L'échocardiographie et le Doppler

Michel Slama

Amiens

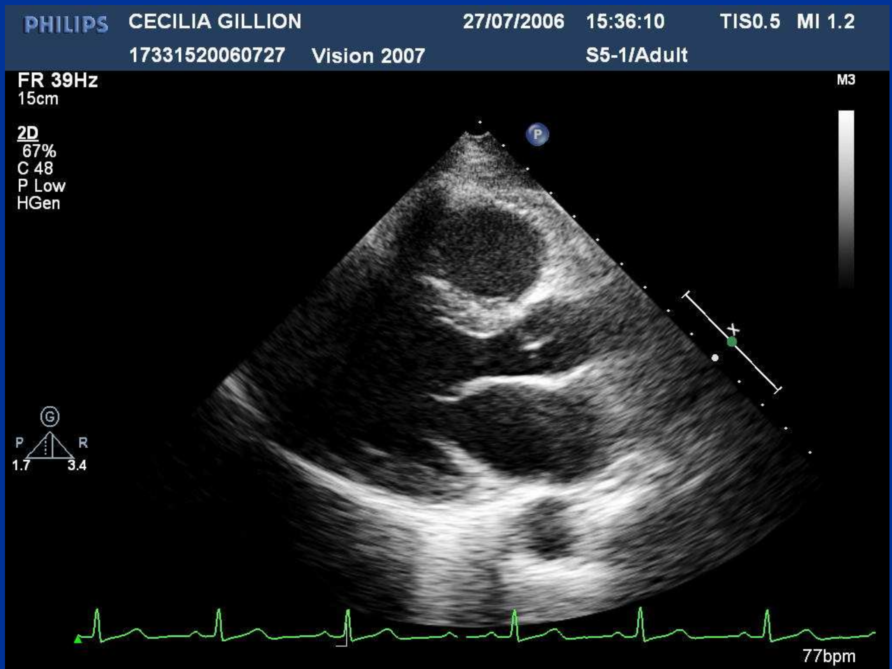
France

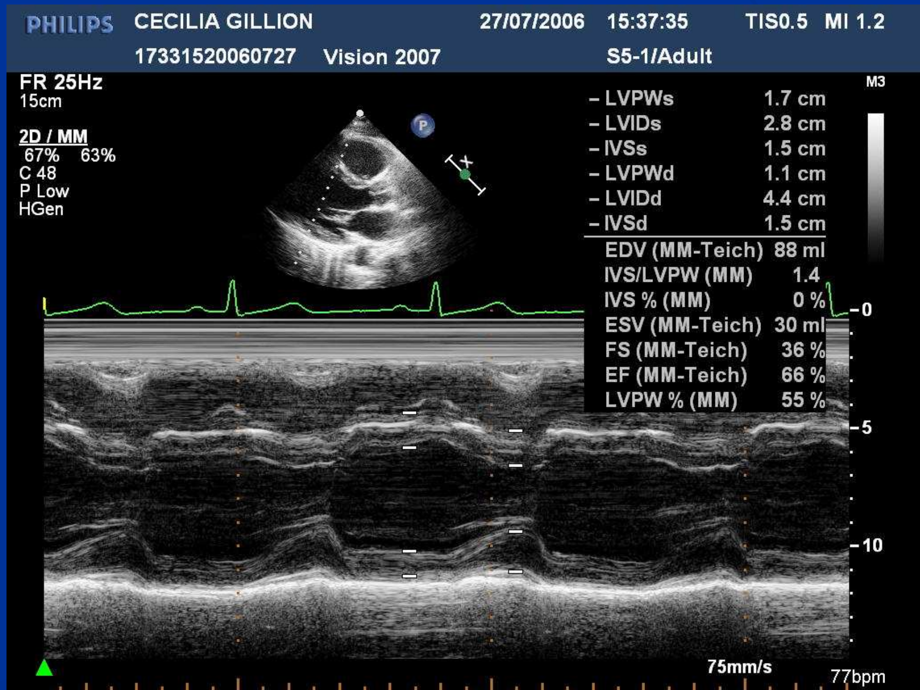
Echocardiographie

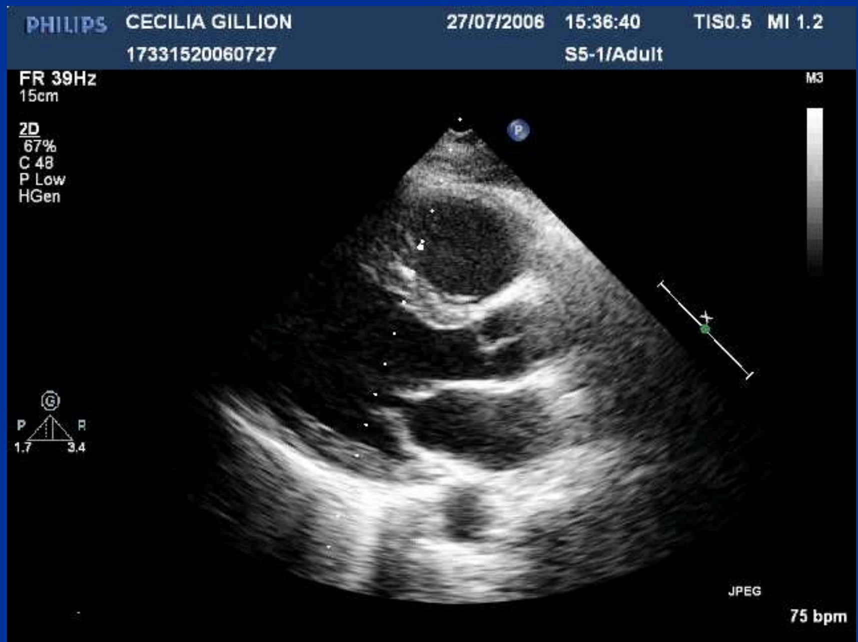
- Deux grandes techniques
  - Echocardiographie = structure
  - Doppler = hémodynamique
- Deux voies d'abord du cœur
  - Voie transthoracique
  - Voie transoesophagienne

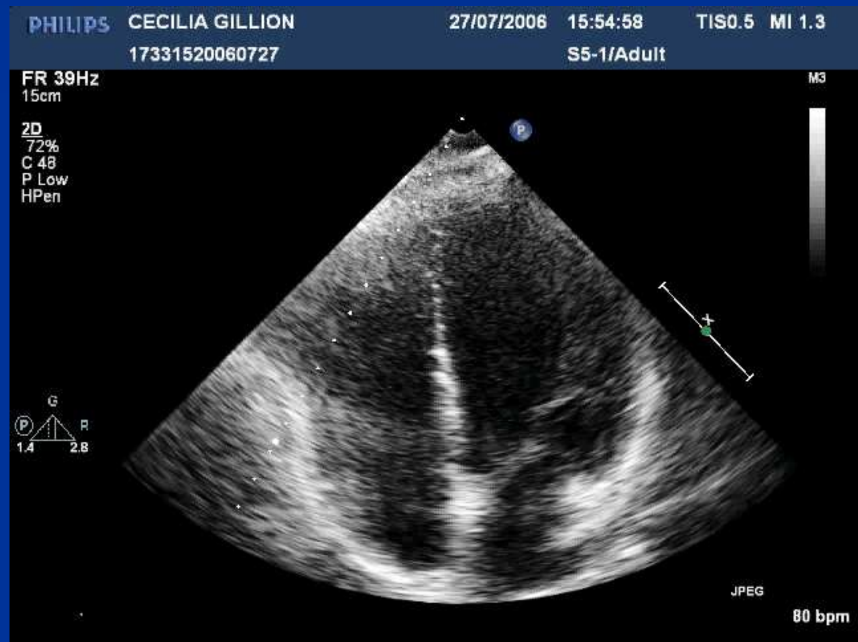
Echocardiographie = analyse des structures

Echocardiographie = analyse des structure





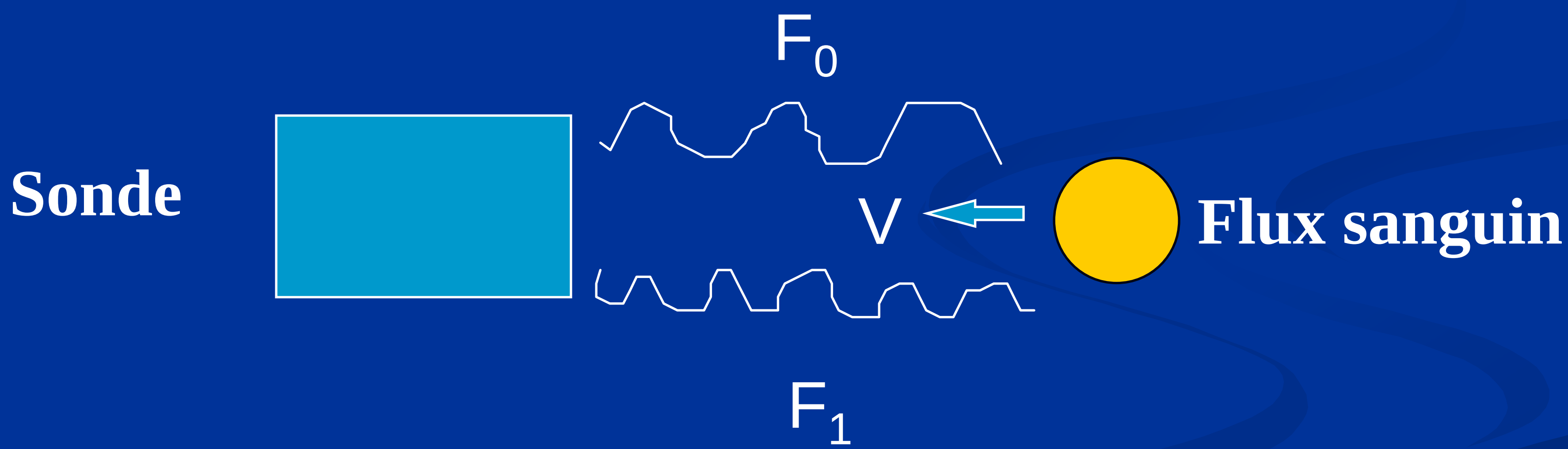

PHILIPS
CECILIA GILLION
17331520060727
27/07/2006 15:36:40
TIS0.5 MI 1.2
S5-1/Adult
FR 39Hz
15cm
2D
67%
C 48
P Low
HGen
M3
JPEG
75 bpm


PHILIPS
CECILIA GILLION
17331520060727
27/07/2006 15:54:58
TIS0.5 MI 1.3
S5-1/Adult
FR 39Hz
15cm
2D
72%
C 48
P Low
HPen
M3
JPEG
80 bpm

Doppler = hémodynamique non invasive (pression, débit)

Vélocité

- **Accessible par le Doppler cardiaque : glissement de fréquence**

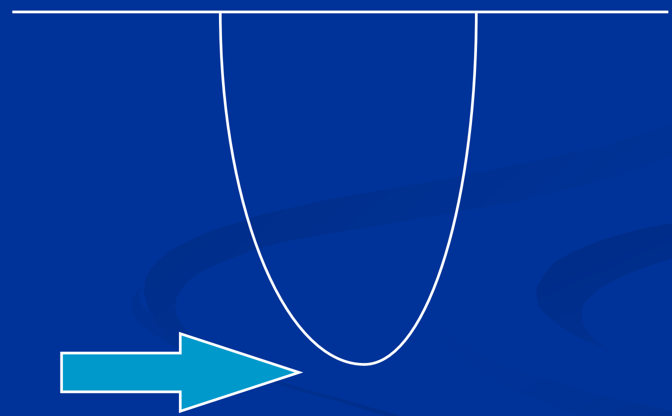


$$V = (F_1 - F_0) \times C / 2 \times F_0 \times \cos a$$

Vélocité

Flux systolique enregistré en Doppler au niveau de l'anneau aortique

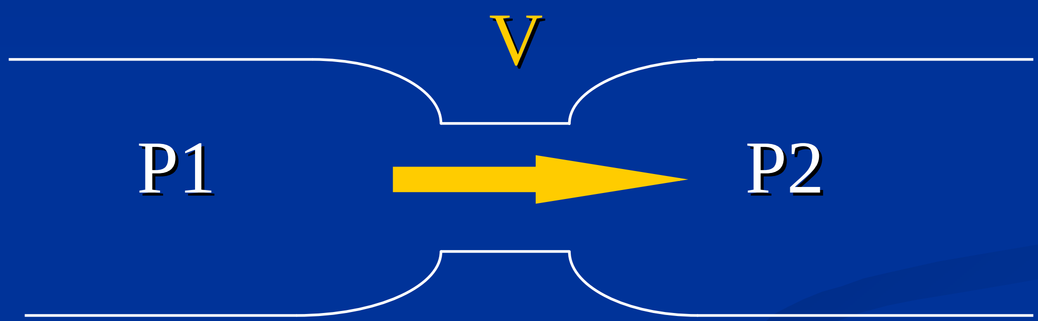
0 m/s



V max = 1 m/s

VTI : intégration des vitesses pendant la systole

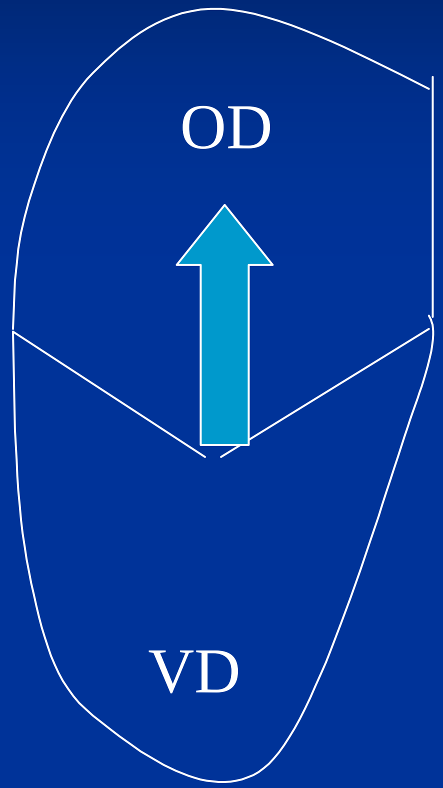
Le Doppler : "Swan-Ganz" non-invasive

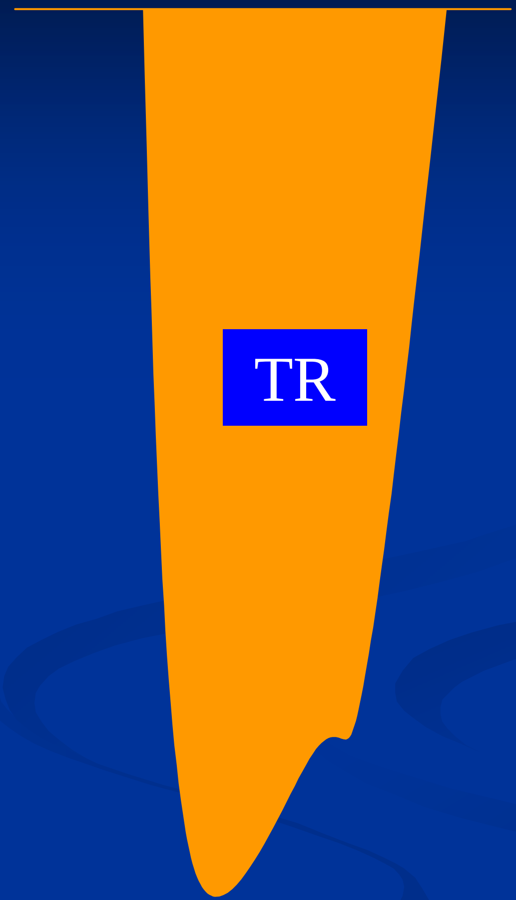


$P2 - P1 = 4 \times V^2$

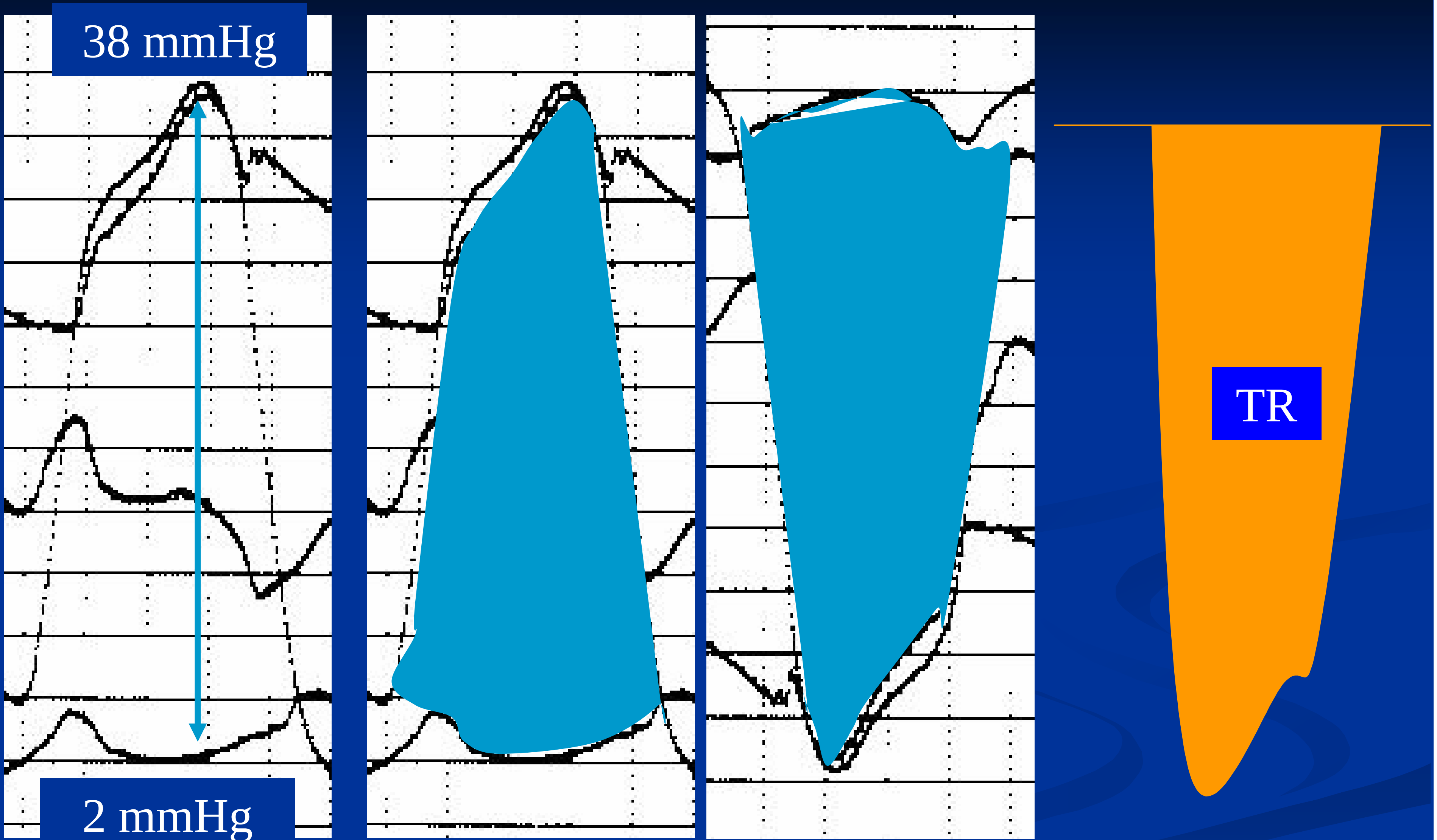
Insuffisance tricuspidienne

Vitesse = 3 m/s





Velocity= 3 m/s

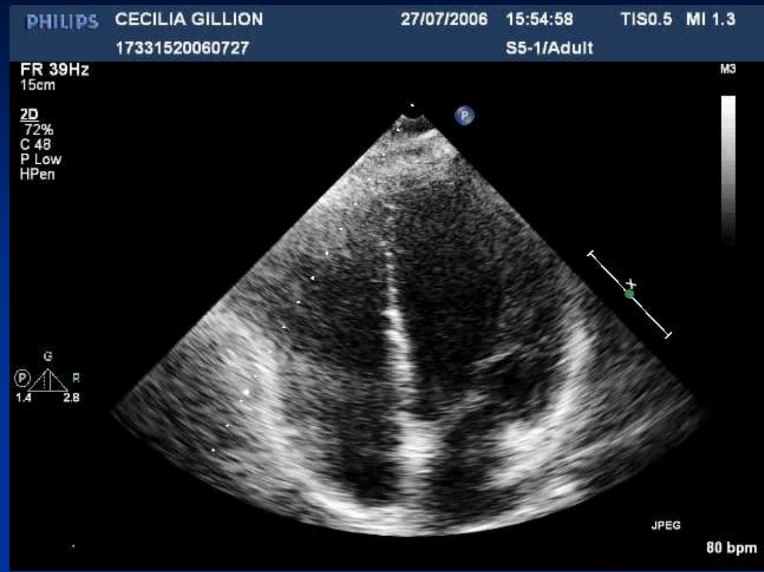


Gradient = 36 mmHg

dP = 4 V^2

Velocity= 3 m/s

Echocardiographie transthoracique


PHILIPS
CECILIA GILLION
17331520060727
27/07/2006
15:54:58
S5-1/Adult
TIS0.5 MI 1.3
FR 39Hz
15cm
2D
72%
C 48
P Low
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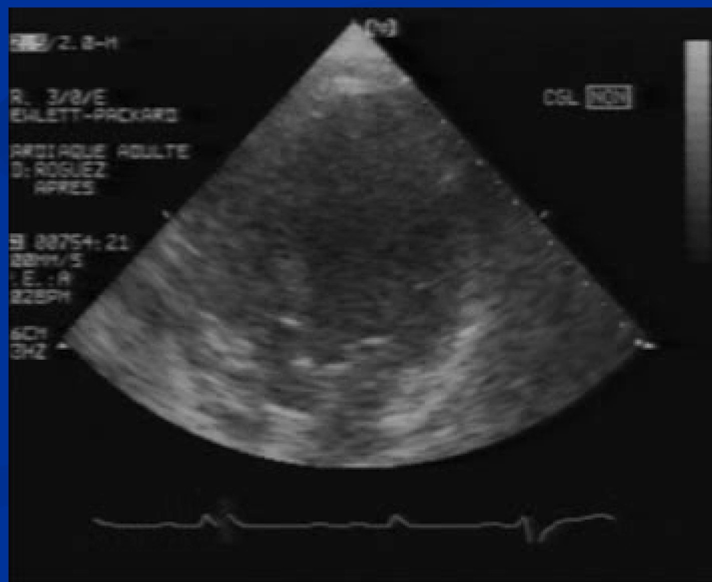
M3
JPEG
80 bpm



Echocardiographie Transoesophagienne

Technique en réanimation

- Patient intubé et ventilé
- Ne pas oublier de sédater
- Anesthésie locale
- Vacuité buccale
- A jeun ou vider le contenu gastrique
- Laisser la sonde gastrique
- Vérifier le déblocage de la sonde ETO
- Passage à l'aveugle sans forcer

Technique en réanimation

- Si passage difficile :
  - Mettre la main pour guider la sonde
  - Faire passer a droite ou à gauche de la sonde d'intubation
  - Flexion de la tête
  - Dégonfler légèrement le ballonnet de la sonde d'intubation
  - Enlever la sonde gastrique
  - Utiliser le laryngoscope

Respecter les contre-indications absolues

- Tumeur ORL ou oesophagienne
- Diverticule oesophagien
- Chirurgie récente oesophagienne
- Dysphagie non explorée
- Fracture cervicale non fixée (y penser en cas de poly traumatisme)
- Radiothérapie thoracique
- Poids < 45 kg pour une sonde multi plan adulte

Respecter les contre-indications relatives ou temporaires

- Hypoplaquettose : discuter en fonction de l'apport attendu
- Patient instable non intubé (OAP, choc)
- Lésion hémorragique oesophagienne voire gastrique
- Estomac plein
- Attention : anévrysmes aortique, gros thrombus OG ou OD

Complications de l'ETO

- Ambulatoire :
  - Mortalité : 0,0098 (10218 patients, Daniel Circulation 1991)- 0,026 (3827 patients, Seward JASE 1992)
  - Perforation oesophagienne : quelques cas (2 Amiens) entre des mains expertes.
  - Complications majeures : bronchospasme, tachycardie ventriculaire, insuffisance cardiaque : 0,2% (Seward JASE 1992;5:288) à 0,5 (Khandheria JACC 1991)
  - Complications mineures : 2,7% (Seward JASE 1992;5:288) : dysphagie…

Complications de l'ETO

- Réanimation
  - 20% saignements mineurs de la cavité buccale
  - 4-5% de complications (Slama, Oh, Pearson)
  - Perforation oesophagienne (petite taille, radiothérapie).
  - Embolie périphérique (anévrysme aortique, thrombus de l'OG).
  - Fractures cervicales.
  - Autres : tachycardie, détresse respiratoire, état de mal épileptique, vomissements, ischémie myocardique. Pas séquelles de ces complications.
  - Replacer la sonde naso-gastrique!!

Echec d'introduction

Mais ETO parfois difficile voire impossible dans 2-5% des cas : cause la plus fréquente inexpérience du manipulateur, mais aussi du à une anomalie anatomique (Daniel, Slama)


Xylocaïne Visqueuse 2%

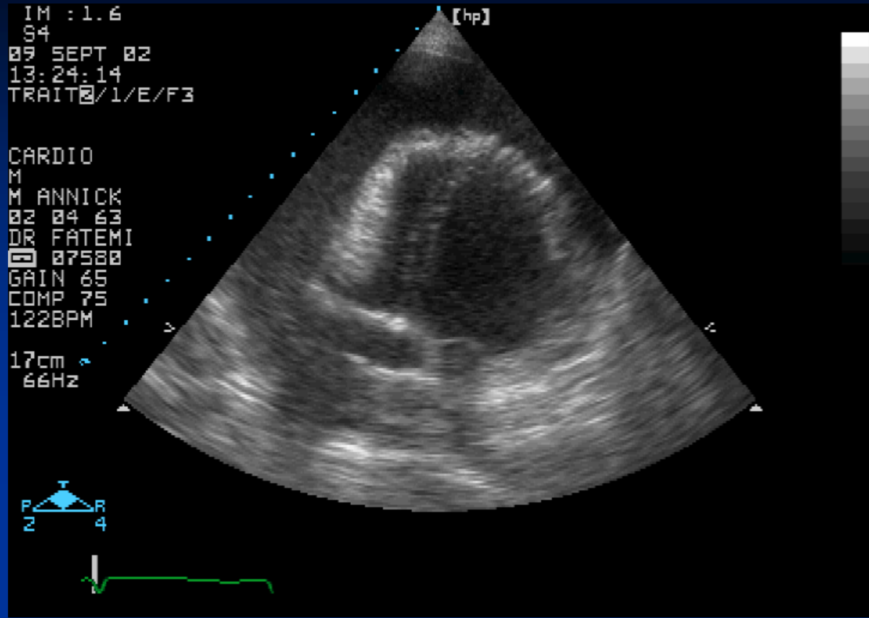
Indications

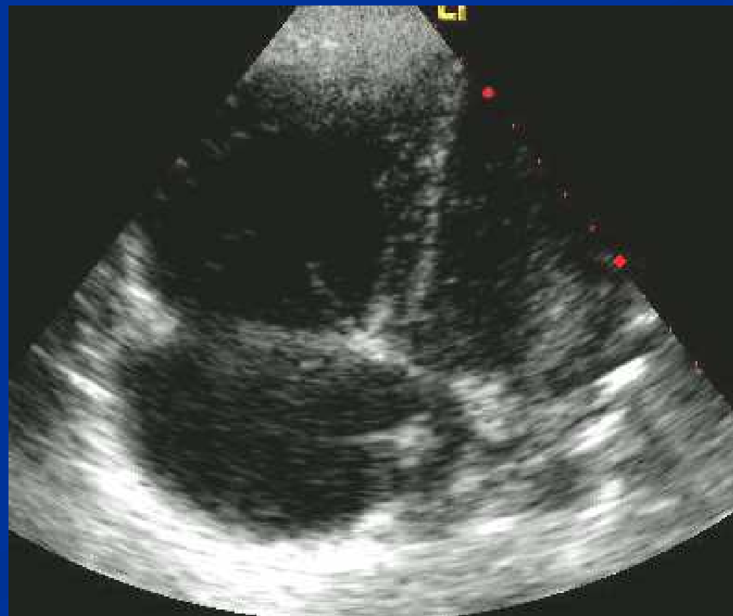
- Etat de choc
- SDRA et OAP
- Endocardites
- Traumatisme thoracique
- Suspicion de dissection aortique
- Etat de choc inexpliqué après chirurgie cardiaque
- OAP inexpliqué après chirurgie cardiaque
- Suspicion de dysfonction de prothèse

Impact thérapeutique de l'ETO

Auteurs (années)	Patients (n)	Impact thérapeutique (%)
Oh (1990)	51	24
Vignon (1994)	96	44
Heidenreich (1995)	61	68
Poelaert (1995)	108	43
Sohn (1995)	124	52
Slama (1996)	61	20
Colreavy (2002)	255	34

Causes évidentes

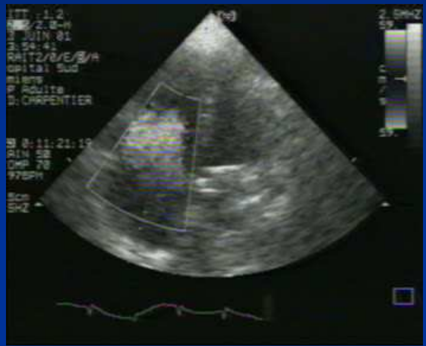

IM : 1.6
S4
09 SEPT 02
13:24:14
TRAIT2/1/E/F3
CARDIO
M
M ANNICK
02 04 63
DR FATEMI
07580
GAIN 65
COMP 75
122BPM
17cm
66Hz
[hp]



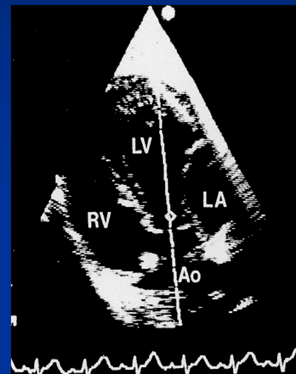
Echocardiographie et Doppler = hemodynamique

ECHOCARDIOGRAPHIE

Pressions droites

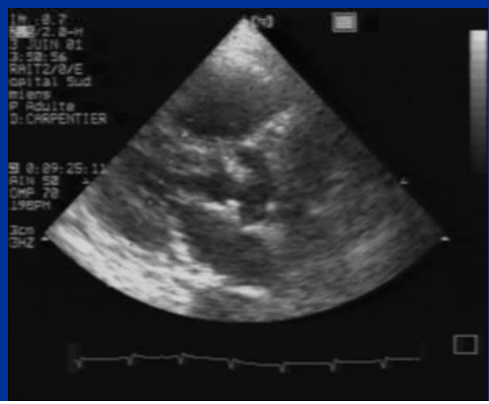


Débit cardiaque



Fonction VD

Fonction VG et la PAPO



Volémie

Dois je remplir mon patient?

critical care review

Predicting Fluid Responsiveness in ICU Patients*

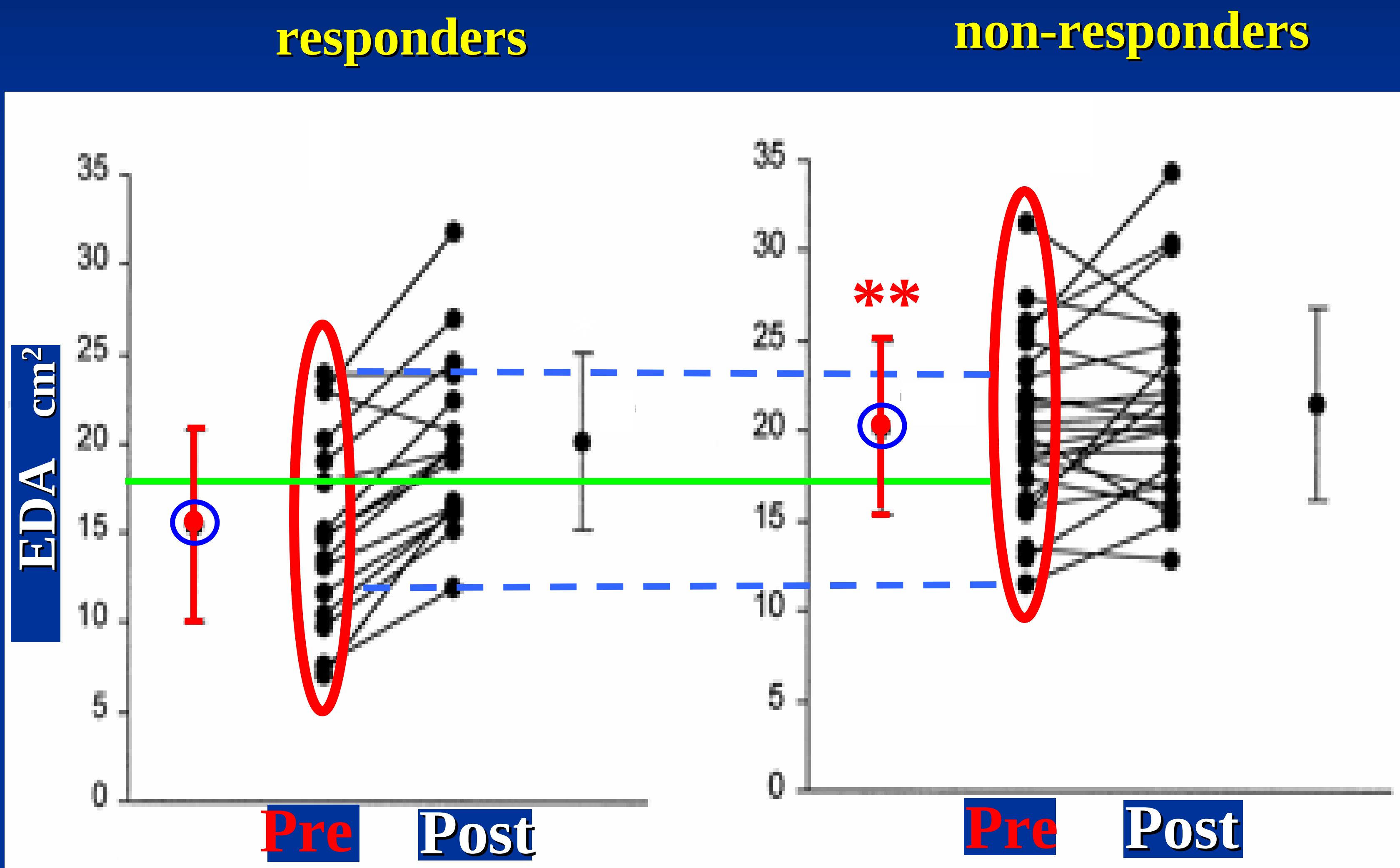
A Critical Analysis of the Evidence

Frédéric Michard, MD, PhD; and Jean-Louis Teboul, MD, PhD

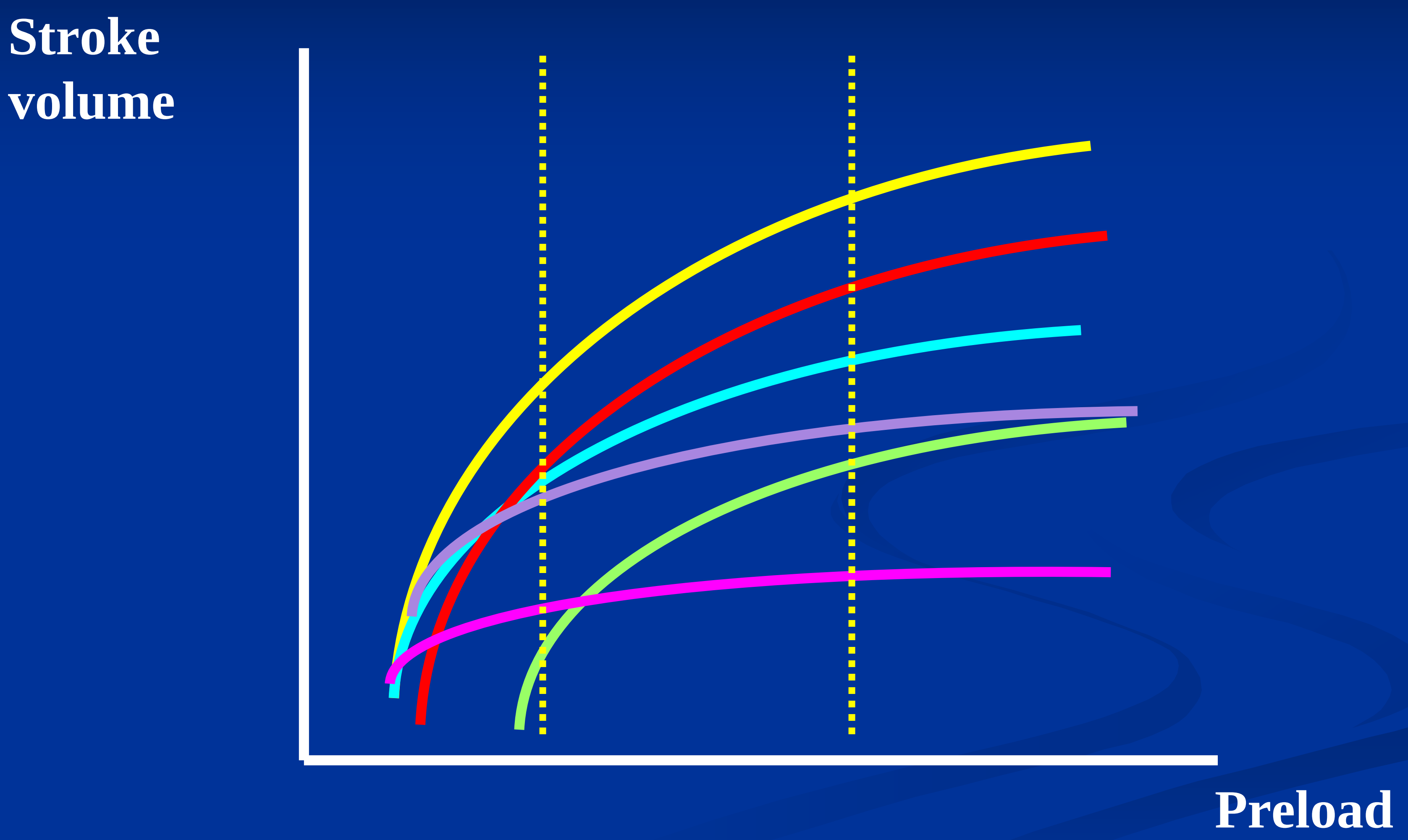
CHEST 2002, 121:2000-8

	R / NR	R (%)
Calvin (Surgery 81)	20 / 8	71 %
Schneider (Am Heart J 88)	13 / 5	72 %
Reuse (Chest 90)	26 / 15	63 %
Magder (J Crit Care 92)	17 / 16	52 %
Diebel (Arch Surgery 92)	13 / 9	59 %
Diebel (J Trauma 94)	26 / 39	40 %
Wagner (Chest 98)	20 / 16	56 %
Tavernier (Anesthesio 98)	21 / 14	60 %
Magder (J Crit Care 99)	13 / 16	45 %
Tousignant (A Analg 00)	16 / 24	40 %
Michard (AJRCCM 00)	16 / 24	40 %
Feissel (Chest 01)	10 / 9	53 %
Mean	**211 / 195**	**52 %**

The use of transesophageal echocardiography for preload assessment in critically ill patients. Tousignant CP, Walsh F, Mazer CD. Anesth Analg 2000;90:351-355

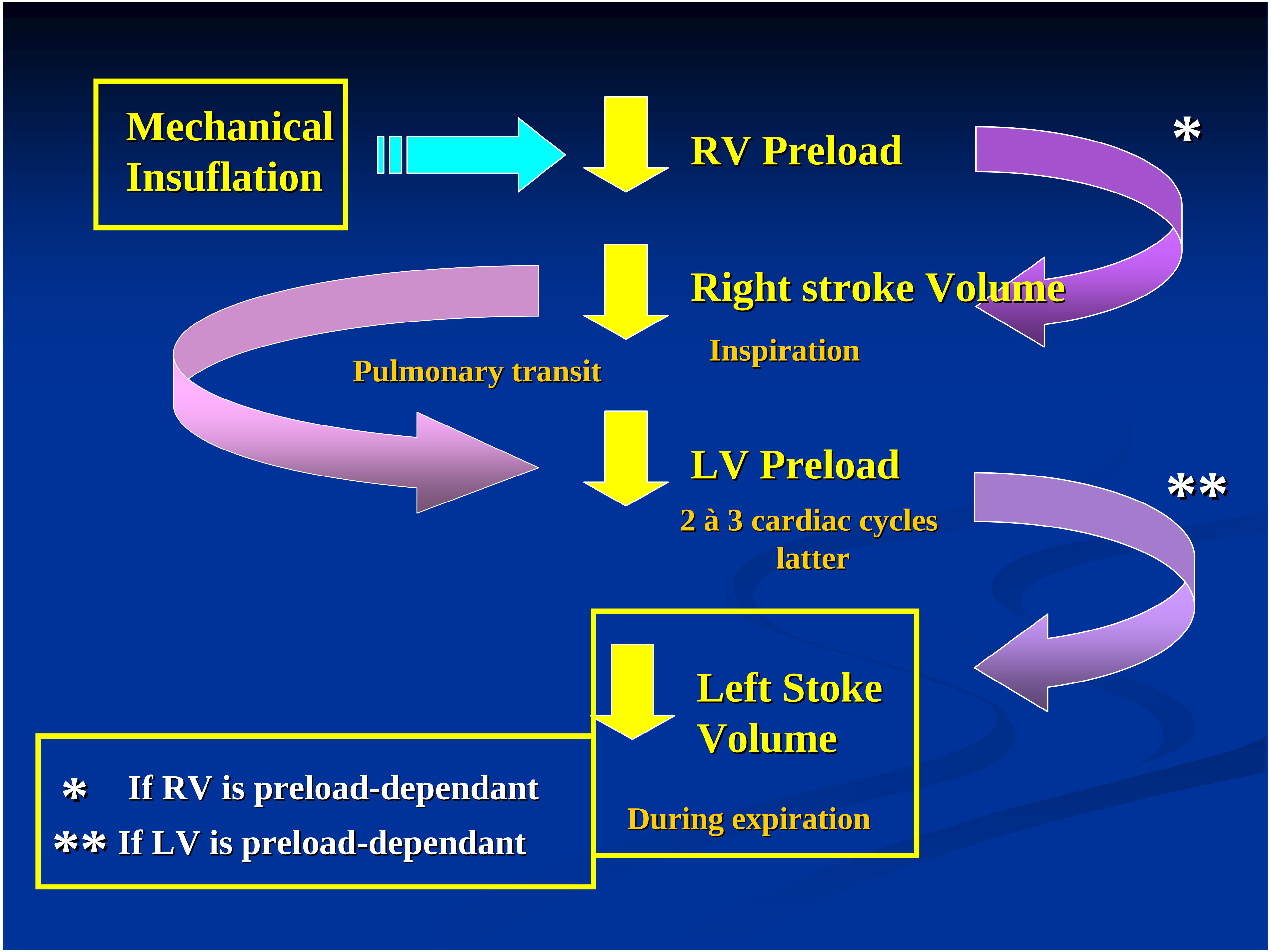


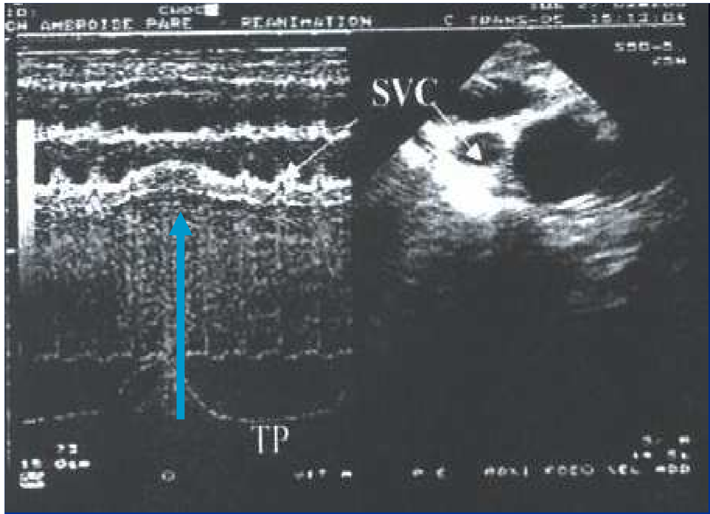
STARLING RELATIONSHIP



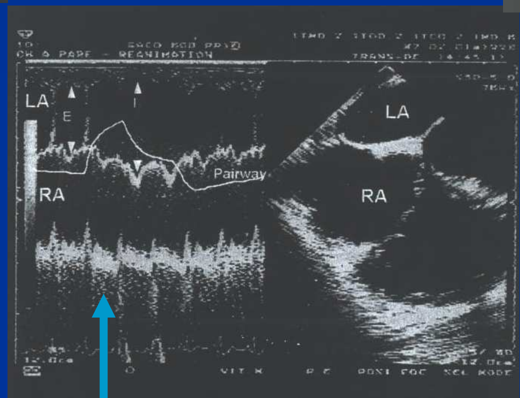
All indices of preload poorly predict fluid responsiveness !

DDB USI

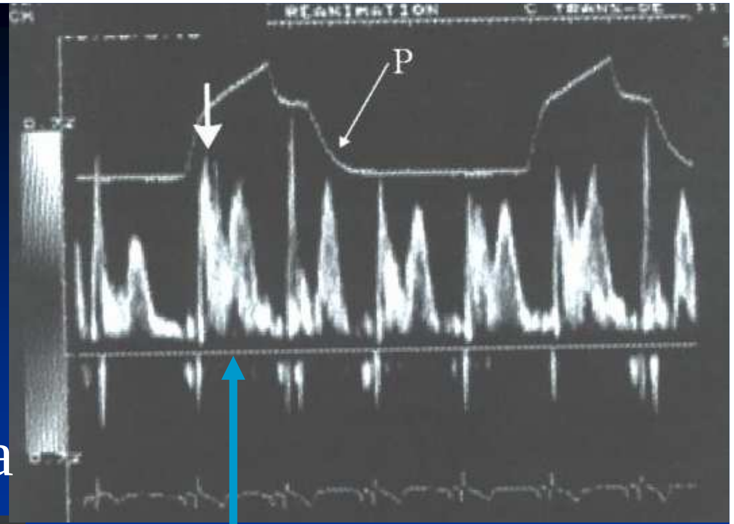

Mechanical Insuflation
RV Preload
*
Right stroke Volume
Inspiration
Pulmonary transit
LV Preload
**
2 à 3 cardiac cycles latter
Left Stoke Volume
During expiration
* If RV is preload-dependant
** If LV is preload-dependant



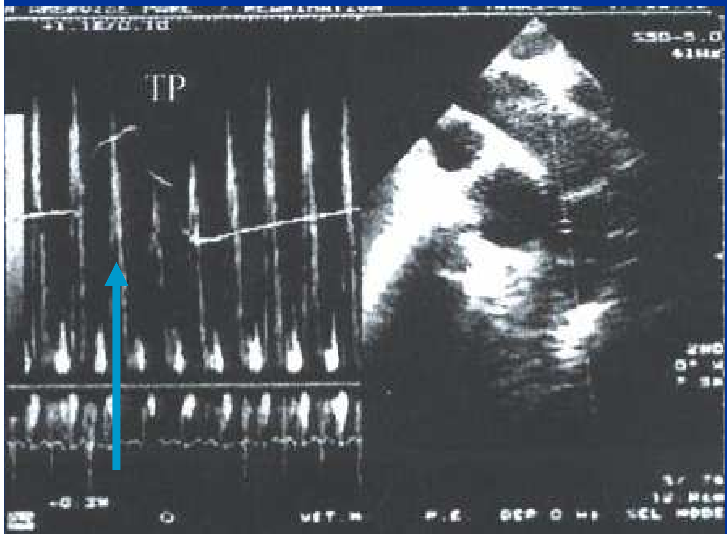
Superior vena cava



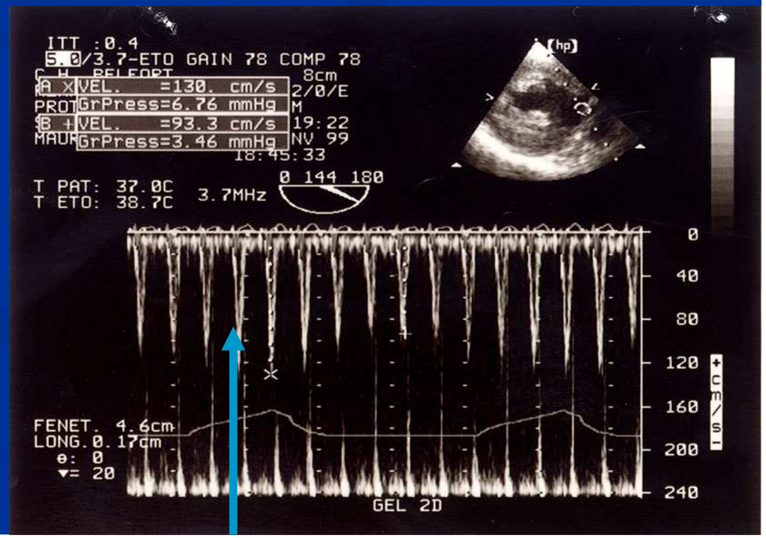
Right and left atria



Mitral flow

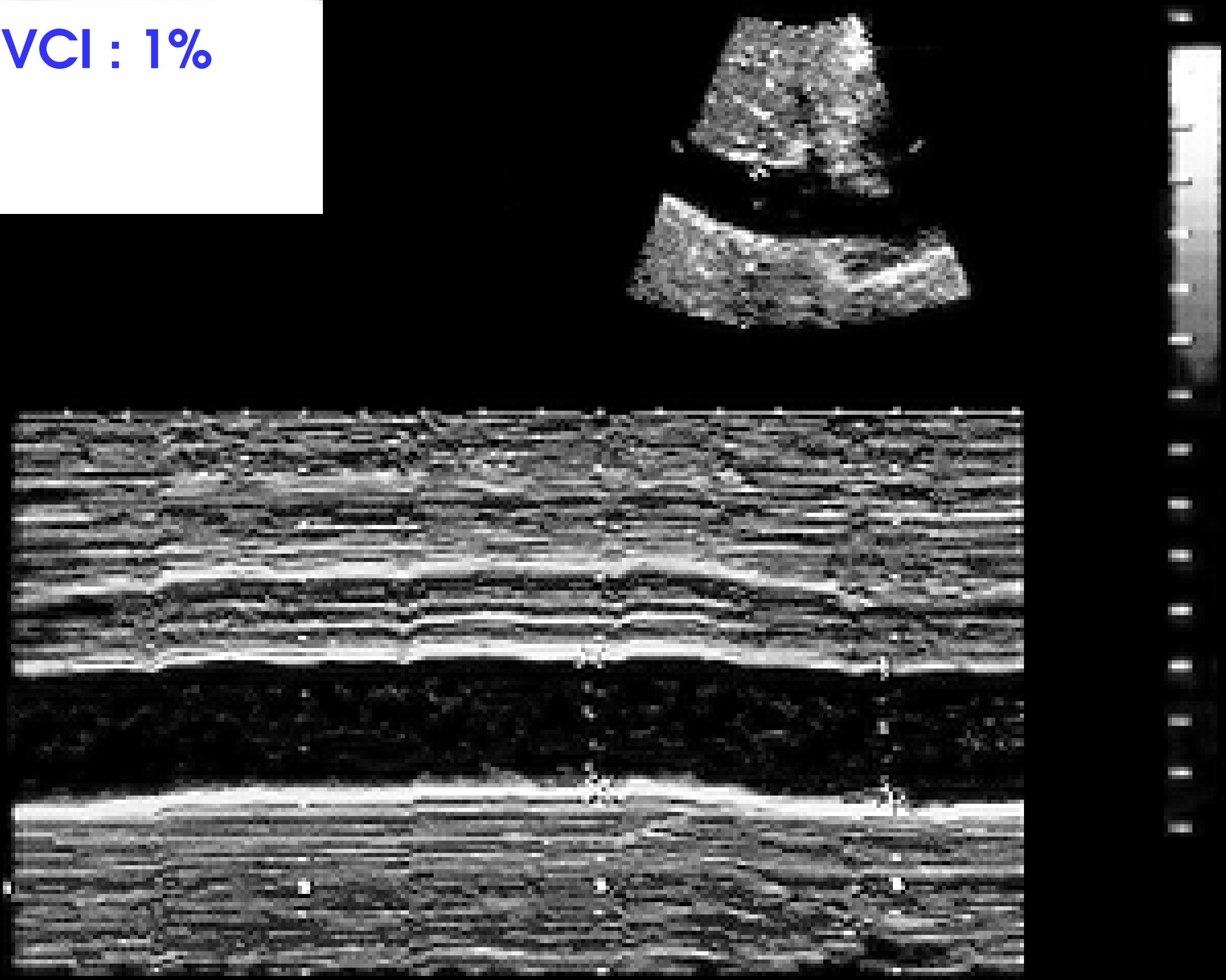


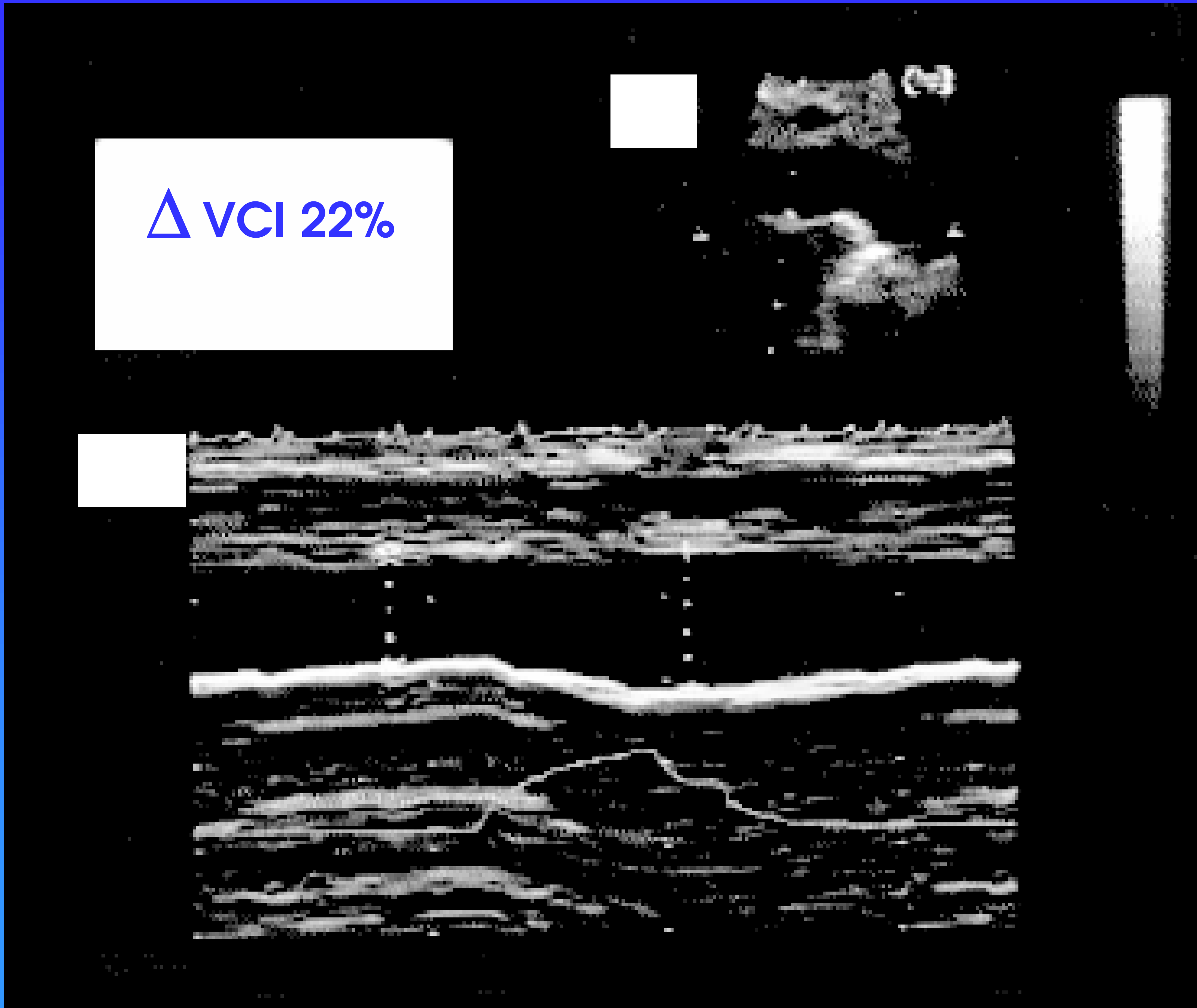
Pulmonary arterial flow

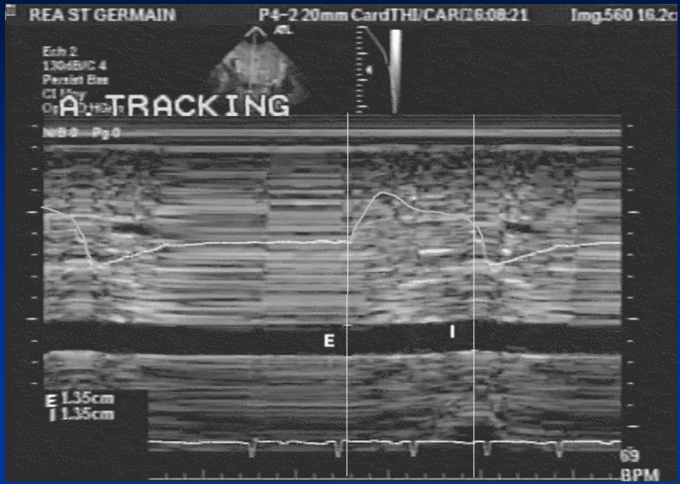
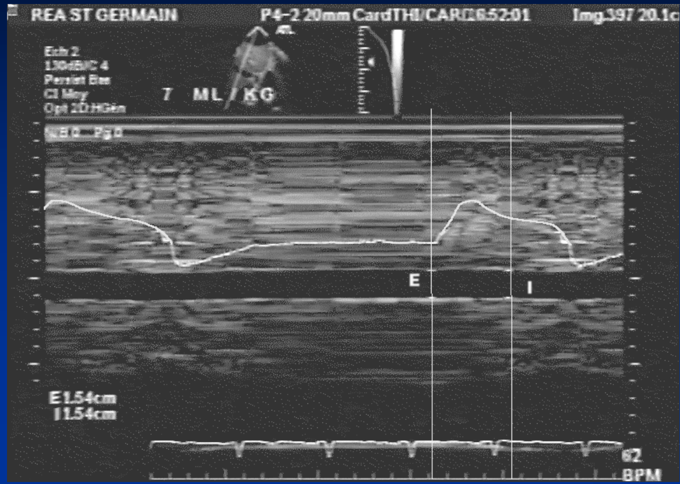
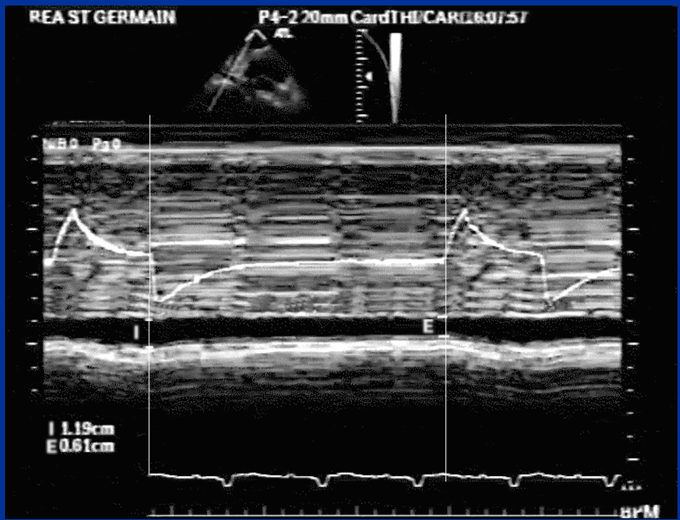
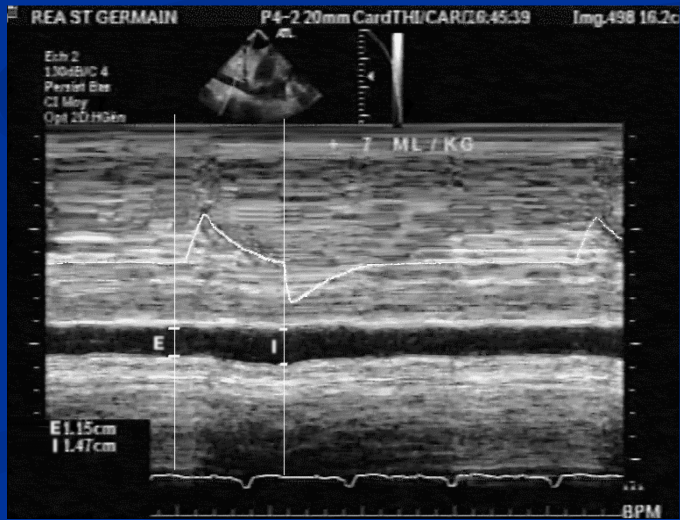


Aortic flow

Δ VCI : 1%



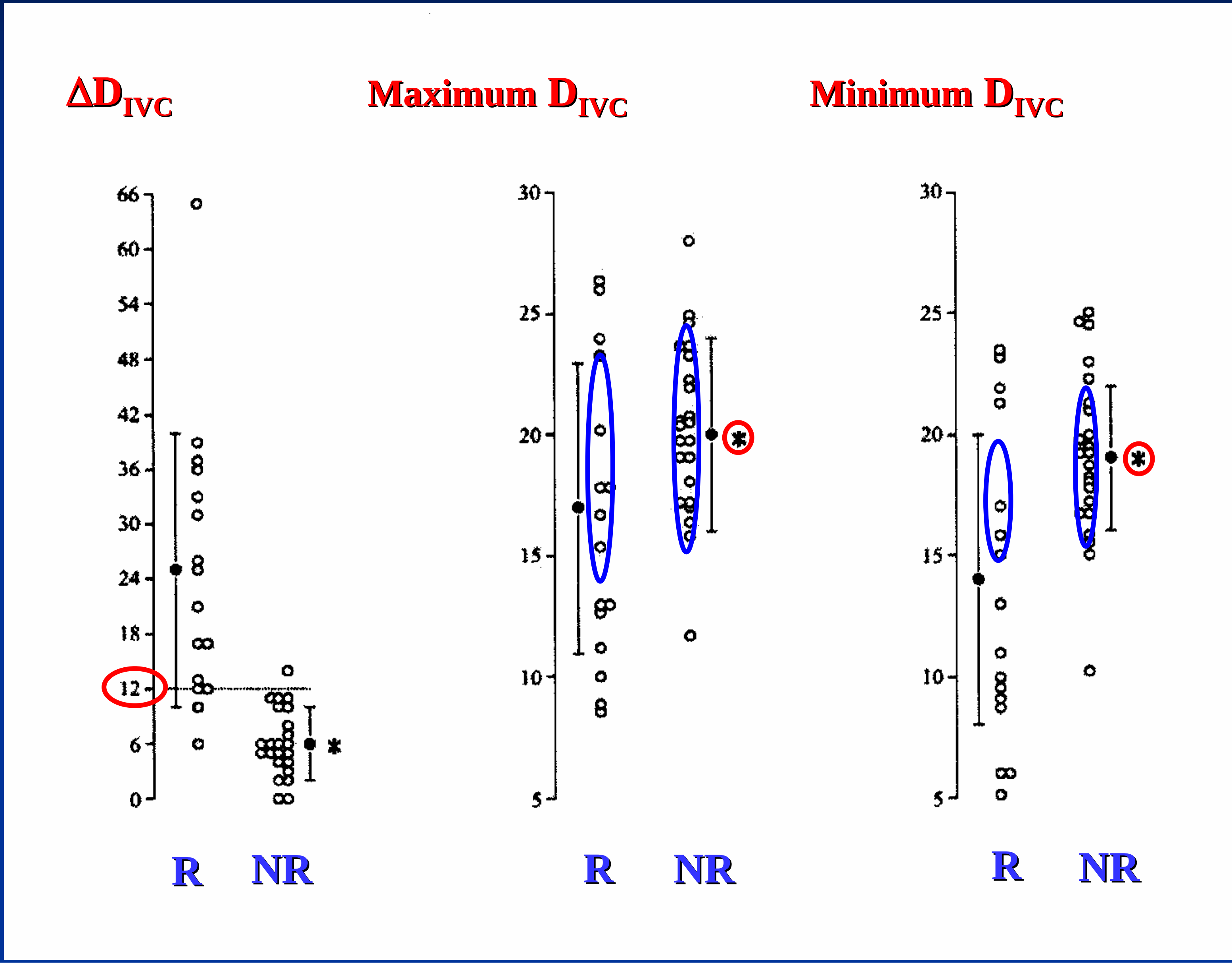

Δ VCI 22%

	Baseline	After blood volume expansion
A		
	dIVC = 0 % CI = 2.3 L/min/m^2	dIVC = 0 % CI = 2.3 L/min/m^2
B		
	dIVC = 95 % CI = 1.8 L/min/m^2	dIVC = 28 % CI = 2.6 L/min/m^2

The respiratory variation of inferior vena cava diameter as a a guide to fluid therapy

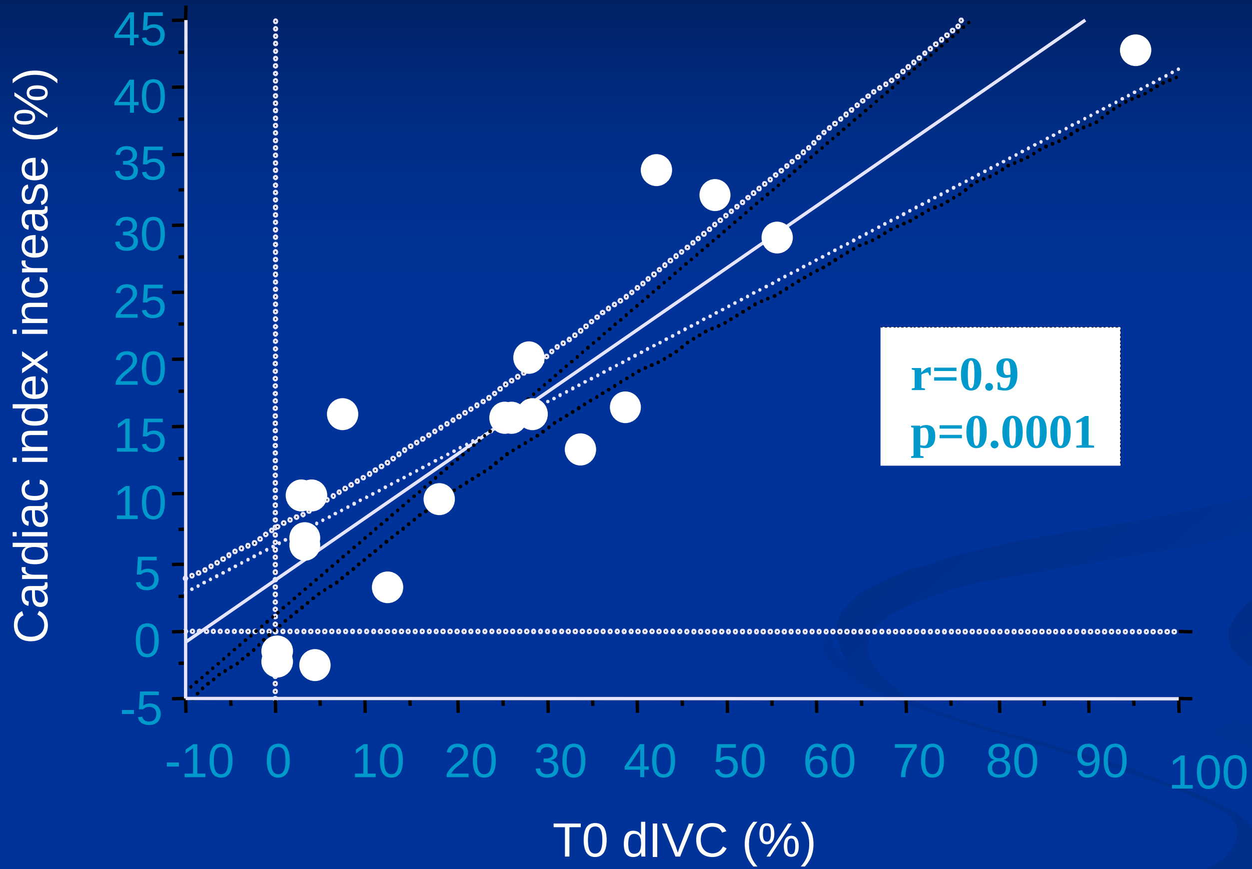
Feissel M, Michard F, Faller JP, Teboul JL

Intensive Care Med 2004, 30:1834-1837



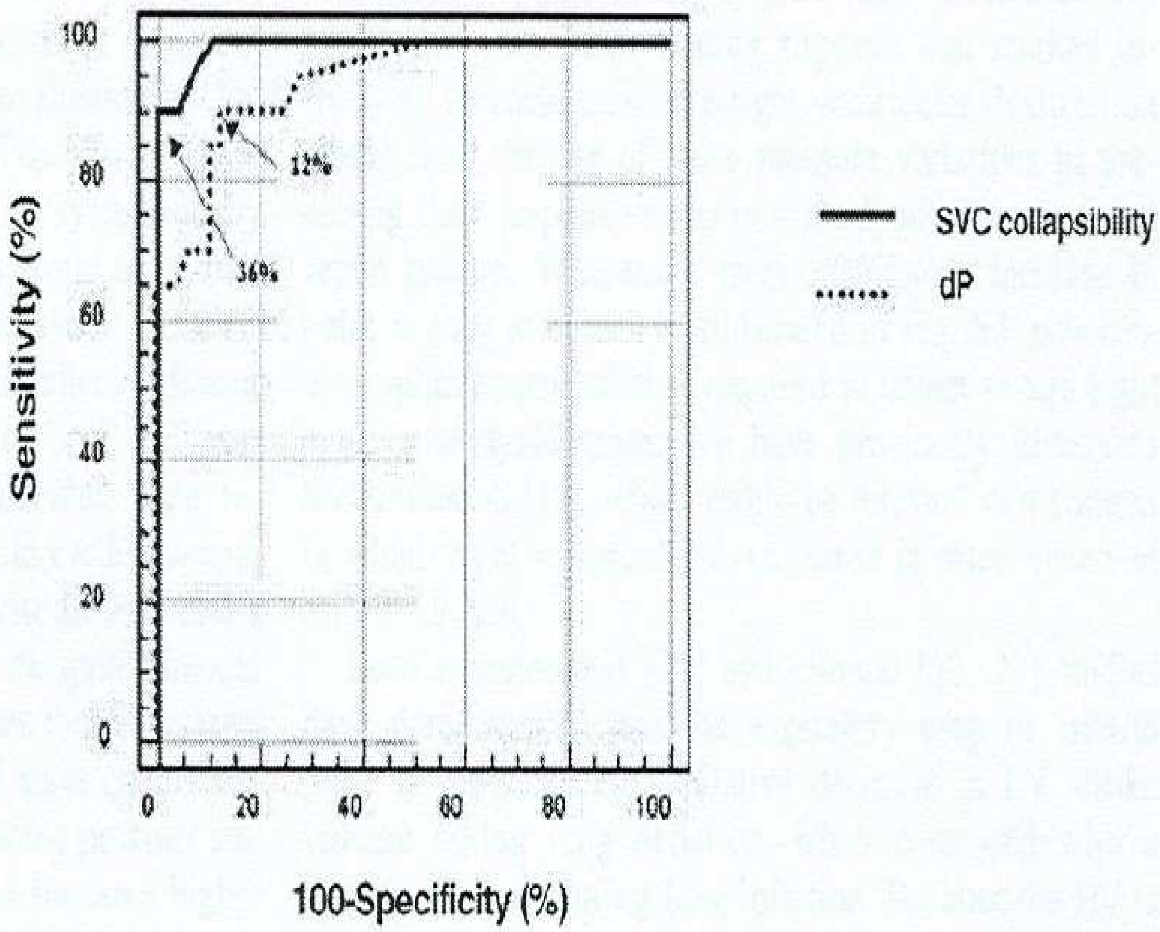
Superior Vena Cava

(A. Vieillard-Baron)



A Vieillard-Baron Int Care Med 2004

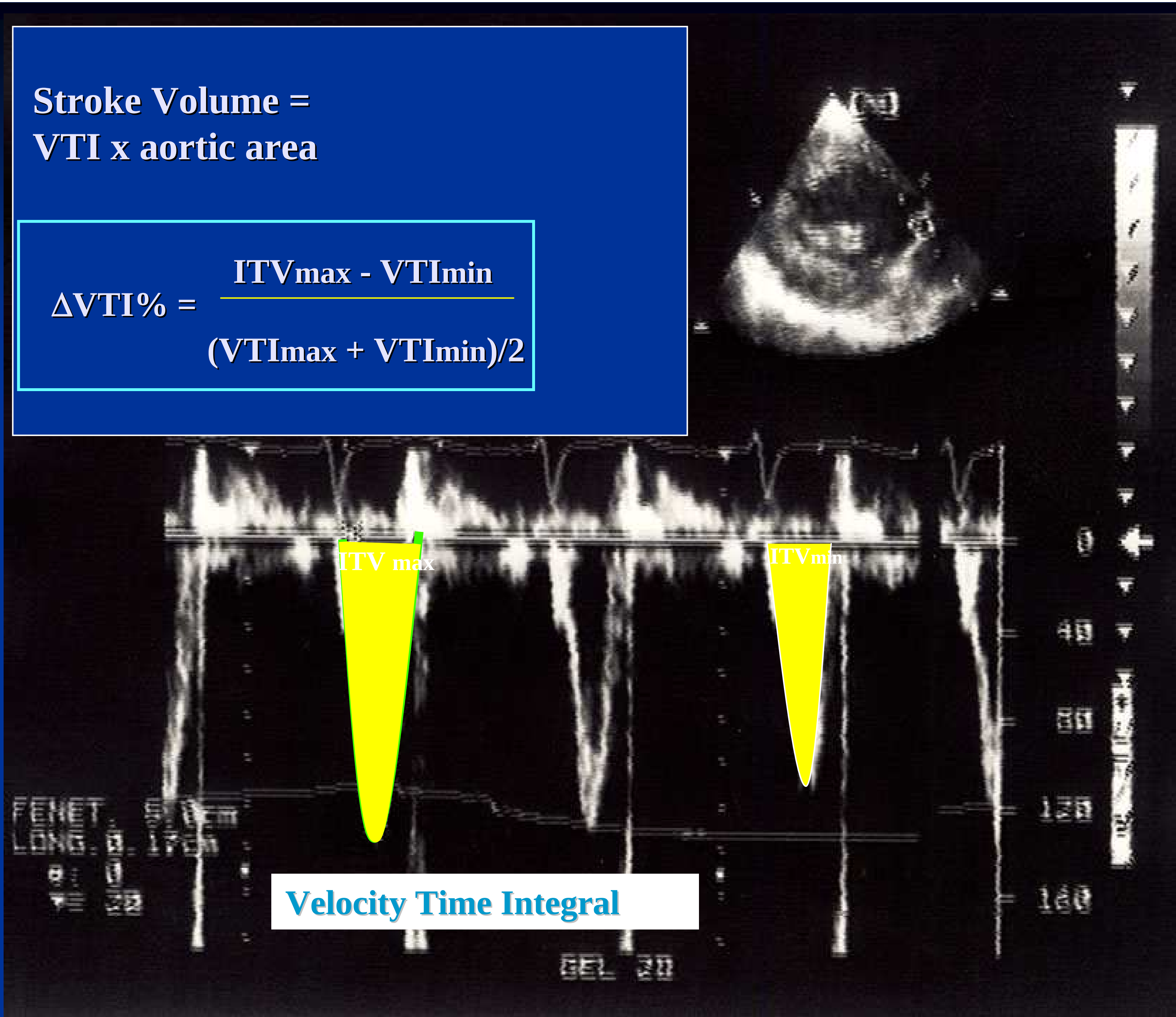
Fig. 3 ROC curves comparing the ability of SVC collapsibility and d*P* to discriminate responders (CI increase ≥11%) and nonresponders to VE. The area under the ROC curve for SVC collapsibility (0.993±0.013) did not differ significantly from the area under the ROC curve for d*P* (0.940±0.038; *P*=0.19) Threshold values for these parameters (*arrows*) are 36% and 12%, respectively



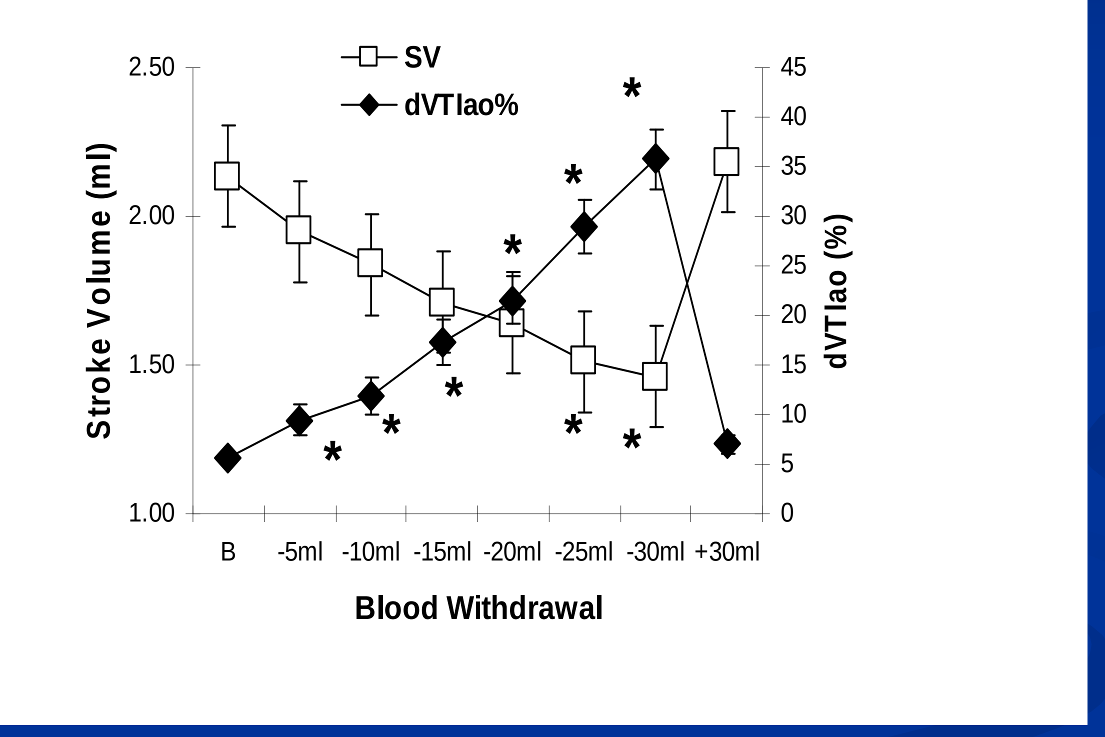
Vieillard-Baron A Anesthesiology 2001;95:1083-8

Stroke Volume =
VTI x aortic area

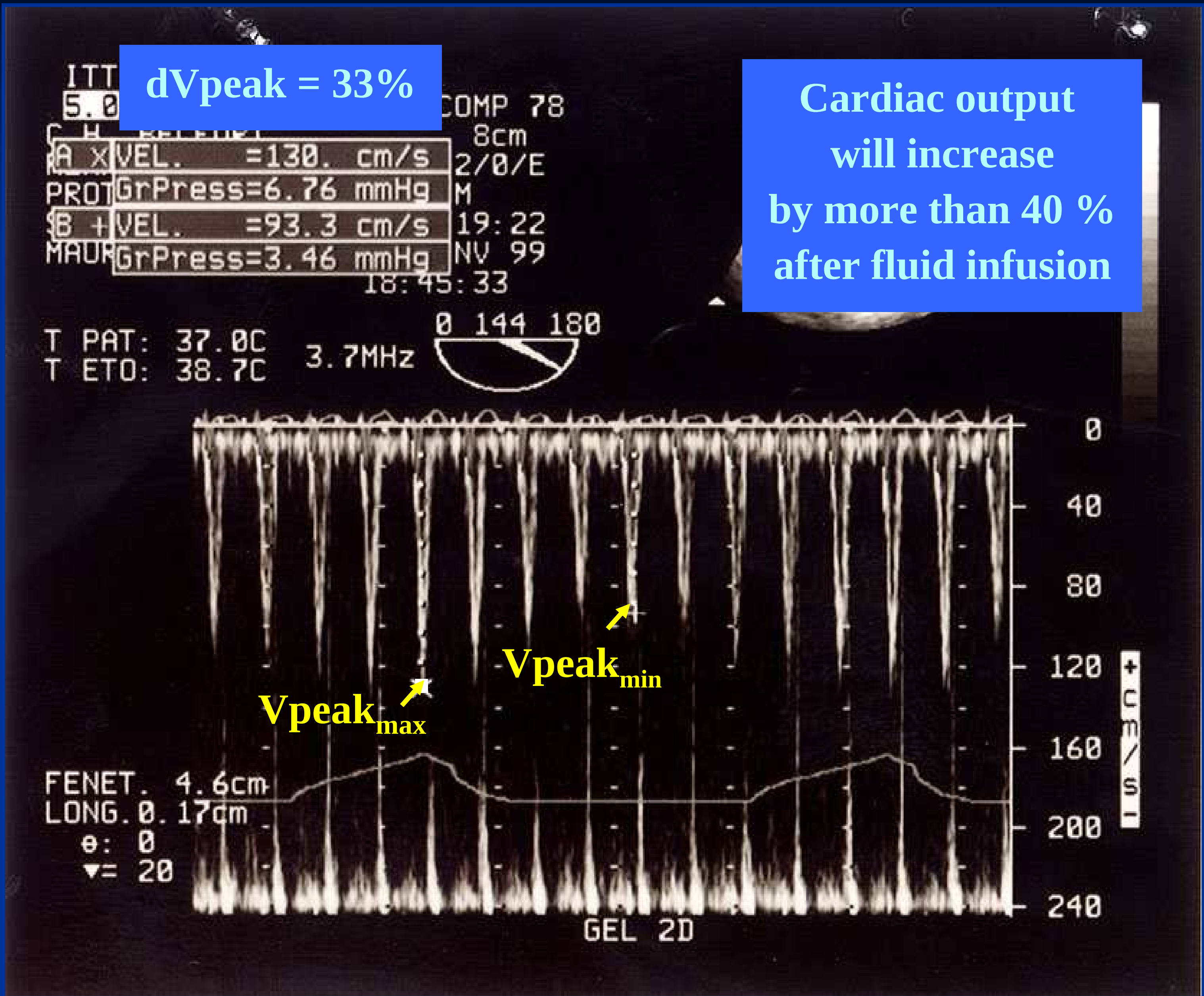
$$\Delta VTI\% = \frac{ITVmax - VTImin}{(VTImax + VTImin)/2}$$

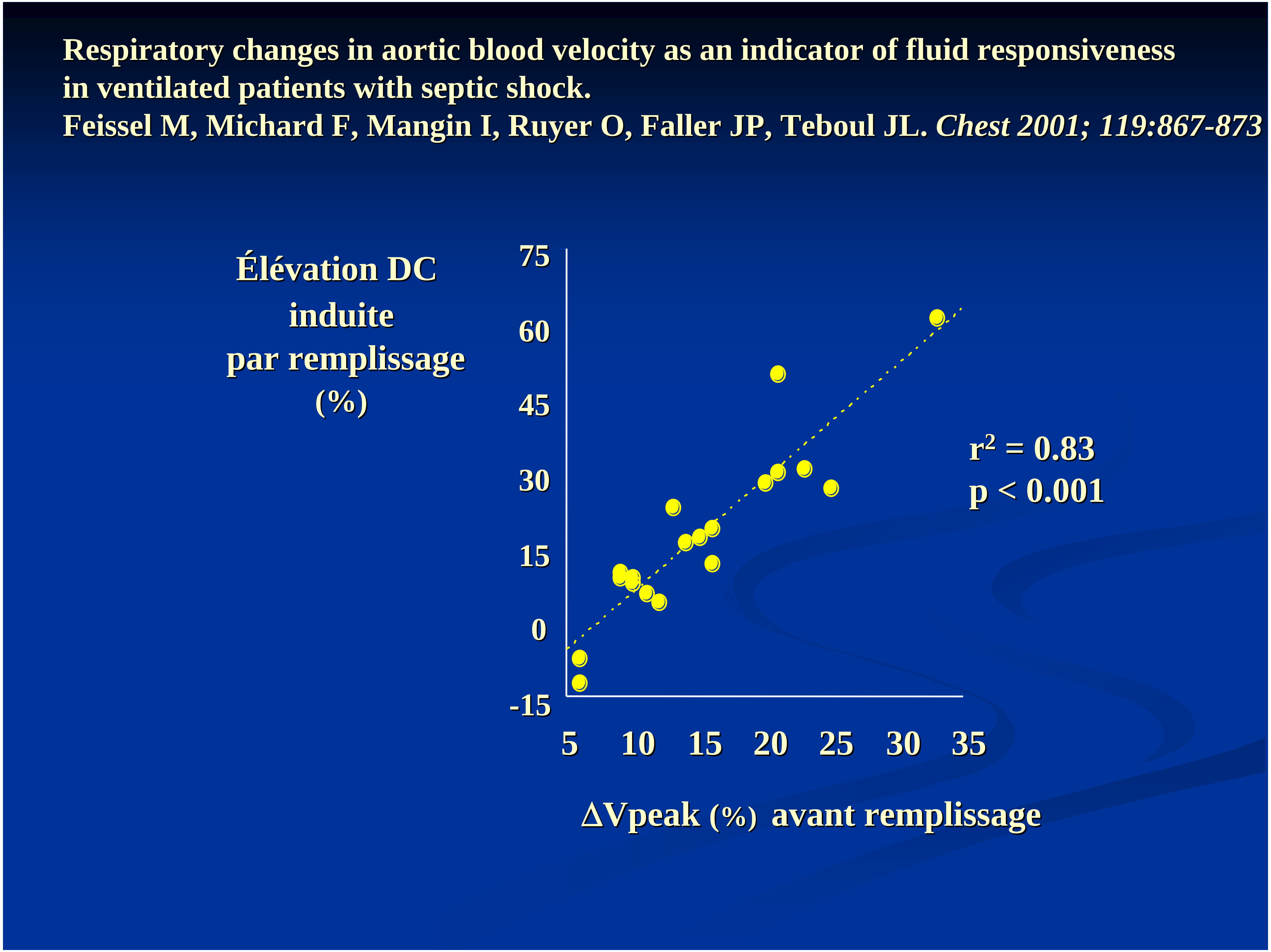


Cyclic Changes During Positive Pressure MV: indices of fluid responsiveness



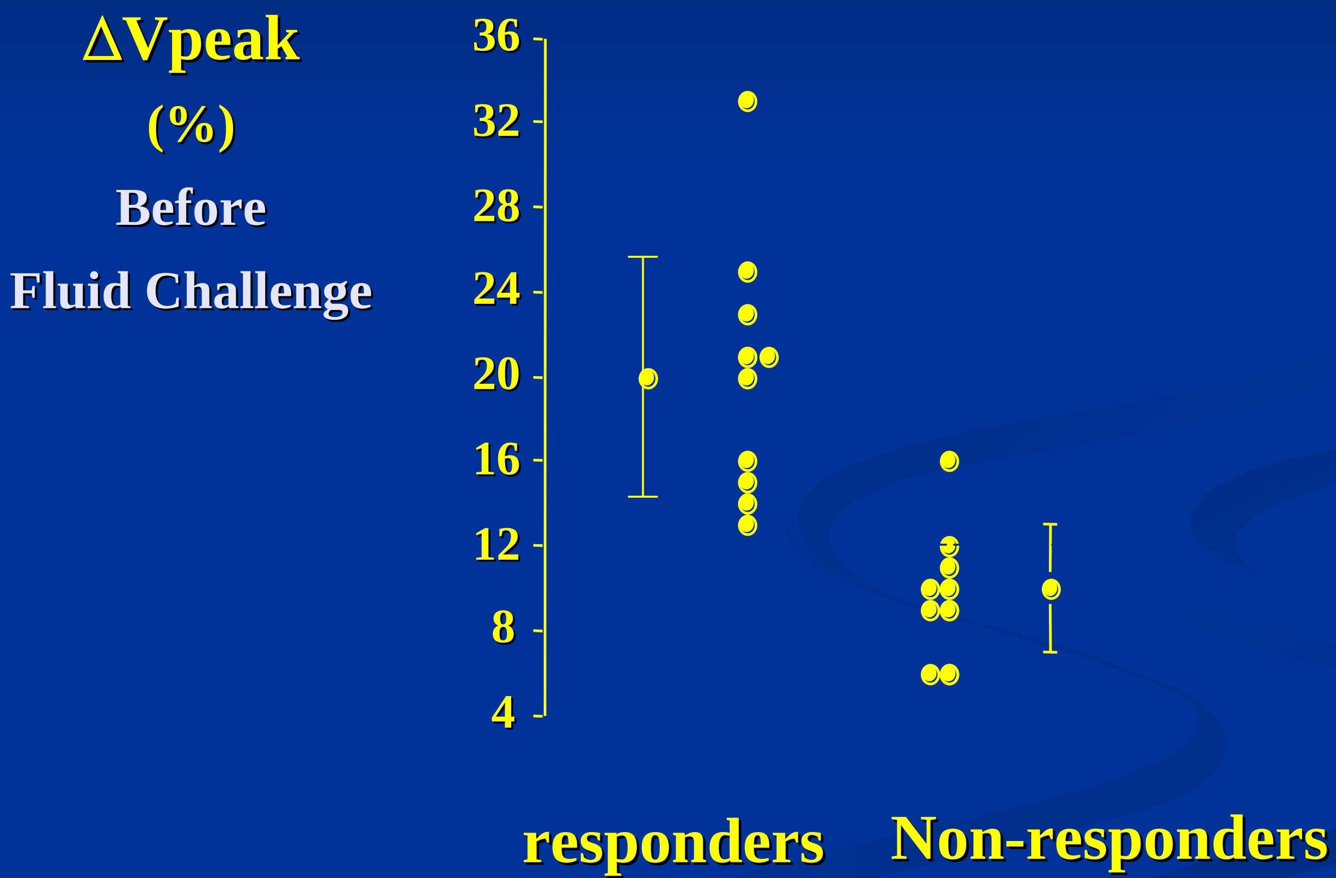
Slama M Am J Physiol 2002;283:H1729


dVpeak = 33%
Cardiac output
will increase
by more than 40 %
after fluid infusion
Vpeak max
Vpeak min
VEL. =130. cm/s
GrPress=6.76 mmHg
VEL. =93.3 cm/s
GrPress=3.46 mmHg
8cm
2/0/E
19:22
NV 99
18:45:33
T PAT: 37.0C
T ETO: 38.7C
3.7MHz
0 144 180
FENET. 4.6cm
LONG.0.17cm
0
40
80
120
160
200
240
cm/s
GEL 2D

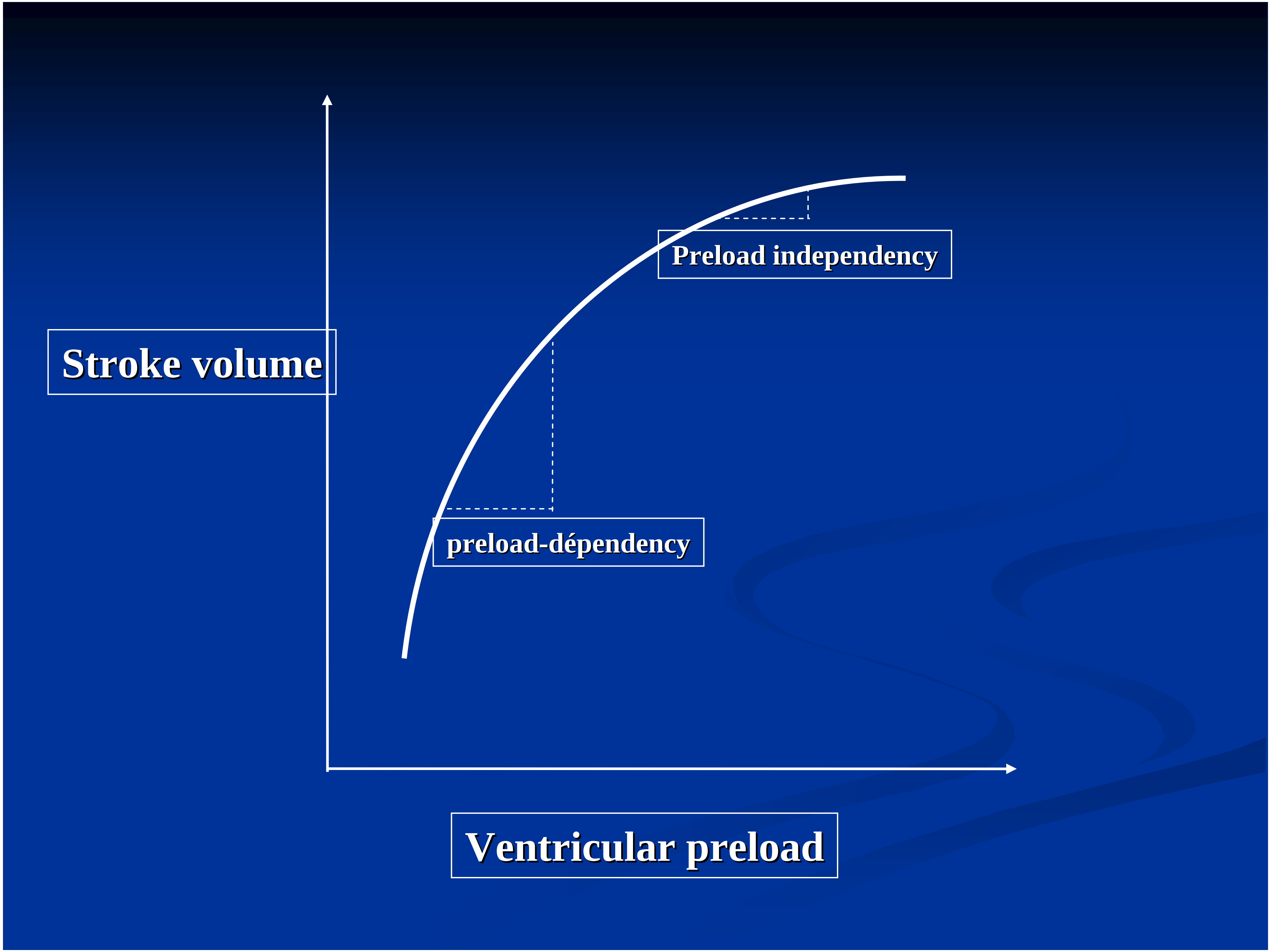

Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness
in ventilated patients with septic shock.
Feissel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. Chest 2001; 119:867-873
Élévation DC
induite
par remplissage
(%)
75
60
45
30
15
0
-15
5
10
15
20
25
30
35
r2 = 0.83
p < 0.001
ΔVpeak (%) avant remplissage

Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock.

Feissel F, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. ***Chest 2001; 119:867-873***

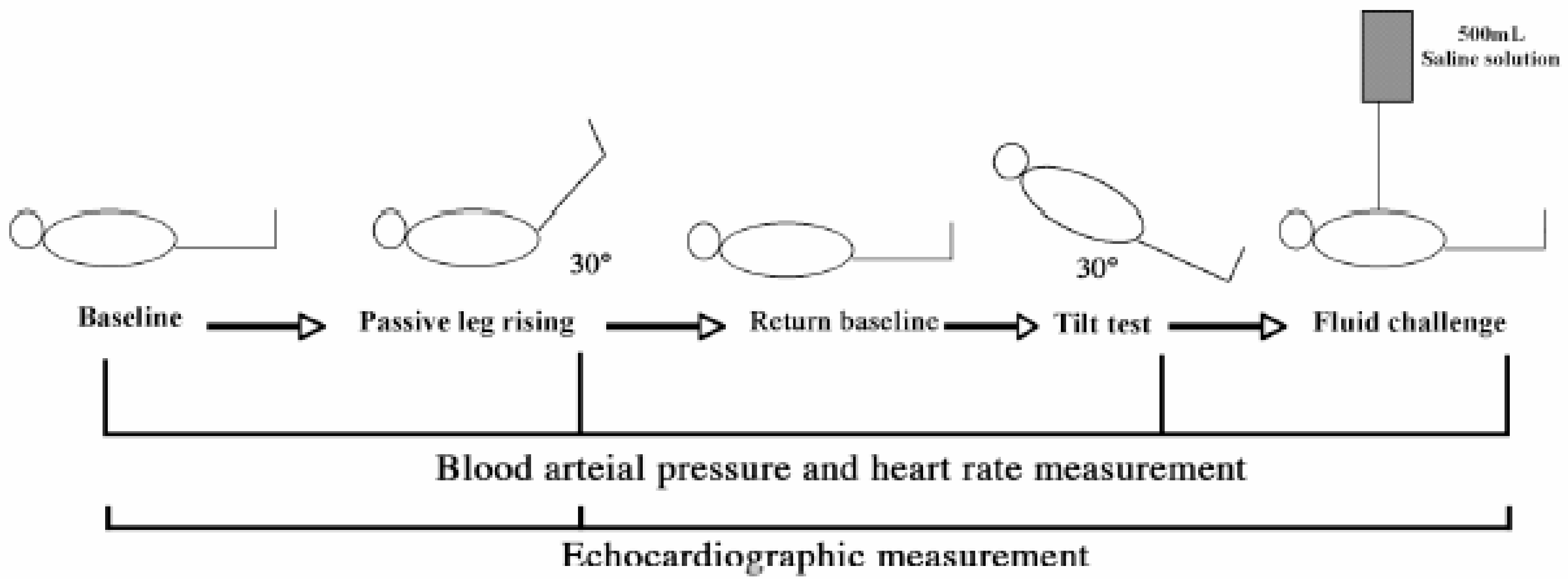


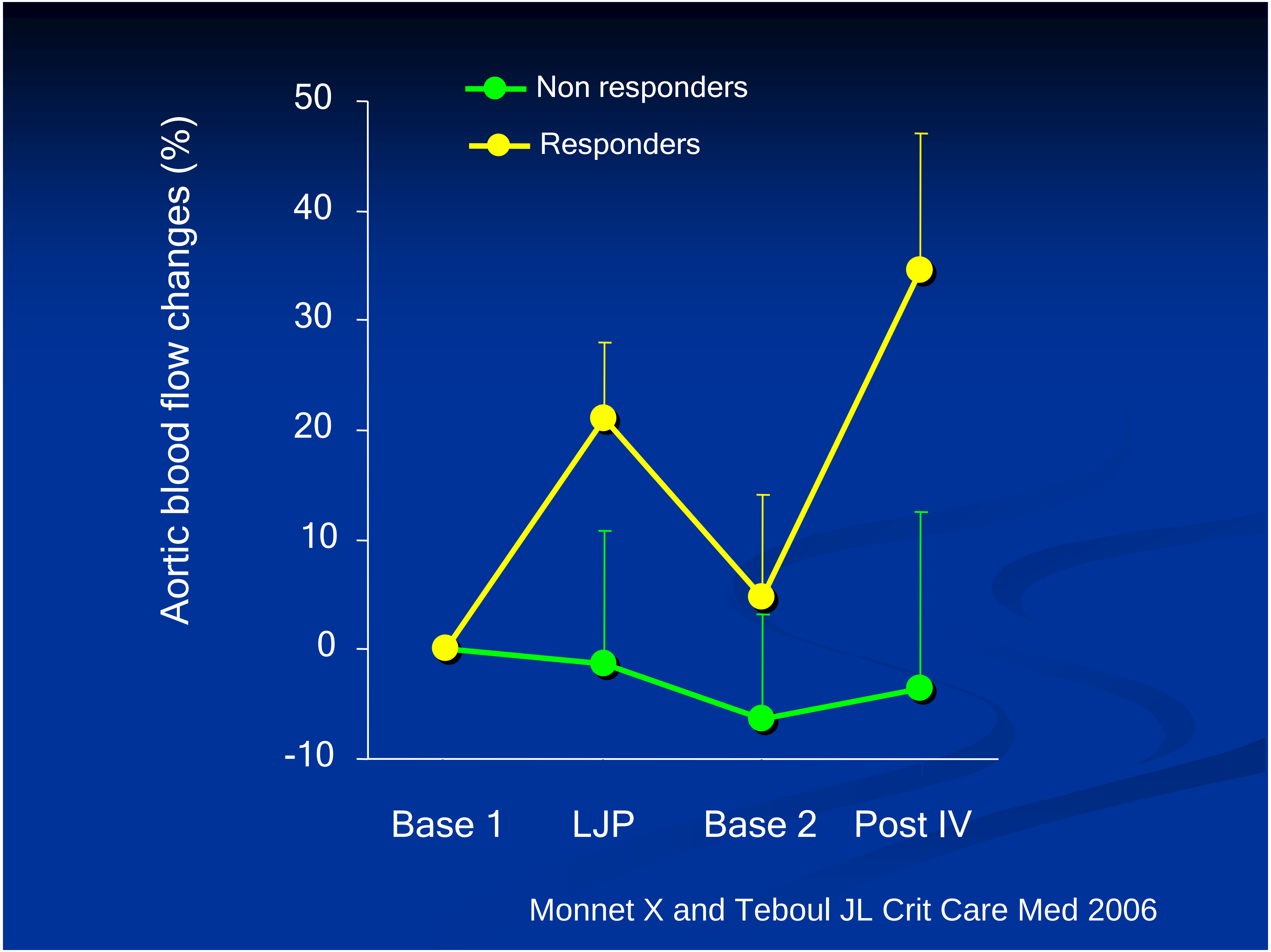
Spontaneously breathing patient

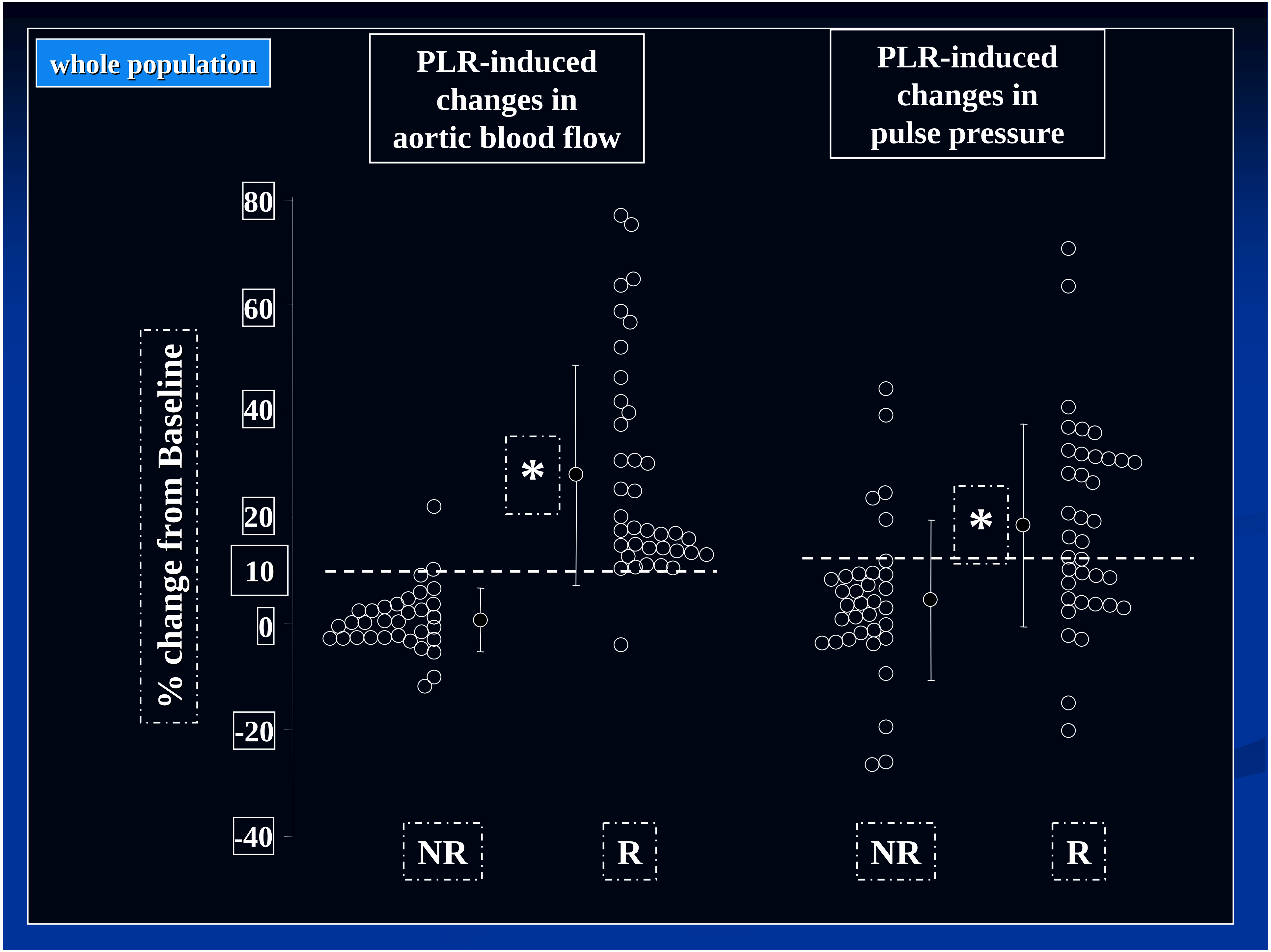


Stroke volume
Preload independency
preload-dépendency
Ventricular preload

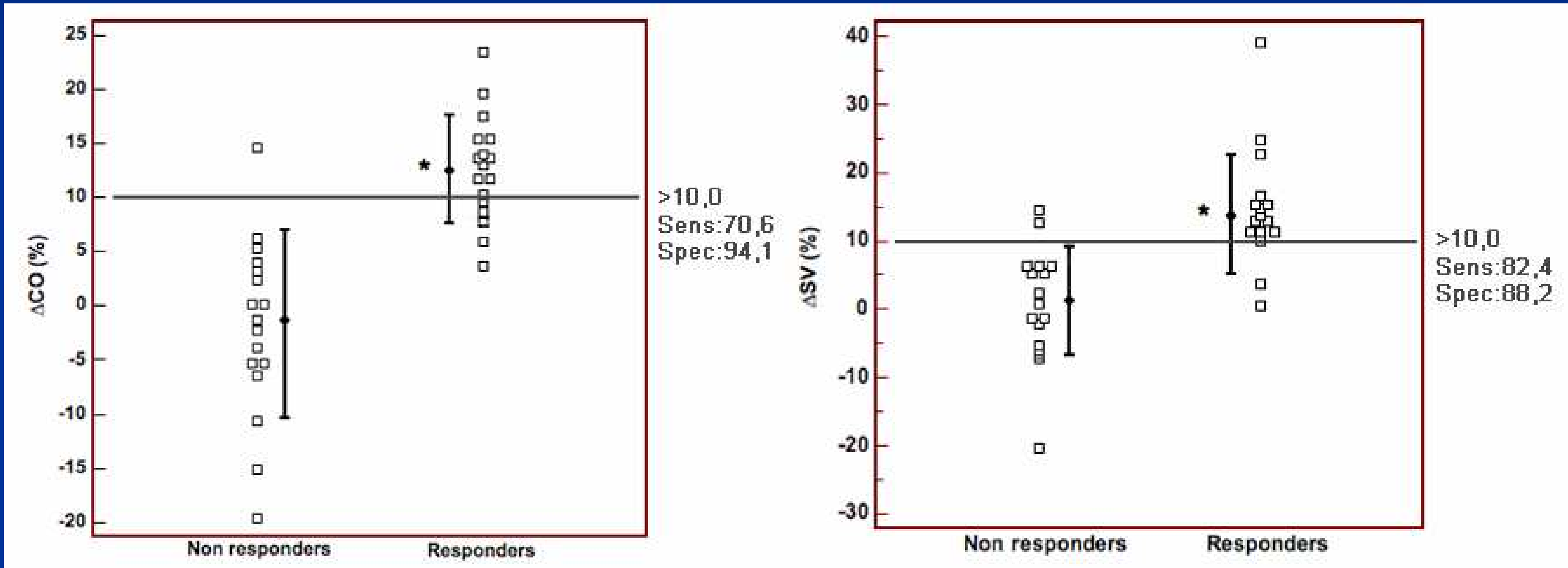
Passive Leg Raising




Non responders
Responders
Aortic blood flow changes (%)
50
40
30
20
10
0
-10
Base 1
LJP
Base 2
Post IV
Monnet X and Teboul JL Crit Care Med 2006


whole population
PLR-induced changes in aortic blood flow
PLR-induced changes in pulse pressure
% change from Baseline
80
60
40
20
10
0
-20
-40
*
*
NR
R
NR
R

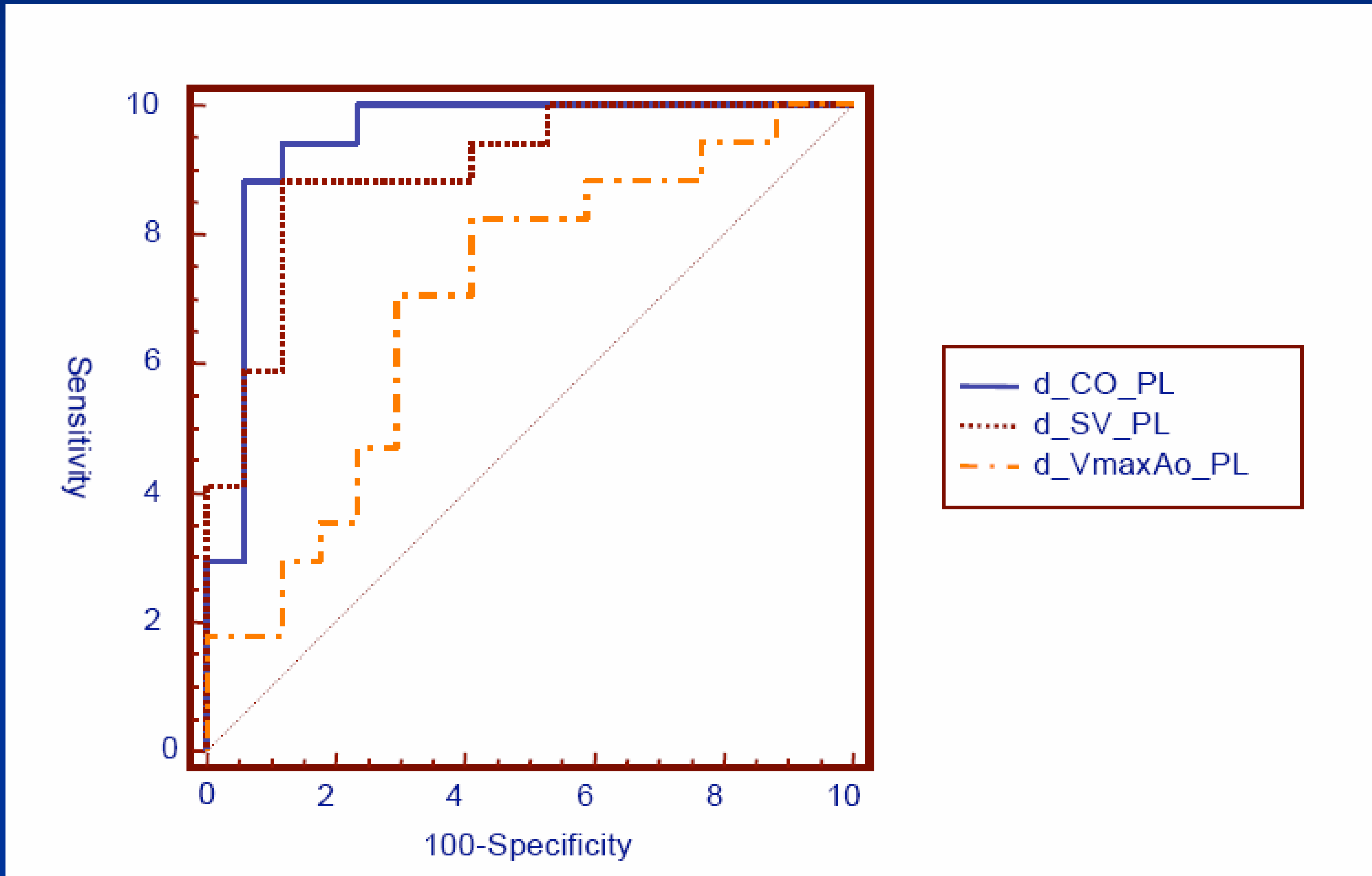
Passive Leg Raising



Increase in CO or SV by >10%

Maizel and Slama 2006 submitted

Passive Leg Raising

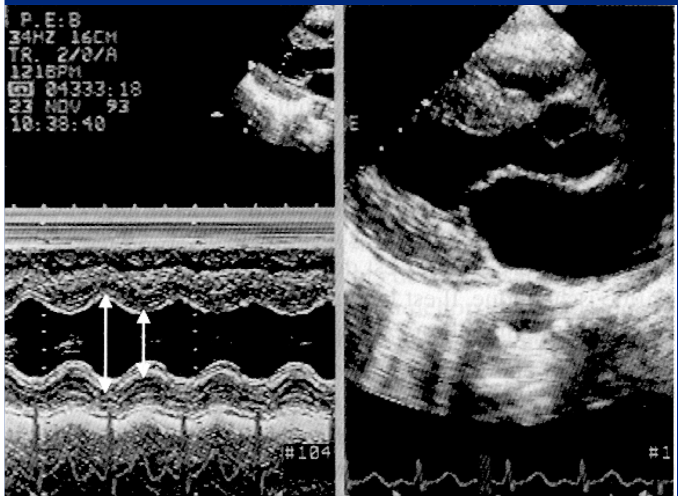


Maizel and Slama 2006 submitted

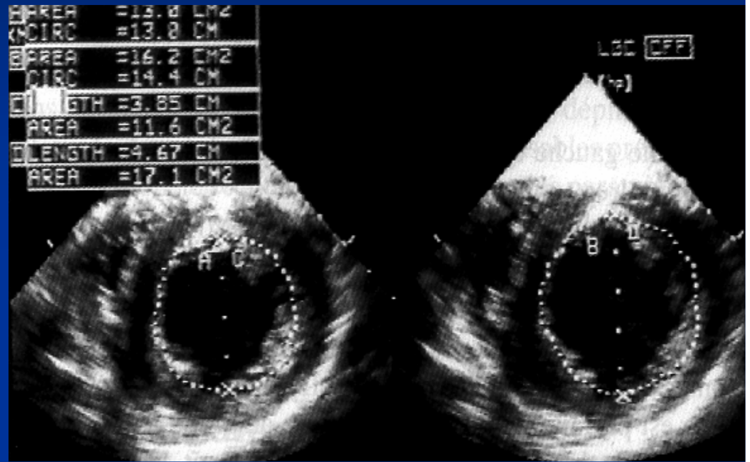
Dois je donner de la dobutamine?

Systolic Function

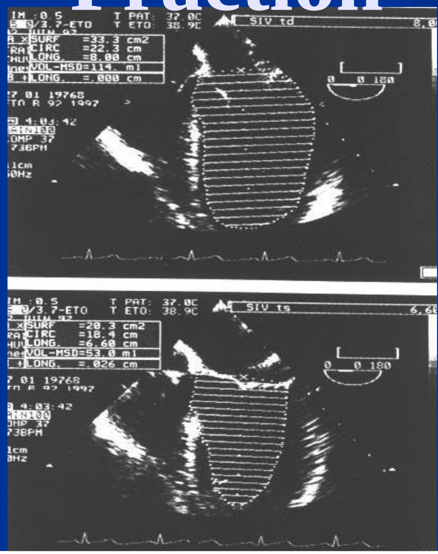
Shortening Fraction



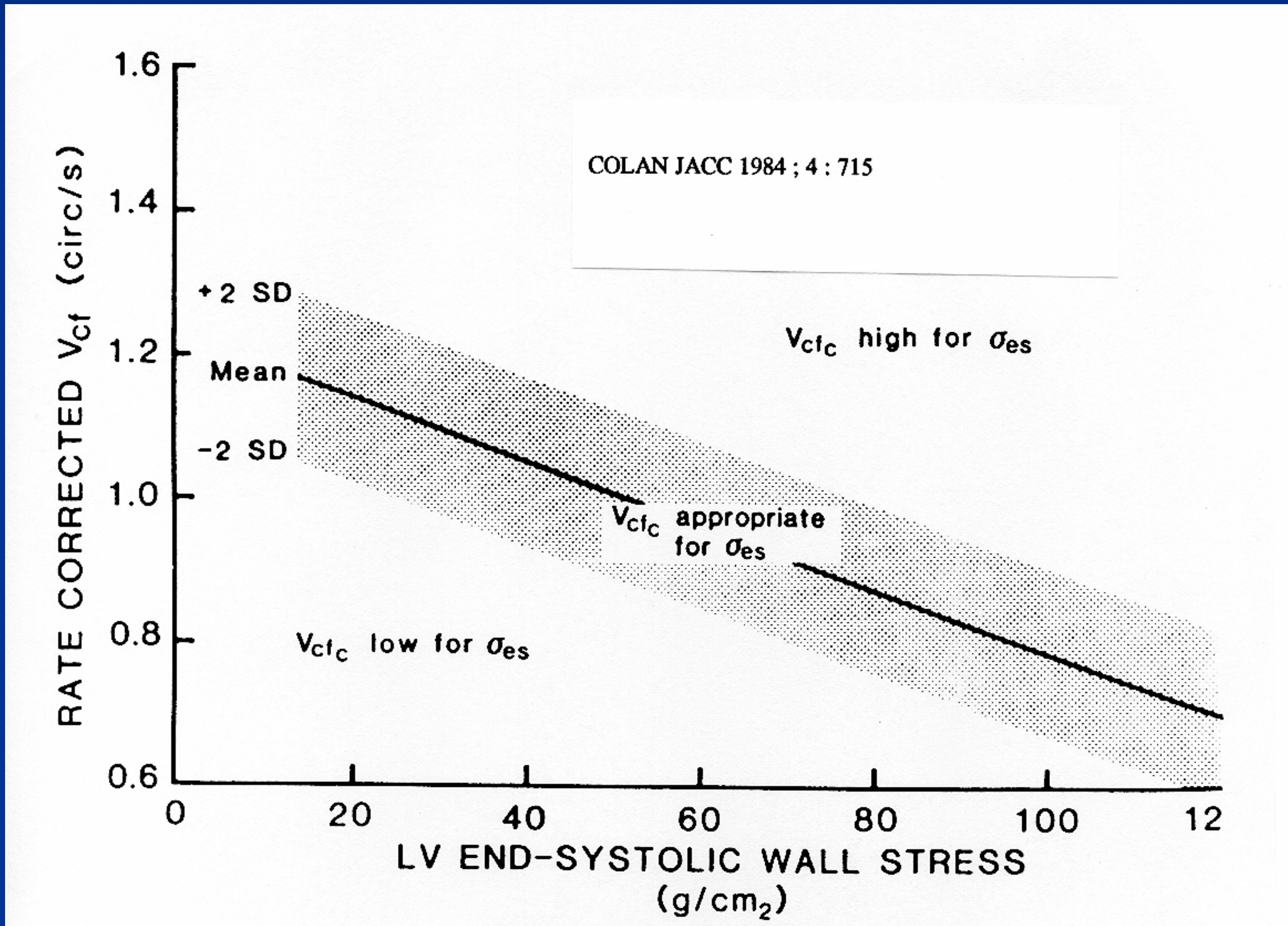
Shortening Fraction of LV Area



Ejection Fraction

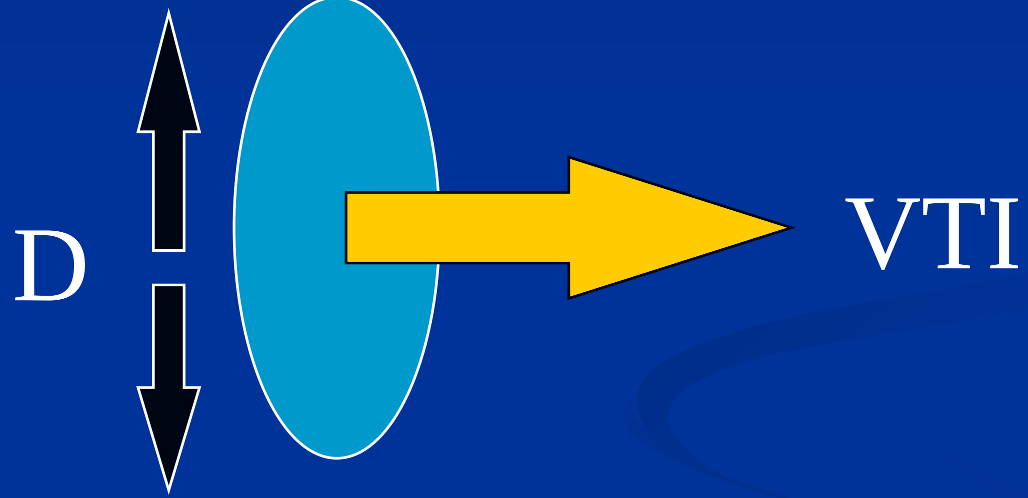


Ejectional indices are afterload dependent (and preoad)



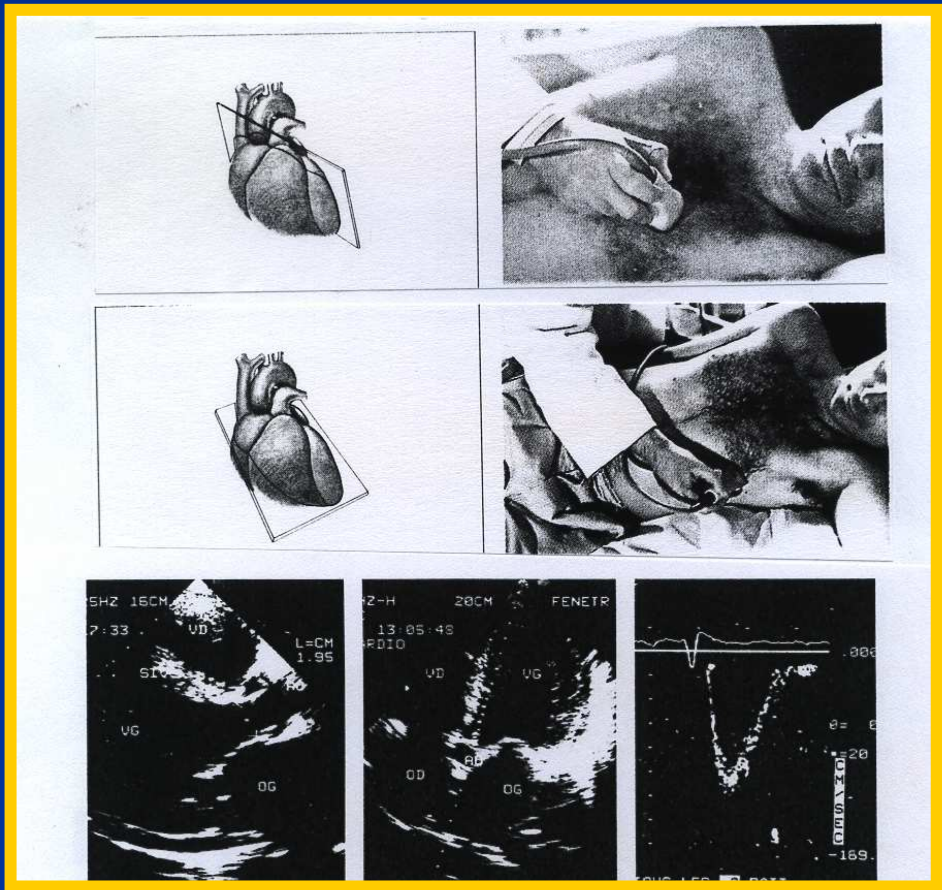
Dois je donner un vasoconstricteur?

Cardiac Output Measurement

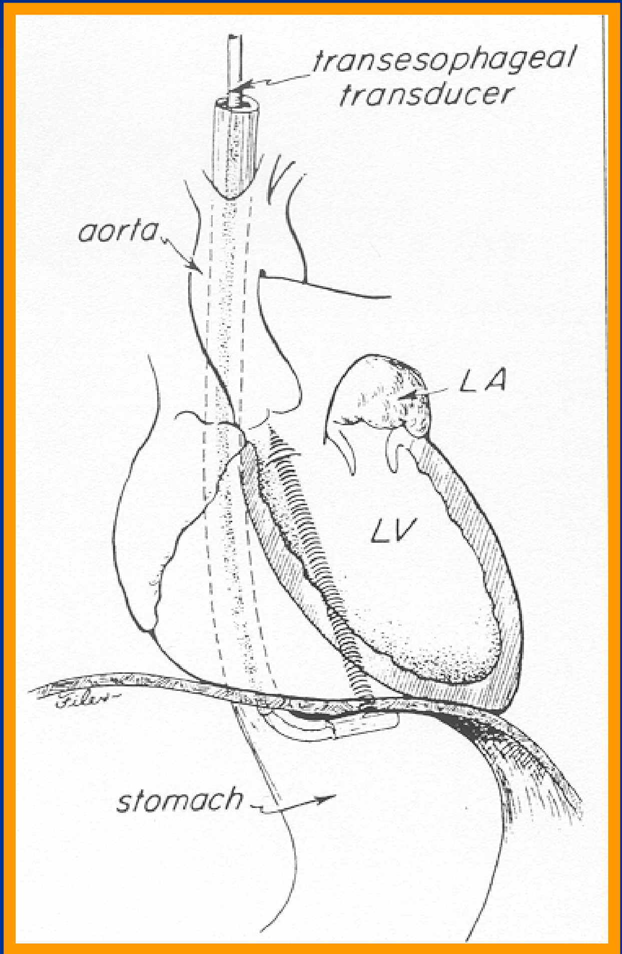


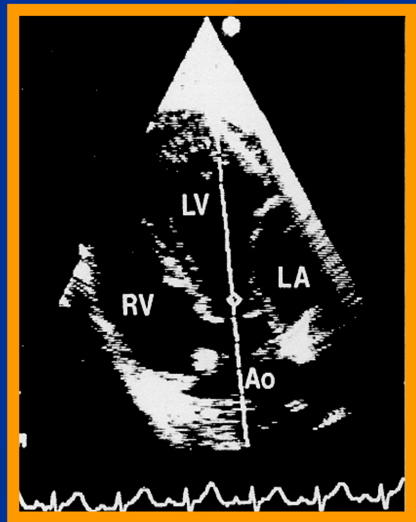
$SV = (\Pi D^2/4) \times VTI$

Transthoracic Cardiac Output



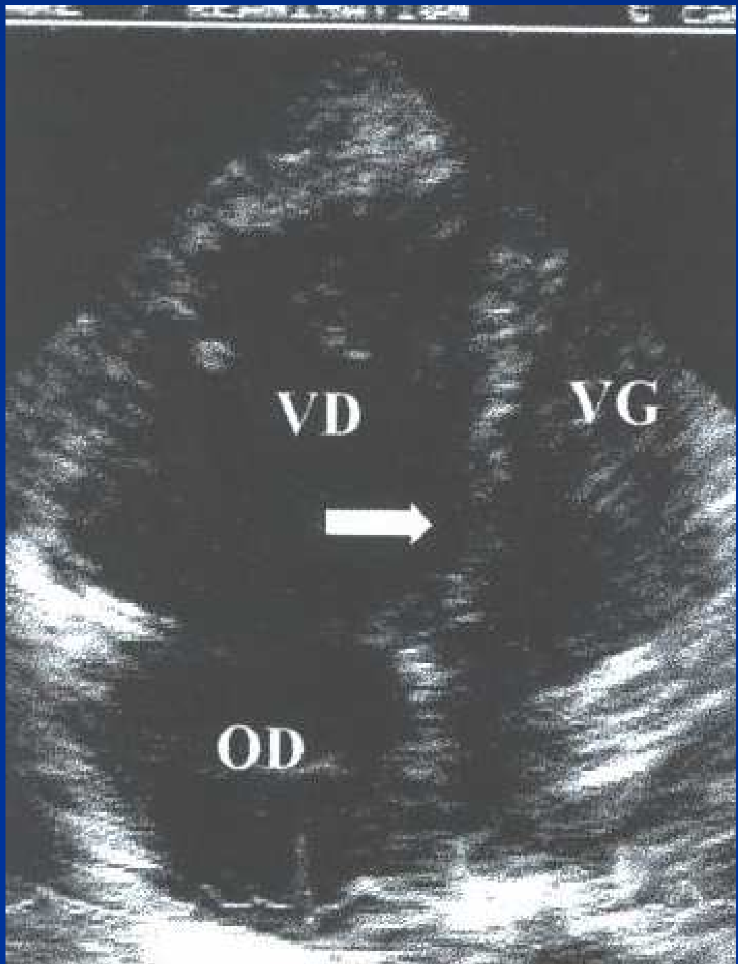
Transesophageal Cardiac output





Dois je modifier mes constantes sur mon respirateur? Dois je donner du NO?

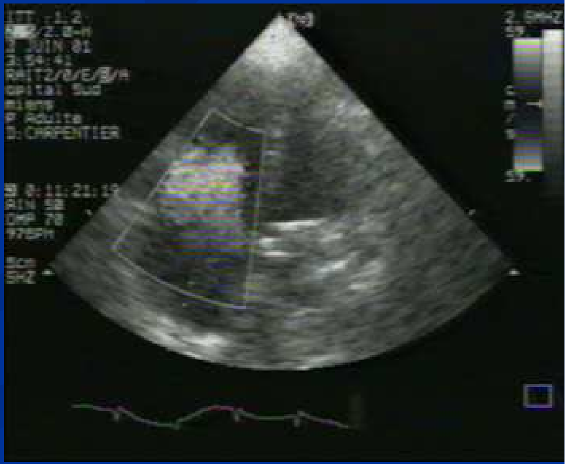
Right Ventricular Size



Pulmonary Arterial Pressures

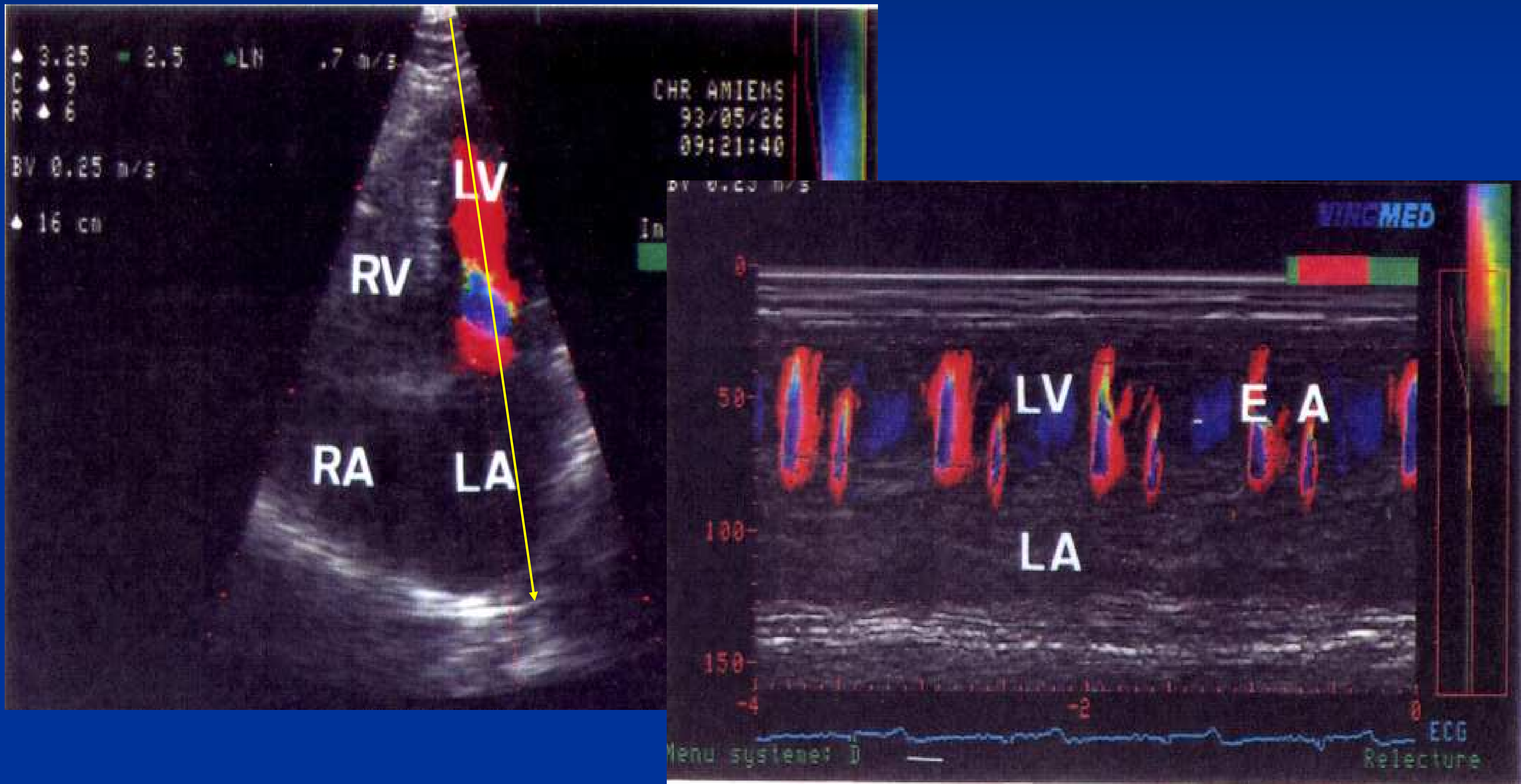
- Systolic Arterial Pressure
  - Recorded in 70-100% of case (70% in ICU patients or patients with COPD, 100% in healthy volunteers)
  - Correlations with invasive methods : 0.8 to 0.99 (Yock PG Circulation 1984, Pepi M JASE 1994)
- Diastolic Arterial Pressure
  - Detected in 60-100% of case
  - Correlations: 0.93-0.97 (Stevenson

JASE 1989, Masuyama Circulation 1986)

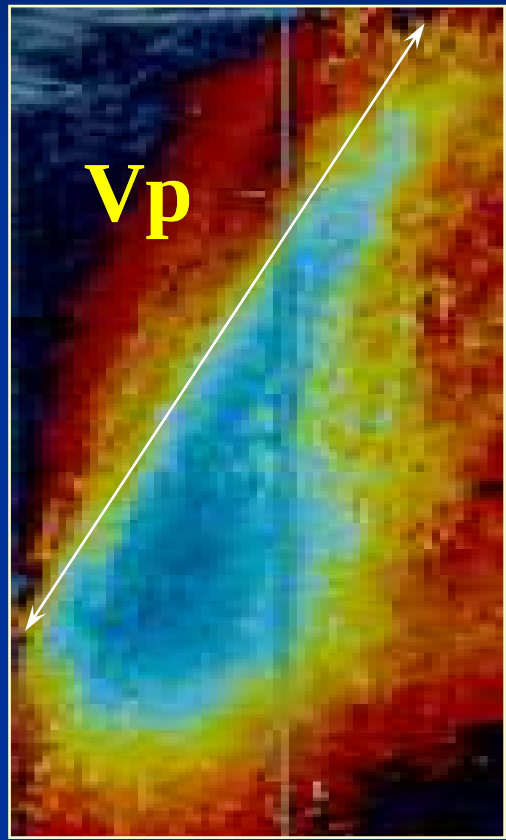


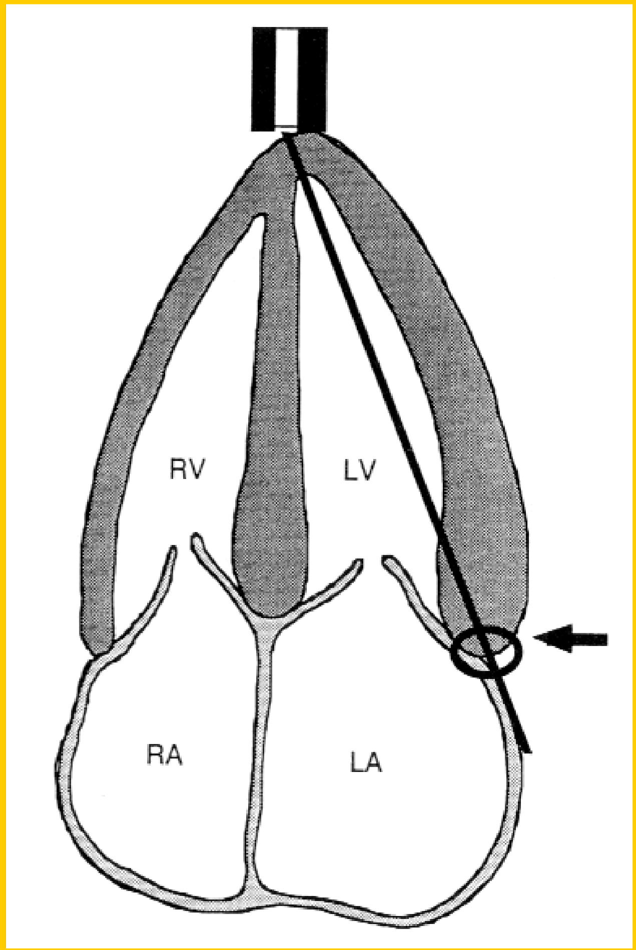
Est-ce un OAP cardiogénique ou un SDRA?

M-Mode Color Doppler Propagation of Mitral Flow (Vp)



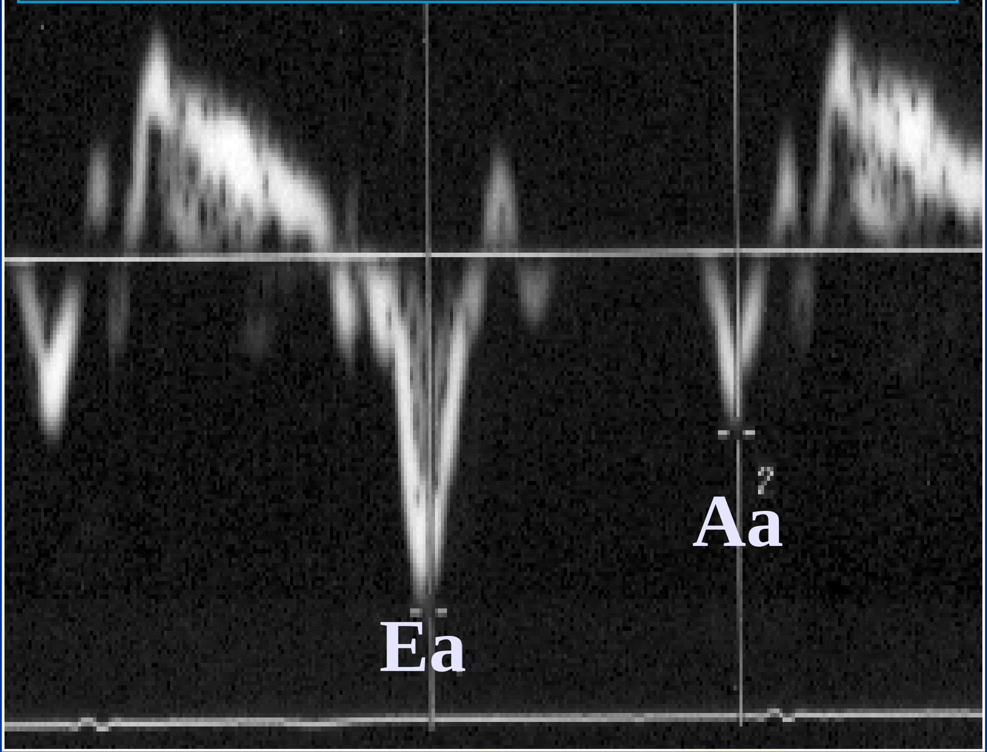
M-Mode Color Doppler Propagation of Mitral Flow (Vp)





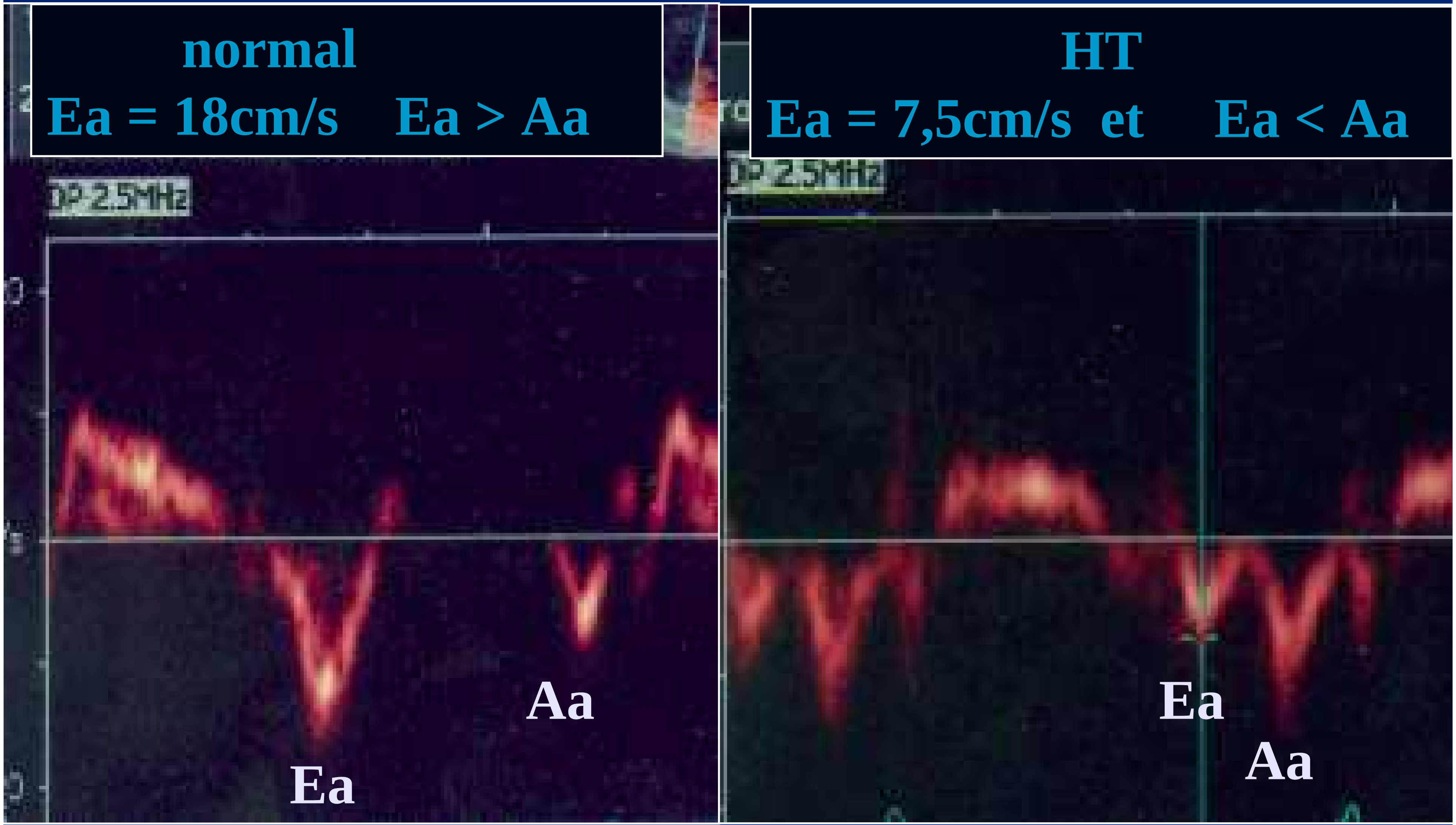
Normal Values

Ea > 8 cm/s et Ea / Aa > 1



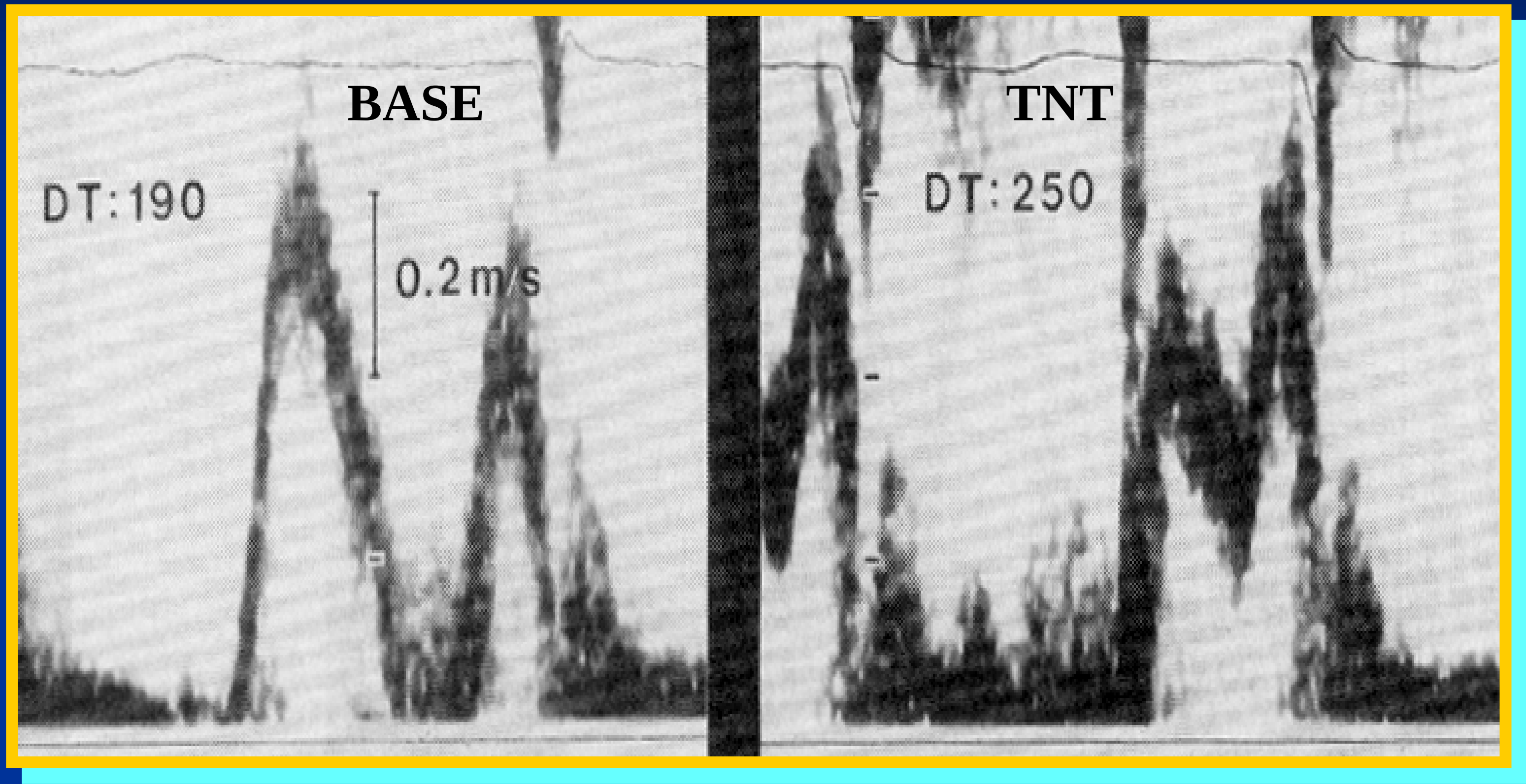
- **DTI**
- **Mitral Annulus**

Early diastolic velocity (Ea)
DTI



LV Diastolic Pressure

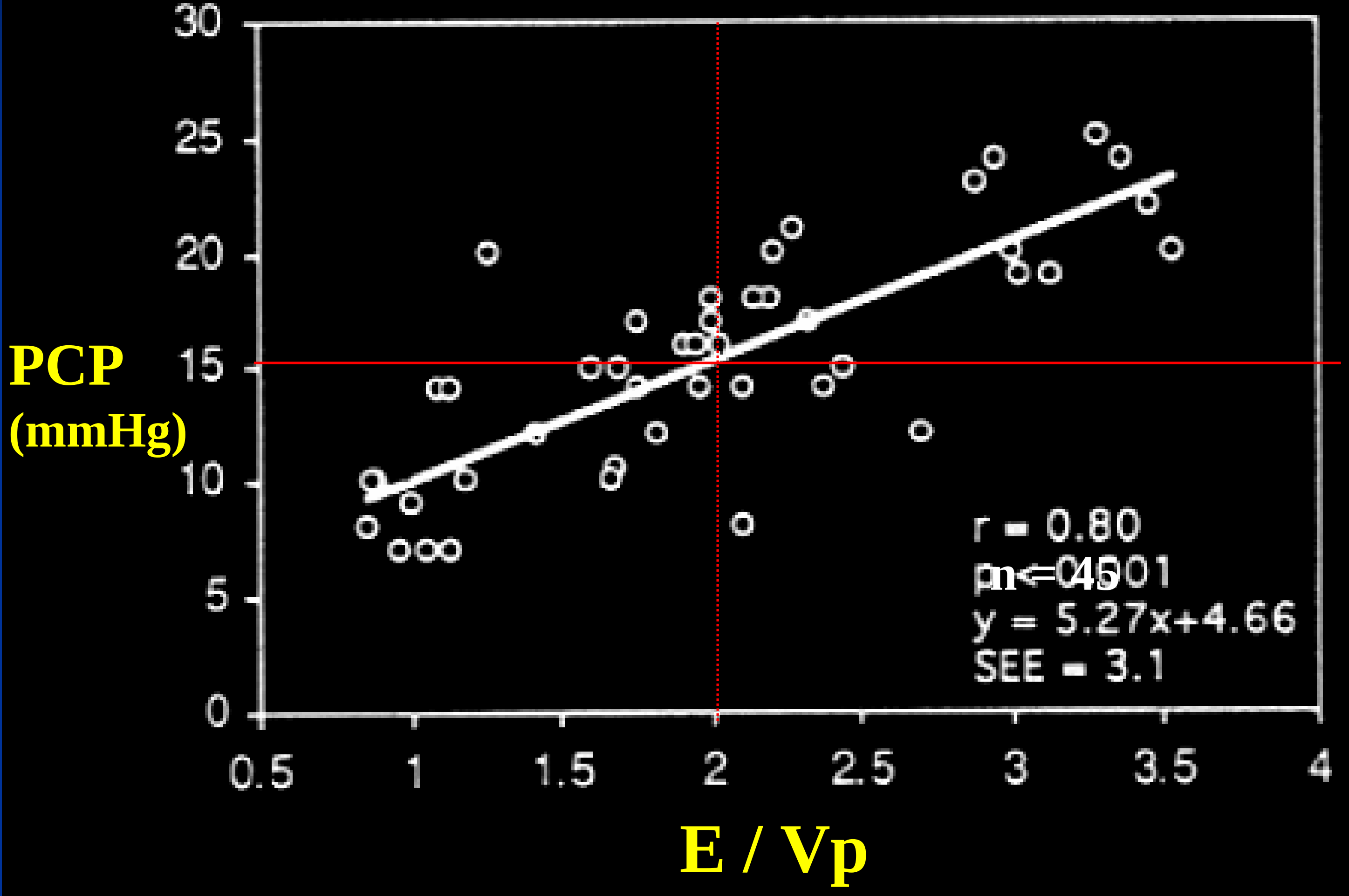
Preload dependance of mitral flow



SOHN, JACC 1997

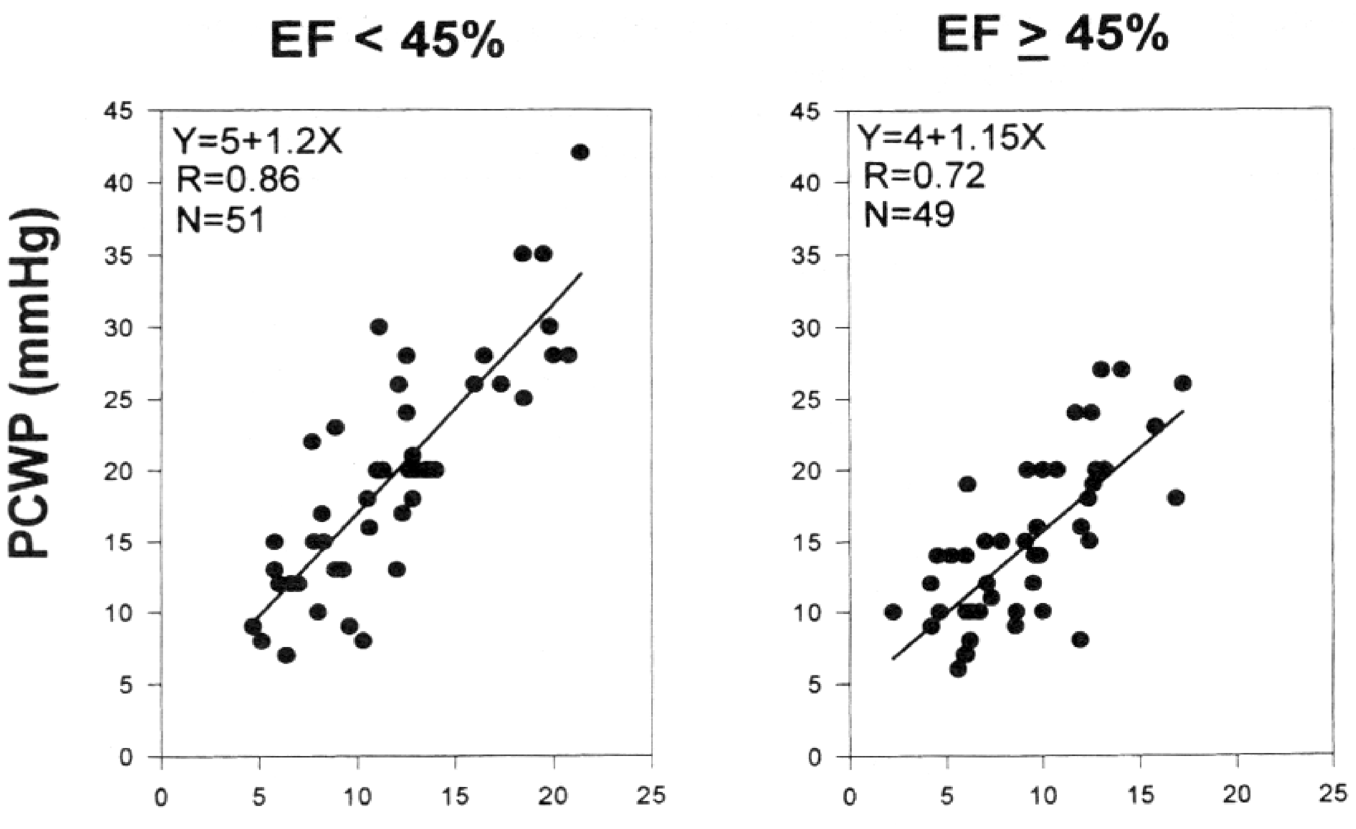
E/Vp

(Garcia et al. JACC 1997; 29: 448-54)



Elevated diastolic pressure : E/Vp > 2.5

E/Ea



Nagueh; Circulation 1998

Elevated Diastolic Pressure

E/Ea > 10 (Nagueh JACC 1997) or E/Ea > 15 (Ommen Circulation 2000)

Shock

- Images : major informations in
  - Valvular disease
  - Tamponade
  - Infarction, severe left ventricular dysfunction
  - Pulmonary embolism
  - Endocarditis
  - Aortique dissection
  - Trauma

Shock

- In many patients no information with images from echocardiography
- Complex situations :
  - Septic shock in patient with previous cardiac dysfunction
  - Right dilation in septic shock patient
- Therefore, hemodynamic evaluation is needed to anderstand the clinical situation

Question 1: can we give fluids? « Yes »

- In the presence of respiratory variations of (>12-15%) IVC, SVC, Aortic velocity or VTI.
- In the presence of small LV (<5 cm2/m2)
- In the presence of mitral flow with A<<E (without LV relaxation impairement), SF> 55%, Ap/Am<1, E/Em<8, E/Vp< 1.5

Question 2 : vasoconstrictive agents?

- Yes
  - Low résistances
  - Low systolic stress
  - Take care in case of cardiogenic impairement

Question 3 : inotropic agents?

- Yes
  - Cardiogenic shock :
    - Hypokinetic LV
    - Low cardiac output
    - High LV diastolic pressures : E/A>2, SF<55%, Am<Ap, E/Em>12, E/Vp>2.5, TDE<150ms
  - LV systolic dysfunction associated with septic shock
- No : in case of cardiogenic shock due to hyperkinesia and LV obstruction

Pulmonary edema

- Question 1: is the pulmonary wedge pressure high?
  - Yes
  - High LV diastolic pressures : E/A>2, SF<55%, Am<Ap, E/Em>12, E/Vp>2.5, TDE<150ms
- Question 2: which is the cause of this pulmonary edema?
  - Echocardiography images

Conclusions

- Echocardiographic and Doppler techniques may help the clinician in decision making process in case of shock or pulmonary edema in giving not only the cause but also <u>the mechanism with a complete hemodynamic evaluation</u>