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Shock and Pressors

During my time at the VA MICU, I experienced a vast amount of patients that presented to the ED in some form of shock. It is estimated that more than one million patients a year are requiring emergent resuscitation. When a patient presents with persistent hypotension, an understanding of the different forms of shock is imperative in initiating the appropriate management.

Shock is defined as **impaired tissue perfusion and oxygenation causing organ dysfunction**. Clinical markers of shock that can be seen include **lactate >2, urine output < 0.5cc/kg/hr, altered mental status, evidence of myocardial ischemia, and mottling of skin**. Early shock may have normal systolic and diastolic blood pressures due to compensatory mechanisms; therefore, **tachycardia** may be the first sign of shock. There are four types of shock that are important to consider in the persistently hypotensive patient (SBP <90). These are: **distributive, hypovolemic, obstructive, and cardiogenic**. Each form differs in hemodynamic properties (cardiac output, systemic vascular resistance, and pulmonary capillary wedge pressure) but all lead to shock through different mechanisms. It is key to remember that BP = CO x SVR (CO = HR x SV).

Distributory
-Sepsis, Neurogenic impairment, Anaphylaxis
-High CO, Low SVR
-Low PCWP

Hypovolemic
-Hemorrhage, Dehydration
-Low CO, High SVR
-Low PCWP

Cardiogenic
-Acute MI, Decompensated HF, Arrhythmia
-Low CO, High SVR
-High PCWP

Obstructive
-Tension PTX, Tamponade, Massive PE
-Low CO, High SVR
-High PCWP

The goals to treating shock are to **maintain perfusion to vital organs, increase MAP/CO, and to correct metabolic derangements**. Fluid resuscitation, commonly with isotonic crystalloids, is critical in patients with hypovolemic shock and sepsis. Once adequate intravascular volume expansion has been achieved (2-4L), vasopressor administration is required for persistent hypotension. Different vasopressors work on receptors found in peripheral vasculature and myocardium to produce CO and SVR changes. Stimulation of alpha 1 receptors produce vasoconstriction, while stimulating beta 2 receptors causes vasodilation. Beta 1 receptors are found in the myocardium and produce an inotropic effect. The following table of vasopressors/inotropes shows the primary impact, side effects and form of shock each is used in.

Vasopressor/ Inotrope	Primary Receptor	Primary Impact	Potential side effect	Clinical scenario used

Phenylephrine	**α_1**	Increased SVR, No inotropy	Reflexive decreased HR	Neurogenic shock **Sepsis + arrhythmias**
Vasopressin	V (smooth muscles)	Increased SVR	Decreased splanchnic flow	**Adjunct for septic shock With Norepinephrine**
Dobutamine	**β_1, β_2** > α_1	Increased inotropy	Transient decreases in SVR (β_2 agonism), **Decreases BP**	**Cardiogenic shock**
Dopamine (low-dose)	**D, β_1**	Increased inotropy and heart rate	Tachyarrhythmias	Cardiogenic shock, particularly if bradycardic
Epinephrine	**α_1, α_2, β_1, β_2**	Increased SVR and inotropy	Tachyarrhythmias and decreased splanchnic flow	**Anaphylaxis**, Septic shock
Norepinephrine	**α_1, α_2, β_1**>> β_2	Increased SVR and inotropy	Decreased splanchnic and renal flow Reflex bradycardia	**Septic shock**
Dopamine (high-dose)	D, **α_1**, β_1>> β_2*	Increased SVR and inotropy	Tachyarrhythmias and decreased splanchnic/renal flow	Bradycardic Hemorrhagic Septic shock (3rd line)

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3. Seigel, Todd A., MD. "Choosing the Right Vasopressor Agent in Hypotension." *ALiEM*. N.p., 20 Aug. 2013. Web. 26 Sept. 2014.