VALVULAR DISORDERS:
AORTIC AND MITRAL VALVE

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AORTIC VALVE STENOSIS

- Etiology
- Severity: Follow the Guidelines
- Quantification
# Applying Classification of Recommendations and Level of Evidence

**“SIZE of TREATMENT EFFECT”**

<table>
<thead>
<tr>
<th>Level A</th>
<th>Level B</th>
<th>Level C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Multiple (3-5) population risk strata evaluated</strong></td>
<td><strong>Limited (2-3) population risk strata evaluated</strong></td>
<td><strong>Very limited (1-2) population risk strata evaluated</strong></td>
</tr>
<tr>
<td>- Recommend that procedure or treatment is useful/effective</td>
<td>- Recommend that procedure or treatment is useful/effective</td>
<td>- Recommend that procedure or treatment is useful/effective</td>
</tr>
<tr>
<td>- Sufficient evidence from multiple randomized trials or meta-analyses</td>
<td>- Limited evidence from single randomized trial or non-randomized studies</td>
<td>- Only expert opinion, case studies, or standard-of-care</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class I</th>
<th>Class IIa</th>
<th>Class IIb</th>
<th>Class III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benefit &gt;&gt; Risk</td>
<td>Benefit &gt;&gt; Risk</td>
<td>Benefit &gt; Risk</td>
<td>Risk &gt; Benefit</td>
</tr>
<tr>
<td>No additional studies needed</td>
<td>Additional studies with focused objectives needed</td>
<td>Additional studies with broad objectives needed; Additional registry data would be helpful</td>
<td>No additional studies needed</td>
</tr>
<tr>
<td>Procedure/Treatment SHOULD be performed/administered</td>
<td>IT IS REASONABLE to perform procedure/administer treatment</td>
<td>IT IS NOT UNREASONABLE to perform procedure/administer treatment</td>
<td>Procedure/Treatment should NOT be performed/administered since it is NOT helpful and may be harmful</td>
</tr>
</tbody>
</table>

- **Level A**
  - Recommendation that procedure or treatment is useful/effective
  - Sufficient evidence from multiple randomized trials or meta-analyses

- **Level B**
  - Recommendation that procedure or treatment is useful/effective
  - Limited evidence from single randomized trial or non-randomized studies

- **Level C**
  - Recommendation that procedure or treatment is useful/effective
  - Only expert opinion, case studies, or standard-of-care
Aortic Valve Disease
Module: Aortic Valve Disease

Aortic Stenosis – Background
Module: Aortic Valve Disease

Aortic Stenosis – Background

- Most common valvular lesion in USA
- Etiology
  - Bicuspid aortic valve (1%-2% of general population)
  - Rheumatic (almost always requires MV involvement)
  - Degenerative-calcific (age-related)
ETIOLOGY: BICUSPID AORTIC VALVE
Bicuspid Aortic Stenosis

- Bicuspid aortic valve – 1%-2% of population
  - Most commonly fusion of right-left cusps
- Majority never develop stenosis
  - Those that do – younger age of presentation than degenerative (40-60)
- Associated with coarctation and dissection
  - Especially in younger patient with hypertension
ETIOLOGY: UNICUSPID AORTIC VALVE
RARE VALVE DISEASE
DEGENERATIVE CALCIFIC AORTIC VALVE:
THE MOST COMMON CAUSE OF AORTIC STENOSIS IN DEVELOPED COUNTRIES
Aortic Stenosis – Rheumatic

- Fusion of commissures
- Mitral valve involvement
ETIOLOGY: RHEUMATIC
OTHERS
LVOT OBSTRUCTION-HOCM
DISC RETE SUBVALVULAR MEMBRANE
## Aortic Stenosis Quantification Methods

<table>
<thead>
<tr>
<th>Portion of Echocardiogram</th>
<th>Characteristics and Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>2-Dimensional Exam</strong></td>
<td>Valvular thickening, calcification and restricted leaflet motion</td>
</tr>
<tr>
<td></td>
<td>(commissural fusion if inflammatory)</td>
</tr>
<tr>
<td></td>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td></td>
<td>Poststenotic dilation of ascending aorta</td>
</tr>
<tr>
<td></td>
<td>AV area by planimetry using TEE</td>
</tr>
<tr>
<td><strong>Doppler Exam</strong></td>
<td>Maximal and mean transvalvular pressure gradients (apical 4ch or right parasternal)</td>
</tr>
<tr>
<td></td>
<td>AV area by continuity equation</td>
</tr>
</tbody>
</table>
Aortic Valve Planimetry (TTE parasternal short-axis view)
Aortic Stenosis – Valve Area – Echo

- Top – planimetry
- Bottom – continuity equation – $a_1v_1 = a_2v_2$
THE CONTINUITY EQUATION

**Continuity Equation**

\[ A_{AV} \cdot V_{AV} = A_{LVOT} \cdot V_{LVOT} \]

\[ A_{AV} = 0.785 \times (D)^2 \]

**Left Ventricular Outflow Tract Area**

\[ A_L = \pi d^2 = \pi \left( \frac{d}{2} \right)^2 \]

\[ A_L = d^2 \times 0.786 \]
ERRORS IN VALVE AREA ESTIMATION

- LVOT DIAMETER
- MISALIGNMENT: UNDERESTIMATION OF THE GRADIENT
- MEASURING AN ECCENTRIC MR JET: OVERESTIMATION
- CE ASSUMES THAT THE LVOT DIAMETER IS CIRCULAR; IT IS ELLIPTICAL

USE THE DIMENSIONLESS VELOCITY RATIO

\[( \text{DVR} ) = \frac{\text{PEAK LVOT VEL}(VTI)}{\text{PK AV VEL}(VTI)} \]
CWD USING THE PEDOFF TRANSDUCER FROM THE APICAL AND SSN WINDOWS- INSTANTANEOUS GRADIENT
DISCREPANCIES WITH CARDIAC CATHETERIZATION
Module: Aortic Valve Disease

Aortic Stenosis – Valve Area – Cath

- Gorlin: \[ \text{Aortic Valve Area} = \frac{\text{Cardiac Output}}{\text{Heart rate} \cdot \text{Systolic ejection period} \cdot 44.3 \cdot \sqrt{\text{Gradient}}} \]

- Hakki: \[ \text{Aortic Valve Area} = \frac{\text{Cardiac Output}}{\sqrt{\text{Gradient}}} \]
<table>
<thead>
<tr>
<th>Variable</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jet velocity (m/sec)</td>
<td>&lt;3</td>
<td>3-4</td>
<td>&gt;4</td>
</tr>
<tr>
<td>Mean gradient (mmHg)</td>
<td>&lt;25</td>
<td>25-40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
<td>&gt;1.5</td>
<td>1-1.5</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Valve area indexed (cm²/m²)</td>
<td>NA</td>
<td>NA</td>
<td>&lt;0.6</td>
</tr>
</tbody>
</table>
52 year old male: Class II dyspnea
LVOT diam 2.0 cm
LVOT TVI 25 cm

AV TVI 98 cm
Area 1 \times TVI_1 = Area 2 \times TVI_2

0.785 \times (2.0)^2 \times 25 = AVA \times 98

AVA = 78.8 / 98

= 0.8\text{cm}^2
Question 1

Which of the following Doppler parameters may predict event-free survival in patients with AS?

A. E/E’ ratio
B. Aortic valve peak jet velocity
C. Rapid E wave deceleration time
D. Reversal of systolic PV flow
Event-Free Survival

Predictors
- V_max
- ΔV_max
- NYHA Class

ERRORS IN VALVE AREA ESTIMATION

LEFT VENTRICULAR DYSFUNCTION

LOW FLOW LOW GRADIENT: DEFINITION
1. EOA < 1 CM  2. EF < 40%  3. MEAN PG < 30 MM HG

USE DOBUTAMINE STRESS ECHO FOR A BETTER ESTIMATION

LEFT VENTRICULAR HYPERTROPHY WITH DIASTOLIC DYSFUNCTION

LOW SV/GRADIENT CAN UNDERESTIMATE SEVERITY—RECENTLY
TERMED PARADOXICAL LOW FLOW, LOW GRADIENT AS ASSOCIATED
WITH HIGHER AFTERLOAD AND REDUCED SURVIVAL

SYSTEMIC HYPERTENSION

AORTIC REGURGITATION

HIGH CARDIAC OUTPUT—CHECK FOR EARLY PEAKING OF THE JET

MITRAL REGURGITATION—SEVERE MR—UNDERESTIMATION OF AS SEVERITY
Aortic Stenosis

Low cardiac output
Low pressure gradient

Baseline Doppler hemodynamics

Dobutamine stress

↑↑ Gradient
↓↓ AV area
Severe AS

Left→Right Gradient
↑ AV area
Not severe AS
Case. Question 2

- 54 year old man: progressive DOE
- NYHA class III/IV
- Edema, orthopnea, PND
- Exam: II/VI late-peaking SEM
LVOT TVI 13 cm  SV 59cc
Mean gradient 27 mmHg AVA 0.93 cm²

LVOT TVI 17cm  SV 77 cc
Mean gradient 38 mmHg AVA 0.89 cm²
Question 2: What to Advise?

91%  A. Aortic valve replacement
0%   B. Biventricular pacemaker
1%   C. Heart transplant
8%   D. Continue medical therapy
Low Gradient AS

*Take home points*

- EF < 40%; MG < 30 mmHg; AVA < 1 cm²
- Dobutamine stress echo: best way to assess contractile reserve and distinguish between true and pseudo AS
- AVR is the best option
Low Gradient AS

- In low-flow states (LV dysfunction)
  - Aortic stenosis may be the cause
    - Due to afterload mismatch
    - Resultant low gradient
  - Low flow may lead to decreased valve excursion
    - Low gradient because low-flow and normal valve
    - Appearance of stenosis – “pseudostenosis”

- Need to determine whether low output/low gradient is due to valve or to myocardium

- To differentiate – dobutamine
  - Start at 5 mcg/kg/min – titrate up
  - If increase in gradient – then valve is culprit and pt will likely benefit from AVR

Module: Aortic Valve Disease

Low Gradient AS cont’d

- Left: increase in gradient with dobutamine
  - At AVR – severe AS
- Right: increase in CO, not in gradient
  - At AVR – minimal AS

DOBUTAMINE STRESS TEST

According to recent consensus statement, three reliable conclusions can be drawn of a dobutamine stress echo:

1. An AVA at peak dobutamine dose of over 1 cm² excludes severe disease.

2. If at any dose, the aortic velocity exceeds 4 m/sec or mean gradient exceeds 40 mm Hg at any stage, the AS is severe as long as the calculated AVA is less than 1 cm².

3. If the stroke volume or LVEF does not increase by 20%, this signifies a lack of contractile reserve that suggests poor surgical and long-term outcomes.
Module: Aortic Valve Disease

AS – Pathophysiology

- Decrease in valve area to < 2 cm² produces pressure overload on LV
- Concentric hypertrophy is compensatory
- Hypertrophied myocardium
  - Decreased coronary flow reserve
    - Leads to diastolic and systolic dysfunction
      - Causing symptoms
        - Angina – coronary insufficiency
        - Syncope – decreased cardiac output (fixed stenosis)
        - CHF – ventricular dysfunction
Aortic Stenosis

- Natural history
  - Asymptomatic disease – no increased mortality
  - Symptomatic disease – limited life expectancy
    - Angina – 5 years
    - Syncope – 3 years
    - CHF – 1-2 years

Question: A 76 y.o. male presents as a referral for aortic stenosis and a recent syncopal event. He is otherwise asymptomatic and has no other medical complaints. Exam is unremarkable except for a mid-late peaking 3/6 SEM at the right sternal border. S2 is present. Echocardiogram demonstrates an aortic valve area of 0.8 cm² with a peak velocity of 4.2 m/s and mean gradient of 44 mmHg. What is the next step in management?

A. Repeat echocardiogram in one year
B. Dobutamine stress test
C. Cardiac catheterization with surgical AVR
D. Surgical AVR
Aortic Stenosis – F/U

- Echo indicated on initial evaluation
- After initial evaluation
  - Change in symptoms
  - Asymptomatic disease
    - Yearly for severe AS
    - Every 2 years for moderate AS
    - Every 5 years for mild AS
- Expectation
  - Jet velocity increases by 0.3 m/s per year
  - Gradient increases by 7 mmHg per year
  - Valve area decreases by 0.1 cm² per year
Aortic Stenosis – Treatment

- Medical
  - Statins – potentially slow progression of AS
    - Retrospective Data for Delayed Progression and Decreased Aortic Valve Calcification
    - SALTIRE*
      - No clinical, echo, CT benefit to statin
  - ACE inhibitor
    - Benefit possibly mediated by drug effects on inflammation

*NEJM, 2005
Module: Aortic Valve Disease

Aortic Stenosis – Treatment cont’d

- Surgery
  - Aortic valve replacement
    - Pros
      - 2%-4% mortality for stand-alone AVR
      - Excellent long-term outcomes
    - Cons
      - Mortality approaches 1.5%-20% in high risk subsets
      - Many patients with severe, symptomatic disease do not undergo surgery
        - Patient and physician factors
Module: Aortic Valve Disease

Aortic Stenosis – Treatment

Question: A 63 y.o. female with history of a heart murmur is referred after an echocardiogram by her primary care physician demonstrated severe aortic stenosis. She is asymptomatic and exam is unremarkable except for a late peaking 3/6 SEM at the right sternal border. S2 is soft but present. Echocardiogram demonstrates normal LV function, aortic valve area of 0.8 cm² with a peak velocity of 3.7 m/s and mean gradient of 32 mmHg. What is the next step in management?

A. Repeat echocardiogram in one year
B. Dobutamine stress test
C. Cardiac catheterization
D. Surgical AVR
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A. Repeat echocardiogram in one year  
B. Dobutamine stress test  
C. Cardiac catheterization  
D. Surgical AVR
Module: Aortic Valve Disease

AS – Guidelines

Severe Aortic Stenosis
Vmax greater than 4 m/s
AVA less than 1.0 cm²
Mean gradient > 40 mm Hg

Undergoing CABG or other
Heart surgery?

Yes

No

Symptoms?

Yes

Espironolactone

No

Exercise test

Normal

LV ejection fraction

Less than 0.50

Severe valve calcification,
rapid progression, and/or
expected delay in surgery

Yes

Aortic valve replacement

Preoperative coronary angiography

Symptoms

BP

Clinical follow-up, patient education,
risk factor modification, annual echo

Prep	Adv	Adv	Adv	Adv
	Adv	Adv	Adv	Adv
	Adv	Adv	Adv	Adv
	Adv	Adv	Adv	Adv
AVR – Surgical Mortality

In-Hospital Mortality (Percent)

Year


AVR + CABG

AVR

Society of Thoracic Surgeons Database, 2005
Adapted from Bonow RO TCT 2006
Percutaneous Therapy
PARTNER Cohort B
All Cause Mortality at one year

- Standard Rx
- TAVI

HR [95% CI] = \( \frac{\Delta \text{ at 1 yr}}{\text{NNT}} = 5.0 \text{ pts} \)

P (log rank) < 0.0001

Numbers at risk:

<table>
<thead>
<tr>
<th>TAVI</th>
<th>179</th>
<th>138</th>
<th>122</th>
<th>67</th>
<th>26</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard Rx</td>
<td>179</td>
<td>121</td>
<td>88</td>
<td>41</td>
<td>12</td>
</tr>
</tbody>
</table>

Leon et al. NEJM 2010 363 1597
PARTNER – Cohort A
Primary Endpoint - All-Cause Mortality at 1 Year

HR [95% CI] = 0.93 [0.71, 1.22]
P (log rank) = 0.62
TAVR – “approved” indications

- Severe aortic stenosis – Guideline Based
- STS greater than 8%
  - Or mortality estimate > 15% in opinion of cardiologist and two cardiac surgeons
- Life expectancy greater than 1 year
  - Outside of Aortic stenosis
- Requires multidisciplinary heart team
- Inoperable transapical technically “off label”
Question: A 48 y.o. female with no significant past history is referred for evaluation of a heart murmur. She is asymptomatic. On examination, blood pressure is 140/60 and she has a 2/6 systolic ejection murmur and a holodiastolic murmur heart best along the right sternal border. Echocardiography demonstrates a dilated LV with EDD 60 mm and ESD 45 mm, normal LV function a normal root and a bicuspid, nonstenotic aortic valve with severe insufficiency. The most appropriate management strategy is:

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Aortic Regurgitation

Etiology

**VALVE**
- BAV DISEASE
- RHEUMATIC
- IE
- MYXOMATOUS
- APLA
- TRAUMA

**ROOT**
- CT DISORDER
- DISSECTION
- IE
- AORTITIS
- HTN
- OTHER (Congenital)
AORTIC REGURGITATION ETIOLOGIES
Aortic Insufficiency

- **Etiology**
  - Formerly, syphilis was most common cause
  - Today
    - Aortic root dilatation
    - Bicuspid valve
    - Calcific degeneration
    - Dissection
    - Rheumatic heart disease
    - Endocarditis
    - Connective tissue disease
AI – Aortic Root Dilatation

- Dilated root
- Malcoaptation of aortic valve leaflets
- Resultant aortic insufficiency
Question 3

EDD: 76 mm; ESD: 51 mm
Question 3

Regurgitant volume: 140 mL; ERO: 0.62 cm²
Question 3: Severity of AR?
1. Moderate
2. Moderate-Severe
3. Severe

TVI SSN: 16 cm

$T_{\frac{1}{2}}$: 200 msec
Summary Severe AR: TTE

- Color jet width > 60%
- Vena contracta > 6 mm
- $T_{1/2}$ AR CW < 200 msec
- TVI flow reversal (SSN): 13-15 cm
- RV > 60 mL
- $ERO \geq 30 \text{ mm}^2$
Pressure Half Time

>500 ms  <200 ms

Mild  Severe

500 ms  119 ms
AORTIC REGURGITATION
Module: Aortic Valve Disease

AI – Severity

- Top – vena contracta
  - Mild: <0.3; severe: >0.6
- Bottom – pressure half-time
  - Mild: >450; severe <250
AORTIC REGURGITATION
FLOW REVERSAL
Root Aneurysm with Bicuspid Ao Valve
AORTIC REGURGITATION ETIOLOGIES
AORTIC REGURGITATION
ETIOLOGIES
AORTIC REGURGITATION
Severe AI – Echocardiography

- Jet dimension >60% LVOT diameter
  - May be misleading on eccentric jets
- Flow reversal in proximal descending aorta
  - >0.6 m/s initially, 0.2 m/s holodiastolic
- Regurgitant volume >60 ml
- Regurgitant fraction >55%
- Look for supportive signs
  - Eccentric LVH
  - LV Dilation
- Catheterization if discrepancy
### Application of Specific and Supportive Signs, and Quantitative Parameters in the Grading of Aortic Regurgitation Severity

<table>
<thead>
<tr>
<th>Specific signs for AR Severity</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Central jet, width &lt;25% of LVOT&lt;sup&gt;T&lt;/sup&gt;</td>
<td>• Signs of AR-mild present but no criteria for severe AR</td>
<td>• Central jet, width ≥65% of LVOT&lt;sup&gt;T&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>• Vena contracta ≤ 0.3 cm²</td>
<td></td>
<td>• Vena contracta</td>
<td></td>
</tr>
<tr>
<td>• No or brief early diastolic flow reversal in descending aorta</td>
<td></td>
<td>• &gt;0.8 cm²</td>
<td></td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>Supportive signs</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pressure half-time &gt;500 ms</td>
<td>• Intermediate values</td>
<td>• Pressure half-time &lt;200 ms</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Quantitative parameters&lt;sup&gt;P&lt;/sup&gt;</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>R Vol, ml/beat</td>
<td>&lt;30</td>
<td>30-44</td>
<td>45-59</td>
</tr>
<tr>
<td>RF, %</td>
<td>&lt;30</td>
<td>30-39</td>
<td>40-49</td>
</tr>
<tr>
<td>ERA0, cm²</td>
<td>&lt;0.10</td>
<td>0.100-0.19</td>
<td>0.20-0.29</td>
</tr>
</tbody>
</table>
Acute AI

- Sudden increase in LV end diastolic volume and pressure
  - Murmur may be short or inaudible
  - Physical exam findings diminished or even absent
- Insufficient time for LV to dilate
  - Leads to tachycardia (compensatory) and pulmonary edema
- On echo – may see diastolic mitral regurgitation

Bonow RO et al. ACC/AHA Guidelines on Valvular Disease 2006
Module: Aortic Valve Disease

Chronic AI

- Increased end diastolic volume, increased preload
- Compensatory LV dilatation and hypertrophy
  - Produces increase in afterload
  - Thus, chronic AI is a state of both pressure and volume overload
    - This compensated state may persist for decades
      - Systolic performance remains normal
      - Stroke volume is increased – widened pulse pressure
AI – Natural History

- Asymptomatic patients with normal LVSF
  - Progress to Sx or LV dysfunction – 6%/yr
  - Sudden death – 0.2%/yr

AI – Natural History

- Asymptomatic patients with normal LVSF
  - Progress to Sx or LV dysfunction – 6%/yr
  - Sudden death – 0.2%/yr
- Asymptomatic patients with LV dysfunction
  - Symptoms – 25%/yr
- Symptoms
  - Death – 10%/yr

Chronic AI – Treatment

- Echocardiography
  - Initial evaluation
  - Yearly for severe disease or root dilatation
- Medical therapy
  - Long-term vasodilator therapy
    - Retrospective data exist for both nifedipine and ACE-I
      - Slows LV dilatation
      - Reduces LV dysfunction with eventual surgery
      - Recently called into question
Module: Aortic Valve Disease

AI – Surgery
AI – Surgery Based on EF

- Operative mortality
  - EF >50% - 3.7%
  - EF 35%-50% - 6.7%
  - EF <35% - 14%

- Long-term survival also affected by EF
Indications for AVR: Severe AR

**Class I**
- Symptoms
- EF < 0.50
- Need for Ao surgery

**Class II**
- LVESD > 55 mm or > 25 mm/M²
- LVEDD > 75 mm
- LVESD > 50 mm or LVEDD > 70 mm and progressing

ACC/AHA Valve Guidelines 2006
Question: A 48 y.o. female with no significant past history is referred for evaluation of a heart murmur. She is asymptomatic. On examination, blood pressure is 140/60 and she has a 2/6 systolic ejection murmur and a holodiastolic murmur heart best along the right sternal border. Echocardiography demonstrates a dilated LV with EDD 60 mm and ESD 45 mm, mild LV dysfunction with EF 50%, a normal root and a bicuspid, nonstenotic aortic valve with severe insufficiency. The most appropriate management strategy is:

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The most appropriate management strategy is:

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B. Repeat echocardiogram in one year
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AI – Indications for Surgery

- Symptoms (1)
- LV dysfunction
  - EF <50% (1)
- LV dilatation
  - ESD > 55 mm or EDD > 75 (2a)
  - ESD 50-55 mm or EDD 70-75 mm (2b)
Question 4

Below is shown the CT scan of an asymptomatic 34 year old software engineer with an ejection click and a grade 3 mid-systolic murmur at the 2nd RICS.
Question 4

In addition to restricting his activities, which of the following management strategies do you recommend?

- **A.** Lisinopril 10 mg daily
  - 9%
- **B.** Losartan 25 mg daily
  - 13%
- **C.** Endovascular stenting
  - 0%
- **D.** Aortic valve and ascending aortic surgery
  - 79%
Bicuspid Aortic Valve

* Dilated Aortic Root *

Class I Indications for Surgery

- Maximal dimension > 5.0 cm or annual increase in size > 0.5 cm / year.*
- Maximal dimension > 4.5 cm and surgery indicated for severe AS or AR.*

* Consider lower threshold values for patients of small stature of either gender

ACC/AHA Valve Guidelines 2006
61 woman: MV commissurotomy 34 years ago NYHA Class III
MITRAL VALVE STENOSIS
<table>
<thead>
<tr>
<th>Grade</th>
<th>Mean gradient (mmHg)</th>
<th>Mitral valve Area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt; 5</td>
<td>1.6-2</td>
</tr>
<tr>
<td>Moderate</td>
<td>5-9</td>
<td>1.1-1.5</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 10</td>
<td>≤ 1</td>
</tr>
</tbody>
</table>
Pitfalls Pressure Half-time

\[ MVA = \frac{220}{T^{1/2}} \]

- Post balloon valvuloplasty
- Significant AR
- Significant diastolic dysfunction
- Heart rate

Pitfalls Continuity Equation

\[ \frac{SV \ AV}{TVI_{MS \ Jet}} \]

- AF
- Significant MR
- Significant AR
Mitral Stenosis
Anticoagulation
Class I

• AF: Paroxysmal, Persistent, Permanent
• Hx TIA/CVA, systemic embolus
• Presence of LA thrombus
Question 5

A 27 year old woman who is 26 weeks pregnant is admitted to the ICU with pulmonary edema. She is intubated, paralyzed and treated with broad spectrum antibiotics. TTE shows MS with mean gradient of 16 mm Hg at HR 115 BPM. Her BP is 105/76. No murmur is audible. Fetal heart sounds are normal and the maternal fetal medicine group are following.

Select the best initial management strategy:
1. Terminate pregnancy
2. IV beta-blocker
3. Heparin
4. Consult cardiac surgery
PMBV

Class I Indications

- Symptoms
- PA HTN (PA > 50 rest, > 60 ex)

Predicated on:
1. Favorable morphology (ECHO score)
2. Operator and Lab experience

Absent:
1. Moderate to severe MR
2. LA thrombus
3. Inability to perform trans-septal puncture
### Assessment of Mitral Valve Anatomy According to the Wilkins Score

<table>
<thead>
<tr>
<th>Grade</th>
<th>Mobility</th>
<th>Thickening</th>
<th>Calcification</th>
<th>Subvalvular Thickening</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Highly mobile valve with only leaflet tips restricted</td>
<td>Leaflets near normal in thickness (4-5 mm)</td>
<td>A single area of increased echo brightness</td>
<td>Minimal thickening just below the mitral leaflets</td>
</tr>
<tr>
<td>2</td>
<td>Leaflet mild and base portions have normal mobility</td>
<td>Midleaflet normal, considerable thickening of margins (5-8 mm)</td>
<td>Scattered area of brightness confined to leaflet margins</td>
<td>Thickening of chordal structures extending to onethird of the cusp length</td>
</tr>
<tr>
<td>3</td>
<td>Valve continues to move forward in diastole, mainly from the base</td>
<td>Thickening extending through the entire leaflet (5-8 mm)</td>
<td>Brightness extending into the mid-portions of the leaflets</td>
<td>Thickening extended to distal third of the chords</td>
</tr>
<tr>
<td>4</td>
<td>No or minimal forward movement of the leaflets in diastole</td>
<td>Considerable thickening of all leaflet tissue (&gt;8-10 mm)</td>
<td>Extensive brightness throughout much of the leaflet tissue</td>
<td>Extensive thickening and shortening of all chordal structures extending down to the papillary muscles</td>
</tr>
</tbody>
</table>
PERCUTANEOUS VALVULOPLASTY

**Immediate Impact of Valvuloplasty**

<table>
<thead>
<tr>
<th>prePMV</th>
<th>postPMV</th>
</tr>
</thead>
<tbody>
<tr>
<td>max PG = 44.9 mm Hg</td>
<td>max PG = 20.6 mm Hg</td>
</tr>
<tr>
<td>mean PG = 29.4 mm Hg</td>
<td>mean PG = 9.0 mm Hg</td>
</tr>
</tbody>
</table>

**Transesophageal Echocardiogram**

Demonstrating LAA With Significant Thrombus

LAA thrombus
EVALUATION OF SEVERITY OF MITRAL STENOSIS
## Approaches to Evaluation of Mitral Stenosis

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Units</th>
<th>Formula / Method</th>
<th>Concept</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve area</td>
<td>cm²</td>
<td>Direct measurement of anatomic MVA</td>
<td>- Independence from other factors</td>
<td>- Experience required - Not always reliable (poor acoustic window, severe valve calcification)</td>
<td></td>
</tr>
<tr>
<td>Pressure half-time</td>
<td>cm²</td>
<td>$220 / T_{LVOT}$</td>
<td>Rate of decrease of transmural flow is inversely proportional to MVA</td>
<td>Easy to obtain</td>
<td>Dependence on other factors (AR, LA compliance, LV diastolic function...)</td>
</tr>
<tr>
<td>Continuity equation</td>
<td>cm²</td>
<td>$\frac{MV-\text{CSA}<em>{aoa}(V</em>{\text{max}})}{V_{\text{max}}}$</td>
<td>Volume flows through mitral and aortic orifices are equal</td>
<td>Independence from flow conditions</td>
<td>Multiple measurements (source of errors) - Not valid if significant AR or MR</td>
</tr>
<tr>
<td>V̇EPA</td>
<td>cm²</td>
<td>$\frac{MV-\text{CSA}<em>{aoa}(V</em>{\text{max}})}{(p_{\text{aw}}+p_{\text{paw}})/2}$</td>
<td>MVA assessed by dividing initial volume flow by the maximum velocity of diastolic mitral flow</td>
<td>Independence from flow conditions</td>
<td>Technically difficult</td>
</tr>
<tr>
<td>Mean gradient</td>
<td>mm Hg</td>
<td>$\Delta P = \frac{\Sigma 4v^2}{N}$</td>
<td>Pressure gradient calculated from velocity using the Bernoulli equation</td>
<td>Easy to obtain</td>
<td>Dependent on heart rate and flow conditions</td>
</tr>
<tr>
<td>Systolic pulmonary artery pressure</td>
<td>mm Hg</td>
<td>$s\text{PAP} = 4\text{V}_{\text{max}} + \text{RA pressure}$</td>
<td>Addition of RA pressure and maximum gradient between RV and RA</td>
<td>Obtained in most patients with MS</td>
<td>- Arbitrary estimation of RA pressure - No estimation of pulmonary vascular resistance</td>
</tr>
<tr>
<td>Mean gradient and systolic pulmonary artery pressure at exercise</td>
<td>mm Hg</td>
<td>$\frac{\Delta P - \Sigma 4v^2}{N}$</td>
<td>$s\text{PAP} = 4\text{V}_{\text{max}} + \text{RA pressure}$</td>
<td>Assessment of gradient and $s\text{PAP}$ for increasing workload</td>
<td>Incremental value in assessment of tolerance</td>
</tr>
<tr>
<td>Valve resistance</td>
<td>dyn. sar cm⁻¹</td>
<td>$\frac{\Delta P_{\text{max}}}{(\text{CSA}<em>{\text{aoa}})(V</em>{\text{max}})}$</td>
<td>Resistance to flow caused by MS</td>
<td>Initially suggested to be less flow-dependent, but not confirmed</td>
<td>No prognostic value - No clear threshold for severity - No additional value vs. valve area</td>
</tr>
</tbody>
</table>
## Recommendations for Classification of Mitral Stenosis Severity

<table>
<thead>
<tr>
<th>Specific findings</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve area (cm²)</td>
<td>&gt;1.5</td>
<td>1.0-1.5</td>
<td>&lt;1.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Supportive findings</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean gradient (mm Hg)</td>
<td>&lt;5</td>
<td>5-10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mm Hg)</td>
<td>&lt;30</td>
<td>30-50</td>
<td>&gt;50</td>
</tr>
</tbody>
</table>
SECONDARY EFFECTS OF MITRAL STENOSIS
Mitral Regurgitation

Etiology

**Acute MR**
- Acute MI (Inf-Post)
- Endocarditis
- Trauma
- “Acute on chronic”

**Chronic MR**
- Myxomatous
- Ischemic
- DCM
- Rheumatic
- MAC
- HOCM
- Other (APLS, etc.)
# Mitral Regurgitation

**Pathophysiology**

<table>
<thead>
<tr>
<th>LAC</th>
<th>LAP</th>
<th>EDV</th>
<th>EF</th>
<th>Contr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute MR</td>
<td>nl, ↓</td>
<td>↑↑↑↑</td>
<td>↑</td>
<td>nl, ↑</td>
</tr>
<tr>
<td>Chronic MR</td>
<td>↑</td>
<td>nl, ↑</td>
<td>↑↑</td>
<td>↓, ↑, nl</td>
</tr>
</tbody>
</table>
Myxomatous MV

Barlow’s Disease

Leaflet thickening, large redundant leaflets, chordal rupture, annular dilation, often multi-segmental
Myxomatous MV
Fibroelastic Deficiency

Lack of connective tissue → leaflet and chordal thinning, eventually prolapse and rupture. Often single segment
Functional MR
Pulmonary Vein Flow
Pulmonary Vein Flow
Pulmonary Vein Flow
Spectral Doppler Patterns Obtained on TTE (four-chamber view)

1-Normal

2-Systolic Blunting

3-Diastolic Dominant

4-Systolic Flow Reversal
### Mitral Regurgitation Severity Grades

<table>
<thead>
<tr>
<th></th>
<th>ERO (mm²)</th>
<th>RVol (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt; 20</td>
<td>&lt; 30</td>
</tr>
<tr>
<td>Moderate</td>
<td>20-49</td>
<td>30-59</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 40</td>
<td>&gt; 60</td>
</tr>
</tbody>
</table>

*JASE 2003; 16 (7):777*
Summary

Severe Organic MR

- ERO ≥ 40 mm²
- RVol ≥ 60 cc
- RF ≥ 50%
- Vena contracta ≥ 7 mm
- Peak E velocity > 1.2 m/s
- “V” wave configuration on CW
- Flow reversal both pulmonary veins

JASE 2003; 16 (7):777
PCW “v” waves in MR

PCW “v” Waves
- Mitral regurgitation
- Ventricular septal defect
- Noncompliant left atrium
Non-Ischemic Severe MR

NYHA FC I

EF > 0.60
ESD < 40 mm

EF ≤ 0.60
ESD ≥ 40 mm

AF? PHT?
Yes
Class IIa

MV Repair
MVR

Class I

ACC/AHA Valve Guidelines 2006
Non-Ischemic Severe MR

NYHA FC I

EF > 0.60
ESD < 40 mm

EF ≤ 0.60
ESD ≥ 40 mm

Class I

MV Repair*
MVR

AF? Yes

Class Ila

MV Repair Highly Likely?

No

Yes

Class Ila

MV Repair*

ACC/AHA Valve Guidelines 2006
Non-Ischemic Severe MR

NYHA FC I

- EF > 0.60
- ESD < 40 mm

- EF < 0.60
- ESD ≥ 40 mm

AF? PHT?

Yes
- MV Repair*
- MVR

No
- Class Ila

Yes
- MV Repair*

No
- Class Ila

ACC/AHA Valve Guidelines 2006
MITRAL VALVE REGURGITATION
PRIMARY AND FUNCTIONAL (LV DYSFUNCTION)
FOCAL PROLAPSE OF THE ANTERIOR LEAFLET

OTHERS: IP INFARCT-POSTERIOR LEAFLET RESTRICTION
SAM-HOCM
CONGENITAL CLEFT ANTERIOR MV LEAFLET
RHEUMATIC INFECTIVE ENDOCARDITIS WITH PERFORATION OF THE AML
COMPLICATIONS OF MVP
CHORDAL RUPTURE
FUNCTIONAL MITRAL REGURGITATION
3D BEST PARAMETER - THE AREA SUBTENTED BY THE OUTWARDLY TETHERED MITRAL VALVE LEAFLETS
ISCHEMIC MR-PAPILLARY MUSCLE RUPTURE
MITRAL VALVE ENDOCARDITIS
PERFORATION
ENDOCARDITIS
PERFORATION
### Qualitative and Quantitative Parameters

**Useful in Grading Mitral Regurgitation Severity**

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structural parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LA size</td>
<td>Normal*</td>
<td>Normal or dilated</td>
<td>Usually dilated**</td>
</tr>
<tr>
<td>LV size</td>
<td>Normal*</td>
<td>Normal or dilated</td>
<td>Usually dilated**</td>
</tr>
<tr>
<td>Mitral leaflets or support apparatus</td>
<td>Normal or abnormal</td>
<td>Normal or abnormal</td>
<td>Abnormal/flail leaflet/ ruptured papillary muscle</td>
</tr>
<tr>
<td><strong>Doppler parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Color flow jet area²</td>
<td>Small, in central jet (usually &lt;4 cm² or &lt;20% of LA area)</td>
<td>Variable</td>
<td>Large in central jet (usually &gt;10 cm² or &gt;40% of LA area or variable size wall-thinning jet swirling in LA</td>
</tr>
<tr>
<td>Mitral inflow-PW</td>
<td>A wave dominant⁶</td>
<td>Variable</td>
<td>E wave dominant⁶/C&gt; (E usually 1.2 m/s)</td>
</tr>
<tr>
<td>Jet density-CW</td>
<td>Incomplete or faint</td>
<td>Dense</td>
<td>Dense</td>
</tr>
<tr>
<td>Jet contour-CW</td>
<td>Parabolic</td>
<td>Usually parabolic</td>
<td>Early peaking - triangular</td>
</tr>
<tr>
<td>Pulmonary vein flow</td>
<td>Systolic dominance⁷</td>
<td>Systolic blunting⁸</td>
<td>Systolic flow reversal⁹</td>
</tr>
<tr>
<td><strong>Quantitative parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VC width (cm)</td>
<td>&lt;0.3</td>
<td>0.3 – 0.69</td>
<td>≥0.7</td>
</tr>
<tr>
<td>RVol (ml/beat)</td>
<td>&lt;30</td>
<td>30–44</td>
<td>45–59</td>
</tr>
<tr>
<td>RE %</td>
<td>&lt;30</td>
<td>30–39</td>
<td>40–49</td>
</tr>
<tr>
<td>ERAO (cm²)</td>
<td>&lt;0.20</td>
<td>0.20–0.29</td>
<td>0.30–0.39</td>
</tr>
</tbody>
</table>
Color Flow Recording of a Mitral Regurgitation Jet
## Qualitative and Quantitative Parameters Useful in Grading Mitral Regurgitation Severity

<table>
<thead>
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<td>LA size</td>
<td>Normal*</td>
<td>Normal or diluted</td>
<td>Usually dilated**</td>
</tr>
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<td>Normal*</td>
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<td>Normal or abnormal</td>
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<td>Color flow jet area</td>
<td>Small, in central jet (usually &lt;4 cm² or &lt;20% of LA area)</td>
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<tr>
<td>Jet contour-CW</td>
<td>Parabolic</td>
<td>Usually parabolic</td>
<td>Early peaking - triangular</td>
</tr>
<tr>
<td>Pulmonary vein flow</td>
<td>Systolic dominance*</td>
<td>Systolic blunting*</td>
<td>Systolic flow reversal*</td>
</tr>
<tr>
<td><strong>Quantitative parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VC width (cm)</td>
<td>&lt;0.3</td>
<td>0.3 – 0.69</td>
<td>≥0.7</td>
</tr>
<tr>
<td>RVol (ml/beat)</td>
<td>&lt;30</td>
<td>30-44</td>
<td>45-59</td>
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<tr>
<td>RE %</td>
<td>&lt;30</td>
<td>30-39</td>
<td>40-49</td>
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<tr>
<td>ERAO (cm²)</td>
<td>&lt;0.20</td>
<td>0.20-0.29</td>
<td>0.30-0.39</td>
</tr>
</tbody>
</table>
PROXIMAL CONVERGENCE ZONE- PISA MORE ACCURATE FOR CENTRAL JETS
REGURGITANT VOLUME
DIFFERENCE OF THE FLOW ACROSS THE MR AND THE LVOT
RV OVER 60 CC - SEVERE MR
RV = (0.785 \times \text{MVD}^2) \times \text{VTI MV} - (0.785 \times \text{LVOT}^2) \times \text{LVOT VTI}
REGURGITANT FRACTION

RF = \( \frac{RV}{MV \text{ flow}} \) \times 100

EFFECTIVE REGURGITANT ORIFICE

ERO (MV) = \( \frac{RV}{VTI (MR)} \)
Management Strategy for Patients With Chronic Severe Mitral Regurgitation

Chronic Severe Mitral Regurgitation?

- Clinical Evaluation + Echo

  - Symptoms?
    - Yes
      - LV Function?
        - Yes
          - EF > 0.30
        - No
          - EF < 0.30
            - EF < 0.30
            - EF ≥ 0.30
        - LV Dysfunction
          - EF ≤ 0.90
          - LV Function
            - EF ≥ 0.30
            - EF ≤ 0.30
              - Medical Therapy
              - MV Repair
                - If not possible, MVR
      - No
        - Reevaluation

  - No
    - LV Function?
      - Yes
        - EF > 0.30
      - No
        - EF < 0.30
          - EF > 0.30
          - EF ≤ 0.30

Clinical Evaluation Every 6 Months
Echo Every 6 Months
MITRAL VALVE REPAIR
OTHER ROLES OF ECHOCARDIOGRAPHY

PROSTHETIC VALVE FUNCTION
MitraL VALE ClIP
ETIOLOGIES OF TRICUSPID VALVE DISEASE

EPSTEIN ANOMALY
RIGHT VENTRICULAR SYSTOLIC PRESSURE
# Echocardiographic and Doppler Parameters Used in Grading Pulmonary Regurgitation Severity

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricuspid valve</td>
<td>Usually normal</td>
<td>Normal or abnormal</td>
<td>Abnormal/flail leaflet/poor coaptation</td>
</tr>
<tr>
<td>RV/RA/IVC size</td>
<td>Normal*</td>
<td>Normal or dilated</td>
<td>Usually dilated**</td>
</tr>
<tr>
<td>Jet area-central jets (cm²)</td>
<td>&lt;5</td>
<td>5-10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>VC width (cm)</td>
<td>Not defined</td>
<td>Not defined, but &lt;0.7</td>
<td>&gt;0.7</td>
</tr>
<tr>
<td>PISA radius (cm)</td>
<td>≤0.5</td>
<td>0.6-0.9</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>Jet density and contour-CW</td>
<td>Soft and parabolic</td>
<td>Dense, variable contour</td>
<td>Dense, triangular with early peaking</td>
</tr>
<tr>
<td>Hepatic vein flow</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
<td>Systolic reversal</td>
</tr>
</tbody>
</table>
Findings Indicative of Haemodynamically Significant Tricuspid Stenosis

<table>
<thead>
<tr>
<th>Specific findings</th>
<th>Supportive findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean pressure gradient</td>
<td>Enlarged right atrium ≥ moderate</td>
</tr>
<tr>
<td>Inflow time-velocity integral</td>
<td>Dilated inferior vena cava</td>
</tr>
<tr>
<td>$T_{1/2}$</td>
<td></td>
</tr>
<tr>
<td>Valve area by continuity equation$^a$</td>
<td></td>
</tr>
<tr>
<td>≥5 mm Hg</td>
<td></td>
</tr>
<tr>
<td>&gt; 60 cm</td>
<td></td>
</tr>
<tr>
<td>≥190 ms</td>
<td></td>
</tr>
<tr>
<td>≤1 cm$^2$</td>
<td></td>
</tr>
</tbody>
</table>
## Grading of Pulmonary Stenosis

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Peak velocity (m/s)</strong></td>
<td>&lt;3</td>
<td>3.4</td>
<td>&gt;4</td>
</tr>
<tr>
<td><strong>Peak gradient (mm Hg)</strong></td>
<td>&lt;36</td>
<td>36-64</td>
<td>&gt;64</td>
</tr>
</tbody>
</table>
## Echocardiographic and Doppler Parameters Used in Grading Pulmonary Regurgitation Severity

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulmonic valve</strong></td>
<td>Normal</td>
<td>Normal or abnormal</td>
<td>Abnormal</td>
</tr>
<tr>
<td><strong>RV size</strong></td>
<td>Normal*</td>
<td>Normal or dilated</td>
<td>Dilated</td>
</tr>
<tr>
<td><strong>Jet size by color Doppler</strong></td>
<td>Thin (usually &lt;0.1 mm in length) with a narrow origin</td>
<td>Intermediate</td>
<td>Usually large, with a wide origin; May be brief in duration</td>
</tr>
<tr>
<td><strong>Jet density and deceleration rate-CW</strong></td>
<td>Soft; slow deceleration</td>
<td>Dense; variable deceleration</td>
<td>Dense; steep deceleration, early termination of diastolic flow</td>
</tr>
<tr>
<td><strong>Pulmonic systolic flow compared to systemic flow - PW</strong></td>
<td>Slightly increased</td>
<td>Intermediate</td>
<td>Greatly increased</td>
</tr>
</tbody>
</table>
VALVE DISEASE CASES