Acute Aortic Regurgitation
Causes – aortic versus valve leaflets

Stelzer and Adams, Surgical Approach to Valvular Heart Disease
In Otto and Bonow, Valvular Heart Disease, 3rd Ed, 2009
Acute Aortic Regurgitation

Causes

• Aortic Dissection (Type A)
• Endocarditis
• Ruptured fenestration
• Blunt chest trauma
# Acute Aortic Regurgitation

## Hemodynamics and physical exam

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aortic regurgitation</strong></td>
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<tr>
<td><strong>Hemodynamics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac output</td>
<td>↓ N</td>
<td>N</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>N ↓</td>
<td>↑</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>↓ ↑</td>
<td>↑ N</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure</td>
<td>↑ ↑</td>
<td>↑ N</td>
</tr>
<tr>
<td>Left ventricular size</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Examination</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic murmur</td>
<td>Soft, early</td>
<td>Holodiastolic, decrescendo</td>
</tr>
<tr>
<td>S1</td>
<td>Soft</td>
<td>Normal</td>
</tr>
<tr>
<td>S2</td>
<td>Loud P2</td>
<td>Normal</td>
</tr>
<tr>
<td>S3</td>
<td>Present</td>
<td>Absent</td>
</tr>
</tbody>
</table>

N indicates normal; ↑, increased from normal; ↓, decreased from normal; CVP, central venous pressure.
63M bilateral ear pain and numbness in legs

Presented with several hour history of:

- Bilateral “burning” in his ears
- Throat tightness without SOB
- Bilateral leg numbness → severe cramping X 2 hrs

PMH –

- Hypertension.
- No other cardiac history.
- Evaluation for weight loss, anemia and hyponatremia
- Chest CT 2 wks prior (asc. aorta 4.3 cm)

Exam –

- Alert and in no distress
- BP 100/64, P 60, RR 12, Afebrile.
- 2/6 systolic murmur at base, 2/6 diastolic murmur LSB.
- Normal peripheral pulses.

Echocardiogram requested
63M bilateral ear pain and numbness in legs
Aortic dissection with acute severe AR

Dissection Flap (Type A)  Acute severe AR
63M bilateral ear pain and numbness in legs
Aortic dissection with acute severe AR
63M bilateral ear pain and numbness in legs
Aortic dissection with acute severe AR

Underlying valve disease (bicuspid valve)

Aortic dilation (lack of central coaptation)

Dissection into leaflet (flail leaflet)

Commisural displacement (asymmetric AR)
63M bilateral ear pain and numbness in legs
Aortic dissection with acute severe AR

Short Axis AR  Apical long-axis AR
Acute Aortic Regurgitation
Surgical Approach

Transferred to our medical center

Directly to OR

28 mm Dacron graft with valve resuspension

Stelzer and Adams, Surgical Approach to Valvular Heart Disease
In Otto and Bonow, Valvular Heart Disease, 3rd Ed, 2009
63M Aortic Dissection with valve resuspension
Intraoperative Echo
22F with acute hepatic and renal failure
1 month history of fatigue and weakness

**Clinical presentation**
- Transferred to Seattle from Montana with hepatic and renal failure.
- Treated for UTI with ceftriaxone for past 2 weeks.
- Worsening dyspnea, diarrhea and nausea/vomiting.
- No chest pain, fever or chills.
- History of VSD with murmur.

**Physical Examination**
- HR 95, BP 130/27 mm Hg, Temperature 36.3 C, RR 40.
- Alert and oriented.
- Neck – No JVD.
- Lungs -- clear
- Cardiac -- 3/6 systolic murmur.
- Abdomen -- soft and nontender
- Ext -- no edema.
22F with acute hepatic and renal failure
1 month history of fatigue and weakness

*Hospital course*

- Intubated for respiratory distress, developed DIC.
- No cause for hepatic or renal failure identified.

Echocardiogram requested
22F with acute hepatic and renal failure
1 month history of fatigue and weakness

Hospital course
- Intubated for respiratory distress, developed DIC.
- No cause for hepatic or renal failure identified.
22F with acute hepatic and renal failure
1 month history of fatigue and weakness
22F with acute hepatic and renal failure
1 month history of fatigue and weakness
Evaluation of AR Severity
Integrated Approach

Am Society Echo Guidelines 2003
Valve hemodynamics: Chronic vs acute aortic regurgitation
22F with acute hepatic and renal failure
1 month history of fatigue and weakness

Vena Contracta
CWD – density and $T^{\frac{1}{2}}$
Holo-diastolic flow reversal in the distal aorta
### Surgery for native valve endocarditis

**ESC Guidelines**

<table>
<thead>
<tr>
<th>Recommendations: Indications for surgery</th>
<th>Timing</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A - HEART FAILURE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic or mitral IE with severe acute regurgitation or valve obstruction causing refractory pulmonary oedema or cardiogenic shock</td>
<td>Emergency</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Aortic or mitral IE with fistula into a cardiac chamber or pericardium causing refractory pulmonary oedema or shock</td>
<td>Emergency</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Aortic or mitral IE with severe acute regurgitation or valve obstruction and persisting heart failure or echocardiographic signs of poor haemodynamic tolerance (early mitral closure or pulmonary hypertension)</td>
<td>Urgent</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Aortic or mitral IE with severe regurgitation and no HF</td>
<td>Elective</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td><strong>B - UNCONTROLLED INFECTION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Locally uncontrolled infection (abscess, false aneurysm, fistula, enlarging vegetation)</td>
<td>Urgent</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Persisting fever and positive blood cultures &gt; 7–10 days</td>
<td>Urgent</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Infection caused by fungi or multiresistant organisms</td>
<td>Urgent/elective</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td><strong>C - PREVENTION OF EMBOLISM</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic or mitral IE with large vegetations (&gt; 10 mm) following one or more embolic episodes despite appropriate antibiotic therapy</td>
<td>Urgent</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Aortic or mitral IE with large vegetations (&gt; 10 mm) and other predictors of complicated course (heart failure, persistent infection, abscess)</td>
<td>Urgent</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Isolated very large vegetations (&gt; 15 mm)*</td>
<td>Urgent</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>

Native Valve Endocarditis
Impact of early surgery (propensity score)

1555 pts with NVE, 46% with early surgery
Overall mortality 12% (surgery) vs 21% (medical Rx)

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Para-valve</th>
<th>Valve Perf.</th>
<th>Sys. Emboli</th>
<th>CVA</th>
<th>S. aureus</th>
<th>CHF</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>-5.9</td>
<td>-3.1</td>
<td>-6.2</td>
<td>-3.4</td>
<td>-4.5</td>
<td>-2.3</td>
<td>-8.3</td>
</tr>
<tr>
<td>Yes</td>
<td>-17.3</td>
<td>-3.5</td>
<td>-12.9</td>
<td>-13</td>
<td>-20.1</td>
<td>-3.4</td>
<td></td>
</tr>
</tbody>
</table>

Acute Aortic Regurgitation
Surgical Approach

Valve repair

Prosthetic valve

Tissue valve

Mechanical valve

Stelzer and Adams, Surgical Approach to Valvular Heart Disease
In Otto and Bonow, Valvular Heart Disease, 3rd Ed, 2009
Acute Aortic Regurgitation
Surgical Approach

With more extensive tissue destruction

- Aortic homograft
- Other complex repairs

Stelzer and Adams, Surgical Approach to Valvular Heart Disease
In Otto and Bonow, Valvular Heart Disease, 3rd Ed, 2009
22F with acute hepatic and renal failure
1 month history of fatigue and weakness

Blood cultures

Outside hospital:
Not obtained

Our hospital:
Already on antibiotics
All negative

Bioprosthetic AVR

PCR of valve tissue: 
Granulicattella elegans
Acute Aortic Regurgitation

Clinical Presentation and Physical Exam

Blood cultures

Aortic Dissection

Bacterial endocarditis

Medical therapy

Other causes

Echo (TTE)--Severe AR

Aortic root replacement
(valve resuspension or AVR)

Aortic valve surgery
(replacement or repair)
Acute Mitral Regurgitation

Causes

- Chordal rupture
- Endocarditis
- Ischemic
  - Papillary muscle rupture
  - Acute ischemia or infarction
Surgery for Acute Mitral Regurgitation
Outcomes after papillary muscle rupture

N = 54
70±8 yrs, 74% male
91% with
• Cardiogenic shock
• Pulmonary edema
• Cardiac arrest

Op. Mort 8.7%
• After 1990
• With CABG

### Acute Mitral Regurgitation

#### Hemodynamics and physical exam

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<thead>
<tr>
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<td><strong>Mitral regurgitation</strong></td>
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</tr>
<tr>
<td><strong>Hemodynamics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac output</td>
<td>↓</td>
<td>N</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>N ↓</td>
<td>N ↑</td>
</tr>
<tr>
<td>Left ventricular</td>
<td>↑ ↑</td>
<td>N</td>
</tr>
<tr>
<td>end-diastolic pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left atrial compliance</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td>Left ventricular size</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Examination</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Murmur</td>
<td>Soft, decrescendo</td>
<td>Holosystolic</td>
</tr>
<tr>
<td>S3</td>
<td>May be present</td>
<td>Absent</td>
</tr>
<tr>
<td>V waves of CVP</td>
<td>May be present</td>
<td>Absent</td>
</tr>
</tbody>
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N indicates normal; ↑, increased from normal; ↓, decreased from normal; CVP, central venous pressure.
54F with a history of mild MVP
Presented with dyspnea and hypoxia

- Flu-like symptoms while traveling
- Dyspnea on exertion
- Oxygen saturations in 80%
- Right middle and lower lobe pneumonia
54F with a history of mild MVP
Presented with dyspnea and hypoxia

- Intubated, broad spectrum antibiotics
- ICU care for 3 weeks for ARDS
- Outside echo showed normal LV function –
  (my review suggested flail mitral valve)
- “Stable” for transfer to our medical center –
  (by plane, >2000 km)
54F with a history of mild MVP
Presented with dyspnea and hypoxia

On arrival at our medical center (Saturday, 10 PM)

- Cardiogenic shock – IAPB placed but remained hypotensive, hypoxic on 100% oxygen, intubated and unresponsive with no urine output

- pH of 7.15, pCO2 75, pO2 199, bicarb 25

- Cardiac output 2.1 L/min, CI 1.2 L/min

Called CT Surgery and started Echo
54F with a history of mild MVP
Presented with dyspnea and hypoxia

Flail posterior leaflet
Mitral regurgitation
54F with a history of mild MVP
Presented with dyspnea and hypoxia

Flail posterior leaflet
Mitral regurgitation
Mitral Regurgitant Severity

MR color flow imaging

Appears to be > mild MR

Vena contracta

Central jet
No PISA seen
Jet area < 4.0 cm$^2$
or < 20% LA area
CWD not intense

Jet direction

> 0.7 cm

Central

< 0.3 cm

Eccentric

Severe MR

PISA RV and ROA

Pulsed Doppler RV and ROA

Mild MR
Valve hemodynamics: Chronic vs acute mitral regurgitation
54F with a history of mild MVP
Quantitation of MR Severity?

CW Doppler

PV systolic flow blunting

3.5 m/s
54F with a history of mild MVP

Quantitation of MR Severity?

\[ PISA = 1.3 \text{ cm}, \quad V_{\text{alias}} = 30 \text{ cm/s}, \quad ROA = 1.5 \text{ cm}^2 \]
Acute Mitral Regurgitation

Clinical Presentation and Physical Exam

Echo (TEE) – severe MR

Medical therapy + IABP

Ischemia or infarction

Blood cultures

Bacterial endocarditis

Spontaneous chordal rupture

Coronary angiography & consider revascularization

Urgent mitral valve surgery (repair or replacement)

Antibiotics
Acute Mitral Regurgitation
Surgical Approach

Mitral Valve Repair

McCarthy and Malaisrie, Mitral valve repair and replacement
In Otto and Bonow, Valvular Heart Disease, 3rd Ed, 2009
Surgery for Acute Mitral Regurgitation
Long term post-operative outcomes

279 pts -- 62±14 yrs; 62% female; cardiogenic shock 66%

Survival

30 day mortality 22.5%

Predictors of long term outcome
- Acute MI
- CAD

54F with a acute severe MR
Long term outcome

- Mechanical MVR.
- Immediate improvement in BP, CO, renal function and mental status.
- Stormy post-operative course (AF)
- Now (5 yrs later) active, healthy woman; NSR; normal LV-EF; high profile career.
Acute Valve Regurgitation

CONCLUSIONS

• Acute valve regurgitation often is not recognized.

• Echo provides definitive diagnostic data.

• Prompt surgical intervention may be life-saving.

• Medical “stabilization” often is not possible.

• An IABP is recommended for acute MR.

• Education of primary care MDs is needed.