Implications of Cardiopulmonary interactions and its use in ICUs

DM Seminar

Harshith

Outline of discussion

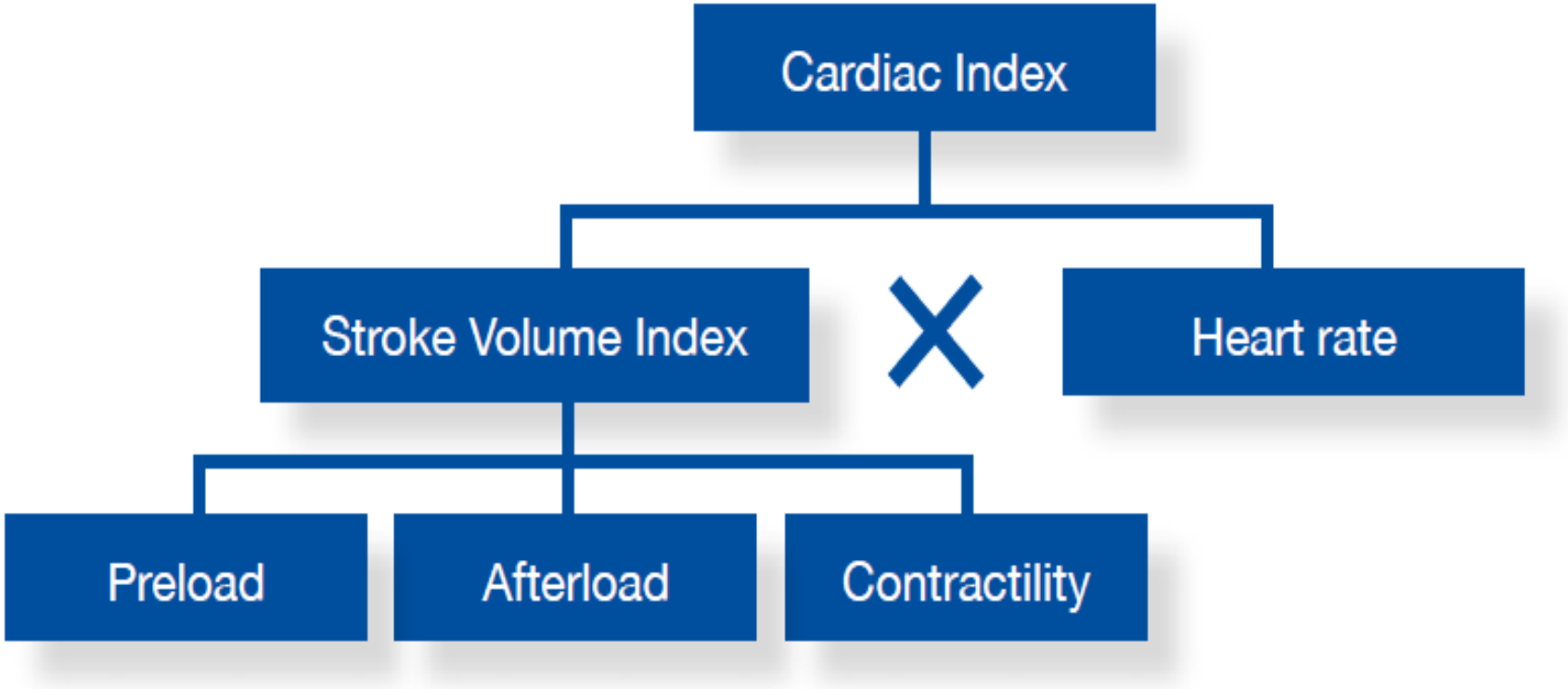
- Overview
- Physiology of cardiopulmonary interactions
- Effects of
  - ↑sed volume
  - ↑sed pressure
- Role in initiation & weaning
- Role in defining CV performance
- Preventing harmful interactions
- Role in hemodynamic monitoring of ventilated pts
- Velocity time integral
- PiCCO

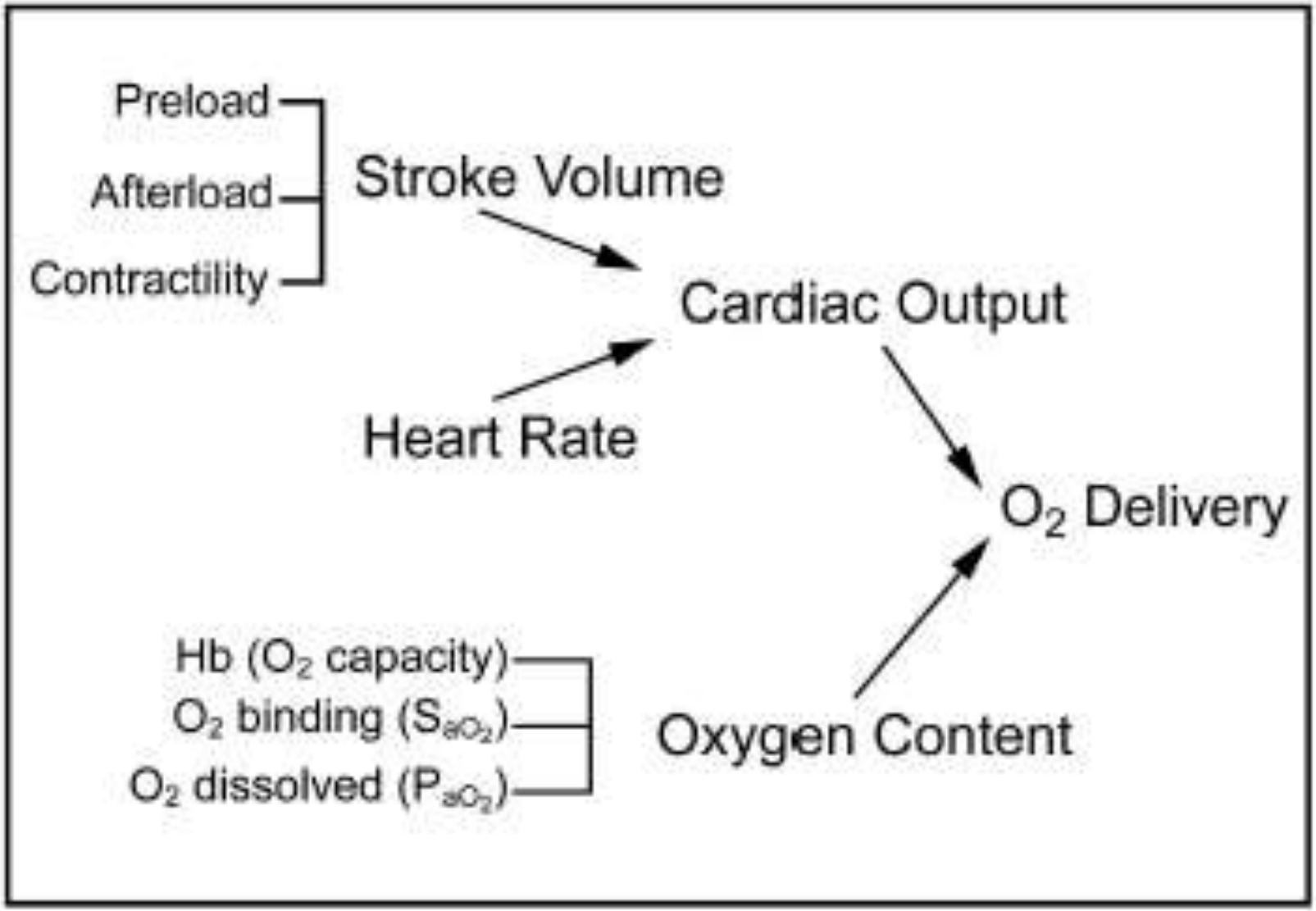
Overview

- Heart and lungs and connected anatomically and physiologically
- In series

- Changes in ventilation significantly affects cardiac physiology

Determinants of cardiac function




Preload
Afterload
Contractility
Stroke Volume
Cardiac Output
Heart Rate
O_2 Delivery
Hb (O_2 capacity)
O_2 binding (S_{aO_2})
O_2 dissolved (P_{aO_2})
Oxygen Content

What happens to lungs during MV?

- ↑sed volume : lung inflation

- ↑sed pressure : Intra thoracic pressure

Effects of normal lung inflation

- Vt <10ml/kg
- causes↓ vagal tone
- ↑ heart rate

- Phasic manner (during inspiration)
- Results in sinus arrythmia

Clinically used to document normal autonomic control

Anrep GV, Pascual W, Rossler R. Respiratory variations in the heart rate in the reflex mechanism of the respiratory arrhythmia. *Proc R Soc Lond B Biol Sci. 1936;119:191–217.*

In case of hyperinflation?

- Vt >10ml/kg
- ↑ vagal tone + sympathetic withdrawal

- ↓ both heart rate and blood pressure
- Compression of heart in cardiac fossa

Lung inflation mediates its reflex CVS effects by modulating central autonomic tone

Glick G, Wechsler AS, Epstein DE. Reflex cardiovascular depression produced by stimulation of pulmonary stretch receptors in the dog. *J Clin Invest. 1969;48:467–472*

Localised hyperinflation?

- In split lung ventilation, COPD
- No autonomic changes noted

Autonomic changes require general increase in lung volumes

Fuhrman BP, Everitt J, Lock JE. Cardiopulmonary effects of unilateral airway pressure changes in intact infant lambs. *J Appl Physiol.* 1984;56:1439–1448.

Role of humoral substances?

- Role of ANP
- Hyperinflation and PPV causes ↓sed RA stretch
- Simulate hypovolemia
- Decreased synthesis of ANP
- ↓sed natriuresis

This is the reason why ventilated pts gain weight early

Frass M, et al. Atrial natriuretic peptide release in response to different positive end-expiratory pressure levels. *Crit Care Med. 1993;21:343–347*

Effect on pulmonary vascular resistance

- By 2 mechanisms
  - ↓ hypoxia induced vasoconstriction
  - Altering cross sectional area of vessels

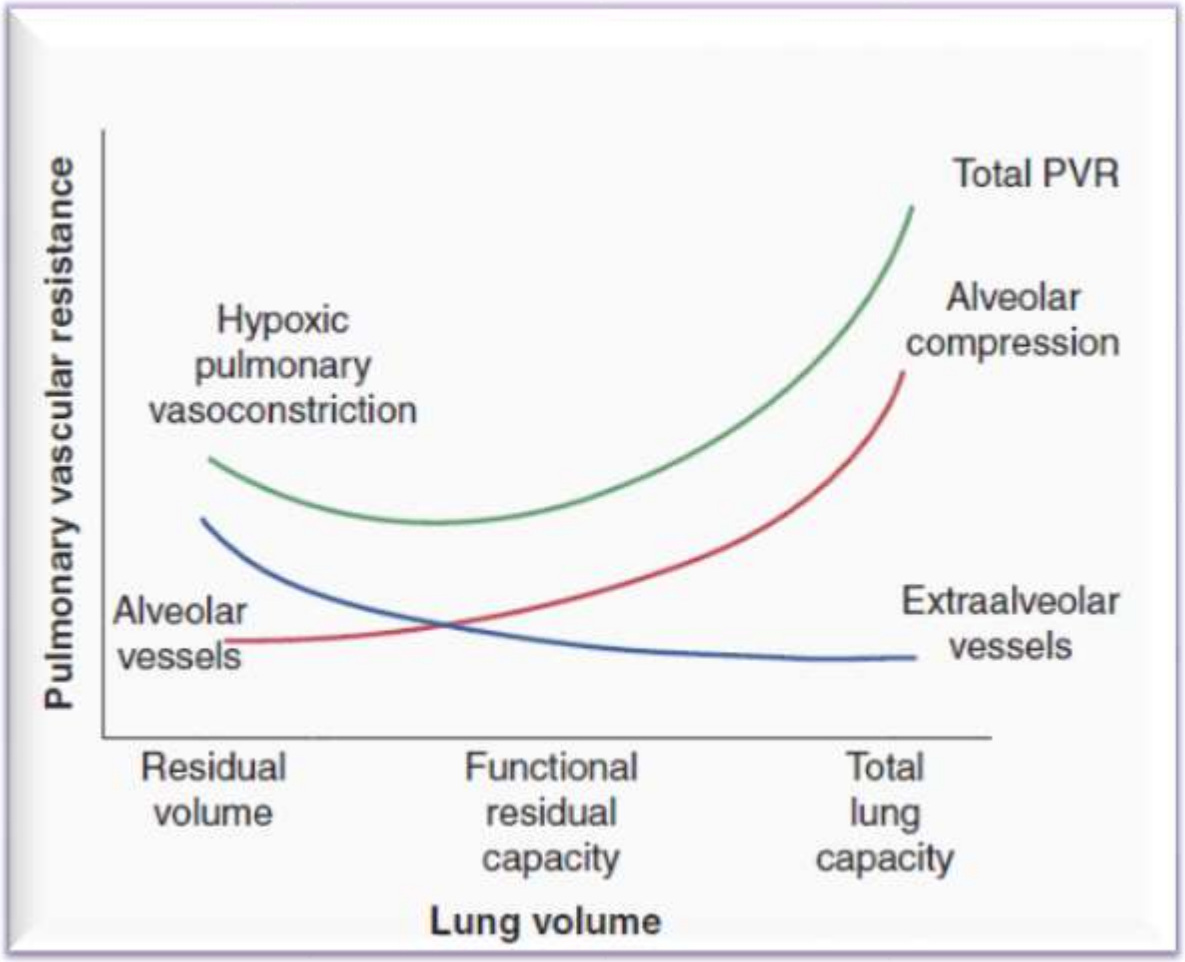
- ↑ ventilation decreases hypoxia and acidosis
- PEEP recruits collapsed alveoli

- Leads to ↓pulm vasomotor tone

NIV, CPAP, Recruitment maneuvers decrease PVR, intubation and mechanical ventilation is not necessary

Brower RG, Gottlieb J, Wise RA, et al. Locus of hypoxic vasoconstriction in isolated ferret lungs. *J Appl Physiol. 1987;63:58–65*

Compression of pulmonary vessels



Net effect on RV afterload

- More effect on alveolar vessels than other mechanisms

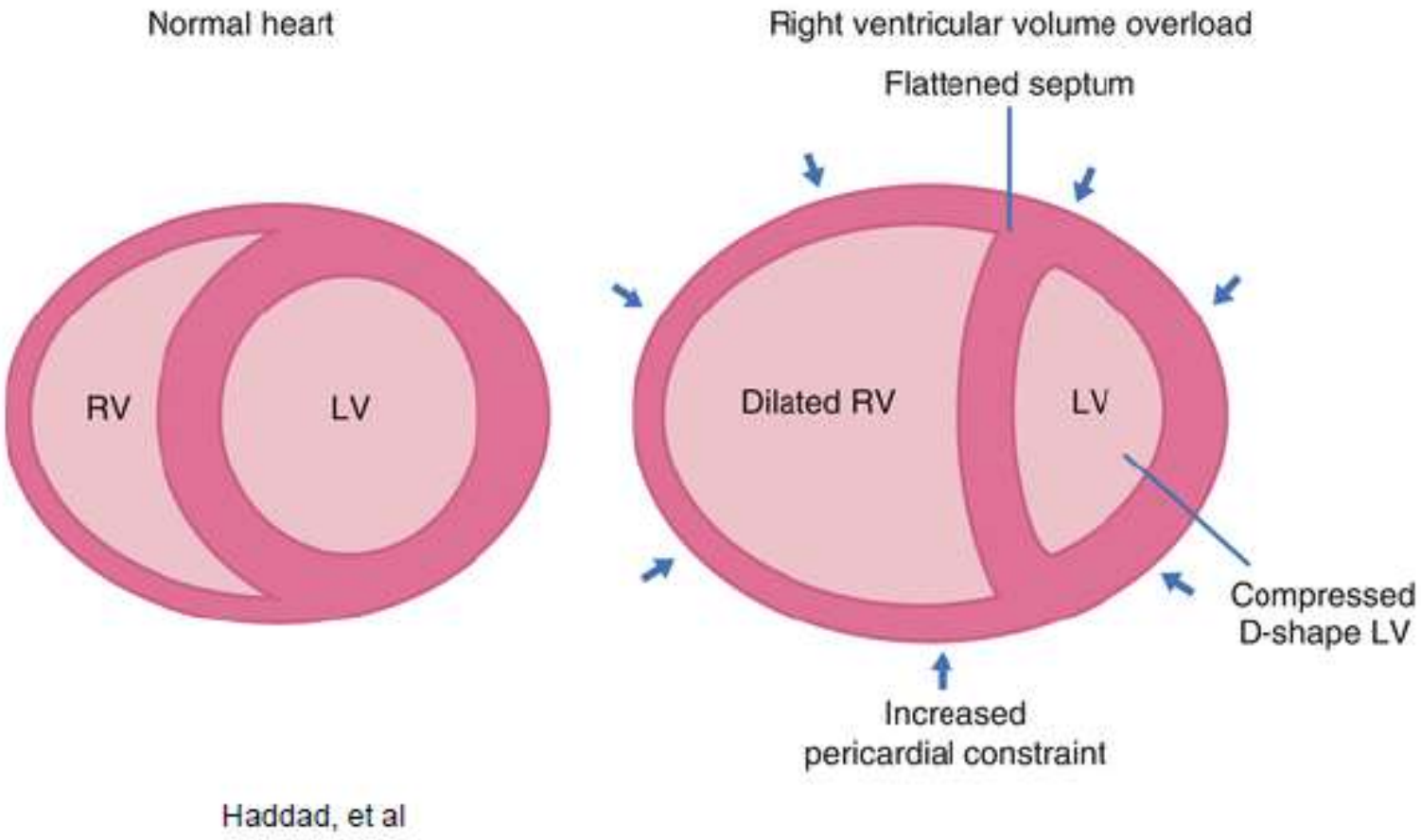
RV afterload increases with positive pressure ventilation

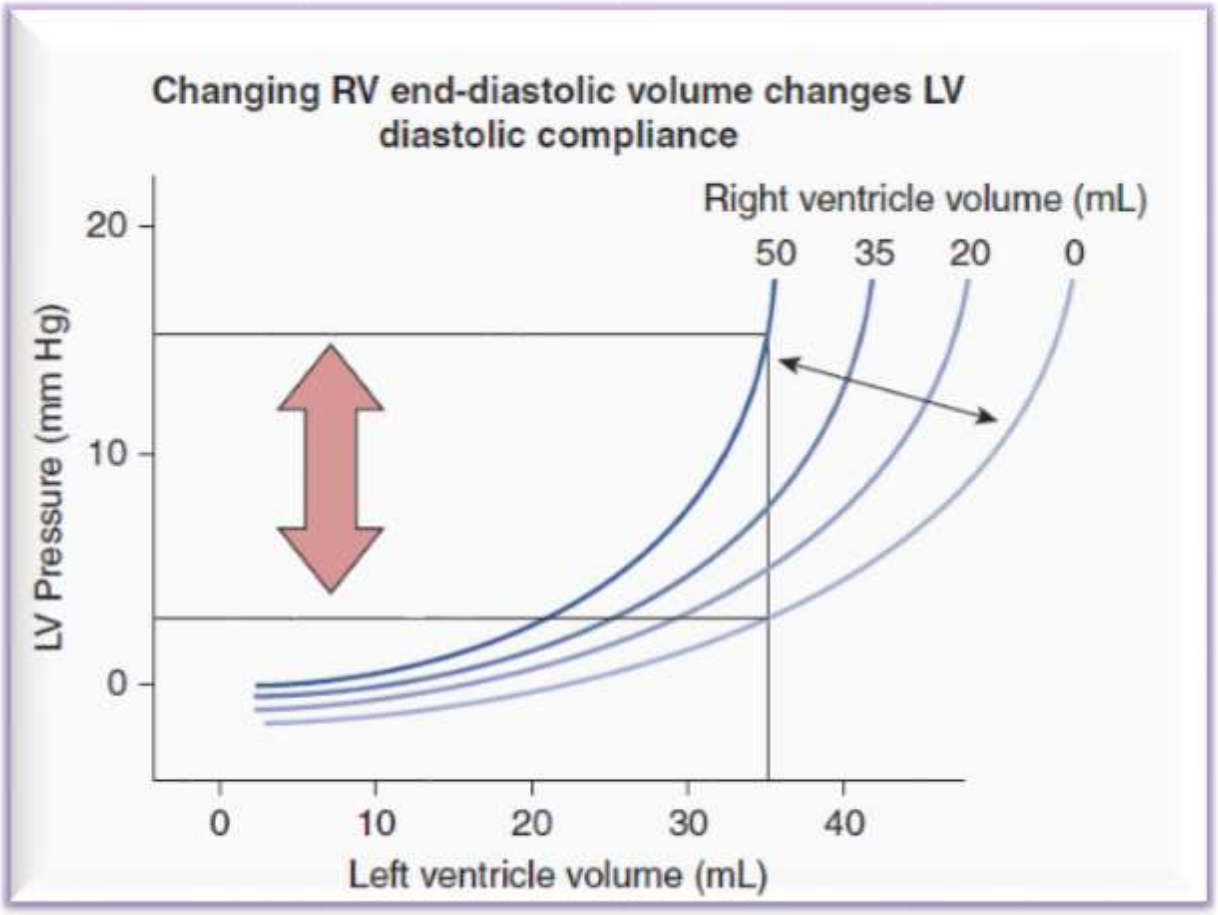
Role in recruitment maneuvres

- High PEEP used
- Significant compression of alveolar vessels
- Sudden ↑se in PVR
- Can cause acute corpulmonale and LV collapse

In borderline RV failure, Recruitment maneuvers are used with caution and is restricted to 10s or less to avoid significant hemodynamic derangements

Ventricular Interdependance




Changing RV end-diastolic volume changes LV diastolic compliance
Right ventricle volume (mL)
50
35
20
0
20
10
0
LV Pressure (mm Hg)
0
10
20
30
40
Left ventricle volume (mL)

VI In PPV?

- Observed that PEEP results in only minimal shift of IV septum to right

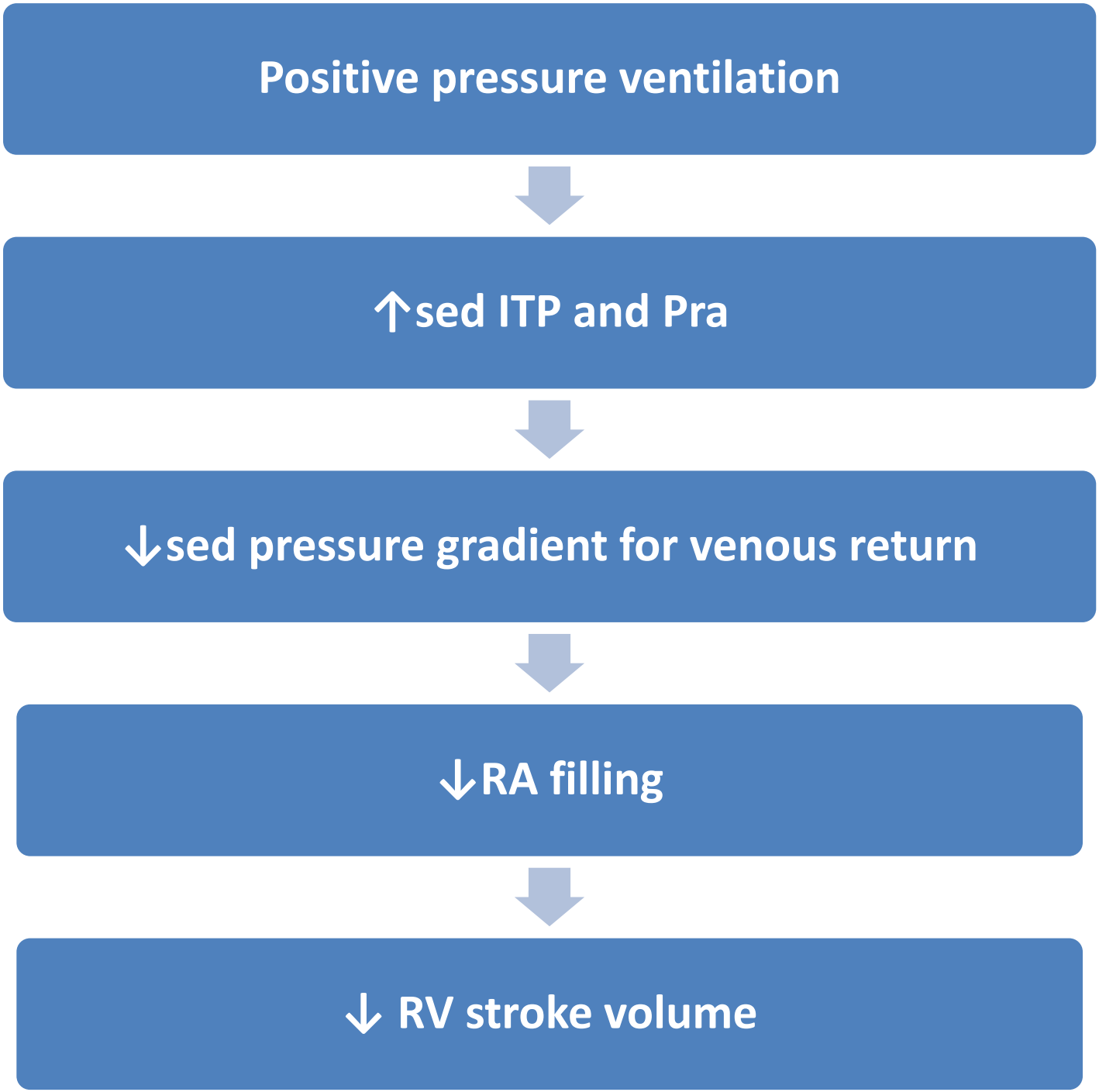
- This VI that occurs during this usual PEEP is found to be small clinically

Jardin F, Farcot JC, Boisante L. Influence of positive end- expiratory pressure on left ventricular performance. *N Engl J Med. 1981;304:* 387–392

Jardin FF, Farcot JC, Gueret P, et al. Echocardiographic evaluation of ventricles during continuous positive pressure breathing. *J Appl Physiol. 1984;56:619–627*

Effects of increased pressure

- Systemic venous return (preload)
- LV afterload
- Myocardial O2 consumption

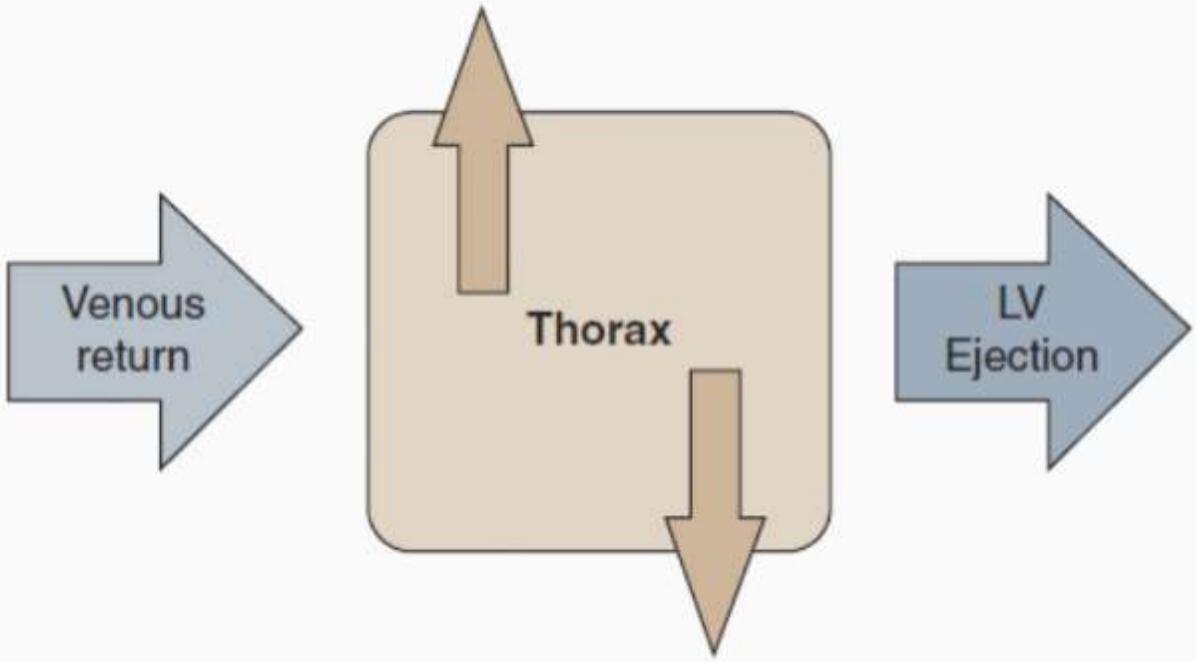

Positive pressure ventilation
↑sed ITP and Pra
↓sed pressure gradient for venous return
↓RA filling
↓ RV stroke volume

Compensatory mechanisms

- Increased sympathetic tone to increase mean systemic pressure
- Diaphragmatic descent causing raised IAP

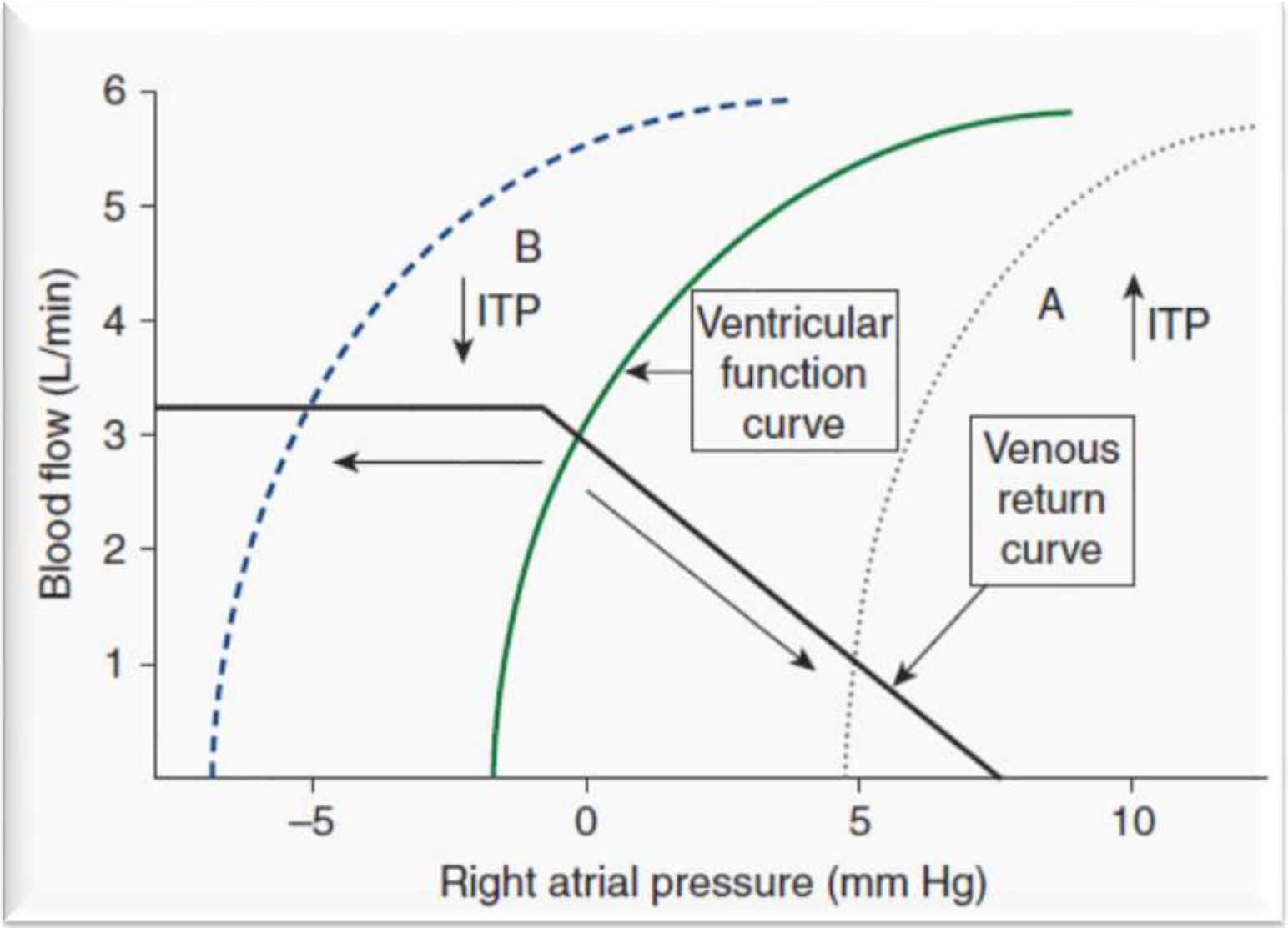
Takata M, Wise RA, Robotham JL. Effects of abdominal pressure on venous return: abdominal vascular zone conditions. *J Appl Physiol.* 1990;69:1961–1972

Hemodynamic effects of changes in intrathoracic pressure



Increasing ITP
Decreases the pressure gradients for venous return and LV ejection

Decreasing ITP
Increases the pressure gradients for venous return and LV ejection


6
5
4
3
2
1
Blood flow (L/min)
B
ITP
A
ITP
Ventricular function curve
Venous return curve
–5
0
5
10
Right atrial pressure (mm Hg)

Can we minimize this? YES!

- Fluid resuscitation
- Prolonging expiratory time
- Low tidal volume
- Low end expiratory pressures

van den Berg P, Jansen JRC, Pinsky MR. The effect of positive-pressure inspiration on venous return in volume loaded post-operative cardiac surgical patients. *J Appl Physiol. 2002;92:1223–1231*

LV afterload

The New England
Journal of Medicine

Volume 301 AUGUST 30, 1979 Number 9

EFFECT OF INTRATHORACIC PRESSURE ON LEFT VENTRICULAR PERFORMANCE

ANDREW J. BUDA, M.D., MICHAEL R. PINSKY, M.D., NEIL B. INGELS, JR., PH.D.,
GEORGE T. DAUGHTERS, II, M.S., EDWARD B. STINSON, M.D., AND EDWIN L. ALDERMAN, M.D.

Abstract Left ventricular dysfunction is common in respiratory-distress syndrome, asthma and obstructive lung disease. To understand the contribution of intrathoracic pressure to this problem, we studied the effects of Valsalva and Müller maneuvers on left ventricular function in eight patients. Implantation of intramyocardial markers permitted beat-by-beat measurement of the velocity of fiber shortening (V_{CF}) and left ventricular volume. During the Müller maneuver, V_{CF} and ejection fraction decreased despite an increase in left ventricular volume and a decline in arterial pressure. In addition, when arterial pressure was corrected for changes in intrapleural pressure during either maneuver it correlated better with left ventricular end-systolic volumes than did uncorrected arterial pressures. These findings suggest that negative intrathoracic pressure affects left ventricular function by increasing left ventricular transmural pressures and thus afterload. We conclude that large intrathoracic-pressure changes, such as those that occur in acute pulmonary disease, can influence cardiac performance. (N Engl J Med 301:453-459, 1979)

LV afterload

- For same tidal volumes, the transmural LV strain decreases with increased ITP

- Raised ITP is known to reduce LV afterload for similar tidal volumes

Buda AJ, Pinsky MR, Ingels NB, et al. Effect of intrathoracic pressure on left ventricular performance. *N Engl J Med. 1979;301:453–459*

Myocardial O2 consumption

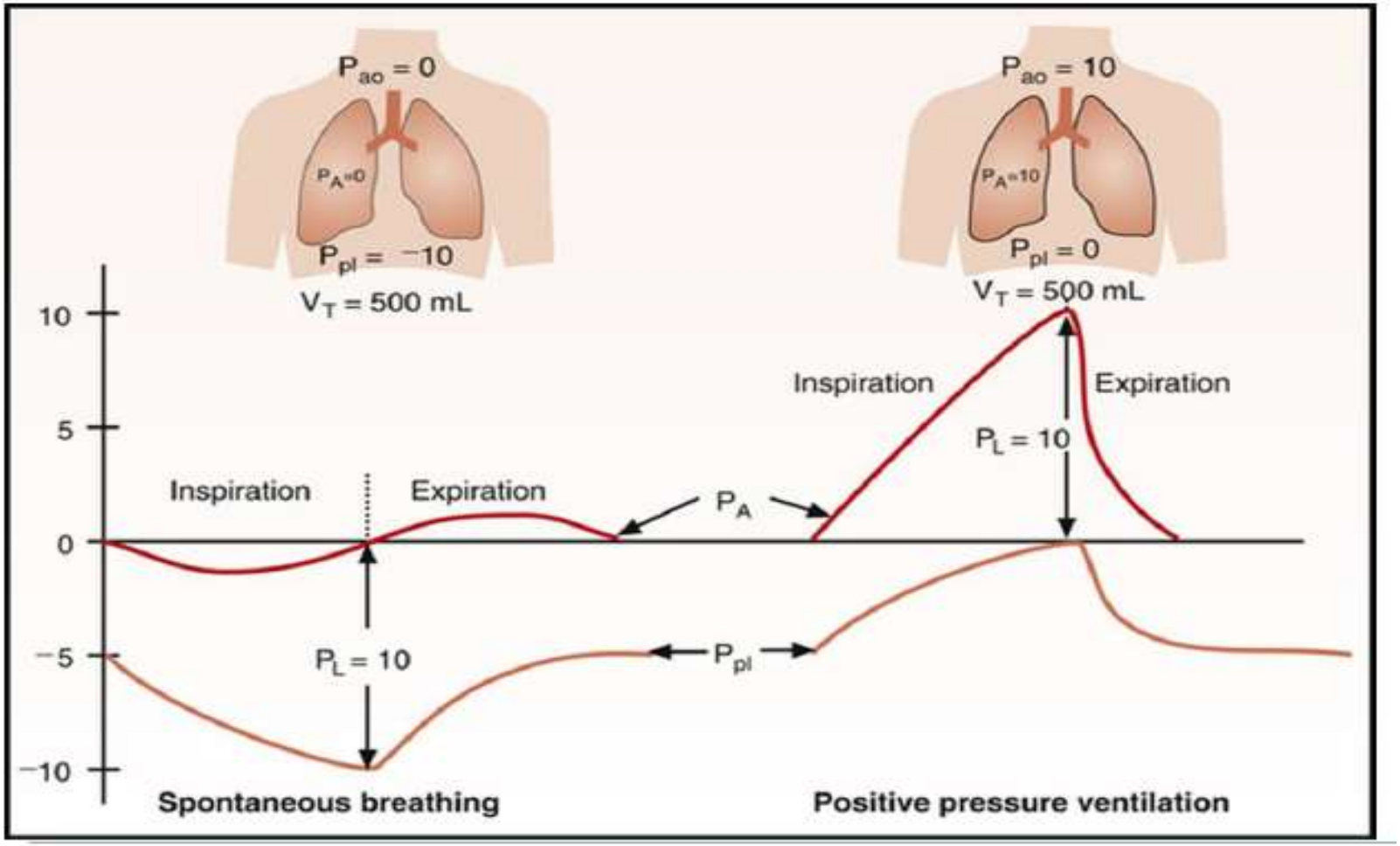
- PPV decreases both LV preload and afterload
- Significant decreased O2 requirements

- Whereas in spontaneous breathing, has shown to increase global O2 demand (irrespective of energy requirements of resp muscles)

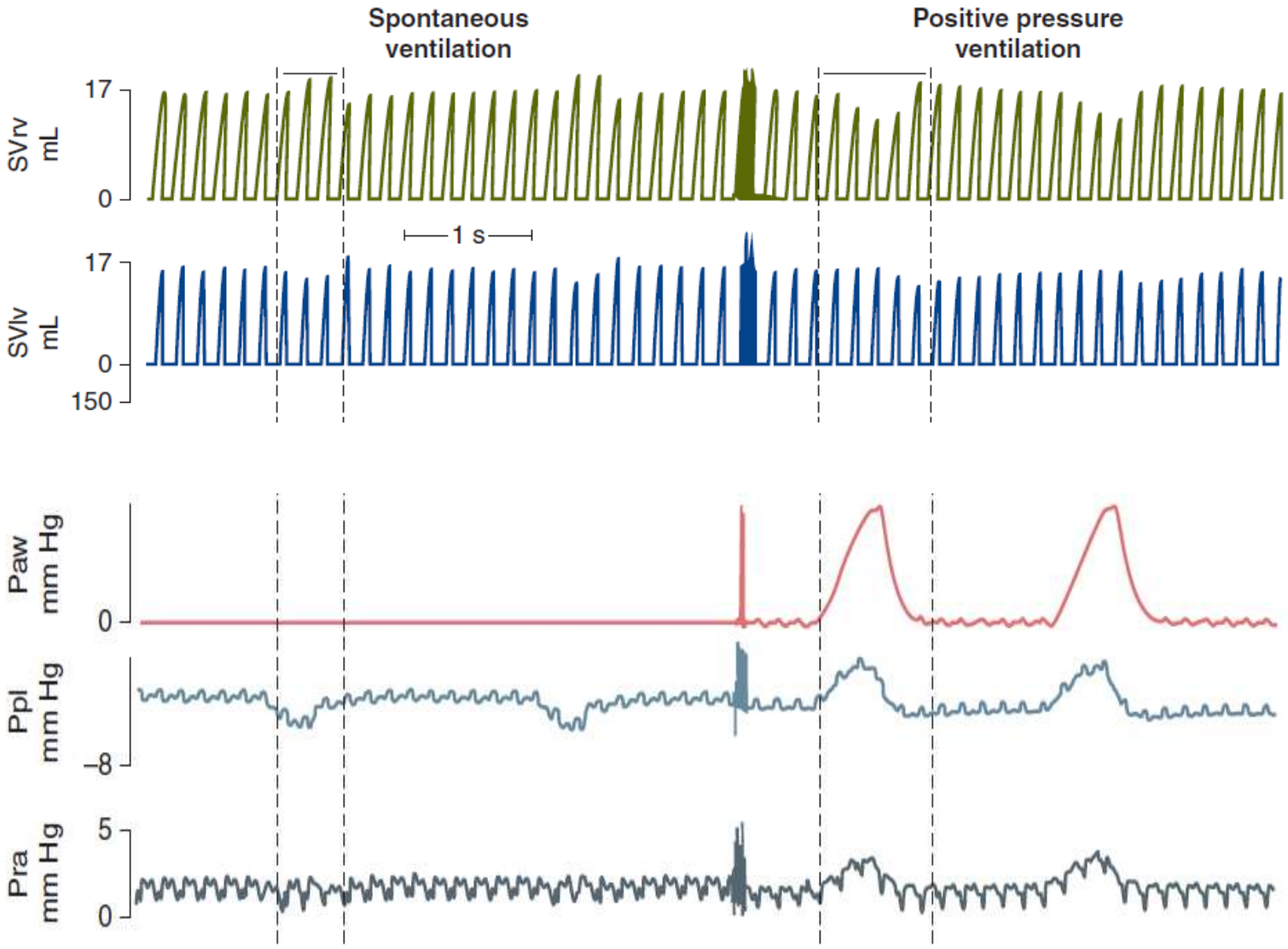
In CHF, increased global LV performance is demonstrated just by applying nasal CPAP and removing negative swings

Buda AJ, Pinsky MR, Ingels NB, et al. Effect of intrathoracic pressure on left ventricular performance. *N Engl J Med. 1979;301:453–459*

Spontaneous vs Positive pressure ventilation

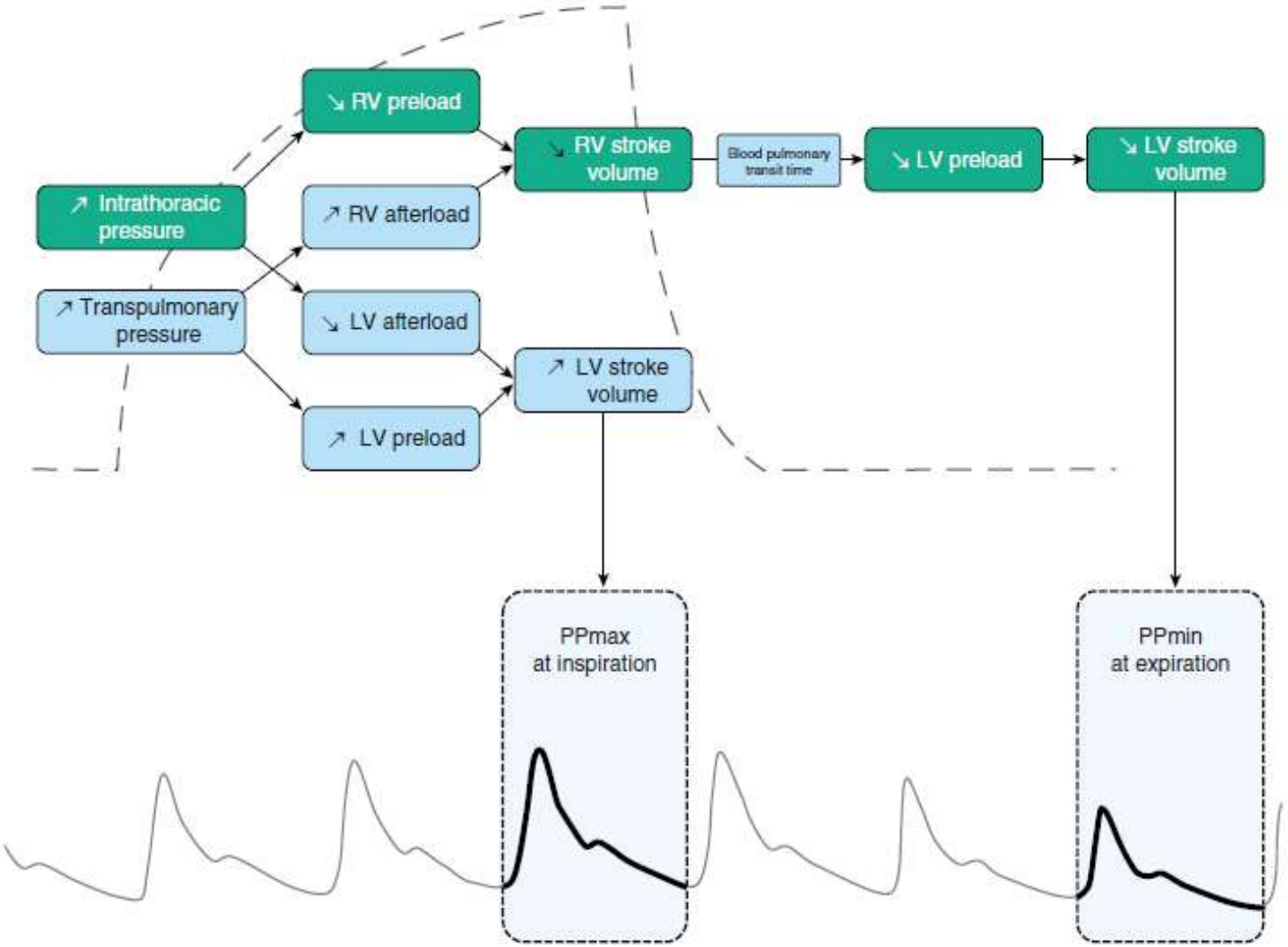


Spontaneous breathing	Parameters	Positive pressure ventilation
↑	lung volume	↑
↓	ITP	↑
↓	Pra	↑
+	Addition of PEEP	++
↑	Venous return	↓
-	RV afterload	↑
↓	Ventricular interdependence LV compliance	-
↑	Myocardial O2 consumption	↓
-	LV afterload	↓


Spontaneous ventilation
Positive pressure ventilation
SVrv mL
17
0
1 s
SVlv mL
17
0
150
Paw mm Hg
0
Ppl mm Hg
–8
Pra mm Hg
5
0

ROLE OF CARDIOPULMONARY INTERACTION DURING INITIATION OF MECHANICAL VENTILATION

To summarize



- Above factors plus
- Blunting of sympathetic responses by drugs used to allow intubation
- Causes hypotension in normovolemic pts

- Not just the effect of drugs (tracheostomised pts)

Does it change among diff modes?

- Differential effect of lung volume and ITP only
- When Vt and PEEP are the same, hemodynamic changes are similar among all the modes of ventilation
- Square flow waveform : ↓sed LV stroke volume

Lessard MR, Guerot E, Lorini H, et al. Effects of pressure-controlled with different I:E ratios versus volume-controlled ventilation on respiratory mechanics, gas exchange and hemodynamics in patients with adult respiratory distress syndrome. *Anesthesiology. 1994;80:* 983–991

ROLE OF CARDIOPULMONARY INTERACTION IN WEANING PROCESS

Changes occurring weaning process

- Shift from PPV to spontaneous breathing (reductions in ITP)
- ↑sed venous return (preload)
- ↑sed LV afterload
- ↓sed LV compliance
- ↑sed global myocardial O2 demand

RV and LV stress test

Clinical applications

- Non compliant RV : acute cor pulmonale, CV collapse
- LV ischemia: ADHF, LVF
- Also seen in severe obstructive lung diseases

During weaning process, pts are carefully observed for signs of heart failure

Lemaire F, Teboul JL, Cinoti L, et al. Acute left ventricular dysfunction during unsuccessful weaning from mechanical ventilation. *Anesthesiology. 1988;69:171–179*

Richard C, Teboul JL, Archambaud F, et al. Left ventricular dysfunction during weaning in patients with chronic obstructive pulmonary disease. *Intensive Care Med. 1994;20:171–172*

Can we prevent?

- In pts with heart failure or ischemia
- Cardiac performance increased by PPV
- Reverse myocardial ischemia

In such patients, preemptive NIV has a role in preventing reintubation and increasing cardiac performance

Rasanen J, et al. Acute myocardial infarction complicated by respiratory failure. The effects of mechanical ventilation. *Chest. 1984;85:21–28.*
Rasanen J, et al. Acute myocardial infarction complicated by left ventricular dysfunction and respiratory failure. The effects of continuous positive airway pressure. *Chest.* 1985;87:156–162

ROLE OF VENTILATION TO DEFINE CARDIAC PERFORMANCE

- LV ejection can be assessed over many end diastolic volumes

- On PPV, Systolic BP monitoring is done
- If SBP ↑ses : volume overload / heart failure
- If SBP ↓ses : hypovolemia (more likely to respond to volume expansion)

Benes J, Giglio M, Brienza N, Michard F. The effects of goal-directed fluid therapy based on dynamic parameters on post-surgical outcome: a meta-analysis of randomized controlled trials. Crit Care 2014;18:584

Its significance?

- Early identification of pts who respond to fluids
- Infusing fluids in others will worsen their cardiovascular status
- Can delay appropriate therapy, if proper subgroup is not identified

Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest. 2002;121:2000–2008*

PARAMETERS FOR HEMODYNAMIC MONITORING

- All these are based on principles of cardiopulmonary interaction

Traditional techniques

- Pulmonary artery catheterization
- Central venous pressure monitoring

Why new techniques?

- Both CVP and PAC poor predictive value for predicting fluid responsiveness
- affected by a number of other physiologic derangements
- Complications: invasiveness
- Data interpretation is difficult

Marik PE, et al. Does central venous pressure predict fluid responsiveness?
A systematic review of the literature and the tale of seven mares. Chest 2008; 134:172.
Michard F, et al. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence.
Chest 2002; 121:2000.

Pulse pressure variation(PPV)

- systolic – diastolic arterial blood pressure varies with respiration
- $PPV = 100 \times (PP_{max} - PP_{min})/PP_{mean}$

- PPV of at least 13 to 15 percent is strongly associated with volume responsiveness
- Sensitivity and specificity of 88%

Marik PE, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. Crit Care Med 2009; 37:2642.

- Accurate in mechanically ventilated(paralysed) pt with VT>7-8ml/kg, when arterial catheter used
- Sinus rhythm

Michard F, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000; 162:134.

Arterial Pulse Pressure Variation with Mechanical Ventilation

Jean-Louis Teboul[1], Xavier Monnet[1], Denis Chemla[2], and Frédéric Michard[3]

[1]Medical Intensive Care Unit, Bicetre Hospital, and [2]Department of Physiology, Bicetre Hospital, Paris-South University Hospitals, Inserm UMR_S999, Paris-South University, Le Kremlin-Bicêtre, France; and [3]MiCo, Denens, Switzerland

Abstract

Fluid administration leads to a significant increase in cardiac output in only half of ICU patients. This has led to the concept of assessing fluid responsiveness before infusing fluid. Pulse pressure variation (PPV), which quantifies the changes in arterial pulse pressure during mechanical ventilation, is one of the dynamic variables that can predict fluid responsiveness. The underlying hypothesis is that large respiratory changes in left ventricular stroke volume, and thus pulse pressure, occur in cases of biventricular preload responsiveness. Several studies showed that PPV accurately predicts fluid responsiveness when patients are under controlled mechanical ventilation. Nevertheless, in many conditions encountered in the ICU, the interpretation of PPV is unreliable (spontaneous breathing, cardiac arrhythmias) or doubtful (low V_T). To overcome some of these limitations, researchers have proposed using simple tests such as the V_T challenge to evaluate the dynamic response of PPV. The applicability of PPV is higher in the operating room setting, where fluid strategies made on the basis of PPV improve postoperative outcomes. In medical critically ill patients, although no randomized controlled trial has compared PPV-based fluid management with standard care, the Surviving Sepsis Campaign guidelines recommend using fluid responsiveness indices, including PPV, whenever applicable. In conclusion, PPV is useful for managing fluid therapy under specific conditions where it is reliable. The kinetics of PPV during diagnostic or therapeutic tests is also helpful for fluid management.

Keywords: fluid responsiveness; cardiac preload; heart–lung interaction; cardiac output

Stroke volume variation (SVV)

- linearly related to pulse pressure
- >10% is associated with fluid responsiveness

- $SVV = 100 \times (SV_{max} - SV_{min})/SV_{mean}$

Jacques D, Bendjelid K, Duperret S, et al. Pulse pressure variation and stroke volume variation during increased intra-abdominal pressure: an experimental study. Crit Care 2011; 15:R33.

- Accurate when arterial catheter is used
- Has same limitations of PPV

- sensitivity and specificity of 94% with threshold value of 10%

Biais M, et al. Uncalibrated pulse contour-derived stroke volume variation predicts fluid responsiveness in mechanically ventilated patients undergoing liver transplantation. Br J Anaesth 2008; 101:761.

Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: A systematic review of the literature*

Paul E. Marik, MD, FCCM; Rodrigo Cavallazzi, MD; Tajender Vasu, MD; Amyn Hirani, MD

Objectives: A systematic review of the literature to determine the ability of dynamic changes in arterial waveform-derived variables to predict fluid responsiveness and compare these with static indices of fluid responsiveness. The assessment of a patient's intravascular volume is one of the most difficult tasks in critical care medicine. Conventional static hemodynamic variables have proven unreliable as predictors of volume responsiveness. Dynamic changes in systolic pressure, pulse pressure, and stroke volume in patients undergoing mechanical ventilation have emerged as useful techniques to assess volume responsiveness.

Data Sources: MEDLINE, EMBASE, Cochrane Register of Controlled Trials and citation review of relevant primary and review articles.

Study Selection: Clinical studies that evaluated the association between stroke volume variation, pulse pressure variation, and/or stroke volume variation and the change in stroke volume/cardiac index after a fluid or positive end-expiratory pressure challenge.

Data Extraction and Synthesis: Data were abstracted on study design, study size, study setting, patient population, and the correlation coefficient and/or receiver operating characteristic between the baseline systolic pressure variation, stroke volume variation, and/or pulse pressure variation and the change in stroke index/cardiac index after a fluid challenge. When reported, the receiver operating characteristic of the central venous pressure, global end-diastolic volume index, and left ventricular end-diastolic area index were also recorded. Meta-analytic techniques were used to summarize the data. Twenty-nine studies (which enrolled 685 patients) met our inclusion criteria. Overall, 56% of patients responded to a fluid challenge. The pooled correlation coefficients between the baseline pulse pressure variation, stroke volume variation, systolic pressure variation, and the change in stroke/cardiac index were 0.78, 0.72, and 0.72, respectively. The area under the receiver operating characteristic curves were 0.94, 0.84, and 0.86, respectively, compared with 0.55 for the central venous pressure, 0.56 for the global end-diastolic volume index, and 0.64 for the left ventricular end-diastolic area index. The mean threshold values were 12.5 ± 1.6% for the pulse pressure variation and 11.6 ± 1.9% for the stroke volume variation. The sensitivity, specificity, and diagnostic odds ratio were 0.89, 0.88, and 59.86 for the pulse pressure variation and 0.82, 0.86, and 27.34 for the stroke volume variation, respectively.

Conclusions: Dynamic changes of arterial waveform-derived variables during mechanical ventilation are highly accurate in predicting volume responsiveness in critically ill patients with an accuracy greater than that of traditional static indices of volume responsiveness. This technique, however, is limited to patients who receive controlled ventilation and who are not breathing spontaneously. (Crit Care Med 2009; 37:2642–2647)

KEY WORDS: arterial waveform; pulse pressure variation; stroke volume variation; pulse contour analysis; heart-lung interactions; fluid responsiveness; preload; stroke volume; fluid therapy; hemodynamic monitoring; critical care; systematic review; meta-analysis

- Both **LV stroke volume variation** and **arterial pulse pressure variation** during phases of resp cycle of ventilation are both sensitive and specific markers of fluid responsiveness

Marik PE, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med.* 37:2642–2647, 2009.

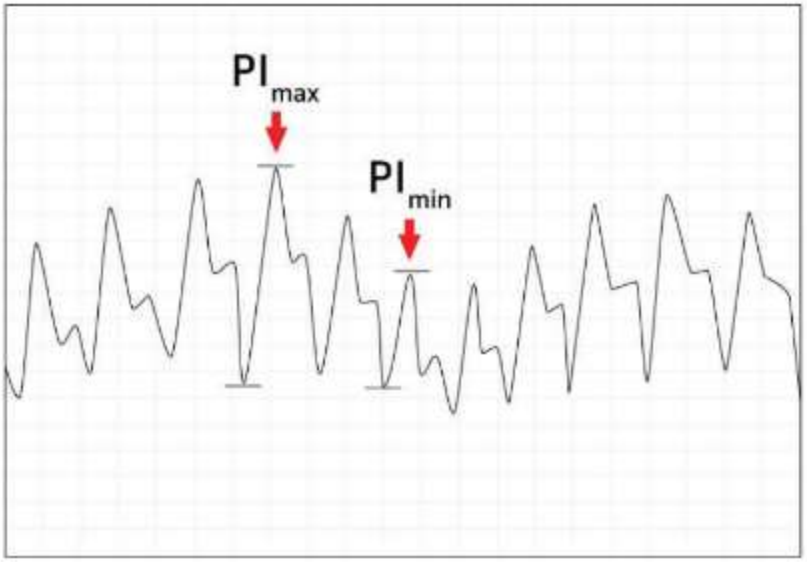
Pleth variability index (PVI)

- Pulse oximetric waveform variation w.r.t. respiration
- measure of the dynamic changes in the Perfusion Index (Pi) that occur during one or more complete respiratory cycles

- Predict fluid responsiveness

Cannesson M, et al. Pleth variability index to monitor the respiratory variations in the pulse oximeter plethysmographic waveform amplitude and predict fluid responsiveness in the operating theatre. Br J Anaesth 2008; 101:200.

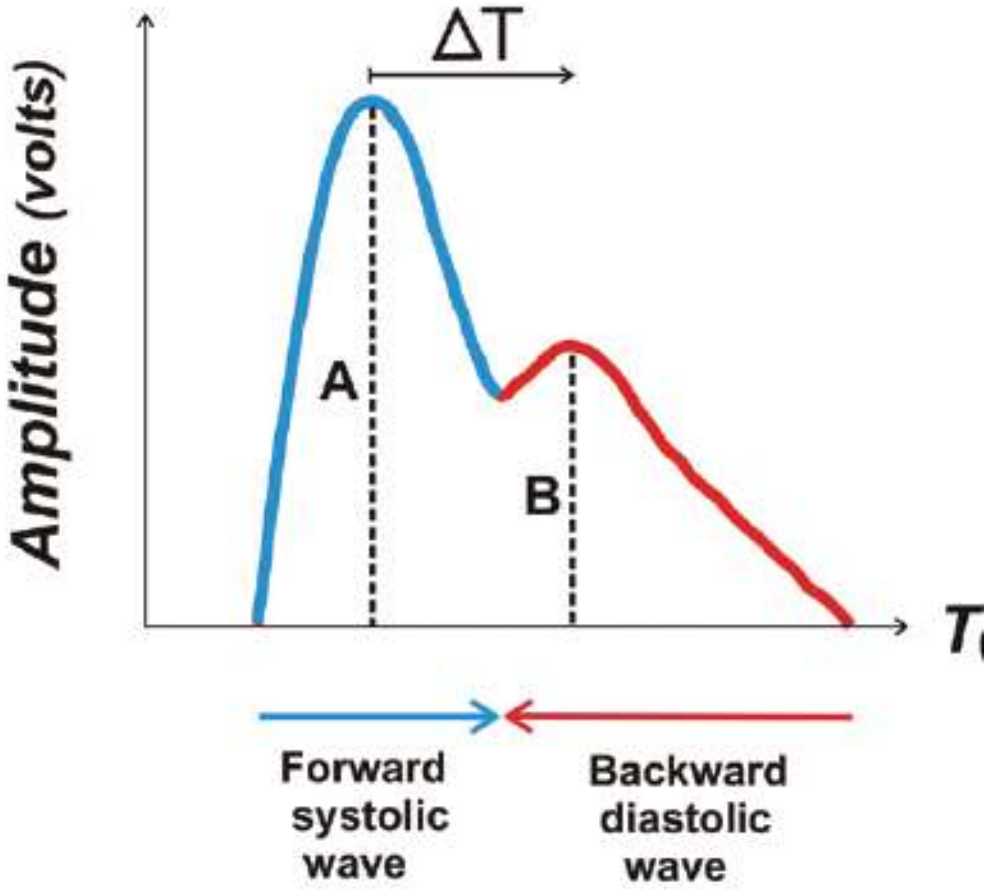
Calculation



$$PVI = \frac{PI_{max} - PI_{min}}{PI_{max}} \times 100$$

- Expressed as a percentage

LV Ejection Time and Pre Ejection Period



Middleton PM, Chan GS, O'Lone E, Steel E, Carroll R, Celler BG, Lovell NH. Changes in left ventricular ejection time and pulse transit time derived from finger photoplethysmogram and electrocardiogram during moderate haemorrhage. Clin Physiol Funct Imaging 2009;29:163–9

Provocative maneuvers

- Fluid challenge (500ml over 10 min)

- Passive leg raise test (45 degrees for one min)
  - sensitivity of 86%, specificity of 92 %

Cherpanath TG, Hirsch A, Geerts BF, et al. Predicting Fluid Responsiveness by Passive Leg Raising: A Systematic Review and Meta-Analysis of 23 Clinical Trials. Crit Care Med 2016; 44:981.

What is Fluid responsiveness?

- Poorly defined
- 10 or 15% increase in Cardiac Output

- reduction in SVV and PPV when provocative maneuvers are used
- Volume responsiveness does not equate to the need for fluids, it only identifies the ability of the heart to increase stroke volume if given fluids

Marik PE, Monnet X, Teboul JL. Hemodynamic parameters to guide fluid therapy. Ann Intensive Care 2011; 1:1.

IVC Diameter

- during positive pressure ventilation, increased intra thoracic pressure pushes blood from the heart into the vena cava, leading to distention of the vessel

- magnitude of these changes has been proposed to correlate with intravascular volume status and fluid responsiveness

Barbier C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. Intensive Care Med 2004; 30:1740.

- Collapse of IVC diameter with inspiration of **12 to 18% :** associated with fluid responsiveness in mechanically ventilated patients

Feissel M, Michard F, Faller JP, Teboul JL. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. Intensive Care Med 2004; 30:1834.

Intensive Care Med (2004) 30:1834–1837
DOI 10.1007/s00134-004-2233-5

BRIEF REPORT

Marc Feissel
Frédéric Michard
Jean-Pierre Faller
Jean-Louis Teboul

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

Abstract *Objective:* To investigate whether the respiratory variation in inferior vena cava diameter (ΔD_{IVC}) could be related to fluid responsiveness in mechanically ventilated patients. *Design:* Prospective clinical study. *Setting:* Medical ICU of a non-university hospital. *Patients:* Mechanically ventilated patients with septic shock (n=39). *Interventions:* Volume loading with 8 mL/kg of 6% hydroxyethylstarch over 20 min. *Measurements and results:* Cardiac output and ΔD_{IVC} were assessed by echography before and immediately after the standardized volume load. Volume loading induced an increase in cardiac output from 5.7±2.0 to 6.4±1.9 L/min (P<0.001) and a decrease in ΔD_{IVC} from 13.8±13.6 vs 5.2±5.8% (P<0.001). Sixteen patients responded to volume loading by an increase in cardiac output ≥15% (responders). Before volume loading, the ΔD_{IVC} was greater in responders than in non-responders (25±15 vs 6±4%, P<0.001), closely correlated with the increase in cardiac output (r=0.82, P<0.001), and a 12% ΔD_{IVC} cut-off value allowed identification of responders with positive and negative predictive values of 93% and 92%, respectively. *Conclusion:* Analysis of ΔD_{IVC} is a simple and non-invasive method to detect fluid responsiveness in mechanically ventilated patients with septic shock.

Keywords Fluid responsiveness · Echography · Inferior vena cava · Septic shock · Mechanical ventilation

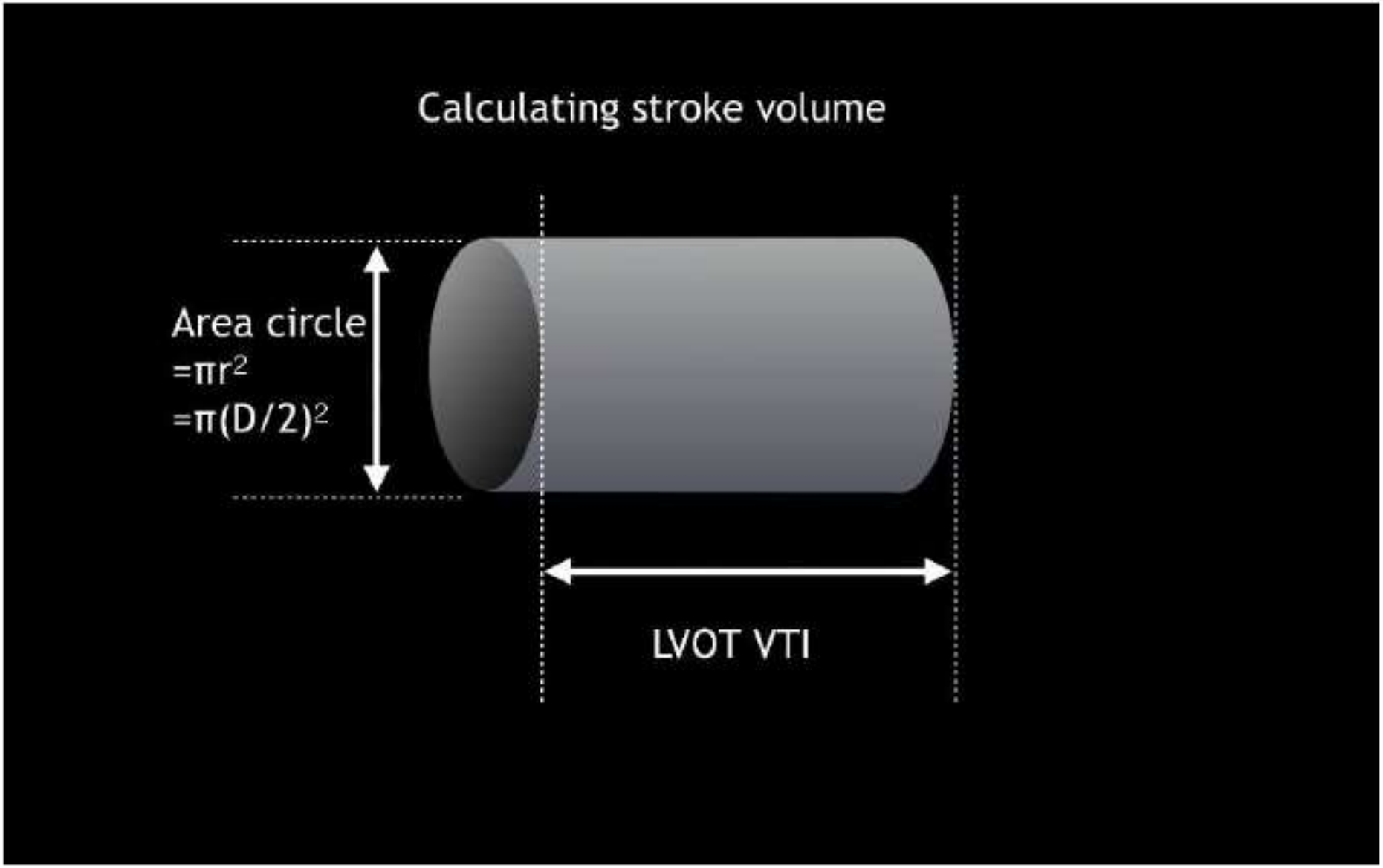
- Operator dependent
- Requires training
- other heart lesions may be confounding

Velocity time integral (VTI)

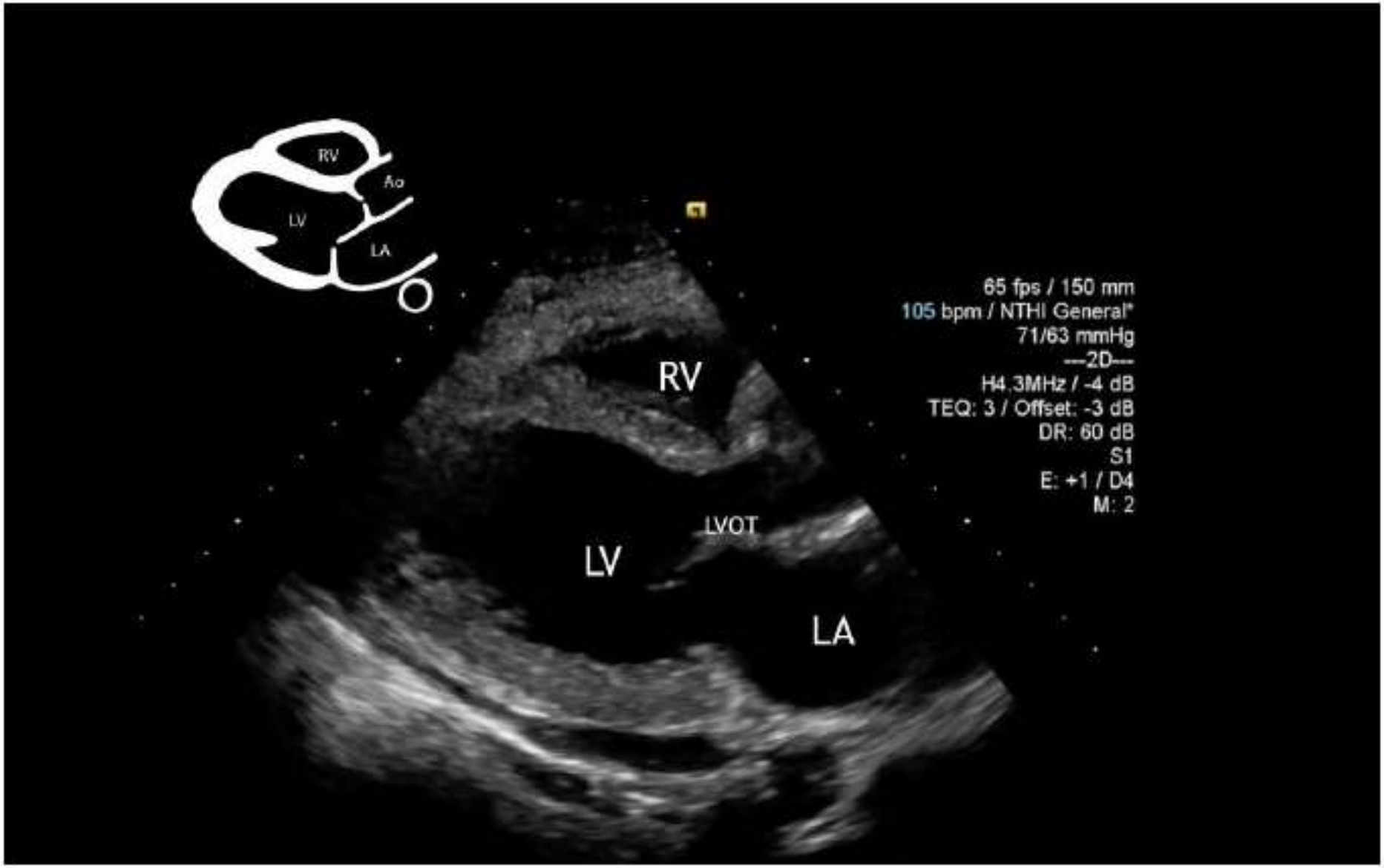
- Commonly done at the level of LVOT
- Using doppler echocardiography : pulsed wave doppler

- Can be used in our RICU!
- Fairly reliable in predicting cardiac output

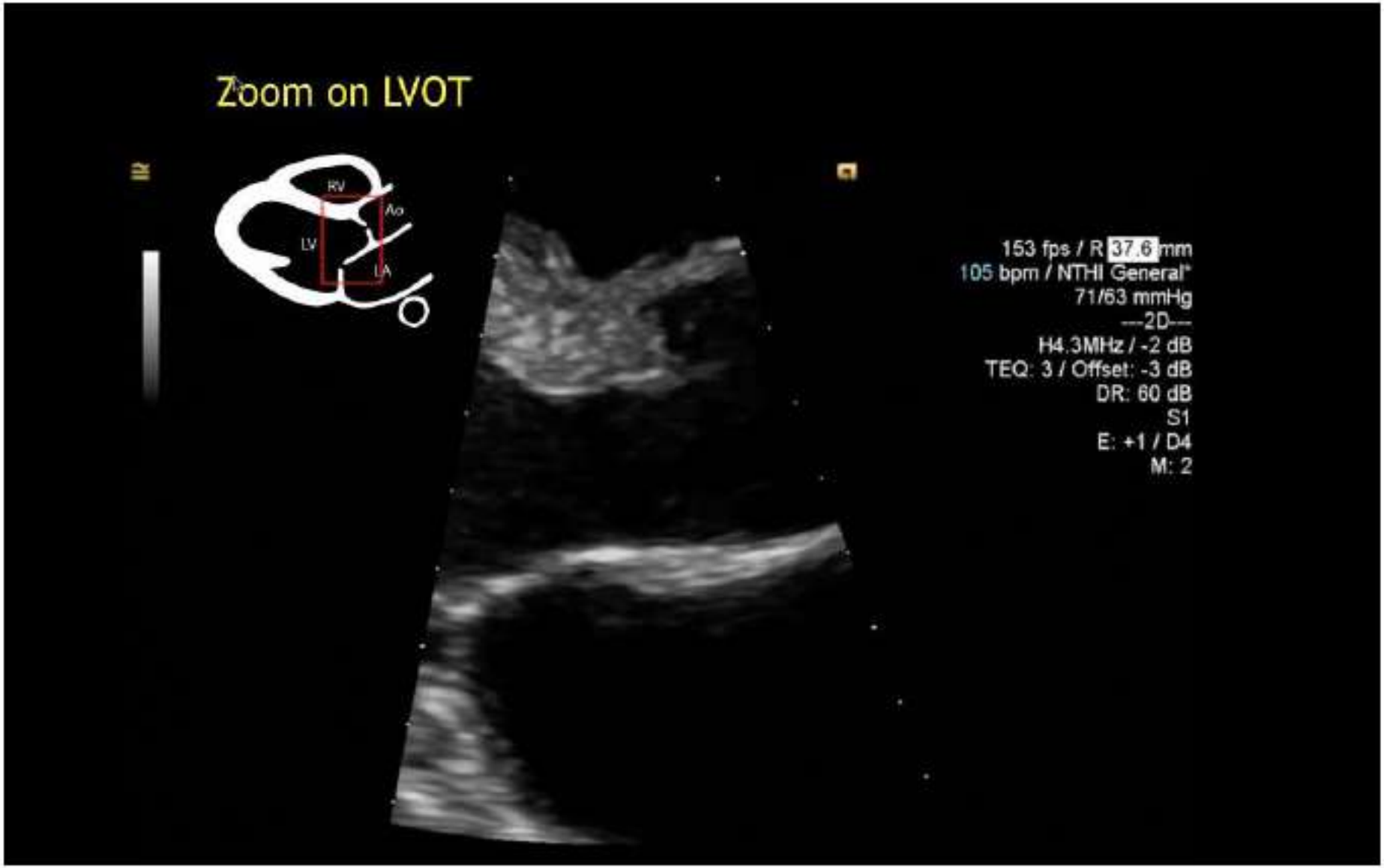
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



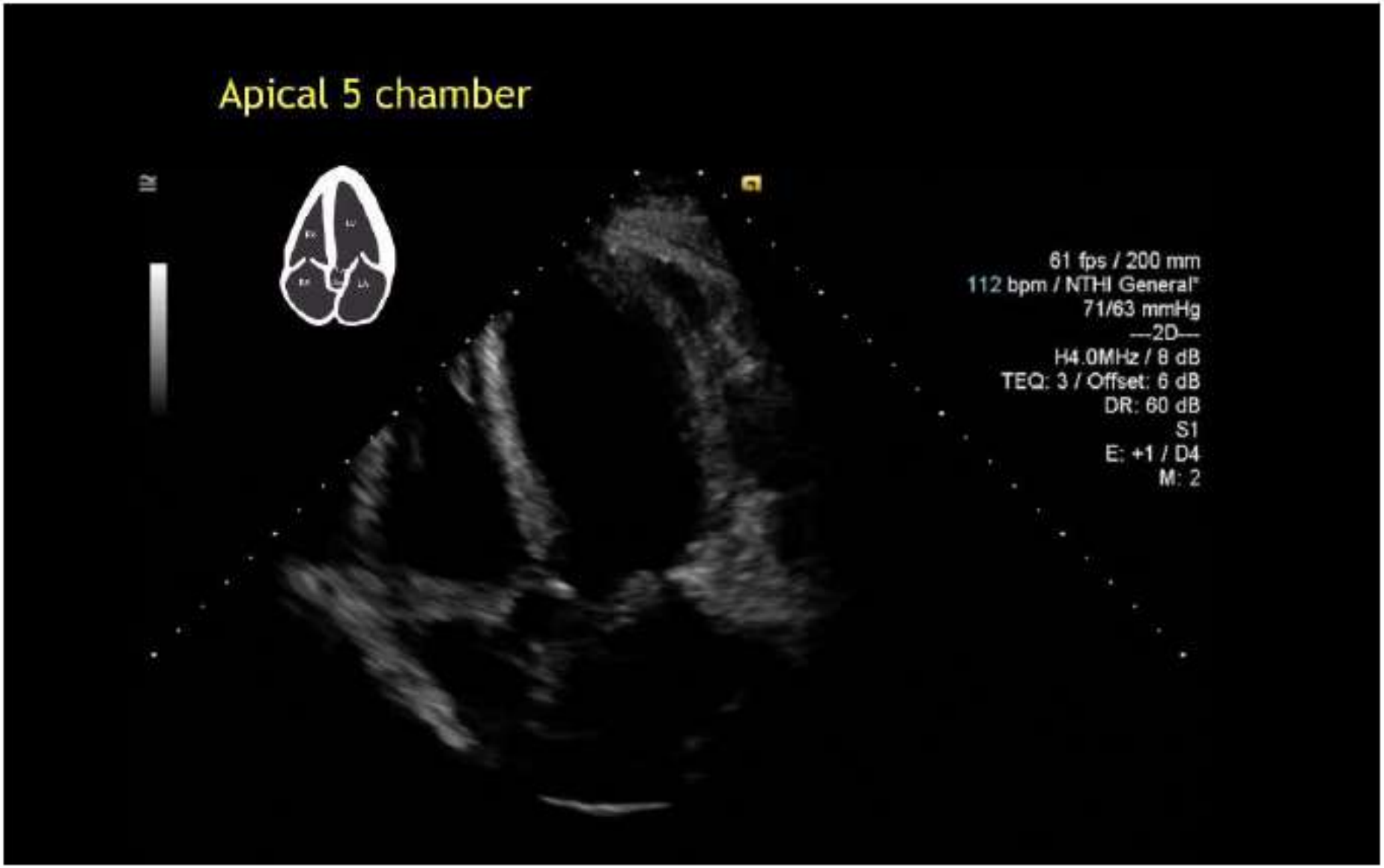
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



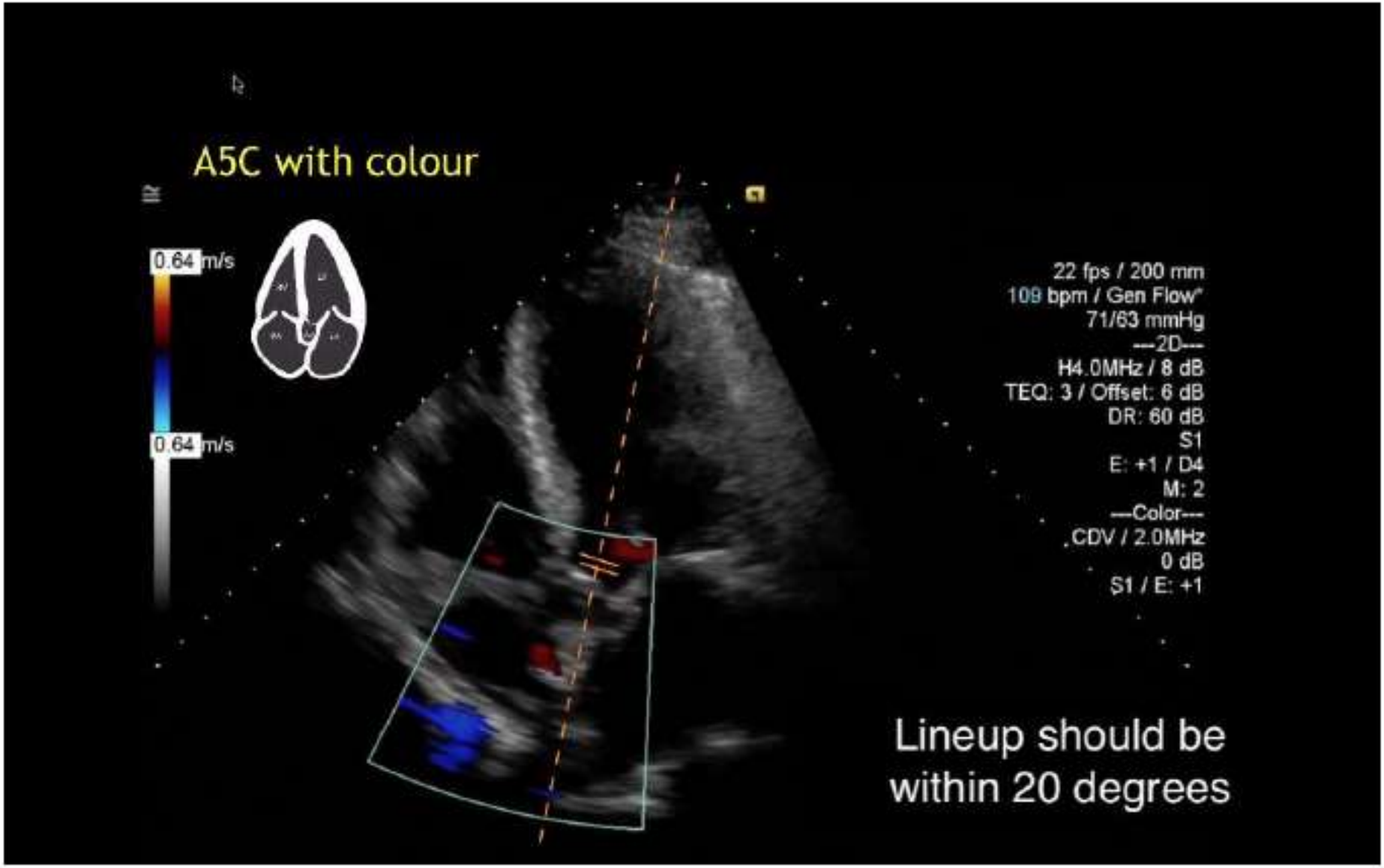
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



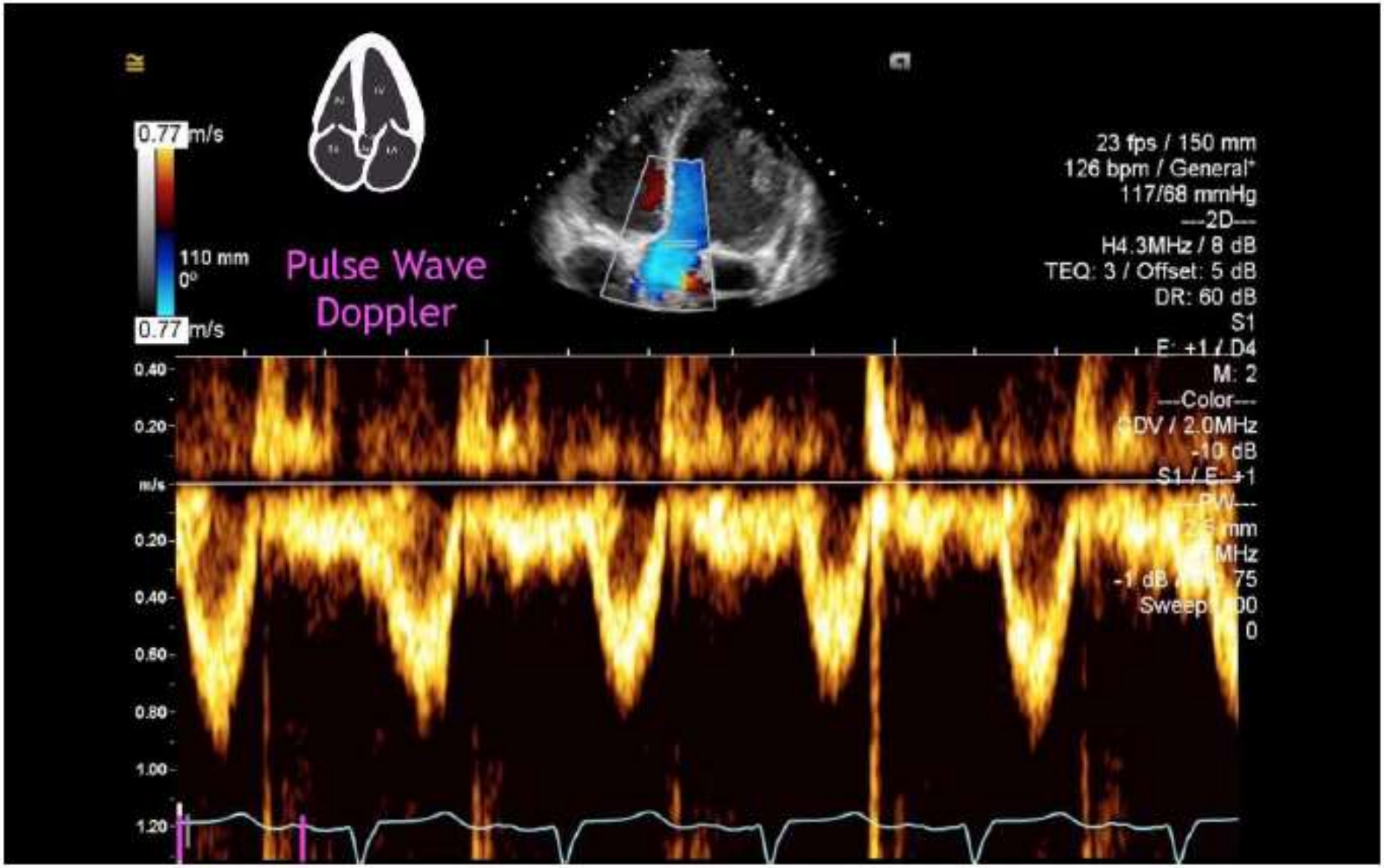
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



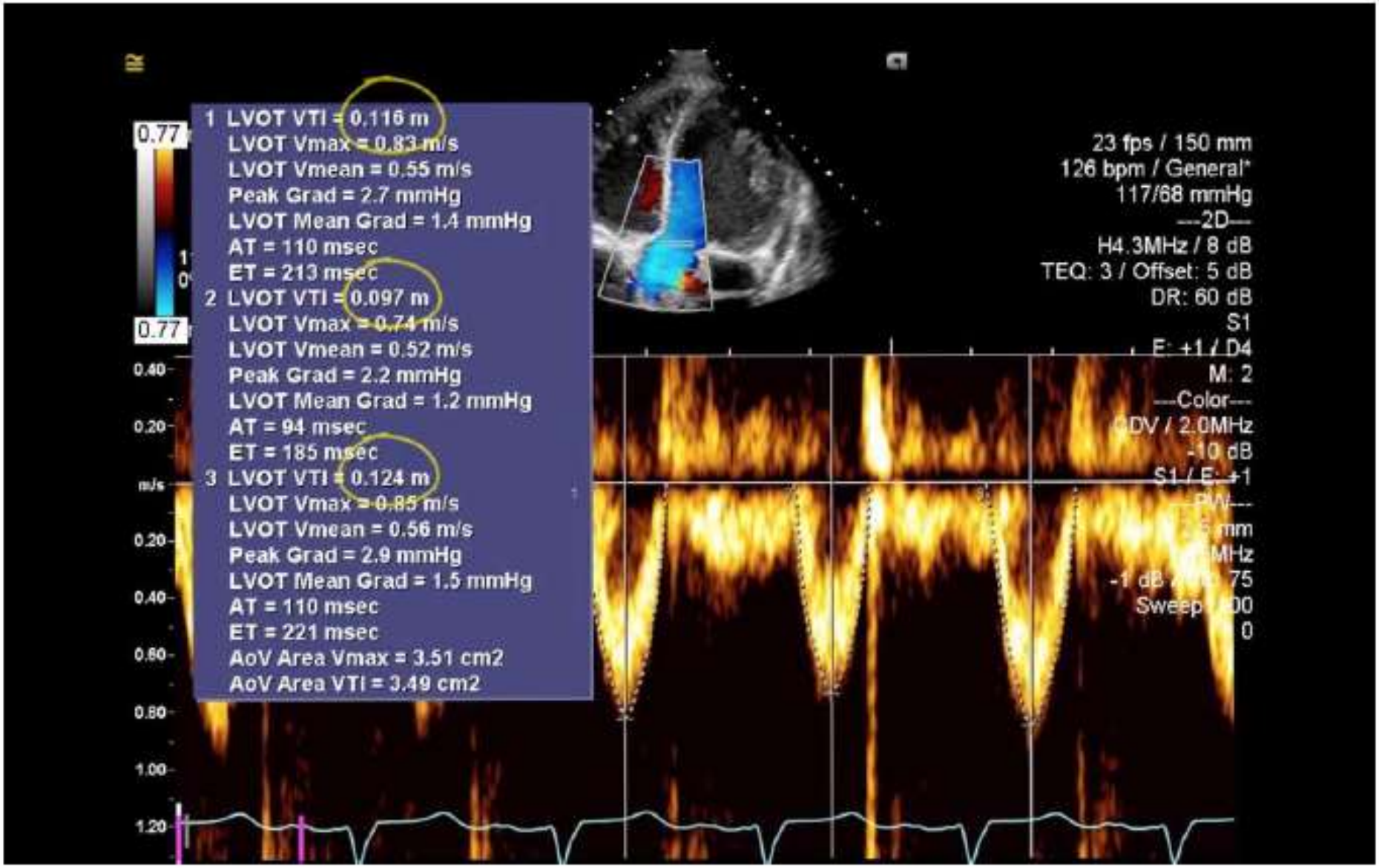
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



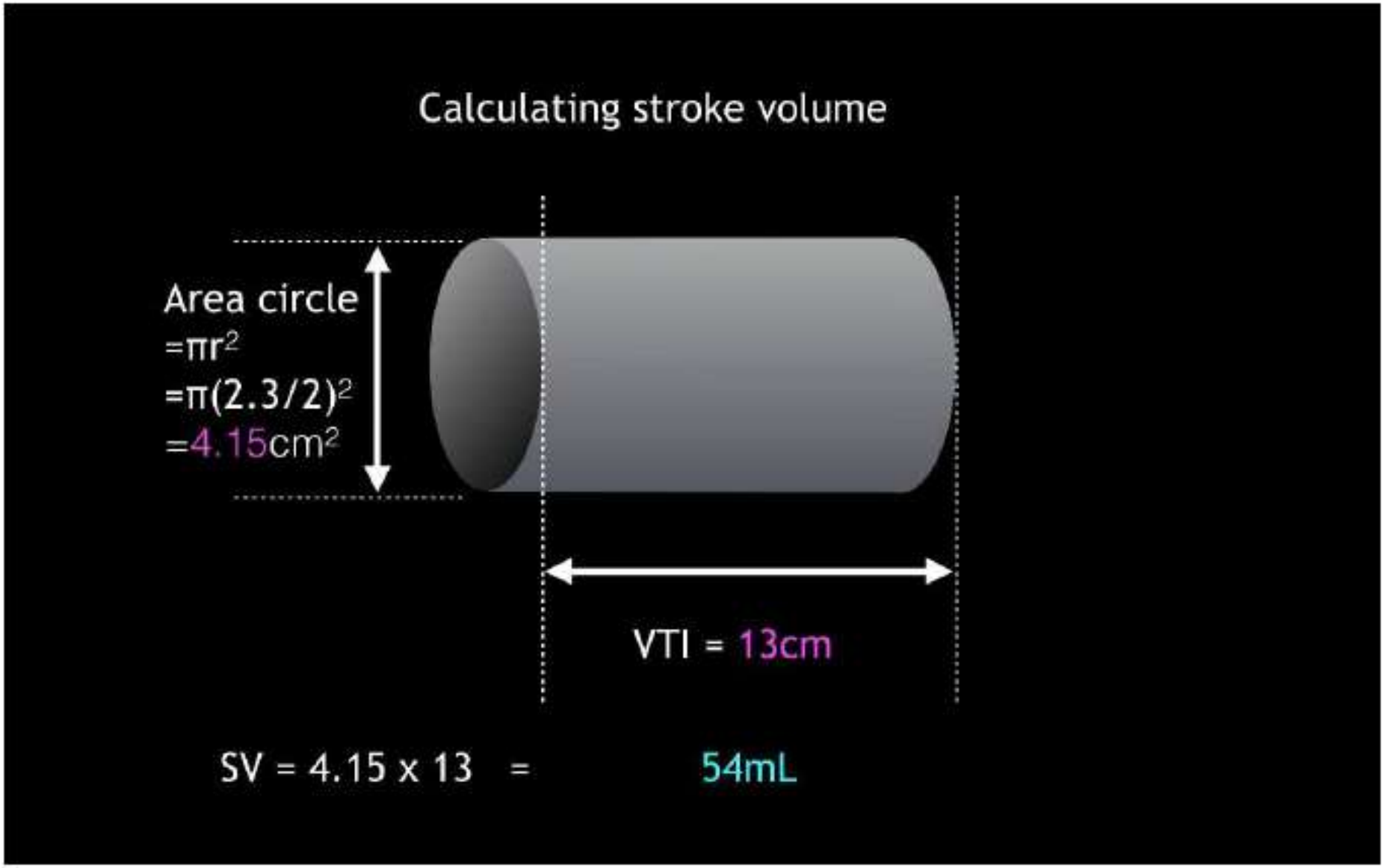
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.



Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.

ELSEVIER

The
American Journal of
Emergency Medicine

www.elsevier.com/locate/ajem

Original Contribution

Measuring cardiac index with a focused cardiac ultrasound examination in the ED☆,☆☆

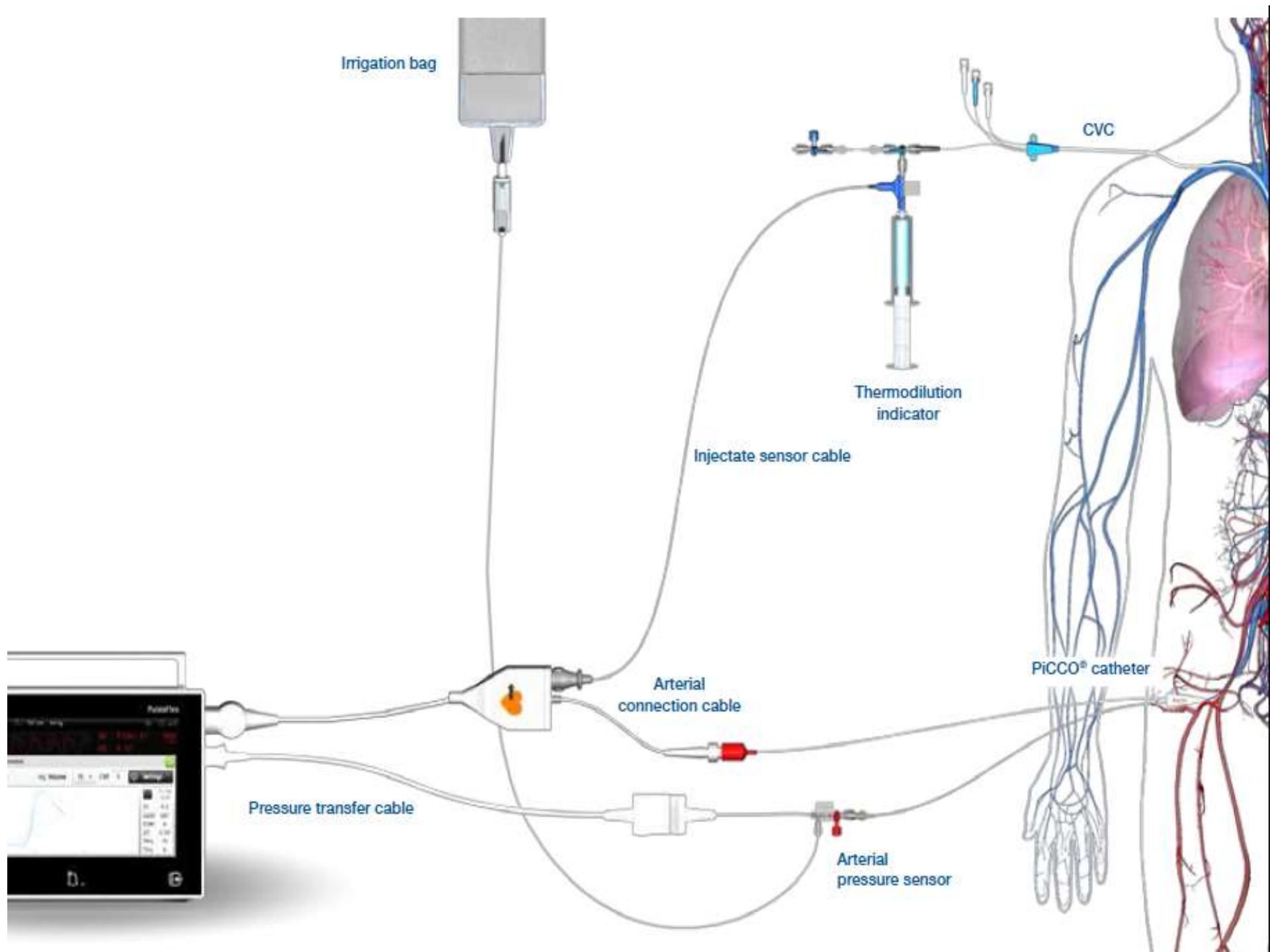
Vi Am Dinh MD[a], H. Samuel Ko MD, MBA[a], Rajiv Rao BS[d],
Ramesh C. Bansal MD, FASE[c], Dustin D. Smith MD[a], Tae Eung Kim MD[a],
H. Bryant Nguyen MD, MS[a,b,*]

- VTI done by emergency doctors co related well with cardiac index

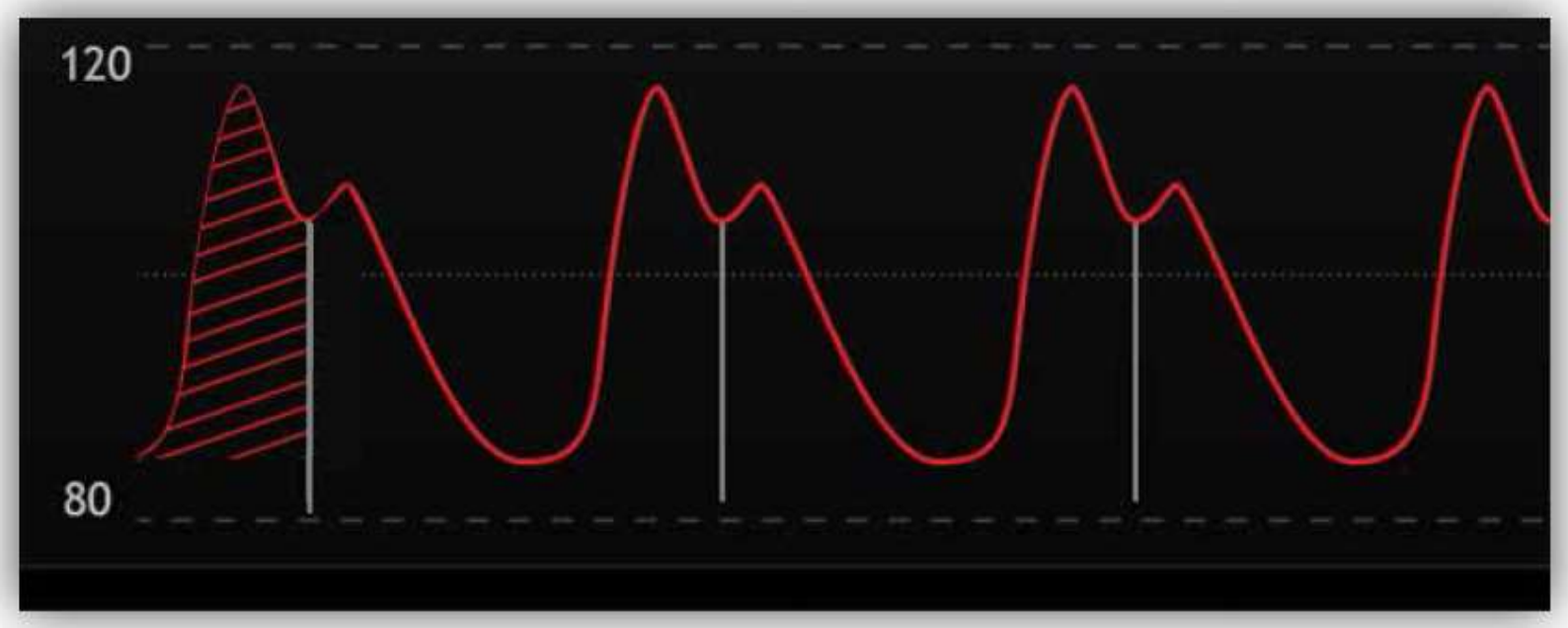
Dinh VA, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012; 30:1845.

PiCCO : Principle

- Calculation of hemodynamic parameters based on
  - Transpulmonary thermodilution (static measurements)
  - Pulse contour analysis (dynamic measurements)
- Independent from breathing or ventilatory cycles
- Depends on heart lung interaction


Irrigation bag
CVC
Thermodilution indicator
Injectate sensor cable
PiCCO® catheter
Arterial connection cable
Pressure transfer cable
Arterial pressure sensor

Arterial pulse contour analysis



The shaded area below the systolic part of the pressure curve is proportional to the stroke volume

Fig. Arterial pulse

Goedje O et al. Accuracy of beat-to-beat cardiac output monitoring by pulse contour analysis in haemodynamical unstable patients. Med Sci Monit 2001;7(6):1344-1350

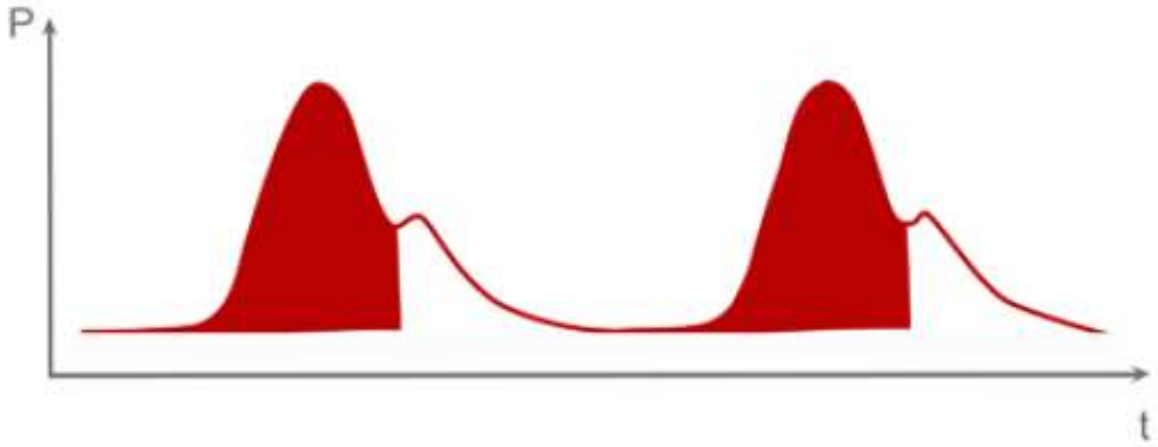


Fig. Analysis of the arterial pressure curve for the area under the systole

With the sophisticated algorithm, the stroke volume is calculated continuously and, by multiplying the stroke volume with the heart rate, a continuous cardiac output is derived, the Pulse Contour Cardiac Output (PCCO)[5].

$$PCCO = cal \times HR \times \int_{systole} \left(\frac{P(t)}{SVR} + C(p) \times \frac{dP}{dt} \right) dt$$

Patient-specific calibration factor (determined with thermodilution)	Heart rate	Area under the pressure curve	Compliance	Shape of pressure curve

Goedje O et al. Accuracy of beat-to-beat cardiac output monitoring by pulse contour analysis in haemodynamical unstable patients. Med Sci Monit 2001;7(6):1344-1350

Transpulmonary thermodilution

- defined bolus, for e.g. 15ml cold normal saline is injected via a central venous catheter
- cold bolus passes through the right heart, the lungs and the left heart and is detected by the PiCCO catheter
- repeated around three times in under 10 minutes to ensure an accurate average is used to calibrate the device and to calculate the thermodilution parameters
- It is recommended to calibrate the system at least 3 times per day.

PiCCO Parameters

- Cardiac Index
- Stroke volume index
- Global end diastolic volume index : preload
- Pulse pressure variation
- Stroke volume variation

Depends on Cardiopulmonary interaction

- Systemic vascular resistance index : afterload
- Extravascular lung water : pulmonary edema

Overcoming these detrimental interaction

- ↓WOB by ↓ resistance & ↓ collapsed alveoli
- ↓negative swings in ITP/ dysynchrony
- Prevent hyperinflation (use lowest PEEP)
- Fluid resuscitation at initiation
- Prevent volume overload during weaning