Hemodynamics Review

CCRN Review
October 2013
Department of Critical Care Nursing

Hemodynamics defined

- Hemo = blood
- Dynamic = changing, moving
- FLOW OF BLOOD

Invasive Hemodynamics

- What does invasive monitoring provide that cannot be obtained from clinical assessment?
  - Cardiovascular pressures
  - Blood flow
  - Evaluation of oxygen delivery and utilization

Non-Invasive Hemodynamics

- skin temperature and color
- capillary refill
- pulses
- blood pressure
- heart rate
- jugular venous distention (JVD)
- urine output

Cardiovascular System

In Cardiac Cycle

Cardiac Cycle
**Systole**
- Follows QRS of ECG
- Ventricular ejection
- Aortic and pulmonic valves open
- Mitral and tricuspid valves closed
- Majority of oxygen consumption

**Diastole**
- Follows T wave on ECG
- Ventricular filling
- Aortic and pulmonic valves closed
- Mitral and tricuspid valves open

**PAC Location**

**Indications-assessment & monitoring**
- Assessment of:
  - Cardiovascular function and response to therapy
  - Pulmonary status and response to therapy
  - Fluid requirements
- Monitoring of:
  - Surgical patients with major organ dysfunction undergoing extensive operative procedures
  - Patients in shock

**Pulmonary Artery Catheter**

**Complications of PAC during insertion**
- Arterial puncture
- Ventricular dysrhythmias
- Pneumothorax/ hemothorax
- Air embolism
- Catheter kinking/ knotting
Complications of PAC during maintenance
- Pulmonary artery rupture
- Pulmonary infarction
- Infection
- Ventricular dysrythmias

Limitations to pressure monitoring
- Assumes that filling pressures reflect ventricular end-diastolic volumes
- Assumes constant ventricular compliance
- Patient factors
  - Elevated intra-thoracic pressure
  - Elevated intra-abdominal pressure
  - Mitral valve disease
  - Incorrect catheter placement

PAC Parameters and Hemodynamic Factors

Right Heart
- Preload: RAP (CVP)
- Afterload: PVR

Left Heart
- Preload: PAWP
- Afterload: SVR

Contractility
- Cardiac output and index
- Stroke volume and index
- Ventricular work and index

Respiratory Variation
- Changes in intrathoracic pressure affect hemodynamic waveforms
- More pronounced variation
  - Stiff chest wall
  - High PEEP
- Always measure parameters at end expiration

End Expiration
- Valleys for ventilators (mechanical breaths)
  - ventilator uses pressure to push air in during inspiration, expiration is passive
- Peaks for people (spontaneous breaths)
  - intrathoracic pressure decreases during inspiration, increases during expiration
**PA catheter-CCOmboV**

- Oximetric with continuous cardiac output and right ventricular volumetrics
- Allows assessment of preload with volume based data

**Benefits of CCOmboV**

- Improved and continuous evaluation of preload
- Improved prediction of patient response to preload augmentation
- Better able to optimize preload to get the best CO/CI

**Volume Calculations**

- Calculated from CO and RVEF
- All are computer calculated numbers

**Hemodynamic Parameters**

- Allows measurement of
  - Continuous cardiac output (CCO)
  - Right ventricular ejection fraction (RVEF)

- Used to calculate
  - Right heart volumes
    - stroke volume (SV)
    - end-diastolic volume (RVEDV)
    - end-systolic volume (RVESV)
Data from PAC

**Measured Parameters**
- RAP
- PAP
- PAOP
- CO
- SVO₂

**Calculated Parameters**
- SV
- SVI
- SVR
- PVR
- DO₂

**Preload**
- Amount of myocardial fiber stretch
- Ventricular filling at end diastole
  - End diastolic volume
- Venous return, blood volume

**Preload effects on CO**
- Starling’s law
  - increased end diastolic volume increases myocardial fiber stretch and thus increases contractility
  - up to a physiologic limit
- Increase in preload increases CO
- Decrease in preload decreases CO

**Cardiac Output**

\[
\text{Cardiac Output} = \text{Heart Rate} \times \text{Stroke Volume}
\]

Preload

- Changes that significantly affect CO
  - Highly individual patient to patient
  - Below 50
  - Above 150
  - Atrial fib/flutter
  - loss of atrial contribution

**Heart Rate**

- Changes that significantly affect CO
  - Highly individual patient to patient
  - Below 50
  - Above 150
  - Atrial fib/flutter
  - loss of atrial contribution
Preload Assessment

- Right side of heart
  - Right atrial pressure (RAP) 2-6 mm Hg
- Left side of heart
  - Pulmonary artery occlusion pressure (PAOP) 6-12 mm Hg
  - Pulmonary artery diastolic pressure (PAD) 8-15 mm Hg

Preload affected by

- Intravascular volume
- Venous tone (dilation/constriction)
- Ventricular function
- Intrathoracic pressure

Preload Treatment

- Goal of preload treatment is to increase ventricular end diastolic blood volume
- Expected hemodynamic changes:
  - Decreased heart rate
  - Increased RAP
  - Increased PAOP
  - Increased SV

Preload Treatment

- Low Preload
  - Replace fluid
    - crystalloids
    - colloids
    - blood
    - hespan
    - plasmanate
  - Increase venous tone
    - vasoconstrictors

- High Preload
  - Remove fluid from intravascular space
    - diuretics
    - dialysis
  - Decrease venous tone
    - vasodilators

What is PAWP?

- Use PAWP to estimate left ventricular pressure or preload to the left side of the heart

How does PAWP reflect left preload?

- The pressure pushing back into the pulmonary veins, comes from the left atrium. Therefore, a catheter in the right side of the heart, can measure pressures in the left side of the heart.
- If the PAWP accurately reflects the left atrial pressure, then we can actually measure the pressure in the left ventricle when the mitral valve is open.
**How does PAWP reflect left preload?**
- Mitral valve is open during diastole
- The pressure the PAWP is measuring is actually the left ventricular pressure during diastole (LVEDP)
- This pressure gives us preload for the left ventricle, (the amount of stretch the left ventricle has just before it contracts)

**When is PAWP not accurate?**
- Pulmonary hypertension
- Pulmonary embolism
- Conditions that specifically alter blood flow from the right side of the heart to the left side
  - Mitral stenosis
  - Mitral regurgitation
  - Cardiac tumors

**Afterload effects on CO**
- Increased afterload decreases stroke volume thus decreasing CO
- Increased afterload causes increased myocardial oxygen consumption
- Decreased afterload increases stroke volume thus increasing CO

**Afterload Assessment**
- Calculated, not directly measured
- Pulmonary vascular resistance (PVR) for right side of heart
  - \( \frac{MPAP - PCWP \times 80}{CO} \)
- <250 dynes/sec/cm³
- Systemic vasculature resistance (SVR) for left side of heart
  - \( \frac{MAP - CVP \times 80}{CO} \)
- 800-1200 dynes/sec/cm³

**Afterload**
- Tension developed by the myocardial muscle fibers during ventricular systolic ejection
- Resistance ventricle must overcome to eject blood

**Afterload affected by**
- Arteriolar tone (constriction/dilatation)
- Arterial pressure
- Viscosity of blood
- Obstruction to flow
Afterload Treatment

<table>
<thead>
<tr>
<th>Low Afterload</th>
<th>High Afterload</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasopressors</td>
<td>Vasodilators</td>
</tr>
<tr>
<td>- Epinephrine</td>
<td>- NTG</td>
</tr>
<tr>
<td>- Norepinephrine</td>
<td>- Sodium Nitroprusside</td>
</tr>
<tr>
<td>- Phenylephrine</td>
<td>- IABP</td>
</tr>
<tr>
<td>- Dopamine</td>
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</tbody>
</table>

Goal of afterload treatment is to decrease cardiac workload by decreasing resistance that the ventricles must overcome.

Expected hemodynamic changes:
- Decreased SVR
- Decreased PVR
- Increased CO/CI
- Increased SV
- Decreased blood pressure

Assessment of contractility

- Stroke volume
- Ejection fraction
- Echocardiography
- Ventricular stroke work

Cardiac Contractility

- Inherent ability of the cardiac fibers to shorten
- Cannot directly measure
- All calculations include determinants of preload and afterload

RVEF (Right Ventricle Ejection Fraction)

- Percentage of total volume of blood in the chamber that is ejected from the ventricle with each beat
- Indicates contractility
- 40-60%

Stroke Volume (SV)

- Volume of blood ejected from the ventricle with each heartbeat
  \[ SV = \text{CO/HR} \times 1000 \]
- Normal: 60-100 ml/beat
-Normally, Right and left ventricles eject the same amount of blood
**Right Ventricular End Diastolic Volume (RVEDV)**
- Volume of blood in the right ventricle at the end of diastole
- Indicates RV preload
- EDV = SV/EF
- Normal: 100-160 ml/beat

**Contractility Treatment**
- Goal is to improve ventricular performance
- Expected hemodynamic changes:
  - Increased CO/CI
  - Increased SV
  - Improved tissue perfusion

**Right Ventricular End Systolic Volume (RVESV)**
- Volume of blood remaining in the ventricle at the end of the ejection phase of systole
- ESV will increase if ventricle meets more resistance (afterload) or if there is impaired contractility
- Normal: 50-100 ml/beat

**Hemodynamic Interventions**

<table>
<thead>
<tr>
<th>Low</th>
<th>High</th>
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</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Beta blockers</td>
</tr>
<tr>
<td>Pacemaker</td>
<td>Ca++ blockers</td>
</tr>
<tr>
<td>Fluids</td>
<td>Diuretics</td>
</tr>
<tr>
<td>Preload</td>
<td>Venodilators</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>Afterload</td>
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<tr>
<td>Afterload</td>
<td>Arterial dilators</td>
</tr>
<tr>
<td>Positive inotropes</td>
<td>Ca++ blockers</td>
</tr>
<tr>
<td>Contractility</td>
<td>ACE inhibitors</td>
</tr>
<tr>
<td>Negative inotropes</td>
<td>Contractility</td>
</tr>
</tbody>
</table>

**Cardiac Output = 4-8 LPM**
**Cardiac Index = 2.5-4 L/min/m2**

**Increased CO**
- sepsis
- exercise
- vasodilation

**Decreased CO**
- MI
- cardiac tamponade
- hypovolemia
- LV failure
- CHF

**Typical Hemodynamic Profiles**

<table>
<thead>
<tr>
<th>Condition</th>
<th>HR</th>
<th>MAP</th>
<th>CO/CI</th>
<th>CVP/RA</th>
<th>PA/PAP</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic Shock</td>
<td>↑</td>
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<tr>
<td>Cardiogenic Shock</td>
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<tr>
<td>Septic Shock</td>
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</tr>
<tr>
<td>Cardiac tamponade</td>
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<td>↓</td>
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<td>↑</td>
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<td>↑</td>
</tr>
<tr>
<td>Right Ventricular Failure</td>
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<tr>
<td>Mesenteric Perfusion</td>
<td>↑</td>
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<td>↑,↑</td>
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<tr>
<td>Aortic Mitral Valve</td>
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<td>↑,↑</td>
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</table>
The goal of resuscitation is to optimize regional organ perfusion and cellular oxygen utilization.

Role of IV Fluids
- Start vasoactive medications only after intravascular volume has been restored
- Can use crystalloids or colloids to restore intravascular volume

Role of IV Fluids-Crystalloids
- Isotonic fluids
  - A solution whose concentrations are matched physiologically to the part of the body in which it will be infused
  - Low risk of allergic reaction
  - Low cost
  - Examples:
    - 0.9%NaCl solution
    - Lactated Ringer’s solution
  - Only 25% remains in the vascular space after 1 hour
**Sodium chloride-0.9%**

- 1 liter of 0.9% NaCl contains:
  - 154 mEq/L sodium
  - 154 mEq/L chloride
  - Calculated osmolarity of 308 mOsm/L
  - pH of 4.5-7

**Lactated Ringers solution**

- Liter of Lactated Ringers contains:
  - 9 calories
  - 130 mEq of sodium
  - 4 mEq of potassium
  - 3 mEq of calcium
  - 109 mEq of chloride
  - 28 mEq of lactate
  - pH is approximately 6.6 (range 6.0-7.5)
  - Calculated osmolarity of 273 mOsm/L

**Vasopressors and inotropes**

**Definitions**

- Vasoactive: agents that cause constriction or dilation of blood vessels leading to a change in blood pressure
- Vasopressors: agents that cause constriction of blood vessels leading to a change in blood pressure
- Vasodilators: agents that cause dilation of blood vessels leading to a change in blood pressure
- Inotropes: agents that influence the force of muscular contractility

**Role of IV Fluids- Colloids**

- Albumin
  - 5%
  - 25%
- PRBC’s
  - As indicated for low hemoglobin states
- FFP, platelets, and cryoprecipitate
  - As indicated for coagulopathy

**Indications for Vasoactive Meds**

- Inotropes and vasopressors are used when there is evidence of inadequate perfusion of vital tissues.
- These drugs may be indicated to either restore overall cardiovascular function or to improve perfusion.
Indications for Vasoactive Meds

- Start vasoactive medications only after intravascular volume has been restored.
- Vasopressors and inotropes are required when volume resuscitation fails to restore tissue perfusion.

Adrenoceptors

- Adrenoceptors are classified as either alpha or beta adrenoceptors
- The physiological response to adrenoceptor stimulation depends on the location of the receptor

Nursing assessment of inadequate perfusion

- What does this look like?
  - Integumentary
  - Renal
  - Hemodynamics
    - MAP/BP
    - Heart rate
    - CVP
    - CI/CO
    - Laboratory values
      - Lactate
      - ScvO2/SvO2

Hemodynamic Effects

- Vasoactive medications act through their interactions with receptors in the heart and vascular system
- The most relevant receptors are the adrenoceptors

Alpha Receptors

- Divided into two main types—Alpha 1 (\(\alpha_1\)) and Alpha 2 (\(\alpha_2\))
- Primary location of \(\alpha_1\) is the arteries, arterioles, and veins
- Primary location of \(\alpha_2\) is the GI tract

- \(\alpha_1\) receptors constrict the vascular system when stimulated
- \(\alpha_2\) receptors decrease tone, decrease motility, and decrease the secretions of the GI tract when stimulated
**Alpha Receptors**

- Medications that stimulate $\alpha_1$ and $\alpha_2$
  - Phenylephrine at all doses
  - Norepinephrine at all doses
  - Epinephrine at doses >0.15mcg/kg/min
  - Dopamine at doses >10mcg/kg/min

**Intended Actions**

- $\alpha_1$ actions
  - Increase systolic and diastolic blood pressure
  - Increase mean arterial pressure
  - Increase systemic vascular resistance
  - Increase cerebral blood flow
- $\alpha_2$
  - Decreases tone, motility, and secretions in the GI tract

**Unintended Actions**

- Decrease peripheral blood flow
- Cold fingers and toes
- Ileus due to decrease GI motility
- Extravasation can lead to tissue necrosis
- Tachycardia
- Hyperglycemia
- Ventricular arrhythmias

**Beta Receptors**

- Divided into two main types—Beta 1 ($\beta_1$) and Beta 2 ($\beta_2$)
- Primary location of $\beta_1$ receptors is the heart
- Primary location of $\beta_2$ receptors is the blood vessels of the skeletal muscle, the bronchial smooth muscle, and the coronary arteries

**Intended Actions**

- $\beta_1$ receptors increase heart rate and force of contraction when stimulated
- $\beta_2$ receptors
  - Dilate the blood vessels of skeletal muscle
  - Dilate the coronary arteries
  - Relax the bronchial smooth muscle

**Unintended Actions**

- Drugs that stimulate $\beta_1$ and $\beta_2$
  - Norepinephrine at all doses
  - Epinephrine at doses <0.15mcg/kg/min
  - Dopamine at doses <10mcg/kg/min
  - Dobutamine at all doses
Intended Actions

- $\beta_1$
  - Increased heart rate
  - Increased cardiac output
  - Increased cardiac index
  - Increased systolic blood pressure
  - Increased stroke volume
  - Increased myocardial oxygen consumption
  - Increased $\text{DO}_2$

- $\beta_2$
  - Dilation of coronary arteries
  - Relaxation of bronchial smooth muscle
  - Decreased vascular tone
  - Decreased systemic vascular resistance
  - Decreased diastolic blood pressure

Unintended Actions

- Tachycardia
- Ventricular arrhythmias
- Hypotension
- Extravasation can lead to tissue necrosis

Vasopressin Receptors

- Vasopressin 1 receptors are located in arterial smooth muscle
- Vasopressin 2 receptors are located in the renal tubules

Vasopressin Receptor Stimulators

- Vasopressin = ADH
  - Inhibits diuresis in patients with Diabetes Insipidus
  - Regulates water balance at lower doses
  - Causes vasoconstriction at higher doses

Intended Actions

- $V_1$ receptors
  - Constriction of arterial smooth muscle
  - Increased MAP / BP
  - Increased SVR
  - Increased cerebral oxygenation

- $V_2$ receptors
  - Constriction of renal tubules
  - Decreased renal blood flow
  - Decreased glomerular filtration rate
  - Stimulation of water resorption causing urine concentration

Unintended Actions

- Decreased CO / CI
- Increased cortisol
- Increased platelet aggregation
- Extravasation can lead to tissue necrosis
Vasopressin
- Vasopressin enhances the sensitivity of the vasculature to other vasopressor agents
- Works best at a pH ≤ 7.20
- ACLS guidelines for cardiac arrest
  - one 40 unit IV push dose to replace first or second dose of Epinephrine

Titration & Weaning
- There is no formula
- Per ordered parameters
- Each drug works differently
- Must know the effects of the drug and the response you are attempting to achieve
- Obtain vital signs including PAC values prior to and after adjusting vasopressors or inotropes
- May need to change concentration of medication

Other Continuous Infusions

Vasodilators
- Sodium Nitroprusside
  - More effective arterial dilator than NTG
  - Used to reduce high afterload
- Nitroglycerin
  - More effective venodilator than SNP
  - Used to reduce high preload

Oxygen Utilization
The ultimate goal of critical care is to ensure adequate oxygen for cellular use throughout the body
**Shock**
- Defined as a state of inadequate tissue perfusion resulting in inadequate oxygen delivery and/or inadequate oxygen utilization by the cells
- Of accomplish your goal, the RN must balance oxygen delivery and consumption

**Steps in the process of oxygenation-cell**
- Oxygen is diffused across the capillary membrane
- Oxygen is transported through the cell for use by the mitochondria

**Steps in the process of oxygenation-lungs**
- Oxygen is taken into the lungs
- Oxygen is diffused across the alveolar-capillary membrane

**Arterial Oxygen Content**
- **SaO₂ vs PaO₂**
  - Oxygen is carried in two forms in the blood
    - SaO₂ is oxygen attached to hemoglobin
      - 98%
    - PaO₂ is oxygen dissolved in the plasma
      - 2%

**Steps in the process of oxygenation-blood**
- Oxygen is dissolved in the blood
- Oxygen attaches to the hemoglobin
- Oxygen is transported via RBC’s
- Oxygen is unloaded from the hemoglobin
- Oxygen is re-dissolved in the blood

**Monitoring**
- **ABG**
  - SaO₂-amount of oxygen bound to hgb. as measured on the ABG
  - PaO₂-amount of oxygen dissolved in the blood
- **Pulse oximetry**
  - SpO₂-amount of oxygen bound to hgb. as measured by pulse oximetry
Oxyhemoglobin Dissociation Curve

- Oxyhemoglobin Dissociation Curve (Bohr Effect): The Bohr effect explains how pH impacts the oxygen saturation of hemoglobin. A low pH (acidity) causes the hemoglobin to have a decreased affinity for oxygen thus giving up oxygen to the tissues more easily, and the opposite occurs when pH increases (alkalinity) with the hemoglobin giving up oxygen to the tissues less easily.

Oxygen Demand

- Amount of O2 cells need to carry out metabolism
- Should be less than O2 delivery
- Should be equal to O2 consumption
- Cannot assume that oxygen consumption meets oxygen demand

Oxygen Balance

Oxygen Delivery (DO₂)

- Amount of O2 delivered to the tissues per minute
- Fick formula
  \[ DO₂ = CO \times CaO₂ \times 10 \]
- Normal is 950-1150 ml/min

Definitions

- Oxygen demand
  - Amount of O₂ needed by cells to carry out metabolism
- Oxygen delivery
  - Amount of O₂ delivered to cells
- Oxygen consumption
  - Amount of O₂ actually used by cells

Oxygen Delivery

\[
\text{DO₂} = \text{Cardiac Output (CO)} \times \text{Arterial oxygen content (CaO₂)}
\]

\[
\text{Stoke Volume} \times \text{Heart Rate} = \frac{\text{CaO₂} \times (\text{Hgb} \times \text{SaO₂}) + (\text{PaO₂} \times 0.03)}{\text{Preload} \times \text{Afterload} \times \text{Contractility}}
\]
Factors Contributing to Oxygen Delivery

1. Cardiac Output
2. Hemoglobin
3. SaO2
4. PaO2

Factors Contributing to Oxygen Delivery

- Cardiac Output is the largest determinate of $DO_2$
- Optimize cardiac output by affecting:
  - Preload
  - Afterload
  - Contractility
  - Heart rate

Causes of Increased O2 Demand

<table>
<thead>
<tr>
<th></th>
<th>Fever (one degree C)</th>
<th>Work of Breathing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10%</td>
<td>40%</td>
</tr>
<tr>
<td>Shivering</td>
<td>50-100%</td>
<td>Dressing change</td>
</tr>
<tr>
<td>10%</td>
<td></td>
<td>10%</td>
</tr>
<tr>
<td>ET suctioning</td>
<td>7-70%</td>
<td>Bath</td>
</tr>
<tr>
<td>23%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sepsis</td>
<td>50-100%</td>
<td>Chest x-ray</td>
</tr>
<tr>
<td>25%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visitors</td>
<td>22%</td>
<td>Position change</td>
</tr>
<tr>
<td>31%</td>
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</tr>
</tbody>
</table>

Oxygen balance

- Under normal conditions, $DO_2$ exceeds $VO_2$ (“supply independent”)
- When delivery becomes compromised, $DO_2$ falls below a critical threshold
- Below the critical delivery threshold, $VO_2$ becomes dependent on $DO_2$
- Called “supply dependency”

Compensatory Response

- When there is a threat to the oxygen balance, a compensatory response occurs:
  - Increase cardiac output to increase delivery

OR

- Increase extraction

Supply dependency

- Occurs when cells cannot compensate for the decrease in $DO_2$ by increasing extraction (because there is no more oxygen available)
- Causes the cells to convert from aerobic metabolism to anaerobic metabolism = oxygen debt
Aerobic metabolism

- Via the Kreb’s Cycle, metabolism of glucose produces CO₂, water, and adequate amounts of ATP
- CO₂ is exhaled
- H₂O is added to the body’s water stores
- 38 molecules of ATP

Factors that decrease VO₂

<table>
<thead>
<tr>
<th>Factor</th>
<th>Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>anesthesia</td>
<td>25%</td>
</tr>
<tr>
<td>NMB</td>
<td>100%</td>
</tr>
<tr>
<td>A/C ventilation</td>
<td>30%</td>
</tr>
</tbody>
</table>

Anaerobic metabolism

- Metabolism of glucose produces lactic acid and less ATP
- Water and CO₂ are not produced
- 2 molecules of ATP
- Accumulation of lactic acid signals organ dysfunction or failure

What’s the Difference?

<table>
<thead>
<tr>
<th>Hypoxemia</th>
<th>Reduced amounts of oxygen in the blood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxia</td>
<td>Reduced amounts of oxygen in the body</td>
</tr>
</tbody>
</table>

Oxygen Consumption (VO₂)

- Amount of oxygen used by cells to generate energy
- Gold standard is indirect calorimetry
  - Expensive, special equipment
  - Reverse the Fick formula
    \[ VO₂ = (CaO₂ – CvO₂) \times CO \times 10 \]
- Normal is 250 ml/min

Evaluating Cellular Oxygenation (Hypoxia)
**Hypoxia**

- State of oxygen deficiency in the body
- Results in cellular oxygen deficit
- Difficult to evaluate

**Causes of Hypoxia**

- Hypoxemic Hypoxia
  - Low arterial blood O₂ content
- Anemic Hypoxia
  - Low Hgb, results in decreased O₂ carrying capacity
- Circulatory Hypoxia
  - Cardiovascular failure, low delivery of oxygen
  - Shock states
- Histotoxic Hypoxia
  - Impaired oxygen utilization by mitochondria of cell
  - Cyanide poisoning

**Clinical Assessment of Hypoxia**

- Assessment findings that may signal hypoxia
  - Brain—decreased mentation, confusion, agitation
  - Heart—chest pain
  - Kidney—decreased urine output
  - Integumentary—cool skin, dusky nailbeds
- Cannot actually measure cellular oxygenation
- Use more global methods to evaluate oxygen balance
- Compare oxygen delivery to oxygen consumption

**Laboratory values in oxygen evaluation**

- SaO₂
- PaO₂
- Lactate
- Base deficit
- SvO₂
- ScvO₂

**Serum Lactate**

- End product of anaerobic metabolism
- Normal <2 mMol/L
- Clinical gold standard as a marker of inadequate cellular oxygenation

**Base Deficit**

- Amount of base required to titrate 1 L of arterial blood to pH of 7.40
- Normal –2 to +2
- Base deficit can accumulate from accumulation of lactate associated with anaerobic metabolism
- Sensitive measure of inadequate oxygenation
**Venous Oxygen Saturation** (SvO\textsubscript{2} or ScvO\textsubscript{2})

- SvO\textsubscript{2} measured with pulmonary artery catheter
  - Measured in the pulmonary artery
  - 5-7 percentage points lower than ScvO\textsubscript{2}
- ScvO\textsubscript{2} measured with central catheter
  - Measured in the superior vena cava

**SvO\textsubscript{2}**

- Measures oxygen saturation in venous blood after cellular extraction
- Reflects changes in O\textsubscript{2} extraction
- Reflects balance between oxygen delivery and oxygen consumption

**Decreased SvO\textsubscript{2}**

- Decreased O\textsubscript{2} delivery
  - Decrease in Hgb
  - Decrease in arterial oxygen content
    - SaO\textsubscript{2}, PaO\textsubscript{2}
  - Decrease in cardiac output
- Increased O\textsubscript{2} consumption

**Increased SVO\textsubscript{2}**

- Adequate delivery
- Decreased consumption
- Signals that cells are not extracting oxygen from the blood
- Causes:
  - Shunting
  - VQ mismatch

**Significance to clinical practice**

- Used to monitor the adequacy of oxygen balance
- Use with other pieces of the assessment to determine pt's oxygenation balance
- Determine oxygen balance to determine pt's readiness for interventions:
  - Weaning of supplemental oxygen/ vent support
  - Weaning of vasoactive medications
  - Personal care/ activity

**SvO\textsubscript{2}**

- Early indicator of threat to cellular oxygenation
- Normal SvO\textsubscript{2} 60%-75%
- Significant changes
  - Outside normal range
  - +/- 10% for more than 3-5 minutes
Case Studies

- See Hemodynamic case studies