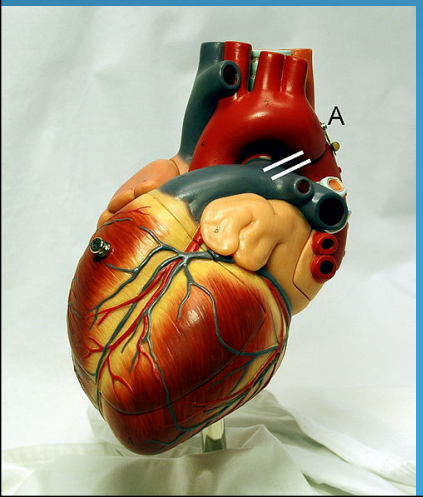
Cardiovascular System Block

Cardiac Arrhythmias

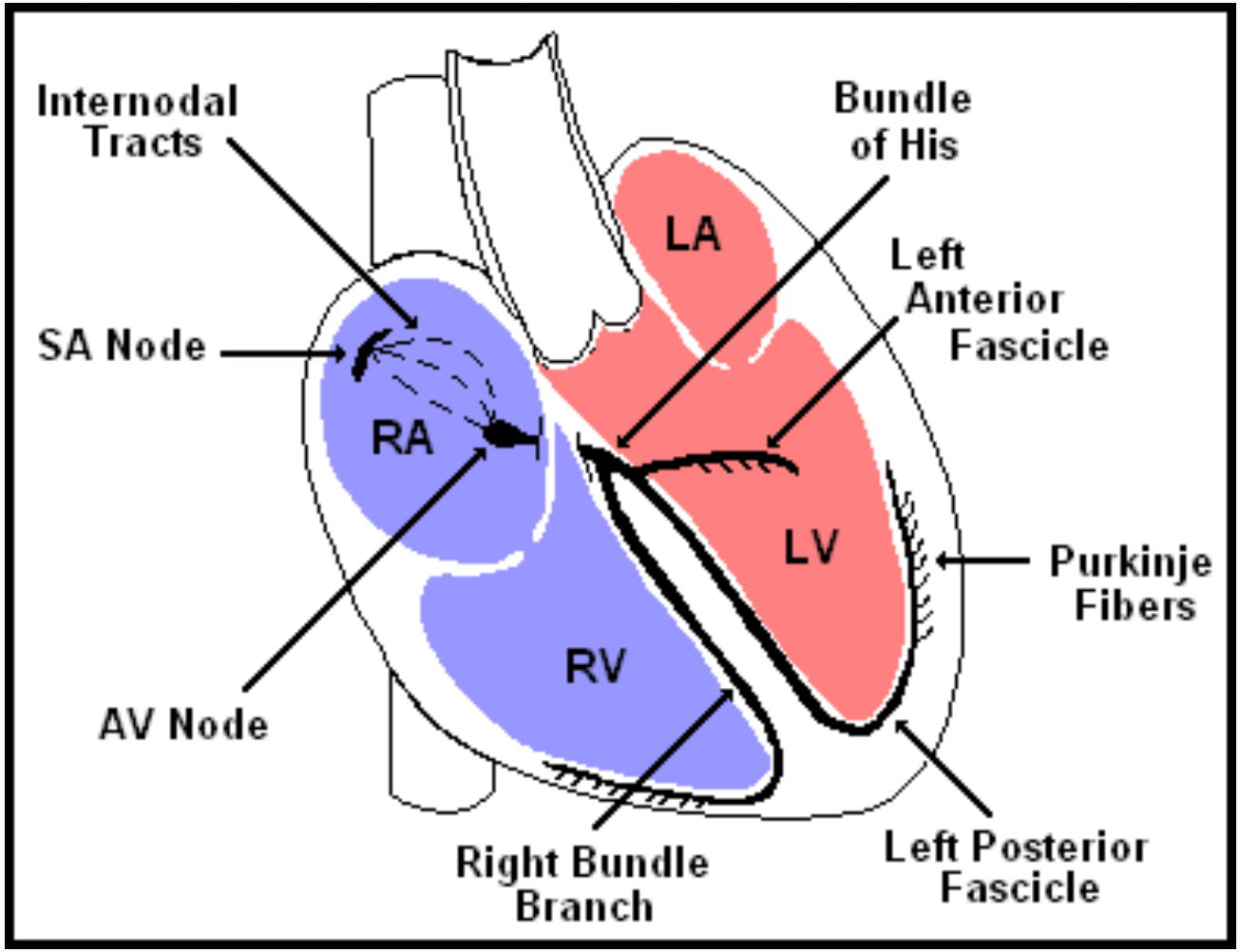
(Physiology)



Lecture Objectives

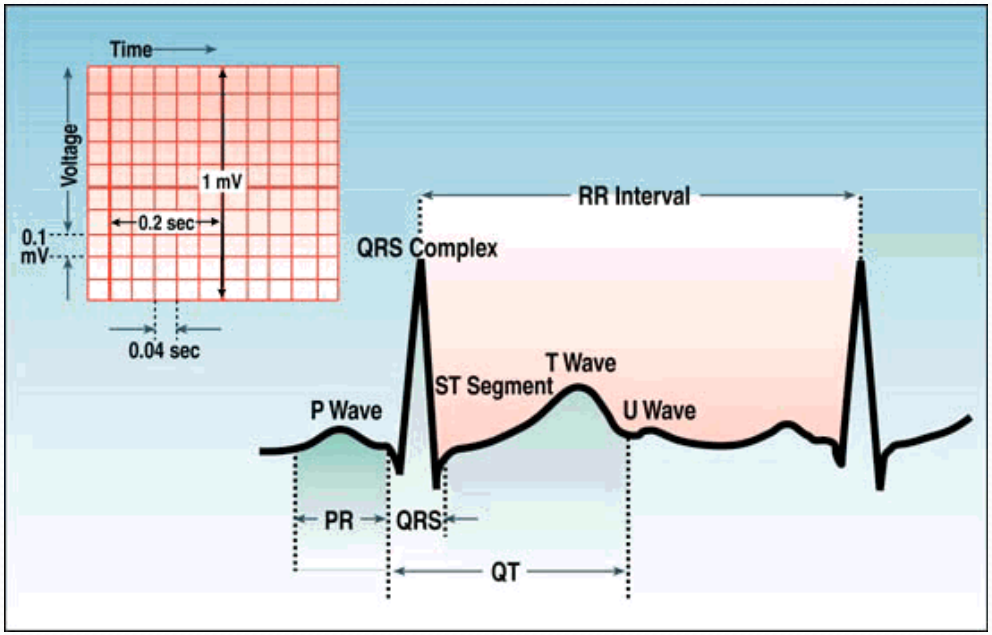
- Describe sinus arrhythmias
- Describe the main pathophysiological causes of cardiac arrhythmias
- Explain the mechanism of cardiac block
- Explain the origin of an ectopic foci
- Enumerate the common arrhythmias and describe the basic ECG changes

The Normal Conduction System

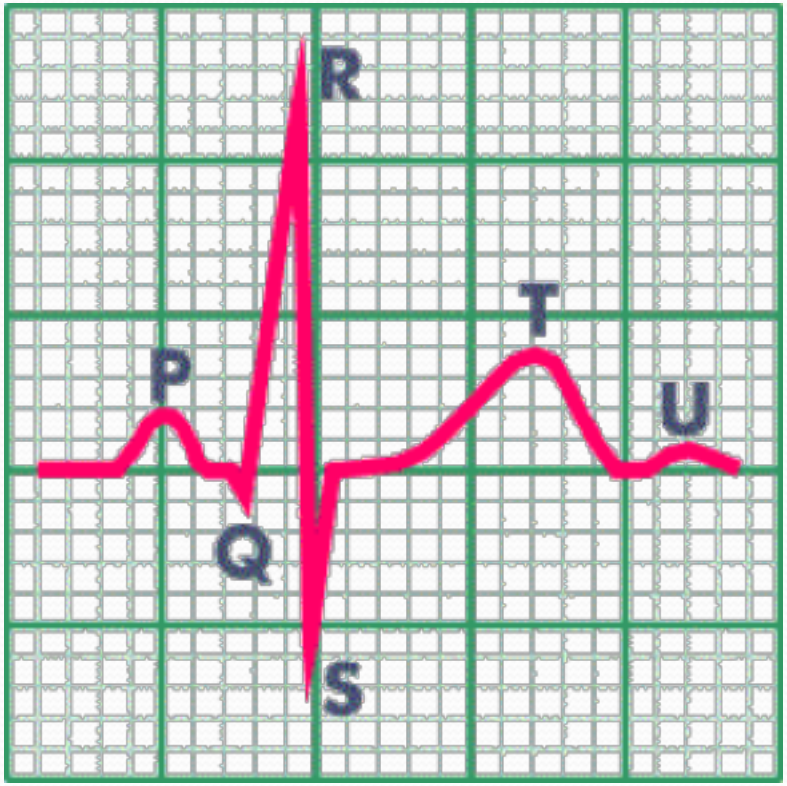


Rhythm

- Sinus
  - Originating from SA node
  - P wave before every QRS
  - P wave in same direction as QRS



Waveforms



Interpretation

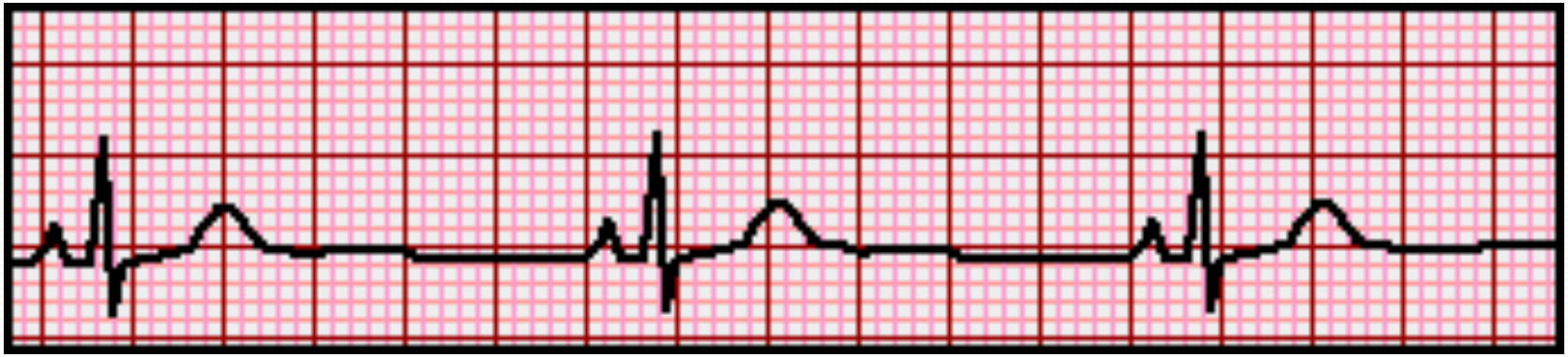
- Develop a systematic approach to reading EKGs and use it every time
- The system we will practice is:
  - Rate
  - Rhythm (including intervals and blocks)
  - Axis
  - Hypertrophy
  - Ischemia

Rate

- Rule of 300- Divide 300 by the number of boxes between each QRS = rate

Number of big boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50

What is the heart rate?



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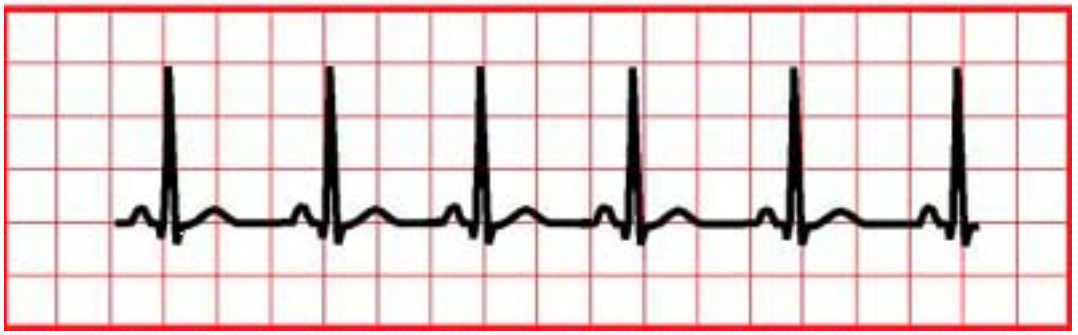
(300 / 6) = 50 bpm

Rate

- HR of 60-100 per minute is normal
- HR > 100 = tachycardia
- HR < 60 = bradycardia

Normal Sinus Rhythm

- Regular
- Single p-wave precedes every QRS complex
- P-R interval is constant and within normal range
- P-P interval is constant



COMMON ARRHYTHMIAS

Location	Bradyarrythmia	Tacharrythmia
SA node	Sinus Bradycardia	Sinus tachycardia
	Sick Sinus Syndrome	
Atria		Atrial Premature Beats
		Atrial Flutter
		Atrial Fibrillation
		Paroxysmal SVT
		Multifocal Atrial Tachycardia
AV node	Conduction Blocks (1,2 and 3)	
	Jxal escape rhythm	
Ventricles	Ventricular escape rhytm	Ventricular premature Beats
		VT
		Torsades de pointes
		Ventricular Fibrillation

Differential Diagnosis of Tachycardia

Tachycardia	Narrow Complex	Wide Complex
Regular	ST SVT Atrial flutter	ST w/ aberrancy SVT w/ aberrancy VT
Irregular	A-fib A-flutter w/ variable conduction MAT	A-fib w/ aberrancy A-fib w/ WPW VT

Causes of Cardiac Arrhythmias

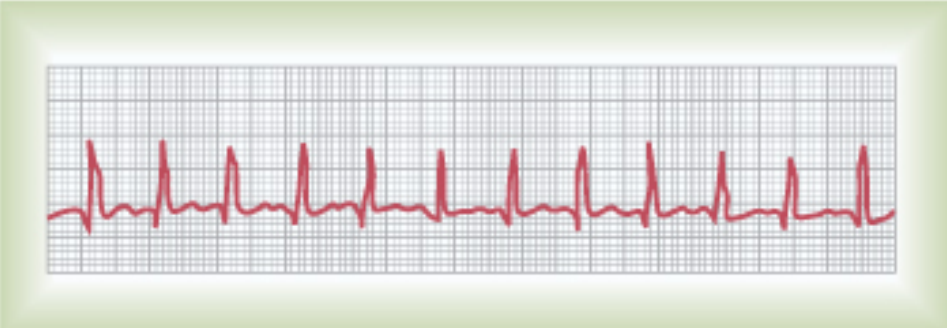
1. Abnormal rhythmicity of the pacemaker
2. Shift of the pacemaker from the sinus node to another place in the heart
3. Blocks at different points during the spread of the impulse through the heart
4. Abnormal pathways of impulse transmission through the heart
5. Spontaneous generation of spurious impulses in almost any part of the heart

Causes of Cardiac Arrhythmias

- Rate above or below normal
- Regular or irregular rhythm
- Narrow or broad QRS complex
- Relation to P waves

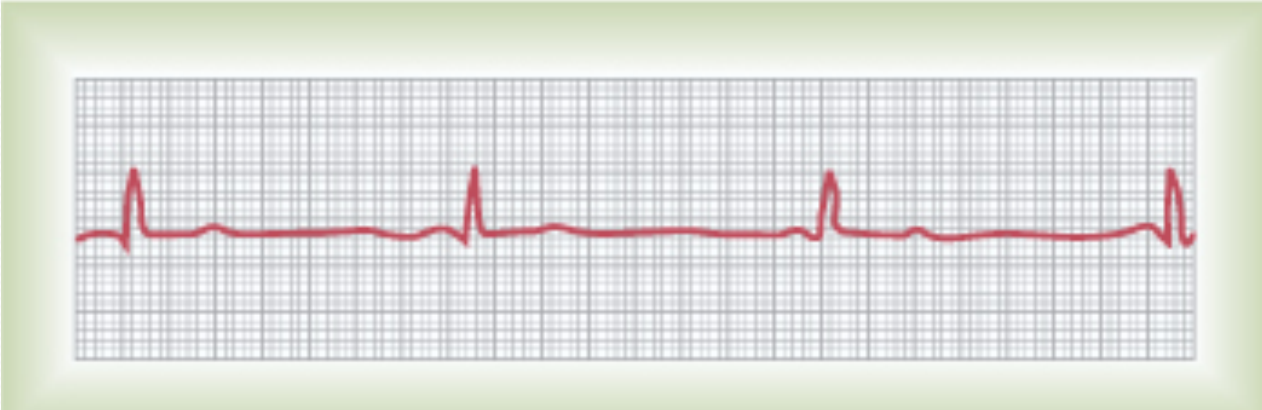
Abnormal Sinus Rhythm

- Tachycardia: an increase in the heart rate
  - Heart rate > 100 beats per minute
  - Causes:
    - Increased body temperature
    - Sympathetic stimulation
    - Drugs: digitalis
    - Inspiration



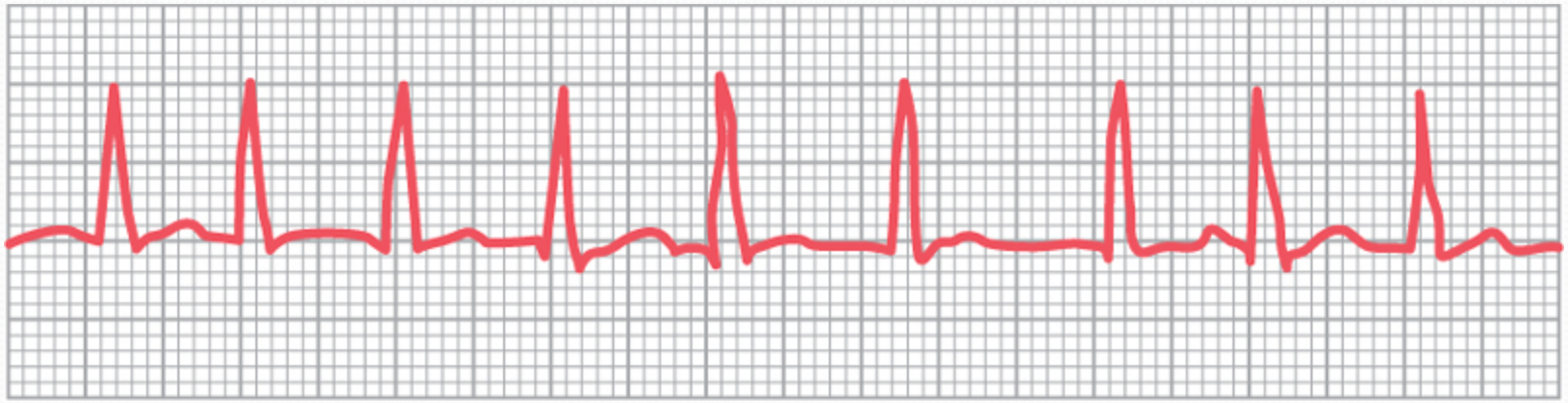
Abnormal Sinus Rhythm

- Bradycardia:
  - Slow heart rate < 60 beats per minute
  - Causes:
    - Parasympathetic stimulation
    - Expiration



Abnormal Cardiac Rhythms that Result from Impulse Conduction Block

- **Sinoatrial Block**
  - Blockasde of the S-A node impulse before entering atrial muscle
- Cessation of P wave
- Causes:
  - Ischemia of the A-V node
  - Compression of the A-V node by scar formation
  - Inflammation of the A-V node
  - Strong vagal stimulation



Abnormal Cardiac Rhythms that Result from Impulse Conduction Block

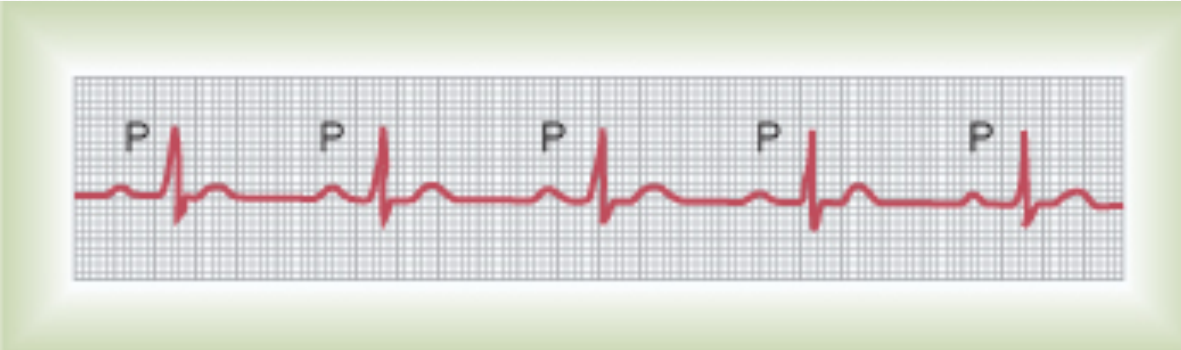
- **A-V Block**
  - When impulse from the S-A node is blocked
  - Causes:
    - Ischemia of the A-V node
    - Compression of the A-V node by scar formation
    - Inflammation of the A-V node
    - Strong vagal stimulation

Types of the A-V Block

- First degree block
- Second degree block
- Third degree block

Types of the A-V Block

- **First degree block**
  - **Prolong P-R interval (0.2 seconds)**



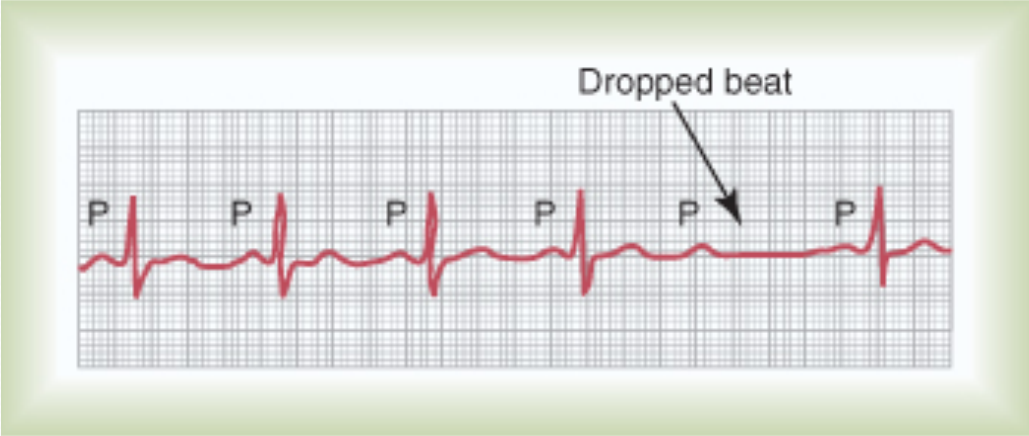
Types of the A-V block

Second Degree Block

- P-R interval > 0.25 second
- Only few impulses pass to the ventricles

  → atria beat faster than ventricles

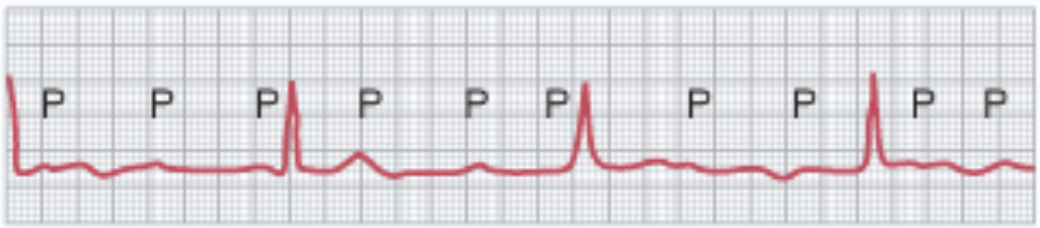
  → "dropped beat" of the ventricles



Types of the A-V block

Third degree block (complete)

- Complete dissociation of P wave and QRS waves
- Ventricle escape from the influence of S-A node
- Atrial rate is 100 beats/min
- Ventricular rate is 40 beats/min
- *Stokes-Adams Syndrome*: AV block comes and goes

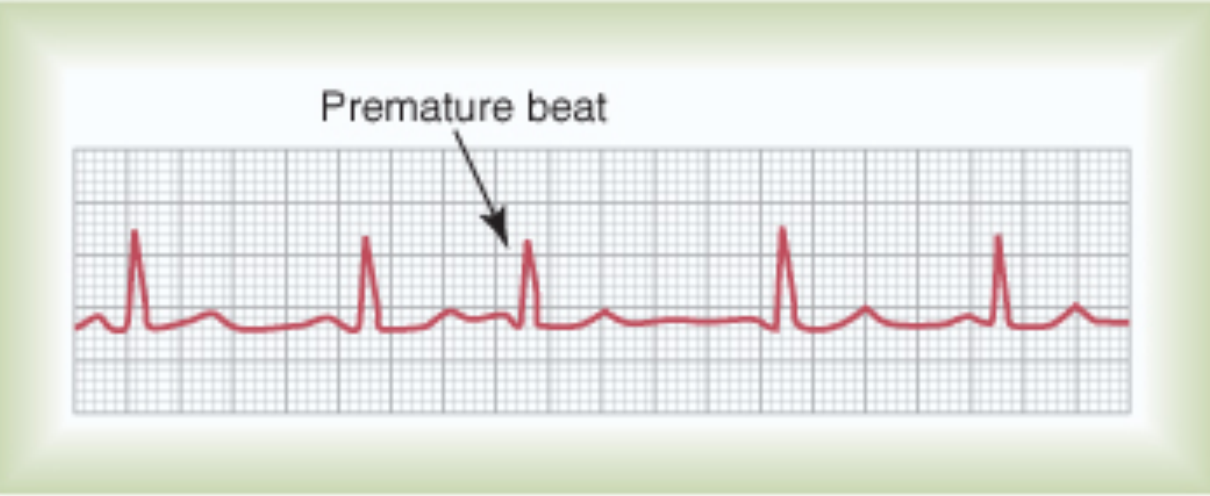


Premature contractions

- Premature contractions, *extrasystoles, or* ectopic beat result from *ectopic foci* that generate abnormal cardiac impulses (pulse deficit)
- Causes:
  - Ischemia
  - Irritation of cardiac muscle by calcified foci
  - Drugs like caffeine
- Ectopic foci can cause premature contractions that originate in:
  - The atria
  - A-V junction
  - The ventricles

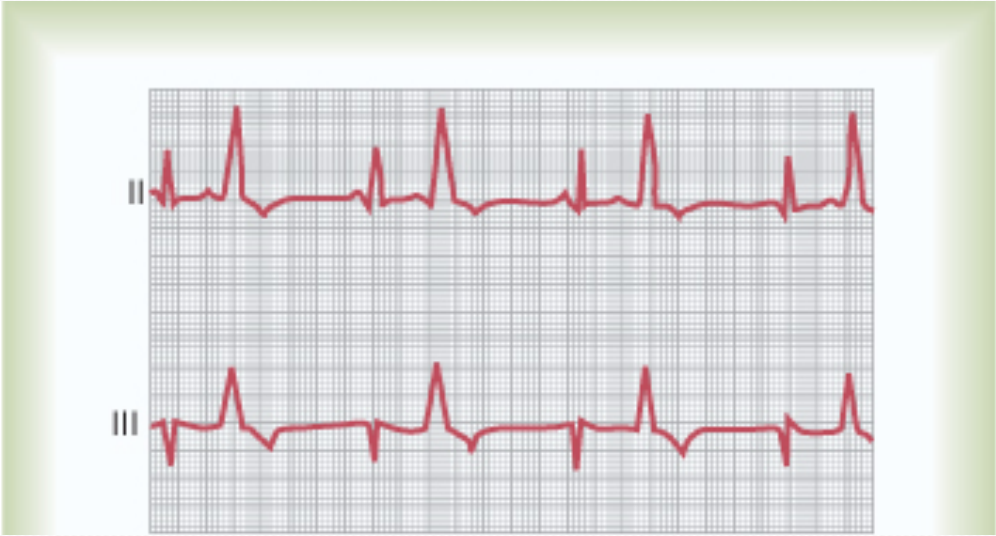
Premature Atrial Contractions

- Short P-R interval depending on how far the ectopic foci from the AV node
- Pulse deficit if there is no time for the ventricles to fill with blood
- The time between the premature contraction and the succeeding beat is increased (*Compensatory pause*)



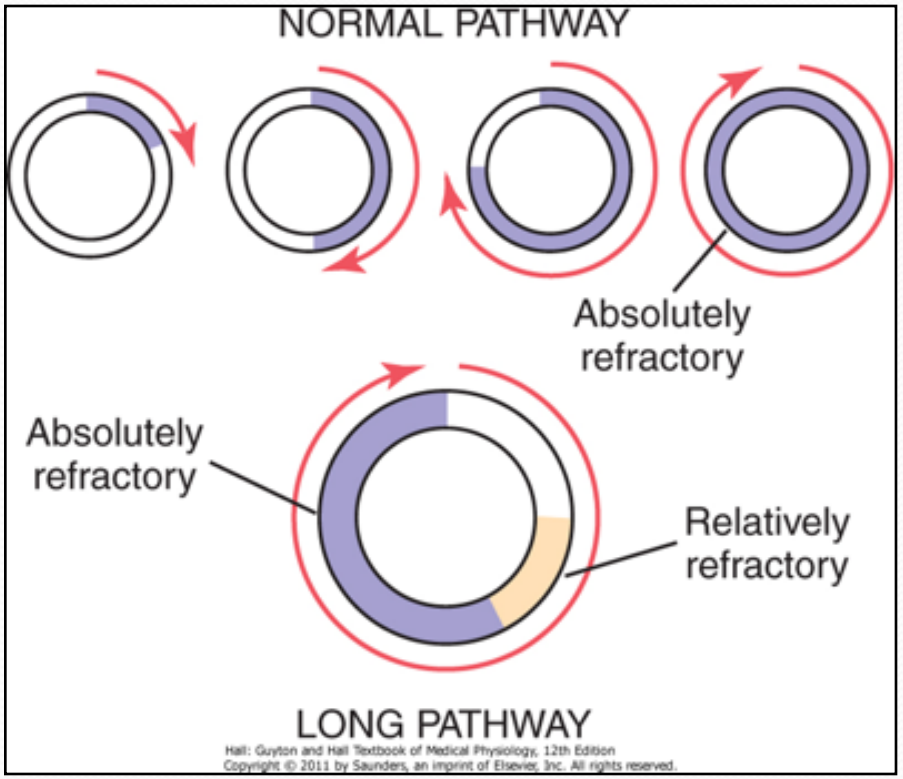
Premature Ventricular Contractions (PVCs)

- Prolong QRS complex because the impulses are carried out with myocardial fibers with slower conduction rate than Purkinje fibers
- Increase QRS complexes voltage because QRS wave from one ventricle can not neutralize the one from the other ventricle
- After PVCs, the T wave has an electrical potential of opposite polarity of that of the QRS because of the slow conduction in the myocardial fibers, the fibers that depolarizes first will repolarize first
- Causes: drugs, caffeine, smoking, lack of sleep, emotional irritations



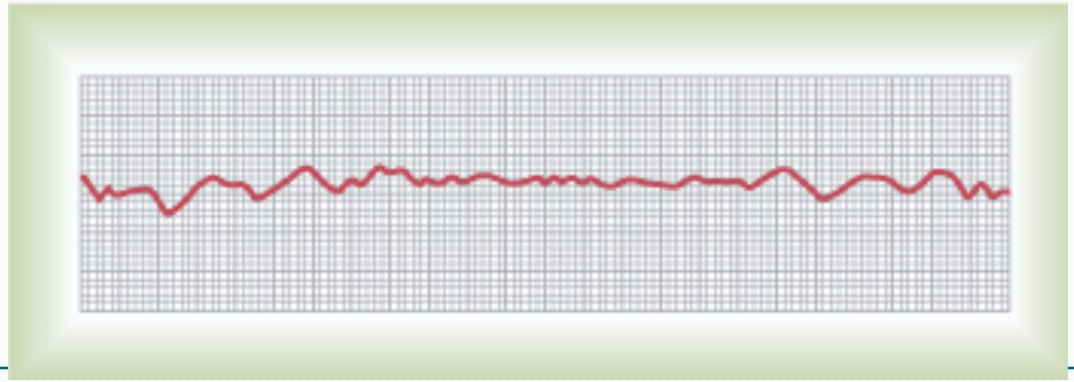
Ventricular Fibrillation

- The most serious of all arhythmias
- Cause: impulses stimulate one part of the ventricles, then another, then itself. Many part contracts at the same time while other parts relax *(Circus movement)*



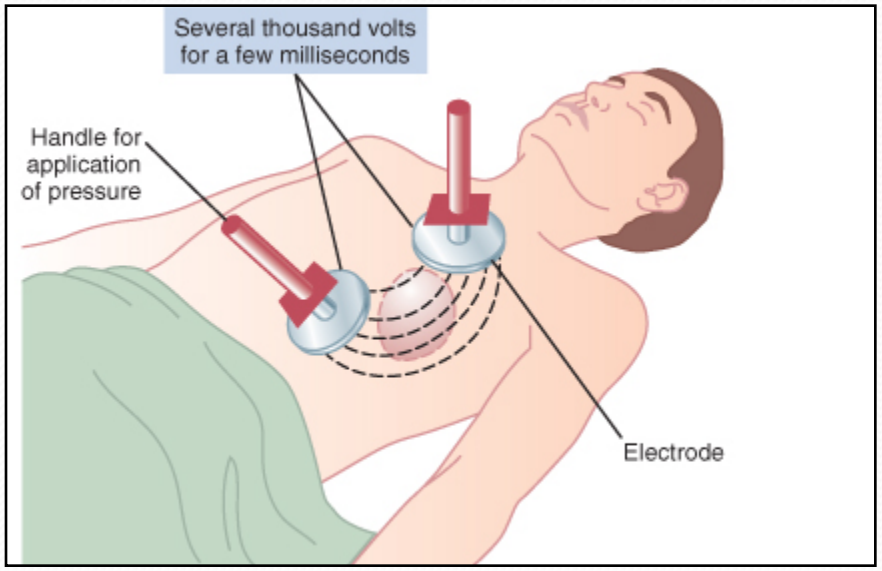
Ventricular Fibrillation

- Causes: sudden electrical shock, ischemia
  - Tachycardia
  - Irregular rhythm
  - Broad QRS complex
  - No P wave



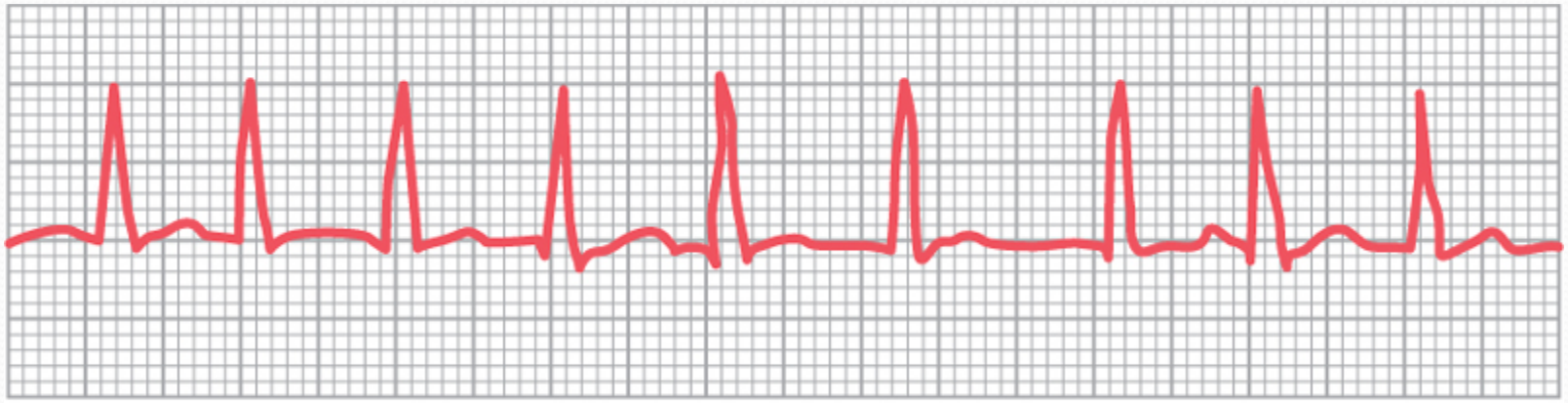
Ventricular Fibrillation

- *Treatment : DC shock*



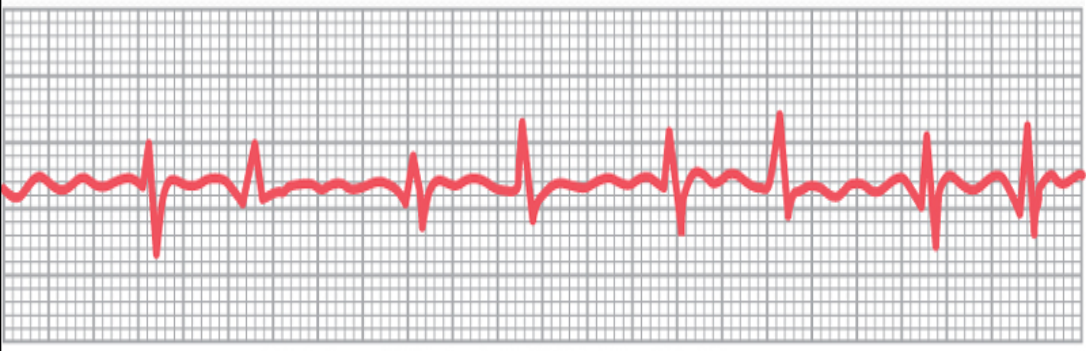
Atrial Fibrillation

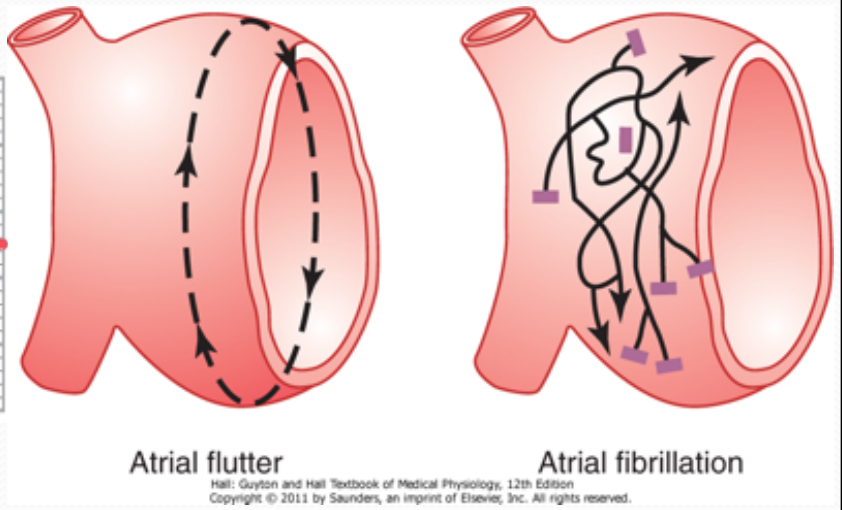
- Same mechanism as ventricular fibrillation. It can occur only in atria without affecting the ventricles
- It occurs more frequently in patients with *enlarged heart*
- The atria do not pump if they are fibrillating
- The efficiency of ventricular filling is decreased 20 to 30%
- No P wave, or high frequency of low voltage P wave
- Treatment: DC shock



Atrial Flutter

- A single large wave travels around and around in the atria
- The atria contracts at high rate (250 beats/min)
- Because one area of the atria is contracted and another one is relaxed, the amount of blood pumped by the atria is slight
- The refractory period of the AV node causes 2-3 beats of atria for one single ventricular beat 2:1 or 2:3 rhythm



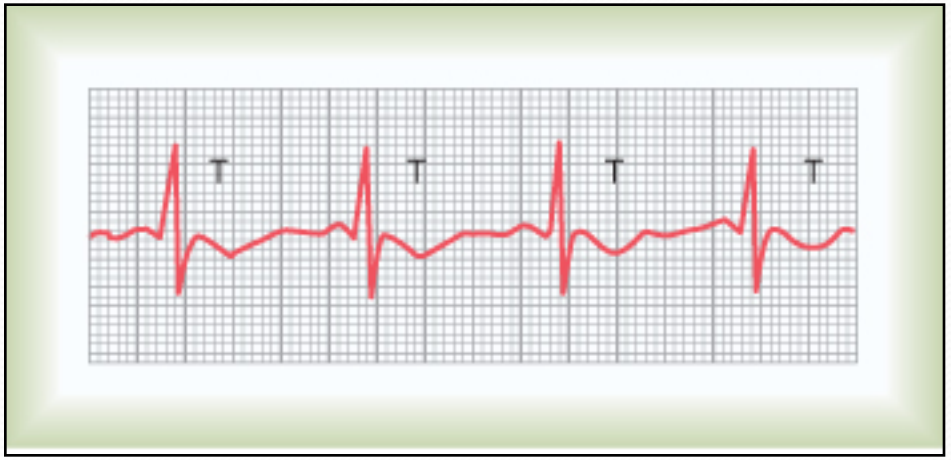


Ischemia and the ECG

- One of the common uses of the ECG is in acute assessment of chest pain
- Cause: restriction of blood flow to the myocardium, either:
  - Reversible: angina pectoris
  - Irreversible: myocardial infarction
- ***Ischemia → injury → infarction***

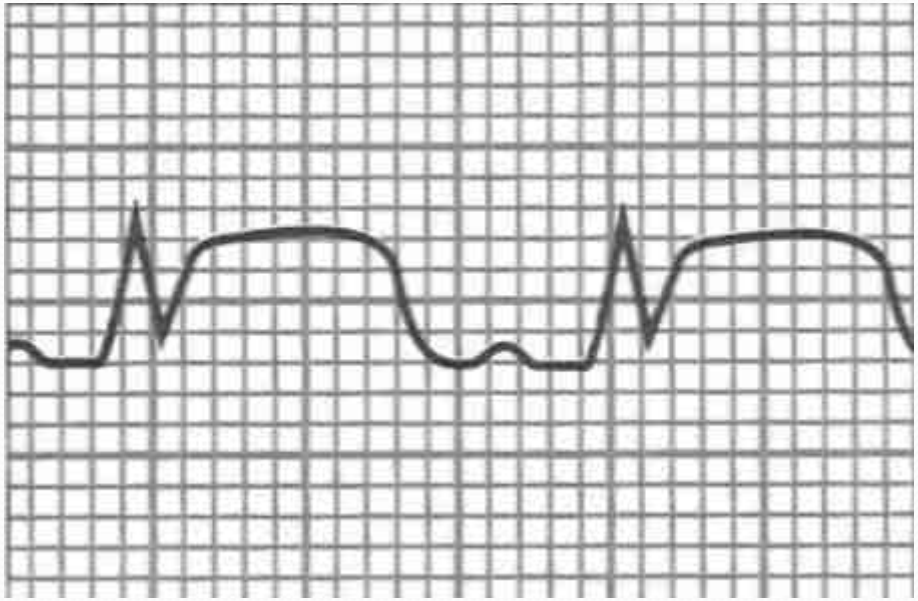
Reversible ischemia

- *Inverted T wave*
- *ST segment depression*



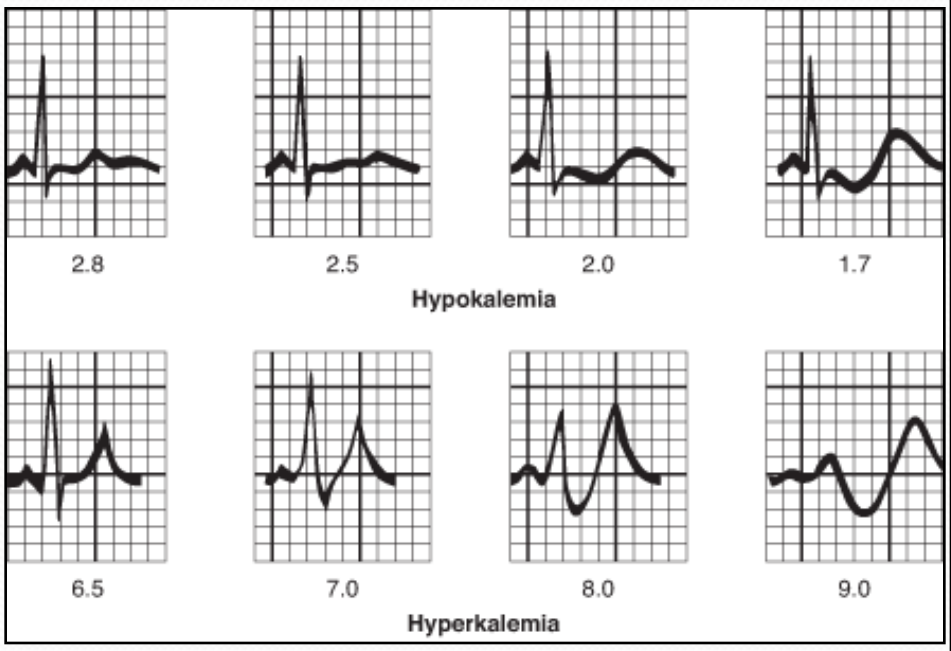
Myocardial Infarction

- Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue
  - *ST segment elevation*
  - *Deep Q wave*



Potassium and the ECG

- Hypokalemia:
  - flat T wave
- Hyperkalemia:
  - Tall peaked T wave



For further readings and diagrams:

Textbook of Medical Physiology by Guyton & Hall

Chapter 10 (Cardiac Arrhythmias and their Electrocardiographic Interpretation)