Echo Evaluation of Hemodynamics in Pericardial Disease

Natesa G. Pandian

Disclosure - None
Pre-Presentation Question 1

In cardiac tamponade, the pressure gradient between pulmonary venous system and left atrium

1. Increases during inspiration
2. Decreases during inspiration
3. Decreases during expiration
4. Shows no change
Pre-Presentation Question 2

In established constrictive pericarditis, septal bounce is recognized

1. In every heart beat
2. With breath hold
3. During regular breathing
4. Only during premature beats
For tamponade to occur, pericardial effusion should be at least

1. 200 ml
2. 500 ml
3. 1000 ml
4. None applicable
The following annular or septal velocity (tissue Doppler) is useful in recognizing constriction (in contrast to restrictive CMP):

1. $e' > 3-5$
2. $e' > 7$
3. $e' > 27$
The Pericardium

Visceral pericardium: single cell layer
Parietal: <2mm thick (collagen)
Pericardial fluid volume: ~ 50 cc
Normal Intrapericardial Pressure (mm Hg):

a) - 3
b) 0
c) + 2
d) + 5
The Pericardium

Intrapericardial pressure

end-inspiration - 6 mmHg

end-expiration - 3 mmHg
Pericardial Pr-Vol Relationship

Pressure

Volume Over Time

Limit of pericardial stretch
Pericardial Pr-Vol Relationship

- Pressure
- Volume Over Time

Chronic

Limit of pericardial stretch

Tamponade

Effusion
Pericardial Pr-Vol Relationship

- Pressure
- Volume Over Time

- Chronic
- Acute

Limit of pericardial stretch

Effusion → Tamponade
INTRAPERICARDIAL PRESSURE
INTRAPERICARDIAL PRESSURE

Diastolic Filling Pressures
INTRAPERICARDIAL PRESSURE

Diastolic Filling Pressures

Filling

Stroke Volume

Cardiac Output

Arterial Pressure

ADRENERGIC STIMULATION

SVR

HEART RATE

INOTROPY
INTRAPERICARDIAL PRESSURE

- Diastolic Filling Pressures
  - Filling
  - Stroke Volume
  - Cardiac Output
  - Arterial Pressure

- ADRENERGIC STIMULATION
  - SVR
  - HEART RATE
  - INOTROPY
At certain times during the cardiac cycle, intrapericardial pressure may exceed chamber pressures.
Case
RV diastolic collapse appears

a) Early RV diastole
b) Mid RV diastole
c) Late RV diastole
d) Early RV systole
e) Late RV systole
RV Diastolic Collapse
RV diastolic collapse could be absent in tamponade in the presence of:

a) RV Hypertrophy
b) Pulmonary hypertension
c) RV volume overload
d) COPD
e) All of the above
Case
RA collapse appears during:

a) Early RV diastole
b) Mid RV diastole
c) Late RV diastole
d) Late RV systole
Ventricular Interaction
Ventricular Interaction
During Inspiration
During Inspiration

- Intrathoracic pressure
- Pulmonary venous pressure
- Intrapericardial pressure
- Intracardiac fill pressures
Tamponade
Tamponade
Tamponade

- Pul Vein
- Ins
- Exp
- Peric
Dissociation between Intrathoracic and Intracardiac Pressures

Tricuspid Flow

Mitral Flow
Exp Velocity – Insp Velocity

Exp Velocity
Hep V/SVC Flow
• Insp Increase
• Exp Decrease
• Exp Increase in reversal flow

Pulm Vein Flow
• Insp Decrease
• Exp Increase
Hemodynamics behind Echo Findings, Symptoms and Signs

- Elevated intraperic & filling Pressures
- Intraperic pr rising above chamber pr
- Exaggerated RV-LV Interaction
- Dissociation Intrathor & Intracard Prs
- Decreased Cardiac Output
Typical Tamponade

- RV diastolic collapse
- RA collapse
- LA collapse
- Abnormal septal motion
- Resp change in LV, RV size
- Dilated IVC, Plethora
- Incr resp variation in flow

Mitral > 25-30%, Tricuspid > 35%
Case

Etiology

1. Viral
2. Post MI
3. Traumatic
4. Post-Qp
Case

Post-CABG
Mild Hypotension
Management

1) Percutaneous drainage
2) Surgical drainage off-pump
3) Surgical repair on pump
4) Fluids, Pressors
5) CT scan, MRI or Cath
Regional Effusion and Tamponade
Regional Tamponade

LV Diastolic Collapse
A post-op pt
Regional Tamponade

- Clinical signs not reliable
- Right sided signs may be absent
- Look for
  - LA collapse
  - LV diastolic collapse
  - Flow velocity changes
1. Hematoma
2. Fat pad
3. Pus
4. Malignant mass
Patient with Dyspnea

Tricuspid Flow
Patient with Dyspnea

Tricuspid Flow

COPD
Asymptomatic Subject

Mitral Flow
Asymptomatic Subject

Mitral Flow

Deep Breathing
Constrictive Pericarditis
Constrictive Pericarditis
Thick Pericardium Filling Dysfunction
Pericardial Thickness
Const Pericarditis: 2DE & M-mode

- Dense rigid pericardial shell
- Normal sized LV and RV
- Septal bounce, notch
- Rapid LV expansion with abrupt halt
- Premature pul valve opening
- IVC dilation and plethora
- Atria normal or mildly enlarged
Filling Dysfunction

- Elevated filling pressures
- Ventricular interdependence
- Dissociation between intrathoracvic and intracardiac pressures
Septal bounce
Septal Bounce, Diastolic notch
TVI: septal dynamics
Septal bounce
Respiratory Flow Variation

Mayo Publications
Mitral flow
Tricuspid flow
Tricuspid Flow Velocity

35% increase during inspiration
Mitral Flow Velocity

32% decrease during inspiration
Mitral Flow Velocity

E/A ratio: 2.2    Decel Time: 150 msec
Filling Dysfunction

Tricuspid Flow

Mitral Flow
Hepatic vein flow
Tissue Doppler

Lateral Annulus

Septal

E/e’  8

E/e’  7

Systolic  7 cm/s
## Diagnostic Value of Doppler Echocardiography

<table>
<thead>
<tr>
<th>Study</th>
<th>Respiratory variations</th>
<th>n</th>
<th>Sn</th>
<th>Sp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oh, Hatle et al.</td>
<td>+/- 25% in MV inflow velocity</td>
<td>28</td>
<td>88</td>
<td>67</td>
</tr>
<tr>
<td>Rajgopalan et al.</td>
<td>+/- 10% in MV inflow velocity</td>
<td>19</td>
<td>84</td>
<td>91</td>
</tr>
<tr>
<td>Klein et al.</td>
<td>PV systolic/diastolic flow ratio &gt;= -65%</td>
<td>14</td>
<td>86</td>
<td>94</td>
</tr>
<tr>
<td>Rajgopalan et al.</td>
<td>PV peak diastolic flow velocity &lt;= -18%</td>
<td>19</td>
<td>79</td>
<td>91</td>
</tr>
<tr>
<td>Von Bibra et al.</td>
<td>Hepatic veins ‘W’ wavepattern</td>
<td>13</td>
<td>68</td>
<td>100</td>
</tr>
</tbody>
</table>

Dal-Bianco & Sengupta, JASE. 2009; 22: 24-33
### MITRAL ANNULAR VELOCITY

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Age (yr)</th>
<th>E’ (cm/s)</th>
<th>Sn</th>
<th>Sp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Garcia et al</td>
<td>8</td>
<td>62 +/- 13</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Rajgopalan et al</td>
<td>19</td>
<td>56 +/- 13</td>
<td>&gt;=8</td>
<td>89</td>
<td>100</td>
</tr>
<tr>
<td>Ha et al</td>
<td>23</td>
<td>59 +/- 13</td>
<td>&gt;=8</td>
<td>95%</td>
<td>96%</td>
</tr>
<tr>
<td>Sohn et al</td>
<td>17</td>
<td>46 +/- 14</td>
<td>*</td>
<td>76%</td>
<td>82%</td>
</tr>
<tr>
<td>Sengupta et al</td>
<td>45</td>
<td>24 +/- 12</td>
<td>&gt;8</td>
<td>89%</td>
<td>73%</td>
</tr>
<tr>
<td>Choi et al</td>
<td>17</td>
<td>55 +/- 12</td>
<td>&gt;8</td>
<td>70%</td>
<td>100%</td>
</tr>
<tr>
<td>Sengupta et al</td>
<td>16</td>
<td>62 +/- 13</td>
<td>&gt;6.6</td>
<td>93%</td>
<td>93%</td>
</tr>
<tr>
<td>Sengupta et al</td>
<td>26</td>
<td>56 +/- 13</td>
<td>&gt;5”</td>
<td>92%</td>
<td>90%</td>
</tr>
</tbody>
</table>

Consider constriction if E’ >7cm/s
## Constrictive vs Restrictive CMP on 2DE

<table>
<thead>
<tr>
<th></th>
<th>Constriction</th>
<th>Restrictive CM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic function</strong></td>
<td>Normal</td>
<td>Initially NL</td>
</tr>
<tr>
<td><strong>Atrial size</strong></td>
<td>+/- mild bi-atrial enlargement</td>
<td>Bi-atrial enlargement</td>
</tr>
<tr>
<td><strong>Pericardial thickness</strong></td>
<td>+/- increased</td>
<td>NL</td>
</tr>
<tr>
<td><strong>Wall thickness</strong></td>
<td>NL</td>
<td>+/- “LVH”</td>
</tr>
<tr>
<td><strong>Septal motion</strong></td>
<td>Abnl- “bounce”</td>
<td>NL</td>
</tr>
<tr>
<td><strong>Myocardial texture</strong></td>
<td>NL</td>
<td>May be “sparkling” w/ amyloid</td>
</tr>
</tbody>
</table>
## Doppler

<table>
<thead>
<tr>
<th></th>
<th>Constriction</th>
<th>Restrictive CM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resp variation mitral inflow</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Pulmonary HTN</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Hepatic veins</td>
<td>Predominant forward flow-systolic</td>
<td>Predominant forward flow-diastolic</td>
</tr>
<tr>
<td></td>
<td>Increased diastolic flow reversal w/expiration</td>
<td>Increased diastolic flow w/inspiration</td>
</tr>
<tr>
<td>Septal mitral annular e’</td>
<td>Usually &gt;/=8cm/s; higher than lateral e’</td>
<td>Usually &lt;8cm/s; lower than lateral e’</td>
</tr>
</tbody>
</table>
## Strain Parameters

<table>
<thead>
<tr>
<th></th>
<th>Constriction</th>
<th>Restrictive CM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doppler based strain</td>
<td>Ventricular septal strain usually NL</td>
<td>Ventricular septal strain usually decr</td>
</tr>
<tr>
<td>Speckle tracking</td>
<td>Preserved longitudinal strain</td>
<td>Decreased longitudinal strain</td>
</tr>
<tr>
<td>Speckle tracking</td>
<td>Lateral long strain $&lt;_{\mathrm{septal}}$</td>
<td>Lateral longitudinal strain $&gt;_\mathrm{septal}$</td>
</tr>
<tr>
<td>Speckle tracking</td>
<td>Decreased circumferential strain</td>
<td>Preserved circumferential strain finally decr w/ progression of disease</td>
</tr>
</tbody>
</table>
Restrictive CMP
Constrictive Pericarditis

No myocardial involvement

Myocardial fibrosis
Doppler flow pattern early after pericardiectomy

Normal 40%
Restrictive 40%
Constrictive 20 %

43% abnormal flow at a mean FU of 20 months

Michele Senni et al, JACC April 1999
Doppler Flow Velocities

Limitations

False positive:
- COPD, RV infarction
- Obesity, massive PE

Marked increase in LA pressure

Irregular rhythm e.g. AF
Effusive – Constrictive Pericarditis
Constr Pericarditis
Atypical Scenarios

- Changing clinical spectrum
- Co-existing disease
- Effusive-constrictive pericarditis
Constrictive Pericarditis

- Chronic
- Subacute
- Acute

- Classic, Occult
- Effusive-Const
- Reversible

- Diffuse
- Localized
- Not thickened

- Pure Constriction
- Peri-Myocardial
- Disease
Occult Constrictive Pericarditis
Hypovolemic states
Give fluids and study

Transient Constrictive Pericarditis
Viral, Post-op

Medical Follow-Up
CMR Delayed Enhancement
Myocardial Fibrosis in Constrictive Pericarditis

- Direct subepicardial penetration
- Throttling of coronaries by scar tissue
- Subendocardial hypoperfusion
- Concomitant pathologic process

Levine H et al, Circulation 1973
Differential Diagnosis

Any disease leading to HF
Restrictive CMP
Nephrotic syndrome
Cirrhosis of liver
Tricuspid valve disease
SVC syndrome
Obstructive RH tumors
Pathoanatomical forms of constrictive pericarditis

(a) (b) (c) (d) (e) (f) (g)
Pericardial thickening
... a constant finding?

18% of patients with pathologically proven constrictive pericarditis have **normal** pericardial thickness.

1.5 mm in depth

3 mm of fat on exterior surface of pericardium

Main etiologies:
cardiac surgery (42%), infection (20%), thoracic irradiation (19%), previous MI (12%), idiopathic (12%).

Talreja et al, Circulation 2003
• Thickened pericardium does not mean it is constriction

• Pericardial pathology has varied distribution

• Constrictive physiology can develop with normal thickness pericardium
Surgical Results

- Operative mortality in 6-19%
- Low output syndrome in 14 - 28%
- Symptoms improvement in 90% but Symptom relief in 50%
- Five year survival is 74-87%
Predictors of poor surgical response

- Poor pre-op functional class
- Severe constriction
  - presence of unresectable calcification
Post-Presentation Question 1

In cardiac tamponade, the pressure gradient between pulmonary venous system and left atrium

1. Increases during inspiration
2. Decreases during inspiration
3. Decreases during expiration
4. Shows no change
Post-Presentation Question 2

In established constrictive pericarditis, septal bounce is recognized

1. In every heart beat
2. With breath hold
3. During regular breathing
4. Only during premature beats
For tamponade to occur, pericardial effusion should be at least

1. 200 ml
2. 500 ml
3. 1000 ml
4. None applicable
The following annular or septal velocity (tissue Doppler) is useful in recognizing constriction (in contrast to restrictive CMP)

1. $e' \geq 3-5$
2. $e' > 7$
3. $e' > 27$
Thank you!