Hemodynamic: Meassurement, Interpretation and Application

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History Of Haemodynamic Monitoring

- **Assessment of hemodynamic function:**
  - It start from patient's physiologic variables or **"vital signs"** such as heart rate, blood pressure, and urinary output
- **→1960's:**
  - Shock is **tachycardia** and **hypotension**
  - But, as clinicians gained more experience in treating critically ill patients, it became apparent that **normalization** of vital sign was **not necessarily** sufficient to reverse a patient's shock . mfda

History Of Haemodynamic Monitoring

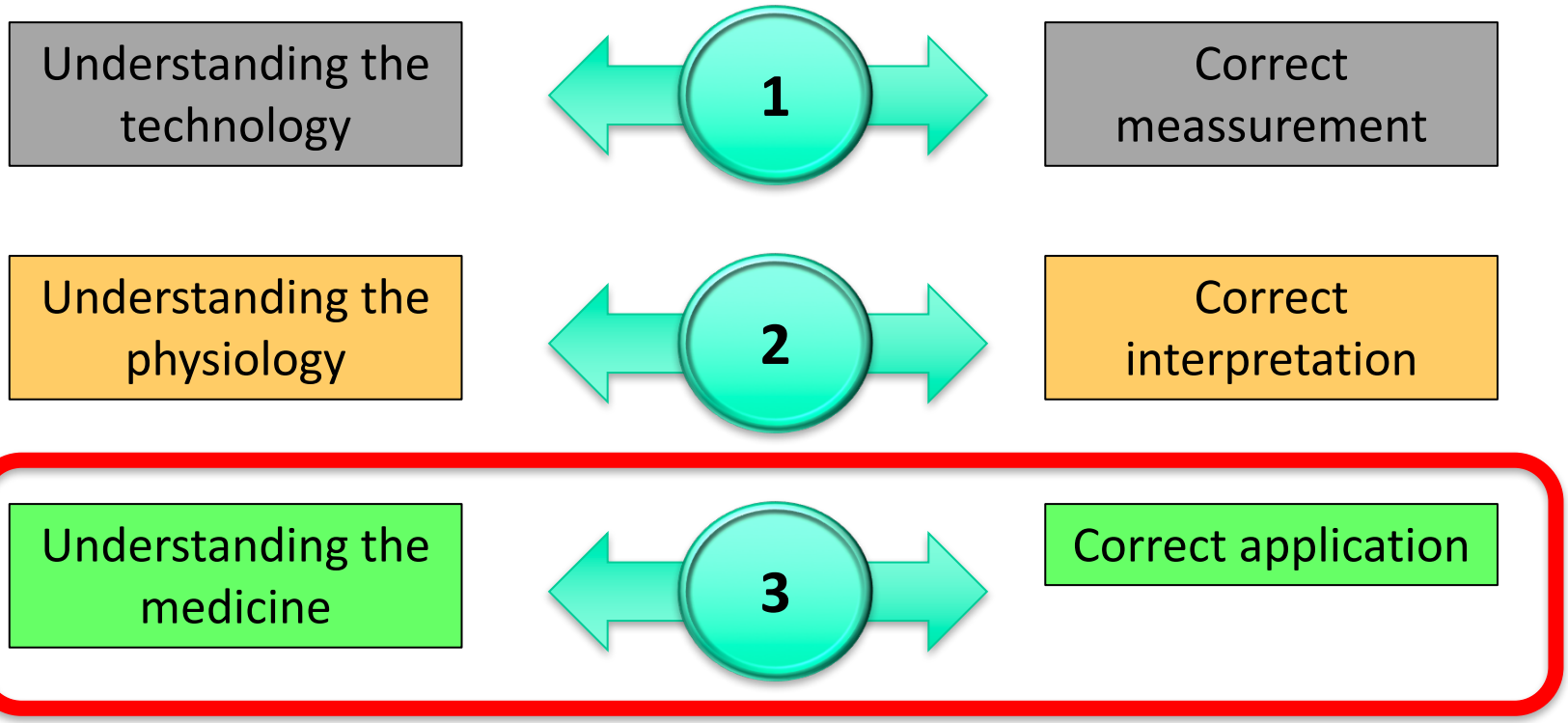
- **1970:**
  - Swan and Ganz introduced the flow-directed pulmonary artery catheter at the bedside
- **1972:**
  - **calculate cardiac output** using the thermodilution technique.
  - This revolutionary advance in physiologic monitoring became the **standard of care** by the late 1970's in patients with multisystem organ dysfunction or refractory shock.
- **1980's:**
  - Continuous mixed venous oximetry (SvO_2) capability was added as the importance of **oxygen delivery, oxygen consumption, and oxygen transport balance** in the diagnosis and management of shock states became clear.
- **Early1990's :**
  - Catheters capable of calculating right ventricular volumes became available further improving **preload assessment** in the critically ill.

Introduction..3

- **2000:**
  - **Bio-impedance, pressure-wave contour analysis, transpulmonary thermodilution, doppler technology and transesophageal echocardiography)** designed to **continuously** assess hemodynamic function and oxygen transport
  - Although most modern bedside monitoring systems will readily measure and calculate standard hemodynamic variables, ***a working knowledge*** of the equations necessary to arrive at such variables is essential to understanding those the **pathophysiology, technology and appropriate treatment** for the various shock states encountered in the intensive care unit.
  - It is only by understanding the derivation of the equations that the clinician will truly understand the therapeutic interventions that will benefit the patient

Fundamentals of Hemodynamic monitoring

The 3 magic push-button



Techniques
of Hemodynamic monitoring

→ Continuous !

As the patient's haemodynamic status may change **rapidly**, **continuous hemodynamic monitoring** will provide information allowing rapid adjustment of therapy

Goal

of Hemodynamic monitoring

To ass

optimize CA

ve and maintain

adeq TISSUE PERFUSION

It's all about cardiac function

So, first you have to know the Cardiac Physiology

Cardiovascular Physiology

1) Blood – carries nutrients

2) Heart – creates pressure gradient – blood flow

3) Peripheral Circulation – carries blood to tissues

*The **primary function** of the **Cardiovascular system** is to*

1) deliver nutrients/oxygen and

2)remove wastes/CO2

from the cells in your body

Physiology of cardiac output

Cardiac output

1. Cardiac Output (in liters/minute) is defined as the amount of blood ejected from the ventricle (primarily the left ventricle) in a minute.
2. Cardiac output is the term that is used when discussing the pumping effectiveness and ventricular function of the heart – **the cardiac performance**

Cardiac output

Cardiac Output = Heart Rate x Stroke Volume

Where:

Heart Rate = beats/min

Stroke Volume = amount of blood ejected from ventricle in one beat

By altering either heart rate or stroke volume, cardiac output can be manipulated.

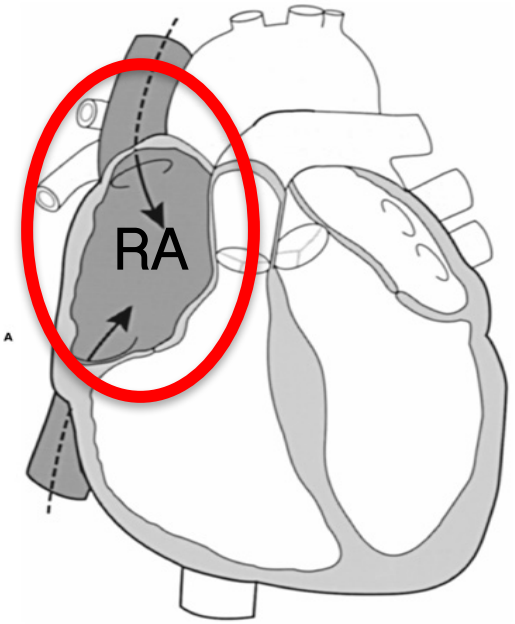


Figure 5-4 A, Illustration depicting blood flow into the right atrium during diastole.

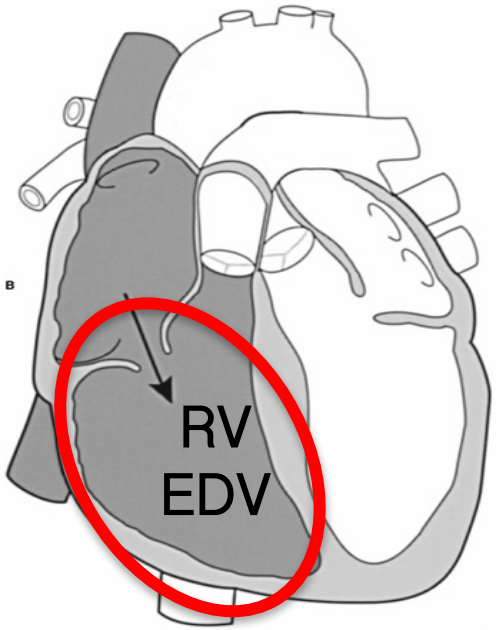
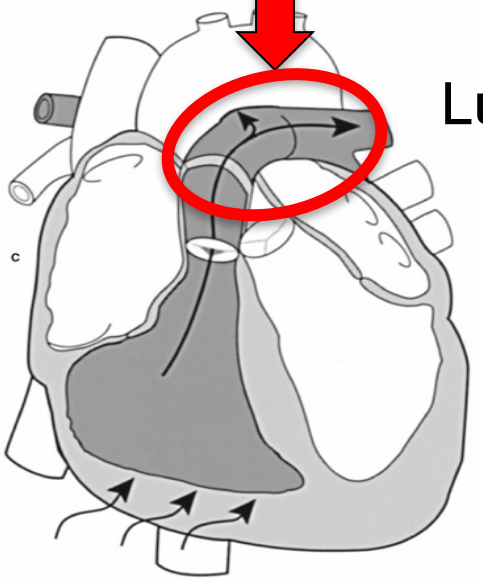


Figure 5-4 B, As blood continues to flow [illegible] the right atrium, the resulting pressure causes the tricuspid valve to open. Blood [illegible] able to enter the right ventricle.



Lung

Figure 5-4 C, During systole, the right ventricle contracts, squeezing its contents through the pulmonic valve and into the pulmonary circulation.

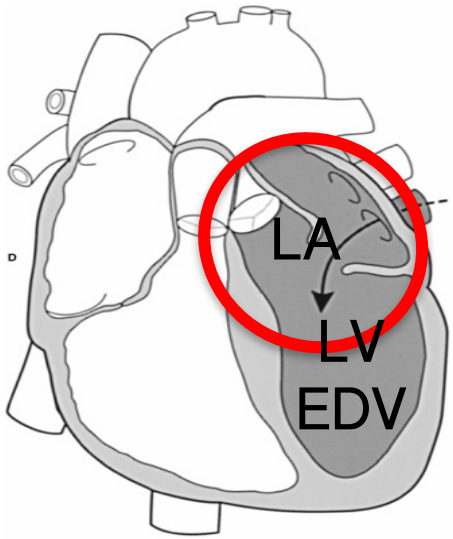
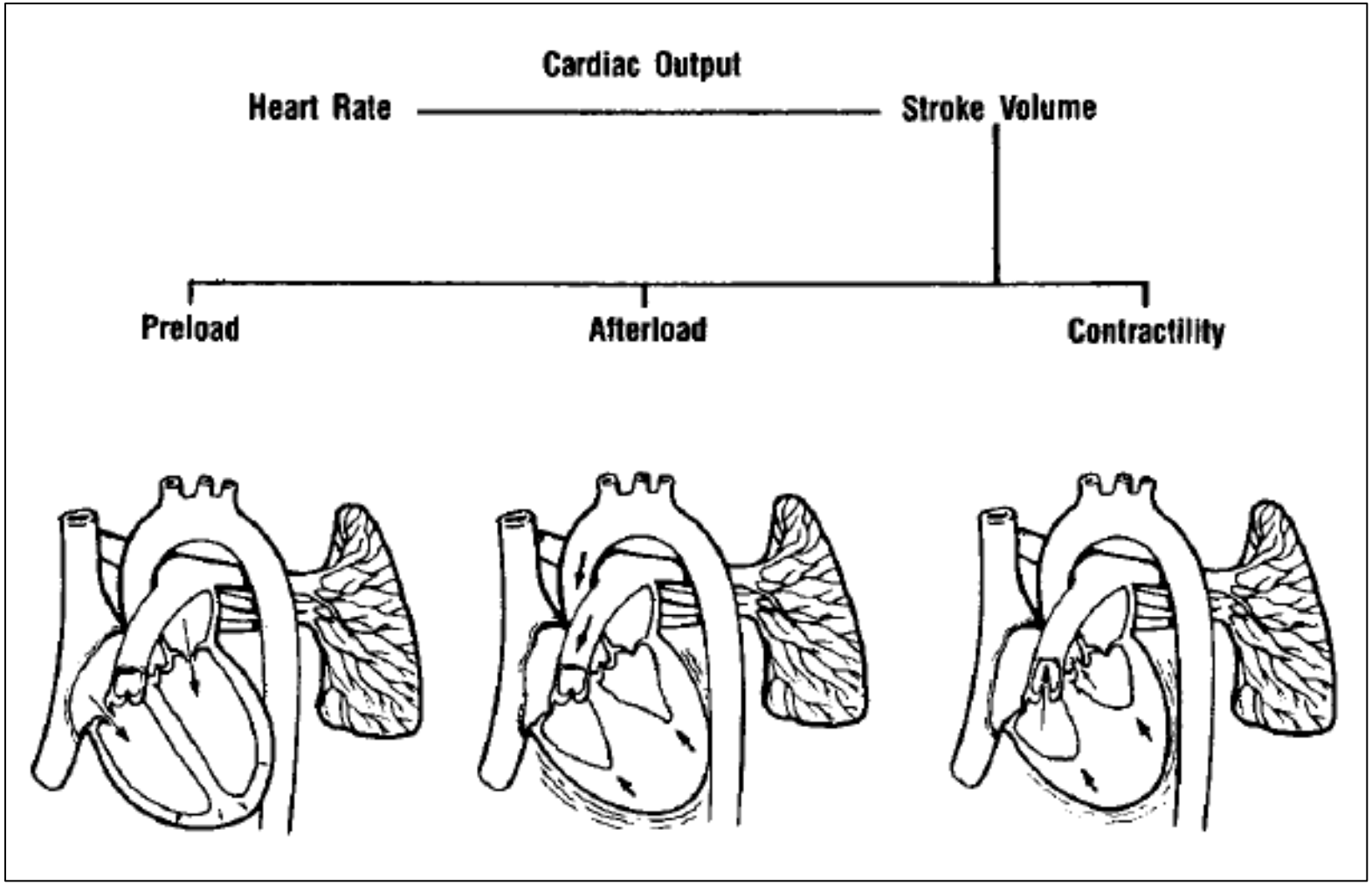


Figure 5-4 D, Oxygenated blood from the lungs now enters the left atrium. As atrial pressure builds, the mitral valve will be forced open so that blood is able to pass into the left ventricle.

Determinants Of Cardiac Output



Preload

1. Preload refers to the amount of myocardial fiber stretch at the end of diastole.
2. Preload also refers to the amount of volume in the ventricle at this phase.
3. It is very difficult to actually measure fiber length or volume at the bedside.
4. It has been clinically acceptable to measure the pressure required to fill the ventricles (LVFP) as a measure of left ventricular end diastolic volume (LVEDV) or fiber length

Preload

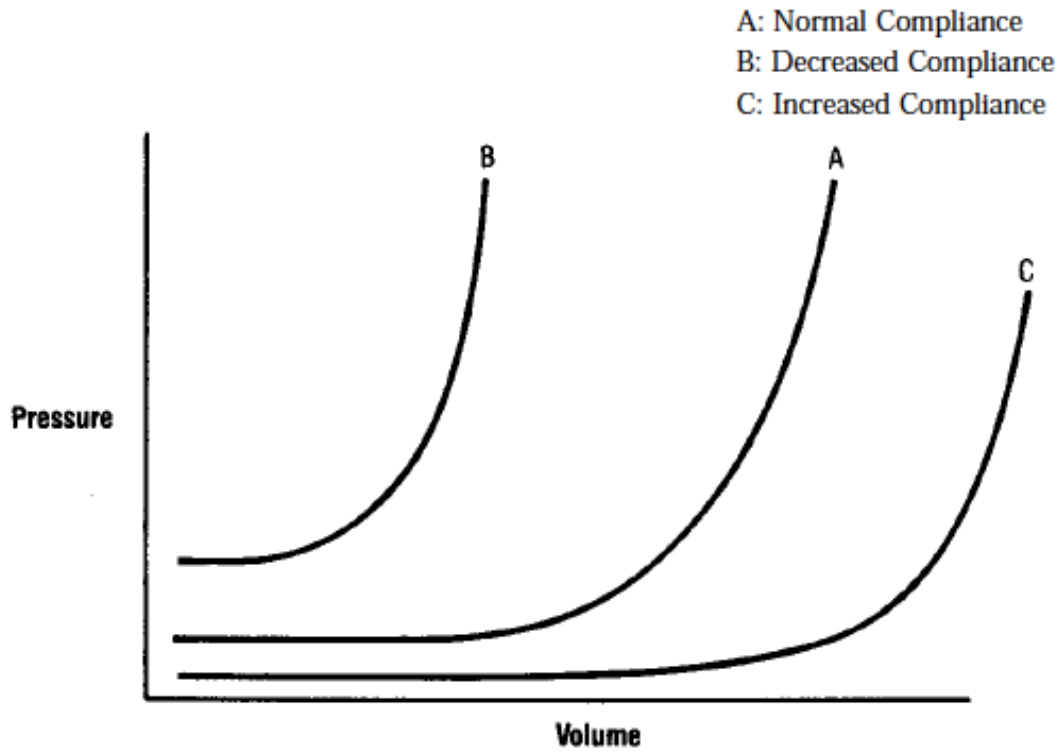


Figure 6
Compliance Curves

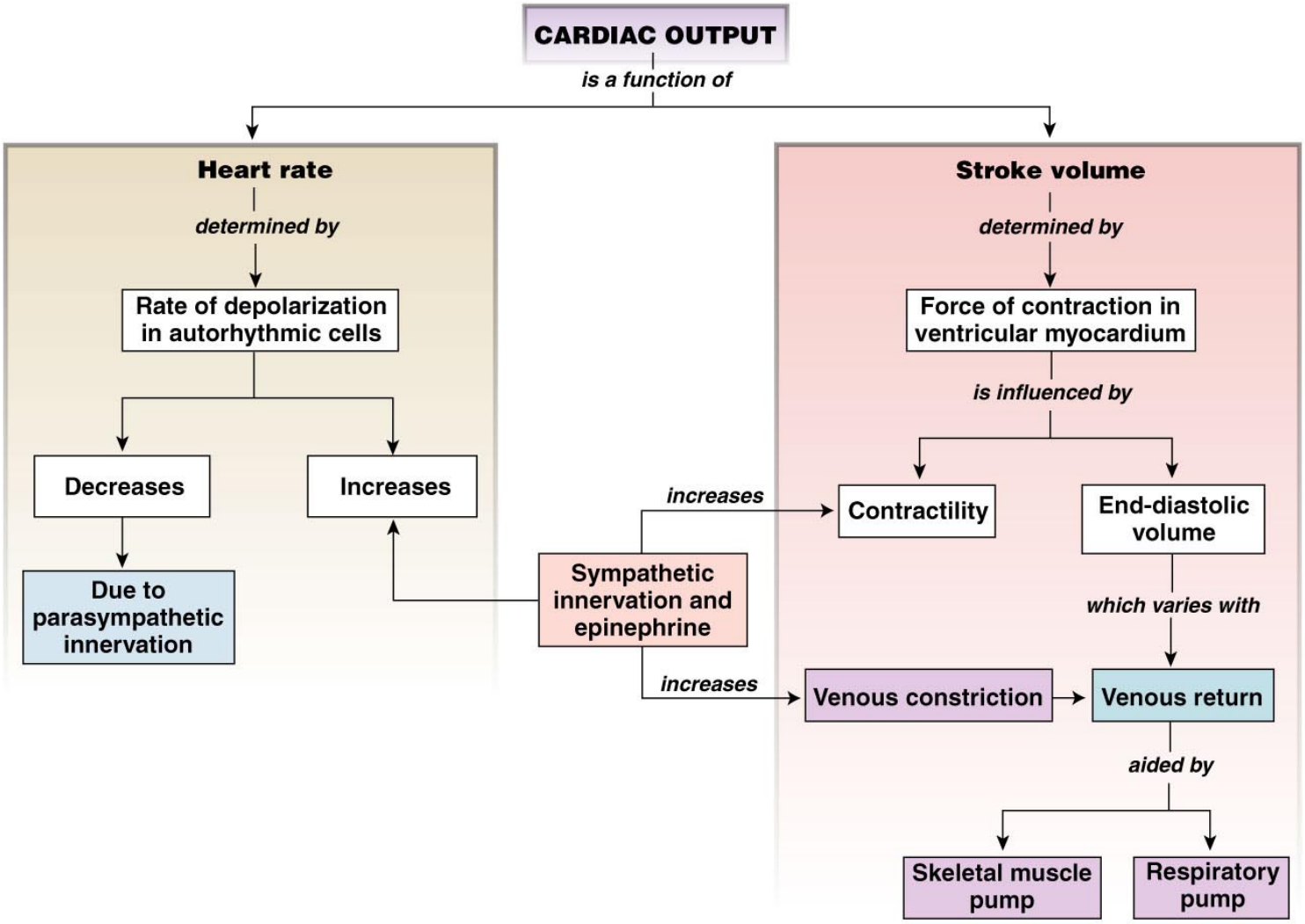
THE RELATIONSHIP BETWEEN ARTERIAL PRESSURE AND FLOW (CARDIAC OUTPUT)

Blood pressure is the “driving pressure” to perfuse organs and it is directly related to

cardiac output

MAP = CO x SVR

Summary: Altering Heart Performan



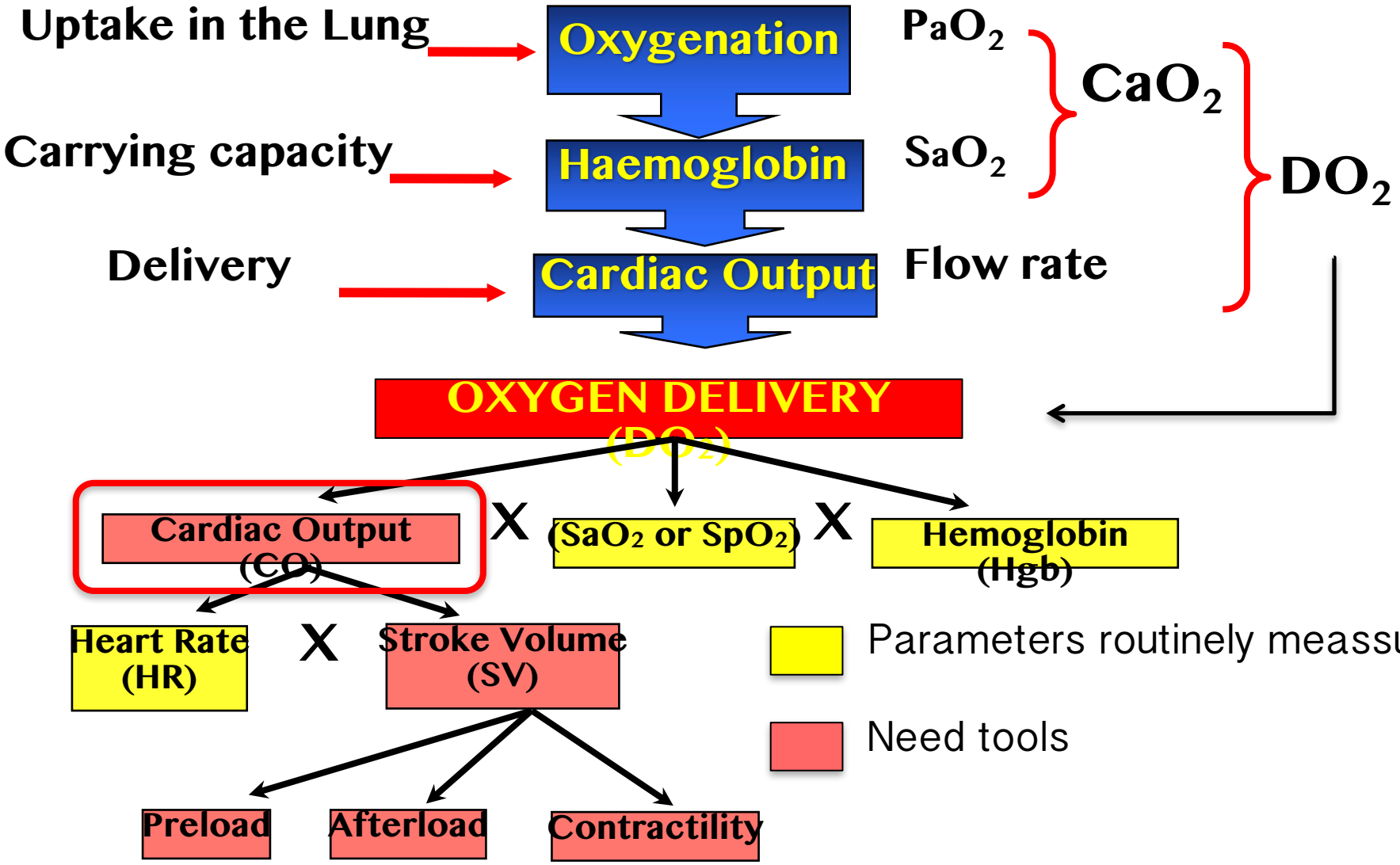
Blood Pressure = CO x SVR

"Normal BP" = Normal SVR x Normal C (e.g. Healthy person)

"Normal BP" = High SVR x Low CO (e.g. Hemorrhagic or cardiogenic shock)

"Normal BP" = Low SVR x High CO (e.g. Sepsis)

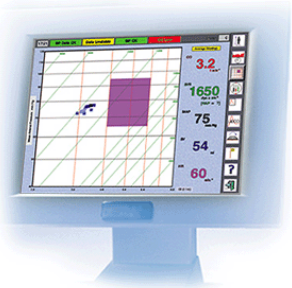
Why Cardiac Output Is Important?

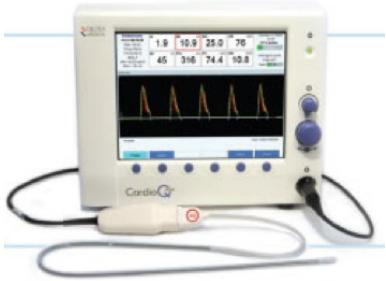

Uptake in the Lung
Oxygenation
PaO_2
Carrying capacity
Haemoglobin
SaO_2
CaO_2
Delivery
Cardiac Output
Flow rate
DO_2
OXYGEN DELIVERY (DO_2)
Cardiac Output (CO)
X
(SaO_2 or SpO_2)
X
Hemoglobin (Hgb)
Heart Rate (HR)
X
Stroke Volume (SV)
Parameters routinely meassure
Need tools
Preload
Afterload
Contractility

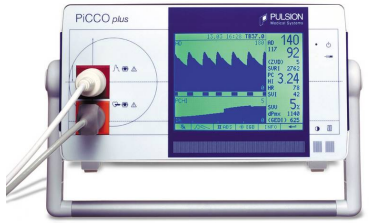
Methods Of Meassuring Cardiac Output

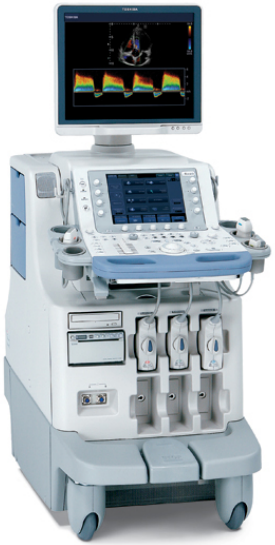
Cardiac Output Assessment Based On The Methods

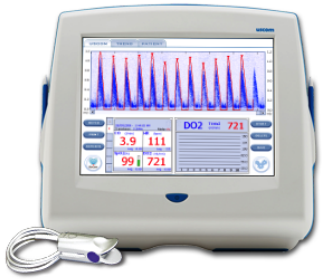
Method	System	Preload and Additional variable	Limitations
Transcardiac Thermodilution	PA catheter Catheter Ccombo	PAOP, PAP, SvO2 RVEF, RVEDV	Invasiveness, Training required
Transpulmonary Indicator Dilution (calibrated)	PiCCO LiDCO	GEDV, EVLW, SVV, PPV	Invasiveness, Need for dedicated catheter
Arterial-pressure waveform derived (not calibrated)	Vigileo MostCare	SVV, dP/dT, CCE, PPV, SVV	Need for optimal arterial signal
Doppler's principle	Esophageal CardiacQ Suprasternal USCOM	Ftc, peakV	Partial meassurement of CO Estimation of AoCSA
Fick's principle	NiCO	Shunt calculation	Intubated patients Less reliable in respiratory failure
Bioimpedance	Lifegard, TEBCO	None	Not applicable in cardiothoracic surg

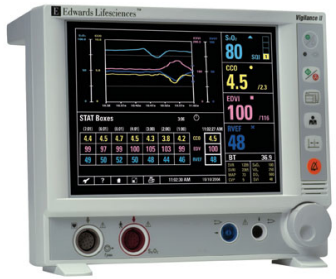


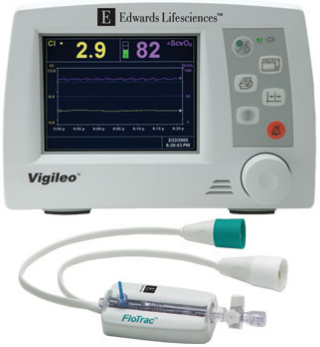


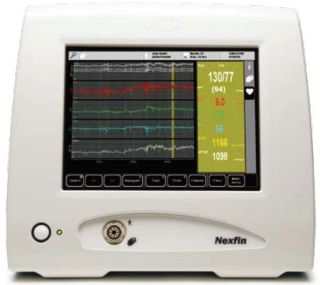

PiCCO plus
PULSION






Edwards Lifesciences


Edwards Lifesciences
Vigileo
FloTrac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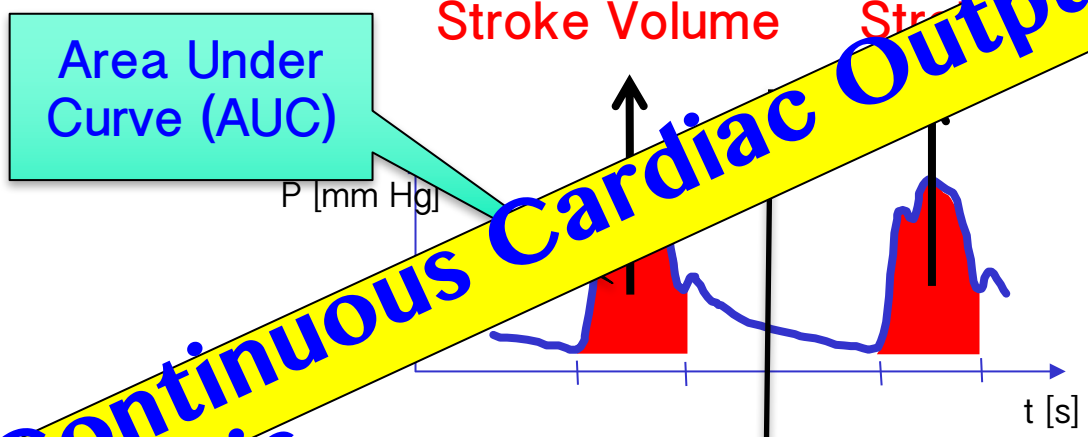
Arterial Pulse Contour Analysis as a basis for Cardiac Output measurement

1. Arterial pulse contour analysis provides continuous *beat-by-beat* parameters obtained from the shape of the arterial pressure wave.
2. The algorithm is capable of computing each stroke volume (SV)



Continuous Cardiac Output (CCO) analysis..

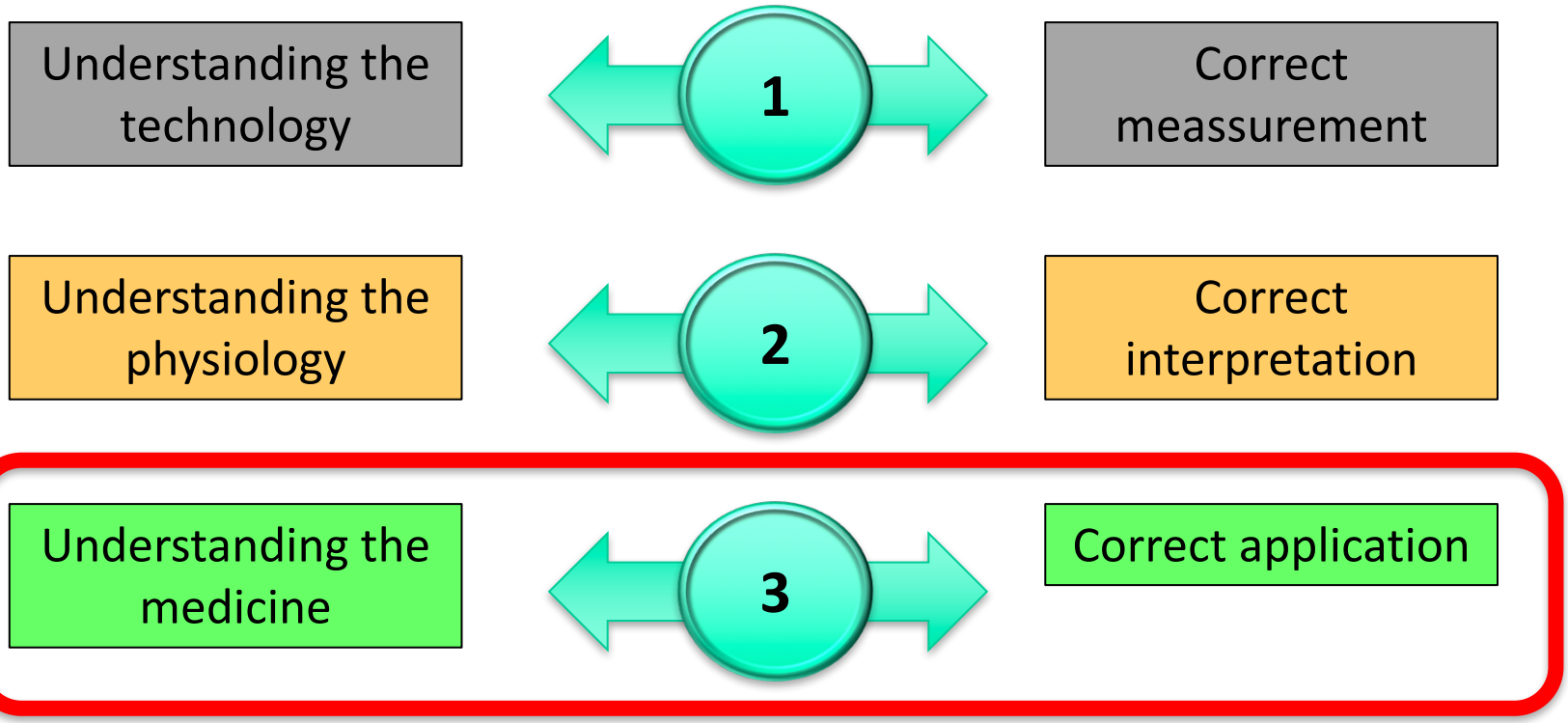
SV x HR = Cardiac Output

The Methods of Pulse Contour Technique

1. Requiring external calibration:
    1. LiDCO (Lithium indicator CO)
    2. PiCCO (Pulse indicator Continuous CO)
2. Without external calibration:
    1. Vigileo
    2. MostCare

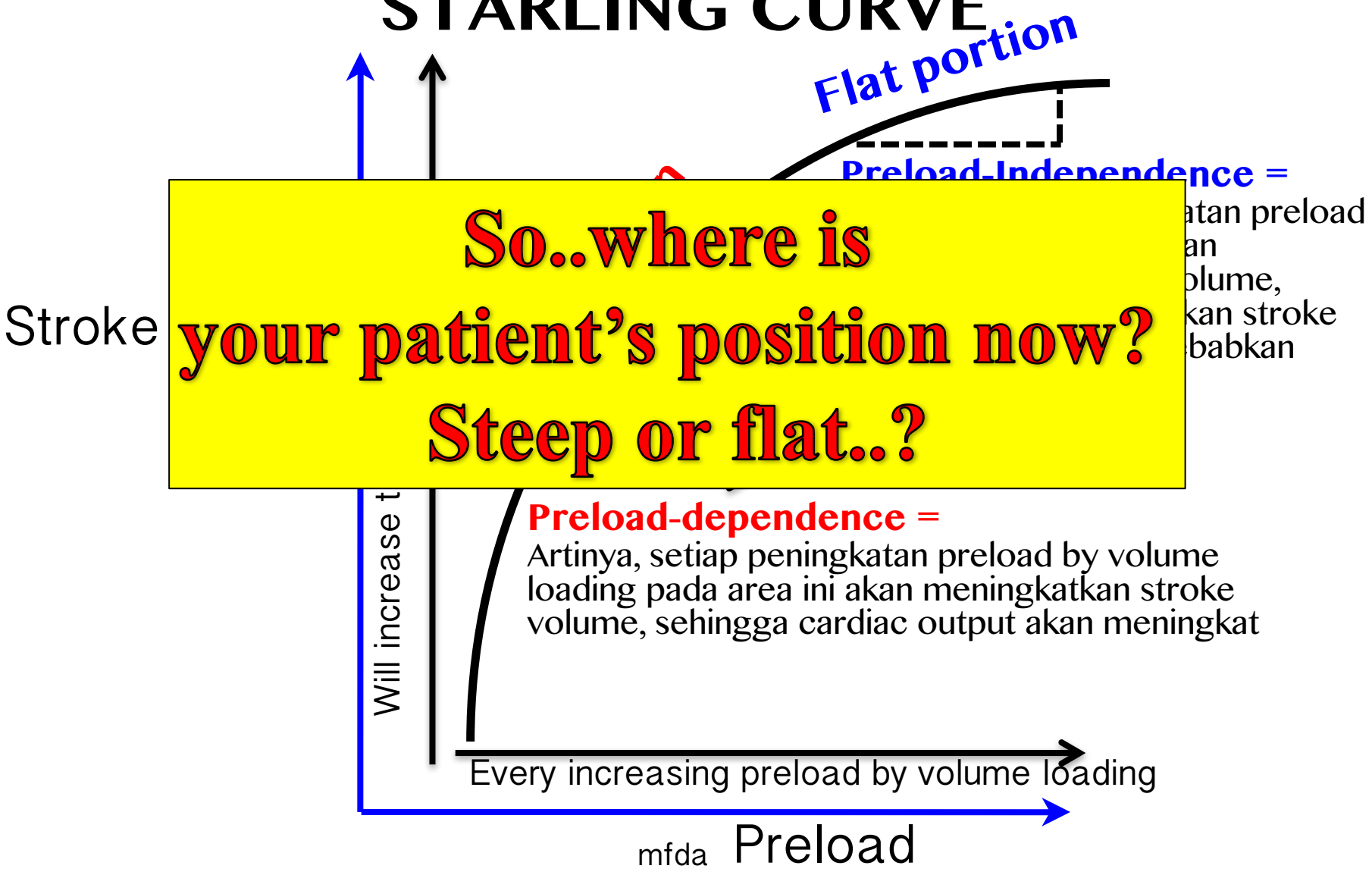
Fundamentals of Hemodynamic monitoring

The 3 magic push-button

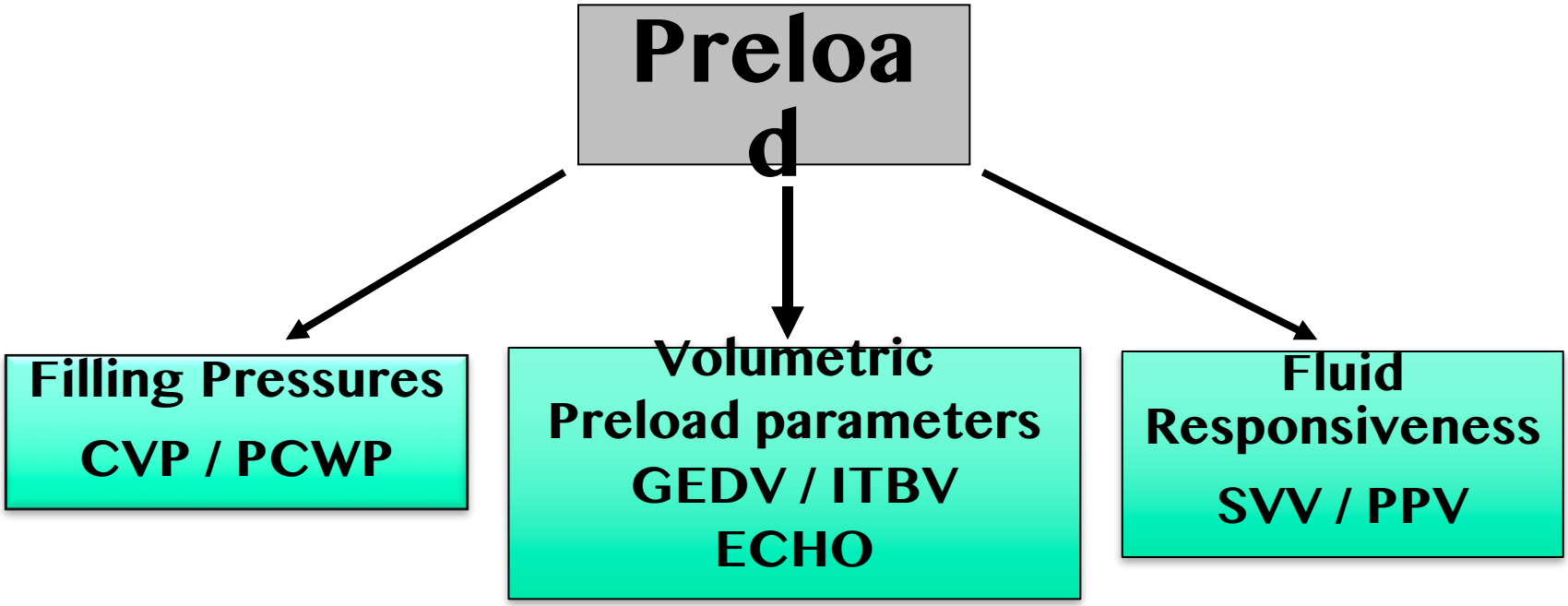


Preload monitoring

RELATIONSHIP BETWEEN STROKE VOLUME AND PRELOAD IN FRANK STARLING CURVE

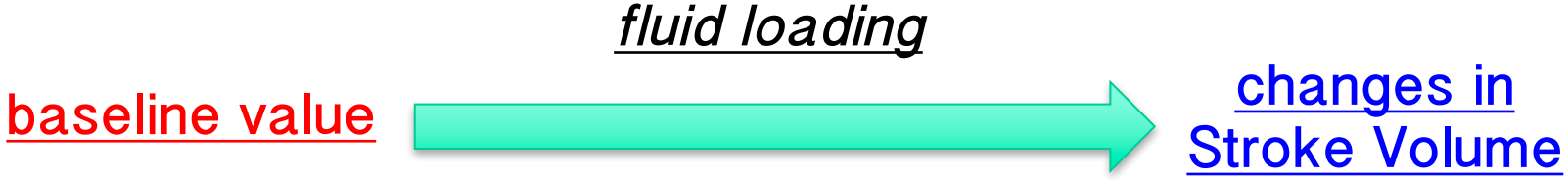


PRELOAD MONITORING

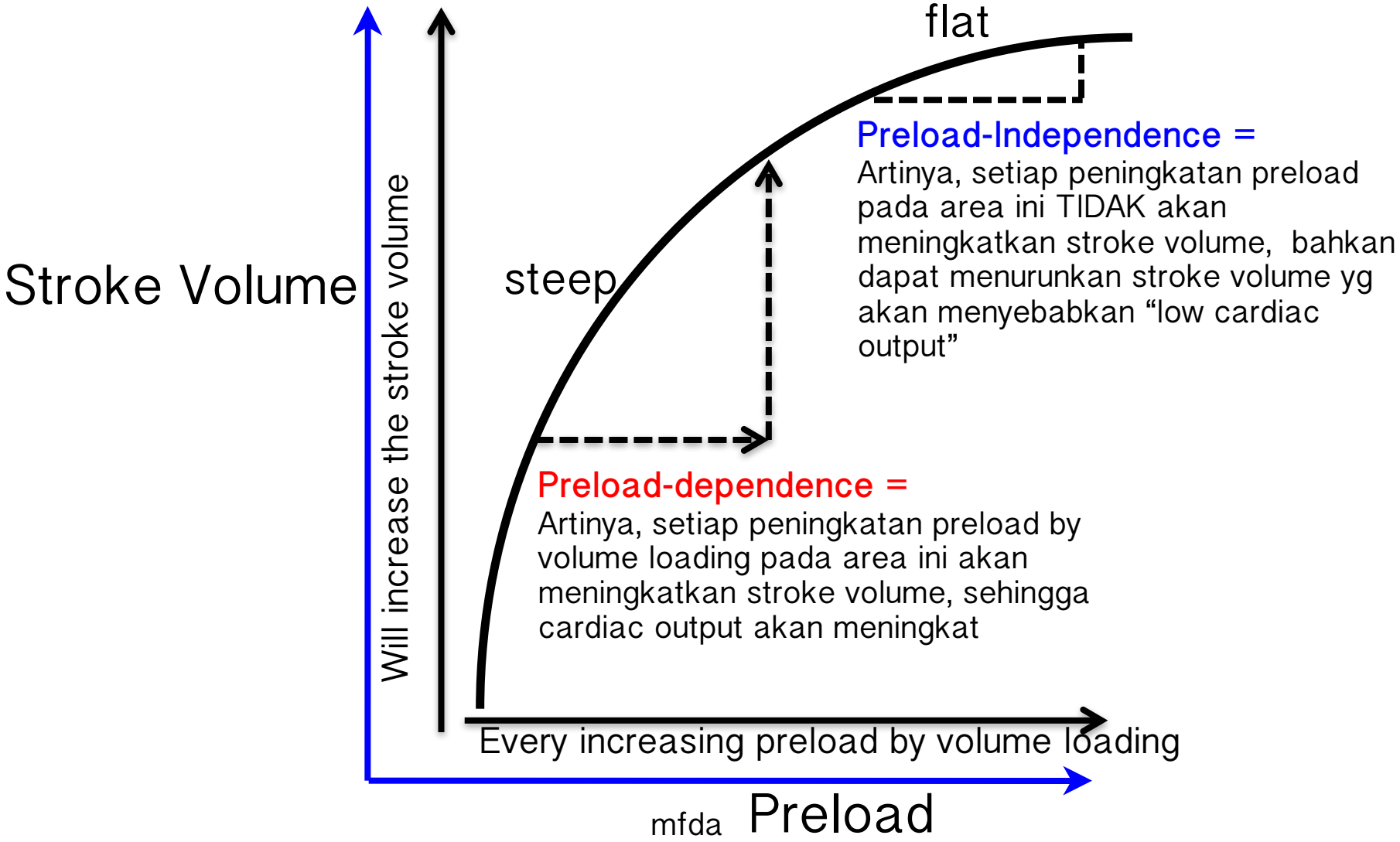


FLUID RESPONSIVENESS

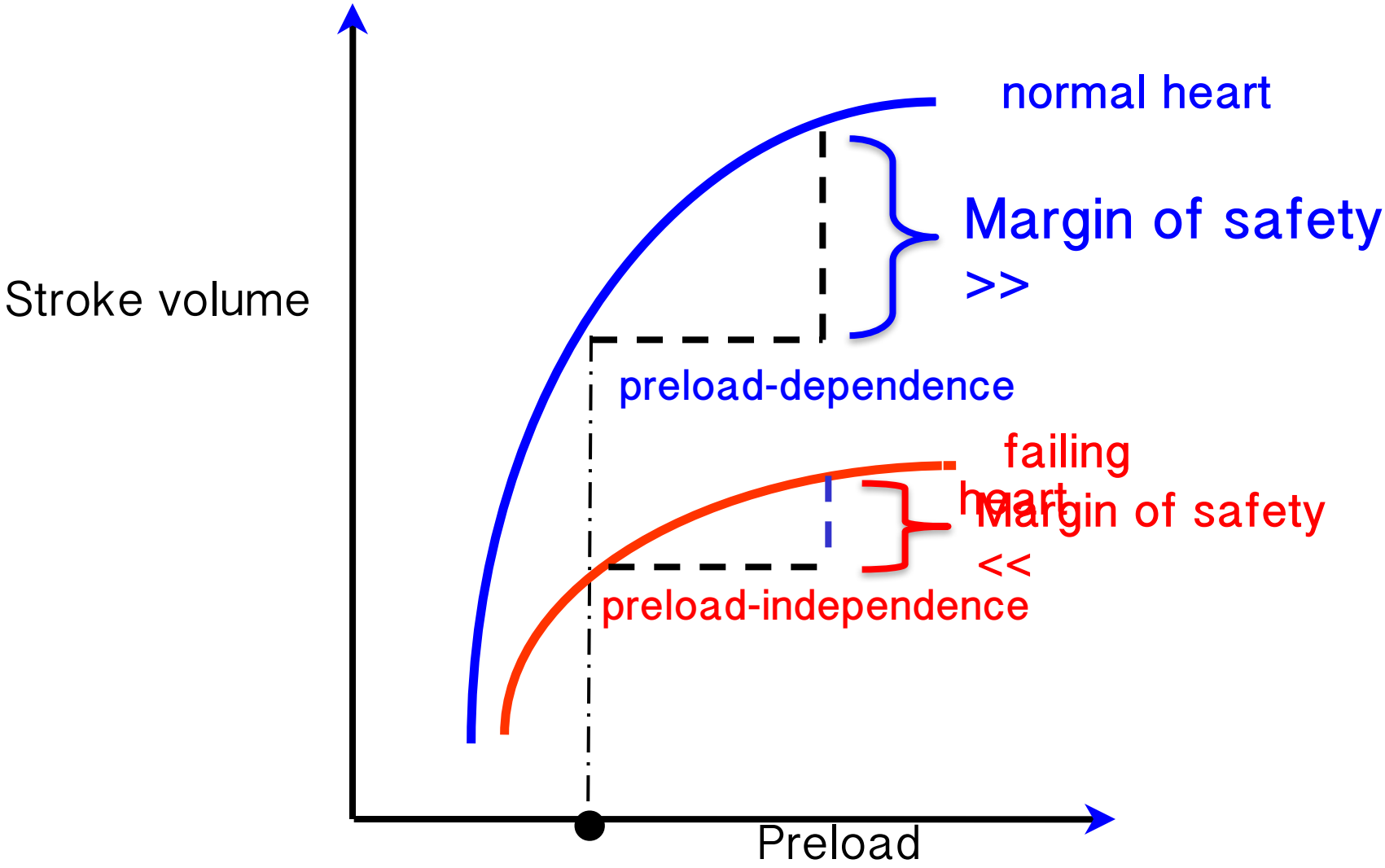
1. The basis of monitoring Fluid Responsiveness is Frank Starling Curve
2. A variable is a predictor of fluid responsiveness if there is a relationship between the baseline value of that variable and changes in Stroke Volume ***after fluid loading***



FRANK STARLING CURVE IN IN NORMAL HEART

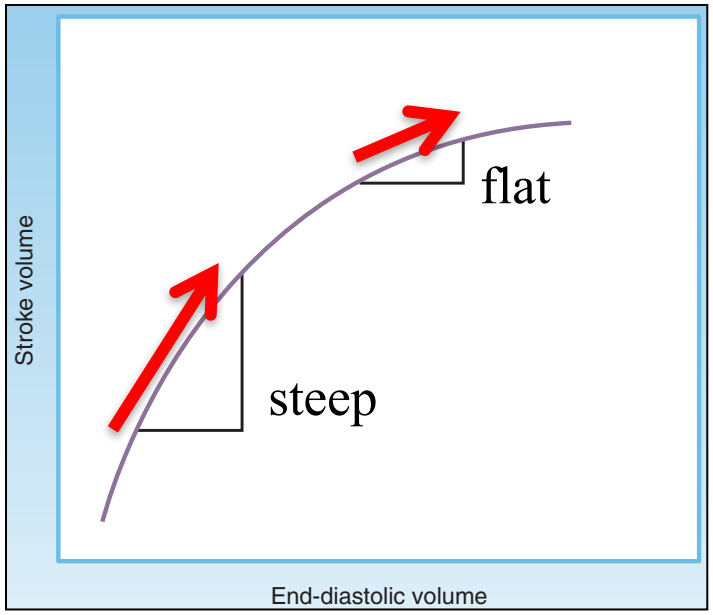


FRANK STARLING CURVE IN FAILING HEART



FLUID RESPONSIVENESS DURING RESUSCITATION

- During resuscitation, we always want to be on the **steep** portion of curve
  - Increasing end-diastolic volume means big increase in stroke volume
- We do not want to be on the **flat** portion of the curve
  - Increasing end-diastolic volume results in little or no increase in stroke volume



TECHNOLOGY AND STRATEGIES TO MONITOR PRELOAD USING FLUID RESPONSIVENESS

1. Stroke Volume Variation
2. Passive Leg Raising
3. Fluid Challenges

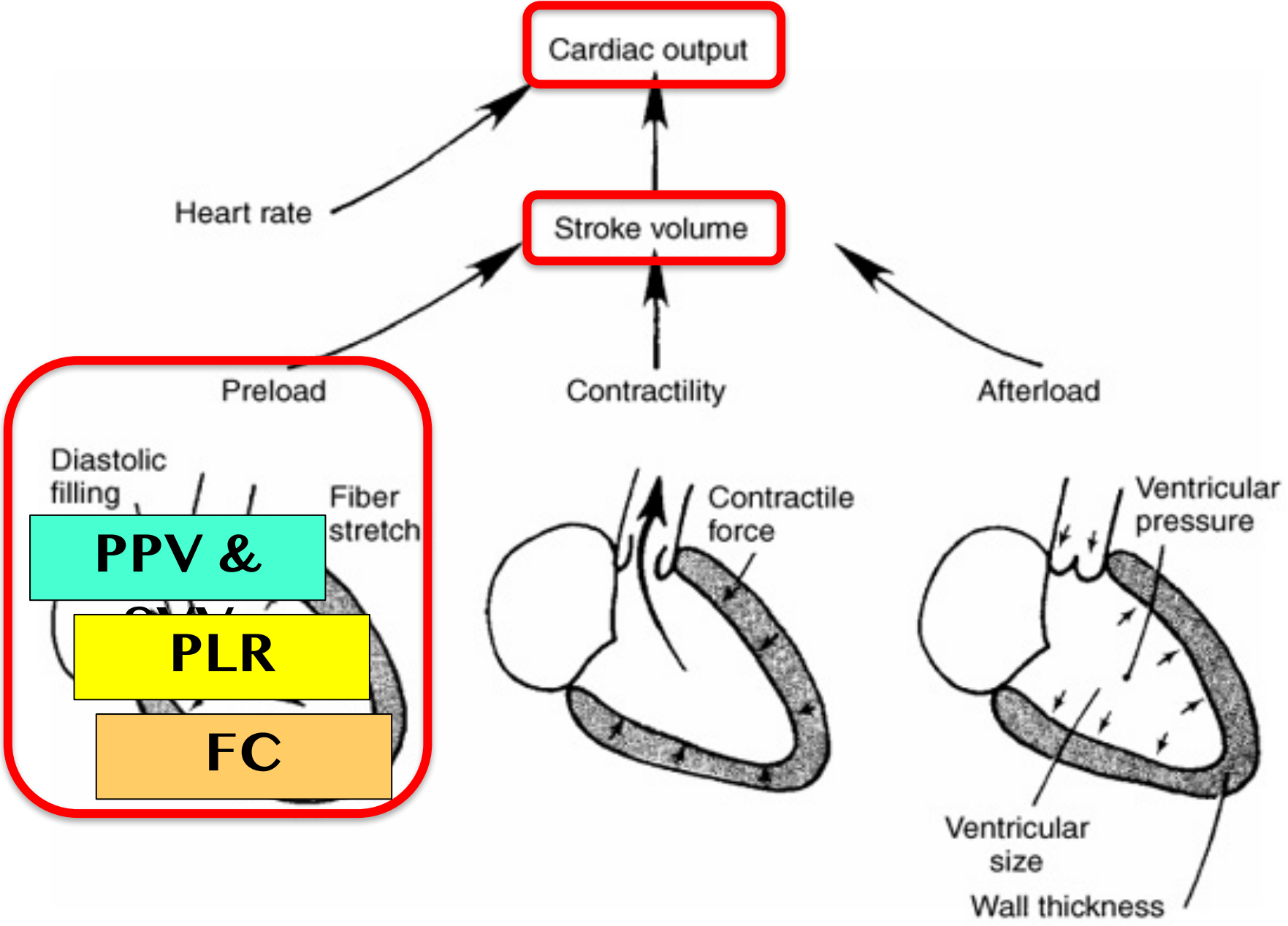


Figure 5-5 Factors affecting cardiac output. (From Price, S., & Wilson, L. [1986]. *Pathophysiology: Clinical concepts of disease processes* [3rd ed.] [p. 351]. New York: McGraw-Hill.)

1. Stroke Volume Variation (SVV), Pulse Pressure Variation (PPV)

Stroke Volume Variation (SVV)

Pulse Contour Analysis and Heart-lung Interaction are a basic concept for SVV measurement

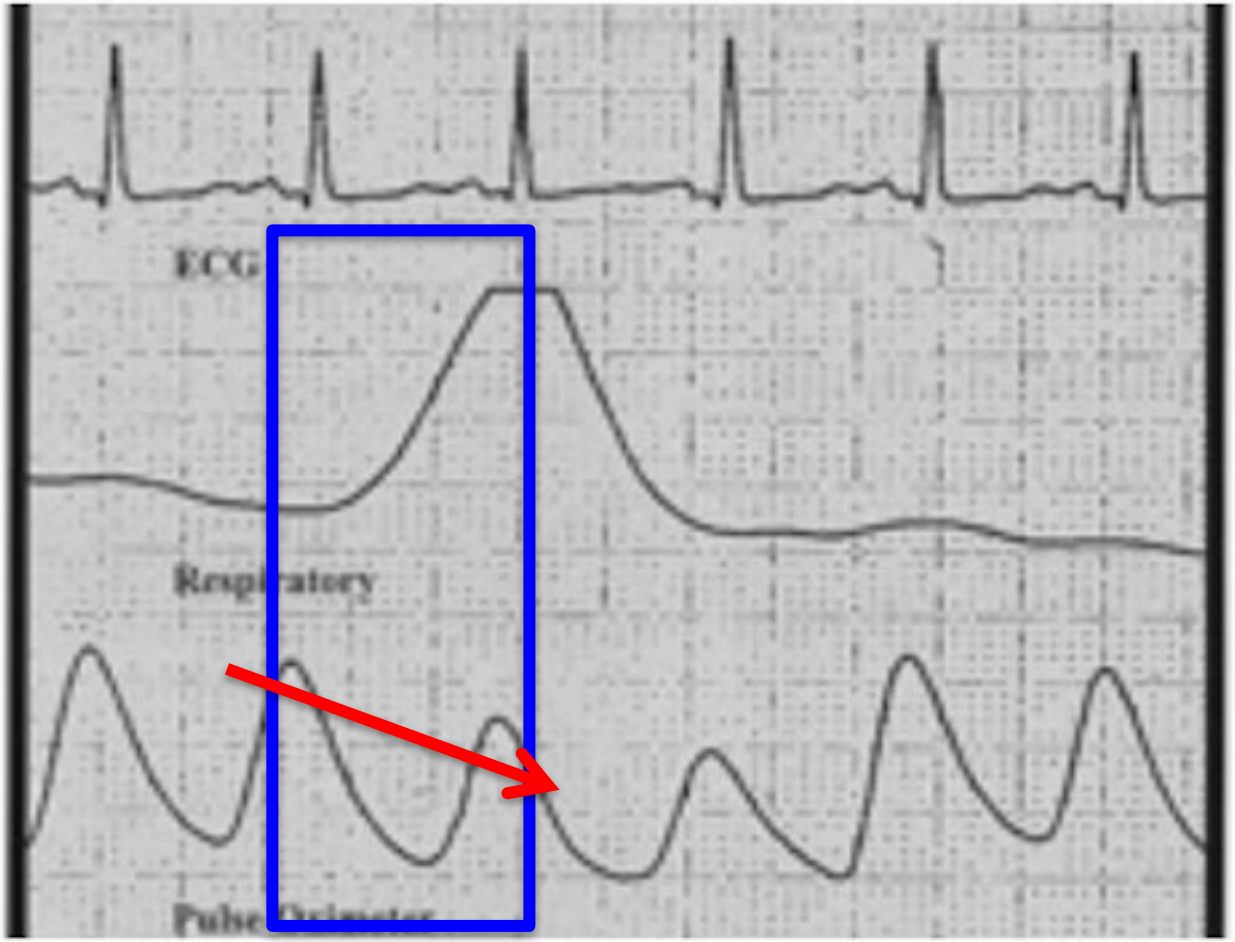
Heart-lung Interaction concept is the basis of monitoring Stroke Volume Variation

Pulsus Paradoxus

Reversed Pulsus Paradoxus

Pulsus Paradoxus

- Pada orang normal, pada waktu inspirasi akan terjadi penurunan tekanan sistolik sebesar 10 mmHg.
- Jika penurunan tekanan sistolik lebih dari 10 mmHg disebut sebagai pulsus paradoxus.
- Beberapa faktor2 fisiologi atau patologi dapat menyebabkan pulsus paradoxus selama inspirasi:
  - Pada kondisi tamponade jantung, tekanan intrapericardial yg tinggi akan menurunkan kapasitas pengisian jantung sehingga menurunkan fraksi ejeksi ventrikel kiri.
  - Selama inspirasi, akan terjadi peningkatan venous return ke jantung kanan, dan bersamaan dengan tingginya tekanan sekeliling jantung waktu inspirasi, maka pengisian jantung kanan akan meningkat juga, sehingga akan menggeser septum interventrikular ke ventrikel kiri, mengakibatkan berkurangnya pengisian ventrikel kiri, sehingga fraksi ejeksi LV menurun waktu inspirasi.



PULSUS PARADOXICUS

NORMAL < 10 mmHg

PALPABLE > 15 mmHg

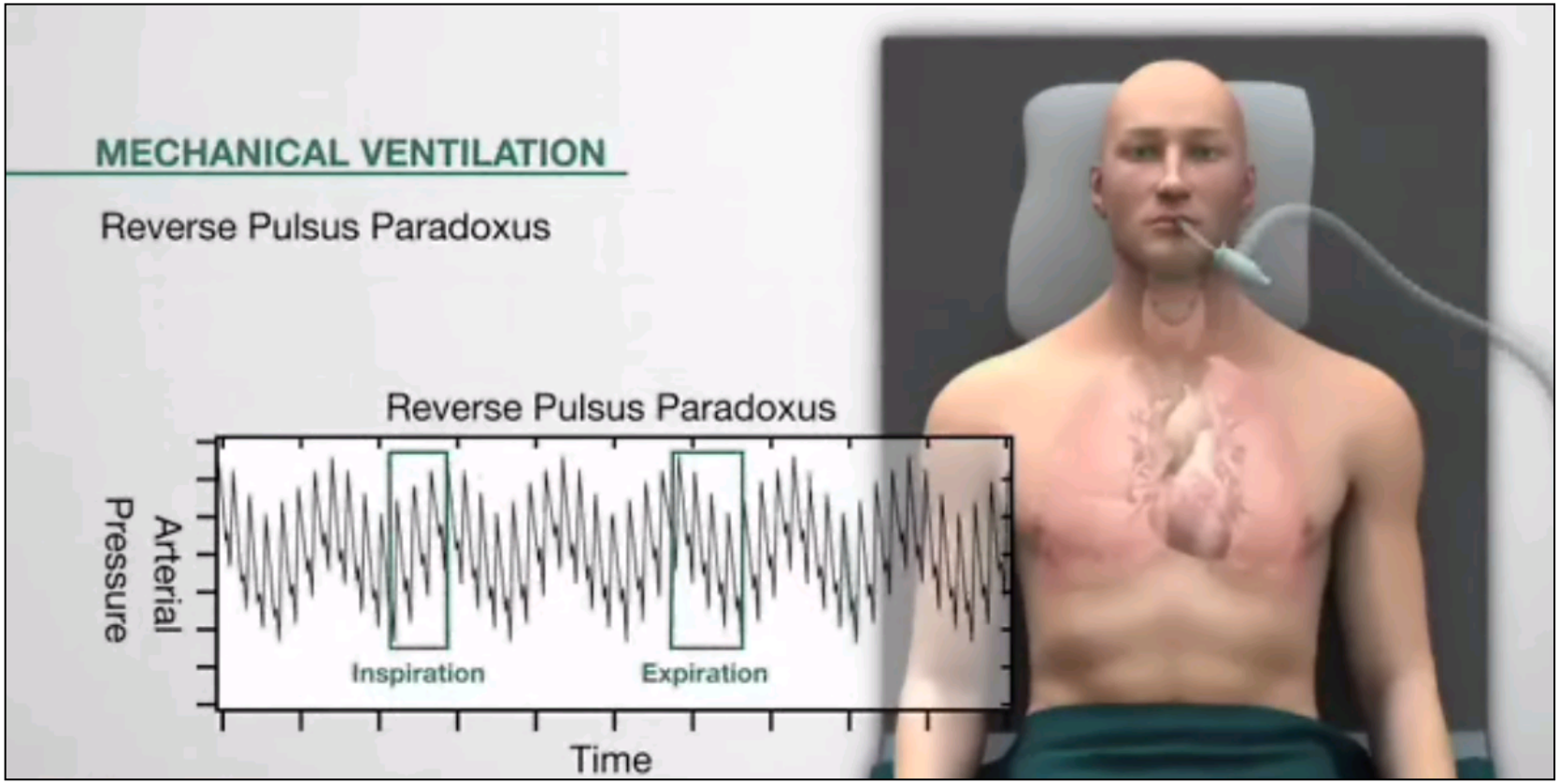
Reversed Pulsus Paradoxus

- Pada reversed pulsus paradoxus terjadi proses yg berlawanan, dimana pada waktu inspirasi terjadi kenaikan tekanan sitolik yang lebih besar dari 15mmHg.
- Hal ini terjadi akibat tekanan positif pada ventilasi mekanik akan meningkatkan tekanan intra toraks sehingga meningkatkan curah jantung ventrikel kiri.
- Ventilasi mekanik (VM) bekerja berbeda thd jantung kiri dan kanan, thd jantung kiri VM akan memompa darah lbh banyak ke ventrikel kiri dari vena pulmonalis, sehingga meningkatkan stroke volume LV dan sistolik pressure. Terhadap jantung kanan, VM akan menekan IVC dan menurunkan aliran darah ke ventikel kanan, sehingga tidak menimbulkan pendorongan septum ke kiri, sehingga lebih banyak darah mengisi LV

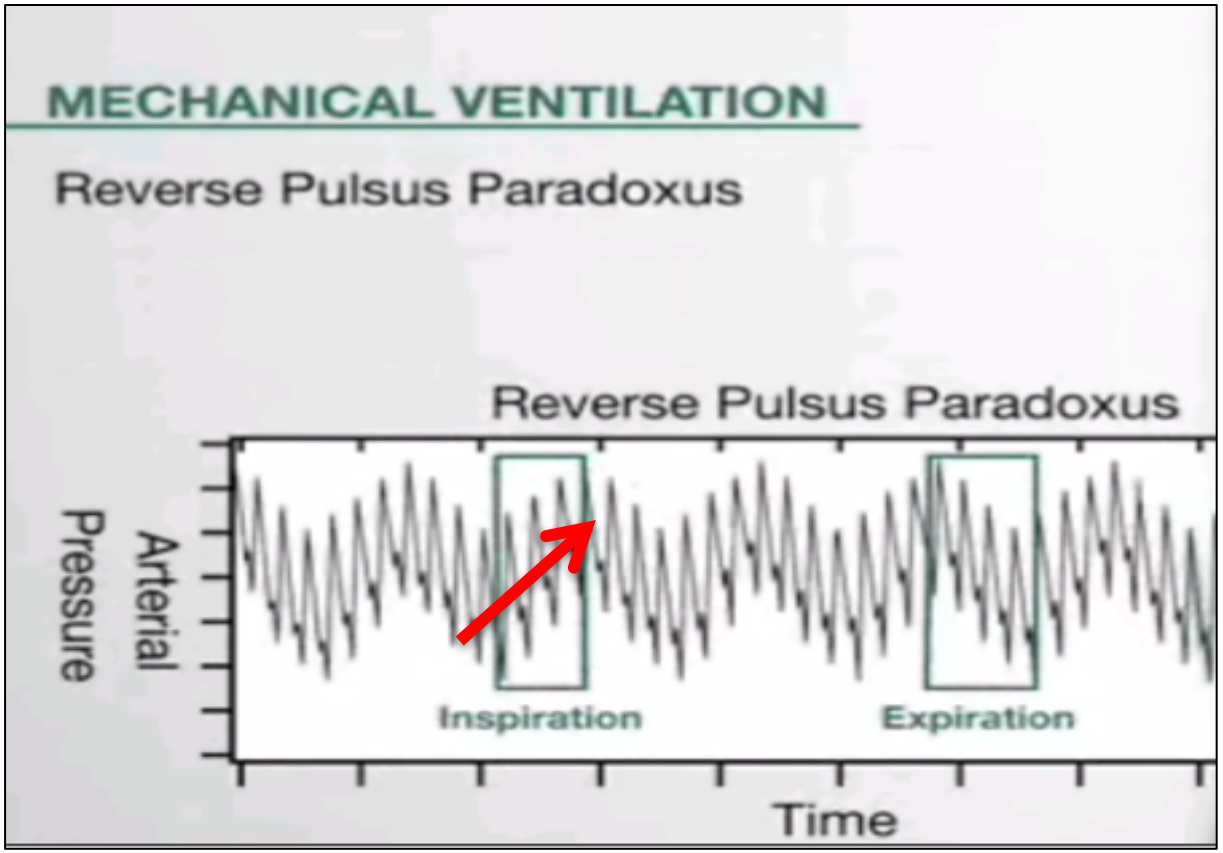
Reversed Pulsus Paradoxus

- Reverse pulsus paradoxus dapat terjadi pada kondisi2 dibawah ini:
  - Pasien gagal jantung kiri yang menggunakan ventilasi mekanik.
  - Idiopathic hypertrophic subaortic stenosis
  - Isorhythmic ventricular rhythms
  - Hypertrophic cardiomyopathy
  - Pada pasien hipovolemia yang menggunakan ventilasi mekanik

Reversed Pulsus Paradoxus



Reversed Pulsus Paradoxus

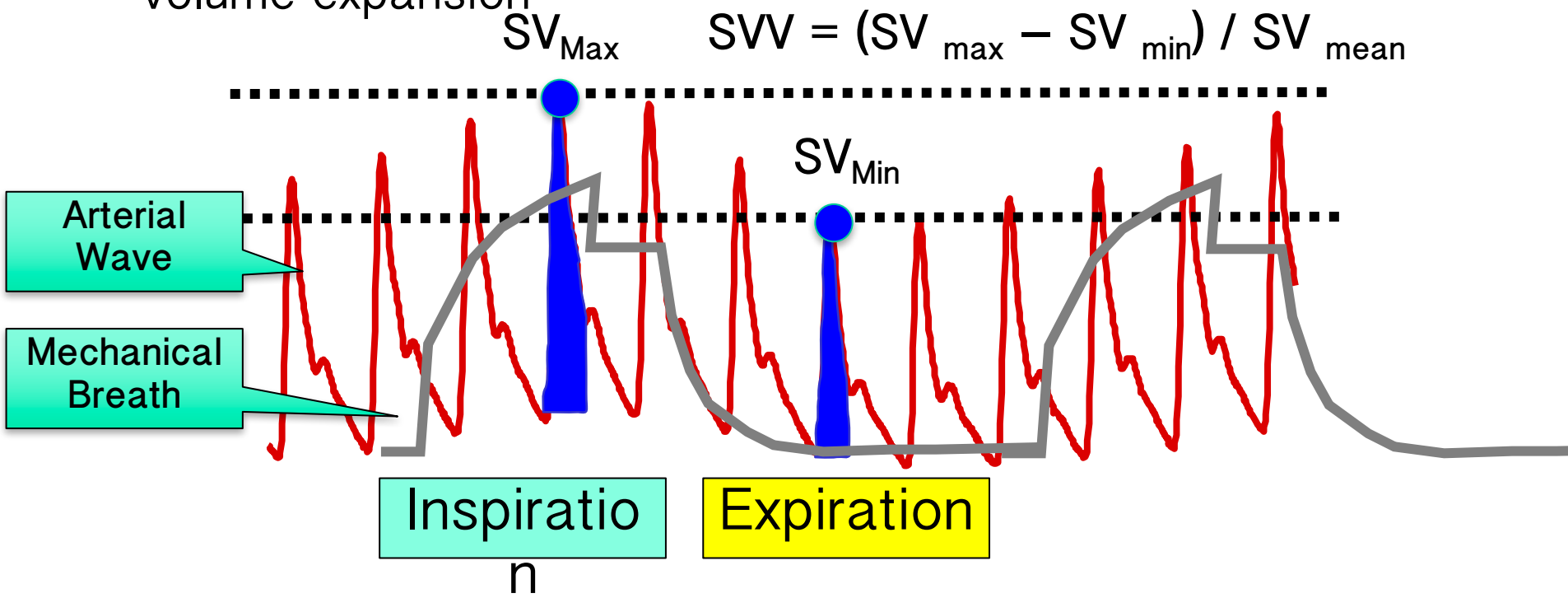


Tekanan sistolik (arterial wave pressure) pada pasien hipovolemia yang menggunakan ventilasi mekanik akan meningkat > 15 mmHg saat inspiratory breath

Stroke Volume Variation (SVV)

In mechanically ventilated patients without arrhythmia,

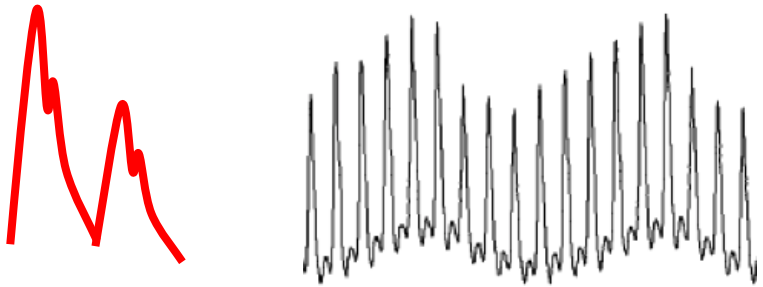
1. SVV reflects the sensitivity of the heart to the cyclic changes in cardiac preload induced by mechanical ventilation
2. SVV can predict whether stroke volume will increase with volume expansion



How to interpret the Stroke Volume Variation

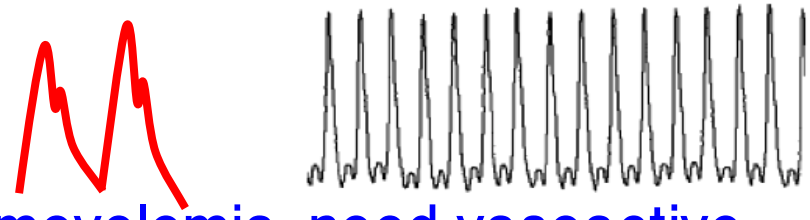
Interpretation of Stroke Volume Variation

Large SVV



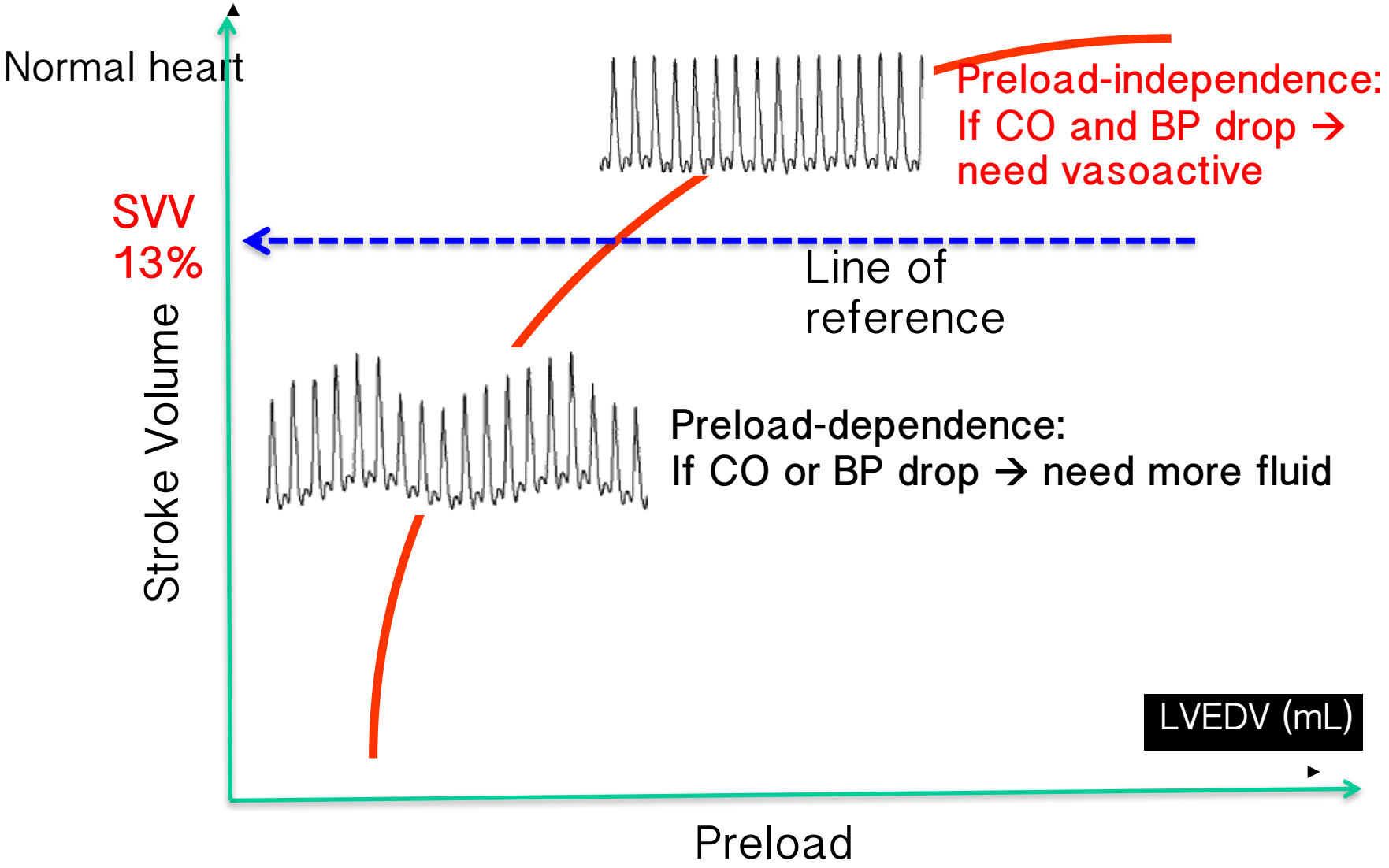
Hypovolemia, need volume loading

Low SVV

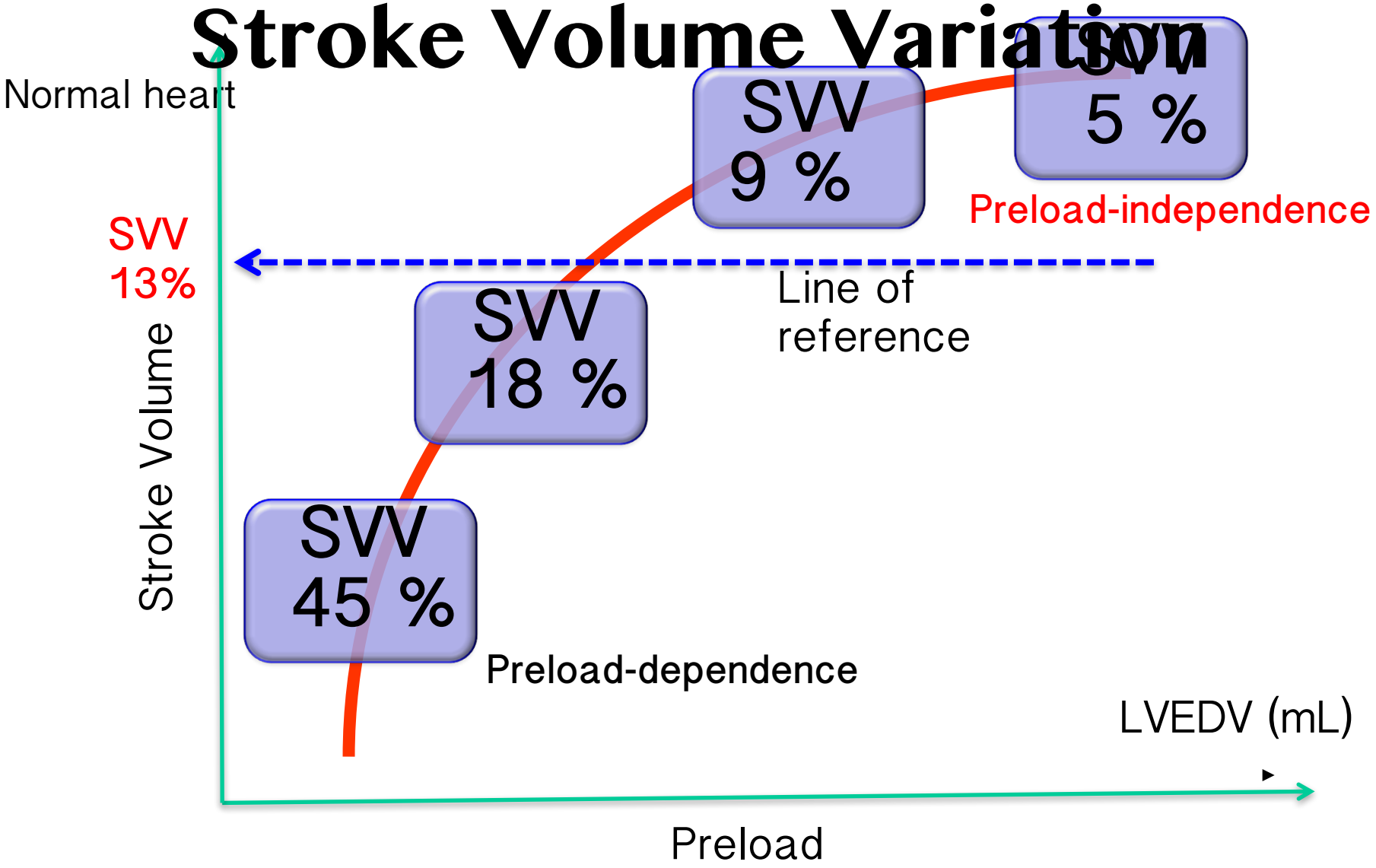


Normovolemia, need vasoactive

Interpretation of Stroke Volume Variation



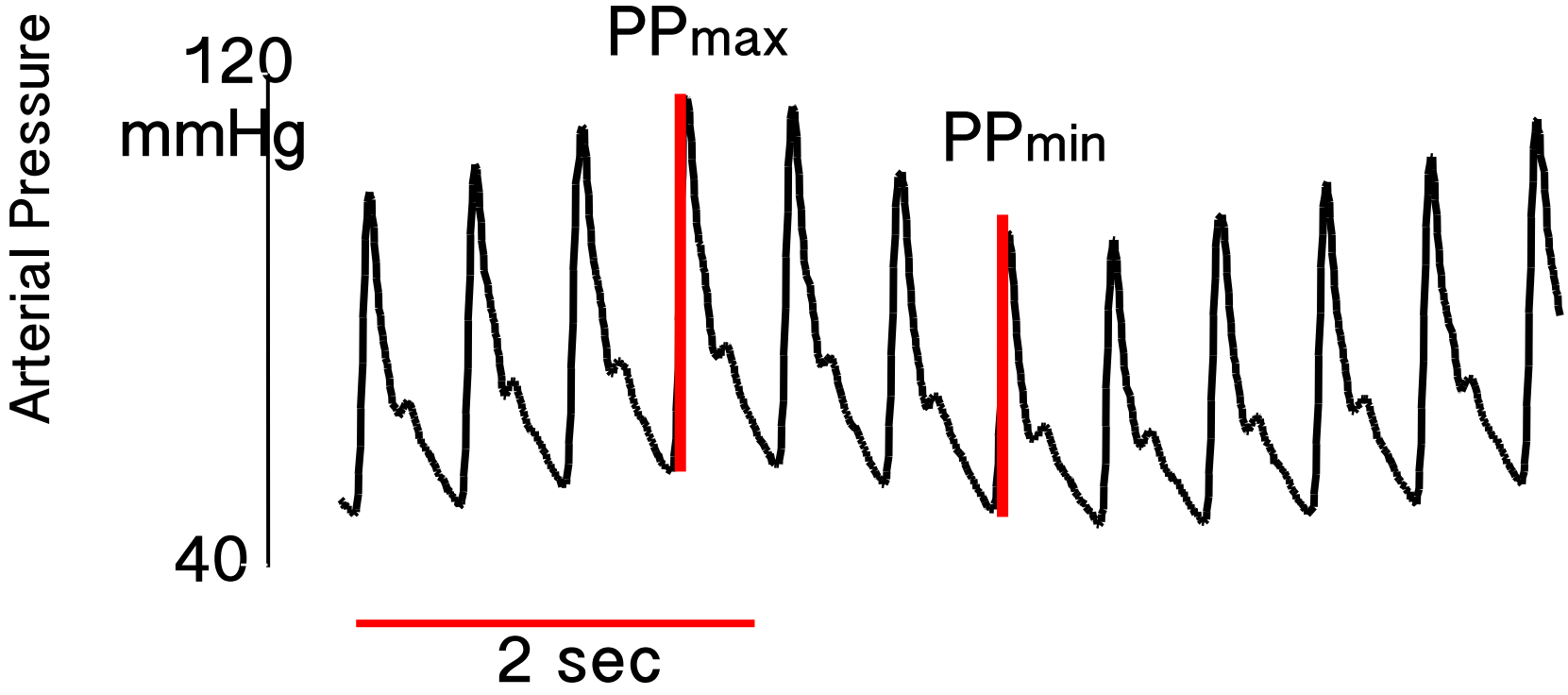
The Value of Interpretation of Stroke Volume Variation



Pulse Presure Variation (PPV)

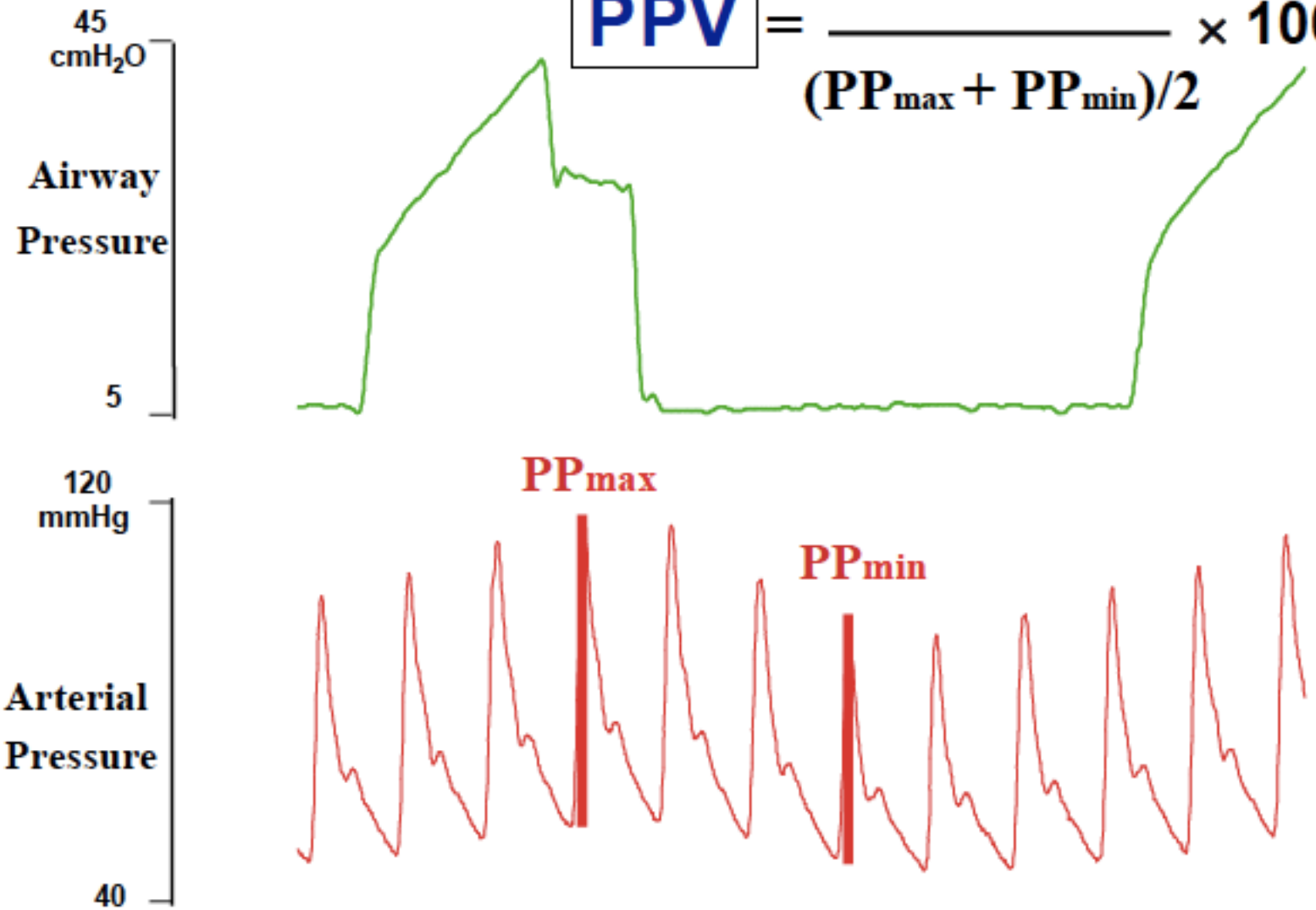
Pulse Pressure Variation

$$\Delta PP = \frac{PPmax - PPmin}{(PPmax + PPmin)/2}$$



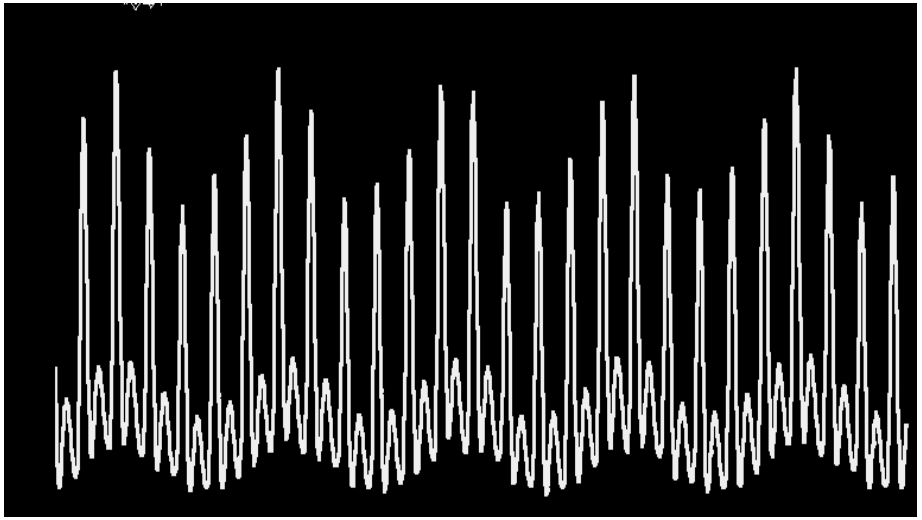
Michard F, et al. *Am J Respir Crit Care Med.* 2000;162(1):134-138.

$$PPV = \frac{PP_{max} - PP_{min}}{(PP_{max} + PP_{min})/2} \times 100$$

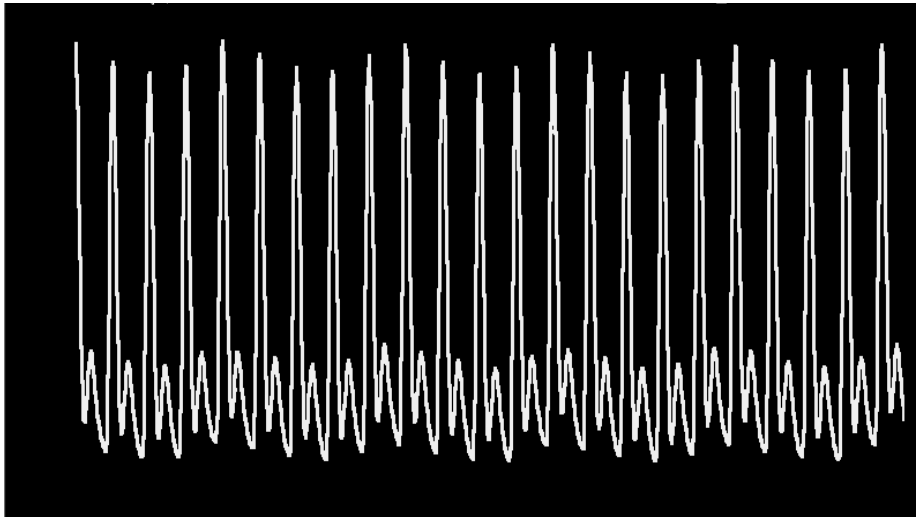


Pulse Pressure Variation

Pulse Pressure Variation



PPV = 32%



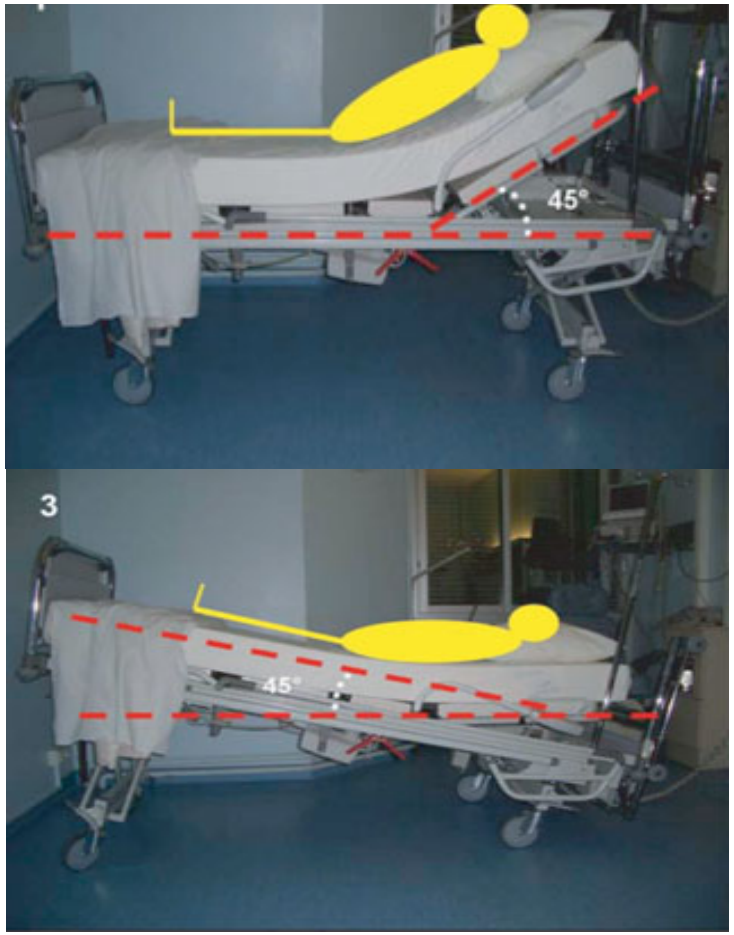
PPV = 5%

2. Passive Leg Raising

- Using Ultrasound
- Using Semi-invasive CO (flow-directed)

Passive Leg Raising

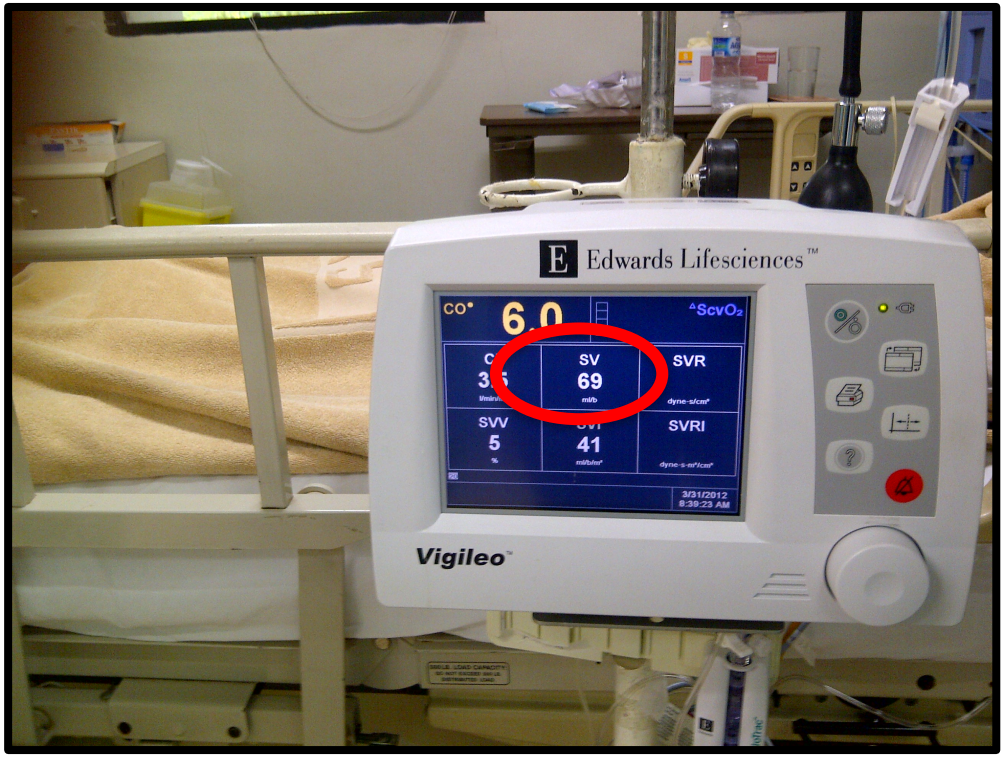
- This is the only dynamic method shown for spontaneously breathing patients
- There is 300mL of blood in a patient's lower extremities and it is used as a bolus
- This technique avoids patients receiving excess fluid boluses which may be harmful in the long-term



mfda

Passive Leg Raising using CCO (Vigileo)

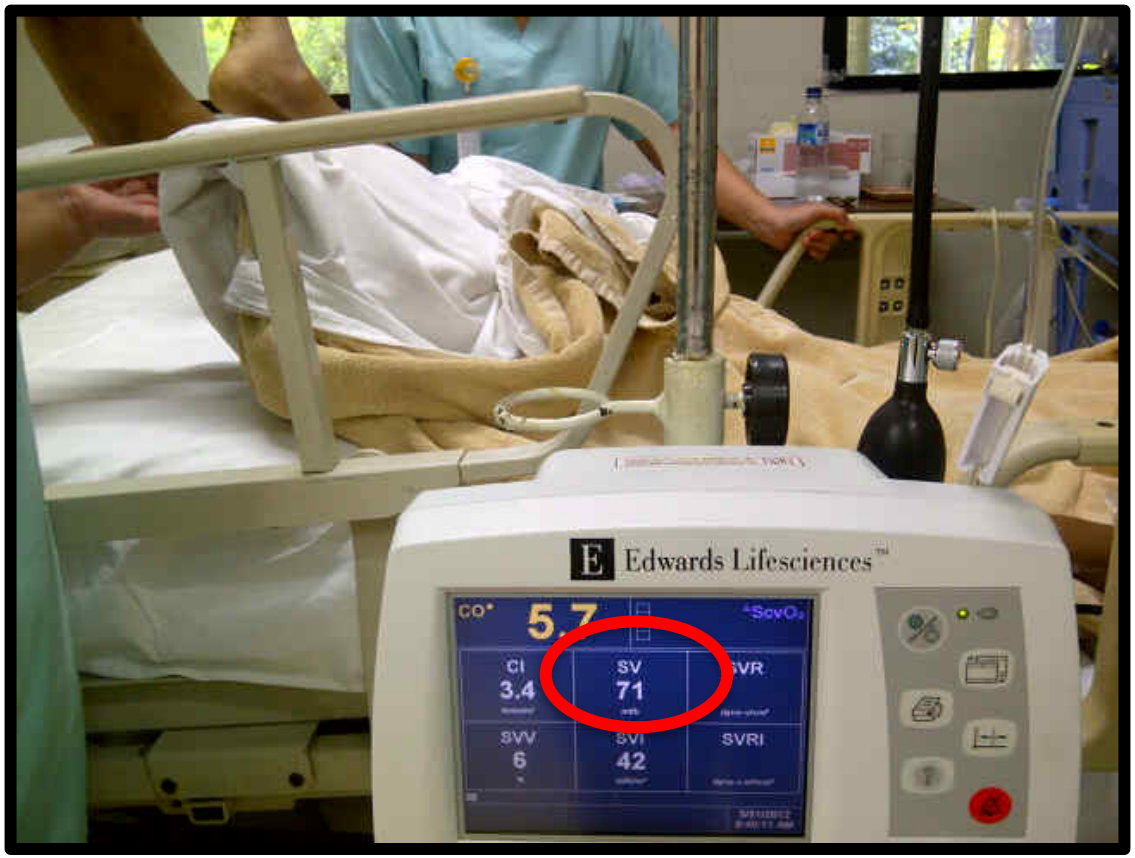
- PLR dilakukan untuk menilai kecukupan cairan pada pasien2 nafas spontan, Teknik: ekstremitas bawah di elevasi 40 derajat, jika SV naik 10% dari nilai awal, berarti pasien masih butuh cairan, jika tidak naik, atau naik tapi <10% artinya ps tidak perlu cairan



mfda

Passive Leg Raising using CCO (Vigileo)

- Setelah 30 detik elevasi dinilai perubahan pada SV dan CO



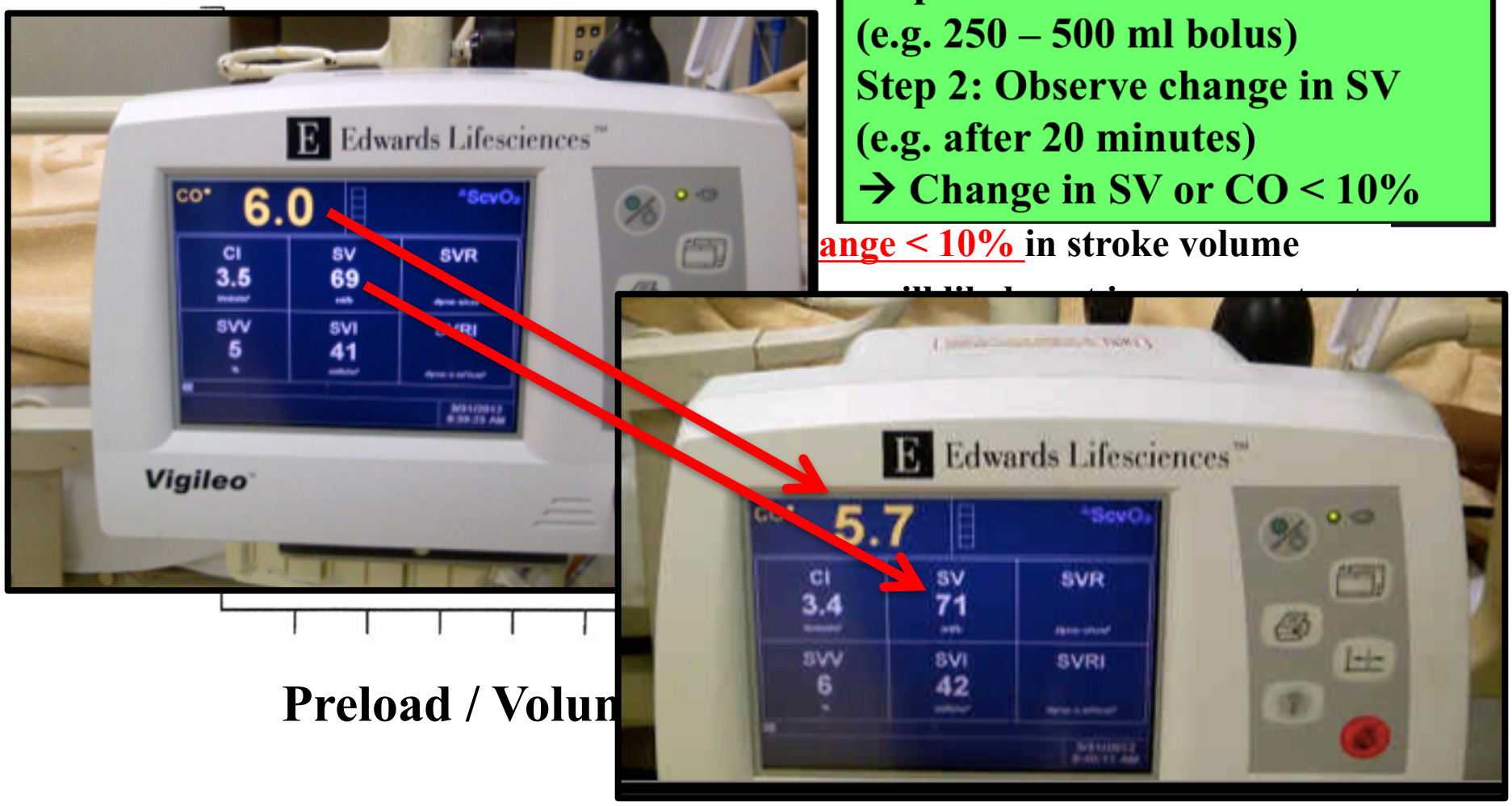
3. Fluid Challenges

- Using Flow-directed CO
- Using Central Venous Pressure

FLUID CHALLENGES USING FLOW-DIRECTED CO

Step 1: Give volume
(e.g. 250 – 500 ml bolus)
Step 2: Observe change in SV
(e.g. after 20 minutes)
→ Change in SV or CO < 10%

ange < 10% in stroke volume



Preload / Volun

FLUID CHALLENGES USING dIVC ULTRASOUND

- IVC variation in a spontaneously breathing patient is a static measurement and only estimates central venous pressure
- Several authors believe that CVP by ultrasound is a **not** a useful measurement of cardiac preload

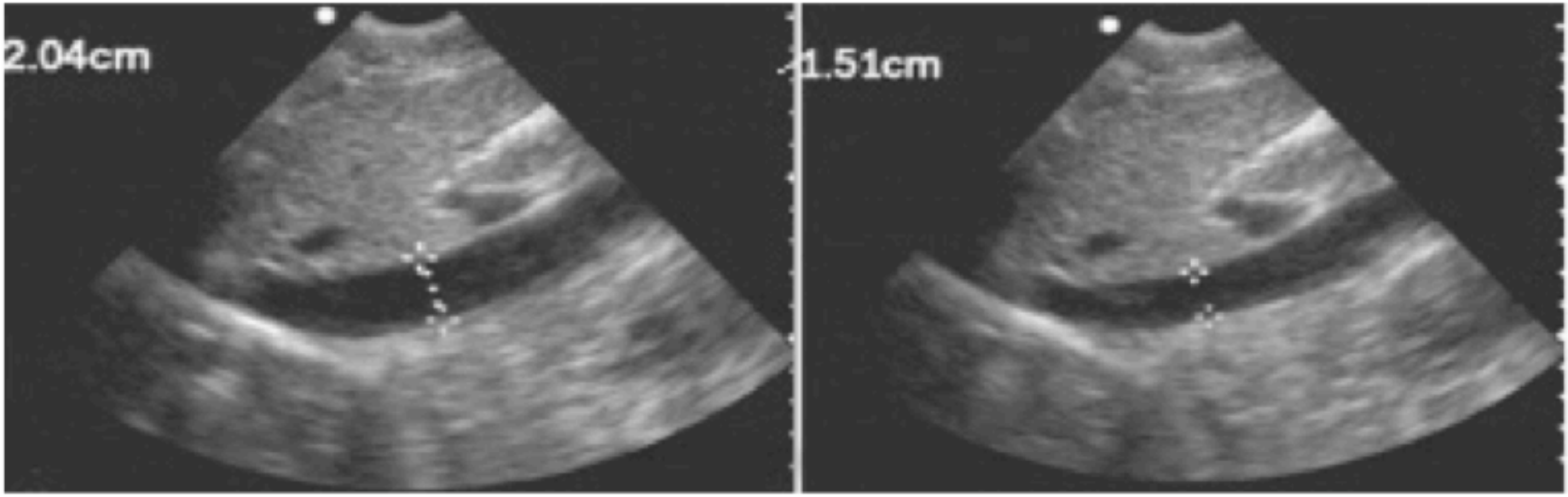
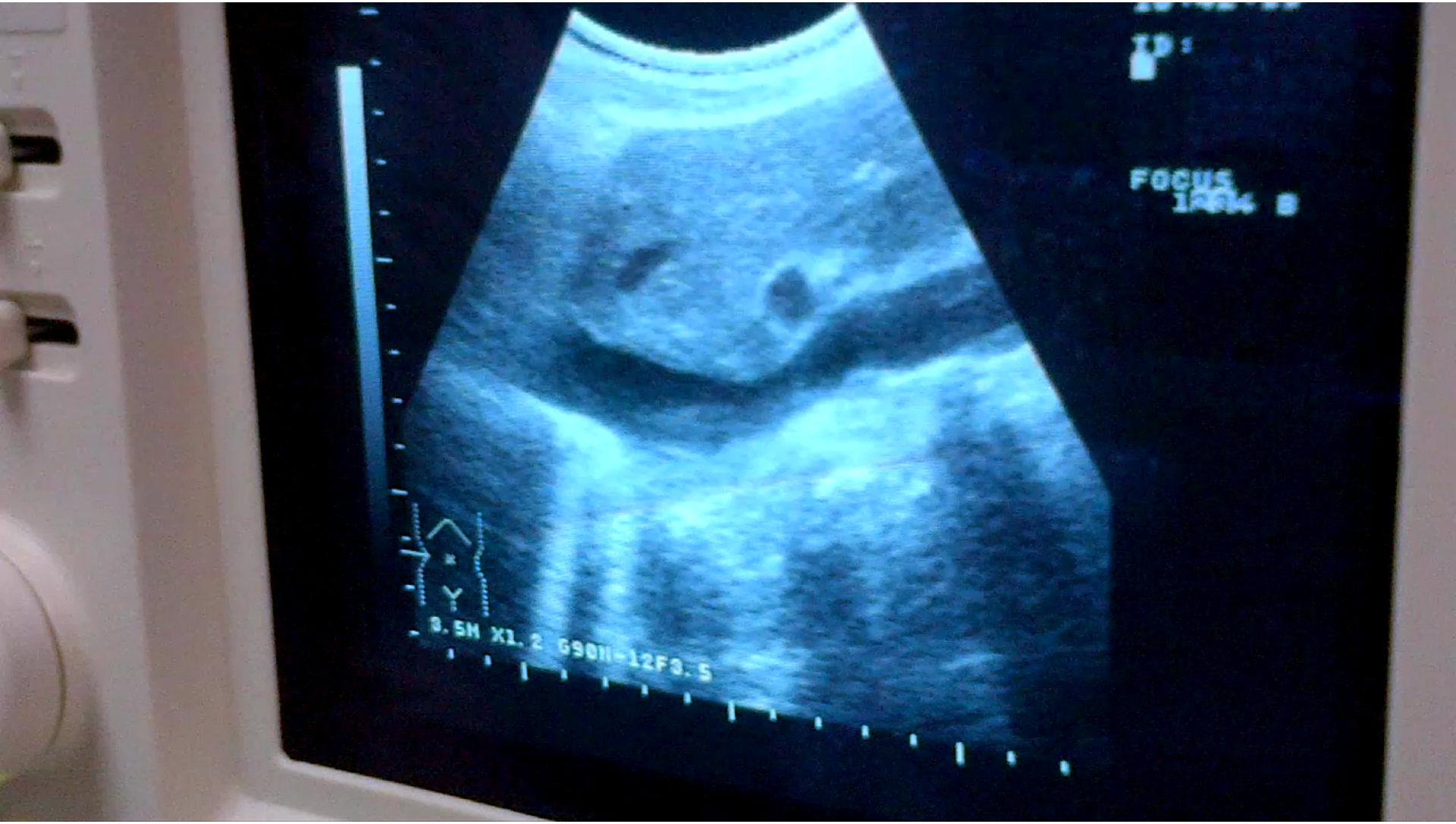


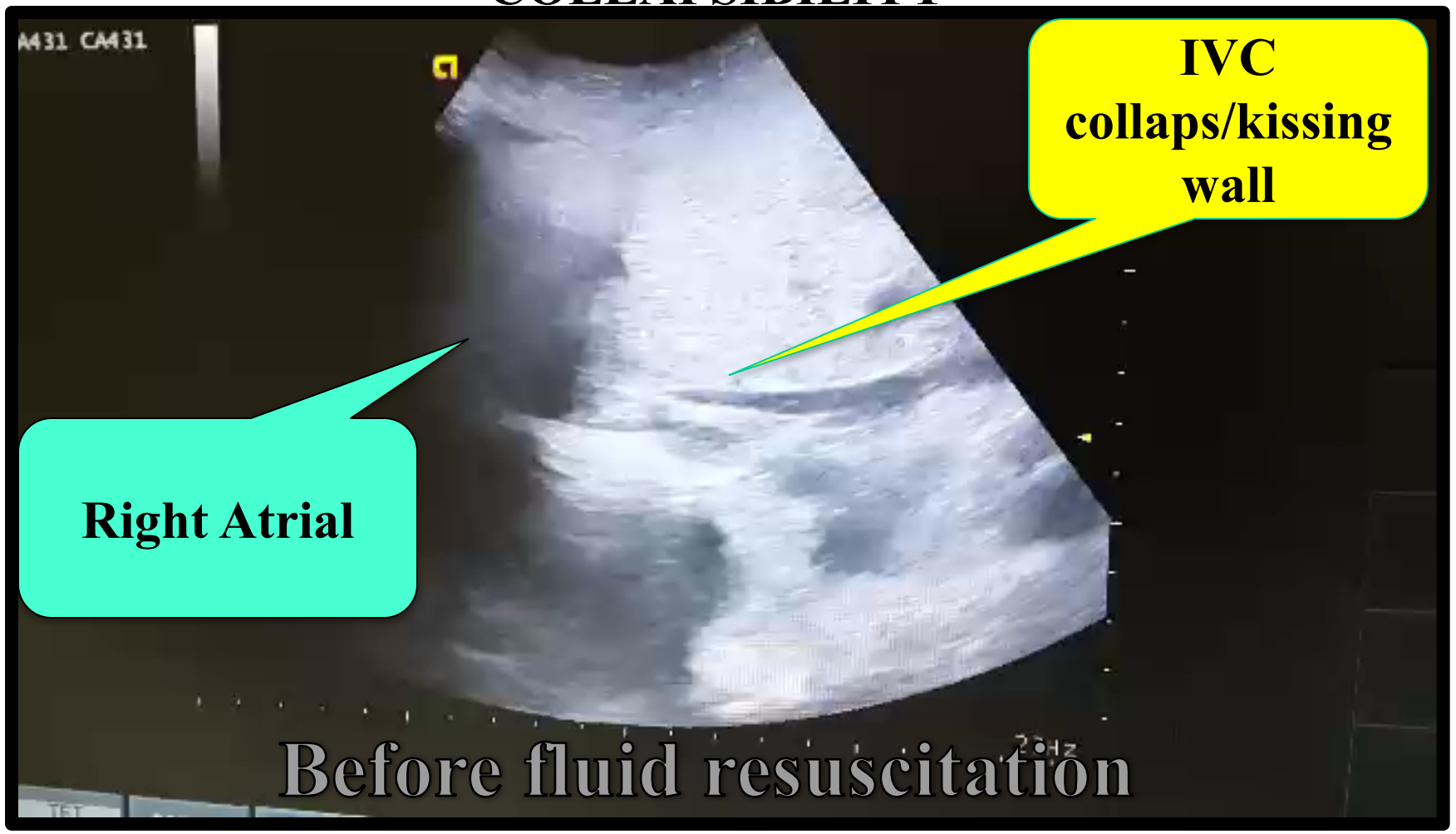
Fig. (2). IVC diameter measured on expiration (**A**) and on inspiration (**B**) [5].

- IVC collapsible index = (IVCd exp – IVCd insp)/ IVCd exp
- 0% is overloaded, 100% is volume depleted
- 2.04 - 1.51/2.04 = 25%. (Volume overloaded)



mfda

MONITORING FLUID RESPONSIVENESS IN 6 YO BOY SUFFERED FROM DENGUE SHOCK SYNDROME USING INFERIOR CAVA VEIN COLLAPSIBILITY

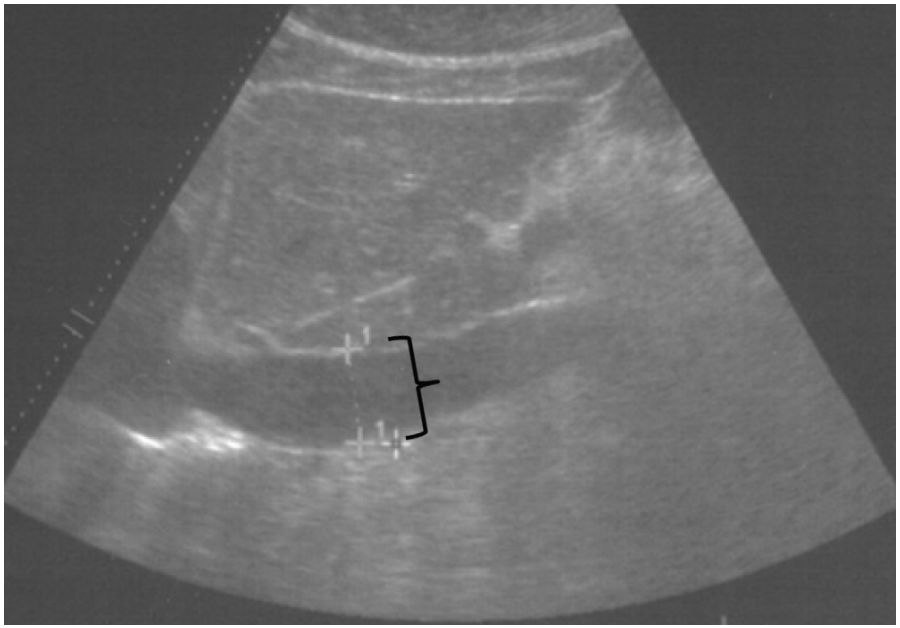


MONITORING FLUID RESPONSIVENESS IN BOY SUFFERED FROM DENGUE SHOCK SYNDROME USING INFERIOR CAVA VEIN COLLAPSIBILITY

After 6 hours fluid resuscitation with Crystalloid and albumin iso-oncotic

Inferior Vena Cava Collapse

- Ultrasound measure of iIVC and eIVC
- N = 30 septic ICU patients



	Correlation with eIVC	*P*-value
CVP	0.56	0.001
EVLW	0.59	0.001
EVLW index	0.63	0.001
ITBV	0.51	0.004
ITBV index	0.35	0.05
ITTV	0.68	0.001
PaO_2/FiO_2	0.47	0.008

EVLW = Extravascular lung water
ITBV = Intrathoracic blood volume
ITTV = Intrathoracic thermal volume

Schefold JC, et al. *J Emerg Med*. 2010;38(5):632-637.

FLUID CHALLENGES USING CVP

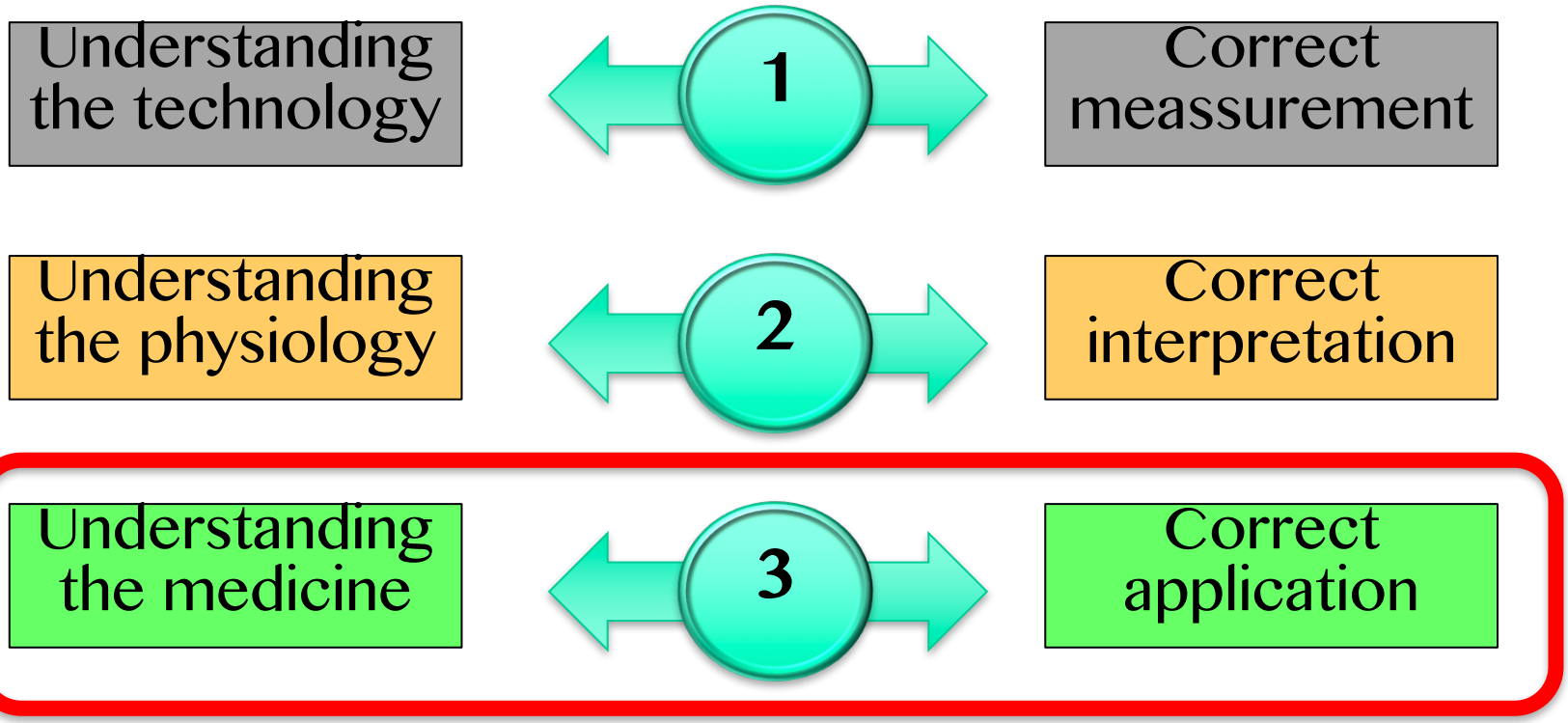
Guided by	CVP (cmH2))	PAOP mmHg)	Infusion
Start	<8	<10	200 ml/10 mnt
	<12	<14	100 ml/10 mnt
	12	14	50 ml/mnt
During infusion	↑>5	↑>7	Stop
After 10 min	2	3	Continue
	2>↑ = 5	3>↑=7	Wait 10 min
	↑>5	↑>7	Stop
After waiting 10 min	Still ↑>2	Still ↑>3	Stop
	↑ =2	↑ =3	Repeat

10 cm H_20 = 7.3 mm Hg. CVP, central venous pressure; PAOP, pulmonary artery occlusion pressure

Weil MH, Henning RJ: New concepts in the diagnosis and fluid treatment of circulatory shock. Anesth Analg 1979;S8:124

Fundamentals of Hemodynamic monitoring

The 3 magic push-button



Stratifikasi hemodinamik monitoring perioperative

mfda

Modular stepwise monitoring concept

Level 1

- Arterial line
- CVC

Targets

HR	= 100 bpm
MAP	= 65 mmHg
CVP	= 8-12 mmHg
Diuresis	= 1 ml/kg/h
Lactate	= 1-2 mmol/l
$ScVO_2$	= 70%
Hb	= 8-10 g/l

Consider level 2 or 3 if

- Diuresis < 1 ml/kg/h
- Lactate > 2 mmol/l
- $ScVO_2$ < 70%

despite fluid trial

Adapted from Hofer CK et al - Eur J Anaesth 2009

Modular stepwise monitoring concept

Level 1

- Arterial line
- CVC

Targets

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Hb	= 8-10 g/l

Consider level 2 or 3 if

- Diuresis < 1 ml/kg/h
- Lactate > 2 mmol/l
- $ScVO_2$ < 70%

despite fluid trial

Level 2

- Minimally invasive
- Continuous $ScvO_2$

Targets

CI	> 2.5 l/min/m^2
SVV	< 12 %
PPV	< 12 %

GEDVI = 600-800 ml/m^2

EVLWI < 7 ml/kg

Consider level 3 if

- Left/right heart failure
- PAH
- Contraindication/ limitations of minimally invasive HM

Adapted from Hofer CK et al - Eur J Anaesth 2009

Modular stepwise monitoring concept

Level 1

- Arterial line
- CVC

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HR	= 100 bpm
MAP	= 65 mmHg
CVP	= 8-12 mmHg
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- Lactate > 2 mmol/l
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despite fluid trial

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- Continuous $ScvO_2$

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SVV	< 12 %
PPV	< 12 %
GEDVI	= 600-800 ml/m²
EVLWI	< 7 ml/kg

Consider level 3 if

- Left/right heart failure
- PAH
- Contraindication/ limitations of minimally invasive HM

Level 3

- PAC

Targets

CI	> 2.5 l/min/m²
PCWP	= 12-15 mmHg
SvO_2	> 65 %
CEDVI	= 110-130 ml/m²

Consider

- Contraindication/ limitations of PAC

Adapted from Hofer CK et al - Eur J Anaesth 2009

Modular stepwise monitoring concept

Echocardiographic evaluation

- Arterial line
- CVC

Targets

HR	= 100 bpm
MAP	= 65 mmHg
CVP	= 8-12 mmHg
Diuresis	= 1 ml/kg/h
Lactate	= 1-2 mmol/l
$ScVO_2$	= 70%
Hb	= 8-10 g/l

Consider level 2 or 3 if

- Diuresis < 1 ml/kg/h
- Lactate > 2 mmol/l
- $ScVO_2$ < 70%

despite fluid trial

- Minimally invasiv
- Continuous $ScvO_2$

Targets

CI	> 2.5 l/min/m²
SVV	< 12 %
PPV	< 12 %
GEDVI	= 600-800 ml/m²
EVLWI	< 7 ml/kg

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- PAH
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- PAC

Targets

CI	> 2.5 l/min/m²
PCWP	= 12-15 mmHg
SvO_2	> 65 %
CEDVI	= 110-130 ml/m²

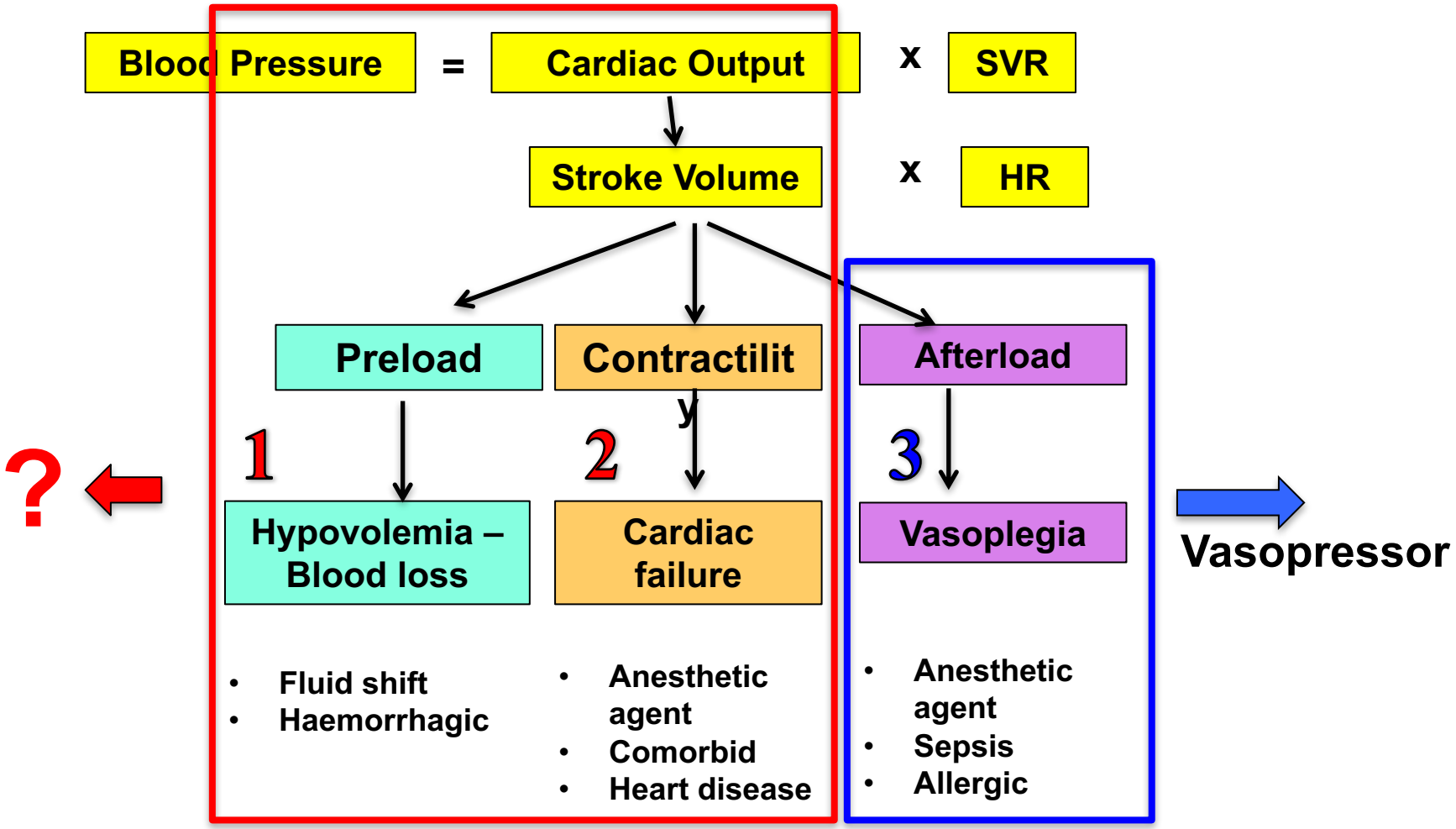
Consider

- Contraindication/ limitations of PAC

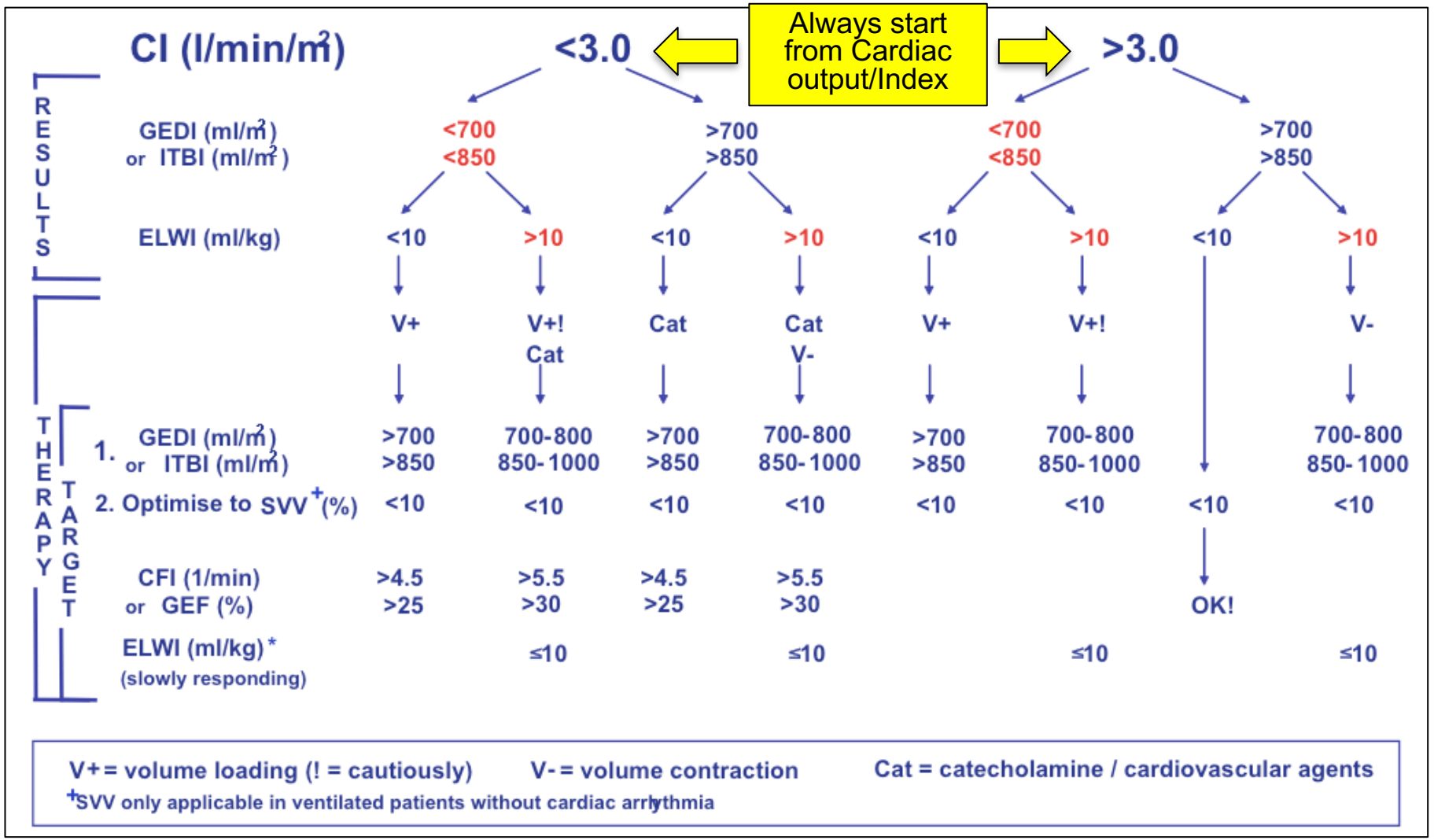
Adapted from Hofer CK et al - Eur J Anaesth 2009

Application And Stratification Of Hemodynamic Monitoring In Critically Ill Patients

Perioperative haemodynamic failure: 1) hypovolemia 2) Cardiac failure 3) Vasoplegia



Application of PiCCO Hemodynamic Monitoring in ICU



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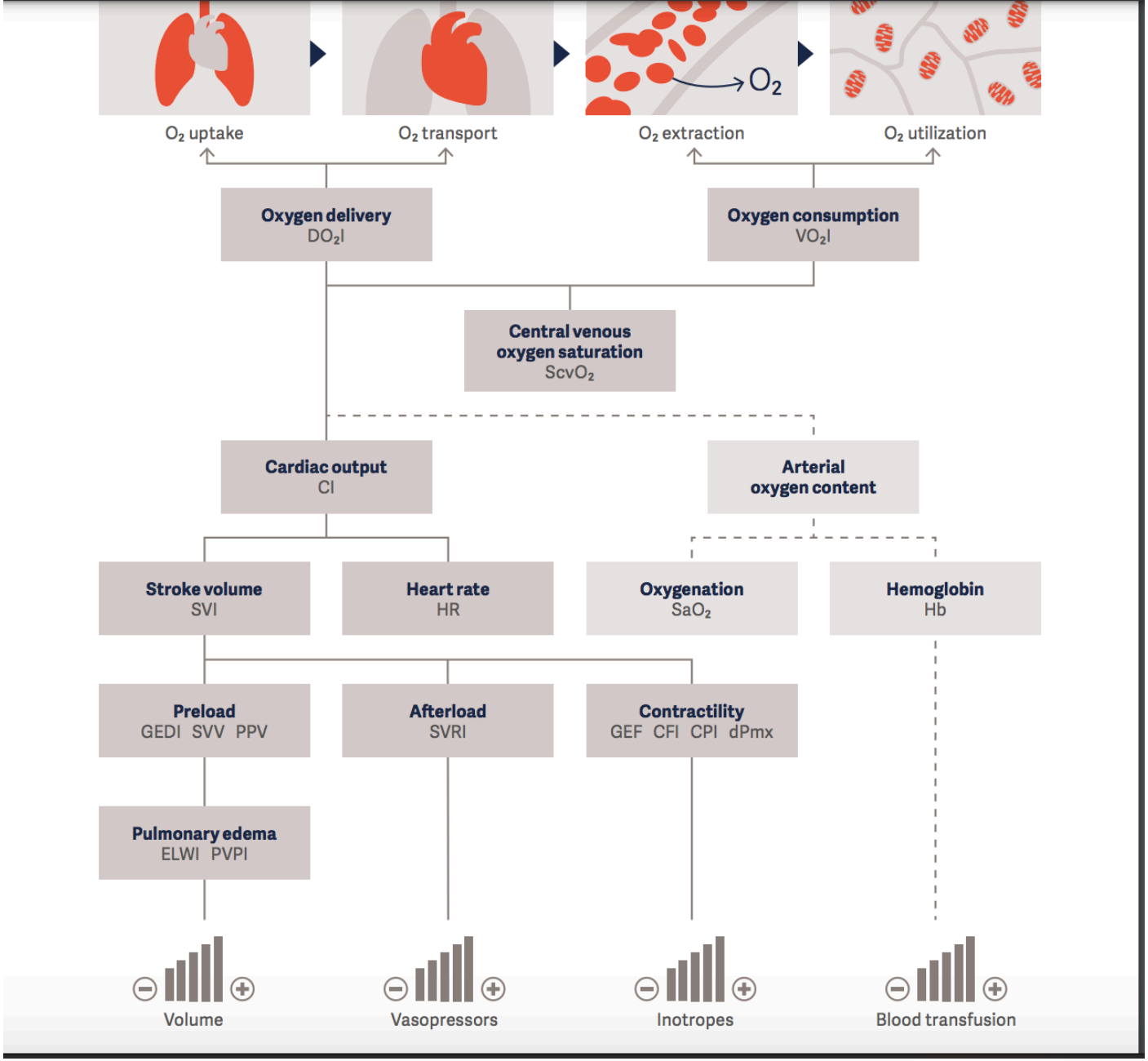
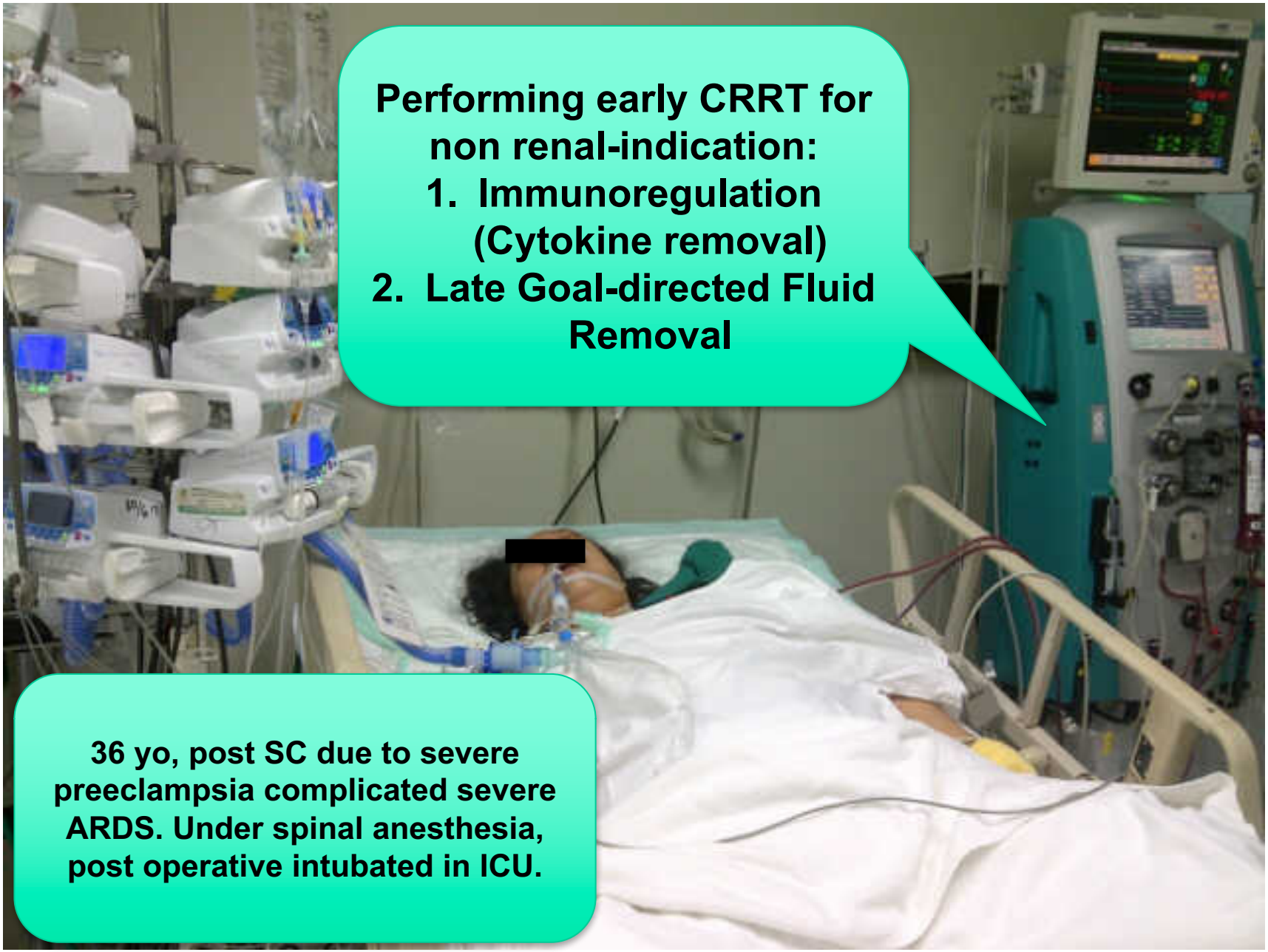

O_2
O_2 uptake
O_2 transport
O_2 extraction
O_2 utilization
Oxygen delivery
DO_2I
Oxygen consumption
VO_2I
Central venous oxygen saturation
$ScvO_2$
Cardiac output
CI
Arterial oxygen content
Stroke volume
SVI
Heart rate
HR
Oxygenation
SaO_2
Hemoglobin
Hb
Preload
GEDI SVV PPV
Afterload
SVRI
Contractility
GEF CFI CPI dPmx
Pulmonary edema
ELWI PVPI
Volume
Vasopressors
Inotropes
Blood transfusion

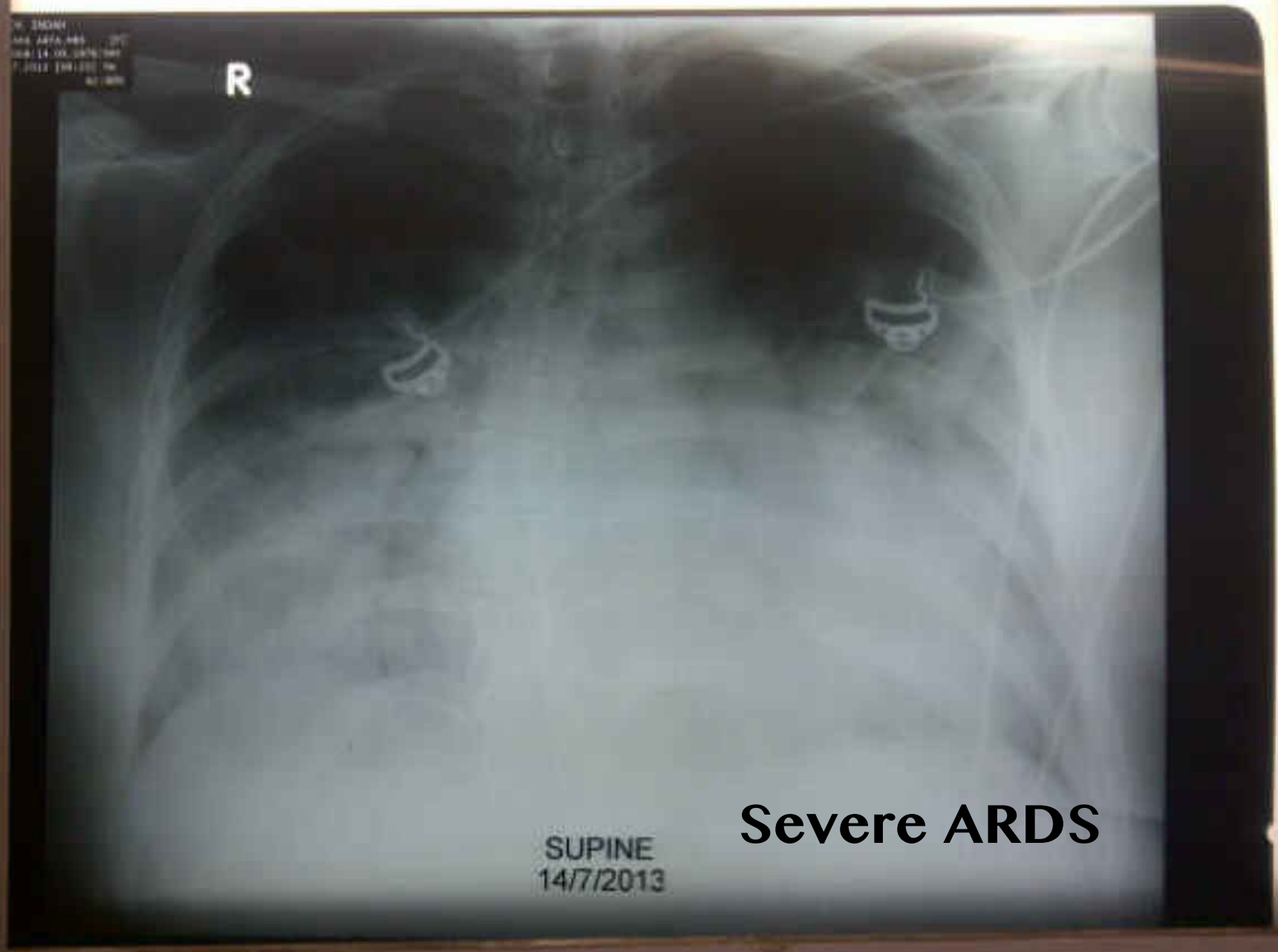
Case 5

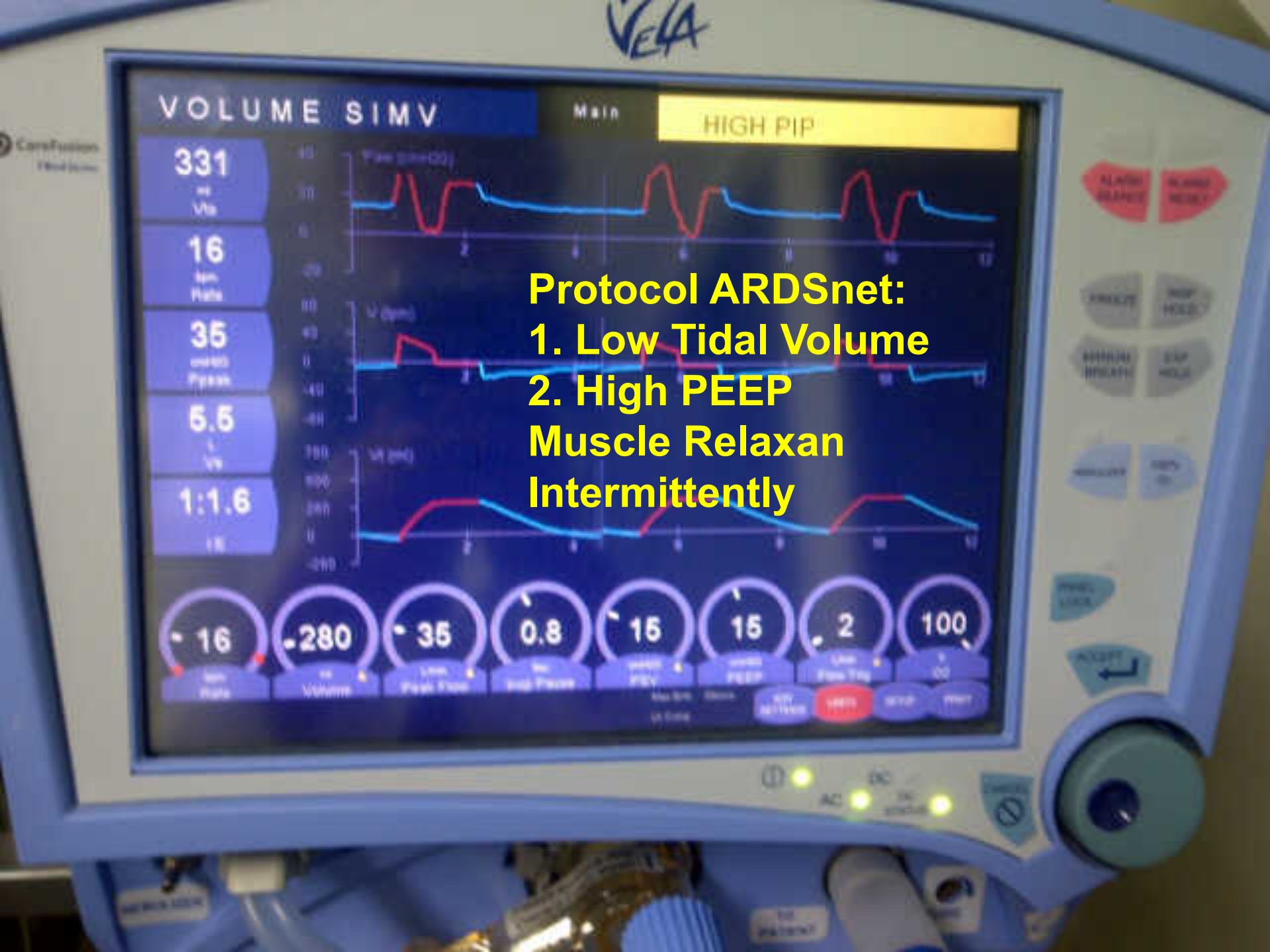
- Male, 66 yo, admited to ICU because of dyspneu, intubated put on SIMV.
- History of CHF FC class 3-4. with cardiomyopathy chronic hypertension.
- Vitals: BP 185/85, HR 130 AF. Temp 38. PE: rares both side , AF rapid, JVP >2.
- Lab: leucocyte 11.000, segmen 80, BNP 33.000, Lactate 5. ECG no ST-Change.
- PiCCO inserted. CI 1.78, GEDI 1156, SVRI 4000, CPI 0.2, GEF 20%. EVLWI 25 PVPI 2.0
- What is the diagnose and hemodynamic interpretation?

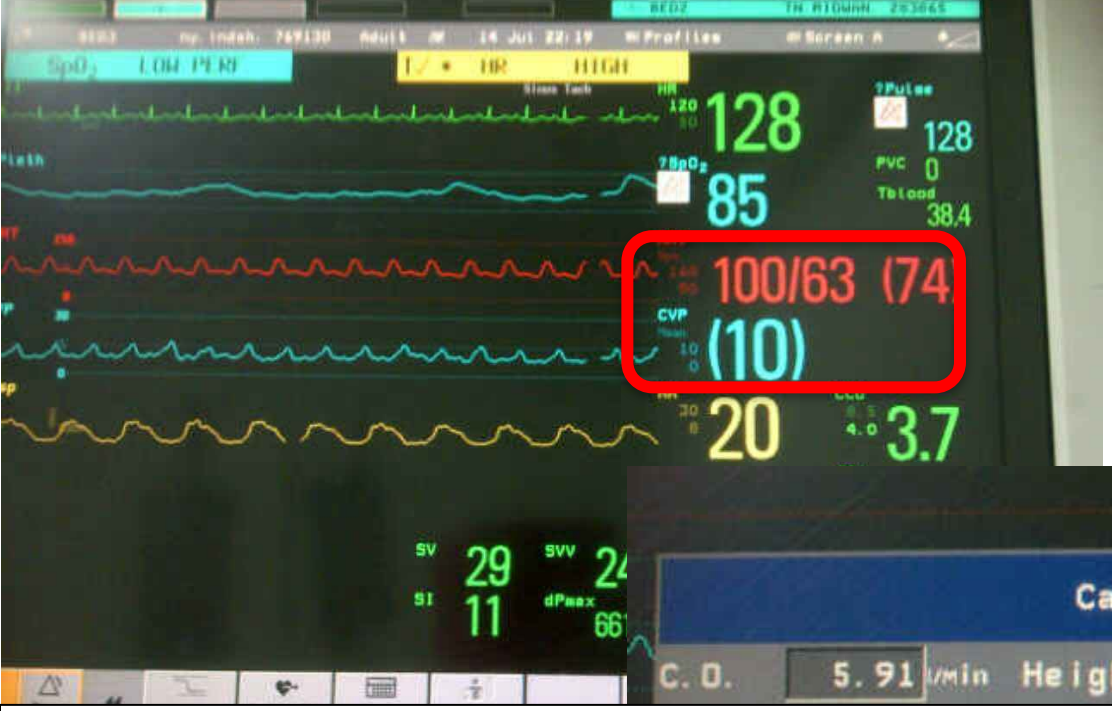
Case cont..

- **Severe acute heart failure forrester subset IV**
- **Dobutamine start 5 mikro, NTG 10 mikro, furosemide drip start 5 mg,**


Performing early CRRT for non renal-indication:
1. Immunoregulation (Cytokine removal)
2. Late Goal-directed Fluid Removal
36 yo, post SC due to severe preeclampsia complicated severe ARDS. Under spinal anesthesia, post operative intubated in ICU.


R
SUPINE
14/7/2013
Severe ARDS

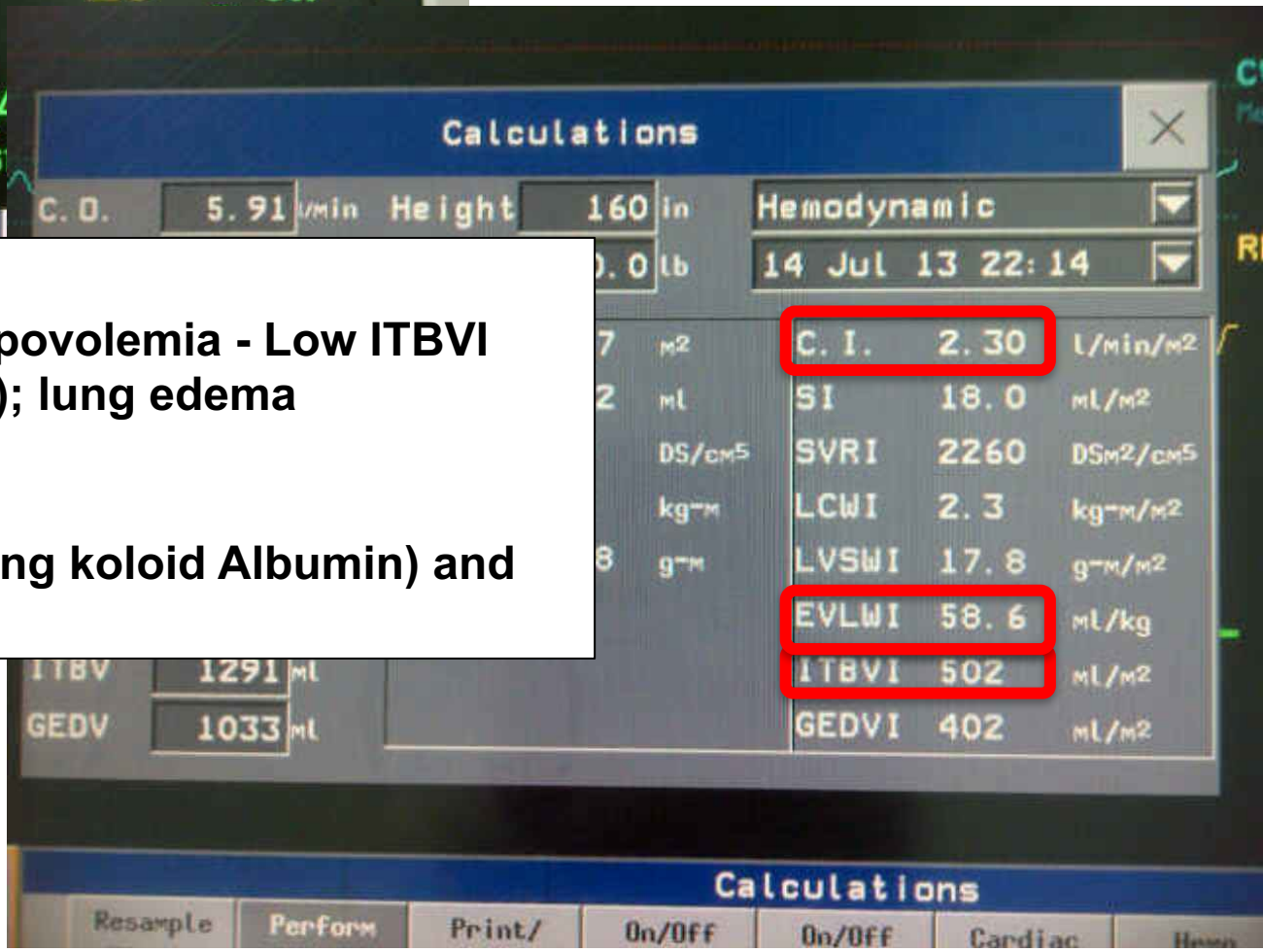

VOLUME SIMV
Main
HIGH PIP
331
16
35
5.5
1:1.6
16
280
35
0.8
15
15
2
100
Protocol ARDSnet:
1. Low Tidal Volume
2. High PEEP
Muscle Relaxan
Intermittently



Day 1 - Severe hypoxemia

Filling Pressure Monitoring:

Looks normal



Flow Monitoring:

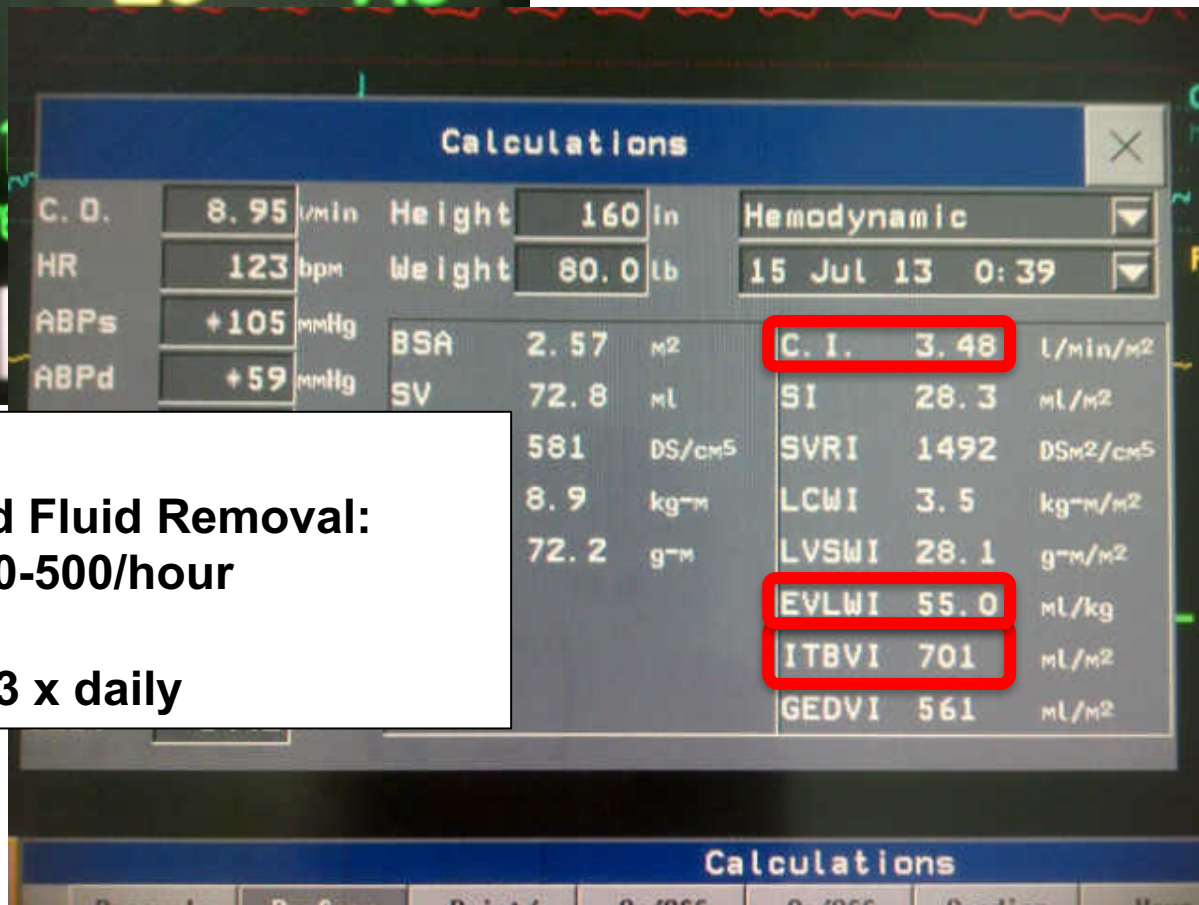
A: Low CI (2.30) due to Hypovolemia - Low ITBVI (502) with High ELWI (58.6); lung edema permeability (PVPI 6):

Therapy:

Fluid Resuscitation (Loading koloid Albumin) and Fluid Removal

Day 1, Post loading;

1. Increased CI because of increased ITBVI
2. Maintain permissive hypoxemia 12 hours to prevents too much increased Intra Thoracic Pressure



Post loading;

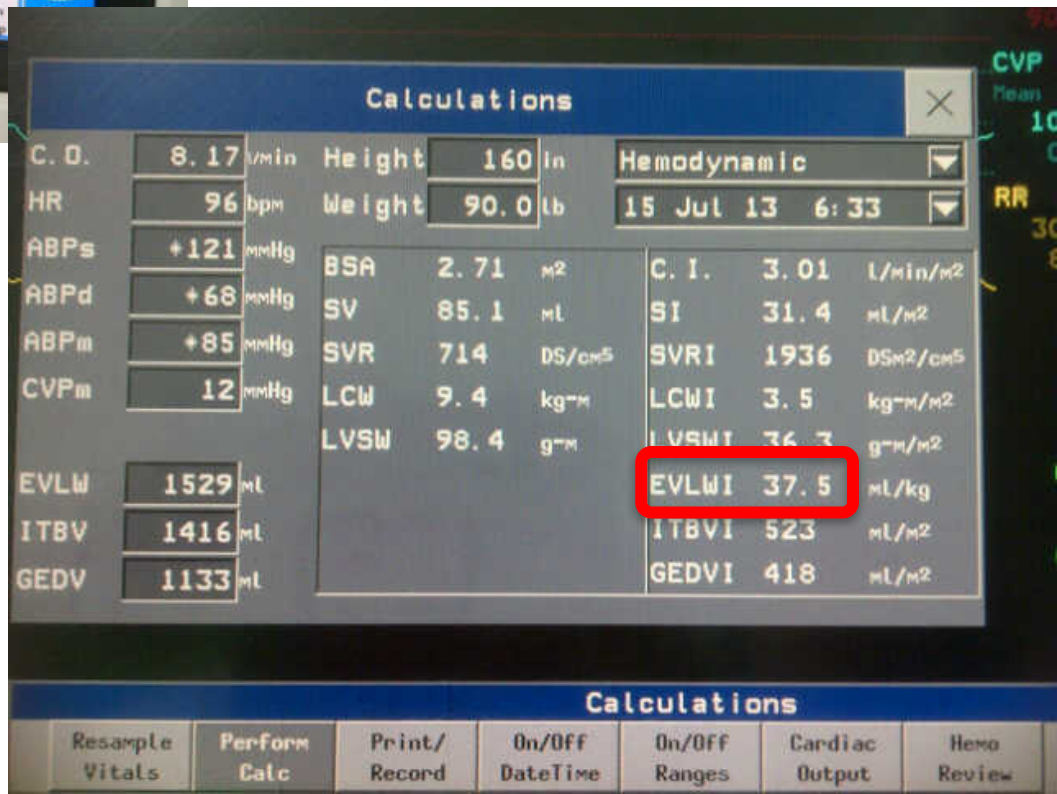
1. Continyu Late Goal-directed Fluid Removal:
    1. CRRT; removal fluid 300-500/hour
    2. Furosemide 10 mg/h
    3. Albumin Hyperoncotic 3 x daily

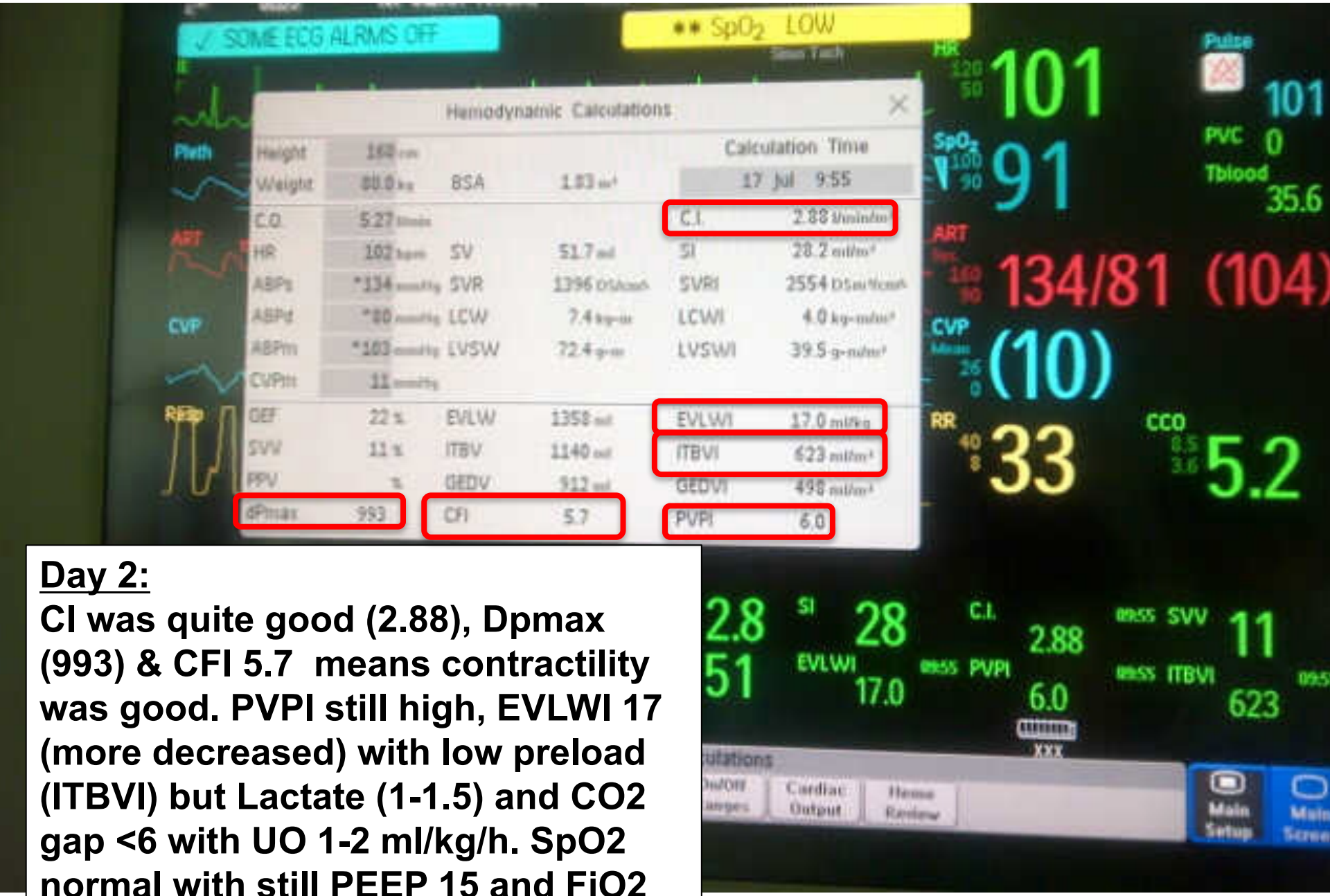
After 12 hours:

1. **Decreased EVLWI with increased SpO2**
2. **Decreased ITBVI (preload) due to restricted Strategy**
3. **Lactate 1-2 with CO2 gap keep normal < 6**

After Intervention (PEEP):

1. **Increased SpO2**
2. **Decreased ELWI from 58 to 37.5**
3. **Decreased ITBVI (preload)**
4. **Action: : another loading Alb 5%**





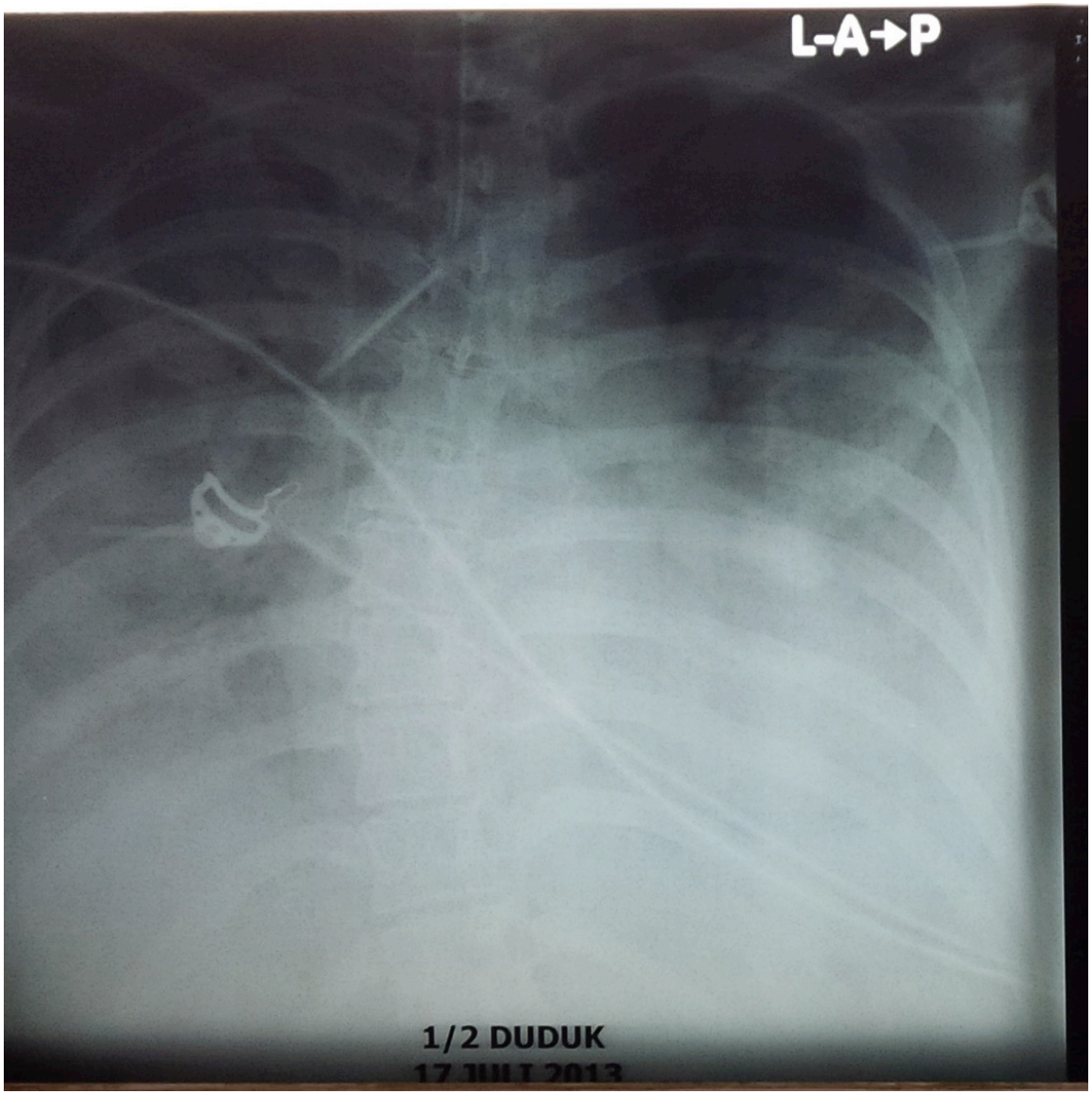
Day 2:

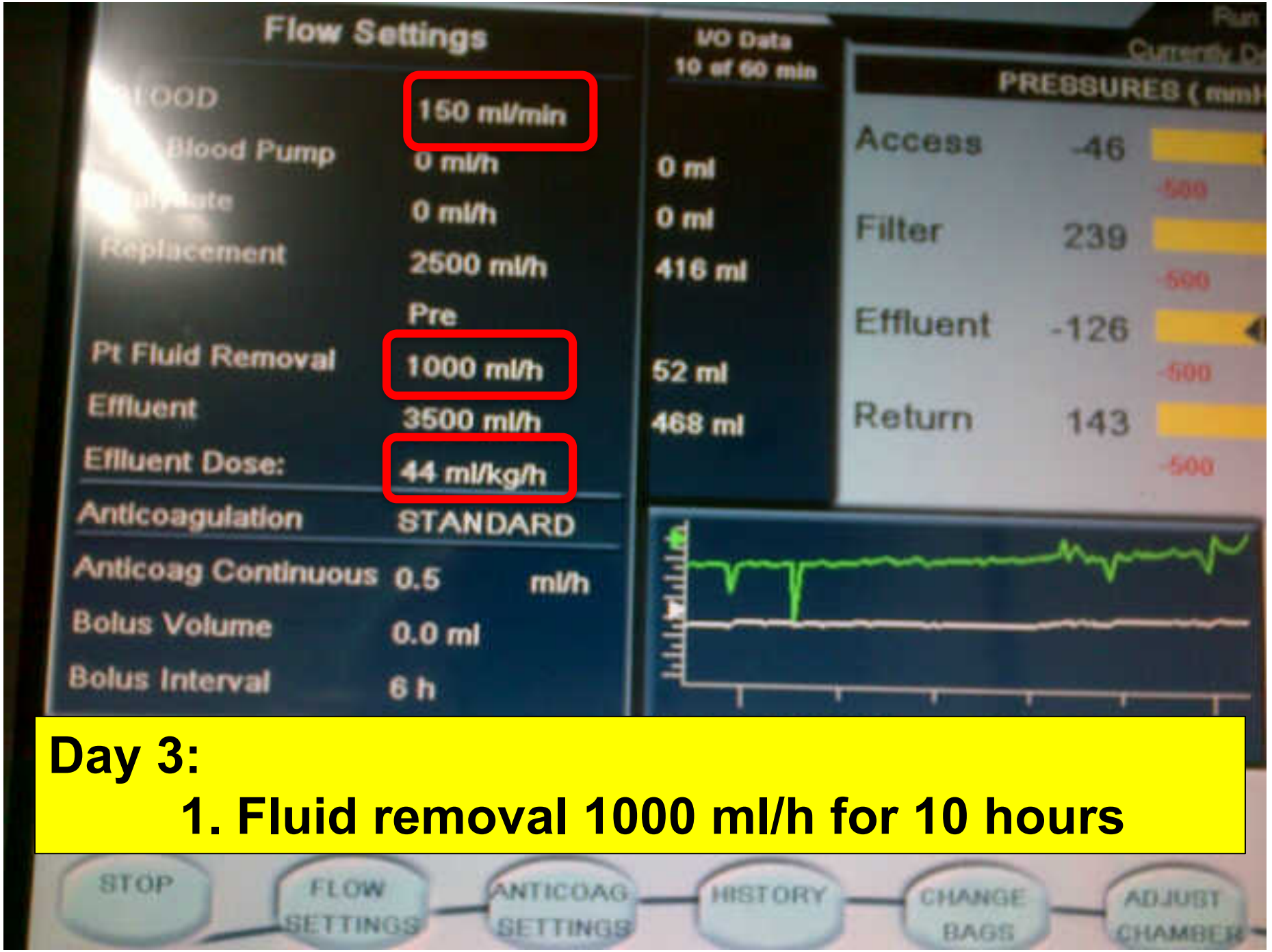
CI was quite good (2.88), Dpmax (993) & CFI 5.7 means contractility was good. PVPI still high, EVLWI 17 (more decreased) with low preload (ITBVI) but Lactate (1-1.5) and CO2 gap <6 with UO 1-2 ml/kg/h. SpO2 normal with still PEEP 15 and FiO2 90%

Day 3:

1. **Early morning, after 3 hours CRRT stop for priming and nursing care, suddenly desaturation, high BP with high CVP.**
2. **HD Calc: Low CI with quite low ITBVI (708) and low Cardiac Function Index - CFI (hypoxemia).**
3. **Possible cause overload due to temporary stop fluid removal for 3 hours.**
4. **Action: Change Pressure Control, start CRRT with 1000 ml/h removal.**

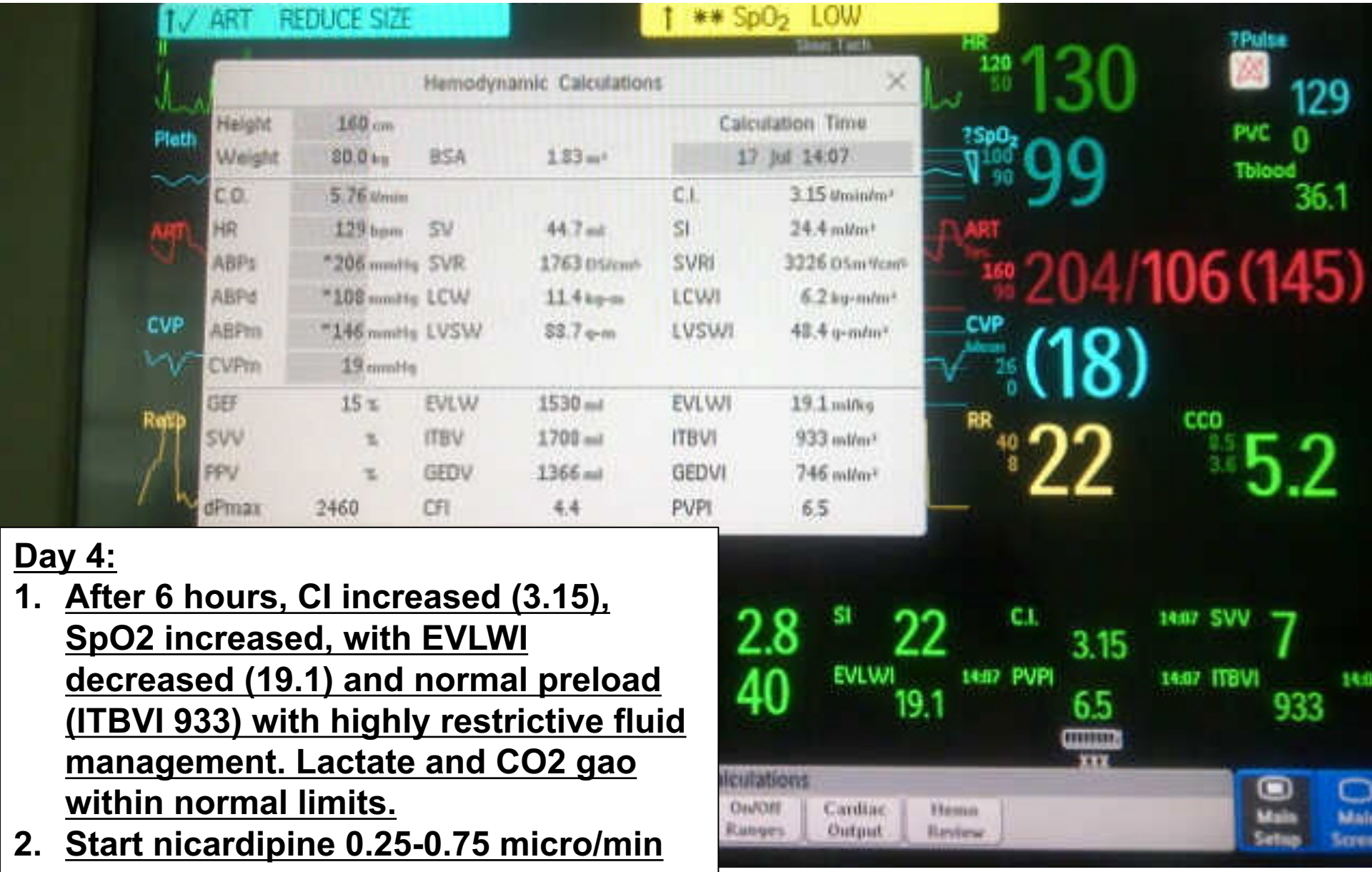
Day 3: CXR performing after desat showing with ELWI 27





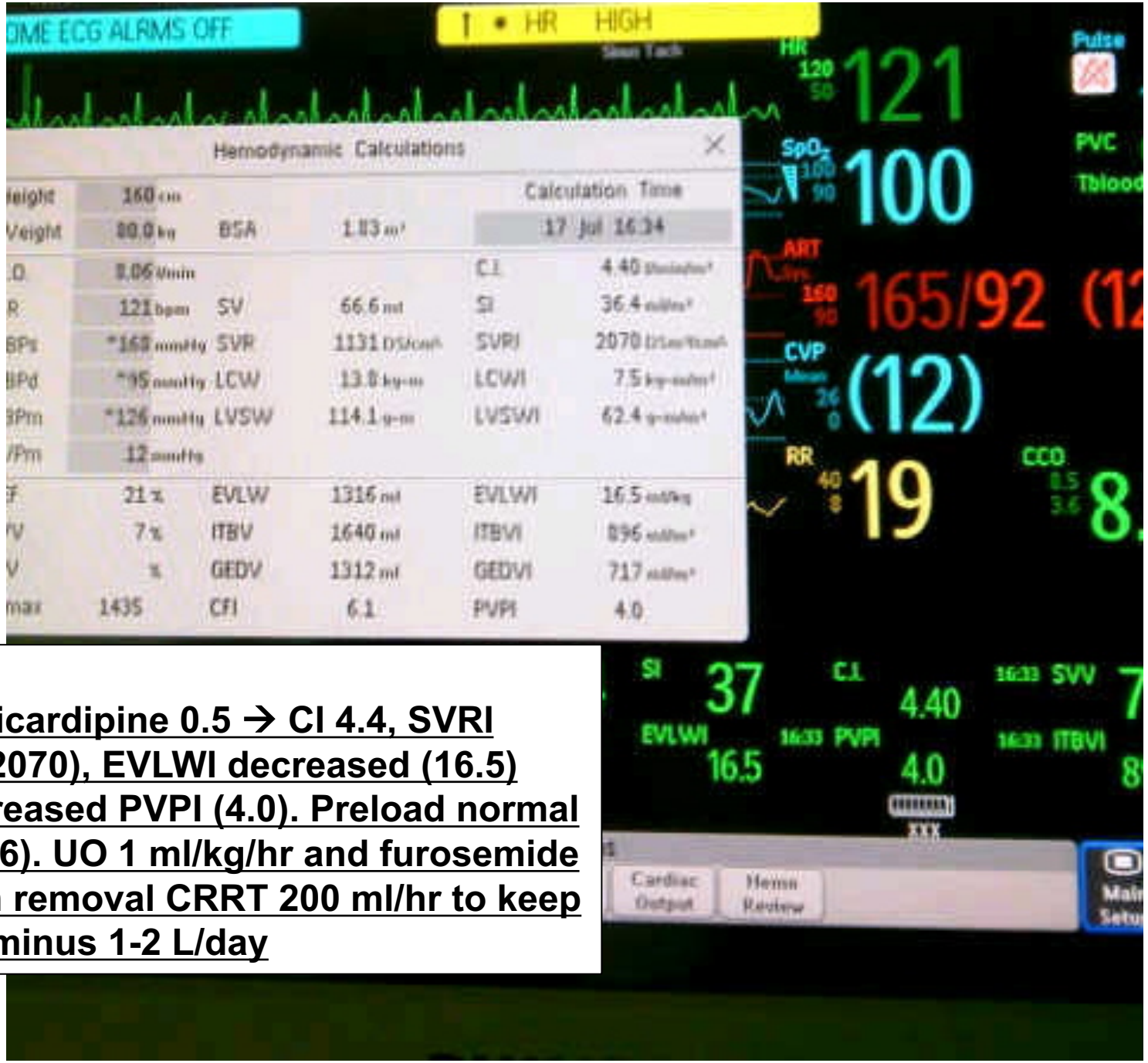
Day 3:

1. Fluid removal 1000 ml/h for 10 hours



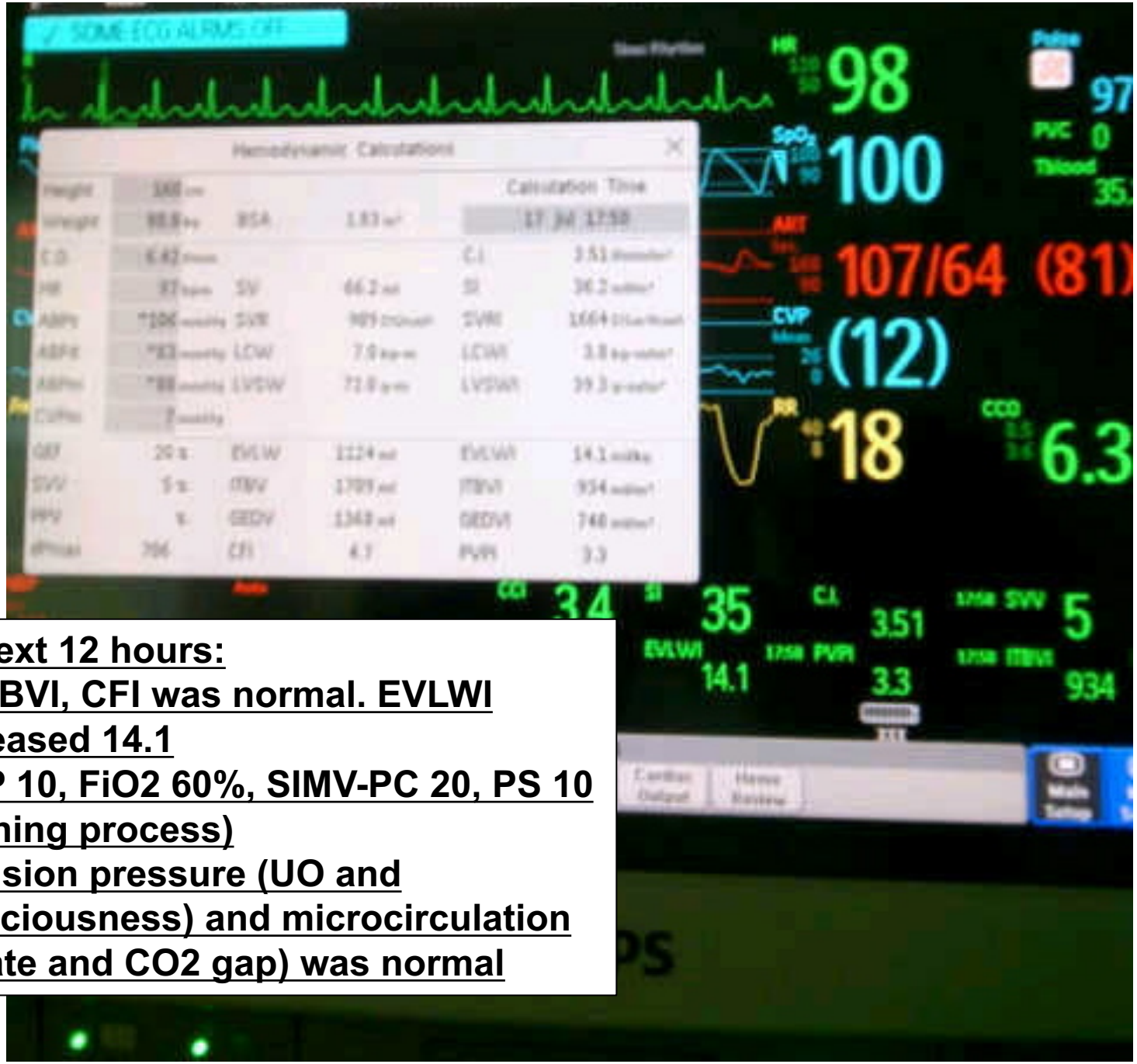
Day 4:

1. **After 6 hours, CI increased (3.15), SpO2 increased, with EVLWI decreased (19.1) and normal preload (ITBVI 933) with highly restrictive fluid management. Lactate and CO2 gao within normal limits.**
2. **Start nicardipine 0.25-0.75 micro/min because very high SVRI 3226**



Day 5:

1. With nicardipine 0.5 → CI 4.4, SVRI normal (2070), EVLWI decreased (16.5) with decreased PVPI (4.0). Preload normal (ITBVI 896). UO 1 ml/kg/hr and furosemide stop with removal CRRT 200 ml/hr to keep balance minus 1-2 L/day

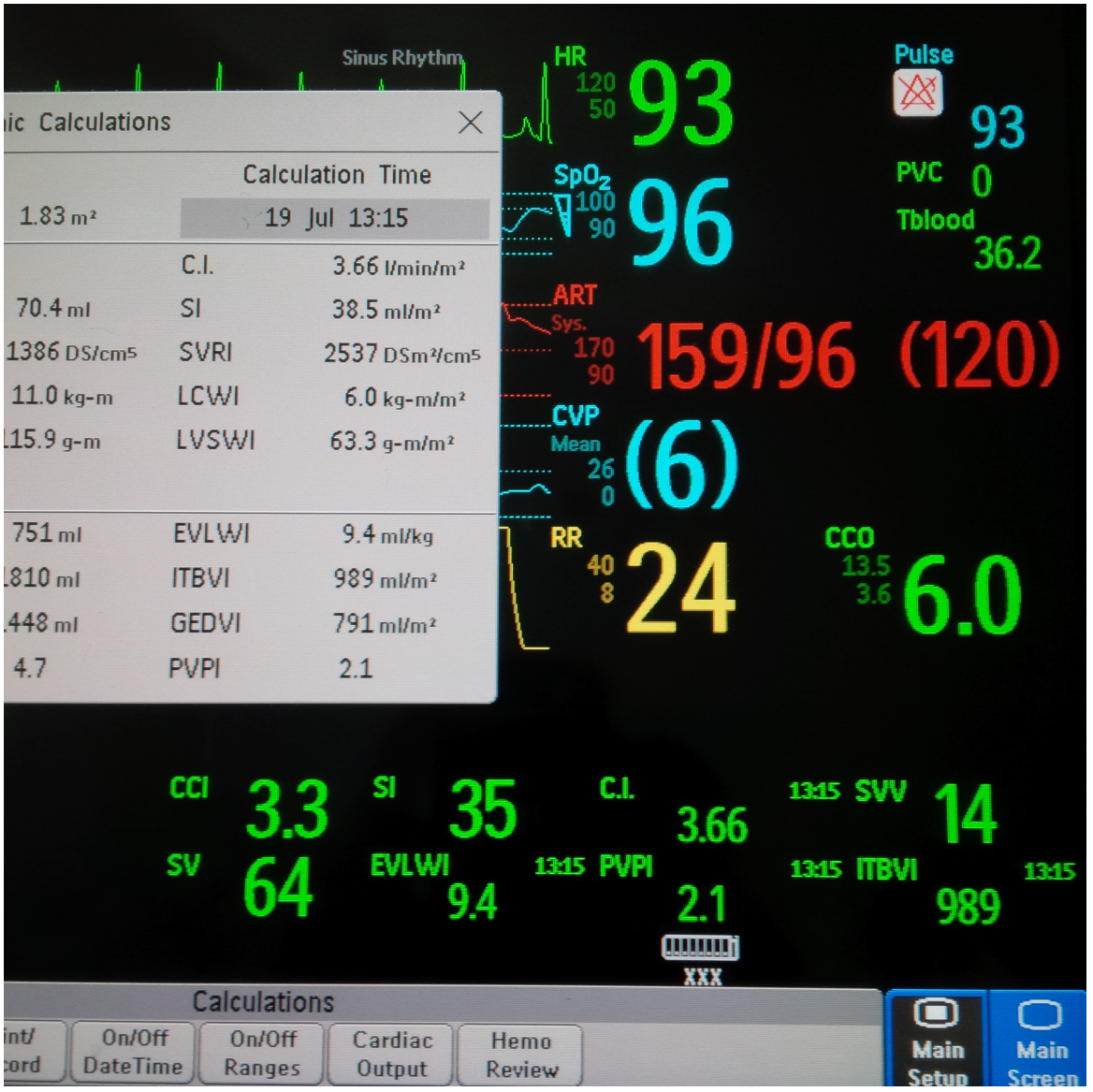


Day 5, next 12 hours:

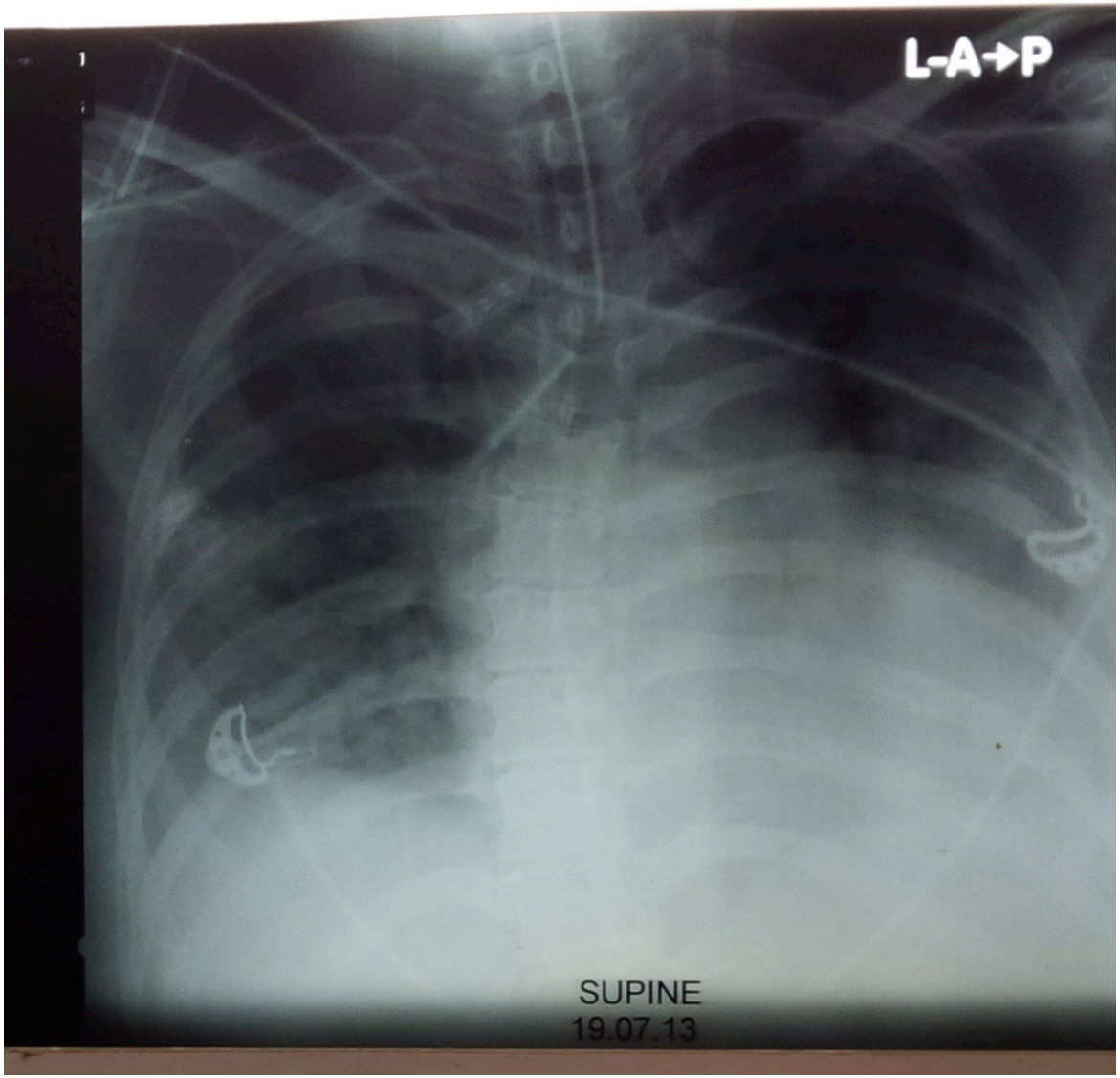
1. **CI, ITBVI, CFI was normal. EVLWI decreased 14.1**
2. **PEEP 10, FiO2 60%, SIMV-PC 20, PS 10 (weaning process)**
3. **Perfusion pressure (UO and Consciousness) and microcirculation (lactate and CO2 gap) was normal**

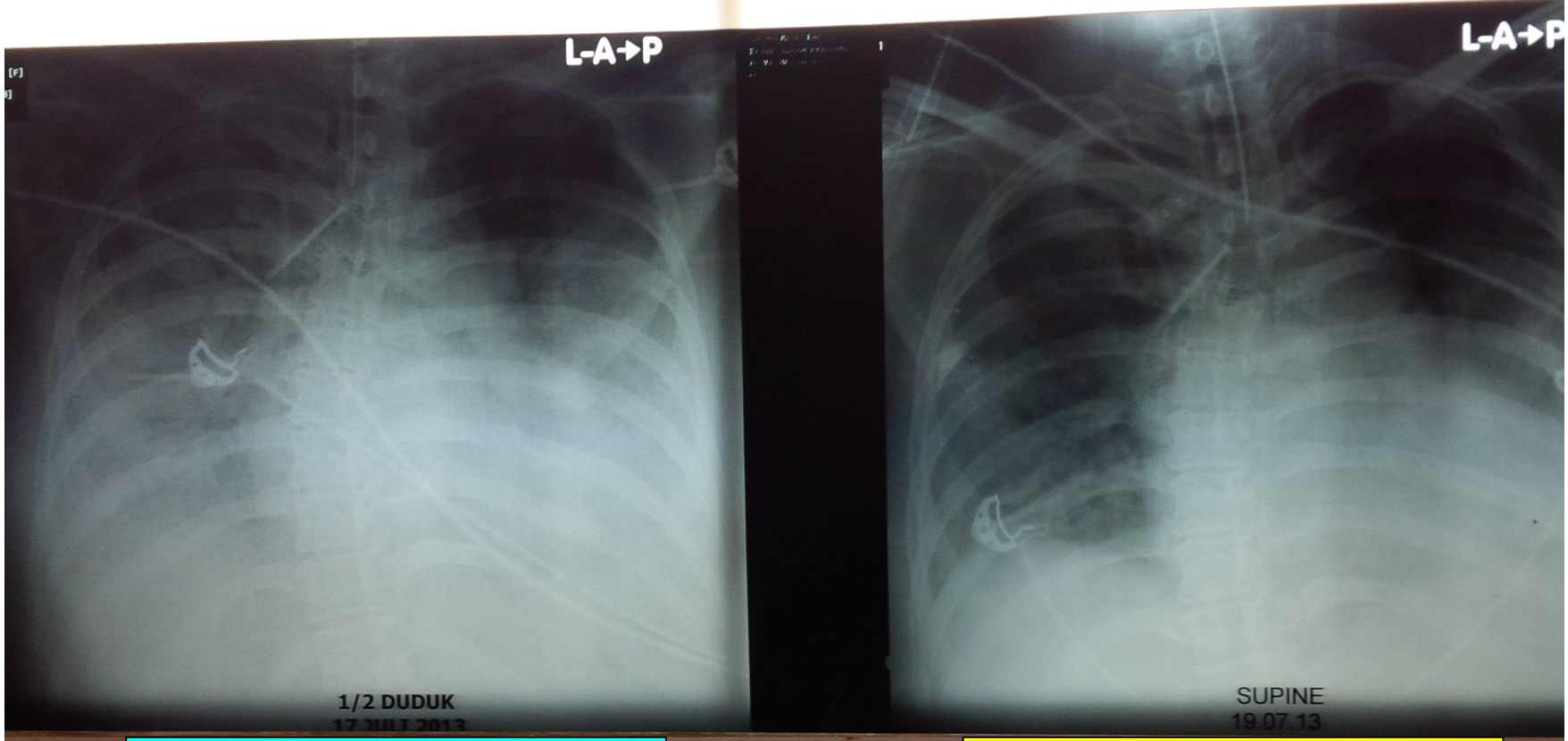

BED3
BED6
Adult
19 Jul 9:37
Profiles
6 Waves*
Calculations
Calculation Time
19 Jul 9:23
C.I. 3.64 l/min/m²
SI 36.8 ml/m²
SVRI 2523 DSm²/cm⁵
LCWI 5.9 kg-m/m²
LVSWI 60.1 g-m/m²
EVLWI 12.4 ml/kg
ITBVI 790 ml/m²
GEDVI 632 ml/m²
PVPI 3.5
HR 95
Pulse 94
SpO2 100
PVC 0
Tblood 35.7
ART 146/91 (114)
CVP (4)
RR 20
CCO 6.0
CCI 3.3
SV 63
SI 35
C.I. 3.64
SVV 17
EVLWI 12.4
PVPI 3.5
ITBVI 790


PRESSURE SIMV
Main
458 ml Vte
25 bpm Rate
19 cmH2O Ppeak
12.4 L Ve
1:1.7 I:E
V (lpm)
Vt (ml)
12 bpm Rate
20 cmH2O Insp Pres
0.8 Sec Insp Time
10 cmH2O PSV
6 cmH2O PEEP
2 L/min Flow Trig
45 % O2
FLOW TERMINATION


Sinus Rhythm
Calculations
Calculation Time
1.83 m²
19 Jul 13:15
C.I. 3.66 l/min/m²
70.4 ml SI 38.5 ml/m²
1386 DS/cm⁵ SVRI 2537 DSm²/cm⁵
11.0 kg-m LCWI 6.0 kg-m/m²
115.9 g-m LVSWI 63.3 g-m/m²
751 ml EVLWI 9.4 ml/kg
1810 ml ITBVI 989 ml/m²
1448 ml GEDVI 791 ml/m²
4.7 PVPI 2.1
HR 93
Pulse 93
SpO2 96
PVC 0
Tblood 36.2
ART 159/96 (120)
CVP (6)
RR 24
CCO 6.0
CCI 3.3
SV 64
SI 35
C.I. 3.66
SVV 14
EVLWI 9.4
PVPI 2.1
ITBVI 989
Calculations
On/Off DateTime
On/Off Ranges
Cardiac Output
Hemo Review
Main Setup
Main Screen

Day 6: CXR performing with EVLWI 9.4 (normal 5-7)





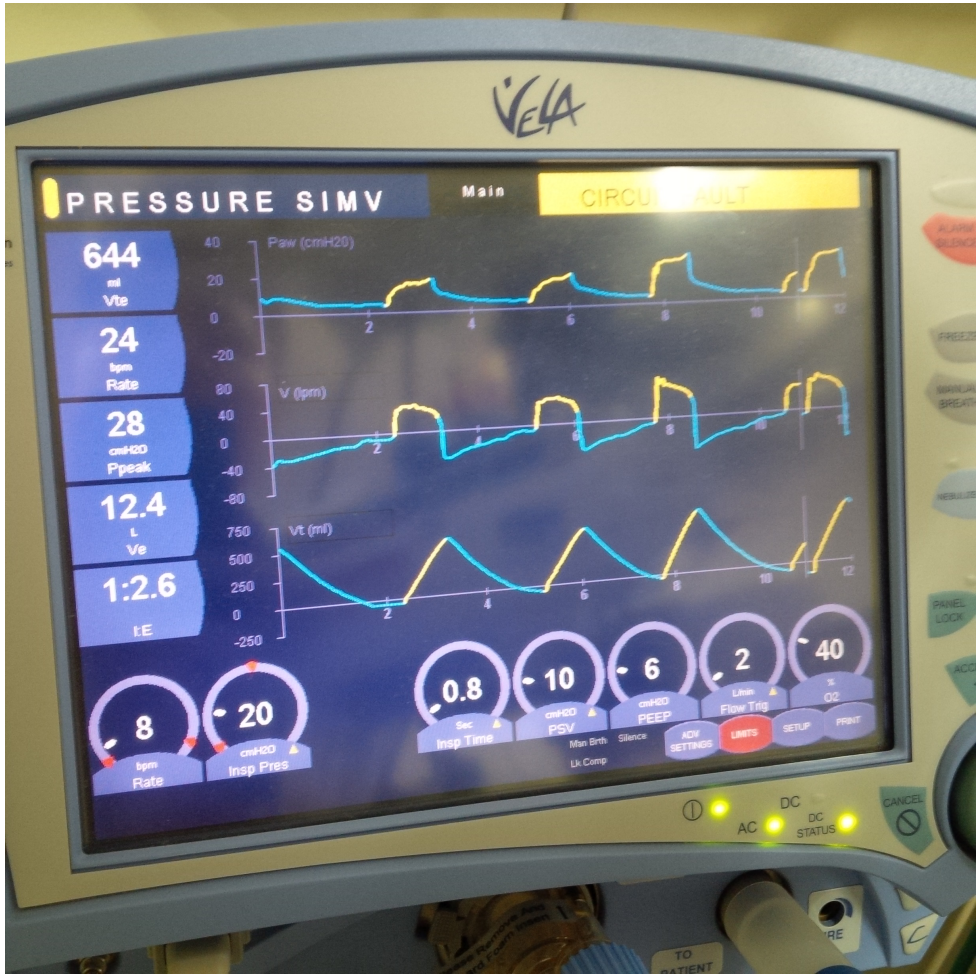
EVLWI 27

EVLWI 9.4

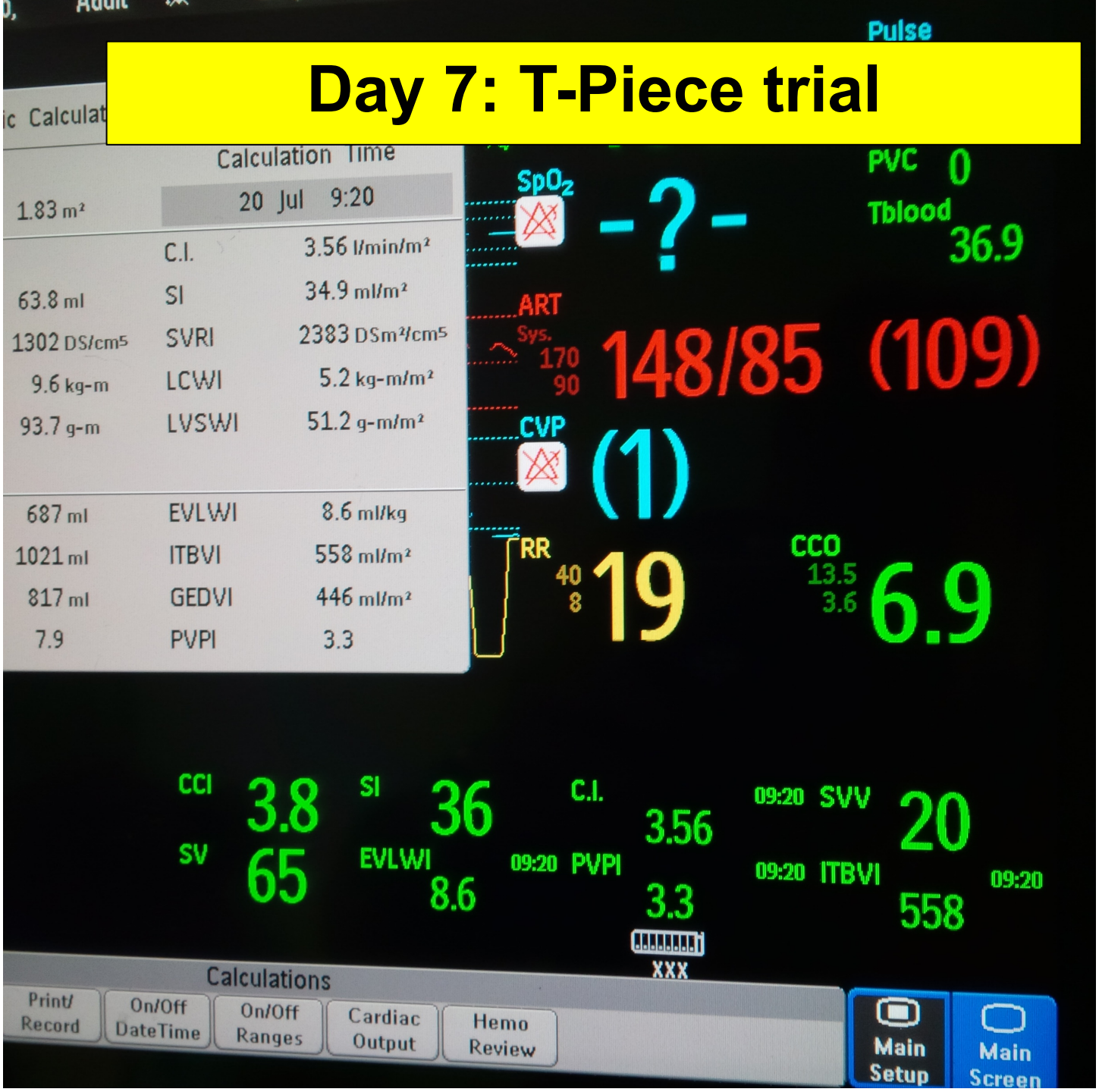
PVPI 6.0

PVPI 2.1

Day 6 next 12 hours: Continyu weaning with SIMV-PC. UO normal without furosemide. Removal CRRT 100/h



Day 7: T-Piece trial



THANK YOU