Hemodynamic Principles for Practice

Cynthia Webner  DNP, RN, CCNS, CCRN, CMC
Karen Marzlin  DNP, RN, CCNS, CCRN, CMC

Paradigm Shift

• Hemodynamics does not equal invasive monitoring

One must be comfortable with shades of grey!!
The Heart as a Pump

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

**Flow** is determined by:
- √ Pressure
- √ Resistance
- √ Volume
Right Sided versus Left Sided System

Basic Hemodynamic Formula

Cardiac Output

Heart Rate X Stroke Volume

Preload  Afterload  Contractility

Same four components also determine myocardial oxygen demand
Emerging Additional Factors

• Synergy

• Synchrony

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/m²

• **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%
More Understanding

KEY PHYSIOLOGICAL PRINCIPALS

Key Principles in Understanding Hemodynamic Assessment

- Arterial pulse waveform (and pulse pressure) are proportional to SV
- Pulse pressure inversely related to arterial compliance
- MAP = Calculated

- SBP: Reflects LV volume and contraction
  - Very dynamic
- Diastolic BP
  - Continuous / less dynamic pressure
  - Drives capillary opening pressure
Key Principles in Understanding Hemodynamic Assessment

- Vascular tone is affected by:
  - Large vessel compliance
  - Peripheral vascular resistance (smaller vessels)
- Vessel resistance changes more quickly than large vessel compliance
- Increased resistance = increased DBP

Key Principles in Understanding Hemodynamic Assessment

- Pressure does not always = Flow

  - “We measure BP because we can” – Barbara Mclean
Cardiac Output Assessment

TRADITIONAL TO LESS INVASIVE TECHNOLOGIES

Incident wave, reflective wave, Windkessel effect
Cardiac Output Measurements

- Thermodilution method using intermittent bolus injections of fluid
- Continuous measurement using a specialized catheter that emits heat
- Pulse contour analysis (PiCCO)
- Lithium derived continuous cardiac output (LiDCO)
- Transthoracic electrical bioimpedance

Thermodilution Cardiac Output

- Injection of known amount of D5W or NS into the RA through the proximal port of the PA catheter.
- The injectate fluid is warmed by the blood.
- The temperature of the blood (flowing past the thermistor at the distal end of the catheter) is measured.
- The CO computer calculates CO based on the temperature change between the injectate and the blood at the thermistor.
- The more blood (higher CO), the more the temperature change; the less blood (low CO) the less the temperature change.
Thermodilution Cardiac Output

- Injectate can be iced or at room temperature. 5 or 10 ml can be used. 5 ml injectate has more potential for error.
- A cardiac output constant is set in the computer to match the catheter type, injectate volume, and injectate temperature.
- Complete injection of the bolus should occur within 4 seconds. Injection should be smooth.
- At least 3 bolus measurements are obtained (to be averaged) with each assessment.
- The monitor displays the injection curve. Irregular or notched curves are deleted.
- **Tricuspid regurgitation or septal defects do not allow for accurate cardiac output assessment. Dysrhythmias can also interfere with an accurate measurement.**

Continuous Cardiac Output

- CO measurement utilizes a catheter that emits frequent bursts of heat to warm the blood in the RA, and calculates CO based on the temperature difference between RA and blood flowing past the thermistor.
  - Cardiac output values are averaged over 3 to 6 minutes and are updated every 30 to 60 seconds.
  - **Displayed values can be delayed by as much as 10 minutes with changes in CO (Bridges, 2005c).**
Pulse Contour Analysis (PiCCO)

- Requires central venous access and arterial access (not right heart access)
- Continuous cardiac output calculated by evaluating arterial pulse contour
  - Requires accurate assessment of dicrotic notch
  - Requires external calibration
- Additional hemodynamic parameters can be measured

Lithium Derived Continuous Cardiac Output (LiDCO)

- Similar to the PiCCCO system
- Utilizes an arterial line and either a central or peripheral venous line
- System is calibrated using a small dose of lithium chloride
- Utilizes the arterial blood pressure system to calculate continuous stroke volume, cardiac output and other hemodynamic measurements
Flo Trac System

- Vigileo Monitor (Flo Trac Sensor)
  - Assessment of CCO, SVV / SV, SVR
  - Used in patients with arterial line
  - SVV methodology
  - ScvO2 also be obtained when used with PreSep catheters.
- **Important consideration:**
  - Not validated in patients on IABP.
  - Cannot be used in patients who are hypothermic.

Transthoracic Electrical Bioimpedance

- Assesses the change in impedance as blood is ejected into the aorta.
- Electrodes on the neck and chest are used to assess electrical impedance.
- Blood ejected into the aorta results in a decrease in impedance proportional to the volume of ejected blood.
  - Magnitude of the decrease in impedance allows for a calculation of stroke volume (cardiac output) and SVR
- Several patient factors can interfere with the accuracy of this technique
- This technique does not provide an assessment of cardiac preload
- **Bioimpedance monitoring technology not yet recommended for use in routine clinical practice due to inconclusive trial results.**
Preload

The ventricle is preloaded for ejection.

- 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
### Assessing Preload with PA Catheter

<table>
<thead>
<tr>
<th>Right Ventricular</th>
<th>Left Ventricular</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Measured by right atrial pressure (RAP) or central venous pressure (CVP)</td>
<td>• Measured by the pulmonary artery occlusive pressure (PAOP) [previously called pulmonary artery catheter wedge pressure]</td>
</tr>
</tbody>
</table>

Changes in pressure do not always = the same changes in volume:

Dilated versus noncompliant ventricle
Non Invasive Assessment of Preload

Right Ventricular
- JVD
- 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
- Weight *
Measuring JVD

- 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 \( \leq 3 \text{ cm above the sternal angle} \)

- Sternal angle is 5cm above right atrium
- JVP of 3 cm + 5cm = estimated CVP of 8cm \( H_2O \)

Estimated CVP> 8 cm\( H_2O \)
- 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.

<table>
<thead>
<tr>
<th>Jugular Vein</th>
<th>Carotid Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pulsations palpable.</td>
<td>Palpable pulsations.</td>
</tr>
<tr>
<td>Pulsations obliterated by pressure above the clavicle.</td>
<td>Pulsations not obliterated by pressure above the clavicle.</td>
</tr>
<tr>
<td>Level of pulse wave decreased on inspiration; increased on expiration.</td>
<td>No effects of respiration on pulse.</td>
</tr>
<tr>
<td>Usually two pulsations per systole (x and y descents).</td>
<td>One pulsation per systole.</td>
</tr>
<tr>
<td>Prominent descents.</td>
<td>Descents not prominent.</td>
</tr>
<tr>
<td>Pulsations sometimes more prominent with abdominal pressure.</td>
<td>No effect of abdominal pressure on pulsations.</td>
</tr>
</tbody>
</table>
### Dynamic Preload Parameters

- **SPV**: Systolic Pressure Variation (mmHg):
  - $\text{SPMax} - \text{SPMin}$ **Normal** < 10 mmHg
    
    (6.25 sweep with arterial line waveform)

- **PPV**: Pulse Pressure Variation (%):
  - $\text{PPMax} - \text{PPmin}/ \text{PP mean}$ **Normal** < 13%

- **SVV**: Stroke Volume Variation (SVV%):
  - measured over the 20 second cycle.
  - $\text{SVMax} - \text{SVMin}/ \text{SV mean}$ **Normal** < 15 %

---

### Dynamic Preload Parameters

- **Passive Leg Raise Test**

- **Systolic Pressure Variation**
- **Stroke Volume Variation**
- **Pulse Pressure Variation**
  - All 3 measure the difference between the maximum and minimum values over a full respiratory cycle.
More on Dynamic Preload Parameters

- **SPV**: Systolic Pressure Variation (mmHg):
  - SPMax – SPMin **Normal < 10 mmHg**
  (6.25 sweep with arterial line waveform)

- **PPV**: Pulse Pressure Variation (%):
  - PPMax – PPmin/ PP mean **Normal <13%**

- **SVV**: Stroke Volume Variation (SVV%):
  measured over the 20 second cycle.
  - SVMax – SVMin/SV mean **Normal < 15 %**

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
Afterload

After the ventricle is loaded, it must work!

- Pressure ventricle needs to overcome to eject blood volume

- Left ventricle:
  - Systemic vascular resistance
  - Other components
    - Valve compliance
    - Viscosity of blood
    - Arterial wall compliance
      - Aortic compliance

- Right ventricle:
  - Pulmonary vascular resistance
BP and Afterload

- Blood pressure does not equal afterload

- Blood Pressure (MAP) = Cardiac Output x Systemic Vascular Resistance (Afterload)

BP = CO x 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

Invasive and Non Invasive Assessment of Afterload

<table>
<thead>
<tr>
<th>Right ventricular afterload</th>
<th>Left ventricular afterload</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Measured by pulmonary vascular resistance (PVR)</td>
<td>• Measured by systemic vascular resistance (SVR)</td>
</tr>
<tr>
<td>• Normal: 150-250 dynes/sec/cm(^{-5})</td>
<td>• Normal 900-1200 dynes/sec/cm(^{-5})</td>
</tr>
<tr>
<td><strong>Note:</strong> Any hypoxemia, positive pressure ventilation, and PEEP increase the workload of the right ventricle</td>
<td><strong>Diastolic BP is closest noninvasive measurement (narrow pulse pressure)</strong></td>
</tr>
</tbody>
</table>
Use of Pulse Pressure

- **PP < 35 with tachycardia** (in absence of beta blocker)
  - Early sign of inadequate blood volume

- **PP > 35 with tachycardia**
  - Early sign of oxygenation failure
    - Delivery cannot meet demand

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
Contractility

- 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

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

Contractility

- Low cardiac output does not necessarily mean diminished contractility

- Correct preload and afterload problems first
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)

- Consider as first line strategy to increase cardiac output when temporary pacemaker in place

Left Ventricular Function Curves

Preload: PAOP, S4, lung sounds, S3, etc.)
Changing Preload: moves patient along the curve they are on.

Changing Contractility: moves patient to a higher curve.
### Changing Afterload

*Moves patient up and to the left (improves forwards flow and reduces preload)*

![Graph showing the effect of changing afterload on CI and Preload](image)

### 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.
## Relationship of CI to Clinical Signs of Hypoperfusion

<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.

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**Backwards Failure:** Pulmonary Congestion

**Forwards Failure:** Hypoperfusion
Preload: PWP, lung sounds (dry or wet)

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

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

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

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

Hemodynamic and Clinical Subsets

Warm and Dry
- Normal Perfusion
- No Congestion

Warm and Wet
- Normal Perfusion
- Congestion

Cold and Dry
- Low Perfusion
- No Congestion

Cold and Wet
- Low Perfusion
- Congestion
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

![Diagram showing the relationship between preload and forwards flow/cardiac index with skin temperature (warm or cold).]

- **Preload Flow**: Cardiac Index
- **Skin temp (warm or cold)**

**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**
  
  \[ \text{CO} = \text{HR, preload, afterload, contractility} \]

- **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)

Contractility changes: move patient to a higher curve

Preload: PWP, lung sounds (dry or wet)
Linking Knowledge to Practice

And: Practice to Outcomes

Why is my patient compensating?
TACHYCARDIA
Use of Pulse Pressure

• PP < 35 with tachycardia (in absence of beta blocker)
  – Early sign of inadequate blood volume

• PP > 35 with tachycardia
  – Early sign of oxygenation failure
  • Delivery cannot meet demand

Stroke Volume Variation

• Remember – think LV ejection
  – Volume
  – Contractility

• Give volume 1st
• Then evaluate
• If needed – add inotrope
BP = CO x 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, drugs, anaphylaxis

Blood Pressure: CO x SVR

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

- BP: 82/30
  - Is problem cardiac output or SVR?
    - How to treat?
Pulmonary Artery Catheter Insertion Wave Forms
Pulmonary Artery Catheter and Cardiac Pressures

Right Atrial (RA) Waveform

- **a wave** = atrial contraction
  - Occurs after the P wave
- **c wave** = closure of tricuspid valve
  - Often not visible
  - Occurs at end of QRS complex
- **v wave** = atrial filling
  - Blood entering atrium from superior and inferior vena cava
  - Occurs after the T wave
- RA pressure is the clinical indicator of RV preload
- Direct measurement of right atrial pressure but an indirect measurement of right ventricular end diastolic volume.
- Document the mean of the **a wave** to obtain measurement
- Importance of normal tricuspid valve

Normal Value: 2-6 mm Hg
Right Ventricular (RV) Waveform

- Measured only during insertion
- PVCs or RBBB during insertion
- Rapid up stroke and rapid down stroke

Normal value: 15-30 / 2-8 mmHg

Pulmonary Artery (PA) Waveform

- Systolic pressure close to RV systolic pressure
  - Correlates with RV function
- Diastolic pressure rises
  - Indirect reflection of left heart
- Rapid up stroke and down stroke
- Dicrotic notch represents closure of pulmonic valve

Normal value:
  Systolic (PAS): 15-30 mm Hg
  Diastolic (PAD): 5-15 mm Hg
  Mean: 10-20 mm Hg
Pulmonary Artery Occlusive Pressure (PAOP) Waveform

- Intermittently assessed by inflating the balloon
- Inflation of the balloon stops forward flow of blood past the catheter tip
- Results in a static column of blood between the tip of the catheter and the left atrium
- Pressure in the left atrium is transmitted back to the catheter tip

![Diagram of PAOP Waveform]

Pulmonary Artery Occlusive Pressure (PAOP) Waveform

- **a wave** = atrial contraction
  - Occurs after QRS in ST segment
- **c wave** = closure of mitral valve
  - Not typically seen
- **v wave** = atrial filling
  - Occurs after T wave
- Document the mean of the **a wave** for measurement

**Normal value: 8-12 mmHg**
PAOP versus PAD

- PAD cannot be used to measure preload in the presence of pulmonary hypertension (hypoxia causes pulmonary hypertension)
- PAD > 5mmHg of PAOP = Pulmonary HTN
- PAD and PAOP should be within 5mmHg
- PAD is always higher than PAOP except in severe mitral regurgitation

Limitations of PAOP

- PAOP is not an accurate assessment of left ventricular end diastolic volume (LVEDV) with mitral valve disease, pulmonary vein disease, abnormalities with ventricular compliance, high alveolar pressures, and tachycardia.
- PEEP > 10 cm H₂O may falsely elevate PAOP.
Safety Principles for PAOP Monitoring

- Never inject > 1.5 cc of air into the balloon
- Stop injection of air when waveform changes to PAOP waveform
- Use minimal amount of inflation time; do not exceed 15 seconds
- Allow air to passively escape from the balloon; never aspirate air
- Never inject anything other than air into the balloon
- Always display waveform from distal tip on monitor
- PA waveform should always be visible unless balloon is inflated
- The presence of a PAOP waveform in the absence of balloon inflation indicates catheter migration, and the catheter needs to be withdrawn slowly until the PA waveform reappears
- Catheter should be securely sutured in place and catheter tip placement confirmed via chest X-ray

More on a Waves

- a waves occur during atrial contraction.
- Large a waves can occur with tricuspid or mitral stenosis, decreased ventricular compliance, and when there is a loss of AV synchrony.
- Large a waves are not an accurate reflection of filling pressures.
- Large a waves are called cannon waves when due to an arrhythmia
- A waves are absent in atrial fibrillation, junctional and ventricular rhythms, and ventricular paced rhythms.
- When a waves are absent, the PAOP is read at the end of the QRS complex.
More on a Waves

- a waves occur during atrial filling.
- The peak of the a wave occurs after the T wave.
- Large a waves can occur with tricuspid (RA) or mitral regurgitation (PAOP) or with non-compliant atria. Read the CVP or PAOP the same way as always: locate the a waves and document the mean of the a wave for CVP and PAOP pressures.
- Note: The sudden appearance of large a waves in a PAOP waveform suggests acute mitral valve regurgitation. Large a waves may cause the tracing to look more like a PA waveform giving the impression that the catheter did not wedge.

More on v waves

- V waves occur during atrial filling.
- The peak of the v wave occurs after the T wave.
- Large v waves can occur with tricuspid (RA) or mitral regurgitation (PAOP) or with non-compliant atria.
- Note: The sudden appearance of large v waves in a PAOP waveform suggests acute mitral valve regurgitation. Large v waves may cause the tracing to look more like a PA waveform giving the impression that the catheter did not wedge.
More on v waves

Expected Practice:
- Verify the accuracy of the transducer-patient interface by performing a square waveform test at the beginning of each shift.
- Position the patient supine prior to PAP/RAP (CVP) measurements. Head of the bed (HOB) elevation can be at any angle from 0° (flat) to 60°.
- Level the transducer air-fluid interface to the phlebostatic axis (4th ICS/½ AP diameter of the chest) with the patient in a supine position prior to PAP/RAP measurements.
- Obtain PAP/RAP measurements from a graphic (analog) tracing at end-expiration.
- Use a simultaneous ECG tracing to assist with proper PAP/RAP waveform identification.
- PA catheters can be safely withdrawn and removed by competent registered nurses.
Square Wave Testing

• Produced by a fast flush
• Repeated 2-3 times and record
  – Should return quickly to baseline
  – Should have 1 to 2 bounces before returning to the PA waveform
  – Second bounce should be \( \leq \frac{1}{3} \) the height of the first bounce (assesses damping)
  – Should be space of \(< 1\) block between bounces (assesses frequency)

Underdamped

• Greater than 2 blocks between bounces
• 2nd bounce close to height of 1st bounce
  • Overestimation of systolic pressure
  • Underestimation of diastolic pressures

• Causes:
  – Long tubing
  – Excessive stopcocks
  – Pinpoint air bubbles
Overdamped

- Absence of bounces
- Gradual descent to baseline
- Underestimation of systolic pressures
- Overestimation of diastolic pressures

**Causes:**
- Air
- Blood in catheter
- Kinks
- Soft (compliant) or long tubing

The Phlebostatic Axis

- 4th intercostal space, mid anterior-posterior chest
- Zero and take each measurement at the level of the phlebostatic axis
- Measurements can be obtained with backrest elevation from 0 – (45-60) degrees (no side lying)
Respiratory Patterns

- Spontaneous Breathing
  - Waveforms drop during inspiration
  - Waveforms rise during expiration
- Positive Pressure Breathing (Mechanical Ventilation)
  - Waveforms rise during inspiration
  - Waveforms drop during expiration

Potential Complications Related to PA Catheter

<table>
<thead>
<tr>
<th>During Insertion</th>
<th>Arterial puncture</th>
<th>Pneumothorax / hemothorax</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Avoid subclavian entry in COPD patients</td>
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<tr>
<td></td>
<td></td>
<td>Air embolism</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ventricular arrhythmias</td>
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<tr>
<td></td>
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<td>Heart block</td>
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<tr>
<td></td>
<td></td>
<td>Avoid insertion with LBBB or have pacing equipment available.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catheter knotting or kinking</td>
</tr>
<tr>
<td>After Insertion</td>
<td>PA rupture</td>
<td>PA infarction</td>
</tr>
<tr>
<td></td>
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<td>Infection</td>
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<td></td>
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<td>Ventricular arrhythmias</td>
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<td></td>
<td></td>
<td>Heparin induced thrombocytopenia (HIT) from heparin coated catheters</td>
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<tr>
<td></td>
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<td>Venous thrombosis</td>
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<td></td>
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<td>Endocardial / valvular damage</td>
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</table>
PA Rupture

<table>
<thead>
<tr>
<th>Prevention:</th>
<th>Treatment:</th>
</tr>
</thead>
<tbody>
<tr>
<td>– Avoid distal migration of catheter tip</td>
<td>– Deflate balloon / pull back catheter tip</td>
</tr>
<tr>
<td>– Use PAd instead of PAOP if accurate</td>
<td>– Stop anticoagulants</td>
</tr>
<tr>
<td>– Inflate with only the amount of air needed for occlusion and for the least amount of time possible</td>
<td>– Place patient in lateral position with affected side down</td>
</tr>
<tr>
<td>– Observe for spontaneous PAOP pressure indicating need to pull back PA catheter tip</td>
<td>– Selective bronchial intubation</td>
</tr>
<tr>
<td>– Pulling back is also indicated if able to wedge with &lt; 1.25 cc of air</td>
<td>– PEEP</td>
</tr>
<tr>
<td>– Pull back catheter tip before the initiation of cardiopulmonary bypass</td>
<td>– Surgical repair</td>
</tr>
</tbody>
</table>

Preventing Infection

– Strict sterile technique during insertion
– Dead end caps on all stopcocks
– Sterile sleeve over catheter
– Avoid glucose in IV solutions
– Change solution and lines no more frequently than every 72 to 96 hours
– Remove catheter as soon as clinically indicated
Transport of Gases in the Blood

• **Definition:** movement of oxygen and carbon dioxide through the circulatory system; oxygen being moved from the alveolus to the tissues for utilization and carbon dioxide being moved from the tissues back to the alveolus for exhalation.
Oxygen Delivery To Tissues

• Oxygen delivery measured as $DO_2$: Volume of oxygen delivered to tissues each minute

• $DO_2 = \text{cardiac output} \times \text{arterial oxygen content} \ (\text{hemoglobin} \times \text{arterial oxygen saturation})$

Oxygen Delivery to Tissues

• $DO_2$ formula = \textbf{CO x Hgb x SaO2} x 13.4 (constant)

• Normal $DO_2 = 900 - 1100 \text{ ml/min}$ (1000)

• Normal $DO_2$ I = 550 – 650 ml/min
Oxygen Delivery to Tissues

- Oxygen delivery can be improved by increasing cardiac output, hemoglobin or SaO2

Some interventions more effective in clinical practice; interventions can be performed simultaneously

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
Causes of Increased VO$_2$

- Fever per 1 degree C
- Shivering
- Suctioning
- Sepsis
- Non Family Visitor
- Position Change
- Sling Scale Weight
- Bath
- CXR
- Multi Organ Failure

- 10%
- 50-100%
- 7-70%
- 5-10%
- 22%
- 31%
- 36%
- 23%
- 25%
- 20-80%

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

<table>
<thead>
<tr>
<th>( \text{DO}_2 )</th>
<th>( \text{VO}_2 ) (extraction is independent of delivery)</th>
<th>( \text{SVO}_2 ) (( \text{SV}0_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>

Relation of Delivery to Consumption

- When oxygen delivery reaches a critical level then consumption will depend on delivery.
- \( \text{SVO}_2 \) will not increase with increased delivery while you are in this dependent state.
- Anaerobic metabolism occurs here because you have an oxygen deficit.
**SVO\textsubscript{2} Monitoring**

- 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\textsubscript{2}**

- SVO\textsubscript{2} < 60%
  - Decreased delivery
  - Increased consumption
- SVO\textsubscript{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\textsubscript{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)
Shock

• Definition:
  – The 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
## Signs and Symptoms of Shock Stages

### Sub clinical

**Hypoperfusion**
- CI 2.2-2.5
- No clinical indications of hypoperfusion yet something seems different or not right

### Compensatory

**with SNS Stimulation**
- CI 2.0 – 2.2
- Tachycardia
- Narrowed pulse pressure
- Tachypnea
- Cool Skin
- Oliguria
- Decreased Bowel sounds
- Restlessness / confusion

### 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
Outcome Indicators

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

• Others:
  – Mentation
  – Urine Output

Shock Classifications

• Hypovolemia
• Cardiogenic
• Distributive
  – Anaphylactic
  – Septic
  – Neurogenic
  – Nonspecific vasodilatory
• Obstructive
The Body Knows the Algorithm

• When perfusion to the tissues decrease for whatever reason the body launches a response
• The body begins to adjust to improve cardiac output

• Two primary responses
  – Sympathetic nervous system
  – Renin angiotensin aldosterone system

• Thus:
  – Increase HR
  – Increase in SVR
  – Unless the body cannot!!

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
Hypovolemic Shock

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

**Hemodynamic Profile**
- ___ HR
- ___ Preload (RA, PAOP)
- ___ Afterload (SVR)
- ___ Contractility
- ___ Stroke Volume
- ___ CO / CI
- ___ Blood Pressure
- ___ $SvO_2$

---

**Table 4.6**
Clinical Findings Associated with Progressive Loss of Blood

<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>
<tr>
<td>500-1,000 cc</td>
<td>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.</td>
</tr>
<tr>
<td>1,000-2,000 cc</td>
<td>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. $SvO_2 &lt; 60%$. 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.</td>
</tr>
<tr>
<td>2,000-3,000 cc</td>
<td>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. $SvO_2 &lt; 55$ to 60%. Oliguria or anuria. Mental status: Encephalitis cold. Peripheral pulses poor. Peripheral cyanosis. Cardiac output decreased. Severe acidosis.</td>
</tr>
</tbody>
</table>
Hypovolemic Shock
Treatment

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

- **CI**
  - Volume replacement
    - Isotonic crystalloids first (0.9NS or Lactated Ringers)
    - Colloids if crystalloids not effective

- **Hemoglobin**
  - Replace blood loss if necessary for tissue oxygenation
    - Need hemoglobin to carry blood
    - Treat cause of loss

- **SaO2**
  - Oxygen
  - Intubation and mechanical ventilation if needed

---

Fluid Replacement

<table>
<thead>
<tr>
<th><strong>Crystalloids</strong></th>
<th><strong>Colloids</strong></th>
<th><strong>Blood Products</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Isotonic</strong></td>
<td>Albumin (costly) Plasma protein fraction</td>
<td>Whole Blood Packed red cells Fresh Frozen Plasma</td>
</tr>
<tr>
<td>NS, LR (D₅NS, D₅LR)</td>
<td>Dextran 70/75* Hetastarch (Hespan) *</td>
<td></td>
</tr>
<tr>
<td><strong>Hypotonic</strong></td>
<td>•Decrease platelet aggregation</td>
<td></td>
</tr>
<tr>
<td>.45NS, (D₅.45NS), D₅W</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hypertonic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3%NS, TPN</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CNEA / Key Choice
Crystalloids and Colloids

- **Isotonic Crystalloids**
  - Osmolality close to blood osmolality
  - Stay in vascular space better than other crystalloids
  - Safe, effective and inexpensive; usually first line

- **Colloids**
  - Large molecule solutions (either protein or starch)
  - Stay in vascular space better than crystalloids
  - Support intravascular colloidal oncotic pressure to help pull more fluid into the vascular space
  - Consider in hypovolemic shock when crystalloids are not effective
    - Non-protein colloids
      - Dextran or Hetastarch

More About Fluid Replacement

- **Hypovolemic and Neurogenic Shock**
  - Crystalloids and Colloids may be used
  - Blood for hemorrhagic

- **Septic and Anaphylactic Shock**
  (and early burns)
  - "leaky states" with increased capillary permeability
  - Crystalloids only (at least initially)

- **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**
Cardiogenic Shock
(Pump Failure)

- Massive insult to the left ventricle resulting in profound left ventricular dysfunction
  - Myocardial infarction
  - End stage heart disease
  - Acute Myocarditis
- Decreased contractility
  - Decreased stroke volume / cardiac output
  - Backward failure / increased preload
- Compensatory results
  - Increased systemic vascular resistance (increased afterload) and heart rate
  - Further impedes ejection and increases myocardial oxygen demand

Presentation
- Systolic BP < 90 mm Hg
- Decreased sensorium
- Cool, pale, moist
- Peripheral cyanosis
- Decreased urine output
- Tachycardia
- Weak thready pulse
- Tachypnea
- Hypoxia
- S3, S4
- JVD

Hemodynamic Profile
- ___ HR
- ___ Preload (RA, PAOP)
- ___ Afterload (SVR)
- ___ Contractility
- ___ Stroke Volume
- ___ CO / CI
- ___ Blood Pressure
- ___ SV\textsubscript{O2}
Treatment

• Treatment Goal: Maximize the delivery of oxygen to the tissues
  – Remember: CI, Hemoglobin, SaO2

• CI
  – Optimize preload and afterload first
    • Preload reduction when PAOP > 20-22 mm Hg or when pulmonary edema is present
      – Diuretics
      – Venous vasodilators
    • Afterload reduction
      – Careful consideration in hypotension
      – Arterial vasodilators
      – IABP
      – Vasopressor use can adversely impact myocardial function
  – Improve contractility to maximize stroke volume if needed
    • Inotropes
      – Dobutamine
      – Milrinone
    – Revascularization if ischemia or infarction

• Hemoglobin
  – Maintain hemoglobin to support myocardial oxygen supply

• SaO2
  – Oxygen
  – Intubation and Mechanical Ventilation

Anaphylactic Shock
(Distributive)

• 1<sup>st</sup> Exposure to allergen (drugs, contrast, foods, food additives, latex, blood products, insect venom)
  – Release of IgE
    • IgE attaches to mast cells (tissues) and basophils (vascular)

• 2<sup>nd</sup> Exposure
  – Immune reaction (releasing histamines)
    • Decreased circulating volume and hypotension
      – Potent vasodilator
      – Increased capillary permeability
      – Bronchoconstriction
    – Other mediators released / complement system activated
Anaphylactic Shock

Presentation
- Pruritus
- Generalized erythema, urticaria
- Angioedema
- Restless, uneasy, apprehensive, anxious
- Laryngeal edema
- Bronchoconstriction
  - Coughing, choking, wheezing, shortness of breath
- Hypotension
- Chest tightness
- Reflex tachycardia
- Cramping

Hemodynamic Profile
- ___ HR
- ___ Preload (RA, PAOP)
- ___ Afterload (SVR)
- ___ Contractility
- ___ Stroke Volume
- ___ CO / CI
- ___ Blood Pressure
- ___ SV$O_2$

Anaphylactic Shock

Treatment
- **Treatment Goal:** Maximize the delivery of oxygen to the tissue
  - **Remember:** CI, Hemoglobin, SaO2
- Remove offending agent or slow absorption (ice, flush skin, lavage)
- Modify or block effects of mediators
  - Epinephrine (SNS stimulation)
  - Antihistamines
  - Diphenhydramine (H1 blocker)
  - Ranitidine (H2 blocker)
  - Steroids (decrease capillary permeability, prevent delayed reaction)
- **CI**
  - Volume replacement – crystalloid or colloid
  - Vasopressors
- Hemoglobin
- SaO2
  - Manage airway
  - Bronchodilatation – epinephrine, aminophylline
  - Oxygen
  - Intubation and Mechanical Ventilation
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

Hemodynamic Profile
Early Septic Shock
• ___ HR
• ___ Preload (RA, PAOP)
• ___ Afterload (SVR)
• ___ Contractility
• ___ Stroke Volume
• ___ CO / CI
• ___ Blood Pressure
• ___ $S_VO_2$

Hemodynamic Profile
Late Septic Shock
• ___ HR
• ___ Preload (RA, PAOP)
• ___ Afterload (SVR)
• ___ Contractility
• ___ Stroke Volume
• ___ CO / CI
• ___ Blood Pressure
• ___ $S_VO_2$
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

- **CI**
  - Volume replacement
    - Crystalloids 4-8 liters
  - Vasopressors
  - May need inotropes

- **Hemoglobin**
  - Transfuse as necessary to support tissue oxygenation

- **SaO2**
  - Oxygen
  - Intubation and Mechanical Ventilation

### Early Goal Directed Therapy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVP</td>
<td>8-12 mm Hg</td>
</tr>
<tr>
<td>PAOP</td>
<td>12-18 mm Hg</td>
</tr>
<tr>
<td>MAP</td>
<td>&gt; 65 mm Hg</td>
</tr>
<tr>
<td>U.O.</td>
<td>&gt; 0.5 cc/kg/hr</td>
</tr>
<tr>
<td>SaO2</td>
<td>&gt; 90%</td>
</tr>
<tr>
<td>Central venous oxygen saturation</td>
<td>&gt; 70%</td>
</tr>
</tbody>
</table>

Neurogenic Shock (Distributive)

- Loss of autonomic control (sympathetic tone)
  - defect in vasomotor center of the brainstem
  - or interruption in the outflow communicating pathways to the vessels

- Profound venous and arterial vasodilatation

- Decreased venous return and decreased SVR

- Decreased preload and stroke volume

- No sympathetic HR response

- Least common form of shock

- Most common in spinal cord injuries above T4

- Hypotension
- Bradycardia
- Hypothermia
- Warm and dry
Neurogenic Shock
Treatment

- **Treatment Goal:** Maximize the delivery of oxygen to the tissue
  - *Remember:* CI, Hemoglobin, SaO2
- **CI**
  - Fill vascular spaces without increasing pressure on spinal column
  - Volume replacement with colloids
  - Avoid hypotonic solutions – can increase cord pressure
  - Atropine for HR
  - Vasopressors – response may be abnormal – interruption of SNS
    - Vasopressin may be needed
- **Hemoglobin**
- **SaO2**
  - Oxygen
  - Intubation and Mechanical Ventilation

- Maintain normal body temperature to prevent shift in oxyhemoglobin curve

---

Nonspecific Vasodilatory Shock
(Distributive)

- Newer category
- May be seen post operative CABG
- Loss of response to catecholamines
- Utilization of vasopressin or methylene blue
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
  - Constrictive pericarditis
  - Pneumothorax
  - Hemothorax
  - Dissecting aneurysm

Treatment Focus
- Removing obstruction to flow
  - Surgery
  - Fibrinolytic therapy
- Bedside cardiopulmonary bypass if CPR needed

Linking Knowledge to Practice

And: Practice to Outcomes
## HEMODYNAMIC PROFILES VARY BY TYPE OF SHOCK

### 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>
</tbody>
</table>
Treatment Focus

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>Cardiogenic</th>
<th>Septic (Distributive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>√ Replace volume</td>
<td>√ Support stroke volume</td>
<td>√ Fill up container</td>
</tr>
<tr>
<td>√ Stop volume loss</td>
<td>√ Decrease preload</td>
<td>√ Use vasopressors to restore vascular</td>
</tr>
<tr>
<td></td>
<td>√ Decrease afterload (think IABP)</td>
<td>tone</td>
</tr>
<tr>
<td></td>
<td>√ May need to support contractility</td>
<td>√ Treat underlying cause</td>
</tr>
</tbody>
</table>

Case Study 1

- 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. A SCVO2 catheter is placed in the ED. The CVP is 1 mmHg and the venous oxygen saturation is 50%.
Case Study 1

- Preload?
- Afterload?
- Type of shock?

- Needed interventions?
- Follow up assessment for effectiveness?

Case Study 2

- 85 year old female is in the coronary unit after experiencing CP for 6 hours. History of two previous MI’s. Admission EKG shows old inferior MI and ST elevation from V_2-V_6. Received thrombolytic therapy. On 1\textsuperscript{st} hospital day EKG show Q waves in V_2-V_6. She is now hypotensive with an S3 and crackles in the lung bases. Urine output is marginal for the last two hours. Pulmonary artery catheter is placed.
Case Study 2

- BP: 88/70
- MAP: 76
- HR: 128
- RA: 8 mmHg
- PA: 42/26 mmHg
- PAM: 31 mmHG
- PAOP: 22 mmHG
- CO: 3.0 L/min
- CI: 1.8 L/min
- SV: 23 ml/beat
- SVR: 1813 dynes/sec/cm⁻⁵
- PVR: 240 dynes/sec/cm⁻⁵
- LVSWI: 10.3g m/m²
- RVSWI: 2.7g m/m²

Case Study 3: The Nuances of the Patient with Poor LV Function

- 85 year old female living independently at home with help of family is admitted to the hospital with shortness of breath.
- She has become increasingly short of breath the past 2 days. Her weight is up 6 pounds from 3 days ago.
- She has not taken her medications for 4 days because her prescriptions ran out and she was waiting on her oldest son to get back in town to get them filled.
Physical Assessment

BP: 88/60
HR: 110’s to 130’s
Rhythm: Atrial Fib
Frequent ventricular ectopy
RR 28-32

SaO2 88% on 4L nasal cannula

Pale and cool to touch
Somewhat lethargic
Mild right sided weakness
Faint pedal pulses

Heart Sounds
S3
Systolic murmur 3/6
Lungs with crackles ½ up bilaterally

JVD
Right Upper quadrant tenderness
2+ peripheral edema to mid calf

Urine output is 10cc in first hour

Evaluation of Cardiac Output

Cardiac Output

• What is the formula for Cardiac Output?
• What non invasive parameter correlates with SV?

• Preload?
  • JVD
  • Lung sounds
  • S3
• Afterload?
  • Pulse pressure
• Contractility?
  • EF
• HR?
  • Compensation
  • Beta blockade
Hemodynamic Profile for Cardiogenic Shock

- CO
- SV
- Preload
- Afterload
- Contractility
- HR

What is the profile for our patient?

Case Study 3

- 65 year old obese female S/P abdominal surgery two weeks ago. She has been recovery at home. She present to the ED with chest pain, shortness of breath and a feeling of doom. ABG’s show respiratory alkalosis and hypokalemia. After she is transferred to the ICU a pulmonary artery catheter is inserted.
Case Study 4

- BP: 112/84 mm Hg
- MAP: 93 mm Hg
- HR: 110 BPM
- RA: 18 mmHg
- PA: 55/32 mmHg
- PAM: 40 mmHg
- PAOP: 6 mmHg
- CO: 4.4 L/min
- CI: 2.75 L/min
- SV: 40 ml/beat
- SVR: 1364 dynes/sec/cm$^5$
- PVR: 618 dynes/sec/cm$^5$
- LVSWI: 30g m/m$^2$
- RVSWI: 7g m/m$^2$

Always Rule Out Obstructive Shock: Treatment is to correct obstruction to forward flow.

Think:
PE
Aortic Dissection
Tamponade
Tension pneumothorax
Assessment Considerations for Emergencies

• Tamponade
  – Risk factors (trauma, post OHS / procedure, large MI – late presentation)
  – Beck’s triad
  – Pulses Paradoxus
  – Pulses / Electrical Alternans

• Tension Pneumothorax
  – Risk factors (Conversion to PPV, existing chest tube, trauma)
  – Lung sounds
  – Hypotension
  – JVD
  – Mediastinal shift (very late sign)

• Pulmonary Embolus
  – Risk factors (Virchow's triad)
  – Tachypnea (most common sign)
  – Respiratory Alkalosis
  – ECG signs
    • Right axis deviation
    • RBBB
    • Tall P waves inferior leads
    • T wave inversion (limb and precordial leads)
    • Prominent S waves I and aVL
    • S1, Q3, T3
  – ST / New atrial arrhythmia

Assessment Considerations for Emergencies

• Acute Aortic Dissection
  – Risk factors (HTN most common)
  – Tearing or ripping description of CP or back pain
  – Diastolic murmur of Aortic regurgitation
  – Bilateral arm BP variation
  – 4 extremity pulse variation
  – Co-existing Inferior MI

• Mechanical Complications of MI
  – Warning signs – regional pericarditis (failure of T wave to invert within 48 hours, premature reversal of T wave inversion, Relevation of ST segments)
  – Papillary Muscle Rupture
    • Holosystolic murmur
    • Acute pulmonary edema
  – VSD
    • Continuous murmur
Knowing is not enough; we must apply.
Willing is not enough; we must do.

Johann Wolfgang von Goethe