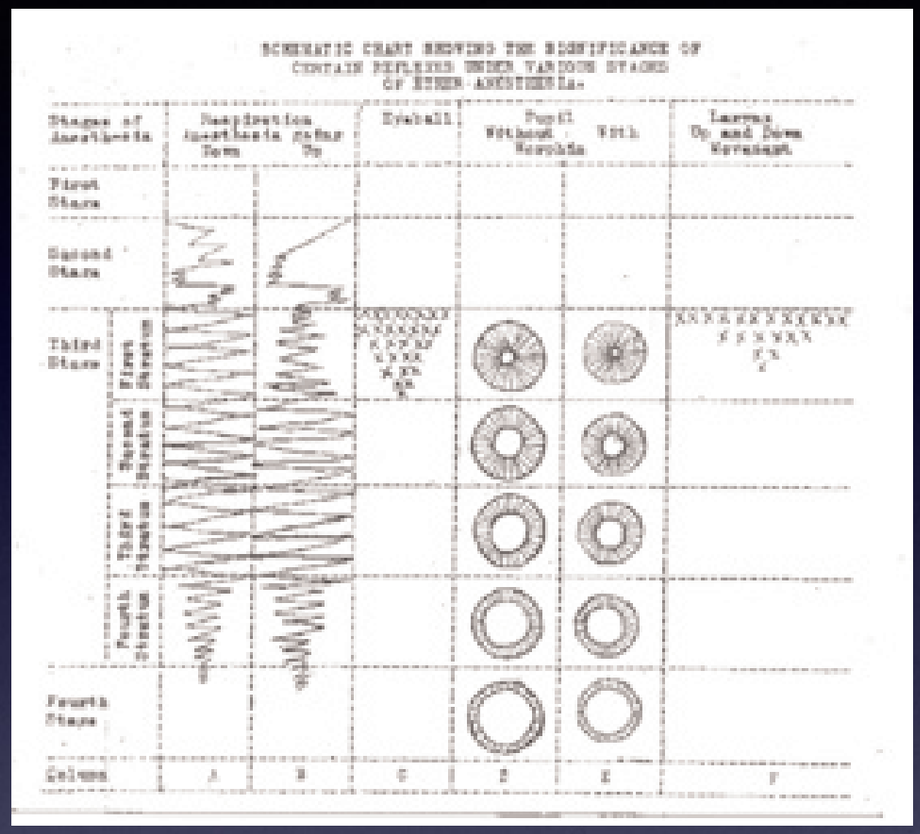
Why bother learning the basics?

The first protocol?



Oxygen Delivery -

Understanding the Physiology

Optimising Haemodynamics with Fluid

Dr J Vogel FRCA

Clinical Case

77 yr old lady

C. Difficile toxic mega colon

Peripherally very oedematous

Received 6 L fluid

Blood Pressure = 95/55

Heart Rate 110

Respiratory Rate = 35

Urine output = 15 ml/hr

Oxygen delivery ~

Cardiac output x Hb x % Sat O2

Cardiac output x Hb x % Sat O2

Preload

Effective blood volume
Capacitance
Obstruction
Septal shift
IV fluid
    volume
    C.O.P.

Pump failure

Arrhythmias
Ischaemia
Valvular problems
Septal shift

Afterload

RAA adaptaion
Sepsis
Valvular problems
Pulmonary embolism
Hypertension
Shunts

Heart rate

Anaemia

Fe def
Dilutional
Inflammatory
Vitamin deficiency
Aplastic

Abnormal Hb

Sickle cell
Thalassaemia
met Hb
CO Hb

Hemolysis

free Hb and NO
Pulmonary hypertension
Hypercoagulability

Hyperviscosity

PRV
Acclimatisation

Inspired O2

Altitude
Hyperbaric O2

Hypoventilation

Decreased respiratory drive
    drug induced
CVA
Fatigue (asthma)
Obstruction
    Sleep apnoea syndrome
    Decreased consciousness

Ventilation/perfusion abnormalities

Shunt
    Pneumonia
    Pulmonary oedema
Dead space
    Pulmonary embolism
    Fat embolism
Mixed
    COPD
    Asthma

Summary of this lecture

- Importance of cardiac output
- Physiology of cardiac output and venous return
- Consequences of "guessing" wrong
  - Too much fluid
  - Too little fluid

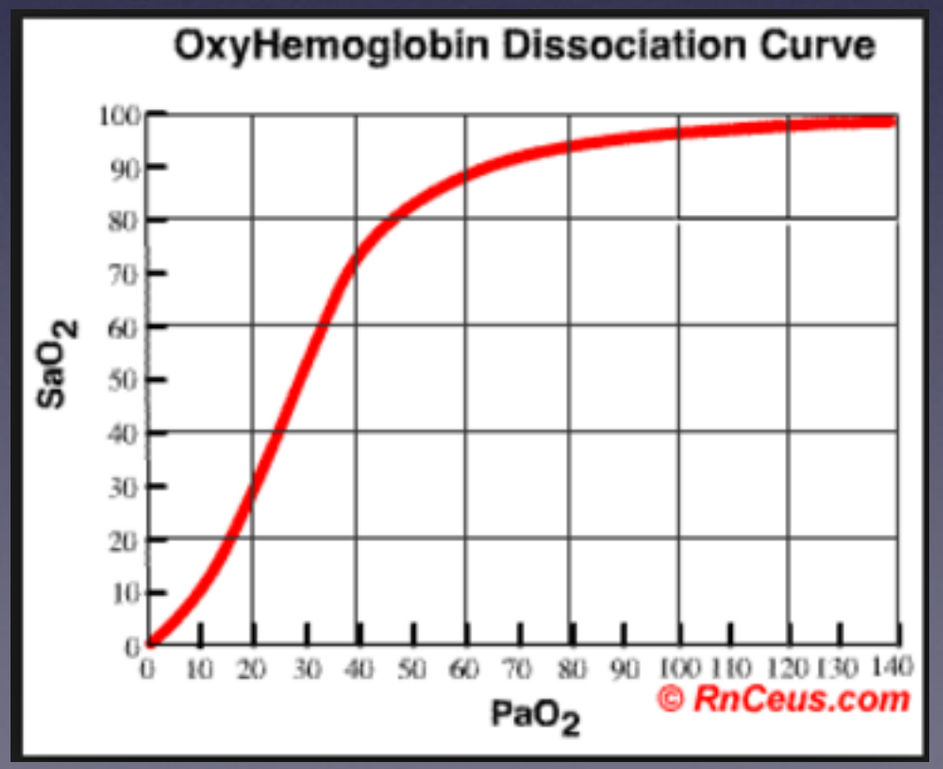
Summary of this lecture

- Importance of cardiac output
- Physiology of cardiac preload and venous return
- Consequences of "guessing" wrong
  - Too much fluid
  - Too little fluid

Why is cardiac output so important ?

Cardiac output x Hb x % Sat O2

Cardiac output the only parameter that:



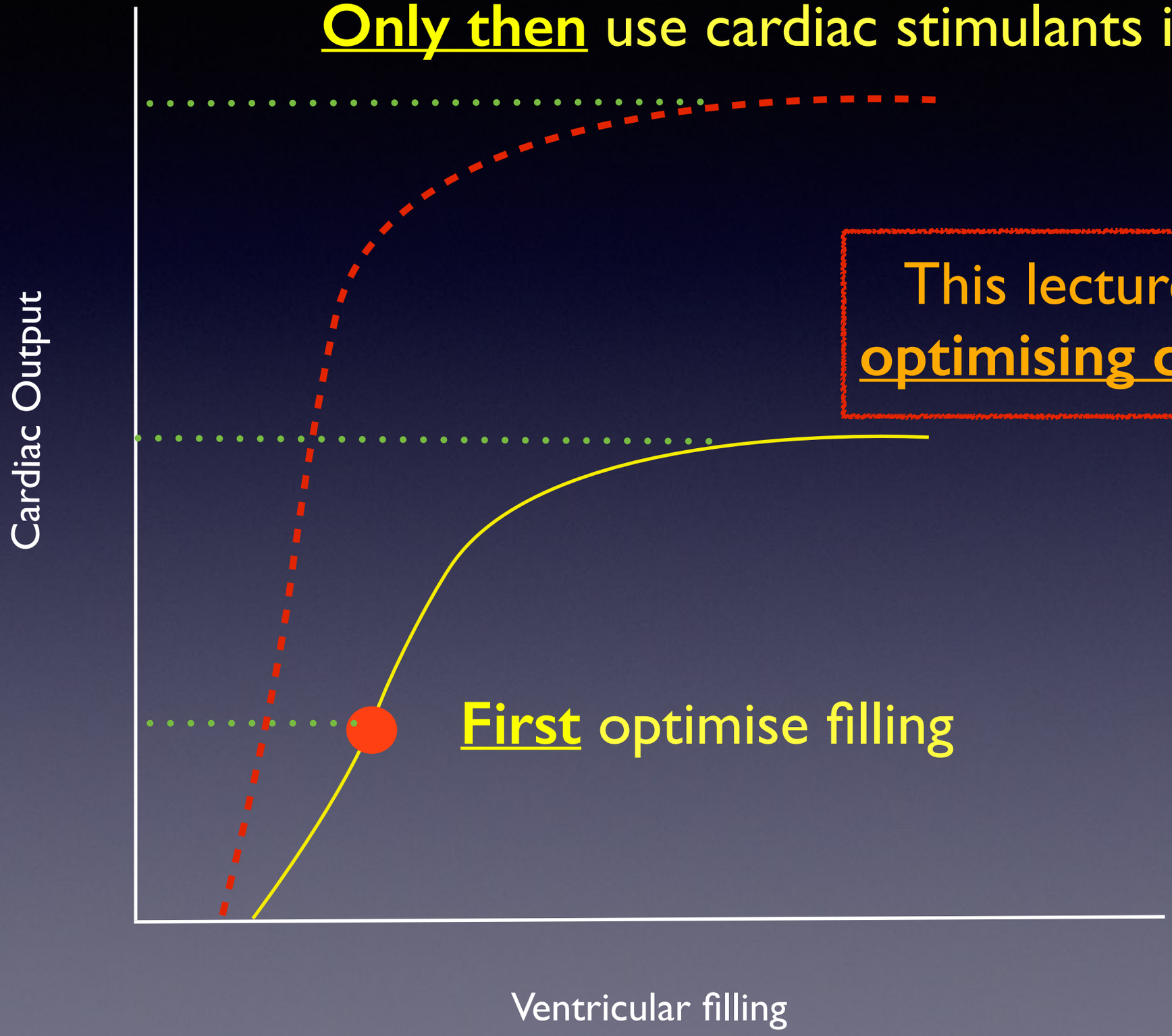
Consider:

- We can easily measure Hb and O2 Sat.
- The most important factors, **cardiac output** is estimated clinically.
- Imagine if we had to look for cyanosis or pale conjunctiva.

Achieving effective cardiac output

Only then use cardiac stimulants if needed

This lecture will be about **optimising cardiac stretch** !



Clinical Case

77 yr old lady

C. Difficile toxic mega colon

Peripherally very oedematous

Received 6 L fluid

Blood Pressure = 95/55

Heart Rate 110

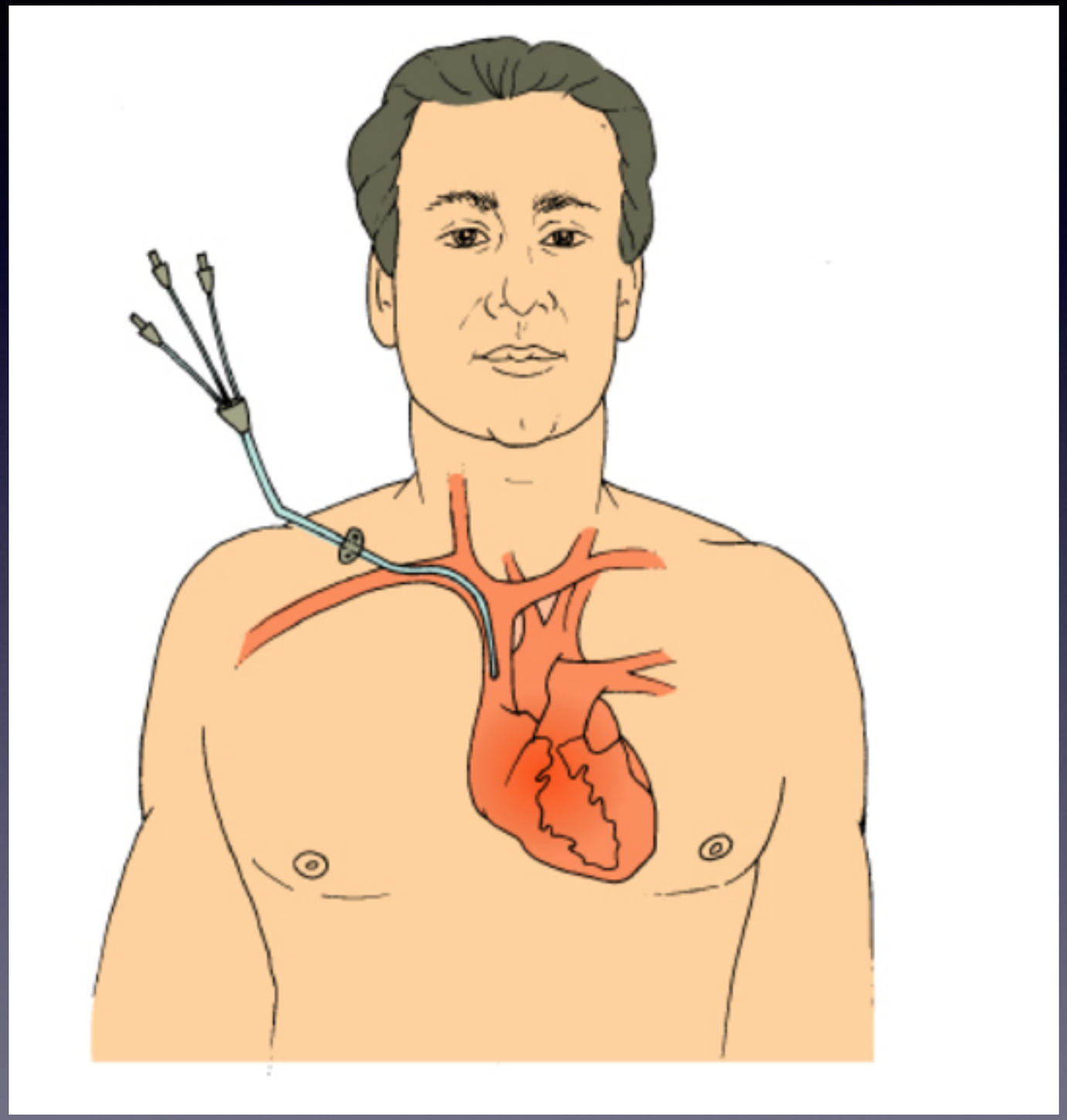
Respiratory Rate = 35

Urine output = 15 ml/hr

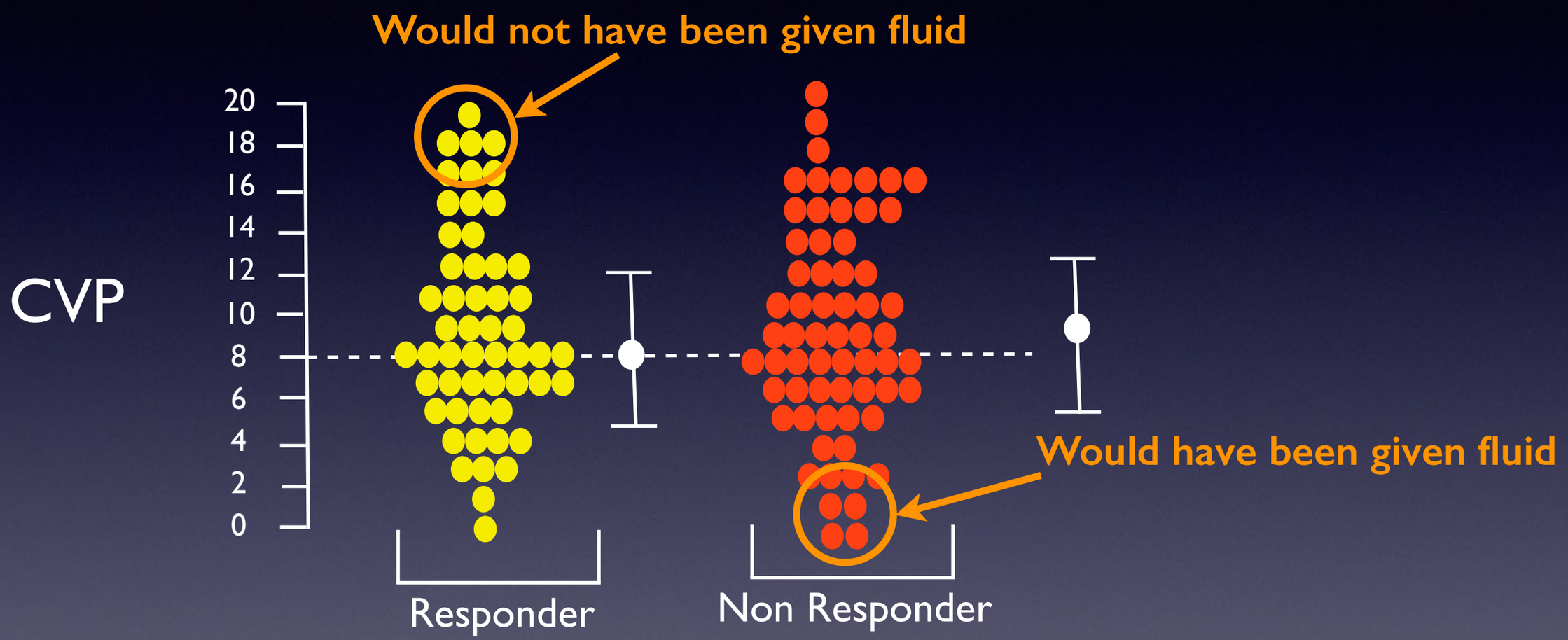
So do you give more fluid or not?

How do you know?

How about measuring the central venous pressure ?

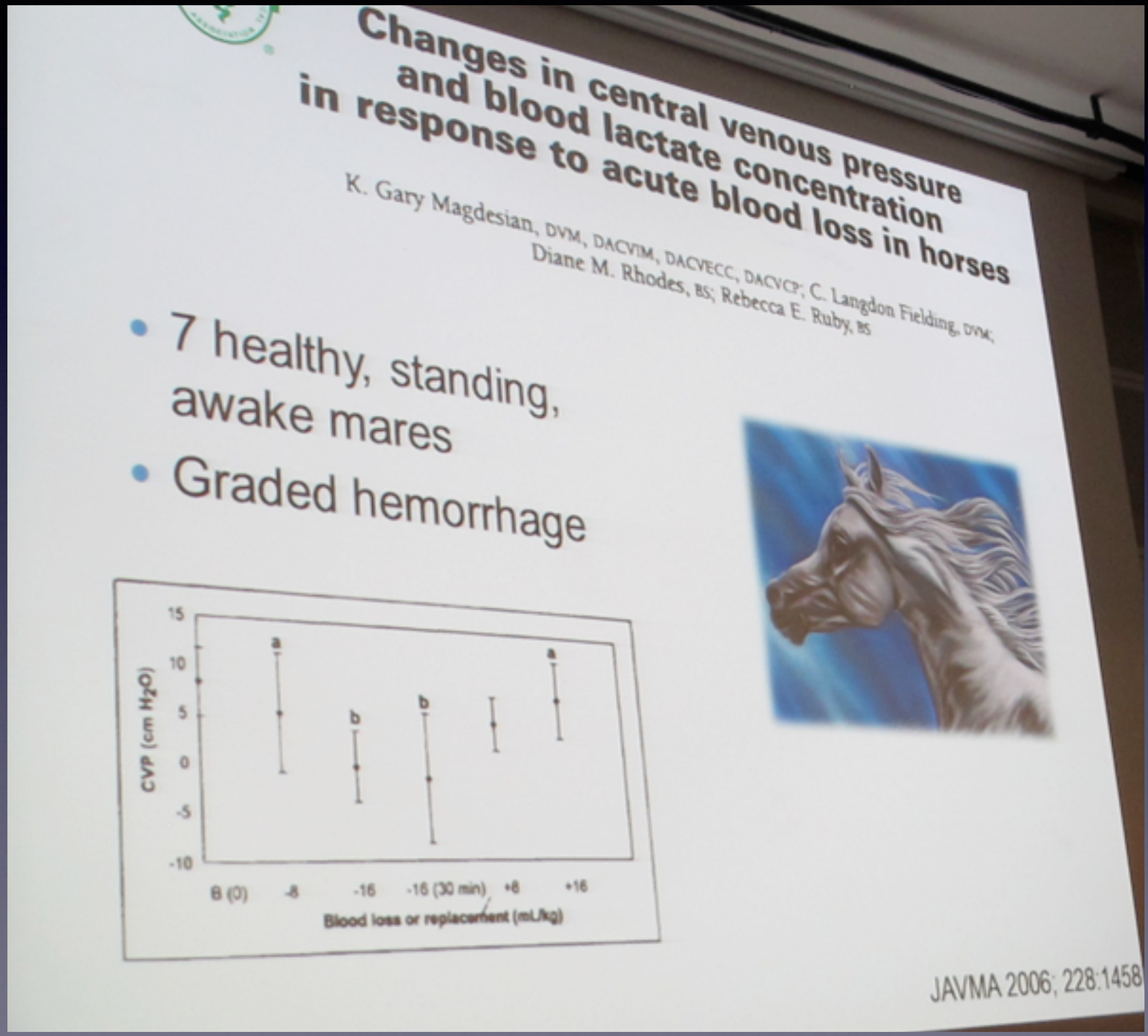


How about the central venous pressure ?



Crit Care Med 2007 Vol. 35, No.

In fact....the only "hard" evidence



Don't you just love guidelines?

Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock: 2012

MANAGEMENT OF SEVERE SEPSIS

Initial Resuscitation and Infection Issues (Table 5)

A. Initial Resuscitation

a) CVP 8–12 mm Hg
b) MAP ≥ 65 mm Hg
c) Urine output ≥ 0.5 mL·kg·hr
d) Superior vena cava oxygenation saturation ($ScvO_2$) or mixed venous oxygen saturation (SvO_2) 70% or 65%, respectively.

British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients

GIFTASUP

Alternatively, the clinical response may be monitored by measurement/estimation of the pulse, capillary refill, CVP and blood pressure before and 15 minutes after receiving the infusion. This procedure should be repeated until there is no further increase in stroke volume and improvement in the clinical parameters.

RCS
ADVANCING SURGICAL STANDARDS

Care of the Critically Ill Surgical Patient (CCrISP)

"resuscitate with fluids, pushing the CVP up to a maximum of 17 mm Hg"!!!

Summary of this lecture

- Importance of cardiac output
- Physiology of cardiac preload and venous return
- Optimising cardiac output with fluids
  - What works and what doesn't

Understanding the Physiology of Preload

What does a bag of lettuce have to do with Starling's Law?

Illustration of transmural pressure

Illustration of transmural pressure

The NEW ENGLAND JOURNAL of MEDICINE

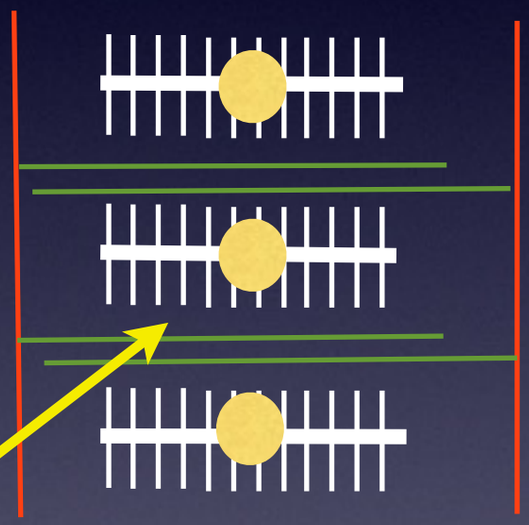
CORRESPONDENCE

Boyle's Law and Breast Implants

N Engl J Med 1994; 331:483-484 | August 18, 1994 | DOI: 10.1056/NEJM199408183310720

Starlings Law of the Heart

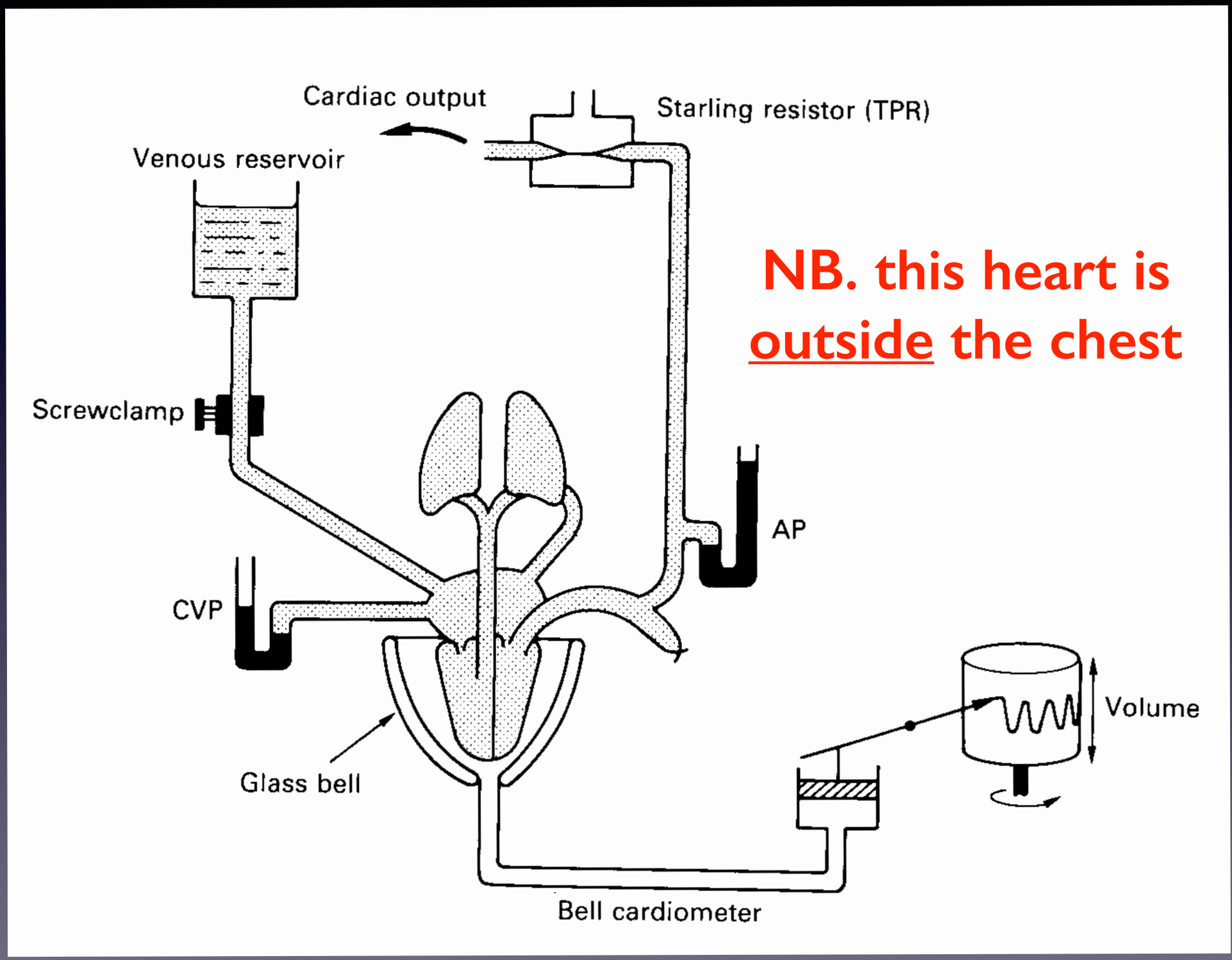
'the greater the stretch of the ventricle in diastole, the greater the stroke work achieved in systole"



Greater overlap of actin-myosin crossbridges

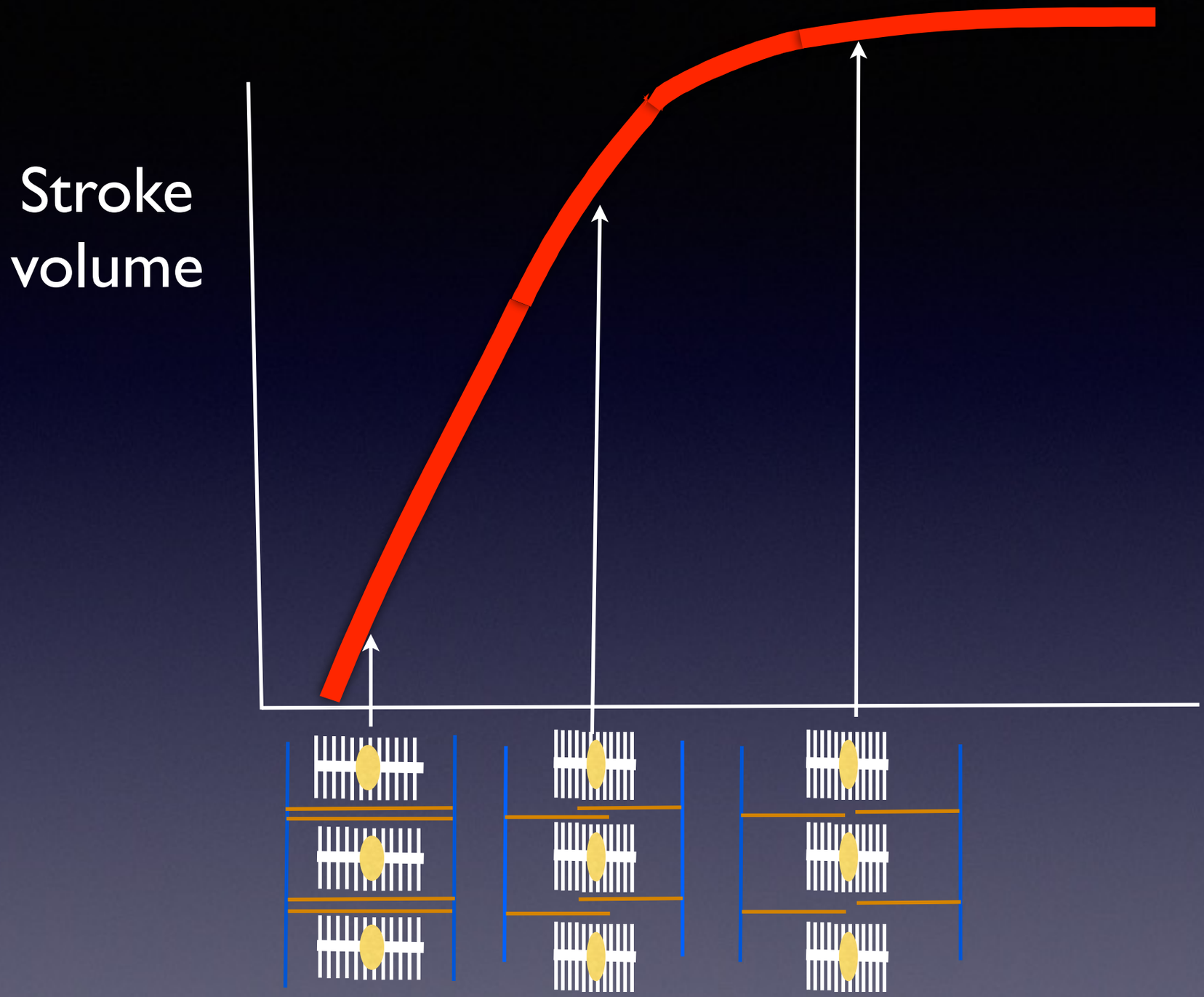
Sarcomere

Starling's experiment



NB. this heart is <u>outside</u> the chest

Journal of Physiology; 1914, 48, 465-511



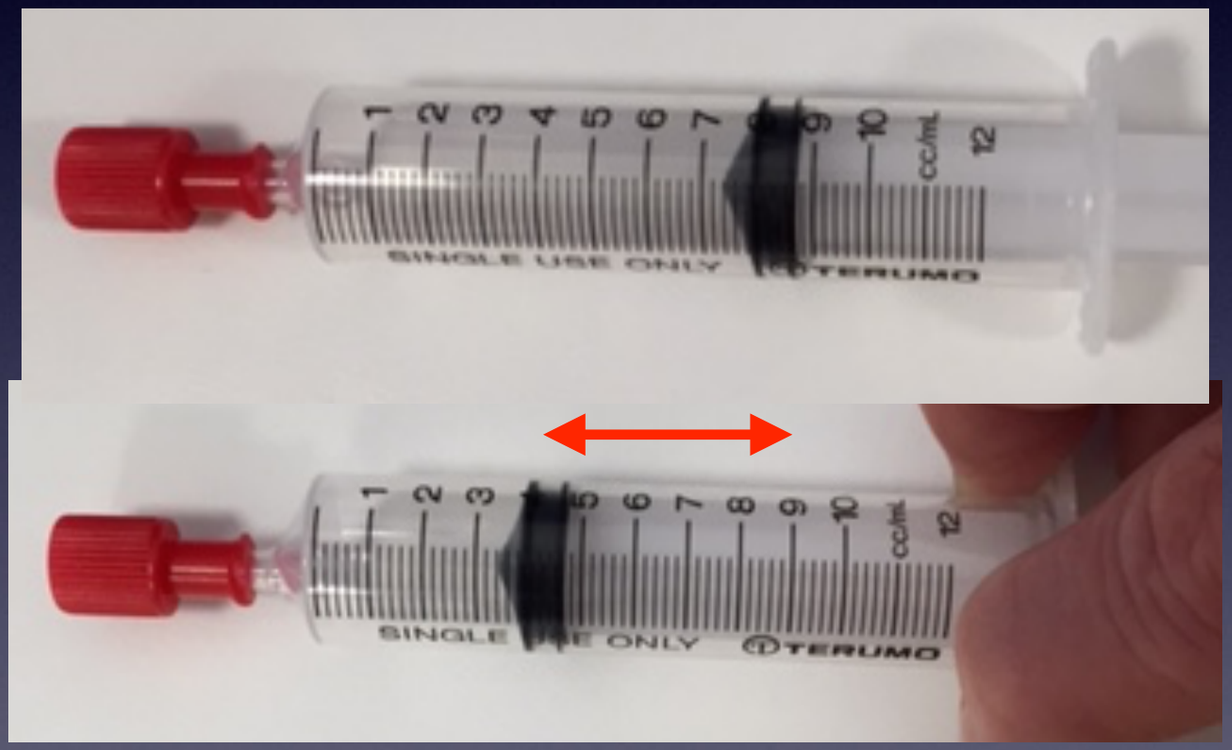
Preload (= muscle stretch)

Intra-thoracic presure

Nature 1969; 221 : 1199-1204

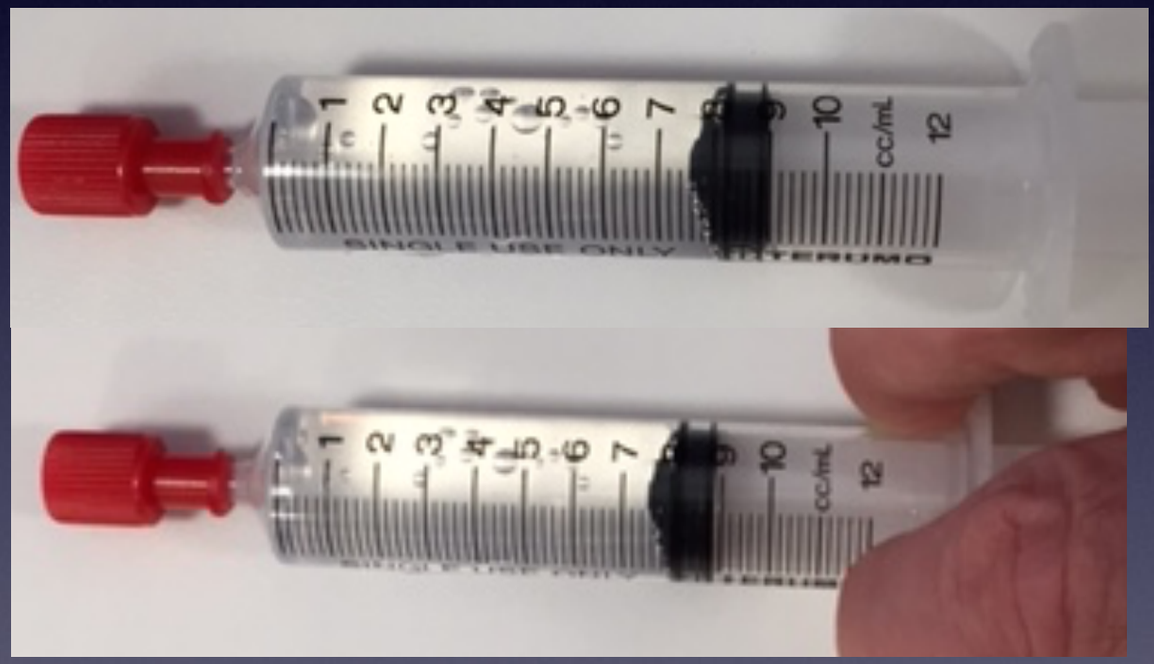
Squeezing the heart

Air



Compressible
Little increase in pressure

Fluid



Non compressible
Large increase in pressure

CVP
+5

0
Inside
thorax

+5

+5

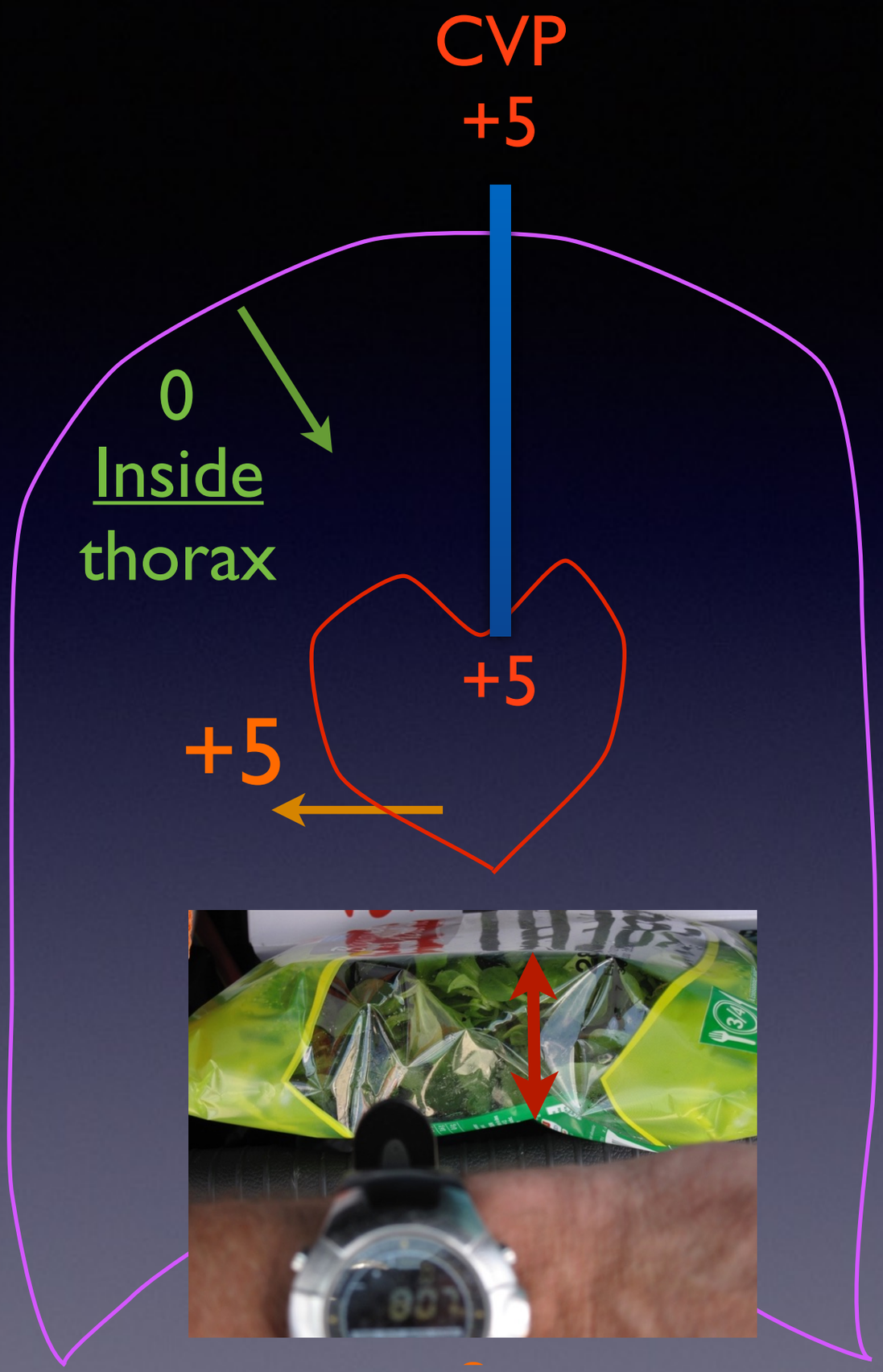
CVP
+155

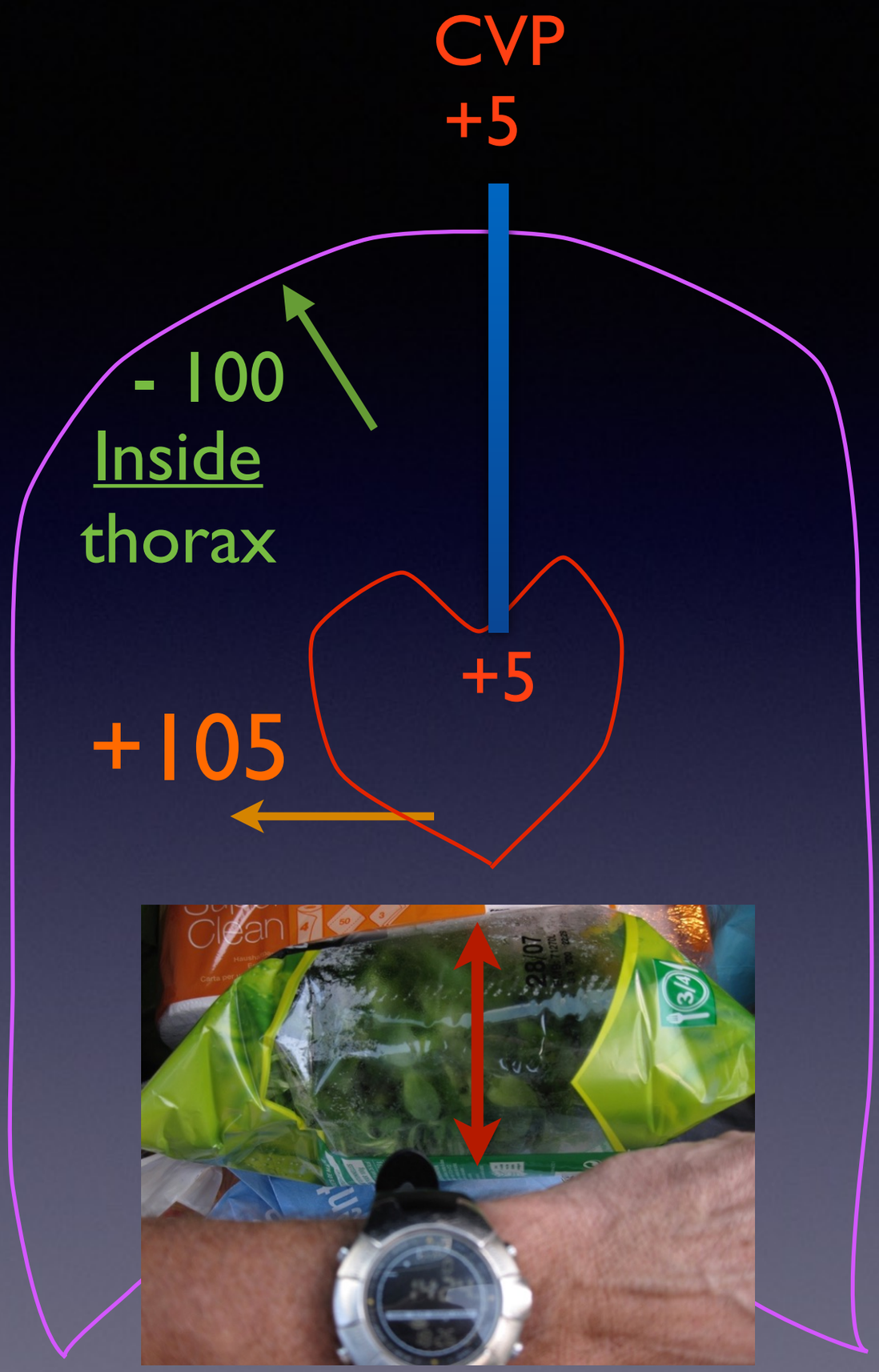
+150
Inside
thorax

+155

+5

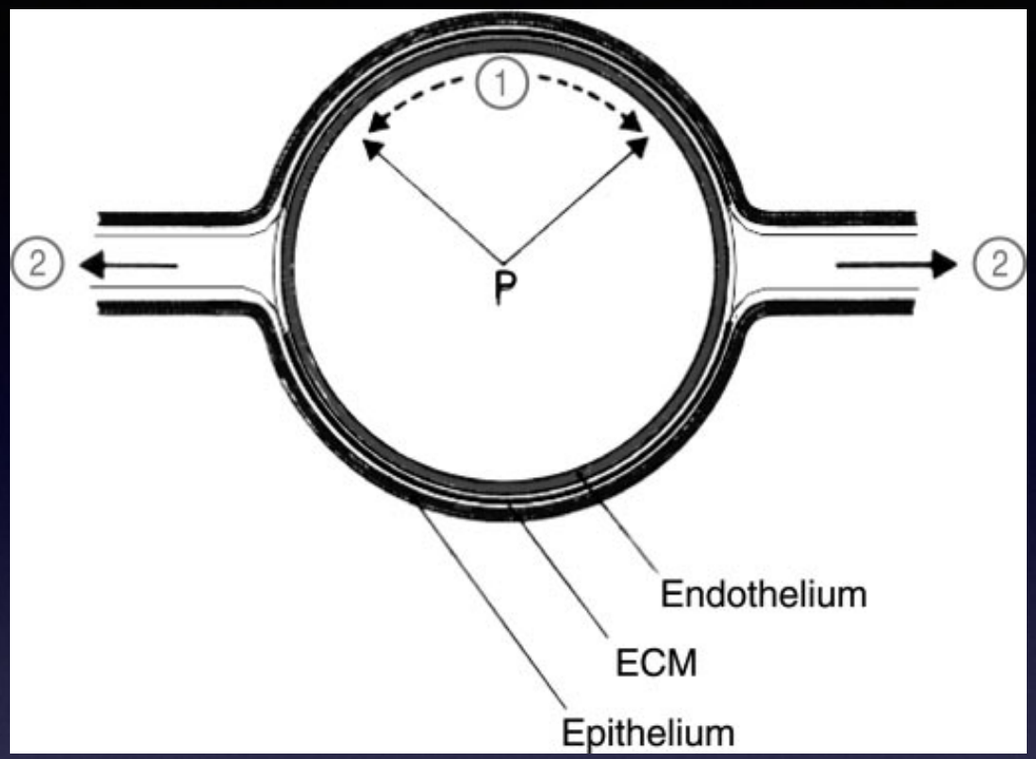
Distending pressure of the heart is the same !

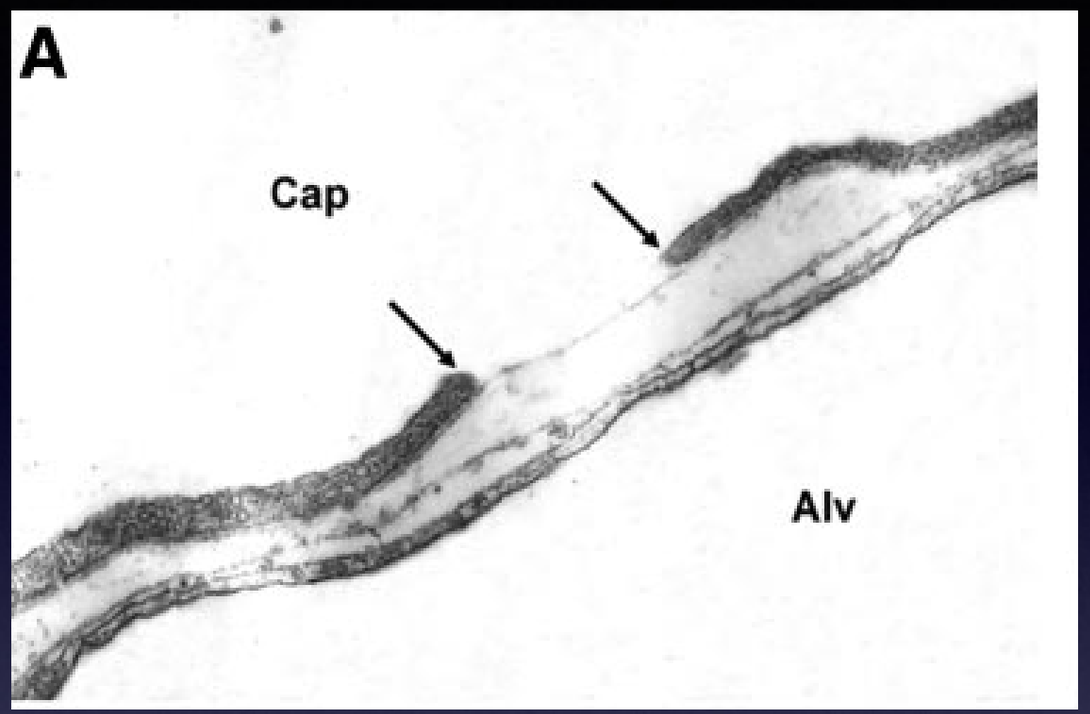


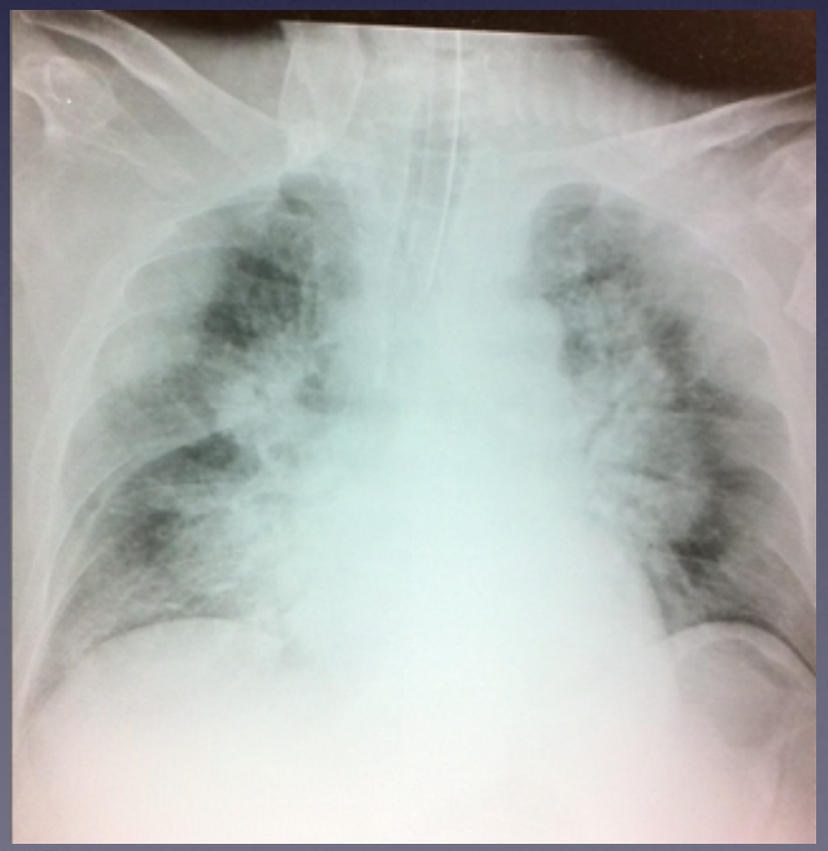


Negative pressure pulmonary oedema

Negative pressure pulmonary oedema







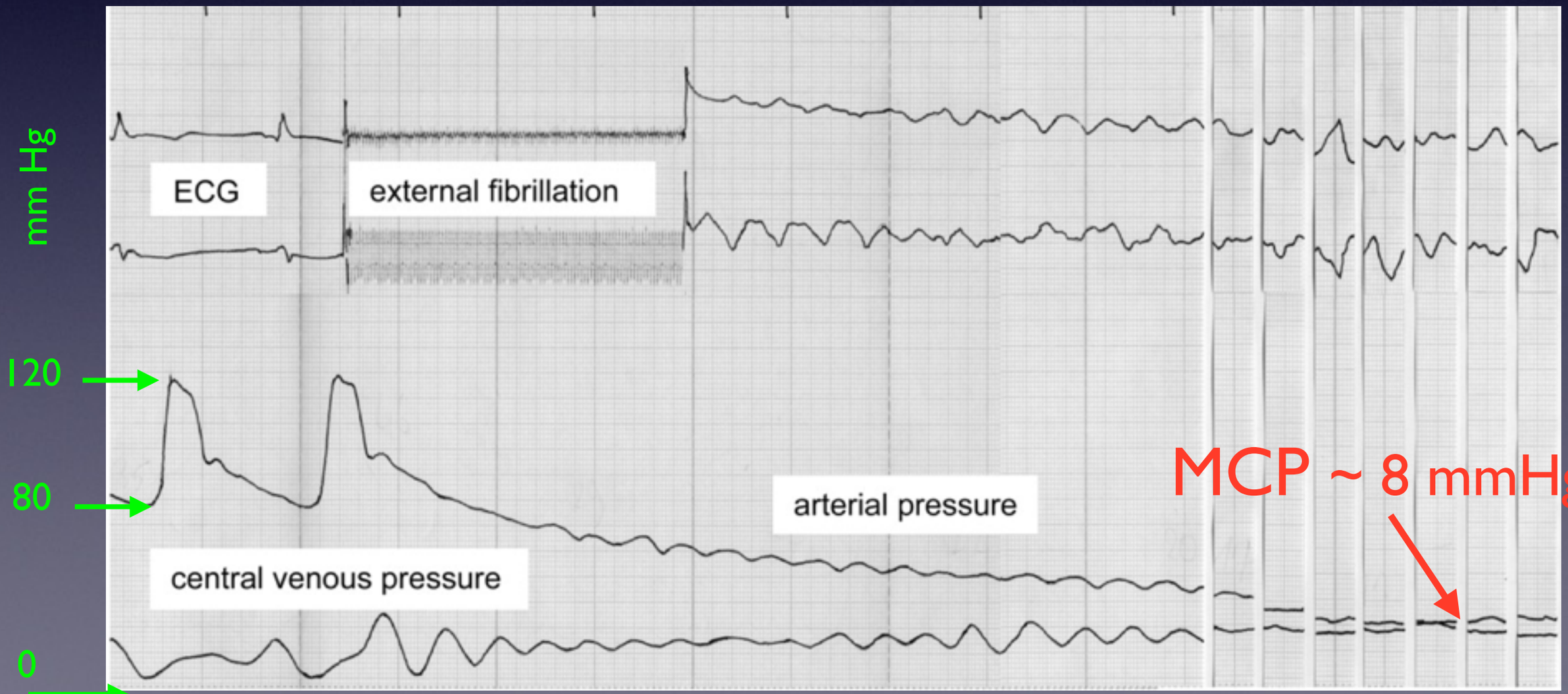
Physiol Rev • VOL 85 • JULY 2005 • www.prv.org

Clinical Case

Fractured femoral shaft
Distended abdomen
Partially resuscitated with fluids
In great pain, so you give morphine
Blood pressure crashes
Why?

Mean circulatory pressure

Mean circulatory pressure ("MCP") = pressure throughout vascular circuit if <u>no flow</u>

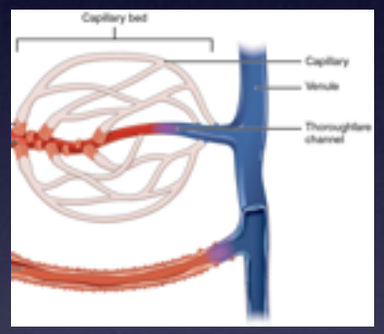


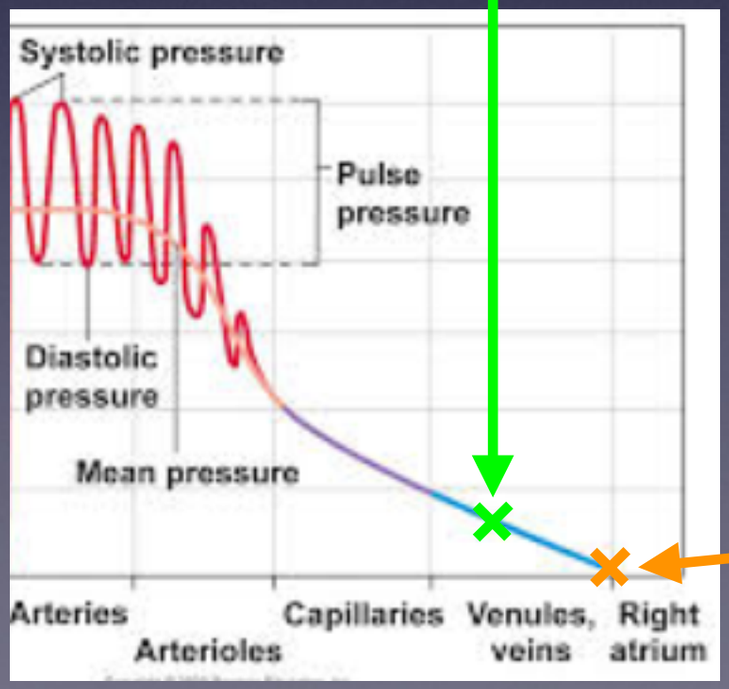
Venous return

Venous return =MCP - CVP

upstream pressure

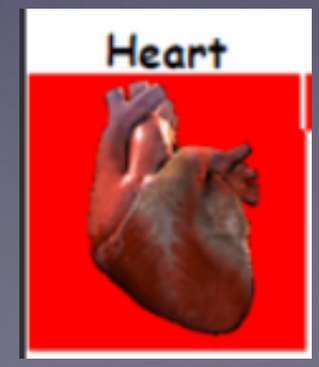
"MCP"





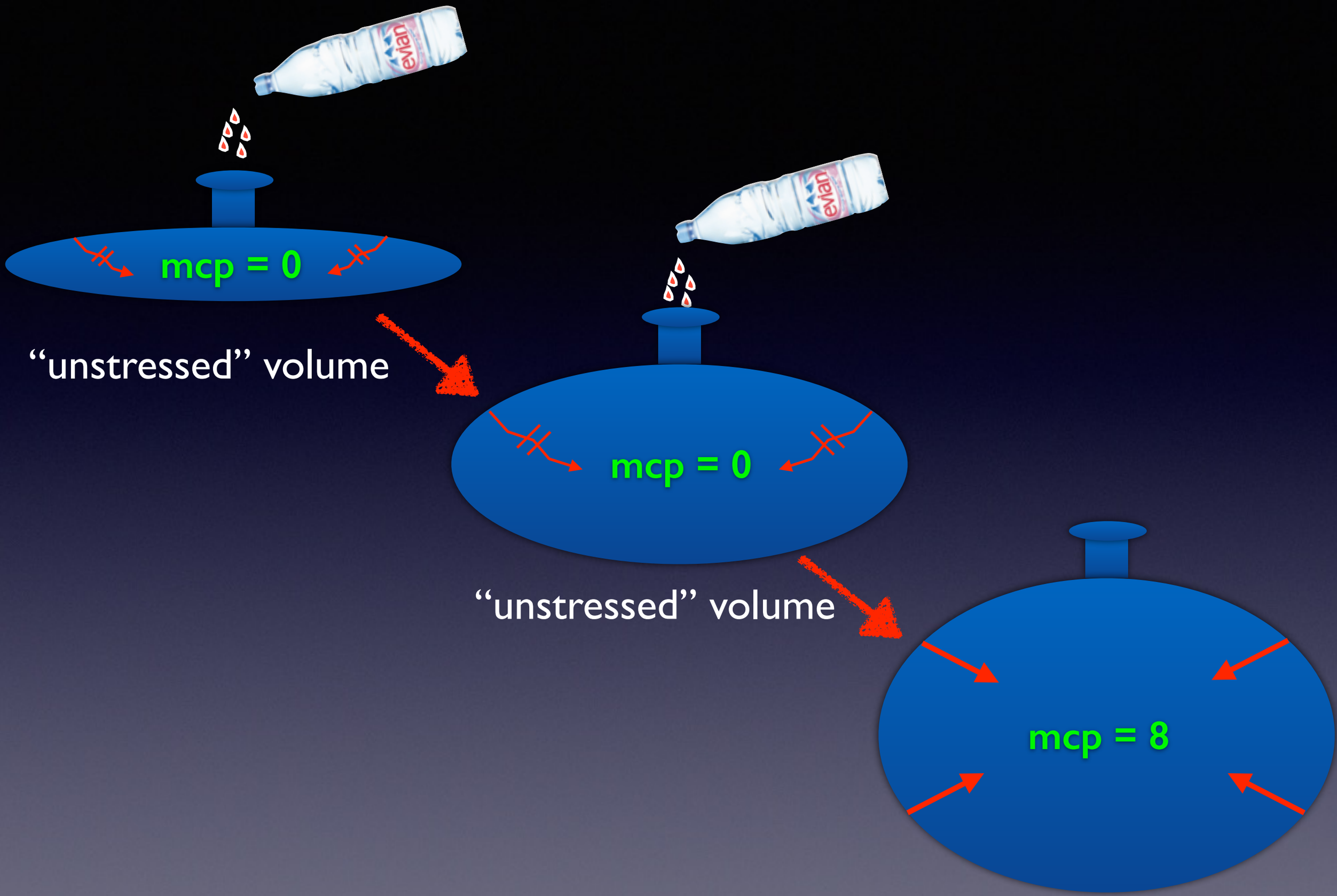
downstream pressure

"CVP"



Stressed venous blood volume =

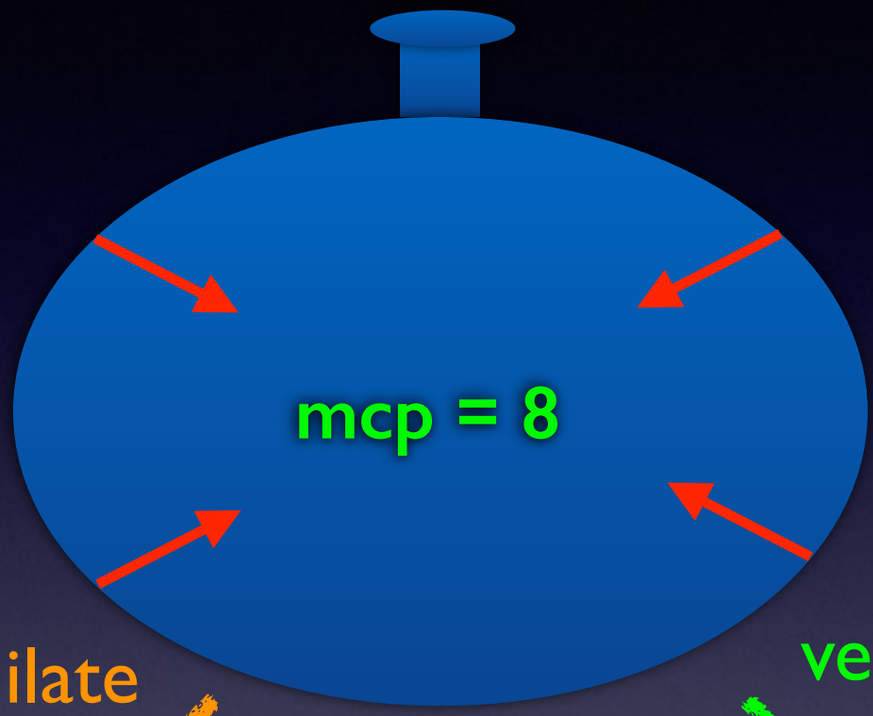
the volume of blood in **excess** of the total volume of the heart and blood vessels at a relaxed, **nondistended** state.


evian
mcp = 0
"unstressed" volume
evian
mcp = 0
"unstressed" volume
mcp = 8
"stressed" volume

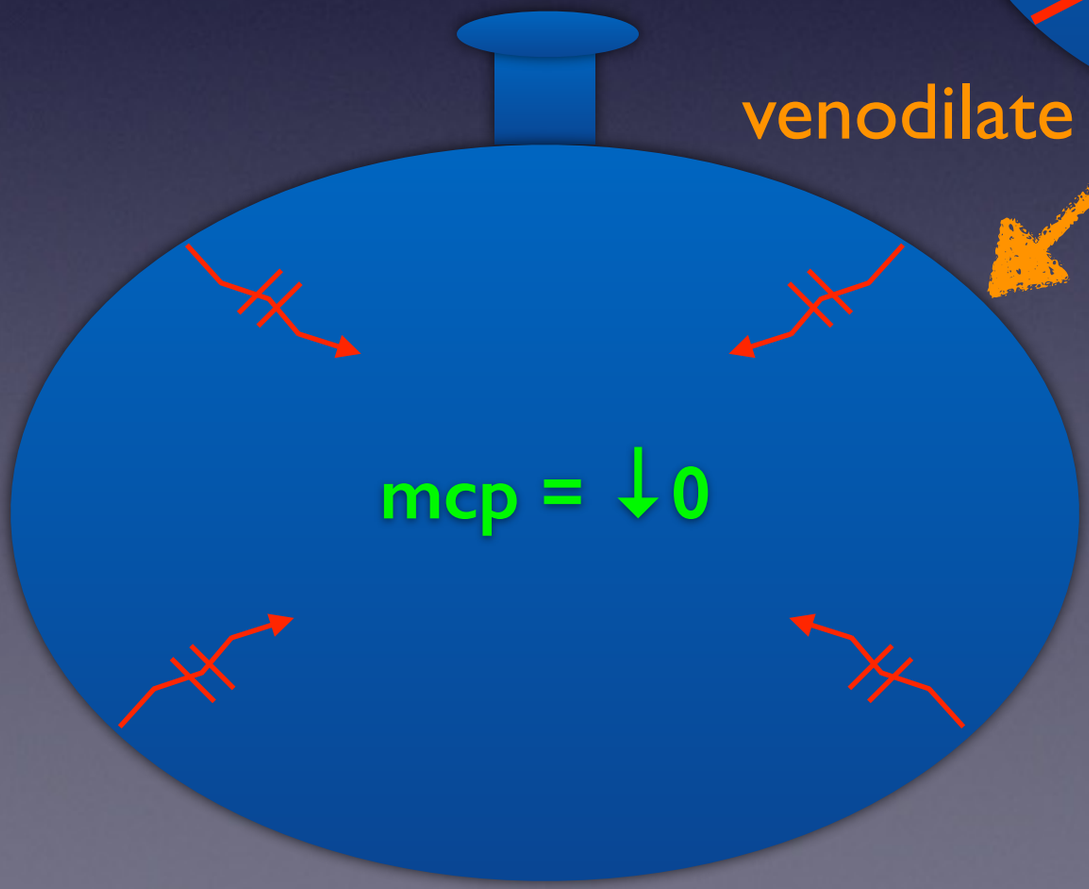
Vasopressors are <u>5 X more potent</u> on the venous (capacity) side then on the arterial (resistance) side

Opioids
GTN
Sedatives

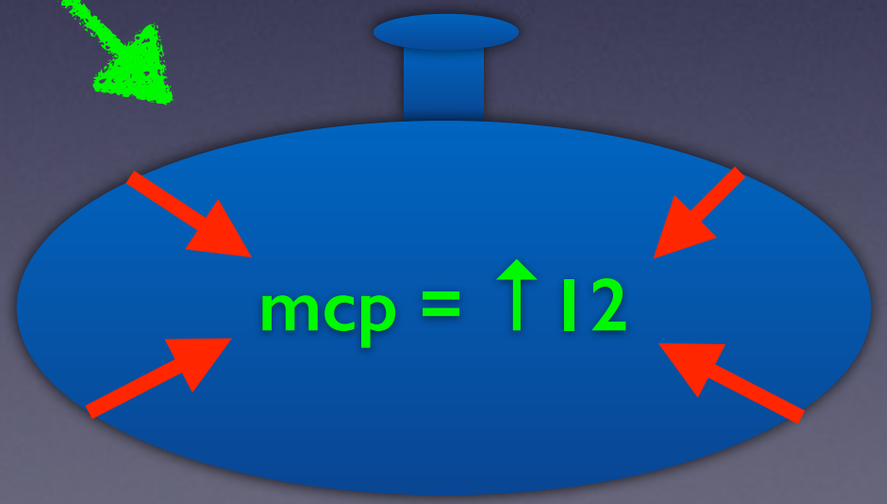
BEWARE!



venodilate

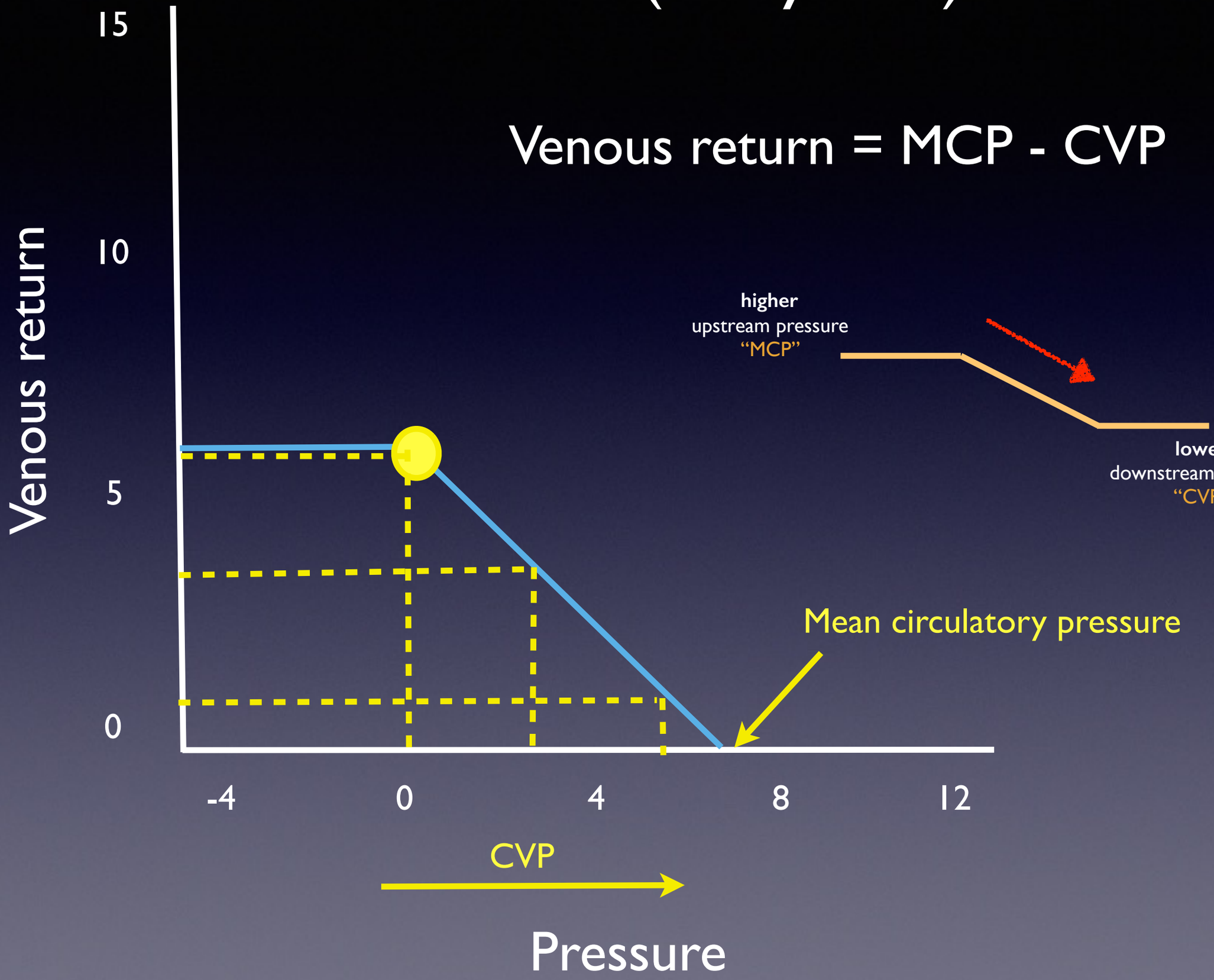


venoconstrict

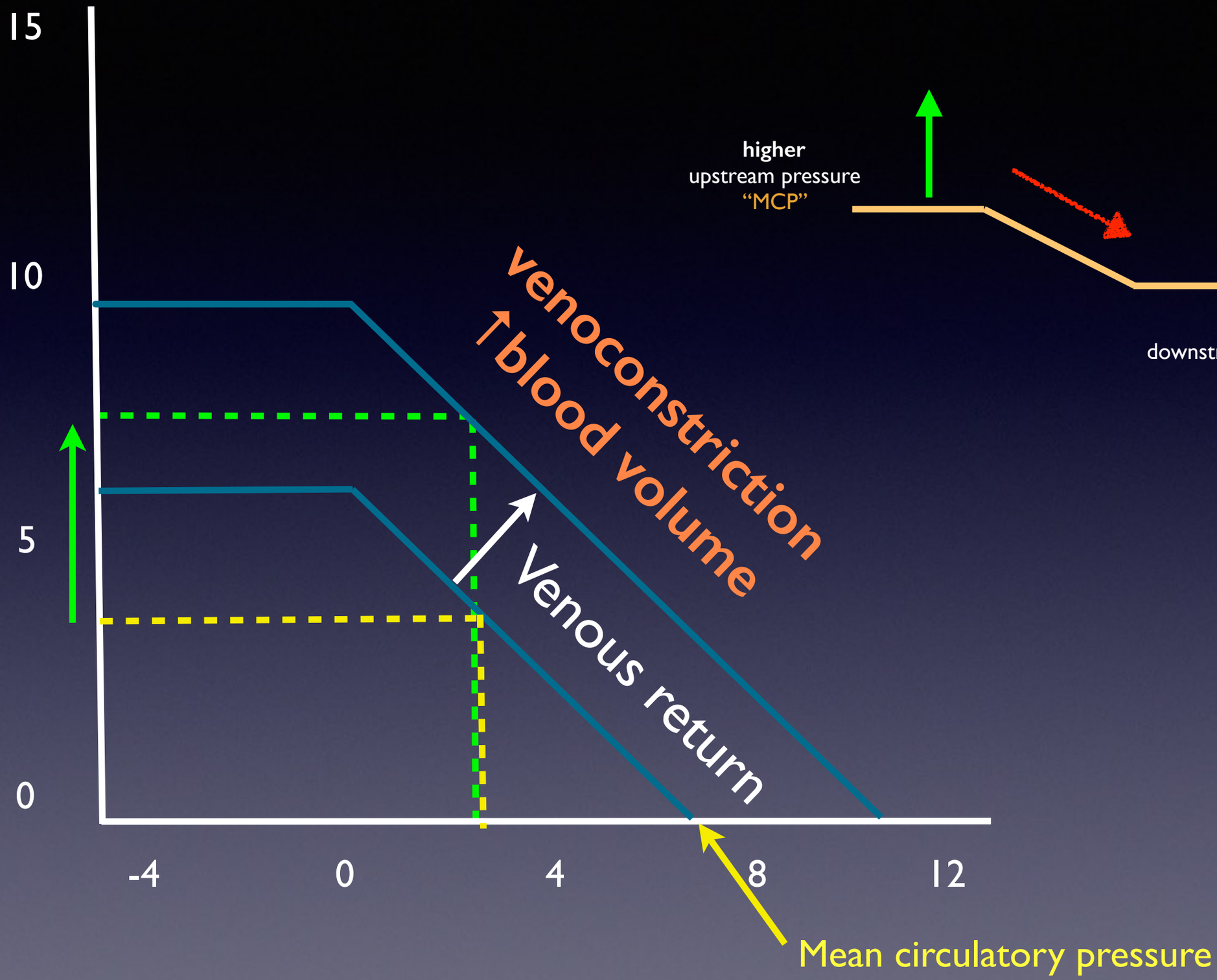


Venous return ("Guyton") curve

Venous return = MCP - CVP



Venous return curve



Starling meets Guyton

- The cardiac output and venous return axes are same

- Venous return = Cardiac output

but measured at the venous end

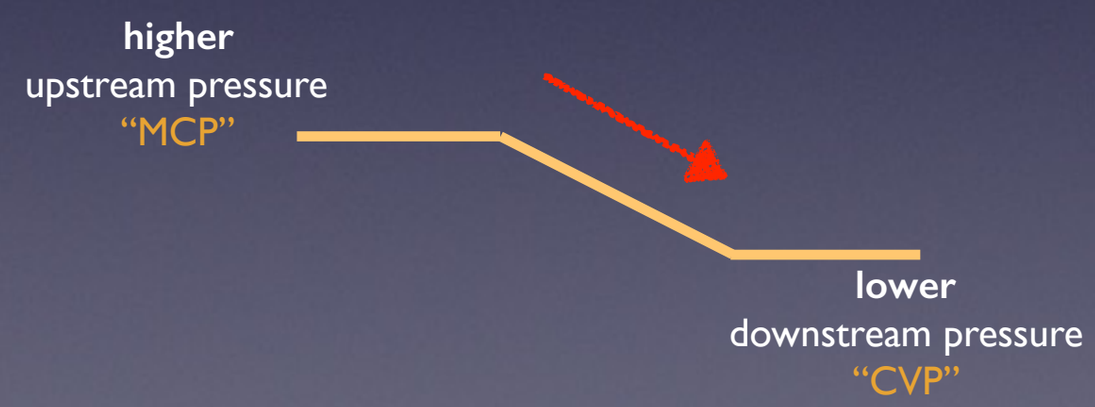
- The circulation is in steady state **only at one point**

  = where **CVP creates the <u>same</u> output and return**

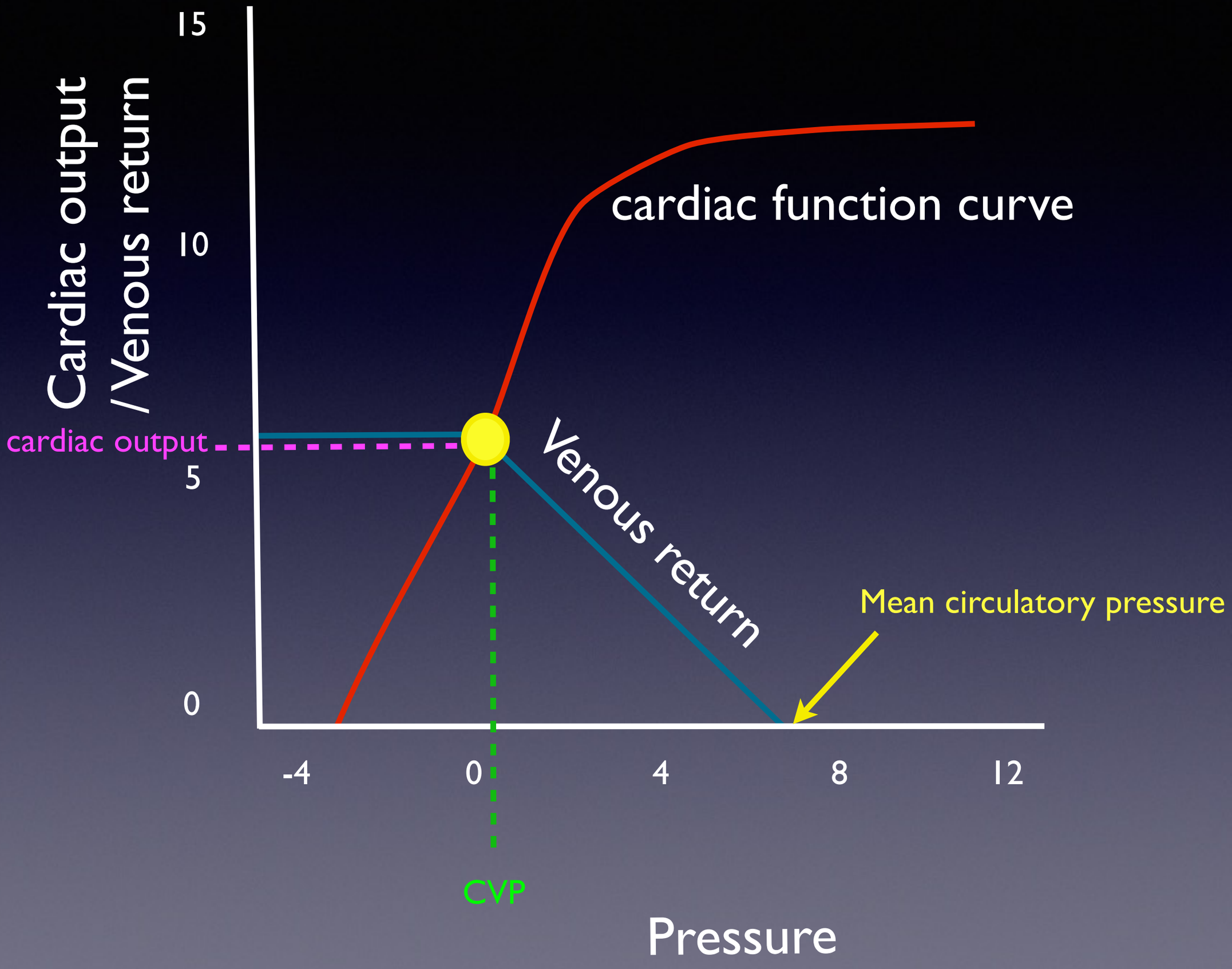
CVP serves 2 functions

"Opposes" venous return
(*Intraluminal* "Guyton")

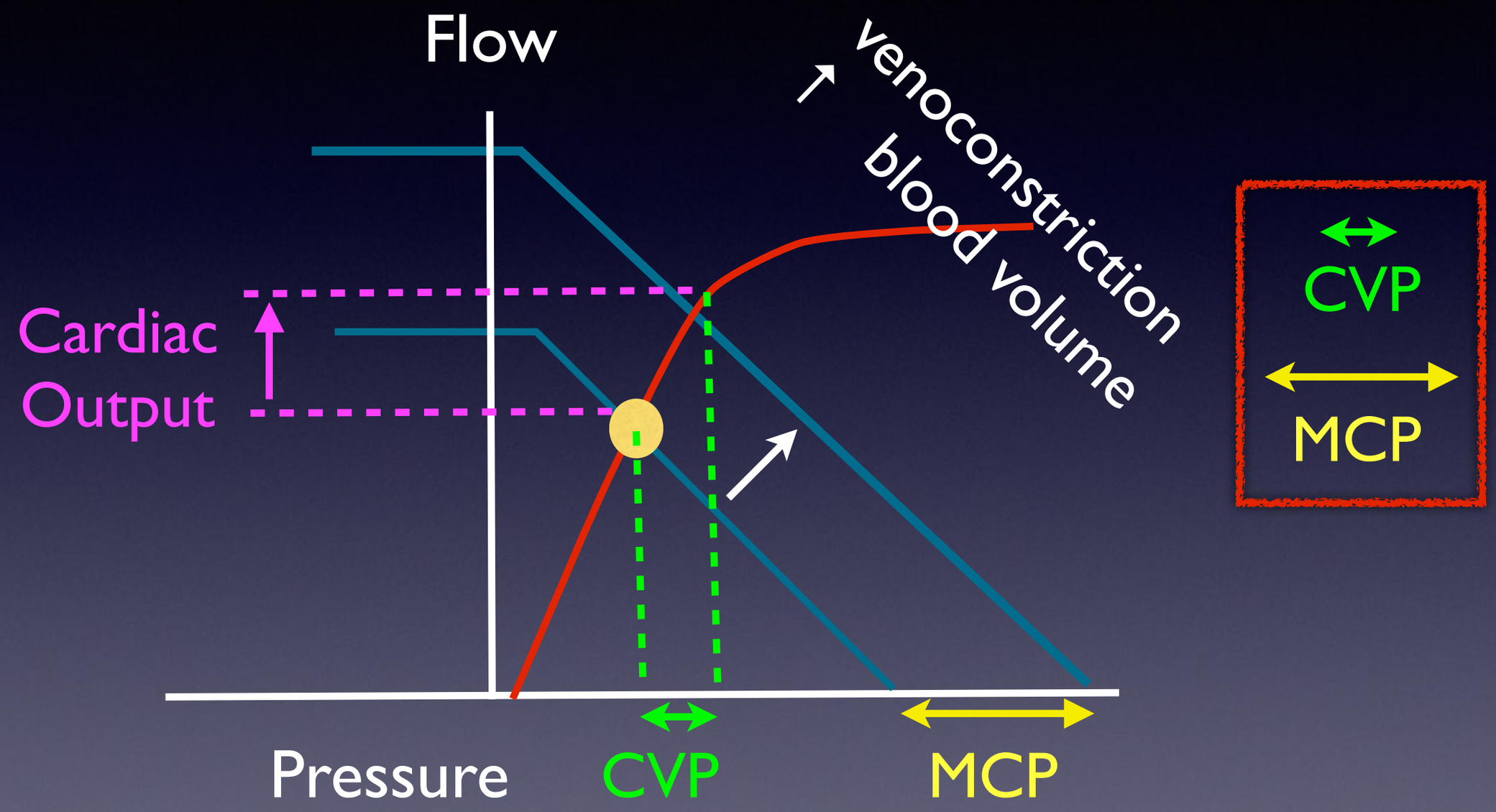
"Drives" the ventricle
(*Transmural* "Starling")



Starling meets Guyton

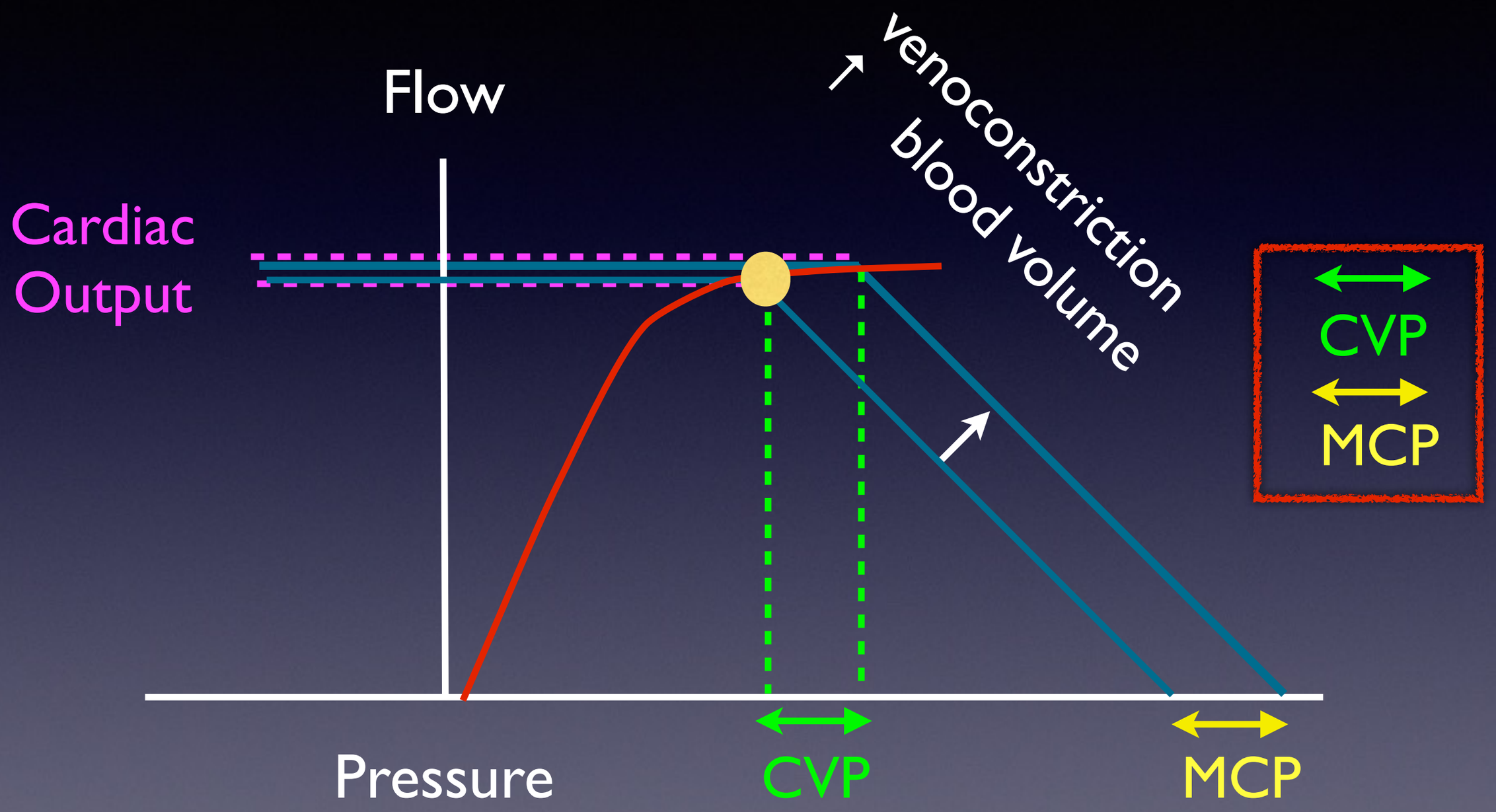


Increase in cardiac output by venoconstriction or increased blood volume



The heart is volume responsive, cardiac output increases……
the MCP **increases more** than CVP

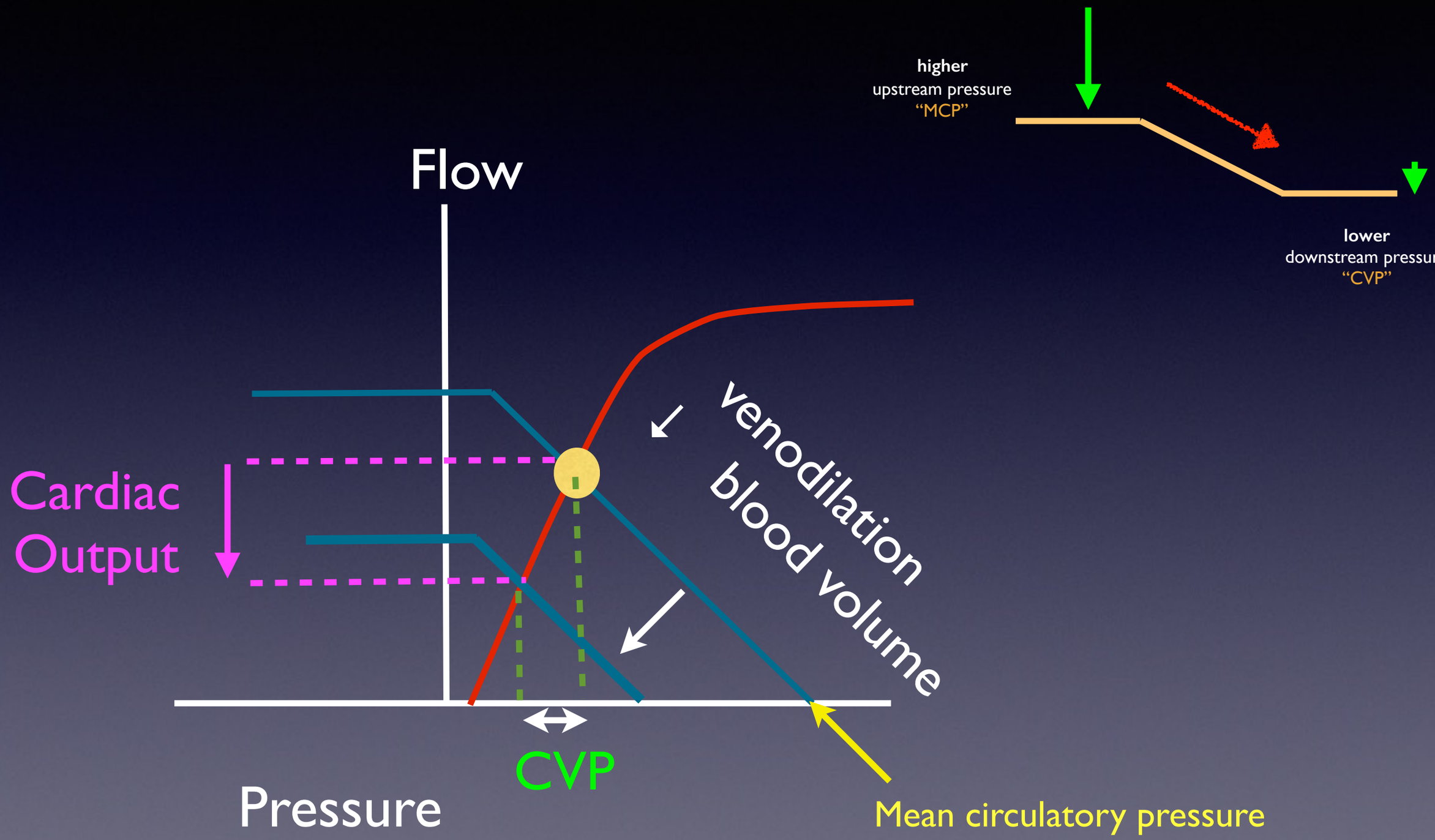
No change in cardiac output by venoconstriction or increased blood volume



The heart is volume unresponsive, cardiac output does not increase…..

the change in MCP ~ **equals** CVP

Decrease in cardiac output by venodilation or decreased blood volume



Current Opinion in Critical Care 2005, 11:264 -270

Clinical Case

Patient has suspected small bowel obstruction

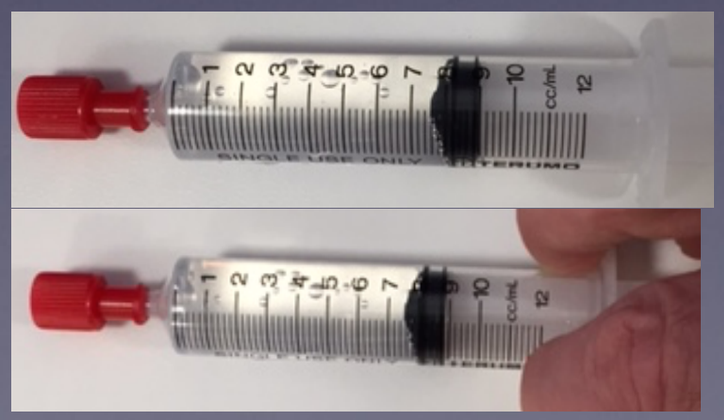
Taken to the operating room for laparotomy

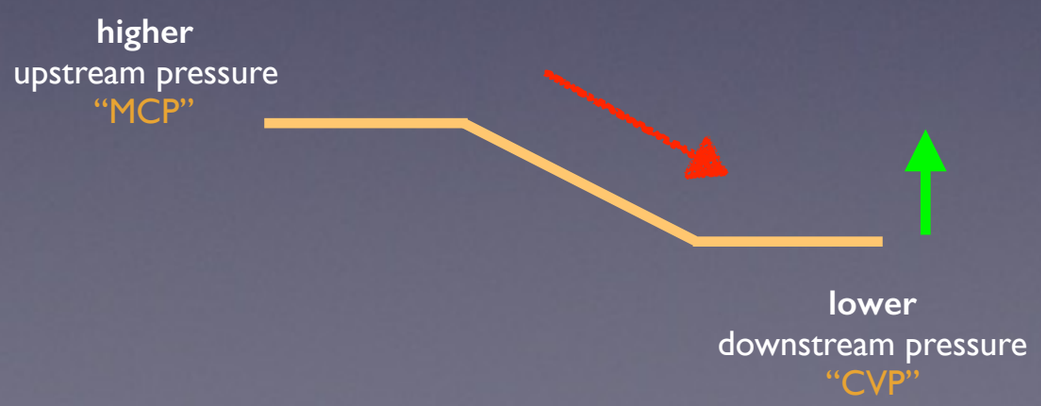
An “rapid sequence induction” performed

Drugs used are Propofol and Rocuronium

BP crashes

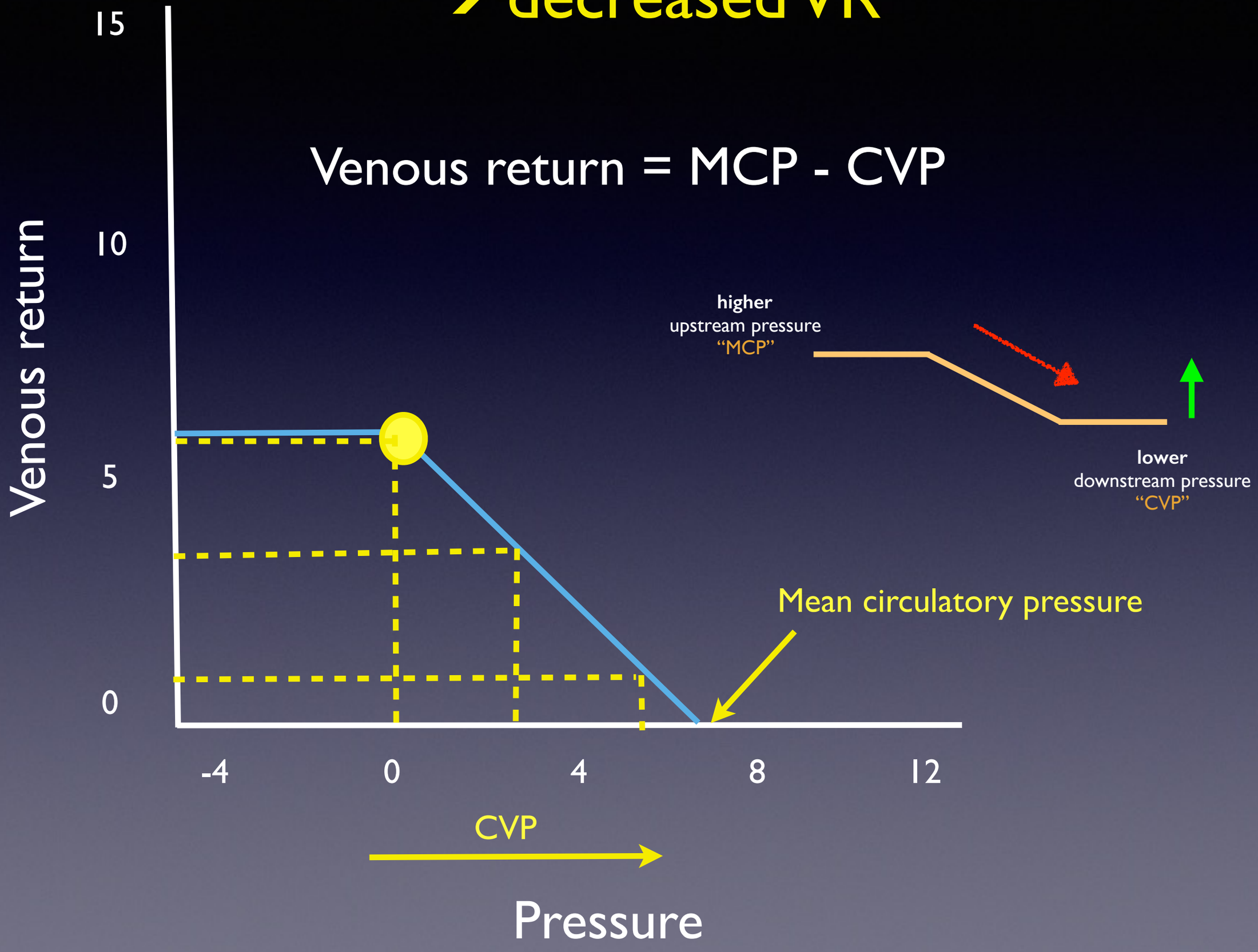
Why?





High airway pressure → high CVP → decreased VR

Venous return = MCP - CVP



Clinical Case

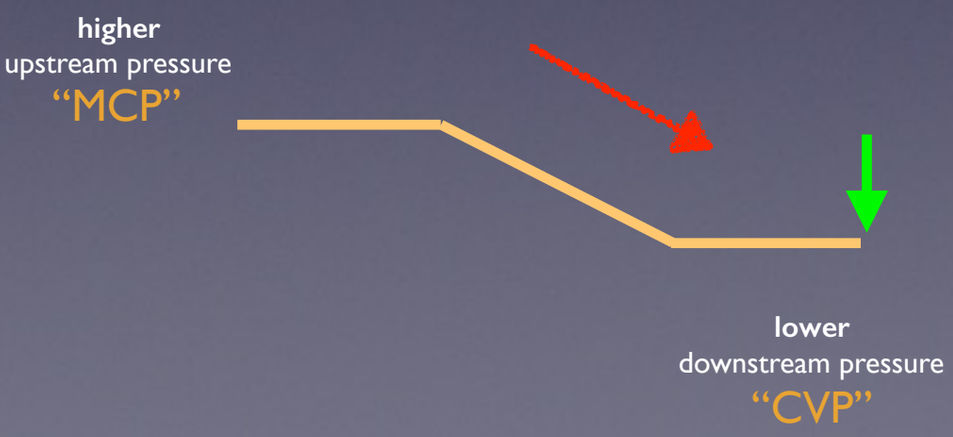
The flip side

During weaning from ventilation

"….intrathoracic pressure changes abruptly….

….venous return significantly increases, an acute increase in preload is imposed on the left ventricle.

….. shown by increased concentrations of brain natriuretic peptides."



Lancet 2016; 387: 1856–66

Recap

Double role of CVP

Inside chest

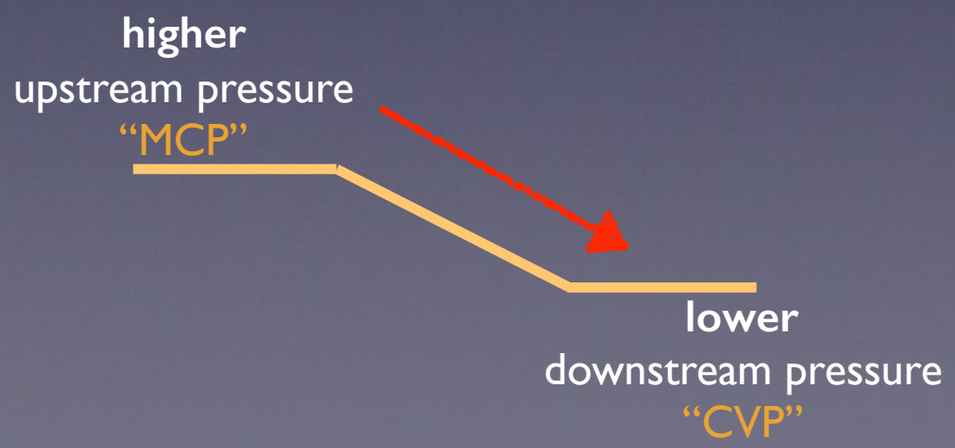
Determines cardiac "stretch" (intra-thoracic pressure-CVP)

"Starling" curve

Outside chest

Determines venous return (MCP-CVP)

"Guyton" curve



Recap

Think O2 Delivery

- Cardiac output - most important factor
  - Cardiac output x Hb x O2 saturation
- Cardiac filling - most commonly treated
  - Physiology of filling :
    - CVP - 2 roles
      - Starling
      - Guyton

???

www.jvsmedicscorner.com

Mallory/Everest2013

Fluid resuscitation - Effects of getting it wrong

Part 2

Dr J Vogel FRCA

Summary of this lecture

- Importance of cardiac output
- Physiology of cardiac preload and venous return
- Consequences of getting it wrong
  - Too much fluid
  - Too little fluid

Cardiac output - what are we trying to achieve?

- Adequate "effective" cardiac output
- Adequate blood pressure
- Adequate macro and micro-circulation

Correcting general haemodynamics is a pre-requisite but **not necessarily enough**.

“Adequate” cardiac output?

- Clinical signs
  - Normal BP
  - Warm toes
  - < 3 sec capillary refill
  - Normal sensorium
  - Urine output
  - Small core-peripheral temperature gradient
- Biochemistry
  - ScV02
  - Lactate
  - Base deficit
- Advanced technology
  - “Visualising” the micro-circulation

Clinical Case

59 yr old male

Previous healthy and active

Undergoing routine surgery

Suddenly develops massive myocardial infarction

Taken to ITU with cardiogenic shock

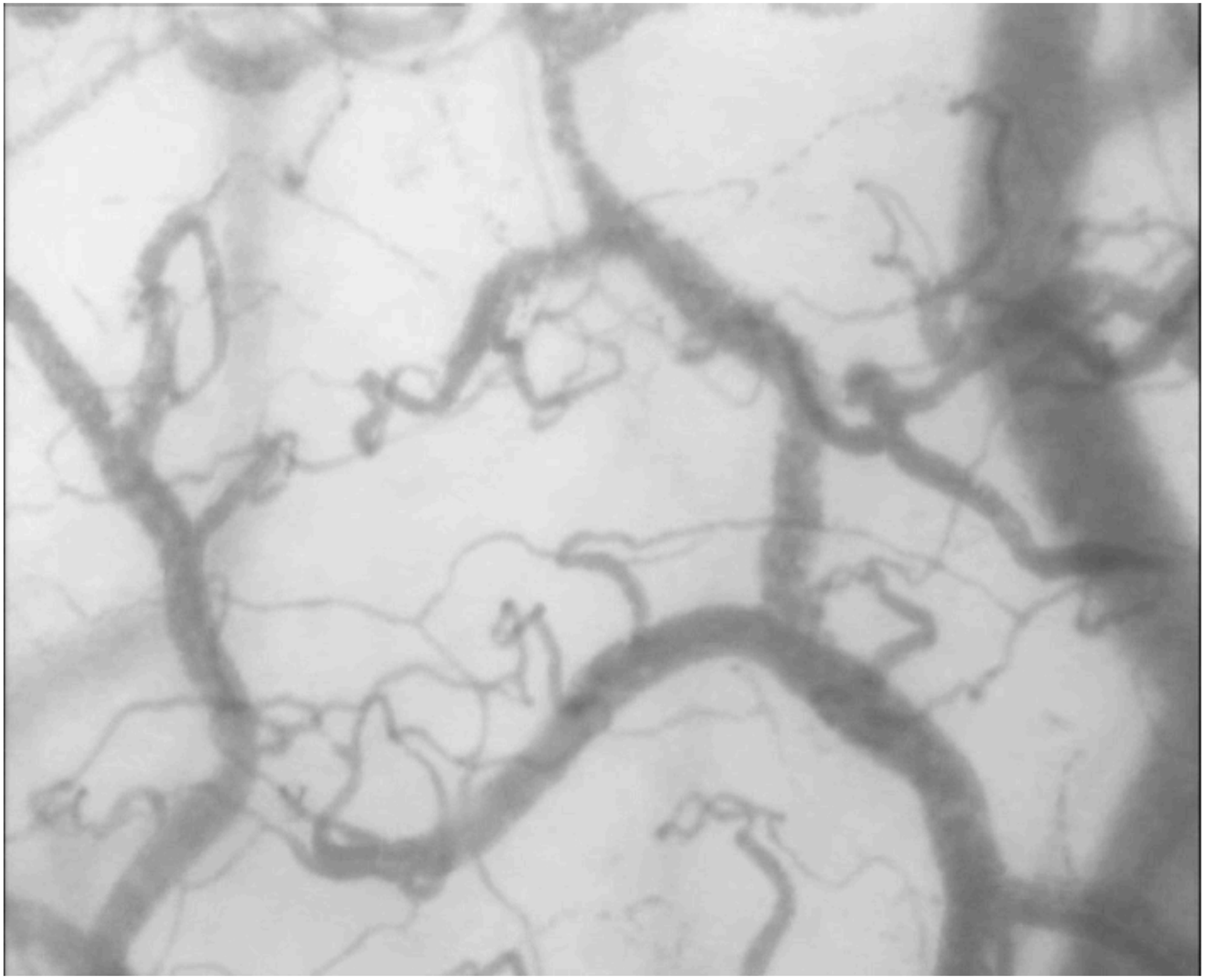
low BP, low cardiac output, low O2 sats and pulmonary oedema

On maximal doses of inotropes

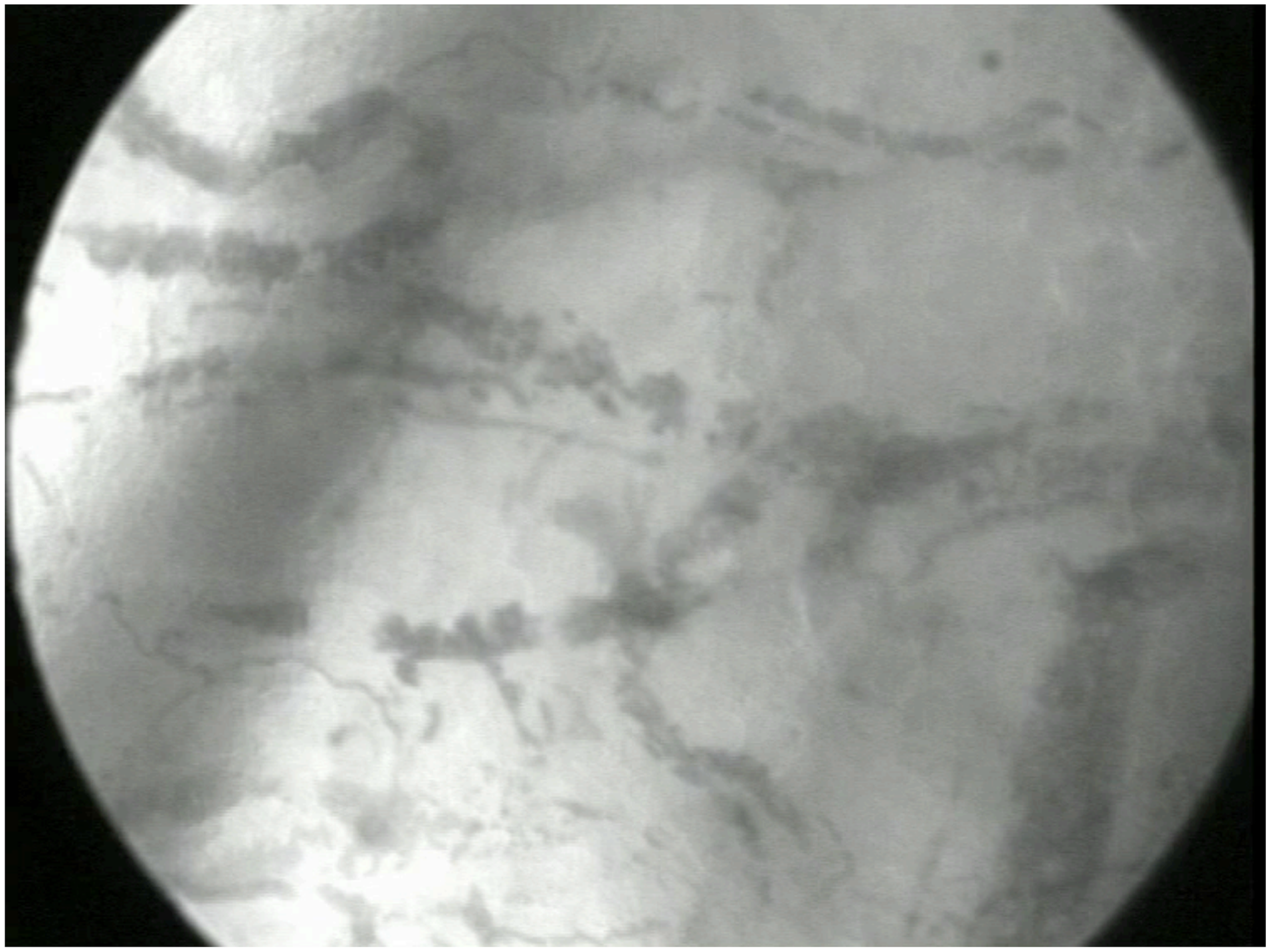
Fluid or not?

Do we have “adequate” microcirculatory flow?

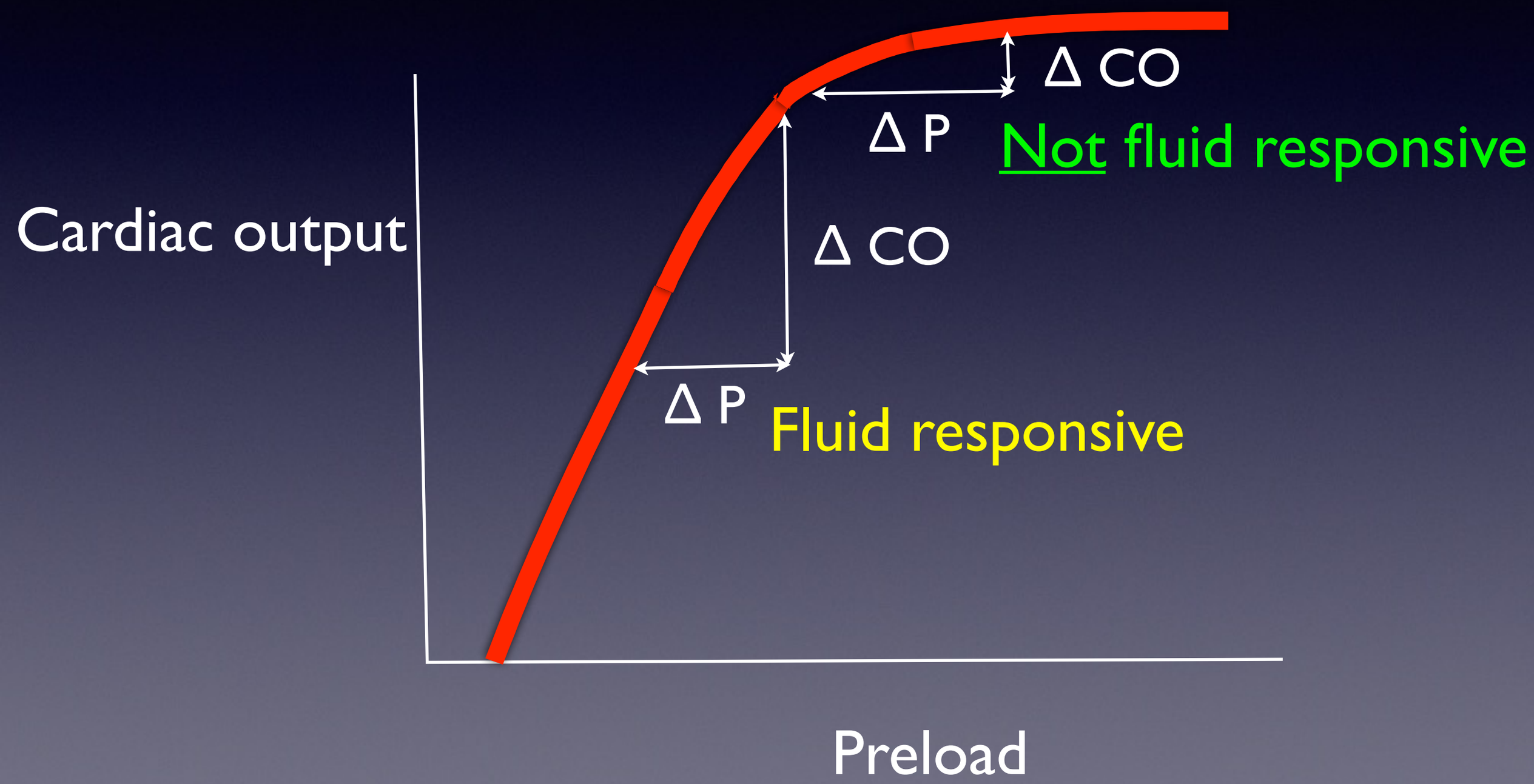
Normal microcirculatory flow



Microcirculation in cardiogenic shock



Fluid responsiveness
= where is patient on the Starling curve?



Clinical Case

63 year old woman

3 days nausea, vomit, diarrhoea

Arrives on the Thursday at 16:30

BP 70/40; HR 110; urine output 10ml/hr; lactate 7

9.5 L crystalloid

BP 80/40; HR 115; urine output 5ml/hr; lactate 14

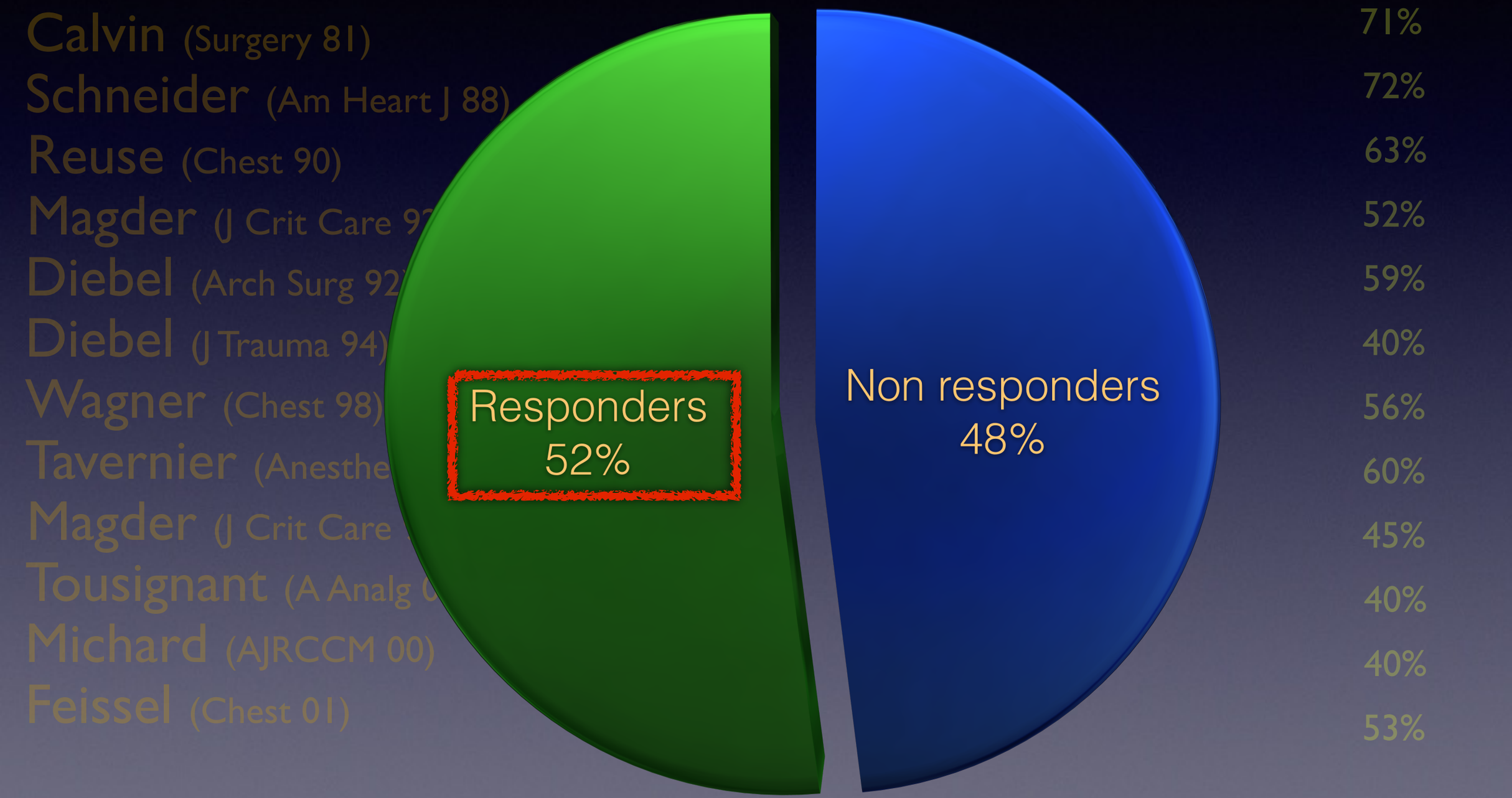
Friday 07:30

Call ITU and ask for admission

20 min later patient arrests and dies

What were the 2 things that went wrong?

Half of ITU patients are fluid responders



Michard & Teboul. Chest 121:2000-8, 2002

How to tell if fluid responsive

Either

- Give a bolus and watch response

  - Probably safe if small volumes required

  - If small risk of pulmonary oedema

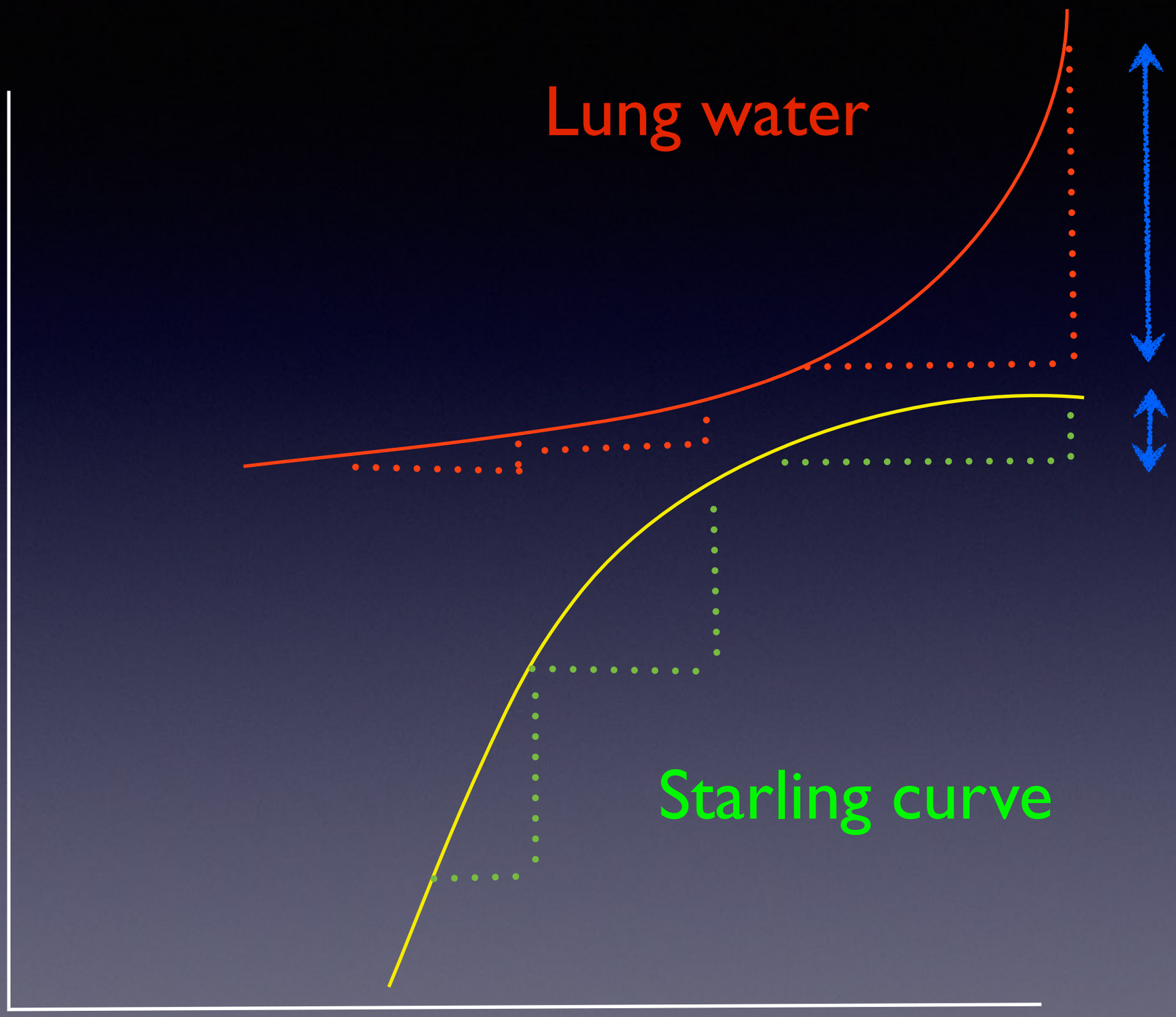
Or

- Try to **predict** how patient will respond

Predicting fluid responsiveness

Consequences of too much fluid

Why try predicting fluid responsiveness?

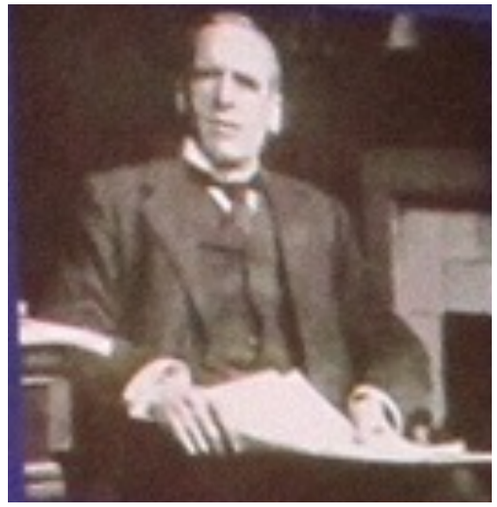


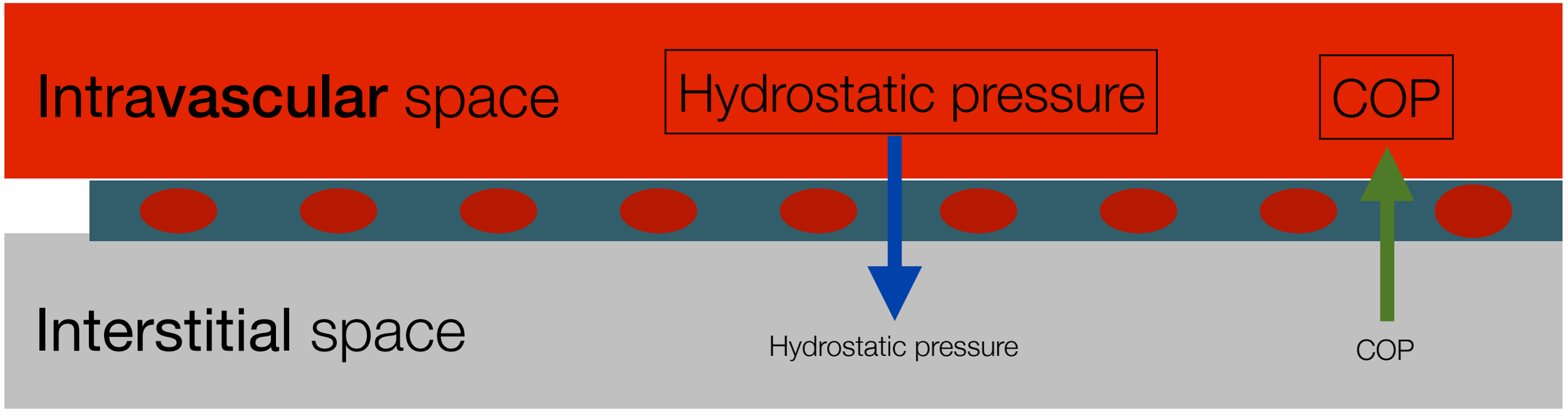
Too fluid much endangers the ……..Glycocalyx

The glyco…..what?

Single vascular barrier

Ernest Starling: 1866-1927





Microvascular fluid exchange and the revised Starling principle

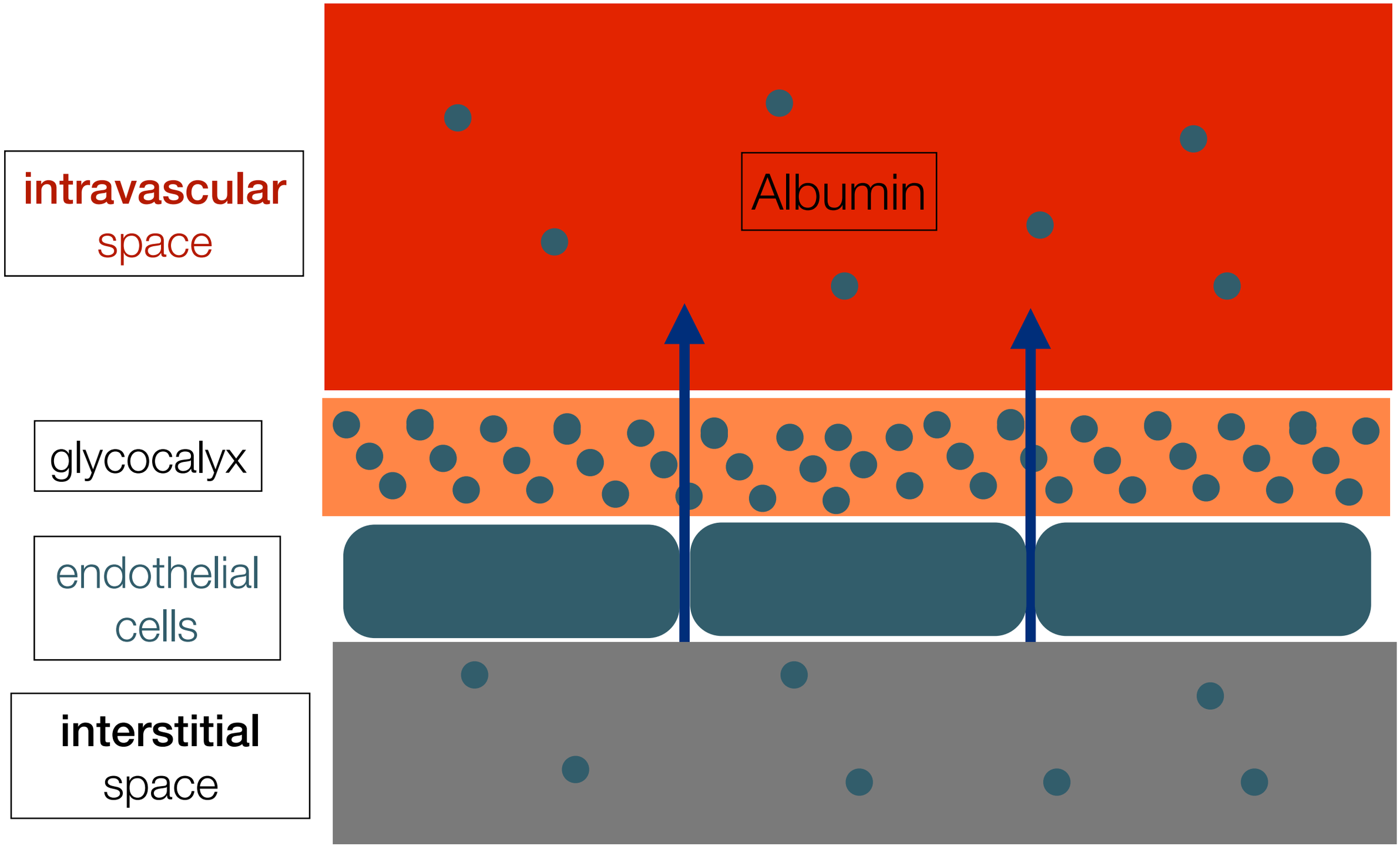
But.....

- **lymph** flow produced is **orders of magnitude smaller** then predicted

- In experiments, even when the COP inside and outside of the vessel were **equal**, there was **still effective COP drawing fluid in !**

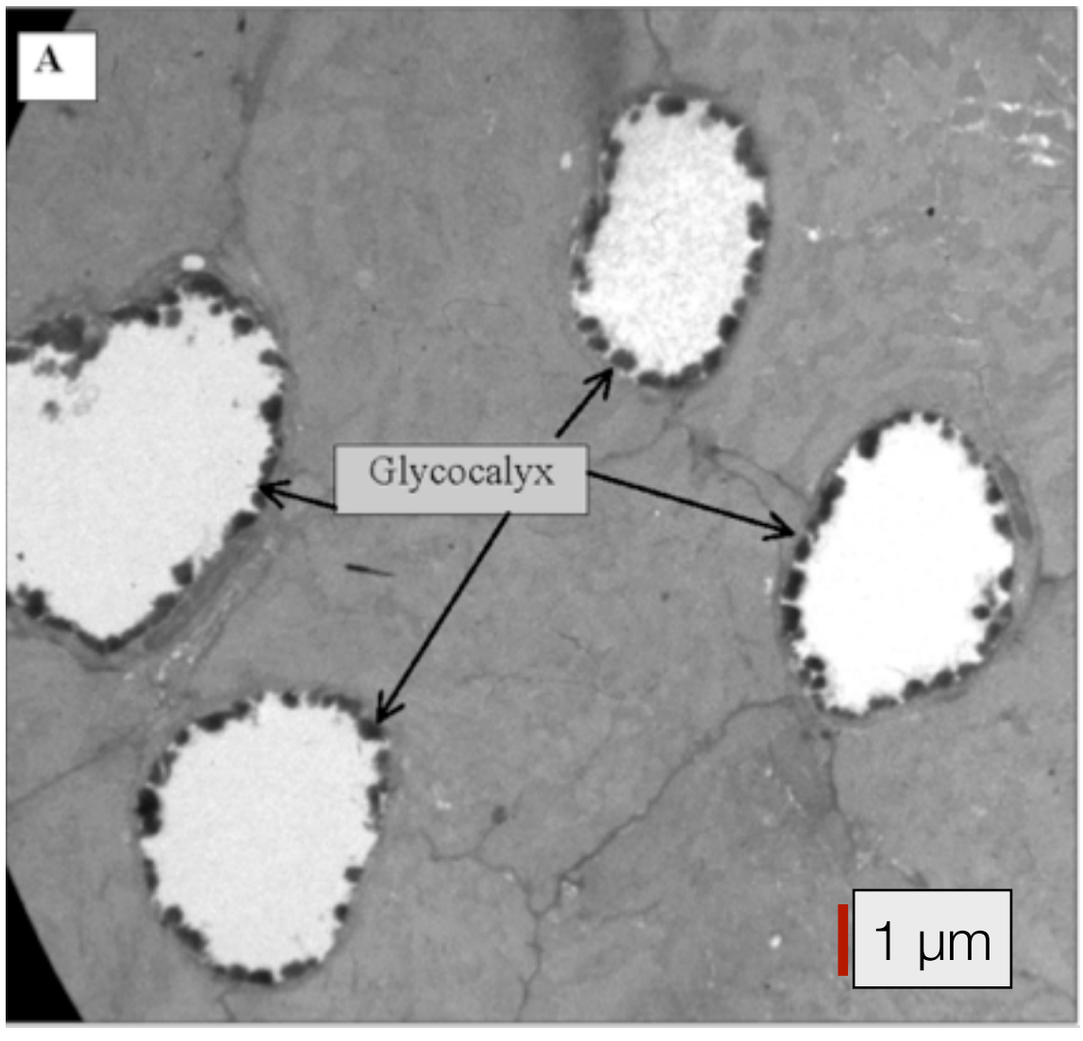
Cardiovascular Research (2010) 87, 198–210

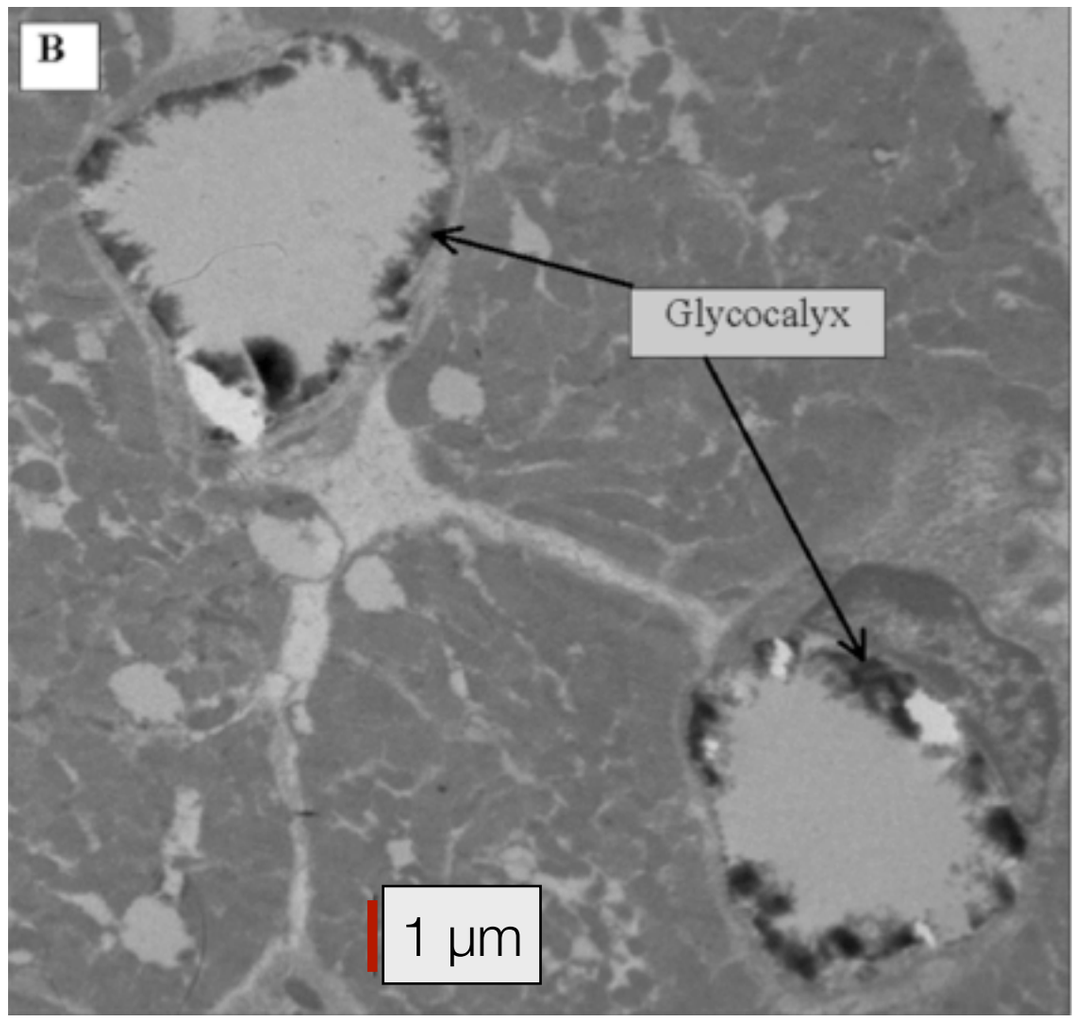
Double vascular barrier



Rehm et al Anesthesiology 2004;100:1211

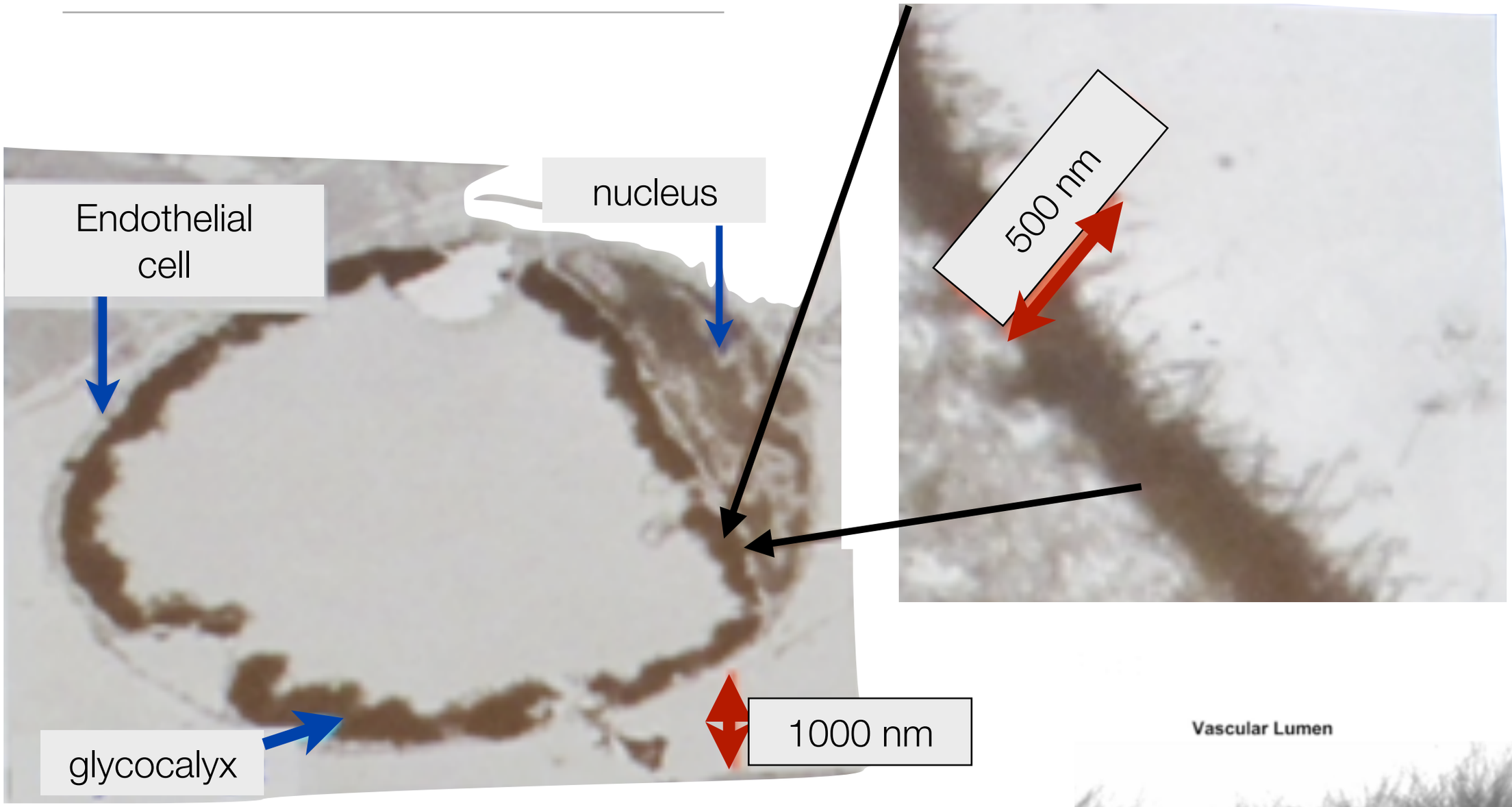
Glycocalyx - electron microscopy

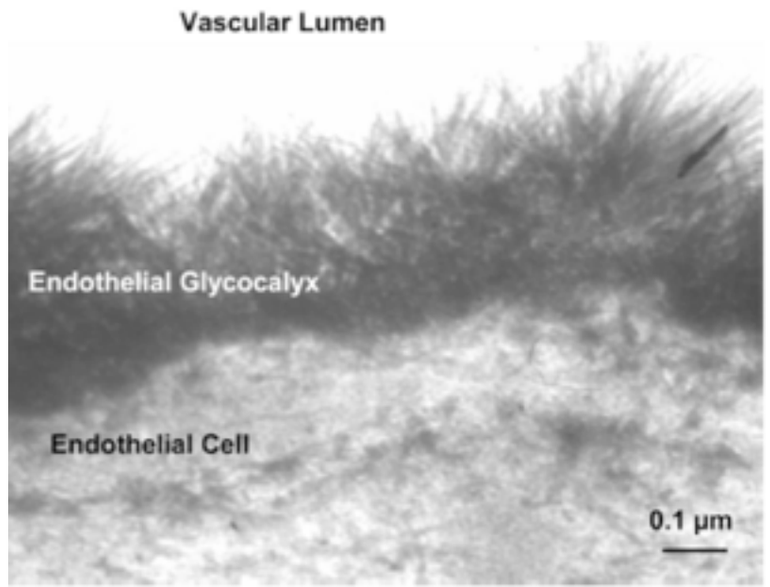




Rehm et al Anesthesiology 2004;100:1211

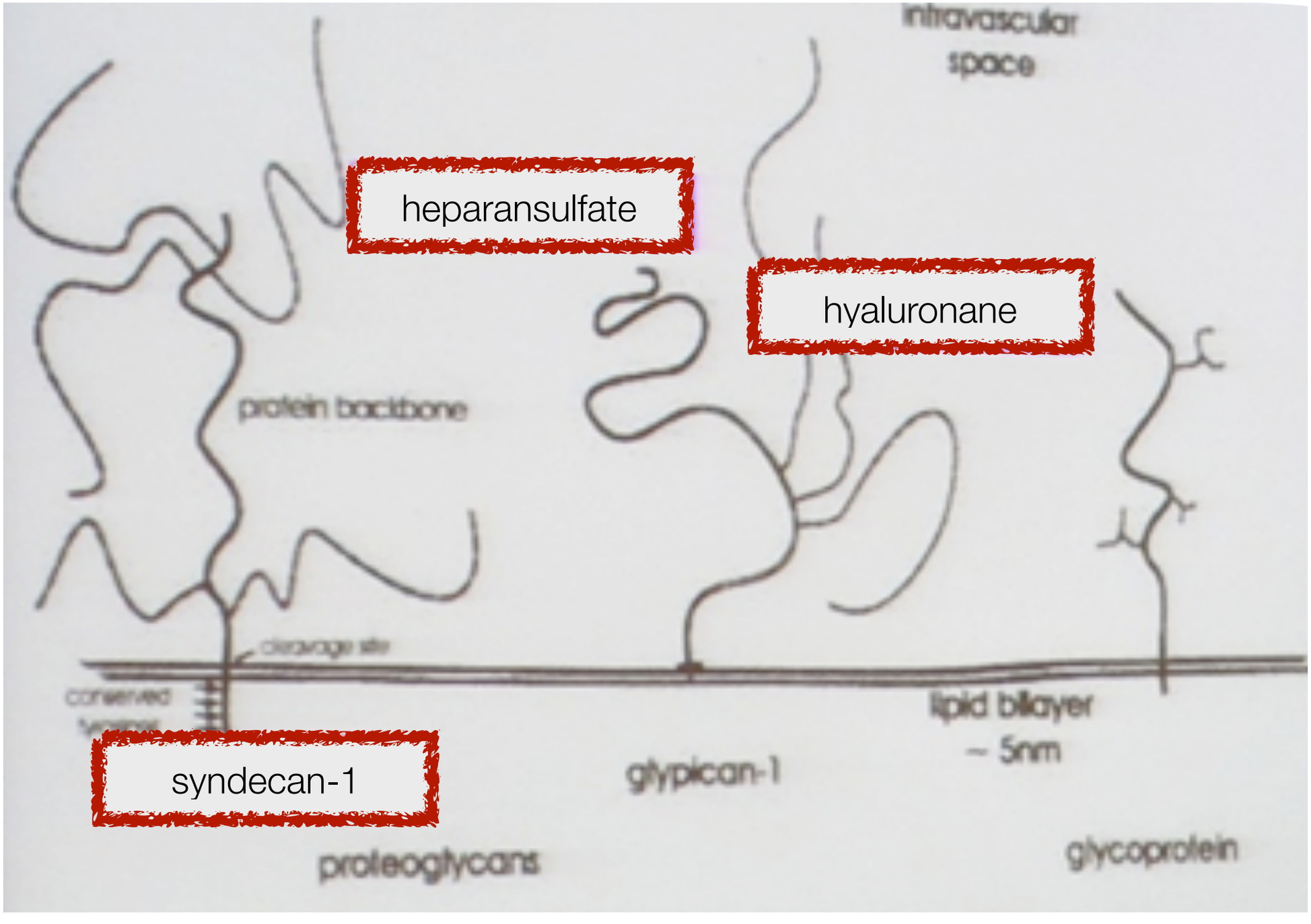
Electron microscopy - glycocalyx





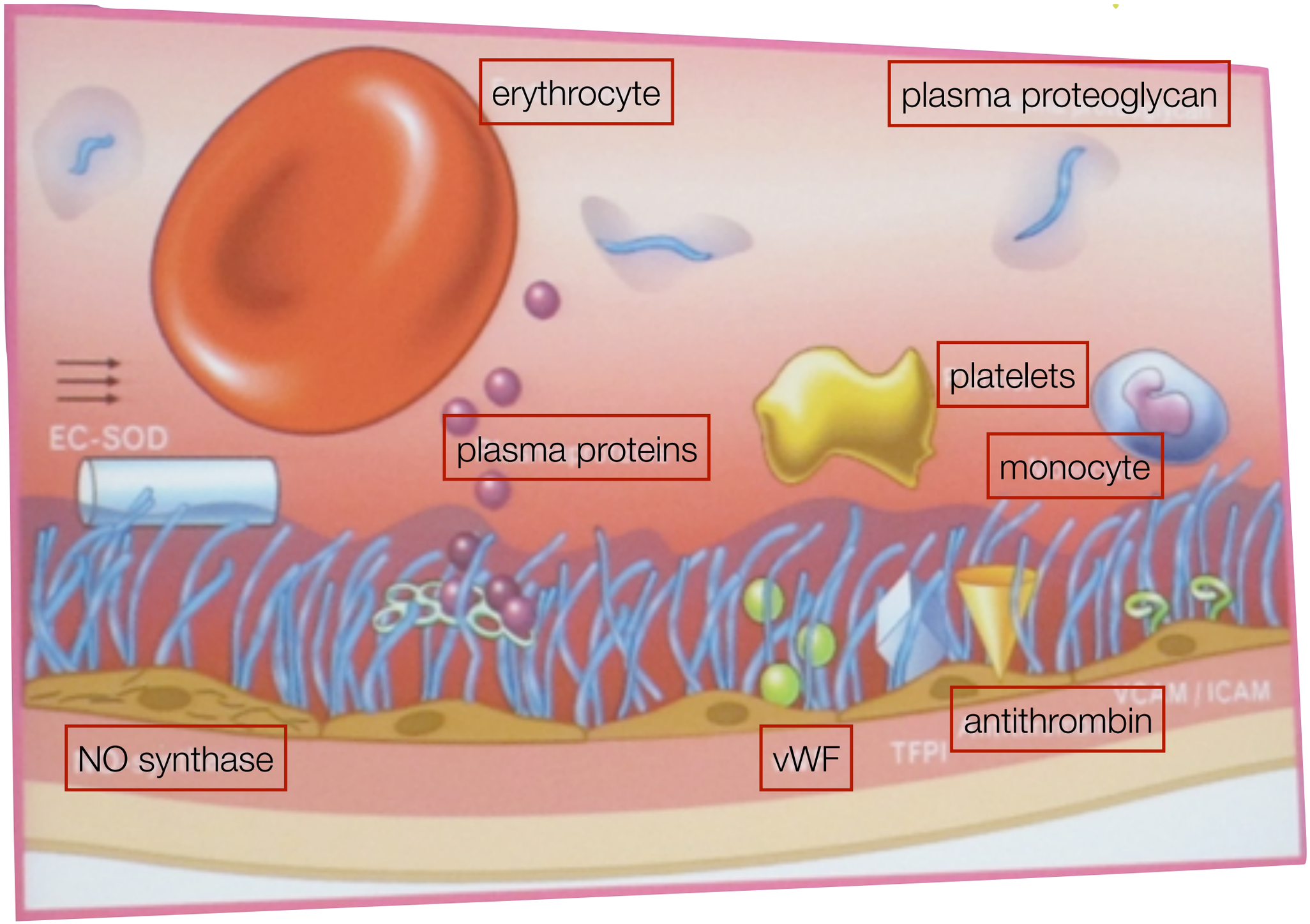
Rehm et al Anesthesiology 2004;100:1211

Glycocalyx - components



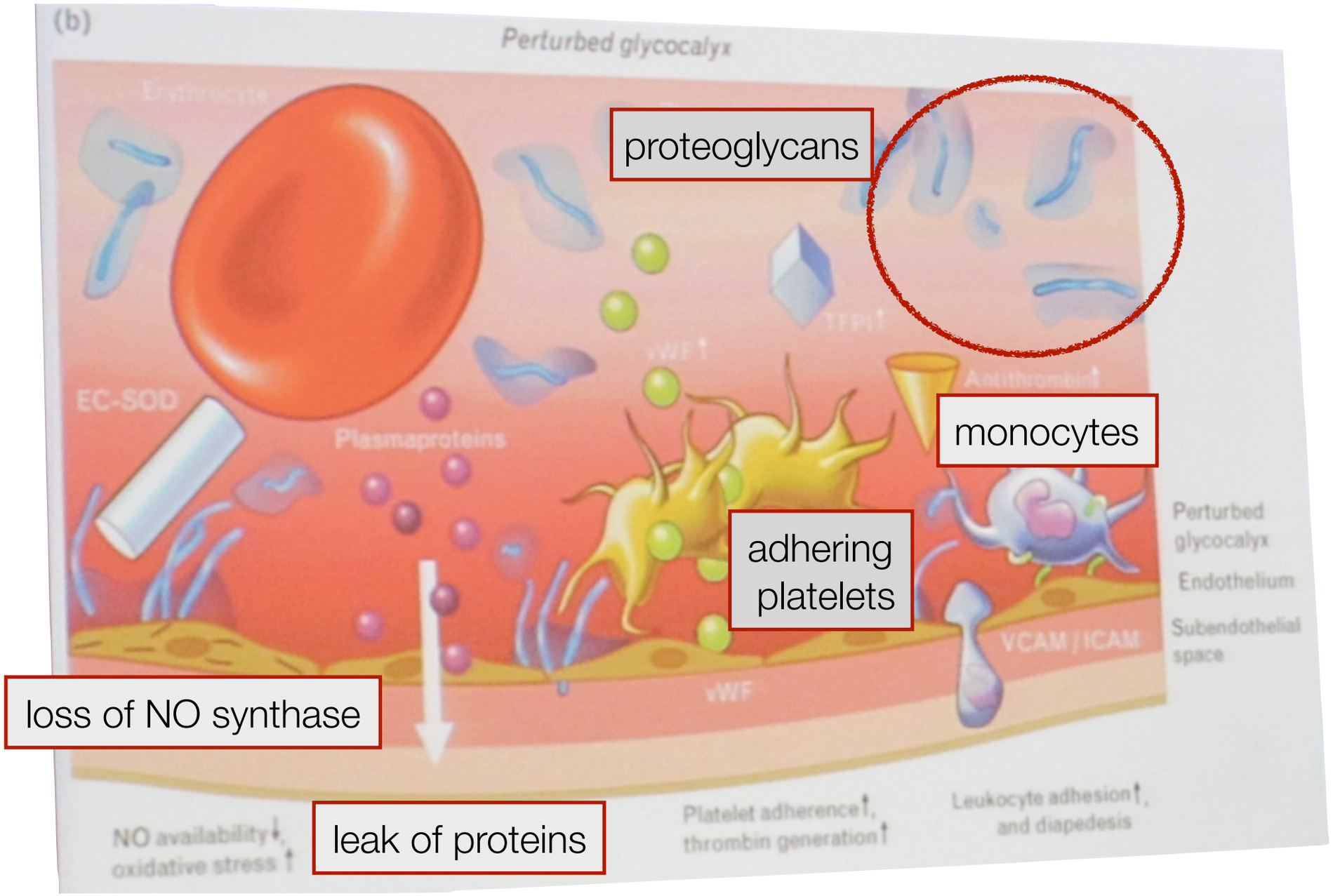
Pries et al Eur J Physiol; 440:653-666, 2000

Healthy endothelial glycocalyx



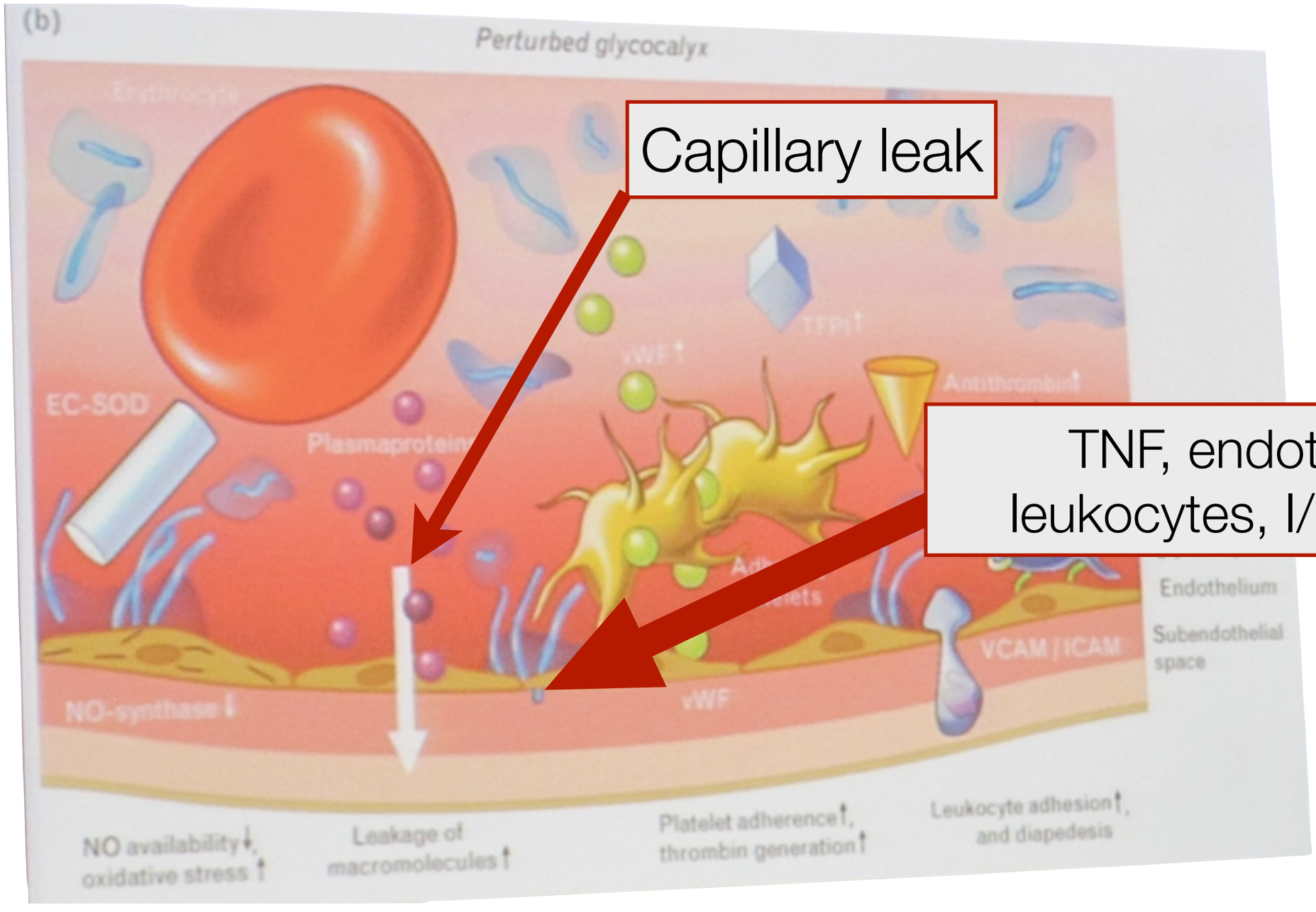
Nieuwdorp et al Curr Opin Lipidol 2005; 16:507

Destruction of the glycocalyx



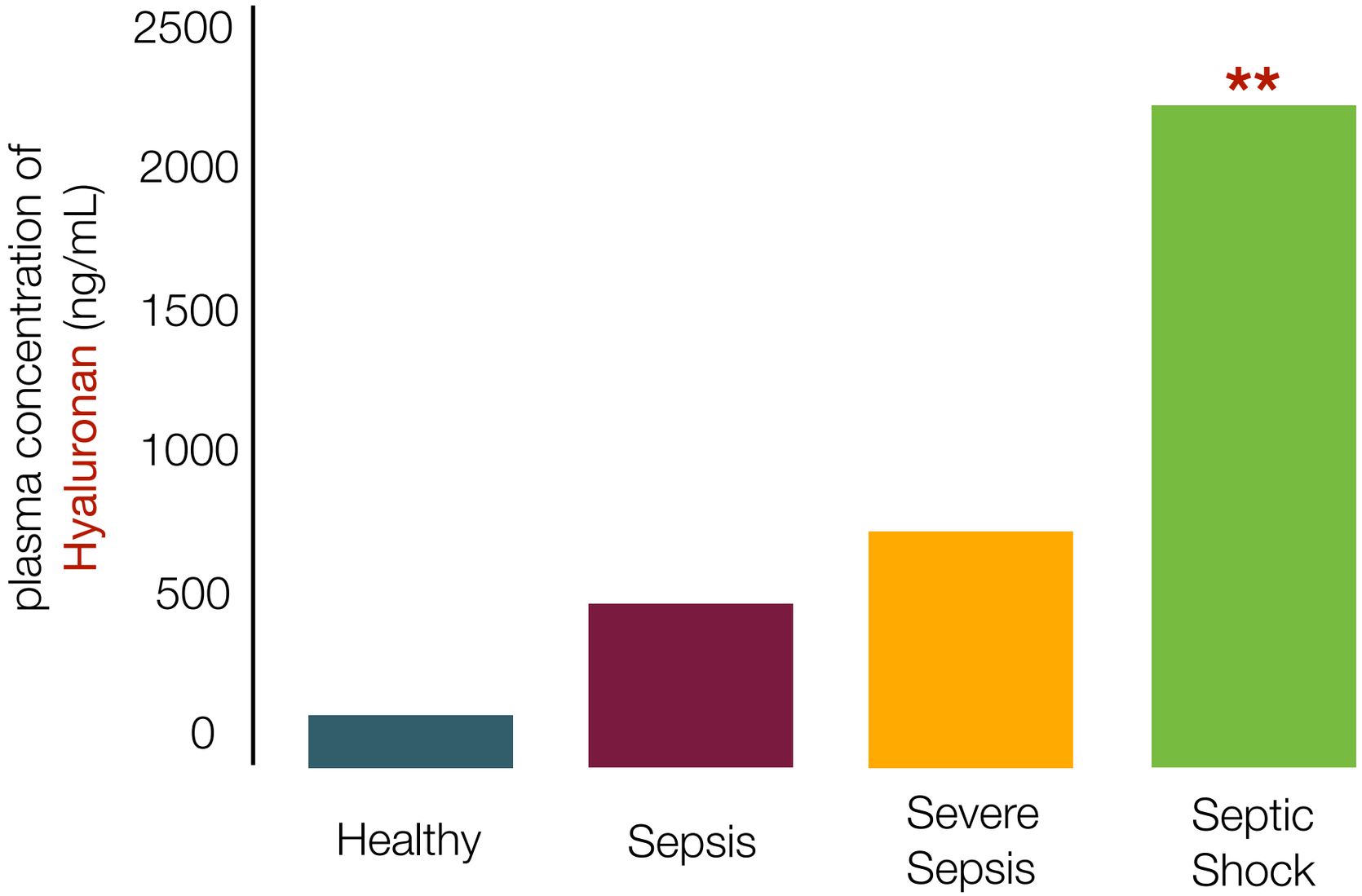
Nieuwdorp et al Curr Opin Lipidol 2005; 16:507

Sepsis-destruction of glycocalyx



Nieuwdorp et al Curr Opin Lipidol 2005; 16:507

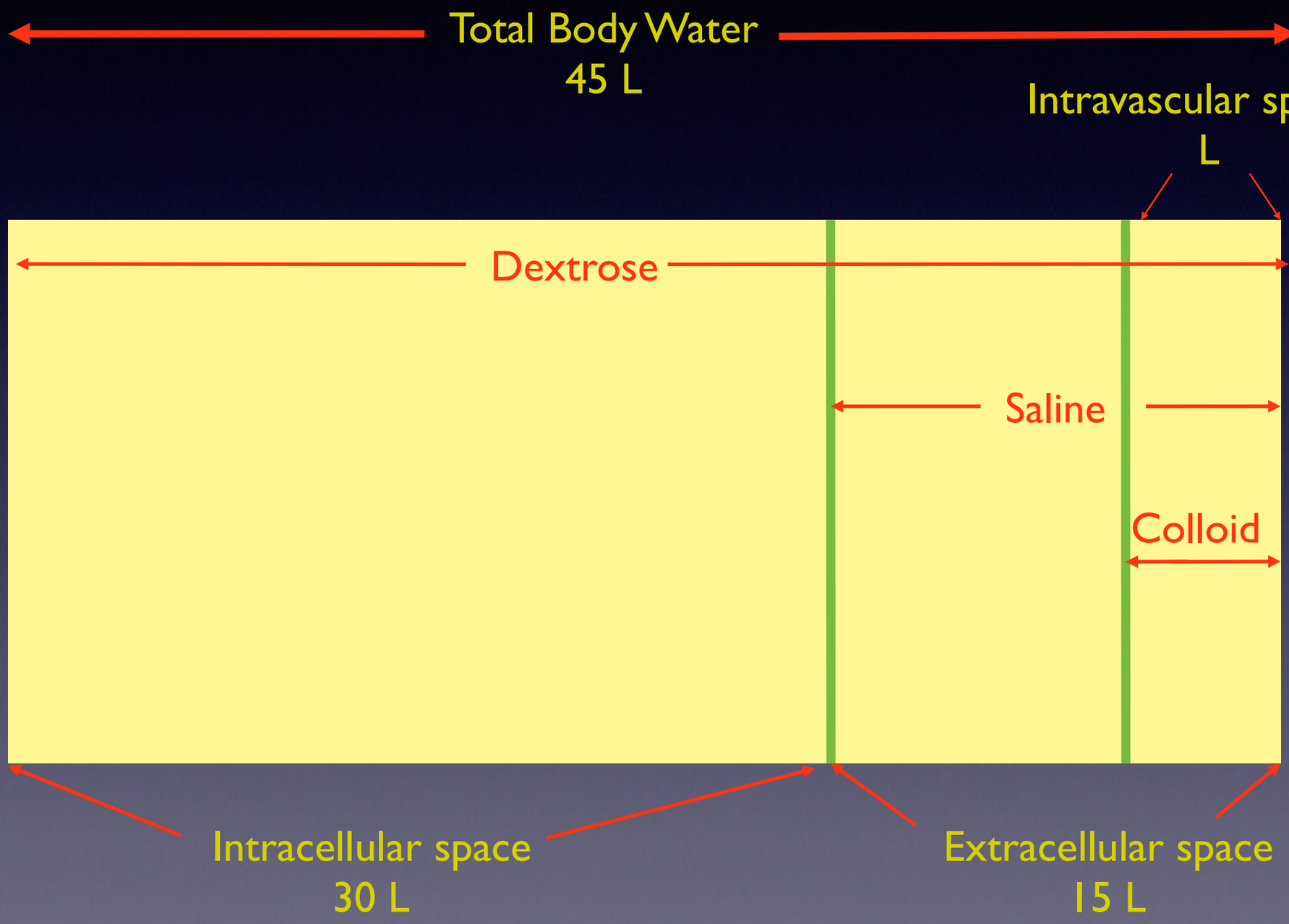
Glycocalyx in sepsis



Increased shedding of glycocalyx in plasma with increasing severity of illness...a **prognostic factor**

Kohler et al Infection 39:117-118; 2011

All Fluids are Not Created Equal



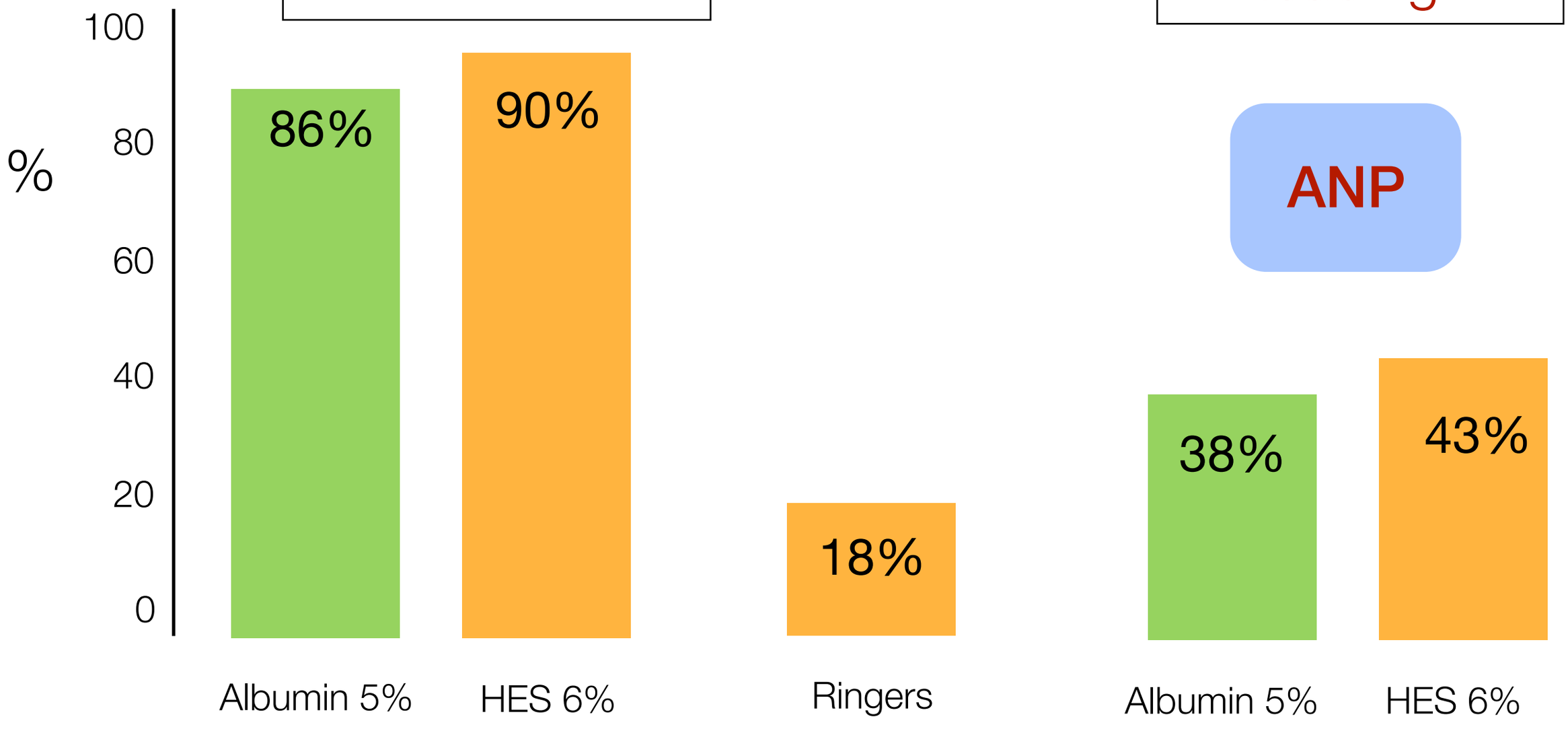
1 L infused, how much stays **intravascular ?**

- Dextrose - **70** ml
- Saline - **250** ml
- Colloid - **1000** mL

Glycocalyx - volume of colloids effects are “context sensitive”

normovolaemic hemodilution

hypervolaemic “volume loading”



Alterations of the glycocalyx reduces the volume effects of colloids

Jacob et al Lancet 2007 16;369:1984-6

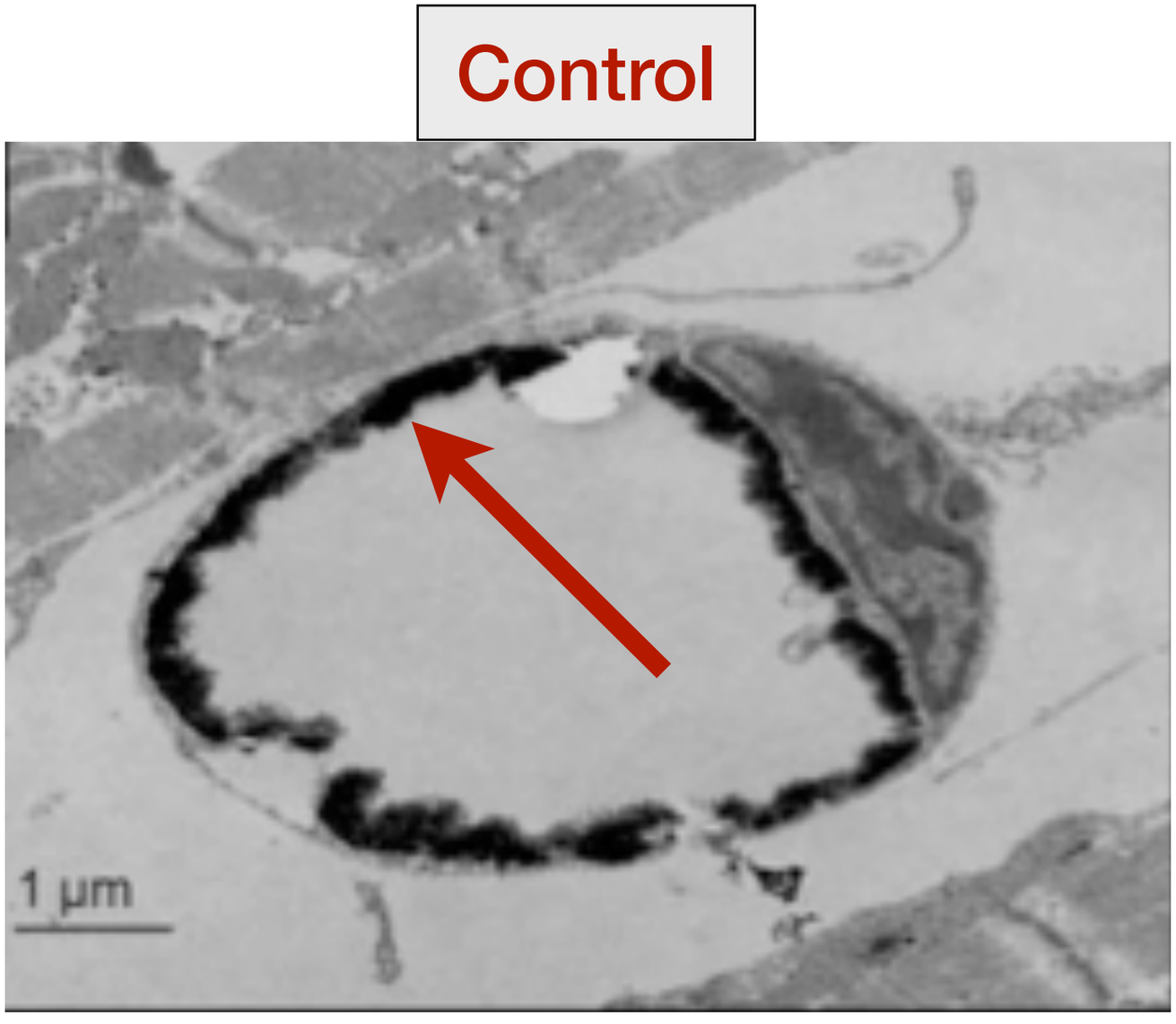
Atrial Natriuretic Peptide (ANP)

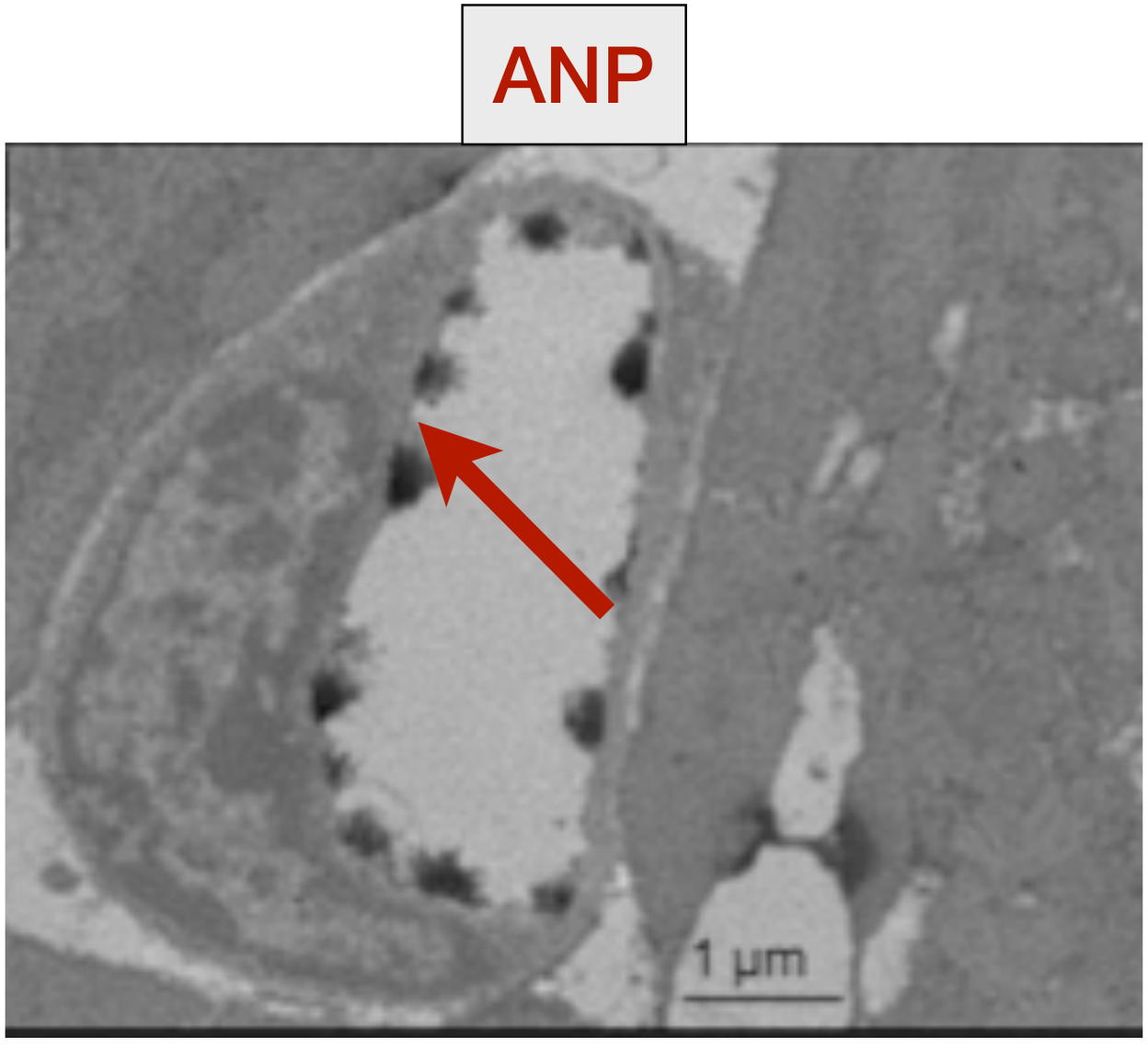
a cardiac hormone released by acute volume loading, plays a key role in blood volume regulation

Isbister (1997) Trans Sci; 18:409-423

Tucker (1996) Am J Physiol; 271:R591

ANP “strips off” the glycocalyx





Am J Physiol Heart Circ Physiol 289: H1993–H1999, 2005

Glycocalyx - summary

Large structure with **important functions**

- Vascular barrier function
- Thrombocyte and leucocyte adhesion (“teflon”)
- Inflammation
- Vessel diameter

Jacob et al Anesthesiology 2006, 104:1223-1231

Jacob et al Cardiovasc Res 2007, 73:1235-1242

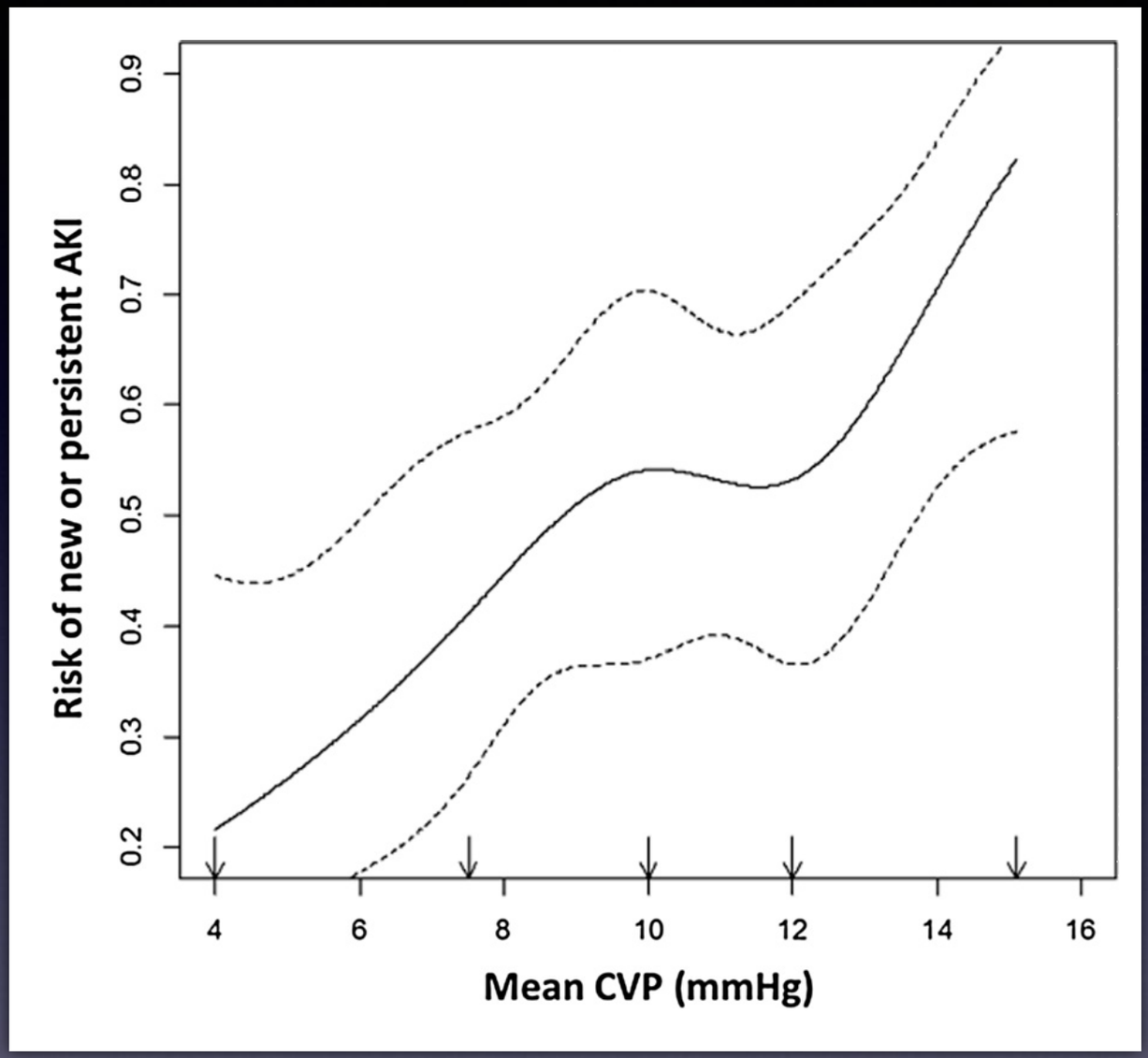
Other consequences of too much fluid

If <u>extreme</u>, high venous pressures do have negative "upstream" consequences

- Right heart
  - Septal shift (impairs Lt Ventricle)
  - Kidney
  - Liver (cardiac cirrhosis)
- Gut
- Head (raised ICP)
- Lungs (reduced lymph flow)

Use CVP to measure "safe limit" when fluid resuscitating

Venous congestion: are we adding insult to kidney injury in sepsis?

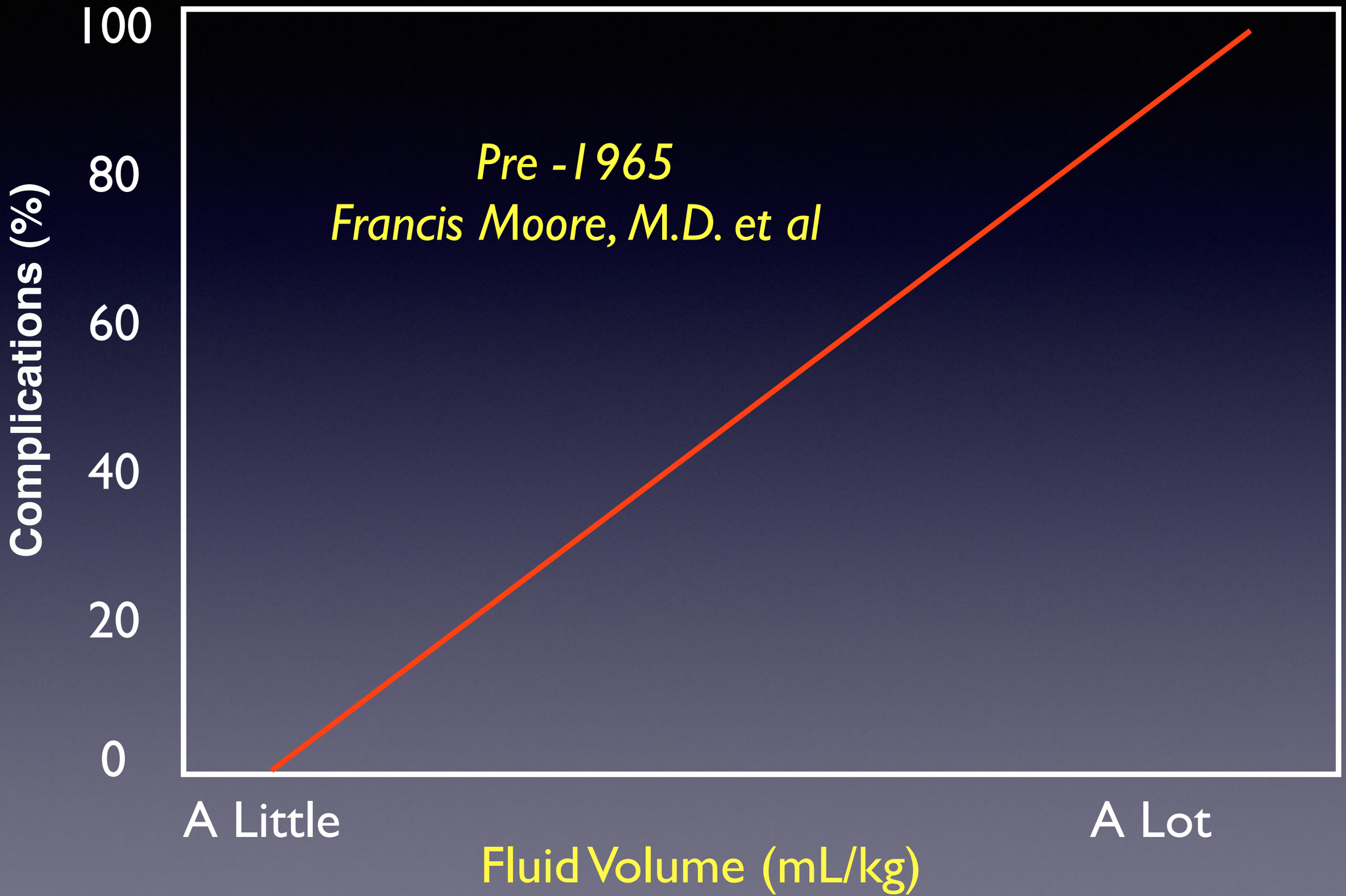


"… a **5 mmHg** increase in CVP predicted **2.7-fold odds** of **AKI**."

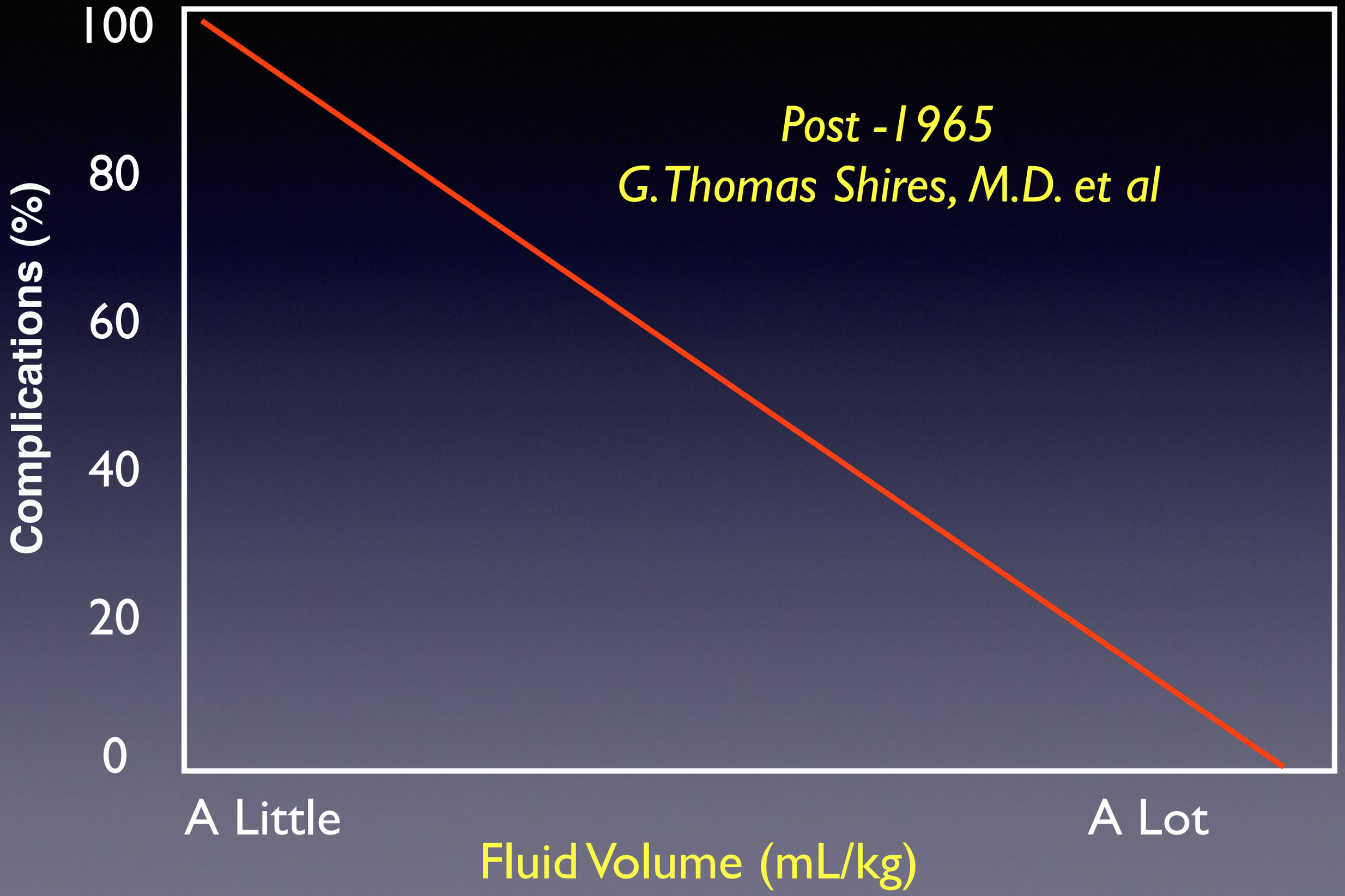
"Renal outcomes were worse for all CVPs from 4 mm Hg and above"

Critical Care 2014, 18:104

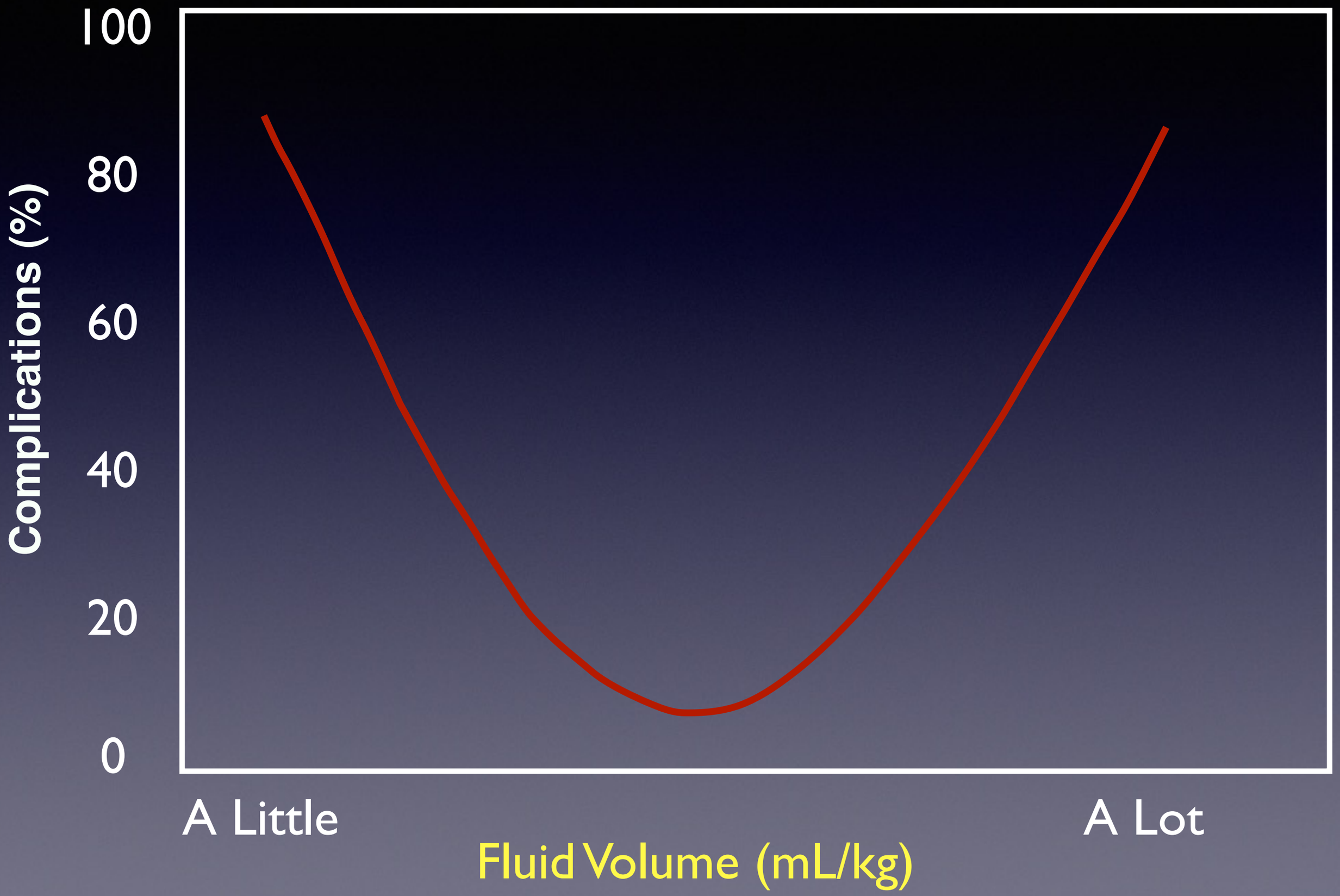
"Fluid Restriction"



"Fill that Third Space"



The True Picture ?



Consequences of too little fluid

Effects of compensated "hypovolaemia" on gut

6 healthy volunteers bled 2 x 600 ml

600 ml ->

No significant change in BP, HR, stroke volume

Decrease in gut intramucosal pH

1200 ml ->

No significant change in BP, HR

Decrease in stroke volume,

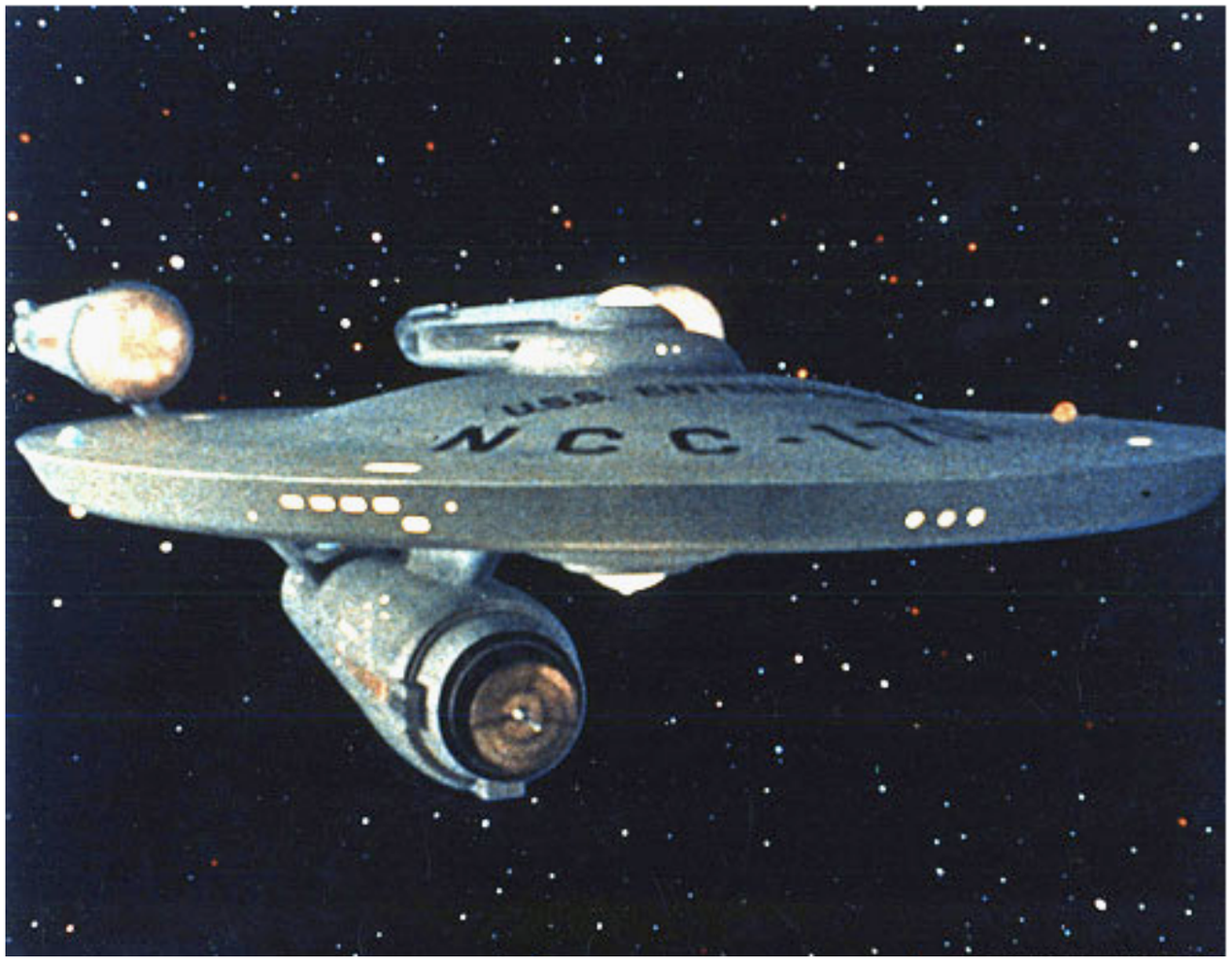
Gut intramucosal pH worsened

Re-transfusion -> variables recovered, but **'flu-like" symptoms**

ICM (1997) 23: 276–281

Adequate microcirculatory flow?

This seems to be the new “frontier”



Microvascular dysfunction

- 50 ICU patients resuscitated to adequate global haemodynamic endpoints
- After successful resuscitation, peripheral perfusion assessed:
  - Capillary refill, Core-peripheral temperature, Peripheral Flow Index
- Compared lactate levels, on-going organ failure

Crit Care Med 2009 Vol. 37, No. 3

Microvascular dysfunction

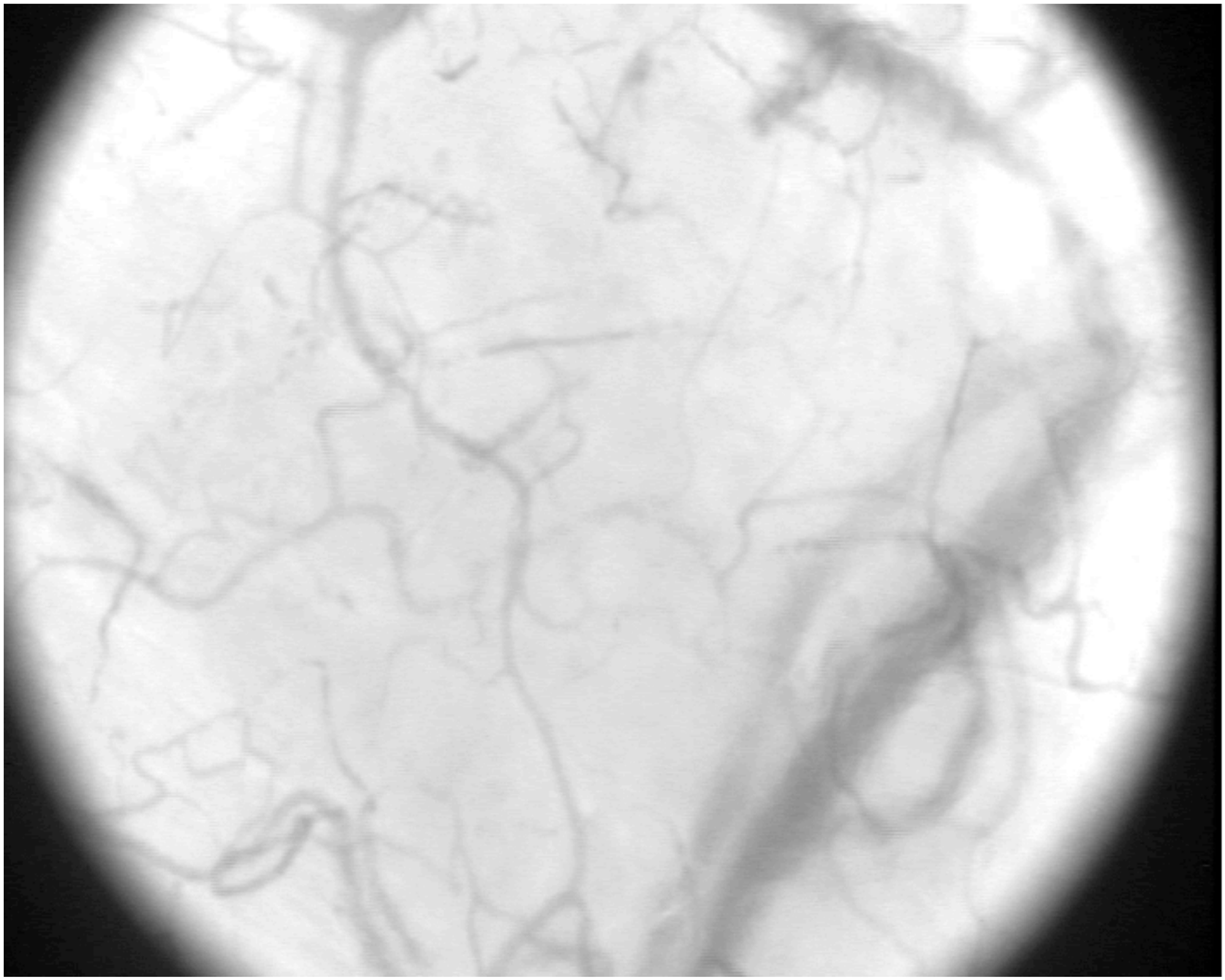
Peripheral perfusion <u>after</u> resuscitation

	Normal (27)	Abnormal (23)
HR	90	94
MAP	80	81
CVP	14	13
% Normal Lactate	69	31 **
△SOFA >0	23	77 **

Adequate global values with poor peripheral perfusion probably a sign of compensatory mechanisms still present.

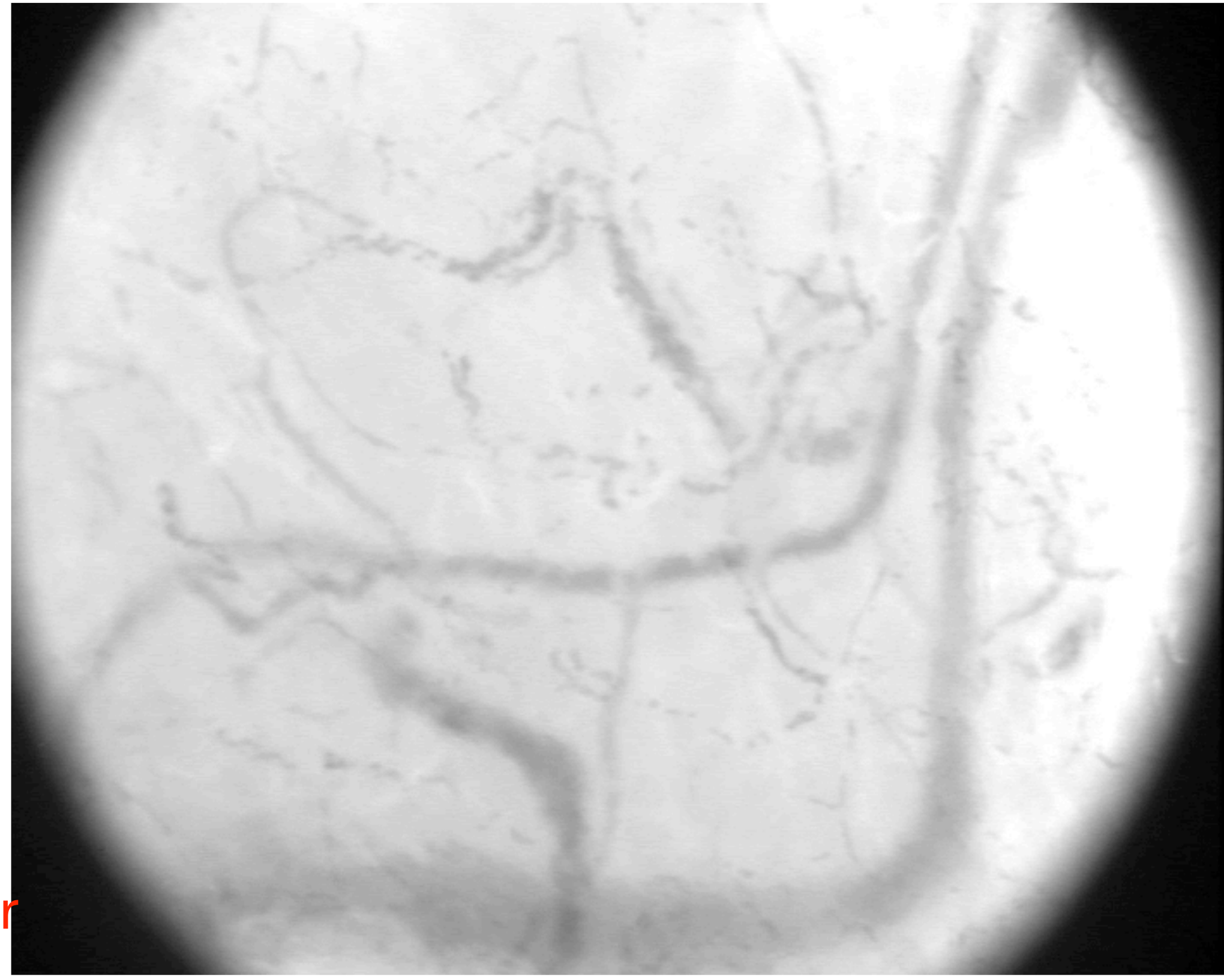
Crit Care Med 2009 Vol. 37, No. 3

Before Terlipressin

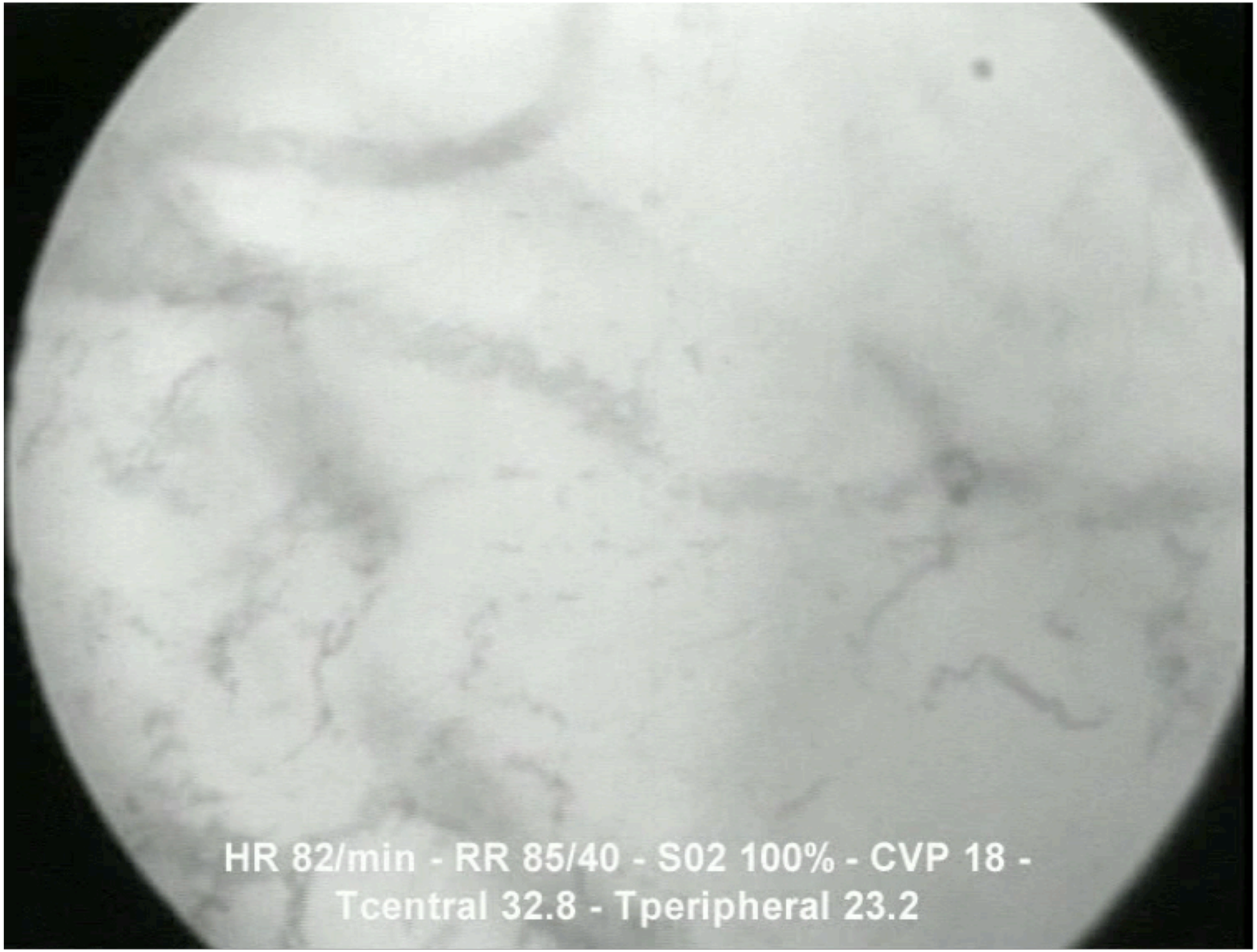


MAP 58
HR 98
CVP 13
UO 20 ml/hr

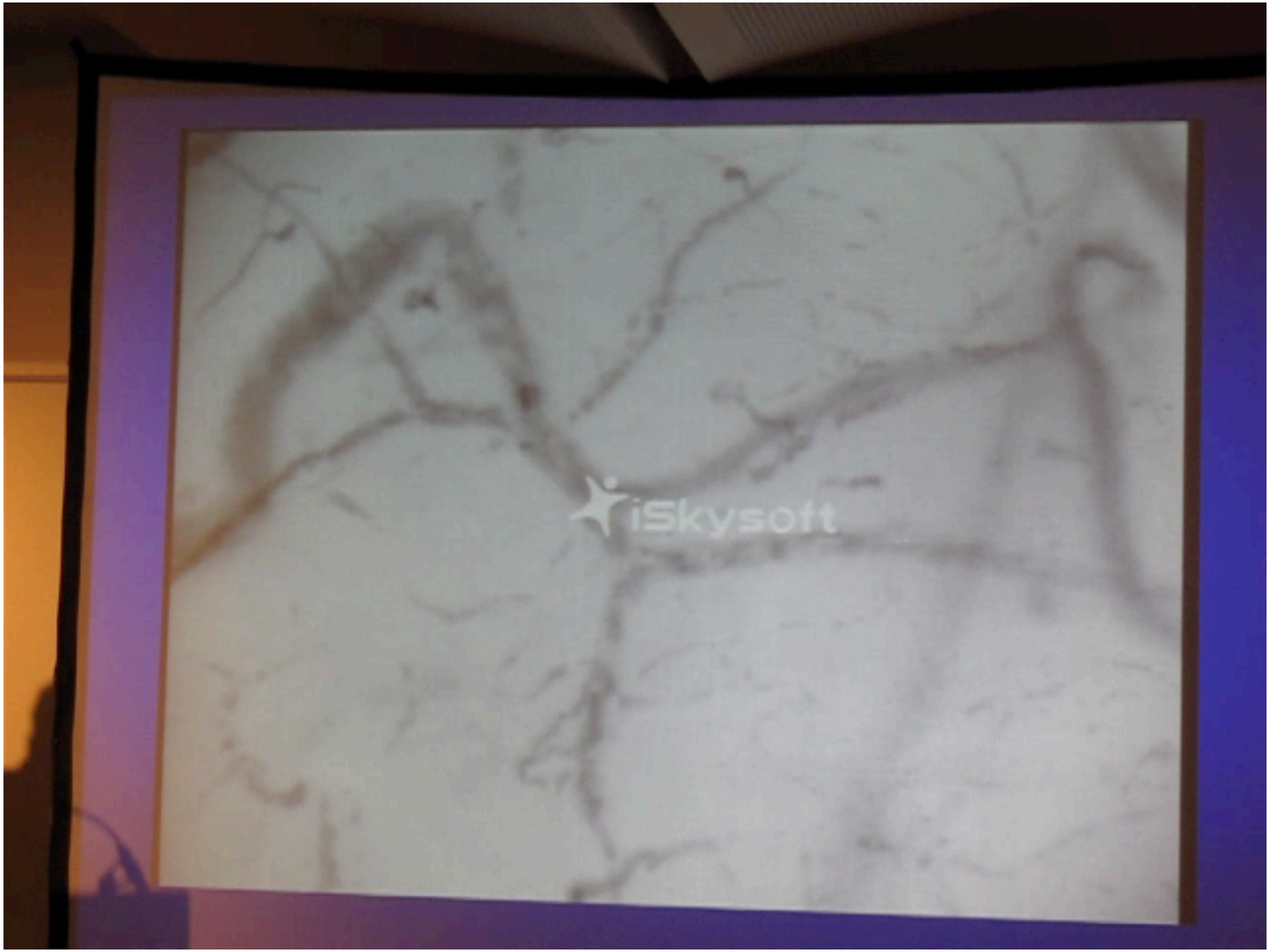
After Terlipressin



MAP 80
HR 98
CVP 12
UO 110 ml/hr


HR 82/min - RR 85/40 - S02 100% - CVP 18 -
Tcentral 32.8 - Tperipheral 23.2

Disseminated intravascular coagulation

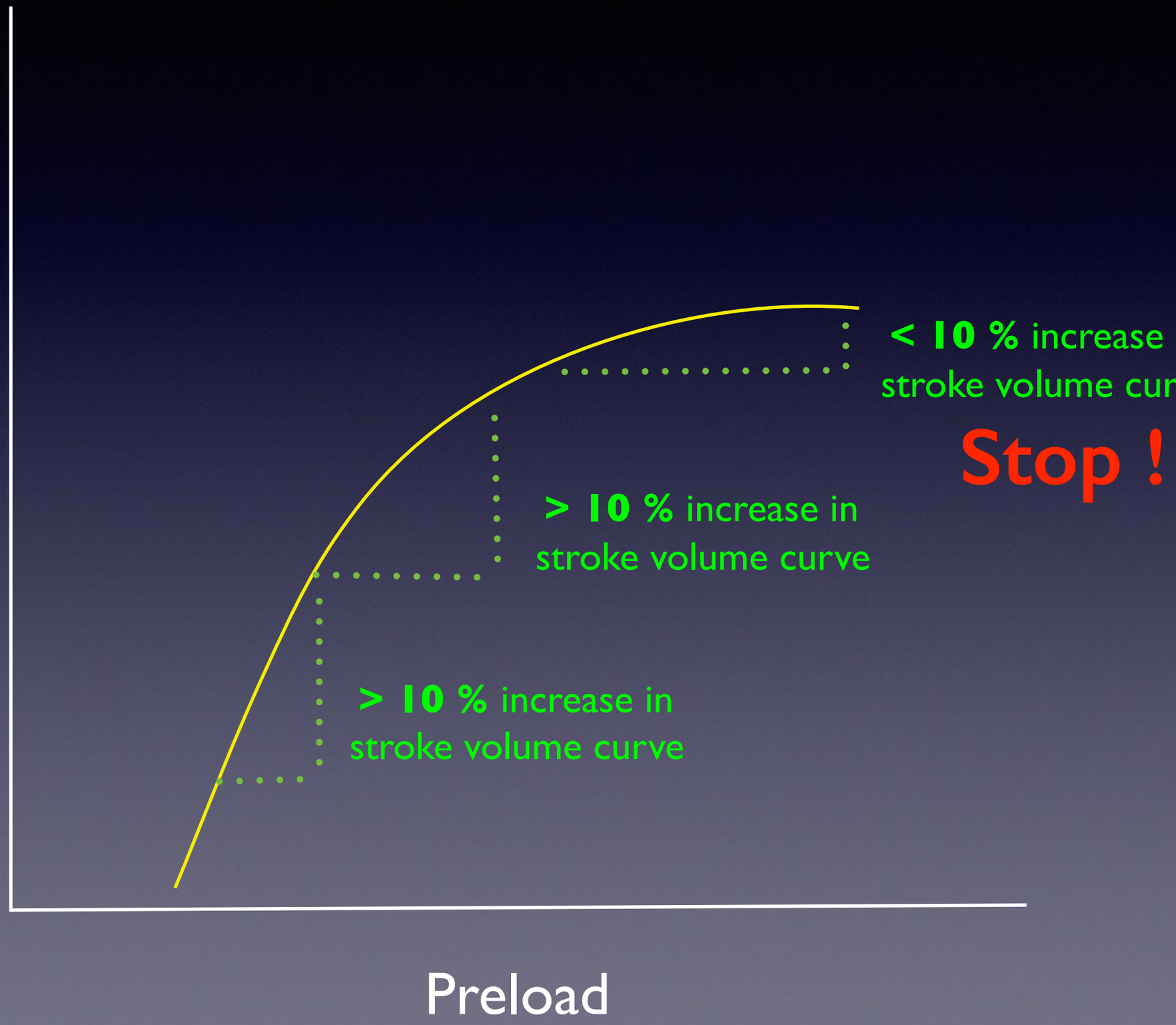


How do you fluid load ?

Give small volume (~ 250 ml) quickly

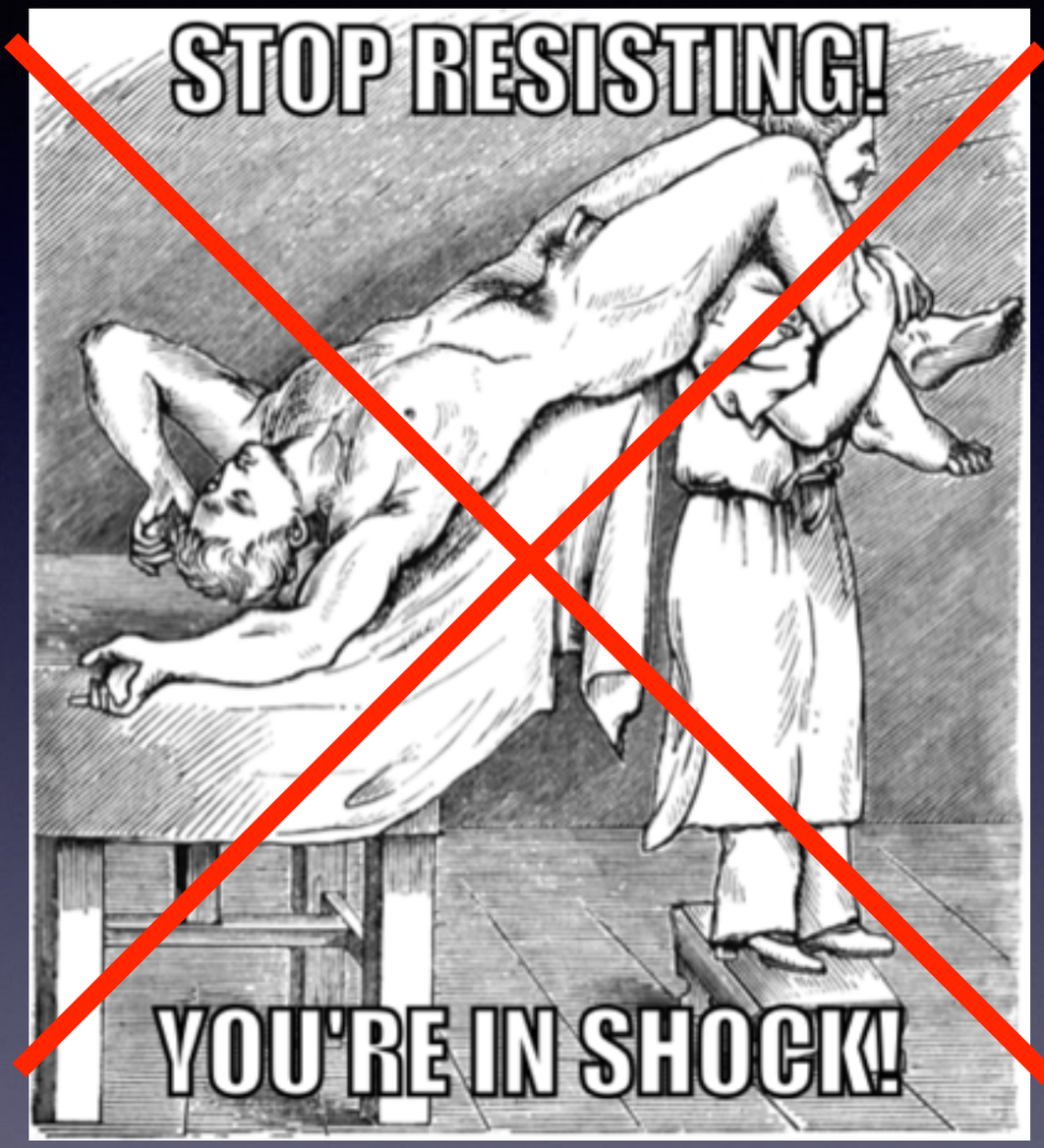
and measure response immediately

How do you fluid load ?



OR

Or raise ze legs!



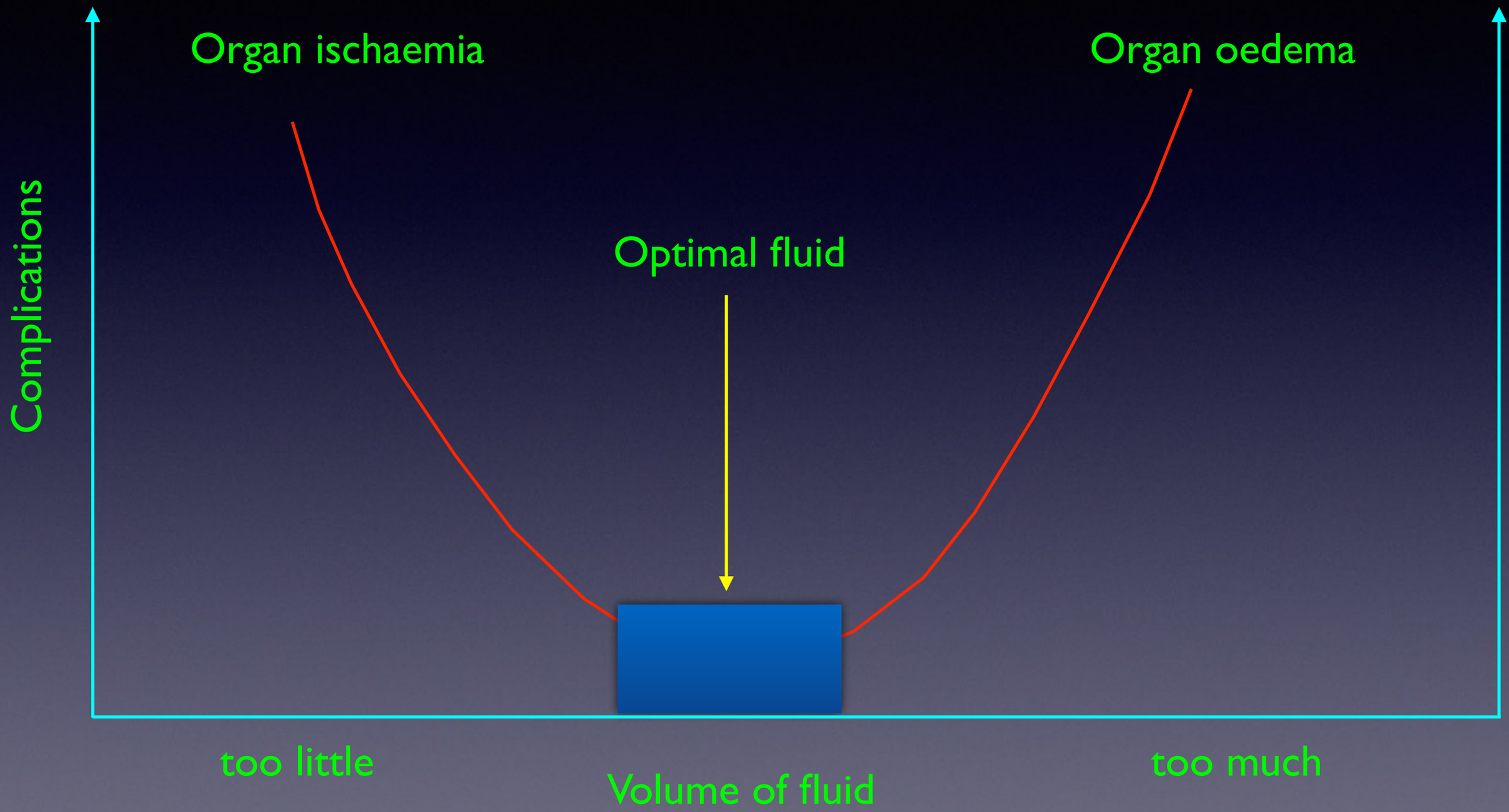
Rapidly "transfuses" ~ 500 mL

Not Trendelenburg

Intensive Care Med (2008) 34:659–663

Recap

Under- and over-resuscitation are associated with increased mortality

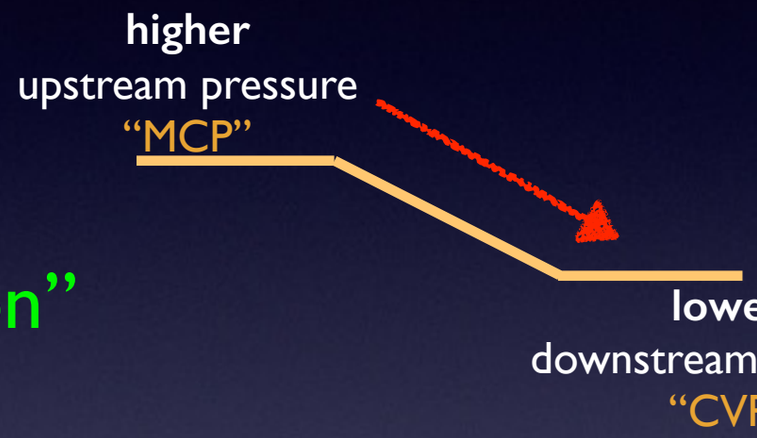


"The end-point of fluid resuscitation remains the Holy Grail of ITU medicine"

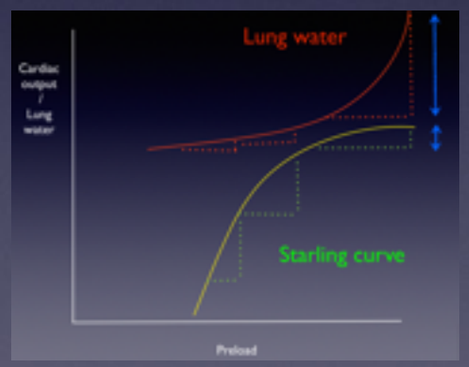
Crit Care Med 2012 Vol. 40, No. 6

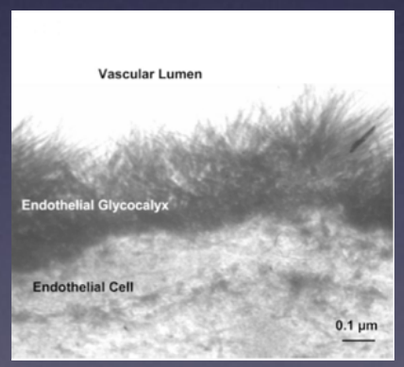
Recap

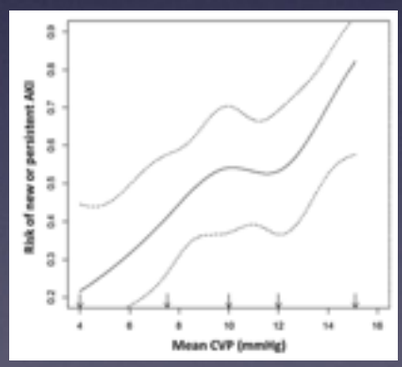
- Cardiac output the most important determinant of O2 delivery
- Venous return determined by <u>vascular factors</u>
  - stress vs unstressed volume → MCP



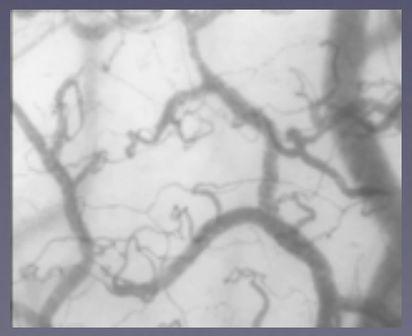
- "Fluid responsive or not? - that is the question"
- Don't guess
  - Too much fluid -







  - Too little fluid -



- Only after fluids optimised consider inotropes

???

www.jvsmedicscorner.com

(Mallory / Everest2013)

Acute Respiratory Distress Syndrome (ARDS)

Oxygen delivery ~

Cardiac output x Hb x % Sat O2

Cardiac output x Hb x % Sat O2

Preload

Effective blood volume
Capacitance
Obstruction
Septal shift
IV fluid
    volume
    C.O.P.

Pump failure

Arrhythmias
Ischaemia
Valvular problems
Septal shift

Afterload

RAA adaptaion
Sepsis
Valvular problems
Pulmonary embolism
Hypertension
Shunts

Heart rate

Anaemia

Fe def
Dilutional
Inflammatory
Vitamin deficiency
Aplastic

Abnormal Hb

Sickle cell
Thalassaemia
met Hb
CO Hb

Hemolysis

free Hb and NO
Pulmonary hypertension
Hypercoagulability

Hyperviscosity

PRV
Acclimatisation

Inspired O2

Altitude
Hyperbaric O2

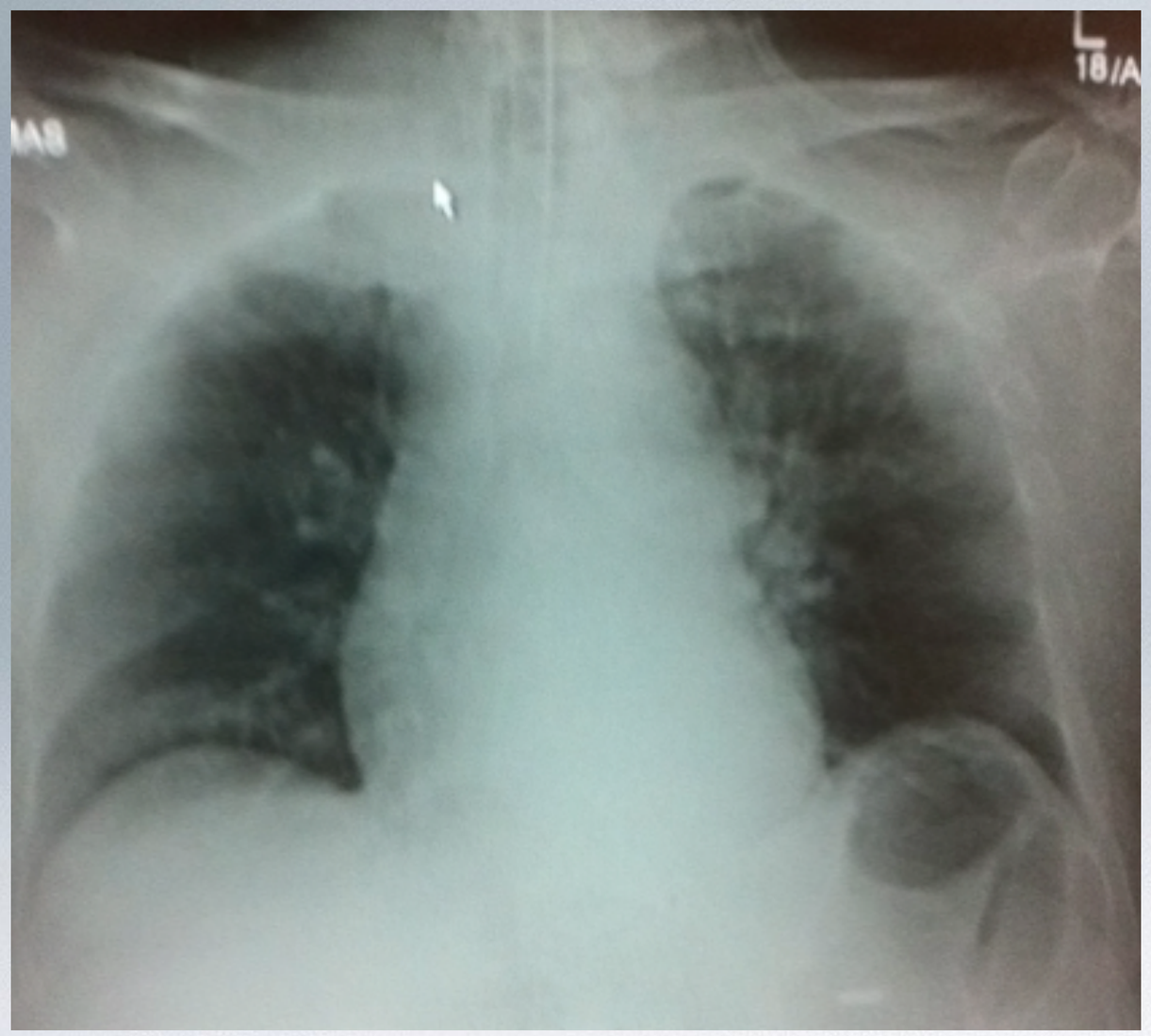
Hypoventilation

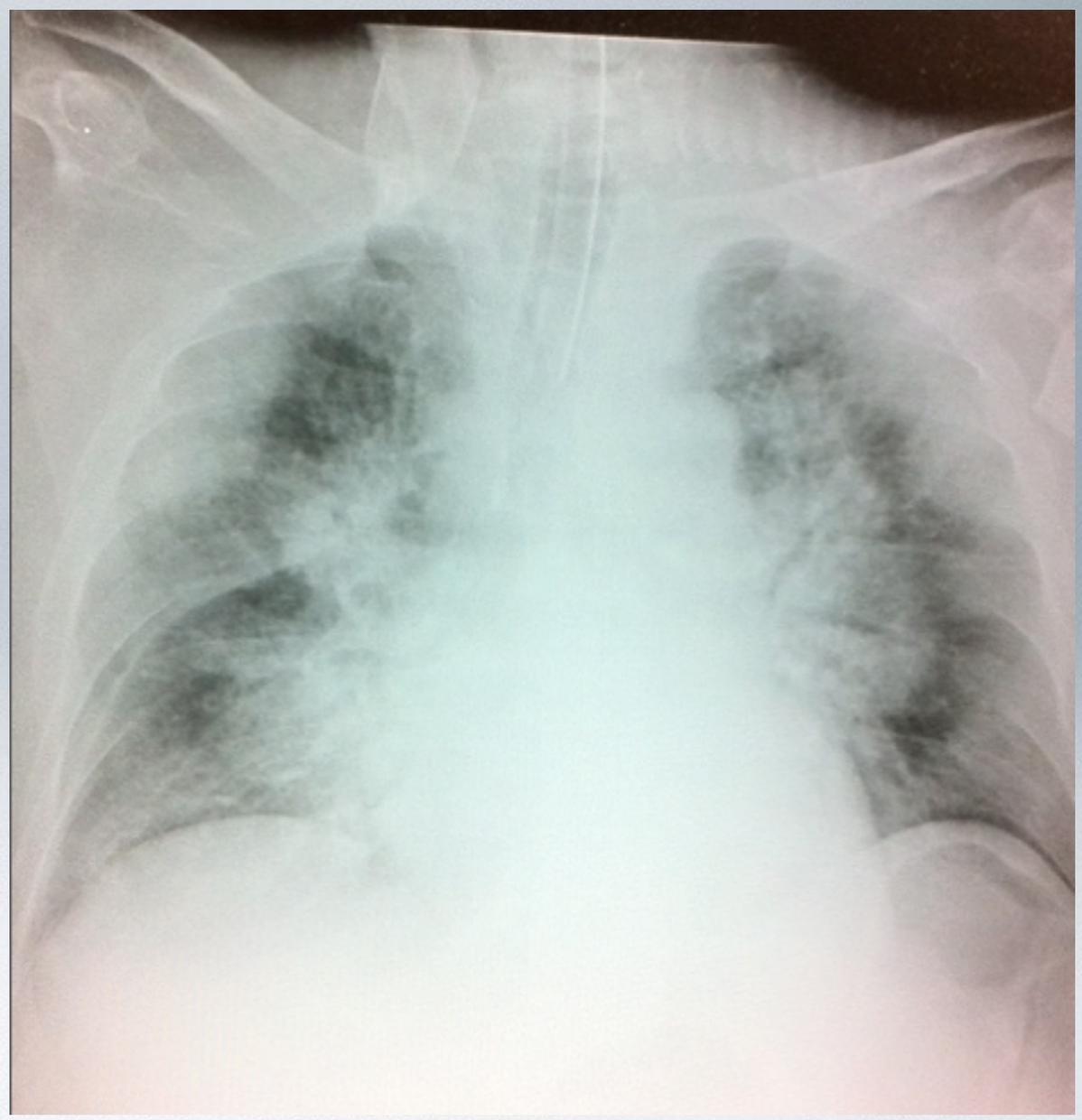
Decreased respiratory drive
    drug induced
CVA
Fatigue (asthma)
Obstruction
    Sleep apnoea syndrome
    Decreased consciousness

Ventilation/perfusion abnormalities

Shunt
    Pneumonia
    Pulmonary oedema
Dead space
    Pulmonary embolism
    Fat embolism
Mixed
    ARDS
    COPD
    Asthma

A patient admitted with H1N1 pneumonitis (“Swine flu”)



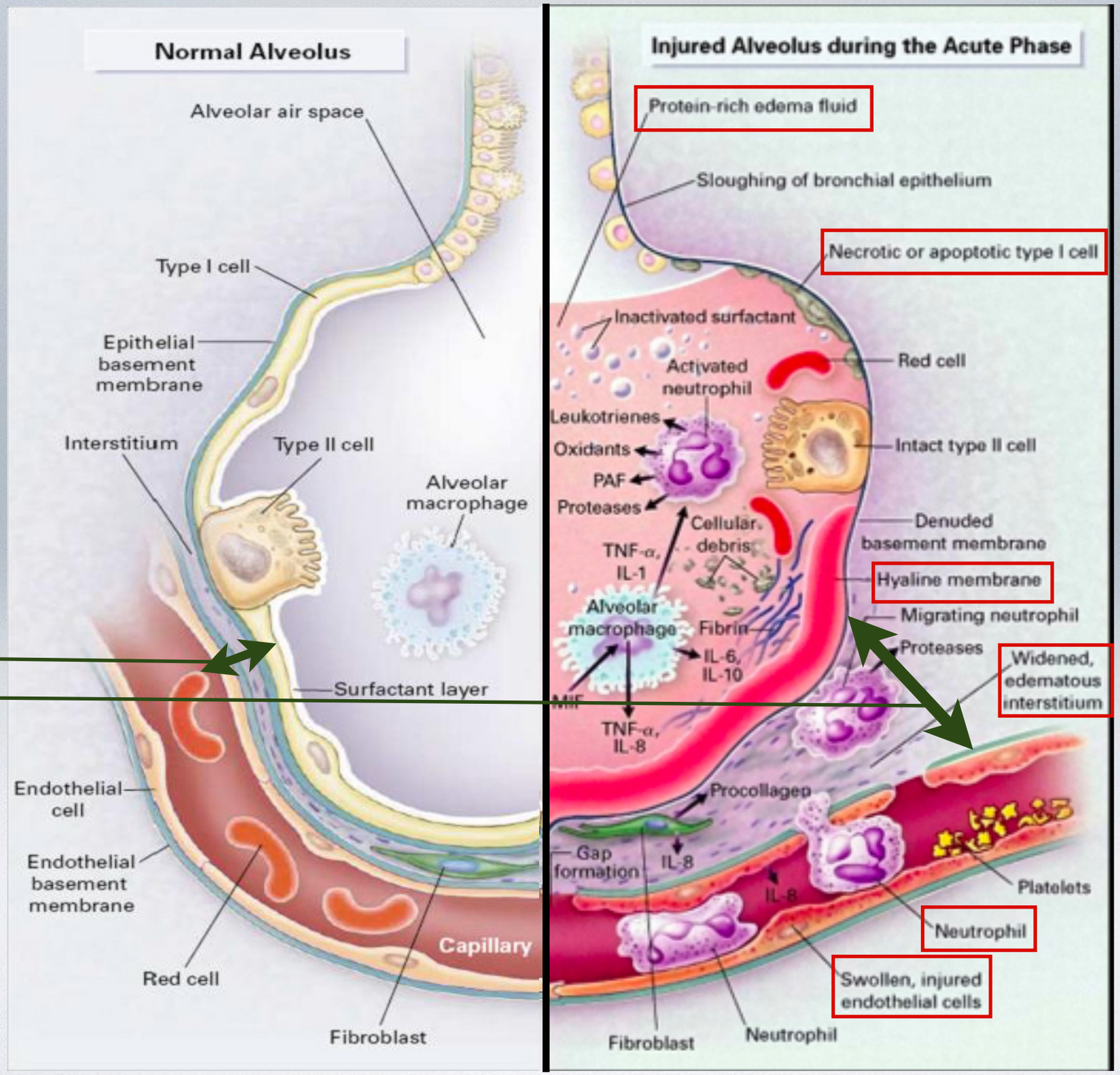


24 hours later

Histopathology of ARDS

ARDS alveolus

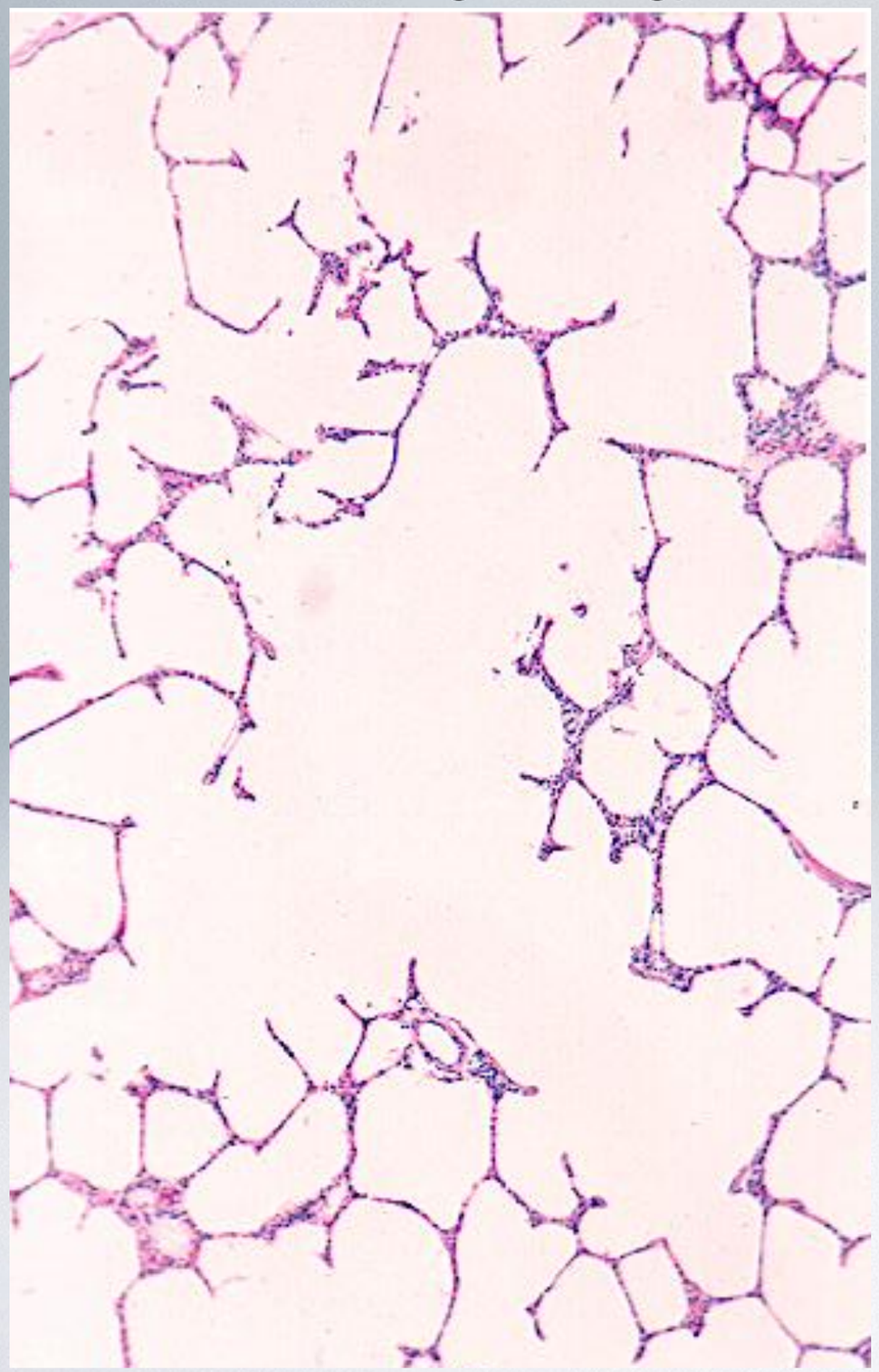
Normally respiratory membrane thickness = 0.2-0.3 micron



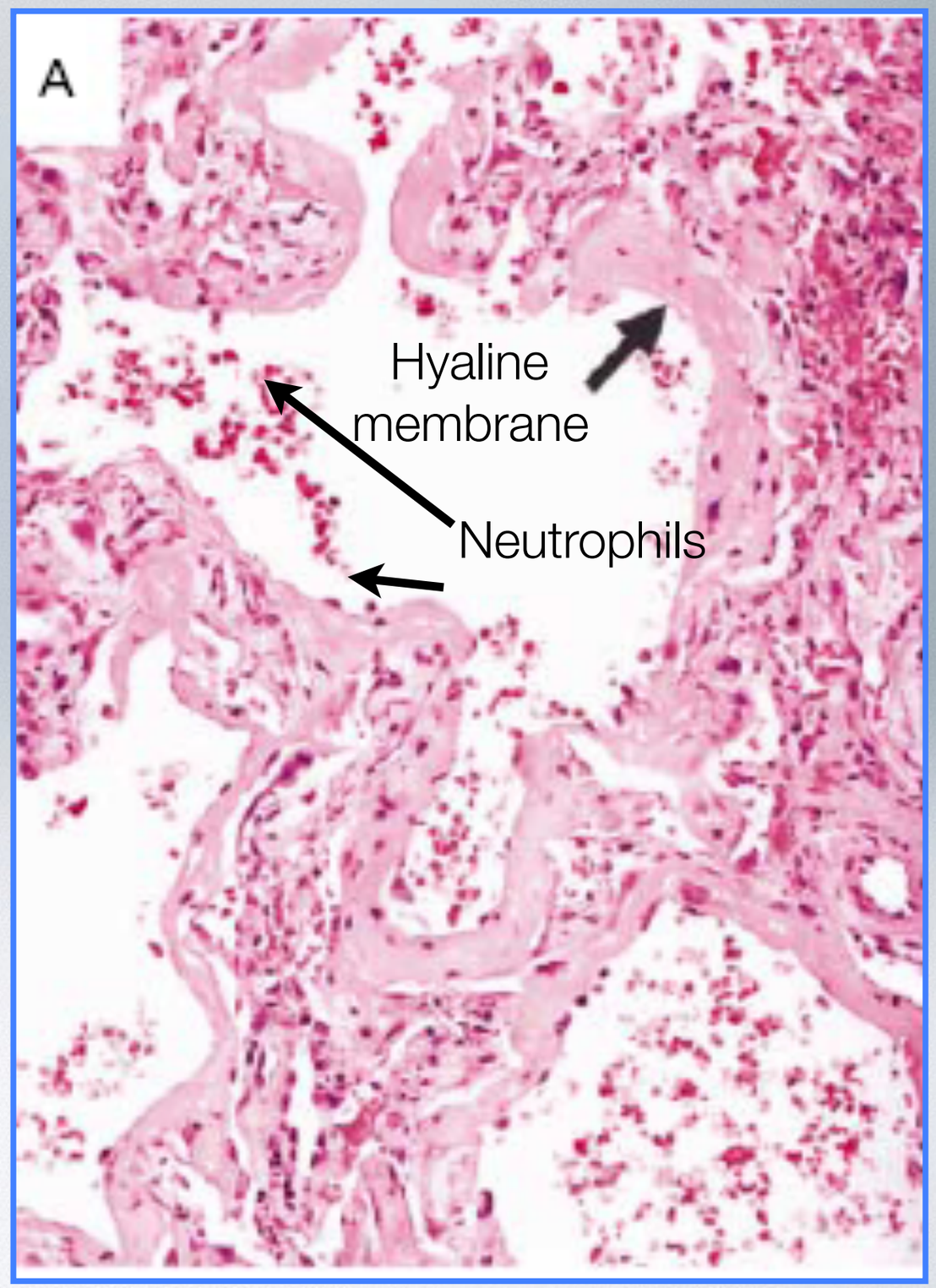
Ware LB, Matthay MA. The acute respiratory distress syndrome. NEJM. 2000;342:1334

ARDS Histology

Normal lung histology



Destruction of lung/alveolar architecture

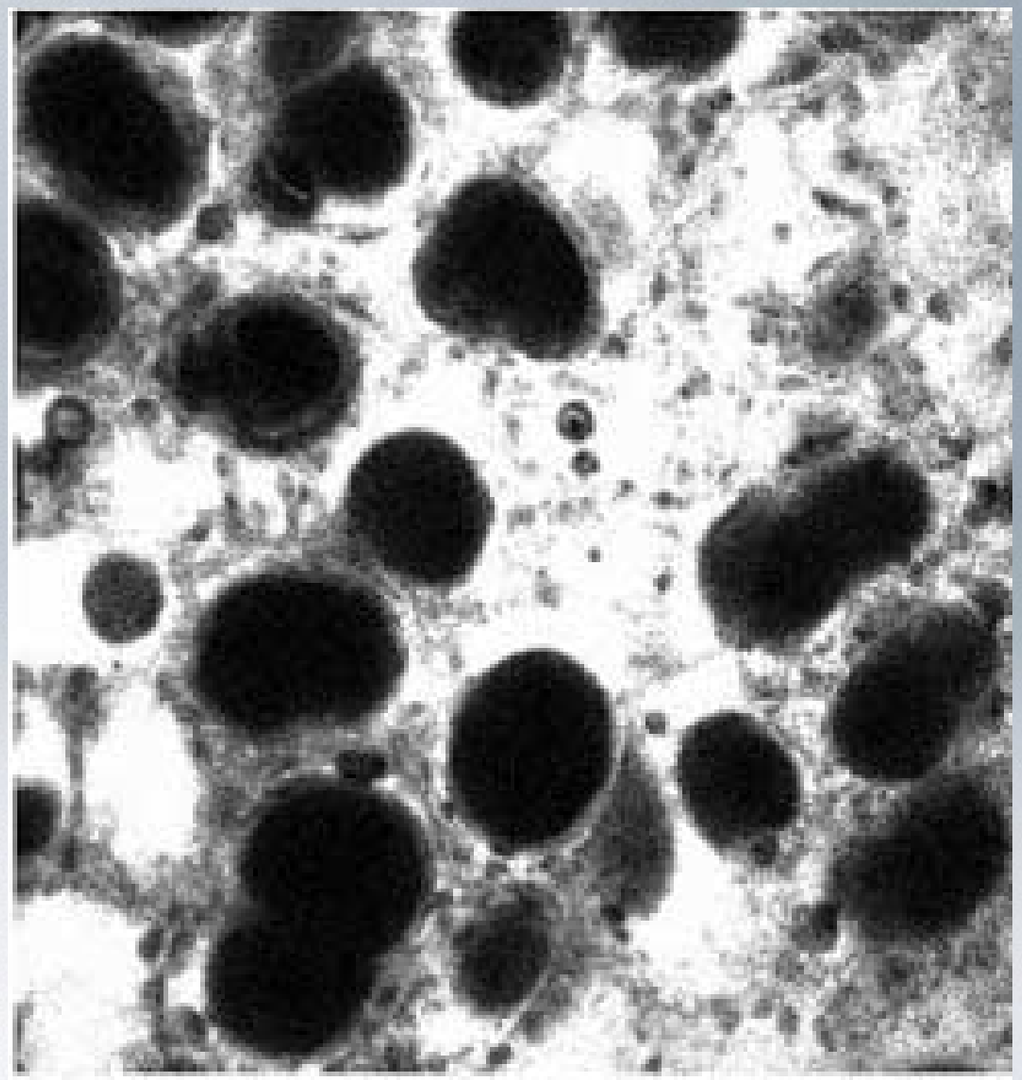


Ware LB, Matthay MA. The acute respiratory distress syndrome. NEJM. 2000;342:1334

ARDS Histology

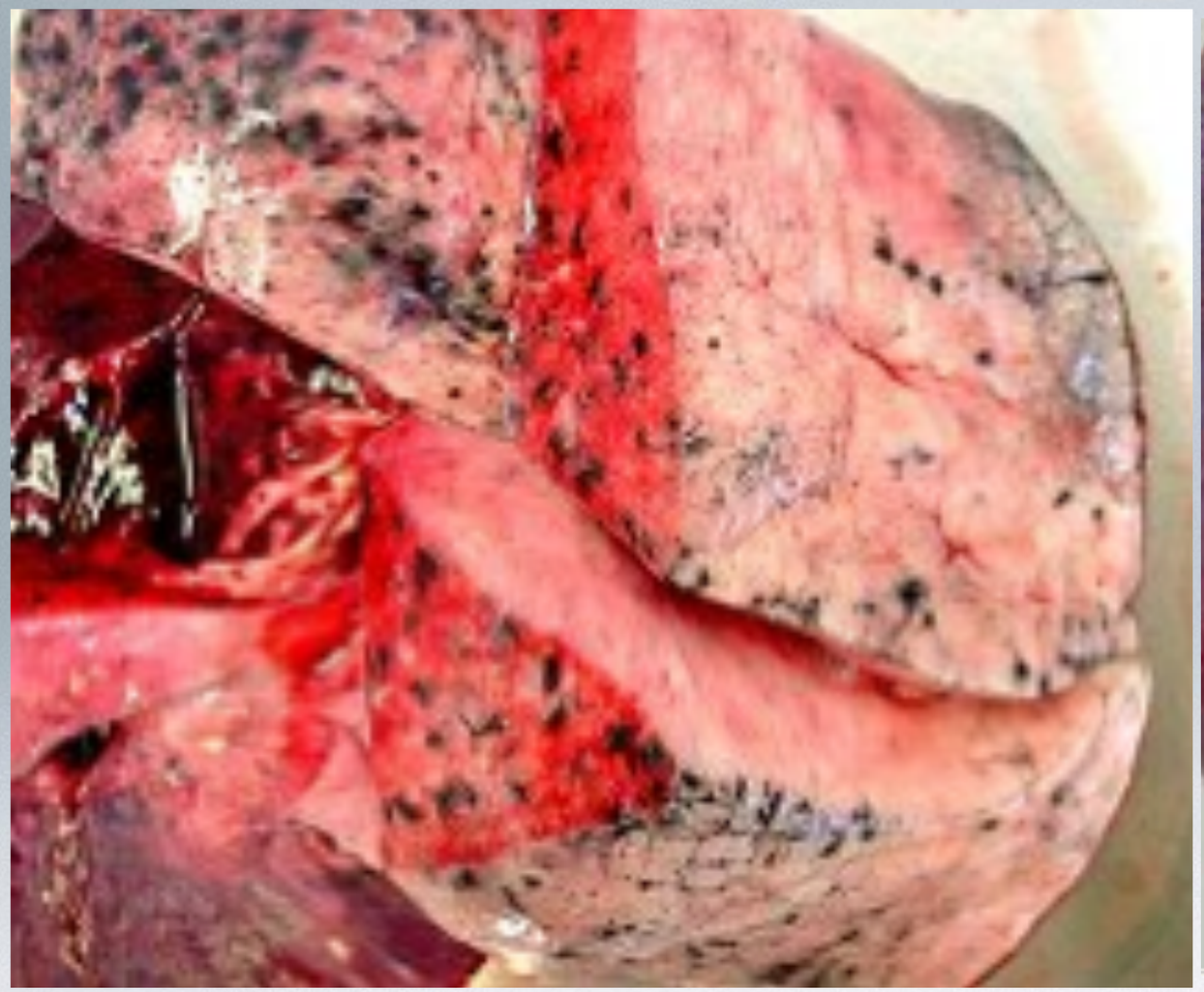
Normal

Oedematous



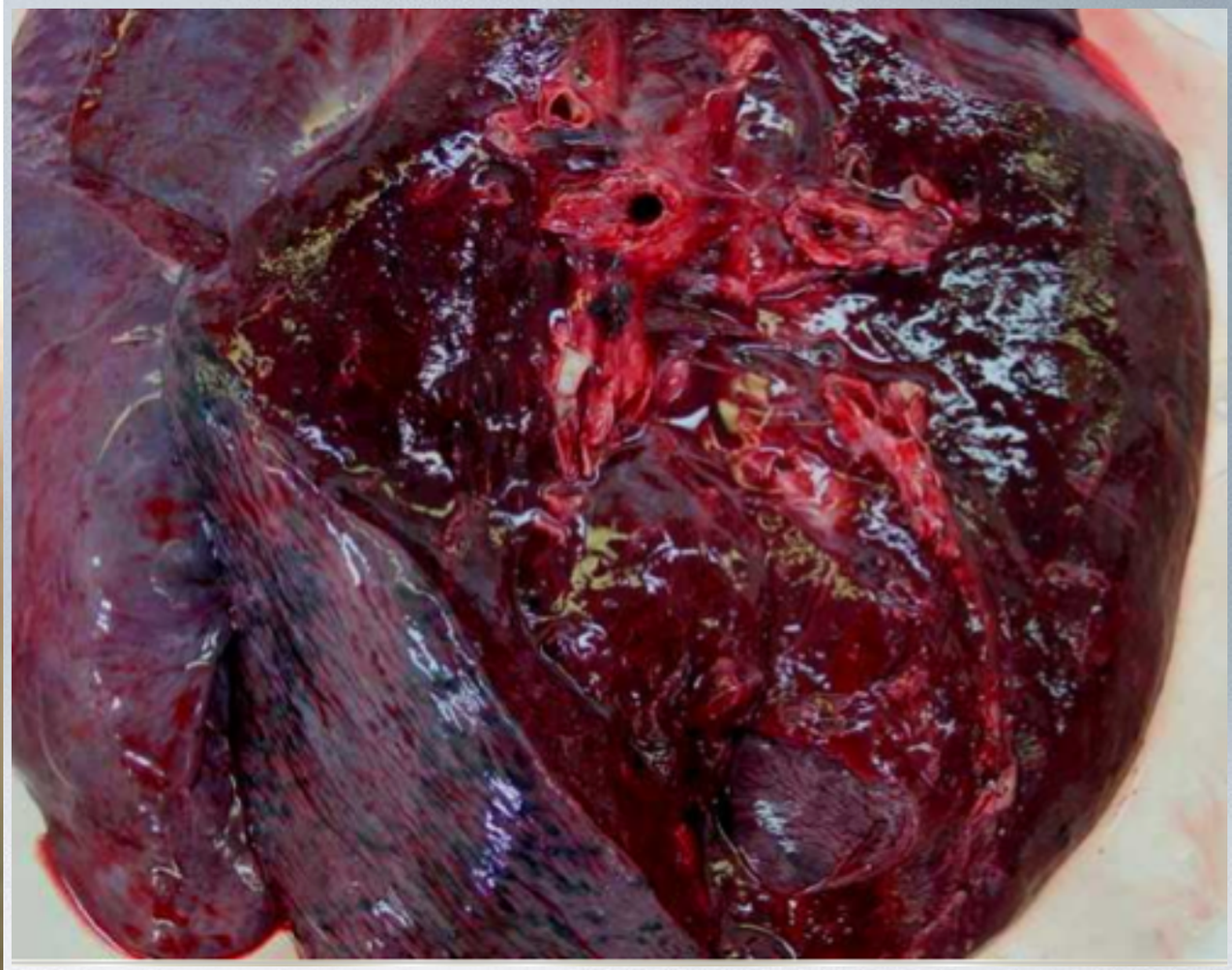
Am J Respir Crit Care Med Vol 165. pp 1647–1653, 2002

ARDS Pathology



Normal lung

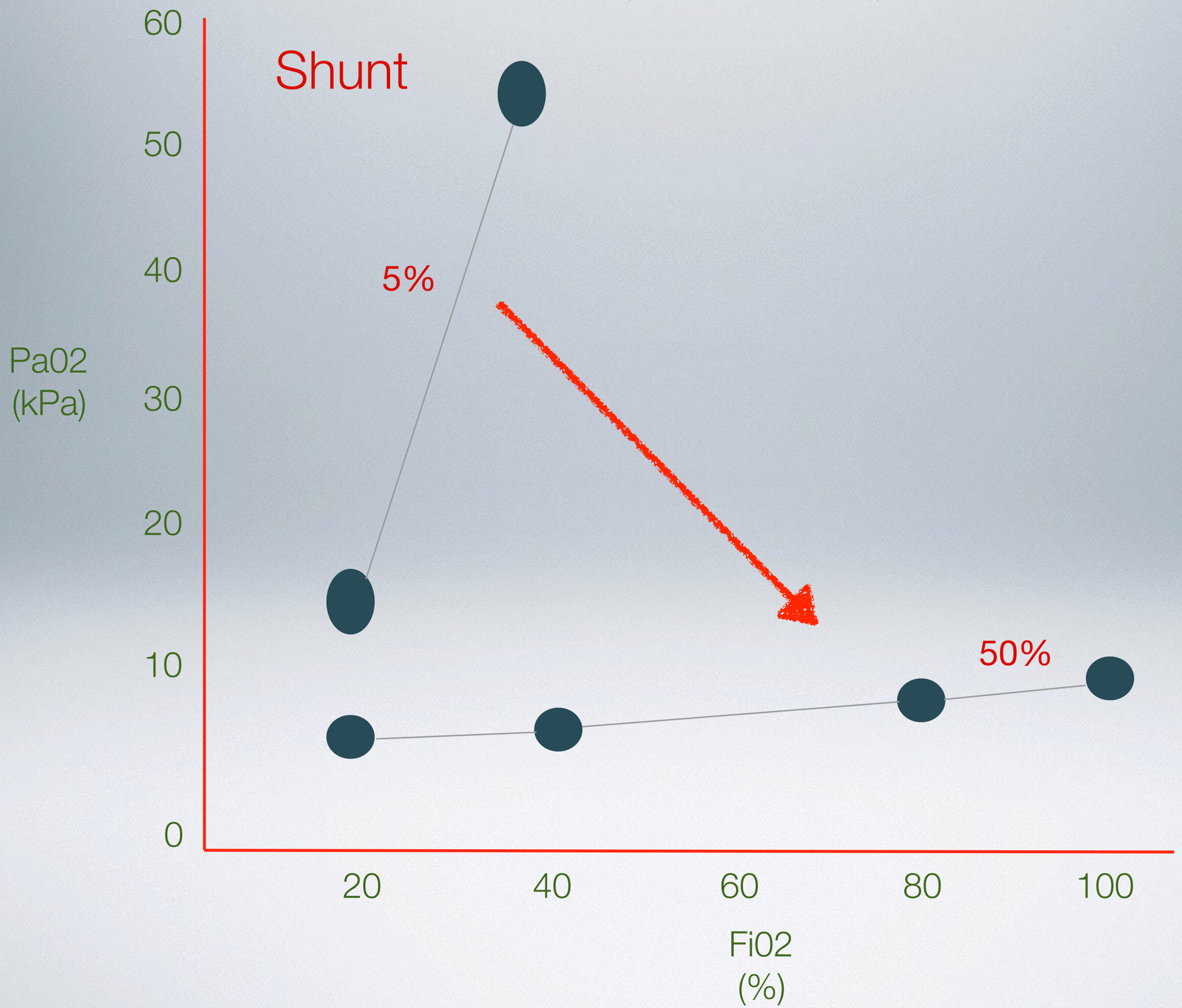
weight 800 gms



ARDS lung

weight 1600 gms

Physiological derangement



Goals of treatment

- **Treat primary condition**
- **<u>Avoid further harm:</u>**
  - Volutrauma
  - Barotrauma
  - Biotrauma
  - Recruitment/de-recruitment
  - Fluid overload

Only really effective treatment is to **avoid further harm !!**

Clinical Case

49 yr old female

Acute respiratory distress following H1N1 flu

Ventilated for 8 days with high Fi02

Tidal volumes ~ 500 mL

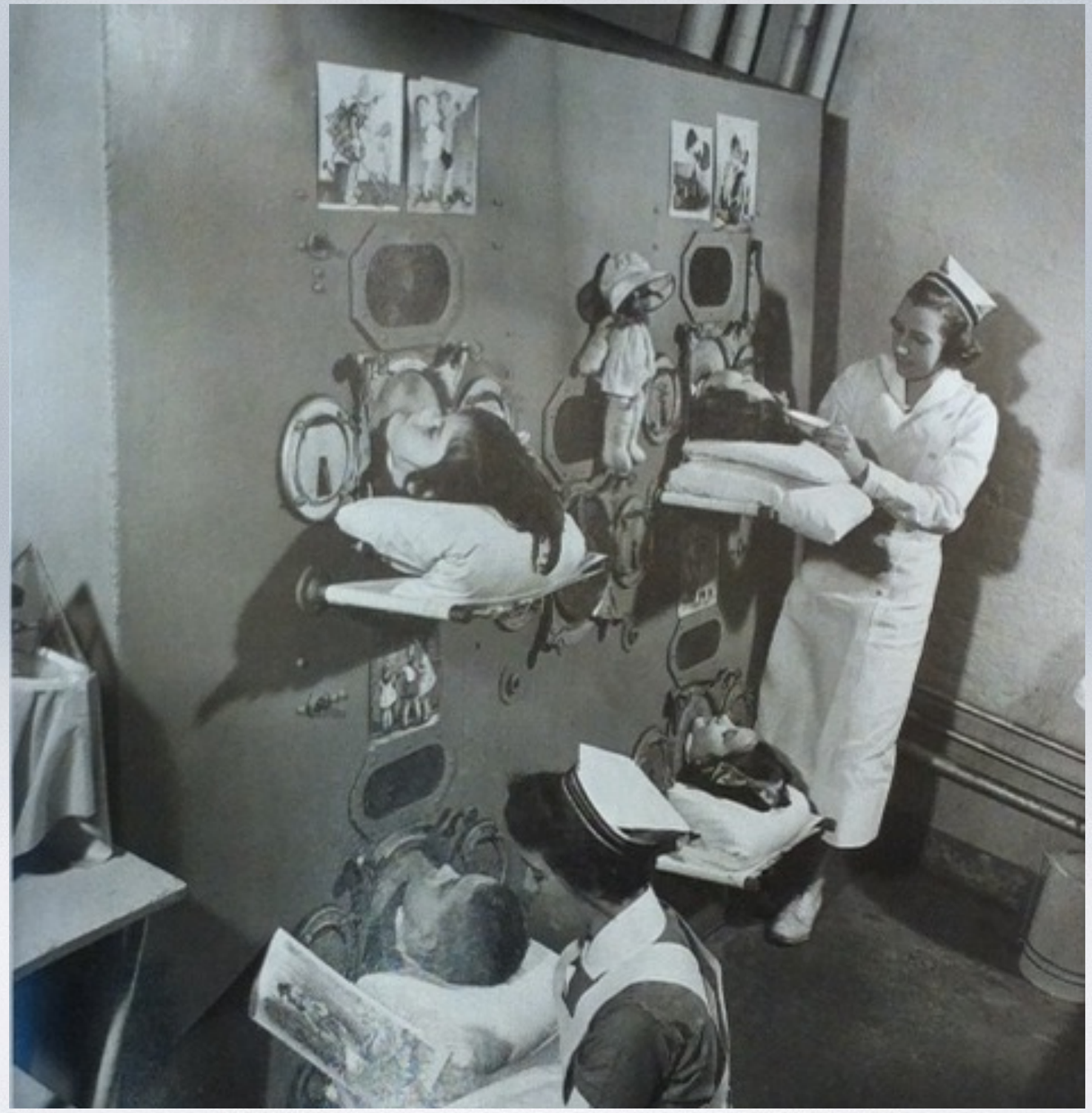
No improvement in deteriorating 02 sats

Called Leicester ECMO center for transfer

Refused!

Why?

Ventilator Induced Lung Injury-“VILI”



Vincent *et al. Critical Care* 2010, **14**:311
http://ccforum.com/content/14/3/311

CRITICAL CARE

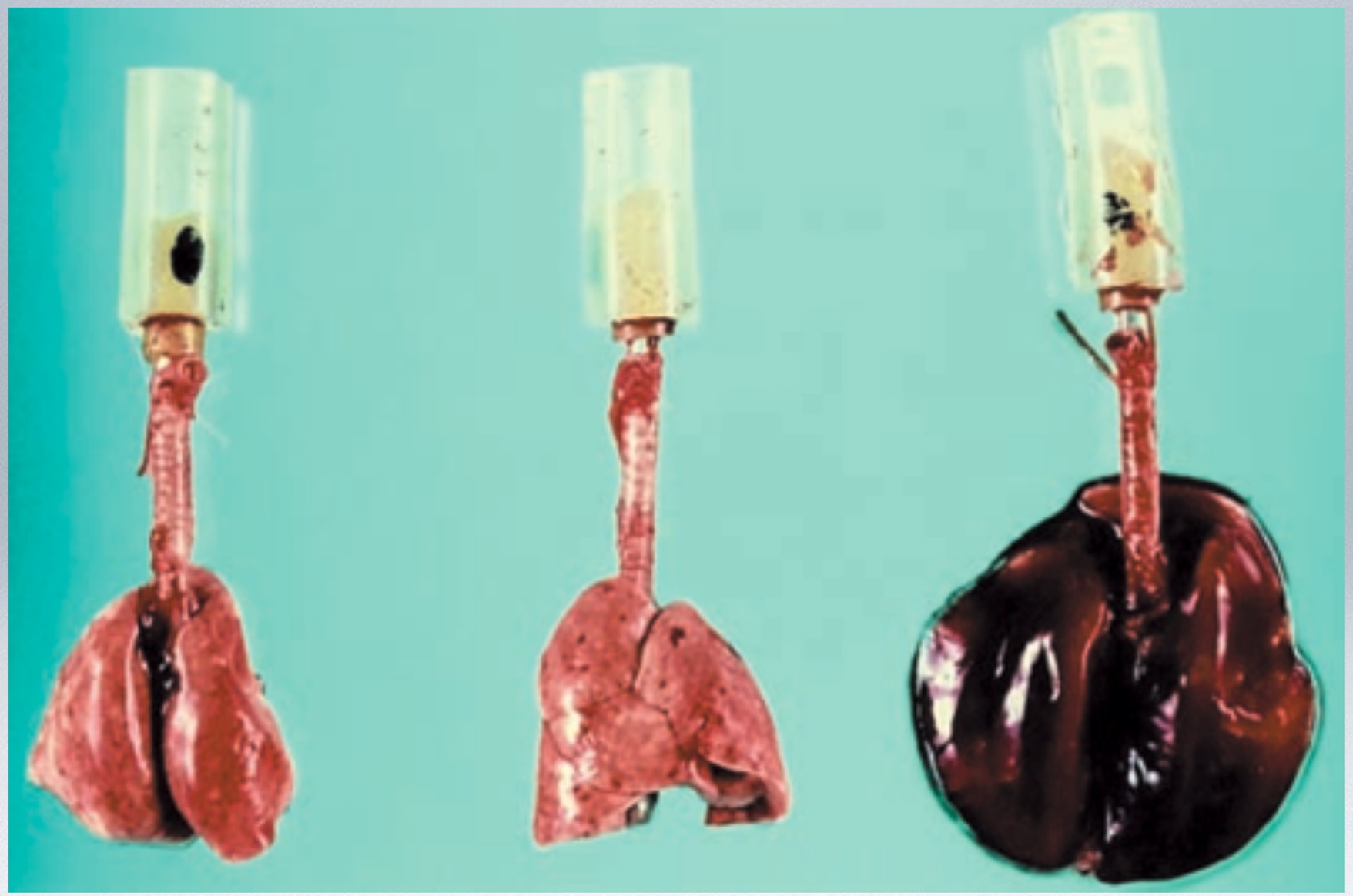
VIEWPOINT

Thirty years of critical care medicine

Jean-Louis Vincent*[1], Mervyn Singer[2], John J Marini[3], Rui Moreno[4], Mitchell Levy[5], Michael A Matthay[6], Michael Pinsky[7], Andrew Rhodes[8], Niall D Ferguson[9], Timothy Evans[10], Djillali Annane[11] and Jesse B Hall[12]

"...we have made major progress …. over the past 30 years through the recognition and avoidance of iatrogenic ventilator-induced lung injury (VILI) by **limiting tidal volumes** and airway pressures."

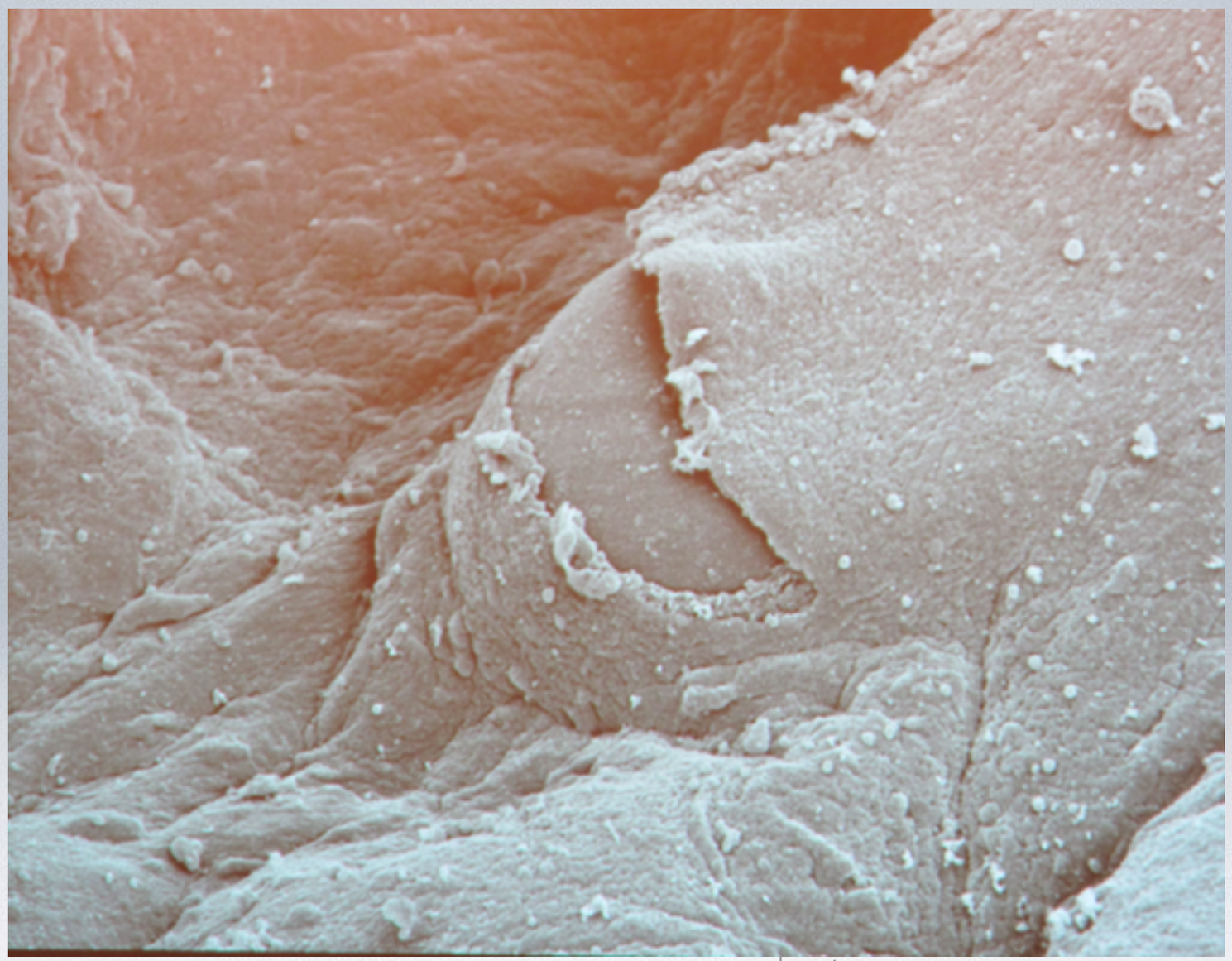
Avoid over-stretch of lungs



Control 5 min 20 min

PIP of 45 cm H20

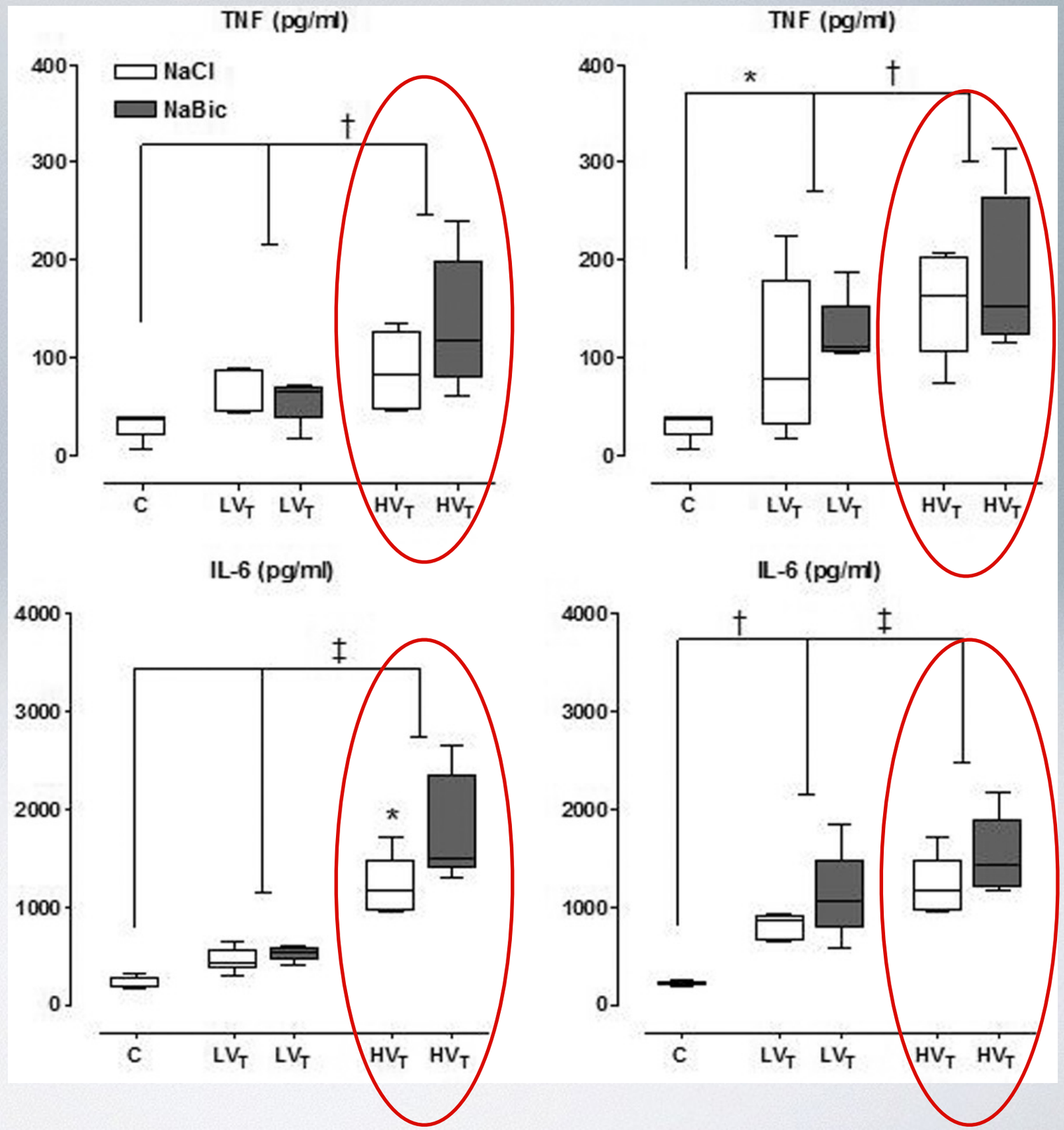
Endothelium and epithelium are injured at high lung volumes and pressures



Fu Z et al J Appl Physiol 1992 73: 123-133

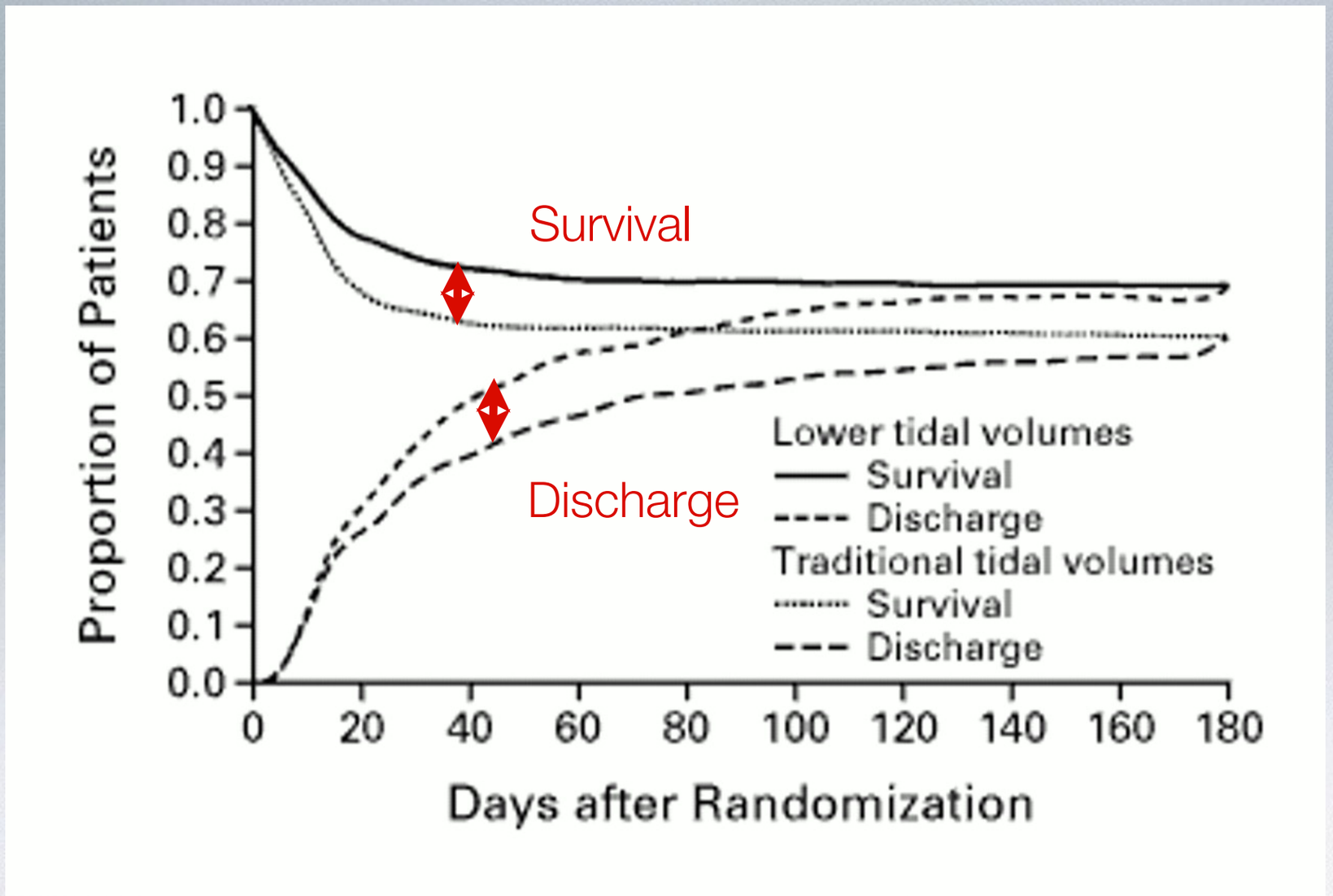
Systemic effects of volutrauma

High tidal volumes associated with increased release of cytokines



Critical Care 2009, 13:R1

Low vs high tidal volumes in ARDS/ALI



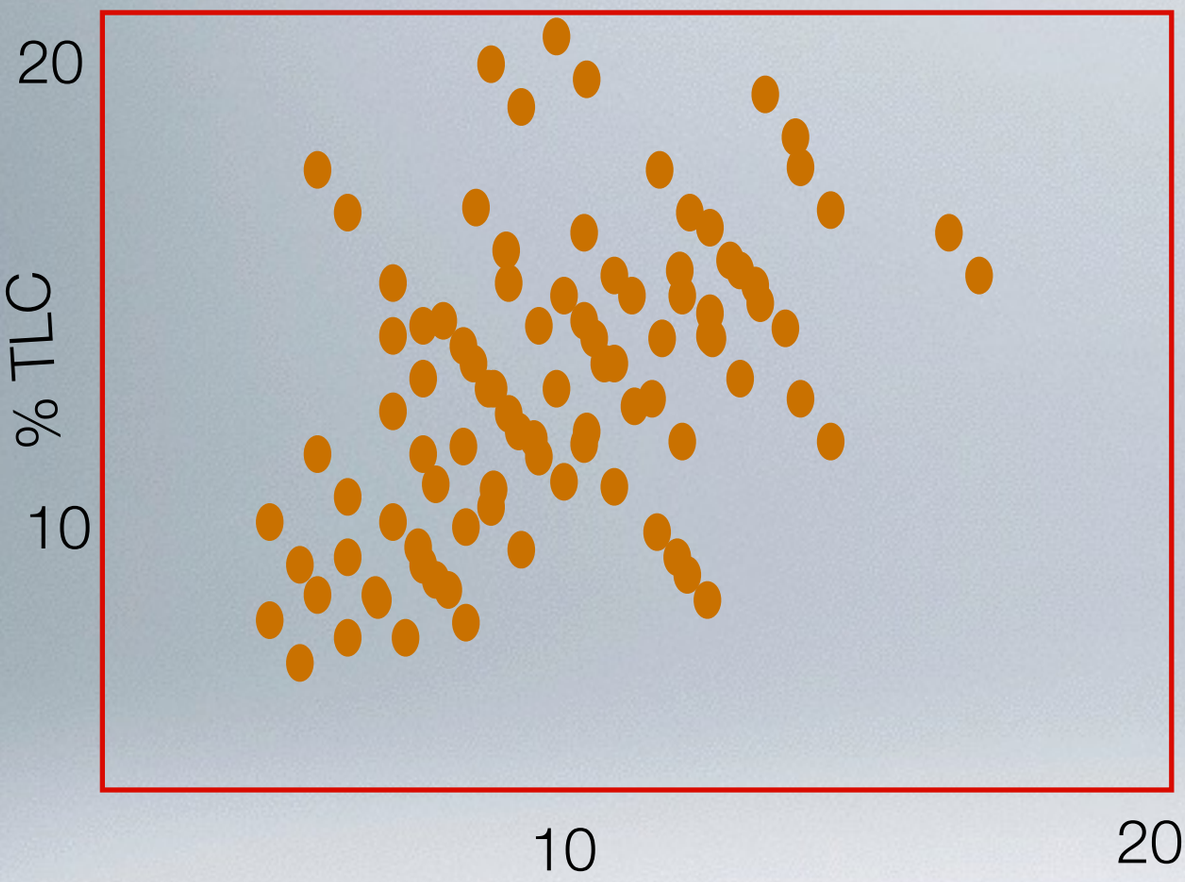
Mortality

12 ml/kg pbw
40%

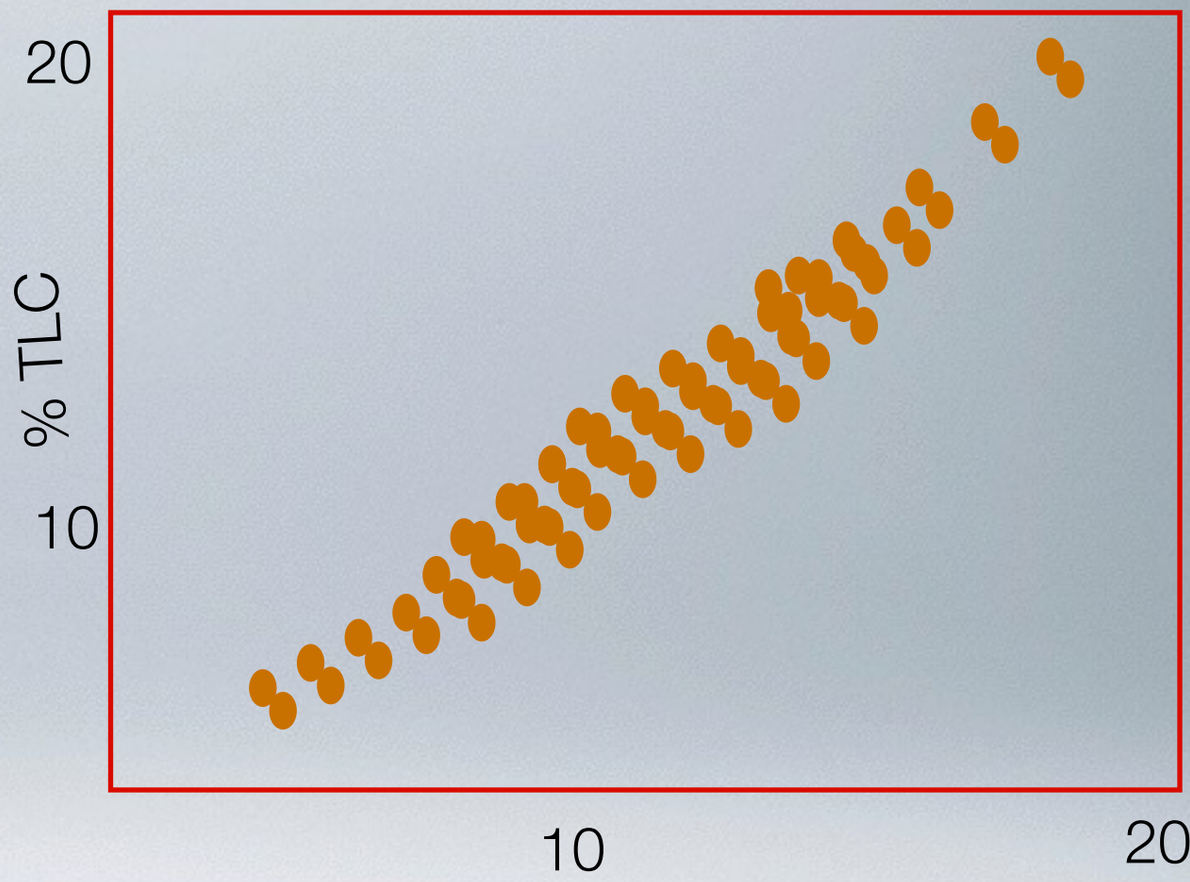
6 ml/kg pbw
30%

NEJM 2000;342:1301-1308

How do you best predict lung volume ?



Vt mL/kg **actual** body weight



Vt mL/kg **predicted** body weight

The correlation between **actual** body **weight** and %Total Lung Capacity is extremely **poor**

Crit Care Med 2004 Vol. 32, No. 9

Don’t forget protective tidal volumes are based on **ideal** (or predicted) body weight, which are based on **SEX** and **HEIGHT** (NOT weight!!)

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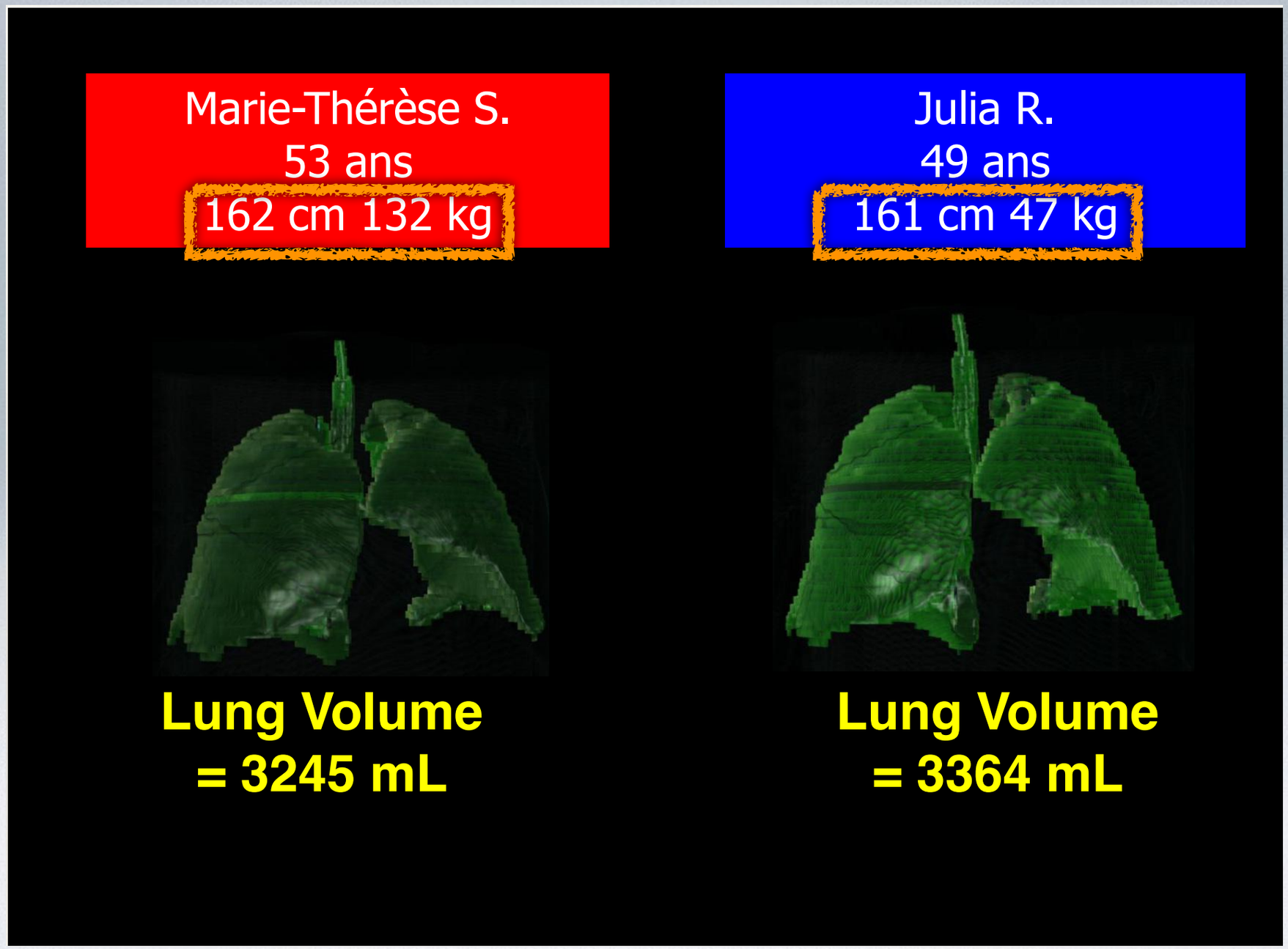
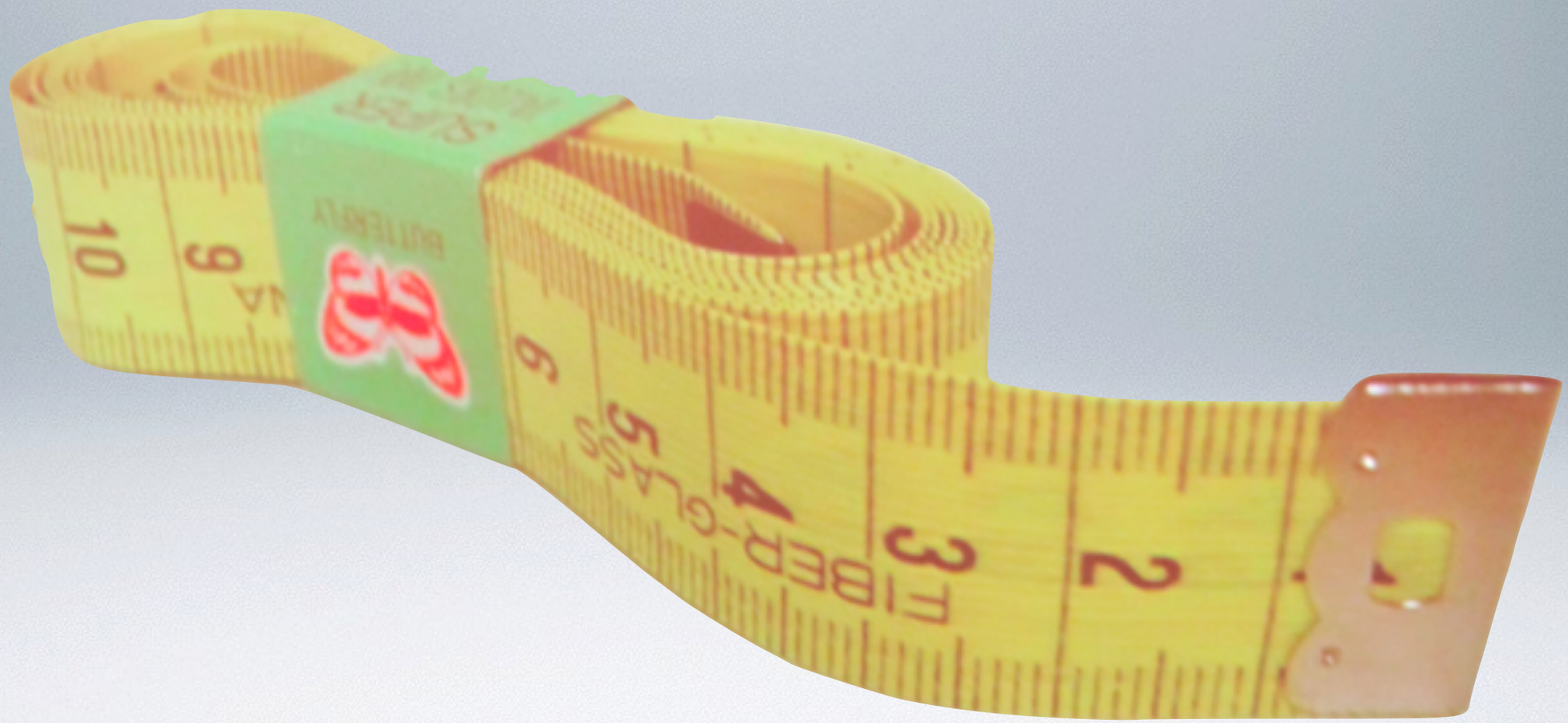


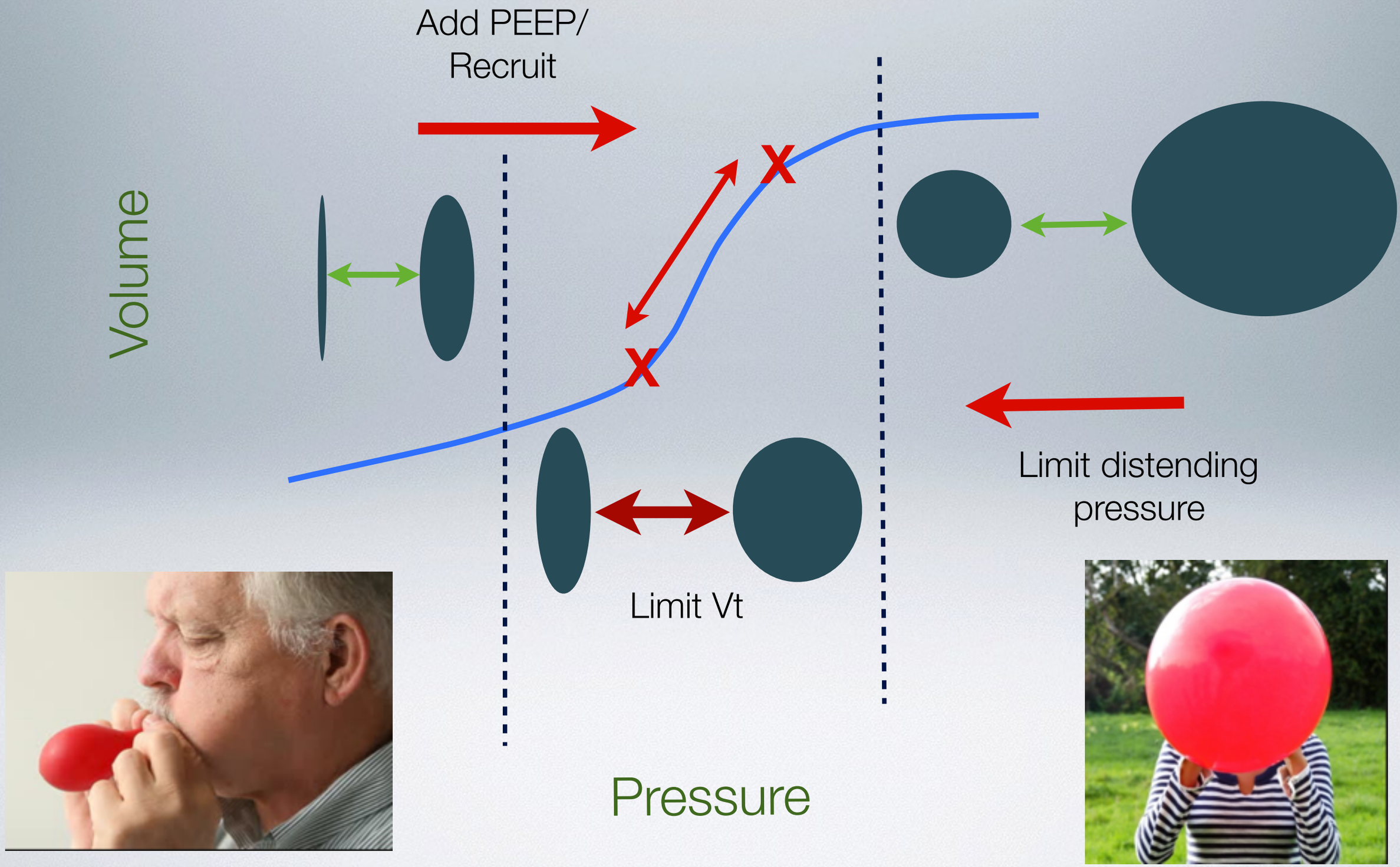
So don’t forget the tape measure!



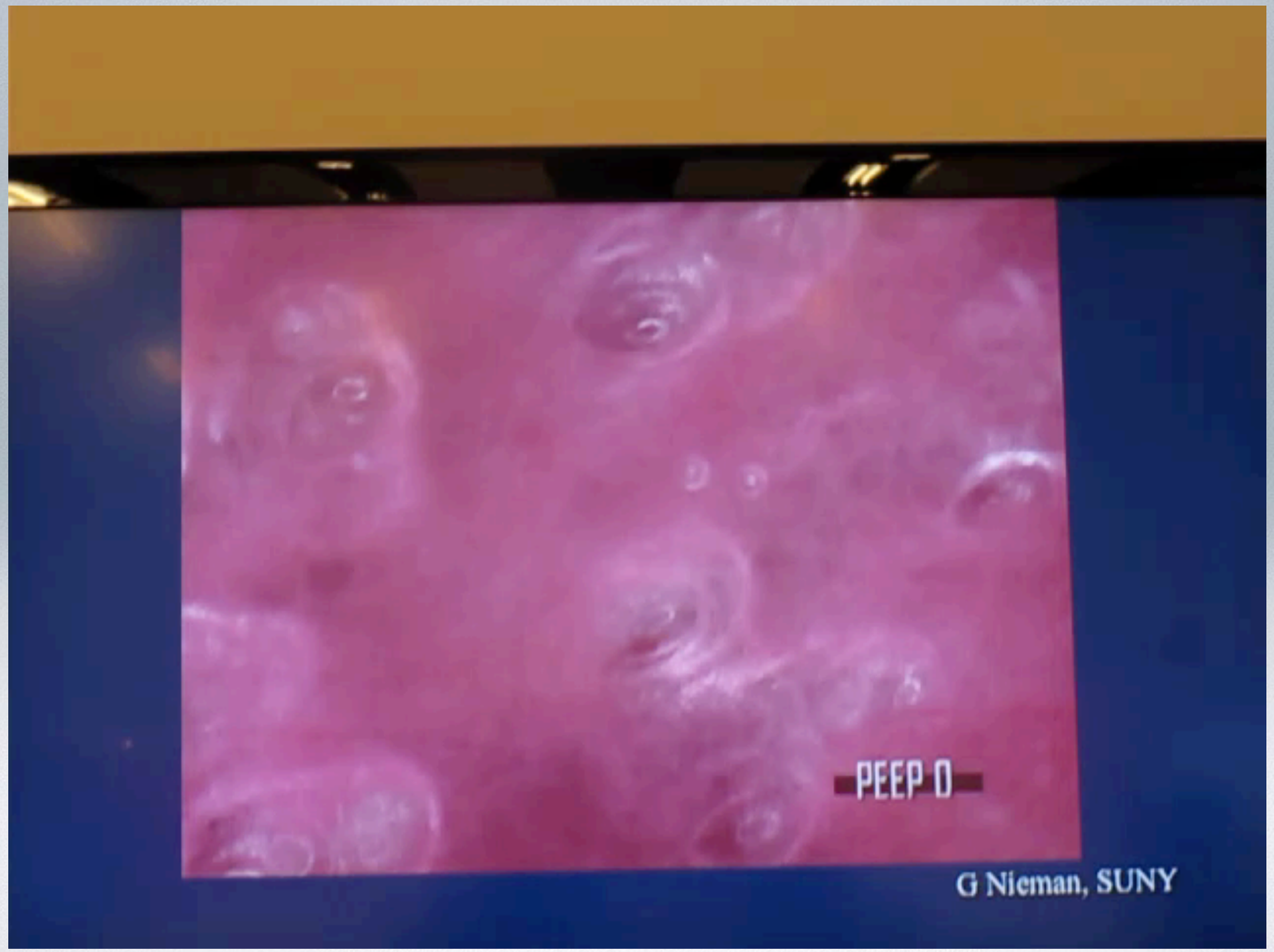
“Lung protective ventilation”

Avoid recruitment/de-recruitment

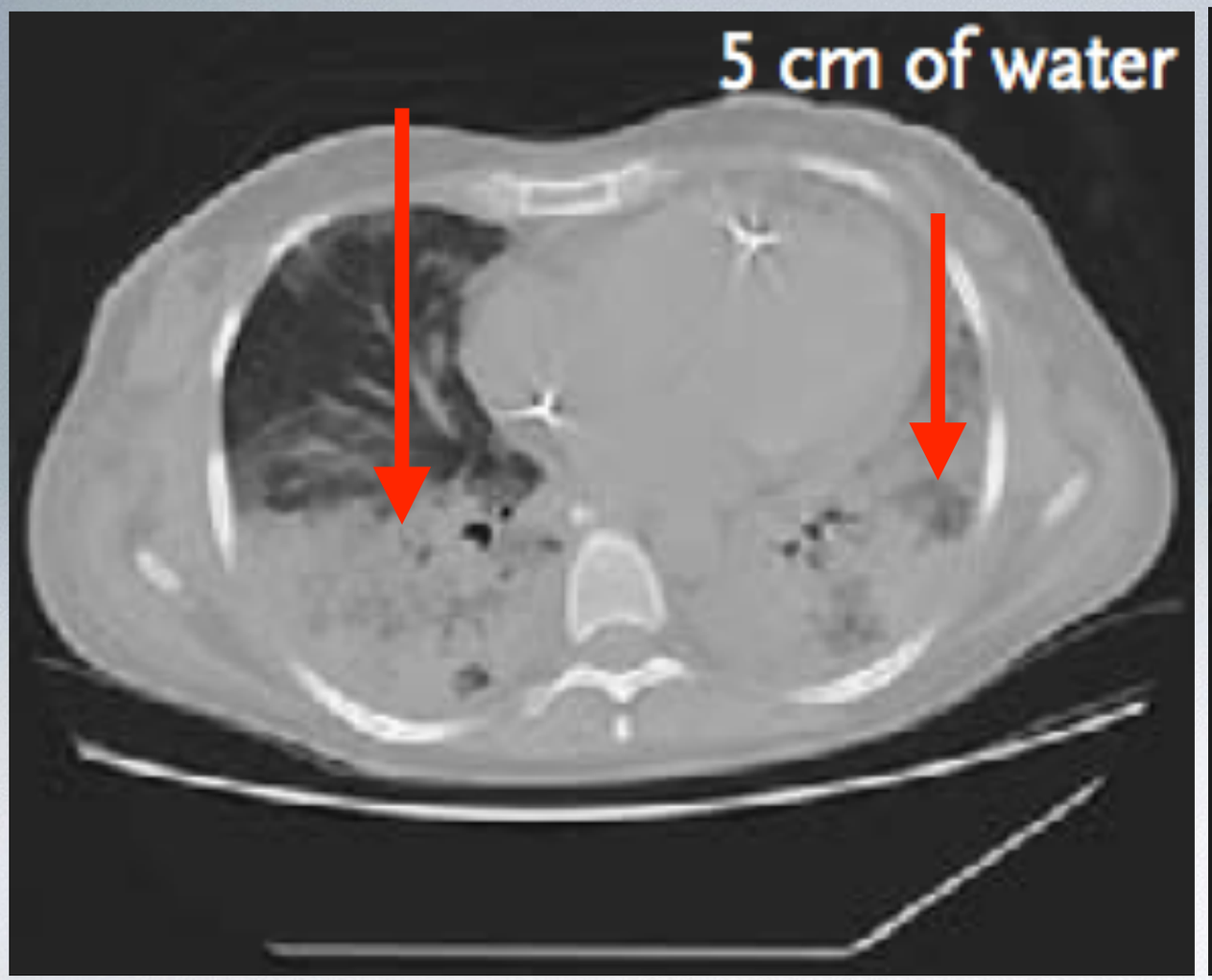
Preventing overdistension and under-recruitment injury

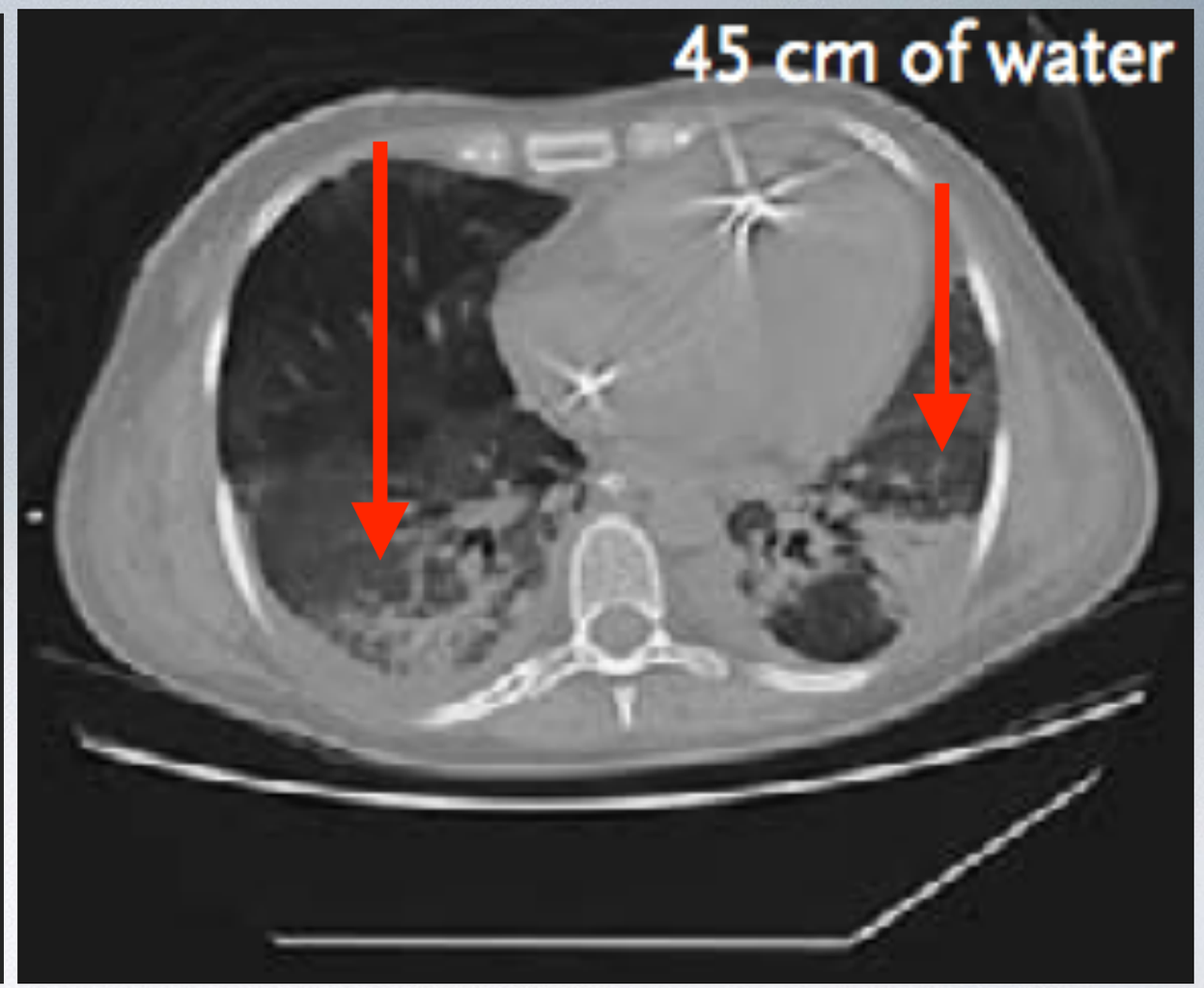


Recruitment/de-recruitment

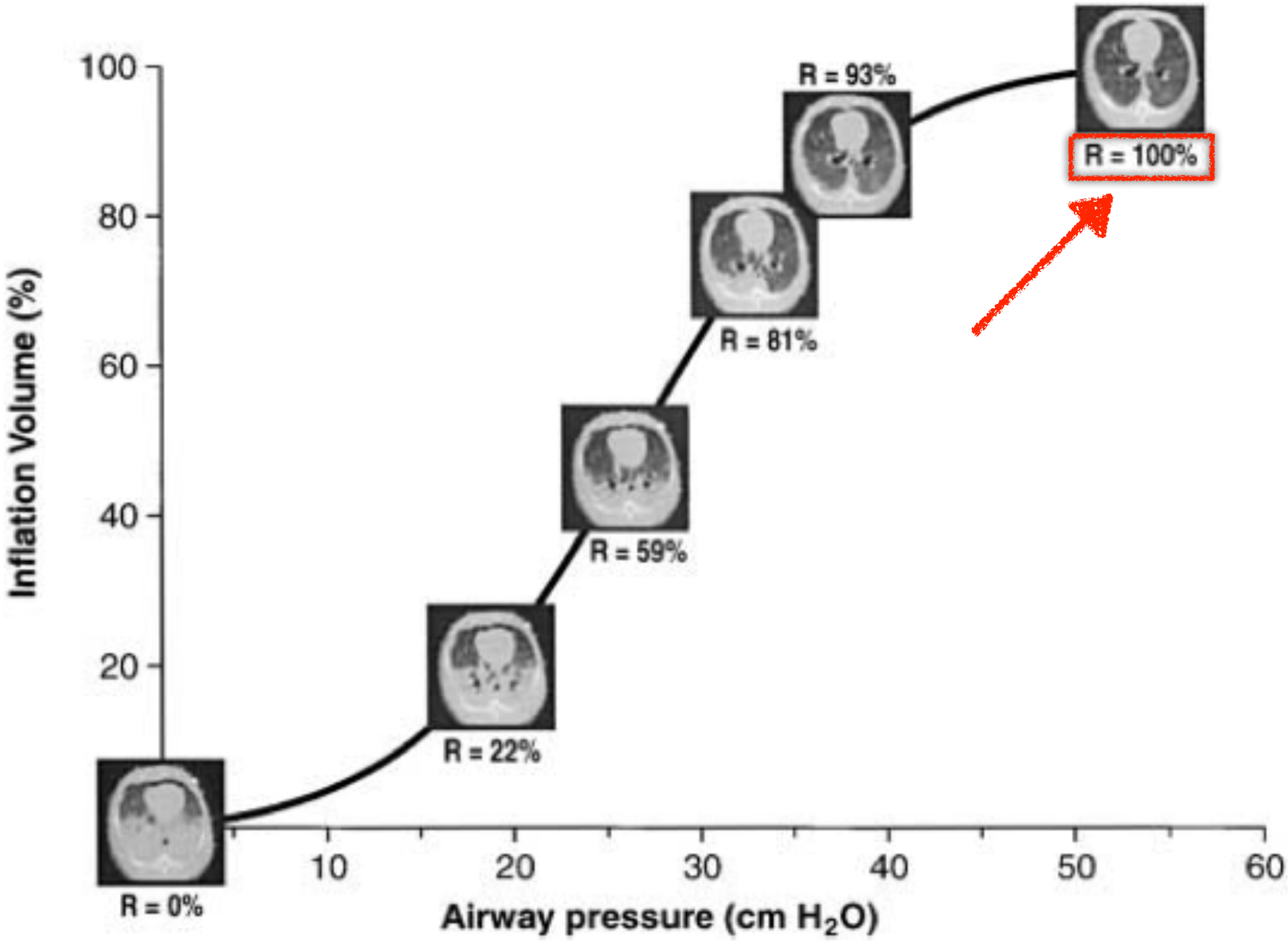


Recruitment manoeuvre and PEEP

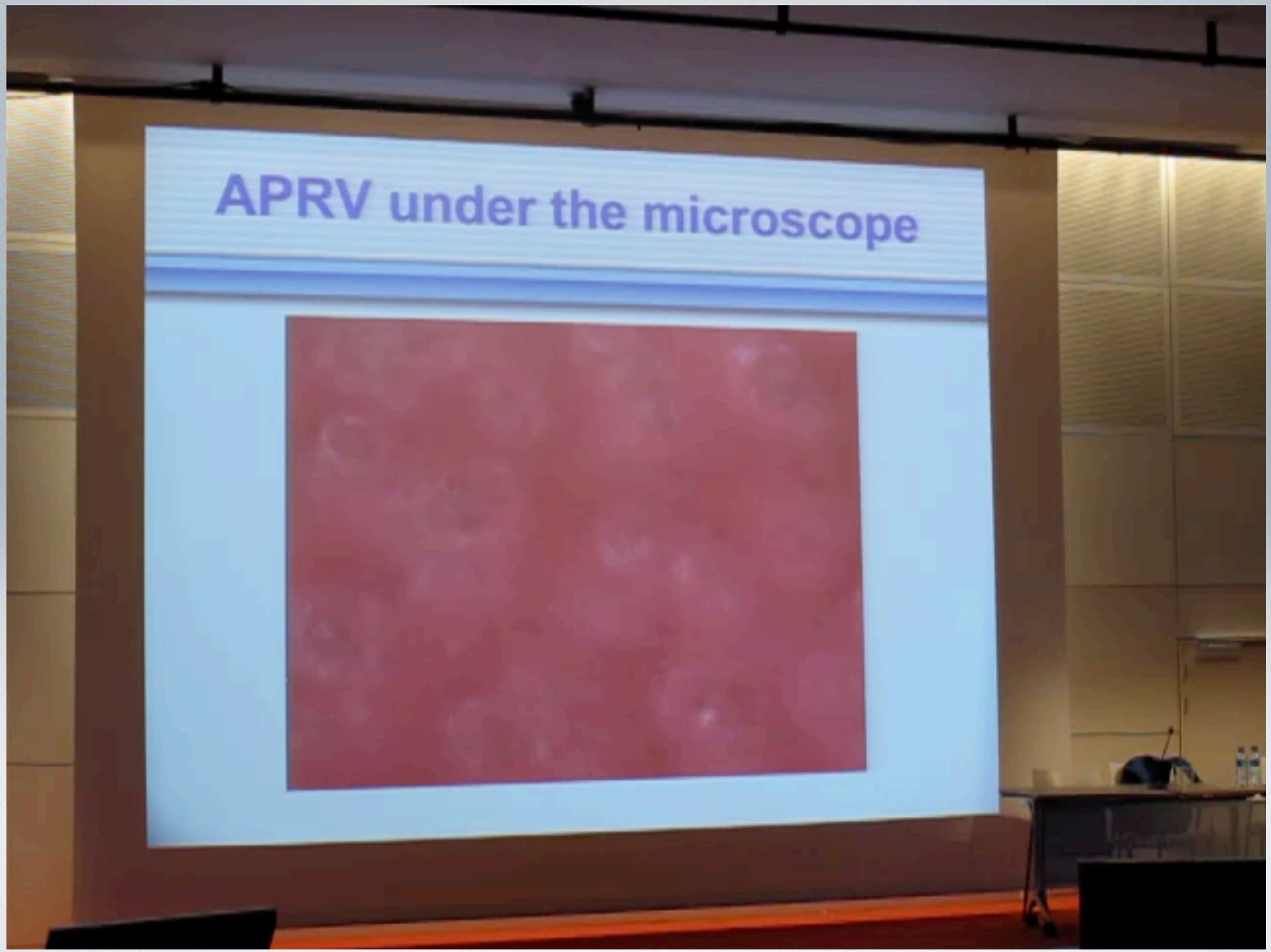




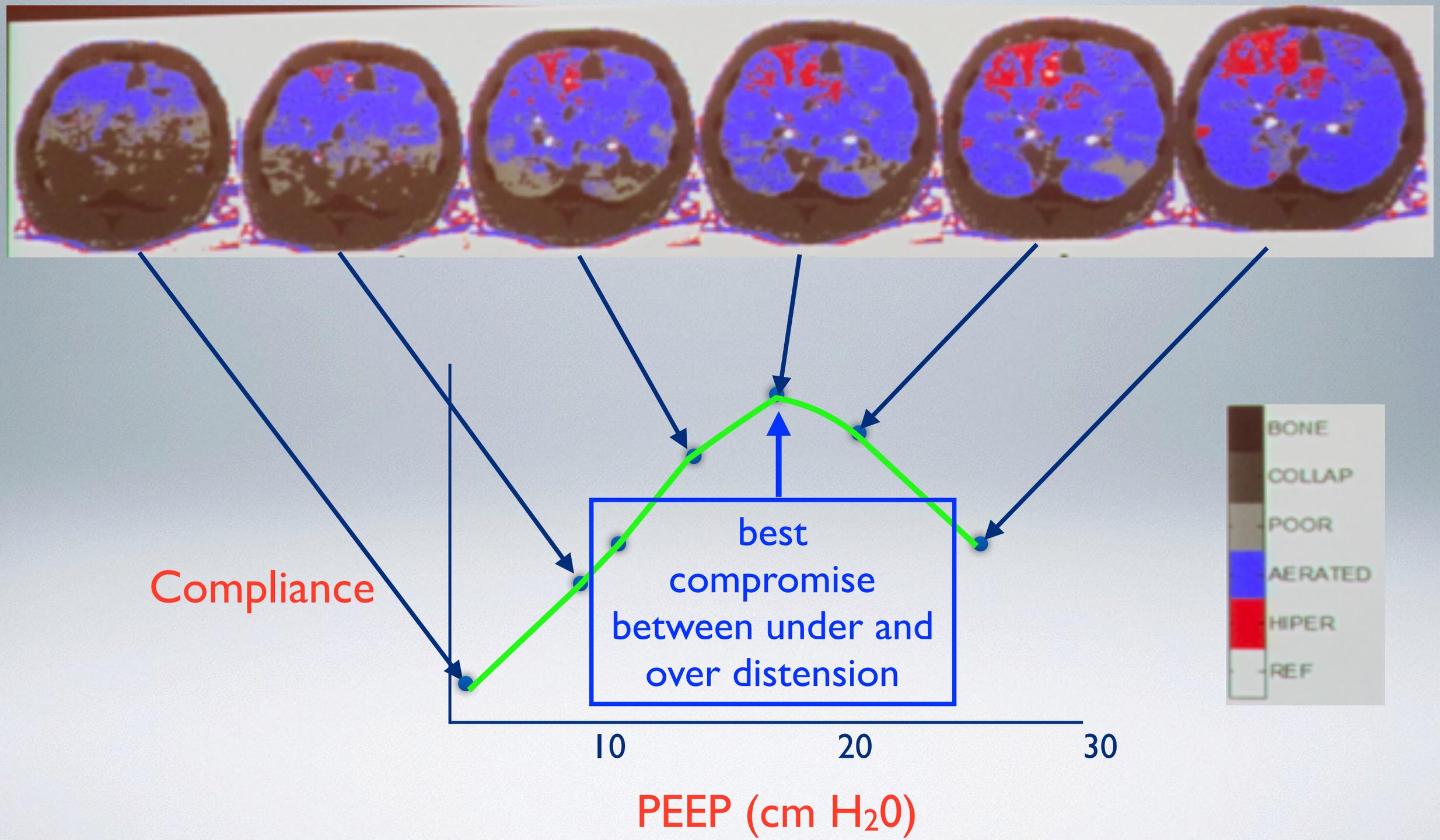
Recruitment manoeuvre and PEEP



Airway Pressure Release Ventilation ("APRV")



Use compliance to titrate PEEP in ARDS



Carvalho AR et al. Intensive care medicine 2008 Dec; 34(12):2291-9

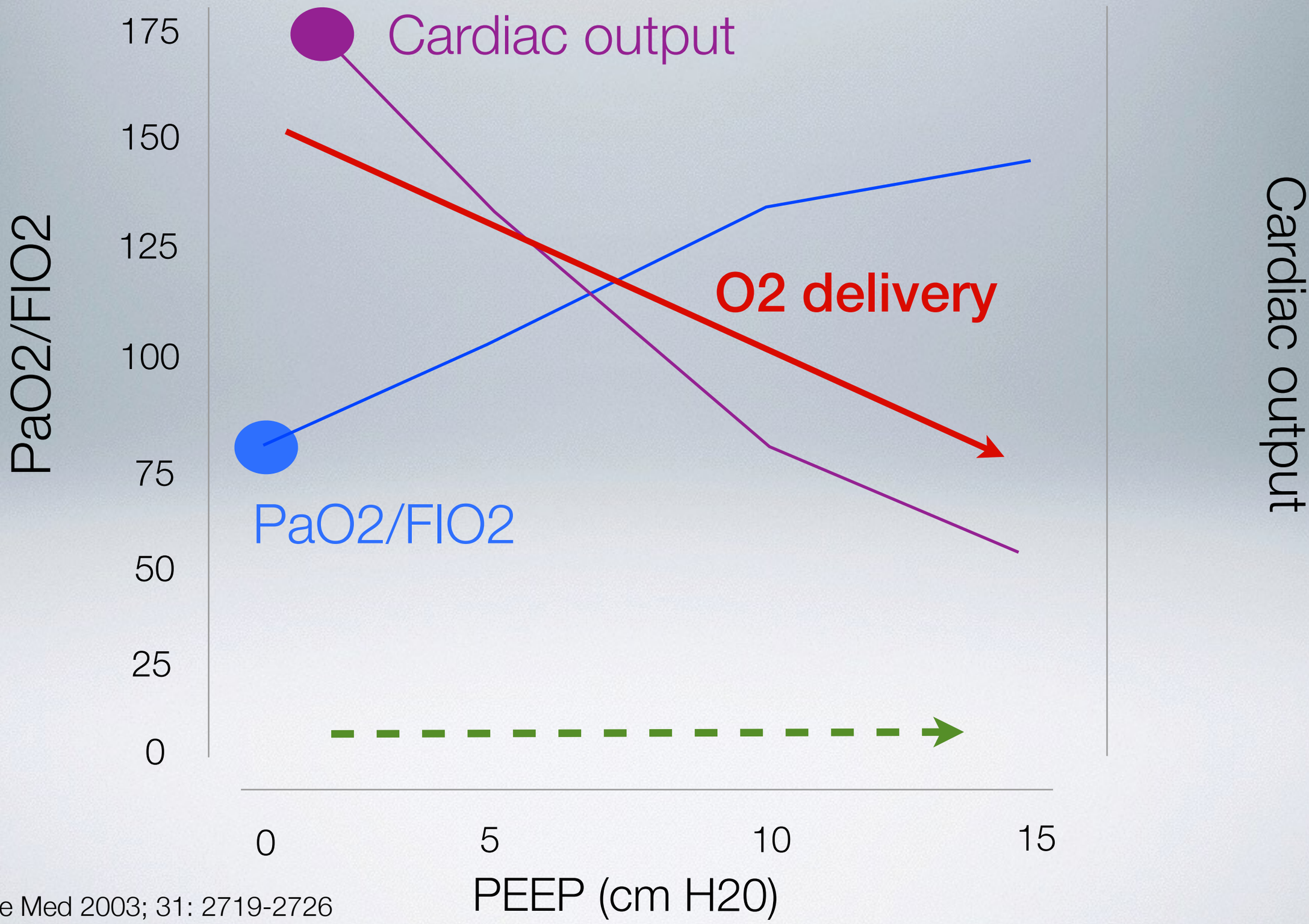
Other considerations

Clinical Case

21 yr old female
Acute respiratory distress following flu-like symptoms (H1N1)
Requires intubation
02 sats continues to drop rapidly from 87% to 78% on 100% 02
Central venous saturation 72%
Attempt at higher PEEP of 25cm H20
Little improvement in 02 sats
But now central venous saturation is 52%

Why?
What does this signify?
What might we do?

Effect of PEEP on lung efficiency vs. cardiac output



Crit Care Med 2003; 31: 2719-2726

Clinical Case

21 year old woman

Very recent Cesarian Section

Increasing respiratory difficulty

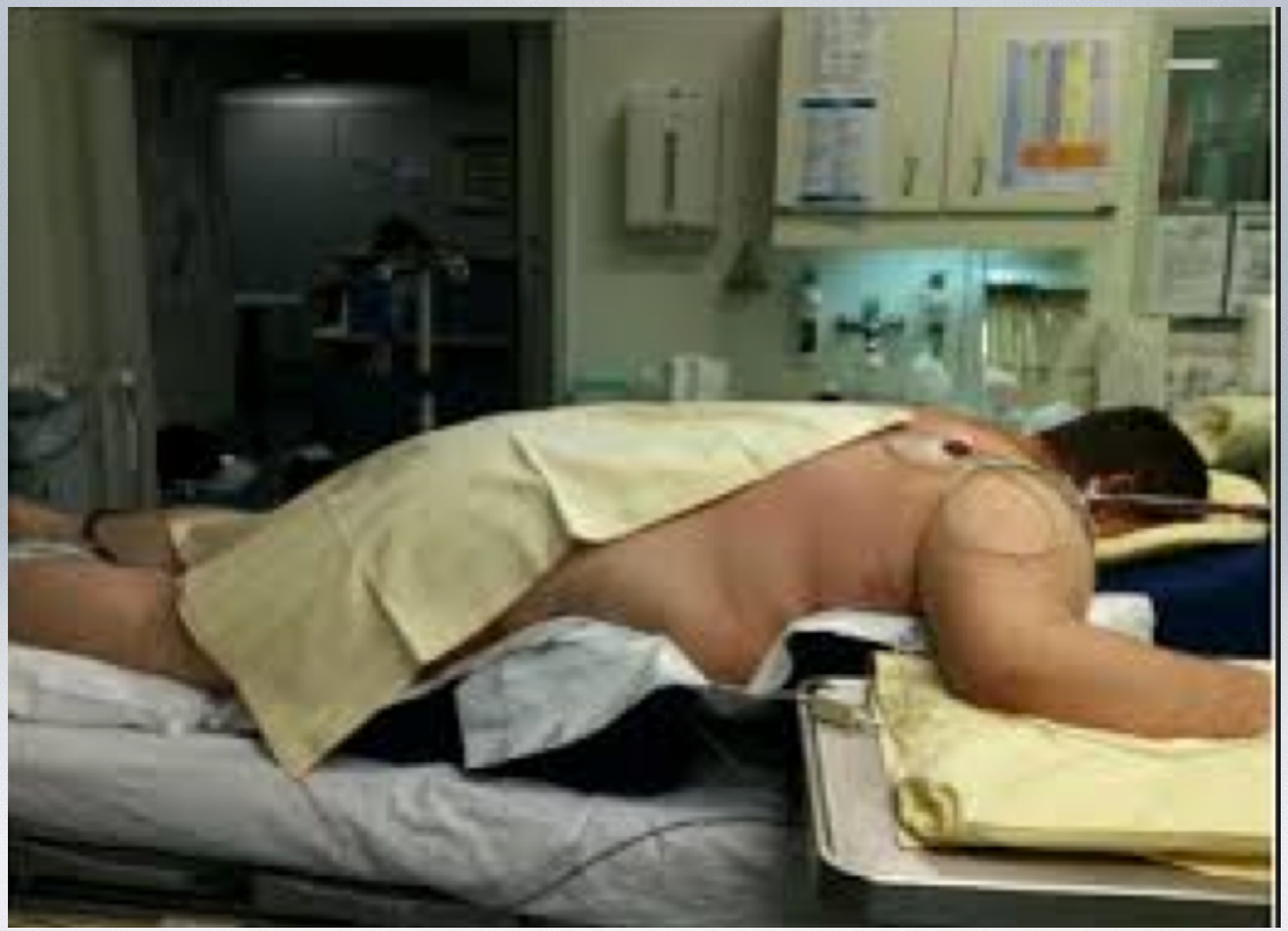
Diagnosis of H1N1 (“swine flu”)

O2 saturations dropping to 75% despite 100% O2

and pressure manoeuvres

What would you do?

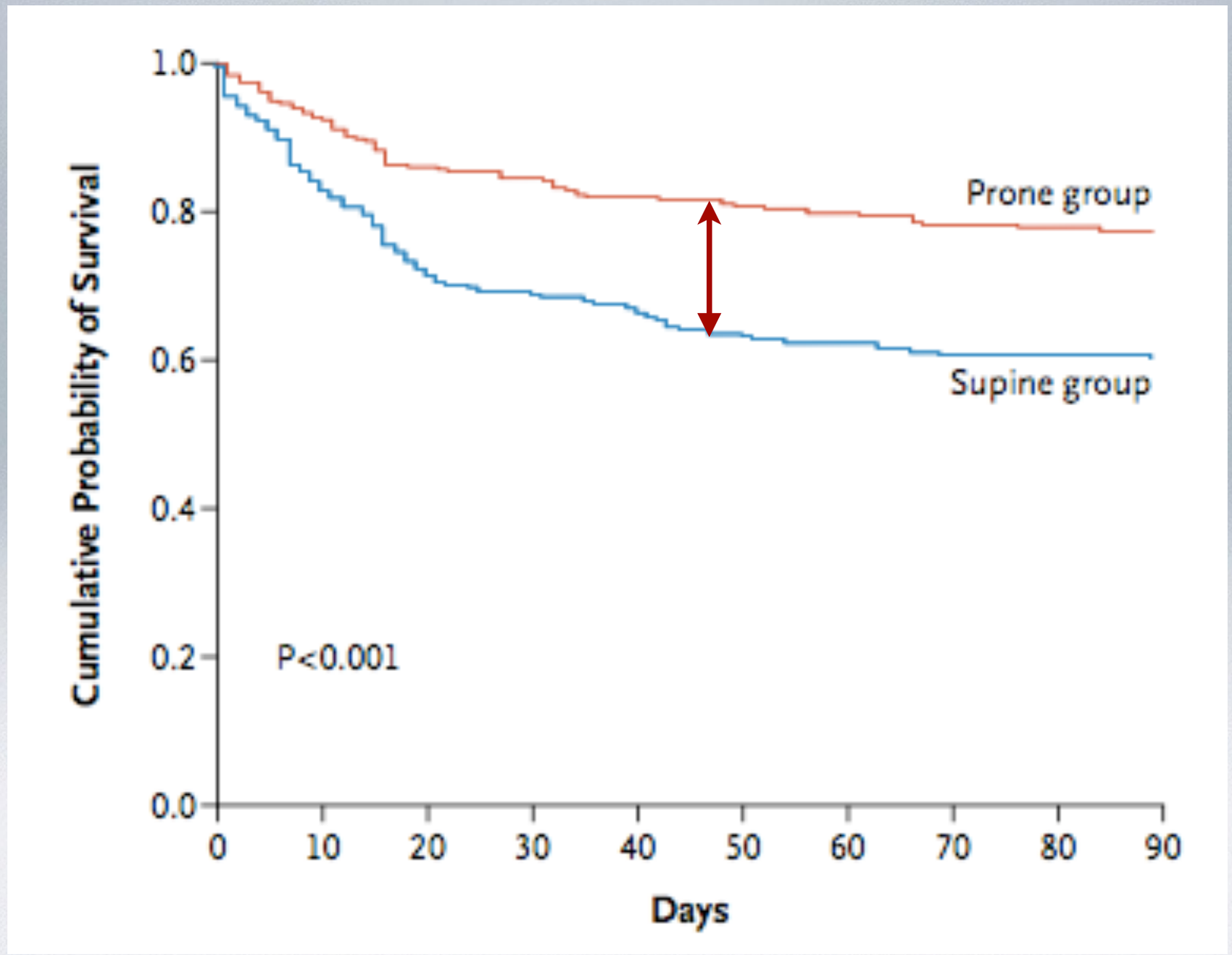
WE TURNED HER PRONE



AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE VOL 188 2013

ORIGINAL ARTICLE

Prone Positioning in Severe Acute Respiratory Distress Syndrome

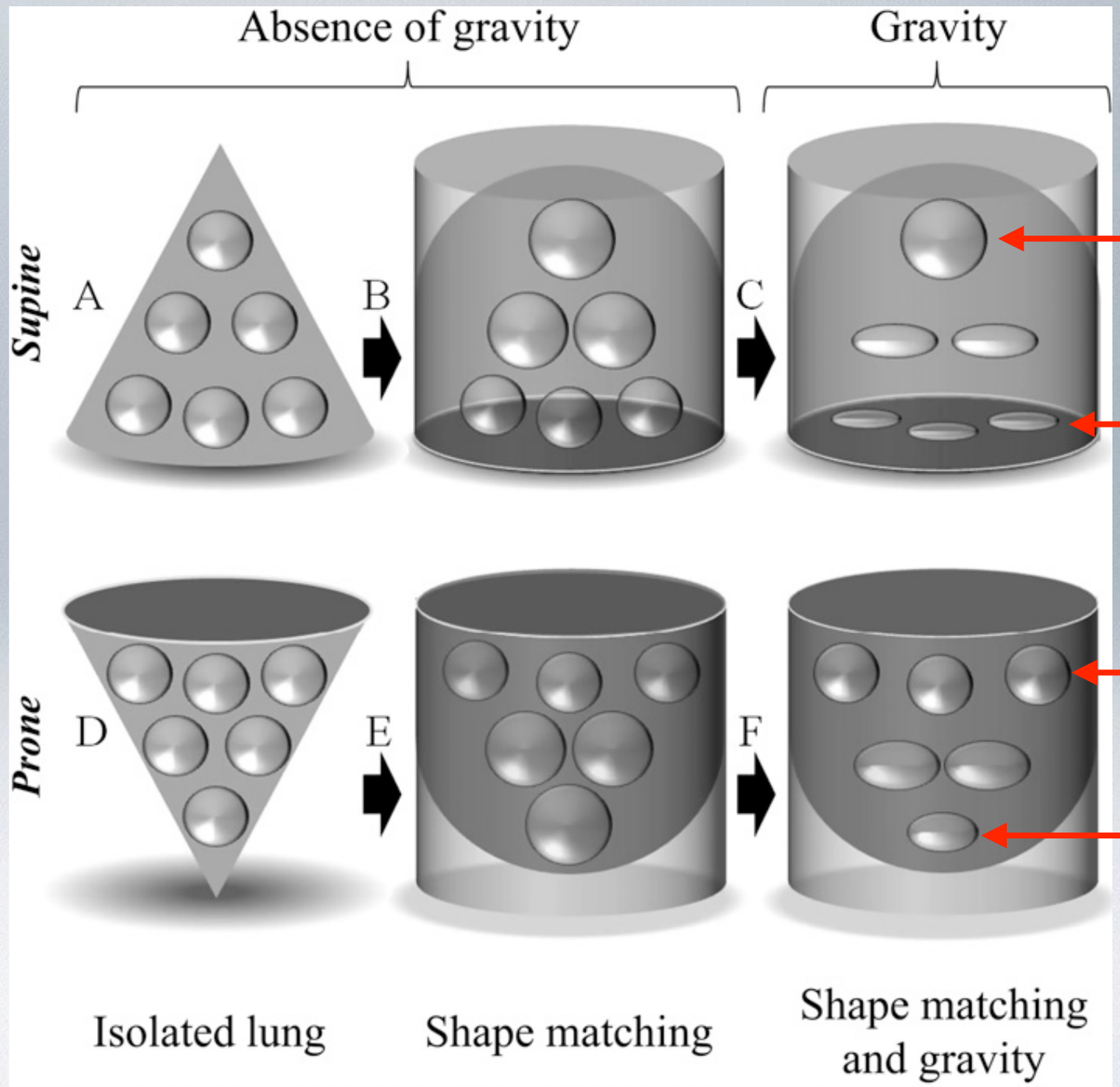


N Engl J Med 2013; 368:2159-2168

HOW DOES PRONING WORK?

2 forces on alveoli:

* **shape mismatch**
* **gravity**



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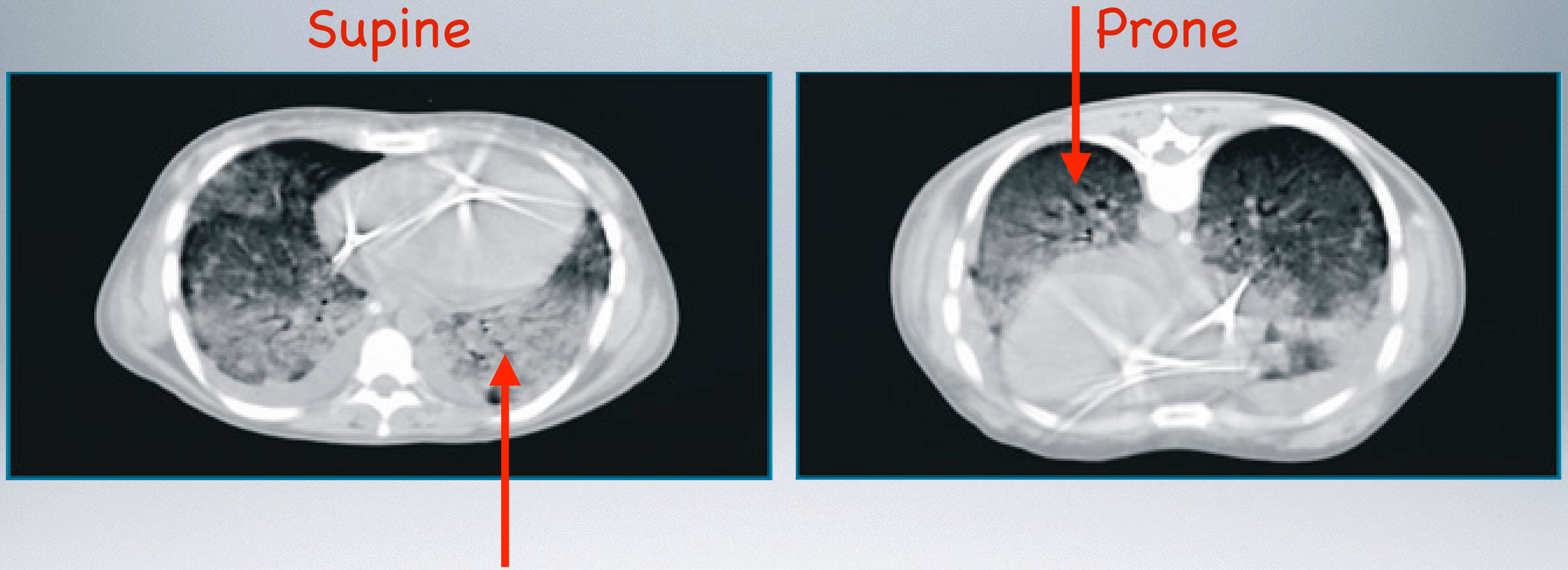
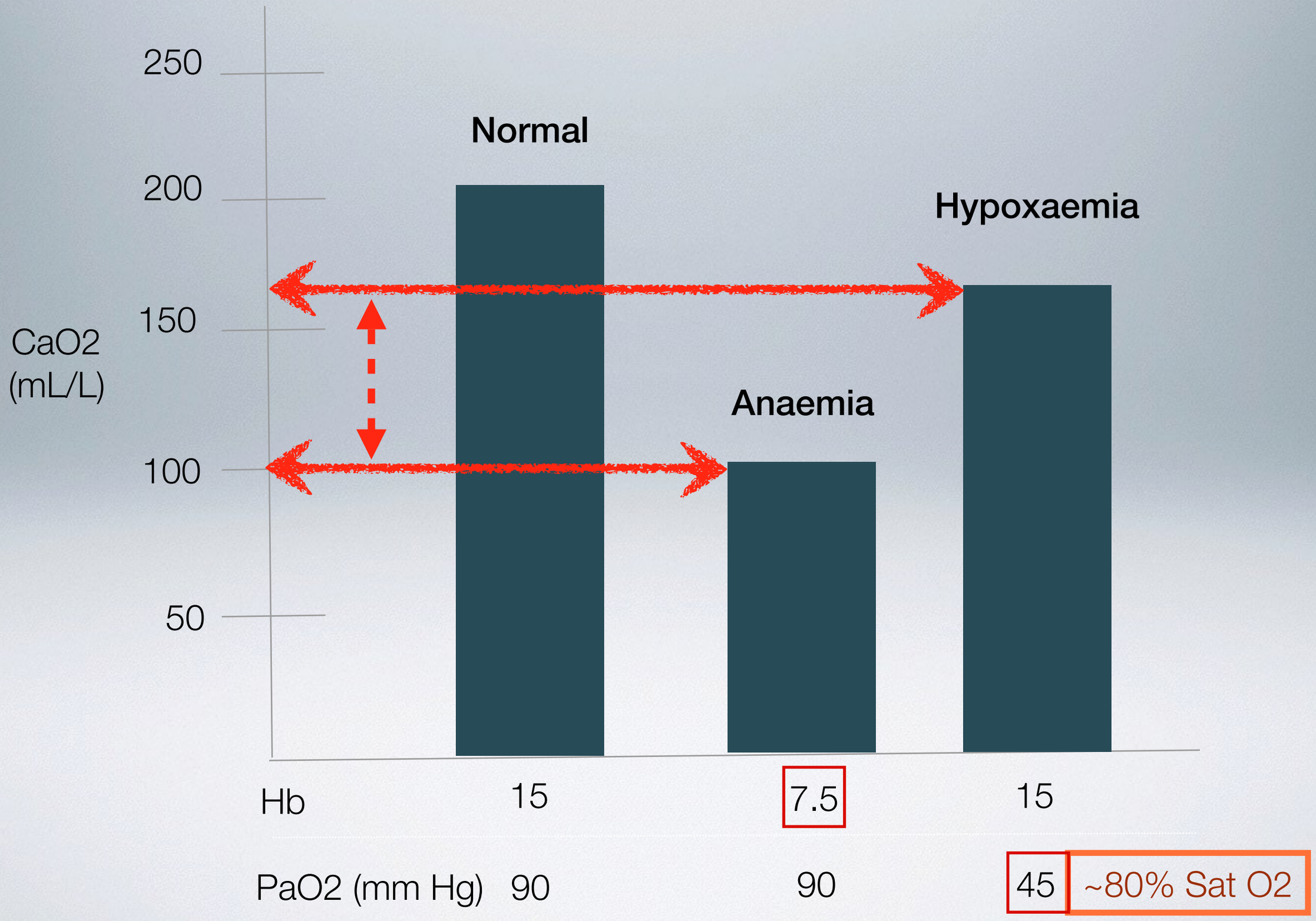
Prone position in ARDS



Am J Respir Crit Care Med Vol 188, Iss. 11, pp 1286–1293, Dec 1, 2013

Effects of a equivalent 50% reduction in Hb and pO2 on **O2 content** in arterial blood



Recap

- We can cause harm to the lungs by
  - overstretching
  - allowing collapse
- Use “low” (i.e., normal tidal volumes)
  - calculate ideal weight using **height and sex**
- Follow efficiency of gas exchange using P/F ratio

Don’t forget, we are in the **oxygen delivery** business

Cardiac output x Hb x % Sat O2

???

Download at

http://www.jvsmedicscorner.com

Mallory / Everest2013

Thanks for listening