Aortic regurgitation. Physiopathology

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Acute AR

**Etiology:**
Infective endocarditis, aortic dissection, trauma, post balloon valvuloplasty, post surgical commissurotomy, idiopathic fenestration or cusp rupture

**Pathophysiology:**
Sudden large regurgitant volume is imposed on a normal size LV

Rapid increase in LVEDP and LAP
Equalization of aortic and LV pressures in diastole

Pulmonary edema, cardiogenic shock
Diminished myocardial perfusion pressure
In the subendocardium
<table>
<thead>
<tr>
<th></th>
<th>Chronic AR compensated</th>
<th>Chronic AR decompensated</th>
<th>Acute AR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physiology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV Volume</td>
<td>Increased (ESD&lt;55)</td>
<td>Increased (ESD&gt;55)</td>
<td>Normal</td>
</tr>
<tr>
<td>Ejection Fraction</td>
<td>Normal (&gt;55%)</td>
<td>Normal or decreased</td>
<td>Normal or decreased</td>
</tr>
<tr>
<td>LV EDP</td>
<td>Normal</td>
<td>Normal</td>
<td>Increased</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
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<tr>
<td><strong>Physical exam</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic murmur</td>
<td>Holodiastolic,</td>
<td>Holodiastolic</td>
<td>Early diastole</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Wide</td>
<td>Wide</td>
<td>Normal</td>
</tr>
<tr>
<td>Peripheral signs</td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Clinical Presentation</strong></td>
<td></td>
<td>Gradual onset of</td>
<td>Sudden onset,pulmonary</td>
</tr>
<tr>
<td></td>
<td>Asymptomatic</td>
<td>symptoms,</td>
<td>edema</td>
</tr>
</tbody>
</table>
Acute AR in aortic dissection
**Acute AR in Aortic Dissection.**

Movsowitz HD, et al. JACC 2000;36:884

<table>
<thead>
<tr>
<th>Degree of Aortic Regurgitation</th>
<th>None/Trace/Mild (n = 27)</th>
<th>Moderate/Severe (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incomplete leaflet closure</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Aortic leaflet prolapse</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Intimal flap prolapse</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Bicuspid aortic valve</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Leaflet thickening</td>
<td>17</td>
<td>5</td>
</tr>
</tbody>
</table>
Acute AR in IE
Chronic AR. Pathophysiology

VOLUME-PRESSURE OVERLOAD

Increased stroke volume  
Ventricular dilatation  
Increase in LV dimension and systolic pressure  
Excessive afterload: depression of contractility

Increased afterload  
Increase in wall thickness  
Wall stress maintained  
Wall thickening fails to keep pace  
Increase in wall stress
<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk</td>
<td>Patients with risk factors for the development of VHD</td>
</tr>
<tr>
<td>B</td>
<td>Progressive</td>
<td>Patients with progressive VHD (mild-to-moderate severity and asymptomatic)</td>
</tr>
<tr>
<td>C</td>
<td>Asymptomatic severe</td>
<td>Asymptomatic patients who have reached the criteria for severe VHD&lt;br&gt;C1: Asymptomatic patients with severe VHD in whom the left or right ventricle remains compensated&lt;br&gt;C2: Asymptomatic patients who have severe VHD, with decompensation of the left or right ventricle</td>
</tr>
<tr>
<td>D</td>
<td>Symptomatic severe</td>
<td>Patients who have developed symptoms as a result of VHD</td>
</tr>
</tbody>
</table>
CHRONIC AR

Stage A: At risk of AR

Stage B: Mild- moderate AR

Stage C1: Compensated severe AR (balanced preload/hypertrophy/afterload)
FE>50%, LVESD < 50 mm

Stage C2: Decompensated severe AR (progressive LV enlargement, decline EF)
FE<50% or LVESD >50 mm or >25 mm/m2

Stage D: Irreversible LV dysfunction
Chronic AR. Stages A and B
CHRONIC AR

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Stage C1: Compensated severe AR (balanced preload/hypertrophy/afterload)
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FE<50% or LVESD >50 mm or >25 mm/m²

Stage D: Irreversible LV dysfunction

SYMPTOMS

CHRONIC AR
Chronic AR. Stage C1
Chronic AR. Stage C1
Chronic AR Stage C1
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(progressive LV enlargement, decline EF)
FE<50% or LVESD >50 mm or >25 mm/m2

Stage D: Irreversible LV dysfunction
Chronic AR. Stage C2
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Stage C1: Compensated severe AR (balanced preload/hypertrophy/afterload)
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Stage D: Irreversible LV dysfunction
Final comments

• The pathophysiology of both acute and chronic AR is well established

• Clinical and Echo-Doppler evaluation are needed for diagnosis and follow up

• Knowledge of pathophysiology is crucial for the correct management of patients with AR