UPMC Cardiology Handbook for Residents

Version 2022

Editors' Note and Acknowledgments

We are thrilled to introduce the Second Edition of the UPMC Cardiology Handbook. This handbook represents the hard work and dedication of 26 UPMC residents. A special thanks to Dr. Michael Bashline and Dr. Joshua Levenson for taking us under their wings as the supportive cardiology fellow and attending in this endeavor.

We have collaborated to collate clinical information as well as logistical information of providing cardiac care at UPMC. Our aim is that this collection of evidence-based recommendations and institution-specific pearls helps you in your day-to-day practice of clinical care, whether looking up something in a pinch or reading a section to brush up on a topic. We hope that this handbook serves as a tool to facilitate evidence-based, quality patient care, a reference point to guide continued learning, and an inspiration to future residents of what we can accomplish when we work together.

Priyanka, Scott, & Harnoor

August 20, 2022

Priyanka Solanki, MD
Class of 2024, Internal Medicine
UPMC

Scott Baumgartner, MD
Class of 2023, Internal Medicine
UPMC

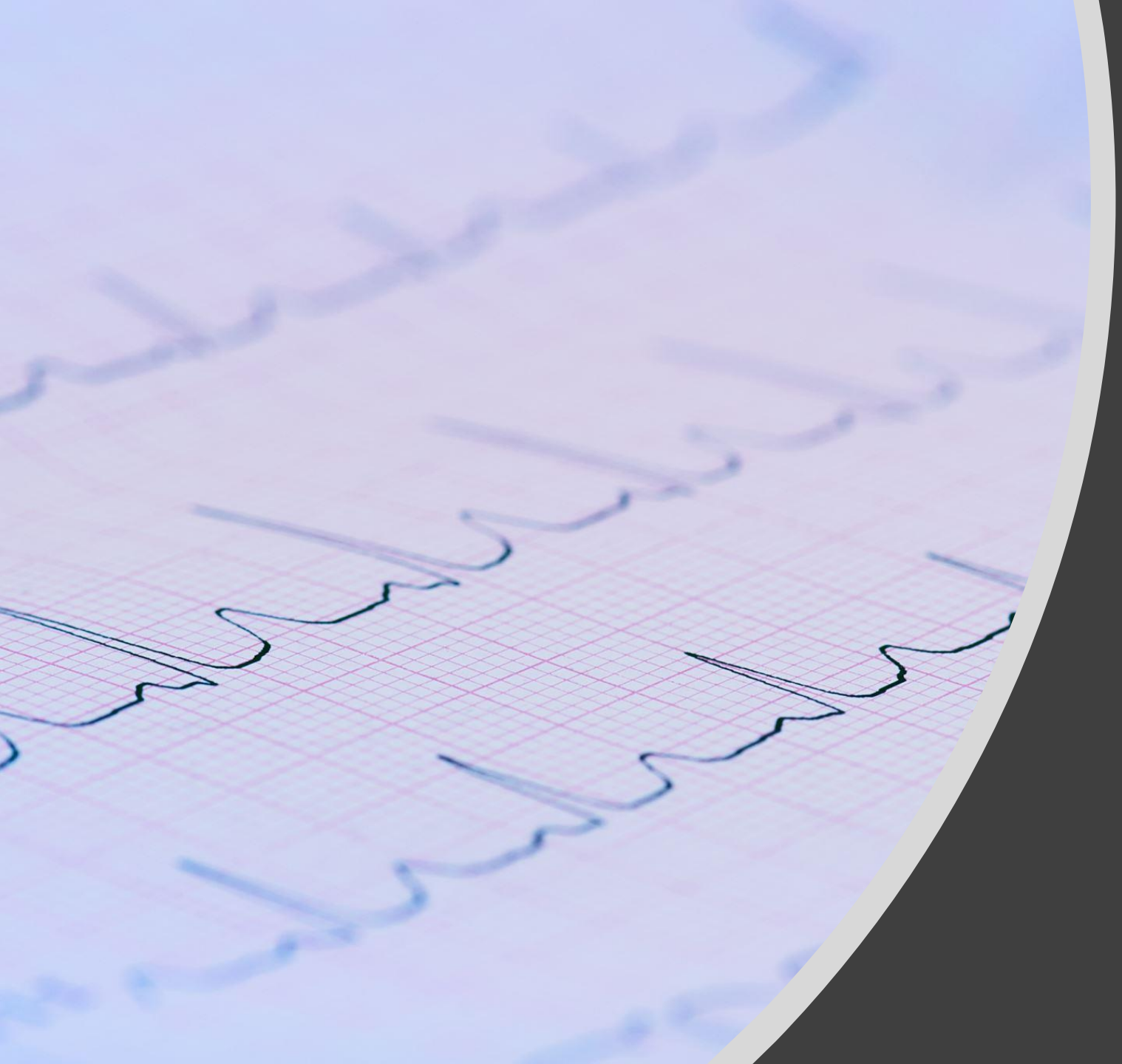
Harnoor Mann, MD
Class of 2024, Internal Medicine
UPMC

Table of Contents

Table of Contents

Table of Contents

Table of Contents



Cardiology Rotation Info

Section Editor:
Harnoor Mann, MD

Cardiology Rotation Guide

Pavilion Structure

- 4 intern-resident teams with 1 elective rotating resident (Recommend that you discuss days off the week prior! Can be tricky to coordinate with elective rotator's clinic day)
- Rotation split between: "Pavilion" (general cardiology), Advanced Heart Failure service, Night Float
- NF admissions are presented by the day team to the proper attending (Pavilion/general vs Heart Failure

CCU structure

- 3 intern-resident teams
- Fellow coverage weekdays, Saturday AM, and nights (7 days/week)
- Closed unit: cardiology attending must accept all patients
- Pulmonary consultation on vented patients (unless Cards CCM attending) and ICU overflow

<table>
<tr><th>Service (admit pager)</th><th>Attending</th><th>7a–4p</th><th>4p–6p</th><th>Nights</th><th>Weekends</th></tr>
<tr><td>EP (33342)</td><td>EP/private</td><td>EP APPs</td><td>EP fellow</td><td rowspan="2">On-call fellow</td><td>AM: EP APPs, then on-</td></tr>
<tr><td>Intervnt'l (7008)</td><td>Private model</td><td colspan="2" rowspan="2">Interventional/CHF APPs</td><td rowspan="2">AM: Interv/CHF APPs, then on-call fellow</td></tr>
<tr><td>HF/APP (7008)</td><td rowspan="2">CHF</td><td rowspan="2">Night float (7p-7a)</td></tr>
<tr><td>Pav/HF (9393)</td><td colspan="2">Pav/HF team</td><td>Pav/HF team</td></tr>
<tr><td>Pav 1&2 (6363)</td><td>Pavilion</td><td colspan="2">Pav 1&2 teams</td><td>Pav 1&2 teams</td></tr>
</table>

Team Contact Information

CCU Intern	802-6622
CCU Resident	802-6621
CCU Fellow	802-6623
Pulm Fellow	864-2942
Pulm Fellow (7p-7a)	647-2295
Pavilion 1 Intern	6262
Pavilion 1 Resident	6363
Pavilion 2 Intern	7575
Pavilion 2 Resident	7373
Pavilion/CHF Intern	9595
Pavilion/CHF Resident	9393
Cards Fellow On-call	864-1916
CHF Fellow (7:30a-6p)	864-1917
Consult Fellow	Medtrak
CHF Consult Fellow	Medtrak
EP Fellow	Medtrak
Interventional/CHF APP	7008

Pagers and Phone Numbers

Pavilion Rotation
Pav 1 Intern: 6262
Pav 1 Resident: 6363
Pav 2 Intern: 7575
Pav 2 Resident: 7373
Pav Fellow: 864-1916
CHF Intern: 9595
CHF Resident: 9393
CHF Fellow: 864-1917
5D Pharmacy: 148-6642

CCU Rotation
CCU Intern: 802-6622
CCU Resident: 802-6621
Pulm Fellow (days): 864-2942
Pulm Fellow (nights): 647-2295
CCU Respiratory Therapy: 692-4267
CCU Pharmacist: 146-8718

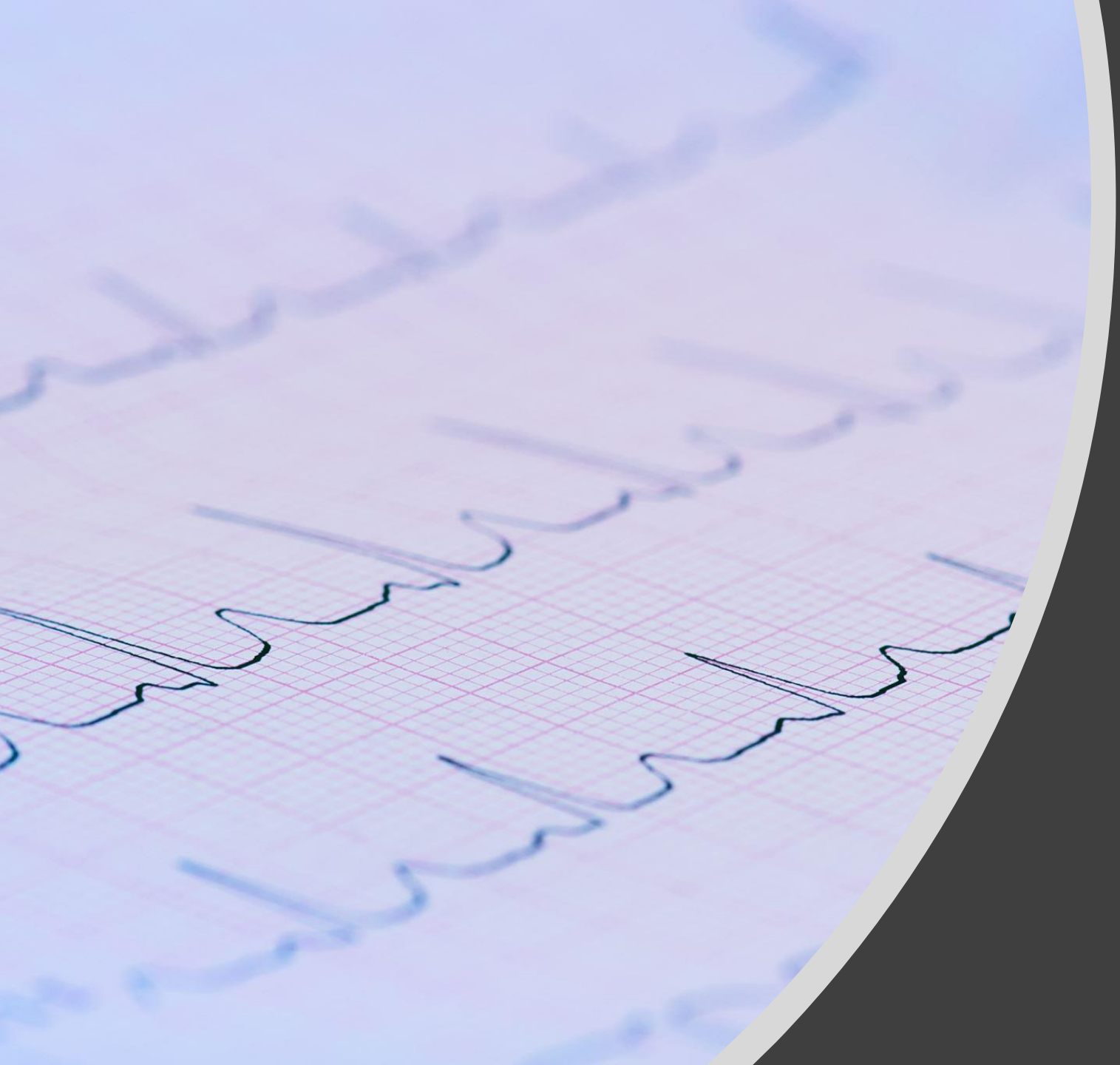
Cardiology Subspecialty Services
Nuclear Cardiology: 647-7142
Cath Lab Scheduling: 647-9000
TTE Service: 647-6175
TEE Service ?fellow? 647-0103
Cardiac MRI: 864-3333
EP Lab: 647-3651
TAVR (Lisa): 951-4396
Event Monitor: 647-3425

Advanced Practice Services
Cards Fellow On Call 8641916
Interventional: Pager 7008
EP Pager: 33342
Heart Failure: 7008
Overnight Coverage: 7008

Nursing Stations:
3E: 647 - 3034
4D: 647 - 8027
5D: 647 - 0900

General Numbers:
Inpatient Pharmacy: 647-3350
Portable XR: pager 6793
Inpatient Lab: 647-5227
ED: 647-3334
RT1:692-4322
RT2:692-4256

Cardiac Surgery
Days: 605-0448 or 605-0446
Consults: 605-0447
CT NF Resident: 864-2240
CTICU NF Resident: 864-2241
Thoracic Surgery: 864-0937
Heart Transplant NP (days): 648-5431
Heart Transplant Fellow/Attending: pager 8919



General Cardiology

Section Editor:
Priyanka Solanki, MD

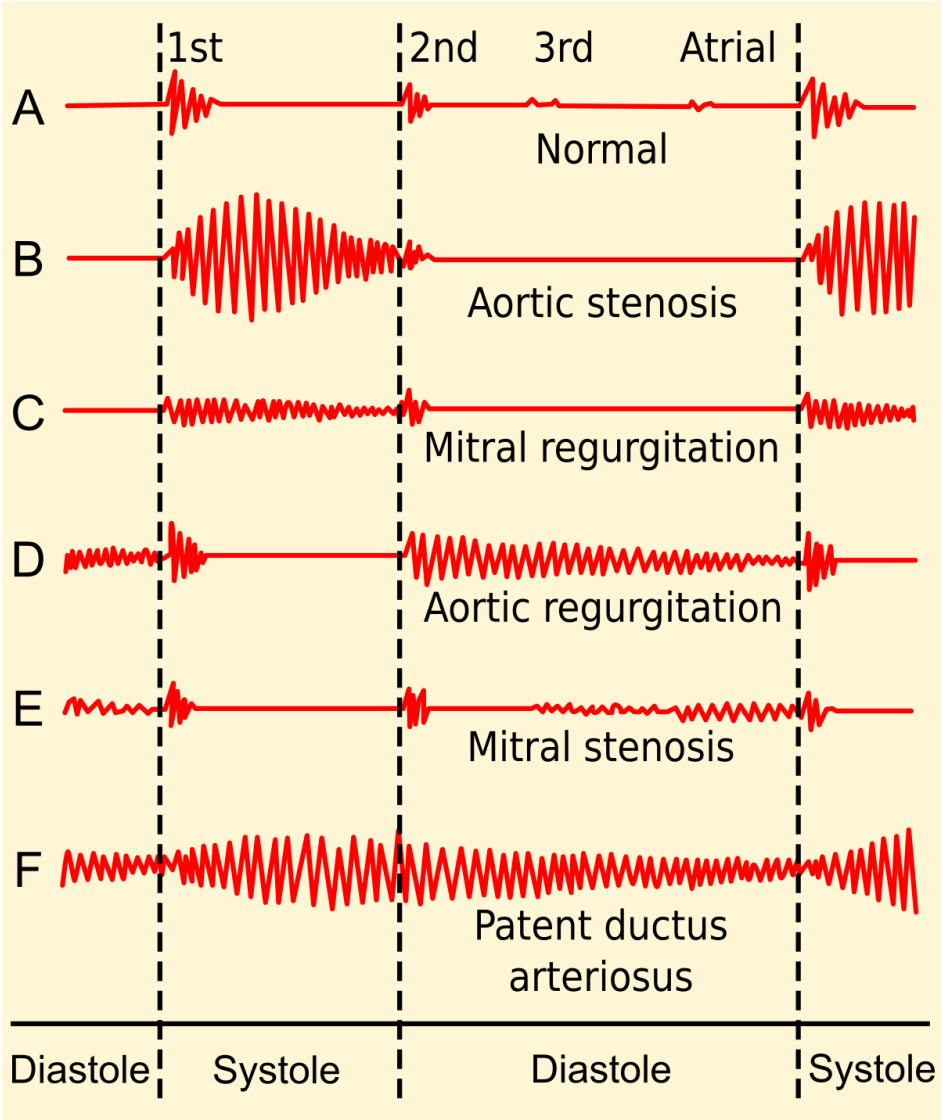
Cardiac Physical Exam

Jugular Venous Pressure (Normal 7-9 cm)	
Steps:	
1	Head of bed at 30° angle
2	Rotate neck to left or right
3	Apply hepatojugular reflux (press on RUQ) and watch pulsation rise
4	Measure from top of pulsation to sternal angle + 5cm
***Remember: JVP is biphasic and changes with respiration unlike the carotid pulse	

Pitting edema scale		
Score	**Indentation**	**Rebound**
+1	≤2 mm	Instantly
+2	2-4 mm	10-15 sec
+3	4-6 mm	<1 min
+4	6-8 mm	Several minutes

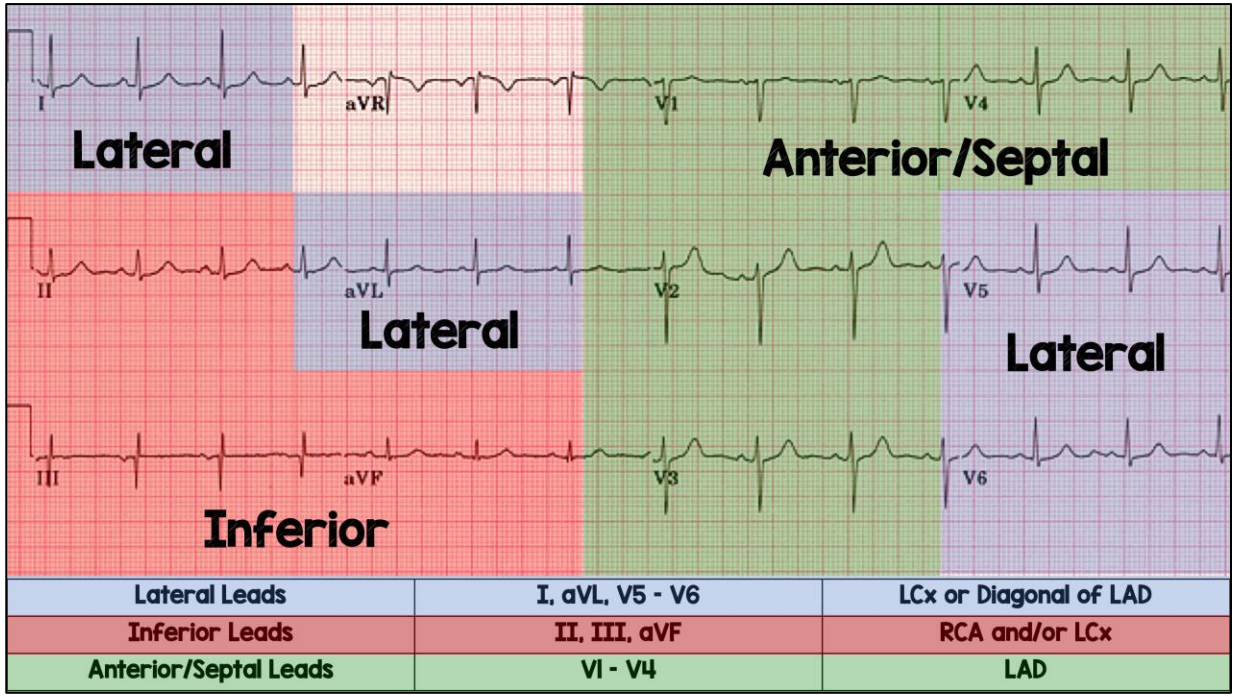
Murmur Grading	
Grade	**Description**
I	Almost inaudible
II	Soft without thrill
III	Moderate without thrill
IV	Loud with palpable thrill
V	Very loud with light touch of stethoscope
VI	Audible without stethoscope

Cardiac Physical Exam



Extra Heart Sounds
S3 (KEN-tuck-y): associated with heart failure, dilated cardiomyopathy, L to R shunts; normal <40yrs
S4 (Te-NNE-ssee): associated with LVH, hypertrophic cardiomyopathy, aortic stenosis

EKG Highlights

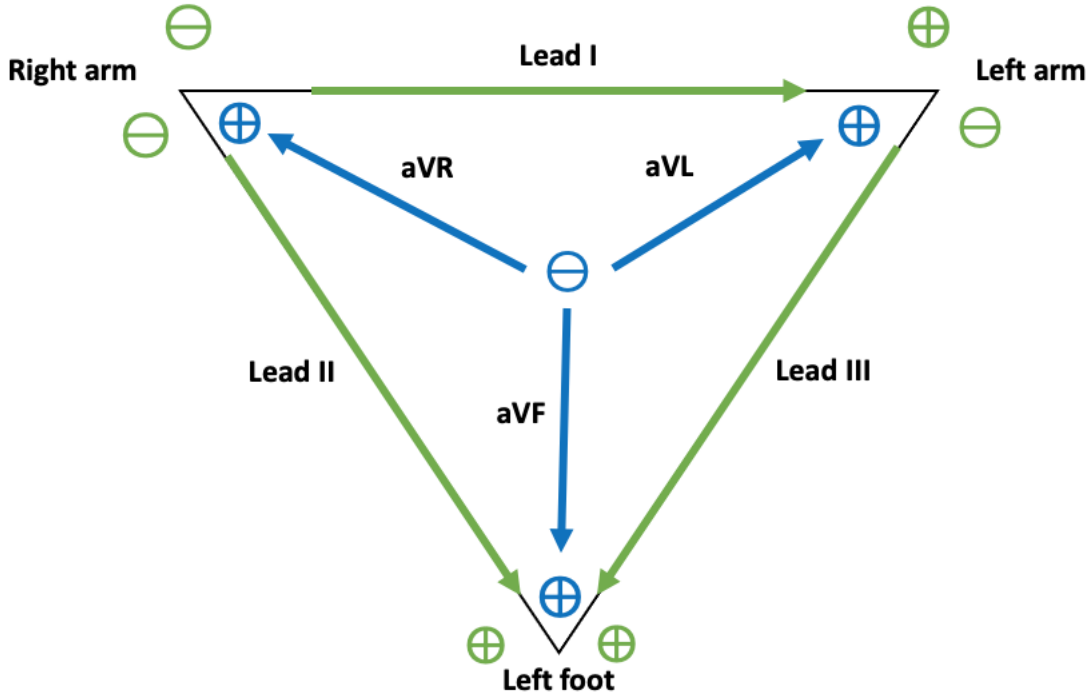


Lateral Leads	I, aVL, V5 - V6	LCx or Diagonal of LAD
Inferior Leads	II, III, aVF	RCA and/or LCx
Anterior/Septal Leads	V1 - V4	LAD

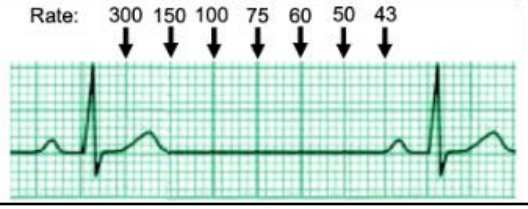
Leads	
Limb Leads	I-III, aVR, aVL, aVF
Precordial Leads	V1-V6

Einthoven's Triangle

Describes electrical conduction measured by EKG



EKG Highlights

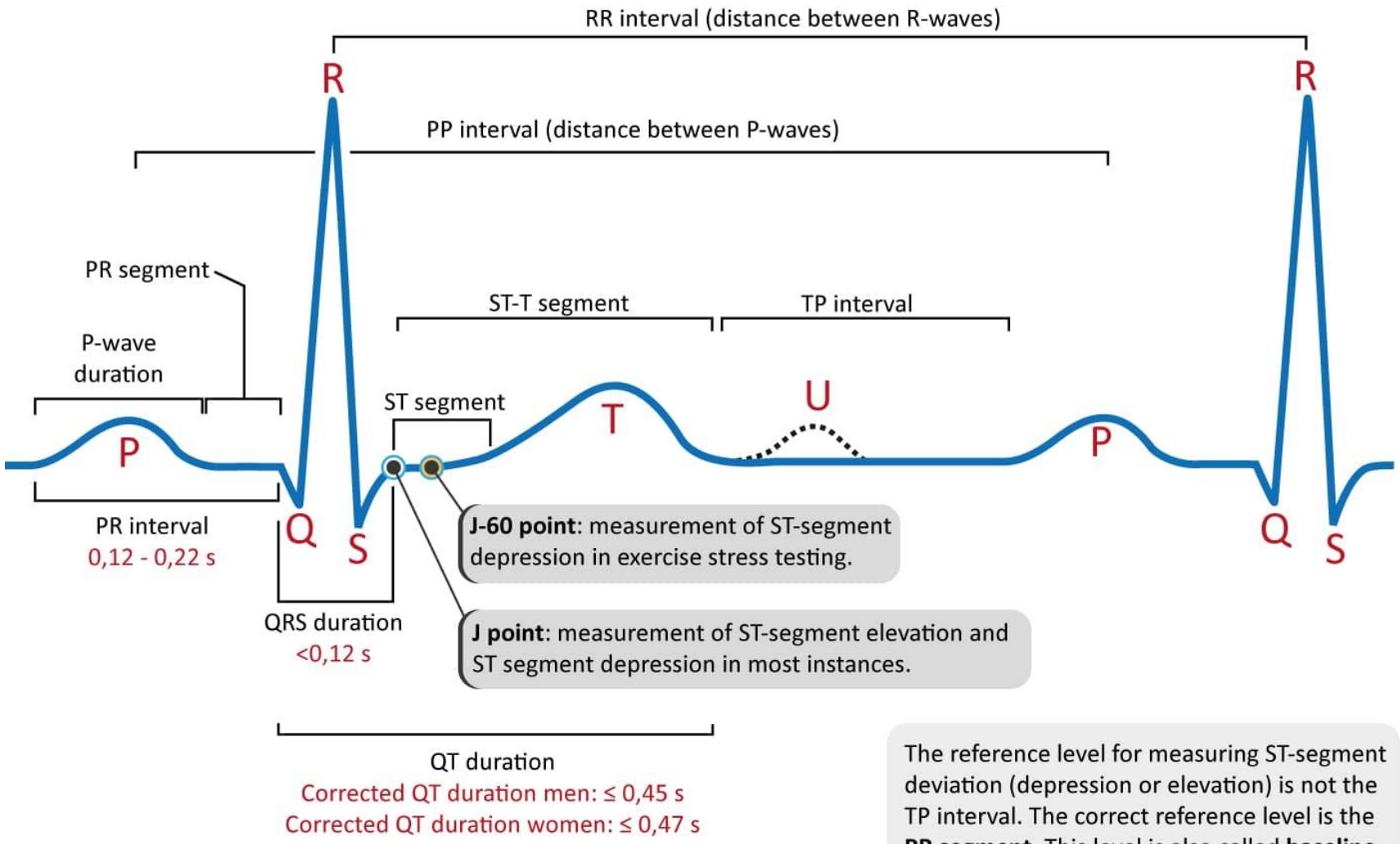
Rate	
Method I	In Lead II, count R waves x 10
Method II	

Rhythm
P before every QRS, QRS after every P

Axis		
I	**aVF**	**Axis**
+	+	Normal
+	-	Positive in II = Normal
+	-	Negative in II = LAD
-	+	RAD
-	-	Extreme Axis Deviation

Important/Common Patterns to Know*			
ACS	• New LBBB • Women: 1.5 mm ST elevation in V2, V3 • Men: 2 mm ST elevation in V2, V3	**Sgarbossa Criteria (if score ≥ 3, diagnoses MI in underlying LBBB)**	• if Concordant ST elevation > 1mm when QRS positive (5 points) • Concordant ST depression > 1mm in V1-V3 (3 points) • Discordant ST elevation > 5mm when QRS negative (2 points)
Hyperkalemia	peaked T waves	**LVH**	Sokolov-Lyon criteria: S wave in V1 + tallest R wave in V5 or V6 > 35mm
RBBB	1. QRS > 120ms 2. RSR' in V1-V3 3. Wide, slurred S wave in I, aVL, V5, V6 (lateral leads)	**LBBB**	1. S > 120ms 2. Large S wave in V1 3. Broad R wave and no Q waves in I, aVL, V5, V6 (lateral leads) 4. R wave peak > 60ms in V5-V6
Mobitz I	Lengthening of PR interval, then dropped P wave	**Mobitz II**	Constant PR interval, then dropped P wave
Pulmonary Embolism (order of most to least common patterns)	• Non-specific ST segment and T wave changes • Sinus tachycardia • T wave inversions in V1-V4 (precordial leads) and II, III, aVF (inferior leads) • Complete or incomplete RBBB • S1Q3T3		*see litfl.com/ecg-library for more EKG criteria

EKG Highlights



The reference level for measuring ST-segment deviation (depression or elevation) is not the TP interval. The correct reference level is the **PR segment**. This level is also called **baseline** level or **isoelectric level**.

Secondary and Resistant Hypertension

AHA 2017 Blood Pressure Criteria		
Category*	**Systolic**	**Diastolic**
Normal	< 120 mmHg	< 80 mmHg
Elevated	120 – 129 mmHg	< 80 mmHg
Stage 1 Hypertension	130 – 139 mmHg	80 – 89 mmHg
Stage 2 Hypertension	≥ 140 mmHg	≥ 90 mmHg
*The higher value determines the category		

Initial workup in new hypertension diagnosis	
Labs	Hgb, Hct, Cr, fasting serum glucose, Ca2+, lipid panel
Diagnostic tests	EKG, UA

Resistant Hypertension Definition	
1	Elevated BP with: • 3 anti-hypertensives at maximal doses • 3 different classes (RAAS blocker, long acting CCB, diuretic)
2	Controlled BP on ≥ 4 anti-hypertensives

When to consider secondary causes of hypertension

• Resistant hypertension diagnosis
  • Hypertension and ≤30yos
  • Accelerated hypertension
• New hypertension when previously stable

Secondary and Resistant Hypertension

Important Hypertension Trials		
	ALLHAT Trial (2002)	**ACCOMPLISH Trial (2008)**
Question	Chlorthalidone vs amlodipine vs lisinopril vs doxazosin for monotherapy HTN treatment	Benazepril/amlodipine vs benazepril/HCTZ for dual-therapy HTN treatment
Conclusions	• Chlorthalidone > amlodipine = lisinopril in improving HTN • Doxazosin arm terminated early for HF side effect	• Benazepril/amlodipine arm had fewer CVD events • Trial terminated early as Benazepril/HCTZ showed significantly worse outcomes

Non-pharmacological interventions	Pharmacological interventions*
• Low sodium (<2400 mg/day) diet • Reduced alcohol intake • ≥6 hours of uninterrupted sleep • Physical activity: ≥150 min/week of moderate exercise	1. ACEi/ARBs (ex. lisinopril, valsartan) 2. Dihydropyridine CCB (ex. amlodipine) 3. Diuretic (ex. HCTZ, chlorthalidone, torsemide) 4. MRA (ex. spironolactone or eplerenone) 5. β-blocker (ex. metoprolol) or combined α-β-blocker (ex. labetalol, carvedilol) 6. Non-dihydropyridine CCB (ex. diltiazem) 7. Hydrazinophthalazine (ex. hydralazine) 8. Substitute hydralazine for minoxidil *Underlying comorbidities should guide the addition of anti-HTNs

Hypertensive Urgency and Emergency

Hypertensive Urgency	
Definition	SBP ≥180 and/or DBP ≥120 *without* end-organ damage
Treatment	• If volume ↑ then diuresis • If euvolemic, then captopril or clonidine (onset time of around 90 min) • If med non-compliant, restart home medications
Goals	Reduce MAP by 25-30% over several hours to days to achieve goal BP of <160/90

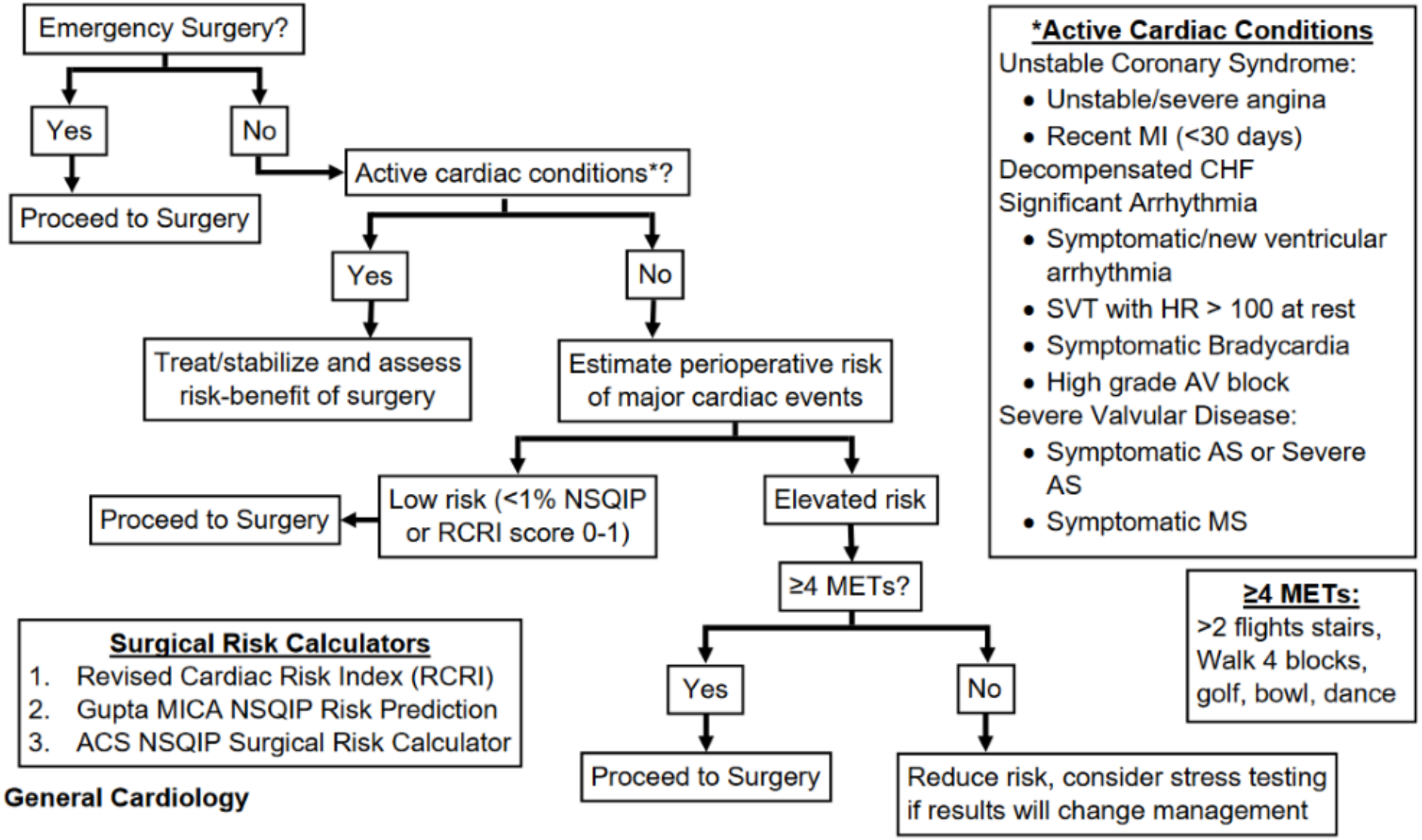
Hypertensive Emergency	
Definition	SBP ≥180 and/or DBP ≥120 *with* end-organ damage (e.g. MI or CVA, eclampsia, ↑ ICP, retinopathy)
Treatment	• If volume ↑ then diuresis • IV medications on table (e.g. Hydralazine, Labetalol)

Goals	• Within first hour, reduce MAP by 15%
	• Within first 24 hours, reduce MAP by 25-30% from initial BP; target goal of <160/90
	• Exceptions:
	  • acute ischemic stroke: allow for permissive HTN for cerebral auto-regulation
	  • aortic dissection rapid BP goal <120/80

Hypertensive Urgency and Emergency

IV drugs	Initial Dose	Max Dose	Onset	Duration	Pearls
Sodium nitroprusside	0.25-0.5 mcg/kg/min	8-10 mcg/kg/ min	≤1 min.	10 min	Requires ICU monitoring. Risk of cyanide toxicity in renal or hepatic failure. Not for use in CNS injury.
Nitroglycerin	5mcg/min	100mcg/min	2-5min	5-10min	Greater venodilation than arterial dilation; fewer anti-HTN effects in general; useful in patients with symptomatic CAD
Nicardipine	5 mg/hr ↑ by 2.5 mg/hr q5-15 min	15 mg/hr	45 mins (peak)	≤ 8 hours	Suitable for patients with ACS or CNS injury.
Labetalol	20 mg bolus + 20-80 mg q10 min.s	300 mg total	5 min	5-8 hours	Suitable for patients with renal failure, CNS injury, or tachycardia
Esmolol	0.5 mg/kg x 1 min + 50 mcg/kg/min x 4 mins (titrate in 50 mcg/kg/min min increments q4 min.s)	300 mcg/kg/min	2-10 min	10-30 min	Suitable for patients with ACS, renal failure, or CNS injury
Hydralazine	10-20 mg IVP q30min	400mg	10-30 min	2-4 hours	Not for use in CNS injury or renal failure. Indicated for eclampsia

Peri-operative Risk Assessment



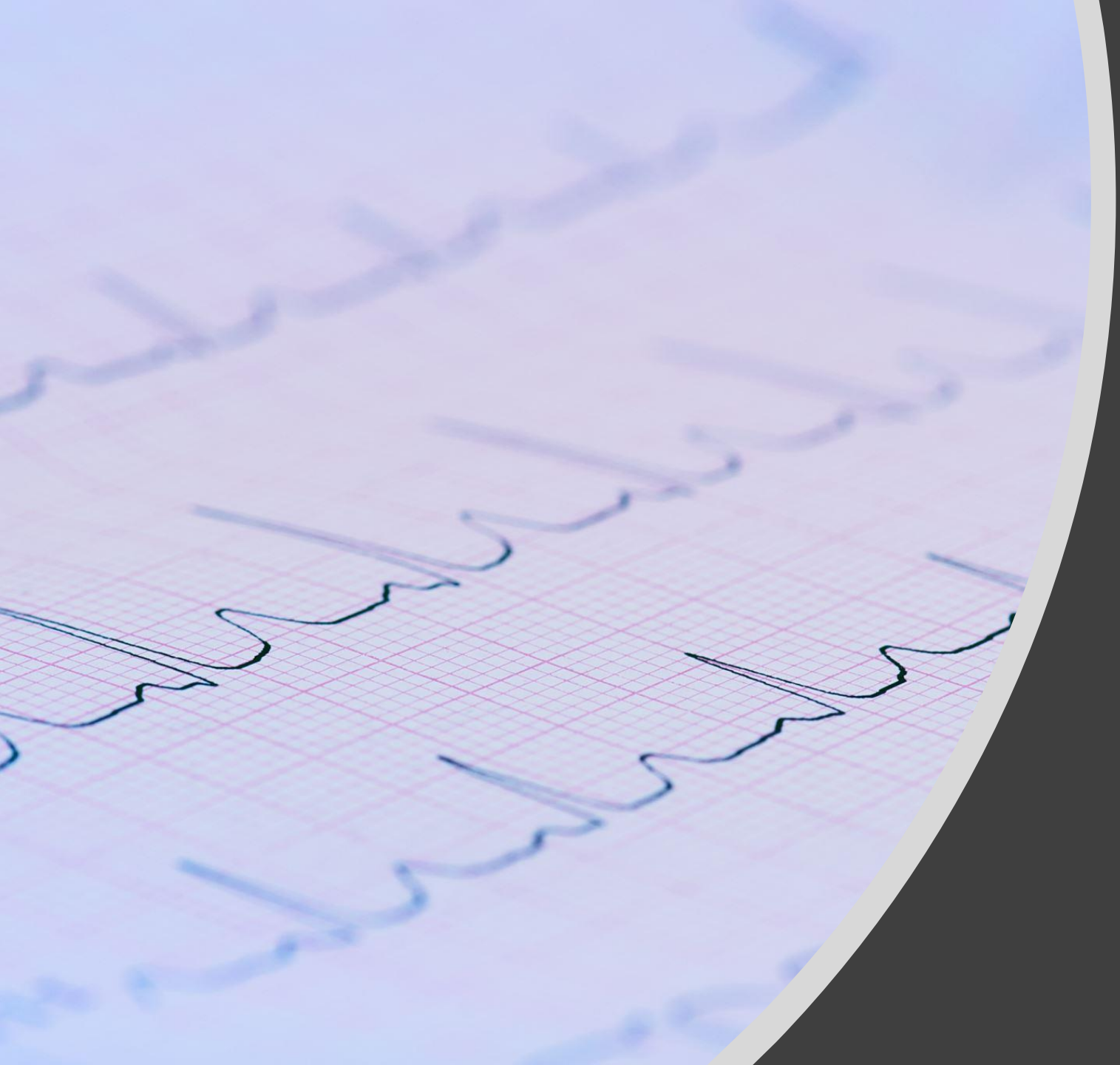
Cardio-obstetrics

Physiologic Cardiovascular Changes During Pregnancy	
Cardiac Output	Increases by 30-50% in first two trimesters
Heart rate	Increases by 20-25% by third trimester
Systematic Vascular Resistance	Decreases in 1st trimester, nadir in 2nd trimester, slight increase in 3rd trimester
Blood Pressure	MAP decreases by 6-8 mmHg in first two trimesters and slowly returns to normal 3-4 months postpartum
Relative anemia	Plasma volume increases by 10-15% in first trimester (relatively more than increase of RBC mass)

Pregnancy Associated Cardiovascular Diseases	
Pre-existing Pulmonary Hypertension	Pregnancy not recommended due to increased mortality related to physiologic changes
New-onset Hypertension	BP > 140/90 mmHg after 20 weeks; Managed with labetalol, nifedipine, or metoprolol
Peripartum Cardiomyopathy	Presents with CHF symptoms as early as 2nd trimester and as late as 6 mo post-partum; managed with diuretics and GDMT; recommended LV function recovery prior to repeat pregnancy
New-onset Ischemic Disease	Mostly commonly coronary artery dissection and coronary artery thrombosis; Risk 3-4x compared to non-pregnant counterparts
Myocardial Infarction	Recommended to undergo PCI and treatment with dual-antiplatelet therapy instead of thrombolysis

Cardio-oncology

Types of Cancer Treatment	Examples	Indications	Dose Dependent?	Cardiotoxic Effect	Reversible?
Anthracycline	Doxorubicin Daunorubicin	Lymphoma, Leukemia, MM, breast, Ovarian, stomach, Lung, thyroid, SCC H&N	Yes, cumulative dose exceeding 400mg/m2	Impaired LV function due to irreversible cardiomyocyte damage	Often permanent
Antimetabolite (Fluoropyrimidines)	Capecitabine 5-fluorouracil	Colon, pancreas, Stomach, breast, ovarian	Unknown	Arterial vasospasm, Myocardial ischemia, Thrombosis	Often yes
Anti-HER2	Trastuzumab Lapatinib	Breast, Gastric	Yes	Antagonism of HER2 pathway → Impaired LV function	Often yes
VEGF pathway inhibitors	Sunitinib Imatinib Dasatinib	Wide range of heme and solid malignancies	Likely	Varies by agent; coronary microvascular damage	Often yes
BTK inhibitor	Ibrutinib	Waldenstrom macroglubulinemia, CLL	Unknown	Unclear mechanism	Often yes
Immune Checkpoint inhibitors	Nivoluma Pembrolizumab Ipilumab	Melenoma, NSCLC, RCC, H&N, Hodgkin's lymphoma, bladder	Unknown	Myocarditis	Unknown
Ionizing radiation		Lymphoma	Yes	Inflammatory, CAD, direct cardiomyocyte damage	



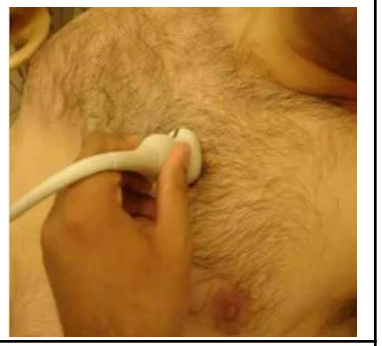
Cardiac Imaging

Section Editor:
Effimia Maria Zacharia, MD PhD

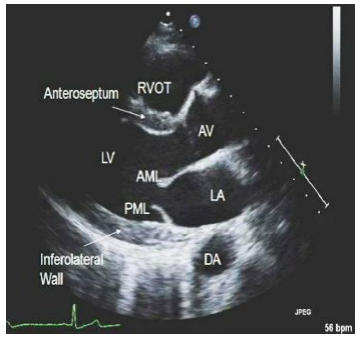
Cardiac POCUS: Views

Parasternal Long Axis (PLAX)

- 2-3 inches left of sternum between 4-5th ribs
- Notch faces right shoulder



- Heart is horizontal
- Apex is not visible
- Depth is correct if DA is visualized

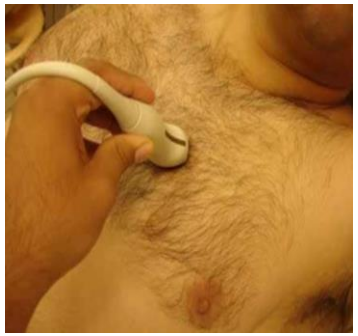


Things to Remember

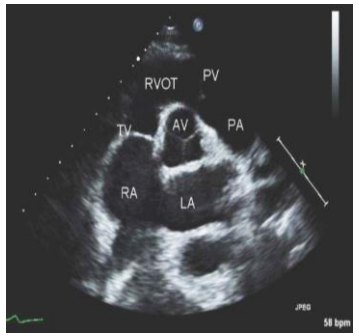
- Fluid above DA = pericardial effusion
- Fluid below DA = pleural effusion
- If RVOT ≠ AV ≠ LA, think dilation
- If RV >> LV, think dilation
- If LV looks like a "**D**", think increased RV afterload
- POCUS cannot classify vegetations

Parasternal Short Axis

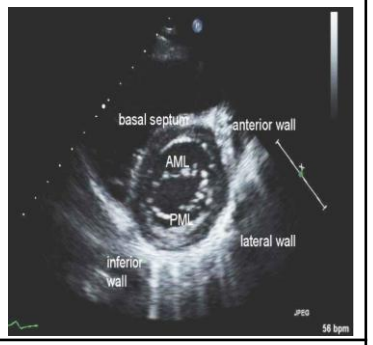
- From PLAX position, rotate prob clockwise so notch faces left shoulder
- Fan probe to see all views



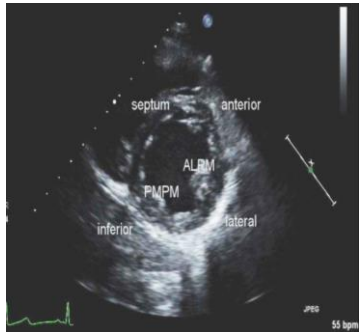
Aortic Valve Level



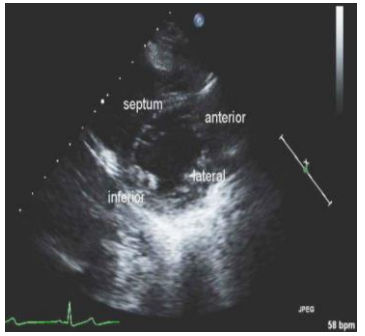
Mitral Valve Level



Papillary Muscle Level



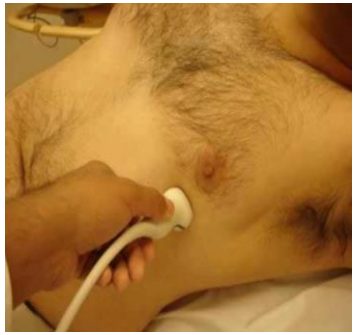
LV Apex Level



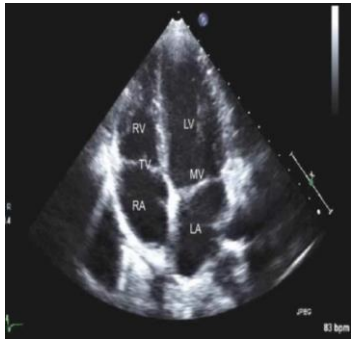
Cardiac POCUS: Views

Apical 4 or 5 Chamber View

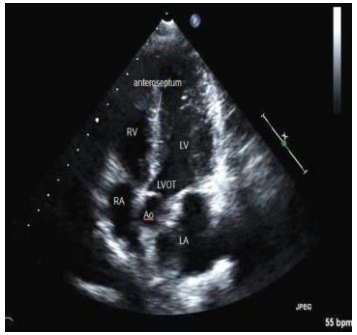
- From short axis position, bring probe downwards and place at PMI
- Notch faces left shoulder/axilla



Four Chamber View



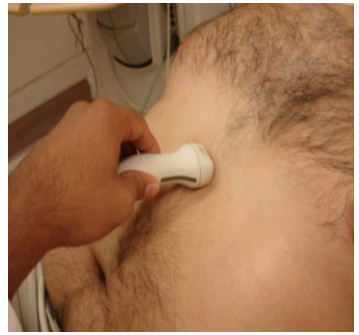
Five Chamber View



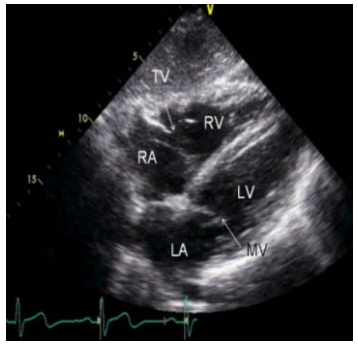
- IVS must be in the center
- DA should be visualized (correct depth)
- LV should appear parabolic
- If apex appears round, then image is off axis
- For 5 chamber, tilt probe upwards

Subcostal View and IVC

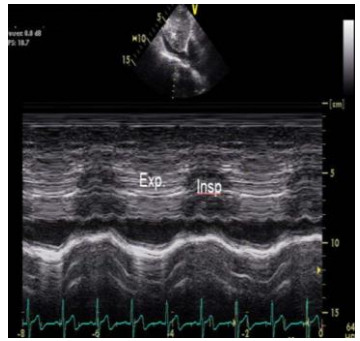
- Pt bends legs
- Place probe under subxiphoid process
- Notch faces axilla
- With RA in center, rotate anti-clockwise (notch towards head) to see IVC



Four Chamber View



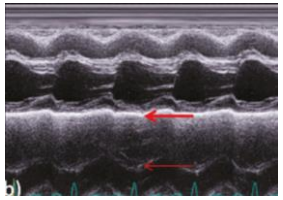
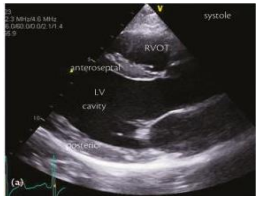
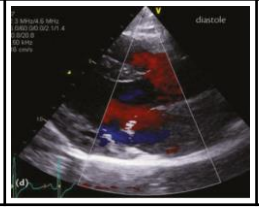
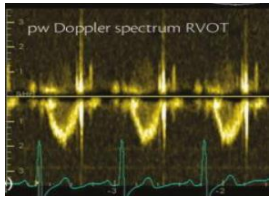
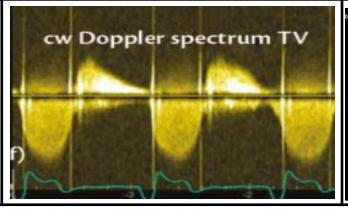
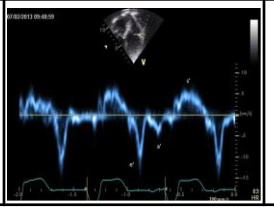
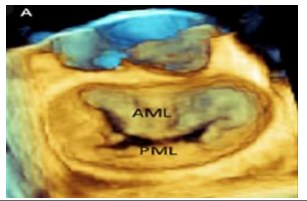
IVC with M mode



Things to Remember

- McConnel's sign: hyperdynamic RV apex, rest of RV akinetic; signifies increased RV afterload (i.e. PE)
- If IVC is collapsing <30% or IVC > 2 cm, think volume overload, RV dysfxn/PE, tamponade, large TV regurgitation

Echocardiography Terminology

ECHO Terminology							
Setting	M Mode	B Mode	Color Doppler	Pulse Spectral Doppler	Continuous Wave Doppler	Pulsed Spectral Tissue Doppler	3D Echo
Description	Reflections of single sound beam plotted against time	Two-dimensional; sectional planes	Adds information of blood flow	Measures flow velocities at a very specific location	Detects very high velocities without recording their location	Adds information of blood flow	Adds depth to 2D (B-mode); "surgical view"
Purpose	measure LV/ LA/ IVC dimensions, wall thickness; TAPSE	measure sizes and volumes, to estimate LVEF, RV function	visualize valve pathologies	assess for diastolic dysfunction, E/A ratio	estimate PASP; max and mean velocities and pressure gradients across valves; for valve pathologies	measure E/E' ratio, to assess diastolic dysfunction	Helpful in procedural planning i.e., valvuloplasty Suboptimal frame rates
Example							

Echo Modifiers		
Contrast Echo	**Bubble Study**	**Speckle tracking/strain analysis**
• Useful if ≥2 contiguous segments or coronary artery territory cannot be visualized • Looks for LV thrombus	• Bubbles appear if ASD present • Looks for patent foramen ovale (PFO)	• Looks for amyloidosis (order echo with strain analysis) • Assesses LV fxn for pts on cardiotoxic chemotherapy

Different Types of Echos

TTE (Transthoracic Echo)

Indication	Findings of interest	
Symptoms of Potential Cardiac Etiology	Chest Pain	WMA, Pericardial effusion
	Shortness of Breath	WMA, HF, pulmonary HTN
	Syncope	LVOT gradient, AS
Stroke/TIA/Embolism	Intracardiac thrombus, Shunts, Vegetations	
Endocarditis	Vegetations Duke Criteria; sensitivity 70% for native valves vs 96% for TEE	
CHF / Cardiomyopathy	LVEF, WMA, echogenicity of wall, strain pattern	
Frequent or Exercise-Induced PVCs, AFib, SVT, or VTach	WMA, low EF, arrhythmogenic RV	
Pulm HTN	R Ventricular function, TAPSE, PA pressure	
Hemodynamic Instability, Respiratory Failure	Cardiac output, complication of recent MI (e.g. acute MR, VSD, etc), volume responsiveness	
Post ACS	WMA/ LVEF	
Known acute PE	R heart strain to guide therapy or evaluate for efficacy of intervention	
Murmur	Evaluate for valvular stenosis or regurgitation	
Prosthetic Valve	surveillance >3yrs after implantation	

Stress echo

Indication	• Detection of WMA or transient ischemic dilation during stress (i.e. global myocardial ischemia or triple vessel disease) • Assess myocardial viability and contractile reserve (hibernating myocardium)
Types	Exercise (treadmill/bicycle) Pharmacologic (dobutamine ± atropine or vasodilators)
Notes	Hold beta blockers night before to maximize sensitivity

TTE (Transthoracic Echo)

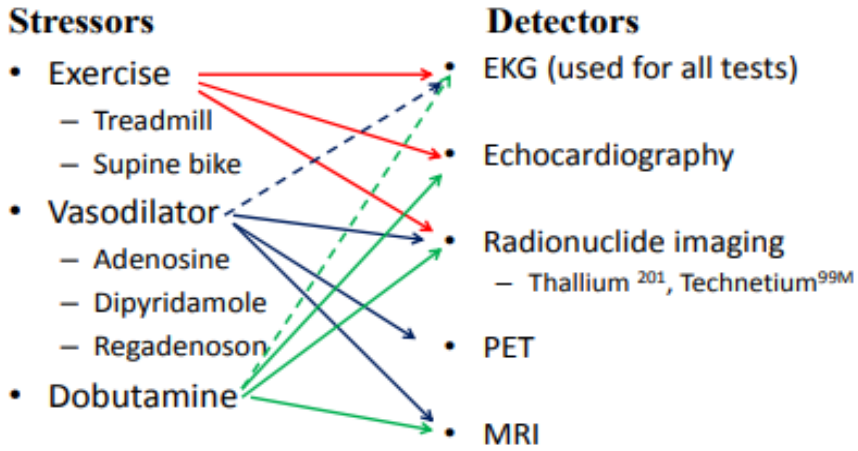
Indication	General Comments
When TTE is nondiagnostic	• Endocarditis (96% sensitive for native valve vegetations, paravalvular abscess or fistulas) • Prosthetic heart valves evaluation (i.e. thrombus or regurgitation) • Acute aortic pathologies (e.g. dissection, intramural hematoma) • Thrombus of left atrium/left atrial appendage (to determine safety of cardioversion and for anticoagulation decisions)
Cryptogenic embolism	In young <50 yo if TTE is normal
Intraoperative	Used in all cardiac (particularly valvular) and thoracic aortic surgeries, some CABGs
Transcatheter procedures	e.g. septal defect closures, atrial appendage obliteration, cardioversion
Critically ill	e.g. unexplained hypotension, unexplained hypoxemia
Relative contraindications	Coagulopathy (INR > 4), thrombocytopenia (<50k), esophageal varices, active esophagitis/PUD, history of radiation, recent GIB, Barrett's esophagus, hiatal hernia, poor neck motility, dysphagia
Absolute contraindications	Perforated viscus, esophageal tumor/stricture/perforation/laceration, active upper GIB
NPO status	At least NPO for 8 hours, when ordering place NPO at midnight status
Notes	• Typically done in echo lab for stable patients, but can be done at bedside if requested (especially for ICU patients) • For stat echos or weekend requests, call the echo lab and/or page the cardiology fellow

How to Choose a Stress Test

Test	Pros	Cons
Exercise EKG	• Provides prognostic info • No radiation • Cost-effective	• Less sensitive/specific • Cannot localize ischemia
Exercise or dobutamine echo	• Localizes ischemia • Assesses heart function, valves, PASP • No radiation • Provides prognostic info	• Baseline WMA makes hard to interpret • Poor window in some patients
Reg-SPECT	• Localizes ischemia • Good if arrhythmia, HTN, baseline WMA • Not affected by b-blockers	• Radiation • Must lay flat/still • Cannot detect global ischemia (only regional) • Attenuation artifacts
Reg-PET	• High resolution, fewer artifacts • Good for obese pts • Defects both regional and global ischemia • Assesses viability with FDG • Quick	• Radiation • Short half life of tracer limits stressor modalities
Reg-MRI	• Assesses anatomy, function, infarct size, location, viability	• Needs expertise • Long time • Must lay flat, hold breath • Regular HR necessary

Why stress?
• Diagnose or risk stratify known or suspected CAD • Localize ischemia prior to revascularization • Determine if medical therapy is adequate • Evaluate the severity of valvular disease or coronary heart disease

To Image or Not To Image?	
Exercise EKG	• Patient can exercise • Baseline EKG is interpretable for ischemia
Image (we usually image)	• Baseline EKG is abnormal • Known CAD or prior revascularization • Need to assess: LF fxn, valvular disease, PASP, viability

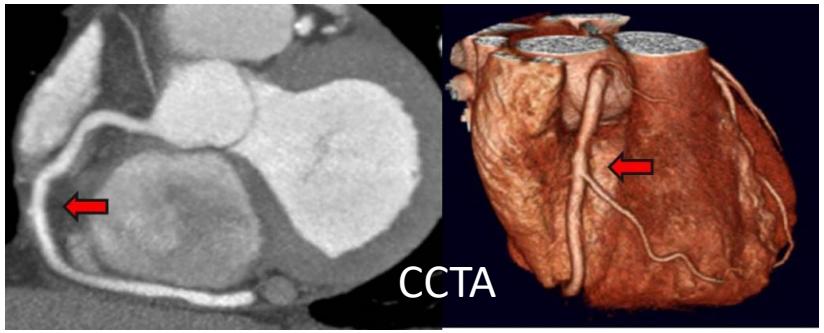


Cardiac CT

Coronary CT angiography (CCTA)	
Diagnostic purpose	• Use for intermediate pre-test probability (defined by Diamond-Forrester score) • Has diagnostic accuracy for detection of obstructive CAD (>50% luminal narrowing in major epicardial vessels)
What info does it provide?	• LVEF • Myocardial perfusion • Fractional Flow Reserve • MACE prediction

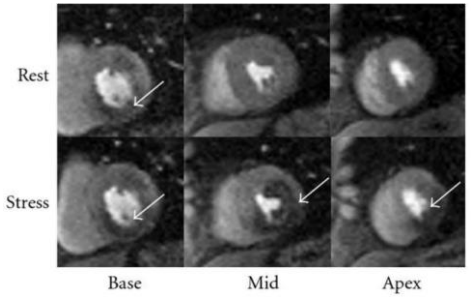
Dual-energy computer tomography (DECT)	
Diagnostic purpose	• Maps iodine distribution in myocardium as surrogate for perfusion
What info does it provide?	• Characterizes plaques • Identifies high-risk plaques prone to rupture

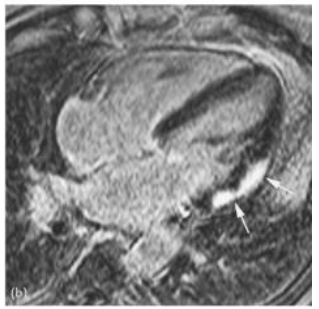
Contraindications and Considerations
• Renal insufficiency • Iodine allergy • Cardiac tachyarrhythmias • Radiation exposure • Patient must cooperate and hold breath for 5-10 sec

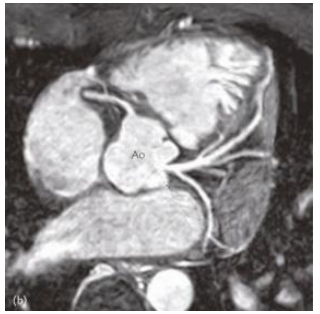


Cardiac MRI (cMR)

cMR Overview	
What info does it provide?	• Global functional parameters (EDV, ESV, LVEF, myocardial mass) • Regional function • Valvular function
Non-contrast enhanced	• Can assess smallest anatomical details • T1: pericardium, aortic wall, fatty infiltration of myocardium • T2: myocardial edema • Infiltrative diseases i.e. iron in hemochromatosis, rejection in transplanted hearts, extent of fibrosis/ inflammation/ necrosis and deposition of amyloid
Contrast enhanced	• Late gadolinium enhancement seen in MI and non-ischemic diseases, i.e. myocarditis, amyloidosis, hypertrophic/dilated cardiomyopathy, arrhythmogenic RV





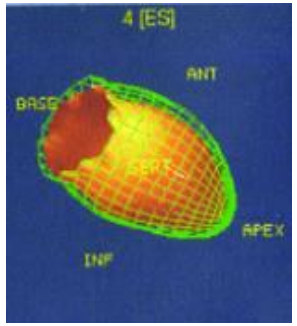


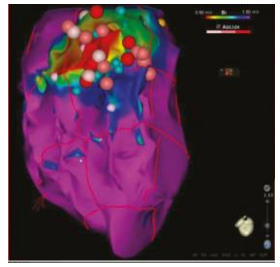
Contraindications and Considerations

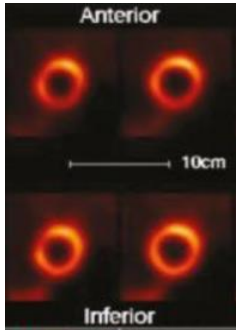
- Metallic and electrical implants, devices or foreign bodies
- Severe renal insufficiency
- Arrhythmias/irregular breathing can decrease image quality
- Long test (40-60 min)

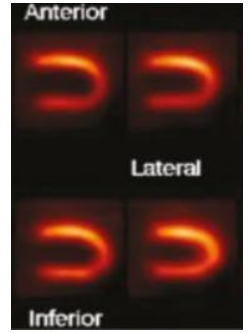
SPECT and PET

SPECT Overview	
What info does it provide?	• Systolic and diastolic parameters • Can monitor slight LVEF changes (indicator of cardiotoxicity 2/2 other medications)
Stress test	• Marker: Tc-sestamibi • Compares images at rest and stress • Perfusion defect appears as dark spot
Molecular imaging	<u>MIBG labelling</u> • Assess for impaired cardiac adrenergic innervation (predicts MACE) • Identify ablation targets <u>99mTc0labeled pyrophosphate (PYP)</u> • Diagnose cardiac ATTR amyloidosis

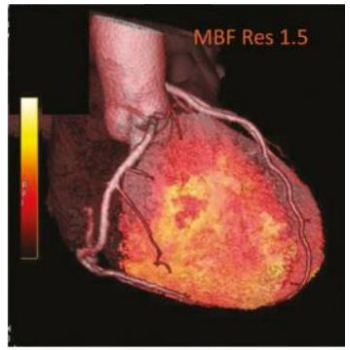


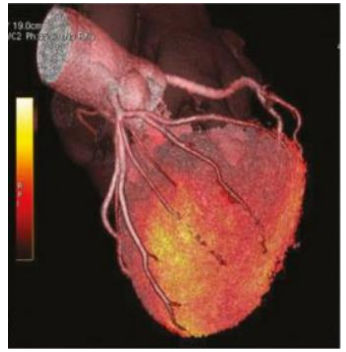


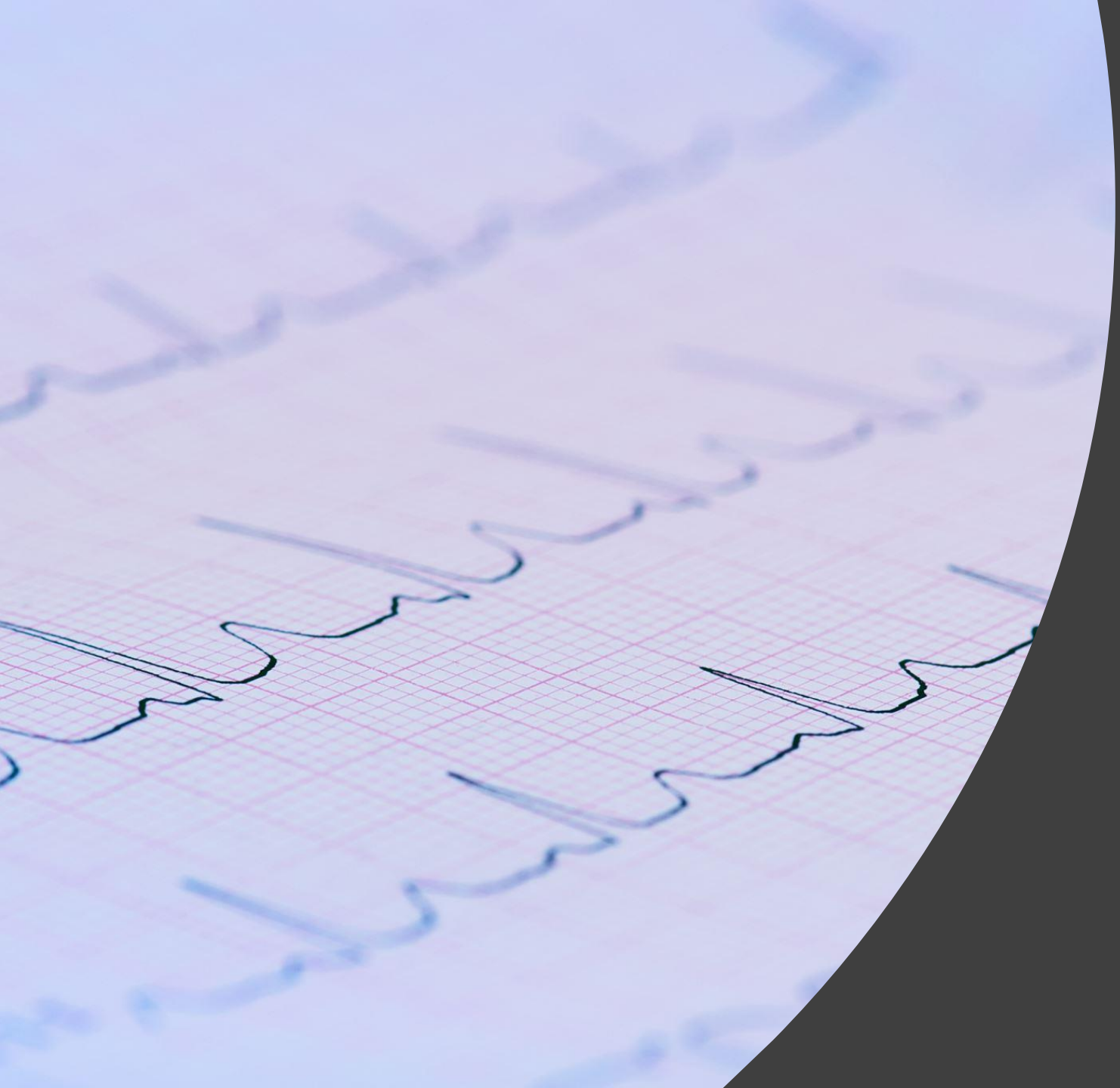




PET Overview	
What info does it provide?	• Regional and global myocardial perfusion • Assessment of global ischemia (diffuse atherosclerosis, triple vessel disease, microvascular defects) • Assessment of decrease in myocardial blood flow in cardiomyopathies
Fluorodeoxyglucose tracer	• Uses glucose metabolism to assess ischemia/viability • Increased uptake: hypoxia/mild ischemia • Decreased update: severe ischemia • No uptake: scarring
11C-HED tracer	• Assess impaired cardiac adrenergic innervation after MI or in CHF • Predicts MACE
PIB (Pittsburgh compound B) tracer	• Diagnoses cardiac amyloidosis





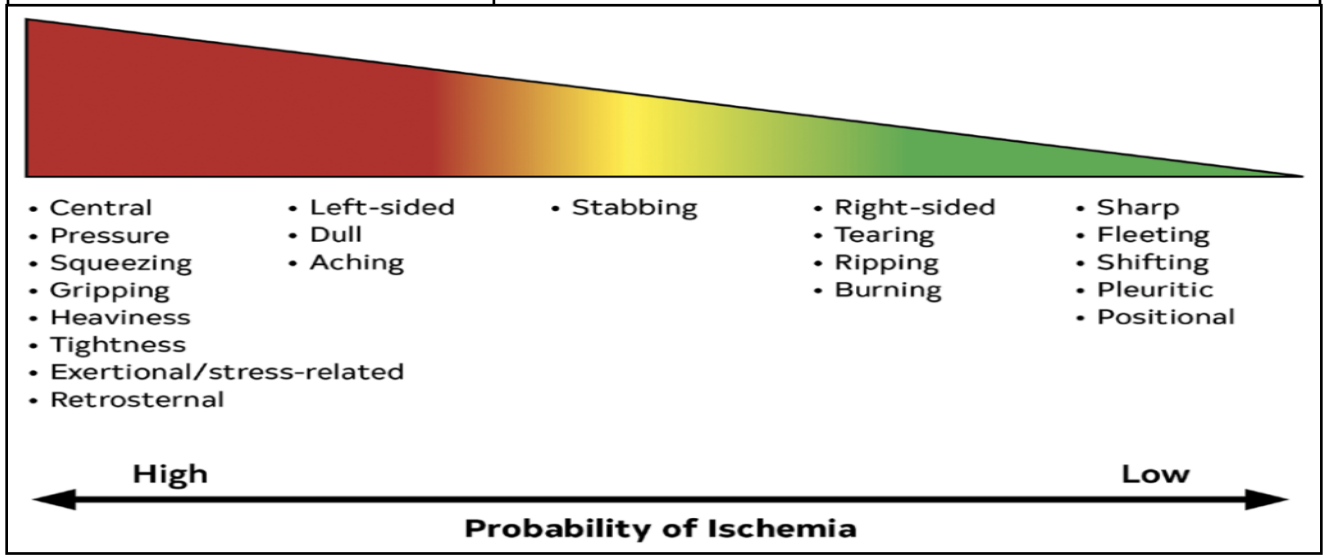


Coronary Artery Disease

Section Editor:
Valentina Jaramillo Restrepo, MD

ACS & Chest Pain

Clinical Evaluation of Chest Pain	
Chest Pain Description	Cardiac Possibly Cardiac Non-Cardiac
Initial Triage based off of HEART Score	Low Risk: 0-3 (2.5% MI/PCI/CABG/death) Moderate Risk: 4-6 (20.3%) High Risk: ≥ 7 (72.7%)
Concerns for cardiac chest pain ACS/Non-ACS	Admit to PAV
STEMI	STAT page Cardiology to activate Cath lab



HEART Score (found on MD Calc)		
History	Highly suspicious	2
	Moderately suspicious	1
	Slightly suspicious	0
EKG	Significant ST-deviation	2
	Non-specific repolarization disturbance	1
	Normal	0
Age	≥ 65 years	2
	45– 65 years	1
	≤ 45 years	0
Risk Factors	≥3 risk factors or hx of CAD	2
	1 or 2 risk factors	1
	No risk factors	0
Troponin	≥3x normal limit	2
	1-3x normal limit	1
	≤ normal limit	0

Risk Factors for CAD:

Hypertension	Diabetes Mellitus	Family History
Hypercholesterolemia	Smoking	Obesity (BMI > 30)

ACS & Chest Pain

	Unstable Angina	NSTEMI	STEMI
Definition	EKG ⊖ or ⊕ Biomarkers ⊖	EKG ⊖ or ⊕ Biomarkers ⊕	EKG ⊕ Biomarkers ⊕
EKG Findings	• No change • ≥ 0.5mm ST depression (horizontal/downward sloping more concerning • new TWI >1mm	• No change • ≥ 0.5mm ST depression (horizontal/downward sloping more concerning) • new TWI >1mm	• 1mm new ST-segment elevation in two contiguous leads (all leads except V2, V3) • V2-V3: ≥2 mm in men ≥40 years; ≥2.5 mm in men <40 years; ≥1.5 mm in women • new LBBB
Biomarkers	hsTrop <4 with pain OR △ <4 in 1 hour	hsTrop peaks above 18 (ULN)	hsTrop peaks above 18 (ULN)
Treatment	Load with ASA+P2Y12i, heparin gtt BB and statin during admission	Load with ASA+P2Y12i, heparin gtt BB and statin during admission	Load ASA and P2Y12i
Cardiac Cath?	Risk Stratification	Risk Stratification	STAT

Early risk stratification for UA/NSTEMI	
Hemodynamic/Electrical instability Refractory Chest Pain	• Immediate cardiac cath
TIMI for UA/NSTEMI (on MDCalc)	• Predicts 14 day all cause mortality, new/recurrent ischemia • TIMI ≥3: high risk group that benefit from early cardiac cath (within 24 hours of chest pain) • Trials: TACTICS-TIMI, TIMACS
GRACE (on MDCalc)	• Predicts 6-month morality from admission

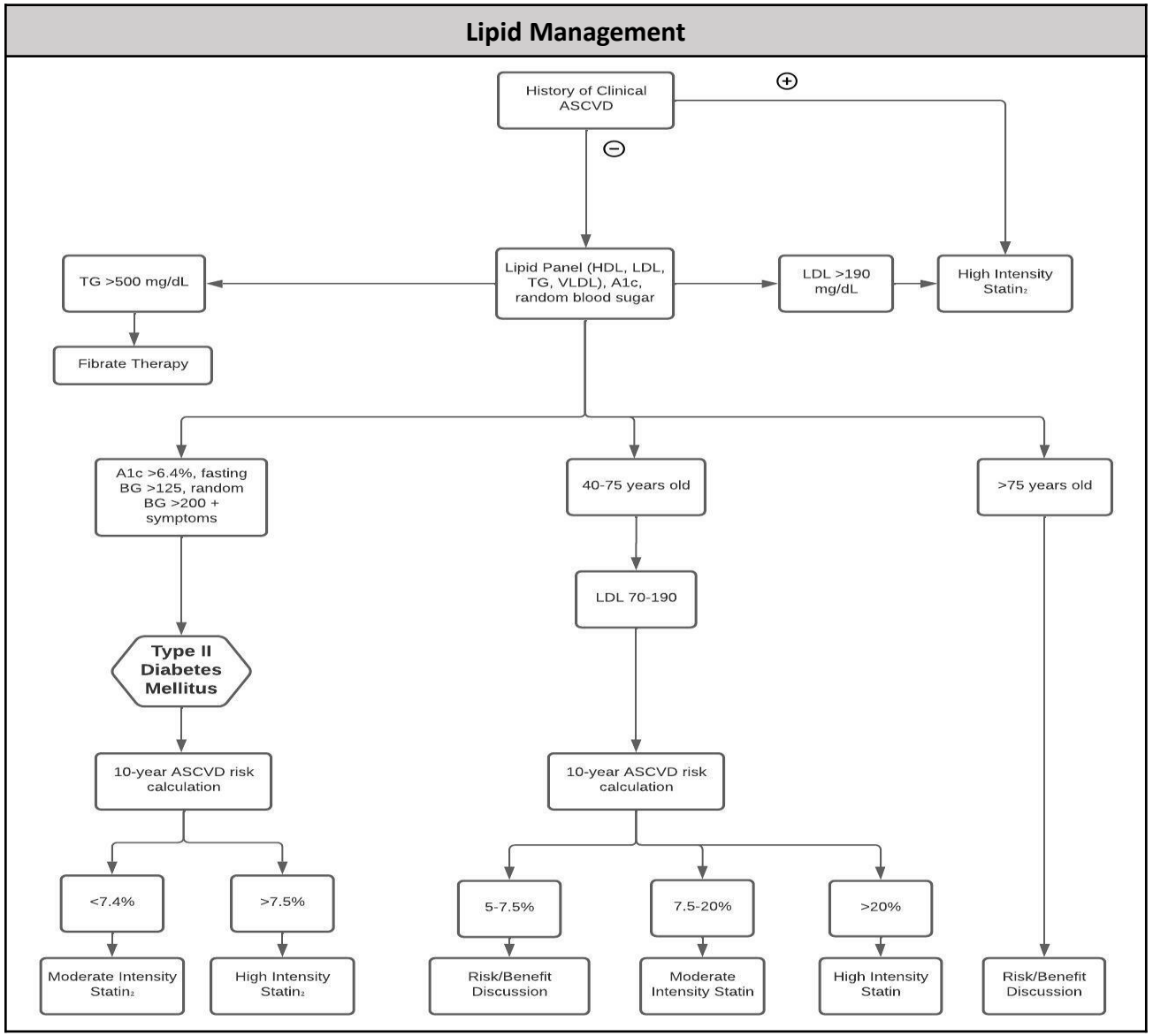
ACS & Chest Pain

Hs and Ts (Causes of Myocardial Ischemia)	
Hypovolemia	**T**oxins/**T**ablets
Hypoxia	**T**amponade
Hydrogen Ions (acidosis)	**T**ension Pneumothorax
Hyperkalemia/**H**ypokalemia	**T**hrombosis
Hypothermia	**T**hromboembolism (pulmonary)
Hypoglycemia	**T**rauma

Biomarkers	
High sensitivity troponin	Reference range: < 18ng/L Critical value: > 90ng/L OR delta >15 within 12 hours
PPV	Directly correlated with high sensitivity troponin value
NPV	High if chest pain >6 hours
Other reasons to be high (besides ACS)	Myocardial injury heart failure, rapid atrial fibrillation, myocarditis, anthracycline cardiotoxicity, subendocardial wall stress, myopericarditis, sepsis

Acute Treatment for ACS			
Anti-platelet (cont. DAPT 12 mo)	ASA	Load 324mg	Daily 81mg
	Clopidogrel	Load 300-600mg	Daily 75mg
	Prasugrel	Load 60mg	Daily 10mg
	Ticagrelor	Load 180mg	Twice Daily 90mg
GP IIb/IIIa	• Early invasive strategy for pts with high thrombus burden • Defer to cath lab for length of time for therapy		
Anti-coagulation	• If on DOAC, heparin gtt on DOAC Interference UA/NSTEMI protocol with no bolus • If GP IIb/IIIa, load UFH 60-unit/kg bolus (maximum 4000 units) followed by a 12 unit/kg/hr infusion		
Oxygen	• Goal: SpO2 > 92%		
Nitrates	• Sublingual NTG q5 min x3 if pain • IV NTG if persistent ischemia or HTN (avoid if on PDEi)		
Morphine	• Try to avoid unless uncontrolled, persistent pain		
Beta Block	• Begin metoprolol or carvedilol if no signs of heart failure or shock • Reduces mortality per COMMIT/CCS-2 trial		
Statins	• Begin high intensity statin (ex. atorvastatin 80mg) • Reduces mortality per PROVE-IT TIMI trial		
ACE inhibitors	• Begin Lisinopril, enalapril, or benazepril if LV dysfxn (LVEF < 40%)		

ASCVD Risk



Risk Factors	
Family hx premature CVD (men <55y, women <65y)	Inflammatory disease (RA, lupus, psoriasis, HIV)
LDL > 160, total chol > 190	Premature menopause (<40y)
CKD	South Asian ancestry
Metabolic syndrome	Biomarkers: TG > 175, Lp(a) > 50, CRP > 2, Apolipoprotein B > 130, ABI < 0.9
Preeclampsia/Eclampsia/ HELLP syndrome	

Coronary Calcium Score	
CAC 0	No statin necessary Repeat CAC in 5-10 years
CAC 1-100	Moderate intensity statin
CAC >100 or 75th %-tile	High intensity statin
Order coronary artery calcium testing for: • ASCVD 7.5-19.9% • High risk patients with ASCVD 5.0-7.4%	

Percutaneous Coronary Intervention (PCI)

Types of PCI	
Diagnostic coronary artery angiography performed prior to assess anatomy and disease burden to plan for appropriate intervention	
Significant stenosis: ≥70% for non-left main disease, ≥50 % for left main disease	
Balloon angioplasty	Inflation of balloon
Stent implantation	Drug eluting vs bare metal
Atherectomy	Physically removes plaque
Intravascular US	Estimates stenosis and appropriate stent deployment
Shockwave lithotripsy	Circumferential, pulsatile mechanical energy to disrupt calcium
Fractional flow reserve (FFR) Instantaneous wave-free ration (iFR)	Indices of severity Normal: FFR > 0.80, iFR > 0.89

Absolute Contraindications
Non-compliance with procedure
Inability to take DAPT
High bleeding risk (thrombocytopenia, peptic ulcer disease, severe coagulopathy)
Multiple PCI restenosis

PCI vs CABG	
STEMI	• PCI: within 12 h, if cardiogenic shock; after >12 h if ongoing ischemia/HF/electrically unstable • CABG: if PCI not feasible • No benefit for revascularization if chronically totally occluded
Complex disease (multivessel disease, complex lesions)	CABG > PCI
Diabetics	PCI: if poor surgical candidate CABG: ≥2 vessels and/or LAD involvement LAD: discussion
Stable ischemic heart disease	If refractory angina, consider revascularization based off of coronary anatomy

Percutaneous Coronary Intervention (PCI)

Prior to PCI	
Labs	CBC, BMP, PT/INR
Premedication	• Load with ASA 324mg + P2Y12inh + GP IIb/IIIa (if high risk) • Enoxaparin vs Heparin gtt
Diet	• NPO not required • If non-urgent, NPO at midnight + no caffeine
Medications	• Hypoglycemic agents: hold metformin 48hrs before and after, insulin correction to accommodate NPO status • BB: continue • ACEi/ARBs and diuretics: hold AM of procedure, resume afterwards
After PCI	
Hydration	Goal UOP: 150cc/hr (decrease kidney injury)
Discharge	Same day discharge after uncomplicated diagnostic coronary angiography without high-risk findings/uncomplicated planned PCI

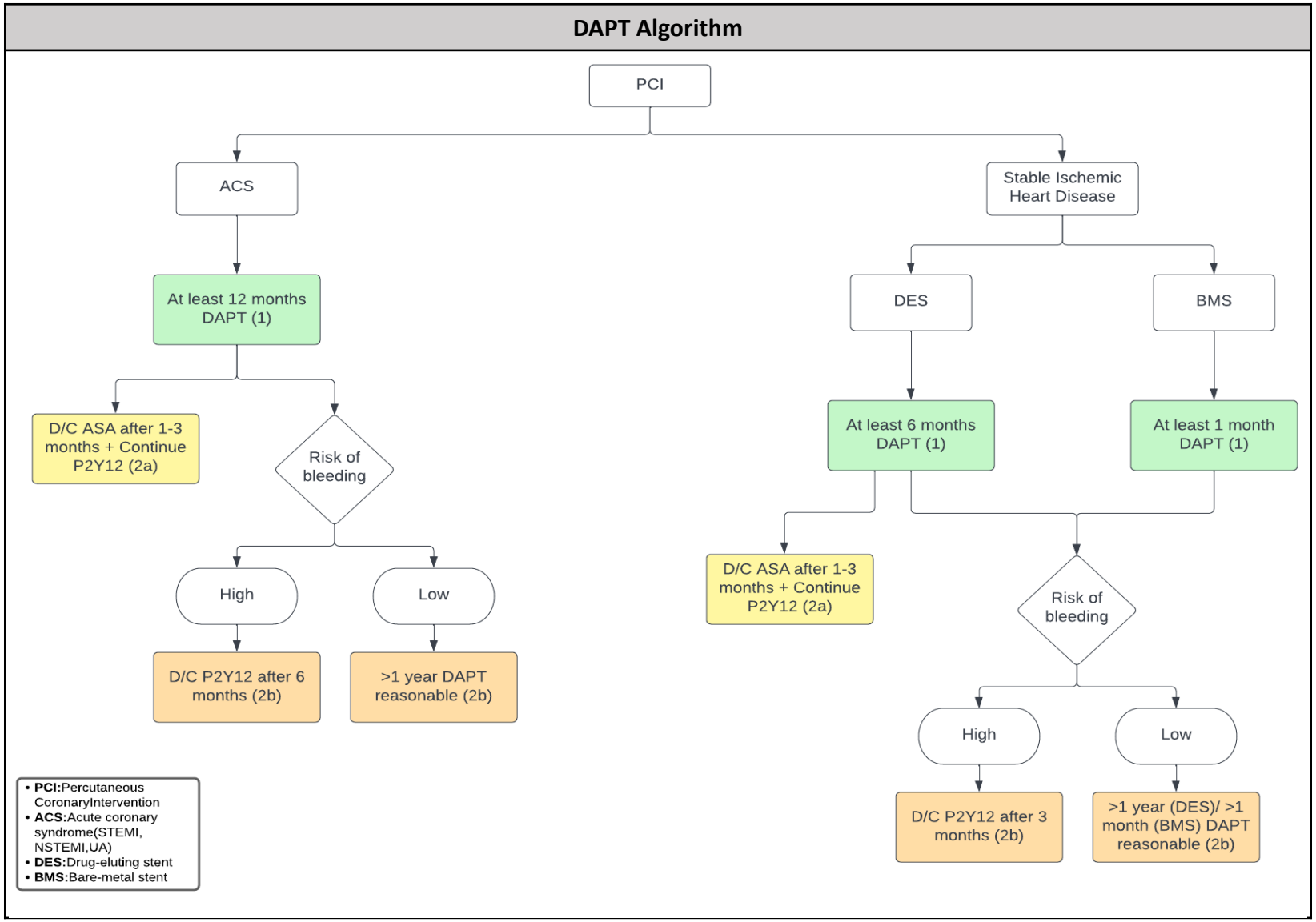
Complications

- Coronary artery or aortic injury (dissection, rupture)
- Bleeding (site or due to DAPT)
- Site complications: Pseudoaneurysm, RP bleed, infection
- Renal injury or failure
- Distal embolization (stroke, MI)
- Increased risk: > 65y, females, renal dysfunction, diabetes

Access Considerations

Femoral Artery	**Radial Artery**
• Manual compression and vascular closure devices; if rebleeding hold pressure for 10-15 min + for persistent bleeding, page cath fellow • Ambulation recommended 1-8 hr post-procedure and determined by sheath size and success of vascular closure device	• Radial hemostatic placed for 30 min for diagnostic cath and 90 min for PCI • 3 mL of air is deflated q15 min until band is deflated • If no hemostasis, air is reinjected and clock restarts

Dual Anti-Platelet Therapy (DAPT)



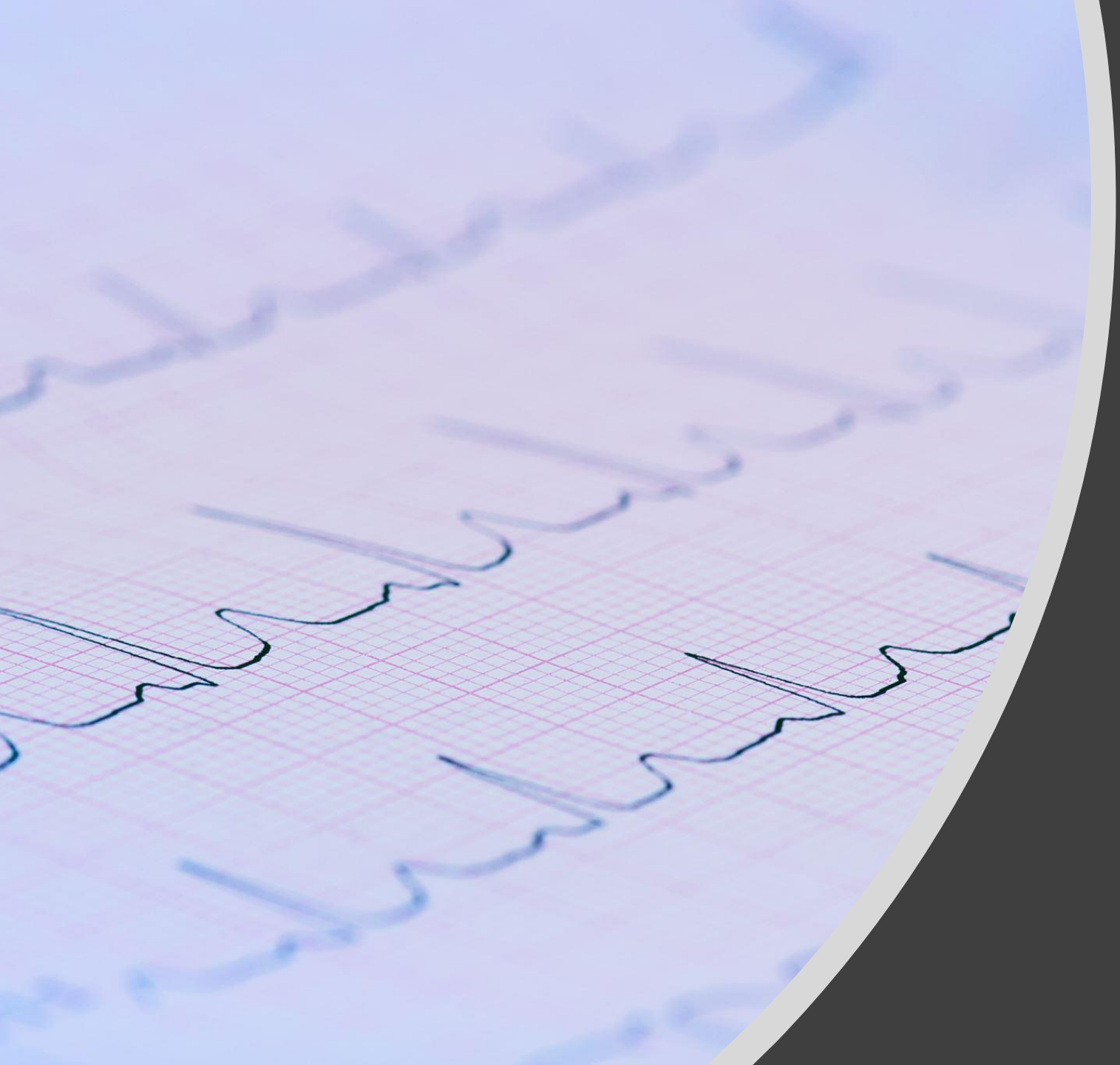
Dual Anti-Platelet Therapy (DAPT)

Drug eluting stent (DES)	Bare metal stent (BMS)
Decreased risk of restenosis, MI, and acute stent thrombosis compared to BMS	Preferred if unable to tolerate DAPT long-term (minimum 1 mo required)

DAPT and PPI
• Recommended if prior GI bleeding, advanced age, NSAID/steroid/warfarin use • Not recommended if low risk of GI bleeding

Triple Therapy (DAPT and anticoagulation)	
Anticoagulation for atrial fibrillation	Discontinue ASA after 1-4 weeks Continue P2Y12inh + DOAC/warfarin
Anticoagulation for coagulability disorder	Consult hematology
Anticoagulation for prosthetic heart valves	Discuss with cardiology

DAPT and CVA	
Small vessel disease Extracranial large artery atherosclerosis Intracranial stenosis 50-69%	NIHSS <6: DAPT for 21 days then monotherapy NIHSS >5: single agent
Large intracranial artery stenosis 70-99%	DAPT for 90 days then single agent
Large infarct and comorbid condition requiring AC	ASA for 2 weeks then restart oral AC If bleeding: hold all therapy until stable



Electrophysiology

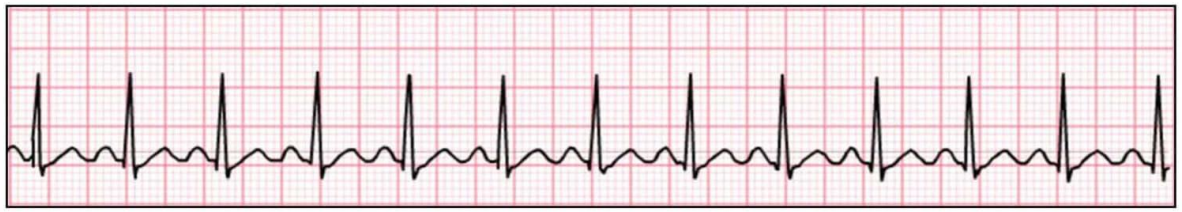
Section Editors:
Ronaldo Correa, MD
Balvindar Singh, MD PhD

Supraventricular Tachycardias (SVT)

Definition: narrow complex tachycardia, QRS < 120 ms, HR > 100

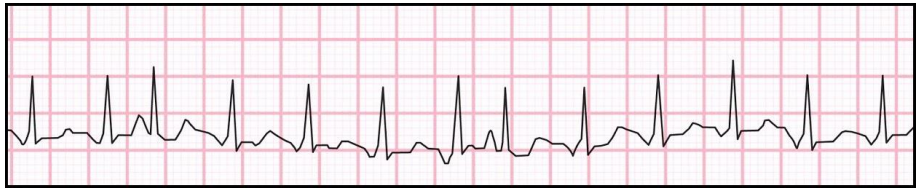
Sinus tachycardia

- Regular rhythm, normal P waves
- Physiological response to underlying condition
- *Gradual* in onset (review tele for gradual increase in HR vs sudden shift – more suggestive of other SVT)
- Causes: Hypovolemia, hemorrhage, withdrawal (EtOH, BZD, opiate, BB), intoxication, fever/infection, pain, hypoxemia, anemia, PE, hyperthyroid, adrenal insufficiency, pheochromocytoma



Multifocal atrial tachycardia (MAT)

- AKA wandering atrial pacemaker if not tachycardic
- Irregular rhythm
- Multiple, abnormal P wave morphologies
- Non-SA node origin, > 3 foci (> 3 P wave morphologies)
- Irregular rhythm
- **Causes:** COPD, pulmonary HTN, CAD, electrolyte derangements

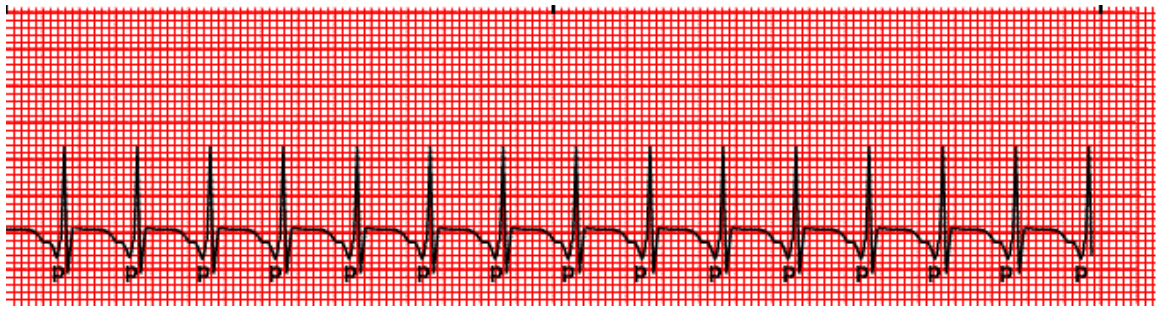


Supraventricular Tachycardias (SVT)

Definition: narrow complex tachycardia, QRS < 120 ms, HR > 100

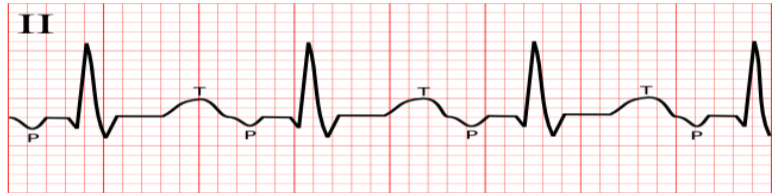
Junctional tachycardia

- Regular rhythm
- Increased automaticity in AV junction
- P waves present? Must be down/negative in aVF



Atrial tachycardia

- Regular rhythm
- Single but abnormal P wave morphology
- Non-SA node origin
- Often 2/2 digoxin toxicity

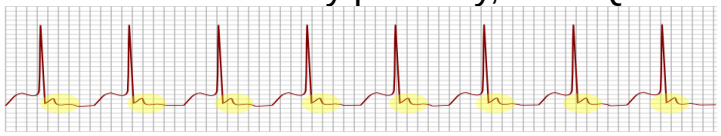


Supraventricular Tachycardias (SVT)

Definition: narrow complex tachycardia, QRS < 120 ms, HR > 100

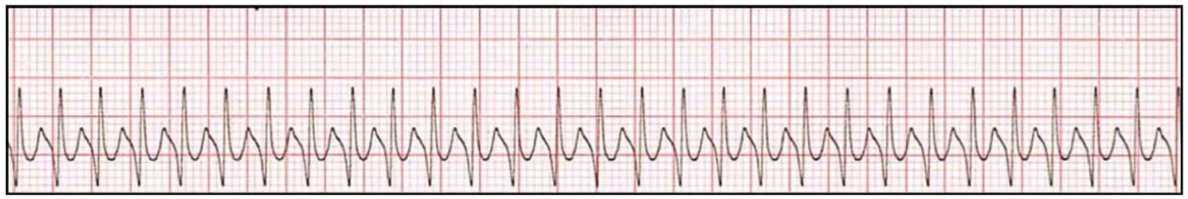
Atrioventricular nodal re-entrant tachycardia (AVNRT)

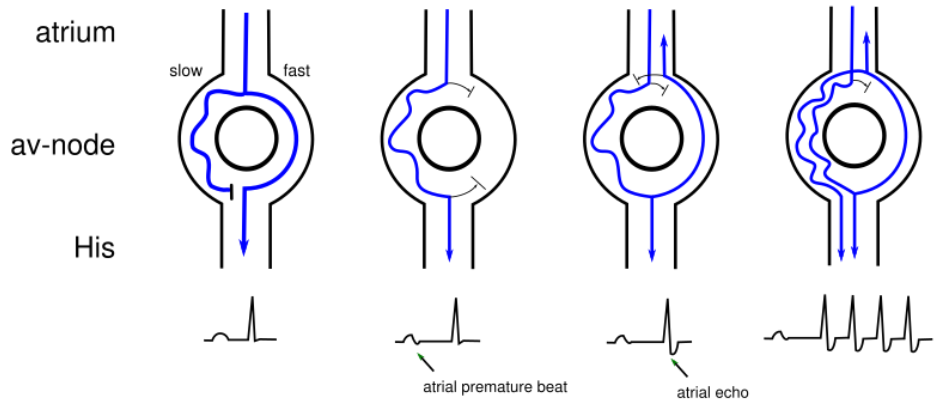
- Regular rhythm
- Arises from re-entry in AV node
- Short RP
- Ventricular activation:
  - Orthodromic: AV node, narrow QRS
  - Antidromic: accessory pathway, wide QRS

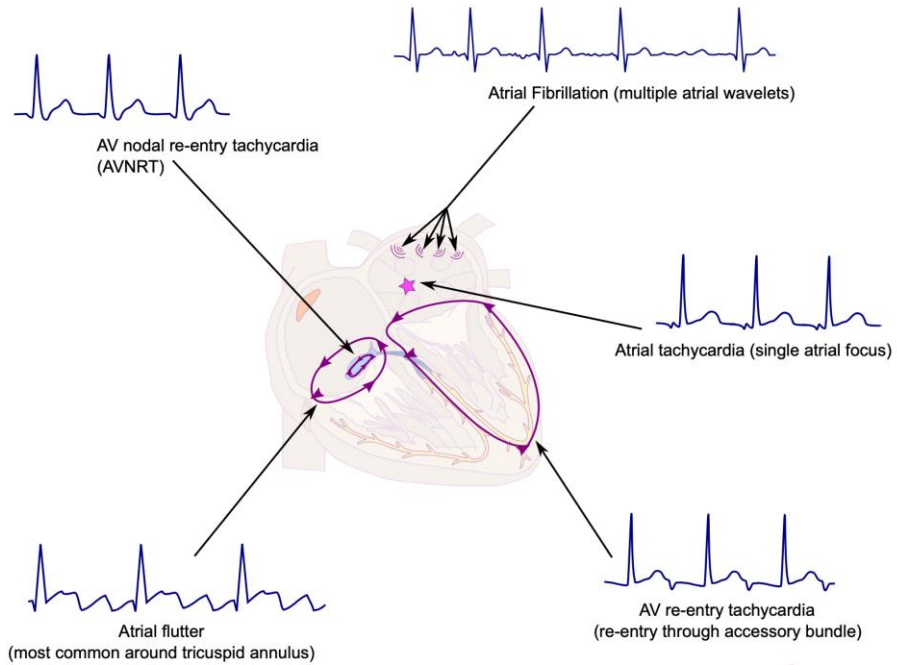


Atrioventricular re-entrant tachycardia (AVRT)

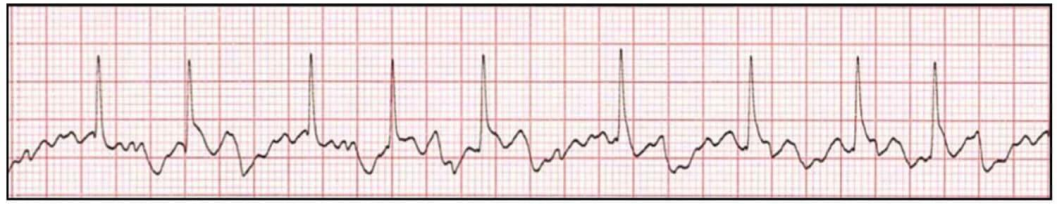
- Regular rhythm
- Arises from re-entry in AV node
- Short or no RP
- Triggers: PAC, PVC
- Pt: young, F > M





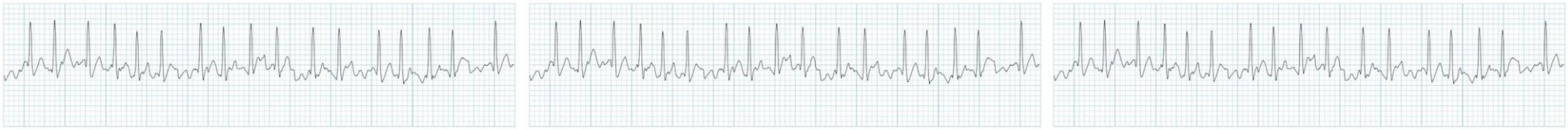


Atrial Fibrillation (Afib)

Atrial Fibrillation Overview	
Definition	• Irregularly irregular rhythm • No P waves • Variable ventricular rate • Symptoms: palpitations, chest pain, dyspnea, fatigue, light-headed 
Epidemiology	• RF: increased age, obesity, HTN, smoking , EtOH, DM, prior MI, HF, OSA • Often due to precipitant: surgery, infection, MI, PE • Co-exists w/ atrial flutter
Valvular AF	• Secondary to mitral stenosis, prosthetic valves, or valve repair

Types of Afib	
Paroxysmal	Termination within 7 days (either self or cardioverted)
Persistent	Continuous lasting > 7 days
Long-standing persistent	Continuous lasting > 12 months
Permanent	Used with decision to stop further treatment attempts for NSR

Afib with RVR

Afib with RVR Management		
Definition • Afib with HR > 110 		
Stable Systolic BP > 90	**Peri-stable** Systolic BP 80-90	**Unstable** Systolic BP < 80

• Rate control: BB, non-DHP CCB
• IV if HR > 130 or with symptoms
  ○ Metoprolol IV 2.5-5.0 mg over 2 min (repeat as required q5min for 15 mg max)
  ○ Metoprolol PO: up to 400 mg daily
  ○ Diltiazem IV: IV bolus 0.25 mg/kg (avg 10 -25 mg) over 2 min (Repeat as required q10-15min)
  ○ Diltiazem PO: up to 360 mg daily
  ▪ Caution in pts with renal dysfxn, hepatic dysfxn, LVEF < 40% (can results in cardiogenic shock), ADHF
	• Consider BP-sparing agents (amiodarone, digoxin)
	• Amiodarone 150 mg IV over 10 min, then repeat x1, then gtt 1 mg/min
	  ○ Amiodarone okay in pts w/ long QTc in this situation
	• Digoxin 0.5 mg SIV then 0.25 mg IV q6hr x2
		• Symptoms: HR > 150, shock (AMS, cool extremities, pulmonary edema)
		• Synchronized cardioversion (start 150 J)
		• First line pressor: phenylephrine

Atrial Fibrillation Treatment

Treatment algorithm	
• Rate and rhythm control not significantly different for symptoms, CV mortality, stroke risk • Recent trials suggest rhythm control as first line [AFFIRM, EARLY-AF, EAST-AFNET4, AF-CHF]	
Rate control [Trials: RACE, RACE II]	**Rhythm control**
• Goal HR < 110 (if patient stable) • BBs (first line and superior to CCBs), non-DHP CCBs • Stricter HR control (HR < 80) non-superior with respect to outcomes (CV death, stroke, bleeding, arrhythmia, hospitalization) • Stricter HR goal in pts that are younger and w/ HF	• Necessary if sx persist while on rate control, adverse effects on BP, comorbid HF (systolic dysfxn) • No structural heart disease: amiodarone, dofetilide, dronedarone, sotalol • Structural heart disease, CAD: amiodarone, dofetilide, dronedarone, sotalol • HF or LVH: amiodarone, dofetilide • Pill in pocket: indicated for pts w/ paroxysmal AF, infrequent episodes, and may not warrant long term medications (PRN flecainide)

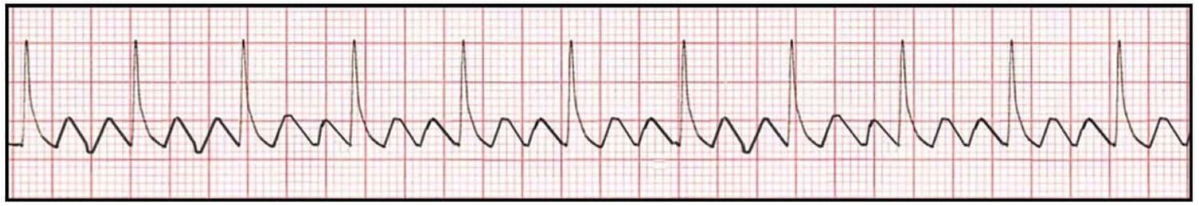
Cardioversion	
Patient selection	Hemodynamically unstable w/ sxs hypotension, AMS, HF
Elective	3-6 weeks of anticoagulation required TEE prior to cardioversion necessary to rule out clot
Electrical (synchronized cardioversion)	Ideal for first episodes, younger pts (< 65), no valve dz, no LA thrombus or prior thromboembolic event, on therapeutic anticoagulation
Pharmacological (i.e. gtt)	Flecainide, amiodarone
After cardioversion	At least 4 weeks anti-coagulation

Catheter ablation	
How	Pulmonary vein isolation
Affect on morbidity/mortality	Decreased [Trails: CASTLE-AF, CABANA]
Comparison to anti-arrhythmic agents	Decreases AF recurrence rate [Trials: MANTRA-PAF, RAAFT-2, SARA]

Atrial Flutter

Definition

- Narrow complex tachycardia
- "Sawtooth" pattern: II, III, aVF
- Regular atrial activity at 300 BPM
- Reentrant circuit around tricuspid valve isthmus
- Ventricular activity is a fraction of atrial rate
  - 2:1 block = 150 BPM
  - 3:1 block = 100 BPM
  - 4:1 block = 75 BPM
- Symptoms: palpitations, chest pain, dyspnea, fatigue, light-headed



Management

Stable and asymptomatic (HR < 110)	Medications
Stable and symptomatic	Medications (HR goal < 110)
Hemodynamically unstable	• Synchronized cardioversion • Failure: pharmacological approach via amiodarone load (150 mg over 10 min) or diltiazem load (2.5 mg/min until HR < 100, max 50)

Catheter ablation

How	Pulmonary vein isolation Anatomic target: Cavotricuspid isthmus (between tricuspid valve and IVC)
Affect on morbidity/mortality	Decreased [Trails: CASTLE-AF, CABANA]
Comparison to anti-arrhythmic agents	Decreases AF recurrence rate [Trials: MANTRA-PAF, RAAFT-2, SARA]
Comparison to AF	More successful for aflutter

Anticoagulation

AC for Non-Valvular Afib and Aflutter	
Afib vs Aflutter	Thromboembolism risk lower for aflutter
CHA2DS2-VASc (MDCalc) CHA_2DS_2-VASc Score CHF (heart failure) 1 Hypertension 1 Age ≥ 75 2 Diabetes 1 Stroke 2 Vascular Disease 1 Age 65-74 1 *Sex Category (female)* 1	Initiate AC (AHA/ACC/HRS 2019 guidelines): ≥2 for males ≥3 for females First line: DOAC >> warfarin Lifelong

Agents for anticoagulation	
IV/gtt	• Lovenox therapeutic (1 mg/kg) when GFR > 30 • Heparin gtt (monitor Xa)
PO	• DOAC (non-valvular AF): apixaban, rivaroxaban, dabigatran, edoxaban • Warfarin (monitor INR)
Bridging	Stop DOAC and start heparin product at time of next scheduled DOAC dose

Valvular afib	
Anticoagulant of choice	Warfarin Goal INR: 2.0 – 3.0
Valvular replacements (mitral and/or aortic)	Warfarin Goal INR: 2.5 – 3.5
Low risk patients (CHA2DS2-VASc ≤1)	ASA 81 qD

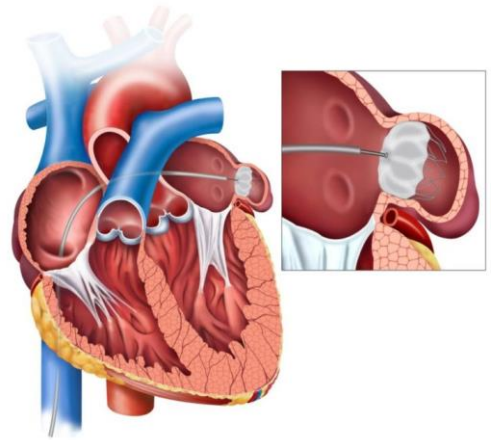
When to dose reduce

Meet 2 out of 3 criteria

1. Age ≥ 80
2. Body weight ≤ 60 kg
3. Serum Cr ≥ 1.5 mg/dL

Dose reduce from 5mg BID to 2.5 mg BID

LAA appendage closure	
Most common source of thrombi afib	LA appendage
Watchman procedure	Device-mediated LAA closure



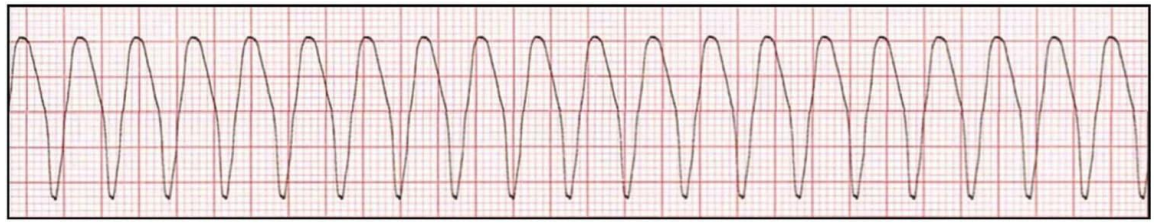
Ventricular Tachycardias

Definition: wide complex tachycardia (WCT), QRS > 120 ms, HR > 100

Monomorphic VT

Single ventricular focus
DDx: ischemia, structural heart disease, idiopathic

- **Non-sustained VT:** < 30 sec
  Manage via AV node blockade (BB, CCB), replete lytes
- **Sustained VT:** > 30 sec
  Manage via antiarrhythmics (amiodarone)
- **Unstable**: hypotension, AMS
  + pulse: synchronized cardioversion (100 J)
  - pulse: defibrillation



'R on T' phenomenon
- Common mechanism for TdP and polymorphic VT initiation
- Occurs when PVC (ventricular ectopic beat) occurs during preceding T wave (of preceding beat)

Polymorphic VT

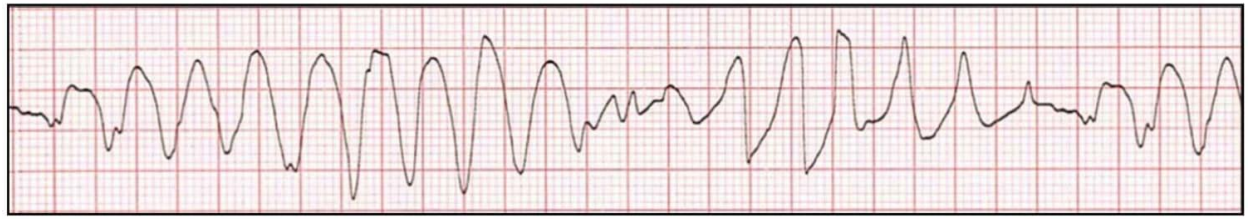
Multiple ventricular foci
DDx: ischemia (acute, ICM, CAD), prolonged QTc

First step: evaluate for ischemia and need for revascularization

Stable:
- Magnesium 2-4 g IV x1
- Increase HR via chronotropic agents dopamine, epinephrine; or overdrive pacing (pace HR faster than intrinsic rate)
- Decrease QTc via lidocaine
- Avoid bradycardia (avoid amiodarone, BB, CCB)

Unstable: hypotension, AMS
- Defibrillation

Torsades de Pointes: form of polymorphic VT often 2/2 prolonged QTc; manage via polymorphic VT algorithm



Ventricular Tachycardias

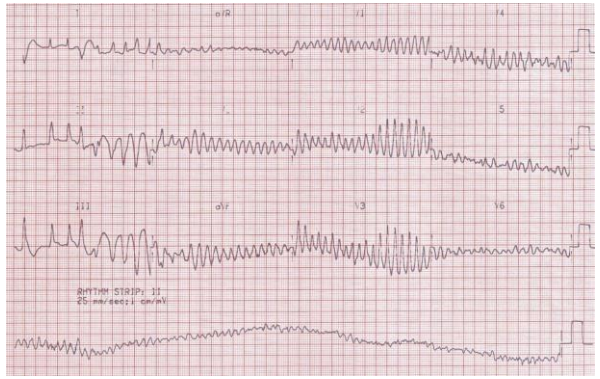
Definition: wide complex tachycardia (WCT), QRS > 120 ms, HR > 100

VT Storm

Multiple sustained (> 30 sec) episodes of VT within 24 hrs

Management:

- Reduce autonomic tone: intubation, sedation
- Treat underlying ischemia: revascularization (improve coronary perfusion), IABP (reduce afterload)
- Overdrive pacing: pace at rate faster than VT
- Amiodarone: 150 mg IV bolus + gtt. Administer with propranolol 60 mg q6hr
- In persistent VT with ICM and ICD, ablation is superior to escalation of antiarrhythmic drugs (lower rates of death, ICD shocks, VT storm events) [VANISH Trial]

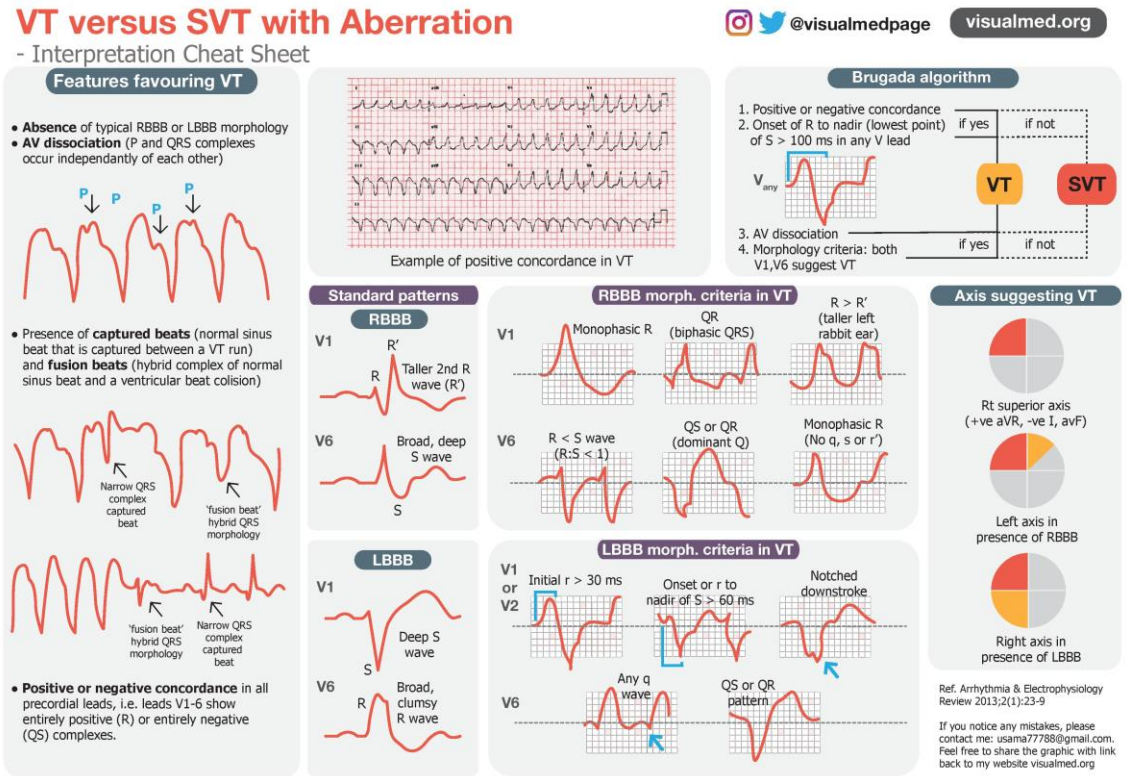


VT Management

Hemodynamically stable	• First line unless Torsades: amiodarone 150 mg IV bolus + gtt @ 1 mg/min x 6hr, then 0.5 mg/min x 18hr • Second line but preferred if VT 2/2 prolonged QT: lidocaine 1-1.5 mg/kg (100 mg), then 0.5-0.75 mkg/kg q5-10min. Continue at 1-4 mg/min if VT recurs • Third line, avoid in prolonged QT: procainamide 20-50 mg/min until VT terminates
Hemodynamically unstable	ACLS algorithm
All	Address underlying process: active ischemia, CAD, ischemic scar, electrolyte changes (K > 4.0, Mg > 2.0)
Chronic treatment	• BB: initial therapy for symptomatic non-sustained VT (prevent ectopy) • Antiarrhythmics • AICD • Ablation

Ventricular Tachycardias

Differentiating VT from Supraventricular VT with aberrant conduction	
Favoring VT	• Age > 35, prior MI, structural heart disease, family hx sudden cardiac death • EKG: broad QRS complex (> 160 ms) • Extreme NW axis • Concordance of precordial lead QRS complexes (V1-V6 all + or -) • AV dissociation • Capture beats: SA node transiently captures ventricles (meaning P wave with narrow/normal QRS < 120 ms) • Fusion beat: partial depolarization of ventricular by underlying supraventricular rhythm
Favoring supraventricular VT with aberrant conduction	• Pre-existing BBB • QRS with initial sharp deflection, followed by terminal broad deflection



Bradycardia

Definition and Differential	
Sinus bradycardia	Heart rate < 60
Symptoms	• Hypotension • Shortness of breath • Lightheadedness/syncope • AMS • HF (pulmonary edema)
Extrinsic	• Increased parasympathetic tone • Illness (hospitalization) • Medications (BB, CCB, digoxin, amiodarone) • Increased ICP
Intrinsic	• Nocturnal • Post-operative (stent, valve replacement) • Sick sinus syndrome • AV nodal blockade • Hypothermia • Hypoxia • K imbalance • Endocarditis • Athletic hearth • Infiltrative disease (amyloid, sarcoid, hemochromatosis) • Lyme disease

AV Nodal Blocks	
First degree	Prolonged PR interval (> 200 ms, nrl = 120-200 ms)
Second degree Mobitz I (Wenckebach)	Progressively increasing PR interval leading to dropped/skipped beat (non-conducted P wave)
Second degree Mobitz II	Fixed PR interval with random/sporadic non-conduced P waves

Third degree	• Complete AV dissociation (P and QRS occur entirely independent of each other)
	• Produces two types of rhythms:
	  ○ Junctional escape (HR 40-60, narrow QRS)
	  ○ Ventricular escape (HR 20-40, wide QRS)

Sick Sinus Syndrome (SSS)	
What	Sinus node dysfunction Symptomatic bradycardia, can alternate with tachycardia Chronotropic incompetence (failure to elevate HR with exertion)
Commonly seen with	Atrial fibrillation
Management	Treat underlying reversible causes Consider permanent pacemaker (dual chamber 2/2 concomitant AV nodal dysfunction)

Bradycardia

Management	
Asymptomatic	No treatment indicated
Symptomatic	Workup to address underlying, reversible causes (ex. infxn, ACS)
Hemodynamically unstable	ACLS algorithm • Atropine 0.5 - 1.0 mg bolus, repeat q3-5 min for max dose 3g

Bradycardia Toxicity	Antidote
Beta blocker	Glucagon 5 mg q10min (up to 3 doses). Insulin 1 U/kg bolus
Calcium channel blocker	Calcium gluconate 3 g, insulin 1 U/kg bolus
Digoxin	Digoxin ab 10-20 vials
Opioid	Naloxone 0.4-0.8 mg IV
Organophosphate	Atropine 2 mg IV (x2 doses q5-30 min), pralidoxime 1-2 g IV over 15 -min

QT-QTc Prolongation

Overview	
QT interval	Repolarization time of ventricles Interval inversely proportional to heart rate
QTc	Corrected QT interval for heart rate Estimates QT at standard heart rate 60 bpm (standardized comparison)
Prolonged QTc risk	Male: > 450 ms Female: > 470 ms Increased risk of developing Torsades de Pointes → VF
Monitoring QT interval	EKG on admission Check QTc before, 12 hrs after initiation/dose change of QT-prolonging medication, new bradyarrhythmia, severe electrolyte imbalances

Long QT syndrome (LQTS)	
Congenital LQTS	Genetic Most often incidentally found on EKG, but sx include (pre)syncope, sudden cardiac death, hemodynamic compromise
Management	Beta blockers, if prior arrest (ICD)
Acquired LQTS	Often medication-related Stop offending agent if QTc > 500 ms, or if QTc change > 60 ms Electrolyte monitoring (K > 4.0, Mg > 2.4) Aggressive repletion acceptable in TdP (K > 4.5-5.0)

QT-QTc Prolongation

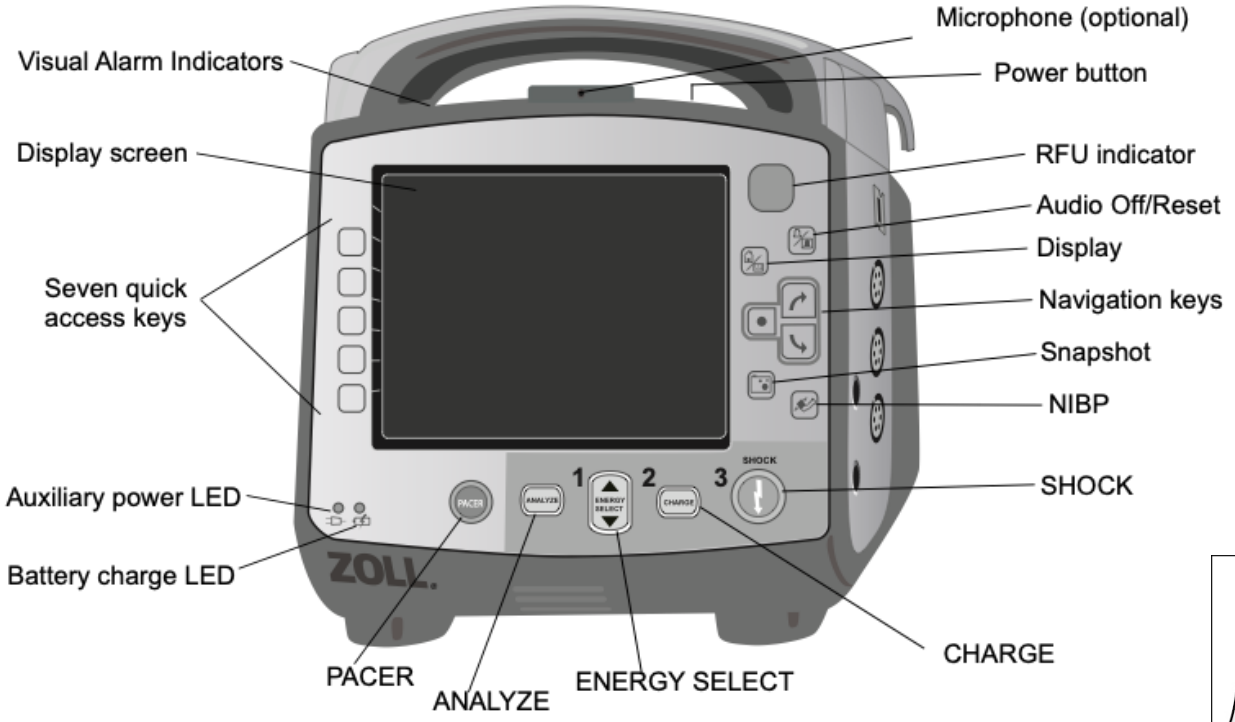
QT prolongation and TdP risk factors	
Demographics	Elderly, female, congenital LQTS
Comorbidities	Renal failure, HF, MI, liver dysfunction
Rhythm-related	QTc > 500, PVCs, bradycardia, AV block
Electrolytes	Hypomagnesemia, hypokalemia, hypocalcemia
Medications	QTc-prolonging meds, diuretics, beta blockers
Other	Hypothermia, myocardial ischemia, post-cardiac arrest, elevated ICP

QT prolonging medications	
Antiarrhythmics	Class IA: quinidine, disopyramide, procainamide Class III: amiodarone, dofetilide, ibutilide, sotalol
Antidepressants	TCAs, SSRIs (citalopram, escitalopram, fluoxetine)
Antiemetics	Droperidol, Ondansetron, Metoclopramide
Antimicrobials	Macrolides (-mycin), Fluoroquinolones (-oxacin), Anti-fungals (fluconazole, voriconazole), Anti-malarial (quinine, chloroquine)
Antipsychotics	Haloperidol, Thioridazine, Chlorpromazine, Quetiapine, Risperidone, Olanzapine, Aripiprazole
Others	Donepezil, Hydroxyzine, Methadone, Propofol

Zoll X Series Overview

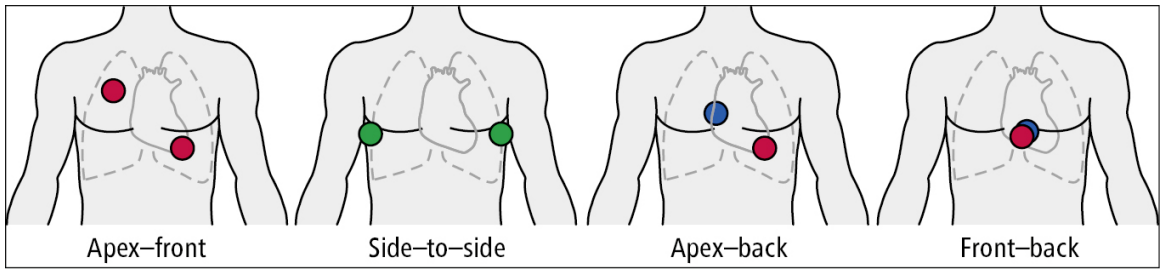
Zoll X Series

Device for external defibrillation, cardioversion, and transcutaneous pacing used at UPMC MUH, PUH, SHY, VAPHS



Tips and Tricks	
Patient set-up	Remove all clothing, ensure dry, remove chest hair (shave if necessary) Ensure patient is clear before any shock or defibrillation
Failure to capture	Ensure pad placement is correct Increase output (J)
Failure to sense	Reposition pads Trial asynchronous pacing

Pad placement options



Instructions for Zoll X Series

Synchronized cardioversion

Indications: unstable SVT, VT, unstable Afib, unstable Aflutter

1. Turn ON
2. Press *Manual* > change to *ALS*
3. If there is shockable rhythm on pulse+rhythm check, then press *Charge* and shock when ready
4. If no shockable rhythm, proceed with defibrillation

Defibrillation

Indications: VT without pulse, vfib

1. Turn ON
2. Press *Manual* > change to *ACLS*
3. Select desired energy (J) based on rhythm
4. Pressy *Sync On/off* button. Confirm Sync marker (arrow) appears over each R wave
5. Charge and shock when ready
6. if additional shocks are necessary, increase energy as needed, confirming Sync each time

Transcutaneous pacing

Indications: unstable bradycardia

1. Turn ON
2. Press Manual > change to ALS
3. Switch to *Pacer*
4. Set pacer rate to 10-20 BPM higher than intrinsic HR (over pace)

Unknown or absent HR? Start at 100 BPM

Cardioversion Protocol

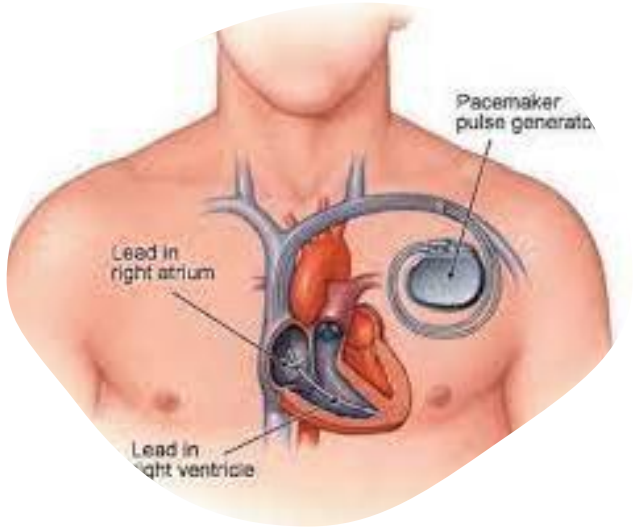
Sedation Options
• Lidocaine 0.5 mg/kg • Fentanyl 1 mcg/kg (typically 50 mcg) • Midazolam 2 mg • Etomidate 0.1 mg/kg > 0.05 mg/kg • Acceptable alternative if available faster: morphine 4 mg then lorazepam 2 mg

Anticoagulation Recommendations Options	
Pre-procedure	Onset < 48 hours: proceed without AC
	Onset > 48 hours: AC 3 weeks prior to electrical cardioversion or TEE immediately prior
Post-procedure	AC for at least 4 weeks post-synchronized cardioversion

Current of choice	
Indication	**Current***
Unstable atrial fibrillation (narrow, irregular)	120-200 J
Unstable atrial flutter	200 J
Supraventricular VT (SVT) (narrow, regular)	200 J
VT w/ pulse (wide, regular)	200 J
VT w/o pulse (wide, irregular)	*defibrillate*
VF	*defibrillate*
**While one may see different currents used, 200 J is standard first line, especially for emergent/urgent situations*	

Permanent Pacemakers

Overview	
What	• Performs sensing and pacing functions
Components	• Pulse generator • One or more electrodes (leads) to deliver the impulse
Transvenous systems	• Used by majority of PPMs • Transvenous leads conduct pacing signal to myocardium
Leadless systems	• Only for RV pacing • Single unit is both pulse generator and electrode (placed in RV)
Lead locations and chamber paced	• Single chamber (one lead in RA or RV) • Dual chamber (two leads, RA and RV) • Biventricular (three leads, RA, RV, LV) • LV lead (placed through coronary sinus)



Indications
• Well-documented persistent symptomatic bradycardia not due to reversible causes • Symptomatic second-degree AV block, type I (Wenkebach) • Second-degree AV block, Type II (Wenkebach) • Complete (third degree) AV block

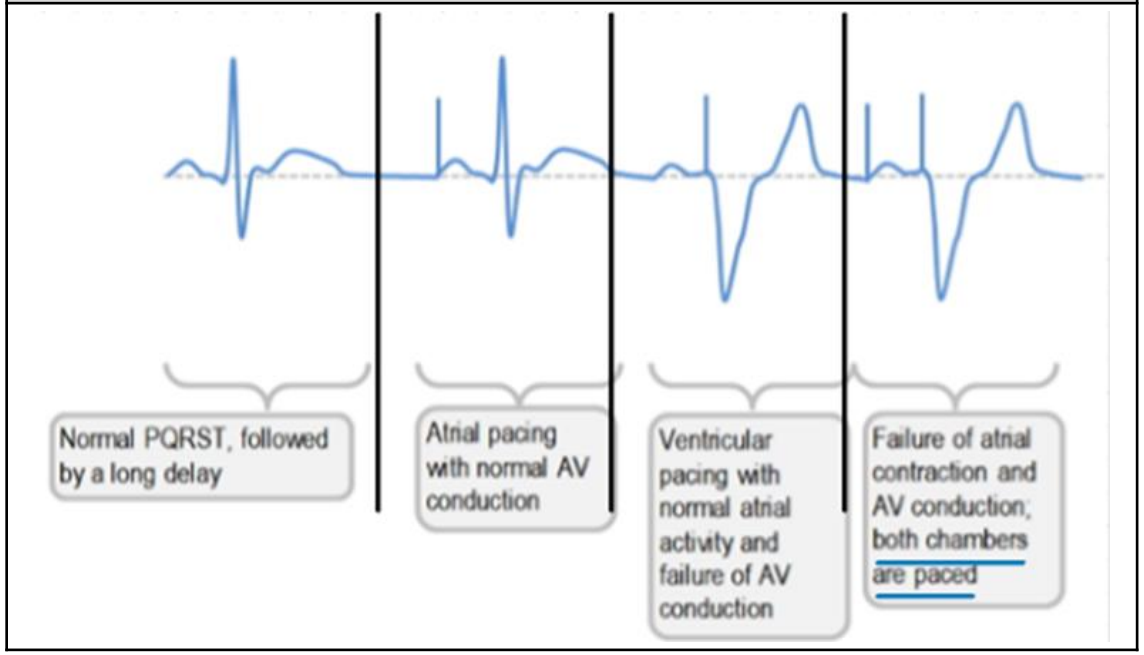
Permanent Pacemakers

Nomenclature

Mode and function designated by 5-letter code, first 3 letters always reported

1	2	3	4	5
Chambers paced	Chambers sensed	Response to sensing	Rate response	Multisite pacing
O = None A = Atrium V = Ventricle D = Dual (A+V)	O = None A = Atrium V = Ventricle D = Dual (A+V)	O = None I = Inhibition T = Triggered D = Dual (T+ I)	O = None R = Rate responsive (can increase rate to meet physiologic demand)	O = None A = Atrium V = Ventricle D = Dual (A+V)
Most common pacing modes				
AAI (Single chamber)	Atrium paced (A), senses atrial activity (A), atrial activity (high atrial rate) inhibits atrium pacing (I)			
VVI (Single chamber)	Ventricle paced (V), senses ventricular activity (V), ventricular activity (high ventricular rate) inhibits ventricular pacing (I)			
DDD (Dual chamber)	Can pace atrium and/or ventricle (D), sensed atrial and ventricular activity (D), atrial activity (high or lower rate or AV block) determines atrium and/or ventricular pacing (D)			

Chamber paced illustration



Device interrogation

When	ICD shocks, failure to capture or sense, decompensated heart failure, symptoms of palpitations or syncope, surgery
How	Call EP fellow on call Know device manufacturer (St. Jude, Boston Sci, Medtronic)

Applying a magnet to…

Pacemaker	Converts it on **asynchronous mode**, meaning a pre-determined fixed pacing with no sensing (second letter is O)
ICD	Shocks will not be given (Anti-arrhythmia function off)
When to use magnet	Surgery Concern with sensing dysfunction or inappropriate shock

Implantable Cardiac Defibrillators (ICDs)

ICD Overview	
Function	• Terminate ventricular arrhythmias by *shocking* (cardioversion if synchronized or defibrillation if not synchronized) • More recent ICDs have antitachyarrhythmia pacing (except subcutaneous ICD)
Leads locations and numbers	• ICDs can have one (always RV) or two leads (RV and RA) • Cardioverter/Defibrillator lead (coiled lead)**:** located in the RV (always); can have two coils in the same lead • Sensing/pacing lead: not always present; 2nd lead and located in RA; possesses no shock function or coil

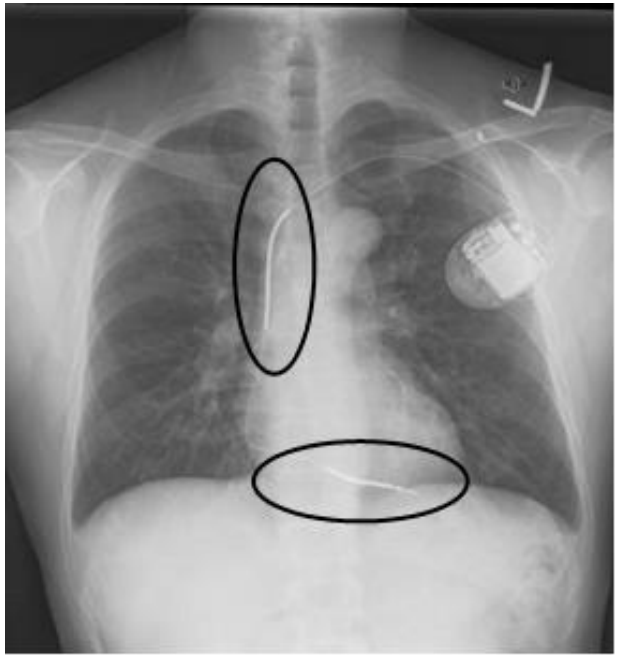
Indications	
Primary Prevention	• Prior MI with LVEF ≤30%, >40 days after MI • NYHA class II-III with LVEF ≤35%
Secondary Prevention	• Survivors of arrest from VF or VT without reversible cause • Structural heart disease with sustained VT • Syncope with inducible VT or VF at EP Study
Class IIa Indications	• Unexplained syncope with sign LV dysfunction and NICM • HOCM with 1 or more risk factors (RF) • ARVD/C with 1 or more RF for SCD • Long QT syndrome, syncope or VT while receiving beta-blockers, non- hospitalized patients awaiting heart transplant • Brugada syndrome

ICD Contraindications
• Arrhythmia with a reversible etiology • VT/VF occurring within 48 hours of acute MI • Life expectancy < 1 year • Incessant VT/VF, consider other therapies (ablation) first • Inability to follow up • NYHA IV HF refractory to medical management and not a transplant or CRT candidate • Syncope without inducible VT/VF or heart disease

CXR ICDs and PPMs

Chest Xray ICD characteristics

ICD with one RV lead and two coils (black circles)

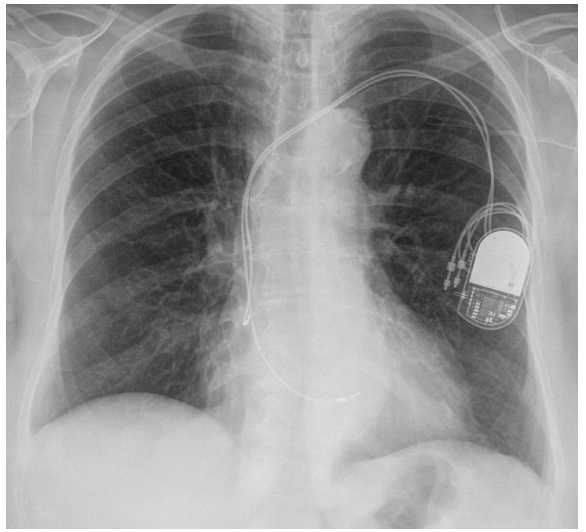


ICD quick facts

1. ICDs have coils (black circle) while pacemakers do not have it (no coils, only wires)
2. ICD can also pace (assuming a PPM function) and may have more than one lead

Chest Xray dual chamber (RA and RV leads) pacemaker

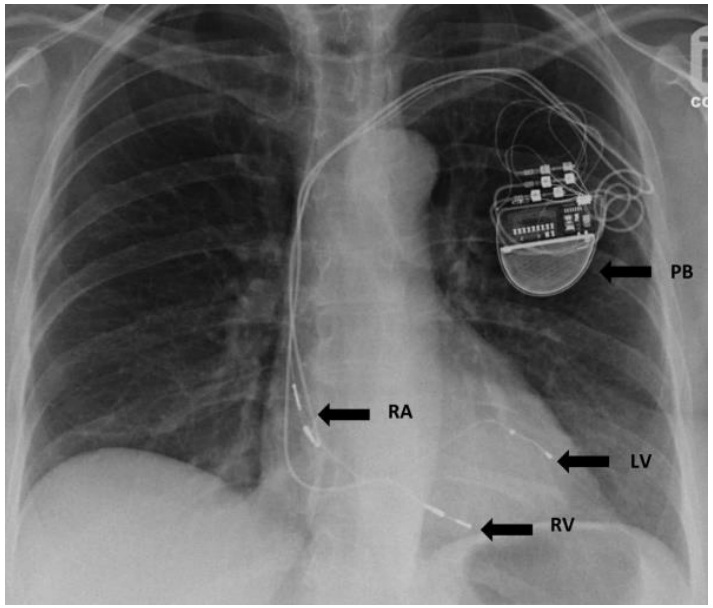
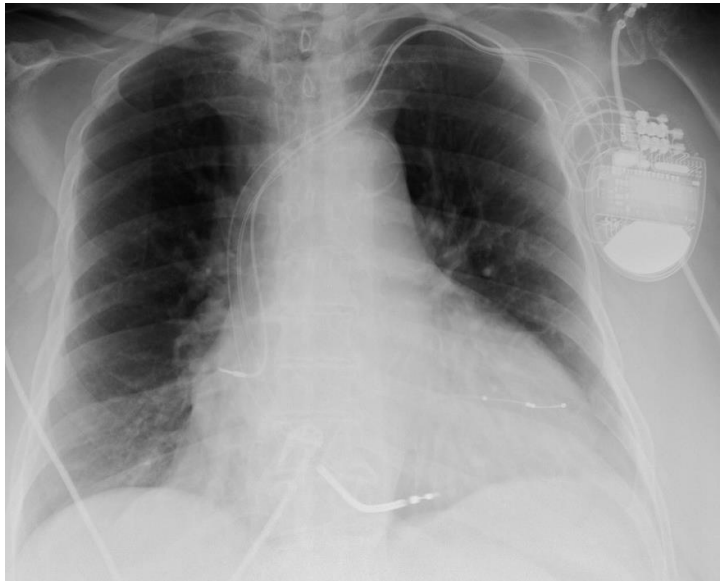
No coils present, therefore, it is a "pure" pacemaker without ICD function (no cardioversion or defibrillation)



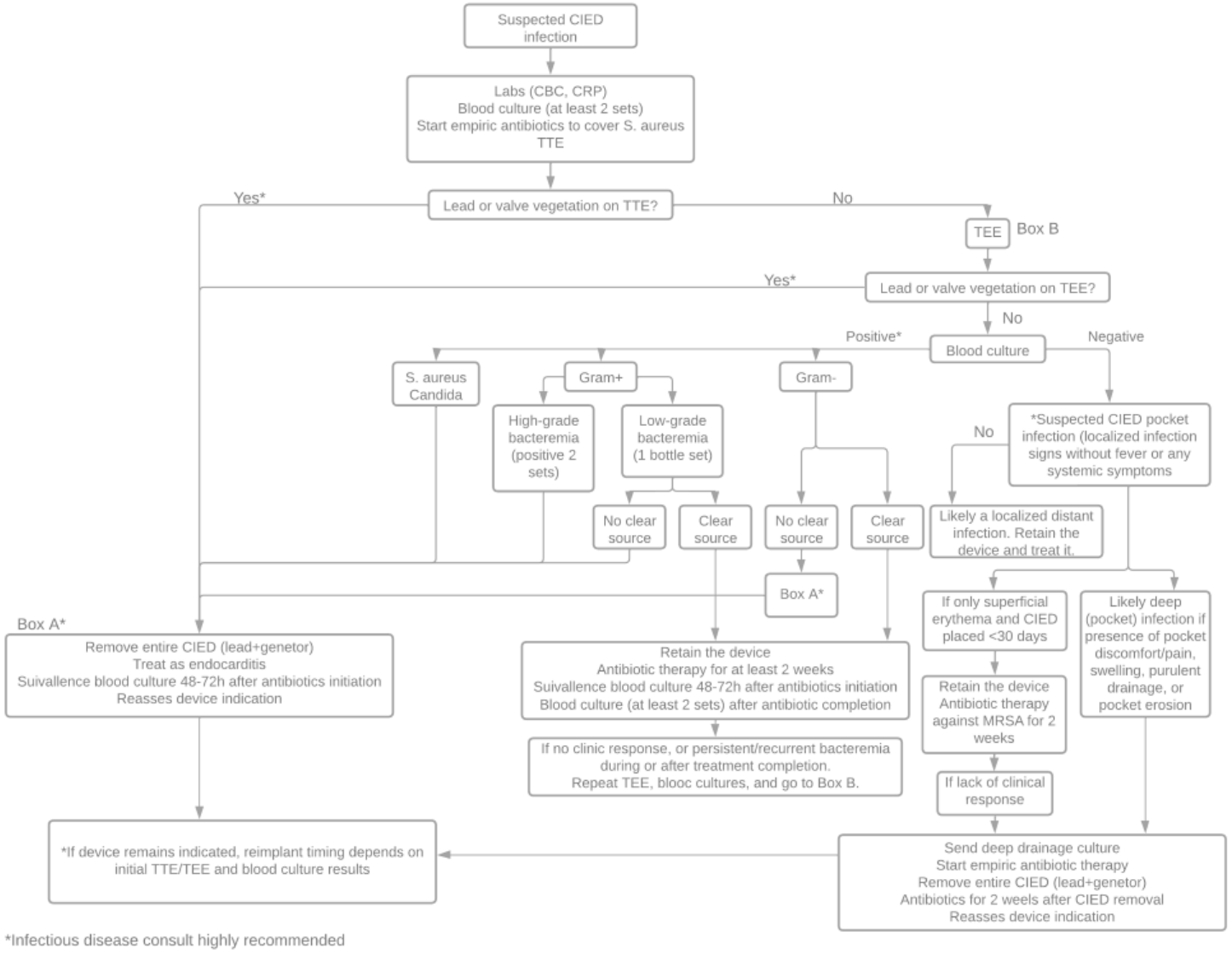
Cardiac Resynchronization Therapy (CRT)

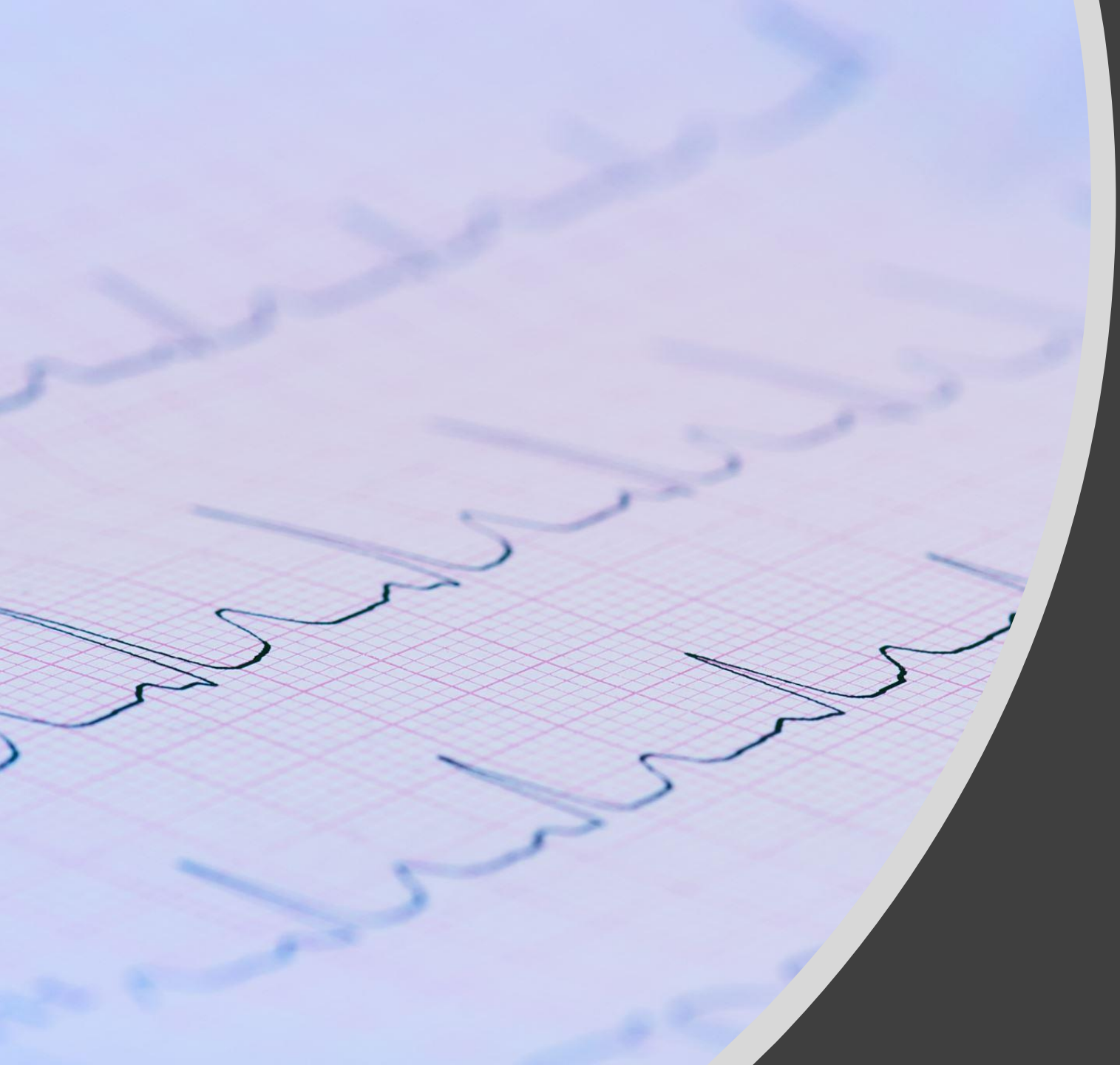
CRT Overview	
Function	• Ensures both ventricles contract together (without pathological delay) through biventricular pacing • Resynchronization therapy is performed by a LV lead • CRTs have three leads
Types of leads (RV, RV, LV leads)	• LV lead: implanted through the coronary sinus vein • CRT-P: Pure biventricular pacing (no ICD function, no coils) • CRT-D: CRT-P plus ICD functions (coils)
Indications	**Indications: LVEF ≤35%** and **sinus rhythm** and **NYHA II/III/IV** plus one of the following: • Class I: LBBB and QRS≥150s • Class IIa: LBBB and QRS 120-149s • Class IIb: Non-LBBB and QRS≥150s • Class III: Non-LBBB and QRS 120-149s
Contraindications	• LVEF ≤35% and sinus rhythm and NYHA II/III/IV and non-LBBB and QRS <120s • System infection (risk of device or lead infections)
Notes	• Patients with PPM or ICD with CRT should be upgraded • PPMs treats bradycardia (no coils); ICDs cardiovert/defibrillate terminating arrhythmias (coils); CRT synchronize both ventricles contraction by biventricular pacing (three leads)

Cardiac Resynchronization Therapy (CRT)

CRT-P	CRT-D (CRT-P plus ICD function)
LV lead through the coronary sinus vein. There is no coils, so it is a CRT-P 	Three leads (RA, RV, LV) with a coil on the RV lead (ICD function), making it CRT-D 

Cardiac Device Infections





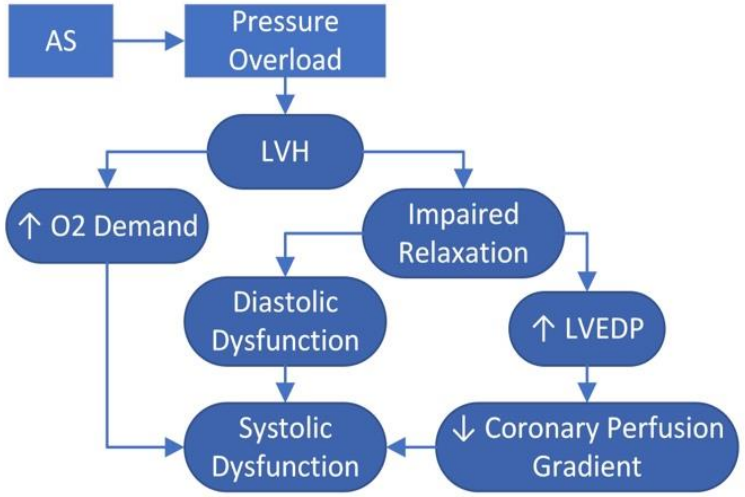
Valvular Heart Disease

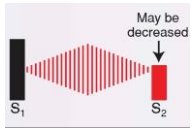
Section Editors:
Mohanad Hamadi, MD
Gautam Rangavajla, MD

Aortic Stenosis Symptomatology

Types	
Calcific	Most common, usually > 65-75yrs
Bicuspid (congenital)	Presents earlier ~50yrs
Rheumatic	Uncommon, typically associated with mitral valve disease

Pathophysiology



Physical Exam	
Pulsus parvus et tardus	Slow rising, late peaking, low volume carotid pulse
Evidence of severe disease	Soft or absent A2
Murmur 	• Systolic ejection murmur in R 2nd intercostal space • Radiation to carotids or apex (Gallavardin phenomenon) • Increased intensity: leg raise (inc LV volume) • Decreased intensity: hand grip (inc afterload), Valsalva (dec LV volume) • Pitch: crescendo-decrescendo (late peaking = more severe)

Symptoms and Associations

- Angina (supply and demand mismatch)
- Syncope
- Dyspnea on exertion
- Heart failure
- Symptoms of IE or cardioembolic stroke
- GI bleeding secondary to colonic AVMs (Heyde Syndrome)

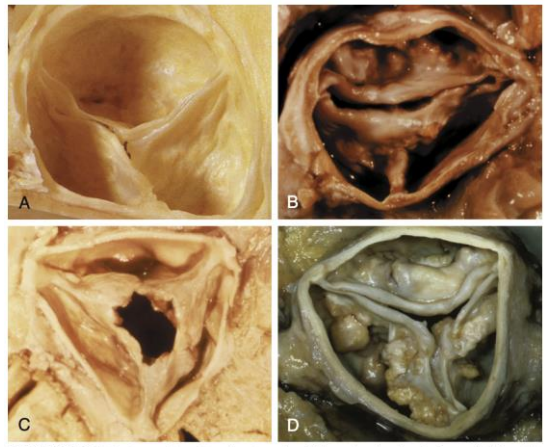


FIGURE 72.1 Major types of aortic valve stenosis. A, Normal aortic valve. **B,** Congenital bicuspid aortic stenosis. A false raphe is present at 6 o'clock. **C,** Rheumatic aortic stenosis. The commissures are fused with a fixed central orifice. **D,** Calcific aortic stenosis. (**A** from Manabe H, Yutani C, editors. *Atlas of Valvular Heart Disease*. Singapore: Churchill Livingstone; 1998:6, 131; **B-D** courtesy Dr. William C. Roberts, Baylor University Medical Center, Dallas, Tex.)

Aortic Stenosis Workup

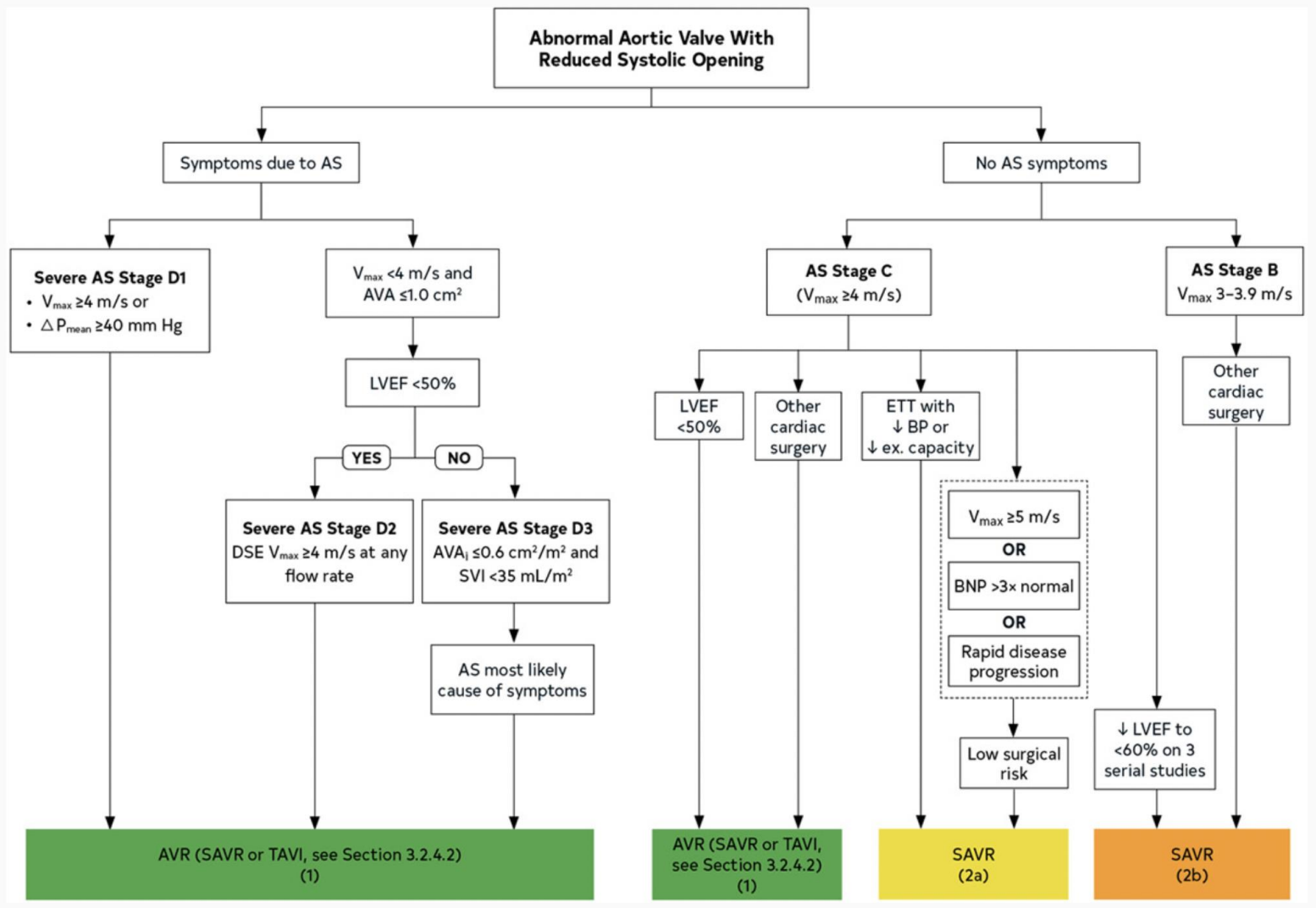
Grading Severity (TTE)	Mild	Moderate	Severe
Valve Area (AVA)	> 1.0 cm^2	> 1.0 cm^2	< 1.0 cm^2
Peak Velo (AV Vmax)	2.0-2.9 m/s	3.0-3.9 m/s	> 4.0 m/s
Mean Gradient (MG)	< 20 mmHg	20-39 mmHg	> 40 mmHg

LFLG AS Post Dobutamine	AVA	AV Vmax	Mean Gradient
Pseudostenosis	> 1.0 cm^2	< 4.0 m/s	< 40 mm Hg
True Stenosis	< 1.0 cm^2	> 4.0 m/s	> 40 mm Hg

Diagnostics	
EKG	LVH (chronic)
TTE	• Shows severity (AV area, peak AV jet velocity, mean transvalvular gradient) • Determines cause (calcific vs bicuspid)
Cardiac CT	Useful in assessing AV calcification, aortic dilatation, surgical planning (esp. before TAVR)
Cardiac MR	Useful in assessing LV function, aortic dimension, myocardial fibrosis (poor prognostic sign)
Cardiac cath	Used when non-invasive tests are inconclusive

Low Flow Low Gradient Aortic Stenosis	
Definition	AVA < 1.0 cm^2, Vmax < 4.0 m/s or MG < 40mmHg, LVEF < 50%
Dobutamine stress echo	Distinguish true stenosis from pseudostenosis
	• True stenosis: valve is truly restricted from opening independent of flow • Pseudostenosis: valve appears stenotic as poor flow/contractility does not fully open valve; increased contractility/flow open valve

Aortic Stenosis Treatment



Disease Monitoring

- Mild aortic stenosis: TTE q3-5y
- Moderate aortic stenosis: TTE q1-2y
- Severe aortic stenosis: TTE q6-12 mo

Surgical Treatment

Balloon valvuloplasty	Limited role (bridge to AVR or palliative if not surgical candidate)
Aortic Valve Replacement (AVR)	• Symptomatic severe AV disease (stage D) • Asymptomatic severe AV disease w/ LVEF < 50% (Stage C2) • Severe AS undergoing other cardiac surgery (stage C or D)

Chronic Medical Treatment

- Does not slow progression of valve disease
- Treat HTN with afterload reduction (ACE/ARB)
- Treat other comorbidities: CAD, CHF, Afib

SAVR vs TAVR

SAVR	Preferred in pts < 65yo
TAVR	Preferred in pts > 80yo or high surgical risk candidates

Transcatheter Aortic Valve Replacement (TAVR)

Pre-Procedural Planning	
H&P	Last AC/AP dose, contrast allergy?, neurocognition assessment, peripheral pulses
Orders	CBC, CMP, PT/INR, T&S, Carotid US, PFTs
Mortality risk assessment	30 day (STS score), In-hospital (TAVR score)
QOL	KCCQ score
Frailty	Katz ADL, Four item score
Multimodality imaging	TTE: AVA, peak velocity, mean gradient Cardiac CT: coronary heights, annulus size, LVOT calcification CTA A/P: vessel diameter >5.5mm, tortuosity Coronary angiography and RHC: CAD, PAH, transaortic gradient

Who should get TAVR?

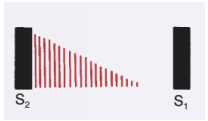
- Severe symptomatic aortic stenosis
- Predicted post-TAVR survival > 12 months
- Low risk patients with severely calcified aorta, chest wall deformity, O2 dependence, frailty

Post-Procedural Risks/Complications

Observe access site for complications
Conduction abnormalities (AVB, branch blocks)
Stroke
Paravalvular leak
Coronary artery occlusion
Valve malposition
Mortality

Aortic Regurgitation Symptomatology

Etiology		
Acute	*NA*	IE, aortic dissection, trauma
Chronic	Valvular disease	Bicuspid, calcific, IE, myxomatous, RA, SLE, valvuloplasty, AVR
	Aortic root disease	Chronic HTN, aortic dissection, aneurysm, Marfan's syndrome, Ehlers's Danlos syndrome, tertiary syphilis

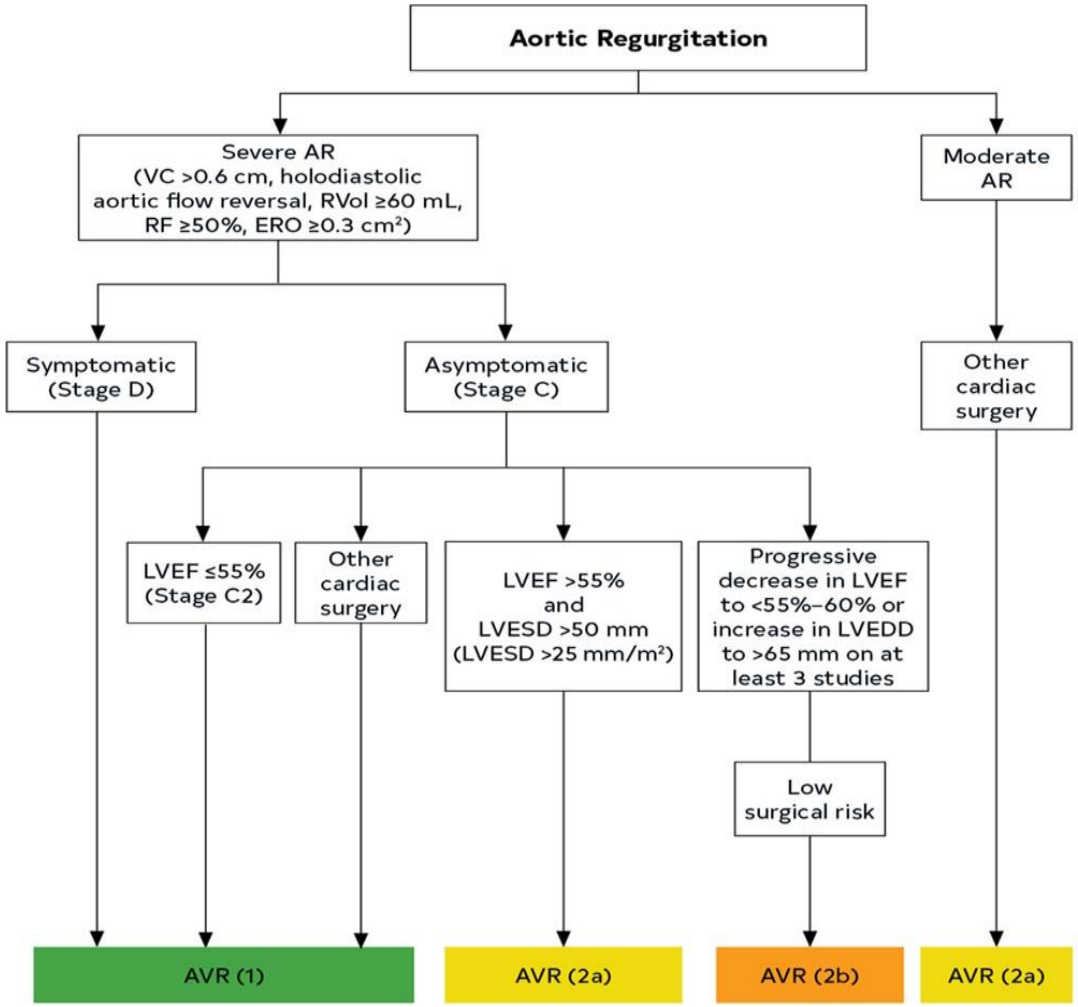
Physical Exam (Chronic)	
Systemic findings	Asymptomatic period → DOE, orthopnea, PND, exertional angina, palpitations Water-Hammer pulse Nailbed capillary Widened pulse pressure with decreased diastolic pressure
Evidence of severe disease	Longer duration of murmur
Murmur  	• Diastolic decrescendo blowing murmur immediately after A2 (best heard in LLSB in valvular disease or RLSB in aortic root disease) • Increased intensity: leaning forward on exhalation, hand grip (inc LV volume)
Note	Always look for physical signs of IE when AR is suspected

Physical Exam (Acute)
Sinus tachycardia Cyanosis Hypotension Acute pulmonary edema Cardiogenic shock

Aortic Regurgitation Diagnostics

TTE Findings for Severe AR					
Jet Width	**Vena Contracta**	**Regurgitant Volume**	**ERO**	**RF**	**Holodiastolic Flow Reverse**
> 65%	> 0.6 cm	> 60mL/beat	> 0.3 cm^2	> 50%	Present
Width of regurgitant flow expressed as percentage of LVOT	Narrowest diameter of flow stream	Volume of back flow across the aortic valve	Effective regurgitant orifice	Rvol / SV	Within the abdominal aorta

Aortic Regurgitation Treatment



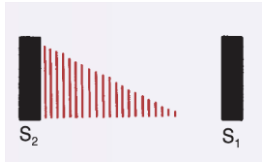
Acute AR	
Medical treatment (for stabilization)	Ionotropic support (i.e. Dobutamine)
	IV nitroprusside for decreased afterload
	Mechanical circulatory support (IABP, Impella, ECMO) contraindicated
Surgical treatment (for definitive management)	Urgent SAVR

Chronic AR	
Medical treatment	Not shown to improve outcomes
Surgical treatment	Symptomatic severe AR (Stage D)
	Asymptomatic severe AR w/ LVEF < 55% (Stage C2)
	Severe AR undergoing other cardiac surgery (Stage C or D)

Mitral Stenosis Symptomatology

Etiology
• Rheumatic heart Disease (>70%) • Congenital • Mitral Annular calcification • Carcinoid • Lupus/ Rheumatoid Arthritis

Symptoms
• Dyspnea, particularly w/ tachycardia (fever, exercise, stress) • Fatigue • Palpitations (esp. in setting of afib) • Thromboembolism (commonly cerebral) • Hemoptysis • Chest pain • Hepatomegaly • JVD • Lower extremity edema • Sx of left heart failure: orthopnea, PND • Sx of right heart failure: abd pain/edema, LE edema

Physical Exam	
Mitral facies	Pink/purple patches on cheeks 2/2 cutaneous vasoconstriction (sign of severe disease)
Evidence of severe disease	• Longer duration of murmur • Earlier snap • Mitral facies
Murmur 	• Loud S1 and Loud P2 • Opening snap (diastolic) • Reduced splitting of S2 (severe pHTN) • Apical, low-pitched rumbling mid-diastolic during expiration in L lateral decubitus position

Mitral Stenosis Diagnostics

Stages of Mitral Stenosis			
Stage	**Morphology**	**Hemodynamic Markers**	**Associated changes**
Stage A (At risk stage)	Domed appearance of valve	N/A	None
Stage B (Progressive)	Annulus > 1.5 cm^2 Commissural fusion + Doming	Increased trans-mitral flow velocity	Left atrial enlargement
Stage C (Severe asymptomatic)	Annulus <1.5 cm2 Commissural fusion + Doming	Pressure ½ time 150 ms or greater Trans-mitral pressure >5-10 mmHg	Left atrial enlargement Pulmonary artery systolic pressure >30mmHg
Stage D (Severe symptomatic)	As in Stage C	As in stage C	As in Stage C
Very Severe	Annulus <1 cm2	Pressure 1/2 time 220 ms or greater	

Workup	
TTE (to establish diagnosis)	• Mean diastolic pressure gradient (5-10 mmHg in severe disease) • Valve area • Pressure half time
TEE (when under consideration for intervention)	• Eval for left atrial thrombus • Additional information regarding valve anatomy for surgical planning
Exercise stress test	• If symptoms and TEE are discordant

Mitral Stenosis Treatment

Medical Treatment
• AVN blockade to decrease HR and increase diastolic time to improve LV filling across the stenotic mitral valve • Diuresis for pulmonary edema

Surgical Treatment		
Options	**Rheumatic mitral stenosis**	**Non-rheumatic calcific mitral stenosis**
Percutaneous Mitral Balloon Commissurotomy (PMBC)	NYHA class II-IV with MV area <1.5 cm^2	NYHA III-IV with MV area <1.5 cm^2 may be considered
Mitral Valve Repair or Replacement	• Stage D and not candidate for PMBC • Failed PMBC • No access to PMBC access • Requirement of other cardiac procedures	NYHA III-IV with MV area <1.5 cm^2 may be considered (Must discuss high risk and and patient values)

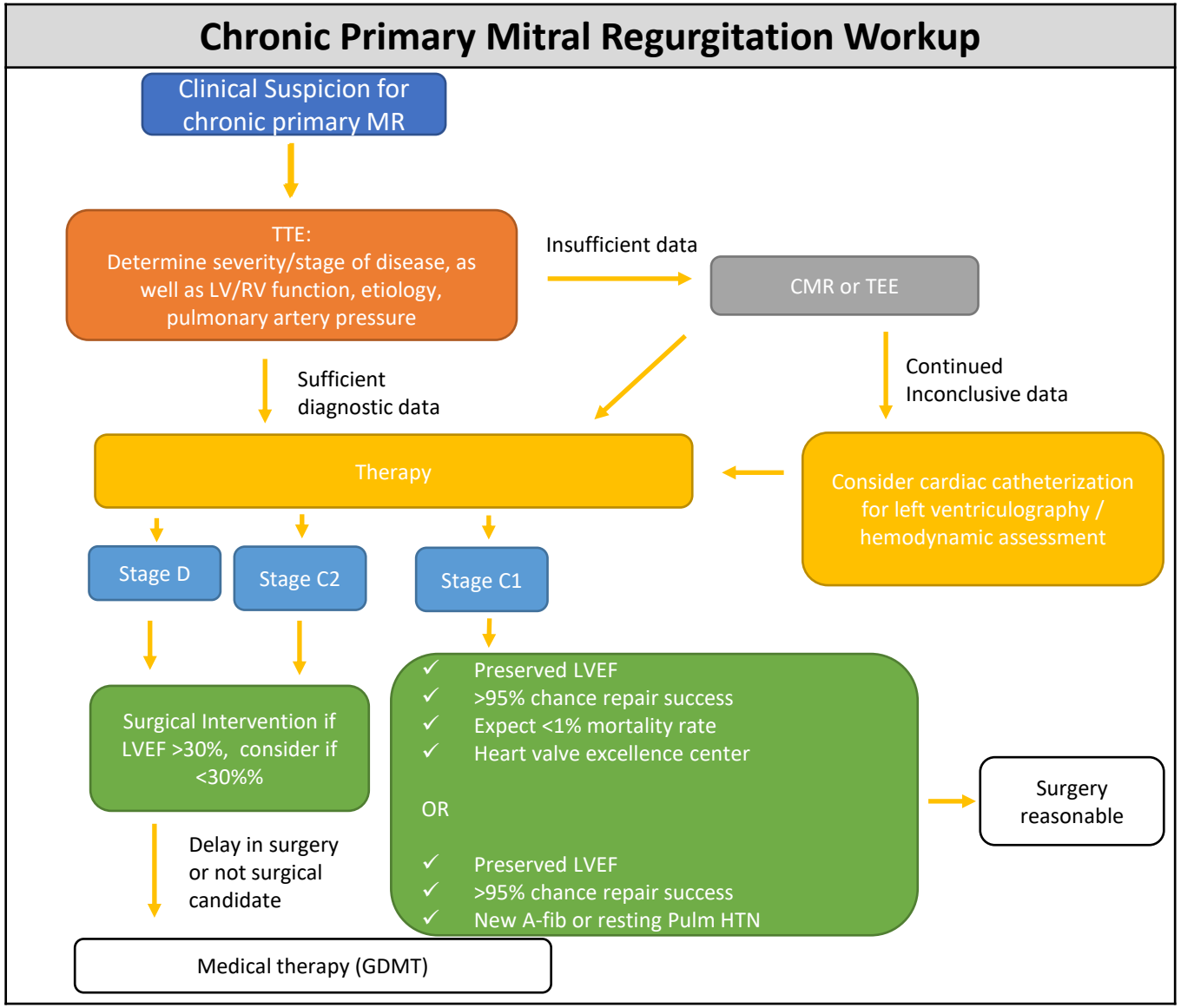
Mitral Regurgitation Symptomatology

Etiology	
Primary mitral regurgitation (valve components specifically)	• Degenerative Mitral disease • Infective endocarditis • Mitral annular calcification • Connective tissue disorders • Congenital • Radiation damage
Secondary mitral regurgitation (ventricular changes)	<u>Ischemic</u> Ischemic cardiomyopathy
	<u>Non-ischemic</u> • Dilated CM • HOCM • Chronic Atrial Fibrillation

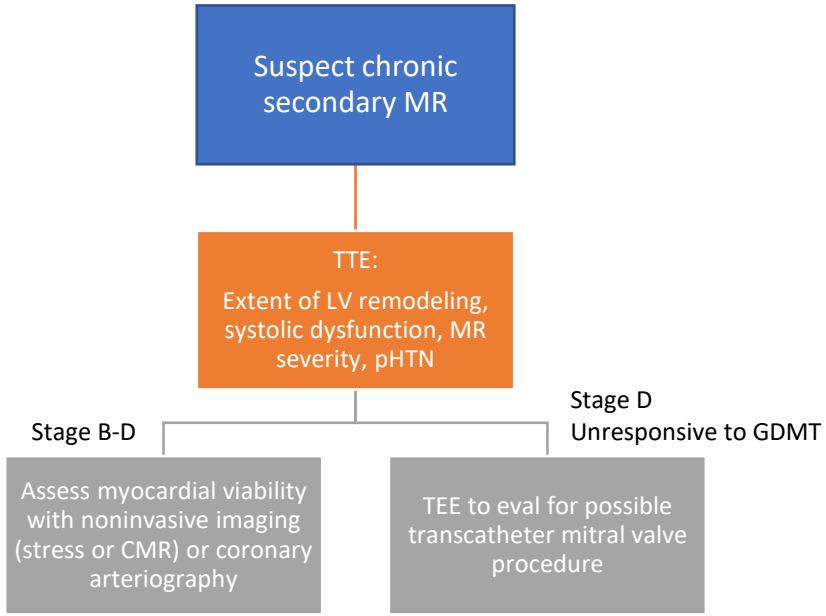
Symptoms	
Primary symptoms	<u>Acute MR</u> • Pulmonary edema (left heart cannot adapt to increased preload) • Cardiogenic shock (poor forward flow)
	<u>Chronic MR</u> • Initially asymptomatic • Gradually progressive fatigue, orthopnea, DOE, PND
Abnormal leaflets	<u>Anterior</u> regurgitant jet heard at spine
	<u>Posterior</u> regurgitant jet heard parasternally
Murmur	• Apical, holosystolic • Radiates to axilla and back • MVP mid-systolic click with mid or late apical systolic murmur • Increases with afterload (ex. handgrip) or LV volume (ex. CHF) • Laterally displaced PMI with left parasternal systolic lift secondary to increased LA volume/size

Mitral Regurgitation Diagnostics

Chronic Primary Mitral Regurgitation Workup



Chronic Secondary Mitral Regurgitation Workup



Mitral Regurgitation Diagnostics

	Morphology	Hemodynamics	Associated Changes
Stage A At Risk	Mild MVP Thickening of valve Restriction of leaflets	Possible central jet less than 20% of LA Vena contracta (smallest stream diameter) <0.3 cm	No associated changes at this stage
Stage B Progressive	Severe prolapse Coaptation normal Prior IE Rheumatic changes: restriction and no coaptation	Jet is 20-40% LA Vena contracta <0.7 cm Regurgitant volume and fraction <60 mL and 50% ERO<0.4 cm	LA enlargement
Stage C Severe Asymptomatic	MVP with flail leaflet vs loss coaptation Rheumatic changes as above Prior IE as in stage B Thickening from radiation	Jet >40% LA or holosystolic nature Vena contracta >0.7 cm Regurgitant volume and fraction >60 mL and >50% respectively ERO>0.4 cm	Begin to see LV enlargement & LA enlargement Pulmonary HTN Differentiate C1 (from C2) by EF >60% and LVESD<40mm
Stage D Severe Symptomatic	As in stage C, with symptoms of dyspnea on exertion and decreased capacity for exercise.		

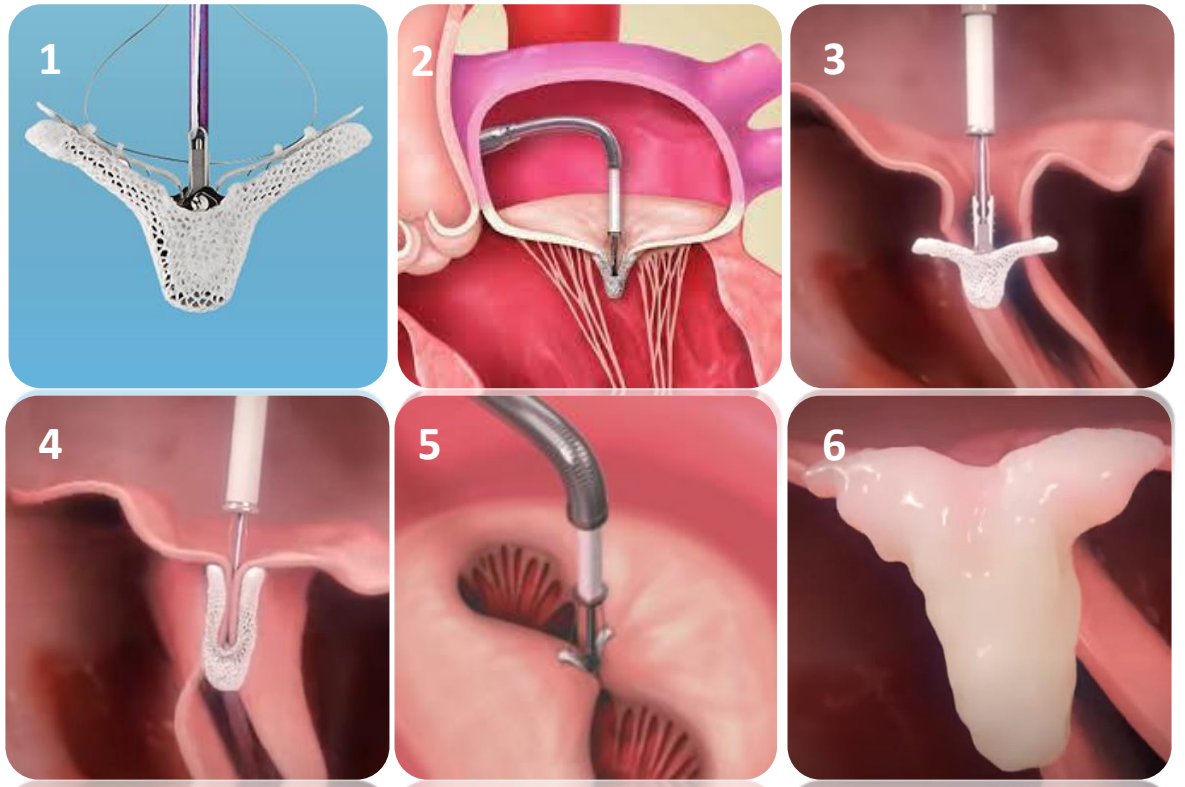
Mitral Regurgitation Treatment

Treatment	
Acute mitral regurgitation	<u>Medical therapy</u> • Vasodilator (allowing forward flow) if blood pressure can tolerate • Intra-aortic balloon pump to improve hemodynamics until surgery
	<u>Surgical therapy</u> • Immediate for if severe • Repair specifically indicated over replacement in case of chordae tendineae rupture
Chronic primary mitral regurgitation	• Treat underlying etiology (E.g. Abx for IE) • If HFrEF: Treat medically with GDMT • Stage C2/D: Mitral valve intervention recommended (repair preferred over replacement) • Stage C1, repair considered if preserved EF & 95% repair success, <1% mortality rate, valve center OR progressive LV dilation or EF decrease in 3+ serial images • Stage D NYHAII/IV: Consider TEER
Chronic secondary mitral regurgitation	• HFrEF and MR: Treat medically with GDMT • Chronic severe MR meeting indication for resynchronization therapy should undergo this intervention • Stage B and other cardiac surgery: Consider repair • Stage C and D undergoing CABG or AVR: MV surgery • NYHA III/IV and Stage D: Consider surgery • NYHA II-IV with LVEF between 20-50%, LVESD <70 mm, and pulm artery pressure <70 mmHg: Consider TEER

Monitoring	
How?	TTE to determine LV function and pulmonary artery pressure
Which conditions?	• Severe primary MR, Stage B-C1, every 6-12 months • Any MR presenting with new or changing symptoms

Transcatheter Edge-to-Edge Repair (MitraClip)

What is TEER?	
How does it work?	Mimics the surgical Alfieri stitch procedure for mitral valve prolapse (involves suturing cusps A2 and P2 on the MV)
Types	• MitraClip (Abbott Cardiovascular) is currently the only TEER therapy approved in the US • Other therapies such as PASCAL (Edwards Lifesciences) are under investigation
COAPT Trial (2018)	• Established role for TEER in chronic severe secondary MR • Improved all-cause mortality and HF hospitalization with MitraClip compared to GDMT alone



TEER (MitraClip) Procedure

1. The TEER (e.g. MitraClip) device
2. Transcatheter delivery from the femoral vein to the left atrium via transseptal puncture from the right atrium
3. Positioning of the clip along the A2-P2 cusps of the MV
4. Grasping A2 and P2
5. Interior view prior to retraction of the catheter
6. Epithelialization of the clip over time

Transcatheter Edge-to-Edge Repair (MitraClip)

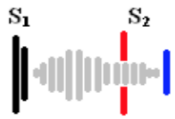
TEER Indications	
Chronic severe secondary MR	<u>Meet Class 2a recommendations</u> • VEF 20-50% • NYHA II, III, or IV symptoms despite optimal GDMT • PASP < 70 mmHg and LVESD < 70 mm
Chronic severe primary MR	<u>Meet Class 2a recommendations</u> • NYHA III or IV symptoms • Prohibitive surgical risk • Life expectancy > 1 yr

Contraindications
• Active MV endocarditis • Rheumatic MV disease • Current intracardiac, IVC, or femoral thrombus • Inability to tolerate post-procedural DAPT (aspirin, Plavix) • Prohibitive MV anatomy on TEE

Post-TEER Complications and Considerations	
Worsened outcomes	Presence of afib, pHTN (>50 mmHg), RV failure
Post-op complications	Iatrogenic ASD, renal failure, clip detachment
Post-TEER mitral stenosis	If multiple clips are required to correct MR
Effect on ejection fraction	Typically “unmasks” a worse EF by decreasing the ability of a weak LV to pump blood retrograde into the lower-pressure LA

Pulmonary Stenosis

Etiology
• Congenital (MCC) • Carcinoid • Tumor • Vegetation

Physical Exam	
Murmur 	• Increases with inspiration • Mid-systolic • LUSB • crescendo-decrescendo

Diagnostics (for severe disease)	
Anatomy	• Thickened leaflets with domed appearance in systole • Restricted excursion may be immobile
Hemodynamic changes	• Vmax > 4 m/s • Peak gradient > 64 • Mean gradient >35 mmHg
Associated changes	• RV or RA enlargement • RV hypertrophy

Treatment	
Mild	No medical therapy required
Moderate	Treat heart failure with diuretics Consider surgical intervention*
Severe	Treat heart failure with diuretics Consider surgical intervention*
*balloon valvuloplasty, valvotomy, surgical replacement	

Follow up in severe disease	
EKG TTE	Stage C or D: annual
16-minute walk test/CPET	Stage C: biennial Stage D: annual
Cardiologist	Stage C: 6-12 months Stage D: 3-6 months

Pulmonary Regurgitation

Etiology	
Primary	Congenital Endocarditis Carcinoid Syndrome
Annular enlargement	Marfan syndrome Pulmonary hypertension

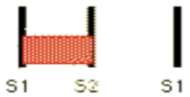
Physical Exam	
Murmur 	• Increases with inspiration • Diastolic • Decrescendo • High pitched (Graham Steell) • LSUB • Palpable P2 if pHTN

Diagnostics (for severe disease)	
Anatomy	• Distorted leaflets (can be absent altogether) • Annular dilation
Hemodynamic changes	• Filling of RVOT with jet on color doppler imaging • Jet density and contour with steep slope of deceleration
Associated changes	• RV enlargement • Paradoxical septal motion

Treatment	
Primary	<u>Asymptomatic</u> Treatment not recommended regardless of severity <u>Symptomatic</u> Consider pulmonary valve replacement
Secondary	Treat underlying disease

Tricuspid Stenosis

Etiology
• Rheumatic Heart Disease • Congenital Heart Disease

Physical Exam	
Concomitant rheumatic MS	Assess for TS (Difficult to assess at bedside)
Findings	Venous congestion (ascites, edema)
Murmur  	Diastolic murmur Worsens with inspiration

Criteria for severe disease	
Anatomy	• Leaflets with calcification, distortion, and thickening
Hemodynamic changes	• Pressure half-time (time from max pressure to half max pressure) is 190 ms or more • Area of valve<1cm^2 • Mean pressure gradient of >5mmHg
Associated changes	• RA enlargement • Tricuspid regurgitation • Left-sided disease (multiple valves common in rheumatic disease)

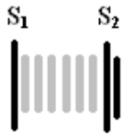
Treatment	
Medical	• Loop diuretics (Improves hepatic and systemic congestion) • Treatment of concomitant left-sided heart disease (MS/MR/AS)
Surgical (for severe TS)	• If undergoing left-sided valve intervention: repair recommended unless severe damage requiring replacement • If symptomatic: Repair over commissurotomy (usually simultaneous TR exists)

Tricuspid Regurgitation

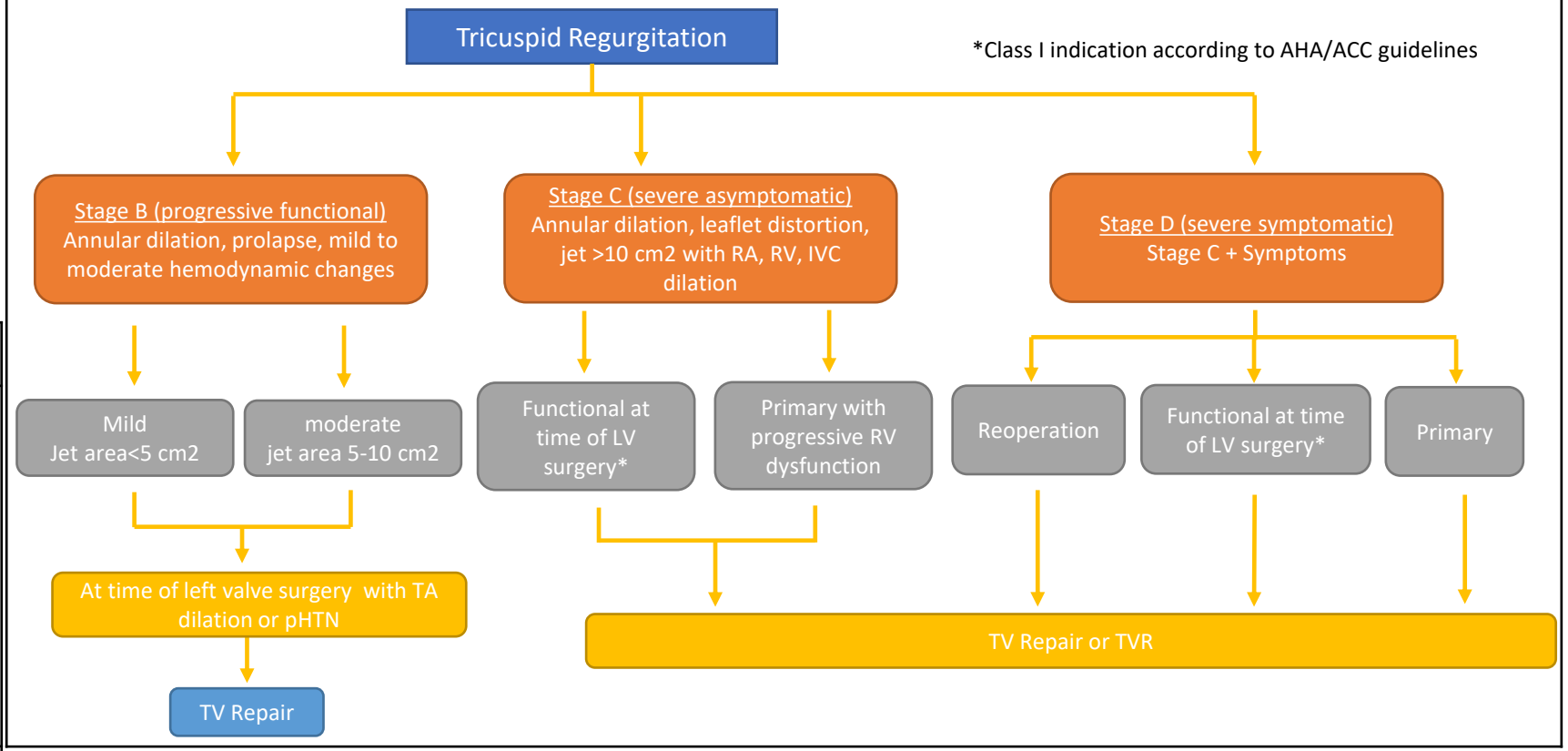
Etiology

- Left heart disease (functional)
  - Rheumatic disease
- Leaflet impingement due to pacemaker leads

Physical Exam

Major symptoms	• Symptoms of left-sided overload when due to chronic remodeling • Dyspnea • Fatigue • Abdominal bloating • Anorexia • Rales • Ascites • Edema
Murmur 	• Holosystolic murmur • LLSB • Blowing quality • Increases during inspiration, decreases with expiration or straining

Diagnostics



Treatment

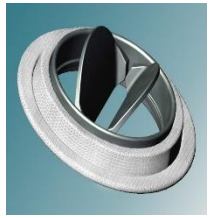
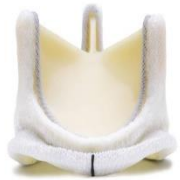
- Diuretics in stage C and D with signs of right-sided heart failure
  - Treat secondary causes including pHTN, a-fib, and HFrEF

Anticoagulation for Prosthetic Valves

Anticoagulation and Valves	
Which valves need AC?	• All mechanical prosthetic valves need warfarin
Valves with high risk of thrombosis	• Mechanical mitral valves > mechanical aortic valves due to lower flow rate
Types of valves	• Surgically placed valves are mechanical or bioprosthetic (bovine or porcine tissue) • All TAVRs are bioprosthetic • Mechanical valves last longer than bioprosthetic valves but require lifelong AC. Better for younger pts (↓ risk of bleed)

Anticoagulation Bridging	
Minor surgical procedures	Continue warfarin at goal
Bileaflet or On-X mechanical AVR and no embolic RFs	Temporarily hold AC without bridging
Other patients	Bridge with heparin gtt

Embolic Risk Factors
• Atrial fibrillation • Prior VTE • LV dysfunction • Hypercoagulable state

Types of valves	
	Ball in cage valve (Discontinued and no longer in use)
	Bileaflet mechanical valve (Most common type of mechanical valve, accounting for ~80% of all prosthetic valves)
	On-X valve
	Surgical bioprosthetic valve

Anticoagulation and Antiplatelet Goals for Prosthetic Valves

Type of valve	Goal	Class Recommendation
1. Old generation mechanical AVR (e.g. ball-in-cage) 2. Any mechanical AVR with embolic risk factors 3. Any mechanical MVR	Warfarin (goal INR 3.0)	Class 1a
	Aspirin 81mg daily	Class 2b
Bileaflet mechanical AVR	Warfarin (goal INR 2.5)	Class 1a
	Aspirin 81mg daily	Class 2b
On-X mechanical AVR	Warfarin (goal INR 2.5) for 3 months	Class 1a
	Warfarin (goal INR 1.5-2.0) after 3 months if also on aspirin	Class 2b
	Aspirin 81mg daily	Class 2b
Bioprosthetic surgical AVR Bioprosthetic MVR	Warfarin (goal INR 2.5) for 3-6 months	Class 2a
	Aspirin 81mg daily	Class 2a
TAVR valve	Aspirin 81mg daily	Class 2a
	DAPT (aspirin, Plavix) for 3-6 months OR warfarin (goal INR 2.5) for at least 3 months	Class 2b

Infective Endocarditis: Duke Criteria

Definition of Terms Used in the Modified Duke Criteria for Diagnosis of Infective Endocarditis

Major Criteria

Blood culture findings positive for IE

Typical microorganisms consistent with IE from two separate blood cultures:

- *Viridans streptococci, Streptococcus gallolyticus* (formerly known as *S. bovis*), *Staphylococcus aureus,* HACEK group, or
- Community-acquired enterococci, in the absence of a primary focus, or

Microorganisms consistent with IE from persistently positive blood culture findings, defined as:

- ≥2 positive culture findings of blood samples drawn >12 hr apart, or
- 3 or most of ≥4 separate culture findings of blood (with first and last sample drawn ≥1 hr apart)
- Single positive blood culture for *Coxiella burnetii* or anti–phase I IgG titer ≥1:800

Evidence of endocardial involvement

Echocardiographic findings positive for IE (TEE recommended in patients with prosthetic valves, rated at least possible IE by clinical criteria or complicated IE [paravalvular abscess]; TTE as first test in other patients), defined as follows:

- Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation, or
- Abscess, or
- New partial dehiscence of prosthetic valve

New valvular regurgitation; worsening or changing of preexisting murmur not sufficient

Minor Criteria

- Predisposition, predisposing heart condition, or intravenous drug use
- Fever—temperature >38°C
- Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway lesions
- Immunologic phenomena: glomerulonephritis, Osler nodes, Roth spots, and rheumatoid factor
- Microbiologic evidence: positive blood culture finding but does not meet a major criterion as noted above (excludes single positive culture findings for coagulase-negative staphylococci and organisms that do not cause endocarditis) or serologic evidence of active infection with organism consistent with IE

Duke Criteria Diagnosis	
Definitive IE (either or)	• 2 major • 1 major + 3 minor • 5 minor
Possible IE (either or)	• 1 major + 1 minor • 3 minor

Prosthetic valve IE Timing

<u>After Placement:</u>

- Early: < 2 months
- Intermediate: 2-12 months
- Late: > 12 months

Infective Endocarditis: Diagnosis

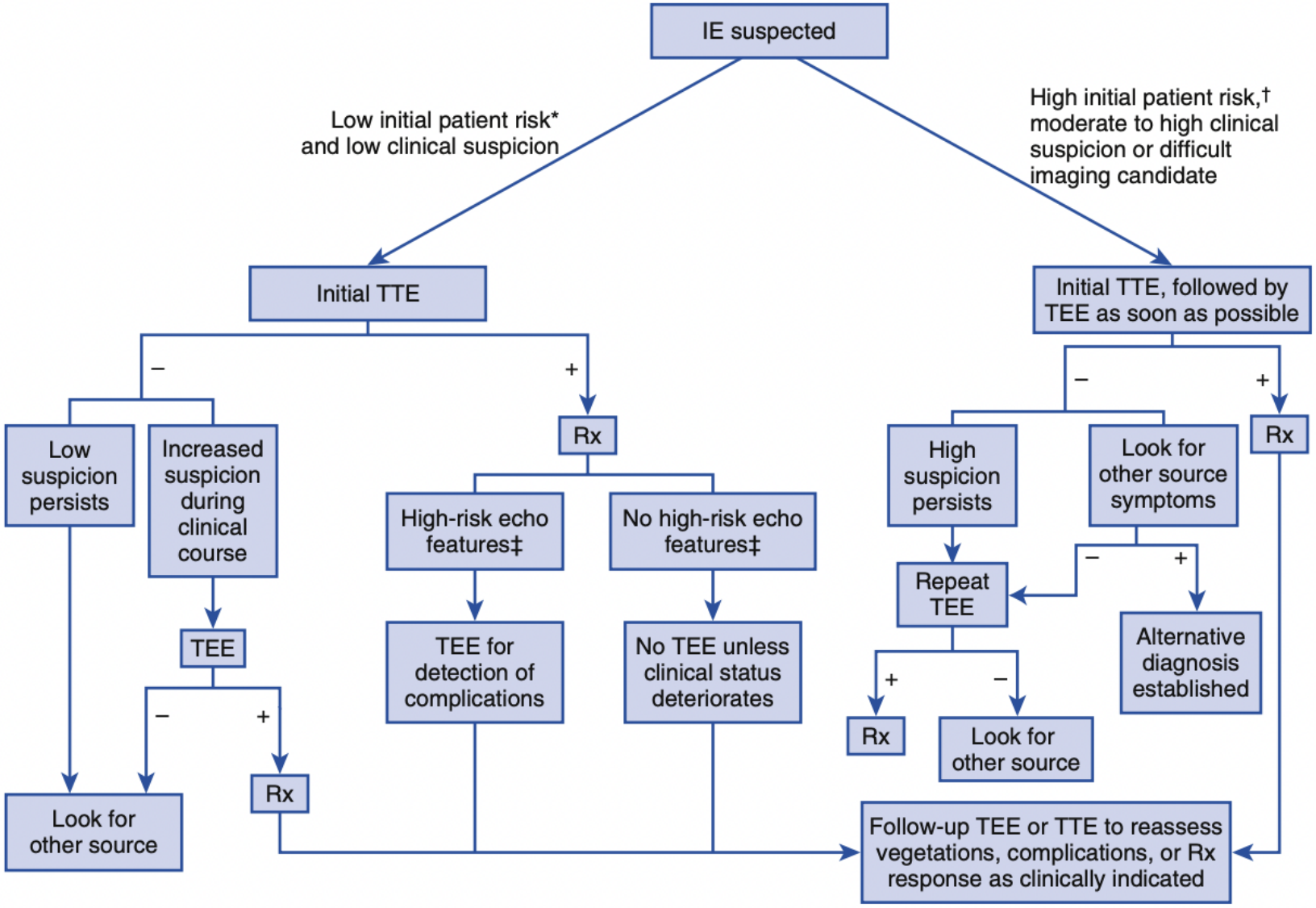
Predisposing factors	
Intrinsic	• Valvular heart disease (MR due to MVP most common, bicuspid AV) • congenital heart disease • skin/dental infections
Extrinsic	• IV Drug Use (Tricuspid valve - Right Side IE) • Hemodialysis (HD) • IV Catheter • CIED

Diagnostics	
Blood cultures	• ≥3 sets within first 24 hours (at least 1 hr apart) • Negative culture more often due to antibiotic administration prior to cultures being drawn than culture negative organisms (e.g. HACEK)
EKG	• AV block or bundle branch block raises suspicion for paravalvular abscess
Echo	• TEE more sensitive than TTE for both native and prosthetic valve IE • TTE: 1st test in most patients → perform TEE if TTE is non-diagnostic and IE is still suspected • TEE: Perform 1st if 1) Prosthetic Valve endocarditis 2) CIED infection 3) Complicated IE (e.g. Paravalvular abscess)

Clinical Features	
Local	• CHF (DOE, PND, orthopnea) • Perivalvular extension w/ conduction abnormality (e.g. High grade AV block, bundle branch block)
Bacteremic/ Infectious	• Fever (95%) • Constitutional symptoms (40%) such as chills, myalgia, fatigue, anorexia, weight loss • Back pain (vertebral seeding) • Septic arthritis
Embolic	• MI (rare) • Stroke and seizure • Abdominal pain (visceral embolism/splenic abscess) • Multi-focal consolidation/pleural effusion (right sided IE)
Immune mediated	• Glomerulonephritis

Physical Exam	
Local	New regurgitant murmur, worsening murmur, CHF (JVD, S3, B/L rales)
Embolic	Splinter hemorrhages, Janeway lesions (painless erythematous macules on palms/soles), petechiae
Immune mediated	Osler nodes (painful erythematous nodules on pulp of fingers and toes), Roth spots (retinal hemorrhage with pale center), splenomegaly

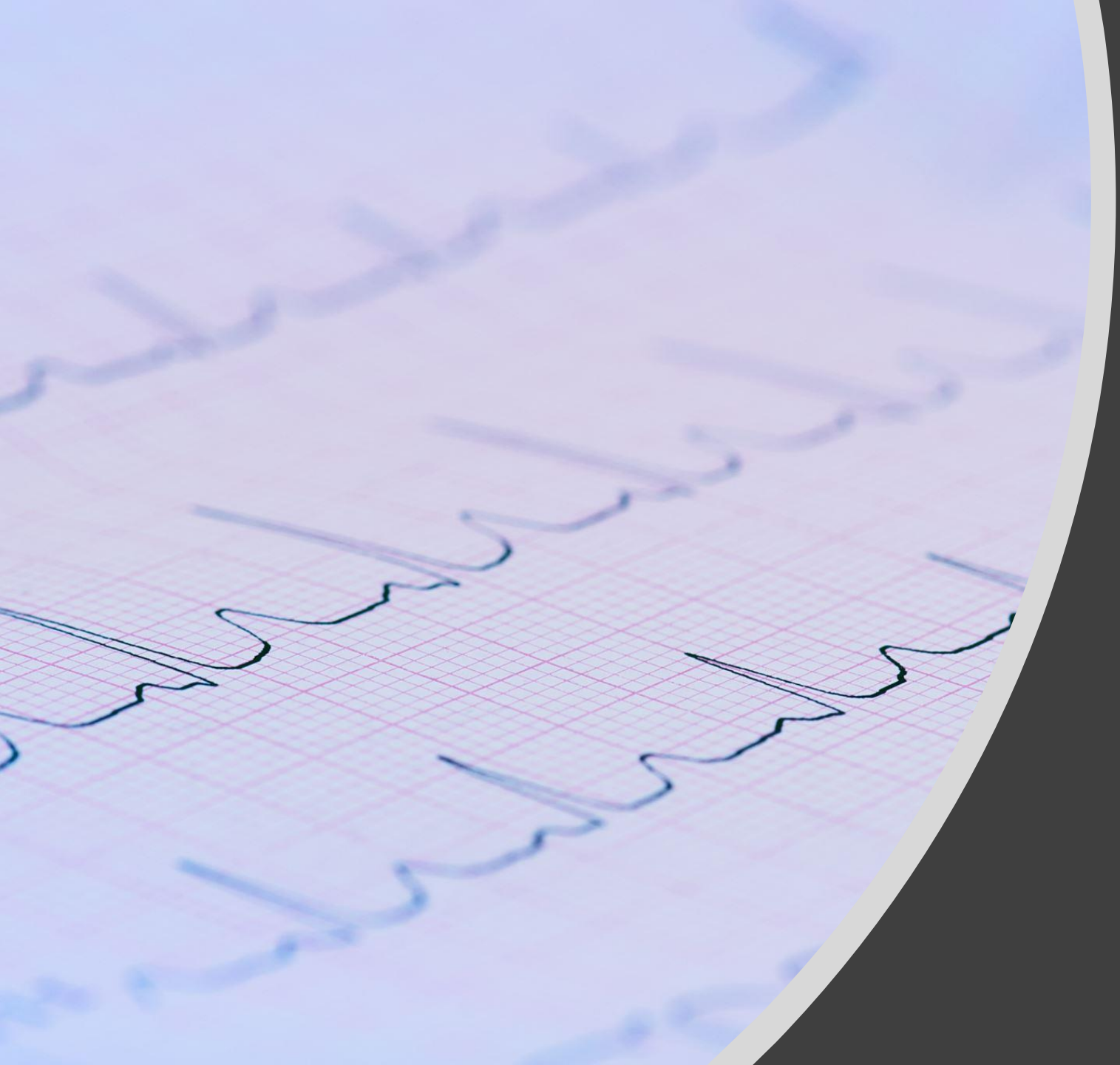
Infective Endocarditis: Diagnostic Algorithm



Infective Endocarditis: Treatment

Organism	Native Valve	Prosthetic Valve
Streptococcus	IV Penicillin (4 wk)	IV Penicillin (6 wk) + Gentamicin (2 wk)
MRSA	Vancomycin (6 wk)	Vancomycin (6 wk) + Rifampin (6 wk) + IV Gentamicin (2 wk)
MSSA	IV Oxacillin (6 wk)	IV Oxacillin (6 wk) + Rifampin (6 wk) + IV Gentamicin (2 wk)
Enterococcus	Based on Sensitivity	Based on Sensitivity
See *UPMC Guide to Antimicrobial Chemotherapy* for detailed antibiotic choice and duration (pg 50-54). No RCTs to support initiation of antiplatelet or anticoagulation to reduce embolic events		

Indications for Surgical Interventions		
L sided native valve	R sided native valve	Prosthetic valve IE
• Stenosis or regurgitation resulting in CHF • IE caused by fungi or resistant organisms • Complicated by CHB, abscess, or penetrating lesions • Antibiotic failure: fever, enlarging vegetation, emboli >5-7d post treatment start • Mobile vegetations > 10mm in diameter • Heart failure, hemodynamic instability	• RV failure • Antibiotic failure: fever, enlarging vegetation, emboli >5-7d post treatment start • Tricuspid vegetation > 20mm with recurrent PE • If IVDU, consider risk of relapse/infection	• Same as native valves • Valvular dehiscence • Intracardiac fistula • Severe prosthetic valvular dysfunction • Relapsing IE



Heart Failure

Section Editor:
Greg Olenginski, MD

Heart Failure Classification

NYHA Functional Classes*	
Class I	Without limitations of physical activity. Ordinary physical activity does not result in symptoms
Class II	Slight limitation of physical activity. Patients are not comfortable at rest and ordinary activity results in symptoms
Class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms.
Class IV	Inability to carry on any physical activity without symptoms. Symptoms may be present at rest and if any physical activity if undertaken.
*Quantifies the degree of **functional limitation** imposed by HF and is determined by degree of effort needed to illicit symptoms	

ACC/AHA Stages*	
Stage A	At risk for heart failure without structural heart disease or symptoms.
Stage B	Structural heart disease but without signs or symptoms. Includes NYHA functional class I with no prior or current symptoms of heart failure.
Stage C	Structural heart disease with prior or current symptoms of heart failure. Includes patients in any NYHA functional class (including class I with prior symptoms).
Stage D	Refractory heart failure requiring specialized interventions. Includes patients in NYA functional Class IV with refractory heart failure.
*Describes the progressive nature of HF and defines the therapeutic approach for each stage	

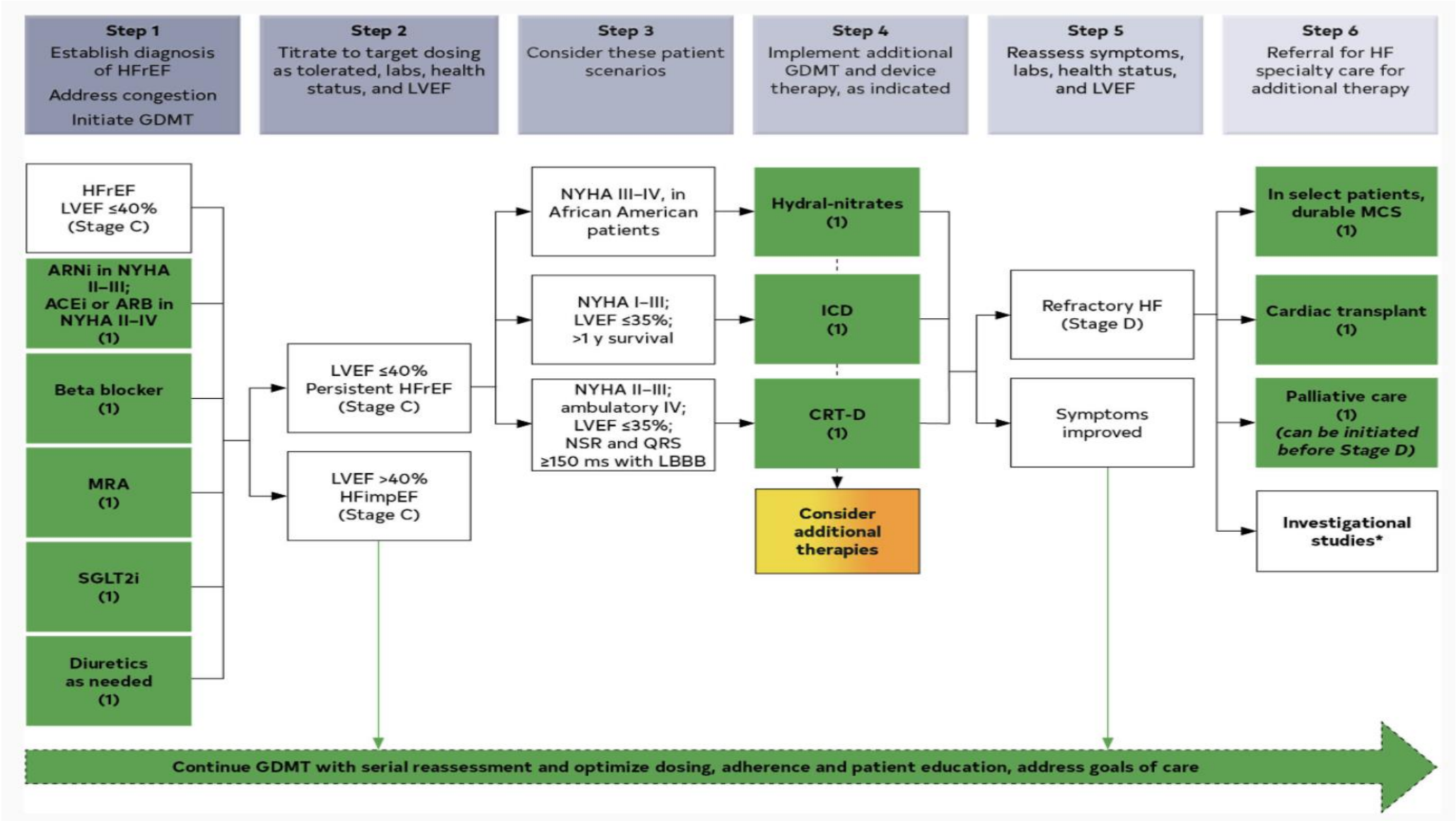
Heart Failure Categories

Classification	EF (%)	Description
HFrEF	≤40	<u>Systolic HF</u> Most common causes include ischemic, HTN, dilated cardiomyopathies, valvular disease
HFpEF	≥50	<u>Diastolic HF</u> • Heterogeneous clinical entity • Most common causes include ischemic (more microvascular dysfunction), HTN, hypertrophic obstructive cardiomyopathy, restrictive cardiomyopathies
HFmrEF	41-49	• Borderline/intermediate group • Characteristics with clinical profile and outcomes similar to HFpEF

Heart Failure Diagnostics

Work-up	
History	Screen for risk factors for CAD (HLD, HTN, DM, FHx), stroke, cancer, recent pregnancy, cirrhosis, and exposures (EtOH, radiation, anthracyclines)
Physical exam	Quality of S1/S2, S3 gallop, displaced PMI, JVD, signs of R-sided overload, pulmonary edema, abdominal distension, nausea, vomiting, ascites, and dependent edema
EKG changes	ST changes, Q waves, LVH, limb lead voltage, QRS duration (bundle branch blocks or conduction delays)
Echocardiogram	Define phenotype and narrows differential (e.g. regional wall motion abnormalities, LVH, valvular pathology)
Stress testing	Evaluate reversible ischemia vs. infarction; exercise tolerance
Coronary angiography	Evaluate for obstructive CAD and occult coronary disease
RHC	Evaluate intra-cardiac filling pressures, cardiac output/index, mixed venous O2 saturation
Cardiac MR	Evaluate for infiltrative disease, late gadolinium enhancement (LGE) in MI, HCM, ARVCM, sarcoid, amyloid, hemochromatosis, and myocarditis
Endomyocardial biopsy	When amyloid or myocarditis is suspected; not routinely indicated
PYP SPECT	High sensitivity for ATTR amyloid; insensitive for AL. AA amyloid rarely involves the heart
Secondary tests	HIV, iron studies, ANA/ENA, viral serologies, SPEP, genetic testing, thiamine/carnitine/selenium levels, TSH, free T4

HFrEF Management



Guideline Directed Medical Therapy (GDMT) for HFrEF

Drug Class	Drug	Clinical Trial	Conclusions
RAAS inhibitors (e.g. ACEi/ARB/ARNI)	1. ARNI: sacubitril-valsartan 2. ACEi: enalapril 3. ARB: candesartan, losartan, valsartan	1. PARADIGM-HF, PIONEER-HF (sacubitril-valsartan vs. enalapril) 2. CONSENSUS, SOLVD (enalapril vs. SOC) 3. Val-HeFT[5] (valsartan vs. SOC), CHARM (candesartan vs. SOC +/- ACEi), ELITE II (losartan vs. captopril)	• PARADIGM-HF: reduction in CV mortality or HF hospitalization (NNT 21) • PIONEER-HF: reduction in NT-proBNP at 4-8 weeks • CONSENSUS: reduction in 6-month all-cause mortality (NNT 6) • SOLVD: significant reduction in 4-year all-cause mortality • Val-heFT; no effect on mortality; reduction in combined end-point • CHARM: no effect on mortality; reduction in CV death and HF hospitalization • ELITE II: no effect on mortality; better tolerated than captopril
Beta-blockers	1. Bisoprolol 2. Metoprolol-succinate 3. Carvedilol	1. CIBIS-II (bisoprolol vs. placebo) 2. MERIT-HF (metoprolol vs. placebo) 3. COMET (carvedilol vs. metoprolol), COPERNICUS (carvedilol vs. placebo)	• CIBIS-II: reduction in all-cause mortality • MERIT-HF: reduction in all-cause mortality and hospitalization • COMET: reduction in all-cause mortality • COPERNICUS: reduction in annual mortality (NNT 15)
Mineralocorticoid receptor antagonist	1. Spironolactone 2. Eplerenone	1. RALES (spironolactone vs. placebo) 2. EPHESUS (eplerenone vs. placebo in post-MI LV dysfunction), EMPHASIS-HF (eplerenone vs. placebo in NYHA II)	• RALES: reduction in all-cause mortality • EPHESUS: reduction in all-cause mortality and CV mortality or hospitalization for CV event • EMPHASIS-HF: reduction in all-cause mortality or HF hospitalization
SGLT-2 Inhibitor	1. Dapagliflozin 2. Empagliflozin	1. DAPA-HF (dapagliflozin vs. SOC) 2. EMPEROR-Reduced (empagliflozin vs. placebo)	• DAPA-HF: reduction in worsening HF (hospitalization or IV therapy for HF) or CV mortality • EMPEROR-Reduced: reduction in CV death or HF hospitalization
I_f channel inhibitor	1. Ivabradine	1. SHIFT (Ivabradine vs. SOC)	• SHIFT: reduction in HF death or hospitalization
Hydralazine + Isosorbide-dinitrate		1. A-HeFT (Hydral + iso-dinitrate vs. SOC in black patients)	• A-HeFT: stopped early due to reduction in survival and hospitalizations

HFpEF Management

Class Recommendation	Treatment
Class I	Diuretics (as needed)
Class 2a	SGLT2 inhibitors
Class 2b	ACE inhibitors ARBs ARNIs MRAs
Class 3	Avoid routine use of nitrates and PDE-5 inhibitors

Other	Treatment
Management of comorbidities	ASCVD risk factors HTN Afib Obesity Anemia DM CKD OSA

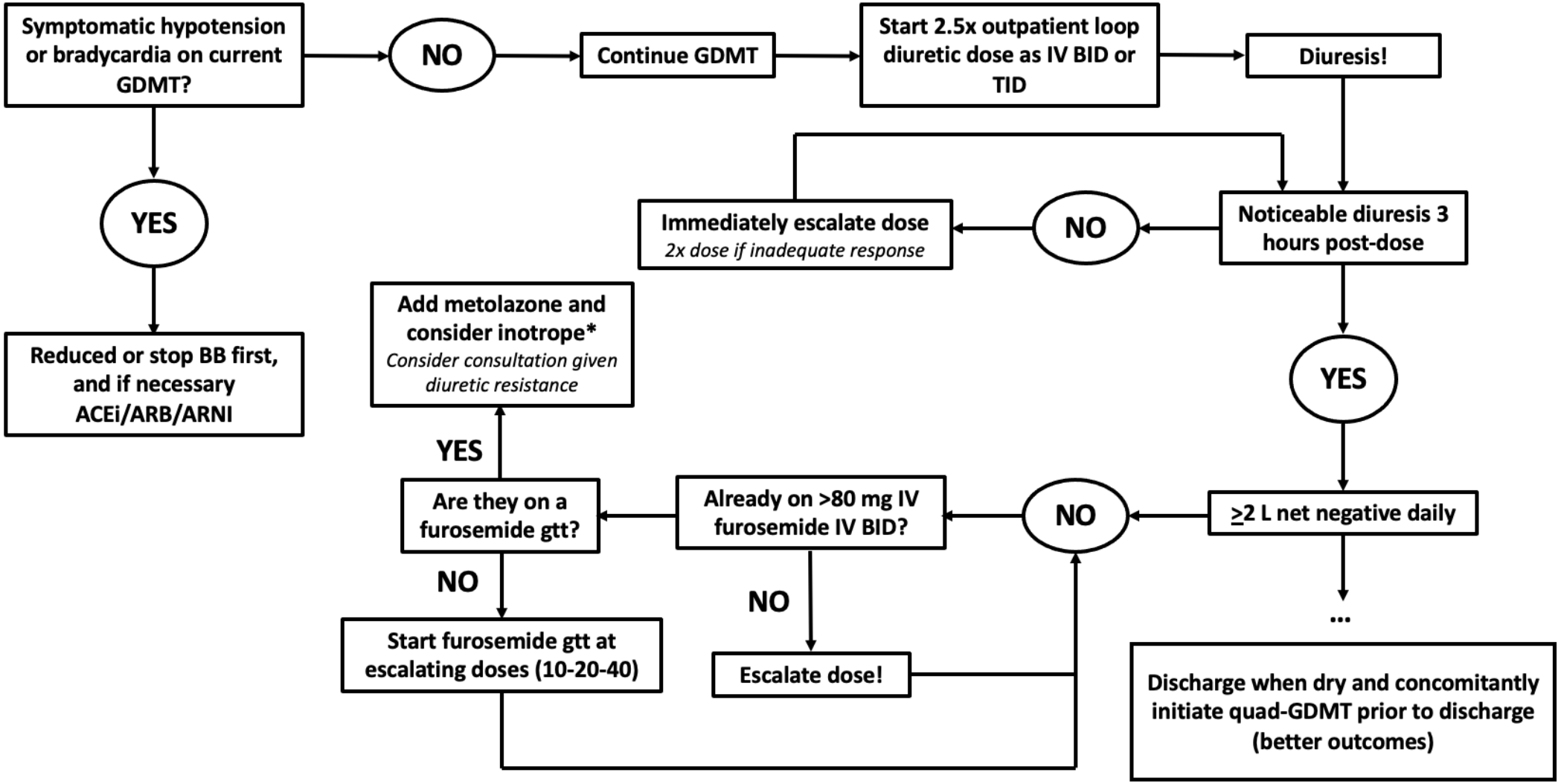
Acute Decompensated Heart Failure Symptomatology

Precipitants (**FAILURE** mnemonic)
• **F**orgetting medication (or taking beta-blockers, NSAIDs, methamphetamine, or cocaine) • **A**rrhythmia or **A**nemia • **I**schemia or **I**nfarction • **L**ifestyle choices – dietary indiscretions, medication noncompliance • **U**pregulation of cardiac demand from either pregnancy or hyperthyroidism • **R**enal failure from the progression of kidney disease or insufficient dialysis • **E**mbolus – pulmonary embolism • **S**tenosis from worsening renal artery stenosis, aortic stenosis, or other valvular diseases

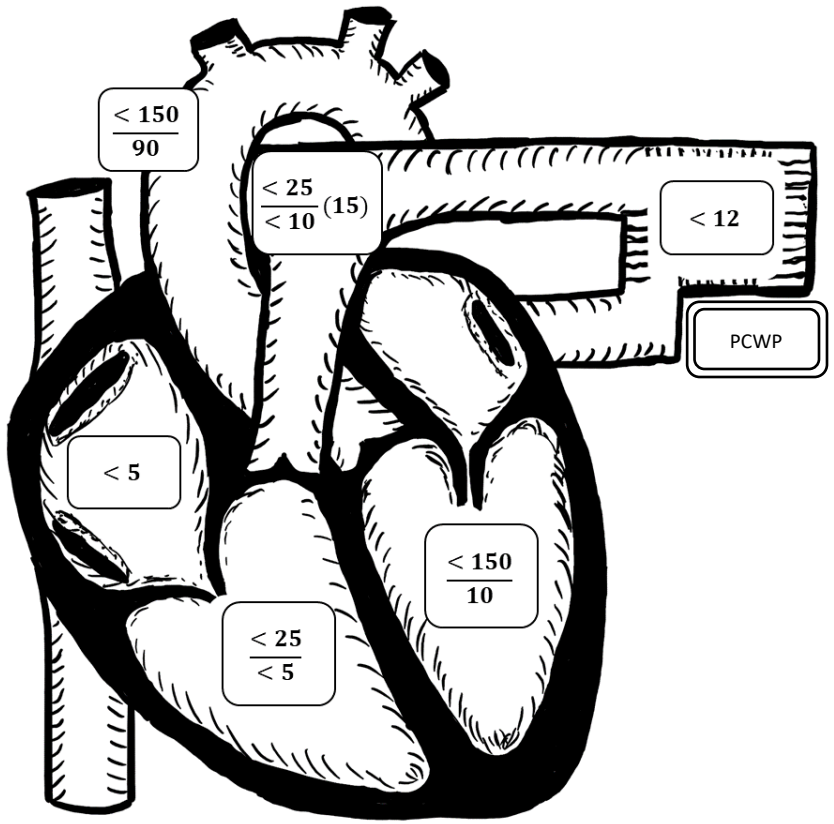
Clinical symptoms
• **Profile**: volume status and perfusion (e.g. dry/wet, cold/warm) • PE evidence of peripheral edema • Take note of the patient's admission weight and weight at discharge

Laboratory evidence
• **Elevated filling pressures:** JVP >8 cm, +S3, displaced PMI, hepato-jugular reflux (HJR) • Elevated sCr and hyponatremia (markers of advanced illness) • Higher BNPs correlate with increased risk of mortality and recurrent hospitalization (no utility in trending) • RHC when clinical picture is ambiguous or not responding to initial therapy (e.g. IV diuretics)

ADHF Management



Right Heart Catheterization (RHC)



Management
- Heart ***transplant*** evaluation
- ***Peri-operative*** management for cardiac surgery
- To guide ***pharmacologic/hemodynamic*** therapy in shock
- To guide ***fluid*** management in shock

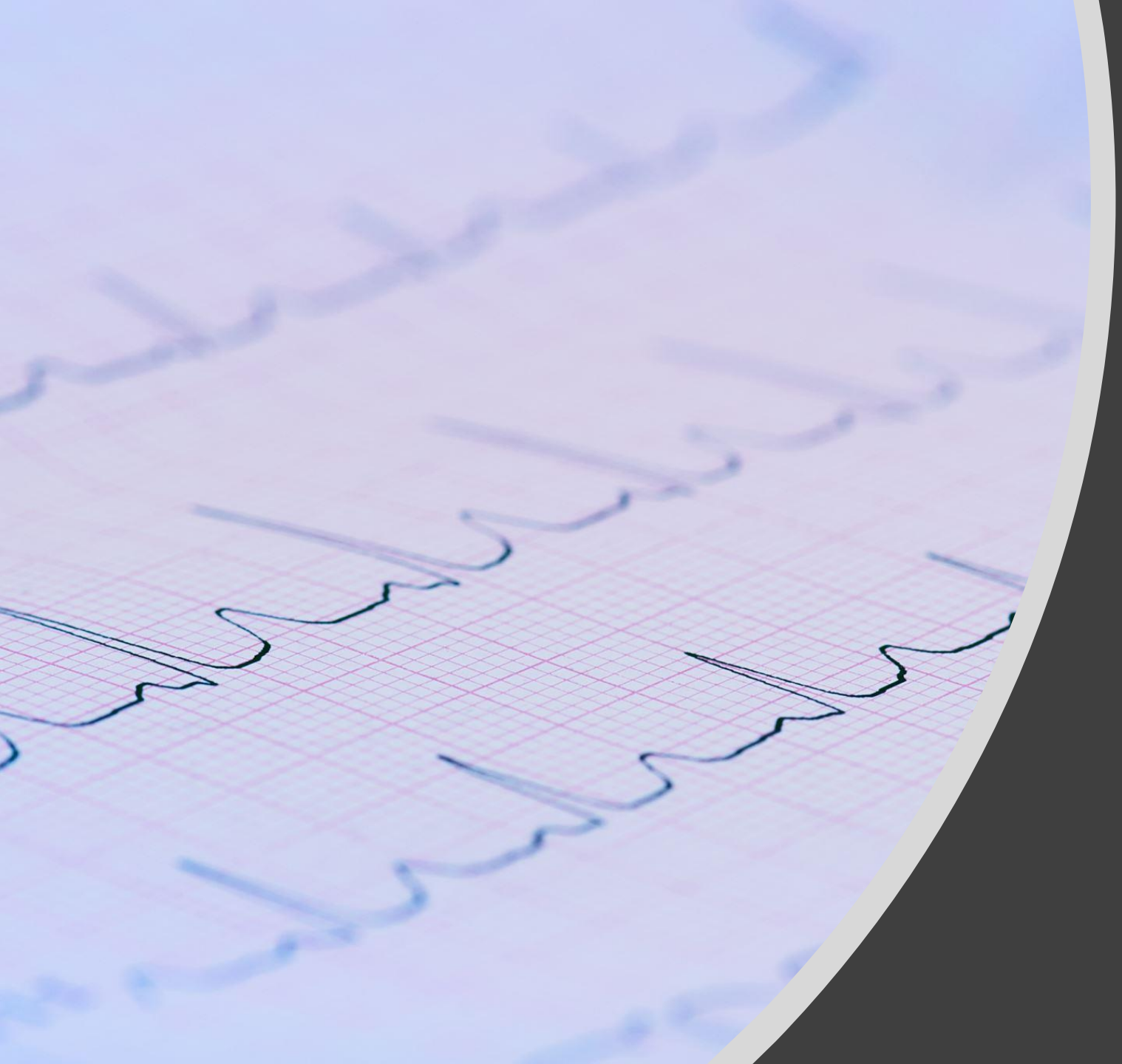
Common Indications	
Diagnosis	Management
• Evaluation of ***pulmonary hypertension*** (PH) • Differentiate etiologies of ***shock*** • Differentiate etiologies of ***pulmonary edema*** • Diagnosis of left-to-right intra-cardiac ***shunt***	• Heart ***transplant*** evaluation • ***Peri-operative*** management for cardiac surgery • To guide ***pharmacologic/hemodynamic*** therapy in shock • To guide ***fluid*** management in shock

Pressures (mmHg)			
Central Venous Pressure (CVP)	3 – 12	**Cardiac Output (CO)**	4.0 – 8.0 (l/m)
Right Atrium (RA)	0 – 5	**Cardiac Index (CI)**	2.8 – 4.2 (L/m/m^2)
Right Ventricle (RV)	15 – 25/0 – 10	**Transpulmonary Gradient (TPG)**	≤ 12 (mmHg)
Pulmonary Artery (PA) (mean)	15 – 25/5 – 15 **(<25)**	**SVR**	< 15 (WU); or < 1200 (dynes/s/cm^5)
Pulmonary Capillary Wedge Pressure (PCWP)	≤ 12	**PVR**	≤ 3 (WU); or ≤ 240 (dynes/s/cm^5)
Mixed Venous Oxygenation (SvO2)*	65 – 80%	*Measured in Wood Units (WU). CCU will often convert to dynes/sec/cm^5. To calculate, multiply result by '80'	

Using Right Heart Catheterization Data

Useful CCU Equations		
Maximal Oxygen Consumption (VO_2)	$VO_2 = BSA * (161 - (Age * 0.54))$	Used to calculate cardiac output
Fick Cardiac Output (CO_f)	$CO_f = \frac{VO_2}{13.4 * Hgb\left(\left(\frac{SaO2}{100}\right) - \left(\frac{PAsat}{100}\right)\right)}$	***Shock*** – correlates with overall function of heart and ability to deliver oxygen to end-organs
Fick Cardiac Index (CO_i)	$CI_f = \frac{CO_f}{BSA}$	
Cardiac Power Output (CPO)	$CPO = \frac{MAP * CO_f}{451}$	***Shock –*** hydraulic energy from LV; correlates with in-hospital mortality for <u>*cardiogenic*</u> shock (CPO < 0.6)
Systemic Vascular Resistance (SVR)*	$SVR = \frac{MAP - RA}{CO_f}$	***Shock*** – helps differentiate between types of shock
Pulmonary Arterial Pressure Index (PAPi)	$PAPi = \frac{PAPs - PAPd}{CVP}$	***Shock*** – helps predicts RV failure (PAPi <u>≤</u> 0.9)
Transpulmonary Gradient (TPG)	$TPG = mPAP - PCWP$	***pHTN*** – differentiates between subtypes of pulmonary hypertension - 1 Wood Unit = 80 dynes/sec/cm^5 - Normal PVR – 0.125-1.5 (WU)
Pulmonary Vascular Resistance (PVR)*	$PVR = \frac{mPAP - PCWP}{CO_f} = \frac{TPG}{CO_f}$	

*Measured in Wood Units (WU). CCU will often convert to dynes/sec/cm^5. To calculate, multiply result by '80'



Pulmonary Hypertension

Section Editor:
Ben Zuchelkowski, MD

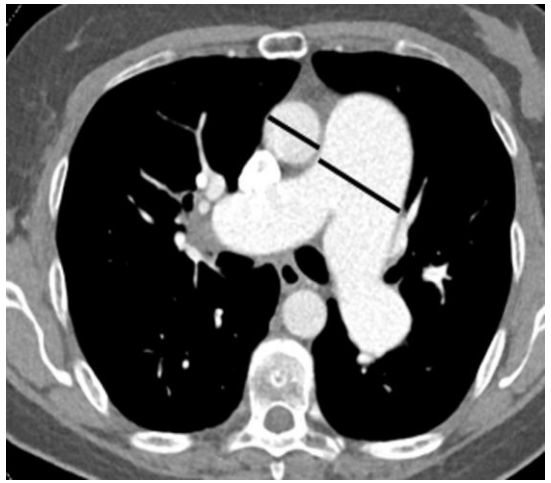
WHO Classes of Pulmonary Hypertension

Group		Examples
I	**Pulmonary arterial hypertension (PAH), pre-capillary PH**	• Idiopathic/Heritable: BMPR2, ALK1, etc. • Drugs/toxins: appetite suppressants, dasatinib (+ other chemotherapeutics), St. John's wort, cocaine, interferon • CTD: systemic sclerosis, Raynaud's, SLE, RA • CHD: septal defects, Eisenmenger syndrome • HIV • Porto-pulmonary hypertension • Schistosomiasis (especially in those with hepatosplenic involvement) • Pulmonary veno-occlusive disease-associated PH/pulmonary capillary hemangiomatosis • Persistent pulmonary hypertension of the newborn
II	**Left heart disease, post-capillary PH**	HFpEF, HFrEF, valvular heart disease, impaired inflow/outflow (restrictive CM, constrictive pericarditis, congenital)
III	**Intrinsic lung disease, post-capillary PH**	Obstructive & restrictive lung disease, mixed patterns, hypoxia without lung disease, developmental lung disorders
IV	**PH due to pulmonary artery obstructions**	• CTEPH • Other PA obstructions: sarcoma, congenital pulmonary artery stenosis, hydatidosis, arteritis without connective tissue disease, malignant and non-malignant tumors
V	**Multifactorial, unclear etiologies, and miscellaneous**	• Hematologic: chronic hemolytic anemia (sickle cell, beta-thalassemia, spherocytosis), myeloproliferative disorders • Systemic/Metabolic: Sarcoidosis, Pulmonary Langerhans cell histiocytosis, Gaucher disease, glycogen storge disease, neurofibromatosis • Other: chronic renal failure with or without hemodialysis, fibrosing mediastinitis

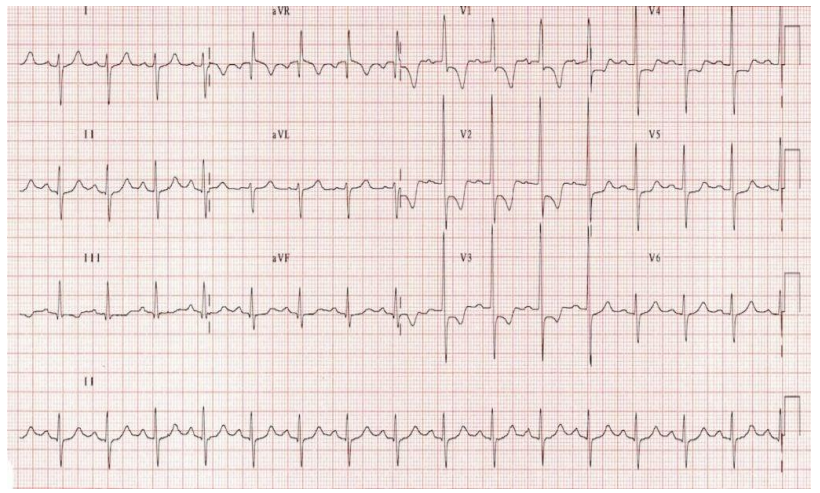
Pulmonary Hypertension Symptomatology

Physical Exam	
RV failure symptoms	• JVD hepatomegaly • Pulsatile/tender liver • Peripheral edema • Ascites • Pleural effusion
Murmur	• Pronounced pulmonary component of S2 <u>Right-sided auscultatory findings</u> • Right-sided S3/S4 • Wide-splitting S2 • Holosystolic murmur of TR • Diastolic PR murmur

CT findings: main pulmonary artery to ascending aorta ratio ≥ 1 suggests the diagnosis

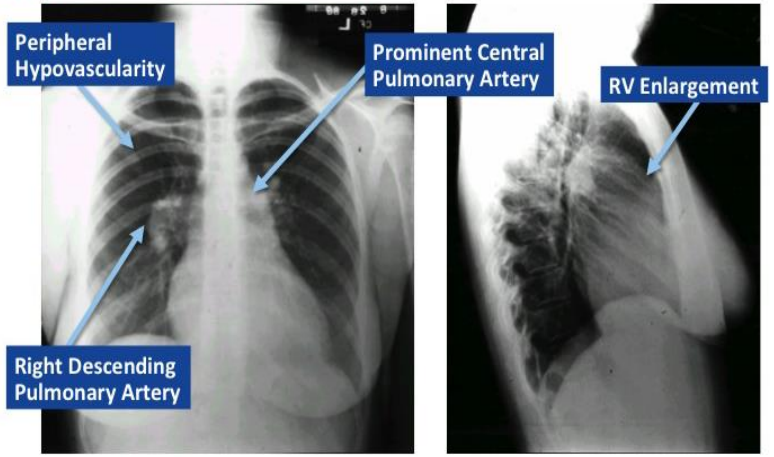


EKG with RV strain pattern due to RVH



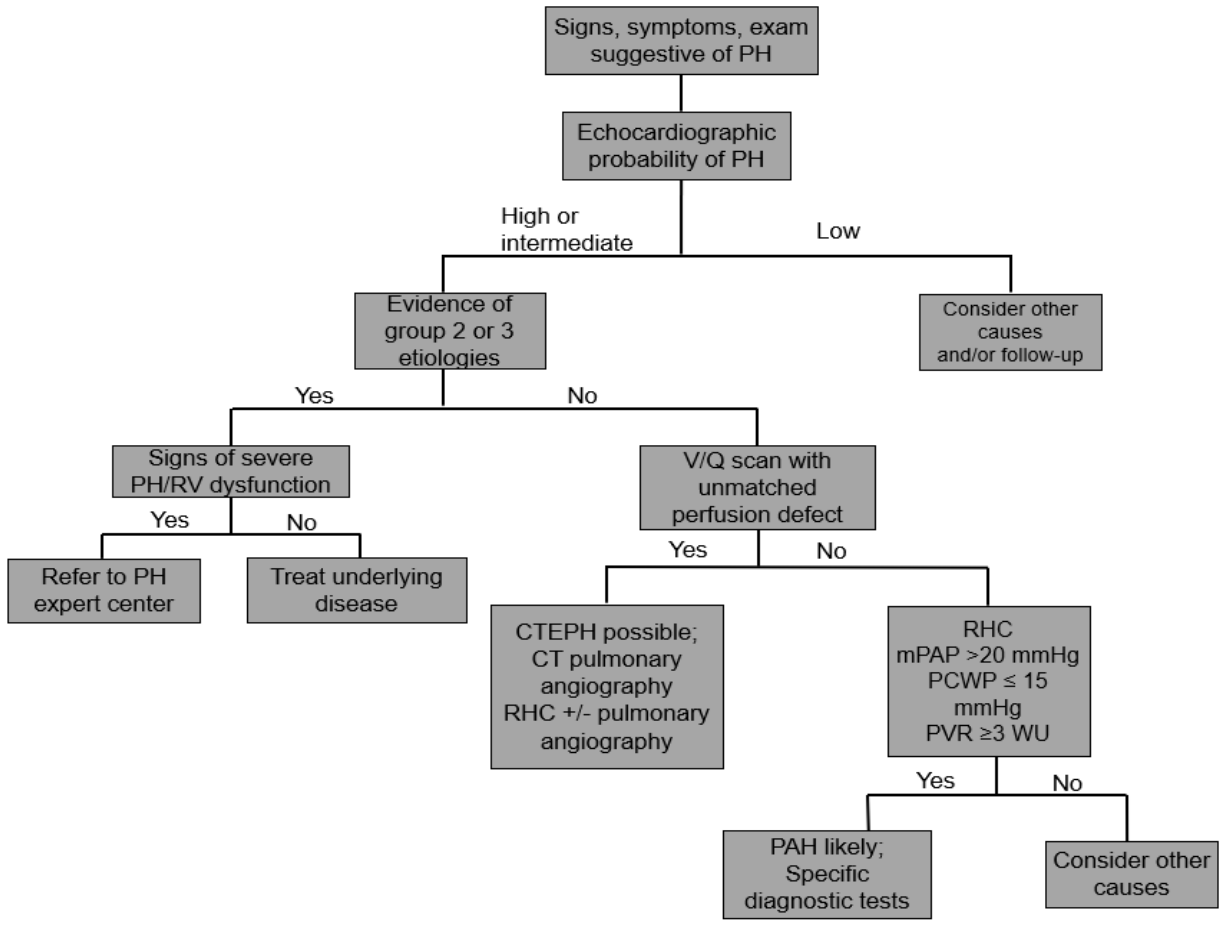
Symptoms	
Most common	• Exertional dyspnea • Fatigue • Symptoms of RV failure develop as PH progresses
Less common	• Cough • Hemoptysis • Hoarseness (Ortner's syndrome due to impingement of the left recurrent laryngeal nerve by a dilated main PA)

Figure 1. Characteristic X-Ray of a Patient with PAH



Pulmonary Hypertension Diagnostics

Diagnostic algorithm



Definition: mean pulmonary artery pressure (PAP) > 20 mmHg (gold standard as measure via RHC when euvolemic)

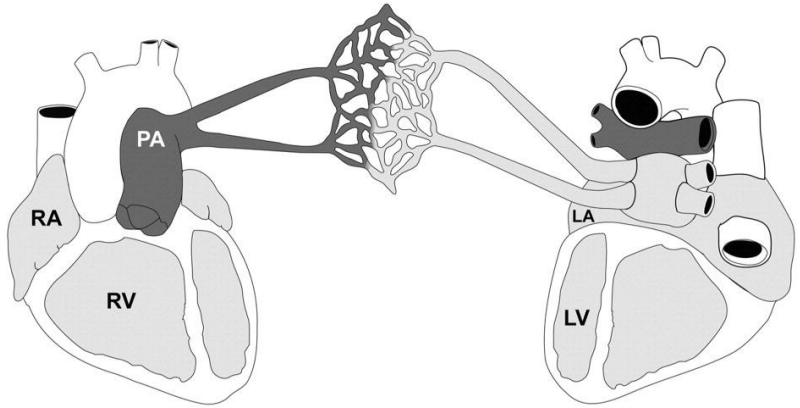
Diagnostic Schema	
Initial work-up	Echocardiogram, 6MWT, NYHA functional class, ECG, PFTs with DLCO, CXR, nocturnal oximetry, CBC w/ plt, LFTs, and TSH
Differentiating types of PH	"Look at the company it keeps." Combine history, exam, and echocardiographic findings to predict etiology.
CTEPH suspected?	V/Q is the gold standard in ruling out CTEPH (NPV ~100%). V/Q scan suggestive of CTEPH would show an unmatched perfusion defect.
PAH risk factors	HIV, schistosomiasis, CHD, portopulmonary HTN, CTD (SLE, scleroderma, CREST syndrome, MCTD), heritable, and drugs/toxins (fenfluramine, maternal SSRI use, amphetamines, cocaine, alkylating chemotherapies)
PAH suspected?	Check HIV and hepatitis serologies, schistosomiasis serologies, CTD panel, and screen for drugs and family hx

Pulmonary Hypertension Diagnostics

Echocardiographic probability of pulmonary hypertension		
Peak TRV (m/s)	**PH signs?**	**Echo probability**
≤ 2.8†	No	Low
≤ 2.8†	Yes	Intermediate
2.9 – 3.4	No	
2.9 – 3.4	Yes	High
> 3.4	N/A	
† denotes "or not measurable"		

Echocardiographic signs to assess probability of pulmonary hypertension		
Ventricles	**Pulmonary artery (PA)**	**IVC and RA**
RV/LV basal diameter ratio >1.0	RV outflow Doppler acceleration time < 105 ms and/or midsystolic notching	IVC diameter >21 mm with ↓ inspiratory collapse
Flattening of the interventricular septum	Early diastolic pulmonary regurgitation velocity >2.2 m/s PA diameter > 25 mm	RA area (end-systole) >18 cm^2

Pulmonary Hypertension Pre- vs Post- Capillary



Pre-capillary PH

WHO groups 1, 3, 4, 5

Post-capillary PH

WHO groups 2, 5

Transpulmonary gradient (TPG) = mPAP - PCWP

$$\text{PVR (WU)} = \frac{\text{TPG}}{\text{CO}} \times 80 \text{ dynes} \cdot \text{sec} \cdot \text{cm}^{-5}$$

Definitions		
	PCWP ≥ 15 mmHg	**PCWP < 15 mmHg**
PVR ≥ 3 WU	Combined pre- and post-capillary PH	Isolated pre-capillary PH
PVR < 3 WU	Isolated post-capillary PH	Consider high-flow state

RHC Interpretation in Pulmonary Hypertension

Pulmonary Hypertension = Mean pulmonary artery pressure (mPAP) > 20 mmHg [by RHC]

PVR vs. PCWP	
PVR	• PVR ≥ 3 WU – suggests ***pre-***capillary PH • PVR < 3 WU – suggests ***post***-capillary PH
PCWP	• PCWP ≥ 15 – suggests ***post***-capillary PH; or (in other words) • suggests high ***left-sided filling pressures***

Distinguishing PAH from PH due to LV Failure			
	1º PH	**LV Failure**	**Mixed**
mPAP (mmHg)	> 20	> 20	> 20
PCWP (mmHg)	< 15	< 15	< 15
PVR (Wood Units)	> 3	> 3	> 3

Distinguishing PAH from PH due to LV Failure		
	PCWP ≥ 15 mmHg	**PCWP < 15 mmHg**
PVR ≥ 3 WU	Combined ***pre-*** & ***post***-capillary PH	Isolated ***pre***-capillary PH
PVR < 3 WU	Isolated ***post***-capillary PH	Suggests a high flow state

RHC Interpretation in Pulmonary Hypertension

1 — Pulmonary arterial hypertension

- Idiopathic PAH
- Heritable PAH
- PAH associated with infection, drugs, toxins, connective tissue disease

Pre-capillary
PAWP ≤ 15 mmHg
PVR ≥ 3 WU

2 — Pulmonary hypertension due to left-sided heart disease

- PH due to HFrEF, HFpEF, valvular heart disease
- Post-capillary PH due to other congenital or acquired CV conditions

Isolated postcapillary
PAWP > 15 mmHg
PVR < 3 WU

Combined pre-/post-capillary
PAWP > 15 mmHg
PVR ≥ 3 WU

3 — Pulmonary hypertension due to lung disease, hypoxia, or both

- Obstructive, Restrictive, Mixed lung disease
- Hypoxia without lung disease
- Developmental lung disorders

Pre-capillary*
PAWP ≤ 15
PVR ≥ 3 WU

**Unless associated with coexisting condition*

4 — Pulmonary hypertension due to pulmonary-artery obstructions

- Chronic thromboembolic PH
- Other pulmonary-artery obstructions

Pre-capillary*
PAWP ≤ 15
PVR ≥ 3 WU

**Unless associated with coexisting condition*

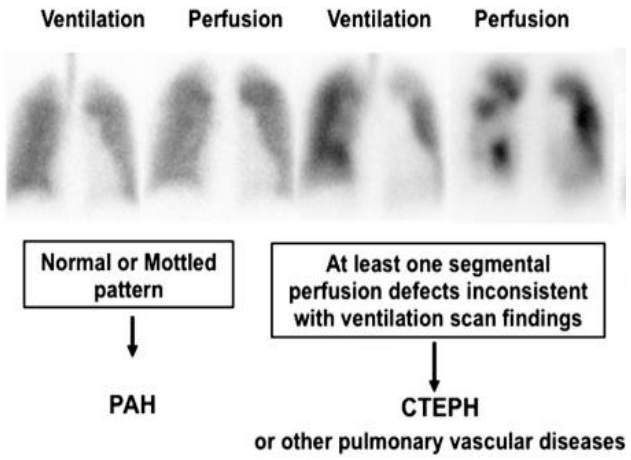
5 — Pulmonary hypertension with multifactorial or unclear mechanisms

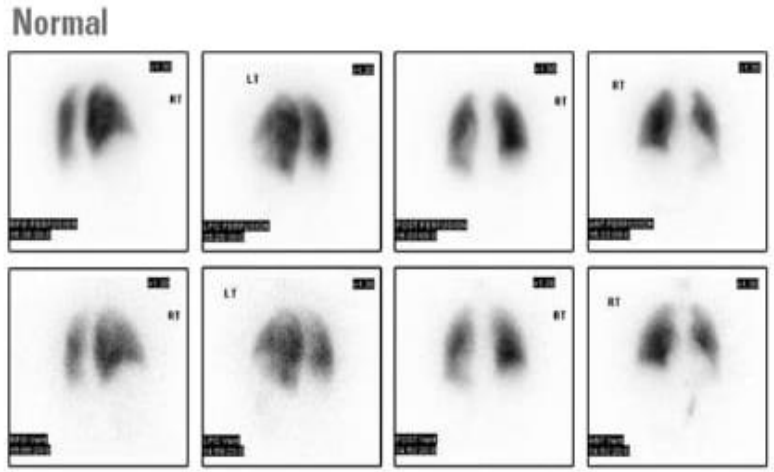
- Hematologic disorders
- Systemic disorders – sarcoidosis, etc.
- Metabolic disorders
- Chronic renal failure

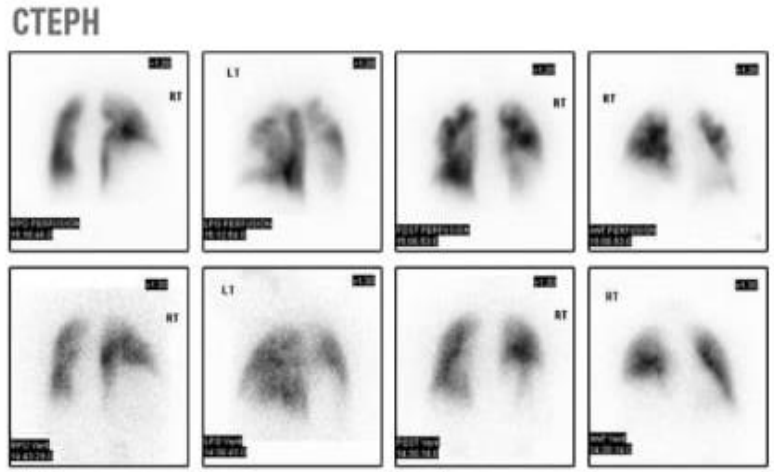
Isolated postcapillary
Isolated postcapillary
Combined pre-/post-capillary

Chronic Thrombotic Pulmonary Hypertension (CTEPH)

Overview of CTEPH	
Definition	PH arising from ≥1 pulmonary emboli
Etiology	Exact mechanism unknown, some experts suggest an underlying hypercoagulable state
Epidemiology	Incidence of ~2.3% among survivors of acute PE
Diagnosis	PH (mPAP >20 mmHg) + persistent perfusion defect(s) despite therapeutic anticoagulation
Evaluation	Assess for PH (Echo, RHC). Detect and quantify degree of vascular occlusion (VQ scan, pulmonary angiography (CT, invasive)
Management	• All patients should receive lifelong anticoagulation (IVC filter if high risk of bleed). Evaluate all patients for suitability for pulmonary artery thromboendarterectomy (PTE), as it is the only definitive therapy • For patients who are not candidates for PTE, consider PH-specific therapy (riociguat in moderate disease, IV epoprostenol in advanced disease), in addition to anticoagulation • If refractory, consider lung transplant







Pulmonary Hypertension Treatment By WHO Class

Treatment Principles by WHO Classification	
Group 1	Pulmonary vasodilators; choice of agents depends on WHO functional class (I-IV) (See next page)
Group 2	Focus on management of comorbidities (HFrEF, valvular disease, etc.)
Group 3	Avoid PAH-directed therapy, unless combined Group I and 3 suspected. Focus on management of comorbidities
Group 4	Lifelong anticoagulation, pulmonary artery thromboendarterectomy, riociguat
Surgical	Atrial septostomy (palliative), lung transplant (definitive)

WHO Functional Classification and Treatment		
Class I*	No limitation of physical activity. Ordinary physical activity does not cause undue symptom burden.	Consider starting single oral agent, low threshold to add second agent
Class II	Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity causes undue symptom burden.	ERA + agent against NO-cGMP pathway (PDE5I)** Ambrisentan + tadalafil (AMBITION trial)
Class III	Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes undue symptom burden.	Same for Class II Consider parenteral or inhaled prostanoid with rapid progression or markers of poor prognosis
Class IV	Inability to carry out any physical activity without symptoms. Signs of right heart failure present. Dyspnea and/or fatigue may even be present at rest. Discomfort is increased by any activity.	Addition of a parenteral prostanoid combination regimen

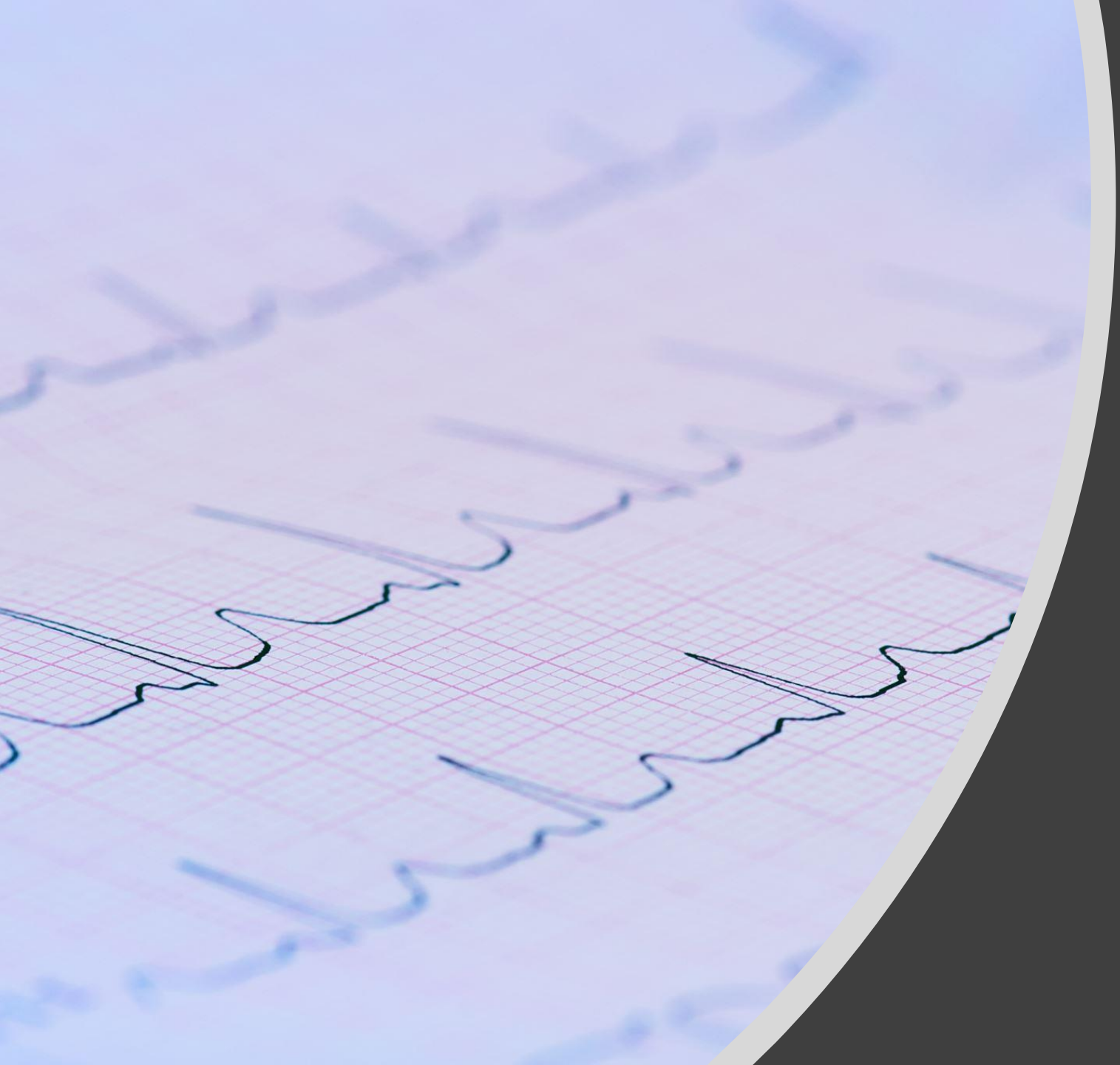
*Patients do not typically present in Class I. Rationale for initiating treatment in Class I is that the disease will progress if left untreated; best to treat early to stave off progression.
**Data exist for specific alternate combinations. Caution against freely choosing agent within each class.

Group I Pulmonary Hypertension Pharmacotherapy

Class	Names	Considerations
Endothelin receptor antagonists*	Bosentan Macitentan Ambrisentan	Considered first-line in combination with PDE5-I for Group 1 (AMBITION trial) unless RV dysfunction
PDE-5 inhibitors*	Sildenafil Tadalafil	Considered first-line in combination with ERA for Group 1 (AMBITION trial) unless RV dysfunction
Prostacyclin analogs*	Epoprostenol: (IV:Flolan/Veletri) Iloprost: (inhaled: Ventavis) Treprostinil (Inhaled: Tyvaso, IV/SC: Remodulin, PO: Orenitram)	Used if RV dysfunction
Guanylate cyclase stimulant	Riociguat	Use in CTEPH (CHEST-1 trial) and Group 1, Class II/III Riociguat may be used in Group 4
IP-receptor agonist*	Selexipag	Can be used as single agent or in combination for Class II/III
CCBs	Nifedipine Diltiazem	Less used now with newer vasodilators available
* All pulmonary vasodilators can cause systemic hypotension, rebound pulmonary hypertension with abrupt withdrawal, worsen V/Q mismatch (unless inhaled). Cautious use in elevated left atrial pressure (can cause pulmonary edema).		

Acute Pulmonary Hypertension Treatment

Management for Decompensated Patients
• Target SpO_2: 88-92% • Optimize RV preload • Ensure parenteral vasodilators infusing at appropriate dose (call specialty pharmacy if unsure) • Vasopressors/inotropes if cardiogenic shock/failure • Avoid intubation (risk of cardiopulmonary collapse due to increased RV afterload with positive pressure) • Ensure back-up IV access in ICU if giving parenteral vasodilators!!



Cardiac Intensive Care and Emergencies

Section Editor:
Amar Patel, MD

Overview of Shock

Type of shock	Preload (PCWP)	Pump function (CO)	Afterload (SVR)	Tissue perfusion (SVO2)
Hypovolemic	←→ (early) or ↓ (late)	←→ (early) or ↓ (late)	↑	> 65% (early) or < 65% (late)
Cardiogenic	↑	↓	↑	< 65%
Distributive (sepsis, anaphylaxis, neurogenic, toxic, adrenal)	←→ (early) or ↓ (late)	↑ or ↓ (late)	↓	> 65%
Obstructive (PE, pHTN, tension PTX)	←→ (early) or ↓ (late)	←→ (early) or ↓ (late)	↑	> 65%
Obstructive (tamponade)	↑	↓	↑	< 65%

Cardiogenic shock

- CI < 2 L/min/m2
- Hypotension (SBP < 90 mmHg or MAP 30 mmHg below baseline)
- Organ hypoperfusion
- Elevated filling pressures (LVEDP > 18 mmHg or RVEDP > 10 mmHg)

Important Formulas		
Parameter	**Equation**	**Normal value**
Fick CO	$CO = VO2/[10 \times (C_aO_2\text{-}C_vO_2)]$	4-8 L/min
Cardiac Index (CI)	CI = CO/BSA	2.5-4 L/min/m^2
SVR	SVR = [(MAP – CVP)/CO] x 80	750-1500 dynes-sec/cm^5
PVR	PVR = [(mPAP – PAOP)/CO] x 80	50-200 dynes-sec/cm^5

Cardiogenic Shock

Shock	
Definition	Cellular and tissue hypoperfusion/hypoxia
Signs and Symptoms	• Hypotension • Vital sign instability • Altered mental status • Cool skin • End organ damage
Lab abnormalities	• Metabolic acidosis • Elevated lactate • Kidney and liver dysfunction
Shock Index $Shock\ Index = \frac{HR}{SBP}$	> 0.8: suggests instability, possible shock

Pearls		
SVR – will see *increase* in SVR in cardiogenic or hypovolemic shock due to compensatory response • *Advanced cardiogenic shock* – SVR will become normal or low	**Skin temperature** can be used as a surrogate of SVR • *Cool skin* – suggests higher SVR • *Warm skin* – suggests lower SVR	**"Cold" profile** – shock with a CI <2.2

RHC Interpretation in Shock

Physiologic variables	Preload		Pump Function		Afterload	Tissue perfusion	
	Pulmonary capillary wedge pressure (PCWP)		Cardiac output (both CO and CI used in CCU)		Systemic vascular resistance (SVR)	Mixed venous oxyhemoglobin saturation (SvO2)	
Type of Shock	*Early*	*Late*	*Early*	*Late*		*Early*	*Late*
Hypovolemic	↔	↓	↔	↓	↑	> 65%	< 65%
Cardiogenic	↑		↓		↑	< 65%	
Distributive	↔	↓	↑	↓	↓	> 65%	
Obstructive							
PE, pHTN, Tension PTX	↔	↓	↔	↓	↑	> 65%	
Pericardial tamponade	↑		↓		↑	< 65%	

DISTRIBUTIVE

- **Septic**
- **Non-Septic**
  - *Inflammatory/SIRS*
  - *Neurogenic*
  - *Anaphylactic*
  - *Burns*
  - *Etc.*

- Fluids
- Vasopressors
- Antibiotics

Cardiogenic

- **Cardiomyopathic**
  - *MI, Myocarditis*
- **Arrhythmogenic**
- **Mechanical**
  - *Valvular, etc.*

- Fluids vs. Diuresis
- Inotropes
- Balloon pumps, etc.

Hypovolemic

- **Hemorrhagic**
  - *Trauma / Bleeding*
  - *GI Bleed, etc.*
- **Non-hemorrhagic**
  - *GI Losses*
  - *Renal Losses*
  - *Etc.*

- Fluids, Blood
- Surgery, intervention
- Underlying cause, etc.

Obstructive

- **Pulmonary Vascular**
  - *Pulmonary Embolus*
  - *Severe pHTN, etc.*
- **Mechanical**
  - *Pneumo-/hemothorax*
  - *Cardiac tamponade*
  - *Pericarditis, etc.*

- PE Response Team
- Pericardiocentesis
- Thoracentesis, etc.

MIXED

- **Endocrine**
- **Metabolic**

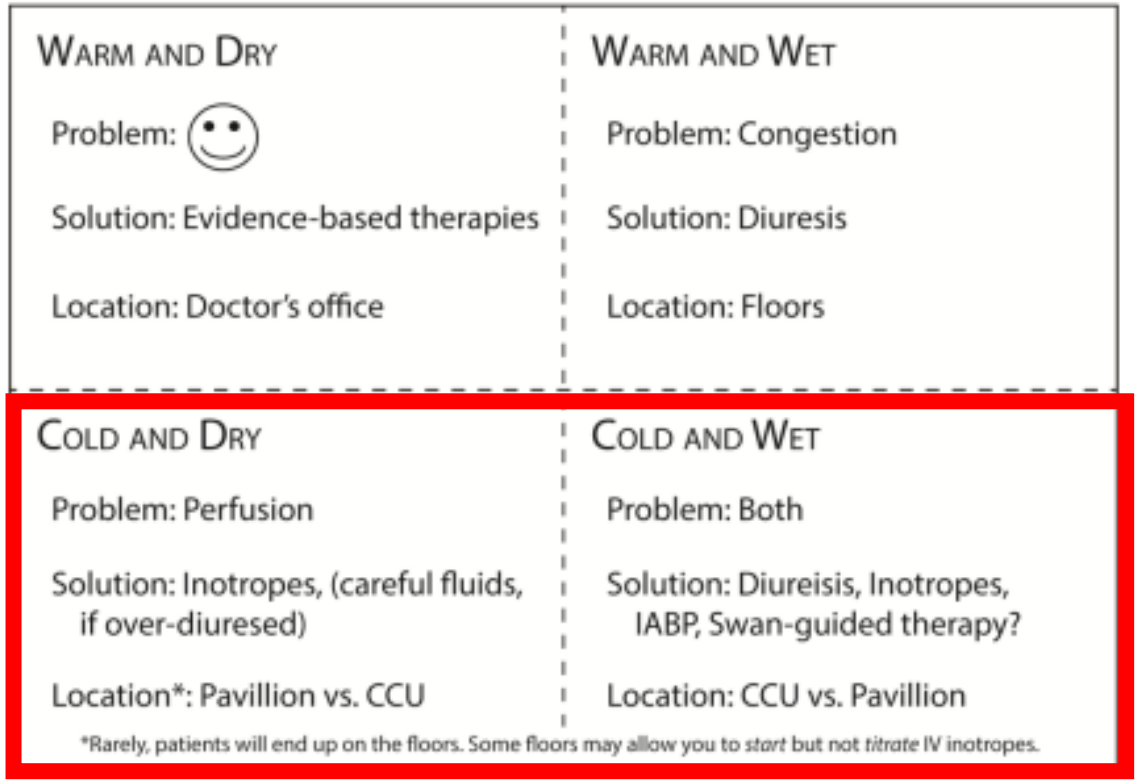
Treat underlying cause

Low-Output Heart Failure

Diagnosis	
Definition	• End-organ malperfusion due to decreased CO/CI • Can't miss diagnosis! If concerned (or not responding initially), empiric inotrope and PA catheter placement
History	• Encephalopathy/drowsy • Abdominal pain/poor appetite • Decreased UOP
Physical exam	• Cool extremities (i.e. shins, forearms) • Volume overloaded
Labs	Elevated ALT/AST, T. bili, lactate, sCr

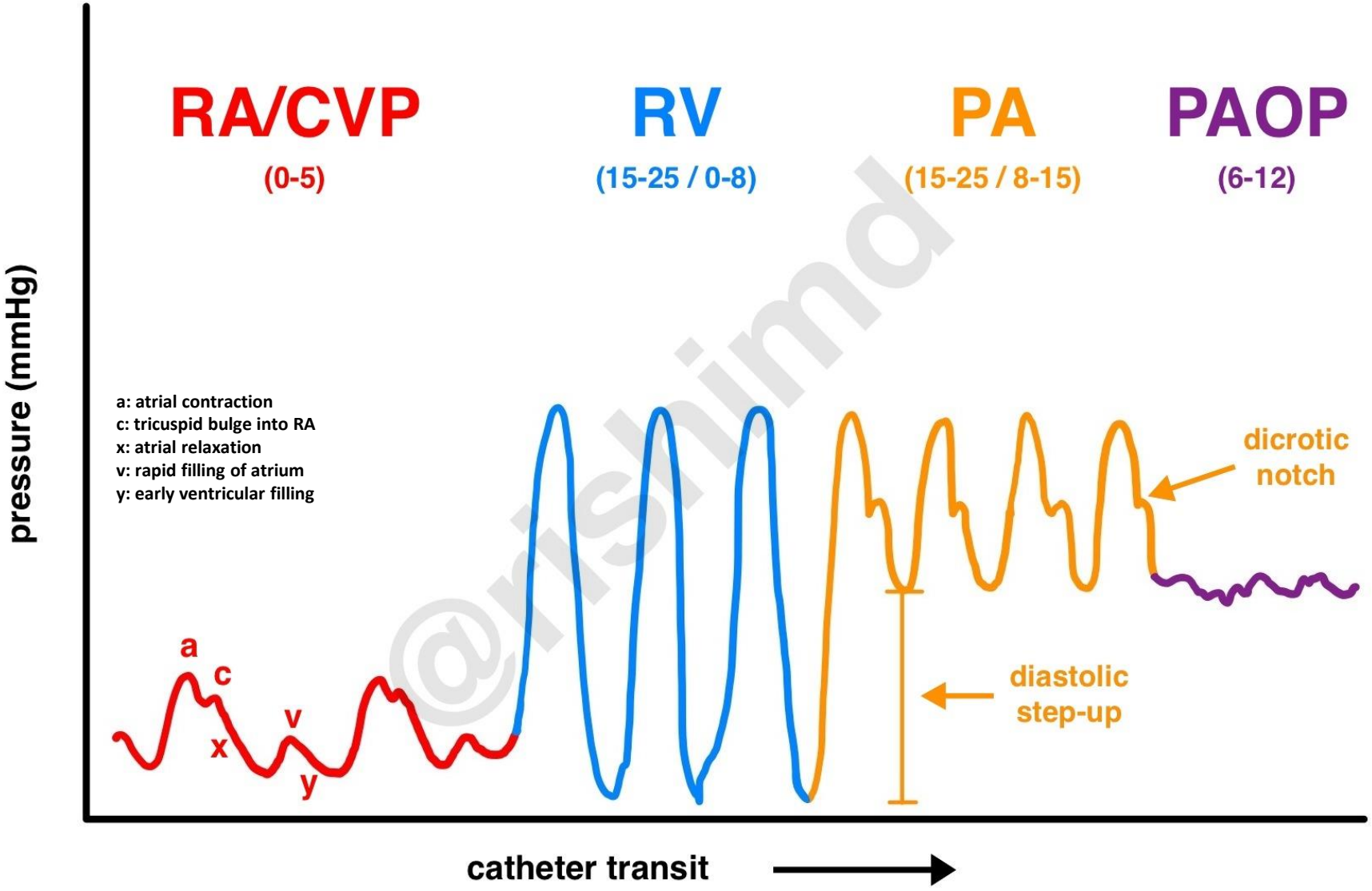
Management	
Optimize preload	IV diuresis or rarely IVF (fill the tank)
Augment contractility	IV inotropes (hold beta-blockers)
Reduce afterload	Nitro gtt, ACE/ARB/ARNI, inotropes are also vasodilatory
Mechanical support	Advanced mechanical support with bridge to LVAD or transplantation

Forrester Classification of Hemodynamic Status (adapted)



JAMA. 2002;287:628–40. N Engl J Med. 1976;295:1356.

Swan-Ganz Waveforms

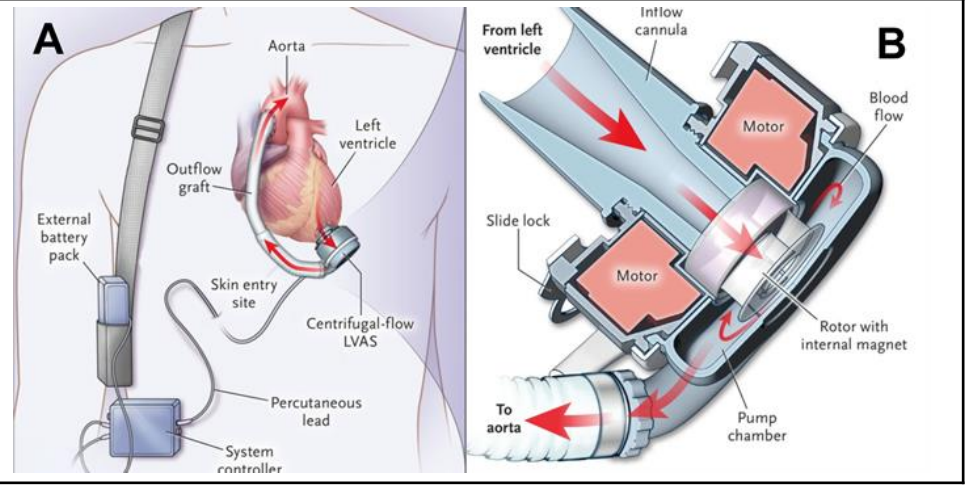


Advanced Therapies for End-Stage Heart Failure

When to refer to HF specialist

I: IV inotropes
N: NYHA class IIIB/IV or persistently elevated natriuretic peptides
E: End-organ dysfunction
E: EF ≤ 35%
D: Defibrillator shocks
H: Hospitalizations >1
E: Edema despite escalating diuretics
L: Low SBP ≤ 90, high HR
P: Prognostic medication; progressive intolerance or down-titration GDMT

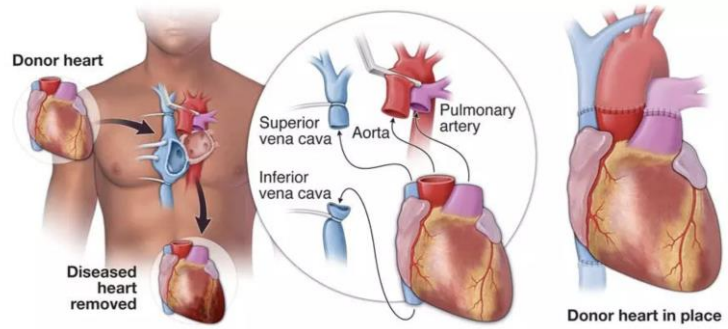
HeartMate3



Advanced Therapies Considerations

Types	Heart transplant Device replacement therapy (ex. LVAD, total artificial heart)
Considerations	Transplant candidacy Eliminate/optimize infection risk Cancer screening Hemodynamic parameters of end-stage remodeling (e.g. TPG < 15 and PVR < 4 WU) Frailty Psychosocial support Substance use

Heart Transplantation



Mechanical Circulatory Support: LVAD

When to Consider Left Ventricular Assist Devices (LVADs)
1. NYHA IV refractory to maximal medical therapy 2. LVEF < 25% 3. Reduce functional capacity (VO_2 < 14 mg/kg/min)

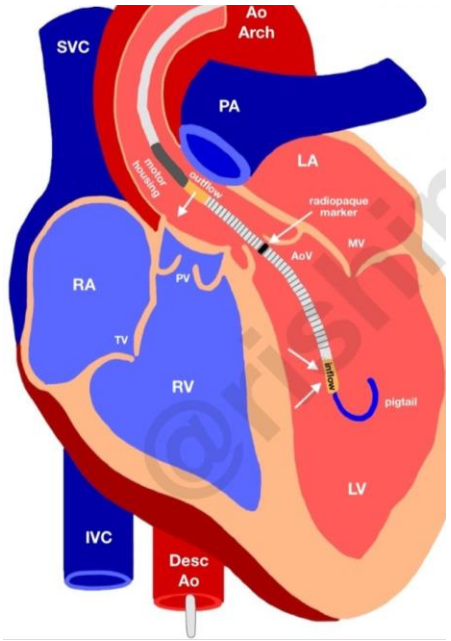
Why it is Used
• **Destination therapy:** if patient not transplant candidate • **Bridge to transplant:** for transplant candidates • **Bridge to decision:** not transplant candidates at time of LVAD placement, but may become eligible • **Bridge to recovery:** rare; improvement in LV function after LVAD placement and can undergo explantation

Contraindications
Limited life expectancy (age >80, active malignancy) Severe comorbidities (ESRD, severe liver, lung, vascular, or neuromuscular disease, unresolved CVA, etc.) Hematologic (active severe bleeding, severe thrombocytopenia, active infection, refusal of blood transfusions, intolerance of anticoagulation, confirmed HIT) Anatomic (CHD, HCM, large VSD, high BMI) Hemodynamic (RV failure, significant AI, PVR >6, TPG >15) Psychosocial (ongoing tobacco, alcohol, or drug use, inability to adhere to medication regimen, inability to maintain device, psychosocial instability)

Common Complications	
Hemocompatibility	**Stroke:** insufficient aspirin or AF (ischemic). INR >3, MAP >90 (hemorrhagic). **GI bleed:** Caused by AVMs from acquired vWF deficiency. Treatment involves standard resuscitative measures, temporarily holding aspirin and warfarin, and endoscopy. ARBs decrease risk of GIB. **Pump thrombosis:** triggers either left-sided heart failure symptoms or increases in LVAD power. Other signs: increased LDH and cola-colored urine (hemolysis). Treatment is pump-exchange.
Drive line infection	Prevention is key. Post-surgical stitch, dressing teaching and supplies, chlorhexidine. When they occur, antibiotics +/- debridement.
Persistent left-heart failure	Due to imbalance of afterload, LVAD rate, and preload. Rarer causes: cannula malposition, pump thrombosis, inflow/outflow cannula obstruction.
Right heart failure	CVP >15, CVP/PCWP ~1, hypotension, low CI, low PA sat on post-op RHC indicate right-heart failure manifesting as cardiogenic shock.
Aortic insufficiency and arrhythmia	

Mechanical Circulatory Support: Impella

Impella Overview	
What is it?	An axial heart pump placed retrograde across the aortic valve into the LV (i.e. unloads LV)
Improves	Cardiac output MAP
Reduces	LVEDP Myocardial workload Oxygen consumption
Dependent on	LV preload, thus dependent on intact RV function

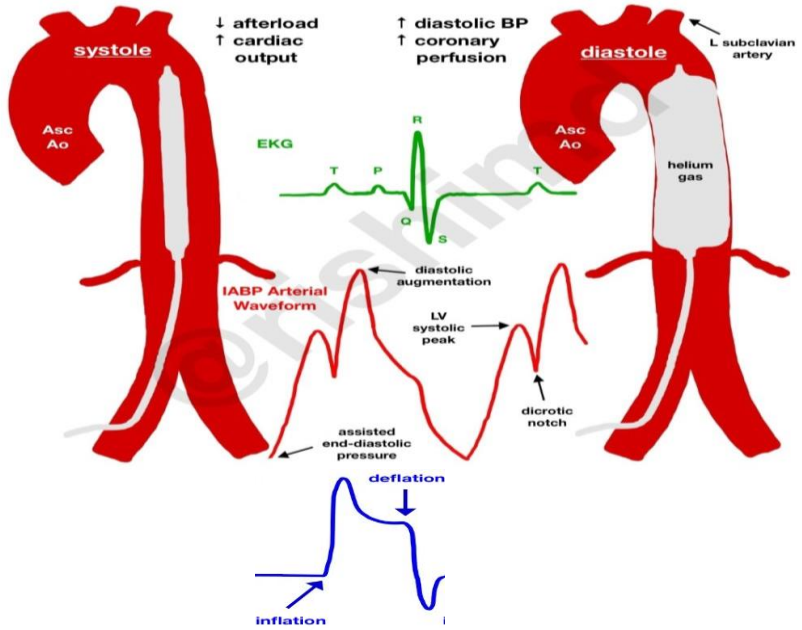


Components
• Motor • Pigtail: stabilizes catheter in the LV • An inflow: suctions blood from the LV • An outflow: propels blood into the ascending aorta

Usage	
Indications	• High-risk PCI • Cardiogenic shock • Ongoing ischemia • Bridge to other forms of circulatory support
Contraindications	• Mechanical aortic valve • Significant AS/AR • LV thrombus • Cardiac tamponade • Severe PAD
Complications	• Bleeding from heparinization • Hemolysis and thrombocytopenia from axial pump • Intracardiac injury from AV and papillary muscles • Peripheral vascular ischemia • Suction events (LV collapse due to low preload) • Acquired von Willebrand Syndrome
Management	• Heparin gtt • Removal: decrease P-level by 2 q2-4 hr; contact interventional cards fellow once stable on P1 (lowest level)

Mechanical Circulatory Support: IABP

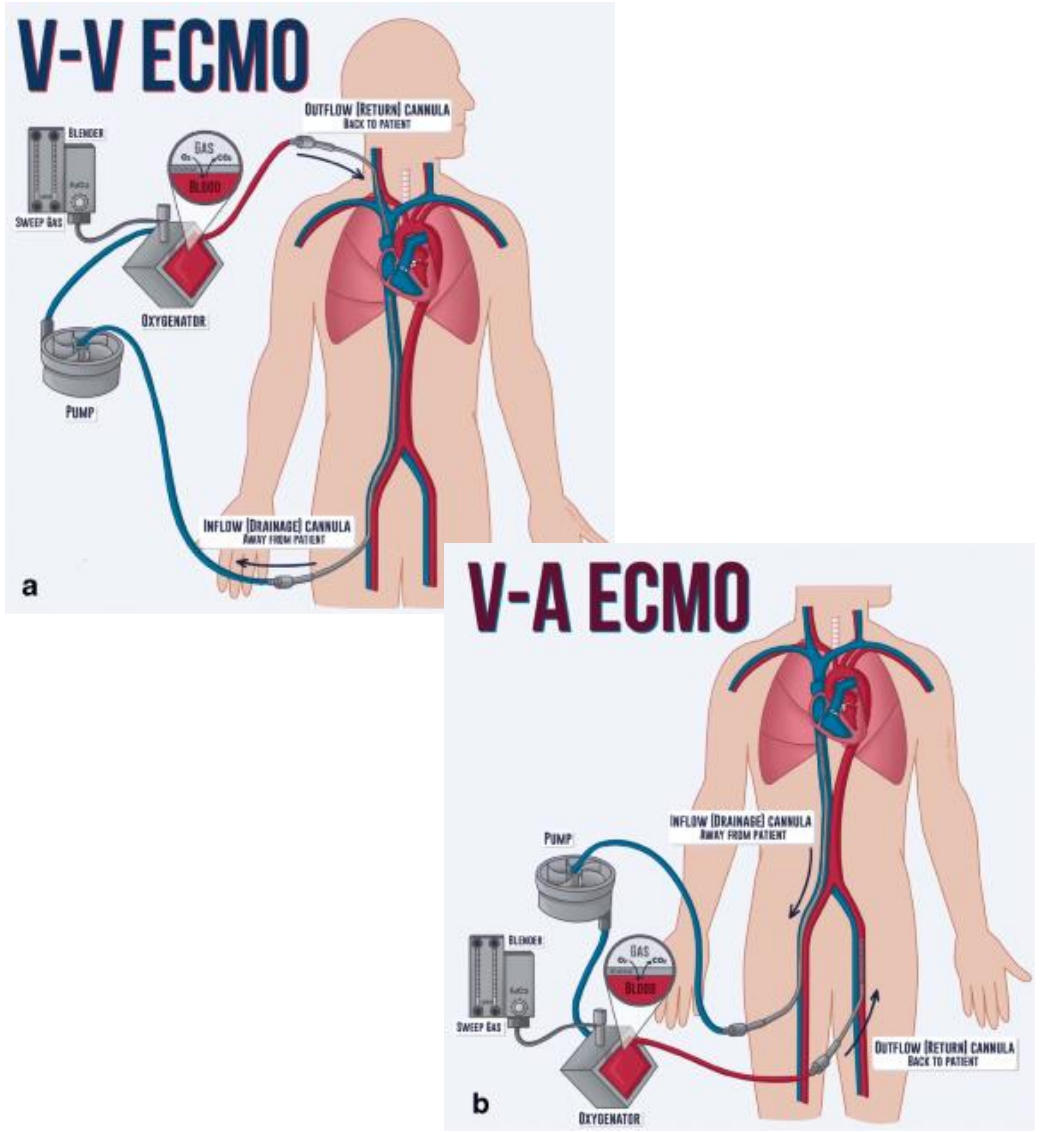
Intra-aortic balloon pump (IABP) Overview	
What is it?	Increases aortic diastolic pressure to improve coronary perfusion
Positioning	Within the aorta, distal to the aortic arch and proximal to renal artery takeoff
Inflation	Occurs with helium during diastole Inflates at dicrotic notch on arterial waveform
Deflation	Occurs during systole (decreases afterload → improves CO) Deflates on R wave



Usage	
Indications	• Complex PCI • Cardiogenic shock • Unprotected left main and LAD angioplasty • Papillary muscle rupture • Severe ischemic MR, VSD
Contraindications	• Aortic insufficiency • AAA • Aortic dissection • Severe PAD
Complications	• Acute limb ischemia • Severe bleeding • CVA/cholesterol emboli • Renal artery obstruction
Management	• Heparin gtt • Neurovascular checks q4 • Daily CBC to monitor for mechanical hemolysis, consumptive thrombocytopenia • Daily CXR (check tip is distal to left subclavian artery)
Removal	• Wean IABP from 1:1 → 1:2 → 1:4 while heparin gtt on • If stable on 1:4, go back to 1:1 and turn off heparin gtt • Call interventional cards fellow to pull IABP

Mechanical Circulatory Support: ECMO

Extracorporeal Membrane Oxygenation (ECMO) Overview	
What is it?	Drains blood from large vein, oxygenates it via membrane oxygenator, pumps it back to body via a vein (VV) or artery (VA)
Types	<u>Veno-venous (VV) ECMO</u> • Used for isolated respiratory failure (ARDS, ILD, etc.) <u>Veno-arterial (VA) ECMO</u> • Used for combined cardiopulmonary failure (PE, MI, high risk PCI, etc.)
Components	• Inflow and outflow cannulas • Pump • Membrane oxygenator



Heart Transplantation

Indications
• Cardiogenic shock requiring either continuous IV inotropic support or circulatory support • Persistent NYHA IV symptoms refractory to maximal medical and surgical therapies • Intractable angina with CAD not amenable to PCI • Intractable, life-threatening arrhythmias unresponsive to medical therapy, catheter ablation, surgery, or ICD • Select patients with restrictive or hypertrophic cardiomyopathies and NYHA III-IV

Absolute Contraindications
Limited life expectancy < 2 years
Irreversible PH with PVR >4-6 WU (case-by-case)
Severe and symptomatic cerebrovascular disease
Active substance (drug and alcohol) abuse
Multiple demonstrations of inability to comply with drug therapy
Multisystem disease with severe extra-cardiac organ dysfunction

Relative Contraindications
Age >70
Obesity (BMI >35)
Diabetes mellitus with poor glycemic control (HgbA1c >7.5%) despite optimal effort or end-organ damage other than non-proliferative retinopathy
Irreversible renal dysfunction
Neoplasm
Infection
Acute PE
Tobacco use within 6 months
Substance (drug or alcohol) abuse within 6 months
Inadequate psychosocial support
Other conditions that increase the risk of perioperative complications or limit tolerance of immunosuppression

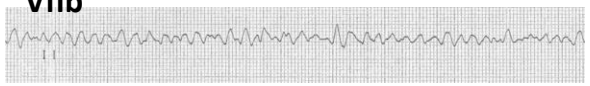
High-yield drug therapies and interactions in heart transplant	
Maintenance immunosuppression	calcineurin inhibitor (CNI) (tacrolimus > cyclosporine), antimetabolite (MMF > AZA), and tapering doses of glucocorticoids over first year post-transplant
Drugs that increase CNI levels	CCBs, antifungals, macrolides, fluoroquinolones, HIV-protease inhibitors, amiodarone
Drugs that decrease CNI levels	rifampin, phenytoin, phenobarbital, octreotide
Drugs with synergistic nephrotoxicity with CNI	aminoglycosides, amphotericin B, and NSAIDs
Drugs whose concentrations increase with CNI	statins, ezetimibe, and colchicine

Vasopressors

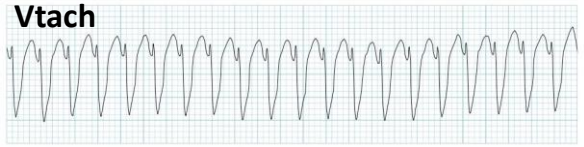
Drug	MoA	HR and Inotropy	Systemic Vascular Resistance	Cardiac Output	Clinical Indications	Notes
Inopressors						
Norepinephrine	α1+++ β1+++	↑	↑↑	↔/↑	Most types of shock *Good first line agent	Peripheral dose: reduced concentration through 18G PIV above elbow
Epinephrine	α1+++ β1+++ β2++	↑↑↑	↑↑	↑↑	Septic shock refractory to NE and IVF resuscitation Bradycardic shock	Also causes venoconstriction (↑preload) SE: stress cardiomyopathy in long periods
Pure vasopressors						
Vasopressin	V1/V2	↔	↑↑	↔	Distributive shock (vasopressin depleted states) *Good second agent to add to NE **Effective in severe acidosis	Also causes venoconstriction (↑preload)
Phenylephrine	α1++	↔	↑↑	Variable	Distributive shock (eg. neurogenic, anaphylactic shock = vasodilatory states with high CO) Sepsis with accompanying tachyarrhythmia	
Inodilators (or Inotropes)						
Dobutamine	β1+++ β2+	↑↑↑	↓	↑↑↑	Cardiogenic shock	SE: hypotension, tachyarrhythmia*
Milrinone	PDE4i (↑cAMP)	↑↑↑	↓	↑↑↑	Cardiogenic shock (theoretical preference in pulmonary hypertension and/or RV dysfunction)	Renally eliminated SE: hypotension, tachyarrhythmia*

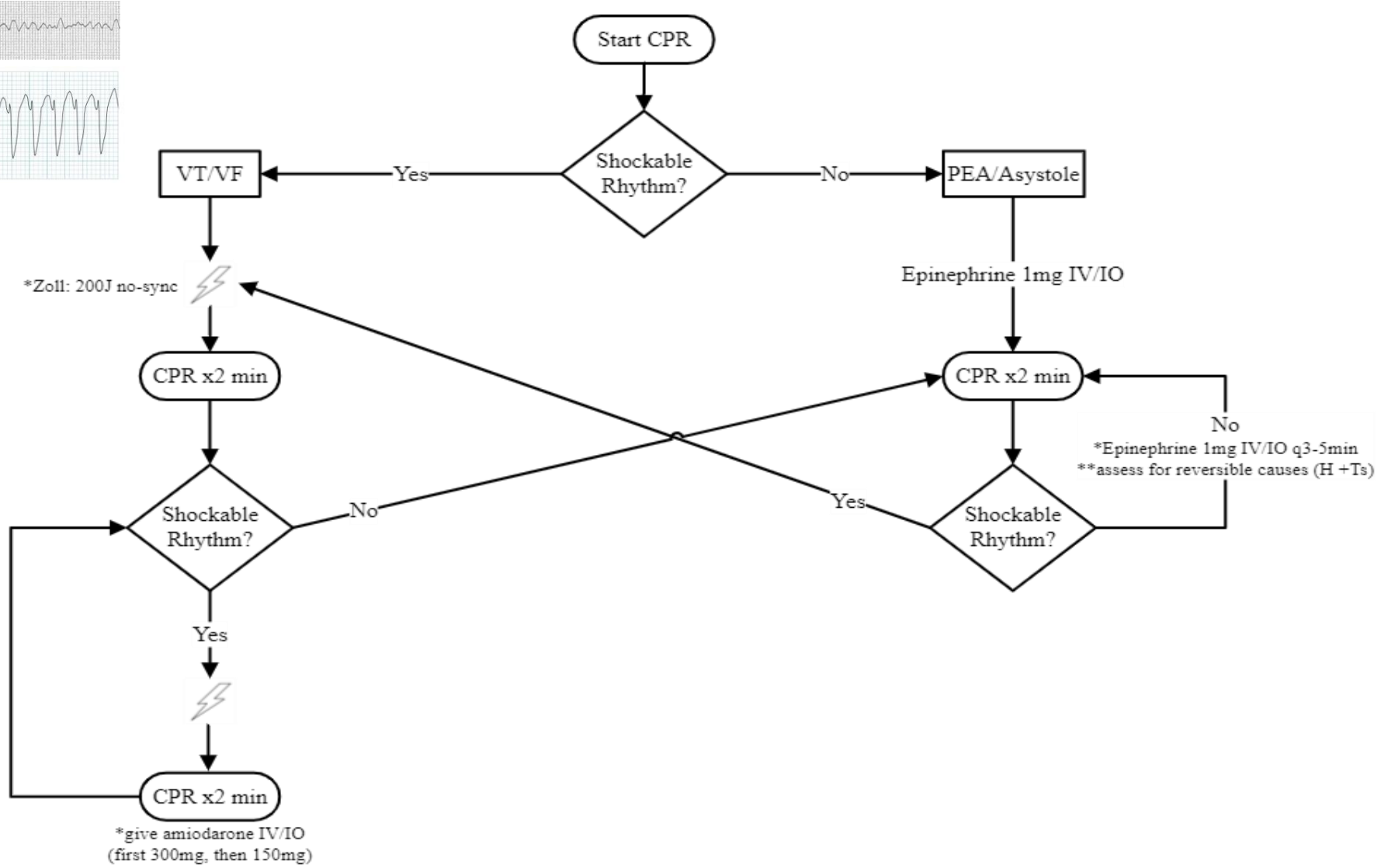
ACLS Algorithm

Vfib



Vtach





Post-Arrest Care

1. Early Post-Arrest Care

Determine Etiology of Cardiac Arrest

Etiology	Evaluation	Treatment
Acute Coronary Syndrome	EKG Troponin (Expected: hsTrop 0-4000 from compressions/defib) TTE (Expected: some degree myocardial stunning)	PCI
PE	CTA Chest TTE (McConnell Sign, RV Strain)	PE Team: lytics vs. thrombectomy vs. AC
GI Bleed	Digital Rectal Exam Nasogastric Lavage CT Abdomen/Pelvis w/ contrast CTA Abdomen/Pelvis Abdominal Angiography	Endoscopy vs. Surgery
Brain Bleed	CT Head	Reverse coagulopathy Interval CTHead in 4-8hrs Neurosurgery consult
Sepsis	CBC Blood Cultures Lactate	IV Fluids Antibiotics Source Control

*remainder of Hs and Ts per ACLS page

Neurologic Resuscitation

Measure	Goal	Reason
PaCO2	40-50	Hyperventilation = cerebral vasoconstriction
O2	SpO2 >94% PaO2 100-120	Hypoxia and hyperoxia (PaO2 > 300) are associated with worse outcomes
MAP	>65 (Ideally 80-100)	Optimizes brain perfusion *NE = vasopressor of choice
Head elevation	Head of bed @ 30 degrees	Decreases intracranial pressure and aspiration risk
Osmolality	Stable	Decrease cerebral edema risk *Avoid dextrose and/or hypotonic fluids

2. PCAC Scoring *link to app

PCAC = Pittsburgh Cardiac Arrest Care

	Examination	Survival	Meaningful Neurologic Recovery
PCAC1	Awake Purposeful movements Follows simple commands	80%	60%
PCAC2	Coma Minimal ventilator + vasopressor support (eg. NE < 0.1)	60%	40%
PCAC3	Coma High ventilator + vasopressor support (eg. NE > 0.1)	30%	10%
PCAC4	Coma Absent pupil and/or corneal reflex No movement of extremities	10%	5%

*PCAS can assist with goals of care discussions

3. Targeted Temperate Management (indicated in PCAC Score ≥ 2)

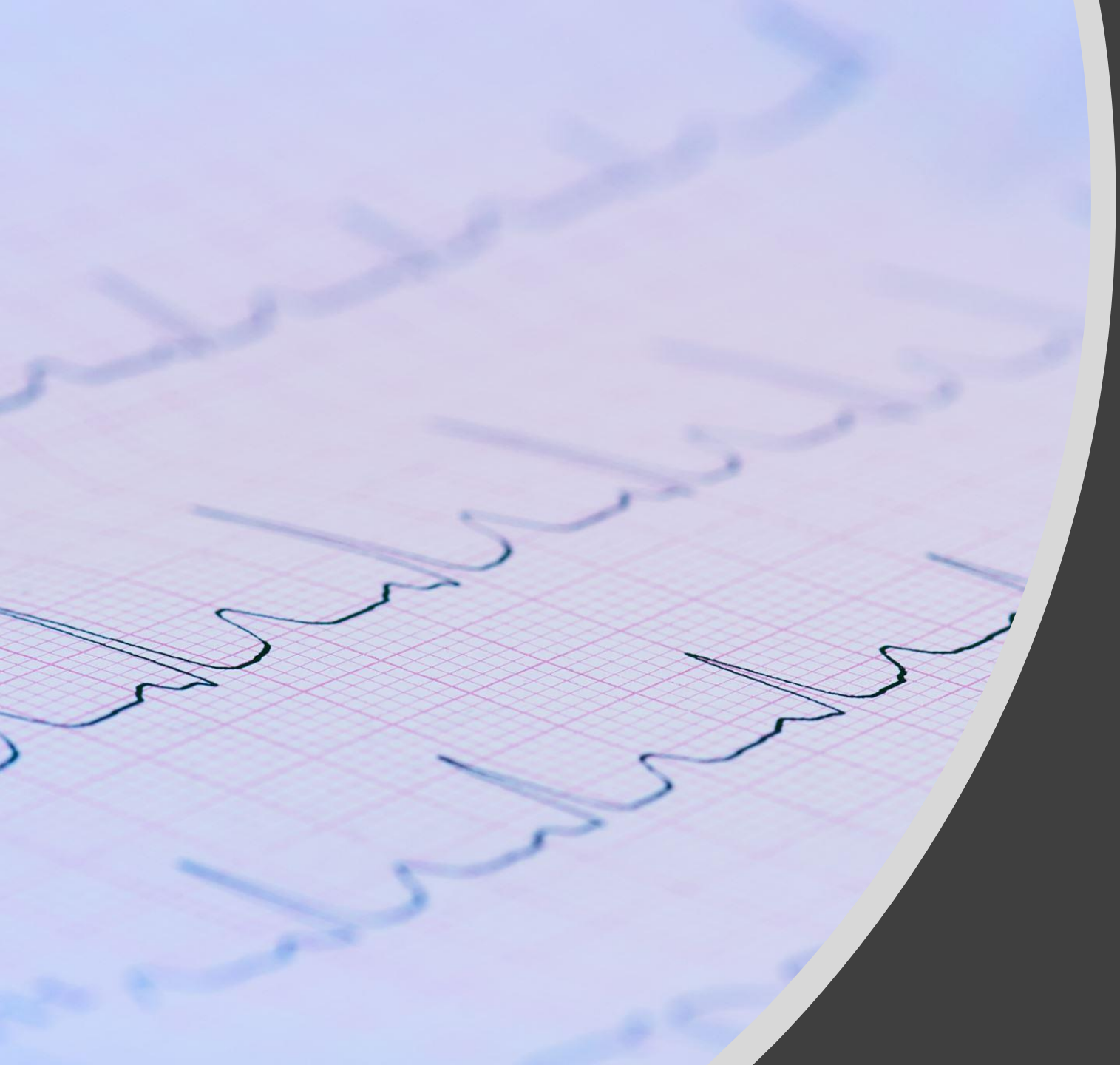
- PCAC 2: 36C
- PCAC 3-4: 33C

Sedation: propofol (easy reversibility for neurologic testing, role in ameliorating shivering and seizures) or ketamine + fentanyl

4. Late Post-Arrest Care

Neuroprognostication (NO SINGLE TEST IS PROGNOSTIC)

Modality	Timing	Useful Prognostic Signs
CT Head w/o contrast	On Admission	Poor outcomes: - Quantitative loss of grey-white differentiation - Qualitative Cerebral Edema (effacement of sulci) - Herniation/Impending herniation
EEG	Day 0-3	"Burst suppression with identical burst" = poor outcome Lance Adams' Syndrome = 50% good outcome
Clinical Exam	Continuous	Early (day 0-1) Malignancy Myoclonus *differentiate from Lance-Adams Syndrome which has better prognosis PCAC Scoring as above Absent of pupils on Day 3 = poor prognosis Improvement in exam = favorable
SSEP	Day 3	Preserved subcortical responses with absent N20 (cortical) responses = poor outcome
MRI Brain w/o contrast	Day 3-5	Diffuse cortical injury (>4 locations) = poor outcome

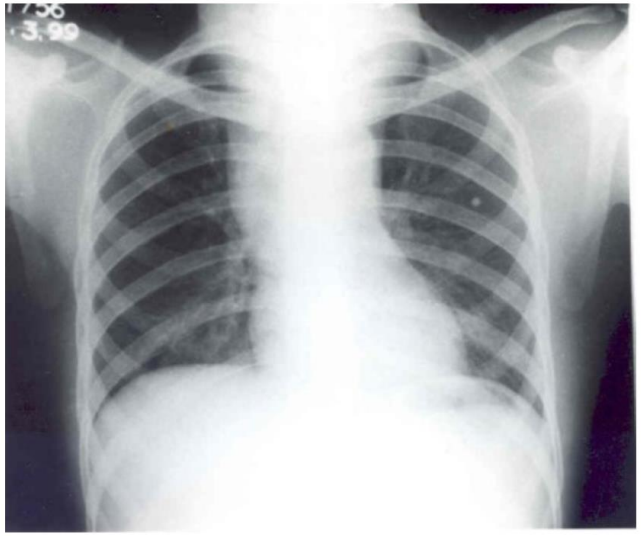


Vascular Disease

Section Editor:
Harnoor Mann, MD

Aortic Dissection

Aortic Dissection Overview		
When to suspect	• Chest pain (ascending aorta, aortic root dissection); presents similarly to an MI 2/2 disruption of the blood supply to the coronary vasculature. • Back pain (descending aorta) • Painless (~10%; usually in those with neurologic complications or Marfan syndrome) • Neurologic deficits (syncope, CVA, AMS)	
Exam Findings	• Hypertension (catecholamine surge v. underlying HTN) • Hypotension (excessive vagal tone, cardiac tamponade, hypovolemia) • Signs of heart failure (right or left-sided) • Other: cardiac tamponade, superior vena cava syndrome, wide pulse pressure, peripheral nerve ischemia (numbness/tingling), hoarseness (recurrent laryngeal nerve compression), Horner syndrome	
CXR Findings	• Widened mediastinum • Hemothorax • Abnormal aortic contour • Blunted aortic knob • Pleural effusion	• Left apical cap • Tracheal deviation to the right • Depression of the left mainstem bronchus • Esophageal deviation • Loss of the paratracheal stripe

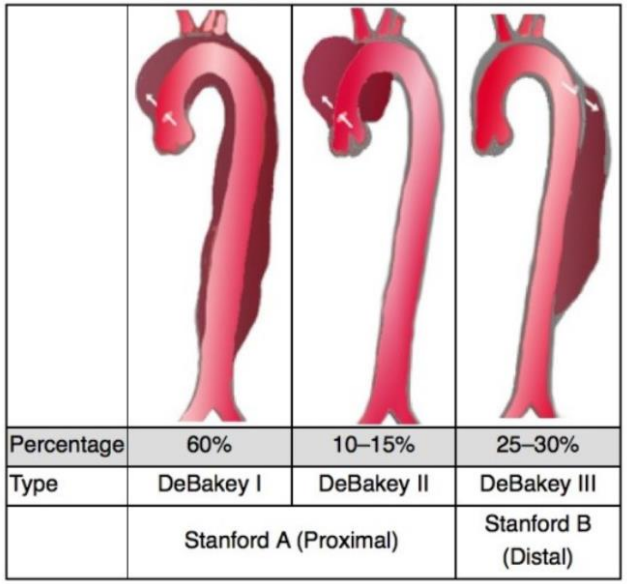


Chest X-ray with widened mediastinum

Aortic Dissection

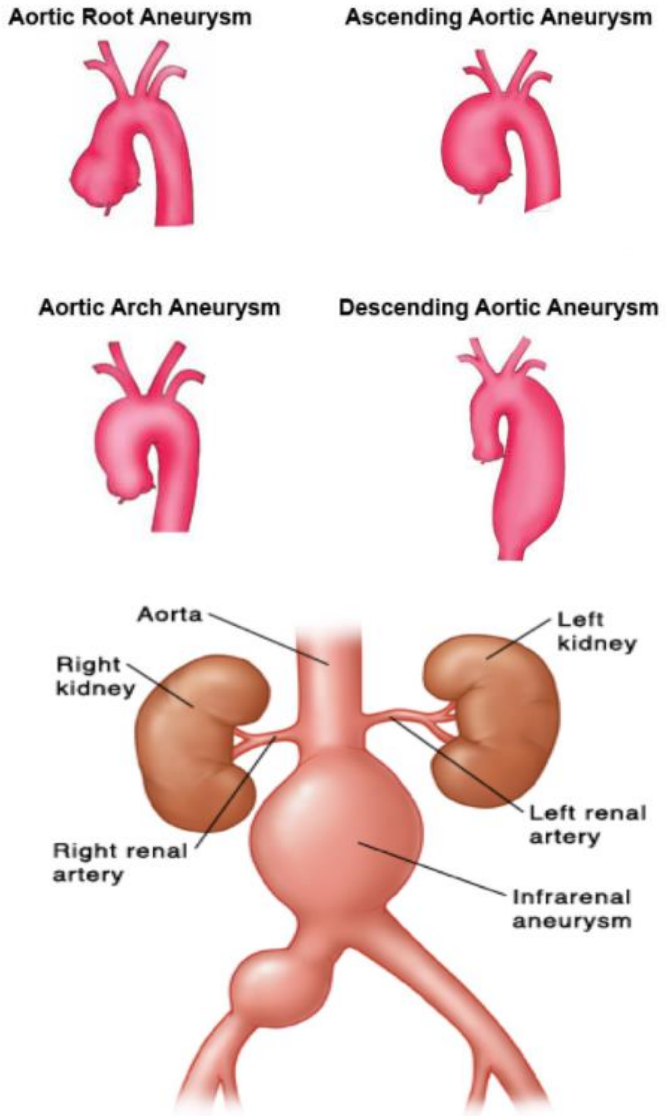
Aortic Dissection Diagnostics	
Confirmatory Testing	• First-line imaging choice in stable patient: CT angiography +/- 3D reconstruction; MRI if IV contrast contraindicated • First-line imaging choice in unstable patient: echocardiography (TEE is more sensitive and specific than TTE) • Smooth muscle myosin heavy-chain assay, with a cut-off of 2.5 has a sensitivity of 91%, specificity of 98%, and accuracy rate of 96%
Management	• HR goal: 60s • SBP goal: 100-120 mmHg or the lowest that maintains cerebral, cardiac, and renal perfusion with beta blockers (metoprolol, propranolol, esmolol, labetalol) +/- nitroprusside for minute-to-minute control of blood pressure • Use arm with higher pressure to monitor hemodynamics • Pressors: norepinephrine or phenylephrine preferred over dopamine, epinephrine • Contraindicated: thrombolytics (even in the setting of MI) • Baseline imaging required prior to discharge
Treatment	<u>Stanford A</u> Surgical repair <u>Stanford B (descending aortic dissection) and stable</u> Stable: Medical management Unstable (end organ ischemia): Surgical repair

Classification of aortic dissection



Thoracic and Abdominal Aortic Aneurysms

Aortic Aneurysm Overview	
Definition	Full thickness dilation involving ≥50% diameter increase involving all three layers of arterial wall (intima, media, adventitia)
Morphology	Fusiform (symmetrically dilated) Saccular (localized outpouching)
Locations	<u>Thoracic</u> Ascending aorta (50% of cases), aortic arch (10%), or descending aorta (40%) <u>Abdominal</u> Suprarenal, Pararenal, or Infrarenal (most common)
Surveillance	Thoracic AA: q12 months CTA Aortic AA: q6-12 months US
Screening	Males aged 65-75 ever-smokers should receive a one-time US for AAA
Conservative management	Smoking cessation, beta blockers, statin therapy



Thoracic and Abdominal Aortic Aneurysms

	Thoracic Aortic Aneurysm	Abdominal Aortic Aneurysm
Dimensions	Normal: 2.5 – 2.8 cm TAA: > 4.0 cm	Normal: 2.0 cm AAA: > 3.0 cm
Prevalence	0.3% (Rarer; affiliated with genetic and familial syndromes)	Most common: 2-8% More common in males >50yo with smoking history (4-8%)
Risk factors	Family history Connective tissue disorders (Marfan, Ehlers Danlos) Aortic stenosis Bicuspid Aortic valves (Ascending TAA's)	Atherosclerotic risk factors (smoking, hypertension, diabetes) Age>50 Male gender
Natural History	Expand at 0.1 – 0.3 cm/year 6.9% annual rate of rupture or dissection for aneurysms > 6.0 cm	Expand at 0.3 – 0.4 cm/year 10.2% rate of rupture for aneurysms size 6.0 to 6.9 cm 32% annual rate of rupture for aneurysms above 7.0 cm
Clinical Presentation	Most are silent Large aneurysms: dysphagia Ruptured symptoms: Tearing chest pain, pulse differential, hypotension	Pulsating abdominal mass, with or without abdominal pain Ruptured symptoms: Classic triad of abdominal pain, pulsating abdominal mass, and hypotension
Diagnostic Imaging	CT Angiography or MR Angiography (or TEE if anaphylactic IV contrast allergy + acute clinical situation)	CT gold standard for symptomatic patients Abdominal Ultrasound for screening

Surgical Interventions for Aortic Aneurysms

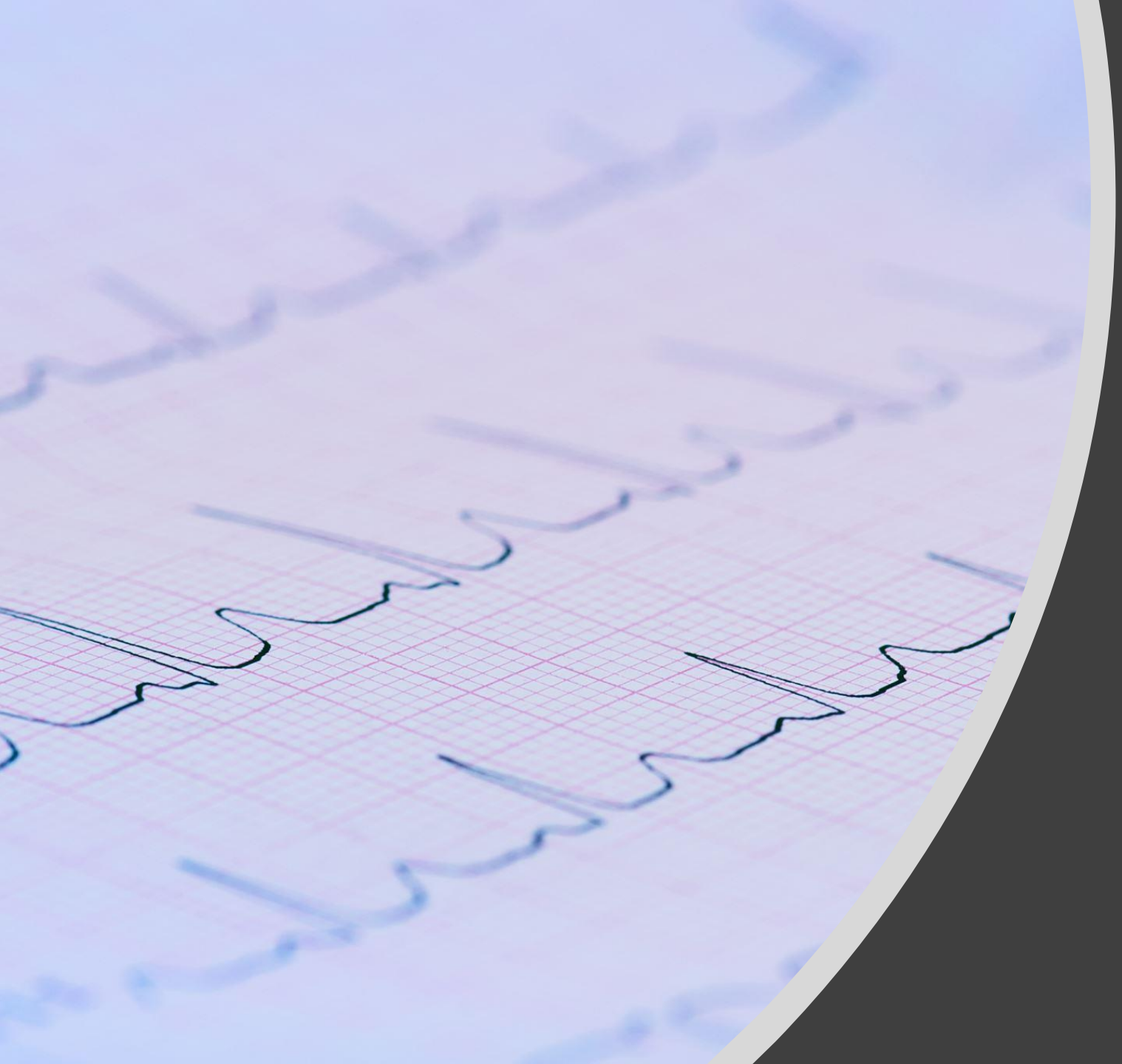
Invasive options for aortic aneurysms		
Operative Indications	Symptoms	Pain
	Size	TAA > 5.5cm TAA > 4.5 – 5.0 cm if certain conditions AAA > 5.5cm
	Rate of expansion	10mm growth in 12 months
	Complicated aneurysm	Ruptured, dissection
Conservative management	Thoracic aortic aneurysm	All ascending TAA's should be managed surgically Descending TAA's may be managed endovascularly
	Abdominal Aortic aneurysm	Surgical or endovascular (↓30-day morbidity)

Peripheral Artery Disease

PAD Overview	
Definition	• Stenosis of peripheral arteries (excluding coronaries), classically those supplying lower extremities: Iliac, femoral, popliteal, tibial, and peroneal vessels
Epidemiology	• Prevalence is 3-7% in general population; up to 20% in those over age 70 yrs
Symptoms	• Intermittent claudication: Pain in calves, thighs, or buttocks on walking that is relieved with rest. • Symptoms suggestive of advanced disease: Blue or cold skin, skin atrophy, diminished hair growth, and painful skin ulcers
Risk factors	• Smoking (most important), diabetes, hypertension, hyperlipidemia, known atherosclerotic disease (CAD, carotid dx, AAA, etc.), age, male gender
Diagnosis	• Ankle Brachial Index (ABI) = Systolic BP of lower limb / Systolic BP of Upper Limb
Emergency scenarios	• Gangrene (emergent surgical evaluation for possible amputation to minimize risk of infection) • Acute Limb Ischemia: resting pain, numbness, or motor weakness should warrant emergent surgical revascularization
Revascularization options	• Balloon angioplasty, atherectomy, bypass, and thrombolysis

Therapies
• Quit smoking • Supervised walking exercise prevent disease progression • Glycemic control • HTN control • High-intensity statin therapy • Aspirin 81 mg or Clopidogrel 75 mg • Cilostazol 100 mg BID may be added to improve walking endurance

ABI	Interpretation
>1.40	Noncompressible, calcified vessel (uninterpretable)
1.0-1.4	Normal
0.91-0.99	Borderline
0.41-0.90	Mild to moderate PAD
.00-0.40	Severe PAD



Myocardial & Pericardial Disease

Section Editor:
Scott Baumgartner, MD

Hypertrophic Cardiomyopathy (HCM)

<table>
<tr><th colspan="3">Overview</th></tr>
<tr><td>What</td><td colspan="2">• Heterogenous disorder caused by mutations associated with hypertrophy of the LV with systolic and diastolic dysfunction</td></tr>
<tr><td>Clinical Presentation</td><td colspan="2">• Fatigue, dyspnea, presyncope
• Chest pain: ↑ O2 due to myocyte hypertrophy, increased muscle mass, and LVOT obstruction
• Syncope: inadequate cardiac output
• Arrhythmia: AF, NSVT, VT, VF
• Acute hemodynamic collapse: ↓ preload or afterload, tachyarrhythmia, or acute mitral insufficiency</td></tr>
<tr><td>Physical Exam</td><td colspan="2">• Systolic murmur: harsh crescendo-decrescendo heard best at apex; ↑intensity with Valsalva and sitting-to-standing (↓preload; ↑ LVOT obstruction)
• S3 and S4 common in young patients; rare older patients</td></tr>
<tr><td>Diagnosis</td><td colspan="2">• EKG: prominent Q waves, inverted (giant negative) T waves
• TTE: LVH (>13-15mm), typically asymmetric (most commonly basal anterior septum), systolic anterior motion of mitral valve, LVOT obstruction (gradient > 35-50 mmHg)
• MRI: anatomical evaluation, assess for fibrosis
• Exercise: risk stratification, LVOT assessment
• 1° relatives: H & P, EKG, TTE- q 1 year (< 18 y/o), q 5 y (>18 y/o)</td></tr>
<tr><td rowspan="2">Treatment</td><td><u>Pharmacologic</u>
Goals: (1) reduce LVOT obstruction; (2) increase LV filling by slowing HR and prolonging diastole; (3) decrease myocardial O2 demand
• Beta Blocker: bisoprolol, nadolol
• Non-DHP CCB: verapamil
• Disopyramide</td><td><u>Non-Pharmacologic</u>
• Alcohol septal ablation
• Surgical myomectomy
• ICD considered if VT/VF, wall thickness >30 mm, unexplained syncope, family history of arrest</td></tr>
<tr><td colspan="2"><u>Contraindicated</u>
Agents that reduce preload
• Diuretics, ACE-I/ARB
• DHP CCB, Nitro</td></tr>
</table>

Dilated Cardiomyopathy

Dilated Cardiomyopathy Etiologies	
Ischemic	Most common cause of HFrEF
Infectious	Viral, Chagas disease, Lyme disease
Toxic	Alcohol, cocaine, and medications (e.g. anthracyclines, anti-retrovirals)
Stress induced (Takotsubo)	Uncommon but increasingly reported
Peripartum	Rare; occurs in late pregnancy and the early postpartum period
Tachycardia-mediated	Chronic SVTs
Sarcoidosis	Immune granulomatous infiltration of the myocardium
Genetic	~50% of patients with idiopathic DCM have a familial disease
Idiopathic	Diagnosis of exclusion

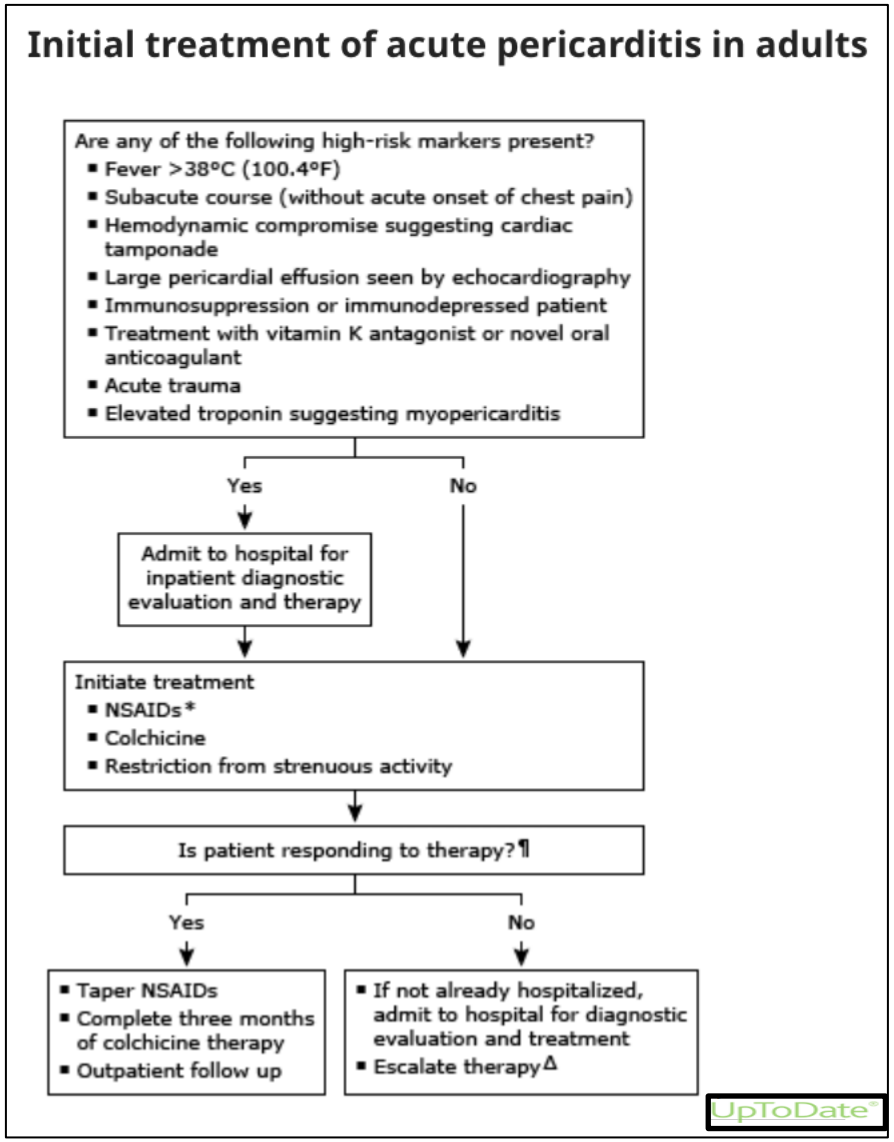
Restrictive Cardiomyopathy

Overview	
Clinical Presentation	• Signs of left and right sided heart failure • Atrial fibrillation (dilated LA due to increased filing pressures, arrhythmia from infiltrative diseases)
Diagnosis	• CXR: cardiomegaly w/ significant atrial enlargement, pulmonary venous congestion, and pleural effusions • TTE: normal, non-dilated LV size and function, normal wall thickness (except amyloid and glycogen storage disease), dilated bilateral atria, accentuated early diastolic filling of ventricles, diminished atrial filling (high E-to-A ratio) on mitral inflow velocities • Cardiac MRI: global sub-endocardial late gadolinium enhancement • Endomyocardial biopsy and staining definitive diagnosis of underlying condition
Treatment	• Treat underlying disease, beta blockers, CCB • Preload dependent given elevated filing pressures; avoid diuretics • Heart transplant is definitive treatment

Etiologies	
Familial non-infiltrative	Genetic variants associated with myosin, troponin, titin, etc.
Infiltrative	Protein (e.g. amyloid), iron, eosinophils, metabolic products, inflammation (e.g. sarcoid), and tumors
Inflammatory	Sarcoidosis
Treatment-related	Radiation, hydroxychloroquine, anthracyclines
Other	Diabetic cardiomyopathy, scleroderma, endomyocardial fibrosis, Noonan Syndrome, Werner Syndrome
Idiopathic	Diagnosis of exclusion

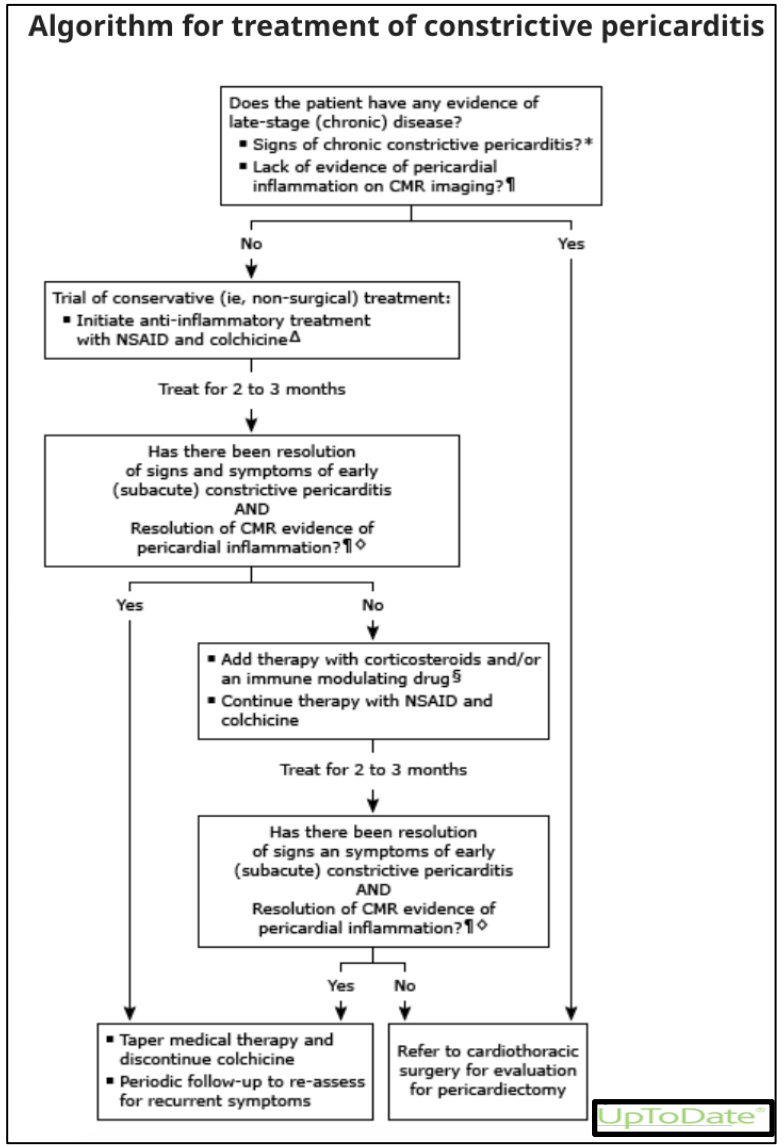
Acute Pericarditis

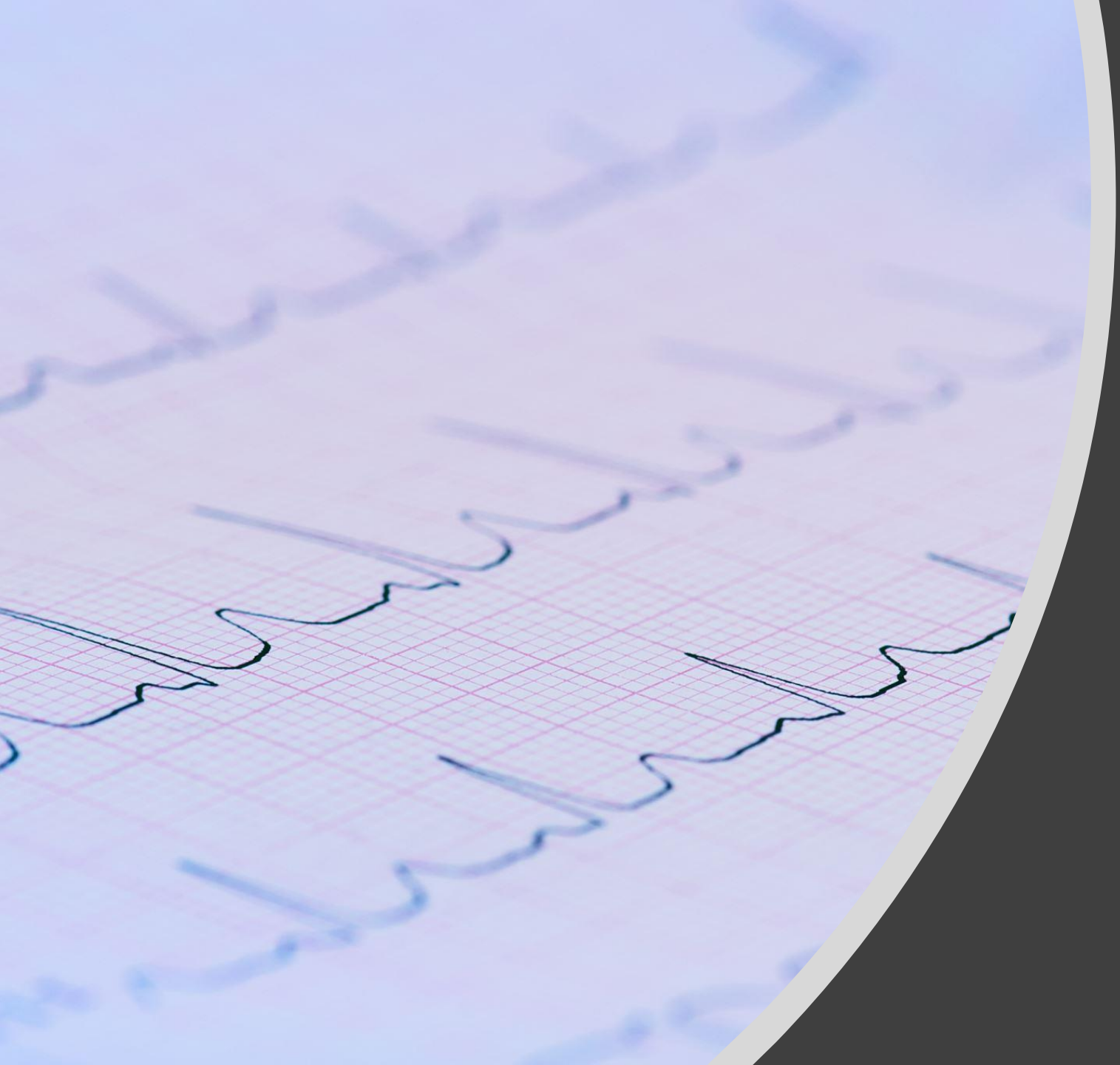
Acute Pericarditis Overview	
Etiology	• Most common: idiopathic or viral • Infectious: bacterial, fungal, TV, HIV • Other: malignancy, trauma, uremia, thoracic radiation, autoimmune, post-MI (Dressler syndrome), drugs (hydralazine, PCN, INH, chemo)
Clinical Presentation	• Chest pain: sharp, pleuritic, improved with sitting forward • Friction rub: heard best LLSB
Diagnostic Testing	• EKG: diffuse concave up ST elevations and PR depressions in all leads except aVR • TTE: pericardial effusions • Labs: CBC, hs-Troponin, ESR, CRP, blood culture • Consider TB, HIV, ANA, CT, and CMR based on clinical suspicion • Viral studies are low yield



Constrictive Pericarditis

Constrictive Pericarditis Overview	
Etiology	• Idiopathic, viral, post-cardiac surgery, post-radiation, connective tissue disorder, post-infectious (TB)
Clinical Presentation	• Symptoms: dyspnea on exertion, lower extremity edema • Signs: right hear failure, JVD, prominent ”x”, rapid ”y” descent, hepatosplenomegaly, ascites, edema, pleural effusions • Kussmaul’s Sign: ↑ JVP with inspiration • Pericardial knock best heard at left sternal border or apex in early diastole
Diagnostic Testing	• TTE: dopplers show severely impaired filling with respiratory variation but normal apparent relaxation • CT, CMR provides information about calcifications, pericardial thickness, and extent of pericardial involvement • RHC/LHC: equalization of RV and LV diastolic pressures (ventricular interdependence)



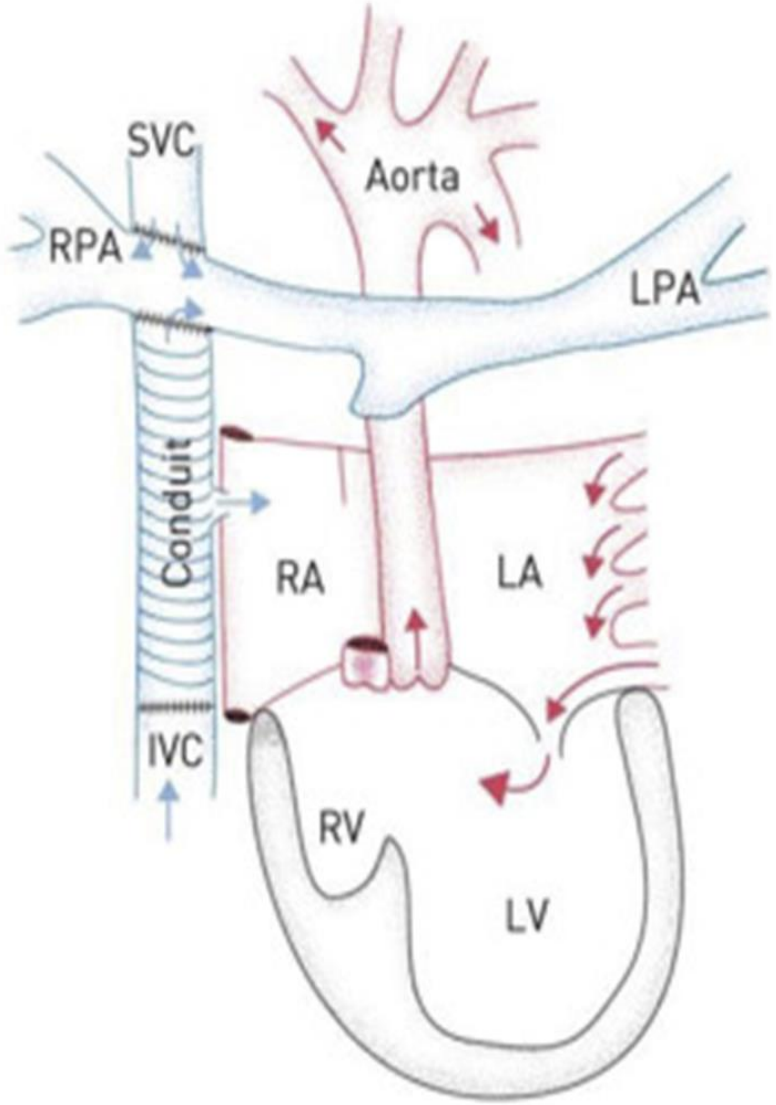


Congenital Heart Disease

Section Editor:
Talya Mandelkern, MD

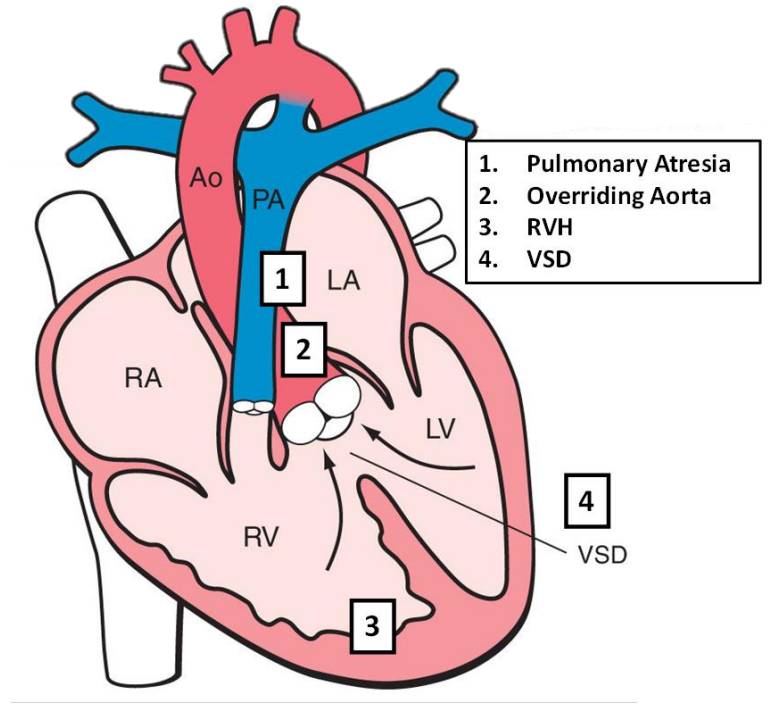
Cyanotic: Fontan

Fontan Overview	
What	Early childhood repair of single ventricle physiology • Tricuspid atresia • Pulmonary atresia • Hypoplastic left heart • Ebstein anomaly
Steps	• Norwood → Glenn → Fontan • Anastomosis of SVC/IVC to PA, bypassing RV with ligation of Main PA
Goal	• SpO2 90-95% • CVP 6-10mmHg
Complicated by	Arrhythmia, congestive hepatopathy, protein losing enteropathy, heart failure, PH, plastic bronchitis, FTT, kidney disease
Consider	• Frank-Starling Curve: all venous return is passive and preload dependent • Associated w/ hypoalbuminemia, coagulopathy, malnutrition • Increased risk for thrombosis, endocarditis



Cyanotic: Tetralogy of Fallot

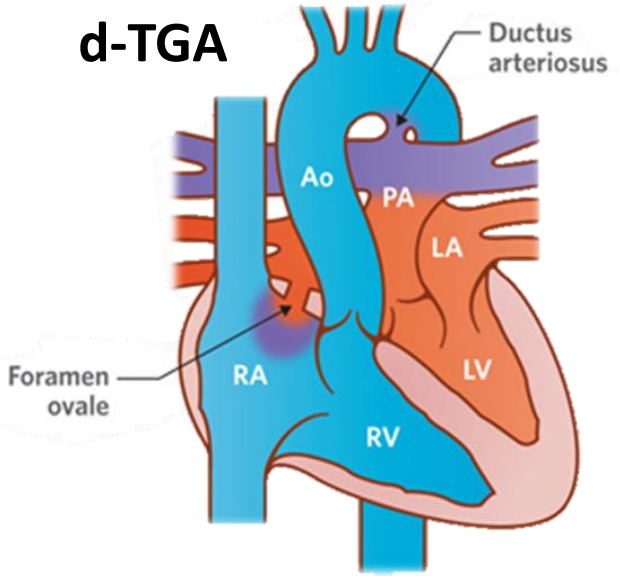
Tetralogy of Fallot Overview	
What	• Ventricular septal defect • Overriding aorta • Pulmonary stenosis • Right ventricular hypertrophy
Repair	• VSD patch closure • Pulmonic valve repair or replacement • Right ventricle → pulmonary artery conduit (Rastelli)
Complicated by	• Arrhythmia • RV dysfunction • Aortic valve/pulmonic valve dilation and insufficiency • Hepatopathy
Consider	• Conduit stenosis • Thrombosis • Risk for endocarditis • CVA



Transposition of Great Arteries

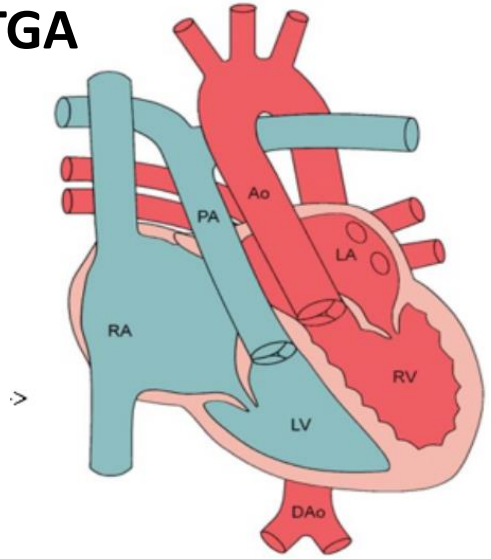
d-TGA (Dextro Transposition of Great Arteries)	
How it works	<u>Cycles in Parallel</u> Body → right atrium → right ventricle → aorta Pulmonary vein → left atrium → left ventricle → pulmonary artery

d-TGA



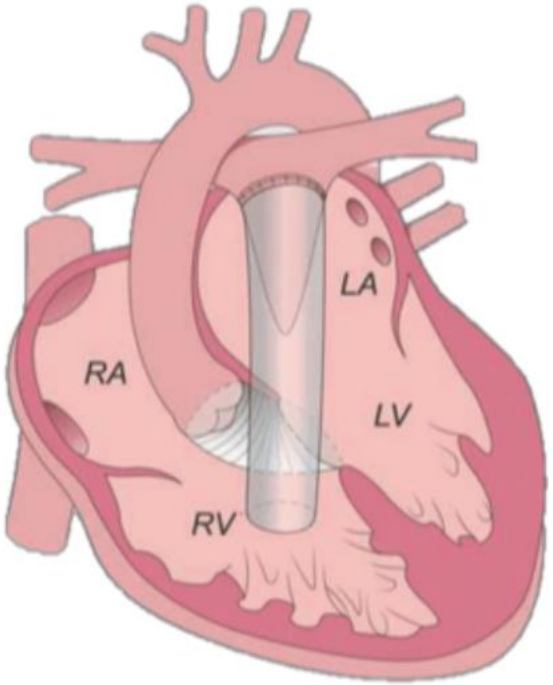
CC (L-) TGA (Congenitally Corrected or Levo Transposition of Great Arteries)	
How it works	<u>Cycles in Series</u> Body → right atrium → anatomic left ventricle → pulmonary artery → lungs → pulmonary vein → left atrium → anatomic right ventricle

CC-TGA



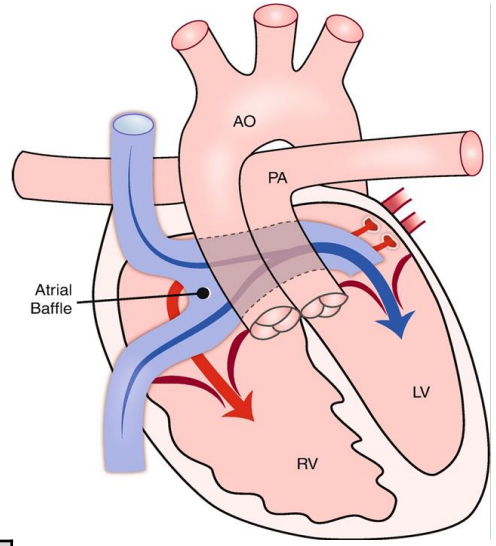
Surgical Repair: Rastelli

Rastelli Procedure Overview	
What	Surgical procedure to correct transposition of the great arteries (TGA), ventricular septal defect (VSD), and pulmonary stenosis
Repair	• Right ventricle → pulmonary artery conduit, patch directing flow • Left ventricle → aorta through VSD • <u>Repair of VSD and RV outflow obstruction</u> Truncus arteriosus [combined MPA/Aorta] d-TGA DORV VSD/PA atresia
Consider	• PA stenosis • Thrombosis • Conduit size

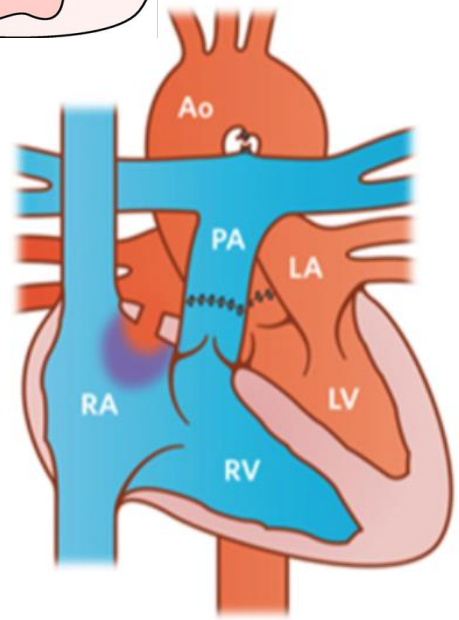


Surgical Repair: Switch Procedures

Mustard and Senning (i.e. ATRIAL Switch) Procedures Overview	
What	Surgical procedure to correct transposition of the great arteries (TGA)
Repair	Baffle (a.k.a. a bridge) of superior vena cava or inferior vena cava to left atrium
Consider	• Tricuspid regurgitation • Right ventricular failure (as RV managing systemic pressures)



ARTERIAL Switch Procedure Overview	
What	Surgical procedure to correct transposition of the great arteries (TGA)
Repair	<u>Anastomosis of:</u> • Aorta → LVOT • Main pulmonary artery -> RVOT • Repair d-TGA
Consider	• Arrhythmia • PA/coronary stenosis • Neovalvular insufficiency

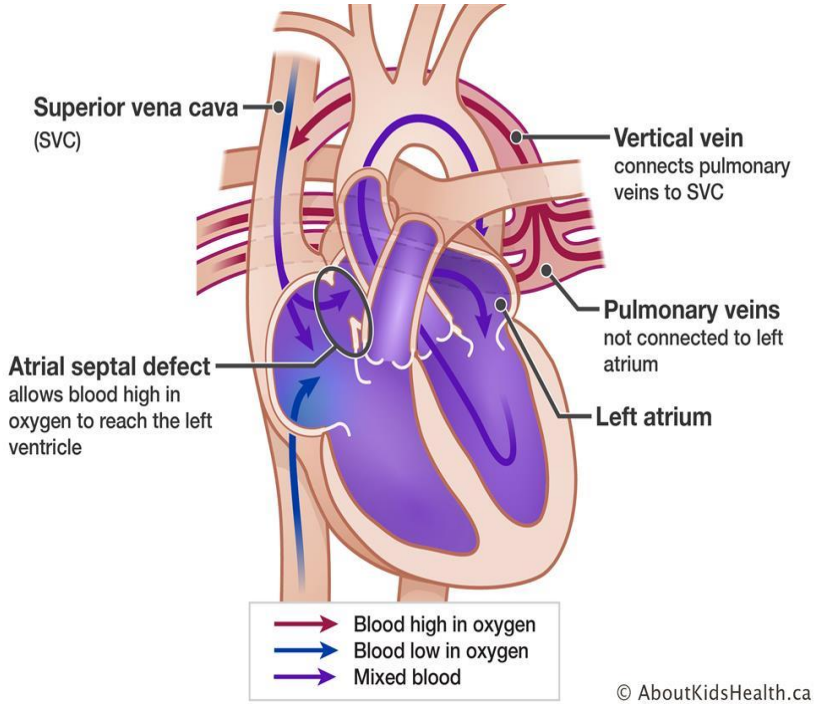


Common Acyanotic Abnormalities

ACYANOTIC		UNREPAIRED	REPAIRED	Consider	If echo shows…
Atrial Septal Defect (ASD)	LA → RA shunt ∴ RH overload → RH Failure	RA/RV dilation RVH, pHTN, arrhythmia	LV dysfxn, conduction abnormalities	↑ risk CVA, endocarditis if shunt	Hypermobile atrial septum, RA/RV overload
Ventricular Septal Defect (VSD)	LV →RV shunt ∴ LH overload → LH Failure	LA/LV dilation, ↑ pulm flow, pHTN, AR/AS	Repaired early if symptoms, arrhythmia	↑ risk endocarditis if shunt	LVH, systolic septal flattening, ↑ pulm flow
Atrioventricular Canal Defect	L→R with flow throughout	Treat as Eisenmenger	AV insufficiency, LVOTO, pHTN, MR/MS, arrhythmia	Associated with Trisomy 21	RVH, ↑RVP
Pulmonary Stenosis	Muscular, supra/valvular, isolated or complex	RH Failure, RVOTO	Transcatheter balloon valvuloplasty or surgical; c/b dynamic RVOTO	Associated with Noonan, Alagille, Williams, congenital Rubella	RVH, ↑RVP, RV dysfunction; PV velocity/gradient
Patent Ductus Arteriosus	Persistence of fetal ductus arterosis; Aorta → PA	If small enough ø; Else, PAH	Coil embolism or surgical ligation if symptoms	↑ risk CVA, endocarditis, aneurysm	LVH, LAD, aortic diastolic reversal of flow
Coarctation of Aorta	Narrowing of proximal thoracic aorta; isolated or complex	Proximal HTN; distal hypoperfusion; collateral vessels	Transcatheter balloon angioplasty/surgical resection + anastomosis	Associated with Turner's, bicuspid aortic valve; mesenteric ischemia, ICH, HTN encephalopathy, AAA	Dilated ascending aorta; forward diastolic flow; collateral flow
Eisenmenger	Systemic-pulmonary shunt → pulmonary vascular disease → pHTN → shunt reversal ∴ R→L flow → cyanosis MOST UNREPAIRED DEFECTS LEAD TO EISENMENGER			Anemia; 2° polycythemia; ↑ uric acid; PA thrombosis; paradoxical air emboli	RV dilation, RVH

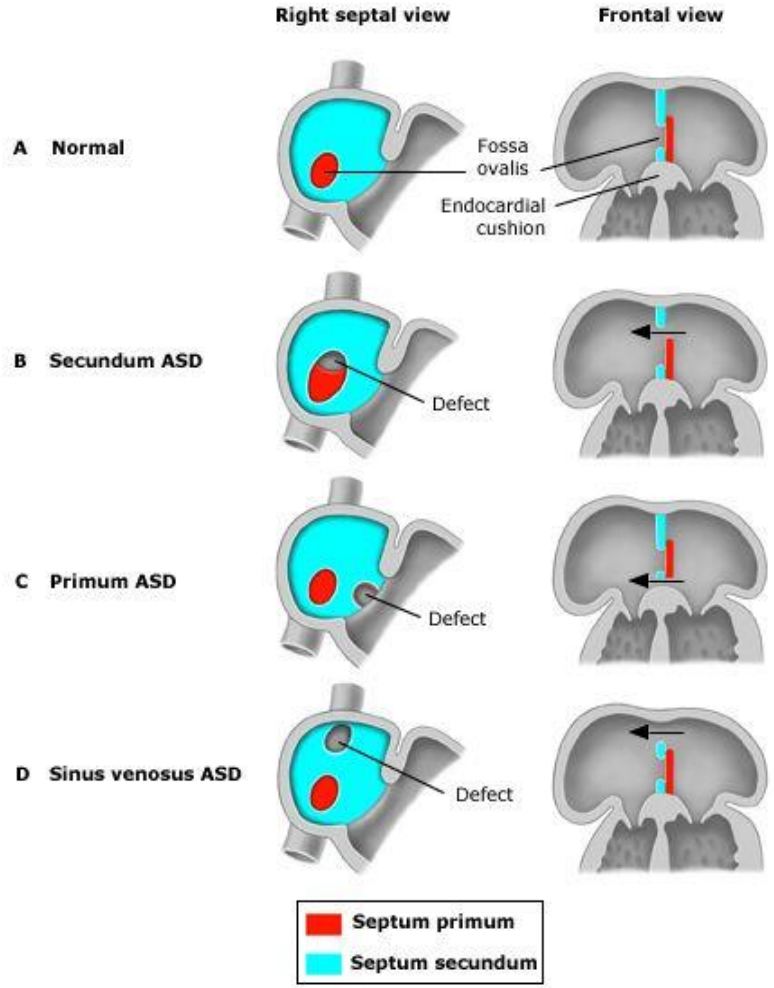
Acyanotic: Anomalous Pulmonary Venous Return

Anomalous Pulmonary Venous Return Overview	
Types	• Total (TAPVR) • Partial (PAPVR)
Pathophysiology	Pulmonary venous return → systemic venous circulation via embryological connection
Repair	• Total or symptoms: repair in infancy • Partial: can go unnoticed until adulthood
CXR	• Right atrium prominence • Right ventricle enlargement • Increased pulmonary vasculature
Echo	• Right atrial enlargement (RAE) • Right ventricular hypertrophy (RVH) • Superior/inferior vena cava dilation
Consider	• Scimitar syndrome • Recurrent bronchopulmonary infections



Acyanotic: Atrial Septal Defect

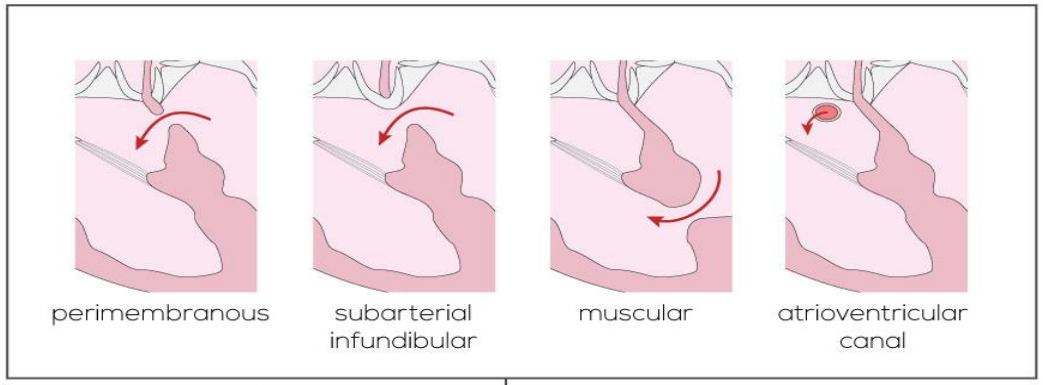
Atrial Septal Defect Pathophysiology
1. Poor growth or absorption of secundum septum (70-75% ASDs) • Familial (often isolated) • Associated with partial anomalous pulmonary venous return (PAPVR), pulmonary stenosis 2. Failure of primum septum to fuse with endocardial cushion (15-20% ASDs) • Associated with trisomy 21 3. Abnormalities in insertion of superior vena cava/inferior vena cava (5-10% ASDs) • Associated with partial anomalous pulmonary venous return (PAPVR) 4. Coronary sinus defect (<1% ASDs) • ”Unroofed” sinus due to absence of coronary sinus or atrial

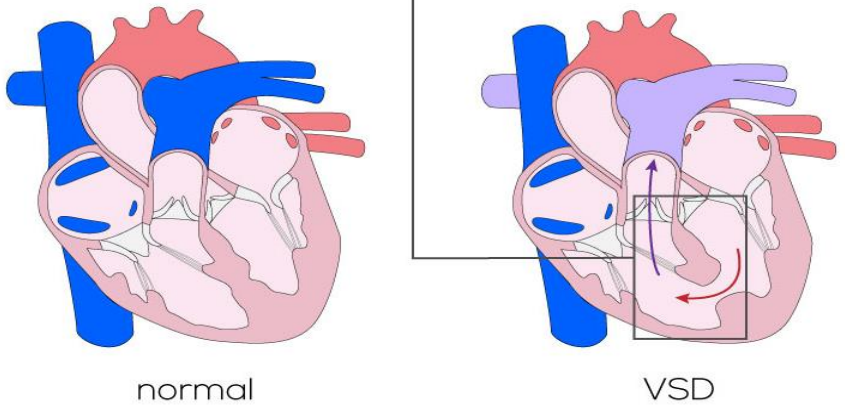


Acyanotic: Ventricular Septal Defect

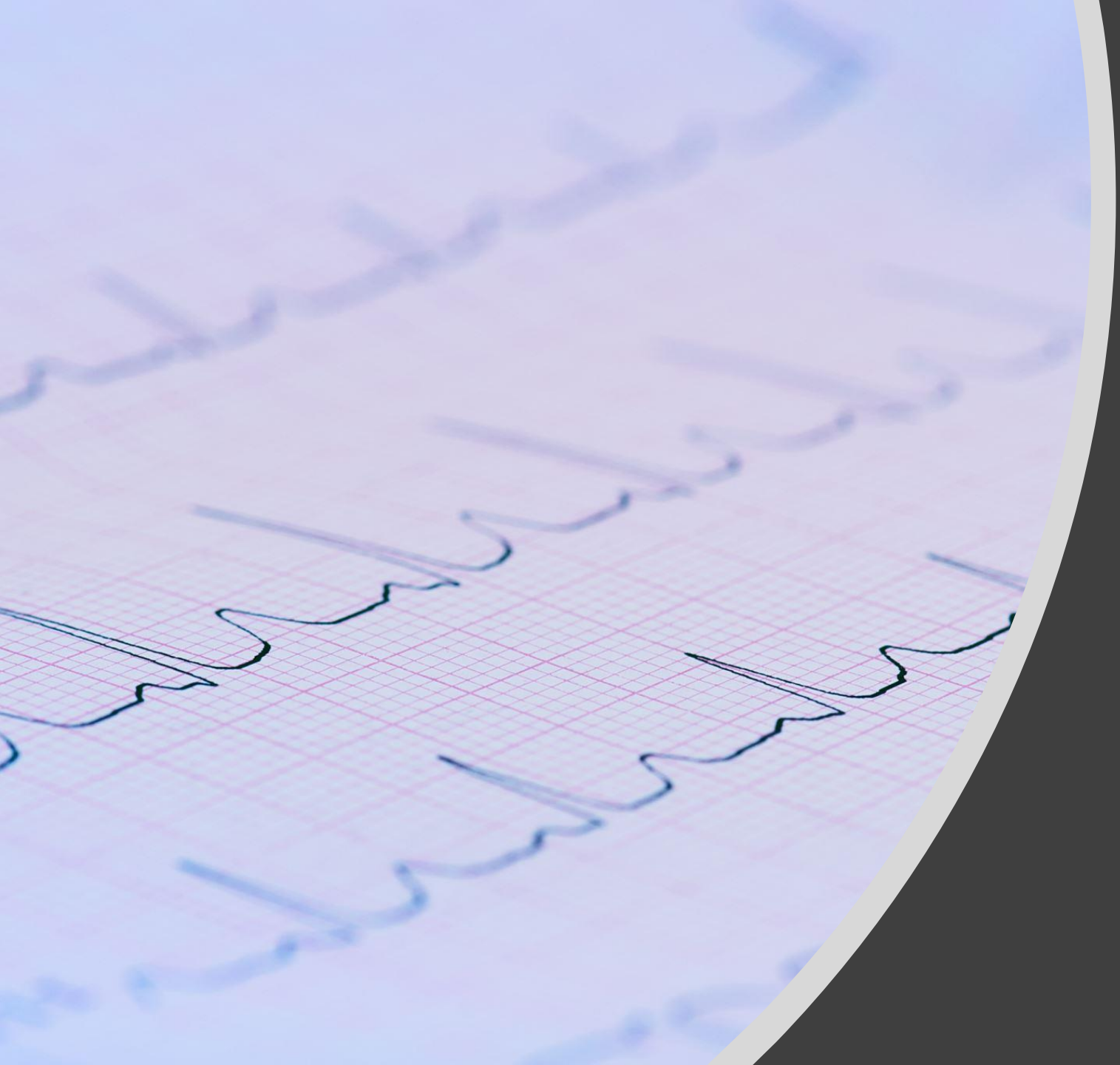
Ventricular Septal Defect (VSD) Pathophysiology
1. Perimembranous (75-80% VSDs) • Most close spontaneously • Post-op conduction defects 2. Muscular (10-15% VSDs) • Usually close spontaneously 3. Subarterial infundibular (5-7% VSDs) • Rarely self-resolve 4. Atrioventricular canal (5% VSDs) • Rarely isolated • Associated with defect of canal and endocardial cushions

Anatomy





Bloodflow



Contributors

Contributors

Editors in Chief
Priyanka Solanki
Harnoor Mann
Scott Baumgartner

Fellow/Faculty Editors
Michael Bashline
Joshua Levenson

Section Editors
Scott Baumgartner
Ronaldo Correa
Mohanad Hamadi
Talya Mandelkern
Harnoor Mann
Greg Olenginski
Amar Patel
Gautam Rangavajla
Valentina Jaramillo Restrepo
Balvindar Singh
Priyanka Solanki
Effimia Maria Zacharia

Article Authors
Anne Arnason
Scott Baumgartner
Ronaldo Correa
Michael Creager

Article Authors Continued
Anisha Das
Nick Faraci
Farid Farkouh
Mohanad Hamadi
Pranav Jain
Michael Lu
Talya Mandelkern
Harnoor Mann
Sarbajeet Nagdas
Greg Olenginski
Amar Patel
Gautam Rangavajla
Mark Rizko
Balvindar Singh
Priyanka Solanki
Nick Sumzin
Brad Very
Jessica Walker
Ben Zuchelkowski

THRID EDITION

The cardiology handbook needs your help for the third edition!

In putting together a relatively concise handbook that covers most cardiology topics, we undoubtedly left out (or put in too much) helpful information for some of the articles. We would welcome your input regarding how to improve the handbook. Additionally, as our evidence base grows and guidelines change, updates will be necessary. Please share your recommendations with the editors by emailing solankip3@upmc.edu, baumgartners2@upmc.edu, or mannhk@upmc.edu .

We also would encourage emails from individuals who are interested in taking over the handbook to facilitate a third edition.

Sincerely,
Priyanka, Scott, and Harnoor