
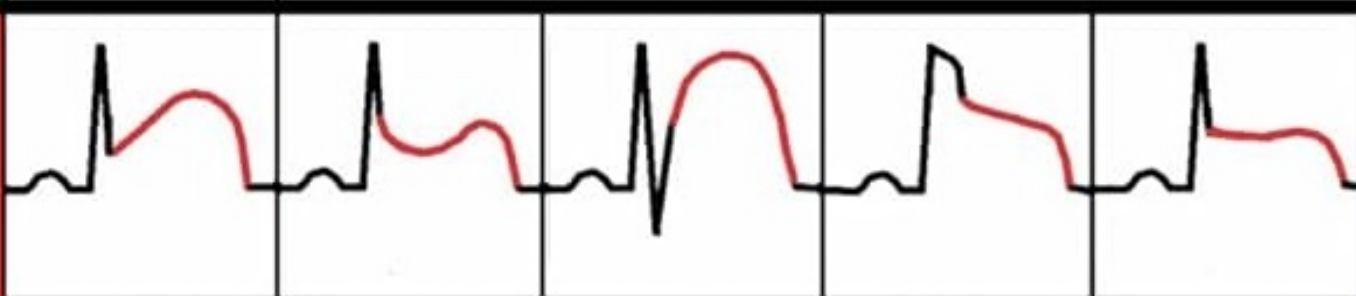
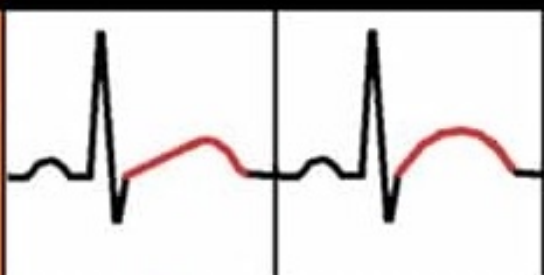
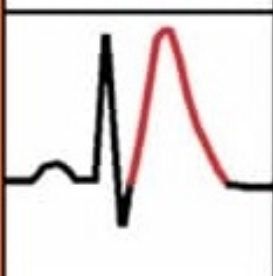
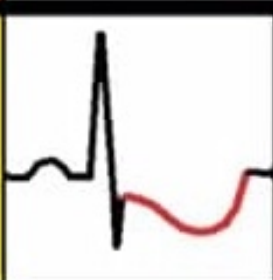


Arrhythmia Review



ECG PATTERNS of MYOCARDIAL PERFUSION *

<p>NORMAL PERFUSION</p>		<ul style="list-style-type: none"> - J POINT ISOELECTRIC - ST SEGMENT: slight POSITIVE inclination - ST-T JUNCTION is CONCAVE - T WAVE smaller than R wave, SMOOTH, ROUNDED (NOT HYPER-ACUTE). T Wave INVERTED in Lead AVR, upright in all other leads.
<p>STEMI</p>		<ul style="list-style-type: none"> - J POINT ELEVATED 1mm or more in 2 or more contiguous leads (except V2 & V3 men can have up to 2mm, women 1.5mm of normal J Point elevation). - ST SEGMENT usually CONVEX, may be UPSLOPING, FLAT, or DOWNSLOPING as seen in the examples above.
<p>PATTERNS CONSISTENT WITH EARLY PHASE ACUTE MI</p>		<p>J POINT to APEX of T WAVE:</p> <ul style="list-style-type: none"> - FLAT (example to far left) or - CONVEX (example to immediate left)
		<p>HYPERACUTE T WAVES</p> <ul style="list-style-type: none"> - J POINT ISOELECTRIC or ELEVATED - PEAK of T Wave POINTED, may exceed amplitude of R Wave - Consider Acute MI / Pending MI - Can ALSO indicate: Transmural Ischemia, Hyperkalemia
<p>ACUTE MI or ISCHEMIA</p>		<p>J POINT DEPRESSION, Downsloping ST Segments, inverted T Waves, consider NSTEMI: check cardiac markers (Troponin):</p> <ul style="list-style-type: none"> - If ST Depression noted in Leads V1-V4 consider Posterior Wall STEMI: obtain Posterior Lead ECG (details on page 7) - If Acute MI not present, rule out MYOCARDIAL ISCHEMIA



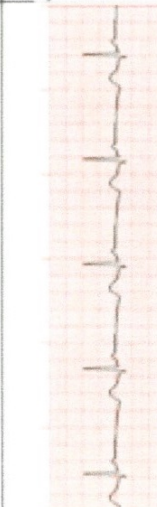
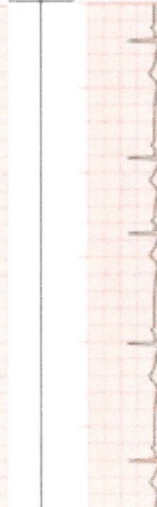
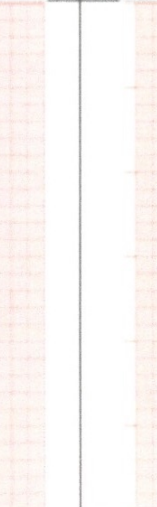
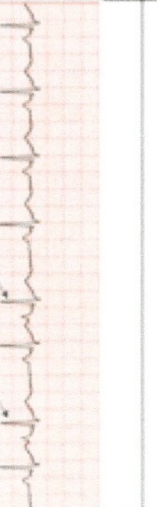
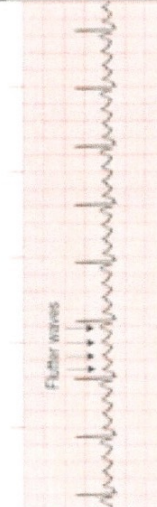
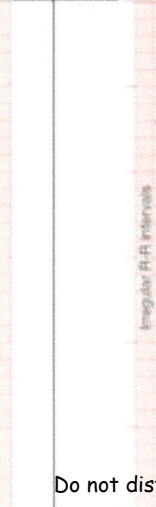
A SUMMARY OF ARRHYTHMIAS

RHYTHM	SIGNIFICANCE	EKG	TREATMENT
SINUS BRADYCARDIA	Low cardiac output Low heart rate	Rate less than 60. PR constant and normal	Atropine Pacemaker
SINUS TACHYCARDIA	Low cardiac output (filling time inadequate)	Rate more than 100. PR constant and normal	Treat underlying problem.
SINUS ARRHYTHMIA	Usually none.	PR constant and normal Variable R-R interval	None
ATRIL FLUTTER	May indicate congestive failure. Cardiac output decreased.	Flutter waves, 300 per minute. Usually 2:1, 3:1, 4:1 or variable A-V conduction.	Digitalis Unstable – cardioversion
ATRIAL FIBRILLATION	Often indicates congestive failure. Cardiac output may fall.	Fibrillation waves, 350-500/min., with variable contour.	Digitalis, Betablockers, Verapamil Unstable = cardioversion
ASYSTOLE	No cardiac output	Straight Line	Confirm another lead, CPR, Epinephrine
VENTRICULAR PREMATURE CONTRACTIONS	May lead to ventricular tachycardia or fibrillation.	QRS premature, broad, “bizarre” and not preceded by premature P.	Lidocaine, Pronestyl.
VENTRICULAR TACHYCARDIA	Rapid decompensation to ventricular fibrillation	QRS broad. Ventricular rhythm nearly regular. P unrelated to QRS. Average rate 180.	Defibrillation Lidocaine
VENTRICULAR FIBRILLATION	No cardiac output. Fatal if untreated.	No well-defined QRS. Irregular undulations.	Defibrillation, CPR, Epinephrine
FIRST DEGREE A-V BLOCK	May progress to higher degree block. May indicate excess Quinidine, Pronestyl or Digitalis.	PR >0.20 sec. or more. Each P conducted	Observe for higher degree block. May need to hold medication.
SECOND DEGREE A-V BLOCK	May progress to complete block.	Some P waves not conducted.	Atropine Pacemaker
THIRD DEGREE A-V BLOCK	Low cardiac output. May produce shock, congestive failure, syncope, ventricular arrhythmias.	P unrelated to QRS. Ventricular rate 20-45. QRS narrow or broad.	Atropine-may or may not help. Pacemaker Dopamine

Complications	Cause/Detection	Prevention/Intervention Recommendations
Simple and tension pneumothoraces	Accidental perforation of lung during passage of pulmonary artery (PA) catheter May be detected on chest x-ray film, analysis of arterial blood gases or patient symptoms of respiratory distress	Careful passage of PA catheter by physician Chest x-ray film is done after PA catheter insertion to evaluate placement of catheter and presence of pneumothorax Higher incidence of pneumothorax or vessel laceration associated when PA catheter placed into subclavian vein
Atrial and ventricular dysrhythmias	Endocardial irritation during passage of PA catheter Transient right bundle branch block (RBBB) can occur as PA catheter passes through the right ventricle (RV) (RV is located superficially in the RV endocardium) Prolonged PA catheterization can cause mechanical damage of the tricuspid and pulmonic valves	Ensure balloon is fully inflated during passage (insertion) of the catheter as well as limiting the time of passage from the right ventricle to pulmonary artery Although temporary, document the occurrence and presence of RBBB Use PA catheter if indicated; remove the PA catheter when no longer clinically indicated
Infection at the insertion site or systemically	PA catheter breaks and invades the skin barrier	Minimize the number of times the catheter-tubing system is entered for blood sampling, cardiac output measurements, and catheter repositioning Wash your hands and wear gloves whenever necessary to enter the system
Systemic air embolism	Balloon rupture or after removal of the PA catheter (through the tissue fibrin tract)	If balloon rupture is suspected, the balloon port is sealed and the PA catheter is removed For care in removing the PA catheter, refer to Table 8
Pulmonary artery perforation and rupture	Risk increased with eccentric or excessive balloon inflation, vigorous flushing with large amounts of fluid, and distal migration of PA catheter	Use recommended balloon inflation volume of 1.25-1.5 mL, avoid flushing distal port of PA catheter, and reposition PA catheter if necessary Remove syringe from balloon port to avoid inadvertent inflation with patient movement Identify patients at risk: pulmonary hypertension, mitral valve dysfunction, receiving anticoagulant therapy
Thrombosis formation	Thrombus formation begins as early as 48 hr on the heparin-bonded PA catheter Damped waveform will appear on oscilloscope	Removal of PA catheter is recommended if thrombus formation is suspected as possible sequelae includes pulmonary embolus, endocardial irritability, and septic phlebitis
Thrombocytopenia	Induced by the heparin-bonded PA catheter and heparin flush solution	Minimize the amount of heparin needed in the flush solution or remove the PA catheter

Troubleshooting the Pulmonary Artery Catheter

Clinical Problem	Implications	Possible Etiology/Cause	Interventions
Migration of the PA catheter into RV	Presence of RV dysrhythmias Decreased diastolic pressure	Accidental or spontaneous withdrawal of catheter into RV	Measures to correct include: Inflate balloon fully to engulf tip of catheter and reduce ectopy. If written protocol allows, or inform physician to, reposition catheter into PA. If dysrhythmias are compromising the patient's hemodynamics allow balloon to passively deflate and withdraw catheter into right atrium (15-20 cm marking on PA catheter).
Inappropriate pressure with proper pressure waveform	Potential for erroneous hemodynamic data and inappropriate treatment measures	Inaccurate reference points – air-fluid interface of stopcock improperly leveled so phlebostatic axis or phlebostatic axis not currently marked on patient's chest Inaccurate calibration Change in patient's condition	Measures to correct include: Open system to air and zero the system, measure phlebostatic axis, level air-fluid interface (stopcock opened to air) to patient's phlebostatic level with a carpenter's level, and calibrate monitor scale or strip chart recorder. Reassess pressure waveforms and data. Consider possible causes
Overwedging	Eccentric balloon inflation is a potential risk for PA perforation and rupture	Catheter migration Small pulmonary vessels	Measures to correct include: Remove syringe to allow passive deflation of balloon and slowly inflate balloon, observing the waveform on the screen. If loss of waveform morphology components and a gradual elevation of waveform off the screen are observed, remove syringe and notify physician to <u>reposition the catheter</u>
Spontaneous wedge	Potential for loss of blood supply to branch of pulmonary vessel and risk of PA infarction	Catheter migration	Measures to correct include: Instruct patient to cough, turn onto side, or straighten arm to dislodge catheter. Notify physician to reposition the catheter

Rhythm	Normal Sinus Rhythm (NSR)	Dysrhythmias originating Sinus Node			Dysrhythmias originating in the Atria			
		Sinus Tachycardia	Sinus Bradycardia	Sinus Arrhythmia	Premature Atrial Complex (PAC)	Atrial Flutter	Atrial Fibrillation (A-fib)	Supraventricular Tachycardia (SVT)
Rate bpm	60 - 100	> 100	< 60	60 - 100 Frequently: ↑ w/inspiration ↓ w/expiration	Depends on rate of underlying rhythm	May be Normal/Tachy	Usually Tachy	> 150
Regularity	Regular	Regular	Regular	Irregular; varies w/respiration	Irregular whenever a PAC occurs	Atria - Regular Ventricles - Reg or Irreg	Irregular	Regular
P wave	Normal/Upright/Rounded	Normal/Upright/Rounded	Normal/Upright/Rounded	Normal/Upright/Rounded	P wave is present: in PAC may have different shape	Sawtooth pattern on P waves. More P waves than QRS	No true P waves; chaotic atrial activity	P waves hidden or not present
P-R interval	0.12 - 0.20 sec	0.12 - 0.20 sec	0.12 - 0.20 sec	0.12 - 0.20 sec	Varies in PAC, otherwise normal	Variable	Absent	Absent
QRS	< 0.12 sec	< 0.12 sec	< 0.12 sec	< 0.12 sec	< 0.12 sec	< 0.12 sec	< 0.12 sec	< 0.12 sec
Drugs	n/a	Treat the underlying cause, i.e. fluid replacement, relief pain, reduce fever, ...	If symptomatic: Atropine Epi 1:10,000	Do NOT require tx unless accompanied by slow heart rate that causes blood flow compromise, if so admin Atropine			Amiodarone	Amiodarone
Clinical Tip	A normal ECG does not exclude heart disease	May be caused by exercise, anxiety, fever, hypoxemia, hypovolemia, or cardiac failure. It is the response to the body's demands for increase O ₂ .	It is normal in athletes and during sleep. In acute MI, it may be protective and beneficial of the slow rate may compromise cardiac output. Certain medications, such as beta blockers, may cause it.	The SA node discharges irregularly. The pacing rate varies w/respiration, especially in children and elderly people.	Stimuli originates within atria, but not in the SA. In patients w/heart disease, frequent PACs may precede paroxymal SVT, A-fib, or A-flutter.	Its presence may be the first indication of cardiac disease. s/s depend on ventricular response rate.	Rapid, erratic electrical discharge comes for multiple points in the atria => ineffective atrial contraction => ↓stroke volume ↓cardiac output It is usually a chronic arrhythmia associated w/heart disease. s/s depend on ventricular response rate.	The rate is so fast that the P waves may not be seen. It may be related to caffeine intake, nicotine, stress, or anxiety in healthy adults.
								

ECG Changes Due to Hyperkalemia

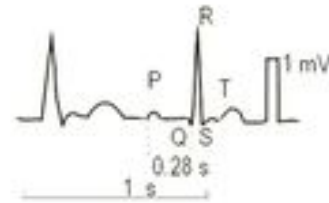


Hyperkalemia Treatment			
Intervention	Onset	Duration	Mechanism
Membrane stabilization			
Calcium gluconate or chloride	Immediate	30-60 min	Membrane stabilization
Redistribution			
Insulin	20-30 min	4-6 hr	Shifts potassium into cell
Albuterol	15-30 min	2-4 hr	Upregulates cAMP & shifts potassium into cells
Sodium bicarbonate	5-10 min	1-2 hr	Shifts potassium into cell
Elimination			
Furosemide	15-30 min	2-3 hr	Increases renal potassium excretion
Sodium polystyrene	1-4 hr	4-6 hr	Increases GI potassium excretion
Hemodialysis	Immediate	2-6 hr	Removes potassium

NURSING MNEMONICS & TIPS

"THE HEART BLOCK POEM"

If the R is far from P,
then you have a **FIRST DEGREE.**



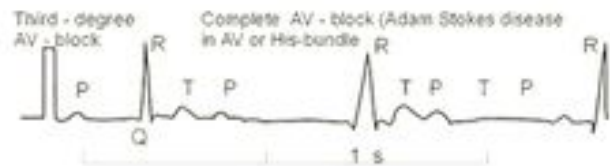
Longer, longer, longer, drop!
Then you have a **WENCKEBACH.**



If some Ps don't get through,
then you have **MOBITZ II.**



If Ps and Qs don't agree,
then you have a **THIRD DEGREE.**



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LEARN MORE: **HEART BLOCKS**

Heart blocks are abnormal heart rhythm where the heart beats too slowly. In this condition, the electrical signals that tell that heart to contract are partial or totally blocked between the upper chambers (atria) and lower chambers (ventricles).

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