ELECTROPHYSIOLOGY OF HEART

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CONTENTS

PHYSIOLOGY OF CARDIAC MUSCLE

INTRODUCTION

Three Major type of cardiac muscle fiber:-

- ✓ Atrial muscle
- ✓ Ventricular muscle
- ✓ Specialized excitatory and conductive muscle fibers

- ✓ Atrial and ventricular muscle contract in a same way as skeletal muscle but the duration of contraction is longer
- ✓ The specialized excitatory and conductive fibers
- Contract feebly because they contain few contractile fibrils
- Exhibit automatic rhythmical electrical discharge in the form of action potentials

CARDIAC MUSCLE AS SYNCYTIUM

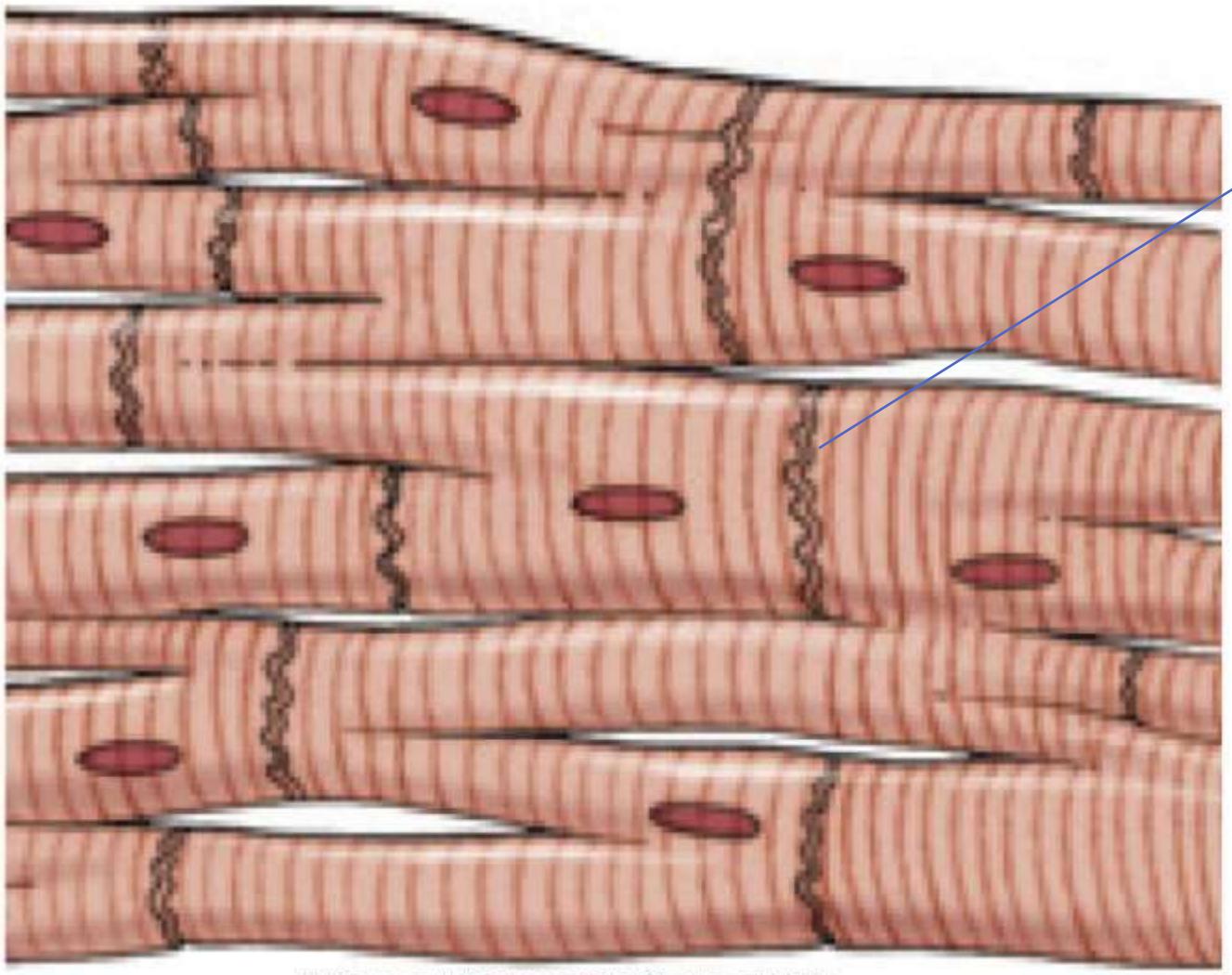


Figure 9-2 "Syncytial," interconnecting nature of cardiac muscle fibers.

- ✓ Intercalated discs are cell membranes that separate individual cardiac muscle cells from one another.

- ✓ At each intercalated disc there are communicating gap junctions that allow rapid diffusion of ions so that action potentials travel easily from one cardiac muscle cell to the next

ACTION POTENTIAL IN CARDIAC MUSCLE

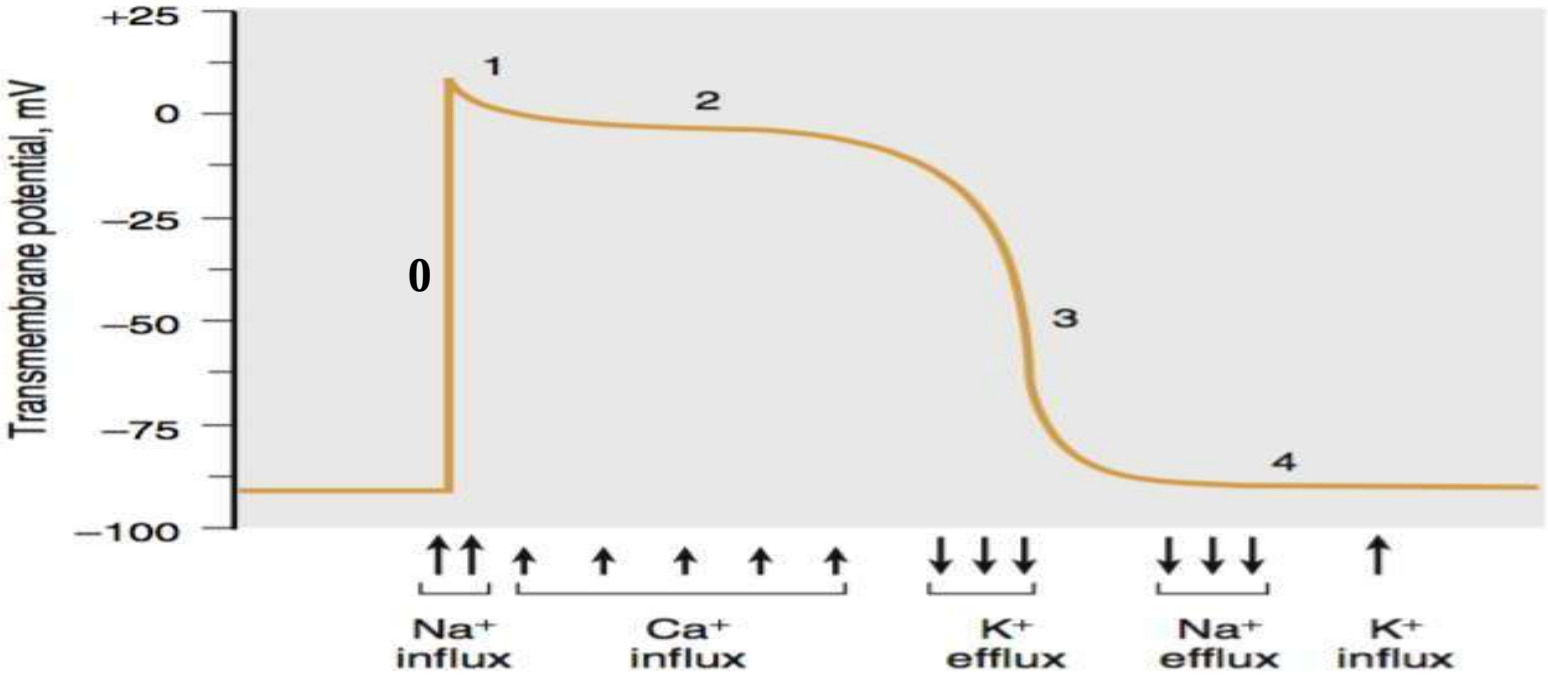


Figure 20-11. Phases of cellular action potentials and major associated currents in ventricular myocytes. The initial phase (0) spike and overshoot (1) are caused by a rapid inward sodium (Na^+) current, the plateau phase (2) by a slow calcium (Ca^{2+}) current through L-type Ca channels, and repolarization (phase 3) by outward potassium (K^+) currents. Phase 4, the resting potential (Na^+ efflux, K^+ influx), is maintained by Na^+-K^+-adenosine triphosphatase (ATPase). The Na^+-Ca^{2+} exchanger is mainly responsible for extrusion of Ca^{2+}. In specialized conduction system tissue, spontaneous depolarization takes place during phase 4 until the voltage resulting in opening of the Na channel is reached. *(From LeWinter MM, Osol G: Normal physiology of the cardiovascular system. In Fuster V, Alexander RW, O'Rourke RA, editors:* Hurst's the heart, *ed 10. New York, 2001, McGraw-Hill, pp 63-94.)*

Phase 0 (rapid depolarization)

External stimulus to excitable tissue

↓

opens the voltage gated sodium ion channels

↓

sodium ions enter the cells down their electrochemical gradient

↓

intracellular movement of sodium ion depolarizes the membrane

↓

increases the membrane conductance to sodium ion

↓

displaces the membrane potential to +30mV

Phase 1 (early repolarization)

- Following phase 0, the membrane repolarizes rapidly and transiently to almost 0 mV because of the inactivation of sodium ion channel and simultaneous transient increases in outward potassium currents

Phase 2 (plateau)

- Membrane potential remains approximately 0 mV for a relatively prolonged duration

- A balance between slow inward Ca2+ and outward K+ currents mediates the plateau phase of the action potential.

Phase 3 (repolarization)

- Inactivation of Ca2+ channels and a simultaneous increase in outward K+ current through K+ channels produces a net outward movement of positive charge and repolarization of the membrane

Phase 4 (resting membrane potential)

- The membrane potential of ventricular myocytes remains at the resting membrane potential until the cell is stimulated again

The types of action potential in the heart can be separated into two categories:

(1) **fast-response action potentials**, which are found in the His-Purkinje system and atrial or ventricular cardiomyocytes

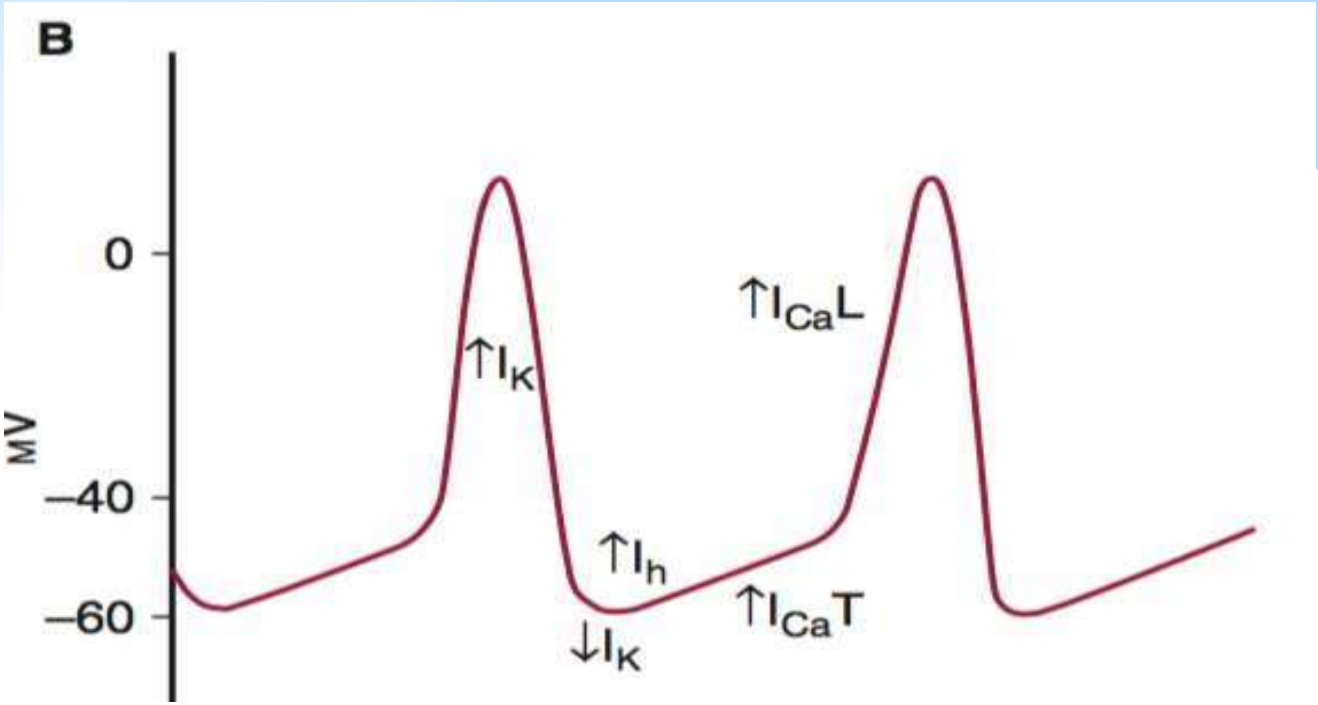
(2) **slow- response action potentials**, which are found in the pacemaker cells in the SA and AVnodes

- **Two types of action potential in the heart**:-

1. **fast-response action potentials:-** His-Purkinje system and atrial or ventricular muscle

2. **slow- response action potentials:-** pacemaker cells in the SA and AVnodes

PACEMAKER POTENTIAL



- Ik:- K+ current
- Ih:- Funny current/’f’channel
- ICaT:- Transient Ca2+ channel
- ICaL:-Long lasting Ca2+ channel

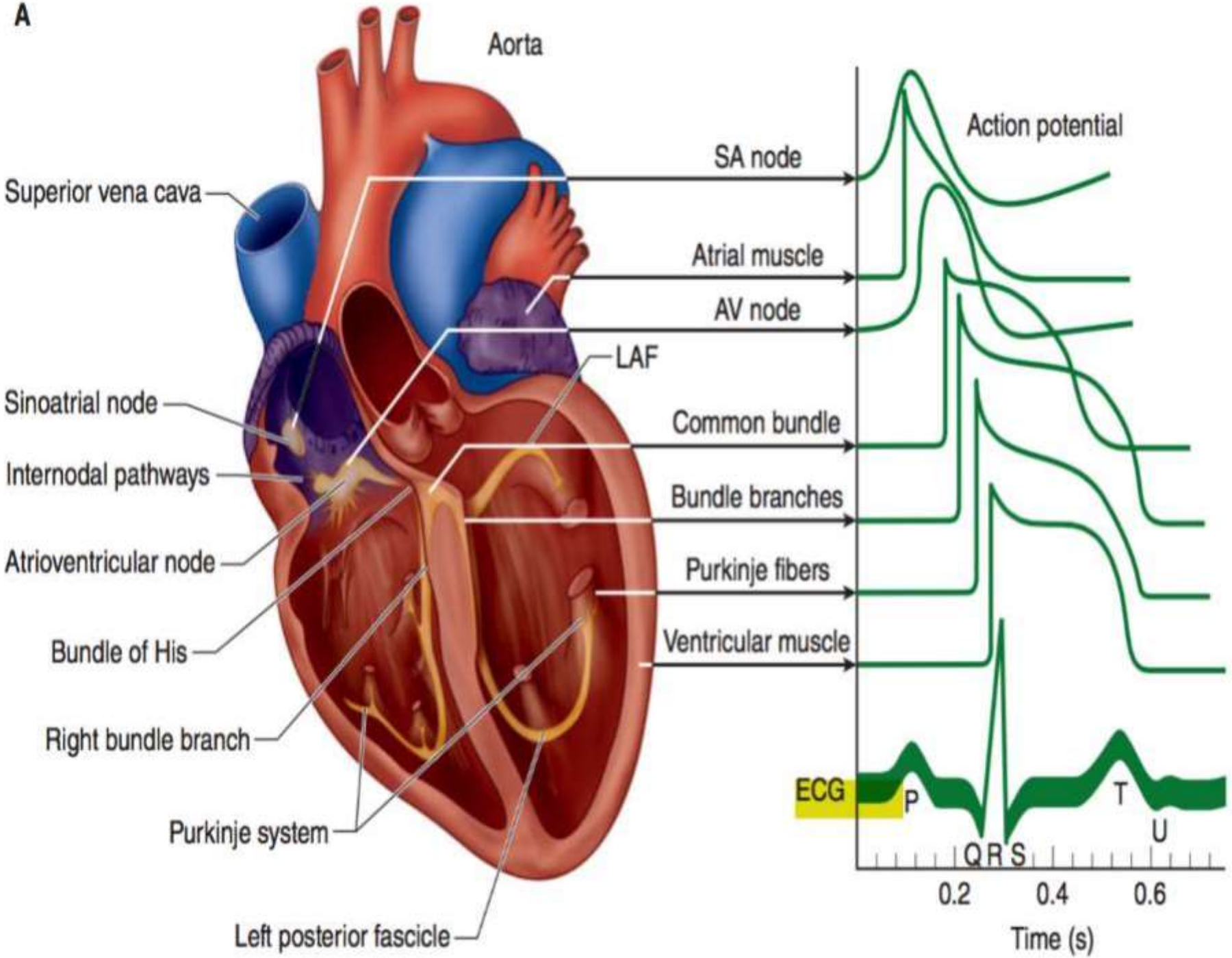

A
Aorta
Superior vena cava
Sinoatrial node
Internodal pathways
Atrioventricular node
Bundle of His
Right bundle branch
Purkinje system
Left posterior fascicle
LAF
SA node
Atrial muscle
AV node
Common bundle
Bundle branches
Purkinje fibers
Ventricular muscle
Action potential
ECG
P
Q R S
T
U
0.2
0.4
0.6
Time (s)

TABLE 20–1 Cardiac action potential.

Phase	Name	Event	Cellular Ion Movement
0	Upstroke	Activation (opening) of voltage-gated Na^+ channels	Na^+ entry and decreased permeability to K^+
1	Early rapid repolarization	Inactivation of Na^+ channel and transient increase in K^+ permeability	K^+ out (I_{To})
2	Plateau	Activation of slow calcium channels	Ca^{2+} entry
3	Final repolarization	Inactivation of calcium channels and increased permeability to K^+	K^+ out
4	Resting potential	Normal permeability restored (atrial and ventricular cells)	Na^+–K^+-ATPase pumps K^+ in and Na^+ out
	Diastolic repolarization	Intrinsic slow leakage of Ca^{2+} into cells that spontaneously depolarize	Ca^{2+} in

Specialized Excitatory and Conductive System of the Heart

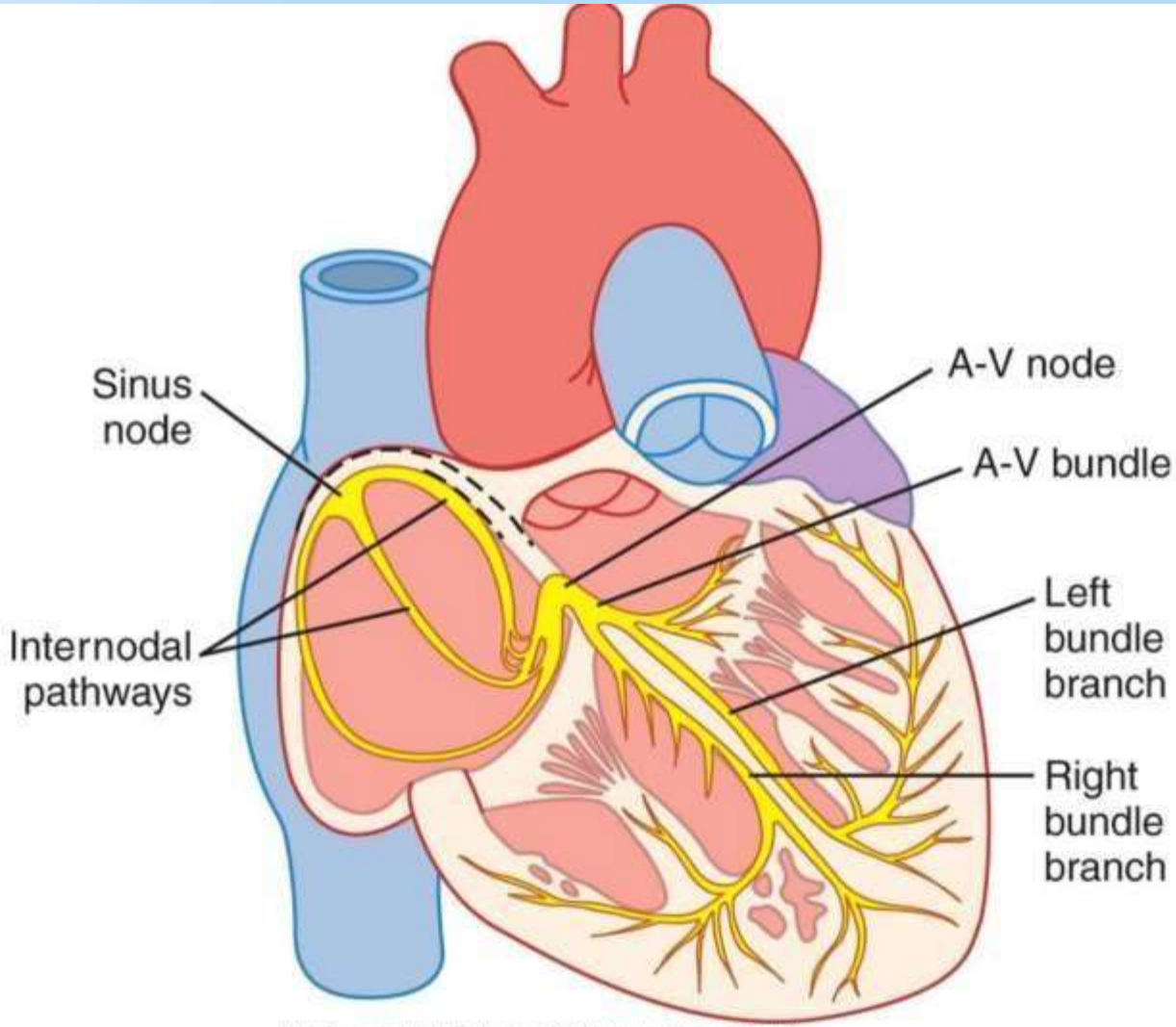


Figure 10-1 Sinus node and the Purkinje system of the heart, showing also the A-V node, atrial internodal pathways, and ventricular bundle branches.

A. SA NODE

- The sinus node/ sinoatrial node is located in the superior posterolateral wall of the right atrium immediately below and lateral to the opening of the superior vena cava

- The sinus nodal fibers connect directly with the atrial muscle fibers so that any action potential that begins in the sinus node spreads immediately into the atrial muscle wall

❑ Mechanism of Sinus Nodal Rhythmicity

- Resting membrane potential is -55mV. At this -55mV,the fast sodium ion channel closes but slow sodium and calcium channels remain open and there by causing action potential

❑ Self-Excitation of Sinus Nodal Fibers

- The inherent leakiness of the sinus nodal fibers to sodium and calcium ions causes their self-excitation

❑ Automatic Electrical Rhythmicity of the Sinus Fibers

- Because of self excitation, sinus fibers has automatic rhythmical discharge and contraction

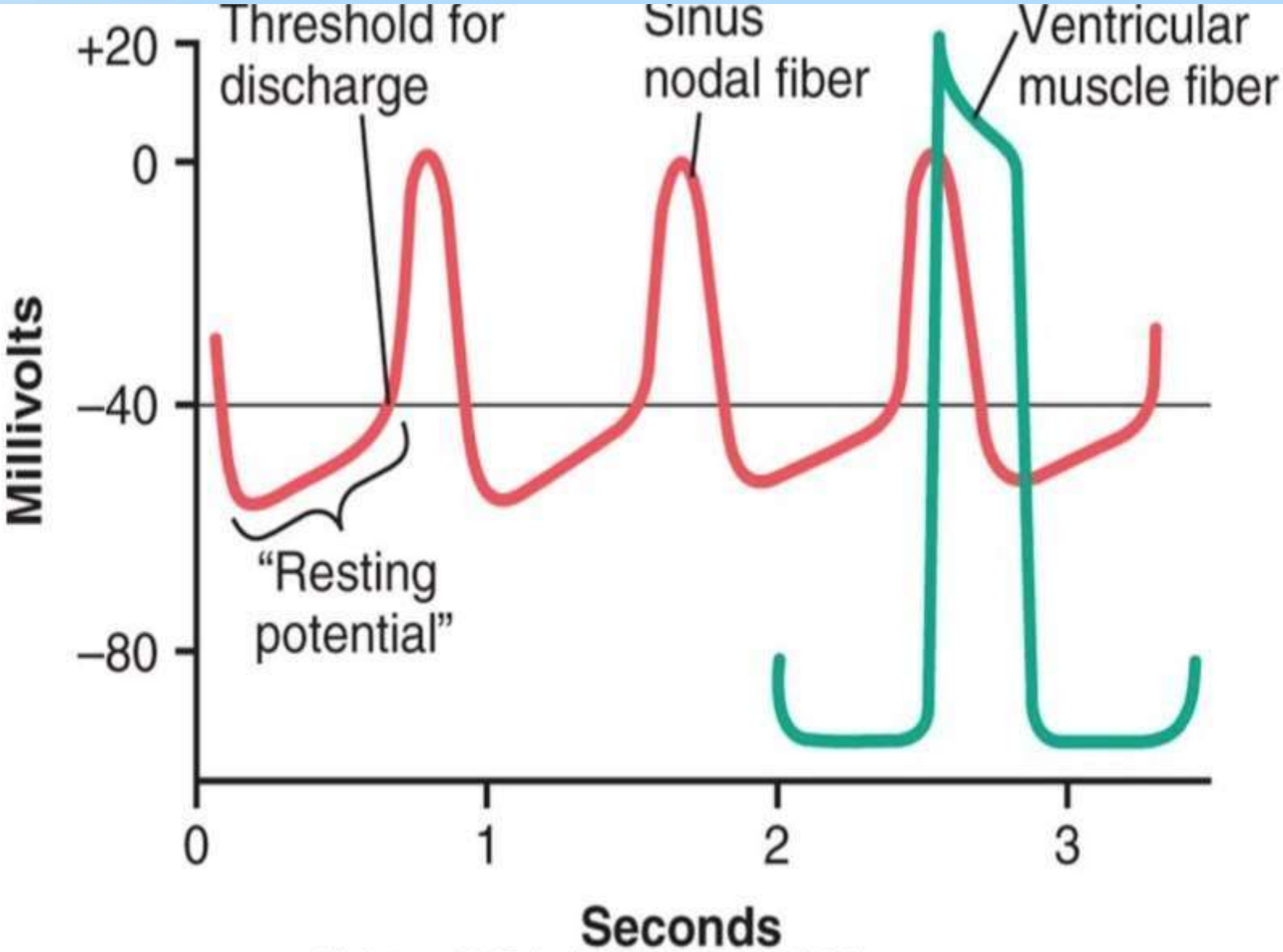


Figure 10-2 Rhythmical discharge of a sinus nodal fiber. Also, the sinus nodal action potential is compared with that of a ventricular muscle fiber.

B. INTERNODAL PATHWAYS

- Anterior (Bachmann)
- Middle(Wenckebach)
- Posterior(Thorel) internodal pathways

C. AV NODE

- The A-V node is located in the posterior wall of the right atrium immediately behind the tricuspid valve

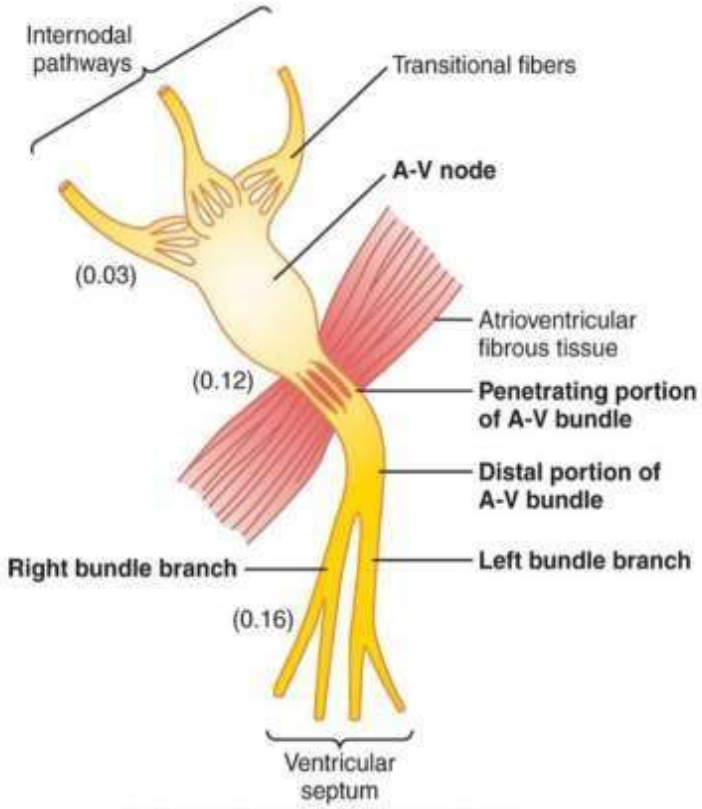
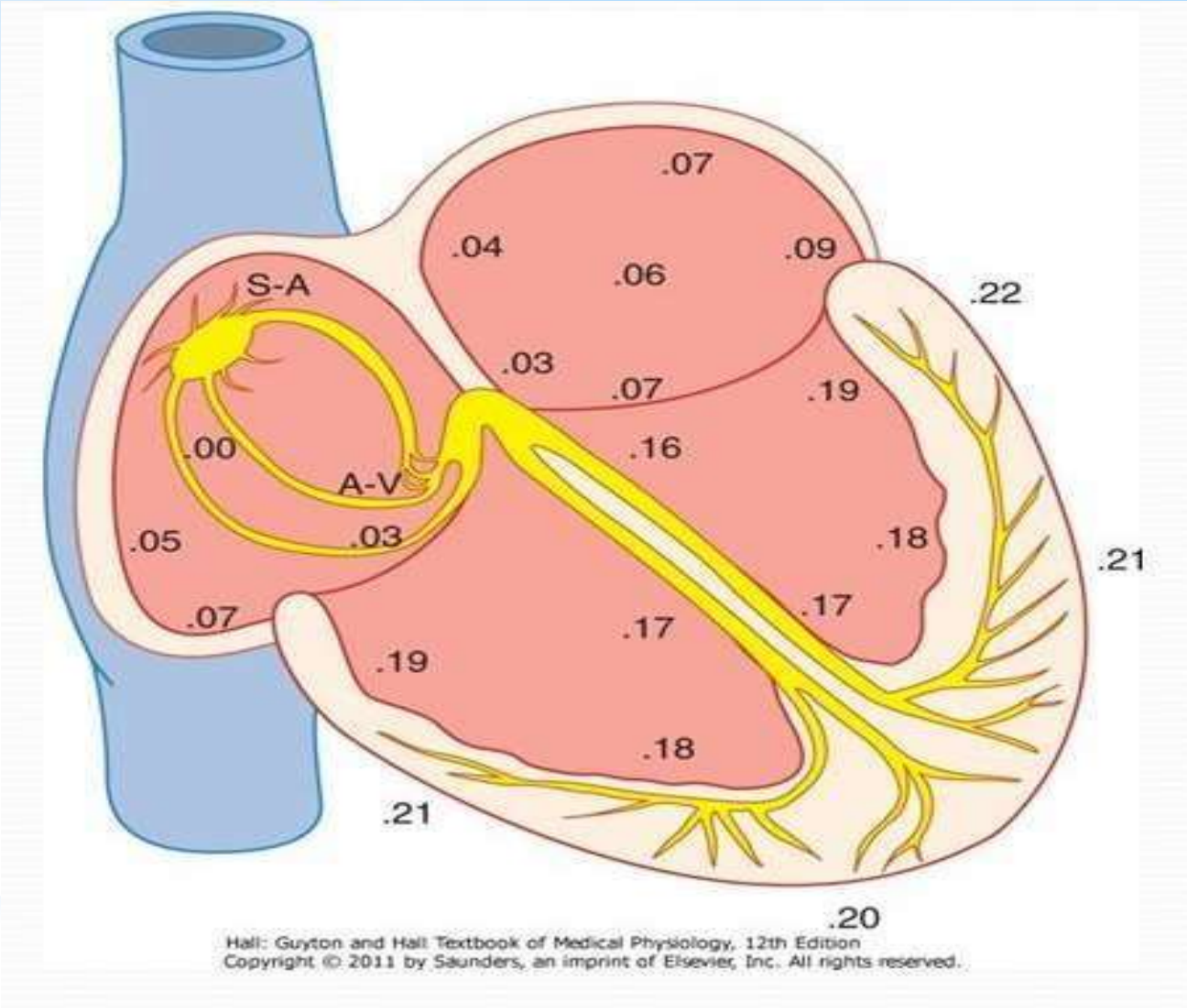


Figure 10-3 Organization of the A-V node. The numbers represent the interval of time from the origin of the impulse in the sinus node. The values have been extrapolated to human beings.

D.PURKINJE FIBERS

- Velocity of conduction is 1 to 4m/sec
- The ends of the Purkinje fibers penetrate about one third of the way into the muscle mass and finally become continuous with the cardiac muscle fibers
- Once the impulse reaches the ends of the Purkinje fibers, it is transmitted through the ventricular muscle mass by the ventricular muscle fibers themselves. The velocity of transmission is now only 0.3 to 0.5 m/sec, one sixth that in the Purkinje fibers.

TRANSMISSION OF CARDIAC IMPULSE THROUGH THE HEART



CONDUCTION SPEEDS IN CARDIAC TISSUES

SA Node	0.05m/sec
Atrial pathways	1m/sec
AV Node	0.05m/sec
Bundle of his	1m/sec
Purkinje fibers	4m/sec
Ventricular muscle	1m/sec

ELECTROCARDIOGRAM(ECG)

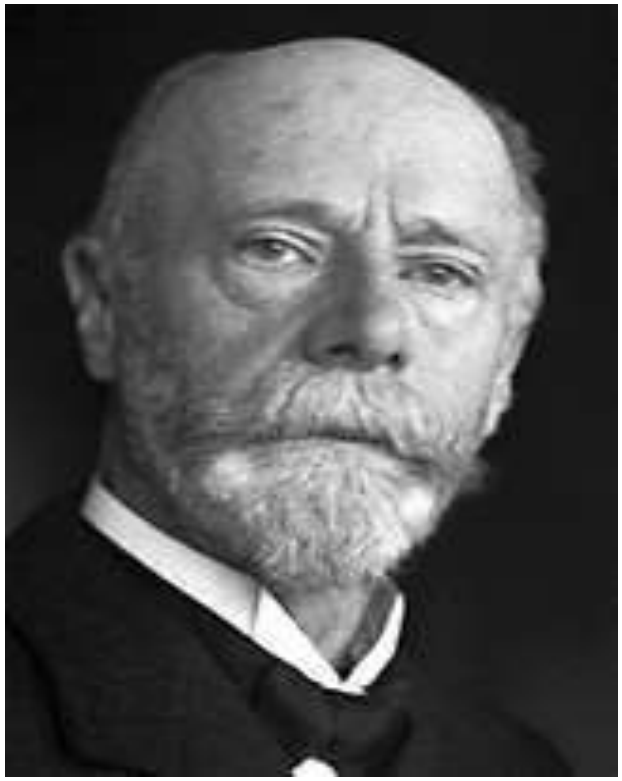
HISTORY

- 1842- Italian scientist Carlo Matteucci realizes that electricity is associated with heart

- 1876- Irish scientist Marey analyzes electrical pattern of frog's heart

- 1887- The first electrocardiogram (ECG) from the intact human heart was recorded with a mercury capillary electrometer by Augustus Waller . The tracings were poor and exhibited only 2 distorted deflections

- Willem Einthoven (1860-1927) was awarded the Nobel Prize in 1924 in physiology and medicine, "for the discovery of the mechanism of the electrocardiogram."



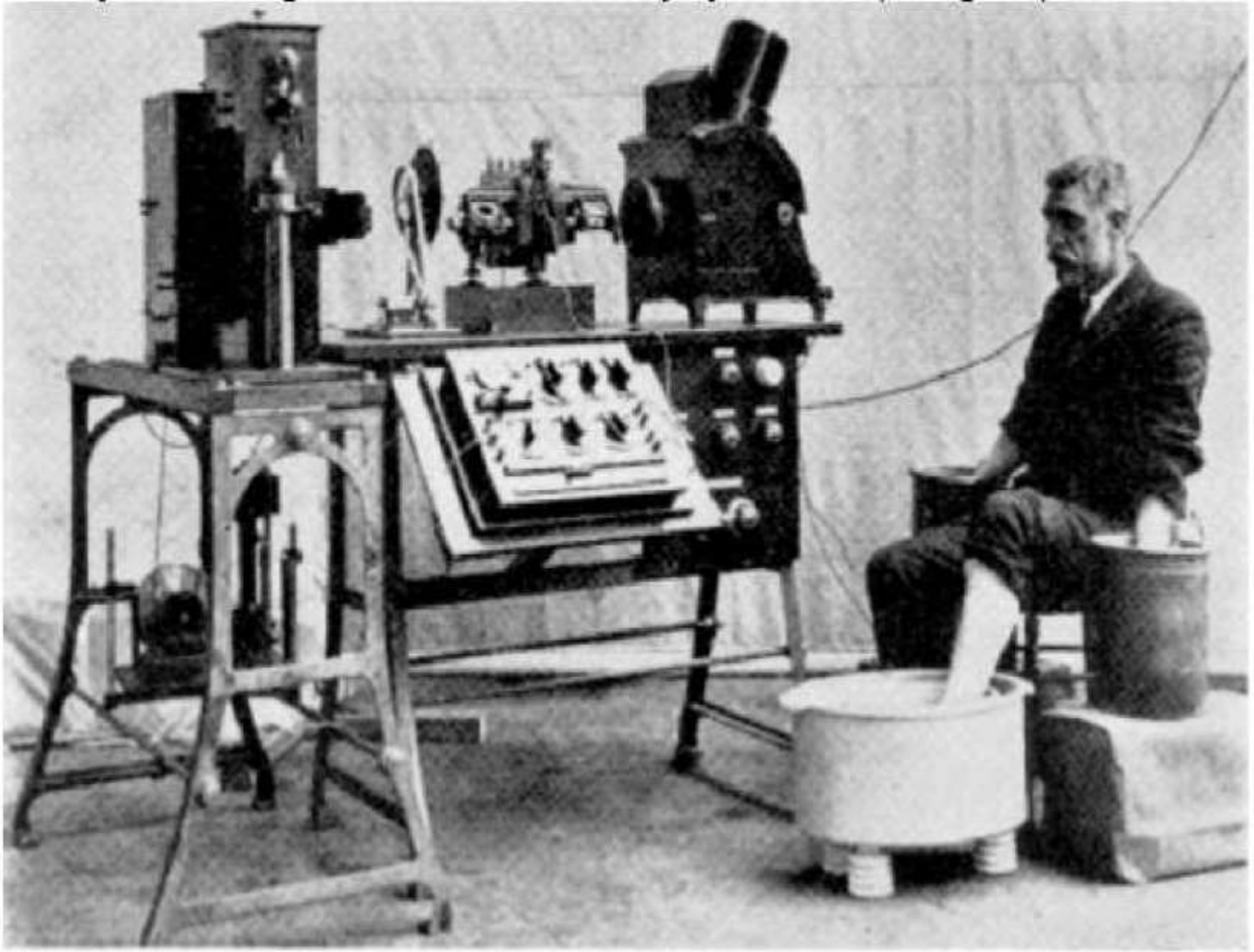


Fig. 6. The first table-model Einthoven electrocardiograph manufactured by the Cambridge Scientific Instrument Company of London in 1911. On the right hand side the arch lamp, in the centre on the table the string galvanometer, and below the switching board for the leads, next left to the camera the timer (rotating wheel with spokes), and on the left hand side the falling-plate camera (from Burch, De Pasquale, A History of Electrocardiography p 33 [14]).

WHAT IS ECG?

- Electrocardiogram/ECG/EKG is the graphical representation of electrical events of heart
- Each event has a distinctive waveform
- When the cardiac impulse passes through the heart, electrical current also spreads from the heart into the adjacent tissues surrounding the heart

CHARACTERSTICS OF NORMAL ELECTROCARDIOGRAM

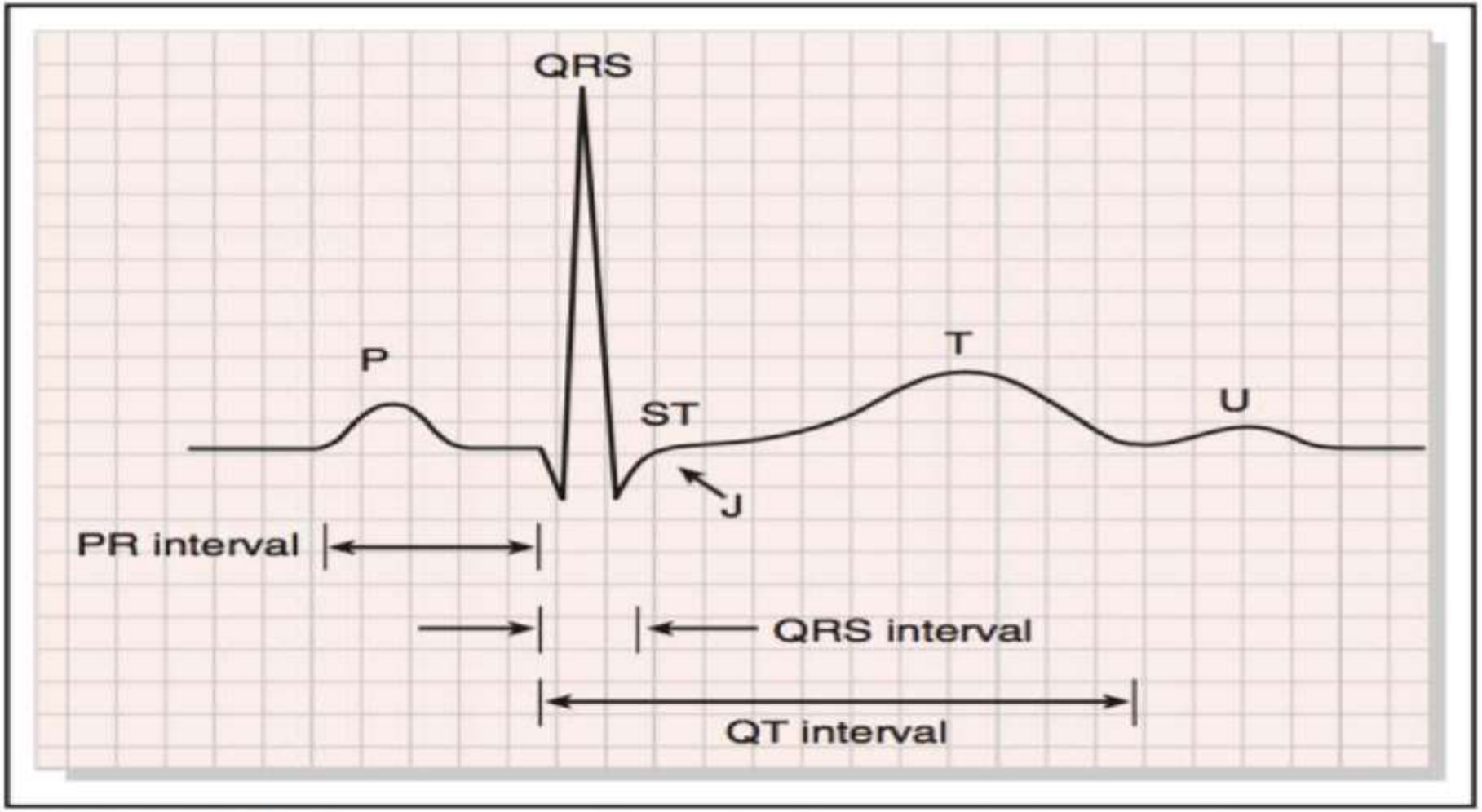


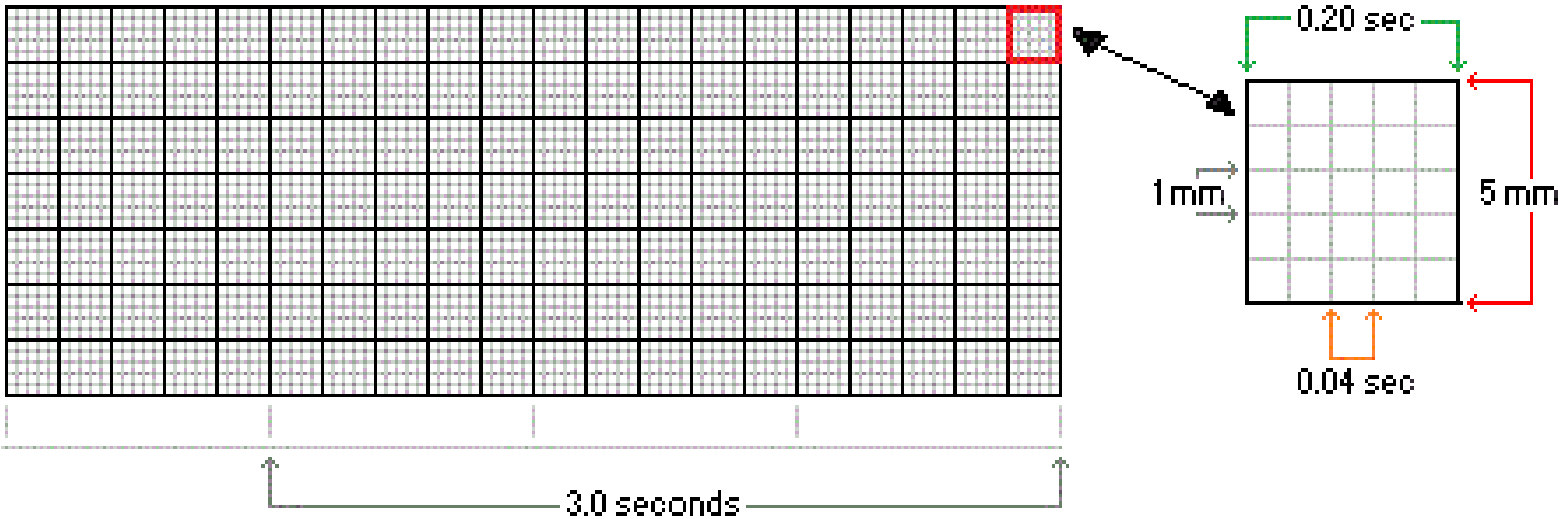
FIGURE 13-8 The waves and intervals of a normal electrocardiogram. *(From Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 7th ed. St. Louis, CV Mosby, 2006.)*

- The normal electrocardiogram is composed of a P wave, a QRS complex, and a T wave.
- The P wave is caused by electrical potentials generated when the atria depolarize before atrial contraction begins.
- The QRS complex is caused by potentials generated when the ventricles depolarize before contraction, that is, as the depolarization wave spreads through the ventricles.
- Therefore, both the P wave and the components of the QRS complex are depolarization waves.

- J point is the point where QRS complex ends
- The T wave is caused by potentials generated as the ventricles recover from the state of depolarization
- In some ECGs an extra wave can be seen on the end of the T wave, and this is called a U wave. It represent repolarization of the papillary muscles.
- If a U wave follows a normally shaped T wave, it can be assumed to be normal. If it follows a flattened T wave , it may be pathological

THE ECG PAPER

- ✓ ECG Machines runs at a standard rate of 25mm/s and use paper with standard-sized squares
- ✓ Horizontally, one large box=0.2 sec and one small box=0.04 sec
- ✓ Vertically, one large box=0.5mV



ELECTROCARDIOGRAPHIC LEADS

- Electrocardiographic leads measures the electrical potential between the two points

- **Bipolar leads:-** electrocardiogram is recorded from two electrodes located on different sides of the heart

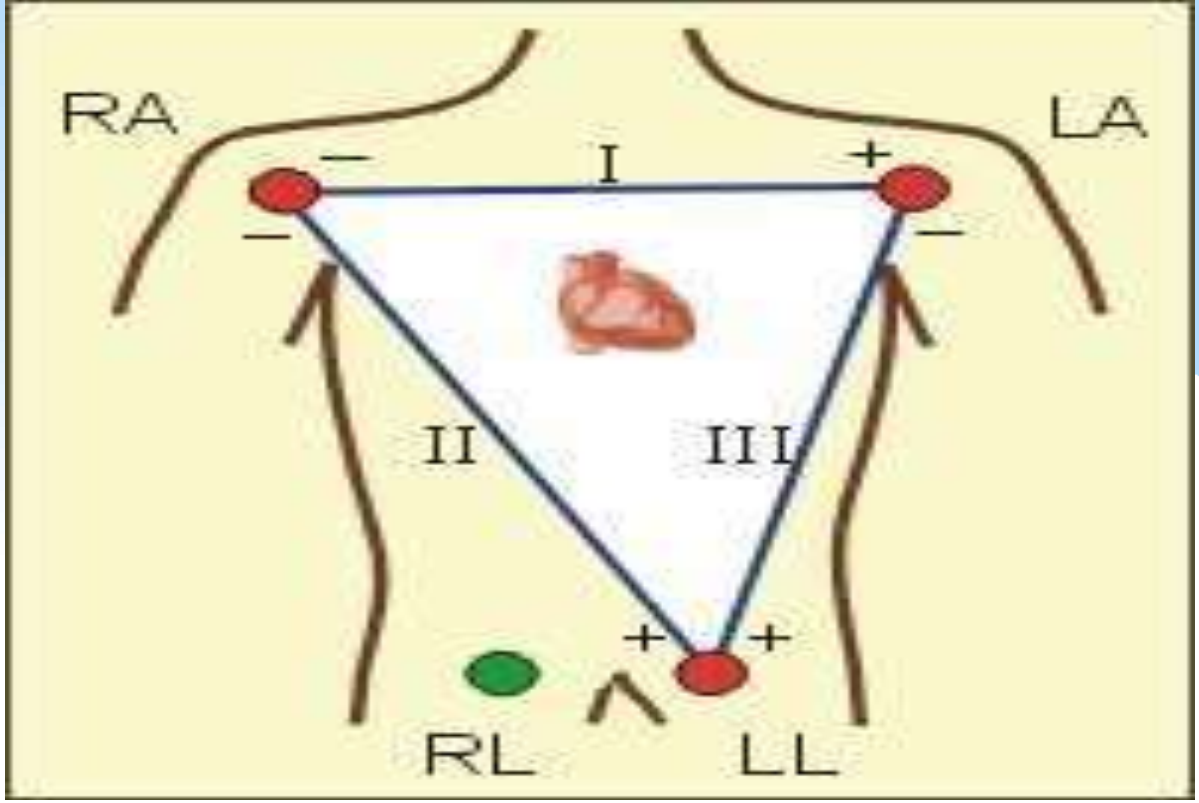
- **Unipolar leads:-** one point on the body and a virtual reference point with zero electrical potential located in the center of heart

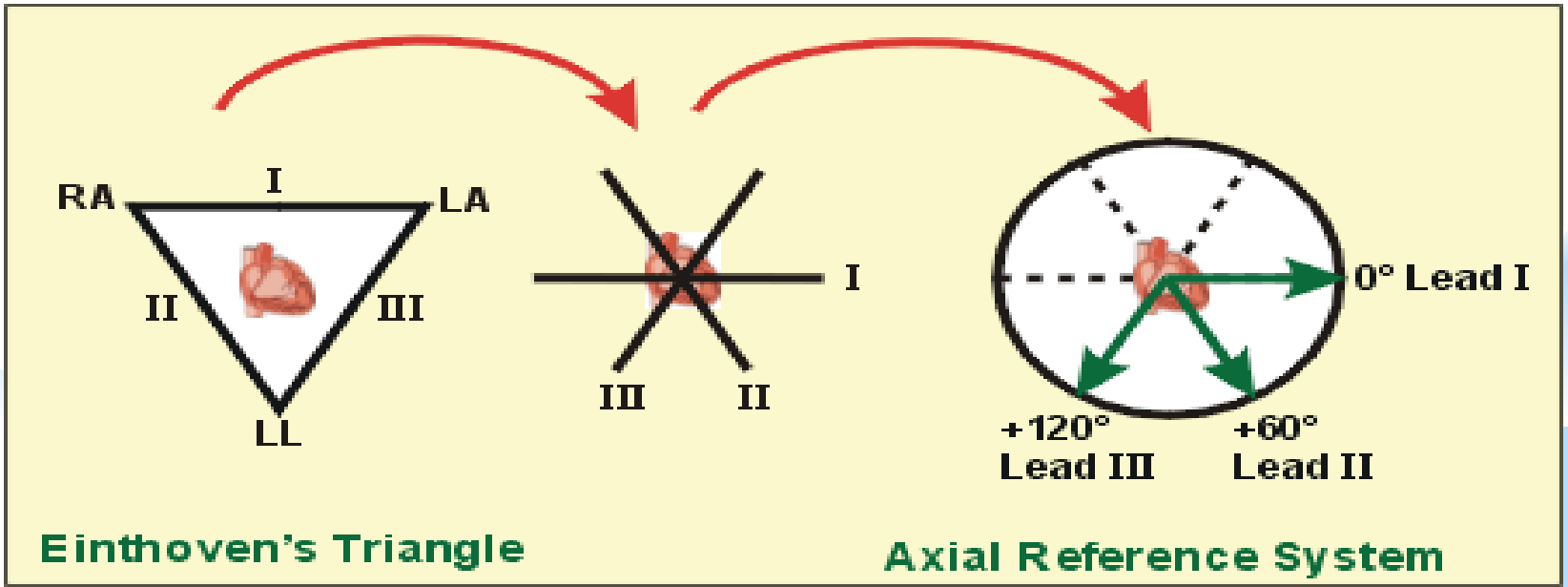
The standard ECG has 12 leads:-

1.Three standard limb leads(Bipolar)

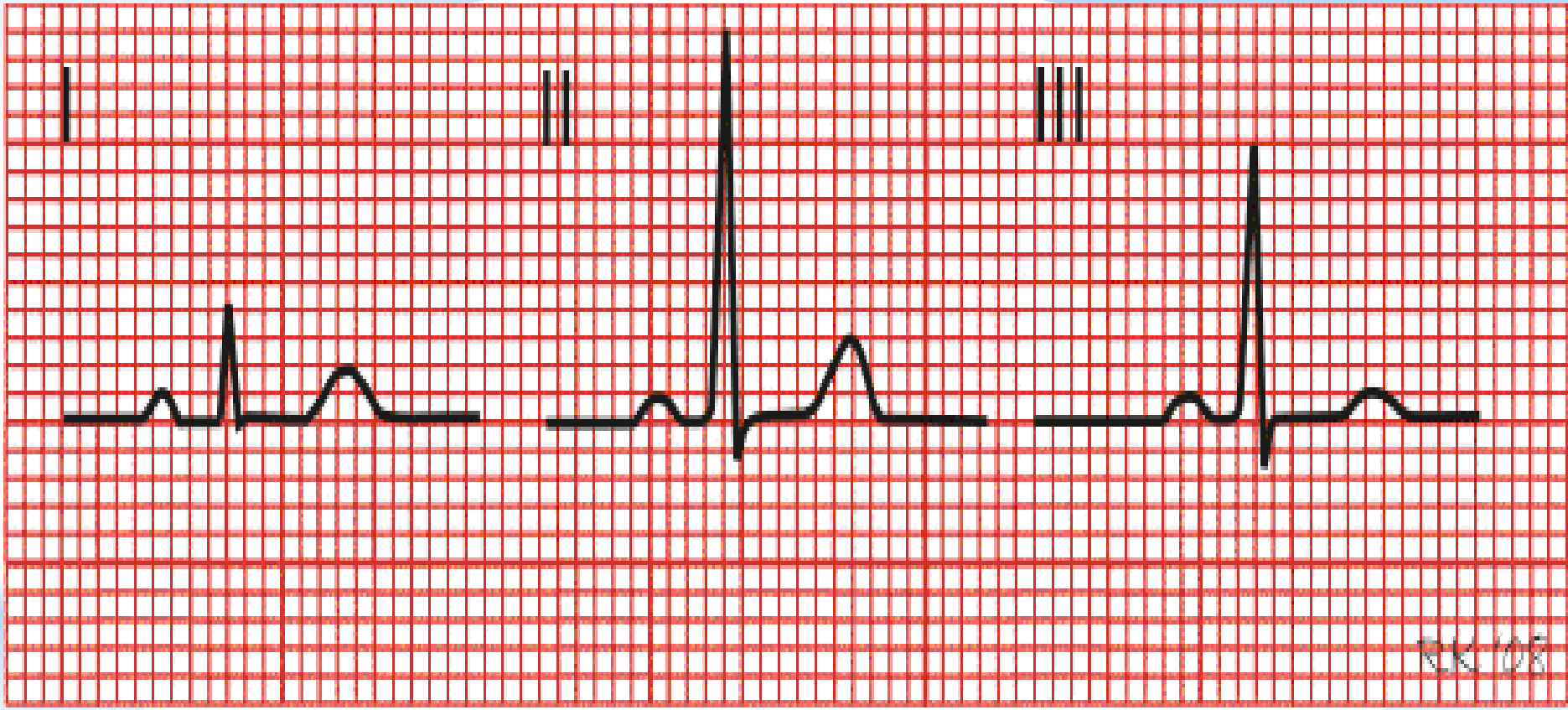
2.Three Augmented limb leads(unipolar)

3.Six chest/precordial leads(unipolar)


RA
LA
I
II
III
RL
LL


RA
I
LA
II
III
LL
I
III
II
0° Lead I
+120°
Lead III
+60°
Lead II
Einthoven's Triangle
Axial Reference System

For heart with normal ECG and mean cardiac axis of +60, the standard limb leads will appear as:-



Einthoven's Law

Einthoven's law states that if the electrical potentials of any two of the three bipolar limb electrocardiographic leads are known at any given instant, the third one can be determined mathematically by simply summing the first two

Lead I + Lead III= Lead II

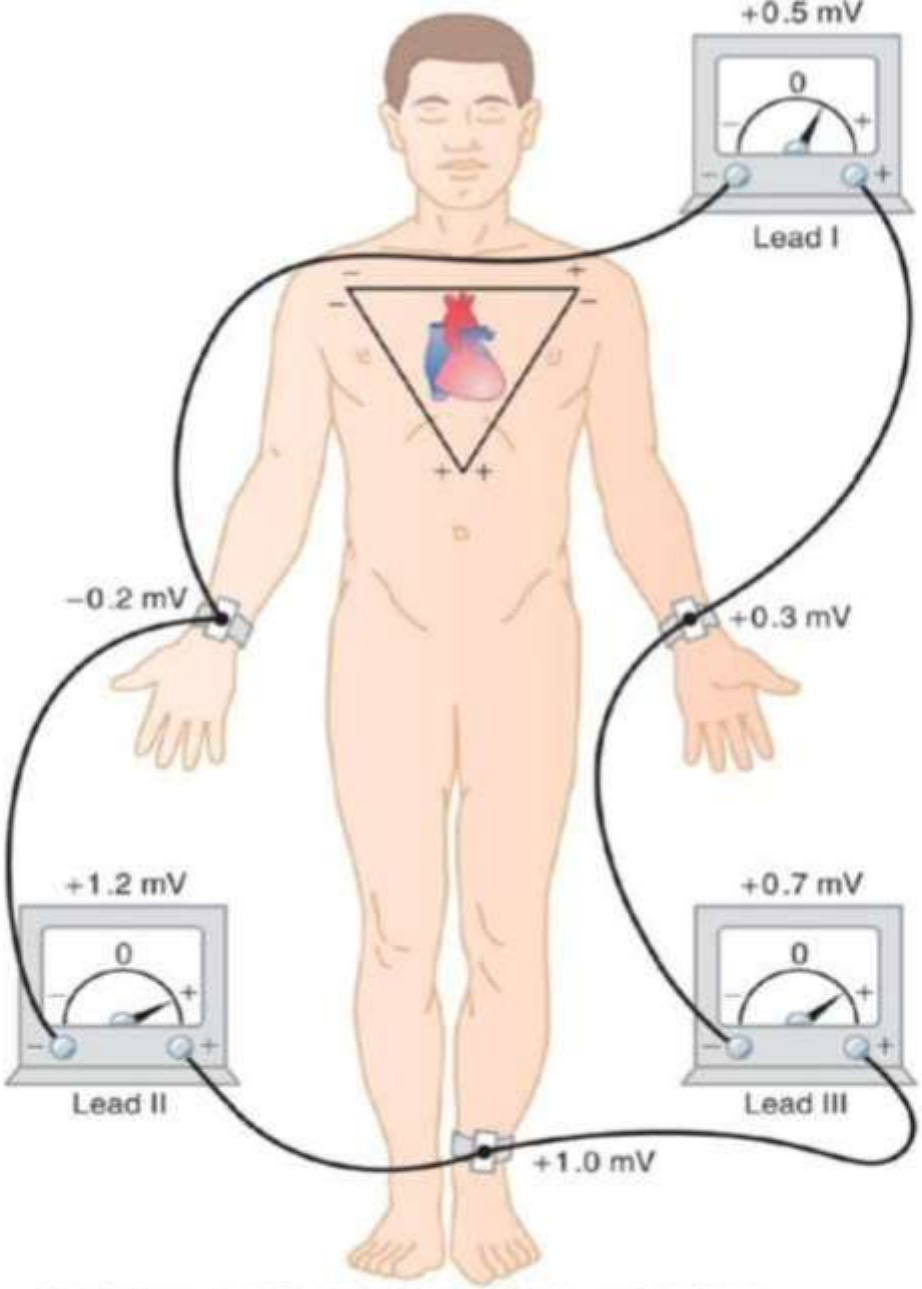
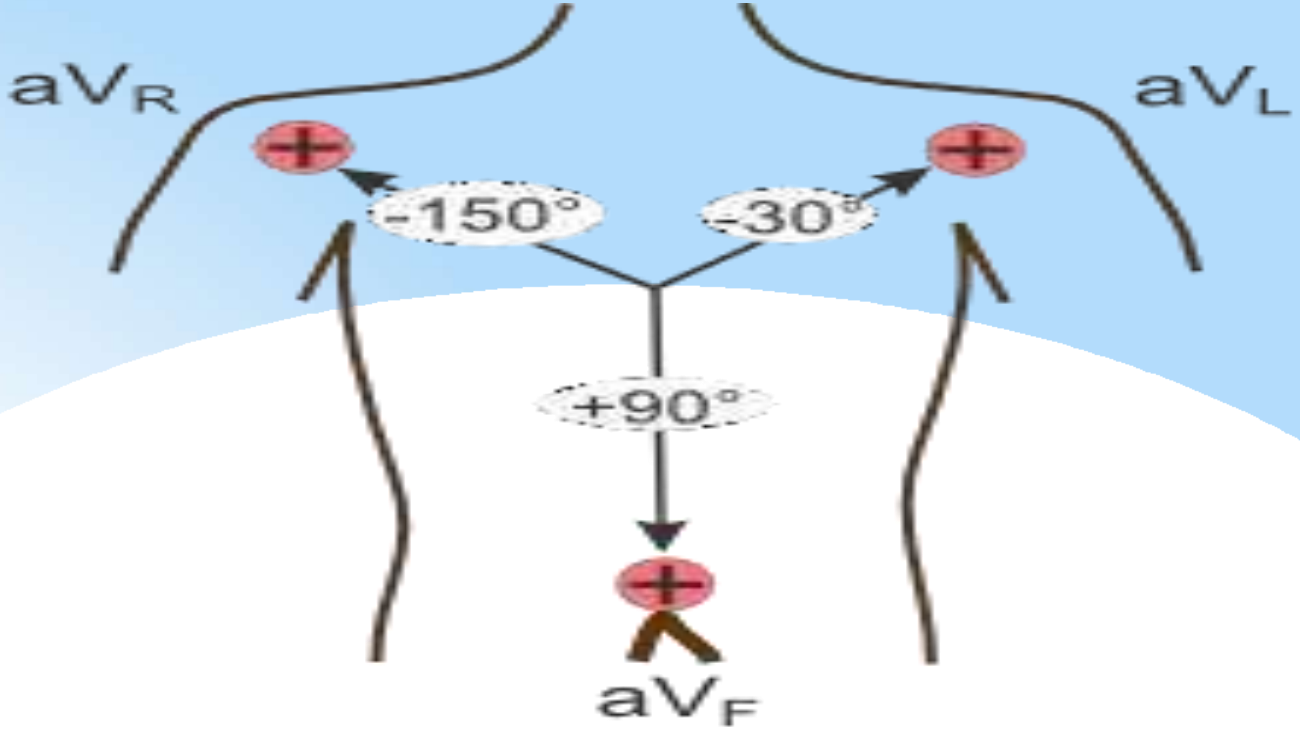
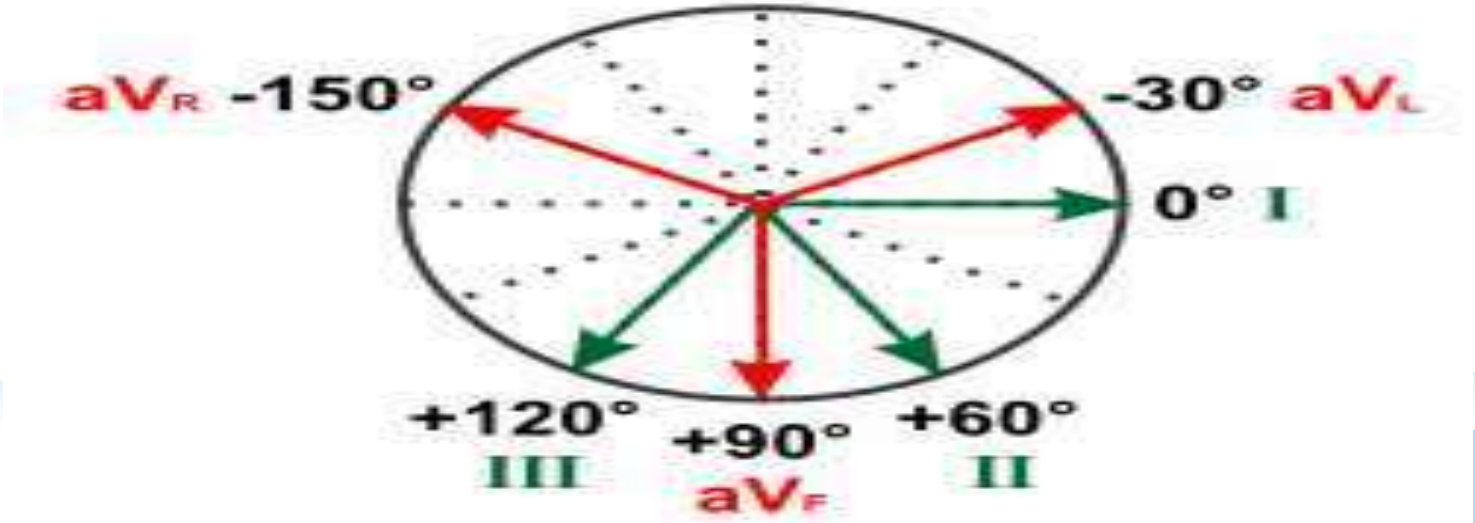
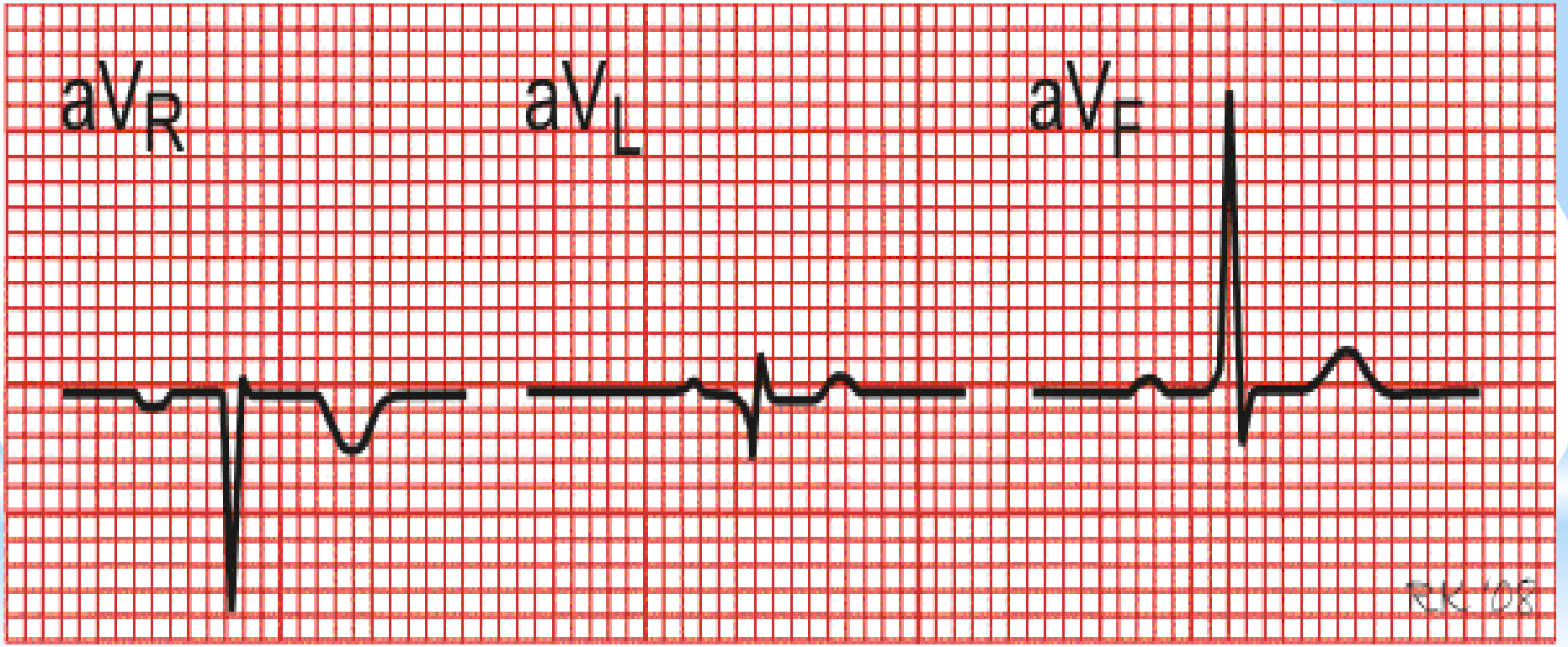


Figure 11-6 Conventional arrangement of electrodes for recording the standard electrocardiographic leads. Einthoven's triangle is superimposed on the chest.

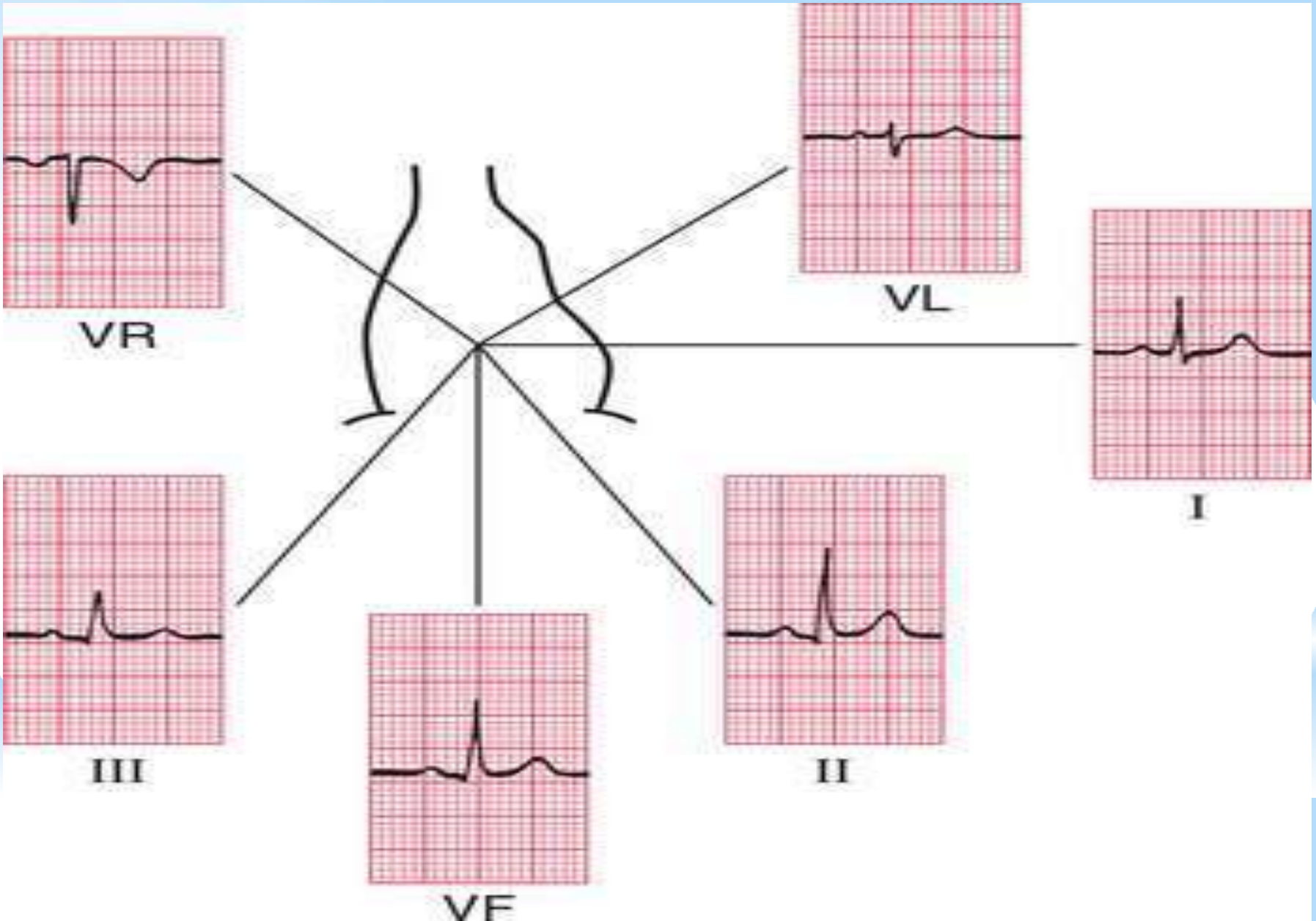

aVR
aVL
-150°
-30°
+90°
aVF


aVR -150°
-30° aVL
0° I
+120°
III
+90°
aVF
+60°
II

For heart with normal ECG and mean cardiac axis of +60, the augmented limb leads will appear as:-



ECG pattern recorded by six standard leads



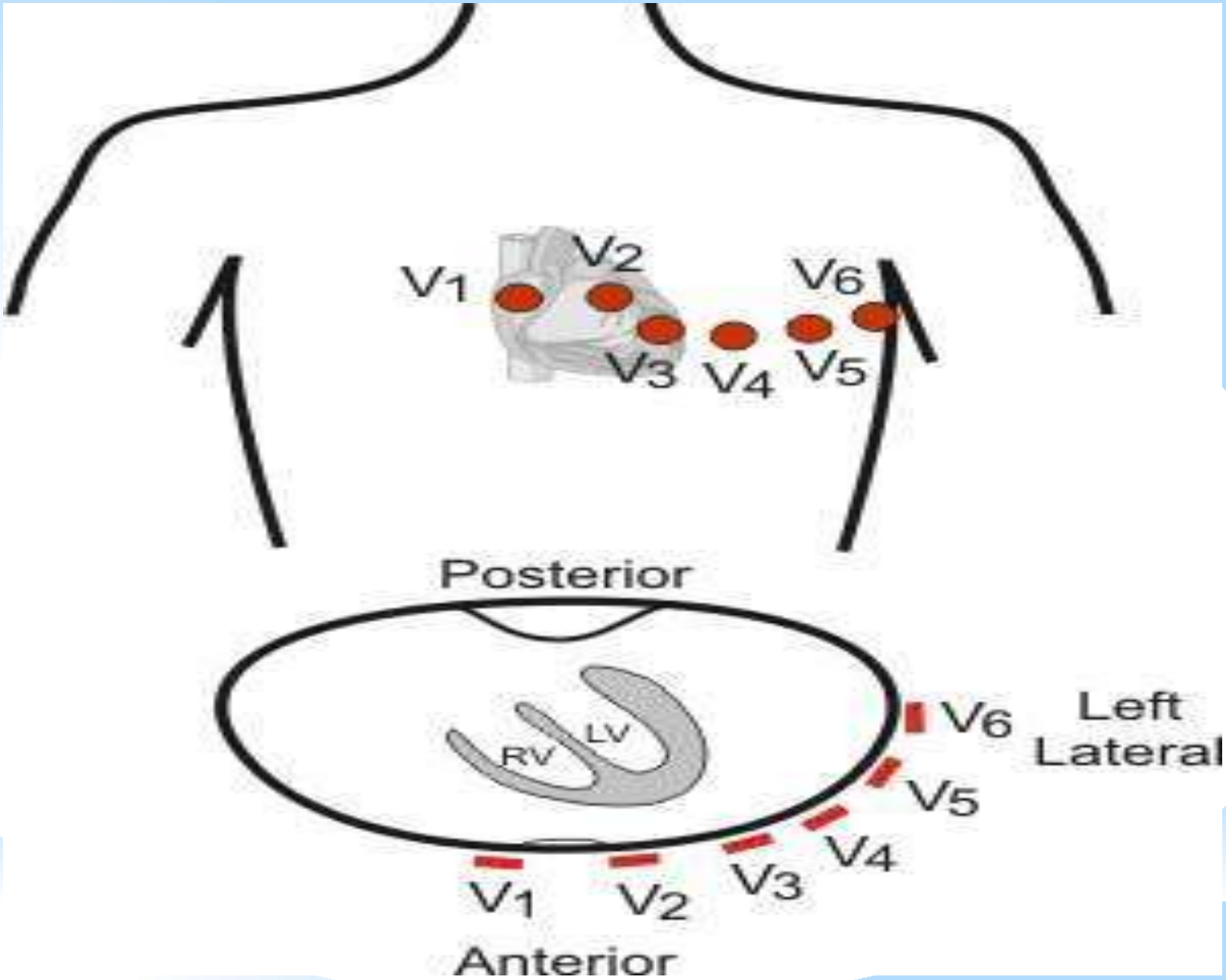
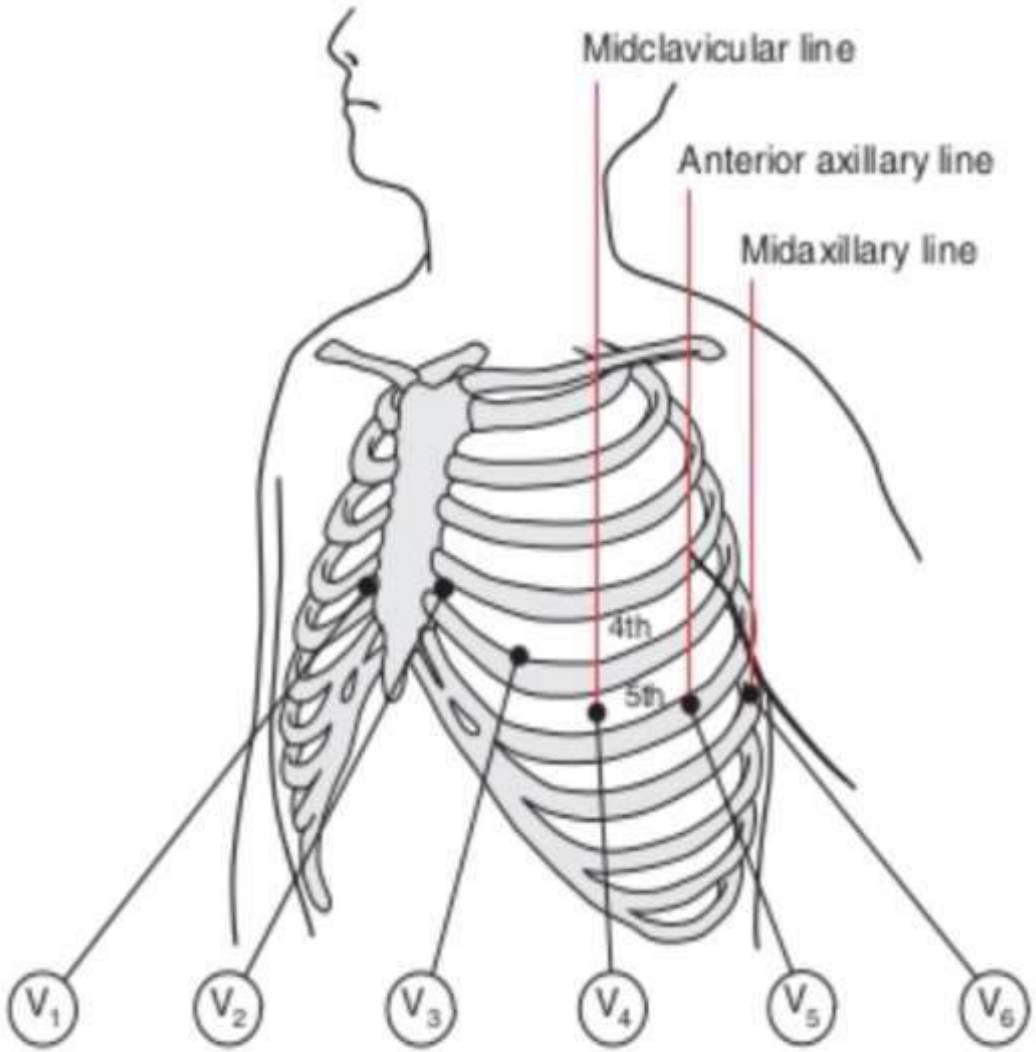

V1
V2
V3
V4
V5
V6
Posterior
RV
LV
V6
V5
V4
V3
V2
V1
Left
Lateral
Anterior

Fig. 1.24

The positions of the chest leads: note the fourth and fifth rib spaces



V1 : Over 4th intercostal space near right sternal margin

V2 : Over 4th intercostal space near left sternal margin

V3 : In between V2 and V4

V4 : Over left 5th intercostal space on the mid clavicular line

V5 : Over left 5th intercostal space on the anterior axillary line

V6 : Over left 5th intercostal space on the mid axillary line.

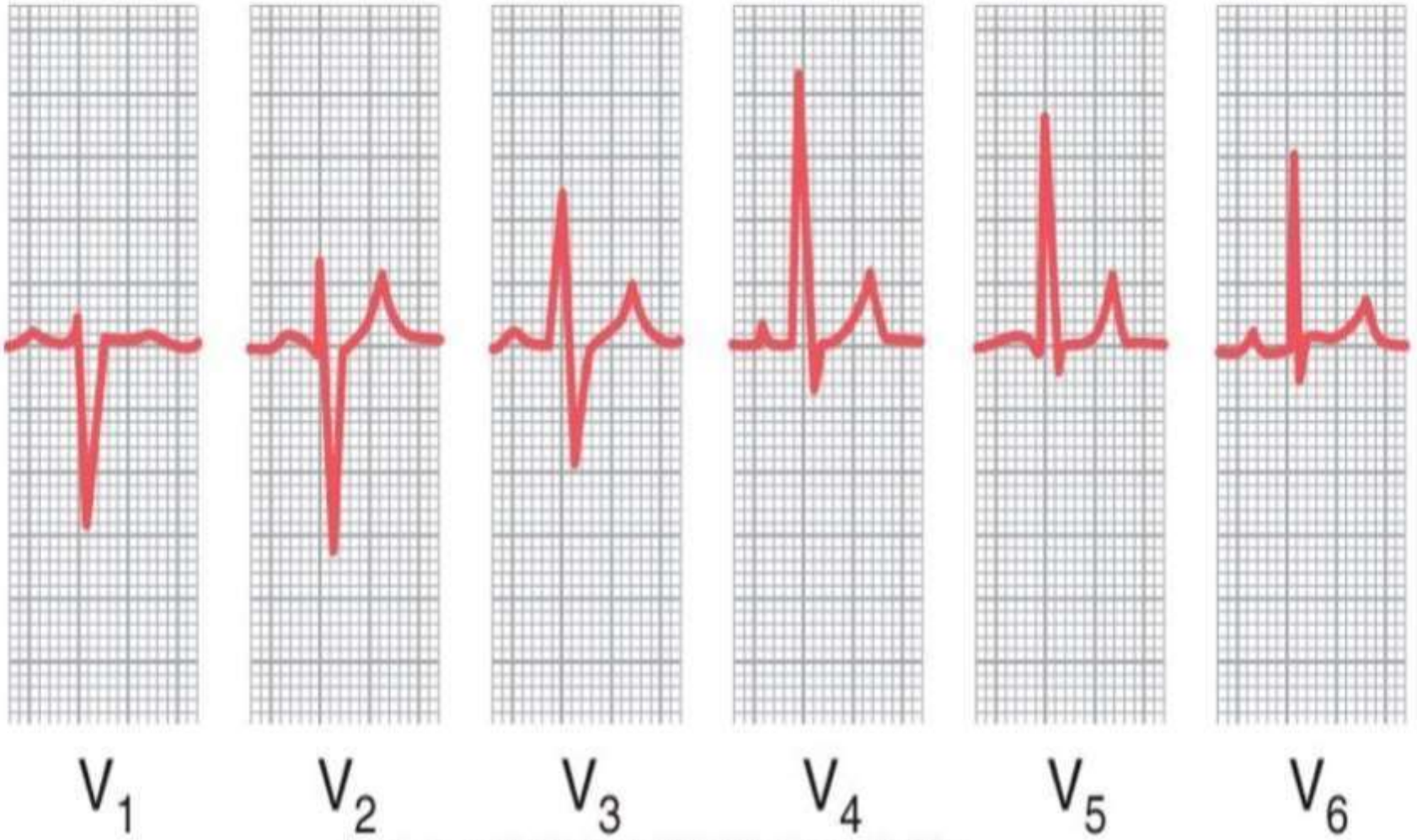


Figure 11-9 Normal electrocardiograms recorded from the six standard chest leads.

TABLE 13-1 Location of Electrodes and Lead Connections for the Standard 12-Lead Electrocardiogram and Additional Leads

LEAD TYPE	POSITIVE INPUT	NEGATIVE INPUT
Standard Limb Leads		
Lead I	Left arm	Right arm
Lead II	Left leg	Right arm
Lead III	Left leg	Left arm
Augmented Limb Leads		
aVR	Right arm	Left arm plus left leg
aVL	Left arm	Right arm plus left leg
aVF	Left leg	Left arm plus right arm
Precordial Leads*		
V_1	Right sternal margin, fourth intercostal space	Wilson central terminal
V_2	Left sternal margin, fourth intercostal space	Wilson central terminal
V_3	Midway between V_2 and V_4	Wilson central terminal
V_4	Left midclavicular line, 5th intercostal space	Wilson central terminal
V_5	Left anterior axillary line†	Wilson central terminal
V_6	Left midaxillary line†	Wilson central terminal

ANATOMIC GROUPS

- Lead I, aVL,V5,V6:- lateral wall
- Lead II,III,aVF:-Inferior wall
- Lead V1,V2:- Septal
- Lead V3,V4:- Anterior wall

HOW TO REPORT AN ECG?

The description should always be in the sequence:-

1 Rhythm, Rate

2 Conduction intervals

3 Cardiac axis

4 A description of the QRS complexes

5 A description of the ST segments and T waves

1. Rhythm, Rate

- The word ‘rhythm’ is used to refer to the part of the heart which is controlling the activation sequence. The normal heart rhythm, with electrical activation beginning in the SA node, is called ‘sinus rhythm’

- If the intervals between QRS complexes (R-R intervals) are consistent, ventricular rhythm is regular. If intervals between P waves (P-P intervals) are consistent, the atrial rhythm is regular

- If the rhythm is regular,

$$\text{Rate} = \frac{300}{\text{Number of large squares between R-R interval}}$$

OR

$$\text{Rate} = \frac{1500}{\text{Number of small squares between R-R interval}}$$

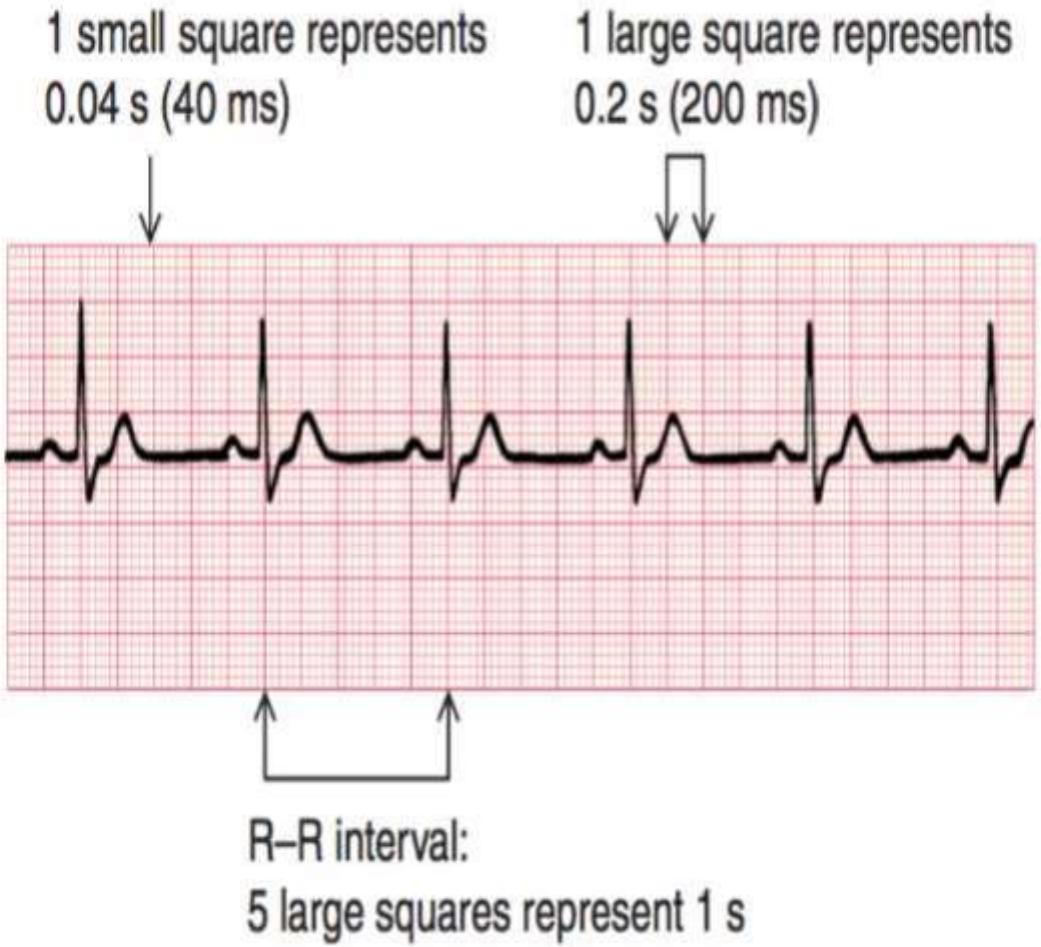
- If the rhythm is irregular, count the number of QRS complexes in a 6-second segment and multiply by 10. Rates below 60 indicate bradycardia; those above 100 indicate tachycardia.

Table 1.1 Relationship between the number of large squares between successive R waves and the heart rate

R–R interval (large squares)	Heart rate (beats/min)
1	300
2	150
3	100
4	75
5	60
6	50

Fig. 1.4

Relationship between the squares on ECG paper and time. Here, there is one QRS complex per second, so the heart rate is 60 beats/min



2. CONDUCTION INTERVALS

TABLE 29–2 ECG intervals.

Intervals	Normal Durations		Events in the Heart during Interval
	Average	Range	
PR interval[a]	0.18[b]	0.12–0.20	Atrioventricular conduction
QRS duration	0.08	to 0.10	Ventricular depolarization
QT interval	0.40[c]	to 0.43	Ventricular action potential
ST interval (QT minus QRS)	0.32	…	Plateau portion of the ventricular action potential

[a]Measured from the beginning of the P wave to the beginning of the QRS complex.

[b]Shortens as heart rate increases from average of 0.18 s at a rate of 70 beats/min to 0.14 s at a rate of 130 beats/min.

[c]Can be lower (0.35) depending on the heart rate.

3. CARDIAC AXIS

- Leads VR and II look at the heart from opposite directions. When seen from the front, the depolarization wave normally spreads through the ventricles from 11 o'clock to 5 o'clock, so the deflections in lead VR are normally mainly downward (negative) and in lead II mainly upward (positive)

- The average direction of spread of the depolarization wave through the ventricles as seen from the front is called the 'cardiac axis'

Fig. 1.13

The cardiac axis

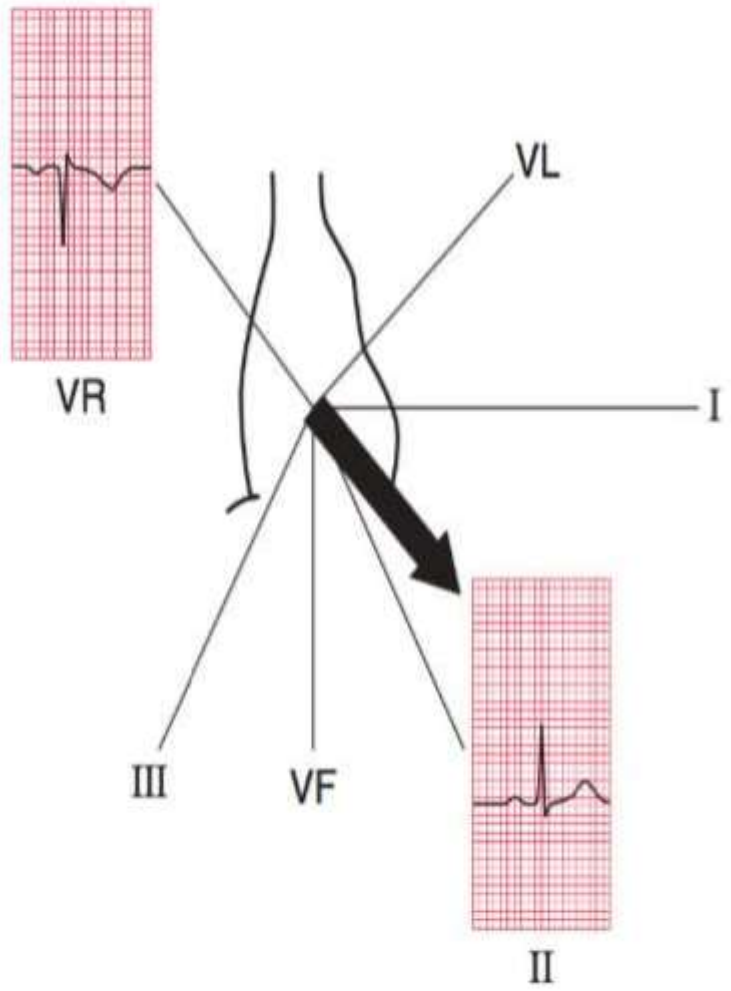


Fig. 1.14

The normal axis

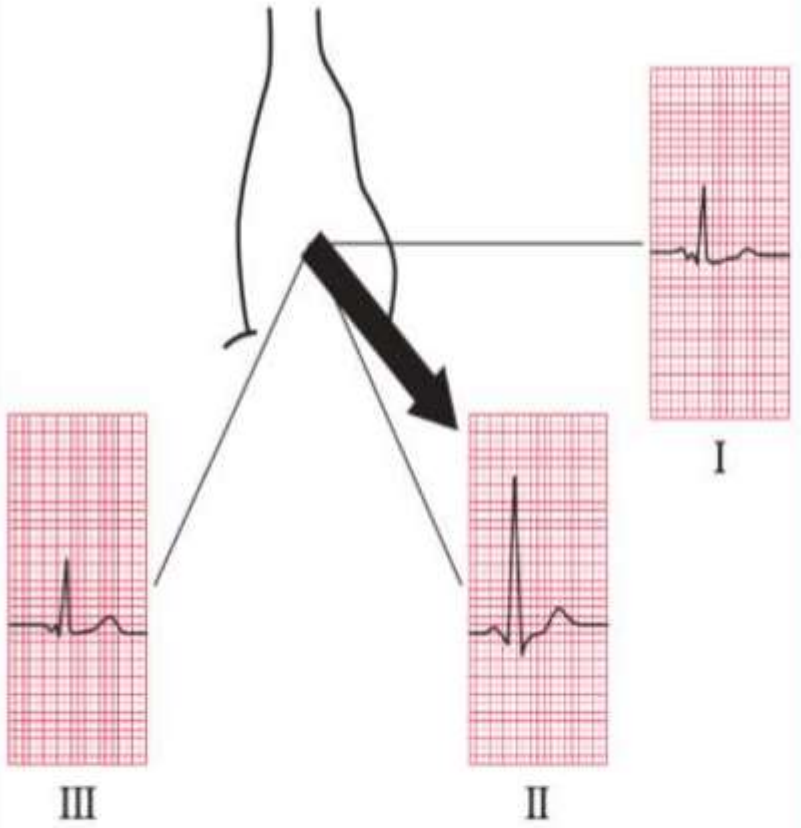
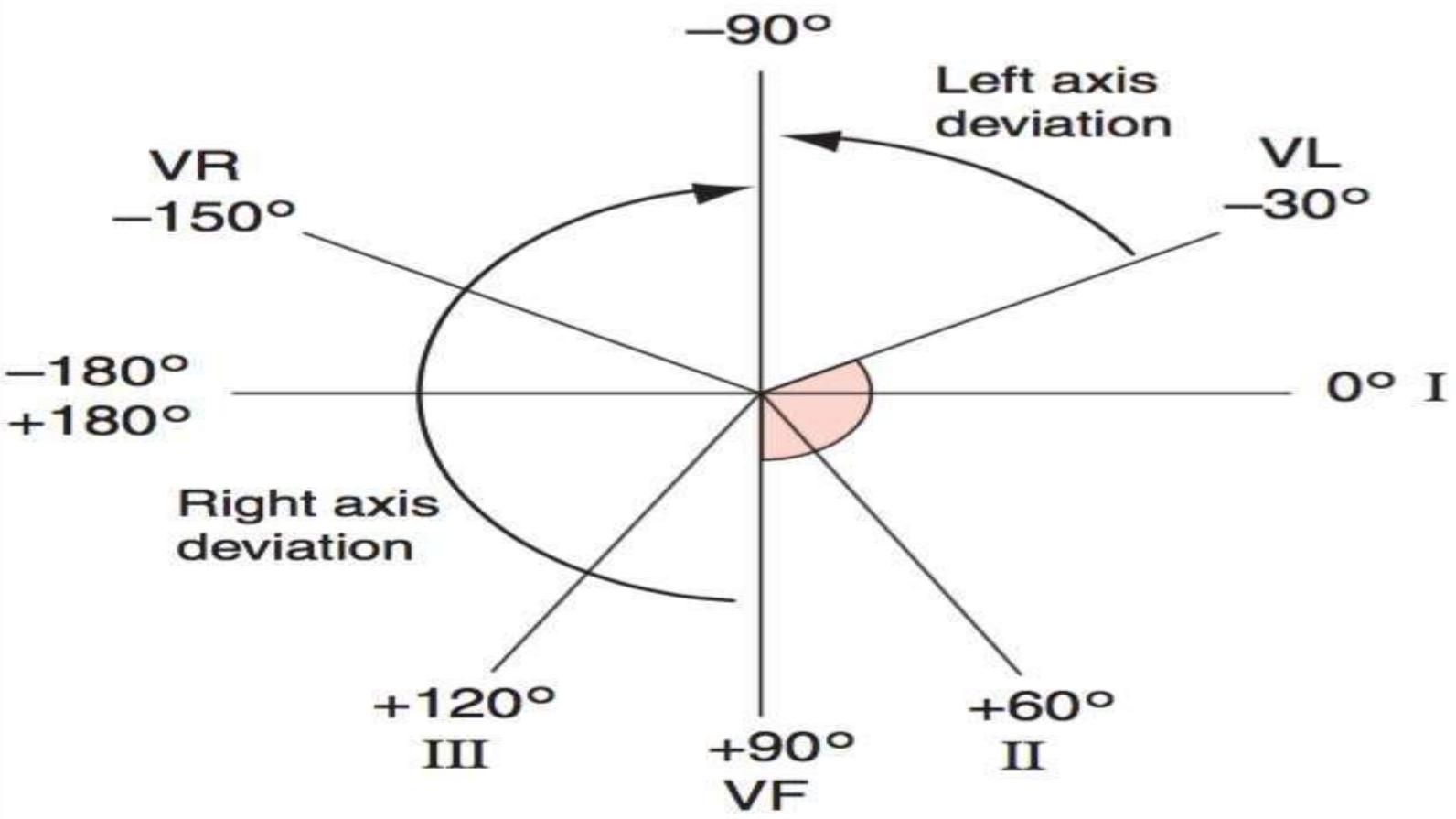


Fig. 1.17

The cardiac axis and lead angles



Limit of the normal cardiac axis

Fig. 1.15

Right axis deviation

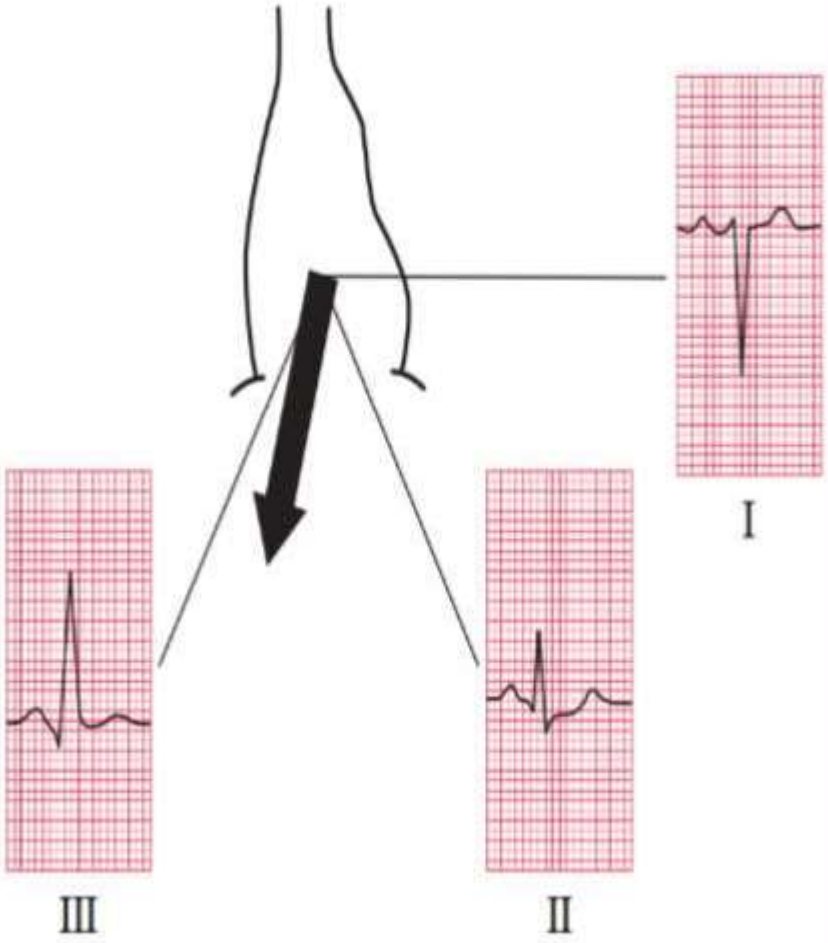
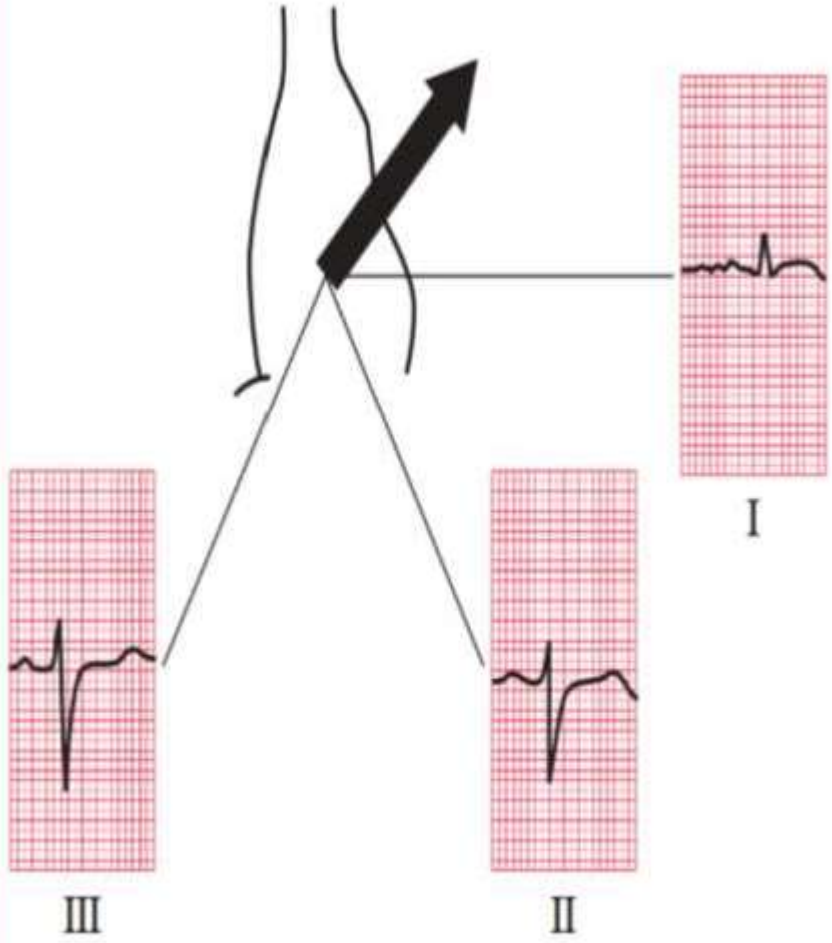


Fig. 1.16

Left axis deviation



4. Shape of QRS complexes

Fig. 1.18

Shape of the QRS complex: first stage

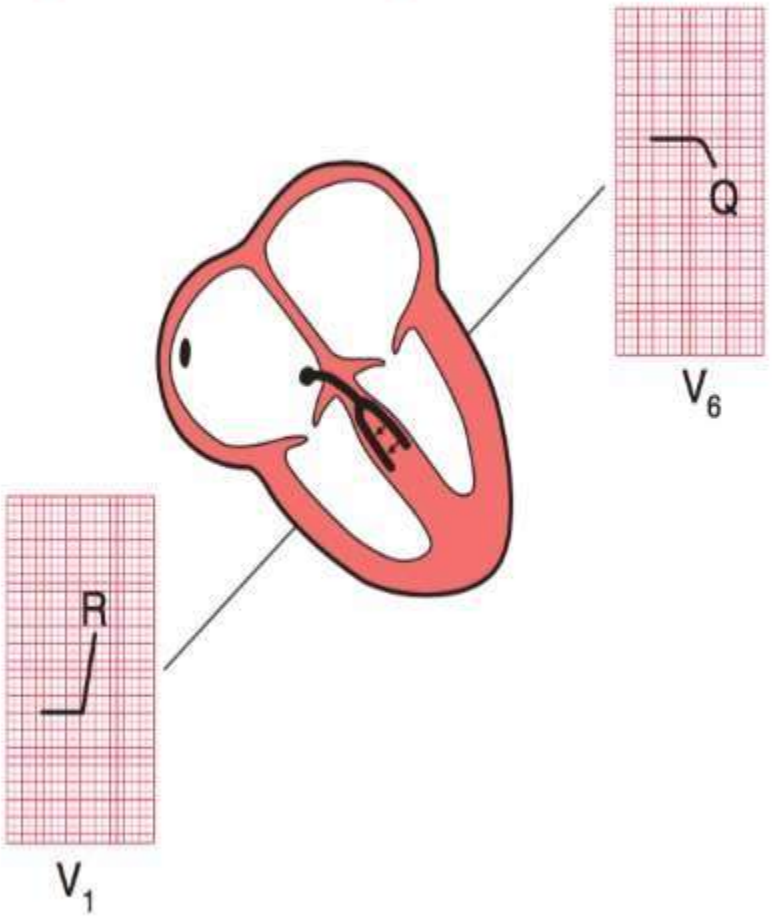


Fig. 1.19

Shape of the QRS complex: second stage

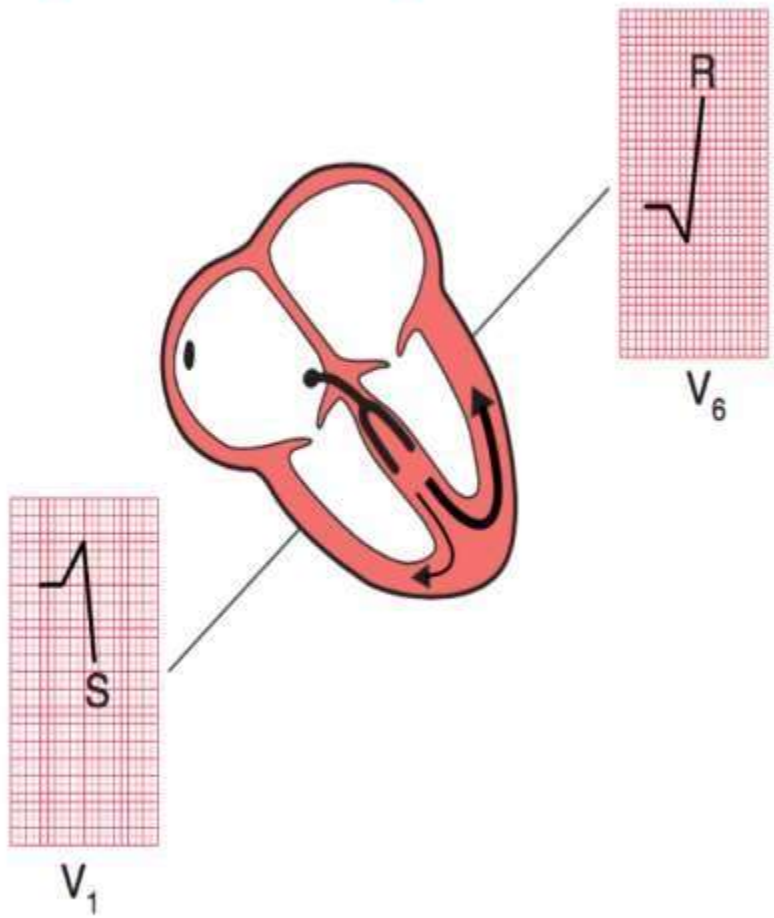


Fig. 1.20

Shape of the QRS complex: third stage

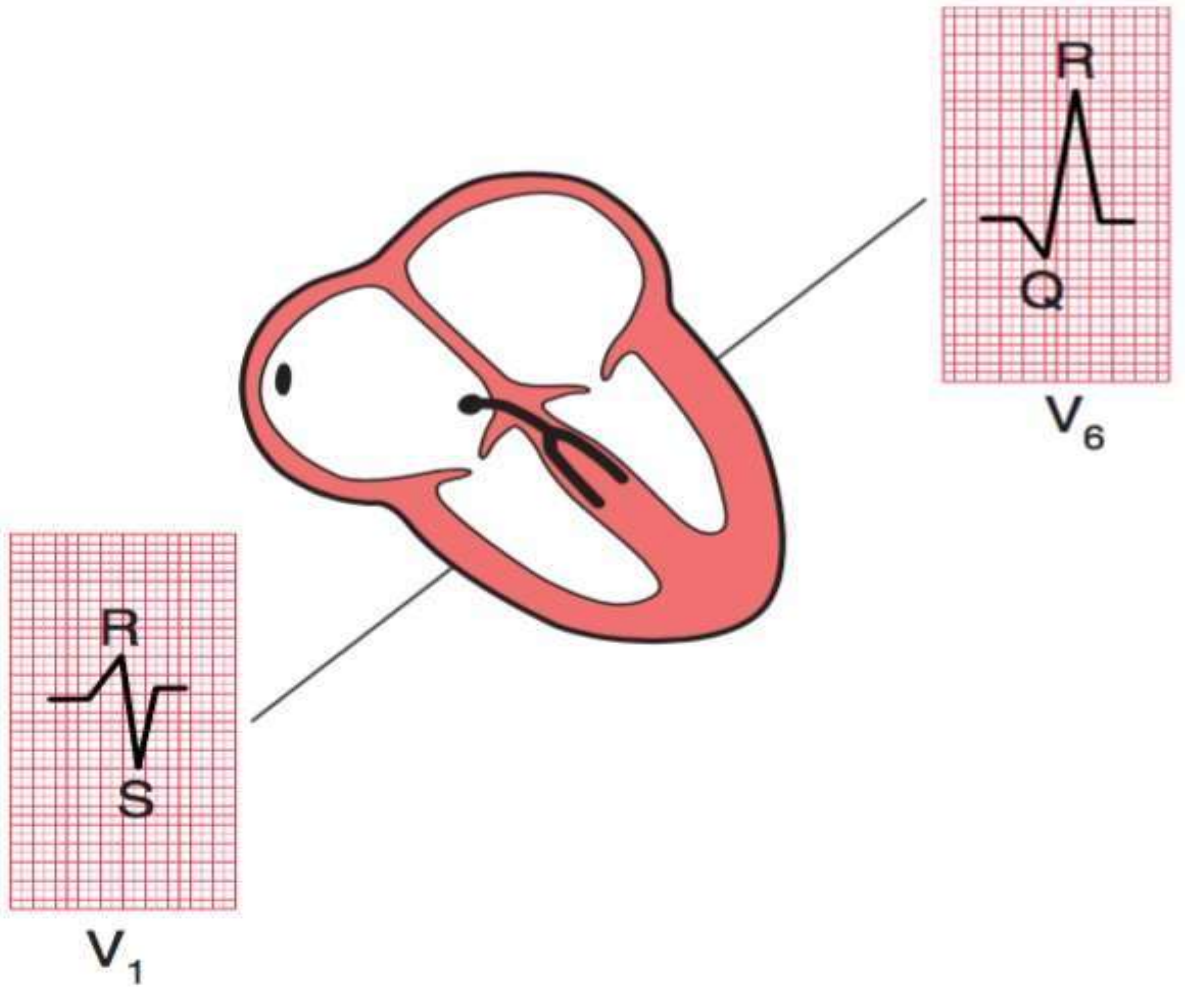
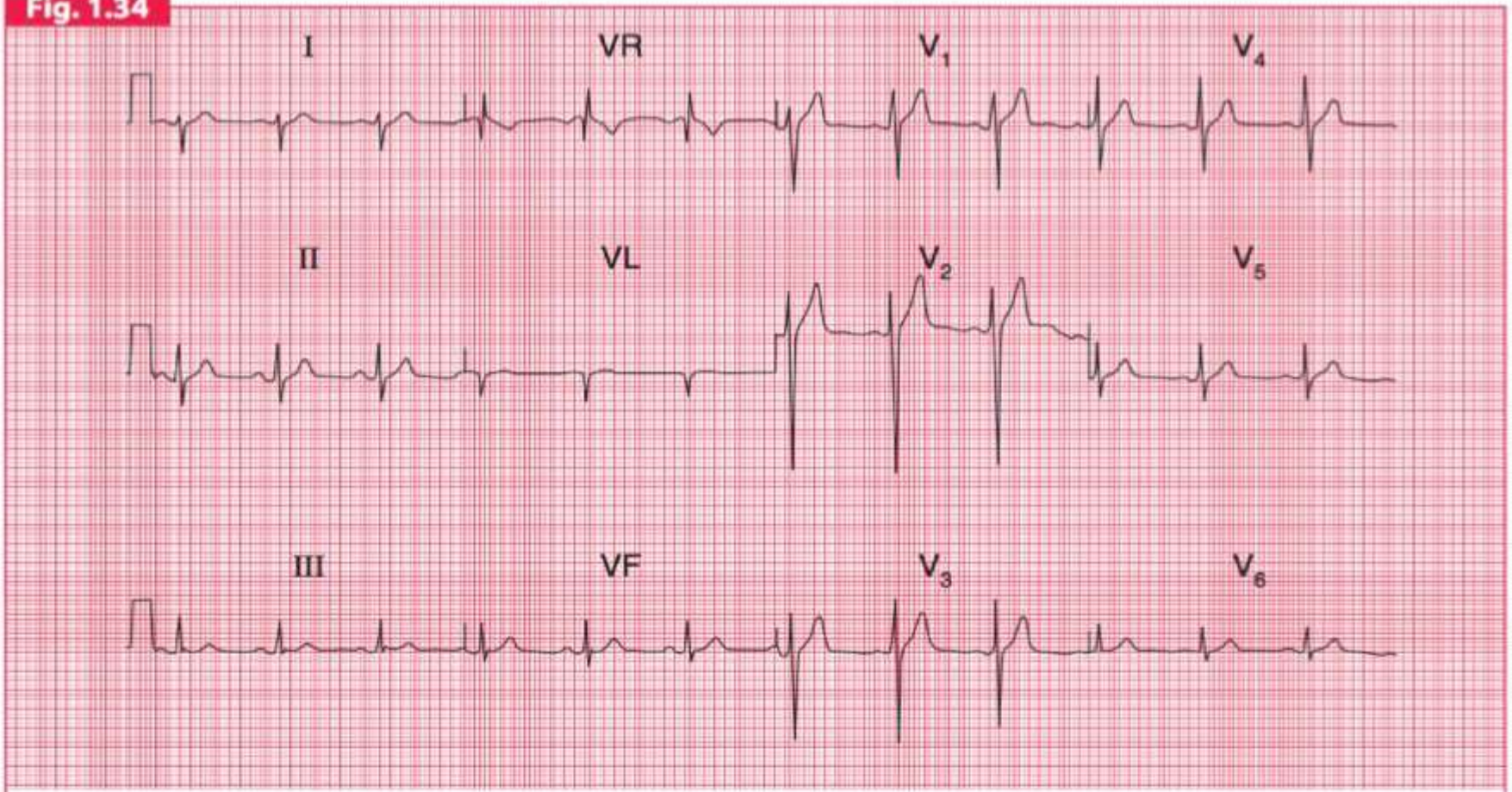


Fig. 1.34



Variant of a normal ECG

Note

- Sinus rhythm, rate 75/min
- Normal PR interval (200 ms)
- Normal QRS complex duration (120 ms)
- Right axis deviation (prominent S wave in lead I)
- Normal QRS complexes
- Normal ST segments and T waves

Interpretation

- Normal ECG – apart from right axis deviation, which could be normal in a tall, thin person

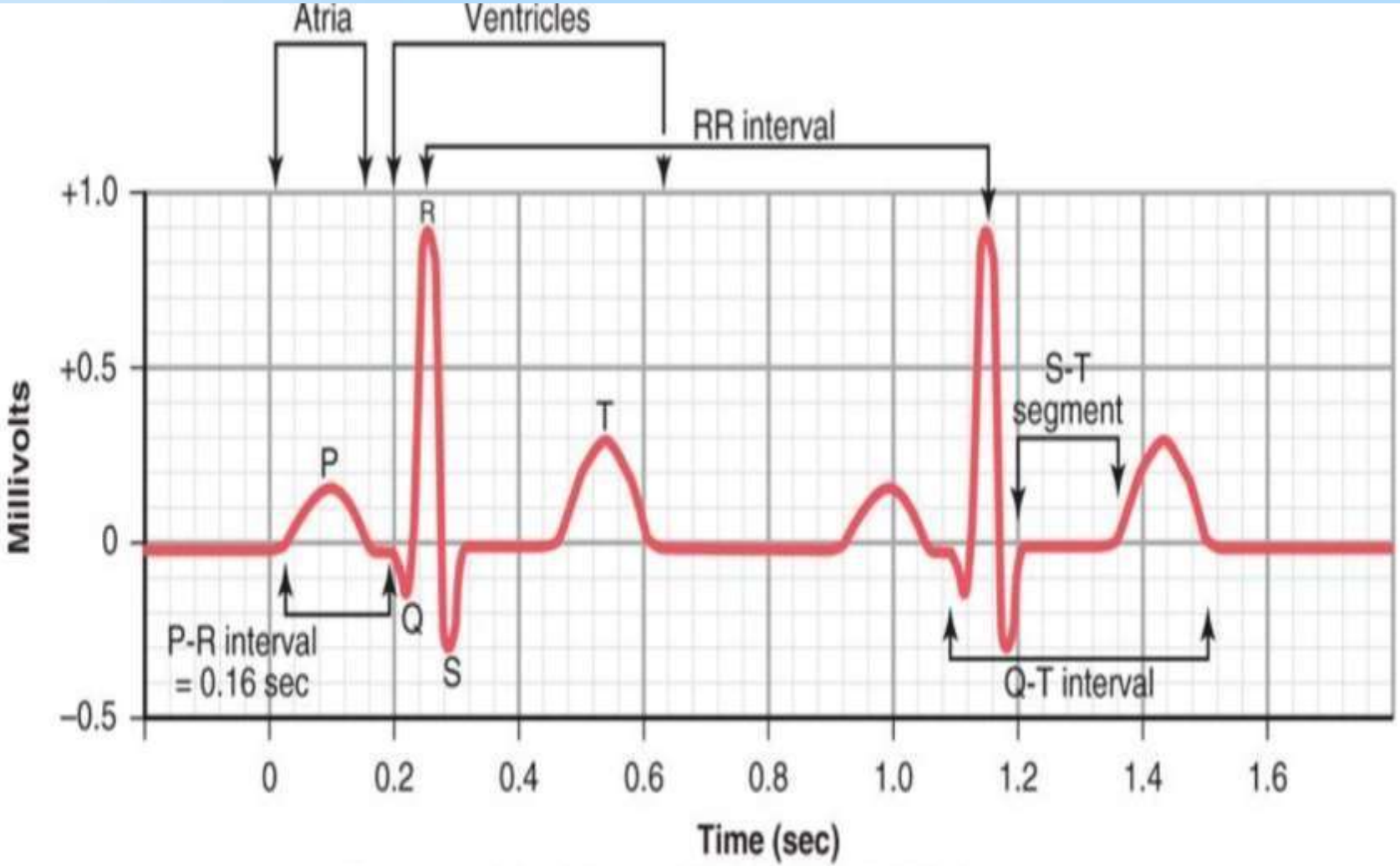


Figure 11-1 Normal electrocardiogram.

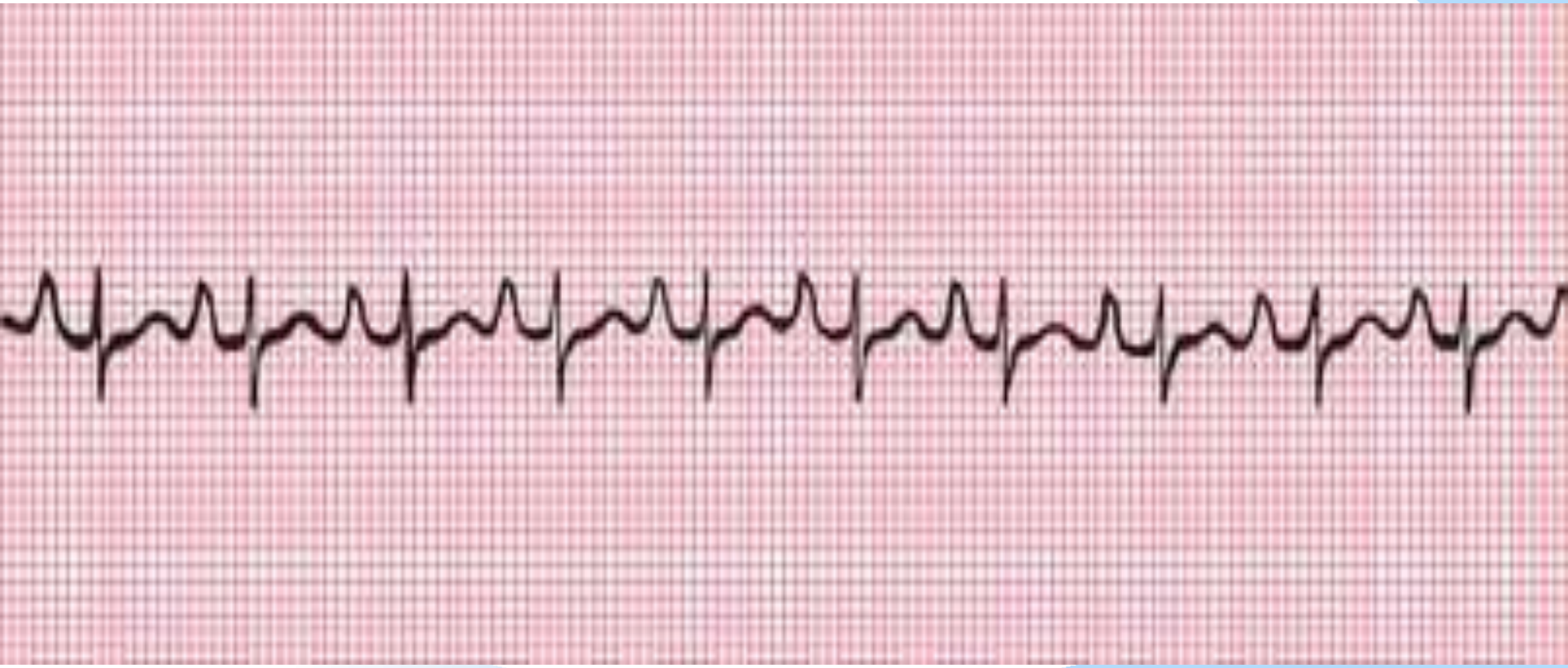
P WAVE

- ✓ Always positive in lead I and II
- ✓ Always negative in lead aVR
- ✓ <3 small square in duration
- ✓ <2.5 small square in amplitute
- ✓ May be biphasic in lead V1
- ✓ Best seen in lead II

Abnormalities of P Wave

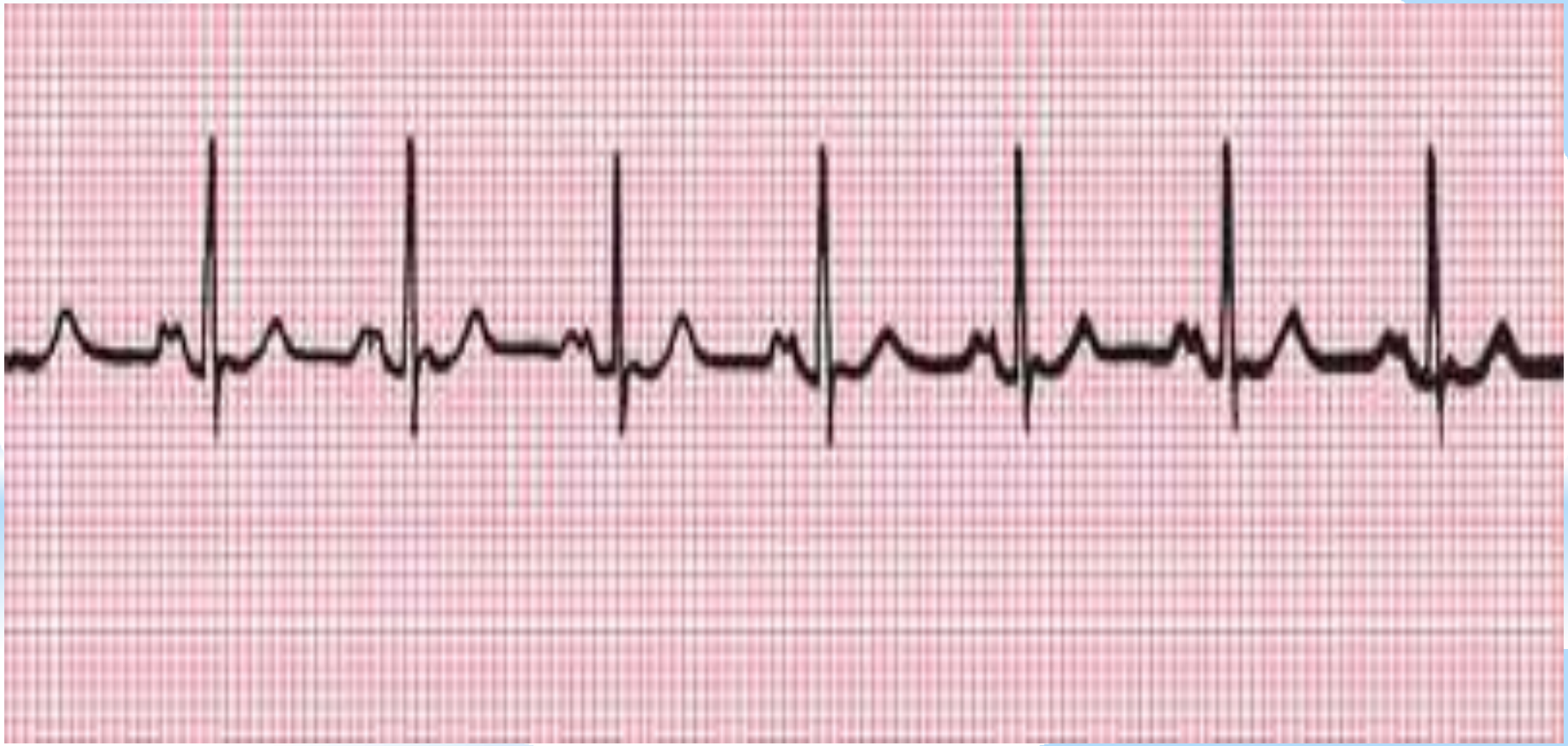
1. Right atrial enlargement:-

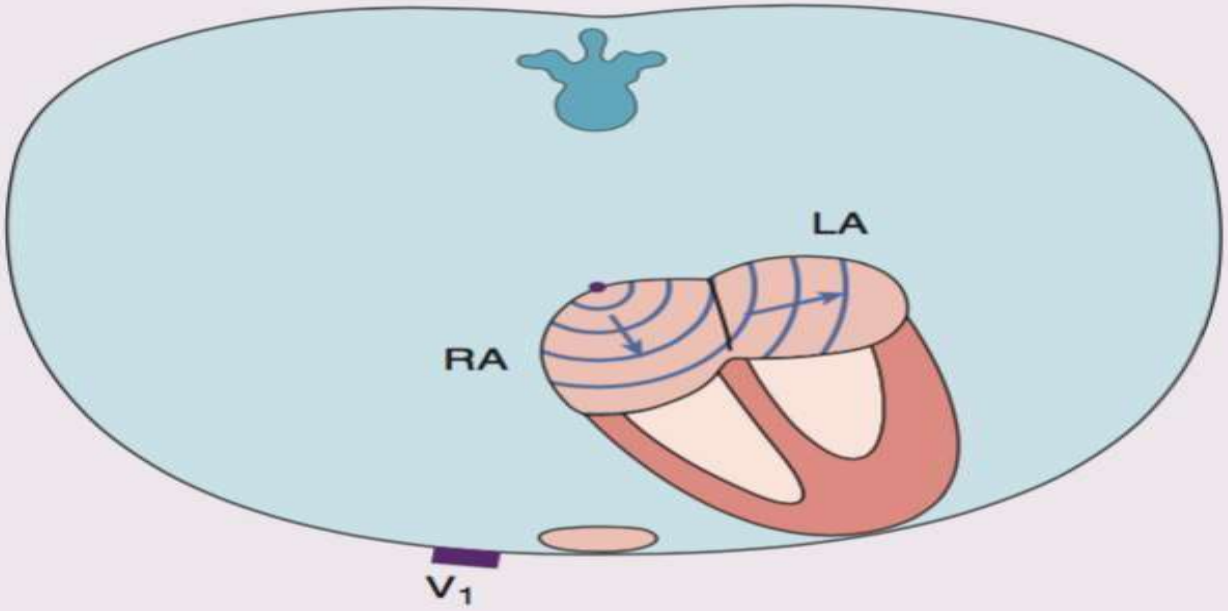
- **Tall(>2.5mm), Pointed P wave('P Pulmonale')**



2. Left atrial enlargement:-

- **Notched/bifid (‘M’ shaped) P wave (‘P Mitrale’) in limb leads**





	Normal	Right	Left
II	RA LA	RA LA	RA LA
V_1	RA LA	RA LA	RA LA

FIGURE 13-14 Schematic representation of atrial depolarization (diagram) and P wave patterns associated with normal atrial activation **(left panel)** and with right **(middle panel)** and left **(right panel)** atrial abnormalities. *(Modified from Park MK, Guntheroth WG: How to Read Pediatric ECGs. 3rd ed. St. Louis, Mosby-Year Book, 1993.)*

TABLE 13-3 Common Diagnostic Criteria for Left and Right Atrial Abnormalities

LEFT ATRIAL ABNORMALITY	RIGHT ATRIAL ABNORMALITY*
Prolonged P wave duration > 120 msec in lead II	Peaked P waves with amplitudes in lead II > 0.25 mV (P pulmonale)
Prominent notching of P wave, usually most obvious in lead II, with interval between notches of 0.40 msec (P mitrale)	Prominent initial positivity in lead V_1 or V_2 > 0.15 mV
Ratio between the duration of the P wave in lead II and duration of the PR segment > 1.6	Increased area under initial positive portion of the P wave in lead V_1 to > 0.06 mm-sec
Increased duration and depth of terminal- negative portion of P wave in lead V_1 (P terminal force) so that area subtended by it > 0.04 mm-sec	Rightward shift of mean P wave axis to more than +75 degrees
Leftward shift of mean P wave axis to between −30 and −45 degrees	

*In addition to criteria based on P wave morphologies, right atrial abnormalities are suggested by QRS changes, including (1) Q waves (especially qR patterns) in the right precordial leads without evidence of myocardial infarction and (2) low-amplitude (<600 μV) QRS complexes in lead V_1 with a threefold or greater increase in lead V_2.

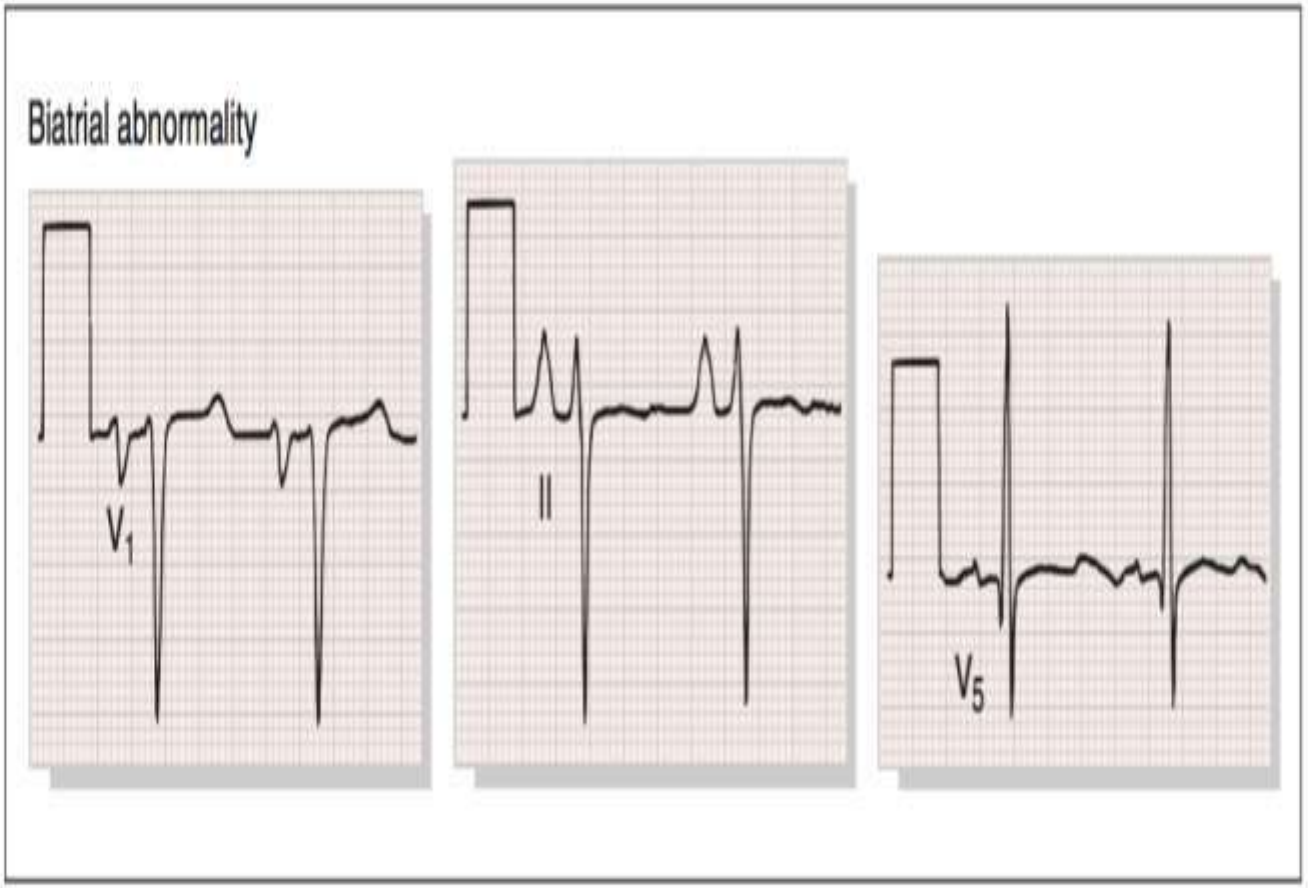


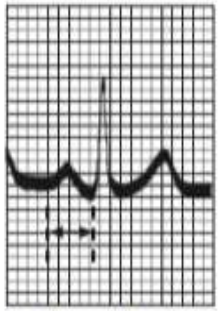
FIGURE 13-15 Biatrial abnormality, with tall P waves in lead II (right atrial abnormality) and an abnormally large terminal negative component of the P wave in lead V_1 (left atrial abnormality). The P wave is also notched in lead V_5.

P-Q or P-R Interval

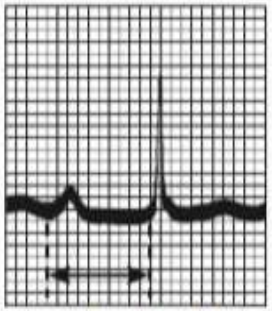
- The time between the beginning of the P wave and the beginning of the QRS complex is the interval between the beginning of electrical excitation of the atria and the beginning of excitation of the ventricles. This period is called the P-Q interval.

- The normal P-R interval is 3-5 small squares/120-220ms (Often this interval is called the P-R interval because the Q wave is likely to be absent.)

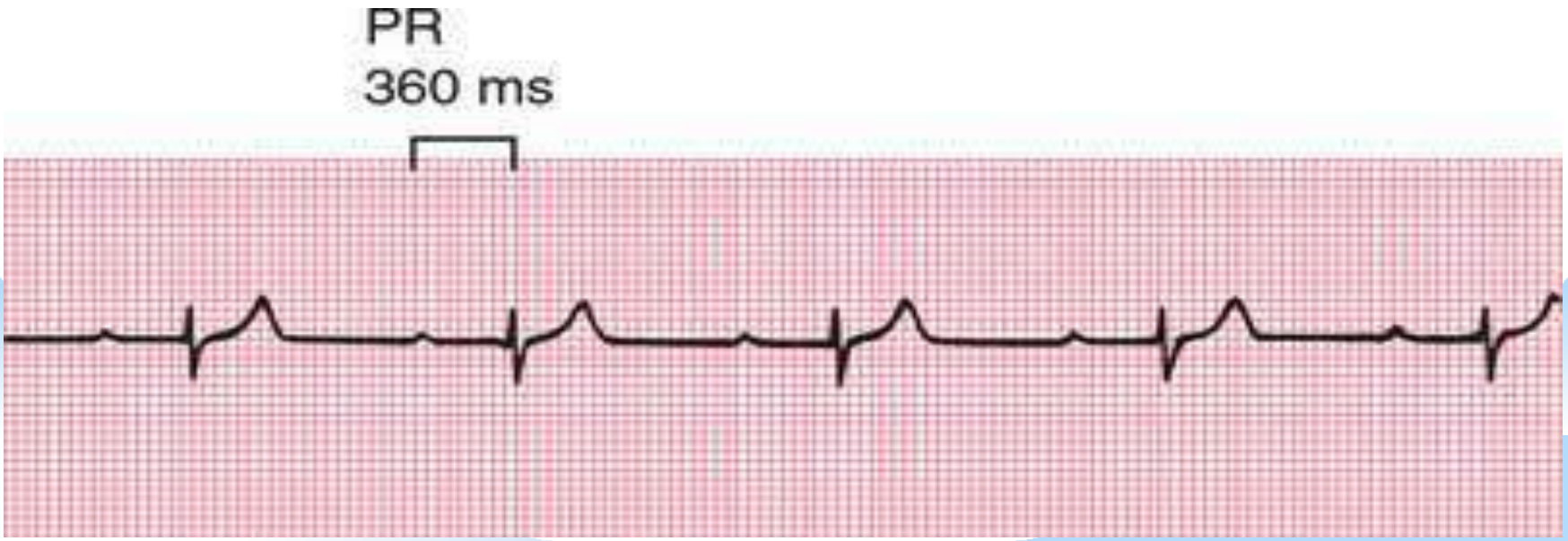
Prolonged PR Interval

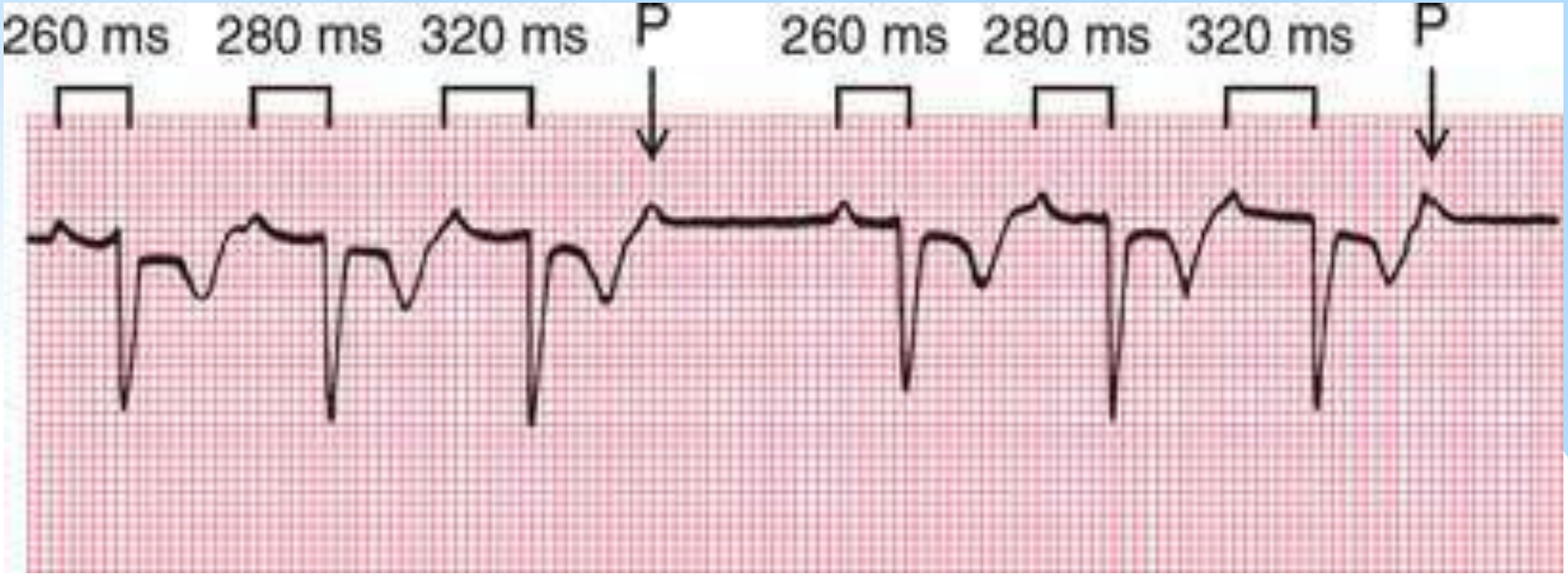


PR = 0.16 s
Normal complex



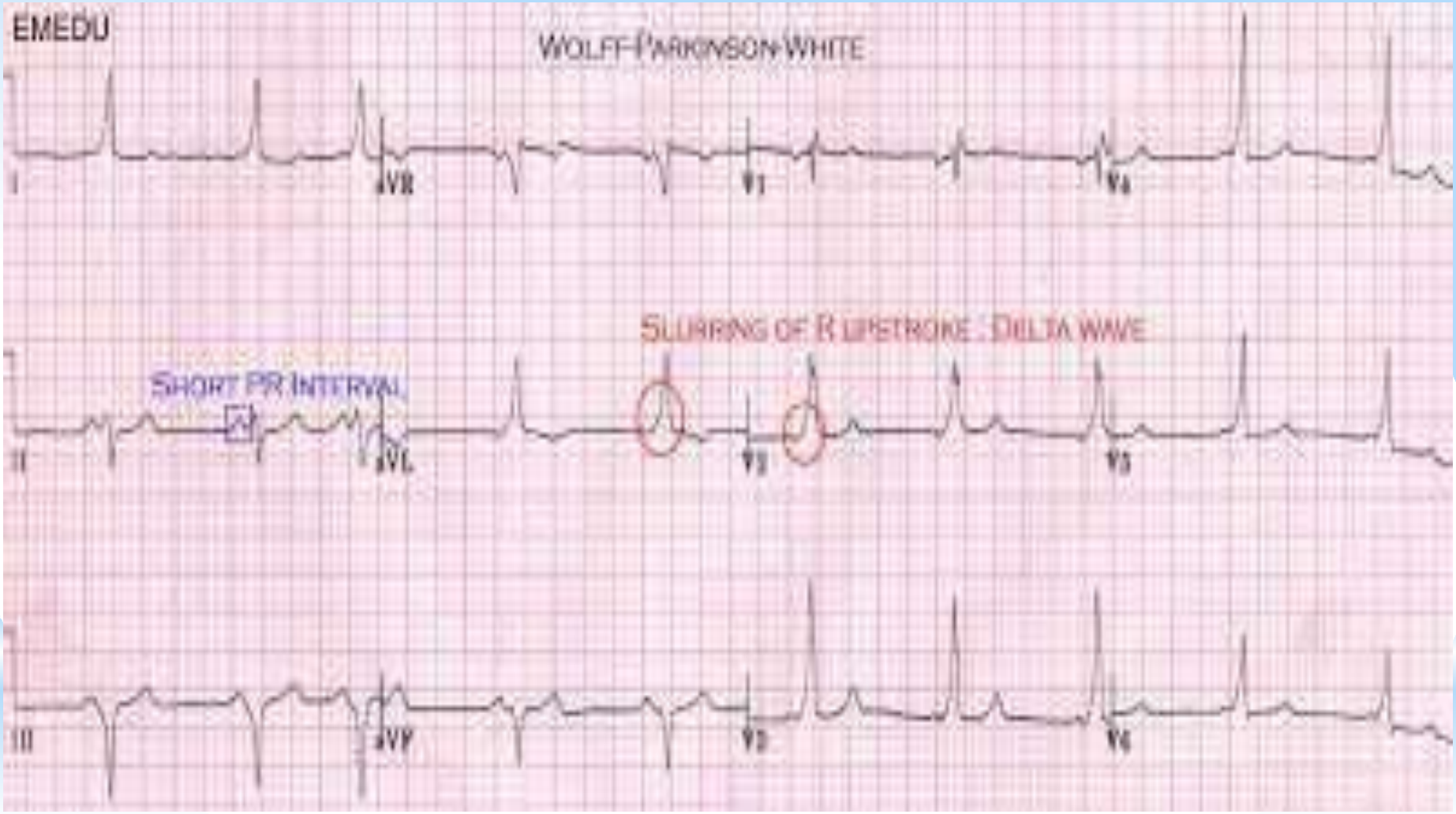
PR = 0.38 s
First-degree heart block





Second degree heart block(Wenckebach/mobitz type 1) showing progresssive lengthening of the PR interval, one non conducted P wave, next conducted beat has a shorter PR interval than the preceding conducted beat

Short PR Interval



QRS Complexes

- The duration of the QRS complex shows how long excitation takes to spread through the ventricles.

- The QRS complex duration is normally 120ms (3 small squares) or less, but any abnormality of conduction takes longer, and causes widened QRS complexes

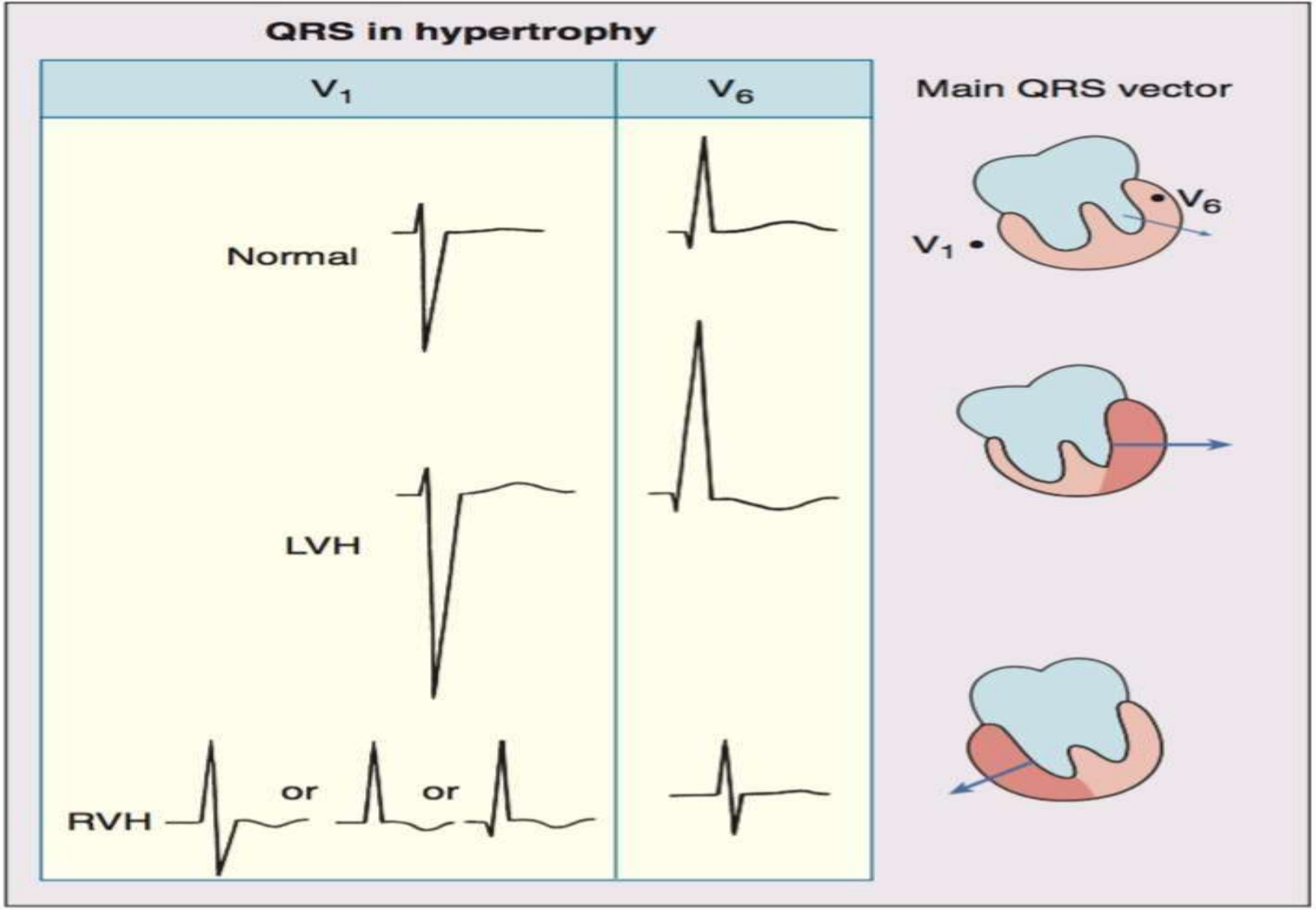
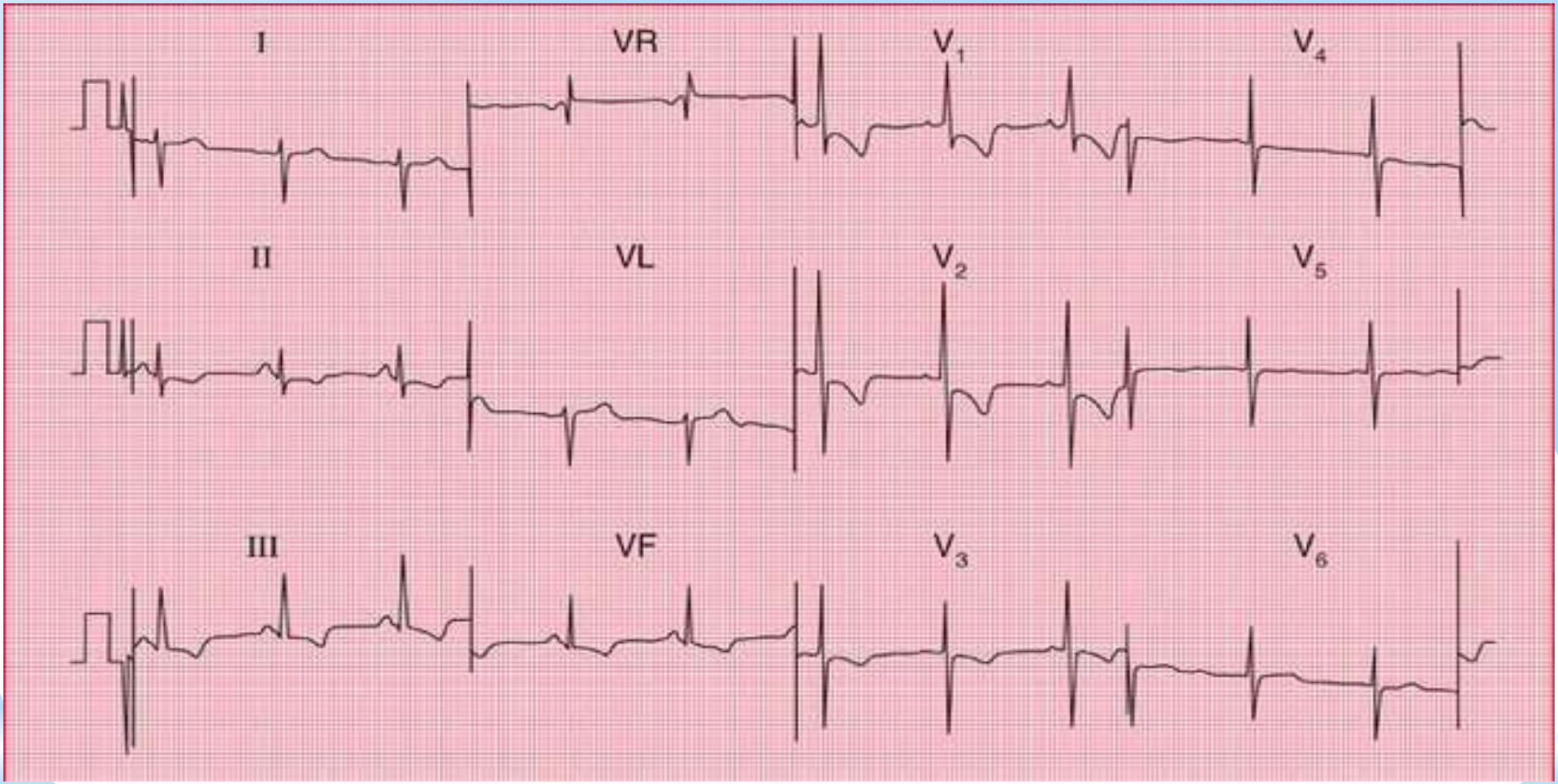


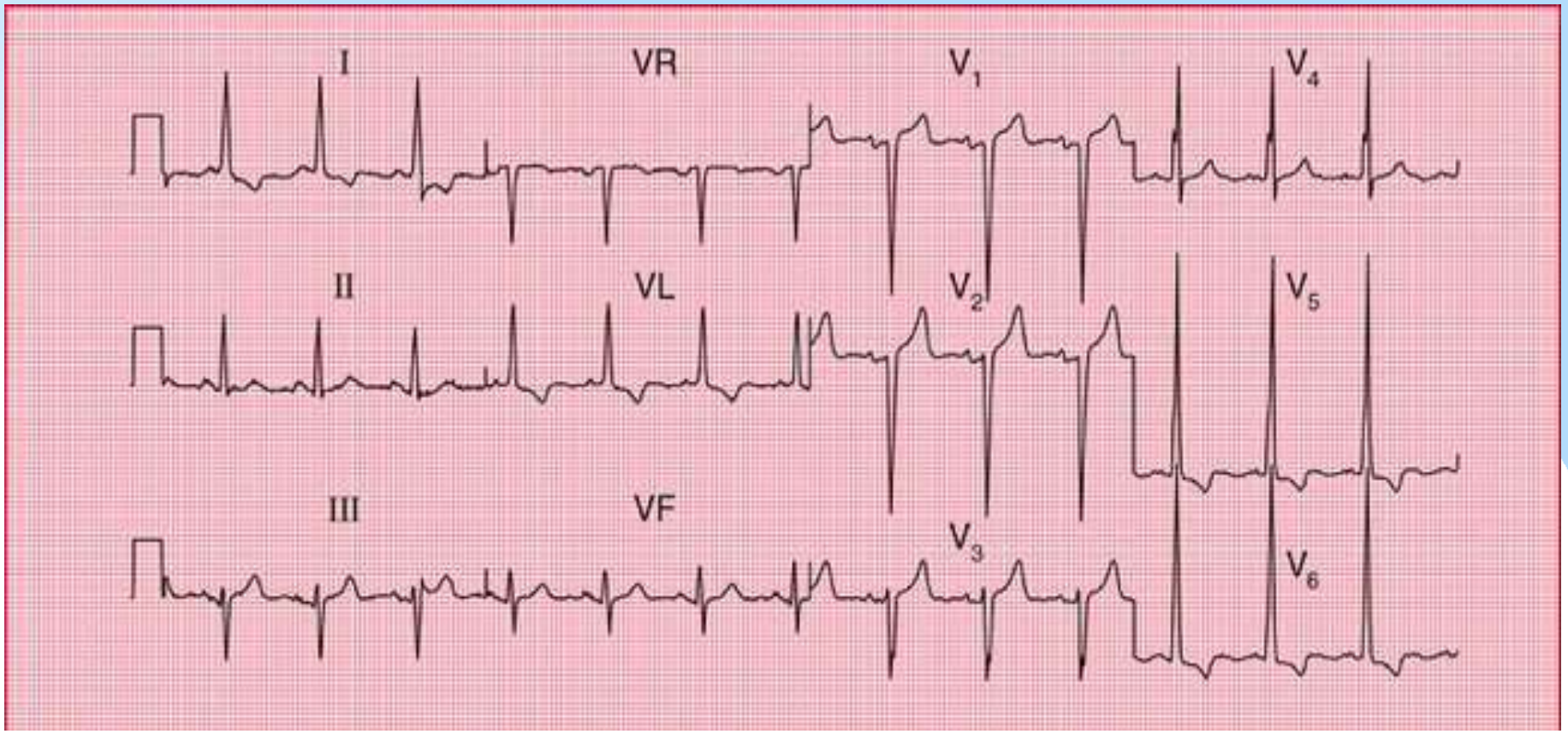
FIGURE 13-16 LVH increases the amplitude of electrical forces directed to the left and posteriorly. In addition, repolarization abnormalities can cause ST-segment depression and T wave inversion in leads with a prominent R wave (formerly referred to as a strain pattern). RVH can shift the QRS vector to the right; this effect is usually associated with an R, RS, or qR complex in lead V_1, especially when caused by severe pressure overload. T wave inversions may be present in the right precordial leads. *(From Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 7th ed. St. Louis, CV Mosby, 2006.)*

SEVERE RVH



- Sinus Rhythm, rate 63/min
- RAD (deep S wave in lead I)
- Dominant R wave in lead V1
- Inverted T wave in lead II,III,VF and V1-V3
- Flat T wave in V4-V6

LVH



- Sinus rhythm,rate 83/min
- Normal axis
- Tall R wave in V5-V6 and deep S waves in V1-V2
- Inverted T waves in lead I, aVL and V5-V6

TABLE 13-4 Common Diagnostic Criteria for Left Ventricular Hypertrophy

MEASUREMENT	CRITERIA
Sokolow-Lyon voltages	$SV_1 + RV_5 > 3.5$ mV RaVL > 1.1 mV
Romhilt-Estes point score system*	Any limb lead R wave or S wave > 2.0 mV (3 points) *or* SV_1 or $SV_2 \geq 3.0$ mV (3 points) *or* RV_5 to $RV_6 \geq 3.0$ mV (3 points) ST-T wave abnormality, no digitalis therapy (3 points) ST-T wave abnormality, digitalis therapy (1 point) Left atrial abnormality (3 points) Left axis deviation ≥ −30 degrees (2 points) QRS duration ≥ 90 msec (1 point) Intrinsicoid deflection in V_5 or $V_6 \geq 50$ msec (1 point)
Cornell voltage criteria	$SV_3 + RaVL \geq 2.8$ mV (for men) $SV_3 + RaVL > 2.0$ mV (for women)
Cornell regression equation	Risk of LVH = $1/(1+ e^{-exp})$†
Cornell voltage duration measurement	QRS duration × Cornell voltage > 2,436 mm-sec‡ QRS duration × sum of voltages in all leads > 1,742 mm-sec

Voltages are in mV, QRS is QRS duration in msec, PTF is the area under the P terminal force in lead V_1 (in mm-sec), and gender = 1 for men and 2 for women. LVH is diagnosed present if exp < −1.55.

*Probable left ventricular hypertrophy is diagnosed if 4 points are present and definite left ventricular hypertrophy is diagnosed if 5 or more points are present.

†For subjects in sinus rhythm, exp = 4.558 − 0.092 (SV_3 + RaVL) − 0.306 TV_1 − 0.212 QRS − 0.278 $PTFV_1$ − 0.559 (gender).

‡For women, add 8 mm.

TABLE 13-5 Common Diagnostic Criteria for Right Ventricular Hypertrophy

R in $V_1 \geq 0.7$ mV
QR in V_1
R/S in $V_1 > 1$ with $R > 0.5$ mV
R/S in V_5 or $V_6 < 1$
S in V_5 or $V_6 > 0.7$ mV
R in V_5 or $V_6 \geq 0.4$ mV with S in $V_1 \leq 0.2$ mV
Right axis deviation (>90 degrees)
S_1Q_3 pattern
$S_1S_2S_3$ pattern
P pulmonale

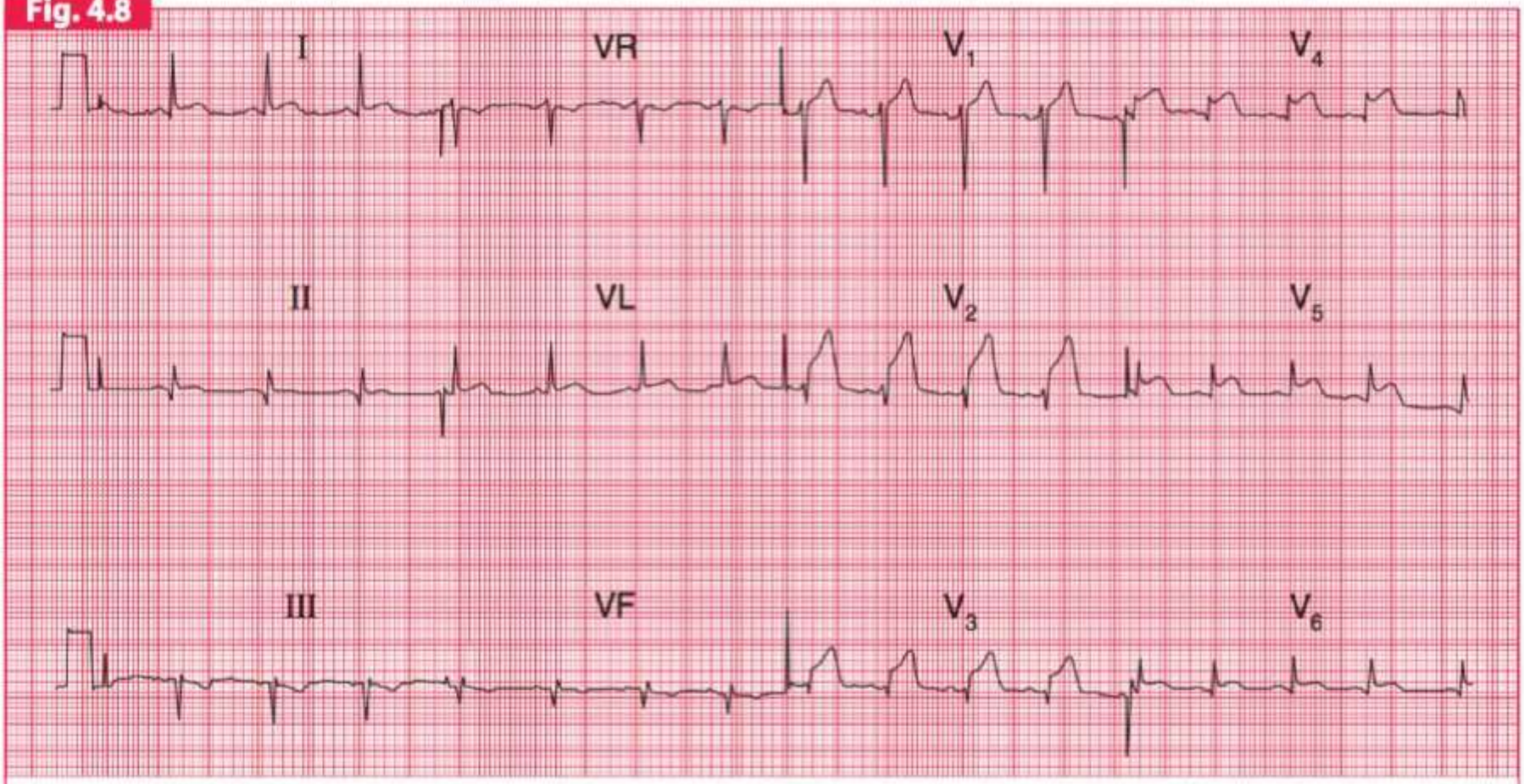
From Murphy ML, Thenabadu PN, de Soyza N, et al: Reevaluation of electrocardiographic criteria for left, right and combined cardiac ventricular hypertrophy. Am J Cardiol 53:1140, 1984.

THE ORIGIN OF Q WAVES

- Small (septal) ‘Q’ waves in the left ventricular leads result from depolarization of the septum from left to right

- Q wave greater than one small square in width and at least 2mm deep therefore indicate a myocardial infarction and the lead in which the Q wave appears give some indication of the part of the heart that has been damaged

Fig. 4.8



Acute anterior myocardial infarction, and probable old inferior infarction

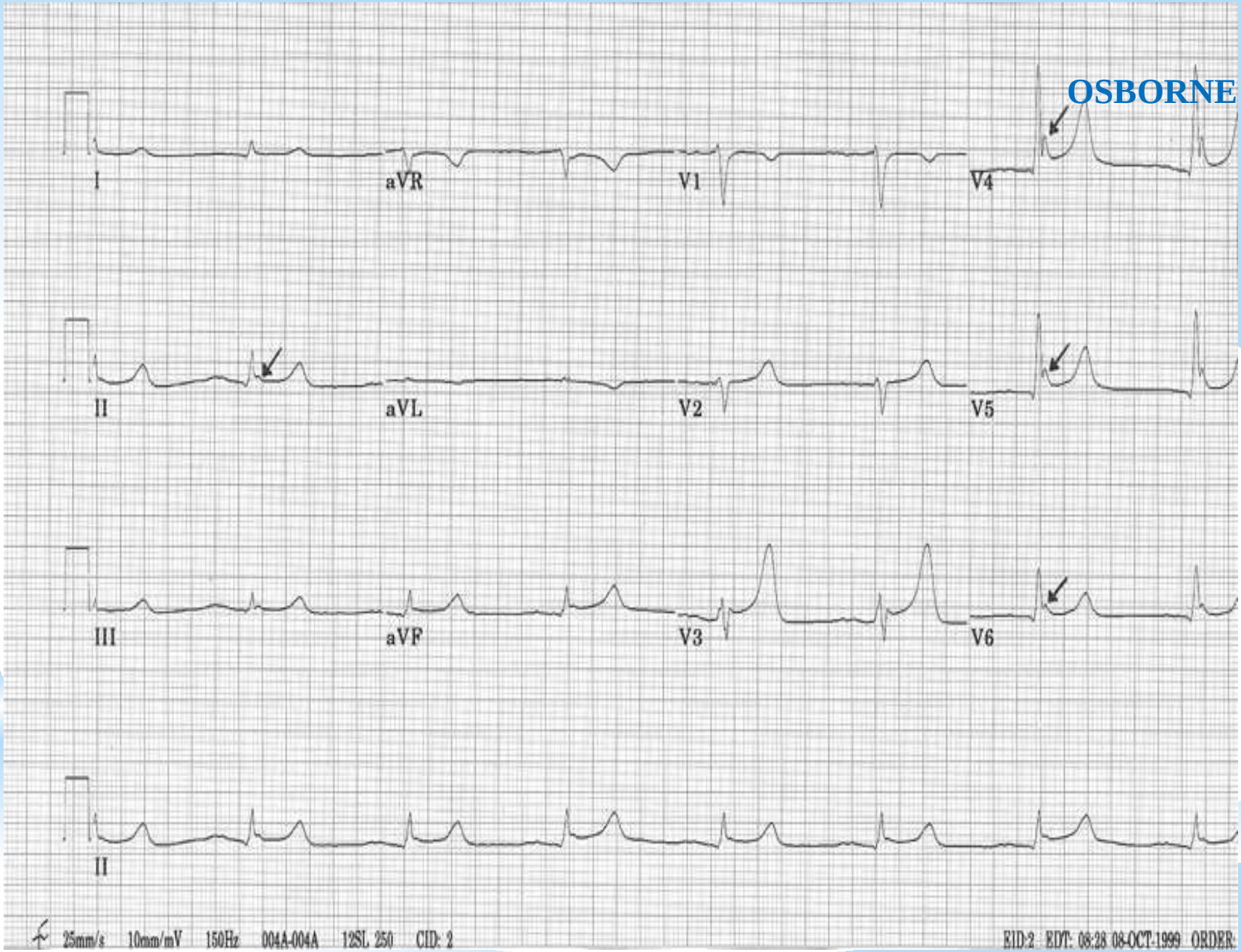
Note

- Sinus rhythm, rate 80/min
- Normal axis
- Small Q waves in leads II, III and VF – associated with flat ST segments and inverted T waves – indicate old inferior infarction
- Small Q waves in leads V_3–V_4 – associated with raised ST segments – indicate acute anterior infarction

J point

- J point is the junction between the termination of QRS complex and beginning of ST segment

- A positive deflection at the J point is termed a **J wave** (**Osborn wave**) and is characteristically seen with hypothermia


OSBORNE WAVE
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25mm/s 10mm/mV 150Hz 004A-004A 12SL 250 CID: 2
EID:2 EDT: 08:28 08-OCT-1999 ORDER:

ST Segment

- ST segment lies between the QRS complex and the T wav
- Should be isoelectric, indicates plateau portion of ventricular action potential
- Normal duration= 0.32 sec
- Elevation of the ST segment is an indication of acute myocardial injury, usually due either to a recent infarction
- or to pericarditis
- Horizontal depression of the ST segment, associated with an upright T wave, is usually a sign of ischemia as opposed to infarction

Fig. 4.12

The ST segment

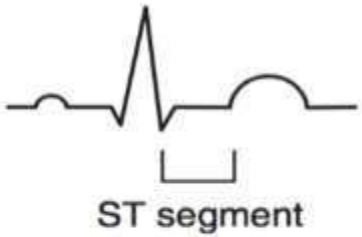
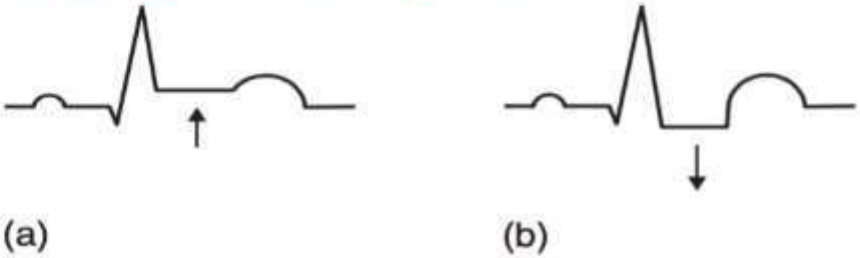


Fig. 4.13

(a) Elevated ST segment. (b) Depressed ST segment

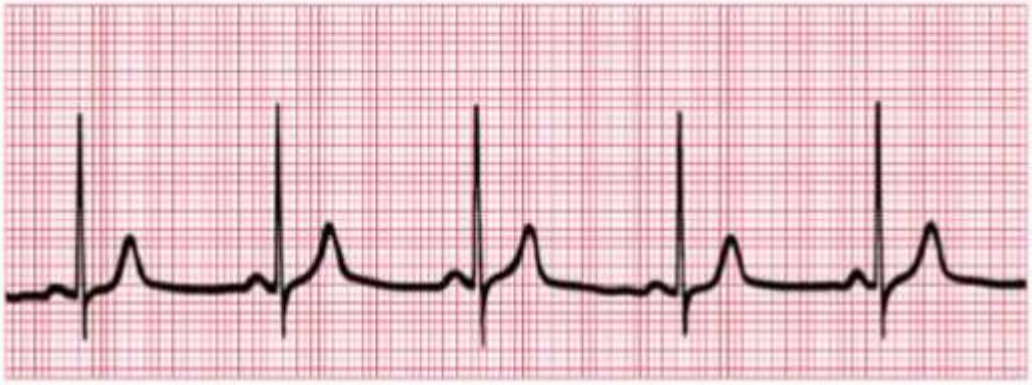


- To be considered significant elevation or depression , the ST segment must deviate 1mm below or above the baseline in at least 2 or more correlating leads

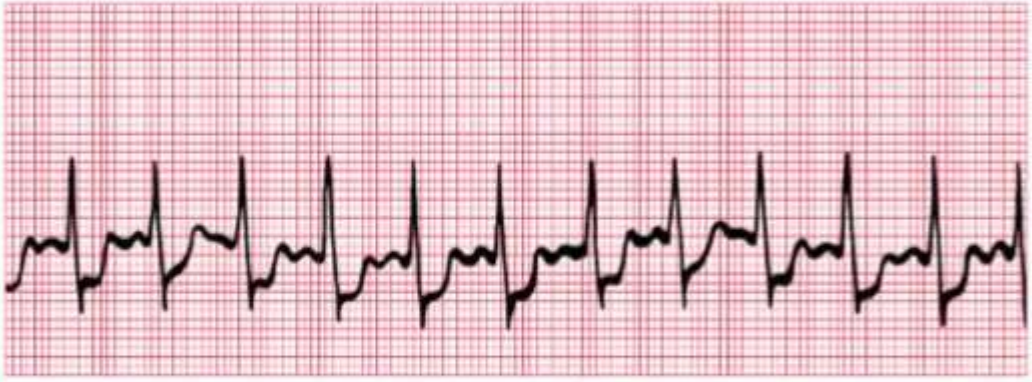
Fig. 4.14

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

T Wave

- Positive deflection (above baseline<5mm)
- Should appear rounded and symmetrical
- Should be atleast 1/8th but less than 2/3rd of the amplitude of R wave
- Positive in all standard bipolar limb leads (caused by repolarization of the apex and outer surfaces of the ventricles ahead of the interventricular surfaces)
- Normally inverted in lead aVR and V1

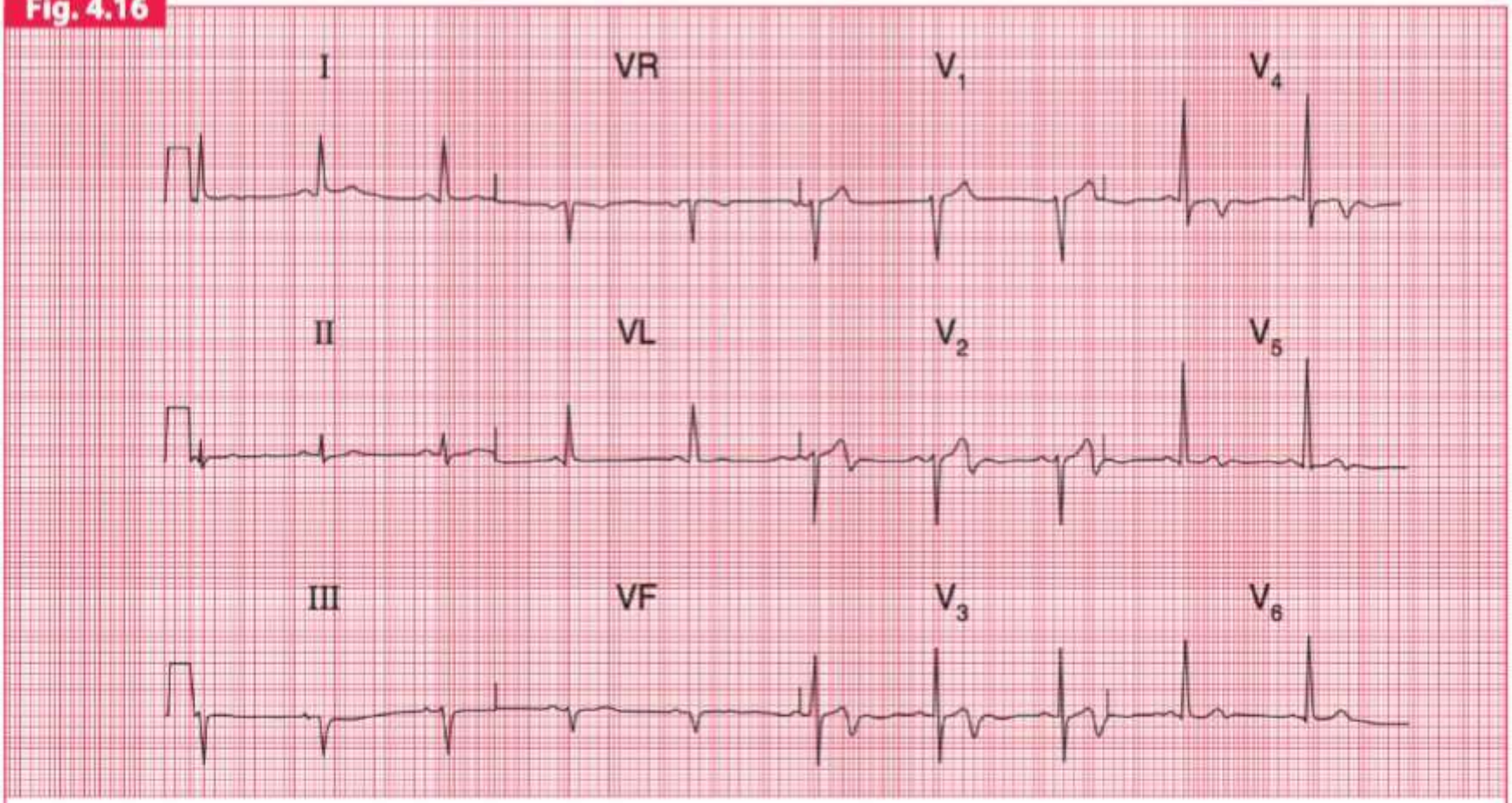
- Elevated T wave:-

✓ positive deflection >5mm
✓ Tall, pointed(tented) wave in hyperkalemia or myocardial injury

- T wave inversion is seen in the following circumstances:

1.Ischemia
2.Ventricular hypertrophy
3.Bundle branch block
4.Digoxin treatment.

Fig. 4.16



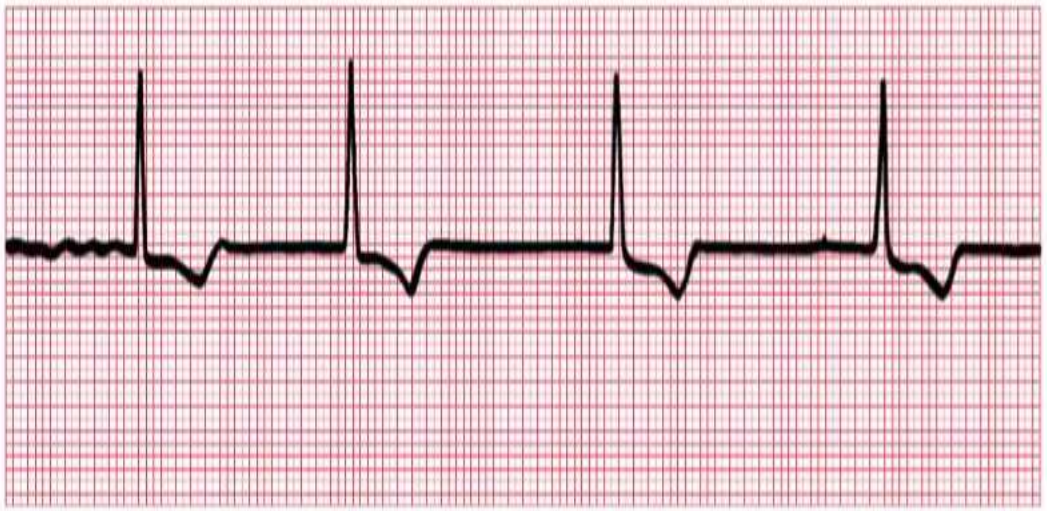
Anterior non-ST segment elevation myocardial infarction

Note

- Sinus rhythm, rate 62/min
- Normal axis
- Normal QRS complexes
- Inverted T waves in leads V_3–V_4
- Biphasic T waves in leads V_2 and V_5

Fig. 4.17

Digoxin effect



Note

- Atrial fibrillation
- Narrow QRS complexes
- Downward-sloping ST segments ('reversed tick')
- Inverted T waves

‘U’ Wave

- Indicates:-

✓ late repolarization of the Purkinje fibers
✓ long action potential of midmyocardial M cells
✓ delayed repolarization in areas of the ventricle that undergo late mechanical relaxation.

- < 0.1 mV in amplitude
- normally has the same polarity as the preceding T wave
- largest in the leads V1 and V2
- most often seen at slow heart rates

QT INTERVAL

- Total duration of depolarization and repolarization

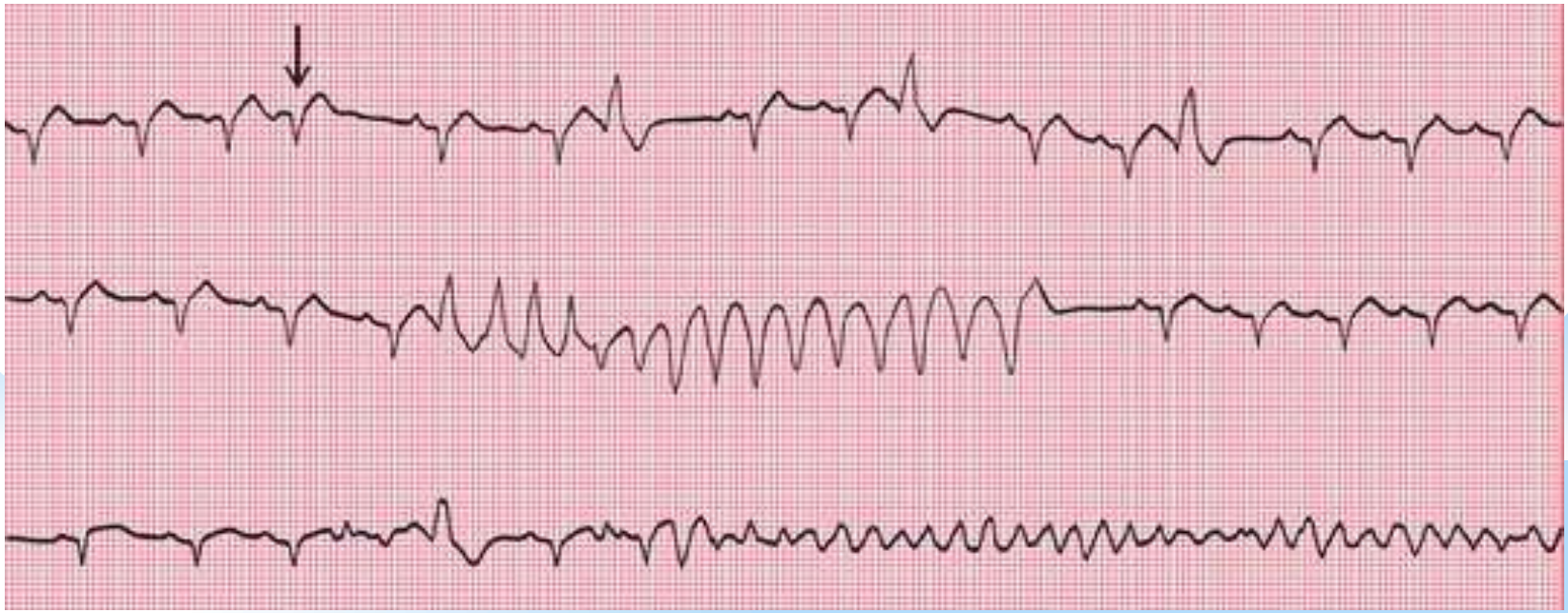
- Contraction of the ventricle lasts from the beginning of the Q wave (or R wave, if the Q wave is absent) to the end of the T wave. This interval is called Q-T interval and is about 0.35 second.
- The corrected interval (QTc) can be calculated using **Bazett's formula**:

QTc = QT ÷(square root of R–R interval)

- AHA/ACC:- QTc= QT+1.75(HR - 60)

- A QTc interval longer than 450 ms is considred to be abnormal.

- Whatever its cause, a corrected QT interval of 500 ms or longer can predispose to paroxysmal ventricular tachycardia of a particular type called ‘torsade de pointes’, which can cause either symptoms typical of paroxysmal tachycardia or sudden death



Box 7.1 Causes of prolonged QT interval

Congenital

- Jervell–Lange–Nielson syndrome
- Romano–Ward syndrome
- Several other genetic abnormalities also identified

Antiarrhythmic drugs

- Procainamide
- Disopyramide
- Amiodarone
- Sotalol

Other drugs

- Tricyclic antidepressants
- Erythromycin

Plasma electrolyte abnormalities

- Low potassium
- Low magnesium
- Low calcium

TABLE 13G-3 ACC/AHA Guidelines for Electrocardiography in Patients with No Apparent or Suspected Heart Disease or Dysfunction

SETTING	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Baseline or initial evaluation	Persons aged 40 yr or older undergoing physical examination Before administration of pharmacologic agents known to have high incidence of cardiovascular effects (e.g., antineoplastic agents) Before exercise stress testing People of any age in special occupations that require very high cardiovascular performance (e.g., fire fighters, police officers) or whose cardiovascular performance is linked to public safety (e.g., pilots, air traffic controllers, critical process operators, bus or truck drivers, railroad engineers)	To evaluate competitive athletes	Routine screening or baseline ECG in asymptomatic persons <40 yr with no risk factors
Response to therapy	To evaluate patients in whom prescribed therapy (e.g., doxorubicin) is known to produce cardiovascular effects	None	To assess treatment known not to produce any cardiovascular effects
Follow-up	To evaluate asymptomatic persons >40 yr of age	None	To evaluate asymptomatic adults who have had no interval change in symptoms, signs, or risk factors and who have had a normal ECG within recent past
Before surgery	Patients >40 yr of age Patients being evaluated as donor for heart transplantation or as recipient of noncardiopulmonary transplant	Patients 30-40 yr of age	Patients <30 yr with no risk factors for coronary artery disease

TABLE 13G-1 ACC/AHA Guidelines for Electrocardiography in Patients with Known Cardiovascular Disease or Dysfunction

INDICATION	CLASS I (INDICATED)	CLASS II (EQUIVOCAL)	CLASS III (NOT INDICATED)
Baseline or initial evaluation	All patients	None	None
Response to therapy	Patients in whom prescribed therapy is known to produce changes in the ECG that correlate with therapeutic responses or progression of disease Patients in whom prescribed therapy may produce adverse effects that may be predicted from or detected by changes in the ECG	None	Patients receiving pharmacologic or nonpharmacologic therapy not known to produce changes in the ECG or to affect conditions that may be associated with such changes
Follow-up	Patients with a change in symptoms, signs, or relevant laboratory findings Patients with an implanted pacemaker or antitachycardia device Patients with symptoms such as the following, even in the absence of new symptoms or signs, after an interval of time appropriate for the condition or disease: syncope and near-syncope, unexplained change in the usual pattern of angina pectoris, and who have no new chest pain or worsening dyspnea Extreme and unexplained fatigue, weakness and prostration; palpitations, new signs of congestive heart failure; new organic murmur or pericardial friction rub; new findings suggesting pulmonary hypertension; accelerating or poorly controlled systemic arterial hypertension; evidence of a recent cerebrovascular accident; unexplained fever in patients with known valvular disease; new onset of cardiac arrhythmia or inappropriate heart rate; chronic known congenital or acquired cardiovascular disease	None	Adult patients whose cardiovascular condition is usually benign and unlikely to progress (e.g., patients with asymptomatic mild mitral valve prolapse, mild hypertension, or premature contractions in absence of organic heart disease) Adult patients with chronic stable heart disease seen at frequent intervals (e.g., 4 months) and unexplained findings
Before surgery	All patients with known cardiovascular disease or dysfunction, except as noted under Class II	Patients with hemodynamically insignificant congenital or acquired heart disease, mild systemic hypertension, or infrequent premature complexes in absence of organic heart disease	None

TABLE 13G-2 ACC/AHA Guidelines for Electrocardiography in Patients Suspected of Having or at Increased Risk for Cardiovascular Disease or Dysfunction

SETTING	CLASS I (APPROPRIATE)	CLASS II (EQUIVOCAL)	CLASS III (INAPPROPRIATE)
Baseline or initial evaluation	All patients suspected of having or being at increased risk for cardiovascular disease Patients who may have used cocaine, amphetamines, or other illicit drugs known to have cardiac effects Patients who may have received an overdose of a drug known to have cardiac effects	None	None
Response to therapy	To assess therapy with cardioactive drugs in patients with suspected cardiac disease To assess response to administration of any agent known to result in cardiac abnormalities or abnormalities on the ECG (e.g., antineoplastic drugs, lithium, antidepressant agents)	To assess response to administration of any agent known to alter serum electrolyte concentration	To assess response to administration of agents known not to influence cardiac structure or function
Follow-up once	Presence of any change in clinical status or laboratory findings suggesting interval development of cardiac disease or dysfunction Periodic follow-up of patients (e.g., every 1 to 5 yr) known to be at increased risk for cardiac disease Follow-up of patients after resolution of chest pain	None	Follow-up ECGs more often than yearly not indicated for patients who remain clinically stable, not at increased risk for development of cardiac disease, and not demonstrated to have cardiac disease with previous studies
Before surgery	As part of preoperative evaluation of any patient with suspected, or at increased risk of developing, cardiac disease or dysfunction	None	None

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THANK YOU