CARDIOLOGY

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BASIC CLINICAL CARDIAC EXAM

CARDIAC HISTORY

- ❑ coronary artery disease: chest pain (CP) (location, radiation, duration, intensity, activities associated with onset; alleviating factors (associated with rest, NTG)
- ❑ heart failure: fatigue, presyncope
  - left-sided symptoms: decreased exercise tolerance, shortness of breath on extertion (SOBOE)/chest pain on exertion (CPOE)
  - right-sided symptoms: paroxysmal nocturnal dyspnea (PND)/orthopnea, SOB at rest, ascites,
- ❑ Arrhythmia: presyncopal/syncopal episodes, palpitations
- ❑ Baseline function: exercise tolerance (# flights of stairs/blocks), need for nitroglycerin (NTG), symptoms during low impact activities/daily activities (combing hair, showering) or at rest

FUNCTIONAL CLASSIFICATION OF CARDIOVASCULAR DISABILITY

Table 1. Canadian Cardiovascular Society (CCS) Functional Classification

Class	Function
I	ordinary physical activity does not cause angina; angina only with strenuous or prolonged activity
II	slight limitation of physical activity; angina brought on at > 2 blocks on level (and/or by emotional stress)
III	marked limitation of physical activity; angina brought on at ≤ 2 blocks on level
IV	inability to carry out any physical activity without discomfort; angina may be present at rest

Table 2. New York Heart Association (NYHA) Functional Classification

Class	Function
I	ordinary physical activity does not evoke symptoms (fatigue, palpitation, dyspnea, or angina)
II	slight limitation of physical activity; comfortable at rest; ordinary physical activity results in symptoms
III	marked limitation of physical activity; less than ordinary physical activity results in symptoms
IV	inability to carry out any physical activity without discomfort; symptoms may be present at rest

CARDIAC EXAMINATION

General Examination

- ❑ Skin – peripheral vs. central cyanosis, clubbing, splinter hemorrhages, Osler's nodes, Janeway lesions
  brownish-coloured skin – hemochromatosis
- ❑ Eyes – conjunctival hemorrhages, Roth spots, emboli, copperwire lesions, soft/hard exudates

Blood Pressure (BP)

- ❑ should be taken in both arms with the patient supine and upright
- ❑ be wary of calcification of the radial artery in the elderly as it may factitiously elevate BP (Osler's sign)
- ❑ orthostatic hypotension - postural drop > 20 mm Hg systolic or > 10 mm Hg diastolic
  - increased HR > 30 bpm (most sensitive - implies inadequate circulating volume)
    patient unable to stand - specific sign for significant volume depletion
- ❑ pulse pressure(PP) (PP = systolic BP (SBP) - diastolic PB (DBP))
  - wide PP: increased cardiac output (CO) (anxiety, exercise, fever, thyrotoxicosis, AR, HTN), decreased total peripheral resistance (TPR) (anaphylaxis, liver cirrhosis, nephrotic syndrome, AVM)
  - narrow PP: decreased CO (CHF, shock, hypovolemia, acute MI, hypothyroidism, cardiomyopathy), increased TPR (shock, hypovolemia), valvular disease (AS, MS, MR), aortic disease (e.g. coarctation of aorta)
- ❑ pulsus paradoxus (inspiratory drop in SBP > 10 mmHg): cardiac tamponade, constrictive pericarditis, airway obstruction, superior vena cava (SVC) obstruction, COPD (asthma, emphysema)

The Arterial Pulse

- ❑ remark on
  - rate, rhythm, volume/amplitude, contour
  - amplitude and contour best appreciated in carotid arteries
- ❑ pulsus alternans - beat-to-beat alteration in PP amplitude with cyclic dip in systolic BP; due to alternating LV contractile force (severe LV dysfunction)
- ❑ pulsus parvus et tardus – slow uprising of the carotid upstroke due to severe aortic stenosis (AS)
- ❑ pulsus bisferiens – a double waveform due to AS + AR combined
- ❑ spike and dome pulse – double carotid impulse due to hypertrophic obstructive cardiomyopathy (HOCM)

Precordial Inspection
❑ observe for apex beat, heaves, lifts

Precordial Palpation
❑ apex - most lateral impulse
❑ PMI - point of maximal intensity
❑ location: normal at 5th intraclavicular space (ICS) at midclavicular line (≤10 cm from midline), lateral/inferior displaced in dilated cardiomyopathy (DCS)
❑ size : normal is 2-3 cm in diameter, diffuse > 3 cm
❑ duration: normal is <1/2 systole (duration > 2/3 systole is considered sustained)
❑ amplitude (exaggerated, brief - AR, MR, L to R shunt)
❑ morphology (may have double/triple impulse in HOCM)
❑ abnormal impulses
- palpable heart sounds (e.g. S1 in MS, P2 = pulmonary artery (PA) pulsation, S3, S4)
- left parasternal lift (right ventricular enlargement (RVE), left atrial enlargement (LAE) , severe left ventricular hypertrophy (LVH))
- epigastric pulsation (RVH especially in COPD)
- thrills (tactile equivalents of murmurs) over each valvular area

Clinical Pearl
❑ **Left parasternal lift - DDx - RVH (with pulmonary hypertension (HTN), LAE (secondary to severe MR), severe LVH, rarely thoracic aortic aneurysm.**

Auscultation - Heart Sounds
❑ S_1
- composed of audible mitral (M1) and tricuspid (T1) components
- may be split in the normal young patient

❑ if S_1 is loud
- short PR interval
- high left atrial (LA) pressure (e.g. early MS)
- high output states or tachycardia (diastole shortened)

❑ if S_1 is soft
- first degree AV block
- calcified mitral valve (MV) (e.g. late MS)
- high LV diastolic pressures (e.g. CHF, severe AR)
- occasionally in MR

❑ if S_1 varies in volume
- AV dissociation (complete AV block, ventricular tachycardia (VT))
- AFib

❑ S_2
- normally has 2 components on inspiration: A_2 and P_2
- normal splitting of S2 (A2 < P2) should vary with respiration

Exp.	Insp.	
S_2	A_2 P_2	normal • increased venous return to right side of heart with inspiration results in delayed closure of pulmonary valve (PV) (widens split)
A_2 P_2	A_2 P_2	wide fixed splitting • atrial septal defect (ASD)
S_2	A_2 P_2	widened splitting (delayed RV or early LV emptying) • right bundle branch block (RBBB), pulmonary HTN, MR, ventricular septal defect (VSD)
P_2 A_2	S_2	paradoxical splitting (delayed LV or early RV emptying) • left bundle branch block (LBBB), tight AS, systemic HTN, LV fib, paced rhythm, tricuspid regurgitation (TR), Wolfe Parkinson White (WPW)

❑ soft S_2
- aortic (A_2) or pulomonic (P_2) stenosis

❑ loud S_2
- systemic (A_2) or pulmonary HTN (P_2)

❑ soft heart sounds
- low cardiac output
- obesity
- emphysema
- pericardial effusion ("muffled" = tamponade)

❑ S3 (see Figure 1): volume overloaded ventricle
- occurs during period of rapid ventricular filling
- low frequency - best heard with bell at apex
- causes
  - may be normal in children and young adults (age < 30)
  - LV failure (systolic dysfunction, acute MI)
  - rapid ventricular filling (MR or high output states), RV S_3 (TR, MS, RV failure)
- DDx - split S_2, opening snap, pericardial knock, tumour plop

❑ S_4 (Figure 1): pressure overloaded ventricle (decreased capacitance, increased contribution of atrial kick to ejection fraction (EF))
- occurs during atrial contraction
- best heard with bell at apex
- always pathological (associated with diastolic dysfunction), ischemia (ventricular relaxation needs ATP), hypertrophy (HTN, AS, HCM), RCM, RV S_4 (pulmonary HTN, PS)
- DDx - split S_1, ejection clicks, prolapse clicks

Extra Sounds
- ❑ opening snap - early-diastolic (see Figure 1) - MS (A_2-opening snap (OS) interval shortens as MS worsens)
- ❑ ejection clicks (AS, PS)
- ❑ non-ejection mid-systolic clicks (mitral and tricuspid valve prolapse (MVP/TVP))
- ❑ pericardial (friction) rub: pericarditis, triphasic - ventricular systole, ventricular diastole and atrial systole ("scratchy" sound, like velcro)
- ❑ tumour plop

Auscultation - Murmurs
- ❑ Classification: timing (systolic/diastolic), location, radiation, intensity (grade murmurs I-VI), shape, pitch (quality), variation with respiration or maneuvers
- ❑ presence or absence of accompanying thrills, association with extra heart sounds

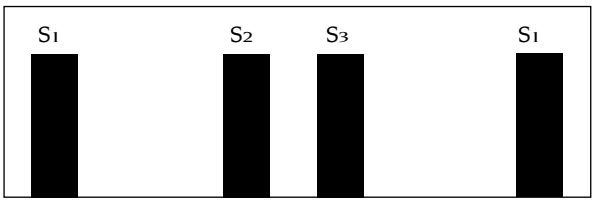
Clinical Pearl
- ❑ **Inspiration augments all right-sided murmurs and sounds, except pulmonary ejection click.**
- ❑ **Expiration augments AR (heard best on full exhalation, sitting leaning forward).**

- ❑ postural maneuvers
  - left lateral decubitus (LLD) for MS, S3, S4
  - squatting to standing position (MVP, hypertrophic obstructive cardiomyopathy (HOCM))
  - passive leg elevation (MVP, HOCM)

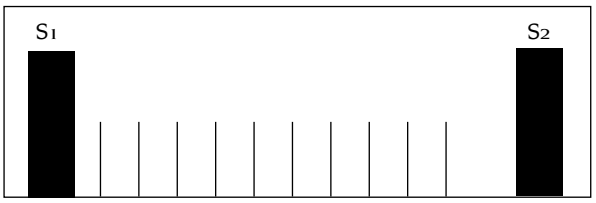
Table 3. Maneuvers for Auscultation of Heart Murmurs

Maneuvers	• Quiet inspiration • Sustained abdominal pressure	• Transient arterial occlusion (using 2 sphygmomanometers) • Fist clenching	• Standing to squatting • Passive leg elevation	• Valsalva
Physiological Effect	↑ venous return	↑ systemic arterial resistance	↑ venous return ↑ systemic arterial resistance	↓ venous return ↑ systemic arterial resistance
Effect on Intensity of the Mummer	• ↑ right-sided murmurs • ↑ TR • ↑ PS	• ↑ left-sided murmurs • ↑ MR • ↑ VSD	• ↓ HCM • ↓ MVP	• ↓ AS

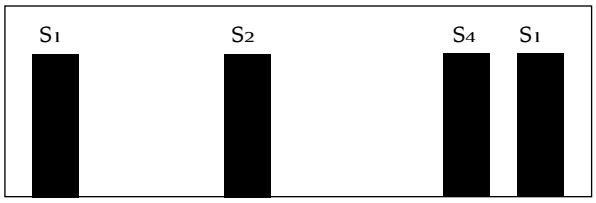
- ❑ systolic "ejection" murmurs (see Figure 1)
  - diamond-shaped, crescendo-decrescendo
  - outflow obstruction: AS, HOCM, PS
  - high output or "flow" murmurs
    - anemia
    - thyrotoxicosis
    - pregnancy
    - arteriovenous fistula
    - children
    - fever
- ❑ pansystolic murmurs (see Figure 1)
  - require a sustained pressure difference throughout systole
    - MR
    - TR
    - VSD
- ❑ high-pitched diastolic decrescendo murmurs (see Figure 1)
  - AR
  - PR
- ❑ low-pitched diastolic murmurs (mid-diastolic rumble) (see Figure 1)
  - MS
  - TS
  - severe AR may produce Austin Flint murmur
- ❑ high flow murmurs (result from 'relative' stenosis)
  - MR, persistent ductus arteriosus (PDA), VSD (increased left atrial (LA) filling)
  - ASD (increased right atrial (RA) filling)
- ❑ continuous murmurs (see Figure 1)
  - PDA
  - mammary souffle - goes away with pressure on stethoscope
  - coronary arteriovenous fistula
  - venous hum
    - due to high blood flow in the jugular veins
    - heard in high output states



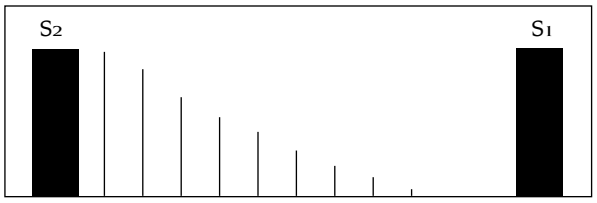
S3



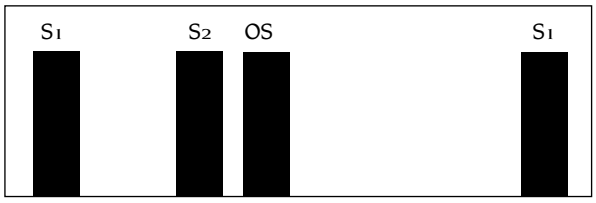
Pansystolic Murmur



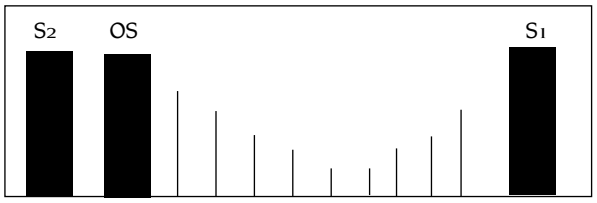
S4



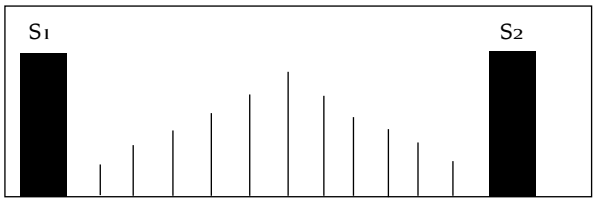
High Pitched Diastolic Murmur



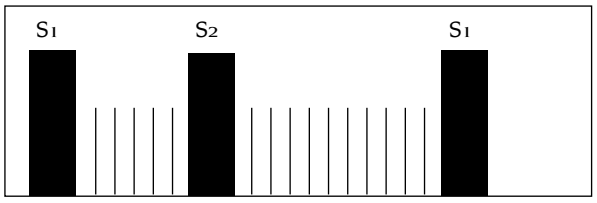
Opening Snap



Low Pitched Diastolic Murmur



Systolic Ejection Murmur



Continuous Murmur

Figure 1. Heart Sounds and Murmurs

Jugular Venous Pulsations - JVP (see Figure 2)

- ❑ height of column of blood filling internal jugular vein, related to RA and RV filling and dynamics, measured as X cm above sternal angle (ASA) (which lies 5cm above the RA; normal JVP is 2-4 cm ASA)
- ❑ distinguishing features of the JVP vs carotid impulse
  - location - between heads of the sternocleidomastoid muscle, coursing towards angle of jaw
  - multiple waveforms in normal patient
  - non-palpable
  - obliterated with pressure at base of neck
  - soft, undulating quality
  - changes with degree of incline and inspiration (normally drops on inspiration)
  - transient increase with abdominal pressure/Valsalva maneuver
  - descents are clinically more prominent than waves at the bedside
- ❑ normal waveforms
  - "a" wave = atrial contraction - precedes carotid pulse
  - "x" descent = atrial relaxation
  - "c" wave = bulging up of TV during RV systole (may reflect carotid pulse in neck)
  - "x prime" descent = descent of base of heart during ventricular systole
  - "v" wave = passive atrial filling against closed AV valve
  - "y" descent = early rapid atrial emptying following opening of AV valve - occurs after carotid pulse felt

- ❑ pathological waveforms
  - loss of "a" wave
    - A fib, atrial standstill
  - absent venous pulse
    - RHF/CHF, SVC obstruction, cardiac tamponade
  - giant "a" waves
    - contraction of atrium against increased resistance (RVH, PS, TS, pulmonary HTN) with every beat
  - cannon “a” waves
    - contraction of atrium against closed TV as in AV dissociation (AV dissociation, PVC); not with every beat
  - systolic venous pulsation (c-v waves)
    - regurgitation of blood into venous system with ventricular contraction as in TR (rapid “y”)
  - sharp "y" descent
    - increased venous pressure as in constrictive pericarditis (“y”>”x” phenomenon)
- ❑ Hepatojugular reflux (HJR)
  - positive response correlates better increased pulmonary capillary wedge pressure (PCWP) (L-sided failure) than R-sided failure
  - sustained > 4 cm rise in JVP with firm abdominal compression
  - postivie response seen in TR, RV failure, pulmonary HTN, CHF, increased PCWP
- ❑ Kussmaul's sign – a paradoxical rise in the JVP on inspiration
- ❑ differential diagnosis: constrictive pericarditis, right ventricular MI high venous pressure

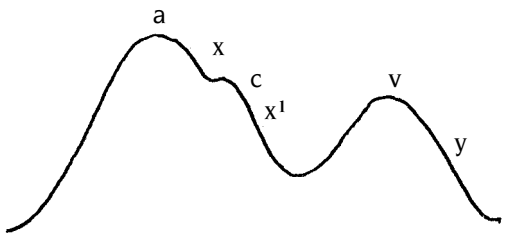


Figure 2. Jugular Venous Pulsations

CARDIAC DIAGNOSTIC TESTS

ECG INTERPRETATION-THE BASICS

Key Features

- ❑ rate
- ❑ rhythm
- ❑ axis
- ❑ waves and segments
- ❑ hypertrophy and chamber enlargement
- ❑ ischemia/infarction
- ❑ miscellaneous

Rate

- ❑ each small box is 0.04 sec; each large box is 0.2 sec.
- ❑ if rhythm is regular, rate is obtained by dividing 300 by number of large squares between two R waves
- ❑ with irregular rhythms note the average ventricular rate over 10 seconds
- ❑ normal adult rate = 60-100 bpm
- ❑ bradycardia < 60 bpm
- ❑ tachycardia > 100 bpm

Rhythm

- ❑ ask four questions
  - Are there P waves present?
  - Are the QRS complexes wide or narrow?
  - What is the relationship between the P waves and QRS complexes?
  - Is the rhythm regular or irregular?
- ❑ definition of normal sinus rhythm
  - has a P wave preceding each QRS complex, and a QRS after each P
  - P wave axis is normal (negative in aVR and positive in II)
  - PR interval is normal and constant
  - P wave morphology is constant

Axis (see Figure 3)
- ❑ deviation - limb leads: normal = positive QRS in I and aVF
  - axis is perpendicular to lead in which QRS is isoelectric
  - QRS axis points toward ventricular hypertrophy and away from infarction
  - see Ventricular Hypertrophy and Hemiblocks sections
- ❑ rotation - precordial leads: normally isoelectric QRS in V_3, V_4
  - clockwise = isoelectric QRS in V_5, V_6
  - counterclockwise = isoelectric QRS in V_1, V_2 (i.e. tall R wave in V_1, see below)

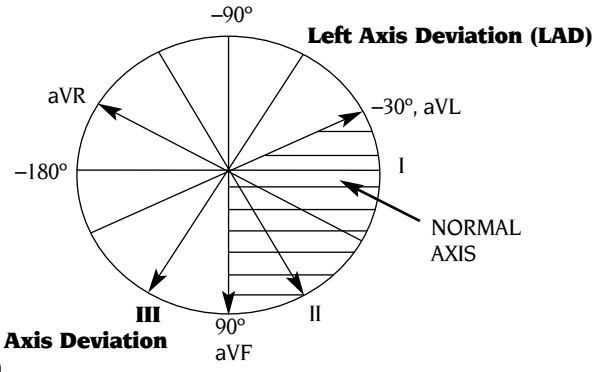


Figure 3. Axes of Electrocardiographic Limb Leads

Waves and Segments
- ❑ P wave - atrial depolarization, smooth contour, entirely positive or negative
- ❑ PR interval - rate dependent; reflects slowing of impulse through the AV node which is governed by parasympathetic and sympathetic discharge
- ❑ QRS complex - ventricular depolarization; any Q wave in V_{1-3} is abnormal;
  R wave increases in amplitude and duration through V_1-V_5;
  S wave is largest in V_2 and gets progressively smaller
- ❑ ST segment - above or below the baseline; point the QRS meets the ST segment is called the J point
- ❑ QT interval - should be < 1/2 of the RR interval
  - interval is rate related (increased HR —> decreased QT)
- ❑ T wave - ventricular repolarization
  - normal = negative in aVR, flat or minimally negative in limb leads; otherwise positive

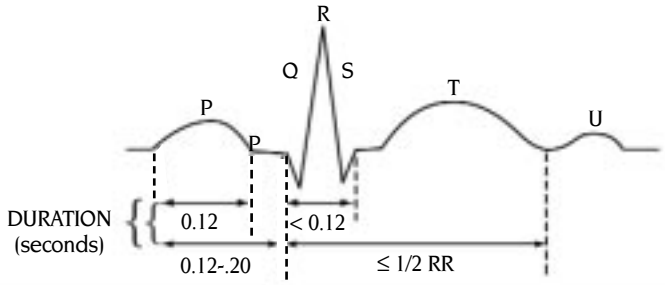


Figure 4. ECG Waveforms and Normal Values

Illustration by Marc Dryer

HYPERTROPHY AND CHAMBER ENLARGEMENT

Right Ventricular Hypertrophy (RVH)
- ❑ QRS < 0.12 seconds, R/S ratio > 1 in V_1, R/S ratio < 1 in V_5 and V_6, R > 7 mm in V_1
- ❑ right axis deviation (RAD) (> 90°)
- ❑ Asymmetric ST segment depression and T wave inversion in V_1 and V_2 (RV strain pattern)

Left Ventricular Hypertrophy (LVH)
- ❑ S in V_1 or V_2 + R in V_5 or V_6 > 35 mm
- ❑ S in V_1 or V_2 or R in V_5 or V_6 > 25 mm
- ❑ R in aVL > 11 mm
- ❑ R in I + S in III > 25 mm
- ❑ left axis deviation (LAD) (> –30°) with slightly widened QRS
- ❑ asymmetric ST segment depression and T wave inversion (LV strain) leads I, aVL, V_{4-6}
- ❑ left atrial enlargement (LAE)

Right Atrial Enlargement(RAE) (P Pulmonale)
❑ P wave > 2.5 mm (in height) in leads II, III or aVF

Left Atrial Enlargement (LAE) (P Mitrale)
❑ P wave duration > 0.11s best seen in leads I, II, aVL, V4-V6
❑ large, biphasic P wave in V1 with deep terminal component that is at least one square wide (0.04 sec) and one square deep (1 mm)
❑ notched P with interpeak interval > 0.04 seconds in I, II or aVL

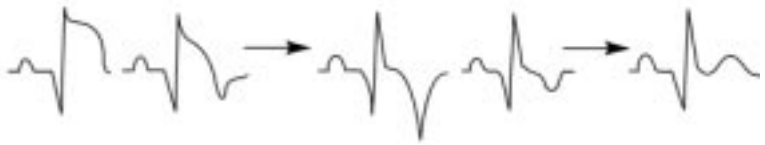
Clinical Pearl
DDx of tall R wave in V1
❑ **RVH, Posterior MI, WPW, HCM (septal hypertrophy), Duchenne muscular dystrophy, and dextrocardia.**

ISCHEMIA / INFARCTION (see Figure 5)

During an ischemic event/acute MI, the ECG changes with time may include:
❑ ischemia: T waves invert at site of injury
❑ injury: ST segment elevation +/– tall peaked T waves, "hyperacute" T waves at area of injury, with reciprocal ST segment depression
  • acute MI = ST elevation in 2 or more contiguous leads in an arterial territory
❑ necrosis: Q waves develop: signifies completed transmural infarct
  • significant if > 1 mm wide (> 0.04 seconds) or if > 1/3 the amplitude of QRS
  • NOTE: Q waves are normally present in lead V1 and non-significant Q's often present in lead III

DDx for ST Segment Changes
❑ elevation
  • early repolarization (normal variant)
  • acute MI
  • post MI
  • Prinzmetal's angina (coronary vasospasm)
  • acute pericarditis
  • ventricular aneurysm
  • LBBB
❑ depression
  • angina (ischemia)
  • subendocardial infarction (non Q-wave MI)
  • acute posterior wall MI (V_1 and V_2)
  • LVH or RVH with strain
  • digitalis effect ("scooping" or "hockey stick")
  • hypokalemia, hypomagnesemia
  • LBBB, RBBB, WPW

T Wave



Acute	**Recent**	**Old**
days	weeks-months	months-years
(avg. 3-5)	(avg. 2-6 months)	(avg. > 6 months)
ST segment elevation	T wave inversion	Just significant Qs

Figure 5. ECG changes with Ischemia/Infarction

Illustration by Victoria Rowsell

Table 4. Areas of Infarction

Infarct Area	Usual Involved Vessel	Q waves
anteroseptal	left anterior descending (LAD)	V_1, V_2
anterior		V_3, V_4
anterolateral		I, aVL, V_3-V_6
extensive anterior		I, aVL, V_1 - V_6
inferior	right coronary artery (RCA)	II, III, aVF
lateral*	circumflex	I, aVL, V_5, V_6
posterior	RCA (accompanies inf. MI) circumflex (isolated post. MI)	V_6, mirror image V_1 and V_2
right ventricle	RCA (most often)	V_4R (V_5R and V_6R) (right sided chest leads)

*often no ECG changes because small infarcts and lateral wall is late in the depolarization (QRS complex)

Variations in Cardiac Vascular Anatomy

❑ Table 4 describes anatomy of "right-dominant" circulation (80%)
❑ compare with
- left-dominant circulation (15%)
  - posteroinferior LV supplied by LCA
- balanced circulation (5%)
  - dual supply of posteroinferior LV by RCA and LCA

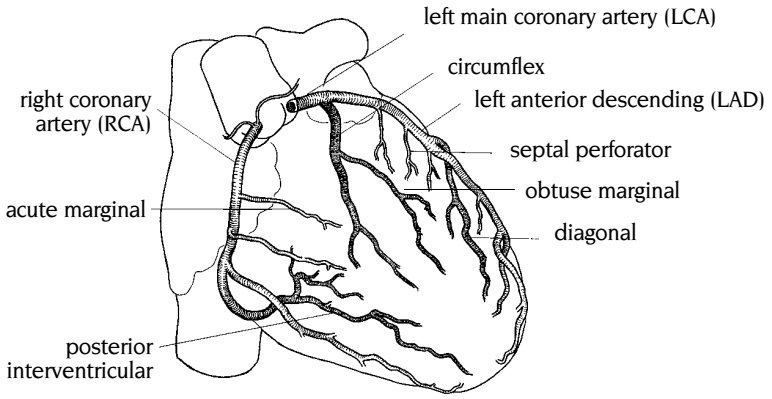


Figure 6. Anatomy of the Coronary Arteries (right anterior oblique projection)

MISCELLANEOUS ECG CHANGES

Electrolyte Disturbances

❑ hyperkalemia
- peaked T waves, flat P, wide QRS, long PR interval, elevated ST segment

Illustrations by Pascale Tranchemontagne

❑ hypokalemia
- flattened T waves, U waves, ST depression, prolonged Q-T interval

Illustrations by Pascale Tranchemontagne

❑ hypocalcemia
- prolonged Q-T interval

❑ hypercalcemia
- shortened Q-T interval

Hypothermia
- ❑ prolonged intervals, sinus bradycardia, slow A fib
- ❑ beware of muscle tremor artifact
- ❑ Osborne or J wave deflection

Early Pericarditis
- ❑ early - diffuse ST segment elevation +/– "PR segment depression"
- ❑ early upright T waves
- ❑ later - isoelectric ST segment and T waves flat or inverted
- ❑ tachycardia

Low Voltages
- ❑ definition - total QRS height in precordial leads < 10 mm, limb leads < 5 mm
- ❑ DDx
  - inappropriate voltage standardization
  - pericardial effusion (e.g. tamponade)
  - barrel chest (COPD), obesity
  - hypothyroidism
  - dilated cardiomyopathy, myocardial disease, myocarditis
  - amyloidosis/infiltrative cardiomyopathy

Drugs
- ❑ Digoxin
- ❑ therapeutic levels may be associated with "Dig effect"
  - T wave depression or inversion
  - ST downsloping or "scooping"
  - QT shortening +/– U waves
  - slowing of ventricular rate in A Fib

Illustration by Seline McNamee

- ❑ toxic levels associated with
  - tachyarrhythmias (especially paroxysmal atrial tachycardia (PAT)) with conduction blocks
  - PVC's, bigeminy
  - classic "regularization" of ventricular rate in A fib due to complete AV dissociation
- ❑ Quinidine, phenothiazines, tricyclic antidepressants (TCA's)
  - prolonged QT interval, U waves

Other Cardiac Conditions
- ❑ hypertrophic cadiomyopathy (HCM)
  - ventricular hypertrophy, LAD, septal Q waves
- ❑ Myocarditis
  - conduction blocks, low voltage

Pulmonary Disorders
- ❑ chronic obstructive pulmonary disease (COPD)
  - low voltage, RAD, poor R wave progression
  - chronic cor pulmonale can produce RAE and RVH with strain
  - multifocal atrial tachycardia (MAT)
- ❑ Massive pulmonary embolus (PE)
  - sinus tachycardia and A fib are the most common arrhythmias
  - RVH with strain, RBBB, S_I, Q_{III}, T_{III} (inverted T) ($S_1Q_3T_3$)

AMBULATORY ECG (HOLTER MONITOR)
- ❑ 24-48 hr ECG recording with patient diary of symptoms to determine correlation between symptoms and abnormalities
  - indications
    1. detect intermittent arrhythmias
    2. relate symptoms to dysrhythmias
    3. detect myocardial ischemia

ECHOCARDIOGRAPHY
- ❑ Two-dimensional (2-D) ECHO: anatomy - ultrasound (U/S) reflecting from tissue interfaces
  - determines
    - LV systolic ejection fraction (LVEF)
    - chamber sizes
    - wall thickness
    - valve morphology
    - pericardial effusion
    - wall motion abnormalities
    - complications of acute MI
- ❑ Doppler: blood flow – U/S reflecting from intracardiac RBCs
  - determines blood flow velocities to estimate valve areas and determine intracardiac gradients
- ❑ Colour flow imaging determines:
  - valvular regurgitation
  - valvular stenosis
  - shunts

❑ Transesophageal Echo (TEE)
- high quality images but invasive
- more sensitive for
  - prosthetic heart valves
  - to identify cardiac sources of systemic emboli, intracardiac thrombi, tumours, debris within the aorta, valvular vegetations, and infective endocarditis
  - aortic dissection

CORONARY ANGIOGRAPHY (see Cardiac and Vascular Surgery Chapter)

❑ technique: injection of radiopaque dye into coronary arteries via percutaneous femoral catheter
❑ information obtained: coronary anatomy, LVEF with ventriculography, hemodynamic indices
❑ Indications:
- Diagnosis: gold standard for detecting and quantifying CAD
- Prognosis: post-MI
- Guiding Therapy: e.g. CABG vs. PTCA vs. medical therapy

❑ complications (%): death (0.1), stroke (0.07), MI (0.07), other major (1.0-2.0), minor (10)

CARDIAC STRESS TESTS AND NUCLEAR CARDIOLOGY

❑ indications
- assessment of chest pain (detection of CAD)
- risk stratification post-MI
- preoperative screening and risk assessment
- assessment of response to therapy
- assessment of myocardial viability

❑ stressors
- physical stressors: treadmill or bicycle
- pharmacological stressors
  - increased coronary flow: dipyridamole (Persantine), adenosine
  - increased myocardial O_2 demand: dobutamine (β1-selective agonist)

❑ ischemia detectors
- ECG: observe for ischemic changes during stress
- ECHO: visualize myocardial effects of ischemia
- SPECT myocardial nuclear perfusion studies
  - tracers infused during stress
  - thallium-201 (201Tl, a K^+ analogue)
  - technetium-99 (99Tc)-labelled tracer (sestamibi = Cardiolyte)
- SPECT images of the heart obtained during stress and at rest 4h later
  - fixed defect = impaired perfusion at rest and during stress (infarcted)
  - reversible defect = impaired perfusion only during stress (ischemic)
- Other imaging techniques: PET, MRI, ultrafast CT, TEE (uncommonly used)

❑ ventricular function assessment (LVEF, RVEF, ventricular size and volume, wall motion anomalies, etc.)
- Radionuclide angiography (MUGA): 99Tc- radiolabelled RBCs
- ECHO
- Ventriculography

Table 5. Attributes and Limitations of Various Stress Tests

Factor	Treadmill Test (GXT)	Stress Echo	Nuclear Perfusion	Radionuclide Angiography
Sensitivity	65-70%	90%	80-85%	80-85%
Specificity	65-70%	90%	90%	
Localizing ischemia	poor	good	good	good
Additional info compared with GXT	N/A	rest & exercise LVEF, plus all other echo parameters	rest LVEF, lung uptake, infarct size, LV size	rest & exercise LVEF, regional wall motion, LV volumes, RV function
Clinical or technical limitations	abnormal resting ECG, pretest probability very important	COPD, obesity	obesity, attenuation artifacts	arrhythmias
Relative cost	$	$$	$$$$	$$$

Modified from: Anon. Mayo Clinic Proceedings. 1996; 71:43-52.

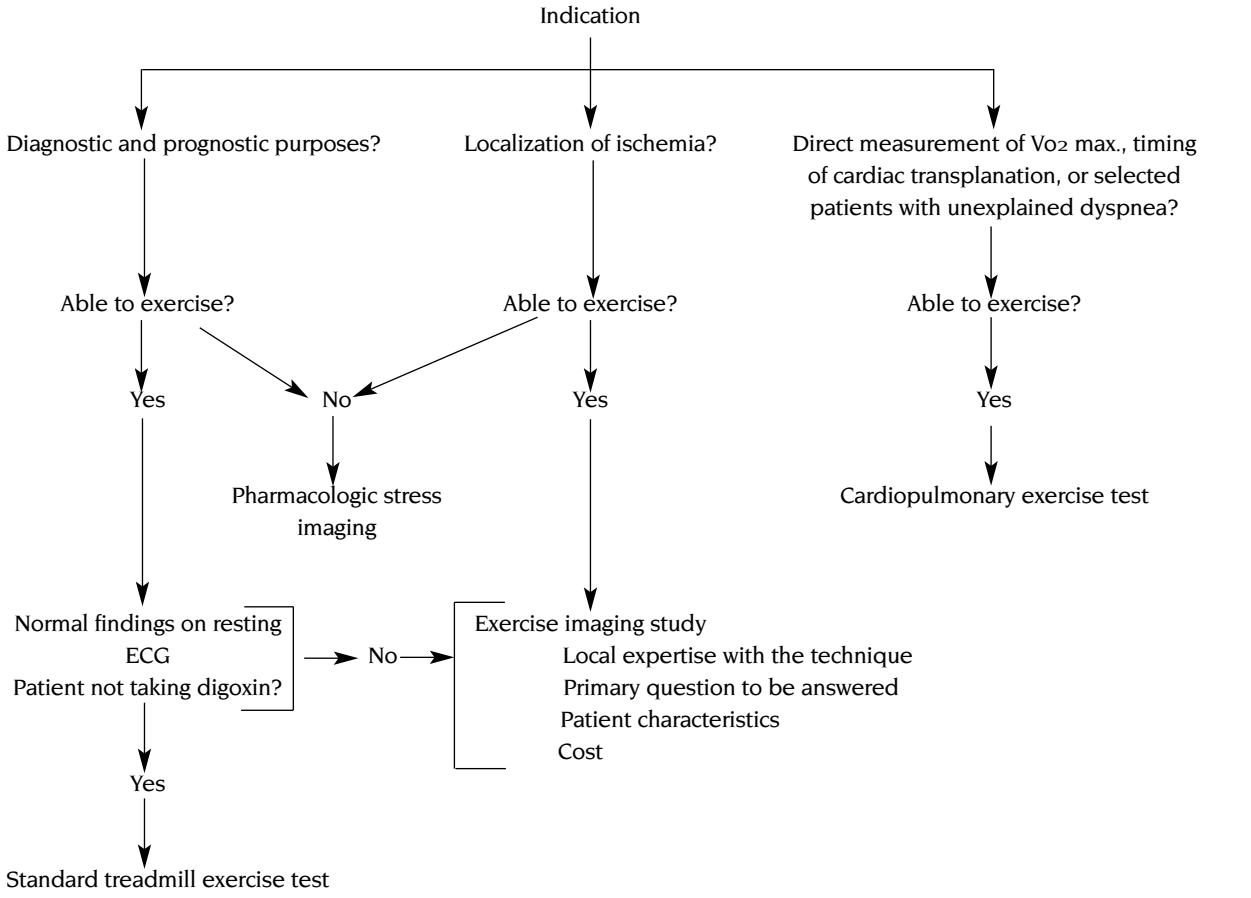


Figure 7. Algorithm for test selection for an individual patient.

From: Anon. *Mayo Clinic Proceedings* 1996; 71;43-52.

ARRHYTHMIAS

MECHANISMS OF ARRHYTHMIAS

1. ALTERED IMPULSE FORMATION

❑ divided into two potentially arrhythmogenic processes:
❑ AUTOMATICITY = the ability of a cell to depolarize itself to threshold and, therefore, generate an action potential
❑ cells with this ability are known as "pacemaker" cells
- SA node, purkinje cells throughout atria
- bundle of His, bundle branches
- purkinje cells in fascicles and peripheral ventricular conduction system

❑ automaticity is influenced by
- neurohormonal factors: sympathetic and parasympathetic
- drugs: e.g. Digoxin has vagal effect on SA and AV nodes but sympathetic effect on other pacemaker sites
- local ischemia/infarction or other pathology
- blockage of proximal pacemaker (SA node) impulses which allows more distal focus to control the ventricular rhythm

❑ TRIGGERED ACTIVITY = abnormal depolarization occurring during or after repolarization
- oscillations of the membrane potential after normal depolarization lead to recurrent depolarization
- prolonged QT interval predisposes (e.g. electrolyte disturbances, antiarrhythmic drugs)
- postulated mechanism of Torsades de Pointes

2. ALTERED IMPULSE CONDUCTION

- ❑ re-entry
  - phenomenon which requires parallel electrical circuit in which two limbs have different refractory periods, e.g. AVNRT
- ❑ conduction blocks - partial or total
- ❑ ventricular pre-excitation
  - congenital abnormality in which ventricular myocardium is electrically activated earlier than by the normal AV nodal impulse
  - e.g. bypass tract in WPW syndrome

OTHER ETIOLOGIC FACTORS

- ❑ increased LA size —> increased risk of A fib
- ❑ bradycardia predisposes via temporal dispersion in refractory periods; e.g. tachy-brady syndrome
- ❑ hypoxia/acidosis lowers the threshold for V fib
- ❑ electrolyte disturbances, e.g.: hypokalemia, imbalances of Ca^{+2}, Mg^{+2}
- ❑ infection, e.g.: myocarditis or infective endocarditis (causing abscess and complete heart block)
- ❑ cardiomyopathies, degenerative disease, infiltration (e.g. sarcoid)
- ❑ ischemia, increased sympathetic tone

CLINICAL APPROACH TO ARRHYTHMIAS

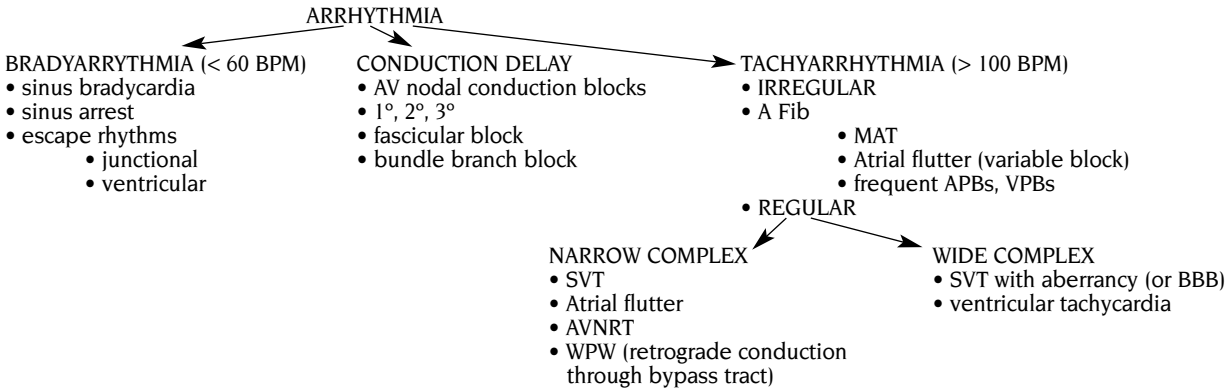


Figure 8. Clinical Approach to Arrhythmias

BRADYARRHYTHMIAS

Presentation

- ❑ often asymptomatic
- ❑ symptoms can include dizziness, fatigue, dyspnea and presyncope or syncope
- ❑ effects of bradycardia depend on rate, and patient's co-morbid conditions (e.g. heart failure)

DDx

Sinus Bradycardia

- ❑ sinus rhythm at regular heart rate less than 60 bpm
- ❑ caused by excessive vagal tone: spontaneous (vasovagal syncope), acute MI (inferior), drugs, vomiting, hypothyroidism, increased intracranial pressure (ICP)
- ❑ treatment: if symptomatic, atropine +/– electrical pacing (chronic)

Sinus Arrhythmia

- ❑ irregular rhythm with normal P wave and constant, normal PR interval
- ❑ normal variant - inspiration accelerates the HR; expiration slows it down
- ❑ pathological - uncommon, variation not related to respiration

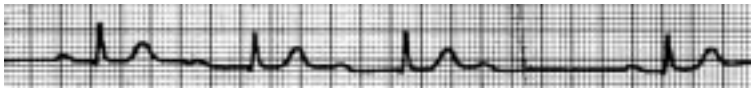
Sinus Arrest or Exit Block

- ❑ sinus node stops firing (arrest) or depolarization fails to exit the sinus node (exit block)
- ❑ depending on duration of inactivity, escape beats or rhythm may occur
  - next available pacemaker will take over, in the following order
    - atrial escape (rate 60-80): originates outside the sinus node within the atria (normal P morphology is lost)
    - junctional escape (rate 40-60): originates near the AV node; a normal P wave is not seen
      - may occasionally see a retrograde P wave representing atrial depolarization moving backward from the AV node into the atria
    - ventricular escape (rate 20-40): originates in ventricular conduction system
      - no P wave; wide, abnormal QRS (ECG tracing)
- ❑ treatment: stop meds which suppress the sinus node (β blockers, CCB, Digoxin); may need pacing

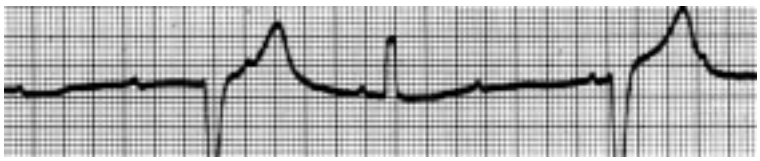
Sick Sinus Syndrome (SSS)
- ❑ includes above sinus node disturbances, when pathologic
  - causes: structural SA node disease, autonomic abnormalities, or both
- ❑ bradycardia may be punctuated by episodes of SVT, especially A fib or atrial flutter (tachy-brady syndrome)
- ❑ treatment: pacing for bradycardia; meds for tachycardia

CONDUCTION DELAYS

AV Node Conduction Blocks
- ❑ look at the relationship of the P waves to the QRS complexes
- ❑ 1st degree - constant prolonged PR interval (> 0.2 seconds)
  - all beats are conducted through to the ventricles
  - no treatment required if asymptomatic
- ❑ 2nd degree (Mobitz) - not all P waves followed by QRS; distinguish Type I from Type II
  - Mobitz Type I (Wenckebach) - due to AV node blockage
    - progressive prolongation of the PR interval until a QRS is dropped
    - treatment: none unless symptomatic; atropine

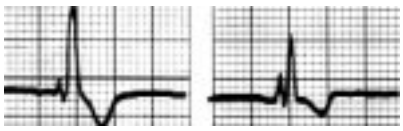


  - Mobitz Type II - due to His-Purkinje blockage
    - all-or-none conduction; QRS complexes are dropped at regular intervals (e.g. 2:1, 3:1, etc.) with stable PR interval (normal or prolonged)
    - risk of developing syncope or complete heart block
    - treatment: pacemaker (ventricular or dual chamber)
- ❑ 3rd degree or complete heart block - no P wave produces a QRS response
  - complete AV dissociation (atria and verntricles contracting independently; may see P waves "marching through" QRS's)
  - can have narrow junctional QRS or wide ventricular QRS (junctional vs ventricular escape rhythm)
  - rate usually 30-60 bpm
  - may cause Stokes-Adams attacks: syncope associated with brief cardiac arrest
  - treatment: pacemaker (ventricular or dual chamber)

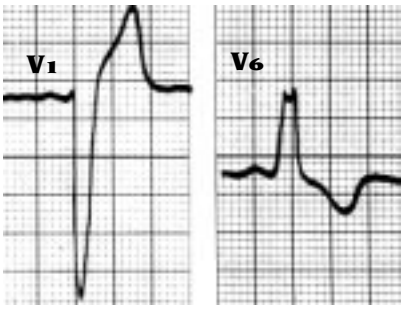


Bundle Branch and fascicular Blocks
- ❑ RBBB, left anterior fascicle and left posterior fascicle should each be considered individually, and combination (i.e. bifascicular) block S should also be noted

Bundle Branch Blocks (BBB)
- ❑ QRS complex > 0.12 seconds
- ❑ RBBB
  - RSR' in V_1 and V_2 (rabbit ears), with ST segment depression and T wave inversion
  - presence of wide (or deep) S wave in I, V_6
  - widely split S_2 on auscultation
- ❑ LBBB
  - broad or notched monophasic R wave with prolonged upstroke and absence of initial Q wave in leads V_6, I and aVL, with ST segment depression and T wave inversion
  - large S or QS in V_1
  - paradoxically split S_2 on auscultation
- ❑ note
  - with BBB the criteria for ventricular hypertrophy become unreliable
  - with LBBB, infarction is difficult to determine



RBBB



LBBB

Hemiblock
- ❑ block of anterior or posterior fascicle of LBB
- ❑ anterior hemiblock
  - normal QRS duration; no ST segment or T wave changes
  - left axis deviation (> –45°), with no other cause present
  - small Q in I and aVL, small R in II, III and aVF
- ❑ posterior hemiblock
  - normal QRS duration; no ST segment or T wave changes
  - right axis deviation (> 110 degrees), with no other cause present
  - small R in I and aVL, small Q in II, III and aVF

TACHYARRHYTHMIAS

Presentation
- ❑ symptoms, when present, include palpitations, dizziness, dyspnea, chest discomfort, presyncope or syncope
- ❑ may precipitate CHF, hypotension, or ischemia in patients with underlying disease
- ❑ incessant untreated tachycardias can cause cardiomyopathy (rare)
- ❑ includes supraventricular and ventricular rhythms

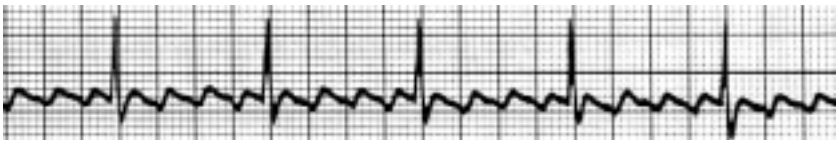
DDx

1. SUPRAVENTRICULAR TACHYARRHYTHMIAS
- ❑ narrow (i.e., normal) QRS complex or wide QRS if aberrant ventricular conduction or pre-existing BBB
- ❑ aberrancy = intraventricular conduction delay associated with a change in cycle length (i.e., with tachycardia); not normal pattern for the individual

Sinus Tachycardia
- ❑ sinus rhythm at a rate greater than 100 bpm
- ❑ Etiology: fever, hypotension, thyrotoxicosis, anemia, anxiety, hypovolemia, PE, CHF, MI, shock, drugs (EtOH, caffeine, atropine, catecholamines)
- ❑ treatment: treat underlying disease; consider propranolol if symptomatic

Premature Beats
- ❑ Atrial Premature Beat (APB)
  - single ectopic supraventricular beat originating in the atria
  - P wave contour of the APB differs from that of a normal sinus beat
- ❑ Junctional Premature Beat
  - a single ectopic supraventricular beat that originates in the vicinity of the AV node
  - there is no P wave preceding the premature QRS complex, but a retrograde P wave may follow the QRS if AV nodal conduction is intact
- ❑ treatment: none unless symptomatic; β blockers or CCB

Atrial Flutter
- ❑ regular; atrial rate 250-350 bpm, usually 300
- ❑ etiology: IHD, thyrotoxicosis, MV disease, cardiac surgery, COPD, PE, pericarditis
- ❑ ventricular conduction is variable e.g. 2:1, 3:1, 4:1 block, etc.
- ❑ ECG: sawtooth inferior leads; narrow QRS (unless aberrancy)
- ❑ carotid massage (check first for bruits), Valsalva or adenosine: increases the block (i.e. slows pulse), brings out flutter waves
- ❑ treatment
  - rate control: β blocker, verapamil, Digoxin
  - medical cardioversion: procainamide, sotalol, amiodarone, quinidine
  - electrical cardioversion: DC shock (@ low synchronized energy levels: start at 50 J)

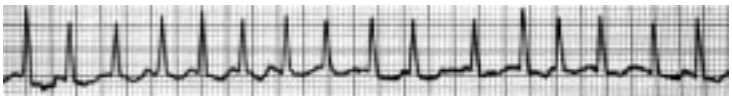


Clinical Pearl
- ❑ **Narrow complex tachycardia at a rate of 150 is atrial flutter with 2:1 block until proven otherwise.**

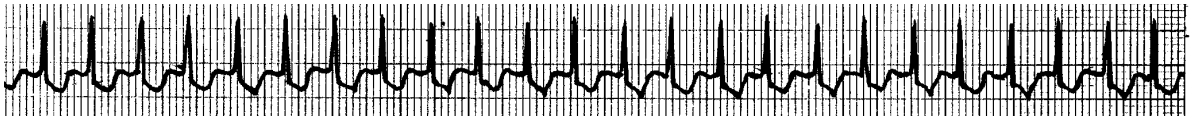
Multifocal Atrial Tachycardia (MAT)
- ❑ irregular rhythm; atrial rate 100-200 bpm; at least 3 distinct P wave morphologies and 3 different P-P intervals present on ECG
- ❑ probably results from increased automaticity of several different atrial foci
- ❑ hence varying P-P, P-R, and R-R intervals, varying degrees of AV block
- ❑ common in COPD, hypoxemia, hypokalemia, hypomagnesemia, sepsis, theophylline or Digoxin toxicity
- ❑ if rate < 100 bpm, then termed a Wandering Atrial Pacemaker
- ❑ carotid massage has no effect in MAT
- ❑ treatment: treat the underlying cause; if necessary try metoprolol (if no contraindications)

Atrial Fibrillation (A fib)
- ❑ seen in 10% of population over 75 years old
- ❑ the majority of cardiogenic strokes and peripheral thromboembolic events occur in association with A fib
- ❑ Etiology: CAD, valvular disease, pericarditis, cardiomyopathy, PE, HTN, COPD, thyrotoxicosis, tachy-brady syndrome, EtOH (holiday heart)
- ❑ irregularly irregular ventricular rate; narrow QRS unless aberrancy,
  - undulating baseline; no P waves
- ❑ atrial rate 400-600 bpm, ventricular rate variable depending on AV node, around 140-180 bpm
- ❑ wide QRS complexes due to aberrancy may occur following a long short R-R cycle sequence ("Ashman phenomenon")
- ❑ lose atrial contribution to ventricular filling (no "a" waves seen in JVP)
- ❑ carotid massage: may slow ventricular rate
- ❑ A fib resistant to cardioversion - significant LA enlargement, longer duration of A fib
- ❑ major issues to be addressed with A fib:
  - Rate control (ventricular) – beta blocker, verapamil, digoxin
  - Anti-coagulation (prevention of thromboembolic phenomenon)
    - warfarin for paroxysmal or chronic A fib
    - balance risk of bleeding 1%/year vs. risk of clot (warfarin reduces thromboembolic event rate by 67% in nonrheumatic A fib)
  - Cardioversion
    - OK without anticoagulation within 48 hours of onset (by history) of A fib
    - if > 48 hours after onset MUST anticoagulate at least 3 weeks prior to cardioversion and 4 weeks after cardioversion
    - alternate option is TEE prior to eraly electrical cardioversion to rule out clot (controversial)
    - medical - sotalol, amiodarone, Class I agent if normal LV function (e.g. IV procainamide, propafenone)
    - electrical - synchronized DC cardioversion (Diltiazem)
  - treat any etiology that can be identified

- ❑ Note – drug - refractory symptomatic A fib may be referred for AV node ablation followed by permanent pacemaker insertion



Paroxysmal Supraventricular Tachycardia (PSVT)
- ❑ sudden onset regular rhythm; rate 150-250 bpm
- ❑ usually initiated by a supraventricular or ventricular premature beat
- ❑ common mechanisms are AV nodal reentry and accessory tract reentry
- ❑ atrioventricular nodal tachycardia (AVNRT) accounts for 60-70% of all SVTs
- ❑ retrograde P waves may be seen but are usually lost in the QRS complex
- ❑ treatment
  - acute: Valsalva or carotid massage (check first for bruits), adenosine especially if associated with WPW (adenosine is 1st choice if unresponsive to vagal maneuvers); if no response, try metoprolol, digoxin, verapamil; DC shock if signs of cardiogenic shock, angina, or CHF
  - chronic: β blocker, verapamil, Digoxin, anti-arrhythmic drugs, EPS catheter ablation

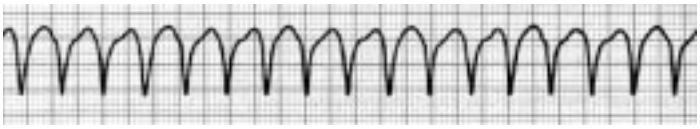


2. VENTRICULAR TACHYARRHYTHMIAS

Premature Ventricular Contraction (PVC) or Ventricular Premature Beats (VPB)
- ❑ QRS width greater than 0.12 seconds, no preceding P wave, bizarre QRS morphology
- ❑ premature in the cardiac cycle, may be followed by a prolonged pause (compensatory)
- ❑ origin: LBBB pattern = RV site; RBBB pattern = LV site
- ❑ rules of malignancies with PVC's
  - frequent, (> 10/hour), consecutive (≥ 3 = VT) or multiform (varied origin)
  - PVC's falling on the T wave of the previous beat ("R on T phenomenon"): vulnerable time in cycle with risk of VT or V fib)
- ❑ PVCs in isolation not treated, as risks not altered, no effect on mortality
- ❑ treatment: since no evidence to suggest that treatment decreased mortality, PVCs are not usually treated
- ❑ consider β blockers if symptomatic palpitations

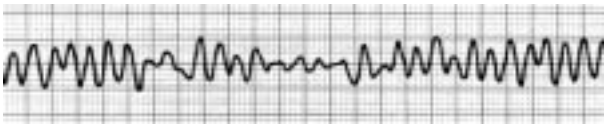
Accelerated Idioventricular Rhythm
- ❑ benign rhythm - originates in terminal Purkinje system or ventricular myocardium
- ❑ represents a ventricular escape focus that has accelerated sufficiently to drive the heart
- ❑ Etiology: sometimes seen during acute MI (especially during reperfusion) or Digoxin toxicity
- ❑ regular rhythm, rate 50-100 bpm
- ❑ rarely requires treatment
- ❑ treatment: if symptomatic, lidocaine, atropine

Ventricular Tachycardia (VT)
- ❑ a run of three or more consecutive PVCs rate > 100 minute is called VT
- ❑ etiology
  - note: only with memomorphic VT
  - CAD with MI is most common underlying cause
- ❑ sustained VT (longer than 30 seconds) is an emergency, prestaging cardiac arrest and requiring immediate treatment
- ❑ rate 120-300 bpm
- ❑ broad QRS, AV dissociation, fusion beats, capture beats, left axis deviation, monophasic or biphasic QRS in V_1 with RBBB, concordance V_1-V_6



- ❑ fusion beat
  - occurs when an atrial impulse manages to slip through the AV node at the same time that an impulse of ventricular origin is spreading across the ventricular myocardium
  - the two impulses jointly depolarize the ventricles producing a hybrid QRS complex that is morphologically part supraventricular and part ventricular
- ❑ capture beat
  - occurs when an atrial impulse manages to "capture" the ventricle and get a normal QRS
- ❑ treatment (for acute sustained VT)
  - hemodynamic compromise – DC cardioversion
  - no hemodynamic compromise - DC shock, lidocaine, amiodarone, type Ia agents (procainamide, guinidine)

Ventricular Fibrillation (V fib)
- ❑ medical emergency; pre-terminal event unless promptly cardioverted
- ❑ most frequently encountered arrhythmia in adults who experience sudden death
- ❑ mechanism: simultaneous presence of multiple activation wavefronts within the ventricle
- ❑ no true QRS complexes - chaotic wide tachyarrhythmia without consistent identifiable QRS complex
- ❑ no cardiac output during V fib
- ❑ refer to ACLS algorithm for complete therapeutic guidelines



Torsades de Pointes
- ❑ polymorphic VT - means "twisting of the points"
- ❑ looks like VT except that QRS complexes rotate around the baseline changing their axis and amplitude
- ❑ ventricular rate greater than 100, usually 150-300
- ❑ etiology: seen in patients with prolonged QT intervals
  - congenital long QT syndromes
  - drugs - e.g. Class IA (quinidine), Class III (sotalol), phenothiazines (TCAs), erythromycin
  - electrolyte disturbances - hypokalemia, hypomagnesemia
  - other - nutritional deficiencies
- ❑ treatment: IV magnesium, temporary pacing, isoproterenol and correct underlying cause of prolonged QT, DC cardioversion if hemodynamic compromise present

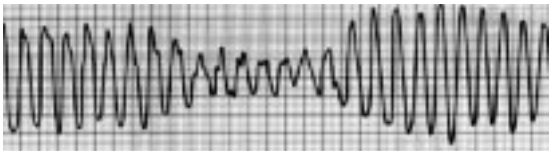


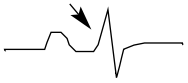
Table 6. Differentiation of VT vs. SVT with Aberrant Conduction*		
	VT	**SVT**
Clinical Clues		
carotid massage	no response	may terminate
cannon "a" waves	may be present	not seen
neck pounding	may be present	not seen
ECG Clues		
AV dissociation	may be seen	not seen
fusion beats	may be seen	not seen
initial QRS deflection	may differ from normal QRS complex	same as normal QRS complex
axis	extreme axis deviation	normal or mild deviation
* if patient > 65, presence of previous MI or structural heart disease then chance of VT > 95%		

PREEXCITATION SYNDROMES

Wolff-Parkinson-White (WPW) Syndrome

- ❑ bypass pathway called the Bundle of Kent connects the atria and ventricles
- ❑ congenital defect, present in 3:1.000
- ❑ criteria
  - PR interval is less than 0.12 seconds
  - wide QRS complex due to premature activation
  - repolarization abnormalities
  - delta wave seen in leads with tall R waves
    - slurred initial upstroke of QRS complex

(delta wave)



- ❑ the two tachyarrhythmias most often seen in WPW are PSVT and A fib
- ❑ carotid massage, vagal maneuvers, and adenosine can enhance the degree of pre-excitation by slowing AV nodal conduction
- ❑ note: if wide complex A fib, concern is that anterograde conduction is occurring down a bypass tract; therefore do not use agents that slow AV conduction (e.g. Digoxin) as may increased conduction through the bypass tract and precipitate V fib. In WPW and A fib use IV procainamide

Lown-Ganong-Levine Syndrome

- ❑ the PR interval is shortened to less than 0.12 seconds
- ❑ the QRS complex is narrow and there is no delta wave

PACEMAKER INDICATIONS

- ❑ SA node dysfunction
  - symptomatic bradycardia
- ❑ AV nodal - infranodal block
  - Mobitz II
  - complete heart block
- ❑ symptomatic carotid sinus hypersensitivity

PACING TECHNIQUES

- ❑ temporary: transvenous (jugular, subclavian, femoral) or external pacing
- ❑ permanent: transvenous into RA, apex of RV or both; power source implanted under clavicle
  - can sense and pace atrium, ventricle or both
  - new generation = rate responsive, able to respond to physiologic demand
- ❑ nomenclature e.g. "VVIR"

V - chamber paced : ventricle
V - chamber sensed : ventricle
I - action : inhibit
R - rate responsive

ISCHEMIC HEART DISEASE (IHD)

BACKGROUND

Epidemiology
- ❑ commonest cause of cardiovascular morbidity and mortality
- ❑ male: female ratio
  - = 2:1 with all age groups included (Framingham study)
  - = 8:1 < age 40
  - = 1:1 > age 70
  - disparity due to protective effect of estrogen
- ❑ peak incidence of symptomatic IHD is from ages 50 to 60 in men and ages 60 to 70 in women
- ❑ spectrum of IHD/CAD ranges anywhere from asymptomatic to sudden death

Atherosclerosis and IHD
- ❑ atherosclerosis and thrombosis are by far the most important pathogenetic mechanisms in IHD

Major Risk Factors For Atherosclerotic Heart Disease
- ❑ smoking
  - risk can be halved by cessation of smoking
- ❑ diabetes mellitus (DM)
  - micro and macrovascular complications
- ❑ hypertension (HTN)
  - depends on degree and duration
- ❑ family history (FHx)
  - first degree male relative < 55 or first degree female relative < 60
- ❑ hyperlipidemia

Other Minor Risk Factors
- ❑ obesity
  - > 30% above ideal weight
- ❑ sedentary lifestyle
- ❑ hyperhomocysteinemia

Preventative Measures
- ❑ smoking cessation
- ❑ tight glycemic control in diabetics
- ❑ BP control
  - major reason for the recent decrease in IHD
- ❑ lipid-modifying therapy
- ❑ dietary measures e.g. mild alcohol consumption
- ❑ weight loss
- ❑ exercise improves weight, HTN, cholesterol and glycemic control
- ❑ family screening (high risk groups)

ANGINA PECTORIS

Definition
- ❑ symptom complex resulting from an imbalance between oxygen supply and demand in the myocardium

Pathophysiology of Myocardial Ischemia

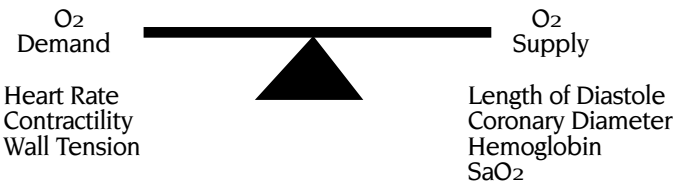


Figure 9. Physiological Principles

Etiology
- ❑ decreased myocardial oxygen supply
  - atherosclerotic heart disease (vast majority)
  - coronary vasospasm (variant angina= Prinzmetal's Angina)
  - severe aortic stenosis or insufficiency
  - thromboembolism
  - severe anemia
  - arteritis (e.g. Takayasu's syndrome, syphilis, etc.)
  - aortic dissection
  - congenital anomalies

- ❑ increased myocardial oxygen demand
  - myocardial hypertrophy
  - severe tachycardia
  - severe hyperthyroidism
  - severe anemia

DDx
- ❑ musculoskeletal (MSK) disease
  - rib fracture
  - intercostal muscle tenderness
  - costochondritis (Tietze's syndrome)
  - nerve root disease (cervical radiculitis)
- ❑ gastrointestinal (GI) disease
  - peptic ulcer disease (PUD)
  - reflux esophagitis
  - esophageal spasm and motility disorder (may be improved by NTG)
- ❑ pulmonary disease
  - pulmonary embolism (PE)
  - pneumothorax
  - pneumonia
- ❑ cardiovascular (CV) disease
  - aortic dissection (asymmetrical BP and pulses, new AR murmur)
  - pericarditis
- ❑ Other
  - intercostal neuritis (shingles)
  - anxiety
- ❑ note
  - careful history and physical required
  - consider risk factors for each entity
  - beware cardiac and non-cardiac disease may coexist

Diagnosis of Angina Pectoris
- ❑ history
  - classically precordial chest pain, tightness or discomfort radiating to left shoulder/arm/jaw
  - dyspnea or fatigue may present as "chest pain equivalents," especially in females
  - associated with diaphoresis or nausea
  - predictably precipitated by the "3 E's" Exertion, Emotion and Eating
  - brief duration, lasting < 10-15 minutes and typically relieved by rest
  - note: always list the presence or absence of the cardiac risk factors in a separate subsection in the history (e.g., + FHx, + HTN, +DM, + smoking, - hypercholesterolemia)
- ❑ stress testing (see Cardiac Diagnostic Tests section)

Variant Angina (Prinzmetal's Angina)
- ❑ vasospasm of coronary arteries results in myocardial ischemia
  - may occur in normal or atherosclerotic vessels
- ❑ typically occurs between midnight and 8 am
- ❑ unrelated to exercise; relieved by Nitrates
- ❑ typically ST elevation on ECG (may be confused with acute MI)
- ❑ diagnose by provocative testing with ergot vasoconstrictors (rarely done)

Syndrome X
- ❑ patient has typical symptoms of Angina yet has normal angiogram
- ❑ may show definite signs of ischemia during exercise testing
- ❑ pathogenesis thought to be due to inadequate vasodilator reserve of coronary resistance vessels
- ❑ has better prognosis than patient with overt atherosclerotic disease

Medical Treatment
- ❑ β blockers (first line therapy)
  - decrease overall mortality
  - decrease heart rate, contractility, and to a lesser degree, blood pressure (afterload)
  - increase coronary perfusion
  - avoid agents with intrinsic sympathomimetic activity (ISA) (e.g. Acebutolol) (these increase demand)
- ❑ nitrates
  - used for symptomatic control
  - no clear impact on survival
  - decrease myocardial work and, therefore, oxygen requirements through venous dilatation (decrease preload) and arteriolar dilatation (decrease afterload)
  - dilate coronary arteries
  - maintain daily nitrate-free intervals to try to prevent nitrate tolerance
- ❑ calcium channel blockers (CCB)
  - variably decrease afterload, decrease heart rate and decrease contractility, produce coronary dilatation
- ❑ ECASA
  - all patients
  - decrease platelet aggregation
- ❑ lipid lowering

Coronary Artery Disease (CAD) Lipid Therapy

Trial		Drug	Dose	CHD Event Reduction
primary	WOSCOPS	pravastatin	40	31%
prevention	AFCAPS	lovastatin	20-40	24%
secondary	LIPID	pravastatin	40	23%
prevention	4S	simvastatin	20-40	34%
	CARE	pravastatin	40	24%

2000 Canadian Guidelines for Treatment of Dyslipidemia

Level of Risk (Definition)	Target Values LDL	TC:HDL Ratio	Tryglycerides
Very High (History of cardiovascular disease or 10 yr risk of CAD > 30%)	< 2.5	< 4	< 2
High (10 yr risk of CAD 20-30%)	< 3	< 5	< 2
Moderate (10 yr risk 10-20%)	< 4	< 6	< 2
Low (10 yr risk < 10%)	< 5	< 7	< 3
Risk calculated based on Framingham data: dertermined by gender, age group, total cholesterol level, HDL level, SBP, history of smoking			

- ❑ treatment strategy
  - short acting nitrates on PRN basis to relieve acute attacks and PRN prior to exertion
  - be careful when combining β blockers and verapamil/diltiazem
    - both decrease conduction and contractility and may result in sinus bradycardia or AV block
  - use nitrates and CCB for variant angina

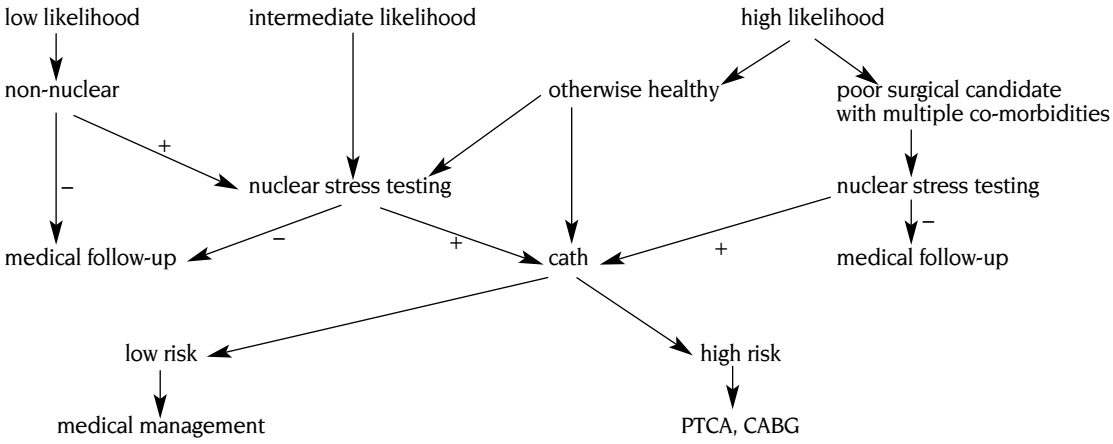


Figure 10. Diagnostic Strategies in the Management of IHD

Indications for Angiography

- ❑ strongly positive exercise test
- ❑ significant, reversible defects on thallium scan
- ❑ refractory to medical therapy or patient unable to tolerate medical therapy
- ❑ unstable angina

Percutaneous Transluminal Coronary Angioplasty (PTCA)
- ❑ uses a balloon inflated under high pressure to rupture atheromatous plaques
- ❑ may be used as primary therapy in angina, acute MI, post-MI angina or in patients presenting with bypass graft stenosis
- ❑ optimally used for proximal lesions free of thrombus and distanced from the origins of large vessel branches – not in Left Main
- ❑ primary success rate is > 80%
- ❑ use of intracoronary stent is associated with a lower restenosis rate (compared with PTCA alone)
- ❑ complications (overall 3-5%)
    - mortality < 1%
    - MI 3-5%
    - intimal dissection + vessel occlusion requiring urgent CABG

Surgical Treatment- Coronary Artery Bypass Grafting (CABG)
- ❑ indications - for survival benefit, or symptomatic relief of angina
    - stable angina (survival benefit for CABG shown)
        - left main coronary disease
        - three-vessel disease with depressed LV function
        - multi-vessel disease with significant proximal LAD stenosis
    - unstable angina (see below)
        - above indications or
            - continuing angina despite aggressive medical therapy
            - complications/failed PTCA
- ❑ comparison of CABG with PTCA
    - studies: RITA, GABI, BARI, EAST, ERACI, CABRI
    - highly select patient population - no left main disease and minimal LV dysfunction
    - overall no difference in survival, but PTCA group had more recurrent ischemia and required more interventions
    - BARI, subset analysis - CABG superior in patients with DM and multi-vessel IHD
- ❑ predictors of poor outcome
    - poor LV function (EF < 40%), history of CHF, NYHA III or IV
    - previous cardiac surgery
    - urgent/emergent case, preoperative IABP
    - gender (relative risk for F:M = 1.6:1)
    - advanced age (> 70), DM, co-morbid disease
- ❑ CABG operative mortality

• elective case	< 1%
• elective case, poor LV function	1-3%
• urgent case	1-5%
• overall (1980-1990)	2.2%

- ❑ efficacy: > 90% symptomatic improvement in angina
- ❑ conduits and patency

• internal mammary (thoracic) artery	90% patency at 10 years
• saphenous vein graft	50% patency at 10 years
• radial/gastroepiploic/inferior epigastric arteries	85% patency at 5 years (improving with experience)

ACUTE CORONARY SYNDROMES (ACS)

Spectrum of ACS
A. Unstable Angina
B. Acute Myocardial Infarcion
C. Sudden Death

A. UNSTABLE ANGINA/NON ST ELEVATION MI (NSTEMI)

Definition
- ❑ accelerating pattern of pain
    - increased frequency
    - longer duration
    - occurring with less exertion
    - less responsive to treatment (eg. require higher doses or more frequent doses)
- ❑ angina at rest
- ❑ new onset angina
- ❑ angina post-MI
- ❑ post-angiography
- ❑ post-CABG
- ❑ note that unstable angina is a heterogeneous group and can be divided into a higher and lower risk groups

Significance
❑ thought to represent plaque rupture and acute thrombosis with incomplete vessel occlusion

Diagnosis
❑ history
❑ ECG changes
- ST depression or elevation
- T wave inversion

❑ no elevation of cardiac enzymes

Management
❑ oxygen
❑ hospitalization/monitoring
❑ bed rest
❑ anti-anginal medications
- sublingual or IV nitroglycerine
- β blockers are first line therapy
  - aim for resting heart rate of 50-60
- CCB are second line therapy (use if β blockers contraindicated, or if patient has refractory symptoms despite aggressive treatment with ECASA, nitrates, and β blockers)
  - evidence suggests that they do not prevent MI or decrease mortality
  - be cautious using verapamil/diltiazem with β blockers
  - use non-dihydropyridines if cannot use β blockers otherwise may use amlodipine or long-acting nifedipine if concomitant β blockade

❑ ECASA
- 160-325 mg/day

❑ IV heparin or Plavix (GPIIB/IIIA inhibitor)
❑ angiography with view to potential PTCA or CABG – used to map areas of ischemia
❑ if aggressive medical management is unsuccessful
- may use intra-aortic balloon pump (IABP) to stabilize before proceeding with revascularization – used to increase coronary perfusion during diasole
- proceed to emergency angiography and PTCA or CABG

B. ACUTE ST ELEVATION MYOCARDIAL INFARCTION (STEMI)

Definition
❑ syndrome of acute coronary insufficiency resulting in death of myocardium

Infarct Diagnosis Based on 2 of 3 - History, ECG, Cardiac Enzymes
❑ history
- sudden onset of characteristic chest pain for > 30 minutes duration
- may be accompanied by symptoms of heart failure (e.g. SOB, leg edema, etc.)

❑ ECG changes
- criteria: ST elevation of at least 1 mm in limb leads and 2 mm in precordial leads
- evolution of ECG changes in Q-wave MI
  - 1st – abnormal T waves
  - 2nd – ST-T elevations (hours post-infarct)
  - 3rd – significant Q waves (hours to days post-infarct)
  - 4th – inverted T waves, or may become flat or biphasic (days to weeks)

❑ cardiac enzymes
- follow CK-MB q8h x 3, Troponin q8h x 3

❑ cardiac Troponin I and/or T levels provide useful diagnostic, prognostic information and permit early identification of an increased risk of mortality in patients with acute coronary syndromes
- Troponin I and T remain elevated for 5 to 7 days

❑ beware
- up to 30% are unrecognized or "silent" due to atypical symptoms
- DM
- elderly
- patients with HTN
- post heart-transplant (because of denervation)

❑ draw serum lipids within 24-48 hours because the serum values are unreliable after 48 hours, but become reliable again 8 weeks post-MI

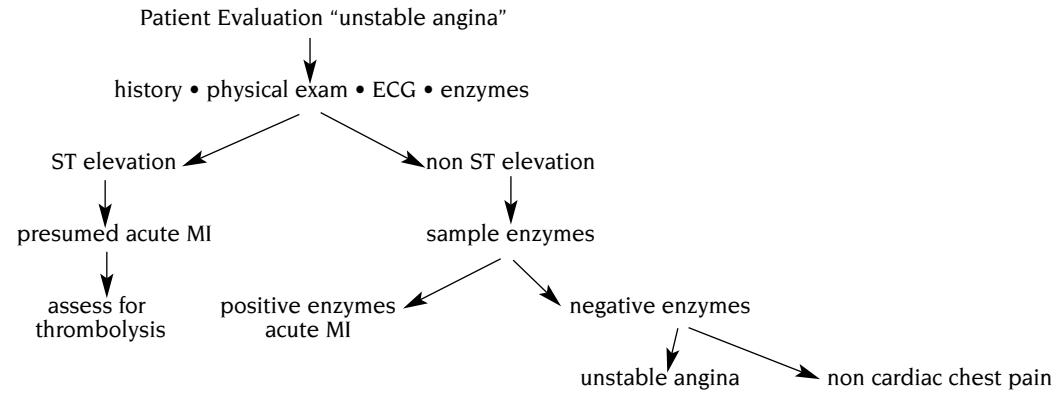


Figure 11. Diagnostic algorithm in acute IHD

Etiology

- ❑ coronary atherosclerosis + superimposed thrombus on ruptured plaque (vast majority)
  - vulnerable "soft" plaques more thrombogenic
- ❑ coronary thromboembolism
  - infective endocarditis
  - rheumatic heart disease
  - intracavity thrombus
  - cholesterol emboli
- ❑ severe coronary vasospasm
- ❑ arteritis
- ❑ coronary dissection
- ❑ consider possible exacerbating factors
  - see Angina Pectoris section

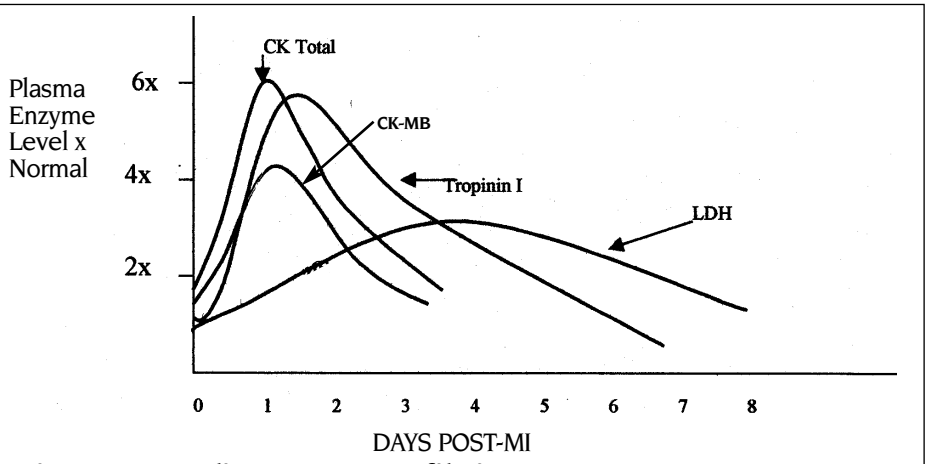


Figure 12. Cardiac Enzyme Profile in Acute MI

Further Classification of MIs

- ❑ Q wave
  - associated with transmural infarctions, involving full thickness of myocardium
- ❑ non-Q wave
  - usually associated with non-transmural (subendocardial) infarctions, involving 1/3 to 1/2 of myocardial thickness
  - in-hospital mortality from non-Q wave infarction is low (< 5%) but 1 year mortality approaches that of Q wave infarction

Management

- ❑ goal is to minimize the amount of infarcted myocardium and prevent complications
- ❑ **emergency room measures**
  - ECASA 325 mg chewed stat
  - oxygen
  - sublingual nitroglycerine
  - morphine for pain relief, sedation, and venodilation
  - β blockers to reduce heart rate if not contraindicated

❑ thrombolytic therapy (see Table 7)
- benefits of thrombolysis shown to be irrespective of age, sex, BP, heart rate, or history of MI or DM
- indications for thrombolytic therapy
  - A. at least 0.5 hours of ischemic cardiac pain and
  - B. any of the following ECG changes thought to be of acute onset
    - at least 1 mm of ST elevation in at least two limb leads
    - at least 1 mm of ST elevation in at least two adjacent precordial leads or
    - new onset complete BBB
  - C. presentation within 12 hours of symptom onset
    - choice of thrombolytic agents include streptokinase and rt-PA
    - patients having previously received streptokinase must receive alternate agent due to development of immunity

❑ heparin
❑ PTCA, CABG

Long-Term Measures
❑ antiplatelet/anticoagulation therapy
❑ ECASA 325 mg daily
❑ nitrates
- alleviate ischemia but may not improve outcome

❑ β blockers (first line therapy)
- start immediately and continue indefinitely if no contraindications
- decrease mortality

❑ CCB
- NOT recommended as first line treatment - Short Acting Nifedipine is contraindicated!
- Diltiazam and Verapamil are contraindicated in MI with associated LV dysfunction

❑ ACEI
- decrease mortality
- stabilize endothelium and prevent adverse ventricular remodeling
- strongly recommended for
  - symptomatic CHF
  - reduced LVEF (< 40%) starting day 3 to 16 post-MI (SAVE trial)
  - anterior MI

❑ lipid lowering agent (HMG-C0A reductase inhibitors or niacin)
- if total cholesterol > 5.5 or LDL > 2.6

❑ coumadin (for 3 months)
- for large anterior MI, especially if LV thrombus seen on 2D-ECHO

❑ see Figure 13 for post critical care unit (CCU) strategy

Table 7. Contraindications to Thrombolytic Therapy in AMI

Absolute	Relative
• active bleeding • aortic dissection • acute pericarditis • cerebral hemorrhage (previous or current)	• GI, GU hemorrhage or stroke within past 6 months • major surgery or trauma within past 2-4 weeks • severe uncontrolled hypertension • bleeding diathesis or intracranial neoplasm • puncture of a noncompressible vessel • significant chest trauma from CPR

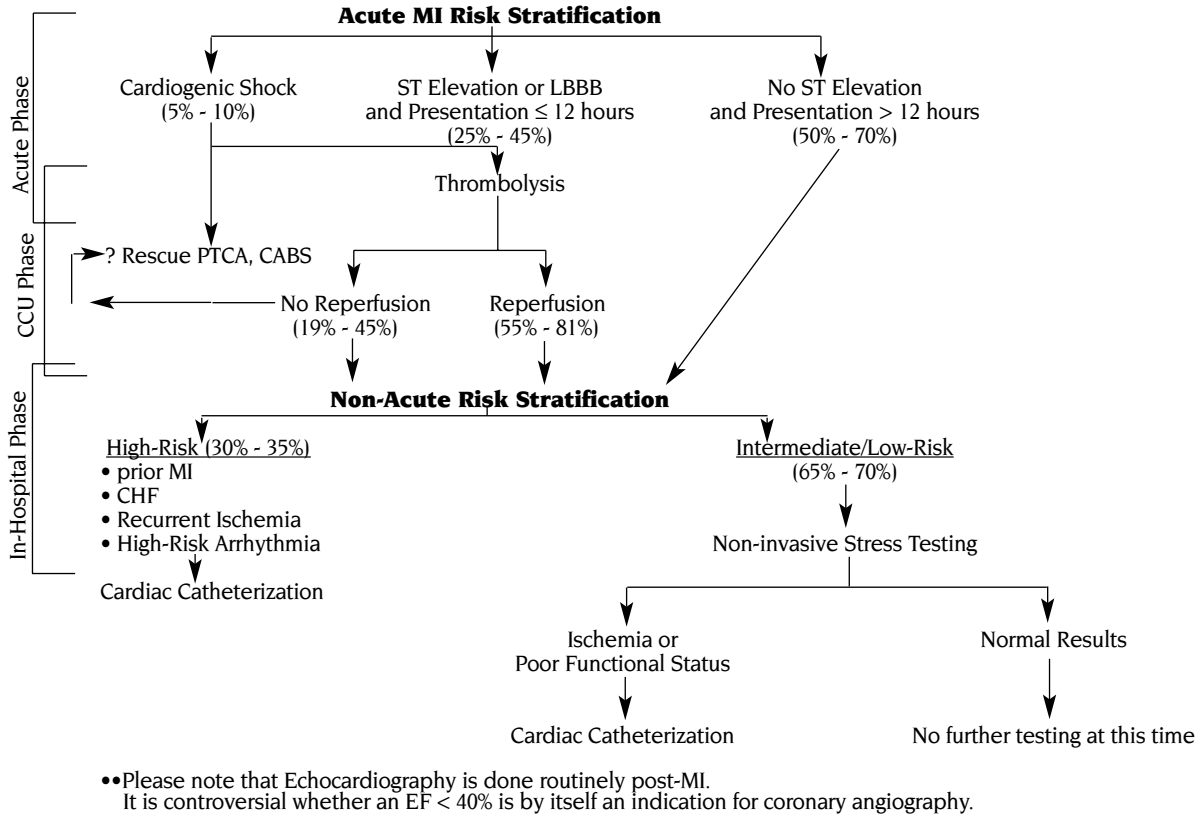
❑ Indications for Post-thrombolysis Heparin
- tPA used for thrombolysis
- Anterior MI
- Ventricular aneurysm
- Post-thrombolysis angina
- A fib
- Previous deep vein thrombosis (DVT), PE, or ischemic stroke

Prognosis
❑ 20% of patients with acute MI die before reaching hospital
❑ 5-15% of hospitalized patients will die
- risk factors
  - infarct size/severity
  - age
  - co-morbid conditions
  - development of heart failure or hypotension

❑ post-discharge mortality rates
- 6-8% within first year, half of these within first 3 months
- 4% per year following first year
- risk factors
  - LV dysfunction
  - residual myocardial ischemia
  - ventricular arrhythmias
  - history of prior MI
- resting LVEF is most useful prognostic factor

Table 8. Complications of Myocardial Infarction

Complication	Etiology	Presentation	Therapy
Arrhythmia			
(a) tachycardia	sinus, AF, VT, VF	early/late	see Arrhythmia section
(b) bradycardia	sinus, AV block	early	
Myocardial Rupture			
(a) LV free wall	transmural infarction	1-7 days	pericardiocentesis or surgery
(b) papillary muscle (MR)	inferior infarction anterior infarction	1-7 days	surgery
(c) ventricular septum (VSD)	septal infarction	1-7 days	surgery
Shock/CHF	LV/RV infarction aneurysm	within 48 hours	fluids, inotropes, IABP
Post Infarct Angina	persistent coronary stenosis multivessel disease	anytime	aggressive medical therapy PTCA or CABG
Recurrent MI	reocclusion	anytime	see above
Thromboembolism	mural thrombus in Q wave infarction	7~10 days, up to 6 months	heparin, warfarin
Pericarditis (Dressler's)	post-MI autoimmune (Dressler's)	1-7 days 2-8 weeks	NSAIDs NSAIDs, steroids



••Please note that Echocardiography is done routinely post-MI.
It is controversial whether an EF < 40% is by itself an indication for coronary angiography.

Figure 13. Acute MI and Predischarge Risk Stratification

C. SUDDEN DEATH

Definition
- ❑ unanticipated, non-traumatic death in a clinically stable patient, within 1 hour of symptom onset
- ❑ immediate cause of death is
  - V fib (most common)
  - ventricular asystole

Significance
- ❑ accounts for ~ 50% of CAD mortalities
- ❑ initial clinical presentation in up to 20% of patients with CAD

Etiology
- ❑ primary cardiac pathology
  - ischemia/MI
  - LV dysfunction
  - severe ventricular hypertrophy
    - hypertrophic cardiomyopathy (HCM)
    - AS
  - QT prolongation syndrome
  - congenital heart disease
- ❑ high risk patients
  - multi-vessel disease
  - LV dysfunction

Management

Acute
- ❑ resuscitate with prompt CPR and defibrillation

Long Term Survivors
- ❑ identify and treat underlying predisposing factors
- ❑ IHD
  - cardiac catheterization to evaluate cardiac anatomy, LV function and need for revascularization
- ❑ Holter monitoring
- ❑ electrophysiologic studies

Treatment
- ❑ antiarrhythmic drug therapy
  - amiodarone, β blockers
- ❑ surgery
  - revascularization to treat ischemia
  - map-guided subendocardial resection
  - cryoablation, radiofrequency ablation
- ❑ implantable cardioverter-defibrillator

Prognosis
- ❑ 1 year mortality post-resuscitation 20-30%
- ❑ predictors of recurrent cardiac arrest in the "survivor" of sudden cardiac death
  - remote MI
  - CHF
  - LV dysfunction
  - extensive CAD
  - complex ventricular ectopy
  - abnormal signal-averaged ECG

HEART FAILURE

- ❑ overall, CHF is associated with a 50% mortality rate at five years
- ❑ **see Colour Atlas R3 and R4**

Definitions and Terminology
- ❑ inability of heart to maintain adequate cardiac output to meet the demands of whole-body metabolism and/or to be able to do so only from an elevated filling pressure(forward heart failure)
- ❑ inability of heart to clear venous return resulting in vascular congestion (backward heart failure)
- ❑ either the left side of the heart (left heart failure) or the right side of the heart (right heart failure) or both (biventricular failure) may be involved
- ❑ there may be components of ineffective ventricular filling (diastolic dysfunction) and/or emptying (systolic dysfunction)
- ❑ most cases associated with poor cardiac function (low-output heart failure) but some are not due to intrinsic cardiac disease (high-output heart failure; this is discussed separately below)
- ❑ CHF is not a disease itself - it is a syndrome involving variable degrees of both forward and backward heart failure

Pathophysiology
- ❑ two components
  - primary insults initiating the disease process
  - compensatory responses which exacerbate and perpetuate the disease process in chronic heart failure

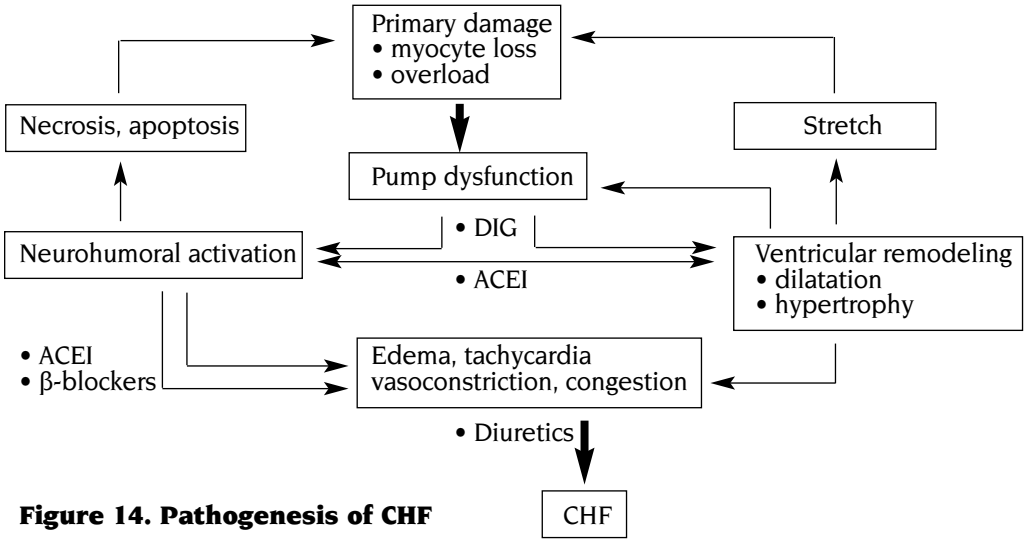


Figure 14. Pathogenesis of CHF

Clinical Pearl
- ❑ **What are the five commonest causes of CHF?**
  - **coronary artery disease (60-70%)**
  - **idiopathic (20%) often in the form of dilated cardiomyopathy**
  - **valvular (e.g. AS, AR and MR)**
  - **HTN**
  - **alcohol (may cause dilated cardiomyopathy)**

Etiologies of Primary Insults
- ❑ consider predisposing, precipitating and perpetuating factors
- ❑ the less common causes of CHF
  - toxic e.g. adriamycin, doxorubicin, radiation, uremia, catecholamines
  - infectious e.g. Chagas disease(very common cause worldwide), Coxsackie, HIV
  - endocrine e.g. hyperthyroidism, DM, acromegaly
  - infiltrative e.g. sarcoidosis, amyloidosis, hemochromatosis
  - genetic e.g. hereditary hypertrophic cardiomyopathy, Freidriech's Ataxia
  - metabolic e.g. thiamine deficiency, selenium deficiency
  - peripartum
- ❑ precipitants (**H-E-A-R-T F-A-I-L-E-D**)
  - **H** - HTN (common)
  - **E** - endocarditis/environment (e.g. heat wave)
  - **A** - anemia
  - **R** - rheumatic heart disease and other valvular disease
  - **T** - thyrotoxicosis
  - **F** - failure to take meds (very common)
  - **A** - arrhythmia (common)
  - **I** - infection/ischemia/infarction (common)
  - **L** - lung problems (PE, pneumonia, COPD)
  - **E** - endocrine (pheochromocytoma, hyperaldosteronism)
  - **D** - dietary indiscretions (common)
- ❑ it is important to differentiate an exacerbation due to a reversible cause from progression of the primary disease for treatment and prognosis

COMPENSATORY RESPONSES IN HEART FAILURE
- ❑ cardiac response to myocardial stress
  - pressure overload results in hypertrophy (e.g. HTN)
  - volume overload results in cardiac dilatation (e.g. AR)
- ❑ systemic response to ineffective circulating volume
  - activation of sympathetic nervous and renin-angiotensin systems result in
    - salt and H_2O retention with intravascular expansion
    - increased increased heart rate and myocardial contractility
    - increased afterload
- ❑ "compensated" heart failure becomes "decompensated" as cardiac and systemic responses overshoot
- ❑ treatments are directed at these compensatory overshoots

SYSTOLIC vs. DIASTOLIC DYSFUNCTION

Systolic Dysfunction (impaired ejection of blood from the heart)
- ❑ impaired myocardial contractile function
- ❑ hallmark is impaired stroke volume and/or ejection fraction
- ❑ symptoms predominantly due to decreased cardiac output
- ❑ examples
  - MI
  - myocarditis
  - dilated cardiomyopathy

Diastolic Dysfunction (defect in ventricular filling)
- ❑ 1/3 of all patients evaluated for clinical diagnosis of heart failure have normal systolic function (ejection fraction (EF))
- ❑ ability of LV to accept blood is impaired due decreased compliance
  - transiently by ischemia
  - permanently by severe hypertrophy (HTN, AS), infiltrative disease, MI (due to scarring) or HCM
- ❑ ischemia causes stiffness of LV because relaxation of myocardium is active and requires energy/ATP
  - increased LV filling pressures produce venous congestion upstream (i.e. pulmonic and systemic venous congestion)
- ❑ clues to diagnosis: S_4, HTN, LVH on ECG/ECHO, normal-size heart on CXR, normal EF
- ❑ apex beat sustained but not displaced
  - treatment: β blockers, verapamil, diltiazem or ACEI

Table 9. Signs and Symptoms of L vs. R Heart Failure

	Left Failure	Right Failure
low cardiac output (forward)	fatigue syncope systemic hypotension cool extremities slow capillary refill peripheral cyanosis MR Cheyne-Stokes breathing pulsus alternans S_3	TR S_3 (right-sided)
venous congestion (backward)	dyspnea orthopnea PND basal crackles cough hemoptysis	peripheral edema hepatomegaly hepatic tenderness pulsatile liver increased JVP positive HJR Kussmaul's sign

SLEEP-DISORDERED BREATHING
- ❑ 45-55% of patients with CHF (systolic and diastolic heart failure) have sleep disturbances, which include Cheyne-Stokes breathing, central and obstructive sleep apnea
- ❑ associated with a worse prognosis and greater LV dysfunction
- ❑ nasal continuous positive airway pressure (CPAP) is effective in treating Cheyne-Stokes respiration/sleep apnea with improvement in cardiac function and symptoms

HIGH-OUTPUT HEART FAILURE
- ❑ a variety of factors may create a situation of relative heart failure by demanding a greater than normal cardiac output for a variety of reasons
- ❑ rarely causes heart failure in itself but often exacerbates existing heart failure or puts a patient with other cardiac pathology "over the edge"
- ❑ DDx: anemia, thiamine deficiency, hyperthyroidism, A-V fistula, Paget's disease of bone

Investigations
- ❑ work up involves assessment for precipitating factors and treatable causes of CHF
- ❑ blood work
  - CBC (increased WBC - possible infectious precipitant; decreased Hb - anemia as a precipitant/exaccerbating factor)
  - electrolytes
    - dilutional (hypervolemic) hyponatremia indicates end-stage CHF
      - sign of neurohormonal activation and poorer prognosis
    - hypokalemia secondary to high renin state
  - BUN, Cr
    - may be elevated due to prerenal insult
    - be wary of ATN with diuretic therapy

- ❑ ECG
  - chamber enlargement
  - abnormal rhythms
  - ischemia/infarction
- ❑ chest x-ray
  - signs of pulmonary congestion
    - peribronchiolar cuffing
    - vascular redistribution
    - Kerley B Lines
    - interstitial pattern
    - fluid in lung fissures
    - alveolar filling if gross pulmonary edema
  - also look for
    - cardiomegaly (cardiac/thoracic (C/T) > 0.5)
    - atrial enlargement
    - pericardial effusion
    - pleural effusion
- ❑ echocardiography is the primary diagnostic method to determine
  - EF (LV Grade I (EF = 60%), II (40-59%), III (21-39%), IV (= 20%)
  - atrial or ventricular dimensions
  - wall motion abnormalities
  - valvular stenosis or regurgitation
  - pericardial effusion
- ❑ radionuclide angiography (MUGA) provides more accurate ejection fraction measurements than echocardiography; however, it provides little information on valvular abnormalities
- ❑ myocardial perfusion scintigraphy (Thallium or Sestamibi SPECT)
  - determines areas of fibrosis/infarct or viability
- ❑ angiogram in selected patients

Long-term Management of CHF

- ❑ short term goals of therapy are to relieve symptoms and improve the quality of life
- ❑ long term goal is to prolong life by slowing, halting, or reversing the progressive LV dysfunction
- ❑ treat the cause/aggravating factors
- ❑ symptomatic measures
  - oxygen, bed rest, elevation of head of bed
- ❑ control of sodium and fluid retention
  - sodium restriction (2 gm/d), requires patient education
  - fluid restriction and monitor daily weights
  - diuretics - for symptom control, mortality benefit demonstrated with spironolactone (RALES study)
    - furosemide (40-500 mg/day) for potent diuresis
    - metalozone may be used with furosemide to increase diuresis
- ❑ vasodilators
  - goal is to arteriodilate (decrease afterload) and venodilate (increase preload), thereby improving cardiac output and venous congestion
  - in hospital, monitor response to therapy with daily weights and measurement of fluid balance and follow renal function
  - ACEI: standard of care (improves survival)
    - strongly recommended for
      - all symptomatic patients
      - all asymptomatic patients with LVEF < 35%
      - post-MI setting if
        - symptomatic heart failure
        - asymptomatic LVEF < 40%
        - anterior MI
      - clearly shown to decrease mortality and slow progression in these settings
  - hydralazine and nitrates
    - second line to ACEI
    - decrease in mortality not as great as with ACEI
  - amlodipine
    - may be of benefit in dilated cardiomyopathy
  - angiotensin II receptor blockers e.g. losartan
    - preliminary evidence suggests benefit
- ❑ inotropic support
  - digitalis
    - inhibits Na/K ATPase leading to decreased Na/Ca exchange and increased intracellular [Ca^{2+}], hence increasing myocardial contractility
    - improves symptoms and decrease hospitalizations (DIG trial); patients on digitalis glycosides may worsen if these are withdrawn
    - no impact on survival
    - excellent choice in setting of CHF with atrial fibrillation

- ❑ other agents
  - β blockers - recommended for functional class (FC) II-III patients
    - should be used cautiously, titrate slowly because may initially worsen CHF
    - postulated that these agents interfere with neurohormonal activation
    - carvedilol confers survival benefit in FC II-III CHF
    - metoprolol has been shown to delay time to transplant, decreased hospitalizations in dilated cardiomyopathy and to decrease mortality (MERIT study)
  - CCB (have equivocal effect on survival)
  - antiarrhythmic drugs
    - if required, amiodarone is drug of choice
    - class I anti-arrhythmics associated with increased mortality in CHF

ACUTE CARDIOGENIC PULMONARY EDEMA

Definition

- ❑ left-sided backward heart failure leading to severe pulmonary congestion with extravasation of capillary fluid into the pulmonary interstitium and alveolar space

Clinical Manifestations

- ❑ tachycardia, tachypnea, diaphoresis
- ❑ severe left-sided venous congestion

Management, use mnemonic "LMNOP"

- ❑ make sure to treat any acute precipitating factors (e.g. ischemia, arrhythmias)
- ❑ sit patient up with legs hanging down if blood pressure is adequate
- ❑ L - Lasix - furosemide 40 mg IV, double dose q1h as necessary
- ❑ M - Morphine 2-4 mg IV q5-10 minutes
  - decreased anxiety
  - vasodilation
- ❑ N - Nitroglycerine topical 2 inches q2h (or IV)
- ❑ O - Oxygen
- ❑ P - Positive airway pressure
  - (CPAP or BiPAP) decreased need for ventilation and decreased preload
- ❑ other vasodilators as necessary in ICU setting
  - nitroprusside (IV)
  - hydralazine (PO)
  - sympathomimetics
    - potent agents used in ICU/CCU settings
    - dopamine
      - agonist at dopamine D1 (high potency), β1-adrenergic (medium potency), and α1-adrenergic receptors (low potency)
      - "low-dose" causes selective renal vasodilation (D1 agonism)
    - "medium-dose" provides inotropic support (β1 agonism)
    - "high-dose" increase systemic vascular resistance (SVR), which in most cases is undesirable (α1 agonism)
  - dobutamine
    - acts at β1 and α1 adrenoceptors
    - selective inotropic agent (β1 agonism)
    - also produces arterial vasodilation (α1 antagonism)
  - phosphodiesterase inhibitors (amrinone, Inocor)
    - effects similar to dobutamine (inhibits PDE —> cAMP —> inotropic effect and vascular smooth muscle relaxation (decrease SVR)
    - adverse effect on survival when used as long-term oral agent
- ❑ inotropic support (dopamine, dobutamine) if necessary
- ❑ consider PA line to monitor capillary wedge pressure
- ❑ consider mechanical ventilation if needed
- ❑ rarely used but potentially life-saving measures
  - rotating tourniquets
  - phlebotomy

CARDIAC TRANSPLANTATION

- ❑ indications - end stage cardiac disease (CAD, DCM, etc.)
  - failure of maximal medical/surgical therapy
  - poor 6 month prognosis
  - absence of contraindications
  - ability to comprehend and comply with therapy
- ❑ 1 year survival 85%, 5 year survival 70%
- ❑ complications: rejection, infection, graft vascular disease, malignancy

CARDIOMYOPATHIES

Definition
- ❑ intrinsic myocardial disease not secondary to CAD, valvular heart disease, congenital heart disease, HTN or pericardial disease
- ❑ The diagnosis of any of the following mandates exclusion of the above conditions:
  - dilated cardiomyopathy (DCM)
  - hypertrophic cardiomyopathy (HCM)
  - restrictive cardiomyopathy (RCM)
  - myocarditis

DILATED CARDIOMYOPATHY

Etiology
- ❑ idiopathic (risk factors: male, black race, family history)
- ❑ alcohol
- ❑ inflammatory (subsequent to myocarditis)
- ❑ collagen vascular disease: SLE, PAN, dermatomyositis, progressive systemic sclerosis
- ❑ infectious: post-viral (Coxsackie), Chagas disease, Lyme disease, Rickettsial diseases, acute rheumatic fever
- ❑ neuromuscular disease: Duchenne muscular dystrophy, myotonic dystrophy, Friedreich ataxia
- ❑ metabolic: uremia, nutritional deficiency (thiamine, selenium, carnitine)
- ❑ endocrine: thyrotoxicosis, DM
- ❑ familial
- ❑ peripartum
- ❑ toxic: cocaine, heroine, glue sniffing, organic solvents
- ❑ radiation induced
- ❑ drugs: chemotherapeutics (adriamycin)

Pathophysiology
- ❑ clinical manifestations
  - CHF
  - systemic or pulmonary emboli
  - arrhythmias
  - sudden death (major cause of mortality due to fatal arrhythmia)

Investigations
- ❑ 12 lead ECG
  - ST-T wave abnormalities
  - poor R wave progression
  - conduction defects (e.g. BBB)
  - arrhythmias
- ❑ chest x-ray
  - global cardiomegaly (globular heart)
  - signs of CHF
- ❑ echocardiography
  - 4-chamber enlargement
  - depressed ejection fraction
  - MR and TR secondary to cardiac dilatation
- ❑ endomyocardial biopsy: not routine, used to diagnose infiltrative RCM and myocarditis, or to rule out a treatable cause
- ❑ angiography: selected patients

Natural History
- ❑ prognosis
  - depends on etiology
  - generally inexorable progression
  - overall once CHF - 50% 5 year survival
  - cause of death usually CHF or sudden death
  - systemic emboli are significant source of morbidity

Management
- ❑ treat underlying disease - e.g. abstinence from EtOH
- ❑ treat CHF (see Heart Failure section), β blockade (e.g. metoprolol, carvedilol) and ACEI (+/– AII receptor inhibitors) to decrease remodeling
- ❑ anticoagulation to prevent thromboembolism (coumadin)
  - absolute - A fib, history of thromboembolism or documented thrombus
  - clinical practice is to anticoagulate if EF < 20%
- ❑ treat symptomatic or serious arrhythmias
- ❑ immunize against influenza and pneumococcus
- ❑ surgical therapy
  - cardiac transplant - established definitive therapy
  - LVAD
  - volume reduction surgery (role remains unclear)
  - cardiomyoplasty (latissimus dorsi wrap)

HYPERTROPHIC CARDIOMYOPATHY (HCM)

Pathophysiology
- ❑ defined as unexplained ventricular hypertrophy (not due to systemic HTN or AS). Histopathologic features are myocardial fiber disarray, myocyte hypertrophy, and interstitial fibrosis
- ❑ cause is felt to be a genetic defect involving 1 of the cardiac sarcomeric proteins (> 100 mutations associated with development of autosomal dominant inheritance)
- ❑ clinical manifestations
  - asymptomatic
  - dyspnea
  - angina
  - presyncope/syncope- LV outflow obstruction or arrhythmia
  - CHF
  - arrhythmias
  - sudden death (may be first manifestation)

Henodynamic Classification
- ❑ hypertrophic obstructive cardiomyopathy (HOCM): dynamic outflow tract (LVOT) obstruction
  - either resting or provocable LVOT obstruction
- ❑ nonobstructive hypertrophic cardiomyopathy: decreased compliance and diastolic dysfunction (impaired filling)
- ❑ complications: obstruction, arrhythmia, diastolic dysfunction

Hallmark Signs of HOCM
- ❑ pulses
  - rapid upstroke pulse
  - bifid pulse
- ❑ precordial palpation
  - PMI: localized, sustained, double impulse, 'triple ripple' (triple apical impulse)
- ❑ precordial auscultation
  - normal or paradoxically split S_2
  - S_4
  - harsh, systolic, diamond-shaped murmur at LLSB or apex, enhanced by squat to standing or valsalva (murmur secondary to LVOT obstruction and asociated mitral regurgitation)

Table 10. Factors Influencing Obstruction in Hypertrophic Cardiomyopathy

Increased Obstruction (decreased murmur)	Decreased Obstruction (decreased murmur)
inotropes, vasodilators, diuretics	negative inotropes
hypovolemia	vasoconstrictors
tachycardia	volume expansion
squat to standing position	bradycardia
Valsalva maneuver	squatting from standing position
Amylnitrite inhalation	sustained handgrip (isometrics)

Investigations
- ❑ 12 lead ECG
  - LVH
  - prominent Q waves or tall r wave in V_1
- ❑ echocardiography
  - LVH - asymmetric septal hypertrophy (most common presentation)
  - systolic anterior motion (SAM) of anterior MV leaflet
  - resting or dynamic ventricular outflow tract obstruction
  - MR (due to SAM and associated with LVOT obstruction)
  - diastolic dysfunction
  - LAE
- ❑ cardiac catheterization
  - increased LV end-diastolic pressure
  - variable systolic gradient across LV outflow tract

Natural History
- ❑ variable
- ❑ potential complications: A fib, VT, CHF, sudden death
- ❑ risk factors for sudden death
  - most reliable
    - history of survived cardiac arrest/sustained VT
    - family history of multiple sudden deaths
  - other factors associated with increased risk of sudden cardiac death (SCD)
    - syncope
    - VT on ambulatory monitoring
    - marked ventricular hypertrophy
  - prevention of sudden death in high risk patients
    = amiodarone or implantable cardioverter defibrillator (ICD)

Management
- ❑ avoid extremes of excertion
- ❑ avoid factors which increase obstruction
- ❑ infective endocarditis prophylaxis for patients with obstructive HCM
- ❑ treatment of obstructive HCM
  - medical agents
    - β blockers
    - disopyramide
  - CCB only used in patients with no resting/provocable obstruction
  - patients with drug-refractory symptoms
    - options
      1. surgical myectomy
      2. septal ethanol ablation
      3. dual-chamber pacing
- ❑ treatment of ventricular arrhythmias - AMIO or ICD
- ❑ adult first-degree relatives of patients with HCM should be screened (physical exam, ECG, 2D-ECHO) serially every 5 years

RESTRICTIVE CARDIOMYOPATHY (RCM)

Etiology
- ❑ infiltrative
  - amyloidosis (especially in primary amyloidosis associated with light chain disease), sarcoidosis
- ❑ non-infiltrative
  - scleroderma, idiopathic myocardial fibrosis
- ❑ storage diseases
  - hemochromatosis (especially in DM, cirrhosis), Fabry's disease, glycogen storage diseases
- ❑ endomyocardial
  - endomyocardial fibrosis (Africans), eosinophilic: Loeffler's endocarditis or eosinophilic endomyocardial disease
  - radiation heart disease
  - pseudoxanthoma elasticum
  - carcinoid syndrome (associated TV or PV dysfunction)

Pathophysiology
- ❑ infiltration of the myocardium —> decreased ventricular compliance —> diastolic dysfunction
- ❑ clinical manifestations
  - CHF - diastolic dysfunction predominates
  - arrhythmias

Investigations
- ❑ 12 lead ECG
  - low voltage
  - non-specific, diffuse ST-T wave changes (no correspondence with vascular territory) +/– nonischemic Q waves
- ❑ chest x-ray
  - mild cardiac enlargement
- ❑ echocardiography
  - normal pericardium, normal or only slightly decreased systolic function, impaired ventricular filling and diastolic dysfunction
- ❑ cardiac catheterization
  - end-diastolic ventricular pressures
- ❑ endomyocardial biopsy to distinguish etiology (especially for infiltrative RCM)

Natural History
- ❑ depends on etiology
- ❑ generally poor prognosis

Management
- ❑ exclude constrictive pericarditis
- ❑ treat underlying disease
- ❑ supportive care
- ❑ treat coexisting CHF, arrhythmias
- ❑ anticoagulation
- ❑ consider cardiac transplantation - depending on etiology

MYOCARDITIS

❑ inflammatory process involving the myocardium (an important cause of dilated cardiomyopathy)

Etiology

❑ idiopathic
❑ infectious
- viral: Coxsackie virus B, Echovirus, Poliovirus, HIV, mumps
- bacterial: S. aureus, C. *perfringens*, C. *diphtheriae*, Mycoplasma, Rickettsia
- fungi
- spirochetal (Lyme disease – B*orrelia burgdorferi*)
- Chagas disease (T*rypanosoma cruzi*), toxoplasmosis

❑ acute rheumatic fever (Group A β-hemolytic S*treptococcus*)
❑ drug-induced: emetine, doxorubicin
❑ collagen vascular disease: systemic lupus erythematosus (SLE), polyarteritis nodosa (PAN), rheumatoid arthritis (RA), dermatomyositis (DMY)
❑ sarcoidosis
❑ giant cell myocarditis

Clinical Manifestations

❑ constitutional illness
❑ acute CHF
❑ chest pain - associated pericarditis or cardiac ischemia
❑ arrhythmias (may have associated inflammation of conduction system)
❑ systemic or pulmonary emboli
❑ sudden death

Investigations

❑ 12 lead ECG
- non-specific ST-T changes +/– conduction defects

❑ blood work
- increased CK, Troponin, LDH, and AST with acute myocardial necrosis +/– increased WBC, ESR, ANA, rheumatoid factor, complement levels

❑ perform blood culture, viral titers and cold agglutinins for Mycoplasma
❑ chest x-ray
- enlarged cardiac silhouette

❑ echocardiography
- dilated, hypokinetic chambers
- segmental wall motion abnormalities

Natural History

❑ usually self-limited and often unrecognized
❑ most recover
❑ may be fulminant with death in 24-48 hours
❑ sudden death in young adults
❑ may progress to dilated cardiomyopathy
❑ few may have recurrent or chronic myocarditis

Management

❑ supportive care
❑ restrict physical activity
❑ treat CHF
❑ treat arrhythmias
❑ anticoagulation
❑ treat underlying cause if possible

VALVULAR HEART DISEASE

❑ see Cardiac Surgery Chapter

INFECTIVE ENDOCARDITIS (IE)

Etiology
❑ *Strep viridans* (commonest, spontaneous bacterial endocarditis (SBE) on abnormal valve – prosthetic, MVP, etc.)
❑ *Enterococcus* (Group D strep, SBE)
❑ *Staph aureus* (enter through break in skin: IV drug abusers, usually rightsided, catheter-associated sepsis)
❑ *Staphylococcus epidermidis* (prosthetic valve)
❑ *Strep bovis* (underlying GI malignancy)
❑ others: gram-negative bacteria, Candida, HACEK organisms (*Haemophilus species*, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella*, and *Kingella*), *Pseudomonas* (IV drug)
❑ frequency of valve involvement: MV >> AV > TV > PV
❑ risk of IE in various cardiac lesions (JAMA 1997;227:1794)
- high risk: prosthetic heart valves, previous IE, complex cyanotic congenital heart disease, surgically constructed systemic to pulmonary shunts or conduits
- moderate risk: most other congenital cardiac malformations, acquired valvular dysfunction, HCM, MVP with MR and/or thickened leaflets

Pathogenesis and Symptomatology
❑ usually requires source of infection, underlying valve lesion, +/– systemic disease/immunocompromised state
❑ portal of entry: oropharynx, skin, GU, drug abuse, nosocomial infection —> bacteremia —> diseased valve/high flow across valve —> turbulence of blood across valve —> deposition of bacteria on endocardial surface of valve (vegetation = clump of fibrin, platelets, WBCs and bacteria) —> endocarditis —> septic embolization
❑ symptoms
- fever, chills, rigors
- night sweats
- 'flu-like' illness, malaise, headaches, myalgia, arthralgia
- dyspnea, chest pain

Signs
❑ fever, regurgitant murmur (new onset or increased intensity), constitutional symptoms, anemia
❑ signs of CHF (secondary to acute MR, AR)
❑ peripheral manifestations: petechiae, Osler's nodes ("ouch!" raised, painful, 3-15 mm, soles/palms), Janeway lesions ("pain away!" flat, painless, approx. 1-2 cm, on soles/plantar surfaces of toes/palms/fingers), splinter hemorrhages (especially on proximal nail bed, distally more commonly due to local trauma)
❑ CNS: focal neurological signs (CNS emboli), retinal Roth spots
❑ clubbing (subacute)
❑ splenomegaly (subacute)
❑ microscopic hematuria (renal emboli or glomerulonephritis) ± active sediment
❑ weight loss

Investigations
❑ blood work: anemia, uncreased ESR, positive rheumatoid factor
❑ serial blood cultures (definitive diagnosis)
❑ echocardiography (transesophageal > sensitivity than transthoracic)
- vegetations, degree of regurgitation valve leaflet perforation, abscess
- serial ECHO may help in assessing cardiac function

Natural History
❑ adverse prognostic factors
- CHF, Gram (–) or fungal infection, prosthetic valve infection, abscess in valve ring or myocardium, elderly, renal failure, culture negative IE

❑ mortality up to 30%
❑ relapses may occur - follow-up is mandatory
❑ permanent risk of re-infection after cure due to residual valve scarring

Complications
❑ CHF (usually due to valvular insufficiency)
❑ systemic emboli
❑ mycotic aneurysm formation
❑ intracardiac abscess formation leading to heart block
❑ renal failure: glomerulonephritis due to immune complex deposition; toxicity of antibiotics

Management
- ❑ medical
  - antibiotic therapy tailored to cultures (penicillin, gentamicin, vancomycin, cloxacillin) minimum of 4 weeks treatment
    - serial ECGs - increased PR interval
  - prophylaxis (JAMA 1997;227:1794)
    - dental/oral/respiratory/esophageal procedures
      - amoxicillin 2 g 1 hour prior
    - GU/GI (excluding esophageal) procedures
      - high risk: ampicillin + gentamicin
      - moderate risk: amoxicillin, ampicillin, or vancomycin
- ❑ surgical
  - indications: refractory CHF, valve ring abscess, valve perforation, unstable prosthesis, multiple major emboli, antimicrobial failure, mycotic aneurysm

RHEUMATIC FEVER

Epidemiology
- ❑ school-aged children (5-15 yr), young adults (20-30 yr), outbreaks of Group A β-hemolytic *Streptococcus*, upper respiratory tract infection (URTI), social factors (low socioeconomic status (SES), crowding)

Etiology
- ❑ 3% of untreated Group A β-hemolytic *Streptococcus* (especially mucoid, highly encapsulated stains, serotypes 5, 6, 18) pharyngitis develop acute rheumatic fever

Diagnosis
- ❑ 1. Modified Jones criteria (1992): 2 major, or 1 major + 2 minor
  - major criteria
    - pancarditis
    - polyarthritis
    - Sydenham's chorea
    - erythema marginatum
    - subcutaneous nodules
  - minor criteria
    - previous history of rheumatic fever or rheumatic heart disease
    - polyarthralgia
    - increased ESR or C-reactive protein (CRP)
    - increased PR interval (first degree heart block)
    - fever

  plus
- ❑ 2. Supported evidence confirming Group A *Streptococcus* infection: history of scarlet fever, group A streptococcal pharyngitis culture, rapid Ag detection test (useful if positive), anti-streptolysin O Titers (ASOT)

Clinical Features
- ❑ Acute Rheumatic Fever: myocarditis (DCM/CHF), conduction system(sinus tachycardia, A fib), valvulitis (acute MR), pericarditis (does not usually lead to constrictive pericarditis)
- ❑ Chronic: Rheumatic Valvular heart disease: fibrous thickening, adhesion, calcification of valve leaflets resulting in stenosis/regurgitation, increased risk of IE +/– thromboembolic phenomenon. Onset of symptoms usually after 10-20 year latency from acute carditis of rheumatic fever. Mitral valve most commonly affected.

Management
- ❑ acute treatment of Streptococcal infection (benzathine penicillin G 1.2 MU IM x 1 dose)
- ❑ prophylaxis to prevent colonization of URT (age < 40): benzathine penicillin G 1.2 MU IM q3-4 weeks, within 10 yr of attack
- ❑ management of carditis in rheumatic fever: salicylates (2g qid x4-6 wk for arthritis), corticosteroids (prednisone 30 mg qid x4-6wk for severe carditis with CHF)

AORTIC STENOSIS

Etiology
- ❑ congenital (bicuspid > > unicuspid) —> calcified degeneration or congenital AS
- ❑ acquired
  - degenerative calcified AS (most common) - "wear and tear"
  - rheumatic disease

Definition
- ❑ AS = narrowed valve orifice (aortic valve area: normal = 3-4 cm^2
  severe AS = < 1.0 cm^2
  critical AS = < 0.75 cm^2)
- ❑ Note: low gradient AS with severely reduced valve area (< 1.0 cm^2) and normal gradient in setting of LV dysfunction

Pathophysiology
- ❑ pressure overloaded LV: increased LV end-diastolic pressure (EDP), concentric LVH, subendocardial ischemia —> forward failure
- ❑ outflow obstruction: fixed cardiac output (CO)
- ❑ LV failure, pulmonary edema, CHF

Symptomatology
- ❑ ASD (triad of Angina, Syncope, and Dyspnea; prognosis associated with onset)
- ❑ angina (exertional): due to concentric LVH and subendocardial ischemia (decreased subendocardial flow and increased myocardial O_2 demand), may have limitation of normal activity or resting angina in tight AS (associated with < 5 year survival)
- ❑ syncope: due to fixed CO or arrhythmia (< 3 year survival)
- ❑ dyspnea (LV failure): systolic +/– diastolic dysfunction, pulmonary edema, may have orthopnea, if secondary RHF may have ascites, peripheral edema, congestive hepatomegaly (< 2 years)

Signs of AS
- ❑ pulses
  - apical-carotid delay
  - pulsus parvus et tardus (decreased amplitude and delayed upstroke) narrow pulse pressure, brachial-radial delay
  - thrill over carotid
- ❑ precordial palpation
  - PMI: sustained (LVH) +/– diffuse (displaced, late, with LV dilation)
  - +/– palpable S_4
  - systolic thrill in 2nd right intercostal space (RICS) +/– along left lower sternal bender (LLSB)
- ❑ precordial auscultation
  - most sensitive physical finding is SEM radiating to right articular head
  - SEM – diamond shaped (crescendo-decrescendo), harsh, high-pitched, peaks progressively later in systole with worsening AS, intensity not related to severity, radiates to neck, musical quality of murmur at apex (Gallavardin phenomenon)
  - +/– diastolic murmur of associated mild AR
  - S_2 – soft S_2, absent A_2 component, paradoxical splitting (severe AS)
  - ejection click
  - S_4 – early in disease (increased LV compliance)
  - S_3 – only in late disease (if LV dilatation present)

Investigations
- ❑ 12 lead ECG
  - LVH and strain +/– LBBB, LAE/A fib
- ❑ chest x-ray
  - post-stenotic aortic root dilatation, calcified valve, LVH + LAE, CHF (develops later)
- ❑ ECHO
  - test of choice for diagnosis and monitoring
  - valvular area and pressure gradient (assess severity of AS)
  - LVH and LV function
  - shows leaflet abnormalities and "jet" flow across valve
- ❑ cardiac catheterization
  - r/o CAD (i.e. especially before surgery in those with angina)
  - valvular area and pressure gradient (for inconclusive ECHO)
  - LVEDP and CO (normal unless associated LV dysfunction)

Natural History
- ❑ asymptomatic patients have excellent survival (near normal)
- ❑ once symptomatic, untreated patients have a high mean mortality
  - 5 years after onset of angina, < 3 years after onset of syncope; and < 2 years after onset of CHF/dyspnea
- ❑ the most common fatal valvular lesion (early mortality/sudden death)
  - ventricular dysrhythmias (likeliest cause of sudden death)
  - sudden onset LV failure
- ❑ other complications: IE, complete heart block

Management
- ❑ asymptomatic patients - follow for development of symptoms
  - serial echocardiograms
  - supportive/medical
    - avoid heavy exertion
    - IE prophylaxis
    - avoid nitrates/arterial vasodilators and ACEI in severe AS
- ❑ indications for surgery
  - onset of symptoms: angina, syncope, or CHF
  - progression of LV dysfunction
  - moderate AS if other cardiac surgery (i.e. CABG) required
- ❑ surgical options (see Cardiac and Vascular Surgery Chapter)
  - AV replacement
    - excellent long-term results, procedure of choice
  - open or balloon valvuloplasty
    - children, repair possible if minimal disease
    - adults (rarely done): pregnancy, palliative in patients with comorbidity, or to stabilize patient awaiting AV replacement - 50% recurrence of AS in 6 months after valvuloplasty
  - complications: low CO, bleeding, conduction block, stroke

AORTIC REGURGITATION (AR)

Etiology

- ❑ supravalvular (aortic root disease with dilatation of ascending aorta)
  - atherosclerotic dilatation and aneurysm; cystic medial necrosis annuloaortic ectasia (Marfan syndrome); dissecting aortic aneurysm; systemic HTN; (idiopathic Aortic root dilation); syphilis; connective tissue diseases (ankylosing spondylitis, psoriatic arthritis, Reiter syndrome, rheumatoid aortitis)
- ❑ valvular
  - congenital abnormalities (bicuspid AV, large VSD); connective tissue diseases (SLE, rheumatoid arthritis, etc.); rheumatic fever (+/– associated AS); IE; myxomatous degeneration; deterioration of prosthetic valve
- ❑ acute AR
  - IE
  - aortic dissection
  - trauma
  - acute rheumatic fever
  - failed prosthetic valve

Pathophysiology and Symptomatology

- ❑ AR = blood flow from aorta back into LV (diastolic run-off)
- ❑ volume overload —> LV dilatation —> increased SV and more diastolic run-off —> high SBP and low DBP (wide pulse pressure)
- ❑ LV dilatation combined with increased SBP —> increased wall tension = pressure overload —> LVH
  - symptoms
    - dyspnea/orthopnea/PND
    - fatigue and palpitations (arrhythmias or hyperdynamic circulation)
- ❑ decreased DBP —> decreased coronary perfusion; LVH —> increased myocardial O_2 demand
  - symptoms
    - syncope, angina (only if severe AR)
- ❑ usually symptomatic only after onset of LV failure (late in disease), LAE presents earlier onset of symptoms

Signs of chronic AR

- ❑ pulses
  - increased volume, Waterhammer (bounding and rapidly collapsing)
  - Bisferiens pulse - twice beating in systole; occurs inpresence of combined AS and AR
  - de Musset's sign - head bobbing due to increased PP
  - pistol-shot sounds over femoral artery (without compression)
  - Traube's sign: double sound heard with the stethoscope lightly applied over the artery
  - Quincke's sign - pulsatile blushing of nail beds (nonspecific)
  - Corrigan's pulse - visible carotid pulse
  - Hill's test:- femoral-brachial SBP difference > 20 (greater differences correlate with more severe AR)
  - Duroziez's test: light proximal compression of femoral artery produces systolic-diastolic murmur over femoral artery
  - other - pulsating uvula (Mueller), liver (Rosenbach), pupil (Gandolfi), or spleen (Gerhard)
- ❑ precordial palpation
  - heaving apex (hyperdynamic), displaced point of maximal impulse (PMI) (volume overload)
- ❑ precordial auscultation
  - S_1 - soft in severe AR (early closure of MV)
  - S_2 - soft or absent (severe AR), may be loud if calcified
  - S_3 in severe AR (early LV decompensation)
  - early diastolic decrescendo murmur (EDM) - high-pitched, at LLSB (cusp disease) or RLSB (aortic root disease), length correlates with severity, best heard with patient sitting, leaning forward on full expiration
  - systolic ejection murmur (SEM) (physiologic, high flow murmur)- in aortic area
  - Austin Flint murmur - diastolic rumble at apex, secondary to regurgitant jet on anterior MV leaflet
- ❑ acute AR - most of these signs are absent (SV not yet increased)
  - patient usually presents in CHF, tachycardia, soft S_1, soft or absent S_2, short early diastolic murmur, preclosure of MV (ECHO)

Investigations

- ❑ 12 lead ECG
  - LVH, LAE
- ❑ chest x-ray
  - LV enlargement, LAE, aortic root dilatation
- ❑ echocardiography (TTE)
  - gold standard for diagnosis and assessment of severity of AR
  - regurgitant jet from aorta into LV
  - association of aortic leaflet morphology, LV size, LVF, aortic root size
  - fluttering of anterior MV leaflet
  - Doppler most sensitive
- ❑ radionuclide imaging
  - serial resting and exercise EF (normal increased with exercise > 5%) for serial monitoring of patients with asymptomatic severe AR
  - sensitive sign of decreased LV function: failure to increase EF with exercise
- ❑ cardiac catheterization
  - coronary angiography indicated if age > 40
  - increased LV volume; CO normal or decreased (LV dysfunction); increased LVEDP

Natural History
- ❑ mild to moderate AR - few symptoms
- ❑ chronic progression to severe AR (may be asymptomatic more than 10 years)
- ❑ once symptomatic, prognosis is much worse
    - mean mortality 4 years after onset of angina, 2 years after CHF
- ❑ severe acute AR - only 10-30% live more than 1 year after diagnosis
- ❑ late complications: arrhythmias, CHF, IE

Management
- ❑ asymptomatic
    - follow with serial ECHO - assess LV size and function
    - +/– afterload reduction: nifedipine, ACE inhibitors
    - IE prophylaxis
- ❑ medical
    - restriction of activities
    - treat CHF (non-pharmacological, afterload reduction, Digoxin, and diuretics)
    - acute AR: may stabilize with IV vasodilators before surgery
- ❑ surgical
    - acute AR leading to LV failure - best treated surgically
    - chronic severe AR - indications for surgery (generally operate prior to onset of irreversible LV dysfunction):
        - symptomatic patients with chronic severe AR
        - progression of LV dilatation
        - consider if poor LVEF (< 55%) at rest, or failure to increase EF with exercise (with serial MUGA assessment)
- ❑ surgical options
    - AV replacement
        - mechanical, bioprosthetic, homograft, or sometimes pulmonary autograft (Ross procedure) valve may be used
    - valve repair (rare in AR)
        - subcommissural annuloplasty for annular dilatation

MITRAL STENOSIS

Etiology
- ❑ congenital (rare)
- ❑ acquired
    - RHD (most common) (especially developing nations; F > M):

Pathophysiology and Symptomatology
- ❑ normal MV area = 4-6 cm^2, hemodynamically significant MS with MV orifice < 2 cm^2
- ❑ MS = LV inlet obstruction —> LAE —> increased LA pressure —> increased pulmonary vascular resistance —> increased right-sided pressure —> right-sided CHF
    - symptoms (2-5 year progression from onset of serious symptoms to death, slower progression seen in the elderly)
    - early: SOB/cough only with exertion or during high output states (fever)
    - late: resting SOB/CP, activity limitation, orthopnea, hemoptysis
    - complications: recurrent PE, pulmonary infections (bronchitis, pneumonia), LA thrombi (systemic emboli: brain, kidney, spleen, arm)
        - dyspnea (exertional, increased HR —> decreased diastolic filling time —> increased LA pressure and pulmonary congestion)
        - orthopnea/PND (increased venous return —> increased LA pressure —> pulmonary congestion)
        - cough, hoarseness, hemoptysis
        - palpitations (A fib secondary to LAE)
- ❑ LV inlet obstruction —> fixed CO
    - symptoms
        - dyspnea
        - fatigue
        - low exercise tolerance
- ❑ atrial kick crucial - CO may decrease with A fib (loss of atrial kick), pregnancy, or tachycardia (shortened diastolic filling period)

Signs of MS
- ❑ general examination
    - mitral facies (mitral flush, pinched and blue facies),
    - hepatic enlargement/pulsation, ascites, peripheral edema (all secondary to TR and RV failure)
- ❑ pulse
    - +/– irregularly irregular (A fib), may be small volume
- ❑ JVP
    - giant "a" waves (Pulmonary HTN, TS), "a" waves lost in A fib, elevated plateau (RV failure), "v" waves (TR)
- ❑ precordial palpation
    - apex - inconspicuous LV (tapping apex)
    - palpable S_1
    - palpable P2 (in severe MS, pulmonary HTN)
    - left parasternal lift (RV tap) palpable diastolic thrill at apex

❑ precordial auscultation
- loud S_1 (when valves are heavily calcified and not pliable —> no closure of MV (no S_1)
- loud P2 (widely split S_2)
- OS (lost if heavily calcified and not pliable), heard best in expiration at apex after P2
- mid-diastolic rumble (low pitch, heard with bell) - at apex, best in LLDB position and post-exercise
- a longer murmur and a shorter A_2-OS interval (both caused by ↑ LAP) correlate with worse MS
- presystolic accentuation of diastolic murmur due to atrial kick (lost with A fib)
- if pulmonary HTN present - loud P2, PR (Graham Steel murmur) associated murmurs: soft systolic apical murmur (MR), Pansystolic murmur at LSB (TR)

❑ chest examination
- crackles (pulmonary congestion)

Investigations

❑ 12 lead ECG
- normal sinus rhythm/A fib, LAE (P mitrale), RVH (RAD)

❑ chest x-ray
- LA enlargement (LA appendage, double contour, splaying of carina), pulmonary congestion (Kerley B lines), pulmonary hemosiderosis (diffuse nodularity) MV calcification, flattened left heart border

❑ echocardiography (TTE)
- gold standard
- thickened calcified valve, fusion of leaflets, LAE, PAP, associated TR
- Doppler can estimate valvular area

❑ cardiac catheterization/ coronary angiography
- concurrent CAD in patients if age > 45 yr (males), > 55 yr (females)

Natural History

❑ symptoms arise > 15-20 years after initial rheumatic involvement of the valve, followed by severe incapacitation (i.e. class IV NYHA symptoms) about 3 years later

❑ complications of A fib: acute respiratory decompensation; systemic and cerebral embolization (often no evidence of residual atrial thrombus)

❑ other complications: IE, pulmonary hemorrhage, cardiac cachexia

Management

❑ avoid factors that increase LA pressure (tachycardia, fever, vigorous exercise, etc.)

❑ medical
- treat A fib (rate control, cardioversion)
- anticoagulation - if A fib or previous embolus
- IE prophylaxis
- diuretics and rate control (beta-blockers)

❑ indications for surgery
- MV area < 1.0 cm^2 with symptoms
- NYHA class III or IV
- worsening pulmonary HTN
- IE
- systemic embolization
- unacceptable lifestyle limitations due to symptoms

❑ surgical options (see Cardiac and Vascular Surgery Chapter)
- closed commisurotomy
  - rarely performed in North America
- balloon valvuloplasty
  - transthoracic echo (TTE) determines suitability for valvuloplasty (based on morphology of leaflets and subchordal apparatus)
- open commisurotomy
  - best procedure if valve amenable to repair
- all the above "turn the clock back" - re-stenosis will develop
- MV replacement
  - if immobile leaflets/heavy calcification, severe subvalvular disease, MR

MITRAL REGURGITATION

Etiology

❑ annulus
- LV dilatation (CHF, DCM, myocarditis); mitral annular calcification; IE (abscess)

❑ leaflets
- congenital (e.g. clefts); myxomatous degeneration (MVP, Marfan's); IE; rheumatic heart disease; collagen vascular disease

❑ chordae
- trauma/tear; myxomatous degeneration; IE; acute MI

❑ papillary muscles and LV wall
- ischemia/infarction; rupture; aneurysm; HCM

Pathophysiology and Symptomatology
- ❑ chronic MR = gradually increase flow across MV (into LA) during systole —> progressive LAE —> decreased fraction of SV flows forward —> LV dilatation (to decrease SV and maintain CO) —> increased LV wall tension —> CHF
- ❑ "MR begets MR" - MR causes LV dilatation which in turn leads to annulus dilatation increased MR
  - symptoms
    - few symptoms initially (LAE generally can prevent an increase in PAP and the subsequent pulmonary edema)
    - later: dyspnea, PND/orthopnea, fatigue and lethargy
    - palpitations
- ❑ acute MR = sudden onset of MV incompetence —> increased LA pressure —> increased PAP —> pulmonary edema —> RV failure (acute onset CHF)

Signs of MR
- ❑ pulse
  - quick and vigorous (unless LV failure)
- ❑ precordial palpation
  - apex - displaced, hyperdynamic, enlarged due to LV dilatation
  - +/– left parasternal lift (LA expands with MR), apical thrill
- ❑ precordial auscultation
  - S_1 normal, soft, or buried in murmur
  - S_3 usually present
  - holosystolic murmur - at apex, usually radiates to axilla, sometimes to base or back (posteriorly directed jet)
    - MR murmur secondary to mitral valve prolapse (MVP) - usually mid-systolic
    - papillary muscle dysfunction - typically a late systolic whoop or honk
  - mid-diastolic rumble - increase flow across valve (often no MS)
  - severity - gauge by LV dilatation, S_3, diastolic flow rumble
- ❑ A fib, CHF, pulmonary HTN develop late
- ❑ acute MR —> CHF, S_3 and S_4 present; usually S_1 and S_2 normal with soft or absent murmur early in systole; often a diastolic flow murmur

Investigations
- ❑ 12 lead ECG
  - LAE, left atrial delay (bifid P waves), possible LVH
- ❑ chest x-ray
  - LVH, LAE, pulmonary venous HTN
- ❑ echocardiography
  - etiology - flail leaflets, vegetations, etc.
  - severity - regurgitant volume/fraction/orifice area
  - LV function - increased LV/LA size; EF
  - color flow mapping shows abnormal jet from LV to LA
- ❑ cardiac catheterization
  - assess coronary arteries
  - ventriculography - contrast fills LA to assess flow and chamber contours
  - prominent left atrial "v" wave on Swan-Ganz

Management
- ❑ medical
  - asymptomatic - serial echocardiograms to monitor progress
  - IE prophylaxis
  - symptomatic - decreased preload (diuresis) and decreased afterload (ACEI) for severe LV dysfunction and MR in poor surgical candidate
- ❑ surgical
  - acute MR - generally best managed surgically
  - chronic MR - indications for surgery
    - persistent symptoms (NYHA class II) despite optimal medical therapy
    - onset of LV dysfunction or increased LV volume or size, even if asymptomatic
- ❑ surgical options (see Cardiac and Vascular Surgery Chapter)
  - valve repair for MR secondary to myxomatous degeneration
    - preferred (low mortality), often technically difficult
- ❑ MV replacement
    - if unable to repair MV
    - attempt to conserve chordal structures/connections, correction of MR achieved

MITRAL VALVE PROLAPSE (MVP)

Etiology
- ❑ myxomatous degeneration of chordae and leaflets which are thickened, voluminous and redundant (too big for the orifice)
- ❑ leaflets displaced into LA during systole
- ❑ 3-5% of population (F > M)
- ❑ alone, or with connective tissue diseases (e.g. Marfan's)
- ❑ may be associated with pectus excavatum, straight back syndrome, and other MSK abnormalities

Symptoms
- ❑ click-murmur syndrome
- ❑ atypical chest pain (prolonged, non-exertional, stabbing)
- ❑ dyspnea, hyperventilation, anxiety, panic, palpitations, presyncope, fatigue - no causal relations or mechanisms found
- ❑ +/– symptoms of MR

Signs of MVP

Clinical diagnosis based on presence of mid-systolic click +/– murmur
- ❑ mid-systolic click (tensing of redundant valve tissue, billowing of posterior leaflet in mid-systole)
- ❑ mid to late systolic murmur(regurgitation after prolapse of MV leaflets)
- ❑ maneuvers to change LV volume (exaggerate the disproportion of the valve with respect to the annulus) - squat to stand, or Valsalva —> decreased venous return, decreased ventricular filling —> earlier click and louder and longer murmur

Investigations
- ❑ 12 lead ECG
  - nonspecific ST-T wave changes, PSVT, ventricular ectopy
- ❑ ECHO
  - posterior systolic prolapse of MV leaflets into LA
  - assess severity of MR

Natural History
- ❑ excellent prognosis (usually benign)
- ❑ risk of complications is most dependent on degree of MR
  - progressive MR; severe MR (beware of ruptured chordae); IE; arrhythmias; thromboembolism; sudden death

Management
- ❑ asymptomatic without MR - excellent prognosis (vast majority)
  - follow-up q 3-5 years
- ❑ β blockers - for palpitations, pain, anxiety
- ❑ anticoagulation - if systemic embolism
- ❑ for MR - IE prophylaxis, standard indications for MV repair/replacement

TRICUSPID VALVE DISEASE

Etiology
- ❑ TS: rheumatic, congenital, carcinoid syndrome, fibroelastosis
- ❑ TR: RV dilatation (commonest cause), IE (iv drug users), rheumatic, Ebstein anomaly, AV cushion defects, carcinoid, tricuspid prolapse, trauma

Symptoms
- ❑ right heart failure
  - fatigue
  - pedal edema, abdominal pain (liver congestion), ascites
  - dyspnea (may reflect right heart forward failure)

Signs
- ❑ carotid pulse: irregular if A fib and low volume
- ❑ JVP
  - increased JVP
  - prominent "a" waves in TS
  - large “v” waves in TR ("cv" waves)
  - positive HJR and Kussmaul's sign (rise in JVP with inspiration)
- ❑ precordial palpation for left parasternal lift (RV) in TR
- ❑ precordial auscultation
  - note: all right sided sounds are louder with inspiration, except a pulmonary ejection click
  - TS: diastolic rumble in 4th left intercostal space (LICS)
  - TR: holosystolic murmur along LLSB (Carvallo's murmur); may behave like an ejection murmur
  - RV S3 along LLSB (with inspiration)
- ❑ abdominal examination
  - hepatomegaly (congestion) with systolic pulsations from TR
  - edema, ascites: 2º to fluid retention

Investigations
- ❑ 12 lead ECG
  - TS: RAE
  - TR: RAE, RVH, A fib
- ❑ chest x-ray
  - TS: dilatation of RA without pulmonary artery enlargement
  - TR: RA + RV enlargement
- ❑ ECHO
  - diagnostic

Management
- ❑ supportive
  - diuretics, preload reduction
  - TV surgery usually determined by need for other interventions (e.g. MVR of r associated MS)

PULMONARY VALVE DISEASE
- ❑ much less commonly involved

Etiology
- ❑ pulmonary stenosis (PS): usually congenital; rheumatic uncommon; carcinoid
- ❑ pumonary regurgitation (PR): secondary to dilatation of valve ring
  - pulmonary HTN (MS - most common, COPD, recurrent PE)
  - rheumatic, IE

Symptoms
- ❑ chest pain, syncope, dyspnea, leg edema (RV failure and CHF)

Signs
- ❑ PS
  - systolic murmur - maximum at 2nd LICS
  - pulmonary ejection click; normal/loud/soft P2; right sided S_4
- ❑ PR
  - early diastolic murmur at base
  - Graham Steel (diastolic) murmur at 2nd and 3rd LICS increasing with inspiration; no peripheral stigmata of AR

Investigations
- ❑ 12 lead ECG
  - RVH
- ❑ chest x-ray
  - prominent pulmonary arteries if pulmonary HTN
  - enlarged RV
- ❑ ECHO
  - diagnostic - RVH, RV dilatation; PS or PR by Doppler

Management
- ❑ IE prophylaxis
- ❑ PR
  - rarely requires treatment (well tolerated if systemic vascular resistance is normal)
  - valve replacement may be required
- ❑ PS
  - balloon valvuloplasty, depending on severity

PROSTHETIC VALVES
- ❑ bioprosthetic valves
  - porcine heterograft, bovine pericardial, human homograft
  - low incidence of thromboembolism, anticoagulation often not required (use ASA only), ideal for those with contraindications to anticoagulation (pregnancy)
  - degeneration of valve after 10 years on average
  - higher failure rate in the mitral position
  - contraindicated in children due to rapid calcification
- ❑ mechanical valves
  - better predictability of performance and durability
  - used preferentially if risk of reoperation is high
  - always requires anticoagulation to prevent thromboembolism
    - contraindications: bleeding tendency (e.g. peptic ulcer disease (PUD)), pregnancy (Coumadin is teratogenic)
    - target INR = 2.5-3.5
- ❑ post-op complications
  - valve failure
  - valve thrombosis (< 1%/year)
  - valve degeneration
  - IE (often < 1 year after surgery, Staph. epidermidis)
  - bleeding problems due to anticoagulation (major: 1%/year)
  - thromboembolism (2-5% per patient-year despite adequate anticoagulation)
  - conduction abnormalities

PERICARDIAL DISEASE

ACUTE PERICARDITIS

Etiology
- ❑ idiopathic is most common: usually presumed to be viral
- ❑ infectious
  - viral: Coxsackie virus A, B (most common)
  - bacterial: Staph, Strep, septicemia
  - TB
  - fungal: histoplasmosis, blastomycosis
  - protozoal
- ❑ post-MI: acute (direct extension of myocardial inflammation, 1-7 days), Dressler's syndrome (autoimmune, 2-8 weeks)
- ❑ post-pericardiotomy (e.g. CABG), other trauma
- ❑ metabolic: uremia (common), hypothyroidism
- ❑ neoplasm: Hodgkin's, breast, lung, renal cell carcinoma, melanoma
- ❑ collagen vascular disease: SLE, periarteritis, RA, scleroderma
- ❑ vascular: dissecting aneurysm
- ❑ infiltrative disease (sarcoid), drugs (e.g. hydralazine), radiation

Presentation
- ❑ diagnostic triad: chest pain, friction rub, and ECG changes
- ❑ chest pain - alleviated by sitting up and leaning forward, pleuritic, worse with deep breathing and supine position
- ❑ pericardial friction rub - may be uni-, bi- or triphasic
- ❑ +/– fever, malaise

Investigations
- ❑ 12 lead ECG
  - initially elevated ST in anterior, lateral and inferior leads +/– depressed PR segment, the elevation in the the ST segment is concave upwards —> 2-5 days later ST isoelectric with T wave flattening and inversion
- ❑ chest x-ray
  - normal heart size, pulmonary infiltrates
- ❑ echocardiography
  - assess pericardial effusion

Management
- ❑ treat the underlying disease
- ❑ anti-inflammatory agents (NSAIDs, steroids if severe or recurrent); analgesics

Complications
- ❑ recurrences, atrial arrhythmias, pericardial effusions, tamponade, residual constrictive pericarditis

PERICARDIAL EFFUSION

Etiology
- ❑ two types of effusions:
  - transudative (serous)
    - CHF, hypoalbuminemia/hypoproteinemia, hypothyroidism
  - exudative (serosanguinous or bloody)
    - causes similar to the causes of acute pericarditis
    - may develop acute effusion secondary to hemopericardium (trauma, post MI myocardial rupture, aortic dessection)
- ❑ physiological consequences depend on type and volume of effusion, rate of effusion development, and underlying cardiac disease

Symptoms
- ❑ none or similar to acute pericarditis
- ❑ dyspnea, cough
- ❑ extra-cardiac (esophageal/recurrent laryngeal nerve/tracheo-bronchial/phrenic nerve irritation)

Signs
- ❑ JVP: increased with dominant "x" descent
- ❑ arterial pulse: normal to decreased volume, decreased PP
- ❑ auscultation: distant heart sounds +/– rub

Investigations
- ❑ 12 lead ECG
  - low voltage, flat T waves
- ❑ chest x-ray
  - cardiomegaly, rounded cardiac contour (water bottle)
- ❑ ECHO (procedure of choice)
  - fluid in pericardial sac
- ❑ pericardiocentesis
  - establishes diagnosis

Management
- ❑ mild: frequent observation with serial ECHO, treat the cause, anti-inflammatory agents for inflammation
- ❑ severe: may develop cardiac tamponade

CARDIAC TAMPONADE

❑ major complication of pericardial effusion
❑ cardiac tamponade is a clinical diagnosis

Pathophysiology and Symptomatology

❑ high intra-pericardial pressure —> decreased venous return —> decreased diastolic ventricular filling —> decreased CO —> hypotension + venous congestion
- symptoms
  - tachypnea, dyspnea, shock

Signs

❑ "x" descent only, absent "y" descent
❑ hepatic congestion

Clinical Pearl
❑ **Classic quartet: hypotension, increased JVP, tachycardia, pulsus paradoxus.**
❑ **Beck's triad: hypotension, increased JVP, muffled heart sounds.**

Investigations

❑ 12 lead ECG
- electrical alternans (pathognomonic variation in R wave amplitude), low voltage

❑ ECHO
- pericardial effusion, compression of cardiac chambers (RA and RV) in diastolic

❑ cardiac catheterization
- mean RA, LA, LV and RV diastolic pressures all high and equal

Management

❑ pericardiocentesis – ECHO-, ECG-guided
❑ pericardiotomy
❑ avoid diuretics and vasodilators (these decrease venous return to already under-filled RV —> decrease LV preload —> decrease CO)
❑ fluid administration may temporarily increase CO
❑ treat underlying cause

CONSTRICTIVE PERICARDITIS

Definition

❑ chronic pericarditis resulting in fibrosed, thickened, adherent, and/or calcified pericardium

Etiology

❑ any cause of acute pericarditis may result in chronic pericarditis
❑ major causes are tuberculous, radiation-induced, post-cardiotomy, idiopathic

Symptoms

❑ dyspnea, fatigue, palpitations
❑ abdominal pain

Signs

❑ general examination - mimics CHF (especially right-sided HF)
- ascites, hepatosplenomegaly, edema

❑ increased JVP, Kussmaul's sign (paradoxical increase in JVP with inspiration), Friedrich's sign (prominent "y" descent > "x" descent)
❑ pressures: BP normal to decreased, +/– pulsus paradoxus
❑ precordial examination: +/– pericardial knock (early diastolic sound)

Investigations

❑ 12 lead ECG
- low voltage, flat T wave, +/– A fib

❑ chest x-ray
- pericardial calcification, effusions

❑ CT/MRI/TEE
- pericardial thickening

❑ cardiac catheterization
- equalization of RV and LV diastolic pressures, RVEDP > 1/3 of RV systolic pressure

Management

❑ medical: diuretics, salt restriction
❑ surgical: pericardiectomy

Table 11. Differentiation of Constrictive Pericarditis vs. Cardiac Tamponade

Characteristic	Constrictive Pericarditis	Tamponade
JVP	"y" > "x"	"x" > "y"
Kussmaul's sign	Present	Absent (JVP too high to see change)
Pulsus paradoxus	1/3 of cases	Always
Pericardial knock	Present	Absent
Hypotension	Mild-moderate	Severe

SYNCOPE

Definition
❑ sudden, transient disruption of consciousness and loss of postural tone with spontaneous recovery
❑ usually caused by generalized cerebral hypoperfusion

Etiology
❑ cause of 50% of cases of syncope is unknown
❑ cardiac
- electrical
  - tachycardia: VT, Torsades de pointes, SVT, rapid A fib
  - bradycardia: sick sinus syndrome, 2° or 3° (Stokes-Adams attack) AV block
  - pacemaker failure
- mechanical
  - outflow obstruction: left-sided (AS, HOCM, MS, LA myxoma), right-sided (PS, PE, pulmonary HTN)
  - myocardial: CAD/MI, LV dysfunction
  - other: tamponade

❑ extra-cardiac
- neurally mediated vasomotor
  - vasovagal - the "common faint " (50%)
  - situational/visceral: micturition/defecation syncope, cough syncope, Valsalva, ocular pressure, etc.
  - carotid sinus syncope
  - psychiatric: somatization, panic, anxiety
  - other: exercise, high altitude, drug-induced
- orthostatic hypotension: drug-induced (e.g. antihypertensives), venous pooling (postural, pregnancy), autonomic neuropathy (primary: Shy-Drager, secondary: DM), hypovolemia (blood loss, diuresis), pheochromocytoma
- neurological: vertebrobasilar TIA/stroke, subarachnoid hemorrhage, cervical spondylosis, seizure, subclavian steal
- metabolic: hypoxia, hypoglycemia, hypocapnia

Clinical Manifestations
❑ history and physical examination are critical - reflect underlying pathology in 40-50% (attention to cardiac and neurological exams) (see Neurology Chapter)

Table 12. Differentiation of Seizure vs. Syncope

Characteristic	Syncope	Seizure
Facial colour	Pale	Cyanotic
(lateral) tongue biting	Rare	Common
Aura	No	Sometimes
Nausea, diaphoresis	Common before	Uncommon
Level of concsciousness (LOC)	Brief	May be longer
Reoriention	Within seconds	Within minutes
Todd's paralysis	No	Sometimes
Setting	Rare when recumbent	Anytime
Attacks	Infrequent	Repeated
Age	Variable	Younger (< 45)
CK	Normal	Increased
Positive EEG	No	Sometimes

Investigations
❑ directed by results of history and physical examination
❑ blood work: CBC, electrolytes, MgV, Ca^{+2}, BUN, creatinine, glucose, ABG, CK-MB
❑ ECG
❑ ECHO
❑ carotid Doppler
❑ Holter monitor, loop Holter
❑ tilt-table testing
❑ electrophysiological study (EPS)

Management
- ❑ treatment of underlying cause

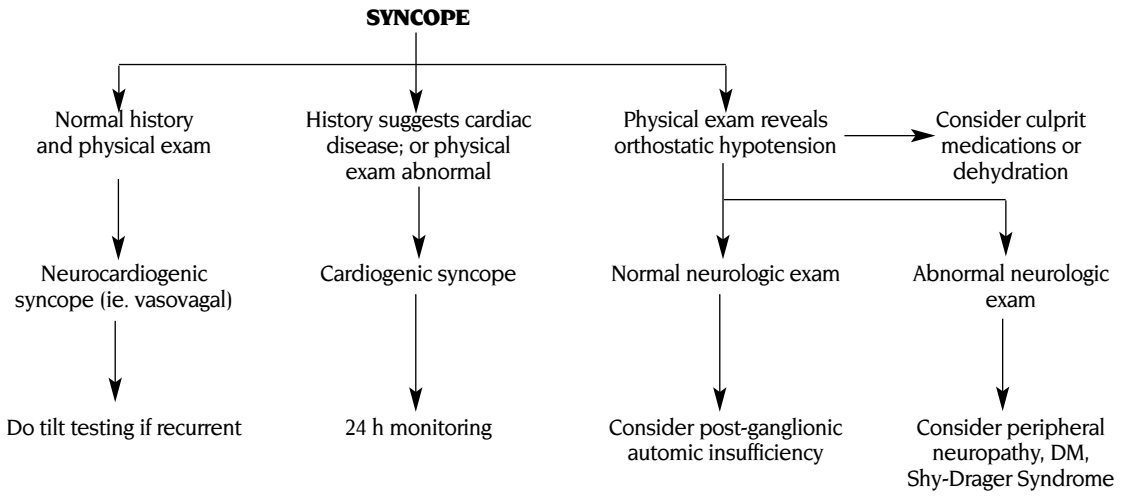


Figure 15. Approach to the Patient with Syncope

EVIDENCE-BASED CARDIOLOGY

CONGESTIVE HEART FAILURE
- ❑ VeHEFT-I: Hydralazine/Isorbide Dinitrate decreases mortality in patients with CHF. (NEJM 1986; 314:1547)
- ❑ VeHEFT-II: Enalapril decreases mortality compared to Hydralazine/Isorbide Dintrate in patients with CHF. (NEJM 1991; 325:303)
- ❑ CONSENSUS: Enalapril decreases mortality compared to placebo in severe CHF. (NEJM 1987; 316:1429)
- ❑ DIG TRIAL: Digoxin decreased rate of hospitalization, improves symptoms and exercise capacity, but has no mortality benefit compared to placebo. (NEJM 1997; 336:525)
- ❑ PRAISE: Amlodipine has no mortality benefit over placebo in CHF, except decreases mortality in patients with non-ischemic dilated CM (NEJM 1996; 335:1107)
- ❑ US-CARVEDILOL STUDY: Carvedilol is superior to placebo for morbidity and mortality in class II and III heart failure (NEJM 1996;334:1349)
- ❑ MERIT: Metoprolol is superior to placebo for morbidity and mortality in class II and III heart failure (Lancet 1999; 353:2001)
- ❑ RALES: Aldosterone antagonism with Spironolactone in addition to standard treatment decreases mortality in patients with FC III-IV heart failure (NEJM 1999;341: 709)

ISCHEMIC HEART DISEASE
- ❑ GUSTO I: There is increased survival after acute MI in patients treated with rt-Pa and IV Heparin compared to Streptokinase (NEJM 1993; 329:673)
- ❑ ESSENCE: Enoxaparin decreases mortality vs. unfractionated heparin in patients with unstable angina or non-Q wave MI. (NEJM 1997; 337:447)
- ❑ PURSUIT: Integrelin (IIb/IIIa inhibitor) decreased mortality when given to patients with high risk unstable angina (e.g. resting chest pain for >15 mins within last 24hrs + increases TnI/ECG changes) or non-Q wave MI, and benefit increases if patients go for PTCA or CABG (Circulation 1996; 94:2083)
- ❑ BARI: subset analysis - CABG as an initial strategy has survival benefit over PTCA in diabetic patients with multivessel disease (NEJM 1996;335:217)
- ❑ HOPE: Ramipril decreases rate of death, MI, and CVA in patients with CAD, Hx of CVD, PVD, or DM +1 other cardiac risk factor, all who are not known to have any LV dysfunction. (NEJM 2000; 342:145)

ATRIAL FIBRILLAITON
- ❑ 5 RCT's (SPAF-I, AFASAK, SPINAF, CAFA, BAATAF) level demonstrated 67% decrease in thromboembolic rate in patients treated with coudamin in setting of nonrheumatic AF)

Table 13. Commonly Used Cardiac Therapeutics

DRUG CLASS	EXAMPLES	MECHANSIM OF ACTION	INDICATIONS	SIDE EFFECTS	CONTRA-INDICATIONS
β-BLOCKERS	• metoprolol, atenolol (β_1) • acebutolol (β_1, ISA) • labetalol (α_1, β_1, β_2) • carvedilol (α_1, β_1, β_2) and anti-oxidant) • sotalol (β1, β2, class III anti- arrhythmic)	• Lowers myocardial O_2 demand by ↓ HR, BP and contractility	• IHD • HTN • A Fib • stable class II to III CHF • SVT	• bradycardia • fatigue • dizziness • nightmares, memory loss, depression, hallucinations • depression of counterregulatory responses to hypoglycemia in diabetes • +/– adverse effects on lipid profile • bronchospasm • exacerbation of Raynaud's phenomenon and claudication • impotence	• severe bradycardia, high-degree heart block • caution in asthmatics (contraindicated if severe asthma/bronchospasm) • caution in patients with peripheral claudication phenomenon and Raynaud's • caution in CHF
CALCIUM CHANNEL BLOCKERS (CCB)	diltiazem	see Table 15	• HTN • 2nd line agent for IHD (1st line β-blockers) • SVT	• anorexia, nausea • edema • bradycardia • CHF	• sick sinus syndrome • second or third degree AV block • severe CHF • AMI with CHF • pregnancy
	verapamil	see Table 15	• HTN • 2nd line agent for IHD (1st line β-blockers) • SVT • diastolic dysfunction	• bradycardia • CHF • constipation	• sick sinus syndrome • second or third degree AV block • severe CHF • AMI (relative) • pregnancy (relative) • A Fib with bypass tract with anterograde conduction
	nifedipine	see Table 15	• HTN	• hypotension • edema • flushing • dizziness • headache	• NOTE evidence that short acting nifedipine is associated with increased mortality (AMI) • severe AS • HCM • poor LV function • pregnancy • unstable angina or threatened MI in absence of β-blocker
ACE INHIBITORS	captopril enalapril ramipril	• peripheral vasodilator —> afterload reduction with little change in CO, HR or GFR • also cause ↓ in fluid volume due to inhibition of aldosterone production	• CHF (including post-MI) • HTN • post-MI EF<40%) • anterior MI	• dry cough (5-15% of patients) • hypotension • hyperkalemia • renal insufficiency • angioedema (rare) • reversible neutropenia • proteinuria • membranous GN • fatigue	• bilateral renal artery stenosis • pregnancy (absolute) • documented angioedema 2° to ACEI
ANGIOTENSIN II BLOCKER	losartan (cozaar)	• blocks angiotensin II receptor so peripherally vasodilates and blocks aldosterone effects	• CHF • HTN	• dizziness (< 2%) • hypotension/syncope • renal dysfunction	• bilateral renal artery stenosis • pregnancy

COMMONLY USED CARDIAC MEDICATIONS . . . CONT.

DRUG CLASS	EXAMPLES	MECHANSIM OF ACTION	INDICATIONS	SIDE EFFECTS	CONTRA-INDICATIONS
DIURETIC	Furosemide	• loop diuretic • interferes with creation of hypertonic medullary interstitium • diuretic effect within 1 hour after oral administration, within 30 minutes after IV administration	• acute pulmonary edema • severe CHF • refractory edema • hypercalcemia (use furosemide with saline infusions)	• hypokalemia • hypovolemia • azotemia • hyperuricemia • hypochloremic metabolic alkalosis	• severe hypovolemia • severe hypotension • hypersensitivity to furosemide or sulfonamide
NITRATES	• sublingual/ patch/ IV nitroglycerin • isosorbide dinitrate	• produce venous, arteriolar and coronary vasodilation	• symptomaltic relief of angina • CHF in isosorbide dinitrate form (always combine with hydralazine in CHF)	• headaches • dizziness • weakness • postural hypotension • tolerance develops rapidly with continuous use; maintain at least 8 nitrate-free hours per day	• hypersensitivity • active peptic ulcer

ANIT-ARRHYTHMIC	Digoxin	• $Na^+ – K^+$ – ATPase inhibitor causes ↑ intracellular Na resulting in exchange of Na+ for Ca^{2+}
		• ↑ Ca^{2+} results in inotropic action, and cell stabilization
		• positive inotrope-increases force contraction
		• blocks AV node (decreased refractory period and conduction time) and depresses SA node
			• A Fib
			• CHF
				• cardiac toxicity
				  • AV blocks (e.g. Wenkebach, atrial tachycardia with block)
				  • tachycardias (eg VT atrioventricular dissociation, accelerated junctional rhythm)
				• bradyarrhythmias (e.g. sinus bradycardia, sinus arrest, sinoatrial block)
				• regularization of R-R interval in A Fib
				• GI
				  • anorexia, nausea/vomiting
				• CNS
				  • blurred or yellow vision
				  • headache
				  • weakness/apathy
				  • psychosis
					Absolute
					• high degree AV block
					• hypersensitivity
					
					Relative
					• arrhythmogenic states (e.g. hypokalemia, acute MI, acute/chronic myocarditis, frequent PVCs, WPW with anterograde conduction down bypass tract, acute hypoxemia, chronic cor pulmonale , diastolic dysfunction in the absense of systolic dysfunction)
					• risk of complete AV block/ bradycardia
					  • sick sinus syndrome
					  • incomplete AV block
					• HCM

ANTI-PLATELET	ASA	• cyclooxygenase inhibitor
		• interferes with platelet aggregation by impairing production of thromboxane A2
			• acute MI
			• Post-MI
			• Post CABG
			• Post PTCA
			• TIA/ CVA
				• GI
				  • nausea,vomiting, diarrhea
				  • dyspepsia, peptic ulcers
				• ototoxicity
				  • tinnitus, vertigo, hearing loss
				• hematological
				  • leukopenia, anemia
				  • purpura, thrombocytopenia
				• bronchoconstriction
				• impaired renal perfusion leading to fluid retention
				• dermatological or anaphylactic hypersensitivity reactions
					• hypersensitivity
					• active peptic ulcer

Table 14. Beta-Blocker Actions

Clinical Effects	Propranolol	Atenolol	Acebutolol	Labetalol
β-Activity	non-selective	β1	β1	non-selective
α-Activity	N	N	N	α1
ISA	N	N	+++	+
Bronchoconstriction	+++	+	+	++
Orthostatic Hypotension	–	–	–	+++
Lipid Adverse Effects	++	++	–	+
CNS Adverse Effects	+++	+	++	++

CALCIUM CHANNEL BLOCKERS (CCB)

- major subtypes are represented by diltiazem (benzothaizepine), verapamil (phenylalkylamine) and nifedipine (dihydropyridine)
- diltiazem and verapamil are strong cardiodepressants, whereas the dihydropyridines are strong vasodilators

Table 15. Calcium Channel Blocker Actions

Clinical Effects	Diltiazem	Verapamil	Nifedipine
Coronary Vasodilator	++	++	+++
Peripheral Vasodilator	+	++	+++
Contractility	<—>	↓	<—>
Sinus Rate	↓	↓	↑
AV Conduction	↓	↓	<—>

ANTI-ARRHYTHMIC DRUGS

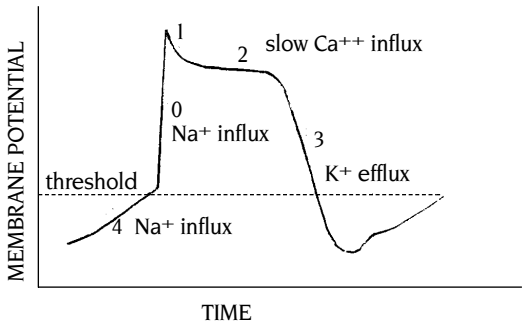


Figure 16. Representative Action Potential

Table 16. Antiarrhythmic drugs (Vaughn-Williams Classification)

Class	Agent	Indications	Side Effects	Mechanism of Action
Ia	Quinidine Procainamide Disopyramide	SVT, VT	Torsades de Pointes (all Ia) diarrhea lupus-like syndrome anti-cholinergic effects	• moderate Na^+ channel blockade • slows phase O upstroke • prolongs repolarization and thus slows conduction
Ib	Lidocaine Mexiletine	VT	confusion, stupor, seizures GI upset, tremor	• mild Na^+ channel blockade • shortens phase 3 repolarization
Ic	Propafenone Flecainide Encainide	SVT, VT[1] A Fib[2]	exacerbation of VT (all Ic) negative inotropy (all Ic) bradycardia and heart block (all Ic)	• marked Na^+ channel blockade • markedly slows phase 0 upstroke
II	Propranolol Metoprolol etc.	SVT, A Fib[1]	bronchospasm, negative inotrophy, bradycardia, AV block, impotence, fatigue	• β blockers • decreases phase 4 depolarization
III	Amiodarone*	SVT, VT A Fib	photosensitivity, pulmonary toxicity, hepatotoxicity, hyper/hypothyroidism	• blocks K channel • prolongs phase 3 repolarization and so prolongs the effective refractory period
	Sotalol Bretylium (IV)	SVT, VT , A Fib VT	beta-blocker effects, Torsades de Pointes, hypotension	
IV	Verapamil Diltiazem	SVT A Fib	bradycardia, AV block hypotension	• CCB • slow phase 4 spontaneous depolarization and so slows conduction in areas such as AV node
Amiodcrone hcs class I, II, III, IV, properties				

- ❑ All anti-arrhythmics have potential to be pro-arrhythmic
- ❑ In the landmark CAST trial, two class Ic agents (encainide, flecainide) prevented VPB's post MI but significantly increased mortality

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