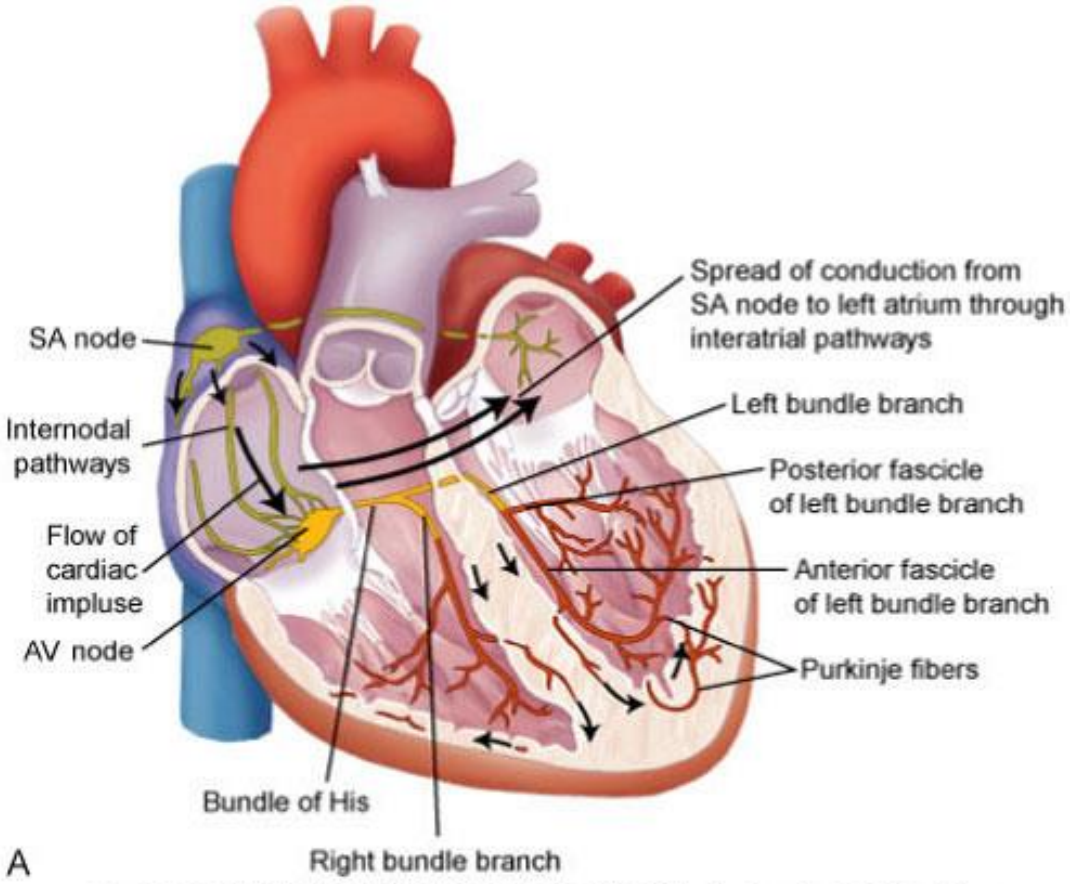
Dysrhythmias

LUCENT NCLEX REVIEWS

Properties of Cardiac Cells

- Automaticity
- Excitability
- Conductivity
- Contractility

Conduction System of the Heart



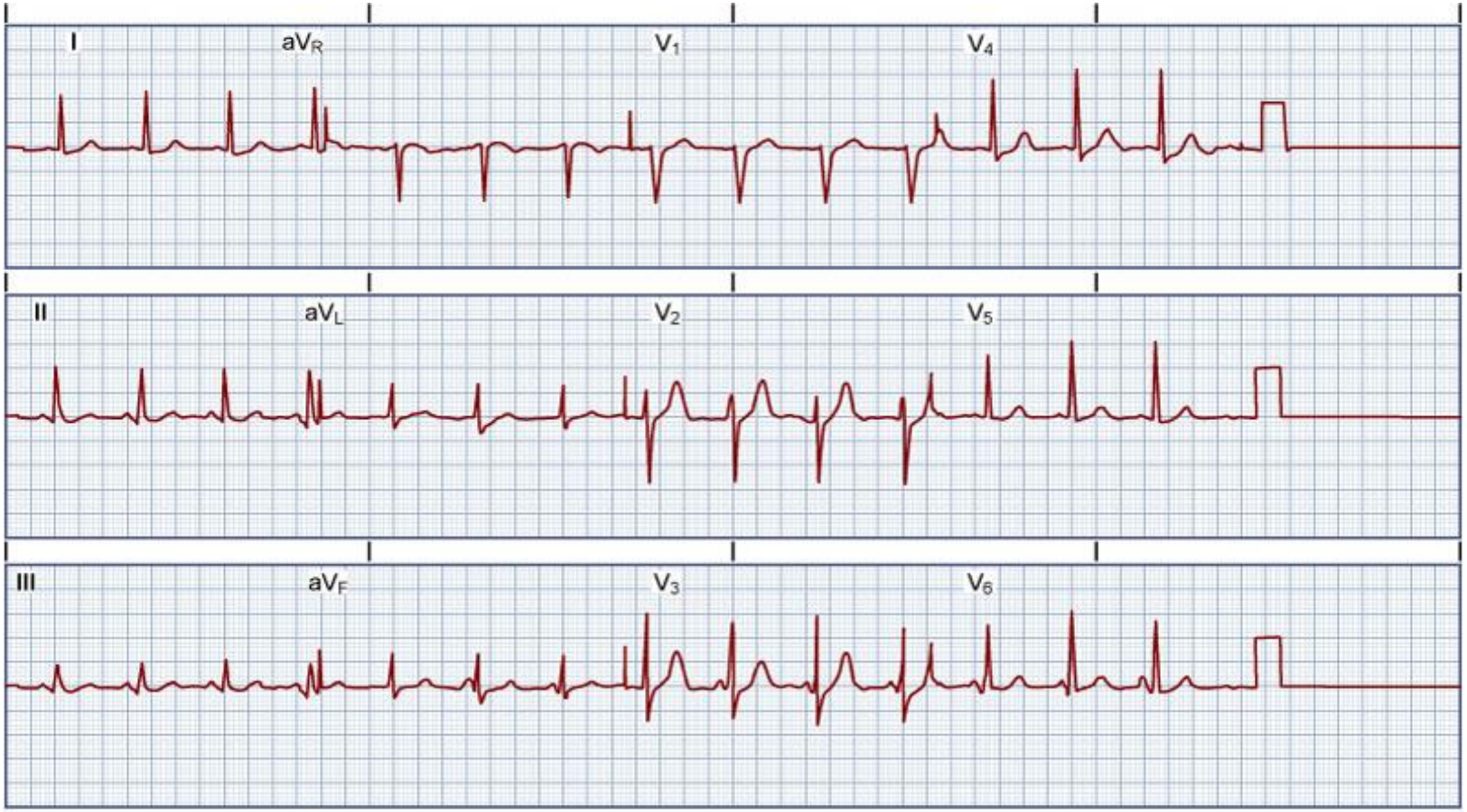
Nervous System Control of Heart

- Autonomic nervous system controls
  - Parasympathetic nervous system
    - Decreases rate of SA node
    - Slows impulse conduction of AV node
  - Sympathetic nervous system
    - Increases rate of SA node
    - Increases impulse conduction of AV node
    - Increases cardiac contractility

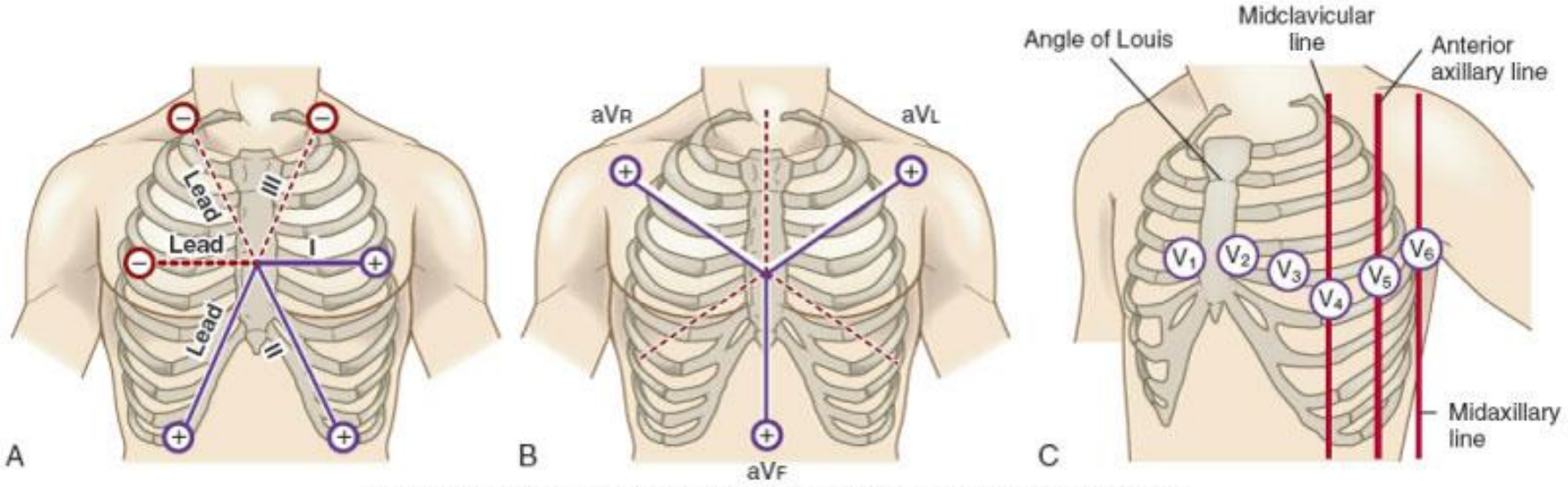
Dysrhythmias

- Disorder of impulse formation, conduction of impulses, or both
- SA node normal pacemaker of heart (60–100 beats/minute)
- Secondary pacemakers
  - AV node (40–60 beats/minute)
  - His-Purkinje fibers (20–40 beats/minute)

12-Lead ECG



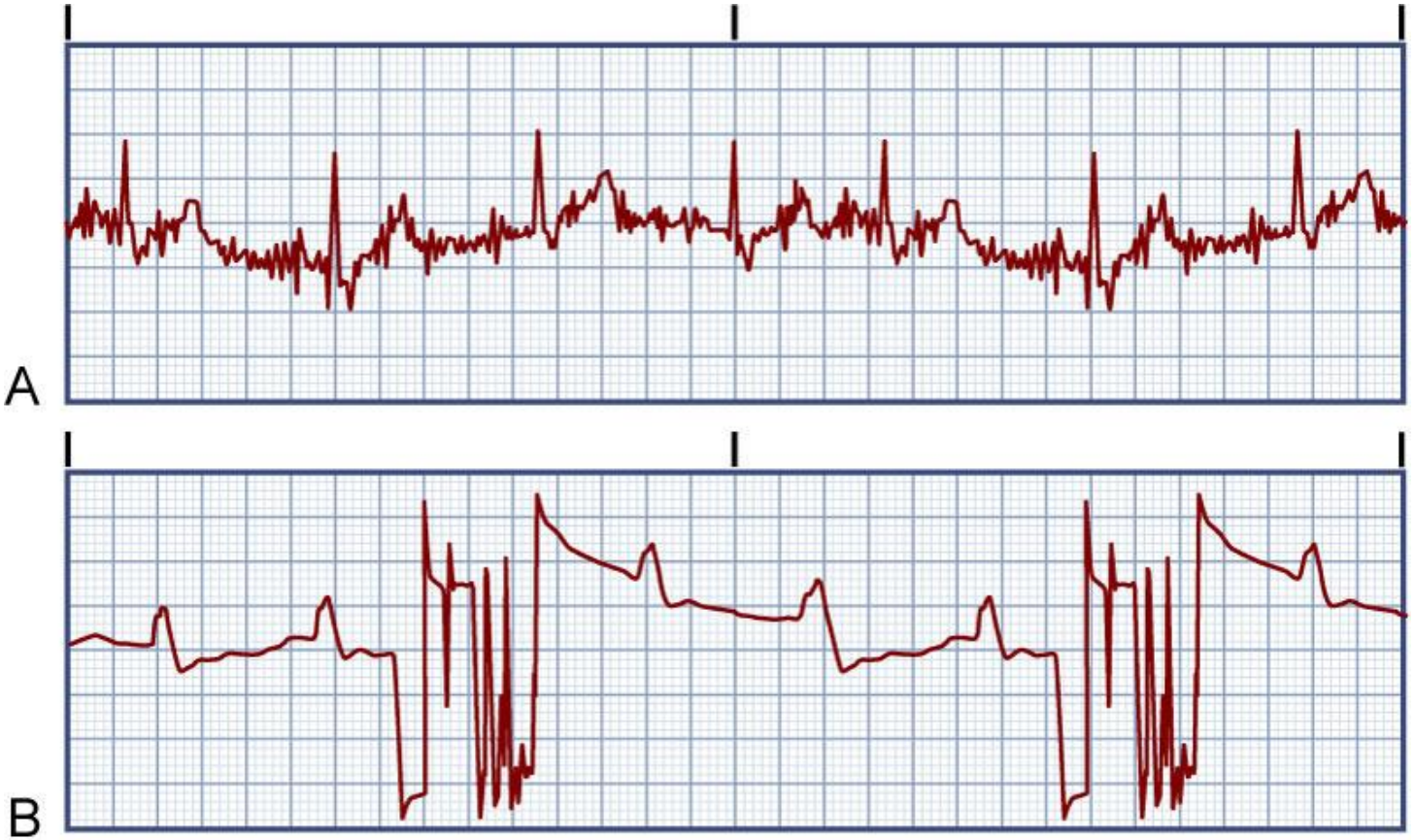
Lead Placement



Patient Preparation

- Clip excessive hair on chest wall
- Rub skin with dry gauze
- May need to use alcohol for oily skin
- Apply electrode pad

Artifact



Telemetry Monitoring

- Observation of HR and rhythm at a distant site
- Two types
  - Centralized monitoring system
  - Advanced alarm system alerts when it detects dysrhythmias, ischemia, or infarction

Assessment of Cardiac Rhythm

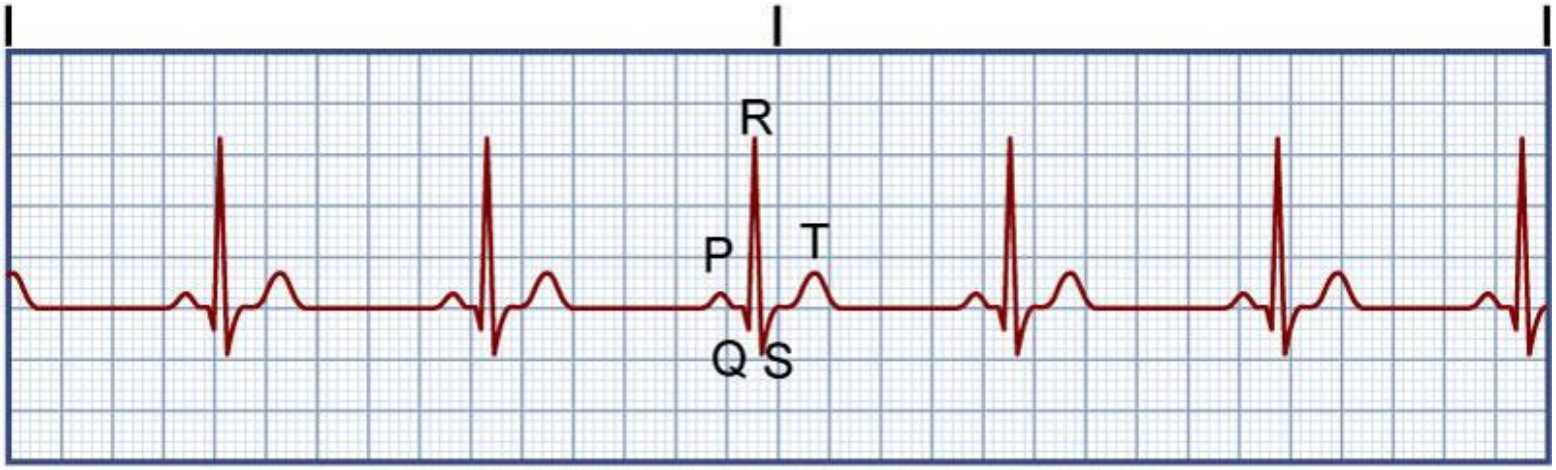
- Interpret the rhythm AND evaluate the clinical status of the patient
- Is the patient hemodynamically stable?
- Determine cause of dysrhythmia
- Treat the patient, not the monitor!

Assessment of Cardiac Rhythm

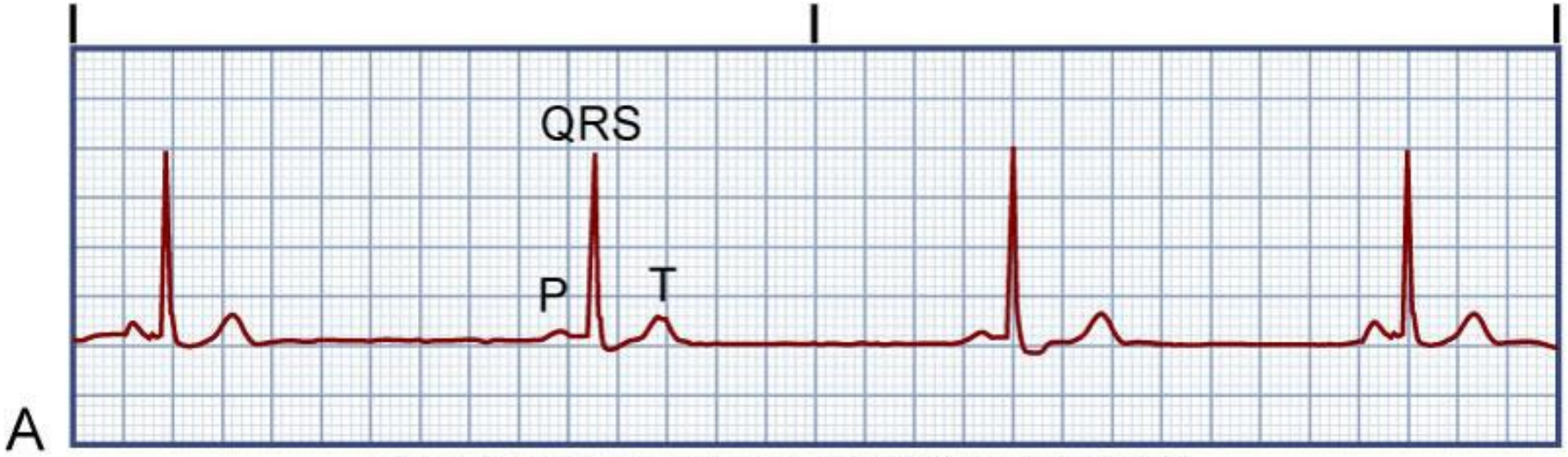
1. P wave
2. Atrial rate and rhythm
3. P-R interval
4. Ventricular rate and rhythm
5. QRS complex
6. ST segment
7. Q-T interval
8. T wave

Normal Sinus Rhythm

- Sinus node fires 60–100 beats/minute
- Follows normal conduction pattern



Sinus Bradycardia



Sinus Bradycardia

- Normal rhythm in aerobically trained athletes and during sleep
- Can occur in response to parasympathetic nerve stimulation and certain drugs
- Also associated with some disease states

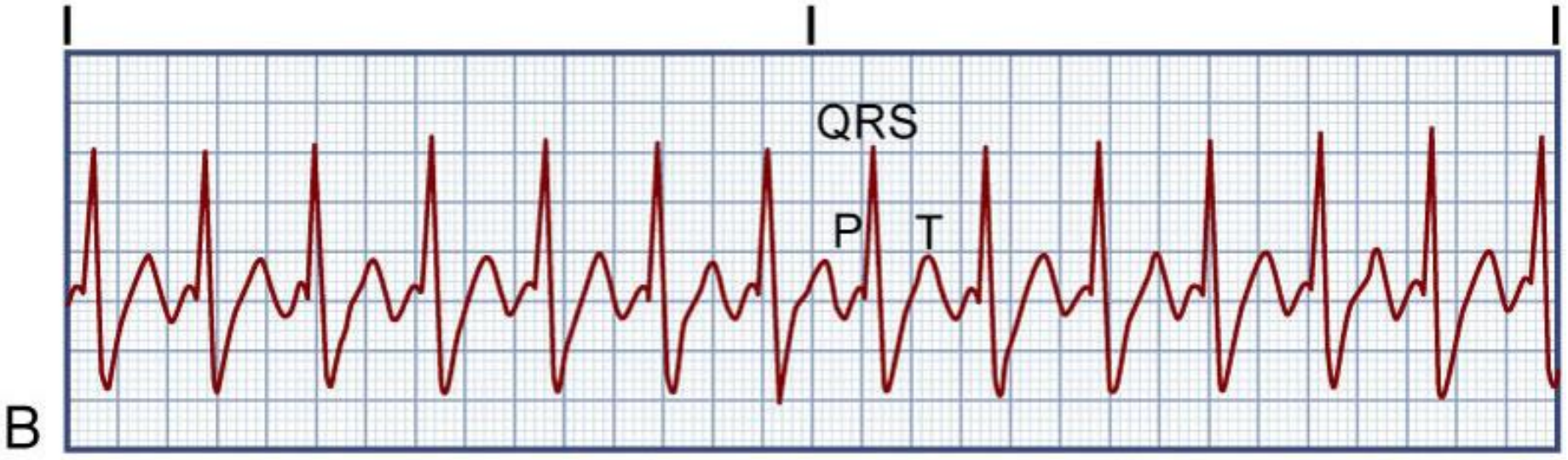
Sinus Bradycardia

- **Manifestations**
  - **Hypotension**
  - **Pale, cool skin**
  - **Weakness**
  - **Angina**
  - **Dizziness or syncope**
  - **Confusion or disorientation**
  - **Shortness of breath**

Sinus Bradycardia

- **Treatment**
  - **Atropine**
  - **Pacemaker**
  - **Stop offending drugs**

Sinus Tachycardia



Sinus Tachycardia

- Caused by vagal inhibition or sympathetic stimulation
- Associated with physiologic and psychologic stressors
- Drugs can increase rate

Sinus Tachycardia

- **Manifestations**
  - Dizziness
  - Dyspnea
  - Hypotension
  - Angina in patients with CAD

Sinus Tachycardia

- Treatment
  - Guided by cause (e.g., treat pain)
  - Vagal maneuver
  - β-adrenergic blockers

Premature Atrial Contraction

Premature Atrial Contraction

- Contraction originating from ectopic focus in atrium in location other than SA node
- Travels across atria by abnormal pathway, creating distorted P wave
- May be stopped, delayed, or conducted normally at the AV node

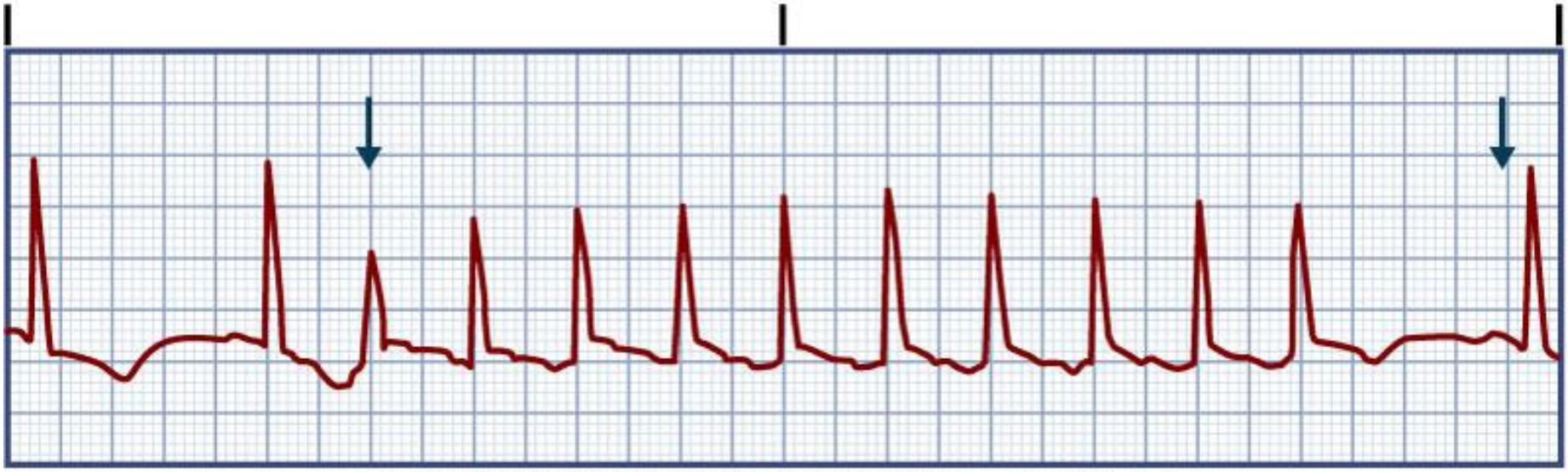
Premature Atrial Contraction

- **Causes**
  - **Stress**
  - **Fatigue**
  - **Caffeine**
  - **Tobacco**
  - **Alcohol**
  - **Hypoxia**
  - **Electrolyte imbalance**
  - **Disease states**

Premature Atrial Contraction

- Manifestations
  - Palpitations
  - Heart "skips a beat"
- Treatment
  - Monitor for more serious dysrhythmias
  - Withhold sources of stimulation
  - β-adrenergic blockers

Paroxysmal Supraventricular Tachycardia (PSVT)



Paroxysmal Supraventricular Tachycardia (PSVT)

- Reentrant phenomenon: PAC triggers a run of repeated premature beats
- Paroxysmal refers to an abrupt onset and termination
- Associated with overexertion, stress, deep inspiration, stimulants, disease, digitalis toxicity

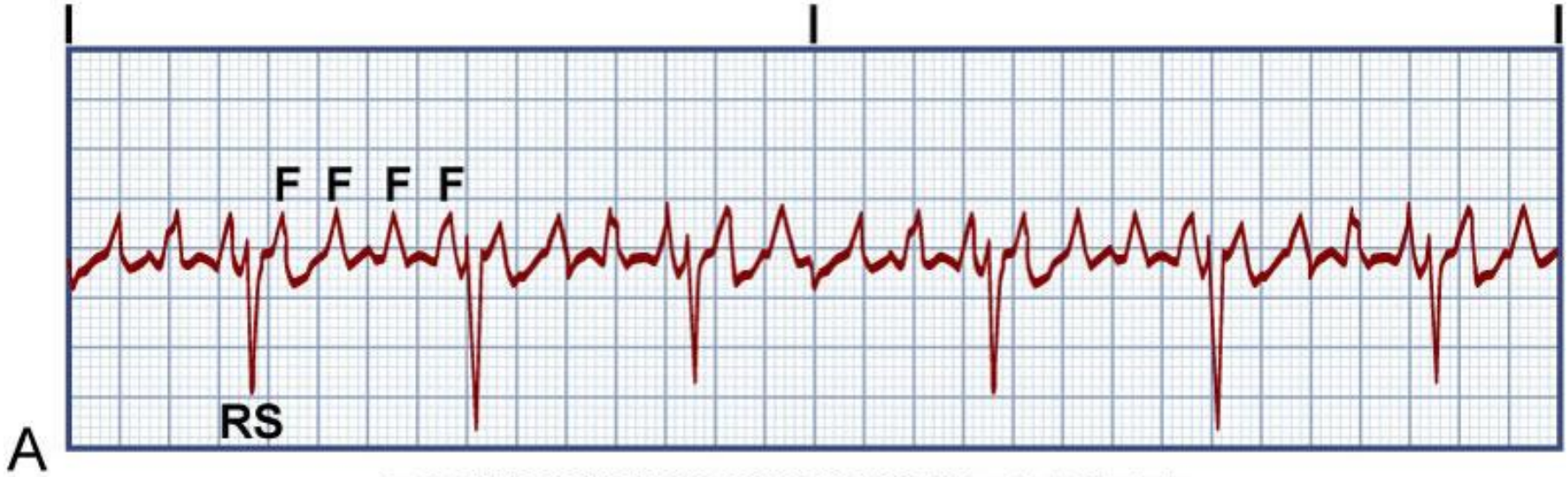
Paroxysmal Supraventricular Tachycardia (PSVT)

- **Manifestations**
  - **HR is 150–220 beats/minute (add for clarification)**
  - **HR > 180 leads to decreased cardiac output and stroke volume**
  - **Hypotension**
  - **Dyspnea**
  - **Angina**

Paroxysmal Supraventricular Tachycardia (PSVT)

- **Treatment**
  - **Vagal stimulation**
  - **IV adenosine**
  - **IV β-adrenergic blockers**
  - **Calcium channel blockers**
  - **Amiodarone**
  - **DC cardioversion**

Atrial Flutter



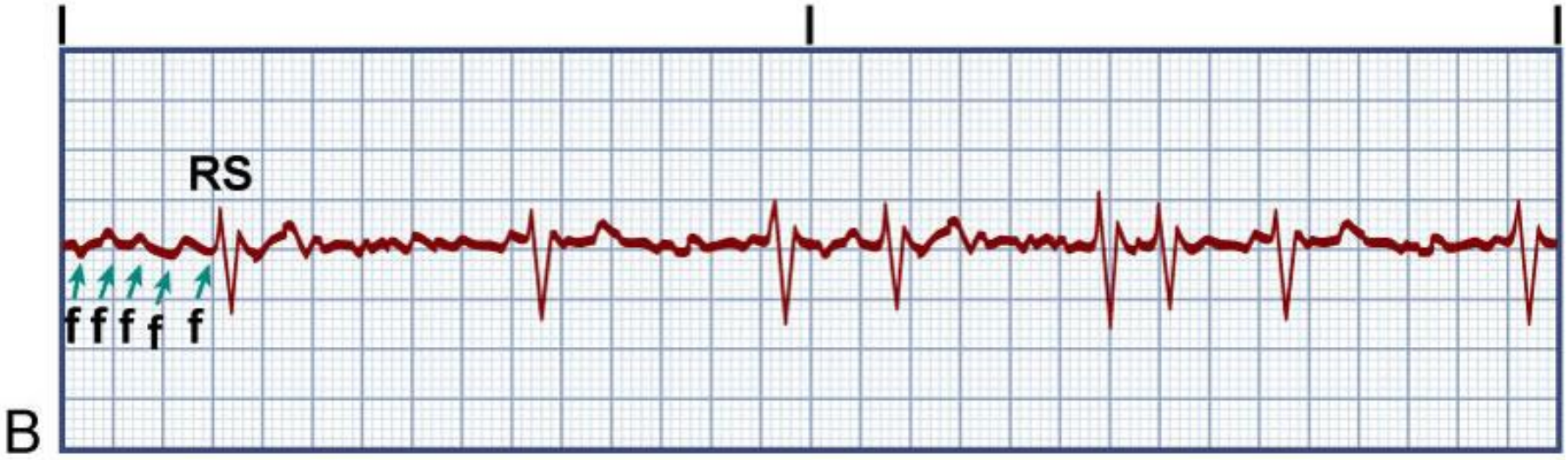
Atrial Flutter

- Typically associated with disease
- Symptoms result from high ventricular rate and loss of atrial "kick" → decreased CO → heart failure
- Increases risk of stroke

Atrial Flutter

- Treatment
  - Pharmacologic agent
  - Electrical cardioversion
  - Radiofrequency ablation

Atrial Fibrillation



Atrial Fibrillation

- Paroxysmal or persistent
- Most common dysrhythmia
- Prevalence increases with age
- Usually occurs in patients with underlying heart disease
- Can also occur with other disease states

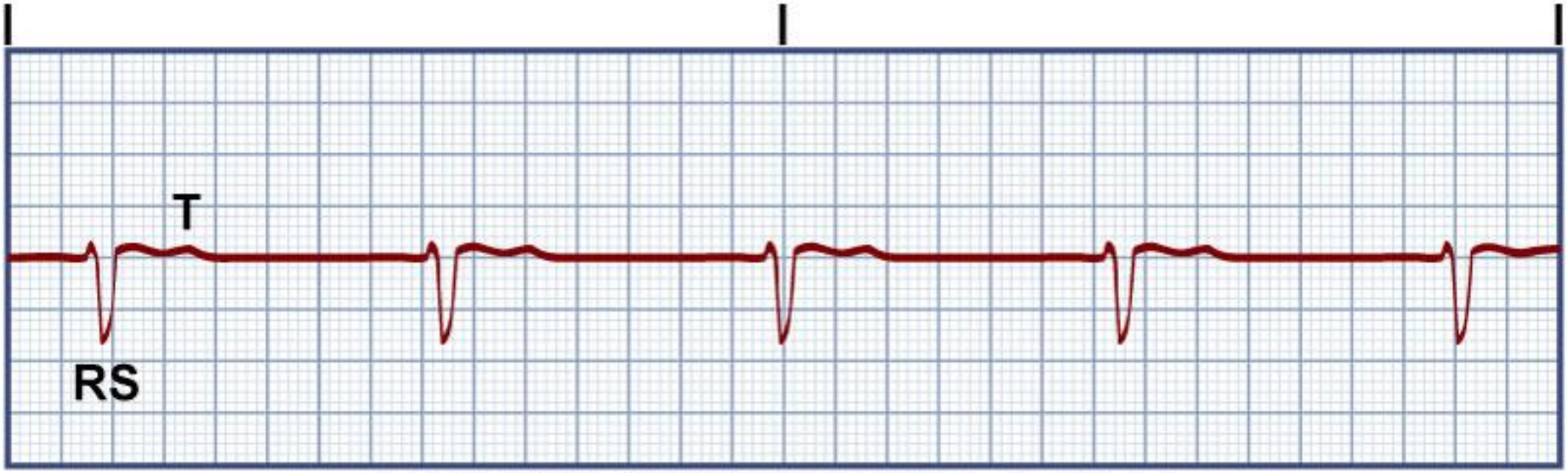
Atrial Fibrillation

- Treatment
  - Drugs to control ventricular rate and/or convert to sinus rhythm (amiodarone and ibutilide most common)
  - Electrical cardioversion
  - Anticoagulation
  - Radiofrequency ablation
  - Maze procedure with cryoablation

Junctional Dysrhythmias

- Dysrhythmias that originate in area of AV node
- SA node has failed to fire, or impulse has been blocked at the AV node
- AV node becomes pacer—retrograde transmission of impulse to atria
- Abnormal P wave; normal QRS
- Associated with disease, certain drugs

Junctional Dysrhythmias



Junctional Dysrhythmia

- Serves as safety mechanism—do not suppress
- If rhythms are rapid, may result in reduction of CO
- Treat if patient is symptomatic
  - Atropine for escape rhythm
  - Correct cause
  - Drugs to reduce rate if tachycardia

First-Degree AV Block

First-Degree AV Block

- Associated with disease states and certain drugs
- Typically not serious
- Patients asymptomatic
- No treatment
- Monitor for changes in heart rhythm

Second-Degree AV Block, Type 1 (Mobitz I, Wenckebach)

Second-Degree AV Block, Type 1 (Mobitz I, Wenckebach)

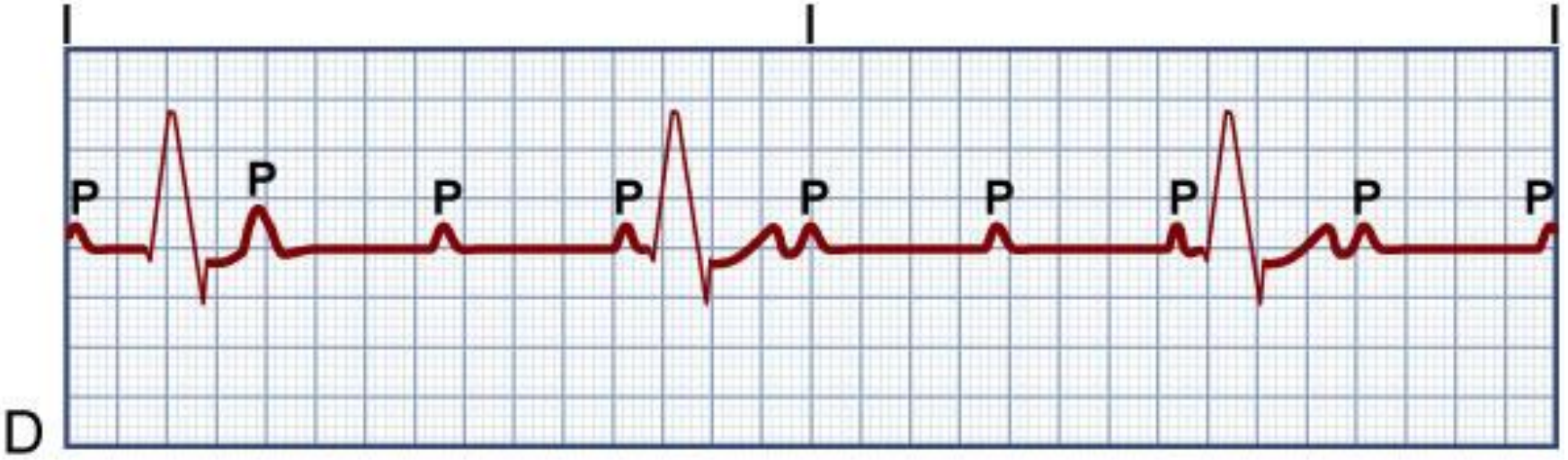
- May result from drugs or CAD
- Typically associated with ischemia
- Usually transient and well tolerated
- Treat if symptomatic
  - Atropine
  - Pacemaker
- If asymptomatic, monitor closely

Second-Degree AV Block, Type 2 (Mobitz II)

Second-Degree AV Block, Type 2 (Mobitz II)

- Associated with heart disease and drug toxicity
- Often progressive and results in decreased CO
- Treat with pacemaker

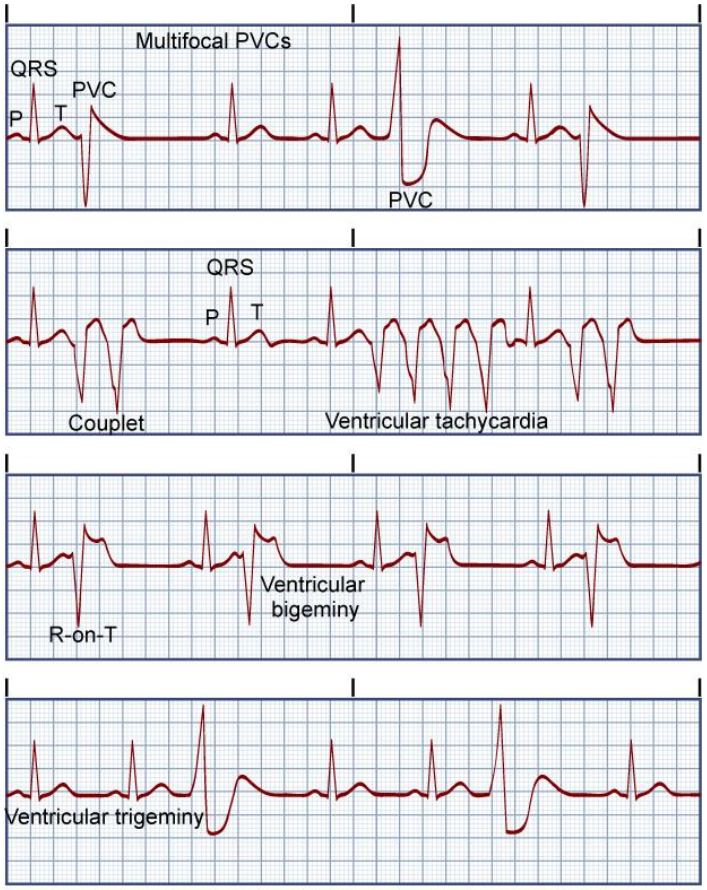
Third-Degree AV Heart Block (Complete Heart Block)



Third-Degree AV Heart Block (Complete Heart Block)

- Associated with severe heart disease, some systemic diseases, certain drugs
- Usually results in decreased CO
- Can lead to syncope, HF, shock
- Treat with pacemaker
- Drugs to increase heart rate if needed while awaiting pacing

Premature Ventricular Contractions



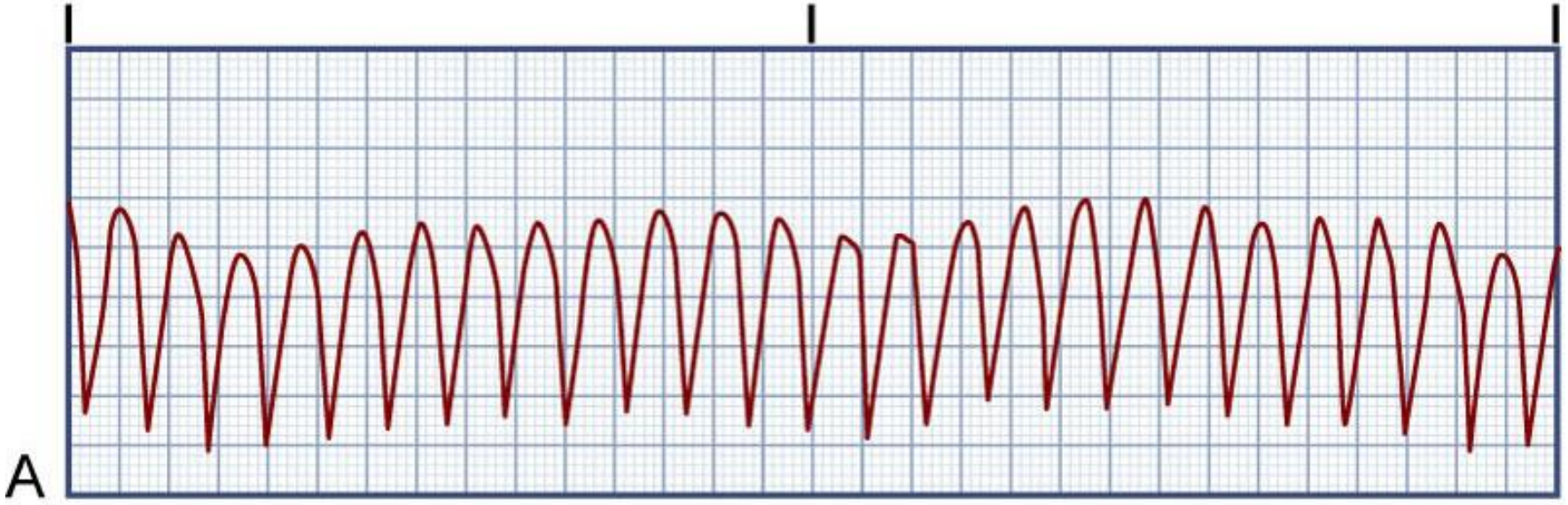
Premature Ventricular Contractions

- Associated with stimulants, electrolyte imbalances, hypoxia, heart disease
- Not harmful with normal heart but CO reduction, angina, and HF in diseased heart
- Assess apical-radial pulse deficit

Premature Ventricular Contractions

- Treatment
  - Correct cause
  - Antidysrhythmics

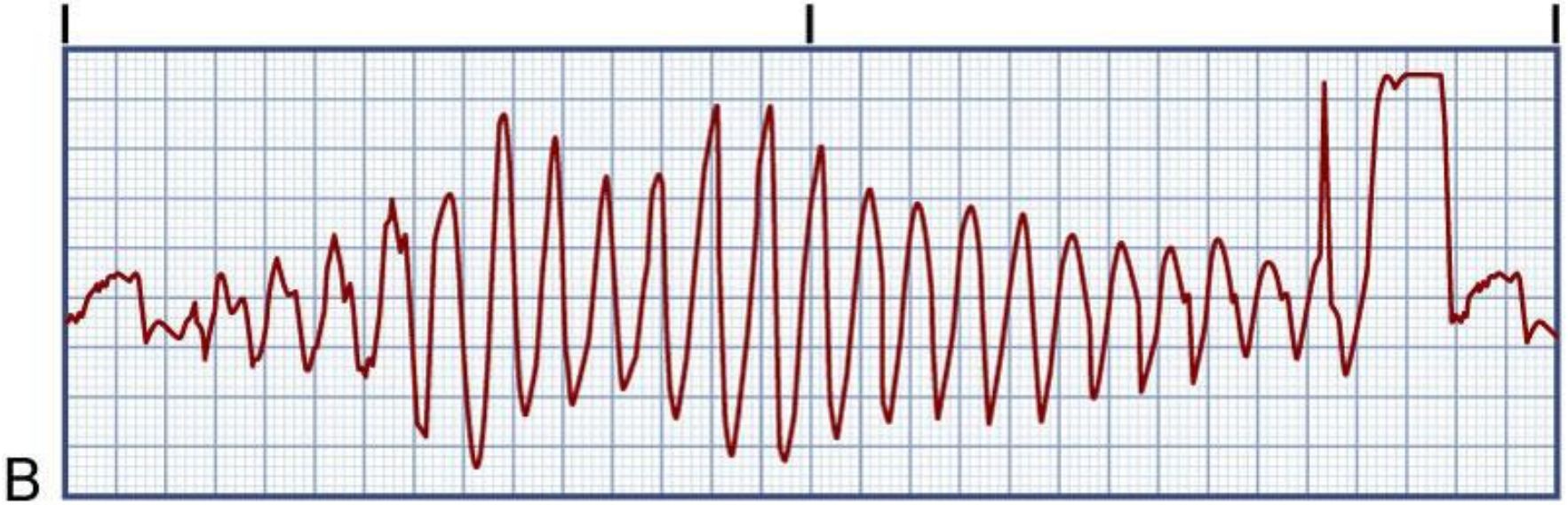
Ventricular Tachycardia



Ventricular Tachycardia

- Ectopic foci take over as pacemaker
- Monomorphic, polymorphic, sustained, and nonsustained
- Considered life-threatening because of decreased CO and the possibility of deterioration to ventricular fibrillation

Ventricular Tachycardia
Torsades de Pointes



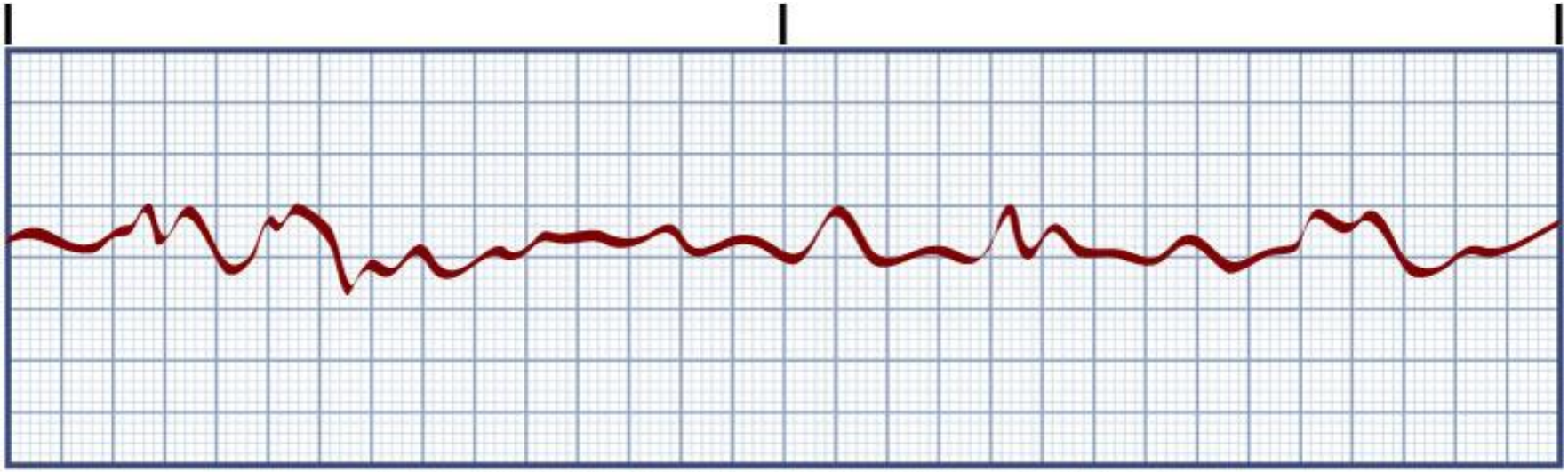
Ventricular Tachycardia

- Associated with heart disease, electrolyte imbalances, drugs, CNS disorder
- Can be stable (patient has a pulse) or unstable (pulseless)
- Sustained VT causes severe decrease in CO
  - Hypotension, pulmonary edema, decreased cerebral blood flow, cardiopulmonary arrest

Ventricular Tachycardia

- Precipitating causes must be identified and treated (e.g., hypoxia)
- VT with pulse (stable) treated with antidysrhythmics or cardioversion
- Pulseless VT treated with CPR and rapid defibrillation

Ventricular Fibrillation



Ventricular Fibrillation

- Associated with MI, ischemia, disease states, procedures
- Unresponsive, pulseless, and apneic
- If not treated rapidly, death will result
- Treat with immediate CPR and ACLS
  - Defibrillation
  - Drug therapy (epinephrine, vasopressin)

Asystole

- Represents total absence of ventricular electrical activity
- No ventricular contraction
- Patient unresponsive, pulseless, apneic
- Must assess in more than one lead

Asystole

- Usually result of advanced cardiac disease, severe conduction disturbance, or end-stage HF
- Treat with immediate CPR and ACLS measures
  - Epinephrine and/or vasopressin
  - Intubation
- Poor prognosis

Pulseless Electrical Activity

- Electrical activity can be observed on the ECG, but no mechanical activity of the ventricles is evident, and the patient has no pulse
- Prognosis is poor unless underlying cause quickly identified and treated

Pulseless Electrical Activity

Hs and Ts Pneumonic

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hyper-/hypokalemia
- Hypoglycemia
- Hypothermia

- Toxins
- Tamponade (cardiac)
- Thrombosis (MI and pulmonary)
- Tension pneumothorax
- Trauma

Pulseless Electrical Activity

- Treatment
  - CPR followed by intubation and IV epinephrine
  - Treatment is directed toward correction of the underlying cause

- **END**