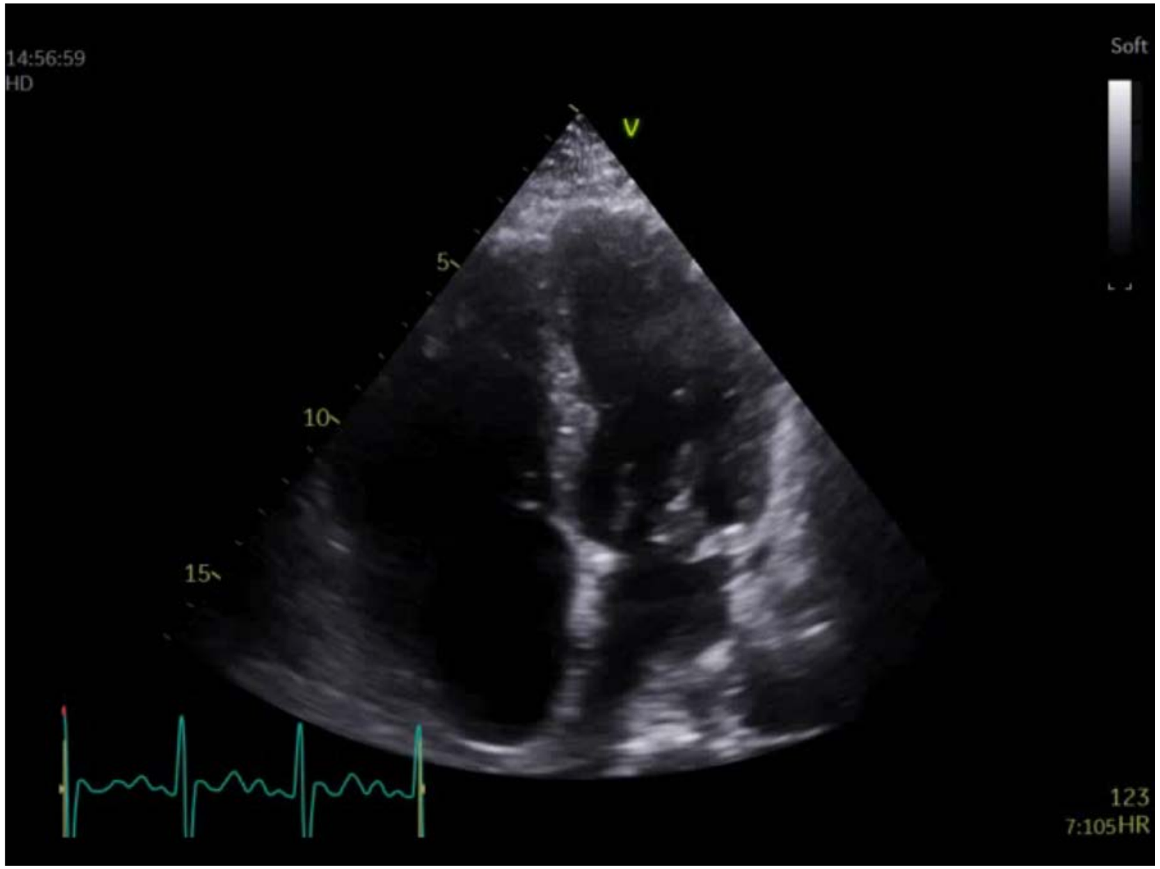
Analyse de la taille du VD

Rapport des surfaces ou des diamètres:

VD/VG < 0.6


Ventricule droit
Ventricule gauche
Oreillette droite
Oreillette gauche
Dilatation modérée
Ventricule droit
Ventricule gauche
Oreillette droite
Oreillette gauche
Dilatation sévère


14:57:22
HD
Soft
V
5
10
15
124
7:105HR


14:56:59
HD
Soft
V
5
10
15
123
7:105HR

Obtenir la bonne incidence

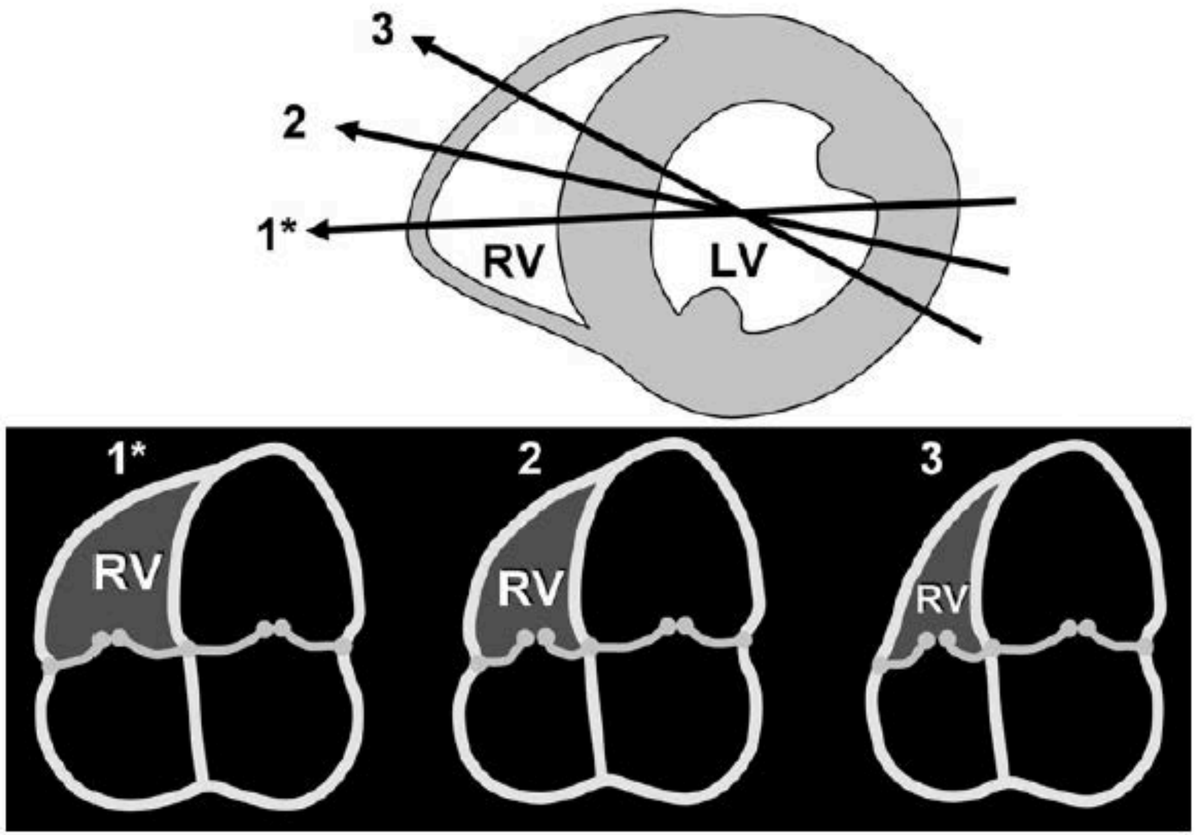
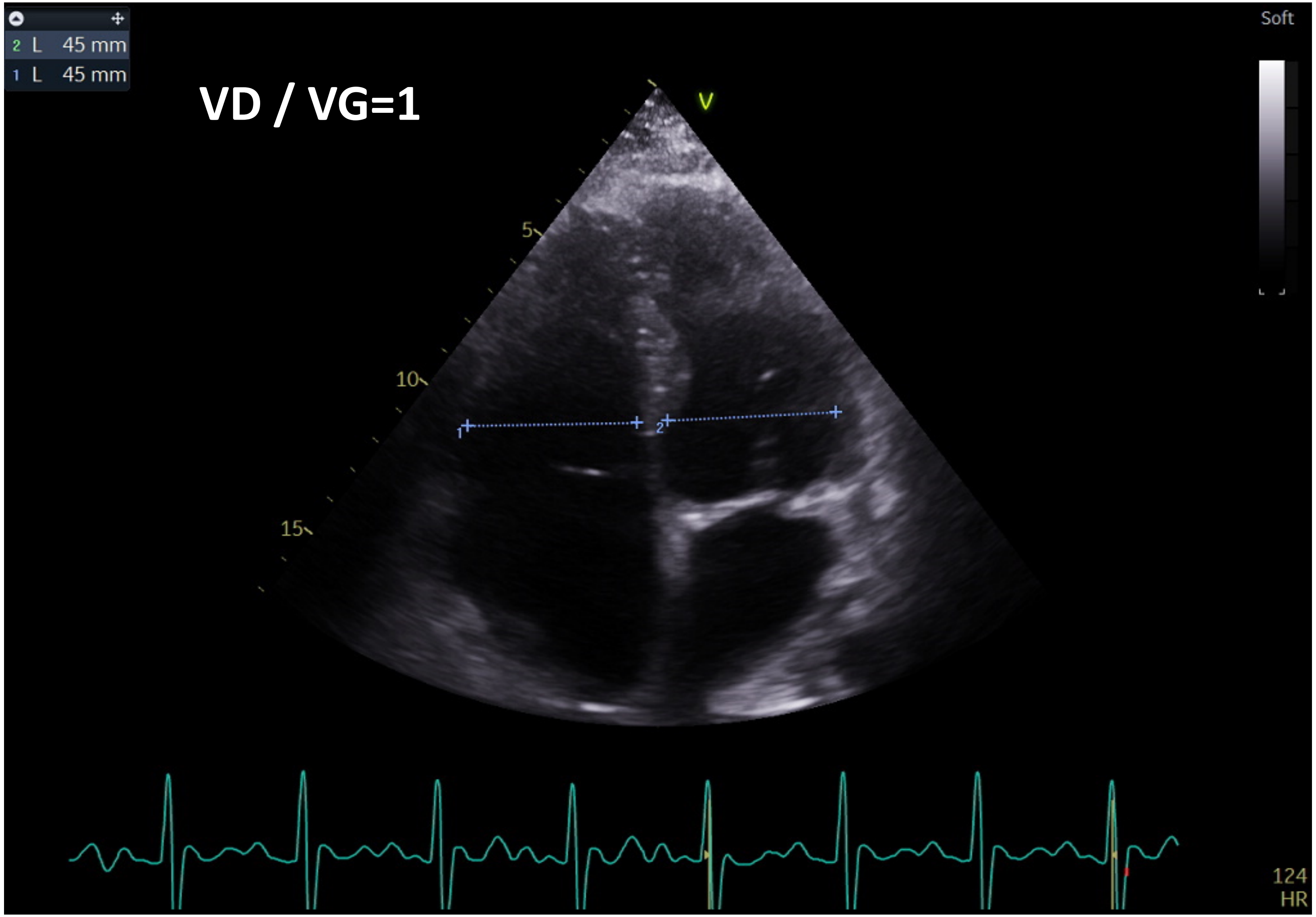


Figure 6 Diagram showing the recommended apical 4-chamber (A4C) view with focus on the right ventricle (RV) (1*) and the sensitivity of right ventricular size with angular change (2,3) despite similar size and appearance of the left ventricle (LV). The lines of intersection of the A4C planes (1*,2,3) with a mid left ventricular short-axis are shown above and corresponding A4C views below.

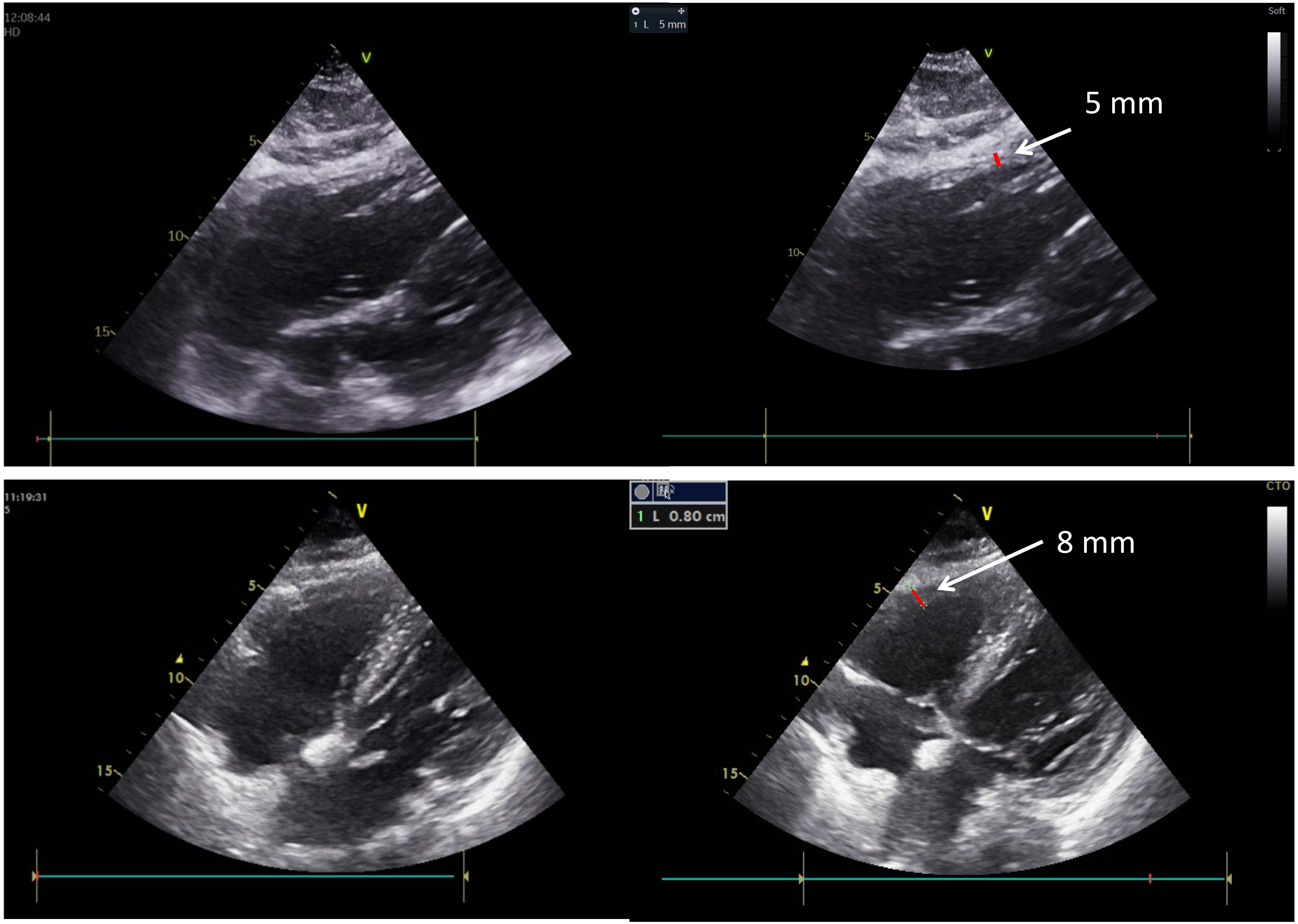
Rudski JASE 2010

VD / VG=1

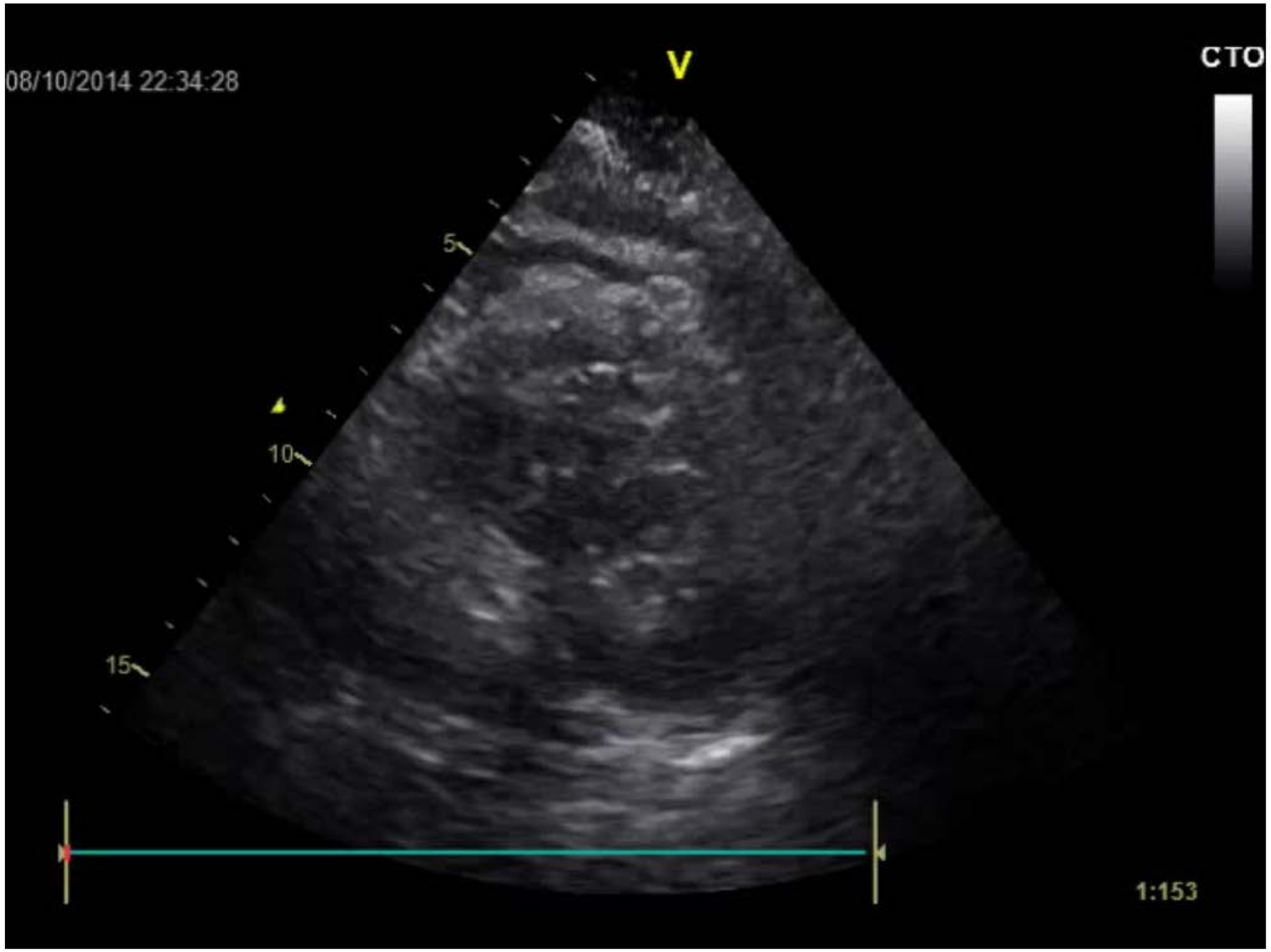

2 L 45 mm
1 L 45 mm
Soft
V
5
10
15
1
2
124
HR

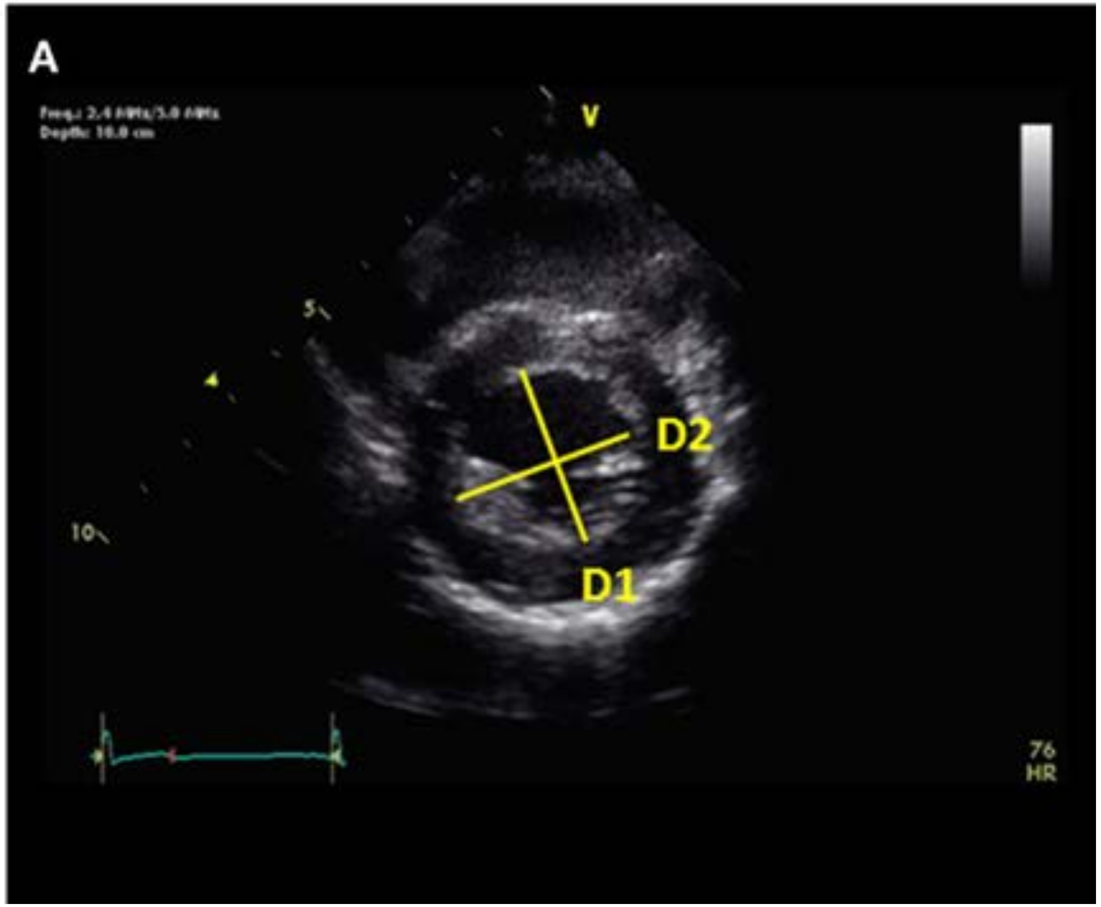
CPA ou CPC ?

Epaisseur normale de la paroi libre du VD: < 6 mm


12:08:44
HD
V
5
10
15
1 L 5 mm
Soft
5 mm
11:19:31
1 L 0.80 cm
CTO
8 mm

Analyse de la taille du VD: conséquence sur VG


08/10/2014 22:34:28
V
CTO
5
10
15
1:153


A
V
5
10
D2
D1
76
HR

D2/D1 compris entre 0.9 et 1.1

An Echocardiographic Index for Separation of Right Ventricular Volume and Pressure Overload

THOMAS RYAN, MD, OLIVERA PETROVIC, MD, JAMES C. DILLON, MD, FACC,
HARVEY FEIGENBAUM, MD, FACC, MARY JO CONLEY, WILLIAM F. ARMSTRONG, MD, FACC
Indianapolis, Indiana

JACC Vol. 5, No. 4
April 1985:918–24

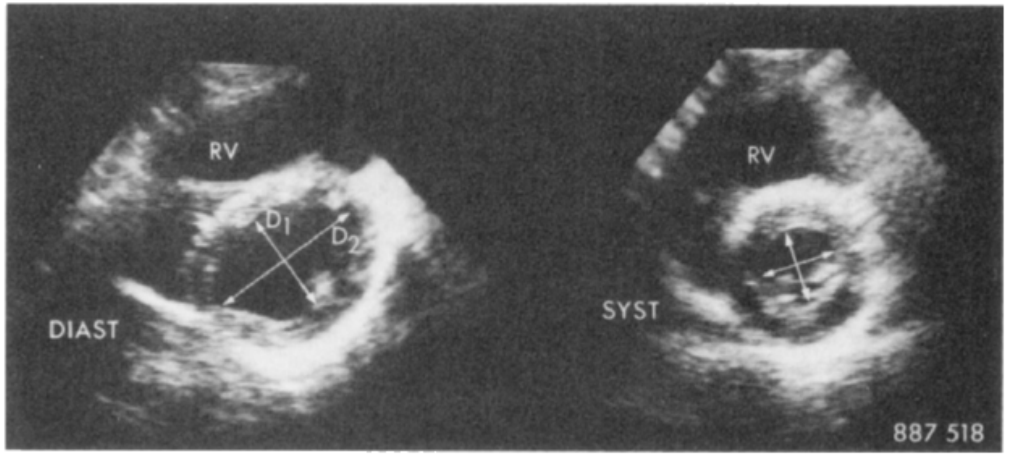
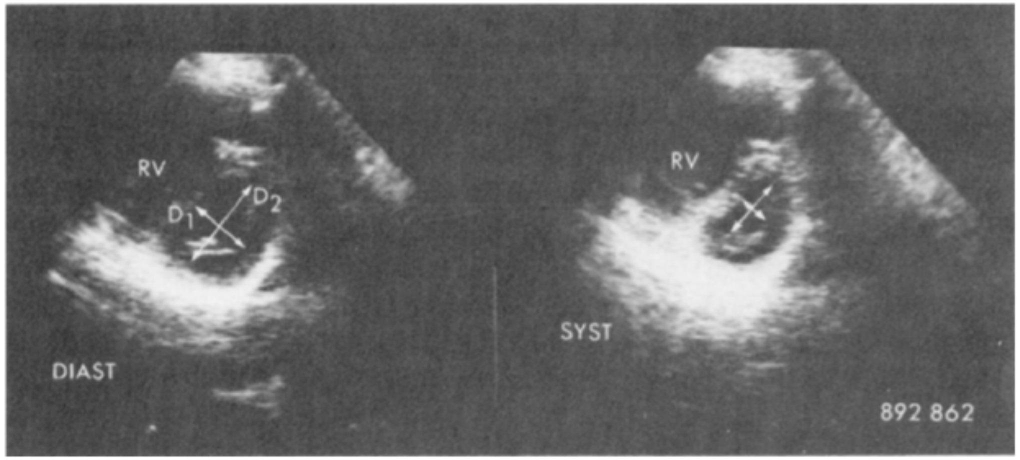


Figure 4. Right ventricular volume overload. Parasternal short-axis views from a 20 year old woman with an atrial septal defect and a pulmonary artery systolic pressure of 18 mm Hg. Abnormal left ventricular shape, apparent at end-diastole, resolved at end-systole. Eccentricity index was 1.52 and 1.04, respectively. Abbreviations as in Figure 3.

Surcharge volumiqe

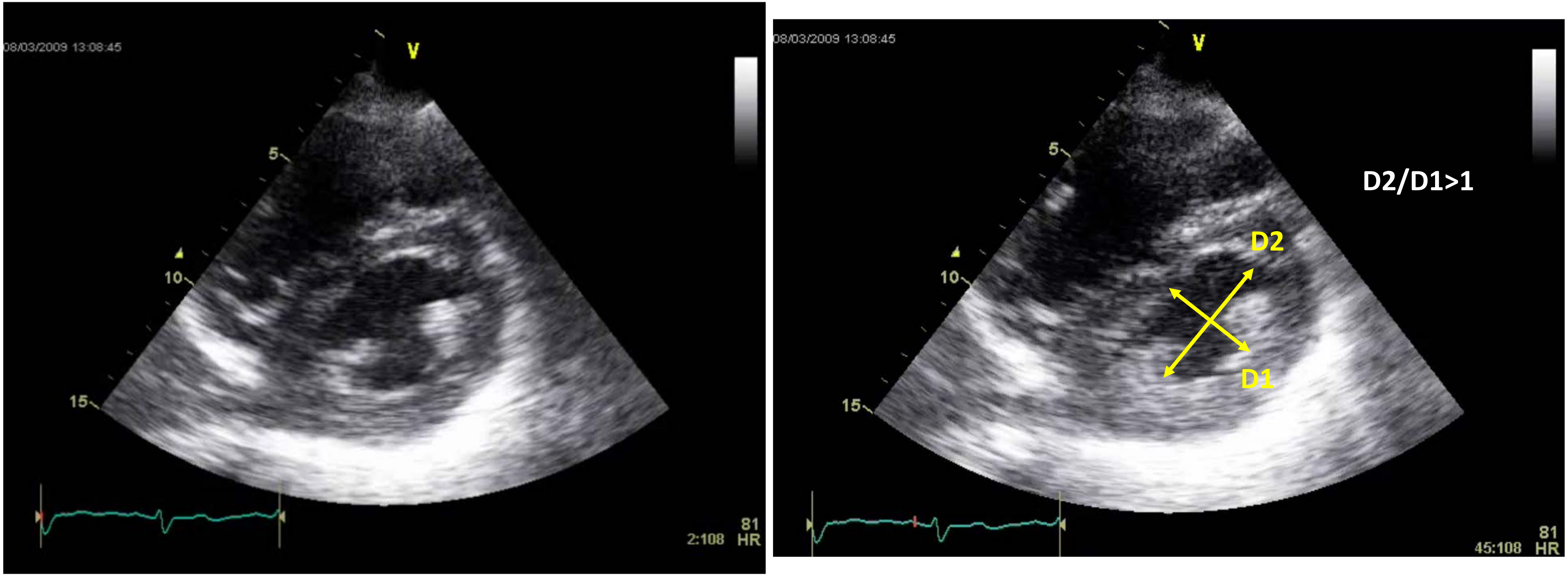
D2/D1 normal en systole
D2/D1 >1.1 en diastole

Figure 5. Right ventricular pressure overload. Parasternal short-axis views from a 28 year old woman with primary pulmonary hypertension (pulmonary artery systolic pressure of 68 mm Hg). Left ventricular deformity due to septal flattening is evident at both end-diastole and end-systole. Eccentricity index was 1.45 and 1.68, respectively. Abbreviations as in Figure 3.



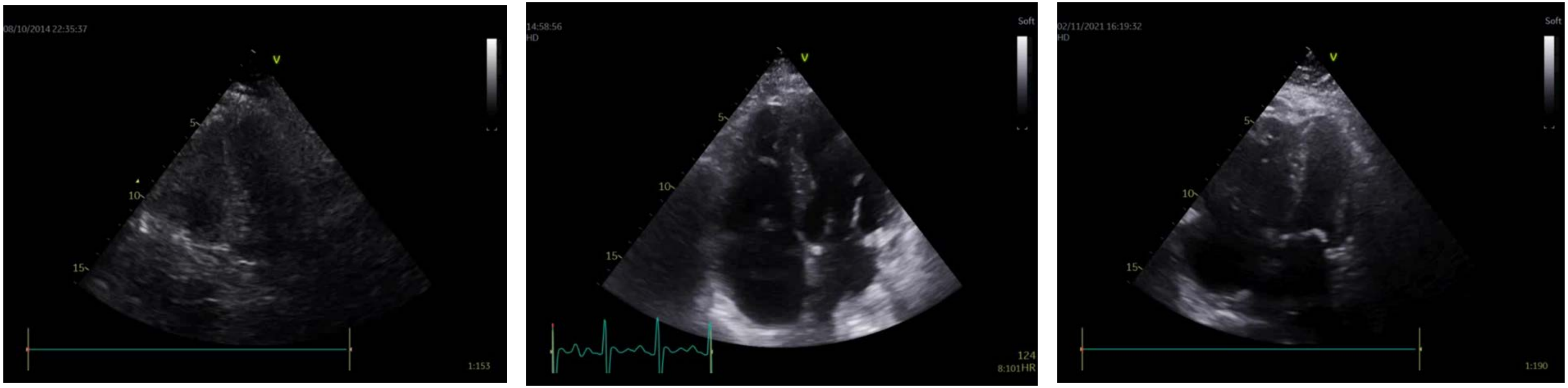
Surcharge en pression

D2/D1 >1.1 en systole
D2/D1 >1.1 en diastole

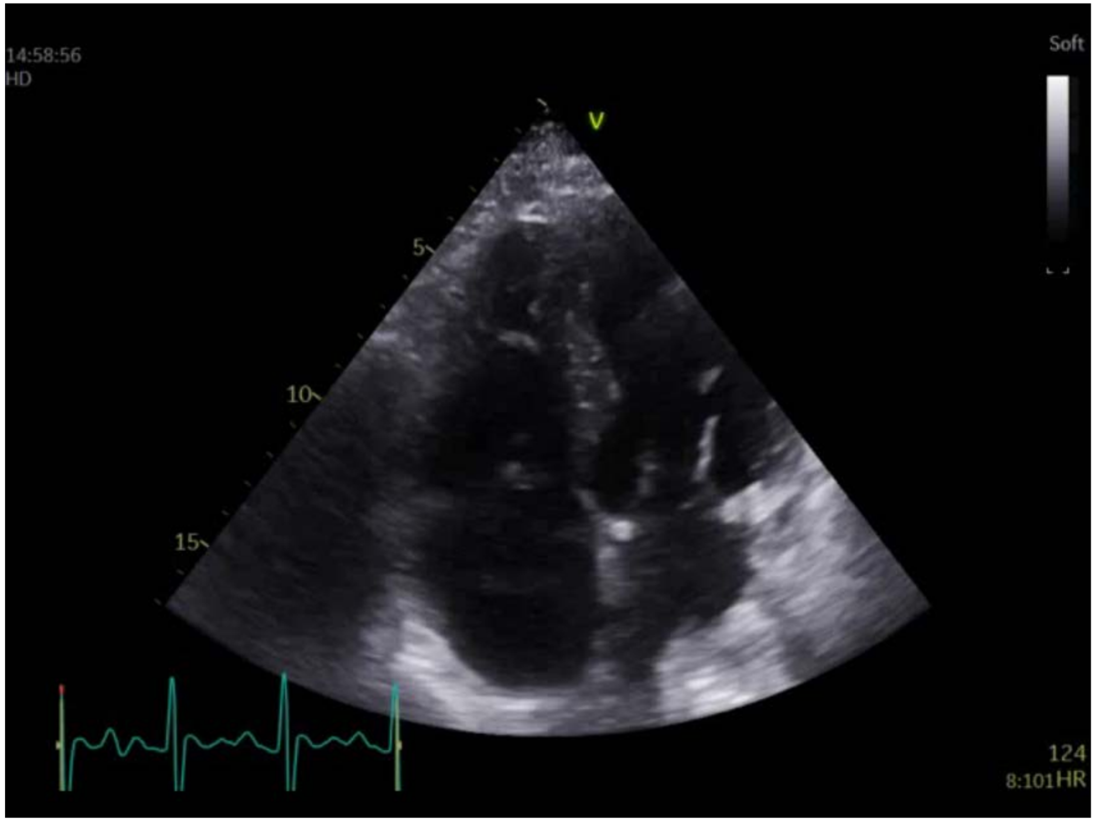

08/03/2009 13:08:45
V
5
10
15
81
2:108 HR
08/03/2009 13:08:45
V
5
10
15
D2/D1>1
D2
D1
81
45:108 HR

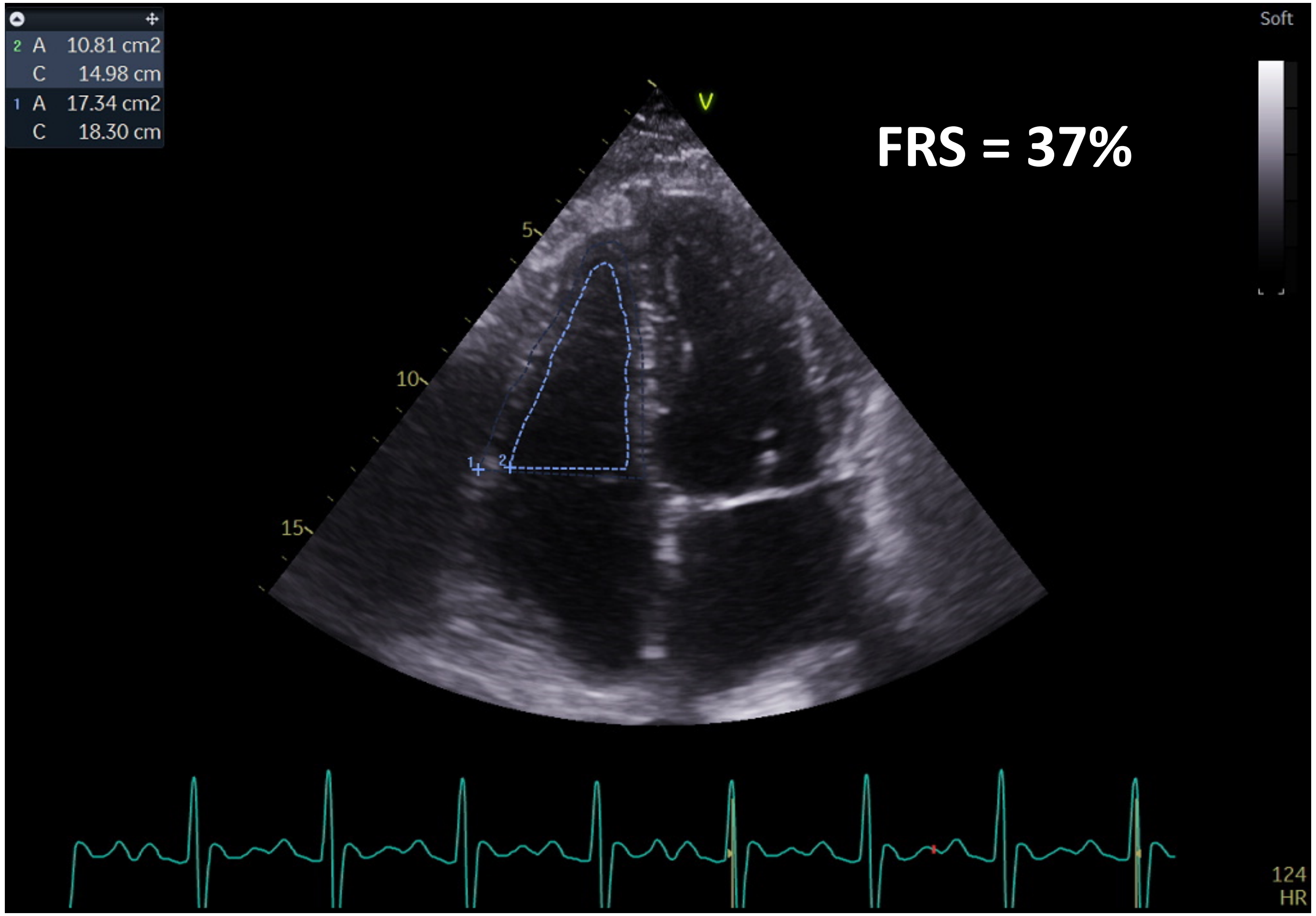
Fonction systolique du VD

1. Fraction de raccourcissement de surface
2. Déplacement de l’anneau tricuspidien:
   - TAPSE
   - Onde S DTI
3. Indice de performance myocardique (MPI)


08/10/2014 22:35:37
V
5
10
15
1:153
14:58:56
HD
Soft
V
5
10
15
124
8:101HR
02/11/2021 16:19:32
HD
Soft
V
5
10
15
1:190

Fraction de raccourcissement de surface du VD


14:58:56
HD
Soft
V
5
10
15
124
8:101HR

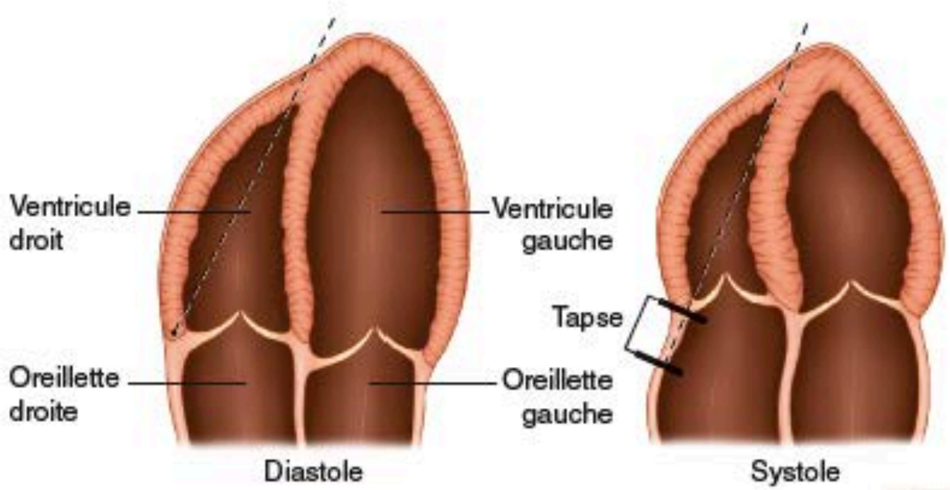

2 A 10.81 cm2
C 14.98 cm
1 A 17.34 cm2
C 18.30 cm

FRS = 37%

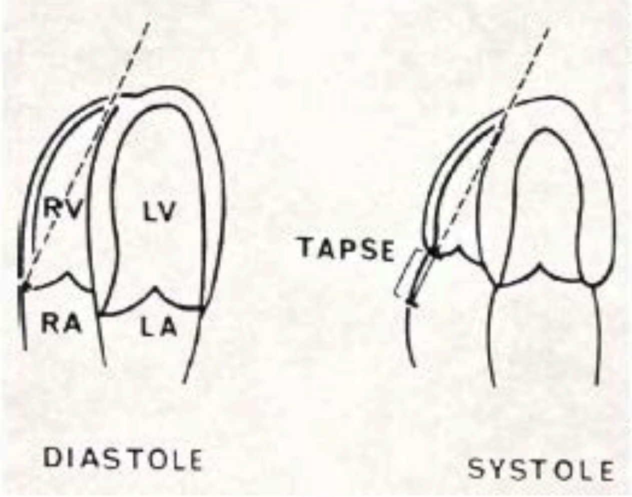
Soft
V
5
10
15
1
2
124
HR

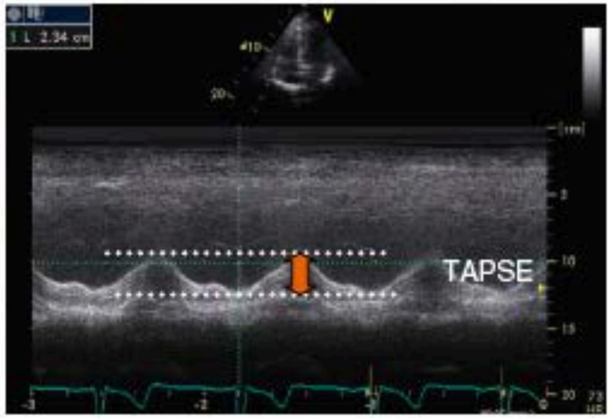
FRS = (STDVD – STSVD) / STDVD >35%

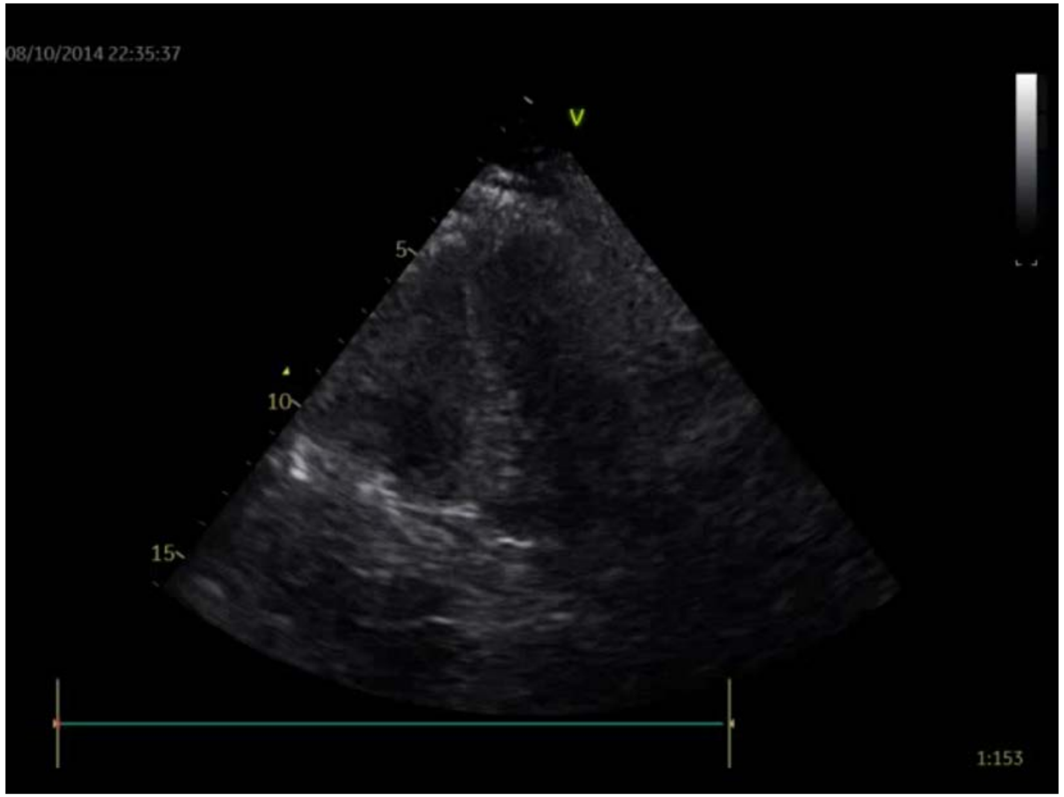
Déplacement de l’anneau Tricuspidien: TAPSE

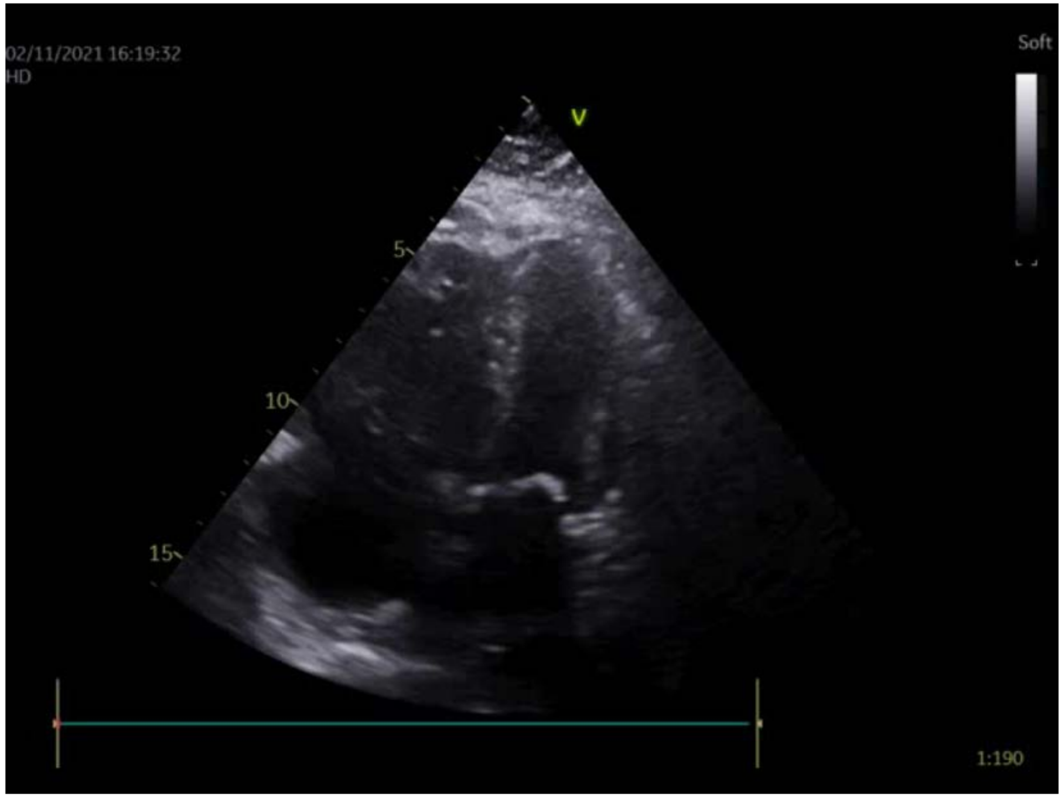

Ventricule droit
Ventricule gauche
Tapse
Oreillette droite
Oreillette gauche
Diastole
Systole

Dysfonction systolique VD:
TAPSE <16mm

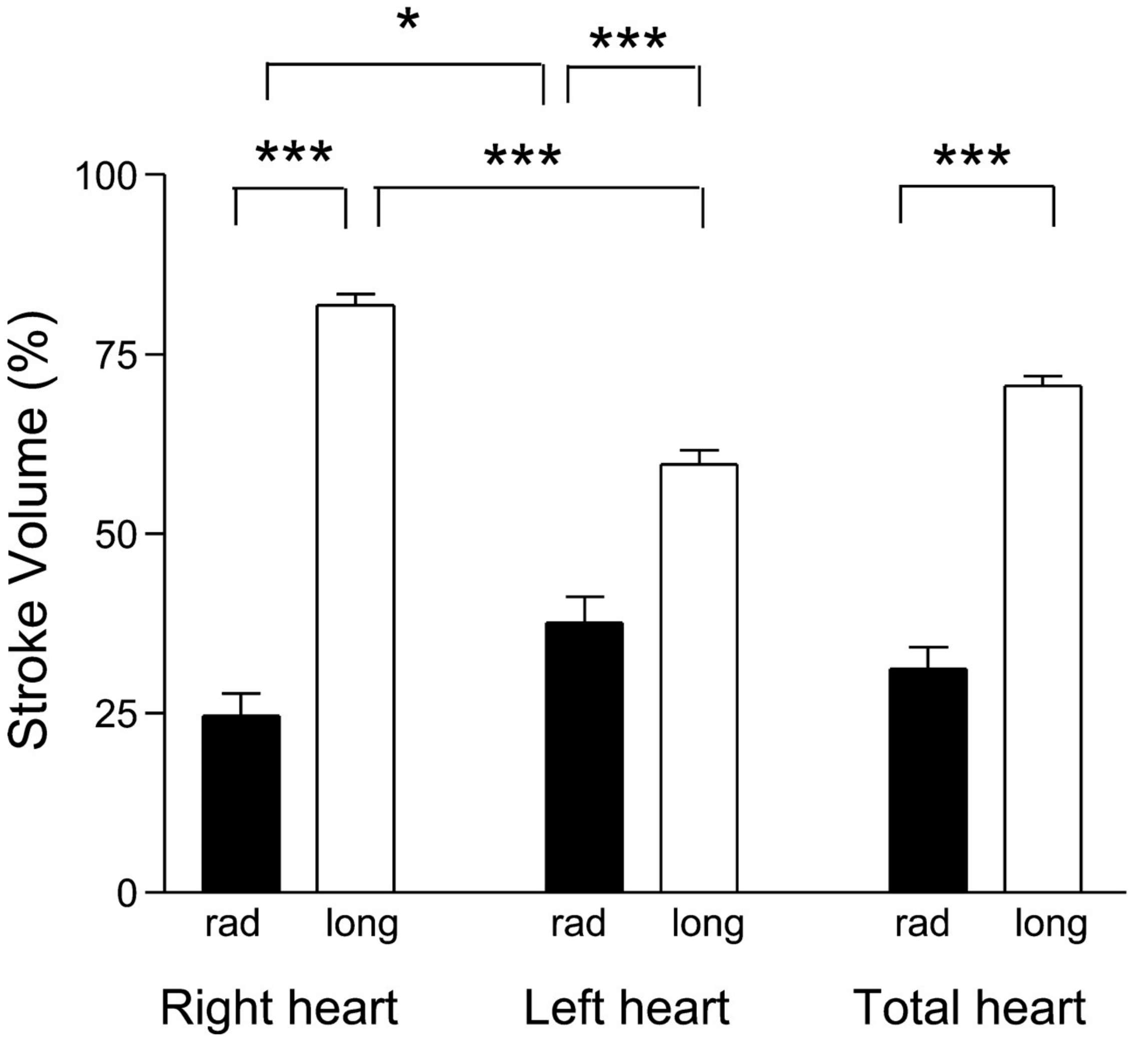

RV
LV
RA
LA
TAPSE
DIASTOLE
SYSTOLE


1 L 2.34 cm
TAPSE


08/10/2014 22:35:37
V
5
10
15
1:153

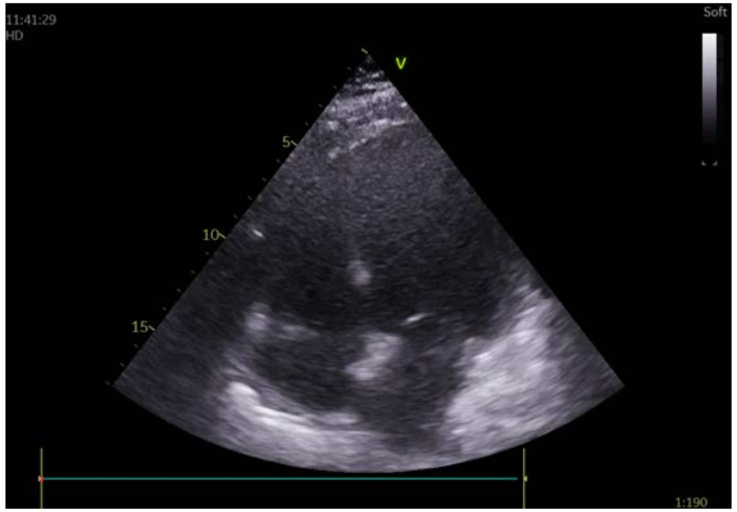

02/11/2021 16:19:32
HD
Soft
V
5
10
15
1:190

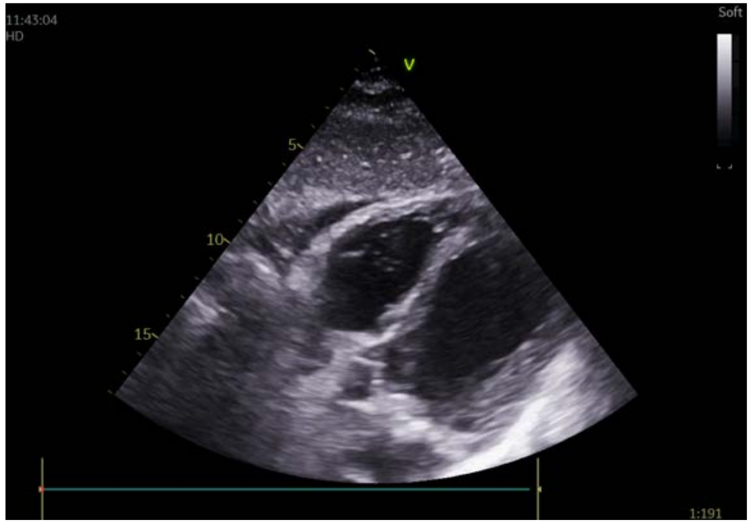
Radial (rad) and longitudinal (long) contribution to SV for the right heart, left heart, and total heart.

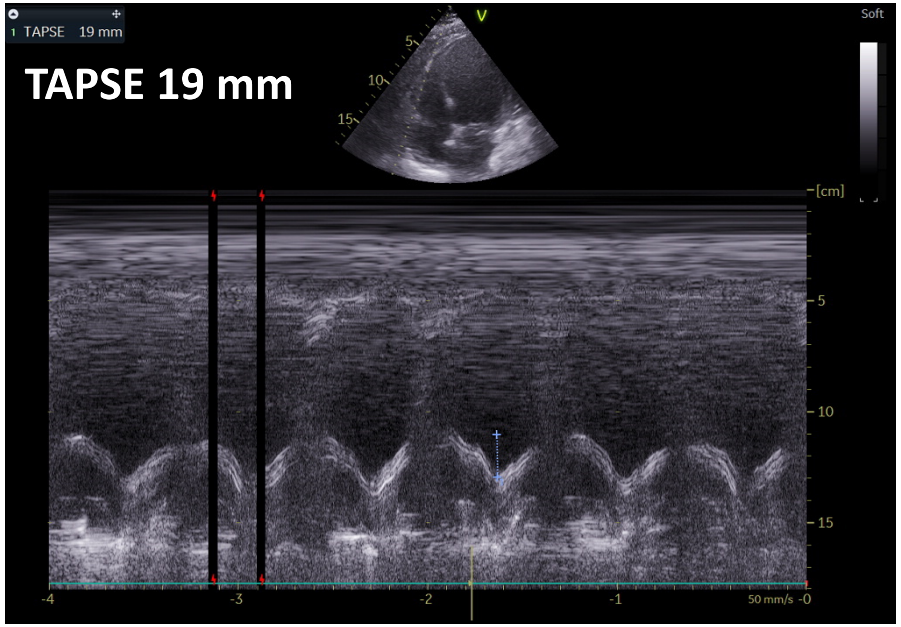


Carlsson M et al. Am J Physiol Heart Circ Physiol 2007;293:H636-H644

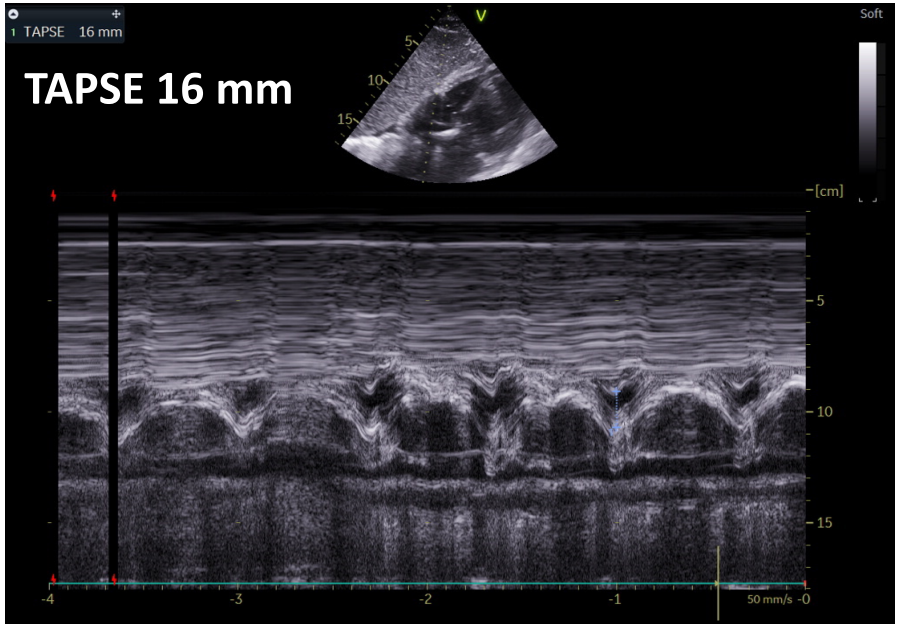
TAPSE measurement: attention à l’axe de mesure


11:41:29
HD
Soft
V
5
10
15
1:190


11:43:04
HD
Soft
V
5
10
15
1:191


TAPSE 19 mm

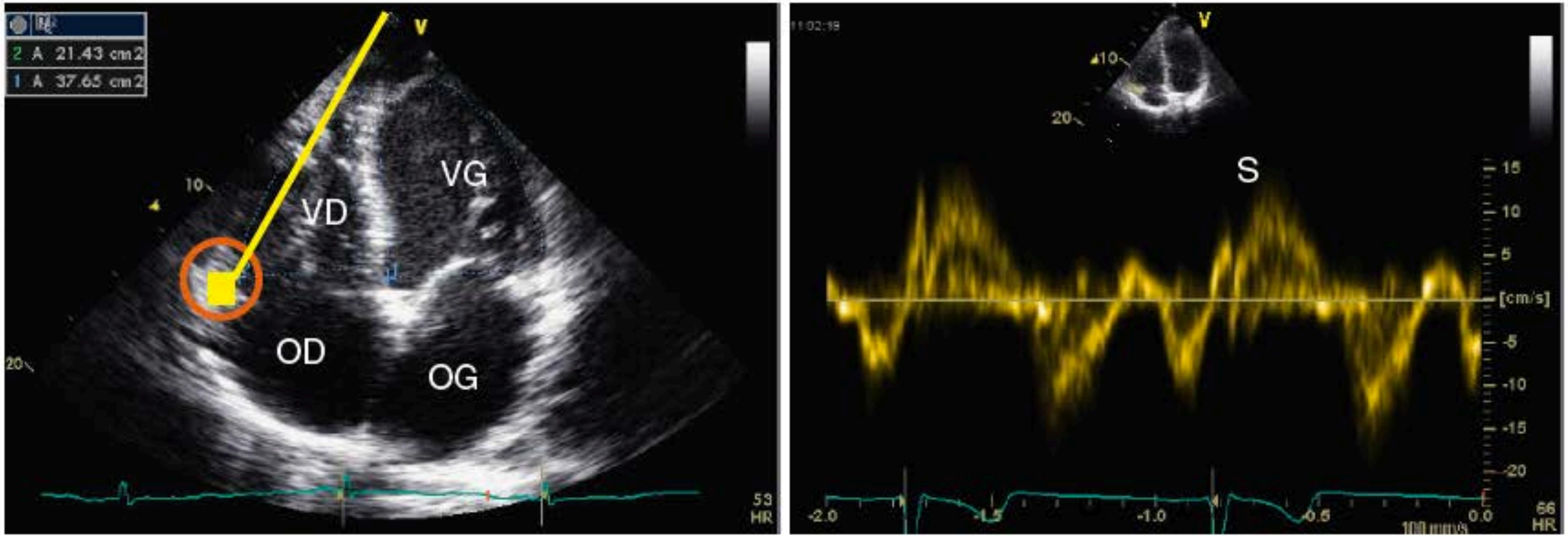
1 TAPSE 19 mm
Soft
V
[cm]
5
10
15
-4
-3
-2
-1
50 mm/s -0


TAPSE 16 mm

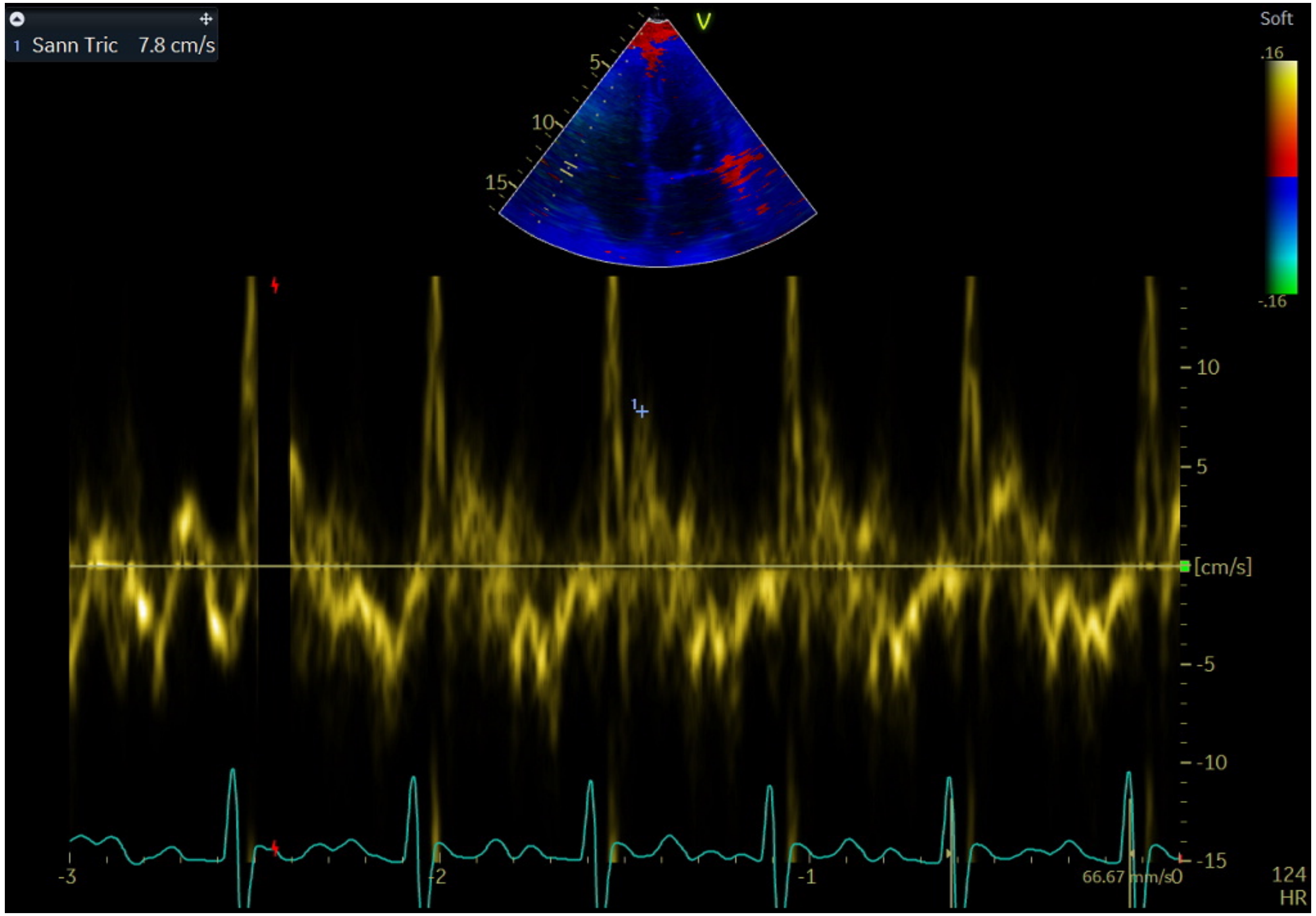
1 TAPSE 16 mm
Soft
V
[cm]
5
10
15
-4
-3
-2
-1
50 mm/s -0

Ondes S DTI

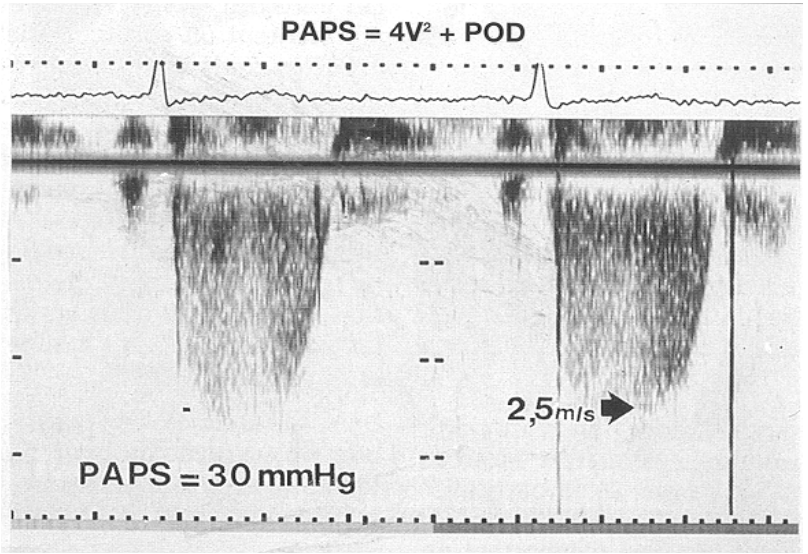
Dysfonction systolique VD:
Onde S < 10 cm/s

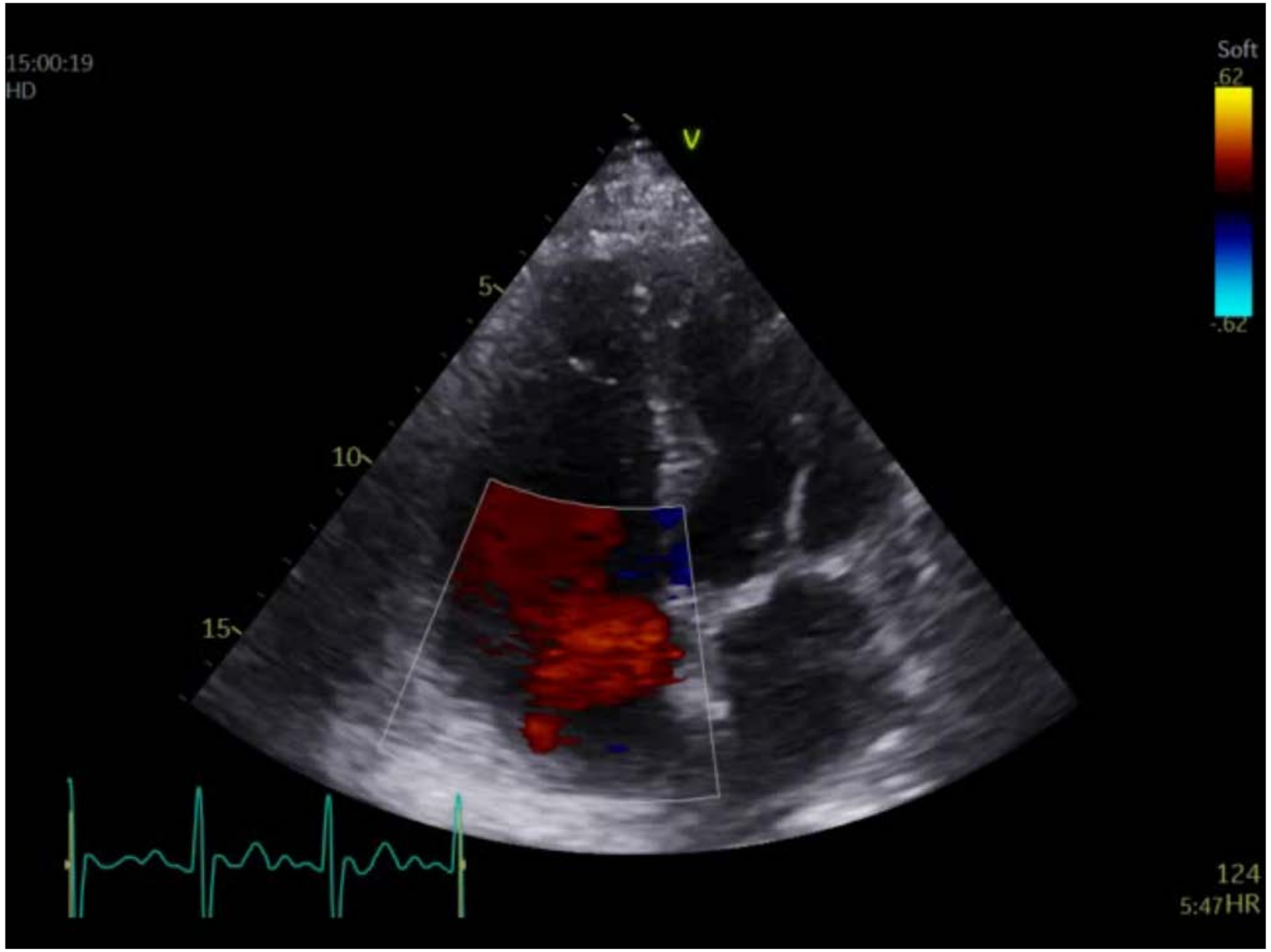

2 A 21.43 cm2
1 A 37.65 cm2
V
10
20
VD
VG
OD
OG
53
HR
11:02:18
V
10
20
S
15
10
5
[cm/s]
-5
-10
-15
-20
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s
66
HR

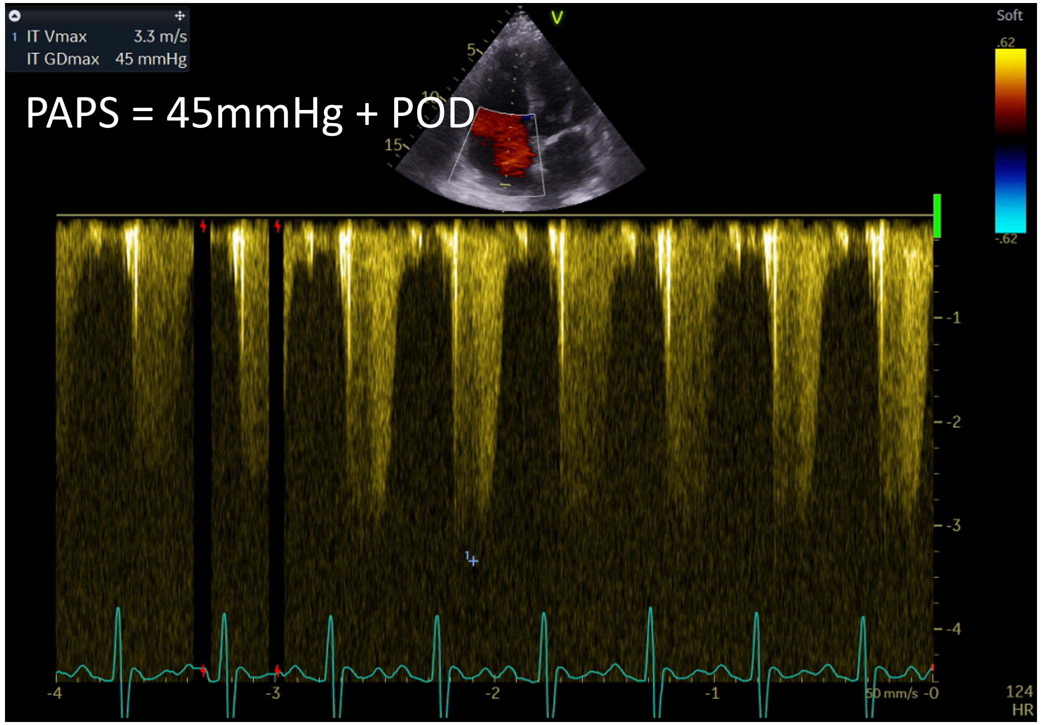
Analyse en DTI de la vitesse de l'anneau tricuspidien à partir d'une coupe apicale des 4 cavités.


1 Sann Tric 7.8 cm/s
V
Soft
.16
-.16
5
10
15
10
5
[cm/s]
-5
-10
-15
-3
-2
-1
66.67 mm/s
0
124
HR

Mesure de la PAPS
Tricuspid Regurgitation


PAPS = 4V² + POD
2,5m/s
PAPS = 30 mmHg


15:00:19
HD
Soft
.62
-.62
V
5
10
15
124
5:47HR


1 IT Vmax 3.3 m/s
IT GDmax 45 mmHg
PAPS = 45mmHg + POD
V
5
15
Soft
.62
-.62
-1
-2
-3
-4
-4
-3
-2
-1
50 mm/s -0
124
HR

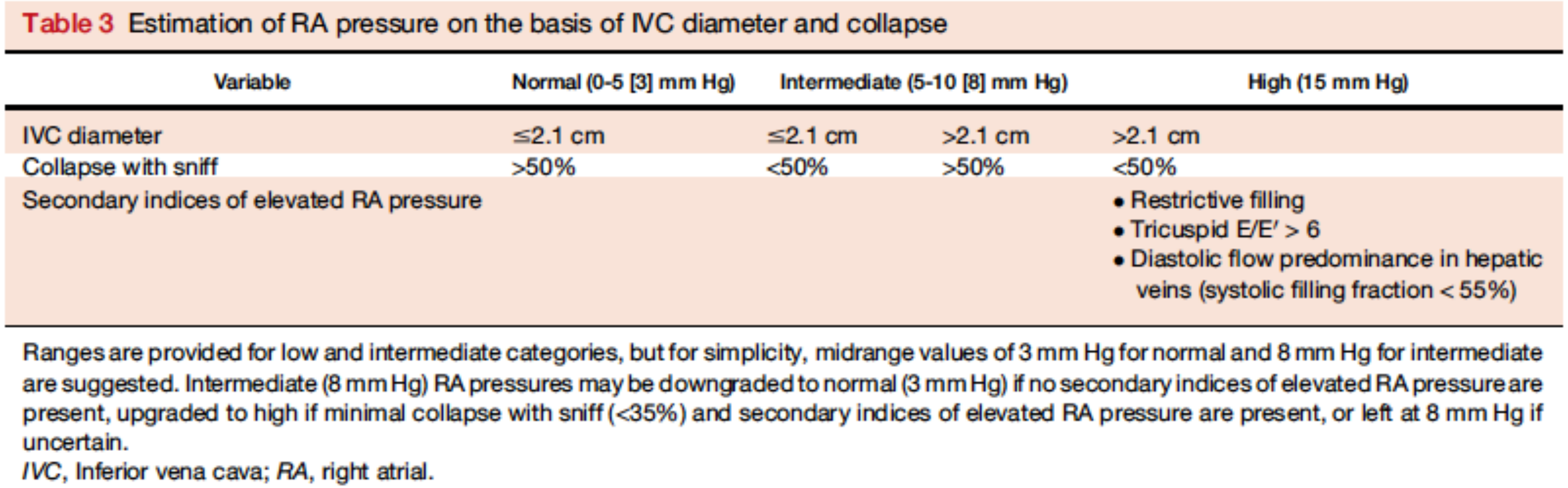
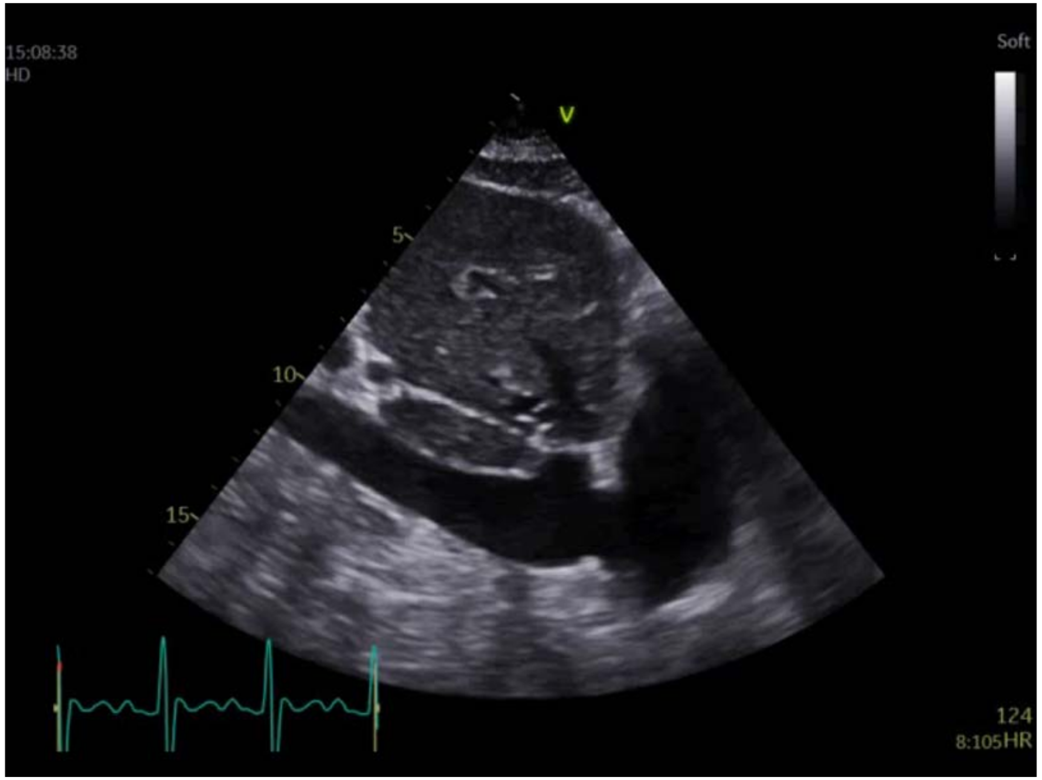


Table 3 Estimation of RA pressure on the basis of IVC diameter and collapse

Variable	Normal (0-5 [3] mm Hg)	Intermediate (5-10 [8] mm Hg)		High (15 mm Hg)
IVC diameter	≤2.1 cm	≤2.1 cm	>2.1 cm	>2.1 cm
Collapse with sniff	>50%	<50%	>50%	<50%
Secondary indices of elevated RA pressure				• Restrictive filling • Tricuspid E/E′ > 6 • Diastolic flow predominance in hepatic veins (systolic filling fraction < 55%)

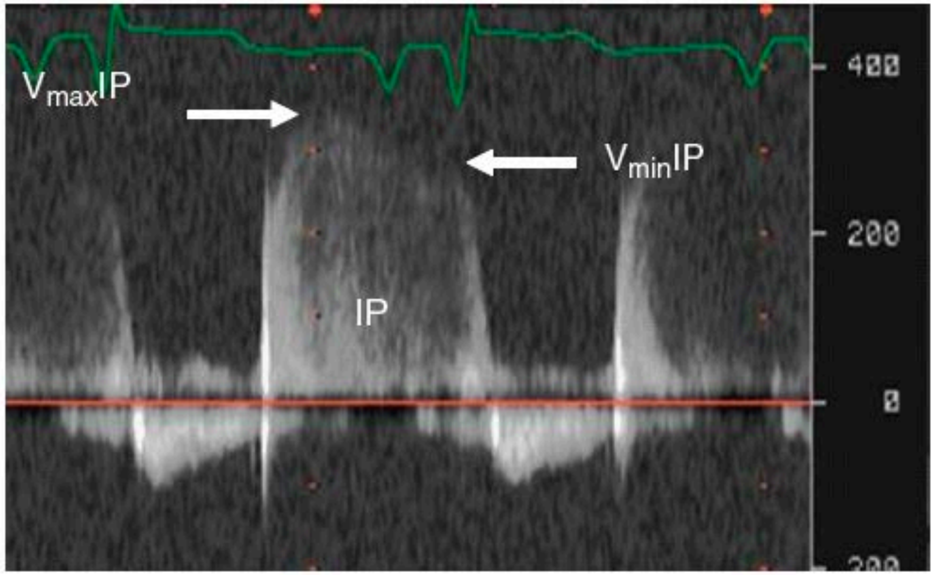
Ranges are provided for low and intermediate categories, but for simplicity, midrange values of 3 mm Hg for normal and 8 mm Hg for intermediate are suggested. Intermediate (8 mm Hg) RA pressures may be downgraded to normal (3 mm Hg) if no secondary indices of elevated RA pressure are present, upgraded to high if minimal collapse with sniff (<35%) and secondary indices of elevated RA pressure are present, or left at 8 mm Hg if uncertain.

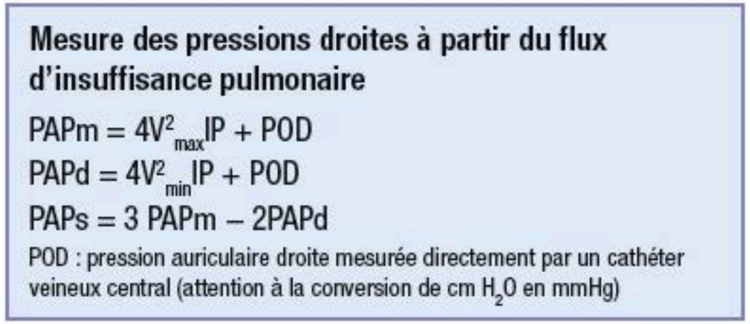
IVC, Inferior vena cava; *RA*, right atrial.



PAPS= 45 + 15 = 60mmHg

Mesure de la PAPS




Mesure des pressions droites à partir du flux d'insuffisance pulmonaire

$PAPm = 4V^2_{max}IP + POD$

$PAPd = 4V^2_{min}IP + POD$

$PAPs = 3\ PAPm - 2PAPd$

POD : pression auriculaire droite mesurée directement par un cathéter veineux central (attention à la conversion de cm H_2O en mmHg)

Calcul des résistances vasculaires pulmonaires

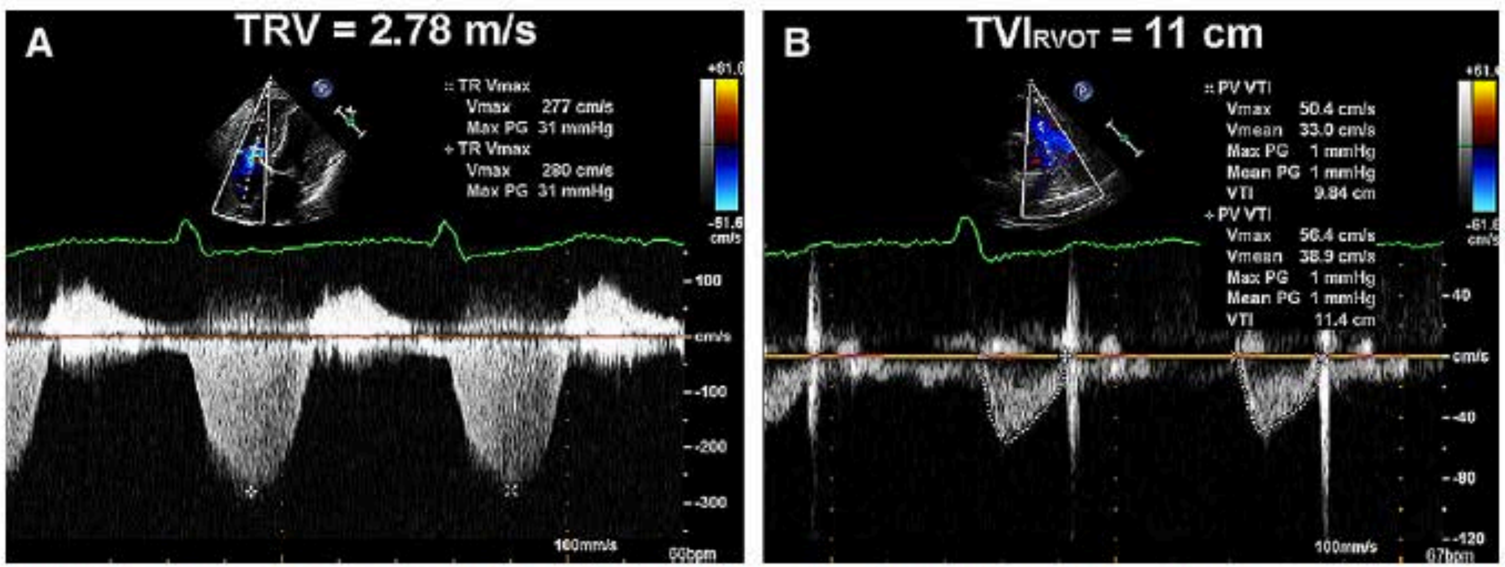

A
TRV = 2.78 m/s
TR Vmax
Vmax 277 cm/s
Max PG 31 mmHg
TR Vmax
Vmax 280 cm/s
Max PG 31 mmHg
100
cm/s
-100
-200
-300
100mm/s
60bpm
B
TVIRVOT = 11 cm
PV VTI
Vmax 50.4 cm/s
Vmean 33.0 cm/s
Max PG 1 mmHg
Mean PG 1 mmHg
VTI 9.84 cm
PV VTI
Vmax 56.4 cm/s
Vmean 38.9 cm/s
Max PG 1 mmHg
Mean PG 1 mmHg
VTI 11.4 cm
40
cm/s
-40
-80
-120
100mm/s
67bpm

Figure 14 The two elements needed to calculate the noninvasive index of pulmonary vascular resistance (PVR) are found in this example. The ratio of peak tricuspid regurgitant velocity (TRV) (2.78 m/s) to the time-velocity integral (TVI) (11 cm) in the right ventricular outflow tract (RVOT) is abnormal at 0.25 (normal, ≤0.15). The estimated PVR is 2.68 using the formula (TRVmax/RVOT TVI) × 10 + 0.16.[84] Adapted with permission from *J Am Soc Echocardiogr*.[52]

Indice de performance myocardique (MPI)

$$\text{MPI} = \frac{\left[\begin{array}{c}\text{Temps de Relaxation IsoVolumique} \\ + \\ \text{Temps de Contraction IsoVolumique}\end{array}\right]}{\text{Temps d'éjection}}$$

Dysfonction VD si:
- MPI >0.4 en Doppler
- MPI >0.55 en DTI

1. Dépendant des conditions de charge: sous estimation en cas de POD élevée
2. Contre indiqué en cas d'arythmie

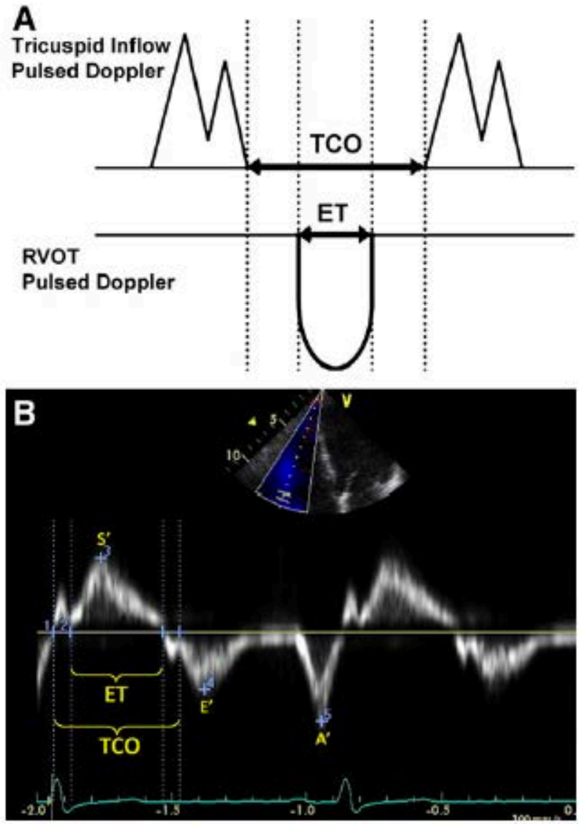
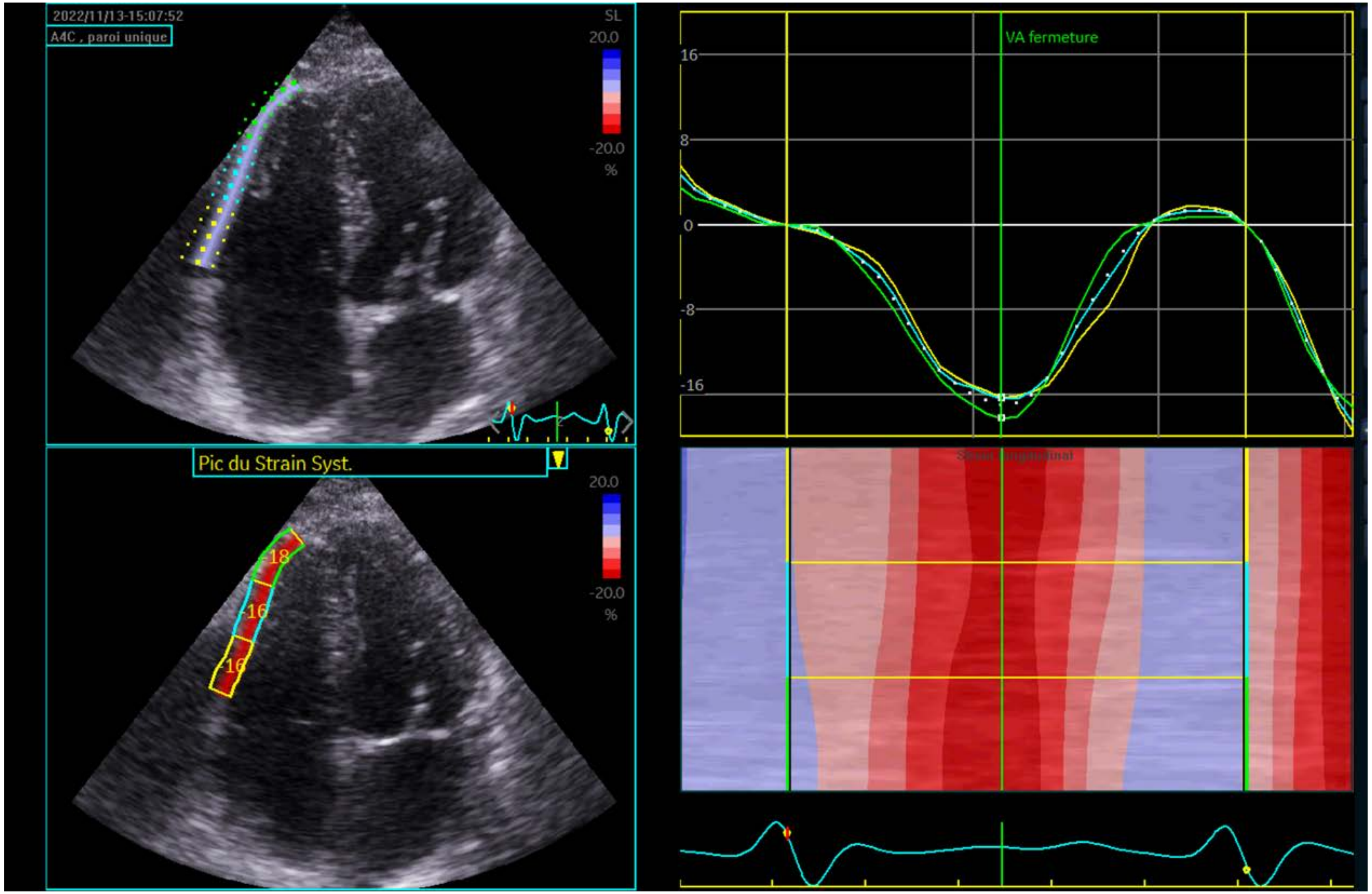


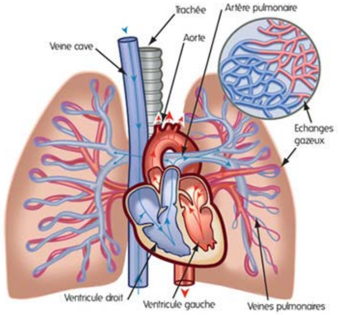
Figure 16 Calculation of right ventricular myocardial performance index (MPI) by pulsed Doppler **(A)** and pulsed tissue Doppler **(B)**. The tricuspid (valve) closure opening time (TCO) encompasses isovolumic contraction time, ejection time (ET), and isovolumic relaxation time. In the pulsed Doppler method, TCO can also be measured by the duration of the tricuspid regurgitation continuous-wave Doppler signal. MPI = (TCO − ET)/ET. Note that S′, E′, and A′ are also measured from the same pulsed Doppler tissue image.

Nouvelles méthodes d’évaluation de la fonction du VD


2022/11/13-15:07:52
A4C , paroi unique
SL
20.0
-20.0
%
Pic du Strain Syst.
-18
-16
-16
20.0
-20.0
%
VA fermeture
16
8
0
-8
-16

Conséquences myocardiques

- Interactions cœur-poumons
- Patients polypathologiques


Trachée
Artère pulmonaire
Veine cave
Aorte
Echanges gazeux
Ventricule droit
Ventricule gauche
Veines pulmonaires

- **Hypothèse :**

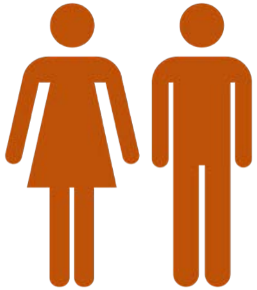
La PEP pourrait diminuer le débit coronaire par majoration des pressions intrathoraciques

- **Objectif :**

Evaluer la variation du débit coronaire en ETO entre une basse PEP et une haute PEP

Caractéristiques démographiques de la population générale

N=32

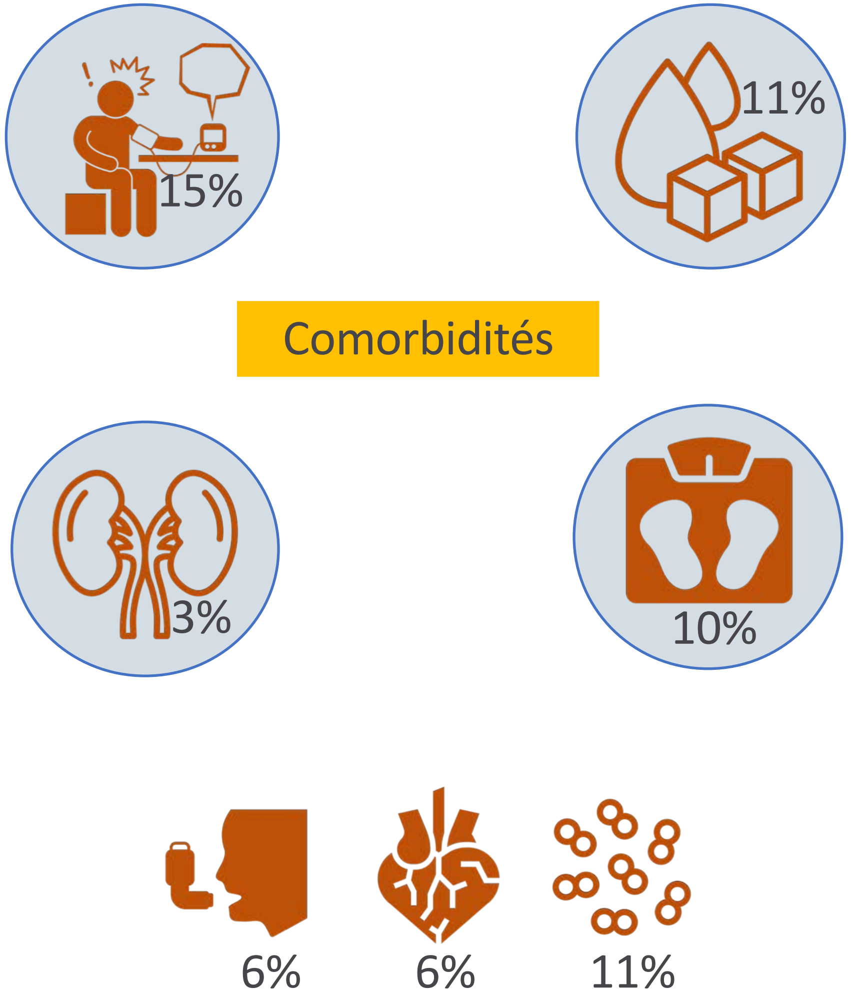


37,5% 62,5%

Âge médian 64,5 ans

IMC médian 28,3 kg/m^2

Comorbidités



SOFA score : 7

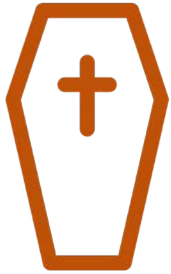
Vasopresseurs 93,8%

Inotropes 9,4%

Etiologie virale 40,6%

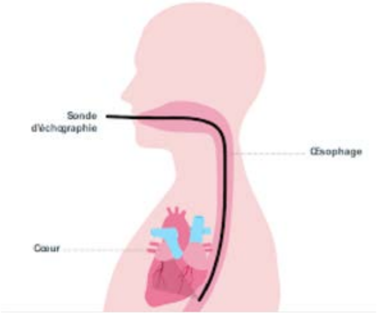
Sévérité du SDRA

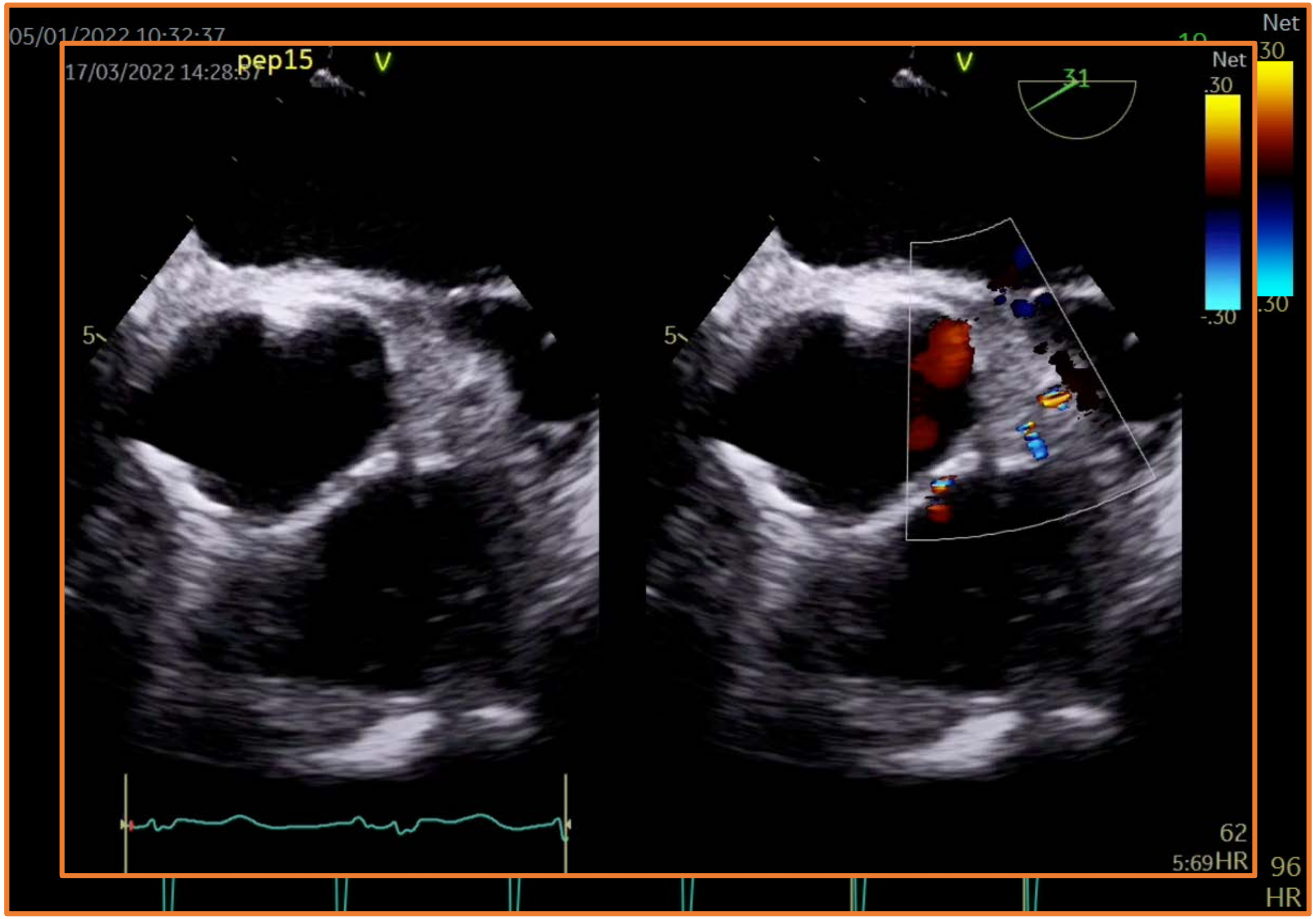
- Léger 15,6%
- Modéré 25%
- Sévère 59,4%

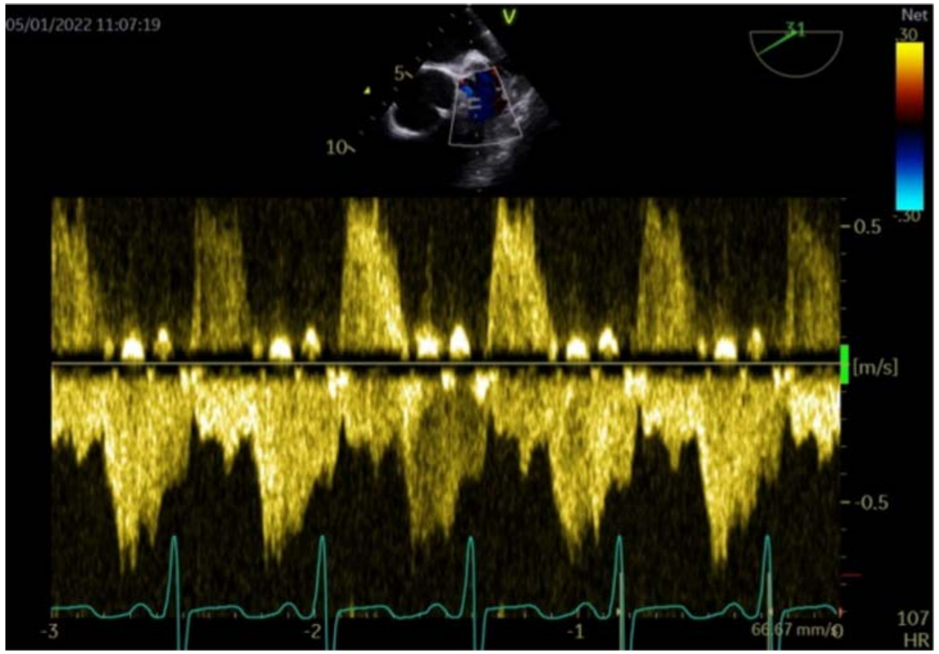


Mesure du débit coronaire en ETO

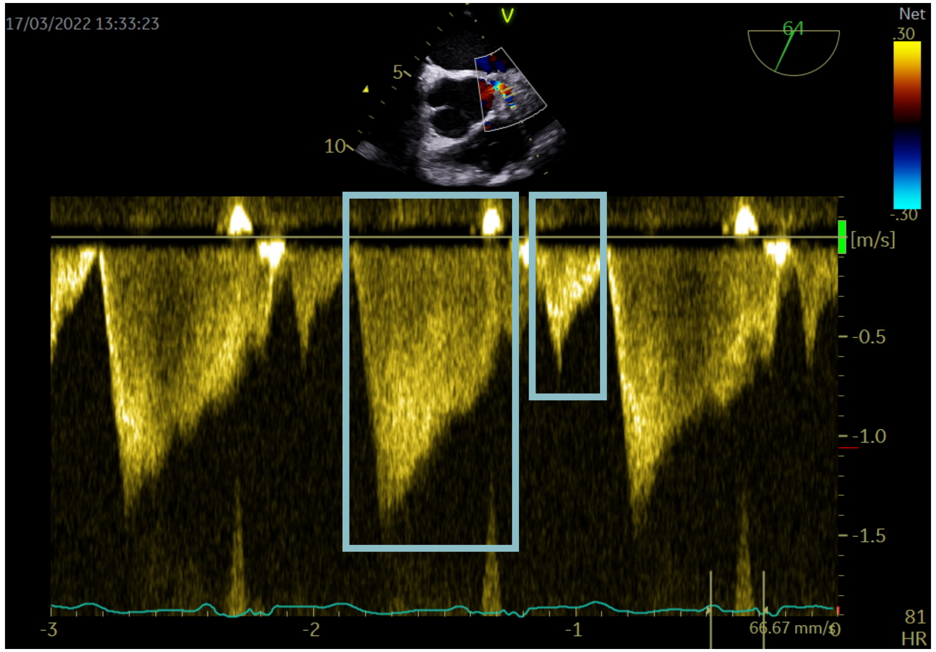
Echographie trans-oesophagienne


Sonde d'échographie
Œsophage
Cœur

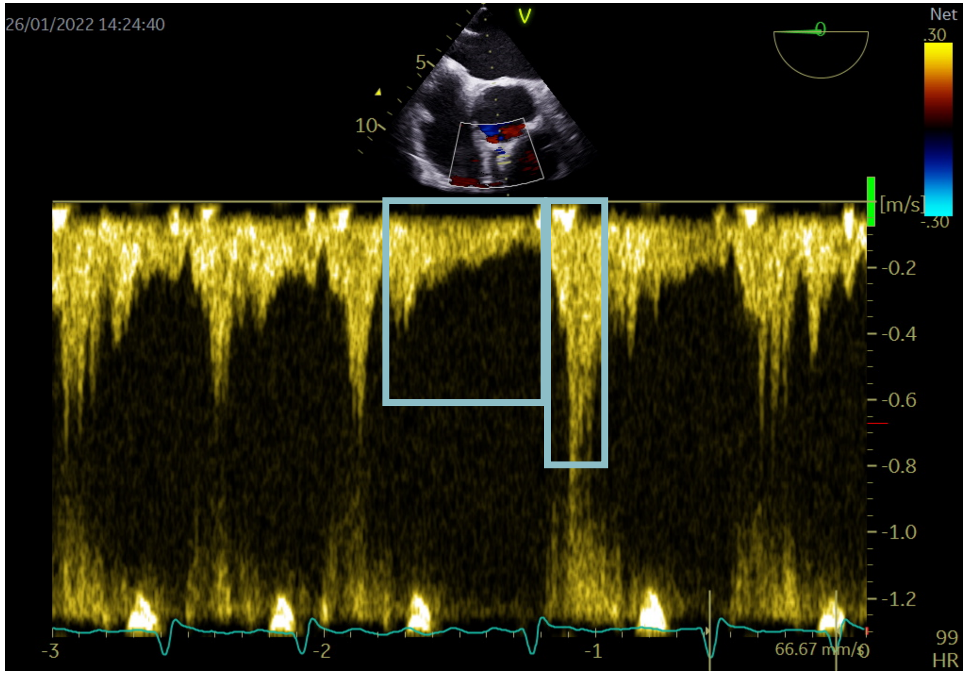

05/01/2022 10:32:37
17/03/2022 14:28:57
pep15
V
V
31
Net
.30
-.30
Net
30
.30
5
5
62
5:69HR
96
HR

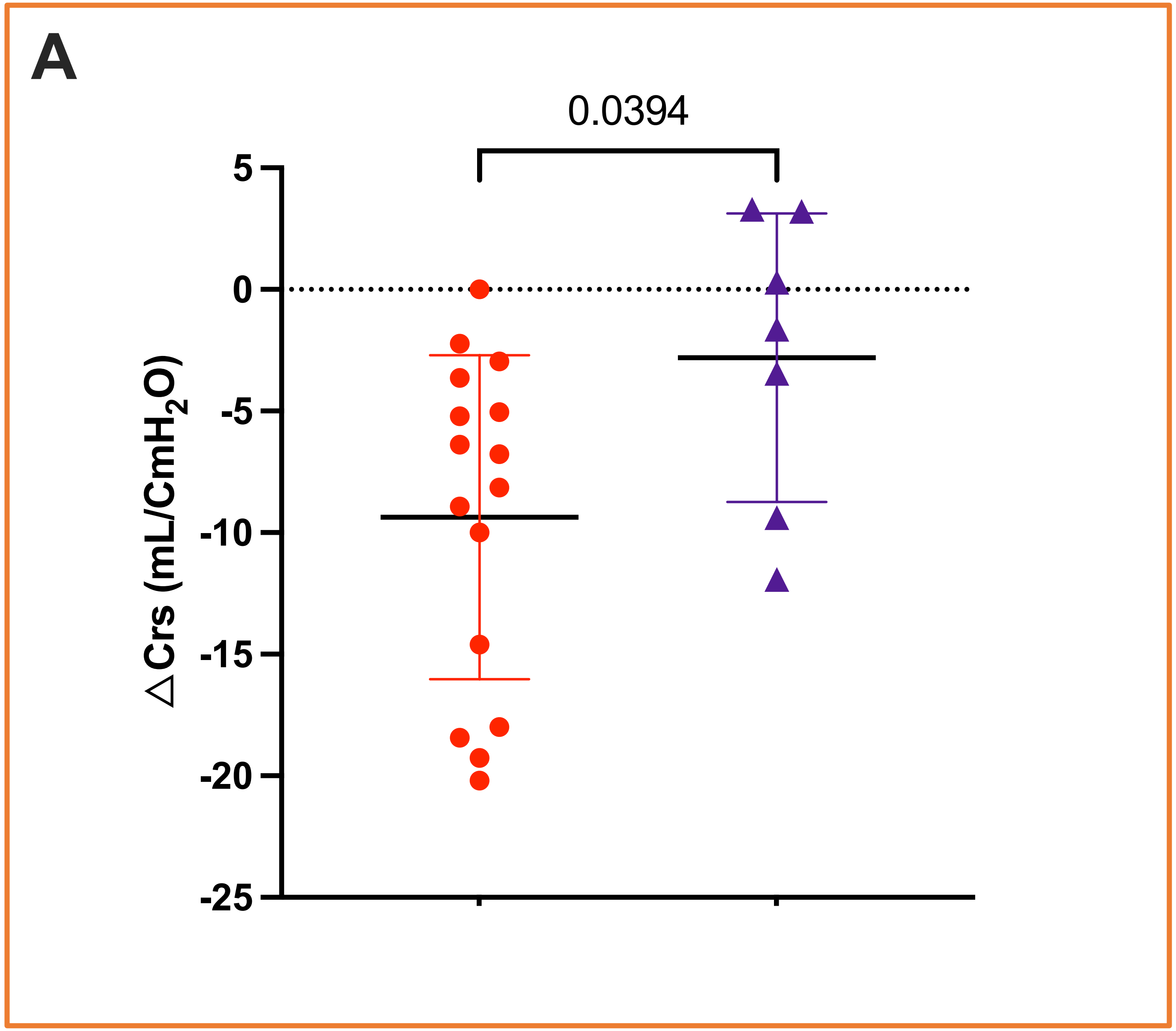

05/01/2022 11:07:19
V
31
Net
30
-30
5
10
0.5
[m/s]
-0.5
-3
-2
-1
66.67 mm/s
0
107
HR

Coronaire gauche


17/03/2022 13:33:23
V
64
Net
30
-30
5
10
[m/s]
-0.5
-1.0
-1.5
-3
-2
-1
66.67 mm/s
0
81
HR

Coronaire droite


26/01/2022 14:24:40
V
0
Net
30
-30
5
10
[m/s]
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
-3
-2
-1
66.67 mm/s
0
99
HR



Comparaison de la variation des variables cliniques et échographiques entre PEP basse et PEP haute dans chaque groupe

▲ Maintien du débit coronaire

● Diminution du débit coronaire

Conclusions

1. On n'évalue pas le VD comme on évalue le VG
    - Pas la même anatomie
    - Pas les mêmes propriétés
    - Pas les mêmes paramètres d'évaluation

2. Gold standard: IRM myocardique
    (fonction et étiologie)

3. Echocardiographie
    - Dimension VD/VG
    - Interaction VD/VG (septum)
    - Fonction systolique (FRS, TAPSE, Stric)
    - PAPS et POD

Bibliographie:

- Chemla D, Rev Mal Respi 2018
- Rudski LG, JASE 2010
- Sanz J, JACC 2019
- Lahm T, AJRCCM 2018