Tissue Perfusion: It really is everything!

Monitoring Tissue Perfusion in ICCU:

B. McLean, MN, RN, CCRN, CCNS, ANP-BC, FCCM

Hemodynamic Monitoring Truth

• No monitoring device, no matter how simple or complex, invasive or non-invasive, inaccurate or precise will improve outcome
• Unless coupled to a treatment, which itself improves outcome


Goals For Monitoring

• To assure the adequacy of perfusion
• Early detection of inadequacy of perfusion
• To titrate therapy to specific hemodynamic end point
• To differentiate among various organ system dysfunctions

Hemodynamic monitoring for individual patient should be physiologically based and goal oriented.
Hemodynamic monitors

- Traditional invasive monitors: Dynamic pressures and oxygenation
  - Arterial line
  - CVP & ScvO2
  - PA catheter, CCO, SvO2
- Functional pressure variation
- Affected pressures and oxygenation
  - Pulse pressure variation
  - Stroke volume variation
  - Tissue oxygenation

Goals for cardiocirculatory therapy

- ScvO2 >70% or SvO2 >65%
- MAP (mean arterial pressure) >65 mmHg
- Cardiac Index >2.0 l/min/m2
- CVP 8–15 mmHg (dependent on ventilation mode)
- SVV < 13 %
- PAOP 12–15 mmHg
- Diuresis >0.5 ml/kgBW/h
- Lactate <3 mmol/l

Surrogate Monitoring

- All left sided measures
  - PAP
  - PaOP
  - CVP
  - B/P
  - RVEDVI/LVEDVI
  - RVSWI/LVSWI

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But it really is about the microcirculation

But it really is about the microcirculation

Multi Organ Failure

Functional Monitoring

• Will cardiac output increase when volume given?
  – How much?
• State of vascular tone?
  – Increased
  – Decreased
  – Normal
• Can the heart maintain cardiac output under pressure without failure?


Review of physiology
Tissue Perfusion: It really is everything!

Gradient of pressure
Gas Distribution

A>a>v Zone 1
+ 10

A>v >A Zone 3

Why are we monitoring?

- Preload, contractility, afterload, and oxygen transport are commonly abnormal in the critically ill
- Inadequate resuscitation and failure to restore cellular oxygen delivery and organ perfusion results in multiple system organ dysfunction syndrome (MODS) and death
- Optimization of critical illness reduces organ failure and improves survival
- Inadequate resuscitation and failure to restore cellular oxygen delivery and organ perfusion results in multiple system organ dysfunction syndrome (MODS) and death
- Accurate assessment of hemodynamic function and goal-directed resuscitation is essential to improving patient outcome

Increase venous pressure (preload) leads to a rise in stroke volume and therefore cardiac output

- ↑ End diastolic volume causes ↑ stroke volume
- "The energy of contraction of a cardiac muscle fiber, like that of a skeletal muscle fiber, is proportional to the initial fiber length at rest."
- Stroke volume increase due to increased force of contraction
- Frank-Starling mechanism or Starling’s Law of the Heart
What does dynamic monitoring offer to me?

Remember…….

A searchlight cannot be used effectively without a fairly thorough knowledge of the territory to be searched.

Fergus Macartney, FRCP

Volume Responsiveness in Critically Ill patients

- PPV causes changes in venous return, which is accentuated in hypovolemic patients
- take advantage of the swings in venous return in order to determine the fluid responsiveness of hypotensive patients
- 2 major tools to look at this:
  - Echo
  - Arterial Line – looking at changes in the pulse contour, and in the pulse pressure
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The Perfect Volume Status Monitor

- Fast
- Easy to learn
- Validated in all critically ill patients. (medical, surgical, trauma, neurosurgical on positive pressure ventilation)
- Available outside of the ICU, not require any highly specialized equipment
- Give an easy answer
- If you do not have a variation, do not give volume

Dynamic Methods to look at Hemodynamics and Volume Status in the MICU

- Take advantage of the Heart-Lung interactions during positive pressure ventilation.
- Arterial Line Monitoring with dynamic analysis of the waveform and pulse pressure variability
- Echocardiography to predict volume responsiveness (not going to be covered in this 20 minute talk!)
  - LV, IVC, SVC

SVV physiology

[Diagram showing Stroke Volume vs Normal heart, Failing heart, Preload vs Independent, Ventricular preload]

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Fluid Responsiveness

- A patient who is fluid responsive will have a significant (>15%) increase in CO in response to a fluid challenge.
- This indicates that the heart is on the steep portion of the Frank-Starling Curve

Cardiac Output Optimisation

Cardiac Output Maximisation

Current state of monitoring
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Swan-Ganz catheter?
The use of pulmonary artery catheters probably needs re-evaluation—but they should not be banned

Trends in the Use of the Pulmonary Artery Catheter in the United States, 1993-2004

Surrogate Monitoring: Static

- All left sided measures
  - PAP
  - PaOP
  - CVP
  - B/P
  - RVEDVI/LVEDVI
  - RVSWI/LVSWI

Meta-analysis of hemodynamic optimization in high-risk patients

Effects of maximizing oxygen delivery on morbidity and mortality in high-risk surgical patients

Surrogate Monitoring: Static

- All left sided measures
  - PAP
  - PaOP
  - CVP
  - B/P
  - RVEDVI/LVEDVI
  - RVSWI/LVSWI

Hemodynamic monitors

- Traditional invasive monitors: Static Measures
  - Arterial line
  - CVP & ScvO2
  - PA catheter, CCO, SvO2
- Functional variation: Dynamic measures
  - Pulse pressure variation
  - Stroke volume variation

Functional Monitoring

- Will cardiac output increase when volume given?
  - How much?
- State of vascular tone?
  - Increased
  - Decreased
  - Normal
- Can the heart maintain cardiac output under pressure without failure?


- Static indicators have been shown to be poor predictors of fluid responsiveness
  - central venous pressure (CVP)
  - pulmonary capillary wedge pressure (PCWP)
  - left ventricular end diastolic area
- Dynamic indicators demonstrated to be better predictors of fluid responsiveness in patients during mechanical ventilation.
- During positive pressure ventilation, the inspiratory right ventricular stroke volume (SV) decrease is proportional to the degree of hypovolemia and is transmitted to the left heart after two or three beats (pulmonary transit time)

Michard F. Anesthesiology 2005;103:419–28,
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Neither CVP or Ppao reflect Ventricular Volumes or tract preload-responsiveness

Kumar et al. Crit Care Med 32:691-9, 2004


• studies have shown that the CVP often does not accurately reflect end-diastolic volume and right ventricular preload.
• Dynamic responses to volume challenge by using either stroke volume variation or pulse pressure variation are both highly sensitive and specific for preload responsiveness in mechanical ventilated patients, whereas the
• passive straight leg test should be used in
• spontaneously breathing patients
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The influence of volume management on outcome
Sean M. Bagshaw* and Rinaldo Bellomo

- Emerging data show that the choice, timing and amount of fluid therapy may affect clinical outcomes
- Early administration of fluid therapy in sepsis may improve survival
- Later fluid therapy in acute lung injury patients will increase the duration of ventilator dependence without achieving better survival

Now what?

Main circumstances in ICU

- Positive pressure ventilation
- Severe pulmonary embolism
- ARDS
- Sepsis induced RV dysfunction
- Exacerbation of medical conditions leading to chronic pulmonary hypertension
- Right ventricle infarction
- Pericardial diseases
- RV failure after cardiac surgery
- After cardiac transplant
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Physiology of the normal pulmonary circulation

- Low pressure system: $P_{RV}$ (syst) = 25 mmHg versus $P_{LV}$ (syst) = 120 mmHg
- The pressure in the pulmonary system depends on cardiac output, resistance and compliance
  - Normally very compliant pulmonary vessels with large diameter and thin wall
  - Normal RV afterload very low
- Alveolar hypoxia leads to pulmonary arterial vasoconstriction and $\uparrow$ pulmonary vascular resistance

Effects of mechanical ventilation

- Increased RV afterload due to positive pressure ventilation
- Hemodynamic failure frequently refractory in PAH patient put on MV
- In ARDS increase in mPAP while increasing tidal volume and PEEP
- Permissive hypercapnia is deleterious (increase in mPAP)
Effect of MV on venous return

Effect of high PEEP on RV

O₂ requirements and blood supply to the RV

- Less O₂ requirements than the LV: ↓ myocardial mass due to ↓ afterload
- Vascularisation: 2/3 RCA, 1/3 left branches
- RV perfused in both systolic and diastolic phases
  - Low systolic pressure (25 mmHg) does not compress vessel
Vicious cycle of auto-aggravation

<table>
<thead>
<tr>
<th>RV pressure overload</th>
<th>Reduced cardiac output</th>
<th>Systemic hypotension</th>
</tr>
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<tr>
<td></td>
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<td>Reduced RV tissue perfusion</td>
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<td>RV free wall ischemia</td>
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<td>Reduced RV free wall contractility</td>
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</table>

Ventricular interdependence

- During systole, LV protrudes in RV
- Surrounding pericardium with limited distensibility
- Compliance of one ventricle can modify the other = Diastolic ventricular interaction

Variation from Ventilation

- SVV, PPV, and SPV are created by tidal volume-induced changes in venous return.
- Presumes a constant R-R interval and are measured from diastole to systole, not vice versa (reflect only changes in venous return and not diastolic filling time)
- Lose their predictive value under conditions of
  - Varying R-R intervals (atrial fibrillation),
  - Tidal volume varies from breath to breath (with assisted and spontaneous ventilation)
**Tissue Perfusion: It really is everything!**

**Monitoring of right-sided heart function.**
Jardin, Francois; Vieillard-Baron, Antoine

![Figure 1.](image1.png)

- **PAD-PAoP gradient > 5 mm Hg**

**Use of Heart Lung Interactions to Diagnose Preload-Responsiveness**

- **Valsalva maneuver**
- **Ventilation-induced changes in:**
  - Right atrial pressure
  - Systolic arterial pressure
  - Arterial pulse pressure
  - Inferior vena caval diameter
  - Superior vena caval diameter

Zema et al., D Chest 85,59-64, 1984

**Key points**

- PAC remains used for the management of patients with right ventricular failure, pulmonary hypertension and weaning failure from cardiac origin.
- PAC can be helpful for the management of complex circulatory conditions in which the knowledge of PAP, PAOP and oxygenation parameters is considered to be particularly important.
- Alternative methods of hemodynamic monitoring which allow intermittent or continuous CO measurements are being evaluated and compared to the single bolus pulmonary THD measurement using the PAC.
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Use of Heart Lung Interactions to Diagnose Preload-Responsiveness

- ValSalva maneuver
- Ventilation-induced changes in:
  - Right atrial pressure
  - Systolic arterial pressure
  - Arterial pulse pressure
  - Inferior vena caval diameter
  - Superior vena caval diameter

Zerwa et al.,; Crit Care Med 85:59-64, 1984
Vieillard-Baron et al. Anesthesiology 96:673-4, 2004
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**Frank-Starling relationship**

- **Stroke Volume**

  - **Preload**

  - **Normal**
  - **Heart Failure**

**Fluid Responsiveness**

- % increase in stroke volume after volume expansion.
  - Responders ≥ 15%
  - Non Responders < 15%

**The Goal is to Improve Oxygen Delivery**

**Respiratory Variations in LV Stroke Volume**

- Airway pressure up
- Ventricular compliance and volume load down
- Pulmonary Vascular transit Time

- Venous Return
- RVEDV
- RSVV
- LVEDV
- LVSV

Static Parameters vs. Dynamic Parameters
"The more sensitive a ventricle is to preload, the more the stroke volume will be impacted by changes in preload due to positive pressure ventilation."
Variation from Ventilation

- SVV, PPV, and SPV are created by tidal volume-induced changes in venous return.
- Presumes a constant R-R interval and are measured from diastole to systole.
- Pressure ventilation causes changes in venous return, which is accentuated in hypovolemic patients.
- Take advantage of the swings in venous return in order to determine the fluid responsiveness of hypotensive patients.

SVV physiology

In a normal individual who is breathing spontaneously, blood pressure decreases on inspiration. The exaggeration of this phenomenon is called pulsus paradoxus.

Normal Variation & Pulsus Paradoxus
Reversed Pulsus Paradoxus

A phenomenon that is the reverse of the conventional pulsus paradoxus has been reported during positive pressure breathing.

F. Michard

Pulse Pressure Variation

- Pulse pressure variation (PPV)
  - Calculated in the same manner as SVV.
  - Also predict preload responsiveness well.
- A 13% PPV predicts a 15% increase in CO for a 500-mL volume bolus
- ANY signal that gives pulse density
- REQUIRED
  - Controlled variables
    • Ventilation
    • Heart rate
What are the Limitations of SVV?

- **Mechanical Ventilation**
  - Currently, literature supports the use of SVV on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8cc/kg and fixed respiratory rates.
- **Spontaneous Ventilation**
  - Currently, literature does not support the use of SVV with patients who are spontaneously breathing.
- **Arrhythmias**
  - Arrhythmias can dramatically affect SVV. Thus, SVV’s utility as a guide for volume resuscitation is greatest in absence of arrhythmias.

What are the Limitations of SVV?

- **Arrhythmias**
  - Arrhythmias can dramatically affect SVV. SVV utility as a guide for volume resuscitation is greatest in absence of arrhythmias.
- **varying R-R intervals (atrial fibrillation),**
- **aortic insufficiency**
- **Peripheral vascular disease**
- **intra-aortic counterpulsation**
What am I looking for?

- Indices of hypovolemia: SVV > 13%
- Volume loading should decrease SVV. If not
  - Stop fluid administration
  - Inotropic support initiated

Are the pulse analysis techniques as accurate as the PAC for monitoring CO?

- Yes, (level of evidence, C)
- But it depends....
  - Not all monitors are the same.
  - In stable patients they perform to a clinically acceptable level and have other advantages.
    - Continuous data
    - Less invasive
    - Offer other variables.
  - In shocked patients the evidence is less clear.

Rhodes, personal communication ESICM 2010
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What is the arterial tone?

Hypotension

- Relationship between PPV/SVV
- Better relationship of elastance /vascular tone than SVR
- No assumption regarding volume distribution
- Physics calculation
  - PP reflects variance
  - SV more regarding EF
- PP/SV normal 1.2-2

What is the arterial tone?

Hypotension, volume responsiveness

- PP/SV normal 1.2-1.5
  - < 0.9 indicates vasoconstriction, SVV > 13%
    - Volume
  - < 0.9 indicates vasoconstriction, SVV<13%
    - Inotrope /vasodilator
    - Volume
  - > 1.5 indicates vasodilation, SVV>13%
    - Volume
    - Vasopressor

Physiological Truth

- There is no such thing as a "Normal Cardiac Output"
- Cardiac output is either
  - Adequate to meet the metabolic demands
  - Inadequate to meet metabolic demands
- Absolute values can only be used as minimal levels below which some tissue beds are under perfused
Tissue Perfusion: It really is everything!

Emerging trends in minimally invasive haemodynamic monitoring and optimization of fluid therapy.

Benington S; Ferris P; Nirmalan M

Are the pulse analysis techniques as accurate as the PAC for monitoring CO?

• Yes, (level of evidence, C)
• but it depends….
  – Not all monitors are the same.
  – In stable patients they perform to a clinically acceptable level and have other advantages.
    • Continuous data
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  – In shocked patients the evidence is less clear.

A. Rhodes, personal communication ESICM 2010

And to validate or measure for those not on ventilation?
Passive Leg Raising Maneuver

- PLR method:
  - Raising the legs 30° above the chest for 1 min
  - About 300-mL blood bolus in a 70-kg man persisting for about 2 to 3 min
  - Transient and reversible in venous return
  - Accurately predict preload responsiveness
  - Both spontaneous and positive pressure ventilation
  - With or without arrhythmias
  - Can be applied in all hemodynamically unstable patients

PASSIVE LEG RAISE TEST (PLRT)

150–300 mL volume
Effects < 30 sec., Not more than 4 minutes
Self-volume challenge
Reversible

Clinical investigations in critical care

Passive leg raising predicts fluid responsiveness in the critically ill

Diagnosis of central hypovolemia by using passive leg raising
Problem: Enough Volume?

Volume expansion is a first line therapy for patients with circulatory failure. However, in only half of such patients is cardiac output increased after a fluid challenge and thus only those can be considered as responders to fluid therapy. Therefore, physicians need reliable criteria to distinguish responders from non-responders. For those patients whose fluid therapy can even be harmful.

• Volume expansion 1st line of therapy.
• Only ½ of patients respond to fluids with increased CO.
• Need a reliable means to determine ability to respond to fluid.

Physiological Truth

• There is no such thing as a "Normal Cardiac Output"
• Cardiac output is either
  – Adequate to meet the metabolic demand
  – Inadequate to meet metabolic demands
• Absolute values can only be used as minimal levels below which some tissue beds are under perfused

So what about lactate?
Lactate Levels

• The other acid: Lactate Levels
  • Lactic acid is a product of carbohydrate metabolism.
  – It is normal to produce 15 to 20 mmol/kg of lactic acid per day.
  – The normal plasma level is 0.5 to 1.5 meq/L.
  • Hyperlactatemia is considered to be present if the level exceeds 4 to 5 meq/L.
  • Lactic acidosis is considered to be present if the elevated lactate level is in conjunction with a gap >20 in the absence of elevated glucose levels..
  – Lactic acid is rapidly buffered by extracellular bicarbonate resulting in lactate.
  – The liver and kidneys convert lactate back to pyruvate which is then converted to CO2 & H2O or glucose.

Lactate Levels

• Is serum Lactate a good marker of adequacy of perfusion?
  – Type A lactic acidosis primarily results from an imbalance between tissue oxygen demand, delivery and use.
  – The blood lactate level in type A lactic acidosis is related to the total oxygen debt and the magnitude of tissue hypoperfusion.
• Elevated blood lactate levels associated with metabolic acidosis are common among critically ill patients with systemic hypoperfusion, tissue hypoxia and metabolic dysfunction.
  – Blood lactate levels also increase with clearance failure, i.e., kidney or liver dysfunction

Lactate Levels

• Utility of a single high initial lactate have been debated
  – poor sensitivity and specificity.
• Lactate clearance is a better predictor of mortality
  – Lac-time: time it takes to clear 10% of lactate
  – Time to clear < 24 hours, improves survival in Severe sepsis
  – Lac-time also directly correlated with number of organ failures
• One lactate (lactic acid) level is not as predictive or evaluative as a series over 24 hours (i.e., Q6H)

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Lactic Acid Check List

<table>
<thead>
<tr>
<th>Lactic Acidosis</th>
<th>Postive</th>
<th>Metabolic Dysfunction</th>
<th>Regional Hypoperfusion</th>
<th>Hypoxic Enhanced Lactic Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver Dysfunction</td>
<td></td>
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<td></td>
<td>Carbohydrate Shock</td>
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<tr>
<td>Renal Dysfunction</td>
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<td>Hypovolemic Shock</td>
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<tr>
<td>Malignancy</td>
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<td>Hemorrhagic Shock</td>
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<tr>
<td>Accelerated Axonal Oxydase SIA</td>
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<td></td>
<td></td>
<td>Hyperpycnic Hypovolemic Syndrome</td>
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<tr>
<td>Methanol, Ethanol</td>
<td></td>
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<td>Severe Pneumocystis Dysfunction</td>
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<td>Anti-vegetarians, Met</td>
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<td>Carbon Monoxide Poisoning</td>
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<td>Opioids/Pulsoxidines</td>
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<td>Certic Arnet</td>
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<td>Vaginal Acid</td>
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<td></td>
<td>Any Condition Where Oxygen</td>
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<td>Early Sepsis</td>
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<td></td>
<td>Delivery is Antidoteic</td>
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<tr>
<td>Pyruvate Dehydrogenase Deficiency</td>
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<td>Advanced Sepsis</td>
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</tbody>
</table>

L-Lactic Acidosis

- Tissue underperfusion (Type A)
  - Shock, shock, shock
  - Hypoxia
  - Asthma
  - CO poisoning
  - Severe anemia

L-Lactic Acidosis

- Medical conditions (w/o tissue hypoxia)
  - Hepatic failure
  - Thianine deficiency (co-factor for pyruvate dehydrogenase)
  - Malignancy
  - Bowel ischemia
  - Seizures
  - Heat stroke
  - Tumor lysis
  - Drugs/Toxins
    - Metformin (particulary associated with hypovolemia and dye)
    - NRTI (especially stavudine and zidovudine)
    - Propofol
    - Nitroprusside

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Conclusions Regarding Different Monitors

- Hemodynamic monitoring becomes more effective at predicting cardiovascular function when measured using performance parameters
  - CVP and arterial pulse pressure (ΔPP) variations predict preload responsiveness
  - ScvO₂, SvO₂, StO₂ predict the adequacy of oxygen transport

Oxygenation Patterns with Normal Function

Oxygen Maintenance

- As tissues’ metabolic activity increases so must O₂ delivery
  - Accomplished via increase in flow
  - Occurs globally via elevated CO
  - Occurs locally by recruitment of capillary beds through auto regulation
  - Tissues able to increase O₂ use if delivery fails to meet the metabolic needs
    - Manifest as lower SvO₂
Assessing $\text{DO}_2$: Mixed Venous Saturation

**Site of measurement of $Sat_{mv}\text{O}_2$**
- superior vena cava vs pulmonary artery
  - Adults with sepsis:
    - $Sat_{svc}$ mean of 7% higher than $Sat_{pa}$
    - $Sat_{svc}$ changes in parallel to $Sat_{pa}$
- inferior vena cava vs superior vena cava
  - ↑ $VO_2$ in hepatosplanchnic region in sepsis
  - ↑ oxygen extraction ratio and ↓ IVC $O_2$ saturation

---

Targeting Mixed Venous Saturation

- normally 70-75%
- may be elevated in sepsis
  - maldistribution of blood flow
  - Increasing LA
- reduced venous saturation with normal arterial saturation → increase in $O_2$ extraction
  - imbalance between $VO_2$ and $DO_2$
  - improve supply

---

Targeting Mixed Venous Saturation

- few studies have specifically targeted resuscitation to a mixed venous saturation of ≥70%
- prospective, randomized trial in adults:
  - treatment to a mixed venous saturation ≥70% did not reduce mortality compared with therapy targeting a normal CI

Tissue Oxygenation

- drop in SvO₂ or ScvO₂ does not necessarily mean tissue hypoxia occurs!

Managing Tissue Oxygenation

Compensation in attempts to sustain Tissue Oxygen

PROBLEM
Oxygen delivery inadequate for oxygen demand
Primary failure

COMPENSATION:
Shift to the right
Release oxygen to save the cells

MEASURE:
ScvO₂ ↓ ↓ ↓ ↓
Always Compensatory
Always an EMERGENCY
Tissue Perfusion: It really is everything!

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Compensation in attempts to sustain Tissue Oxygen

PROBLEM
ScvO2 normal to ↑↑↑↑↑↑↑↑↑↑ in the face of suspicion (HR ↑, RR ↑ persistent acidosis (LA))
MUST BE considered as failure to release oxygen (in the presence of ↑↑LA)

COMPENSATION:
Cardiac output increases
Despite increase, tissue hypoperfusion persists (↑↑LA)

OXYGEN Delivery

OXYGEN release

OXYGEN Demand & Consumption

What Is StO2?
StO2 = hemoglobin oxygen saturation of the microcirculation

SaO2 and SpO2 measure O2 saturation in the arteries.

ScvO2 and SvO2 measure O2 saturation in the superior vena cava. SvO2 measures O2 saturation in the pulmonary artery.

StO2 measures O2 saturation in the microcirculation, where O2 diffuses to tissue cells. Direct measure of tissue oxygenation and sensitive indicator of tissue perfusion status.

Why Measure Thenar Muscle?

- During shock, blood flow to peripheral muscles and core organs (liver, gut and kidneys) is reduced in order to preserve brain and heart oxygenation
- Thenar muscle group is peripheral muscle
- StO2 measured in thenar allows noninvasive monitoring of early changes in perfusion status during shock and resuscitation

Perfusion Changes During Shock
Watching the Value; Watching the Trend

- StO2 low: assess patient, resuscitate if indicated
- StO2 adequate: assess need for further resuscitation, stop if indicated
- StO2 rising: assess need for continued resuscitation
- StO2 falling: assess patient, resume resuscitation if indicated

Low StO2 is associated with poor outcomes and should be investigated

StO2 Monitoring

Patients Who May Benefit

Emergency Medicine
- High Acuity Elderly
  - Over 65 years old with:
    - Injuries from blunt force trauma
    - Congestive heart failure
    - Pulmonary hypertension
- Trauma
  - Any age
  - Mechanism of injury puts at risk for bleeding
  - May have non-alarming clinical signs
- Medical Bleeding
  - All patients at risk for bleeding (susceptible to internal bleeding):
    - Electromechanical
    - Vaginal
    - Nasal

Critical Care
- Patients described above
- Patients considered to be resuscitated; have non-alarming clinical signs
- Patients requiring active treatment
Tissue Perfusion: It really is everything!

- **Vascular Occlusion Test (VOT)**

  - $\Delta S\text{O}_2$ (%)
  - $\Delta B\text{O}_2$ (%)
  - $R\text{n}\Delta S\text{O}_2$
  - $R\text{n}\Delta B\text{O}_2$

  - Arterial occlusion
  - Arterial reperfusion

  - $S\text{O}_2$ (%) vs. Time

  - $B\text{O}_2$ (%) vs. Time

- **DYNAMIC NEAR-INFRARED SPECTROSCOPY MEASUREMENTS IN PATIENTS WITH SEVERE SEPSIS.**

  - Skarda, David; Mulier, Kris; Myers, Dean; Taylor, Jodie; Beilman, Greg

Tissue Oxygenation

- drop in SvO₂ or ScvO₂ does not necessarily mean tissue hypoxia occurs!
- Return of SvO₂ or ScvO₂ does not necessarily mean tissue oxygenation returned to normal!
- StO₂ may be inverse! Think about it!
A-V PCO$_2$ Gradient ($\Delta$PCO$_2$)

- Can the PCO$_2$ gradient between arterial and venous blood gas samples ($\Delta$PCO$_2$) represent adequacy of perfusion?

A-V PCO$_2$ Gradient ($\Delta$PCO$_2$)

- $\Delta$PCO$_2$ = PvCO$_2$ – PaCO$_2$
- The $\Delta$PCO$_2$ is an index to identify the critical oxygen delivery point ($VO_2/DO_2$).
- The critical oxygen delivery point is when consumption ($VO_2$) is dependent on delivery ($DO_2$).

A-V PCO2 Gradient ($\Delta$PCO2)

- Critical oxygen delivery point is associated with an abrupt increase of blood lactate levels and a significant widening in $\Delta$PCO$_2$.
- Since CO$_2$ is 20x more soluble in aqueous solutions than O$_2$, it is logical that $\Delta$PCO$_2$ may serve as an excellent measurement of adequacy of perfusion.
Measures of Tissue Oxygenation

- **Lactate/pH**
  - Normal lactate: 1-2 mmol
  - pH: normal 7.35-7.45
  - If lactate > 4 mmol and pH < 7.30, consider tissue hypoxia
    - Lactate/pyruvate
    - Lactate normally 10 x pyruvate
    - If lactate rising proportionately faster than pyruvate, consider tissue hypoxia (Type A lactic acidosis)

- **StO₂**
  - Reflects tissue perfusion
  - Should not be the same as ScVO₂
  - Potentially earliest indicator of a threat to tissue oxygenation

Comparison of △PCO₂ and SvO₂

- **Key Points:**
  - SvO₂ may reflect the metabolic rate and oxygen consumption
  - △PCO₂ and/or serial lactate levels and clearance may reflect the adequacy of tissue perfusion
Case Presentation 1

- 54 year old man with fever and abnormal liver function for liver biopsy
- Biopsy “well tolerated” until 3 hours afterwards when he developed abdominal distension, with systolic BP 60 and Hg 8.6

Case Presentation 2

- ScV02: 54%, HR 128, RR 26
- 22% SVV what now?
- PP/SV 0.8. Vasoconsticted or dilated?
- Next?

Volume responsive!

Case Presentation 2

- SVV 15%
- PP/SV 1.8
- SvO2 50%
- StiO2 40%
- Next?

Vasopressor
Case Presentation 2

- SVV 20%
- PP/SV  0.8
- SvO₂  80%
- StiO₂  40%
- Next?

Volume inotrope dilator

Remember…..

A searchlight cannot be used effectively without a fairly thorough knowledge of the territory to be searched.
Fergus Macartney, FRCP

Before I came here I was confused about this subject. Having listened to your lecture I am still confused. But on a higher level.
-Enrico Fermi