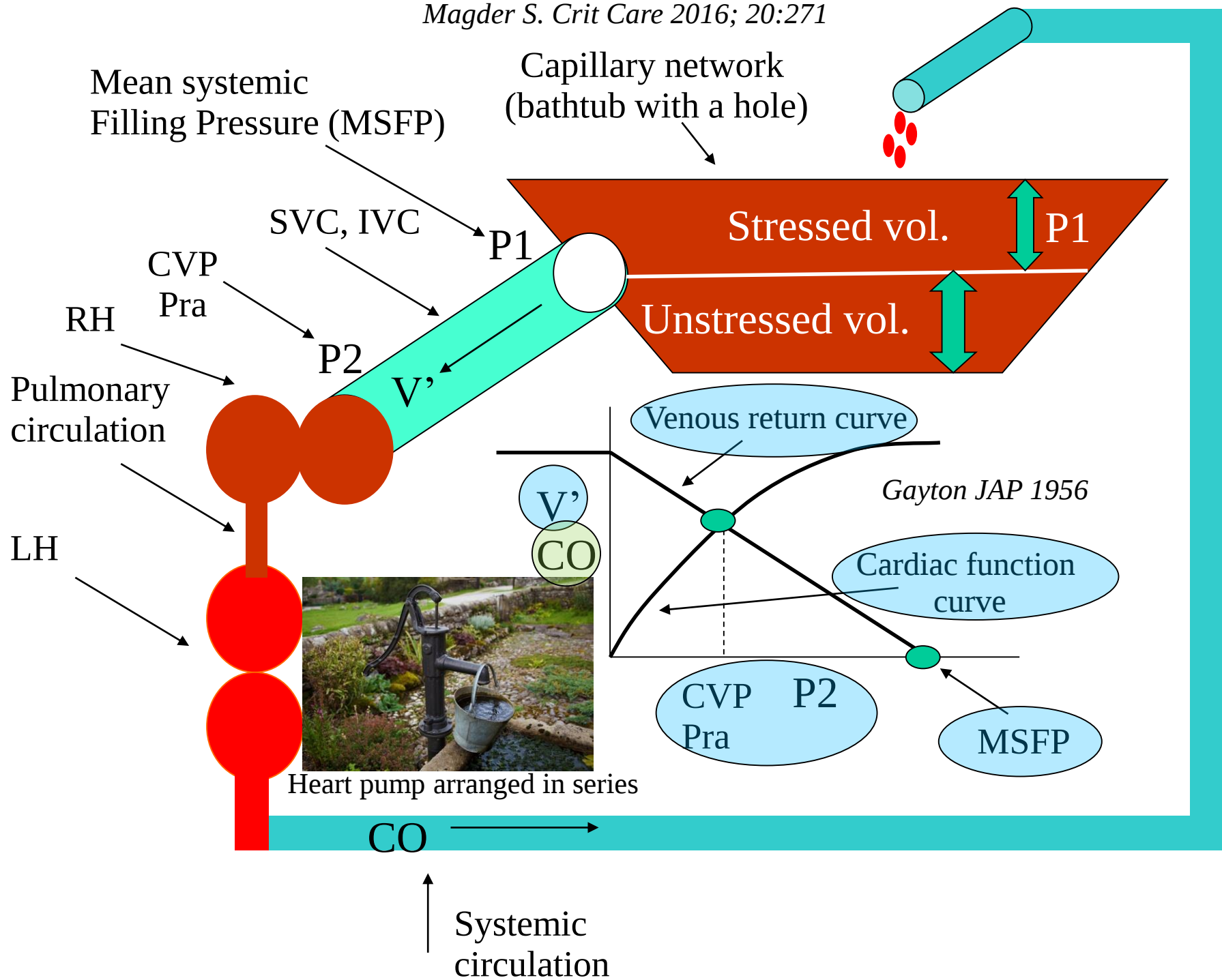
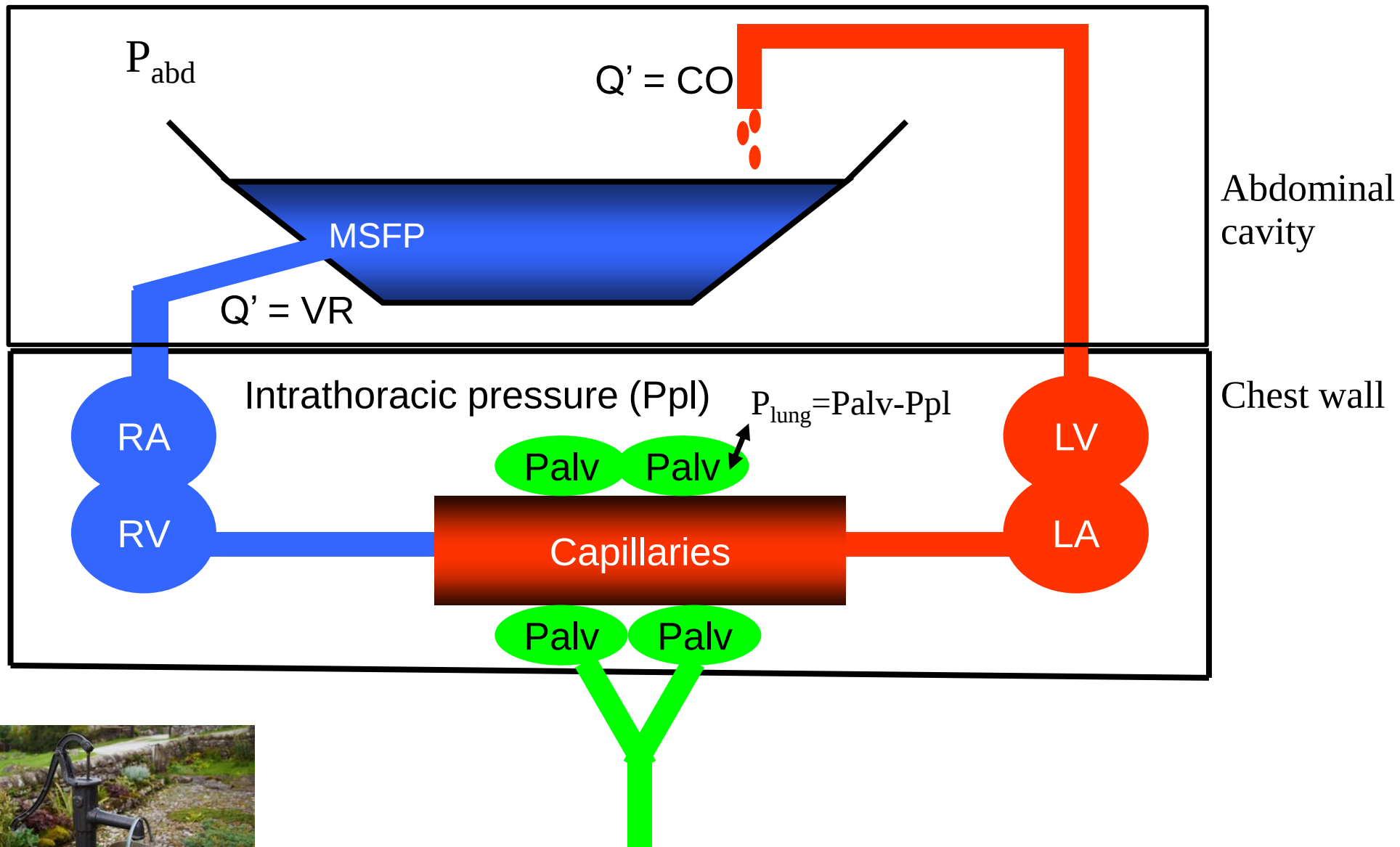


Heart-lung interaction - Its role in pulmonary circulation (Spontaneous breathing –mechanical ventilation)

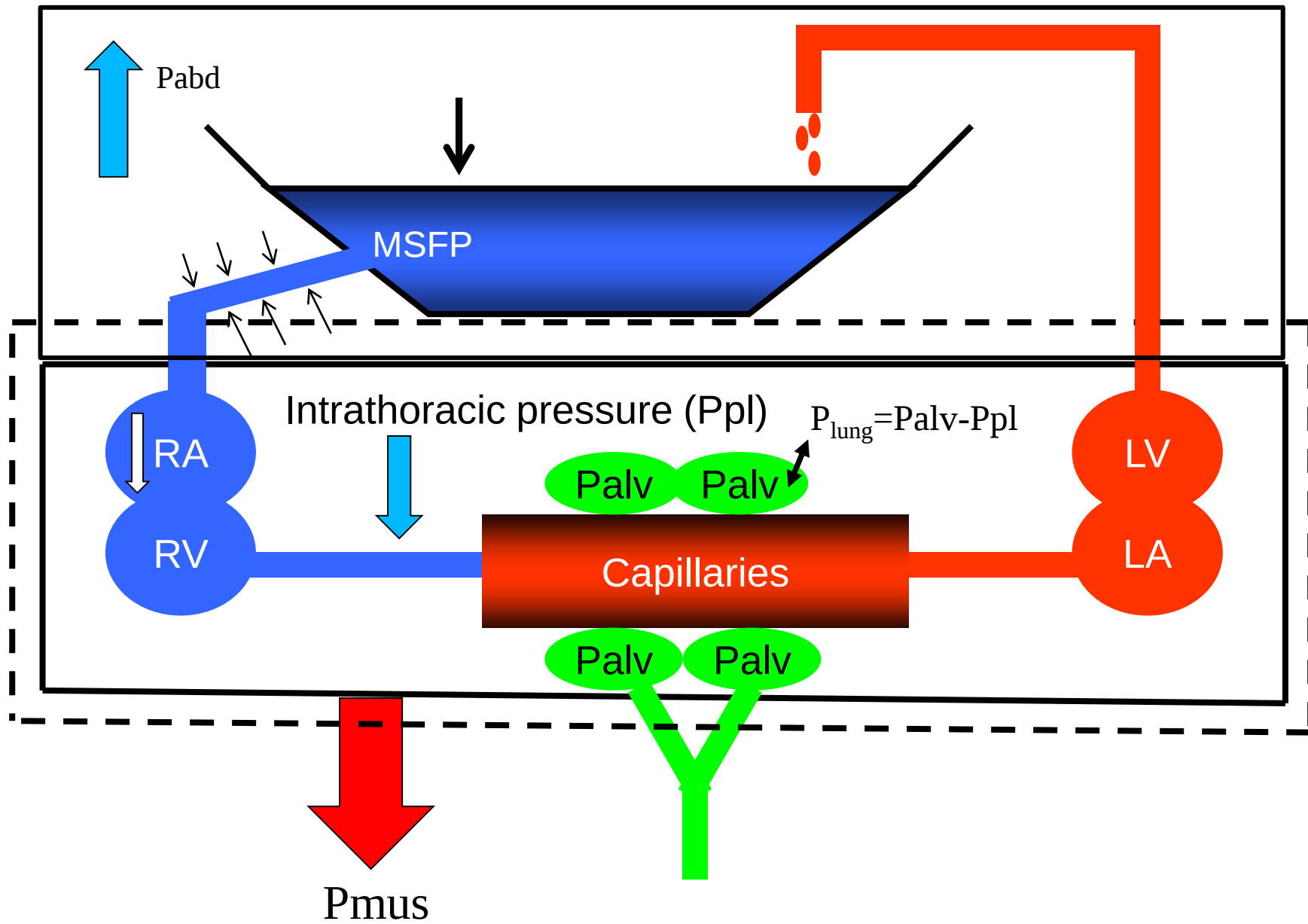


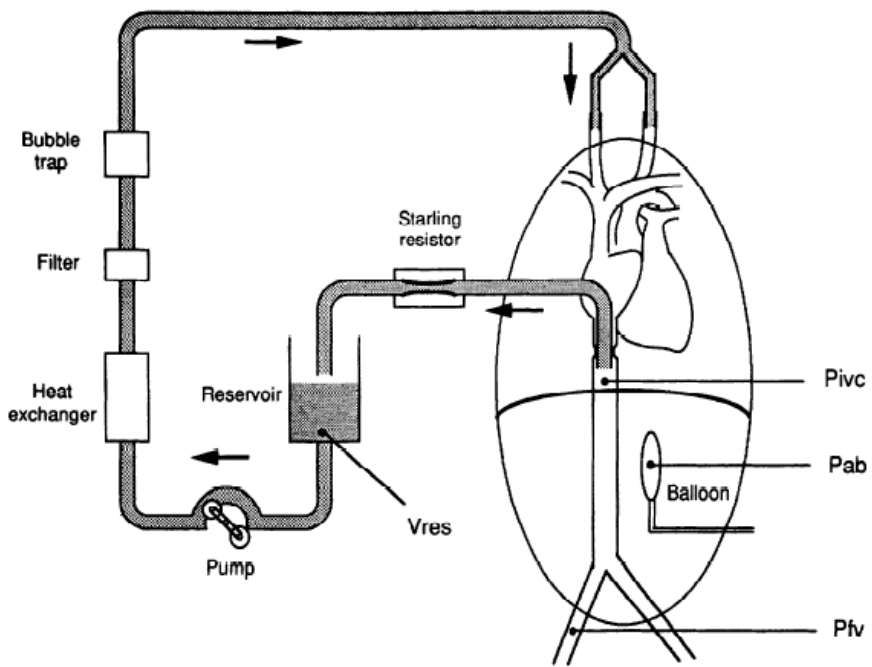
*D. Georgopoulos, Professor of Medicine,
Department of Intensive Care Medicine,
University Hospital of Heraklion,
University of Crete, Greece*





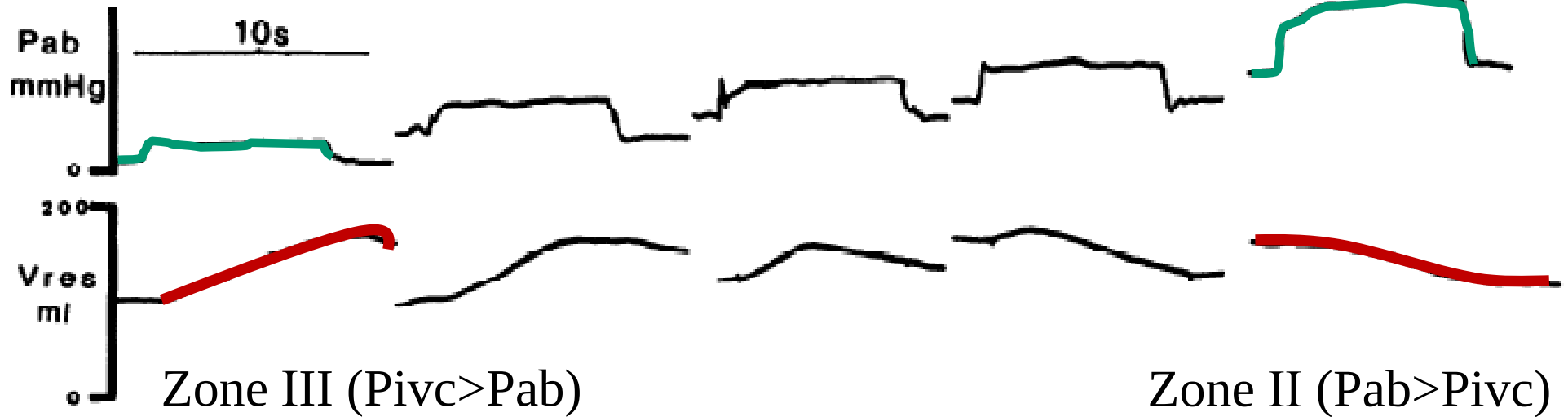
Venous return (VR) = Cardiac output (CO)





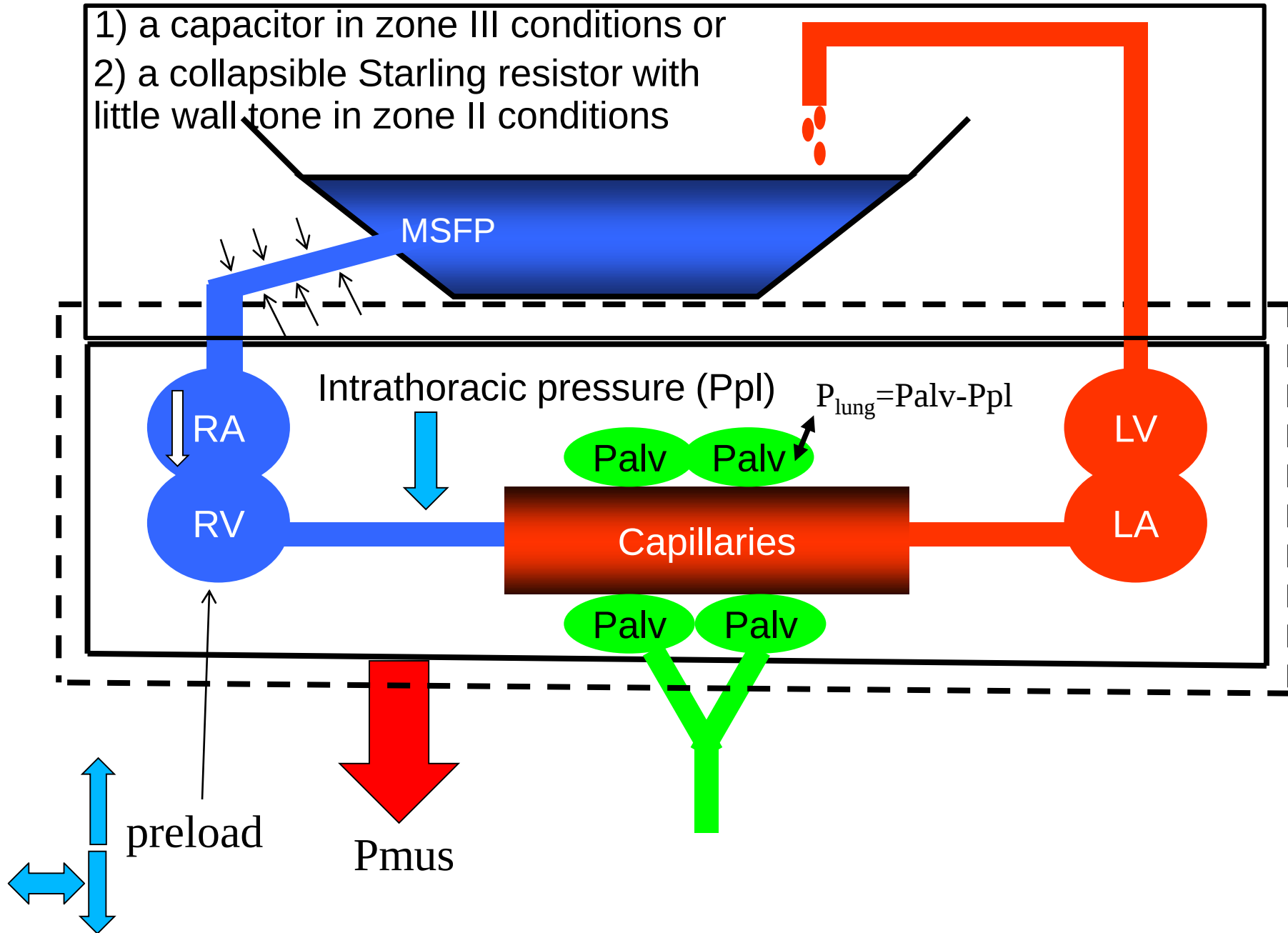
8 sec Phrenic nerve stimulation

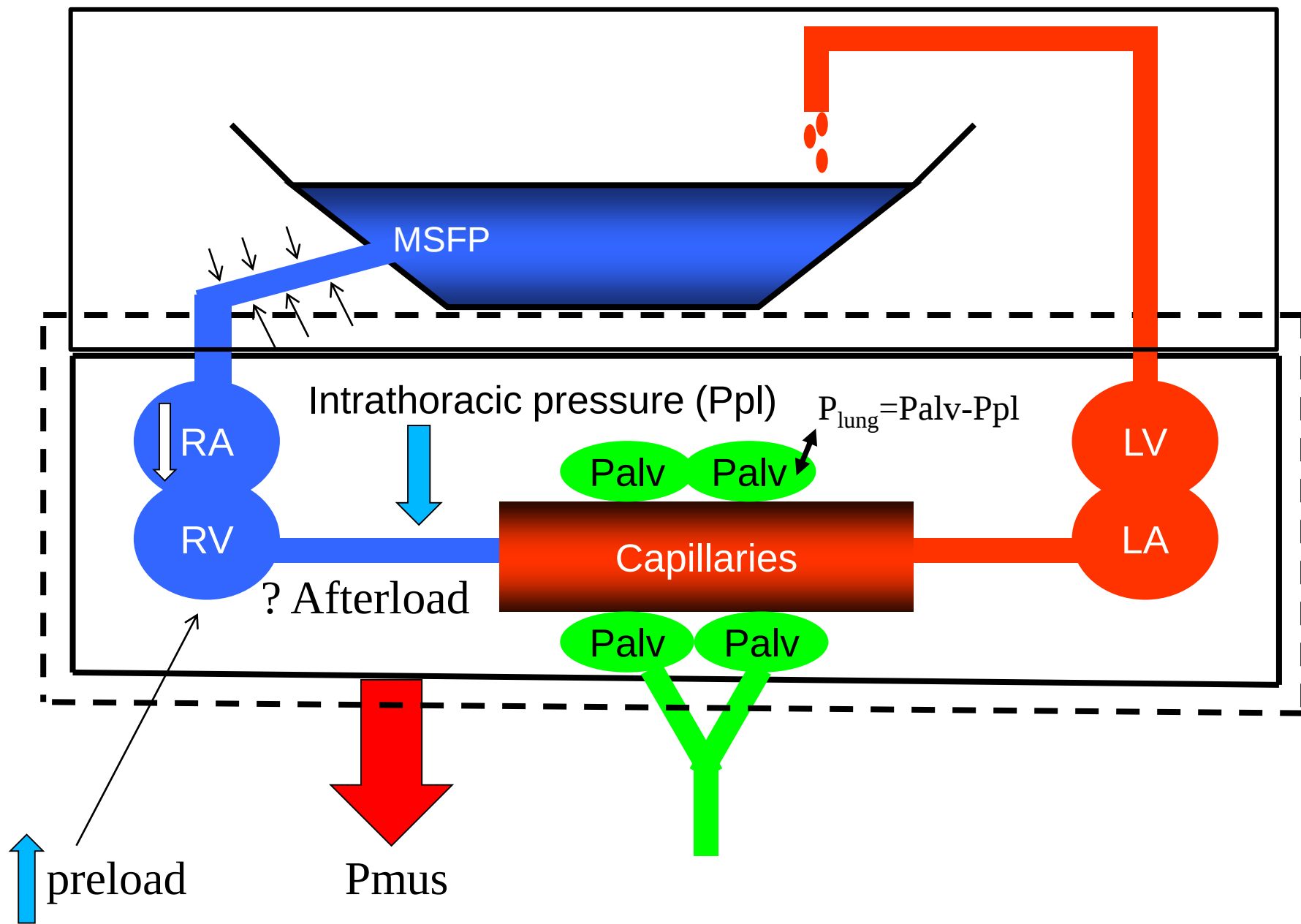
Abdominal hypertension



Abdominal compartment behaves as:

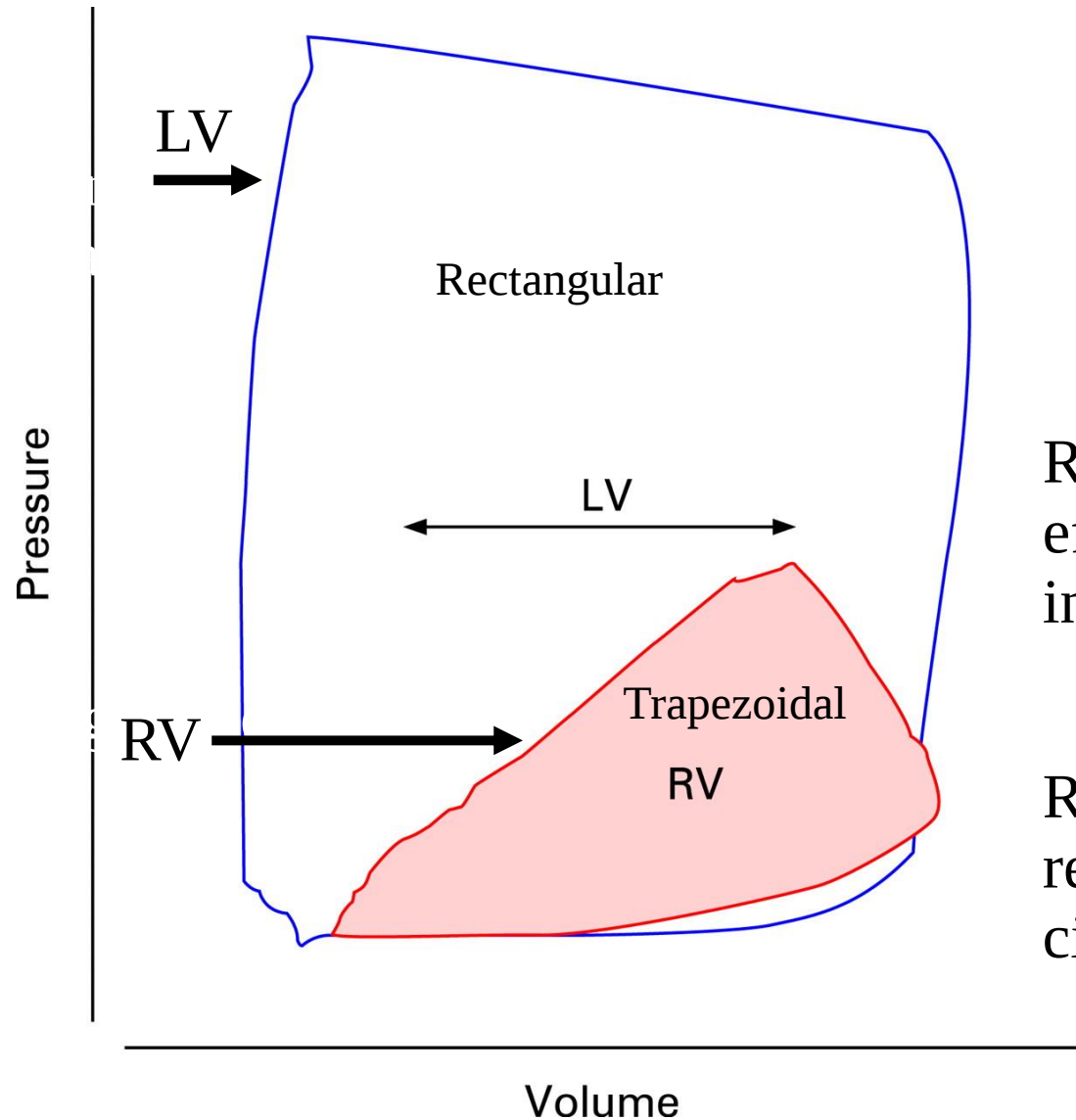
- 1) a capacitor in zone III conditions or
- 2) a collapsible Starling resistor with little wall tone in zone II conditions





Normal volume status, no abdominal hypertension

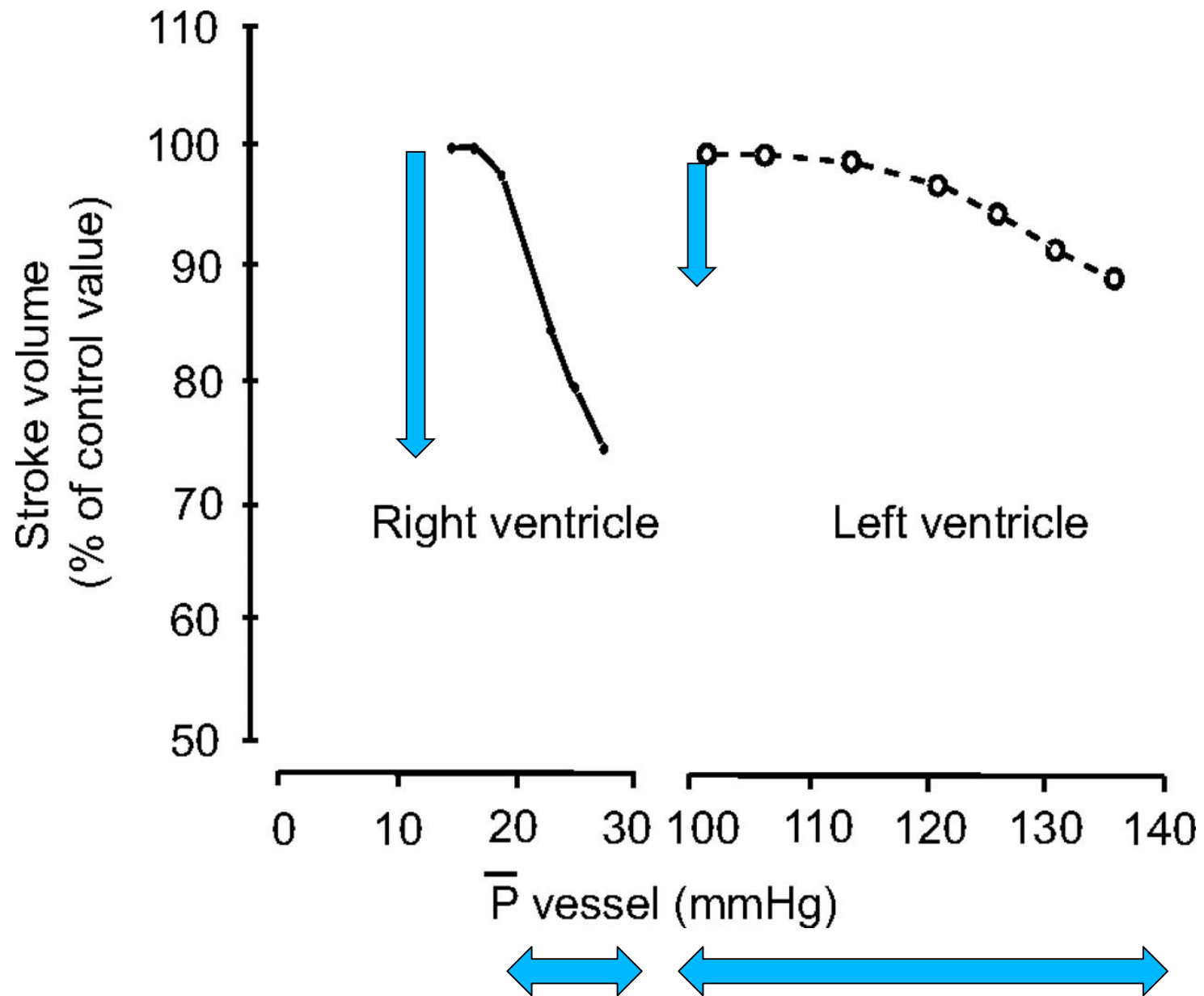
Normal human left (LV) and right ventricular (RV) pressure–volume relationships.

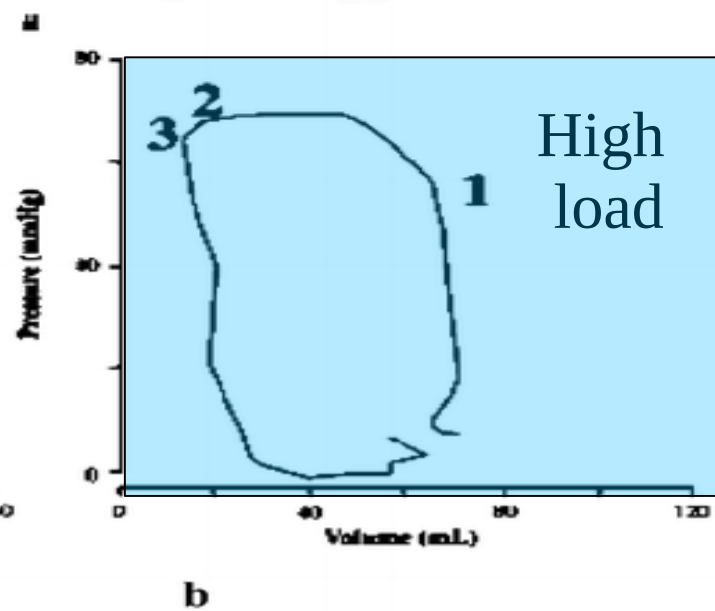
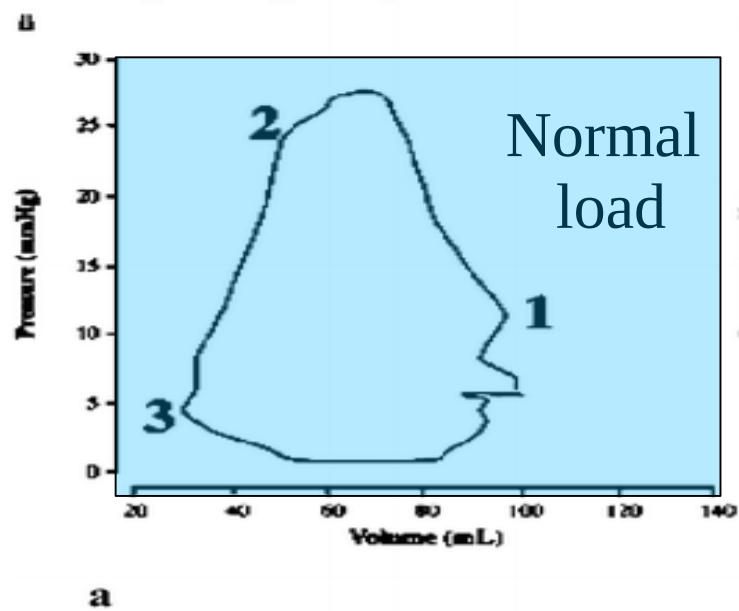
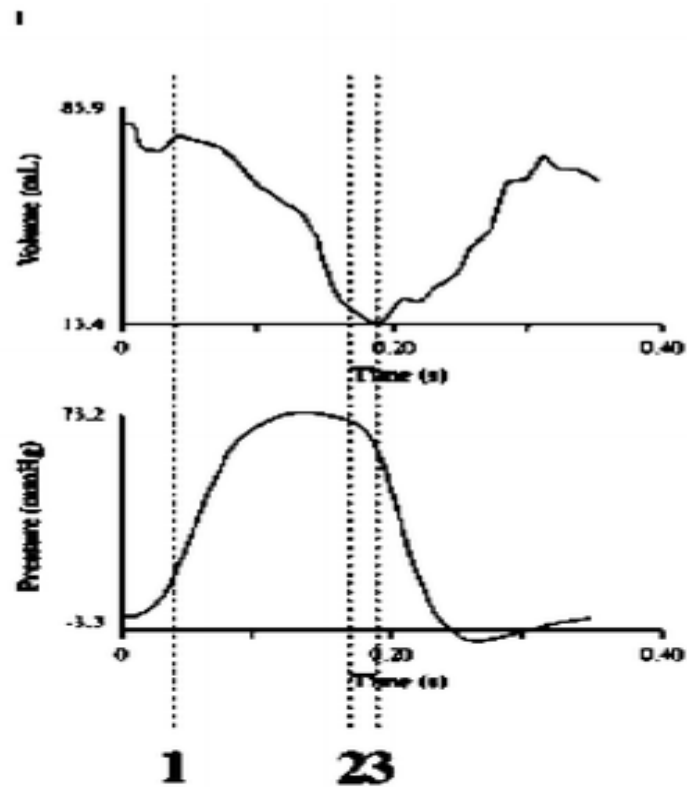
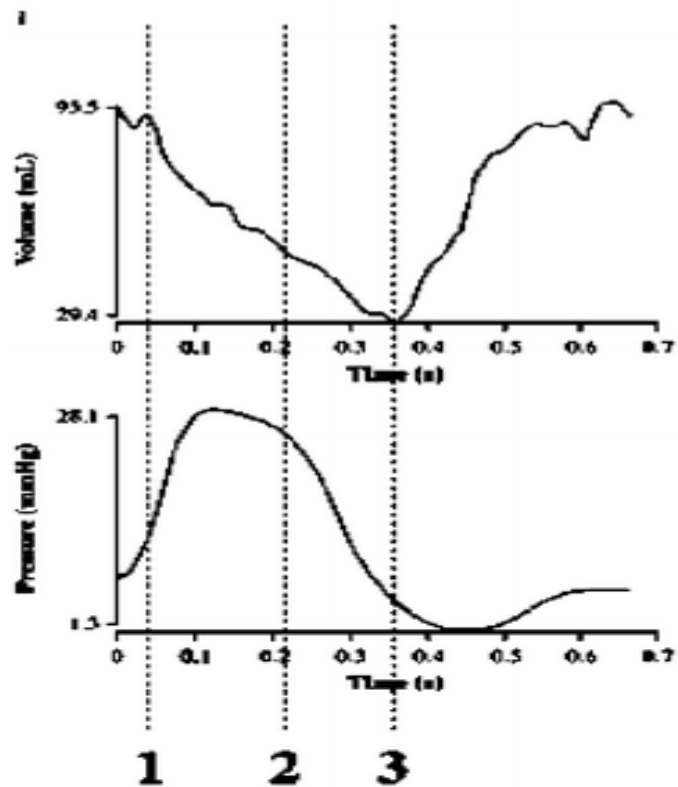


RV can not cope efficiently with sudden increases in after-load

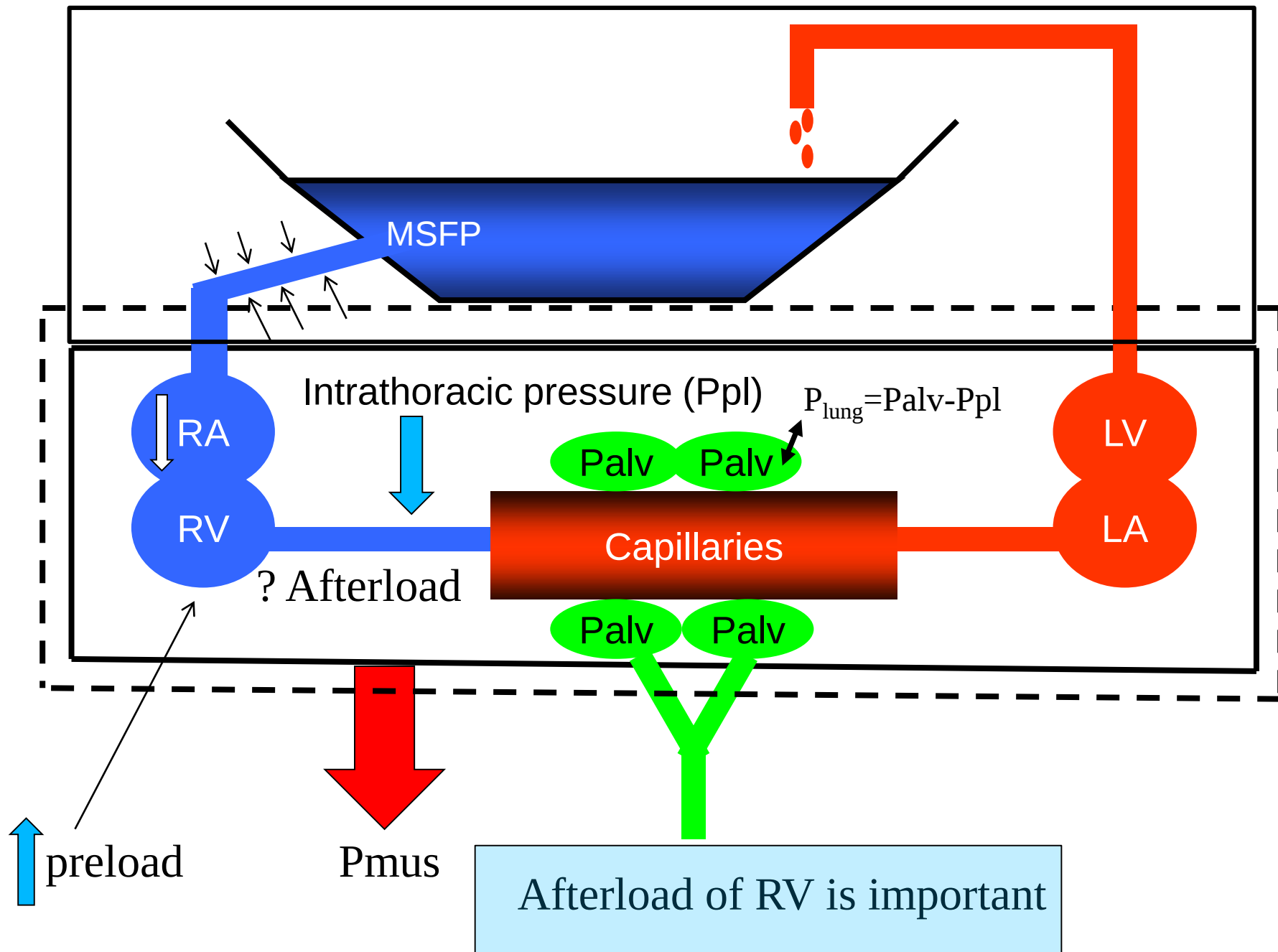
RV ejects into a low resistance, high compliant circuit

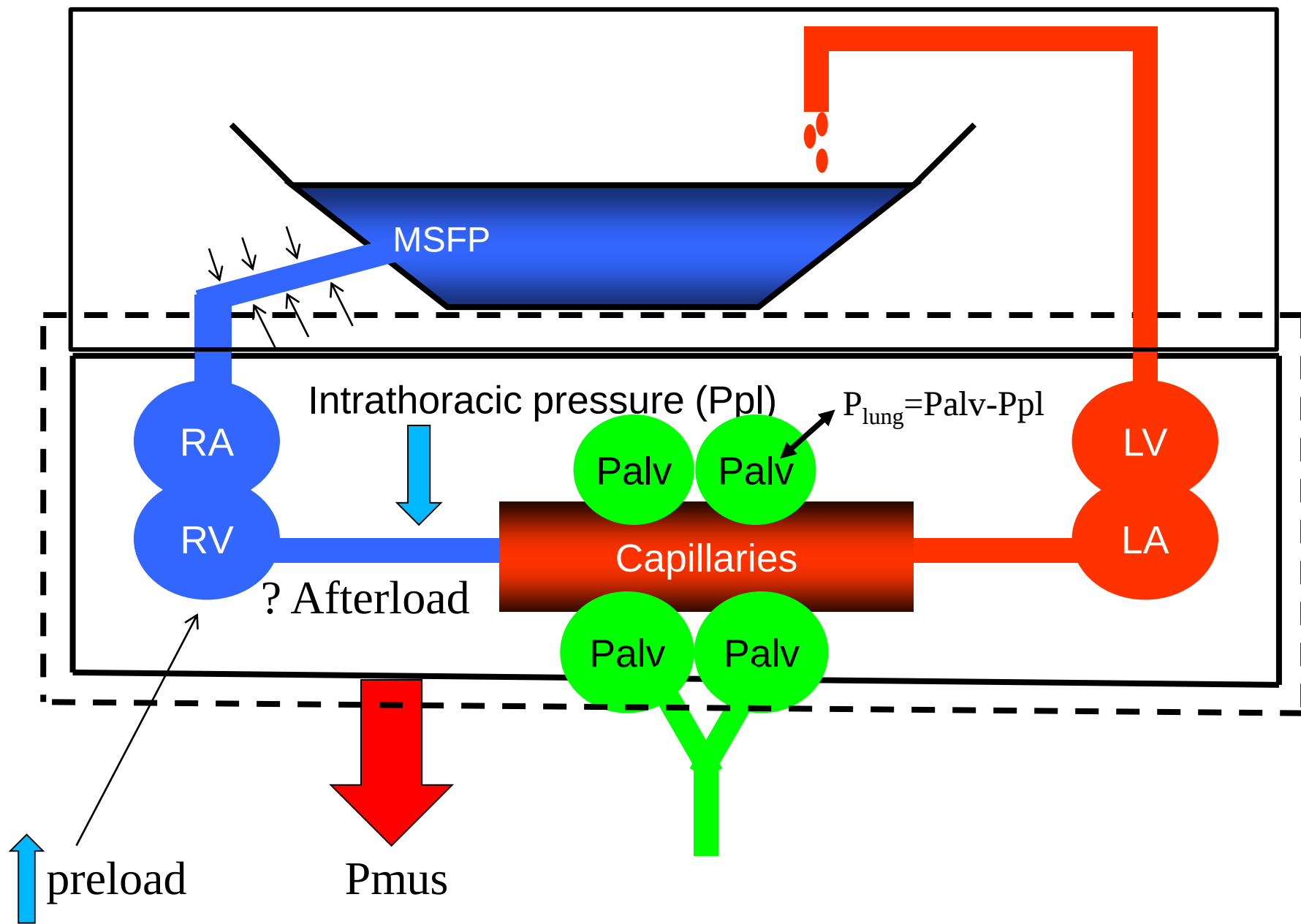
The response of the RV and LV to experimental increase in afterload.



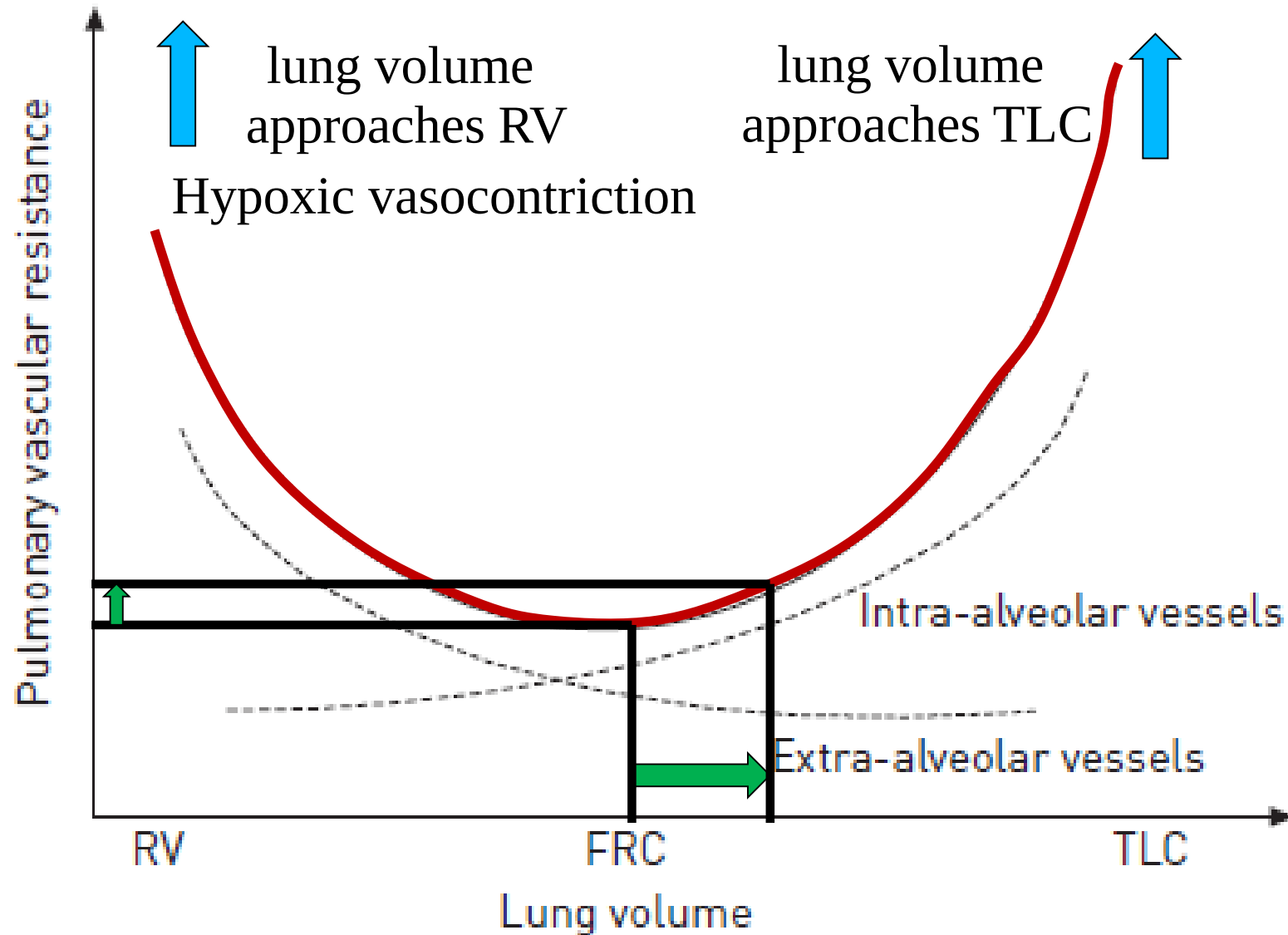


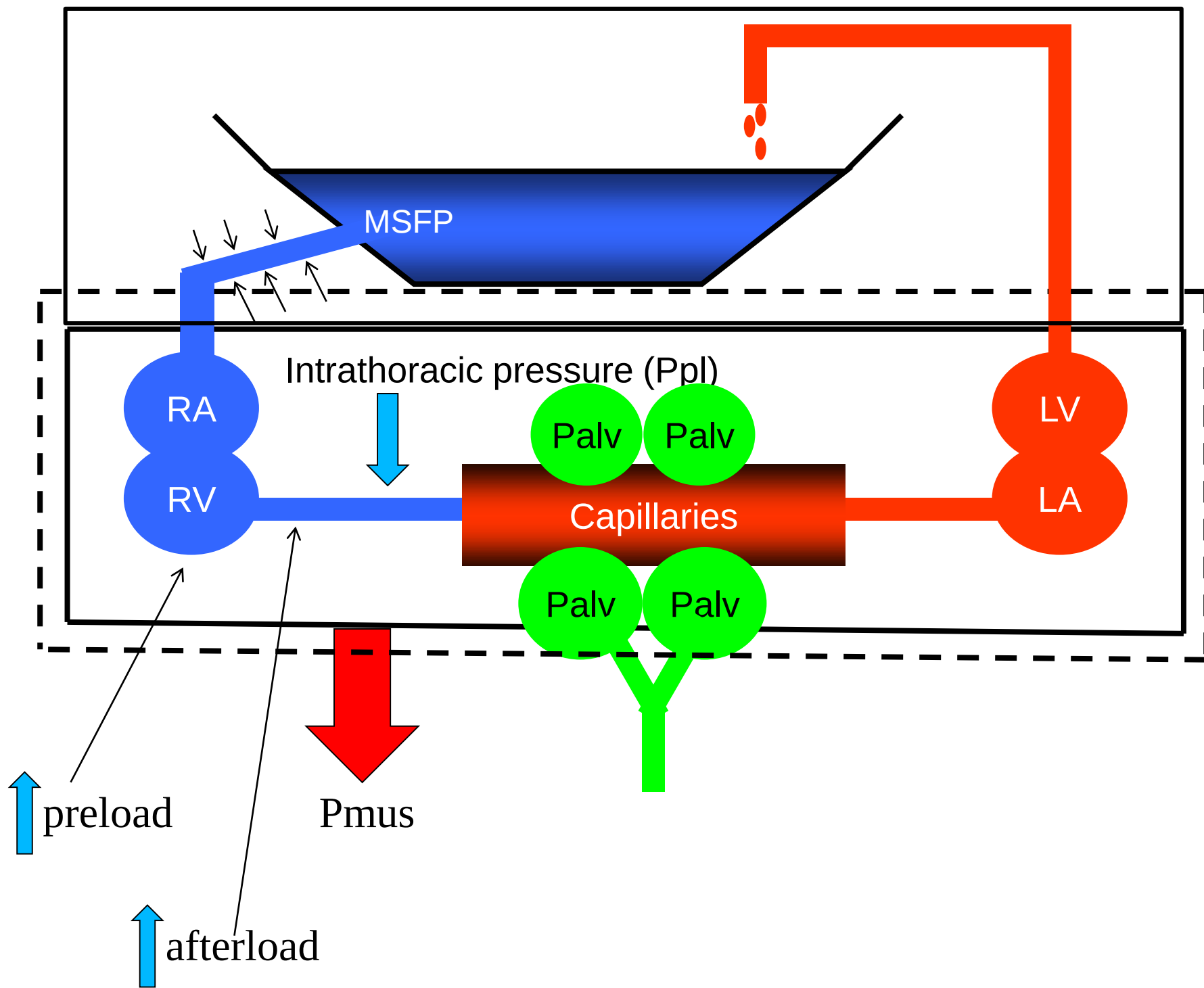
Isometric contraction and relaxation phases

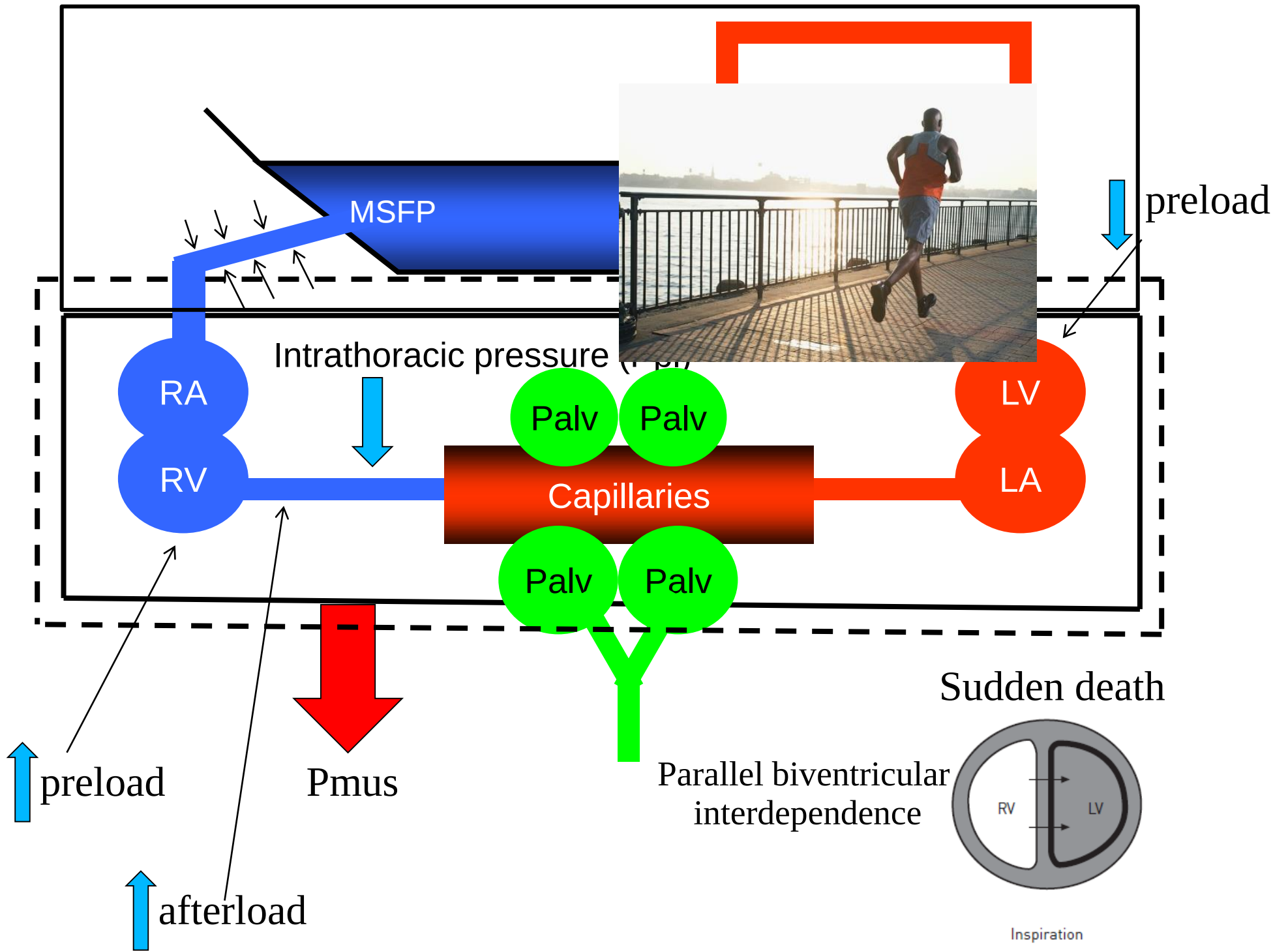


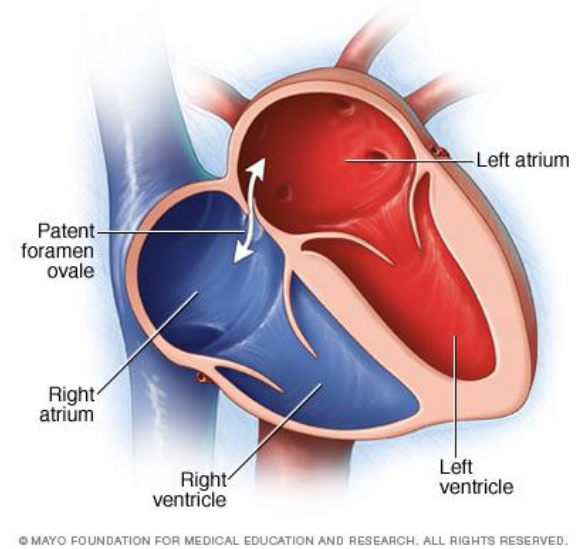
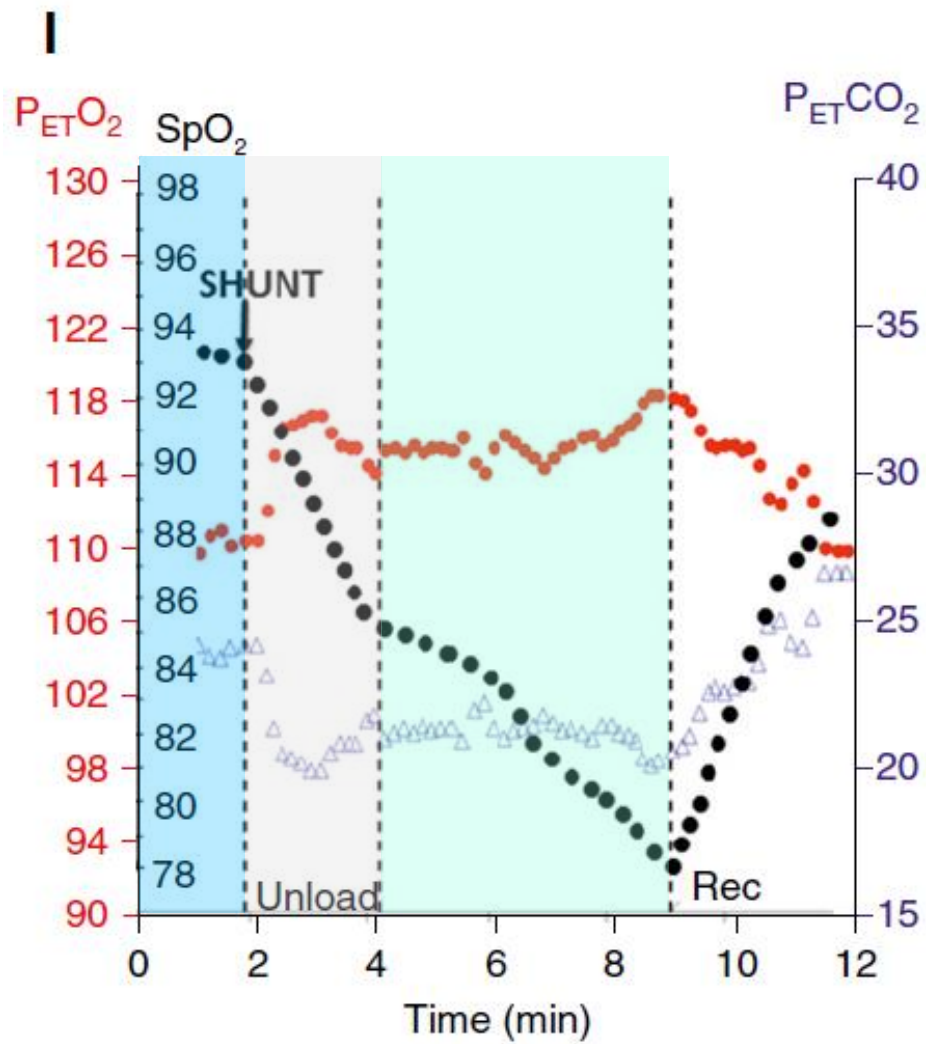


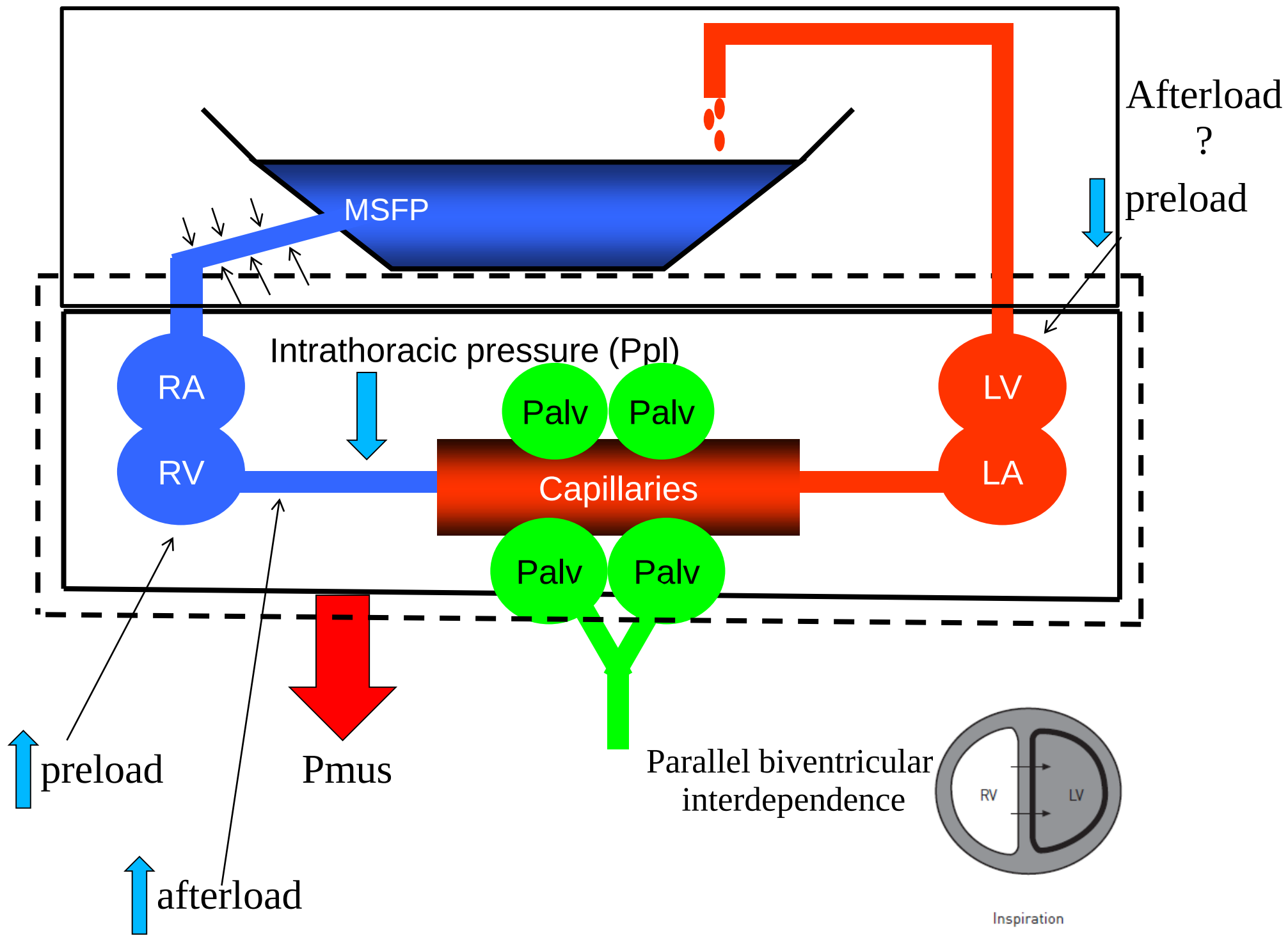
Afterload of the Right Ventricle



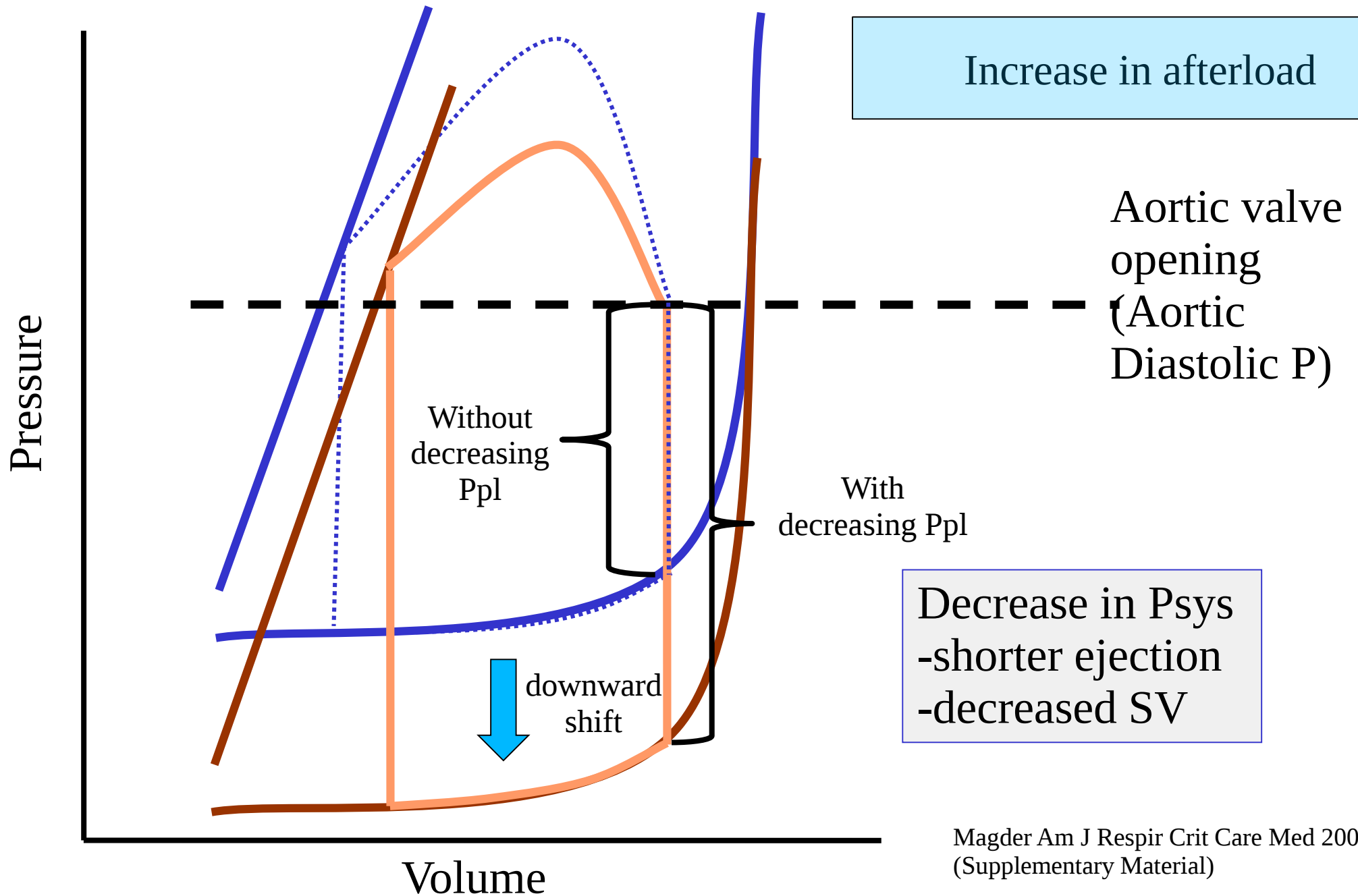


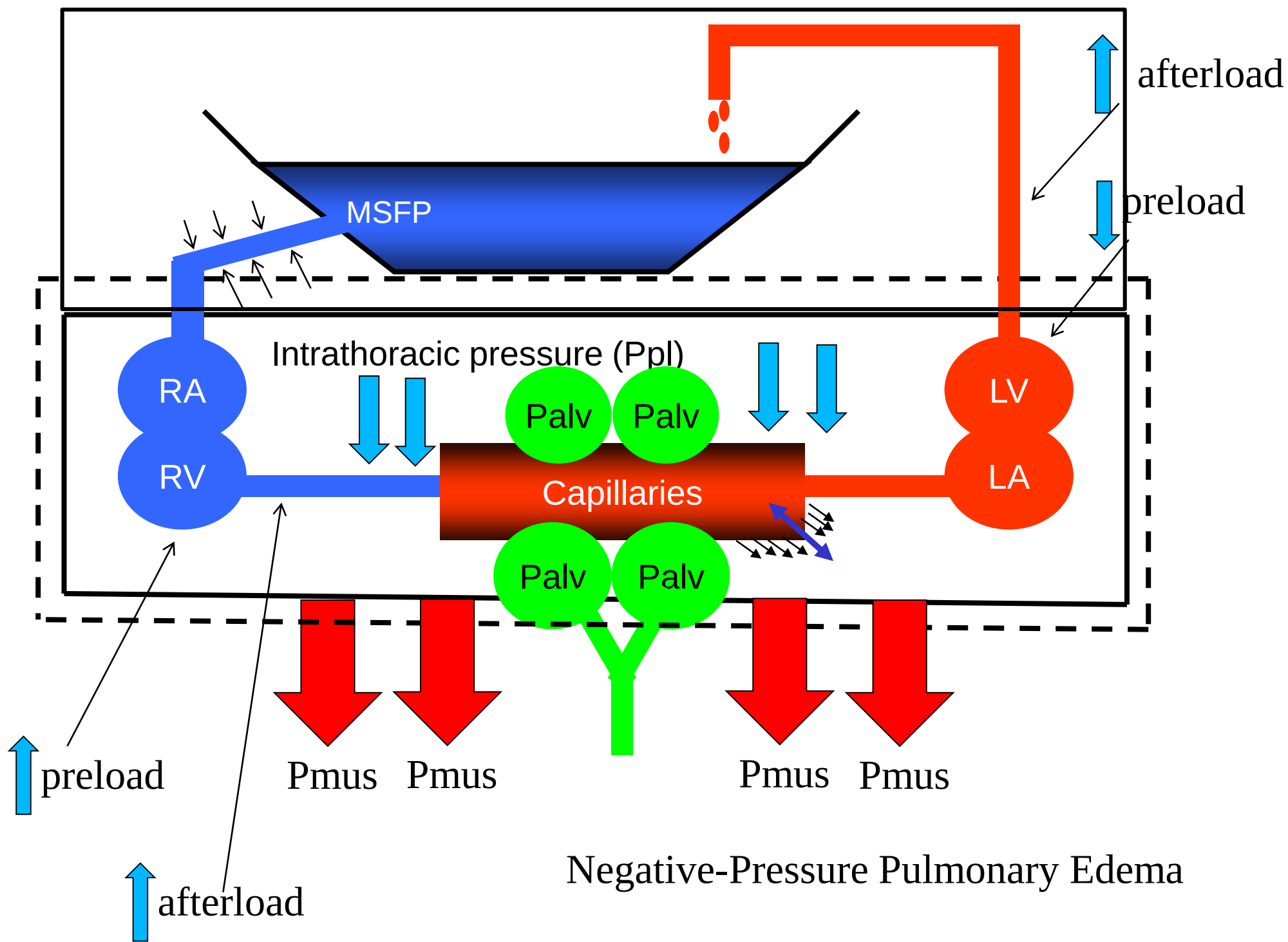






Effect of $\downarrow P_{pl}$ on LV ejection

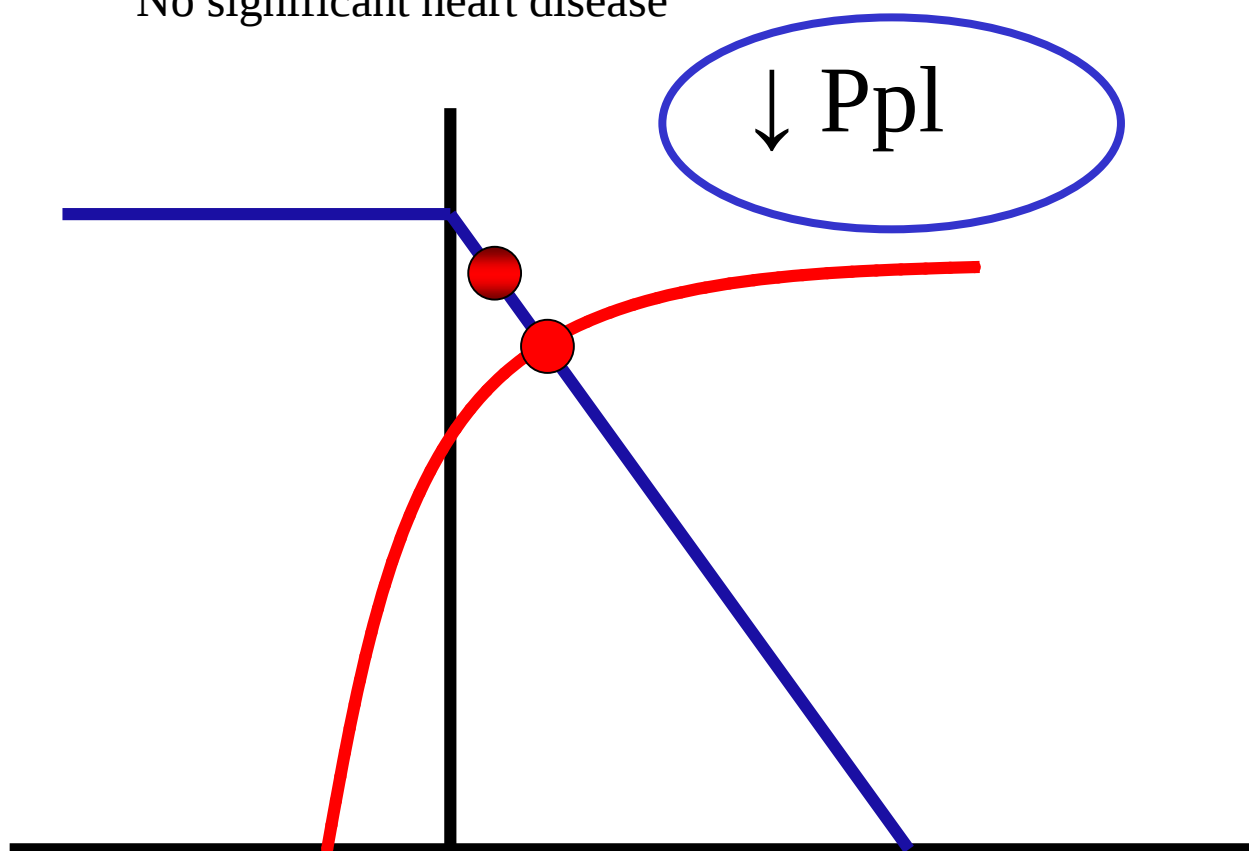




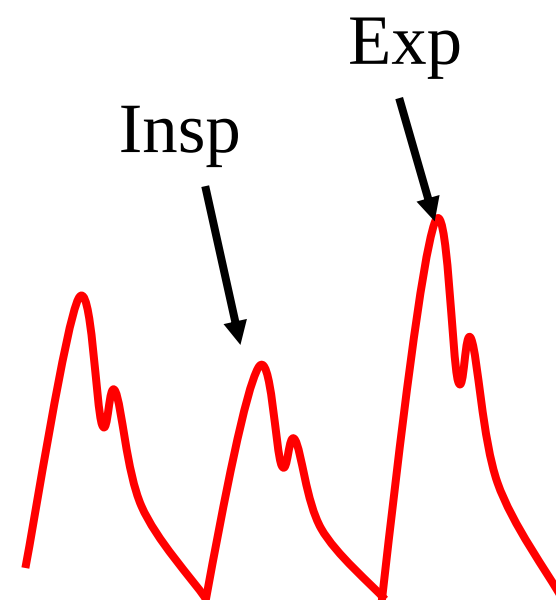
Effect of decrease in Ppl

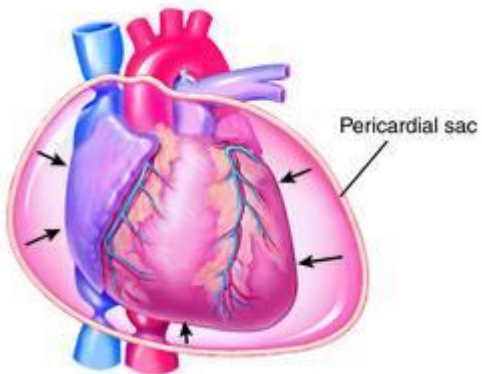
Overall

Volume status: Normal
No significant respiratory system disease
No significant heart disease

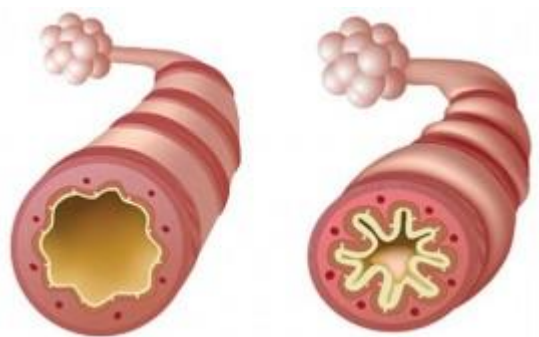


Normal <10 mmHg



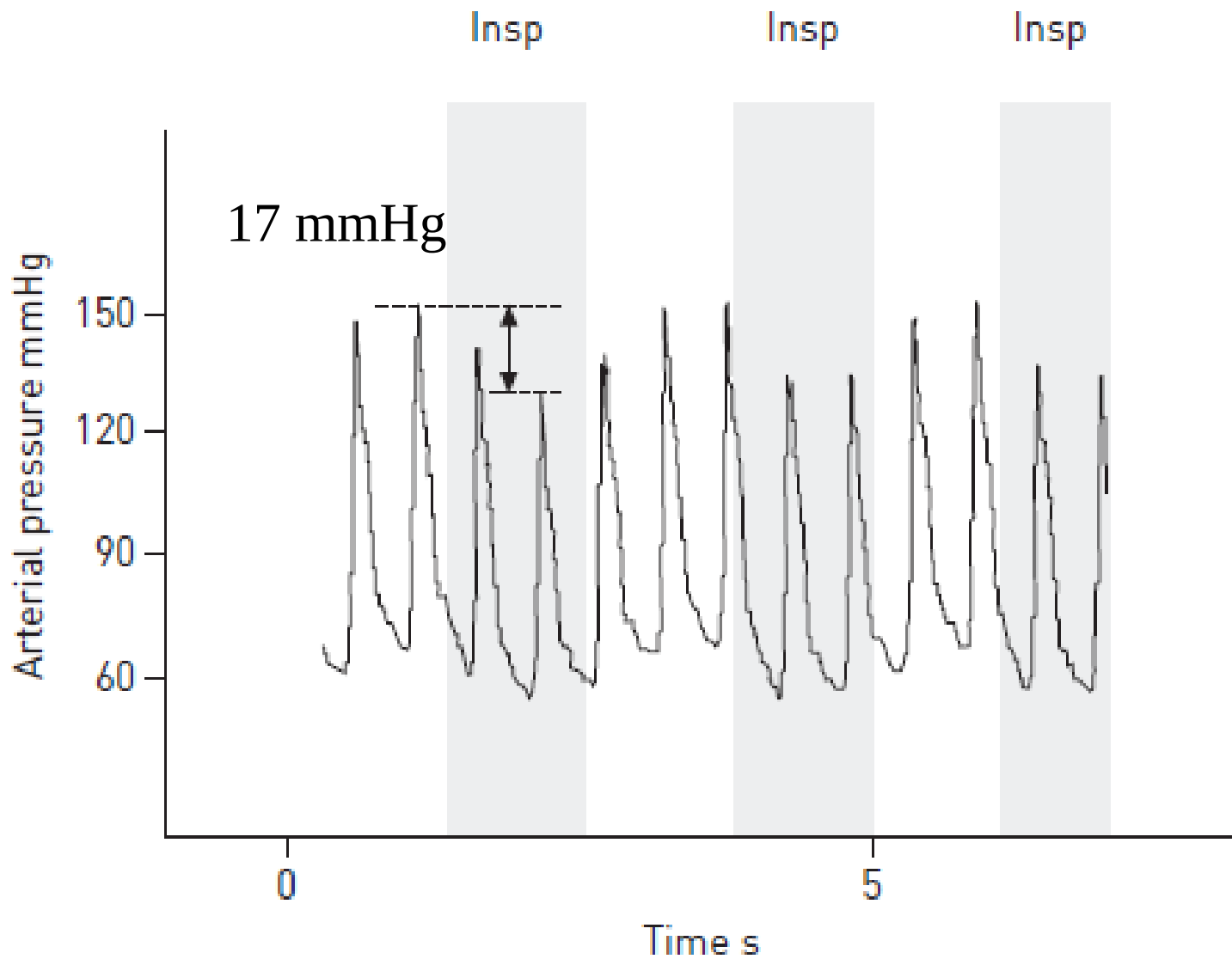


Cardiac tamponade

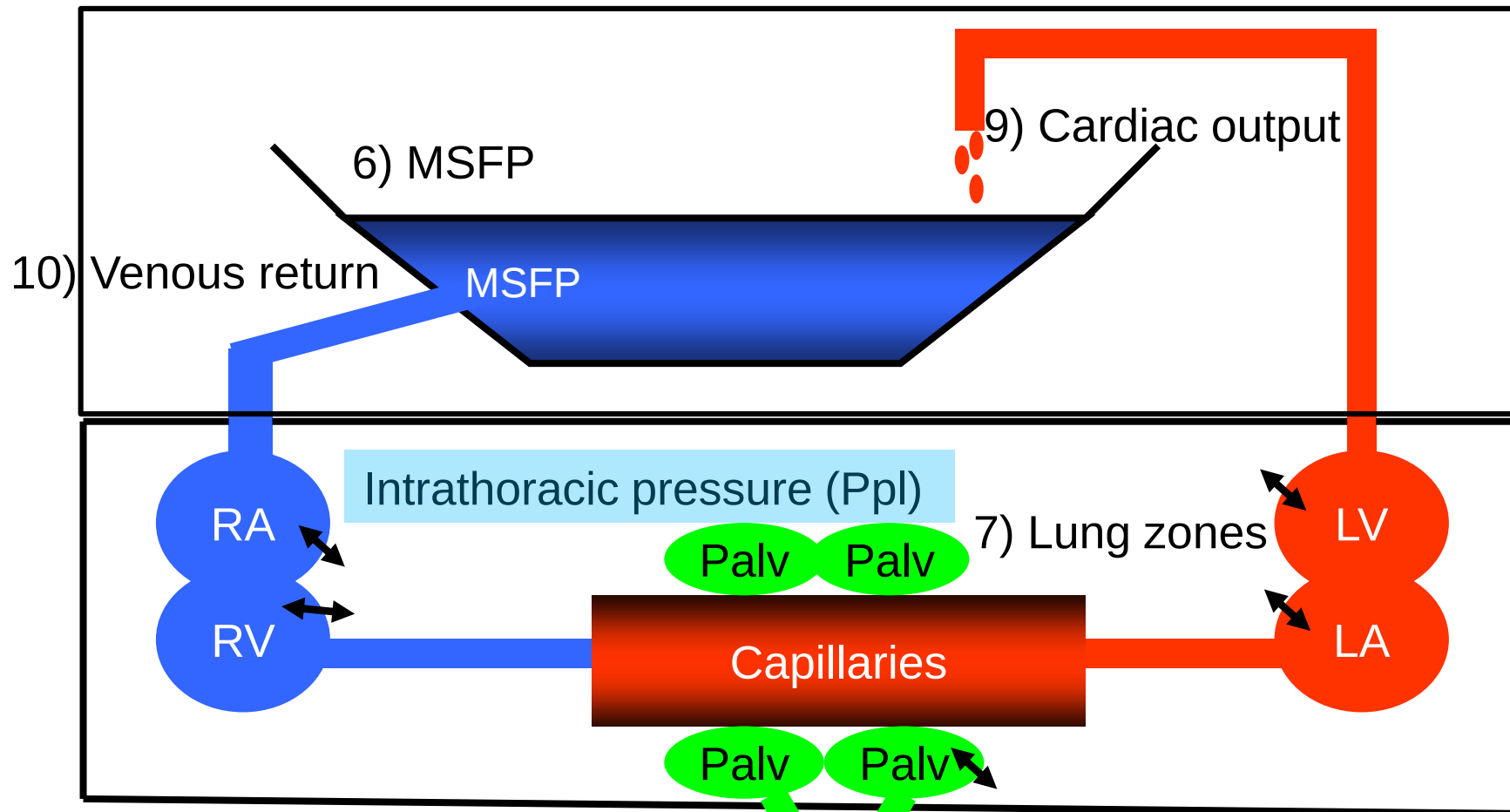


Severe asthma attack
(status asthmaticus)

Pulsus paradoxus



Let's place the patient on MV (passive)



**Mechanical ventilation
changes everything**

- 1) Autonomic tone
- 2) P_{alv}
- 3) P_{pl}

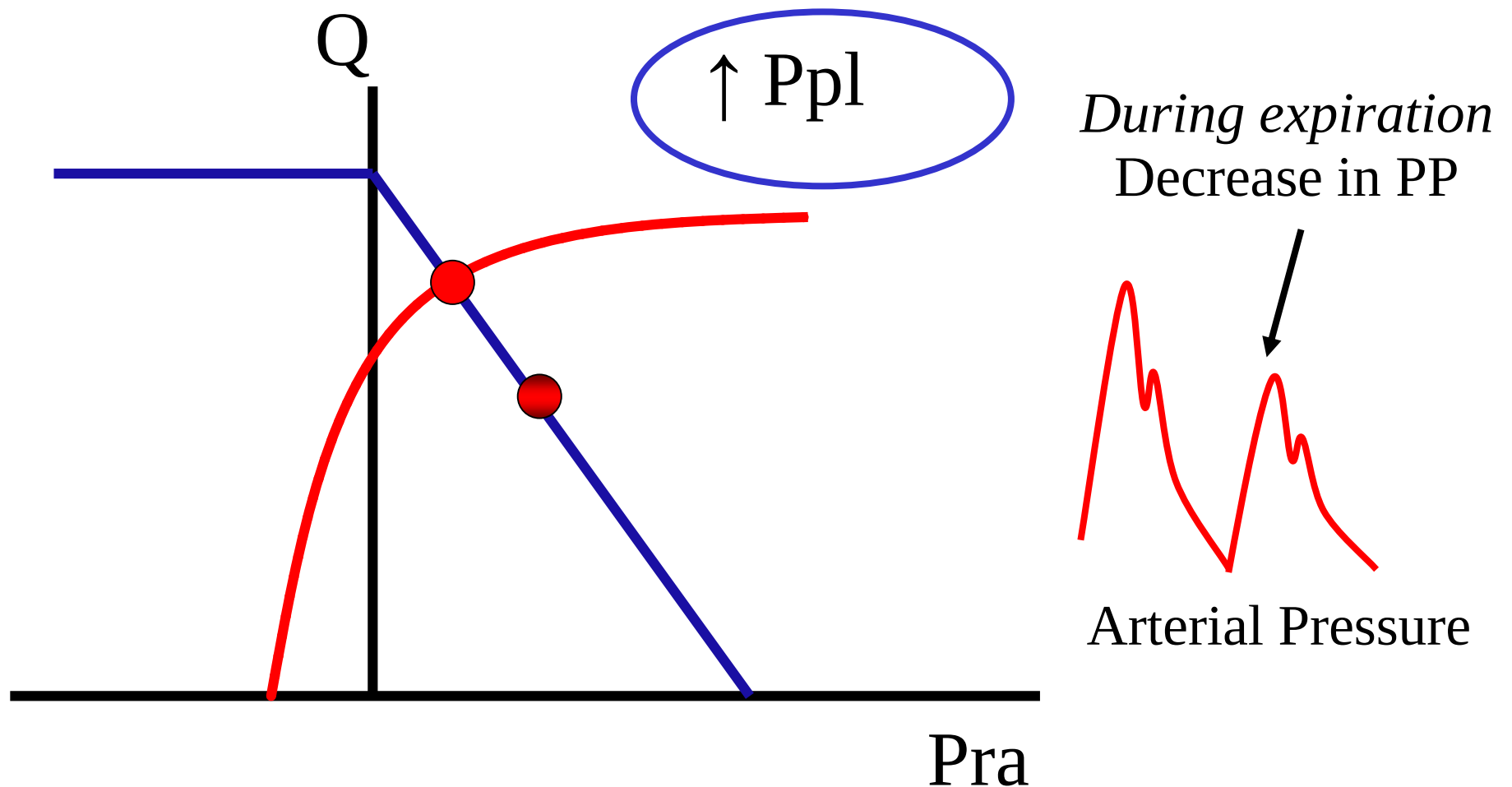
Passive



- 4) Trans-vascular pressure
- 5) Trans-pulmonary pressure
- 8) Lung compliance

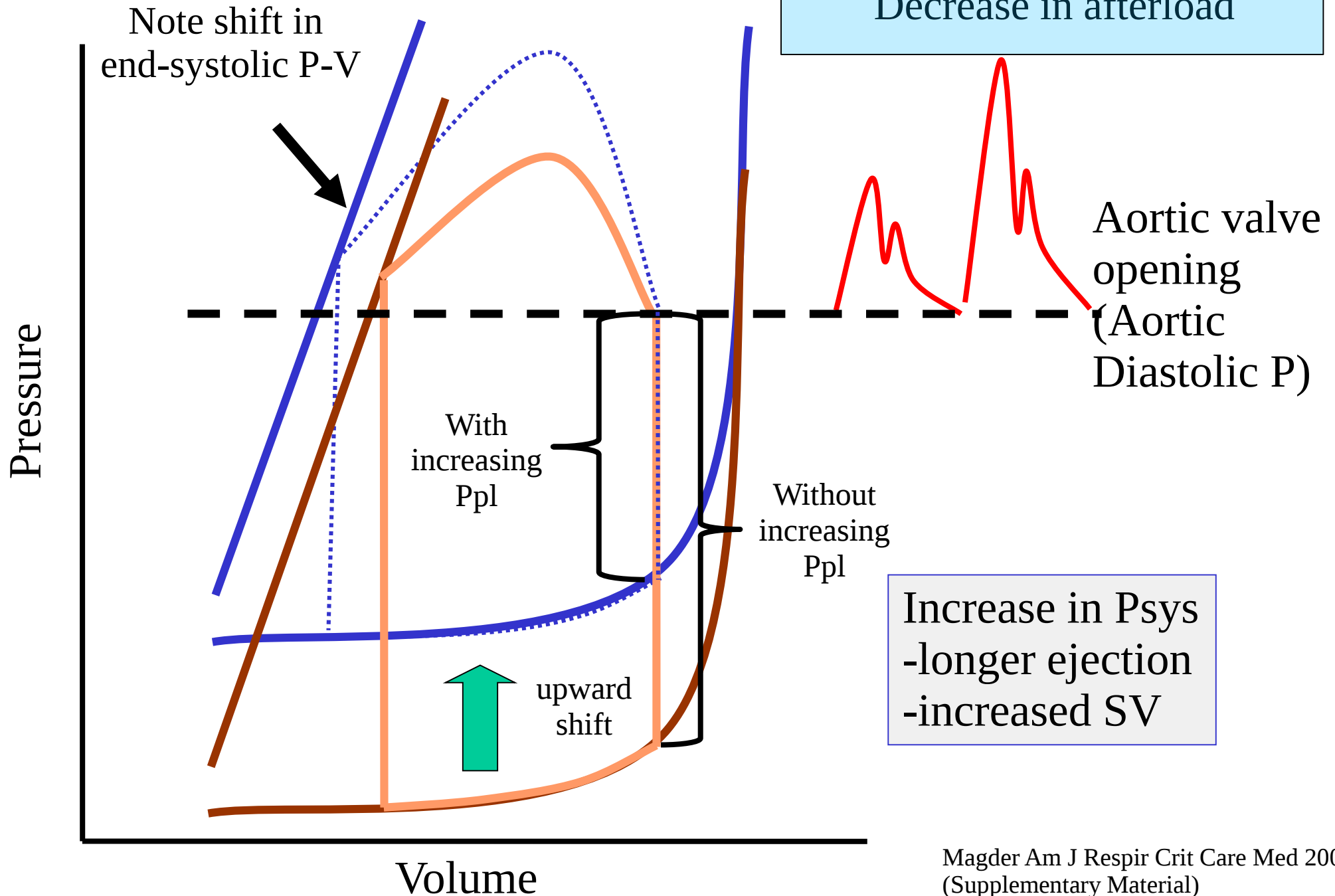
11) Juxtacardiac pressures

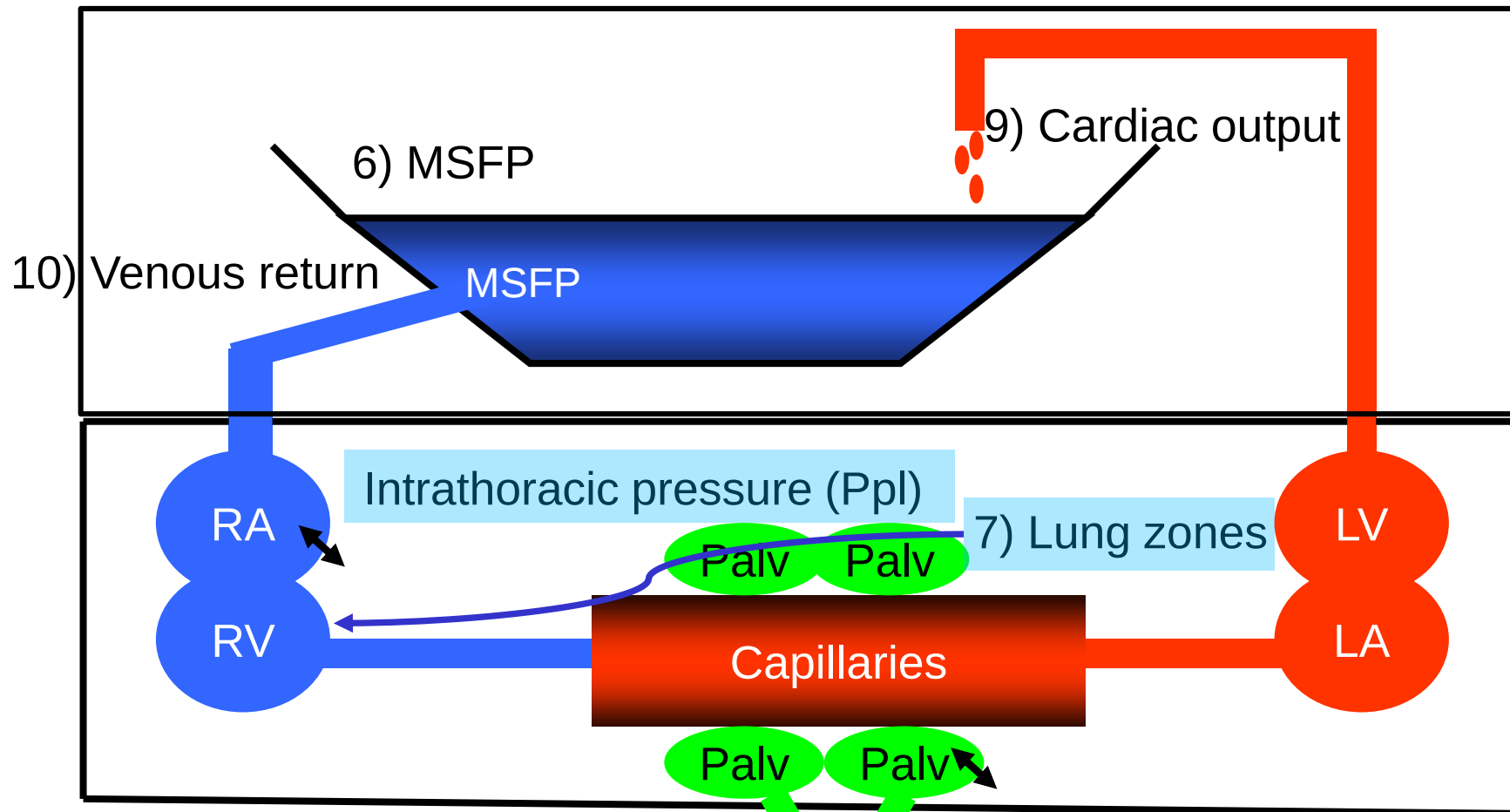
Effect of increase in Ppl



Left
ventricle

Effect of $\uparrow$ Ppl on LV ejection





Mechanical ventilation changes everything

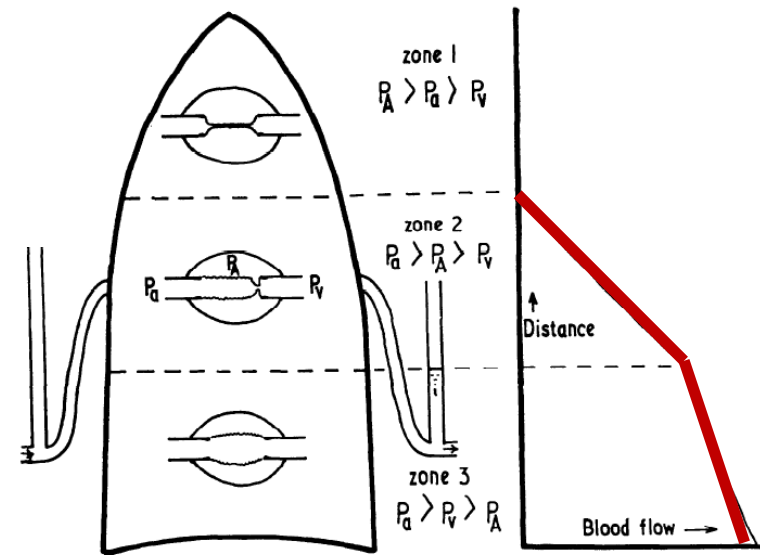
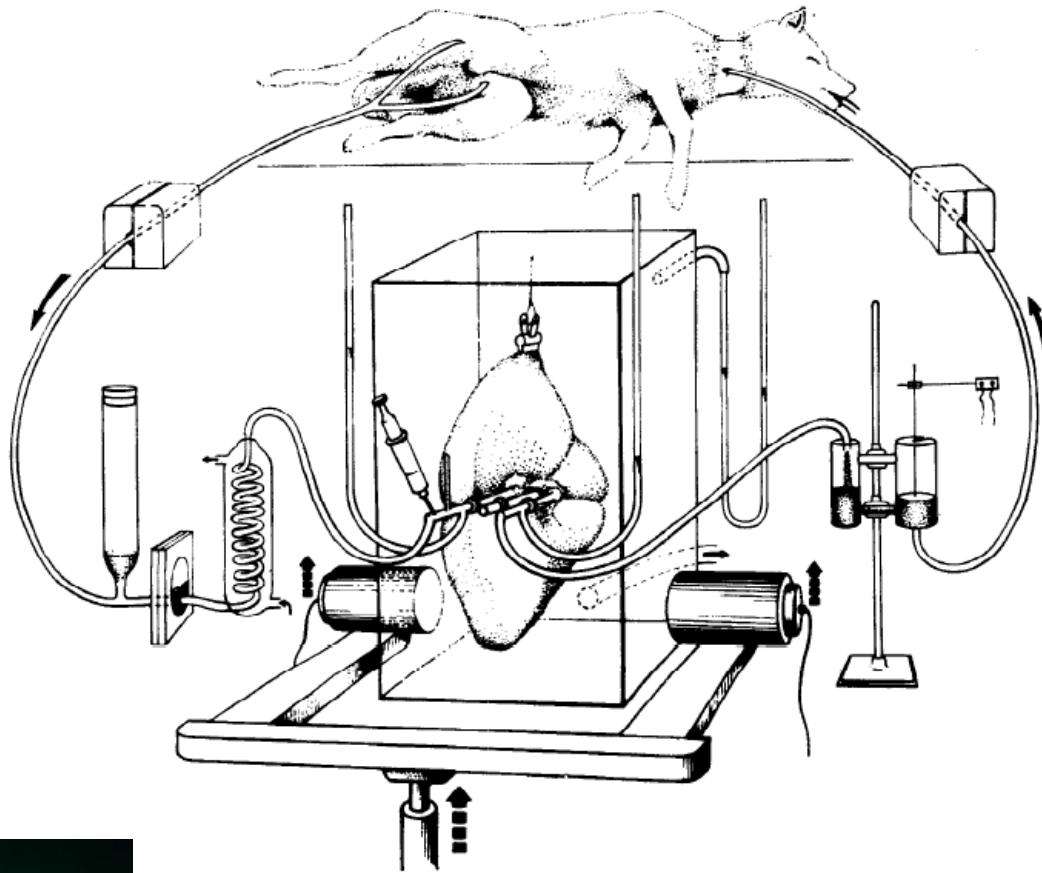
- 1) Autonomic tone
- 2) Palv
- 3) Ppl

Passive



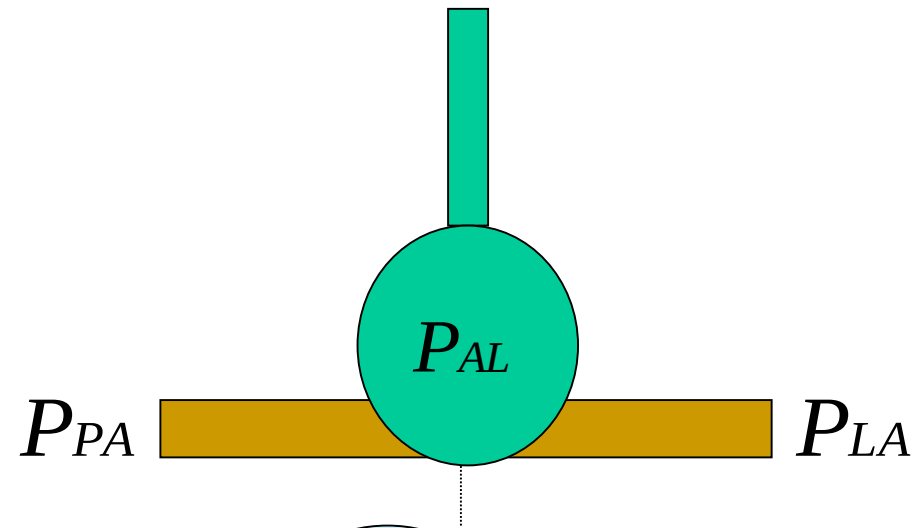
- 4) Trans-vascular pressure
- 5) Trans-pulmonary pressure
- 8) Lung compliance

11) Juxtacardiac pressures



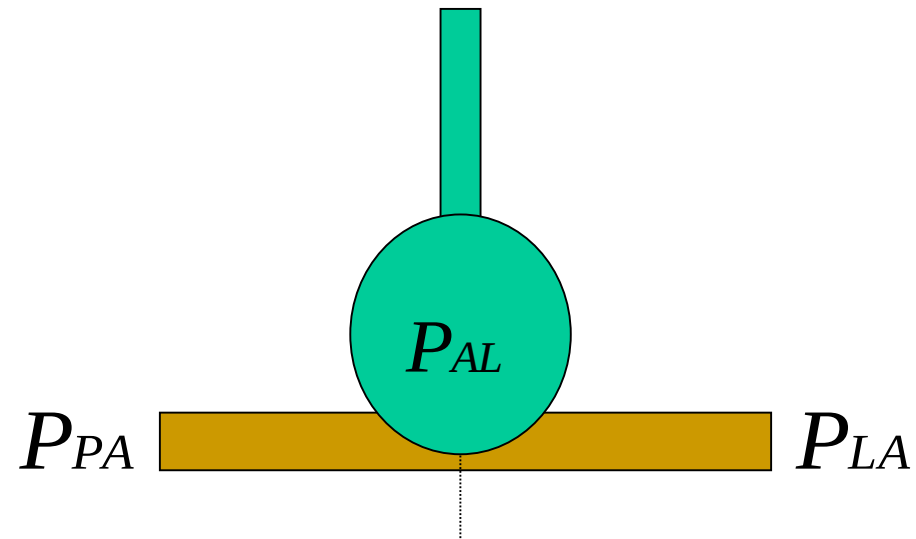
West et al. J Appl Physiol 1964

Zone I



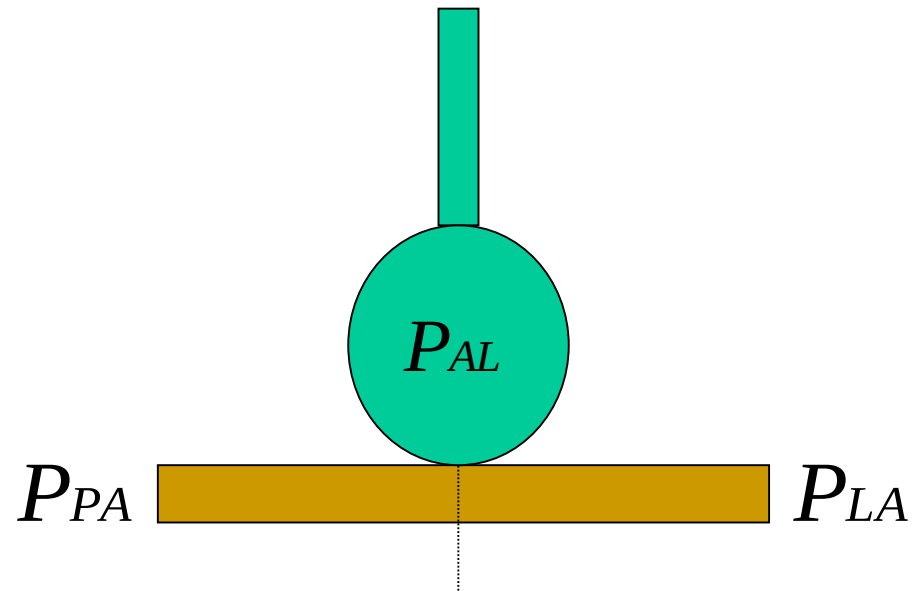
- $P_{AL} > P_{PA}$ and hence, there is **no flow** under these conditions

Zone II



- $P_{PA} > P_{AL} > P_{LA}$ and hence, flow is dependent on the pressure gradient $P_{PA} - P_{AL}$ rather than $P_{PA} - P_{LA}$

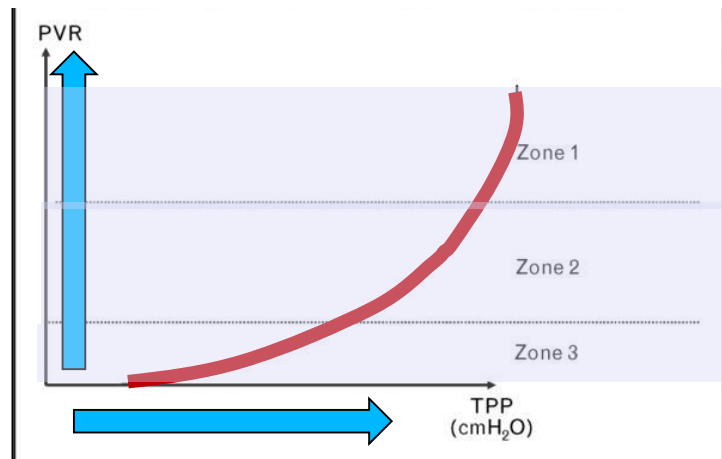
Zone III



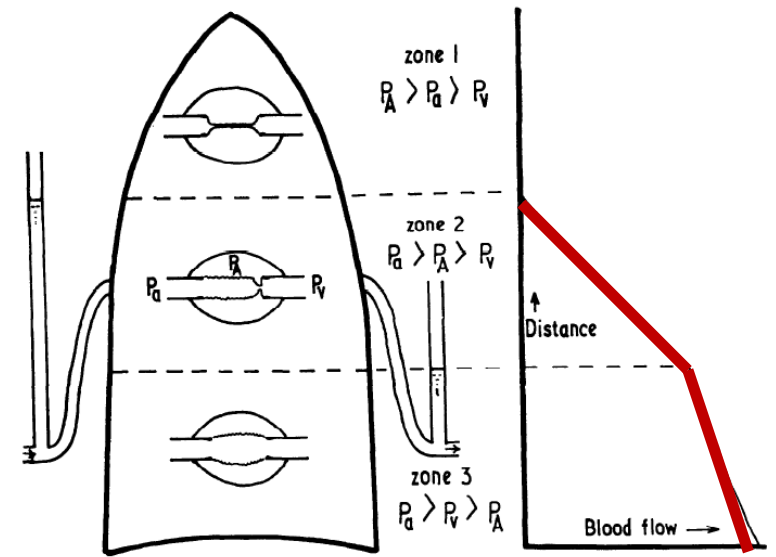
- $P_{PA} > P_{LA} > P_{AL}$ and hence, flow is dependent on the pressure gradient $P_{PA} - P_{LA}$

Why are lung zones important for after-load of RV?

Lung zones



↑ After-load of RV



Bouferrache and Vieillard-Baron:
Current Opinion in Critical Care 2011, 17:30–35

P_{lung} ($P_{\text{alv}} - P_{\text{pl}}$) is the critical variable
(and not only P_{alv})

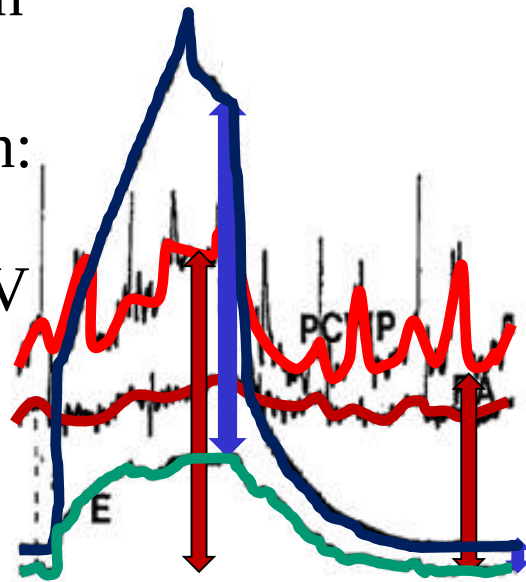
Vieillard-Baron et al. J Appl Physiol 1999; 87:1644



RV suffers
during inspiration

INSP.

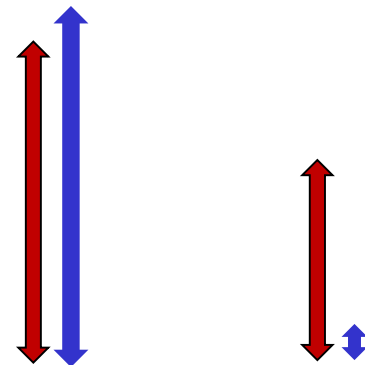
EXP.



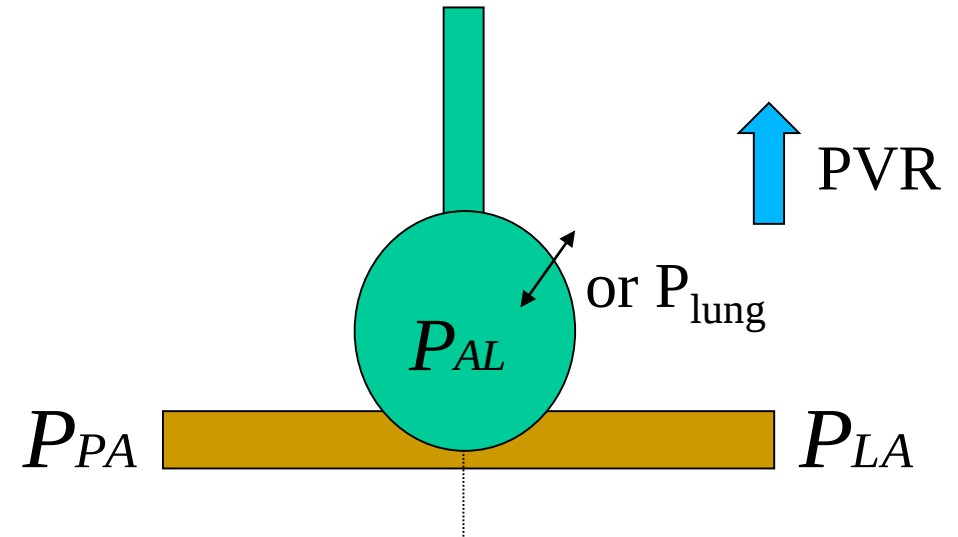
During the breath:
Cyclic changes
in afterload of RV

Let's change V_T

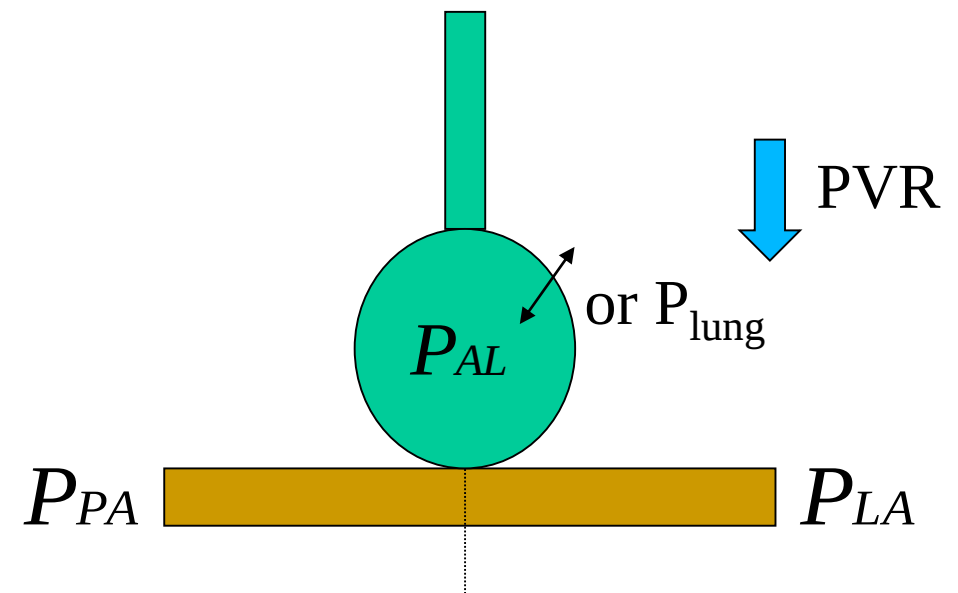
Change in V_T
changes P_{lung}



End inspiration: Zone II



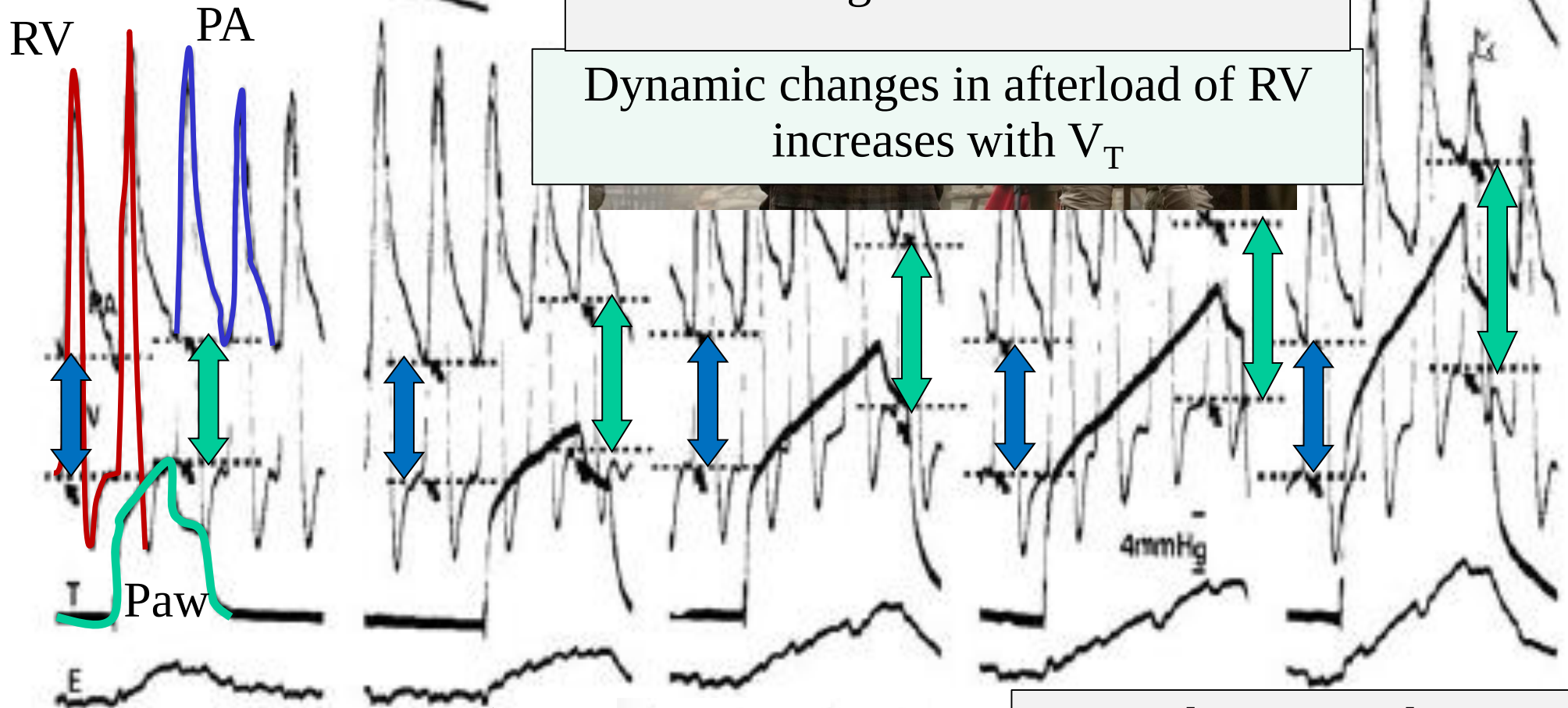
End expiration: Zone III





Static change in afterload of RV?

Dynamic changes in afterload of RV increases with V_T

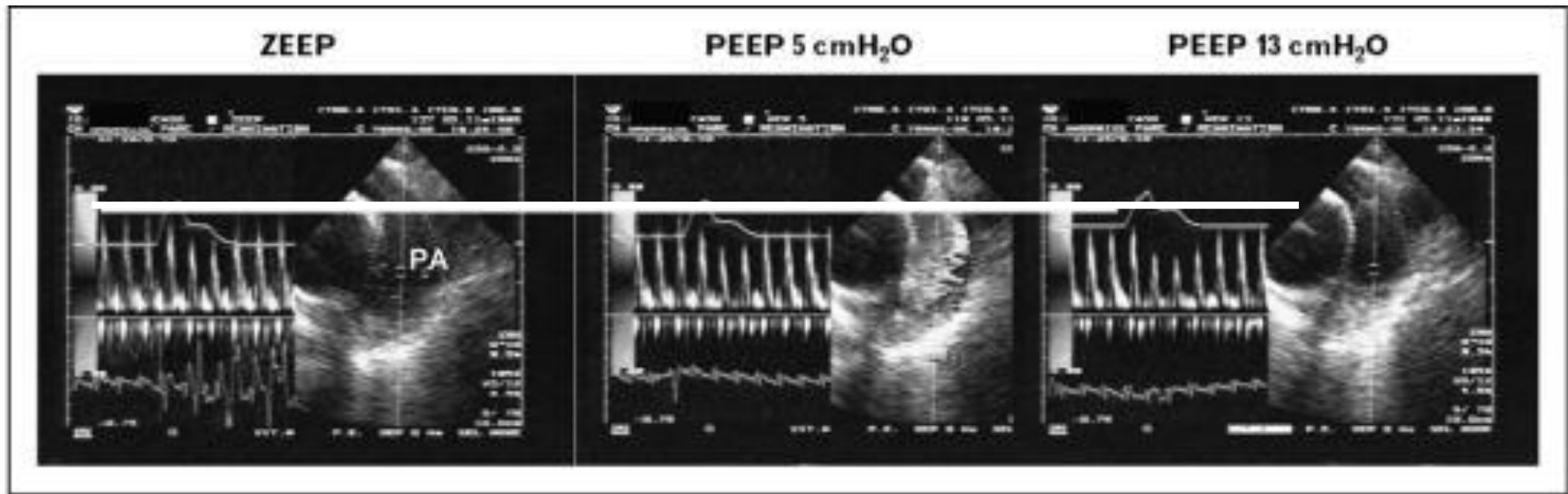


RV needs more and more pressure to open the door to pulmonary circulation at end inspiration



Increasing PEEP caused a progressive decrease in RV ejection flow during expiration

Bouferrache and Vieillard-Baron:
Current Opinion in Critical Care 2011



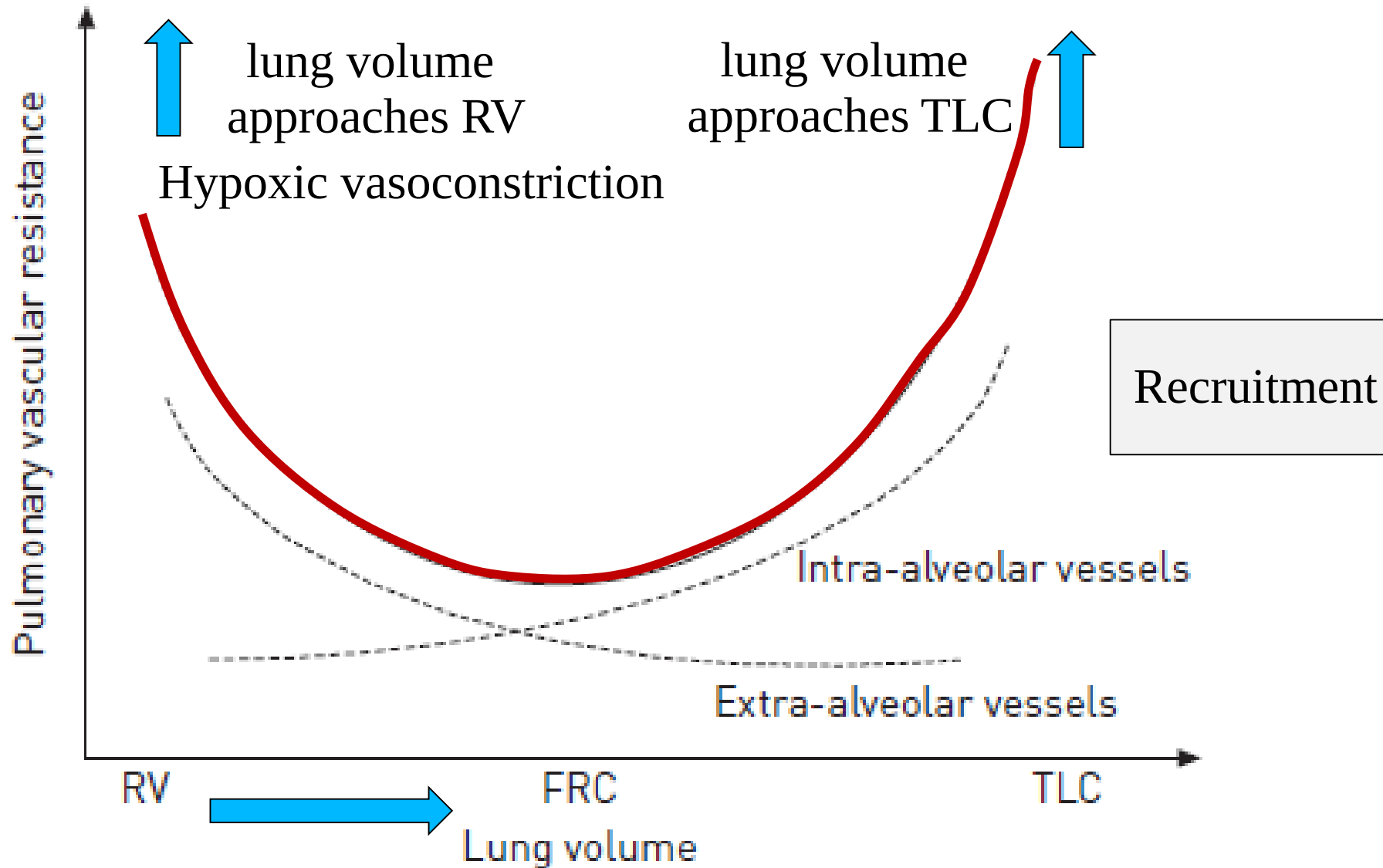
Static increase in transpulmonary pressure (PEEP application)
Static increase in afterload of Right ventricle



HFVO
Quasi-static increase in Palv

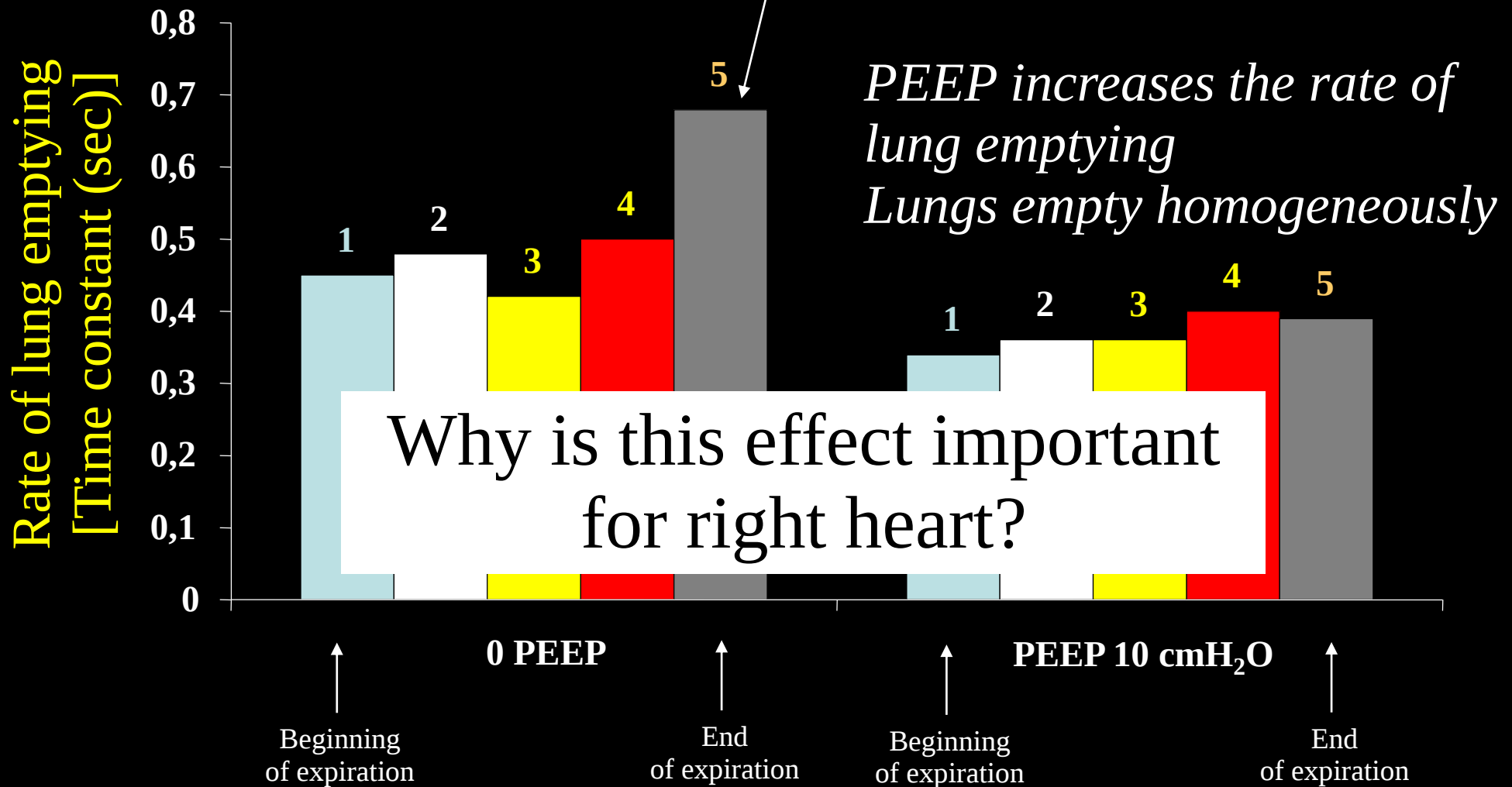
May PEEP have (direct) beneficial effect on Right Ventricle?

Afterload Right ventricle



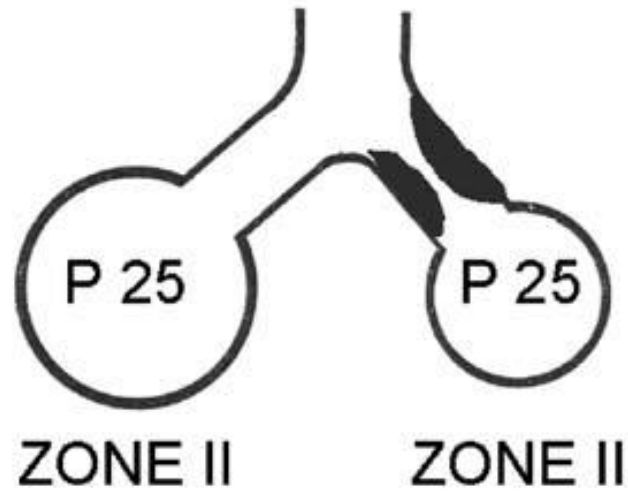
Rate of lung emptying of respiratory system in ARDS patients with and without PEEP (beginning to end of expiration)

Time constant increases abruptly at end-expiration (↓ lung emptying)

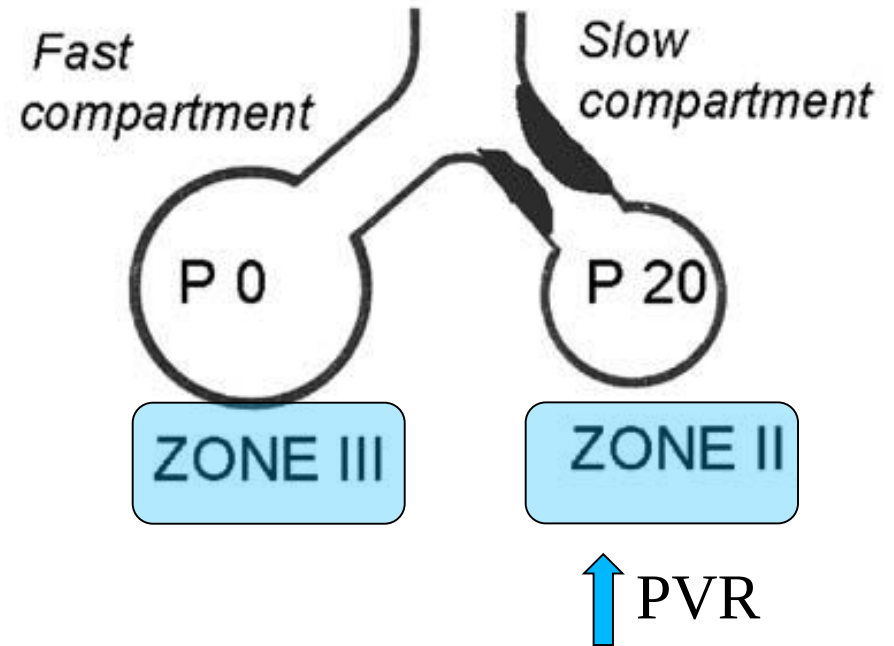


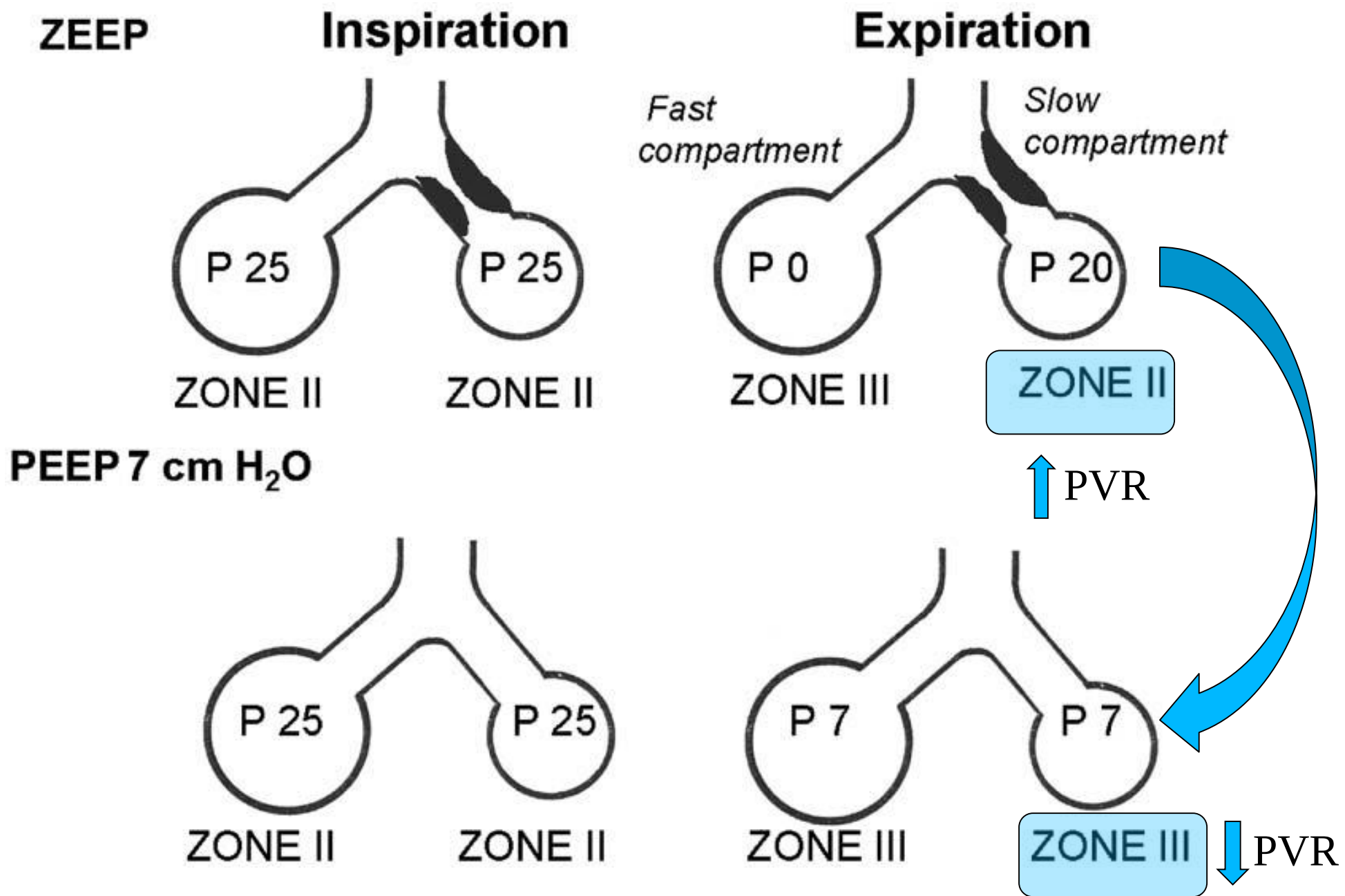
ZEEP

Inspiration

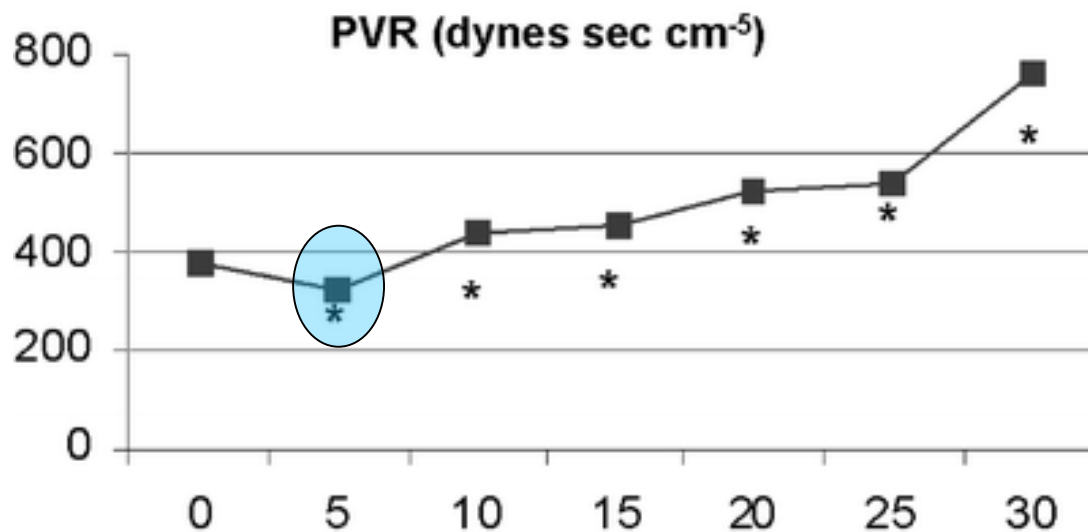


Expiration



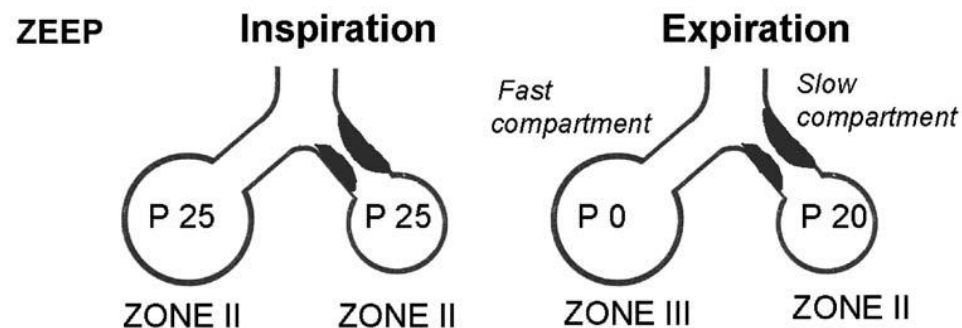


PEEP
may decrease the afterload
of RV

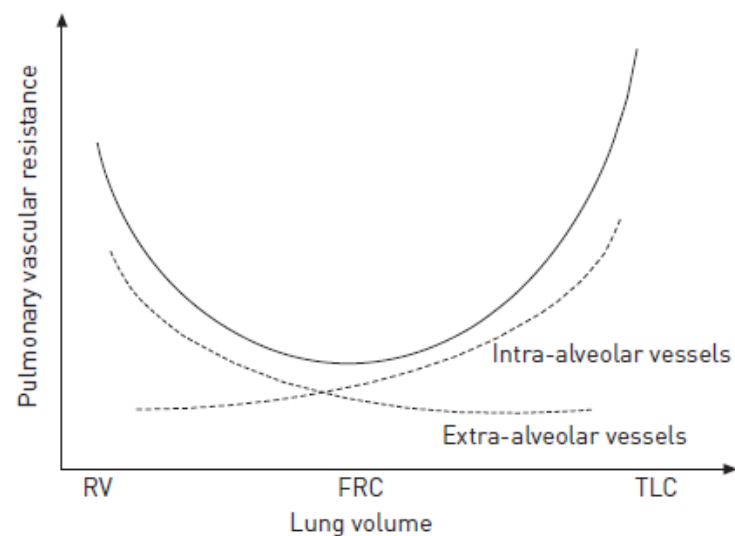
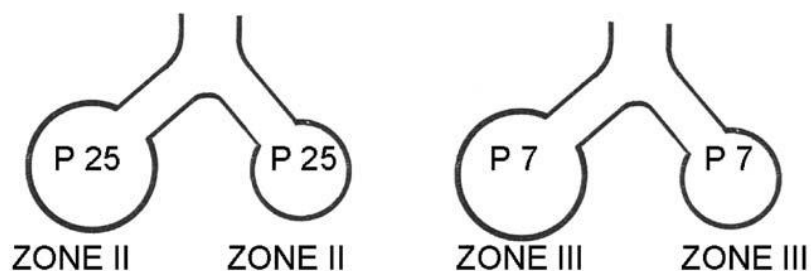


PEEP (cm H₂O)

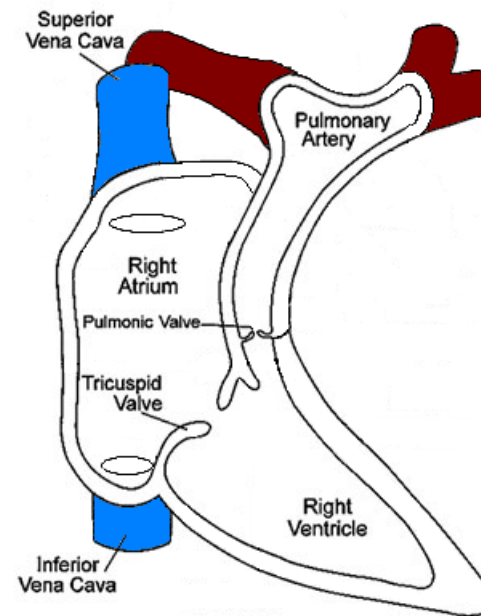
Jardin et al. Intensive Care Med 2003;29:1426



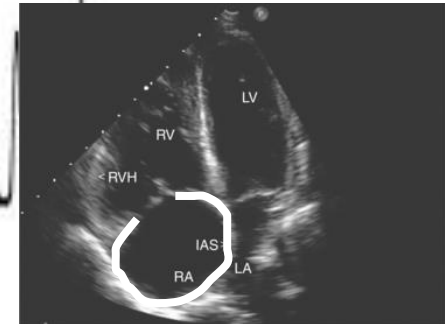
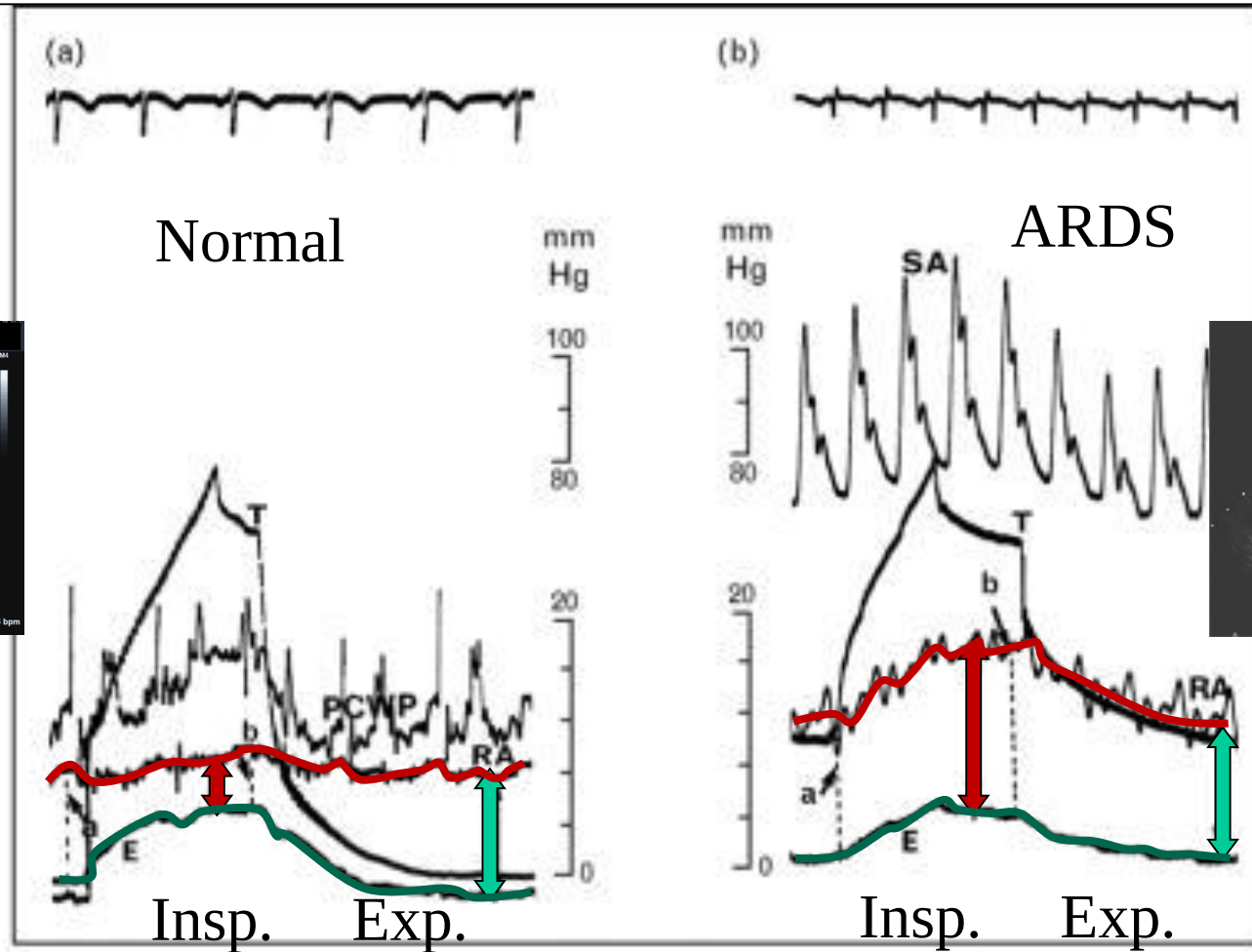
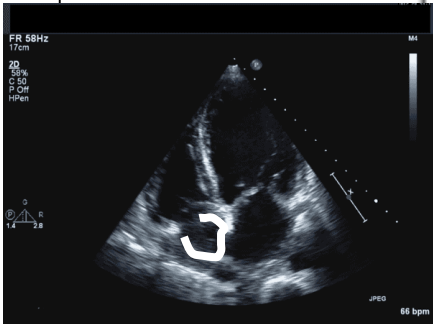
PEEP 7 cm H₂O



Respiratory system mechanics – Right heart interaction during mechanical ventilation



WHY?



Pressure
across the RA

↕

↕

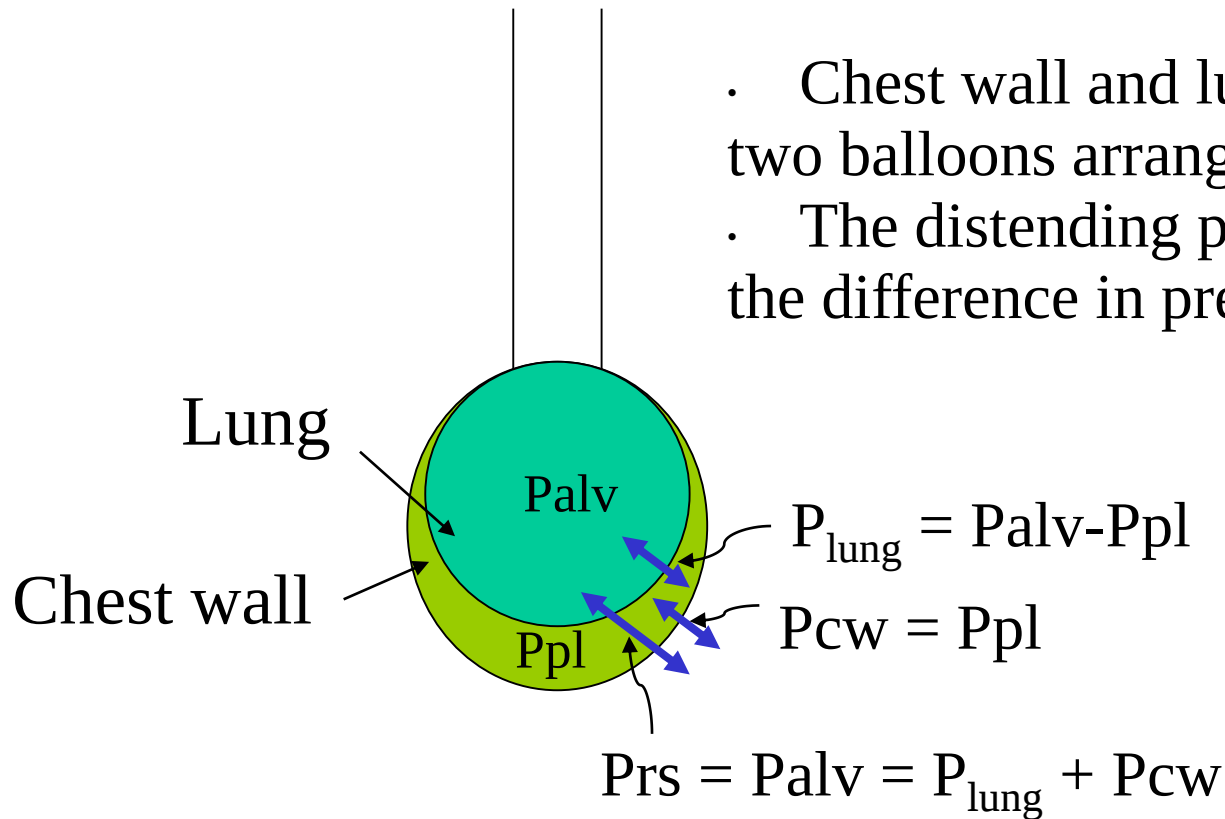
Preload effect
(during inspiration)

↕

↕

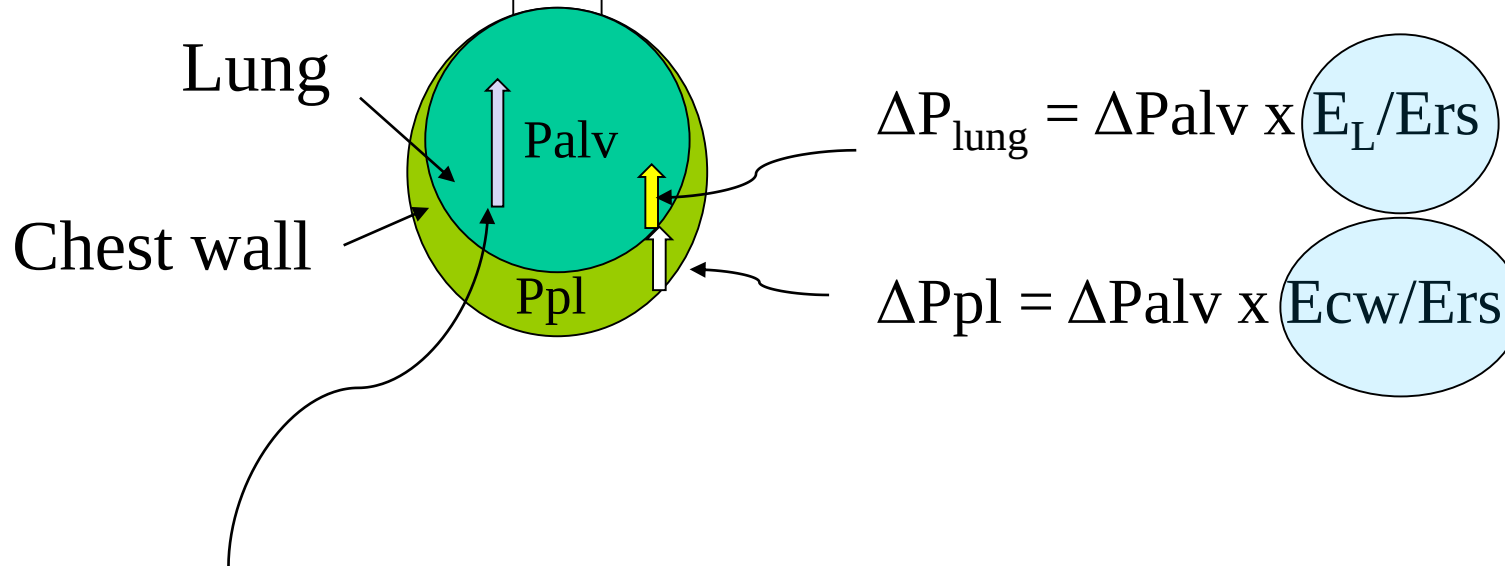
Afterload effect
(during inspiration)

- Chest wall and lung may be considered as two balloons arranged in series
- The distending pressure of each balloon is the difference in pressure across its wall

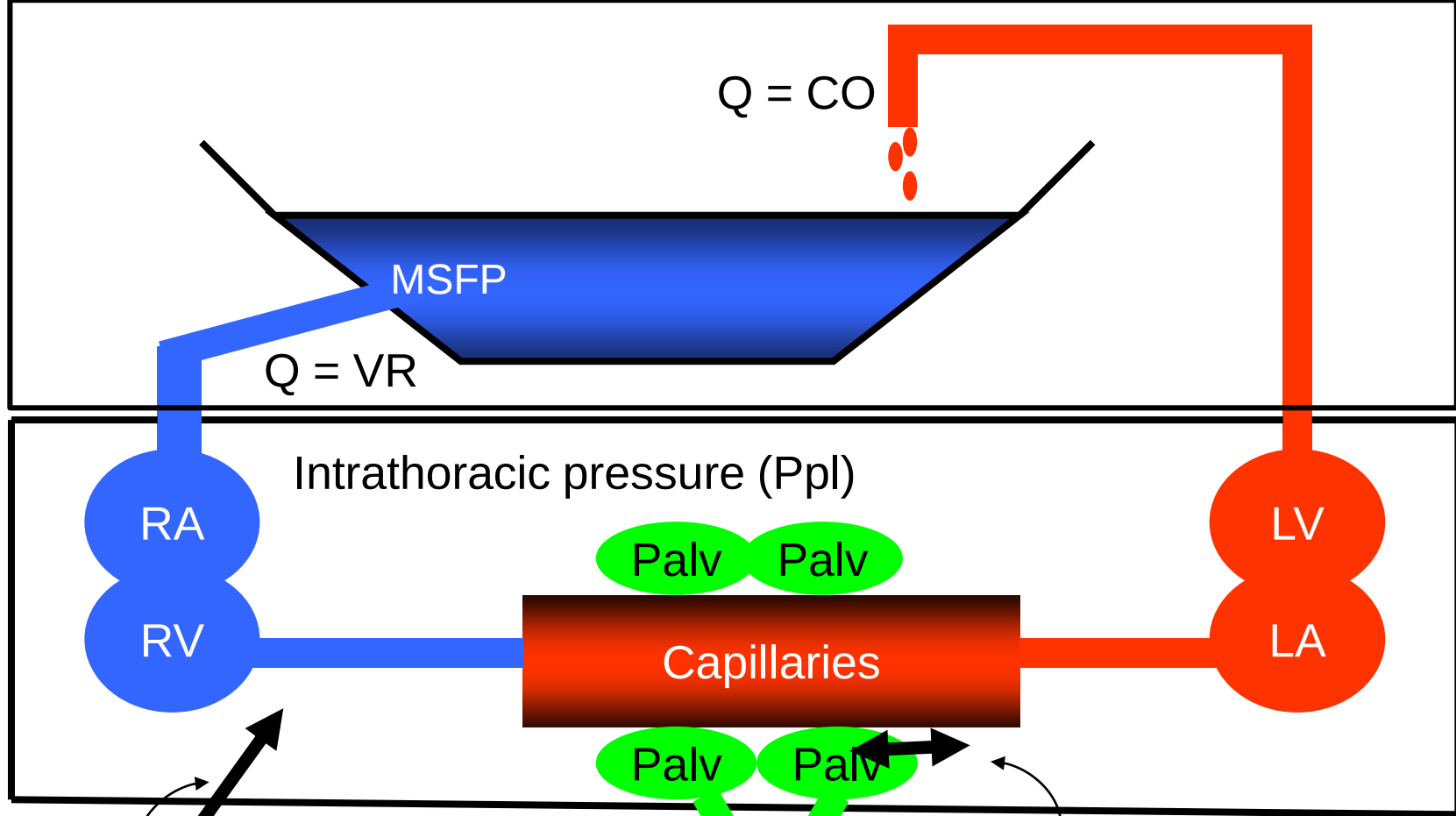




- Chest wall and lung may be considered as two balloons arranged in series
- The distending pressure of each balloon is the difference in pressure across its wall



ΔP_{alv} is dissipated to inflate the lungs and chest wall
 $\Delta P_{alv} = \Delta P_{lung} + \Delta P_{pl}$



$\Delta P_{pl} = \Delta P_{alv} \times E_{cw}/E_{rs}$
 (preload effect)
For a given ΔP_{alv} the change in P_{pl} increases with increasing E_{cw}



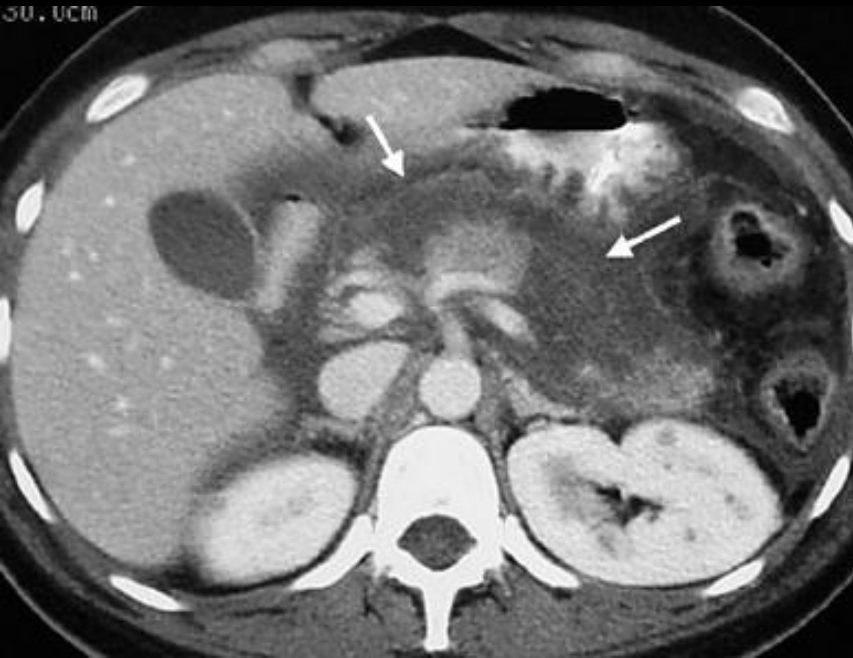
$\Delta P_{lung} = \Delta P_{alv} \times E_L/E_{rs}$
 (afterload effect)
For a given ΔP_{alv} the change in P_{lung} increases with increasing E_L

V_T 6 ml/kg (0.42 ml), PEEP 15 cmH₂O



End inspiratory plateau pressure 30 cmH₂O

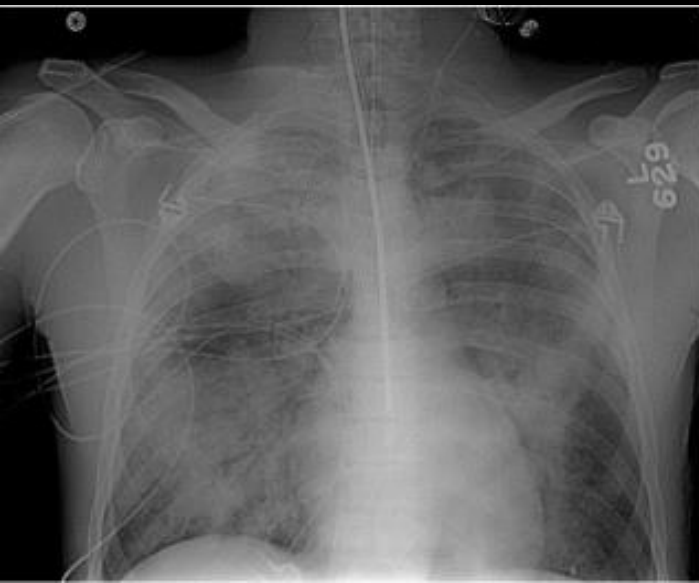
$$E_{rs} = (30 - 15) / 0.42 = 36 \text{ cmH}_2\text{O/L}$$



Patient A:
acute pancreatitis
 $E_{cw} = 21 \text{ cmH}_2\text{O/L}$

$$\Delta P_{\text{lung}} = 6 \text{ cmH}_2\text{O}$$
$$\Delta P_{\text{pl}} = 9 \text{ cmH}_2\text{O}$$

**Predominant
preload effect**



Patient B:
pneumonia
 $E_{cw} = 6 \text{ cmH}_2\text{O/L}$

$$\Delta P_{\text{lung}} = 13 \text{ cmH}_2\text{O}$$
$$\Delta P_{\text{pl}} = 3 \text{ cmH}_2\text{O}$$

**Predominant
afterload effect**



ΔP_{es}



Pre-load effect

Prone position

Jozwiak et al. AJRCCM 2013

Vieillard-Baron et al. Chest. 2007

ΔP_{lung}



After-load effect

Recruitment

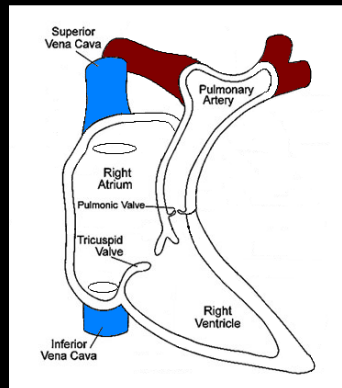
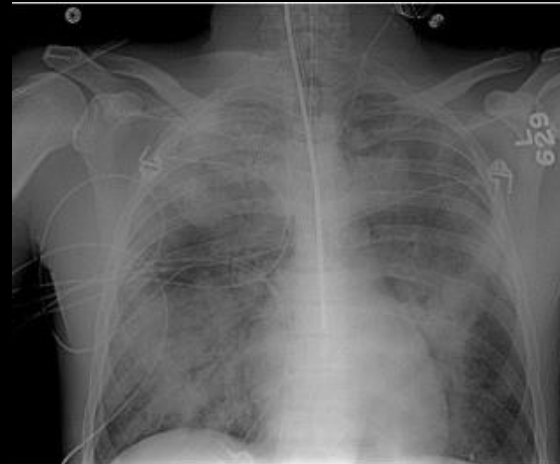
ECCO₂ removal

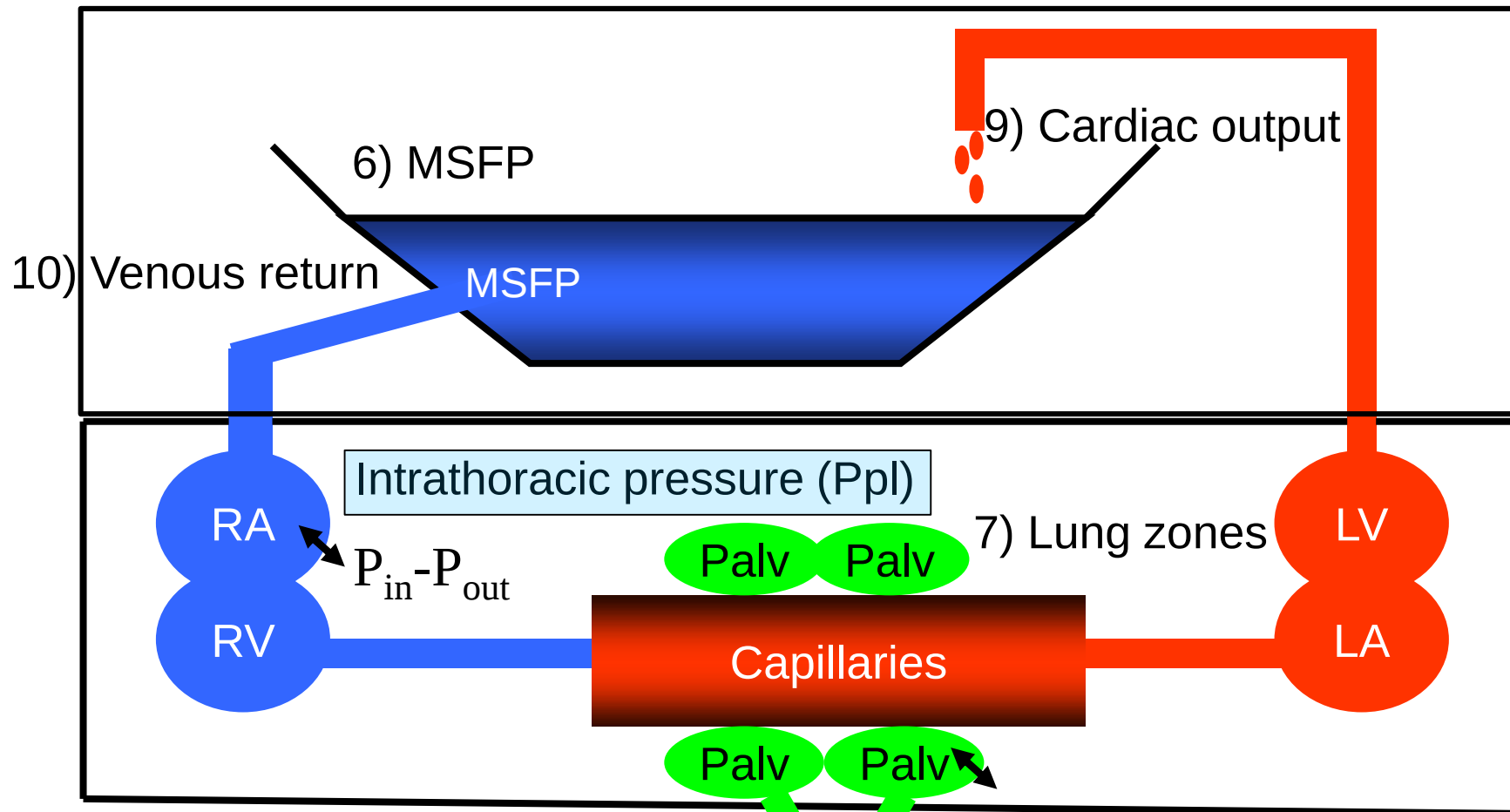
Lower driving P

Lower V_T

Vieillard-Baron et al.

Intensive Care Med 2013





Mechanical ventilation changes everything

- 1) Autonomic tone
- 2) Palv
- 3) Ppl

Passive

4) Trans-vascular pressure

5) Trans-pulmonary pressure

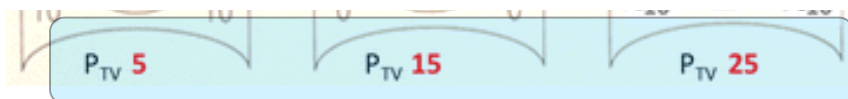
8) Lung compliance

11) Juxtacardiac pressures



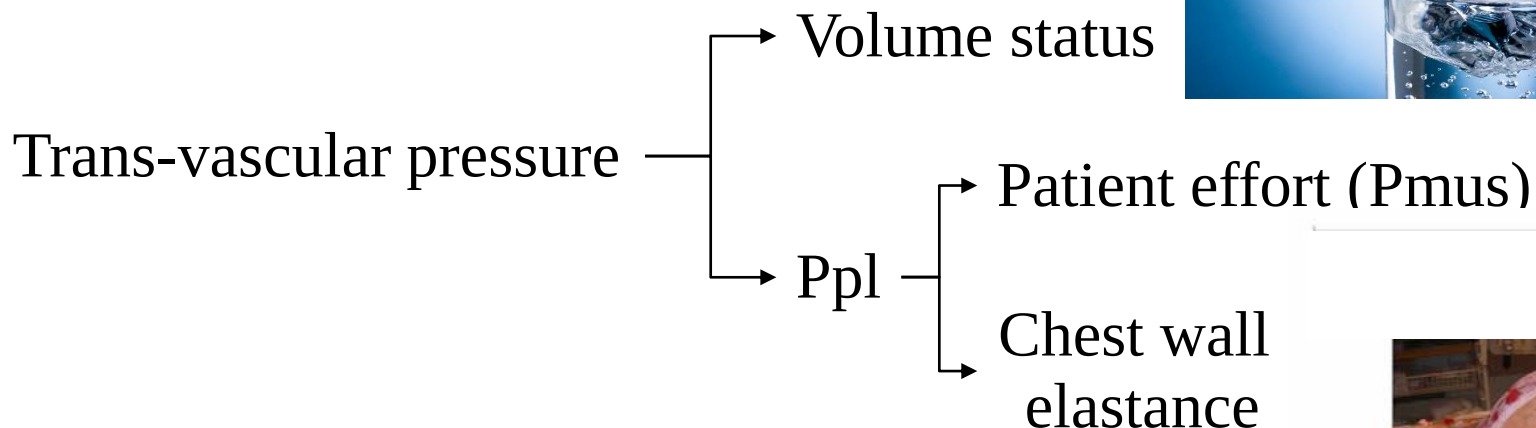
The Application of Esophageal Pressure Measurement in Patients with Respiratory Failure

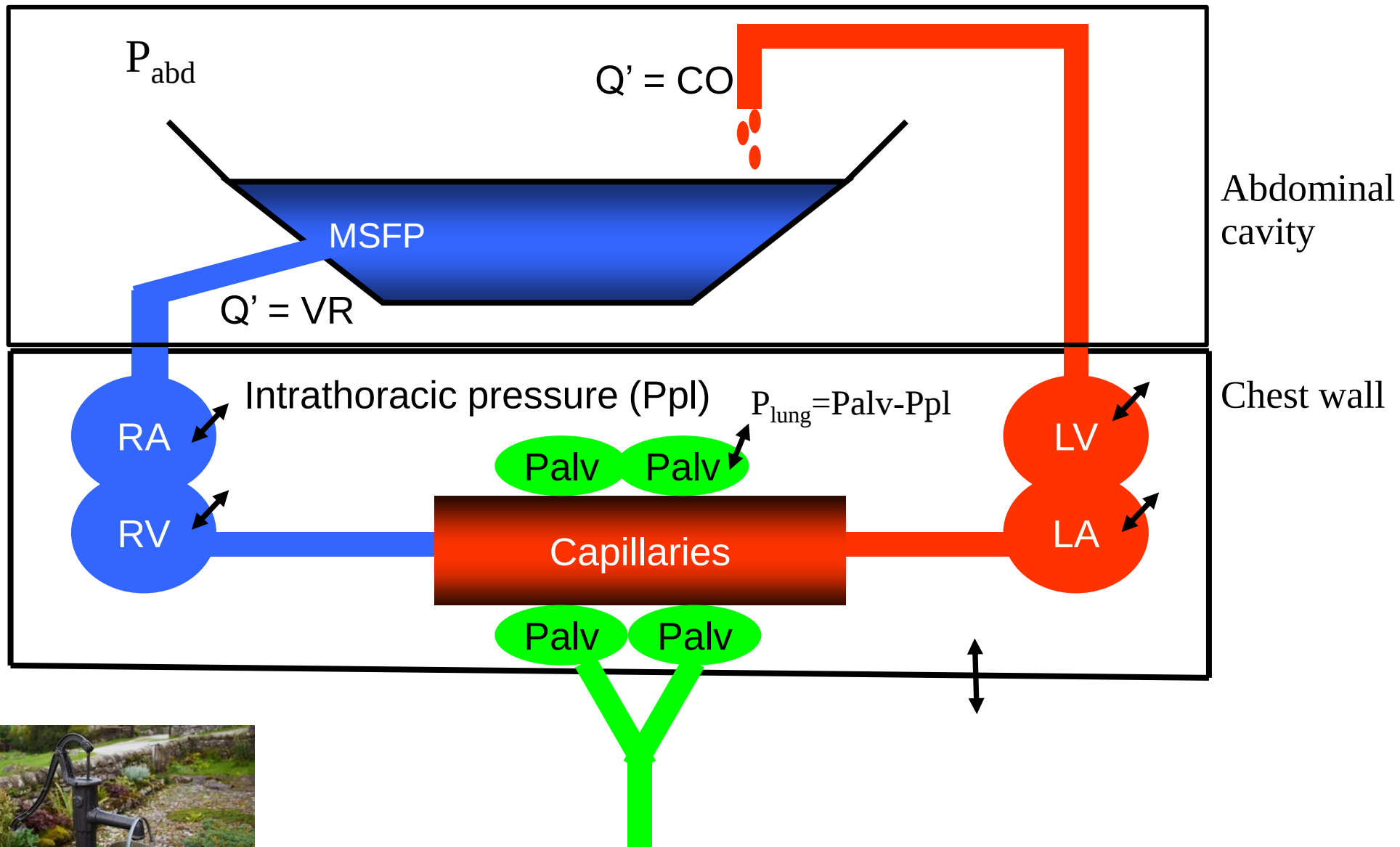
Evangelia Akoumianaki¹, Salvatore M. Maggiore², Franco Valenza³, Giacomo Bellani⁴, Amal Jubran⁵, Stephen H. Loring⁶, Paolo Pelosi⁷, Daniel Talmor⁶, Salvatore Grasso⁸, Davide Chiumello⁹, Claude Guérin¹⁰, Nicolo Patroniti⁴, V. Marco Ranieri¹¹, Luciano Gattinoni¹², Stefano Nava¹³, Pietro-Paolo Terragni¹¹, Antonio Pesenti⁴, Martin Tobin⁵, Jordi Mancebo¹⁴, and Laurent Brochard¹⁵



Veillard-Baron et al. Intensive Care Med 2016; 42:739

For a given CVP





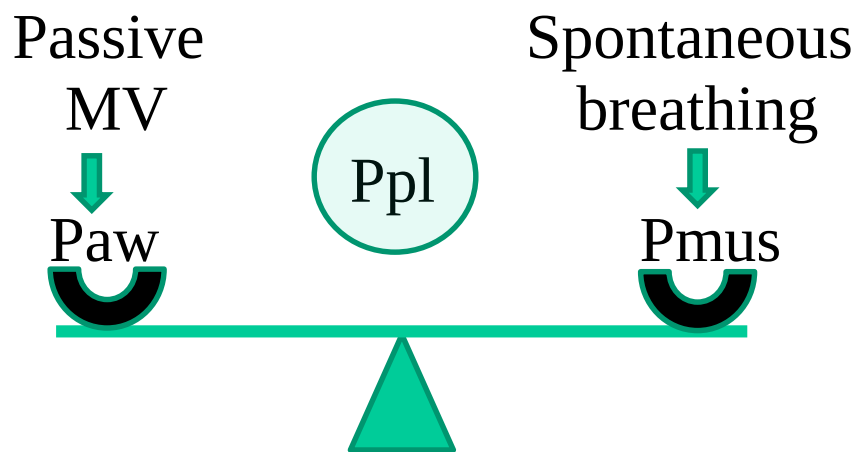
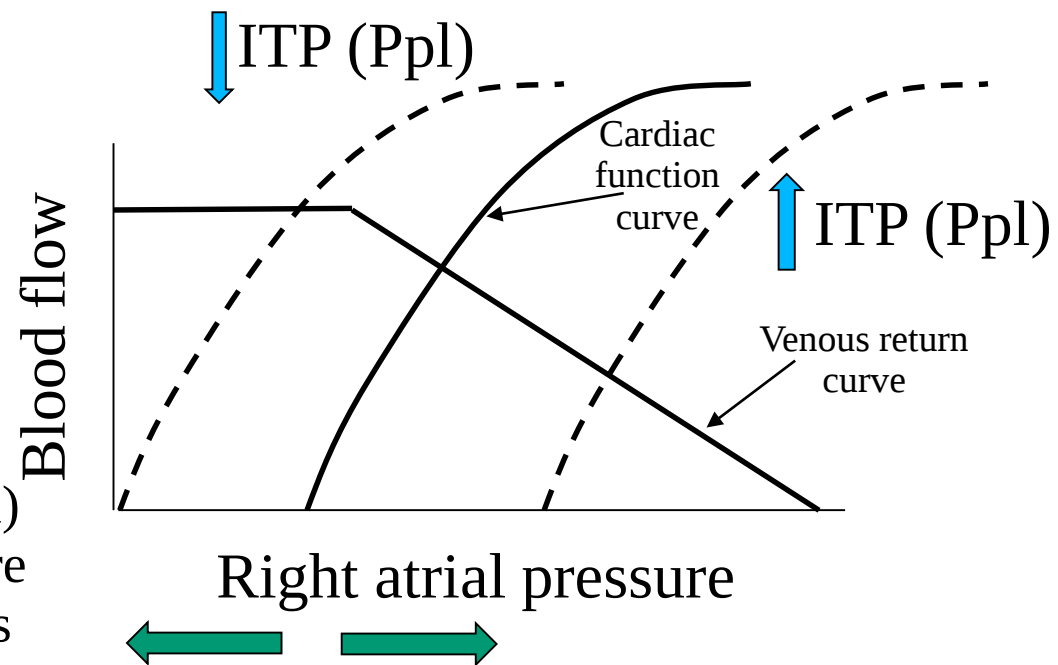
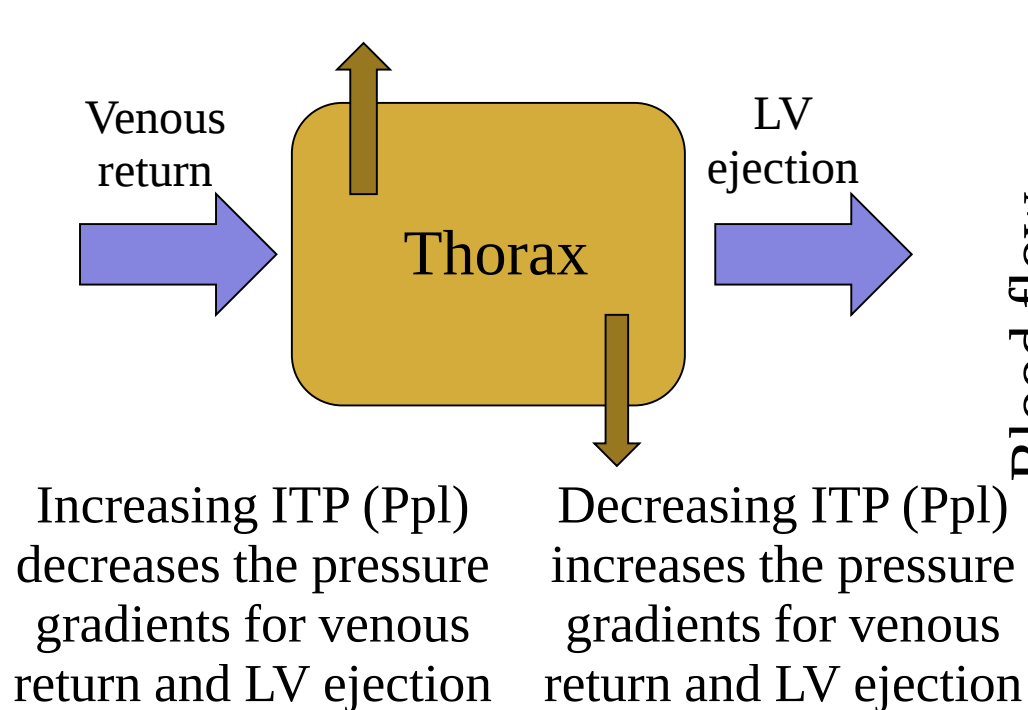
Venous return (VR) = Cardiac output (CO)

Heart-lung
is

Black

interaction
not

White



Assisted modes of MV

Adapted from:
 Gomez and Pinsky. *Principles and Practice of Mechanical Ventilation*
 (ed. Tobin) 2013; pp.821-849