Oxygen Delivery -

Optimising Haemodynamics with Fluid

Understanding the Physiology

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

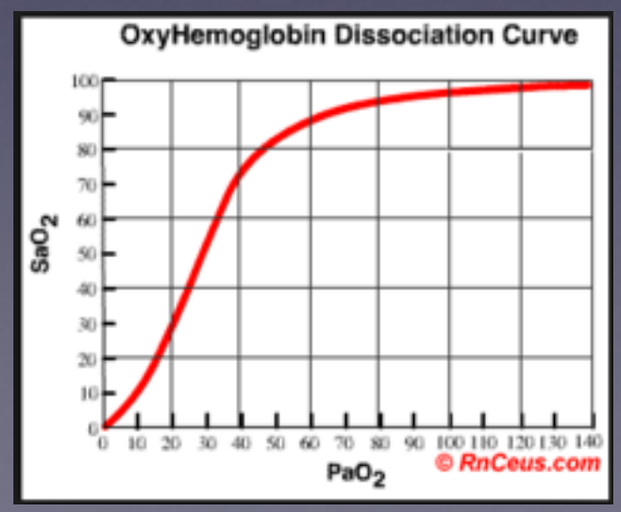
Why is cardiac output so important ?

Cardiac output x Hb x % Sat O2

Cardiac output the only parameter that:

- responds **rapidly**

- does **not plateau**



Consider:

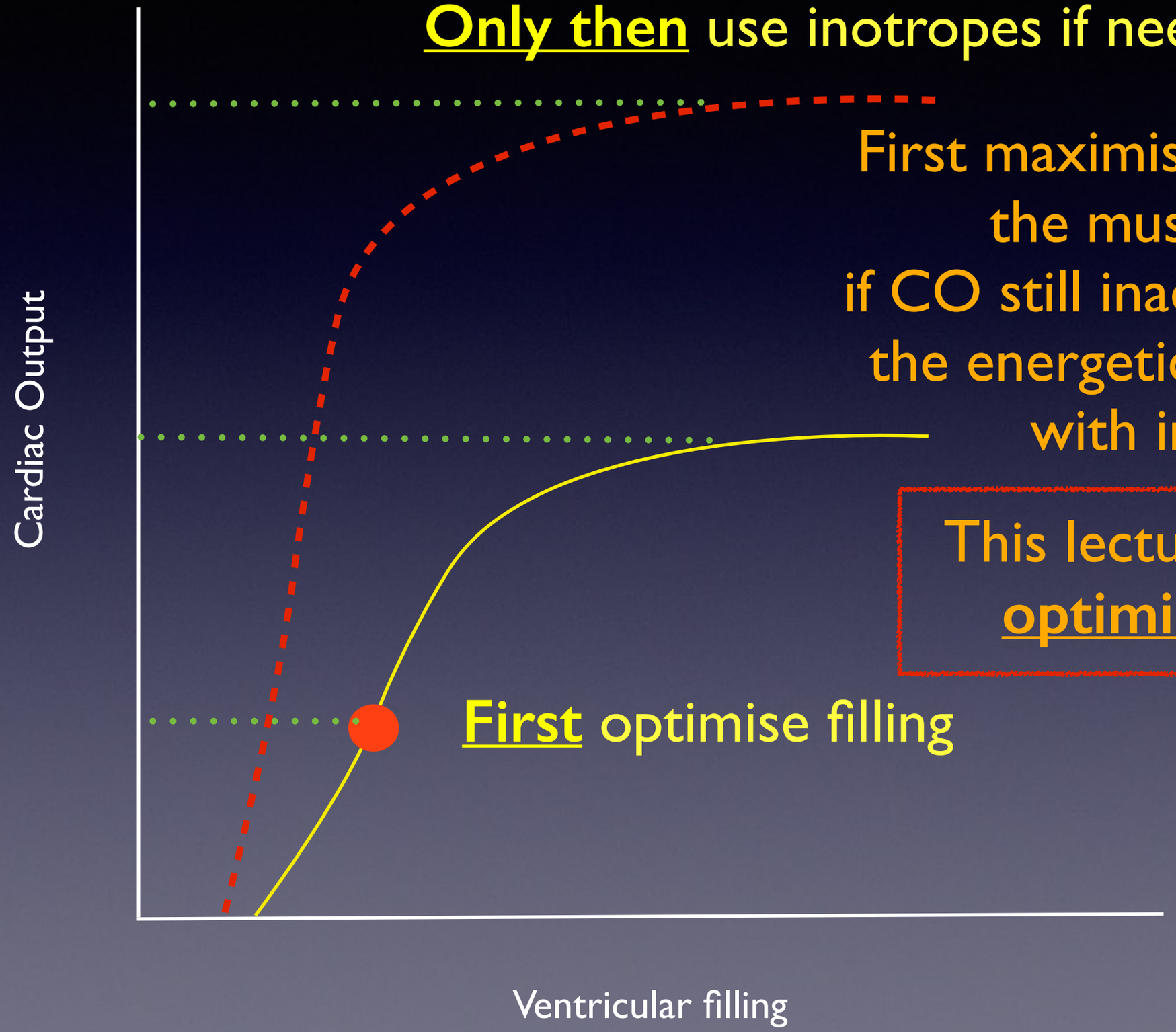
- We can easily measure Hb and O2 Sat.
- The most important factors, **cardiac output**, and its vital component, **preload**, are estimated clinically.
- Imagine if we had to look for cyanosis or pale conjunctiva.
- Future use of pulse contour analysis on the near horizon.

Achieving effective cardiac output

Only then use inotropes if needed

First maximise the **stretch** of the muscle (filling), if CO still inadequate, improve the energetics of the muscle with inotropes.

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



Clinical Case

77 yr old lady

C. Difficile toxic mega colon

Peripherally very oedematous

Received 5.5 l fluid

BP = 95/55 ; HR 90

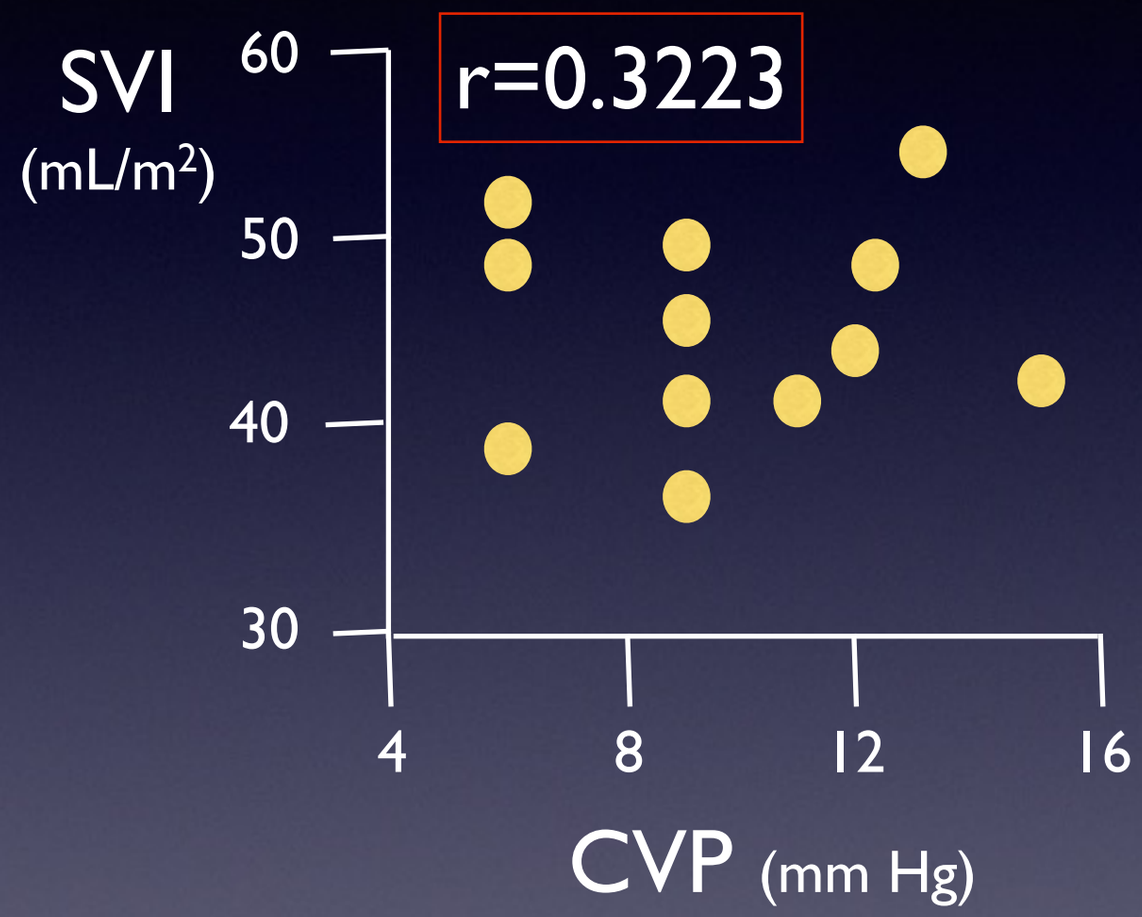
RR = 35

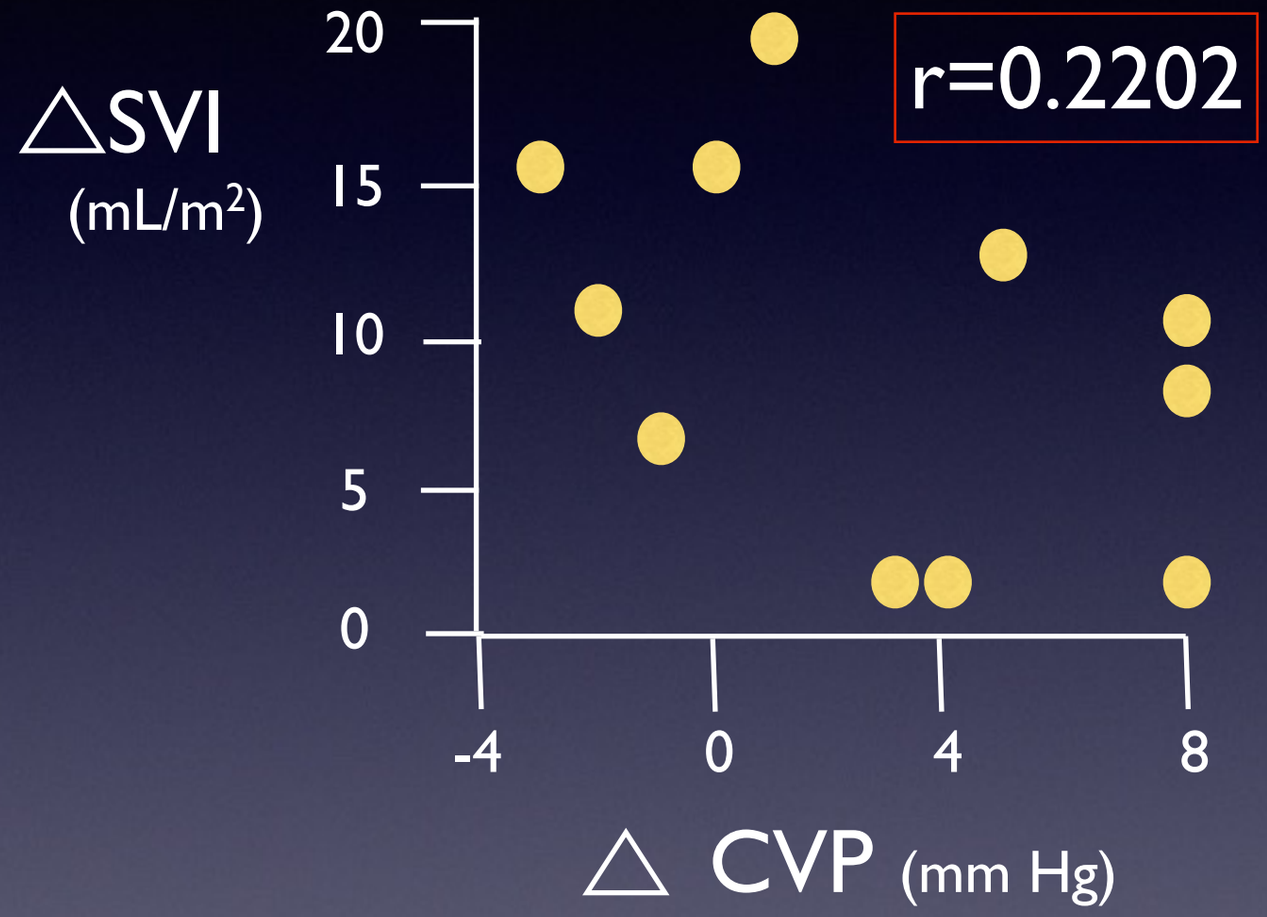
U.O = 15 ml/hr

So do you give more fluid or not?

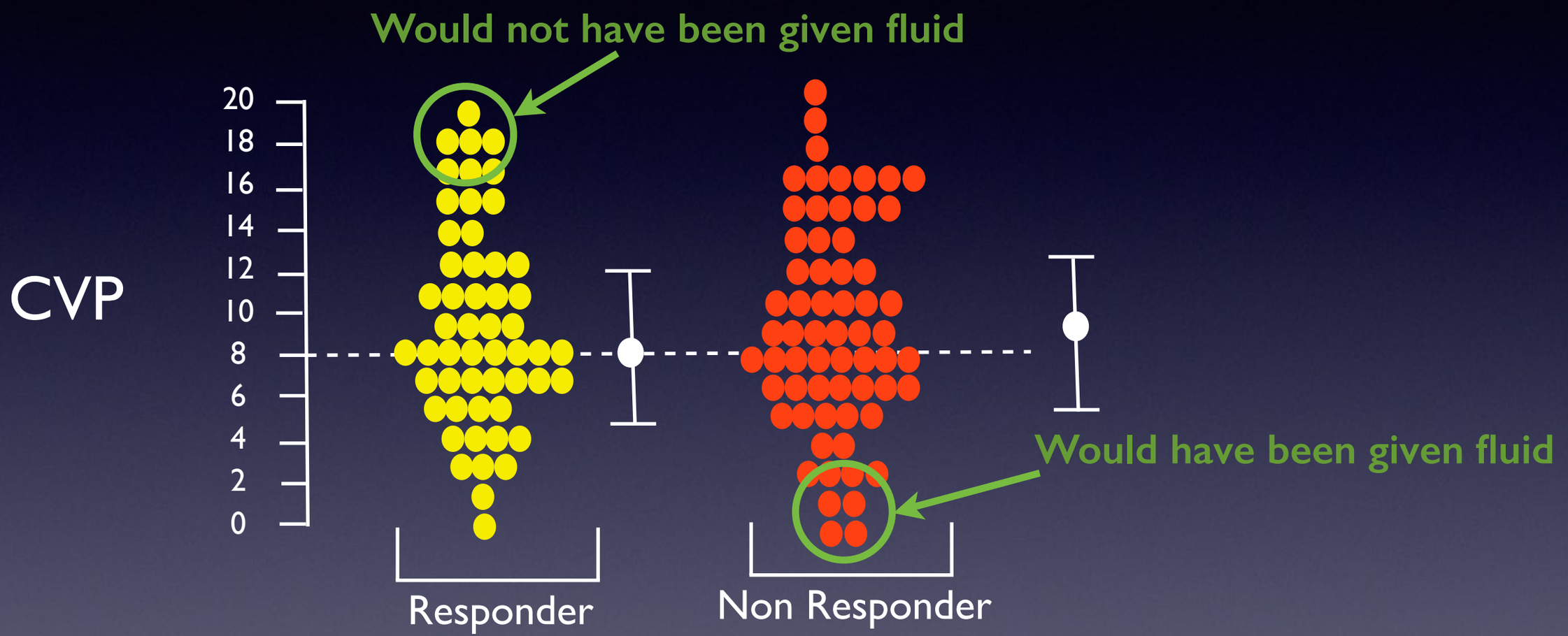
How do you know?

How about the CVP ?





Crit Care Med 2004 Vol. 32, No. 3



Crit Care Med 2007 Vol. 35, No.

CHEST Special Feature

Does Central Venous Pressure Predict Fluid Responsiveness?*

Conclusions: This systematic review (**24 studies**) demonstrated a very poor relationship between CVP and blood volume as well as the inability of CVP/ΔCVP to predict the hemodynamic response to a fluid challenge.

"CVP should not be used to make clinical decisions regarding fluid management."

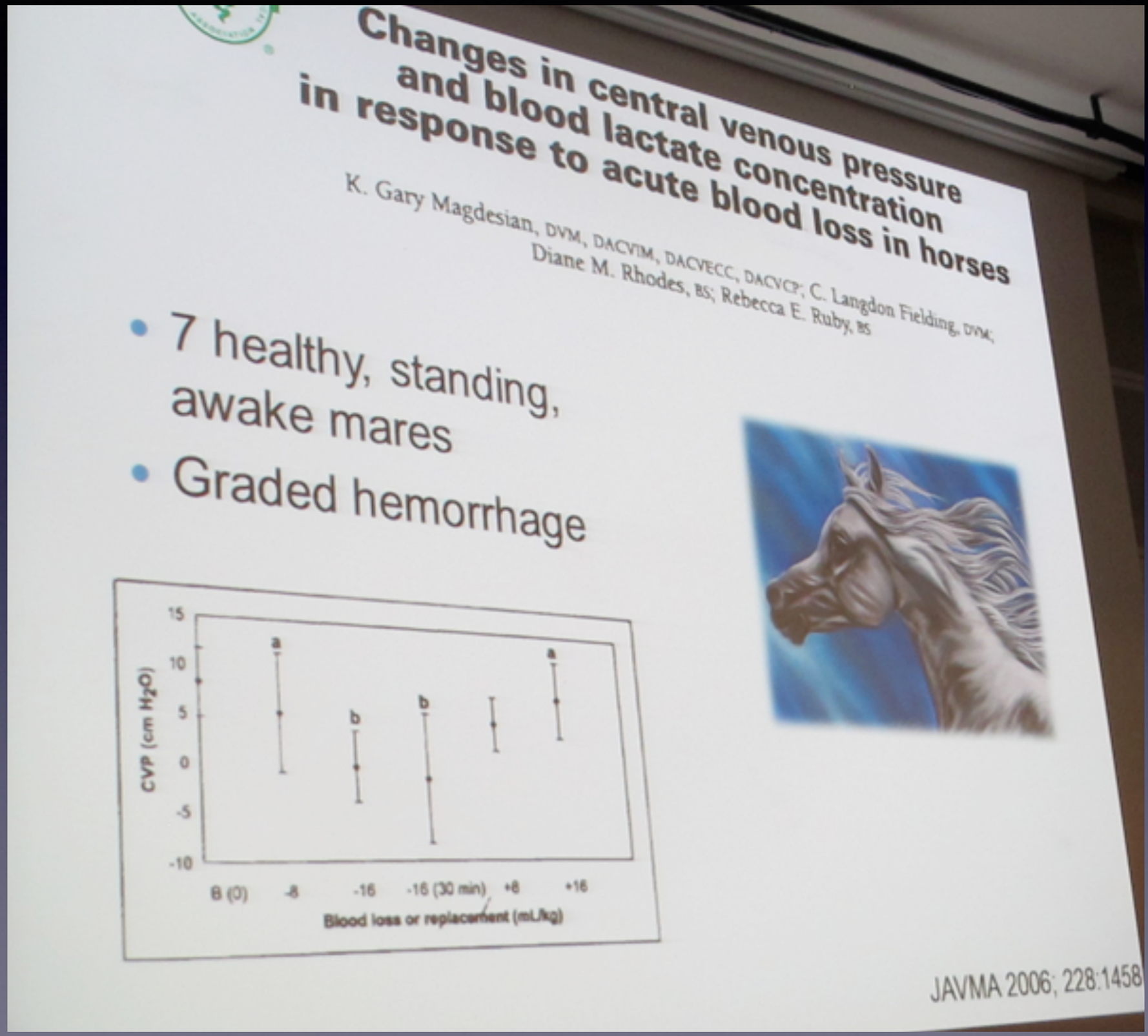
CHEST **2008**; 134:172–178

Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis

"**43 studies** : AUC was 0.56 (coin flip)
There is no data in any group of patients to support using the CVP to guide fluid therapy. This approach **must be abandoned**."

CCM July **2013**; 41:7; 1774

In fact....the only "pro" 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"!!!

Understanding the Physiology of Preload

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

807 m

Illustration of transmural pressure

Illustration of transmural pressure

The NEW ENGLAND JOURNAL of MEDICINE

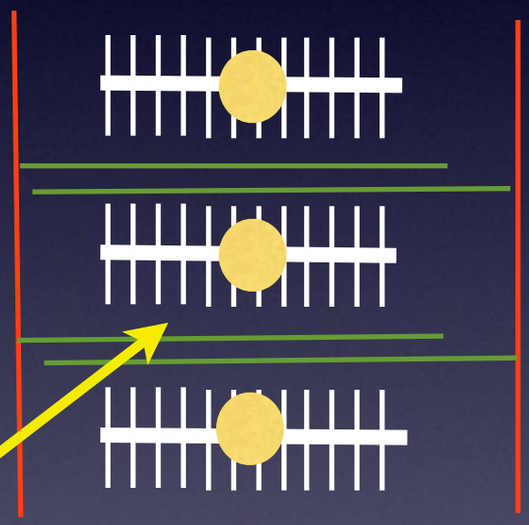
CORRESPONDENCE

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

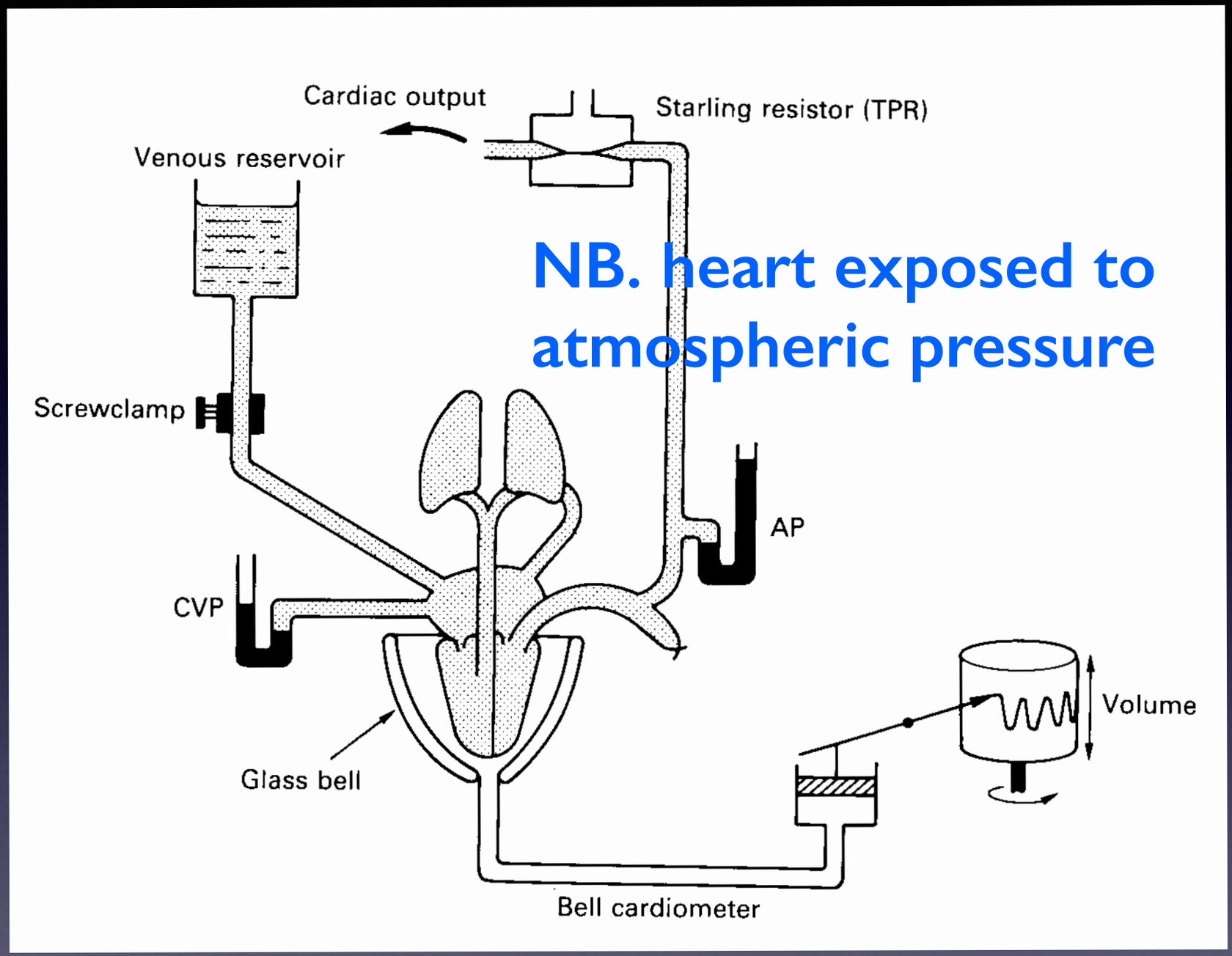
'the greater the stretch of the ventricle in diastole, the greater the stroke work achieved in systole"



Greater overlap of actin-myosin crossbridges

Sarcomere

Starling's experiment

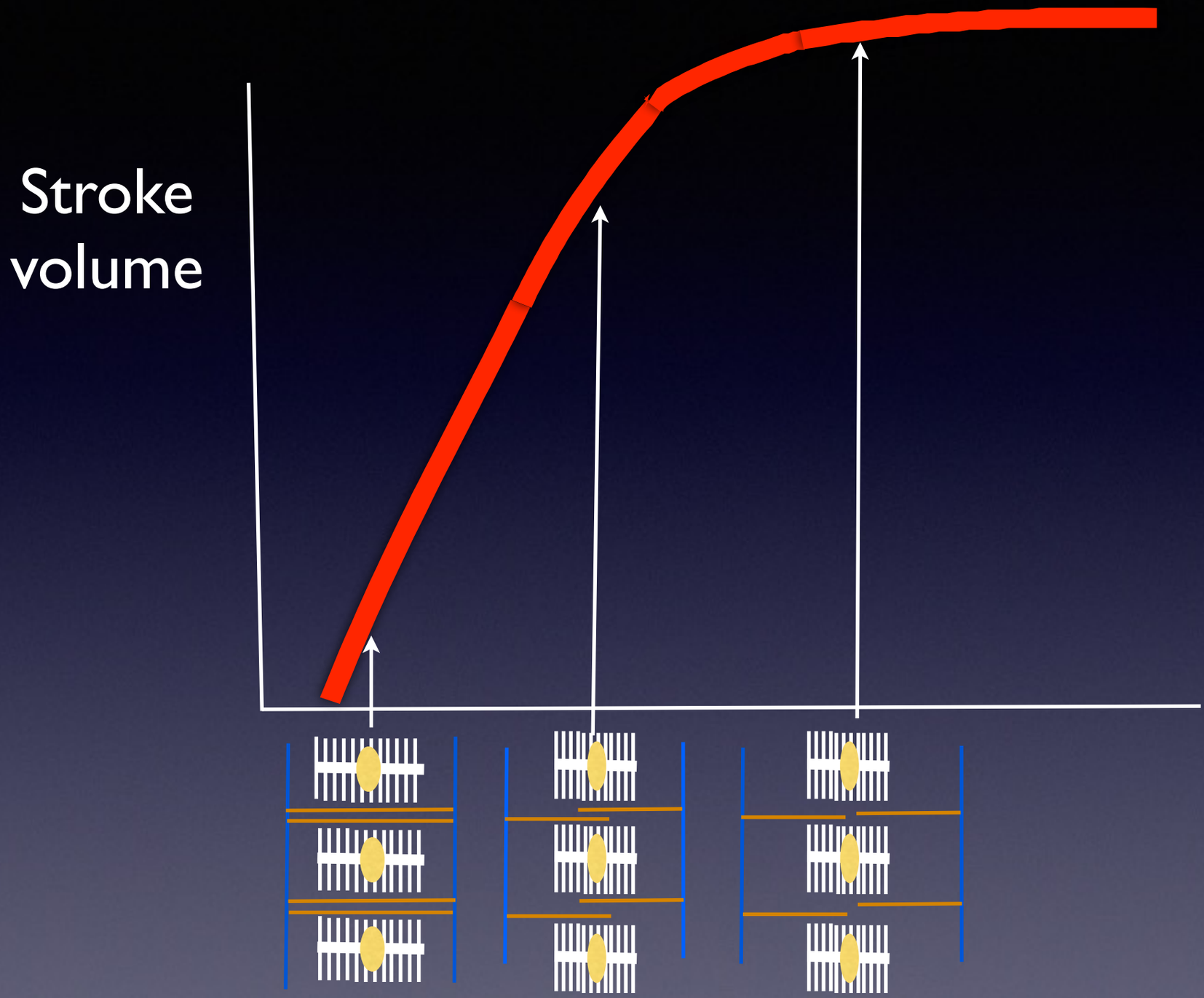


NB. heart exposed to atmospheric pressure

"Starling did <u>not</u> suggest that **right atrial pressure is an independent variable** that controls stroke volume.

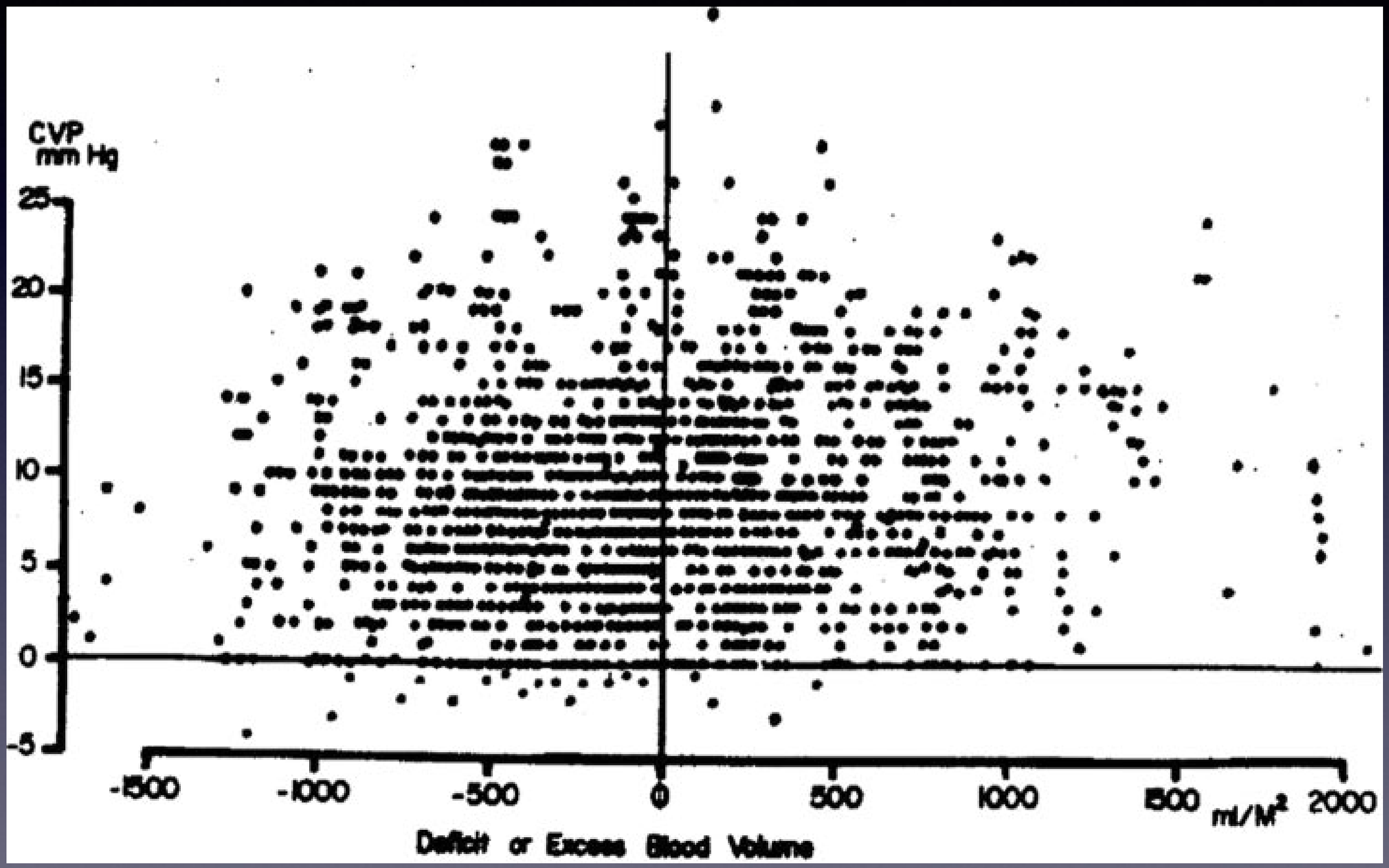
The **independent variable** was the **amount he opened the resistor on the cannula that carried blood back to the heart.**"

Journal of Physiology; 1914, 48, 465-511



Preload (= muscle stretch)

But.....no correlation between CVP and hemodynamic response to fluid!
Was Starling wrong?



(r=0.27)

Crit Care Med 1984; 12:107–112

So, the CVP doesn't work-
So why not ?

- Depends on <u>not</u> just blood volume

  increased afterload can raise right atrial pressure without increasing cardiac output

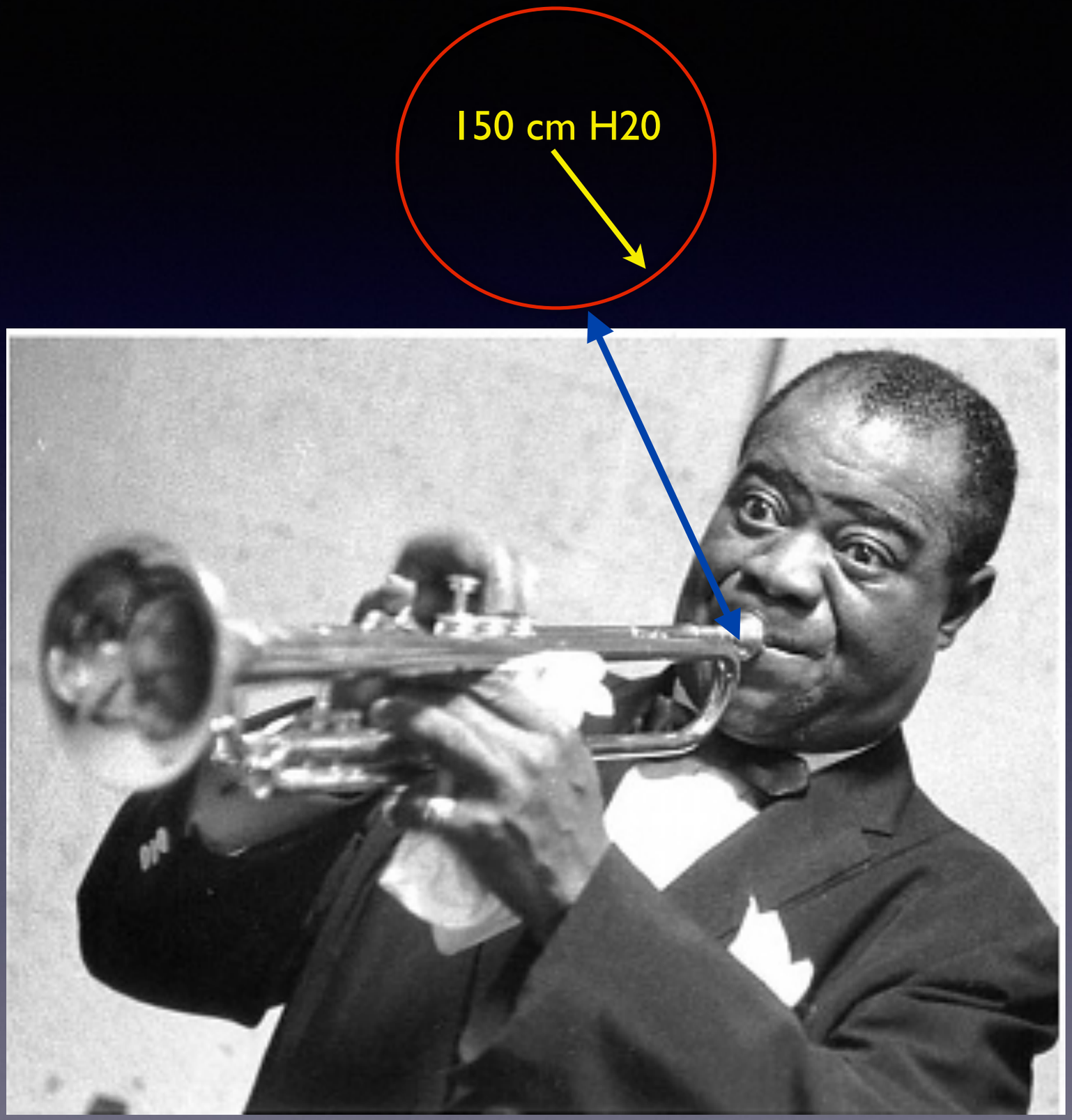
- Doesn't describe "upstream" pressure which dictates venous return

- Doesn't tell you <u>where</u> you are on the Starling curve, nor <u>which</u> Starling curve

- CVP is a composite of the pressure generated by the volume of blood that distends the right atrium and the pressure in the pericardium and thorax

  Ex. COPD with PEEPi, raised intra-abdominal pressure

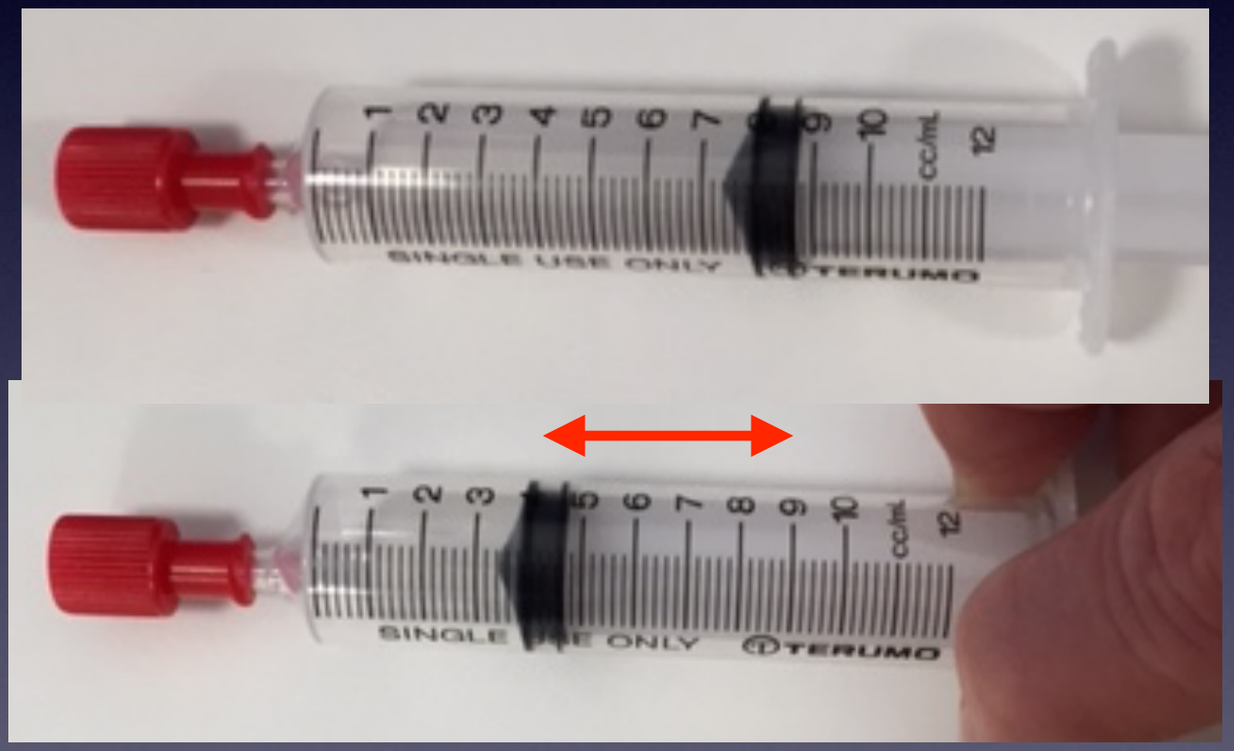
Intra-thoracic presure



Nature 1969; 221 : 1199-1204

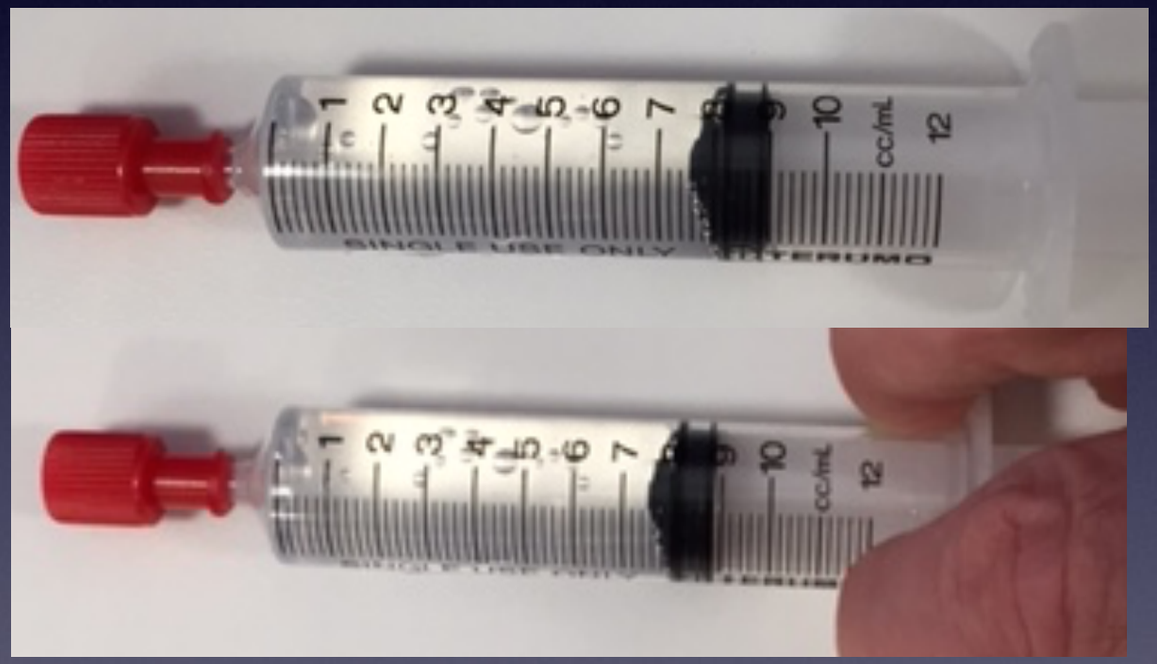
Squeezing the heart

Air



Compressible
less increased pressure

Fluid



Non Compressible
increased pressure

CVP
+5

0
Inside
thorax

+5

+5

CVP
+155

+150
Inside
thorax

+155

+5

Distending pressure of the heart is the same !

CVP
+5

0
Inside
thorax

+5

+5

CVP
+5

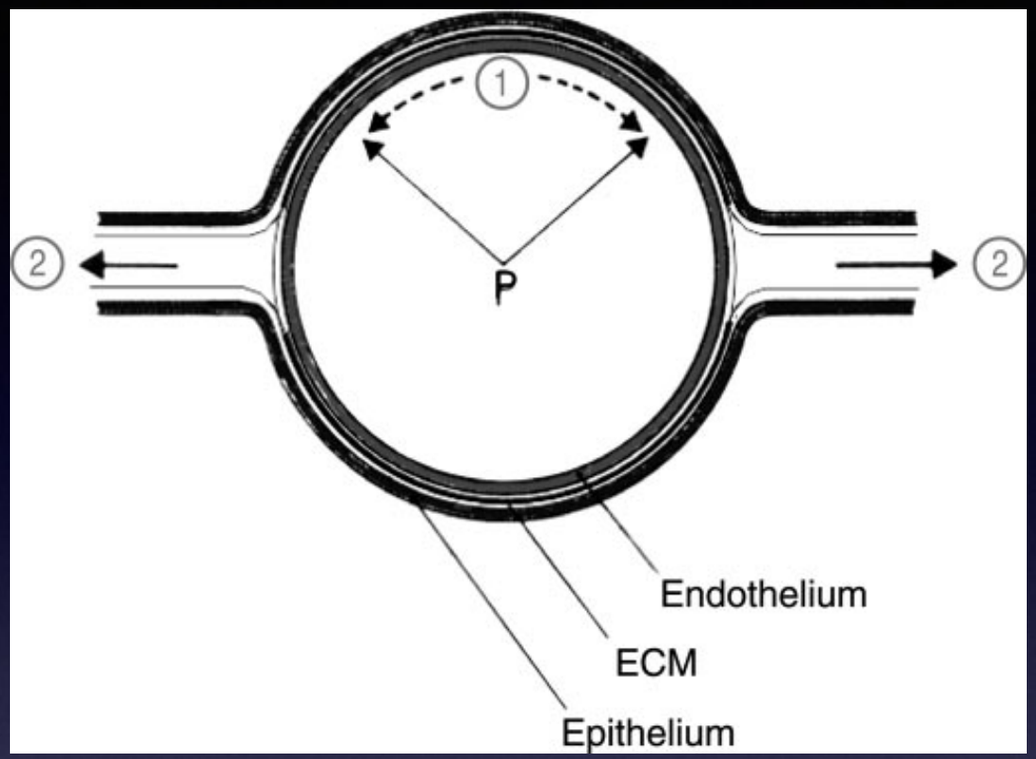
- 100
Inside
thorax

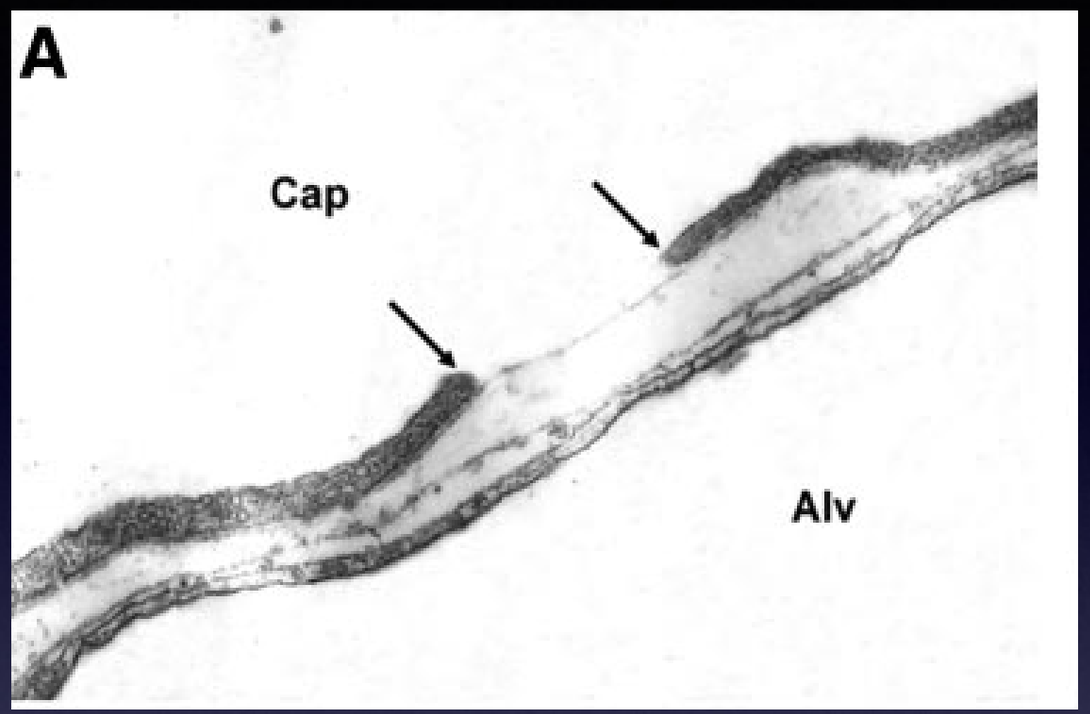
+5

+105

Negative pressure pulmonary oedema

Negative pressure pulmonary oedema





Clinical Case

Fractured femoral shaft
Distended abdomen
Resuscitated with colloid / crystalloid
In great pain, so you give morphine
What happens?
Why?

Venous return

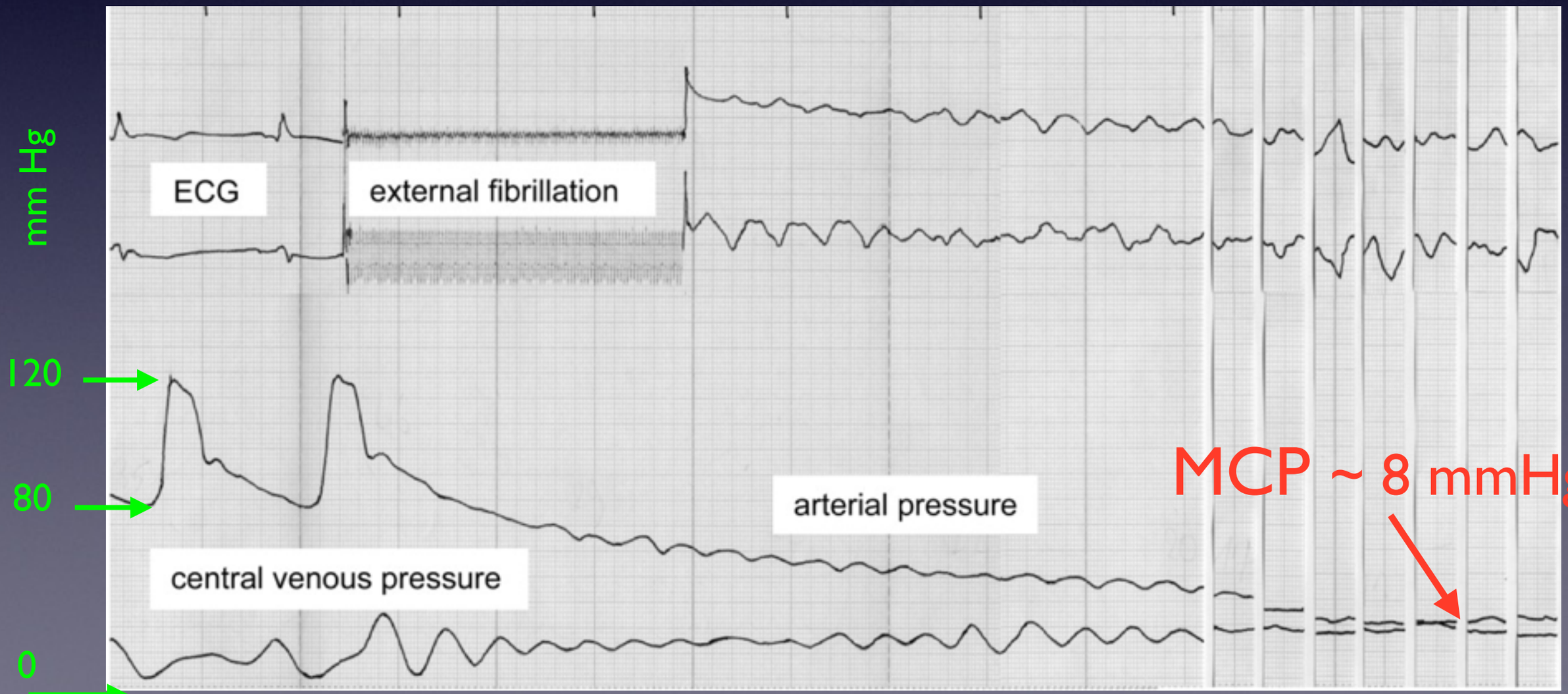
- Mean circulatory pressure (mcp) = pressure throughout vascular circuit if no flow
- Venous return = mcp - CVP
- MCP depends on stressed venous volume ("elastic energy within the system")
- Stressed venous volume depends on venous capacity and volume

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.

Mean circulatory pressure

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

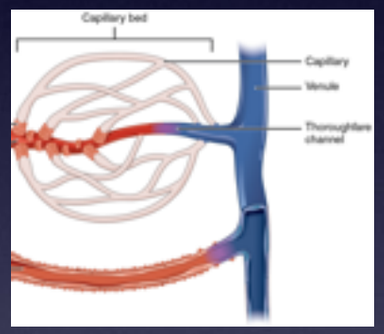


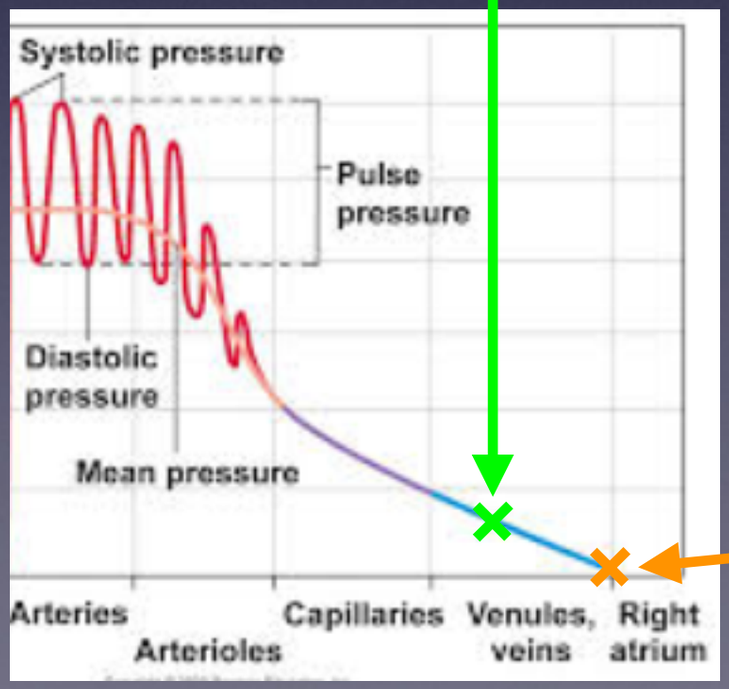
Venous return

Venous return = MCP - CVP

upstream pressure

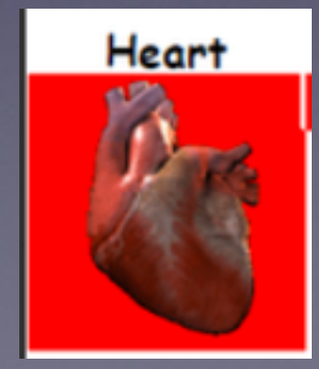
"MCP"





downstream pressure

"CVP"



Venous return

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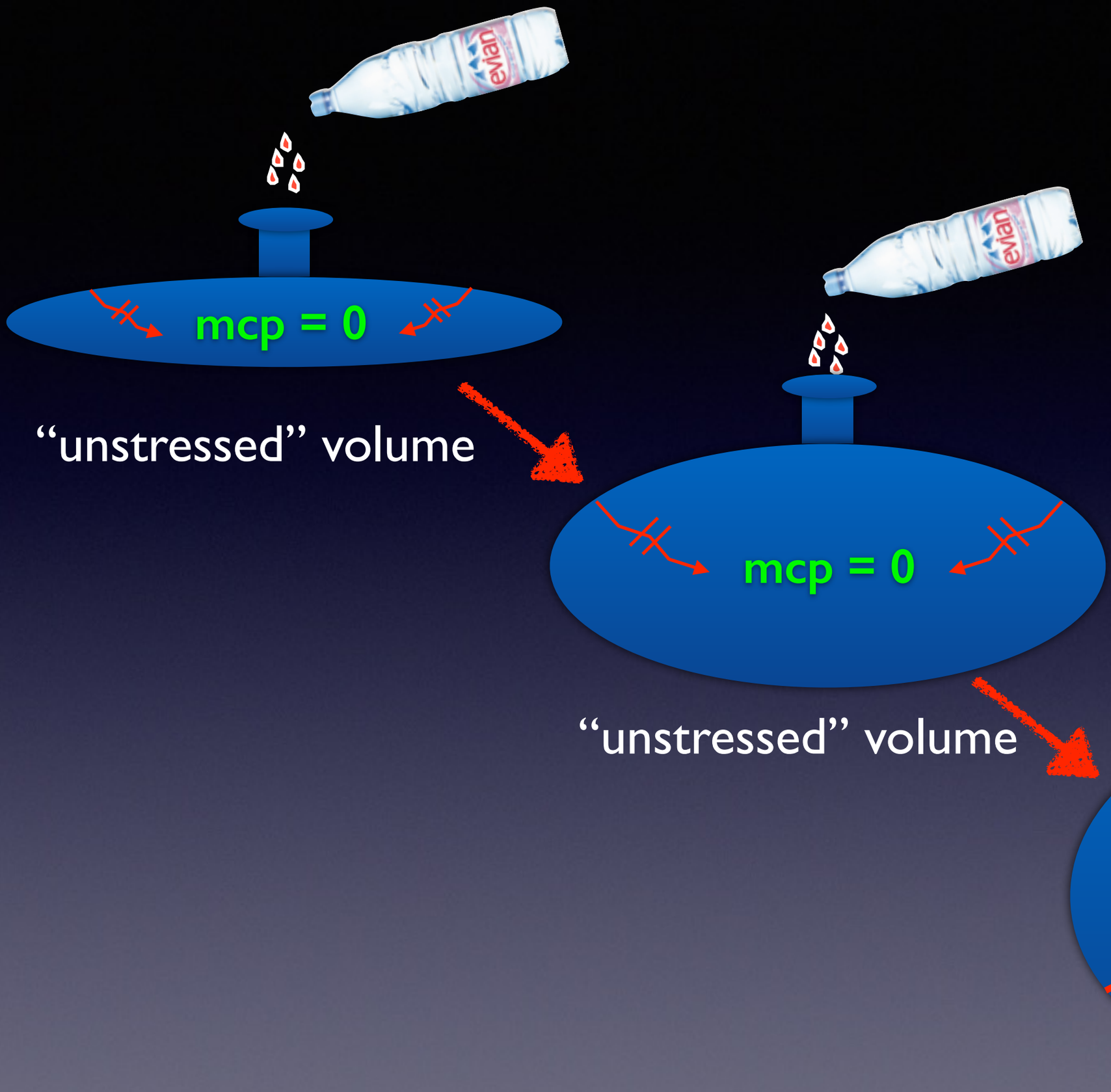
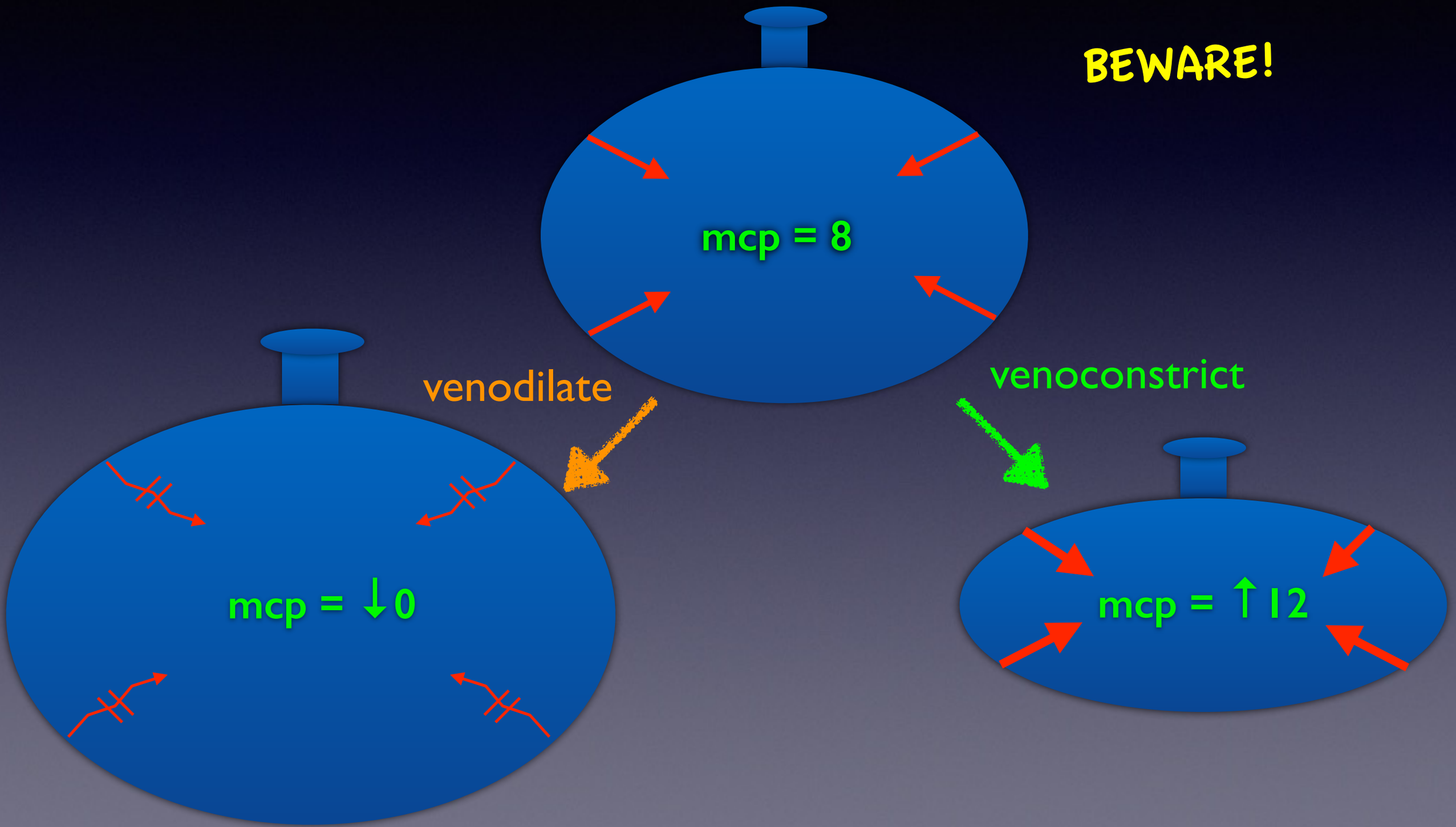

evian
mcp = 0
"unstressed" volume
evian
mcp = 0
"unstressed" volume
mcp = 8
"stressed" volume

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

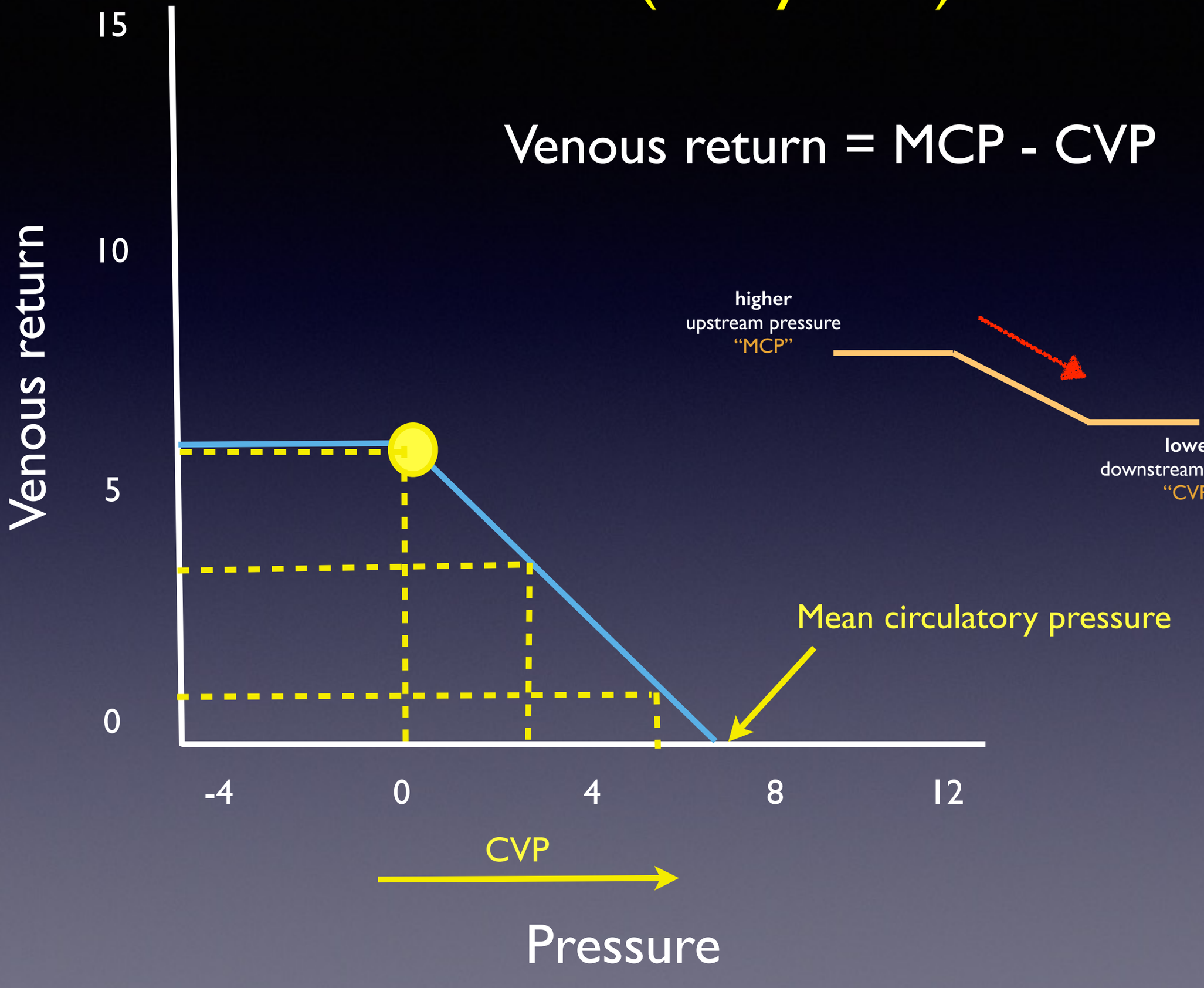
Opioids
GTN
Sedatives

BEWARE!

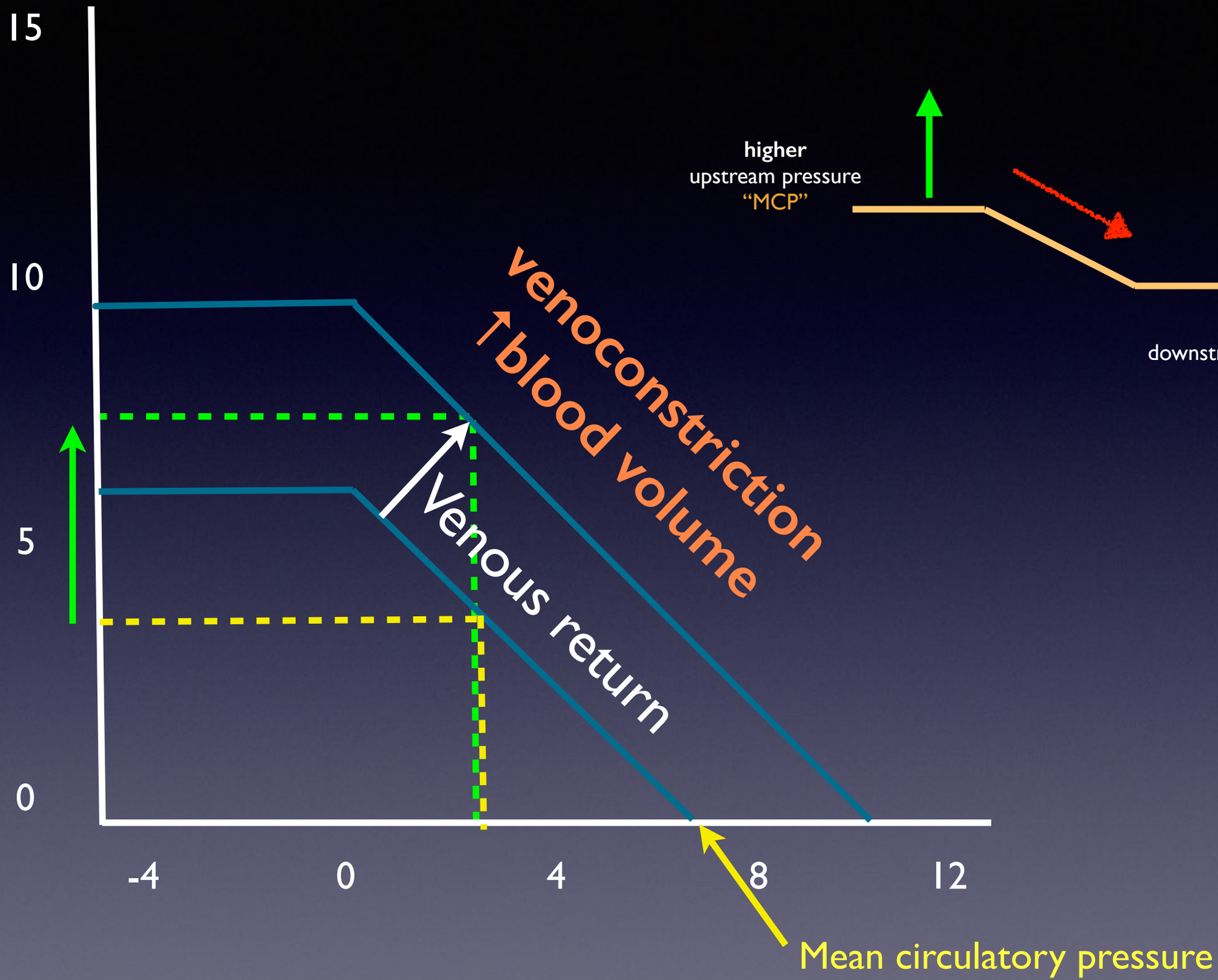


Venous return ("Guyton") curve

Venous return = MCP - CVP



Venous return curve



Starling meets Guyton

- The cardiac output and venous return axes are same

- Cardiac output and venous return must be **equal**

  Venous return = the Cardiac Output measured at the veins

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

  = where **CVP creates the same output and return**

RAP serves 2 functions

1. "Opposes" venous return.

   Each heart beat **lowers the RAP**, enabling venous return

   Intraluminal pressure relative to **atmospheric** pressure and **unaffected** by pleural pressure

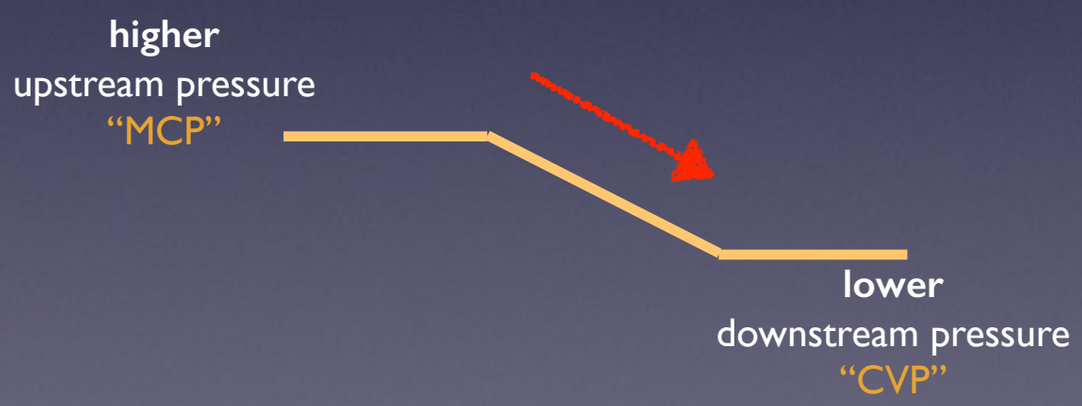
2. "Drives" the ventricle = Starling's law

   transmural pressure relative to **pleural** pressure thus **affected** by changes in pleural pressure, causing a shift of the cardiac function curve

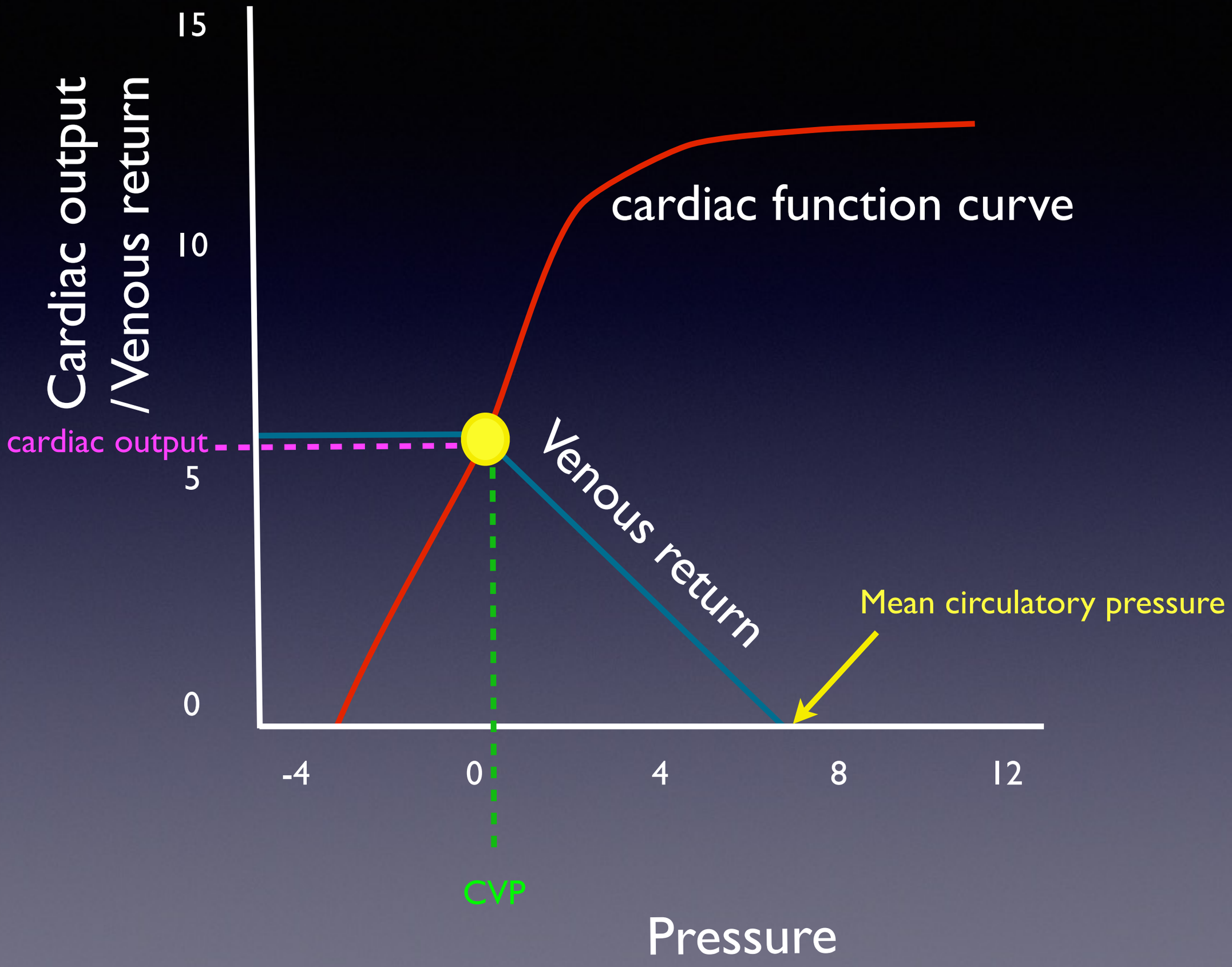
CVP serves 2 functions

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

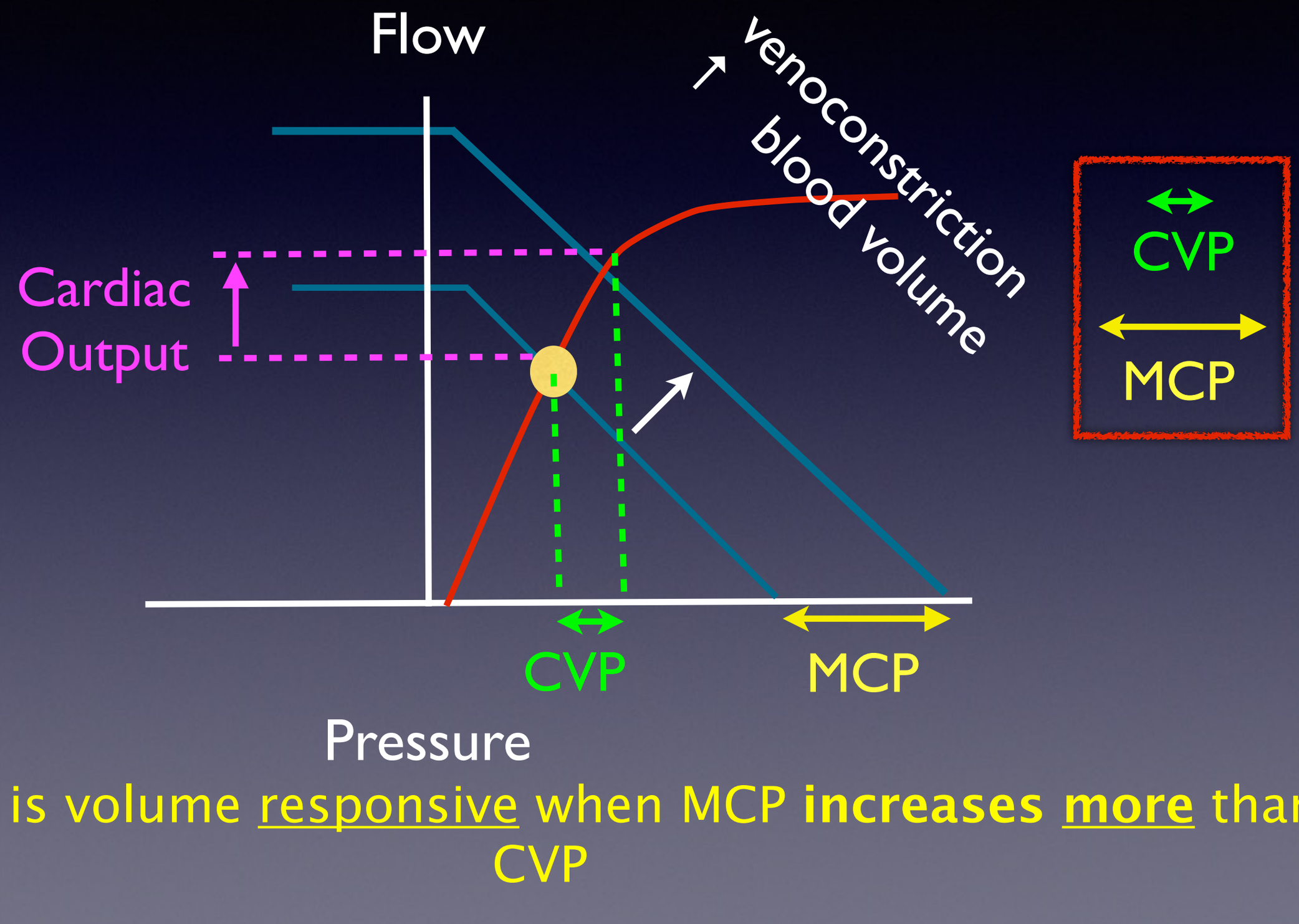
"Drives" the ventricle
(*Transmural* "Starling")



Starling meets Guyton

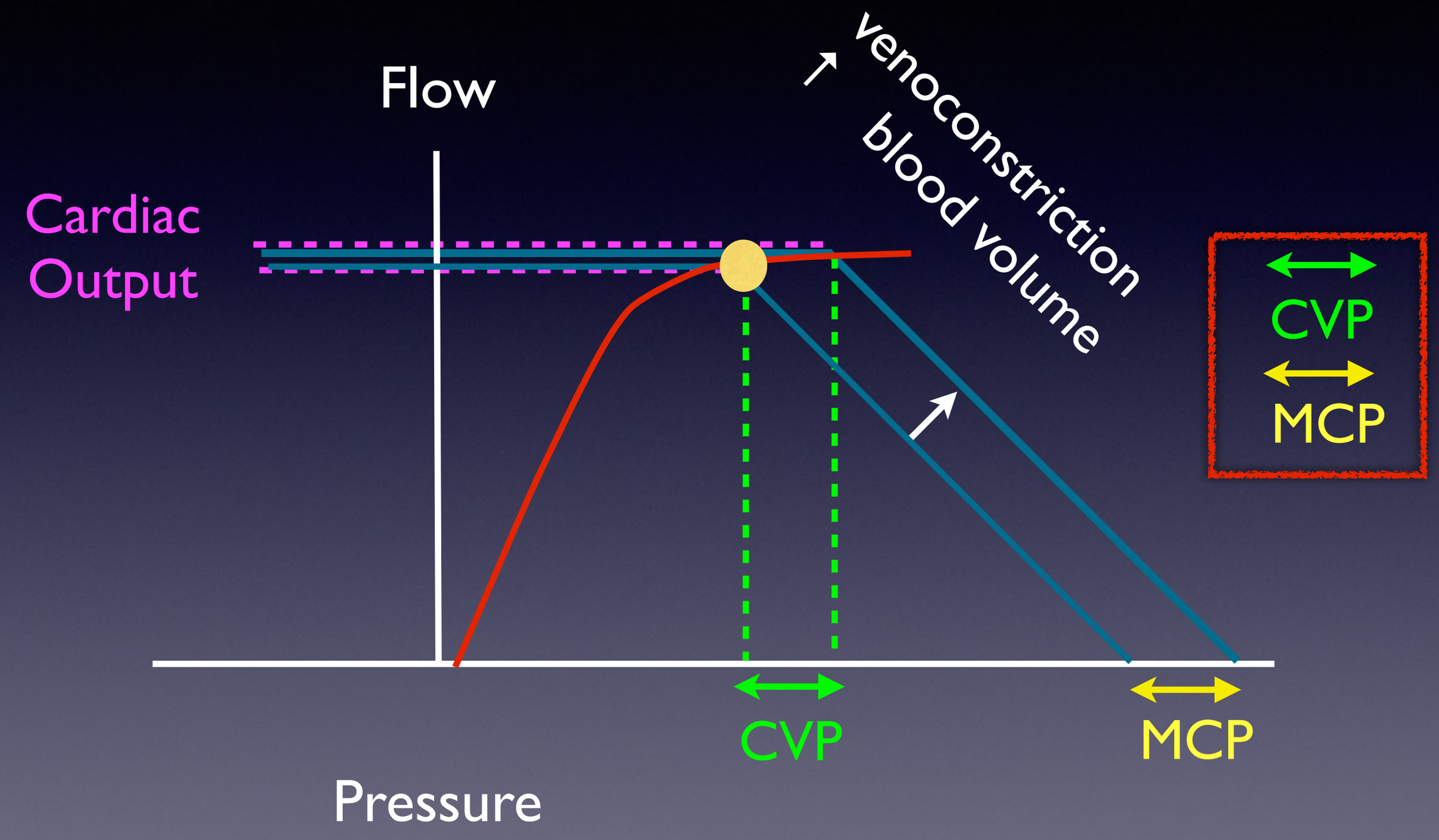


Increase in cardiac output by venoconstriction or increased blood volume



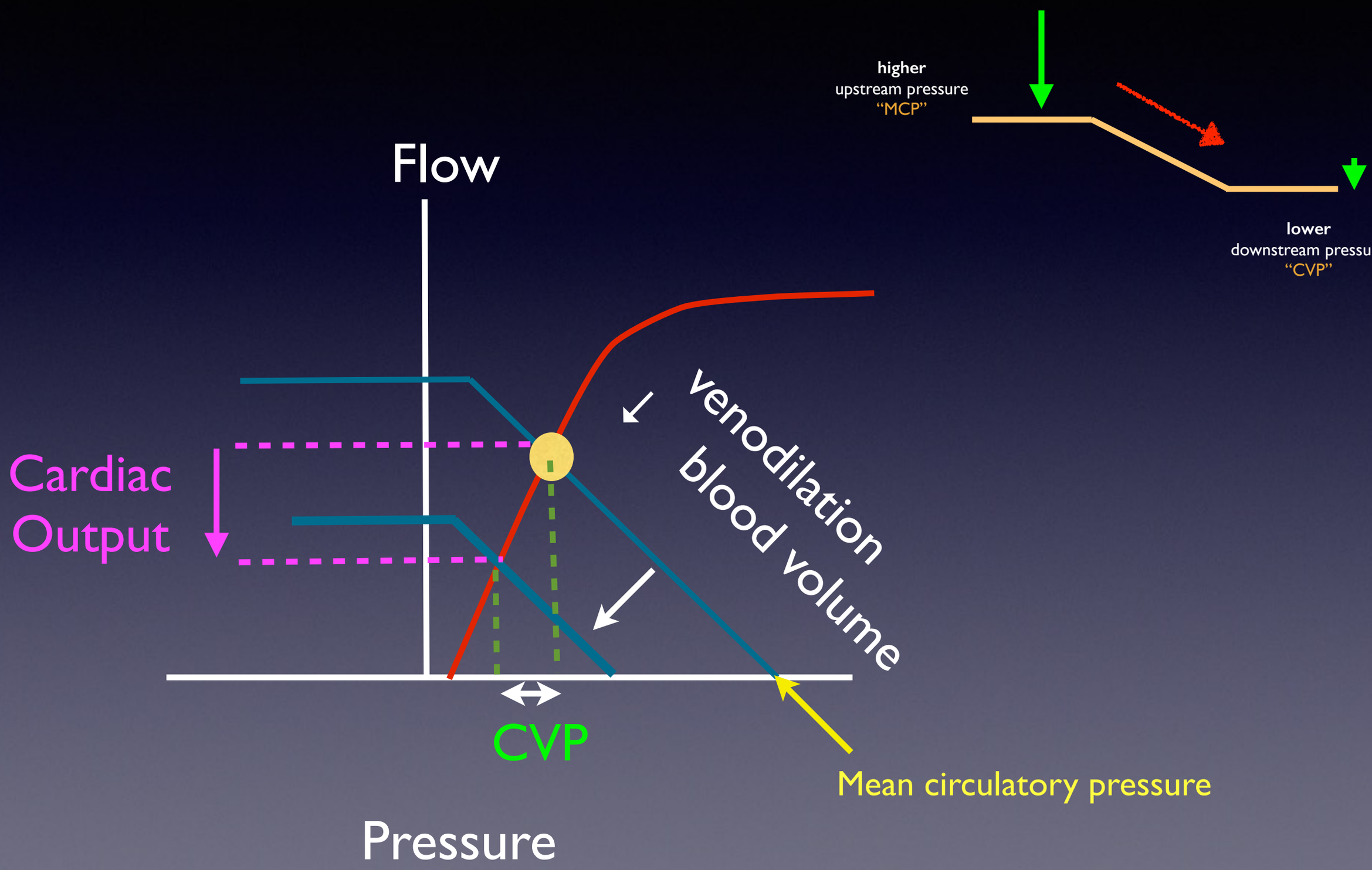
The heart is volume responsive when MCP **increases more** than CVP

No change in cardiac output by venoconstriction or increased blood volume



The heart is volume unresponsive when 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 with severe pneumonia

Hypoxic despite high Fi02

Lungs may be recruitable

Ventilated with PEEP

Paw = 28 cm H20

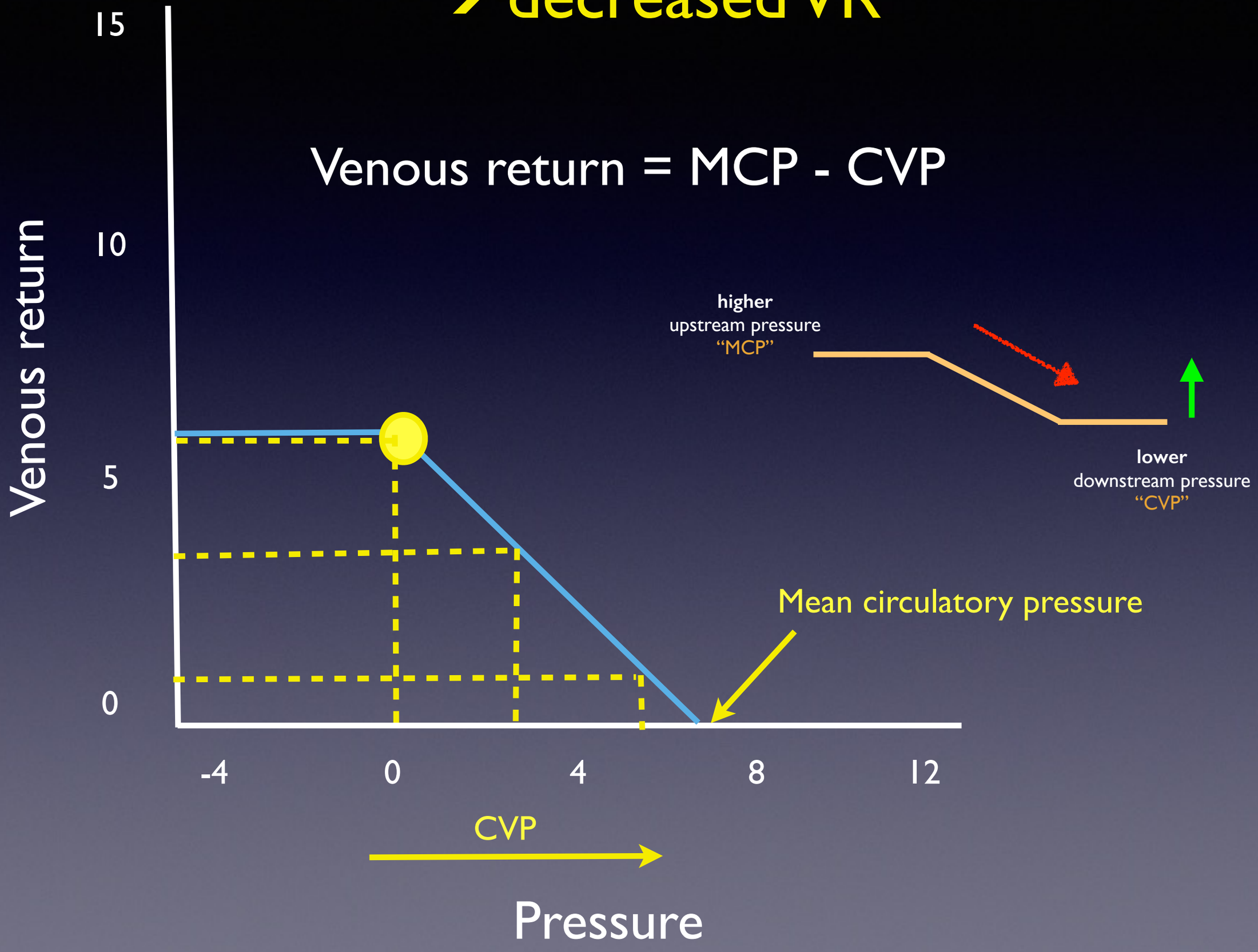
CVP → 18 mm Hg

BP drops

Why?

High airway pressure → high CVP → decreased VR

Venous return = MCP - CVP



Recap

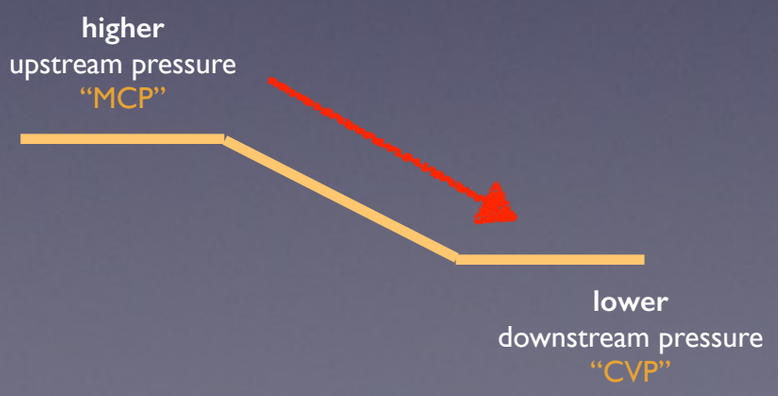
Double role of CVP

Inside chest

Determines cardiac "stretch" and C.O. (intra-thoracic pressure-CVP)
"Starling" curve
We don't measure intra-thoracic pressure
Clinical example: negative pressure pulmonary oedema

Outside chest

Determines venous return (mcp-CVP)
"Guyton" curve
We don't measure mcp pressure
Clinical example: cardiac tamponade



Recap

Think O2 Delivery

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

???

www.jvsmedicscorner.com

Mallory/Everest2013

Optimising Haemodynamics with Fluid

Part 2

Dr J Vogel FRCA

Summary of todays lecture

- Importance of cardiac output
- Physiology of cardiac output and venous return

<u>This lecture:</u>

- Optimising C.O. with fluids
  - What works and what doesn't

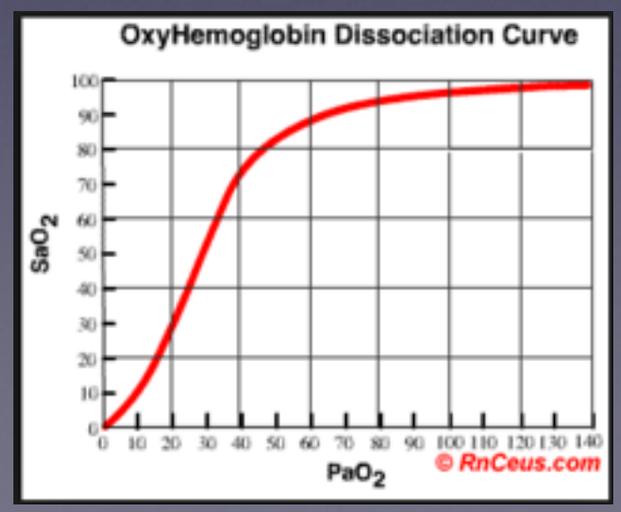
Why is cardiac output so important ?

Cardiac output x Hb x % Sat O2

Cardiac output the only parameter that:

- responds **rapidly**

- does **not plateau**



Recap

Consider:

- We can easily measure Hb and O2 Sat.
- The most important factors, **cardiac output**, and cardiac **preload**, are estimated clinically.

Today we will discuss how best to estimate optimizing filling

Cardiac output - what are we trying to achieve?

- Adequate "effective" cardiac output
- Adequate blood pressure (>65 mean)
- Adequate macro and micro-circulation

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

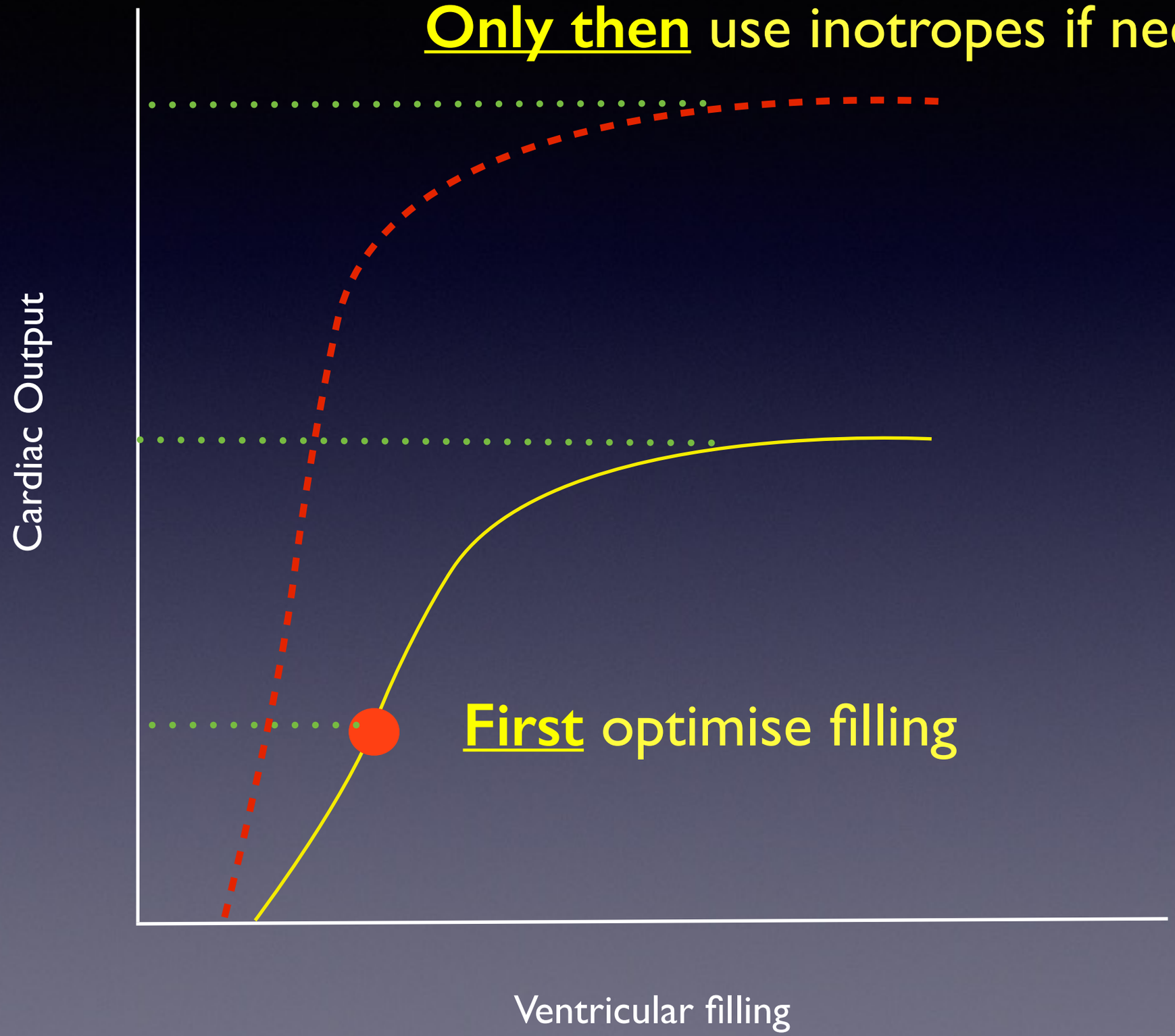
“Adequate” cardiac output?

- 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
  - “Visualizing” the micro-circulation

Recap

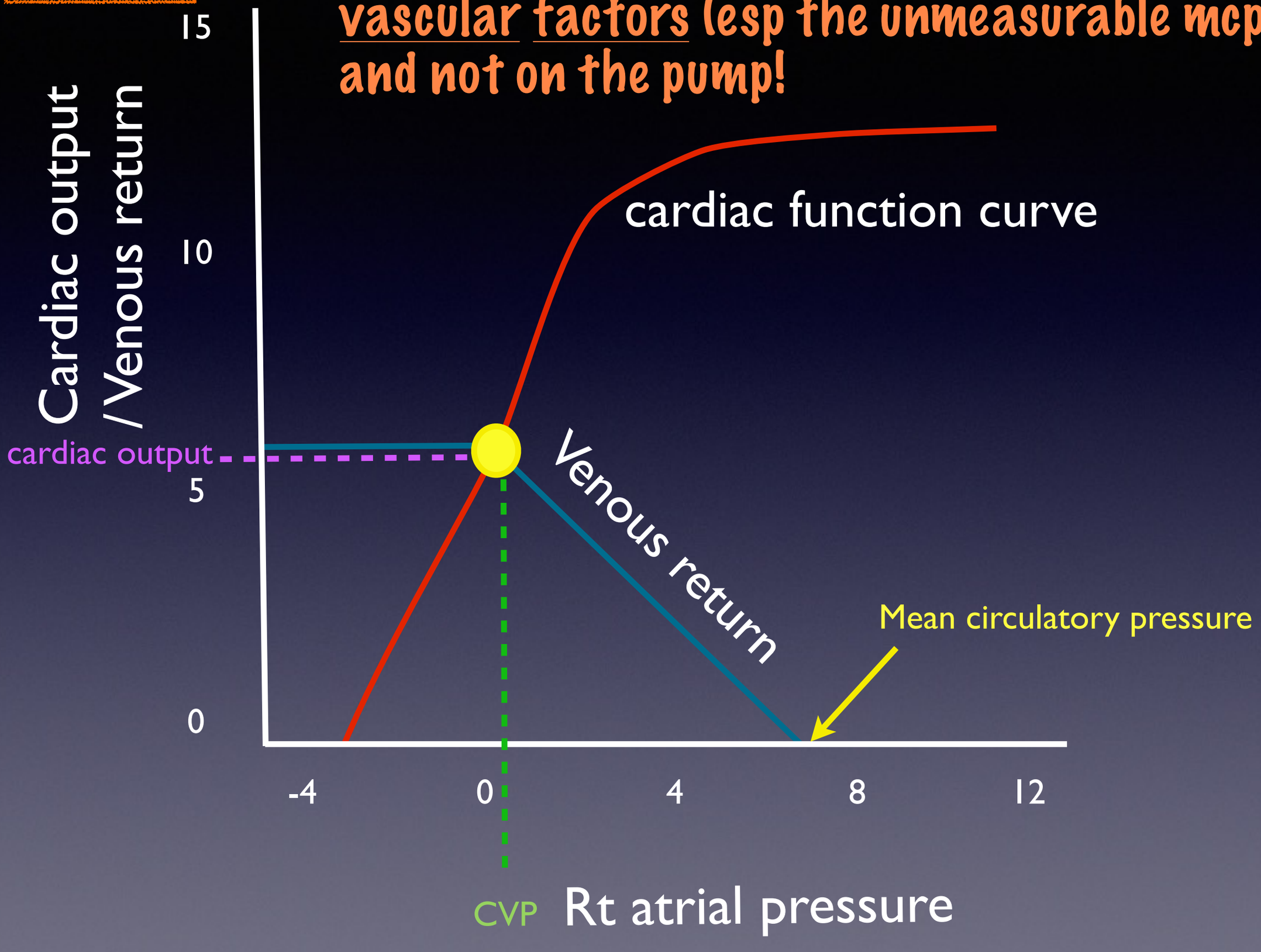
First step, optimise filling!

Only then use inotropes if needed



Recap

Venous return (ie cardiac filling) depends on vascular factors (esp the unmeasurable mcp) and not on the pump!



Recap

Starling

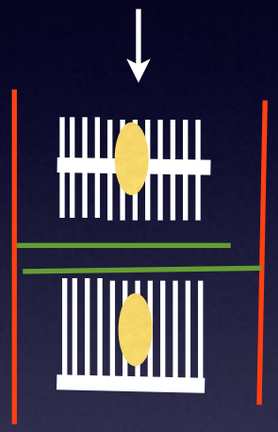
CVP

2 "functions"

Guyton

Transmural (rel. to pleural pressure)

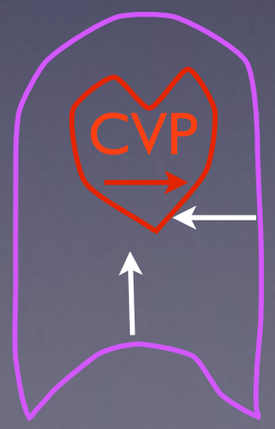
= "**distending pressure**"



Sarcomere

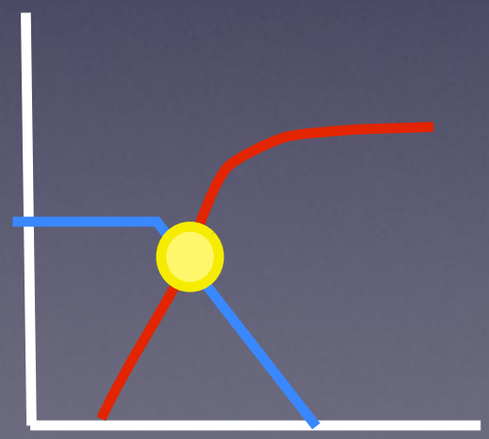
(Distension of ventricle leads to optimal sarcomere length)

Inside thorax



ex. PEEP

ex. Intra abdo pressure



Intramural (rel. to atmosphere)

= "**opposes venous return**"

Venous return depends on pressure gradient from extra-thoracic capillaries (**mcp**) to Rt heart

Outside thorax

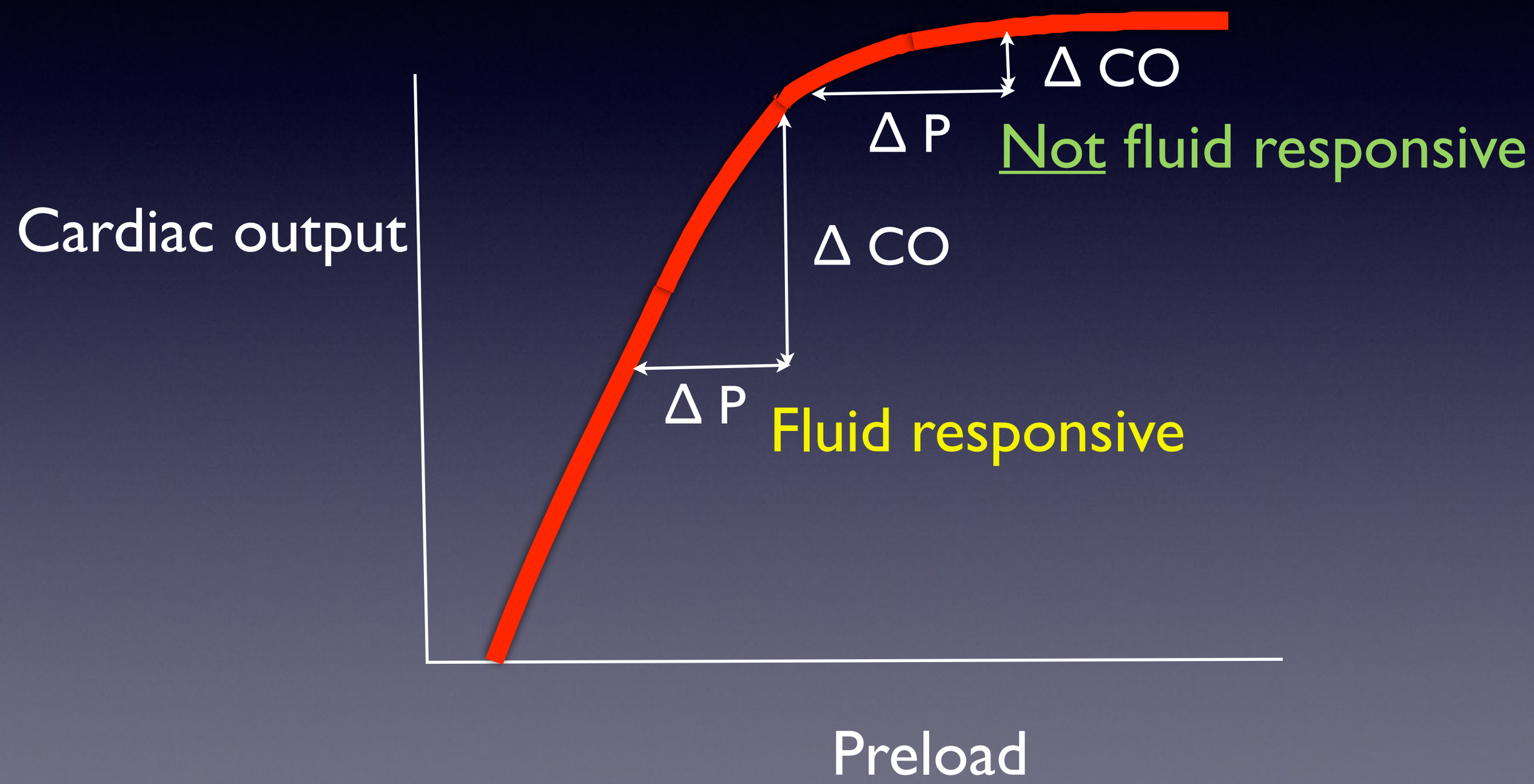
Venous system

Problems with assessing blood volume

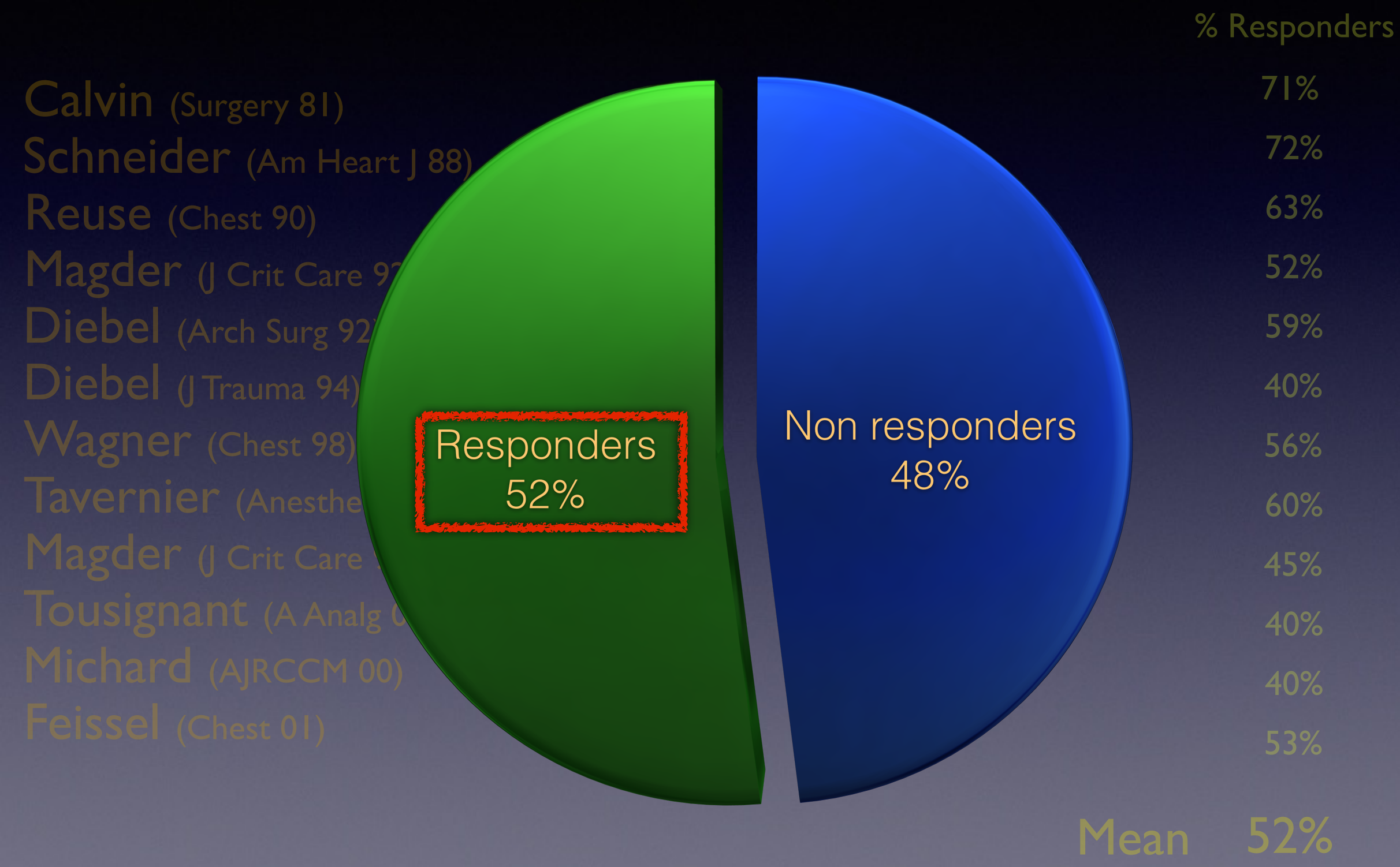
- We can't accurately evaluate blood volume
- We can't accurately identify fluid overload
- We can't accurately identify hypovolaemia
- We can't accurately evaluate tissue hypoperfusion

What we want to know is will CO improve by giving fluids?
Not is this patient's volume status, but are they
fluid responsive?

Fluid responsiveness
= where is patient on the Starling curve?



Half of ITU patients are fluid responders



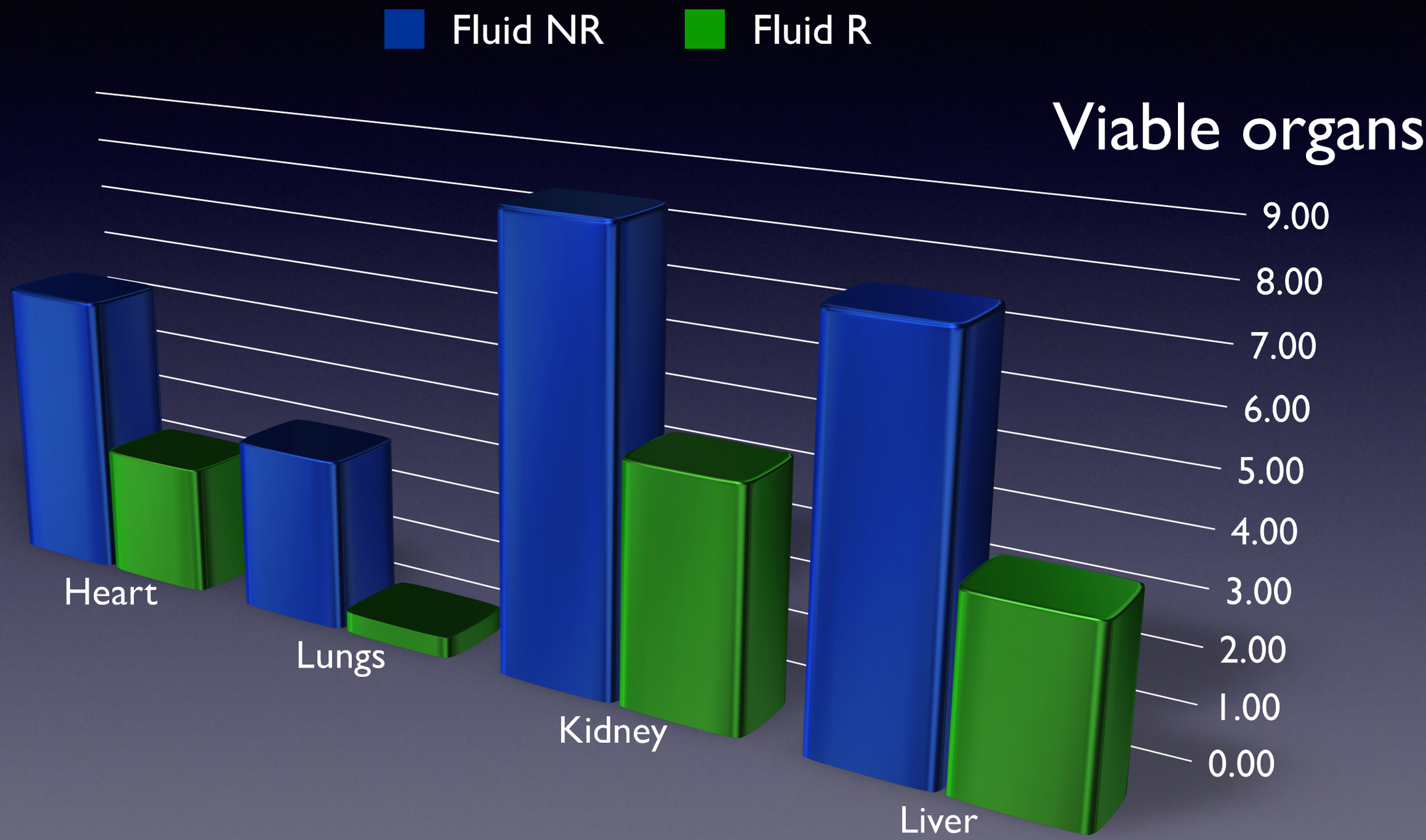
Michard & Teboul. Chest 121:2000-8, 2002

Predicting fluid responsiveness

Why predict?

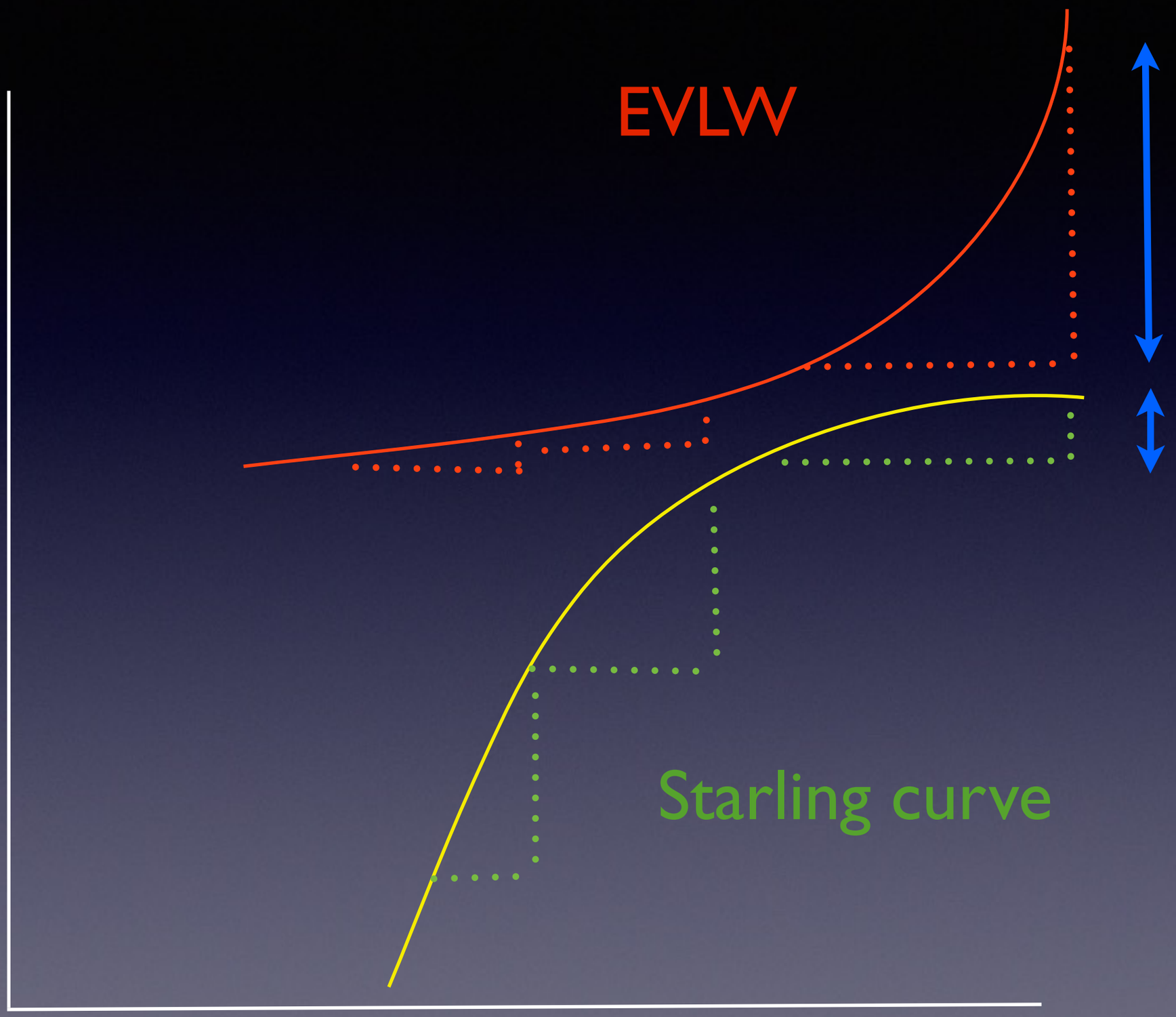
How to predict?

Fluid responsiveness is associated with lower organ yield from brain-dead donors



Murugan et al. Crit Care Med 2009

Why try <u>predicting</u> fluid responsiveness?



How to tell if fluid responsive

Either

- Give a bolus and watch response

  - Probably safe if small volumes required

  - If no risk of pulmonary oedema

Or

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

Predicting fluid responsiveness

How to predict fluid responsiveness

1. **Clinical**
   - Orthostatic response
2. **Static** measures
   - ~~CVP~~
   - ~~ECHO~~
   - ~~GEDI~~

Static measures do not work

3. **Dynamic** measures
   - Heart-lung interactions

J Intensive Care Med 2009;24:329-337

Critical Care Med 2000;4:282-289

Clinical Case

Patient is 2nd day post knee replacement

Vital signs are normal

Physios try and mobilise for the first time

Patient faints and re-fractures femur

Why?

Clinical Signs

Accuracy of Vital Signs in the Detection of Blood Loss

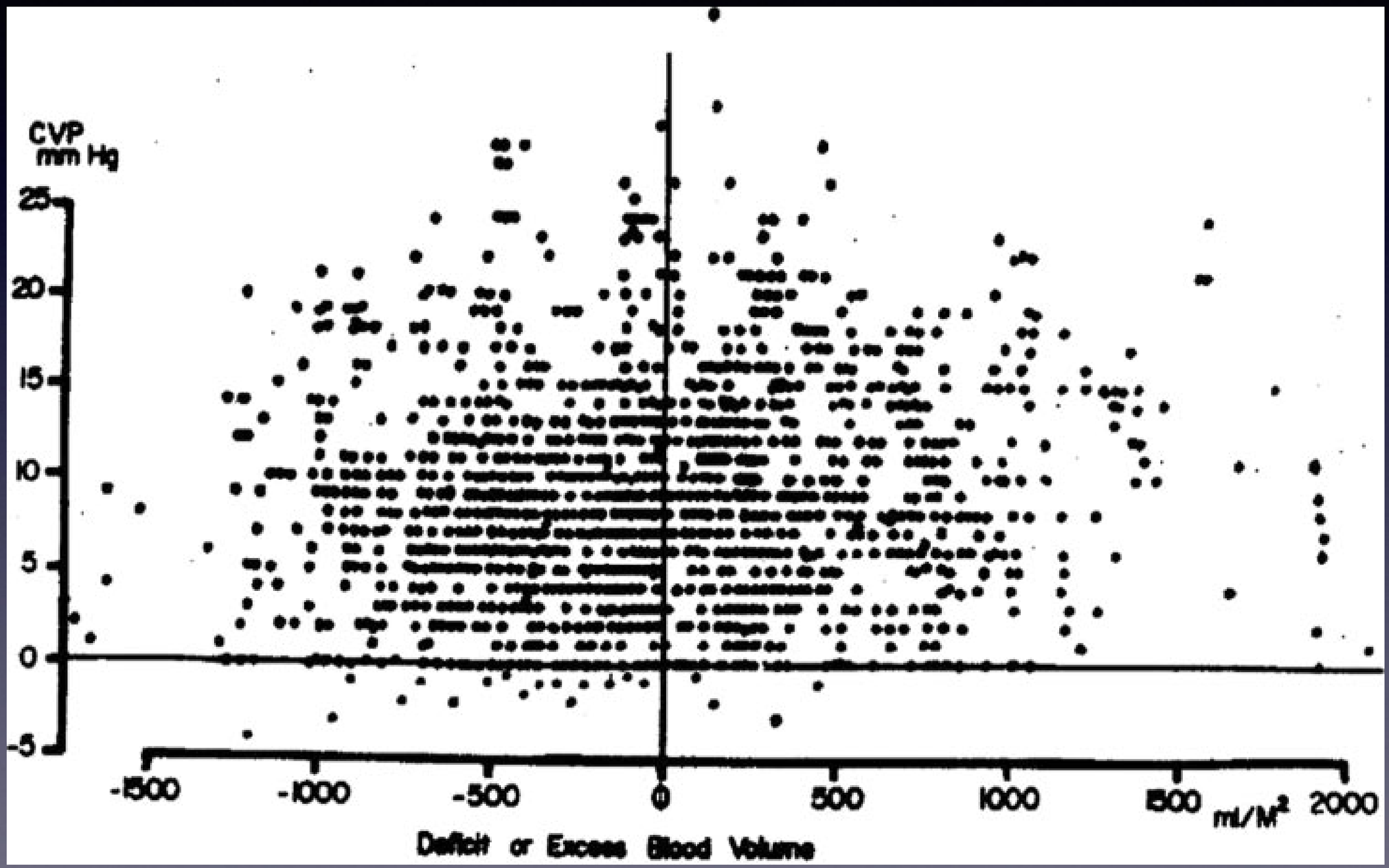
Parameter	Moderate Blood Loss (450-630 mL)	Severe Blood Loss (630-1150 mL)
Supine tachycardia	0-42%	5-24%
Supine hypotension	0-50% ← Flipping a coin Is better →	21-47%
Postural pulse increment or Postural dizziness	6-48%	91-100%

JAMA 1999;281:1022-1029

Static measures

CVP

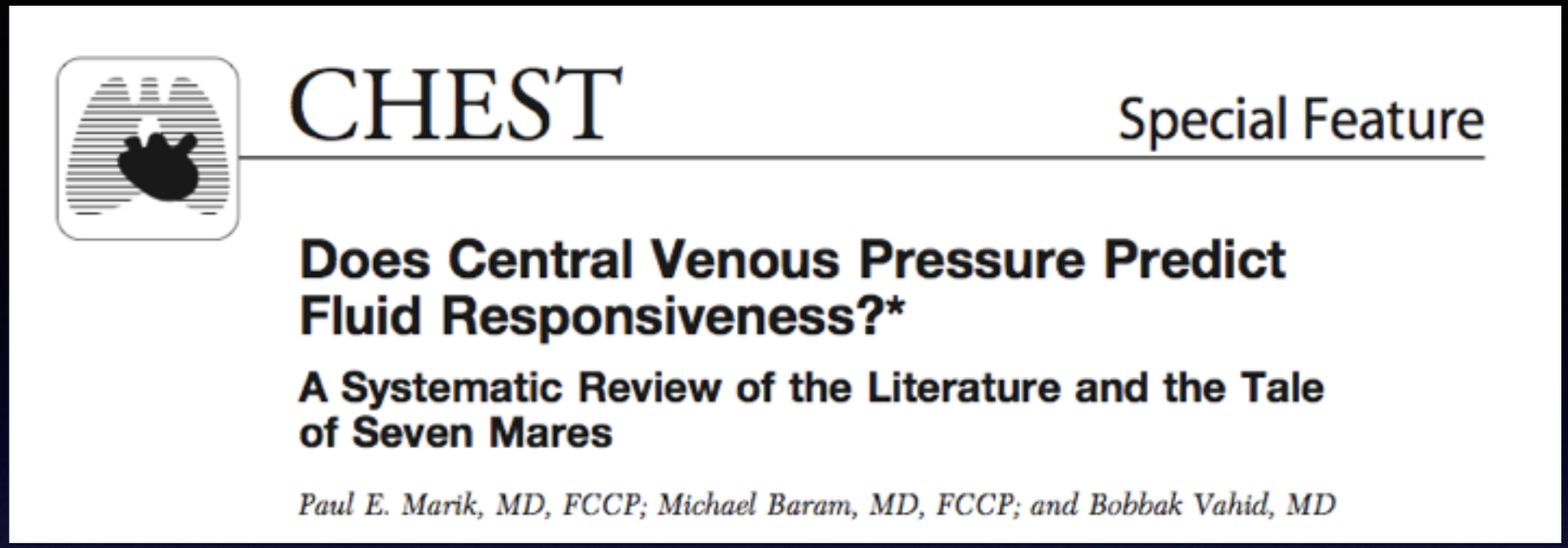
No correlation to measured blood volume !



Crit Care Med 1984; 12:107–112

(r=0.27)

Static measures



Conclusions: This systematic review (24 studies) demonstrated a very poor relationship between CVP and blood volume as well as the inability of CVP/ΔCVP to predict the hemodynamic response to a fluid challenge.

"CVP should not be used to make clinical decisions regarding fluid management."

Chest 2008; 134:172-178

Static measures

British Journal of Anaesthesia **94** (3): 318–23 (2005)
doi:10.1093/bja/aei043 Advance Access publication December 10, 2004

BJA

Assessing fluid responsiveness during open chest conditions

D. A. Reuter[1]*, M. S. G. Goepfert[1], T. Goresch[1], M. Schmoeckel[2], E. Kilger[1] and A. E. Goetz[1]

"**No correlation** between values of global end-diastolic volume (GEDI) nor left ventricular end-diastolic area (ECHO) and response to fluid loading."

Preload is not the same as **preload responsiveness**!

Dynamic measures

Heart-lung interactions

1. Spontaneous ventilation
   - **Drop in CVP**
2. Ventilated patient
   - **CV Variation with inspiration**
     - ❖Pulse pressure variation
     - ❖Stroke volume variation
     - ❖Systolic pressure variation
     - ❖Pulse oximeter variation

Clinical Case

Patient is 1st day postop

BP 125/80; HR 90/min

Spontaneous respiratory rate 20/min

Nurse tells you his urine output is poor

Still has a central line in place

What do you do?

Dynamic measures

Drop in CVP on inspiration?

Spontaneous breathing ->
(**negative** pleural pressure)
-> Increase transmural pressure
of heart

Room to stretch?
is the heart compliant?

Not fluid responsive

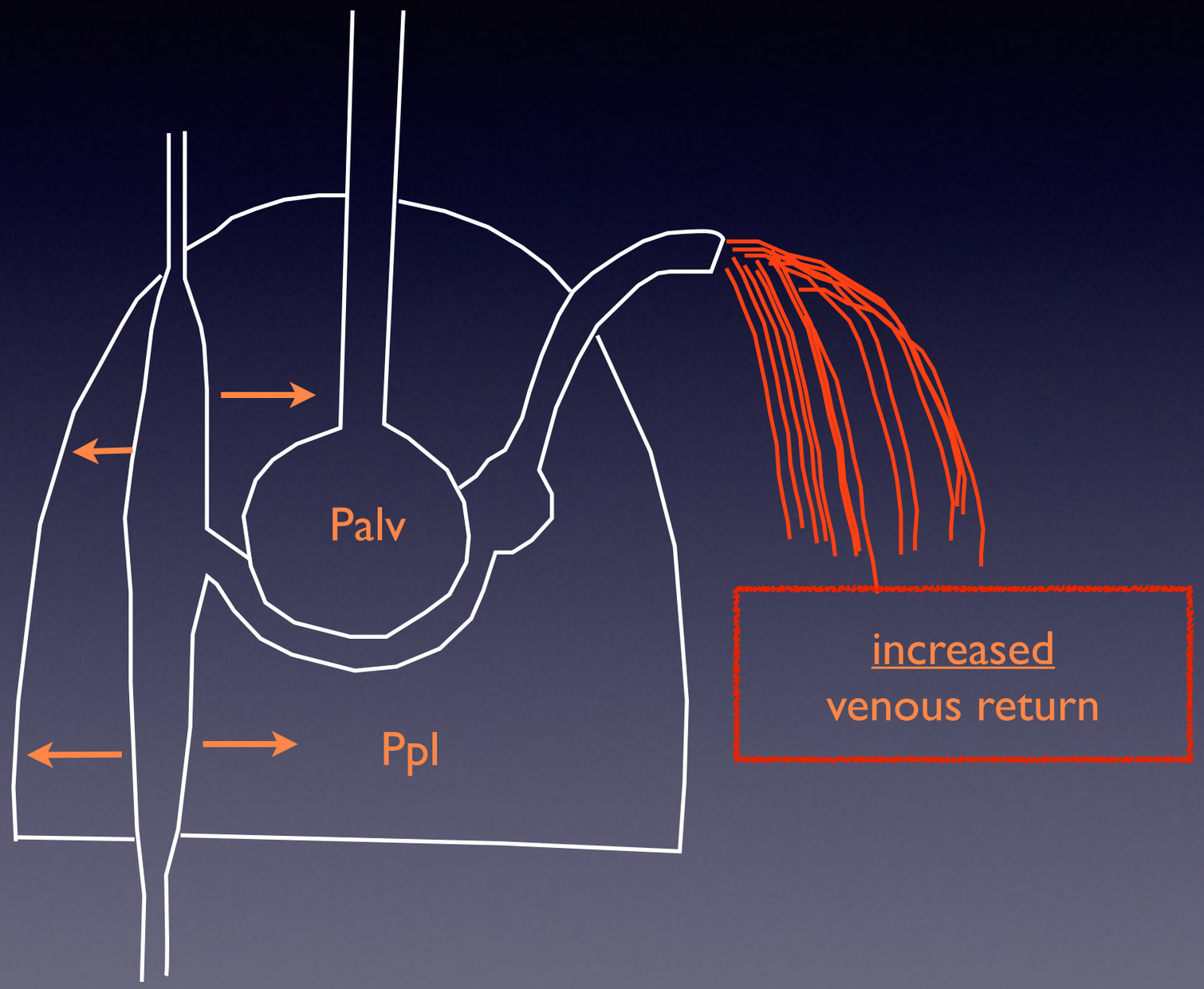
Changes in pleural pressure are **not** transmitted to heart.
Heart maximally full.
no fall in CVP
and no change in CO

Fluid responsive

Changes will result in an increase in volume (a bolus of blood) which the heart can pump out.
fall in CVP
and increase in CO

Spontaneous ventilation

the pleural pressure drops
-> the vena cavae expand (if **compliant**)
-> the CVP drops
-> sucking more blood into the chest
->venous return and cardiac output increased

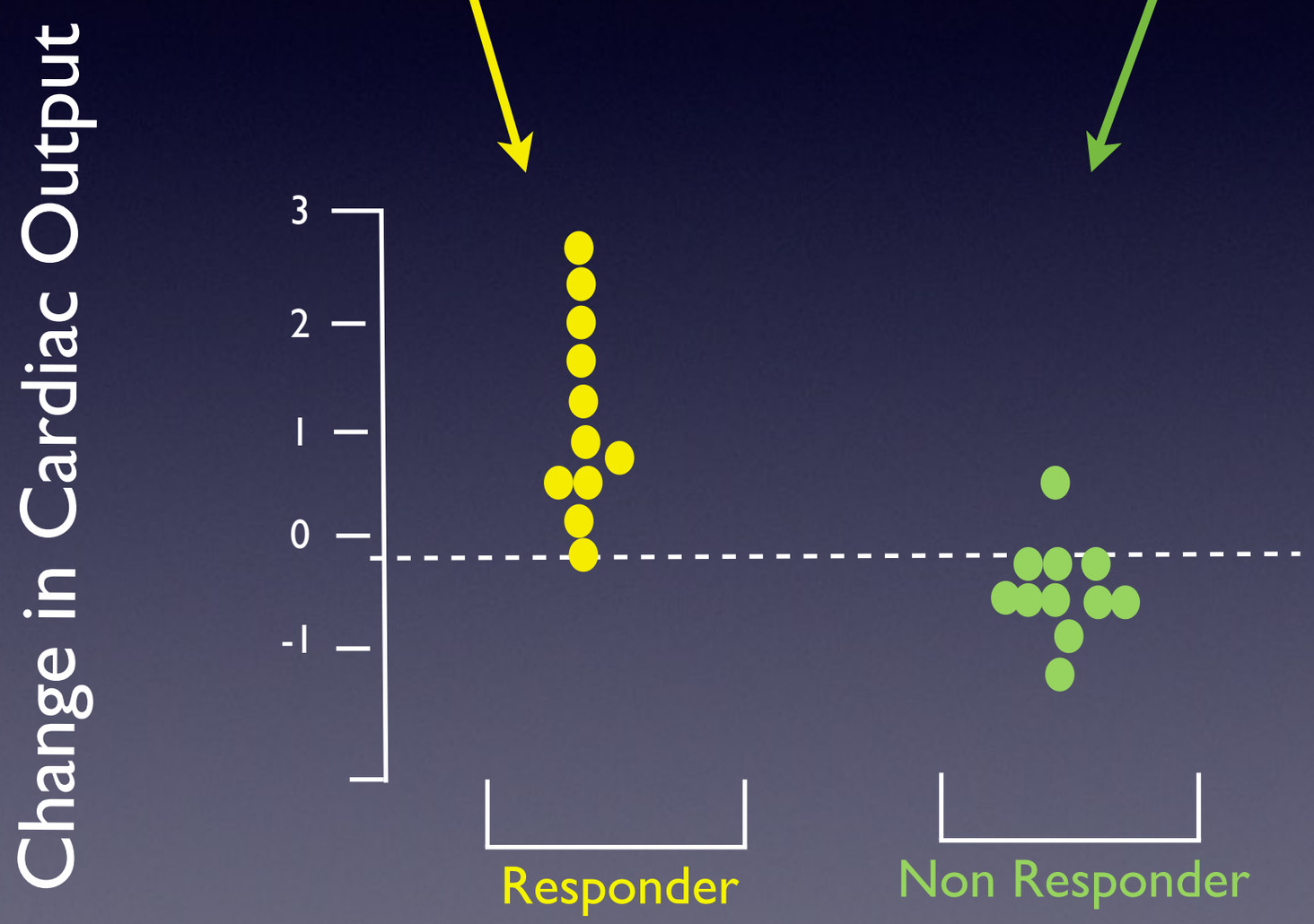


CVP decreases

Change in CVP and spontaneous ventilation - Does it work ?

Inspiratory fall in CVP

No inspiratory fall in CVP



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

Clinical Case

66 yr old woman

PMH:

DM, IHD, Ch Renal impairment (creatinine 117);

RVF, severe tricuspid regurg

CT with contrast 2 days prior

BP+HR-OK; CVP 31

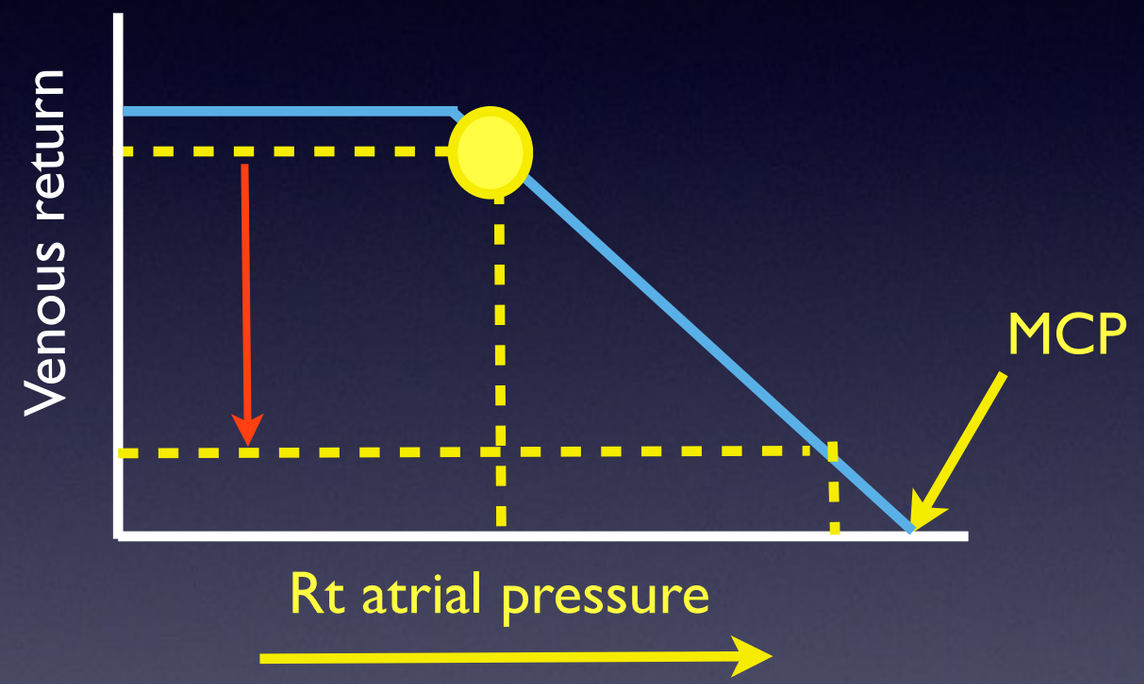
Urine output is dropping

What is going on?

What do you do?

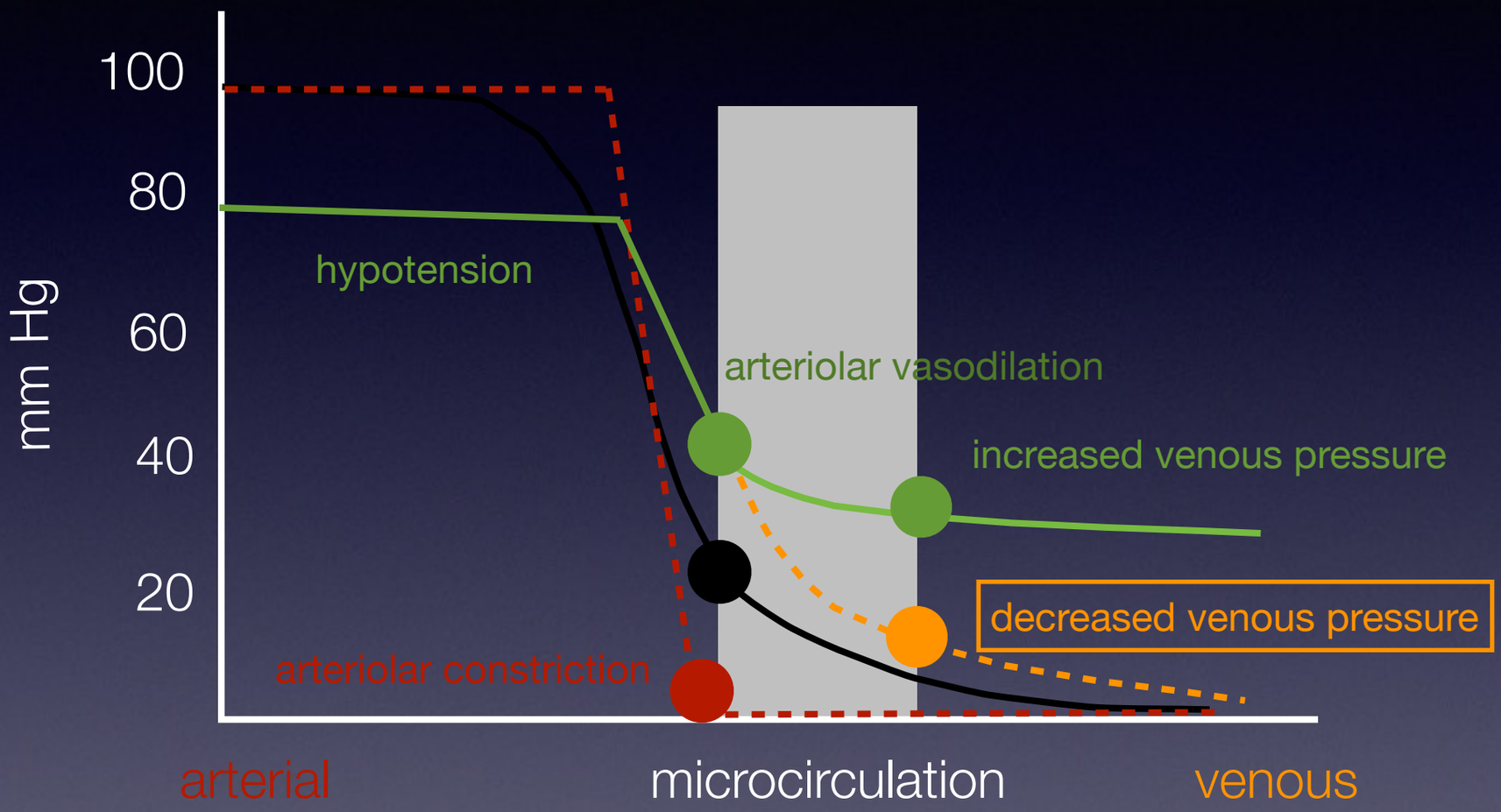
If <u>extreme</u>, high venous pressures do have negative "upstream" consequences

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



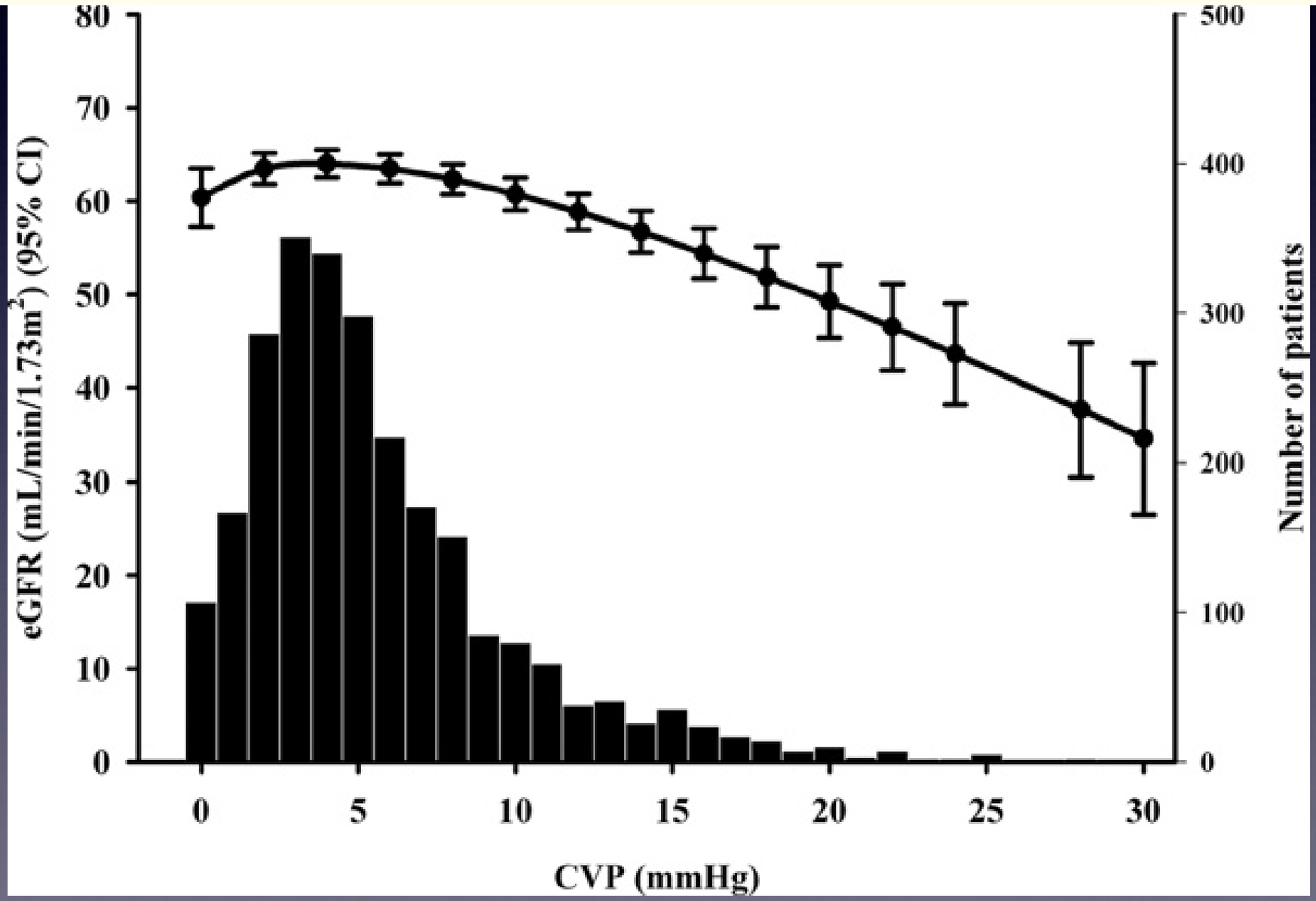
Use CVP to measure "safe limit" when fluid resuscitating

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



Use CVP to measure "safe limit" when fluid resuscitating

Increased Central Venous Pressure Is Associated With Impaired Renal Function and Mortality in a Broad Spectrum of Patients With Cardiovascular Disease



J Am Coll Cardiol 2009;53 582-6

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

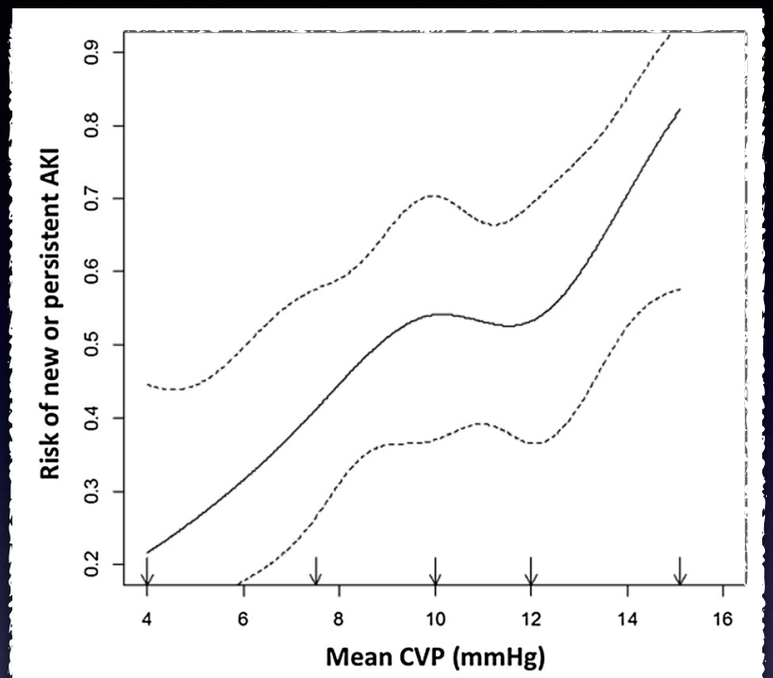


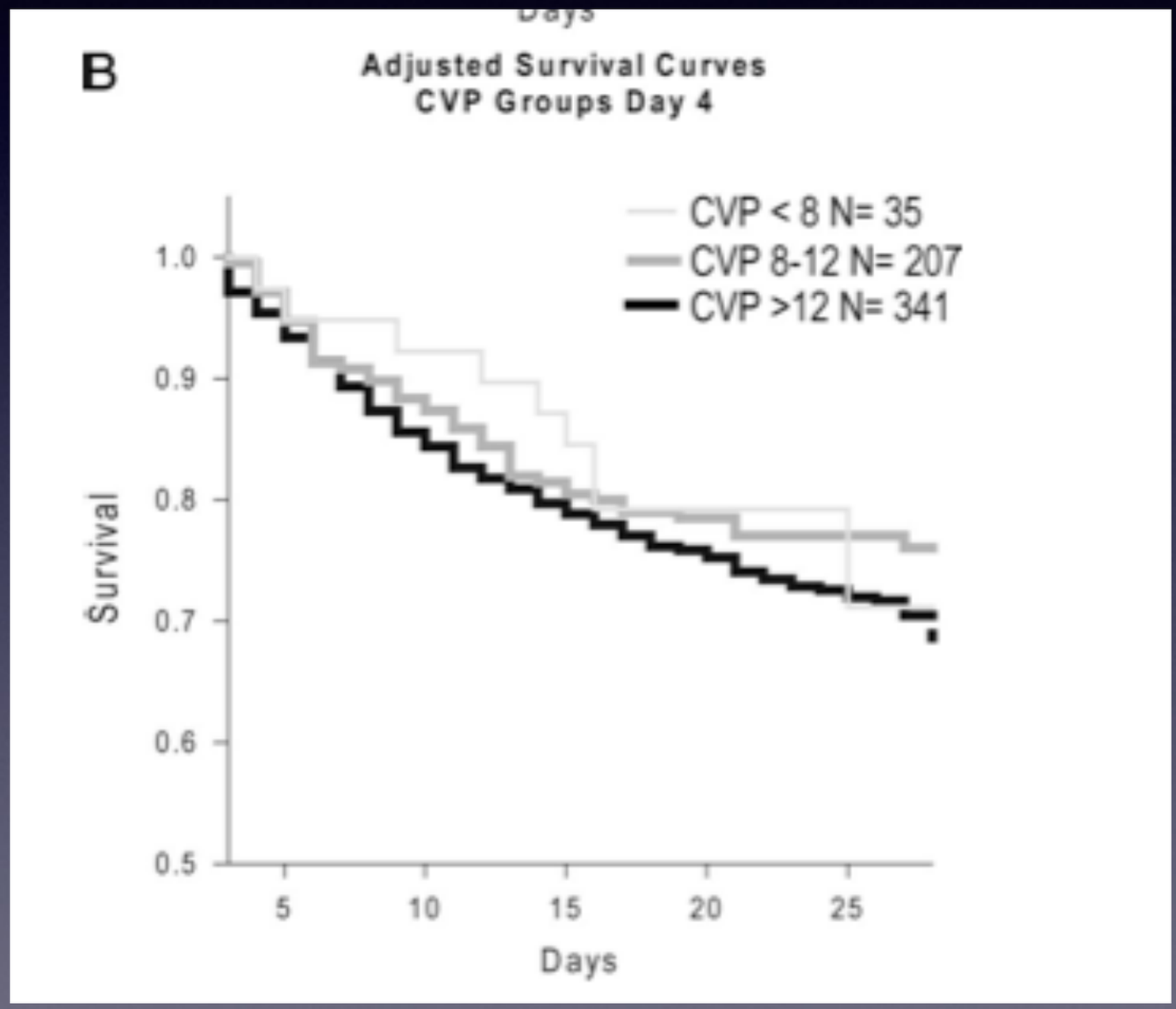
Figure 2 Statistical model of nonparametric logistic regression showing the relationship between mean central venous pressure during the first 24 hours after admission and the probability of new or persistent acute kidney injury. Note the plateau for the incidence of acute kidney injury (AKI) when the lower limit of central venous pressure (CVP) was between 8 and 12 mmHg. Over this limit, the rise in CVP was associated with a sharp increase in new or persistent AKI incidence.

"…**association between CVP and AKI** remained when potentially confounding effects of positive fluid balance and higher positive end-expiratory pressure were accounted … a **5 mmHg increase in CVP predicted 2.7-fold odds** of new or persistent AKI."

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

Critical Care 2014, 18:104

Fluid resuscitation in septic shock: A positive fluid balance and elevated central venous pressure are associated with increased mortality*



Crit Care Med 2011 Vol. 39, No. 2

If you do measure CVP, do it correctly !

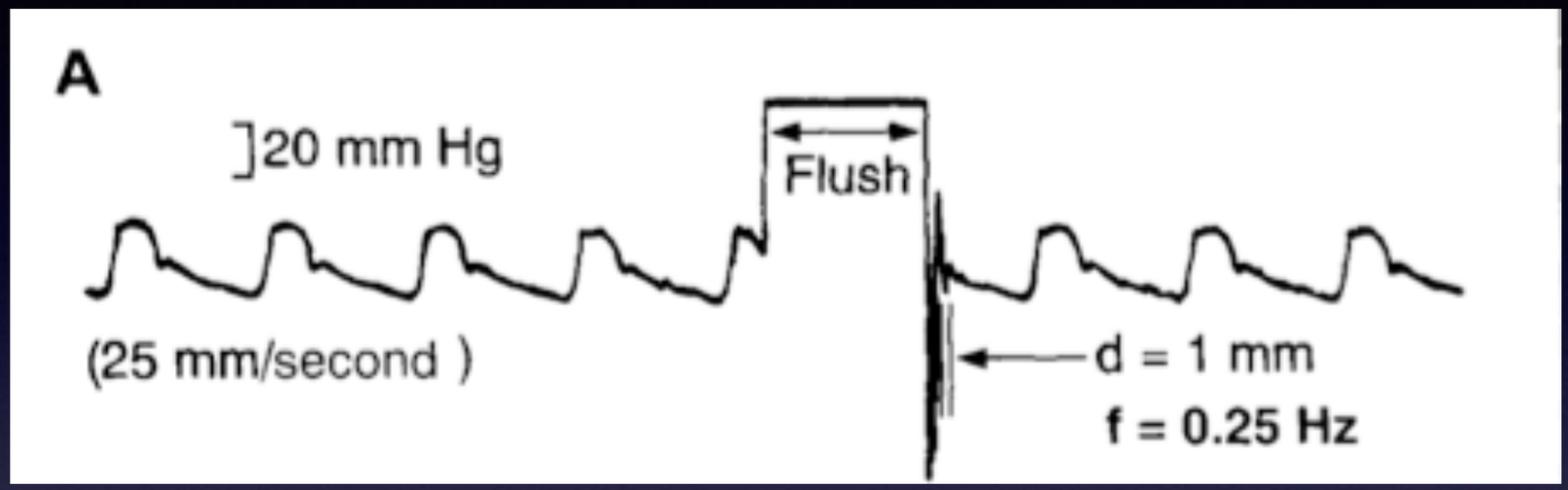
Reference point

1. **Sternal angle**
   Mid point of right atrium is 5 cm vertically below
   True whether person is supine or sitting erect (up to 60o)

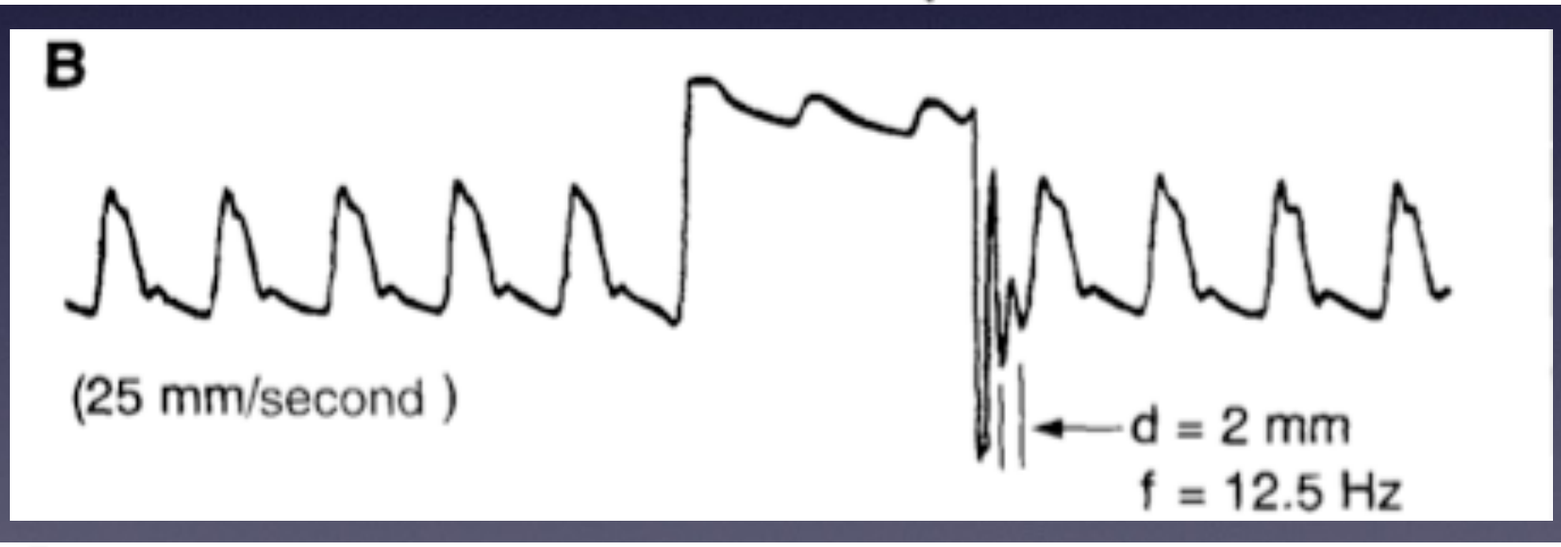
2. **Mid axillary line**
   use only if supine
   on average 3 mm Hg higher than sternal angle

Always test your measuring system

Flush test



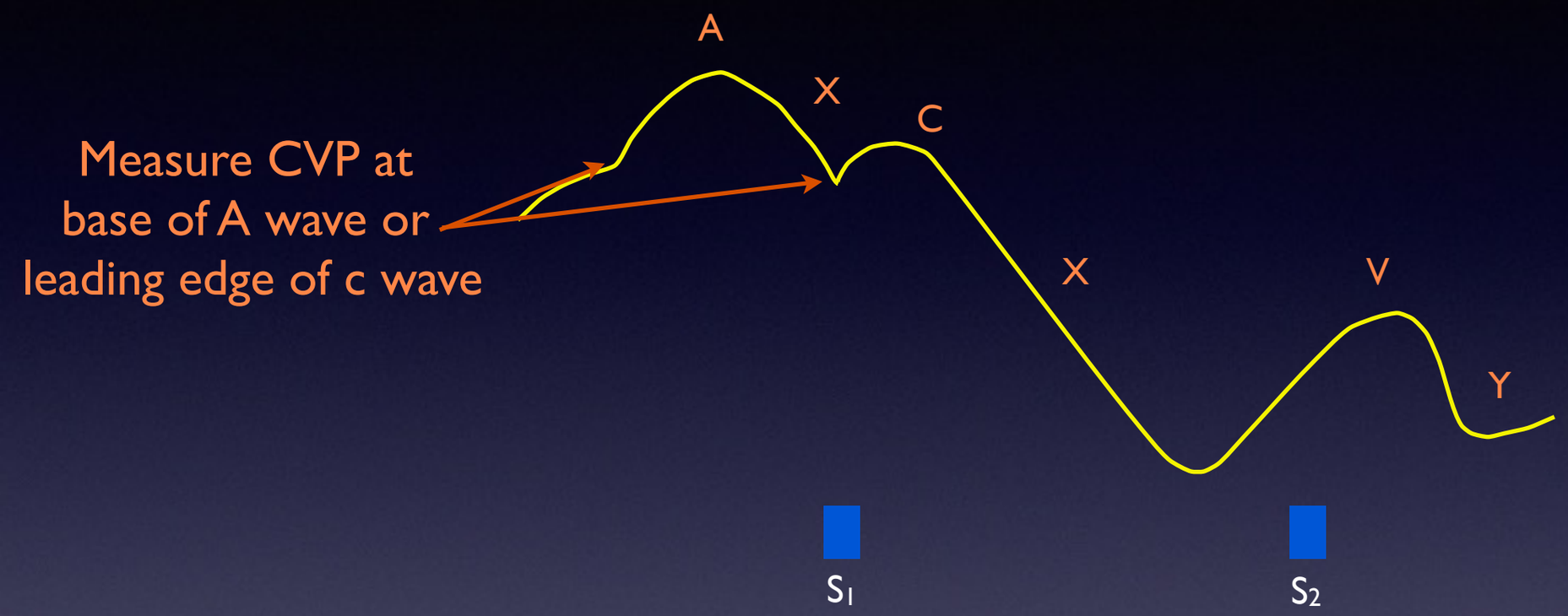
Normal



Under damped

Over damped

The CVP waves give valuable information



A wave = atrial contraction

X descent = atrial relaxation

C wave = pushing up of tricuspid valve on ventricular systole

V wave = atrial filling during systole

Y descent =sudden decrease in atrial pressure at onset of vent. diastole

Clinical Case

78 yr old male

Atrial fibrillation

In HDU post hemi-colectomy

CVP reads 34 mmHg on monitor

What do you do?

Clinical Case

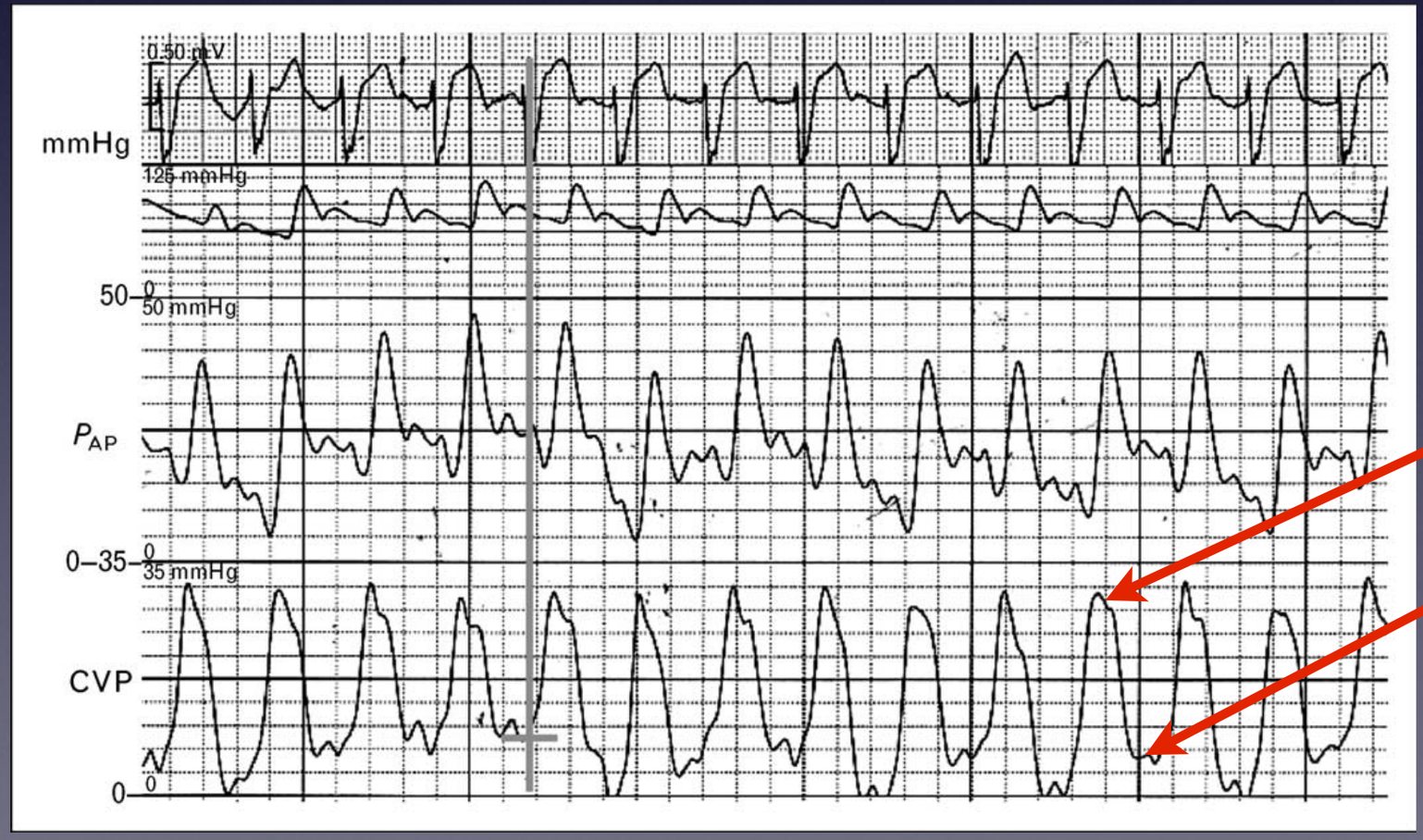
Look at the waveform!

Why the large "a" wave ?

CVP measured from base of "c" was 10 mm Hg

ECHO confirmed diagnosis of tricuspid regurgitation

After GTN, CVP read 10 mm Hg from screen



Dynamic measures

Cardiovascular response to positive pressure ventilation can predict fluid responsiveness

Clinical Case

Patient is in anaesthetic room

Will undergo an emergency laparotomy

Has been deemed adequately fluid resuscitated

Vitals OK

Rapid sequence induction of anaesthesia with

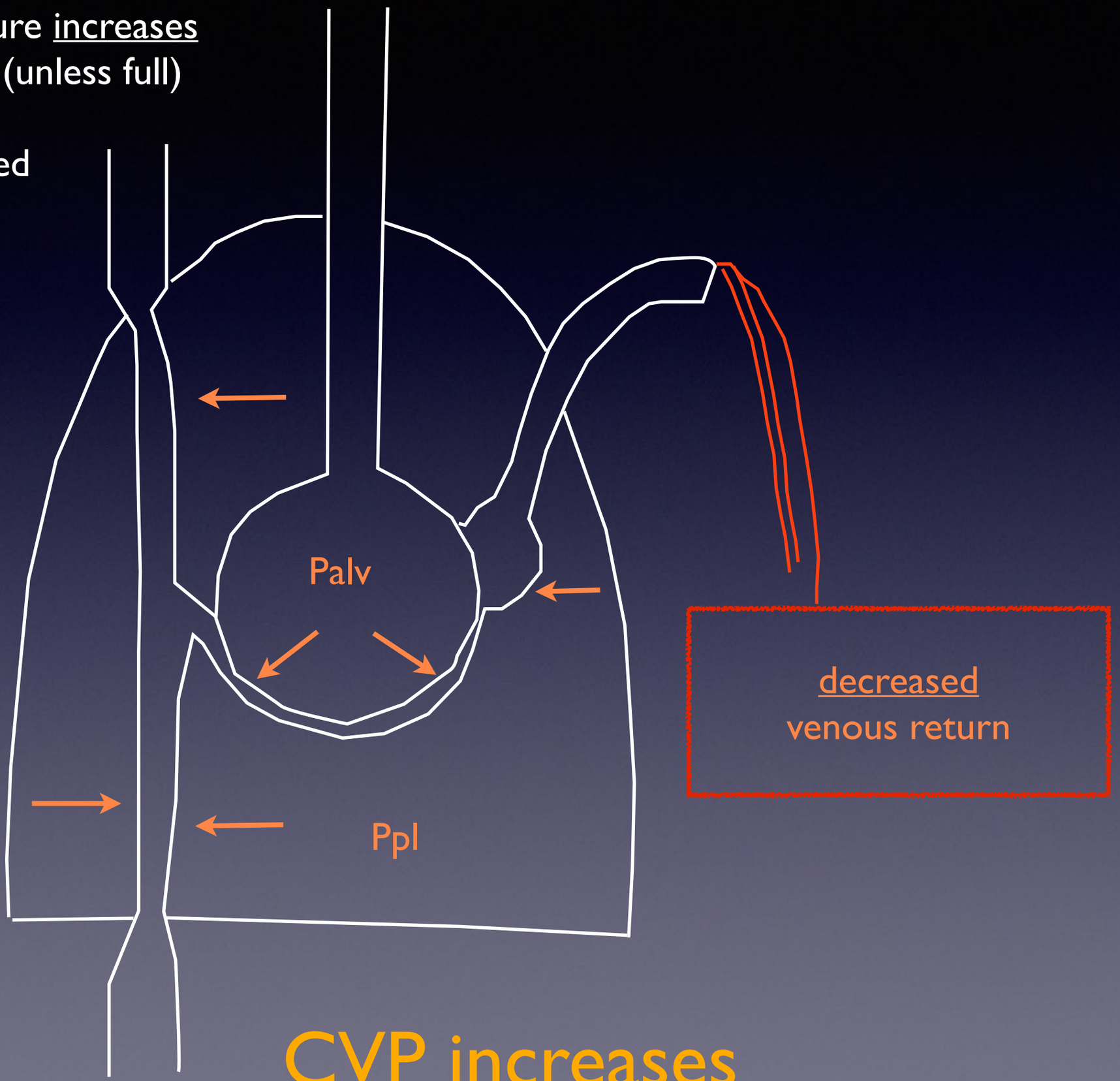
IPPV

BP crashes

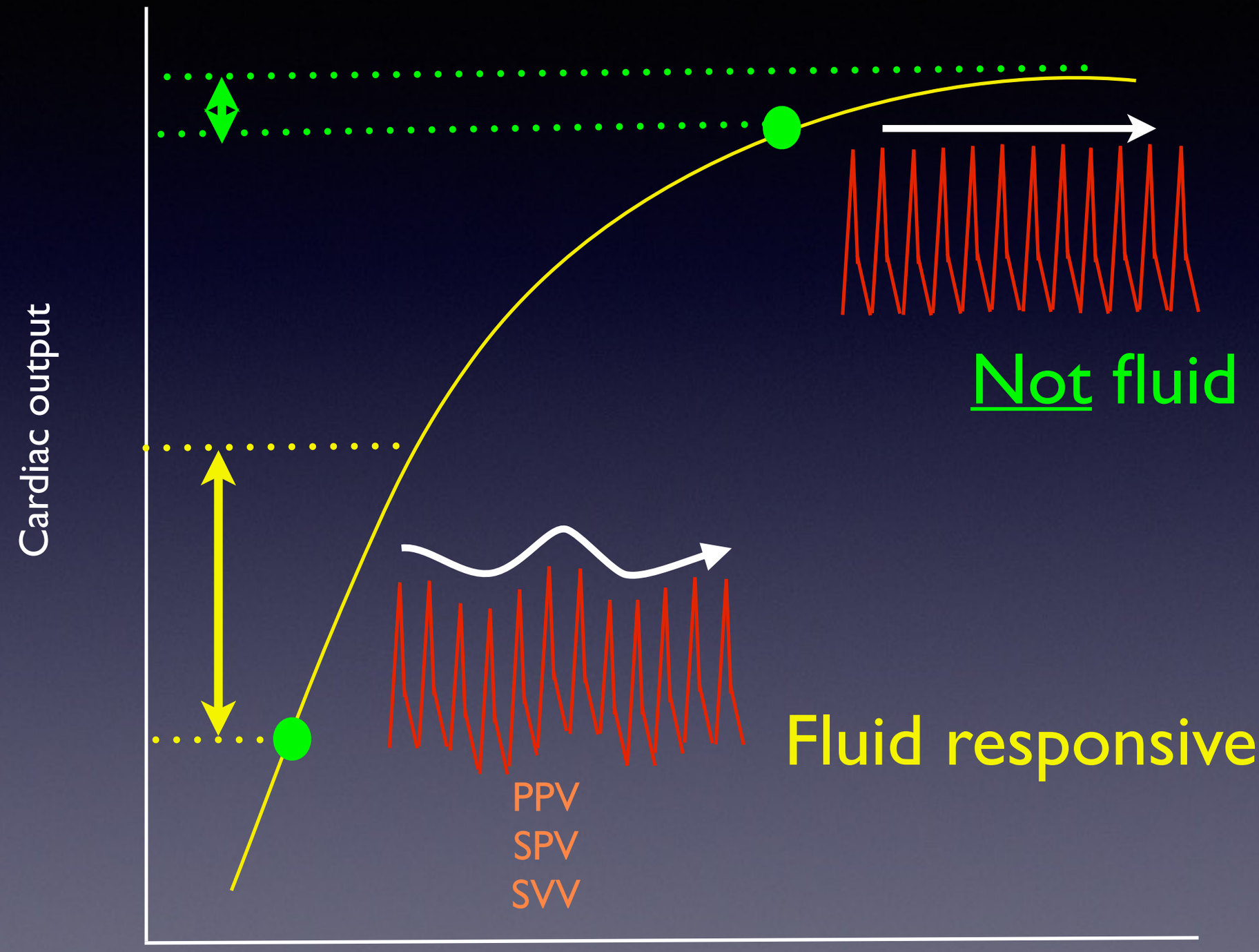
Why ?

IPPV

the pleural and alveolar pressure increases
->the vena cavae compressed (unless full)
-> the CVP increases
-> the venous return is reduced
-> cardiac output is reduced



Response to IPPV tells you where you are on Starling Curve



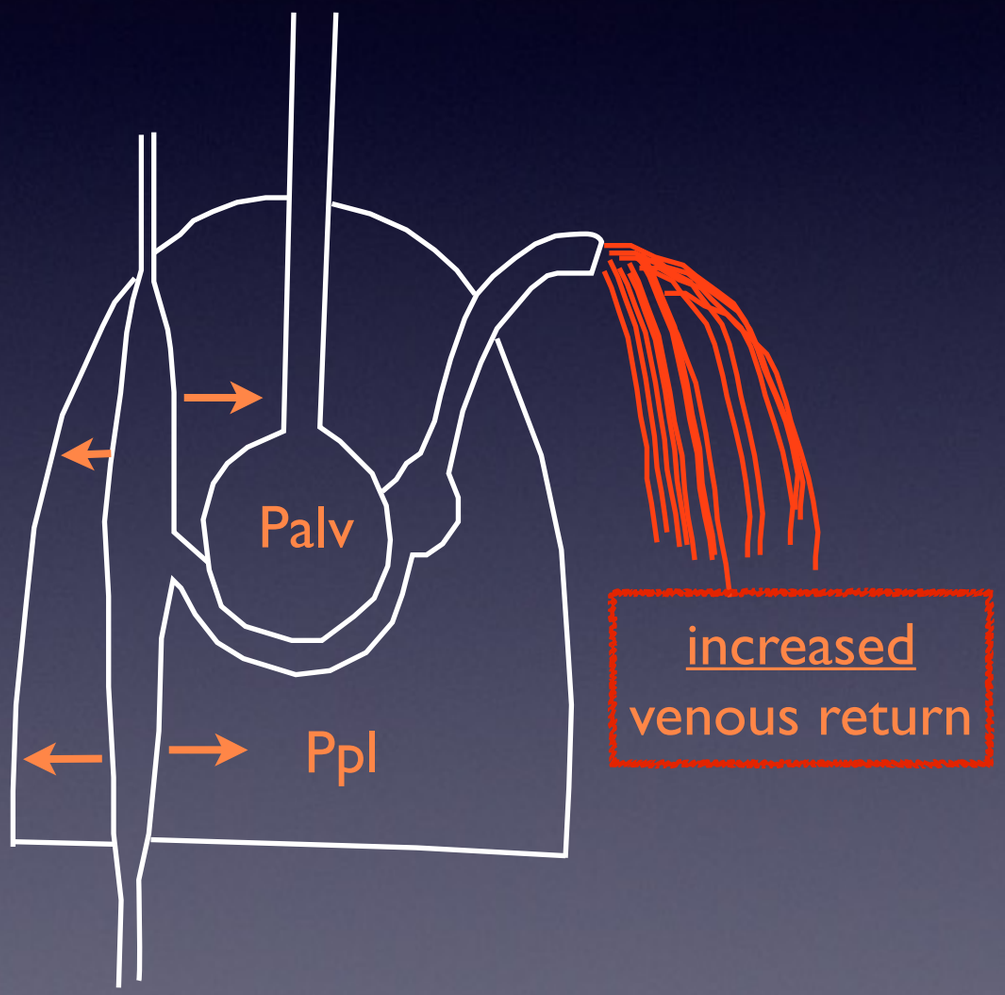
Compare

Spontaneous ventilation

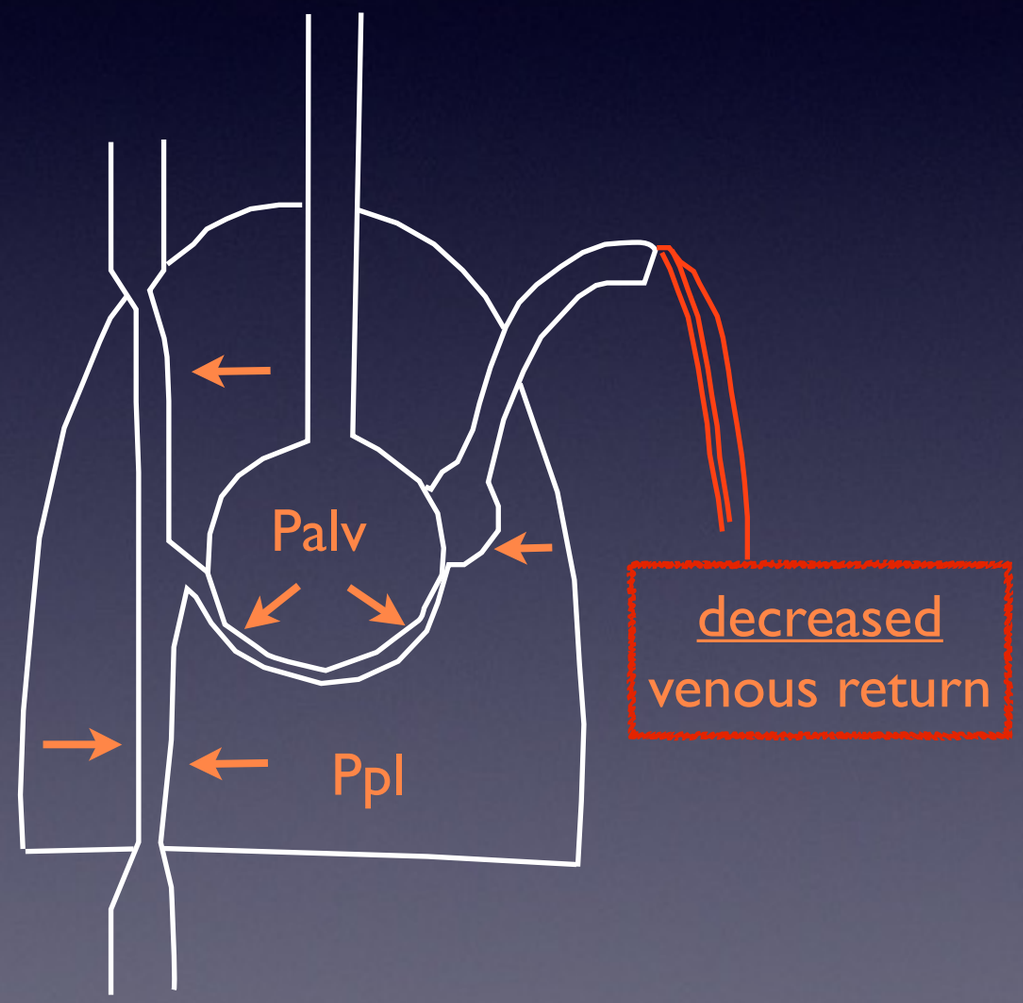
the pleural pressure falls
-> the vena cavae expand (if compliant)
-> the CVP drops
-> sucking more blood into the chest
->venous return and cardiac output increased

IPPV

the pleural and alveolar pressure increases
->the vena cavae compressed (unless full)
-> the CVP increases
-> the venous return is reduced
-> cardiac output is reduced

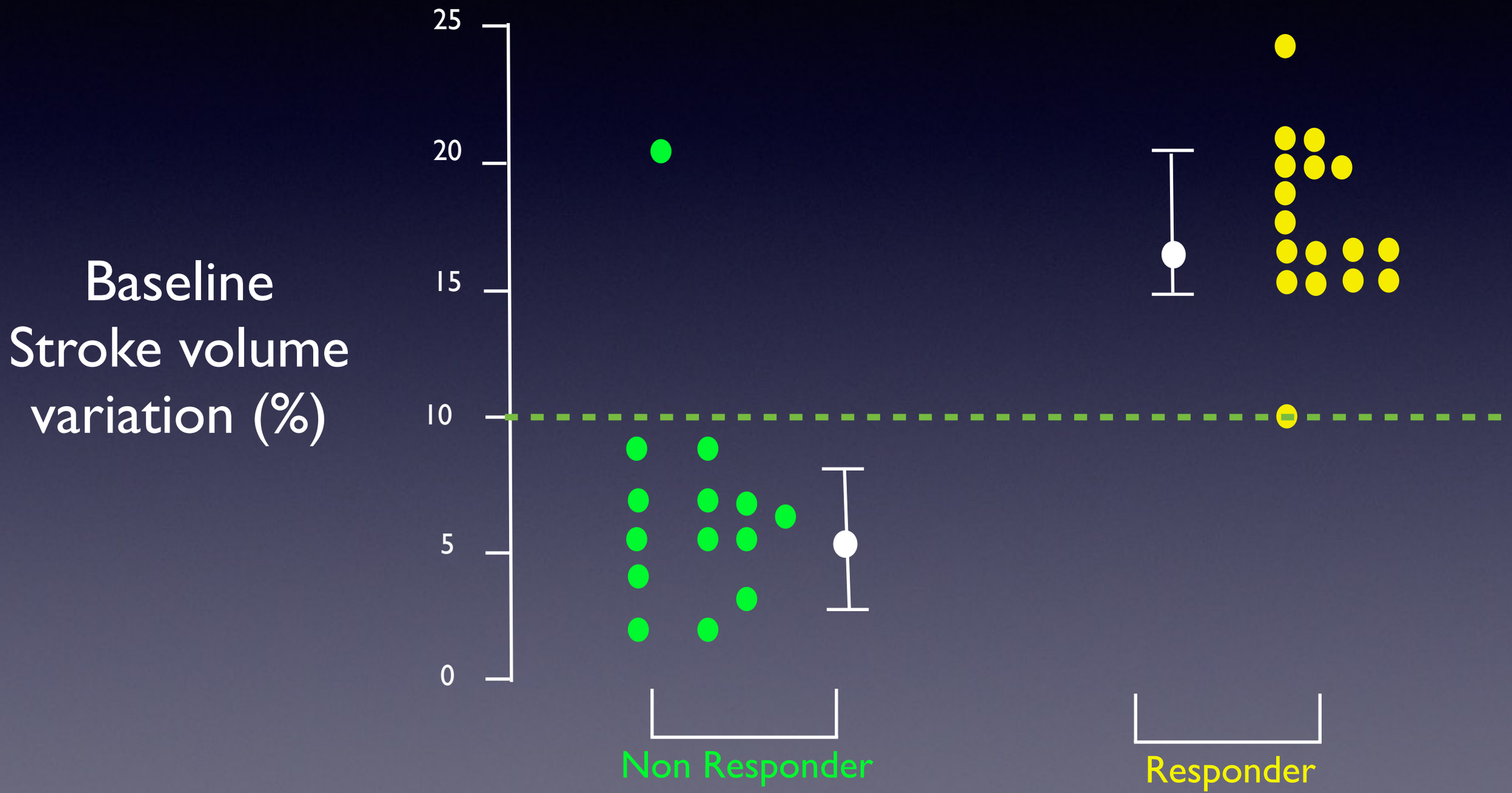


CVP decreases



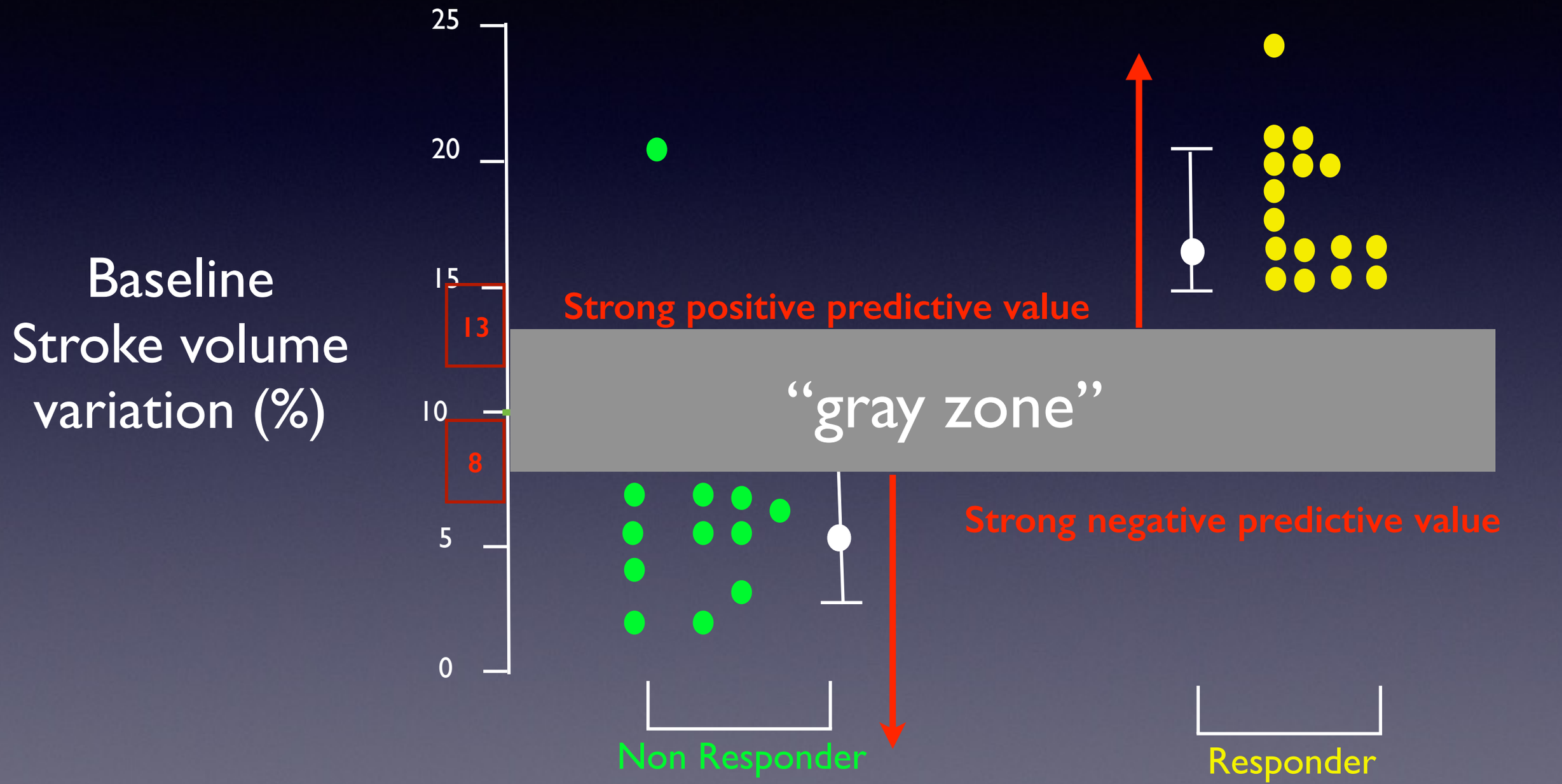
CVP increases

Do variations in stroke volume or pressure predicts fluid responsiveness ?



Br J Anaesth 2008; 101: 761 –8

The “gray zone”



Anesthesiology 2010, 112:1023-1040

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

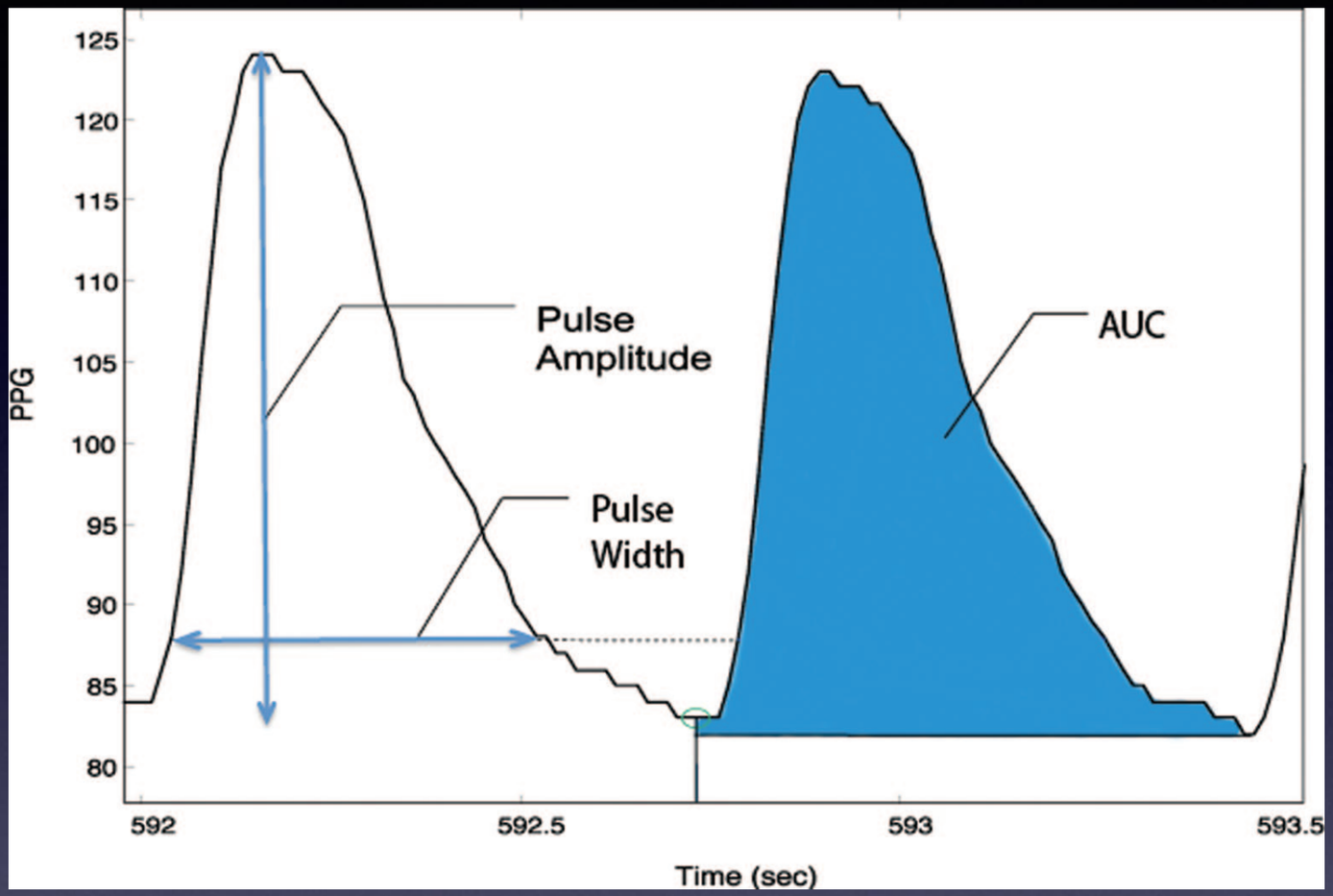
Author	Year	n	Patient	Dynamic Variable		
				SPV	PPV	SVV
Tavernier (31)	1998	15	ICU-sepsis	Y	N	N
Michard (32)	1999	14	ICU-ARDS	N	Y	N
Michard (33)	2000	40	ICU-sepsis	Y	Y	N
Berkenstadt (34)	2001	15	Neurosurg[a]	N	N	Y
Reuter (35)	2002	20	Post C.Surg	Y	N	Y
Reuter (36)	2002	20	Post C.Surg	N	N	Y
Reuter (37)	2003	12	Post C.Surg-a	N	N	Y
		14	Post C.Surg-b			
Bendjelid (38)	2004	16	Post C.Surg	Y	Y	N
Rex (39)	2004	14	Post C.Surg	N	N	Y
Kramer (40)	2004	21	Post C.Surg	Y	Y	N
Marx (41)	2004	10	ICU-sepsis	N	N	Y
Hofer (42)	2005	35	Post C.Surg	N	Y	Y
Preisman (43)	2005	18	Post C.Surg	Y	Y	Y
De Backer (44)[d]	2005	27	ICU-mixed	N	Y	N
Wiesenack (45)	2005	20	C.Surg[a]	N	Y	Y
Feissel (46)	2005	20	ICU-sepsis	N	Y	N
Solus-Biguenet (47)	2006	8	Hepatic surgery	N	Y	N
Charron (48)	2006	21	ICU-mixed	N	Y	N
Natalini (49)	2006	22	ICU-mixed	Y	Y	N
Wyffels (50)	2007	32	Post C.Surg	N	Y	N
Feissel (51)	2007	23	ICU-sepsis	N	Y	N
Lee (52)	2007	20	Neurosurg[a]	N	Y	N
Cannesson (53)	2007	25	C.Surg[a]	N	Y	N
Cannesson (54)	2008	25	C.Surg[a]	N	Y	N
Auler (55)	2008	59	Post C.Surg	N	Y	N
Belloni (56)	2008	19	C.Surg[a]	Y	Y	Y
Cannesson (57)	2008	25	C.Surg[a]	N	Y	N
Hofer (58)	2008	40	Post CABG	N	Y	Y
Biasis (59)	2008	35	Liver transplant	N	Y	Y

→ **High level of evidence**

Meta-analysis of 29 studies, 685 patients

Crit Care Med 2009 Vol. 37, No. 9

Pulse oximeter plethysmographic waveform changes in awake, spontaneously breathing, hypovolemic volunteers

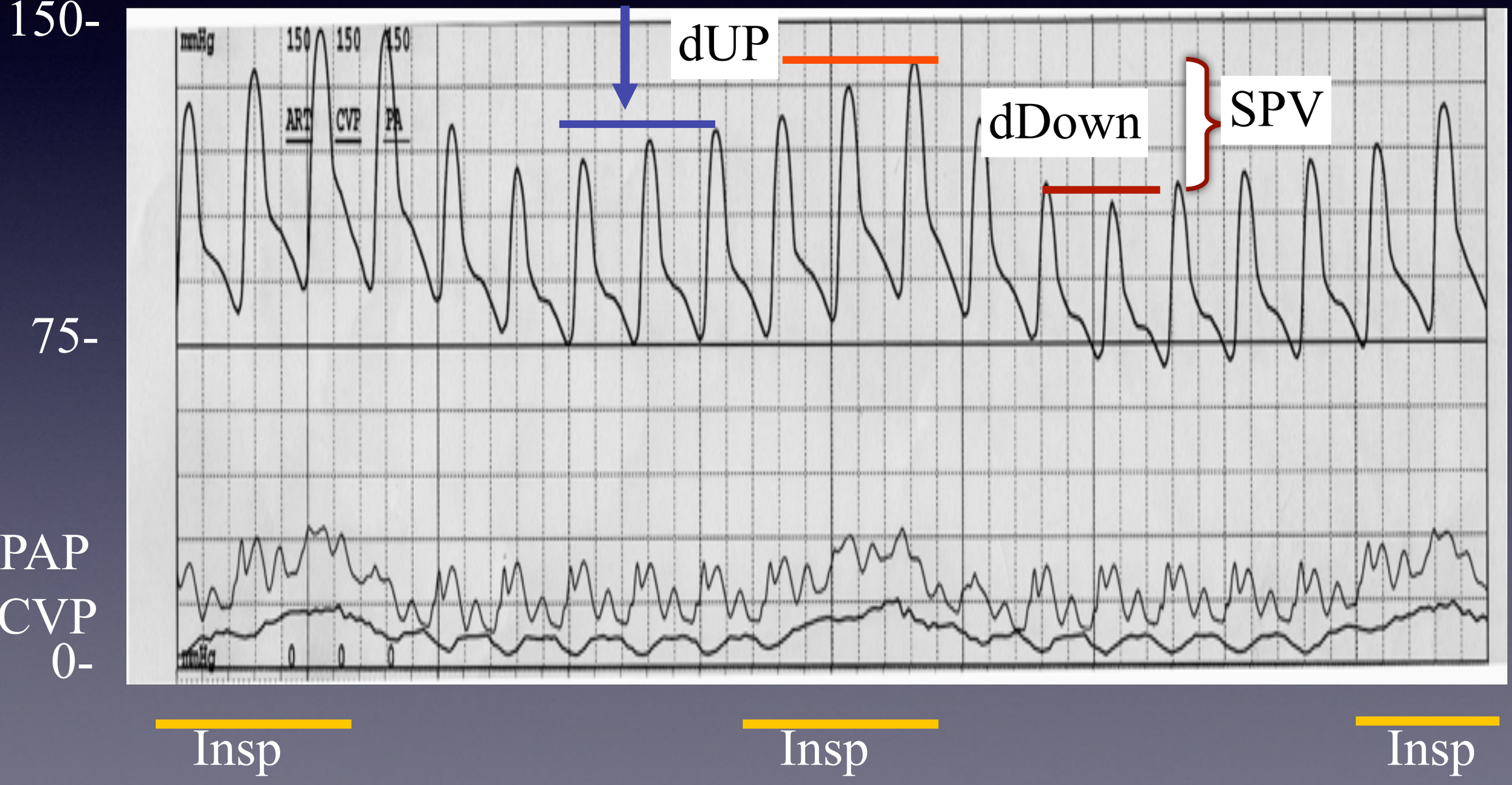


"These results support the use of pulse oximeter waveform analysis as a potential diagnostic tool to **detect clinically significant hypovolemia** before the onset of cardiovascular decompensation in spontaneously breathing patients"

Anesth Analg 2011;112: 368 –74

Example of Systolic Pressure Variation during Positive Pressure Ventilation

Must be ventilated and in sinus rhythm



Limits of Pressure Variation during Positive Pressure Ventilation

		False positive	False negative
L	**Low** HR/RR ratio (Extreme bradycardia or high frequency ventilation)		✓
I	**Irregular** heart beats	✓	
M	**Mechanical** ventilation with low tidal volume		✓
I	**Increased** abdominal Pressure (Pneumoperitoneum)	✓	
T	**Thorax** open		✓
S	**Spontaneous** breathing	✓	✓

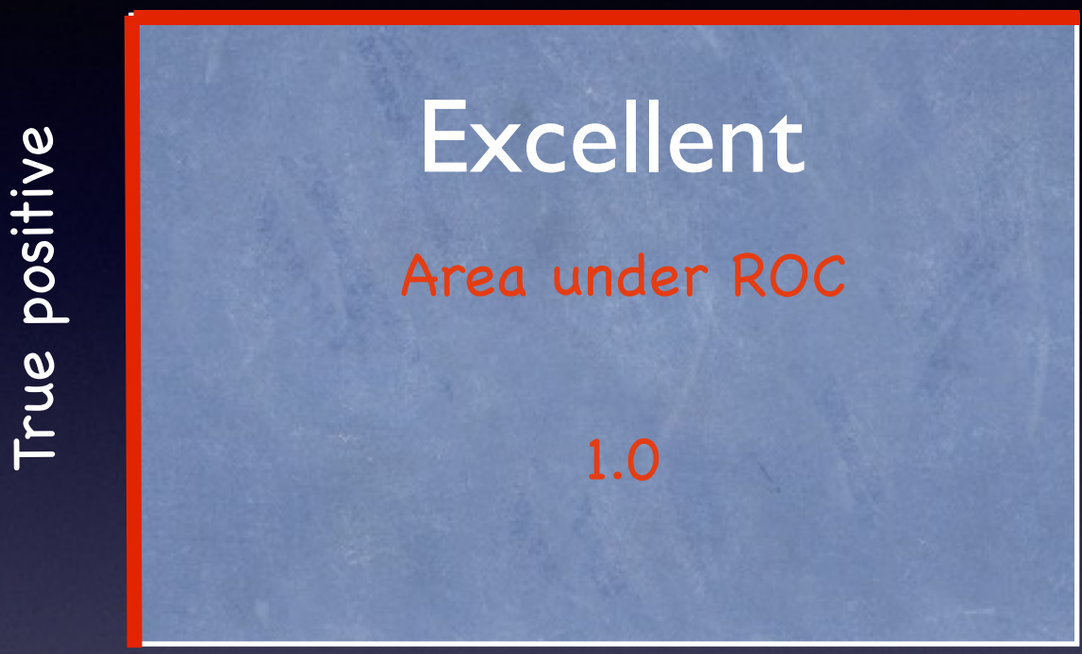
Michard et al. Critical Care (2015) 19:144

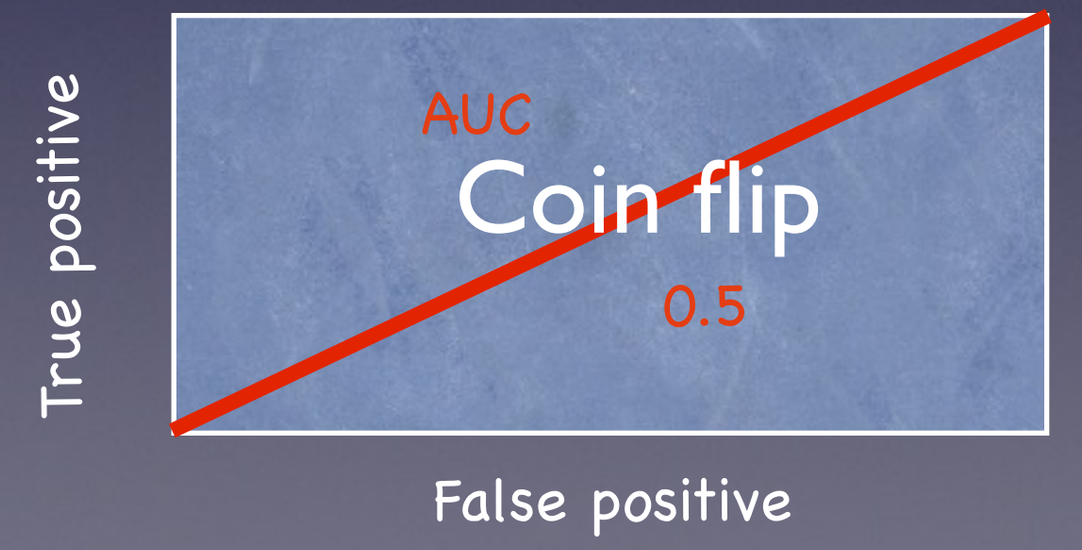
Receiver Operating Characteristic Curve

- ROC curve is a graphical tool allowing one to determine the sensitivity and specificity of a diagnostic test.

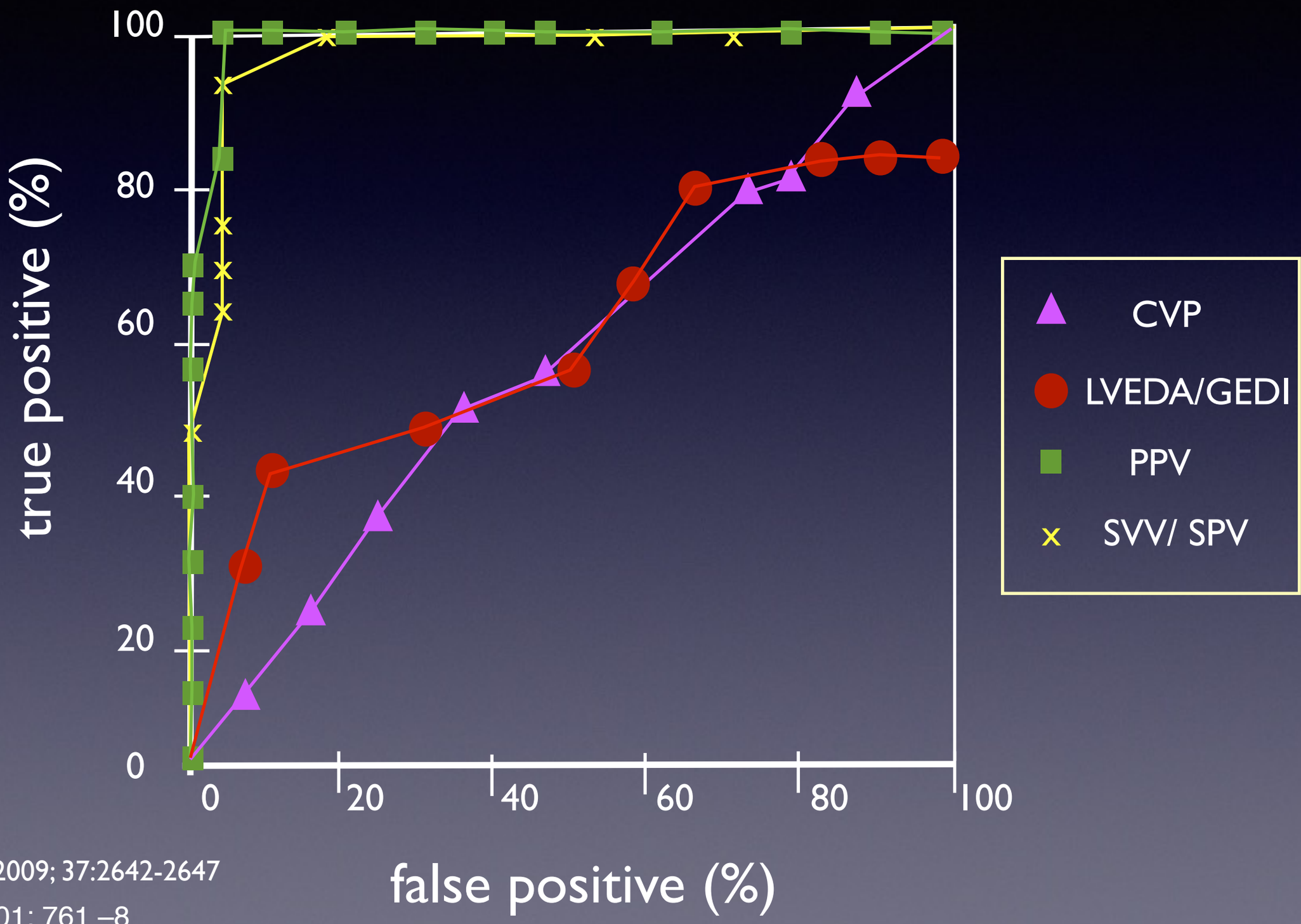
- Statistical tool used by radar operators during WW II to distinguish:

from





How do <u>static</u> compare with <u>dynamic</u> variables in predicting volume responsiveness



Critical Care Medicine 2009; 37:2642-2647

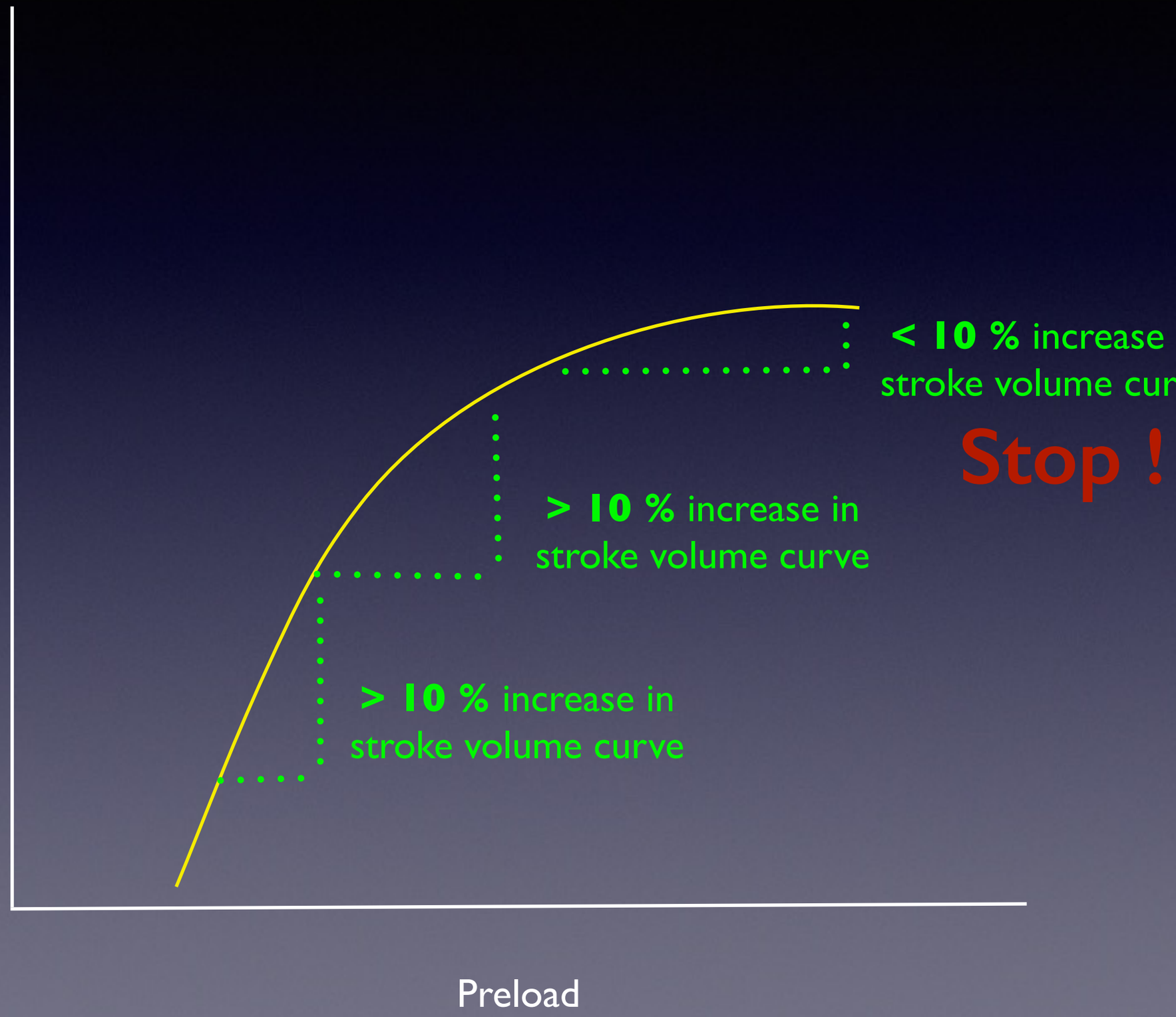
Br J Anaesth 2008; 101: 761 –8

How do you fluid load ?

Give small volume (~ 250 ml) quickly

and measure response immediately

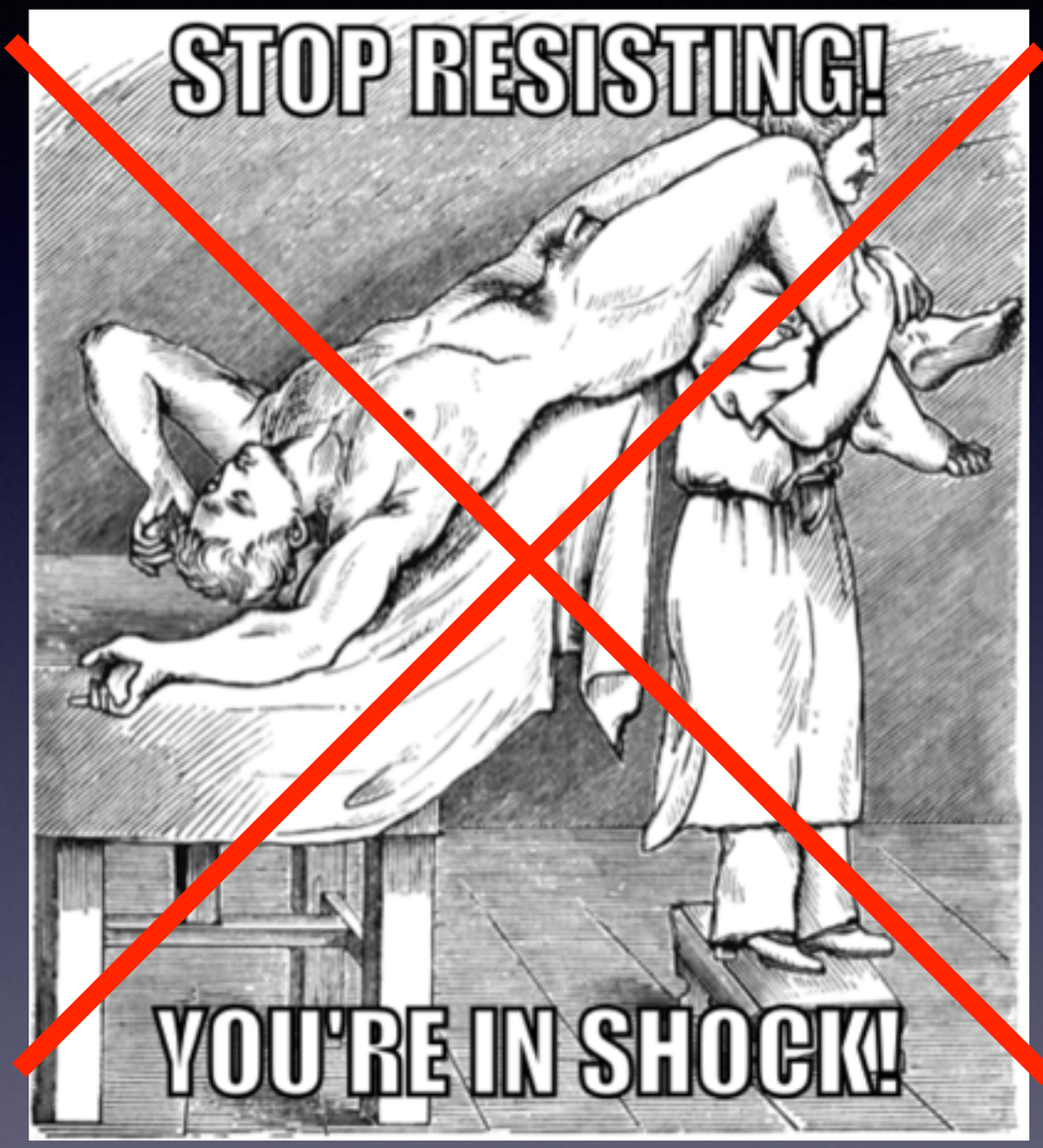
How do you fluid load ?



OR

Or raise ze legs!

Rapidly "transfuses" ~ 500 mL



Not Trendelenburg

Intensive Care Med (2008) 34:659–663

Recap

Cardiac output adequate ?

↓ No

Get to the top of the Starling curve -> optimize fluids

↓

Predict if fluid responsive?

IPPV → SVV/PPV/SPV

Spontaneous respirations → Drop in CVP

SVV/PPV/SPV → Yes → Give fluid

SVV/PPV/SPV → No → Inotropes/vasopressors

Drop in CVP → Yes → Give fluid

Drop in CVP → No → Inotropes/vasopressors

Effective cardiac output adequate ?

The bottom line

To optimise cardiac output,

first maximise the stretch of the sarcomeres(filling),

only then, if CO is still inadequate, improve the energetics of the muscle with inotropes.

This lecture was about

optimising stretch !

Recap

- Cardiac output the most important determinant of O2 delivery
- Delay in treatment = lives lost!
- Give fluids only if increased flow needed and fluid responsive
- "Fluid responsive or not? - that is the question"
- Static measures of blood volume (ex. CVP) does not work
- Dynamic measures predict if fluid responsive
- Only after fluids optimised consider inotropes

???

www.jvsmedicscorner.com

(Mallory / Everest2013)

Thanks for listening