DEFINING SHOCK

- Condition of insufficient perfusion of cells and vital organs causing tissue hypoxia
- Perfusion is inadequate to sustain life
- Results in cellular, metabolic and hemodynamic derangements
It’s all about delivery of oxygen to the tissues!!!
DELIVERY OF OXYGEN TO THE TISSUES

Oxygen
Hemoglobin
Cardiac Output
OXYGEN DELIVERY TO TISSUES

- Oxygen delivery measured as DO\textsubscript{2}: Volume of oxygen delivered to tissues each minute
- \( \text{DO}_2 = \text{cardiac output} \times \text{arterial oxygen content} \)
  - Arterial oxygen content = hemoglobin \( \times \) arterial oxygen saturation
- \( \text{DO}_2 \) formula = \( \text{CO} \times \text{Hgb} \times \text{SaO}_2 \times 13.4 \) (constant)
- Normal \( \text{DO}_2 = 900-1100 \text{ ml/min} \) (1000 ml/min)
OXYGEN CONSUMPTION / RESERVE

- Oxygen consumption is measured as VO₂
- Volume of oxygen consumed by the tissues each minute
- Normal VO₂: 200–300 ml / min (250 ml / min)

- Measured by mixed venous oxygen saturation (SVO₂)
- Normal 60-80% (75%)
- May also be measured by SCVO₂
- Normal 80-85%
- Trends the same as SVO₂
KEY POINTS

- Tissues were delivered 1000 ml / min (DO$_2$)
- Tissues uses 250 ml / min (VO$_2$)
- This leaves a 75% reserve in venous blood

- Oxygen delivery and oxygen consumption are independent until a critical point of oxygen delivery is reached
- Tissues will extract the amount of oxygen needed independent of delivery because delivery exceeds need
## Relationship of Delivery to Consumption

The table below demonstrates the relationship between delivery ($DO_2$), extraction ($VO_2$), and oxygen saturation ($SVO_2$) according to the equation:

$$VO_2 = DO_2 - SV_0_2$$

<table>
<thead>
<tr>
<th>$DO_2$</th>
<th>$VO_2$ (extraction is independent of delivery)</th>
<th>$SVO_2$ (SV0_2 will improve when you increase delivery)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1000 cc</td>
<td>250 cc (25%)</td>
<td>75%</td>
</tr>
<tr>
<td>750 cc</td>
<td>250 cc (33%)</td>
<td>67%</td>
</tr>
<tr>
<td>500 cc</td>
<td>250 cc (50%)</td>
<td>50%</td>
</tr>
</tbody>
</table>
When oxygen delivery reaches a critical level then consumption will depend on delivery

SVO2 will *not* increase with increased delivery while you are in this dependent state

Anaerobic metabolism occurs here because you have an oxygen deficit
Global indicator between oxygen supply and demand

Influenced by oxygen delivery and oxygen extraction

Reflects mixing of venous blood from superior vena cava, inferior vena cava and coronary sinus

Measured using a pulmonary artery fiberoptic catheter
**Significant Changes in SVO$_2$**

- **SVO$_2$ < 60%**
  - Decreased delivery
  - Increased consumption

- **SVO$_2$ > 80%**
  - Increased delivery
  - Decreased demand
  - Sepsis (tissues cannot extract)
  - Wedged catheter

- Clinically significant change is +or – 5 to 10% over 3 to 5 minutes

- **SVO$_2$ < 40%** represents limits of compensation and lactic acidosis will occur (oxygen demand is greater than oxygen delivery and reserve can be depleted = oxygen debt)
ScVO2

- ScVO2 reflects oxygen saturation of blood returning to right atrium via the superior vena cava.
  - Can be obtained without a pulmonary artery catheter, using a modified central venous catheter with fiberoptic technology.
  - Normal value is > 70%.
  - ScVO2 trends higher than SVO2 but trends with SVO2.
LACTIC ACIDOSIS

- Indicator of perfusion / poor perfusion
- Normal Lactate Level
  - 0.5-1 mmol/L
- Concentrations up to 2 mmol/L may be normal in critical illness
- Levels 2-4 mmol/L
  - Mild to moderate
  - Do not always have metabolic acidosis YET
- >5 mmol/L
  - Metabolic acidosis (pH < 7.35)
  - 75% mortality
CARDIAC OUTPUT
The Heart as a Pump

**Goal:** Forward propulsion of blood to perfuse the body.

**FLOW IS**

**DETERMINED BY:**

- SQUARE ROOT **PRESSURE**
- SQUARE ROOT **RESISTANCE**
- **VOLUME**
HEART

ARTERIES

VEINS

CONTAINER

CONTENT

VOLUME
CARDIAC OUTPUT

Heart rate

Stroke volume

Preload

Contractility

Afterload

Diastolic filling

Fiber stretch

Contractile force

Arterial pressure
DEFINITIONS

- **Cardiac Output**: Volume of blood ejected by the ventricle each minute
  - Normal: 4-8 liters/minute

- **Cardiac Index**: Adjustment made for body size
  - Normal cardiac index: 2.5-4 liters/minute/m^2

- **Stroke Volume**: Volume of blood ejected with each beat.
  - Normal 60-120 ml / beat
  - Systolic BP as non invasive indicator

- **Ejection Fraction**: Percent of blood ejected from the ventricle
  - Normal: 55% to 60%
RIGHT SIDED VERSUS LEFT SIDED SYSTEM

Diagram showing the right-sided versus left-sided system of the heart, including the pulmonary artery, pulmonary vein, capillaries, Aorta, LA, LV, RA, and RV.
The ventricle is preloaded with blood at the end of diastole: Creates stretch on myocardial muscles fibers

- Determined by:
  - Volume of blood filling the ventricle at end of diastole
  - Greater the volume the greater the stretch (muscle fiber length)
  - Greater the stretch the greater the contraction
  - Greater the contraction the greater cardiac output

TO A POINT
End-diastolic stretch on myocardial muscles fibers

Determined by:

- Volume of blood filling the ventricle at end of diastole
- Greater the volume the greater the stretch (muscle fiber length)
- Greater the stretch the greater the contraction
- Greater the contraction the greater cardiac output

TO A POINT
RIGHT VENTRICULAR PRELOAD
- Measured by right atrial pressure (RAP) or central venous pressure (CVP)

LEFT VENTRICULAR PRELOAD
- Measured by the pulmonary artery occlusive pressure (PAOP) [previously called pulmonary artery catheter wedge pressure]

Changes in pressure do not always = the same changes in volume:
- Dilated versus noncompliant ventricle
NON INVASIVE ASSESSMENT OF PRELOAD

RIGHT VENTRICULAR
- JV pulsation
- Hepatojugular reflux
- Peripheral edema *
- Weight *

LEFT VENTRICULAR
- Orthopnea / PND/ Dyspnea
- Rales/crackles
  + Consider role of lymph drainage
- S3 Gallop
- Frothy sputum
- Hypoxemia from decreased diffusion of oxygen
- Urine output
- Weight *
MEASURING JVP PULSATION / PRESSURE

- Raise HOB 30 – 45 degrees
- Internal preferred
- May use external
- Use tangential light
- Use centimeter ruler
- Difficult to assess if HR>100

- Normal JVP level is < 3 cm above the sternal angle
- Sternal angle is 5cm above right atrium
- JVP of 3 cm + 5cm = estimated CVP of 8cm H₂O
Estimated CVP > 8 cmH₂O

- Increased blood volume
- Usually RV failure
- Tricuspid valve regurgitation
- Pulmonary hypertension
JVD (JUGULAR VENOUS DISTENSION)

Additional assessment tip:
Sitting or standing patient up to see top of column.
FACTORS INFLUENCING PRELOAD

- Body Position
- Venous Tone
- Intrathoracic pressure
- Intrapericardial pressure
- Dysrhythmias
- Atrial Kick
- LV Function

- Circulating blood volume
  - Hypervolemia
  - Hypovolemia
  - Third spacing

- Distribution of blood volume
  - Sepsis
  - Anaphylaxis
  - Venous vasodilators
After the ventricle is loaded:
Pressure ventricle needs to overcome to eject blood volume

- Blood pressure is major component of LV afterload but it does not equal afterload
- Smaller vessel resistance is major component of LV afterload
- Other components
  - Valve compliance
  - Viscosity of blood
  - Arterial wall compliance
  - Aortic compliance
**AFTERLOAD ASSESSMENT**

- **Left ventricle:**
  - Systemic vascular resistance (SVR)
  - Noninvasive assessment: Diastolic blood pressure and pulse pressure

- **Right ventricle:**
  - Pulmonary vascular resistance (PVR)
Afterload is related to blood pressure but not synonymous
BP = CO \times SVR

- Low BP could be due to:
  - Low CO
    - HR too slow or too fast
    - Preload too low or too high
    - Contractility low
  - Low SVR
    - Vasodilation due to sepsis, anaphylaxis, altered neurological function, drugs
MORE ON VASCULAR TONE

- Increased vascular tone is usually associated with compensation for low SV
  - Acute Cardiogenic shock
  - Hypovolemic shock

- Decreased vascular tone is usually due to abnormally pathology
  - Sepsis
  - Anaphylaxis
  - Altered neurological control
WHEN YOU HAVE TACHYCARDIA ASK YOURSELF:

Why is my patient compensating?

Look to the pulse pressure for your answer.
PP < 35 with tachycardia (in absence of beta blocker)

Early sign of inadequate blood volume
* Can also be seen in cardiogenic shock

PP > 35 with tachycardia

Early sign of vasodilatory state such as sepsis
BLOOD PRESSURE: CO X SVR

- BP: 88/64
  - Is problem low cardiac output or low SVR?
  - How to treat?

- BP: 82/30
  - Is problem low cardiac output or low SVR?
  - How to treat?
STROKE VOLUME VARIATION

- Remember – think LV ejection
  + Volume
  + Contractility

- Give volume 1st
- Then evaluate
- If needed – add inotrope
ETIOLOGY OF HYPOTENSION

Cardiac Output \times SVR = Blood Pressure
**COMPARISON OF 2 HYPOTENSIVE PATIENTS**

**88/70**
- **Cardiogenic Shock**
  - Cause: Decreased C.O.
  - Treatment may involve afterload reduction to increase cardiac output

**82/30**
- **Septic Shock**
  - Cause: Decreased SVR
  - Treatment is focused on filling tank and restoring vascular tone.

Vasodilators? Fix HR Inotropes

Fluid and Vasopressors
Causes of Increased LV Afterload

- Arterial vasoconstrictors
- Hypertension
- Aortic valve stenosis
- Increased blood viscosity
- Hypothermia
- Compensatory vasoconstriction from hypotension in shock

Causes of Decreased LV Afterload

- Arterial vasodilators
- Hyperthermia
- Vasogenic shock states (sepsis and anaphylactic) where the body cannot compensate with vasoconstriction
- Aortic Regurgitation – hyperdynamic cardiac output therefore lowering systemic vascular resistance
INCREASED RIGHT SIDED AFTERLOAD

- Pulmonary hypertension
  - mPAP > 25 mmHg or > 30 mmHg with exercise
  - PVR > 250 dynes/sec/cm$^{-5}$
- Causes
  - Hypoxemia
  - Acidosis
  - Inflammation
  - Hypothermia
  - Excess sympathetic stimulation
  - Pulmonary endothelial dysfunction
    - Impaired nitric oxide and prostacyclin (PGI$_2$) release
  - Primary pulmonary hypertension
CONTRACTILITY

- **Ability of myocardium to contract independent of preload or afterload**
  - Velocity and extent of myocardial fiber shortening
  - Inotropic state
- Related to degree of myocardial fiber stretch (preload) and wall tension (afterload).
- Influences myocardial oxygen consumption
- ↑ contractility
  - ⇒ ↑ myocardial workload
  - ⇒ ↑ myocardial oxygen consumption
IMPORTANT POINTS ABOUT CONTRACTILITY

- No accurate way to measure contractility
  - RVSWI / LVSWI not reliable measures

**Noninvasive Assessment: Ejection Fraction**

- Low cardiac output does not necessarily mean diminished contractility (i.e. hypovolemia)

- Correct preload and afterload problems first in a patient with a low ejection fraction.

- Increasing contractility with medications will also increase myocardial oxygen demand.
FACTORS ALTERING CONTRACTILITY

- Decreased contractility
  + Excessive preload or afterload
  + Drugs – negative inotropes
  + Myocardial damage
  + Ischemia
  + Cardiomyopathy
  + Hypothyroidism
  + Changes in ionic environment: hypoxia, acidosis or electrolyte imbalance

- Increased contractility
  + Drugs
    - Positive inotropes
  + Hyperthyroidism
  + Adrenal Medulla Tumor
Low cardiac output does not necessarily mean diminished contractility

Correct preload and afterload problems first

Medications which increase contractility impact not only cardiac output but also myocardial oxygen demand.
HEART RATE

- Mathematically heart rate increases cardiac output
- Physiological limit where increased heart rate will decrease cardiac output due to decreased filling time (decreased preload)
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal</th>
<th>Component of CO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Output / Index</td>
<td>CO: 4-8 L/min CI: 2.5-4.0 L/min/m²</td>
<td></td>
</tr>
<tr>
<td>CVP/RAP (mean)</td>
<td>2-6 mm Hg</td>
<td>RV Preload</td>
</tr>
<tr>
<td>PAOP/PWP (mean)</td>
<td>5-12 mm Hg</td>
<td>LV Preload</td>
</tr>
<tr>
<td>PA Systolic</td>
<td>15-30 mm Hg</td>
<td>RV Function (Reflects lungs)</td>
</tr>
<tr>
<td>PA Diastolic</td>
<td>6-15 mm Hg</td>
<td>LV Preload (if no Pulm HPTN or MV Disease)</td>
</tr>
<tr>
<td>Pulmonary Vascular Resistance (PVR)</td>
<td>&lt; 200 dynes/sec/cm⁻⁵</td>
<td>RV Afterload</td>
</tr>
<tr>
<td>Systemic Vascular Resistance (SVR)</td>
<td>800-1200 dynes/sec/cm⁻⁵</td>
<td>LV Afterload</td>
</tr>
<tr>
<td>Stroke Volume / SVI</td>
<td>SV: 60-100 ml/beat SVI: 35-60 ml/beat/ m²</td>
<td>SUM of Preload, afterload, contractility</td>
</tr>
<tr>
<td>RVSWI</td>
<td>5 - 10 g/m²/beat</td>
<td>RV Contractility</td>
</tr>
<tr>
<td>LVSWI</td>
<td>50 - 62 g/m²/beat</td>
<td>LV Contractility</td>
</tr>
</tbody>
</table>
LEFT VENTRICULAR FUNCTION CURVES

Forrester Subsets
### SIGNS AND SYMPTOMS OF SHOCK STAGES

<table>
<thead>
<tr>
<th>Stages</th>
<th>( CI )</th>
<th>Signs and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sub clinical Hypoperfusion</td>
<td>2.2-2.5</td>
<td>- No clinical indications of hypoperfusion yet something seems different or not right</td>
</tr>
<tr>
<td>Compensatory with SNS Stimulation</td>
<td>2.0 – 2.2</td>
<td>- Tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Narrowed pulse pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Tachypnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Cool Skin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Oliguria</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Decreased Bowel sounds</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Restlessness / confusion</td>
</tr>
</tbody>
</table>
**SIGNS AND SYMPTOMS OF SHOCK STAGES**

- **Shock: Progressive with hypoperfusion (CI < 2.0)**
  - Dysrhythmias
  - Hypotension
  - Tachypnea
  - Cold, clammy skin
  - Anuria
  - Absent bowel sounds
  - Lethargy to coma

- **Shock: Refractory with profound hypoperfusion (CI < 1.8)**
  - Life threatening dysrhythmias
  - Hypotension despite vasopressors
  - ARDS
  - DIC
  - Hepatic failure
  - ATN
  - Mesenteric ischemia
  - Myocardial ischemia
  - Cerebral ischemia
    - Or infarction for above
**Fluid Overload vs. Hypoperfusion**

**Hypoperfusion**
- Narrow pulse pressure
- Resting tachycardia
- Cool Skin
- Altered mentation
- Decreased urine output
- Increased BUN/Creatinine
- Cheyne Stokes Respirations

**Fluid Overload**
- Weight gain
- Peripheral edema
- Jugular venous distention
- SOB
- Crackles
# Relationship of PAOP (PWP) to Clinical Signs of Pulmonary Congestion

<table>
<thead>
<tr>
<th>PAOP</th>
<th>Clinical State</th>
</tr>
</thead>
<tbody>
<tr>
<td>18 – 20 mmHg</td>
<td>Pulmonary Congestion</td>
</tr>
<tr>
<td>20 – 25 mmHg</td>
<td>Moderate Congestion</td>
</tr>
<tr>
<td>25 – 30 mmHg</td>
<td>Severe Congestion</td>
</tr>
<tr>
<td>&gt; 30 mmHg</td>
<td>Pulmonary Edema</td>
</tr>
</tbody>
</table>

The value for PAOP that best separates patients with and without pulmonary congestion is 18 mmHg.
<table>
<thead>
<tr>
<th>CI</th>
<th>Clinical State</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.7 – 4.7</td>
<td>Normal</td>
</tr>
<tr>
<td>2.2 – 2.7</td>
<td>Subclinical depression</td>
</tr>
<tr>
<td>1.8 – 2.2</td>
<td>Clinical hypoperfusion</td>
</tr>
<tr>
<td>&lt; 1.8</td>
<td>Cardiogenic shock</td>
</tr>
</tbody>
</table>

The value for CI that best separates patients with and without hypoperfusion is 2.2 L/min/M²
Backwards Failure: Pulmonary Congestion

Forwards Failure: Hypoperfusion
HEMODYNAMIC AND CLINICAL SUBSETS

Forwards Flow: CI, Skin temp (warm or cold)

Normal Hemodynamics (I)
No pulmonary congestion:
- PWP < 18; Dry lungs
No hypoperfusion:
- CI > 2.2; Warm skin

Backwards Failure (II)
Pulmonary congestion
- PWP > 18; Wet lungs
No hypoperfusion
- CI > 2.2; Warm skin

Forwards Failure (III)
No pulmonary congestion
- PWP < 18; Dry lungs
Hypoperfusion
- CI < 2.2; Cold skin

The Shock Box (IV)
Pulmonary congestion
- PWP > 18; Wet lungs
Hypoperfusion
- CI < 2.2; Cold skin

Preload: PWP, lung sounds (dry or wet)
Preload: PWP, lung sounds (dry or wet)

Left Ventricular Function Curves

Forwards Flow:

Cl, Skin temp (warm or cold)
<table>
<thead>
<tr>
<th>Warm and Dry</th>
<th>Warm and Wet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Perfusion</td>
<td>Normal Perfusion</td>
</tr>
<tr>
<td>No Congestion</td>
<td>Congestion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cold and Dry</th>
<th>Cold and Wet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Perfusion</td>
<td>Low Perfusion</td>
</tr>
<tr>
<td>No Congestion</td>
<td>Congestion</td>
</tr>
</tbody>
</table>

**Preload:** PWP, lung sounds (dry or wet)
Left Ventricular Function Curves

Forwards Flow: CI, Skin temp (warm or cold)

Preload: PWP, lung sounds (dry or wet)
Changing Preload: moves patient along the curve they are on.

Forwards Flow: CI, Skin temp (warm or cold)

Preload: PWP, lung sounds (dry or wet)
Changing Contractility: moves patient to a higher curve

Forwards Flow: CI, Skin temp (warm or cold)

Preload: PWP, lung sounds (dry or wet)
Changing Afterload: moves patient up and to the left (improves forwards flow and reduces preload)

Forwards Flow: CI, Skin temp (warm or cold)

Preload: PWP, lung sounds (dry or wet)
WHEN TO ALTER PRELOAD

• Hypotension secondary to hypovolemia
  – Goal: Increase preload
  – Therapy: Fluids

• Pulmonary congestion
  – Goal: Decrease preload
  – Therapy: Diuretics, venous dilators
Preload changes: move patient along the current curve
WHEN TO ALTER AFTERLOAD

- **Combined forward and backward failure** (high PWP and low CI)
  - Goal: decrease pulmonary congestion and increase forward flow

- **Mitral regurgitation or VSD**
  - Goal: decrease retrograde blood flow and increase forward flow

- **Therapy**: Arterial dilator drugs
  - ACEI, ARBs, Nitroprusside, Ca\(^{++}\) blockers, Milrinone, Nesiritide
  - OR: IABP
Afterload Changes: move patient up and to the left: improves forwards flow and reduces preload

Forwards Flow: CI, Skin temp (warm or cold)

Must have adequate BP
SVR must be elevated
WHEN TO ALTER CONTRACTILITY

- Subset III patients with adequate preload
- Subset IV patients (high PAOP and low CI)
  - Assume a contractility problem
- Patients with low CO but optimal preload, afterload, and HR
  \[ CO = HR, \text{preload}, \text{afterload}, \text{contractility} \]
- Therapy: inotropes (dobutamine, dopamine, milrinone, epinephrine, digoxin)
  - Increase MVO\(_2\), use with caution in acute MI
Contractility Changes: move patient to a higher curve

Preload: PWP, lung sounds (dry or wet)
DEFINING SHOCK

- Condition of insufficient perfusion of cells and vital organs causing tissue hypoxia
- Perfusion is inadequate to sustain life
- Results in cellular, metabolic and hemodynamic derangements
When signs of hypoperfusion are evident THINK:

- The Heart
- The Blood
- The Vessels
The Heart

Elevated HR
Elevated Preload
Elevated Afterload
Decreased Contractility

- Cardiogenic shock
- Inadequate pump
• Hypovolemic shock
• Not enough volume
• Loss of blood or fluid
• Internally or externally

The Blood

Elevated HR
Decreased Preload
Increased Afterload
Decreased Contractility
The Vessels

Increased HR
Decreased Preload
Decreased Afterload
Increased Contractility (early to compensate)

• Vasogenic Shock
• Inappropriate vasodilatation (Distributive Shocks)
  • Septic
  • Anaphylactic
• Neurogenic
## SHOCK STATE COMPARISONS

<table>
<thead>
<tr>
<th></th>
<th>Hypovolemic</th>
<th>Cardiogenic</th>
<th>Early Septic (Distributive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO / CI</td>
<td>▼</td>
<td>▼</td>
<td>▲ (to compensate)</td>
</tr>
<tr>
<td>Preload</td>
<td>▼ (primary problem)</td>
<td>▲</td>
<td>▼ (primary problem)</td>
</tr>
<tr>
<td>Afterload</td>
<td>▲ (to compensate)</td>
<td>▲ (to compensate)</td>
<td>▼ (primary problem)</td>
</tr>
<tr>
<td>Contractility</td>
<td>Neutral</td>
<td>▼ (primary problem)</td>
<td>▲ (to compensate)</td>
</tr>
<tr>
<td>HR</td>
<td>▲ (to compensate)</td>
<td>▲ (to compensate)</td>
<td>▲ (to compensate)</td>
</tr>
<tr>
<td>SVO2</td>
<td>▼</td>
<td>▼</td>
<td>▼</td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Cardiogenic</td>
<td>Septic (Distributive)</td>
<td></td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>-------------------------------------------</td>
<td>----------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>√ Replace volume</td>
<td>√ Support stroke volume</td>
<td>√ Fill up container</td>
<td></td>
</tr>
<tr>
<td>√ Stop volume loss</td>
<td>√ Decrease preload</td>
<td>√ Use vasopressors to restore vascular tone</td>
<td></td>
</tr>
<tr>
<td></td>
<td>√ Decrease afterload</td>
<td>√ Treat underlying cause</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(think IABP)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>√ May need to support contractility</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
OUTCOME INDICATORS

- SVV < 15%
- ScVO2 > 70%
- HR < 90 BPM
- Lactic acid < 2.0 mMOL

Others:
  - Mentation
  - Urine Output
58 year old male with no prior cardiac events presents to the emergency department with severe midsternal chest pain. The pain started about 60 minutes ago and has been fairly severe and unrelenting. The patient is experiencing nausea and he is short of breath.

- He reports a history of hypertension and hyperlipidemia but admits he does not regularly follow with his physician nor take his medications. His father died of an AMI at age 60 and his bother had an AMI at age 55.

- He is a 1 ½ PPD smoker
PHYSICAL ASSESSMENT / DIAGNOSTICS

- BP: 92/70
- HR: 110 with frequent PVCs
- Rhythm: ST with ST Segment elevation in leads V1-V6, I and aVL
- RR 28-32
- Anxious
- Pale and cool to touch
- Peripheral pulses are weak
- Capillary refill is slow
- JVD +5cm
- Lungs: Fine crackles over the bases
- Heart Sounds: Irregular, normal S1, S2 with no murmur, rub, or gallop.
- Hypoactive bowel sounds

- SaO2 91% on 4L nasal cannula
- PaCO2 43 mmHg
- PaO2 62 mmHg
- Troponin 3.2
- HGB 13.8
EVALUATION

- Signs of hypoperfusion?
  - Delivery of oxygen?
    - Cardiac output
    - Hemoglobin
    - O2 Saturation
  - A closer look at cardiac output?
    + Preload?
    + Afterload?
    + Contractility?
    + HR?
    + Hemodynamic profile?

- BP: 92/70
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- PaCO2 43 mmHg
- PaO2 62 mmHg
- Troponin 3.2 / HGB 13.8
When signs of hypoperfusion are evident THINK:

- The Heart
- The Blood
- The Vessels
Hemodynamic Profile

- Increased HR
- Increased Preload (RA, PAOP)
- Increased Afterload (SVR)
- Decreased Contractility
- Decreased Stroke Volume
- Decreased CO / CI
- Decreased $SVO_2$
Assessment of Cardiogenic Shock

<table>
<thead>
<tr>
<th>Perfusion at Rest</th>
<th>Congestion at Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>Warm and Dry</td>
</tr>
<tr>
<td></td>
<td>No congestion</td>
</tr>
<tr>
<td></td>
<td>No hypoperfusion</td>
</tr>
<tr>
<td>No</td>
<td>Warm and Wet</td>
</tr>
<tr>
<td></td>
<td>Congestion</td>
</tr>
<tr>
<td></td>
<td>No hypoperfusion</td>
</tr>
<tr>
<td>Yes</td>
<td>Cold and Dry</td>
</tr>
<tr>
<td></td>
<td>No congestion</td>
</tr>
<tr>
<td></td>
<td>Hypoperfusion</td>
</tr>
<tr>
<td>Cl 2.2</td>
<td></td>
</tr>
</tbody>
</table>

PWP 18
Treatment Goal: Maximize the delivery of oxygen to the tissues

Remember: CI, Hemoglobin, SaO2

Focus on: Cardiac Output/Cardiac Index

Optimize preload and afterload first

Preload reduction when PAOP > 20-22 mm Hg or when pulmonary edema is present
- Diuretics
- Venous vasodilators

Afterload reduction – Mean arterial pressure 70mm Hg or greater
- Careful consideration in hypotension
- Arterial vasodilators
- IABP
- Vasopressors use can adversely impact myocardial function

Improve contractility to maximize stroke volume if needed

- Inotropes
  - Dobutamine
  - Milrinone

Revascularization if ischemia or infarction
Low Output Cardiogenic Shock

- Systolic BP > 100 mm Hg
  - NTG 10-20 mcg/min IV

- Systolic BP 70-100 mm Hg No S / S of Shock
  - Dobutamine 2-20 mcg/kg/min

- Systolic BP 70-100 mm Hg S/S of Shock
  - Dopamine 5-15 mcg/kg/min

- Systolic BP < 70 mm Hg S/S of shock
  - Norepinephrine 0.5-30 mcg/min
Backer et al. 
Multi Center Randomized Controlled Trial 
New England Journal of Medicine 
March 4\textsuperscript{th} 2010

There were no significant differences between the groups in the rate of death at 28 days or in the rates of death in the ICU, in the hospital, at 6 months, or at 12 months

More patients with arrhythmia in the dopamine group

Rate of death was higher in predefined subgroup analysis for patients with cardiogenic shock treated with dopamine.
CASE STUDY 2

A 62 year old male presents after developing new onset confusion following a one week course of flu and respiratory symptoms. On admission to ED the patients blood pressure is 76/30 mm Hg with a pulse of 128 BPM and a respiratory rate of 32. Initial temperature is 38.9 C, WBCs 14.9, and CXR shows left lower lobe infiltrate.
EVALUATION

- Signs of hypoperfusion?
- Delivery of oxygen?
  - Cardiac Output
  - Hemoglobin
  - Oxygen Saturation
- Cardiac output?
  + Preload?
  + Afterload?
  + Contractility?
  + HR?
  + Hemodynamic profile?

- BP: 76/30 mm Hg
- HR: 128
- RR: 32
- Temp: 38.6
- Skin cool
- New onset confusion
- Lungs: Diminished breath sounds left base
- No JVD
- Heart: Regular Normal  S1 S2
- SaO2: 88% on 4L / NC
- PaCO2 43 mmHg
- PaO2: 59 mmHg
- WBC 14.9
- H & H 12.8 / 38.2
- BUN 22 / Creatinine 0.98
When signs of hypoperfusion are evident THINK:

- The Heart
- The Blood
- The Vessels
Septic Shock

Hemodynamic Profile

Early Septic Shock

- Increased HR
- Decreased Preload
- Decreased Afterload
- Contractility - depressed due to tumor necrosing factor alpha
- Increased CO / CI
  - CO x SVR = BP therefore CO rises in an attempt to compensate for the reduced SVR
- Decreased SV02
SEPTIC SHOCK (DISTRIBUTIVE – EARLY)

- Sepsis with hypotension despite fluid resuscitation, and perfusion abnormalities
- Three key processes involved in sepsis
  + Inflammation
  + Coagulation
  + Impaired fibrinolysis

Presentation
- Tachypnea
- Hypoxia
- Tachycardia
- Profound hypotension
SEPTIC SHOCK TREATMENT

- **Treatment Goal:** Maximize the delivery of oxygen to the tissue
  - *Remember:* CI, Hemoglobin, SaO2
    - Prevention – hand hygiene
    - Treat the cause – early antibiotics
- **Focus on Cardiac Output**
  - Volume replacement
    - Crystalloids 4-8 liters
    - 20ml/kg
  - Vasopressors
    - To maintain MAP > 65 mm Hg
    - Norepinephrine is vasopressor of choice
    - Dopamine (not low dose)
    - Vasopressin for refractory shock
  - May need inotropes
- **Hemoglobin**
  - Transfuse for HCT < 30%
- **Oxygen**
  - Sedate and ventilate

---

**Early Goal Directed Therapy**
- CVP 8-12 mm Hg (12-15 mm Hg)
- PAOP 12-18 mm Hg
- MAP > 65 mm Hg
- U.O. > 0.5 cc/kg/hr
- SaO2 > 90%
- Central venous oxygen saturation > 70%
ETIOLOGY OF HYPOTENSION

Cardiac Output \times SVR = Blood Pressure
COMPARISON OF 2 HYPOTENSIVE PATIENTS

Cardiogenic Shock

- Cause: Decreased C.O.
- Treatment may involve afterload reduction to increase cardiac output

Vasodilators

88/70

Septic Shock

- Cause: Decreased SVR
- Treatment is focused on filling tank and restoring vascular tone.

Vasopressors

82/30
CASE STUDY #3

- 81 year old female post PTCA DES to RCA
- Staged procedure: LAD STENT placed two weeks ago when she had an anterior STEMI
- EF at that time 35%
- Arterial sheath pulled without complication 3 hours ago. Recovery has been uneventful.
- Patient puts on the call light. When nurse comes to the room the patient tells the nurse she is not feeling well, she feels faint and her lower back is really hurting.
PHYSICAL ASSESSMENT

- BP 86/68
- ST 108
- RR 24-28 Pulse Ox 90% on 2L NC
- Pale, cool
- Lungs Clear
- Apical regular, no murmurs, rubs or gallops
- Hypoactive BSP
- Pulses faintly palpable
- Right groin site without bleeding, hematoma, bruit or thrill
**EVALUATION**

- Signs of hypoperfusion?
- Delivery of oxygen?
  - Cardiac Output
  - Hemoglobin
  - Oxygen Saturation
- Cardiac output?
  + Preload?
  + Afterload?
  + Contractility?
  + HR?
- Hemodynamic profile?

- BP 86/68
- ST 108
- RR 24-28  SaO2 90%
- Pale, cool
- Lungs Clear
- Apical regular, no murmurs, rubs or gallops
- Hypoactive BSP
- Pulses faintly palpable
- Right groin site without bleeding, hematoma, bruit or thrill
When signs of hypoperfusion are evident THINK:

The Heart

The Blood

The Vessels
HYPOVOLEMIC SHOCK

- Decreased intravascular volume
  + Usually acute blood loss
- Decreased venous return
- Decreased preload and stroke volume
- Decreased cardiac output
- Decreased mean arterial pressure
- Inadequate tissue perfusion
- Anoxia -> anaerobic metabolism -> lactic acid accumulation -> metabolic acidosis
- Organ failure
<table>
<thead>
<tr>
<th>Estimated Loss</th>
<th>Clinical Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 500cc</td>
<td>No signs or symptoms.</td>
</tr>
</tbody>
</table>
| 500-1,000 cc  | Heart rate up to 20% above baseline.  
|               | Systolic blood pressure 10% below baseline.  
|               | Decreased urine output.  
|               | Weakening pulse.  
|               | Skin cool to touch.  
|               | Cardiac output within normal limits with an elevated systemic resistance.  
|               | Mild acidosis. |
| 1,000-2,000 cc| Heart rate 20 to 30% above baseline.  
|               | Systolic blood pressure 10 to 20% below baseline.  
|               | Respiratory rate 10% above baseline.  
|               | Oxygen saturation may continue to remain normal if receiving supplemental oxygen.  
|               | SvO₂ < 60%.  
|               | Urine output less than 30 cc/hour.  
|               | Changes in level of consciousness.  
|               | Skin cool and diaphoretic.  
|               | Peripheral pulses weak and thready.  
|               | Cardiac output decreased with elevated SVR.  
|               | Progressive acidosis. |
| 2,000-3,000 cc| Heart rate 20 to 30% above baseline.  
|               | Systolic blood pressure 10 to 20% below baseline.  
|               | Respiratory rate 10 to 20% above baseline.  
|               | Oxygen saturation decreased.  
|               | SvO₂ < 55 to 60%.  
|               | Oliguria or anuria.  
|               | Mental stupor.  
|               | Extremities cold.  
|               | Peripheral pulses poor.  
|               | Peripheral cyanosis.  
|               | Cardiac output decreased with elevated SVR.  
|               | Severe acidosis. |

(Adapted from Park, 2004)
HYPOVOLEMIC SHOCK

Presentation
- Tachycardia
- Pulse pressure narrows
- Increased RR and depth
- Hypoxemia
- Decreased urine output
- Pale, cool
- Changes in LOC

Hemodynamic Profile
- Increased HR
- Decreased Preload
- Increased Afterload
- Decreased Contractility
- Decreased CO / CI
- Decreased $S_vO_2$
**HYPOVOLEMIC SHOCK TREATMENT**

- **Treatment Goal:** Maximize the delivery of oxygen to the tissue
  - *Remember:* CI, Hemoglobin, SaO2

- Focus on Cardiac Output
  - Volume replacement
    - Isotonic crystalloids first (0.9NS or Lactated Ringers)
    - Require replacement of 3 cc per 1 cc fluid loss
    - Fluid challenge of 250 – 500 cc over 5 minutes
    - Then: 200cc every 5 minutes until an increase in BP or signs of fluid overload
    - Colloids if crystalloids not effective

- Focus on Hemoglobin
  - Replace blood loss if necessary for tissue oxygenation
    - Need hemoglobin to carry blood
  - Treat cause of loss
CASE STUDY #4

- 82 year old female presented to the ED with chest pain and shortness of breath
- No previous cardiac history
- History of treated hypothyroidism, hypertension, and type II DM on oral medication
- ECG demonstrates ST 112 with no ST elevation, depression or T Wave inversion.
**Physical Assessment**

- BP 86/70
- ST 110 Regular
- RR 28-32 Pulse Ox
  - 90% on 4L NC
- Very Pale, cool
- Lungs Clear
- No murmurs, rubs or gallops
- Hypoactive BSP
- Pulses thready

**Diagnostic Data**

- SaO2: 90% on 4L / NC
- pH 7.31
- PaCO2 37 mmHg
- PaO2: 60 mmHg
- Troponin 2.6
- H & H 6.8 / 20.2
- BUN/Creatinine WNL
EVALUATION

- Signs of hypoperfusion?
- Delivery of oxygen?
  - Cardiac Output
  - Hemoglobin
  - Oxygen Saturation
- Cardiac output?
  + Preload?
  + Afterload?
  + Contractility?
  + HR?
- Hemodynamic profile?

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- pH 7.31
- PaCO2 37 mmHg
- PaO2: 60 mmHg
- Troponin 2.6
- H & H 6.8 / 20.2
- BUN/Creatinine WNL
When signs of hypoperfusion are evident THINK:

The Heart

The Blood

The Vessels
OBSTRUCTIVE SHOCK STATES
OBSTRUCTIVE SHOCK

- Obstruction of ventricular filling or obstruction of blood flow through the heart or great vessels resulting in decreased cardiac output
  - Pulmonary embolus
  - Cardiac tamponade
  - Pneumothorax
  - Dissecting aneurysm
IN SUMMARY: SHOCK STATES

- Condition of insufficient perfusion of cells and vital organs causing tissue hypoxia
- Think: Heart, Blood, or Vessels
- Treatment Goal: Maximize the delivery of oxygen to the tissues
  - Oxygen saturation
  - Hemoglobin
  - Cardiac Output / Index
It's all about delivery of oxygen to the tissues!!!
IT’S ALL ABOUT THE TISSUES BOTH RECEIVING AND UTILIZING OXYGEN!
In late sepsis tissues can decrease extraction of oxygen resulting in increase in SV02.

SV02 reflects venous saturation from the entire body but is not organ specific

SV02 does not reflect cellular utilization

Need better tools to evaluate tissue oxygenation

StO2
Thank You!

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