

ECG

What is an ECG?

- The electrocardiogram (ECG/EKG) is a representation of the electrical events of the cardiac cycle.
- Each event has a distinctive waveform
- The study of waveform can lead to greater insight into a patient's cardiac pathophysiology.

Electrophysiology

Depolarization of the heart is the initiating event for cardiac contraction.

Electric currents in the heart are produced by three components cardiac pacemaker cells, specialised conduction tissue and heart muscle itself

ECG records only the depolarisation and repolarisation of atrial and ventricular myocardium

Electrophysiology

Depolarisation stimulus originates in the SA node a collection of pacemaker cells. These cells have automaticity they fire spontaneously.

First phase of cardiac electrical activation is the spread of the depolarisation wave over the right and left atria followed by atrial contraction.

Electrophysiology

The impulse stimulates pacemaker and specialised conduction tissues in the atrioventricular nodal and His bundle areas

Bundle of HIS bifurcates into right and left branches which rapidly transmit depolarisation wave fronts to the right and left myocardium by way of the Purkinje fibres

Electrophysiology

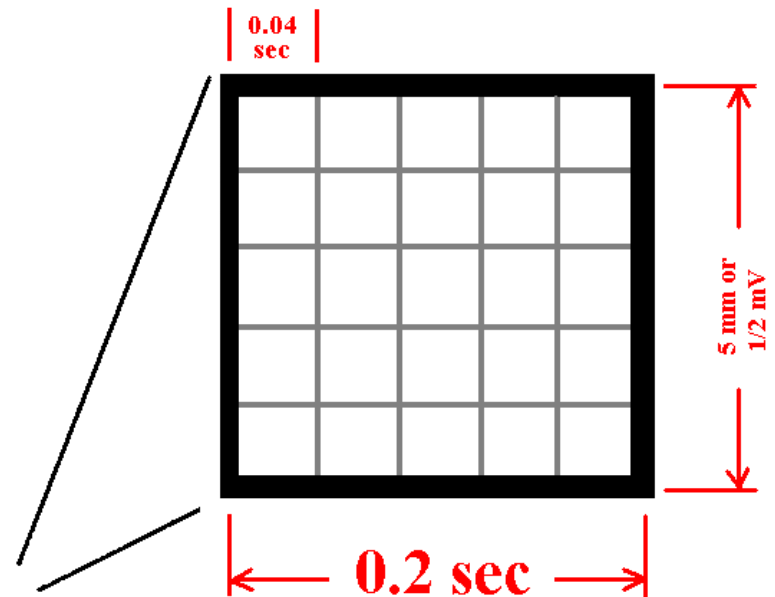
Main left bundle branch bifurcates into two primary subdivisions a left anterior fascicle and a left posterior fascicle

Depolarisation wave fronts then spread through the ventricular wall from endocardium to epicardium

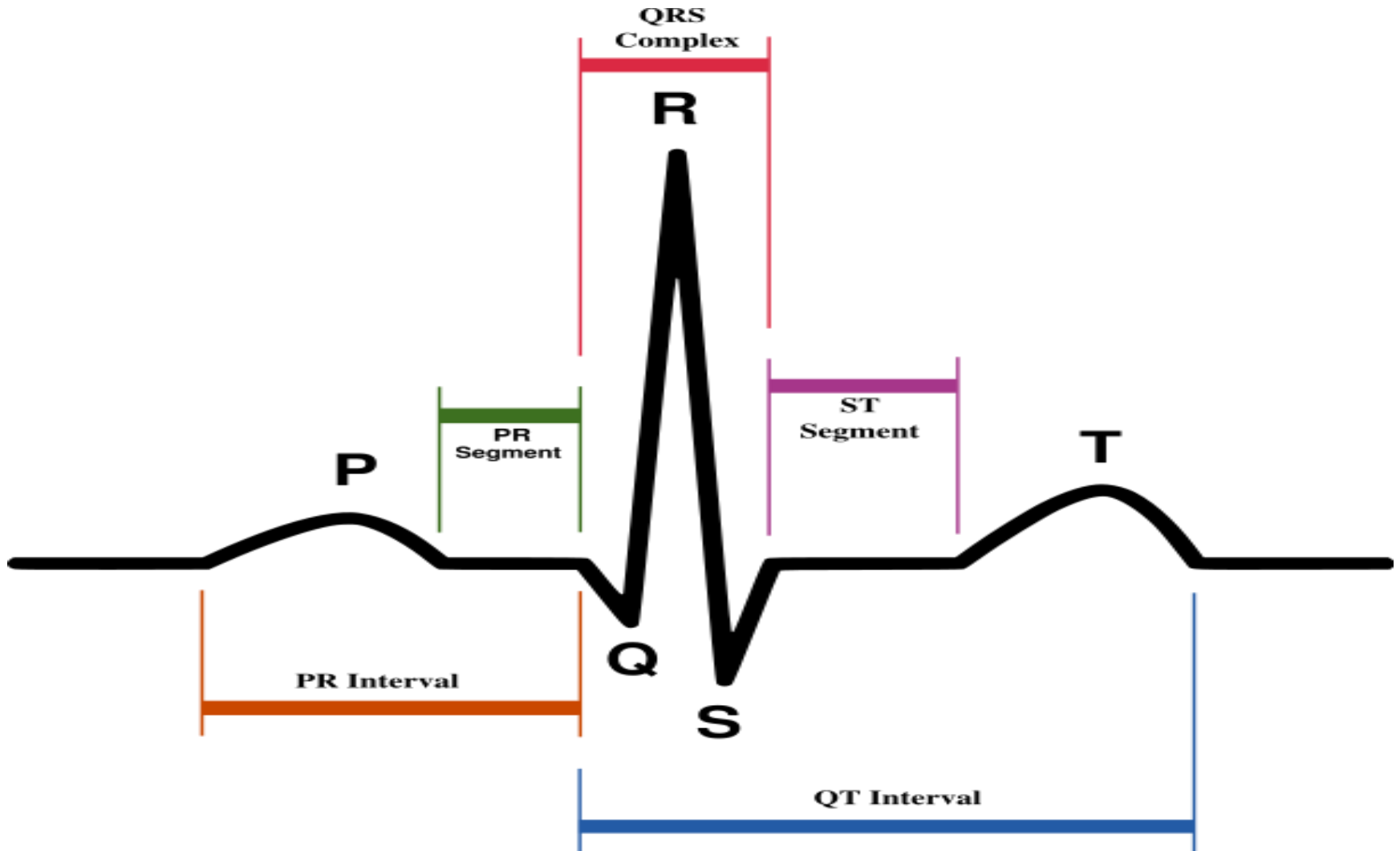
This triggers ventricular contraction

The ECG Paper

- Horizontally
 - One small box - 0.04 s
 - One large box - 0.20 s
- Vertically
 - One large box - 0.5 mV



NORMAL ECG



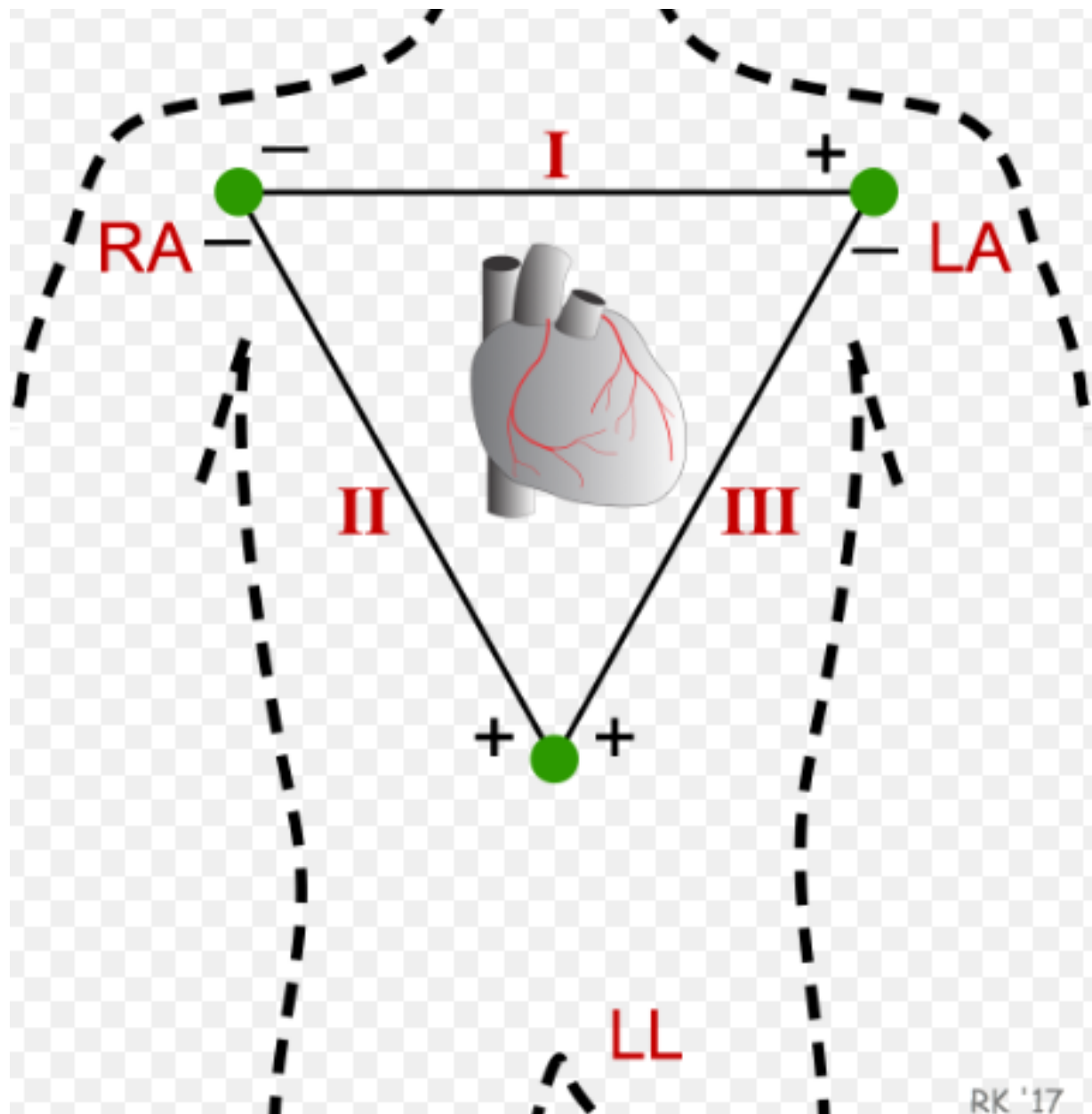
ECG Leads

The standard ECG has 12 leads:

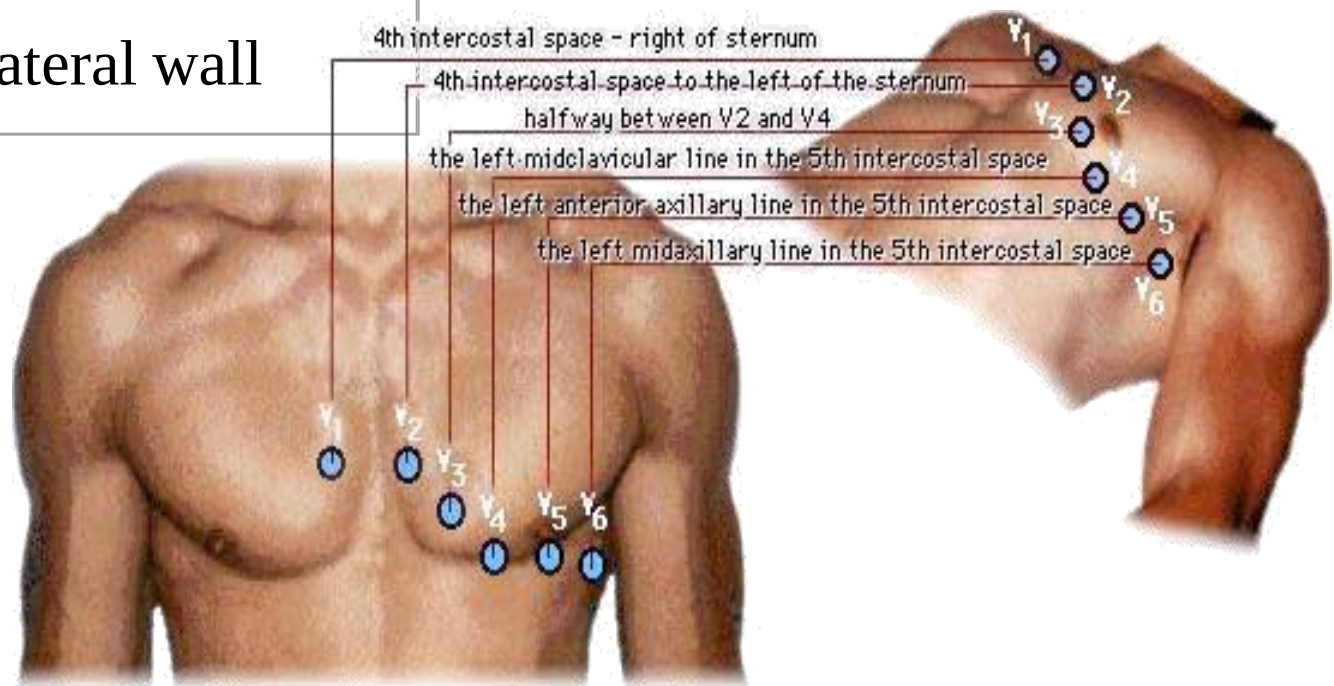
3 Standard Limb Leads

3 Augmented Limb Leads

6 Precordial Leads



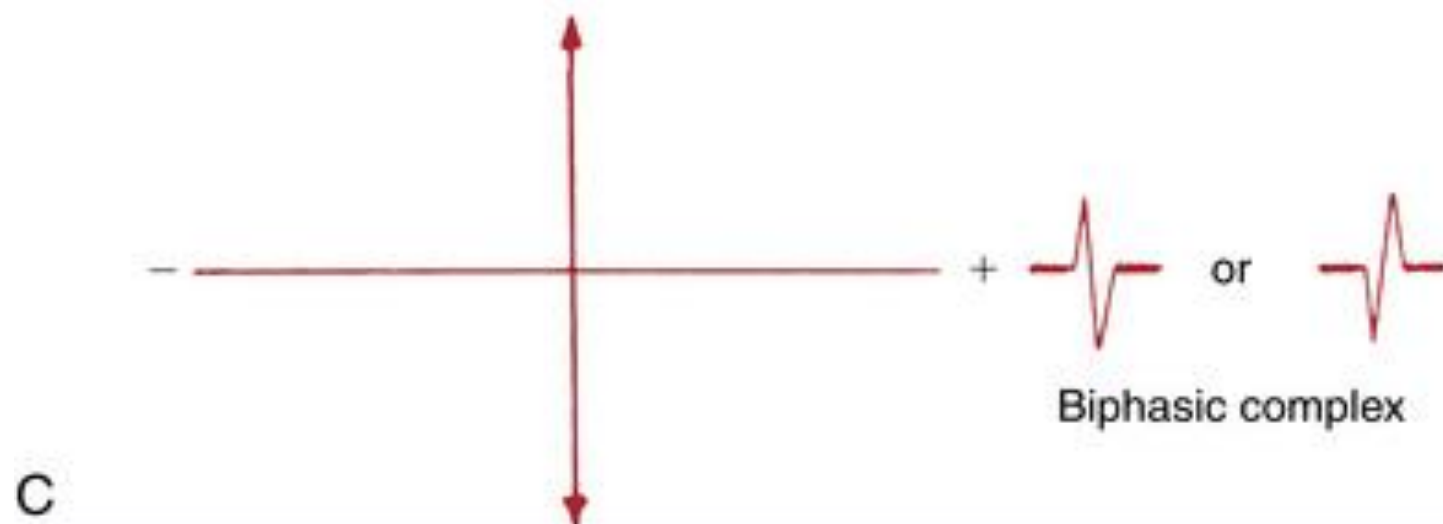
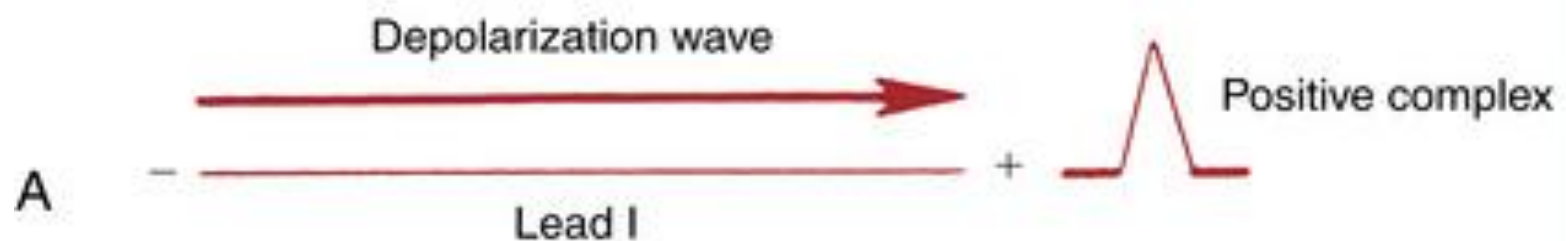
V1-V2	anteroseptal wall
V3-V4	anterior wall
V5-V6	anterolateral wall
II, III, aVF	inferior wall
I, aVL	lateral wall



ECG leads are configured so that a positive deflection is recorded in a lead if a wave of depolarisation spreads towards the positive pole of that lead and a negative deflection is recorded if the wave spreads towards the negative pole

If mean orientation of the depolarisation vector is at right angles to a particular lead axis a biphasic deflection will be recorded

Three Basic Laws of Electrocardiography



Electrophysiology

QRS complex is subdivided into special deflections or waves

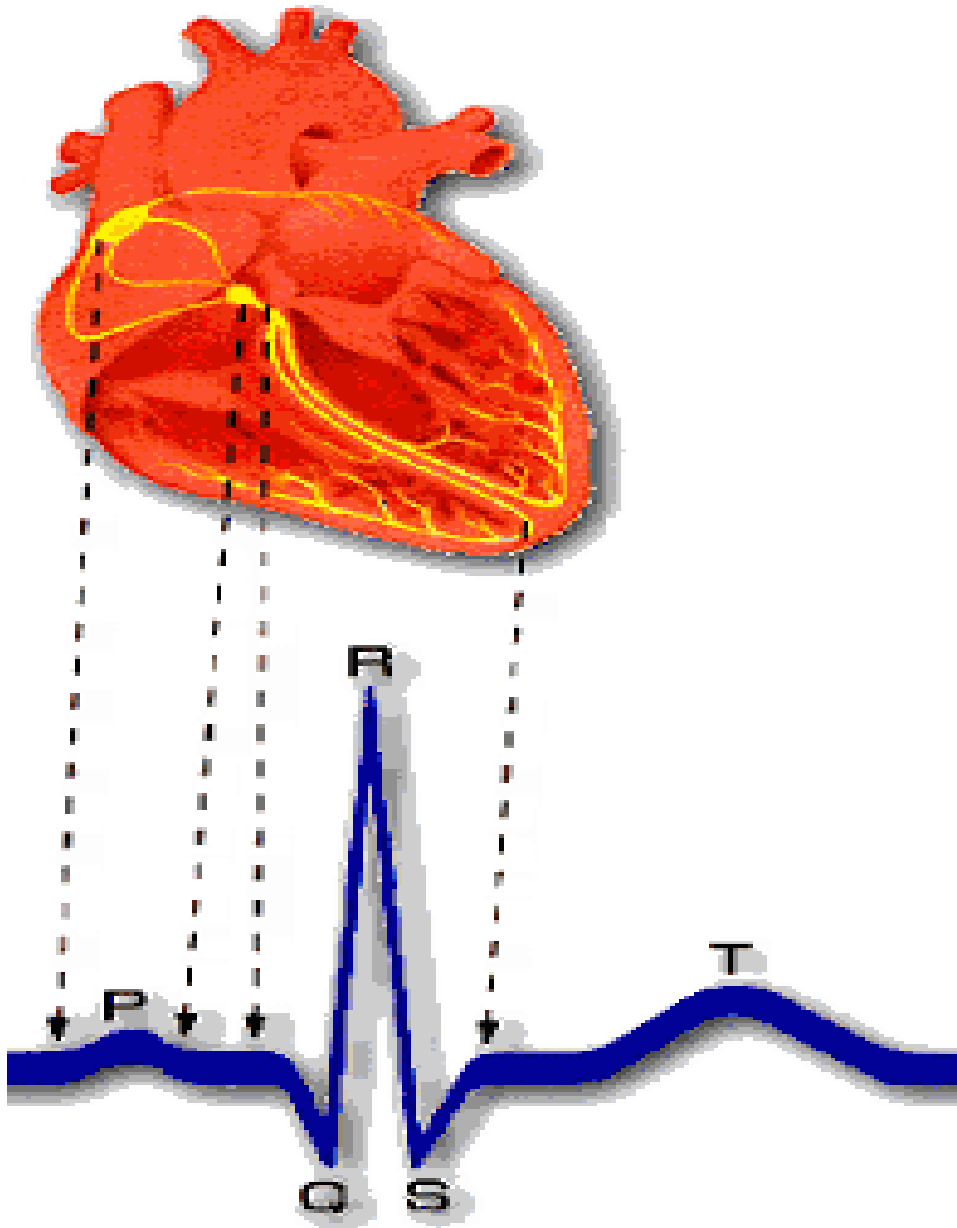
Initial QRS deflection in a lead is negative it is termed as Q wave, first positive deflection is termed as an R wave

A negative deflection after an R wave is an S wave

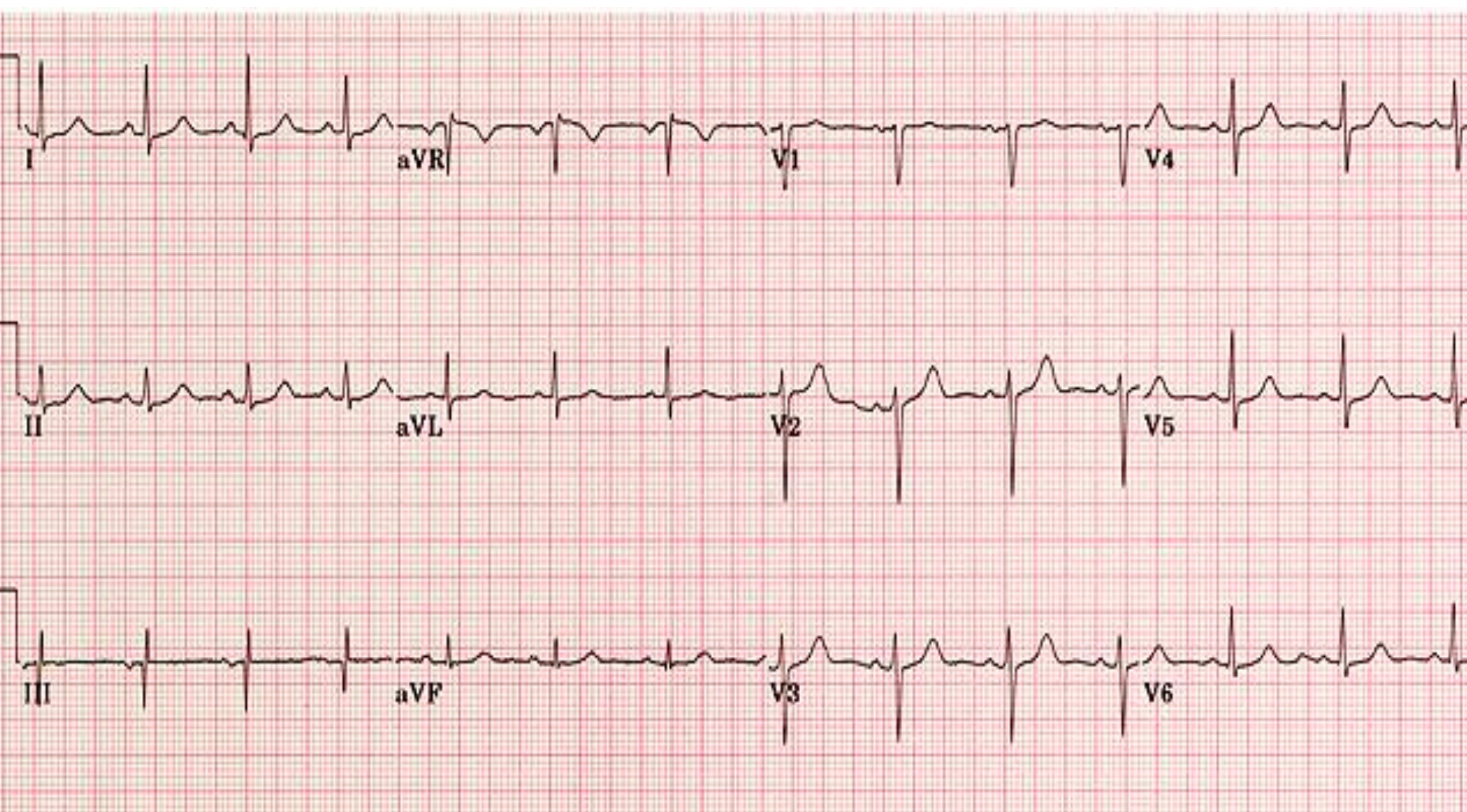
Subsequent positive or negative waves are termed as R' and S' respectively

Lower case letters for small amplitude

Normal ECG pattern



- P wave – atrial depolarization or activation (Amp<0.25, Duration<0.12s)
- PR segment – AV conduction system activation
- PR interval – onset of atrial depolarization to ventricular repolarization
- QRS complex – depolarization of ventricle
- ST – T – ventricular repolarization or recovery
- U wave – repolarization of purkinje
- J point – junction between QRS and ST segment



Genesis of waves

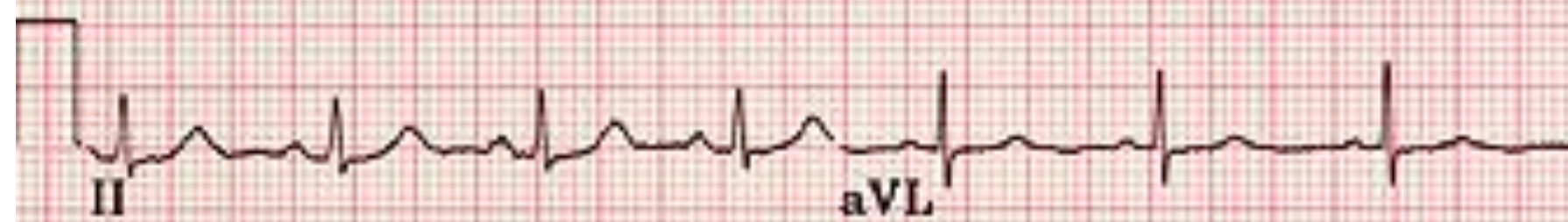
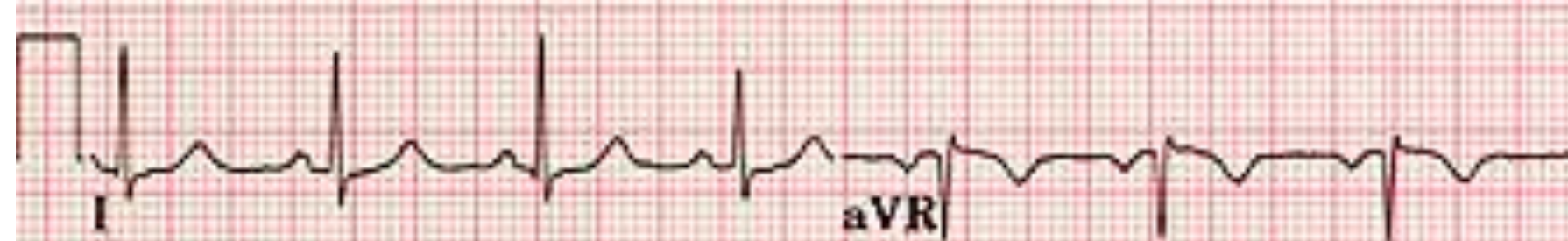
P wave:

Atrial depolarisation vector is oriented down and to the subjects left

The vector points towards lead II.

P wave is positive in Lead II and negative in lead aVR

In lead V1 P wave is biphasic



QRS wave

First phase is depolarisation of the interventricular septum from left to right and anteriorly

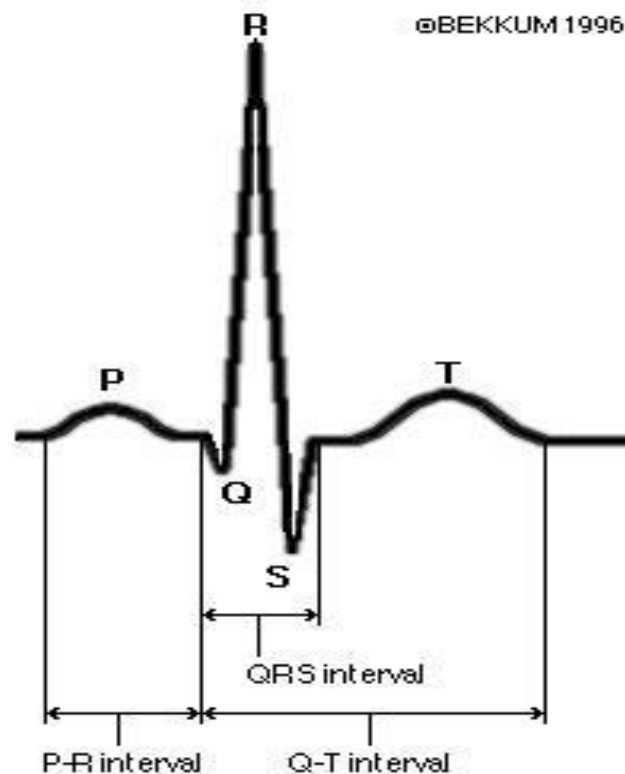
Second results from simultaneous depolarisation of right and left ventricles. As left ventricle is larger the main vector points towards left

Hence in V1 we get a small positive wave first followed by a large deep S wave

In V6 small -ve deflection followed by large +ve wave

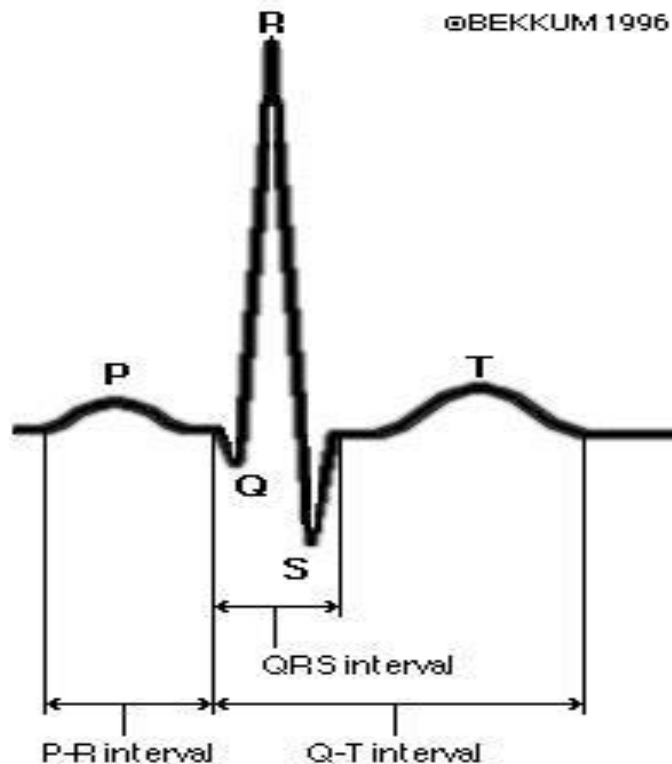
Intervals

An interval is a portion of the baseline and at least one wave. We measure an interval on the horizontal axis in seconds. The PR, QRS, and QT are the intervals which should be routinely scanned on each ECG. For measuring intervals, look at the widest form in any lead



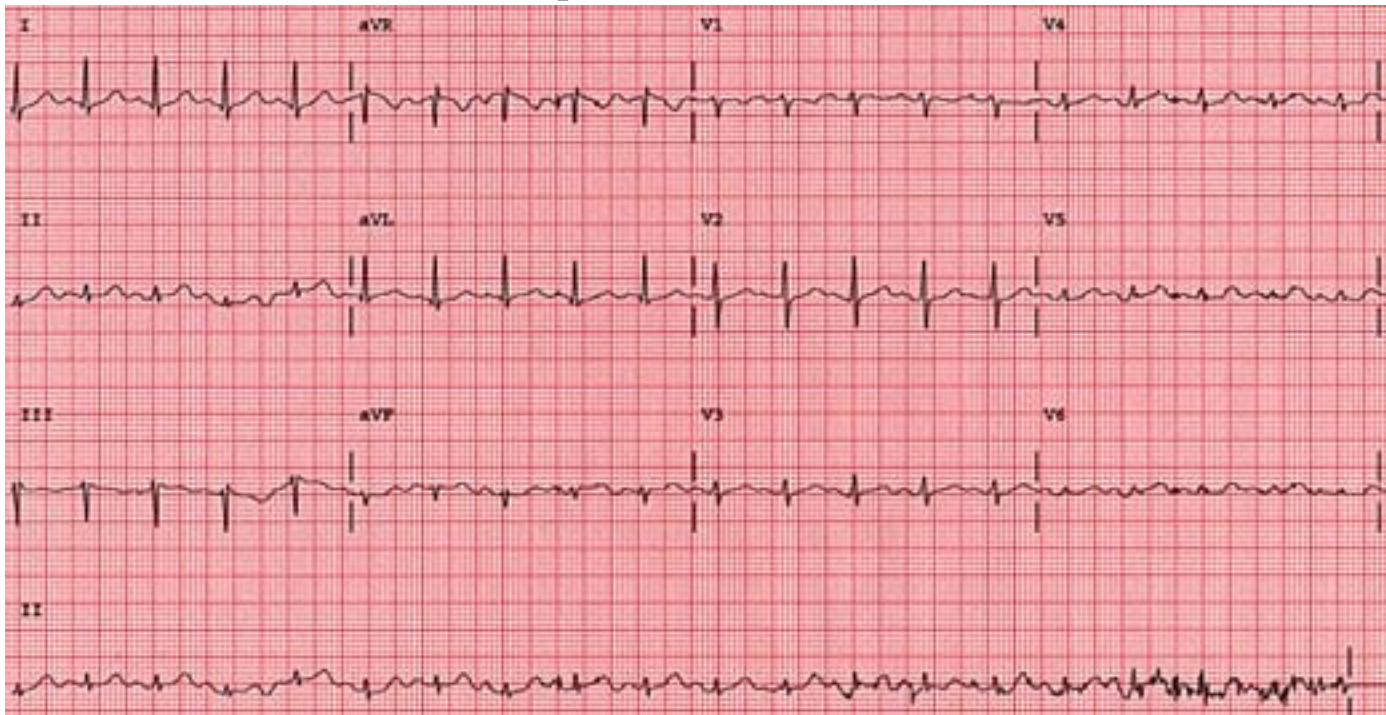
P wave (Amp<0.25, Duration<0.12s)

1. PR interval (beginning of P wave to the beginning of the next QRS). Normally, < .2 seconds or one large box. If it is > .2 seconds, it is a first degree block
2. QRS interval (beginning of Q to the end of the S wave) should be < .12 seconds (< 3 small boxes). If QRS is > .12, check for bundle branch block.
3. A QRS > .12 and RR (2 peaks or R waves in QRS) occurring in the right chest leads (V1-V2) indicates a right bundle branch block



1. QT interval (beginning of QRS to end of T wave) should be less than half of the preceding RR interval - this varies with the rate. For normal rates, $QT < .4$ seconds (2 large boxes). "QT prolongation" (too long) can lead to a refractory form of ventricular tachycardia called *torsades de pointes*.

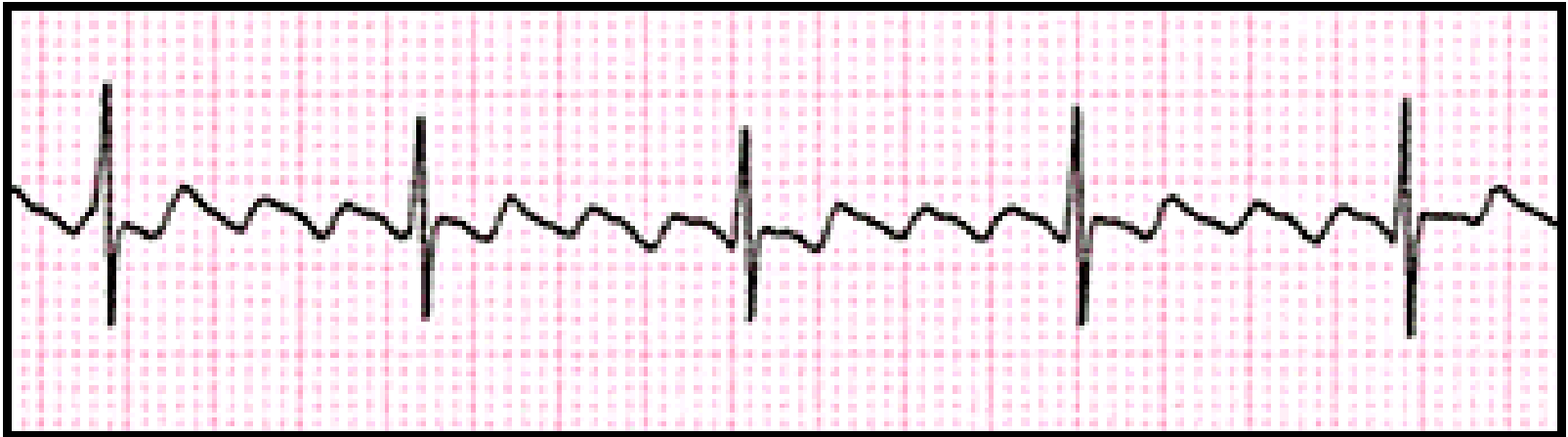
Bazetts formula for $QT_c = QT / \sqrt{RR}$



ECG interpretation: look at five areas, in order, on each ECG.

Rate
Rhythm (Intervals)
Axis
Hypertrophy
Infarct

What is the heart rate?

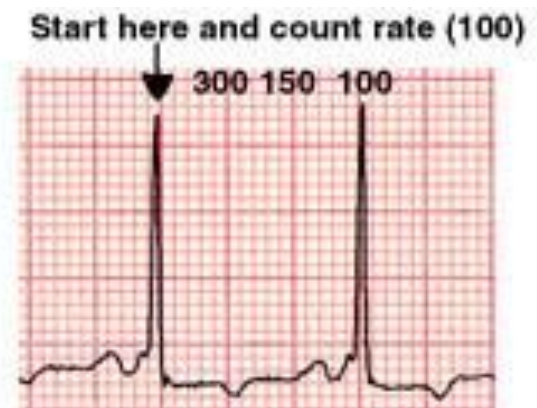
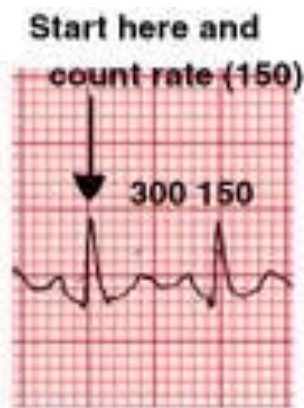


$$(300 / \sim 4) = \sim 75 \text{ bpm}$$

- Rate is cycles or beats per minute.
- Normal rate for the SA node 60-100.
- SA node is the usual pacemaker, other potential pacemakers (if SA node fails) are
 - atrial pacemakers with inherent rates of 60-80,
 - AV node (rate 40-60),
 - or ventricular pacer (rate 20-40).
- In certain pathologic conditions ectopic (out of place) pacemakers can go much faster at rates 150-250 cycles/minute

1. *Most Common Method:*

(Most rates can be calculated this way). Find an R wave on a heavy line (large box) count off "300, 150, 100, 75, 60, 50" for each large box you land on until you reach the next R wave. Estimate the rate if the second R wave doesn't fall on a heavy black line.



1. *Mathematical method:* $300 / [\# \text{ large boxes between two R waves}]$.



The Rule of 300

It may be easiest to memorize the following table:

No of big boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50

1.Rhythm Guidelines

1.1. Check the bottom of the rhythm strip for regularity, i.e. - is it completely regular, mostly regular with a few extra beats, or totally irregular?

1.2. Check for a P wave before each QRS, QRS after each P.

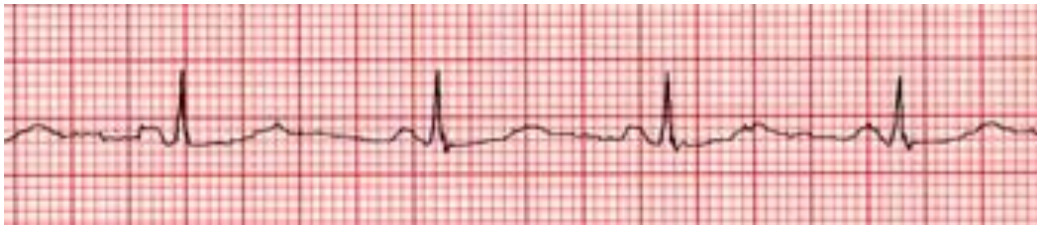
1.3. Check RR interval (for AV blocks) and QRS interval (for bundle branch blocks). Check for prolonged QT.

1.4. Continue to recognize "patterns" such as atrial fibrillation, PVCs, PACs, escape beats, ventricular tachycardia, paroxysmal atrial tachycardia, AV blocks and bundle branch blocks.

The normal conduction pathway is: SA node --> AV node --> Bundle of HIS --> Bundle Branches.

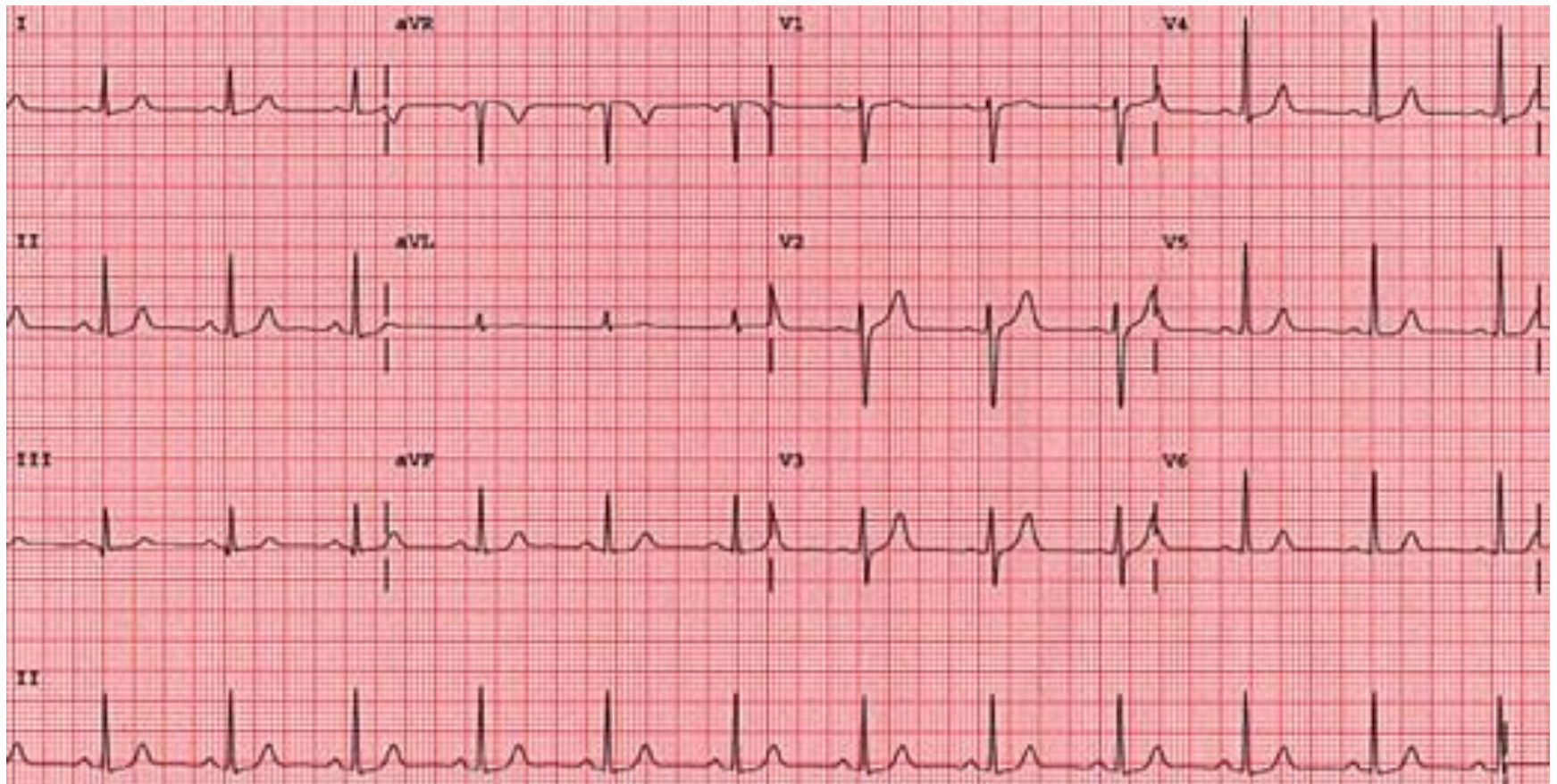
Arrhythmia can be understood by realizing the existence of ectopic (out of place) foci (pacemakers) and understanding the normal conduction pathway of the heart.

if the beat originates in the *atria or AV node* (supraventricular) the *QRS is usually narrow* (normal), because it comes from above along the normal pathway



If the beat is *ventricular in origin*, the *QRS is wide and bizarre* because it doesn't come down the normal pathway.





*Limb Leads I, II, III
aVR, aVF, aVL*

*Chest Leads
V1 - V6*

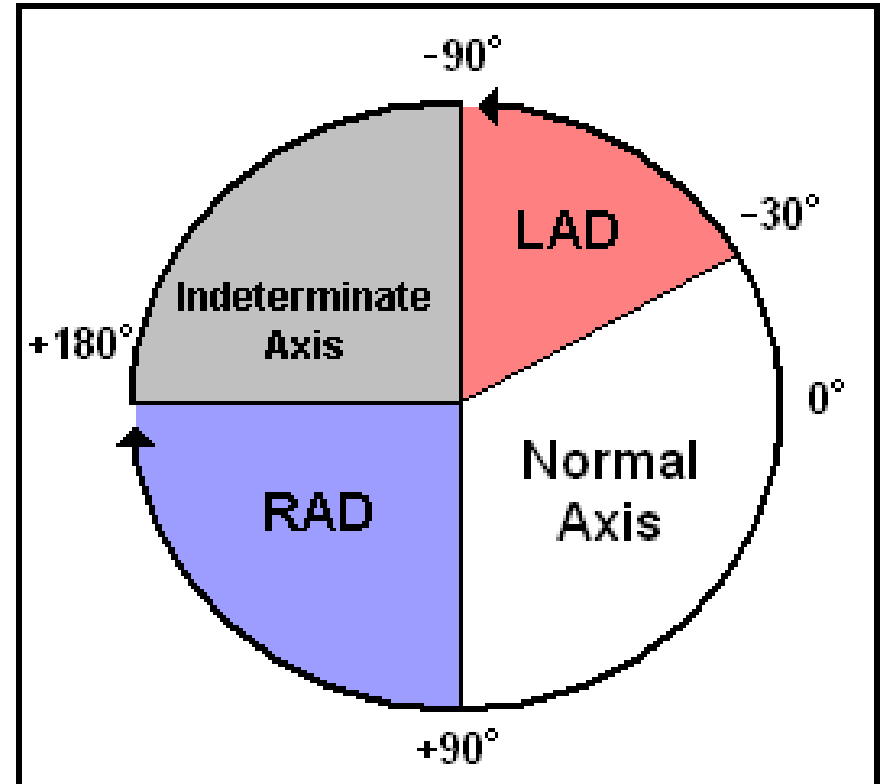
*Rhythm Strip Located on the bottom of the ECG printout.
Selected to give the best relationship of the P wave to the QRS.*

The QRS Axis

Normal QRS axis from -30° to $+90^\circ$.

-30° to -90° is referred to as a left axis deviation (LAD)

$+90^\circ$ to $+180^\circ$ is referred to as a right axis deviation (RAD)



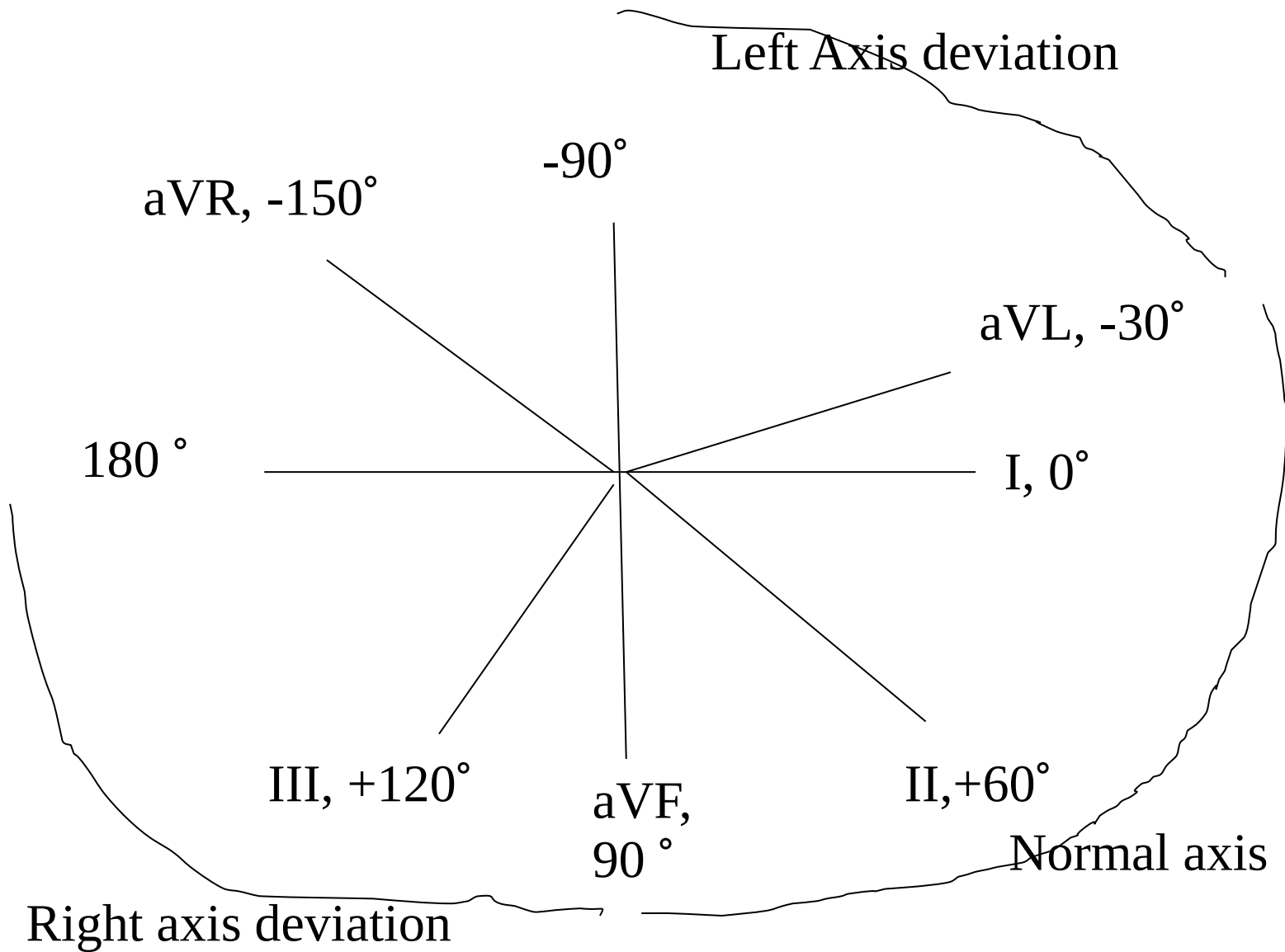
The QRS Axis

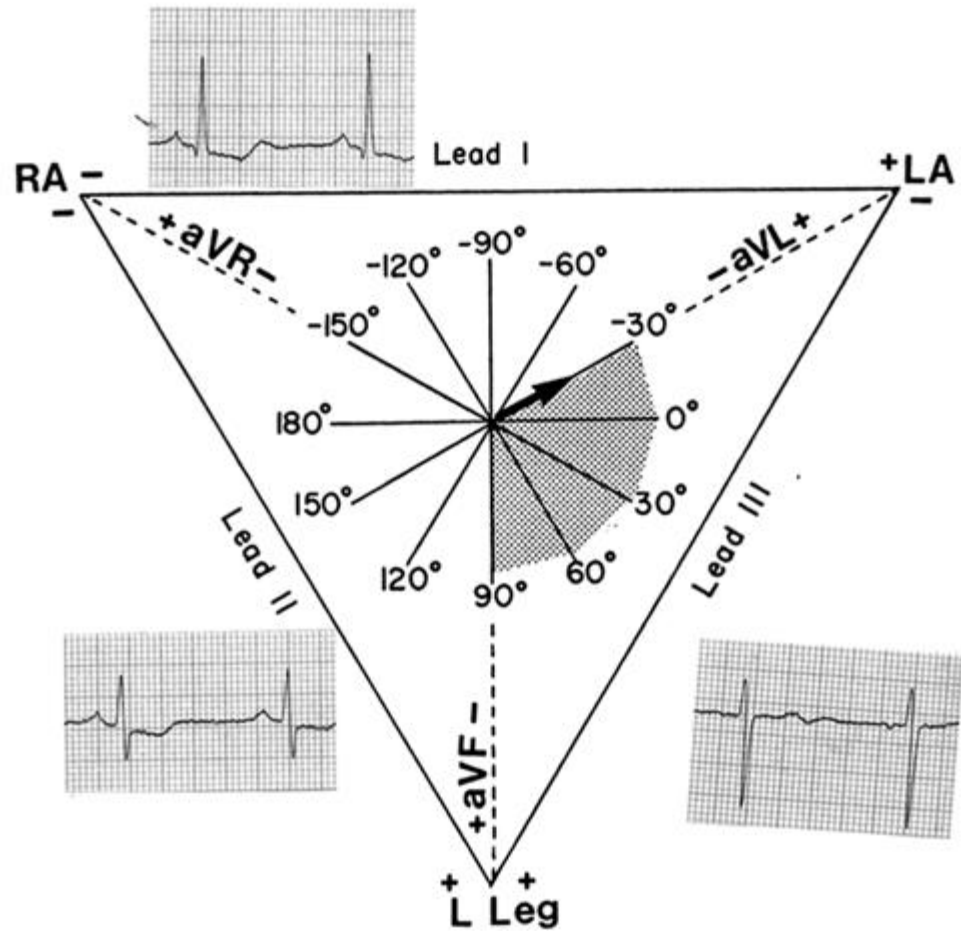
The QRS axis represents overall direction of the heart's electrical activity.

Abnormalities hint at:

- Ventricular enlargement

- Conduction blocks (i.e. hemiblocks)





QRS Axis = -30 degrees

ABNORMAL ECG

P Pulmonale

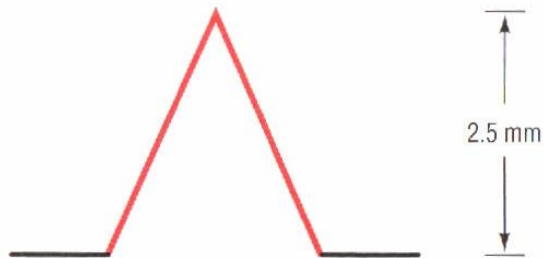
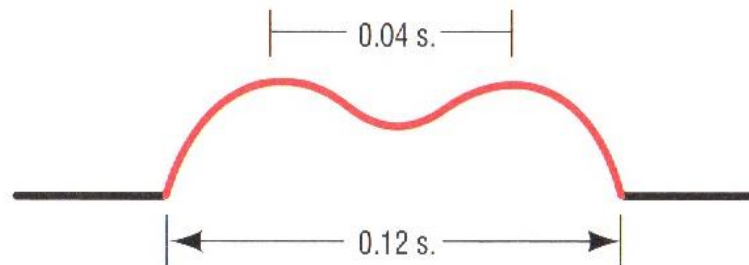


Figure 12-4: A peaked P wave taller than 2.5 mm in the limb leads indicates P-pulmonale.

P Mitrale



Cardiac enlargement and hypertrophy

Right atrial overload may lead to increase in P wave amplitude called as 'p pulmonale'.

Left atrial overload produces a biphasic p wave in V1 with a broad negative component or broad notched p waves called as 'p mitrale'

Ventricular Hypertrophy

- LVH $S1 + R6 > 35$
-
- RVH $R1 + S6 > 10$

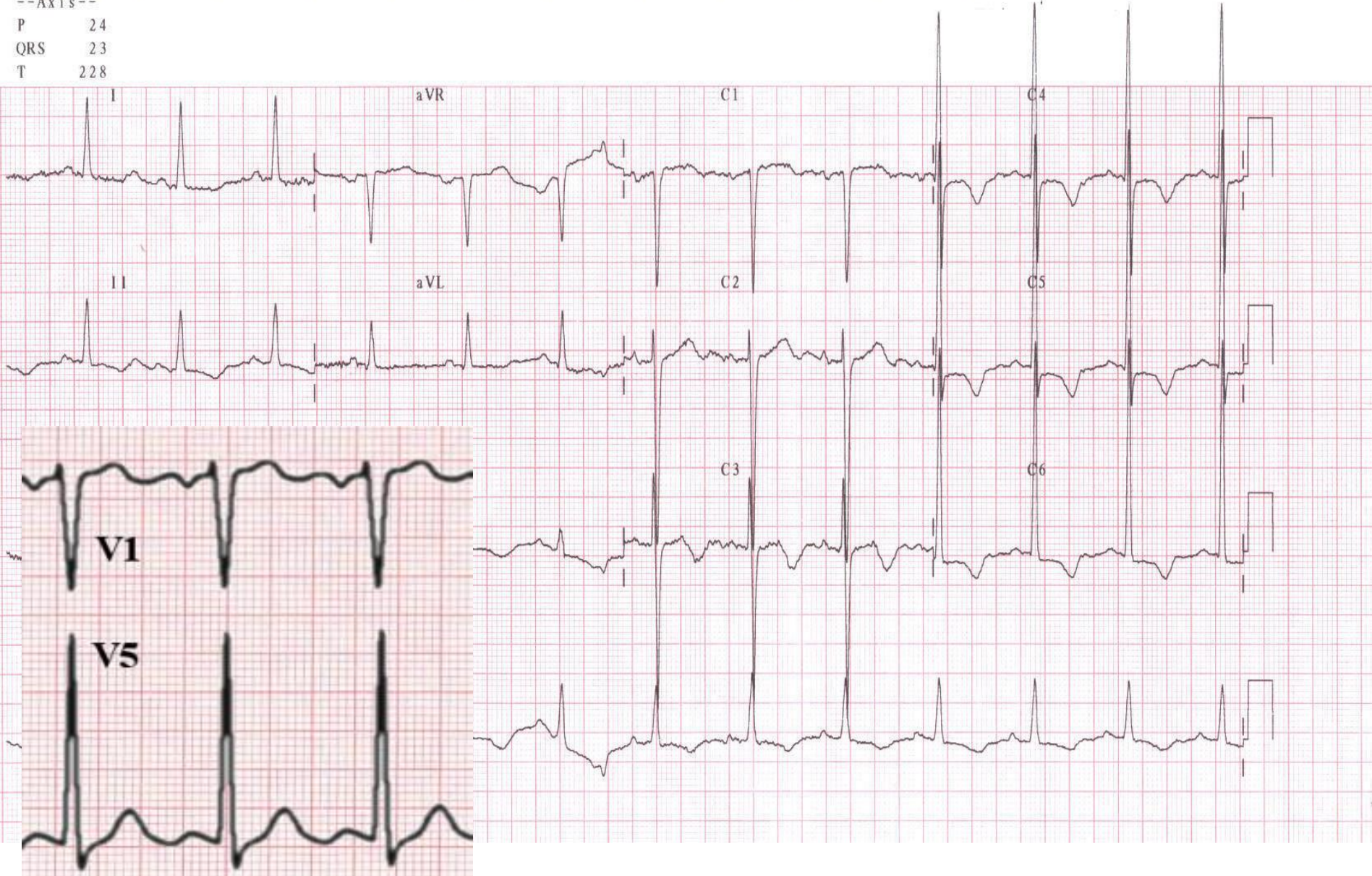
LVH

--Axis--

P 24

QRS 23

T 228



RVH

RVH : Tall R wave in V1 with right axis deviation

ST depression and T inversion in right to mid precordial leads

May be seen in Pulmonary stenosis or PAH

S waves may be seen in the left lateral leads

Acute cor pulmonale

Acute cor pulmonale due to pulmonary embolism may be associated with sinus tachycardia

QRS axis may shift to right along with sometimes S1 Q3T3 pattern

Seen in Pulmonary embolism

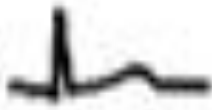
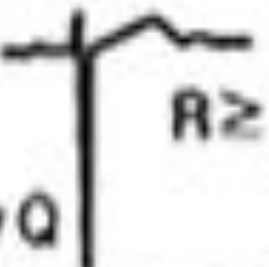
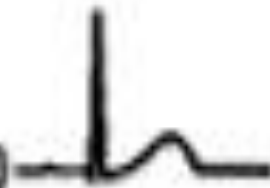
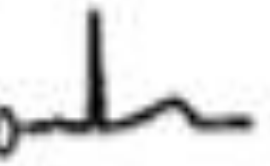
Left ventricular hypertrophy

LVH=

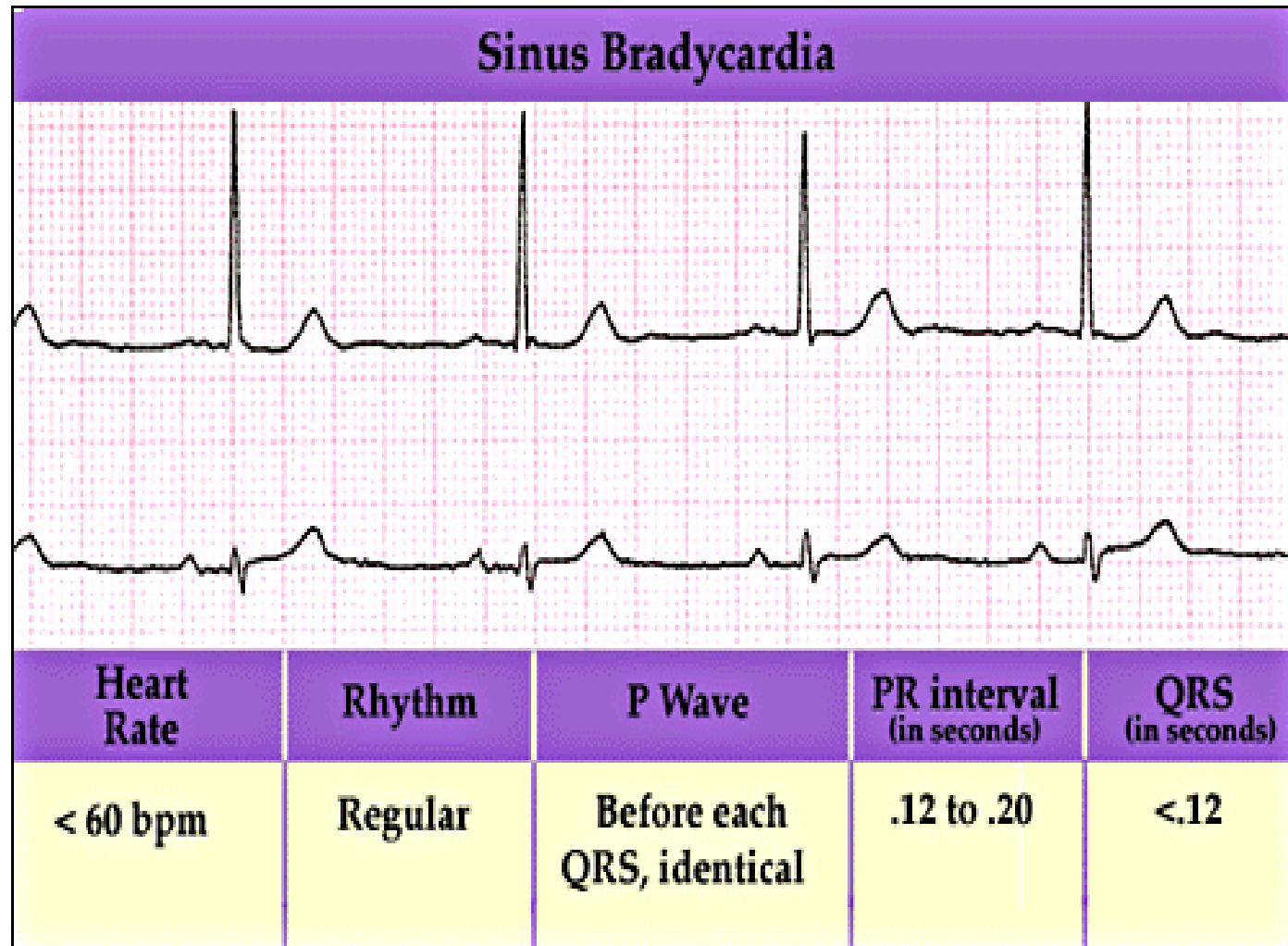
$S V1 + R V5 \text{ OR } R V6 = 35 \text{ MM}$

Repolarisation abnormalities may also appear in leads with prominent R waves

Pathologic widths for Q waves and R waves for selected leads

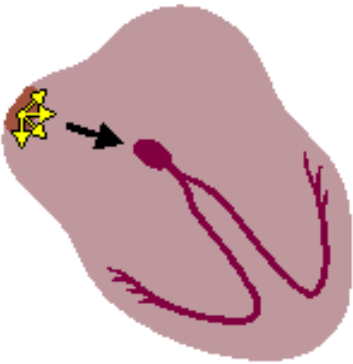
I Q ≥ 30 	aVR 	V ₁ Any Q R ≥ 40 	V ₄ Q ≥ 20 
II Q ≥ 30 	aVL Q ≥ 30 	V ₂ Any Q R ≥ 50 	V ₅ Q ≥ 30 
III 	aVF Q ≥ 30 	V ₃ Any Q 	V ₆ Q ≥ 30 

Sinus bradycardia.



SINUS BRADYCARDIA

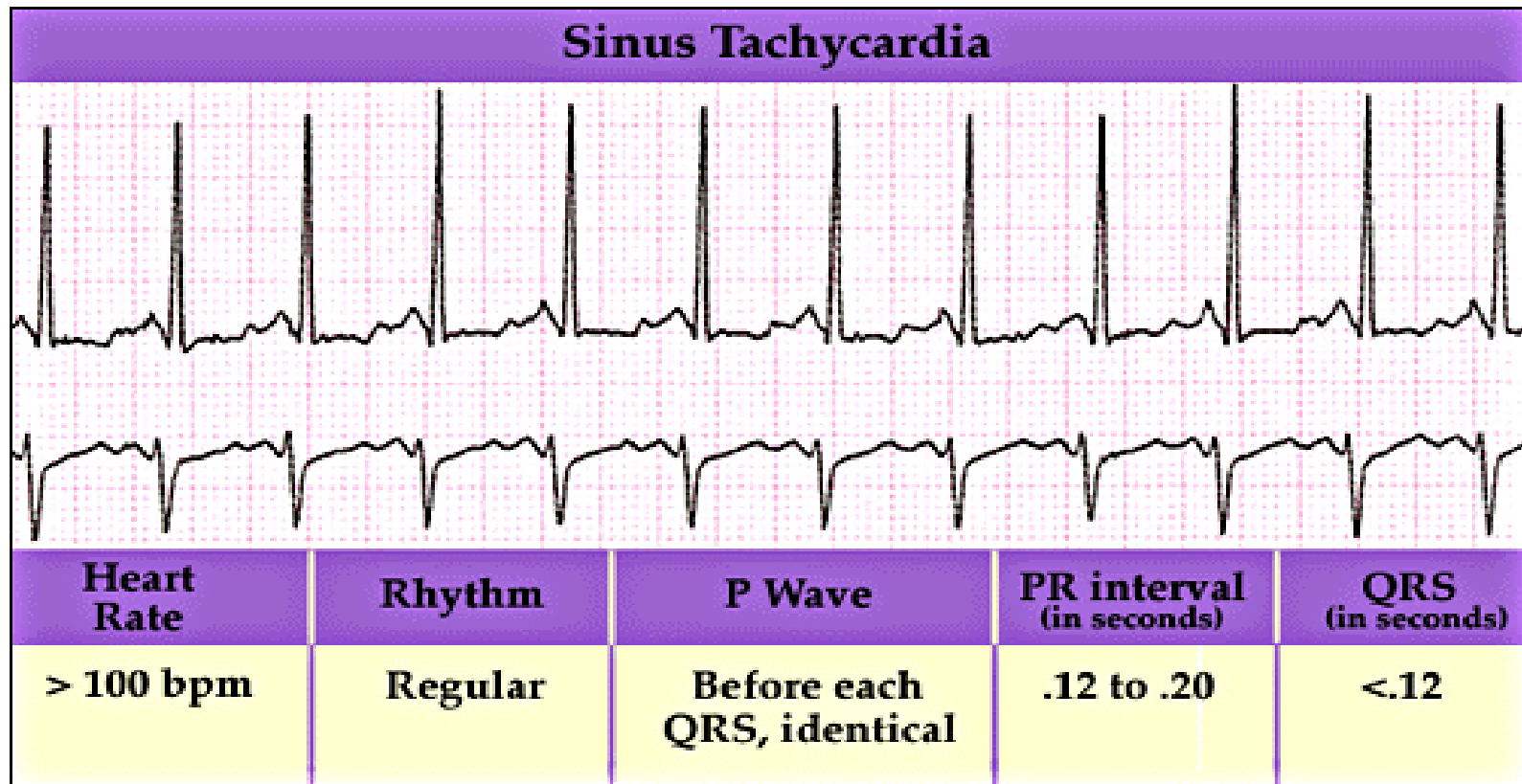
Impulses originate at S-A node at slow rate



All complexes normal, evenly spaced

Rate < 60 - 100/min

SINUS TACHYCARDIA

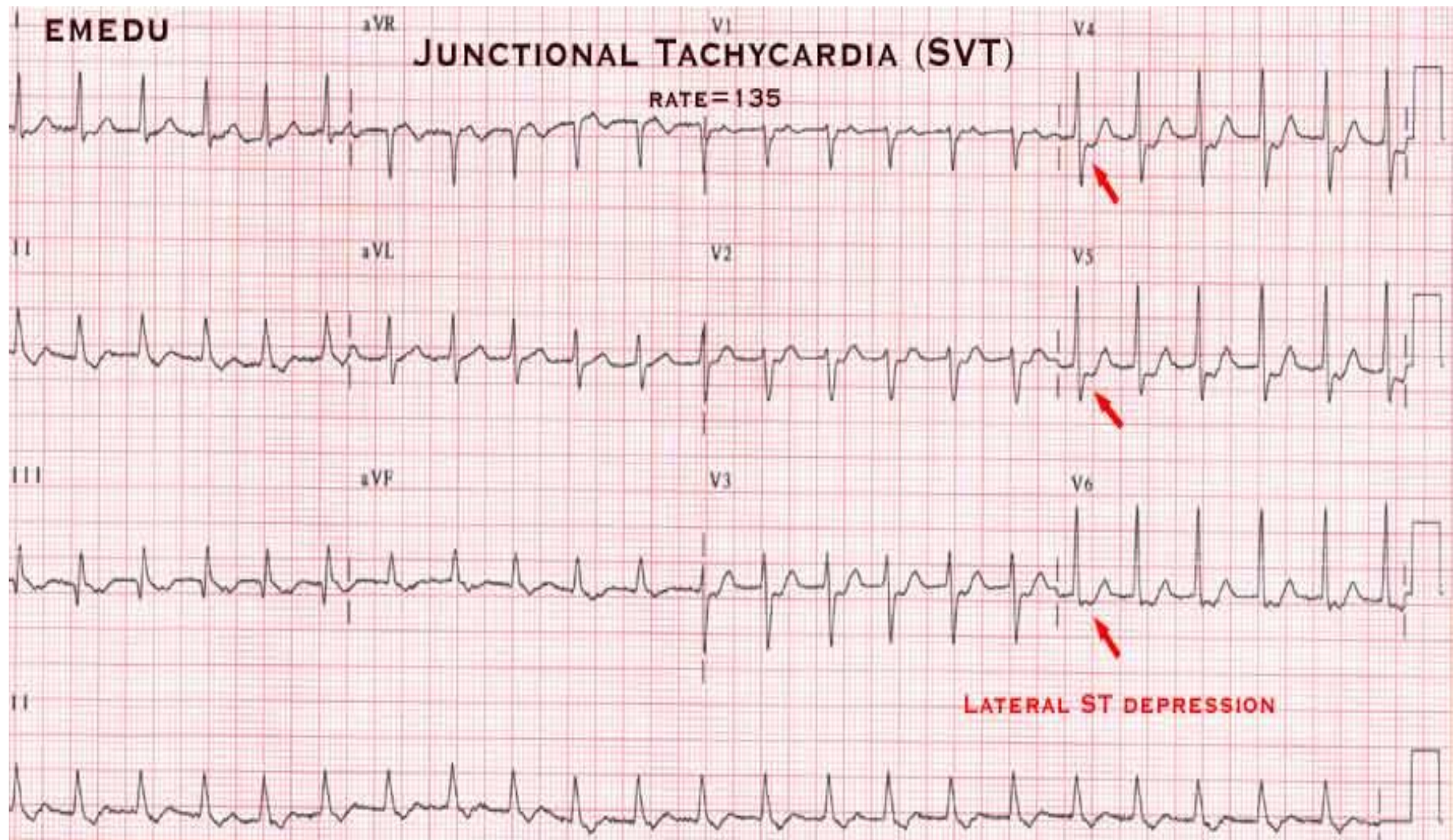


Tachyarrythmias

Supraventricular

- atrial,
- junctional
- narrow,
- wide complex(SVT with aberrancy)
- Ventricular (VT) - Wide complex

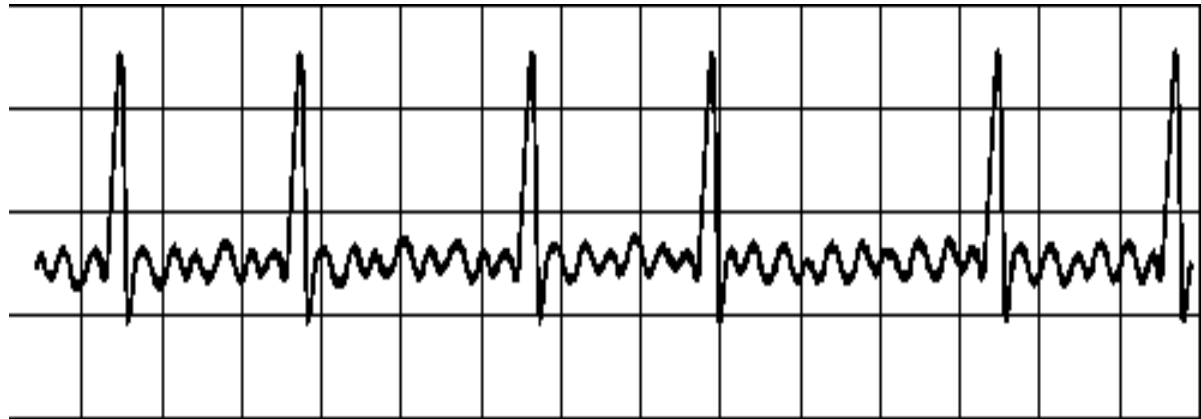
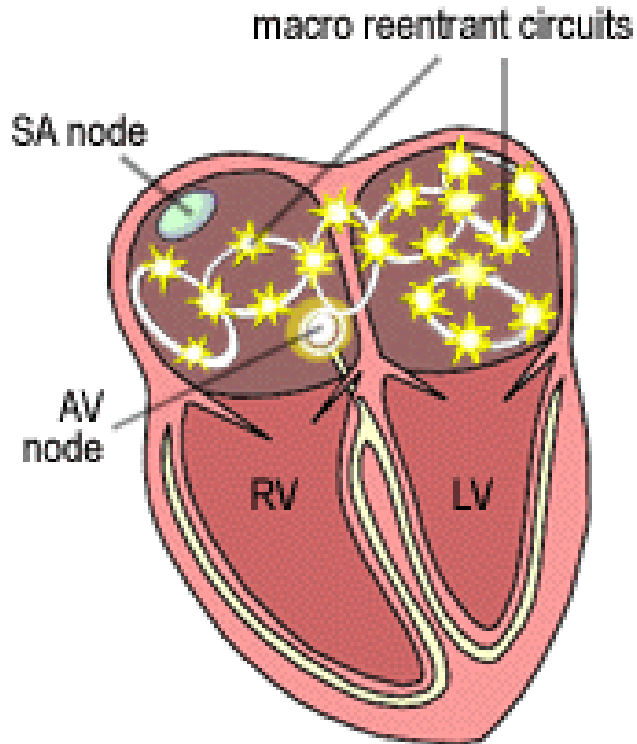
SVT



Supraventricular Tachycardia: Rate 140

ATRIAL FIBRILLATION

Impulses have chaotic, random pathways in atria



Baseline irregular, ventricular response irregular

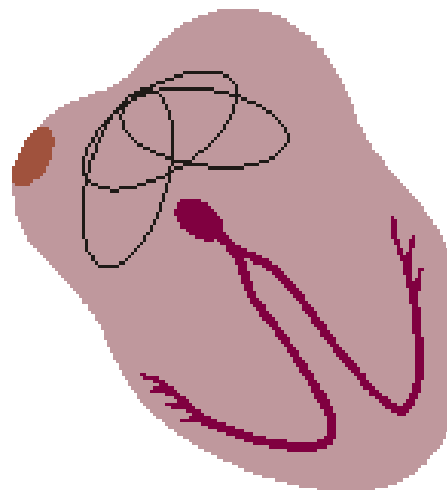
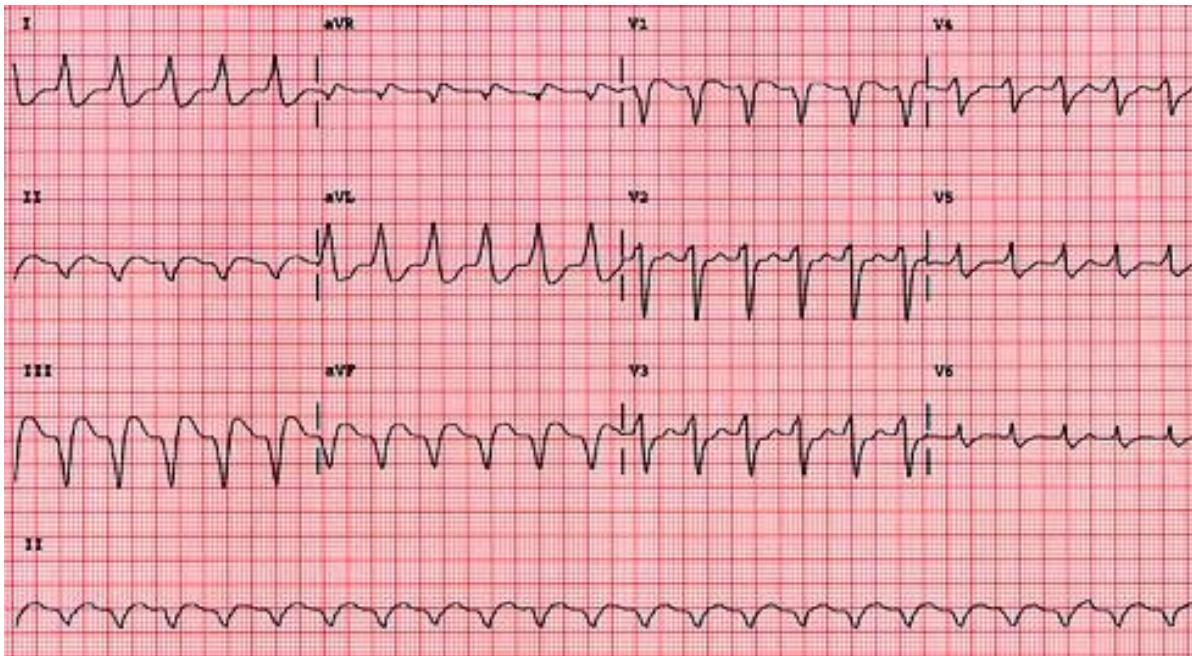
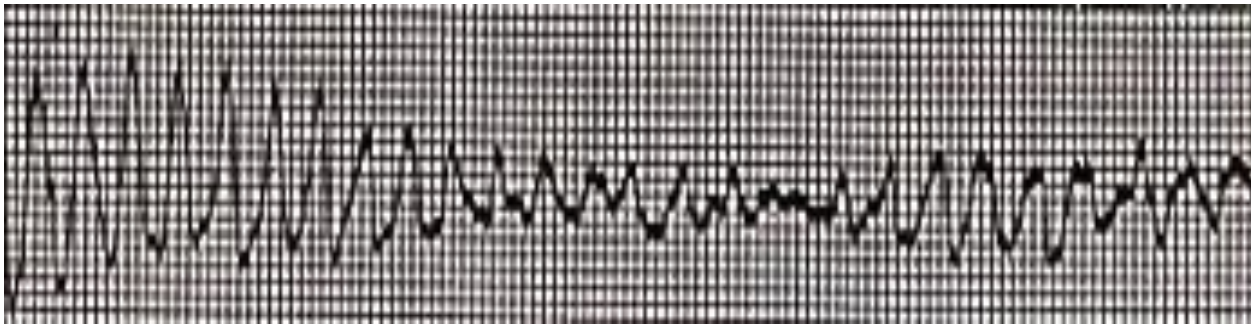


Illustration depicting macro reentrant circuit ("wavelet") activity during a run of complex atrial fibrillation (AF)



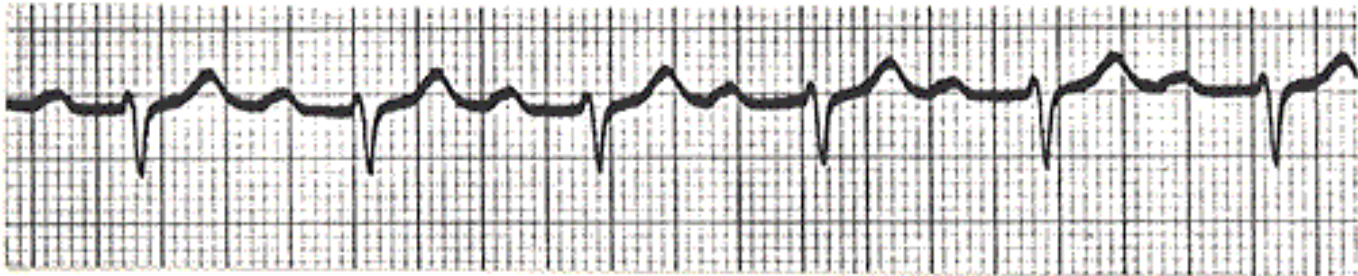
Ventricular
tachycardia:
note fast rate
and wide
bizarre QRS.



Ventricular
fibrillation:
erratic and
wavy
baseline

Long PR Interval

- First degree Heart Block



1st degree AV block (PR = 280 ms)

Group 4 Atrioventricular heart blocks

1st degree

PR interval > 0.2 seconds (1 large box), each P is followed by a QRS. PR interval is measured from the beginning of the P wave to the beginning of the QRS



The PR interval is approximately 0.28 seconds.

2nd degree block - type 1

Also called "Wenkebach". PR interval gets progressively longer each beat until finally a QRS is "dropped" (missing).

Note the increasing PR interval before the QRS is dropped, then the cycle is repeated



2nd degree block - type 2

Also called "Mobitz II". A more serious conduction problem than Type 1. PR intervals are constant and a QRS is "dropped" intermittently.



Note the dropped QRS after the second and sixth P wave

3rd degree block

The atrial rate is independent of the ventricular rate (P wave and QRS march out separately. The clue here is no relationship at all of the P-R intervals). The P-R interval is constantly changing, the QRS is usually wide and bizarre because it is ventricular origin.

P waves and QRS waves are independent of each other



Bundle Branch Blocks

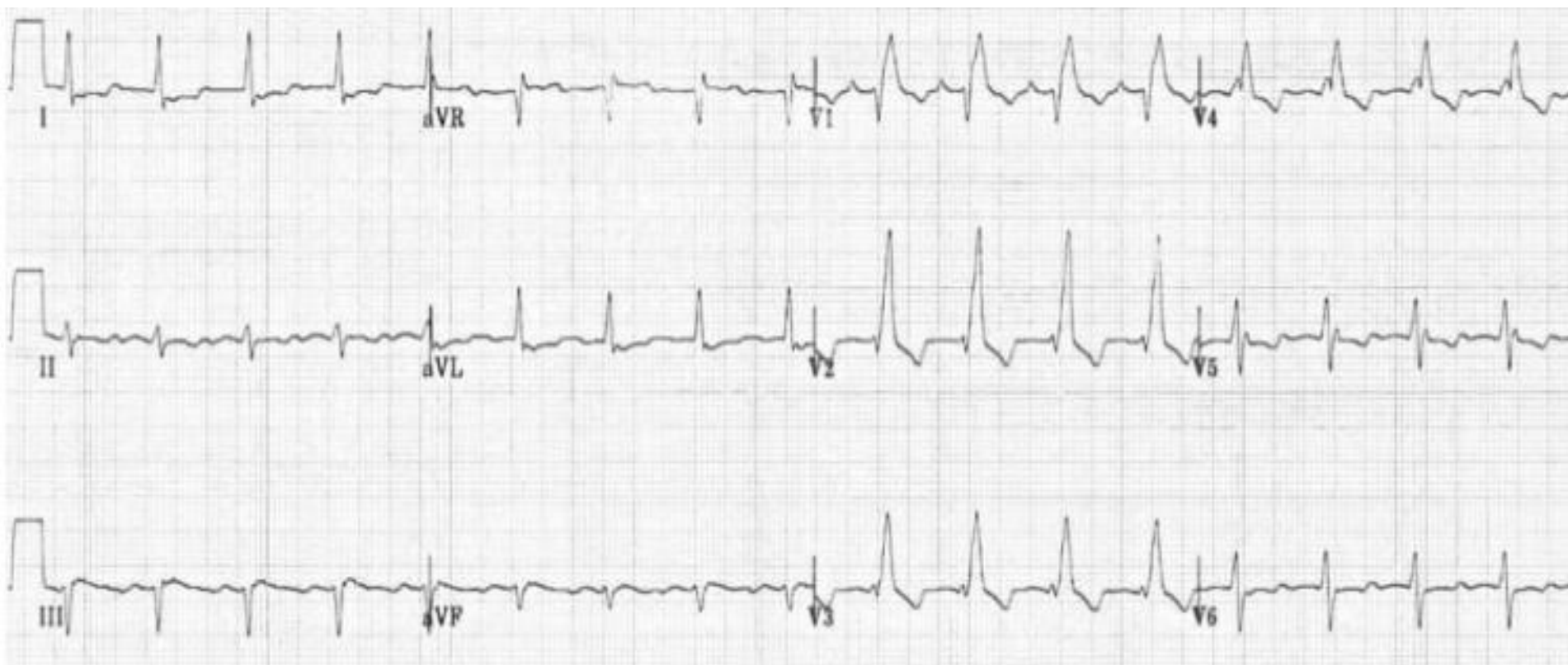
Intrinsic impairment of conduction in either the right or left bundle system leads to prolongation of the QRS interval

With complete Bundle branch blocks the duration of QRS becomes > 120 ms in duration.

RBBB results in rSR' in V1 and qRS in V6 and the QRS vector is oriented towards the right

RBBB

Congenital heart disease ASD
Acquired Valvular and ischemic



Bundle branch blocks

LBBB QRS vector is directed towards the left.

Wide predominantly negative complexes
QS in V1

Large positive R complexes in V6

LBBB

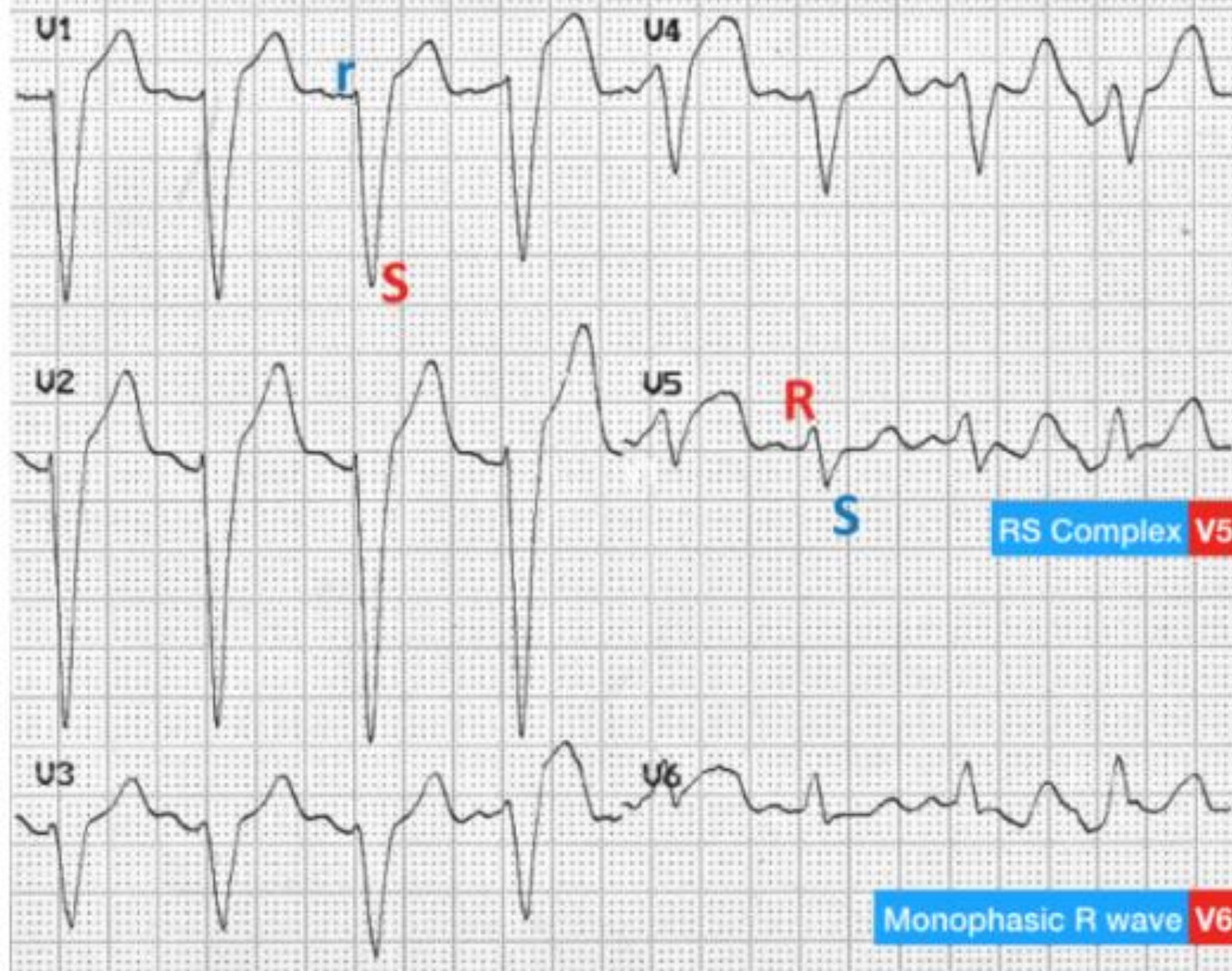
Coronary heart disease

Hypertensive heart disease

Aortic valve disease

Cardiomyopathy

V1 rS Complex



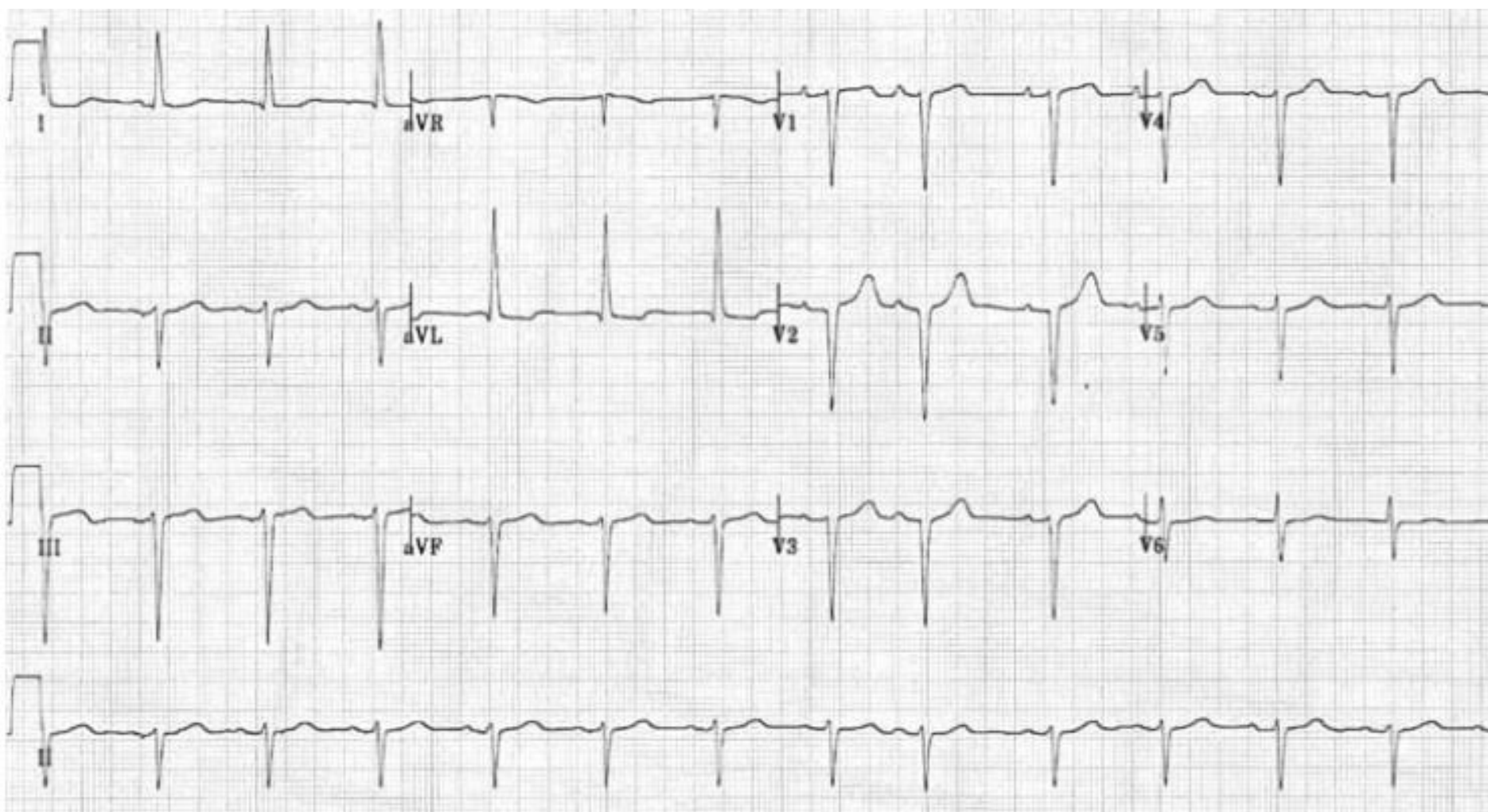
Fascicular blocks

Left anterior fascicular block

Left posterior fascicular block

LAHB is associated with extreme left axis deviation without widening of QRS complex

Isolated LPFB is very rare



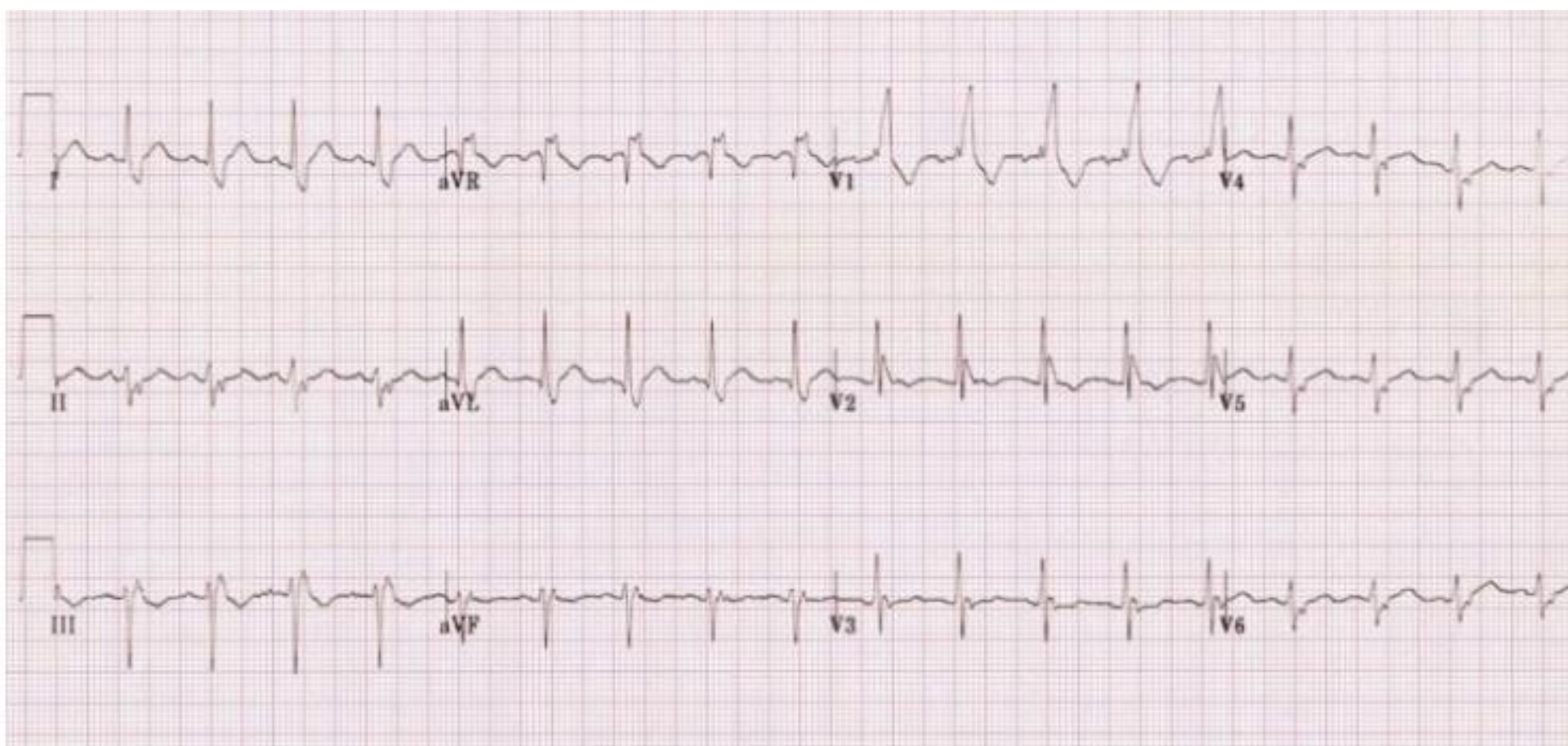
Other blocks

Bifascicular block:

Complete LBBB

RBBB with LAHB

RBBB with LPHB



Ischemia

Findings depend on several factors:

Nature of the process

Duration

Localisation

Other abnormalities

Acute ischemia in myocardium

Acute ischemia lowers the resting membrane potential and shortens the duration of the action potential

Such changes cause a voltage gradient between normal and ischemic zones

When acute ischemia is transmural ST vector shift in direction of epicardium causing ST elevations and sometime Tall T waves called hyperacute T waves

Acute ischemia

When ischemia is confined to the subendocardium St vector shifts to the ventricular cavity leading to St depression

Profound ST changes indicate severe ischemia

Infarct

Division of acute myocardial infarction into ST segment elevation and non- ST elevation types is useful since the efficacy of the acute reperfusion therapy is limited to the ST elevation group

Acute transmural anterior wall ischemia :
ST elevation or hyperacute T waves in
V1-V6 and I, aVL

Inferior wall ischemia : Changes in II, III,
aVF

Infarct

Accurate ECG interpretation in a patient with chest pain is critical. Basically, there can be three types of problems –

- *ischemia* is a relative lack of blood supply (not yet an infarct),
- *injury* is acute damage occurring right now, and finally,
- *infarct* is an area of dead myocardium.

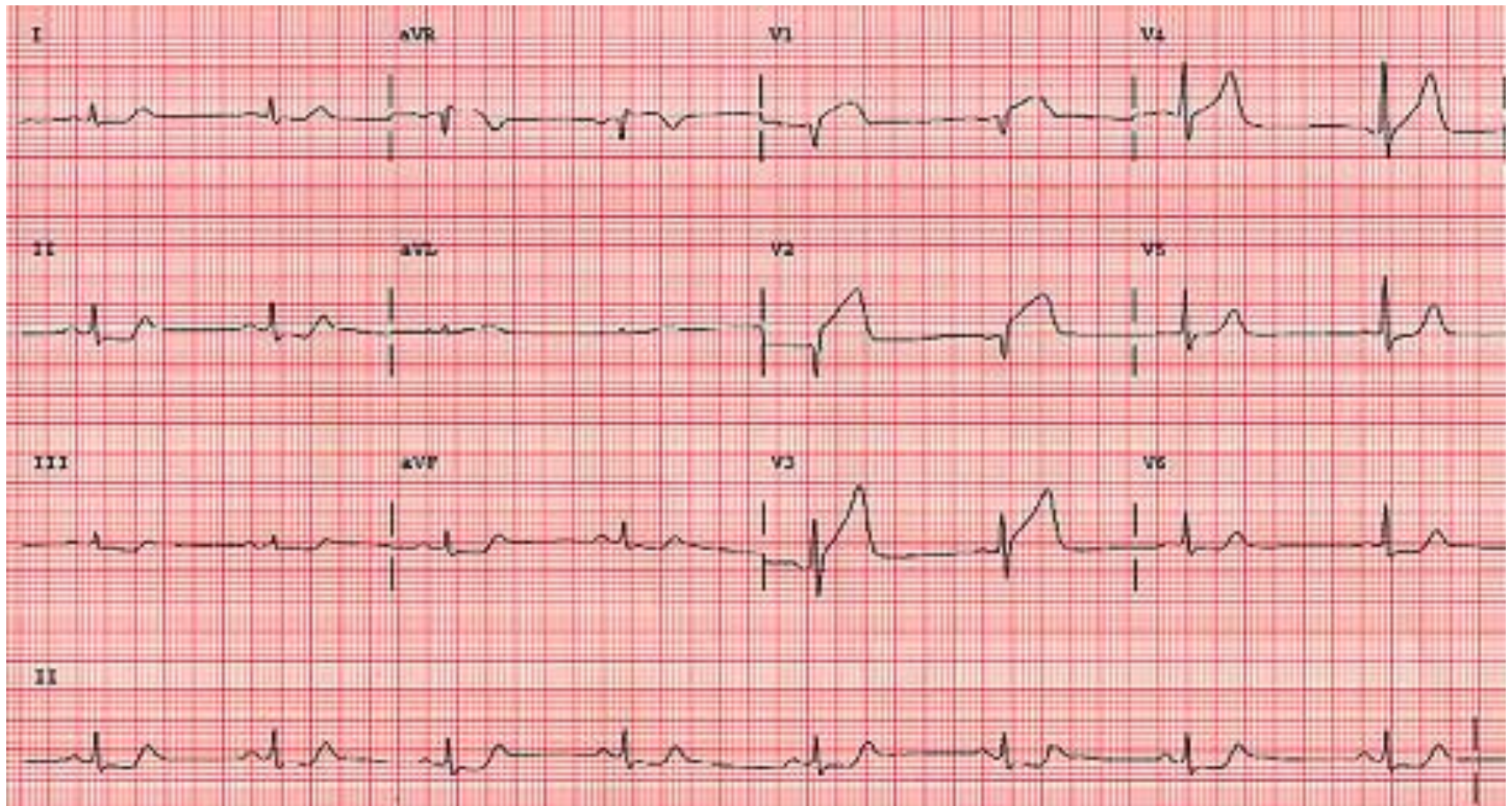
It is important to realize that certain leads represent certain areas of the left ventricle; by noting which leads are involved, you can localize the process.

The prognosis often varies depending on which area of the left ventricle is involved (i.e. anterior wall myocardial infarct generally has a worse prognosis than an inferior wall infarct).

Infarct

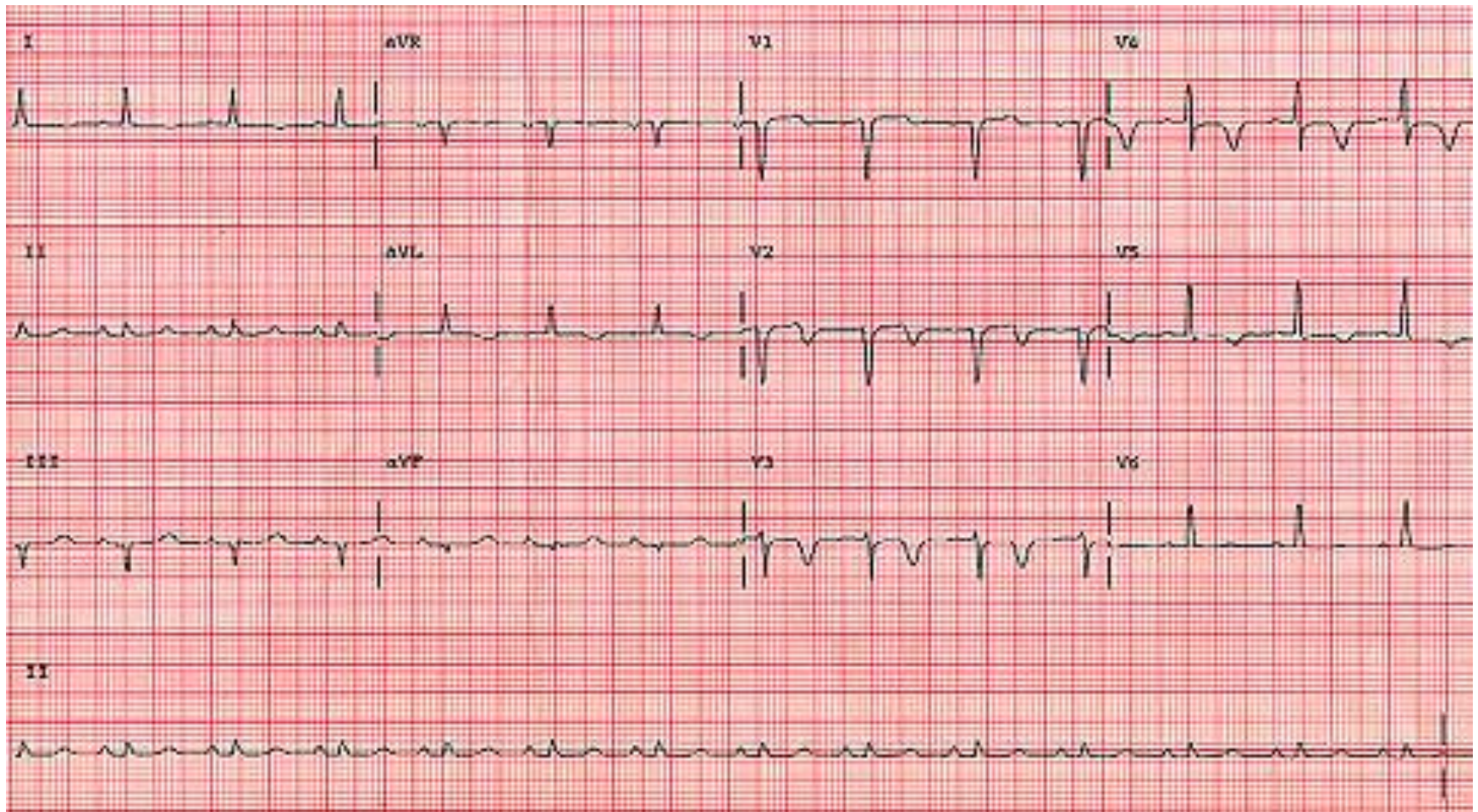
Ischemic ST elevations are followed by hours to days later deep T inversions or the development of Q waves in the same lead distribution

Patients with chest pain who have deep T inversions in multiple precordial leads usually have severe obstruction in the left anterior descending coronary artery system

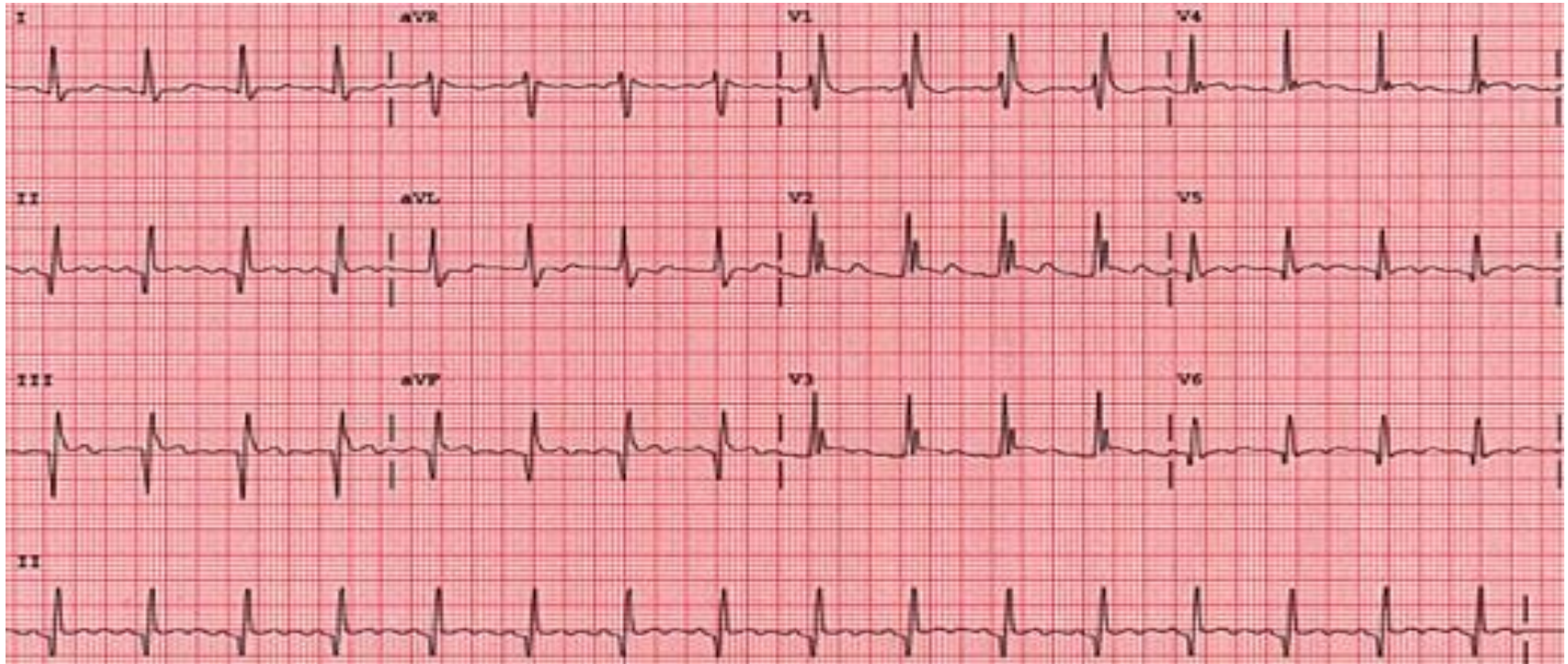


Injury: Note ST segment elevation in leads V2-V3
(anteroseptal/anterior wall)

1. Ischemia	Represented by symmetrical T wave inversion (upside down). The definitive leads for ischemia are: I, II, V2 - V6.
2. Injury	Acute damage - look for elevated ST segments . (Pericarditis and cardiac aneurysm can also cause ST elevation; remember to correlate it with the patient.
3. Infarct	Look for significant "pathologic" Q waves . To be significant, a Q wave must be at least one small box wide or one-third the entire QRS height. Remember, to be a Q wave, the initial deflection must be down; even a tiny initial upward deflection makes the apparent Q wave an R wave.



Ischemia: Note symmetric T wave inversions in leads I, V2-V5.

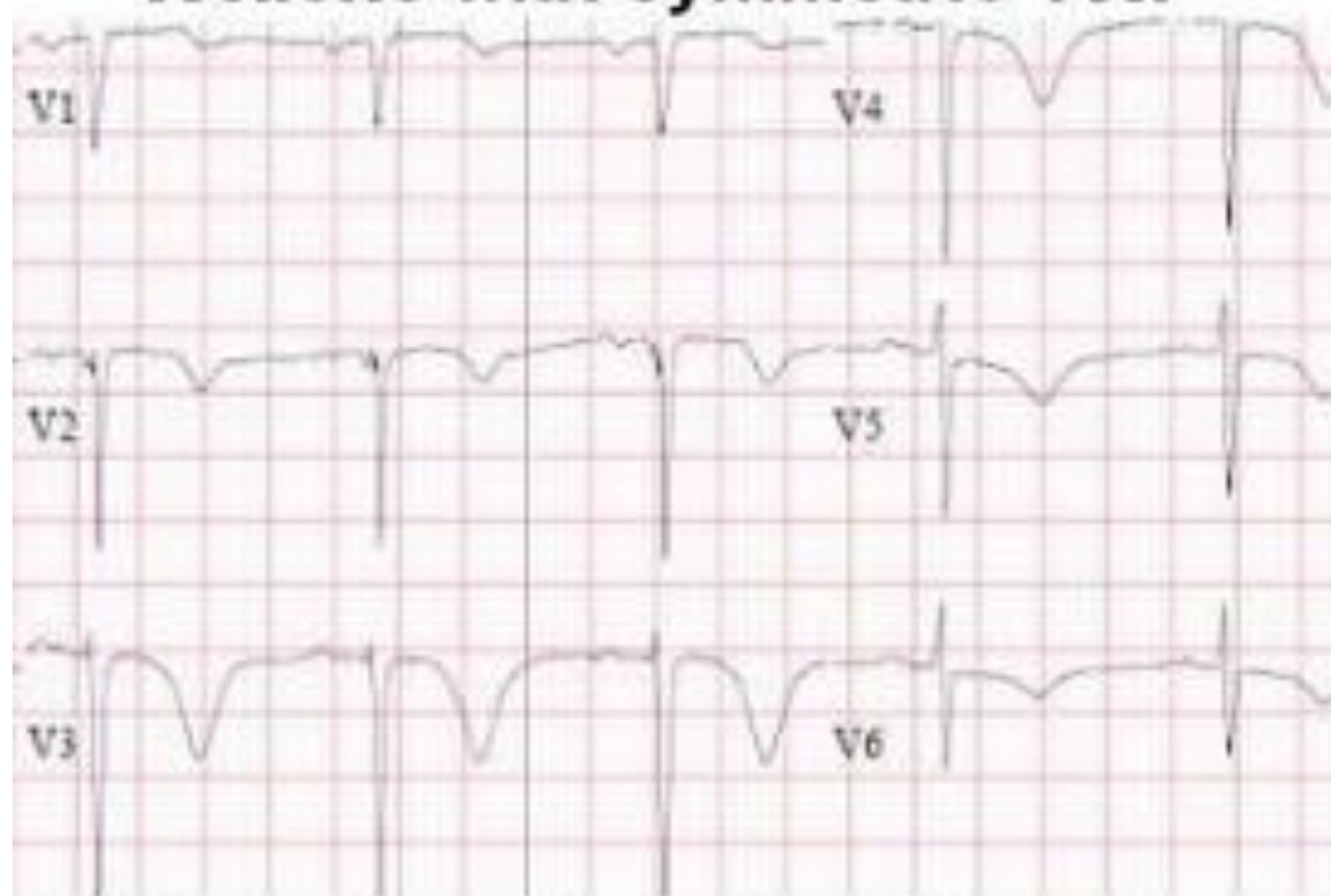


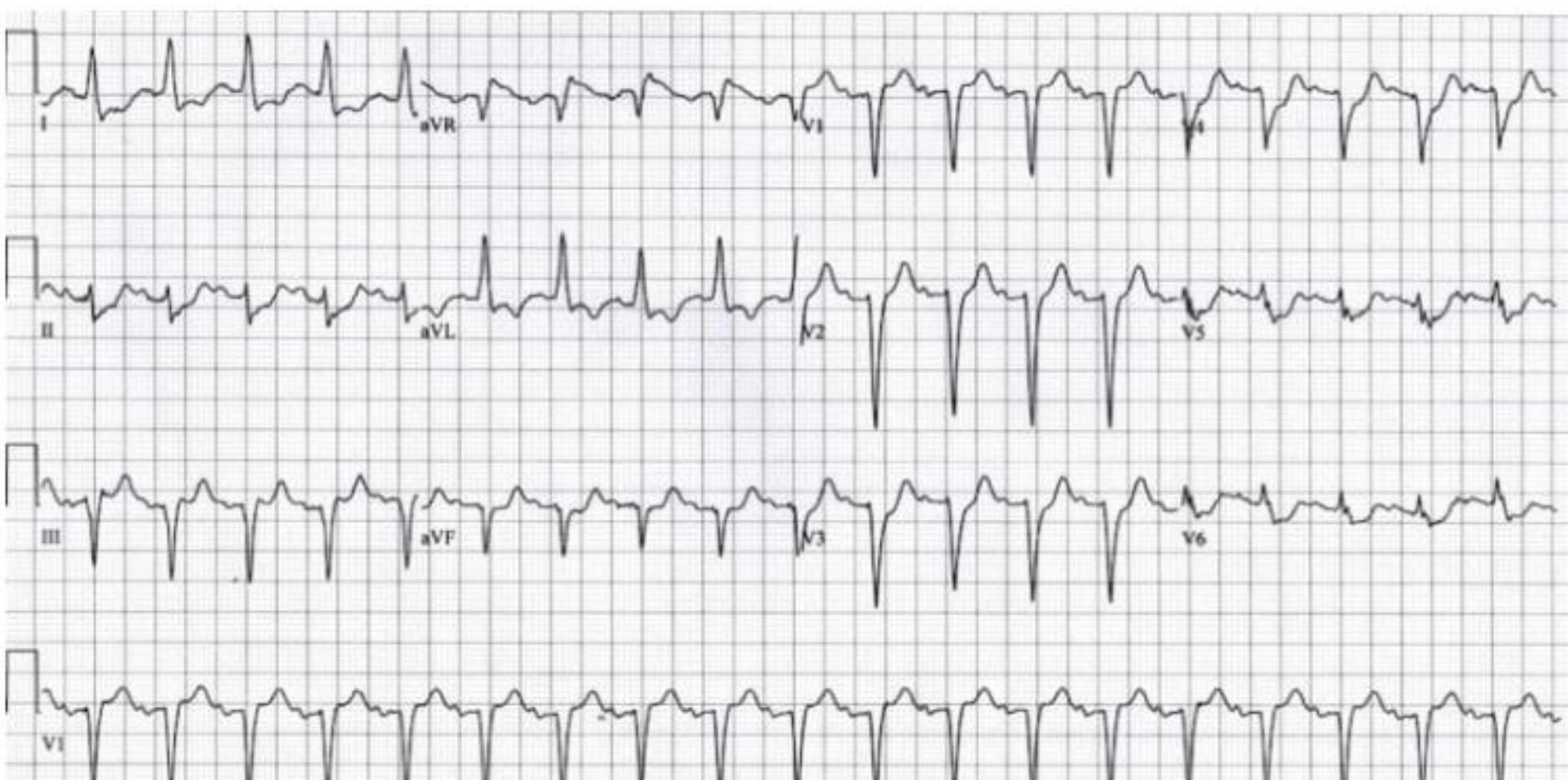
Infarct: Note Q waves in leads II, III, and aVF (inferior wall).

Normally the R wave gets larger as you go to V1 to V6. If there is no R wave "progression" from V1 to V6 this can mean an infarct.

The second caveat is that, with a left bundle branch block, you cannot evaluate "infarct" on that ECG. In a patient with chest pain and left bundle branch block, you must rely on cardiac enzymes (blood tests) and the history.

Wellens with symmetric TWI





THANK YOU