Hemodynamics and Mechanical Ventilation

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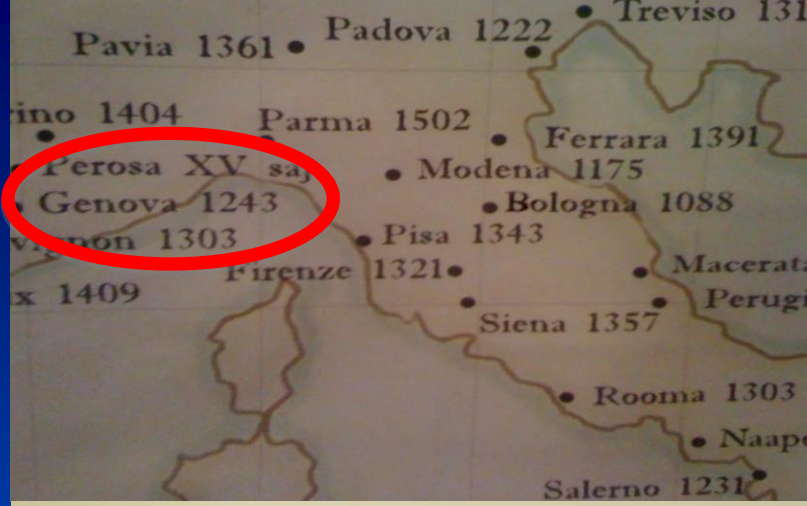
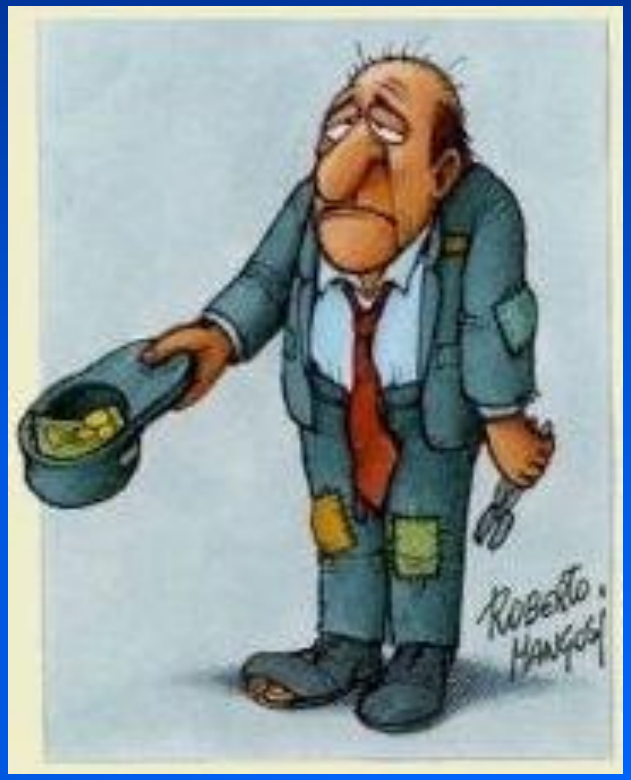


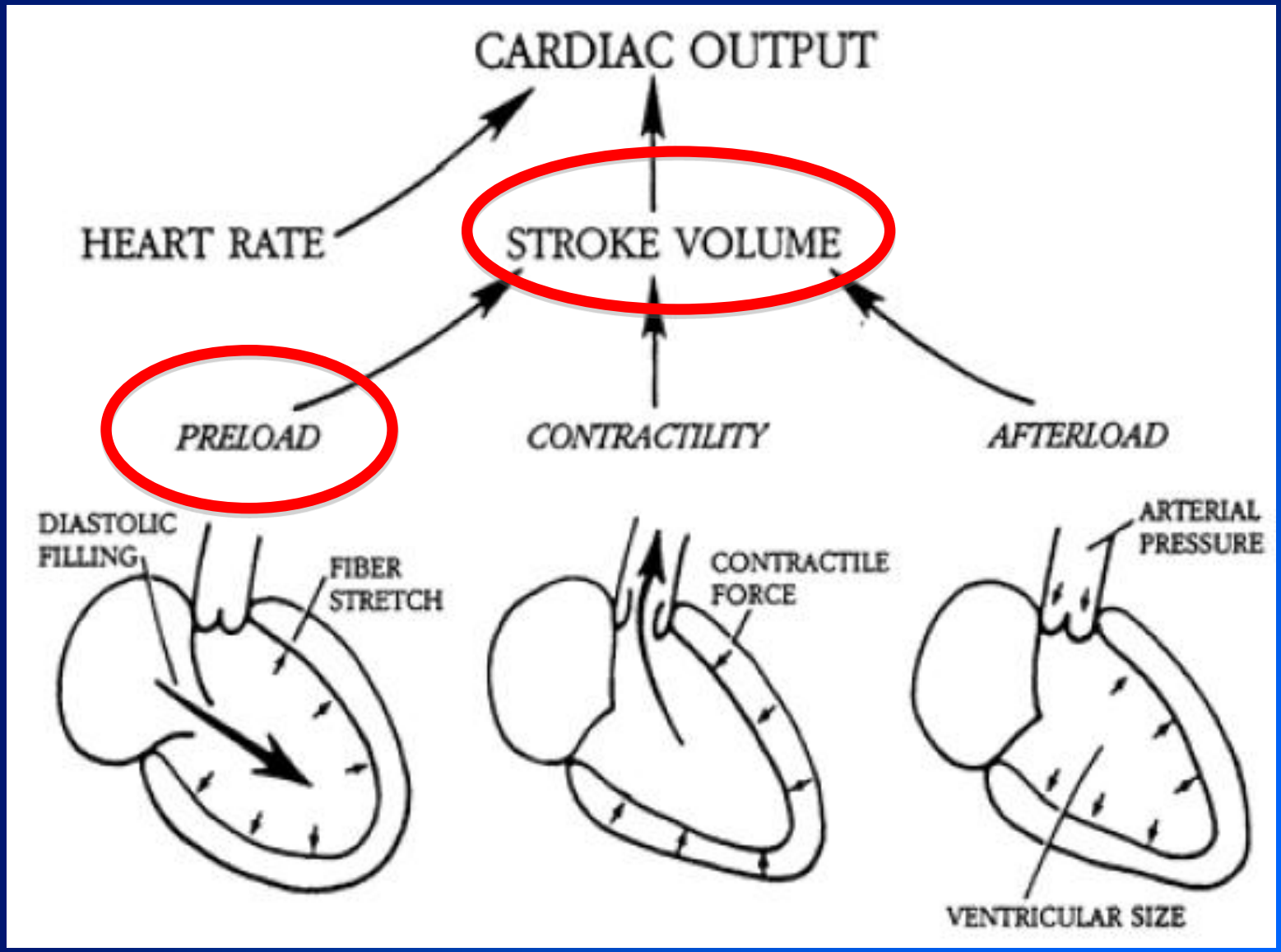
Conflicts of Interest

I declare

NO conflicts of interest



Determinants of Cardiac Output



What should we know for tx decisions ?

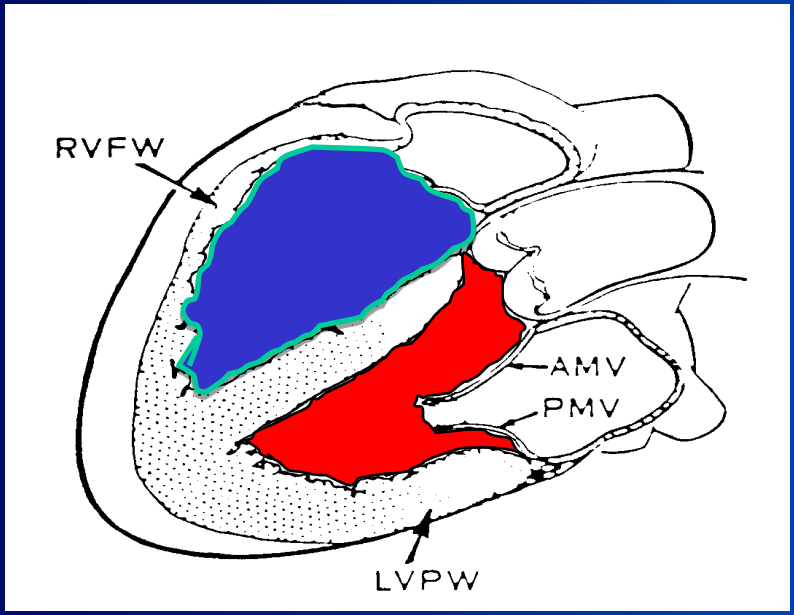
- Preload
- Flow
- Flow adequacy

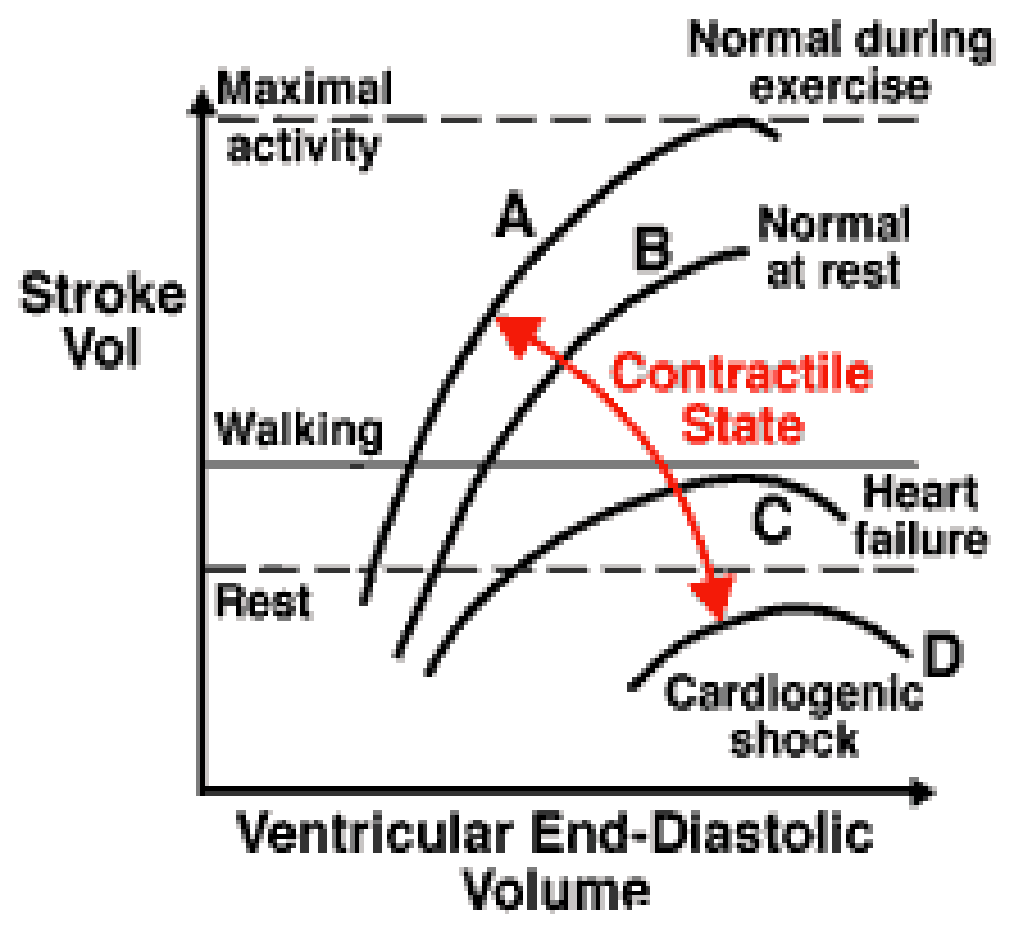
Fluids
Inotrops
Pressors dilators

Treatment !

Preload & Stroke Volume

PRELOAD = RVEDV/LVEDV





(Frank-Starling)

Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects

Anand Kumar, MD; Ramon Anel, MD; Eugene Bunnell, MD; Kalim Habet, MD, MD; Sergio Zanotti, MD; Stephanie Marshall, RN; Alex Neumann, MS; Amjad Ali, MD; Mary Cheang, MS; Clifford Kavinsky, MD, PhD; Joseph E. Parrillo, MD

Objective: Pulmonary artery occlusion pressure and central venous pressure have been considered to be reliable measures of left and right ventricular preload in patients requiring invasive hemodynamic monitoring. Studies in recent years have questioned the correlation between these estimates of ventricular filling pressures and ventricular end-diastolic volumes/cardiac performance variables in specific patient groups, but clinicians have continued to consider the relationship valid in the broader context. The objective of this study was to assess the relationship between pressure estimates of ventricular preload (pulmonary artery occlusion pressure, central venous pressure) and end-diastolic ventricular volumes/cardiac performance in healthy volunteers.

Design: Prospective, nonrandomized, nonblinded interventional study.

Setting: Cardiac catheterization and echocardiography laboratories.

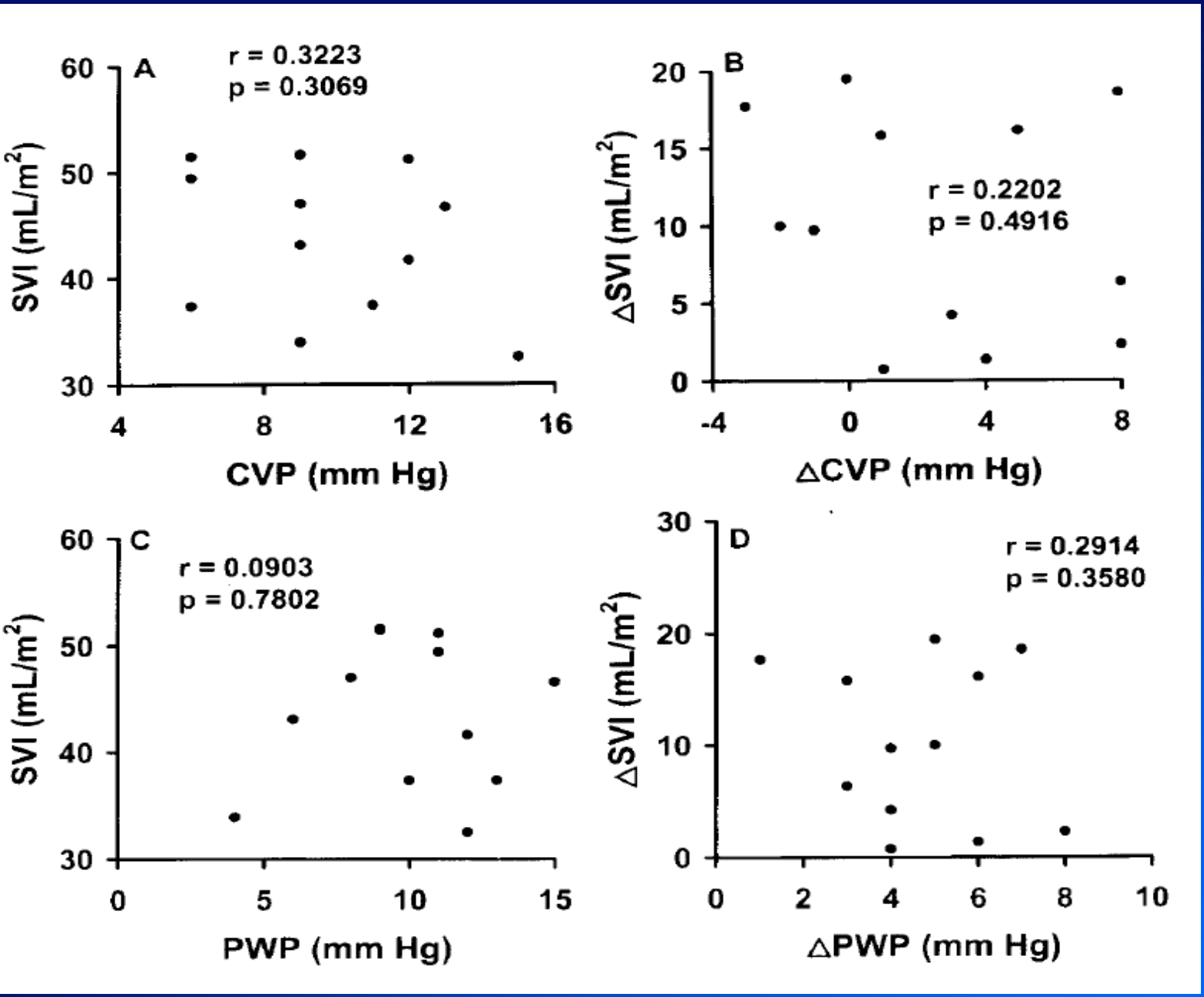
Subjects: Normal healthy volunteers (n = 12 group 1, n = 32 group 2).

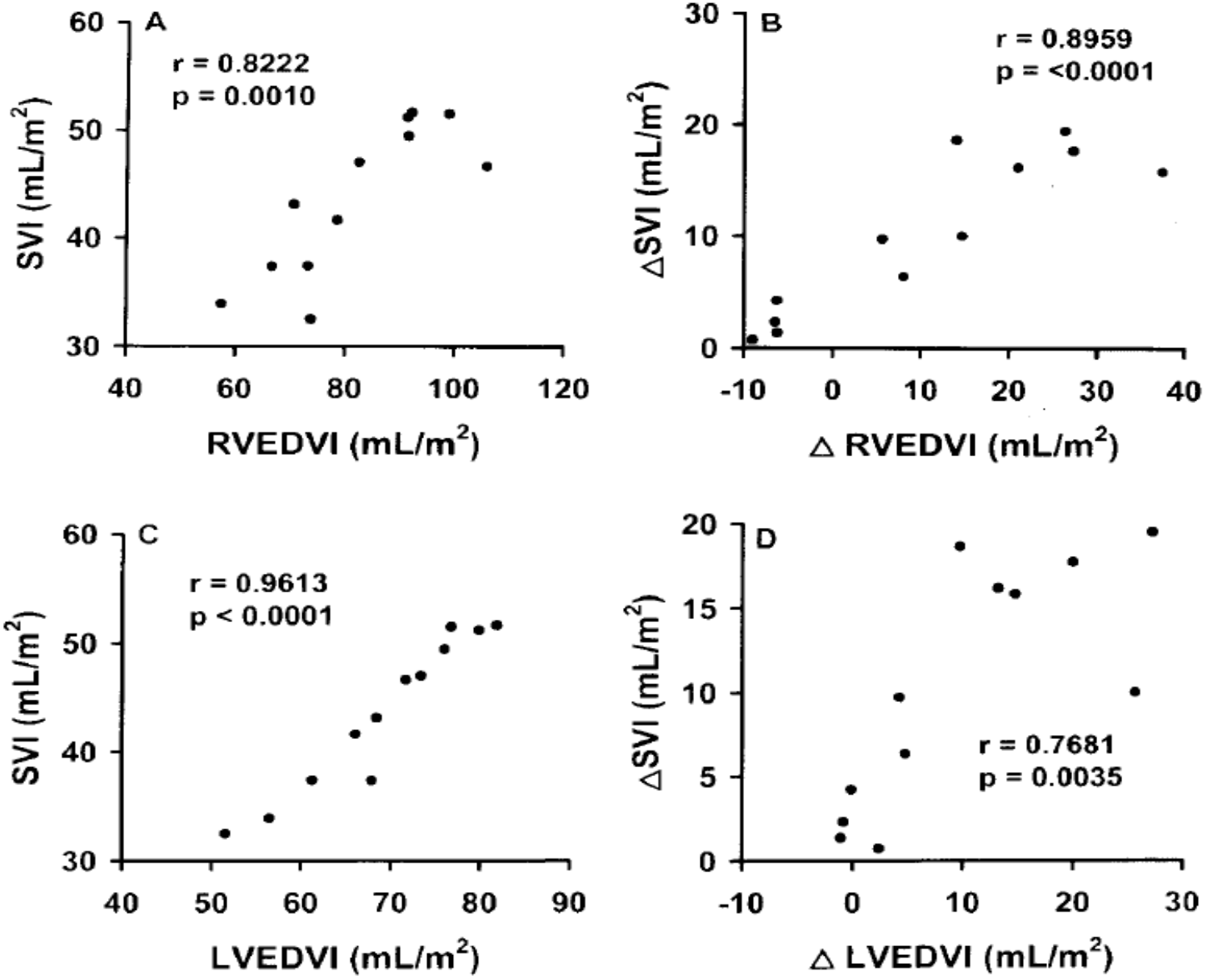
Interventions: Pulmonary catheterization and radionuclide cineangiography (group 1) and volumetric echocardiography (group 2) during 3 L of normal saline infusion over 3 hrs.

Measurements and Main Results: In group 1, the initial pulmonary artery occlusion pressure and central venous pressure did not correlate significantly with initial end-diastolic ventricular volume indexes or cardiac performance (cardiac index and stroke volume index). Changes in pulmonary artery occlusion pressure and central venous pressure following saline infusion also did not correlate with changes in end-diastolic ventricular volume indexes or cardiac performance. In contrast, initial end-diastolic ventricular volume indexes and changes in these ventricular volume indexes in response to 3 L of normal saline loading correlated well with initial stroke volume index and changes in stroke volume index, respectively. The relationship between left ventricular end-diastolic volume index and stroke volume index was confirmed in group 2 subjects using mathematically independent techniques to measure these variables. In addition, initial central venous pressure, right ventricular end-diastolic volume index, pulmonary artery occlusion pressure, and left ventricular end-diastolic volume index failed to correlate significantly with changes in cardiac performance in response to saline infusion in group 1 subjects.

Conclusions: Normal healthy volunteers demonstrate a lack of correlation between initial central venous pressure/pulmonary artery occlusion pressure and both end-diastolic ventricular volume indexes and stroke volume index. Similar results are found with respect to changes in these variables following volume infusion. In contrast, initial end-diastolic ventricular volume indexes and changes in end-diastolic ventricular volume indexes in response to saline loading correlate strongly with initial and postsaline loading changes in cardiac performance as measured by stroke volume index. These data suggest that the lack of correlation of these variables in specific patient groups described in other studies represents a more universal phenomenon that includes normal subjects. Neither central venous pressure nor pulmonary artery occlusion pressure appears to be a useful predictor of ventricular preload with respect to optimizing cardiac performance. (Crit Care Med 2004; 32:691–699)

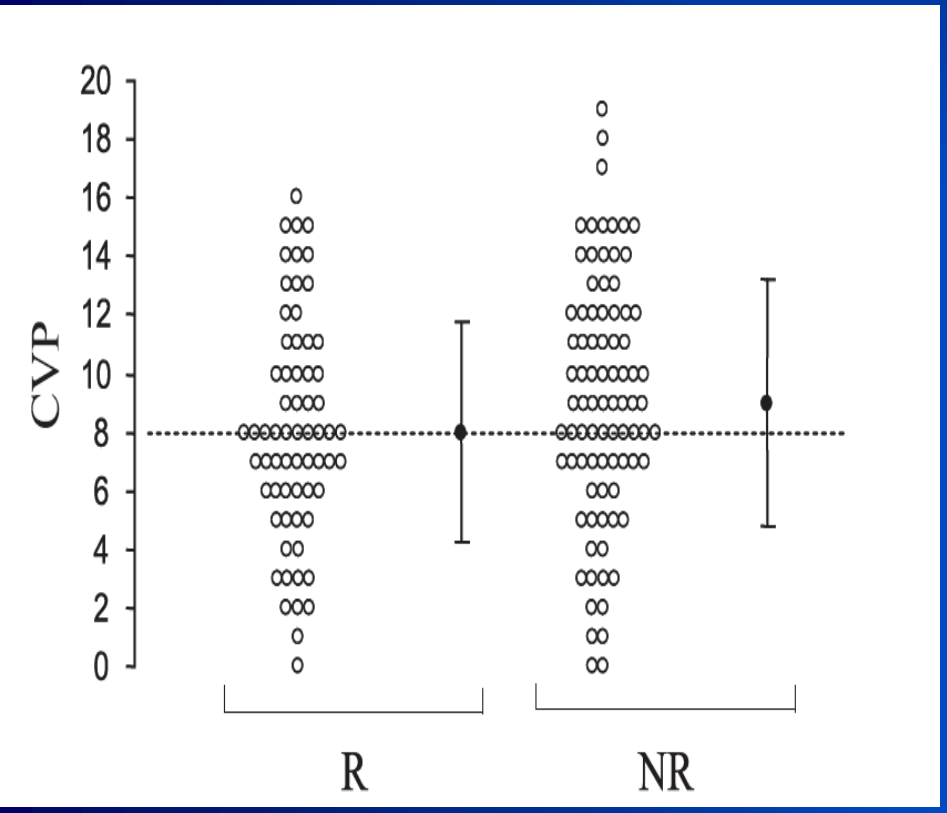
KEY WORDS: volunteers; saline; heart; cardiac output; stroke volume; ventricular volume; cardiac compliance; pulmonary artery occlusion pressure; central venous pressure; preload

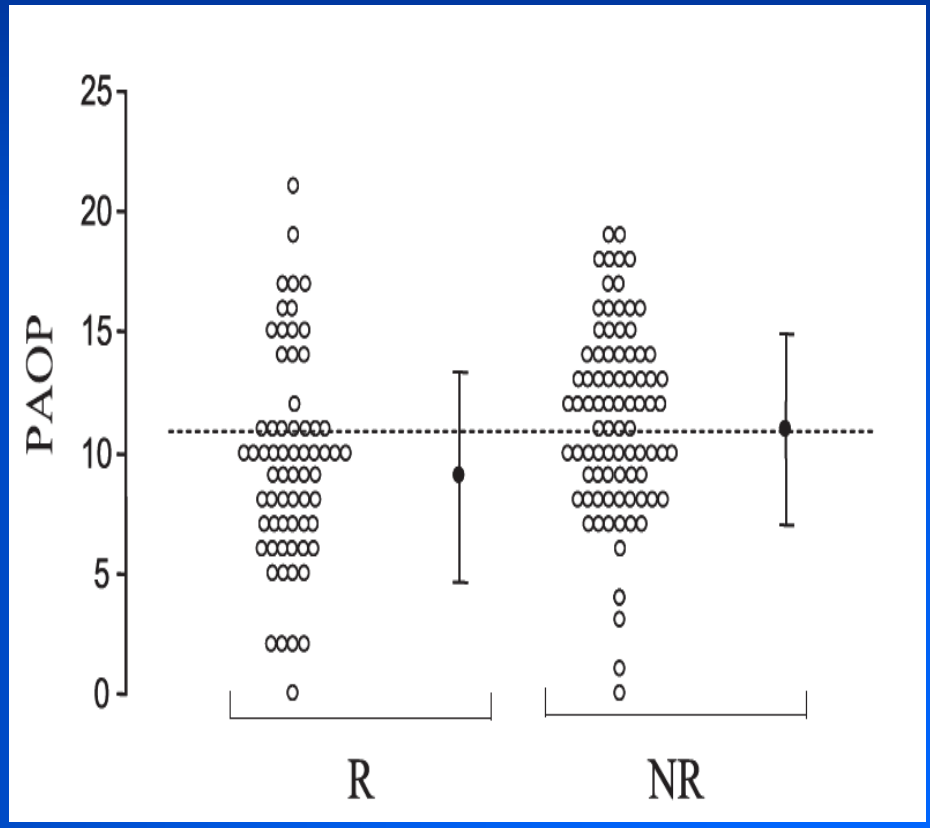

A
r = 0.3223
p = 0.3069
SVI (mL/m²)
60
50
40
30
4
8
12
16
CVP (mm Hg)
B
r = 0.2202
p = 0.4916
ΔSVI (mL/m²)
20
15
10
5
0
-4
0
4
8
ΔCVP (mm Hg)
C
r = 0.0903
p = 0.7802
SVI (mL/m²)
60
50
40
30
0
5
10
15
PWP (mm Hg)
D
r = 0.2914
p = 0.3580
ΔSVI (mL/m²)
30
20
10
0
0
2
4
6
8
10
ΔPWP (mm Hg)


A
r = 0.8222
p = 0.0010
SVI (mL/m²)
RVEDVI (mL/m²)
B
r = 0.8959
p = <0.0001
ΔSVI (mL/m²)
Δ RVEDVI (mL/m²)
C
r = 0.9613
p < 0.0001
SVI (mL/m²)
LVEDVI (mL/m²)
D
r = 0.7681
p = 0.0035
ΔSVI (mL/m²)
Δ LVEDVI (mL/m²)

Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge*

David Osman, MD; Christophe Ridel, MD; Patrick Ray, MD; Xavier Monnet, MD, PhD; Nadia Anguel, MD; Christian Richard, MD; Jean-Louis Teboul, MD, PhD





RAP/CVP IS A FUNCTION OF FOUR INDIPENDENT PARAMETERS

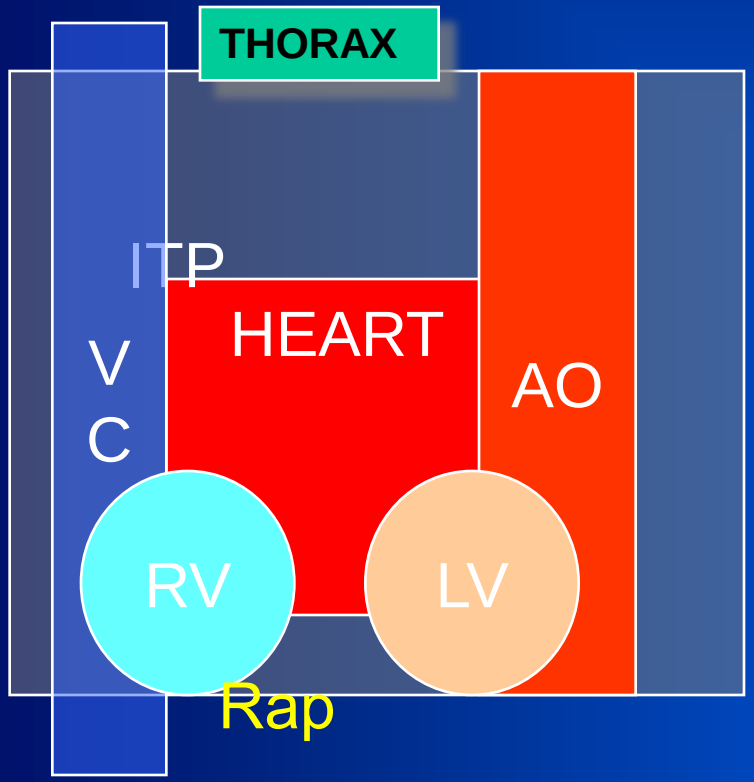
- Blood volume and flow in the central veins
- Right ventricular compliance and contractility during diastolic filling
- Central veins tonus
- Ventricular interactions
- Intrathoracic pressure

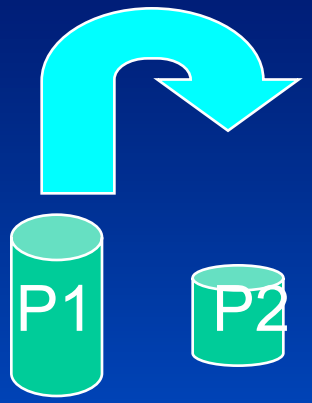
…….RAP/CVP IS A FUNCTION OF MANY OTHER FACTORS

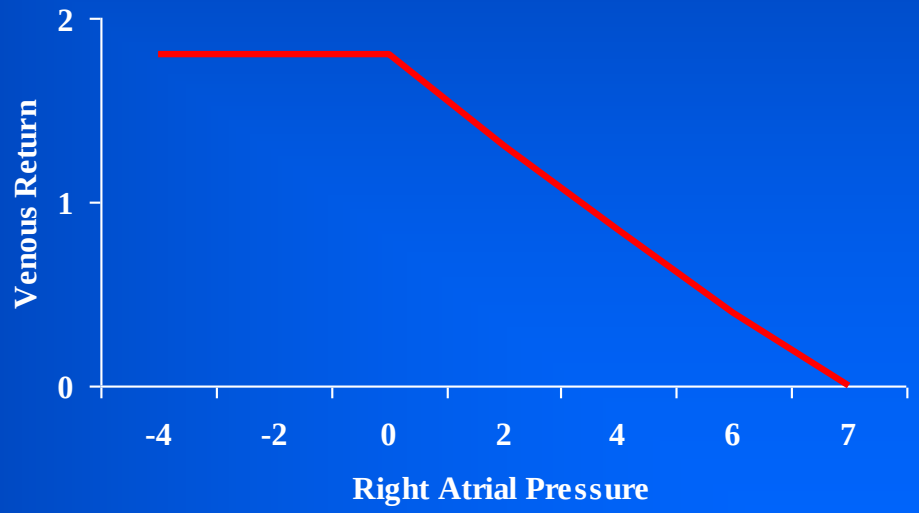
- Patient's posture
- Venus obstruction
- Valvular alterations (stenosis/tricuspidal insufficiency) and ventricular compliance
- Cardiac rythm and respiratory rate

VENOUS RETURN CURVE

$$VR = \frac{PmS - RAP}{r}$$







PAWP

- PAWP → LAP

```
ZONE_WEST_
1 PA>Pa>Pv_
2 Pa>PA>Pv_
3 Pa>Pv>PA_
```

  - shape(eccentric) of the balloon
  - no zone III West
  - venous pulmonary occlusion

- LAP → LVEDP
  - stenosis or insufficiency of the mitral valve
  - tachicardia and premature occlusion of mitral valve
  - atrial contraction during hypovolemia

- LVEDP →LVEDV

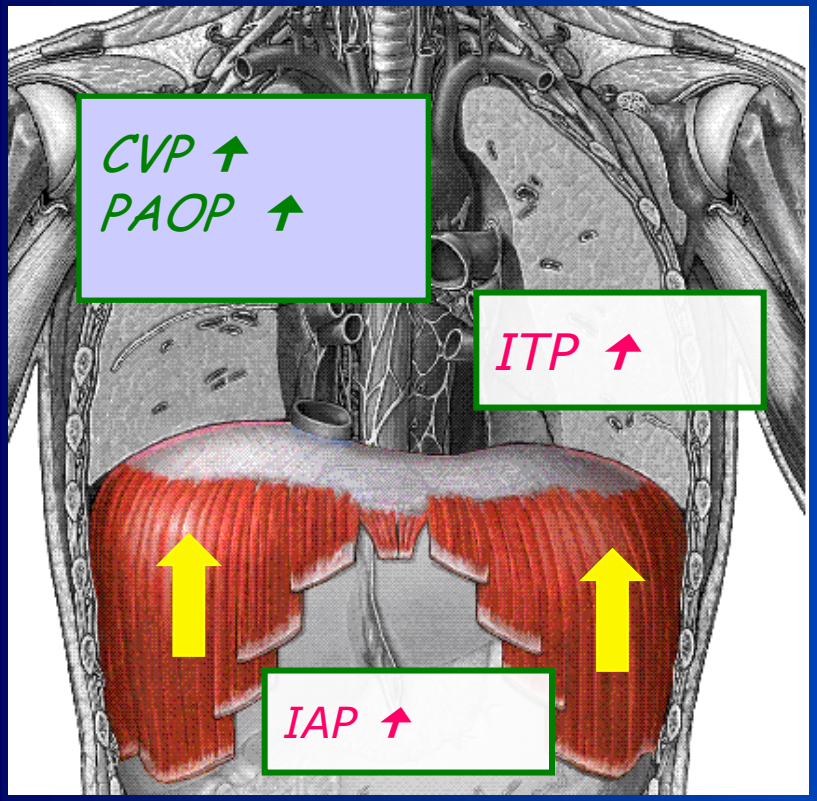
  Compliance variations due to:

  - *Stiffness of LV*
  - *↑ Pressure around pericardium (effusion, ↑ITP)*
  - *Ventricular interdependence*

Paolo Pelosi
Enrico Calzia
Pierre Asfar

It's time to measure intra-abdominal pressure to optimize hemodynamics!

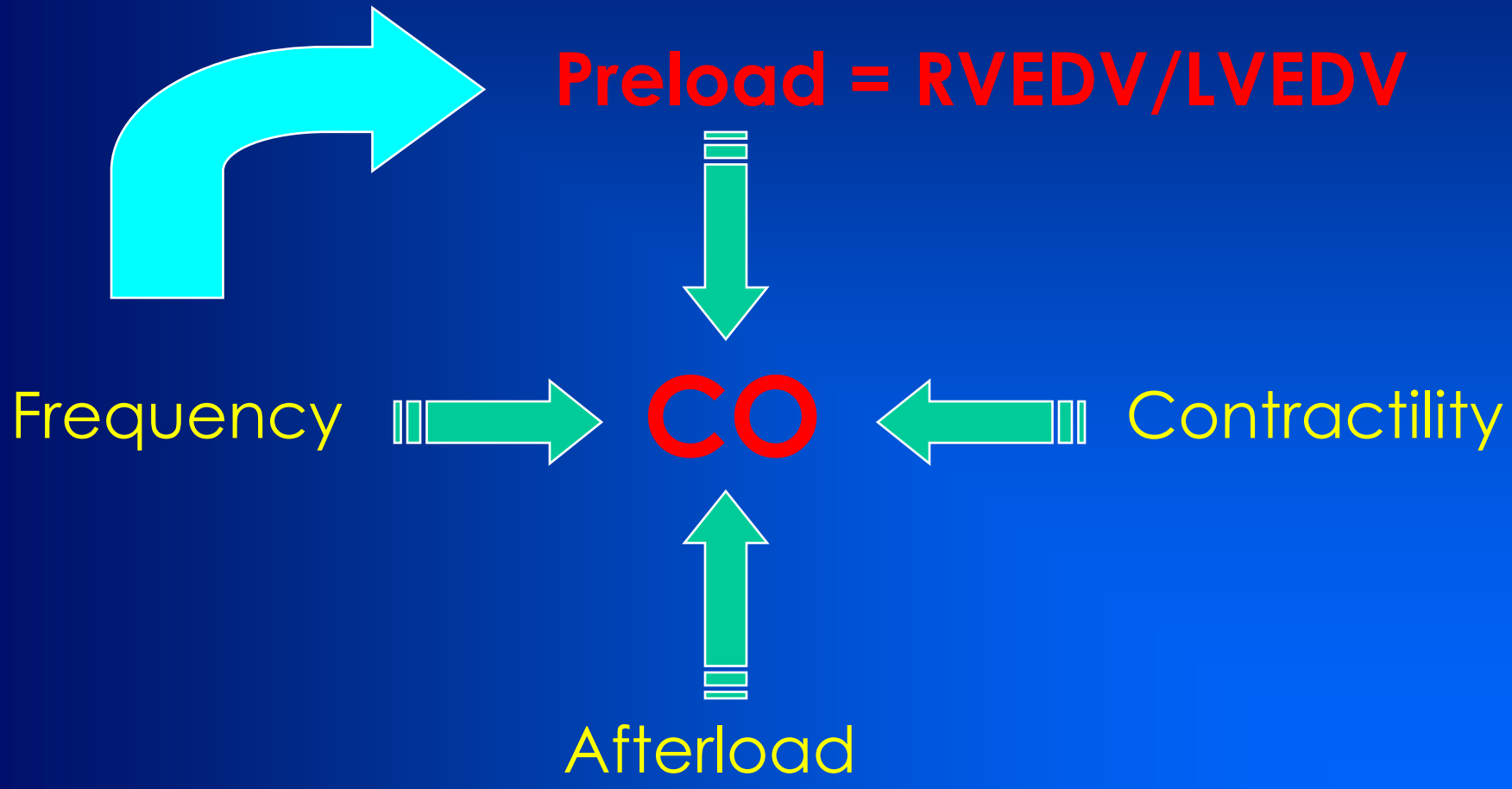
Intensive Care Med 2007; 33(1): 6-8



- **Use transmural filling pressures !**

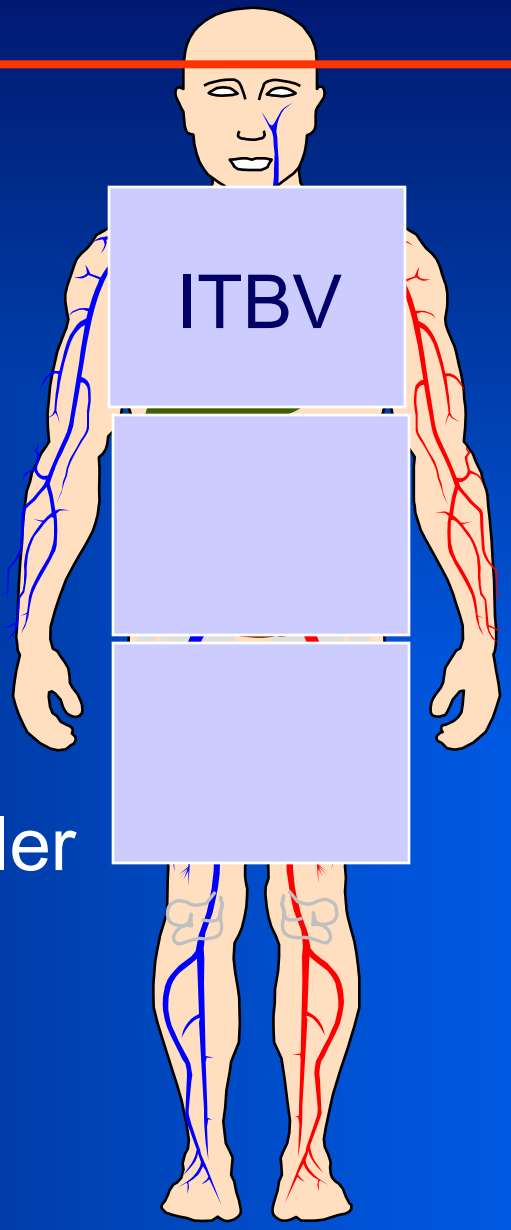
- **CVP^{TM} = CVP - IAP/2**
- **$PAOP^{TM}$ = PAOP - IAP/2**

CO determinants



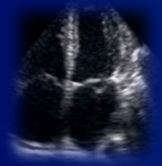
Intra Thoracic Blood Volume: preload?

$$ITBV = \frac{TBV}{3}$$

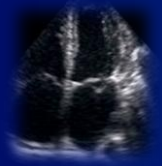


- ✓ ECHO
- ✓ Esophageal Doppler
- ✓ LiDCO
- ✓ Picco
- ✓ Vigileo

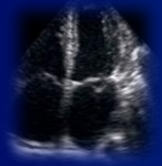
Rapid Assessment by Cardiac Echo (RACE)



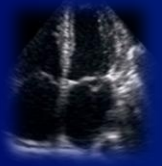
Left ventricular heart function



Right ventricular heart function

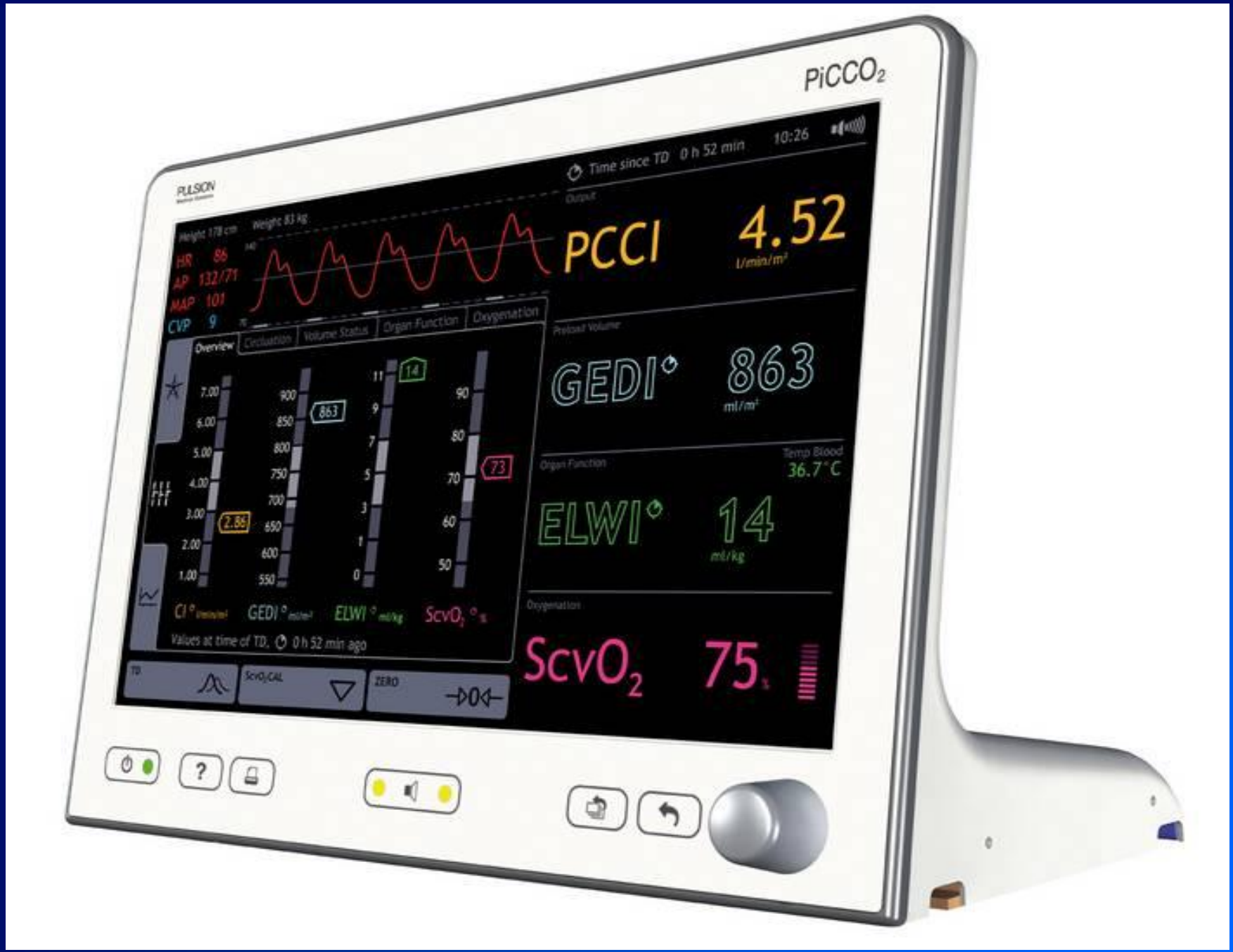


Pericardial effusion

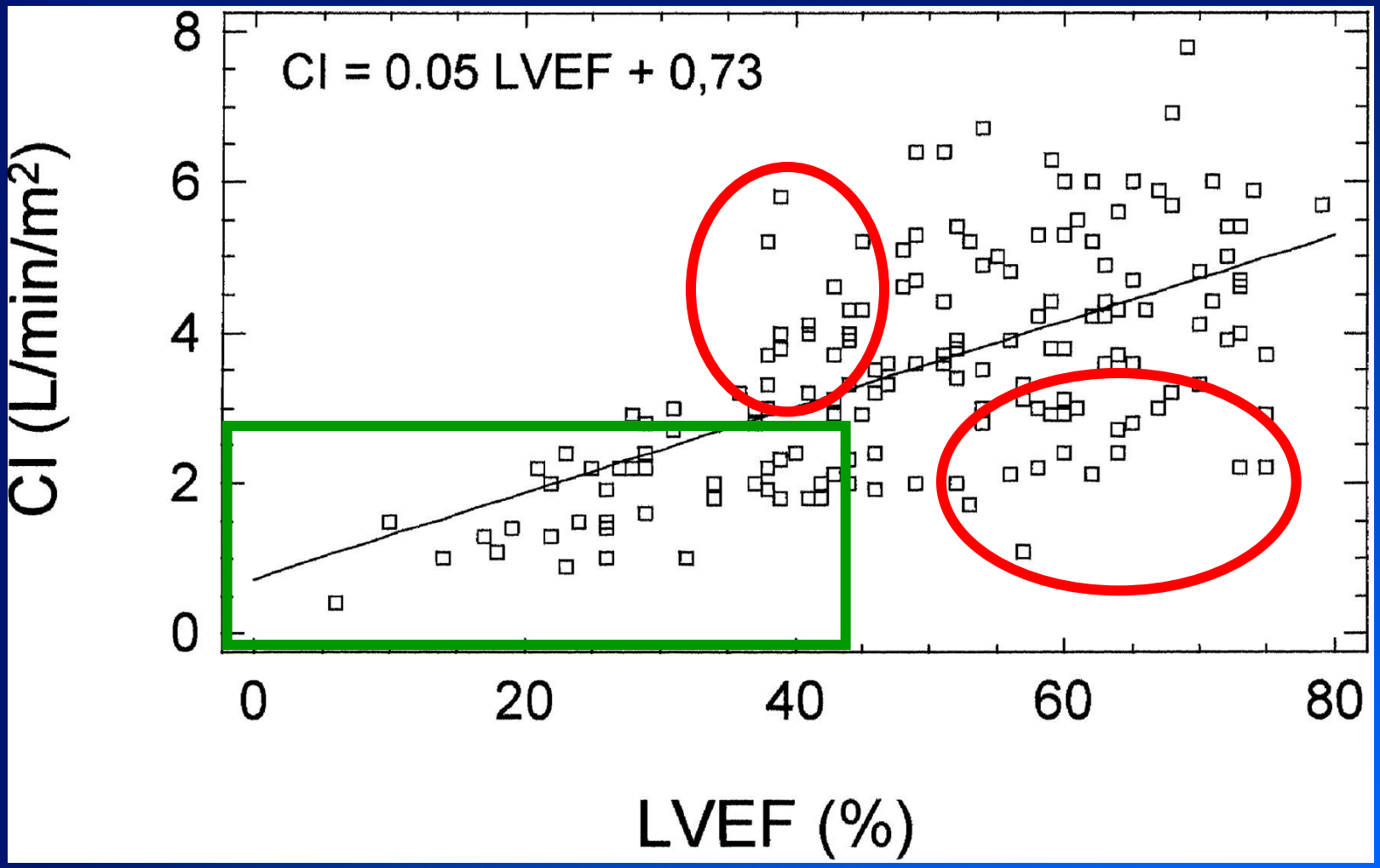


Fluid status

By the Nepean Institute of Critical Care Education and Research

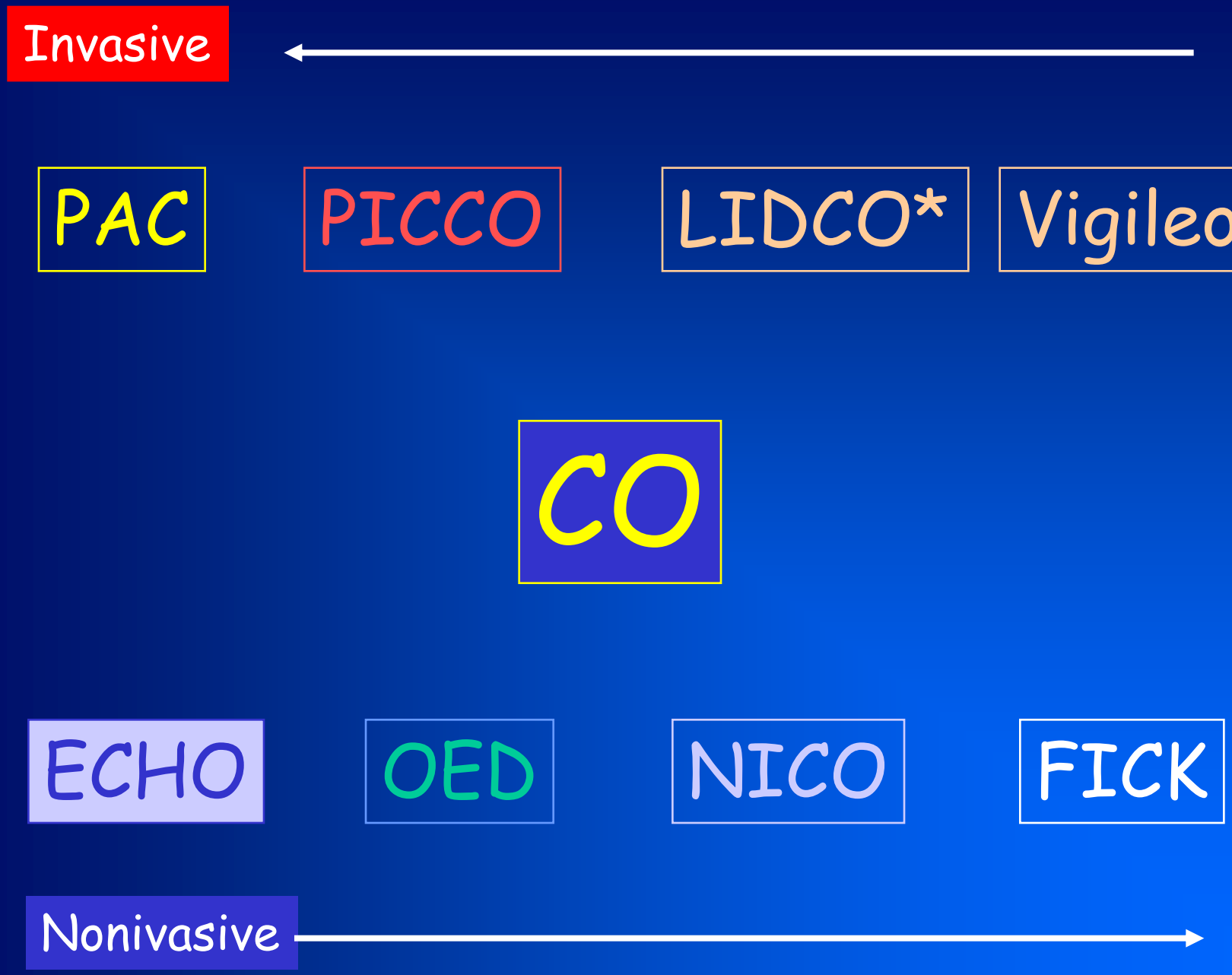

PiCCO2
PULSION
Time since TD 0 h 52 min
10:26
Output
PCCI
4.52
l/min/m²
HR 86
AP 132/71
MAP 101
CVP 9
Overview
Circulation
Volume Status
Organ Function
Oxygenation
Preload Volume
GEDI
863
ml/m²
Organ Function
Temp Blood
36.7 °C
ELWI
14
ml/kg
Oxygenation
ScvO2
75
CI
GEDI
ELWI
ScvO2
Values at time of TD. 0 h 52 min ago
TD
ScvO2CAL
ZERO

Vieillard-Baron et al
AJRCCM 168:1270;2003

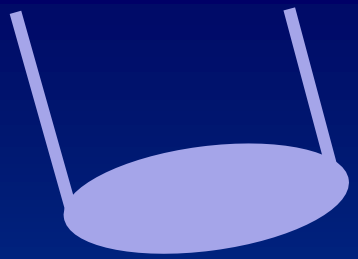


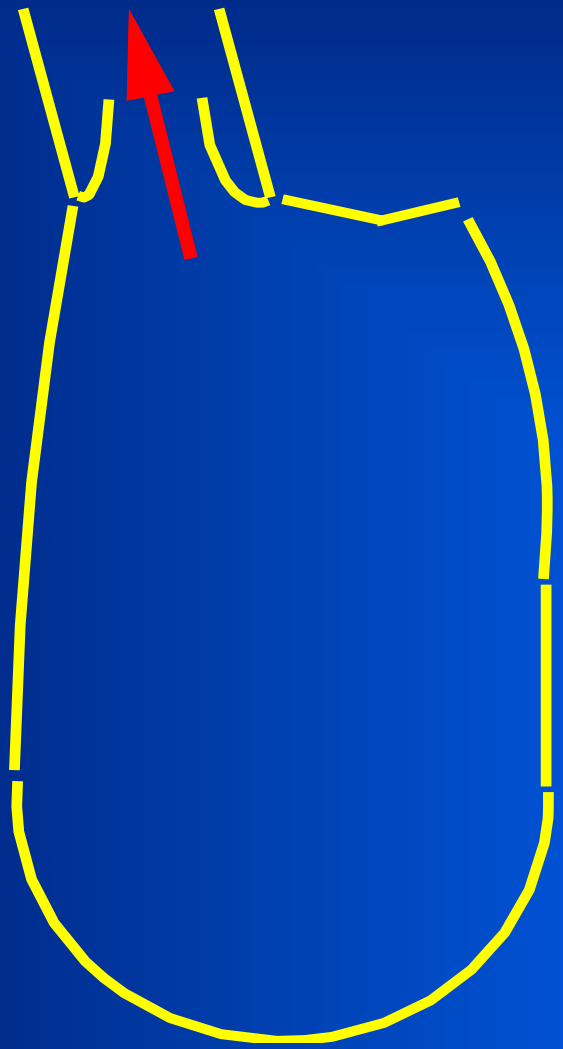
⇒CO can be preserved even when EF is markedly altered

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


Invasive
PAC
PICCO
LIDCO*
Vigileo
CO
ECHO
OED
NICO
FICK
Nonivasive

Measurement of cardiac outp



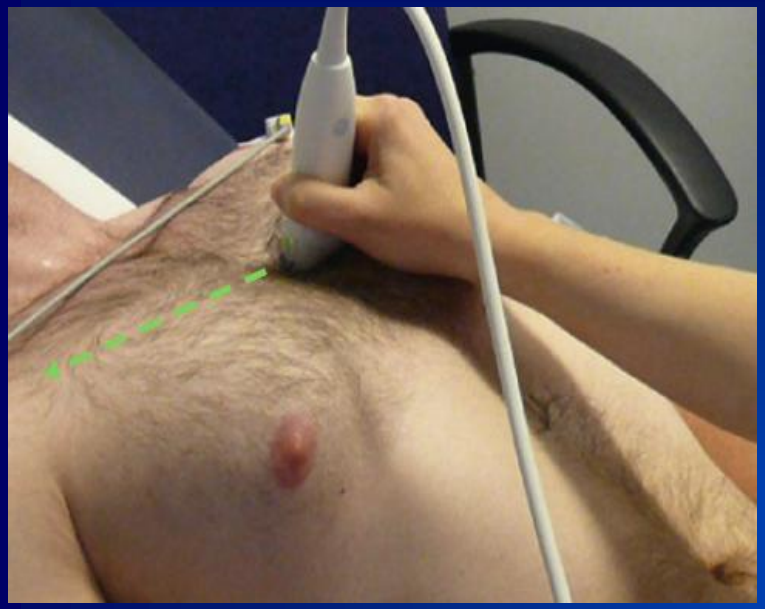


SV = VTI x LVOTarea

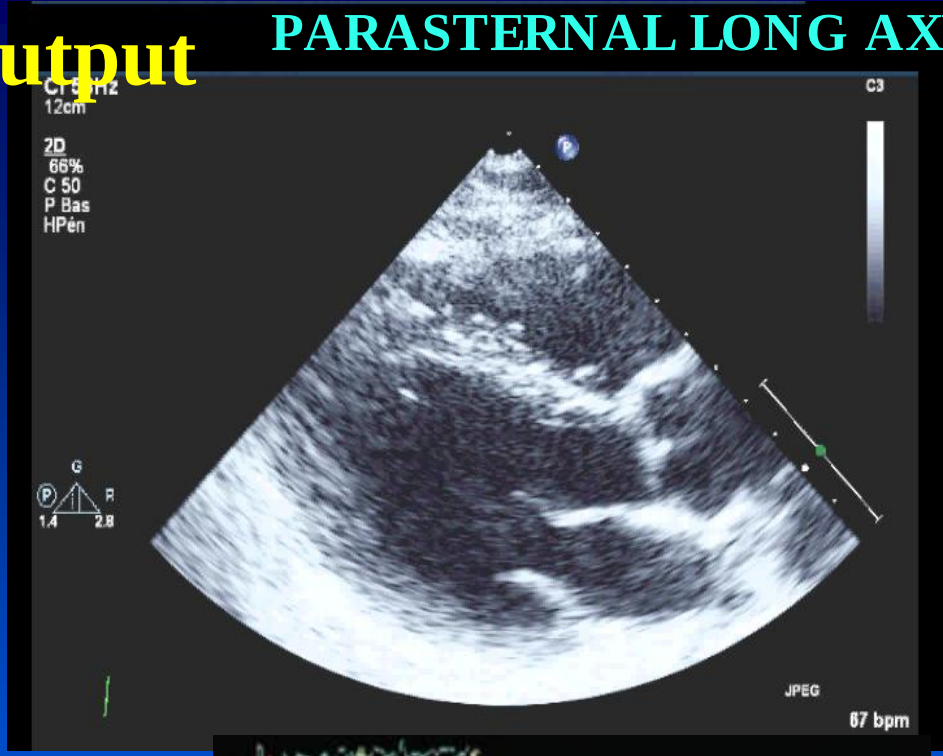
LVOT=
left ventricular outflow tract

VTI= velocity time integral

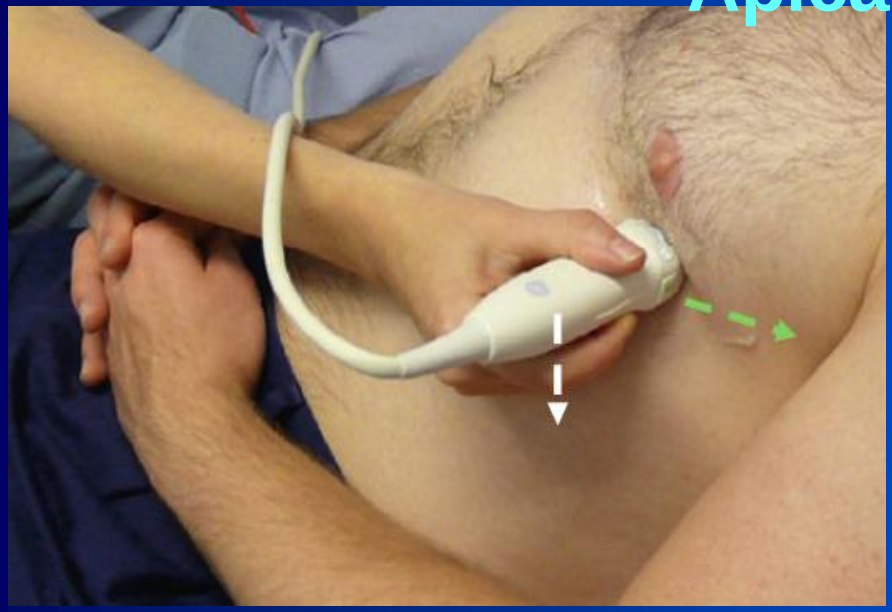
Measurement of cardiac output

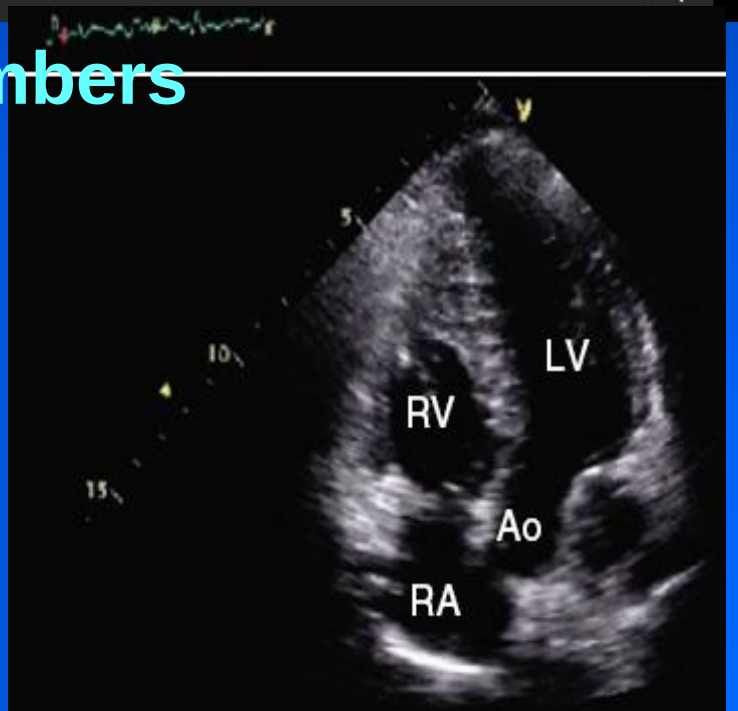


PARASTERNAL LONG AX

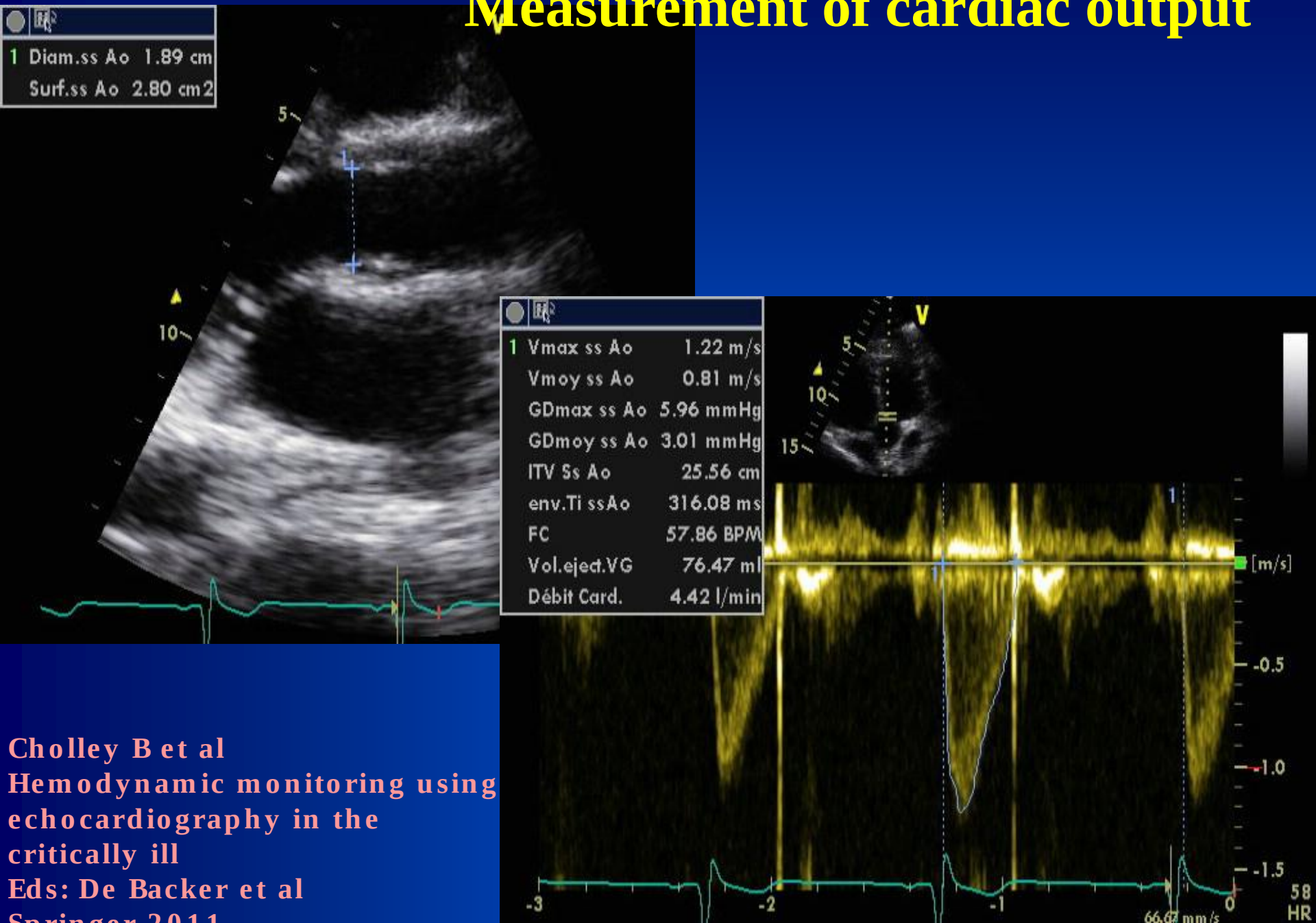


Apical 5 chambers



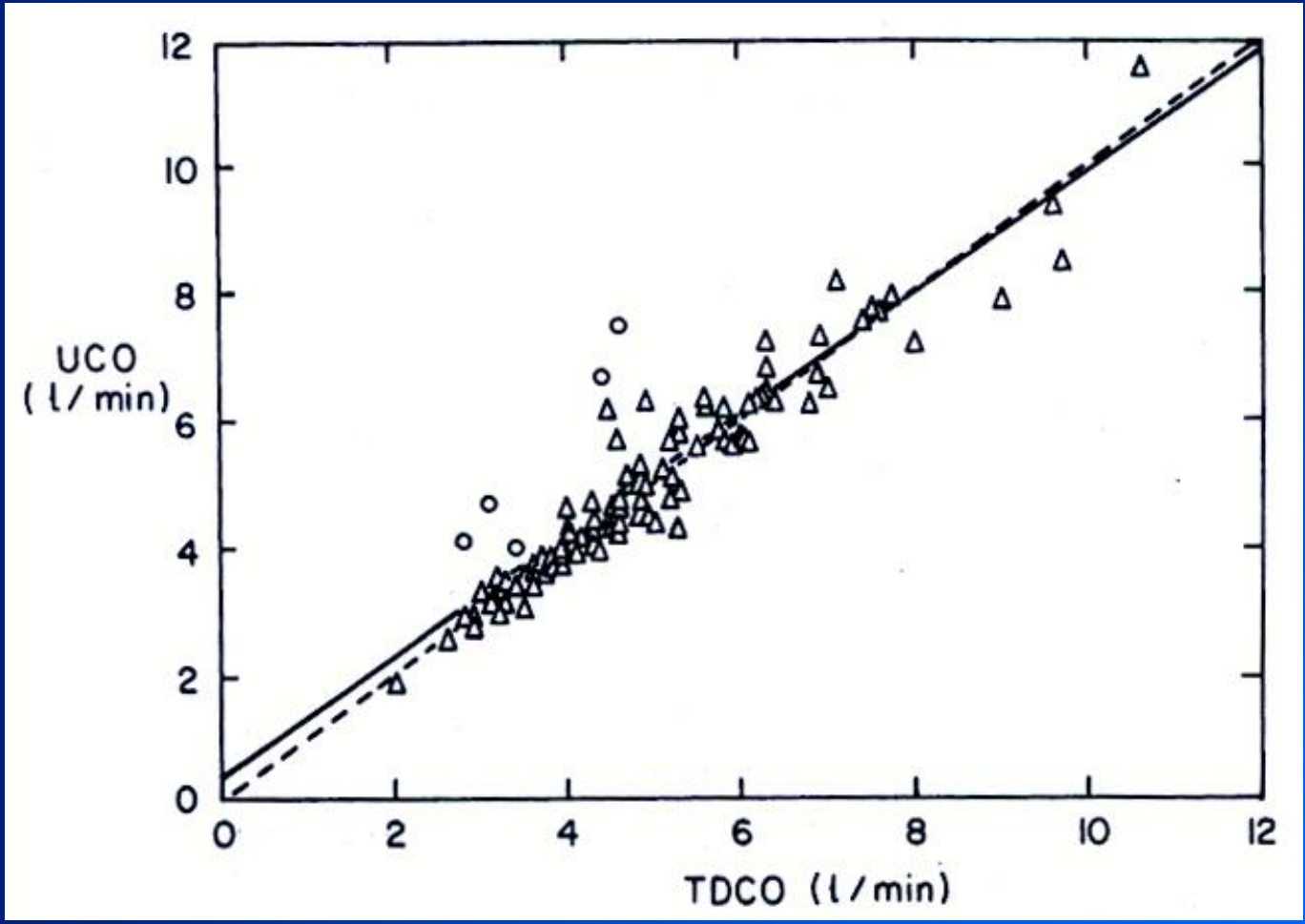


Measurement of cardiac output



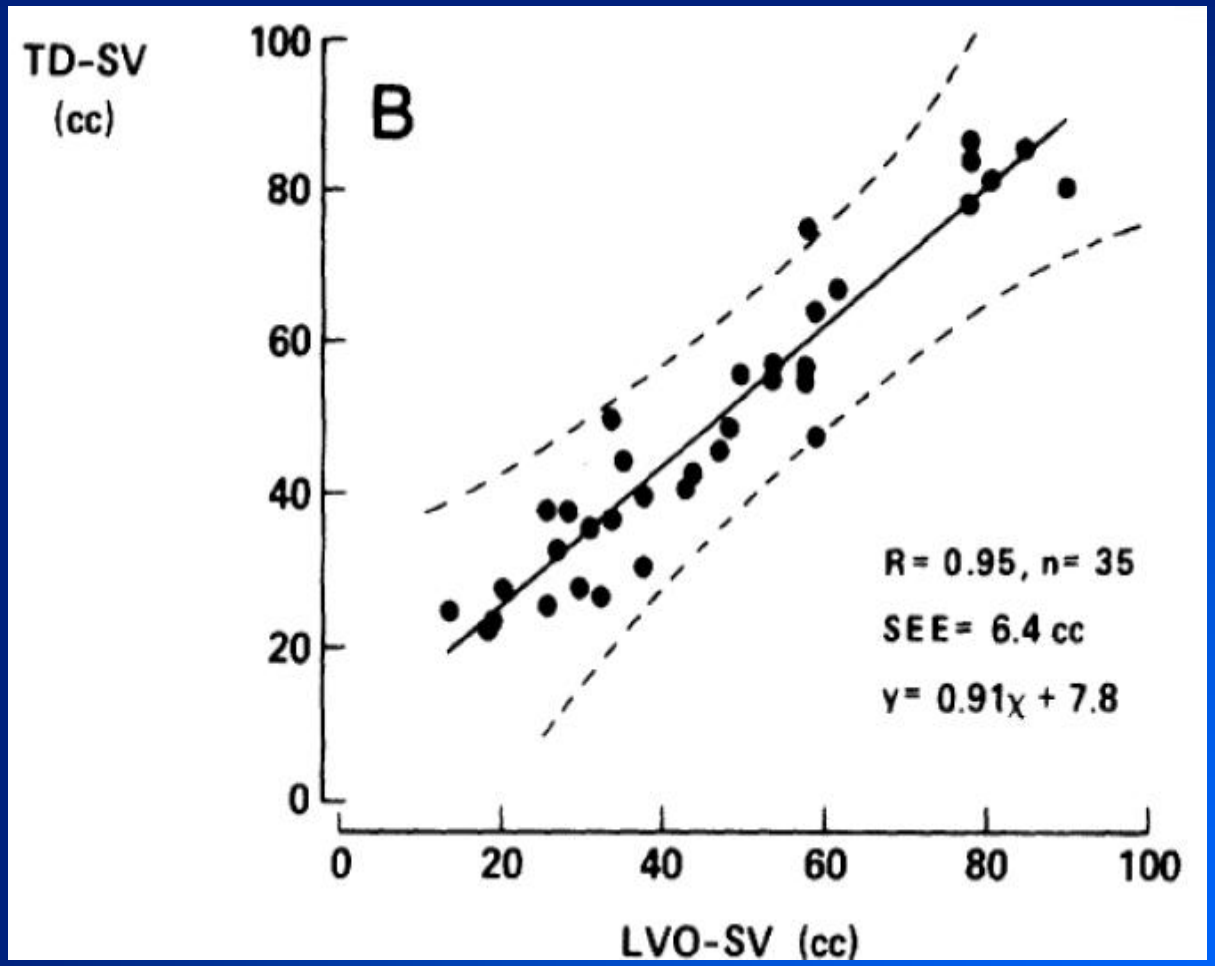
Cholley B et al
Hemodynamic monitoring using echocardiography in the critically ill
Eds: De Backer et al
Springer 2011

Huntsman et al.
Circulation 1983



Echo vs Thermodilution CO measurements

Lewis JA et al.
Circulation 1984



Thermodilution vs echo (stroke volume)

Lewis JA et al.
Circulation 1984

Inter-observer variability

Cross-sectional area	6.0 ± 1.6%
Velocity-time integral	2.4 ± 1.5%
Stroke volume	6.8 ± 5.0%

N = 39

CO with echocardiography

The pros:

- Reliable
- Additional measurements (pressure/volumes)
- Full cardiac evaluation
- Measurement of SVV

The cons:

- Skills required
- Time consuming
- Intermittent

CONTINUOUS PRESSURE-BASED CARDIAC OUTPUT MONITORING SYSTEMS

1) CALIBRATED

PiCCO2®

EV-1000/VolumeView™

LiDCOplus™

COstatus ®

Geisen M, et al. Curr Opin Crit Care **2012**, 18:377–384

CONTINUOUS PRESSURE-BASED CARDIAC OUTPUT MONITORING SYSTEMS

2) UNCALIBRATED

(pre-estimated data - nomograms)

Vigileo™

LiDCOrapid™

PulsioFlex ®

Geisen M, et al. Curr Opin Crit Care **2012**, 18:377–384

"other" CARDIAC OUTPUT MONITORING SYSTEMS

NICOM®

ECOM®

Nexfin®

NICO®

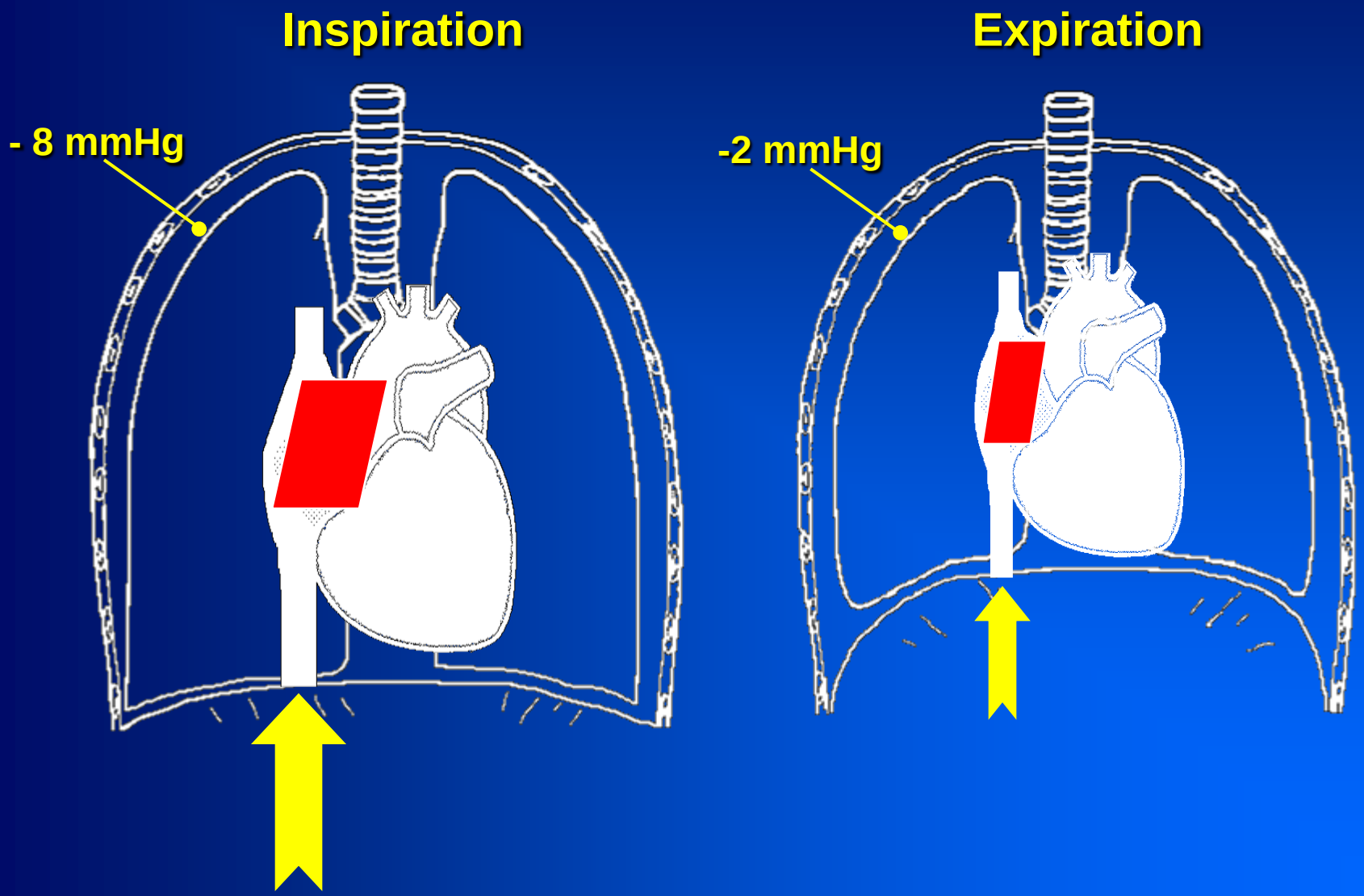
Geisen M, et al. Curr Opin Crit Care **2012**, 18:377–384

What technique should I use to measure cardiac output?
Christoph K. Hofer[a], Michael T. Ganter[b] and Andreas Zollinger[a]

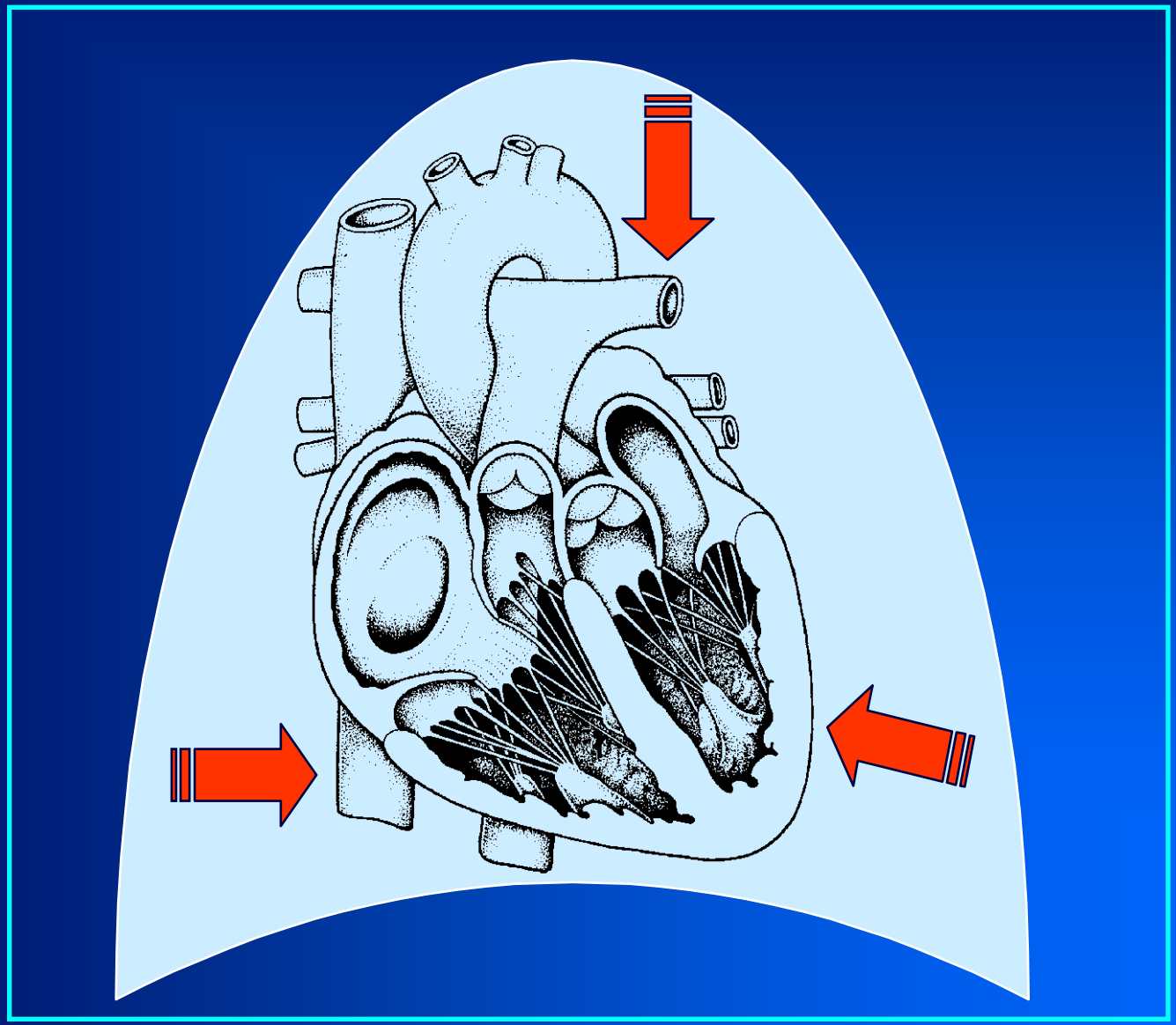
Current Opinion in Critical Care

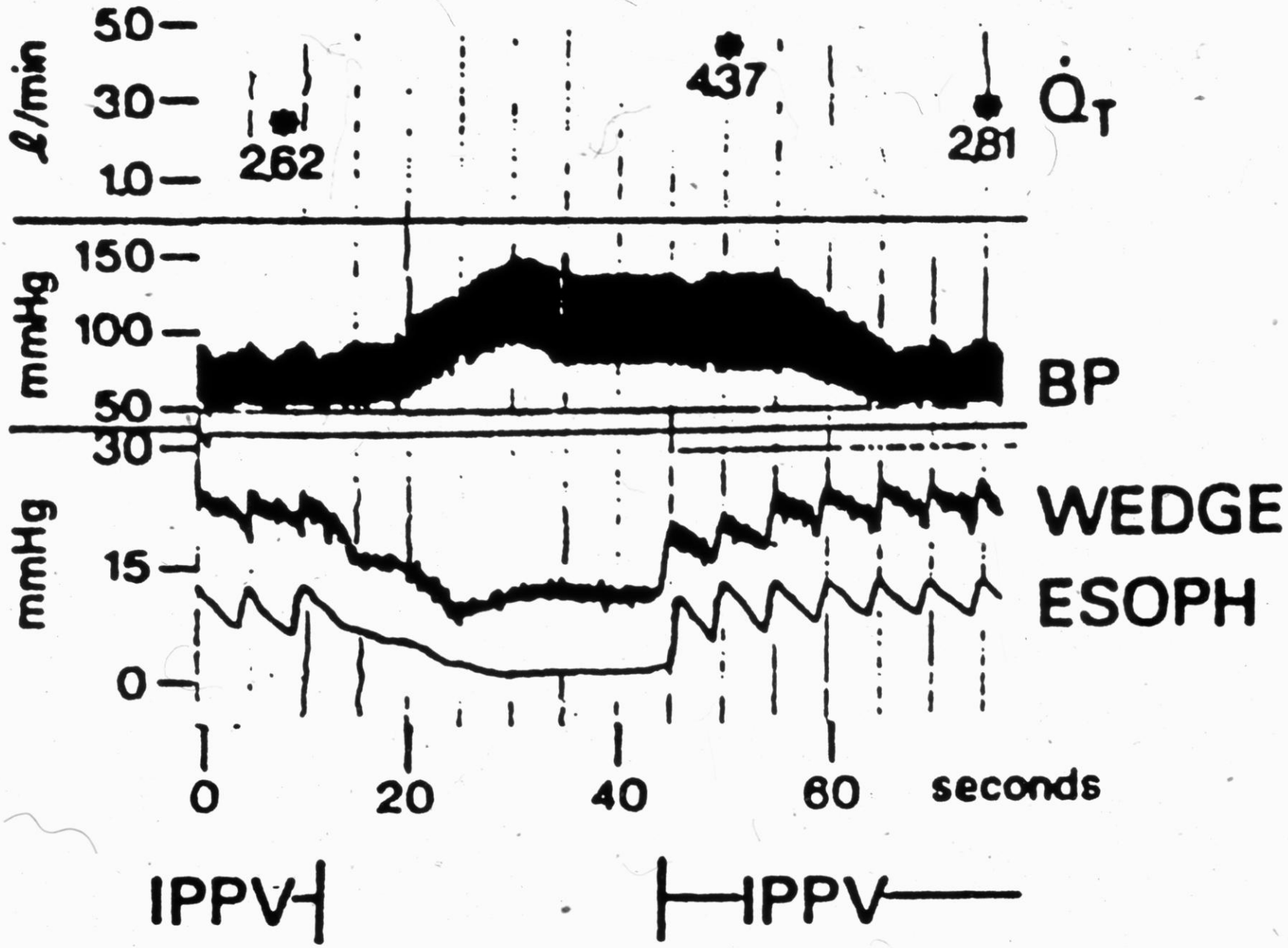
	Intermittent	Continuous	Invasive	Limitations	Additional information
PAC	+	+ (5 -12 min)	+++	Well described complications	PAP, PCWP, SVO2
PiCCO	+	+ (3 sec)	+(+)	Specific arterial catheter	GEDV, EVLW, SVV
LiDCO	+	+	+	Lithium injection	SVV
Flotrac		+ (20 sec)	(+)		SVV

SPONTANEOUS VENTILATION

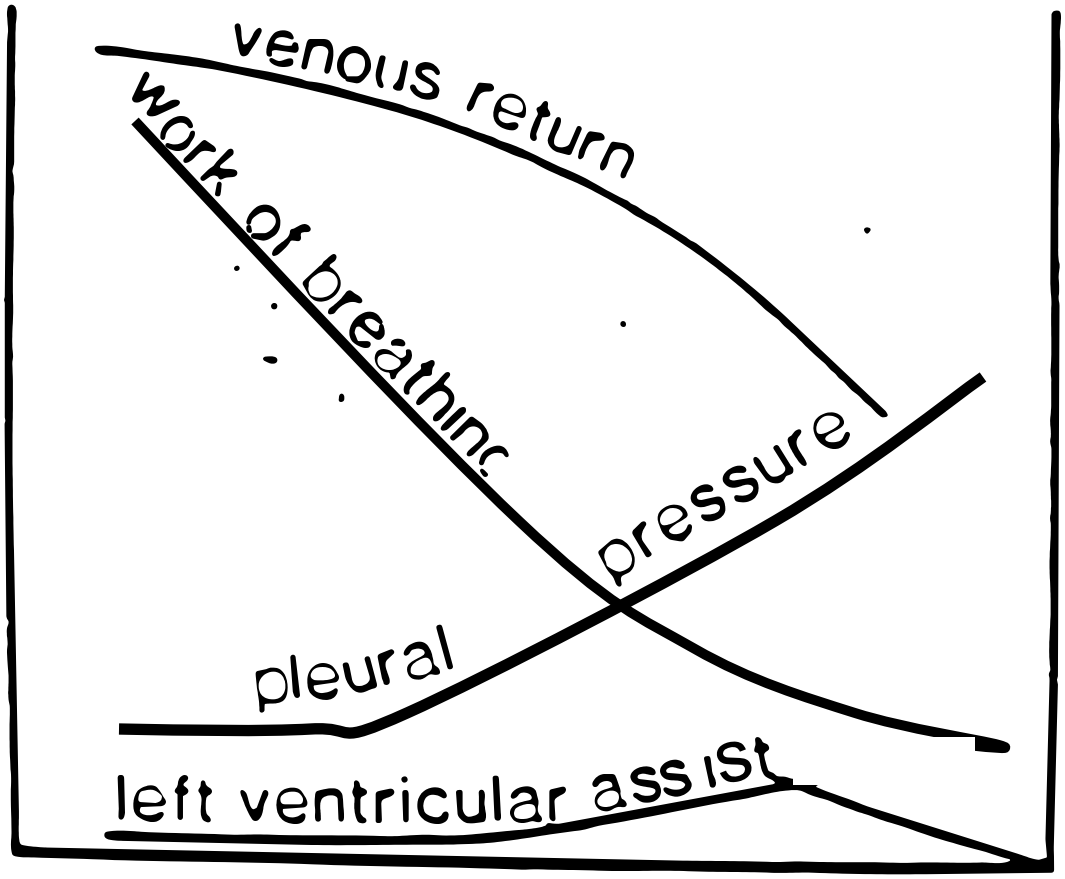


INTRATHORACIC PRESSURE



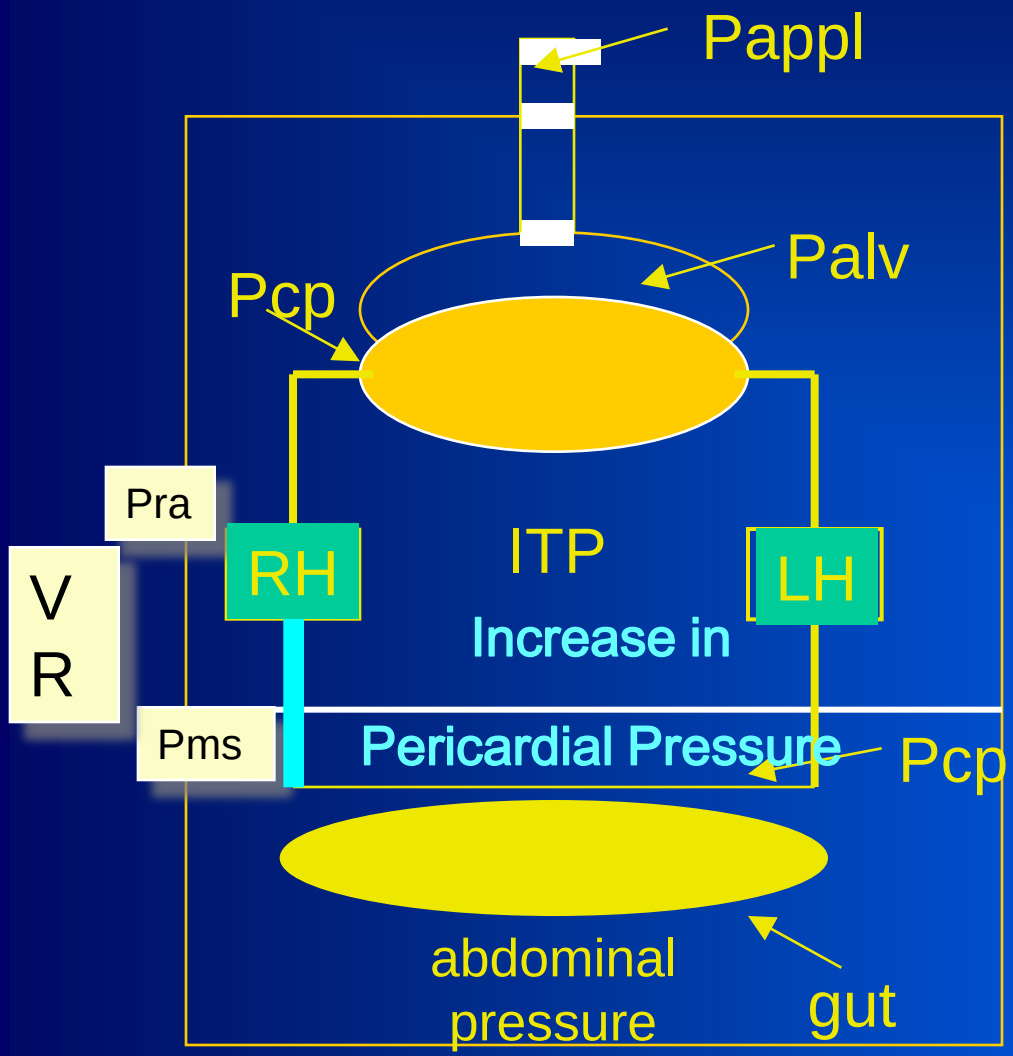

l/min
50
30
10
262
437
281
$\dot{Q}_T$
mmHg
150
100
50
BP
30
mmHg
15
0
WEDGE
ESOPH
0
20
40
60
seconds
IPPV
IPPV

Effects of Different Ventilatory Modes on Cardiopulmonary Performance



Mofied from Synder 1984

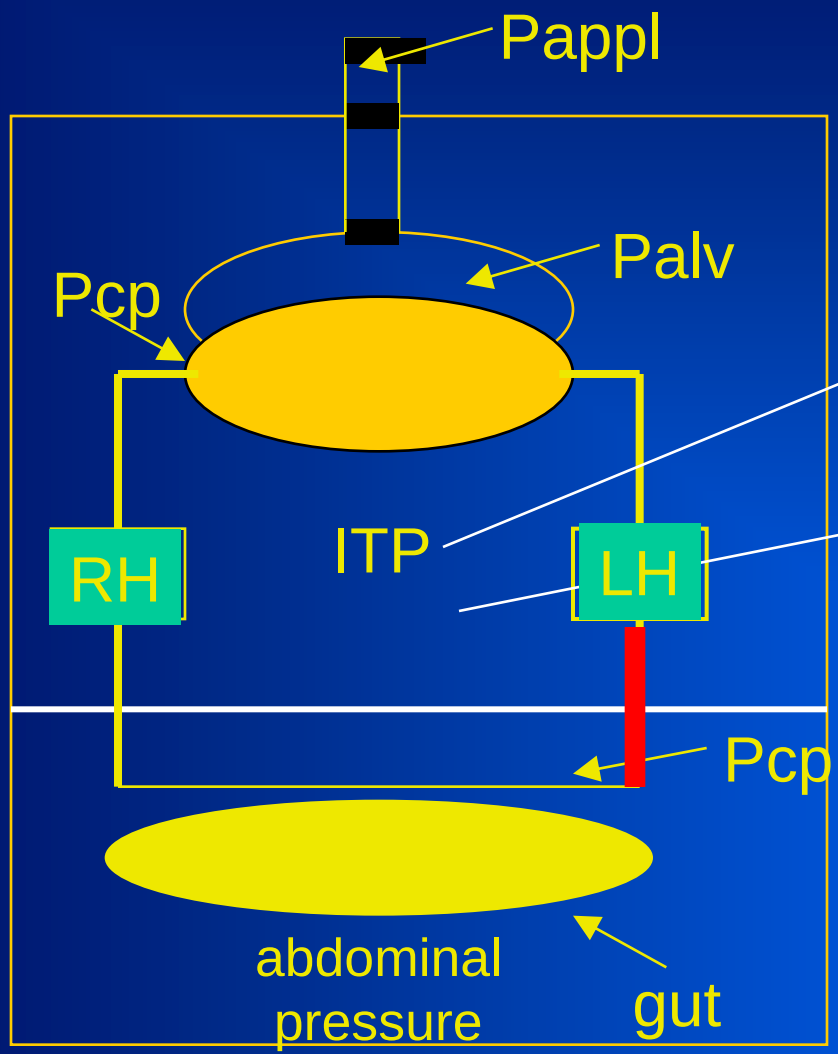
Effects of the increase in **ITP** : ***RV pre-load***



- Changes in ITP will not alter the (RV afterload) pressure gradient
- Decrease in VR are due to increase in Pra because of increased in pericardial pressure

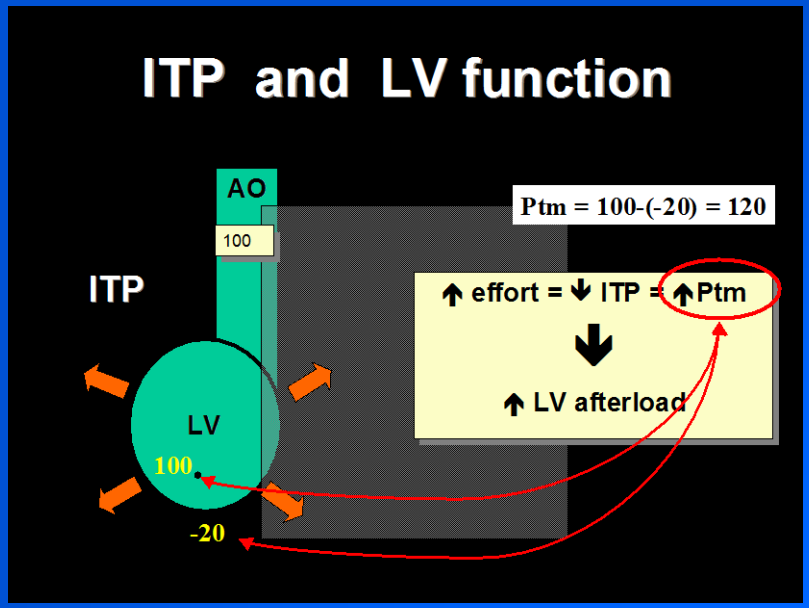
Pms= driving pressure to the RA

effects of changes in ITP: LV afterload

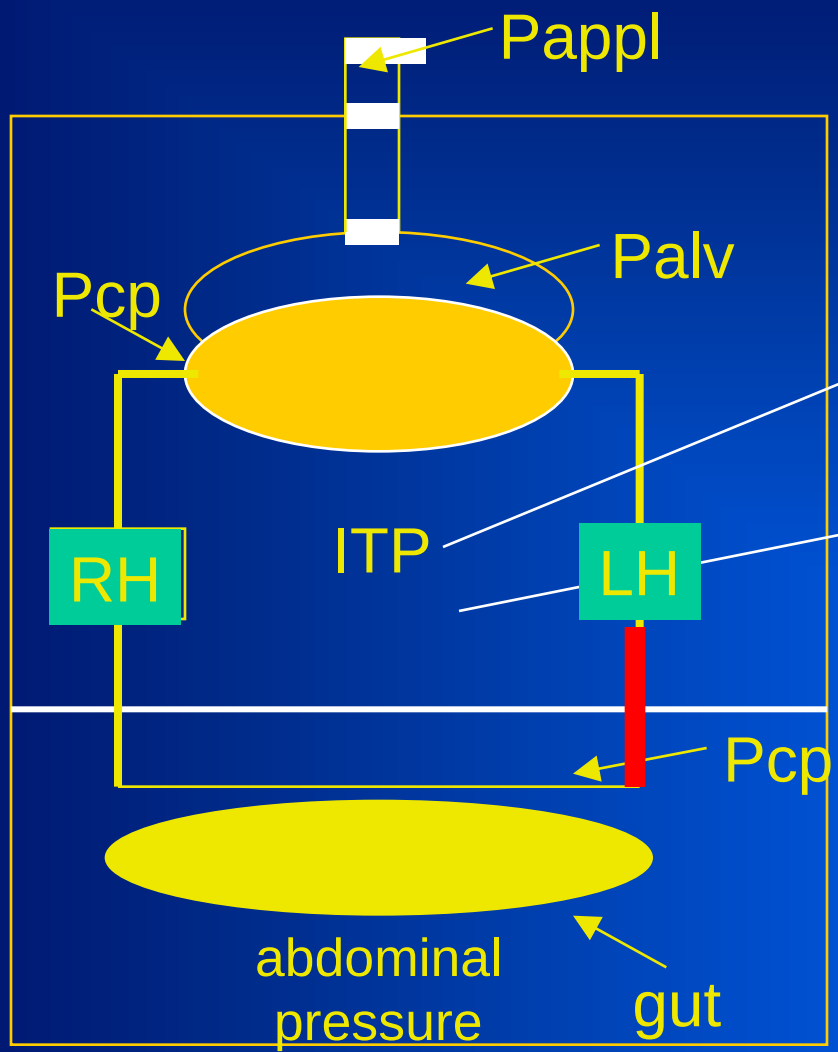


mechanical breath

spontaneous breath

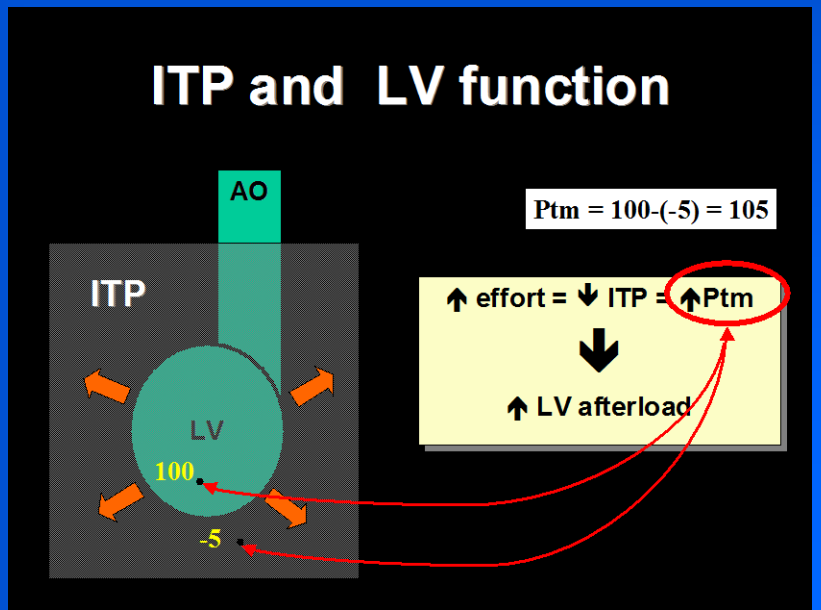


effects of changes in ITP: LV afterload



mechanical breath

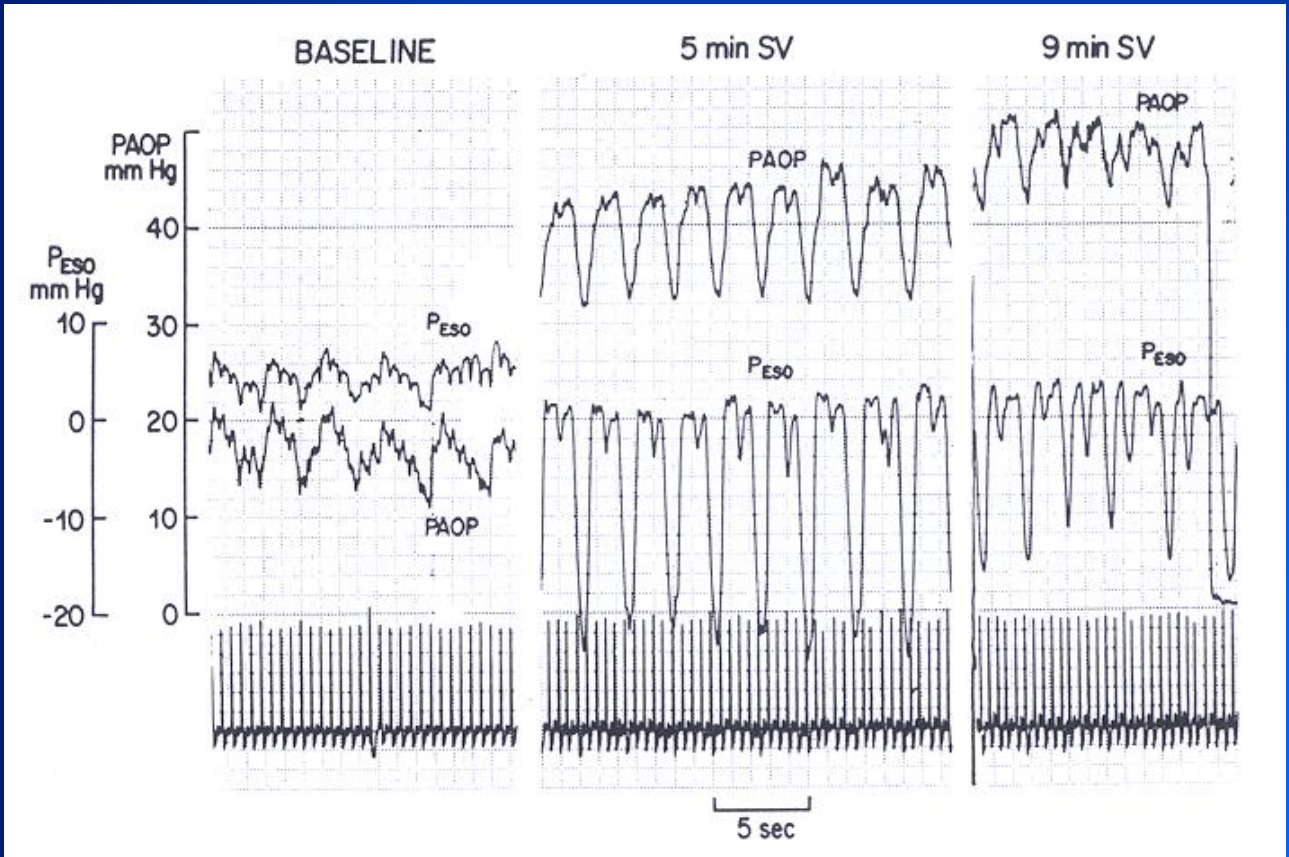
spontaneous breath



Anesthesiology
69:171–179, 1988

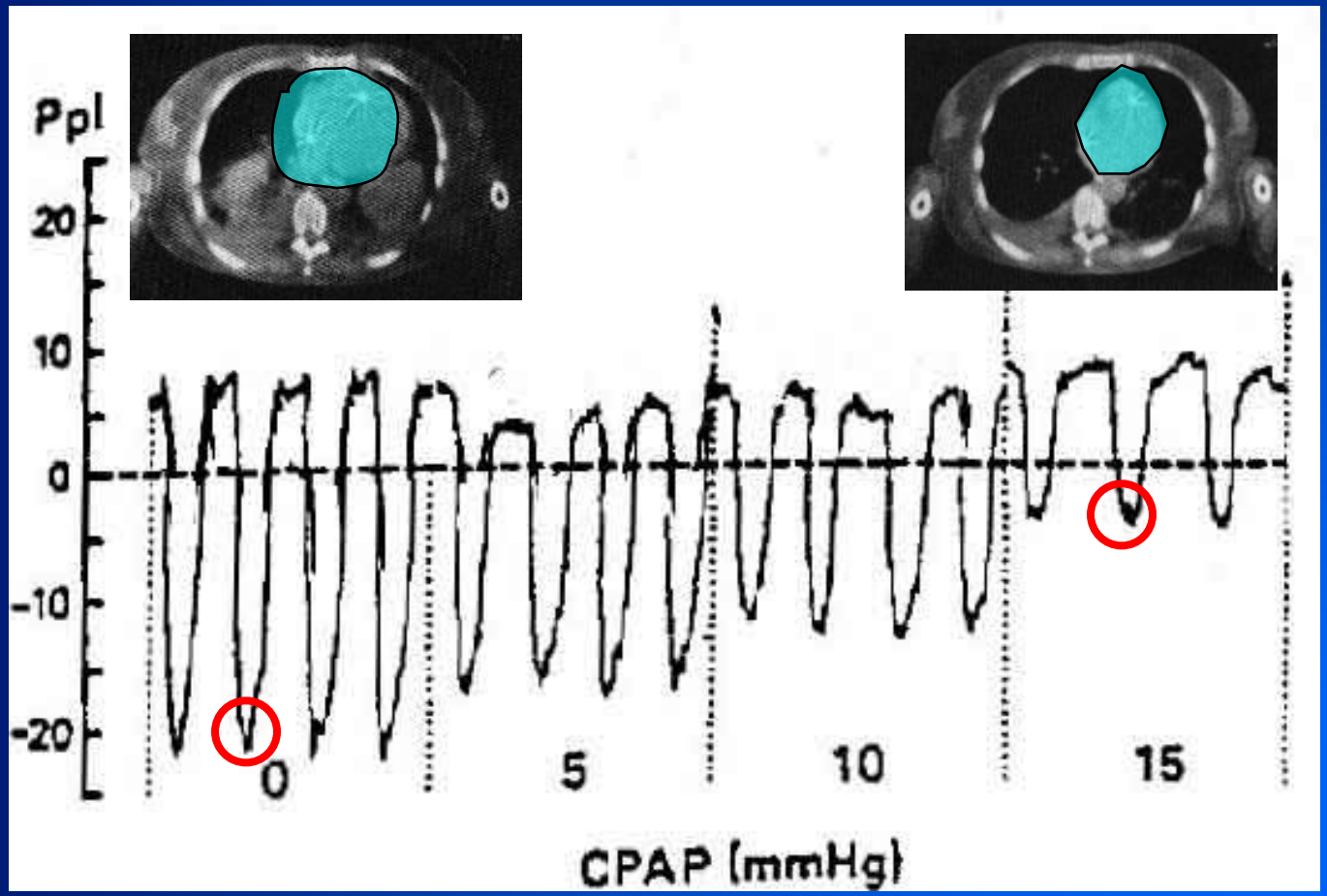
Acute Left Ventricular Dysfunction during Unsuccessful Weaning from Mechanical Ventilation

Francois Lemaire, M.D., Jean-Louis Teboul, M.D.,† Luc Cinotti, M.D.,‡ Guillen Giotto, M.D.,§ Fekri Abrouk, M.D.,§ Gabriel Steg, M.D.,§ Isabelle Macquin-Mavier, M.D.,¶ Warren M. Zapol, M.D.***



CPAP IN CARDIOGENIC PULMONARY EDEMA

Rasen et al: Chest 1985; 87: 158-162



Myocardial Systolic Function Increases During Positive Pressure Lung Inflation

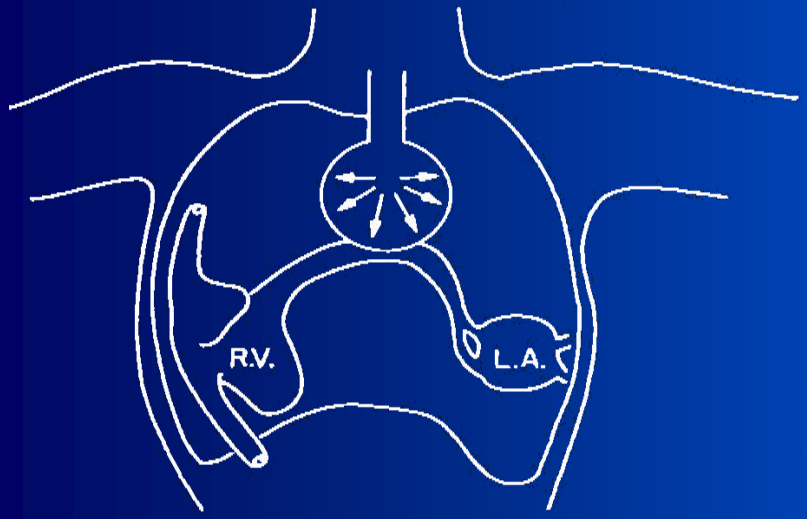
Michael F. Haney, Göran Johansson, Sören Häggmark, Björn Biber,
Michael F. Haney, MD, PhD, Göran Johansson, MS, Sören Häggmark, MS, and
Björn Biber, MD, PhD

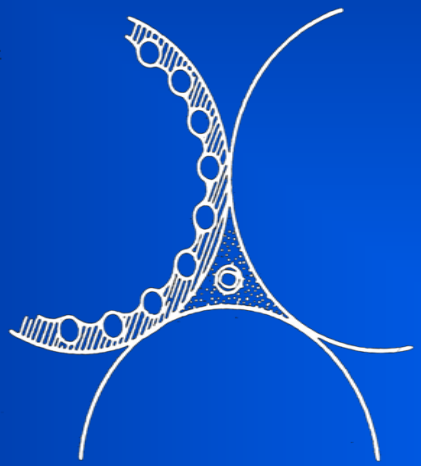
Anesthesiology and Intensive Care Medicine, Umeå University Hospital, Umeå, Sweden

Lung inflation with positive airway pressure may have rapid and dynamic effects on myocardial contractile function. We designed this study to assess the magnitude and time to onset of myocardial function changes during the initiation of single positive pressure lung inflation at clinically relevant inflation pressures. In 8 anesthetized 40-kg pigs, left ventricular pressures and volumes were measured directly (conductance volumetry). A 15 cm H_2O airway pressure plateau with lung inflation (PPLI-15) was performed, and 2 single beats from that sequence, one from resting apnea at zero airway pressure and the second from the point when the lungs were first maximally inflated, were selected for analysis. Systolic function variables for zero airway pressure and PPLI-15 were analyzed. Systolic elastance, derived from bilinear time-varying elastance curves, increased approximately 15% during PPLI-15 from zero airway pressure. This agreed with other systolic function variables that identified an increase in left ventricular contractile function for the lung inflation beat. Serial measurements of myocardial function should be conducted with constant airway pressure and lung inflation conditions.

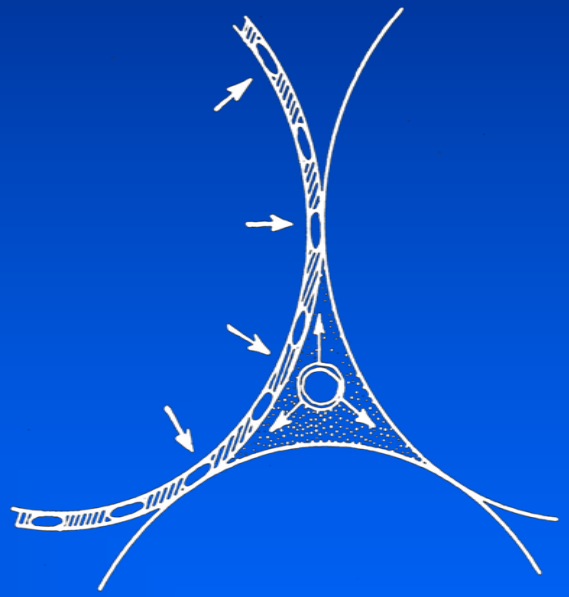
(Anesth Analg 2005;101:1269–74)

EFFECTS OF LUNG VOLUME CHANGES



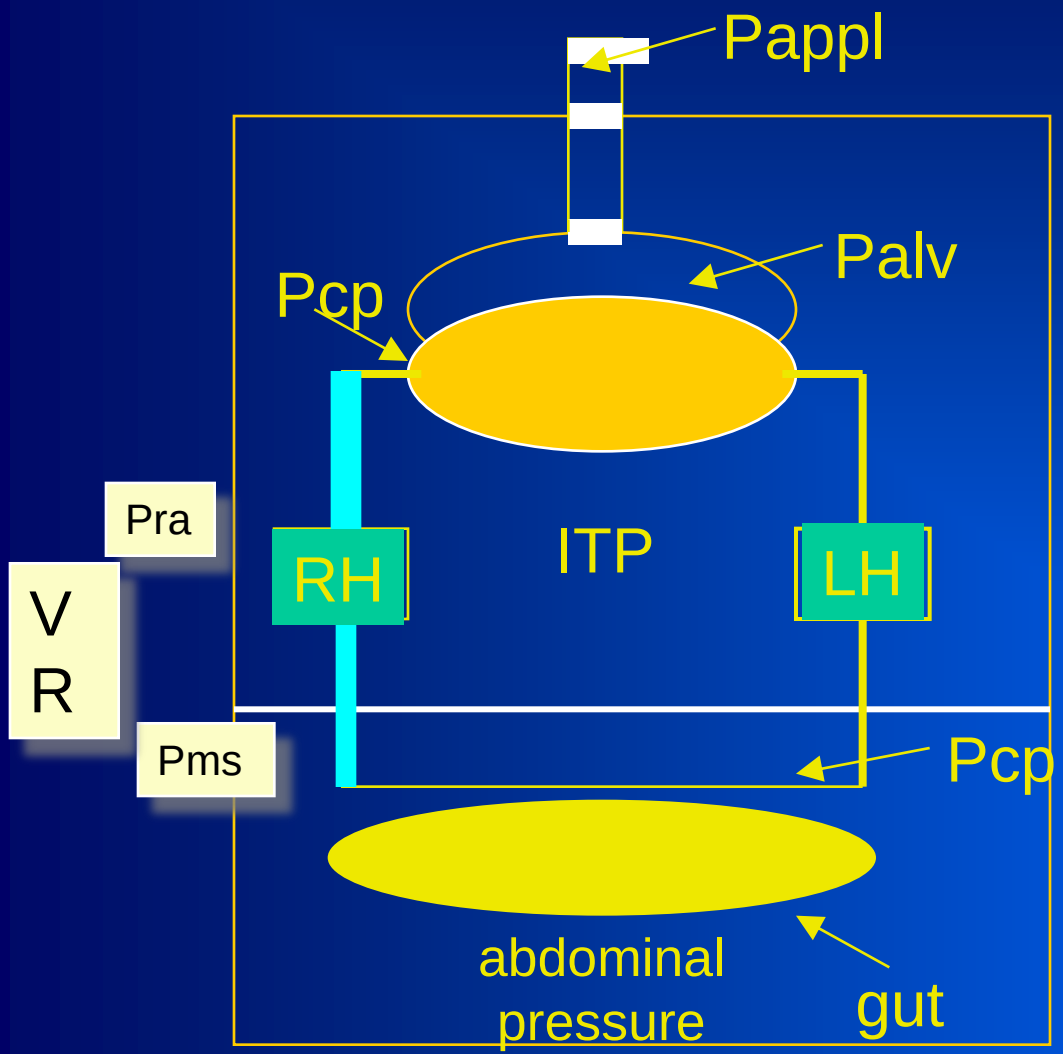


LOW LUNG VOLUME



HIGH LUNG VOLUME

Effects of the increase in **lung volume**: ***RV afterload and preload***



- Incresase in PAP afterload is due to increase in PA resistance

- Decrease in VR are due to increase in Pra because of increased PA resistance

EFFECTS OF POSITIVE INTRATHORACIC PRESSURE ON VENOUS RETURN

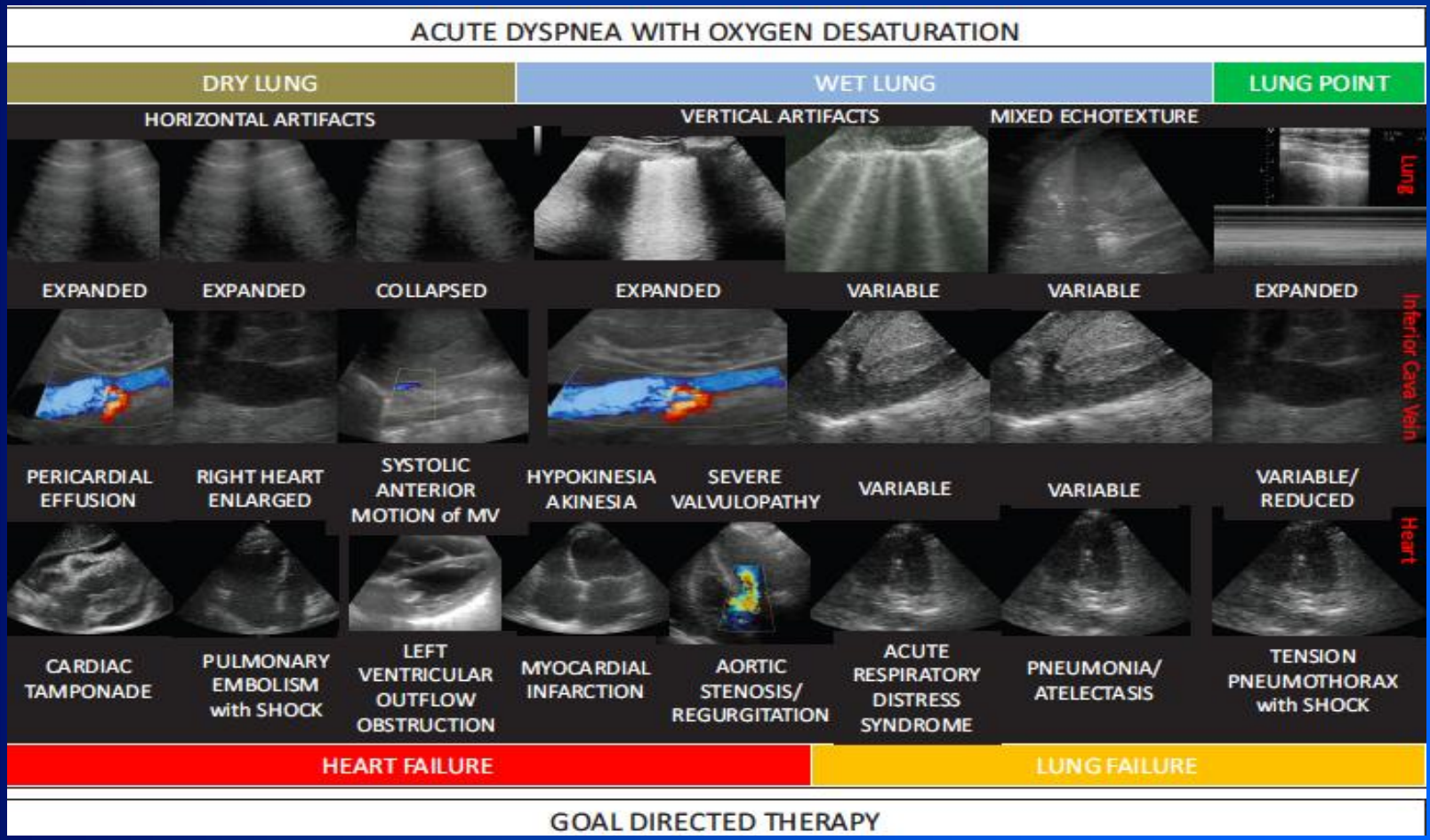
Pulmonary compliance	**HIGH (Emphisema)**	↓
	LOW (ARDS)	—
Chest wall compliance	**HIGH**	—
	LOW	↓

TAKE HOME MESSAGE

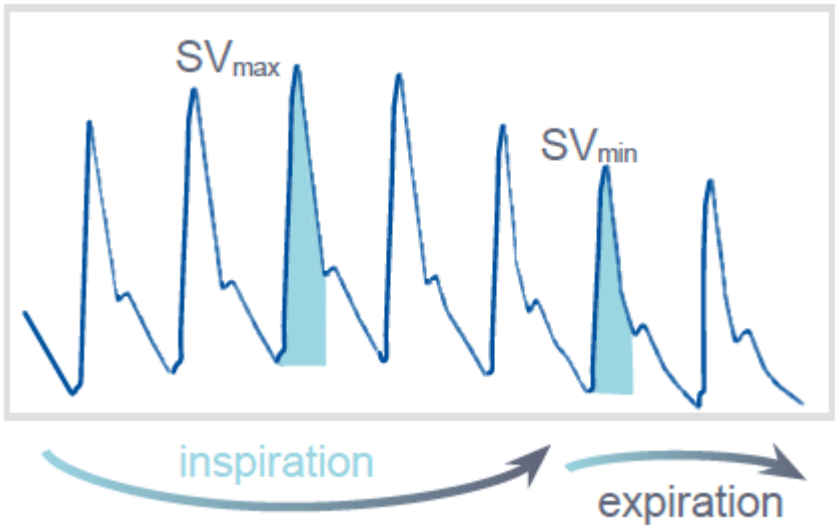
Chest ultrasound in Acute Respiratory Distress Syndrome

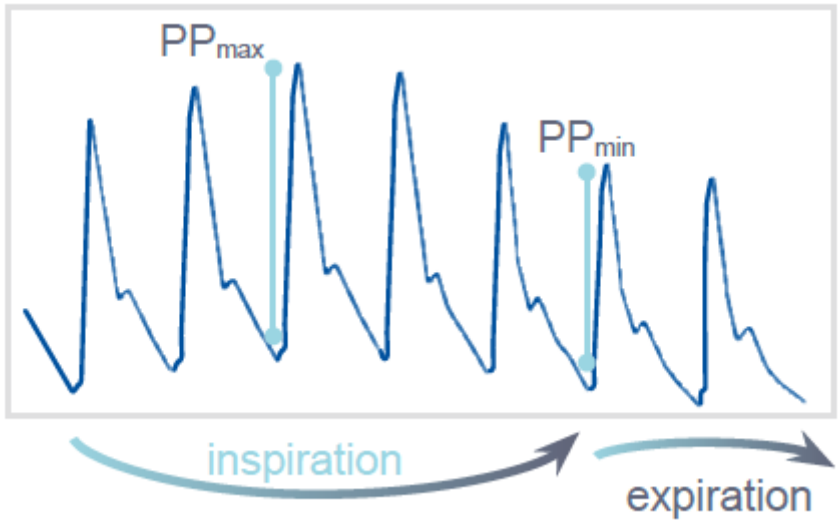
Corradi F., Brusasco C., Pelosi P. Curr Opin Crit Care 2014, 20:98–103

Pelosi P., Corradi F. Anesthesiology 117(4):696-698, 2012



Known Variations to Indexes at the Bedside





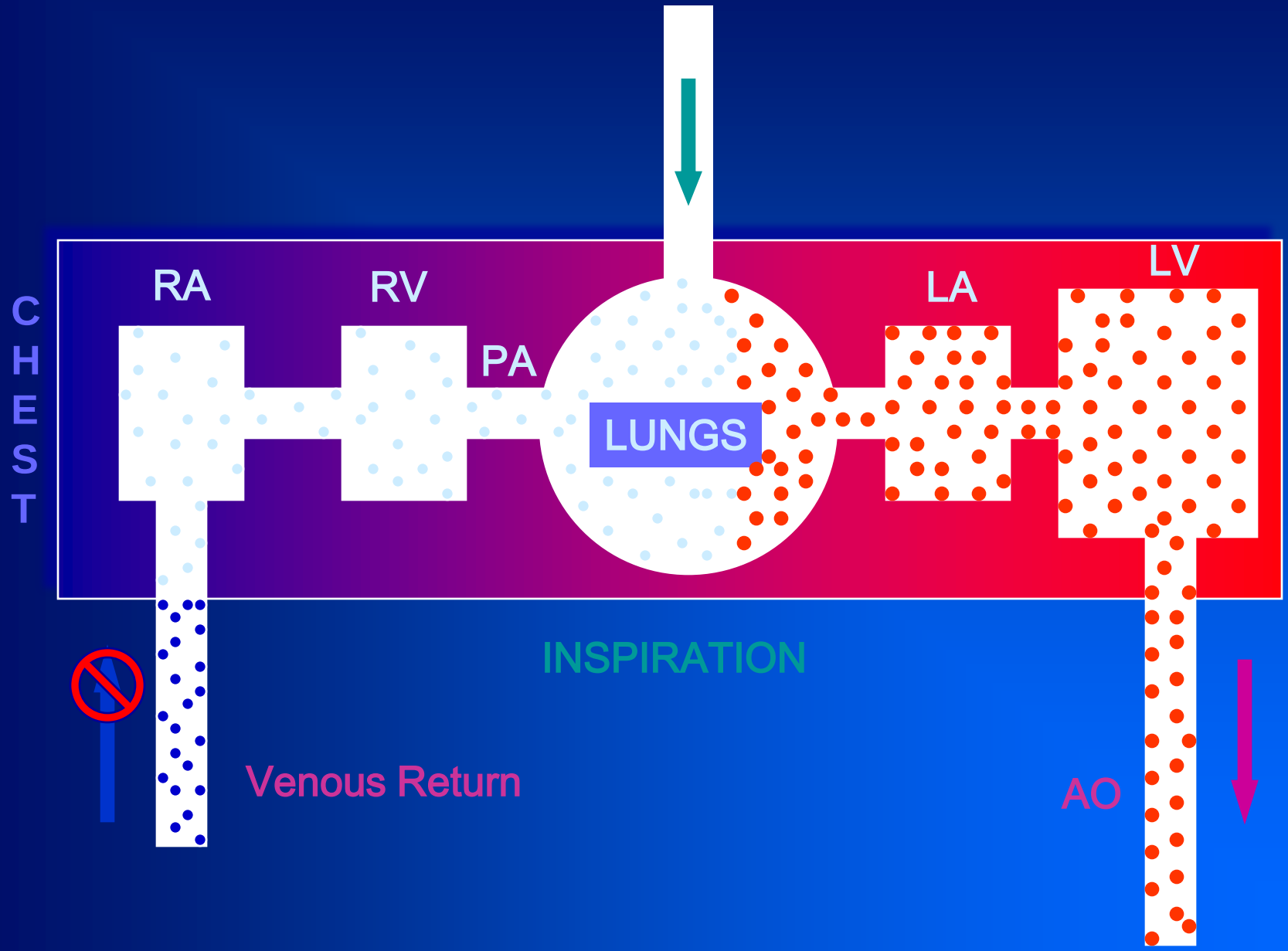
$$SVV\ (\%) = \frac{SV_{MAX} - SV_{MIN}}{(SV_{MAX} + SV_{MIN})/2} \times 100$$

$$PPV\ (\%) = \frac{PP_{MAX} - PP_{MIN}}{(PP_{MAX} + PP_{MIN})/2} \times 100$$

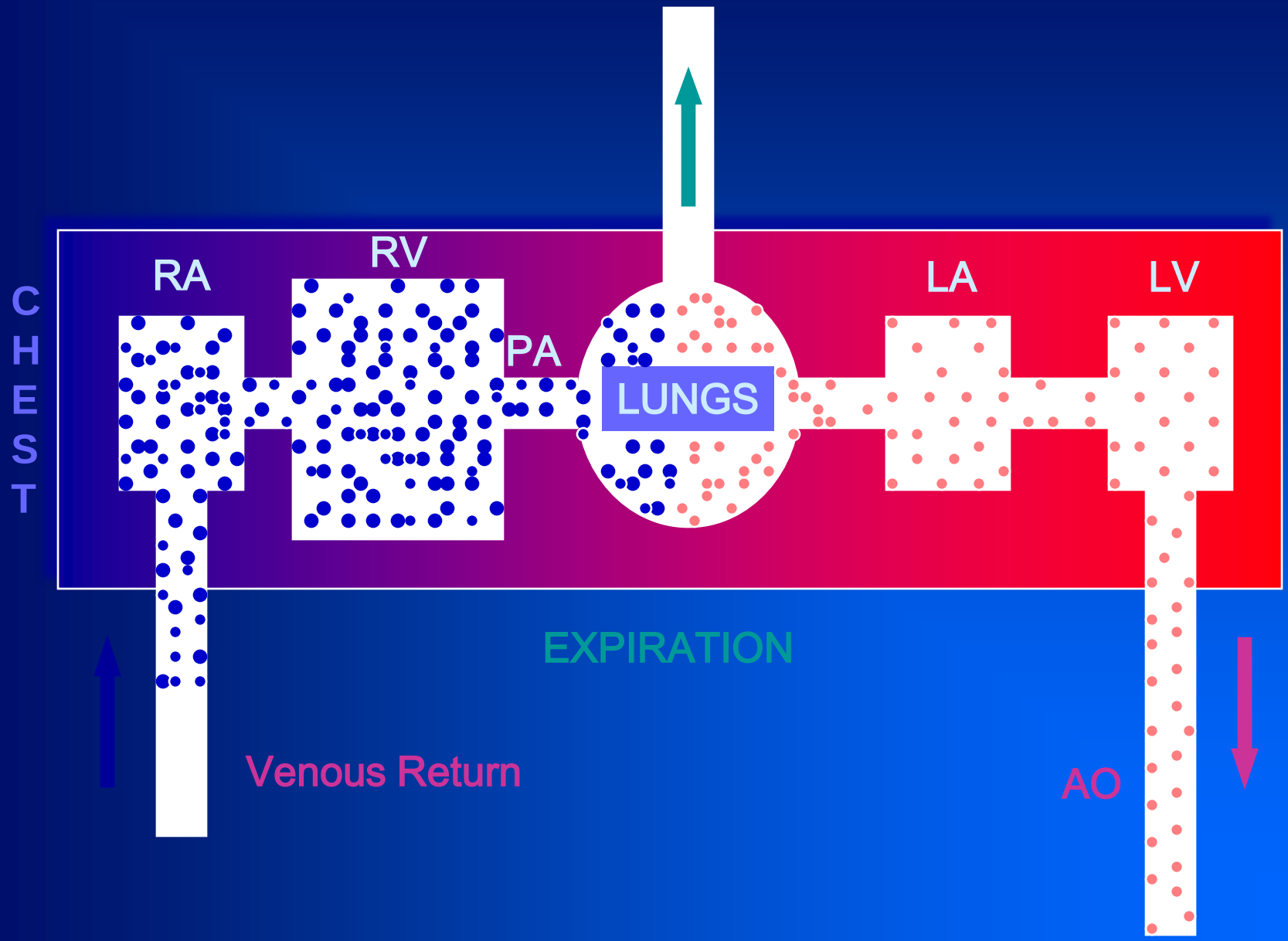
Why use these Dynamic Indexes?

Biais, *Anesthesiology* 2012

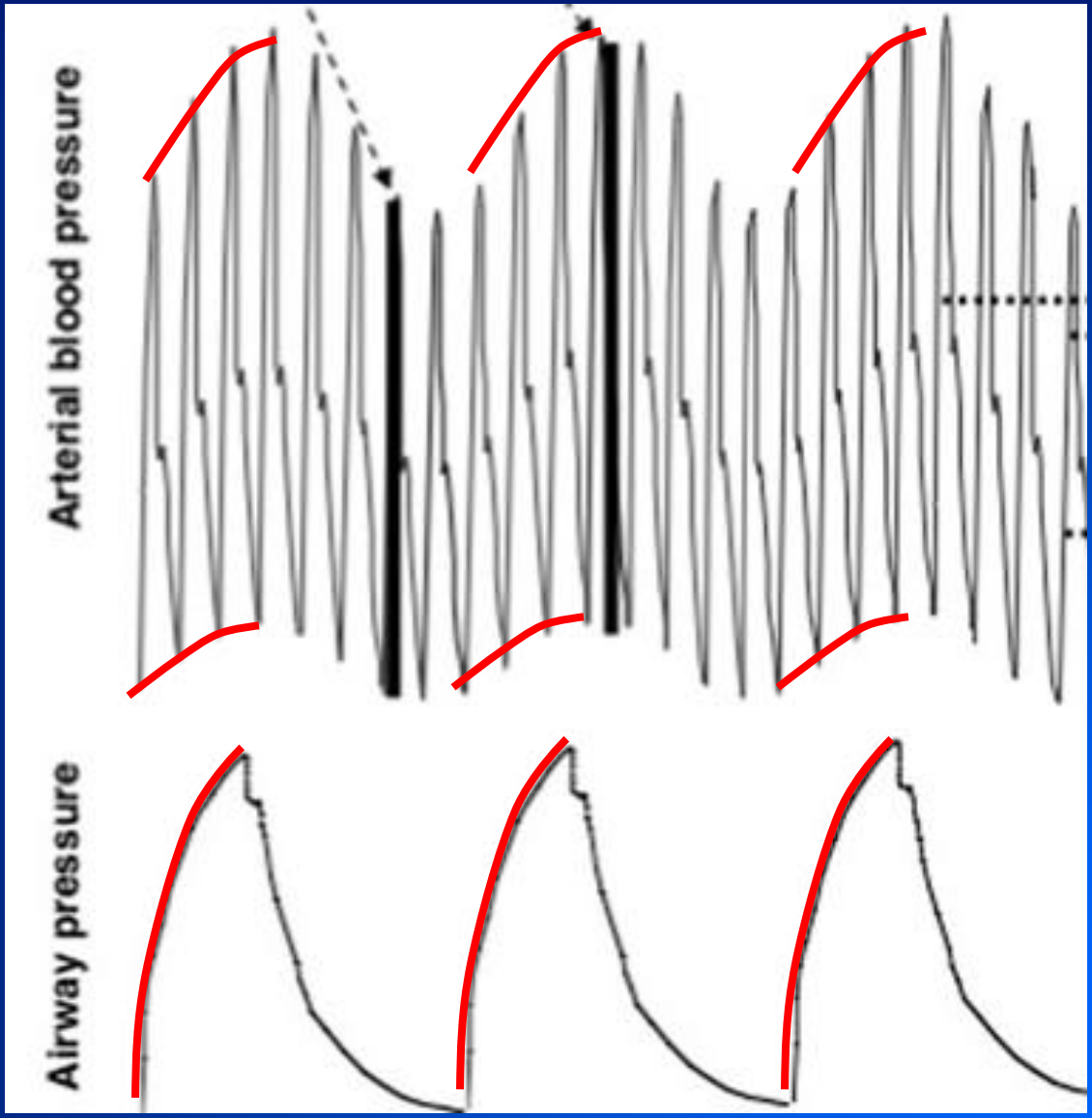
MECHANICAL VENTILATION



MECHANICAL VENTILATION

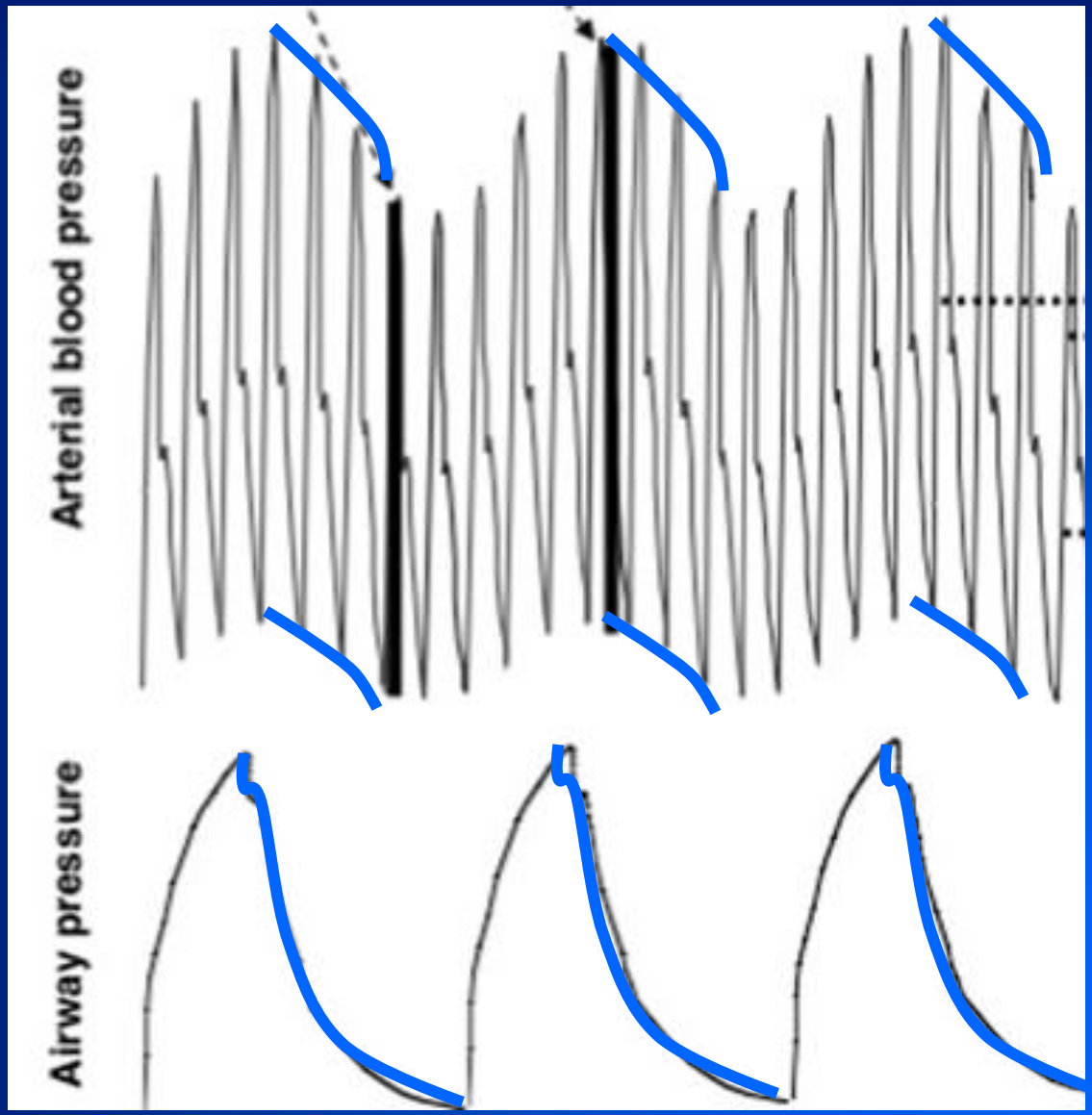


Overall Picture - Inspiration



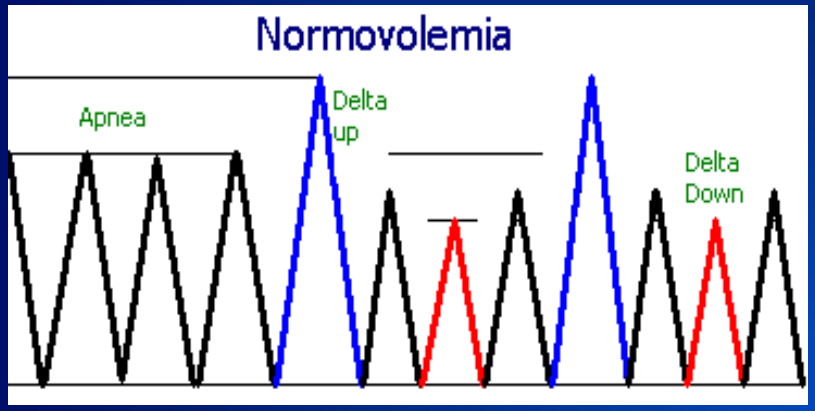
Broccard, *J Clin Monit Comput* 2012

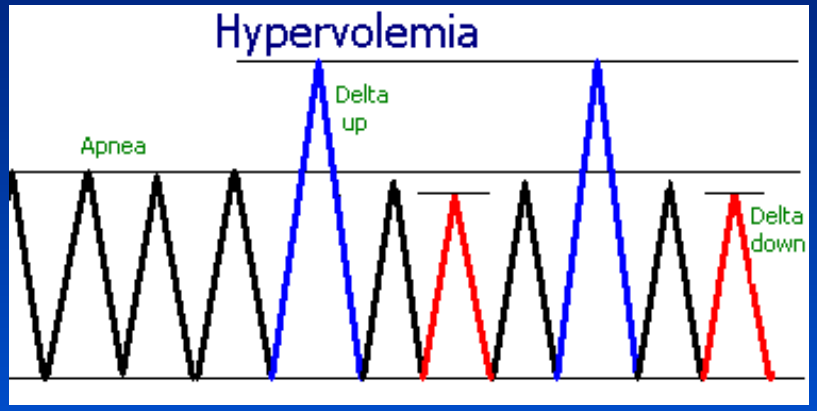
Overall Picture - Expiration

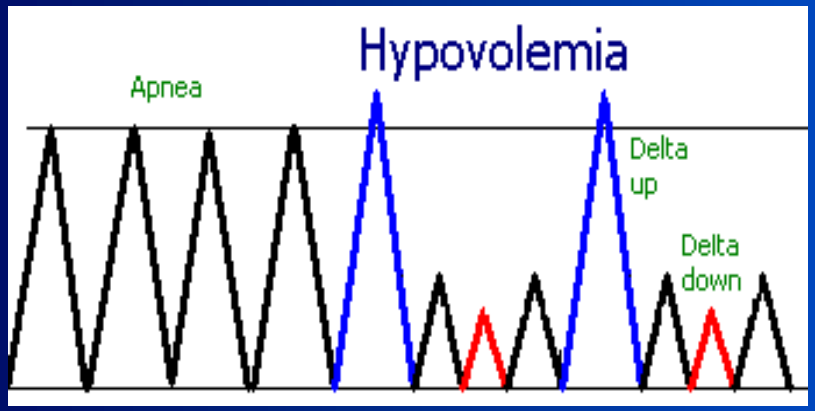


Broccard, *J Clin Monit Comput* 2012

Assessing Fluid Responsiveness by the Systolic Pressure Variation in Mechanically Ventilated Patients



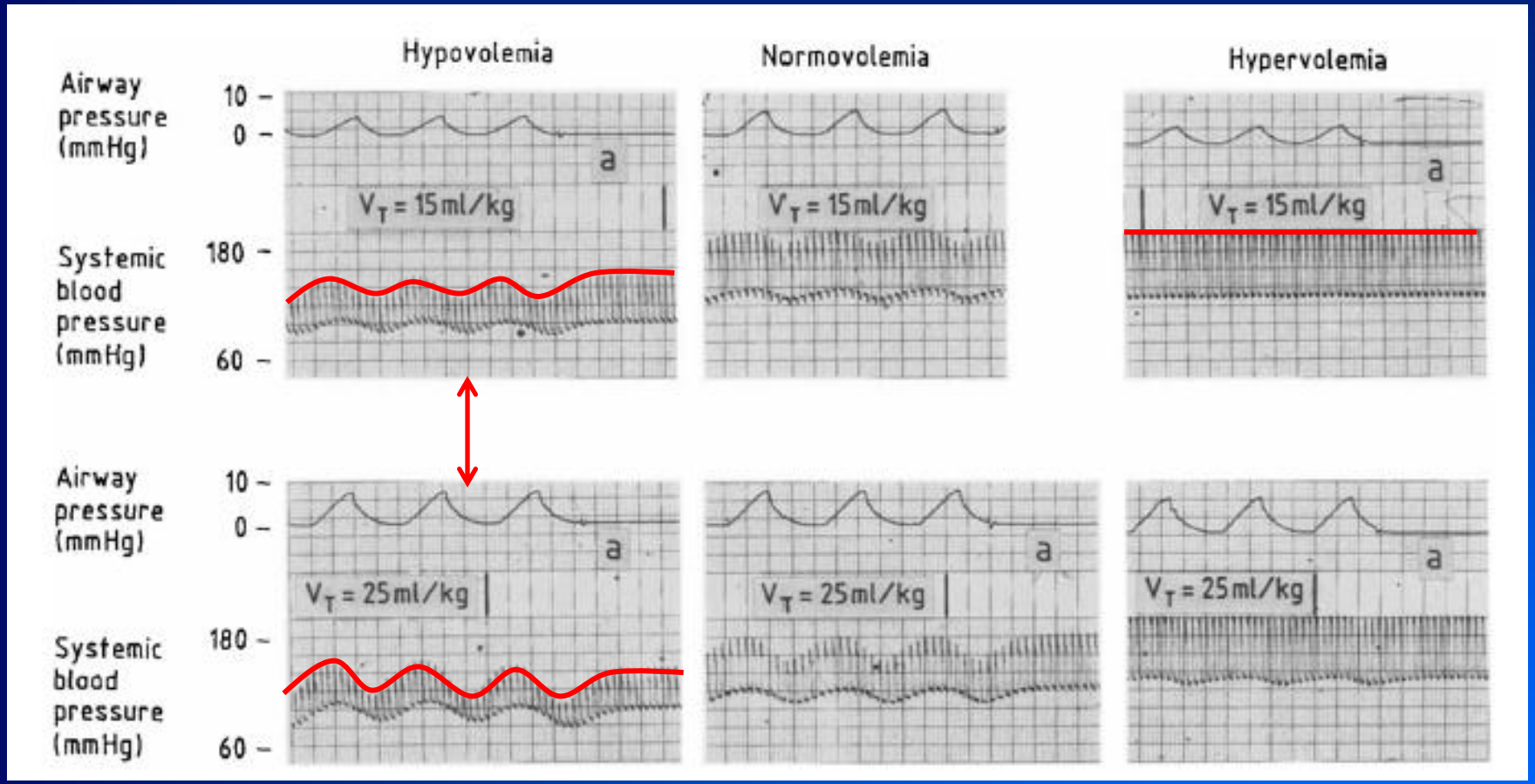




	N.V. mmHg
SPV	8 - 10
Delta down	5 - 6
Delta up	2 - 4

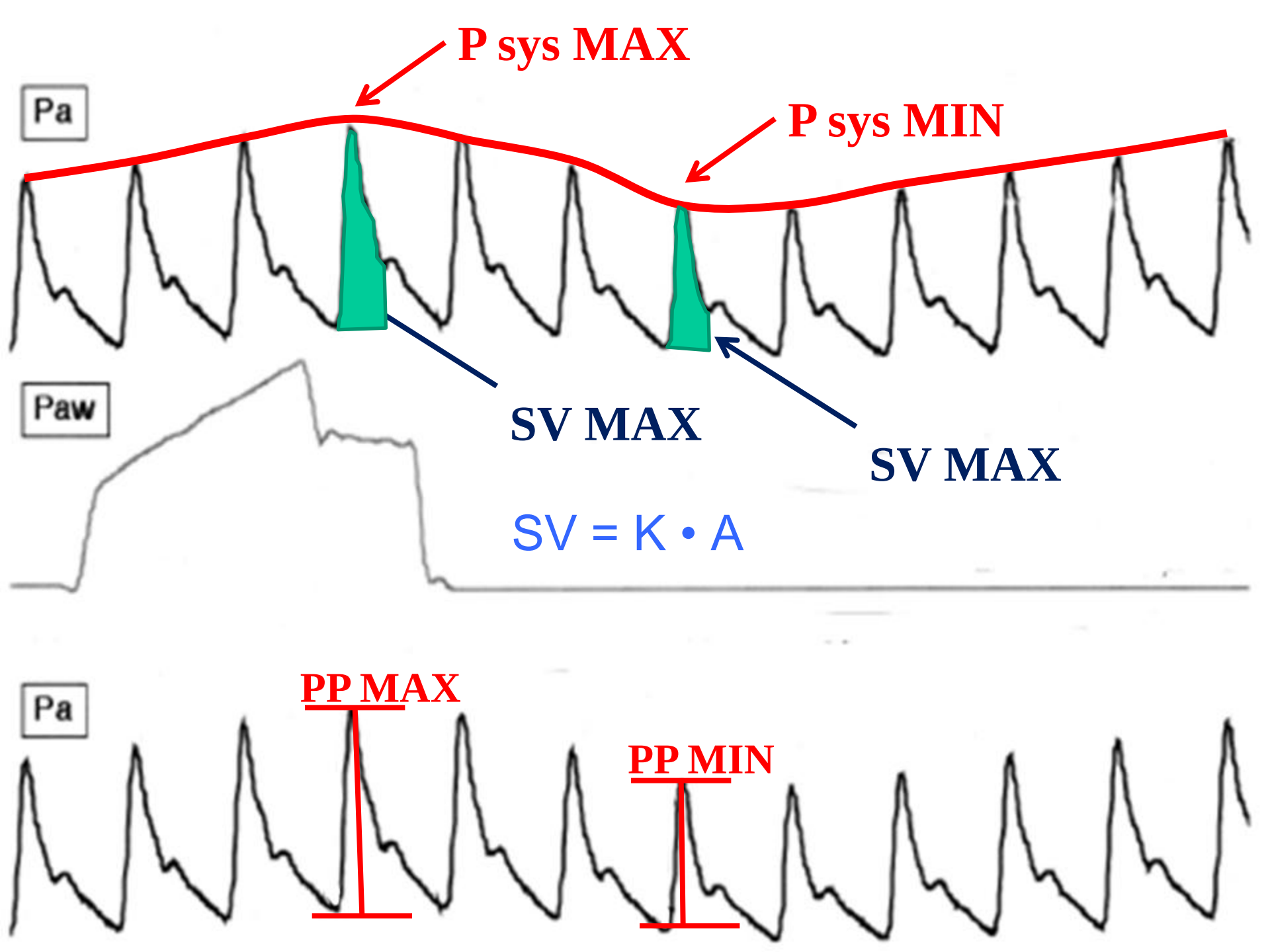
Perel A. *Anesthesiology* 1998

Looking back in 80’s…



Predict who could be responsive to volume restoration

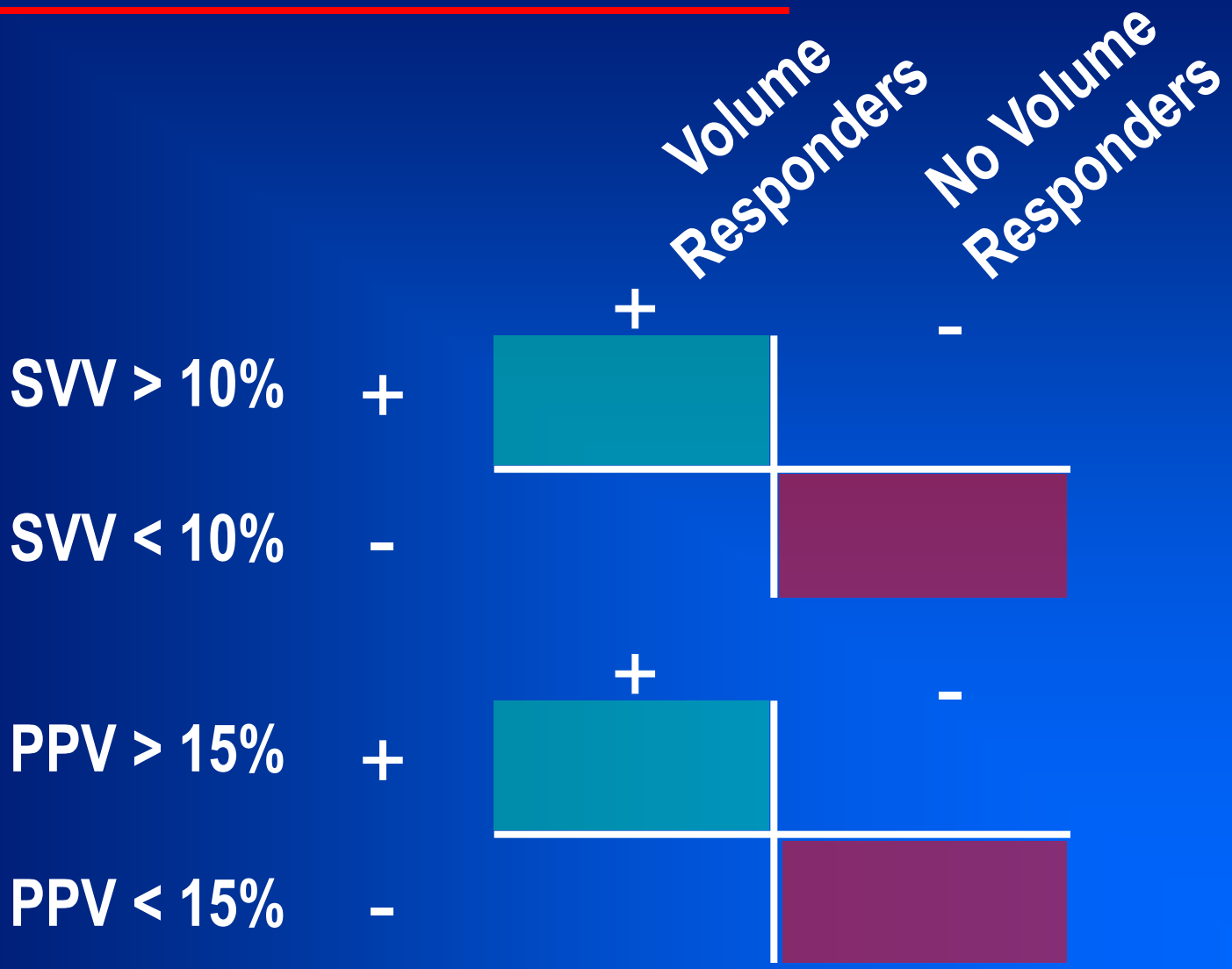
Szold, *Intensive Care Med* 1989


P sys MAX
P sys MIN
Pa
SV MAX
SV MAX
Paw
SV = K • A
Pa
PP MAX
PP MIN

STROKE VOLUME VARIATION

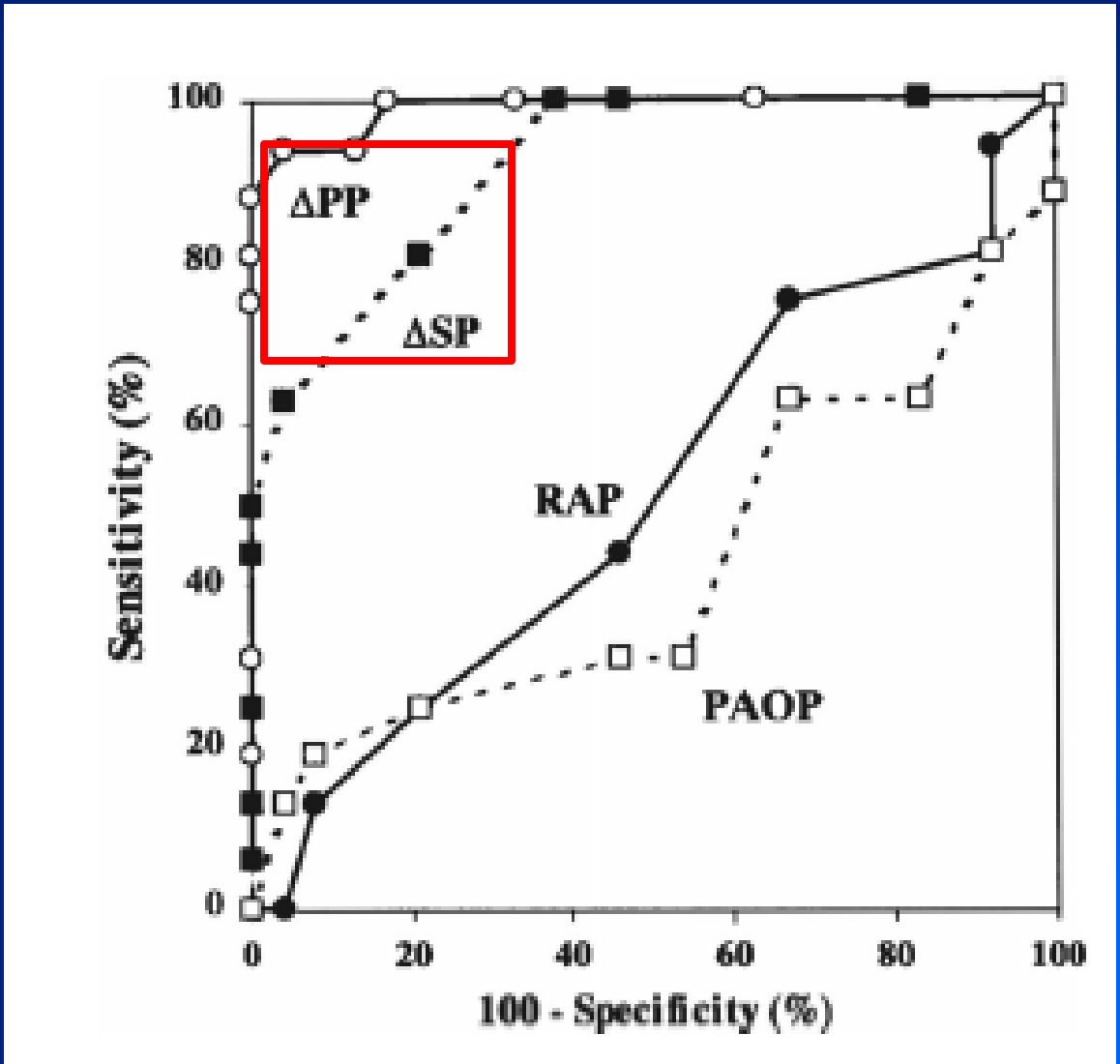
$$SVV = \frac{SV_{max} - SV_{min}}{SV_{mean}}$$

Which Parameter can better recognize it?

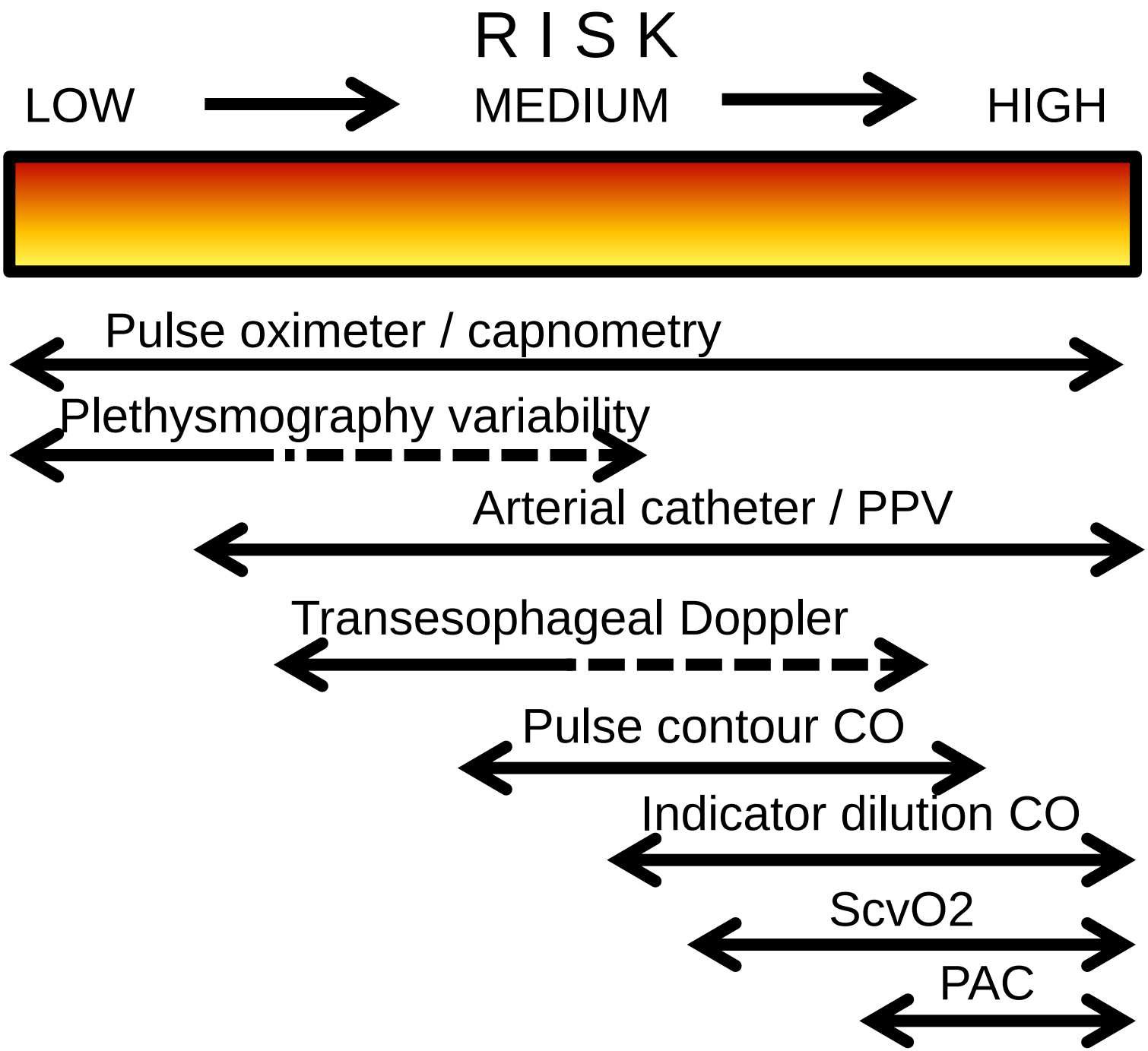


Michard, *Chest 2002*

Which Parameter can better recognize it?



Michard, *Chest 2002*


RISK
LOW
MEDIUM
HIGH
Pulse oximeter / capnometry
Plethysmography variability
Arterial catheter / PPV
Transesophageal Doppler
Pulse contour CO
Indicator dilution CO
ScvO2
PAC

REVIEW

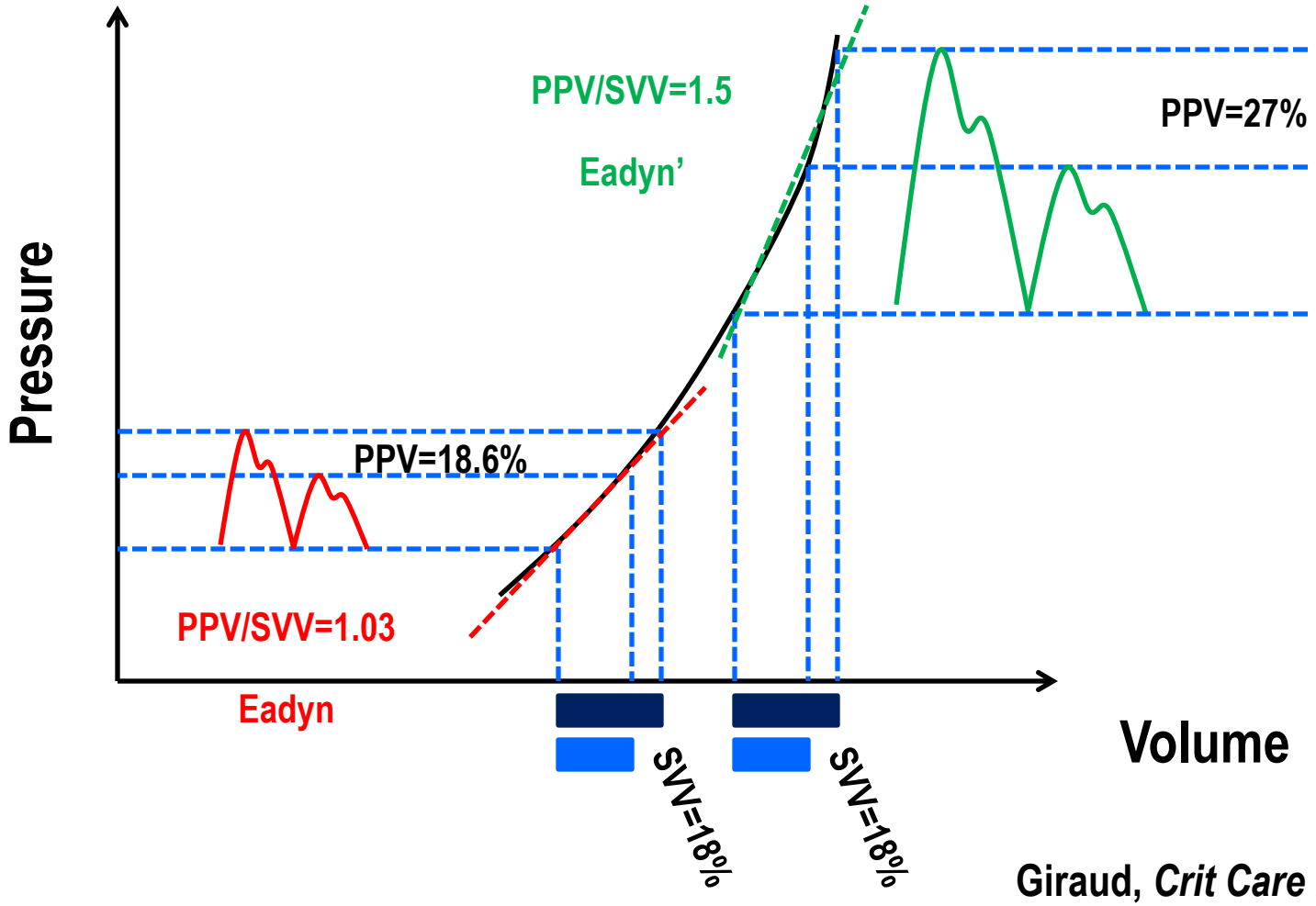
Bench-to-bedside review: Functional hemodynamics during surgery - should it be used for all high-risk cases?

Limitations of functional hemodynamic parameters

- Spontaneous breathing
- Tidal volume should be 8 ml/kg
- Nonstandardized airway pressure/respiratory rate
- Open-chest conditions may affect the FHP
- Pediatric patients
- Nonsinus rithm
- Right heart failure

Perel et al. Critical Care

PPV/SVV – Dynamic Arterial Elastance



Giraud, *Crit Care* 2011

Monge García et al. Critical Care 2011, 15:R15
http://ccforum.com/content/15/1/R15

CRITICAL CARE

RESEARCH Open Access

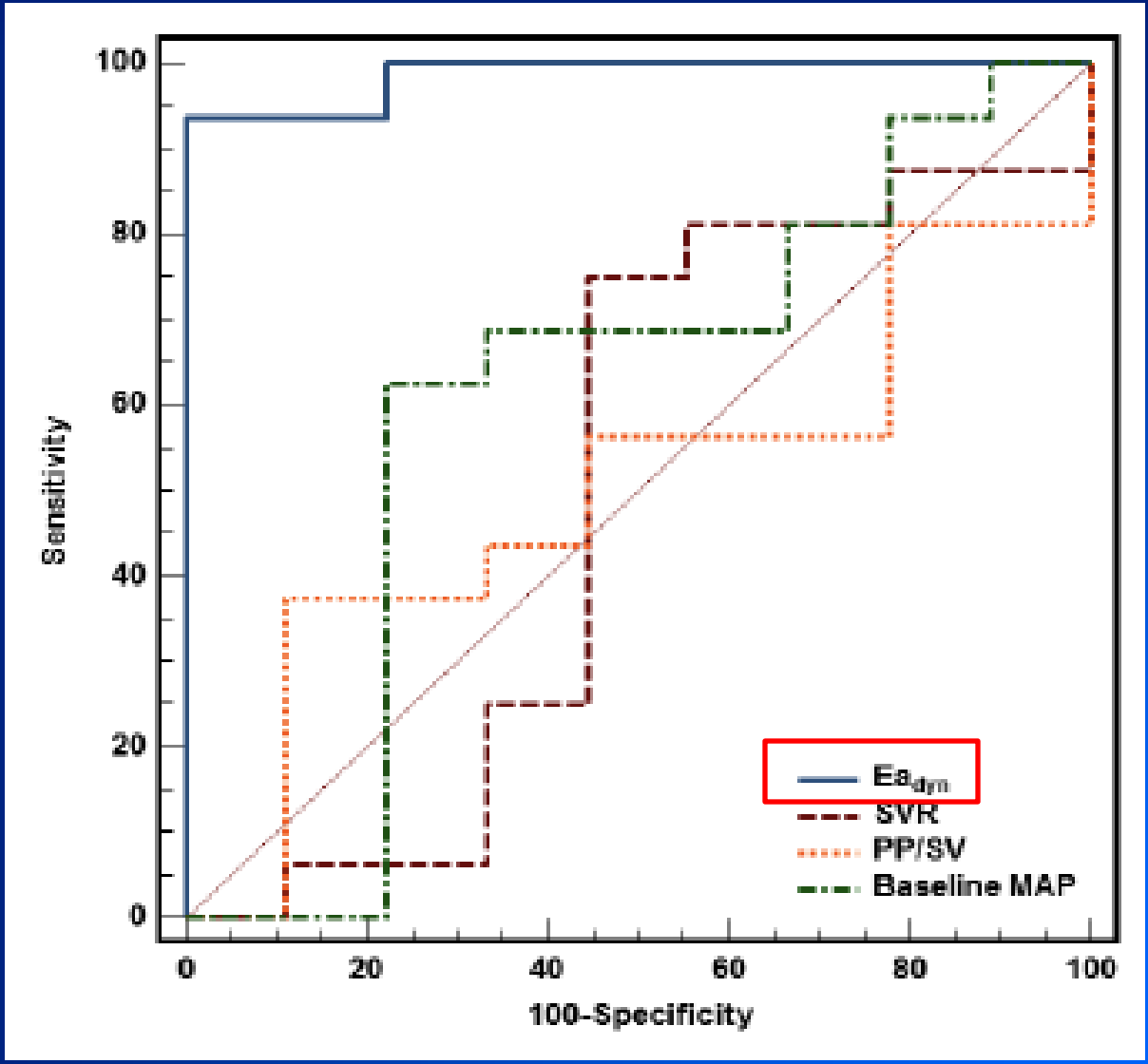
Dynamic arterial elastance to predict arterial pressure response to volume loading in preload-dependent patients

Manuel Ignacio Monge García*, Anselmo Gil Cano, Manuel Gracia Romero

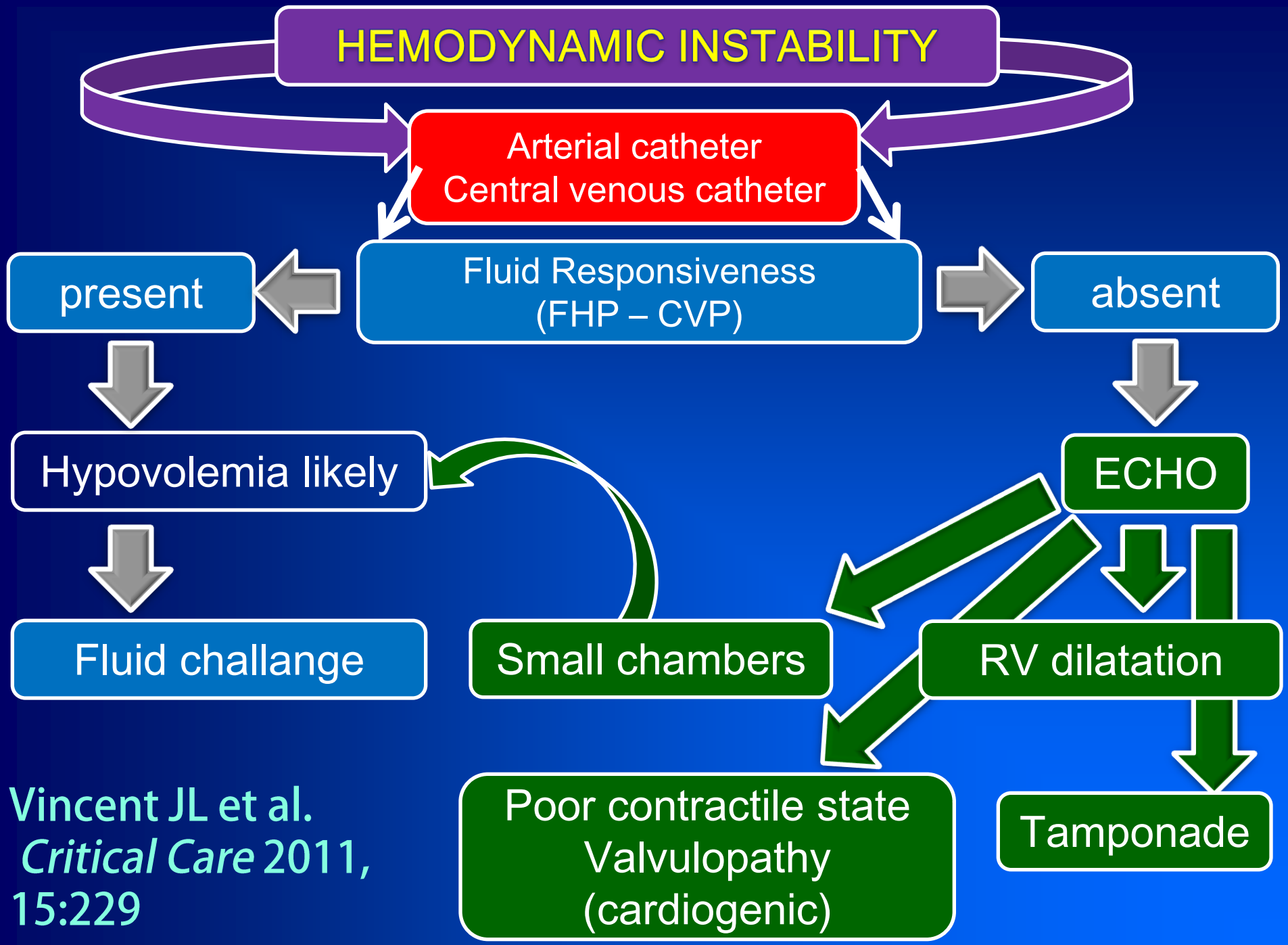
Parameter	Preinfusion	Postinfusion
Dynamic arterial elastance		
Responders	1.34 ± 0.45[c]	0.85 ± 0.21[b,e]
Nonresponders	0.75 ± 0.12	0.64 ± 0.21
SVR, dyn s cm^{-5}		
Responders	889.66 ± 392.03	881.19 ± 344.16
Nonresponders	870.95 ± 379.38	774.42 ± 377.17[d]
PP/SV, mmHg/mL		
Responders	0.79 ± 0.36	0.90 ± 0.37[b,d]
Nonresponders	0.73 ± 0.19	0.66 ± 0.16

Cuttoff Values?

PPV/SVV – Dynamic Arterial Elastance

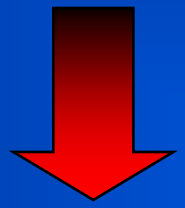


Monge-Garcia, *Crit Care* 2011


HEMODYNAMIC INSTABILITY
Arterial catheter
Central venous catheter
Fluid Responsiveness
(FHP – CVP)
present
absent
Hypovolemia likely
Fluid challange
Small chambers
ECHO
RV dilatation
Poor contractile state
Valvulopathy
(cardiogenic)
Tamponade
Vincent JL et al.
Critical Care 2011,
15:229

Physiological background

$$Sat_vO_2 = Sat_aO_2 - \frac{VO_2\ (mL/min)}{Q\ (L/min)} * \frac{1}{Hb\ (gr/L) * 1.39}$$



$$Sat_vO_2 = \text{Lung} - \frac{\text{metabolism}}{\text{hemodynamic}} * \frac{1}{\text{carrier}}$$

Venous blood

Venous PO_2	→	Reflects the tissue oxygenation
$PvCO_2$	→	Reflects the arterial $PaCO_2$ / the tissue acidosis
Sat_vO_2	→	Reflects Sat_aO_2, VO_2/Q, Hb
Base $excess_v$	→	Assessment of metabolic status (always different from the arterial)

Venous-arterial sampling

It allows

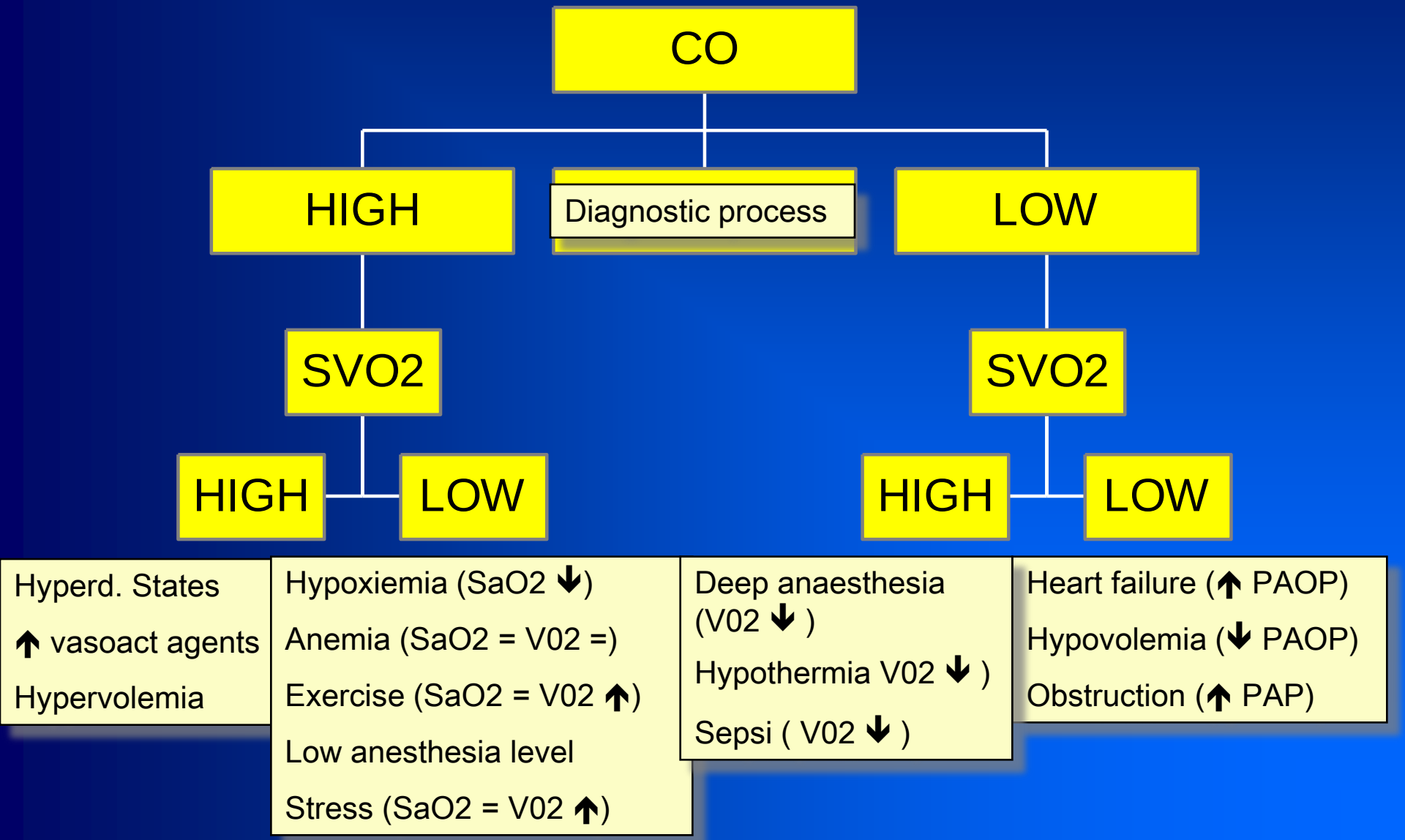
- The shunt computation
- $\Delta a\text{-}vO_2$ difference
- $(Sat_a - Sat_v)/Sat_a$
- ΔPCO_2
- $\Delta PCO_2/\Delta a\text{-}vO_2$ content
- LACTATE !

Venous-Arterial blood gases allow to:

- Define the oxygenation status (PaO_2-Shunt (not replaceable))
- Define the ventilatory status
- Infere on the hemodynamic status ($\Delta a\text{-}vO_2$, Sat_vO_2, $(Sat_aO_2 – Sat_vO_2)/Sat_aO_2$ (not replaceable))
- Define the energy failure – BE, pH, $\Delta PCO_2/\Delta a\text{-}vO_2$

*SVO2 = SaO2 – VO2/Q * 1/(1.36*Hb)* Fick equation

SVO2 = LUNG - METABOLISM/HEMODYNAMIC * 1/ANEMIA



Conclusions

- ✓ Preload, flow and flow adequacy
- ✓ Non invasive and Invasive methods (Echo and other Methods)
- ✓ Ventilatory assist affects hemodynamics
- ✓ Flow adequacy: consider $ScvO_2$ and Lactate