Management of Shock in Critical Care

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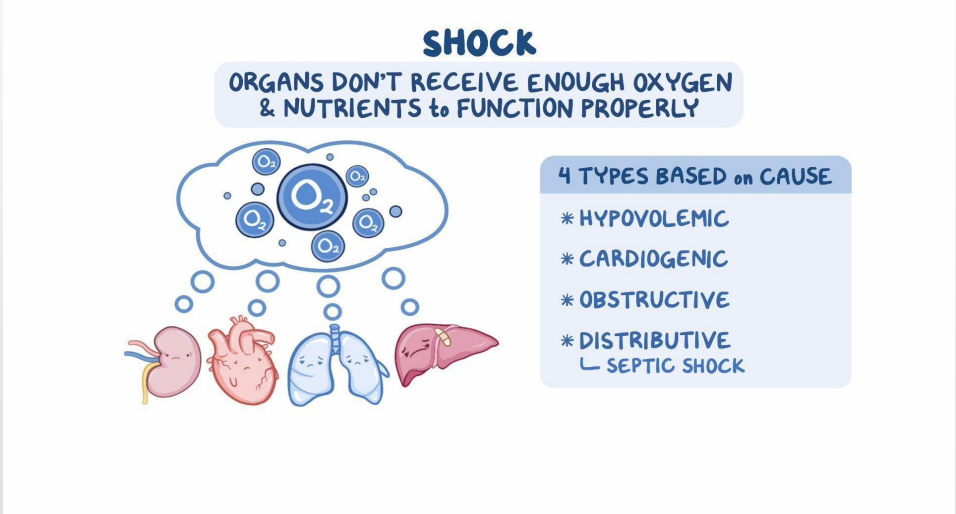
Objectives

- Introduction to shock
- Pathophysiology of shock in critical care
- Diagnose early stages of shock
- Provide initial treatment & stabilization
- Correctly interpret hemodynamic monitoring
- Prevent complications associated with shock

Back to the basics

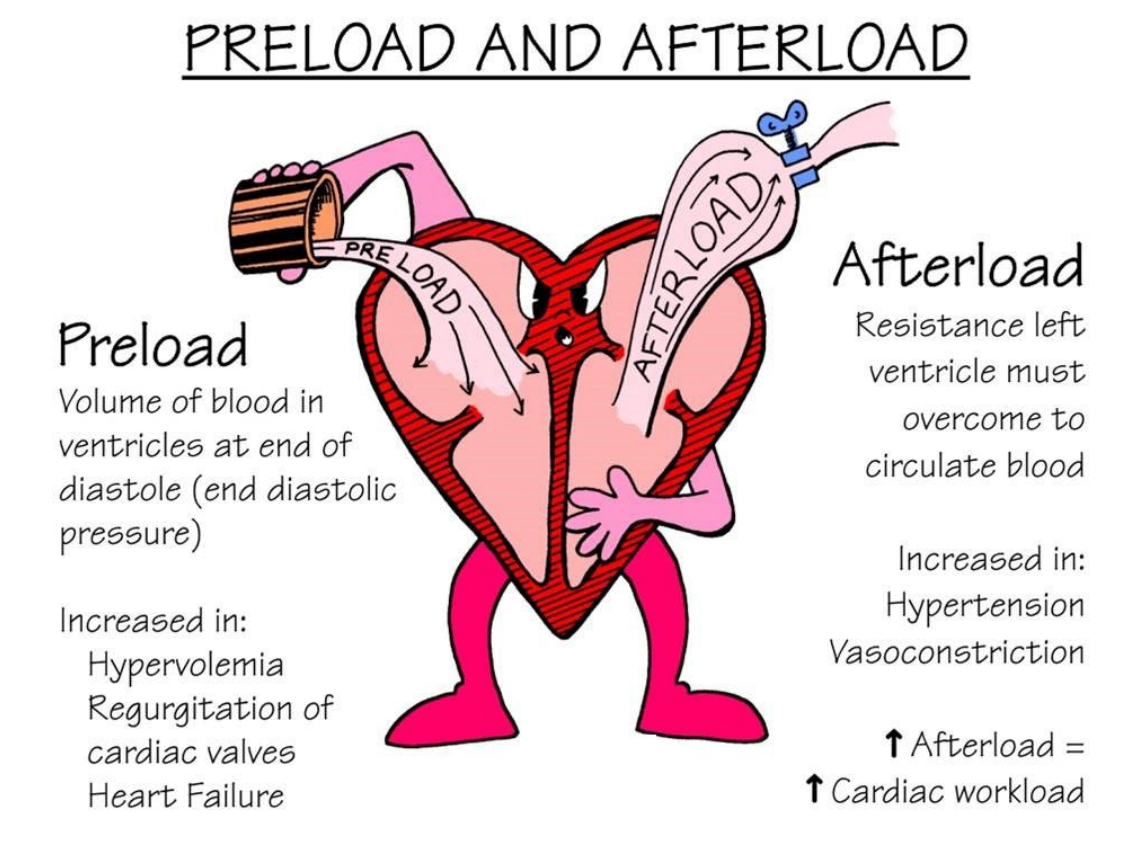
- What is shock?
  - Life-threatening
  - A state of cellular & tissue hypoxia due to either reduced oxygen delivery, increased oxygen consumption, inadequate oxygen utilization, or a combination
  - Essentially inadequate blood flow/tissue perfusion



Pathophysiology of Shock

- Cellular hypoxia from insult (hypovolemia, infection, tamponade, MI, etc…) leads to reduced tissue perfusion
- Tissue perfusion is dictated by systemic blood pressure = CO x SVR
- CO = Heart rate x stroke volume
  - Stroke volume determined by preload, myocardial contractility, afterload
- Therefore-any process that alters or effects one of these parameters can lead to hypotension and ultimately shock

Hemodynamics in Shock



Hemodynamics in Shock

Hemodynamic profiles of shock on pulmonary artery catheter in adults

Physiologic variable	Preload	Pump function	Afterload	Tissue perfusion
Clinical measurement	**Pulmonary capillary wedge pressure**	**Cardiac output***	**Systemic vascular resistance**	**Mixed venous oxyhemoglobin saturation¶**
Hypovolemic	↔ (early) or ↓ (late)	↔ (early) or ↓ (late)	↑	>65% (early) or <65% (late)
Cardiogenic	↑	↓	↑	<65%
Distributive	↔ (early) or ↓ (late)	↑ or ↓ (occasionally)	↓	>65%
Obstructive				
PE, PH, tension pneumothorax	↔ (early) or ↓ (late)	↔ (early) or ↓ (late)	↑	>65%
Pericardial tamponadeΔ	↑	↓	↑	<65%

PE: pulmonary embolus; PH: pulmonary hypertension; PAC: pulmonary artery catheter.

* Cardiac output is generally measured using the cardiac index.

¶ Mixed venous oxyhemoglobin saturation cutoff measured on PAC is 65%, but on triple lumen catheter is 70%.

Δ Equalization of right atrial, right ventricular end-diastolic and pulmonary artery wedge pressures is classic in pericardial tamponade and distinguishes it from primary cardiogenic shock.

Early Stages of Shock

- Management of shock in the critical care setting involves prompt recognition & treatment to restore adequate tissue perfusion & oxygenation
- Early stages = compensation
  - Tachycardia
  - Slight change in systemic BP
  - Mild to moderate elevated lactic

Types of Shock

- Distributive
  - Severe peripheral vasodilatation, low SVR
    - Examples: Septic (most common), neurogenic, anaphylactic
- Hypovolemic
  - Due to reduced intravascular volume (reduced preload) which in turn reduces CO
    - Can be divided into 2 categories
      - Hemorrhagic
      - Non-hemorrhagic (dehydration, GI losses, skin losses, renal losses, third spacing)

Types of Shock

- Cardiogenic
  - INTRAcardiac pump failure resulting in decreased CO
    - Examples: MI, arrhythmias, cardiac arrest, acute heart failure, acute valvular defects, severe aortic or mitral valve insufficiency
- Obstructive
  - EXTRAcardiac causes of pump failure & often poor RV output
    - Can be divided into 2 categories
      - Pulmonary vascular (PE, severe pulmonary HTN)
      - Mechanical (tension PTX, pericardial tamponade, constrictive pericarditis)

Treatment of Obstructive Shock

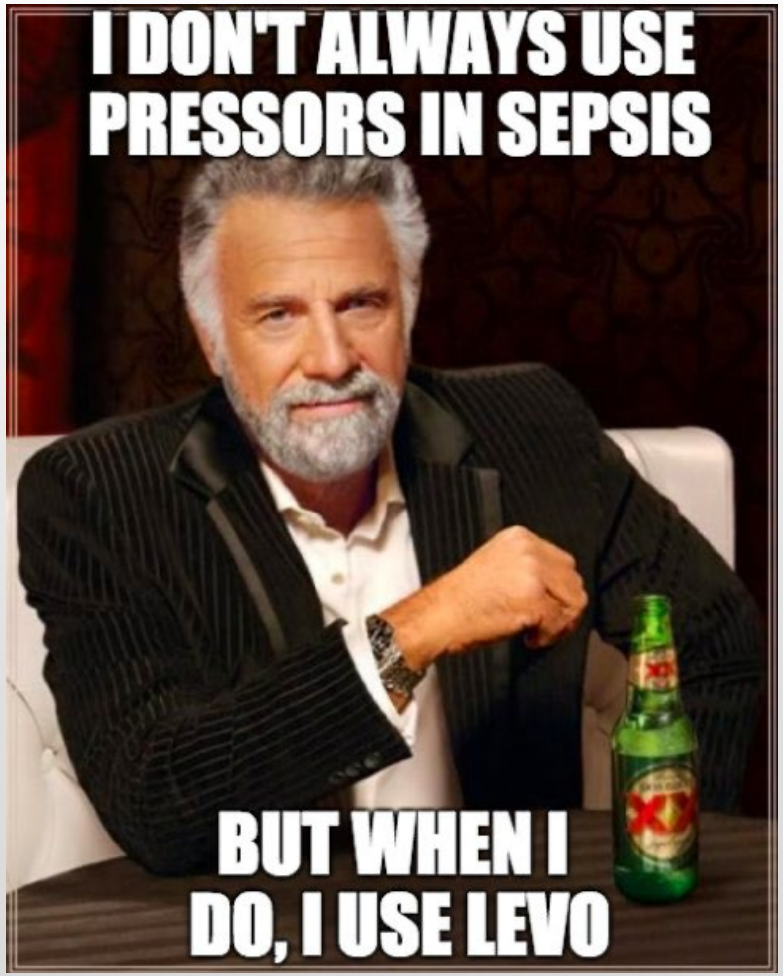
- **Relieve obstruction**
  - Tension PTX
    - Needle decompression &/or chest tube placement
  - Cardiac tamponade
    - Pericardiocentesis
  - Massive PE
    - Thrombolysis or thrombectomy

Treatment of Distributive Shock

- Anaphylactic shock treatment
  - 1st line: Epinephrine
  - IVFs, antihistamines, & corticosteroids
  - Intubation if concern for airway swelling
- Neurogenic shock treatment
  - IVF resuscitation
  - Pressor support
    - 1st Line: Norepinephrine
  - Treat underlying SCI

Treatment of Septic Shock (Distributive)

- Septic shock:
  - Fluid resuscitation
    - 30cc/kg in first 3 hrs
  - Broad spectrum antibiotics w/in 1hr
  - Pressors if needed to maintain MAP>65
    - Norepinephrine = first line
  - Corticosteroids if refractory to fluids & pressors
    - Consider cortisol level



Methylene Blue

- Inhibits nitrous oxide synthesis and therefore decreases vascular smooth muscle relaxation
- Increases oxygen-binding capacity of hemoglobin –hus increasing oxygen delivery to tissues
- May reduce short-term mortality, duration of pressors, & hospital LOS
- Monitor for: GI distress, serotonin syndrome, and bluish discoloration of urine
- G6PD can worsen hemolytic anemia

The good, the bad, and the neo of it all…

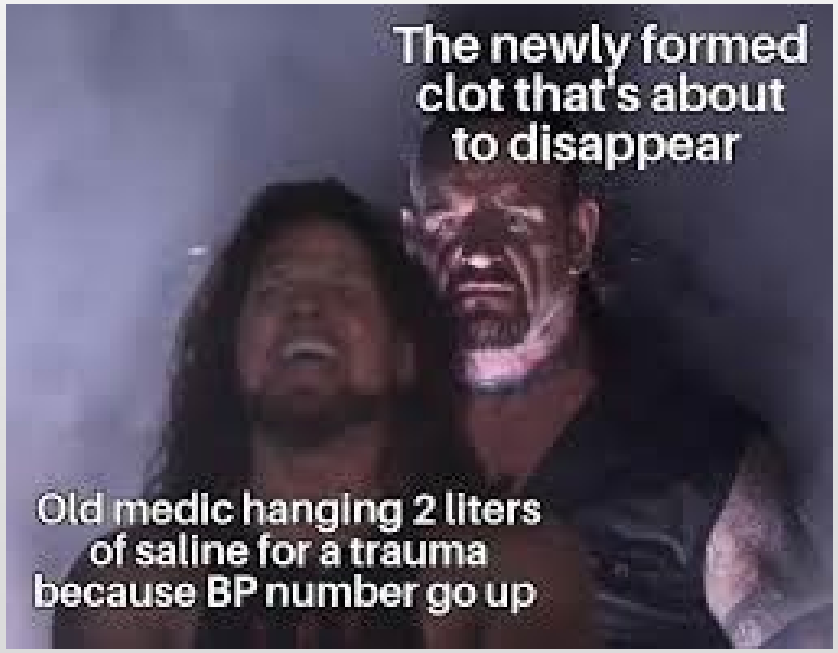
- Neosynephrine in shock has fallen out of favor as first line vasopressor
- Pure alpha-adregeneric vasoconstrictor
  - Can be used in patients with tachyarrythmias
  - Caution for (profound) reflex bradycardia
  - Caution in patient's with significant, known heart disease
- Still used as alternative/additional agent

Treatment of Hypovolemic Shock: Non-hemorrhagic

- Goal: Restore circulating intravascular volume to improve preload, cardiac output & tissue perfusion; treat underlying cause
- Rapid isotonic crystalloid fluid resuscitation
- Consider colloid in burn patients who may have significant plasma losses or in patients with liver disease
- Hypovolemic shock primarily requires fluids so pressors should be used cautiously, especially in hemorrhagic shock

Treatment of Hypovolemic Shock: HEMORRHAGIC

Limit crystalloid



Stop the Bleed

- Apply direct pressure, tourniquet if necessary
  - Timing of tourniquet placement
- Give blood, clotting factors as needed
  - Whole blood preferred if available
  - 1:1:1 or balanced resuscitation
  - Monitor TEG & use to guide resuscitation
- Replete calcium
- Consider TXA in trauma-related hemorrhage (w/in 3 hrs of injury)

Stop the Bleed

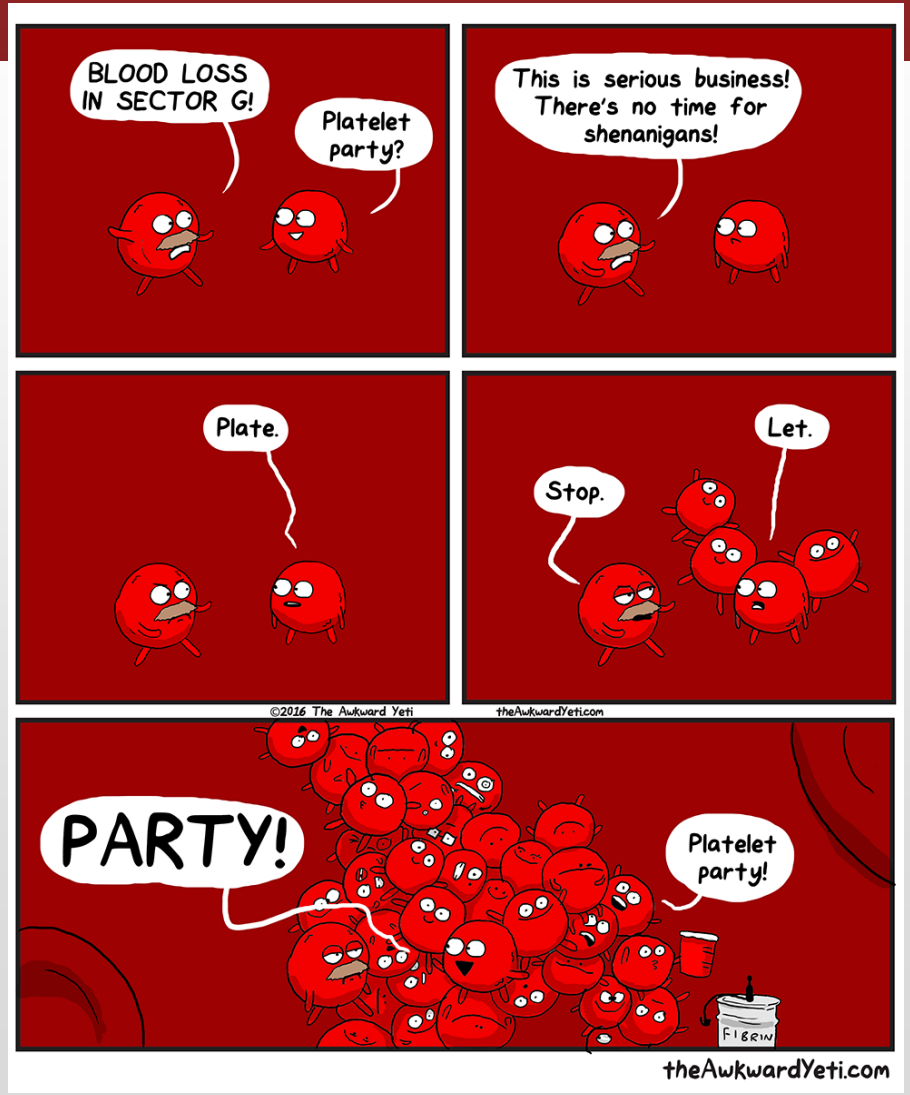
- Anticoagulation reversal if indicated
- Permissive hypotension
- Consider angio, endoscopy, surgical intervention if necessary
- Treat hypothermia

Guiding resuscitation in shock

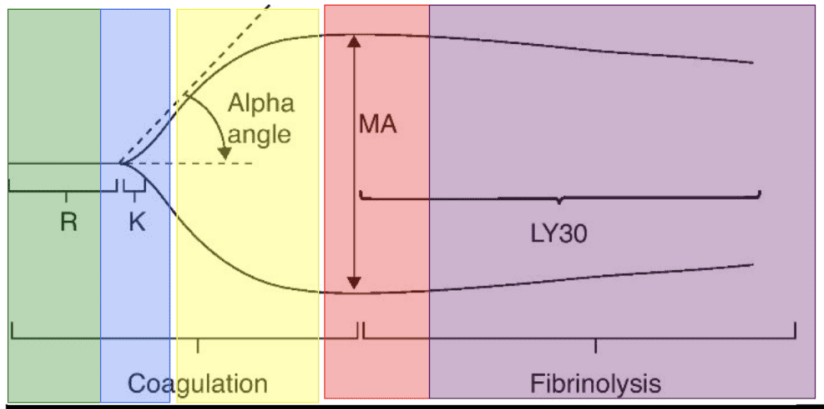
- Reassess frequently/monitor response to interventions
  - HR, BP, pressor requirements
  - UOP
  - lactic, ABG
- Complications:
  - Metabolic acidosis
    - If severely acidotic, bicarb pushes/gttp
  - Coagulopathy with significant blood loss
  - Electrolyte imbalances

What is TEG?

- A test that measures how blood clots form, stabilize/strengthen, & breakdown
- Incorporates platelet function vs just a platelet count
- Essentially offers a "global" view of clotting versus coagulation tests focus on specific coagulation factors in the plasma
- Results are displayed graphically in real-time



TEG

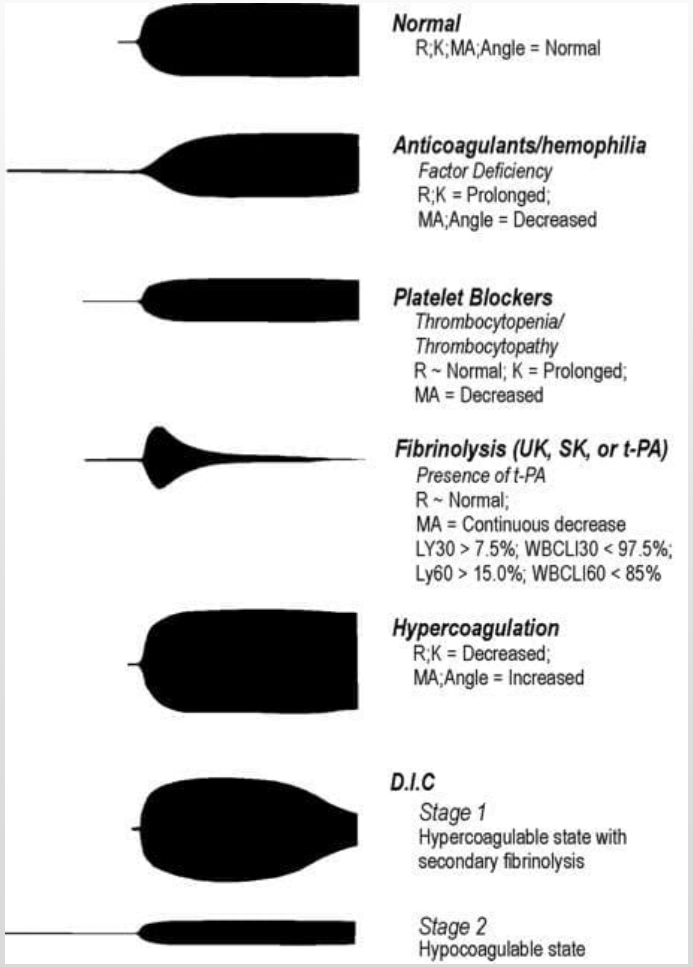


REBEL REVIEWS

Thromboelastogram (TEG)				
Components	Definition	Normal Values	Problem with...	Treatment
R Time	Time to start forming clot	5 - 10 minutes	Coagulation Factors	FFP
K Time	Time until clot reaches a fixed strength	1 - 3 minutes	Fibrinogen	Cryoprecipitate
Alpha angle	Speed of fibrin accumulation	53 - 72 degrees	Fibrinogen	Cryoprecipitate
Maximum Amplitude (MA)	Highest vertical amplitude of the TEG	50 - 70 mm	Platelets	Platelets and/or DDAVP
Lysis at 30 Minutes (LY30)	Percentage of amplitude reduction 30 minutes after maximum amplitude	0 - 8%	Excess Fibrinolysis	Tranexemic Acid and/or Aminocaproic Acid

Source: Rebelem.com

TEG interpretation



Source: LIFTL

Cryoprecipitate

- Derived from FFP
- More concentrated = more clotting factors
- Usually contains:
  - Fibrinogen (factor I)
  - Factor VIII
  - Factor XIII
  - von Willebrand factor
  - Fibronectin

?Case study

- Trauma & SCI patient
- Include POC: how to resuscitate, line placement
- Include POCUS-> is my patient resuscitated?? Consider type of injury -> ptx? FAST?
- 2nd case study: sepsis patient
- Vasopressor of choice, line placement
- POCUS?
- …. It doesn't work? What next?

- EOL, palliative care

Questions

References