



Why is my patient in shock?

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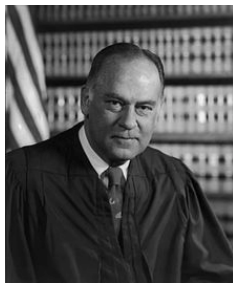
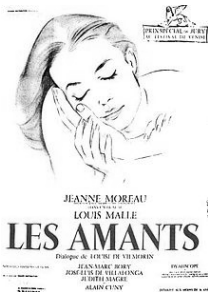
Case vignette

- You are called to the ER to see a 27 year old female with three days of nausea, vomiting, and anorexia. She has a history of AIDS and has recently started antiretroviral therapy. She is on dapsone for PCP prophylaxis. She is tachycardic with a mean arterial pressure of 55 mm Hg. She is confused.

Which of the following findings would indicate shock as the cause of the patient's presentation?

- Elevated venous lactate and central venous oxygenation
- Severe hemolytic anemia, with hemoglobin of 3
- Cool, clammy skin
- Any of the above

Jacobellis v. Ohio (1964)

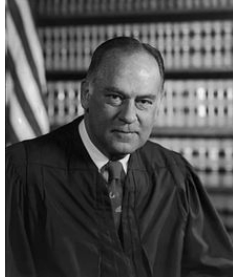


Justice Potter Stewart, 1964

Jacobellis v. Ohio (1964)

Regarding the definition of "hard-core pornography":

I shall not today attempt further to define the kinds of material I understand to be embraced within that shorthand description and perhaps I could never succeed in intelligibly doing so. But I know it when I see it, and the motion picture involved in this case is not that.

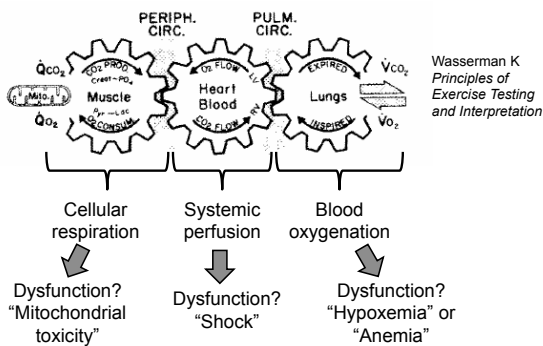


Justice Potter Stewart, 1964

What is shock?

- Inadequate tissue oxygen delivery, with resultant (multiple) organ dysfunction

Oxygen delivery to tissues



What is considered “shock”?

- Inadequate tissue oxygen delivery, perfusion, with resultant (multiple) organ dysfunction
- Determinants of tissue perfusion?
 - Cardiac output
 - Arteriolar tone (vascular resistance)

Impact of local vascular tone on organ perfusion?

- Autoregulation

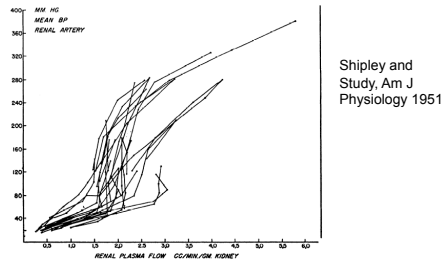
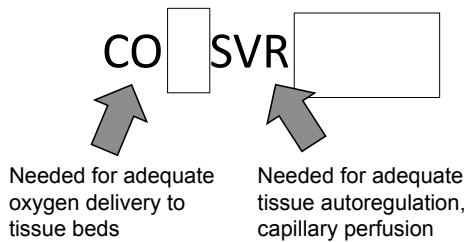


Fig. 2. EFFECT OF ALTERING RENAL ARTERY BP ON renal plasma flow in 13 experiments.

Main determinants of tissue perfusion?



Main determinants of tissue perfusion?

$$CO * SVR = MAP$$

“Ohm’s law”

“Shock” is manifested by loss of SVR or CO. As shock progresses, compensatory mechanisms fail, leading to loss of MAP.

Diagnosis of “shock”

1. Evidence of a systemic decrease in oxygen delivery
Elevated lactate, decreased central venous sats
2. Decrease in oxygen delivery is caused by loss of systemic tissue perfusion
Decreased CO or SVR (and, ultimately, MAP)
3. Dysfunction of multiple organs (suggesting systemic effect)
Decreased UOP, mental status, etc.

Case vignette

- You are called to see a 85 year old nursing home resident sent to the ER for “failure to thrive”. He is confused; physical examination reveals cool, clammy extremities. He is tachycardic and hypotensive. Laboratories reveal an elevated BUN/Cr.

Which of the following best establishes hypovolemia as the cause of his shock?

1. TTE reveals IVC respiratory variation > 12%
2. Flat neck veins
3. No evidence of pulmonary edema
4. Improved urine output after fluid bolus

Causes of shock

$$CO * SVR = MAP$$



Loss of cardiac output

1. Cardiogenic shock
2. Hypovolemic shock

Loss of SVR

3. Distributive shock

1. Cardiogenic shock

- Chronotropic heart failure
 - Low HR
 - Compensated by high stroke volume, high SVR
- Inotropic heart failure
 - Low stroke volume
 - Compensated by high HR, high SVR
 - Right heart failure
 - May be systolic (massive PE, infarction) or diastolic (tamponade)
 - Left heart failure
 - May be systolic (MI, cardiomyopathy, valve rupture, critical AS) or diastolic (LVH, tamponade, tachycardia)

2. Hypovolemic shock

- Lack of volume leads to inadequate preload, loss of cardiac output
 - Elevated heart rate, elevated systemic vascular resistance
 - Often coexists with distributive shock (vascular leak)
- Major difference from cardiogenic shock?
 - Absence of congestion/fluid overload on exam

What about echocardiography?

- Changes management in 24 – 51% of cases Orme Br J Anesth 2009
 - Evaluate cause of cardiogenic shock Noritomi et al Shock 2010
 - Can rule out tamponade, massive PE
 - Estimates RV/LV function, cardiac output
 - Evaluate hypovolemic shock
 - IVC diameter variation > 12% on TTE predicts fluid responsiveness in ventilated septic patients Feissel Int. Care Med 2004
- TTE images often limited in ventilated patients
 - 45% TTEs suboptimal Vignon Chest 1994
 - TEE results had more impact; can be performed safely
- No prospective data exists in care of critically ill

Case vignette

- You admit a 42 y/o female with obstructive pyelonephritis. After percutaneous nephrostomy placement, the patient becomes hypotensive and febrile. She is tachycardic, and her MAP is 50 mm Hg even after goal-directed crystalloid infusion. An echocardiogram reveals moderately depressed LV systolic function.

What is the first agent you should try to improve her blood pressure?

1. Phenylephrine
2. Dobutamine
3. Norepinephrine
4. Dopamine

Causes of shock

$$CO * SVR = MAP$$



Cardiogenic shock?

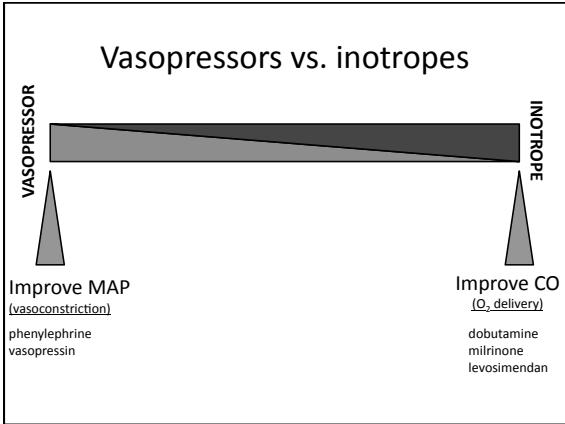
Loss of CO
(Compensatory increase in SVR)

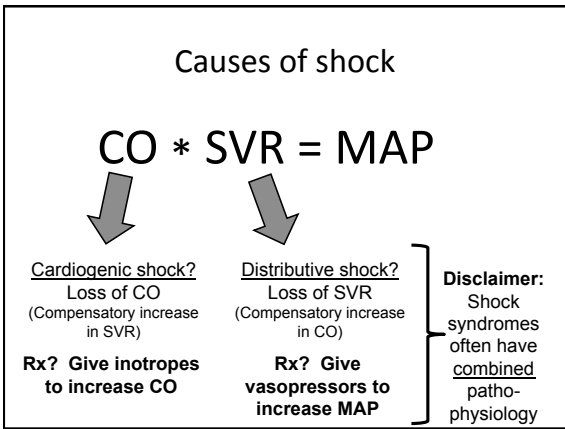
Rx? Give inotropes to increase CO

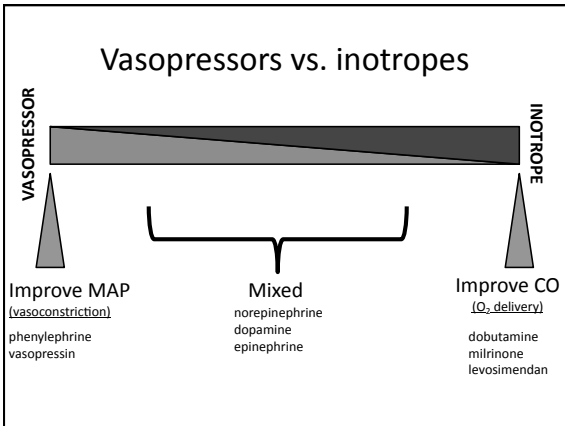
Distributive shock?

Loss of SVR
(Compensatory increase in CO)

Rx? Give vasopressors to increase MAP







Choice of (mixed) vasopressor in undifferentiated shock?

- Patient should be (ideally) euvolemic before vasopressors started

– De Backer et al. NEJM 2010

- Randomized patients with shock to NE or dopamine
- 28 day survival trend favoring NE (p=0.07)
- Dopamine: increased tachycardia

Dopamine is AHA/ACC first line agent for post-MI hypotension! JACC 2004

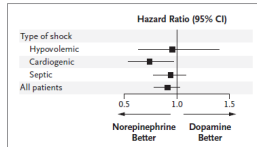


Figure 3. Forest Plot for Predefined Subgroup Analysis According to Type of Shock.

A total of 1044 patients were in septic shock (542 in the dopamine group and 502 in the norepinephrine group), 280 were in cardiogenic shock (135 in the dopamine group and 145 in the norepinephrine group), and 263 were in hypovolemic shock (138 in the dopamine group and 125 in the norepinephrine group). The P value for interaction was 0.87.

Evidence-based choice of vasopressor/inotrope in shock?

- Septic shock

– Early goal-directed therapy (Rivers NEJM 2001)

- 1) Volume resuscitate to goal CVP
- 2) Give vasopressors to correct MAP > 65 mm Hg
 - Surviving sepsis campaign: Dellenger et al. Inten Care Med 2008
 - » Dopamine or norepinephrine 1st line agents
 - » Phenylephrine/vasopression monotherapy discouraged due to negative inotropy
 - No clear benefit from addition of vasopressin to norepinephrine (may be beneficial the in less-sick) Russell NEJM 2008
- 3) Give inotropes (or blood) to improve oxygen delivery
 - Dobutamine used in Rivers study
 - No difference in epinephrine vs. norepinephrine/dobutamine Annane Lancet 2007

Evidence-based choice of vasopressor/inotrope in shock?

- Cardiogenic shock (decreased stroke volume)?

– Inotropes—increase myocardial cAMP

- Beta agonists (dobutamine)
 - PDE III inhibitors (milrinone)
- } No studies comparing two agents

– Active cardiac ischemia?

- IABP
 - Grade IB evidence in ACC/AHA guidelines Antman JACC 2004
 - Unclear benefit post-MI if emergent PCI already performed Prondzinsky et al. Crit Care Med 2010

- Vasodilating agents
 - Levosimendan –calcium “sensitizer” (not yet FDA approved)
 - » no benefit vs. dobutamine in acute CHF Mebazaa et al. JAMA 2009

– Refractory hypotension: Dopamine (caution!) vs NE

Evidence-based choice of
vasopressor/inotrope in shock?

- Cardiogenic shock (decreased HR)?
 - Pacing (per ACLS)
 - Isoproterenol
 - Calcium channel blocker/beta blocker overdose?
 - Drug-specific antidote (calcium, insulin, glucagon)
 - If refractory, lipid emulsion therapy
- Anaphylactic shock
 - Epinephrine is most important therapy
 - Bimodal time course
