Statewide Sepsis Initiative
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APPROPRIATE FLUID RESUSCITATION

WHEN IS IT ENOUGH, AND IS THERE A PATIENT POPULATION THAT SHOULD BE EXCLUDED?
Types of Shock

- ED physicians default to Cardiogenic Shock until proven otherwise

- are not always considering the four other general categories of shock at presentation. (Distributive, Hypovolemic, Obstructive and Combined)

- These 5 general categories can further be broken down to multiple etiologies and pathophysiologies with multiple mechanisms and stages of shock.
Distributive Shock
Distributive shock is characterized by hypotension (systolic blood pressure <90 mm Hg) due to a severe reduction in systemic vascular resistance (SVR), with normal or elevated cardiac output in most instances.
Causes of shock

- Septic shock is the most commonly encountered form of distributive shock.

- Is now the most common cause of non-cardiac death in intensive care units (ICUs) in the US.
Other causes of distributive shock

- Systemic inflammatory response syndrome (SIRS) due to noninfectious inflammatory conditions.
- Toxic shock syndrome (TSS).
- Anaphylaxis; drug or toxin reactions.
- Transfusion reaction.
- Addisonian crisis; hepatic insufficiency; and neurogenic shock due to brain or spinal cord injury.
Pathophysiology:

- Decreased tissue perfusion in distributive shock results primarily from arterial hypotension caused by a reduction in SVR.

- In addition, a reduction in effective circulating plasma volume often occurs due to a decrease in venous tone and subsequent pooling of blood in venous capacitance vessels.
Pathophysiology:

- Loss of intravascular volume into the interstitium due to increased capillary permeability also occurs.
- Eventually, primary myocardial dysfunction often is present as manifested by ventricular dilatation, decreased ejection fraction (despite normal stroke volume and cardiac output), and depressed ventricular function curves.
The hemodynamic derangements observed in septic shock and SIRS are due to a complicated cascade of inflammatory mediators released in response to infection, inflammation, or tissue injury.
Pathophysiology: In anaphylaxis

- In anaphylaxis, decreased SVR is due primarily to massive histamine release from mast cells after activation by antigen-bound immunoglobulin E (IgE), as well as increased synthesis and release of prostaglandins.
Pathophysiology of septic shock and SIRS

- Tumor necrosis factor-alpha (TNF-alpha), interleukin (IL)-1b, and IL-6 act synergistically with other cytokines and phospholipid-derived mediators to produce the complex alterations in vascular and myocardial function, which leads to maldistribution of blood flow.
A Multidisciplinary Community Hospital Program for Early and Rapid Resuscitation of Shock in Nontrauma Patients

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Intravascular Volume

- Euvolemic vs Hypovolemic vs Hypervolemia
- Intravascular volume (IV)
- Difficult to assess clinically
- IV effects venous return (VR) which effects stroke volume (SV) which effects cardiac output (CO)

- \( \text{SV} \times \text{HR} = \text{CO} \)

- Preload (VR) decreased with low systemic vascular resistance (SVR) seen with sepsis (leukotrienes, cytokines, TNF, etc. and usually normal or increased pump function)

- End result=septic shock
Approach to Shock

▪ “Importantly, resuscitative efforts, particularly intravenous fluids, should not be delayed for a detailed clinical assessment, nor should clinicians be conservative in terms of fluid resuscitation to patients with heart failure or kidney injury as a rule. Related to this latter point we know, liberal fluid resuscitation is most certainly of paramount importance in order to save lives in septic patients even with intermediate serum lactate levels, a benefit derived amongst these traditionally under resuscitated sepsis subgroups.” Burton and Rose

▪ ACEP literature supports this so Why aren’t we practicing this??
Treament of Shock

- shock is complex and can have dozens of etiologies but ultimately it boils down to very elementary resuscitative efforts of A,B,C (Airway, Breathing and Circulation).