Swans and Pressors

Vanderbilt Surgery Summer School

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SHOCK≠Hypotension

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SHOCK=Reduction of systemic tissue perfusion, resulting in decreased oxygen delivery to the tissues.

SHOCK≈Hypotension

Reasons for Hypotension

- Not enough preload
- Not enough afterload
- Not enough contractility
- Cardiac obstruction

Reasons for Hypotension

- 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%

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SvO2=O2 delivery - O2 consumption

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SvO2=O2 content x CO – consumption

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SvO2=O2 delivery - O2 consumption

SvO2=O2 content x CO – consumption

SvO2=SaO2 x Hct x CO - 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

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Treatment for Hypotension

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

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Squeeze Vessels

No heart squeeze	Some heart squeeze
Phenylepherine=Neosynepherine	Norepinepherine=Levophed
Vasopressin=Pitissin	Dopamine

Squeeze Vessels

No heart squeeze	Some heart squeeze
Phenylepherine=Neosynepherine Neurogenic Shock/Epidural	Norepinepherine=Levophed 1st agent for Sepsis
Vasopressin=Pitissin 2nd agent for Sepsis	Dopamine 1st agent for Sepsis

Squeeze Heart

Unsqueeze Vessels	No Vessel Squeeze
Milrinone	Dobutamine

Epinepherine

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
- Epinepherine if all else fails

In Depth Info

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

Type of shock	CVP/ PCWP	CO	SVR	Venous O2 Sat
Hypovolemic (including hemorrhagic)	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Septic (hyperdynamic)	↑↓	↑	↓	↑
Septic (hypodynamic)	↑↓	↓	↑	↓
Neurogenic	↓	↓	↓	↓

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.

Mutlu GM; Role of vasopressin in the management of septic shock. Intensive Care Med 2004 Jul;30(7):1276-91. 2004 Apr 21

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 are 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.

http://www.pacep.org/

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/cm5

Pulmonary vascular resistance: 20–130 dynes · s/cm5

SVO2: 60-80% (about 75%)