Cardiopulmonary Interactions

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• Two pumps in series in a chamber where pressure is changing
  • RV reservoir is outside thorax
    – Subject to atmospheric pressure
  • RV venous connections in thorax
    – Subject to thoracic pressure
• LV filling reservoir and LV lie in thorax
• Intrapleural pressure constantly changes
  – Fluctuations in intrathoracic pressure will affect output
    • Alter preload on R
    • Alter afterload on L
• Right and left ventricles share a septum
  – Changes in contractile state of one, affects the other
• Changes in lung volume alter caliber of alveolar vessels, affecting RV afterload
Today, we will step through each bit:
- Venous return and RA filling
- RV output
- Pulmonary Vascular resistance
- LV output
Spontaneous Breathing

Normal heart
Venous Return (Preload)

- Pressure for venous return
  - Mean systemic pressure – right atrial pressure
    - Usually about 5 mmHg and is determined by difference between extrathoracic and intrathoracic venous pressures

- Negative intrathoracic pressure increases caliber of intrathoracic veins
  - Increases preload to RA
  - Flow limited by the collapse of vena cavae at thoracic inlet
Venous Return (Preload)

- Increase in venous return during inspiration is countered by the increase in right atrial pressure, which thus decreases the gradient for further flow.
Right Ventricular Output

- Pumps blood through low resistance pulmonary vascular bed
- Blood flow through the lung to the left atrium sustained by
  - Pressure differential through the pulmonary artery, the downstream pressure in the pulmonary resistance vessels and the LA pressure.
    - Only requires 20-30 mmHg at rest
- RV well suited to this
  - Low pressure, high volume pump
  - In normal conditions, not too needed
    - Fontan physiology demonstrates this.
Increased preload can lead to increased RV systolic function, if pulmonary vascular resistance allows it.
Pulmonary Circulation

- Low resistance vascular bed
- Characteristics dependant on RV and LV function as well as alveolar pressures
- Low pressures, with Mean PA ~10-12mm Hg
  - Transpulmonary pressure gradient from PA to LA is <= 5 mmHg
- Vessels passively dilate to accommodate increases in CO, without increases in pressure
Pulmonary Vascular Bed

- Pulmonary vascular resistance is the afterload for the RV and thus determines preload for LV filling
  - Changes in PVR thus affect both ventricles
- Pulmonary circulation is wholly intrathoracic
  - SO, both RV afterload and LV preload are affected by changing intrathoracic pressure
• Two functional groups of pulmonary vessels
• Relative contribution of each to PVR depends on the pressure around the vessels
• Extra-alveolar vessels
  – Exposed to an extravascular pressure that reflects pleural pressure
  – Large pulmonary veins, arteries
  – Lie outside alveolar wall
• Intra-alveolar vessels
  – Exposed to an extravascular pressure that reflects alveolar pressure
  – Small arterioles, venules and capillaries
  – Reside in alveolar septa
• Intra-alveolar vessels
  – Zone I (PA > Pa > Pla)
    • Pa insufficient to open vessels, so no flow occurs
    • Occurs in apex of lung, or superior portion of supine lung
    • Proportion of lung that is zone 1, and thus dependant on alveolar pressure as primary determinant of resistance, makes sig contribution to PVR
      – More important as alveolar pressure increases, increasing Zone 1’s
Zone 2 (Pa>PA>Pla)
- Flow depends on difference between arterial and alveolar pressures
- Contribution to PVR increases with increasing PA and decreasing Pa (as in hypovolemia, etc.)
- Increased lung volume increases back pressure to RV, meaning RV must compensate with higher wall stress
  - Contributes to high afterload seen in asthma
  - Increased lung volume increases caliber of vessels, causing transient fall in LV preload
• **Zone 3 (Pa>Pla>PA)**
  – Flow is independent of alveolar pressures

• **These zones take on different import in the face of positive pressure**
  – PA often becomes high enough to influence distribution and relative amount of Zone 1/2 areas, and thus plays a major role in pulmonary vascular resistance
• Extra-alveolar vessels
  – Increased lung volume increases caliber of extra-alveolar vessels
    • Decreases pulmonary vascular resistance
    • Decreases RV afterload
      – Lowest afterload at FRC
  – Overdistention or loss of FRC result in increase in RV afterload
• The contributions of both alveolar and extra alveolar vessels add up to the total PVR
• PVR is lowest at FRC, and increases as lung volume increases or decreases from that point
Ventricular Interdependence

- Describes the process by which alterations in ventricular contraction and volume modify the function of the other ventricle
- RV is related to LV by:
  - Common muscle fibers that encircle both chambers
  - A common, deformable septum
  - Pericardium
- Their relationship affects both systolic and diastolic function
• Continuity of the muscle fibers means both ventricles pull towards a shared center of gravity, enhancing contraction

• Their shared septum acts as an anchor for the free RV wall, aiding force generation

• Right ventricular contraction is aided by left ventricular contraction
  – Increased LV volume increases pressures generated by RV
• LV, under normal conditions, operates on linear part of curve
• Large increases in RV volume during overload states, make the LV stiffer
  – Shift curve to left
    • A given change in volume thus requires more pressure
  – LV filling constrained by increases in RV volume

Fig 2. Ventricular compliance curve. Acute right ventricle volume overload shifts the left ventricular volume-pressure curve to the left.
Diastolic Ventricular Interaction

- Ventricular diastolic volumes contribute to cardiac performance
  - It is during diastole that the pressure or volume of either the LV or RV have the greatest impact on the other ventricle
• Acute RV distention shifts the septum toward the LV cavity
  – Decreases LV free wall dimension and compliance
  – So, despite same LV filling pressure, LVEDV decreases and so does CO
• Acute LV distension shifts the septum towards the RV cavity
  – Curve shifts upward and to the left
  – Decreases RV dimensions
• Intrathoracic pressure also affects diastolic function
  – High intrathoracic pressure prevents diastolic relaxation of both ventricles
    • The RV, however, is more susceptible, secondary to the lower intracavity pressures it generates
  – Evidenced by use of DSC in cases of increased cardiac edema following CPB
Left Ventricle

• Left ventricle is thick walled and elliptoid
  – Allows for an efficient creation of high pressures

• LV function is governed by
  – Contractility
  – Rate
  – Afterload
  – Preload
Effect of Spontaneous Ventilation on Ventricular Function

- Intrathoracic pressure and intrapericardial pressure decrease
  - Increasing venous return and RVEDV
    - And, depending on PVR, increased RV output
  - This may result in a transient shift of the septum to the left
    - Particularly when PVR, and thus, RV EDP, is high
  - If the RV dilates, LV diastolic compliance falls, decreasing LVEDV but not LVEDP
    - Decreases LV stroke volume and CO
    - Explains pulsus paradoxus..
• Preload for LV comes from pulmonary venous return and LA filling
  – Both reservoir and chamber lie inside the thorax, so
  – Both preload and afterload of LV are influenced by changes in intrathoracic pressure

• Arterial pressure falls during inspiration
  – Caused by decrease in LV stroke volume
• Reasons include
  – Pooling of blood in pulmonary circulation because of lung expansion
  – RV filling causing a decreased LV diastolic function
  – Negative intrathoracic pressure inhibits LV systolic function by increasing afterload
    • Very important in failing heart
    • More pronounced in cases of decreased lung compliance where intrathoracic pressure must become more negative

• Negative intrathoracic pressure thus adversely effects LV systolic and diastolic function
  – Large swings can result in acute pulmonary edema and LV failure
    • As in croup, epiglottitis and asthma
Pericardial Influence

- Pericardium limits acute changes in chamber size and influences cardiac function both directly and indirectly via ventricular interdependence.

- Effects of constraint
  - Diastole > systole
    - Development of increased intrapericardial pressure by way of effusion or the dilated, failing heart, interferes with diastolic filling.
      - Since coronary blood flow occurs during diastole, pericardial constraint can limit perfusion.
Mechanical Ventilation

Normal heart
Pulmonary Vascular Resistance

• Positive pressure alters PVR via changes in lung volume and alveolar O2 tension
• The diseased lung
  – infiltrates and edema lead to localized regions of alveolar hypoxia and collapse
  – Decreased FRC and hypoxic pulmonary vasoconstriction lead to increased PVR
  – Addition of PEEP may ameliorate these
    • Decreasing PVR and improving RV ejection fraction
• The normal lung
  – Greater compliance means more Paw is transmitted to vasculature and heart
  – Greater PEEP/Paw may lead to increased PVR and decreased RV function
  – Remember West Zone discussion?
    • It applies here, and for diseased lungs, too..
    • When PA>Pa, west zones 1 and 2 begin to predominate, increasing import of Paw as the factor that limits blood flow.
      – Happens in asthma, hypovolemia, ARDS with high pressures, etc…
Mechanical ventilation and Ventricular Function

- Following onset of positive pressure inspiration
  - Vena cava blood flow decreases first
  - Then pulmonary artery flow decreases
  - Then aortic blood flow falls
• Venous return decreases secondary to
  – Increase in RA pressure, secondary to increased intrathoracic pressure
  – Compression of vena cava during inspiratory increase in pleural pressure
• Inspiratory decrease in RV preload decreases RV output
  – Frank Starling
• This thus leads to decrease in LV filling and output
• Three other mechanisms participate in LV stroke volume variation
  – RV afterload increases during inspiration
    • Increase in alveolar pressure (pressure around capillaries) is greater than increase in pleural pressure (pressure surrounding the pulmonary arterial bed)
      – More west zone 1 and 2 versus 3, increasing PVR
  – Left Ventricular preload increases during inspiration
    • Increase in alveolar pressure>pleural pressure increase (pressure surrounding pulmonary venous bed)
    • Blood is squeezed to LA
• Left ventricular afterload decreases
  – Positive pleural pressure decreases transmural gradient

• So, during inspiration
  – LV stroke volume increases
    • LV preload increase
    • LV afterload decreases
  – RV stroke volume decreases
    • RV preload decreases
    • RV afterload increases
  – Because of long (~2 sec) pulmonary blood transit time the inspiratory decrease in RV output causes a decrease in LV filling and output a few heartbeats later
In hypovolemic and vasodilatory states, respiratory variations in arterial pressure and stroke volume are greater, and the expiratory decrease in LV output that follows is too.

- The venous system is more collapsible in hypovolemic conditions
- Inspiratory increase in RA pressure is greater, as RA is underfilled, and thus, more compliant
  - Decreased RV filling leads to decreased RV function, so...
- West Zone I and II states are more likely, and so, effect of inspiration on RV afterload is more marked
- RV and LV are more sensitive to preload when they operate on steep portion of frank starling curve.
**PEEP and LV function**

- **PEEP can decrease cardiac output**
  - Decreases preload and increases afterload of RV
    - Increases RV volume, which adversely affects LV compliance
    - Effects are more important with a compromised ventricle that is unable to generate enough pressure to overcome the higher afterload
      - TOF repair, Fontan (no RV)
    - Results in decreased LV EDV, thus decreasing stroke volume

- **Appropriate PEEP, however, allows FRC to be maintained at end expiration**
  - PVR thus falls
Wedge Pressures....

• In conditions such as ARDS, where RV afterload is already high, increasing PEEP can significantly decrease venous return
  – Leads to systemic hypotension
• RV afterload also increases septal shift and decreases LV EDV and output....
  – which leads to a misleading increase in wedge pressure, in that it is due to poor LV compliance and NOT poor function
• This situation may also occur in states associated with abrupt increases in RV volume.
• Underscores import of cautious use of volume until ventricular compliance is considered.
  – Echo can help with this one....

Table 1. Ventricular Interdependence

<table>
<thead>
<tr>
<th>Right Ventricular Pressure Overload State</th>
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<tbody>
<tr>
<td>• Acute pulmonary hypertension</td>
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<tr>
<td>• Pulmonary embolism</td>
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<tr>
<td>• Chronic pulmonary disease</td>
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<tr>
<td>• Mitral stenosis</td>
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</tbody>
</table>

<table>
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<tr>
<th>Right Ventricular Volume Overload States</th>
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</thead>
<tbody>
<tr>
<td>• Atrial septal defect</td>
</tr>
<tr>
<td>• Tricuspid insufficiency</td>
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<tr>
<td>• Pulmonary insufficiency</td>
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Spontaneous Ventilation

LV dysfunction
• In a normal individual, decreased intrathoracic pressure
  – Augments CO via increased RV preload
  – Diminish CO by increasing LV afterload
• Net result is a balance of these effects
• When LV function is normal, negative intrathoracic pressure results in little or nor hemodynamic change
• However, when negative intrathoracic pressures are large, even in those with normal function, LV ejection can be effected.

• A few instances can illustrate effect of negative intrathoracic pressure on the failing LV.
  – In those with MI, EKG patterns of injury improve once on PPV.
  – In those with LV failure, PPV cannot be weaned until LV is better inotropically supported.
• Increased afterload associated with negative intrathoracic pressure thus results in worsening heart failure

• In failure, with pulmonary congestion and edema, lung compliance falls, thus increasing the negative forces that must be generated, worsening LV function
  – PPV/CPAP fixes this....
Mechanical Ventilation

Cardiopulmonary Disease
RV

• PEEP often used in pulmonary failure as a method of maintaining FRC and improving oxygenation
  – There are, however, adverse effects of PEEP upon the RV

• The magnitude of the hemodynamic changes associated with increased PEEP depend on other cardiopulmonary factors
  – Volume status
  – Ventricular dysfunction
  – PVR
  – Lung compliance
• Indeed, even PEEP up to 25 cm H2O, in the face of decreased pulmonary compliance seen in ARDS, right ventricular ejection can be unhindered

• However, one can see a fall in CO and SV with high levels of PEEP
• Mechanisms behind this:
  – Decreased venous return
    • Most important mechanism
    • Can be overcome by increasing circulating volume, which increases RV preload, and help overcome increased PVR, too
  – Increased RV afterload
    • Via increased PVR caused by high Paw
  – Decreased LV compliance
    • Via ventricular interdependence
  – Decreased ventricular contractility
    • In the instance of underlying coronary ischemia
    • Otherwise PEEP helps with LV contractility
• There is a difference in the way PEEP affects ventricles with systolic versus diastolic dysfunction

• In systole, PEEP
  – reduces the venous return,
  – decreases the right and left ventricular preload,
  – thus improves mechanics in an overloaded ventricle

• In Diastole, PEEP
  – increases pericardial pressure
    • Potentially hurting diastolic function
  – reduces transmural pressure,
    • thus decreasing afterload.
• So PEEP
  – Helps systolic compromise by
    • Decreasing preload and afterload
  – May negatively effect diastolic compromise by
    • Compromising venous return
    • Decreasing LV EDV
    • Thus worsening filling and CO..
Bibliography

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