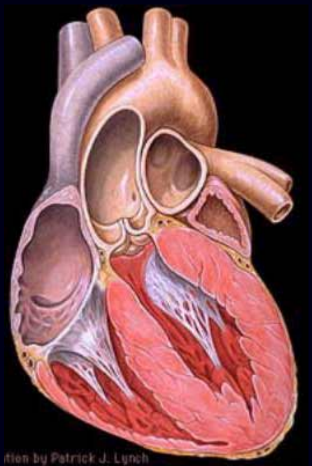
USE OF ECHOCARDIOGRAPHY IN SEPTIC SHOCK



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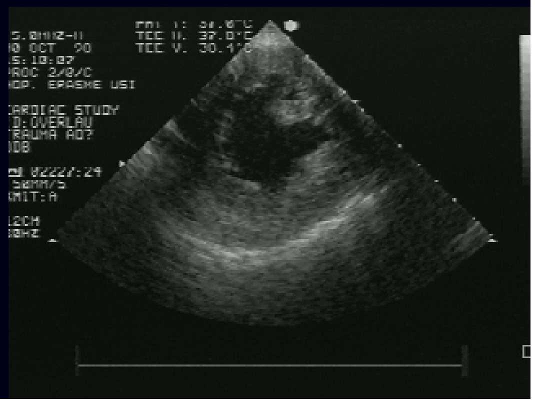


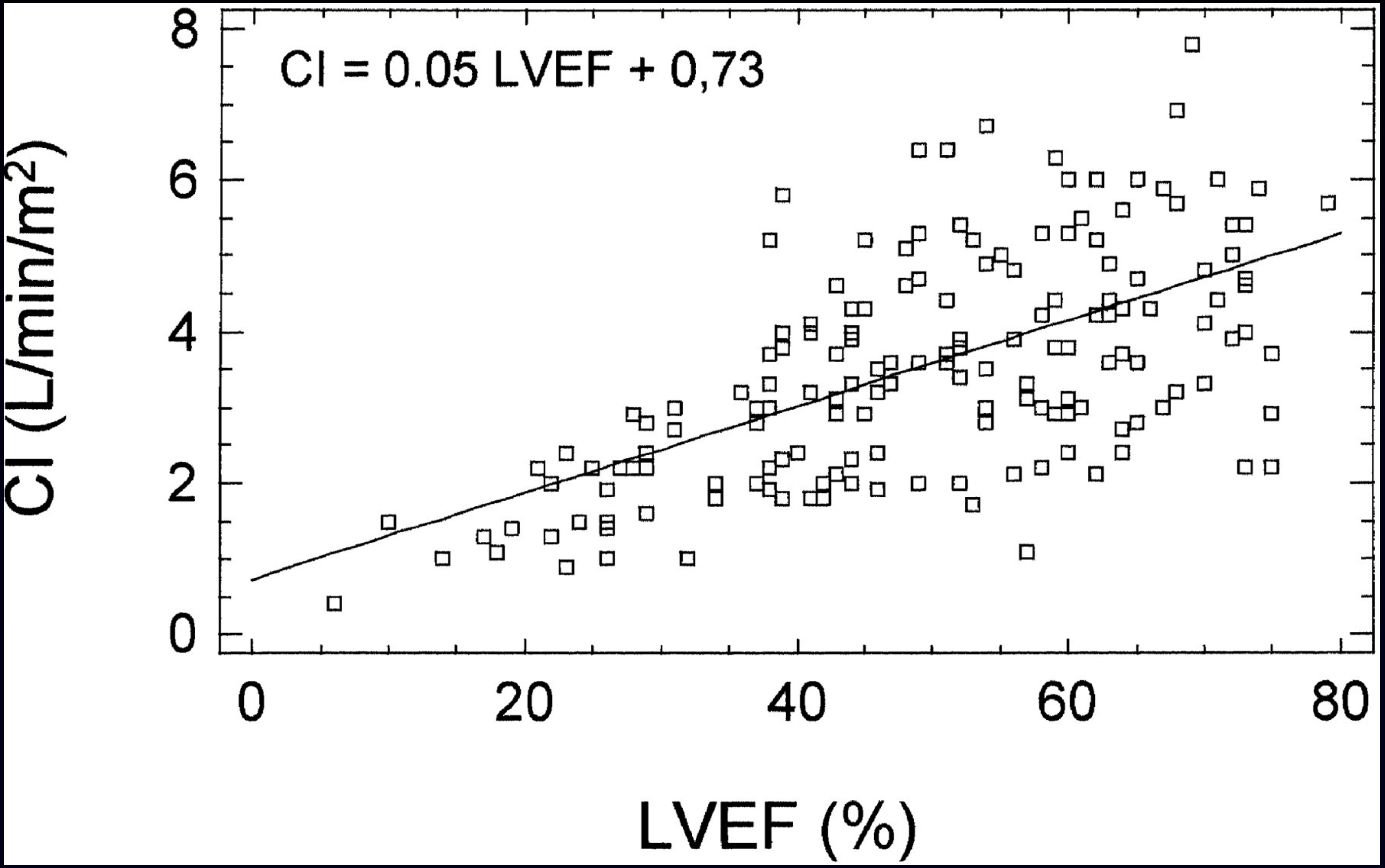
ECHOCARDIOGRAPHY IN SEPTIC SHOCK

PATHOPHYSIOLOGIC ALTERATIONS:

Myocardial depression is frequently observed in septic shock

Echographic evaluation of LVEF in patients with septic shock

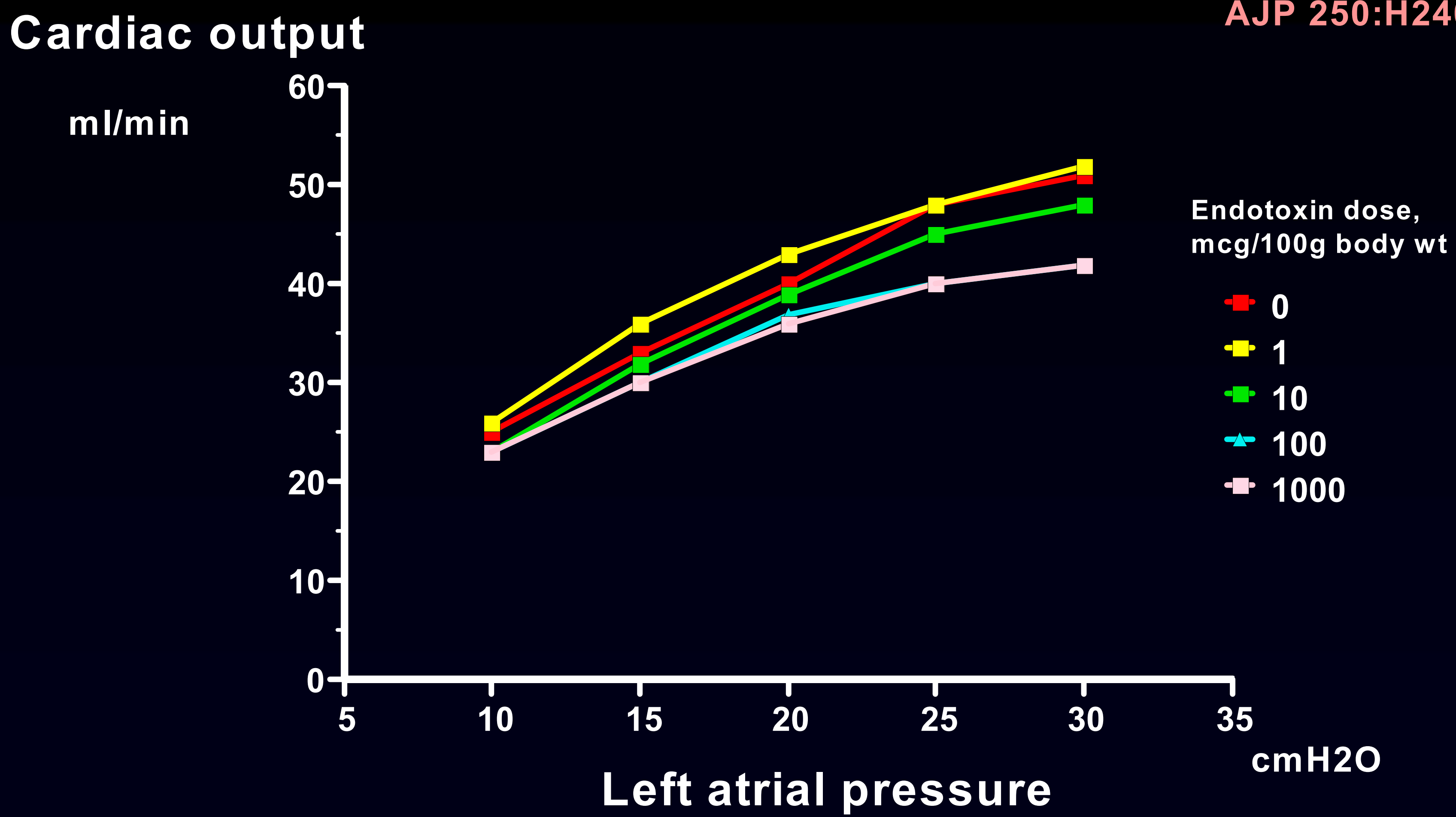
Vieillard-Baron et al
AJRCCM 168:1270;2003



183 patients with septic shock

EFFECTS OF ENDOTOXIN ON MYOCARDIAL FUNCTION

McDonnough et al
AJP 250:H240;1986

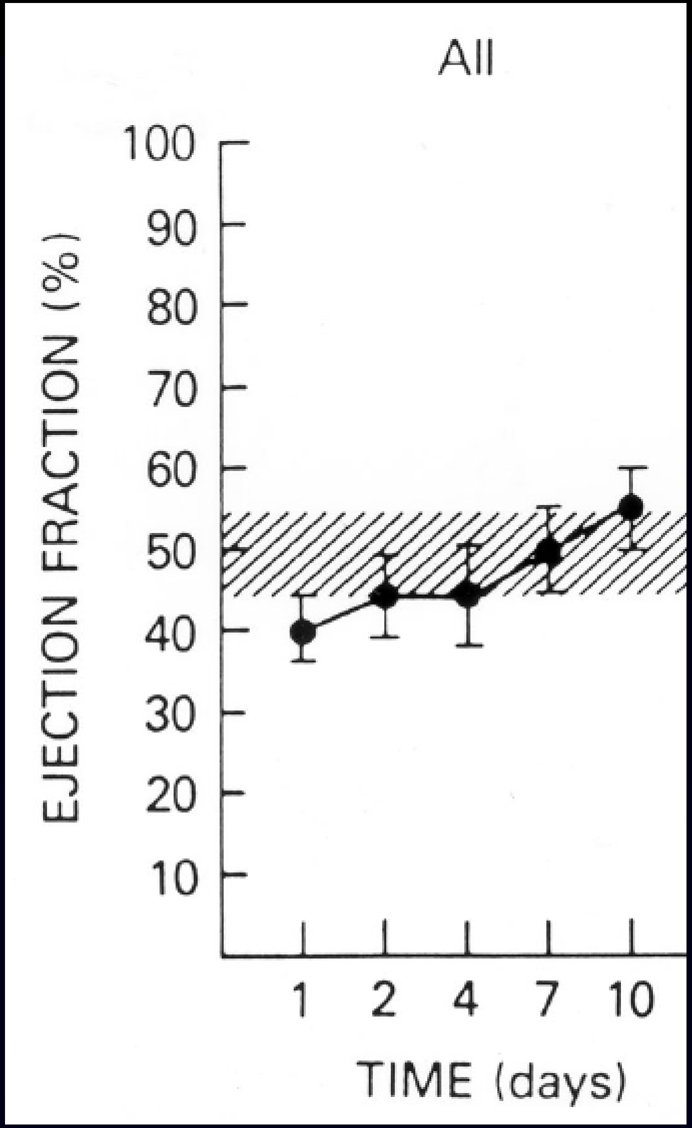


Isolated heart (rat)

DDB USI

Reversible myocardial depression in patients with septic shock

Parker et al. Ann Intern Med 1984: 100; 483-490

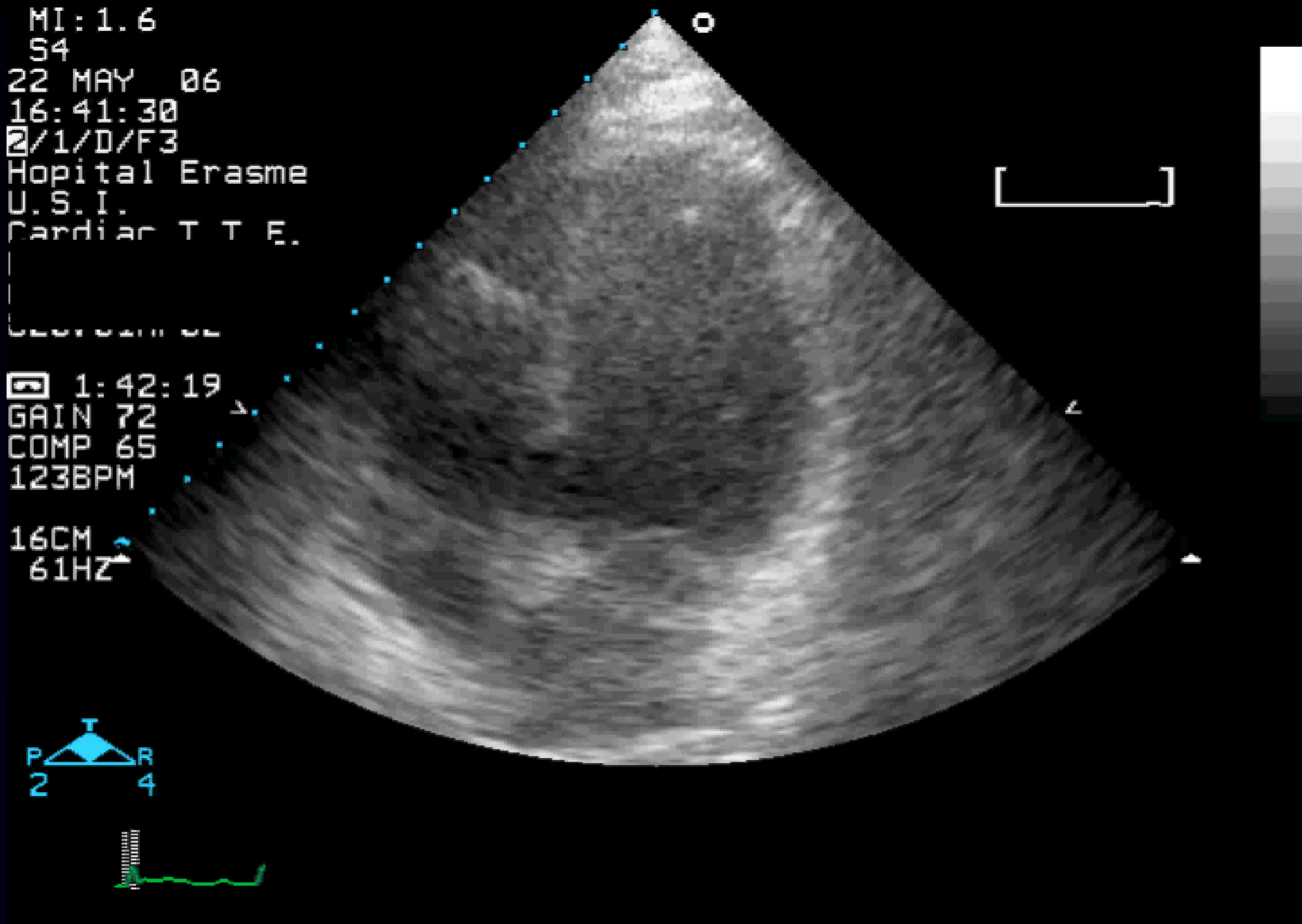


DDB USI

Clinical case:

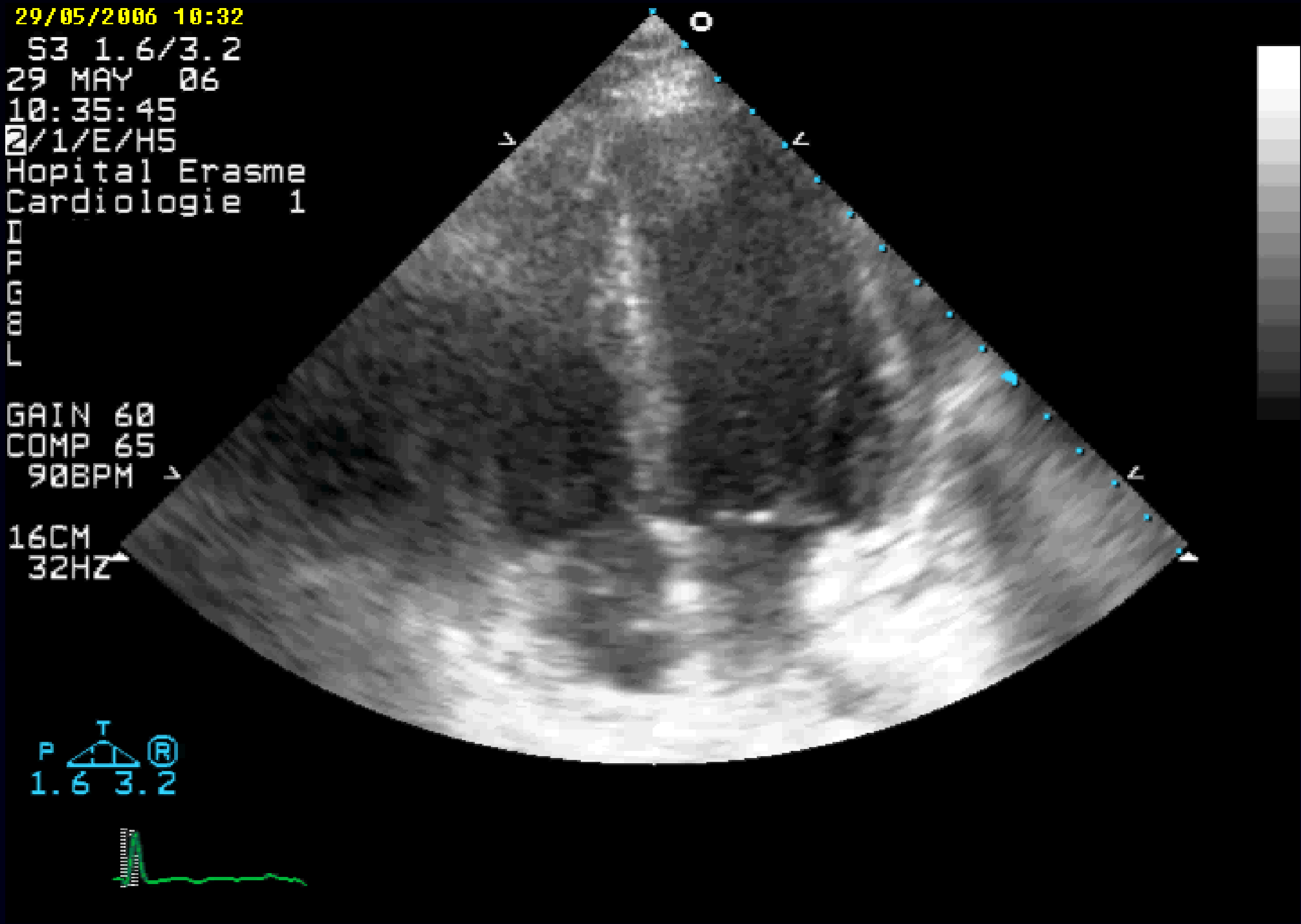
A 30 YO male with history of splenectomy in childhood (trauma) admitted for septic shock due to lung infection (Streptococcus pneumoniae).

Admission



MI:1.6
S4
22 MAY 06
16:41:30
2/1/D/F3
Hopital Erasme
U.S.I.
1:42:19
GAIN 72
COMP 65
123BPM
16CM
61HZ
P
R
2
4

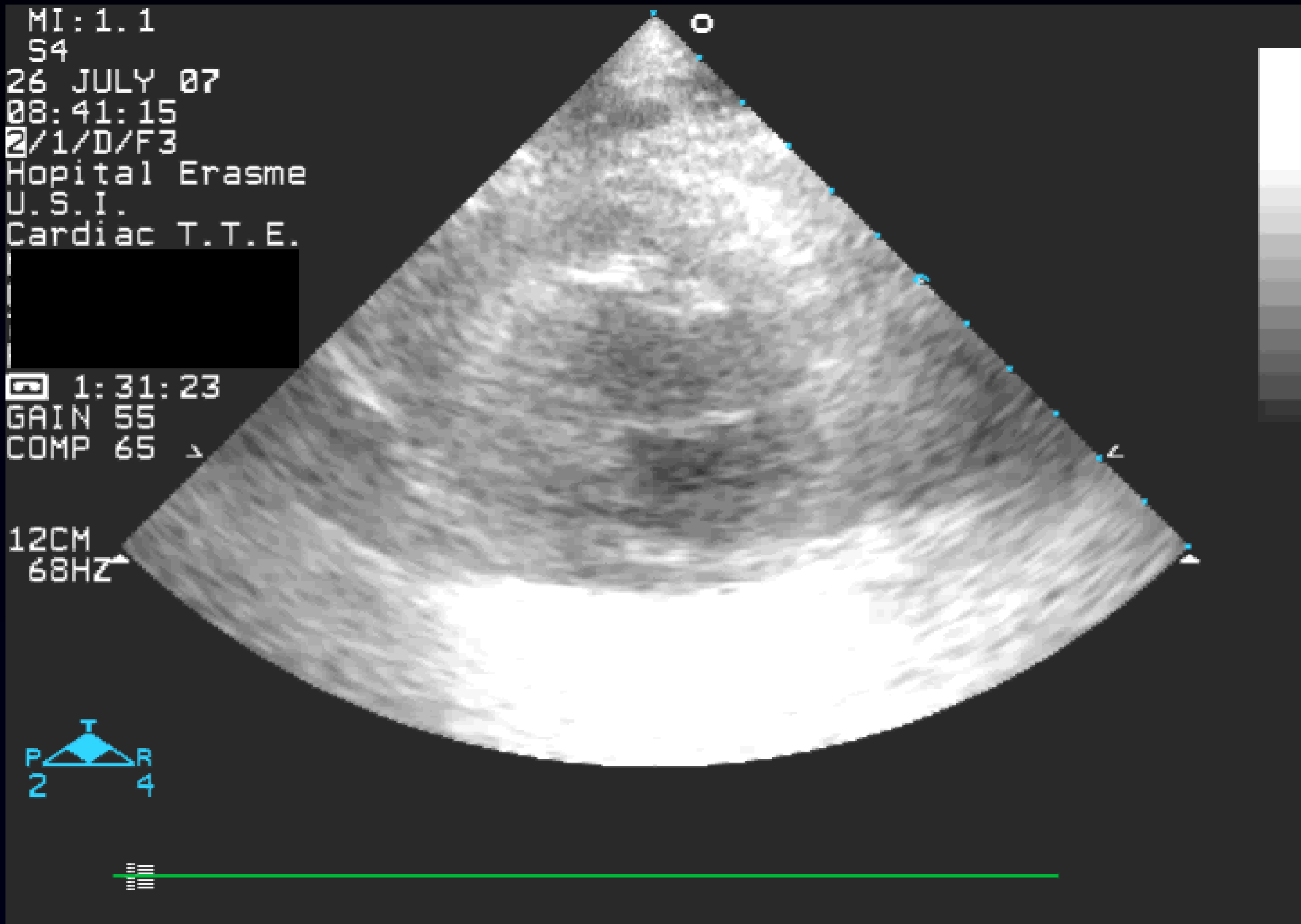
ICU discharge (day 7)

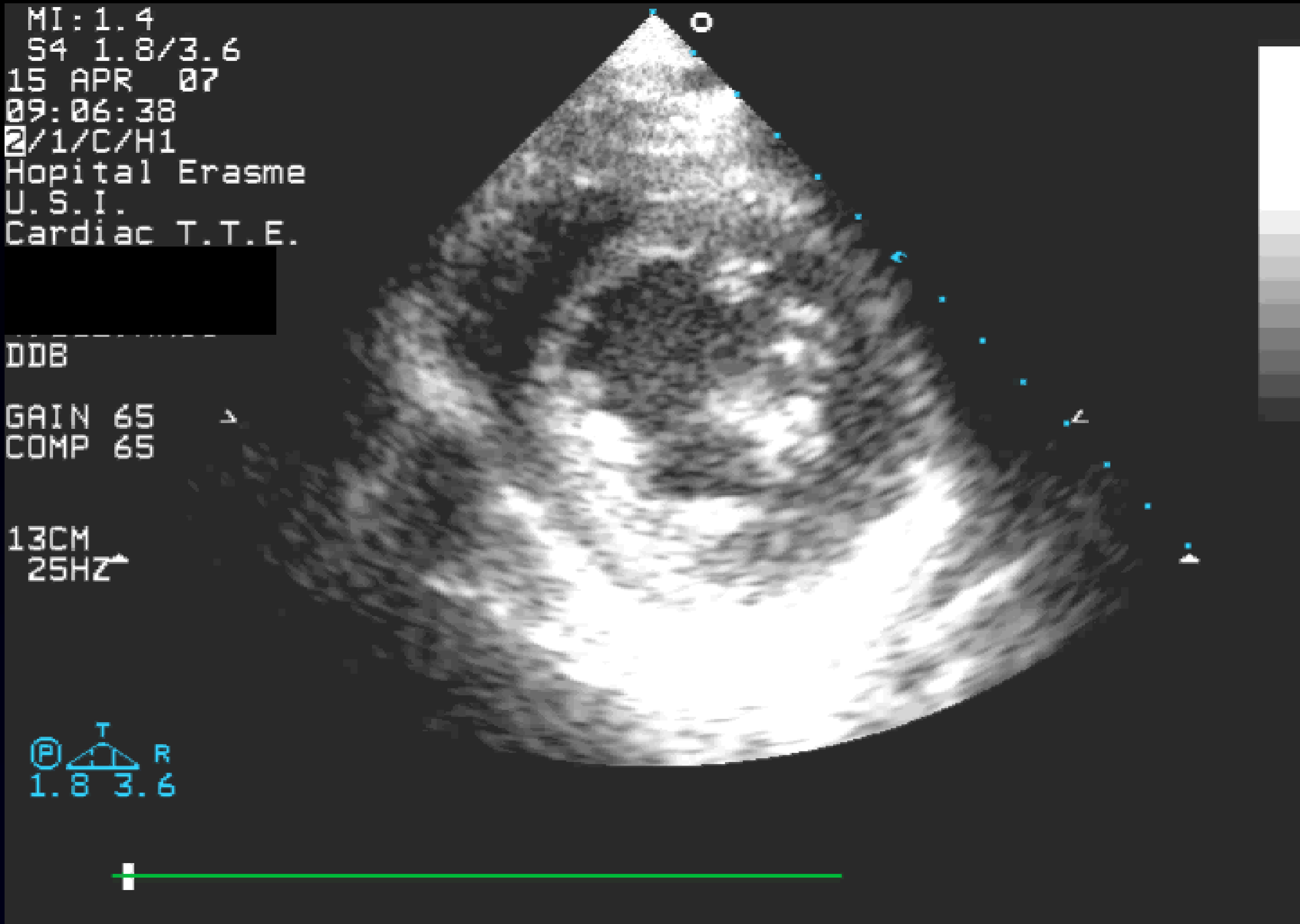


But cardiac output may be preserved…

CO 4.5 L/min

$ScvO_2$ 52%

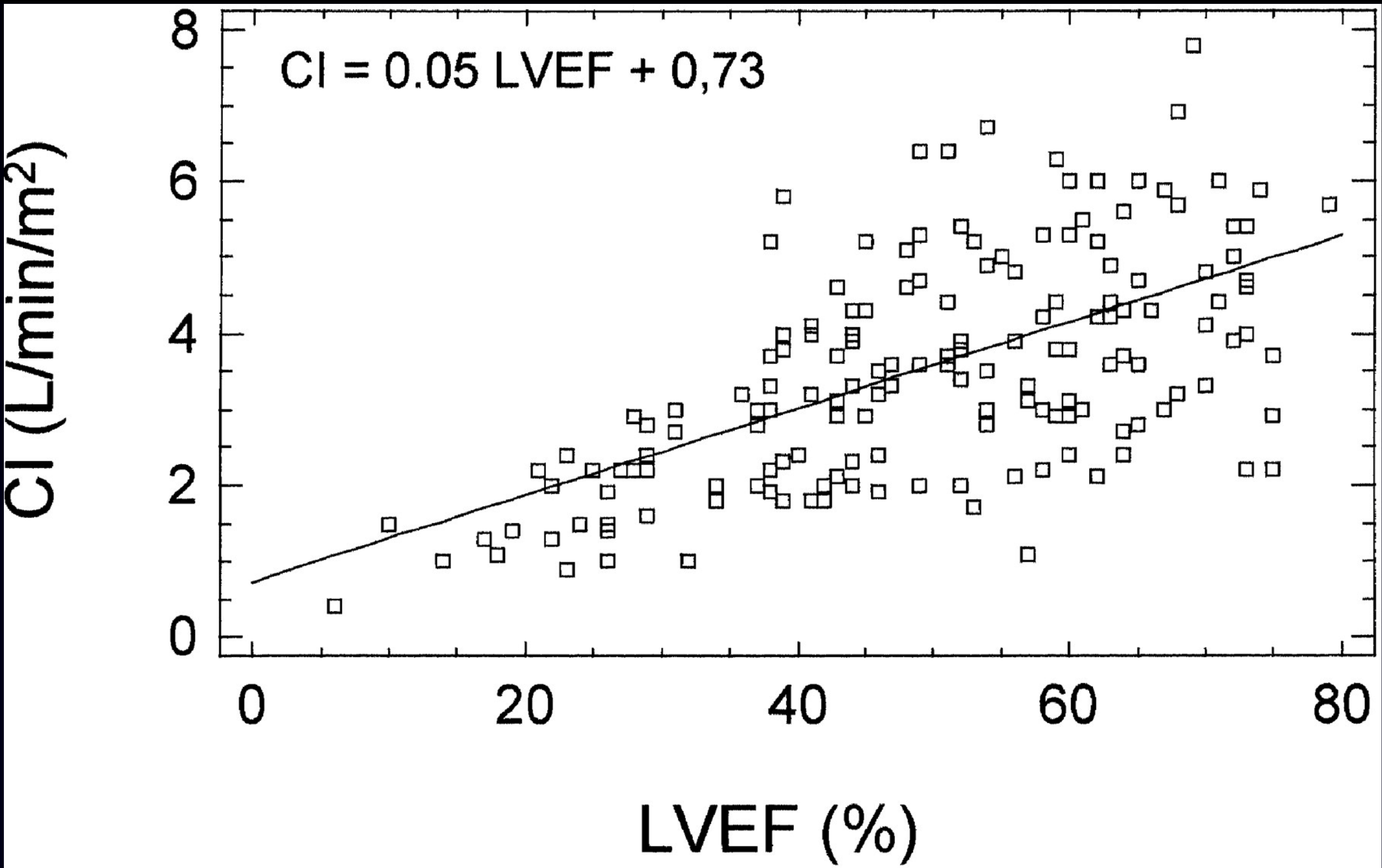




CO 8.2 L/min
ScvO2 68%

Echographic evaluation of LVEF in patients with septic shock

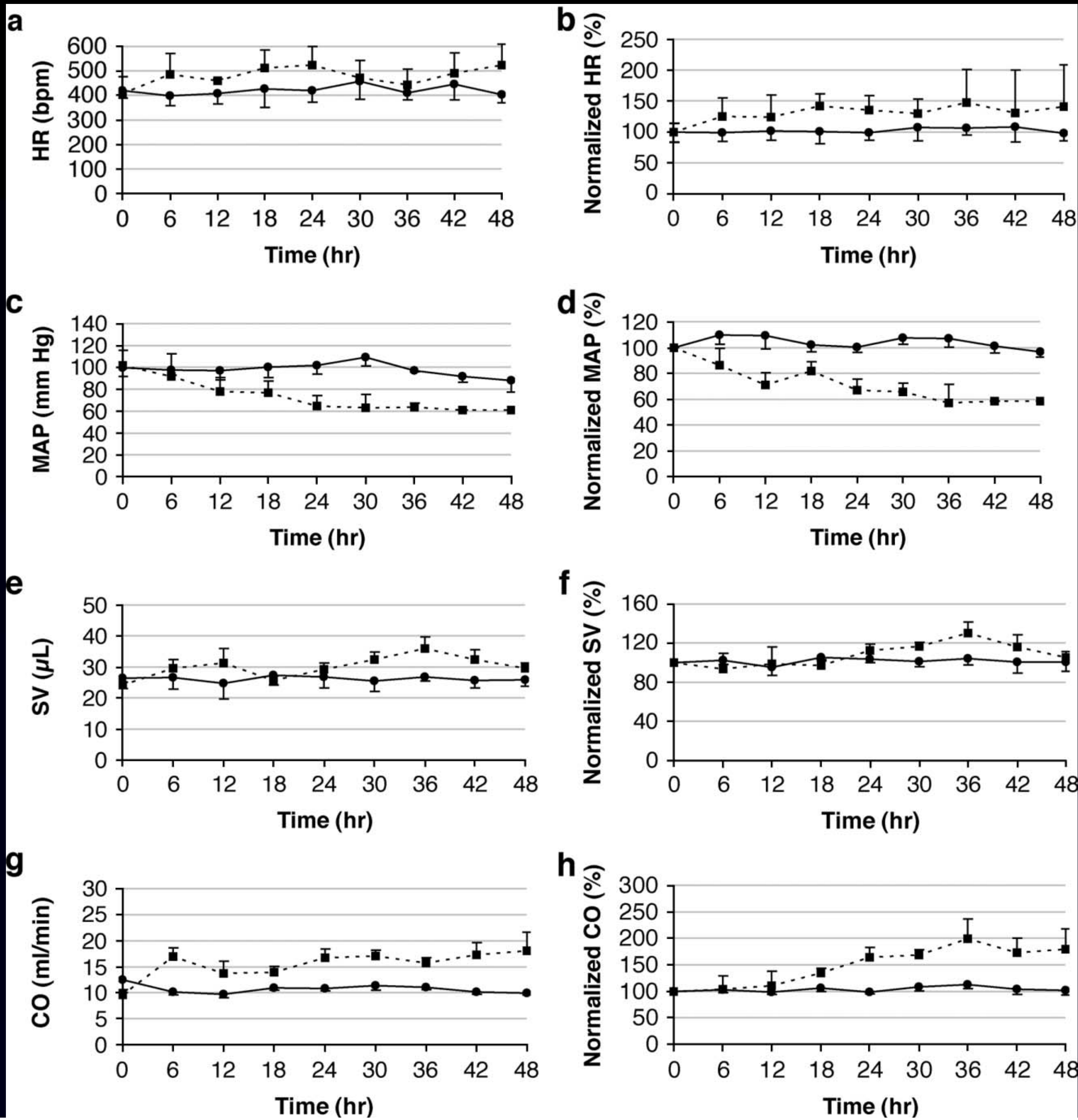
Vieillard-Baron et al
AJRCCM 168:1270;2003



183 patients with septic shock (1/3 hypodynamic)

How can cardiac output be preserved in the context of impaired contractility?

- Increased heart rate
- Decreased afterload
- Preload adaptation ?



Hollenberg et al
Shock 24:58;2005

Mice
CLP
Fluids

Preload adaptation refers to acute LV dilation

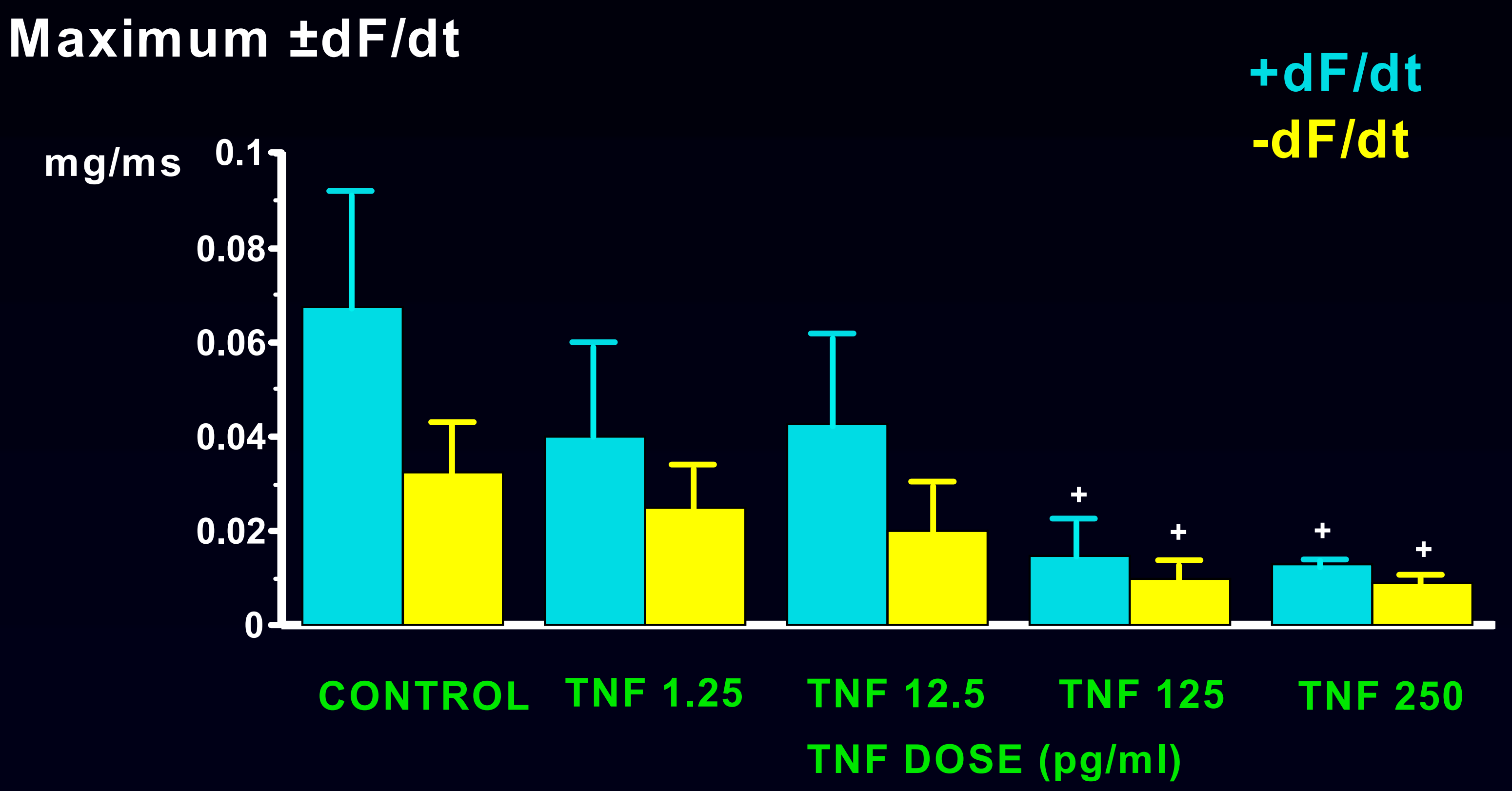
EF = SV / EDV

If SV is maintained in the presence of a decreased EF, this can be achieved only via an increase in EDV (preload adpatation)

Diastolic dysfunction also occurs…

SYSTOLIC AND DIASTOLIC MYOCARDIAL DEPRESSION

Cain et al
CCM 27:1309;1999



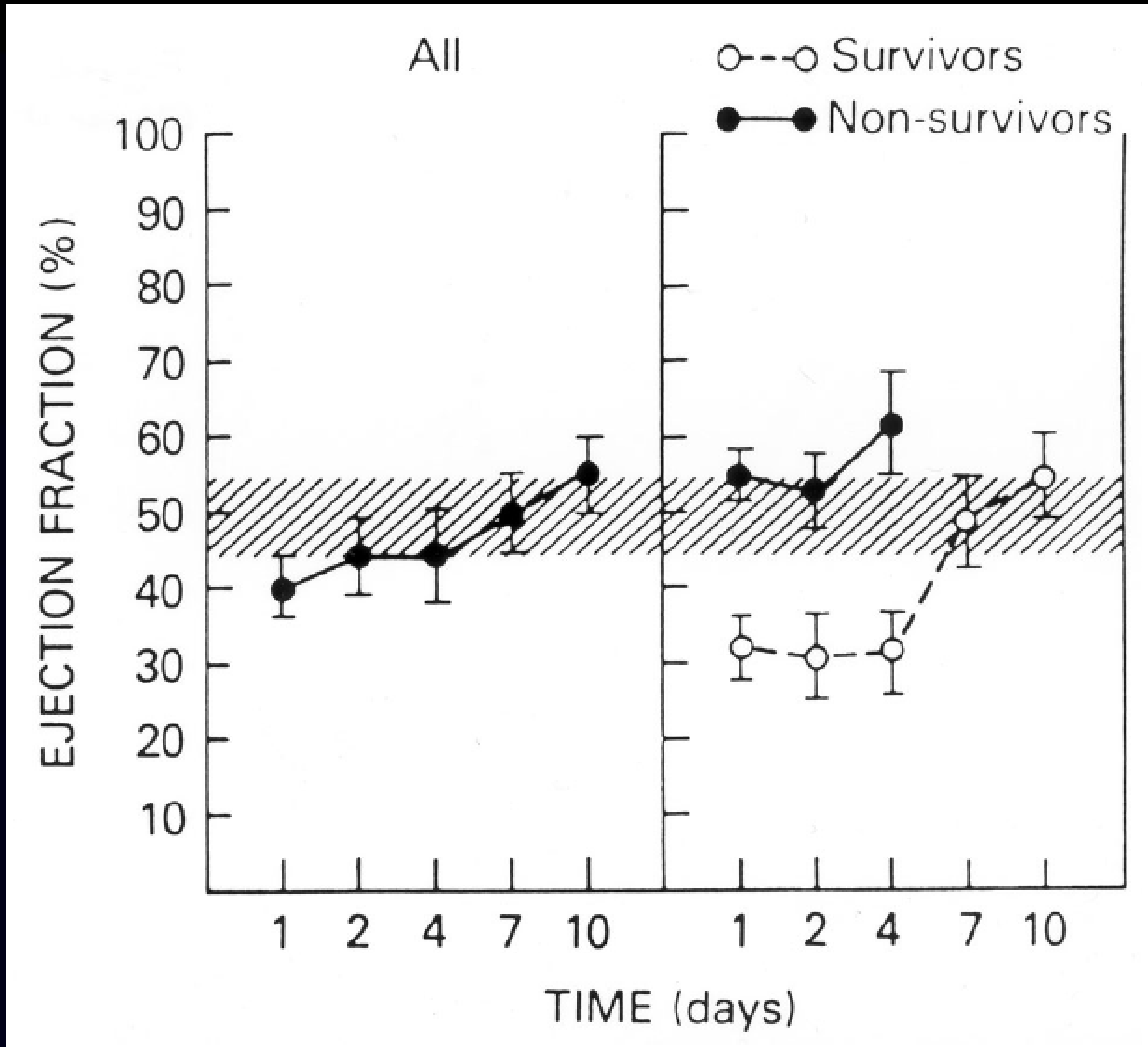
+ $p<0.05$ vs control

Isolated human atrial trabecules

DDB USI

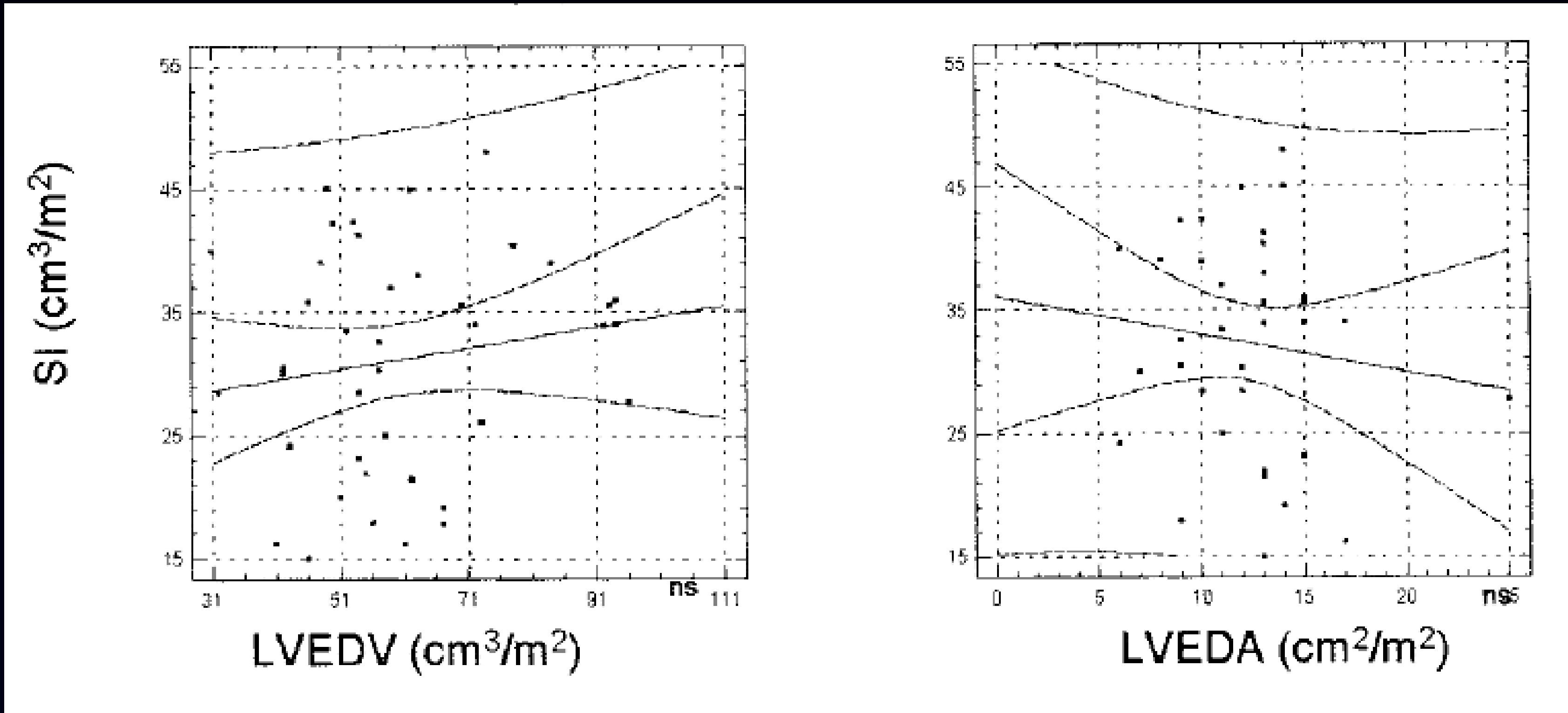
Reversible myocardial depression in patients with septic shock

Parker et al. Ann Intern Med 1984: 100; 483-490



Does preload adpatation occurs ?

Vieillard-Baron
Anesthesiology 94:3;2001



LVEDV within normal values in most instances

McLean et al
CCM 35:1019;2007

	With Normal LV Function, Baseline LVEF ≥55%	With RCD	*p* Value
No.	23	7	
No. with pre-existing cardiac condition	2	0	
Age, yrs	63.8 ± 13.1 [64; 56–71]	57.3 ± 9.5 [61; 42–69]	.169
APACHE II	19.5 ± 6.3 [20; 15–23]	25.3 ± 9.0 [22; 15–39]	.160
Baseline LVEF, %	58.1 ± 3.4 [58; 55–60]	38.3 ± 10.9 [40; 20–50]	<.001
E/E'	14.2 ± 7.5 [14.6; 9.5–16.1]	14.1 ± 7.1 [10.5; 6.7–25.8]	.976
LVEDD, cm	4.2 ± 0.8 [4.2, 3.9–4.8]	4.9 ± 0.7 [4.9, 3.9–5.9]	.034
Baseline cardiac output, L/min	5.9 ± 1.7 [5.9; 5.2–6.5]	6.3 ± 1.4 [5.9; 4.9–9.2]	.623
LOS_{ICU}	11.2 ± 11.1 [9; 5–13]	19.4 ± 17.7 [16; 4–58]	.084
LOS_{HOS}	23.6 ± 28.6 [15; 9–20]	32.4 ± 21.7 [28; 8–65]	.148
Mechanical ventilation:			
n (%)	15 (65.2%)	7 (70%)	.893
Mean ventilation hour, hr	152 ± 256 [73; 0–157]	218 ± 114 [192; 51–385]	.045
Mortality, n (%)	5 (21.7)	0 (0)	.155
Admission BNP, pg/mL	568 ± 811 [279; 110–636]	630 ± 726 [272; 43–1970]	.806

Impact of afterload

Kozerias et al
ICM 33:1619;2007

Septic shock patients

NE MAP 65 =>90 =>65 mmHg

Parameter	Baseline	Intervention	Control
HR (1/min)	98 ± 19 (100)	95 ± 17 (94)	94 ± 19 (95)
CVP (mmHg)	12 ± 5 (12)	13 ± 7 (13)	12 ± 5 (13)
CI ($l/min/m^2$)	3.3 ± 0.9 (3.4)	3.4 ± 0.9 (3.6)*	3.3 ± 0.9 (3.3)
SVR ($dyn*s*cm^{-5}$)	551 ± 106 (535)	746 ± 91 (759)*	566 ± 138 (525)
GEF (%)	22 ± 6 (22)	22 ± 6 (23)	23 ± 7 (22)
GEDVI (ml/m^2)	703 ± 178 (705)	747 ± 175 (761)*	704 ± 170 (696)
ITBVI (ml/m^2)	816 ± 203 (827)	867 ± 195 (915)*	821 ± 205 (853)
EVLWI (ml/kg)	7.0 ± 2.7 (7.0)	7.5 ± 3.0 (7.0)	7.4 ± 3.0 (7.0)
LVEDA cm^2/m^2	10.4± 4.7	12.4±3.8	9.7±3.3
LVEF %	37 ± 12	36 ± 12	38 ± 11

Impact of afterload / preload (MAST)

Loubrires et al
Chest 118:1619;2000

↑ LVSA　　　　↓ LVSA

Variables	Group 1 Baseline	Group 1 MAST	Group 2 Baseline	Group 2 MAST
SAP, mm Hg	122 ± 15	133 ± 14†	121 ± 25	127 ± 25†
DAP, mm Hg	59 ± 8	64 ± 6	65 ± 18	70 ± 16†
HR, beats/min	90 ± 18	90 ± 18	103 ± 20	102 ± 20
LVEDA, cm^2/m^2	14.4 ± 6.2	16.6 ± 7.8†	17.1 ± 6.2	17.8 ± 6.4
LVESA, cm^2/m^2	6.8 ± 6.3	7.6 ± 8	10.5 ± 6.3	12.3 ± 6.8†
LVSA, cm^2/m^2	7.6 ± 1.7	9 ± 1.6†	6.6 ± 2.1	5.5 ± 2.1†
LVFAC, %	59.6 ± 31.2	62.9 ± 20.5	43.0 ± 17.9	35.2 ± 17.7†
LVσ, $kdyn/cm^2$‡	50.4 ± 31.2	59.8 ± 36.7†	65.6 ± 33.4	79 ± 36.2†

*Data are presented as mean ± SD. HR = heart rate; DAP = diastolic arterial pressure.
†$p < 0.05$ (Wilcoxon signed rank test).
‡Values for LVσ were not normalized for BSA.

26 pts septic shock

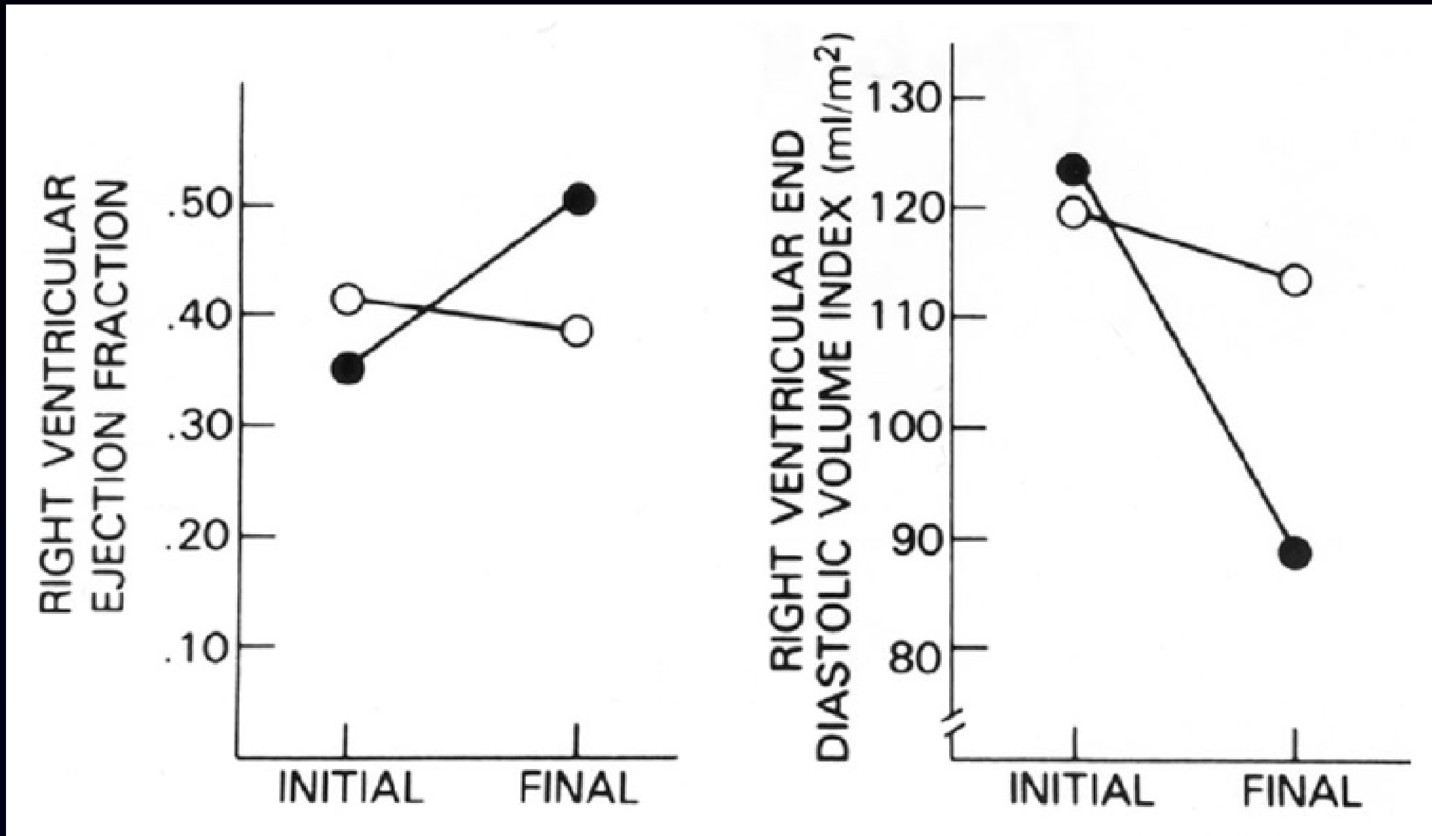
How can cardiac output be preserved in the context of impaired contractility?

- Increased heart rate => yes!
- Decreased afterload => yes
- Preload adaptation => probably but limited

Myocardial dysfunction in sepsis:

Right ventricular dysfunction ?

Parker M et al
Chest 97:126;1990



Myocardial dysfunction in sepsis:

- Alteration in systolic function
- Alteration in diastolic function
- Related with severity of disease and outcome
- Potentially reversible

ECHOCARDIOGRAPHY IN SEPTIC SHOCK

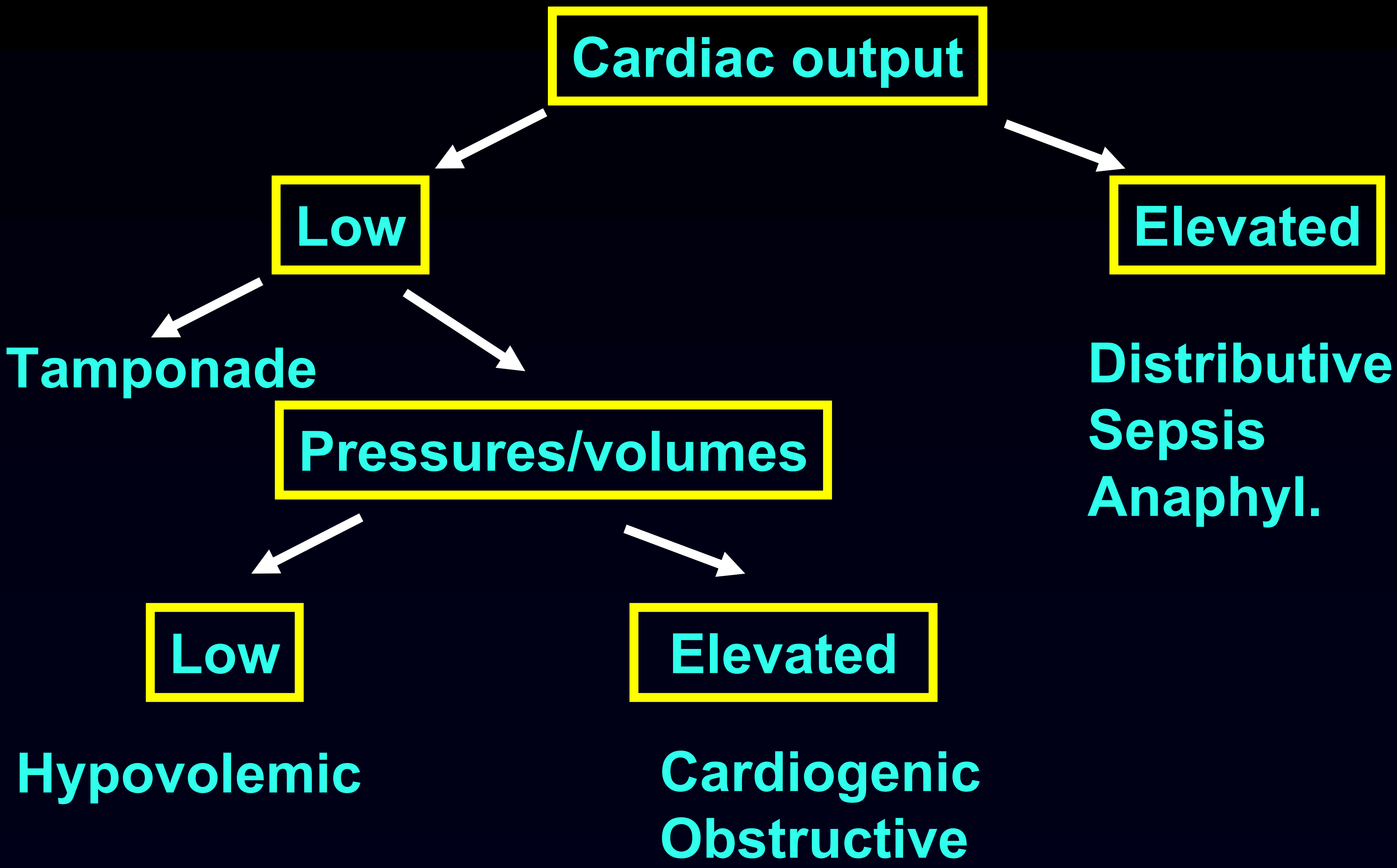
Purpose of echocardiographic evaluation:

- Identify the type of shock
- Hemodynamic evaluation
- Endocarditis

ECHOCARDIOGRAPHY IN SEPTIC SHOCK

How to diagnose septic shock with echo ?

DIAGNOSTIC APPROACH



ECHOCARDIOGRAPHY IN SEPTIC SHOCK

How to diagnose septic shock with echo ?

ECHOCARDIOGRAPHY IN SEPTIC SHOCK

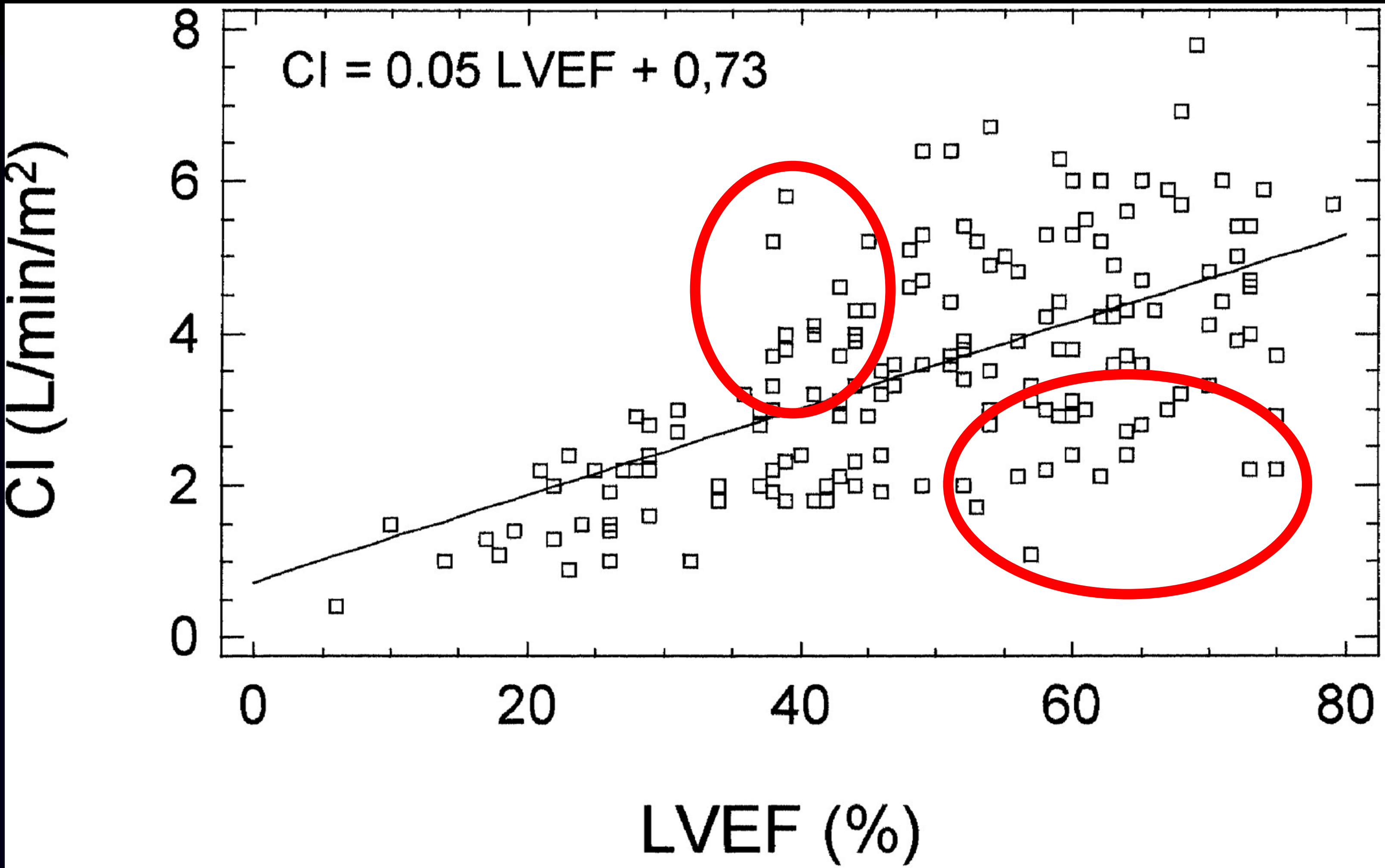
How to diagnose septic shock with echo ?

Difficult !

Identification of a hyperdynamic state is suggestive of sepsis, but hypodynamic state may also occur (1/3 patients) and no definite diagnosis can be made in these cases.

HEMODYNAMIC MONITORING
OF THE PATIENT IN SEPTIC SHOCK

Vieillard-Baron et al
AJRCCM 168:1270;2003



⇒CO can be preserved even when EF is markedly altered
⇒CO can be altered even when EF is normal (hypovolemia)

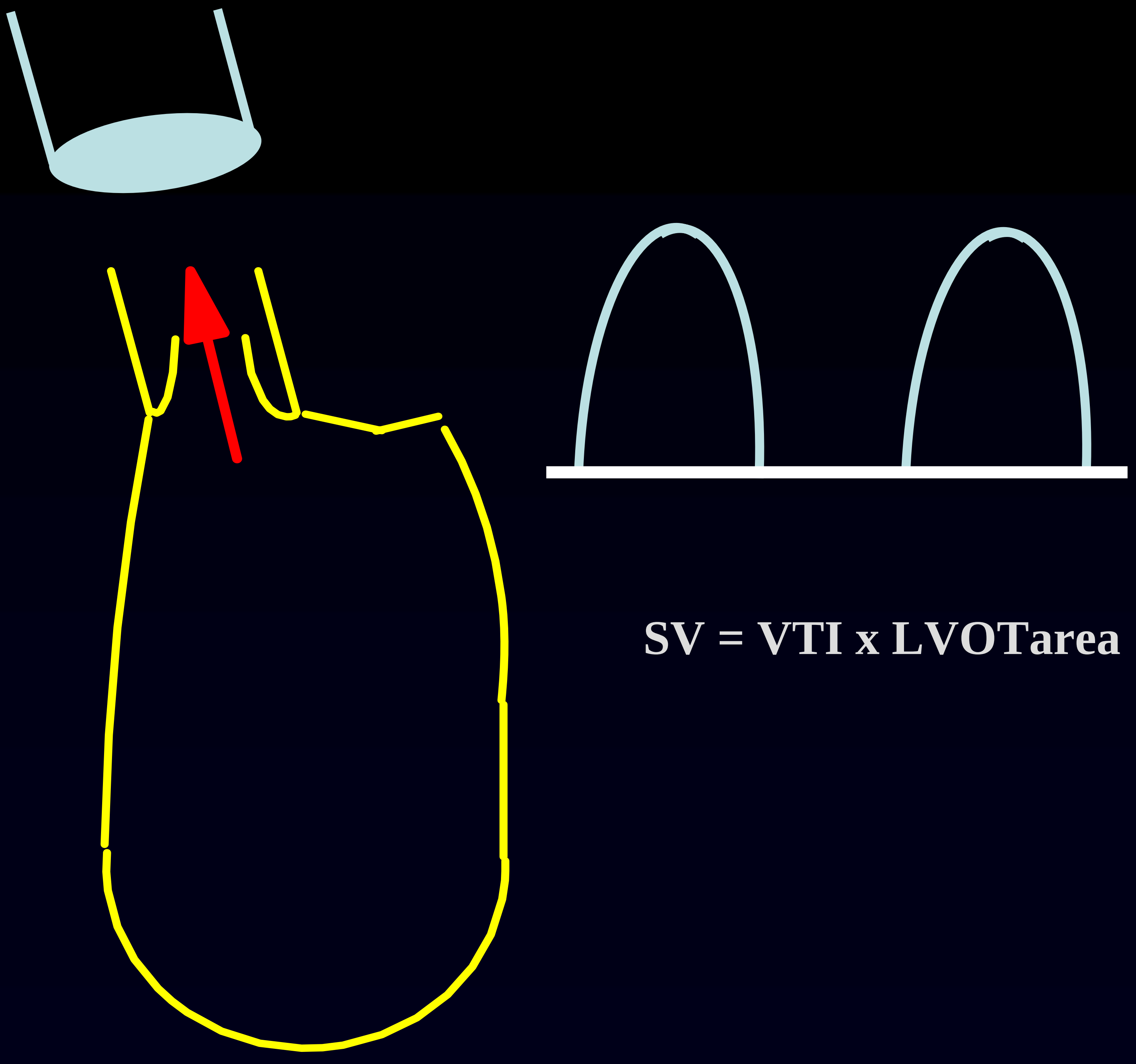
Hemodynamic monitoring in septic shock

- Measurement of cardiac output
- Measurements of determinants of CO
- Measurements of cardiac function ?

CARDIAC OUTPUT

Key determinant of tissue oxygenation

(DO_2 ~CO x Hb x SaO_2)

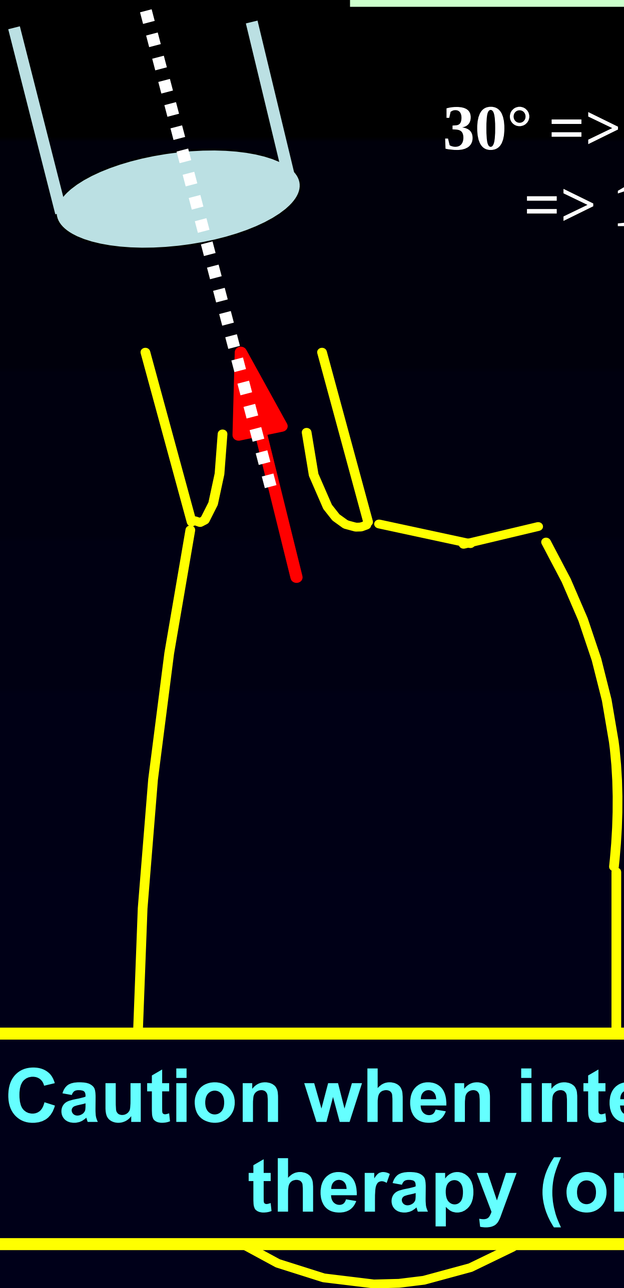


SV = VTI x LVOTarea

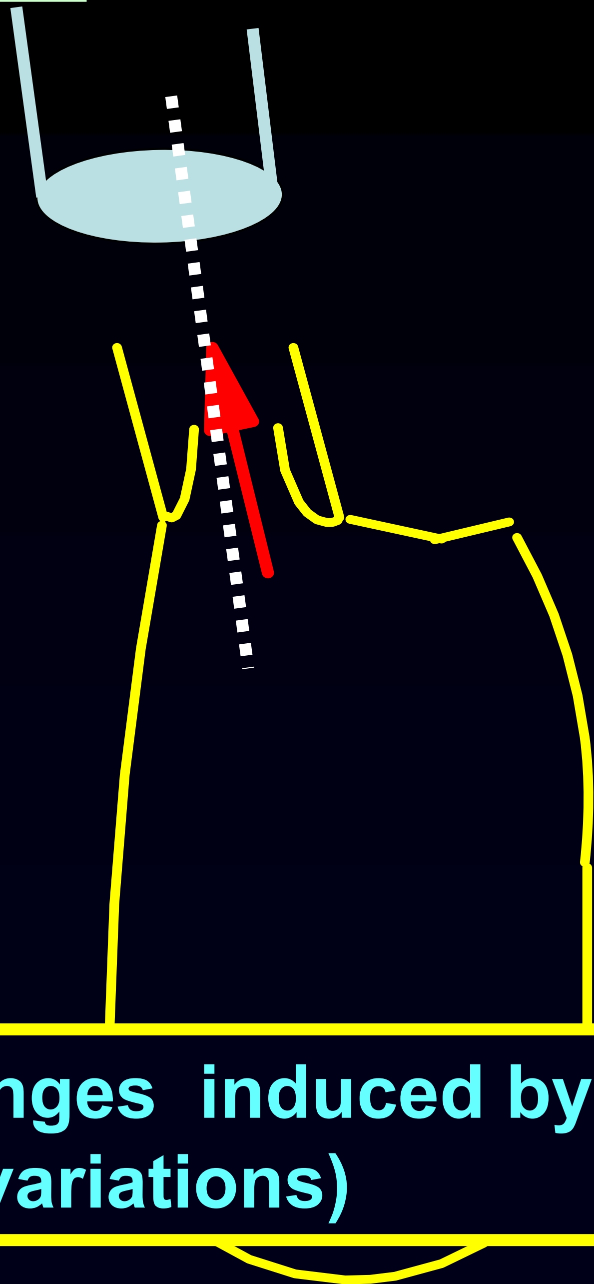
Potential problems:

- **Angle of the beam**

Flow(mes) = Flow x Cos angle



30° => cos 30 = 0.87
=> 13% error



Caution when interpreting changes induced by therapy (or respiratory variations)

Potential problems:

- **Angle of the beam**
- **Measurement of LVOT**

Measurement of LVOT

⇒LVOT area: $\pi D^2/4$

A small error is elevated to the square !

VTI	HR	LVOT	$\pi D^2/4$	CO
17	100	1.7	2.27	3.86
17	100	1.8	2.54	4.33
17	100	1.9	2.84	4.82
17	100	2.0	3.14	5.34
17	100	2.1	3.46	5.89

Measurement of LVOT

⇒LVOT area: pD²/4
A small error is elevated to the square

=> Always use same value or just VTI ?

Heart rate can also vary !
=> Changes in ejected volume
may differ from changes in CO

Potential problems:

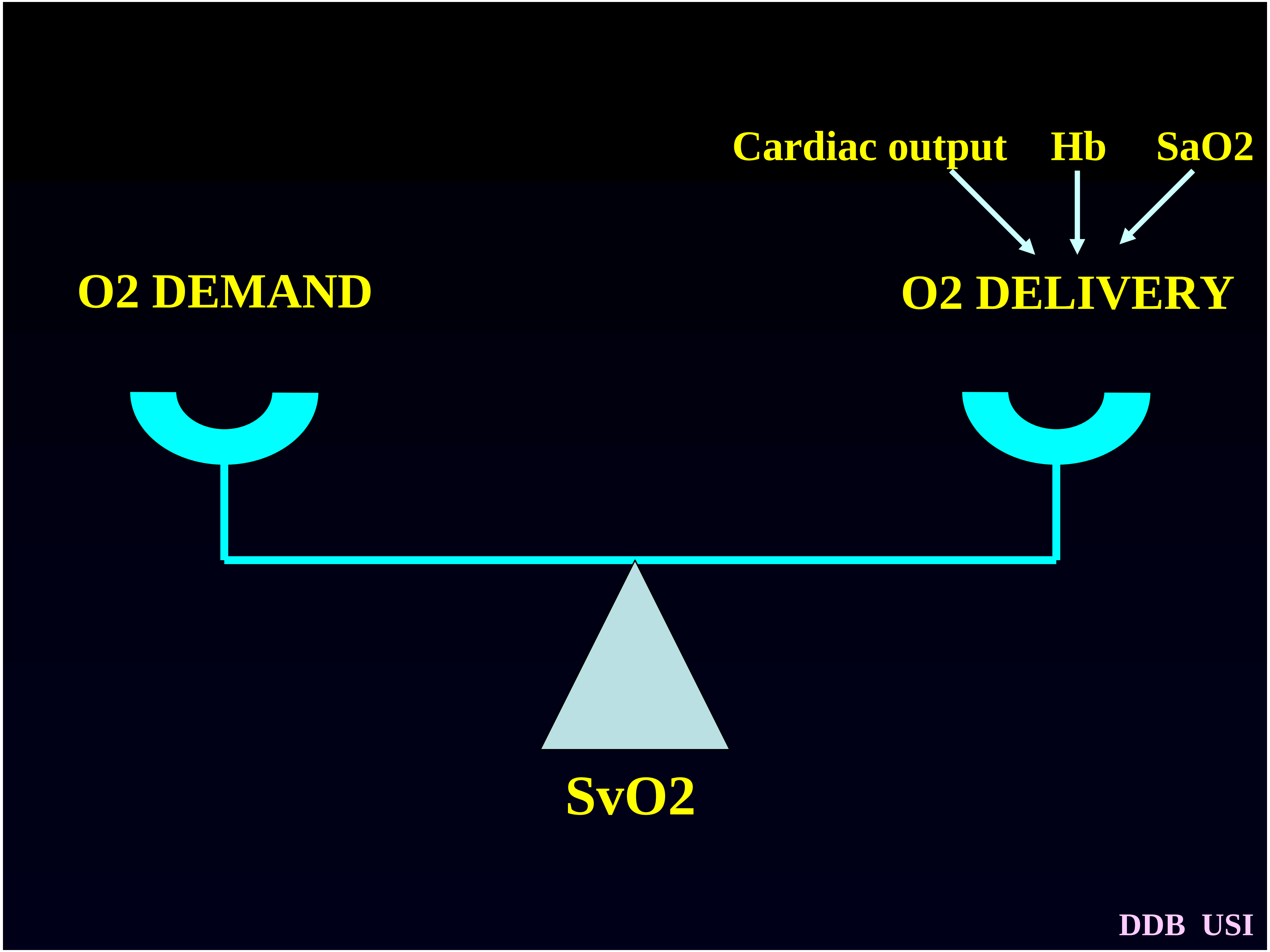
- Angle of the beam
- Measurement of LVOT => always use same value or
- Difficult to repeat (time consuming)

A value of CO alone is of limited value

Cardiac output measurements:

3.0 L/min.M²	Quiet, MV, 36.5°C, Hb 10
3.5 L/min.M²	Quiet, extubated, 36.5°C, Hb 10
4.0 L/min.M²	Quiet, MV, 39.5°C, Hb 10
4.5 L/min.M²	Dyspneic, MV, 38.5°C, Hb 9

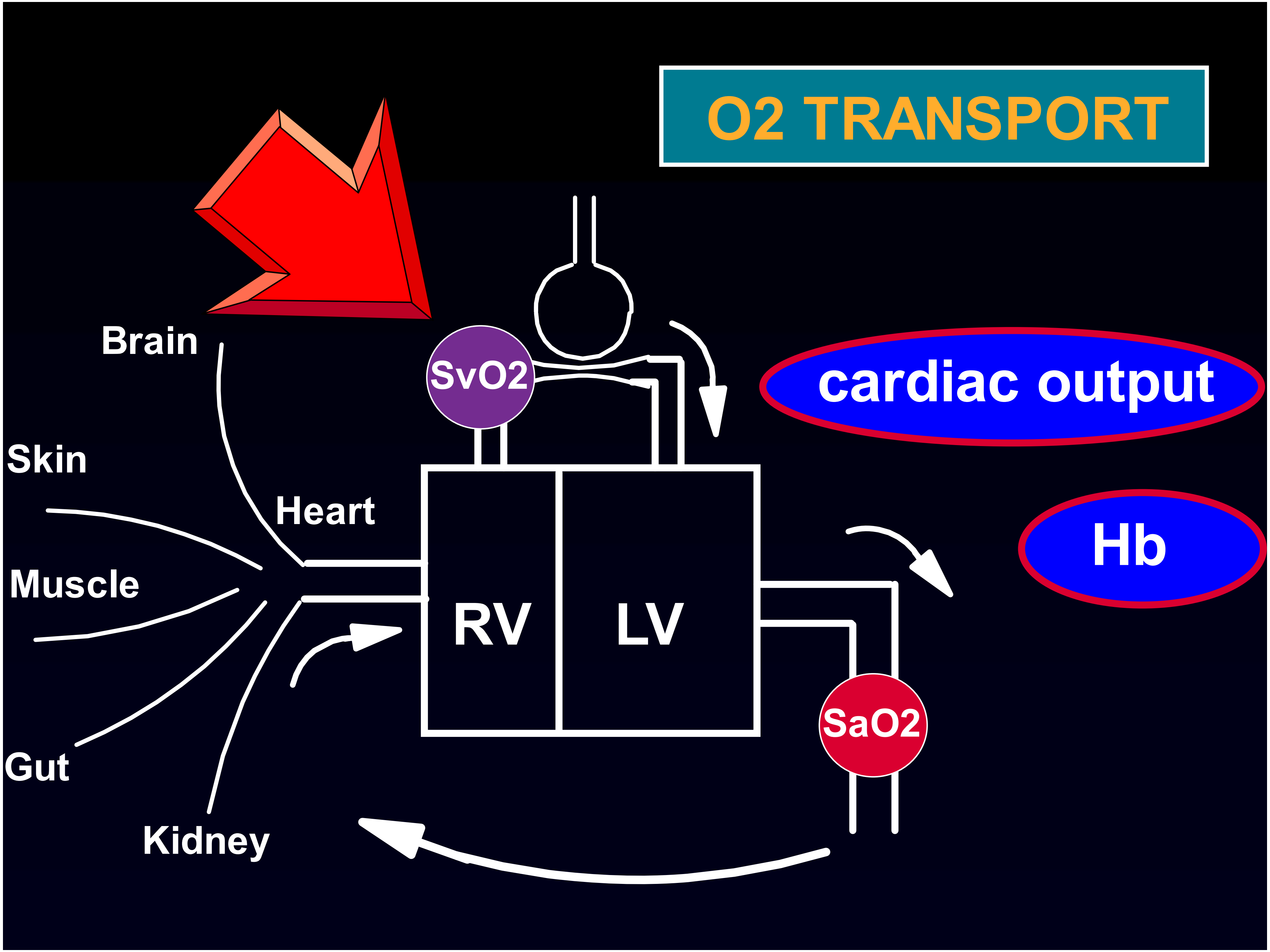
Is cardiac output adequate ?


Cardiac output
Hb
SaO2
O2 DEMAND
O2 DELIVERY
SvO2
DDB USI

Cardiac output measurements:

3.0 L/min.M²	Quiet, MV, 36.5°C, Hb 10	SvO2 70%
3.5 L/min.M²	Quiet, extubated, 36.5°C, Hb 10	SvO2 70%
4.0 L/min.M²	Quiet, MV, 39.5°C, Hb 10	SvO2 60%
4.5 L/min.M²	Dyspneic, MV, 38.5°C, Hb 9	SvO2 35%

Is cardiac output adequate ?

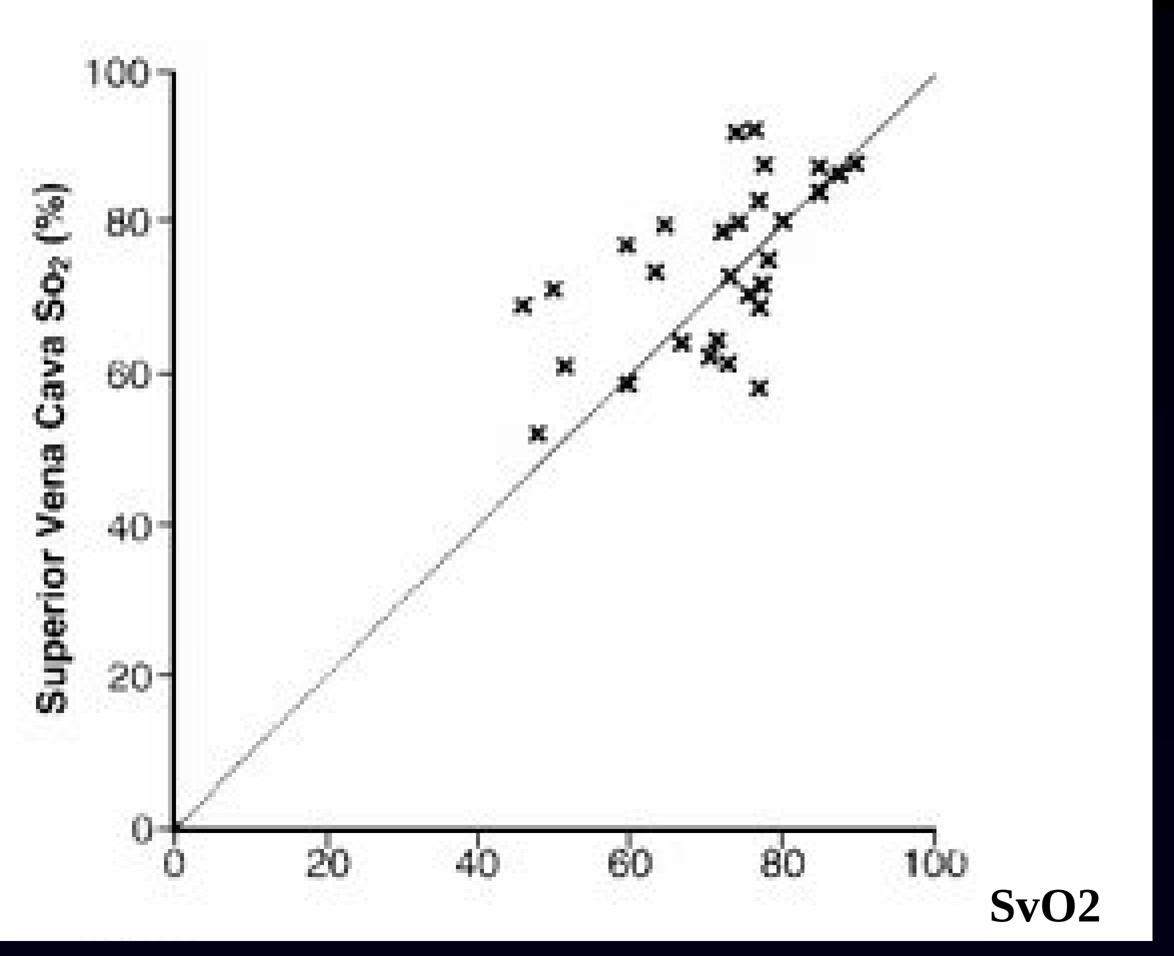

O2 TRANSPORT
Brain
Skin
Heart
Muscle
Gut
Kidney
SvO2
RV
LV
SaO2
cardiac output
Hb

SvO2 cannot be measured without a pulmonary artery catheter.

=> Can SvcO2 be a surrogate ?

SvO2 and SvcO2 are related but not identical

Edwards et al
CCM 26:1356;1998



30 pts with circulatory shock

SvcO2 surrogate of SvO2 ?

Edwards et al
CCM 26:1356;1998

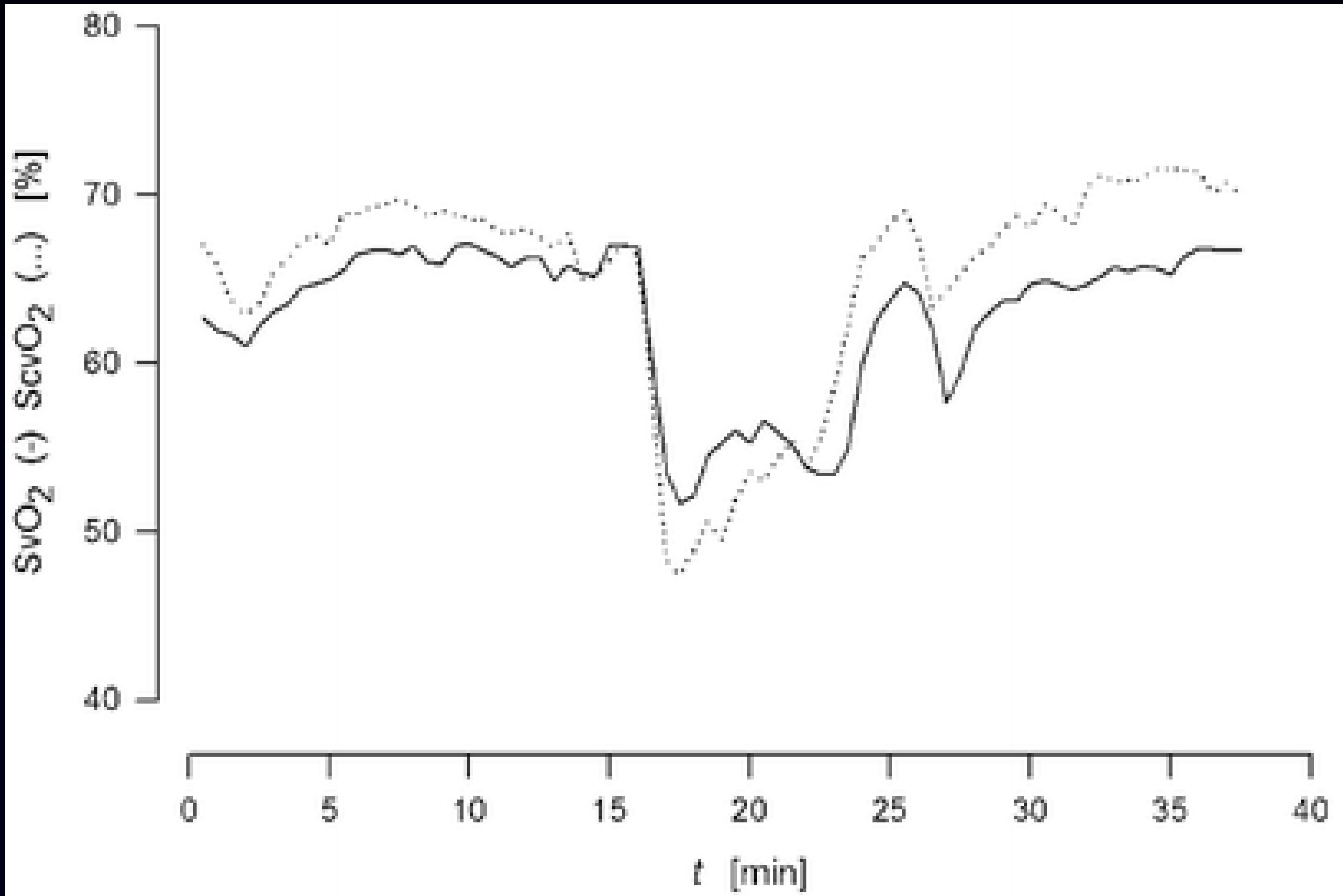
	Oxygen Saturation (% Difference)		
	Mean ± SD	Range	95% CI
Ssvc-Spa	2.9 ± 10.4	–19.3–+23.1	–18.4–+24.2
Sra-Spa	–0.7 ± 8.8	–19.7–+16.7	–18.6–+17.3
Ssvc-Sra	3.6 ± 8.8	–14.3–+32.5	–14.4–+21.6

CI, confidence interval; Ssvc, superior vena caval oxyhemoglobin saturation; Spa, pulmonary artery oxyhemoglobin saturation; Sra, right atrial oxyhemoglobin saturation.

30 pts with circulatory shock

Do changes in SvcO2 track changes in SvO2 ?

Reinhart et al
ICM 30:1572;2004



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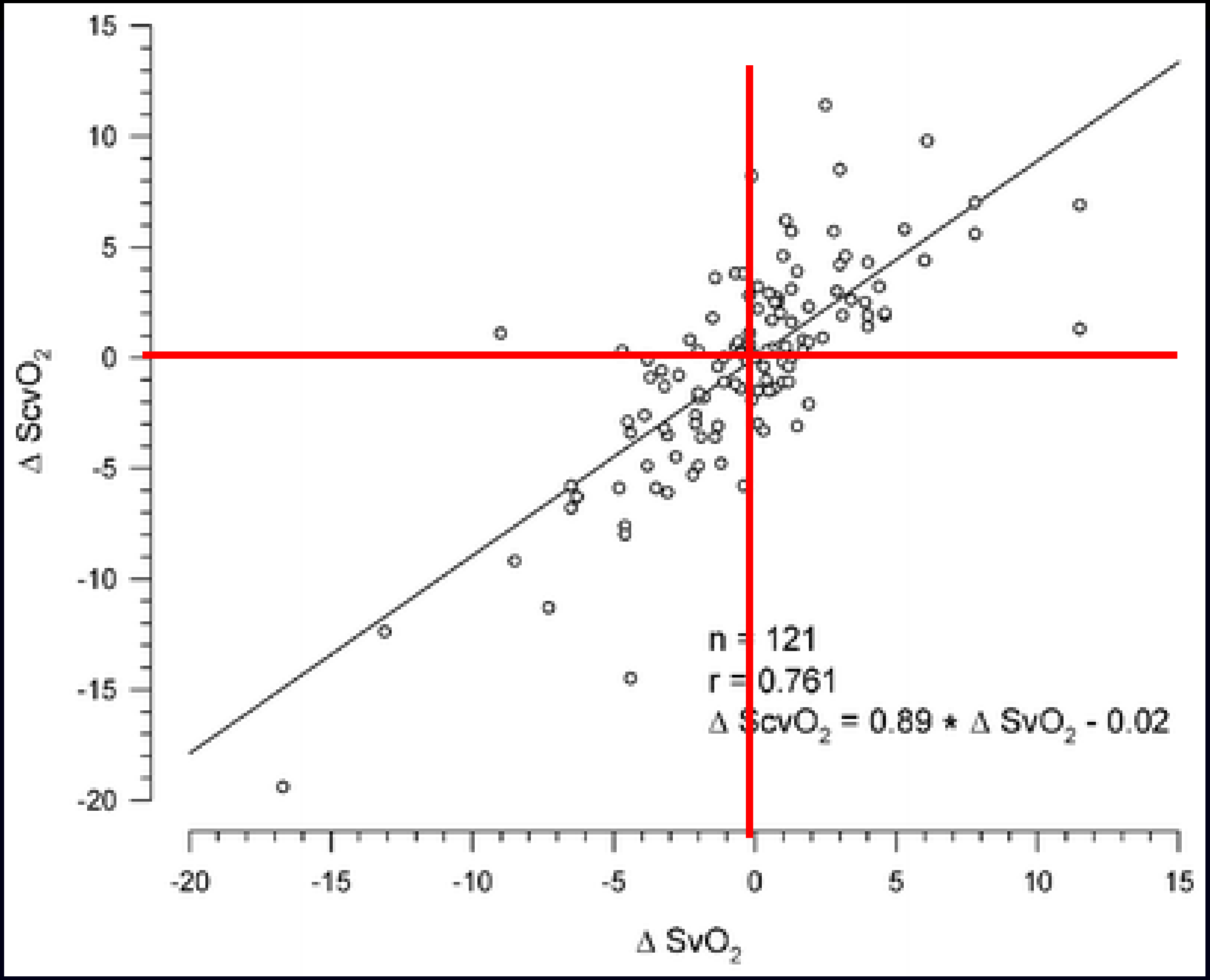

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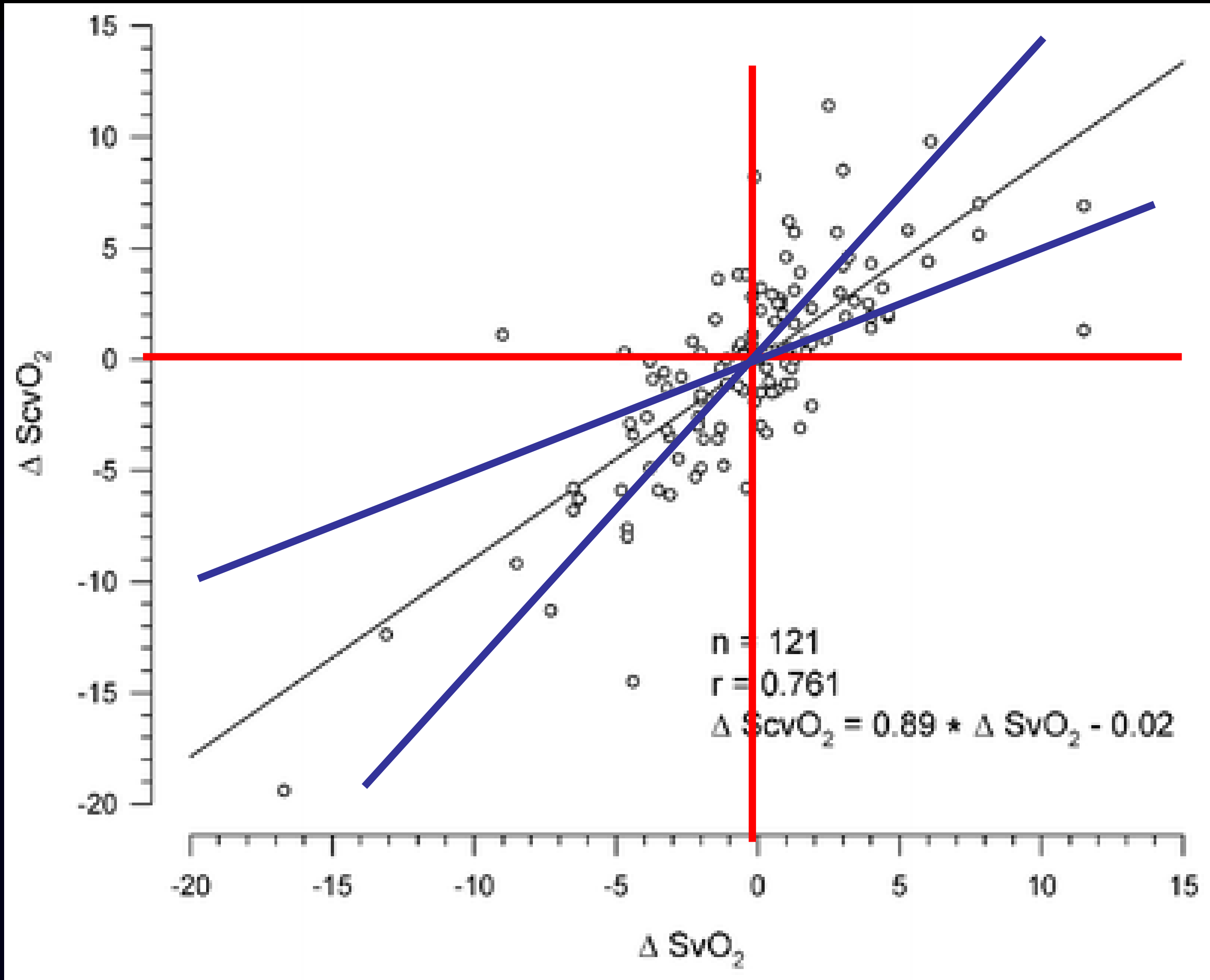
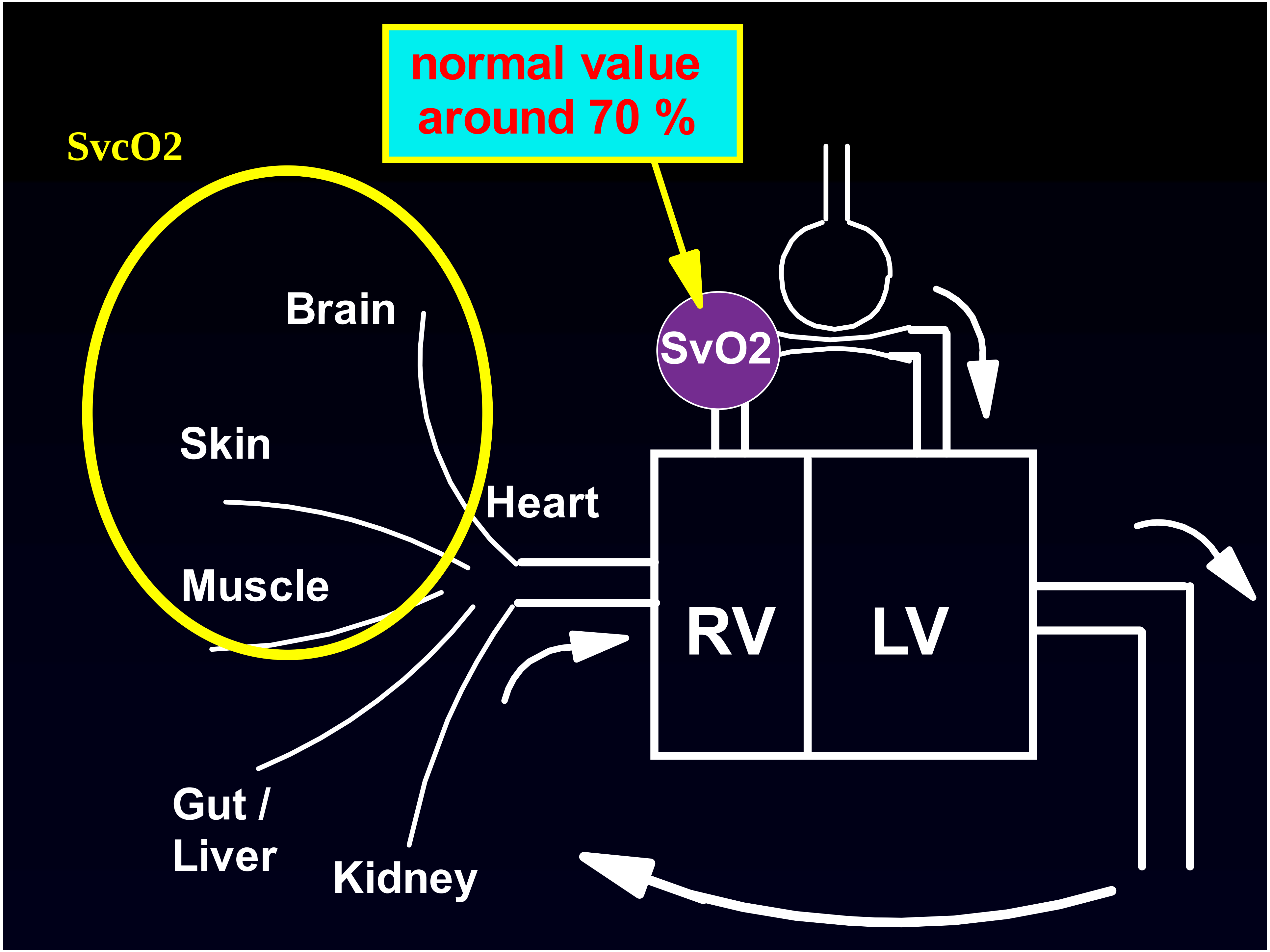


50% over- or under- estimation of SvO2 changes



SvcO2
normal value
around 70 %
SvO2
Brain
Skin
Heart
Muscle
RV
LV
Gut /
Liver
Kidney

HEMODYNAMIC MONITORING OF THE PATIENT IN SEPTIC SHOCK

- A low EF (in isolation) should not be treated
- A value of CO alone is of limited value
- CO measurements should always be combined with ScvO2 measurements

If CO is inadequate, what can I do for my patient ?

DETERMINANTS OF CARDIAC OUTPUT

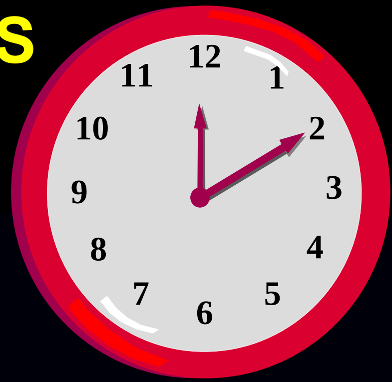
DETERMINANTS OF CARDIAC OUTPUT

- **Ejected volume**
  - **Preload**
  - **Afterload**
  - **Contractility**
- **Heart rate**

DETERMINATION OF CARDIAC FUNCTION

HEMODYNAMIC OPTIMIZATION

HEMODYNAMIC RESUSCITATION IN SEPSIS



THE SEPTIC CLOCK

The golden hour(s)

After the initial phase of resuscitation, no data support the use of any monitoring strategy or resuscitation goal !

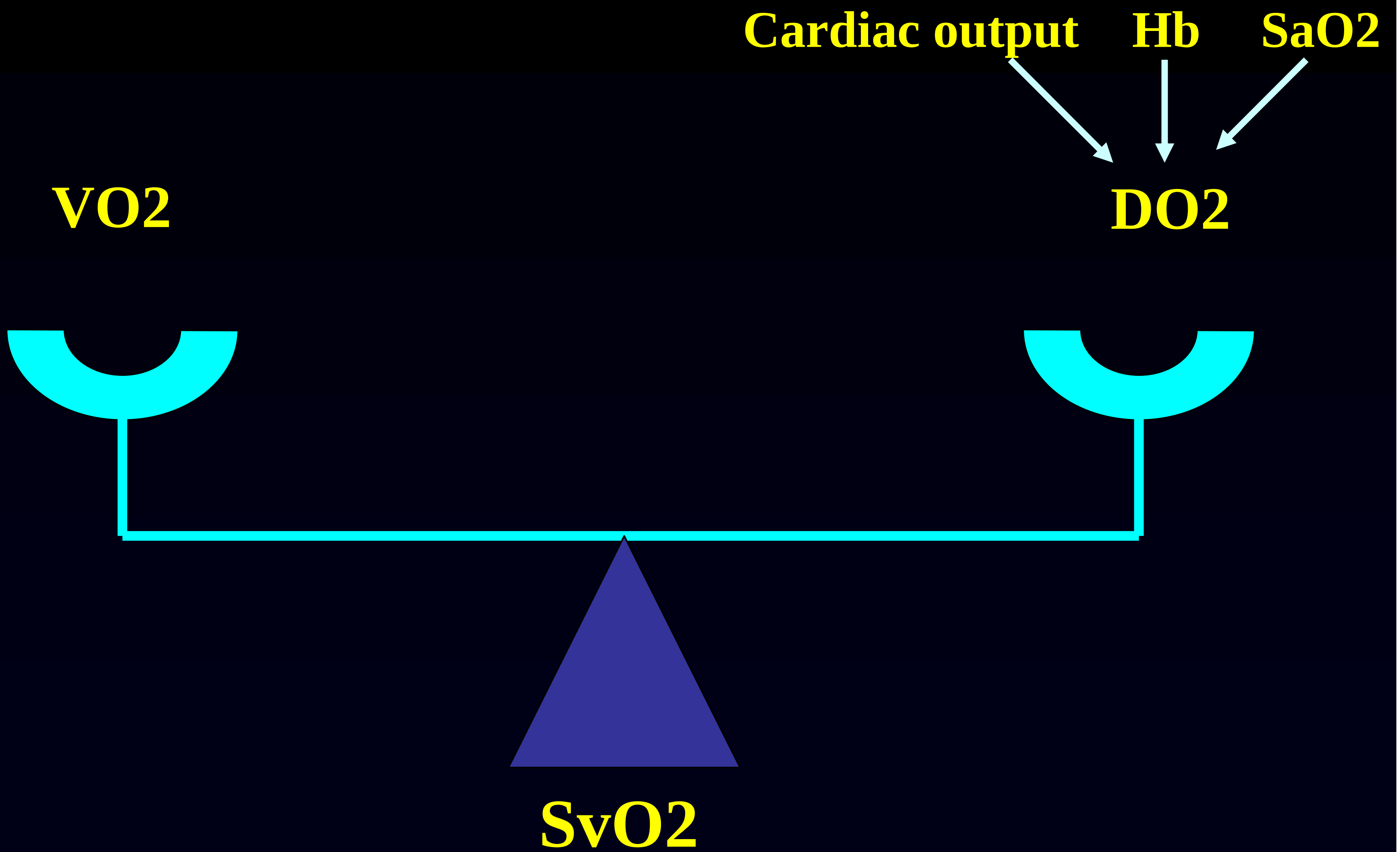
It seems reasonable to provide *some* hemodynamic support in order to prevent further tissue hypoperfusion, but hemodynamic optimization is not useful at this stage.

HEMODYNAMIC RESUSCITATION IN SEPSIS

Q1: Is cardiac output adequate ?

SvO2
Lactate

Should cardiac output be manipulated ?



DDB USI

HEMODYNAMIC RESUSCITATION IN SEPSIS

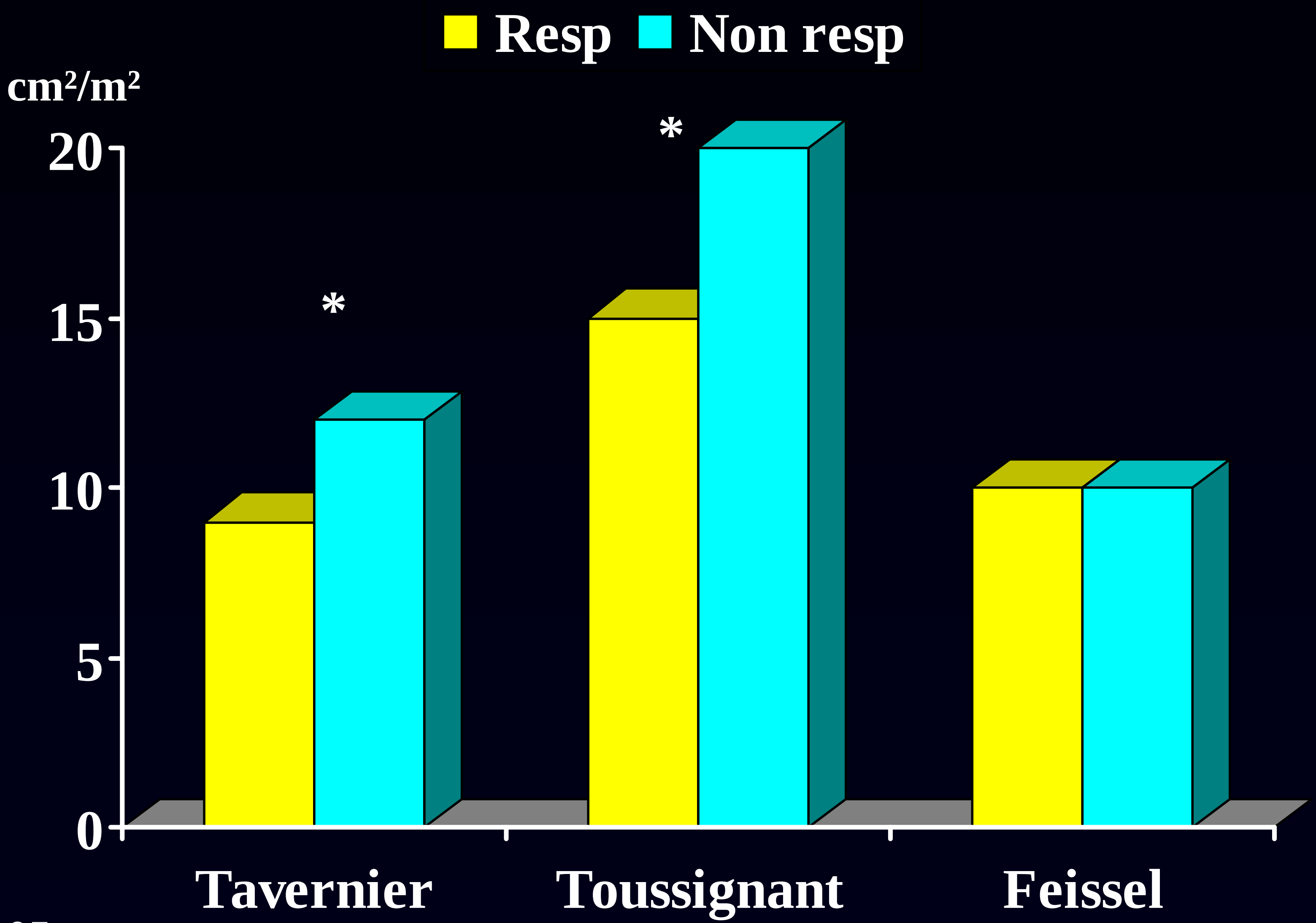
Q1: Is cardiac output adequate ?

Q2: How to manipulate CO?

Assess fluid responsiveness
Assess contractility

Left ventricular end diastolic area

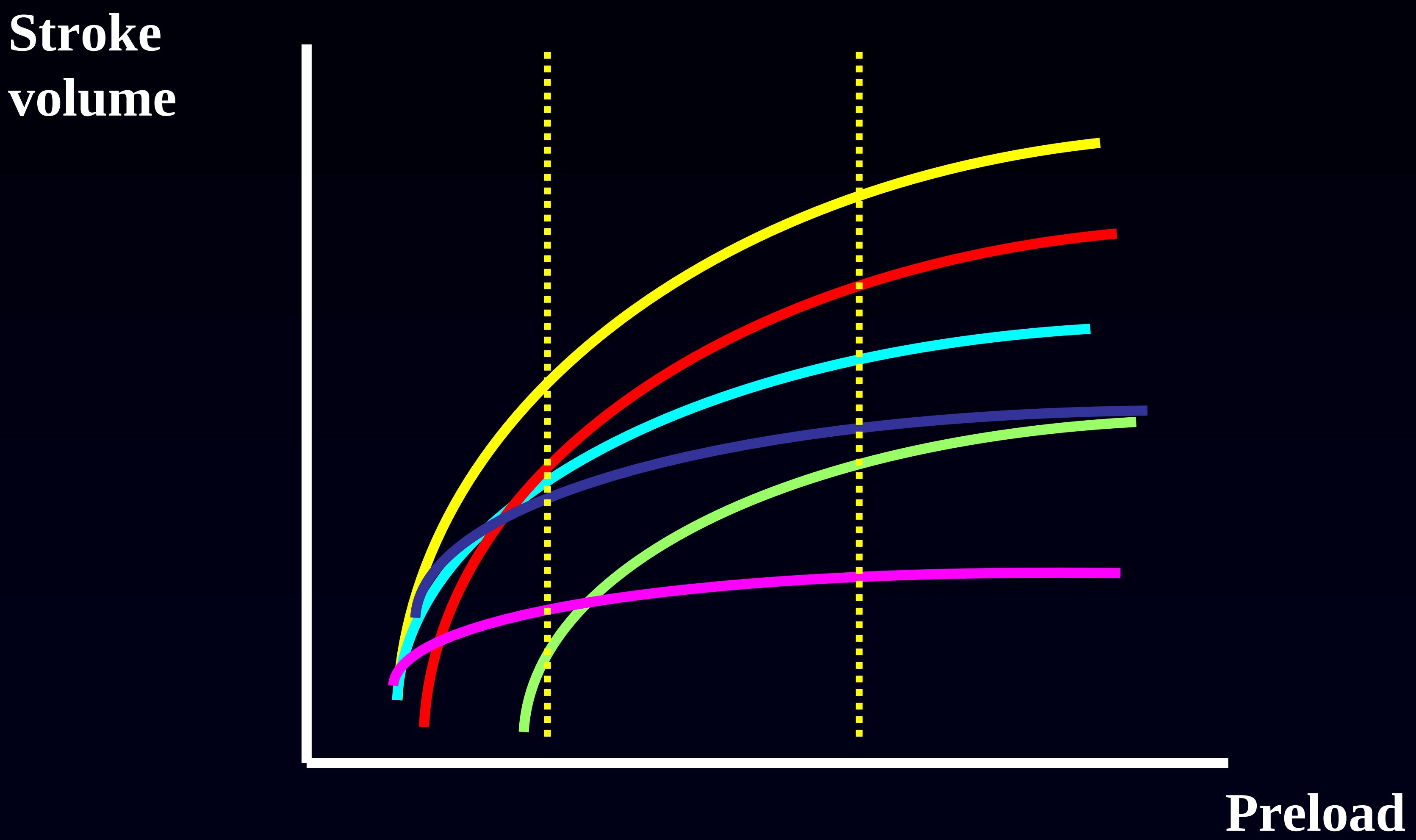
Michard et al
Chest 121:2000;2002



* p<0.05

DDB USI

STARLING RELATIONSHIP



All indices of preload poorly predict fluid responsiveness !

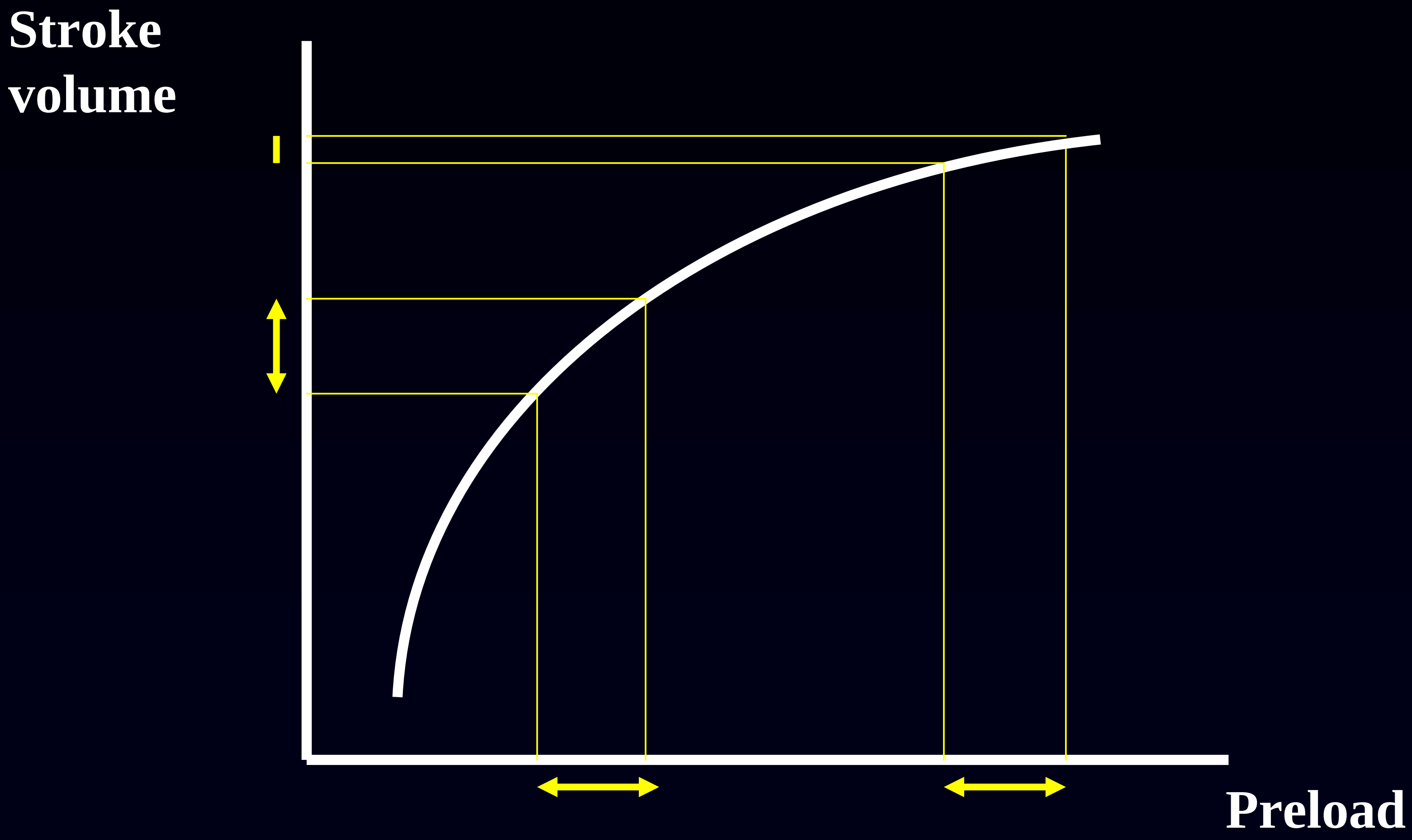
STROKE VOLUME VARIATIONS

Principle

- Ventilation induces changes in preload
- Respiratory variation of stroke volume will reflect preload-dependency (or fluid responsiveness)

DDB

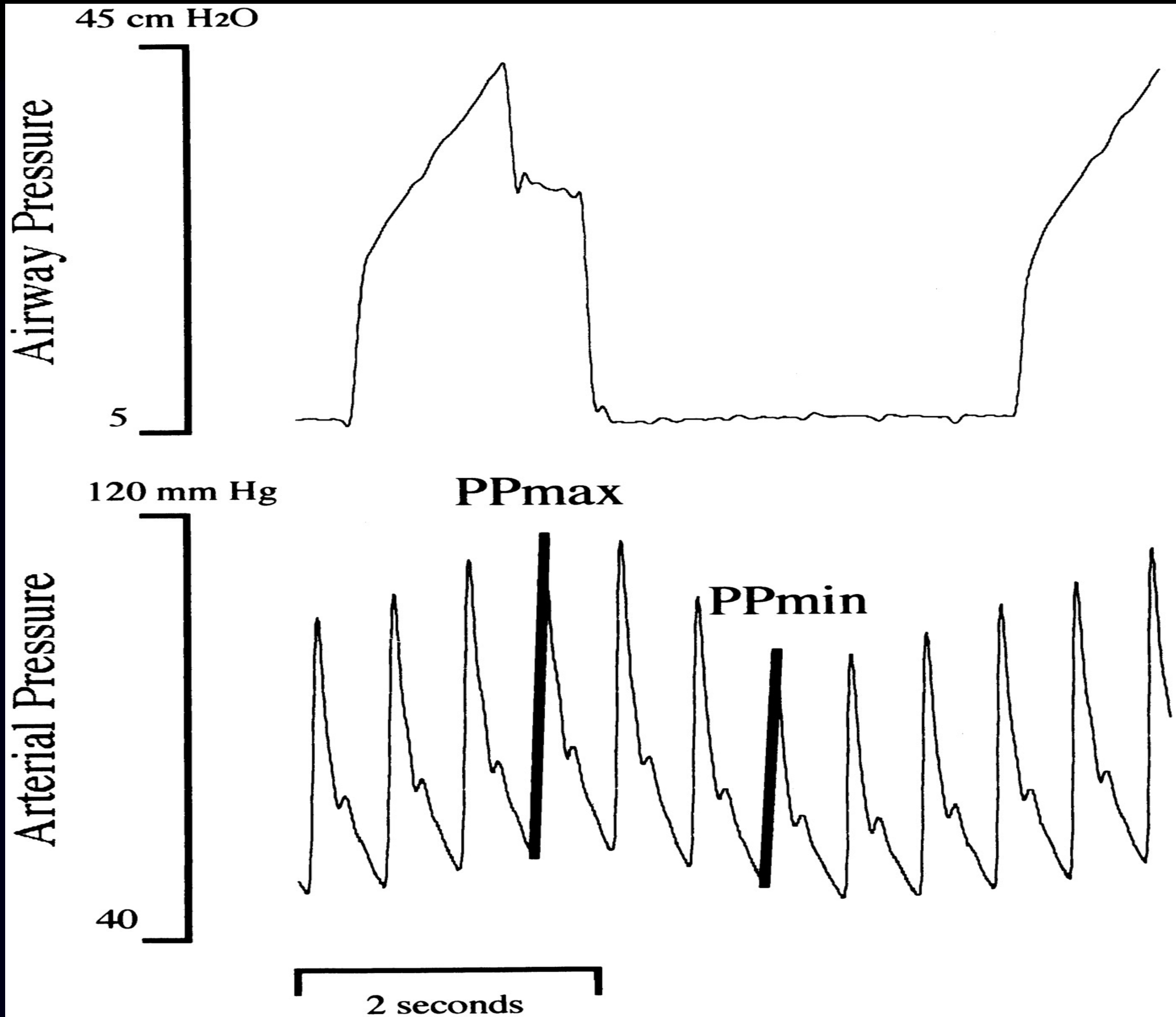
STROKE VOLUME VARIATIONS



DDB

Pulse pressure respiratory variations

Michard et al
AJRCCM 162:134;2000

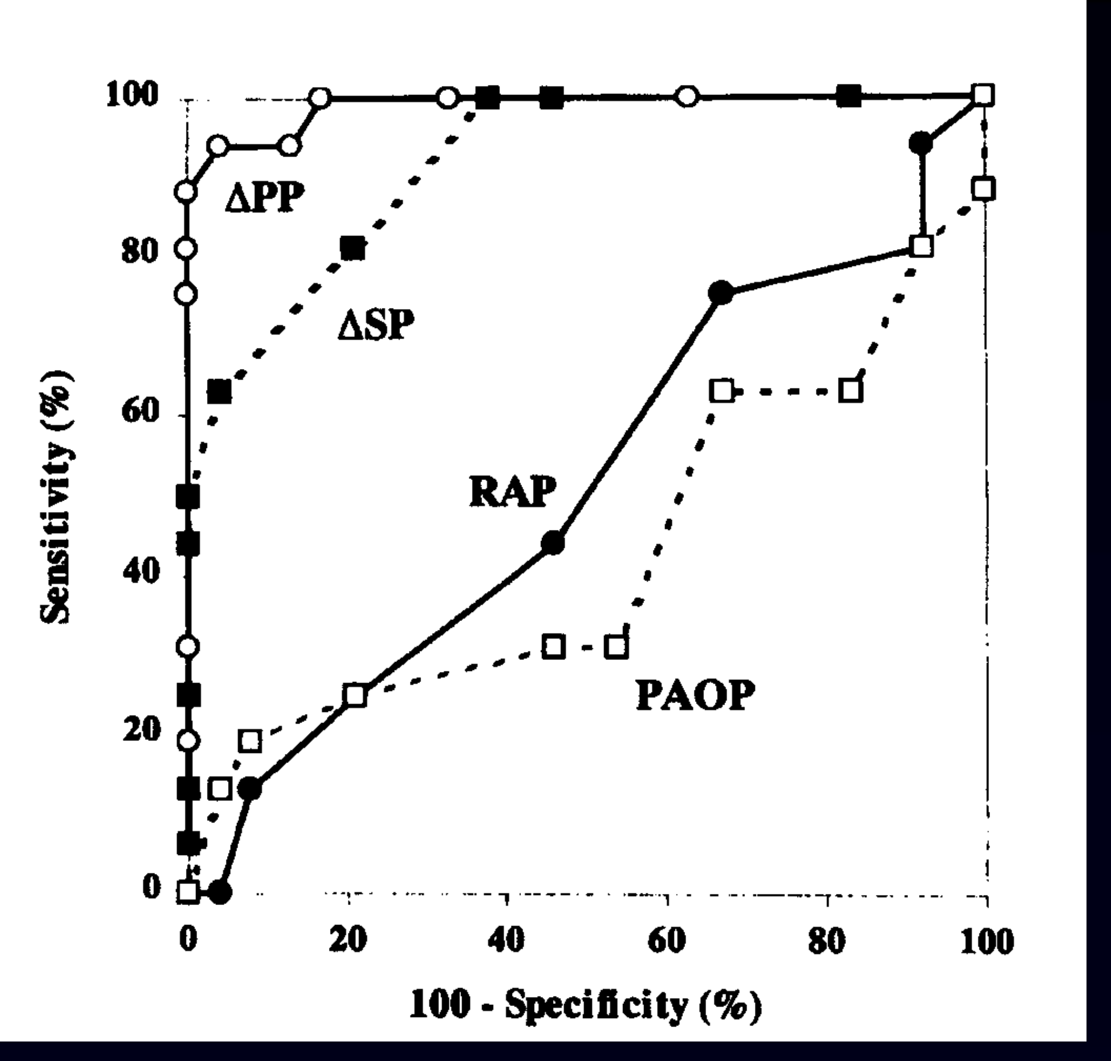


DeltaPP = (PPmax-PPmin) / ((PPmax+PPmin)/2)

DDB

SEPSIS

Michard et al
AJRCCM 162:134;2000

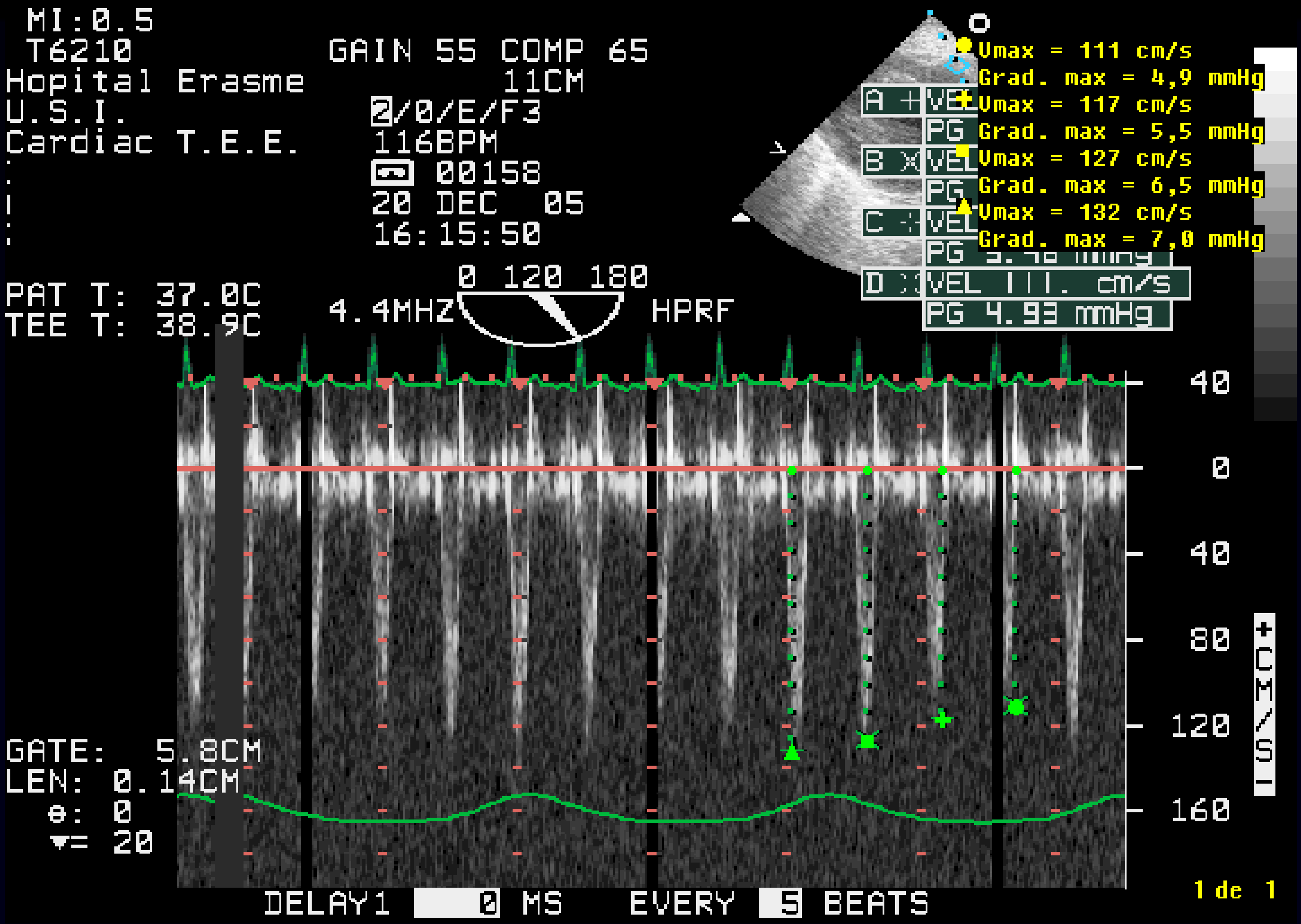


N= 40

Cut-off value: 13%

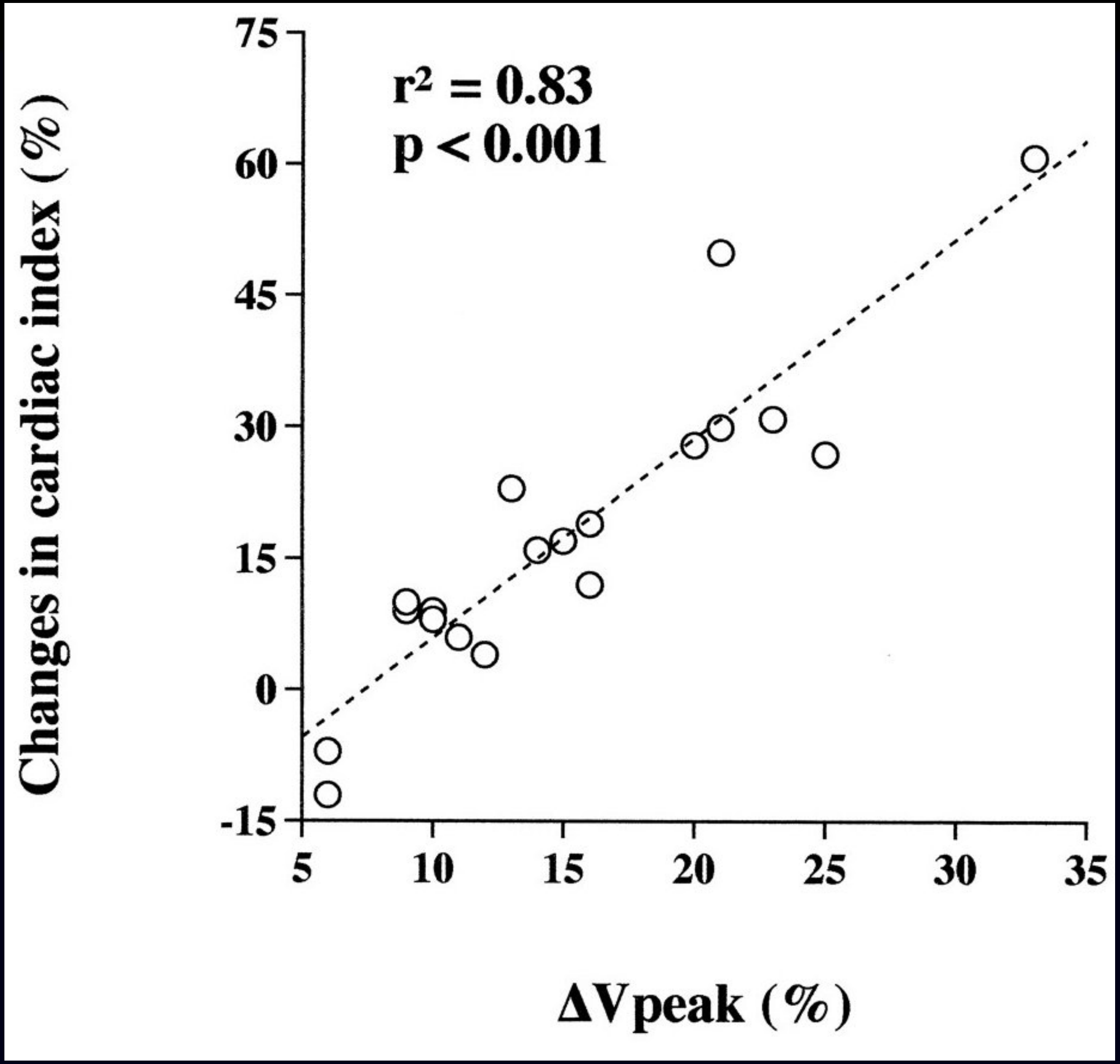
DDB

Aortic flow respiratory variations



SEPSIS

Feissel et al
Chest 119:867;2001



ECHO

N= 19

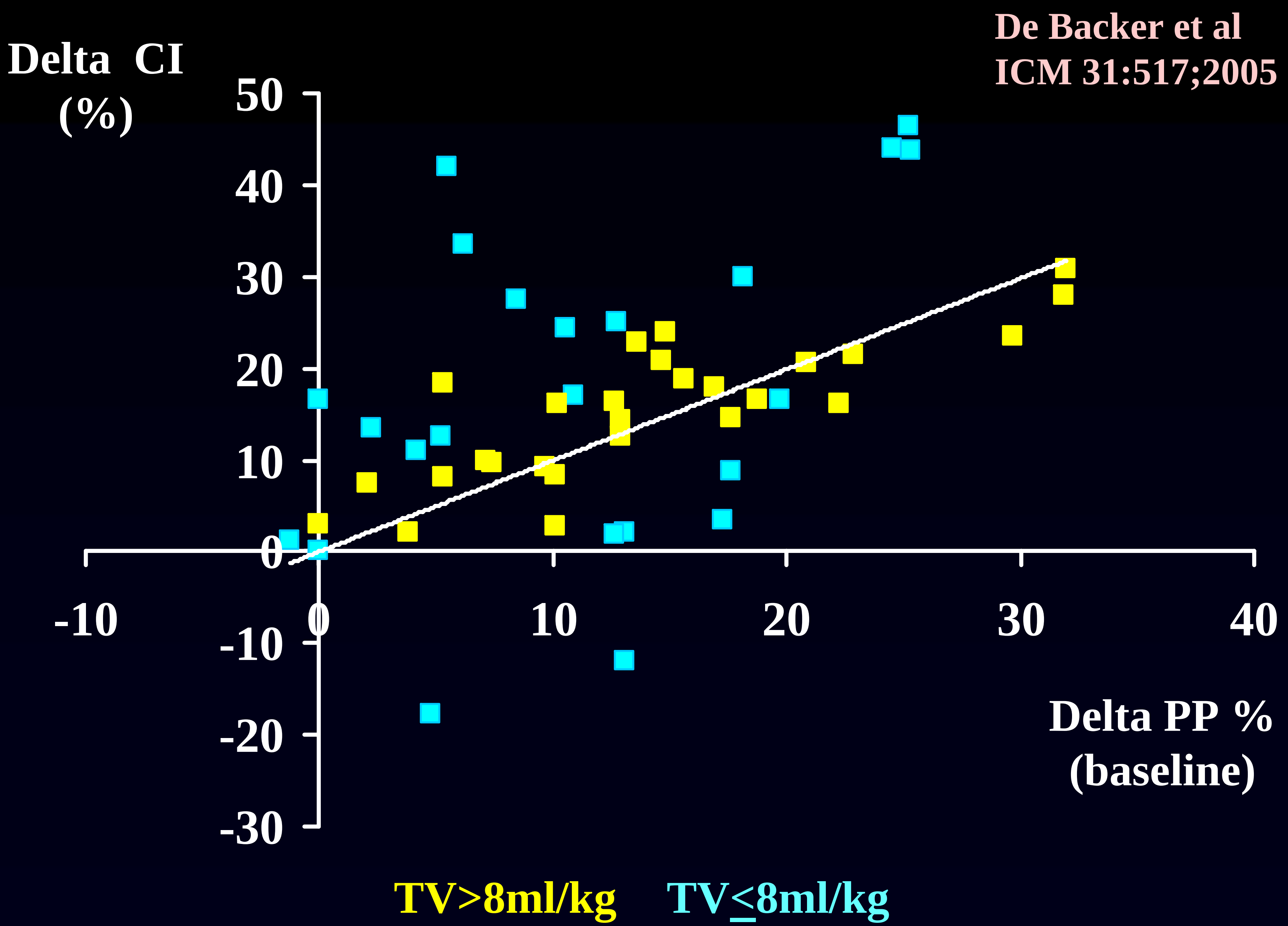
DDB

Limitations:

- **-Arrythmia**
- **-Low tidal volume**
- **-Respiratory movements**
- **-Cor pulmonale**

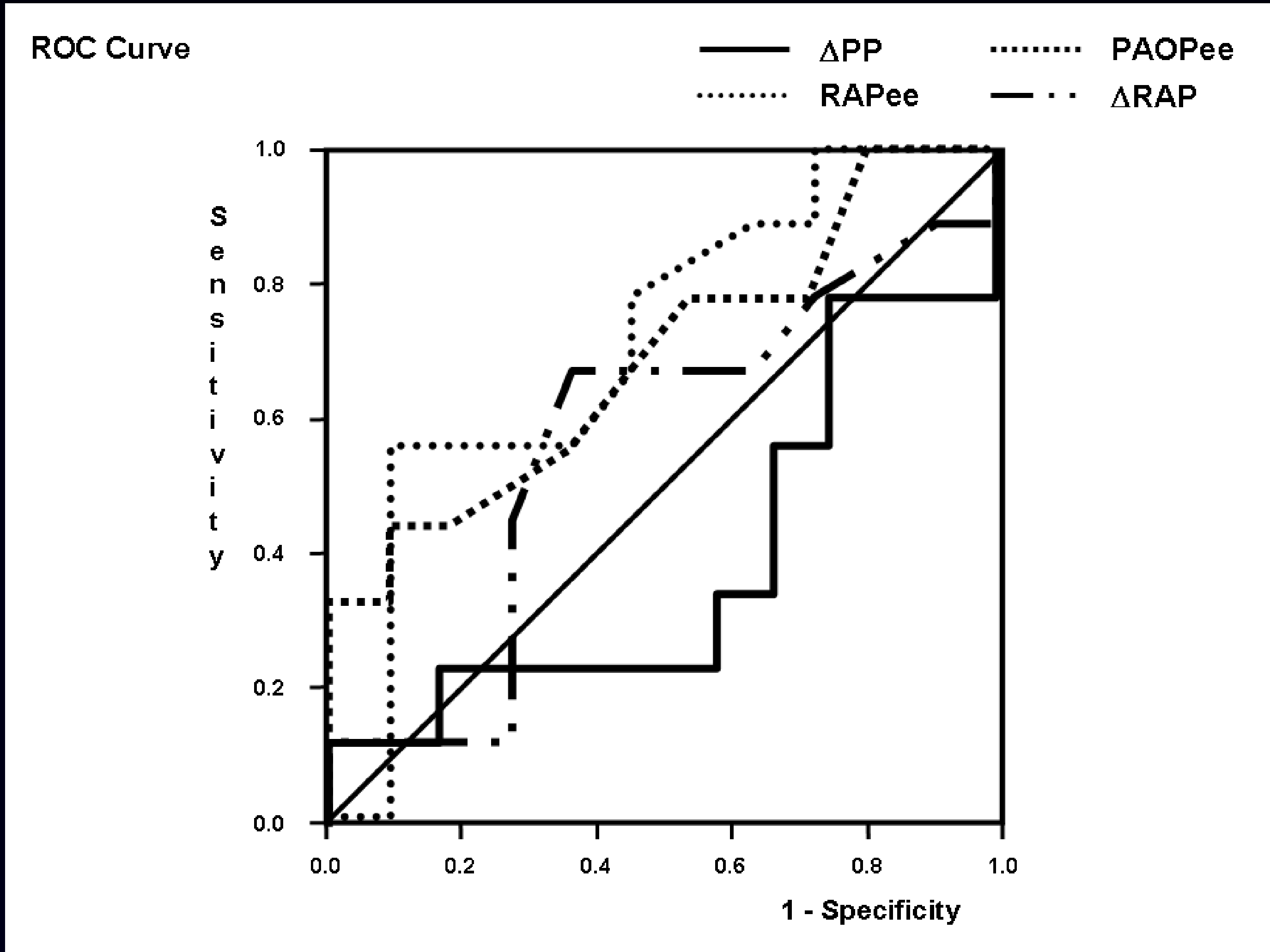
DDB

INFLUENCE OF TIDAL VOLUME

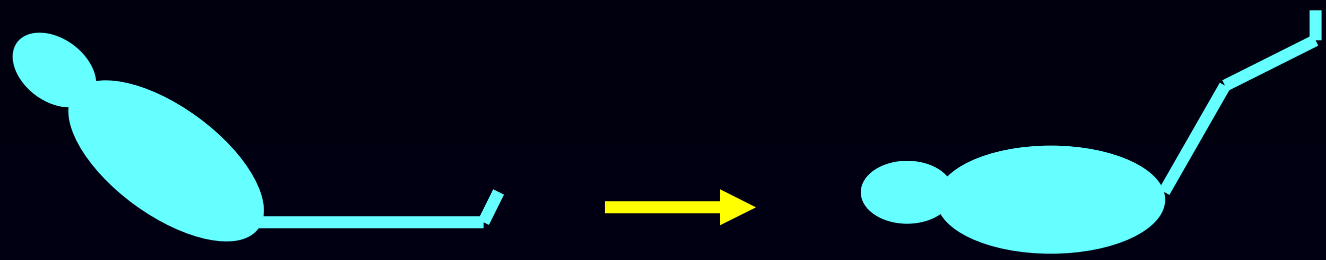


Prediction of fluid responsiveness in patients with respiratory movements

Heenen et al
Crit Care 2006



The passive leg raising test

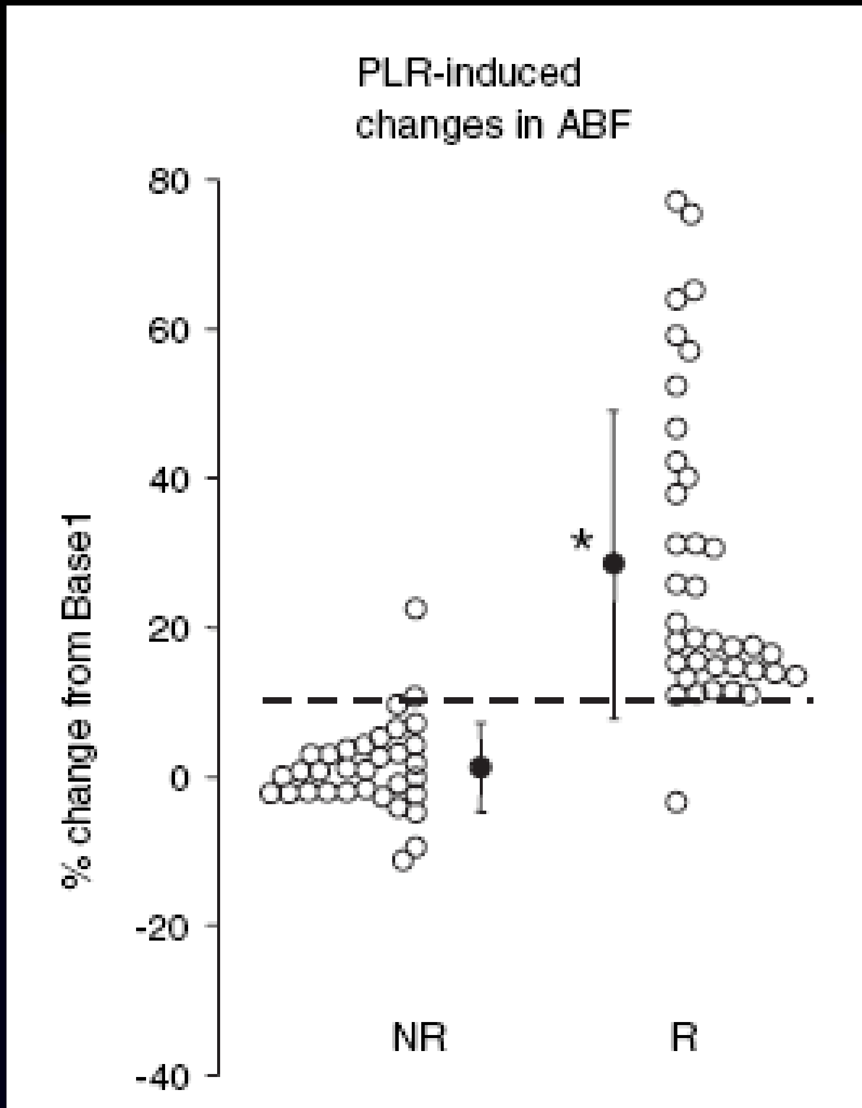


Autotransfusion of ~300 mL of blood

Measurement of cardiac output with fast response device (beat by beat) within 1 min of PLR (transient effect)

The passive leg raising test

Monnet et al
CCM 34:402;2006



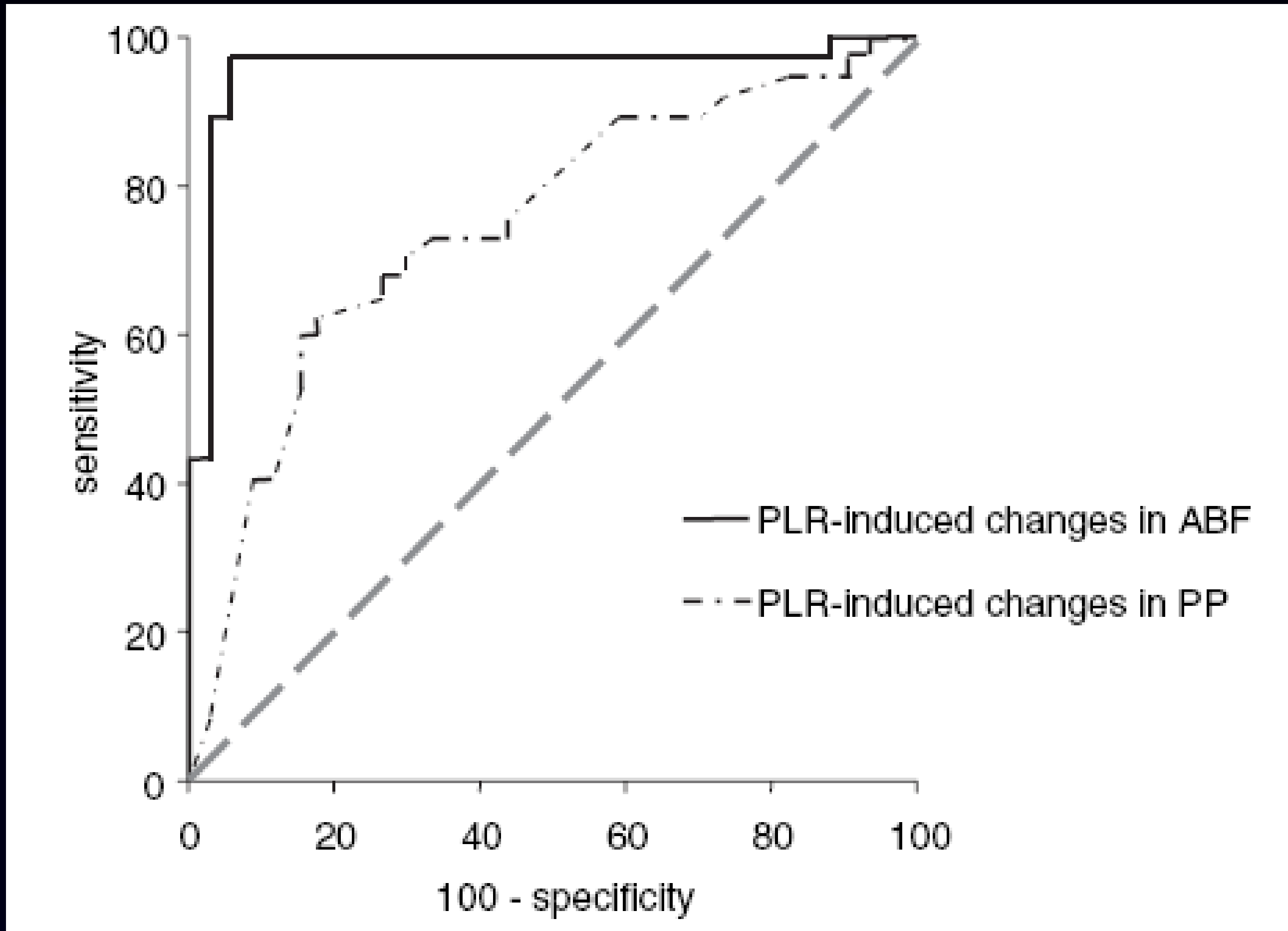
71 patients, 31 with respiratory movements

Fluids 500 mL cryst

The passive leg raising test

Monnet et al
CCM 34:402;2006

Measurement of CO should be preferred to indirect evaluation by PP



71 patients, 31 with respiratory movements

Fluids 500 mL cryst

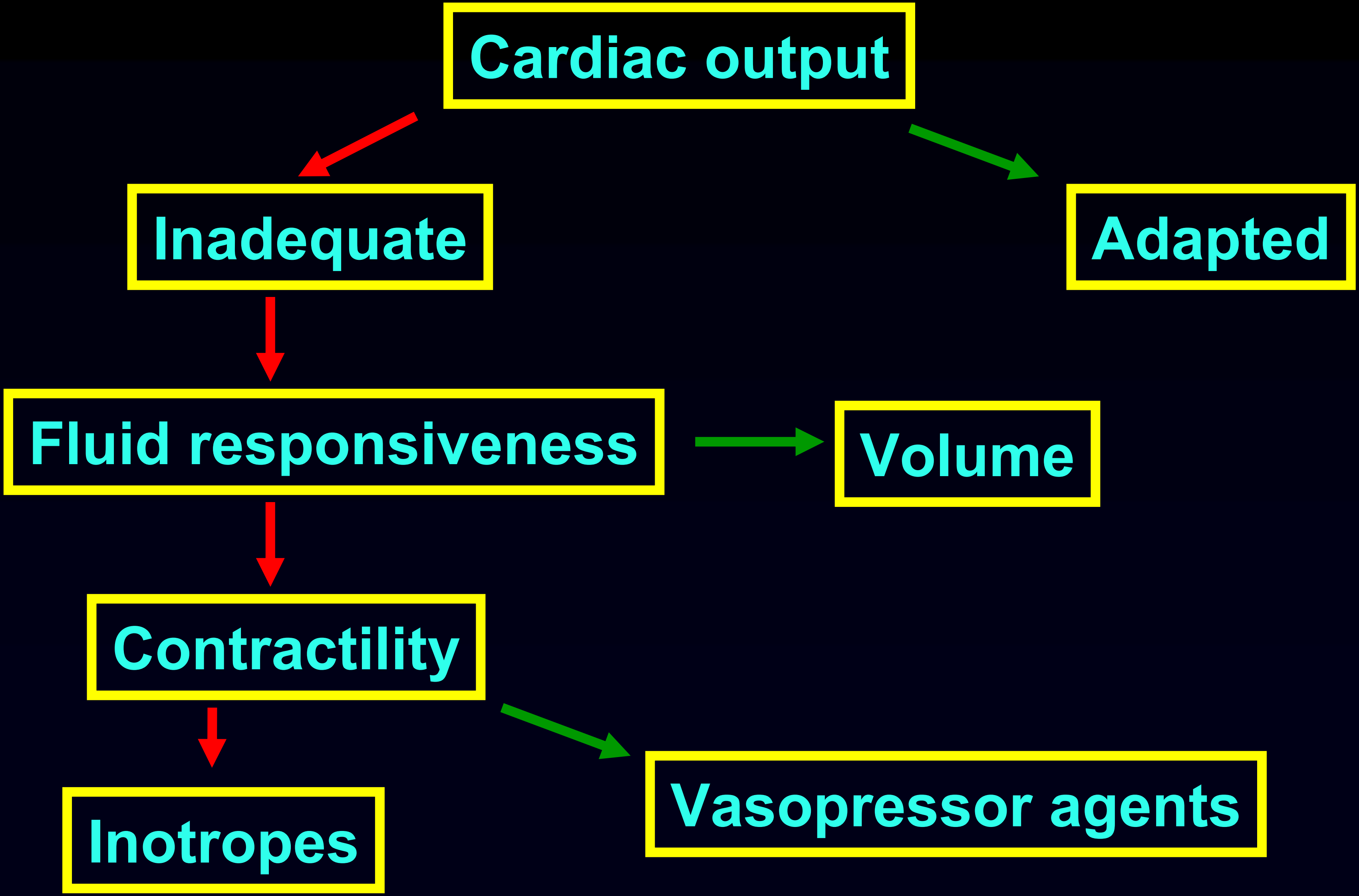
Inotropic support

Morelli et al
ICM 31:638;2005

	Levosimendan		Dobutamine	
	Baseline	24 h	Baseline	24 h
EDVI (ml m^{-1})	75.8±23.8	66.2±24.6*,**	84.2±25.1	82.9±26.4
ESVI (ml m^{-1})	46.7±21.9	36.9±19.4*,**	52.4±25.8	50.5±25.3
LVEF (%)	37.1±3.0	45.4±8.4*	37.3±2.6	40.8±11.3

*$p<0.05$ baseline vs. 24 h, **$p<0.05$ levosimendan vs. dobutamine after 24 h

Therapeutic approach



Echocardiography in sepsis:
Excellent for hemodynamic evaluation, but less convenient for monitoring

Hemodynamic evaluation should be retricted to simple indices !

Antoine Vieillard-Baron
Cyril Charron
Karim Chergui
Olivier Peyrouset
François Jardin

Bedside echocardiographic evaluation of hemodynamics in sepsis: is a qualitative evaluation sufficient?

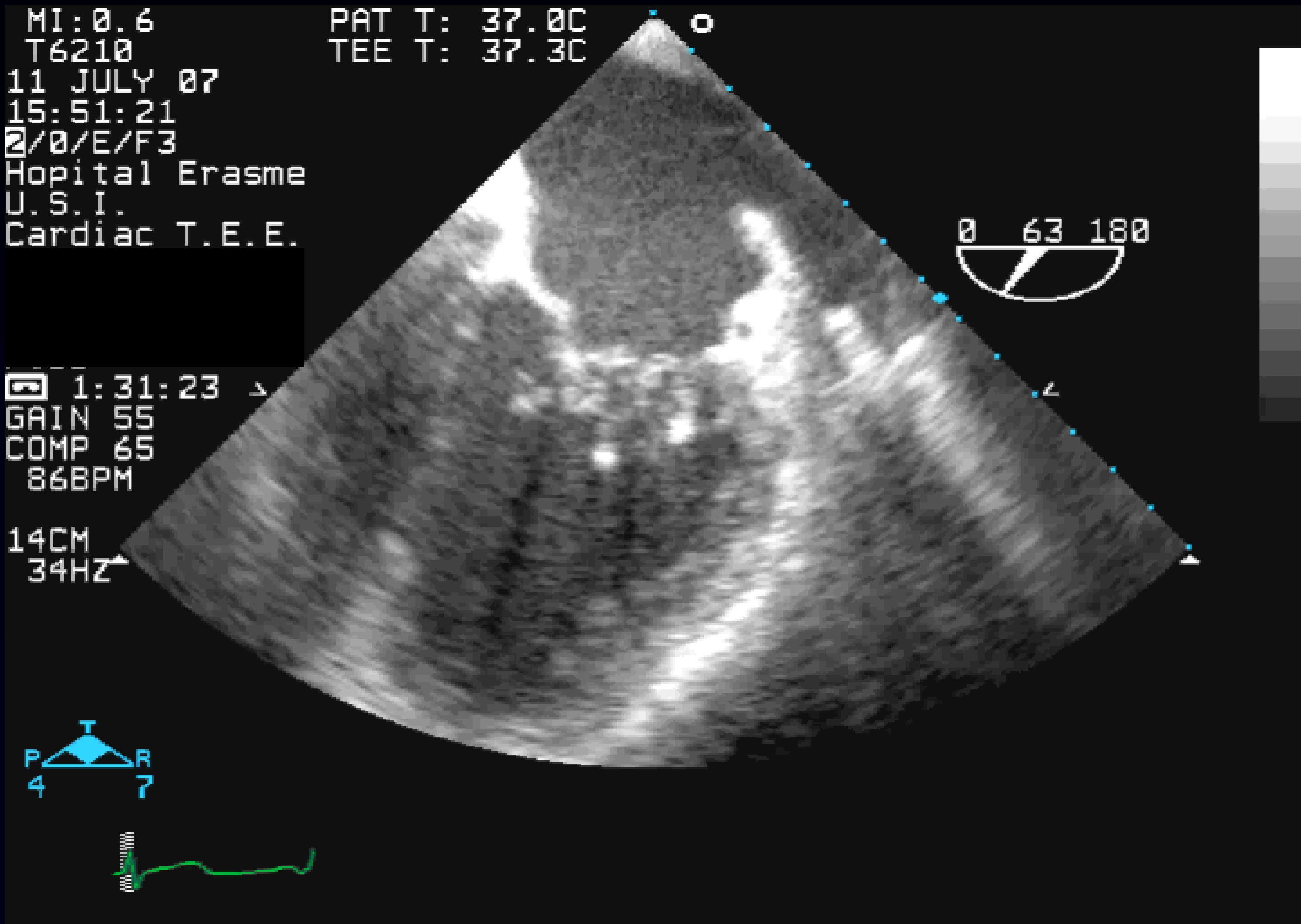
ICM 32:1547;2006

	Grade 0	Grade 1	Grade 2
SVC changes	57 (SVC CI 0%)	22 (SVC CI 19 ± 10%*)	4 (SVC CI 80 ± 1%**)
LV systolic function	38 (LVEF 63 ± 8%)	24 (LVEF 42 ± 5%*)	21 (LVEF 27 ± 8%**)
RV diastolic size	70 (EDA ratio 0.40 ± 0.12)	13 (EDA ratio 0.73 ± 0.1*)	0
IVS kinetics	81 (EI 1.07 ± 0.04)	2 (EI 1.9 and 1.5)*	–

* $p < 0.05$ vs. grade 0, ** $p < 0.05$ vs. grade 1

Echocardiography in sepsis:
its interest is not restricted to hemodynamic assessment !

Shock and respiratory failure developping 7 days after peritonitis



Shock and respiratory failure developping 7 days after peritonitis