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<td>Sinus Bradycardia</td>
<td>-Conduction path same as NSR -SA node fires at &lt;60 bpm -Symptomatic: HR &lt;60 resulting in symptoms (chest pain, syncope)</td>
<td>-Normal in some aerobic athletes and some pts during sleep -Cardioid-sinus massage, Valsalva maneuver, Hypothermia, Increased intracranial pressure, Vagal stimulation</td>
<td>-Depends on pt hemodynamically tolerates -1/5 of symptomatich Bradycardia: pale, cool skin; hypotension; weakness; angina; dizziness or syncope; confusion or disorientation; shortness of breath</td>
<td>-Atropine (anti-cholinergic) if symptomatic -Propranolol pace maker -D/t drugs: d/c, reduce dose, hold -Dopamine IV Inf., Epi IV Inf. -Replace Electrolytes -fix H's and T's</td>
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<tr>
<td>Sinus Tachycardia</td>
<td>-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</td>
<td>-Exercise, fever, pain, hypotension, hypovolemia, anemia, hypoxia, hypoglycemia, MI, HF, hyperthyroidism, anxiety, fear -Drugs: Nicotine, cocaine, epinephrine, norepinephrine, atropine, caffeine, theophylline, Procainamide, hydralazine, -hypercalcemia</td>
<td>-Depends on pt tolerance of ↑ HR -1/5x of myocardial o2 consumption associated with ↑ HR -Angina or ↑Infarct size may accompany it in pt w CAD or acute MI</td>
<td>-Treat the underlying cause -Pain: effective pain managing -Hypovolemia: treat hypovolemia -If stable: vagal maneuvers, IV beta blockers given to reduce HR and myocardial o2 demand - fix H's and T's</td>
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<td>Premature Atrial Contraction</td>
<td>-Originate at 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</td>
<td>-Normal Heart: emotional stress, physical fatigue, caffeine, tobacco, alcohol, pain, Nicotine, cocaine -Electrolyte imbalance, Calcium, hypercalcemia, COPD, -Heart disease: CAD, valvular disease, hypercalcemia</td>
<td>-Not significant if isolated PAC in healthy heart -Pt report &quot;palpitations&quot; &quot;skip a beat&quot; -Heart disease: frequent PAC- enhanced automaticity of atria, or reentry (may warn of more serious dysrhythmias-supraventricular tachycardia)</td>
<td>-Depends on sx -Withdrawal of caffeine or sympathomimeticic drugs - Treat Pain -Replace Electrolytes - fix H's and T's</td>
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<td>Supraventricular Tachycardia</td>
<td>-Originate 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</td>
<td>-Normal Heart: deep inspiration, stimulants (caffeine, Cocaine and tobacco) -hypercalcemia -Rheumatic heart disease, digitalis toxicity, CAD, cor pulmonale</td>
<td>-Depends on associated symptoms -Prolonged episode and HR &gt;180 may precipitate decreased CO d/t reduced stroke volume -Sx often include hypotension, dyspnea, angina</td>
<td>-Vagal stimulation: Valsalva maneuver and coughing -Drug tox: IV adenosine (1'), 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</td>
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<td>Atrial Flutter</td>
<td>-Atrial tach dysrhythmia -ID by recurring, regular, saw tooth shaped flutter waves -Originate from single ectopic focus in R atrium (or L but uncommon)</td>
<td>-Rarely occurs in healthy heart -Diseased states: CAD, HTN, mitral valve disorders, PE, chronic lung disease, atrial flutter, cardiomyopathy, hyperthyroidism -Drugs: digoxin, quinidine, epinephrine, cocaine, -hypercalcemia</td>
<td>-High ventricular rates and loss of atrial &quot;kick&quot; (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</td>
<td>-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</td>
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<td>Atrial Fibrillation</td>
<td>-Total disorganization of atrial electrical activity due to multiple ectopic foci resulting in loss of effective atrial contraction -Paroxysmal or persistent (&gt;7 Days) -Sometimes, atrial flutter and atrial</td>
<td>-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,</td>
<td>-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 fibr responsible for 20% all)</td>
<td>-Goal: ↓ vent response (&lt;100), prevent cerebral embolism, convert to NSR if possible -Drugs (rate control): CCB, B-blockers, digoxin, dronedarone -Anti-dysrhythmia drugs: Amiodarone, Ibutilide</td>
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<td>1° AV Block</td>
<td>-Every impulse conducted to ventricles but AV conduction is long -After through AV, ventricles respond normally</td>
<td>-MI, CAD, rheumatic fever, hyperthyroidism, vagal stimulation -Drugs: digoxin, B-blockers, CCB, flecainide</td>
<td>-HR: Normal -Rhythm: Regular -P wave: Normal -PR interval: Prolonged (&gt;0.20 seconds) -QRS: normal shape/duration</td>
<td>-usually not serious but can be precursor of higher degrees of AV block -asx</td>
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<td>2° AV Block Type 1 (Wenckebach/Mobitz I)</td>
<td>-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 with progressive lengthening of PR interval until another QRS drops</td>
<td>-Digoxin -Beta-blockers/ CCB -CAD -Other dx that slow AV conduction</td>
<td>-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</td>
<td>-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</td>
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<tr>
<td>2° AV Block Type 2 (Mobitz II)</td>
<td>-P wave nonconducted w/o prograde 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</td>
<td>-Rheumatic heart disease -CAD -Anterior MI -Drug toxicity -Beta-blockers/ CCB</td>
<td>-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 &gt;0.12 sec d/t bundle branch block</td>
<td>-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</td>
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<tr>
<td>3° AV Block</td>
<td>-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</td>
<td>Severe heart dx -CAD -MI -Myocarditis -Cardiomyopathy Systemic dx -Amyloidosis -Scleroderma Drugs -Digoxin -Beta-blockers -CCB</td>
<td>-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 <strong>No time relationship b/t P wave and QRS complex</strong></td>
<td>-↓CO→ ischemia, HF, and shock -Syncope d/t severe bradycardia or periods of asystole</td>
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<tr>
<td>Premature Vent Contraction</td>
<td>-Contractions from ectopic focus within ventricles -Premature wide/distorted QRS -Diff dci: diff shape (multifocal) -Same foci: same shape (unifocal) -Couplet, trigeminy, bigeminy -V-Tach if 3+ consecutive PVCs Rate</td>
<td>Stimulants -Caffeine -ETOH -Nicotine -Aminophylline -Epinephrine -Isoproterenol -Hyoxia -Fever -Emotion stress Disease States -MI -Mitral prolapse -HF</td>
<td>-HR: Varies r/t intrinsic rate, # PVCs -Rhythm: Irregular d/t pre existing AV block -P wave: Usually lost in QRS of PVC -PR interval: Not measurable -QRS: Wide, Distorted, &gt;0.12 sec</td>
<td>-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</td>
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**ECG Characteristics**

- **PR interval:** Not measurable
- **QRS:** normal shape/duration
- **HR:** Normal
- **Rhythm:** Regular
- **P wave:** Normal
- **PR interval:** Prolonged (>0.20 seconds)
- **QRS:** normal shape/duration
- **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
- **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
- **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**
- **HR:** Varies r/t intrinsic rate, # PVCs
- **Rhythm:** Irregular d/t pre existing AV block
- **P wave:** Usually lost in QRS of PVC
- **PR interval:** Not measurable
- **QRS:** Wide, Distorted, >0.12 sec
**Rhythm**

- > 100 bmp
  - Can initiate V-Tach or VFib

**Clinical Associations**

- Digoxin
- Electrolyte imbalance
- CAD

**ECG Characteristics**

- 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
- PR Interval: Not measurable
- QRS: Not measurable

**Clinical Significance**

- 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

- Polymorphic VT (R wave Morphication)
  - 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

**Treatment**

- Fix H's and T's

**Strip**

- Polymorphic VT (cont)

**Ventricular Tachycardia (VT)**

- Run of ≥3 PVCs
- Ventricles take control as pacer
- 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

**Ventricular Tachycardia (VT) - Causes**

- MI
- CAD
- Electrolyte imbalance
- Cardiomyopathy
- Mitral valve prolapse
- Long QT syndrome
- Drug toxicity
- CNS disorders
- Pts w no hx CV dx

**Ventricular Fibrillation (VF)**

- Irregular waveforms varying shapes and amplitudes
- Firing of multiple ectopic foci in ventricle (quivering)
- No ventricle contraction... NO CO

**Ventricular Fibrillation (VF) - Causes**

- Acute MI
- Myocardial Ischemia
- HF
- Cardiomyopathy
- During pacing/caths
- Accidental shock
- Hyperkalemia
- Hypoxemia
- Acidosis
- Drug toxicity

**Asystole**

- Absence of ventricular electrical activity (no depolarization occurs)
- Pt unresponsive, pulseless, apneic
- VF may look like Asystole, so rhythm assessed in >1 lead

**Asystole - Causes**

- Result of:
  - Advanced cardiac disease
  - Severe conduction disturbance
  - End stage HF

**Asystole - Features**

- HR: None
- Rhythm: None
- P wave: None, Occasionally seen
- PR Interval: None
- QRS: None

**Asystole - Treatment**

- CPR
- ACLS protocols with defibrillation and definitive drug therapy.
  - CPR, Defib, Epi, Amiodarone, intubation, Lidocaine
  - Fix H's and T's

**Asystole - Results**

- Usually cannot be resuscitated

- CPR
- ACLS initiation with definitive drug therapy, including: Epi, intubation and fix H's and T's