# CARDIOLOGY

## CORONARY ARTERIES & EKG LEADS

<table>
<thead>
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<th>Main Coronary Artery</th>
<th>Main Branches</th>
<th>Additional Branches</th>
<th>Wall and Lead Correlation</th>
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</table>
| Right Coronary Artery (RCA) | | | SA & AV Nodes (in ~60% of people)  
Right atrium and ventricle  
Posterior 1/3 of IV septum  
Leads II, III, aVF (inferior) |
| | SA Nodal Artery | | SA Node |
| | R Marginal Artery (RMA) | | Inferior wall – leads II, III, aVF |
| | AV Nodal Artery | | AV Node |
| | Posterior Descending IV Artery (PDA)* | | Ventricular septum  
Posteroomedial papillary muscle  
*can be supplied by the RCA or LCx – this determines “dominance” |
| | Posterolateral Artery 1&2 | | |
| Left Coronary Artery (LCA) | Left Anterior Descending (LAD) aka Anterior IV artery | | Right & left bundle branches  
Anterior & Anteroseptal wall  
Leads V1-V4  
Diagonal Arteries 1 & 2 |
| | | | |
| | Left Circumflex (LCx) | | Lateral wall  
Leads V5-V6, and I & aVL (high lateral)  
*Supplies SA node in 38% of people  
Obtuse Marginal Branches 1 & 2 (OM1, OM2) |

![Diagram of EKG Leads](image)
AMI ECG, ANATOMY AND PATHOLOGY

RCA ‘TYPE’ LESIONS ±

INFERIOR MI
STE: II, III, aVF
STO: aVL (reciprocal STE)
RCA occlusion distal to RV
50% of MI

Seek and exclude
INFERIOR AND RV MI
STE: II, III, aVF and V1, V4R
RCA occlusion proximal to RV
40% of inferior MI
Increased mortality risk

INFEROLATERAL MI
STE: II, III, AVF and LAVL, V3, V6
aV4R
LAD and LCK occlusion
In a L dominant system

INFEROPosterior MI
STE: II, III, AVF and V7-9
STO: V4, V2 (reciprocal STE)
R5 ± 1/VI-2
TaT: V1-2
RCA and LCK occlusion

LAD LESIONS

Combinations of the following

SEPTAL MI
STE: V1-2
LAD occlusion

ANTERIOR MI
STE: V3, V4
LAD occlusion

LATERAL MI
STE: V5, V6, I, AVL
LAD occlusion

LCX LESIONS ±

POSTERIOR MI
STE: V7-9
STO: V1-2 (reciprocal STE)
R5 ± 1/VI-2
TaT: V1-2
RCA and LCK occlusion

Seek and exclude
POSTEROLATERAL MI
STE: V7-9 and LAVL, V5-6
STO: V1-2
LAD and LCK occlusion

INFEROPOSTERIOR MI
STE: II, III, AVF and V7-9
STO: V1-2 (reciprocal STE)
R5 ± 1/VI-2
TaT: V1-2
RCA and LCK occlusion
EKG QUICK TIPS & CLINICAL PEARLS

- **Brugada Syndrome = RsR' w/ STE in V1-V3** (susceptible to deadly arrhythmias, get ICD)
- **Wellen’s Syndrome = biphasic T waves → deep TWI in V2-V3** indicating critical LAD stenosis
  - If artery remains occluded, pt will develop acute anterior STEMI
- **QT interval should generally be no longer than ½ R--R interval**
- **STE in aVR with diffuse STD = L main artery CAD pattern**
- **Posterior MI - STD and large R in V1-V2**
- **RV infarct → may see hypotension, tachycardia, congestive hepatopathy → tx w/ fluids, nitro CI**
- **Do not give nitro to inferior wall STEMIs (d/t possible concomitant RV infarct)**
- **Broad complex irregular tachycardia at rapid rates w/ variable QRS morph → suspect AF w/ WPW**
- **AFib w/ WPW → procainamide (do NOT given AV nodal blocking agents)**
- **“Rabbit Ears” → taller LEFT rabbit ear = VT, taller RIGHT rabbit ear = RBBB**
- **Pericarditis → Diffuse concave STE w/ PR depressions (ST:T ratio >0.25, whereas BEN <0.25)**
- **Benign Early Repolarization (BER) = normal variant; localized or diffuse "concave up" STE**
- **HFrEF - BB >> CCB**
- **6 PVCs per min = pathological**
- **Run of 3+ PVCs in rapid succession = run of VTach (>30sec = sustained VT)**
- **Evaluate for P waves in leads I, II (upright), and V1 (biphasic)**
- **Axis (mean QRS vector) points toward ventricular hypertrophy & away from infarction**
- **Atrial hypertrophy - diphasic p wave in V1**
- **RVH - large R in V1 (w/ S smaller than R) that gets progressively smaller from V2, V3, etc.**
  - QRS >0.12s w/ 2 of the following: RAD, V1 w/ R>S or qR, or V1 w/ R>6mm or S<2mm
- **LVH - Cornell Criteria = R in avL + S in V3 >28mm in men, >20mm women**
  - May see repolarization abnormalities = ST/TW changes (STD typical in lateral leads)
- **LBBB → wide QRS >120ms, deep S in V1, big R in V6**
  - Tall R in V5-6 + concave STE changes (V1-2?) + Tw in opposite direction of QRS vector → LBBB
- **RBBB → wide QRS >120ms, (RsR') rabbit ears in V1, slurred/deep S in V5-6**
- **Low Voltage = <5mm in limb leads OR <10mm in precordial leads (R+S) → effusion, COPD, infiltrative dz**
- **Causes of R waves in V1 (abnormal) = posterior MI, RVH, RBBB, Brugada**
- **Digitalis tox = atrial & junctional premature beats, sinus block, AV blocks, paroxysmal atrial tachycardia w/ AV block** (rapid spiked P’s w/ 2:1 P:QRS ratio) → marked dig tox can cause PVCs and VT, VF
- **Beta-blocker overdose tx → glucagon**
- **Hyperkalemia → widened QRS, peaked T, may see loss of P waves**
- **Hypokalemia → flat or inverted T wave (U wave may appear)**
- **Hypercalcemia → short QT**
- **Hypocalcemia → prolonged QT**
- **Bipolar leads: RA = (-,-), LA = (+,-), LL = (+,+)**
- **ESRD on HD w/ AFib require anticoagulation → warfarin* >> NOACS**
  - NOACs excreted ~80% renally but Eliquis ~25% renal excretion
- **LVEDP >25mmHg → furosemide**
- **Non-ischemic CM has many causes (including LBBB)**
  - IABP fxn - inflates during diastole and deflates in systole, thereby increasing coronary perfusion during diastole and decreasing myocardial O2 demand
- **NO NSAIDS post-MI**
- **Hypotension 2/2 LV outflow tract obstruction may be seen with large anterior MI, takotsubo, decompensated HCM → levophed can make this worse, use alpha-agonists like phenylephrine**
- **Antiplatelets for ACS/post-Cl → Thienopyridines (P2Y12/ADP receptor inhibitors): Clopidogrel (pro-drug for which 15% people are non-converters, Ticagrelor (faster onset of action, expensive), Prasugrel (higher bleeding risk, BBW for stroke hx, age >75, weight <60)**
EKGS - ISCHEMIA & INFARCT (lecture notes)

- General Evaluation:
  - STE and STD - compare ST segment to TP segment (not PR)
  - T waves – peaked, flattened, inverted?
  - Pathologic Q waves = >1mm deep and >40ms duration

- Coronary Arteries and EKG leads
  - RCA – inferolateral wall (II, III, aVF)
  - LAD – anterior and anteroseptal wall (V1-V4)
  - LCx – lateral wall (V5-6), (high lateral (I, aVL)
  - PDA – supplied by RCA in ~80%, LCx in ~20%, posterior wall

- NSTEMI = subtotal occlusion of vessel → subendocardial injury (often have STD/TWI)
  - Infarction (STD and TWI) → Fibrosis (ST returns to baseline, but TWI persists)

- STEMI = total occlusion of coronary artery → transmural injury
  - Infarction – STE & appearance of Q-waves
  - Late infarction – ST segments and T waves return to normal, Q waves persist
  - Evolution of STEMI: Hyperacute T waves → STE w/ convex ST shape → STE + biphasic T waves → pathologic Q waves
  - EKG Criteria (though consider clinical picture):
    - Limb leads: STE ≥1mm in at least 2 [anatomically] contiguous leads
    - Precordial leads: STE equal to or ≥2mm in at least 2 contiguous lead
    - STD in reciprocal leads to the territory that shows STE
  - Posterior MI – STE in II, III, aVF and STD V1-V2, large R in V1

- Inferior Wall MI
  - STE in Leads II, III, avF (RCA)
  - RCA feeds AV & SA nodal arteries, R marginal artery
  - Complications: beware of hypotension, new murmur, heart block
    - High degree heart block (given ischemia of AV nodal artery)
    - RV infarction (R ventricular artery arises from RCA) - can cause hypoTN from decreased preload
    - Acute mitral regurge (papillary muscle rupture)
    - Rupture of L ventricular free wall
    - Rupture of IV septum
  - Do NOT give nitroglycerine - hypotension, pressures can bottom out

WIDE COMPLEX TACH & VENTRICULAR RHYTHMS (lecture notes)

- PVCs
  - Early QRS originating from ventricles
  - Wide complex, abnormal from other beats, not preceded by P wave
  - 3 PVCs can lead to fusion beats → non-sustained VT!
  - Can be unifocal or multifocal
  - Can be isolated, couplets, triplets → Bigeminy and trigeminy (every other or every 3rd beat is a PVC)
  - Treatment for PVCs = magnesium
  - High PVC burden over time can cause heart burden
● **VT**
  ○ Electrical activity driven by a focus in the R or L ventricle
  ○ Sustained VT lasts >30sec
  ○ Rate often 160-240
  ○ Wide complex tachycardia >160bpm → think VT
  ○ Torsades – usually 200+bpm, seen in all leads
  ○ Polymorphic (torsades) usually related to ischemia or iatrogenic (drugs)
  ○ Monomorphic usually related to scar

● **VF**
  ○ Uncoordinated ventricular activity, rate >240bpm
  ○ Terminal rhythm
  ○ Wide Complex Tachycardia and VT vs. SVT w/ aberrancy

● **DDx for WCT** = VT, SVT w/ aberrancy, AVNRT (antidromic, SVT w/ pre-excitation, WPW)

● Favors VT:
  ○ **AV dissociation** (may see cannon a waves, irregular intensity of 1st heart sound)
  ○ **RAD**
  ○ **Electrical Concordance** (all QRS complexes in precordial leads pointing in same direction; negative is more specific for VT but can be positive)
  ○ **Absence of RS complex in precordial leads**
  ○ Positive QRS complex in avR
  ○ Capture & fusion beats
  ○ VT is typically regular

● Favors SVT: uniformity of RR

● Grossly irregular WCT likely represents AFib w/ aberrant conduction or polymorphic VT

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**CARDIOMYOPATHY**

● Dilated CM → LV dilation and systolic dysfunction
  ○ Causes: EtOH, Aflutter, Afib, thyroid dysfunction, pregnancy, HIV, SLE, Beriberi, cocaine

● Restrictive CM → diastolic dysfunction w/o evidence of dilation or hypertrophy of LV
  ○ Causes: amyloidosis, sarcoidosis, hemochromatosis, idiopathic

● Hypertrophic CM → hypertrophy of ventricles (caused by sarcomere defect)
  ○ Genetic; m/c cause of SCA in young athletes

● Dx: Echo and EKG (HCM - L axis deviation and LV hypertrophy)

● Tx: HCM → CCB and beta blockers vs. surgery (when LV outflow tract obstruction not managed with meds)
CORONARY ARTERY DISEASE & ACUTE CORONARY SYNDROMES (ACS)

- Chest Pain ddx: ACS, pericarditis, myocarditis, aortic dissection, pneumonia, pleuritis, PTX, PE, GERD, esoph spam, PUD, biliary dz, pancreatitis, costochondritis, anxiety, shingles, prinzmetal’s

- Stable Angina
  - S/sx: retrosternal chest pain, squeezing or pressure, gradual radiation to arms/neck/jaw; associated dyspnea, dia phoresis, N/V
  - Dx: EKG (STD, TWI, Qw), troponins (check @ baseline + 3-6hrs after onset)
  - Tx: modify risk factors, nitrates, CCB, BBs, tx contributing dz

- Unstable Angina (ACS)
  - Sx: new onset angina, angina with minimal exertion, worsened angina, angina at rest
  - Dx: EKG (STD, TWI, Qw), (-) troponins, f/u stress test, f/u cardiac cath if high risk
  - Tx: MONAB - morphine, O2, nitro, aspirin 325mg, BB, +/- heparin

- NSTEMI (ACS)
  - Signs of ACS: S3/S4, signs of CHF, bruits, mitral regurg (ischemia of papillary muscle)
  - Dx: ↑ Troponin I, ↑ CK-MB. EKG (STD, TWI, Qw), stress test if mod risk, cath if high risk
  - Tx: MONAB - morphine, O2, nitro, aspirin 325mg, BB, +/- heparin

- STEMI (ACS)
  - Sx: angina at rest
  - Dx: ↑ Troponin I, ↑ CK-MB. EKG w/ STE ≥1mm or Q waves in 2+ contiguous leads
    - Echo → hypokinesis or akinesis in area supplied by occluded vessel
  - Tx: aspirin 325mg, nitrates, O2, BB, morphine, heparin
  - Reperfusion → PCI (angioplasty within 90min of arrival) >> fibrinolytics (IPA)

- General ACS Treatment → control risk factors & maintain perfusion
  - Primary prevention: aspirin + statin (statins stabilize vulnerable plaque)
  - Initial treatment ("MONA") → Morphine, Oxygen, Nitrates, aspirin
  - Anti-anginal therapy → nitrates, morphine, BB (IV lopressor q5min x3)
  - Antiplatelets → aspirin + clopidogrel (Plavix), Prasugrel, Ticagrelor, or IIbIIIa GPI
  - Anticoagulants → LMWH (Lovenox), UFH, DTI (bivalirudin), Xa inh (Fondaparinux)
  - Meds that improve mortality post-MI (d/c meds) → ASA, statins, BB, ~ACEI

- TIMI Risk Score
  - Age 65+ yo
  - 3+ CAD risk factors (DM, HTN, HLD, smoking, Fhx of early MI)
  - Prior coronary stenosis >50%
  - ST deviation on admission
  - 2+ anginal episodes in prior 24hrs
  - Elevated serum cardiac biomarkers
  - Use of aspirin in prior 7days
  - 0-2 = low risk  3-4 = intermediate risk → stress test  5-7 = high risk → cardiac cath

- Stress tests
  - “Stress” options → exercise vs. pharmacologic
    - Vasodilators i.e. regadenoson, adenosine (coronary steal principle)
    - Inotropes i.e. dobutamine
  - “Analysis” options → EKG, echo, nuclear test
    - Nuclear if used intrope - most common, avoid caffeine 24hrs prior
    - Echo if used vasodilator - less useful if known wall abnormality, AS
    - EKG less useful if paced, LBBB, LVH, baseline STD, WPW, digoxin use
CHF

- Precipitants of Acute HF: *dietary indiscretion (chinese food!), *med noncompliance or changes, ischemia/MI, arrhythmias, renal failure, HTN crisis, drugs, EtOH, recent illness/infx, COPD, PE
- Precipitating dz: HTN, valvular dz, CM, ischemic heart dz, arrhythmias, SLE, hemochromatosis, sarcoidosis, cocaine abuse, alcohol abuse
- Systolic dysfunction → dilated LV w/ impaired contractility
- Diastolic dysfunction → impaired LV relaxation & filling (stiffness, hypertrophy, AV stenosis)
- Left-sided (pulm edema) vs. R-sided (peripheral edema, JVD, hepatomegaly)
- Reduced (HRrEF) vs. Preserved (HRpEF) LV EF%
- Sx related to decreased CO: fatigue, weakness, DOE / exercise intolerance, AMS, anorexia
- Sx related to fluid retention / congestion (response to ↓ CO): edema, SOB, PND, orthopnea, wheezing, hemoptysis, RUQ discomfort, abdominal distension, satiety
- Signs: crackles, S3 gallop, peripheral edema, displaced PMI, tachy, JVD, HSM, HJ reflux, ascites
- Diagnosis & Eval: (clinical diagnosis)
  - Physical Exam: congestion (dry vs. wet) and perfusion (warm vs. cold)
  - Labs: BNP >500 (n=<100), increased SCR / low GFR, serum Na <130, TSH
  - Assess for decreased organ perfusion: inc. Cr, dec Na, abnl LFTs
  - Echo → assess EF% and chamber size
    - EF <40% seen in systolic dysfxn (n= >55%; EF preserved in diastolic)
  - EKG: ST-T wave changes
  - CXR: pleural effusion, pulm edema, pulm vasc congestion & cephalization, cardiomegaly
- Tx of Acute Decompensated HF: (LMNOP)
  - Lasix / diuretics with monitoring of urine output and weight (give same dose IV as PO)
    - IV: PO ratio is 1:2 but you want to double PO dose so keep same (if on 40mg PO lasix at home, give 40mg IV in hospital so you are doubling dosage)
  - Morphine (helps sx, venodilator, decreases afterload)
  - Nitrates (vasodilators i.e. nitroglycerin, nitroprusside) decrease preload
  - Oxygen
  - Positioning (sitting with legs dangling)
  - Telemetry, monitor fluids & lytes, weight pt daily, control HTN
  - Continue same home meds (unless hypotensive) but don’t start on new BB for acute sx
  - Inotropes (dobutamine, dopamine) if pt is wet (tx in CCU)
  - If hypoxemic → PPV (CPAP or BiPAP)
  - If advanced, consider PAC, IV vasodilators, inotropes, IABP, LVAD, transplant
- Therapies for Chronic HF with Reduced EF: (* ↓ mortality)
  - Maintenance Loop (Furosemide) +/- Thiazide (HCTZ) diuretics: ↓ decrease preload
  - *ACEIs (Enalapril, etc.): ↓ preload & afterload, ↓ mortality
  - *BBs (Carvedilol, Metoprolol succinate): ↓ HR, ↓ mortality
  - *Aldosterone antagonists (Spironolactone): ↓ fluid retention & preload, arrhythmias, BP
  - *Cardiac Devices: CRT, ICD
  - Digoxin (good if comorbid AFib and hypotension)
  - Hydralazine & nitrates: ↓ afterload (arteriolar vasodilator)
  - For rate control in comorbid AFib → BB, CCB
  - Statins ONLY if hx of MI or ACS
AFIB & ANTICOAGULATION

- Can be paroxysmal, persistent (>7d), permanent (>1yr), lone (no structural dz)
- Eval: EKG, echo, TSH (r/o thyrotoxicosis), CHADS2 score (eval for risk of stroke)
  - TTE to eval for structural heart dz
  - TEE - identify thrombi, thus need for anticoagulation before cardioversion
- Tx Strategies:
  - Rate OR Rhythm control
    - Rate control (slow conduction across AV node) = BB, CCB, digoxin
      - Ex: Metoprolol Tartrate (Lopressor 50mg)x2-3 tries → Diltiazem → Digoxin
      - Can use IV lopressor 100mg prn HR >120bpm
      - Diastolic HF w/ Afib can immediately benefit from beta blocker
      - Digoxin useful if hypotensive and tachycardic
    - Rhythm control = electrical or pharmacologic cardioversion
      - antiarrhythmics, ablation, surgery
  - Antithrombotic therapy to prevent emboli (CHA2DS2-VASc Score)
    - AC before and after cardioversion!
    - CHADS2 Score = CHF, HTN, Age ≥75yo, DM, Stroke/TIA
      - CHADS2 0: aspirin 325mg daily
      - CHADS2 1: aspirin or warfarin
      - CHADS2 2+: anticoagulation
    - Example maintenance regimen: Cardizem, metoprolol, xarelto
  - Anticoagulant therapies
    - DTIs (direct thrombin / II inhibitors): Dabigatran (Pradaxa)
    - Factor Xa inhibitors: Rivaroxaban (Xarelto), Apixaban (Eliquis)
      - Fondaparinux is an indirect Xa inhibitor
    - Vitamin K antagonists: Warfarin
    - Heparin
- INR target in AFib = 2-3 (increase dose if low, decrease dose if high)
TROPNON

- Regulatory proteins that control the calcium-mediated interaction of actin and myosin
- High sensitivity to detect small amounts of myocardial necrosis
- Normal level: <0.003
- Troponin release occurs when there has been damage to the myocardium secondary to myocardial ischemia, trauma, or indeterminate causes
- Troponin concentrations rise 2-3 hours after onset of acute MI
- Gold standard is to repeat q6hrs
  - Get 2 troponins, 3-6hrs apart
  - First trop should be at least 6hrs after sx onset
  - Early repeat (1-2hrs) is useful to rule IN MI
- Sticks around for 10-14 days
- Causes of elevated troponin in absence of ACS: Heart failure/CHF, Heart block, Sepsis, Trauma, Rhabdo, Aortic dissection, Myocarditis, Drug abuse, PE

VALVULAR DISEASE & HEART SOUNDS

- S1 (AV valve closure) and S2 (SLV closure)
- S3 = ventricular gallop → early diastole, low pitch (rapid ventricular filling)
  - Volume overload → CHF, pregnancy
- S4 = atrial gallop → late diastole (decreased ventricular compliance, stiff ventricles)
  - Pressure overload → HTN, hypertrophic cardiomyopathy
- Aortic Stenosis
  - Normal aortic valve area = 3-4cm → critical AS is <0.8cm
  - Common causes: bicuspid valve, degenerative changes w/ aging, rheumatic fever
  - Sx: angina, CHF sx, syncope
  - Signs: n-low BP, parvus et tardus, crescendo-decrescendo systolic murmur @ R 2nd ICS and radiation to neck
  - Tx: nothing if asymptomatic, surgery if symptoms

MURMUR GRADING
**ENDOCARDITIS** = localized infx of endocardium d/t vegetations from colonized bacteria/fungus/virus

- Acute (often normal valves w/ virulent organism) vs. Subacute (often abnormal valves)

**Risk Factors:**
- Abnormal valves (prosthetic valves or congenital valve defects)
- Abnormal risk of bacteremia: IVDU, indwelling venous catheters, poor dentition, hemodialysis, DM, intracardiac devices

**Common orgs:** S. viridans (SBE), S. aureus (ABE, IVDU -esp MRSA), S. epidermis (PVE), enterococci/GNR (GI/GU procedures), HACEK organisms

**Modified Duke Criteria → 2 major OR 1 major + 3 minor criteria OR 5 minor** (definitive)
  - Major criteria:
    - Sustained bacteremia by an organism known to cause endocarditis (or coxiella)
    - *Endocardial involvement* documented by either +endocardiogram (vegetation, abscess, prosthetic dehiscence) or *new valvular regurgitation*
  - Minor criteria
    - Fever
    - Predisposing condition
    - Vascular phenomena → septic arterial or pulmonary emboli, Janeway lesions, mycotic aneurysms, ICH
    - Immune phenomena → +RF, GN, osler's nodes, roth spots
    - +BCx not meeting major criteria

**Clinical manifestations:** fever, fatigue, chest pain, neuro complaints, weight loss
  - Persistent bacteremia (fever), valvular infx (CHF, dysrhythmias), septic emboli (PE, stroke, MI), immune complex phenomena (arthritis, +RF, ↑ESR)

**Exam:** heart murmur, petechiae, roth spots, janeway lesions (macules on palms/soles), osler nodes (tender nodules on finger pads), arthritis

**Labs:** Blood Cx x3 (before abx!), CBC, ESR/CRP, RF, BUN/Cr, UA & UCx

**Imaging:** EKG, Echo (TTE if low suspicion, TEE greater sensitivity)

**Tx:** duration of therapy ~4-6wks
  - native valve ABE → **nafcillin + gent OR vanco**
  - Native valve SBE → **PCN/amp + gent**
  - prosthetic valve → vanc + gent + [cefepime or rifampin]
  - IVDU→ vanco
  - Surgery indication: severe valve dysfunction, refractory CHF, uncontrolled infx, PVE

**Monitor for complications:** CHF, conduction block or arrhythmias, new emboli, drug rxns

**PERICARDITIS**

- Causes: Infx* (coxsackie virus), idiopathic*, lupus/rheum dz, CA, radiation, post-MI
- S/sx: pleuritic/inspirational chest pain relieved when leaning forward, low fever, SOB
- Exam: pericardial friction rub (sit & lean forward), sinus tach, high WBC, high ESR/CRP, diffuse STE on EKG, echo may show pericardial effusion
- Tx: tx underlying cause, pain control (NSAIDs)
- Complication = cardiac tamponade d/t pericardial effusion → dx w/ echo
  - Beck’s triad = JVD, hypotension, muffled heart sounds
  - Other s/sx: SOB, narrow pulse pressure, pulsus paradoxus, tachycardia, EKG w/ low QRS voltage, total electrical alternans
○ Tx: pericardiocentesis if unstable; fluids, inotropes (dopamine)

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<th>ARRHYTHMIAS QUICK REFERENCE</th>
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<td><strong>Supraventricular rhythms with normal rate → narrow QRS</strong></td>
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<td><strong>Sinus arrhythmia</strong></td>
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<td><strong>PACs</strong></td>
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<td><strong>2nd degree AV Block – Mobitz II</strong></td>
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<td><strong>3rd degree AV Block</strong></td>
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<td><strong>RBBB</strong></td>
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<td><strong>LBBB</strong></td>
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<tr>
<td><strong>SVTS → HR &gt;100, narrow or wide complex</strong></td>
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<td>Condition</td>
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<td><strong>Ventricular Arrhythmias → wide QRS ≥ 120ms</strong></td>
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<td>Polymorphic VT</td>
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**RESOURCES & LINKS**
- Mitral Regurg → Mitral Clip: [http://mitraclip.com/the_mitraclip_procedure](http://mitraclip.com/the_mitraclip_procedure)
- EKG practice: [https://ecg.bidmc.harvard.edu/maven/displist.asp?ans=0](https://ecg.bidmc.harvard.edu/maven/displist.asp?ans=0)
- Life in the Fast Lane: [https://lifeinthefastlane.com/ecg-library/basics/diagnosis/](https://lifeinthefastlane.com/ecg-library/basics/diagnosis/)
### Guidelines for choice of pacemaker generator in selected indications for pacing

<table>
<thead>
<tr>
<th>Type of pacemaker</th>
<th>Sinus node dysfunction</th>
<th>AV block</th>
<th>Neurally-mediated syncope or carotid sinus hypersensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single-chamber atrial</td>
<td>No suspected abnormality of AV conduction and not at increased risk for future AV block</td>
<td>Not appropriate</td>
<td>Not appropriate (unless AV block systematically excluded)</td>
</tr>
<tr>
<td></td>
<td>Maintenance of AV synchrony during pacing desired</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rate response available if desired</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single-chamber ventricular</td>
<td>Maintenance of AV synchrony during pacing not necessary</td>
<td>Chronic atrial fibrillation or other atrial tachyarrhythmia or maintenance of AV synchrony during pacing not necessary</td>
<td>Chronic atrial fibrillation or other atrial tachyarrhythmia</td>
</tr>
<tr>
<td></td>
<td>Rate response available if desired</td>
<td>Rate response available if desired</td>
<td>Rate response available if desired</td>
</tr>
<tr>
<td>Dual-chamber</td>
<td>AV synchrony during pacing desired</td>
<td>AV synchrony during pacing desired</td>
<td>Sinus mechanism present</td>
</tr>
<tr>
<td></td>
<td>Suspected abnormality of AV conduction or increased risk for future AV block</td>
<td>Atrial pacing desired</td>
<td>Rate response available if desired</td>
</tr>
<tr>
<td></td>
<td>Rate response available if desired</td>
<td>Rate response available if desired</td>
<td></td>
</tr>
<tr>
<td>Single-lead, atrial-sensing ventricular</td>
<td>Not appropriate</td>
<td>Normal sinus node function and no need for atrial pacing</td>
<td>Not appropriate</td>
</tr>
<tr>
<td></td>
<td>Desire to limit number of pacemaker leads</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>