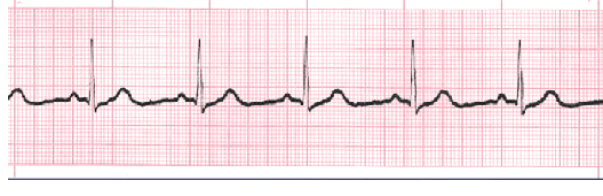





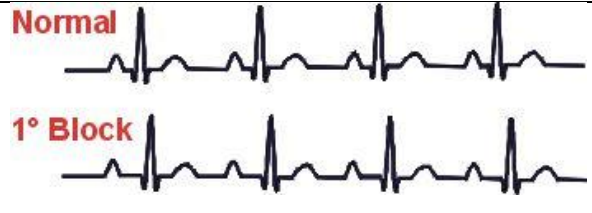


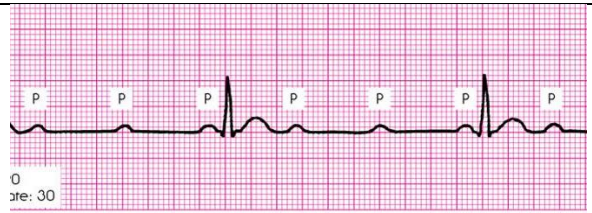


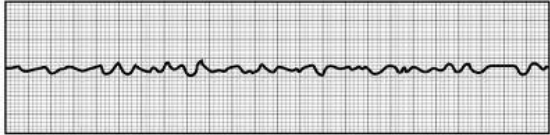


Rhythm	Clinical Associations	ECG Characteristics	Clinical Significance	Treatment	Strip
<p>Sinus Bradycardia</p> <ul style="list-style-type: none"> -Conduction path same as NSR -SA node fires at <60 bpm -Symptomatic- HR <60 resulting in symptoms (chest pain, syncope) 	<ul style="list-style-type: none"> -Normal in some aerobic athletes and some pts during sleep -Carotid sinus massage, Valsalva maneuver, Hypothermia, Increased intraocular pressure, Vagal stimulation -Drugs (b-blockers, CCB) 	<ul style="list-style-type: none"> -HR: <60 bpm -Rhythm: regular -P wave: normal, before each QRS -PR Interval: normal -QRS: normal shape/duration 	<ul style="list-style-type: none"> -Depends on how pt hemodynamically tolerates -S/sx of symptomatic Bradycardia: pale, cool skin; hypotension; weakness; angina; dizziness or syncope; confusion or disorientation; shortness of breath 	<ul style="list-style-type: none"> -Atropine (anticholinergic) if symptomatic -Possible pace maker -D/t drugs: d/c, reduce dose, hold - Dopamine IV Inf., Epi IV Inf. - Replace Electrolytes - fix H's and T's 	
<p>Sinus Tachycardia</p> <ul style="list-style-type: none"> -Conduction path same as NSR -D/c rate from sinus node increases b/c vagal inhibition or sympathetic stimulation -Sinus rate is 101-200 bpm 	<ul style="list-style-type: none"> -Exercise, fever, pain, hypotension, hypovolemia, anemia, hypoxia, hypoglycemia, MI, HF, hyperthyroidism, anxiety, fear -Drugs: Nicotine, cocaine, epinephrine, norepinephrine, atropine, caffeine, theophylline, Procardia, hydralazine, hypercalcemia 	<ul style="list-style-type: none"> -HR: 101-200 bpm -Rhythm: regular -P wave: normal, before each QRS -PR Interval: normal -QRS: normal shape/duration 	<ul style="list-style-type: none"> -Depends on pt tolerance of ↑ HR -Sx: dizziness, dyspnea, hypotension due to decreased cardiac output -↑ myocardial o2 consumption associated with ↑HR -Angina or ↑infarction size may accompany in pt w CAD or acute MI 	<ul style="list-style-type: none"> -Treat the underlying cause -Pain: effective pain management -Hypovolemia: treat hypovolemia -If stable: vagal maneuvers, IV beta blockers given to reduce HR and myocardial o2 demand - fix H's and T's 	
<p>Premature Atrial Contraction</p> <ul style="list-style-type: none"> -Originates at site other than SA -Starts L/R atrium travels across atrium by abnormal path creating distorted P wave -At AV it may be stopped, delayed (long PR interval) or go normally 	<ul style="list-style-type: none"> -Normal Heart: emotional stress, physical fatigue, caffeine, tobacco, alcohol, pain, Nicotine, cocaine -Electrolyte imbalance, Calcium, hyperthyroidism, COPD, -Heart disease: CAD, valvular disease, hypercalcemia 	<ul style="list-style-type: none"> -HR: varies with underlying rate and frequency of PAC -Rhythm: irregular -P wave: different shape (notched, downward, hidden in T wave) -PR Interval: longer or shorter but WNL -QRS: usually normal, if >0.12 abnormal conduction via vents 	<ul style="list-style-type: none"> -Not significant if isolated PAC in healthy heart -Pt report "palpitations" "skip a beat" -Heart disease: frequent PAC- enhanced automaticity of atria, or reentry (may warn of more serious dysrhythmias-supraventricular tachycardia) 	<ul style="list-style-type: none"> -Depends on sx -Withdrawal of caffeine or sympathomimetic drugs - Treat Pain -Replace Electrolytes - fix H's and T's *B-blockers (may decrease PACs) 	
<p>Supraventricular Tachycardia</p> <ul style="list-style-type: none"> -Originates in ectopic focus above bundle of His -Occurs d/t re-excitation of atria when there's a one-way block -Abrupt onset and termination followed by brief asystole -Some degree AV block possible 	<ul style="list-style-type: none"> -Normal Heart: deep inspiration, stimulants (caffeine, Cocaine and tobacco) -hypercalcemia -Rheumatic heart disease, digitalis toxicity, CAD, cor pulmonale 	<ul style="list-style-type: none"> -HR: 150-220 bpm -Rhythm: regular/slightly irregular -P wave: hidden in T wave or irregular shape -PR Interval: shortened or normal -QRS: usually normal 	<ul style="list-style-type: none"> -Depends on associated symptoms -Prolonged episode and HR >180 may precipitate decreased CO d/t reduced stroke volume -Sx often include hypotension, dyspnea, angina 	<ul style="list-style-type: none"> -Vagal stimulation: Valsalva maneuver and coughing -Drug tx: IV adenosine (1st), IV b-blocker, CCB, amiodarone -If pt remains unstable, cardioversion is used -Radiofrequency catheter ablation (burn foci generating ectopic rhythm) - fix H's and T's 	
<p>Atrial Flutter</p> <ul style="list-style-type: none"> -Atrial tach dysrhythmia -ID by recurring, regular, saw tooth shaped flutter waves -Originate from single ectopic focus in R atrium (or L but uncommon) 	<ul style="list-style-type: none"> -Rarely occurs in healthy heart -Diseased states: CAD, HTN, mitral valve disorders, PE, chronic lung disease, cor pulmonale, cardiomyopathy, hyperthyroidism -Drugs: digoxin, quinidine, epinephrine, cocaine, hypercalcemia 	<ul style="list-style-type: none"> -HR: Atrial: 200-350 bpm; Vent: varies r/t conduction ratio -Rhythm: Regular (A and V) -P wave: None (F waves- more F waves than QRS complexes) -PR Interval: Variable/not measurable -QRS: usually Normal 	<ul style="list-style-type: none"> -High ventricular rates and loss of atrial "kick" (sinus P wave) decrease CO and cause serious consequences such as HF, esp if heart disease hx -↑ Stroke risk d/t risk thrombus formation in atria from stasis of blood -Warfarin given to prevent stroke 	<ul style="list-style-type: none"> -Primary goal: slow ventricular response by increasing AV block -Cardioversion if an emergency -Anti-dysrhythmia drugs: Amiodarone, propafenone hcl, Ibutilide, flecainide -Radio frequency catheter ablation - fix H's and T's 	
<p>Atrial Fibrillation</p> <ul style="list-style-type: none"> -Total disorganization of atrial electrical activity due to multiple ectopic foci resulting in loss of effective atrial contraction -Paroxysmal or persistent (>7 Days) -Sometimes, atrial flutter and atrial 	<ul style="list-style-type: none"> -Primarily in pts w/ underlying heart disease (CAD, rheumatic heart dx, cardiomyopathy, HTN, HF, pericarditis) -Often develops acutely w/ thyrotoxicosis, ETOH intox, caffeine use, electrolyte imbalances, stress, 	<ul style="list-style-type: none"> -HR: Atrial: up to 600 bpm; Vent: varies 60-100 controlled, >100 Rapid, <60 slow vent response -Rhythm: Irregular -P wave: Replaced by fibrillation waves 	<ul style="list-style-type: none"> -Results in ↓CO d/t ineffective atrial contractions and/or rapid ventricular response -Thrombi form in atria d/t blood stasis -Thrombi may embolize and cause stroke (A Fib responsible for 20% all) 	<ul style="list-style-type: none"> -Goal: ↓vent response (<100), prevent cerebral embolism, convert to NSR if possible -Drugs (rate control): CCB, B-blockers, digoxin, dronedarone -Anti-dysrhythmia drugs: Amiodarone, Ibutilide 	

Rhythm	Clinical Associations		ECG Characteristics	Clinical Significance	Treatment	Strip
fibrillation may coexist	cardiac surgery		-PR Interval: Not measurable -QRS: normal shape/duration		-Cardioversion or Ablation - fix H's and T's	
1° AV Block -Every impulse conducted to ventricles but AV conduction is long -After through AV, ventricles respond normally	-MI, CAD, rheumatic fever, hyperthyroidism, vagal stimulation -Drugs: digoxin, B-blockers, CCB, flecainide		-HR: Normal -Rhythm: Regular -P wave: Normal -PR Interval: Prolonged (>0.20 seconds) -QRS: normal shape/duration	-usually not serious but can be precursor of higher degrees of AV block -asx	-usually No treatment -Modifications to potentially causative meds may be considered -Monitor pts for new changes in rhythm (more serious AV block) - fix H's and T's	Normal  1° Block 
2° AV Block Type 1 (Wenckebach/Mobitz I) -Gradual lengthening of PR interval d/t prolonged AV conduction time until an atrial impulse is nonconducted and a QRS is blocked -Most common in AV but can occur in His-purkinje system -Once beat is blocked, cycle repeats w progressive lengthening of PR interval until another QRS drops	-Digoxin -Beta-blockers/ CCB -CAD -Other dx that slow AV conduction		-HR: Atrial: normal; vent: possibly slower d/t blocked QRS leading to bradycardia -Rhythm: Pattern of grouped beats -P wave: Normal shape -PR Interval: Gradual lengthening -QRS: normal shape/duration	-Usually d/t myocardial ischemia or infarction -Generally transient and well tolerated -In some pts may be warning sign of a more serious conduction disturbance such as complete heart block	-If sx: atropine to ↑HR or temporary pacemaker (especially if hx MI) -If asx: rhythm observed with transcutaneous pacemaker on standby -Bradycardia more likely to become symptomatic when hypotension, HF or shock is present - fix H's and T's	
2° AV Block Type 2 (Mobitz II) -P wave nonconducted w/o progressive PR lengthening -Usually occurs when block in one of the bundle branches is present -More serious type of block -Certain # of impulses are not conducted into the ventricles -Occur in ratios 2:1, 3:1, etc (2 P waves for 1 QRS complex) -May occur with varying ratios	-Rheumatic heart disease -CAD -Anterior MI -Drug toxicity -Beta-blockers/ CCB		-HR: Atrial: Normal Vent: depends on intrinsic conduction/degree of block -Rhythm: Atrial: Regular Vent: may be irregular -P wave: Normal shape -PR Interval: Normal or prolonged, constant on conducted beats -QRS: Usually >0.12 sec d/t bundle branch block	-Often progresses to 3° block -Associated with poor prognosis -↓HR frequently results in ↓CO with hypotension and myocardial ischemia -Indication for therapy with permanent pacemaker	-Temporary pacemaker may be necessary if pt becomes symptomatic prior to insertion of permanent pacemaker (e.g., hypotension, angina)	
3° AV Block -Complete Heart Block -No impulses from atria conducted -Atria stimulated and contract independently of ventricles -Vent rhythm is escape rhythm, ectopic pacemaker may be above or below the bundle of His	Severe heart dx -CAD -MI -Myocarditis - Cardiomyopathy	Systemic dx -Amyloidosis -Scleroderma Drugs -Digoxin -Beta-blockers -CCB	-HR: Atrial: sinus 60-100 bpm Vent: r/t block site (AV 60-40, etc) -Rhythm: Regular (unrelated) -P wave: Normal shape -PR Interval: Variable -QRS: Normal or Widened **No time relationship b/t P wave and QRS complex**	-↓CO→ ischemia, HF, and shock -Syncope d/t severe bradycardia or periods of asystole	Symptomatic pts -Transcutaneous pacemaker used until temporary trans venous pacemaker can be inserted -Drugs: Atropine, Epinephrine, Isoproterenol, Dopamine are temporary to ↑HR and BP -If d/t CCB toxicity, toxicity w calcium chloride	
Premature Vent Contraction -Contractions from ectopic focus within ventricles -Premature wide/distorted QRS -Diff foci: diff shape (multifocal) -Same foci: same shape (unifocal) -Couplet, trigeminy, bigeminy -V-Tach if 3+ consecutive PVCs Rate	Stimulants -Caffeine -ETOH -Nicotine - Aminophylline -Epinephrine -Isoproterenol	-Hypoxia -Fever -Exercise -Emotion stress Disease States -MI -Mitral prolapse -HF	-HR: Varies r/t intrinsic rate, # PVCs -Rhythm: Irregular d/t pre beats -P wave: Usually lost in QRS of PVC -PR Interval: Not measurable -QRS: Wide, Distorted, >0.12	-Usually benign in pt w/ normal heart -If hx heart dx: may ↓CO and precipitate angina and HF (depends on frequency) -Monitor apical pulse b/c PVCs usually aren't strong enough to illicit peripheral pulses possibly leading to pulse deficit	-Relates to cause PVCs -Assess hemodynamics r/t need for drug tx -Drug tx: Beta-blockers, Procainamide, Amiodarone, Xylocaine -PVCs in CAD or acute MI indicate vent irritability so monitor pt response	

Rhythm	Clinical Associations		ECG Characteristics	Clinical Significance	Treatment	Strip
> 100 bpm -Can initiate V-Tach or VFib	-Digoxin -Electrolyte Imbalance	-CAD	sec -T wave: Large, Opposite direction to direction of QRS		-fix H's and T's	
Ventricular Tachycardia (VT) -Run of ≥3 PVCs -Ventricles take control as pacer -Different forms r/t QRS conf -Monomorphic: QRSs equal -Polymorphic: QRS gradually change size/shape/direction -Torsades de pointes: polymorphic VT r/t prolonged QT interval of underlying rhythm -Sustained (>30 sec) -Non-sustained (<30 sec) -Life threatening d/t ↓CO and possible development of VFib	-MI -CAD -Electrolyte imbalance -Cardiomyopathy -Mitral valve prolapse -Long QT syndrome -Drug toxicity -CNS disorders -Pts w no hx CV dx		-HR: Vent: 150-250 bpm -Rhythm: Regular or Irregular -P wave: Usually buried in QRS *Possible AV dissociation with P wave independent of QRS complex -PR Interval: Not measurable -QRS: *Distorted in appearance *Duration >0.12 sec *ST-T opposite direction as QRS *R-R interval regular or irregular	-VT stable (pt has pulse) or can be -VT unstable (pt has no pulse) -Sustained VT causes severe ↓CO d/t ↓vent diastolic filling times and loss of atrial contraction -Results in hypotension, pulmonary edema, ↓cerebral blood flow and cardiopulmonary arrest -Must treat quickly even if occurs briefly and stops -May reoccur if no prophylaxis -VFib may also develop	-Precipitating cause must be ID and treated *Monomorphic VT -Stable w/ L vent function: IV Procainamide, Sotalol, Amiodarone or Lidocaine -Unstable, poor L vent function: IV Amiodarone or Lidocaine then Synchronize cardioversion * <u>Polymorphic VT (R wave Morphication)</u> -Normal baseline QT interval: Beta-blockers, Lidocaine, Amiodarone, Procainamide, or Sotalol, Defib if no change.. (fix H's and T's)	*Polymorphic VT (cont) -Prolonged QT interval: IV Magnesium, Isoproterenol, Dilantin, Lidocaine OR antitachycardia pacing; D/c drugs that prolong QT interval; Cardioversion needed if not responsive *Pulseless VT: CPR and rapid defibrillation followed by vasopressors and antidysrhythmics if defib unsuccessful
Ventricular Fibrillation (VF) -Irregular waveforms varying shapes and amplitudes -Firing of multiple ectopic foci in ventricle (quivering) -No ventricle contraction.. NO CO	-Acute MI -Myocardial Ischemia -HF -Cardiomyopathy -During pacing/caths -Accidental shock -Hyperkalemia -Hypoxemia -Acidosis -Drug toxicity		-HR: Not measurable -Rhythm: Irregular and Chaotic -P wave: Not visible -PR Interval: Not measurable -QRS: Not measurable	-Results in an unresponsive, pulseless, apneic state -Tx rapidly or pt will die	-CPR -ACLS protocols with defibrillation and definitive drug therapy. CPR, Defib, Epi, Amiodarone, intubation, Lidocaine fix H's and T's	
Asystole -Absence of ventricular electrical activity (no depolarization occurs) -Pt unresponsive, pulseless, apneic -VF may look like Asystole, so rhythm assessed in >1 lead	Result of: -Advanced cardiac disease -Severe conduction disturbance -End stage HF		-HR: None -Rhythm: None -P wave: None, Occasionally seen -PR Interval: None -QRS: None	-Usually cannot be resuscitated	-CPR -ACLS initiation with definitive drug therapy, including: Epi, intubation and fix H's and T's	