

UPMC Critical Care



www.ccm.pitt.edu



Shock and Monitoring

Samuel A. Tisherman, MD, FACS, FCCM

Professor

Departments of CCM and Surgery

University of Pittsburgh

Shock



Anaerobic metabolism

Lactic acidosis

"Dysoxia"

Activation of inflammatory cascades

Inadequate DO_2

Organ system dysfunction

Shock



⊕ **Normal cardiovascular system**

⊕ **Pathophysiology of shock**

⊕ **Categories of shock**

⊕ **Resuscitation**

⊕ **Monitoring**

Key components of CV system



⊕ Intravascular volume



© Nick Peltier

⊕ Cardiac output

- Preload
- Heart rate
- Contractility
- Afterload



www.bradfitzpatrick.com

Key components of CV system



⊕ Resistance circuit – arterioles

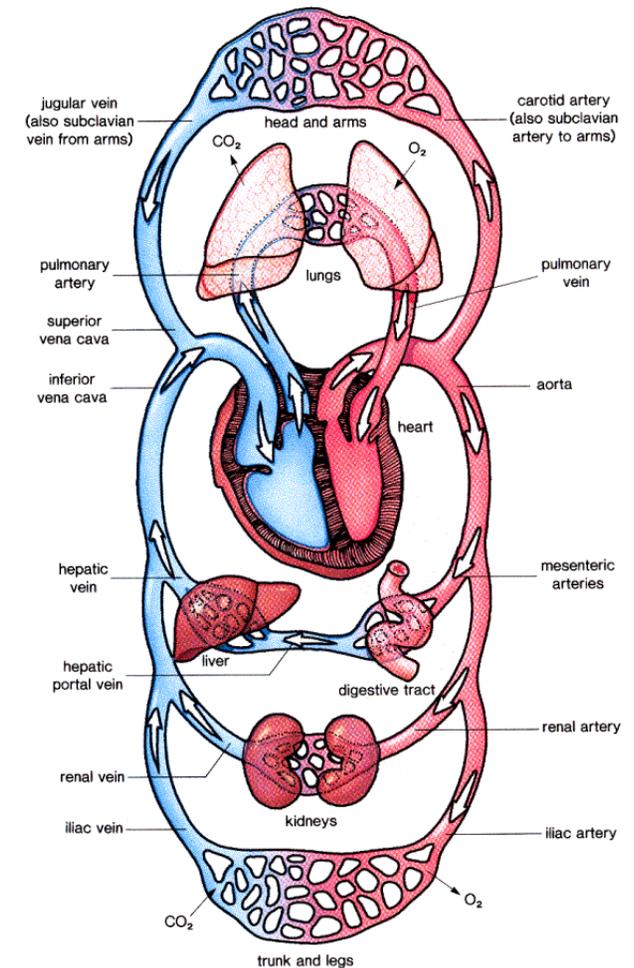
- Distribution of organ flow

⊕ Capillaries

- Leak
- Obstruction to flow
- Shunts

⊕ Venous capacitance vessels

- Vary venous return

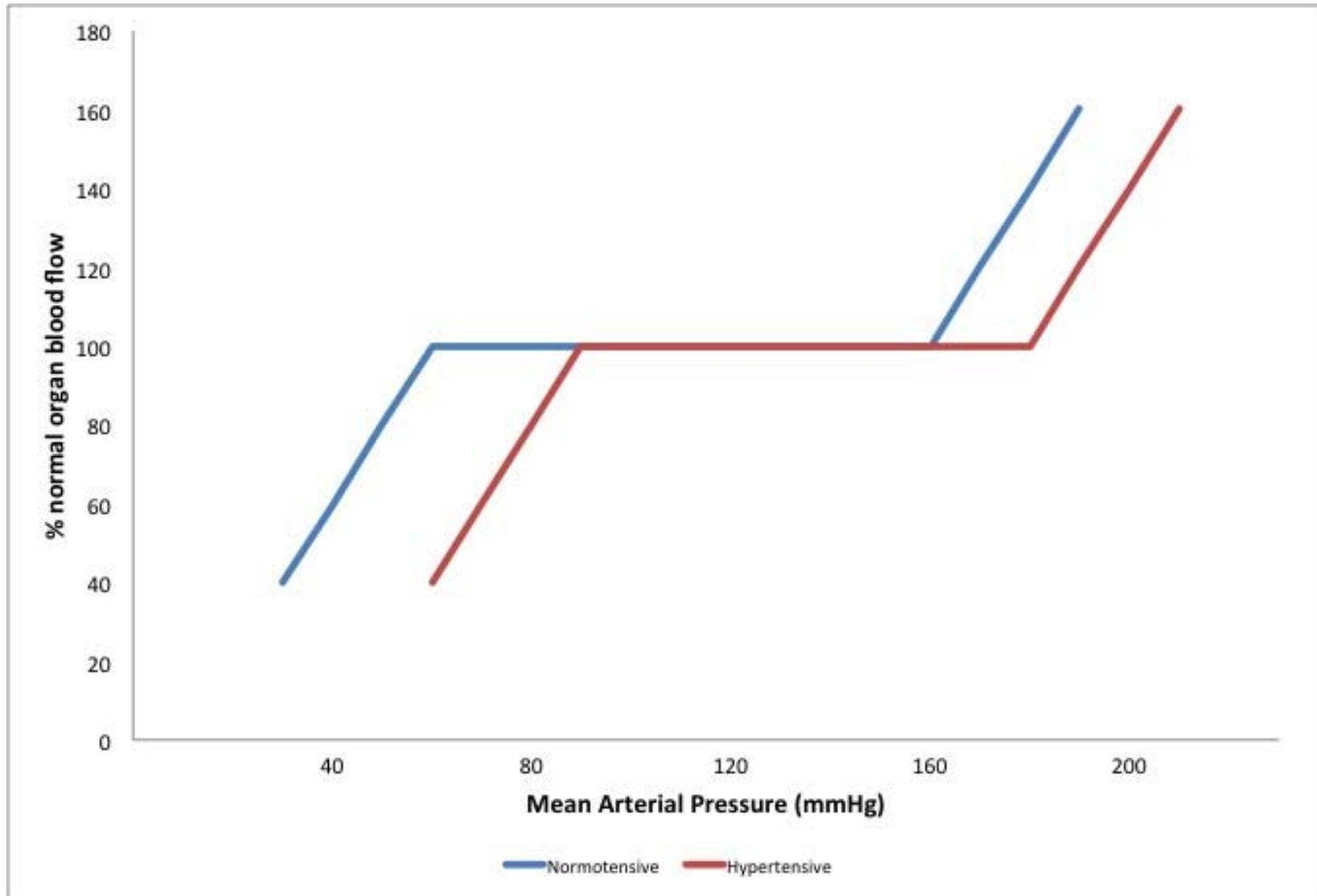




Organ blood flow

- ⊕ **Most dependent upon mean arterial pressure (MAP)**
- ⊕ **Autoregulation**
 - ✿ Maintained during large variations of MAP
 - ✿ 60-130 mmHg
 - ✿ Can be impaired - hypertension

Autoregulation





Supply and demand

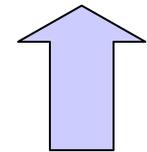
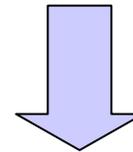
⊕ O₂ delivery

- ⊗ Hemoglobin
- ⊗ Saturation
- ⊗ Cardiac output

⊕ O₂ demand



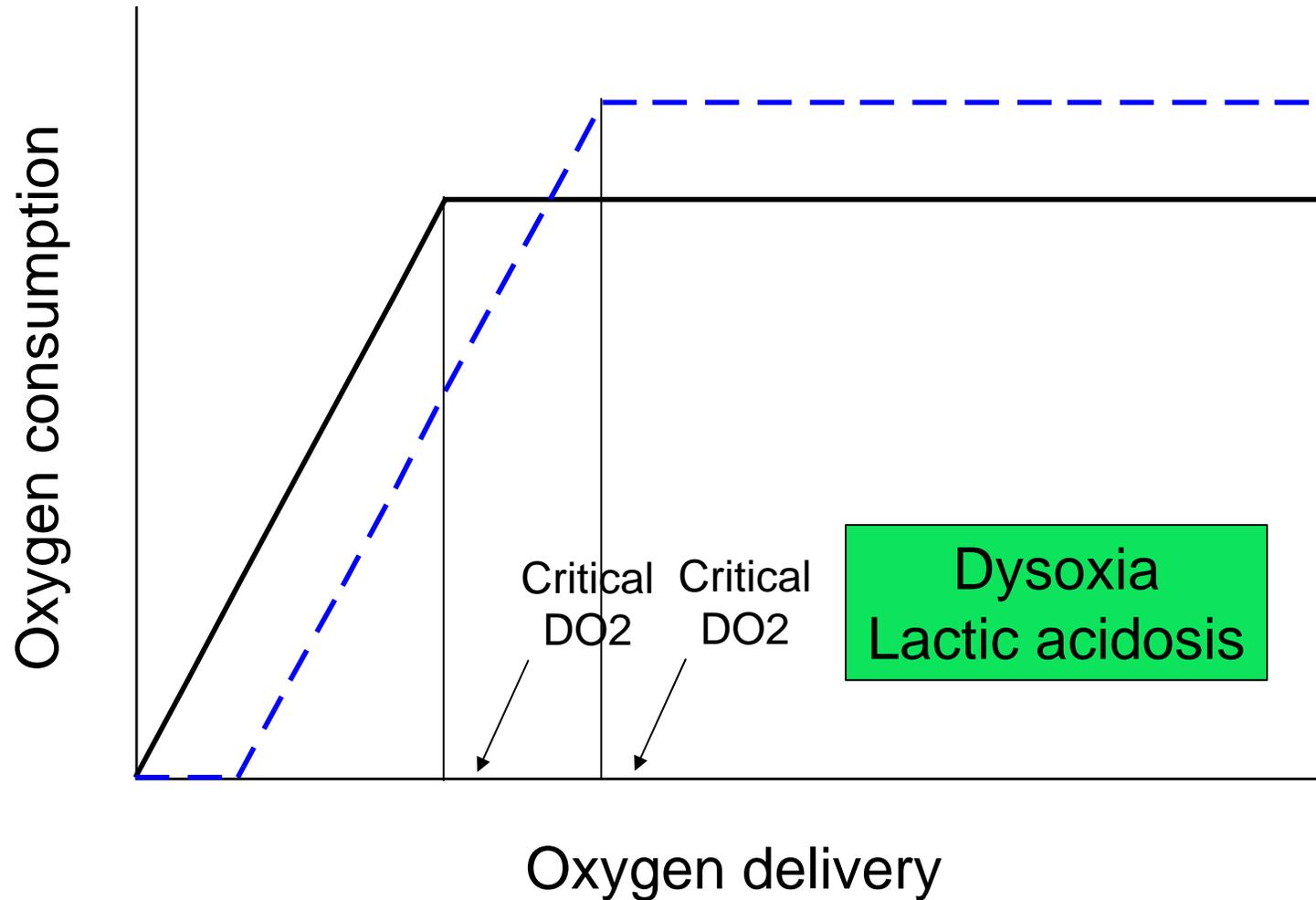
20-60%



$$VO_2 = DO_2 \times ERO_2$$

ER = extraction ratio

O₂ supply dependency



Cytopathic Hypoxia



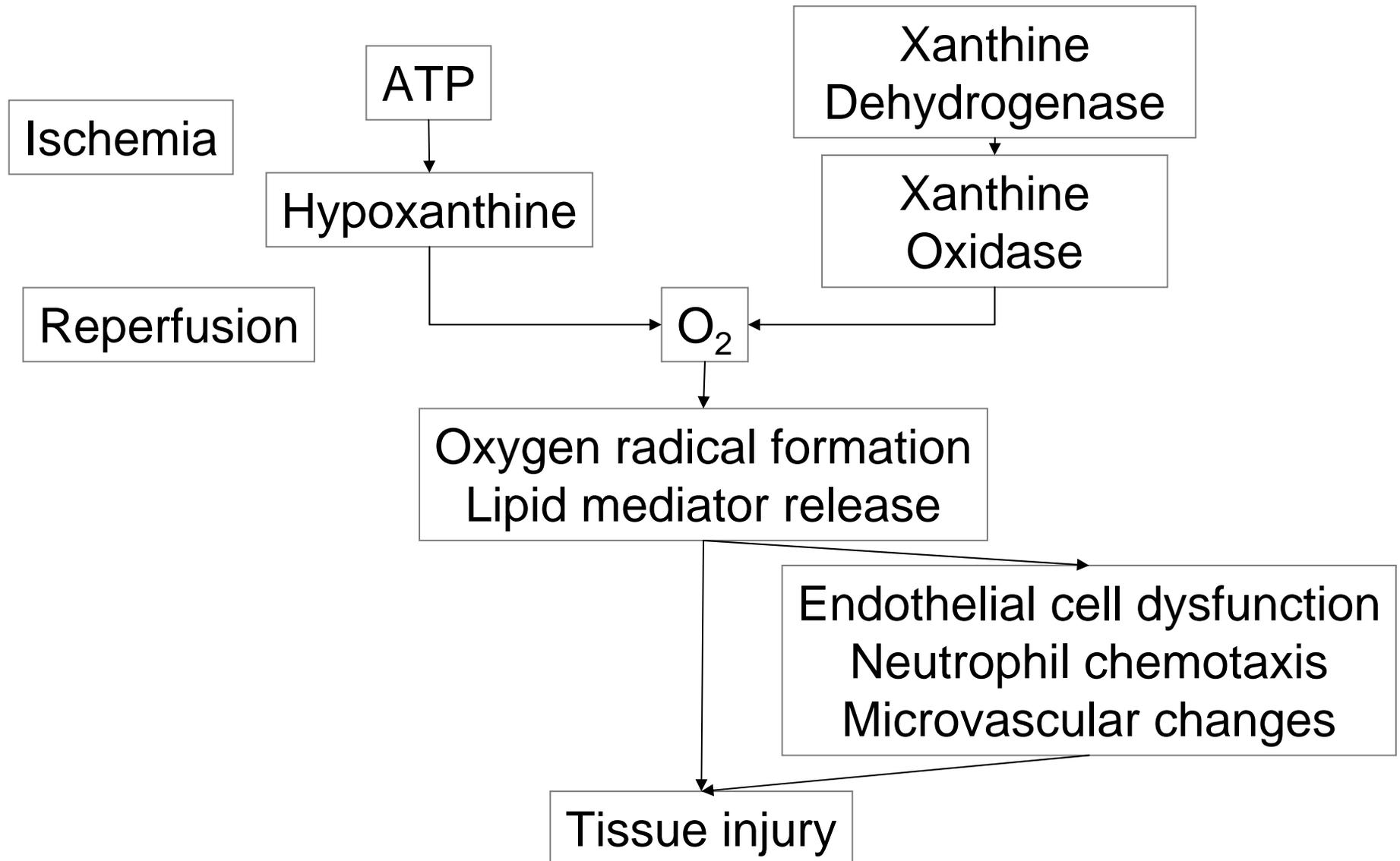
⊕ Sepsis

- ✿ Normal oxygen delivery
- ✿ Impaired mitochondrial O₂ utilization

⊕ Mediators

- ✿ Inflammatory cytokines
- ✿ Inhibition of pyruvate dehydrogenase
- ✿ Inhibition of cytochrome a₃ by NO
- ✿ Irreversible inhibition of one or more mitochondrial respiratory complexes by peroxynitrite
- ✿ Poly(ADP-ribose) polymerase-1

Reperfusion Injury

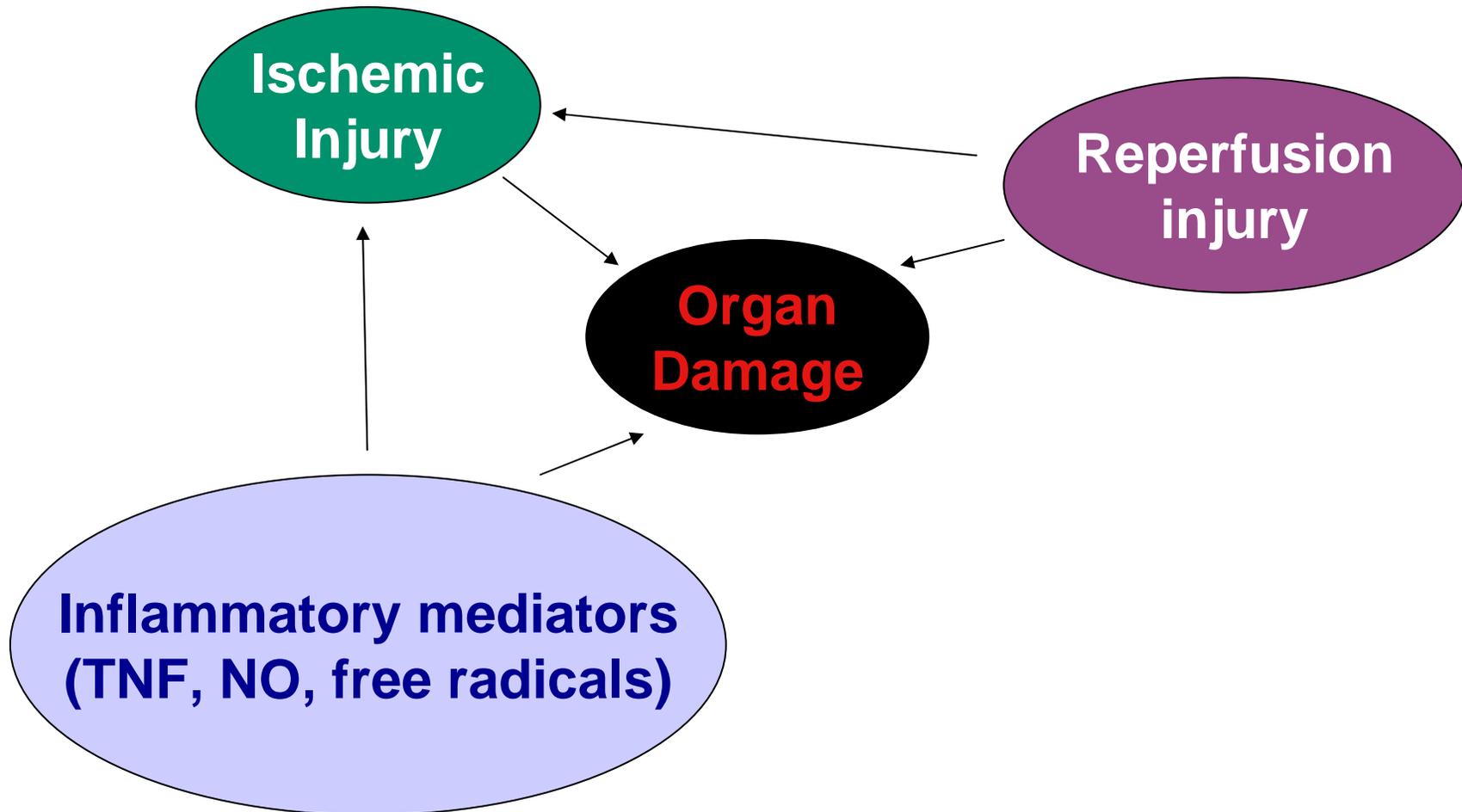


It's not the fall that gets you....

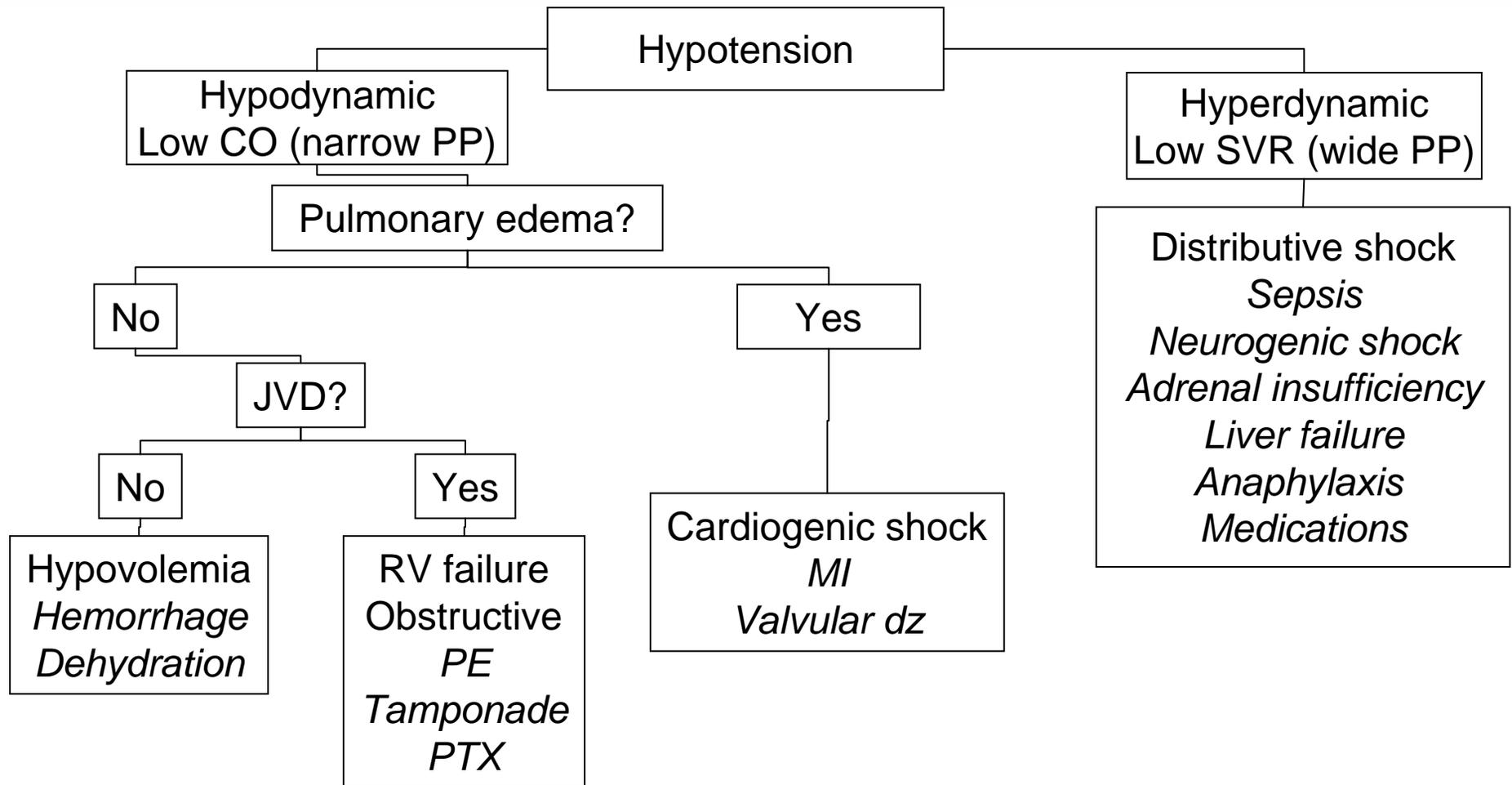


✦ Exemplary Care ✦ Cutting-edge Research ✦ World-class Education ✦

Organ failure



Classifications of shock





Hemorrhagic shock

- ⊕ **Most common cause of hypotension in trauma patients**
- ⊕ **Initial fluid should be crystalloid**
- ⊕ **Transfusion**
 - ✿ Hypotensive after 2 L crystalloid (ATLS)
 - ✿ Earlier for severe shock
- ⊕ **Uncontrolled hemorrhage**
 - ✿ Stop hemorrhage
 - ✿ Limited fluid resuscitation



Cardiogenic shock

- ⊕ Resuscitation simultaneous with revascularization
- ⊕ Patients may need higher than normal filling pressures (poor LV compliance)
- ⊕ Inotropes may worsen ischemia
- ⊕ Pacing may be helpful for relative bradycardia
- ⊕ Intra-aortic balloon pump improves coronary perfusion and decreases afterload



Pulmonary embolism

⊕ Risk factors

- ⊕ Hypercoagulability, stasis, trauma

⊕ Presentation

- ⊕ Respiratory
- ⊕ Cardiovascular

⊕ Diagnosis

- ⊕ CT angio, VQ, echo, angio, d-dimer

⊕ Management

- ⊕ Anticoagulation, thrombolytic, embolectomy
- ⊕ Prevent the next one -?filter



Cardiac tamponade

- ⊕ **Penetrating trauma more common than blunt**
- ⊕ **Beck's triad (hypotension, distant heart sounds, JVD) obscured by hypovolemia**
- ⊕ **High index of suspicion**
- ⊕ **Diagnose: echo or pericardial window**
- ⊕ **Treatment:**
 - ✿ **Need sternotomy or thoracotomy (trauma)**
 - ✿ **Pericardiocentesis (non-trauma)**



Septic shock

⊕ Decreased preload

- ✱ Hypovolemia (capillary leak)
- ✱ Increased venous capacitance

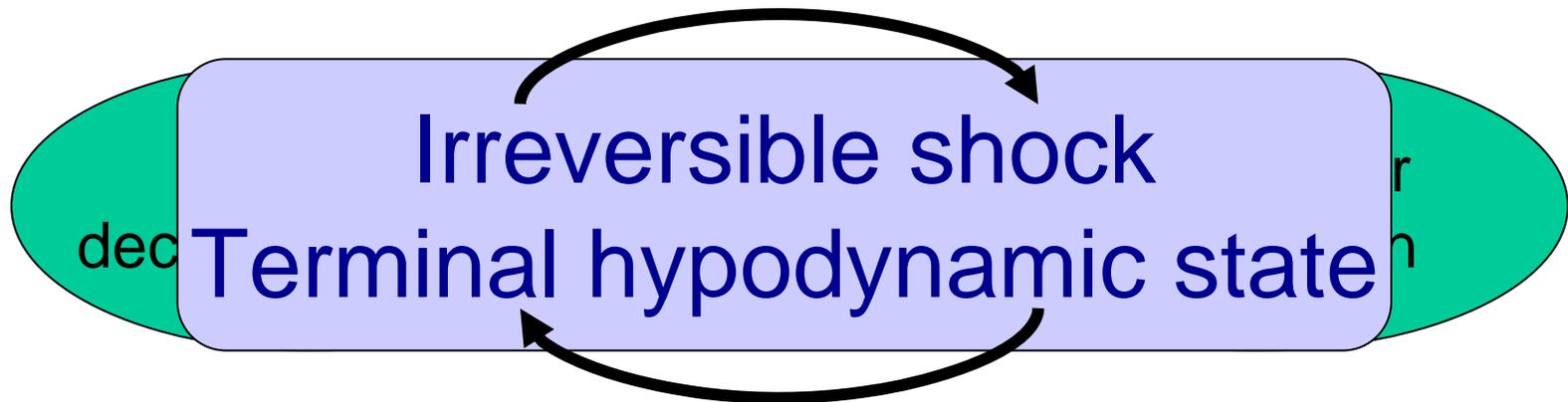
⊕ Vasodilatation

⊕ Cardiac dysfunction

- ✱ Ventricular dilatation
- ✱ Decreased ejection fraction

⊕ Cytopathic hypoxia

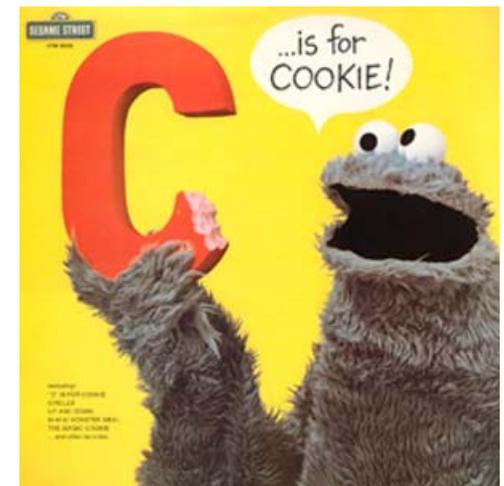
Endstage shock



Initial management



ABCs



Initial management



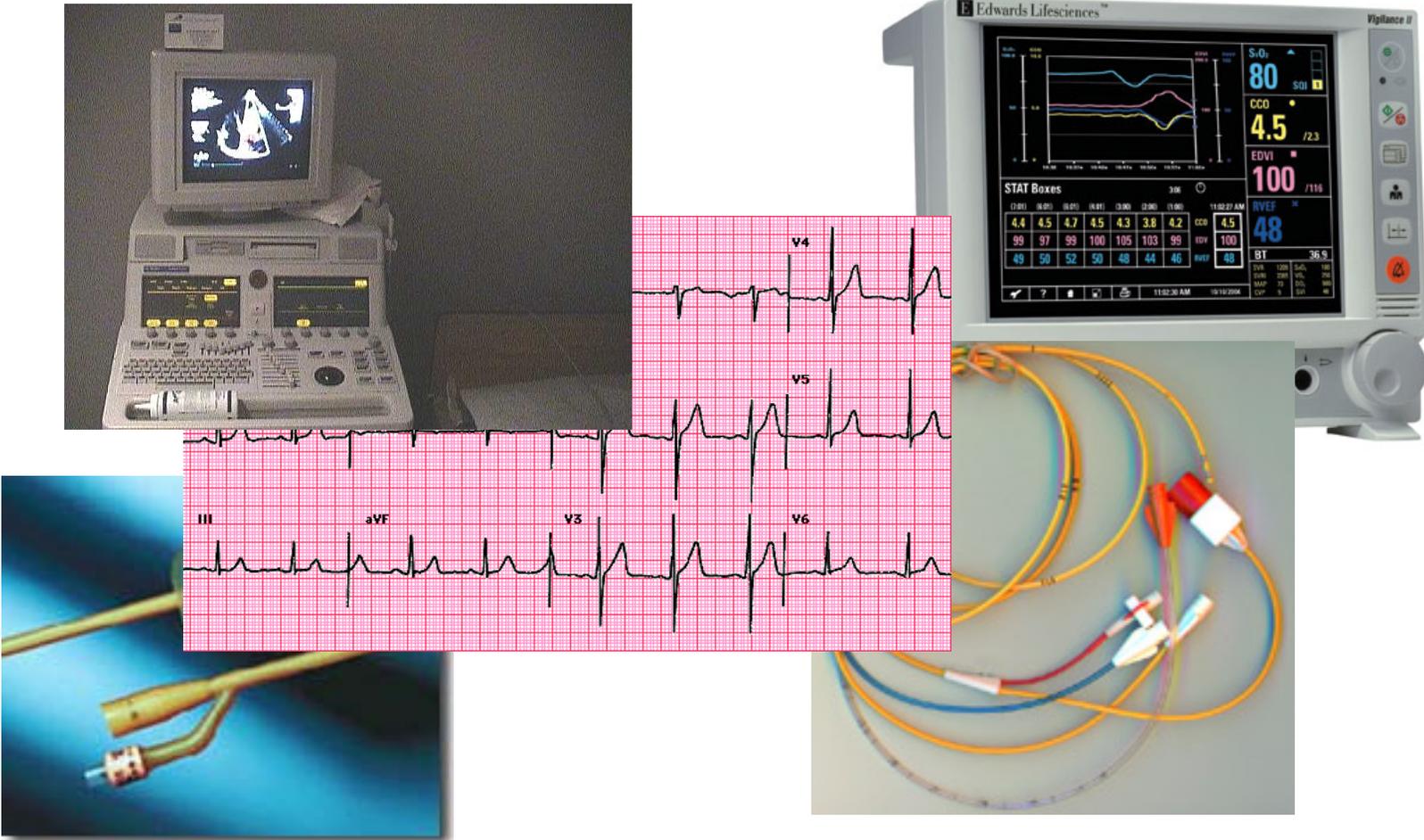
⊕ Resuscitation

- ✱ Ventilation
 - ⊕ ?intubation early
- ✱ Infusion
 - ⊕ Bolus to effect
 - ⊕ ?blood
 - ⊕ Clear lactate
- ✱ Pump therapy
 - ⊕ Inotropic support
 - ⊕ Afterload reduction
- ✱ Vasodilatation
 - ⊕ Vasopressor support

⊕ Definitive therapy

- ✱ Stop bleeding
- ✱ Circulatory assist
- ✱ Antibiotics and drainage/debridement

Monitoring



CVP



⊕ Only elevated in disease

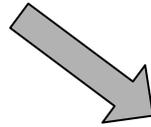
- ✿ RV dysfunction
- ✿ Pulmonary hypertension
- ✿ LV dysfunction
- ✿ Tamponade
- ✿ Hyperinflation
- ✿ Intravascular volume expansion

⊕ Poor correlation with volume responsiveness

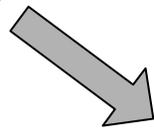
PAOP and Preload



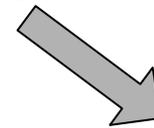
Muscle



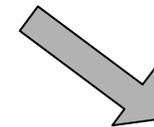
LVEDV



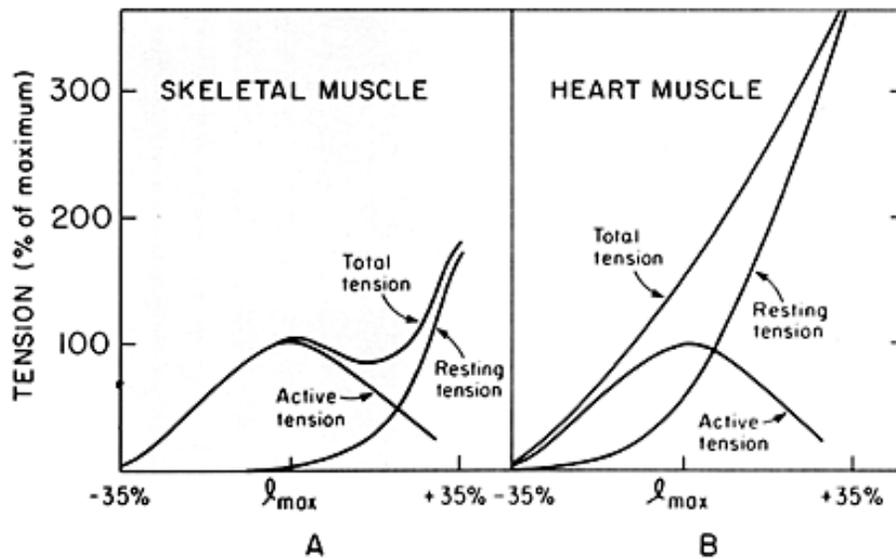
LVEDP



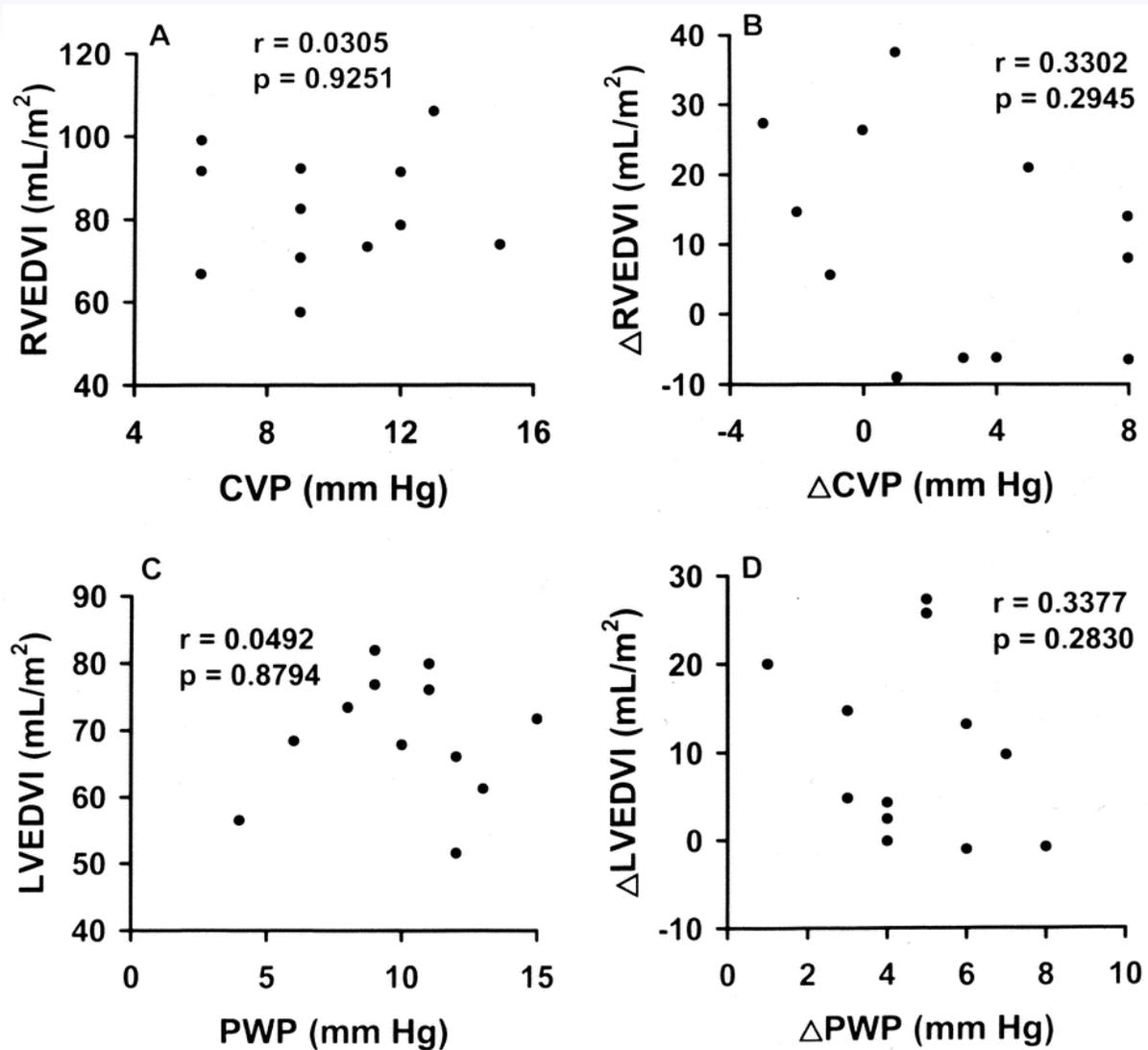
LAP



PAOP



CVP and PAOP



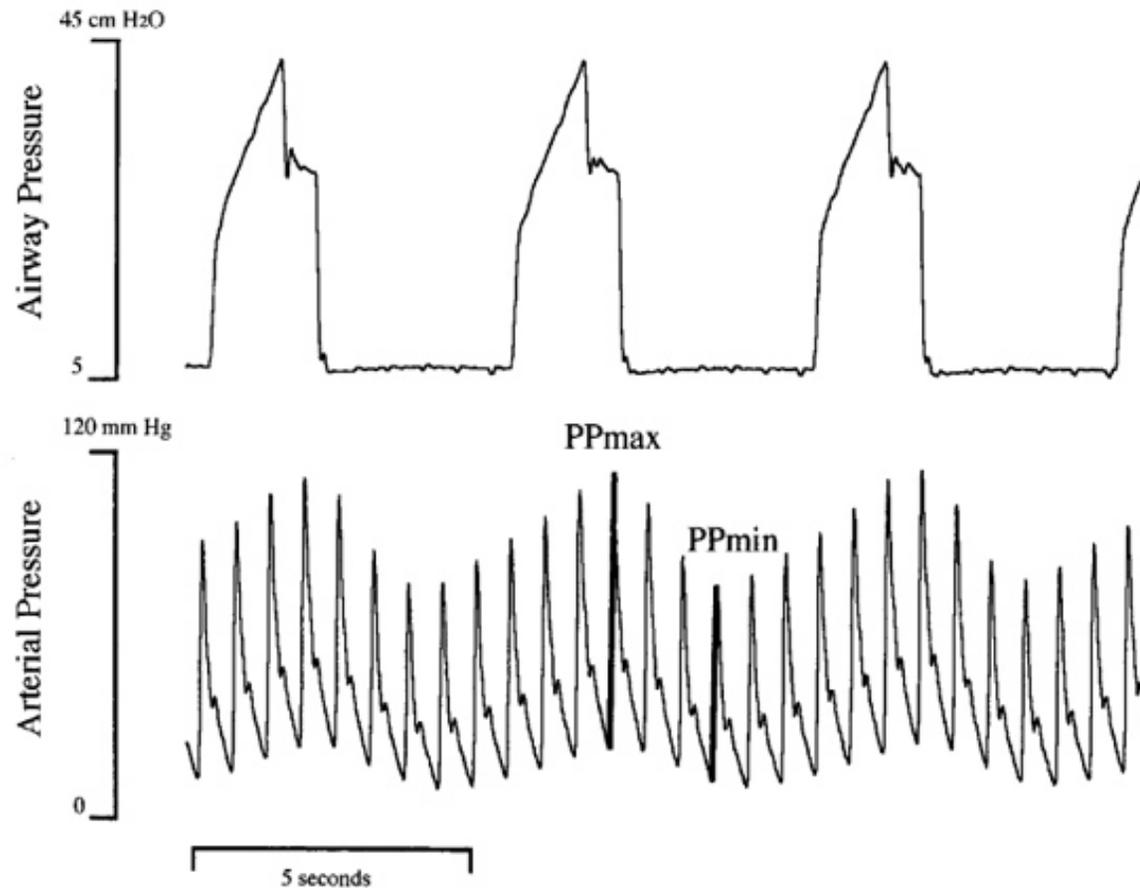
Kumar, et al. CCM, 2004.

Functional Hemodynamic Monitoring



Positive pressure ventilation changes LV output

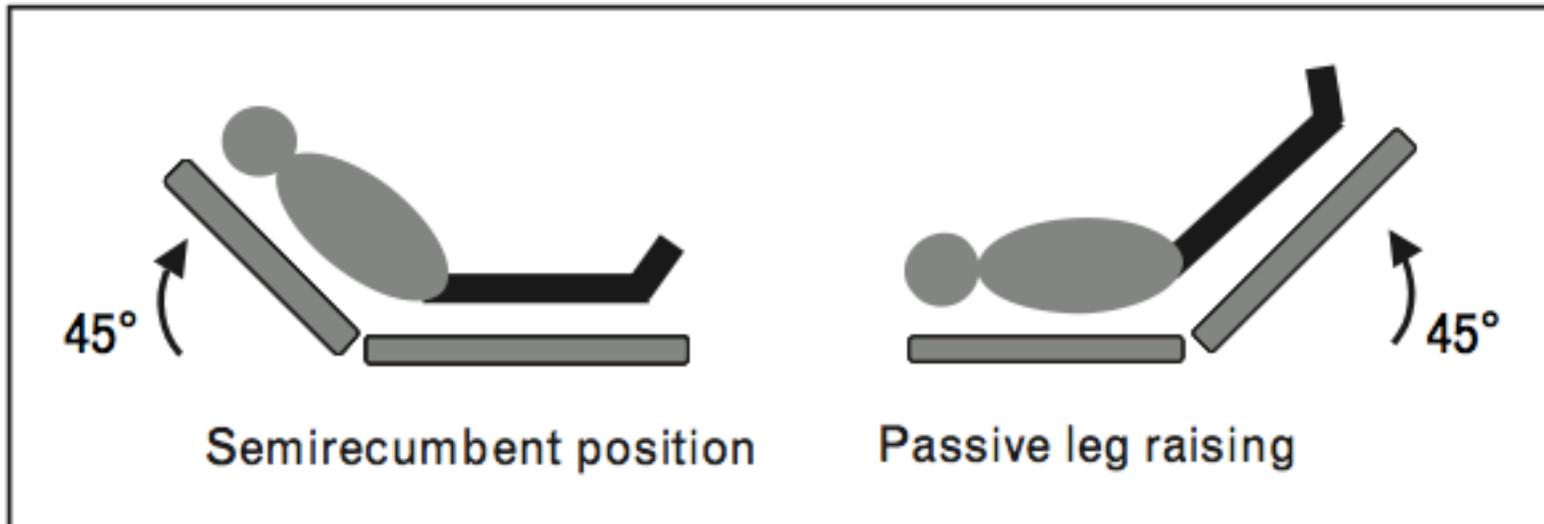
Resp variation in pulse pressure



Limitations: spontaneous breathing, arrhythmias.

Michard and Teboul. *Crit care*, 2000.

Passive Leg Raising



Passive Leg Raising



Variable	Area Under the Curve	<i>p</i> vs. 0.500	Best Cut-Off Value
Compliance of the respiratory system			
>30 cm H ₂ O/mL (n = 26)			
Pulse pressure variation at baseline	0.98 ± 0.03	<.0001	12%
Passive leg-raising–induced changes in CI	0.91 ± 0.06	<.0001	10%
End-expiratory occlusion-induced changes in CI	0.97 ± 0.03	<.0001	5%
Global end-diastolic volume index at baseline	0.69 ± 0.11 ^a	.090	—
Compliance of respiratory system			
≤30 cm H ₂ O/mL (n = 28)			
Pulse pressure variation at baseline	0.69 ± 0.10	.04	4%
Passive leg-raising–induced changes in CI	0.94 ± 0.05 ^a	<.0001	10%
End-expiratory occlusion-induced changes in CI	0.93 ± 0.05 ^a	<.0001	5%
Global end-diastolic volume index at baseline	0.48 ± 0.11 ^a	.980	—

Monnet, et al. *CCM*, 2012.



Preload Echo

⊕ Distensibility index

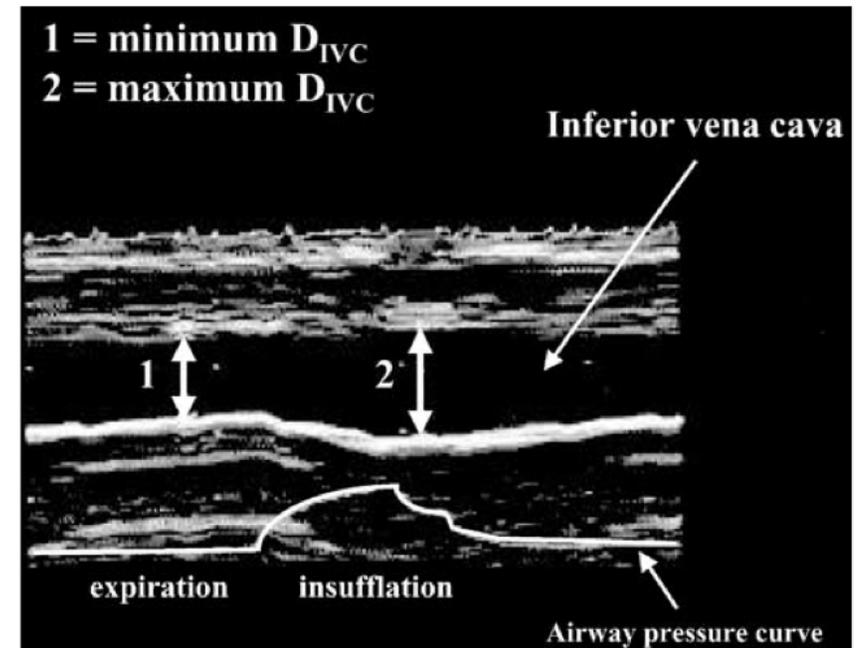
$$\bullet \frac{\text{Max-min } D_{IVC}}{\text{Min } D_{IVC}} \times 100\%$$

⊕ Collapsibility index

$$\bullet \frac{\text{Max-min } D_{IVC}}{\text{Max } D_{IVC}} \times 100\%$$

⊕ Resp variability index

$$\bullet \Delta D_{IVC} = \frac{\text{Max-min } D_{IVC}}{\text{Mean } D_{IVC}} \times 100\%$$



Feissel, et al. *Intensive Care Med*, 2004.

Mixed venous oxygen - SvO₂



- ⊕ **Decrease SvO₂**
 - ✱ Decrease DO₂
 - ⊕ Hb, O₂ sat, cardiac output
 - ✱ Increase O₂ demands
 - ⊕ Fever, sepsis, exercise
 - ✱ Normal >65%
 - ✱ Critical value ~40%

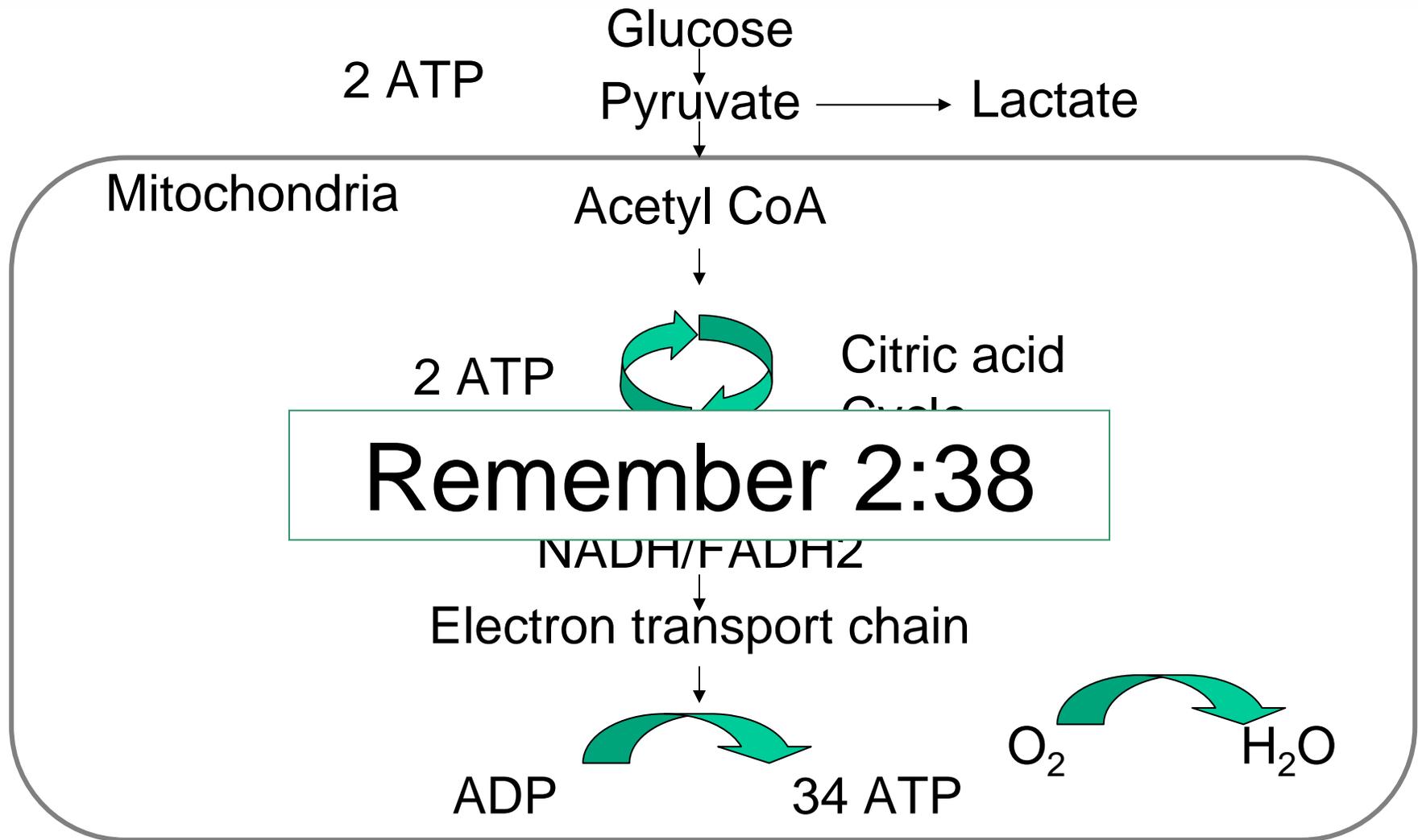


⊕ **Resuscitation endpoint for sepsis and cardiogenic shock**

⊕ **Sepsis -> maldistribution of blood flow**

✱ SvO₂ normal with tissue dysoxia still present

Oxidative Metabolism





Base Deficit

⊕ Definition

- ✿ Amount of base needed to normalize pH with normal PCO_2

⊕ Limitations

- ✿ Administration of bicarbonate
- ✿ Alcohol intoxication
- ✿ Hyperchloremic metabolic acidosis
- ✿ Seizures
- ✿ Pre-existing acidosis



Lactate

⊕ Excess production

- ✱ Anaerobic metabolism
- ✱ Initial level and time to clearance useful

⊕ Decreased metabolism (liver, kidney)

⊕ Washout during reperfusion

⊕ Sepsis

- ✱ Increase flux of alanine from muscle
- ✱ Decreased PDH activity
- ✱ Decreased hepatic clearance
- ✱ Dysfunctional mitochondrial respiration

Near Infrared Spectroscopy



⊕ Measurements

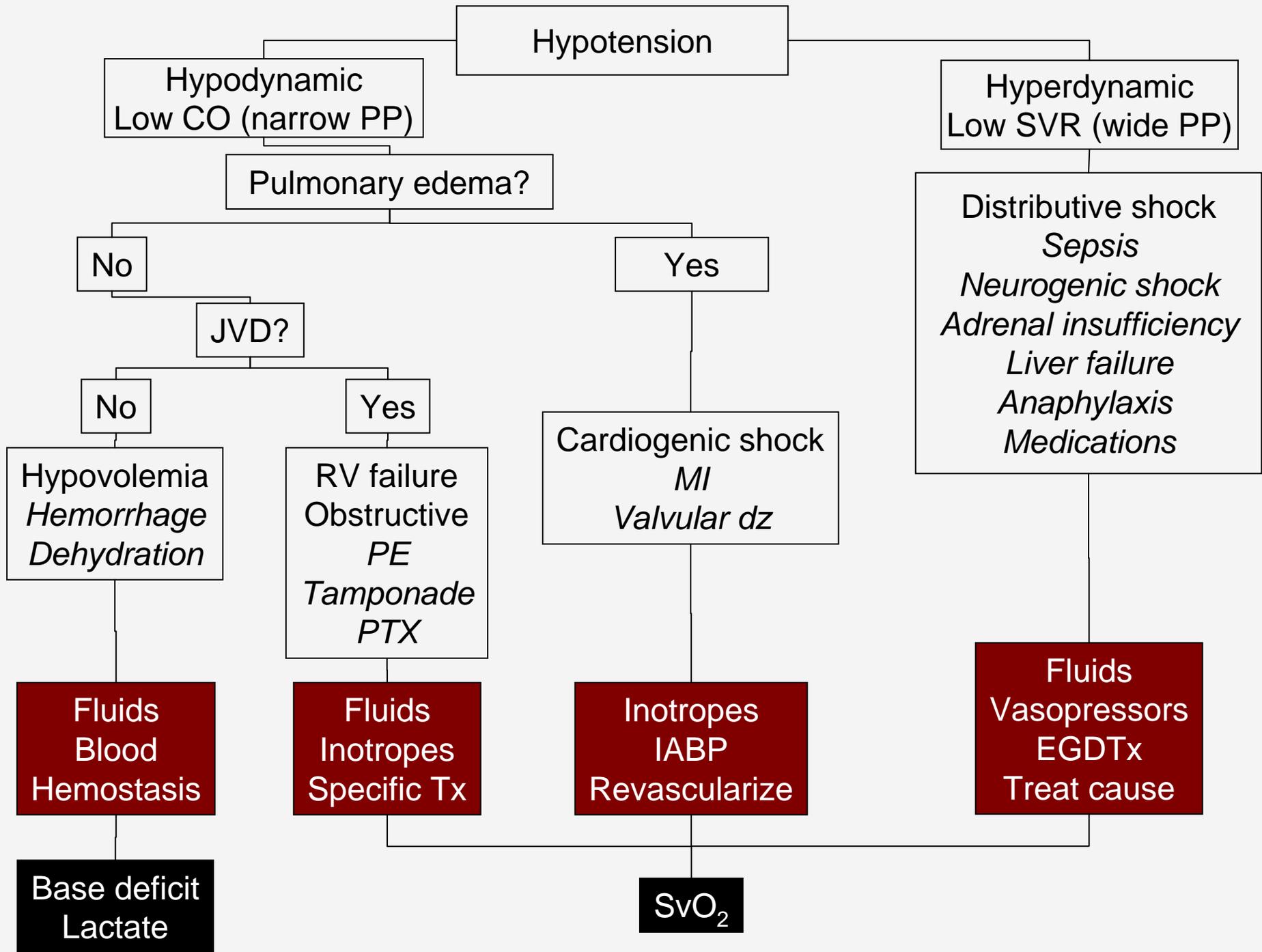
- ✿ PO₂
- ✿ PCO₂
- ✿ pH
- ✿ O₂ saturation of hemoglobin
- ✿ Tissue oxyhemoglobin coupling to cytochrome *a,a3* redox
 - ⊕ Mitochondrial O₂

⊕ Location

- ✿ Muscle
- ✿ Stomach
- ✿ Bowel
- ✿ Liver







Hypotension

Hypodynamic
Low CO (narrow PP)

Hyperdynamic
Low SVR (wide PP)

Pulmonary edema?

No

Yes

JVD?

No

Yes

Hypovolemia
Hemorrhage
Dehydration

RV failure
Obstructive
PE
Tamponade
PTX

Cardiogenic shock
MI
Valvular dz

Distributive shock
Sepsis
Neurogenic shock
Adrenal insufficiency
Liver failure
Anaphylaxis
Medications

Fluids
Blood
Hemostasis

Fluids
Inotropes
Specific Tx

Inotropes
IABP
Revascularize

Fluids
Vasopressors
EGDTx
Treat cause

Base deficit
Lactate

SvO₂

UPMC Critical Care



www.ccm.pitt.edu