

# **CVS**

## **Electrical Activity and the Electrocardiogram**

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# Objectives

- To understand :
  - the origin and spread of cardiac excitation
  - the recording of a normal electrocardiogram
  - Review sample questions

# Introduction

- Anatomic considerations (see figure)
  - **SA node** – located at the junction of at the junction of the **superior vena cava** and the **right atrium**
  - **AV node** – located in the **right posterior portion** of the **interatrial septum**
  - **SA node** and **AV node** connected by 3 Purkinje like fibres
    - (a) anterior internodal tract of **Bachman**, (b) middle internodal tract of **Wenckebach**, and (c) posterior internodal tract of **Thorel**
- Conduction occurs via atrial myocyte but is fastest in the bundles.
- AV node only conducting pathway between atria and ventricles
- SA node and AV node contain P-cells - histology
- Nerve supply and relationship to embryonic development

# The conducting system of the Heart

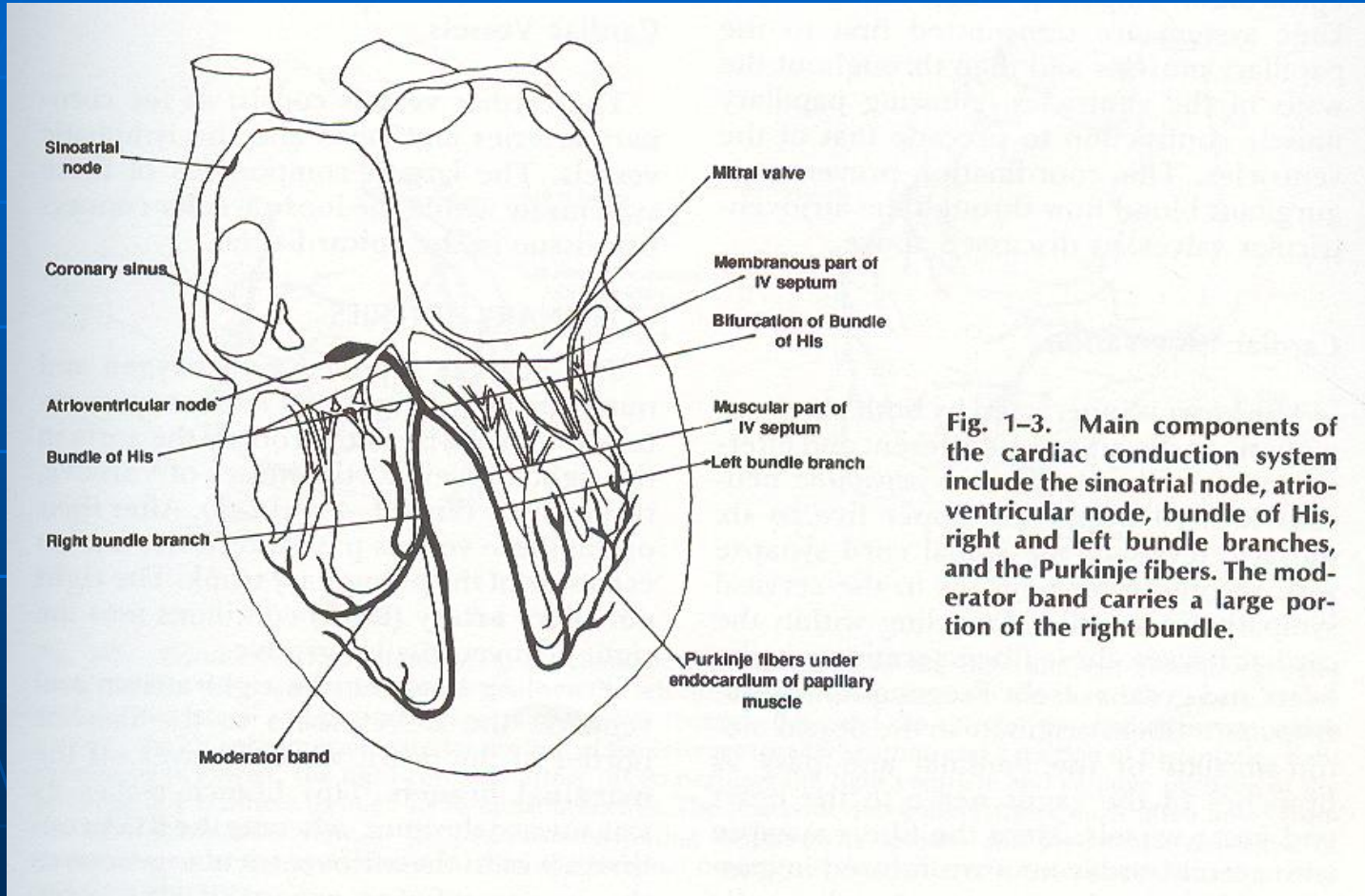


Fig. 1-3. Main components of the cardiac conduction system include the sinoatrial node, atrioventricular node, bundle of His, right and left bundle branches, and the Purkinje fibers. The moderator band carries a large portion of the right bundle.

# Pacemaker cells and automaticity

- **Pacemaker cells** – do not require external stimulation to initiate an Action potential
- They have the property of **automaticity** – undergo spontaneous phase 4 depolarization –  $\text{Na}^+$  responsible
- **Pacemaker behaviour** – cells of SAN, AVN, HIS purkinje fibres, (Ventricular muscle under disease conditions!)
- Action Potential of Pacemaker cell – different from Ventricular muscle cell (see figure)

# AP – Ventricular myocyte vs Pacemaker cells

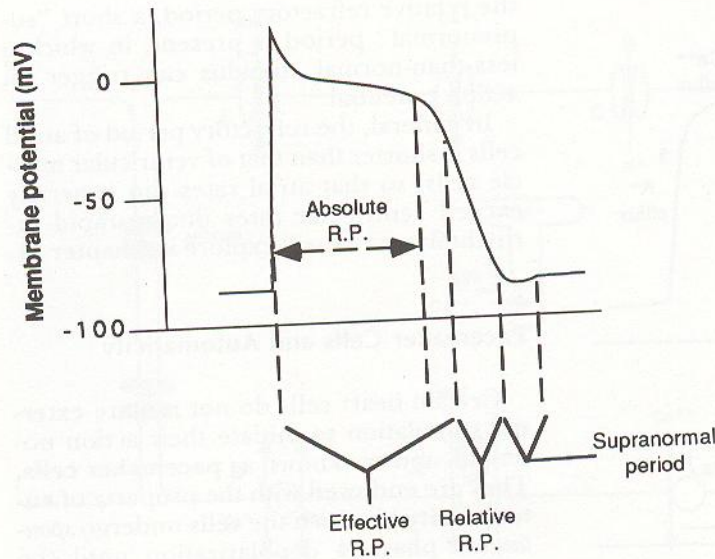


Fig. 1-11. Refractory the myocyte. During the refractory period (ARP) is unexcitable to another action. The effective refractory period includes a brief period beyond the ARP during which stimulation produces a small depolarization that does not propagate. During the relative refractory period, stimulation produces a weak action potential (AP) that propagates but is less than normal. This is due to less-than-normal stimulation being able to trigger an action potential.

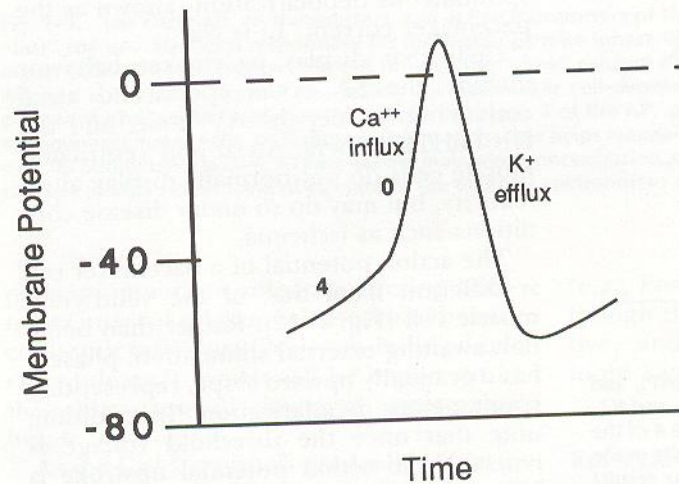


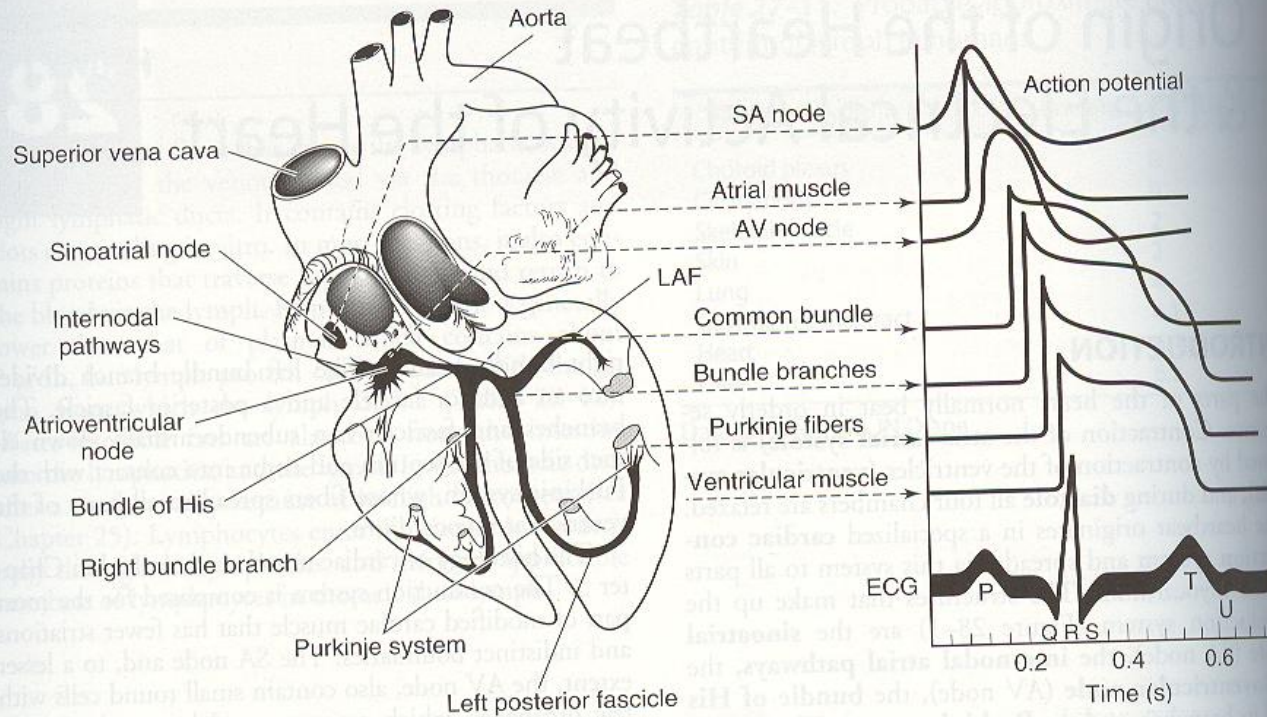
Fig. 1-12. Action potential in a pacemaker cell. It is characterized by slow, steady depolarization. When the membrane potential reaches 0 mV, the upstroke of the action potential follows. The slow depolarization of phase 0 is less than in a nonpacemaker cell.



# Spread of Cardiac Excitation

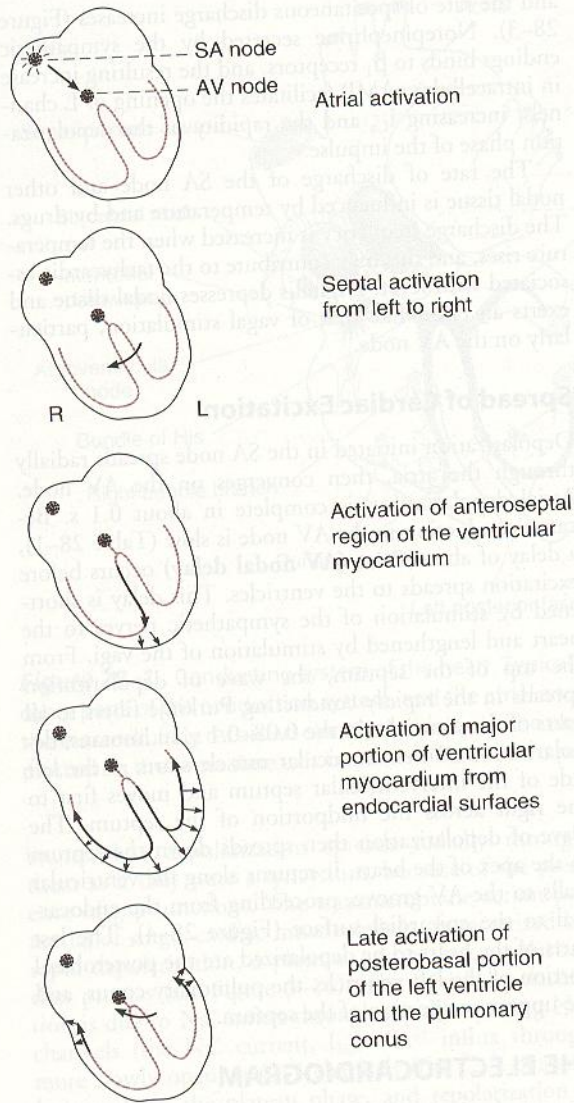
- SA node initiates excitation (rate 60-80 beats/min)
- Spreads to AV Node – delay of approx 0.1 sec (reasons?)
  - ✓ small diameter fibres
  - ✓ Pause allows atria to fully empty their contents
  - ✓ Allows AV-node to serve as a 'gatekeeper' of onward conduction
  - ✓ Role of Autonomic nervous system (explain?)
- Depolarization spreads rapidly from top of the Septum to all parts of the ventricle in 0.08-0.1 sec
- Normal sequence of cardiac depolarization (see figure)

# The conducting system of the heart



**Figure 28-1.** Conducting system of the heart. Typical transmembrane action potentials for the SA and AV nodes, other parts of the conduction system, and the atrial and ventricular muscles are shown along with the correlation to the extracellularly recorded electrical activity, ie, the electrocardiogram (ECG). The action potentials and ECG are plotted on the same time axis but with different zero points on the vertical scale. LAF, left anterior fascicle.





**Figure 28-4.** Normal spread of electrical activity in the heart. (Reproduced, with permission, from Goldman MJ: *Principles of Clinical Electrocardiography*, 12th ed. Originally published by Appleton & Lange. Copyright © 1986 by The McGraw-Hill Companies, Inc.)

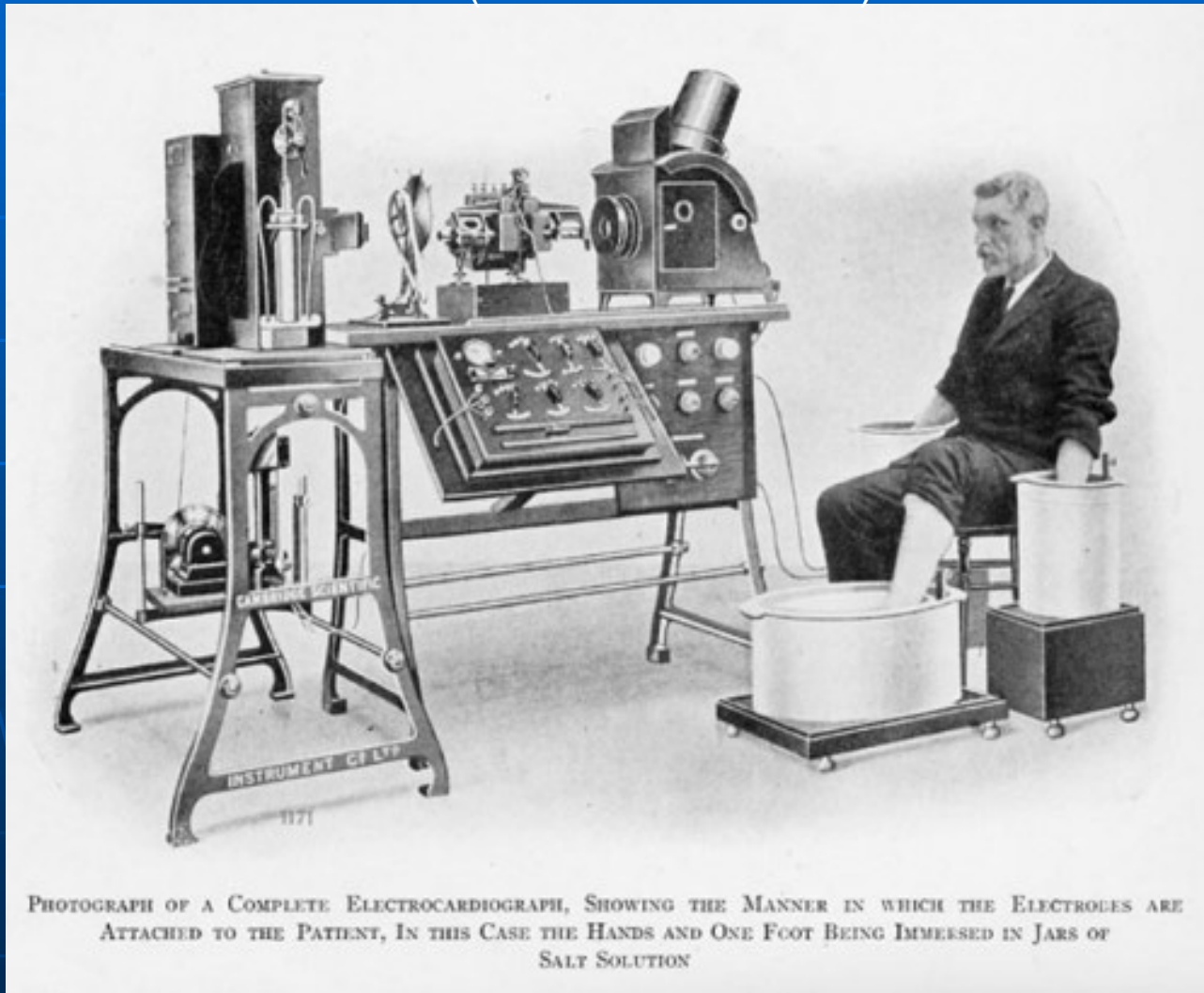
# Normal sequence of cardiac depolarization

# ECG: What is it?

- **Surface recording** of the algebraic sum of the action potentials of cardiac fibres
- Human body is a **volume conductor**
- Action potentials fluctuate and are recorded by an **Electrocardiograph** machine on moving paper strip

# Willem Einthoven

Nobel prize in 1924 for electrocardiogram  
(discovered in 1903)



# ECG: The Lead Reference system I

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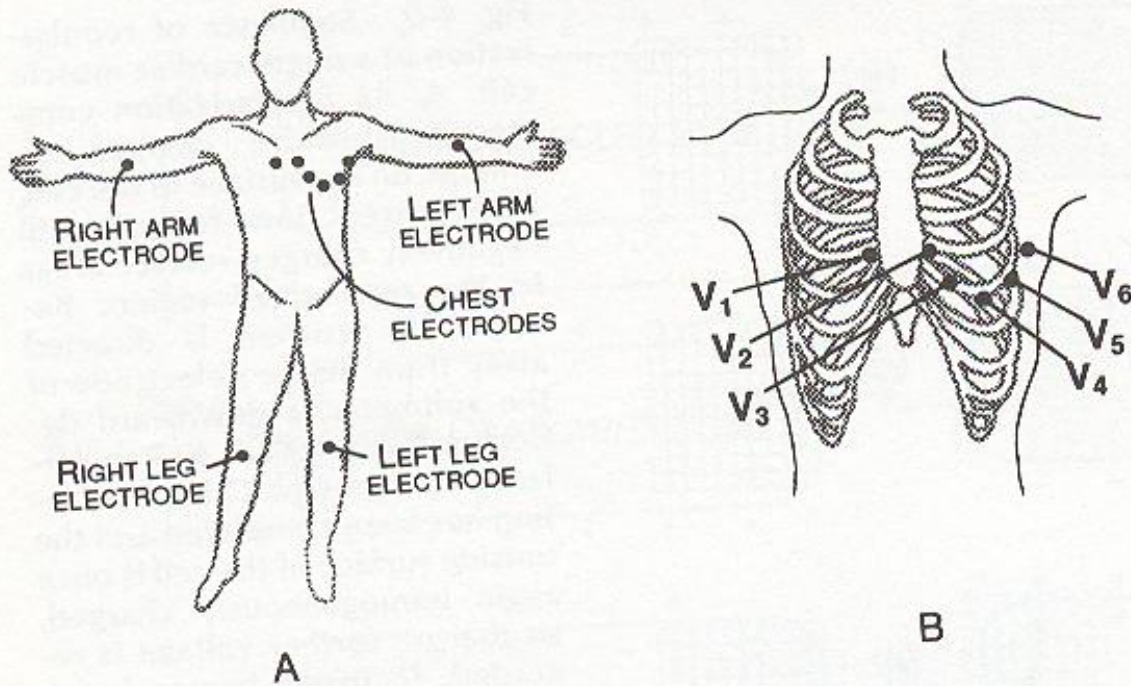
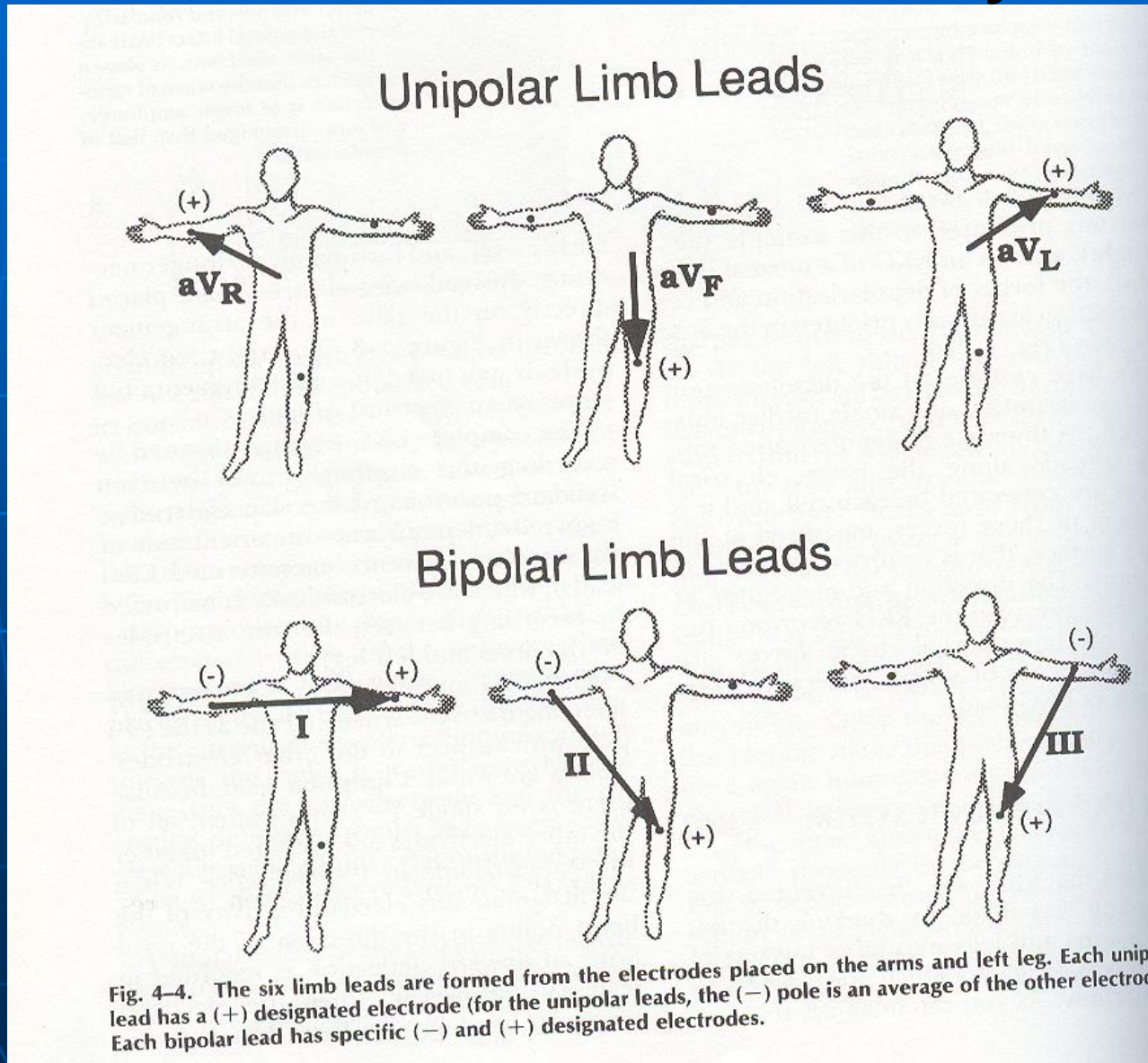


Fig. 4-3. *A*, Standard positions of the EKG electrodes. *B*, Close-up view of chest electrode placement.

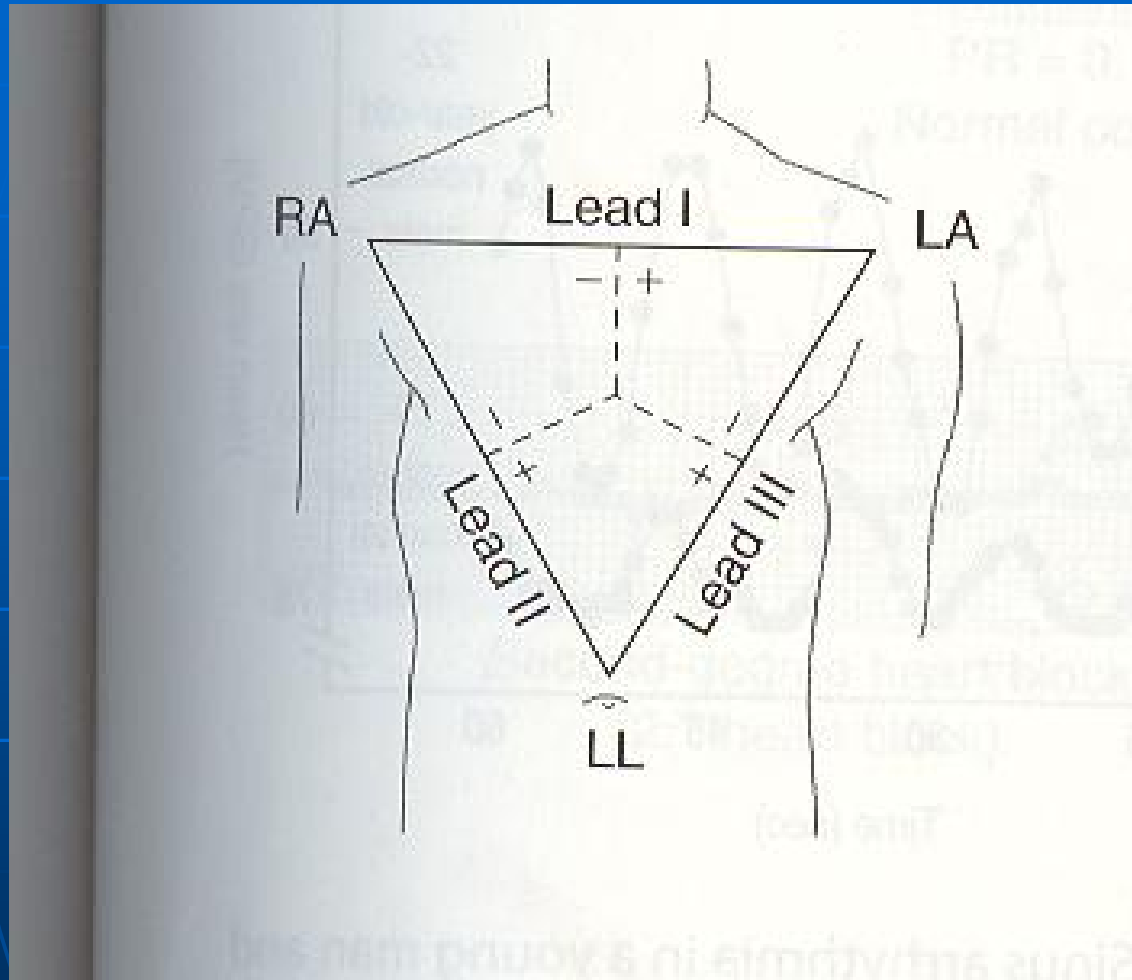


# ECG: The Lead Reference system II





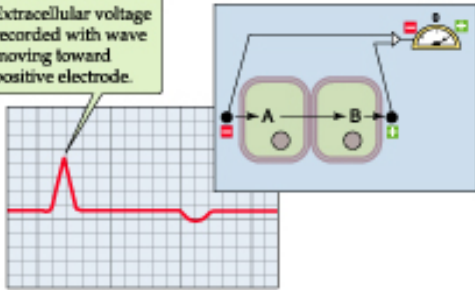
# Einthoven's Triangle



# ECG activity: Two cell model

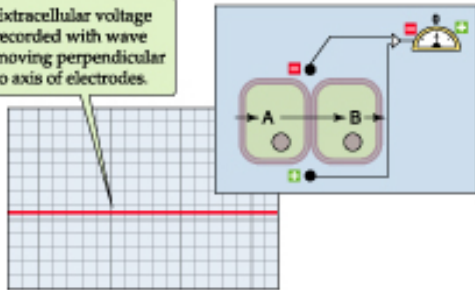
## C DEPOLARIZATION MOVING TOWARD POSITIVE ELECTRODE

Extracellular voltage recorded with wave moving toward positive electrode.



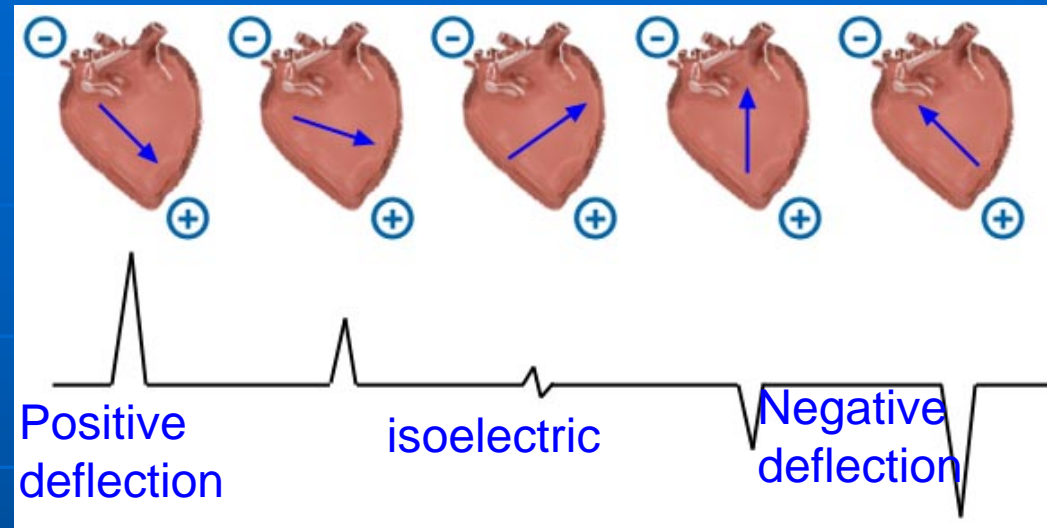
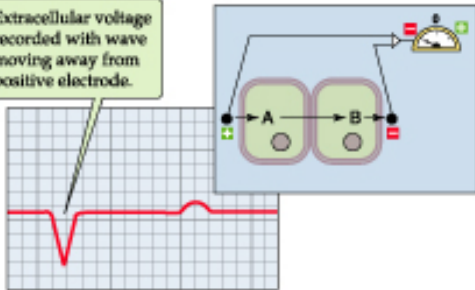
## D DEPOLARIZATION MOVING PERPENDICULAR TO ELECTRODE AXIS

Extracellular voltage recorded with wave moving perpendicular to axis of electrodes.



## E DEPOLARIZATION MOVING AWAY FROM POSITIVE ELECTRODE

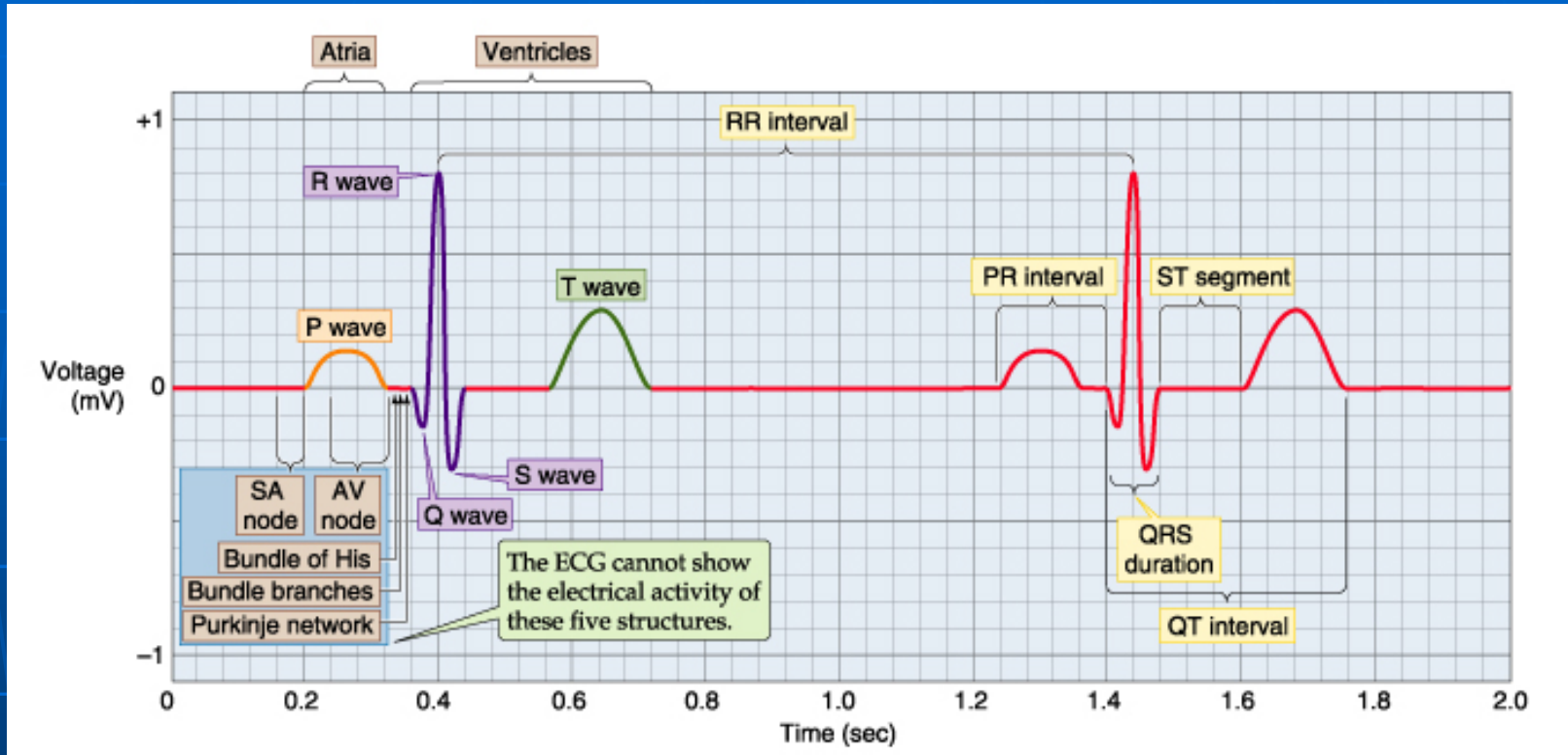
Extracellular voltage recorded with wave moving away from positive electrode.



When wave of depolarization moves towards a positive electrode, the deflection is **positive**.

# The Normal Electrocardiogram

0-6



P wave – depolarization of atria

QRS complex – depolarization of ventricular muscle

T wave – repolarization of ventricular muscle

\*repolarization of atria lies under QRS

# ECG: Interpretation

- Sequence of analysis
  1. Heart Rhythm
  2. Heart Rate
  3. Intervals (PR, QRS, ST)
  4. Abnormalities of the P wave
  5. Abnormalities of the QRS
  6. ST and T wave abnormalities

# Normal ECG: Intervals and segments

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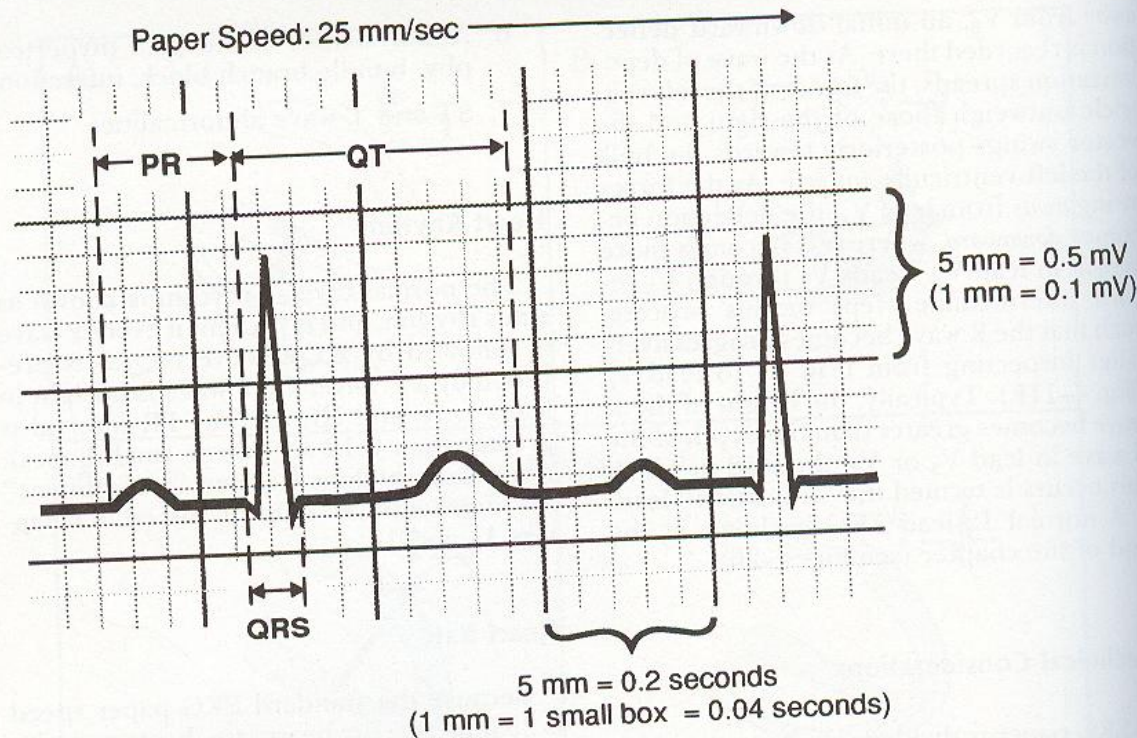


Fig. 4-12. Enlarged view of an EKG strip. The paper travels through the machine at 25 mm/sec, so that each 1 mm on the horizontal axis represents 0.04 sec. Each 1 mm on the vertical axis represents 0.1 millivolt. Interval measurements are: PR (from the beginning of the P wave to the beginning of the QRS) = 4 small boxes = 0.16 sec; QRS duration (from the beginning to the end of the QRS complex) = 4 small boxes = 0.16 sec; QT interval (from the beginning of the QRS to the end of the T waves) = 8 small boxes = 0.32 sec. The corrected QT =  $\frac{QT}{\sqrt{R-R}}$ . Because the R-R = 15 small boxes (0.6 sec), the corrected QT =  $\frac{0.32}{\sqrt{0.6}} = 0.41$  sec.

**Homework!** –  
check up  
the normal  
values of ALL  
Intervals and  
Segments



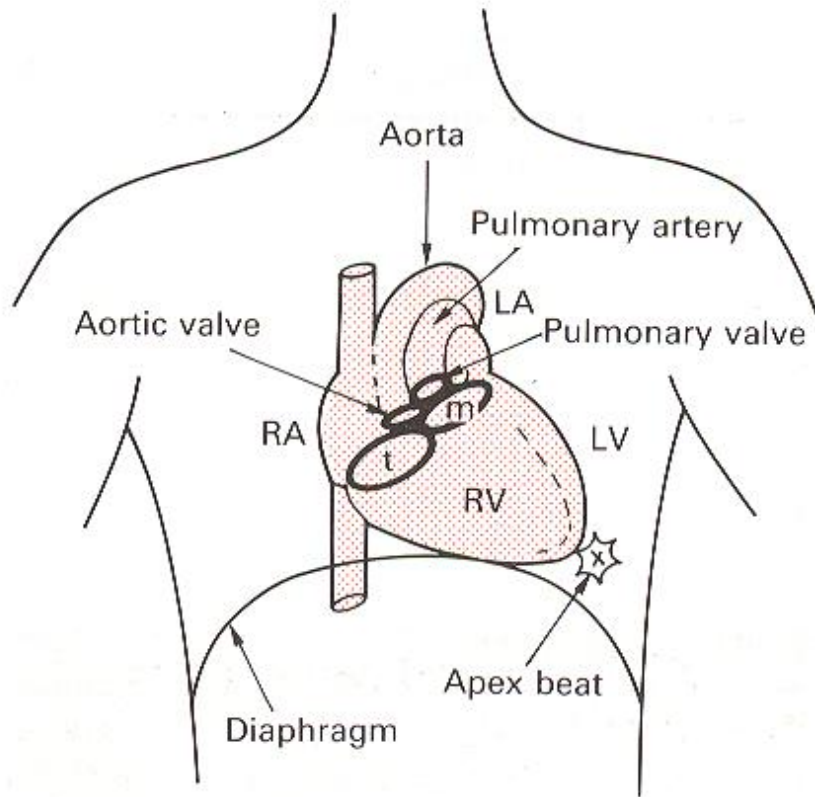
# CVS

## The Heart Mechanical Activity

### Anatomic considerations

- Heart built on a fibrotendinous ring – **Annulus fibrosus** – located at the atrioventricular junction
- Muscular atria and ventricles are attached to either side of the ring
- Ring perforated by 4 apertures each containing **a valve** (see figure)
- Fibrotendinous ring **insulates** the ventricles electrically from the atria

# The Heart: Fibrotendinous Ring

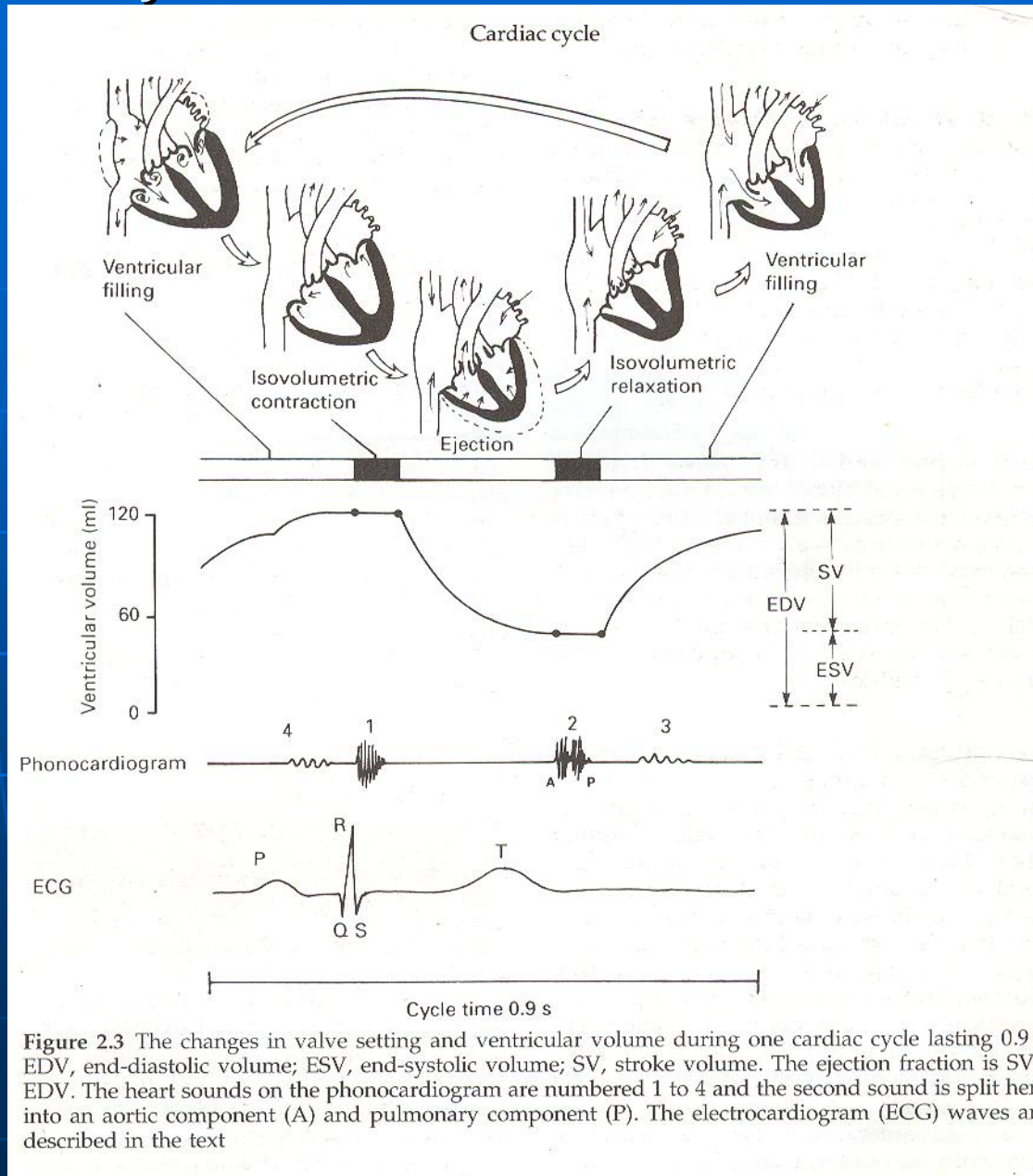


**Figure 2.1** The heart lies obliquely across the chest. The fibrotendinous ring (black) acts as a base for the heart. It contains the tricuspid (t), mitral (m), aortic and pulmonary valves grouped in an oblique plane beneath the sternum. The apex of the heart is formed by the left ventricle (LV), and the anterior surface is formed by the right ventricle (RV) and right atrium (RA). The inferior surface of the heart and the pericardium (not shown) rest on the central tendon of the diaphragm

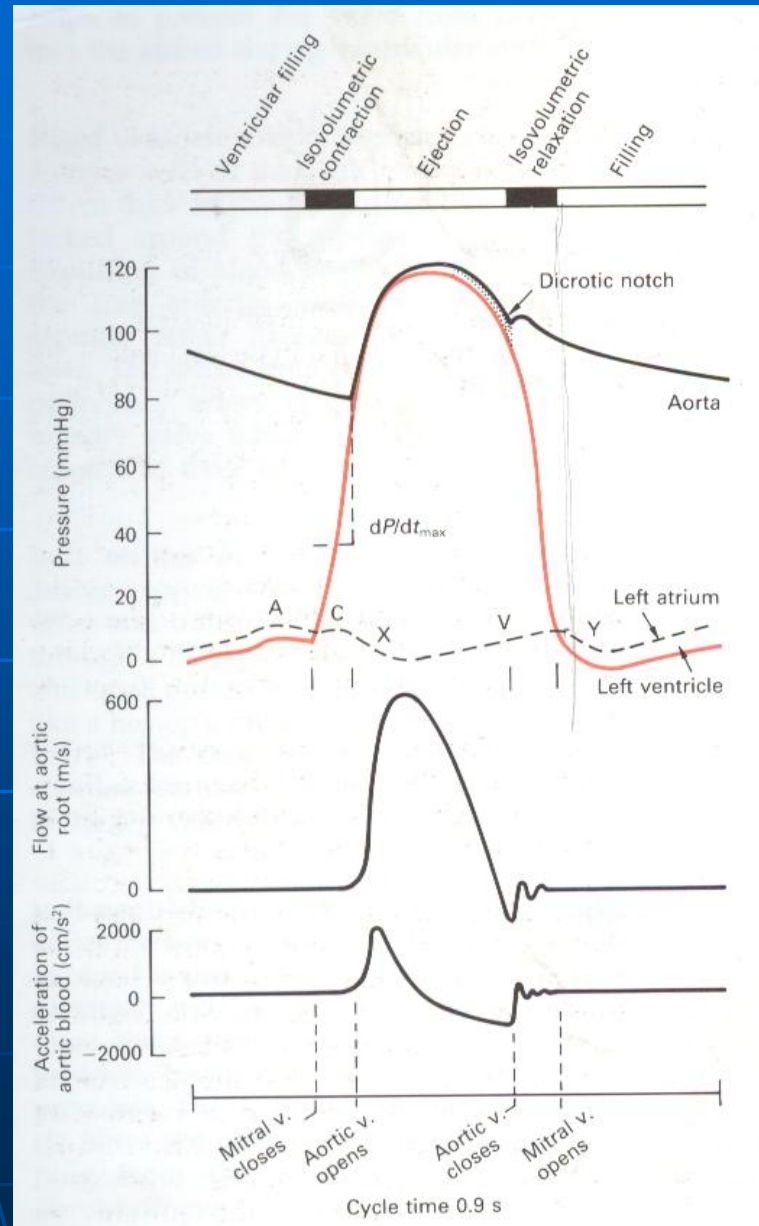
# Mechanical events of the Cardiac cycle

- Atria and ventricles contract in sequence resulting in a cycle of pressure and volume
- Cardiac cycle has 4 phases (arbitrary)
  - (i) ventricular filling
  - (ii) Isovolumetric contraction
  - (iii) Ejection
  - (iv) Isovolumetric relaxation
- **Figure** based on human cycle – 0.9 sec duration, (67 beats/min)

# Cardiac cycle: valve and volume changes



# Pressure and outflow: left ventricle





# Ventricular filling (Diastole)

- Duration 0.5 sec
- Inlet valves (tricuspid and mitral): open
- Outlet valves (pulmonary and aortic): closed
- Lasts about two-thirds of cardiac cycle
- Rapid filling – coincides with atrial diastole – lasts 0.15 sec
- Atrial systole – boosts filling by 15-20%
- End-diastolic volume: volume of blood in the ventricle at the end of filling  $\approx$  120 mls
- Corresponding pressure is End-diastolic pressure – just a few mm Hg

# Isovolumetric contraction

- Duration: 0.05 sec
- Inlet valves: closed
- Outlet valves: closed
- Ventricle is a closed chamber
- Pressure rises steeply

# Ejection

- Duration: 0.3 sec
- Inlet valves: closed
- Outlet valves: open
- Outflow valves open when ventricular pressure exceeds arterial pressure
- $\frac{3}{4}$  of Stroke volume is ejected in the first phase of ejection – rapid phase – 0.15 sec
- Ventricle empties just about two-thirds – average ejection fraction is approx 0.67 – stroke volume of about 70-80 ml

# Isovolumetric relaxation

- Duration: 0.08 sec
- Inlet valves: closed
- Outlet valves: closed
- Ventricle – closed chamber
- Ventricular pressure falls very rapidly
- A-V valves open when ventricular pressure falls below atrial pressure

# The ventricular pressure-volume loop

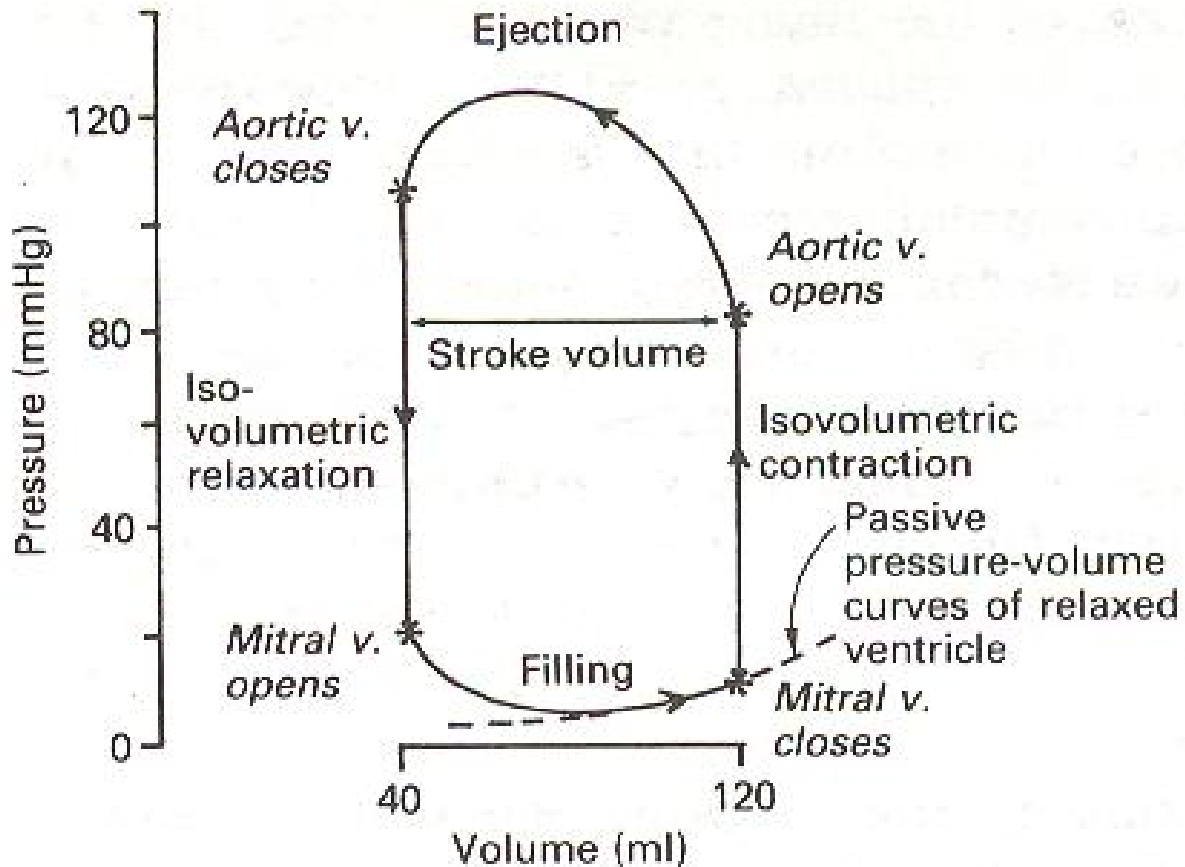


Figure 2.5 Pressure-volume cycle of human left ventricle



# Heart Sounds

- Two sounds typically heard through a stethoscope – “**lub**” first heart sound and “**dup**” second sound.
- **First** : caused by vibrations from sudden closure of the mitral and tricuspid valves
- **Second**: vibrations associated with closure of aortic and pulmonary valves
- **Third**: heard in many normal young persons – one third into diastole –rapid ventricular filling phase
- **Fourth**: heard just before first sound – when atrial pressure is high or ventricular hypertrophy –rarely heard in normal adults

# Effect of heart rate on phase duration

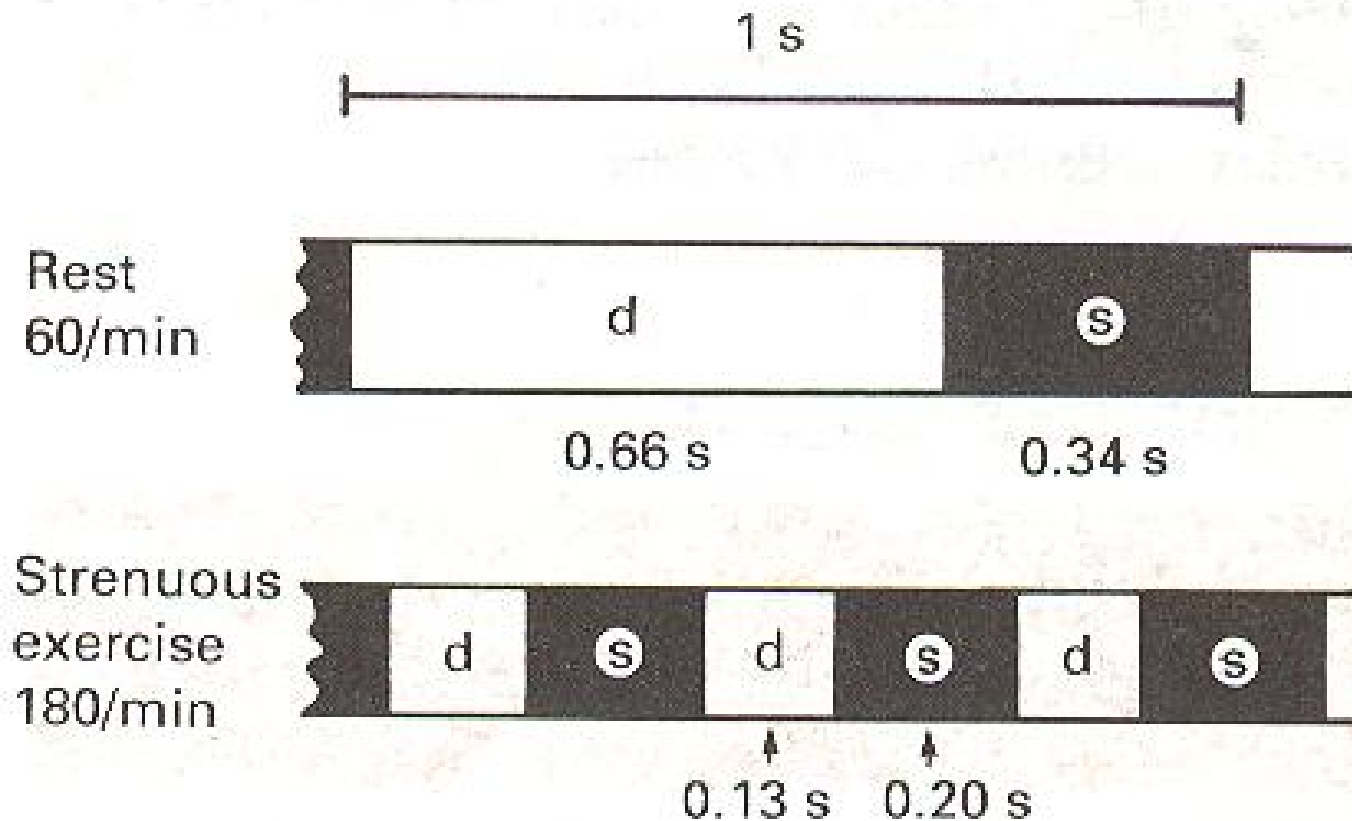


Figure 2.6 Effect of heart rate on the diastolic period available for filling, d, diastole; s, systole. Diastole is curtailed more than systole as heart rate increases

# Homework

- Read about:
  - Arterial pressure
  - Atrial pressure changes
  - The Jugular Pulse

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# Intervals and Segments

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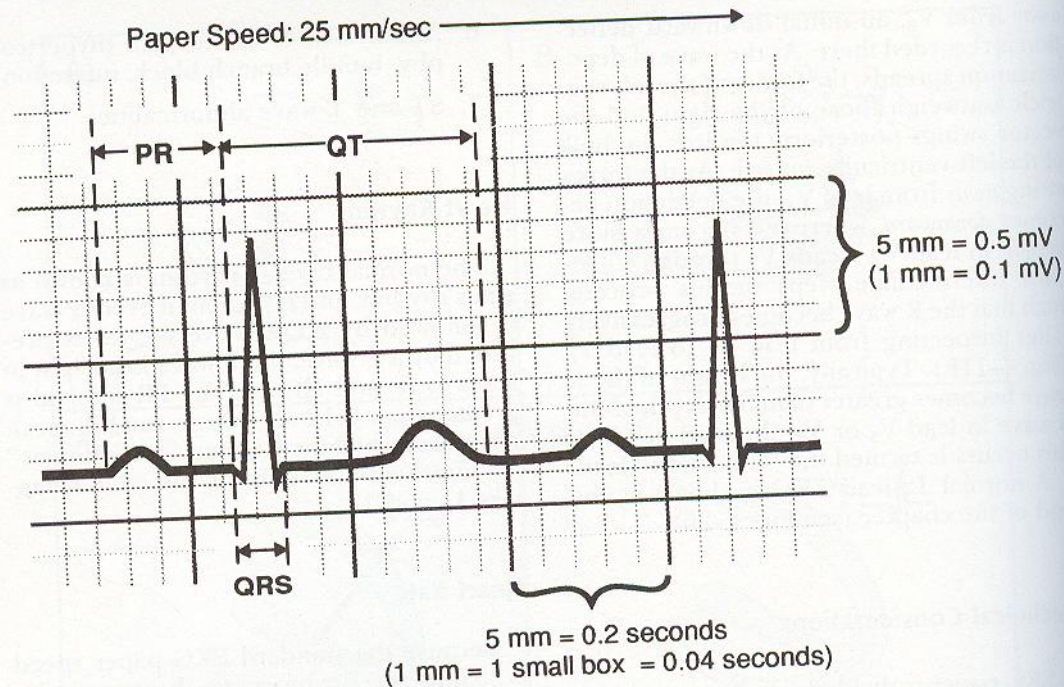
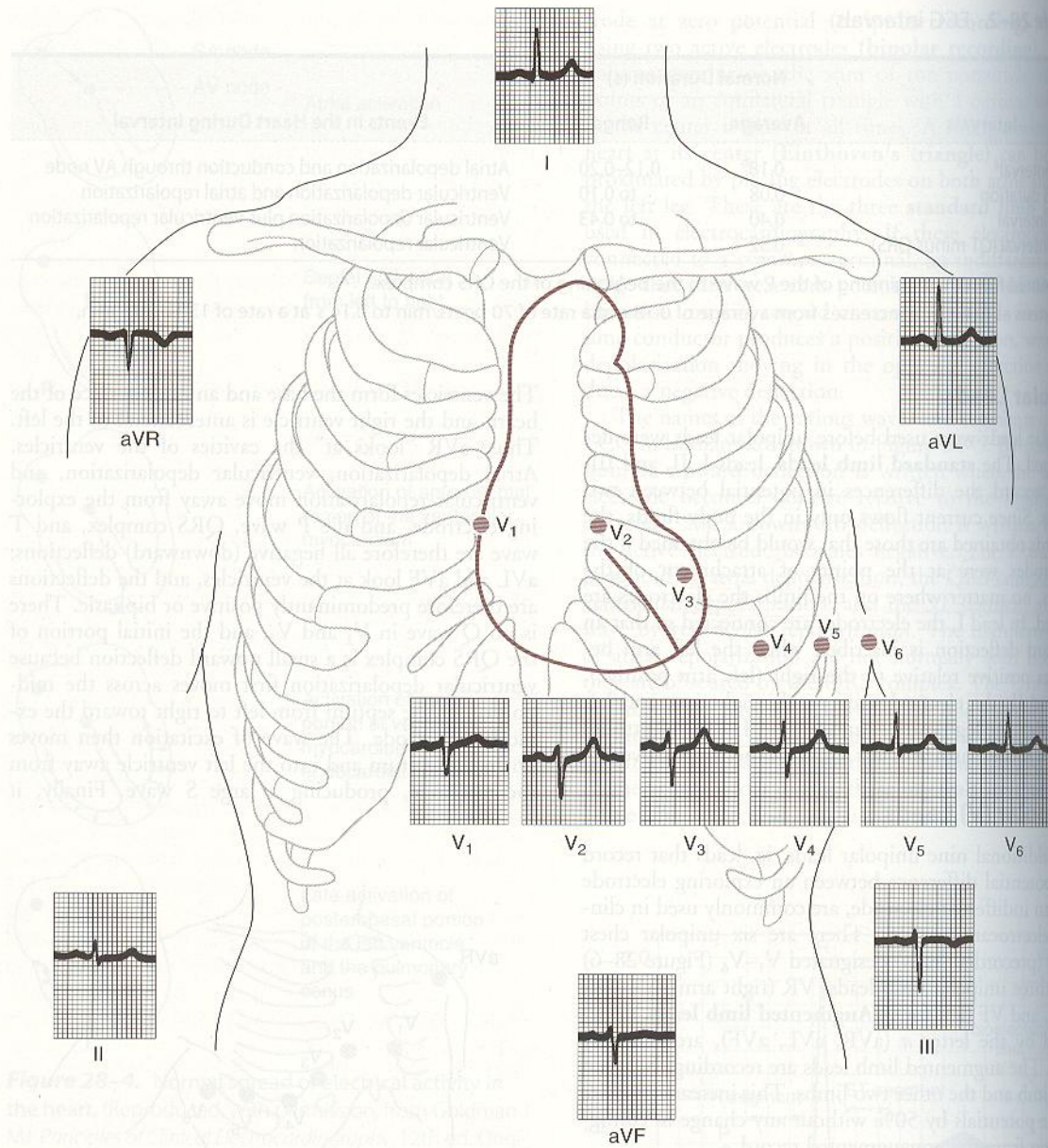


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**Figure 28-7.** Normal ECG. (Reproduced, with permission, from Goldman MJ: *Principles of Clinical Electrocardiography*, 12th ed. Originally published by Appleton & Lange. Copyright © 1986 by The McGraw-Hill Companies, Inc.)