Normal Pericardial Physiology

- Fibrous pericardium
- Parietal layer of serous pericardium
- Visceral layer of serous pericardium (epicardium)
- Pericardial cavity
- Endocardium
- Myocardium (cardiac muscle)
- Trabeculae carneae
• Normal pericardium contains 20-30 ml of lymphoid fluid

• lubricating function that facilitates normal myocardial rotation and translation during each cardiac cycle in that the mesothelial layers of the pericardium produce a serous fluid that serves as a lubricant.

• secrete biochemical substances (e.g., prostacyclin) that may play a role in sympathetic neural regulation, coronary vascular tone and cardiac contractility.
Physiology of the normal pericardium

**Mechanical function**

1. Relatively inelastic cardiac envelope
   - limitation of excessive acute dilatation
   - protection against excessive ventriculoatrial regurgitation
   - maintenance of normal ventricular compliance
   - hydrostatic system
2. ventricular interaction; relative pericardial stiffness
3. maintenance of functionally optimal cardiac shape
4. Provision of closed chamber with subatmospheric pressure

**Membranous function**

**Ligmentous function**
Mechanical function => “Pericardial restraint”

- The compliance of the pericardium varies with the volume
- Initially the pericardium is supple and can expand with minimal increase in intrapericardial pressure with cardiac filling
- At higher volumes, the intrapericardial pressure rises, and impedes systemic and pulmonary venous return
- Volume of the fluid
- Rate of fluid accumulation
  - Slowly accumulation >1L
  - Rapid accumulation; 50-100 cc
Defense of the integrity of the Starling curve: Starling mechanism operates uniformly at all intraventricular pressures because of the presence of pericardium.
Membranous Function: Shielding the Heart

- Reduction of external friction due to heart movements
- Barrier to inflammation from contiguous structures
- Buttressing of thinner portions of the myocardium
  - Atria/ Right ventricle
- Defensive immunologic constituents in pericardial fluid
- Fibrinolytic activity in mesothelial lining
Respiratory-cardiac coupling

• Normal pericardial pressure is subatmospheric (negative)

Transmural pressure; actual chamber distending (filling) pressure

(Intracavitary pressure) - (Intrapericardial pressure)

Normally negative pericardial pressure produces a distending pressure higher than cavitary pressure
Pericardial pressure (Pp) recorded over the LV free wall using an orthogonal catheter.

Respirophasic variation

-6mmHg at the end inspiration
-3mmHg at the end expiration
as measured by a fluid filled non-balloon tipped catheter

For example)
Actual filling pressure spontaneously breathing patient
RA pressure 6mmHg
Intrapericardial pressure -6mmHg

6-(-6) mmHg during inspiration
Normal response of JVP and BP to Inspiration

**JVP**
Mean pressure falls. Wave forms become prominent

**Systemic BP**
Falls < 5-10mmhg

**Mechanism**

- Inspiratory transmission of negative Intra thoracic, intra pleural, Intra-pericardial and Intra cardiac pressures help the right heart chambers to suck the blood from extra-thoracic venous reservoir IVC*
  (*Which is uninfluenced by swinging Intra thoracic pressure Hence a positive gradient towards the right heart is created.)

- During inspiration there is relative pooling of blood volume in lungs.
  (Note: Since both pulmonary circuit and the left heart are intra-thoracic structures Inspiratory gradient is not established between them unlike right side.)

- Septal movement towards LV cavity interferes with LV stroke volume (Reverse Benhiem effect/Ventricular interdependence) Pericardial restraint also augments this.

- Importantly, direct transmission of negative intra-thoracic pressure into the central arteries also contribute to momentary reduction Aortic afterload & BP by few mmhg.

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Inspiration

- Increased venous return
- Right ventricle enlarges
- Septum pushes into LV

- Lungs expand
- Pulmonary vasculature expands
- More blood pools in lungs

Decreased filling of LV

Decreased stroke volume and decreased pulse amplitude
Spontaneous Respiration: Transtricuspid

Spontaneous Respiration: Transmitral
IPPV: Transtricuspid

IPPV: Transmitral
• The absolute values of these velocities affected by several physiologic variables
  age
  heart rate, rhythm
  preload, volume flow rate
  ventricular systolic function, diastolic function
  atrial contractile function.

The transmission of intrathoracic pressures to the intrapericardial structures appears blunted
  pericarditis or pericardial effusions severe enough to elicit tamponade physiology
TAMPONADE Physiology

Impairment of diastolic filling of the LV during inspiration, caused by abnormally elevated intrapericardial pressure.
With increasing intrapericardial pressure
  i.e., negative → positive

(Intracavitary pressure) - (Intrapericardial pressure)

local transmural gradient becomes negative
=> cavity collapse occurs
Relationship Between Intracardiac Filling Pressures and Intrapericardial Pressure and Cardiac Output in Cardiac Tamponade

Ventricular Interdependence

Inspiration

Expiration
Filling of the chamber is competitive

• respiratory changes in venous pressure difference alternately favoring RV and LV filling
• Filling of one ventricle and resultant shifting septum impede filling of the other ventricle
Large, chronic effusion are associated with excessive antero-posterior heart motion as well as counterclockwise rotation in the horizontal plane. Effusions can lead to cardiac translation within the pericardial space \textit{electrical alternans}.
Spontaneous Respiration: Tamponade – Transmitral

Spontaneous Respiration: Tamponade – Transtricuspid
Spontaneous Respiration: Tamponade – Transhepatic
IPPV: Tamponade - Transmitral
Percent change in Doppler Flow Velocity with Inspiration

<table>
<thead>
<tr>
<th></th>
<th>Pandian(^{81})</th>
<th>Leeman(^{83})</th>
<th>Appleton(^{84})</th>
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<tbody>
<tr>
<td><strong>Control</strong></td>
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<tr>
<td>Mitral</td>
<td>-10</td>
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<tr>
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<tr>
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<td>-3</td>
<td>-5(^{*}), -31(\dagger)</td>
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<tr>
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<td>21</td>
<td>32(^{*}), 74(\dagger)</td>
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</tr>
<tr>
<td>Mitral</td>
<td>-42</td>
<td>-35</td>
<td>E -43 ± 9%; A -28 ± 12%</td>
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<tr>
<td>Tricuspid</td>
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<td>80</td>
<td>E 85 ± 53%; A 58 ± 25%</td>
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<td>86</td>
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<tr>
<td>Left ventricular isovolumic relaxation time</td>
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<td>85 ± 14%</td>
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<tr>
<td>Left ventricular ejection time</td>
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<td>-21 ± 3%</td>
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