Hemodynamic Monitoring

University of South Alabama
Cardiorespiratory Care
Definition

Hemodynamic monitoring: continuous monitoring of the movement of blood and the measurement of vascular pressures exerted in the veins, arteries, and chambers of the heart.
Methods of Hemodynamic Monitoring

- Arterial blood pressure
  - non-invasive
  - invasive
- Central venous pressure
- Pulmonary artery catheter
- Cardiac output measurement
- Tissue oxygenation
Arterial Pressure Monitoring

- **Invasive:** more accurate

- **Common Catheter Insertion Sites:**
  - Radial artery
  - Brachial artery
  - Femoral artery
Radial Artery Site

- Ease of access
- Control of bleeding
- Maximal mobility for patient
- Collateral circulation via ulnar and radial arteries
Collateral Circulation in the Hand
Invasive Arterial Pressure Monitoring

Advantages

- Accuracy
- Continuous
- Waveform/ECG
- Access for blood samples
Invasive Arterial Pressure Monitoring

Disadvantages

• Expense
• Infection
• Clot formation
• Damage to artery
• Hemorrhage
• Pain/discomfort
Arterial Waveform

Dicrotic notch: closing of the semilunar valves, especially the aortic valve.

Incisura = dicrotic notch
180 msec delay between R wave & upstroke of systole; delay represents time between actual ventricular depolarization & arrival of signal to transducer.
Arterial waveform in hypertension and peripheral vascular disease

- Reflected wave
- Dicrotic notch
- Reflected wave
The further away the insertion site is from the aorta:

- the higher the systolic pressure
- the further down the dicrotic limb the dicrotic notch is located
- the lower the diastolic pressure
- the wider the pulse pressure

MAP does not change much from aorta to radial artery because resistance to flow changes little between these two sites.

MAP begins to change more when blood reaches the arterioles.

Vasodilation: incisura closer to baseline. Vasoconstriction: incisura further from baseline.
Arterial Waveform

- The arterial pressure wave is a shockwave.
- It travels much faster than the blood which is ejected.
- It represents the impulse of left ventricular contraction, conducted though the aortic valve and systemic arteries along a fluid (blood) column.
  - Shockwave then travels up a catheter
  - Then up another fluid (normal saline) column within stiff tubing
  - Into a Wheatstone bridge, i.e., the pressure transducer
Dicrotic notch

Pulse Pressure (PP)

Diastole

Systole

Time (seconds)
INTERPRETING THE ARTERIAL WAVEFORM

As pressure falls, the aortic valve closes - signaling the onset of diastole. Aortic valve closure produces a characteristic waveform known as the dicrotic notch (see above). As diastole progresses, the pressure falls to its lowest level. The lowest value of the arterial waveform is the diastolic pressure. Normal diastolic pressure ranges between 60 and 90 mmHg.

Mean Arterial Pressure (MAP) = \( \frac{\text{Systolic} + (\text{Diastolic} \times 2)}{3} \) = 93 mmHg

BP (mmHg) anacrotic limb dicrotic limb dicrotic notch
Systolic = 120 mmHg
Pulse Pressure = Systolic - Diastolic = 40 mmHg
Diastolic = 80 mmHg
# Arterial Pressures

## Normal Values

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Normal (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td>90 to 140</td>
</tr>
<tr>
<td>Diastolic</td>
<td>68 to 90</td>
</tr>
<tr>
<td>Mean</td>
<td>70 to 100</td>
</tr>
</tbody>
</table>

**Abnormal Values:**
- **Hypertension:** > 140 mm Hg/ > 90 mm Hg
- **Hypotension:** < 100 mm Hg/ < 60 mm Hg
Dangerously Low Pressures

- Diastolic $< 50$ mm Hg
- Mean $< 60$ mm Hg
- May compromise coronary perfusion
Arterial Catheter Insertion
Arterial Catheter Insertion
Arterial Line Maintenance

- Continuous heparinized flush
- 2 to 3 ml/hour
- 1.0 L of 0.9% NS with 100 to 200 units of heparin
- Watch for infection, ischemia, and bleeding
- Infection risk increased
  - Insertion percutaneously or by cut-down
  - Cannulation > 4 days
  - Altered host defense
Calculations
MAP and SVR

\[
\text{MAP} = \text{Systolic Pressure} + 2 \left( \frac{\text{Diastolic Pressure}}{3} \right)
\]

\[
\text{SVR} = \frac{\left( \text{MAP} - \text{RAP} \right)}{\text{C.O.}} \times 80
\]

Normal range for SVR = 900 to 1400 dynes x sec/cm\(^5\)
(dynes x second x cm\(^{-5}\))
Arterial Pressure Decreases

- Hypovolemia: fluid or blood loss
- Cardiac failure and shock (hypotension)
- Vasodilation
Arterial Pressure Increases

- Increased circulatory volume (hypervolemia)
- Increased cardiac function
  - increased HR
  - increased force of contractility
- Sympathetic stimulation
- Vasoconstriction
- Inotropic agents
- Vasopressors
Pulse Pressure

- Difference between systolic and diastolic pressures (PP = SP – DP)
- Normal: 30 to 40 mm Hg
- Increased pulse pressure: increased SV and decreased arterial compliance
- Decreased pulse pressure: decreased SV and increased arterial compliance
Respiratory Induced Changes

*Pulsus paradoxus*

Decreased SP > 10 mm Hg during inspiration.
Central Venous Pressure

Measured in either:

- Vena cava
- Right atrium
  - $\text{CVP} = \text{RAP}$
Central Venous Line Subclavian Vein
Central Venous Line Subclavian Vein

Diagram showing the insertion site of a central venous line into the subclavian vein, tunnelled under skin, and exiting from a different site.
CVP Catheter and Insertion Sites

- External jugular vein
- Internal jugular vein
- Subclavian vein
- Antecubital fossa (cephalic or basilic veins)
- Femoral vein
Advantages of Internal Jugular Vein

- Ease of insertion
- Low risk of pneumothorax
- Good visibility
Advantages of Subclavian Vein

- Easier to stabilize
- Less likely to kink, separate, or break
Central Venous Pressure

- **Measures**
  - Preload of RV using end-diastolic pressure (EDP) of RV, i.e., RVEDP
  - Right heart function

- **Reflects (in young, healthy patients)**
  - Left heart filling pressures
  - $LVEDP = (2 \times RVEDP) + 2$
    - E.g., RVEDP 2 mm Hg; LVEDP 6 mm Hg
Normal CVP

- 2 to 6 mm Hg via pressure transducer
- < 12 cm H₂O by water column
- **Conversion:** 1.36 cm H₂O = 1 mm Hg
  - 1,034 cm H₂O/760 mm Hg = 1.36 cm H₂O/mm Hg
COMPONENTS OF THE CENTRAL VENOUS PRESSURE (CVP) WAVE

Central venous waves are created by the pressure changes occurring in the right atrium during right atrial systole and right atrial diastole. The central venous waveform consists of the following parts: A wave, C wave, X descent, Y wave, and Y descent.

The A wave is generated during atrial systole and its height is a direct result of how much pressure is occurring in the atrium as the blood is being ejected into the ventricle.
## Central Venous Pressure Waveform Components

<table>
<thead>
<tr>
<th>Waveform Component</th>
<th>Phase of Cardiac Cycle</th>
<th>Mechanical Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>a wave</td>
<td>End diastole</td>
<td>Atrial contraction</td>
</tr>
<tr>
<td>c wave</td>
<td>Early systole</td>
<td>Tricuspid bulging</td>
</tr>
<tr>
<td>v wave</td>
<td>Late systole</td>
<td>Systolic filling of the atrium</td>
</tr>
<tr>
<td>x descent</td>
<td>Mid-systole</td>
<td>Atrial relaxation</td>
</tr>
<tr>
<td>y descent</td>
<td>Early diastole</td>
<td>Early ventricular filling</td>
</tr>
</tbody>
</table>
CVP Decreases

- Decreased venous return (decreased RV preload)
- RVEDP represents RV preload
- Hypovolemia (decreased intravascular volume)
- Decreased intrathoracic pressure
- Improved cardiac function in patients with right heart failure
CVP Decreases

- Inadequate volume or volume loss
- Vasodilation
- Spontaneous inspiration
- Technical problems with measurement
CVP Increases

- Increased venous return (increased volume)
- Increased intrathoracic pressure (compression effect on myocardium)
- Right ventricular failure (e.g., peripheral blood accumulation & JVD)
Causes of High CVP

- Volume overload
- Heart compression
- Pulmonary hypertension
- High intrathoracic pressures
- High ventilator pressures
- I.V. infusion
- RV failure
- LV failure
- Pulmonary stenosis
- Tricuspid valve abnormalities
- Pulmonary embolism
- Vasoconstriction
- Vasodilation
Pulmonary Artery Catheter

Swan-Ganz Catheter
Pulmonary Artery Catheter
Pulmonary Artery Catheter

- PAC has 4-5 lumens:
  - Temperature thermistor located proximal to balloon to measure pulmonary artery blood temperature
  - Proximal port located 30 cm from tip for CVP monitoring, fluid and drug administration
  - Distal port at catheter tip for PAP monitoring
  - +/- Variable infusion port (VIP) for fluid and drug administration
  - Balloon at catheter tip
Pulmonary Artery Catheterization

- A large-bore introducer catheter is used to facilitate PAC insertion

- Inserted through the subclavian or internal jugular vein with the patient in Trendelenburg

- Prior to PAC insertion,
  - Connect the distal port (yellow) to the pressure transducer
  - Level the transducer at the level of the patient’s heart
  - Zero the transducer
PAC Insertion
Characteristics of PAC

- 110 cm long
- Distal lumen at tip
- Balloon at distal tip
- Balloon holds 1.5 ml of air
- Marked in 10 cm increments
- Radiopaque polyvinylchloride
- Proximal lumen 30 cm from distal tip
- Thermistor bead 1½ inches from the distal tip
Pulmonary Artery Catheterization

PAC as seen on chest radiograph
Normal Pulmonary Artery Pressures

- PA systolic = 20 to 30 mm Hg
- PA diastolic = 6 to 15 mm Hg
- Mean PAP (PAP) = 10 to 20 mm Hg
- PCWP = 4 to 12 mm Hg
Synonyms

- Pulmonary capillary wedge pressure (PCWP)
- Pulmonary artery wedge pressure (PAWP)
- Wedge pressure
- Pulmonary artery occlusion pressure (PAOP)
PA Pressures Decrease

- Volume ejected by right ventricle decreases
- Dilation of pulmonary vasculature (↓PVR)
PA Pressures Increase

- Increased pulmonary blood flow
- Constriction of pulmonary vasculature ($\uparrow$PVR)
- Pulmonary hypertension (increased RV afterload)
Causes of ↑ Pulmonary Blood Flow

- Volume overload (hypervolemia)
- Left-to-right intracardiac shunts
  - Atrial septal defect (ASD)
  - Ventricular septal defect (VSD)
  - Patent ductus arteriosus (PDA)
Determinants of Cardiac Output

- **Preload**
  - Estimated by end-diastolic pressure
  - CVP for RVEDV; PCWP for LVEDV

- **Afterload**
  - LV: \( SVR = \frac{[MAP - CVP]}{C.O. \times 80} \)
  - RV: \( PVR = \frac{[PAP - PCWP]}{C.O. \times 80} \)

- **Contractility**
PVR Calculation

- \[ PVR = \frac{PAP - PCWP}{C.O.} \times 80 \]

- Normal = 100 to 250 dynes x sec/sec^5  
  (dynes x second x cm^{-5})
Causes of ↑ PVR

- Pulmonary emboli (obstruct flow)
- Pulmonary parenchymal disease
- Cardiac tamponade
- Increased intrathoracic pressure
- LV failure
- Mitral stenosis
- Hypoxemia
- Hypercapnia
- Acidemia
- Vasoconstrictive drugs
PAd Pressure

Indicates LV function in absence of:

- Severe pulmonary vascular changes
- Marked tachycardia
- Pulmonary embolism
PAd vs PCWP

- PAd usually ~ 2 mm Hg higher than PCWP
- PAd-PCWP gradient > 5 mm Hg
  - ARDS
  - Sepsis
  - Excessive PEEP
  - Other causes of ↑PVR
- PAd-PCWP gradient < 5 mm Hg with pulmonary hypertension
  - Increased LV pressures
Criteria for Accurate PCWP

- Distinct and valid pulmonary artery tracing
- Catheter tip in zone III
- Distinct PCWP tracing immediately when tip wedged
- Free flow with catheter wedged
Criteria for Accurate PCWP

- Change in PCWP < $\frac{1}{2}$ change in airway pressure
- Aspiration of “arterialized” blood from distal port when wedged
Lung Zones

- PAC distal tip must be in zone III
- $P_a > P_v > P_{alv}$
- Continuous column of blood is present between catheter tip and LA
- Avoids compression by pulmonary pressures
The PAWP should always be lower than the mean pulmonary artery pressure (PAP). If it appears higher than mean PAP, suspect an analytical error or that the catheter tip is not in zone III of the lung. If the catheter tip is not in zone III, the PAWP may reflect alveolar or airway pressure and would not accurately reflect left atrial pressure. In addition to the mean PAP pressure being higher than PAWP, pulmonary artery diastolic (PAD) pressure should be higher than PAWP. This is because the higher pressure in the pulmonary artery is needed to push the blood to the left atrium.
Biomedical Recording Instruments

Physiological Signal → Transducer → Amplifier → Recorder
Transducer

• Two stopcocks
  – Catheter
  – Air reference

• Diaphragm

• Flush valve

• Clear fluid-filled chamber

• Monitor connection
Pressure bag with flush solution

2-3 mL/hr

Manometer

Thermocilution monitor connector

CVP port

PA port

Stopcock

To catheter
Phlebostatic Axis

Junction between the transverse plane of the body passing through the 4th intercostal space at the lateral margin of the sternum and a frontal plane of the body passing through the midpoint of a line from the outermost point of the sternum to the outermost point of the posterior chest, i.e. mid-axillary level at the 4th intercostal space.
Leveling and Zeroing

Pressure transducer must be at the same level as catheter tip.

*Phlebostatic axis*
Leveling and Zeroing

Pressure transducer level with phlebostaic axis: pressure measurement accurate.

Pressure transducer below phlebostaic axis: pressure measurement higher than actual.

Pressure transducer above phlebostaic axis: pressure measurement lower than actual.

Leveling corrects for hydrostatic pressure. Zeroing corrects for atmospheric pressure.
Phlebostatic Axis

Mid-chest level/phlebostatic axis:
- Junction of the nipple line and anterior axillary line
- ½ way between anterior and posterior chest wall
- 10 cm from the top of the mattress
- 4th intercostal space and anterior axillary line
- 4th intercostal space and mid-axillary line

Pressure transducer location is based on the patient’s body position.
Cardiac Output

• **Volume (L) of oxygenated blood pumped by LV per minute (L/min)**

• **C.O. = HR X SV**

• **Fick equation:**

\[ \dot{Q}_T = \frac{\dot{VO}_2}{CaO_2 - C\bar{VO}_2} \]

• **Normal C.O. = 4 to 8 L/min**

• **Normal CI = 2.4 to 4 L/min/m^2**
Stroke Volume

• Volume of blood ejected with each beat

• Determined by:
  – Preload: stretch on ventricular (myocardial) fibers before contraction
  – Afterload: resistance (sum of external forces opposing ejection of SV)
  – Contractility: strength of ventricular (myocardial) contraction
Determinants of Pump Function

- C.O. = HR x SV
- Stroke Volume
  - Preload
  - Afterload
  - Myocardial contractility
Factors Affecting Preload

- Ventricular compliance
- Changes in blood volume
- Changes in distribution of blood volume
- Atrial contraction
- Cardiac tamponade
Ventricular Compliance

- **Decreased Compliance:**
  - Myocardial ischemia and infarction
  - Hemorrhage and septic shock
  - Pericardial effusions
  - Right ventricular dilation and overload
  - PEEP
  - Inotropic drugs

- **Increased Compliance:**
  - Relief of ischemia
  - Vasodilator drugs
  - Cardiomyopathies
Changes in Blood Volume

- **Loss of Blood Volume:**
  - Hemorrhage
  - Loss of other body fluids (diuresis, wound drainage, diarrhea, perspiration, vomiting, gastric suctioning, etc.)
  - Shift into interstitial space (third space)

- **Gain of Blood Volume:**
  - Fluids ingested
  - Intravenous fluids
  - Hyperosmolar solutions
  - Respiratory care humidification (children)
Atrial Contraction

- Normally contributes 30% to loading of ventricle
- Lost with certain dysrhythmias
  - Atrial fibrillation
  - Atrioventricular dissociation (3rd degree heart block)
  - Ventricular pacemaker
Spontaneous Ventilation and Preload

- Normal inspiration: ↓ intrapleural pressure to -6 cm H$_2$O & ↑ venous return
- Labored breathing: paradoxical pulse; ↓ in BP during inspiration > 10 mm Hg
- Increased intrapleural pressure (e.g., Valsalva maneuver) ↓ venous return
Mechanical Ventilation and Preload

Positive pressure impedes venous return dependent upon transference of pressure to pleural space which in turn is based on balance between chest wall and lung compliance.

- Compliances are equal: ½ airway pressures transferred
- ↓ lung compliance: less transferred
- ↓ chest wall compliance: more transferred
- ↓ chest wall compliance & ↑ lung compliance: even greater transference
Afterload

- **Tension (Stress) in ventricular wall**
  - Ventricular distension
  - Intraventricular pressure
  - Ventricular wall thickness
  - Intrathoracic pressure

- **Systemic (peripheral) vascular resistance**
  - Elasticity of blood vessels
  - Radius of vessels
  - Viscosity of blood
  - \( \Delta P \) from one end of vessel to the other
Increased SVR

Increased mycardial O$_2$ demand

Decreased C.O.

Decreased ventricular efficiency

Cycle of Coronary Insufficiency

Increased vasoconstriction
Myocardial Contractility

- Change in initial muscle length caused by stretch of muscle (preload): Starling’s law of the heart
- Change in contractility (inotropic state) of the heart
  - Sympathetic nerve stimulation
  - Inotropic drugs
  - Physiological depressants
  - Damage to myocardium
  - Coronary blood flow
C.O. Determination

- Dye-dilution cardiac output
- Thermodilution cardiac output
- Continuous cardiac output monitoring
- Fick Method
C.O. Determination Methods

• New technology – Pulse contour analysis
• PiCCO – Transpulmonary thermodilution
• LiDCO – Lithium chloride indicator dilution method
• PRAM – Self-calibration method
• FloTrac/Vigileo – Self-calibration method
Dye-dilution Method
Thermodilution Method
Thermodilution Method

• The thermodilution method adapts the indicator dilution principle to injectates that cause changes in blood temperature detected downstream.
• An injectate of known volume and temperature is injected into the right atrium and cools the blood.
• Cooled blood traverses a thermistor in the PAC’s distal tip downstream over time.
• C.O. is inversely proportional to mean blood-temperature depression & duration of cooled blood
• C.O. is a function of temperature change over time (i.e., area under the curve).
Thermodilution C.O.

Phase 1
Début de l’injection rapide d’un injectat
Site d’injection dans l’oreillette droite

Phase 2
Injectat franchit le ventricule droit
Site d’injection dans l’oreillette droite

Phase 3
Injectat franchit l’artère pulmonaire
Site d’injection dans l’oreillette droite

Phase 4
Déflection maximale de la variation de température
Site d’injection dans l’oreillette droite

Phase 5
Arrivée de sang frais à l’artère pulmonaire
Site d’injection dans l’oreillette droite
Introduction of cold injectate causes a rapid upslope to a peak.
Gradual downslope, and an exponential decay of the thermal signal.
C.O. computer begins integration of area under the TD curve until the exponential decay reaches a value of about 30%.
Exponential decay to baseline extrapolated to minimize artifacts caused by recirculation of the indicator.
Thermodilution C.O.

**Normal curve**

- Temperature vs. Time
- Injection
- Smooth upstroke to peak, then gradual downslope to baseline
- Computer looks for a smooth curve

**Variation in normal curve**

- Temperature vs. Time
- Injection
- Curve extrapolated
- Respiratory variation
- Increase baseline from recirculation of injectate

**High cardiac output**

- Temperature vs. Time
- Injection
- Small area under the curve is typical of a high cardiac output (small change in injectate temperature over time)

**Low cardiac output**

- Temperature vs. Time
- Injection
- Large area under the curve seen in patients with low cardiac output (greater change in temperature over time)
Thermodilution C.O.

- Stroke volume of RV varies throughout the respiratory cycle, and measurements of RV C.O. vary as much as 50%, depending on the point during the respiratory cycle when the measurements are performed.
- Although reproducibility of consecutive measurements improves when the bolus injections are synchronized to the same phase of the respiratory cycle, true C.O. is estimated better by making numerous injections throughout all phases of the respiratory cycle and then averaging the results.
Fick Method

- C.O. = \frac{\dot{V}O_2}{CaO_2 - C\text{\text{\text{\text{\text{\text{$\bar{O}$}}}}}}_2}

\dot{V}O_2 = O_2 \text{ consumption}

units = ml O_2/minute

- Requires collection of exhaled gas
- Hb, Sao_2, PaO_2, S\text{\text{\text{\text{\text{\text{$\bar{O}$}}}}}}_2, P\text{\text{\text{\text{\text{\text{$\bar{O}$}}}}}}_2
- 3.5 ml O_2/kg/min
- 70 kg adult has \dot{V}O_2 of 250 ml O_2/min
$S\bar{V}O_2$

- Mixed-venous $O_2$ saturation can help assess tissue oxygen delivery.

By using the derivation of the Fick equation, the mixed-venous $O_2$ saturation can be determined by:

- $S\bar{V}O_2 = SaO_2 - \left[ \frac{(\dot{V}O_2)}{(Hb \times 1.36 \times \dot{Q})} \right]$

$S\bar{V}O_2$: mixed venous saturation
$SaO_2$: arterial oxygen saturation
$\dot{V}O_2$: oxygen consumption
$Hb$: hemoglobin
http://www.slideshare.net/pbsherren/cardiac-output-monitoring?related=1


http://www.modernmedicine.com/modern-medicine/content/pa-catheter-refresher-course?page=full