Invasive Mechanical Ventilation & Hemodynamic Monitoring

HEART AND LUNG CONNECTION

- The heart and lungs are pressure-driven systems that share the primary responsibility for oxygen uptake and delivery to the body.
- They share a common space (thorax) and are linked anatomically.
- Heart–Lung Interactions
  - Intrathoracic pressure changes
  - Lung volume changes
  - Pulmonary vascular resistance
  - Mechanical effects of lung expansion
  - Abdominal pressure changes
  - Ventricular interdependence

Some terms

- Stroke Volume (SV) = EDV – ESV
- Ejection Fraction (EF) = (SV / EDV) × 100%
- Cardiac Output (Q) = SV × HR
- Cardiac Index (CI) = Q / Body Surface Area (BSA) = SV × HR/BSA
  - HR is Heart Rate, expressed as BPM (Beats Per Minute). BSA is Body Surface Area in square meters.
Hemodynamic data: BP, CO, $C_{(a-v)}O_2$, PAP, PCWP, PvO$_2$, SvO$_2$, O$_2$ transport (CO x CaO$_2$).

Simple estimates
- end-diastolic volume (EDV) 120 ml
- end-systolic volume (ESV) 50 ml
- stroke volume (SV) 70 ml
- ejection fraction (E$_f$) 58%
- heart rate (HR) 70 bpm
- cardiac output (CO) 4.9 L/m

Bedside estimates
- SV = 2 ml × Pulse Pressure
- Q = 2 ml × Pulse Pressure × HR

- BP = 120/80  HR = 75
- Pulse press = Systolic − Diastolic
- 120−80 = 40
- SV = 2 ml × 40 = 80 ml
- Q = 2 ml × 40 × 75 = 6.0 L/min
### Cardiovascular (CV) Effects of PPV

- **Early studies show marked reduction in cardiac output**
  - Amount of reduction proportional to pressure applied
- **PPV causes \( P_{pl} \) to become positive**
  - Intrathoracic veins are compressed, increased CVP
    - Impedes venous return and venous pooling
    - Cardiac output (CO) decreases within few beats
    - Increases ICP with a reduction in cerebral perfusion, may lead to cerebral ischemia/hypoxia
      - Healthy persons minimized by autoregulation
    - Compensatory mechanisms minimize CV affects
      - Increase HR, SVR, blood to core organs

### Cardiovascular Effects of PPV

Pulmonary vascular pressure, blood flow, and pulmonary vascular resistance (PVR)

- Normally PPV has a minimal impact on all the above
- If alveolar distention occurs, due to high \( V_T \) or excessive PEEP, blood flow is impeded as the capillaries are compressed between over-distended alveoli
  - Increases RV afterload and decreases CO
  - The stiffer the lungs, the more this affect is minimized.

### Cardiovascular Effects of PPV

Right and left ventricular function

- Normal CV systems: there will be no significant affect
- Conditions that cause impairment
  - Hypovolemia, excessive \( V_T \) or PEEP

Effect with left ventricular dysfunction

- May improve CV function due to decreased afterload
- Endocardial blood flow may be impeded
  - Determined by \( \Delta P \) between \( P_{wedge} \) and wedge
  - Decreased by excessive PEEP, MAP, high \( V_T \), long \( I_T \)
Cardiovascular Effects of PPV

CO, CI, and systemic blood pressure
- No alteration if CV system is normal? and PPV is normal
- High MAP may decrease all of these values
- Ways to minimize or avoid these affects are:
  - Adequate fluid balance
  - Proper management of PPV
    - Lowest MAP and PEEP possible
  - Use of vasopressors
- Most common cause of hypotension in PPV is sepsis followed by hypovolemia.

Minimizing the CV Effects of PPV

Mean pleural pressures (MPPs)
- Clinically monitored by following MAP
  - Linear relationship
- Higher the MPP, the greater the CV effects
- Affects are reduced in noncompliant lungs, i.e., stiff lungs seen in ARDS patients.
- Moderate increases in MPP have small effect in most stable patients.
  - If patient is hypovolemic, a small change in MPP could cause a serious decrease in CO.

Minimizing the CV Effects of PPV

Decreasing the mean airway pressure (MAP)
- Determined by f, VT, I, T, I pause, E, I:E, PIP, PEEP, and inspiratory flow pattern
- Changes of the above to decrease MAP
  - If PaO2 is high, reduce PEEP: very effective
    - Follow carefully to avoid desaturation
  - Increased compliance
    - PEEP and diuretics
  - Decreased VT or PC level
  - Decreased I and I:E ratio
  - Changing from A/C to SIMV, PSV, & mixed ventilation modes.
Minimizing the CV Effects of PPV

Fluid management and cardiac output (CO)
- In face of sepsis, high PEEP and MAP, preload and CO may decrease thus worsening DO$_2$
  - Fluid or blood administration to maintain CO
- Pharmacologic maintenance of CO and blood pressure
- First line is fluid administration except CHF
- Shock
  - Fluid to achieve CVP 8–10 or wedge of 18 mm Hg
  - Persistent shock, use dobutamine
  - If BP is low, dopamine
**Preload**
- Is the degree of muscle fiber stretching present in the ventricles right before systole
- Is the amount of blood in a ventricle before it contracts; also known as "filling pressures"
- Left ventricular preload is reflected by the PCWP
- Right ventricular preload is reflected by the CVP [RA]

**Afterload**
- Any resistance against which the ventricles must pump in order to eject its volume
- How hard the heart [either side left or right] has to push to get the blood out
- Also thought of as the " resistance to flow" or how "clamped" the blood vessels are

**Cardiac Output/Index**
- Is the amount of blood ejected from the ventricle in one minute
- Two components multiply to make the cardiac output: heart rate and stroke volume [amount of blood ejected with each contraction]
- Cardiac index is the cardiac output adjusted for body surface area (BSI)
Adequate Oxygen Delivery?

Demand | Consumption

Oxygen Delivery

Cardiac Output

Oxygen Content

Arterial Blood Gas

Hemoglobin

Oxygen Content

Oxygen Delivery

= Cardiac Output

Oxygen Content

Oxygen Consumption

Oxygen Delivery

= Oxygen Consumed

Remaining Oxygen to Heart

Oxygen Uptake by Organs & Tissues

Oxygen Content in CVP & PA
Physiological Truth

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

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 of Monitoring

• To assure the adequacy of perfusion
• Early detection of inadequacy of perfusion
• To titrate ventilator therapy to specific hemodynamic end point

Hemodynamic monitoring for individual patient should be physiologically based and goal oriented.
Hemodynamic monitors (1)

- Traditional invasive monitors
  - Arterial line
  - CVP & ScvO₂, Central Venous Oxygen Saturation
- PA catheter, CCO, SvO₂
- Functional pressure variation
  - Pulse pressure variation
  - Stroke volume variation

Hemodynamic monitors (2)

- Alternative to right-side heart catheterization
  - PICCO
- Echocardiography
  - Transesophageal echocardiography (TEE)
  - Esophageal doppler monitor

Is Cardiac Output Adequate?

Is blood flow adequate to meet metabolic demands?

Pump function?

Adequate intravascular volume?

Driving pressure for venous return?
Is Cardiac Output Adequate?

We Should Know

Left & right ventricular function
The effects of respiration or mechanical ventilation
Preload & preload responsiveness

Ventricular Function

- Left ventricular function
- Right ventricular function
- Depressed right ventricular function was further linked to more severely compromised left ventricular function.


Standard & TEE ECHO w/Doppler
Mechanical Ventilation

Increase RV outflow impedance, reduce ejection, increase RVEDV, tricuspid regurgitation

TEE: SVC diameter: the effect of venous return?

CVP may be misleading

Preload & Preload Responsiveness

Starling’s law is still operated.
If end diastolic volume (EDV) increased in response to volume loading, then stroke volume increased as well.
CVP, PCWP and their changes:
Did not respond with EDV, but
Provide a stable route for drug titration and fluid infusion

Physiological limitations

CVP
dysfunction
Pulmonary hypertension
LV dysfunction
Tamponade & hyperinflation
Intravascular volume expansion

PCWP
diastolic compliance
Pericardial restraint
Intrathoracic pressure
Heart rate
Mitrval regurgitation
Thermodilution Cardiac Output

- Mean (steady state) blood flow
- Functional significance of a specific cardiac output value
- Cardiac output varies to match the metabolic demands of the body

Mixed Venous Oximetry

- $SvO_2$ is the averaged end-capillary oxygen content (essential for $VO_2$ Fick)
- $SvO_2$ is a useful parameter of hemodynamic status in specific conditions
- If $SvO_2 < 60\%$ some capillary beds ischemic
- In sedated, paralyzed patient $SvO_2$ parallels CO
Adequate Oxygen delivery?

• $\text{SvO}_2$: mixed venous oxygen saturation
  
  $\text{C(a-v)O}_2$: arterial-venous oxygen content difference

• Lactate: the demand and need of the use of oxygen

Central Venous and Mixed Venous $\text{O}_2$ Saturation

• $\text{ScvO}_2$ on CVP monitor
• $\text{SvO}_2$ on PA catheter
• $\text{SvO}_2$ is a sensitive but non-specific measure of cardiovascular instability
  
  Although $\text{ScvO}_2$ tracked $\text{SvO}_2$, it tends to be $7 \pm 4\%$ higher.

Arterial Catheterization

• Directly measured arterial blood pressure
• Baroreceptor mechanisms defend arterial pressure over a wide range of flows
• Hypotension is always pathological
• Beat-to-beat variations in pulse pressure reflect changes in stroke volume rather than cardiac output
Pulmonary Arterial Catheterization

- Pressures reflect intrathoracic pressure
- Ventilation alters both pulmonary blood flow and vascular resistance
  - Resistance increases with increasing lung volume above resting lung volume (FRC)
  - Right ventricular output varies in phase with respiration-induced changes in venous return
- Spontaneous inspiration increases pulmonary blood flow
- Positive-pressure inspiration decreases pulmonary blood flow

Functional Hemodynamic Monitors

- Better monitors for preload responsiveness:
  - a significant correlation between the increase of cardiac index by fluid loading by pulse pressure variation and stroke volume variation
- Peripheral continuous cardiac output system (PiCCO): arterial pulse contour and transpulmonary thermal injection:
  - intrathoracic volume and extravascular lung water

PiCCO

Peripheral invasive continuous cardiac output
- Enables continuous hemodynamic monitoring using a femoral or axillary artery catheter and a central venous catheter.
- Using algorithms, PiCCO combines real-time continuous monitoring through pulse analysis with intermittent thermodilution measurement
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
  • CVP, ScvO₂ and PCWP, SvO₂ predict the adequacy of oxygen transport

The Truths in Hemodynamics

• Tachycardia is never a good thing.
• Hypotension is always pathological.
• There is no normal cardiac output.
• CVP is only elevated in disease.
• A higher mortality was shown in patients with right ventricular dysfunction and an increase of pulmonary vascular resistance.

The Truths in Hemodynamic Monitoring

• Monitors associate with inaccuracies, misconceptions and poorly documented benefits.
• A good understanding of the pathophysiological underpinnings for its effective application across patient groups is required.
• The goal of treatments based on monitoring is to restore the physiological homeostasis.