

Valvular Heart Disease

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No disclosures

Learning Objectives

- **Aortic Stenosis-** definition, physical exam findings, clinical manifestations & treatment
- **Mitral Regurgitation-** review primary (degenerative) vs secondary (functional) MR and treatment options
- **Mitral Stenosis-** not every MS is the same, review treatment
- **Tricuspid Regurgitation-** new frontier in structural heart disease

General Concepts

- Echocardiography is the mainstay of diagnosis of valve disease
- Integration of physical exam findings and multiple parameters from imaging studies is essential in accurate diagnosis of valve disease
- Multidisciplinary valve team is key to improved outcomes
- Surgical risk and patient preference play an important role in decision-making regarding valve intervention

Aortic Stenosis

- Supravalvular
- **Valvular**
 - Calcific
 - Congenital (bicuspid, unicuspid)
 - Rheumatic
 - Radiation induced valve disease
- Subvalvular
 - Subaortic membrane
 - Hypertrophic cardiomyopathy

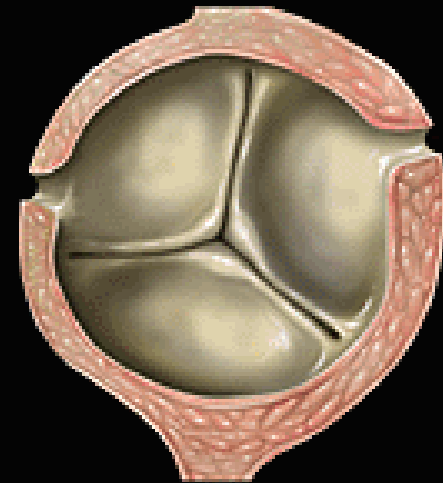
Calcific Aortic Stenosis

- Age related progressive degeneration
 - Inflammatory disease → ? Anti-inflammatory medications
- 2% of persons ≥ 65 years have significant calcific AS, whereas 29% exhibited age-related aortic valve sclerosis without stenosis
- 50% increased risk of cardiovascular death and myocardial infarction over 5 years of follow-up
- $\sim 2/3$ of all heart valve surgeries in U.S. are for aortic valve replacement, mostly for severe AS

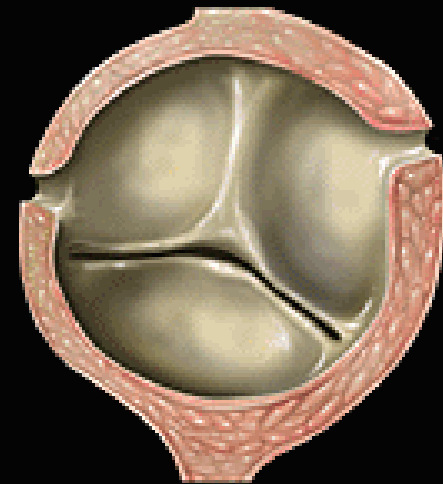
Bicuspid Aortic Valve

- 1-2% of general population; 2:1 male : female ratio
- Can occur as isolated lesion or a/w aortopathy
- Can lead to stenosis, regurgitation or both
- Mid-systolic ejection click
- Among patients with aortic coarctation, 50-60% have bicuspid AV
- First degree relatives need to be screened

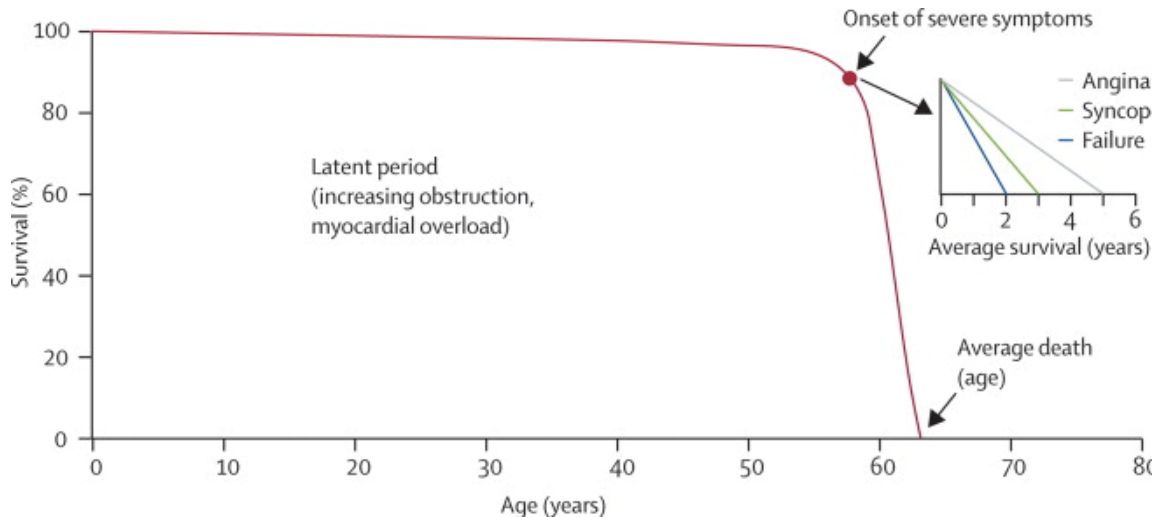
Normal aortic valve



Bicuspid aortic valve



Clinical Manifestations



- Can be complicated by Heyde's syndrome
 - Acquired type 2A von Willebrand deficiency leading to angiodysplasias in GI tract

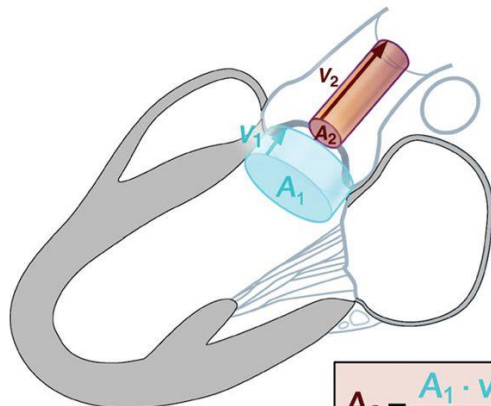
Physical Exam

- Late-peaking systolic crescendo-decrescendo murmur that radiates to the carotids with a
 - Decrease in intensity of S2
 - Slow-rising, late-peaking, low-amplitude carotid pulse
- AS increases in intensity with maneuvers that increase the stroke volume (e.g., squatting)
 - Used to differentiate AS from hypertrophic cardiomyopathy

Diagnosis: Echocardiography

Criteria	Formula/Method	Advantages	Disadvantages
Peak transaortic velocity	Direct measurement	Direct	Requires parallel alignment of ultrasound beam, flow dependent
Mean gradient	$\Delta P = \sum 4v^2/N$	Comparable to invasive measurements	Requires accurate velocity measurement, flow dependent
Aortic valve area	$AVA = (CSA_{LVOT} \times VTI_{LVOT}) / VTI_{AV}$	Relatively flow independent	Depends on accurate LVOT diameter measurement which is prone to errors

Aortic valve area Continuity equation



$$A_2 = \frac{A_1 \cdot v_1}{v_2}$$

Audience Response Question

65-year-old F with osteoporosis is here for yearly exam. She has no cardiac symptoms. She routinely exercises on a recumbent bike without any symptoms. On exam, her BP is 126/ 74, HR is 72bpm. She has 3/6 early peaking systolic murmur at LUSB with preserved S2. TTE is ordered and shows normal biventricular systolic function, aortic valve Vmax is 3.1m/sec, mean transaortic gradient of 28mmHg and calculated aortic valve area of 1.4cm²

What is the severity of her aortic stenosis?

- A- No aortic stenosis
- B- Mild aortic stenosis
- C- Moderate aortic stenosis
- D- Severe aortic stenosis

Severity of AS

Table 3 Recommendations for grading of AS severity

	Aortic sclerosis	Mild	Moderate	Severe
Peak velocity (m/s)	≤2.5 m/s	2.6–2.9	3.0–4.0	≥4.0
Mean gradient (mmHg)	–	<20	20–40	≥40
AVA (cm ²)	–	> 1.5	1.0–1.5	<1.0
Indexed AVA (cm ² /m ²)	–	>0.85	0.60–0.85	<0.6
Velocity ratio	–	> 0.50	0.25–0.50	<0.25

If discordant findings are present (i.e MG < 40 mmHg but AVA is < 1 cms²) → Cardiology referral is indicated

Diagnosis

Cardiac Catheterization

- Invasive assessment of the AS gradient is indicated when
 - Noninvasive testing is inconclusive
 - Discordant with physical exam findings and symptoms

Exercise Stress Testing

- Recommended to evaluate exercise tolerance and hemodynamic response in asymptomatic patients with severe AS
- Contraindications to ETT
 - Established indication for AVR
 - Uncontrolled hypertension
 - Symptomatic or hemodynamically significant arrhythmias
 - Inability to perform the test

For the same patient, when should the follow up TTE be done?

A- 6-12 months

B- 1-2 year

C- 3-5 years

Following a patient with AS

A	At risk	Patients with risk factors for the development of VHD
B	Every 3-5 years if Vmax 2-2.9m/sec; Every 1-2 years if Vmax 3-3.9m/sec	
C	Asymptomatic severe	Asymptomatic patients who have reached the criteria for severe AS C1: Normal LVEF , AV Vmax ≥ 4 m/s or mean $\Delta P \geq 40$ mmHg, AVA ≤ 1.0 cm ²
D	Every 6-12 months	
		<4 m/s or mean $\Delta P < 40$ mm Hg AND SVi <35mL/m ² ; Measured when patient is normotensive (SBP <140 mm Hg)

Audience Response Question

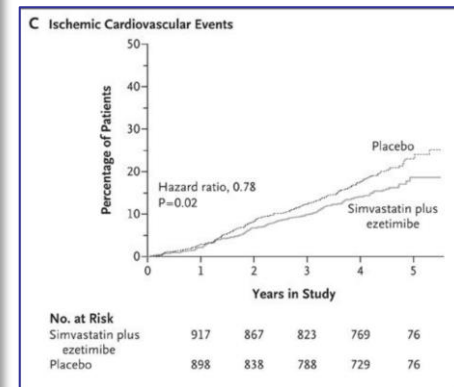
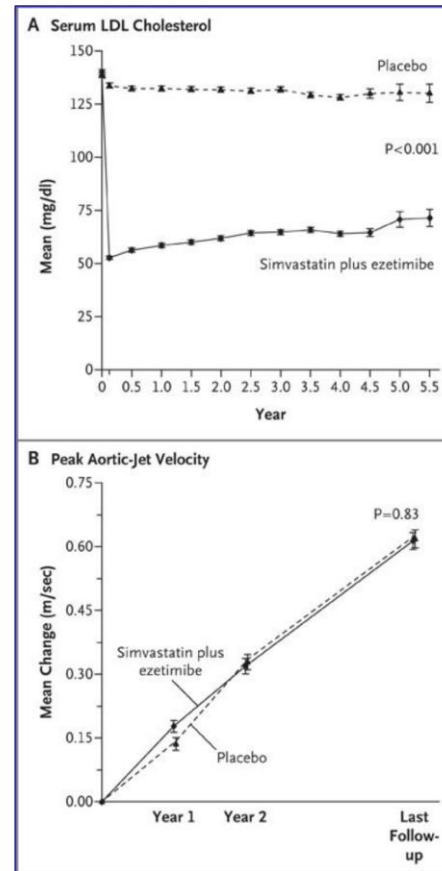
Her most recent lipid profile shows: TC 190, HDL 44, LDL 100, Trig 78. Her ASCVD score is 5.8%.
Would you recommend statin therapy?

A- Yes

B- No

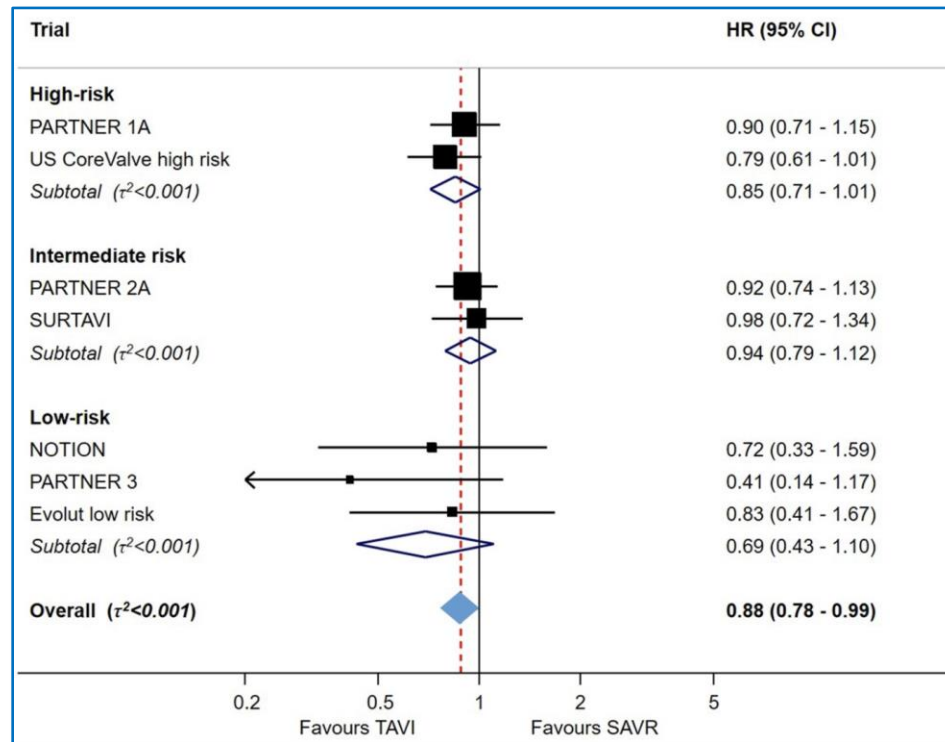
Medical Therapy for AS

- Treat HTN
- Statin therapy in all patients for primary and secondary prevention of atherosclerotic disease



Surgical AVR vs Transcatheter AVR

TAVR approved for all surgical risk groups



Indications AVR for AS

- Symptomatic AS
- Asymptomatic AS if
 - Abnormal ETT (a fall of ≥ 10 mm Hg in sBP from baseline to peak exercise; significant \downarrow in ET as compared with age and sex normal standards)
 - Elevated BNP
 - Very severe AS
 - Rapid progression of AS

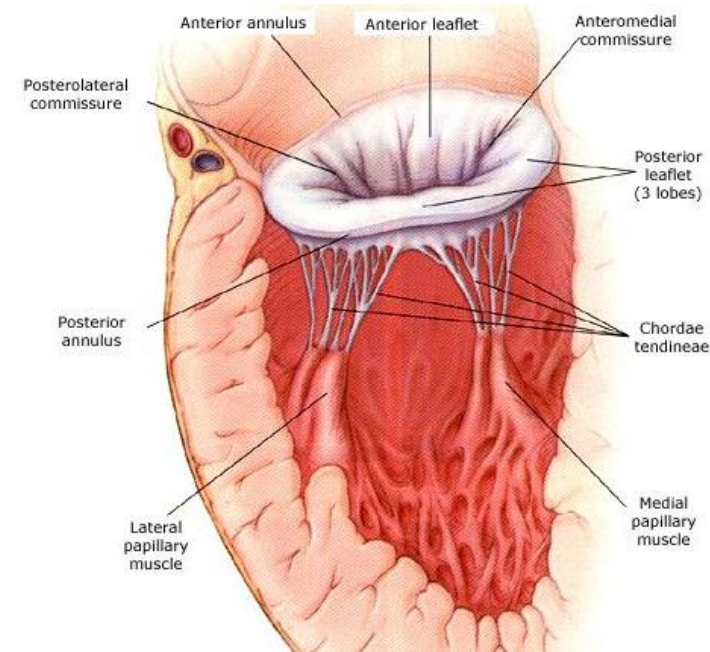
Antithrombotic therapy after AVR

- Aspirin 75 to 100mg post TAVI or SAVR (Class IIA)
- If no bleeding risk, VKA for 3-6 months post SAVR (Class IIA)

Mitral Regurgitation

ACUTE

- Acute chordal rupture (MVP)
- Ischemic MR
- Papillary muscle rupture
- Prosthesis failure



CHRONIC

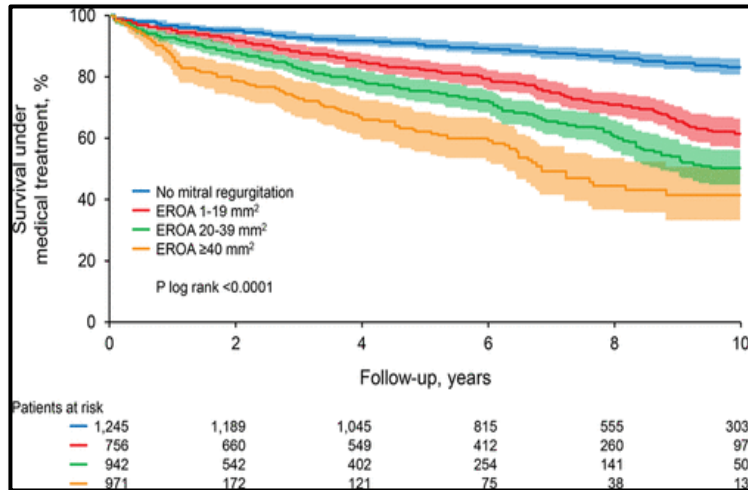
Primary MR

- Mitral valve prolapse (MVP)
- Leaflet perforation (endocarditis)
- Congenitally abnormal MV

Secondary regurgitation (functional)

- Annular dilation
- Papillary muscle dysfunction

Degenerative MR Outcomes and Treatment



Clemence Antoine. Circulation. 2018

Medical treatment : None!

MV repair if:

- Severe symptomatic Primary MR (Class I)
- Severe asymptomatic Primary MR:
 - LVEF ≤ 60%** or LVEDD ≥ 40 mm and *successful and durable repair possible* (Class I)
 - LVEF ≥ 60%** or LVEDD < 40mm and *expected surgical mortality < 1% with > 95% likelihood of successful and durable repair* without residual MR (Class IIa)

*LVEDD: LV End systolic dimension

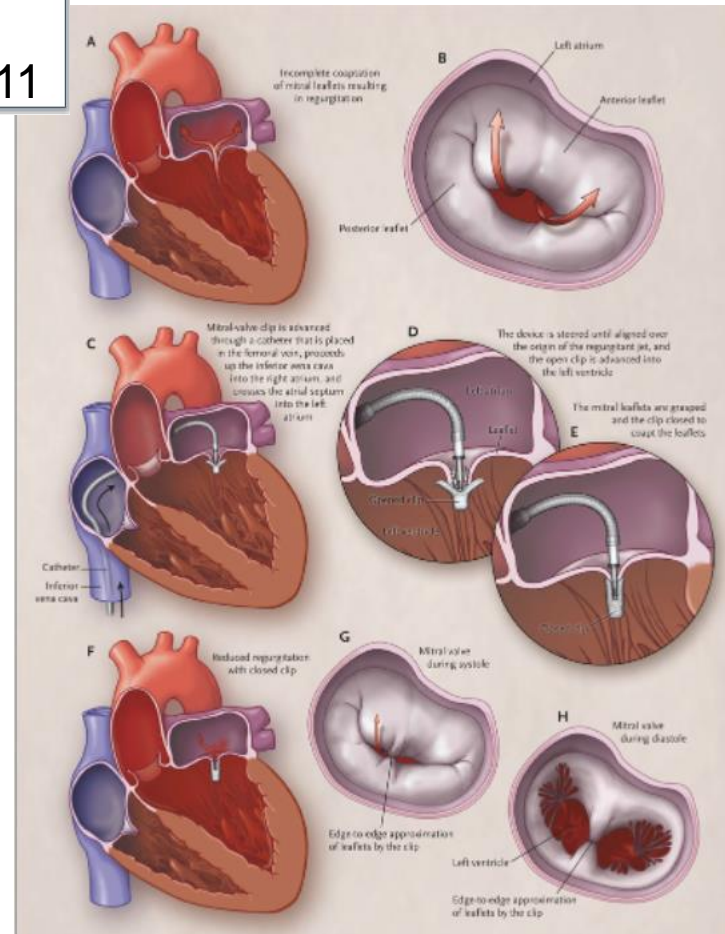
The NEW ENGLAND JOURNAL of MEDICINE

Percutaneous Repair or Surgery for Mitral Regurgitation

Ted Feldman, M.D., Elyse Foster, M.D., Don Glower, M.D., Saibal Kar, M.D., Michael J. Rinaldi, M.D., Peter S. Fail, M.D., Richard W. Smalling, M.D., Ph.D., Robert Siegel, M.D., Geoffrey A. Rose, M.D., Eric Engerson, M.D., Catalin Loghin, M.D., Alfredo Trento, M.D., Eric R. Skipper, M.D., Tommy Fudge, M.D., George V. Letsou, M.D., Joseph M. Massaro, Ph.D., and Laura Mauri, M.D., M.Sc.,
for the EVEREST II Investigators*

2011

- Surgery was superior to MitraClip in MR reduction at discharge but at 12 and 24 months, rates of reduction in MR were similar
- MitraClip is safer than surgery due to lower risk of transfusion



Indication for TEER (Class IIA)

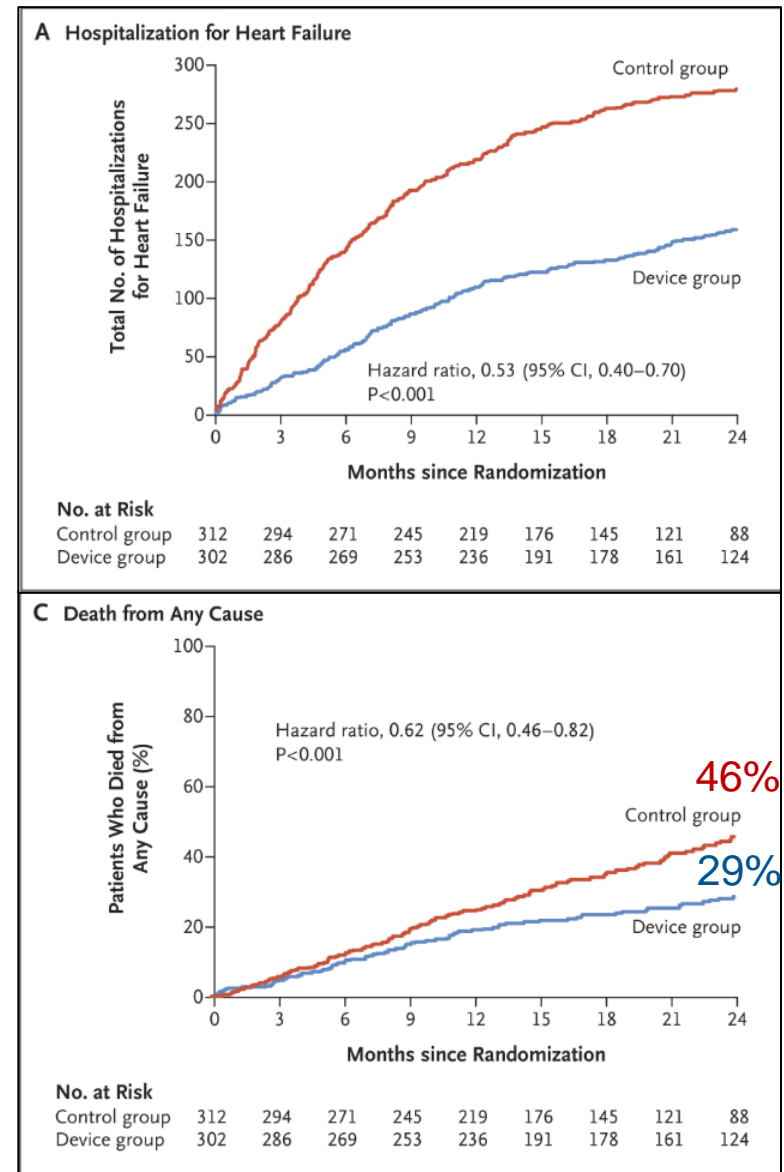
- **Symptomatic** or **Asymptomatic** patients with degenerative MR if high or prohibitively high surgical risk with favorable anatomy for transcatheter approach and life expectancy > 1 year

Treatment of Secondary MR

- **Initial treatment is ALWAYS guideline-directed medical therapy and if indicated CRT (chronic resynchronization therapy)**
- Severe MR + Undergoing CABG → MV surgery (Class IIA)

COAPT Trial

- Randomized controlled trial
- 614 pts with moderate to severe or severe secondary MR who remained symptomatic despite the use of maximal doses of GDMT
- Very strict inclusion/exclusion criteria



TEER for Secondary MR (Class IIA)

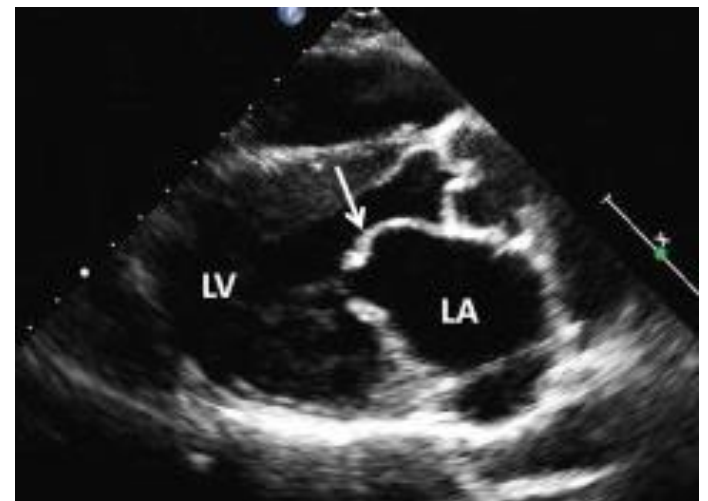
- *Initial treatment is ALWAYS guideline-directed medical therapy and CRT (chronic resynchronization therapy) if indicated*
- Persistent severe functional MR with LVEF 20-50%, LVESD \leq 70mm, PASP \leq 70mmHg (Class IIA)

Mitral Stenosis

- Etiology:
 - Rheumatic (>80%)
 - Calcific (renal failure, calciphylaxis, advanced HTN)
 - Congenital (parachute mitral valve, repaired cleft MV)
 - Status post repair/replacement
- Severe Mitral Stenosis in *Rheumatic Disease*:
 - Mitral valve area $< 1.5\text{cm}^2$ (very severe MS $< 1.0\text{cm}^2$)
 - (mean gradient $>10\text{mmHg}$)
 - PA Systolic Pressure $> 50\text{mmHg}$
- Elevated LA pressures \rightarrow Atrial fibrillation

Rheumatic Mitral Stenosis

- ~ 40M affected by RHD globally in 2019
- Primary prevention: Prompt diagnosis and treatment for Group A strep
- Secondary prevention: Pen G benzathine IM 1.2M units q 21-28 d OR Pen V oral 250mg BID



Rheumatic fever with carditis and residual heart disease	10 years or until 40 y.o.; whichever is longer
Rheumatic fever with carditis but no residual heart disease	10 years or until 21 y.o.; whichever is longer

Cardiac Management

- **Anticoagulation with VKA for AF with MS is a must**
- Rate control to improve diastolic filling time
- Diuretics to reduce LA pressure
- If severe and symptomatic intervention is necessary

Rheumatic Mitral Stenosis

Intervention

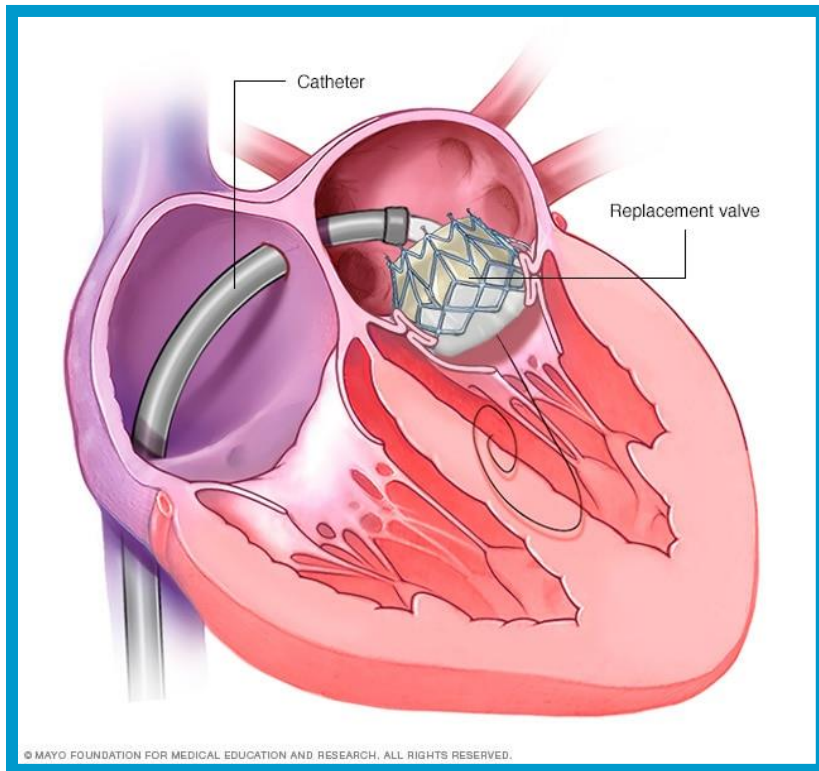
Symptomatic Severe MS

- Percutaneous Balloon Commissurotomy (PMBC)
 - Favorable mitral anatomy \leq moderate mitral regurgitation
 - Complications: stroke, increased MR, tamponade
- Surgical Mitral Valve Replacement
 - If unfavorable mitral anatomy for PMBC or concomitant MR

Asymptomatic Severe MS

- Intervention:
 - New onset atrial fibrillation (Class I if very severe, class IIa if severe)
 - Consideration of pregnancy

Intervention for Calcific Mitral Stenosis



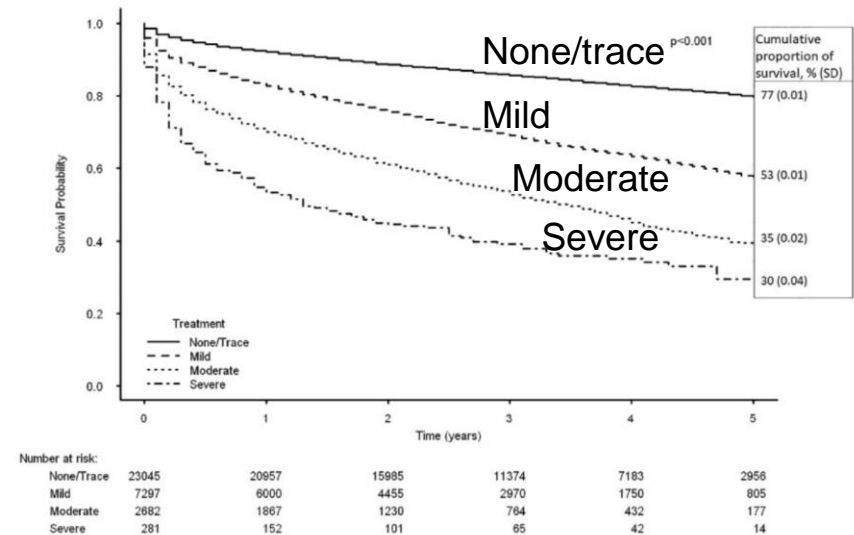
- Valve in mitral annular calcification (calcific mitral stenosis)
- Degenerative bioprosthesis
- Valve in annuloplasty ring

Tricuspid Regurgitation

Common yet rarely treated...

- Trivial/Mild TR is considered a “normal variant”
- 1.6 M people in US with \geq moderate to severe TR; only 5000 TR surgeries/year are done in the US due to historically poor outcomes
- Leads to right sided heart failure, gut edema, renal failure, liver failure, low CO state

Clinical impact of TR in long term outcomes:



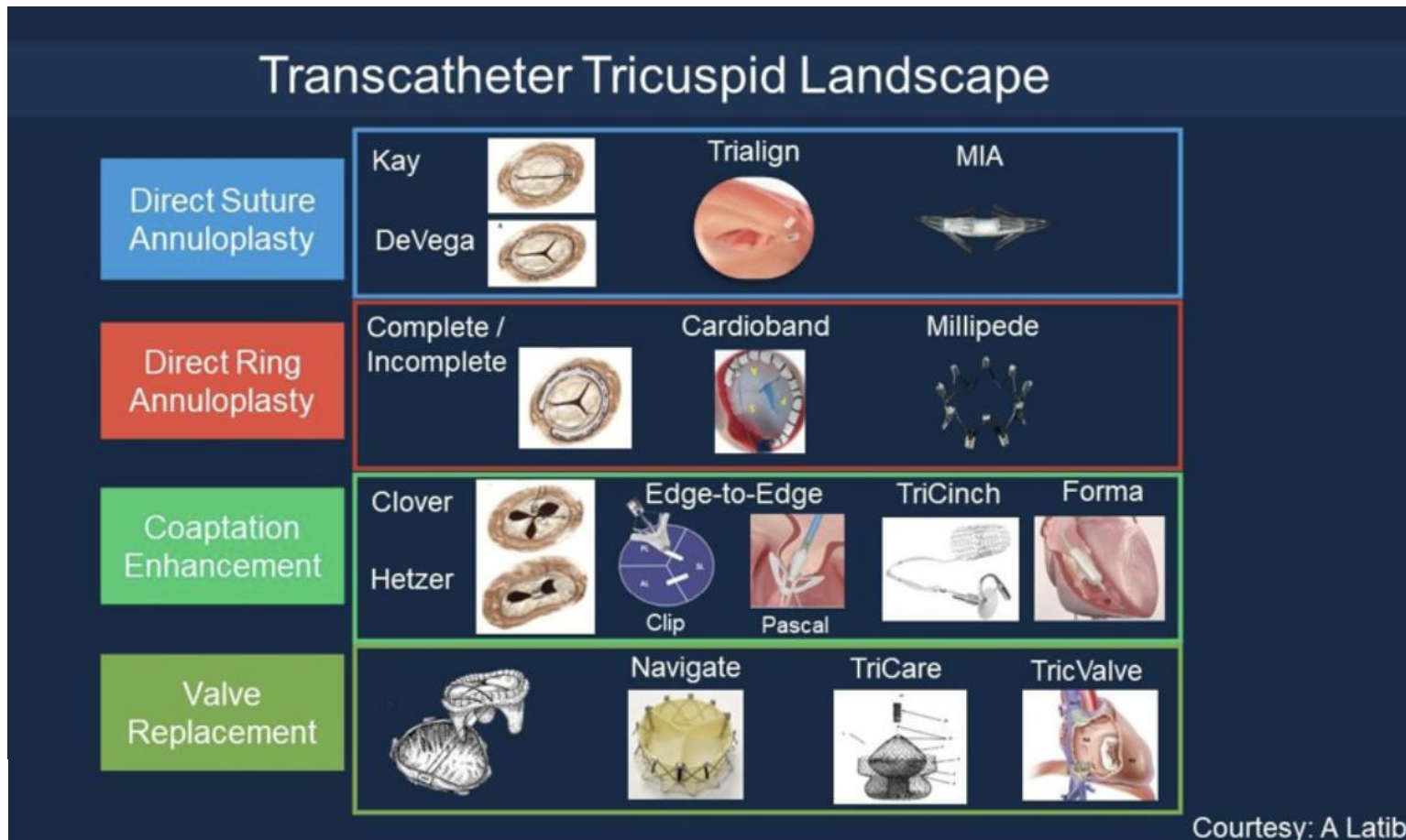
Chorin E, et al. Eur Heart J Cardiovasc Imaging. 2020 Feb 1;21(2):157-165

Medical treatment of TR

Diuretics

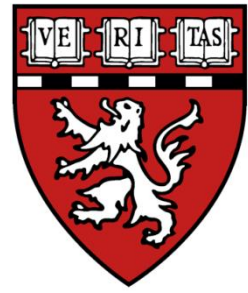
Favor Torsemide > Furosemide due to increased bioavailability in the setting of gut edema

Transcatheter Tricuspid Valve Therapies



Summary

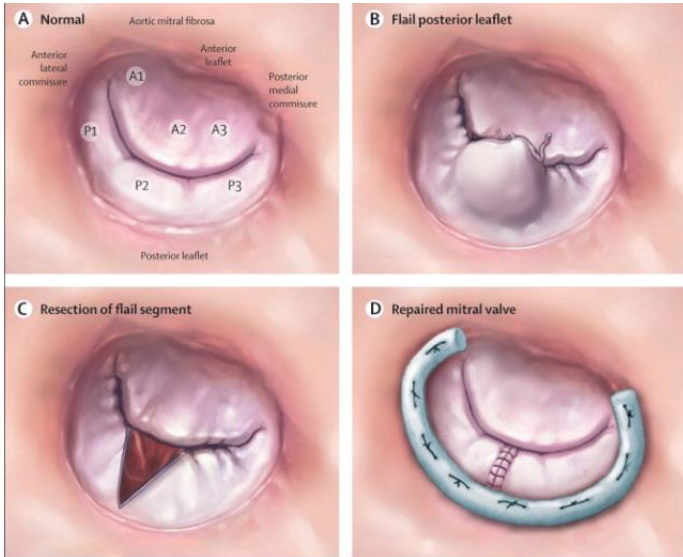
- Aortic Stenosis: Mainstay of diagnosis is TTE. Surveillance imaging is indicated based on the severity
- TAVR: approved for all surgical risk groups but SAVR is still recommended for patients < 65 years old
- Mitral Regurgitation: Treatment depends on the etiology (primary vs secondary), symptoms and LV size and function
- Secondary Mitral Regurgitation: Initial treatment is GDMT. TEER is reasonable among patients who have persistent symptoms despite GDMT.
- Rheumatic Mitral Stenosis: Anticoagulation for atrial fibrillation with mitral stenosis is required along with rate control strategies
- Tricuspid regurgitation: common & under-treated. New transcatheter treatment options are currently under investigation



Thank you

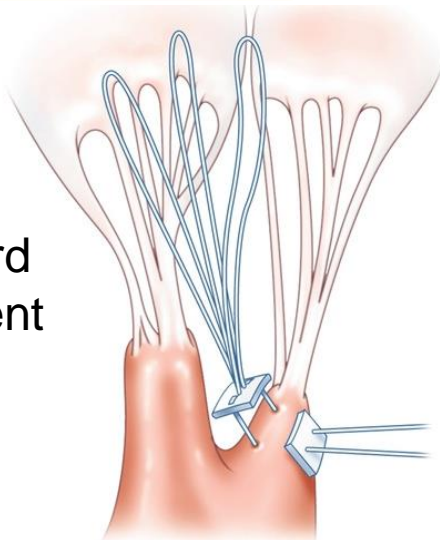
MV Repair Techniques

Triangular Resection

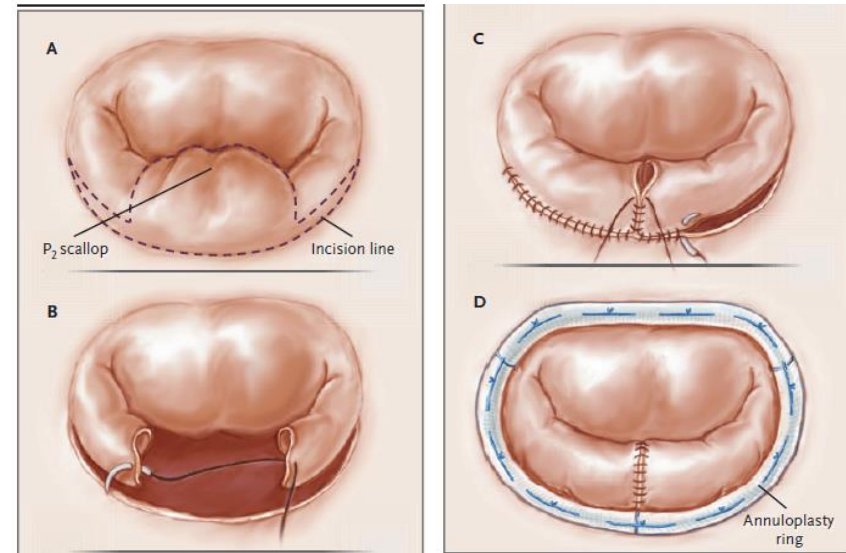


Enriquez-Sarano Lancet 2000

Neochord placement



Quadrangular Resection



Foster NEJM 2010

Alfieri's Stitch

