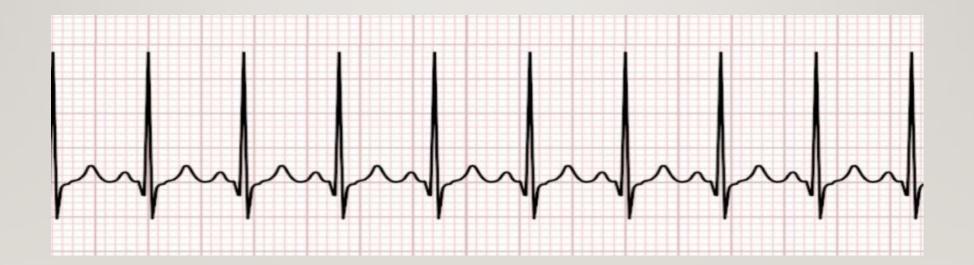
### **Arrhythmias**

- An abnormal heart rhythm.
- Might be associated with tachycardia or bradycardia
- The causes of the cardiac arrhythmias are usually one or a combination of the following abnormalities in the rhythmicity-conduction system of the heart:
- I. 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 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

# Sinus Tachycardia

• Fast heart rate above 100 beats per minute (B.P.M) usually caused by increased sympathetic tone in physiological stress, fever, pain, dehydration, hypovolemia, and anemia



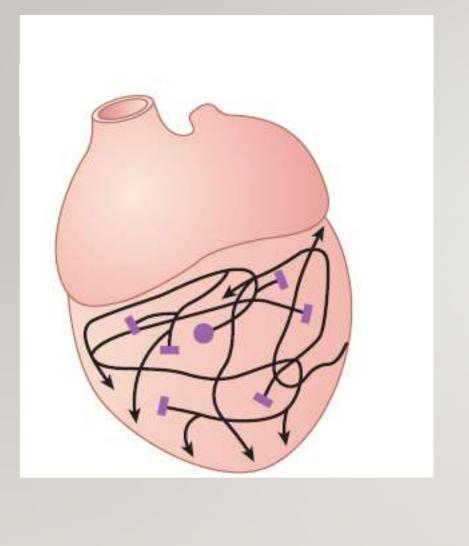
# Sinus Bradycardia

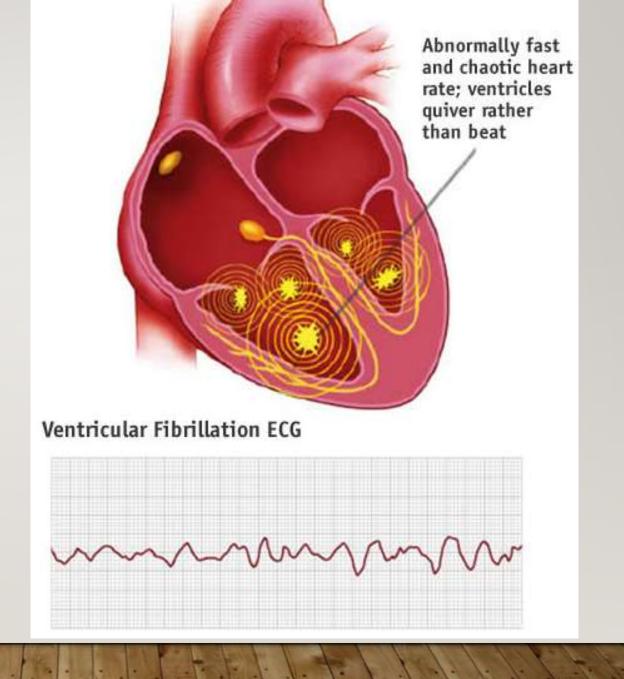
- Slow heart rate below 60 B.P.M.
- Seen normally in athletes at rest
- Vagal stimulation can trigger bradycardia
- Hypothermia, hypothyroidism, certain drugs like beta blockers

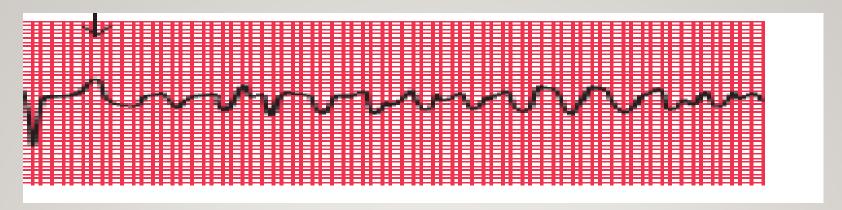


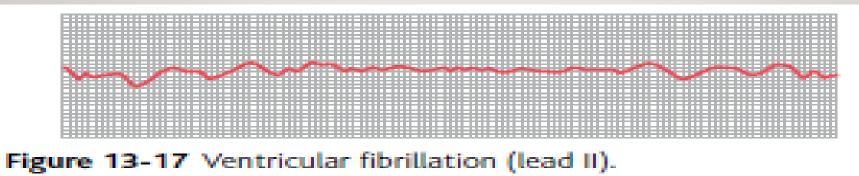
#### Ventricular Fibrillation

- The most serious of all cardiac arrhythmias, if not stopped within 1 to 3 minutes, is almost invariably fatal
- Results from cardiac impulses that have gone berserk within the ventricular muscle mass, stimulating first one portion of the ventricular muscle, then another portion, then another, and eventually feeding back onto itself to re-excite the same ventricular muscle over and over never stopping.
- The ventricular muscle contraction is not coordinated. So no pumping of blood occurs.
- Caused by
- Sudden electrical shock of the heart
- 2. Ischemia of the heart muscle, of its specialized conducting system, or both.
  - 3. Other forms of arrhythmia









- ECG is bizarre and shows no regular rhythm of any type.
- Voltages of the waves in the ECG are usually about 0.5 millivolt when ventricular fibrillation first begins, but they decay rapidly.

#### Atrial fibrillation

- A frequent cause of atrial fibrillation is atrial enlargement
- The normal regular electrical impulses generated by the SA node are overridden by disorganized electrical impulses usually originating in the roots of the pulmonary veins.
- On the ECG either no P waves are seen or only a fine, high frequency, very low voltage wavy record. The QRS complexes are normal in shape but are <u>irregular</u>



### Atrioventricular (AV) Block

- Results from conditions that can either decrease the rate of impulse conduction in the AV bundle or block the impulse entirely:
- Ischemia of the A-V node or A-V bundle fibers
- 2. Compression of the A-V bundle by scar tissue or by calcified portions of the heart.
- 3. Inflammation of the A-V node or A-V bundle
- 4. Extreme stimulation of the heart by the vagus nerves

#### TYPES OF AV BLOCK

- First degree heart block
- Second degree heart block
- Third degree heart block
- Stokes- Adams Syndrome

# First Degree Heart Block

- When the PR interval increases to greater than 0.2 sec, the P-R interval is said to be prolonged and the patient is said to have first-degree heart block.
- Caused by coronary artery disease, acute rheumatic carditis, digoxin toxicity or electrolyte disturbances.



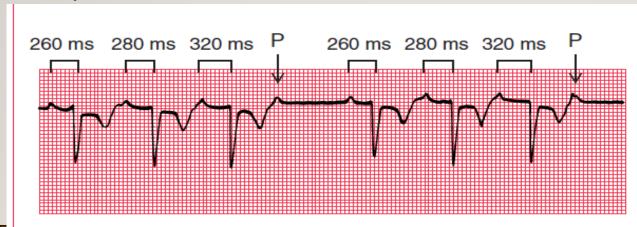
PR interval here is 0.32 seconds

#### SECOND DEGREE BLOCK

- Happens when conduction through the A-V bundle is slowed enough to increase the P-R interval to 0.25 to 0.45 second
- The action potential is sometimes strong enough to pass through the bundle into the ventricles and sometimes not strong enough to do so.
- So occasionally there will be "dropped beats"; an atrial P wave but no QRS-T wave.
- There are two types of second-degree A-V block:
- I. Type I (Wenckebach periodicity)
- 2. Type II (Fixed ratio blocks)

### TYPE I (WENCKEBACH PERIODICITY)

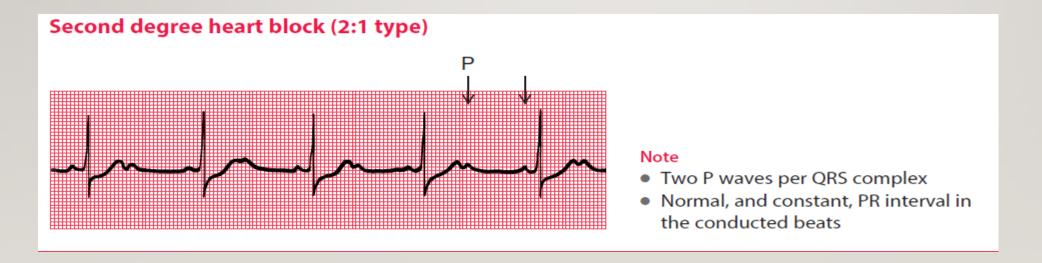
- Progressive lengthening of the PR interval and then failure of conduction of an atrial beat, followed by a conducted beat with a shorter PR interval and then a repetition of this cycle.
- A type I block is almost always caused by abnormality of the A-V node. In most cases no specific treatment is needed.



- Progressive lengthening of the PR interval
- One nonconducted P wave
- Next conducted beat has a shorter PR interval than the preceding conducted beat

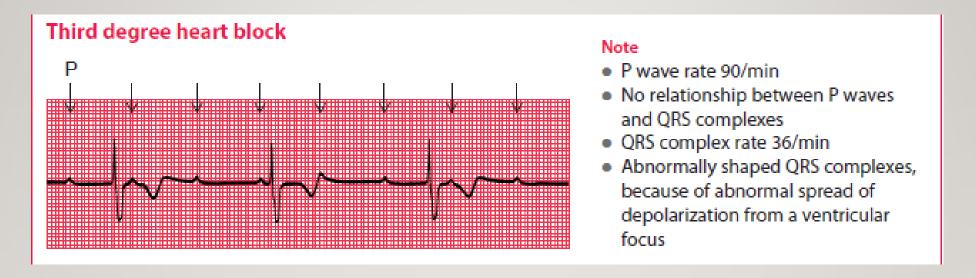
# TYPE II (FIXED RATIO BLOCKS)

- There is usually a fixed number of non-conducted P waves for every QRS complex.
- Alternate conducted and non-conducted atrial beats(2:1), or one conducted atrial beat and then two (3:1) or three (4:1) non-conducted beats.
- Caused by an abnormality of the bundle of His-Purkinje system and may require implantation of a pacemaker



### Third degree block

- Occurs with complete block of the impulse from the atria into the ventricles.
- The ventricles spontaneously establish their own signal, usually originating in the AV node, AV bundle or purkinji fibers. Therefore, the P waves become dissociated from the QRS-T complexes.
- Third-degree block is characterized by:
- I. Regular P-P interval
- 2. Regular R-R interval
- 3. Lack of an apparent relationship between the P waves and QRS complexes
- 4. Atrial rate is higher than ventricular rate
- Occur as an acute phenomenon in patients with myocardial infarction or it may be chronic, usually due to fibrosis around the bundle of His.



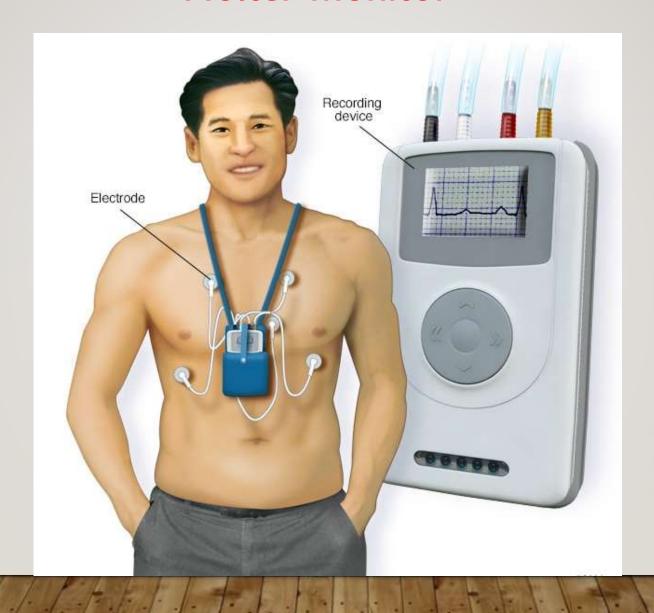
The A-V nodal fibers, when not stimulated by SA node discharge at an intrinsic rhythmical rate of 40 to 60 times per minute, and the Purkinje fibers discharge at a rate somewhere between 15 and 40 times per minute

# Stokes- Adams Syndrome

- The total block comes and goes
- The duration of block may be a few seconds, a few minutes, a few hours, or even weeks
- Overdrive suppression prevents the ventricles from beating until after a delay of 5 to 30 seconds. During this time the patient will faint.



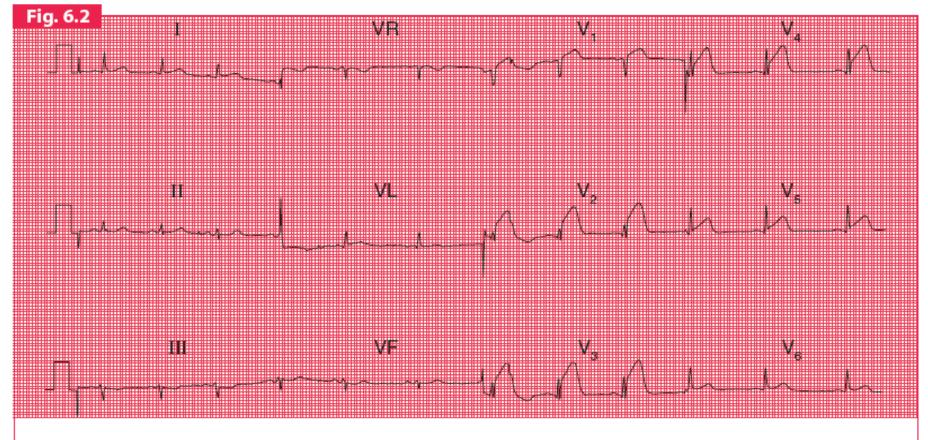
# Holter monitor



# ECG changes seen in MYOCARDIAL INFARCTION (MI) AND ANGINA PECTORIS

- ECG is very useful for diagnosing MI & Angina pectoris and locating the affected areas.
- One of the earliest sign of MI is ST segment changes & it occurs in the leads corresponding to the part of the heart that is damaged:
- Leads VI-V4 with anteroseptal wall infarction
- Lead aVL, I,V5 & V6 with lateral wall infarction
- Leads II, III and aVF with inferior wall infarction.
- To be considered significant, more than I mm of ST segment elevation or depression in at least two contiguous limb leads (e.g. I and VL; III and VF), or more than 2 mm of ST segment elevation or depression in at least two contiguous chest leads

- In case of myocardial infarction, within a day or so, the ST segments return to the baseline.
- Without proper & prompt treatment the T waves in the affected leads become inverted, and Q waves develop within 24 hours. These ECG changes are usually permanent
- In angina ECG changes are noticed while the patient is in pain, once the pain has resolved the ECG returns to normal.
- ST segment changes are very common with angina.
- When the ECG is normal at rest, ST segment changes may be induced by making the patient exercise,
  this test is called stress ECG



#### ST segment elevation in acute anterior ST segment elevation myocardial infarction

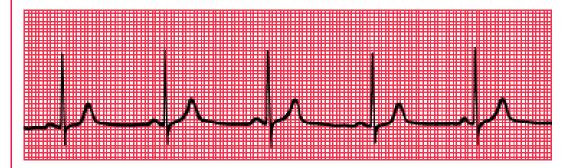
#### Note

- Sinus rhythm, rate 75/min
- Normal axis
- Normal QRS complexes
- ST segments elevated in leads V<sub>1</sub>–V<sub>5</sub>
- Normal T waves

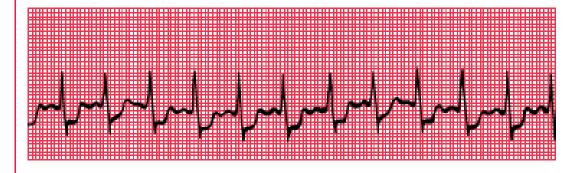
#### Fig. 4.14

#### Exercise-induced ischaemic changes

#### Rest:



#### Exercise:



#### Note

- In the upper (normal) trace, the heart rate is 55/min and the ST segments are isoelectric
- In the lower trace, the heart rate is 125/min and the ST segments are horizontally depressed

## Pathological Q wave

- ► Q waves are considered pathological if:
  - > 0.04 sec
  - > 2 mm deep
  - > 25% of the QRS complex

Pathological Q waves usually indicate ongoing or <u>prior</u> myocardial infarction.

