

APPROACH TO UNDIFFERENTIATED SHOCK by Nick Mark MD



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Shock occurs when there is inadequate blood flow (CO) & oxygen delivery (DO2) to meet demands. Manifestations can be protean and may not initially include hypotension (**cryptic shock**). Identifying the etiology of undifferentiated shock is essential to determine treatment.

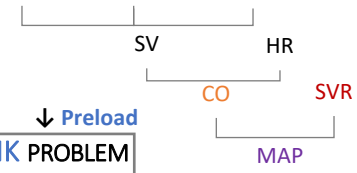
Shock can be broken into 4 categories: **cardiogenic, obstructive, distributive, hypovolemic**

Multiple causes may be present (e.g. sepsis in a patient with decompensated heart failure) and some etiologies may cause **mixed shock**:

- **Endocrine** (adrenal insuf., myxedma, thyrotoxicosis)
- **Metabolic** (hypothermia severe acidosis)

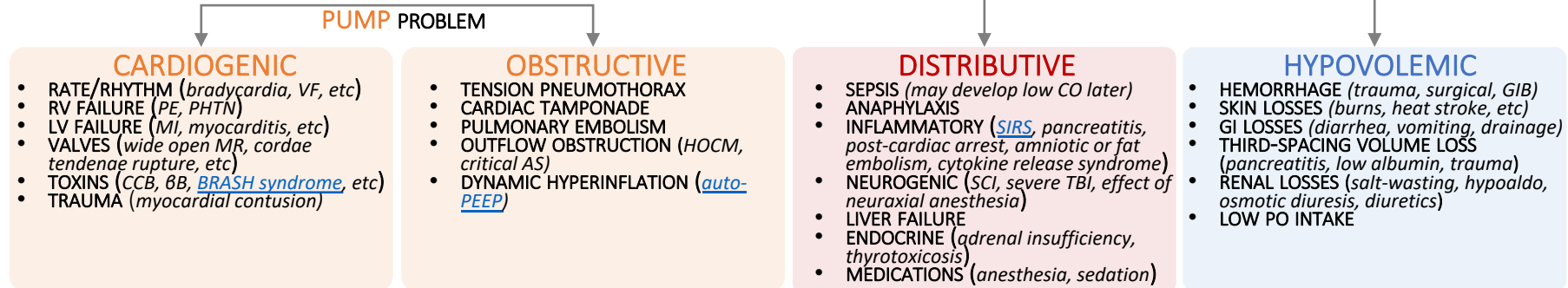
Undifferentiated SHOCK
Hypotension (not required)
Organ dysfunction (AKI, shock liver, etc)
Altered mental status
Lactic acidosis
Low urine output

MAP DETERMINANTS:
Preload Contractility Afterload



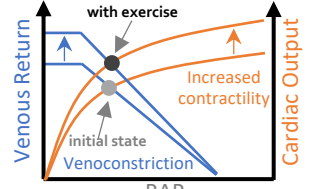
ETIOLOGY

EXAM & POCUS & HEMODYNAMICS
(see RUSH exam for more about POCUS in Shock)

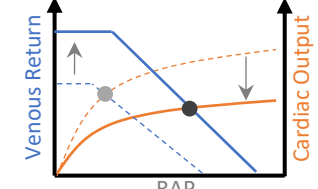


HD	↑CVP, ↑PCWP, ↓CO, ↑SVR	↑CVP, ↑PCWP, ↓CO, ↑SVR	var CVP, var PCWP, var CO, ↓SVR	↓CVP, ↓PCWP, ↑CO, ↑SVR
Heart	± Reduced contractility ± RV dilation ± Wall motion abnormalities ± Valvulopathy	Reduced contractility RV dilation (PE) ± septal D sign (p/v overload) Pericardial effusion, RA collapse (tamponade)	Hyperdynamic (hypodynamic in late sepsis)	Hyperdynamic
IVC	Plethoric IVC, reversal of flow in HV	Plethoric IVC, reversal of flow in HV	Variable IVC	Small/collapsing IVC
Lungs	B-line pattern + pleural effusions	Lack of lung sliding ± lung point (PTX)	A line pattern	A line pattern
Other	Pleural effusions (LV failure)	DVT or clot in transit (PE)	Evidence of infxn (cholecystitis, endocarditis, etc), cirrhosis,	Blood or fluid in abdomen (FAST), Ectopic pregnancy, Aortic dissection,
Skin	Usually cool, Delayed cap refill	Usually cool, Delayed cap refill	Warm, flushed, Brisk cap refill	Usually cool, Delayed cap refill
Neck	Increased JVP	Increased JVP	Variable	Flat neck veins
Other	Weak pulses (narrow pulse pressure)	Weak pulses (narrow pulse pressure) Lung and heart sounds are unreliable indicators of tamponade or PTX.	Bounding pulses (wide pulse pressure)	Weak pulses (narrow pulse pressure) Evidence of blood (pallor) or volume loss (axillary dryness)

PHYSIOLOGIC RESPONSES TO SHOCK USING GUYTON CURVES:

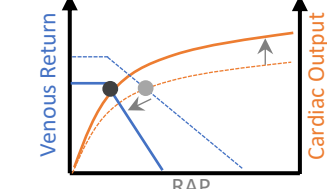


Normally cardiac output (CO) determined by venous return & contractility

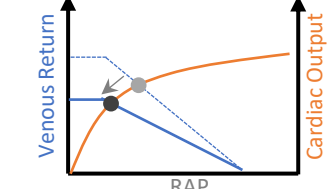


CARDIOGENIC/OBSTRUCTIVE with low CO, RA filling pressures rise to (partially) compensate

(See Guyton Curves in Shock OnePager for more)



DISTRIBUTIVE vasodilation decreases filling, hyperdynamic CO compensates



HYPOVOLEMIC with low preload, venoconstriction increased filling pressure compensates

CALCULATING SVR:

SVR can be useful to understand etiology. You can either measure CO invasively (e.g. PAC) or estimate using POCUS (e.g. LVOT VTI)

$$MAP = CO \times SVR$$

$$SVR = \frac{(MAP - CVP)}{CO} \times 80$$

Normal SVR = 800 – 1600 dyn/cm/sec⁵ = 10 – 20 Wood units

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