Monitoring in the Critical Ill

1) Clinical:
   - GCS
   - Skin temperature, colour, capillary refill
   - Pulse; rythem, rate, quality
   - Urine output
   - JVP
   - Systems examination

2) Biochemical:
   - Blood tests-electrolytes, FBC, coagulation profile, etc.
   - Blood gas
   - TEG

3) Microbiological:
   - Blood cultures
   - LUKI

4) Imaging:
   - X-rays
   - U/S
   - CT scan
   - MRI

5) Mechanical and Electrical:
   - ECG
   - Non-invasive BP
   - Invasive BP monitoring-A-line
   - CVP monitoring
   - Pulmonary Artery Catherization (Swan Ganz)
   - CO2 analyser
   - Capnography
   - Oesophageal Doppler
   - Oxymetry
   - Tonometry
   - Hand held Doppler
   - Endotracheal cuff pressures
   - Bleeding time

Normal Hemodynamic Parameters – Adult

<table>
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<tr>
<th>PARAMETER</th>
<th>EQUATION</th>
<th>NORMAL RANGE</th>
</tr>
</thead>
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<tr>
<td>Arterial Oxygen Saturation</td>
<td>(SaO2)</td>
<td>95 - 100%</td>
</tr>
<tr>
<td>Mixed Venous Saturation</td>
<td>(SvO2)</td>
<td>60 - 80%</td>
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<tr>
<td>Central Venous Oxygen Saturation</td>
<td>(ScvO2) 70%</td>
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<tr>
<td>Arterial Blood Pressure (BP)</td>
<td>Systolic (SBP)</td>
<td>100 - 140 mmHg</td>
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<tr>
<td>Diastolic (DBP)</td>
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<td>60 - 90 mmHg</td>
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<tr>
<td>Mean Arterial Pressure (MAP)</td>
<td>SBP + (2 x DBP)/3</td>
<td>70 - 105 mmHg</td>
</tr>
<tr>
<td>Right Atrial Pressure (RAP)/</td>
<td></td>
<td>2 - 6 mmHg</td>
</tr>
<tr>
<td>Central Venous Pressure (CVP)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PARAMETER</td>
<td>EQUATION</td>
<td>NORMAL RANGE</td>
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<tr>
<td>Right Ventricular Pressure (RVP)</td>
<td></td>
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</tr>
<tr>
<td>Systolic (RVSP)</td>
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<td>15 - 30 mmHg</td>
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<tr>
<td>Diastolic (RVDP)</td>
<td></td>
<td>2 - 8 mmHg</td>
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<tr>
<td>Pulmonary Artery Pressure (PAP)</td>
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</tr>
<tr>
<td>Systolic (PASP)</td>
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<td>15 - 30 mmHg</td>
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<tr>
<td>Diastolic (PADP)</td>
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<td>8 - 15 mmHg</td>
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<tr>
<td>Mean Pulmonary Artery Pressure (MPAP)</td>
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<tr>
<td></td>
<td>PASP + (2 x PADP)/3</td>
<td>9 - 18 mmHg</td>
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<td>Pulmonary Artery Occlusion Pressure (PAOP)</td>
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<td></td>
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<td>6 - 12 mmHg</td>
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<tr>
<td>Left Atrial Pressure (LAP)</td>
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<td></td>
<td>4 - 12 mmHg</td>
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<tr>
<td>Cardiac Output (CO)</td>
<td>HR x SV/1000</td>
<td>4.0 - 8.0 L/min</td>
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<tr>
<td>Cardiac Index (CI)</td>
<td>CO/BSA</td>
<td>2.5 - 4.0L/min/m2</td>
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<tr>
<td>Stroke Volume (SV)</td>
<td>CO/HR x 1000</td>
<td>60 - 100mL/beat</td>
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<tr>
<td>Stroke Volume Index (SVI)</td>
<td>CI/HR x 1000</td>
<td>33 - 47 mL/m2/beat</td>
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<td>Stroke Volume Variation (SVV)</td>
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<tr>
<td></td>
<td>SVmax - SVmin/ SVmean x 100</td>
<td>10 - 15%</td>
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<tr>
<td>Systemic Vascular Resistance</td>
<td>80 x (MAP - RAP)/CO</td>
<td>800-1200dynes</td>
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<tr>
<td>Systemic Vascular Resistance Index (SVRI)</td>
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<td>1970 - 2390 dynes</td>
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<tr>
<td>Pulmonary Vascular Resistance (PVR)</td>
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</tr>
<tr>
<td></td>
<td>80 x (MPAP - PAOP)/CO</td>
<td>&lt;250 dynes- sec</td>
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<tr>
<td>Pulmonary Vascular Resistance Index (PVRI)</td>
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</tr>
<tr>
<td></td>
<td>80 x (MPAP - PAOP)/CI</td>
<td>255 – 285 dynes- sec</td>
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<tr>
<td>Left Ventricular StrokeWork (LVSW)</td>
<td>SI x MAP x 0.0144</td>
<td>8 - 10 g/m/m2</td>
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<td>Left Ventricular StrokeWork (LVSWI)</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>SVI x (MAP - PAOP) x 0.0136</td>
<td>50 - 62 Index</td>
</tr>
<tr>
<td>Right Ventricular StrokeWork (RVSW)</td>
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<td></td>
</tr>
<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>SI x MAP x 0.0144</td>
<td>51 - 61 g/m/m2</td>
</tr>
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<td></td>
<td>SVI x (MPAP - CVP) x 0.0136</td>
<td>5 - 10 Index</td>
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<td>Coronary Artery Perfusion Pressure (CPP)</td>
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</tr>
<tr>
<td></td>
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<td>60 - 80 mmHg</td>
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<tr>
<td>Right Ventricular End-Diastolic Volume (RVEDV)</td>
<td></td>
<td>100 - 160 mL</td>
</tr>
<tr>
<td>Right Ventricular End-Diastolic Volume Index (RVEDVI)</td>
<td></td>
<td>60 - 100 mL/m2</td>
</tr>
<tr>
<td>Right Ventricular End-Systolic Volume (RVESV)</td>
<td></td>
<td>50 - 100 mL</td>
</tr>
<tr>
<td>PARAMETER</td>
<td>EQUATION</td>
<td>NORMAL RANGE</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
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</tr>
<tr>
<td>Right Ventricular Ejection Fraction (RVEF)</td>
<td>SV/EDV x 100</td>
<td>40 - 60%</td>
</tr>
<tr>
<td>Arterial Oxygen Content (CaO2)</td>
<td>(0.0138 x Hgb x SaO2) +0.0031 x PaO2</td>
<td>16 - 22 mL/dL</td>
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<tr>
<td>Venous Oxygen Content (CvO2)</td>
<td>(0.0138 x Hgb x SvO2) +0.0031 x PvO2</td>
<td>15 mL/dL</td>
</tr>
<tr>
<td>A - V Oxygen Content Difference</td>
<td>(C(a - v)O2) CaO2 - CvO2</td>
<td>4 - 6 mL/dL</td>
</tr>
<tr>
<td>Oxygen Delivery (DO2)</td>
<td>CaO2 x CO x 10</td>
<td>950 - 1150 mL/min</td>
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<tr>
<td>Oxygen Delivery Index (DO2I)</td>
<td>CaO2 x CI x 10</td>
<td>500 – 600 mL/min/m2</td>
</tr>
<tr>
<td>Oxygen Consumption (VO2)</td>
<td>C(a - v)O2 x CO x 10</td>
<td>200 - 250 mL/min</td>
</tr>
<tr>
<td>Oxygen Consumption Index (VO2I)</td>
<td>C(a - v)O2 x CI x 10</td>
<td>120 - 160 mL/min/m2</td>
</tr>
<tr>
<td>Oxygen Extraction Ratio (O2ER)</td>
<td>(CaO2 - CvO2)/CaO2 x 100</td>
<td>22 - 30%</td>
</tr>
<tr>
<td>Oxygen Extraction Index (O2EI)</td>
<td>(SaO2 - SvO2)/SaO2 x 100</td>
<td>20 - 25%</td>
</tr>
</tbody>
</table>

**Endotracheal Cuff Pressure**

Tubes for tracheal intubation in adults are equipped with inflatable balloons (cuff) that is used to seal the trachea and isolate the airways from the larynx and oral cavity.

**Cuff related problems:**
Aspiration  Pneumonia thus must suction regularly.
Cuff leaks = check cuff pressure <25 cm H2O
Tracheal Necrosis = low compliance cuffs
Systolic pressure in tracheal mucosa is 20-25 mmHg, so ideal pressure is below 20 cm H2O
If patient is shocked or hypotensive pressure necrosis is possible at much lower pressures.

**ELECTRO CARDIO Gram [ECG].**
- Continuous monitoring -Rate and Rythm.
- Detect pacemaker function, arrythmia, ischaemia and electrolyte abnormalities.
BLOOD PRESSURE MONITORING

Pulse Pressure = SBP - DBP
PULSE PRESSURE varies with stroke volume
Pulse Pressure <30 - Hypovolemia
- Tachycardia
- Aortic stenosis
- Constrictive pericarditis
- Pleural effusion
- Ascitis

Pulse Pressure widen due to:
  - Aortic regurgitation
  - Thyrotoxicosis
  - Aortic coarctation

NON INVASIVE BLOOD PRESSURE MONITORING:

1. Palpation:
SBP is the pressure where the pulse resumes when cuff is deflated, this underestimates the SBP.

2. Auscultation:
Korotkoff sounds. A cuff 20% wider than the diameter of the limb must be used, if the cuff is too narrow the SBP and DBP will be artificially increased.
With pressures >120 auscultation overestimates and with pressures <120 underestimates arterial pressure.

3. Oscillometry:
Automated devices that use a single bladder cuff. It can indicate MAP and requires a few cardiac cycles to measure BP accurately. Unreliable in patients with irregular rythms, too much motion and low flow states.

4. Doppler:
Ultrasonic probe on an artery distal to a compressing cuff. Disadvantage include motion sensetivity, inaccurate placement, and the use of Ultrasonic gel.

5. Tonometry:
Measures BP by sensing the occlusive pressure required to stop flow through a superficial artery.

INVASIVE BLOOD PRESSURE MONITORING

The most accurate technique to monitor blood pressure is to insert a catheter into an artery. Common arteries used are Radial, Ulnar, dorsalis pedis, post tibial ,femoral
and axillary arteries. Radial artery pressure underestimates central pressure in hypotensive septic patients receiving high-dose vasopressor therapy. Clinical management, based on radial pressures, may lead to excessive vasopressor administration. (Crit Care Med 1998; 26: 1646-1649)

CENTRAL VENOUS PRESSURE:

All blood flows back to the Right Atrium, thus filling pressure = CVP. In diastole, the CVP decline to 0 and in systole it raises to 8 mmHg thus CVP fills the R ventricle and controls the end diastolic volume, the stroke volume and the cardiac output. **NORMAL CVP** = -4 to 15 mm Hg

**CVP wave forms**

a- wave = Atrial contraction

c- wave = Tricuspid valve displace into RA with initial V contraction

X-decent = Period of V ejection

V-wave = Increase in Artrial pressure as Venous return rises while valve is still closed

y-decent = Tricuspid valve opens at the end of ventricle contraction and blood enters the RV

**The influence of Ventilation on the CVP**

**Spontaneous Ventilation**: CvP lowers with insp. CVP rise with exp due to negative intrathor pressure.

**Mechanically Ventilated**: CvP rise with isnp & CVP lowers with exp due to positive intrathor pressure.

**PEEP** decreases venous return and increase the CVP

CVP should always be measured at end-expiration

**Vigileo**

1. Cardiac Output: Blood pumped from heart in liters/min.
2. Central Venous Oxygen Saturation: Assessment of balance between DO2 and VO2. Lower values indicate increased oxygen extraction or decreased delivery. Higher levels are seen with impaired oxygen utilization and extraction.
4. Stroke Volume Variation (For use on control ventilated patients): Variation in arterial pulsations caused by volume changes during positive pressure inspiration.
>15% may indicate hypovolemia.

**PULMONARY ARTERY CATHETERIZATION.**

The mean pressure in the LA represents the filling pressure of the LV. This can be measured with a PA catheter and is referred to as wedge pressure

**The function of a Swan Ganz**
1) Provides information related to the L heart filling pressures
2) Allows sampling of pulmonary artery blood for mixed venous oxygen saturation measurement
3) If combined with a thermister it allows us to measure CO(cardiac output) with increased accuracy of cardiac output measurement through the elimination of ventilator cycle and thermal noise effects.

**Continuous Svo2 monitoring** can be useful as a:

- Surveillance and early warning system.
- Guide for adjusting and assessing therapy and routine nursing care. (eg. convenient and accurate means to adjust PEEP rapidly to optimal levels without repeated blood gases and cardiac output determinations.)
- Means for interpreting other variables (eg. If cardiac output is adequate).

**How does it work?**

Inflation of the balloon obstructs blood flow and creates a static column of blood between the tip and the left atrium -thus- the pressure at the tip should be the same as the pressure in the LA.

**Usefulness:** For patients in need of the highest level of monitoring and those who require precision-guided hemodynamic therapy for:

- ARDS
- Cardiac Surgery
- Severe Trauma
- Cardiogenic Shock
- Septic Shock
• Hemorrhagic Shock
• CHF
• High-risk Surgery
• Pulmonary Hypertension
• Surgical Pre-optimization
• Ventilator Patients with PEEP
High $\text{SvO}_2$  
$\uparrow$ Oxygen delivery  
$\uparrow$ $\text{FiO}_2$

Hyperoxia

$\downarrow$ Oxygen demand

Hypothermia
Anesthesia
Pharmacologic paralysis
Sepsis

Low $\text{SvO}_2$  
$\downarrow$ Oxygen delivery

$\downarrow$ Hb  
Anemia, hemorrhage

$\downarrow$ $\text{SaO}_2$  
Hypoxia, suctioning

$\downarrow$ CO  
Hypovolemia, shock
Arrhythmias

$\uparrow$ Oxygen demand

Hyperthermia, pain
Shivering, seizures

Table 2
Clinical applications of $\text{SvO}_2$
Vigilance II Monitor

- Displays continuous assessment of oxygen delivery and consumption
- When used with a Swan-Ganz Advanced Technology pulmonary artery catheter, provides a broad view of hemodynamic performance
- Continuous CO, SvO2, EDV, SVR, and other measured and derived parameters are presented on customized displays selected
- Interface with the bedside monitor for convenience and display of additional measured and derived parameters such as continuous SVR, DO2, DO2I, VO2, VO2I, O2EI, O2ER, and VQI.

The FloTrac System Enables Precise Management to Fluid Optimization

The FloTrac system equips clinicians with a set of tools to assess both dimensions of the Frank-Starling curve. The algorithm of the FloTrac system utilizes advanced waveform processing to adjust dynamically for vascular tone (resistance and compliance) in addition to patient specific variables (age, gender, body surface area, etc.) in order to calculate the key flow related parameters stroke volume and cardiac output. The result is an enhanced ability to determine the adequacy of cardiac flow, which comprises the Y-axis of the Frank-Starling Curve.
Second, the FloTrac system measures **preload responsiveness** for the X-axis of the Frank-Starling curve through one of three distinct, practical methods:

- **Stroke Volume Variation (SVV):** For control-ventilated patients, SVV has been proven to be a highly sensitive and specific indicator for preload responsiveness. As a dynamic parameter, SVV has the advantage of predicting whether a patient will benefit from volume before the fluid is given.

- **Passive Leg Raising (PLR):** In situations where it is not possible to use SVV (i.e., during arrhythmias, when patients are not on control-mode of ventilation, or in patients at risk of complications from fluid loading), simply raising the legs has been proven clinically to act like a “self volume challenge” to indicate the patient’s status on the Frank-Starling curve. If the patient is fluid-responsive, SV will increase substantially.

- **SV Fluid Challenge:** In the rare case when neither SVV nor PLR is feasible, the FloTrac system provides a highly efficient method for assessing fluid responsiveness via a standard fluid challenge. The administration of a small volume of fluid (e.g., 250-500 mL) and observance of the corresponding change in SV and/or CO can indicate whether further volume will improve cardiac performance.

**MIXED VENOUS OXYGEN SATURATION (SV02)**

\[
SV02 = (SaO2 - VO2) \div (Hb \times 1.39 \times Q)
\]

The distal port of the Swan Ganz can be used for sampling true mixed venous blood to be used as SV02 measurement to indicate systemic oxygen utilization as VO2 (Peripheral O2 consumption) is independent of DO2.

Monitoring of central venous oxygen saturation (ScvO2) has been advocated as a simple method of assessing changes in the global oxygen supply-to-demand ratio in various clinical settings. Others, however, have questioned the ability of ScvO2 measurements to track SvO2. For example, in shock the oxygen extraction increases in nonvital organs such as the hepatosplanchnic region, causing a reduced oxygen saturation in the inferior vena cava and thus increasing the difference between SvO2 and ScvO2.

Consideration of ScvO2 may be appropriate, especially if it is continuously monitored.

**CARDIAC OUTPUT:**

*Time-Temperature Curve* – (Thermodilution technology) Inject a known quantity of a cold solution into circulation and measure the CO. The area under the time-temperature curve represents the CO (inversely proportionate)
The liquid to be injected must be at least 12degC colder than body temperature. Injection at end exp improve consistency of measurement, and more than one measurement must be taken.
Other methods of measuring CO:

1. **Indicator Dilution.** Indocyanine green is used like the cold water but light absorption is measured-densitometer

2. **Doppler Ultrasound.** Measure the flow in the ascending aorta and CO is calculated. A continuous flow doppler beam is put in suprasternal notch.
   Errors include: malalignment of the beam, the aorta might not be round and the flow might not be laminer
   Esophageal Doppler (Hemosonic) is better.

3. **Thoracic Bio impedance.** Non invasive and measures stroke volume by small alternating current. 2 electrodes at base of neck and 2 at the Xiphi sternum - placement is important for accuracy

4. **Ficks Method.** CO is calculated by relating to art O2 content and venous O2 content.
   
   \[ CO = \frac{VO2}{(CaO2-CvO2)} \]
   
   VO2 = whole body O2 consumption
   CaO2: Arterial O2 content
   CvO2: Venous O2 content

5. **Continuous Cardiac Output.** Continuous monitoring of cardiac output using a modified pulmonary artery catheter with a heated filament has proven to be accurate and precise in the critically ill patient when compared with the "standard" intermittent bolus thermodilution technique.

**AIRWAY CO2 Monitoring**
Rapid responding infrared CO2 analyzer that measures CO2 in ET tube.
Capnography is the continuous display or recording of CO2 during each breath. This is non invasive. Provides early detection of significant events by displaying changes in CO2 levels and abnormal wave forms.
WHAT CAN BE USED TO MONITOR ORGAN PERFUSION AND MICROCIRCULATION?

Regional monitoring

Initial resuscitation of critically ill patients with shock does not require monitoring of regional variables. After stabilization, however, regional variables (lactate, gastric mucosal pH, mucosal-end tidal Pco2 gap, mucosal-arterial Pco2 gap, indocyanine green plasma clearance, and plasma disappearance rate of dye) are the best predictors of outcome.

Indirect methods to measure tissue perfusion and oxygenation:

1. Oxygen delivery and consumption

\[
\text{DO}_2 = \text{CO} \times \text{CaO}_2
\]

\[
\text{CaO}_2 = (\text{Hb} \times 1.39 \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)
\]

\[
\text{N} = 1000\text{ml/min or 14 ml/kg/min.}
\]

The ratio of \(\text{VO}_2: \text{DO}_2\) = oxygen extraction ratio


\[
\text{SV}_02 = (\text{SaO}_2 - \text{VO}_2) ÷ (1.39 \times \text{Hb} \times \text{CO})
\]

3. Blood LACTATE

Pyruvate > lactate dehydrogenase > Lactate.
If > 2mmol = poor oxygenation, poor perfusion or circulatory failure.

The severity of lactic acidosis in critically ill patients correlates with overall oxygen debt and survival. Lactate determinations may be useful as an ongoing monitor of perfusion as resuscitation proceeds.

According to Critical Care Medicine 2009, blood lactate measurement in critically ill patients: 1) is accurate in terms of measurement technique but adequate understanding of the (an)aerobic etiology is required for its correct interpretation; 2) provides not only diagnostic but also important prognostic information; 3) should be measured directly instead of estimated from other acid-base variables; 4) can alter therapeutic decisions; 5) could potentially improve patient outcome when combined
with a treatment algorithm to optimize oxygen delivery, but this has only been shown indirectly; 6) is likely to have similar benefits in critical care settings worldwide; and 7) has an unknown cost-effectiveness.

It is unknown whether the routine use of lactate as a resuscitation end point improves outcome. This warrants randomized controlled studies on the efficacy of lactate-directed therapy.

4. TONOMETRY
A sterile silicone balloon is placed in the stomach and filled with NaCl -CO2 is absorbed and measured in a bloodgas machine after 30 min and this is a strong indicator of splanchnic hypoperfusion and elevated risk of bacterial translocation and subsequent SIRS. Gastric intramucosal pH is also considered as attractive option in diagnosing and monitoring splanchnic hypoperfusion. Splanchnic hypoperfusion - intramucosal PCO2 increase and pH decrease. CO2 diffuses into hollow viscus, into balloon. PCO2 in a hollow viscus, is called regional PCO2 = PrCO2. PrCO2 is the balance between CO2 production and CO2 transport. Normal = 8-10mm Hg and we aim to keep the gap <25mm Hg.

When should we consider Tonometry?

1. Cardiogenic shock
2. Septic shock
3. Acute Resp failure
4. Acute Pancreatitis
5. Major Burns - acute phase resus
6. After therapy to confirm tissue perfusion

Continuous non invasive measurement of cytochrome aa3
A decrease in DO2 leads to a decrease in cytochrome aa3.

6. Tissue Oxygen tension.
Miniature implantable electrodes measures Oxygen tension (pti O2) in organs and bodily fluids directly on a continuous basis.

7. Transcutaneous O2 monitoring
Patients not in shock responded to changes in inspired oxygen concentration (FIO2) with changes in Pao2 and PtcO2 values; the 95% response time was approximately 2 min. The authors conclude that the normal value for PtcO2 for adult surgical
patients who are hemodynamically stable is 79 +/- 12% of the PaO2 and that PtcO2 values were reliable, continuous, noninvasive trend monitors of PaO2 in these patients. During circulatory problems when PtcO2 values were compared to PaO2 values (PtcO2 index), the changes reflected trends in the severity of low flow shock.

8. Sublingual capnometry

The initial $P_{SL\text{co}_2}$-$P_{aco}_2$ gradient ($P_{SL\text{co}_2}$-diff) and the initial $P_{SL\text{co}_2}$ were highly predictive of outcome. The baseline $P_{SL\text{co}_2}$-diff and $P_{SL\text{co}_2}$ were better predictors of outcome than traditional markers (arterial lactate concentration and SvO2) of tissue hypoxia and were more responsive to therapeutic interventions. The $P_{SL\text{co}_2}$-diff and/or $P_{SL\text{co}_2}$ may prove to be a useful marker for goal-directed therapy and for assessing the response to clinical interventions aimed at improving tissue oxygenation.

OESOPHAGEAL DOPPLER (Haemosonic)

Transesophageal Doppler can provide a noninvasive, clinically useful estimate of cardiac output and detect hemodynamic changes in mechanically ventilated, critically ill patients.

The doppler emits a continuous wave at +/- 5mHz and this determines the flow velocity of red cells.

The probe is positioned ± 30 - 40 cm from the teeth in the esophagus, where the aorta runs parallel to it. The stroke volume is calculated as a product of the systolic red cell velocity and aorta gross sectional area.

Esophageal Doppler monitoring allows a reliable noninvasive estimation of cardiac output by measuring the aortic blood flow (ABF) in the descending thoracic aorta. This monitoring device tracks the changes in cardiac output induced by inotropic drugs or volume replacement. The duration of the aortic velocity signal corrected for heart rate, so-called “flow time corrected” (FTc), is considered a static indicator of cardiac preload. The respiratory variation in aortic blood flow reliably predicts fluid responsiveness in patients with sinus rhythm and without breathing activity. Respiratory variation has superiority over static parameters of preload as filling pressures or echographic left ventricular dimensions.

Analysis of the velocity time wave forms provides information about preload, contractility and after load.

OXIMETRY AND CAPNOGRAPHY

Spectrophotometry is applied to the detection of oxygenated Hb (Hb02) and deoxygenated Hb and this is called oximetry.
The optical detection of CO2 is called capnometry and the continuous recording of CO2 concentration during each breath is called capnography.

Hb configuration changes during chemical reactions and each configuration has a different pattern of light reflection.

At wavelengths of 660nm (the red region of the light spectrum) Hb02 reflects more light than Hb (arterial blood is more red). At 940 nm (infrared spectrum) Hb reflects more light than Hb02. Both wavelengths of light is passed through a sample of blood and the concentrations of Hb and Hb02 are expressed as a fraction of the Hb that is oxygenated.

\[
\text{thus} = \% \text{Saturation} = \frac{\text{Hb}02}{\text{Hb}02 + \text{Hb}} \times 100
\]

**Limitations**
The use of 2 wavelengths is based on the assumption that other forms of Hb - MetHb and COHb- normally to blood samples contribute. In abnormal situations a high COHb (smoke inhalation) and a high MetHb (Nitrogliserine overdose) will lead to false high estimates of Hb02.

Arterial pulsations are associated with changes in blood volume and this changes the intensity of light transmission This eliminates light reflected from nonpulsatile stuctures like veins.

**CO oximetry:**
4 Wavelengths are transmitted through a sample and this can detect MetHb, COHb, Hb and Hb02.

**Mixed Venous oximetry:**
Catheter tip with PAC that reflects red and infrared light in pulmonary artery. The reflected light is then measured and this might give a continuous monitoring of Hb, HbO2 and SV02 (Venous)

**Pulse oximetry**
Records light transmission through pulsatile arteries only. 2 wavelengths are reflected The probe is placed on the fingers. At clinical accepted levels a SaO2 > 70% is recorded very accurately.
NB! - Not calibrated for SaO2 < 70%

**BLOOD GASES**
Continuous intra-arterial blood gas analysis can add substantially to the safety of patients with acute respiratory failure and can reduce blood sampling requirements for blood gas analysis.

The partial pressure of oxygen (PO2) is measured in mm Hg and reflects the tension or pressure that is exerted by oxygen when it is dissolved in plasma.

Oxygen saturation (SO2) is a measurement of the amount of oxygen bound to hemoglobin (Hb).

![Figure 2](image)

The *association segment* of the curve, or upper portion, is essentially flat and represents *oxygen uptake* in the lung. (See Figure 3.) In this portion of the curve, changes in PO2 levels between 60 and 100 mm Hg cause only small changes in oxygen saturation.
The lower portion of the curve (below 45 mm Hg) corresponds to the PO2 levels of venous blood. (See Figure 4.) This steep part of the curve is referred to as the dissociation segment and represents the release of oxygen to the tissues. In this low range of PO2 values, even small changes in oxygen tension produce large alterations in oxygen saturation. This is advantageous to the tissue because large quantities of oxygen can be extracted from the blood for relatively small decreases in PO2.

Haemoglobin-oxygen affinity refers to the strength of the bond between haemoglobin and oxygen. It is expressed as the PO2 value where hemoglobin is 50% saturated with oxygen and is referred to as the P50. The P50 is 27 mm Hg under standard conditions (pH 7.40, T 37°C, PCO2 40 mm Hg, normal 2,3 DPG). Several factors affect the affinity of haemoglobin for oxygen. An increase in hydrogen ion concentration (pH), PCO2, temperature, or 2,3 DPG (a byproduct of red blood cell metabolism) will decrease haemoglobin-oxygen affinity, thereby shifting the oxyhemoglobin curve to the right. (See Figure 5.) This shift results in a higher P50.
value, indicating that a higher PO2 will be required to saturate 50% of the hemoglobin.

Figure 5
Shift in the oxyhemoglobin dissociation curve resulting from changes in the oxygen affinity of hemoglobin

References


