CARDIOLOGY

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FUNCTIONAL CLASSIFICATION OF CARDIOVASCULAR DISABILITY

Table 1. 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

Table 2. 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 3. Clinical Applicability of Classification Schemes

Scale	Validity (%)	Reproducibility (%)
NYHA	51	56
CCS	59	73

Questions to ask on History to Clarify Disability

- ❑ What kind of activities bring on symptoms (fatigue, palpitations, dyspnea, or angina)?
- ❑ How far can you walk before becoming symptomatic?
- ❑ Do low impact activities, such as combing your hair or getting into the shower ever bring on symptoms?
- ❑ Have you ever experienced symptoms at rest?

CARDIAC EXAMINATION

Blood Pressure

- ❑ should be taken in both arms, and with the patient supine and upright
- ❑ orthostatic hypotension – postural drop >20 mmHg systolic or >10 mmHg diastolic, usually accompanied by tachycardia; implies inadequate circulating blood volume
- ❑ pulse pressure – pressure differential between systolic and diastolic BP
  - wide pulse pressure: stiffening of arterial system (e.g. atherosclerosis, hypertension), increased stroke volume (anxiety, exercise, AR), increased CO or decreased peripheral resistence (fever, anemia, thyrotoxicosis, cirrhosis of the liver)
  - narrow pulse pressure: decreased CO (ie. CHF, shock, hypovolemia, acute MI, cardiomyopathy), peripheral vasoconstriction (shock, hypovolemia), valvular disease (AS, MS, MR), aortic disease (e.g. coarctation of aorta)
- ❑ pulsus alterans – beat-to-beat alteration in pulse pressure amplitude (i.e. cyclic dip in systolic BP); due to alternating LV contractile force
  - differential diagnosis: severe LV functional impairment, PSVT
- ❑ pulsus paradoxus – decrease in systolic arterial blood pressure > 10 mmHg during inspiration
  - differential diagnosis: pericardial tamponade, constrictive pericarditis, airway obstruction, superior vena cava obstruction

The Arterial Pulse

- ❑ remark on
  - rate, rhythm, volume/amplitude, contour
  - amplitude and contour best appreciated in carotid arteries

Precordial Inspection

- ❑ observe for apex beat, heaves, lifts

Precordial Palpation

- ❑ apex - definition - most lateral impulse
- ❑ PMI - definition - point of maximal intensity, usually the apex
- ❑ comment on location, size and quality of apex (if difficult to palpate, try LLDB)
- ❑ normal apex is 2-3 cm in diameter in 5th intercostal space at midclavicular line, not > than 10 cm from midline, and a duration < 2/3 systole
- ❑ abnormal impulses
  - LV hypertrophy - sustained (> 2/3 systole), heaving apex
  - LV dilatation - apex displaced "down and out", enlarged > 3 cm
  - RV hypertrophy - sustained, heaving pulsation at LLSB
  - RV dilatation - less-sustained pulsation at LLSB
  - anterior MI - impulse between apex and LLSB
  - pulmonary artery pulsation - second left interspace (pulmonary hypertension)
  - double or triple impulse - HCM
  - exaggerated, brief - AR, MR, L to R shunt
- ❑ palpate over each valvular area for palpable murmurs (thrills)
  - tactile equivalents of murmurs

Clinical Pearl

- ❑ Left parasternal lift - DDX - RVH, LAE (secondary to MR), LV aneurysm, rarely thoracic aortic aneurysm

Auscultation - Heart Sounds

- ❑ S_1
  - composed of audible mitral (M1) and tricuspid (T1) components
  - may be split in the normal patient
- ❑ if S_1 is loud
  - short PR interval
  - high left atrial pressure (e.g. early mitral stenosis)
  - high output states or tachycardia (diastole shortened)
- ❑ if S_1 is soft
  - first degree AV block
  - calcific mitral valve (e.g. late mitral stenosis)
  - high LV diastolic pressures (e.g. CHF, severe AR)
  - occasionally in mitral regurgitation
- ❑ if S_1 varies in volume
  - AV dissociation (complete AV block, VT)
  - atrial fibrillation
- ❑ S_2
  - normally has 2 components: A_2 and P_2
  - normal splitting of S_2 ($A_2 < P_2$) 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 (widens split)
A_2 P_2	A_2 P_2	wide fixed splitting • ASD
S_2	A_2 P_2	widened splitting (delayed RV or early LV emptying) • right bundle branch block • pulmonary hypertension • mitral regurgitation
P_2 A_2	S_2	paradoxical splitting (delayed LV or early RV emptying) • left bundle branch block • aortic stenosis (tight) • systemic hypertension • LV failure • paced rhythm • tricuspid regurgitation

- ❑ soft S_2
  - aortic (A_2) or pulmonary stenosis (P_2)
- ❑ loud S_2
  - systemic (A_2) or pulmonary hypertension (P_2)
- ❑ soft heart sounds
  - low cardiac output
  - obesity
  - emphysema
  - pericardial effusion ("muffled" = tamponade)
- ❑ S_3 (Figure 1)
  - 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)
    - left ventricular failure (systolic dysfunction)
    - rapid ventricular filling, as in mitral regurgitation or high output states
  - differential diagnosis - split S_2, opening snap, pericardial knock, tumour plop
- ❑ S_4 (Figure 1)
  - occurs during atrial contraction
  - best heard with bell at apex
  - almost always pathological
  - heard with conditions that result in a rigid, non-compliant ventricle (i.e. diastolic dysfunction)
    - ischemia (ventricular relaxation needs ATP)
    - hypertrophy (HTN, AS, HCM)
    - restrictive cardiomyopathy
  - differential diagnosis - split S_1, ejection clicks, prolapse clicks
- ❑ extra sounds
- ❑ opening snap - early-diastolic (see Figure 1)
  - mitral stenosis (A_2-OS time shortens as MS worsens)
- ❑ ejection clicks
  - aortic stenosis
  - pulmonary stenosis
- ❑ non-ejection clicks
  - early, mid or late systolic
  - associated with mitral valve prolapse, tricuspid valve prolapse
- ❑ pericardial rubs
  - pericarditis
  - "scratchy" sound
  - up to three components - ventricular systole, ventricular diastole and atrial systole

Auscultation - Murmurs

- ❑ assess location, radiation, timing (relation to systole/diastole), shape, pitch, intensity (grade 1-6), maneuvers
- ❑ presence or absence of accompanying thrills, association with extra heart sounds
- ❑ consider use of maneuvers to accentuate murmurs
- ❑ respiratory maneuvers

Clinical Pearl

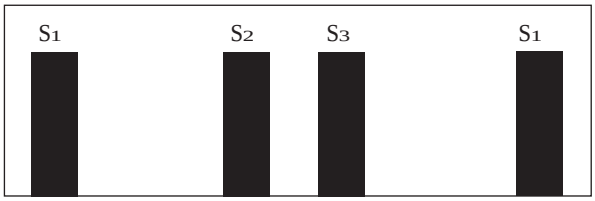
- ❑ Inspiration augments all right-sided murmurs and sounds (Carvallo's sign), except pulmonary ejection click and right sided HCM
- ❑ Expiration augments AR

- ❑ postural maneuvers
  - LLDB for MS
  - upright, leaning forward for AR
- ❑ special maneuvers

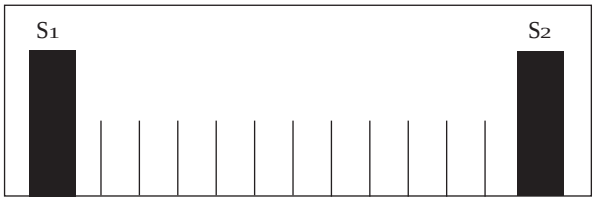
Table 4. Maneuvers for Auscultation of Heart Murmurs

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 • ↑ pulmonic stenosis	• ↑left-sided murmurs • ↑MR • ↑VSD	• ↓HCM • ↓MVP	• ↓ AS

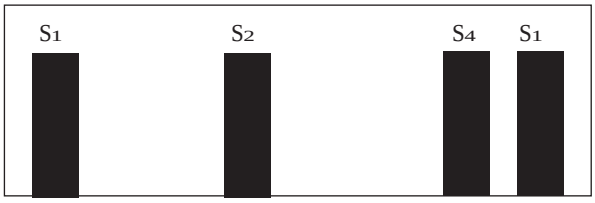
- ❑ systolic “ejection” murmurs (see Figure 1)
  - diamond-shaped, crescendo-decrescendo
  - aortic or pulmonary stenosis
  - high output or "flow" murmurs
    - anemia
    - hyperthyroidism
    - pregnancy
    - arteriovenous fistula
    - children
- ❑ pansystolic murmurs (see Figure 1)
  - require a sustained pressure difference throughout systole
    - mitral regurgitation
    - tricuspid regurgitation
    - VSD
- ❑ high-pitched diastolic decrescendo murmurs (see Figure 1)
  - aortic regurgitation
  - pulmonary regurgitation
- ❑ low-pitched diastolic murmurs (see Figure 1)
  - mitral stenosis
  - tricuspid stenosis
  - severe AR may produce Austin Flint murmur
- ❑ high flow murmurs (result from 'relative' stenosis)
  - MR, PDA, VSD (increased LA filling)
  - ASD (increased 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



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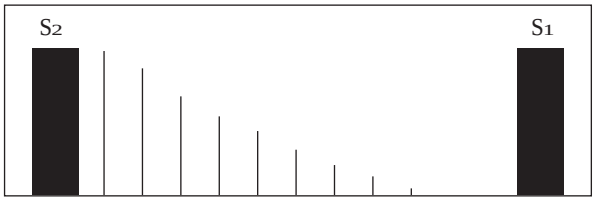
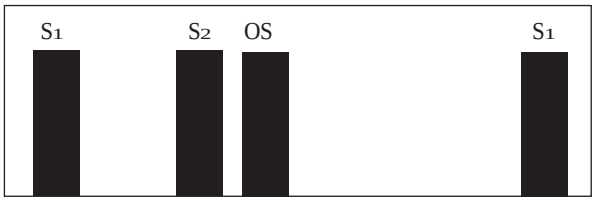
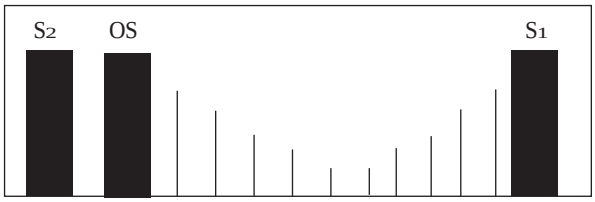


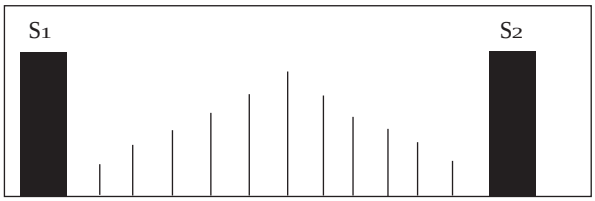
High Pitched Diastolic Murmur



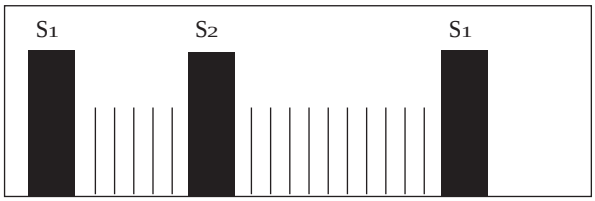
Opening Snap



Low Pitched Diastolic Murmur



Systolic Ejection Murmur



Continuous Murmur

Figure 1. Heart Sounds and Murmurs

Jugular Venous Pulsations (Figure 2)

- ❑ attempt visualization with patient at 30-45 degrees inclination and adjust as necessary to see JVP at mid-neck level
- ❑ identifying features of the JVP
  - location - between heads of the sternocleidomastoid coursing towards angle of jaw
  - multiple waveform in normal patient
  - non-palpable
  - obliterated with pressure at base of neck
  - changes in location with degree of incline and inspiration
  - increases with abdominal pressure: 20-35 mmHg (HJR)
  - normal response is a transient rise [(+) HJR; see below]
  - descents are clinically more prominent than ascents at the bedside
- ❑ normal waveforms
  - "a" wave = atrial contraction – precedes carotid pulse
  - "x" descent = atrial relaxation – occurs during peak of carotid pulse
  - "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
    - atrial fibrillation, atrial standstill

- giant "a" waves
  - contraction of atrium against increased resistance (e.g. TS or RVH [every beat])
- cannon waves
  - contraction of atrium against closed TV as in AV dissociation (not every beat)
- systolic venous pulsation (c-v waves)
  - regurgitation of blood into venous system with ventricular contraction as in TR
- sharp "y" descent
  - raised venous pressure as in constrictive pericarditis

❑ HJR
- positive response (controversial - > 1 definition)
- Sapira says sustained elevation > 4 cm for one minute
- Other - JAMA 1996 = >10s elevation of > 4 cm with abdominal compression
- correlates better with increased PCWP (L-sided failure) than R-sided failure

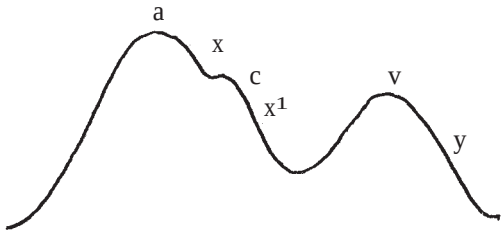


Figure 2. Jugular Venous Pulsations

CARDIAC DIAGNOSTIC TESTS

ECG INTERPRETATION-THE BASICS

Key Features (see ECG appendix)
❑ 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
❑ sinus rhythm = 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?
❑ normal sinus rhythm, has a P wave preceding each QRS complex
❑ P is negative in aVR and positive in II in normal sinus rhythm

Axis
- ❑ deviation - limb leads: normal = positive QRS in I and II
  - axis is perpendicular to lead in which QRS is isoelectric
  - see sections on ventricular hypertrophy and hemiblocks, below
- ❑ rotation - precordial leads: isoelectric QRS in V3, V4
  - heart rotates toward hypertrophy and away from infarction
  - clockwise = isoelectric QRS in V5, V6
  - counterclockwise = isoelectric QRS in V1, V2 (i.e. tall R wave in V1, see below)

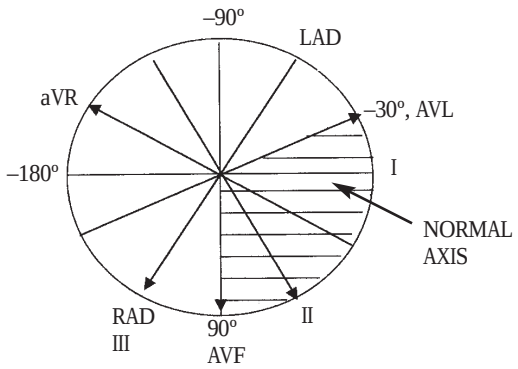


Figure 3. Diagram of Electrocardiogram Limb Leads

Waves and Segments
- ❑ P wave - atrial depolarization
- ❑ PR interval - normal is 0.12 - 0.20 seconds (3-5 small squares)
  - rate dependent
- ❑ QRS complex - ventricular depolarization
  - normal duration < 0.12 seconds (3 small squares)
- ❑ ST segment
  - is it above or below the baseline?
- ❑ QT interval - should be < 1/2 of the RR interval
  - appropriate QT interval is rate related
- ❑ T wave - ventricular repolarization
  - normal = negative in aVR, flat or minimally negative in limb leads; otherwise positive

HYPERTROPHY AND CHAMBER ENLARGEMENT

Right Ventricular Hypertrophy
- ❑ QRS < 0.12 seconds, R/S ratio > 1 in V1, R/S ratio < 1 in V5 and V6, R > 7 mm in V1
- ❑ RAD (> 90º)
- ❑ ST segment depression in V1 and V2 (strain if asymmetrically inverted)

Left Ventricular Hypertrophy
- ❑ S in V1 or V2 (in mm) + R in V5 or V6 > 35 mm
- ❑ S in V1 or V2 or R in V5 or V6 > 25 mm
- ❑ R in aVL > 11 mm
- ❑ R in I + S in III > 25 mm
- ❑ LAD (> –30) with slightly widened QRS
- ❑ asymmetric ST segment depression and T wave inversion (strain) leads I, aVL, V4-V6
- ❑ LAE

Right Atrial Enlargement (P Pulmonale)
- ❑ P wave > 2.5 mm (in height) in leads II, III or aVF
- ❑ P wave duration < 0.12 seconds

Left Atrial Enlargement (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

Clinical Pearl
Differential Diagnosis of tall R wave in V_1
❑ RVH, Posterior MI, RBBB, WPW, Hypertrophic cardiomyopathy (septal hypertrophy), Duchenne's Muscular Dystrophy, counterclockwise rotation

ISCHEMIA/INFARCTION

Criteria for Q wave infarct (two leads serving an arterial territory)
❑ during an AMI, the ECG changes with time may include
❑ ST segment elevation +/– tall peaked T waves "hyperacute T waves" (area of injury)
❑ Q waves develop (transmural infarcts only)
❑ T waves invert (ischemia)

Q Wave
❑ significant if > 1 mm wide (i.e. > 0.04 seconds in duration) or if > 1/3 the amplitude of QRS
❑ note leads where Q waves are present (Q in III and V_1 is normal)

ST Segment
❑ elevation
- acute myocardial infarction
- Prinzmetal's angina (coronary vasospasm)
- other causes - acute pericarditis, ventricular aneurysm
- post MI
- early repolarization (normal variant)

❑ depression
- angina (ischemia)
- subendocardial infarction (non Q-wave MI)
- positive stress test
- acute posterior wall MI (V_1 and V_2)
- LVH "strain", LBBB
- digitalis effect ("scooping" or "hockey stick")

T Wave
❑ adults may have flat or slightly inverted T waves in limb leads
❑ note abnormally inverted T waves or changes from old ECGs
❑ biphatic T waves always present before chemia

Criteria for Non-Q-Wave MI (Subendocardial Infarctions)
❑ nonspecific ECG changes: T wave inversion; ST segment increased, decreased or <—>
❑ diagnosis depends on increased cardiac enzymes in presence of chest pain, +/– abnormal ECG

Table 5. Areas of Infarction

Infarct Area	Vessel	Q waves
anteroseptal	LAD	V_1, V_2
localized anterior		V_3, V_4
anterolateral		V_5, V_6
extensive anterior		V_1 - V_6
inferior	RCA (80-90%)	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)	RV3 and RV4 (right sided chest leads)

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

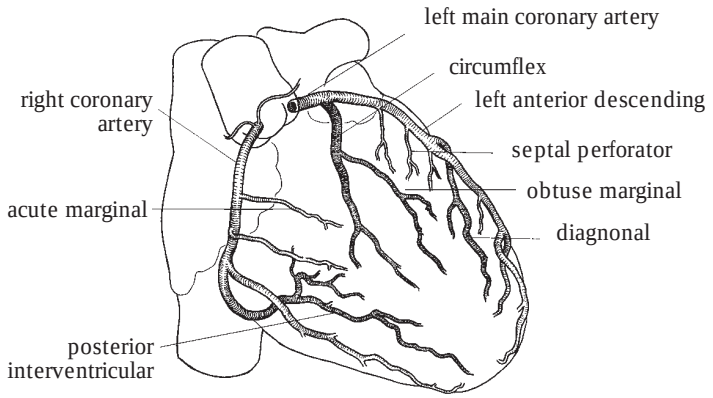


Figure 4. Anatomy of the Coronary Arteries (right anterior oblique projection)

Variations in Cardiac Vascular Anatomy
- ❑ Table 5 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

MISCELLANEOUS ECG CHANGES

Electrolyte Disturbances
- ❑ hyperkalemia (Appendix 5a)
  - peaked T waves (Mexican hat), flat P wave, wide QRS, long PR interval, elevated ST segment
  - ultimately the QRS and T waves merge to form a sine wave and VF may develop
- ❑ hypokalemia
  - T wave flattening, U waves, ST depression, prolonged Q-T interval
- ❑ hypocalcemia
  - prolonged Q-T interval
- ❑ hypercalcemia
  - shortened Q-T interval

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

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

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

Drugs
- ❑ Digoxin (Appendix 5b)
- ❑ 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 atrial fibrillation

❑ toxic levels associated with
- tachyarrhythmias with conduction blocks
- PAT with block is most characteristic
- PVC's, bigeminy
- classic "regularization" of ventricular rate in AF due to complete AV dissociation

❑ Quinidine
- prolonged QT interval, U waves

❑ Phenothiazines and TCAs
- changes similar to quinidine

Other Cardiac Conditions

❑ HCM
- ventricular hypertrophy, LAD, septal Q waves

❑ myocarditis
- conduction blocks, low voltage

Pulmonary Disorders

❑ COPD
- low voltage, RAD, poor R wave progression
- chronic Cor pulmonale can produce P pulmonale and RVH with strain
- multifocal atrial tachycardia

❑ massive pulmonary embolus
- sinus tachycardia and AF are the most common arrhythmias
- RVH with strain, RBBB, S_1, Q_3, T_3 (inverted T)

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 arrythmias
  2. relate symptoms to dysrhythmias
  3. detect myocardial ischemia

ECHOCARDIOGRAPHY

❑ Two-dimentional (2-D) echo = anatomy - U/S reflecting from tissue interfaces
- determines:
  - left ventricular systolic ejection fraction
  - chamber sizes
  - wall thickness
  - valve morphology
  - pericardial effusion
  - wall motion abnormalities
  - complications of AMI

❑ Doppler = blood flow- U/S reflecting from intracardiac RBCs
- determines: blood flow velocities using gradient (= $4v^2$) to estimate aortic and mitral valve areas

❑ Colour flow imaging
- determines:
  - valvular regurgitation
  - valvular stenosis
  - shunts

❑ Transesophageal Echo
- 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 and valvular vegetations, infective endocarditis
  - aortic dissection

EXERCISE TESTS

❑ indications:
- assessment of chest pain
- risk stratification post-MI
- assessment of therapy

❑ Standard Exercise Test
- patient exercises on a treadmill or bicycle

- sensitivity 65-70%, specificity 65-70%
- pretest likelihood of CAD is very important
- patient must be able to exercise
- advantages: assessment of ischemia, functional class, prognosis, accuracy tested in different populations
- disadvantages: sensitivity lower than stress imaging studies, specificity poor with marked ST-T abnormalities on resting ECG, digoxin, LBBB, pacemakers or in females, does not accurately localize site or extent of myocardial ischemia

❑ Pharmacologic induced stress test with imaging (nuclear or echo)
- sensitivity 80%, specitivity 85-90%
- increased coronary flow: dipyridamole/persantine, adenosine
- increased myocardial O_2 demand: dobutamine

❑ Stress Echo
- sensitivity 90%, specificity 90%
- provides information on the presence and extent of coronary disease
- assess multiple parameters (see 2-D echo)

RADIONUCLIDE ANGIOGRAPHY

❑ Tc labelled RBCs to assess EF
- indications: risk-stratification post-MI
  LVF
  CHF
- EF= $\frac{EDV-ESV}{EDV}$
- good images in patients with COPD or obesity

NUCLEAR IMAGING

❑ sensitivity 85%, specificity 90%
- assess:
  - myocardial perfusion
  - blood flow
  - localize and quantify myocardial ischemia and infarction
  - myocardial metabolism

Table 6. Imaging in Cardiac Disorders

Myocardial Ischemia (reversible)	
Stress-delayed-re-injection thallium	low uptake during stress with complete or partial uptake in delayed or re-injection images (ie reversible defect)
Rest stress sestamibi	normal uptake at rest with decreased uptake during stress
Dobutamine stress echo	wall motion abnormality with stress
PET	decrease flow with normal or increased uptake during stress
Myocardial infarct (fixed)	
Stress-delayed-re-injection thallium	low uptake during stress and after re-injection (ie fixed defect)
Rest-stress sestamibi	low uptake in rest and stress images
Dobutamine stress echo	wall motion abnormality at rest and with stress
PET	decreased flow and decreased uptake at rest
"Hibernating " Myocardium:	
rest-delayed thallium	complete or partial uptake or defects after re-injection
dobutamine stress echo	wall motion abnormalities with stress, "contractile reserve"
PET	decreased flow and increased uptake at rest
Assessment of ventricular function:	
Tc-99m RBC gated blood pool imaging	assessment of global left and right ventricular function at rest or during exercise, accurate determination of ejection fraction
Echo	regional wall motion and function, estimate of ejection fraction

MECHANISMS OF ARRHYTHMIAS

- ❑ altered impulse formation
- ❑ altered impulse conduction

ALTERED IMPULSE FORMATION

- ❑ 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 that selectively increase automaticity of pacemakers which are normally slower than SA node
    - e.g. digoxin, which has vagal effect on SA and AV nodes but sympathetic effect on other pacemaker sites
  - local ischemia/pathology
  - blockage of proximal pacemaker (SA node) impulses which allows more distal focus to control the ventricular rhythm
- ❑ triggered activity
  - oscillations of the membrane potential after normal depolarization lead to recurrent depolarization
  - prolonged QT interval predisposes (e.g. electrolyte disturbances, drugs)
  - postulated mechanism of Torsades de Pointes

ALTERED IMPULSE CONDUCTION

- ❑ re-entry
  - phenomenon which requires parallel electrical circuit in which two limbs have different refractory periods, e.g. AF, AVNRT
- ❑ conduction blocks - partial or total
- ❑ ventricular preexcitation
  - 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

- ❑ stretch of myocardial cells is arrhythmogenic; hence, increased LA size —> AF
- ❑ bradycardia predisposes via temporal dispersion in refractory periods; e.g. tachy-brady syndrome; protection via pacing or atropine
- ❑ hypoxia/acidosis lowers the threshold for VF; hence the protective role of O_2 + bicarbonate
- ❑ electrolyte disturbances, e.g.: hypokalemia, imbalances of Ca^{++}, Mg^{++}
- ❑ infection, e.g.: myocarditis or infective endocarditis (causing aortic root abscess)
- ❑ cardiomyopathies, degenerative disease, infiltration (e.g. sarcoid)

CLINICAL APPROACH TO ARRHYTHMIAS

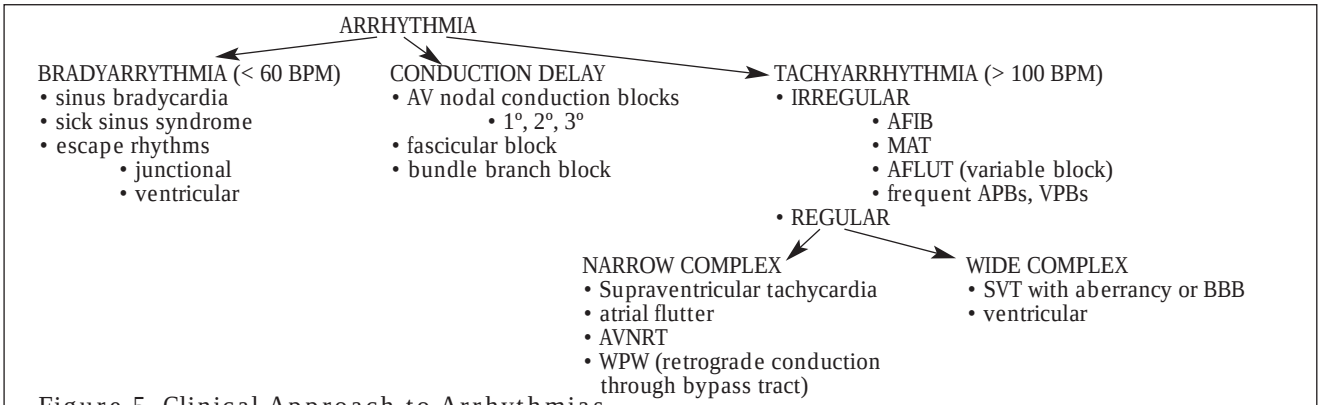


Figure 5. Clinical Approach to Arrhythmias

BRADYARRHYTHMIAS

SA NODE

Sinus Bradycardia (Appendix 1a)

- ❑ regular heart rate less than 60 bpm with normal P wave preceding each QRS
- ❑ excessive vagal tone: spontaneous (vasovagal syncope), acute (inferior) MI, drugs, vomiting, hypothyroidism, increased ICP
- ❑ treatment: if symptomatic, atropine and/or 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

Sick Sinus Syndrome

- ❑ SSS = inappropriate sinus bradycardia
- ❑ bradycardia may be punctuated by episodes of SVT, especially AF or atrial flutter ("Tachy-Brady Syndrome")
- ❑ usually elderly; younger patient. with cardiomyopathies
- ❑ syncope
- ❑ treatment = pacing for brady; meds for tachy

Sinus Arrest or Exit Block (Appendix 1b)

- ❑ 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
        - as a result, a normal P wave is not seen
        - occasionally a retrograde P wave may be seen 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
- ❑ treatment: stop meds which suppress the sinus node (beta-blockers, CCB, digoxin); may need pacing

TACHYARRHYTHMIAS

SUPRAVENTRICULAR

- ❑ 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 (Appendix 2a)

- ❑ regular heart rate greater than 100 bpm with P wave preceding QRS
- ❑ normal P wave morphology
- ❑ occurs with 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)
    - a single ectopic supraventricular beat that originates in the atria
    - the 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; beta-blockers, or calcium channel blockers

Atrial Flutter (Appendix 2b)

❑ regular; atrial rate 250-350 bpm, usually 300

Clinical Pearl

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

❑ etiology: IHD, thyrotoxicosis, MV disease, cardiac surgery, COPD, PE, pericarditis
❑ 2:1, 3:1, 4:1, etc... block (may be variable) i.e. ventricular rate one half, one third, etc... the atrial rate
❑ ECG: sawtooth inferior leads; narrow QRS (unless aberrancy)
❑ carotid massage (check first for bruits), Valsalva or adenosine: increases the block, brings out flutter waves
❑ treatment
- rate control: beta-blocker, verapamil, digoxin
- medical cardioversion: procainamide, sotalol, amiodarone, quinidine
- electrical cardioversion: DC shock (@ low synchronized energy levels: start at 50J)
- anticoagulation usually not necessary

Multifocal Atrial Tachycardia (MAT)

❑ irregular rhythm; atrial rate 100-200 bpm; at least 3 distinct P wave morphologies 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 verapamil; or, if not contraindicated, metoprolol

Atrial Fibrillation (AF) (Appendix 2c)

❑ seen in 10% of population over 75 years old
❑ the majority of cardiogenic strokes and peripheral thromboembolic events occur in association with AF
❑ 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
❑ AF resistant to cardioversion - LA > 50 mm, longer duration of AF
❑ major issues to be addressed with AF: (RACE)
- Rate control (ventricular)
  - digoxin, beta blockers, verapamil, diltiazem
  - maintenance of sinus rhythm - sotalol, amiodarone or Class I if normal LV function
- Anti-coagulation (prevention of thromboembolic phenomenon)
  - warfarin for paroxysmal or chronic AF
  - balance risk of bleeding 1%/year versus risk of clot
- Cardioversion (to sinus rhythm)
  - OK without anticoagulation within 48 hours of onset
  - if > 48 hours of onset MUST anticoagulate prior to cardioversion (at least 3 weeks before and 4 weeks after cardioversion)
  - alternate option is TEE prior to electrical cardioversion to rule out clot

- medical cardioversion- sotalol, amiodarone, Class I agent if normal LV function (e.g. IV procainamide, propafenone)
- electrical cardioversion- synchronized DC cardioversion (start at 300J)

- Etiology
  - CAD, valvular disease, pericarditis, cardiomyopathy, PE, hypertension, COPD, thyrotoxicosis, tachy-brady syndrome, EtOH (holiday heart)

Paroxysmal Supraventricular Tachycardia (PSVT) (Appendix 2d)

- ❑ 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
- ❑ AVNRT accounts for 60-70% of all SVT's
- ❑ retrograde P waves may be seen but are usually lost in the QRS complex
- ❑ asymptomatic or palpitations
- ❑ may precipitate CHF or hypotension if underlying disease
- ❑ treatment
  - acute: Valsalva or carotid massage (check first for bruits), adenosine especially if associated with WPW (adenosine is first choice if unresponsive to vagal maneuvers); if no response, try verapamil, metoprolol, then digoxin; DC shock if signs of cardiogenic shock, angina, or CHF
  - chronic: beta-blocker, verapamil, digoxin, anti-arrhythmic drugs, EPS catheter ablation

VENTRICULAR

Premature Ventricular Contraction (PVC or VPB) (Appendix 2e)

- ❑ QRS width greater than 0.12 seconds, no preceding P wave
- ❑ premature in the cardiac cycle, may be followed by a prolonged pause
- ❑ origin: LBBB pattern = RV site; RBBB pattern = LV site
- ❑ rules of malignancies with PVC's (seen in CAD, HTN, COPD)
  - 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 VF)
- ❑ include risk of sudden death if associated with CAD, HCM, MVP; risk not altered by treatment of PVCs
- ❑ treatment: since no evidence to suggest that treatment reduces mortality, PVCs are not usually treated
  - if symptomatic, use lidocaine acutely and may consider procainamide, quinidine, beta blocker or disopyramide if chronic

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
- ❑ sometimes seen during AMI (especially during reperfusion) or digoxin toxicity
- ❑ regular rhythm; rate 50-100 bpm
- ❑ rarely sustained and rarely requires treatment
- ❑ treatment: if symptomatic, lidocaine, atropine

Ventricular Tachycardia (VT) (Appendix 2f)

- ❑ a run of three or more consecutive PVCs rate > 100 is called VT
- ❑ reentry accounts for the majority
- ❑ sustained VT is an emergency, prestaging cardiac arrest and requiring immediate treatment
- ❑ most common form of heart disease predisposing to VT is CAD with MI
- ❑ rate 120-300 bpm
- ❑ broad QRS, AV dissociation, fusion beats, capture beats, left axis deviation, monophasic or biphasic QRS in V1 with RBBB, concordance V_1-V_6
- ❑ AV dissociation
  - the atria and ventricles beat independently of one another, thereby producing cannon "a" waves in the jugular venous system; P waves "march through" unrelated to QRS complexes

- ❑ 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
    - distinguish from SVT with aberrancy (see table)
    - DC shock, lidocaine, procainamide, bretylium, amiodarone

Ventricular Fibrillation (VF) (Appendix 2g)

- ❑ 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 VF
- ❑ CPR, electrical defibrillation, epinephrine, lidocaine. If VF persists, Bretylium, $MgSO_4$, procainamide, amiodarone
- ❑ refer to ACLS algorithm for complete therapeutic guidelines

Torsades de Pointes (Appendix 2h)

- ❑ polymorphic VT - it 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
- ❑ a form of VT seen in patients with prolonged QT intervals
  - congenital long QT syndromes
  - drugs - Class IA (quinidine), Class III (sotalol), phenothiazines, tricyclic antidepressants
  - electrolyte disturbances - hypokalemia, hypomagnesemia
  - other - nutritional deficiencies
- ❑ treatment: temporary pacing, IV magnesium, correct underlying cause of prolonged QT, DC cardioversion if hemodynamic compromise present

Differentiation of VT vs. SVT with Aberrant Conduction*

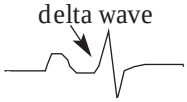
	VT	SVT
Clinical Clues		
carotid massage	no response	may terminate
cannon "a" waves	may be present	not seen
neck pounding	may be present	not seen
ECG Clues		
AV dissociation	may be seen	not seen
fusion beats	may be seen	not seen
initial QRS deflection	may differ from normal QRS complex	same as normal 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 Syndrome (Appendix 3a)

- ❑ bypass pathway called the Bundle of Kent connects the atria and ventricles
- ❑ congenital defect, present in 3:1000
- ❑ 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



- ❑ the two tachyarrhythmias most often seen in WPW are PSVT and AF
- ❑ carotid massage, vagal maneuvers, and adenosine can enhance the degree of preexcitation by slowing AV nodal conduction
- ❑ note: if wide complex atrial fibrillation, 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 increase conduction through the bypass tract and precipitate VF

Lown-Ganong-Levine Syndrome

- ❑ the accessory pathway (James fibres) is intranodal, bypassing the delay within the AV node
- ❑ the PR interval is shortened to less than 0.12 seconds
- ❑ the QRS complex is narrow and there is no delta wave

CONDUCTION DELAYS

AV NODE

Conduction Block

- ❑ look at the relationship of the P waves to the QRS complexes
- ❑ 1st degree - constant prolonged PR interval (> 0.2 seconds) (Appendix 1c)
  - all beats are conducted through to the ventricles
  - no treatment required if asymptomatic
- ❑ 2nd degree - not all sinus P waves are followed by QRS; distinguish Type I from Type II
  - Mobitz type I (Wenckebach) - due to AV node blockage (Appendix 1d)
    - progressive prolongation of the PR interval until a QRS is dropped
    - treatment: none unless symptomatic; atropine
  - Mobitz type II - due to His-Purkinje blockage (Appendix 1e)
    - all-or-none conduction; QRS complexes are dropped at regular intervals without PR prolongation
    - stable PR interval (normal or prolonged)
    - risk of developing syncope or complete HB
    - can have 2:1 or higher blocks
    - requires insertion of a pacemaker (ventricular or dual chamber)
  - 3rd degree or complete HB (Appendix 1f)
    - no P wave produces a QRS response
    - complete AV dissociation (no relationship between and QRS)
    - can have narrow junctional QRS or wide ventricular QRS (junctional vs. ventricular escape rhythm); depends on where escape comes from
    - rate usually 30-60 bpm
    - Stokes-Adams attacks
    - treatment: pacemaker (ventricular or dual chamber)

BUNDLE BRANCH AND FASCICULAR

- ❑ RBBB, left anterior fasciculus and left posterior fasciculus should each be considered individually, and combination (i.e., bifascicular) blocks should also be noted

Bundle Branch Blocks

- ❑ QRS complex > 0.12 seconds
- ❑ RBBB (Appendix 4a)
  - RSR' in V_1 and V_2 (rabbit ears), with ST segment depression and T wave inversion
  - presence of wide slurred S wave in I, V_6
  - widely split S_2 on auscultation
- ❑ LBBB (Appendix 4b)
  - 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

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 degrees), with no other cause present
  - small 'q' in I and aVL, small 'r' in II, III, aVF
- ❑ posterior hemiblock
  - normal QRS duration; no ST segment or T wave changes
  - right axis deviation (>110 degrees), with no other cause
  - small 'r' in I and aVL, small 'q' in II, III and aVF

PACEMAKER INDICATIONS

- ❑ sinus node dysfunction
  - symptomatic bradycardia
- ❑ AV nodal block
  - symptomatic Mobitz I
  - bifascicular block
- ❑ infranodal block
  - Mobitz II
  - complete HB
- ❑ symptomatic carotid hypersensitivity

PACING TECHNIQUES

- ❑ temporary: transvenous (jugular, subclavian, femoral) or external pacing
- ❑ permanent: transvenous into R atrium, 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. V V I

  V - chamber paced : ventricle
  V - chamber sensed: ventricle
  I - action : inhibit

BACKGROUND

Epidemiology

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

Pathophysiology of Myocardial Ischemia

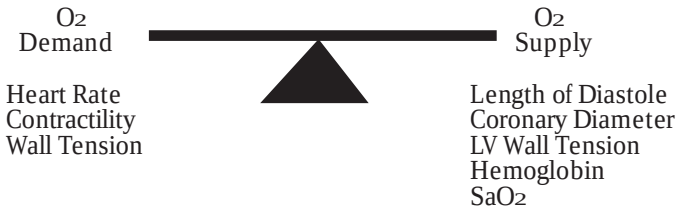


Figure 6. Physiological Principles

Atherosclerosis and Ischemic Heart Disease

- ❑ atherosclerosis and thrombosis are by far the most important pathogenetic mechanisms in ischemic heart disease

Major Risk Factors For Atherosclerotic Heart Disease

- ❑ smoking
  - risk can be halved by cessation of smoking
- ❑ diabetes
  - micro and macrovascular complications
- ❑ hypertension
  - depends on degree and duration
- ❑ family history
  - first degree male relative < 55 or first degree female relative < 60
- ❑ hyperlipidemia

Minor Risk Factors

- ❑ obesity
  - > 30% above ideal weight
- ❑ sedentary lifestyle
- ❑ major depression – increases the risk for fatal and non-fatal IHD and 1/3 of acute post-MI patients are depressed
- ❑ hyperhomocysteinemia

Preventative Measures

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

ANGINA PECTORIS

Definition

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

Etiology
- ❑ reduced myocardial oxygen supply
  - atherosclerotic heart disease (vast majority)
  - coronary vasospasm (variant angina)
  - severe aortic stenosis or insufficiency
  - thromboembolism
  - severe anemia
  - arteritis
  - dissection
  - congenital anomalies
- ❑ increased myocardial oxygen demand
  - myocardial hypertrophy
  - severe tachycardia
  - severe hyperthyroidism
  - severe anemia

Differential Diagnosis
- ❑ MSK disease
  - rib fracture
  - intercostal muscle tenderness
  - costochondritis
  - intercostal neuritis (shingles)
  - nerve root disease (cervical radicultis)
- ❑ GI disease
  - PUD
  - reflux esophagitis
  - esophageal spasm and motility disorder (may be improved by nitro)
- ❑ pulmonary disease
  - PE
  - pneumothorax
  - pneumonia
- ❑ CV disease
  - aortic dissection (assymetrical BP and pulses, new AI murmur)
  - pericarditis
- ❑ 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"
  - 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
- ❑ stress testing (see Cardiac Diagnostic Tests Section)

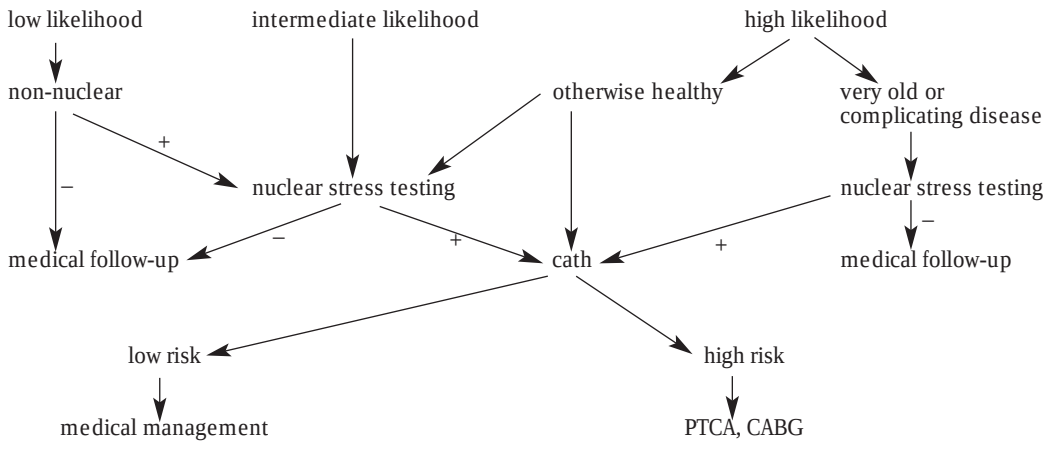


Figure 7. Diagnostic Strategies in the Management of IHD

Variant 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
- ❑ typically ST elevation on ECG (may be confused with acute infarction)
- ❑ diagnose by provocative testing with ergot vasoconstrictors (rarely done)

Medical Treatment

- ❑ beta-blockers (first line therapy)
  - reduce overall mortality
  - reduce heart rate, contractility, and to a lesser degree, blood pressure (afterload)
  - also increase coronary perfusion
  - avoid agents with intrinsic sympathomimetic activity (ISA) unless patient is bradycardic
- ❑ calcium channel blockers (second line therapy)
  - centrally acting: variably decrease afterload and contractility and produce coronary dilatation
- ❑ nitrates
  - used for symptomatic control
  - no clear impact on survival
  - reduce myocardial work and, therefore, oxygen requirements through venous dilatation (decreased preload) and arteriolar dilatation (decreased afterload)
  - also dilate coronary arteries
  - maintain daily nitrate-free intervals to try to prevent tolerance ("drug holiday")
- ❑ ECASA
  - all patients
  - decrease platelet aggregation
- ❑ lipid lowering

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%

CAD-NCEP Guidelines

	Diet	Drug	Goal
Primary Prevention			
< 2 risk factors	>/=4.1	>/=4.9	< 4.1
2 risk factors	>/=3.4	>/+4.1	< 3.4
Secondary Prevention			
based on LDL-C levels	> 2	>/=3.4	</=2.6

- ❑ treatment strategy
  - short acting nitrates on prn basis to relieve acute attacks and prn prior to exertion
  - good prophylactic combination regimens include:
    - beta-blocker and long-acting nitrate
    - beta-blocker and calcium channel blocker (long acting or peripherally acting -second generation dihydropyridine group)
  - be careful when combining beta-blockers and verapamil/diltiazem
    - both depress conduction and contractility and may result in sinus bradycardia or AV block
  - carefully consider non-cardiac adverse effects
  - use nitrates and calcium channel blockers for variant angina

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
- ❑ primary success rate is > 80%
- ❑ restenosis occurs in approximately 30-50% of dilated vessels within the first 6 months (dependent upon location)
- ❑ use of intracoronary stent is associated with a lower restenosis rate and reduces need for urgent CABG in patients with threatened vessel closure at time of PTCA
- ❑ complications (overall 3-5%)
  - mortality < 1%
  - MI 3-5%
  - intimal dissection + vessel occlusion requiring urgent CABG in 3-5%

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 or "equivalent"
    - three-vessel disease with depressed LV function
    - multi-vessel disease with significant proximal LAD stenosis
  - unstable angina
    - continuing angina despite aggressive medical therapy (unstable angina)
    - evolving myocardial infarction (post infarct angina)
    - 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 or MI at 3 years, but more revascularization and recurrent ischemia in PTCA group
  - BARI, subset analysis - CABG superior in patients with diabetes mellitus 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, comorbid 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)

UNSTABLE ANGINA

Definition
- ❑ accelerating pattern of pain
  - increased frequency
  - longer duration
  - occuring with less exertion
  - less responsive to treatment
- ❑ angina at rest
- ❑ new onset angina
- ❑ angina post-MI
- ❑ post-angiography
- ❑ post-CABG
- ❑ note that unstable angina is a heterogenous 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
- ❑ 10-15% will progress to MI
- ❑ 5-10% one year mortality

Diagnosis
- ❑ history
- ❑ ECG changes
  - ST depression or elevation
  - T wave inversion
- ❑ no elevation of cardiac enzymes

Management
- ❑ oxygen
- ❑ bed rest
- ❑ hospitalization/monitoring
- ❑ anti-anginal medications
  - sublingual or IV nitroglycerine
  - beta-blockers are first line therapy
    - aim for resting heart rate of 50-60
  - calcium channel blockers are second line therapy
    - evidence suggests that they do not prevent MI or reduce mortality
    - be cautious using verapamil/diltiazem with beta-blockers
    - may use amlodipine or long acting nifedipine if concomitant beta blockade
- ❑ aspirin
  - 160-325 mg/day, although lower doses have proven beneficial
- ❑ IV heparin
- ❑ angiography with view to potential PTCA or CABG
- ❑ if aggressive medical management is unsuccessful
  - may use intra-aortic balloon pump to stabilize before proceeding with revascularization
  - proceed to emergency angiography and PTCA or CABG

SUDDEN DEATH

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

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

Etiology
- ❑ primary cardiac pathology
  - ischemia/MI
  - left ventricular dysfunction
  - severe ventricular hypertrophy
    - hypertrophic CM
    - AS
  - QT prolongation syndrome
  - congenital heart disease
- ❑ high risk patients may have in common
  - multi-vessel disease
  - ventricular electrical instability (i.e. VPBs)
  - repolarization abnormalities on signal-averaged ECG
  - LV dysfunction
- ❑ antecedent rhythms to VF
  - VT (62%)
  - bradyarrhythmias (16%)
  - torsade de pointes (12%)
  - primary VF (8%)

Management

Acute
- ❑ resuscitate with prompt CPR and defibrillation

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

Treatment
- ❑ antiarrhythmic drug therapy
  - amiodarone, beta-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

ACUTE MYOCARDIAL INFARCTION

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

Diagnosis

(Dx infarction 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
- ❑ ECG changes
  - hyperacute T waves
  - ST segment elevation
  - T wave inversion
  - significant Q wave
- ❑ 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
    - diabetics
    - elderly
    - patients with hypertension
- ❑ draw serum lipids within 24-48 hours because the serum values are unreliable after 48 hours, but become reliable again 8 weeks post MI

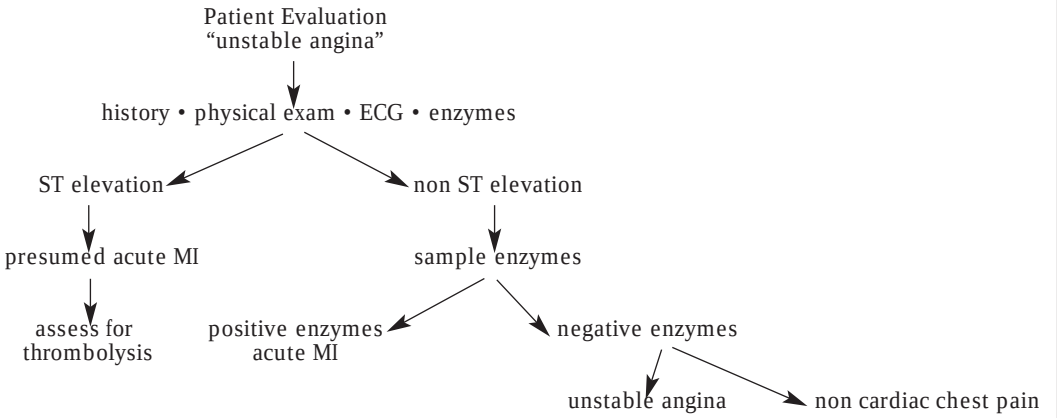


Figure 8. Diagnostic algorithm in acute IHD

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

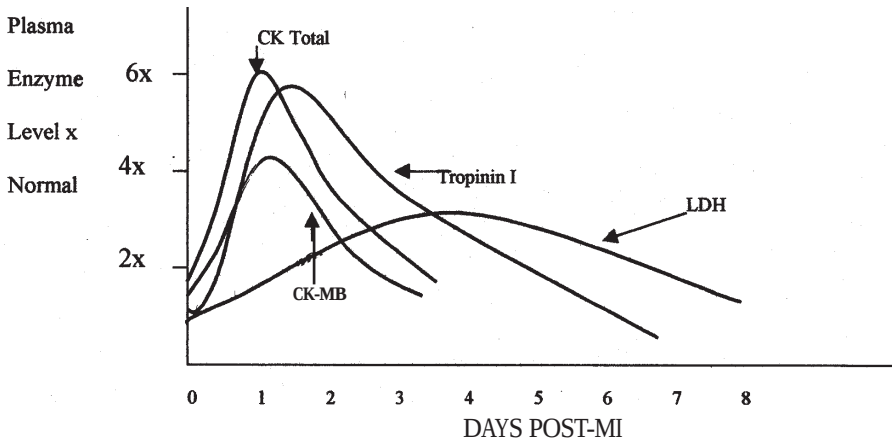


Figure 9. Cardiac Enzyme Profile in Acute MI

Classification of MIs
- ❑ Q wave
  - • associated with transmural infarctions, involving full thickness of myocardium
- ❑ non-Q wave
  - • associated with non-transmural (subendocardial) infarctions, involving one third to one half 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
  - aspirin 325 mg chewed stat
  - oxygen
  - sublingual nitroglycerine x 3 to r/o angina
  - morphine for pain relief and sedation
  - beta-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 diabetes
  - strongly recommended that patients with the following should receive 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 LBBB

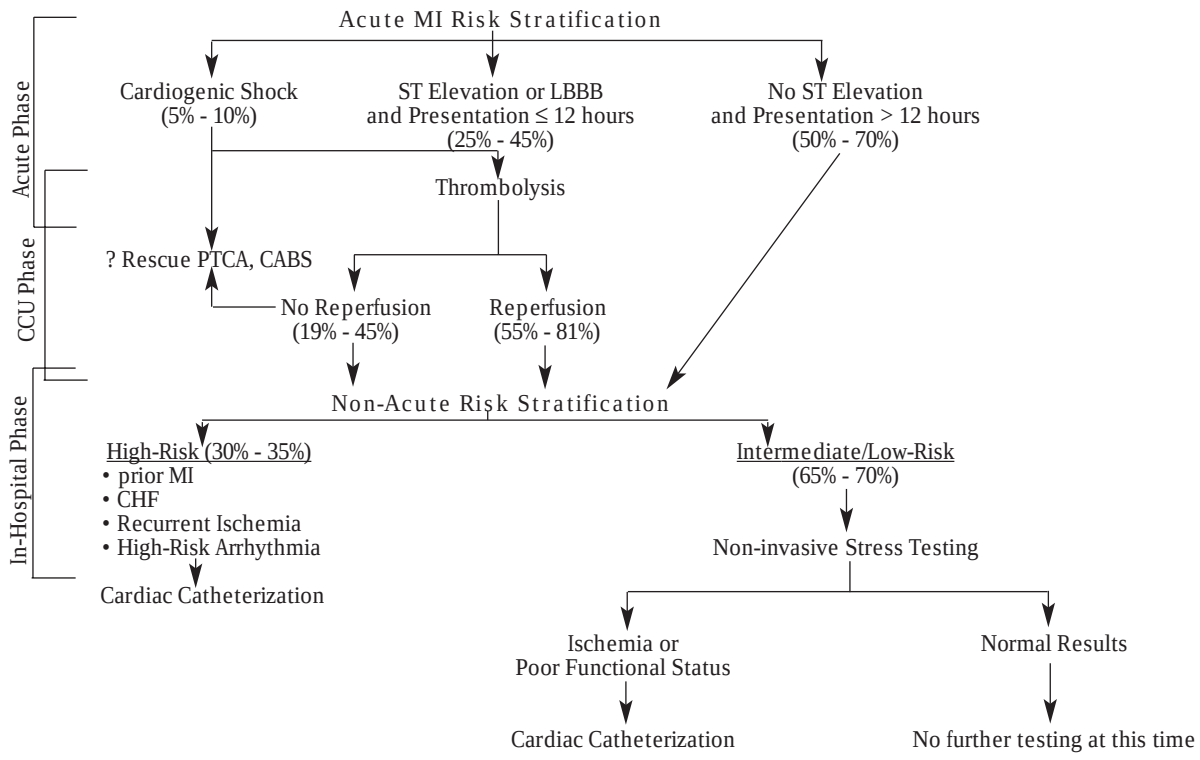
    C. presentation within 12 hours of symptom onset
    - choice of thrombolytic agents include streptokinase and rt-PA
    - patients having previously received streptokinase must receive alternate agent due to development of immunity
- ❑ PTCA, CABG
- ❑ Long-term measures
  - antiplatelet/anticoagulation therapy
  - ECASA 325 mg daily
  - heparin
  - for all patients, especially if high risk of systemic or venous thromboembolism (anterior MI, atrial fibrillation, ventricular aneurysm)
  - nitrates
    - alleviate ischemia but may not improve outcome
  - beta blockers (first line therapy)
    - start immediately and continue indefinitely if no contraindications
    - reduce mortality
  - calcium channel blockers
    - NOT recommended in Q-wave MI
    - diltiazem of questionable benefit in non-Q wave MI (if no LV dysfuntion)
  - ACE-inhibitors
    - all patients should be considered for ACEI
    - reduce mortality
    - 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 10 for post-CCU strategy

Table 7. Contraindications to Thrombolytic Therapy in AMI

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

Table 8. Complications of Myocardial Infarction

Complication	Etiology	Presentation	Therapy
arrhythmia			
(a) tachy	sinus, AF, VT, VF	early/late	see Arrhythmia section
(b) brady	sinus, AV block	early	
myocardial rupture			
(a) LV free wall	transmural infarction	1-7 days	pericardiocentesis or surgery
(b) pap muscle (MR)	inferior infarction anterior infarction	1-7 days	surgery
(c) vent 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 10. Acute MI and Predischarge Risk Stratification

Prognosis
- ❑ 20% of patients with acute MI die before reaching hospital
- ❑ 5-15% of hospitalized patients will die
  - risk factors
    - infarct size/severity
    - age
    - comorbid 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 LV ejection fraction is most useful prognostic factor

HEART FAILURE

- ❑ the overall prognosis of patients with CHF remains 50% mortality at five years

DEFINITION
- ❑ 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)
- ❑ not a disease entity in and of itself but rather a syndrome involving components from the forward and backward heart failure theories

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

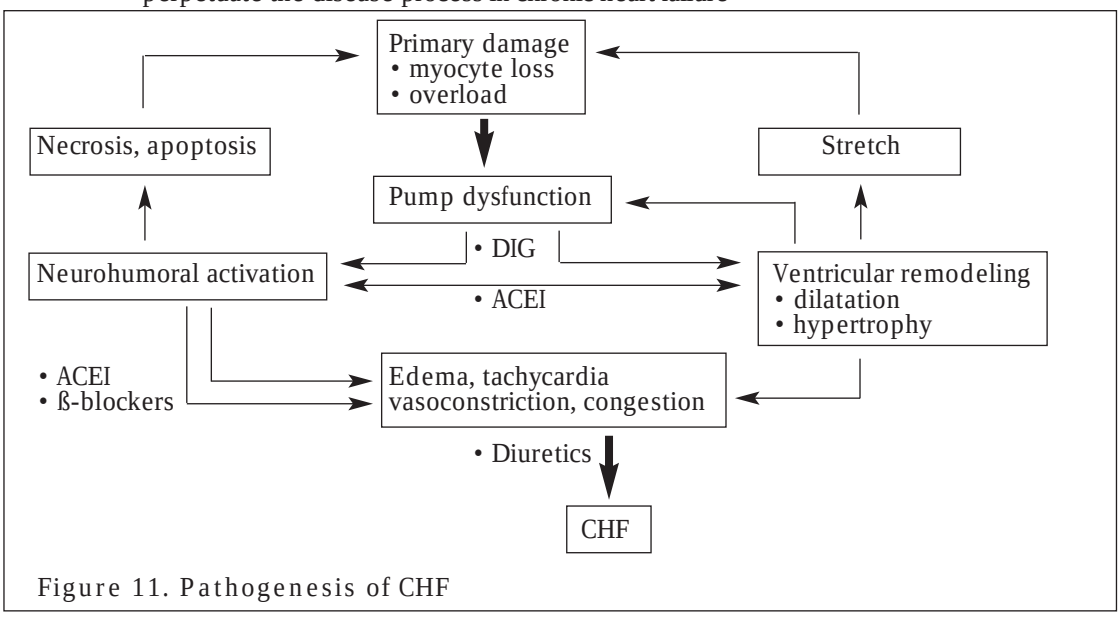


Figure 11. Pathogenesis of CHF

ETIOLOGY OF PRIMARY INSULTS
- ❑ consider predisposing, precipitating and perpetuating factors

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)
- hypertension (may produce hypertrophic cardiomyopathy)
- alcohol (may cause dilated cardiomyopathy)

❑ the less common causes of CHF
- toxic e.g. adriamycin, doxorubicin, radiation, uremia, catecholamines
- infectious e.g. Chagas (very common cause worldwide), coxsackie, HIV
- endocrine e.g. hyperthyroidism, diabetes, acromegaly
- infiltrative e.g. sarcoidosis, amyloidosis, hemochromatosis, neoplasia
- genetic e.g. hereditary hypertrophic cardiomyopathy
- metabolic e.g. thiamine defiency, selenium deficiency
- peripartum
- congenital

❑ precipitants
- lack of compliance with diet and medications, inadequate therapy
- uncontrolled hypertension
- arrhythmias e.g. atrial fibrillation
- recurrent ischemia
- disease progression
- environmental e.g. heat wave
- intercurrent infection, fever
- pulmonary embolism
- thyrotoxicosis

❑ 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. hypertension)
- 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 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

Table 9. "Overshooting" of Compensatory Responses in Heart Failure

Compensatory Response	Result of Excess
hypertrophy	increased O_2 consumption diastolic dysfunction
dilatation	impaired myocardial function
salt and H_2O retention	venous congestion
increased heart rate and contractility	increased O_2 consumption
increased systemic vascular resistance	decreased cardiac output

SYSTOLIC vs. DIASTOLIC DYSFUNCTION

Systolic Dysfunction (defect in the 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

- ❑ systolic dysfunction may lead to diastolic dysfunction when compensatory responses of hypertrophy/dilatation result in increased end-diastolic pressure
- ❑ 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)
- ❑ ability of left ventricle to accept blood is impaired due to a lack of 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 (ie. pulmonic and systemic venous congestion)
- ❑ diastolic dysfunction may lead to systolic dysfunction when compensatory responses of dilatation/hypertrophy lead to decreased EF
  - clues to diagnosis: S_4, HTN, LVH on ECG/ECHO, normal EF
  - treatment: beta blockers, verapamil or diltiazem

Table 10. 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 mitral regurgitation Cheyne-Stokes breathing pulsus alternans S_3	dyspnea tricuspid regurgitation S_3
venous congestion (backward)	dyspnea orthopnea PND basal crackles cough hemoptysis S_4	peripheral edema hepatomegaly hepatic tenderness pulsatile liver elevated JVP positive HJR Kussmaul's sign S_4

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"
- ❑ differential diagnosis includes anemia, thiamine deficiency, hyperthyroidism, A-V fistula, Paget's disease

INVESTIGATIONS

- ❑ work up involves assessment for precipitating factors and treatable causes of CHF
- ❑ bloodwork
  - CBC
  - lytes
    - dilutional 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
    - alveolar filling if gross pulmonary edema
  - also look for
    - cardiomegaly (C/T > 0.5)
    - atrial enlargement
    - pericardial effusion
    - pleural effusion
- ❑ echocardiography is the primary diagnostic method to determine
  - ejection fraction (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

MANAGEMENT

- ❑ 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
- ❑ control of sodium and fluid retention
  - sodium restriction (2 gm), requires patient education
  - fluid restriction and monitor daily weights
  - diuretics (no effect on mortality and purely symptomatic) except spirnolactone (Rales study)
    - thiazides for mild heart failure
    - furosemide for potent diuresis
    - metalozone may be used with furosemide to increase diuresis
- ❑ vasodilators
  - goal is to arteriodilate (decrease afterload) and venodilate (decrease preload), thereby improving systolic function and venous congestion
  - in hospital, monitor response to therapy with daily weights and measurement of fluid balance and follow renal function
  - ACE inhibitors: 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 ACE inhibitors
  - decrease in mortality not as great as with ACE inhibitors
- amlodipine
  - may be of benefit in dilated cardiomyopathy
- angiotensin II receptor blockers e.g. losartan
  - preliminary evidence suggests benefit

❑ inotropic support
- digitalis
  - improves symptoms and decreases hospitalizations (DIG trial)
  - no impact on survival
  - excellent choice in setting CHF with atrial fibrillation
- sympathomimetics
  - potent agents used in ICU/CCU settings
  - dopamine
    - "low-dose" causes selective renal vasodilation
    - "medium-dose" provides inotropic support
    - "high-dose" increases systemic vascular resistance, which in most cases is undesirable
  - dobutamine
    - selective inotropic agent
    - also produces arterial vasodilation
- phosphodiesterase inhibitors
  - effects similar to dobutamine
  - adverse effect on survival when used as oral agent (PROMISE study)

❑ other agents
- beta-blockers - recommended for 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 functional class II-III CHF
  - metoprolol has been shown to delay time to transplant, reduce hospitalizations in dilated cardiomyopathy and to decrease mortality (MERIT study)
- calcium channel blockers (have equivocal effect on survival)
- antiarrhythmic, if required then amiodarone is drug of choice
  - class I anti-arrhythmics associated with increased mortality in CHF

ACUTE CARDIOGENIC PULMONARY EDEMA

Definition
❑ severe pulmonary congestion leading to extravasation of capillary fluid into 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
❑ Lasix - furosemide 40 mg IV, double dose q1h as necessary
❑ Morphine 2-4 mg IV q5-10 minutes
- decreases anxiety
- vasodilation

- ❑ Nitroglycerine topical 2 inches q2h (or IV nitroglycerine)
- ❑ Oxygen
- ❑ Positive airway pressure
  - (CPAP or BiPAP) decrease need for ventilation
- ❑ other vasodilators as necessary in ICU setting
  - nitroprusside (IV)
  - hydralazine (PO)
- ❑ inotropic support
- ❑ 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

- ❑ disease of the myocardium not secondary to coronary artery disease, valvular heart disease, congenital heart disease, hypertension or pericardial disease
- ❑ diagnosis of any of the following conditions mandates exclusion of the above conditions
- ❑ dilated cardiomyopathy
- ❑ hypertrophic cardiomyopathy
- ❑ restrictive cardiomyopathy
- ❑ myocarditis

DILATED CARDIOMYOPATHY

Etiology

- ❑ idiopathic
- ❑ peri-partum
- ❑ inflammatory
- ❑ infectious
  - post-viral (Coxsackie), Chagas, etc...
- ❑ non-infectious
  - collagen vascular disease
- ❑ neuromuscular disease - e.g. Duchenne
- ❑ toxic - alcoholic, adriamycin, cocaine, heroin, organic solvents; glue sniffer's heart
- ❑ metabolic
- ❑ nutritional
  - thiamine deficiency, selenium deficiency, carnitine deficiency
- ❑ endocrine - e.g. thyrotoxicosis, DM
- ❑ familial
- ❑ radiation

Pathophysiology

- ❑ impaired contractile function of the myocardium —> progressive cardiac dilatation and eventually, decreased ejection fraction
- ❑ clinical manifestations
  - CHF
  - systemic or pulmonary emboli
  - arrhythmias
  - sudden death

Investigations
- ❑ 12 lead ECG
  - ST-T wave abnormalities
  - conduction defects
  - arrhythmias
- ❑ chest x-ray
  - global cardiomegaly
  - signs of heart failure
- ❑ echocardiography
  - 4-chamber enlargement
  - depressed ejection fraction
  - mitral and tricuspid regurgitation secondary to cardiac dilatation
- ❑ endomyocardial biopsy: not routine, may help diagnose infiltrative disease or myocarditis
- ❑ angiography: selected patients - if cardiac risk factors to r/o CAD

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)
- ❑ anticoagulation to prevent thromboembolism
  - absolute - AF, 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 therapy
  - volume reduction surgery (role remains unclear)
  - cardiomyoplasty (latissimus dorsi wrap)
  - LVAD

HYPERTROPHIC CARDIOMYOPATHY

- ❑ also known as hypertrophic obstructive cardiomyopathy (HOCM) and idiopathic hypertrophic subaortic stenosis (IHSS)
- ❑ issues are obstruction, arrhythmia, diastolic dysfunction

Pathophysiology
- ❑ symmetrical or asymmetrical hypertrophy of the myocardium either:
- ❑ non-obstructive
  - symptoms secondary to decreased compliance and impaired diastolic filling
- ❑ obstructive (latent [brought on by provocative testing] or resting)
  - symptoms secondary to dynamic ventricular outflow obstruction diminishing cardiac output
- ❑ clinical manifestations
  - asymptomatic
  - dyspnea (90%) - secondary to diastolic dysfunction
  - cardiac ischemia
  - presyncope, syncope - obstruction or arrhythmic
  - CHF
  - arrhythmias
  - sudden death (may be first manifestation)

Hallmark Signs of HCM
- ❑ pulses
  - rapid upstroke pulse
  - bifid or bisferiens pulse
- ❑ precordial palpation
  - localized, sustained, double/triple impulse apex beat

- ❑ precordial auscultation
  - normal or paradoxical S_2 (if severe obstruction)
  - S_4
  - harsh, systolic, diamond-shaped murmur at LLSB or apex
- ❑ +/– murmur of MR
  - manouvers (see table below)

Factors Influencing Obstruction

- ❑ these include any factors that
  - increase ventricular contractility
  - decrease preload
  - decrease afterload

Table 11. Factors Influencing Obstruction in Hypertrophic Cardiomyopathy

Increased Obstruction (increase murmur)	Decreased Obstruction (decrease murmur)
inotropes, vasodilators, diuretics	negative inotropes
hypovolemia	vasoconstrictors
tachycardia	volume expansion
standing	bradycardia
valsalva maneuvre	squatting
	sustained handgrip

Investigations

- ❑ 12 lead ECG
  - LVH
  - Q waves in anterolateral and inferior leads
- ❑ echocardiography
  - LVH - concentric or asymmetric septal hypertrophy
  - systolic anterior motion of anterior MV leaflet (SAM)
  - resting or dynamic ventricular outflow tract obstruction
  - diastolic dysfunction
  - +/– MR
  - LAE
- ❑ cardiac catheterization
  - increased left ventricular end-diastolic pressure
  - variable systolic gradient across LV outflow tract

Natural History

- ❑ variable; some improve and stabilize over time while others suffer from some of the complications
- ❑ AF, IE (< 10%), LV failure (10-15%), sudden death (cause of 50% of all mortality from HCM)
- ❑ risk factors for sudden death
  - most reliable
    - young age < 30 at diagnosis
    - family history
    - genetic abnormalities associated with an increased risk
  - less clear
    - syncope (ominous in children, less so in adults)
    - ventricular tachycardia on ambulatory monitoring
    - marked ventricular hypertrophy
  - prevention of sudden death in high risk patients = amiodarone or ICD

Management

- ❑ supportive care
- ❑ avoid factors which increase obstruction
- ❑ avoid strenuous exercise (guidelines exist)
- ❑ treat arrhythmias
- ❑ IE prophylaxis
- ❑ obstruction
  - beta-blockers, verapamil, or diltiazem (caution if large outflow gradient or very high pulmonary pressure) (NOTE: these therapies do NOT appear to affect sudden death)

- ❑ consider surgical options (myotomy - myectomy, MV replacement)
- ❑ dual chamber pacing - to decrease obstruction
- ❑ arrhythmias - amiodarone +/– ICD

RESTRICTIVE CARDIOMYOPATHY

Etiology

- ❑ infiltrative
  - amyloidosis/sarcoidosis
- ❑ non-infiltrative
  - scleroderma, idiopathic myocardial fibrosis
- ❑ Storage diseases
  - hemochromatosis, Fabry's disease
- ❑ endomyocardial
  - endomyocardial fibrosis
- ❑ Loeffler's endocarditis or eosinophilic endomyocardial disease
- ❑ radiation heart disease

Pathophysiology

- ❑ infiltration of the myocardium —> decreased ventricular compliance —> diastolic dysfunction
- ❑ clinical manifestations
  - CHF - diastolic dysfunction predominates
  - arrhythmias
  - systemic and pulmonary embolism

Investigations

- ❑ 12 lead ECG
  - low voltage
  - non-specific ST-T wave changes
- ❑ chest x-ray
  - mild cardiac enlargement
- ❑ echocardiography
  - normal or only slightly decreased systolic function, diastolic dysfunction
- ❑ cardiac catheterization
  - elevated end-diastolic ventricular pressures

Natural History

- ❑ depends on etiology
- ❑ generally poor prognosis: most die within a few years, usually due to severe CHF

Management

- ❑ exclude constrictive pericarditis
- ❑ treat underlying disease
- ❑ supportive care
- ❑ treat CHF
- ❑ treat 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
  - fungi
  - spirochetal
  - Lyme carditis
  - Chagas disease, toxoplasmosis
- ❑ acute rheumatic fever
- ❑ drug-induced: emetine, doxorubicin
- ❑ collagen vascular disease
- ❑ sarcoidosis

Clinical Manifestations
- ❑ constitutional illness
- ❑ acute CHF
- ❑ chest pain - associated pericarditis or cardiac ischemia
- ❑ arrhythmias
- ❑ systemic or pulmonary emboli
- ❑ sudden death

Investigations
- ❑ 12 lead ECG
  - non-specific ST-T changes +/– conduction defects
- ❑ blood work
  - increased CK, LDH, and AST with acute myocardial necrosis +/– increased WBC, ESR
- ❑ perform blood culture, viral titres 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

INFECTIVE ENDOCARDITIS

Etiology
- ❑ *Streptococcus viridans* (commonest)
- ❑ *Enterococcus*
- ❑ S. *aureus* (IV drug abusers, catheter-associated sepsis)
- ❑ *Staphylococcus epidermidis* (prosthetic valve)
- ❑ *Strep bovis*
  - underlying GI malignancy
- ❑ others: gram-negative bacteria, Candida, Hacek organisms
- ❑ frequency of valve involvement: MV >> AoV > 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/immunocompromise
- ❑ 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 —> endocarditis

- ❑ symptoms
  - fever, chills, rigors
  - night sweats
  - 'flu-like' illness, malaise, H/A, myalgia, arthralgia
  - dyspnea, chest pain

Signs
- ❑ classic triad = fever, murmur (new or changing), anemia
- ❑ signs of HF
- ❑ petechiae, retinal Roth spots, 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 (also seen with local trauma)
- ❑ focal neurological signs (CNS emboli)
- ❑ arthritis
- ❑ clubbing (subacute)
- ❑ splenomegaly (subacute)
- ❑ microscopic hematuria (renal emboli or glomerulonephritis)
- ❑ weight loss

Investigations
- ❑ blood work - anemia, increased ESR, positive rheumatoid factor
- ❑ serial blood cultures (definitive diagnosis)
- ❑ echocardiography (transesophageal > sensitivity than transthoracic)
  - vegetations, valve leaflet rupture, chordal rupture, abscess
  - serial ECHO may help in assessing cardiac function
  - persistence or disappearance of vegetations is not a reliable indication of success or failure

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
  - 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, valvular obstruction, unstable prosthesis, multiple major emboli, antimicrobial failure, splenic abscess, mycotic aneurysm

RHEUMATIC FEVER

- ❑ Jones' criteria for diagnosis: 2 major, or 1 major + 2 minor
  - major criteria
    - carditis
    - polyarthritis
    - Sydenham's chorea
    - erythema marginatum
    - subcutaneous nodules

- minor criteria
  - previous history of rheumatic fever or rheumatic heart disease
  - polyarthralgia
  - increased ESR or CRP
  - increased PR interval
  - fever
- confirmation of streptococcal infection: history of scarlet fever, group A streptococcal pharyngitis culture, ↑ anti-streptolysin O Titers

❑ management: bed rest, ASA, benzathine penicillin G 1.2 MU IM
❑ prophylaxis (age < 40): benzathine penicillin G 1.2 MU IM monthly

AORTIC STENOSIS

Etiology

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

Pathophysiology and Symptomatology

❑ AS = narrowed valve orifice (aortic valve area: normal = 3-4 cm^2; severe AS (usually symptomatic) = < 1.0 cm^2; critical AS = < 0.75 cm^2 or pressure gradient > 50 mmHg)
❑ small orifice —> outflow obstruction —> fixed output —> forward failure
- symptoms
  - syncope (especially with heavy exertion)
  - fatigue

❑ small orifice —> pressure overload —> concentric LVH (fibers in parallel) —> ↑ LVEDP
- symptoms
  - dyspnea (initially exertional)
  - PND/orthopnea
  - peripheral edema + CHF (10% develop RV failure)

❑ ↑LVEDP —> ↓ subendocardial flow and ↑ myocardial O_2 demand
- symptoms
  - angina
  - palpitations

❑ TRIAD: syncope, CHF, angina

Signs of AS

❑ pulses
- apical-carotid delay
- pulsus parvus et tardus (slow upstroke and late peaking)
- brachio-radial delay
- thrill over carotid and suprasternal notch

❑ precordial palpation
- sustained +/– diffuse apex beat
- +/– palpable S_4
- systolic thrill in 2nd RICS +/– along LLSB

❑ precordial auscultation
- SEM - diamond shaped (crescendo-decrescendo), peaks progressively later in systole with worsening AS, intensity not related to severity, radiates to neck, musical quality of murmur at apex (Gallavardin effect)
- +/– diastolic murmur of associated mild AR
- S_2 - paradoxical splitting (severe AS), or single (A2 absent)
- ejection click (more common in mild AS, absent if severe)
- S_3 - late in disease (if LV dilatation present)
- S_4 - early in disease (decreased LV compliance)

Investigations

❑ 12 lead ECG
- LVH and strain +/– LBBB, LAE/AF

❑ chest x-ray
- post-stenotic aortic root dilatation, calcified valve, LVH + LAE, CHF (develops later)

❑ echocardiography
- gold standard for diagnosis
- 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 syncope; 3 years after onset of angina; 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/vasodilators in severe AS
  - treat CHF (see CHF Section)

❑ indications for surgery
- onset of symptoms: angina, syncope, or CHF
- progression of LV dysfunction
- AoV area < 0.8 cm^2 associated with symptoms
- moderate AS if other cardiac surgery (i.e. CABG) required

❑ surgical options
- open or balloon valvuloplasty
  - children, repair possible if minimal disease
  - adults (rare): pregnancy, palliative in patients with comorbidity, or to stabilize patient awaiting AV replacement - 50% recurrence of AS in 6 months
- aortic valve replacement
  - excellent long-term results, procedure of choice
- complications: low CO, bleeding, conduction block, stroke

AORTIC REGURGITATION

Etiology
❑ supravalvular (aortic root disease with dilatation of ascending aorta)
- atherosclerotic dilatation and aneurysm; cystic medial necrosis (Marfan's syndrome); dissecting aortic aneurysm; systemic hypertension; syphilis; connective tissue diseases (ankylosing spondylitis, psoriatic arthritis, Reiter's syndrome, rheumatoid aortitis, etc…)

❑ valvular
- congenital abnormalities (bicuspid AoV, large VSD); connective tissue diseases (lupus, ankylosing spondylitis, rheumatoid arthritis, etc…); rheumatic fever (+/– associated AS); IE; myxomatous degeneration; deterioration of prosthetic valve

❑ acute AR
- IE
- aortic dissection
- acute rheumatic fever
- failed prosthetic valve

Pathophysiology and Symptomatology
❑ AR = blood flow from aorta into LV (diastolic run-off)
❑ volume overload —> LV dilatation —> ↑ SV and more diastolic run-off —> high SBP and low DBP (wide pulse pressure)
❑ LV dilatation combined with↑ SBP —> ↑ wall tension = pressure overload —> LVH
- symptoms
  - dyspnea/orthopnea/PND
  - fatigue and palpitations (arrhythmias or hyperdynamic circulation)

- ❑ ↓DBP —> ↓ coronary perfusion; LVH —> ↑ myocardial O_2 demand
  - symptoms
    - syncope, angina (only if severe AR)
- ❑ usually symptomatic only after onset of LV failure

Signs of chronic AR

- ❑ pulses
  - increased volume (bounding/collapsing)
  - de Musset's sign - head bobbing due to ↑PP
  - pistol-shot sounds over femoral artery (without compression)
  - Duroziez's murmur - to-and-fro murmur over femoral artery with light compression
  - Traube's sign - double sound heard with the stethoscope lightly applied over the artery
  - Quincke's sign - pulsatile blushing of nail beds (nonspecific)
  - water-hammer pulse - strong but rapidly collapsing pulse
  - Corrigan's pulse - visible carotid pulse
  - Hill's sign - femoral-brachial SBP difference > 20 (greater differences correlate with more severe AR)
  - Bisferiens pulse - twice beating in systole; especially if AS also present
  - other - pulsating uvula (Muller), liver (Rosenbach), pupil (Gandolfi), or spleen (Gerhardt)
- ❑ precordial palpation
  - hyperdynamic, displaced apex (volume overload)
- ❑ precordial auscultation
  - S_1 - soft in severe AR (early closure of MV)
  - S_2 - loud, or soft (severe AR or with calcification of valve)
  - S_3 in severe AR (early LV decompensation)
  - diastolic decrescendo murmur - high-pitched, at LLSB (cusp disease) or RLSB (aortic root disease), length correlates with severity, best heard with patient leaning forward
  - SEM - in aortic area, secondary to increased flow
  - 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 S_2, short early diastolic murmur

Investigations

- ❑ 12 lead ECG
  - LVH, LAE (p-mitrale)
- ❑ chest x-ray
  - LV enlargement, LAE, aortic root dilatation
- ❑ echocardiography
  - gold standard for diagnosis and assessment of severity of AR
  - regurgitant jet from aorta into LV; dilated LV, aortic outlet, and LA
  - LV volume overload
  - fluttering of anterior MV leaflet
  - Doppler most sensitive
- ❑ radionuclide imaging
  - serial resting and exercise EF (normal ↑ with exercise > 5%)
  - sensitive sign of ↓ LV function: failure to ↑ EF with exercise
- ❑ cardiac catheterization
  - coronary angiography indicated if age > 40
  - ↑ LV volume; CO normal or depressed (LV dysfunction); ↑ LVEDP

Natural History

- ❑ mild to moderate AR - few symptoms
- ❑ chronic progression to severe AR may be asymptomatic up to 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 delays need for surgery
  - IE prophylaxis
- ❑ medical
  - restriction of activities
  - treat CHF (non-pharmacologic, 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, even if asymptomatic
  - consider if poor LVEF (< 55%) at rest, or failure to increase EF with exercise (with serial MUGA assessment)
- ❑ surgical options
  - valve repair (rare in AR)
    - subcommissural annuloplasty for annular dilatation
  - aortic valve replacement
    - heterograft, homograft, or sometimes pulmonary autograft (Ross procedure) valve may be used

MITRAL STENOSIS

Etiology
- ❑ congenital (rare)
- ❑ acquired
  - RHD (most common) (especially developing nations; F > M)
  - other: atrial myxoma, atrial or valvular thrombus, etc…

Pathophysiology and Symptomatology
- ❑ normal MV area = 4-6 cm^2
- ❑ MS = LV inlet obstruction —> LAE —>↑ LA pressure —> ↑ PVR —> ↑ right-sided pressure —> RVH and 2º TR —> right-sided CHF
  - symptoms
    - dyspnea (exertional, ↑ HR —> ↓ diastolic filling time —> ↑ LA pressure and pulmonary congestion))
    - orthopnea/PND (↑ venous return —> ↑ LA pressure and diastolic PAP (pulmonary congestion)
    - cough, hoarseness, hemoptysis
    - palpitations (AF 2º to LAE)
- ❑ LV inlet obstruction —> fixed CO
  - symptoms
    - fatigue
    - low exercise tolerance
- ❑ atrial kick crucial - CO may ↓ with AF (loss of atrial kick), pregnancy, or tachycardia (shortened diastolic filling period)

Signs of MS
- ❑ general examination
  - mitral facies, peripheral coldness and cyanosis
  - hepatic enlargement/pulsation, ascites, peripheral edema (all 2º to TR and RV failure)
- ❑ pulse
  - +/– irregularly irregular (AF), may be small volume
- ❑ JVP
  - +/– loss of “a” waves (AF), elevated (RV failure), or large “v” waves (TR)
- ❑ precordial palpation
  - apex - inconspicuous LV
  - palpable S_1
  - palpable P_2 (in severe MS)
  - left parasternal lift (RV)
- ❑ precordial auscultation
  - loud S_1 (lost if heavily calcified and not pliable)
  - opening snap (lost if heavily calcified and not pliable)

- mid-diastolic rumble - at apex, heard better in LLDB position and post-exercise, a longer murmur and a shorter A_2-OS duration correlate with worse MS (increased LAP)
- presystolic accentuation (lost with AF)
- if pulmonary hypertension present - loud P_2, pulmonary regurgitation (Graham Steell murmur)

❑ chest examination
- crackles (pulmonary congestion)

Investigations

❑ 12 lead ECG
- normal sinus rhythm/AF, LAE, RVH

❑ chest x-ray
- LA enlargement (LA appendage, double contour, splaying of carina), pulmonary congestion, MV calcification

❑ echocardiography
- gold standard
- thickened calcified valve, fusion of leaflets, LAE
- Doppler can estimate valvular area
- decay of gradient to assess severity

❑ cardiac catheterization
- concurrent CAD in patients if age > 35

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 AF: 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 AF (rate control, cardioversion)
- anticoagulation - if AF, previous embolus, or LAE > 50 mm
- IE prophylaxis
- diuretics and rate control - if mild symptoms, and high risk surgical candidate

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

❑ surgical options
- closed commisurotomy
  - rarely performed in North America
- balloon valvuloplasty
  - if high risk patient, fused commisures, and non-calcified valve with intact chordae, minimal MR
- open commisurotomy
  - best procedure if valve amenable to repair
- all the above “turn the clock back” - re-stenosis will develop
- mitral valve replacement
  - if immobile leaflets/heavy calcification, severe subvalvular disease, MR

MITRAL REGURGITATION

Etiology

- ❑ annulus
  - 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; myxomatous degeneration; IE; acute MI
- ❑ papillary muscles and LV wall
  - ischemia/infarction; aneurysm; HCM

Pathophysiology and Symptomatology

- ❑ chronic MR = gradually increasing flow across MV during systole —> progressive LAE —> ↓ fraction of SV flows forward —> LV dilatation (to maintain CO) —> ↑ LV wall tension —> LVH —> CHF (↓ CO, pulmonary edema)
  - 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 (LVH)
  - because of LV dilatation, "MR begets MR" was coined
- ❑ acute MR = sudden onset of MV incompetence —> ↑ LA pressure —> ↑ PAP —> pulmonary edema —> RV failure (acute onset CHF)

Signs of MR

- ❑ pulse
  - quick and vigorous (unless LV failure)
- ❑ precordial palpation
  - apex - displaced, hyperdynamic, enlarged
  - +/– 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 2º to MVP - usually mid-systolic
    - papillary muscle dysfunction - typically a late systolic whoop or honk
  - mid-diastolic rumble - increased flow across valve (often no MS)
  - severity - gauge by LV dilatation, S_3, diastolic flow rumble
  - opening snap = associated MS, but does not preclude predominant MR
- ❑ AF, CHF, pulmonary hypertension 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), LVH (50% of patients)
- ❑ chest x-ray
  - LVH, LAE, pulmonary venous hypertension
- ❑ echocardiography
  - etiology - flail leaflets, vegetations, etc…
  - severity – regurgitant volume/fraction/orifice area
  - LV function - increased LV/LA size, LVED volume; EF
  - colour flow mapping shows abnormal jet from LV to LA
- ❑ cardiac catheterization
  - assess coronary arteries
  - ventriculography - contrast fills LA
  - prominent left atrial "v" wave on Swan-Ganz

Management

- ❑ medical
  - asymptomatic - serial echocardiograms

- IE prophylaxis
- symptomatic - ↓ preload (diuresis) and ↓ 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 left ventricular dysfunction or increased LV volume or size, even if asymptomatic

❑ surgical options
- valve repair
  - preferred (low mortality), often technically difficult
- mitral valve replacement
  - if unable to repair MV
  - straight forward technique, attempt to conserve chordal structures/connections, complete correction of MR achieved, good prognosis unless age > 75

MITRAL VALVE PROLAPSE
(Barlow's Syndrome)

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)
❑ associated with low weight and BP, and pectus excavatum

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
❑ mid-systolic click (tensing of redundant valve tissue)
❑ mid to late systolic murmur or pansystolic murmur (regurgitation after prolapse)
❑ maneuvers to change LV volume – squat to stand, or Valsalva —> decreased ventricular filling —> earlier click and louder/longer murmur

Investigations
❑ 12 lead ECG
- nonspecific ST-T wave changes, PSVT, ventricular ectopy

❑ echocardiography
- posterior systolic prolapse of MV leaflets
- 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

❑ beta-blockers - for palpitations, pain, anxiety
❑ anticoagulation - if systemic embolism
❑ for MR - IE prophylaxis, consider early MV repair for severe MR, standard indications for MV replacement

TRICUSPID VALVE DISEASE

Etiology
- ❑ TS: rheumatic, congenital, carcinoid syndrome, RA tumours, fibroelastosis
- ❑ TR: RV dilatation (commonest cause), IE (IV drug users), rheumatic, Ebstein's anomaly, AV cushion defects, carcinoid, tricuspid prolpase, 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 AF and low volume
- ❑ JVP
  - elevated pressure
  - prominent "a" waves in TS
  - large "v" waves in TR ("CV" waves)
  - positive hepatojugular reflux and Kussmaul's sign
- ❑ precordial palpation for left parasternal lift (RV) in TR
- ❑ precordial auscultation
  - note: all right sided sounds are louder with inspiration ("Carvallo's sign"), except a pulmonary ejection click
  - TS: diastolic rumble in 4th LICS
  - TR: holosystolic murmur along LLSB ± thrill (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: secondary to fluid retention

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

Management
- ❑ TS: usually determined by the more severely stenotic MV
- ❑ TR: in treating RV failure, also treat LV failure, MS, or MR
  - note: commonest cause of RV failure is LV failure

PULMONARY VALVE DISEASE
- ❑ very rarely of clinical importance

Etiology
- ❑ PS: usually congenital; rheumatic uncommon; carcinoid
- ❑ PR: secondary to dilatation of valve ring
  - pulmonary hypertension (MS (most common), chronic lung disease, recurrent PE)
  - inflammatory (rheumatic, IE, tuberculosis)

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

Signs
- ❑ PS
  - systolic murmur - maximum at 2nd LICS
  - pulmonary ejection click; normal/loud/soft P_2; right sided S_4
- ❑ PR: associated with pulmonary hypertension
  - early diastolic murmur at base - AR until proven otherwise
  - Graham Steell (diastolic) murmur at 2nd and 3rd LICS without peripheral signs of AR

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

Management
- ❑ IE prophylaxis
- ❑ PR
  - rarely requires treatment (well tolerated if PVR 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), 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
- ❑ infectious
  - viral: Coxsackie virus A, B (most common)
  - bacterial: endocarditis, septicemia
  - TB
  - fungal: histoplasmosis, blastomycosis
  - protozoal
- ❑ myocardial infarction: acute (1-7 days), post MI (Dressler's syndrome) (2-8 weeks)
- ❑ post-pericardiotomy (e.g. CABG)
- ❑ collagen vascular disease: SLE, periarteritis, RA, scleroderma
- ❑ metabolic: uremia, hypothyroidism
- ❑ vascular: dissecting aneurysm
- ❑ neoplasm: Hodgkin's, breast, lung, renal cell carcinoma, melanoma
- ❑ infiltrative disease, drugs (e.g. hydralazine), trauma, radiation
- ❑ idiopathic (? viral)

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 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

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

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

PERICARDIAL EFFUSION

Etiology
- ❑ two types of effusions:
    - transudative (serous)
        - CHF, hypoalbuminemia/hypoproteinemia
    - exudative (serosanguinous or bloody)
        - causes similar to the causes of acute pericarditis
- ❑ physiological consequences depend on type and volume of effusion, rate of effusion development, and underlying cardiac disease

Symptoms
- ❑ nil or similar to acute pericarditis
- ❑ dyspnea, cough
- ❑ extra-cardiac (esophageal/recurrent laryngeal nerve/ tracheo-bronchial/phrenic nerve irritation)

Signs
- ❑ JVP: elevated with dominant "x" descent
- ❑ arterial pulse: normal to ↓ volume, ↓ PP
- ❑ pulsus paradoxus (drop of SBP > 10 mm Hg on inspiration)
- ❑ apex normal or absent
- ❑ auscultation: distant heart sounds +/– rub

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

Management
- ❑ mild: frequent observation with serial ECHO, anti-inflammatory agents for inflammation
- ❑ severe: may develop cardiac tamponade
- ❑ if hemodynamic compromise, pericardiocentesis or open drainage
- ❑ medical: treat the cause, therapeutic pericardiocentesis
- ❑ surgical: pericardial window, pericardiectomy

CARDIAC TAMPONADE
- ❑ major complication of pericardial effusion

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)
- ❑ echocardiography: pericardial effusion, diastolic compression of cardiac chambers (RA and RV)
- ❑ cardiac catheterization: mean RA, LA, LV and RV diastolic pressures all high and equal

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

CONSTRICTIVE PERICARDITIS

Etiology
- ❑ any cause of acute pericarditis may result in chronic pericarditis

Symptoms
- ❑ dyspnea, fatigue, palpitations
- ❑ abdominal pain

Signs
- ❑ general examination - mimics CHF (especially right-sided HF)
  - ascites, hepatosplenomegaly, edema
- ❑ pulses: ↑ JVP, Kussmaul's sign (paradoxical ↑ 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, +/– AF
- ❑ chest x-ray: pericardial calcification, effusions
- ❑ CT or MRI: 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 12. 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

Definition

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

Etiology

- ❑ 50% of cases are never diagnosed
- ❑ cardiac
  - electrical
    - tachycardia: VT, Torsades de pointes, SVT
    - bradycardia: SSS, 2º or 3º AV block
    - pacemaker failure
  - mechanical
    - outflow obstruction: LV (AS, HOCM, MS, LA myxoma), RV (PS, PE, pulmonary hypertension)
    - 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 (1º: Shy-Drager, 2º: DM), hypovolemia (blood loss, diuresis pheochromocytoma)
  - neurological: vertebrobasilar TIA/stroke, SAH, 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)

Table 13. Differentiation of Seizure vs. Syncope

Characteristic	Syncope	Seizure
facial color	pale	cyanotic
(lateral) tongue biting	rare	common
aura	no	sometimes
nausea, diaphoresis	common before	uncommon
LOC	brief	may be longer
reorientation	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, serum electrolytes, Mg, Ca, BUN, creatinine, glucose, ABG, CK-MB
- ❑ ECG
- ❑ ECHO
- ❑ carotid Doppler US
- ❑ Holter monitor, loop Holter
- ❑ tilt-table testing
- ❑ EPS

Management

- ❑ treatment of underlying cause

Table 14. Commonly Used Cardiac Therapeutics

DRUG CLASS	EXAMPLES	MECHANSIM OF ACTION	INDICATIONS	SIDE EFFECTS	CONTRA-INDICATIONS
BETA-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- arrhythmia)	• Lowers myocardial O_2 demand by decreased HR, BP and contractility	• ischemic heart disease • hypertension • atrial fibrillation • 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	diltiazem	see Table 15	• hypertension • 2nd line agent for IHD (1st line beta-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	• hypertension • 2nd line agent for IHD (1st line beta-blockers) • SVT • diastolic dysfunction	• bradycardia • CHF • constipation	• sick sinus syndrome • second or third degree AV block • severe CHF • AMI (relative) • pregnancy (relative) • atrial fibrillation with bypass tract with anterograde conduction
	nifedipine	see Table 15	• hypertension	• 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 beta-blocker
ACE INHIBITORS	captopril enalapril ramipril	• peripheral vasodilator —> afterload reduction with little change in CO, HR or GFR • also cause decrease in fluid volume due to inhibition of aldosterone production	• CHF (including post-MI) • hypertension • 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 2º to ACEI
ANGIOTENSIN II BLOCKER	losartan (cozaar)	• blocks angiotensin II receptor so peripherally vasodilates and blocks aldosterone effects	• CHF • hypertension	• dizziness (< 2%) • hypotension/syncope • renal dysfunction	• bilateral renal artery stenosis • pregnancy

DRUG CLASS	EXAMPLES	MECHANSIM OF ACTION	INDICATIONS	SIDE EFFECTS	CONTRA-INDICATIONS
FUROSEMIDE		• loop diuretic • interferes with creation of hypertonic medullary intersitium • 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 hypokalemia • severe hypovolemia • severe hypotension • hypersensitivity to furosemide or sulfonamide • pregnancy
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
DIGOXIN		• positive inotrope- increases force and velocity of myocardial contraction • blocks AV node (decreased refractory period and conduction time) and depresses SA node	• atrial fibrillation • CHF	• cardiac toxicity • AV blocks (e.g. Wenkebach, atrial tachycardia with block) • tachycardias (eg ventricular tachycardia, atrioventricular dissociation, accelerated junctional rhythm) • bradyarrhythmias (e.g. sinus bradycardia, sinus arrest, sinoatrial block) • regularization of R-R interval in AF • 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
ASA		• cyclooxygenase inhibitor • interferes with platelet aggregation by impairing production of thromboxane A2	• AMI • 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 15. Beta-Blocker Actions				
Clinical Effects	Propranolol	Atenolol	Acebutolol	Labetalol
ß-Activity	non-selective	ß1	ß1	non-selective
α-Activity	N	N	N	α1
ISA	N	N	+++	+
brochoconstriction	+++	+	+	++
orthostatic hypotension	–	–	–	+++
lipid adverse effects	++	++	–	+
CNS adverse effects	+++	+	++	++

Carvedilol (α_1-and non-selective ß-blockade)
- ❑ useful in functional class II-III CHF (65% reduction in mortality)
- ❑ antioxidant

CALCIUM CHANNEL BLOCKERS

- ❑ 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 16. Calcium Channel Blocker Actions			
Clinical Effects	Diltiazem	Verapamil	Nifedipine
coronary vasodilator	++	++	+++
peripheral vasodilator	+	++	+++
contractility	<—>	decr	<—>
sinus rate	decr	decr	incr
AV conduction	decr	decr	<—>

ANTI-ARRHYTHMIC DRUGS

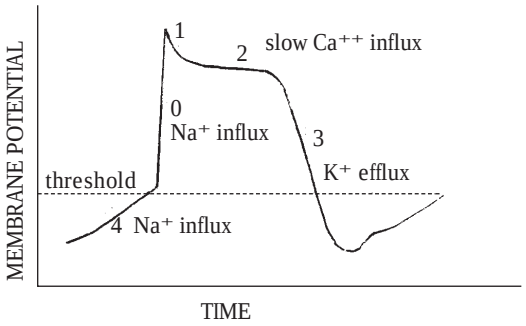


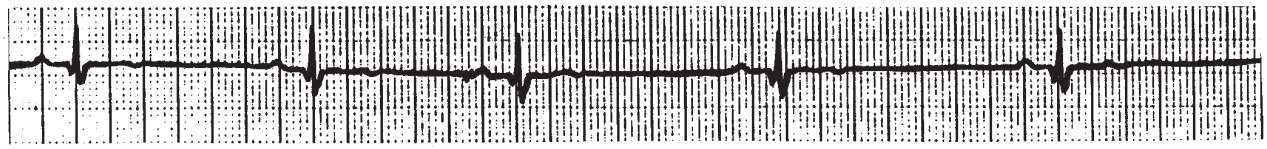
Figure 12. Representative Action Potential

Table 17. Antiarrhythmic drugs

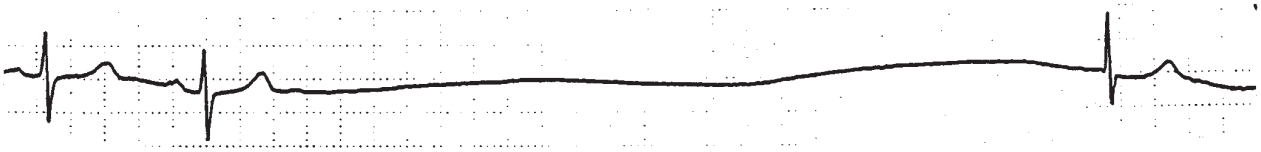
Class	Agent	Indications	Side Effects	Mechanism of Action
Ia	Quinidine Procainamide Disopyramide	SVT, VT	Torsades de Pointes (all Ia) diarrhea lupus-like syndrome anti-cholinergic effects	• moderate Na channel blockade • slows phase O upstroke • prolongs repolarization and thus slows conduction
Ib	Lidocaine Mexiletine	VT	confusion, stupor, seizures GI upset, tremor	• mild Na channel blockade • shortens phase 3 repolarization
Ic	Propafenone Flecainide Encainide	SVT, VT[1] AF[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, AF[1]	bronchospasm, negative inotrophy, bradycardia, AV block, impotence, fatigue	• beta-blockers • decreases phase 4 depolarization
III	Amiodarone (multiple class effects)	SVT, VT AF[2]	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 , AF[1,2] VT	beta-blocker effects, Torsades de Pointes, hypotension	
IV	Verapamil Diltiazem	SVT AF[2]	bradycardia, AV block hypotension	• Ca channel blocker • slow phase 4 spontaneous depolarization and so slows conduction in areas such as AV node

1rate control of atrial fibrillation only
2cardioversionl of atrial fibrillation only

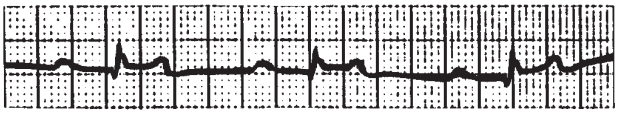
- ❑ All anti-arrhythmics have potential to be pro-arrhythmic
- ❑ In the landmark CAST trial, two class Ic agents (encainide, flecainide) prevented VPB's post MI but significantly increased mortality



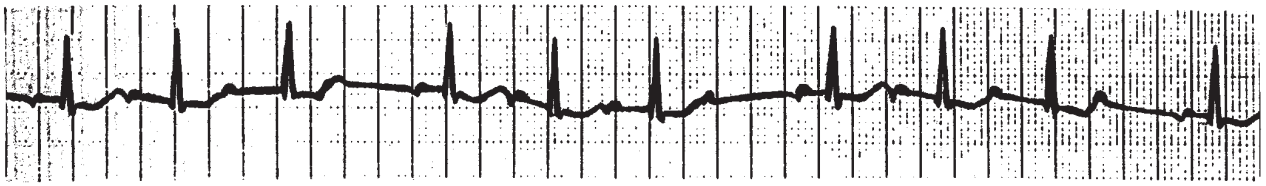
1a) Sinus Bradycardia



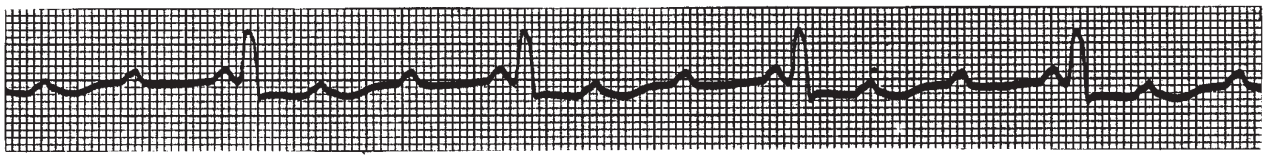
1b) Sinus Arrest



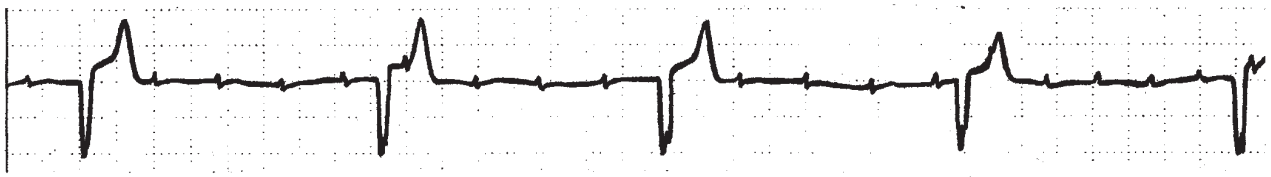
1c) 1° AV Block



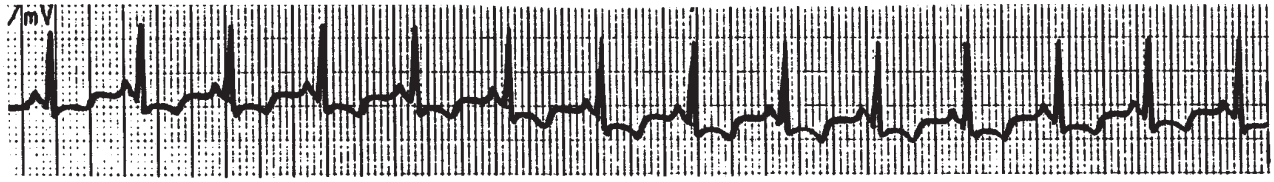
1d) 2° AV Block (Type I or Wenkeback)



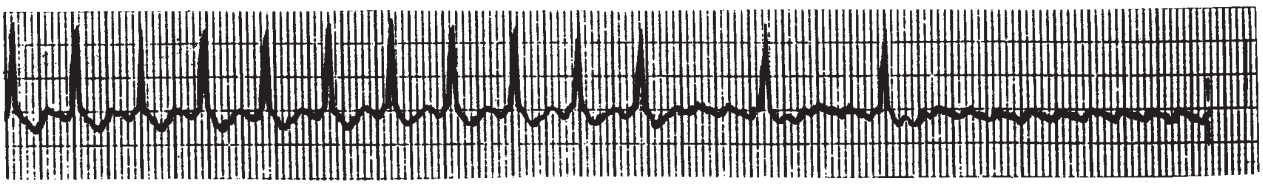
1e) 2° AV Block (Type II)



1f) 3° AV Block

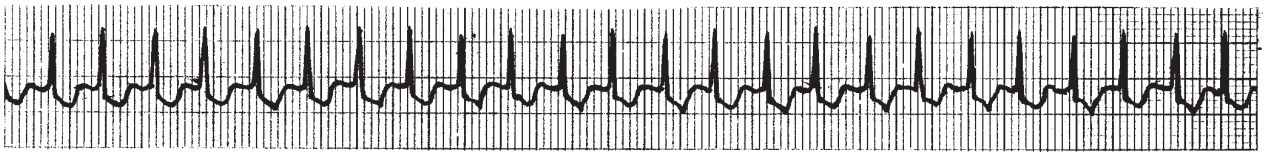


2a) Sinus Tachycardia

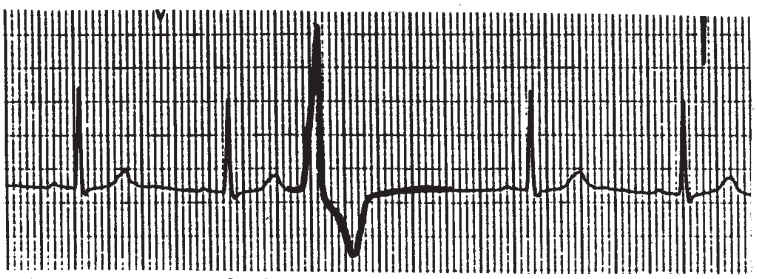


2b) Atrial Flutter (with 2:1 AV block)

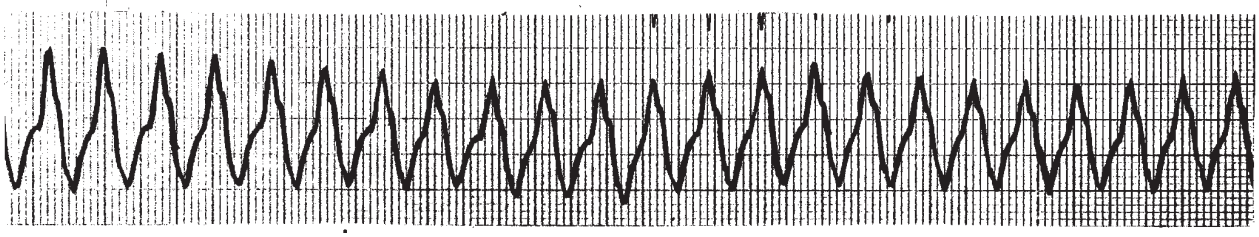
2c) Atrial Fibrillation



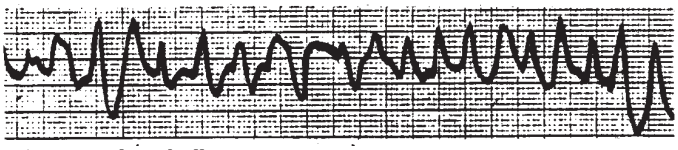
2d) Paroxysmal Supraventricular Tachycardia



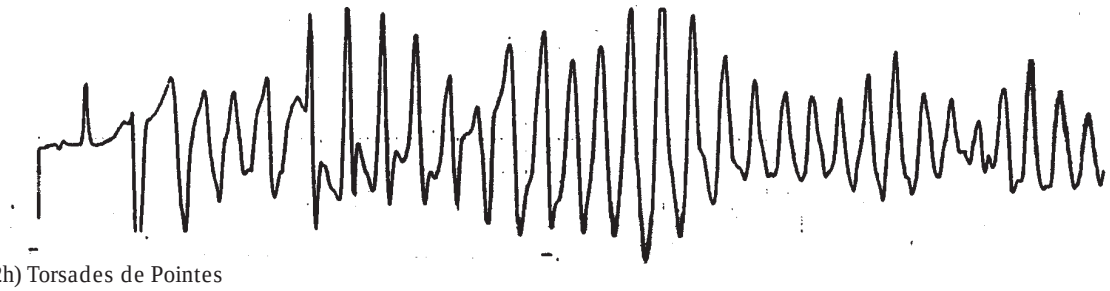
2e) Premature Ventricular Contraction



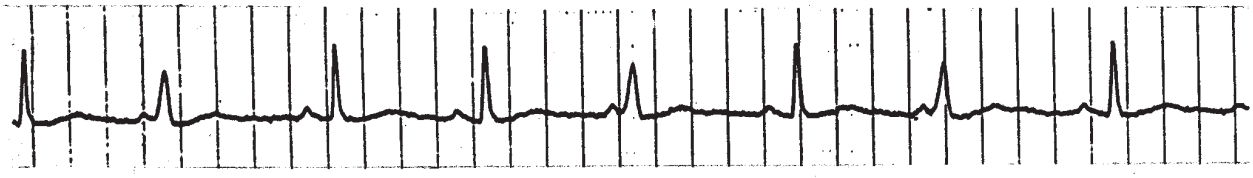
2f) Ventricular Tachycardia



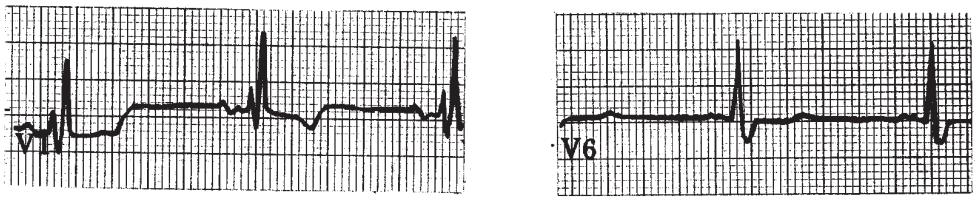
2g) Ventricular Fibrillation



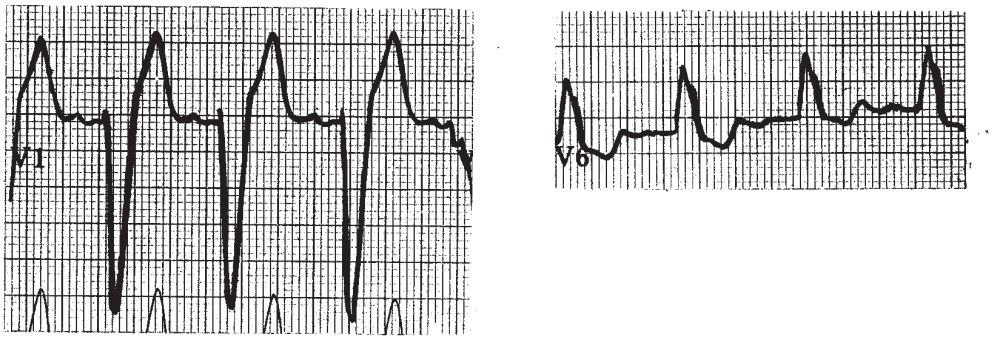
2h) Torsades de Pointes



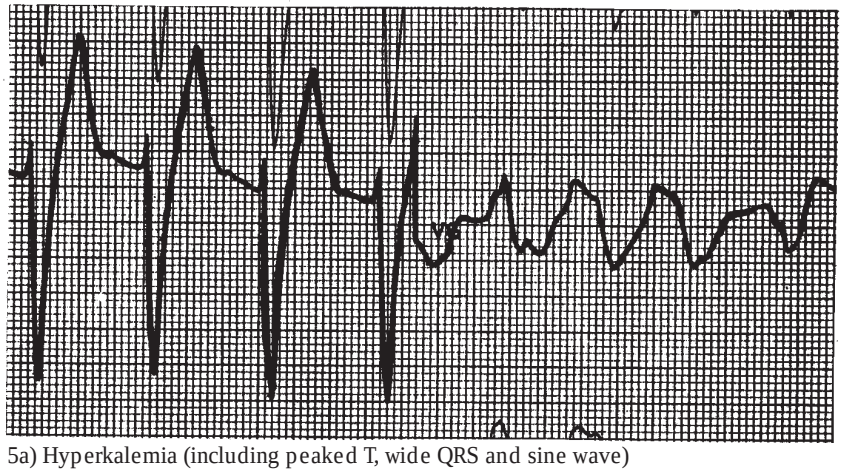
3a) Wolff-Parkinson-White Syndrome (intermittent)



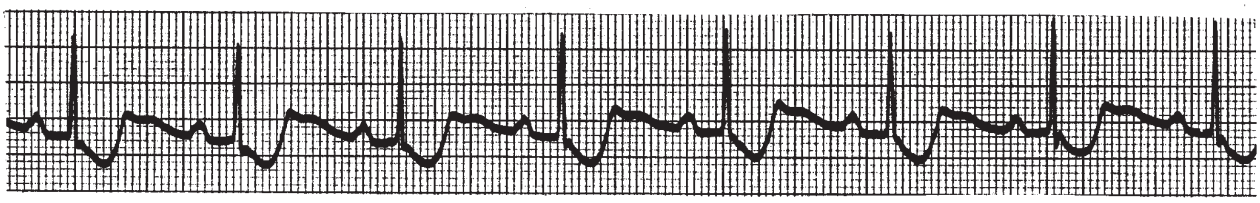
4a) Right Bundle Branch Block



4b) Left Bundle Branch Block



5a) Hyperkalemia (including peaked T, wide QRS and sine wave)



5b) Digitalis Effect (including 1º AV block, scooped ST)