

Ventriculo-Arterial Coupling

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Presenter Disclosure Information

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FINANCIAL DISCLOSURE:

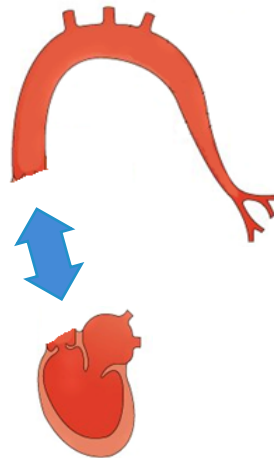
Grants/Research Support: Maquet

Speaker: Maquet, Pulsion Medical

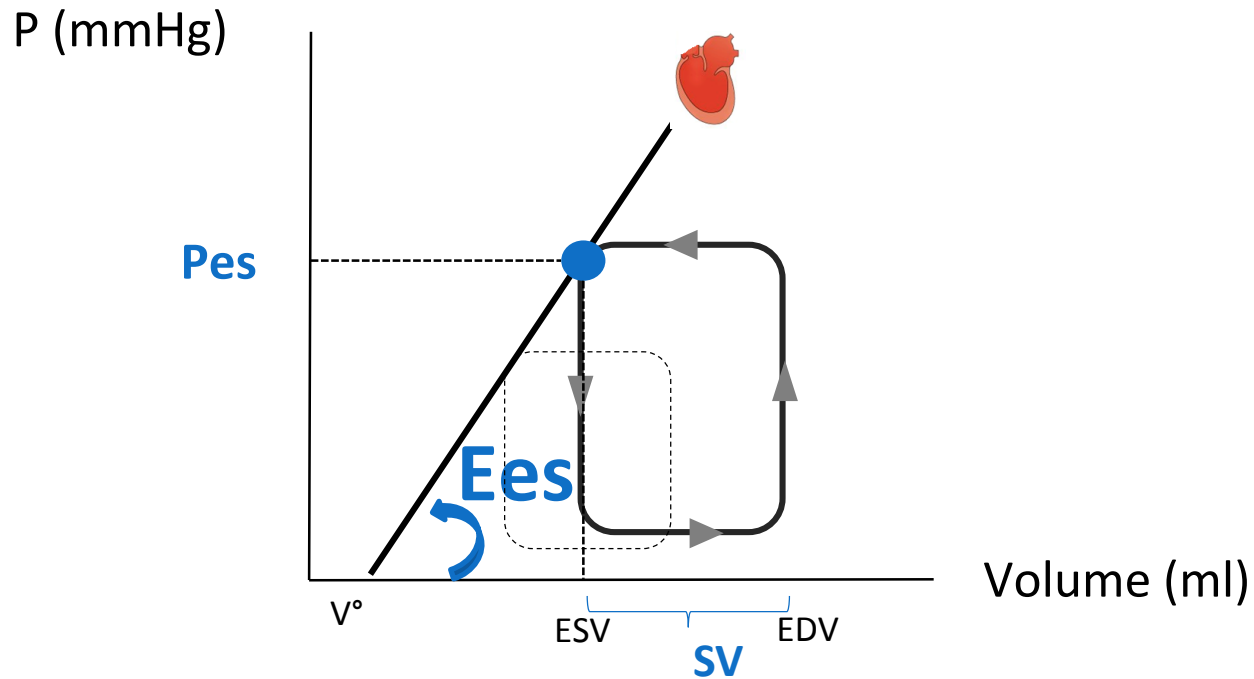
- ✓ Theoretical overview
- ✓ Assessment in experimental settings
- ✓ Assessment at bedside
- ✓ Clinical relevance and applications
- ✓ Conclusion

Why is VA coupling a key point in critically ill patients?

- ❑ Cardiovascular performance results from continuous interaction between ventricular and vascular systems
- ❑ Cardiovascular system works better if ventricular pump and arterial system are perfectly matched
- ❑ This concept may be applied in systemic as well as in pulmonary circulation

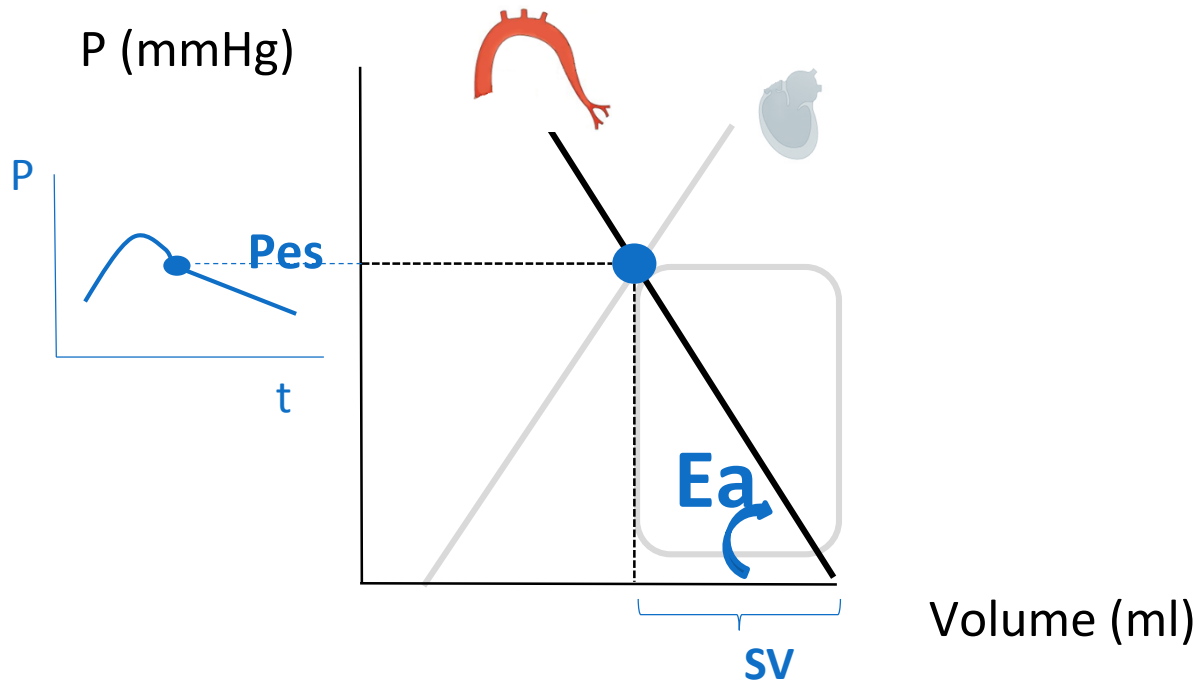


Ventricular system



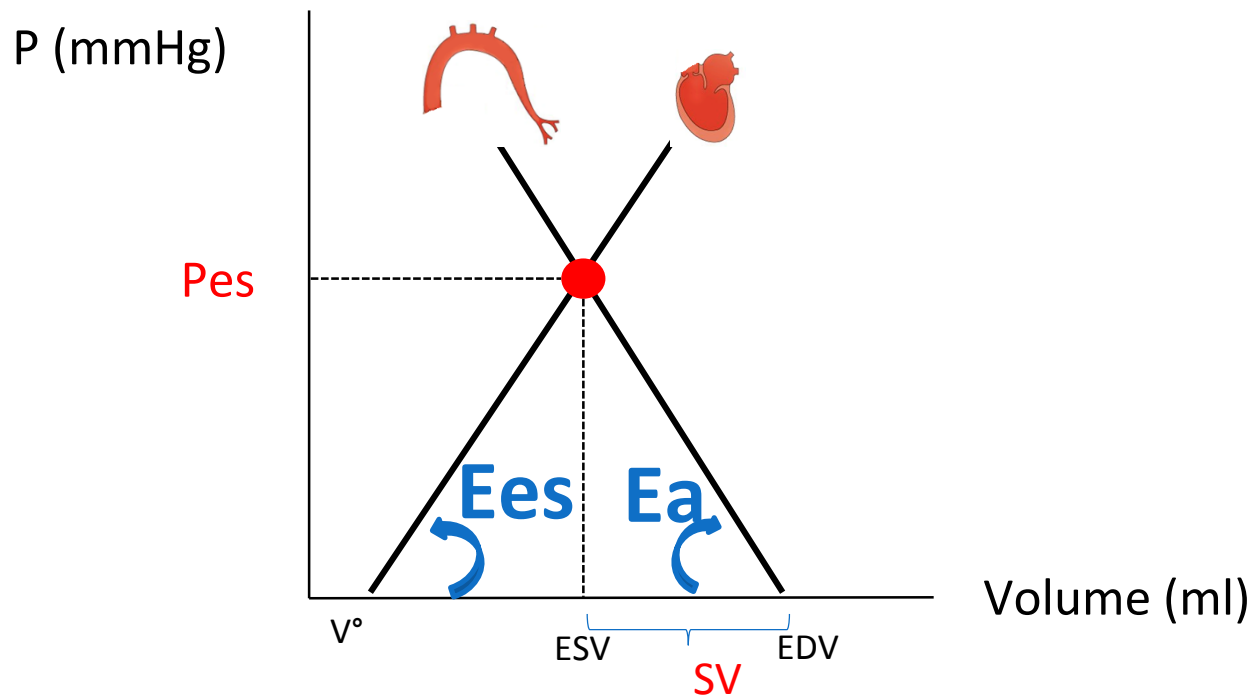
- ❑ The ventricular system can be characterized by an **elastance**:
 E_{es} (mm Hg / mL) = end-systolic elastance = contractility

Arterial system



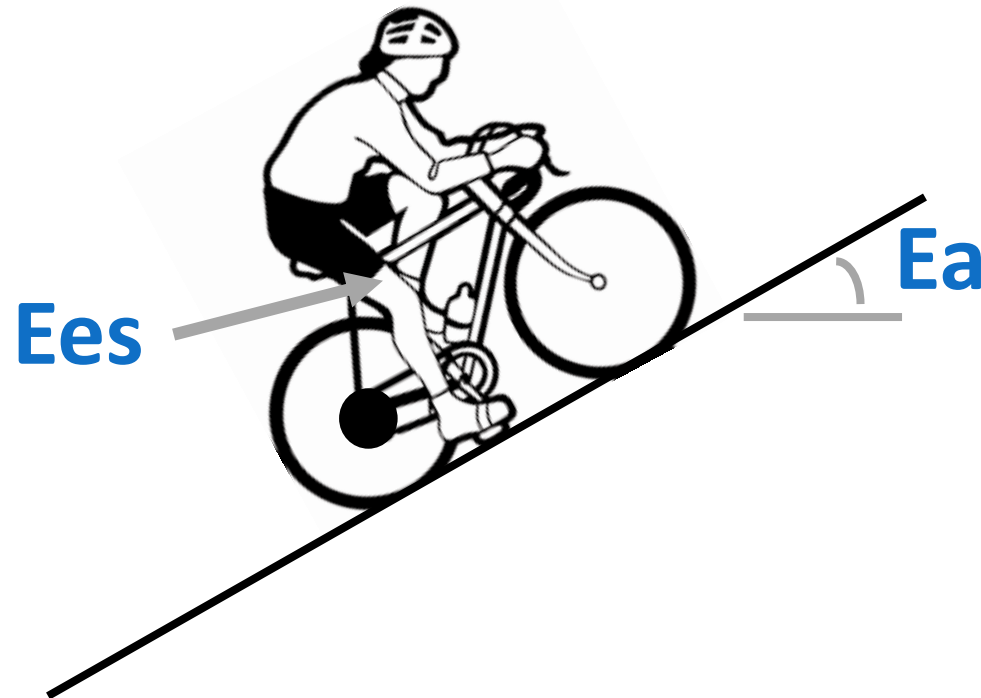
- The arterial system can also be characterized by an **elastance**:
 E_a (mm Hg / mL) = arterial elastance = afterload

VA coupling



- ❑ Intersection of elastance lines gives the working point
- ❑ **SV** and **Pes** result from this intersection

Matching between contractility and afterload

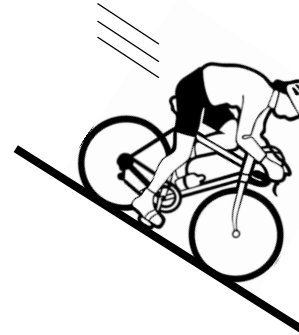


- ❑ Ventricular contractility (E_{es}) = driving force of the biker
 - ❑ Afterload (E_a) = slope of the road
- slope and driving force should be perfectly matched

Energetics

$$\text{efficiency} = \frac{\text{useful energy out}}{\text{total energy in}} \approx \frac{SW}{\text{Vo}_2}$$

$E_{es}/E_a = 2 \rightarrow$ maximum efficiency



$E_{es}/E_a = 1 \rightarrow$ maximum SW

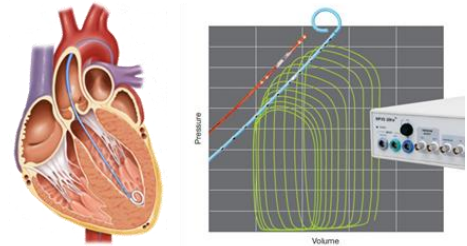


$E_{es}/E_a \ll 1 \rightarrow$ uncoupling

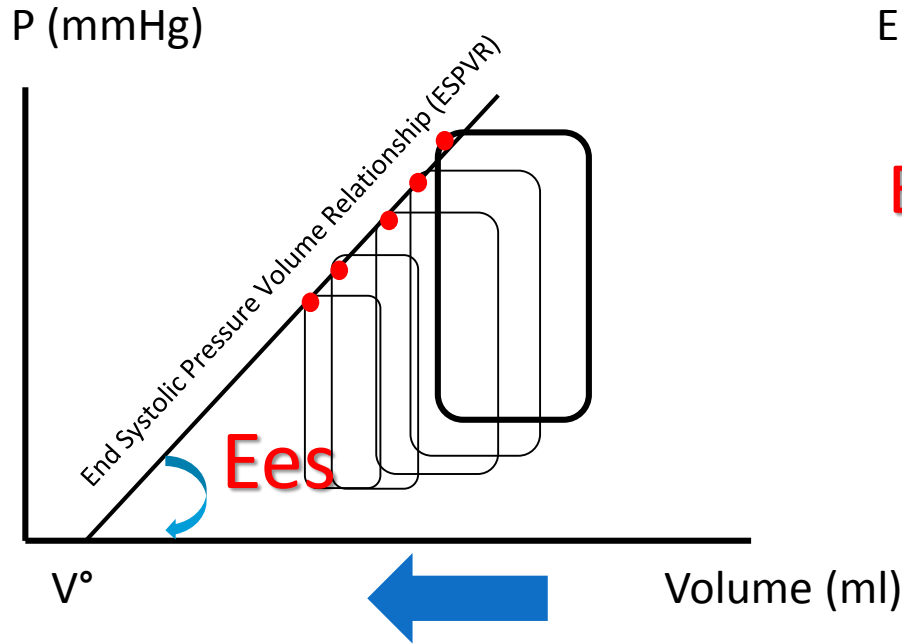




From bench

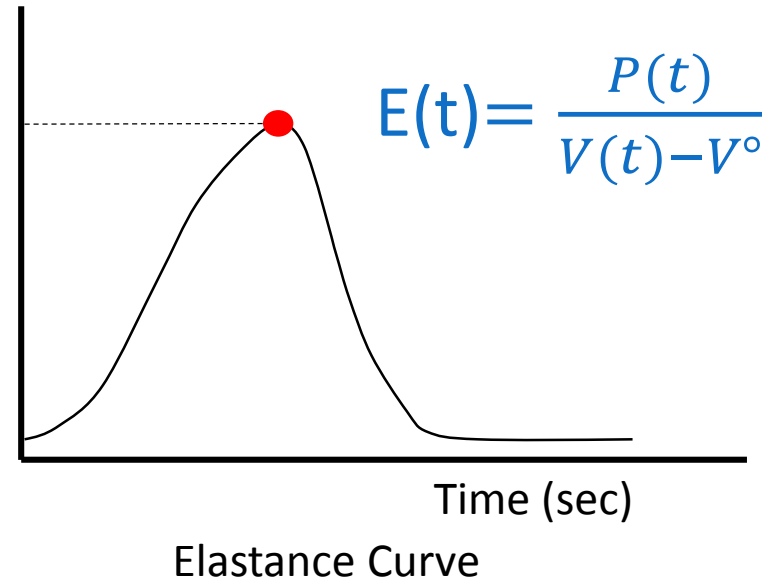


1. Ventricular system: Ees



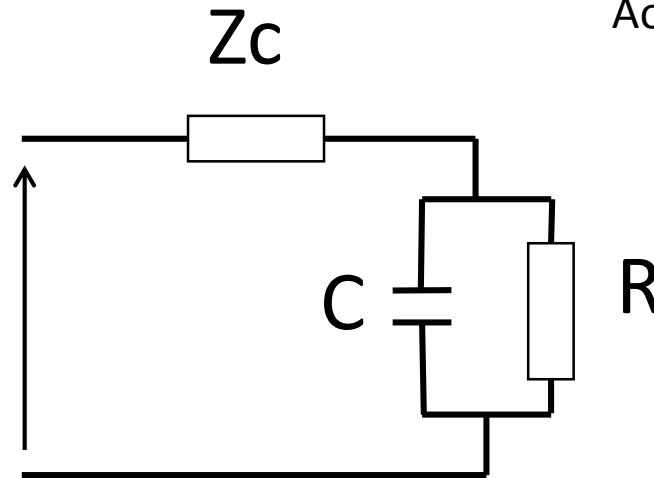
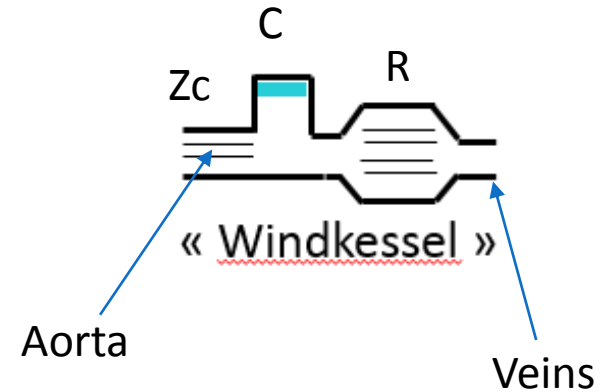
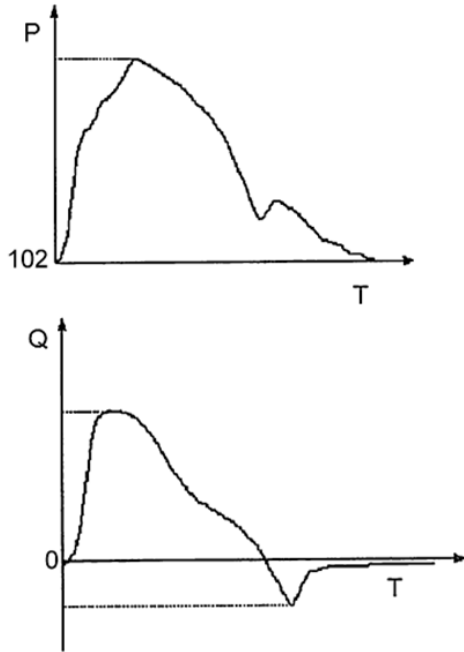
E (mmHg/ml)

E_{es}



- End-systolic points obtained from **preload reduction**
- E_{es} = slope of ESPVR
- Independent of loading conditions
- = « intrinsic » contractility

2. Arterial system: E_a



- ❑ Arterial system can be modeled by an electrical circuit (Windkessel » model)
- ❑ Resistive and compliant elements can be calculated from pressure and flow waves

❑ E_a is precisely derived from the Windkessel model:
$$E_a = \frac{Z_c + R2}{ts + R2.C(1 - e^{-\frac{td}{R2.C}})}$$



From bench

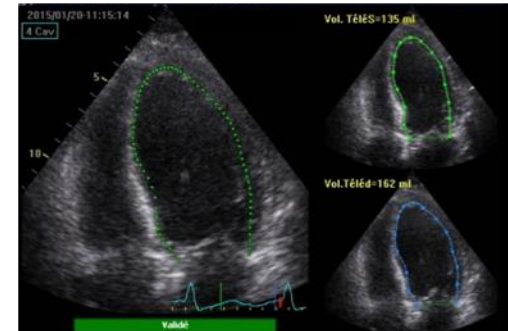
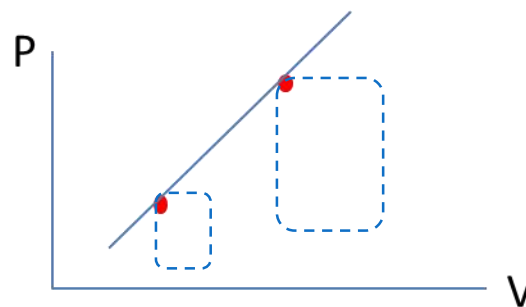
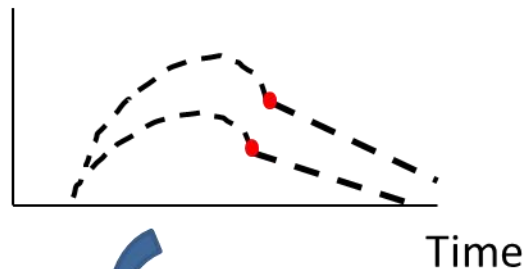
.... to bedside



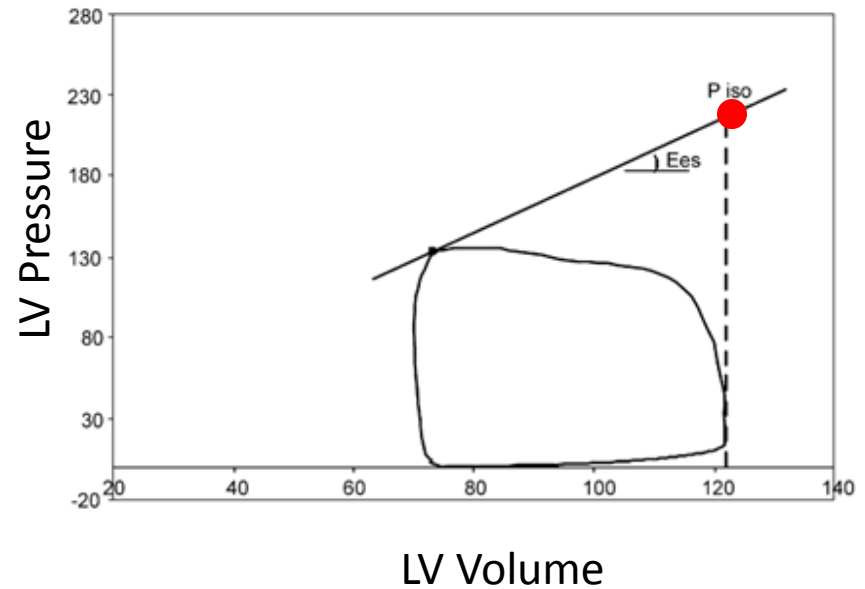
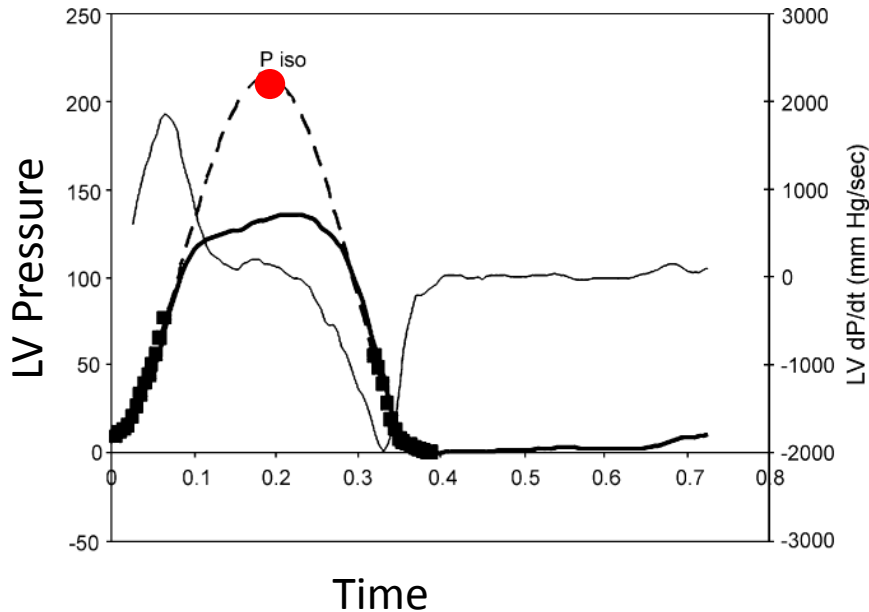
1. How to assess Ees at bedside?

- Pes derived from invasive art P
- ESV derived from echocardiography
- Preload reduction is obtained from inspiratory hold maneuver of 10 cmH2O

P (mm Hg)



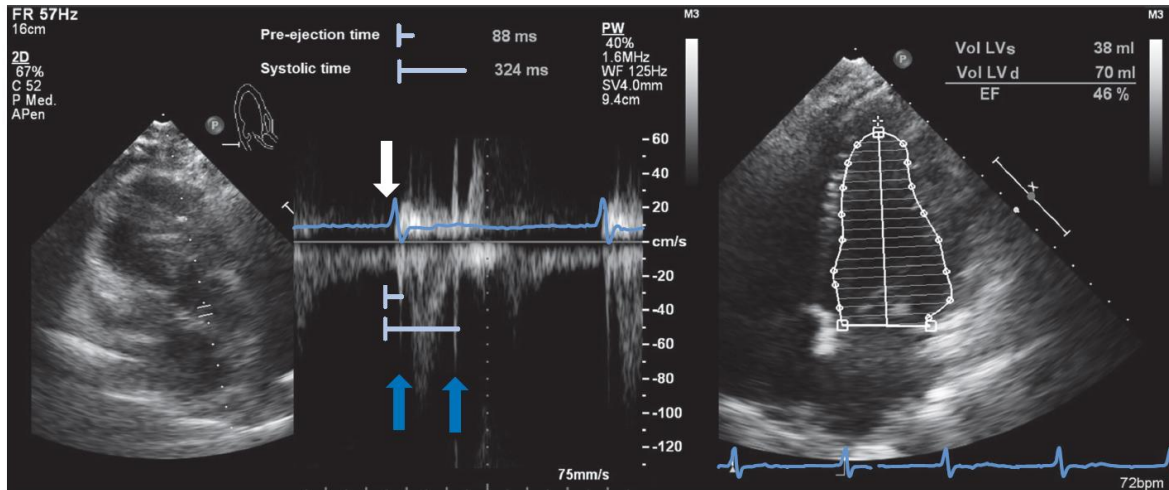
Single beat analysis



- ❑ Isovolumic LV contraction extrapolated to determine **P iso**
- ❑ **P iso** is reported on PV graph to determine a second « fictive point »

Takeuchi M et al. Single-beat estimation of the slope of the end-systolic pressure-volume relation in the human left ventricle. *Circulation* 1991;83:202–12.

Echocardiographic single beat method



Pd
 Ps
 SV
 EF
 Pre-ejection
 period
 Total systolic
 period

$$E_{Nd(est)} \rightarrow E_{es(sb)} = [Pd - (E_{Nd(est)} \times Ps \times 0.9)] / (E_{Nd(est)} \times SV)$$

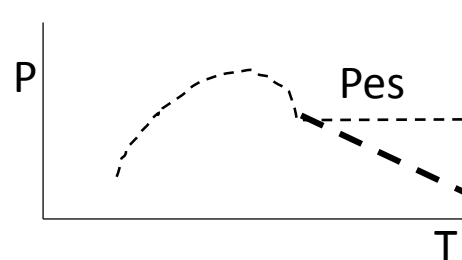
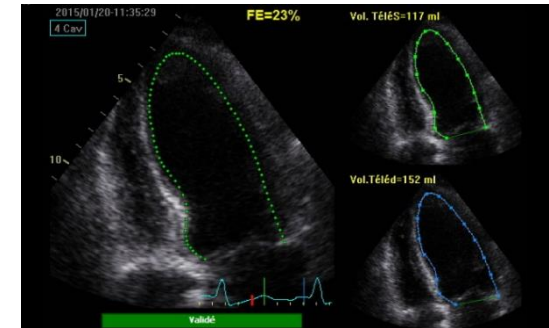
No preload variation

- Chen CH et al. Noninvasive single-beat determination of left ventricular end-systolic elastance in humans. *J Am Coll Cardiol* 2001, 38:2028–2034.
- Guarracino et al. Ventriculoarterial decoupling in human septic shock. *Critical Care* 2014, 18:80

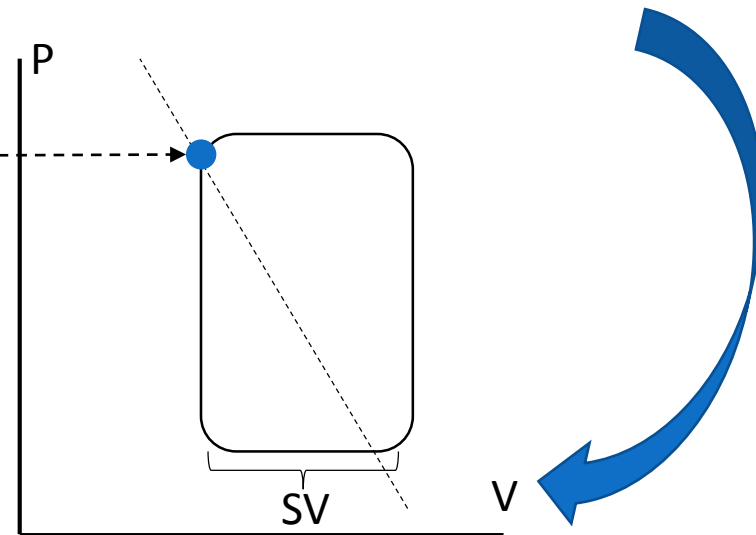
2. How to assess Ea at bedside?

Ea can be approximated as:

$$Ea \approx \frac{Pes}{SV}$$



- ✓ Validation in systemic circulation
- ✓ Validation in pulmonary circulation



- Kelly et al. *Circulation* 1992;86:513—21.
- Morimont P et al. *Am J Physiol Heart Circ Physiol* 2008: 294,H2736-H2742

Clinical relevance in ICU

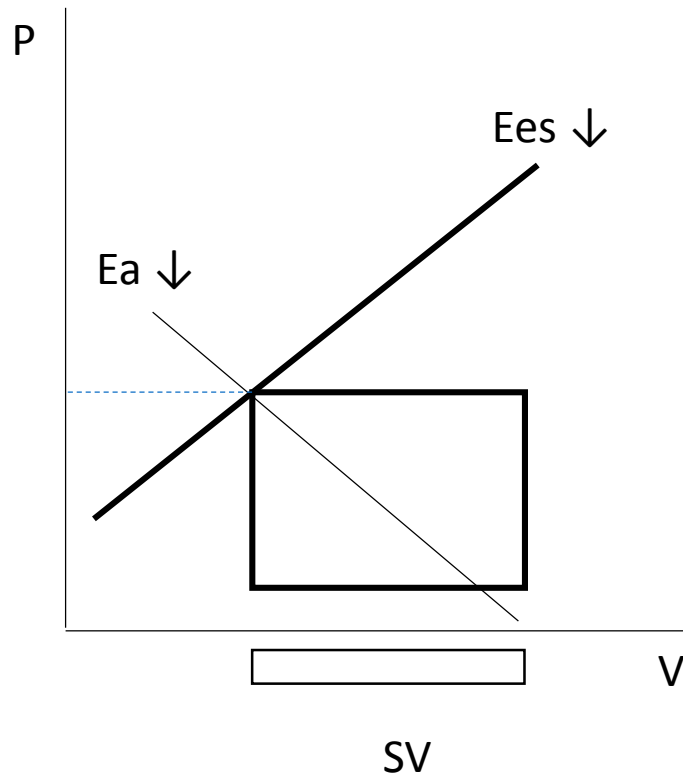
	↑ Ea	↓ Ees
Right VA uncoupling	Pulmonary embolism Pulmonary HT High airway P ARDS Endotoxin Hypercapnic acidosis Hypoxic vasoconstriction ...	RV infarction RV dysfunct° (post Tx) Hypercapnic acidosis Hypoxemia Sepsis ...
Left VA uncoupling	Vasopressors Correct° of mitral regurgit° Aortic stenosis	Myocardial infarction Myocarditis Septic cardiomyopathy Toxic cardiomyopathy

- Ghuysen A et al. *Shock*. 2008;29(2):197-204
- Lambermont B et al. *Cardiovasc Res*. 2003, 59:412-418.
- Segers P, Morimont P et al. *Am Heart J* 2002 ; 144(4) : 568-76

- Price L. et al. *Critical Care* 2010, 14:R169
- Guarracino F. et al. *Critical Care* 2013, 17:213
- Binkley PF et al. *J Am Coll Cardiol* 1990, 15:1127–1135

Septic shock

- ❑ Depressed LV contractility ($E_{es} \downarrow$) is induced by cytokines, NO, acidosis ...
- ❑ Decreased arterial elastance ($E_a \downarrow$) results from vasoplegia

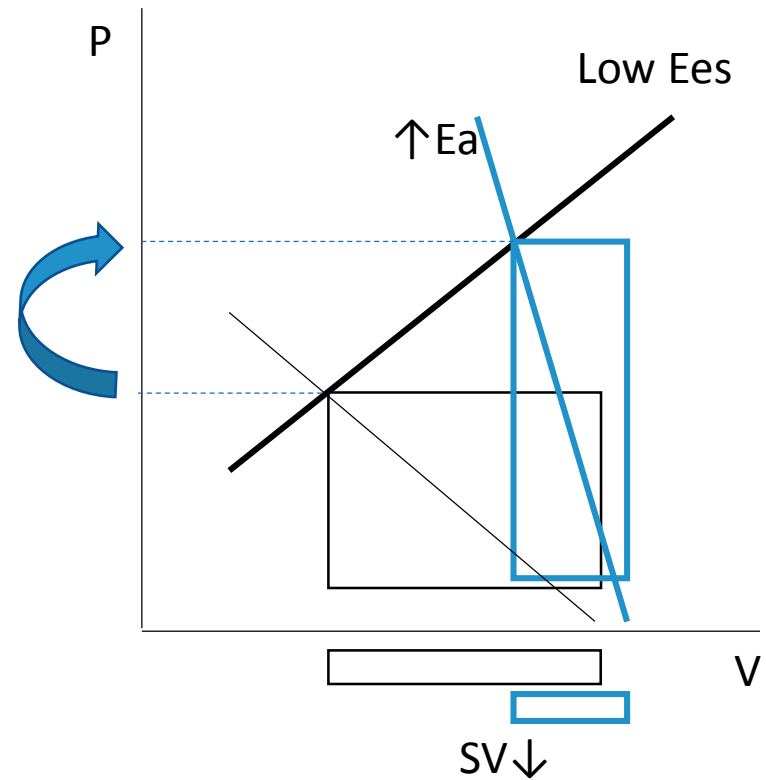


Jardin F et al. Sepsis-related cardiogenic shock. *Crit Care Med* 1990, 18:1055-1060

Guarracino et al. Ventriculoarterial decoupling in human septic shock. *Critical Care* 2014, 18:80

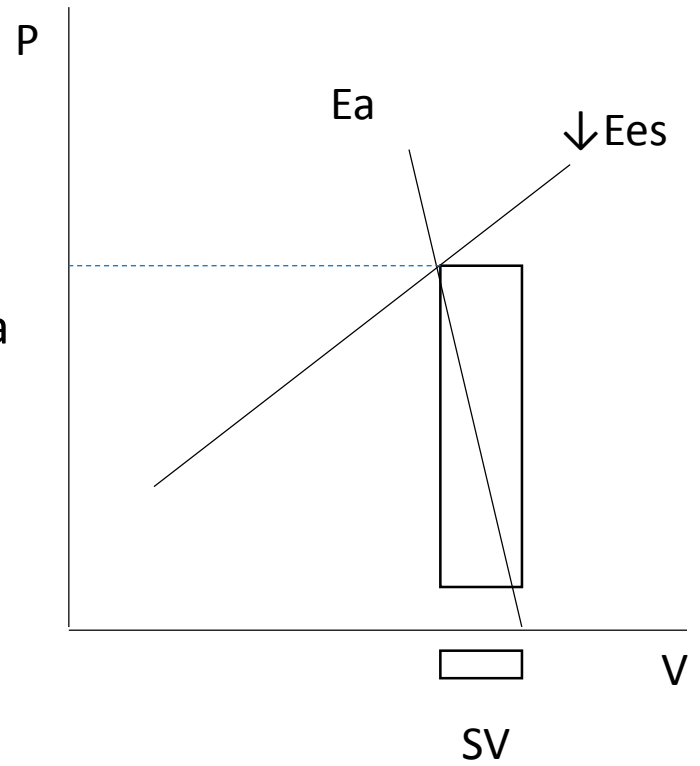
Septic shock + vasopressors

□ Vasopressor: $\uparrow E_a \rightarrow \downarrow \frac{E_{es}}{E_a}$



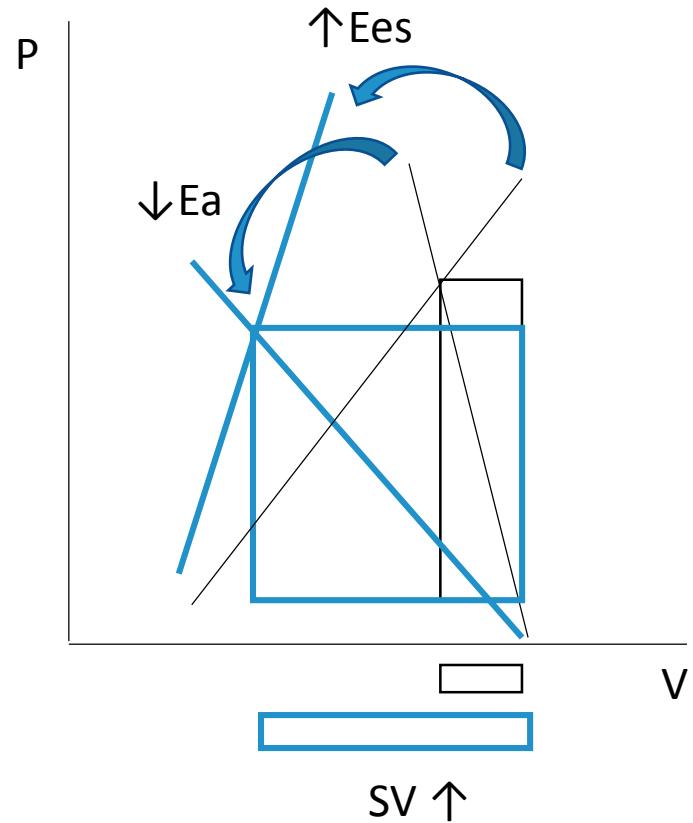
LV failure

Mismatch between depressed Ees and high Ea



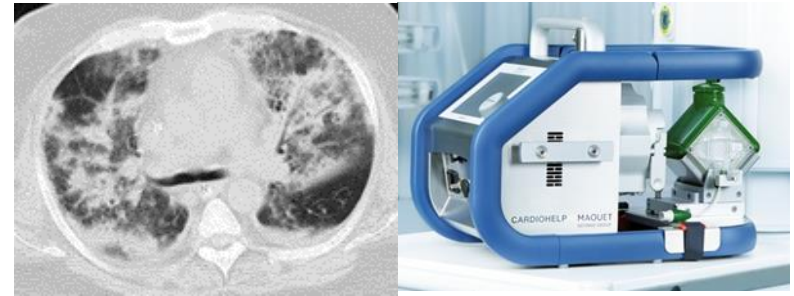
LV failure

Combination of inotropes and vasodilators
improves VA coupling: $\frac{Ees\uparrow}{Ea\downarrow}$



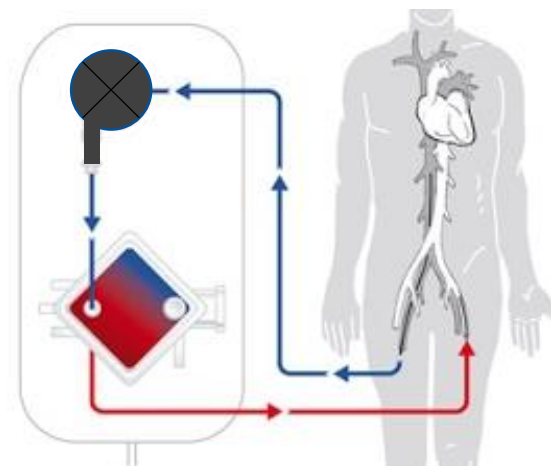
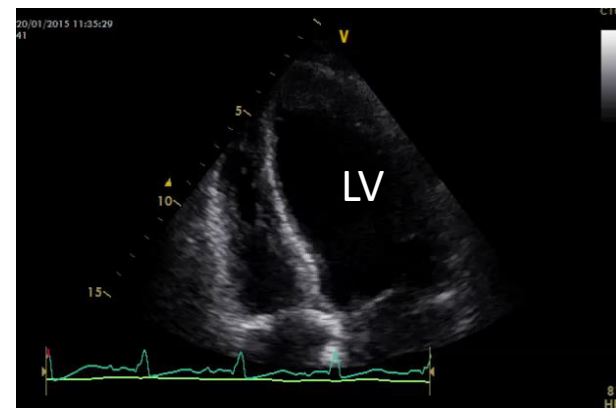
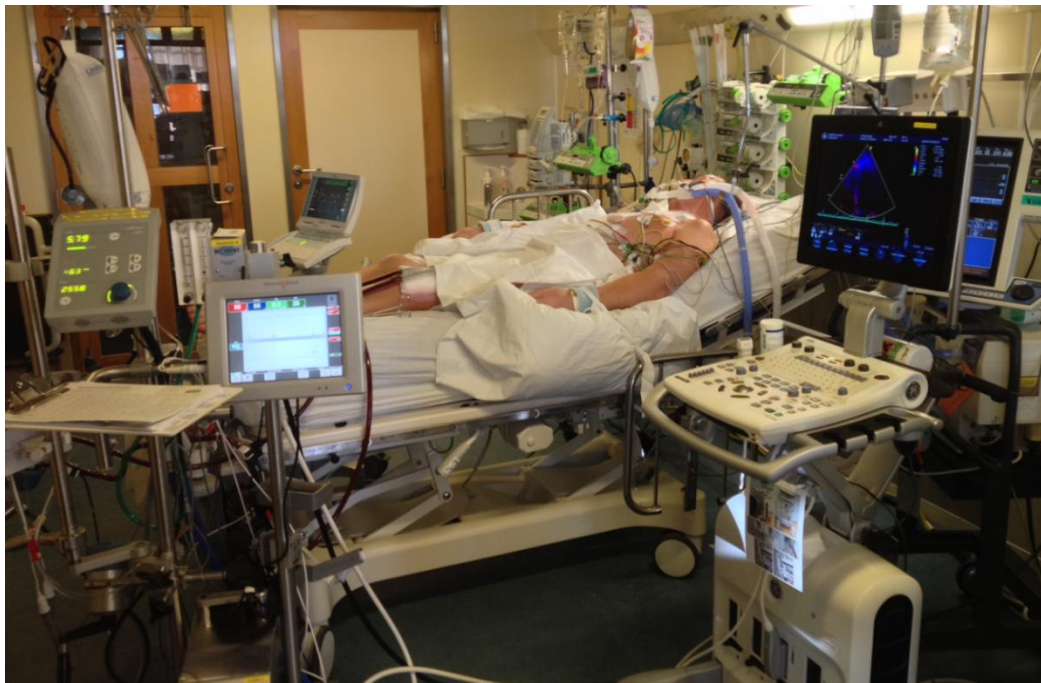
Guarracino F, et al. Effect of levosimendan on ventriculo-arterial coupling in patients with ischemic cardiomyopathy. *Acta Anaesthesiol Scand* 2007, 51:1217–1224.

ECCO2R



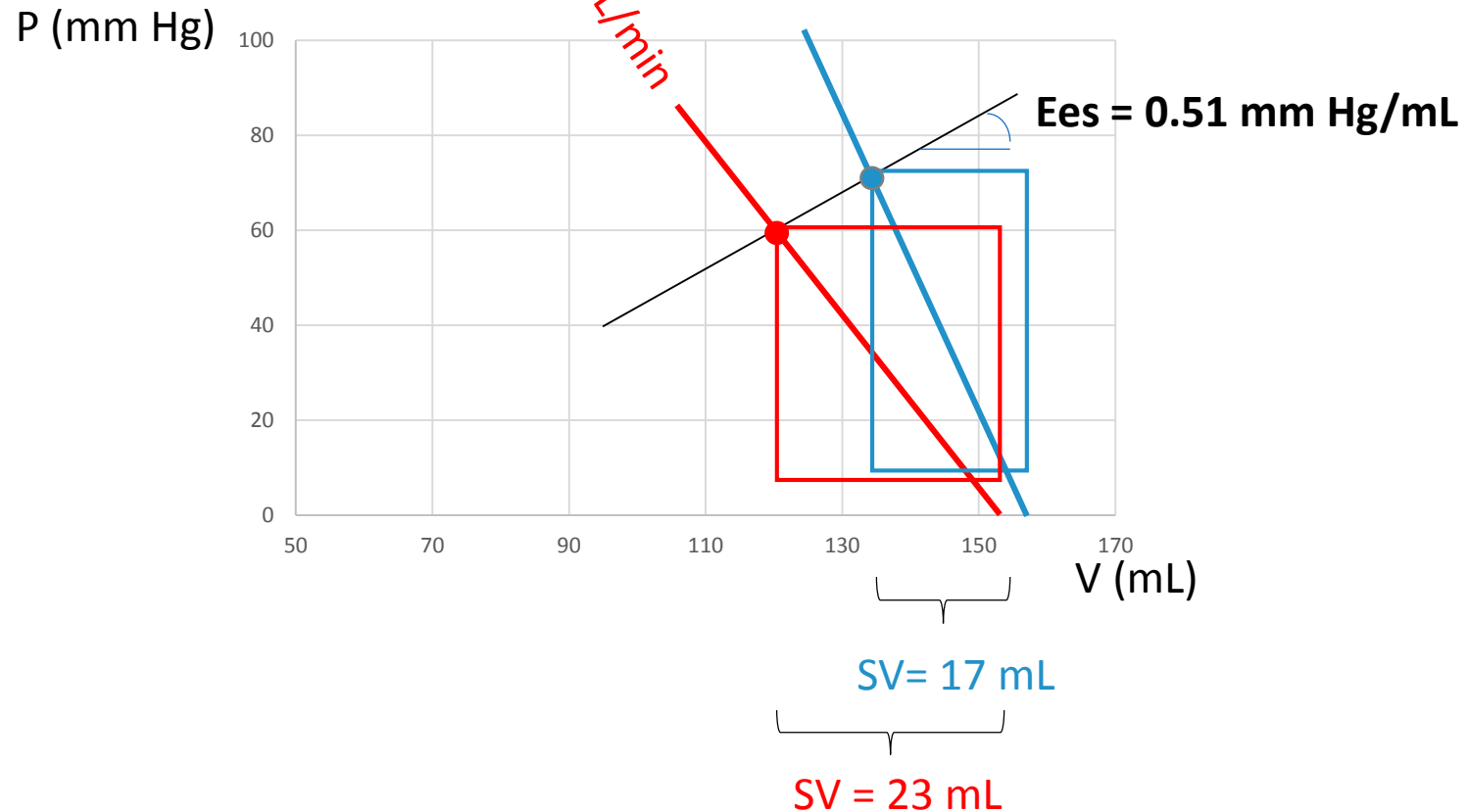
- ❑ In ARDS, hypercapnic acidosis resulting from protective ventilation is responsible for increased PHT ($\uparrow E_a$) and decreased RV contractility: $\frac{E_{es\downarrow}}{E_a\uparrow} \ll 1$
- ❑ CO2 removal decreases PHT ($\downarrow E_a$), improves RV function ($\uparrow E_{es}$) and restores VA coupling in an experimental model of ARDS : $\frac{E_{es\uparrow}}{E_a\downarrow} \approx 1$

VA coupling and VA ECMO



- Mr B. L. 54 YO
- CAD (Cx – IVA)
- Cardiogenic shock post CABP
- ECMO VA (Femoro-Femoral)

PV loops



□ changes in ECMO flow → changes in pre- and after- load → end-systolic points → Ees

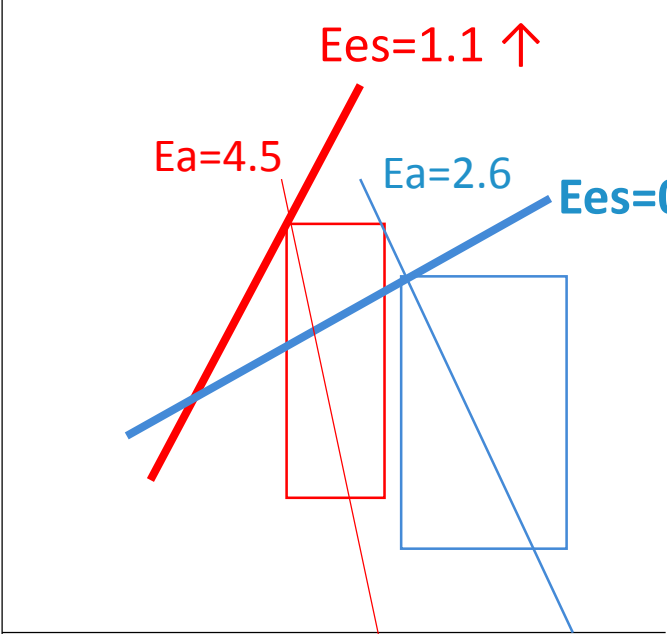
Hemodynamics

	DAY 1	DAY 3
Dobutamine ($\mu\text{g}/\text{kg}/\text{min}$)	5	5 =
Norepineph ($\mu\text{g}/\text{kg}/\text{min}$)	5	15 \uparrow
ECMO Flow (L/min)	5.80	5.50 \approx
MAP (mm Hg)	71	89 \uparrow
SV (mL)	27	20 \downarrow
LVEF (%)	17	14 \downarrow
Ees (mm Hg/mL)	0.51	1.09 \uparrow
Ea (mm Hg/mL)	2.63	4.45 $\uparrow\uparrow$
Ees/Ea	0.33	0.24 $\downarrow\downarrow\downarrow$

- Increased vasopressors \rightarrow increased Ea \rightarrow uncoupling \rightarrow decreased LVEF
- However, LV contractility improved !!!

PV loops

LV pressure (mm Hg)



Day 1

Day 3

LV volume (mL)

→ Recovery is unmasked by using VA coupling

Conclusions

- ❑ Management of critically ill patients with **acutely altered hemodynamic states** can benefit from assessment of VA coupling.
- ❑ Treatment oriented at **normalizing Ees/Ea** ratio improves cardiovascular efficiency.
- ❑ Bedside evaluation of Ees/Ea is becoming **available and reliable** because of the improvement in echocardiographic methods

Acknowledgements

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- Leon Fredericq Foundation of the University of Liège, Belgium