General Principles

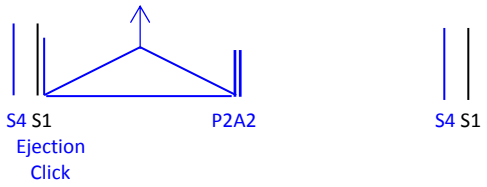
- When do you need to investigate a murmur with TTE?
  - all diastolic murmurs
  - all systolic murmurs w/ symptoms or w/o Sx but ≥3/6, holosystolic, or ejection click
  - NB otherwise likely benign or associated w/ physiologic increase in blood velocity
    - Newborn: Physiologic Pulmonary Branch Stenosis (turbulent flow thru hypoplastic pulmonary arteries), PDA (turbulent flow thru PDA), etc
    - Pre-Schooler: Stills Vibratory Murmur (turbulent flow thru LVOT), Venous Hum (turbulent flow thru systemic venous system as it returns back to heart), Carotid Bruit (turbulent flow from large bore aorta to small bore branches), etc
    - Children: Pulmonary Flow Murmur (turbulent flow thru PV), etc
    - Adults: Innocent Flow Murmur (systolic, soft, at base, no radiation, no Sx)
    - Pregnant Women: Mammary Souffle (systolic, soft, b/t breast/sternum, no radiation, no Sx)
  - Bernoulli Equation: $P = 4V^2$ eg. velocity is 4m/s then pressure is 96mm of Hg
    - NB Pulmonary Pressure = measure TR Velocity on TTE then calculate pressure using equation then add that number to RA pressure
- Management Approach
  - General Stenotic Lesions: Sx always occur before permanent dysfunction therefore Tx based on Sx
  - General Regurgitant Lesions: although most develop Sx prior to permanent dysfunction some do not therefore once regurgitation is identified you need to follow these pts with serial echos such that timing of surgical intervention in asymptomatic pts is based on specific echo parameters noted below (NB once pts are symptomatic you automatically treat)
- Maneuvers to determine how change in afterload/preload changes the murmur
  - 1st: Preload **(increased preload increases every murmur except HCOM and MVP therefore good to distinguish from AS and MS)**
    - Increase: Lying Down, Passive Leg Elevation, Squatting, Hydration, etc
    - Decrease: Standing, Valsalva (poor b/c pts often handgrip and do other stuff that complicates hemodynamics therefore don't do), Diuretics, Dehydration, Nitrates, etc
    - Important in distinguishing HCOM from AS, it is all about LV dimension in HOCM and blood volume in AS, in HOCM with increased preload there is increased LV dimension resulting in wider outflow tract therefore less HOCM murmur but in AS with increased preload there is increased LV dimension but the valve hasn't changed in its stenosis and with increased preload there is now more blood volume to pass thru the same stenotic valve therefore more AS murmur
    - Important in distinguishing MVP from MS, ?
  - 2nd: Afterload **(increased afterload decreases every murmur except MVP therefore good to distinguish from HOCM)**
    - Increase: Isometric Handgrip
    - Decrease: Inhaled Amyl Nitrate
- Tricuspid/Pulmonic Stenosis/Regurgitation (rare) L**E**ft = **E**xpiration Louder vs R**I**ght = **I**nspiration Louder
  - NB common causes unique to R heart are Congenital, Carcinoid, IVDU ABE, Rheumatic Heart Dz
  - TS (above) vs TR (above + Ebstein's Anomaly, s/p pacer lead placement and pulm HTN, LHF, chronic lung dz = most common valve abnormality!!!, hence to measure pulmonary pressure)
  - PS (above + Noonan's Syndrome) vs PR (above + pulm HTN)
  - NB inspiration (sucks blood into chest) results in increased venous return to right heart (Carvallo's Sign) which increases murmurs caused by right sided valve disease
- Other causes of murmurs
  - Congenital Heart Disease
  - Fistulas

Prosthetic Valves

- Types
  - Mechanical
    - more durable but thrombogenic requiring lifelong AC
    - Pt: young (<60yo) and will have good AC compliance
    - St. Jude BiLeaflet Tilting Disk INR 2.5-3.5, Single Tilting Disk INR 3-4, Caged Ball INR 4-5, etc
  - Biologic
    - less durable but only mildly thrombogenic requiring variable AC
    - Pt: old (>60yo) and has AC contraindications or young women desiring pregnancy
    - Autologous, Xenograft (Bovine/Porcine), etc
- PEx: very crisp sounds (absent then abnormal) with a soft murmur similar to the murmur seen in a native stenotic valve (regurg like murmur then abnormal)
- Tx: Coumadin (for all mechanical valves and for all biologic valves depending on RFs), baby ASA for all, IE prophylaxis when needed
- F/U: periodically check valve dysfunction by assessing Sx, change in murmur sounds, and embolic phenomena and checking labs for hemolytic anemia then proceed with TEE

- Complications: valve thrombosis, pannus formation, structure failure esp for biologic valves (30% w/in 10yrs), hemolysis, leak aka regurgitation, IE

Aortic Stenosis

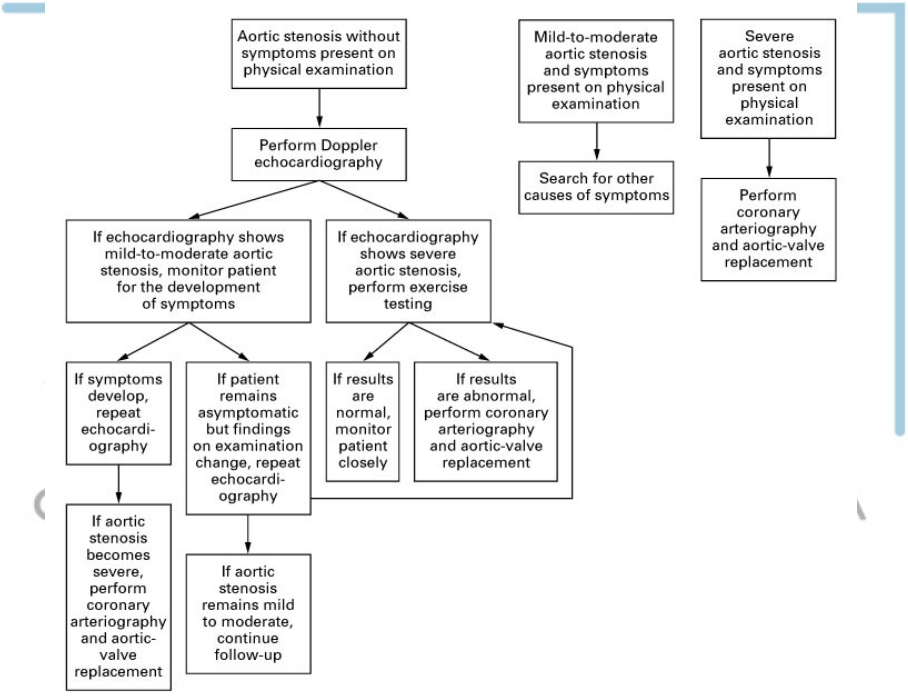


- Work Up
  - PEx: ejection click reflecting partial opening of stenotic valve followed by high pitched (diaphragm) harsh crescendo-decrescendo systolic murmur at RUSB (**later peak INDICATES SEVERE**) radiating to R Carotid w/ pt sitting up and leaning forward, b/c of delayed ejection the A2 component of S2 may fall on P2 during inspiration resulting in a single S2 and then follow P2 during expiration resulting in "paradoxic splitting" or the A2 dissipates such that S2 is soft or absent (**decreased S2 INDICATES SEVERE**), hypertrophy leads to S4, displaced PMI, etc., *Pulsus Tardus* (slow rising) *et Parvus* (low volume) of carotid pulse (**change in pulse INDICATES SEVERE**)
    - NB there are only 3 clinical findings that indicate severity and **loudness is NOT one of them**
  - CXR: CM, calcification, dilation of aortic root, signs of CHF
  - EKG: LVH, LAE, LBBB
  - Echo: Morphology (uni, bi, tri, quadri), Pressure Gradient, Jet Velocity, AV Area, LVEF, presence of calcification/thrombi
  - Cath: to more accurately calculate Pressure Gradient and AV Area but to also r/o CAD as seen in 60% of pts w/ AS 2/2 calcification

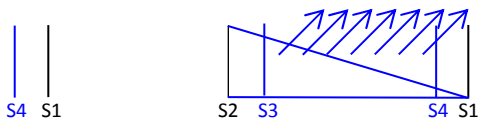
Stage	Pressure Gradient b/t LV and Aortic Root (mm of Hg)	Jet Velocity (m/s)	AV Area (cm^2)
Nl	0	1	3-4
Mild	<25	<3	1.5-2
Mod	25-50	3-4	1.0-1.5
Severe	>50	>4	"Severe" <1.0 "Critical" <0.7

- Etiology (good way to break it down is based on age)
  - **Child (<20yo) 5% Congenital Unicuspid or Quadricuspid** Valve such that the atherosclerosis process below is accelerated
  - **Adult (20-50yo) 25% Rheumatic** Heart Disease (M>A>>>>T>>>>P) suspect if also AI and mitral disease
  - **Old (50-70yo) 35% Congenital Bicuspid** (1/3 nl, 1/3 AS, 1/6 AI, 1/6 other) or **Malformed Tricuspid** Valve such that the atherosclerosis process below is accelerated
  - **Very Old (>70yo) 35% Calcific Degeneration** of a Normal Valve due to an Atherosclerotic-like process w/ the same type of RFs including smoking, HTN, DL, etc
  - Other: IE, Radiation Therapy, Paget's, Fabry's, End Stage Renal Failure (all rare)
  - NB **sclerosis** (aka leaflet thickening) occurs before stenosis (aka outflow obstruction) and has the same murmur but is asymptomatic nevertheless it is important b/c it has been associated w/ increased risk of CAD, progression from sclerosis to stenosis is slow
  - NB psuedostenosis occurs when the stenosis is mild but the leaflets are very sclerotic suggesting significant outflow obstruction when not really present, this can be differentiated thru dobutamine infusion tests during echo/cath
  - NB bicuspid valves have r/o ascending aortic aneurysm
- S/S
  - Cardinal Symptoms: **"532 ASF"**
    - Angina (1/2 will die in 5 yrs if no therapy) 2/2 prolonged ejection time → decreased diastole → decreased coronary filling → less oxygen supply AND increased wall thickness → more oxygen demand
    - Syncope (1/2 will die in 3 yrs if no therapy) 2/2 decreased outflow and arrhythmias
    - CHF (1/2 will die in 2 yrs if no therapy) initially there is mild outflow obstruction which leads to LVH (diastolic dysfxn) but after a while outflow obstruction worsens leading to LV dilation (systolic dysfxn)
    - And those pts who get a new valve approach the survival of the normal population
    - 10% r/o sudden death
  - Other Symptoms:
    - Arrhythmias esp **LBBB** 2/2 calcification of conduction system
    - **Heyde's/William's Syndrome** (acquired von Willebrand Dz 2/2 destruction of vWF from turbulent blood flow around valve resulting in GIB from an AVMs which otherwise would not bleed but they do b/c vWF is low, in addition it is theorized that the same underling connective tissue disorder of the AV that leads to AS is similar to the connective tissue disorder of the mucosa that leads to AVMs, nevertheless it is unclear)

- Tx
  - Algorithm: Tx if symptomatic don't Tx if asymptomatic just follow Sx and TTE Q6-12mo, the question is whether imaging findings (pressures, velocities, areas, LVEF, chamber size) correlate with symptoms, say they have symptoms but values aren't bad then still Tx, say they are asymptomatic but they have critical values (remember that there can be discordance as to what is normal parameters for a pt who is very small or big) then you need to question the history given by the pt and if pt still says asymptomatic then consider stress test to induce symptoms b/c you almost never have NO symptoms with critical values but if they truly have no Sx then don't do surgery but remember that 1% can die suddenly or have very rapid progression of Sx hence there is still some controversy but remember that surgery carries a 4% risk of death, in general Sx and values always correspond w/ Sx developing slightly before change in values therefore follow Sx… hence Stenosis = Symptom, if pt is getting heart surgery for other reasons then change out valve if severe but asymptomatic
  - Medical Therapy (bridge to surgery)
    - Decrease Afterload (but some people say don't!!!)
    - Maintain SR
    - Avoid Vigorous Exercise
    - New studies indicate that statins seem to stunt progression of stenosis given the fact that most cases are 2/2 a atherosclerosis-like process
  - Surgical Therapy (the definitive Tx, age is never a factor, if indicated you always want to replace the valve)
    - Aortic Valve Replacement (AVR)
    - If pt is non-surgical candidate or pt is critically ill and meds are not working before surgery then do percutaneous balloon valvuloplasty but not definitive Tx b/c 50% restenosis in 6-12mo and 10% peri-operative stroke
    - NB remember to always do a coronary angiogram to r/o CAD



Aortic Regurgitation/Insufficiency



- Work-Up
  - PEx: high pitched (diaphragm) soft blowing diastolic decrescendo murmur at LUSB if primarily valve dz or RUSB if primarily aortic root dz w/ pt sitting up and leaning forward, S3 due to atrial blood hitting a "pool" of arterial blood that regurgitated back into the ventricle during early diastole
  - Unique findings that suggest regurgitation:
    - *Austin-Flint Murmur* (regurgitant blood flows over anterior leaflet of the mitral valve causing narrowing and thus a murmur ~ to that seen in mitral stenosis) indicates that AR is severe

        - *Corrigan's / Water Hammer Pulse* (the regurgitant volume results in a lower DBP while the increase in SV b/c of the regurgitant volume results in a higher SBP thus widened pulse pressure w/ a bounding systolic pulse)
        - *de Mussett's Sign* (head bobbing w/ each systolic pulse)
        - *Muller's Sign* (uvula bobbing w/ each systolic pulse)
        - *Traube's Sign* (pistol shot sound over femoral artery when compressed distally)
        - *Duroziez's Sign* (systolic/diastolic murmur over femoral artery when compressed proximally/distally)
        - *Quincke's Sign* (capillary pulsations underneath nails w/ each systolic pulse)
        - *Hill's Sign* (popliteal-brachial SBP >60mm Hg)
    - CXR: CM, Aortic Root Dilation
    - EKG: LVH, LAE
    - Echo: parameters below but also helpful is the presence of flow reversal in descending aorta, aortic dilation, etc
    - Cath
    - MRI/CTA

Stage	Vena Contracta Width aka Width of Regurg at Orifice (mm)	Ratio of AR Jet Width to LV Outflow	Regurg Volume (mL)	Regurg Fraction (%)	Regurg Orifice (mm^2)
Mild	<3	<25	<30	<30	<10
Mod	3-6	25-65	30-60	30-50	10-30
Severe	>6	>65	>60	>50	>30

- Etiology
    - Acute
        - Primary Valve Disease: IE, Trauma
        - Primary Aortic Root Disease: Aortic Dissection or Aneurysm
    - Chronic
        - Primary Valve Disease: Rheumatic Heart Disease (M>A>>>>T>>>>P) suspect if also AS and mitral disease, Congenital Bicuspid, IE, SLE, RA, Increased Serotonin in Blood (Phen-fen, Amphetamines, DA Agonists, etc)
        - Primary Aortic Root Disease: systemic HTN resulting in dilation of the aorta, Inflammation (Syphilitic Aortitis, Relapsing Polychondritis, Behcet's Syndrome, Reiter's Syndrome, Takayasu, Ankylosing Spondylitis, Reactive Arthritis), CTD (Marfan's, Osteogenesis Imperfecta, Ehler-Danlos)
- S/S
    - Acute: LV cannot acutely compensate for the regurgitant volume and thus decreased SV therefore the heart attempts to maintain CO thru *tachycardia* but unsuccessful therefore *hypotensive* and blood backs up into lungs resulting in *dyspnea*, (pts appear quite ill)
    - Chronic Compensation: usually asymptomatic for years b/c the heart compensates by dilating to accommodate the extra volume and hypertrophies to pump this volume out (aka eccentric hypertrophy)
    - Chronic Decompensation: eventually the heart fails
    - Symptomatic especially during bradycardia b/c of the long diastolic period
    - CHF occurs in 50% of pts 10yrs after the dx of severe AR and once developed NYHA II/III-IV have an annual mortality of 5/25%
- Treatment
    - Acute: surgical emergency as likely severely symptomatic

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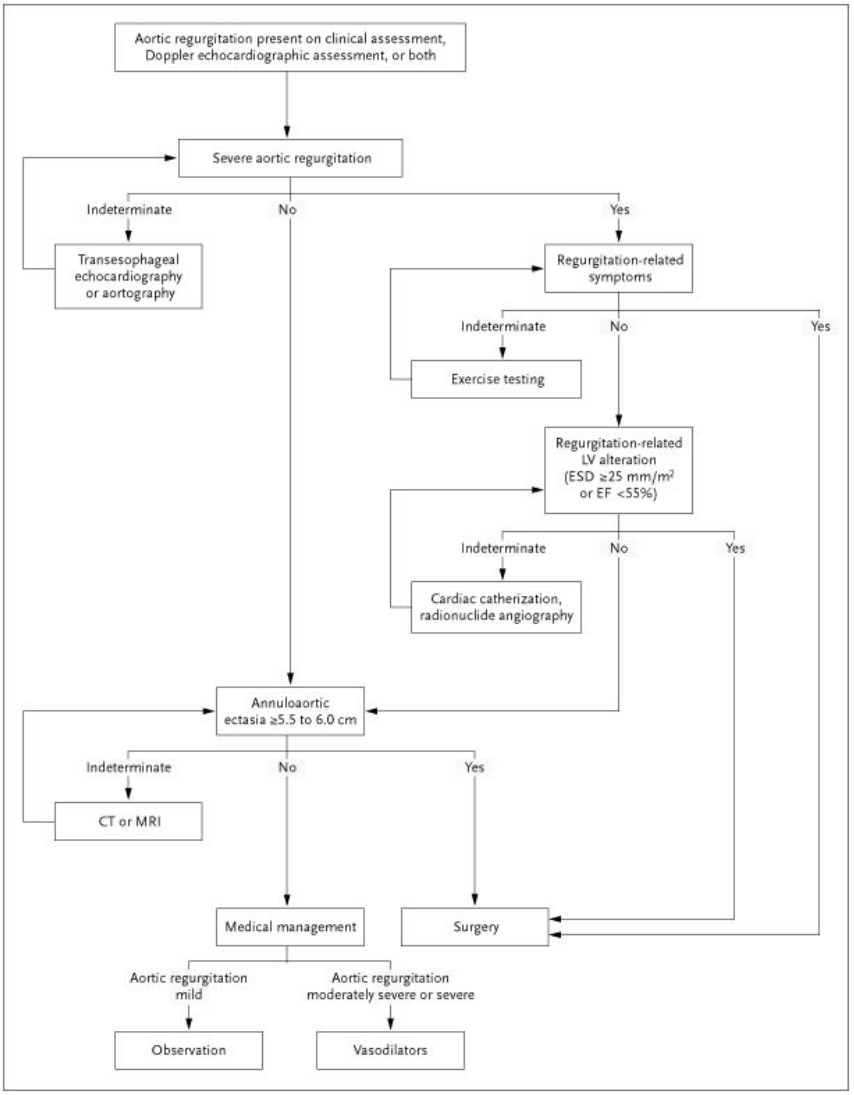
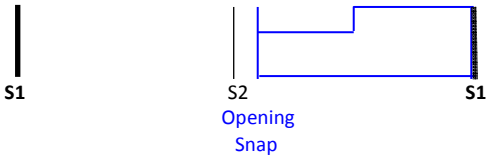


- Chronic (above)
  - Mild AR (no need to follow), Mod AR (Qyr TTE), Severe AR (refer above)
  - Assess Severity Parameters (above in table), LV Fxn/Anatomy (EF and dimensions), and presence of annuloaortic ectasia on TTE and if equivocal then TEE, Aortography, Cath, Angiography, CT, MRI, etc
  - If symptoms are equivocal then exercise pt, if the causal link b/t symptoms and AR is uncertain then rule out other causes and if ruled out then proceed with surgery
  - Surgical Options: aortic valve replacement, valve repair, or valve replacement with pulmonic valve aka Ross procedure along w/ aortic root replacement if needed
  - The important thing to note is that if an asymptomatic pt don't delay surgery until symptoms rather serially check LV fxn/anatomy w/ echo (LVEF <50%, LV end-systolic dimension >55mm, or LV end-diastolic dimension >75mm), serially check aortic dilatation (>55mm), and find out if pt is already going to get heart surgery for other reasons, if so then go ahead with surgery as many studies have shown that delaying surgery until symptoms present is associated w/ higher mortality/morbidity
  - If the pt is truly not in need of surgery and pt has mod-severe AR then medically manage if pt has mild AR then just observe
    - Decrease Afterload w/ ACEI/ARB (but NO IABP) and Increase Chronotropy (decreases diastolic period allowing less time for regurgitation)
    - There is a 5%/yr progression of asymptomatic but severe AR to symptomatic severe AR therefore with medical treatment close f/u is necessary

Mitral Stenosis

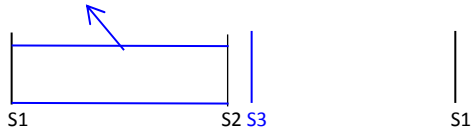


- Work-Up
  - PEx: opening snap (sudden full opening of valve) followed by low pitch (bell) diastolic rumble w/ pre-systolic accentuation at apex (esp w/ pt on L lateral decubitus position and right after exercising), **shorter S2-OS interval and thus longer diastolic murmur = more severe, louder = more severe**, loud S1, load P2 and R heave
  - CXR: LAE, RVH
  - EKG: LAE, RVH, AFib
  - TE/TE: decreased valve area opening aka "fish mouth" w/ "hockey stick" leaflets (Nl 4-6cm^2)
  - Cath: PASP (Nl <25 mm of Hg) and mean transvalvular diastolic pressure gradient (NL 0 mm of Hg) also good to assess CAD and helpful if echo equivocal

Stage	Gradient	MV Area	PA Systolic
Nl	0	4-6	<25
Mild	<5	1.5-2	<30
Mod	5-10	1-1.5	30-50
Severe	>10	<1	>50

- Etiology
  - Rheumatic Heart Disease (most common nonetheless MS is very rare in the US, pt in US usually present 15yrs after pharyngitis, most MS in the US are immigrants, ½ pts remember pharyngitis and RF)
  - Other: Congenital, Myxoma protruding through mitral orifice, IE, Large Mitral Annular Calcific Deposits associated w/ LV outflow obstruction, Valvulitis from SLE, Carcinoid, etc, Infiltration from mucopolysaccharidoses (all exceedingly rare)
- S/S
  - Chronic: progressive S/S consistent w/ low systemic CO along with back up into lungs and hemoptysis
  - Acute: when pt develops Afib from LAE there is a sudden CHF exacerbation
- Treatment
  - Treat when pt symptomatic or MVA (<1.5cm^2) or PASP (>60 mm of Hg aka Pulm HTN) at rest or Transvalvular Diastolic Pressure Gradient (>15 mm of Hg) when exercising otherwise repeat clinical visit Q6mo???
  - Medical Therapy: diuresis (why? b/c they have CHF w/ back up into lungs), BB/CBB to lower HR as much as tolerated (why? to allow enough diastole time for blood to leave atrium and go into ventricle), avoid exercise, anticoagulation, endocarditis prophylaxis, rhythm control or concurrent LA radiofrequency ablation to prevent or Tx Afib (AFib is bad b/c then there is no atrial kick to push blood thru stenotic valve)
  - Surgical Therapy: percutaneous transseptal balloon valvotomy/valvoplasty (initial treatment if NO calcification, NO involvement of subvalvular tissue, NO regurgitation, NO left atrial appendage thrombus), open surgical commissurotomy (if -plasty fails or if contraindications above are present or if primarily commisures are affected), mitral valve replacement (if –plasty fails or if contraindications above are present or if primarily chordae tendinae / papillary muscle are affected)
  - MS often exaggerbates during pregnancy resulting in bad Afib, Tx in pregnancy is limited to w/ digoxin/verapamil (no BB?), heparin (no Coumadin) and cardioversion (is actually safe!!!)

Mitral Regurgitation/Insufficiency

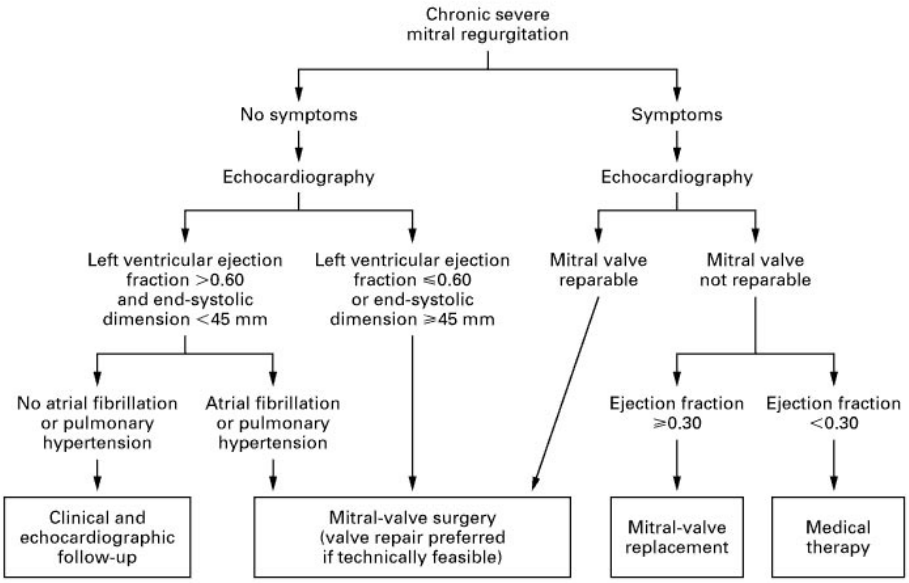


- Workup
  - PEx: high-pitched (diaphragm), blowing, holosystolic murmur at apex radiating to back/clavicular/axilla depending on which leaflet is involved, S3
  - CXR: LAE, LVH
  - EKG: LAE, LVH, Afib
  - Echo:

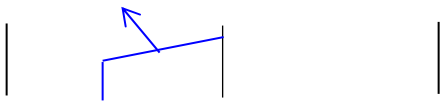
Stage	Regurg Fraction	Jet Area	Jet Width	ERO	Angio
Mild	<30%	<20	<0.3	<0.2	LA clears w/ each beat

Mod	30-50%	20-40	0.3-0.70	0.2-0.40	Does not clear w/ faint opacity after several beats
Severe	>50%	>40	>0.70	>0.40	LA and LV opacity

- Etiology
  - Acute
    - Papillary Muscle Rupture from Infarction (usually posteromedial muscle b/c supplied by only the PDA while the anterolateral muscle is supplied by Diagonals from LAD and OMs from Cx)
    - Chordae Tendinae Rupture from trauma, IE, just spontaneously, etc
    - Flail Leaflet from IE, trauma, etc
  - Chronic
    - MVP
    - Chronic Ischemia resulting in Papillary Muscle Dysfunction
    - Rheumatic Heart Disease
    - there is stretched tissue (myxomatous degeneration) b/c of connective tissue disorders (Marfan's, Ehlers-Danlos, etc)
    - LV Dilation Cardiomyopathy (refer)
    - Anorectic Drugs like Phen-Phen
- S/S
  - Acute: S/S consistent w/ low systemic CO along with back up into the lungs
  - Chronic: asymptomatic as the LV compensates by dilating to take on more volume but then it begins to fail, also there is chronic LAE resulting in Afib
  - Progression from dx to Sx is 16yrs
  - Pts with severe symptomatic MR have 5%/yr mortality due to CHF and ventricular arrhythmias b/c of ventricular dilation
- Treatment
  - Treat when pt symptomatic or EF <60% or LV end systolic diameter <40mm or pulmonary HTN or Afib otherwise repeat TTE Q6-12mo and clinical visit
  - Medical Therapy: decrease preload and decrease afterload (which promotes ventricular blood into aorta and not LA) (overall medical therapy is controversial, only shown to decrease Sx NOT alter course of dz), Tx of Afib, IE prophylaxis, treat the underlying problem that caused MR especially if the cause is ischemia b/c revascularization has been found to decrease MR
  - Surgical Therapy: if still symptomatic despite medical Tx then surgery, percutaneous/surgical valve repair (if anatomy not suitable then replacement but otherwise don't b/c when you replace you have to cut papillary muscles and it turns out that intact papillaries are important in LV fxn such that when cut there is a 10% decrease in EF) if EF is <30% surgical correction is poor therefore just medically manage
  - Mild MR (nothing)
  - Mod MR (TTE Qyr)
  - Severe MR (refer)



Mitral Valve Prolapse



S1 Click S2 S1

- Workup
  - PEx: midsystolic click representing the snapping of tendineae followed by a late systolic murmur (some pts have click and no murmur or vice versa or both) at apex radiating to back/clavicular/axilla depending on which leaflet is involved, earlier Click & Longer/Harder Murmur (more severe) vs Later Click & Shorter/Softer Murmur (less severe)
  - CXR: nothing
  - EKG: nothing
  - Echo: displacement/prolapse of leaflets >2mm above mitral annulus into L atrium in the parasternal long axis view during midsystole
- Etiology
  - Chronic
    - Mild Form: normal redundant tissue of chordae tendinaea for no apparent reason but interestingly pts often have various associated conditions like von Willebrand's Dz and other seemingly random disorders (less likely to progress to MR)
    - Severe Form: abnormal myxomatous changes of valve leaflets and chordate tendinaea (more likely to progress to MR)
- S/S
  - very common (7% of population, mild form mainly young females vs severe form mainly in old males)
  - usually an incidental finding on PEx as pts are usually asymptomatic
  - although benign pts are at slightly high r/o developing IE, MR, or tachyarrhythmias
- Treatment
  - Reassurance
  - Observation for IE, MR, and tachyarrhythmias

Rheumatic Heart Disease

S/S of Acute Rheumatic Fever (**J♠NES** Major Criteria) (in order of incidence) RF is called RHD if the heart is involved

- **J**oint 70%
  - migratory (affecting a new joint as past affected joints resolve) and polyarthritic (affecting more than one joint at a single point in time) of large joints but joints of spine and cranium can also be affected, lasting <1mo, Tx: aspirin x1mo
- **♠** Symptoms 50%
  - Pericarditis: exudation of fibrin rich fluid creating a "bread-butter" appearance and friction rub on auscultation, usually complete resolution with only few adhesions
  - Myocarditis: Aschoff Bodies on Bx composed of Anitschkow Myocytes (large myocytes), Aschoff Cells (multinucleated giant cells), and Collagen resulting in arrhythmias
  - Endocarditis: @ points of greater hemodynamic stress (points of valve closure) verrucae (platelets/fibrin) form which are non-friable (hence do not embolize)

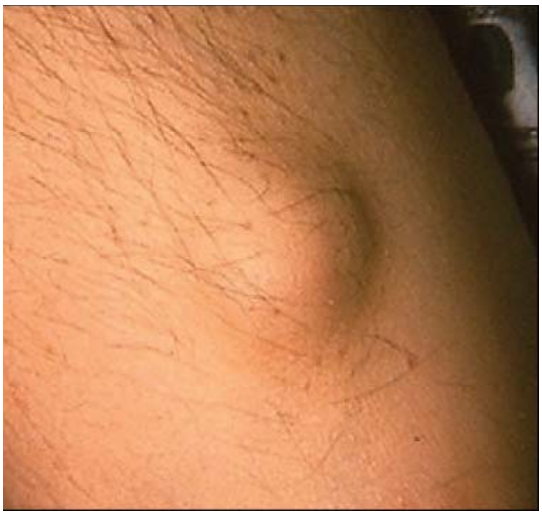
(most pts have combination of both regurgitation and stenosis)

M>A>>>>T>>>>P "MAT"

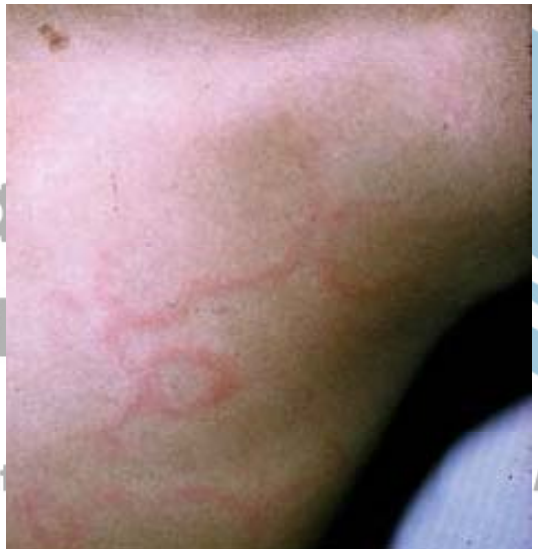
a. Tx: aspirin x1mo and add steroids x1mo if CHF

(2) **N**odules 10%

a. Description: hard, painless, small (0.5-1cm) subcutaneous nodules over bony prominences esp extensor tendons of hand, scalp, spine



(3) **E**rythema Marginatum 10%
   a. Description: clear center w/ serpiginous erythematous ring, nonpruritic, migratory, evanescent (disappears when cold then reappears when warm) found on trunk and proximal extremities, never on face



(4) **S**ydenham's Chorea 10% "St. Vitus' Dance"
   a. Description: initially psych issues (emotionally labile, ADHD, OCD) then motor issues (loss of motor coordination, "milk-maid" hands b/c hands look like milking cow, spontaneous purposeless movement, motor weakness)
   b. very common in prepubertal girls
   c. Lasts 7-17mo!!!

Minor Criteria
(1) F
(2) ↑ APRs
(3) Arthralgia
(4) Prolonged PR
(5) Hx of Rheumatic Fever or Streptococcal Pharyngitis w/ +ASO

Definite Rheumatic Heart Disease
- Clinical Criteria AND evidence of preceding Group A strep infection
   - 2 Major OR
   - 1 Major + 2 Minor
- Dx & Tx (refer to pulm section)

Infective Endocarditis (IE)

- Mechanism: prosthetic valves or natural valves that are damaged 2/2 degenerative, congenital (MVP), acquired processes or any other structural problem to heart such that endothelial injury 2/2 abnl flow resulting in sterile platelet/fibrin adherence forming verrucae → skin/mucosa breakdown during dental cleaning, GI/GU procedures, surgery, IVDU (50% TV 20% AV 20% AV, adulterants in the injected drug like cotton), IV catheters, pacemakers, cardiac surgery, post-op wound infections, etc introduces bacteria into blood → bacteria adhere to verrucae and begin to destroy valve as a vegetation forms (change in murmur)
- Classification
    - Native (*Strep viridians* (most common SBE), *Staph aureus* (most common ABE), *Enterococcus* (GI malignancy, GI/GU procedure), *Strept bovis* and *Clostridium septicum* (GI cancer), GNR esp Psuedomonas, Salmonella, Serratia, Gonorrhea (nosocomial))
    - Prosthetic (<2mo *Staph epi* vs >2mo similar to native) Risk: 3% at 5yrs, 5% at 10yrs esp mitral valve
    - IVDU (Staph, Candida)
    - Culture Negative
        - Slow Growers
            - HACEK (fastidious bacteria that require several weeks of incubation, tend to produce very large friable vegetations such that embolization is very common, SBE, part of normal GI flora, IVDU, high r/o CHF and embolic phenomena)
                - *Haemophilus* spp.
                - *Actinobacillus actinomycetemcomitans*
                - *Cardiobacetrium hominis*
                - *Eikenella corrodens*
                - *Kingella kingae*
            - Fungi (subacute course, fastidious fungi requires several weeks incubation for isolation from blood therefore suspect if cultures are negative but you are sure that pt has IE, tend to produce very large friable vegetations such that embolization is very common, often invade myocardium)
                - *Candida*
                - *Aspergillus*
            - Bacteria
                - *Bartonella hensilae/quintana/elizabethae* (check serology, SBE, homeless pts)
                - *Trophopheryma whipplei* (homeless pts, transmitted via body lice, check serology)
                - *Coxiella burnetti* (check serology, immunosuppressed pts, SBE)
                - *Listeria* (check serology, immunosuppressed pts)
        - True Culture Negative
            - Prior Abx Use
            - Rheumatic Endocarditis
            - Carcinoid Syndrome Endocarditis (found in pts with carcinoid syndrome, sterile deposits on R sided valves)
            - Libman-Sacks Endocarditis (found in pts with SLE, sterile deposits on both sides of aortic valve)
            - Marantic Endocarditis (found in pts with debilitating illnesses like metastatic cancer w/ mets seeding valves, sterile deposits of fibrin and platelets on valves, poor prognosis)

"The 1994 Duke Criteria"
2 Major OR 1 Major + 3 Minor OR 5 Minor

Major Criteria

(1) Evidence of Bacteremia
    a. Positive Blood Culture (draw from different sites, not thru a line, incubate for >21d)
        i. Before Abx: ≥3 spaced >1hr apart OR ≥2 spaced >12hrs apart
        ii. After Abx: ≥2 spaced >1hr apart
    b. Positive Serum Serology (1:1800) w/ an organism known to cause IE
(2) Evidence of Heart Involvement
    a. Clinical: New Regurgitation
    b. Echo: Vegetation, Abscess, Prosthetic Valve Dehiscence, New Regurg
        i. Get a TTE and then a TEE if prosthetic valve, nondiagnostic TTE, negative TTE but suspicion still very high, suspect progressive disease

Sensitivity	NVE	PVE	Abscess
TTE	65%	25%	28%
TEE	90%	90%	87%

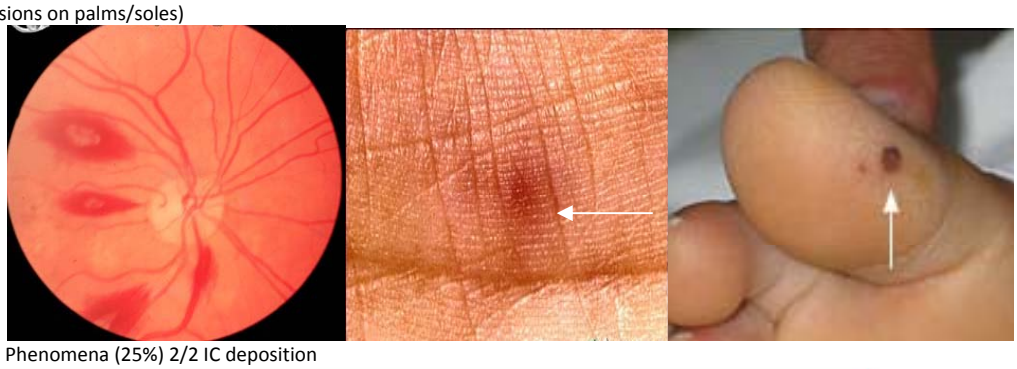
Minor Criteria **"F♠B-VIP"**

(1) Persistent **F**ever w/ other constitutional symptoms as the infected thrombus acts as a reservoir for bacteria (95%)
(2) Evidence of **Heart** Involvement not meeting Major Criteria (85%)
    a. Arrhythmias (get an EKG)
    b. CHF (get an Echo)
    c. Myo/Pericarditis

d. Abscesses

(3) Evidence of **B**acteremia not meeting Major Criteria (85%)

(4) **V**ascular Phenomena (25%) 2/2 septic emboli that can turn into abscesses/infarction
   a. CNS (AMS, Deficits, etc)
   b. Heart (MI, Mycotic Aneurysms, etc)
   c. Kidney (RF)
   d. Spleen (Infarct)
   e. Lung (Infarct)
   f. Skin (Mucosal Petechiae, Splinter Hemorrhages, Subungual Petechiae, Janeway's Lesions = painless, flat erythematous lesions on palms/soles)



(5) **I**mmunologic Phenomena (25%) 2/2 IC deposition
   a. Kidney (Glomerulonephritis)
   b. Joint (Arthritis)
   c. Lab (+ RF)
   d. Skin (Osler's Nodes = painful "Ouch that's painful", raised purple lesions on finger/toe pad)
   e. Eye (Roth's Spots = oval, retinal hemorrhages with a clear, pale centers)

(6) **P**redisposition
   a. Abnormal Valve, IVDU, Distant Infection, Indwelling Venous Catheter, Poor Dentition, HD, DM

General Presentation

- ABE (Acute Bacterial Endocarditis, typically normal valves w/ very aggressive pathogens) w/in wks h/o overt constitutional symptoms and cardiac symptoms
- SBE (Subacute Bacterial Endocarditis, typically damaged/prosthetic valves w/ less aggressive pathogens) w/in months h/o vague constitutional symptoms (low grade F, anorexia, weight loss, flu-like Sx, pleuritic cp, RU/LQ pain), vascular phenomena and immunologic phenomena

Antibiotic Therapy

- Empiric Treatment
   - NVE: Naficillin (Vanc if pen allergic or high prevalence of MRSA, Pen or Amp if SBE) + Gentamycin
   - PVE: Vancomycin + Gentamycin + Rifampin
- Synergy b/t penicillins and aminoglycosides
- Pts should be hospitalized for 2wks to monitor complications and to see that treatment is working
- B/c pts will be getting >1wk of an aminoglycoside they will need baseline/follow-up audiometry
- Fever may persist up to 1wk after appropriate abx treatment
- high dose bactericidal IV (not PO for any part of the treatment) antibiotics for 4-6wks starting after first set of negative BCx x2 hence check BCx Q1-2d until negative
- avoid anticoagulation b/c of high r/o hemorrhagic transformation of embolic strokes except for pts w/ prosthetic valves
- difficult to Tx b/c bacteria lie deep in verrucus
- Bacteremia
   - High Risk: anything not below and remember CPS aka S. aureus is never a contaminant and always check a TTE to r/o IE
   - Low Risk (is usually contaminant): CNS aka *S. epi*, *Propionibacterium spp*
   - Start w/ vanc
   - if pt just has bacteremia and no IE than treat for 2wks from first negative blood culture
   - d/c catheters/lines and Cx tips always for Staph but not always for Strep
   - if persistent consider infected thrombus

Surgical Therapy

- 20% of pts need surgical intervention
- Often done months after sterilization of verrucus
- imminence is hard to assess but is based on:
   - Hemodynamic Instability: CHF refractory to medical therapy (most common)
   - Perivalvular Invasion: conduction disturbance 2/2 abscess, metastatic infections, mycotic aneurysm
   - Big Vegetations: >1cm, recurrent systemic emboli

- Hard To Eradicate Bugs: Culture negative or abx resistant pathogens, persistent bacteremia despite appropriate antibiotic therapy (>72hrs)
- Prosthetic Valve: dehiscence, placed <2mo ago, Staph

Prophylaxis (only 20% of IE is due to invasive procedure)

- If the pt will be undergoing one of the following procedures:
  - Any upper surgery like Dental/Respiratory (GI/GU no longer require prophylaxis at all)
  - NB prophylaxis is not needed for pts who are undergoing device placement, catheterization, CABG
- And if the pt has one of the following risk factors: (NO = ASD, VSD, AS, MVP, HCM)
  - Prosthetic Valve
  - Prior IE
  - Congenital HD: unrepaired cyanotic, repair w/in last 6mo, repair but still w/ residual defects, repair w/ prosthetic material/device
  - Heart Transplant that subsequently develops valvuloplasty
- Then give antibiotic prophylaxis:
  - Amoxicillin 2g PO 1hr prior OR
  - Amp 2g IV 30min prior (if pt cannot tolerate PO) OR
  - Clinda 600mg PO 1hr prior / 600mg IV 30min prior or Azithro/Clarithro 500mg PO 1hr prior (if pen allergic)
- NB most important is good oral hygiene as degree of gingivitis is proportional to risk of bacteremia