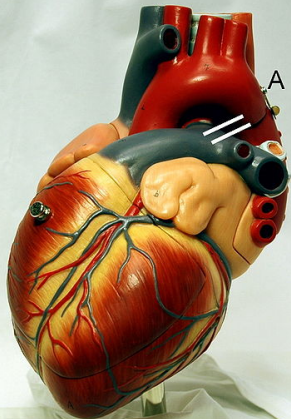


Cardiovascular System Block Cardiac Arrhythmias (Physiology)

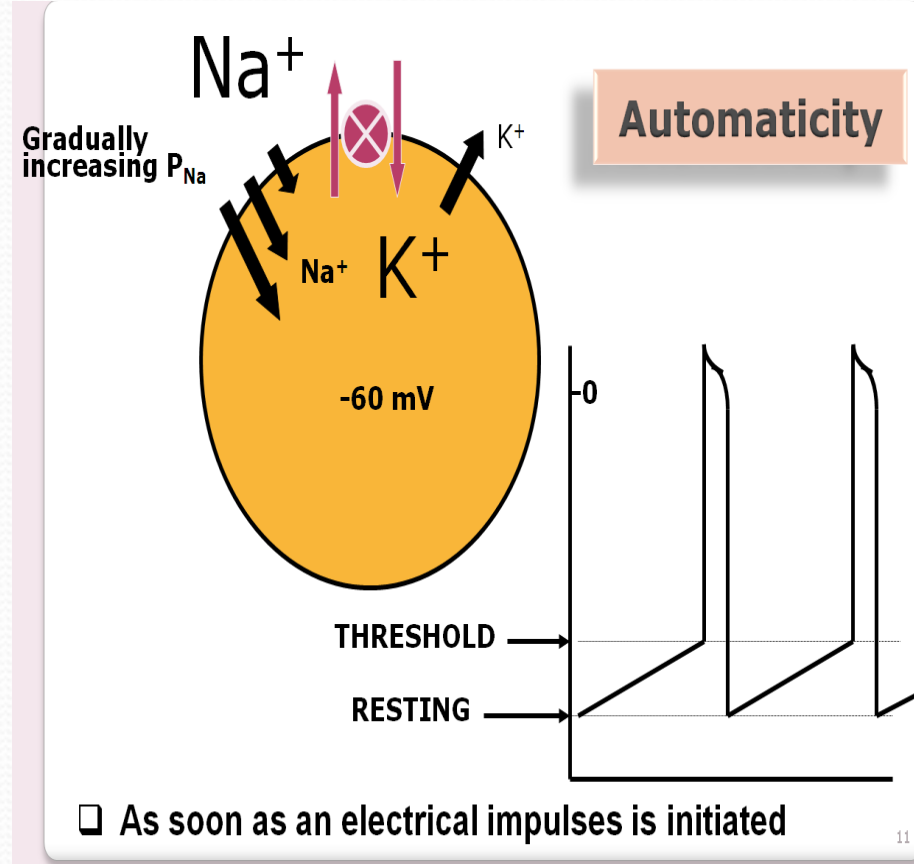
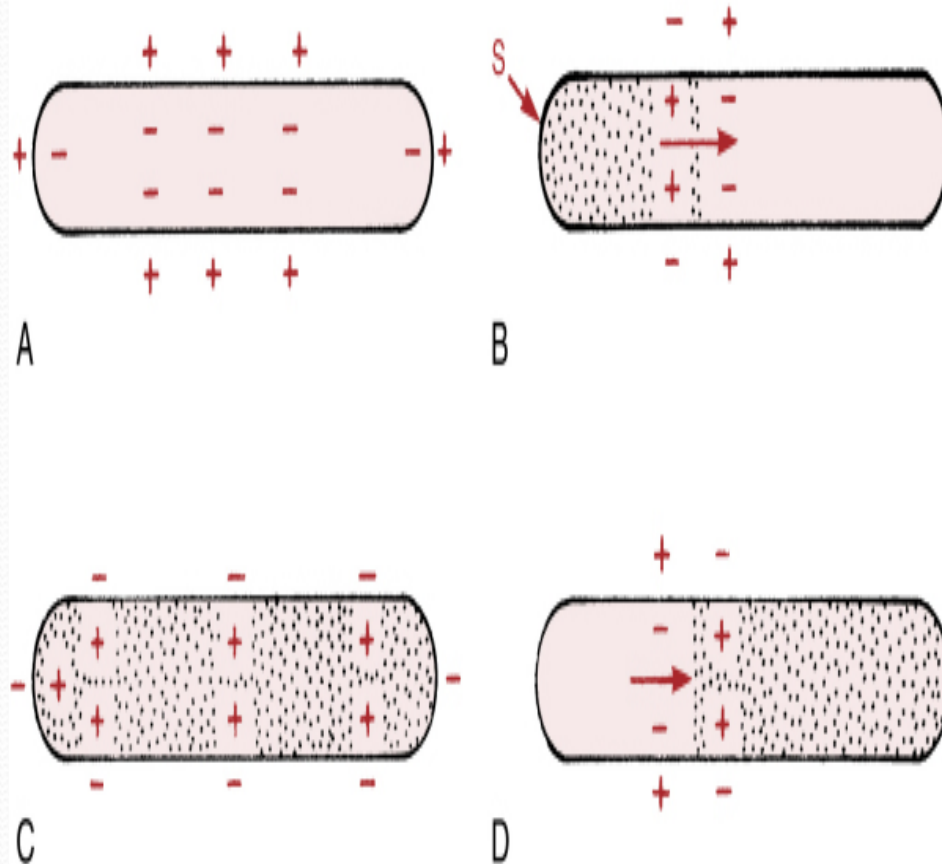
Ahmad Hersi

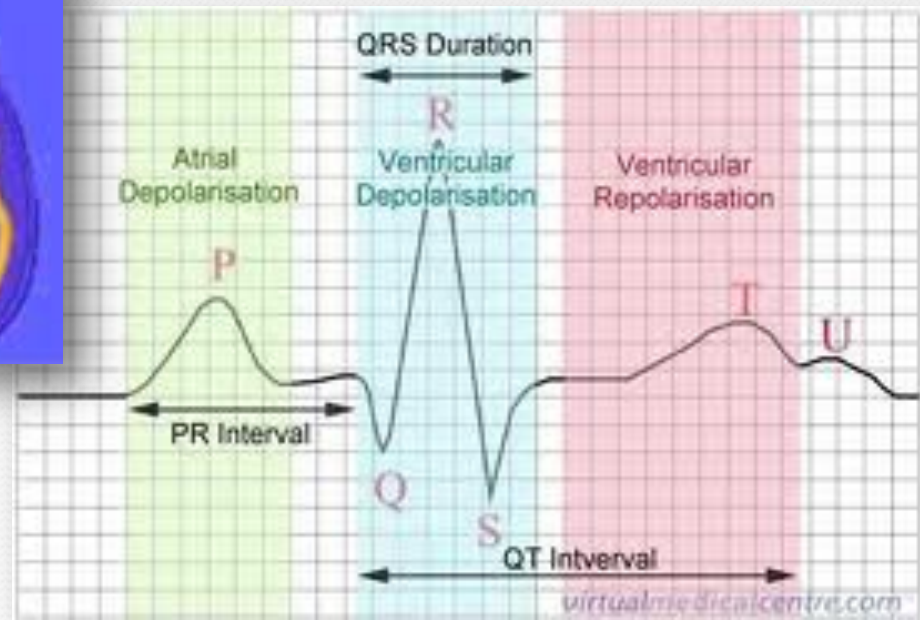


Lecture Objectives

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

Depolarization and Repolarization



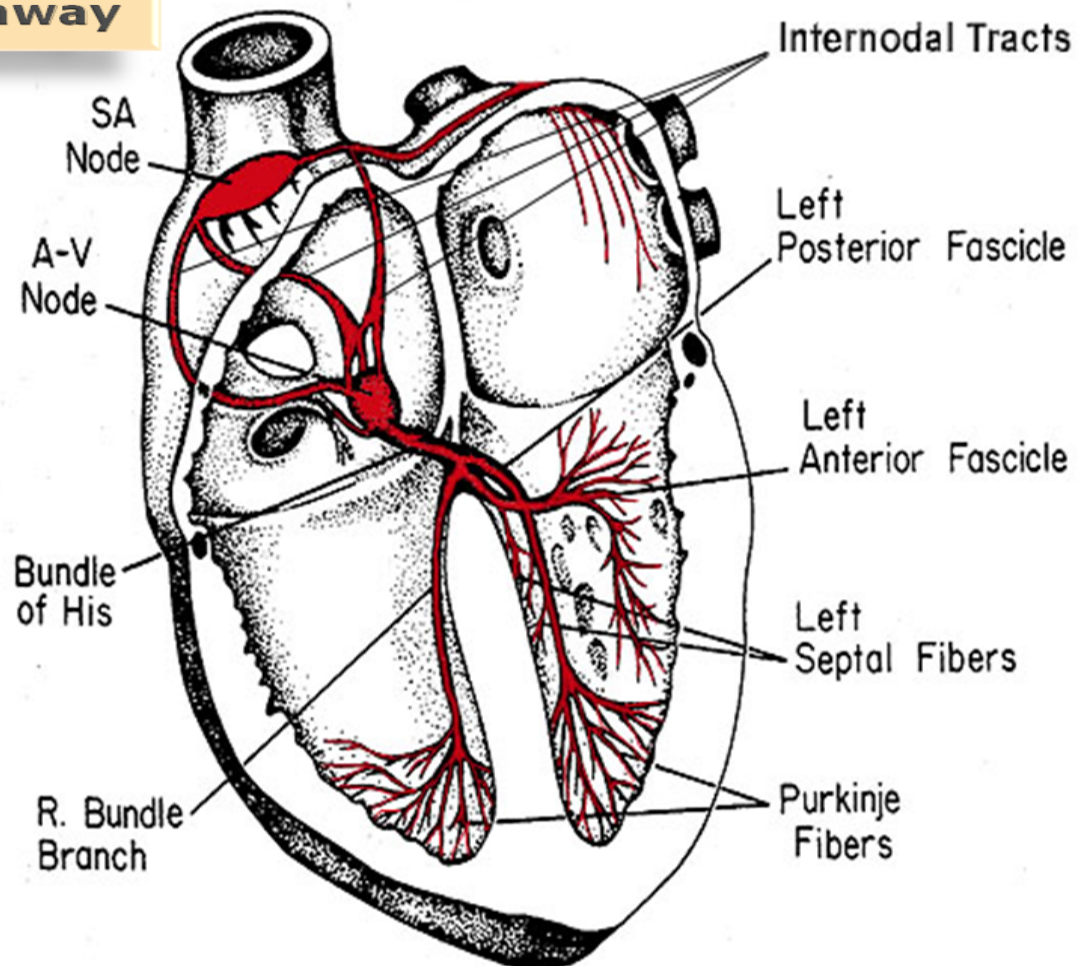


Electrical Conduction

The conduction system

Electrical Conduction Pathway

- **Sinoatrial (SA) node**
- **Internodal & Interatrial pathways**
 - Anterior internodal tract
 - Middle internodal tract (Wenckebach's tract)
 - Posterior internodal tract (Thorel's tract)
- **Atrioventricular (AV) node**
- **AV bundle (bundle of His)**
- **Rt & Lt bundle branches**
- **Purkinje fibers**



Intrinsic Firing Rates

Three potential areas capable of beginning cardiac conduction

❑ SA node:

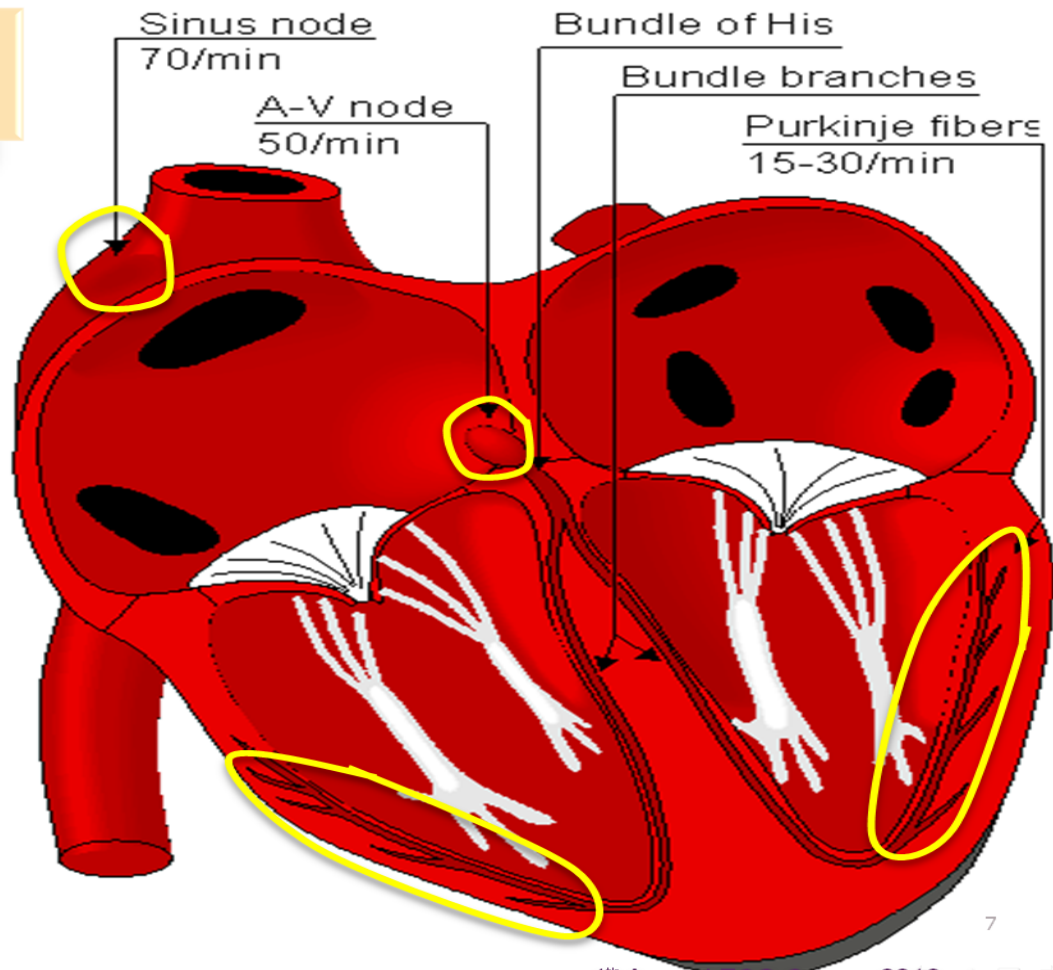
- Cardiac pacemaker
- Paces at a rate of 60–100 bpm
- Average of 70 bpm

❑ AV node:

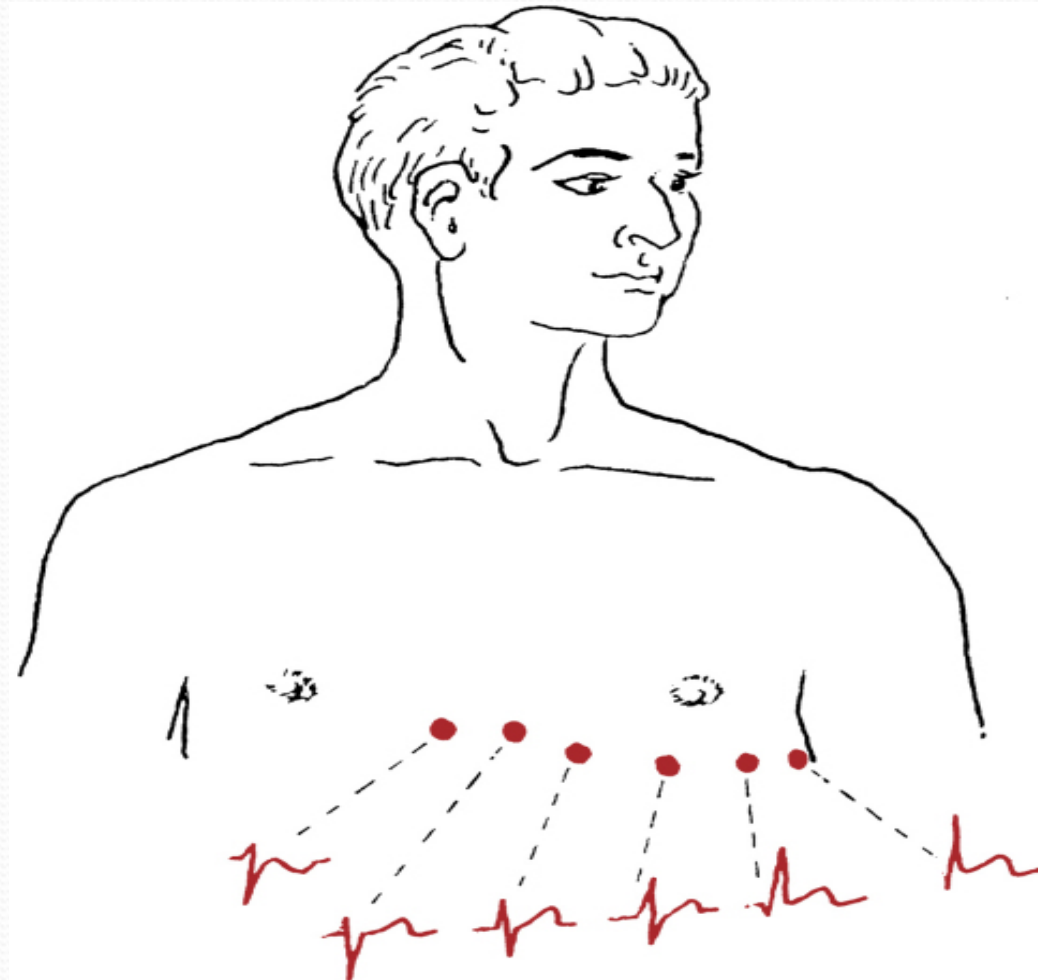
- 45-60 bpm

❑ Purkinje:

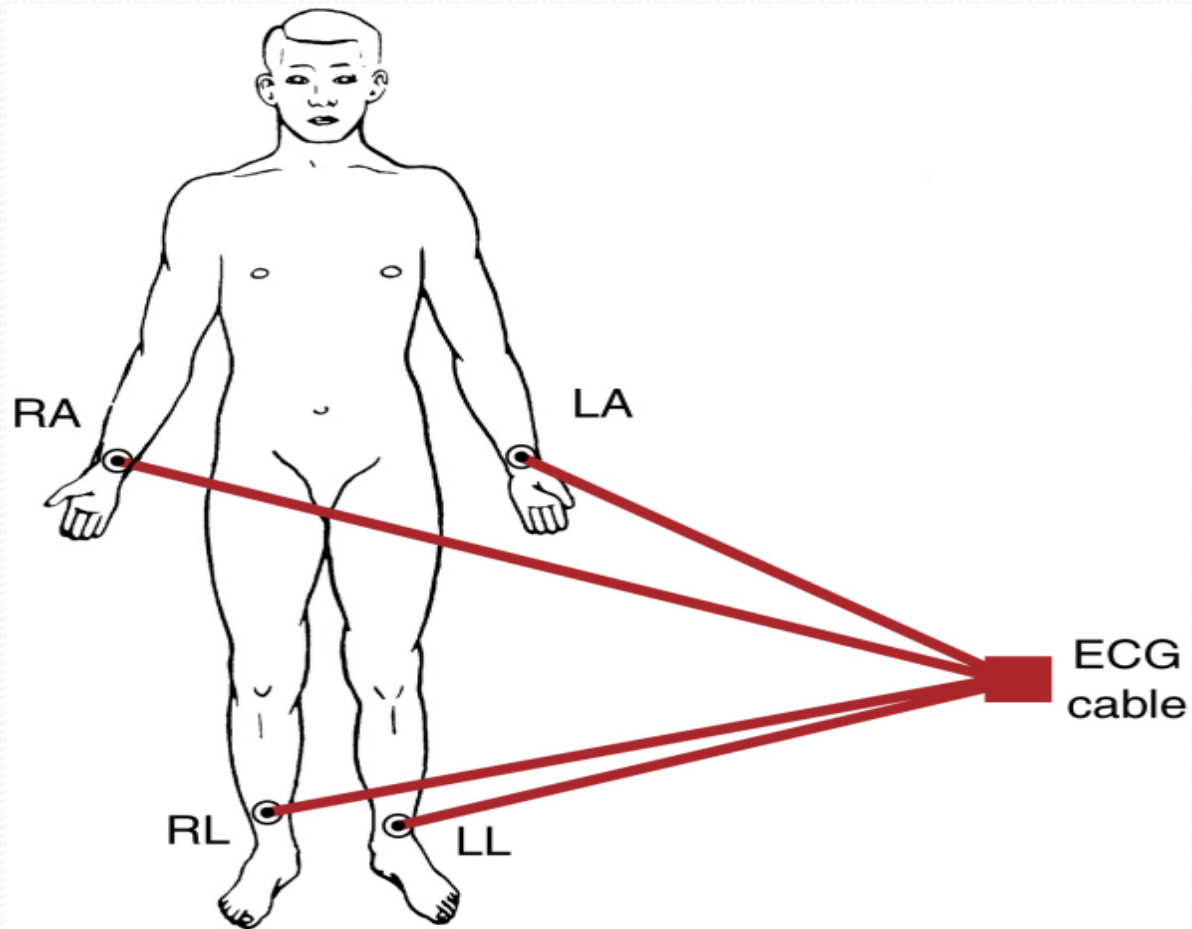
- 15-45 bpm



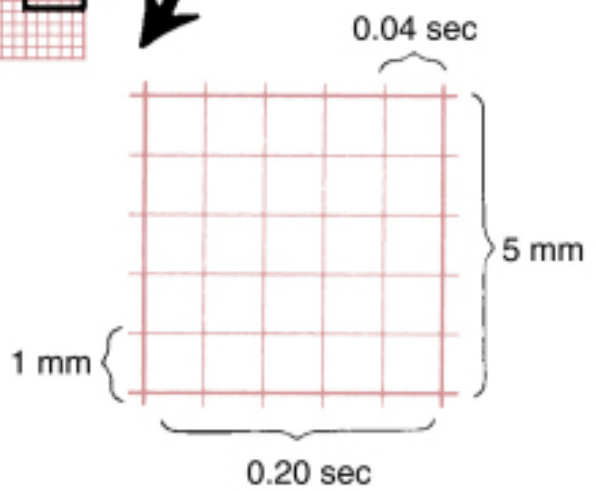
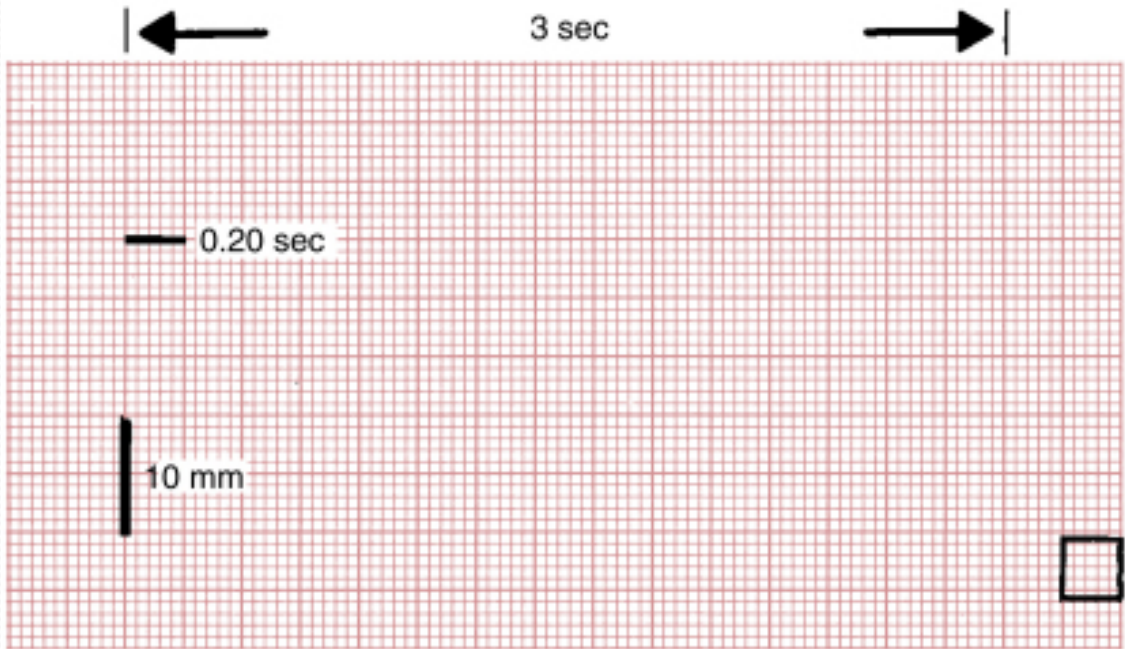
Chest leads



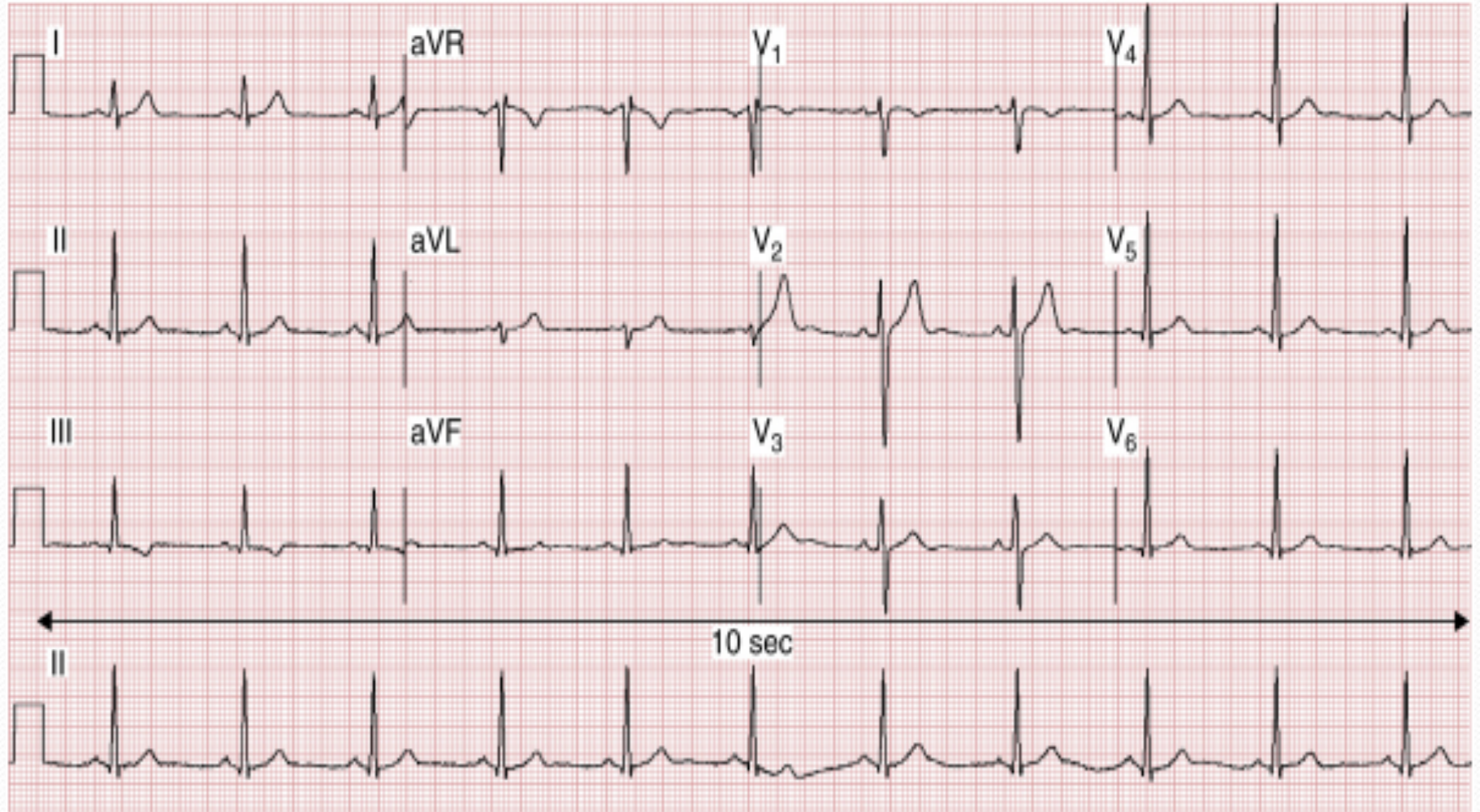
Limb leads



ECG Graph Paper



12 Leads EKG



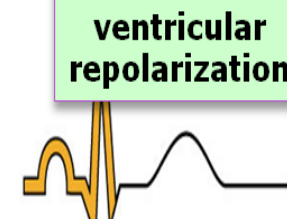
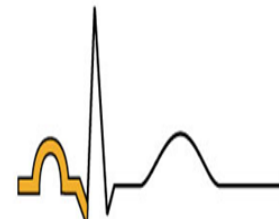
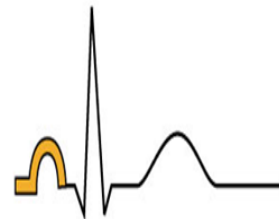
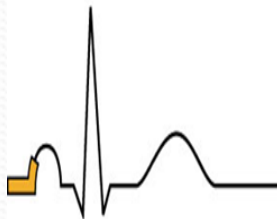
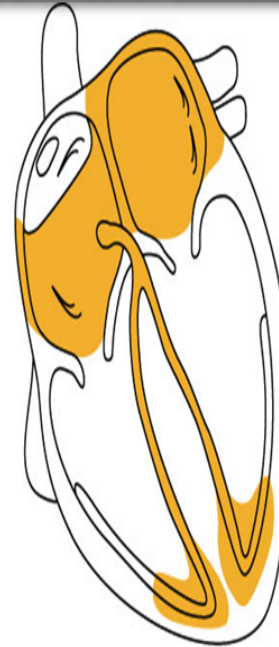
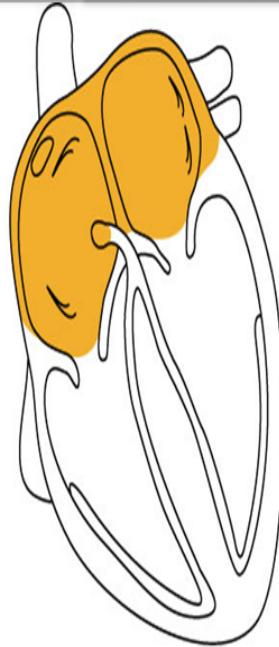
Sequence of cardiac excitation

SA node generates impulse;
atrial excitation begins

Impulse delayed
at AV node

Impulse passes to heart apex;
ventricular excitation begins

Ventricular
excitation
complete



P wave: Atrial
depolarization

QRS complex: ventricular
depolarization

T wave:
ventricular
repolarization

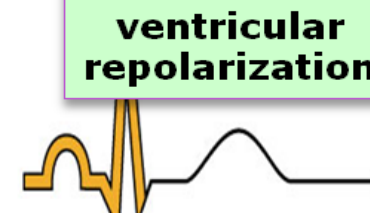
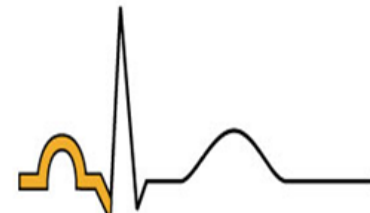
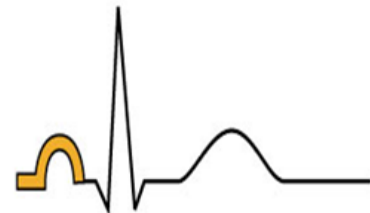
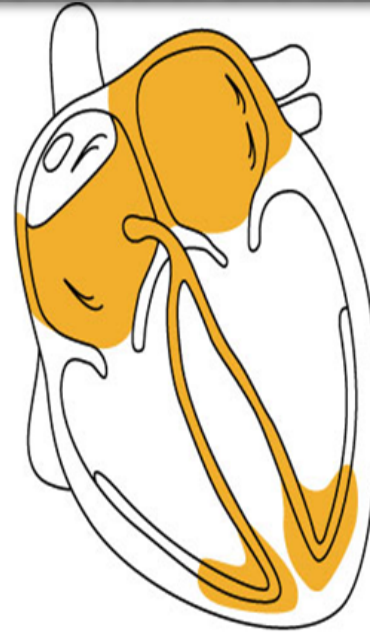
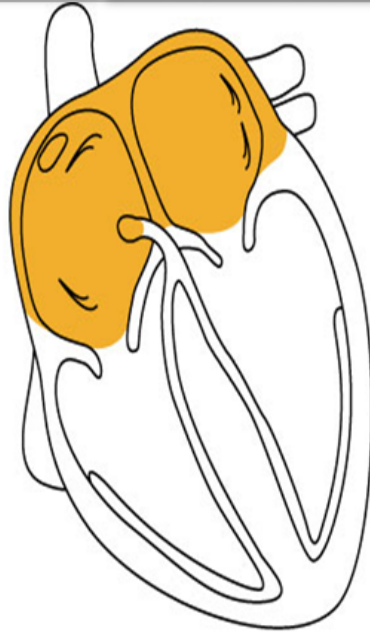
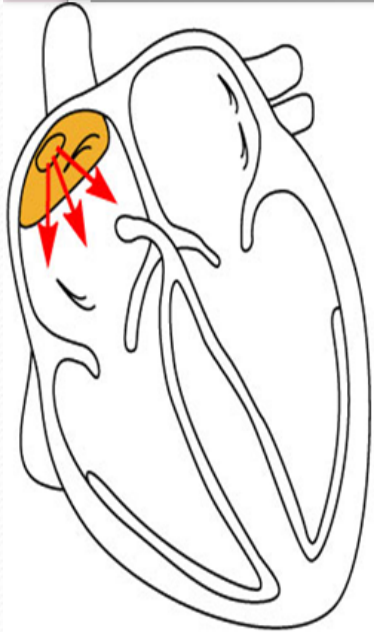
Sequence of cardiac excitation

SA node generates impulse;
atrial excitation begins

Impulse delayed
at AV node

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ventricular excitation begins

Ventricular
excitation
complete



**P wave: Atrial
depolarization**

**QRS complex: ventricular
depolarization**

**T wave:
ventricular
repolarization**

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



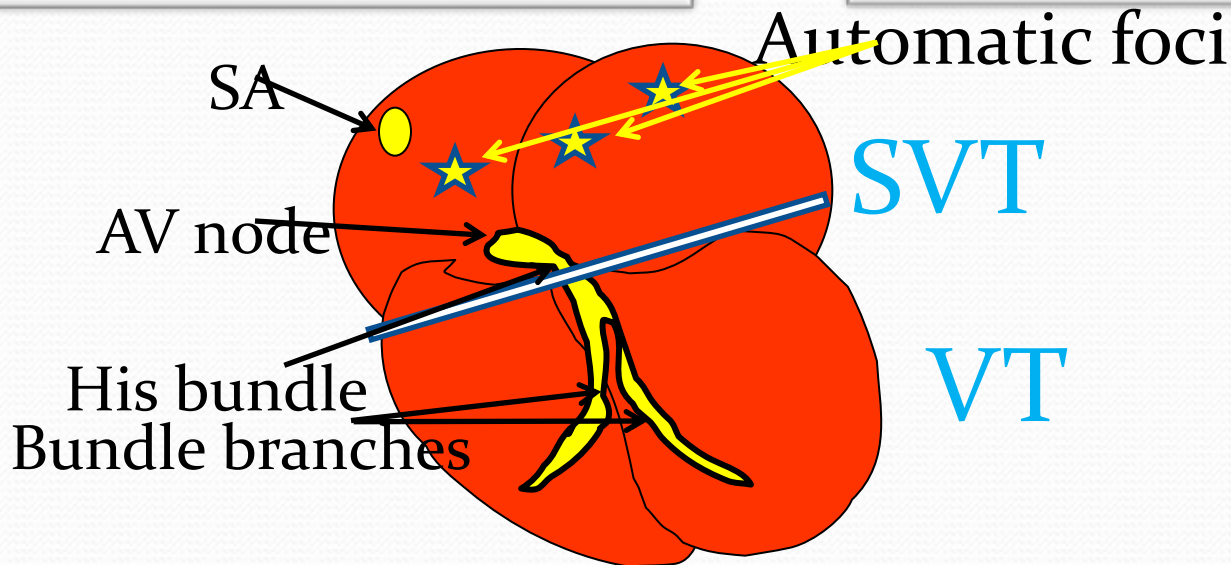
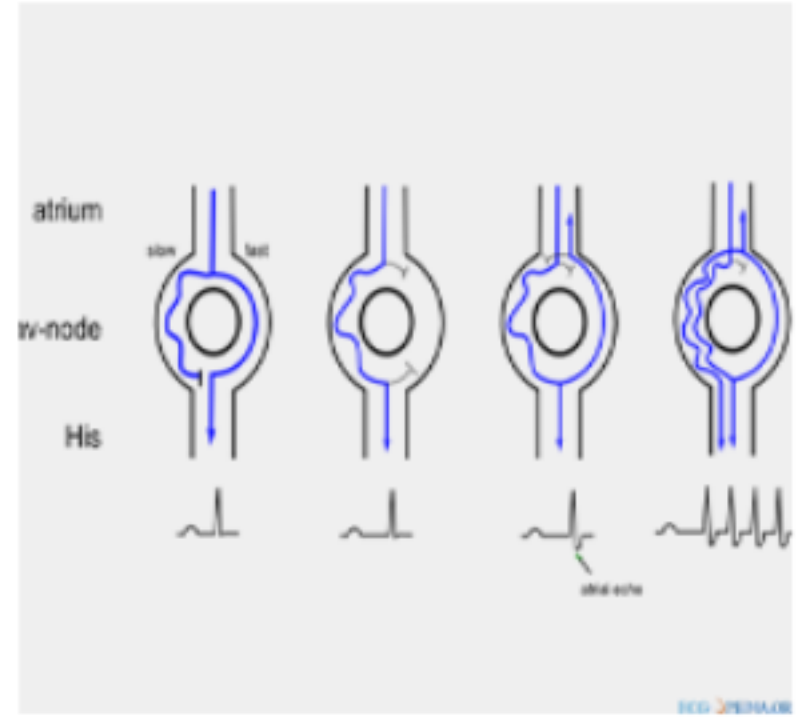
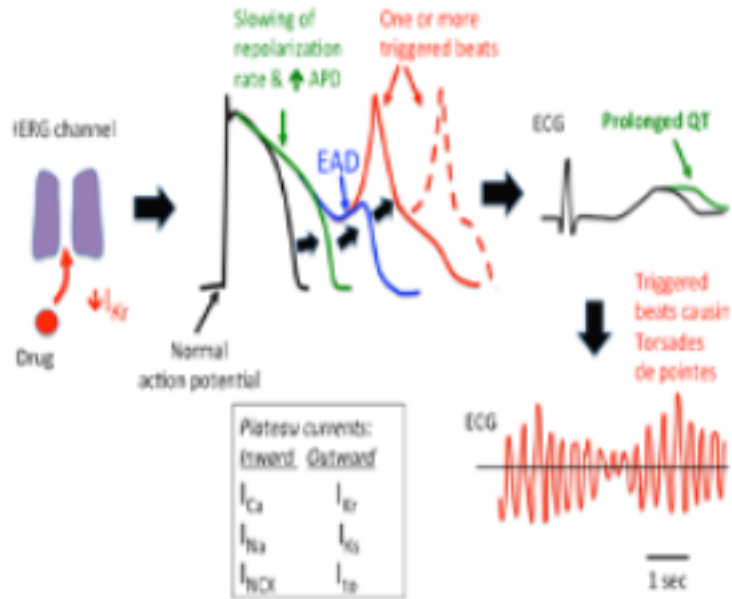
Rate

- **Normal sinus rate:** (60-100).
- **Tachycardia:** >100.
- **Bradycardia:** <60.

Causes/Mechanisms 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 in the spread of impulse through the heart
4. Triger
5. Reentry

EAD's - Mechanism

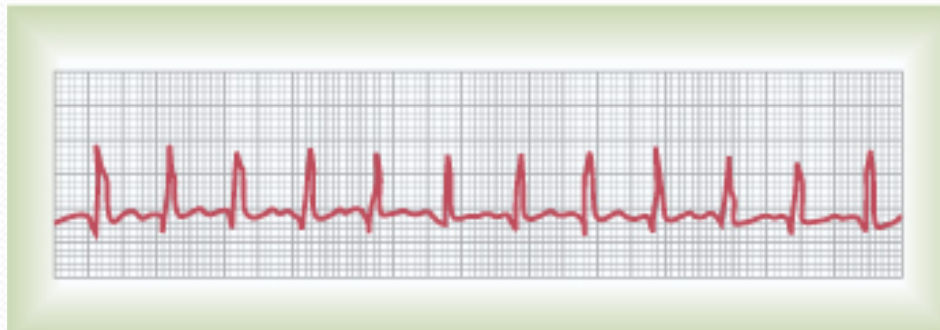


Classification of Cardiac Arrhythmias

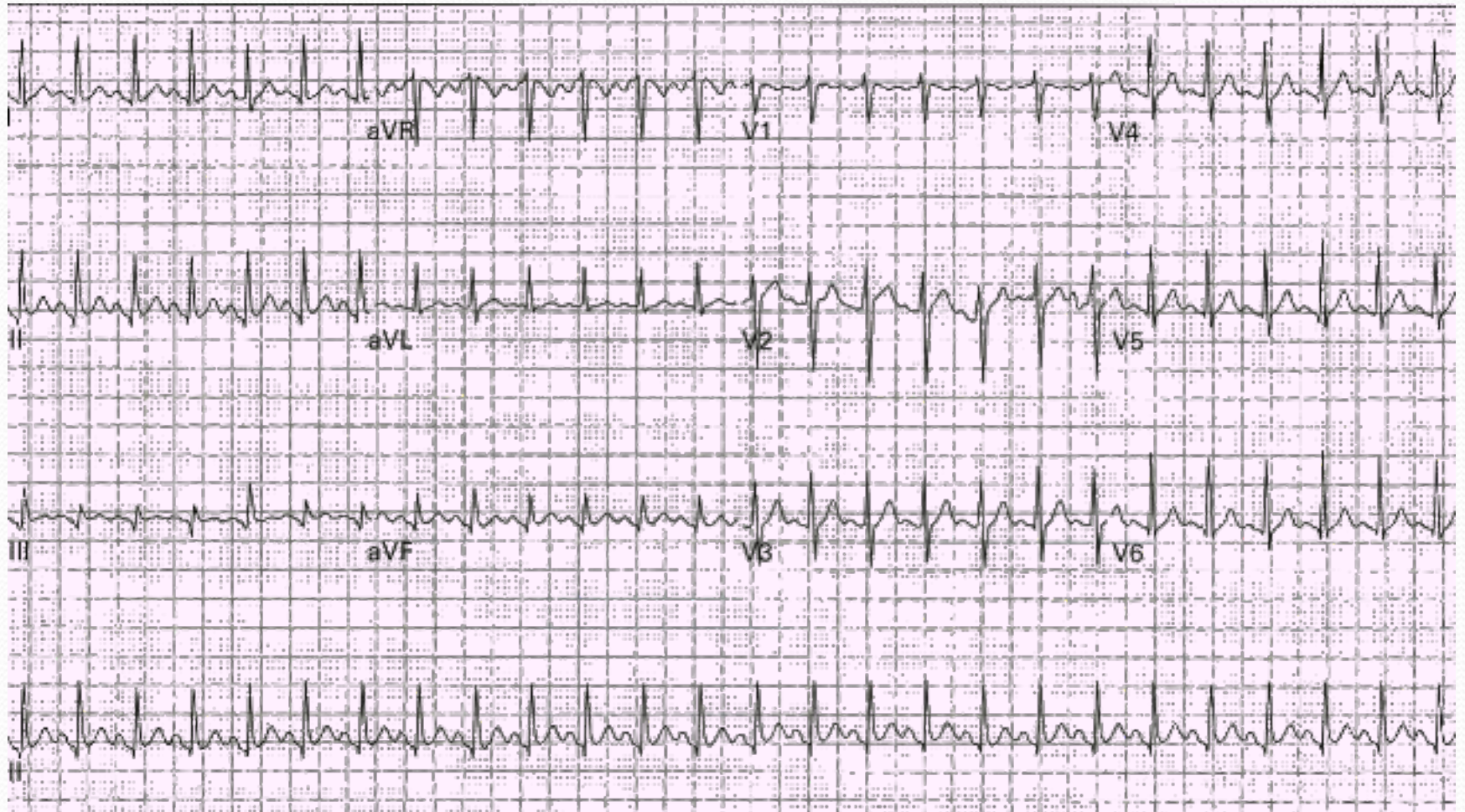
- Rate above or below normal (tachy vs. Brady)
- Regular or irregular rhythm
- Narrow or broad QRS complex
- Relation to P waves
- Supraventricular Vs. ventricular

Abnormal Sinus Rhythm

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

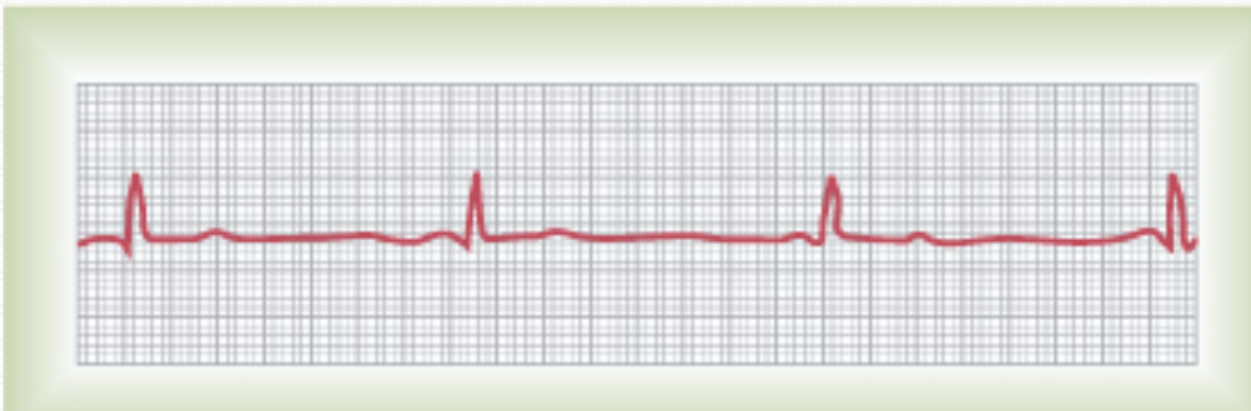


24 year-old pregnant woman with three days of frequent vomiting



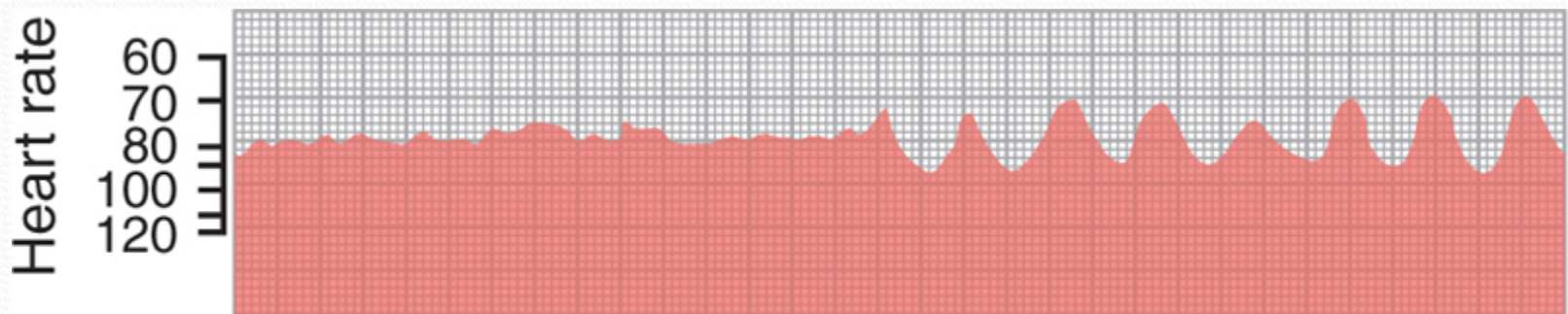
Abnormal Sinus Rhythm

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



Sinus Arrhythmia

- Result from spillover of signals from the medullary respiratory center into the adjacent vasomotor center during inspiration and expiratory cycles of respiration
- The spillover signals cause alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart

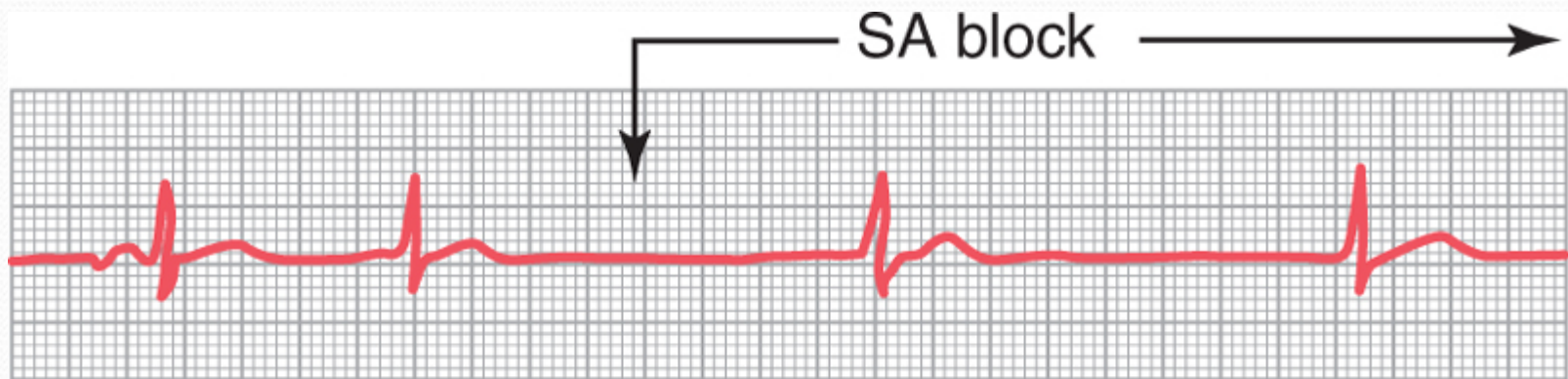


Hall: Guyton and Hall Textbook of Medical Physiology, 12th Edition
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Abnormal Cardiac Rhythms that Result from Impulse Conduction Block

- **Sinoatrial Block**

- The impulse from the S-A node is blocked before it enters the atrial muscle
- Cessation of P waves



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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

Heart Block

1st°

- Constant PR prolongation without drop beat.

2nd°

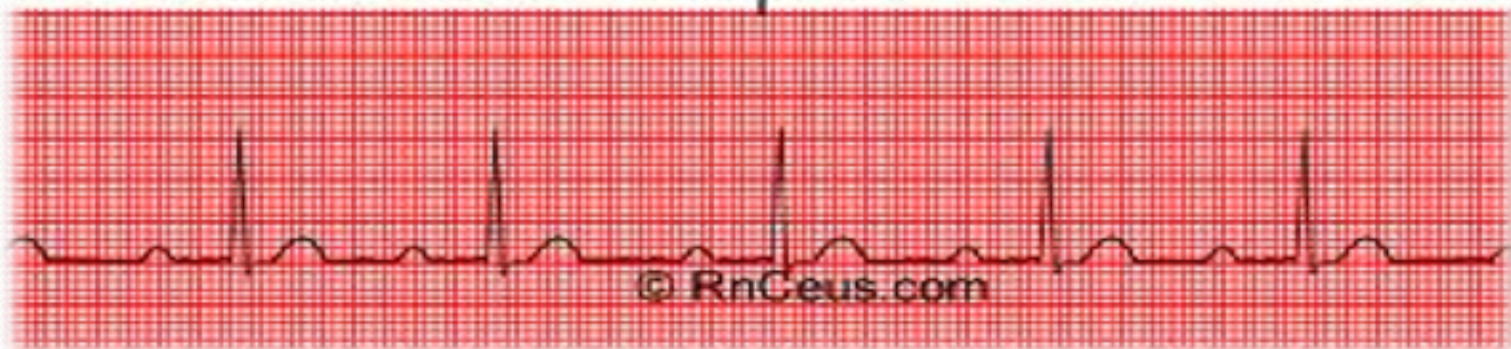
- **Mobitz1:** Progressive PR prolongation + drop beat.
- **Mobitz2:** Constant PR prolongation + drop beat.

3rd°

- Complete dissociation between P and QRS.

First Degree Heart Block

FIRST DEGREE A-V HEART BLOCK



Second Degree Heart Block

- **Mobitz (I):**



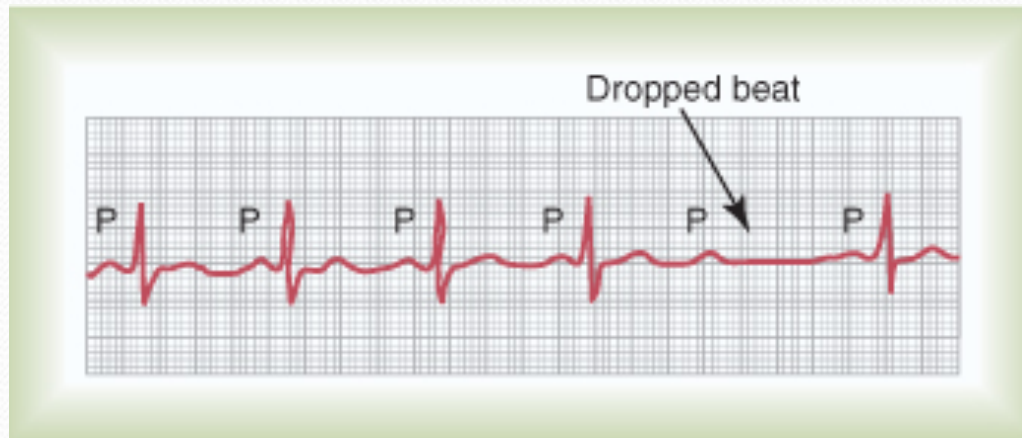
- **Mobitz (II):**



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



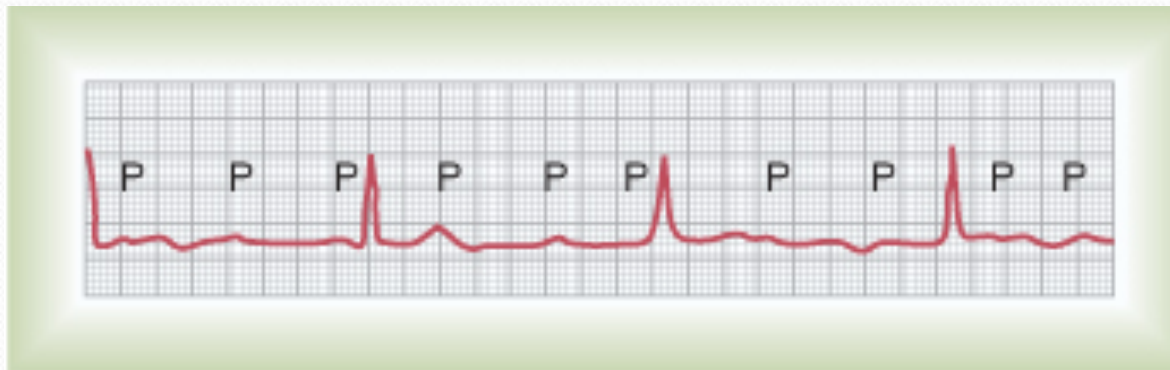
Third Degree Heart Block

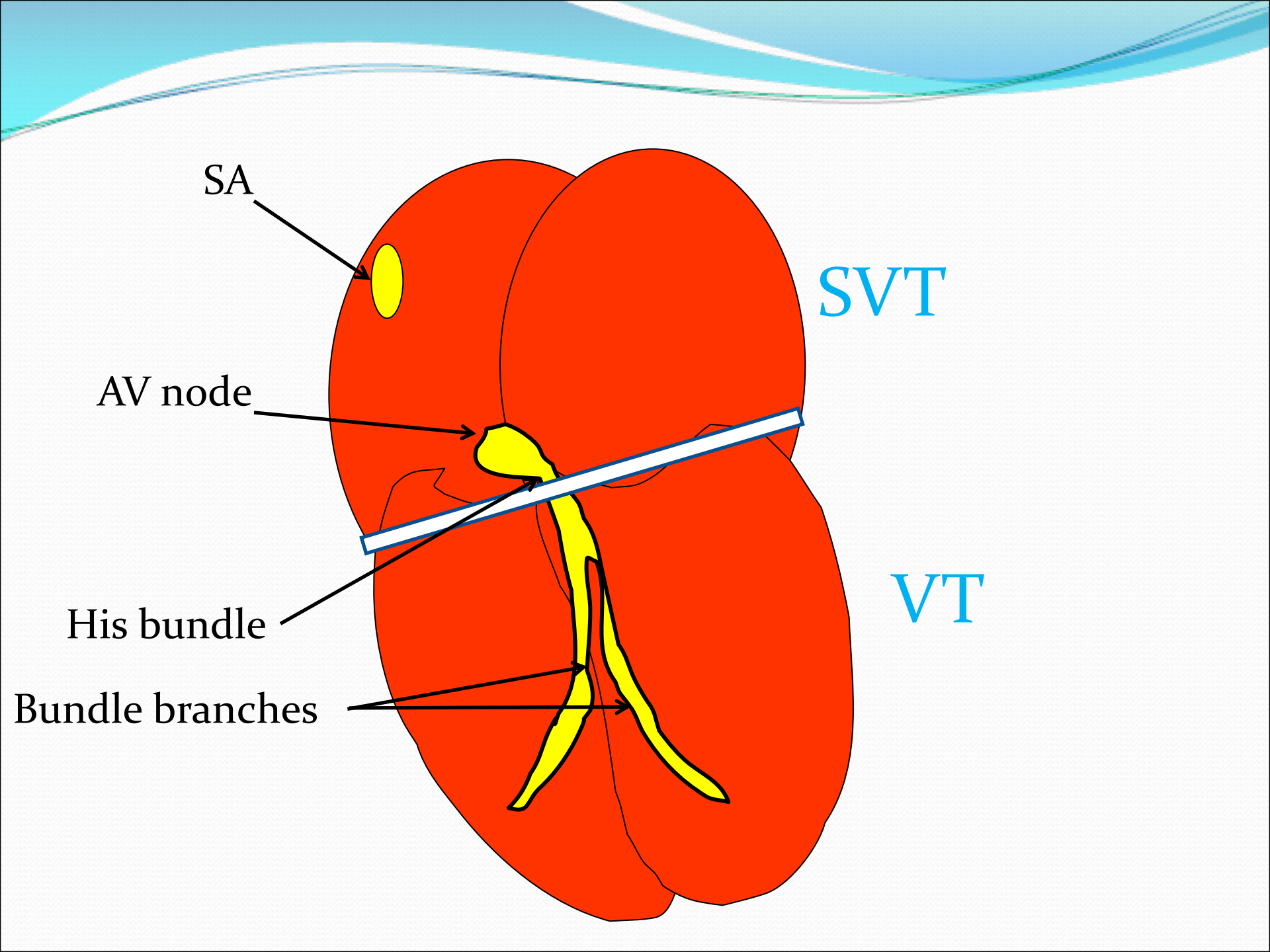


Types of the A-V block

Third degree block (complete)

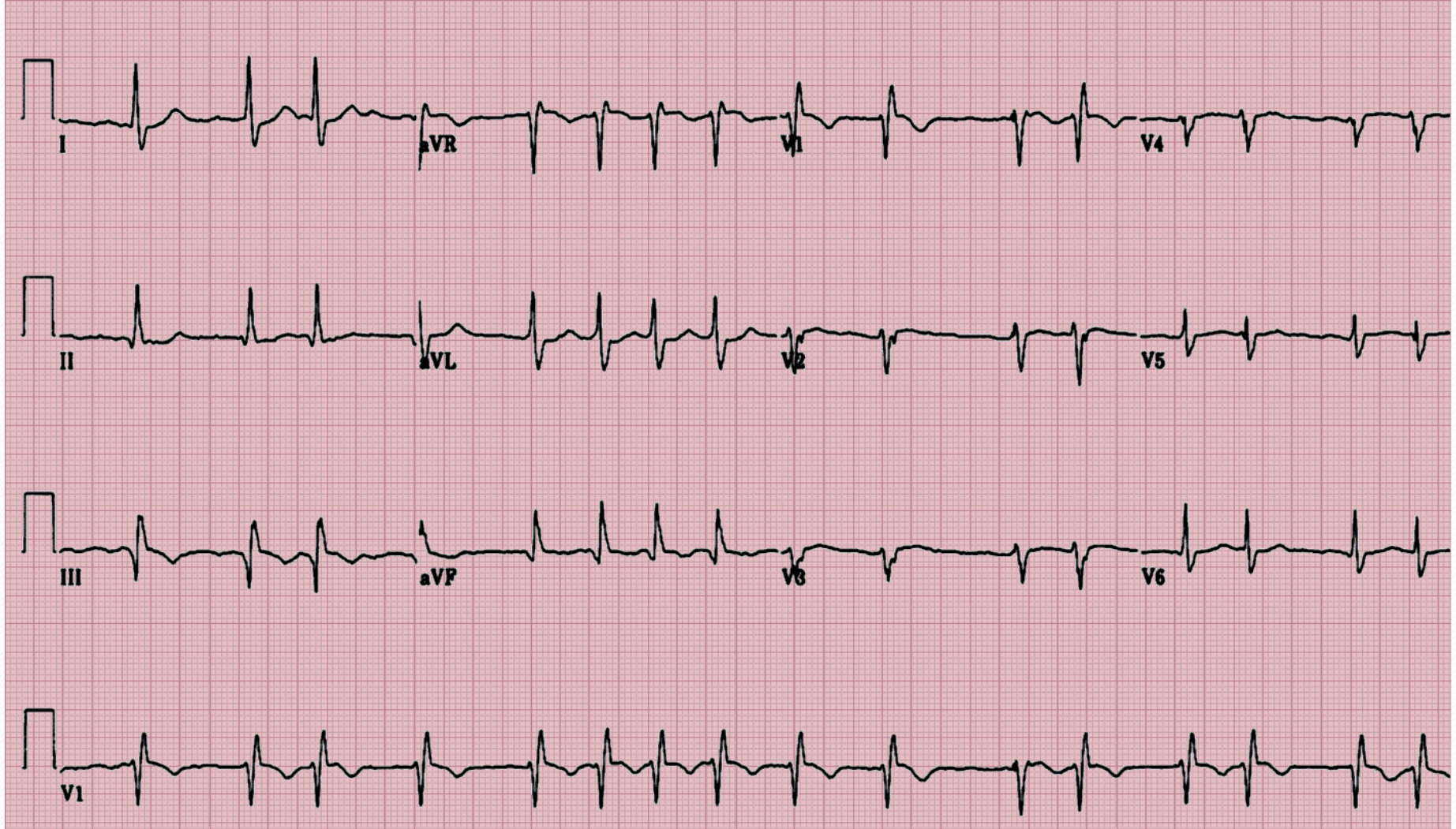
- Complete dissociation of P wave and QRS waves
→ The ventricle escape from the influence of S-A node
- Stokes-Adams Syndrome: AV block comes and goes





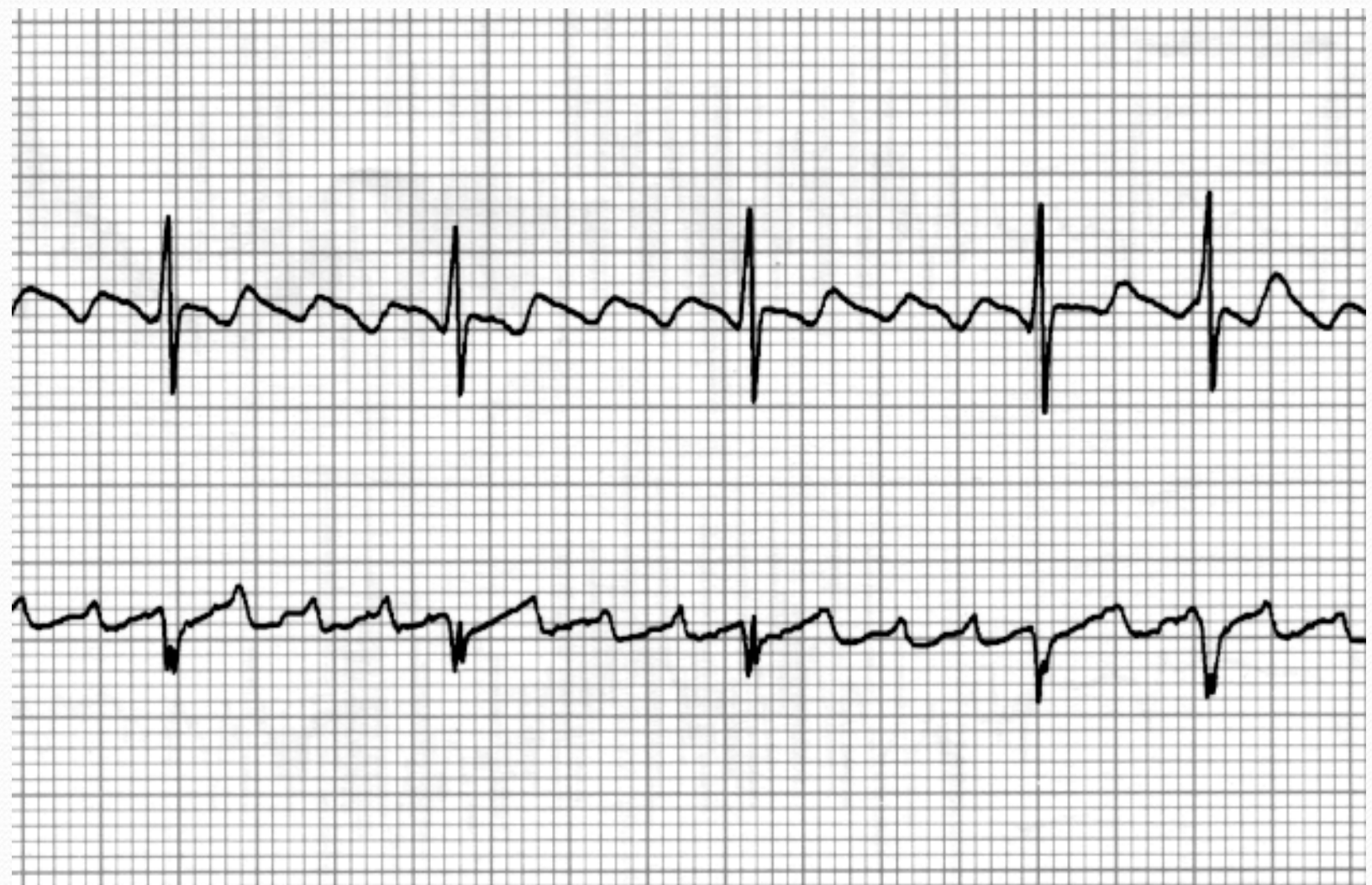
Atrial Fibrillation

- Cause: as ventricular fibrillation
- It occurs more frequently in patients with *enlarged heart*
- The atria do not pump if they are fibrillating
- The efficiency of ventricular pumping is decreased 20 to 30%
- A person can live for years with atrial fibrillation

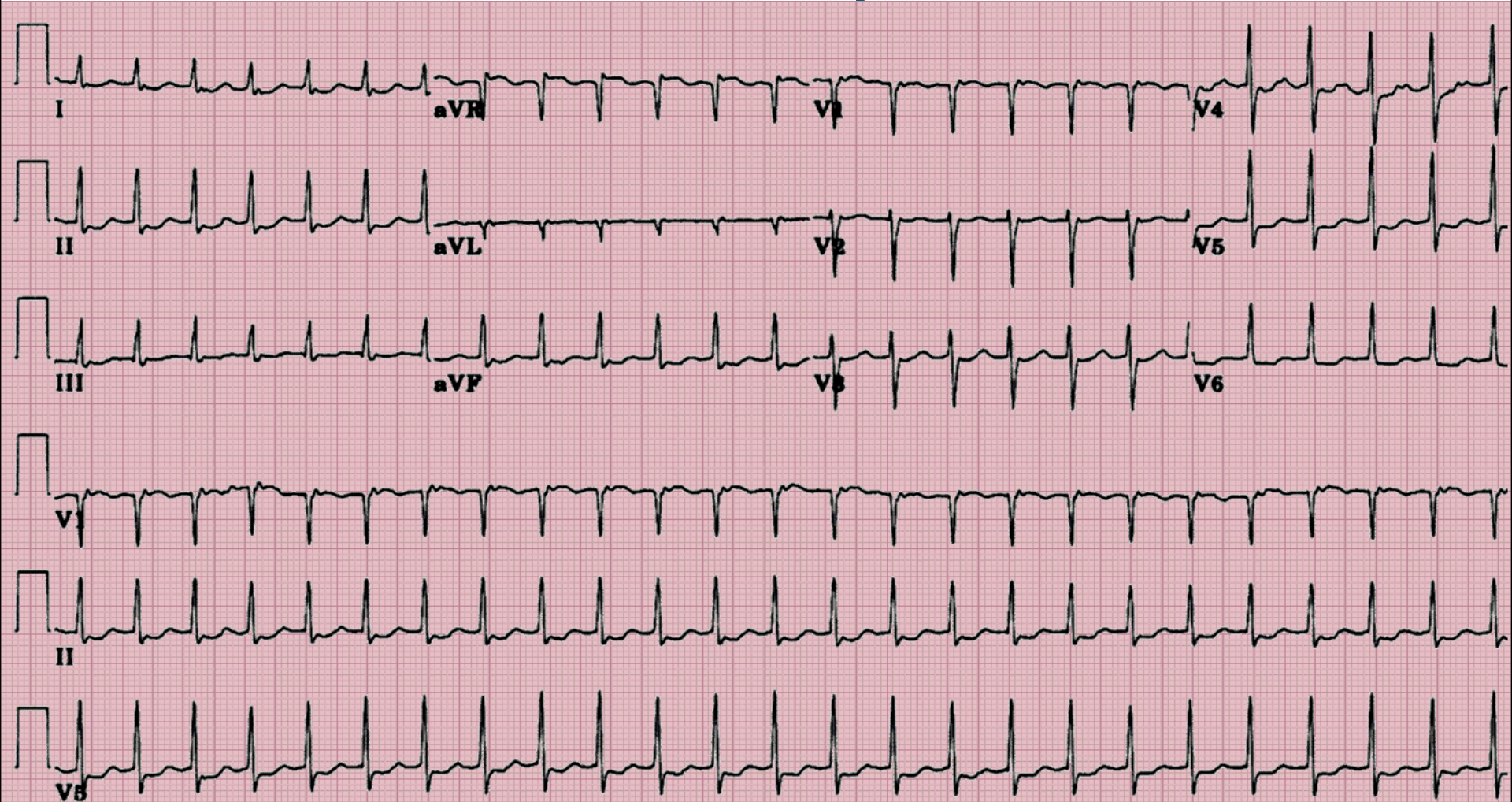


Atrial Flutter

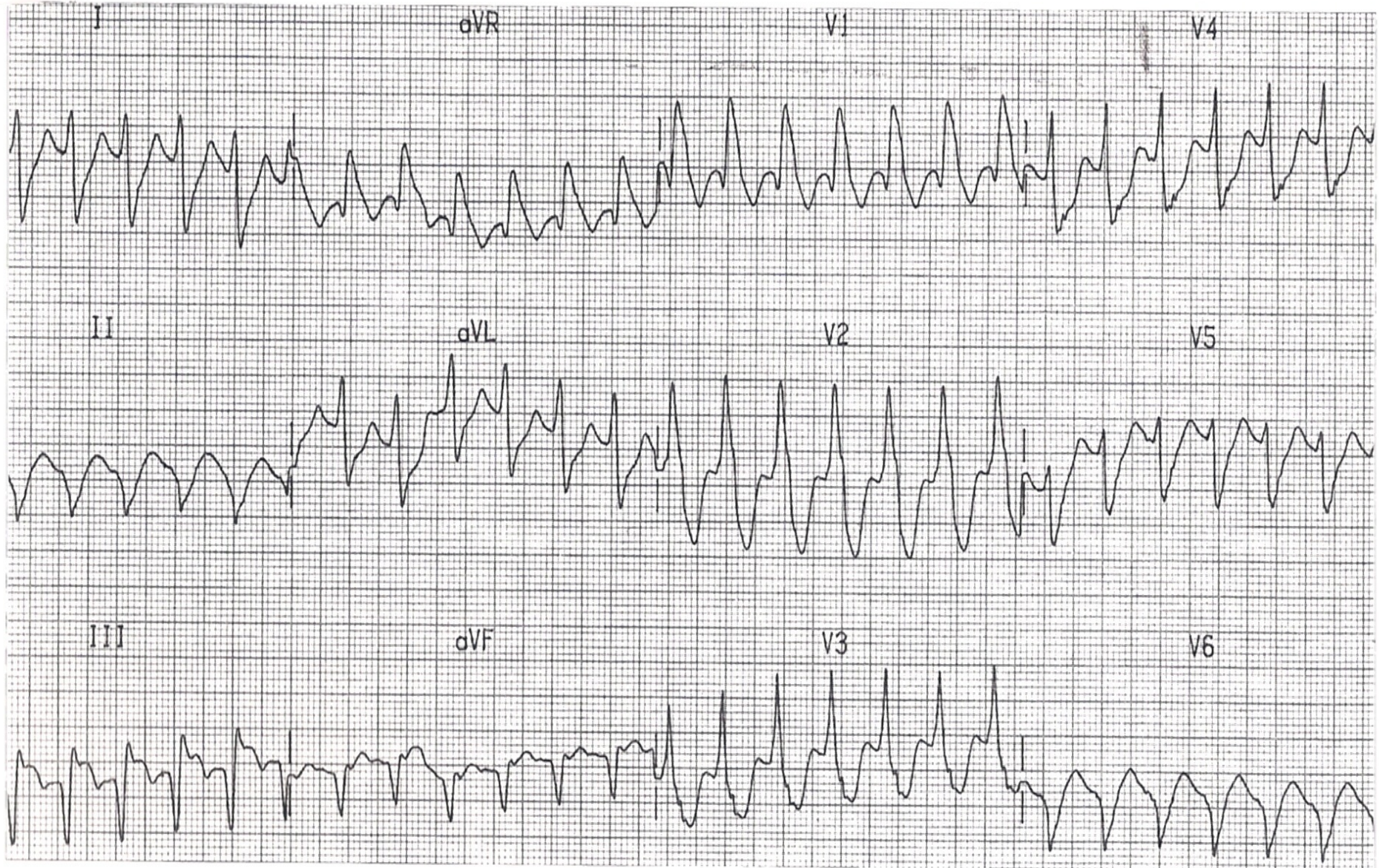
- A single large wave travels around and around in the atria
- The atria contracts at high rate (250 time per minute)



AVRT-Narrow complex

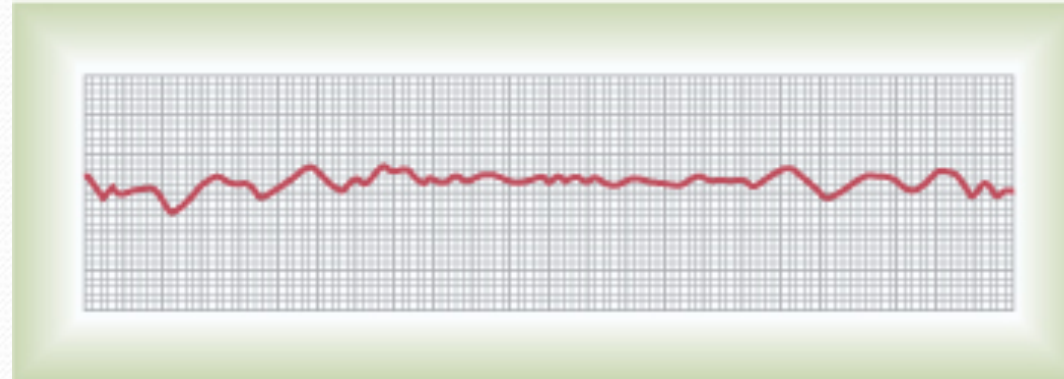


Ventricular Tachycardia



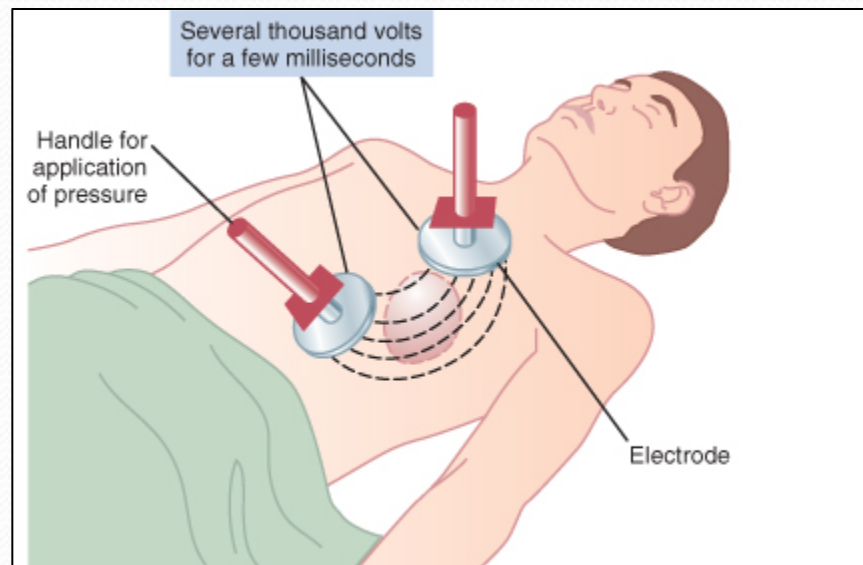
Ventricular Fibrillation

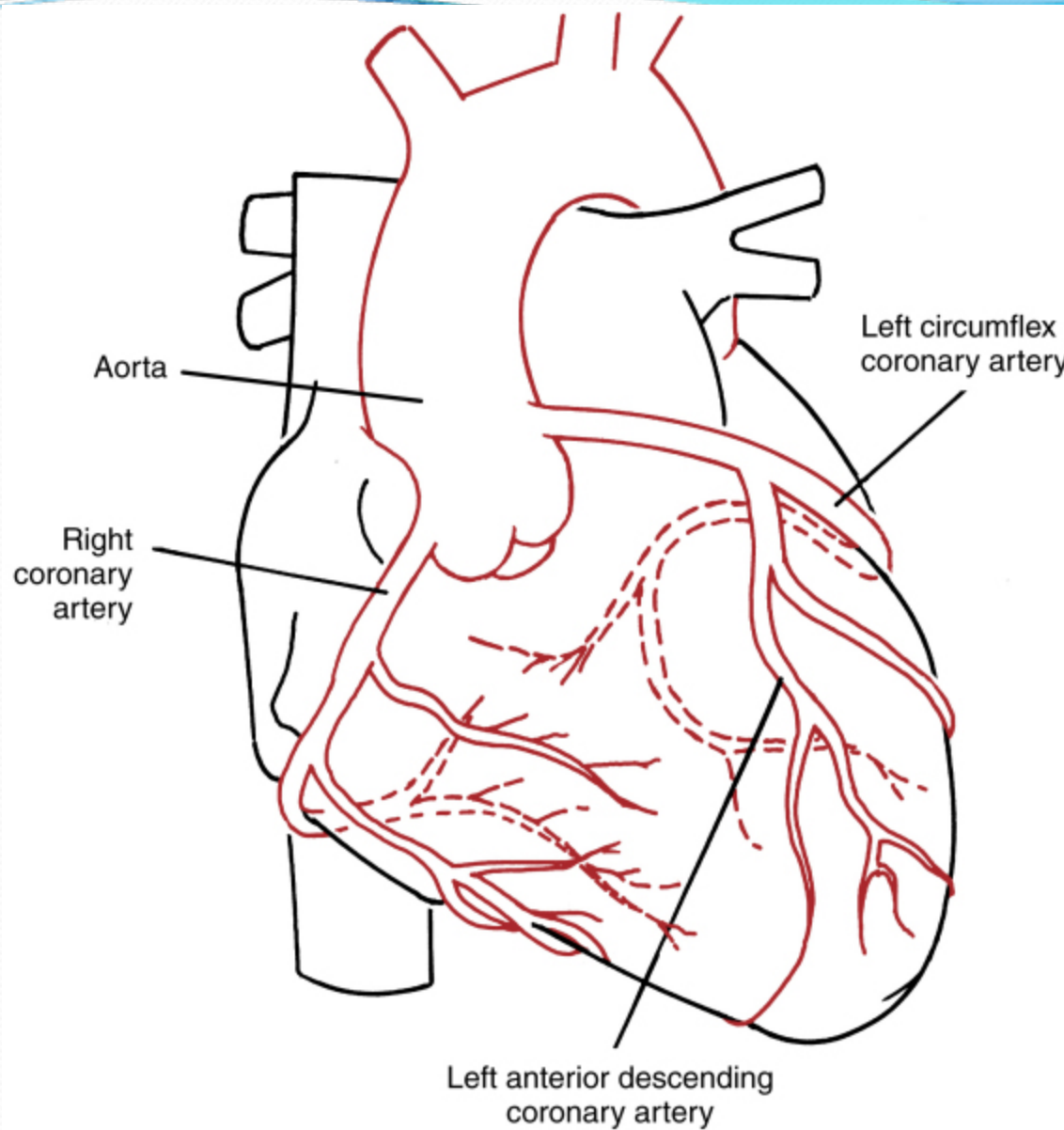
- **The most serious of all arrhythmias**
- Cause: impulses stimulate one part of the ventricles, then another, then itself. Many part contracts at the same time while other parts relax (*Circus movement*)
 - Tachycardia
 - Irregular rhythm
 - Broad QRS complex
 - No P wave
- Treatment : *DC shock*

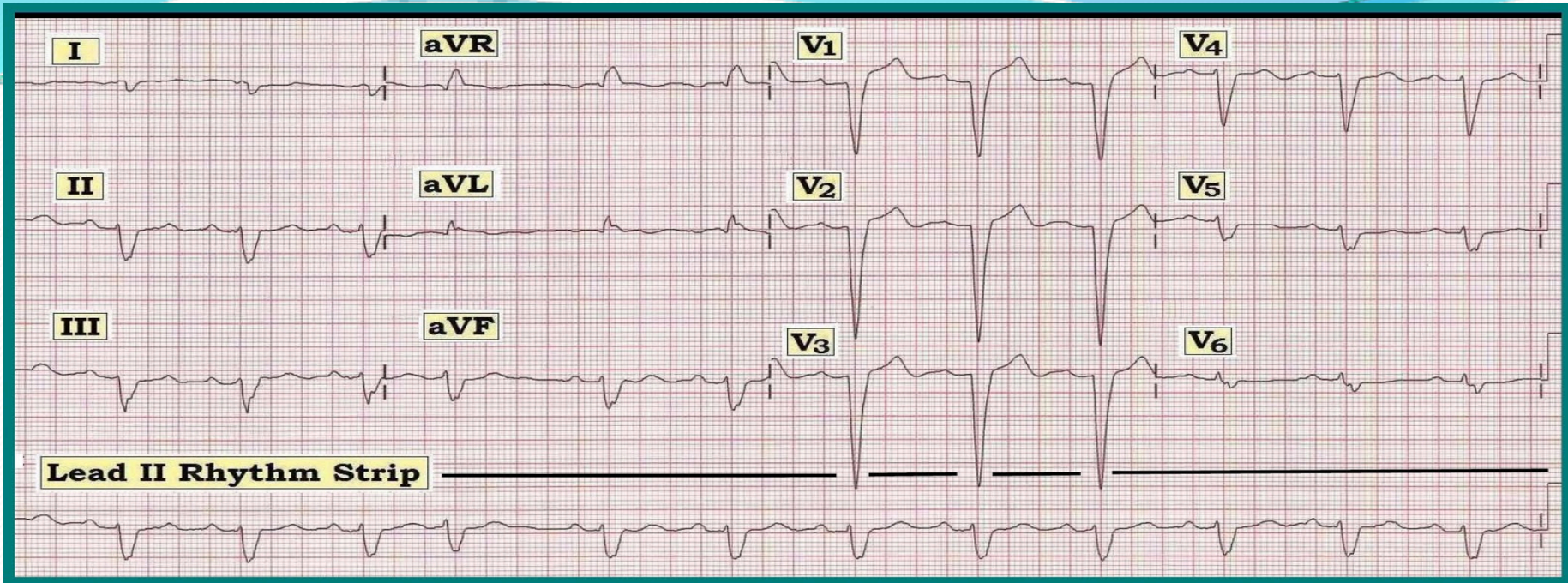


Ventricular Fibrillation

- Treatment : DC shock







I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

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***

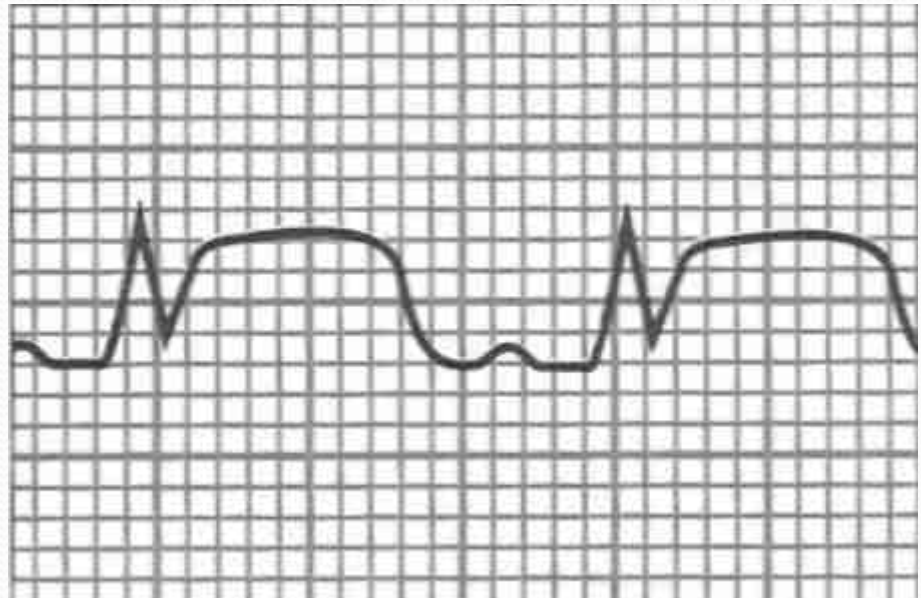
Reversible ischemia

- Inverted T wave
- ST segment depression

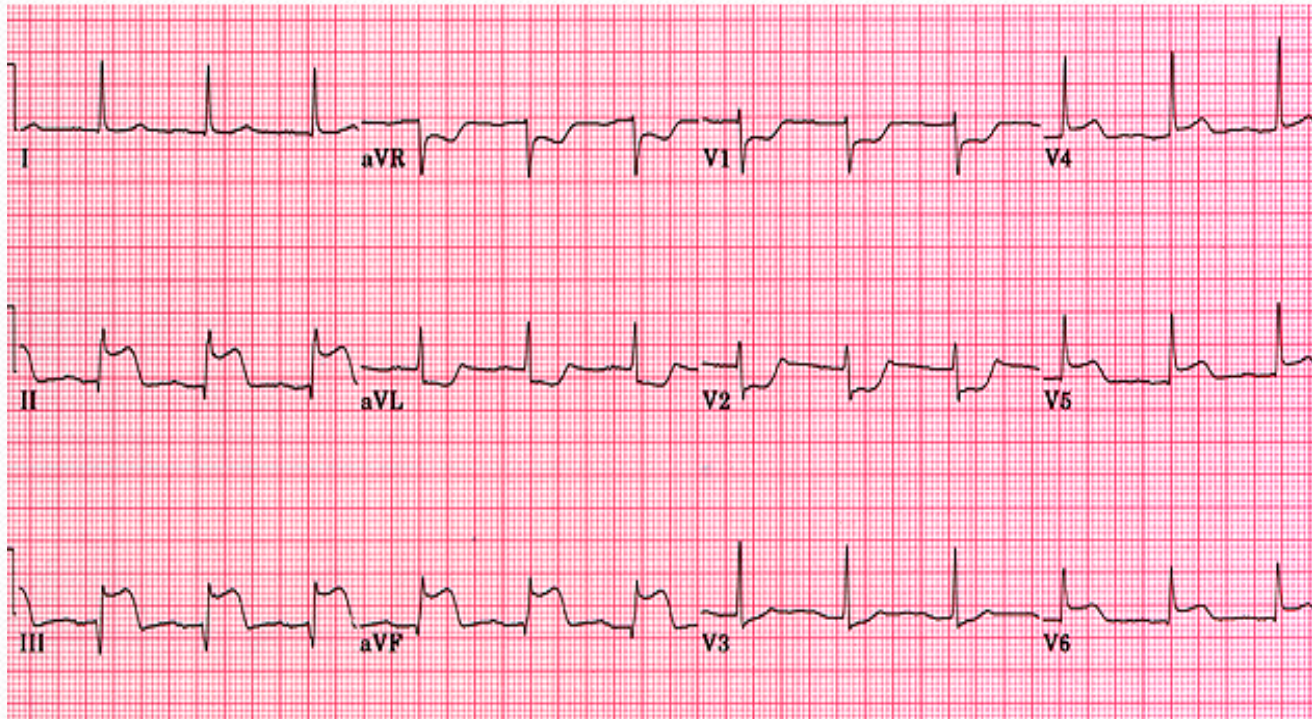


Myocardial Infarction

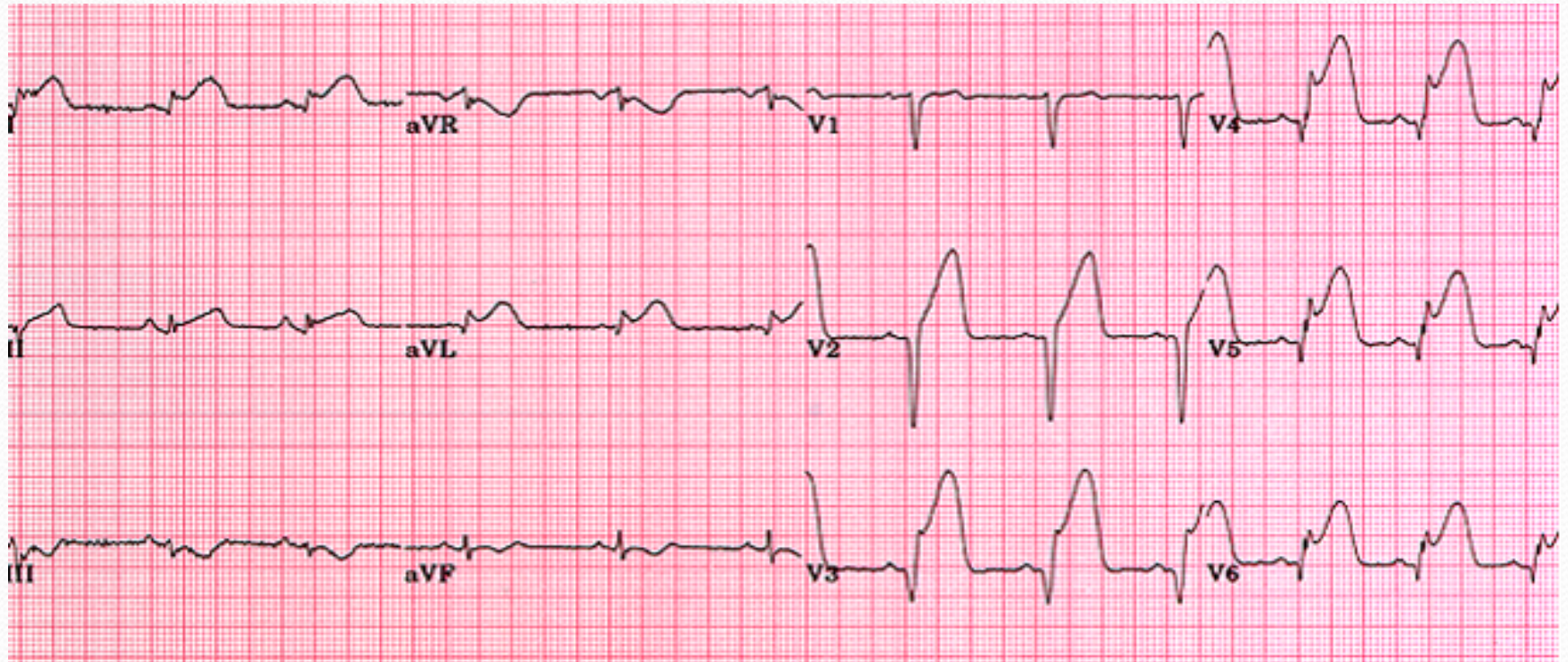
- Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue
- ST segment elevation
- Deep Q wave



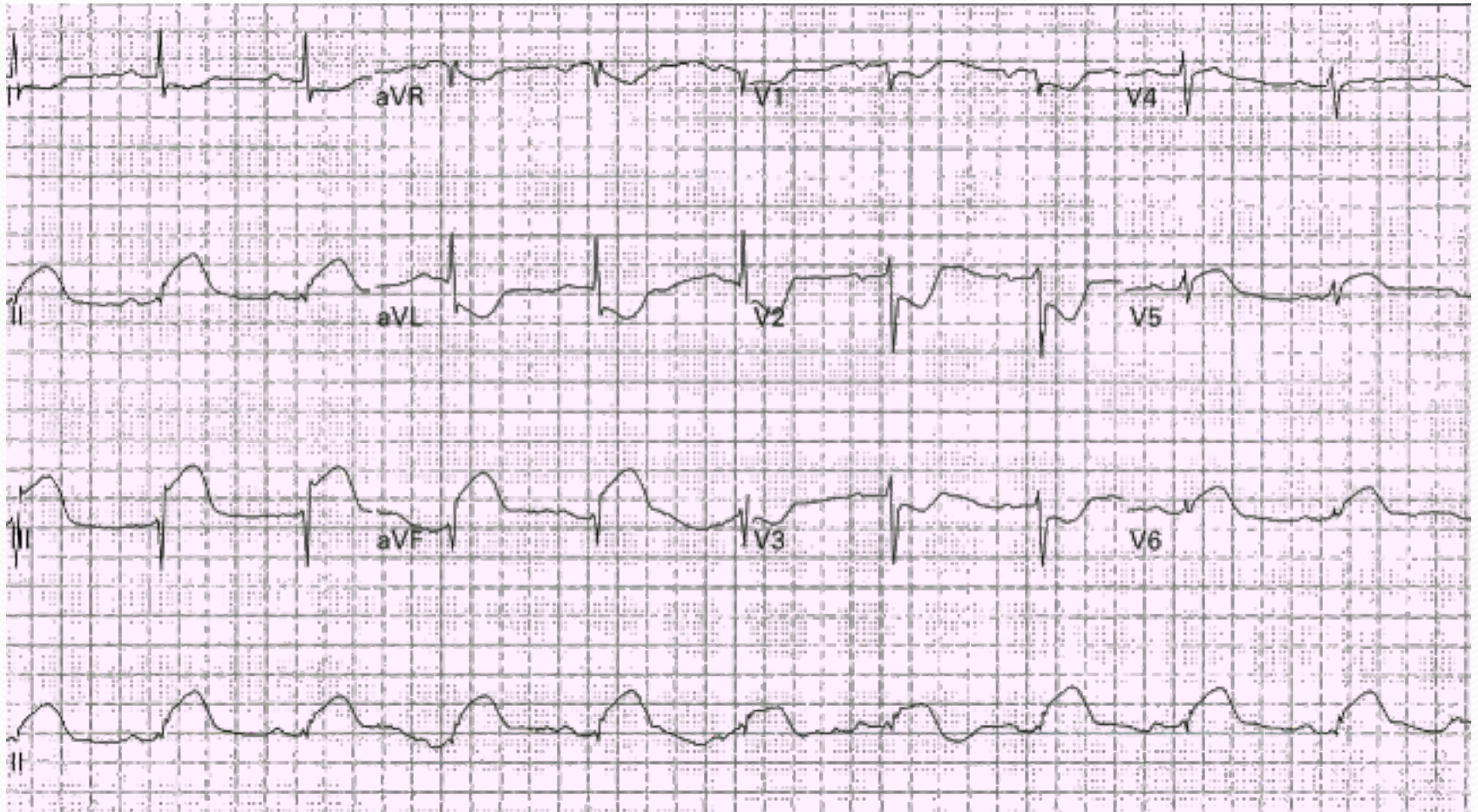
Infero-Posterior MI



Antero-Lateral MI

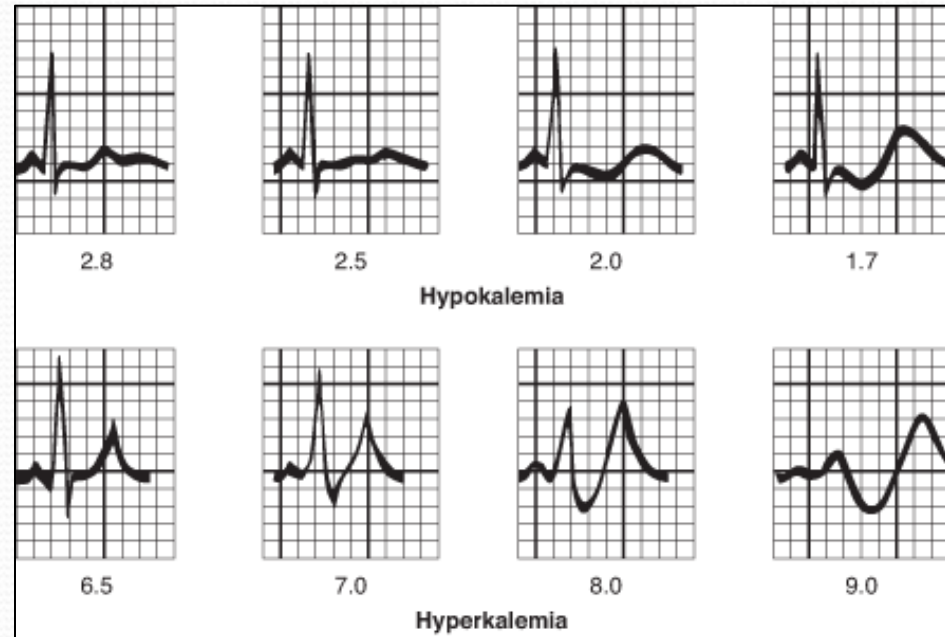


57 year-old man with chest pressure and diaphoresis



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)