Shock, Swans, Pressors in 15 minutes

- 4 Reasons for Shock
- 4 Swan numbers to know
- 7 Pressors

=15 things to know
4 Reasons for Shock

- Not enough preload
- Not enough afterload
- Not enough contractility
- Cardiac obstruction
4 Reasons for Shock

- Not enough preload (Hypovol., Hemor.)
- Not enough afterload
- Not enough contractility
- Cardiac obstruction
4 Reasons for Shock

- Not enough preload (Hypovol., Hemor.)
- Not enough afterload (Neurogenic, Septic)
- Not enough contractility
- Cardiac obstruction
4 Reasons for Shock

- Not enough preload (Hypovol., Hemor.)
- Not enough afterload (Neurogenic, Septic)
- Not enough contractility (Cardiogenic)
- Cardiac obstruction
4 Reasons for Shock

- Not enough preload (Hypovol., Hemor.)
- Not enough afterload (Neurogenic, Septic)
- Not enough contractility (Cardiogenic)
- Cardiac obstruction (Obstructive)
4 Swan Numbers to Know

- SvO2
- EDVI
- CI
- SVR/SVRI
SvO2: Overall Picture

Normal 60-80%
SvO2: Overall Picture

Normal 60-80%

SvO2 = O2 delivery - O2 consumption
SvO2: Overall Picture

Normal 60-80%

SvO2 = O2 delivery - O2 consumption
SvO2 = O2 content × CO – consumption
SvO2: Overall Picture

Normal 60-80%

\[ SvO2 = O2 \text{ delivery} - O2 \text{ consumption} \]

\[ SvO2 = O2 \text{ content} \times \text{CO} - \text{consumption} \]

\[ SvO2 = \text{SaO2} \times \text{Hct} \times \text{CO} - \text{consumption} \]
EDVI: Preload

Normal 100-120

Requires a special type of Swan.

Can also use CVP, PCWP to gauge preload
CI: Contractility

Normal 2.5-4.0

If your preload and afterload are fixed, CI gives an idea of contractility
SVR/SVRI: afterload

SVR Normal:  800-1200
SVRI Normal:  2000-2400
4 Reasons for Shock

- Not enough preload
- Not enough afterload
- Not enough contractility
- Cardiac obstruction
4 Treatments for Shock

- Not enough preload – give volume
- Not enough afterload – squeeze vessels
- Not enough contractility – squeeze heart
- Cardiac obstruction – relieve obstruction
4 Treatments for Shock

- Not enough preload – give volume
- Not enough afterload – squeeze vessels
- Not enough contractility – squeeze heart
- Cardiac obstruction – relieve obstruction
## Squeeze Vessels

<table>
<thead>
<tr>
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<tr>
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## Squeeze Heart

<table>
<thead>
<tr>
<th>Unsqueeze Vessels</th>
<th>No Vessel Squeeze</th>
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<tbody>
<tr>
<td>Milrinone</td>
<td>Dobutamine</td>
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Take Home Points

- SvO2, EDVI, CI, SVR tell you 95% of the important info on a Swan
- Make sure volume status is adequate before starting pressors
- Phenylepherine for sympathectomy only
- Dopamine or Levo for Sepsis, then add Vaso
- Dobutamine or Milrinone for contractility
- Epinephrine if all else fails
Definition of shock

- Reduction of systemic tissue perfusion, resulting in decreased oxygen delivery to the tissues.
- Prolonged oxygen deprivation leads to cellular hypoxia.
- The effects of oxygen deprivation are initially reversible, but rapidly become irreversible. The result is sequential cell death, end-organ damage, multi-system organ failure, and death.
Types of shock

- Hypovolemic/Hemorrhagic
  - loss of blood or plasma volume
- Cardiogenic
  - pump failure or compression
- Septic
  - toxin induced vasodilation
- Neurogenic
  - cervical or high thoracic (T1-T5) injury interrupts thoracic sympathetic outflow
## Physiologic parameters in shock

<table>
<thead>
<tr>
<th>Type of shock</th>
<th>CVP/PCWP</th>
<th>CO</th>
<th>SVR</th>
<th>Venous O2 Sat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic (including hemorrhagic)</td>
<td>↓</td>
<td>↓</td>
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<td>Cardiogenic</td>
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Receptor physiology

- **Alpha adrenergic** — Activation of alpha-1 adrenergic receptors, located in vascular walls, induces significant vasoconstriction.

- **Beta adrenergic** — Beta-1 adrenergic receptors are most common in the heart, and mediate increases in inotropy and chronotropy with minimal vasoconstriction. Stimulation of beta-2 adrenergic receptors in blood vessels induces vasodilation.

- **Dopamine** — Dopamine receptors are present in the renal, splanchnic (mesenteric), coronary, and cerebral vascular beds; stimulation of these receptors leads to vasodilation. A second subtype of dopamine receptors causes vasoconstriction by inducing norepinephrine release.
Norepinephrine (Levophed)

- Stimulates beta1 and alpha-adrenergic receptors.
- Clinically alpha effects (vasoconstriction) >> than beta effects (inotropic and chronotropic effects)
- Dose 1-30 mcg/min as a continuous infusion
- Used for treatment of shock which persists after adequate fluid resuscitation (think sepsis)
- Adverse reactions: bradycardia, digital ischemia, skin necrosis (with extrav)
- Give centrally
Vasopressin (Pitressin)

- ADH analog, increases cAMP, direct vasoconstrictor w/o inotropic/chronotropic effects
- Dose 0.01-0.04 units/minute. (Doses >0.04 with more dysrhythmias)
- Most case reports have used 0.04 units/minute continuous infusion as a fixed dose for the treatment of septic shock.
- Relative deficiency of plasma levels of ADH and relative hypersensitivity to its vasoconstrictive effects during sepsis.
- Causes mesenteric vasoconstriction, particularly at higher doses. Also skin necrosis.

Phenylephrine (Neosynephrine)

- Alpha-adrenergic stimulator with weak beta-adrenergic activity
- Produces systemic arterial vasoconstriction
- Initial dose: 100-180 mcg/minute, or alternatively, 0.5 mcg/kg/minute; titrate to desired response.
- Uses: neurogenic shock, hypotension after epidural placement.
- Adverse effect: increases afterload and decreases stroke volume
Dopamine

- Adrenergic and dopaminergic agonist
- Low-dose (dopaminergic): 1-5 mcg/kg/minute, increased renal blood flow and urine output, mesenteric dilation.
- Intermediate-dose (dopaminergic and beta1): 5-15 mcg/kg/minute, increased renal blood flow, heart rate, cardiac contractility, and cardiac output.
- High-dose (alpha-adrenergic predominates): >15 mcg/kg/minute vasoconstriction, increased blood pressure.

- Good for sepsis or cardiogenic shock.
- Can use on 9N or 10S! Renal Tx service uses this for hypotension.
- If extravasates, short half life (2 minutes) means you can just withdraw the drug. Less likely to cause skin necrosis.

Lauschke A; Teichgraber UK; Frei U; Eckardt KU, “Low-dose' dopamine worsens renal perfusion in patients with acute renal failure.” Kidney Int. 2006 May;69(9):1669-74.
Epinephrine

- Alpha/beta agonist
- Usually used for shock resistant to other pressors (or in CT surgery at VA)
- Causes vasoconstriction, inotropy, chronotropy
- Dose: Initial: 1 mcg/minute; titrate to desired response; usual range: 2-10 mcg/minute
- Give centrally
Inotropes

- **Milrinone**
  - A selective phosphodiesterase inhibitor in cardiac and vascular tissue, resulting in vasodilation and inotropic effects with little chronotropic activity
  - Dose: 0.375-0.75 mcg/kg/minute.
  - >10%: Cardiovascular: Ventricular arrhythmia (ectopy 9%, NSVT 3%, sustained ventricular tachycardia 1%, ventricular fibrillation <1%)
  - Hypotension

- **Dobutamine**
  - Stimulates beta1-adrenergic receptors, causing increased contractility and heart rate, with little effect on beta2- or alpha-receptors
  - Dose: 2.5-20 mcg/kg/minute; maximum: 40 mcg/kg/minute,
  - Less arythmogenic than milrinone, but still a concern
  - Hypotension
Appropriate monitoring

- Central line: monitor CVP and for access
- Arterial line: titrate gtts
- Foley: monitor end organ perfusion by following UOP
- Swan-ganz catheter: optional
How it works (generally)

- Measures pressures from the right atrium, right ventricle, and pulmonary artery.
- The left atrial pressure can be indirectly measured by inflating a balloon at the tip of the catheter and allowing the balloon to occlude a branch of the pulmonary artery (PCWP).
- The systemic vascular resistance and pulmonary vascular resistance can be estimated by calculations derived from Ohm's Law.
- Older pulmonary artery catheters measured cardiac output via the indicator thermodilution method or the Fick calculation.
- Newer catheter designs incorporate continuous oximetric monitoring of pulmonary artery oxygen saturation using fiberoptic reflectance spectrophotometry, thereby enabling continuous estimation of cardiac output.
Normal Hemodynamic Parameters

CVP: 1-11 mmHg
PCWP: 6-15 mmHg
Cardiac output: 4–8 L/min
Cardiac index: 2.6–4.2 (L/min)/m²
Stroke volume: 50–100 mL/beat
Systemic vascular resistance: 700–1600 dynes · s/cm²
Pulmonary vascular resistance: 20–130 dynes · s/cm²
SVO2: 60-80% (about 75%)