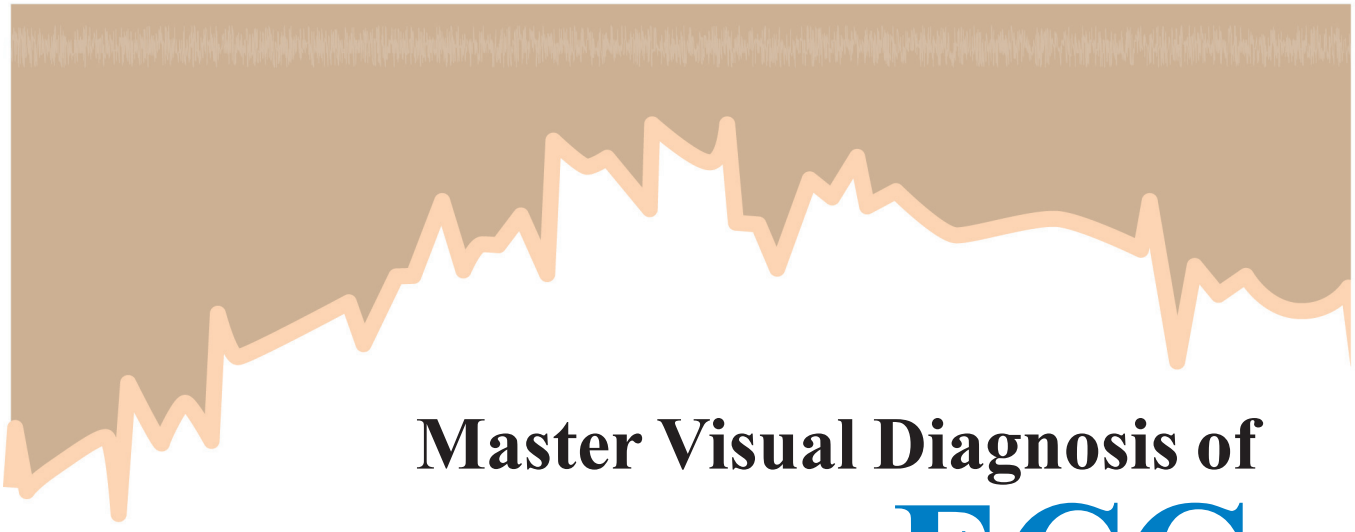
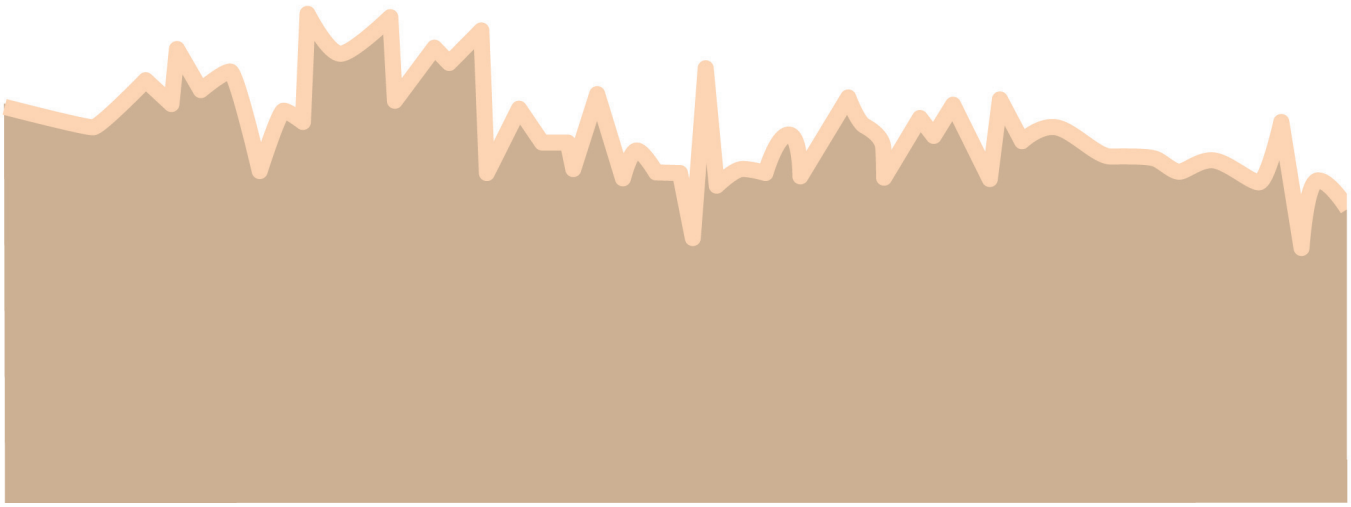


Master Visual Diagnosis of ECG

A Short Atlas



Master Visual Diagnosis of ECG

A Short Atlas (Learn ECG Through ECG)

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Dedicated to

Dr Sahibzada Tasleem Rasool,
Assistant Professor, King Faisal University, Al-Ahsa,
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for his support and valuable guidance in
our studies and in writing of this book.

Preface

This book is written with the intention to present main ECG diagnoses in a very easy, quick and retainable manner. There are many books available on this topic. So what is the reason for writing a new book? Answer is two-fold! Firstly, we have noticed that undergraduate medical students and junior residents cannot find time to go through much detailed books as they have to study other subjects and have to work in the hospital. Secondly, ECG is a visual diagnosis which needs clear "Visual" explanation in terms of ECG graphs and schematic diagrams to show normal and abnormal presentations more clearly. We guarantee the readers that they will find almost every normal and abnormal finding commonly encountered in ECG in wards with its explanation from real ECGs and useful tables and schematic diagrams, while the text stressing more on the diagnostic points so the readers can readily understand characteristic features of conditions and memorize it visually. In order to present ECG diagnoses in a more real-looking situation and to encourage the readers to hunt for abnormalities, we tried to avoid marking the abnormalities with arrows, circles or asterisk as much as possible. This creates an ECG Hunting Reflex in the readers and Look-Note-Diagnose approach instead of Read-Memorize-Diagnose approach seen in other books. In fact, this book may be regarded as a mini atlas for basic ECG diagnosis.

Other feature of this book is that it also presents logical explanation of different ECG findings that why specific conditions present with specific ECG appearance. For example, in right bundle branch block why lead V_1 present with specific morphology. Another feature which the readers will find much helpful is the axis description. It is presented in a very comprehensive and interesting way which will remove fear of the readers for cardiac axis (In fact, we have heard from many of our friends and colleagues that they hesitate from ECG mainly because of the difficulty in understanding and determination of cardiac axis).

The book consists of two sections, the first deals with basic concepts [Deep Analysis Section (DAS)] which makes the readers to understand how normal and abnormal ECG and components of ECG including waves, segments and intervals present, while the second [Quick Diagnosis Section (QDS)] section deals with how to diagnose specific appearance in ECG.

The other benefit of the above-mentioned twosections is that after clarification of basic concepts of ECG, the readers do not need to repeatedly consult the main text while they encounter with ECGs in the wards or during revision for examinations and they can just pay attention to diagnoses.

We thank Dr Wang Wei Na from Department of ECG in Zhongnan Hospital for providing us valuable ECGs. Our special thanks to our Chinese friends Su Yu Tong, Xia Xi Ya, Zhang Wei and Zhang Xiang Yu for providing us much help in necessary translations from Chinese to English. We also thank all writers and publishers from where we have got help. Special thanks to our junior undergraduate fellow Adnan Aslam who profoundly helped us in typing, editing and in index making of the book. We also like to thank Junaid and Umair (Wuhan University Medical College). Finally, we thank Dr Sahibzada Tasleem Rasool (Assistant Professor, King Faisal University, Al-Ahsa, Saudi Arabia) for his valuable guidance.

We hope this book will fulfill the requirements of readers. We welcome every suggestion and correction to improve the book in the next edition.

Shahzad Khan
Ren Jiang Hua

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- www.ecglibrary.com by Dean Jenkins and Stephen Gerred
- ECGpedia, Wiki ECG Course
- Ashman Phenomenon, Ram C Sharma, USA
- Arrhythmia Recognition by Tomas B Garcia, Geoffrey T Miller
- wikipedia.org
- The Only EKG Book you will Ever Need by Malcolm S Thaler

- Journal(s) of the American College of Cardiology (JACC)
- Electrocardiographic Case: A Middle Aged, Seriously Ill Woman with an Unusual ECG and Wide Complex Tachycardia, P Shah, WS Teo, SMJ (Singapore Medical Journal)
- Electrocardiography of Clinical Arrhythmias by Charles Fisch, Suzanne B Knoebel
- Atrioventricular Nodal Reentry Tachycardia (AVNRT): Brian, Chirag M Sandesara
- ECG-SAP III: Electrocardiography Self-Assessment Program
- Electrocardiography: 100 Diagnostic Criteria by Harold L Brooks
- How to Quickly and Accurately Master Arrhythmia Interpretation by Dale Davis
- ECG Pocket Guide by Bradford C Lipman and Bernard S Lipman
- Advanced ECG: Board and Beyond by Brendan P Phibbs
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Some Abbreviations Used in this Book

AF	Atrial fibrillation
AF	Atrial flutter
AMI	Acute myocardial infarction
APC	Atrial premature contraction
ASD	Atrial septal defect
AV	Atrioventricular
AVNRT	Atrioventricular re-entry tachycardia
AVRT	Atrioventricular reciprocating tachycardia
AMI	Anterior myocardial infarction
BPM	Beats per minute
COPD	Chronic obstructive pulmonary disease
CV	Cardiovascular
CVA	Cerebrovascular accident
ECG	Electrocardiogram
EKG	Electrocardiogram
JPC	Junctional premature contraction
LA	Left atrium
LAD	Left anterior descending or Left axis deviation
LAFB	Left anterior fascicular block
LBBB	Left bundle branch block
LCA	Left coronary artery
LCX	Left circumflex

LPFB	Left posterior fascicular block
LV	Left ventricle
LVH	Left ventricular hypertrophy
MAT	Multifocal atrial tachycardia
NSR	Normal sinus rhythm
PAT	Paroxysmal supraventricular tachycardia
PE	Pulmonary embolism
PSVT	Paroxysmal supraventricular tachycardia
RA	Right atrium
RBBB	Right bundle branch block
RCA	Right coronary artery
RV	Right ventricle
RVH	Right ventricular hypertrophy
SA	Sinoatrial
STEMI	ST elevation myocardial infarction
SVT	Supraventricular tachycardia
VF	Ventricular fibrillation
VPC	Ventricular premature complex
VSD	Ventricular septal defect
VT	Ventricular tachycardia
WPW syndrome	Wolff-Parkinson-White syndrome

Section 1: Deep Analysis Section (DAS)

WHAT CONCEPT DO YOU NEED TO HAVE FOR BETTER UNDERSTANDING OF ECG?

A simple ECG strip provides more than 100 diagnoses itself alone. The detailed physiology of electrical activity in the heart resulting in 12-lead ECG usually presented in the other ECG books makes students think ECG is very difficult. Therefore, we highlight here the very core concepts which are easy to understand and will help students to LOOK-NOTE-DIAGNOSE rather than READ-MEMORIZE-DIAGNOSE.

What is ECG?

Electrocardiography is the final outcome of physiological and technological processes which start from the current generator in the heart till you interpret.

Sequence showing (Fig. 1.1) how ECG generates. In fact, any factor on the right or left can change the ECG morphology and its interpretation.

Current Generation in the Heart

Normally current generates from SA node (Sinoatrial node) which is the main generator or pacemaker causes sinus rhythm. AV node (atrioventricular node) which is at the junction of atrium and ventricle, the current passes through it from atrium to ventricle with a inherent capacity of AV node to delay the passage of current from atrium to ventricle (Fig. 1.2).

Sometimes, current can generate from other ectopic areas in the:

- Atrial tissue
- Ventricle tissue
- Tissue surrounding AV node

Impulse arising from these areas causes “Extrasystole” or beats beside the normal beats. Therefore, if these ectopic areas cause any beats these are called “Premature” or extra beats and in a normally going ECG tracing, its shape gives a clue that whether it has arisen from atrium “atrial premature contraction (APC)”, Ventricle “ventricular premature contraction (VPC)”, or around AV junction “Junctional premature contraction (JPC)”.

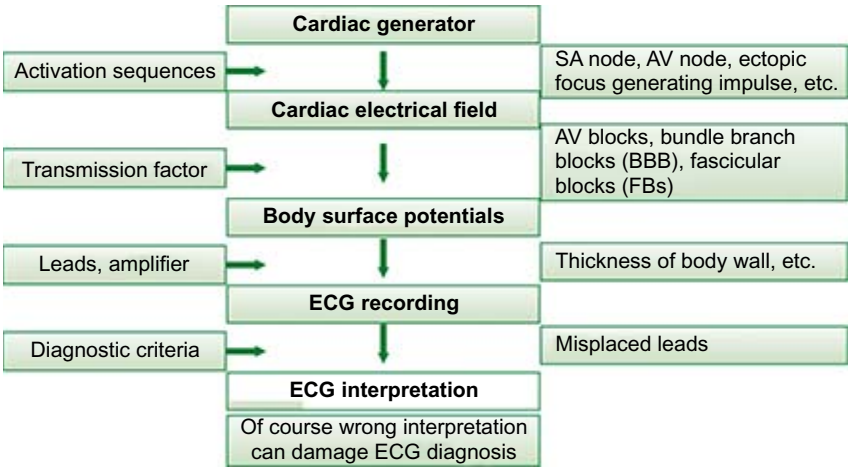


Figure 1.1: ECG from generation to interpretation

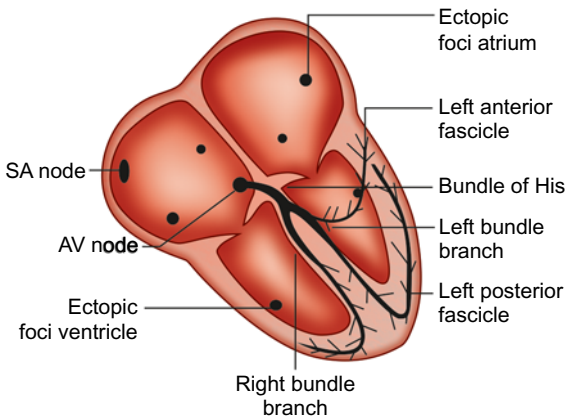


Figure 1.2: The foci which can generate current and the pathways where current flows

Note that in normal state SA node's rhythm over-rides all other tissues which can cause impulse generation. Therefore, it is the only source of currents initiation and rest of the system only transmits. That's why in normal state it is called PACEMAKER. "In conditions when SA node fails to generate current, surrounding parts (AV node, ventricular tissue and Purkinje fibers in descending order) take the place of SA node and become dominant current generator. Since their maximum rate at which they can generate current is not same examining their rate can give us the clue that which part is now pacemaker (See next section for details)".

WHAT ARE ECG LEADS?

There are different leads which can conduct the electrical activity of the heart through body walls to ECG machine, this current is then amplified appropriately to be viewable on a screen or printed on ECG strip.

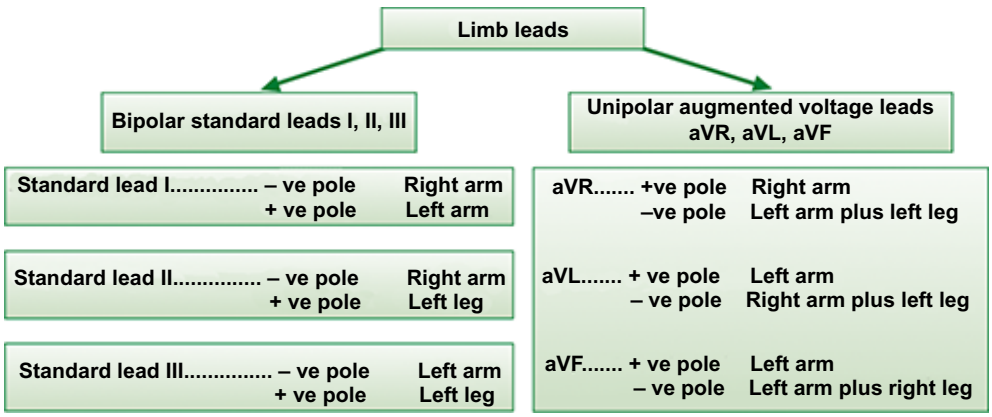
Each lead "looks" heart from a different angle, therefore their recording is different in morphology.

There are two groups of leads:

1. Limb leads
2. Chest leads

In fact, modern ECG machine increases the amplitude of these leads by 50 percent, hence called augmented voltage leads (Figs 1.3, 1.4 and Table 1.1).

Table 1.1: Limb leads arrangement



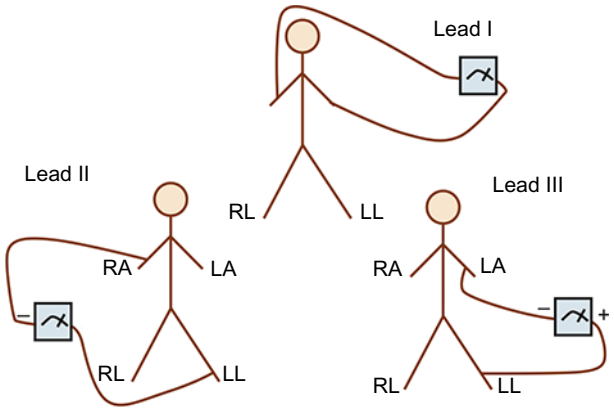


Figure 1.3: Limb leads arrangement in standard limb leads

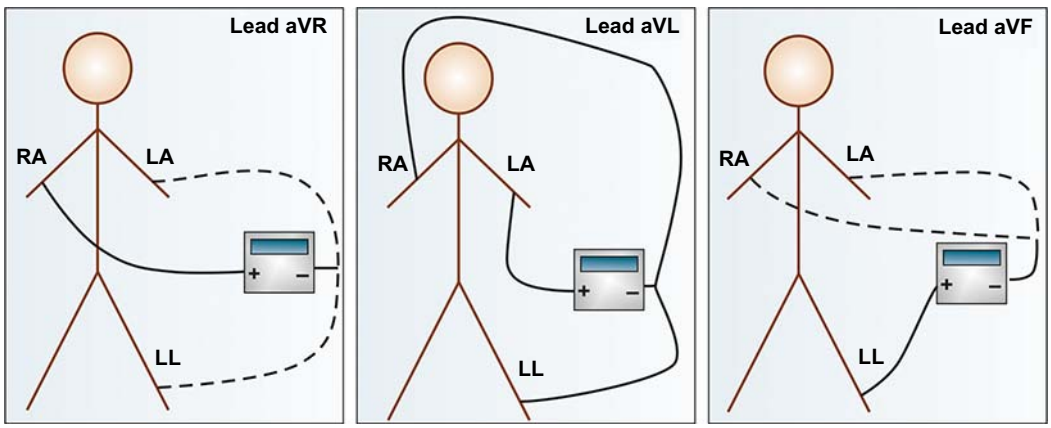
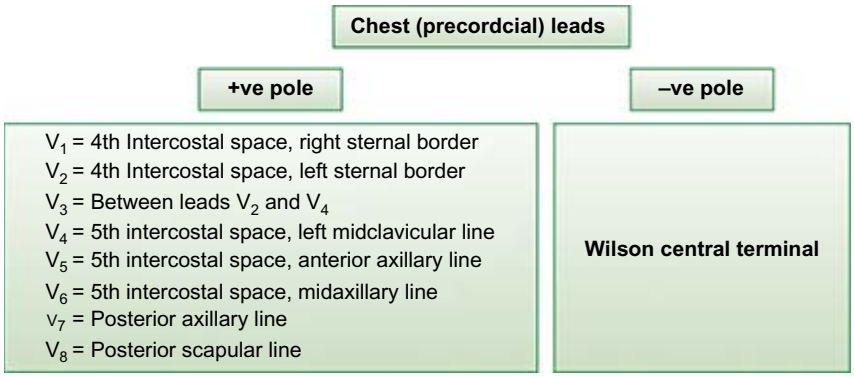


Figure 1.4: Lead arrangement in unipolar augmented voltage leads. Bold line shows the main exploring electrode connection while dotted lines show reference electrode connection which is the main output from the other two electrodes

Precordial or Chest Leads

Precordial leads placed directly on chest because of close proximity to the heart they do not require augmentation. Wilson's central terminal is used for the negative terminal and these leads consider being unipolar. They record heart's electrical activity in horizontal plane.



How Chest (Precordial) Leads Attach?

There are usually 6 chest leads and their positions are:

V_1 = 4th Intercostal space, right sternal border
V_2 = 4th Intercostal space, left sternal border
V_3 = between leads V_2 and V_4
V_4 = 5th Intercostal space, left midclavicular line
V_5 = 5th Intercostal space, anterior axillary line
V_6 = 5th Intercostal space, midaxillary line

Sometime, for better vision of posterior infarction, these leads are added

V_7 = Posterior axillary line
V_8 = Posterior scapular line

Similarly, for right ventricular infarction, it is advisable to take right sided chest leads (Fig. 1.5).

V_4R = Right sided 5th intercostal space.
V_3R = Right sided intercostal space between leads V_2 and V_4.

(*Note:* Chest leads should be placed carefully as small variation can cause different and misleading ECG recording)

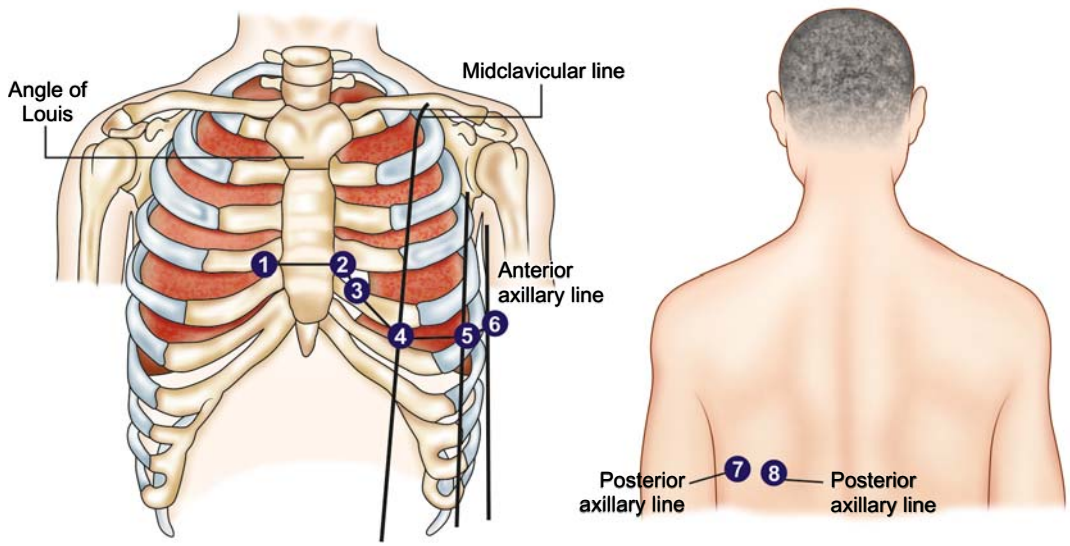


Figure 1.5: Arrangement of chest leads

Each Lead Looks Heart Differently

Standard Limb Lead

You may be confused by so many leads but each lead focuses at different angle and their viewing can be understood just by imagining the heart anatomical position and leads position (Fig. 1.6).
Lead I, aVL..... looks left lateral surface of heart.
Leads II, III, and aVF …....looks inferior surface of heart.
Lead aVR .....Looks right ventricle.

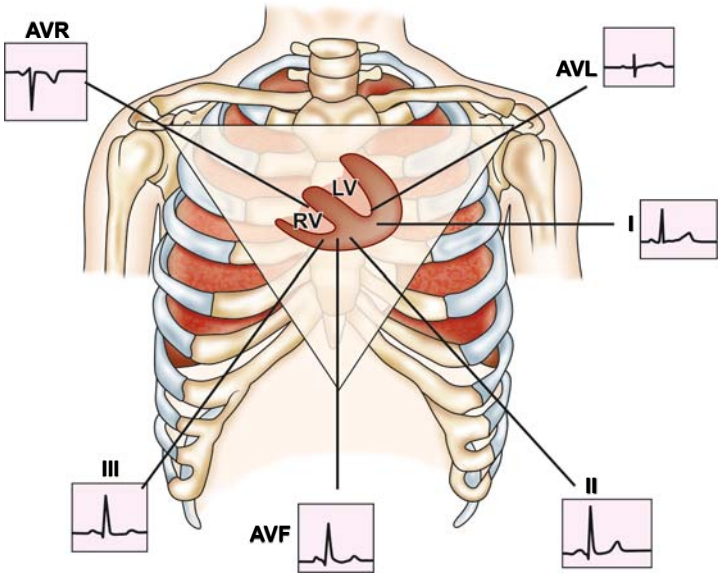


Figure 1.6: Note how different limb leads look different parts of heart and result in different morphology of QRS complex

Good to Remember

1. Lead AVR is inverted in normal conditions (Fig. 1.6).
2. Lead II and AVF are most informative while lead III is the least.

Chest Leads

As you can easily imagine chest leads view in horizontal fashion in anterior-posterior plane. Imagine heart in anatomical position within the chest cavity (Fig. 1.7).

It should be remembered that if heart is deviated from its normal position to acquire more horizontal or vertical position, the normal ECG pattern may vary without any underlying disease.

Note that limb leads look heart in the frontal plane; while chest leads see heart in horizontal plane.

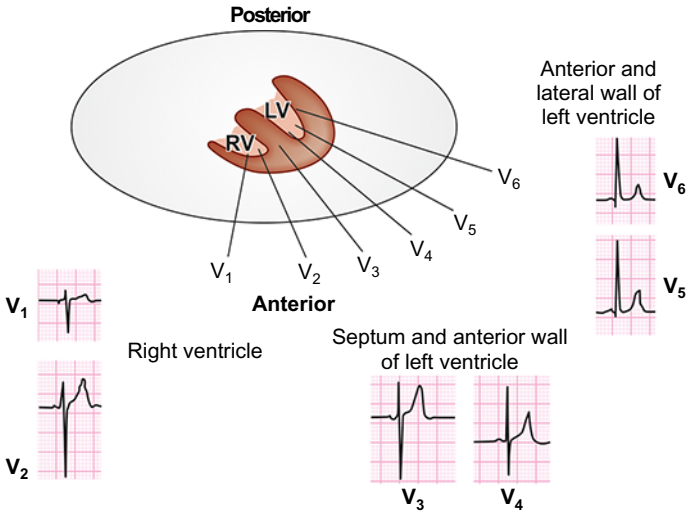


Figure 1.7: Note how chest leads look different parts of the heart and record QRS complex of different morphology

Get Familiar with ECG Strip

It is extremely necessary to know some basic details of ECG strips. These are:

1. The ECG graph paper consists of small and large squares. The length shows voltage and width shows time (in other words, X-axis is time in seconds or milliseconds and Y-axis is voltage in volts or millvolts). The length and width of small box is 1 mm.
2. One small square equals to 0.04sec. (When the speed of strip is 25 mm/sec.)
   And its length equals to 0.1 millvolts. Therefore, a large square consisting of 5 small squares equals to 0.2 seconds and 0.5 mV. And for the 1 second recording we need 5 large squares (5 large square = 1 second)
   Six seconds recordings multiplied by 10 give us one minute recordings (See Quick Diagnoses section).
   One minute record is needed when calculates irregular rates (Fig. 1.8).

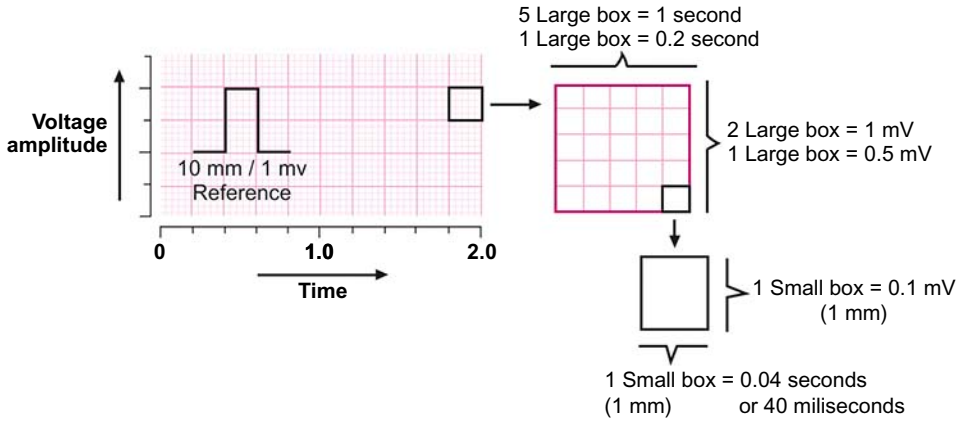


Figure 1.8: ECG paper, interpretation of large and small boxes (Speed of paper = 25 mm/sec)

HOW TO TAKE ECG RECORDING?

ECG leads set up with 6 chest leads, usually with suction grip and the limb leads are clip like. For correct recording:

- Leads must be applied to correct limbs. The colors of the leads usually are a good guide for correct position (Fig. 1.9).

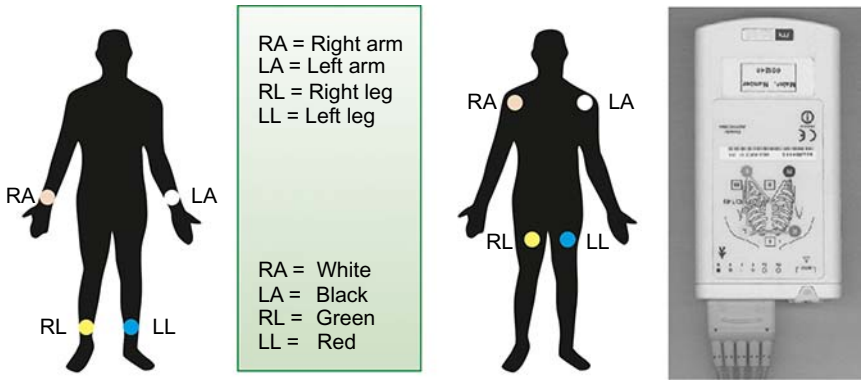


Figure 1.9: Note the limb leads and chest leads have different marks which help for correct diagnosis

Also remember that:

- For limb fleshy area better to provide more contact (little above ankles)
- In female, lower border of breast usually lies on 5th interspace.
- Thick hairy chest may be shaved (only required parts).
- For better grip gel may be used (simple normal saline can do, also)
- Make sure calibration with 1 mV.
- Patient should lie down, shouldn't move and relax in order to prevent artifacts.
- Three or four complexes are enough from each leads.

WHAT CONCEPT YOU NEED TO KNOW TO GET FAMILIAR WITH PQRS COMPLEX?

Detailed generation process of QRS complex and vector physiology can be found in textbooks of physiology and cardiology. However, it is wise to always remember these facts:

1. Current flow in this direction: SA node → AV node → Bundle of His → Left bundle branch (which again divides into anterior fascicular branch and posterior fascicular branch as left ventricle muscle mass is much more than right ventricle) → Purkinje fibers.
2. The current (generated by action potential) when travels toward the positive lead or electrode it causes upward positive deflection in QRS complex.
3. When current going away from positive electrode, it causes negative or downward QRS deflection in QRS complex.
4. Sometimes, QRS complex is isoelectric, i.e. equally or almost equal in positive and negative direction. Here the wave of current is at right angle to this lead, i.e. Perpendicular to it (Fig. 1.10).

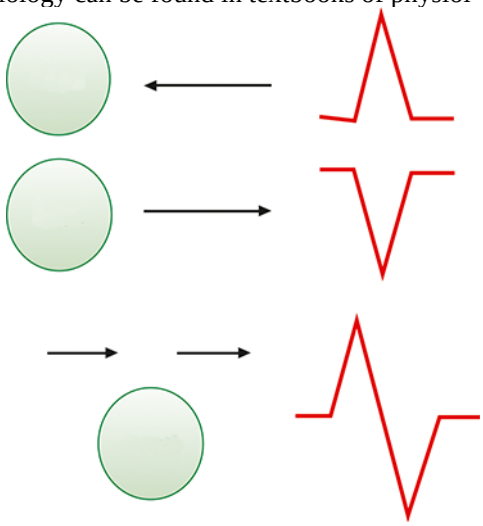


Figure 1.10: Note the direction of current with respect to electrode determines positive or negative polarity of QRS complex

What is Axis?

This will be dealt while discussing Bundle Branch Block later in this book in detail.

The PQRS Complex

Einthoven who discovered ECG formation labeled this as PQRS. It should be, however, noted that if the deflection is small, use of lower case is also common, i.e. instead of R for small R wave use r (qrs- vice versa).

Note: QRS complex coincides with electrical and mechanical activity of heart. In fact, P wave, QRS complex and T wave coincides with different phases of action potential, as described in following text (Fig. 1.11).

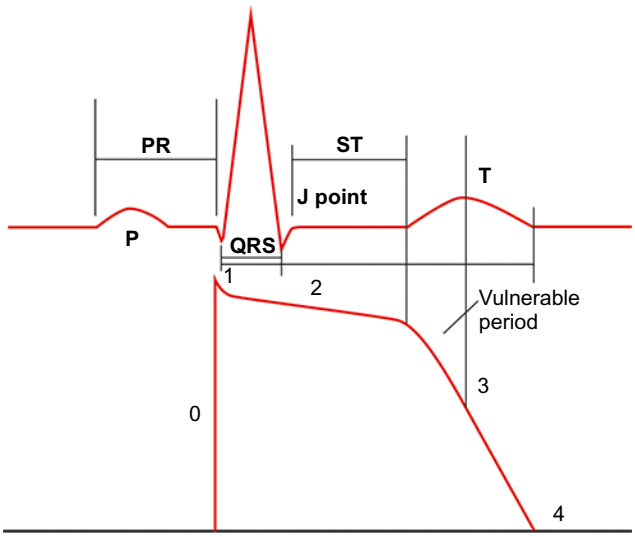


Figure 1.11: Note relationship of cardiac action potential and QRS complex of ECG. Different periods of QRS complex coincide with different phases of action potential

Characteristics of Normal P Wave

- Width less than 3 small squares (0.12 second) and height <2.5 mm.
- Represent atrial depolarization. Initial portion represents right atrium and last portion indicates left atrium activity.
- Normal P wave should be upward in leads I, II and inverted in lead aVR. This is called as sinus P wave. Presence of sinus P wave means impulse generating from SA node and rhythm is regarded as sinus rhythm. It indicates that impulse is generating from sinus node. P wave after the QRS complex (retrograde P wave) or inverted P wave indicates its origin from sources other than sinus node and it is called as ectopic P wave.
- Best seen in leads II and V1.

How Abnormal P Wave can Present? (Fig. 1.12)

Abnormal P wave may be:

1. Peaked (taller than 2.5 mm)
2. Blobbed or notched
3. Biphasic (with one half positive and other half negative)
4. Inverted
5. Buried in QRS complex
6. Retrograde (i.e. after QRS complex)

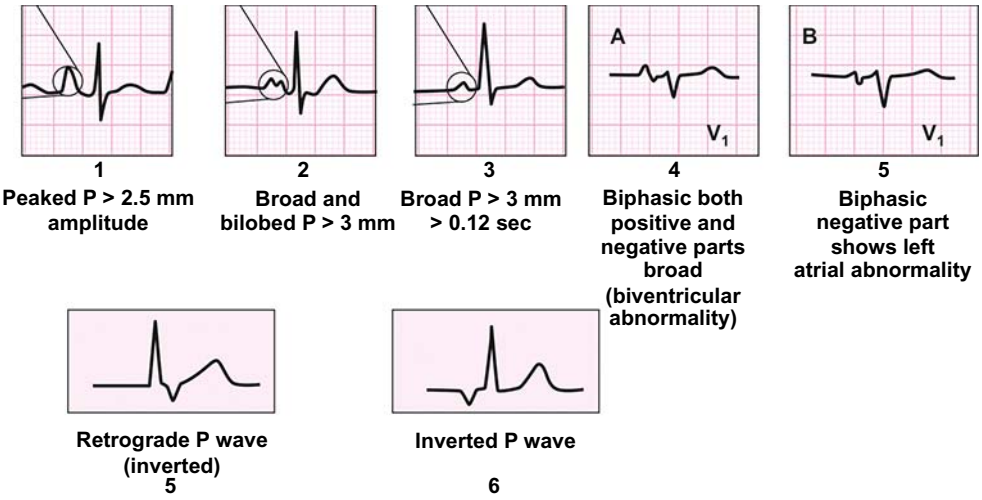


Figure 1.12: Abnormal presentations of P waves

What is P Terminal Force?

In biphasic P wave for more accuracy, the negative half is defined in terms of P- terminal force.

P terminal force = Depth of negative half in mm × Duration of negative half in seconds (Fig. 1.13).

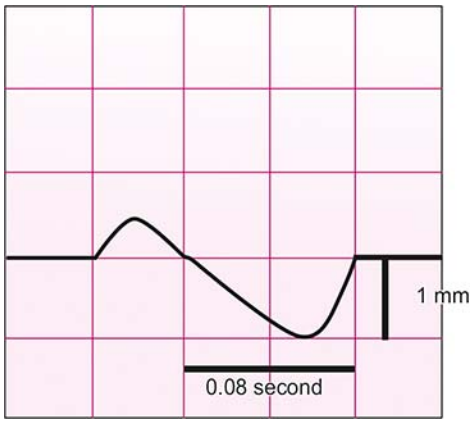


Figure 1.13: P terminal force 1 mm × 0.08 sec. = 0.08 mm.sec

This is important in diagnosing left atrial hypertrophy or abnormality.

Get Familiar with Q Wave

1. Any deflection downward after P wave is a Q wave. So you should check carefully (Note P and R waves may be negative or downward but Q and S always downward!)
2. Presence of Q wave may be normal in lead aVR, III and V_6, e.g. small q wave in lead V_6 is found >75 percent of normal people.
3. Nonsignificant Q is:
   - Width Less than 0.04 sec.
   - Less than 0.03 mm deep
   - Usually does not have specific combinations (See Table 1.2 for combination).
   - Individuals <30 years tend to have deeper Q waves than >30 years old, e.g. in Lead III.
4. Q waves may be of different shapes (Fig. 1.14)
5. Poor R waves progression may mimic with Q wave (See R wave)

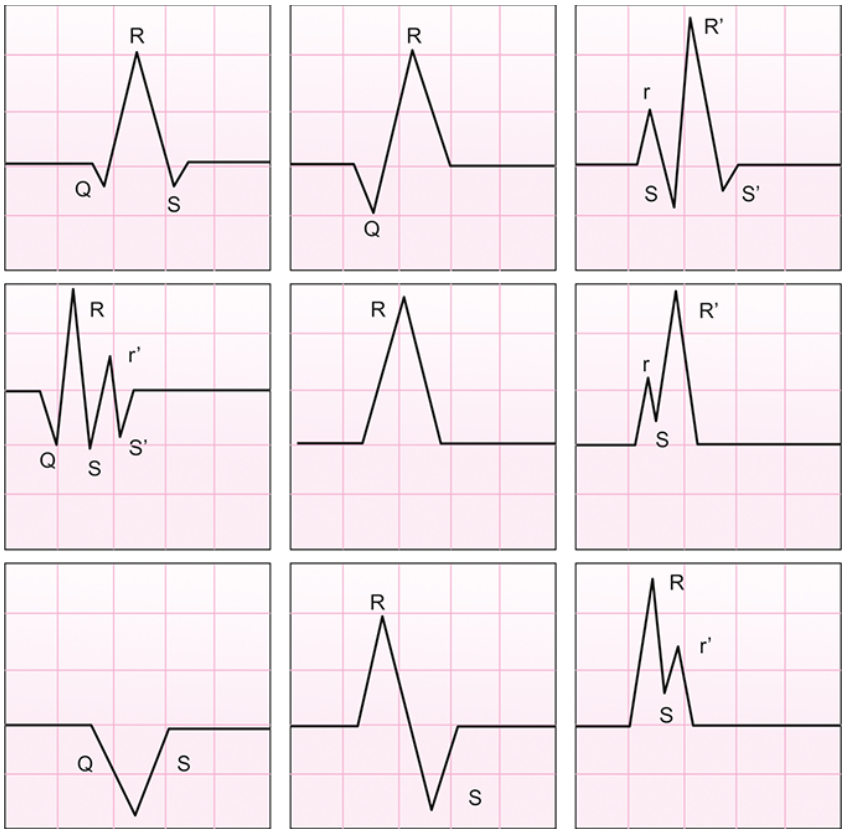


Figure 1.14: Note different morphologies of Q, R and S waves

Table 1.2: Differential diagnoses of Q waves

Q waves and its association (See next section)	
Q wave (significant) in lead II, III, aVF	Inferior infarction
Q wave (significant) in lead I, aVL, V_5, V_6	Anterolateral infarction
Small q wave in lead I + small r wave in lead III + left axis deviation (LAD) ≈ 60°	Left anterior fascicular block (LAFB)
Small r wave in lead I + small q wave in lead III + right axis deviation (RAD)	Right anterior fascicular block (RAFB)
Small q wave from lead V_3–V_6	Extreme counterclockwise rotation

Introduction with R Wave

1. Recalling figure which shows relationship of QRS and cardiac action potential, beginning of QRS coincides with the initial positive deflection of the action potential.
2. It can also present with different shapes (Remember: R wave cannot be negative, any negative deflection after Q wave is S wave) (*See* Fig. 1.14)

What is R Wave Progression?

In chest leads, R wave changes gradually its length from V_1 to V_6 and this variation is in accordance with the leads position to heart.

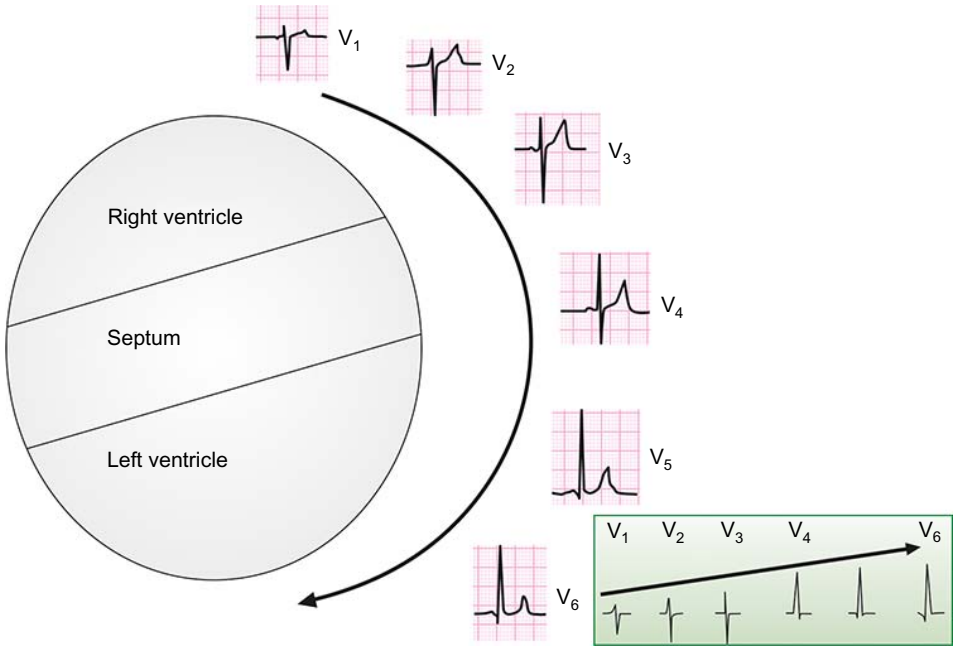
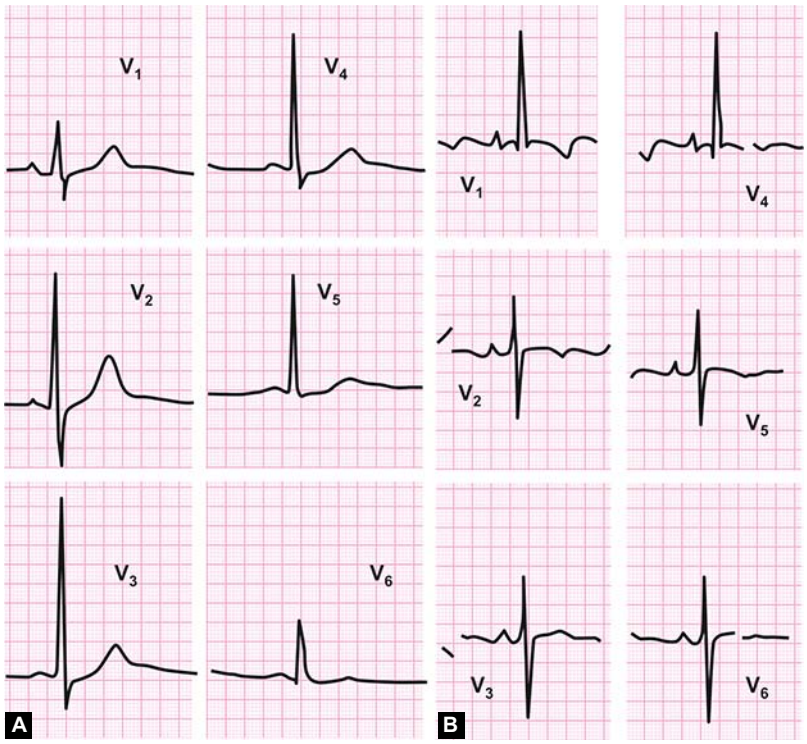


Figure 1.15: Schematic diagram showing chest leads positions and R wave progression. Note transition is V_3/V_4 and small q wave in lead V_6

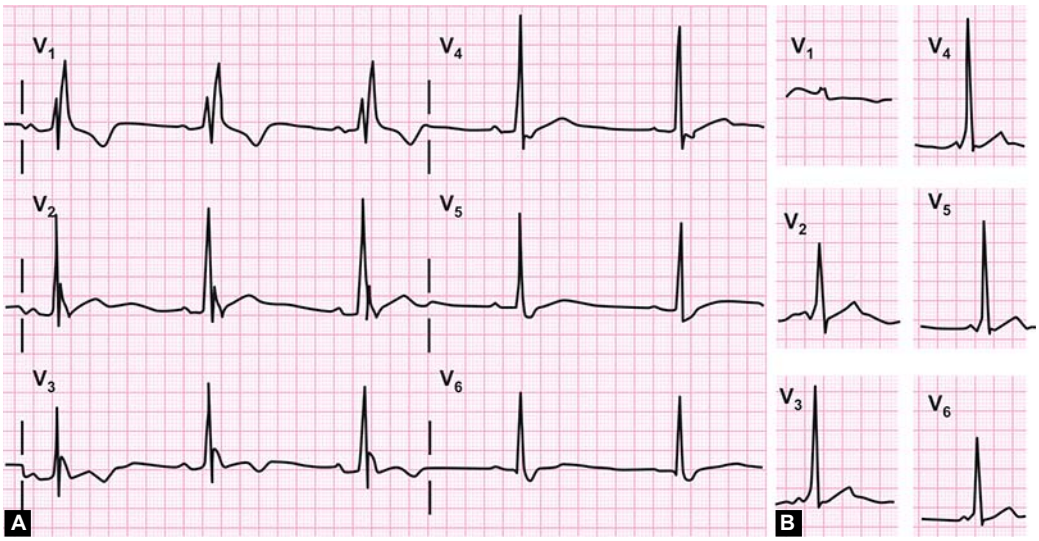
Figure 1.15 shows that leads V_1 and V_2 face right ventricle and leads V_3 and V_4 interventricular septum while V_5 and V_6 to left ventricle. Because muscle mass in left ventricle is more than right ventricle the R wave amplitude is greater in the leads V_5 and V_6 that is why in right ventricular hypertrophy R wave is also tall in leads V_1 and V_2 (Table 1.3).

Table 1.3: Causes of tall R wave in V_1–V_2 (Figs 1.16 to 1.18)

1.	Thin chest wall
2.	Normal variant in younger than 20 years
3.	Right ventricular hypertrophy
4.	Right bundle branch block
5.	WPW syndrome
6.	True posterior infarction
7.	Dextro position
8.	Wrongly placed leads in lower intercostal space
9.	Hypertrophic cardiomyopathy



Figures 1.16A and B: Isolated posterior MI. Note (A) tall R wave in leads V_1 and V_2 (with upright T wave) indicates posterior wall MI. (B) Shows right ventricular hypertrophy, note tall R wave in leads V_1 and V_2



Figures 1.17A and B: (A) Shows right bundle branch block (RBBB); Note tall R wave in leads V_1 and V_2. (B) Shows Wolff Parkinson White (WPW) syndrome. Note tall R wave in V_2. Also note the slurring in the initial portion of the PR segment called delta wave

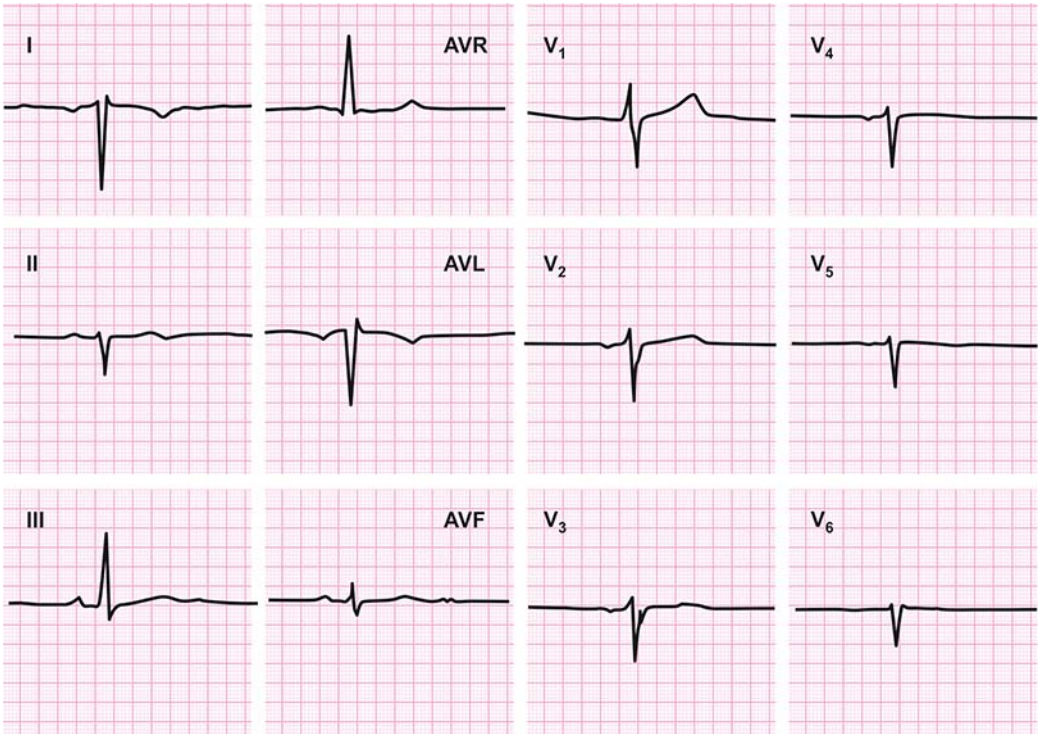


Figure 1.18: Dextrocardia. Note Tall R wave in leads V_1 and V_2. R wave height progressively decreases from V_1–V_6. Also note that P wave, QRS, T wave inverted in lead I and aVL and upright in lead aVR

Generally, R wave length in leads V_1 is as follows:

- 0.5 mm (12–20 years age)
- 0.8 mm (20–30 years)
- 0.6 mm (>30 years)

R Wave Transition

Also note QRS is predominantly negative. In leads V_1 V_2 and it changes to predominantly positive in leads V_5 and V_6 being equal in leads V_3/V_4 this is called Normal Transition. In case of COPD (Chronic Obstructive Pulmonary Disease), because of increase strain of right ventricle, the right ventricle may dilate rotated to clockwise position. In that condition leads V_1 and V_2 face the cavity of the right ventricle thus recording a Q wave and transition points shift to leads V_4/V_5 this is called Late Transition (Figs 1.19 and 1.20).

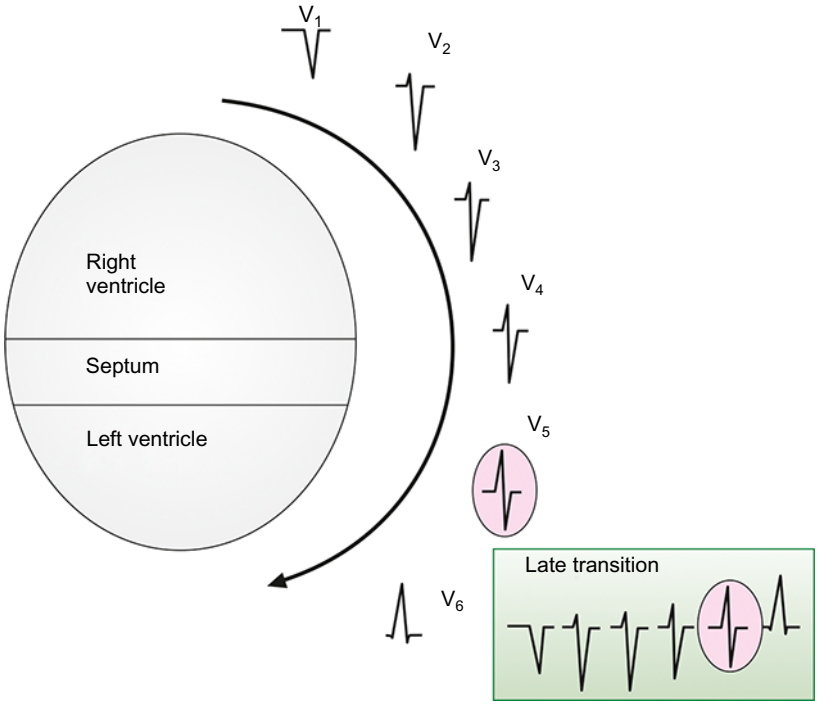


Figure 1.19: Note clockwise rotation of heart and late transition as seen in COPD. Note lead V_1 records a Q wave because it is facing cavity of right ventricle while normally present q in lead V_5 and V_6 is absent here

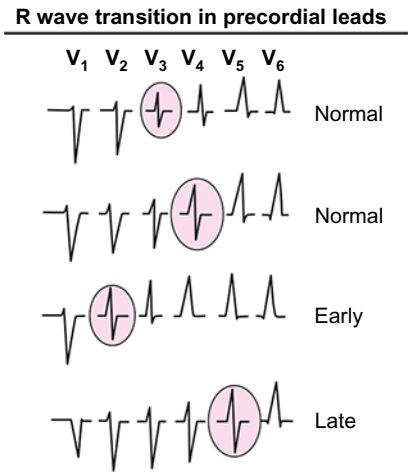


Figure 1.20: Note various patterns of R wave transitions

Intrinsicoid Deflection and its Diagnostic Importance

This is downward deflection after the peak of R wave (Recall current flow toward lead produces positive deflection and going away produces negative deflection). The time of onset of intrinsicoid deflection also known as R wave peak time, and is measured from the beginning of QRS complex to the peak R wave (Fig. 1.21).

Importance

If there is hypertrophy of ventricle or bundle branch block, the depolarization impulse takes longer time to reach the recording (due to increase muscle size or block). Thus, ECG shows delay in the onset of intrinsicoid deflection. This delay in the onset of the intrinsicoid deflection is an important criterion for diagnosis of LVH (Left Ventricular Hypertrophy) and BBB (Bundle Branch Block).

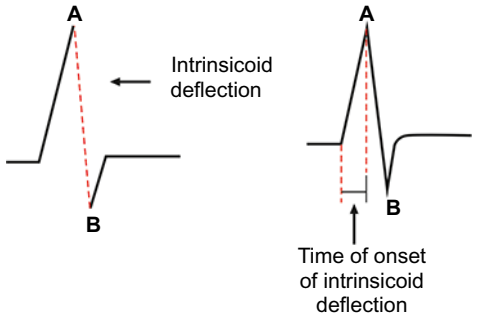
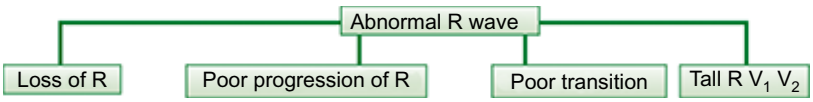


Figure 1.21: Note intrinsicoid deflection and time of onset of intrinsicoid deflection

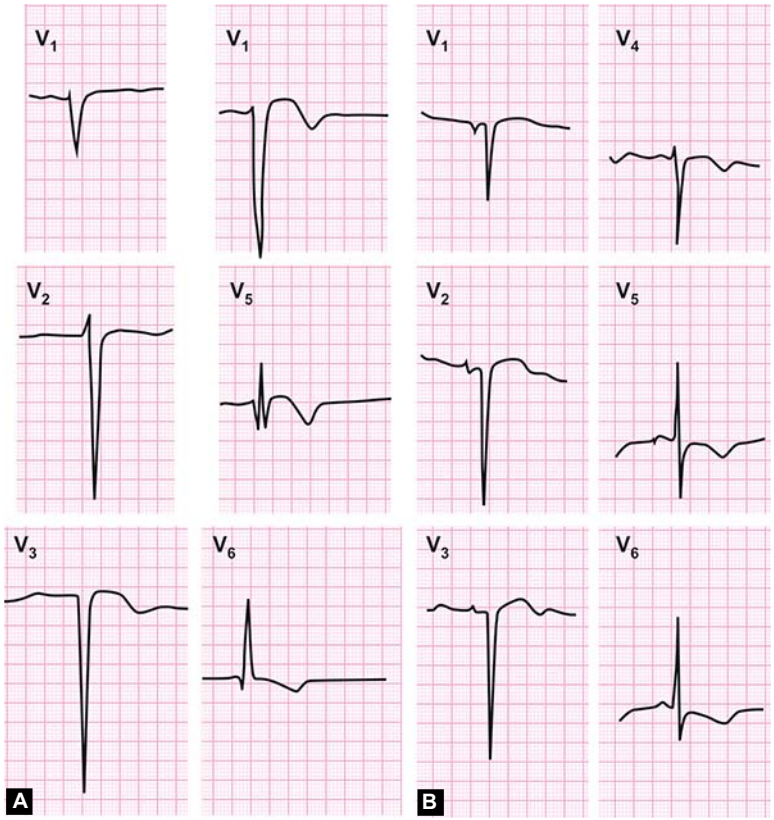
How Abnormal R Wave can Present?



Poor Progression and Loss of R

The above discussed progression of R wave is not seen if the heart muscle is suffering ischemic damage due to MI. For diagnostic purpose, remember that loss of R wave or poor progression should be seen along with ST segment and T wave, e.g.

1. Loss of R wave in leads V_1–V_3 + Isoelectric ST segment (i.e. not elevated or depressed) + T wave inversion = Anterior septal MI (Figs 1.22A and B)
2. Poor progression in leads V_1–V_4 + ST elevation = Acute anterior infarction
   Again note that isolated loss of single R does not carry diagnostic importance.



Figures 1.22A and B: (A) Anteroseptal MI. Note poor R wave progression. Also note deep Q wave in V_5. (B) Anteroseptal MI. Note ST elevation in leads V_1–V_4 with loss of R wave in leads V_4. Also note QS in leads V_1–V_4

Tall R Wave

Contrary to poor progression, R wave in leads V_1 and V_2 may be tall and indicative of underlying pathology (See Table 1.3).

Poor Transition

R wave late transition in clockwise rotation of heart has discussed earlier (*See* Figs 1.19 and 1.20).

Get Familiar with S Wave

Any negative deflection follows the first R wave or Q wave is S wave. Usually, S wave is seen with ST segment

When S Wave Alone is Significant

- Normal S wave is pointed and its duration is no more than 1 small square but in bundle branch block (BBB), it may be wide and slurred in lead I and V_6 (Fig. 1.23).

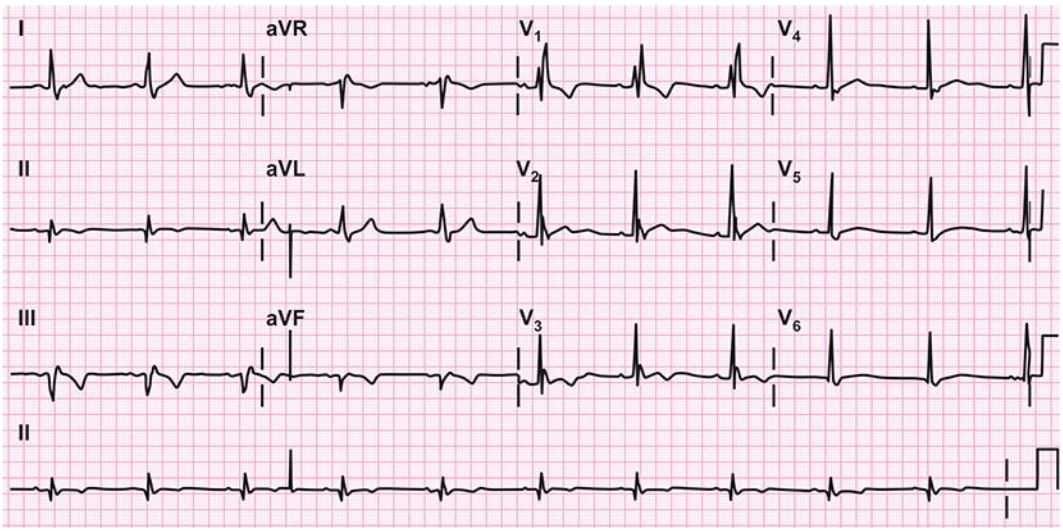


Figure 1.23: ECG with many findings including RBBB. At this stage did you notice slurred and broad S wave in leads I and V_6

- R/S ratio in lead V_1 is less than one (R shorter than S) and in lead V_6, R/S ratio is more than 1 (R > S). This condition helps in making diagnosis of right ventricular hypertrophy (RVH) (Fig. 1.24).

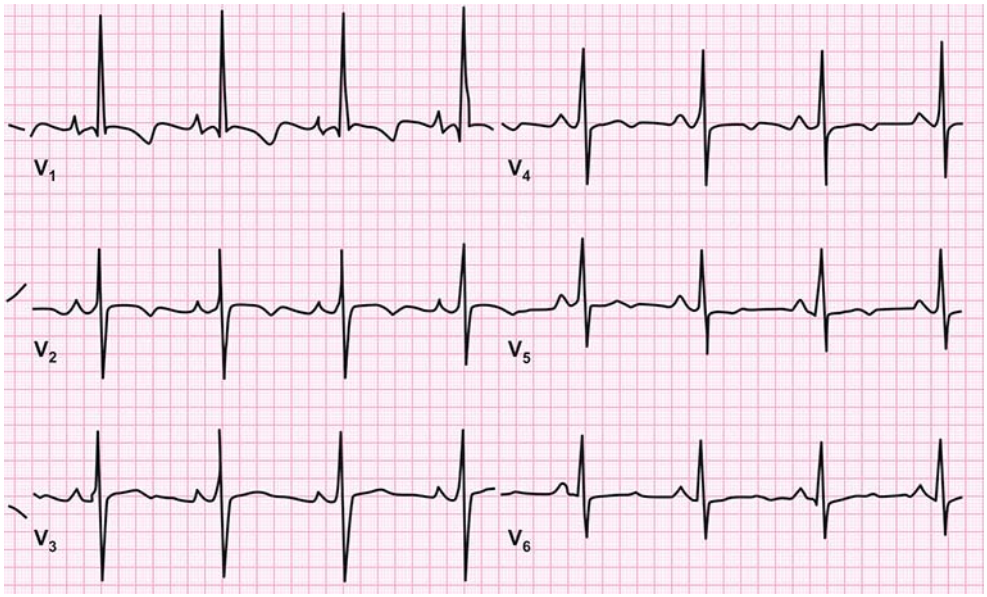


Figure 1.24: Right ventricular hypertrophy (RVH). Note tall R wave in lead V_1 and S wave in lead V_6 which is deeper than usual

T WAVE IN DEPTH

T wave may be upward and downward and it indicates ventricular repolarization.

Note the interval from beginning of QRS to apex of T wave coincides with absolute refractory period while last half of T coincides with relative refractory period (Fig. 1.25).

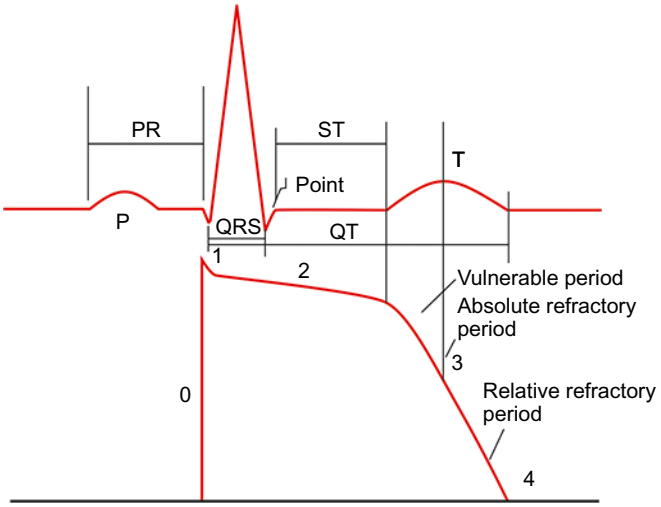


Figure 1.25: P-QRS-T, note T wave has upsloping and downsloping part, the initial upsloping part coincides with absolute refractory period of cardiac action potential

In normal states, T wave is:

- Note that a negative T wave is normal in lead aVR and lead V_1 this is because leads aVR and V_1 look down from above into the negatively charged interior of the heart and record a negative or cavity potential (Fig. 1.26A).
- Upright in lead I, II, V_3–V_6
- Variable in leads III, aVF, aVL and V_2.

Isolated T wave usually does not clue toward diagnosis. So again, recall our rule that to make diagnosis T wave should be taken along with other signs including ST segment depression or elevation, etc.

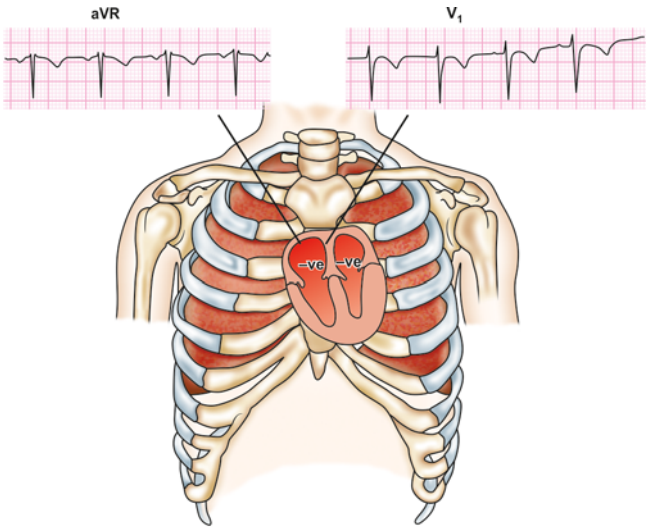


Figure 1.26A: Leads aVR and V_1 look down from above into the negatively charged interior of the heart and record a negative or cavity potential. That is why leads aVR and V_1 normally record inverted T wave

Different Presentations of Abnormal T Wave (Fig. 1.26B)

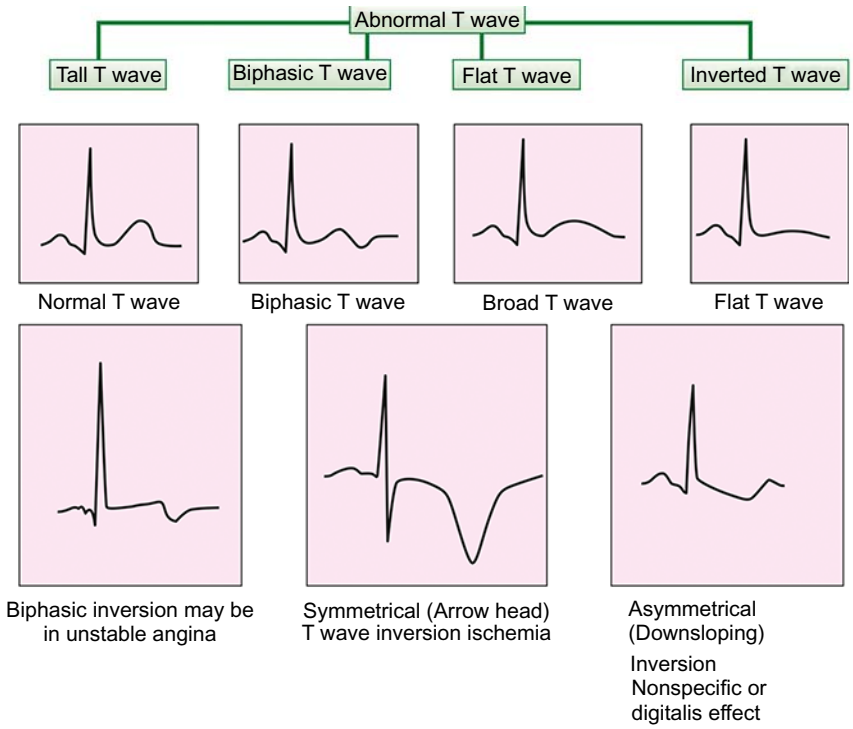


Figure 1.26B: Different presentations of abnormal T wave

T Wave Inversion

For significant T wave inversion, its shape and association with other leads is important.

1. Normal T wave inversion is asymmetrical with downsloping initial segment and second half steep upward. This is called as "downslopping" T wave inversion. Note the digitalis administration can also cause "downsloping" T wave inversion (Fig. 1.27).

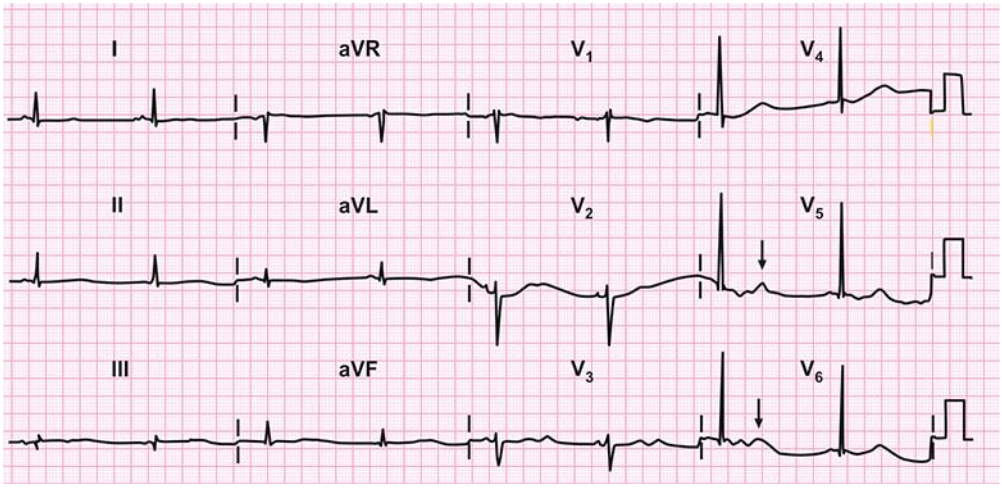


Figure 1.27: Down sloping T wave. At this stage note in leads II, III, aVF and V_4–V_6, T wave with downsloping initial segment. The patient was on digitalis

2. Symmetric T inversion both hands equal. This is also called arrowhead depression seen in ischemia (Fig. 1.28).

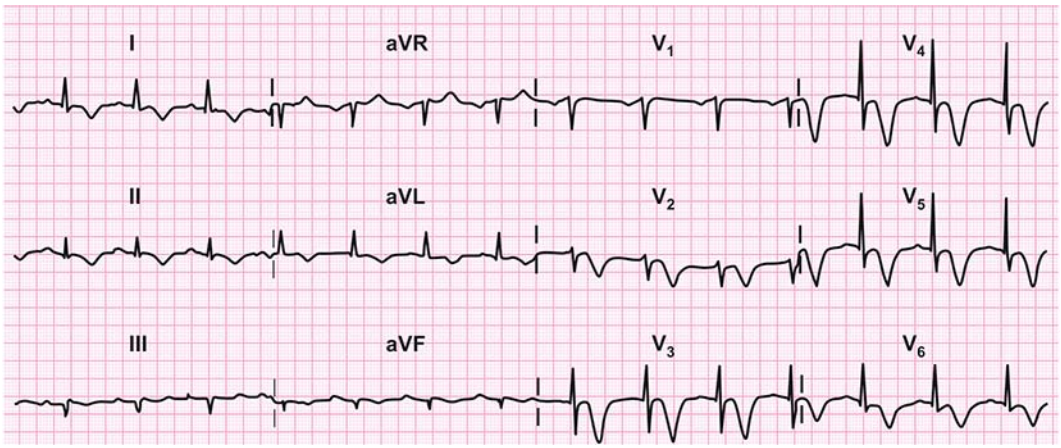


Figure 1.28: Note T wave inversion in most leads (except aVR). T wave inversion is almost symmetrical in leads I, II, aVL, aVF and V_2–V_6 (arrowhead inversion). These features are suggestive of myocardial ischemia

3. Normally during infancy, childhood and adolescence, T wave is inverted in leads V_1–V_3.
This is normal and called as Juvenile Pattern. In certain population this juvenile pattern is persistent in later ages (Fig. 1.29).

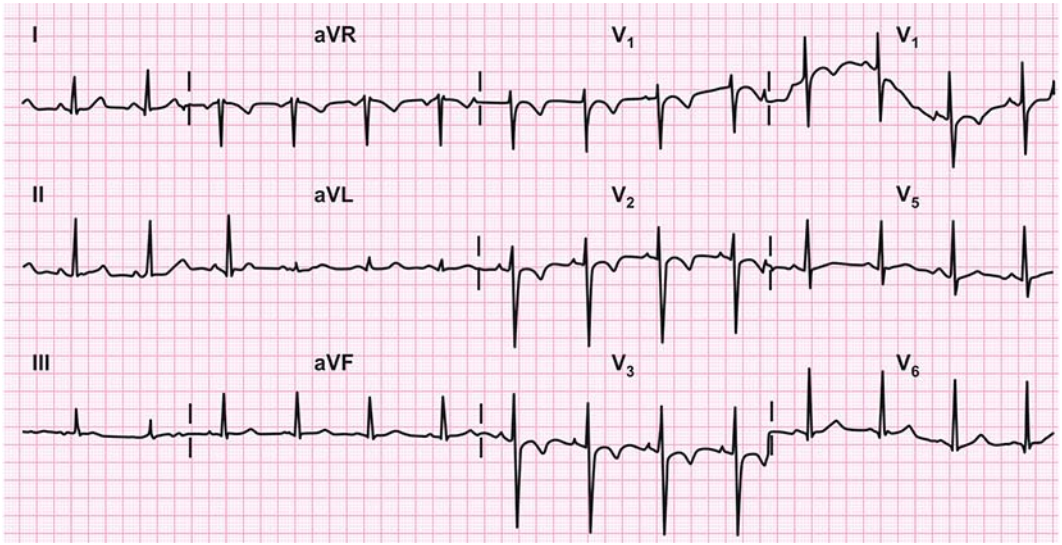
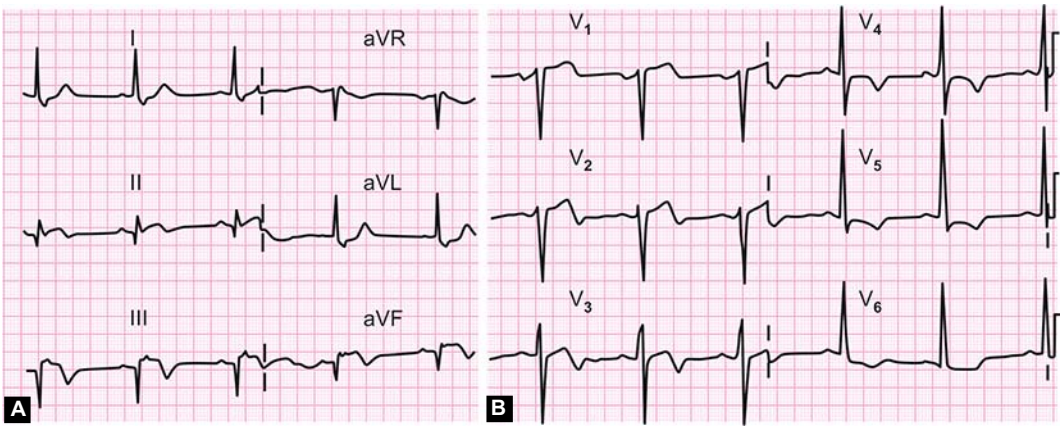


Figure 1.29: Persistent juvenile T wave pattern in a healthy individual. Note symmetrical T wave inversion in leads V_1–V_3

4. Biphasic T wave inversion can be seen in unstable angina, etc. (Figs 1.30A to C)



Figures 1.30A and B: Note bipolar T wave in lead II (A) and in leads V_1–V_3 (B)

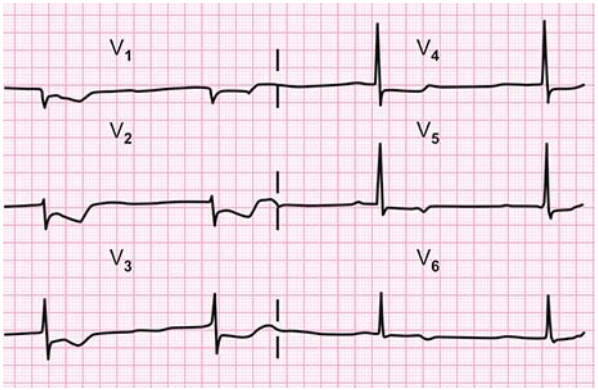


Figure 1.30C: Bipolar T wave in lead V_4–V_6

5. Deep T inversion with long QT interval indicates CNS pathology specially stroke, subarachnoid hemorrhage (SAH), etc. A very broad and symmetrically inverted T wave with prolonged corrected QT interval (QTc) is a common finding in CNS disorders especially subarachnoid hemorrhage (SAH). The pattern of broad and inverted T waves associated with long QTc intervals is commonly termed "cerebral", "neurogenic", and T wave (Fig. 1.31).

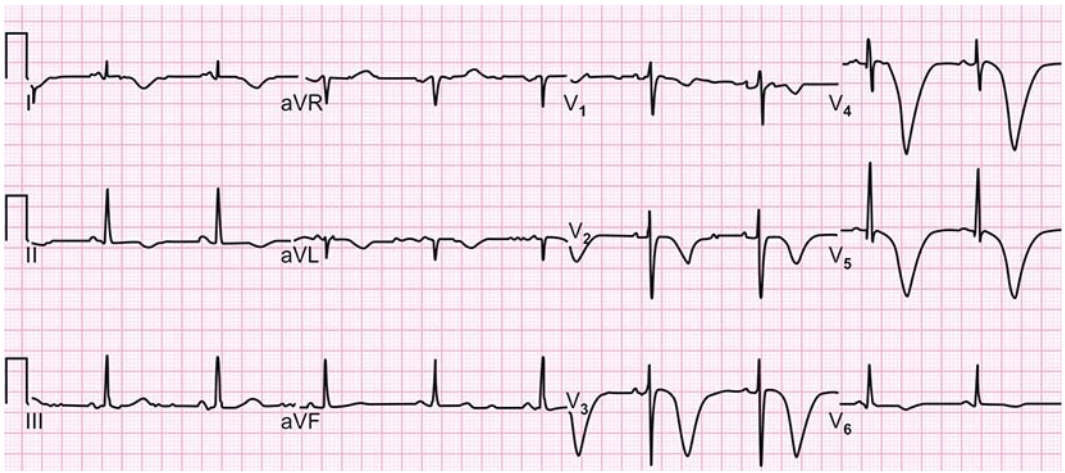
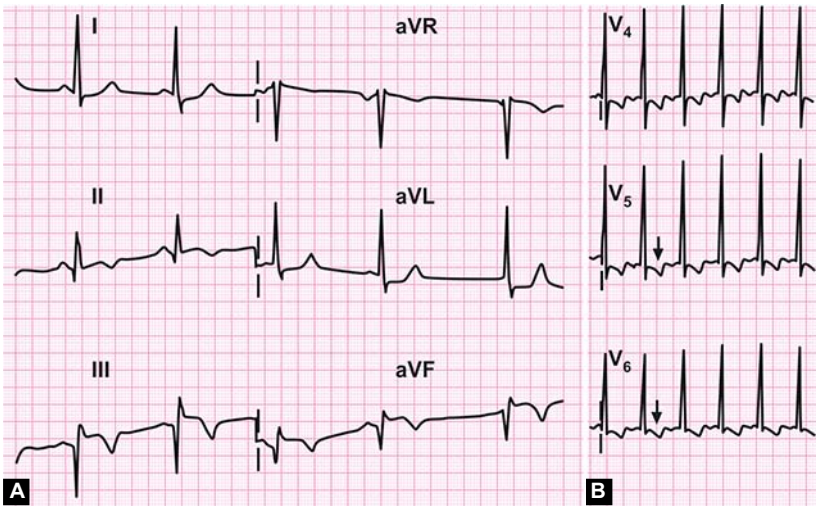


Figure 1.31: Broad, deeply inverted "cerebral" T waves and prolonged, corrected QT interval on 12-lead electrocardiogram of a patient with subarachnoid hemorrhage

T Wave Inversion should Always be Seen with ST Segment

Again T wave presentations are "suggestive" not diagnostic specially when isolated and it should be considered with ST segment elevation/depression (> 1 mm). T wave inversion with ST segment elevation, presences of Q wave or ST segment depression > 1 mm indicate ischemia/infarction (Fig. 1.32A). Note down sloping T wave inversion with ST segment depression is called strain pattern. It shows increase in work load and relative decrease blood supply in LVH (Fig. 1.32B). Sometimes T wave inversion is also associated with "abnormal shaped" ST segment (coved, hitched up, etc.), this also makes T wave inversion significant (*See* Table 1.4).

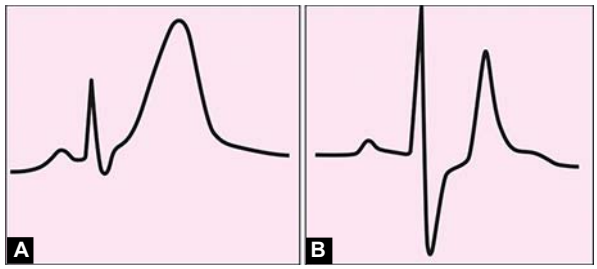


Figures 1.32A and B: Association of T wave and ST segment. (A) Shows T wave inversion with ST segment elevation and Q waves in leads II, III and aVF (acute inferior wall MI). (B) Shows downsloping T wave inversion with ST depression in LVH. This is called strain pattern

AMPLITUDE OF T WAVE

1. T wave may be taller than usual or may loss its length to become flat. Flat T wave is usually nonspecific but may indicate hypokalemia (Fig. 1.34).
2. T wave amplitude may increase resulting tall T wave (hyperacute T wave). Tall T wave may be with broad based and slight asymmetry or with narrow based (tented) and symmetrical. This tented T wave is specific for hyperkalemia (Figs 1.33A and B).

a. Broad based slightly asymmetrical, this may be earliest ECG sign of acute MI or may be non-specific (consider with all other sign. See next portion) (Figs 1.33A and 1.35).
b. Narrow based (tented) with symmetrical shape (Figs 1.33B and 1.36), this is typical for hyperkalemia.



Figures 1.33A and B: (A) Shows broad based, slightly asymmetrical T wave (may be AMI, nonspecific) while (B) Shows tented, symmetrical, narrow based T wave usually found in hyperkalemia

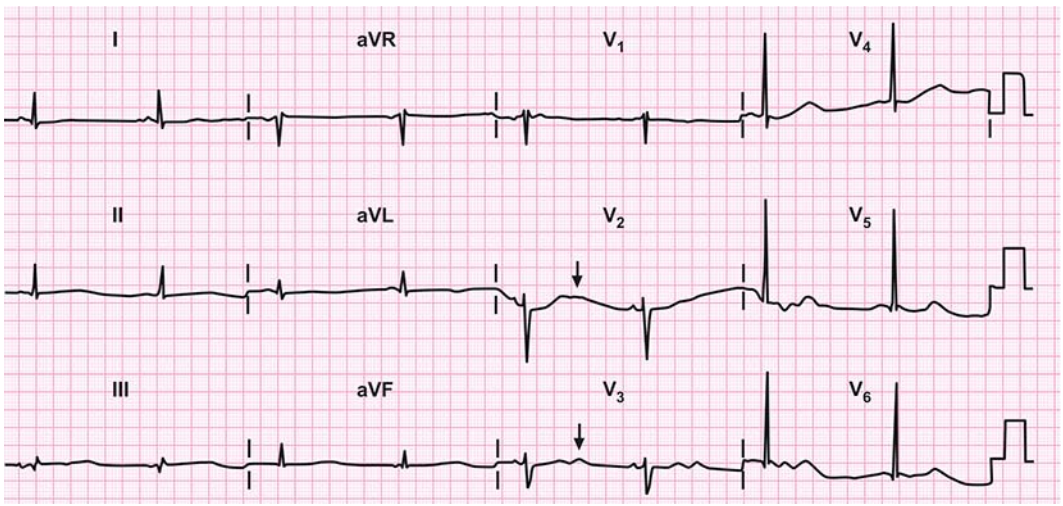


Figure 1.34: ECG of a person with prolonged vomiting. Note flat T wave in limb leads. Prominent U wave can also be seen (arrow)

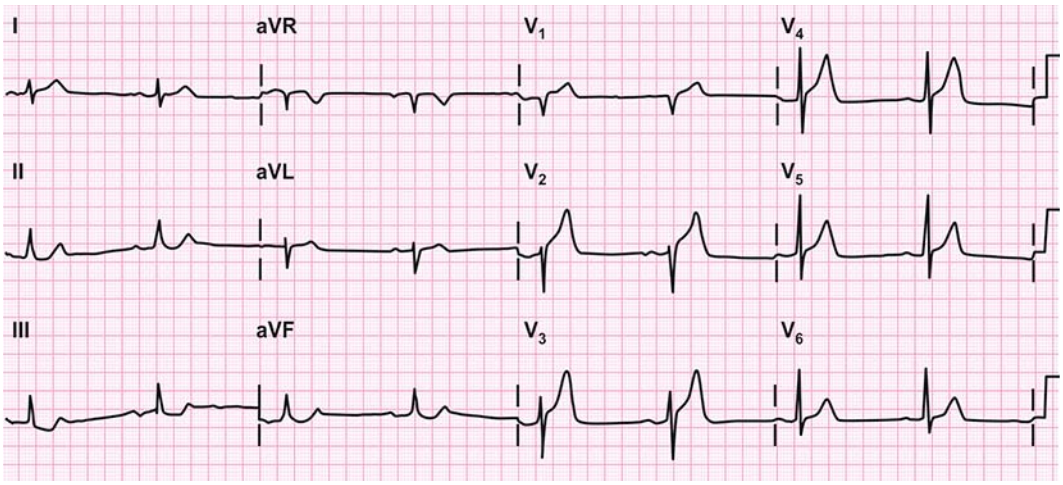


Figure 1.35: Note hyperacute T wave in a case of myocardial ischemia or injury. Here T wave is slightly asymmetrical broad based and associated with ST elevation

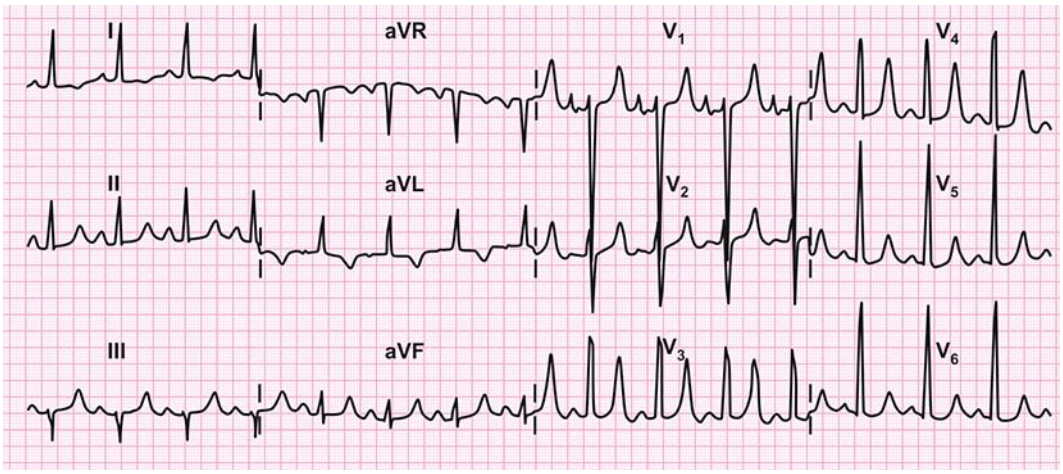
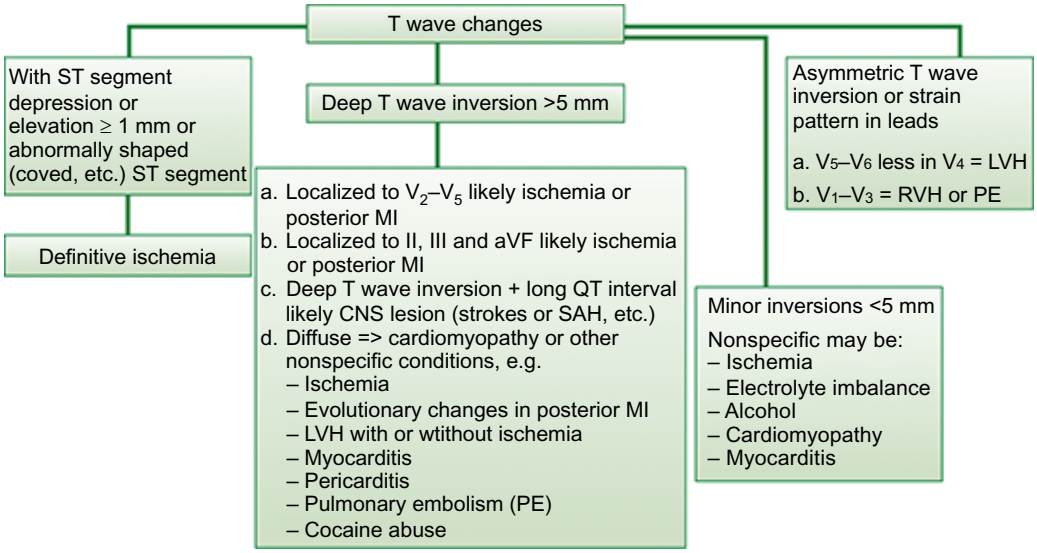


Figure 1.36: Tall tented narrow based symmetrical T wave in most leads indicating hyperkalemia

Table 1.4: T wave changes and their diagnostic importance



WHEN YOU SAY U WAVE, WHAT DO YOU MEAN?

U Wave

By definition, U wave follows T wave.

U wave is not always seen, if present best seen in V_2 and V_3. Its source is not certain however thought to be originate from repolarization of the papillary muscles or purkinje fibers (Figs 1.34, 1.37 and 1.38).

Prominent U wave most often seen in hypokalemia but may be seen in:

- Hypercalcemia.
- Class 1 A and 3 antiarrhythmic drugs [specially digitalis use (Digi effect) and quinidine]
- Thyrotoxicosis
- Congenital T syndrome
- LVH
- As with T wave negative U wave may be seen mostly in severe hypertension.

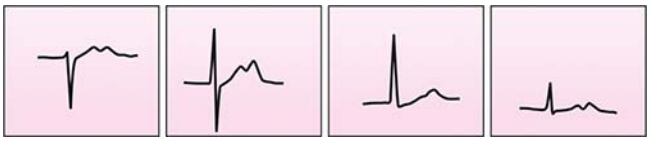


Figure 1.37: Note after T wave presence of U wave in different leads

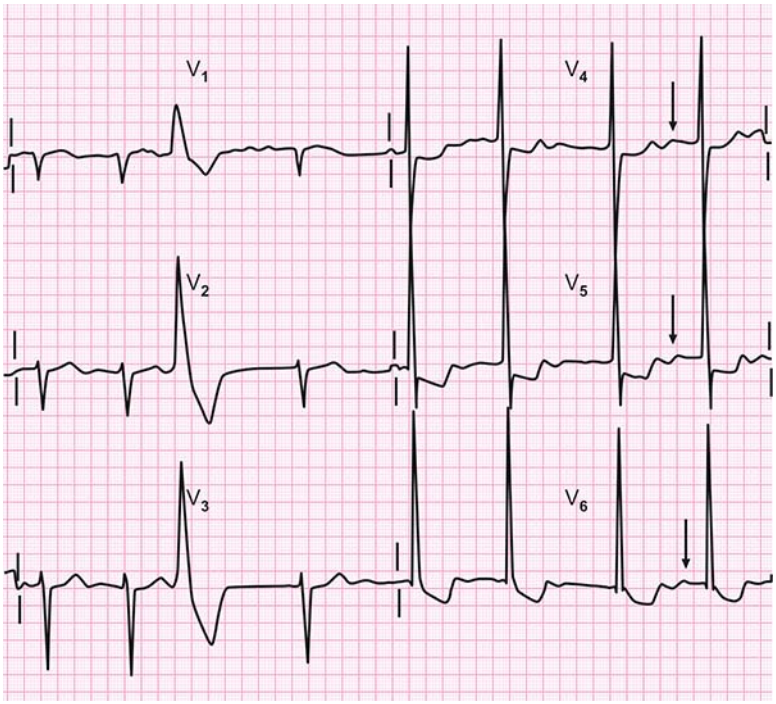


Figure 1.38: Note prominent U waves in leads V_4–V_6 (arrow) in a case of atrial fibrillation (AF). Here U waves are most likely secondary to LVH, and should not be confused with P wave as there is also atrial fibrillation

SEGMENTS + INTERVALS OF ECG

Conventionally, ECG is divided into many interval and periods, as described under.

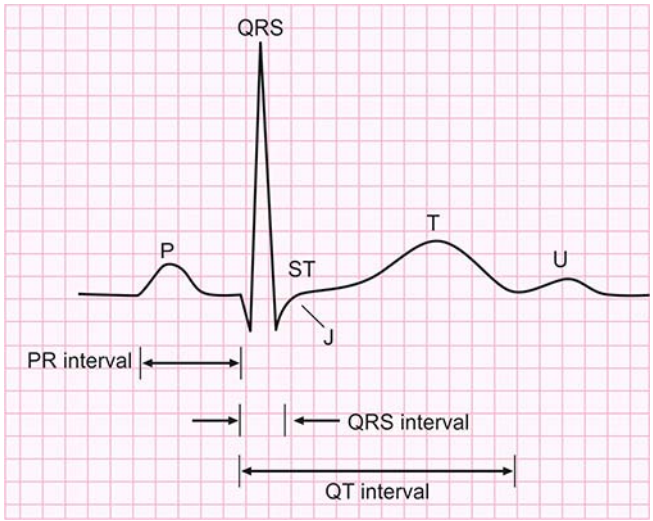


Figure 1.39: Note division of P-QRS-T into different intervals

PR Interval

PR interval measure from beginning of P wave to the beginning of QRS complex (if Q wave presents it is called PQ interval) (Fig. 1.39)

1. PR interval indicates atrial depolarization and repolarization and the delay in AV node conduction, i.e. time delay in passage of current from atrium to ventricle thorough atrioventricular node normal PR interval is from 0.12 to 0.20 seconds (3–5 small square) or 120–200 msec.
2. Since PR interval indicates the delay of current from atrium to ventricle through AV node, if this duration is increased it is said as AV block.

This increased duration may remain same or further increases gradually from one beat to other or sometimes even totally irregular. In the last case in fact there is not any AV conduction and atrium is contracting on its own and ventricle on its own (See AV block discussion in later section) (Figs 1.40 to 1.43).

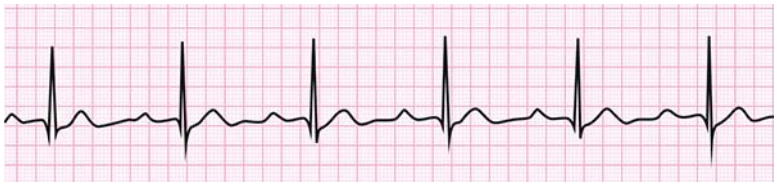


Figure 1.40: First degree AV block. Note PR interval is increased but it remains same

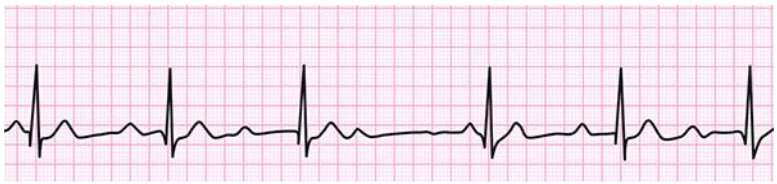


Figure 1.41: Second degree AV block. Note PR interval gradually increases

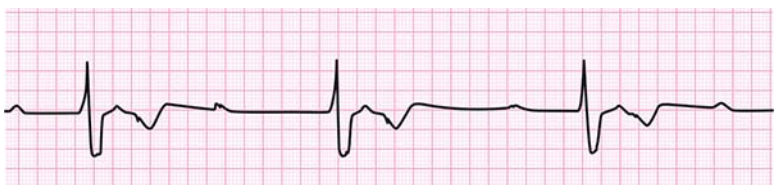


Figure 1.42: Third degree AV block. Note irregular PR interval duration

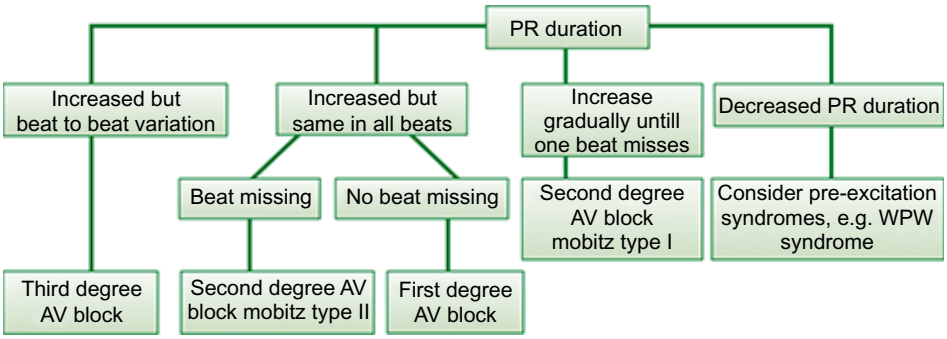


Figure 1.43: Differential diagnosis of PR interval

Is there PR Segment Depression/Elevation?

PR segment depression and elevation indicate atrial injury or pericarditis. In pericarditis PR is elevated in aVR and depressed in other leads specially II, AVF, V_4–V_6 (Fig. 1.44)

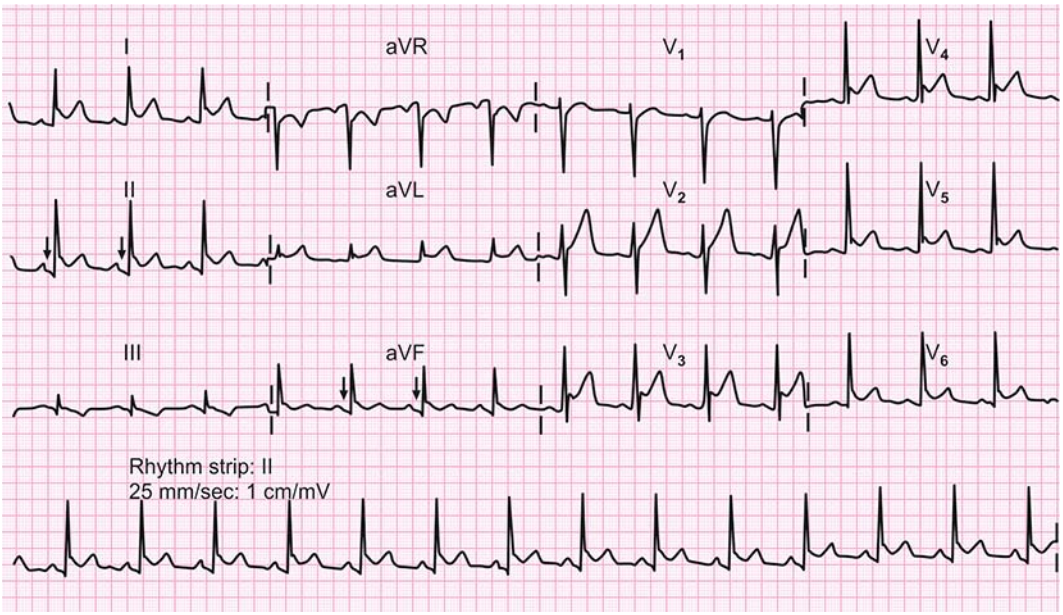


Figure 1.44: Note PR segment depression most prominent in leads II and aVF (also see rhythm strip) in aVR it seems elevated mostly due to ST segment depression

Decrease in PR Interval

Decrease in PR interval shows fast conduction from atrium to ventricle , this may occur if there is some accessory pathway in which conducts the current rapidly from atrium to ventricle resulting decreased PR as happens in WPW syndrome (Fig. 1.45, see next section for detail).

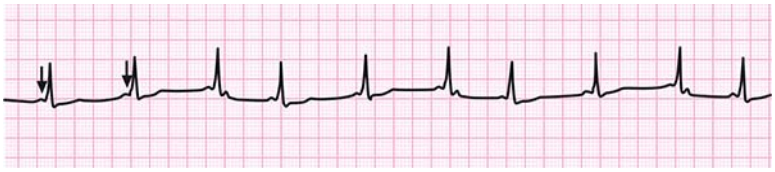
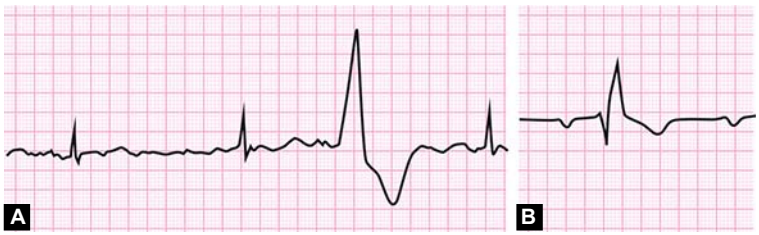


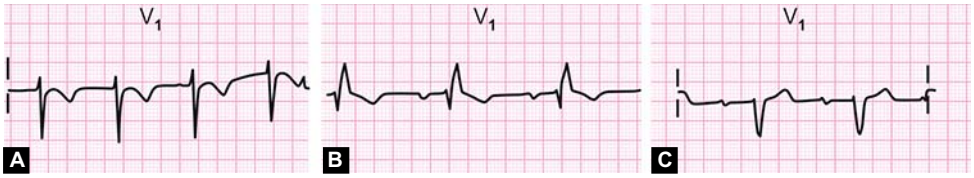
Figure 1.45: Note PR interval is decreased and there is slurring of PR segment. This is a case of Wolff-Parkinson-White (WPW) syndrome

What Does QRS Interval Show?

QRS duration is 0.05 to 0.1 seconds (2 and half small boxes) greater than 0.1 seconds should alert viewer to check for bundle branch block (BBB). While broad QRS also clues its origin from focus lower than AV node or Bundle of His as seen in ventricular premature beats (Figs 1.46 and 1.47).



Figures 1.46A and B: Broad QRS complex. A broad QRS shows its origin from ventricle (A). Ventricular premature contraction VPC, while (B) is a case of BBB



Figures 1.47A to C: Note QRS durations. (A) Shows normal duration. (B) Shows broad QRS in RBBB. (C) Shows broad QRS in LBBB

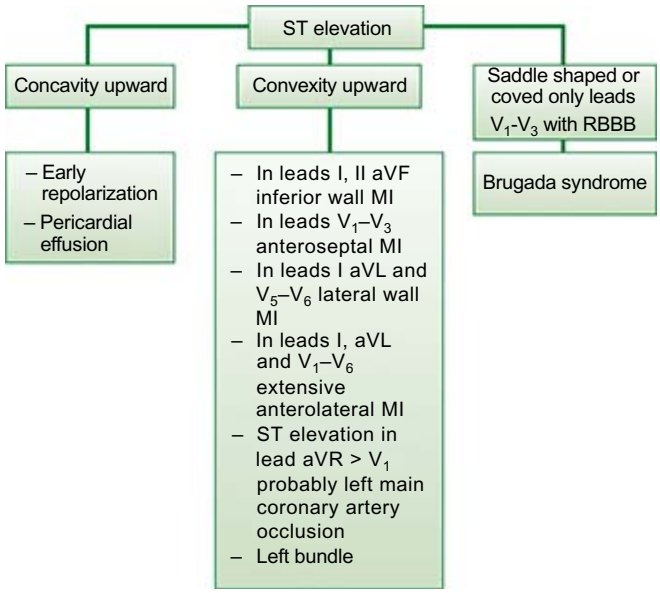
Familiar with ST Segment (See Fig. 1.39)

- ST segment joins QRS complex and T wave and has duration of 0.08–0.12 sec. But its isoelectric position (not elevated or depressed more than 1 mm) is more important than its duration.
- The point which joins QRS and beginning of ST segment is called J point.
- ST segment elevation and ST segment depression both have diagnostic importance.

How ST Elevation can Present?

ST elevation represents epicardial (outer muscle wall) injury or coronary spasm (ST depression shows subendocardial injury). ST elevation may present with different morphologies discussed below (Table 1.5):

Table 1.5: ST elevation patterns and their diagnostic associations



ST Segment Elevation with Convexity Upward

It is also wise approach to look shape of ST while considering its elevation. ST elevation MI (STEMI) usually present with convexity upward (*See* Figs 1.56A to E).

Remember

1. Inferior wall MI–ST elevation in leads II, III and AVF (inferior limb leads) (Fig. 1.48)
2. If ST elevation only in leads V_1–V_3 called anteroapical MI (also anteroseptal)
3. ST elevation in leads I, aVL, and V_5–V_6 indicate lateral wall MI
4. ST elevation in leads I, aVL and V_1 through V_6 (eight or more leads) show extensive *Anterolateral MI (Fig. 1.49).*
   Note: Site opposing the MI shows reciprocal depressions which strengthen the diagnosis or, this is called as reciprocal depression. This also helps to distinguish from pericarditis where ST elevation is present in most leads but ST depression is present only in lead aVR usually.
5. ST elevation in leads V_3R, V_4R (right sided pericardial leads)
   (See earlier). Indicate right ventricular MI.
6. ST elevation with convexity upward can also be seen left bundle branch block (LBBB), but here QRS duration is greater than 0.12 sec. Diagnosis of MI in the presence of bundle branch block is difficult. Sometimes ST segment is with opposite polarity to QRS complex this causes discordant ST elevation while sometimes ST segment and QRS both are with same polarity this is called concordant ST elevation (see later discussion) (Fig. 1.50).
7. ST elevation with convexity upward can also be seen in left ventricular hypertrophy LVH (Fig. 1.51).
8. Persistent ST elevation more than one week after MI indicates ventricular aneurysm (Fig. 1.52).
9. ST elevation can be caused by coronary artery spasm but disappears when relieved by nitroglycerine.
10. Extreme ST segment elevation (≥5 mm) in lead V_1 and V_2 also indicate acute ischemia.

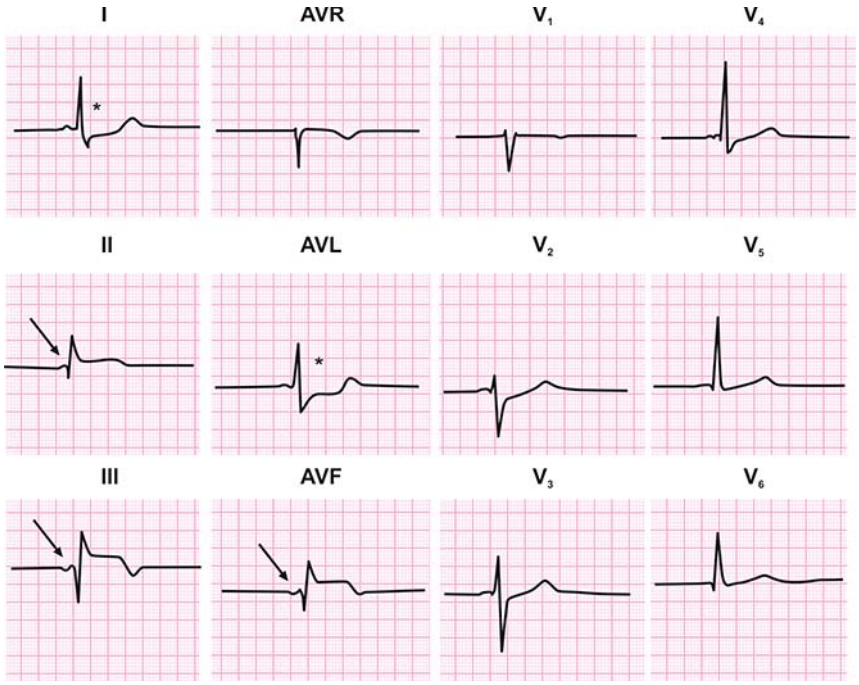


Figure 1.48: Note ST elevation in a case of inferior wall MI in leads II, III and aVF. Arrow shows Q wave, while asterisk shows reciprocal depression in leads I and aVL

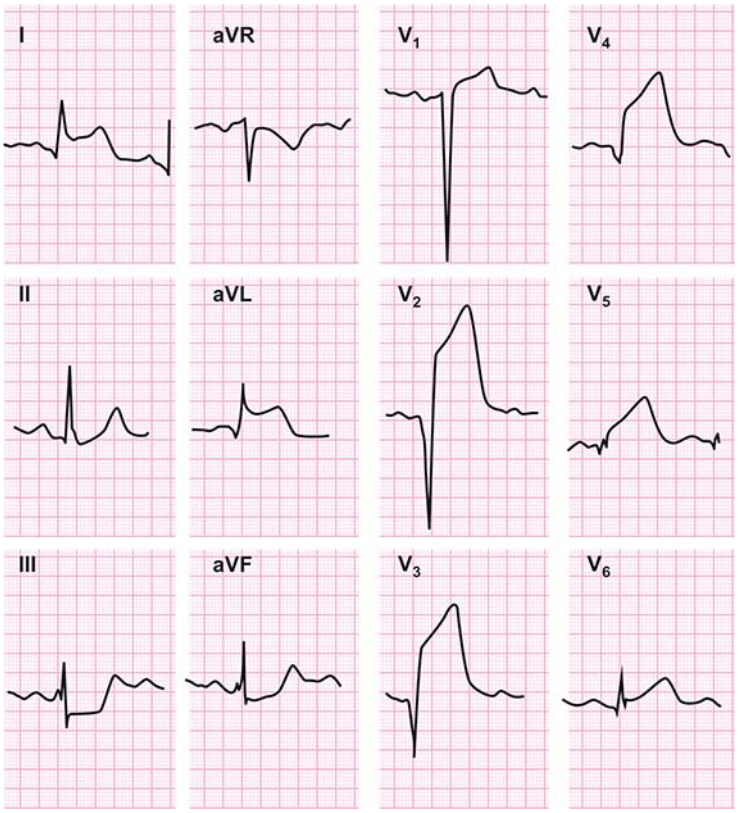


Figure 1.49: Extensive anterolateral MI. Note ST elevation in leads I, aVL and V_1–V_5. Leads II, III and aVF show reciprocal depression

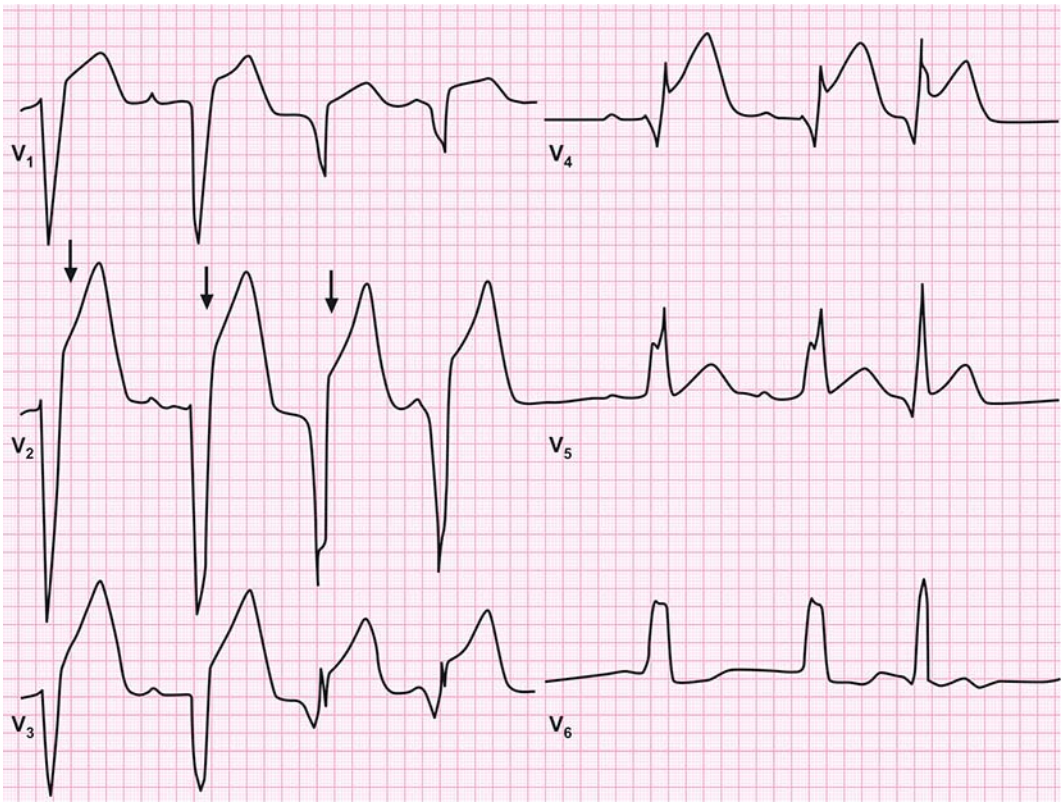


Figure 1.50: ST segment elevation in the presence of LBBB. V_2 shows discordant (arrow) while V_5 shows concordant ST elevation (see text for details)

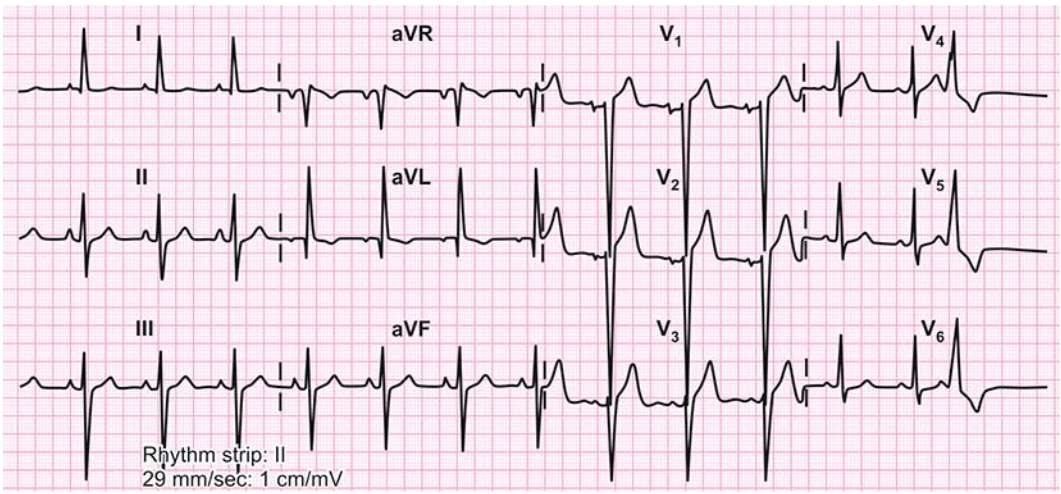


Figure 1.51: ST elevation in LVH. Note leads V_1–V_4 show prominent ST elevation with upward convexity

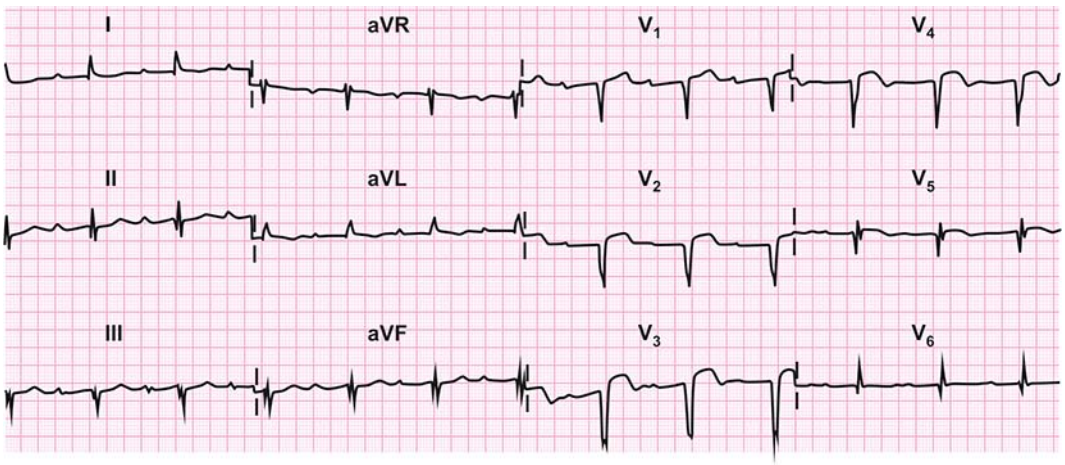


Figure 1.52: Persistent ST elevation (most obvious in leads V_1–V_4) in a person with prior anterolateral MI indicates development of ventricular aneurysm

ST Elevation Clues Toward Specific Coronary Artery

A. ST elevation in lead aVR> elevation in lead V_1 indicates Left main coronary artery disease (Fig. 1.55).
B. ST segment elevation in anterior leads and precordial leads in case of left anterior descending artery (LAD) occlusion (Fig. 1.54).
C. In case of right coronary artery (RCA) the ST segment elevation is more prominent in inferior leads (Fig. 1.53).

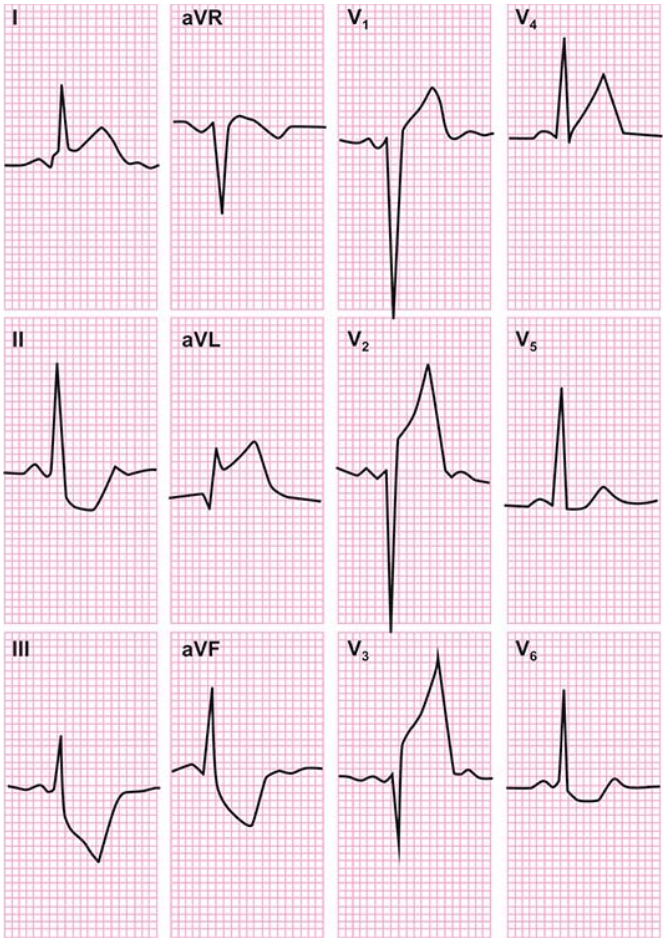


Figure 1.53: Left anterior descending artery (LAD) involvement. Note ST elevation is more prominent in anterior (leads I, aVL) and precordial (especially V_1–V_3) leads

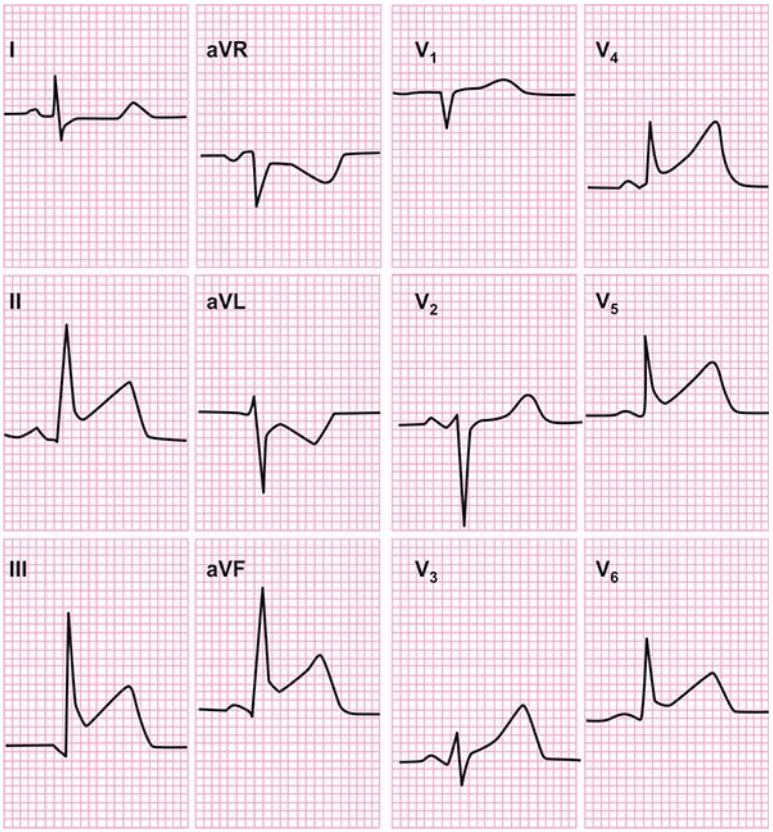


Figure 1.54: Right coronary artery (RCA) involvement. More ST elevation in inferior II, III, aVF leads

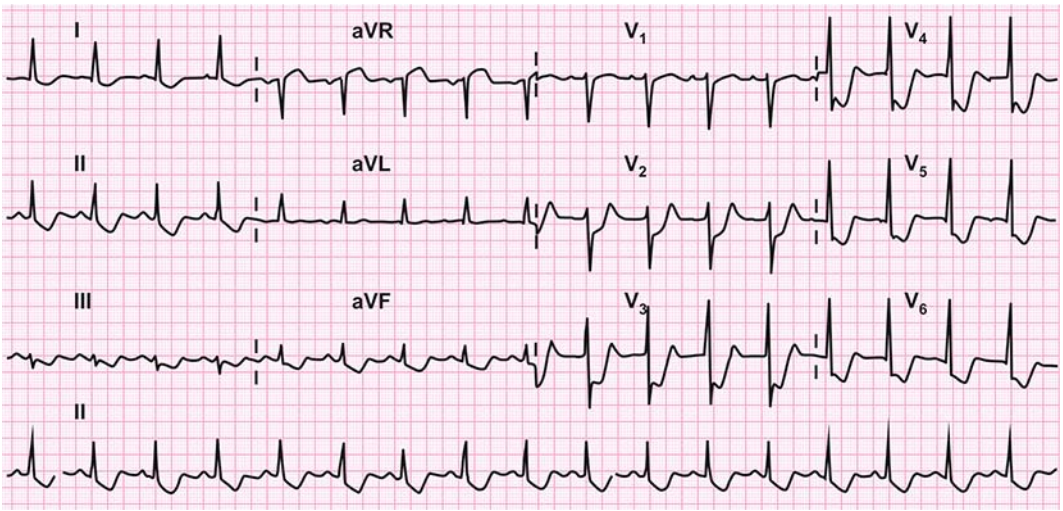
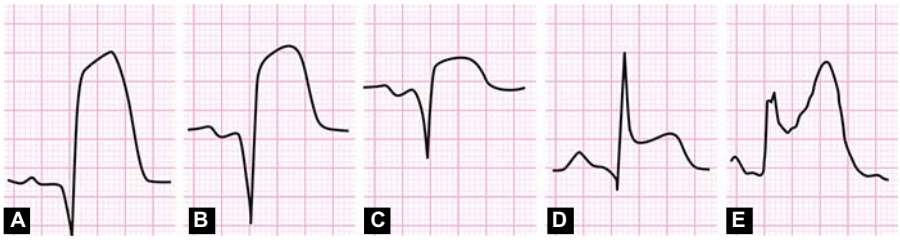


Figure 1.55: ST elevation in lead aVR > ST elevation in lead V_1 indicates left main coronary artery disease



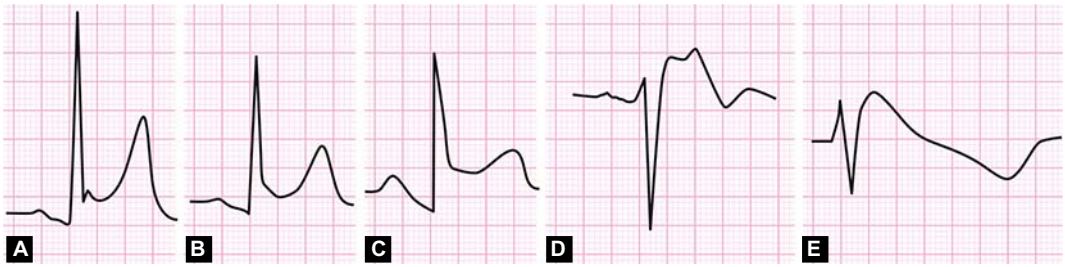
Figures 1.56A to E: Show convexity upward ST elevation usually seen STEMI

ST Elevation with Concavity Upward (Figs 1.57A to E)

This type of elevation is not uncommon in healthy individuals. Most commonly it is seen in the following conditions:

Early Repolarization (High J Point)

Early repolarization is characterized by elevated ST segment with concavity upward and elevated J point with a small notch at the point, common in African Americans (Fig. 1.58).



Figures 1.57A to E: (A to C) Show concave ST elevation which may be seen in pericarditis, early repolarization or may be nonspecific. (D) Shows fish hook like pattern. Note saddle shaped (E) and coved ST segment seen in Brugada syndrome

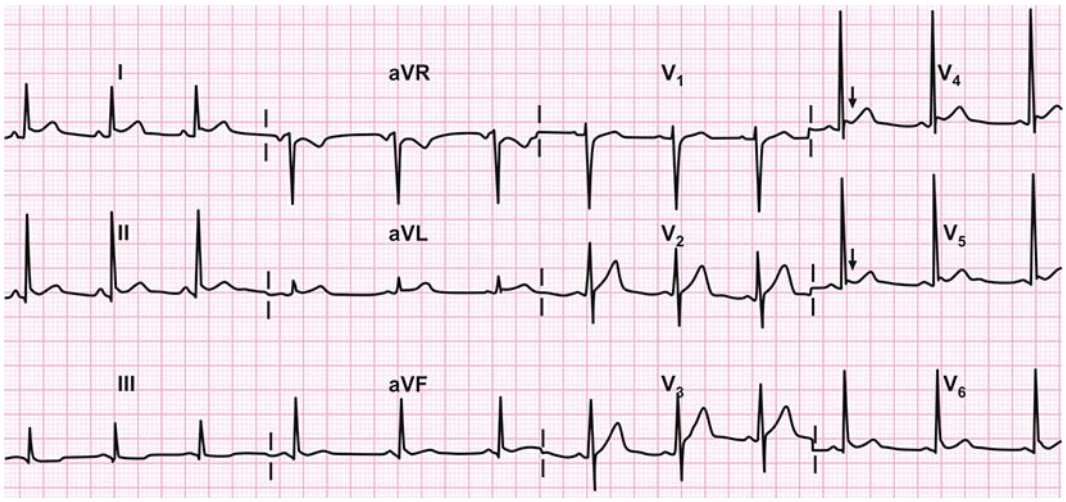


Figure 1.58: High J point with ST segment having upward concavity in leads I, II, aVL, aVF, V_4–V_6 (Arrow shows fish hook like shape with a notch)

Pericarditis

In pericarditis, there is also ST elevation. This can be differentiated from ST elevation in MI by many factors including the following:

- ST elevation in pericarditis is with concavity upward in most cases. In pericarditis, lead II has higher ST elevation than lead III (contrasting with most inferior MI) also the highest STE is in V_3–V_6 and in the inferior leads, with reciprocal depression only in aVR. In MI, T wave inversion is seen in early stages while in pericarditis it is seen in late stage (Fig. 1.59).

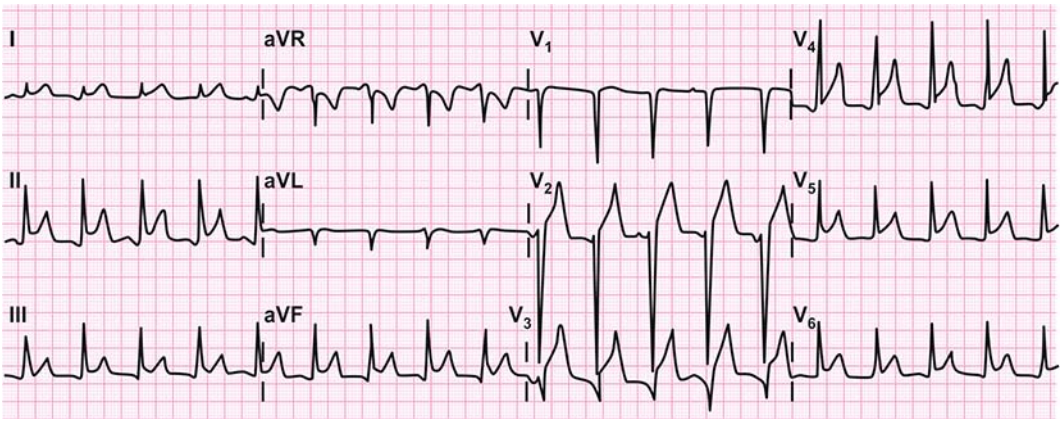


Figure 1.59: ST elevation pattern in case of pericarditis. Note concavity upward, higher ST elevation in lead II than in lead III, ST elevation in V_6 and V_5 is higher than other precordial leads

Saddle Shaped or Coved ST Elevation (Fig. 1.60A)

If ST elevation shows a typical coved or saddle shaped pattern in V_1–V_3 in the presence of complete or incomplete RBBB it is wise to consider Brugada syndrome which is common in Asian population and is a common cause of idiopathic ventricular fibrillation and sudden death (See next section).

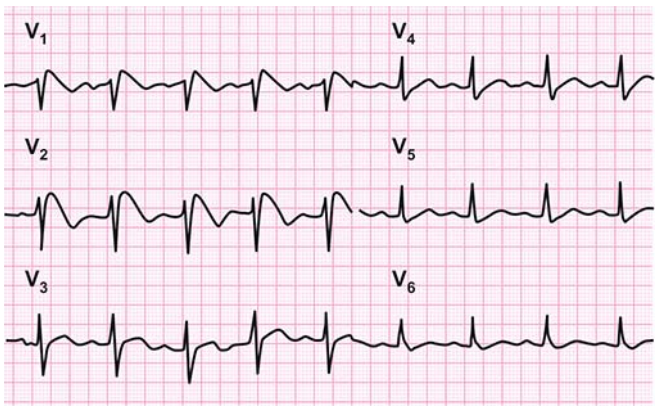


Figure 1.60A: Coved ST elevation in leads V_1–V_3 with RBBB is characteristic of Brugada syndrome

ST Segment Depression

ST segment depression can be flat, down sloping and up sloping (Table 1.6). Up sloping ST depression is non-specific while flat and down sloping usually a good clue for ischemia (Fig. 1.60B).

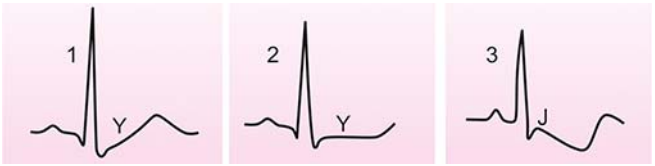


Figure 1.60B: ST depressions. Upsloping or junctional (1), flat (2), and downsloping (3). Note J point is depressed in all three but later portion Y has different patterns

Table 1.6: ST segment depression and its associations

ST segment depression		
Upsloping (junctional) ST segment depression	Flat/downsloping ST segment depression	ST elevation in some leads while ST depression in some leads of same ECG
– Nonspecific – In exercise testing > 4 mm may indicate ischemia	≥ 1 mm, in 2 or more leads (in 2 or more QRS complexes) with/without T wave inversion – Ischemia – ST depression + T wave inversion in $V_1 – V_3$ = RVH – ST depression + T wave inversion in $V_4 – V_6$ = LVH (Strian pattern consider other findings also) – May be nonspecific	– ST elevation in precordial leads and depression in leads II, III, aVF indicate reciprocal depression in anterior MI – ST elevation in leads II, III, aVF and depression in leads I, aVL indicate reciprocal depression in inferior MI – ST depression in $V_1–V_2$ with tall R wave in $V_1–V_2$ check for posterior MI – ST depression in aVR but elevation in most of other leads consider pericarditis

ST segment depression can be divided into two groups (Fig. 1.60).

1. Junctional depression (upsloping)
2. Flat or downsloping depression

Junctional or Upsloping ST Depression (Figs 1.60 and 1.62)

Only the junction of ST with QRS is depressed and rest of the segment slope upward to the T wave (Upsloping depression). ST depression of this type is not significant usually however during exercise if this depression is severe (>4 mm) check for ischemia (Note rate of rise of ST segment in these conditions is also considered).

Flat or Downsloping ST Depression (Figs 1.63A and B)

ST segment depression in flat or downsloping manner usually indicates ischemia and many other nonischemic causes.

Ischemic Causes

1. For ischemia to diagnose, ST depression should be:
   a. Greater than 1 mm.
      0.5 mm depression: possible ischemia
      0.1 mm depression: probably ischemia
      1.5 mm to 2 mm depression: almost certainly ischemia (Table 1.6).
   b. Present in two or more than two leads
   c. Present in two or more consecutive QRS complex.
   d. Flat or down sloping with or without T wave inversion.
      However; ECG findings should be considered in relation with the clinical findings of patients.
2. ST segment depression as a reciprocal depression in leads opposite to infarct area (see earlier discussion) (Fig. 1.64).
3. Left/right ventricular hypertrophy typically causes ST depressions with inverted T wave, sometimes called as strain pattern (Fig. 1.65, also see Fig. 1.32).

Nonischemic Causes

These may be (for specific ECGs see discussion in next part).

1. Pericarditis (interestingly ST segment is elevated in most of the leads but in lead aVR ST segment is depressed)
2. Digoxin use (Digoxin effect) typically causes “reversed check sign” or “scooping” shape ST segment depression (Figs 1.66A and B).
3. Hypokalemia and hyperkalemia
4. Pulmonary embolism (PE).
5. Subarachnoids hemorrhage (SAH) or other CNS pathologies
6. Myocarditis and cardiomyopathy.

Normal Variant ST Depression

1. Pseudo ST depression (wandering baseline due to poor skin electrode contact) (Fig. 1.61)
2. Physiologic junctional depression with sinus tachycardia (must be likely due to atrial repolarization).
3. Hyperventilation induced ST depression
4. After drinking cold water, etc.

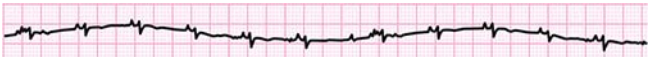


Figure 1.61: Wandering baseline. It may give false impression of ST depression

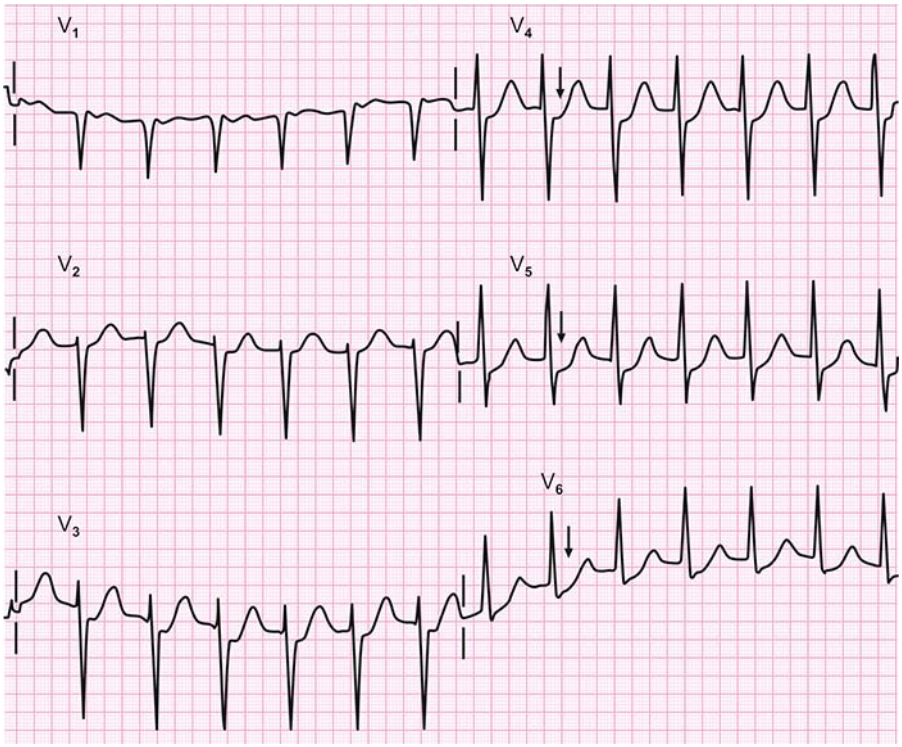


Figure 1.62: Note upsloping ST segment depression (Arrow)

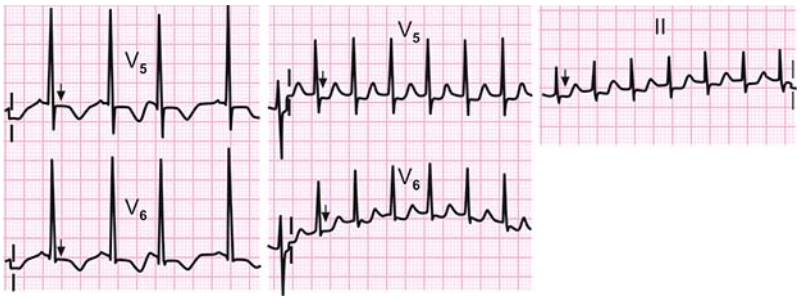


Figure 1.63A: Flat ST segment depression in different ECGs (arrow)

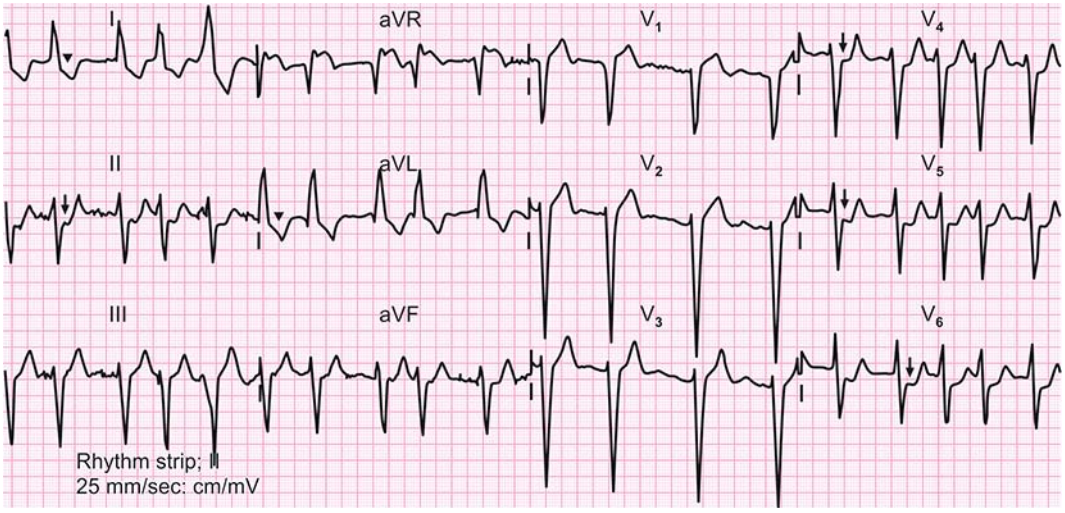


Figure 1.63B: Note flat ST depression (leads II and V_4–V_6, without T wave inversion, arrow) and down sloping ST depression (leads I and aVL, with T wave inversion, arrowhead) in same ECG

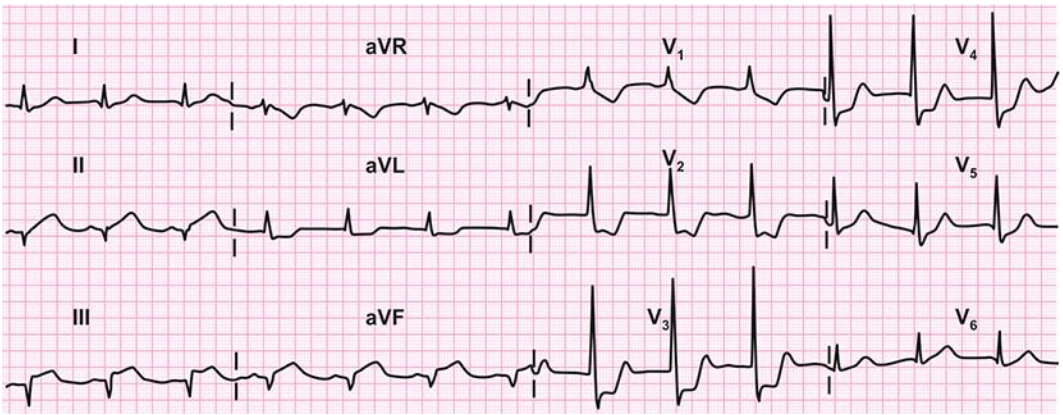


Figure 1.64: ST elevation (II III, aVF) and "reciprocal ST depression" (I, aVL, V_1–V_6) in inferior wall MI

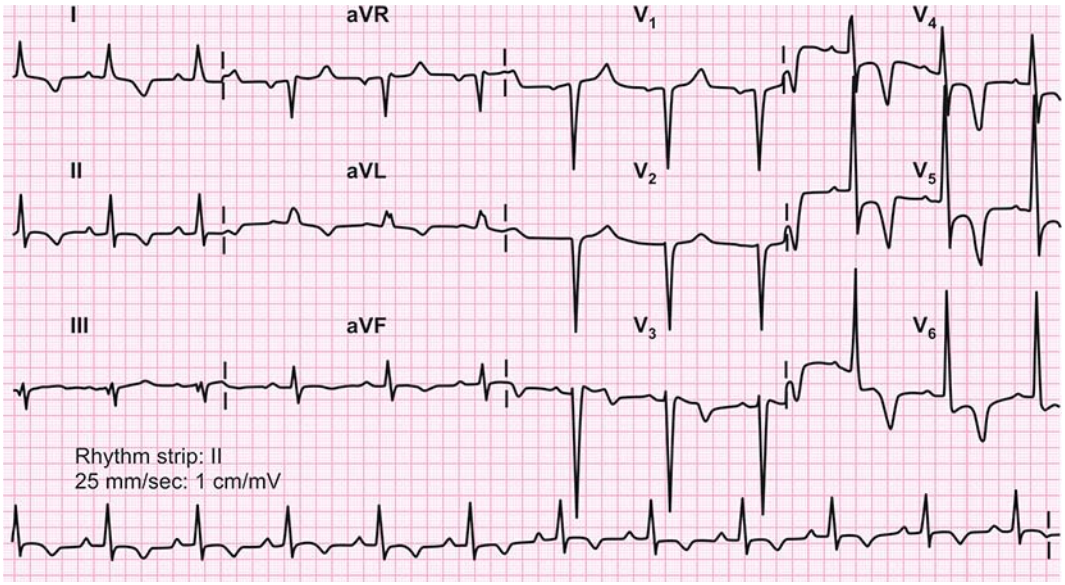
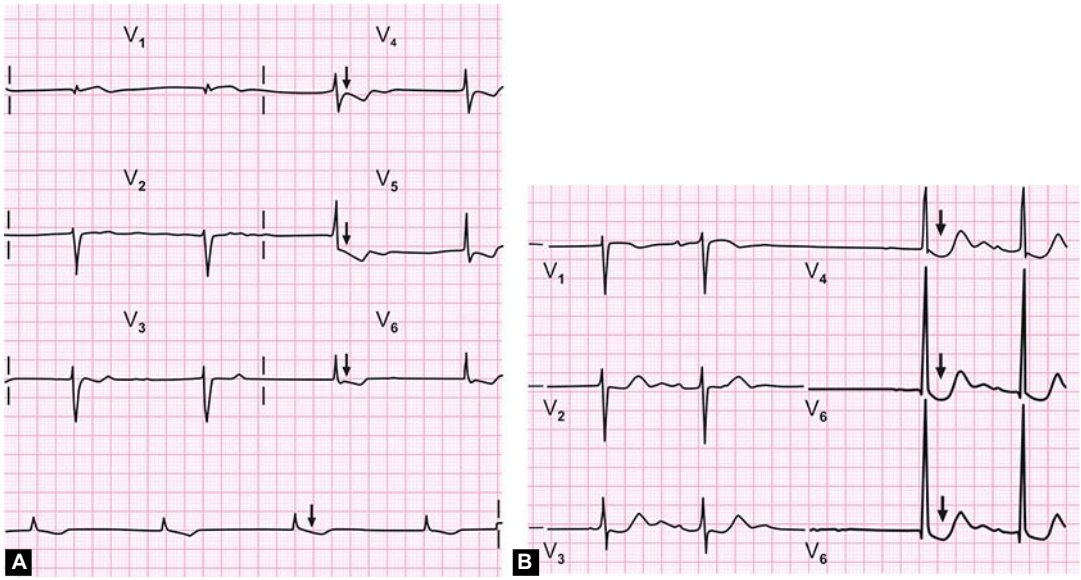


Figure 1.65: ST depression in case of LVH (S wave in lead V_2+R wave in lead V_5>35 mm, see later for criteria). Note flat ST depression in lead V_4–V_6



Figures 1.66A and B: Note reversed check sign ST depression (A-arrow) and scooping ST depression (B-arrow) in digitalis use

QT Interval

The interval between the on sets of ventricular depolarization to the end of the ventricular repolarization (T wave). If prolonged, this represents delayed repolarization of ventricles. The danger in prolonged QT is that a VPC is likely to fall on the vulnerable zone of T wave (just ahead of peak of T wave and can cause torsade de pointes (see diagram of QRS and cardiac action potential) (*see* Fig. 1.25).

How to Diagnose

Several formulas (like Bazett's Formula, Fridericia's formula and currently more reliable Sagie et al. formula, etc.) and tables (Ashman and Hull) has been derived for corrected QT interval QTc (QT interval decreases with increased heart rates.) A rough guideline is that normal QT is less than half of the proceeding PR interval. Do not confuse! A simple chart is also given. In clinical practice, QT should be assessed mainly for excessive prolongation (Table 1.7).

Table 1.7: Simple approximation of heart rates and QT interval

Clinically useful approximation of upper limit of QT interval		
ECG paper speed 25 mm/sec		
Heart rate	**Male**	**Female**
– 45–65 bpm	< 0.47 sec	< 0.48 sec
– 66–100 bpm	< 0.41 sec	< 0.43 sec
> 100 bpm	< 0.36 sec	< 0.37 sec

Table 1.8: Causes of prolonged QT interval

Long QT interval cause

Acquired	Inherited
Drugs: – Class I antiarrhythmics (quinidine, procainamide, etc.) – class III antiarrhythmics (amiodarone, sotalal, etc,) – TCA (tricyclic antidepressants) **CNS:** – Cerebrovascular diseases [stroke, (SAH) subarachnoid hemorrhage] – Ischemic heart disease (myocarditis, rheumatic fever, etc.) **Electrolyte:** – Hypocalcemia **Endocrine:** – Hypothyroidism **Cardiac:** – Myocarditis – Rehumatic fever	– Congenital long QT syndrome – Jewell and Lange–Nielsen syndrome – Romano–Ward syndrome

How to Deal with Long QT?

If QT is prolonged, rule out toxic metabolic causes (Table 1.8) if no apparent cause is found, a careful family history for syncope or sudden death should be sought to rule out hereditary cause (Figs 1.67 to 1.69).

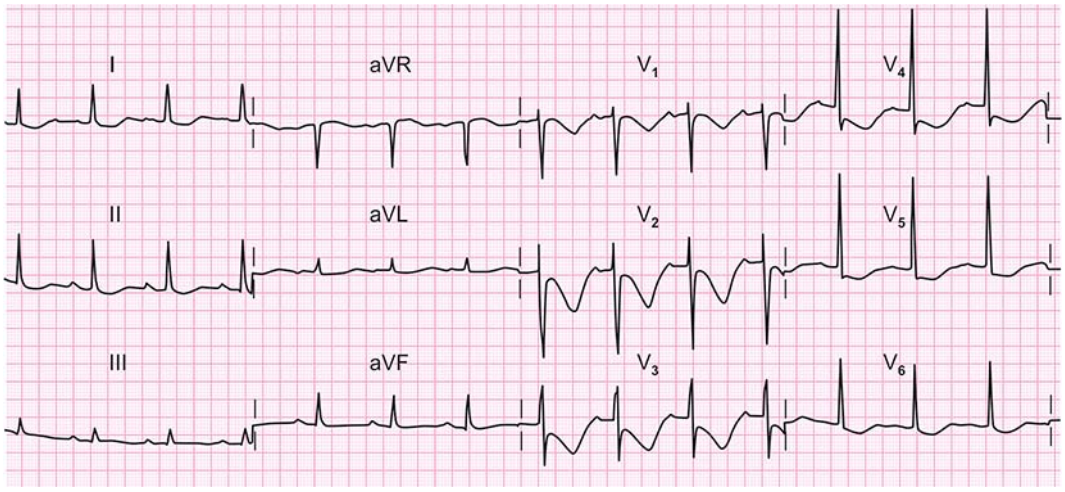


Figure 1.67: Long QT (0.50 sec) for heart rate 85 bpm. This lead also shows S wave in lead V_2+R wave in lead V_5>35 mm which indicate LVH by voltage criteria

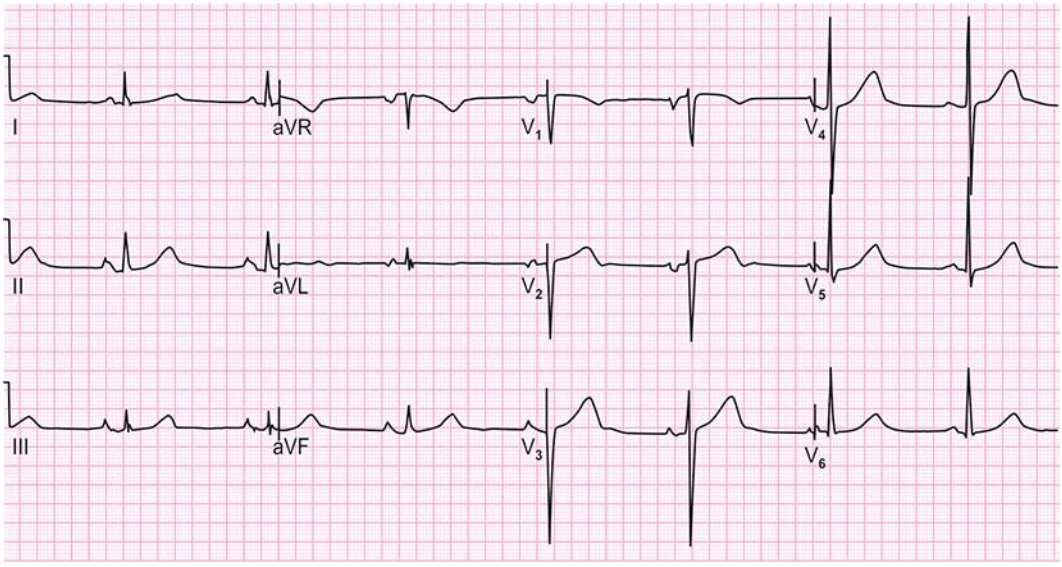


Figure 1.68: Prolonged QT (0.54 sec) with heart rate in case of bradycardia (This ECG also shows prominent negative P wave in V_1 which indicates left atrial abnormality)

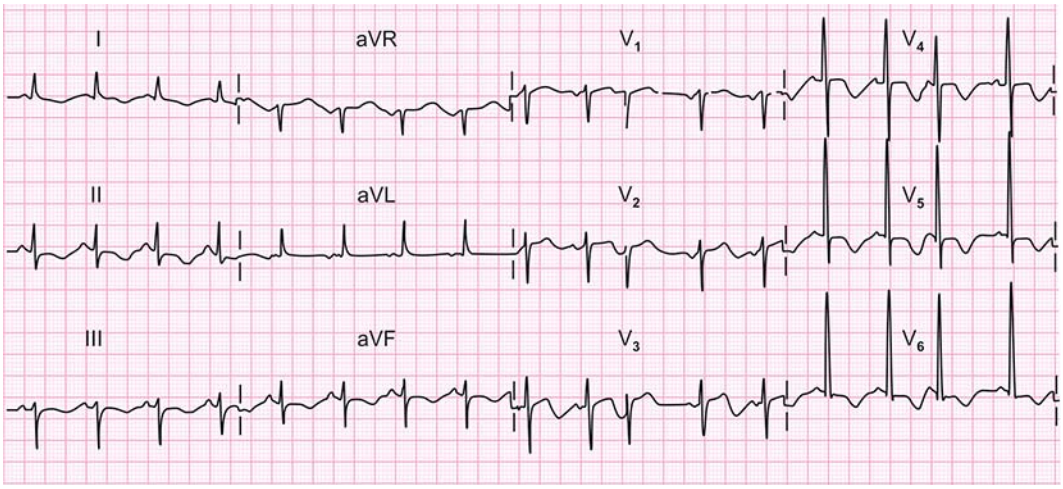


Figure 1.69: Prolonged QT (0.40 sec) interval with heart rate greater than 100 bpm. This lead also shows ST depression and T wave inversion in leads V_4–V_6

HANDSHAKE WITH "ELECTRICAL AXIS"

Many authors stress on electrical axis too much and start with discussing axis (right axis deviation (RAD), left axis deviation (LAD) and fascicular blocks, etc.) which make ECG unpalatable. For diagnosing purpose determination of axis is mainly supporting and for students these are not too many (fortunately), e.g.

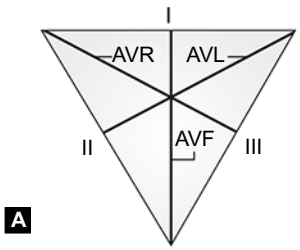
- Right ventricular hypertrophy (RVH). Here the presence of right axis deviation (RAD) is supporting (Note left axis deviation (LAD) is not necessary for diagnosis of LVH).
- Ventricular tachycardia (VT). Here usually left axis deviation (LAD) or sometimes extreme RAD (no man's land) is present. Rarely RAD can be present.

And what about the fascicular blocks? I prefer to consider left anterior fascicular block (LAFB) an especial case of LAD, while consider left posterior fascicular block (LPFB) an especial case of RAD. See Quick Diagnostic Section!

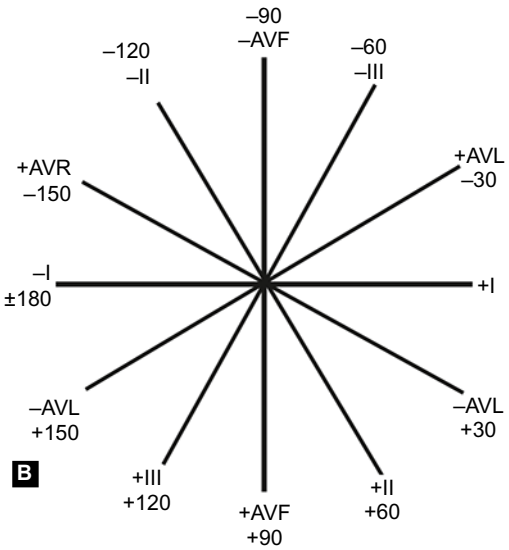
What Does Electric Axis Mean?

As we know, heart produces electric forces. These forces are depolarization forces and repolarization forces. The major direction of these forces forms the mean axis (for example, direction of depolarization forces in atria forms mean P axis, direction of depolarization forces in ventricles forms mean QRS axis. Similarly, direction of repolarization forces in ventricle (mean ST axis, mean T axis) to gauge these forces, they are

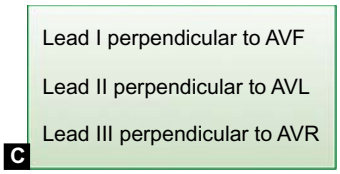
expressed mathematically as vector in a hexaxial reference system. This hexaxial reference system is nothing but just the six-limb leads represented graphically (Figs 1.70A to C). Note that this system sees heart in the frontal plane (Chest leads see heart in horizontal plane).



Unipolar limb leads added to the equilateral triangle (Einthoven's triangle formed by lead I, II, II)



Hexaxial reference system derived from A



Figures 1.70A to C: Formation of hexaxial reference system (A and B). Note some leads are perpendicular to other leads (C)

Note above figure of hexaxial system of limb leads (Fig. 1.70B) shows positive value represent the positive end of axis leads, while the vector of axis lead may point to positive end or negative end depending whether QRS negative or positive.

Figure 1.70C shows wave perpendicular to each other. Since left ventricular mass is much more than the right ventricle, the major direction of ventricular forces (mean QRS axis) is directed toward the left ventricle (toward left side of body downward posteriorly) and in frontal plane left downward (Fig. 1.71).

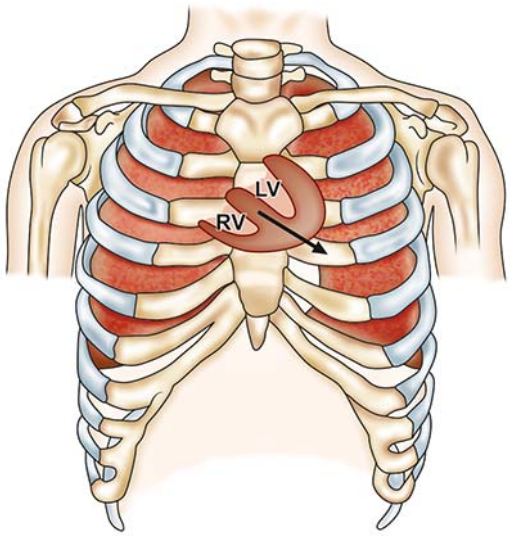


Figure 1.71: Mean QRS vector

Recall from your school mathematics if ventricular force is expressed in form of vector it will have a head and tail and length of tail shows magnitude. Note that it is not necessary that this vector always points to the end of lead in hexaxial system. Simply if QRS direction is positive (upward) in ECG (let's say for lead I) vector point the positive end of lead I, if QRS is negative (downward) in vector points toward negative end of lead I and if QRS is almost equally biphasic, i.e. equiphasic the vector points to perpendicular (right angle) lead either positive or negative (Fig. 1.72).

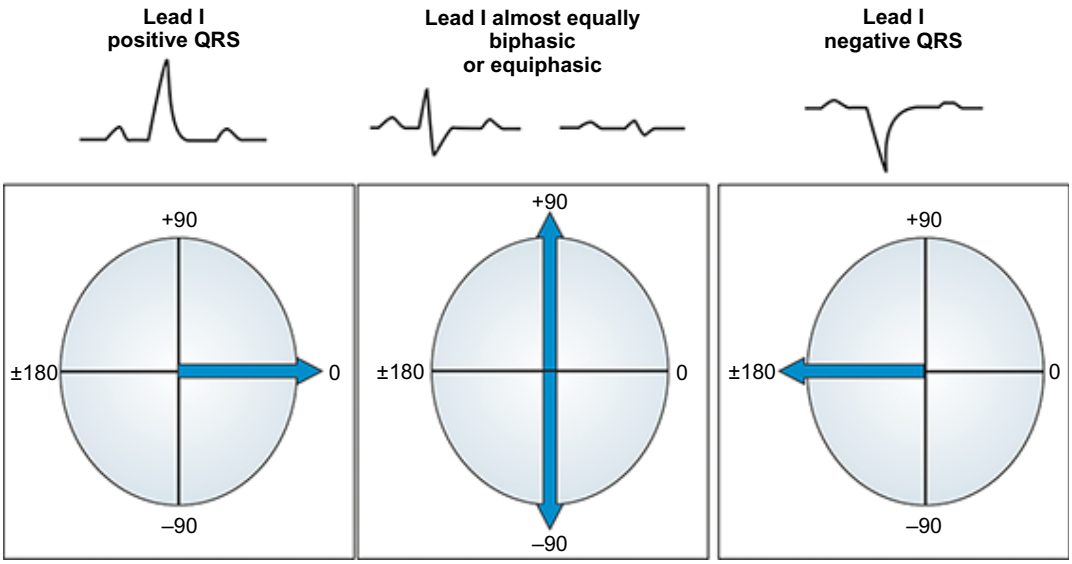


Figure 1.72: Relationship of QRS shape and direction of its vector. Note positive QRS points toward positive end of lead parallel to it, negative QRS points toward negative end of lead parallel to it but equally biphasic or equiphasic QRS points to lead perpendicular to it (for lead I is aVF) either positive or negative

Note in hexaxial system normal axis is left downward identified in ECG by QRS in lead I, aVF directed upward (positive polarity) for memorization remember as "Both Thumbs Up is normal"); It is between 0–110 (younger than 40 years) and between –30 to +90 (older than 40 years). Axis anywhere else is said to be deviated. Axis between –30 to –90 (QRS upward in lead I but downward in lead aVF) is called as left axis deviation (LAD). Axis between +90 to +180/ –180 is called right axis deviation (RAD); here QRS is negative in lead I while QRS in aVF is not necessarily to be checked because of minimal significance of its direction (Table 1.9)

Table 1.9: Relationship of QRS polarity and axis. QRS direction can be memorized by thumb method, e.g. both thumbs up (QRS in lead I and aVF both upward) indicate normal axis and vice-versa

Axis	Degree	QRS in Lead I	QRS in Lead aVF
Normal	0 to +110 (< 40 years) – 30 to +90 (> 40 years)	↑	↑
Left axis deviation [LAD]	– 30 to – 90	↑	↓
Right axis deviation [RAD]	+ 110 to + 180	↓	Not necessary

Now Jump to Simple Two-Step Method

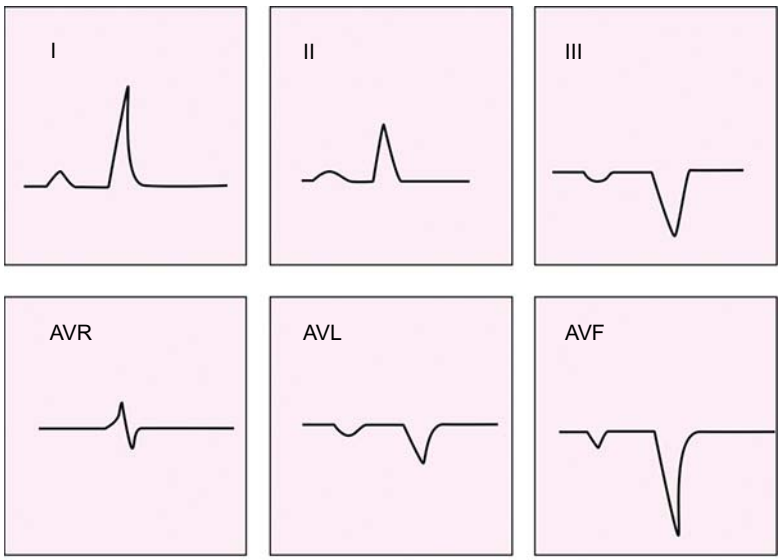
Step I

Keeping above given concepts in mind and specially focus on figures and table given above. First, find axis main direction by QRS position in lead I and AVF (thumb method).

Step II

Now to locate more precisely, find the almost equally positive negative (equiphasic) QRS. The direction is "perpendicular" to this equiphasic QRS and if the QRS position in perpendicular lead is positive, the vector points toward axis lead's positive end.

(Remember, value of positive end may be given negative) and if QRS is negative the vector points towards negative end of axis lead (See illustrated example in Figure 1.73).



Main axis = LAD (I is upward and aVF is downward)
Equiphasic QRS = aVR
Perpendicular lead to aVR = II

Figure 1.73: Simple method of finding axis. First find main axis by checking QRS polarity in lead I and aVF (LAD here) then look for equiphasic QRS (here aVR) then find perpendicular lead to the smallest or isoelectric lead which is lead III. Since here QRS in lead III is negative therefore vector points toward negative end of lead III which is –60; hence the axis is LAD ≈ –60 degree

Visual Impression Method

However, if you find this method little difficult be relaxed there is even simpler method.

This is called as visual impression method, memorize this simple figure to know axis roughly (Fig. 1.74).

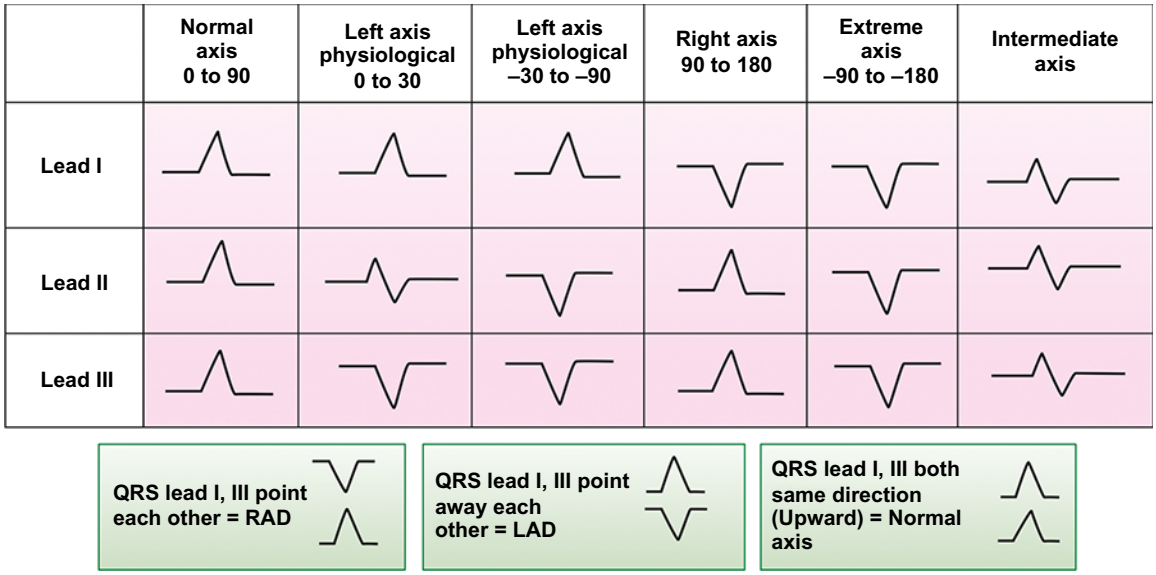


Figure 1.74: Visual impression method. Considering QRS polarity of QRS in leads I, II and III gives impression of axis

Now check Figures 1.75 to 1.77 and do practice.

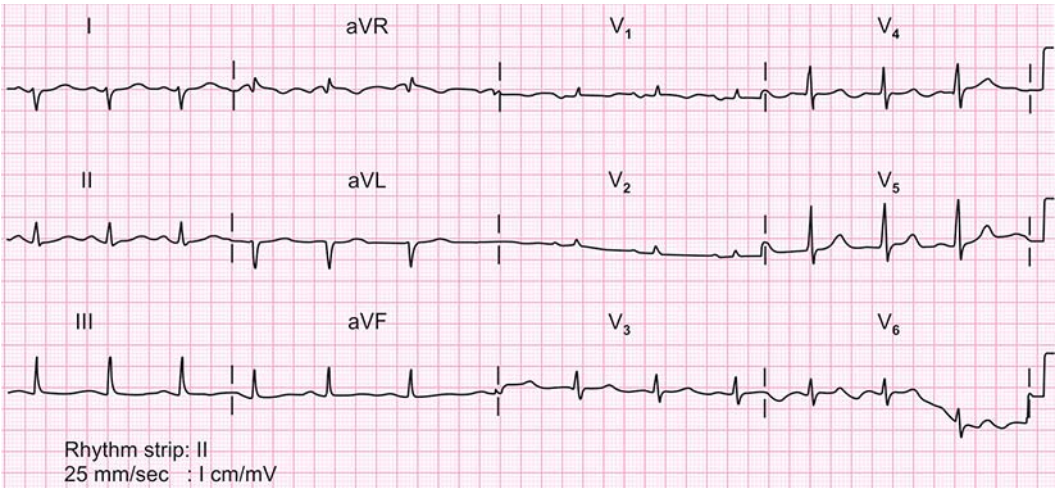


Figure 1.75: ECG from a patient with severe primary pulmonary hypertension with RVH. Note marked right axis deviation (QRS lead I downward and lead aVF upward) with prominent R forces in right precordial leads

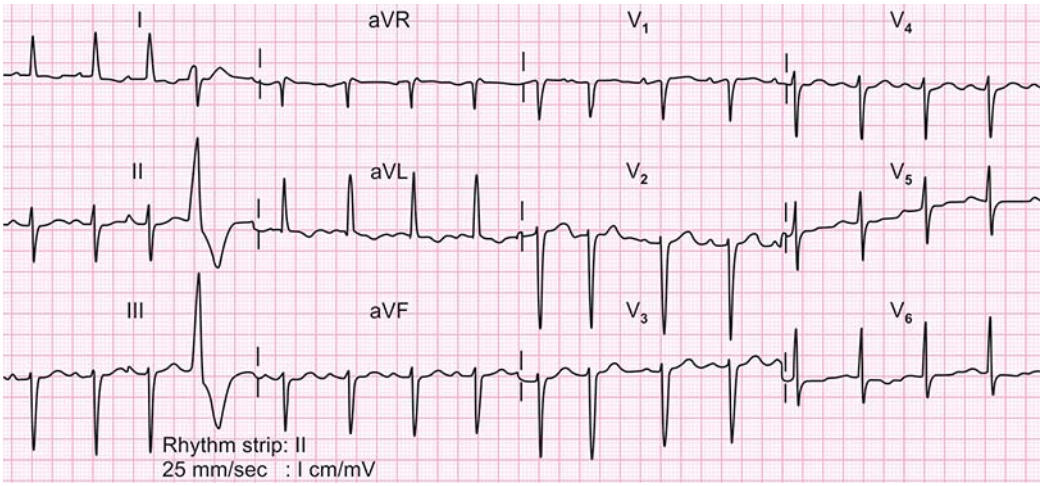


Figure 1.76: QRS in lead I upward and in aVF downward shows LAD in this lead

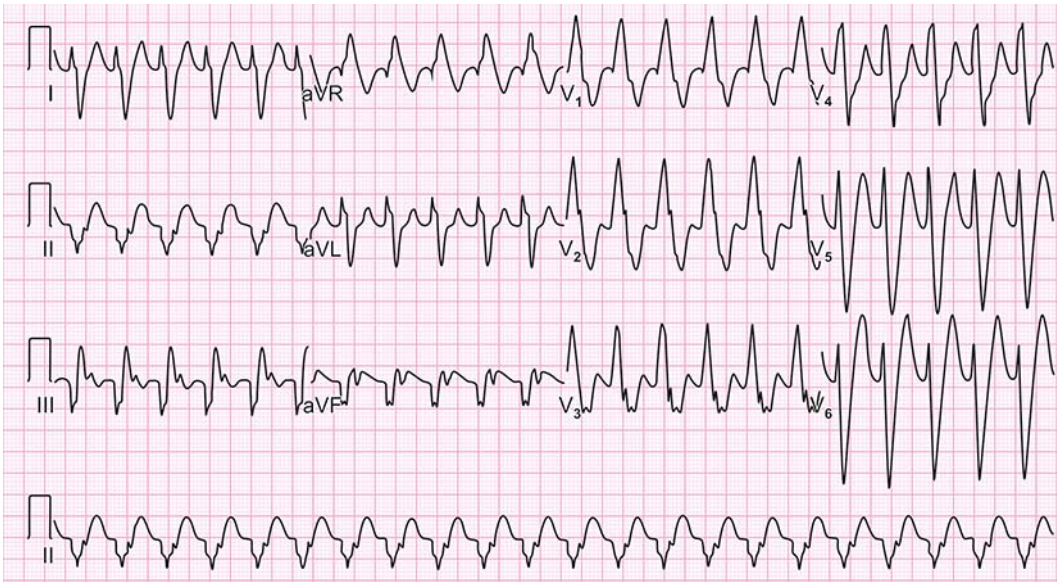


Figure 1.77: Note both downward deflected QRS in lead I and aVF indicates extreme axis deviation (no man's land). In the case of ventricular tachycardia (as in this ECG) is an important feature for diagnosis

Section 2:
Quick Diagnosis Section (QDS)

QUICK DIAGNOSIS SECTION (QDS)

This section will present diagnosis in the forms of diagnostic points and supporting points so that reader can quickly make diagnosis rather than searching from rich text.

These points are divided into two categories. Diagnostic points which are more specific to diagnosis and supporting points which support the diagnosis.

HOW TO READ ECG AND MAKE DIAGNOSIS?

Five-Finger Method

Five-finger method is easy method which enables to gauge ECG from every aspect. Initially, it looks a little long but after sometime, it will become your habit to look upon every point of ECG and will be much quick. And do not miss any diagnosis because usually single ECG has more than one diagnosis (Fig. 2.1).

The three segments of every finger represent features of ECG. So total 15 features to be noted. So let's begin.

These features are:

1. Check lead positions and exclude dextrocardia.
2. Rhythm
3. Rate
4. QRS interval
5. PR interval
6. Exclude other conditions including 'WPW' syndrome (specially if QRS duration is >0.12 sec.
7. ST segment

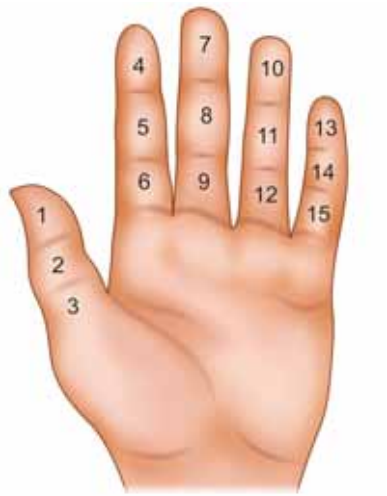


Figure 2.1: Fifteen-finger segments to remember 15 features of ECG (See text)

8. Q wave
9. R wave progression
10. P wave
11. T wave
12. Axis and block (bundle branch blocks and fascicular blocks)
13. Hypertrophy
14. Miscellaneous conditions (See later)
15. Detailed arrhythmia analyses

Step 1: *Check lead positions and exclude dextrocardia.*
It is wise to look for and rule out possible technical errors in placing leads. See details later.

Step 2: *Rhythm*
Check for sinus rhythm; if not check for arrhythmia (jump step 15)

Step 3: *Rate*
Calculate heart rate.

Step 4: *QRS interval*
Assess QRS both in lead V_1 and V_6 for interval and shape in order to diagnose bundle branch blocks, if > 0.12 sec BBB (complete or incomplete) may be present. For shape, look for M or W shape pattern.
(For diagnosis criteria see later).

Step 5: *PR interval*
Assess PR interval, if abnormal (>0.2 sec). Check for AV blocks (see later portion) if less (<0.12) check for delta wave and WPW syndrome.

Step 6: *Exclude other conditions including WPW syndrome if QRS > 0.12 sec*
According to some authors, it is wise after QRS and PR analysis, exclude non-specific causes of intra-ventricular conduction delay, WPW syndrome, electrical pacing and Brugada syndrome, etc. (See diagnostic points later). These conditions are rare but often missed and causes death.

Step 7: *ST segment*
Assess ST segment for abnormality, i.e. depression or elevation (See Tables 1.4 and 1.5).
Look changes suggesting Infarction, Ischemia, etc.

Step 8: *Q wave*
Then look every lead for Q wave for MI diagnosis and determining its age as acute, intermediate or chronic.

Step 9: *R wave progression*
To identify anterior, posterior infarction and BBB or other condition assess R wave in V_1–V_6 and also its progression. Also check whether normally progressing R wave suddenly disappears or not.

Step 10: *P wave*
Assess P wave for its shape, look every P wave is followed by QRS or not, P wave inversion. This will also help in diagnosing hypertrophy of left and right atrium, right atrial hypertrophy, left atrial hypertrophy and arrhythmia. Pay special stress on lead II and V_1.

Step 11: *T wave*
Assess T wave for inversion, its amplitude (tall, flat, etc.) for making some diagnosis (like post infarction, hypercalcemia, hypokalemia, etc.) and strengthening diagnosis of MI and Ischemia (*See* Tables 1.3 and 1.4).

Step 12: *Axis and Block (bundle branch blocks and fascicular blocks)*
Check axis by simple 2-step method and check for left anterior, left posterior fasicular block/hemi block.

Step 13: *RVH, LVH check*
Criteria for diagnosis RVH and LVH are presented in later discussion.

Step 14: *Miscellaneous condition*
Check for miscellaneous condition like electrolyte imbalance (hypokalemia, hyperkalemia, hypercalcemia, etc.), cardiac pathologies (pulmonary embolism, ASD, long QT syndrome) drug effects like digitalis and electrical pacing, etc.

Step 15: *Detailed arrhythmia analyses*
If sinus rhythm is not found, detailed scrutiny for arrhythmia is mandatory and this step should be considered in step 2 instead.

Although, this list took long but by finger counting method, you will easily pick these and can't miss, for example; when you are on step 4 on ST segment an elevated ST segment (with concavity upward) in most leads will recall you to think pericarditis!!

LEAD POSITION REVERSAL

Most common error in lead placement is reversal from right arm to left arm. Therefore, it is wise before embarking on ECG just look that ECG is technically OK or not (it is easy).

Diagnostic Points

1. Lead I shows negative QRS while lead aVR shows positive QRS (normally it is negative).
2. Lead aVL and aVR transposed (therefore now lead aVL is negative instead of aVR).
3. Lead II and III are transposed (Fig. 2.2).
4. Limb leads aVF and V_1–V_6 are not affected (this differentiates from dextrocardia where precordial leads are also reversed) [see Dextrocardia].

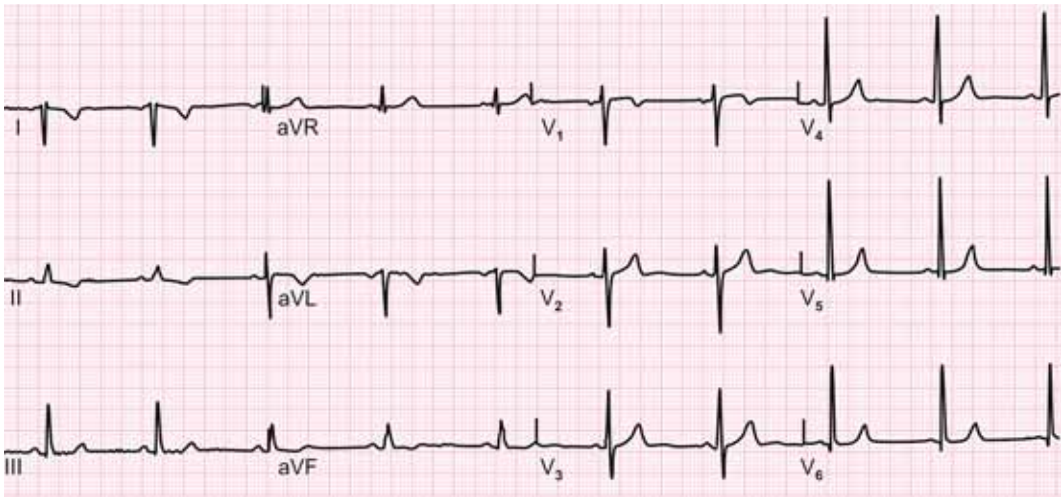


Figure 2.2: Incorrect lead placement with a right to left arm reversal. Note lead I is negative while aVR is surprisingly positive. In fact aVR and aVl are reversed. Similarly II and III are reversed, importantly also note that precordial leads are unchanged which is not the case in dextrocardia where both limb leads and precordial both have reversed configuration

DETERMINE RATE/RHYTHM

Rate

Heart rate >100 beats per minute (bpm) = tachycardia; Heart rate <60 bpm = bradycardia.

a. Determination of rate is not difficult. Note these rates are counted when ECG paper speed setting is 25 mm/sec which is used universally. Count number of large boxes between two consecutive R waves. Number of large boxes show rate. For convenience choose R wave which falls on the bold line of ECG strip (Table 2.1).

b. If rate is high enough that only one or less than one large box is there between two R waves, the number of small box also corresponds to the rate as in Table 2.1.

c. If rate is irregular, it is better to count QRS complexes in one minute interval. One large box equals to 0.2 sec, 5 large boxes equal to 1 sec. So count QRS in 6 seconds then multiply it by 10 to get rate in one minute (6 second × 10=1 minute). For any condition rate is: rate = 1500 divided by number of small squares between two RR intervals (Fig. 2.3).

Table 2.1: Number of large boxes and small boxes between two consecutive R waves and approximate heart rates

Number of large boxes	*Rates bpm*	*Number of small boxes*	*Rates bpm*
1	300	2	750
2	150	3	500
3	100	4	375
4	75	5	300
5	60	6	250
6	50	7	214
7	42	8	107
8	30	9	166
9	33		

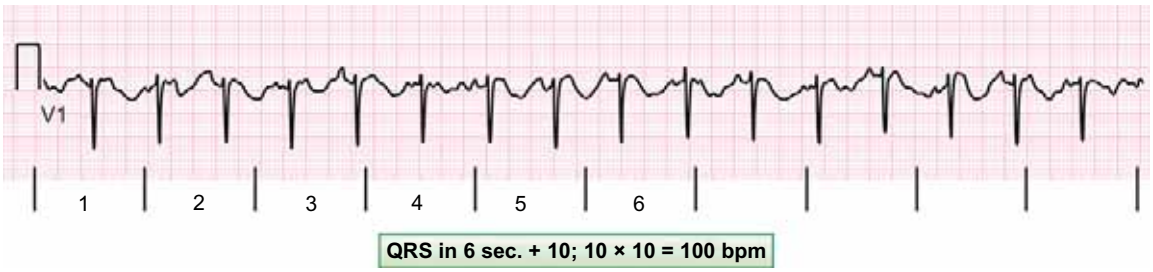


Figure 2.3: Showing determination of heart rate by calculating QRS complexes in 6 seconds strip

Rhythm

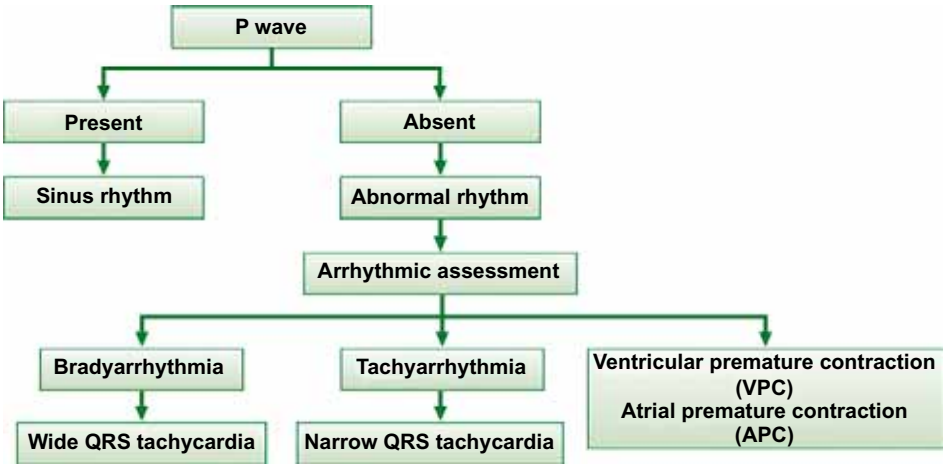
This step may be simple of course if rhythm is regular and derived by SA (sinoatrial) node called sinus rhythm; (Table 2.2). Focus on V_1 and II for best visualization of P wave. Presence of sinus P wave means impulse generating from atrium and rhythm is regarded as sinus rhythm (recall that positive P wave in lead I and II and negative P wave in lead aVR indicates that impulse is generating from sinus node which is called as sinus P wave and rhythm is called as sinus rhythm. Retrograde or inverted P wave indicate its origin from sources other than sinus node and it is regarded as ectopic P wave. In the absence of sinus rhythm this step II becomes most important (irregular rhythm or arrhythmia). See step 15 of our five-finger method (note if arrhythmia is suspected this step should be considered at this stage, i.e. step 2) (See arrhythmia portion).

SINUS RHYTHM

Diagnostic Points (Fig. 2.4)

- The rate is within regular range (60–100 bpm).
- Every QRS is preceded by P wave and every P wave is followed by a P wave (which rules out AV block).
- P wave morphology and PR interval fairly constant.
- PP interval and RR interval also remains constant (equidistant) (Interestingly PP = RR).

Table 2.2: Assessment of P wave in ECG and its outcome



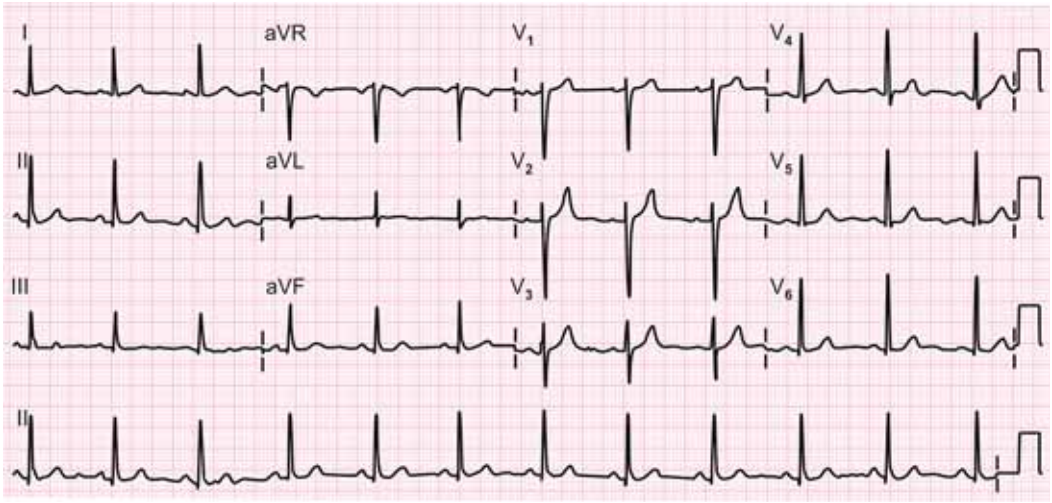


Figure 2.4: Normal sinus rhythm. Note presence of P waves and PP interval are equidistant and equal to RR interval

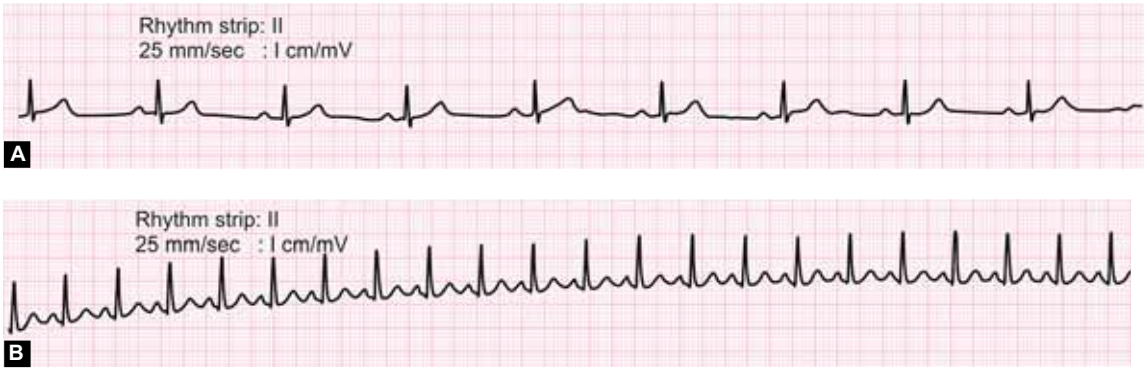
SINUS BRADYCARDIA

Diagnostic Points (Fig. 2.5A)

- Presence of sinus P wave before QRS complex if it is not sinus P wave it is escape rhythm.
- Sinus rate < 60 bpm
- Constant PR interval of normal duration
- P-P interval same to R-R interval and constant (may be slightly irregular).

SINUS TACHYCARDIA

Same above except rate >100 bpm (Fig. 2.5B).



Figures 2.5A and B: Example of sinus bradycardia around 50 bpm (A) and sinus tachycardia around 125 bpm (B)

DEXTROCARDIA

Dextrocardia with Situs inversus is congenital defect and is rare (1:10,000).

Diagnostic Points (*See* Fig. 2.2)

- Lead I, P, QRS, T waves are negative (downward).
- Lead aVR and aVL are interposed, i.e. aVR is positive and aVL is negative (so lead I + AVL both negative).
- R wave shows inverse progression, i.e. it is tallest in V_1 and decrease toward V_6.
  Note in right arm to left arm reversal chest leads are spared and show normal R wave progression.

ATRIOVENTRICULAR BLOCK (AV BLOCK)

Wiring diagram of the heart (below) shows current flow can be interrupted anywhere and this interruption manifests as blocks (AV block, bundle branch block) in ECG. Important blocks are discussed below (Fig. 2.6).

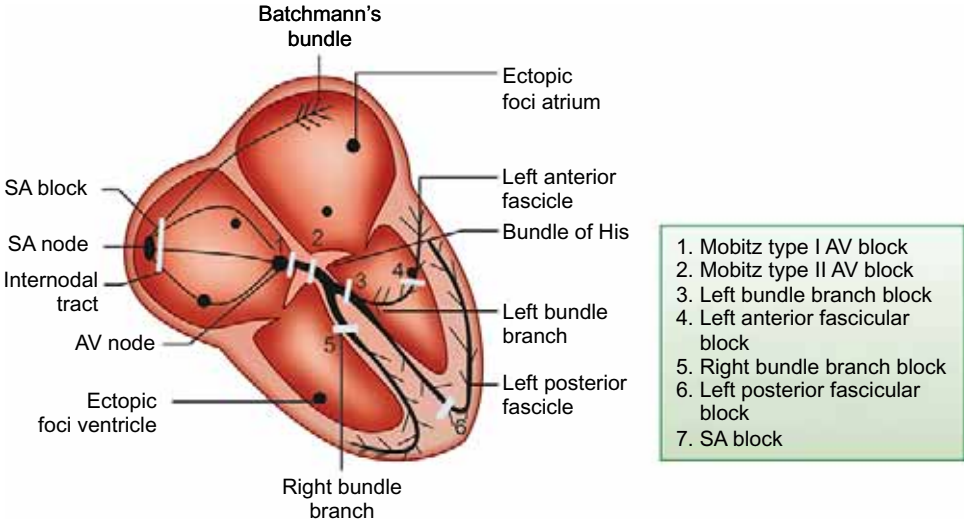


Figure 2.6: Wiring diagram of heart. Note interruption at different positions manifest as different type of blocks. Note that Mobitz type I is located higher almost always in AV node and Mobitz type II in lower position, usually in initial part of bundle branch and, therefore, QRS may be broaden or normal. Type II is more serious than type I

First Degree AV Block

Diagnostic Points (Figs 2.7 and 2.8)

- PR interval more than 0.2 sec (>5 small boxes).
- PR interval is constant.
- Every P is followed by QRS complex.

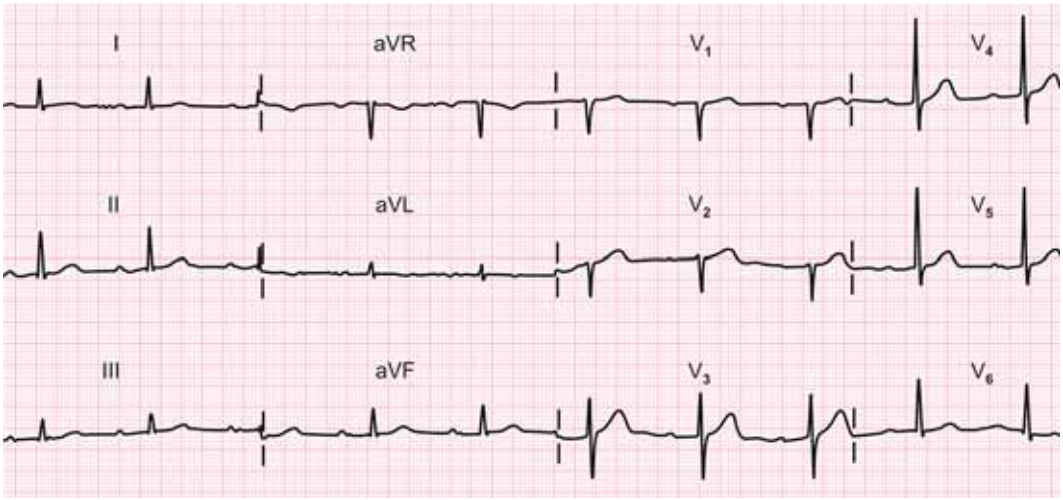
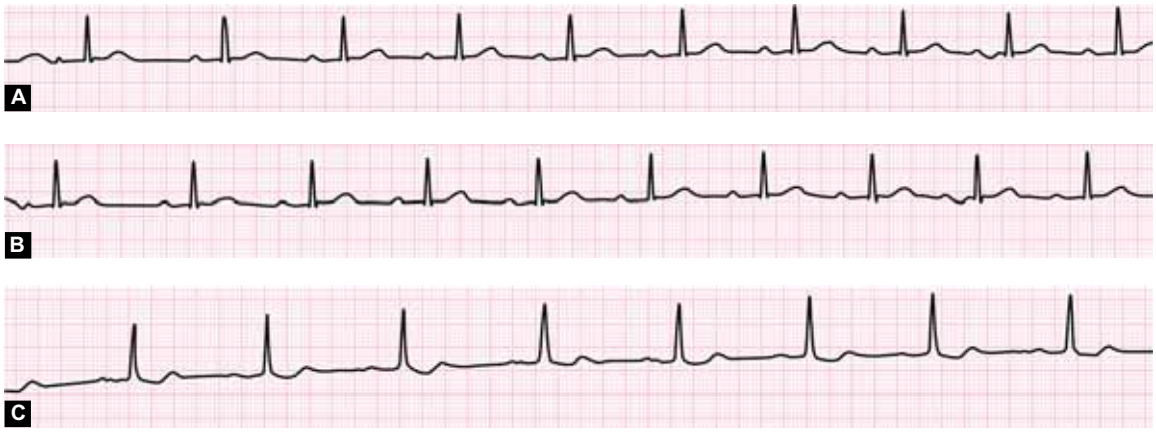


Figure 2.7: Sinus rhythm with first degree AV block. Note prolonged PR interval



Figures 2.8A to C: First degree AV block. Prolonged PR interval (greater than 0.12 sec)

Second Degree AV Block; Mobitz Type I (Wenckebach) Block

Diagnostic Points (Figs 2.9 to 2.11)

- Progressive increase in PR interval until impulse fails to conduct to ventricle and P wave is not followed by QRS (blocked P).
- Recovery after blocked P wave is always with short PR interval.
- The RR interval containing blocked P is less than sum of two consecutive RR interval before the dropped QRS. This is because after blocked P wave the next PR interval is always shorter.

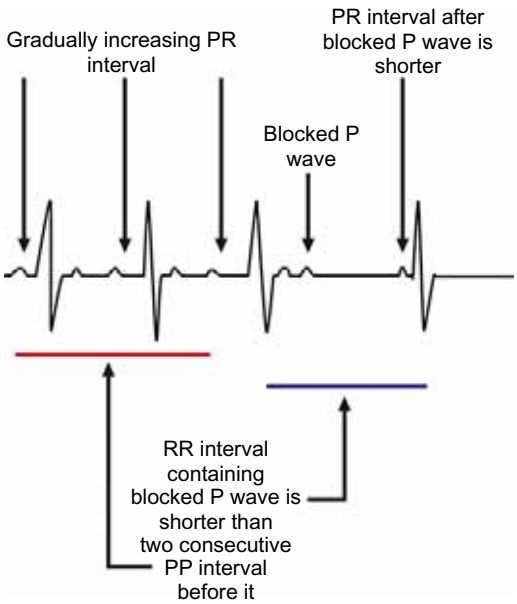
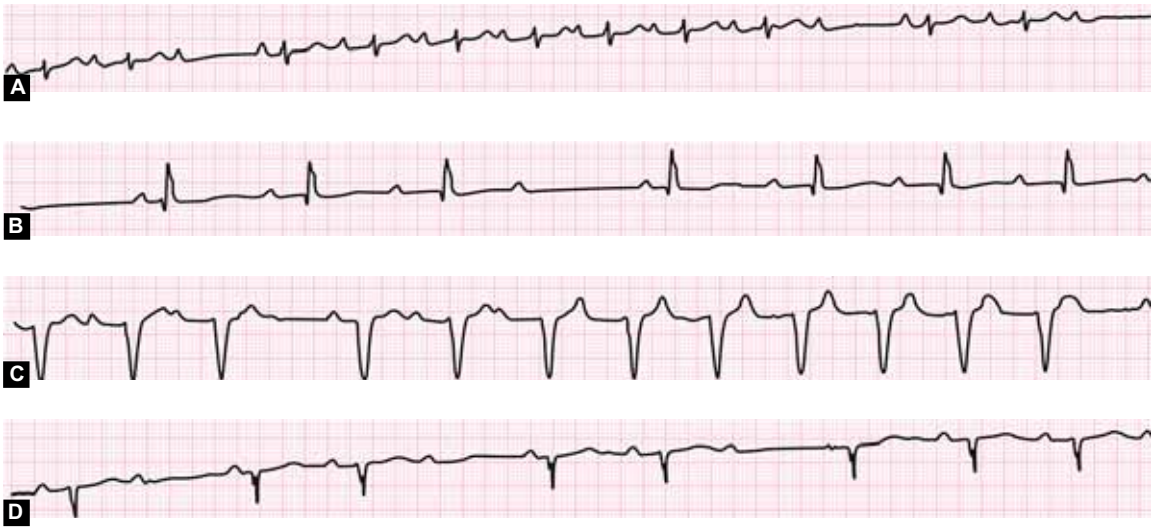


Figure 2.9: Showing second degree AV block. Also note that PP interval is constant

- PP interval is constant (this may be overlooked, but every P originates in constant time interval and it is QRS which is pushed toward next P wave so increased PR).
- QRS complex is usually narrow because block is high (if type II).

- RR interval gradually decreases (if present also typical for Wenckebach) but may be same or increases)
- Sometimes it presents with 2:1 block (pairs) in this situation we cannot differentiate whether there is gradual increase in the PR interval or not. Therefore, we cannot differentiate between type 1 and type 2.



Figures 2.10A to D: Different examples of second degree AV block Mobitz type 1. Note gradual prolongation of PR interval until P wave is blocked (failed to conduct)

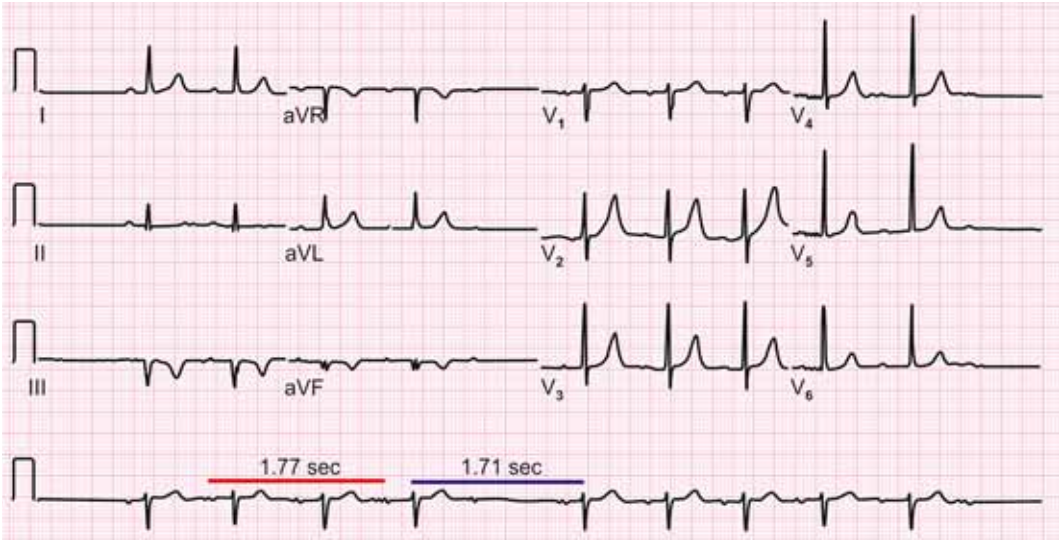


Figure 2.11: Second degree AV block Mobitz type 1. Note PR interval gradually increased until P wave is blocked (Wenckebach periodicity). Note after the blocked P wave PR interval is short. RR interval containing blocked P wave is smaller than the two consecutive PP interval. This ECG also shows inferior MI and posterior wall involvement

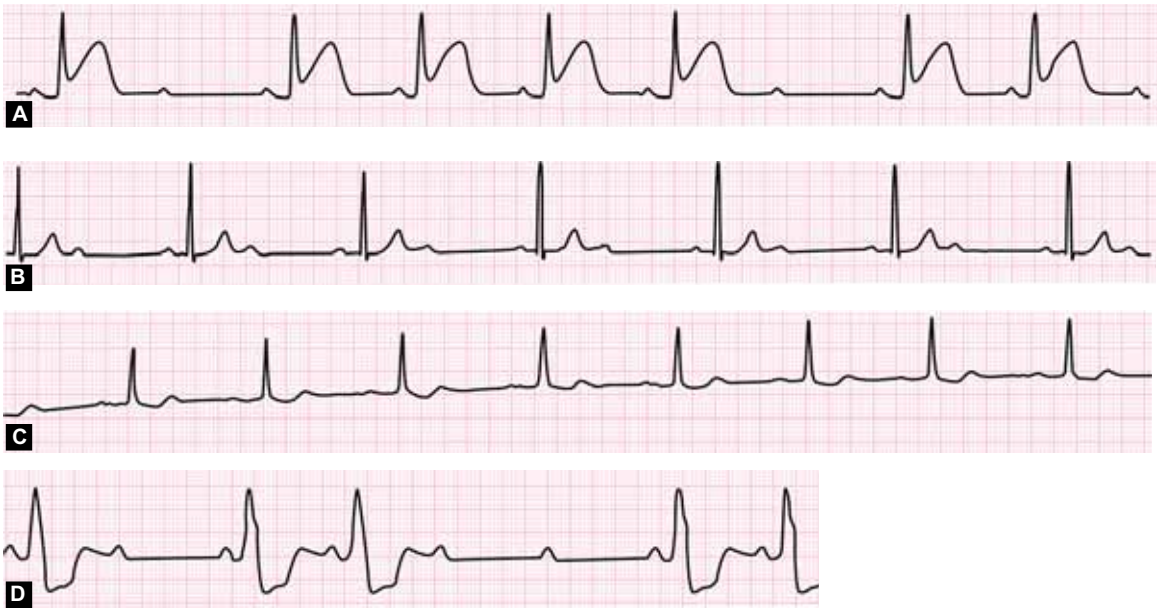
Second Degrees AV Block (Mobitz Type II) (Figs 2.12 and 2.13)

As described earlier here the site of block is lower than type I, i.e. in the initial portion of bundle branch

- At least two regular and consecutive atrial impulses are conducted with constant PR interval.
- A blocked (nonconducted P wave without QRS) sinus impulse.
- PR interval after the block is same as previous PR interval.
- Because of above fact, RR interval containing nonconducted P wave is equal to two RR interval (in other words, twice the heart rate compare; type I where it is shorter than the sum of two consecutive RR interval before the dropped QRS).

Supporting Points

- Since block is at the bundle of His or below it in the bundle branch QRS maybe > 0.12 sec (cf. type I where AV node is blocked) (Fig. 2.12D).
- High grade second degree AV blocks two or more consecutive P may be blocked.
- This is worse than type I Mobitz with bad prognosis with risk of catastrophic a systole = 36 percent approximately.
- If rate 2:1 you cannot surely diagnose it because you cannot check PR interval for gradual increase or constant.



Figures 2.12A to D: Different examples of second degree AV blocks Mobitz Type II. Note also PR interval before and after the dropped P wave is same

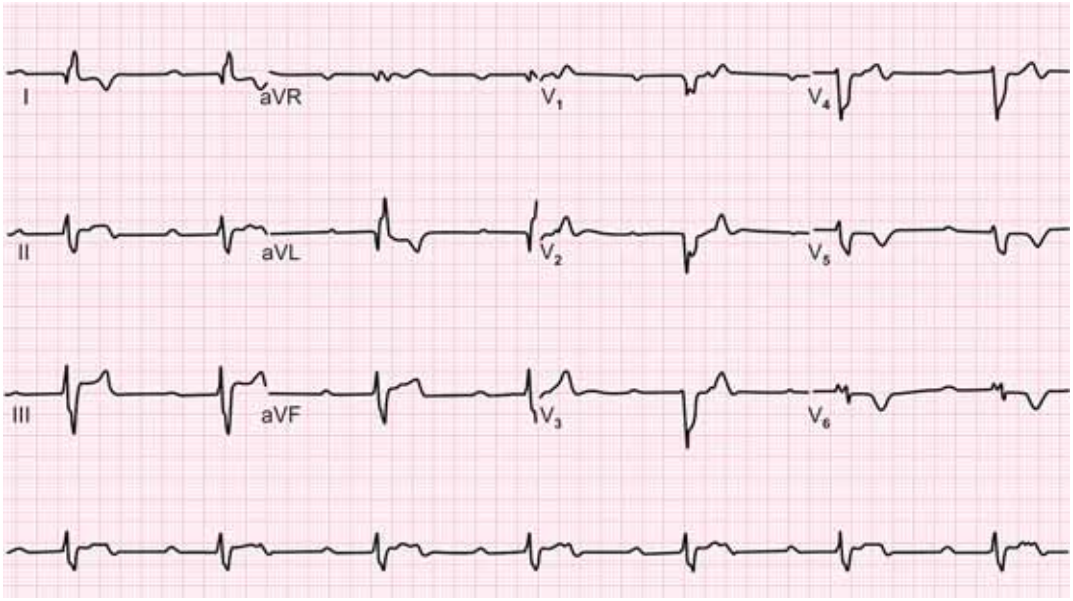


Figure 2.13: Second degree AV block Mobitz type II. 2:1 conduction, identical PR interval with one P wave blocked and broad QRS complex are the clues

Complete AV, or Third Degree Block

Diagnostic Points (Figs 2.14 and 2.15)

- AV conduction is completely blocked so atrial impulses do not deliver to ventricle and ventricle contracts its own. This is called AV dissociation, i.e. no relationship between atria and ventricle.
- Ventricle rate around 45/min (if impulse arise from AV junction rate may be higher).
- RR is constant. Note atrial rate > ventricular rate (this condition is reversed if AV dissociation is present in the absence of third degree AV block)

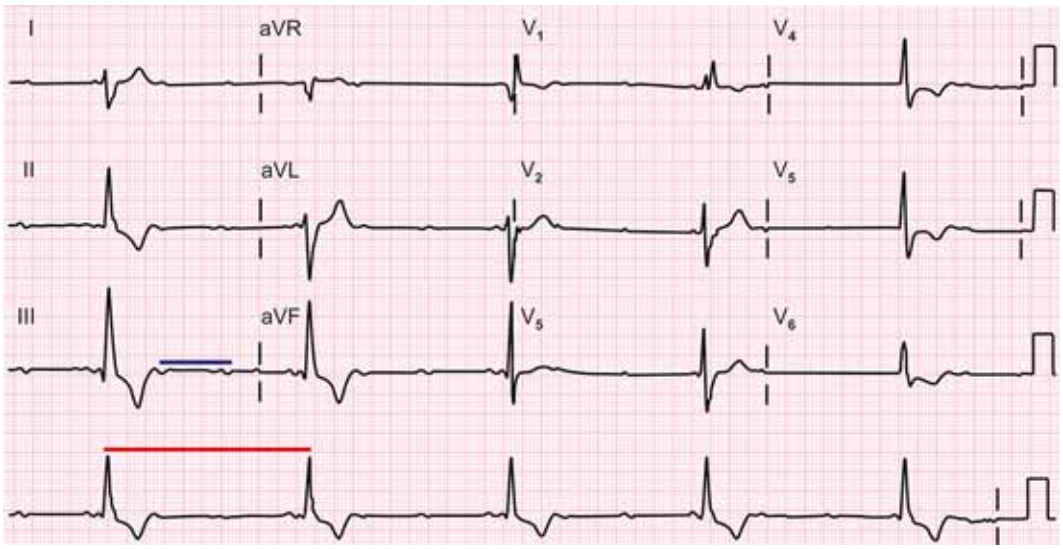


Figure 2.14: Complete third degrees AV block idioventricular AV escape rhythm 30 bpm. In AV dissociation (atrial rate (AR) >ventricular rate (VR) which differentiates it from other forms of AV dissociation where VR > AR. Also note RBBB morphology

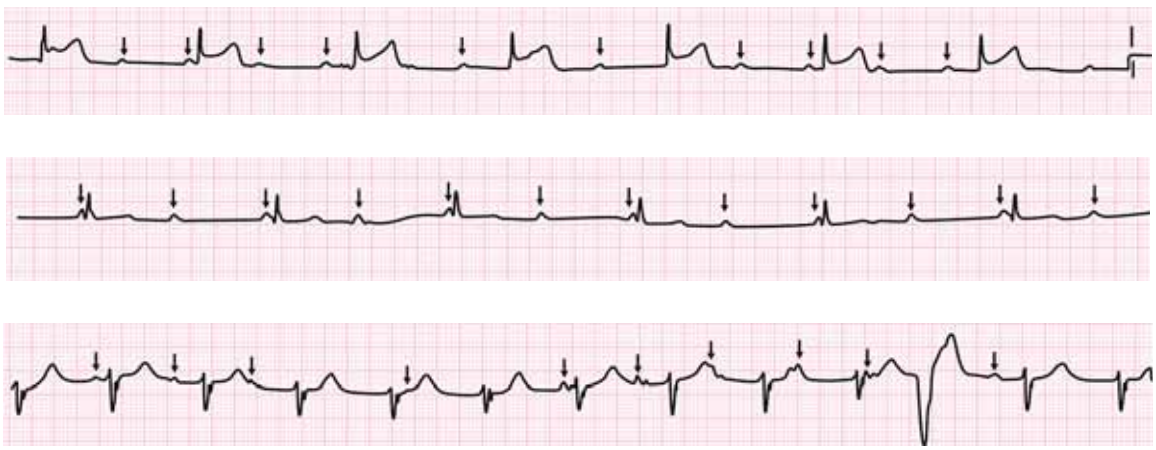


Figure 2.15: Third degree; complete AV block with junctional escape rhythm. Arrows show P wave, note AV dissociation

INTRODUCTION TO ELECTROCARIOGRAPHIC FEATURES OF MYOCARDIAL INFARCTION

Myocardial Infarction (MI)

Area suffering MI and leads showing changes depends on the coronary arteries involved (Fig. 2.16).

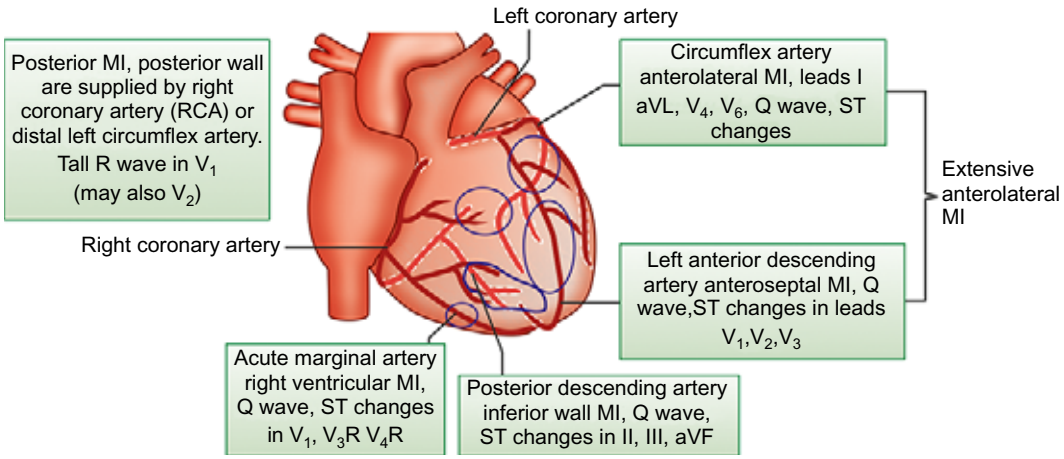
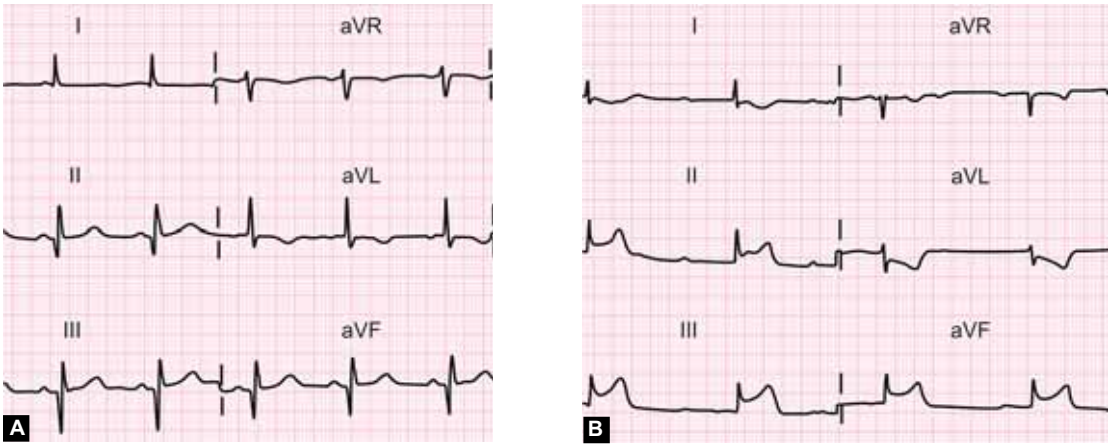


Figure 2.16: Schematic diagram showing coronary arteries areas infarcted and leads changes

ST Elevation Infarction v/s Non ST Elevation MI or Q Wave v/s Non Q Wave MI

The outer portion of cardiac wall, subepicardium (thicker wall) gets blood from major branches of coronary arteries. Coronary arteries obstruction results in infarction of entire wall, called transmural infarction and it manifests with ST elevation and Q wave formation, this is called ST elevation MI or Q wave MI (STEMI). Contrary to this the inner (thinner) layer subendocardial layer is supplied by smaller branches of the major coronary arteries, also this is farther from the main arteries, also during from the contraction, and there is more decrease in the blood supply. Therefore, subendocardium is more prone to infarction and here MI is sub-endocardial. This infraction does not show ST elevation and Q wave (hence called non ST elevation MI, NSTEMI, non Q wave MI) and may show pattern of ischemia (ST depression and T inversion) and need to be verified by checking levels of cardiac enzymes to differentiate from ischemia (troponin T, INI , CKMB, etc) (Figs 2.17A and B).



Figures 2.17A and B: STEMI and Q wave MI. (A) Shows prominent Q waves with ST elevation in leads II, III, aVF while (B) Shows prominent ST elevation in the same leads in the case of inferior wall MI

Inferior Wall MI

Diagnostic Points (Figs 2.18 and 2.19)

- ST segment elevation in leads II, III, aVF (acute MI).
- ST reciprocal ST segment depression in lead I, aVL strengthens diagnosis.
- Significant Q wave in II, III, AVF.
- In old MI, ST - T segments should be in same line.
- Area involved is inferior wall of LV + posterior part of intraventricular septum supplied by postdescending coronary arteries (80% cases from RCM, 20% LCX.)

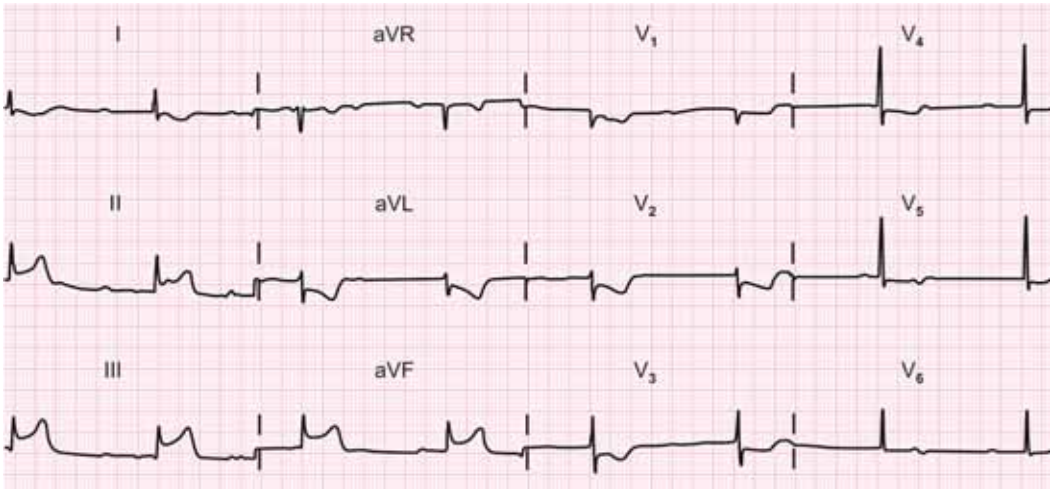


Figure 2.18: A patient with complete AV block and junctional escape rhythm. Note ST elevation in leads II, III, aVF showing acute inferior wall infarction. Leads I, aVL show ST depression with T wave inversion probably due to reciprocal changes

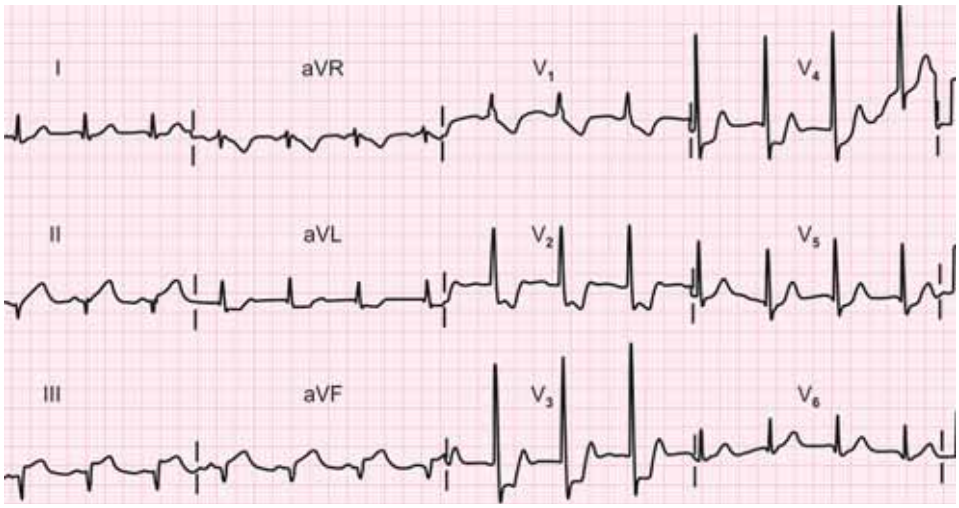


Figure 2.19: Inferior wall infarction of intermediate age. Note there are prominent Q waves along with ST elevation in leads II, III, aVF. Tall R wave with R wave greater than S wave in leads V_1V_2 indicating posterior wall infarction also. ST depression in leads I, aVL, V_1–V_6 shows reciprocal depression or lateral wall ischemia

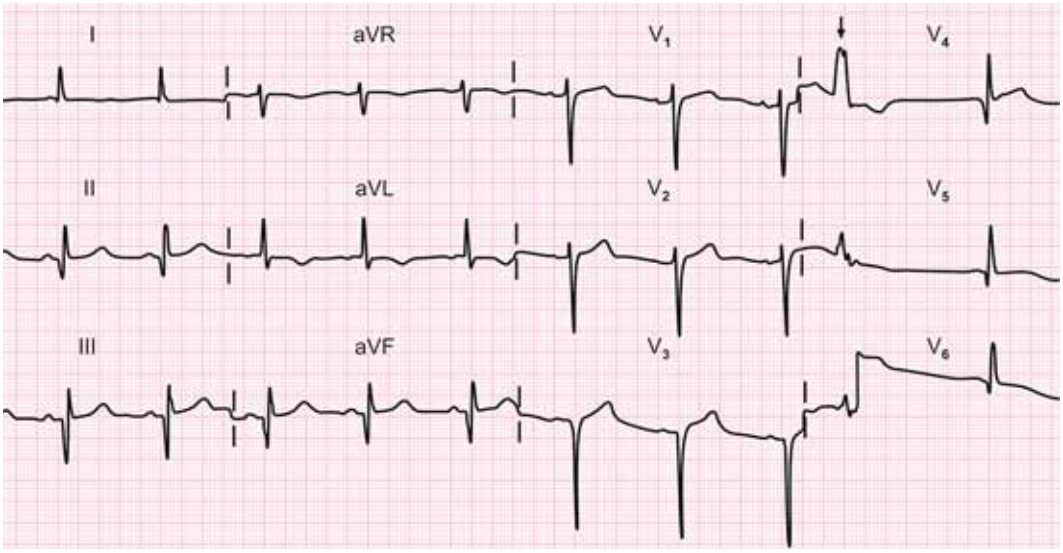


Figure 2.20: Note prominent Q waves with slight ST elevation inferior wall MI. Lead V_3 shows QS wave and Q waves V_4–V_6 suggestive of anterolateral MI also. Note R in lead aVL+S wave in lead V_3 greater than 20 mm in this woman suggest LVH (Arrow shows VPC)

Anterior Infarction

Depending upon the arteries supplying the specific parts of heart muscle, anterior MI is named anterolateral, anteroseptal or extensive anterior STEMI (Figs 2.20 to 2.22).

Anteroseptal or Anteroapical MI

Diagnostic Points

- ST elevation V_1–V_3 (acute)
- Q waves V_1–V_3
- Evolving Q waves + settling ST segment.

Supporting Points

- T wave decreases V_1–V_3.
- Reciprocal depression in other ECG leads.
- Poor R progression especially if R wave present in V_1 or V_2 and then disappears or becomes smaller in later leads.
  - The cause is blockade of LAD artery causing infarction of inferior or anterior septum + medial anterior wall of LV.

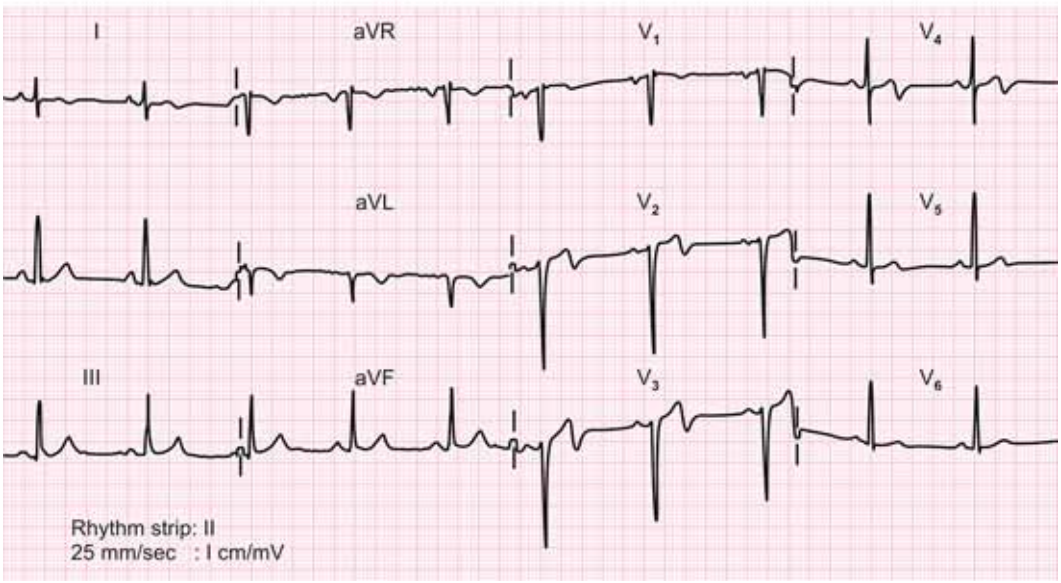


Figure 2.21: Acute anteroseptal MI. Note R wave is less than 3 mm and ST elevation in lead V_1–V_3

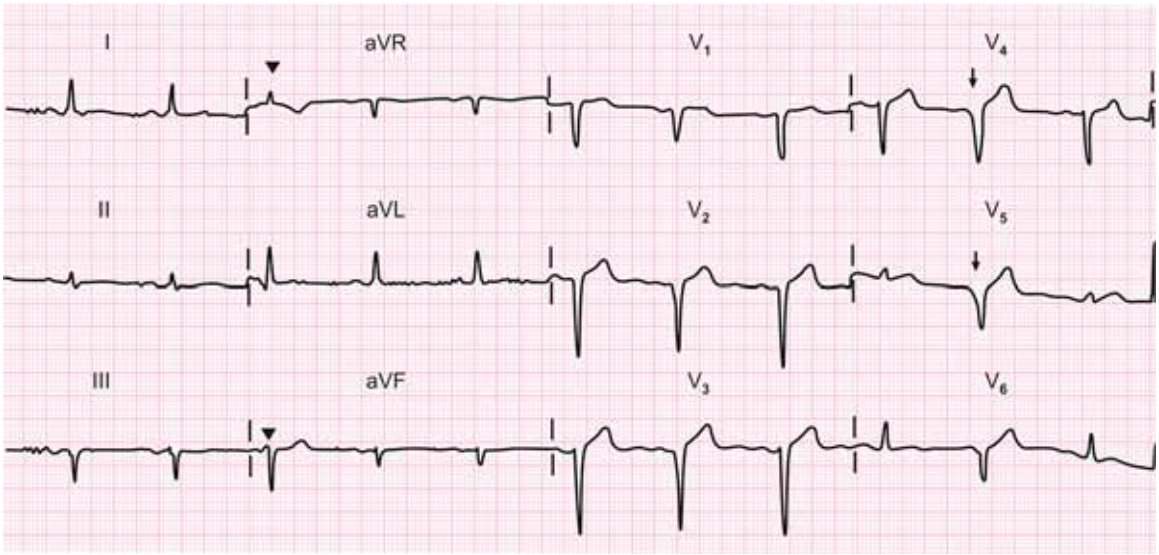


Figure 2.22: Note QS waves in V_1–V_2 and small R wave in V_3 in a case of old anteroseptal MI. This ECG also shows VPC (arrow) and fusion beat formed by fusion of supraventricular and ventricular premature conduction (arrowhead). Note fusion beats have variable morphology because variation in the extent of fusion

Lateral Wall MI

Diagnostic Points (Figs 2.23 and 2.24)

- ST elevation V_4–V_6 and or I, aVL (acute)
- Q waves in V_4–V_6 (old)
- Both Q wave and ST change in intermediate age MI.
  - There is blockade of LCX artery causing infarction of anterolateral part of LV.

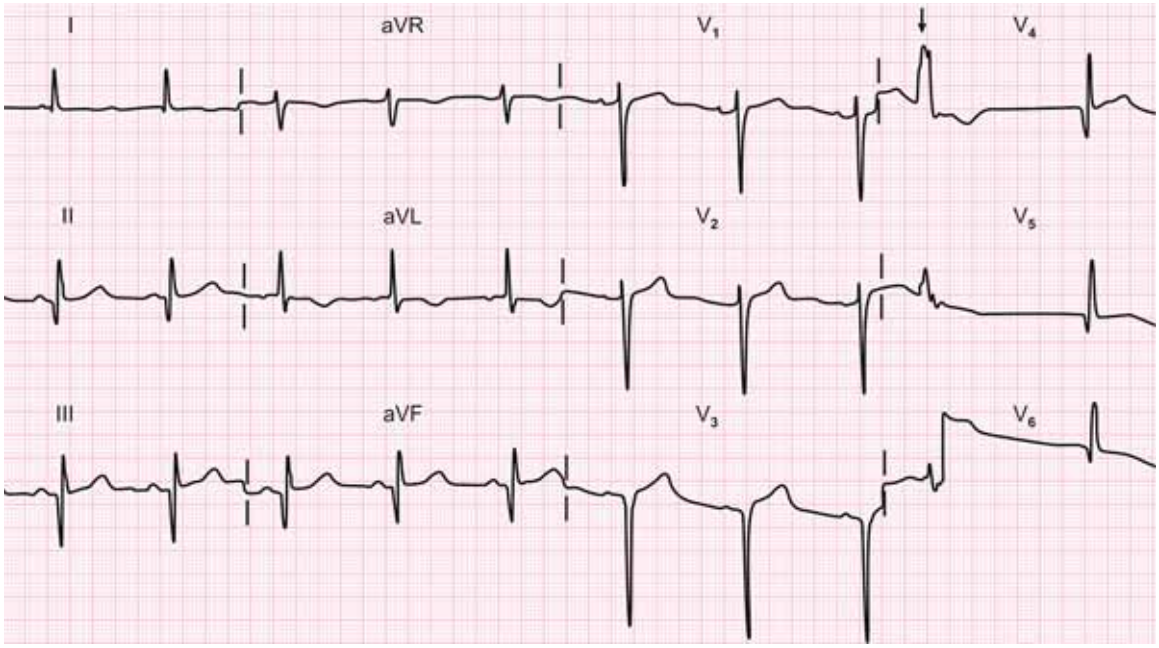


Figure 2.23: This is an ECG of a patient with the diagnosis of aortic aneurysm. This ECG shows Q waves in leads II, III, aVF and V_4–V_6 and QS wave in lead V_3. Also note ST elevation in these leads (II, III, aVF, and V_3–V_6) while ST depression and inverted T wave aVL. These findings are indicative of inferior wall MI of intermediate age and anterolateral MI of intermediate age. Prominent Q wave with still ST segment elevated is suggestive of ventricular aneurysm. R wave in lead aVL+S wave in lead V_3 are greater than 20 mm in this woman which is suggestive of LVH. Arrow shows VPC. There is long QTc also

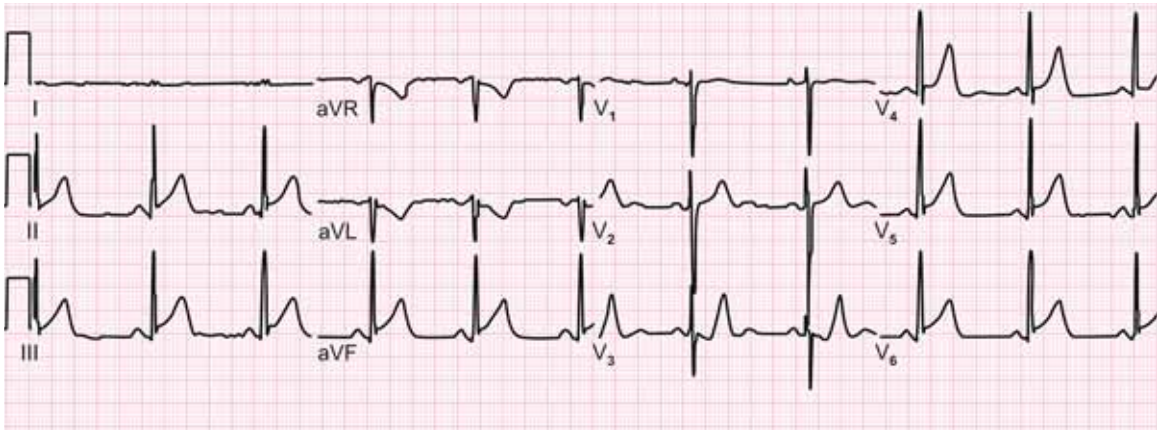


Figure 2.24: Note ST elevation in leads V_4–V_6 indicating anterolateral (better say lateral wall MI) wall infarction, in these cases ST segment in leads I, aVL is also elevated but ST elevation in lead aVL is attenuated by simultaneous inferior all infarction (look ST elevation in leads II, III, aVF) and shows reciprocally depression

Extensive Anterior Wall MI (Anterolateral MI)

Diagnostic Points (Figs 2.25 to 2.27)

- ST changes or Q wave in eight or more leads (chest leads + limb leads), i.e. leads I, aVL and V_1–V_6.
- LCX artery and LAD artery both may be blocked.

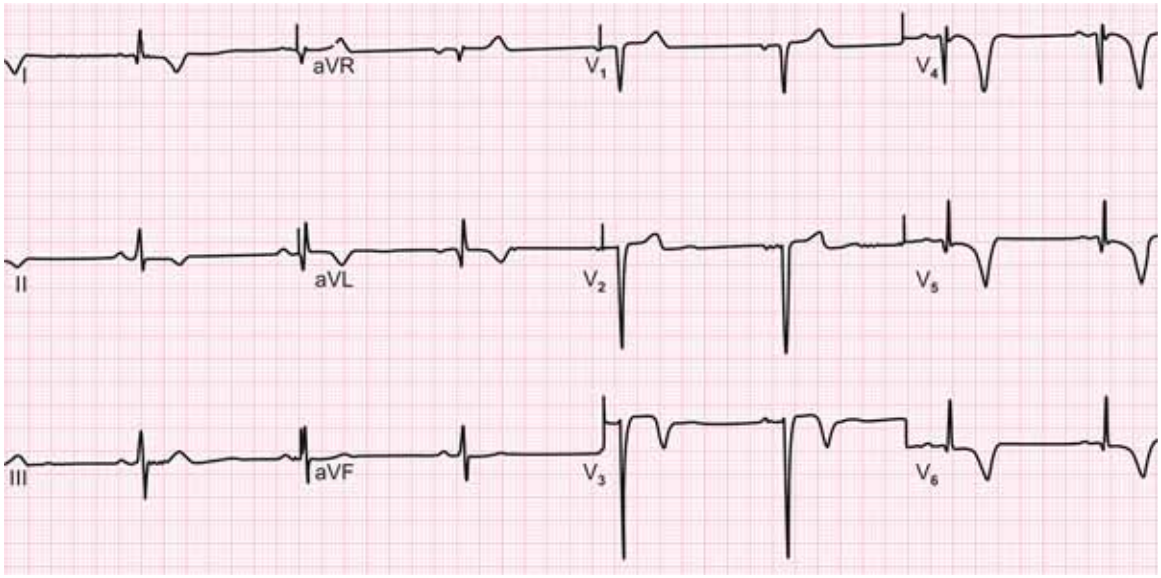


Figure 2.25: Note Q waves in leads I, aVL and V_4–V_6 and poor R wave progression V_1–V_3. There is also slight ST elevation in leads I, aVL and V_1–V_5. These changes are suggestive of recent extensive anterior and lateral wall infarction. Other findings are bradycardia and LVH (R wave in lead aVL+S wave in lead V_3 is greater than 28 mm in this man)

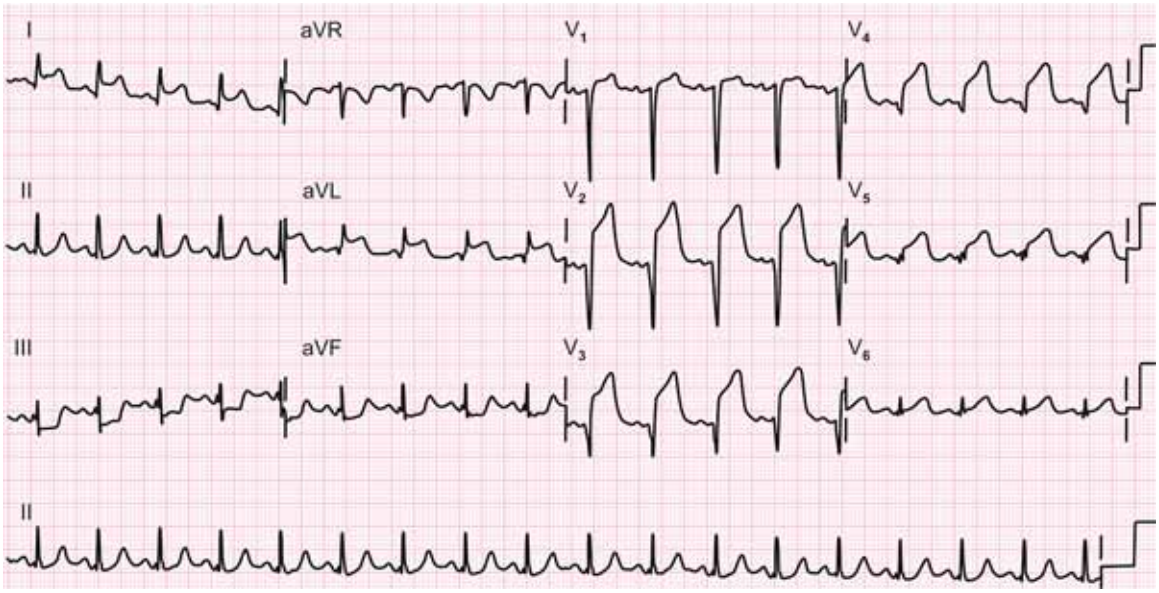


Figure 2.26: Extensive anterior wall MI. Note ST elevation in leads I, aVL, V_1–V_6, Q waves in leads V_2–V_6 and reciprocal ST depression in leads III, aVF

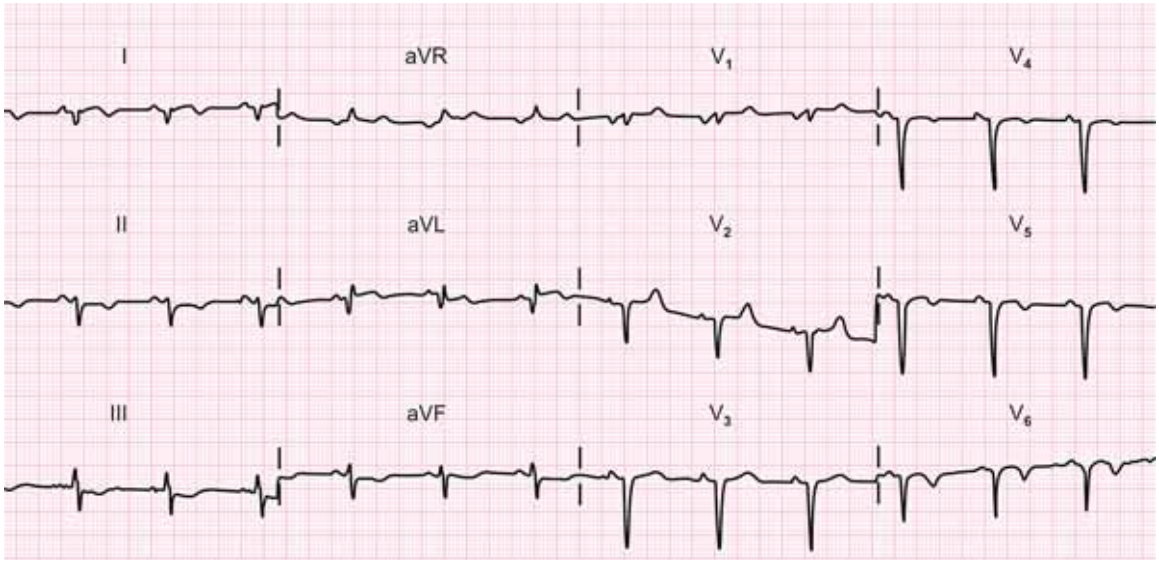


Figure 2.27: Extensive anterior wall MI. Note Q waves I, aVL, loss of R wave in V_1–V_6. There is slight ST elevation in leads I aVL V_5–V_6 and reciprocal ST depression in leads II, III, aVF and T wave inversion in leads II, III, aVF V_4–V_6. Also there is right axis deviation. Lead V_1 shows significant p terminal force indicates left atrial abnormality

Posterior MI

Diagnostic Points (Figs 2.28 and 2.29)

- Tall R waves in V_1 and V_2 and associated with ST segment depression. This is sometimes confirmed by positive mirror test: inverted mirror image will show classic QR pattern and ST elevation.
- Posterior infarction occurs virtually always in association with inferior or right ventricular infarction. If only these changes are not present, it is better to confirm with cardiac enzyme.
- If dorsal leads are placed (V_7–V_9) they show classical infarction pattern.
- T wave which is usually negative in V_1 is positive and peaked in lead V_1.
- R wave ratio to S wave (R/S) is greater than 1 in leads V_1–V_2.
- Better to consider other causes of tall R wave in V_1 (Table 1.3).

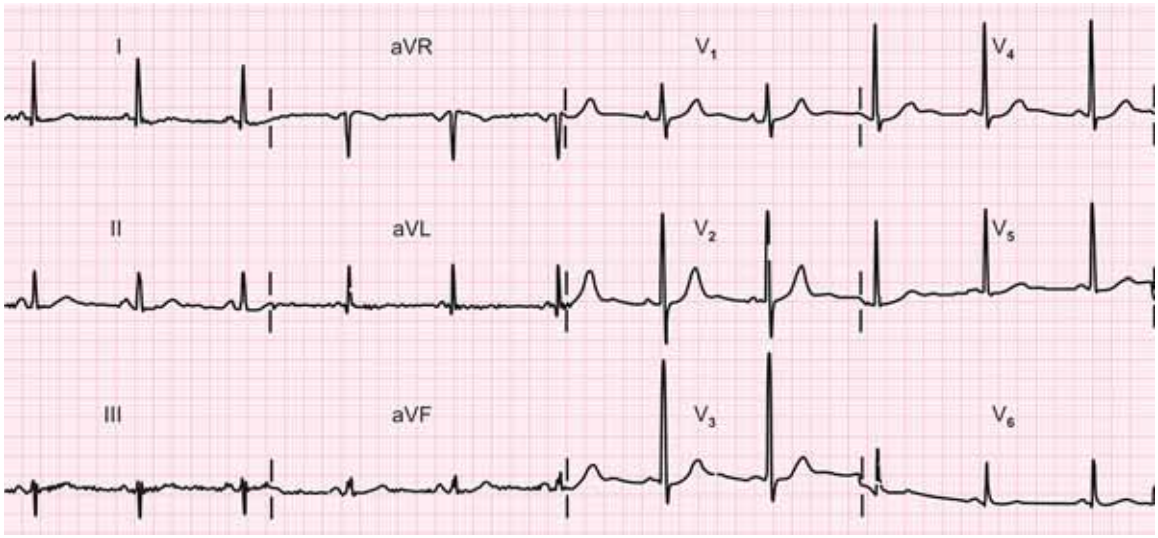


Figure 2.28: Isolated posterior wall MI. Note R wave greater than S wave in leads V_1 and V_2 and upright T wave in V_1. Tall R wave may also occur in RVH but upright T wave in V_1 or concomitant inferior wall MI supports posterior wall MI

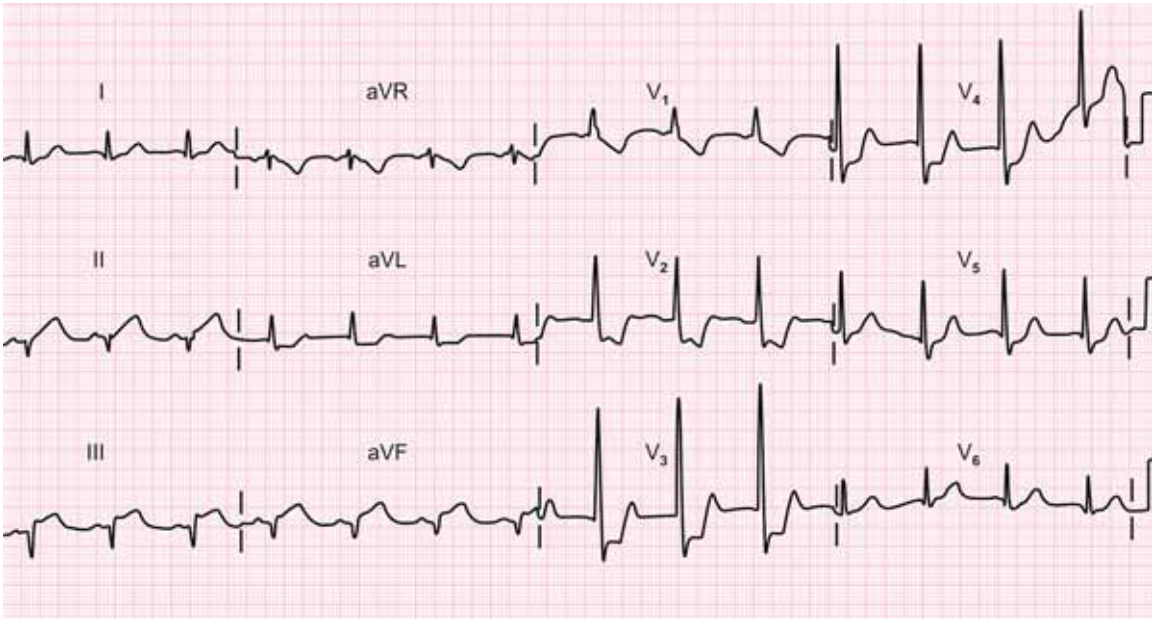


Figure 2.29: ST elevation in leads II, III, aVF with Q waves indicating inferior wall infarction, also tall T wave with R wave greater than S wave in leads V_1, V_2 indicating posterior wall infarction. ST depression in leads I, aVL and V_1–V_6 shows reciprocal depression or lateral wall ischemia

Right Ventricle MI

Diagnostic Points (Figs 2.30 and 2.31)

- Right ventricular infarction occurs in the presence of inferior infarction
- ST elevation in V_1.
- ST elevation in leads V_3R and V_4R (chest leads placed in right precordial sides).
- As said earlier, signs of inferior MI (Q wave in leads II, III, aVF).
- Note in the presence of inferior MI; ST elevation is more in lead III than II indicates probability of right ventricular MI (Fig. 2.30).

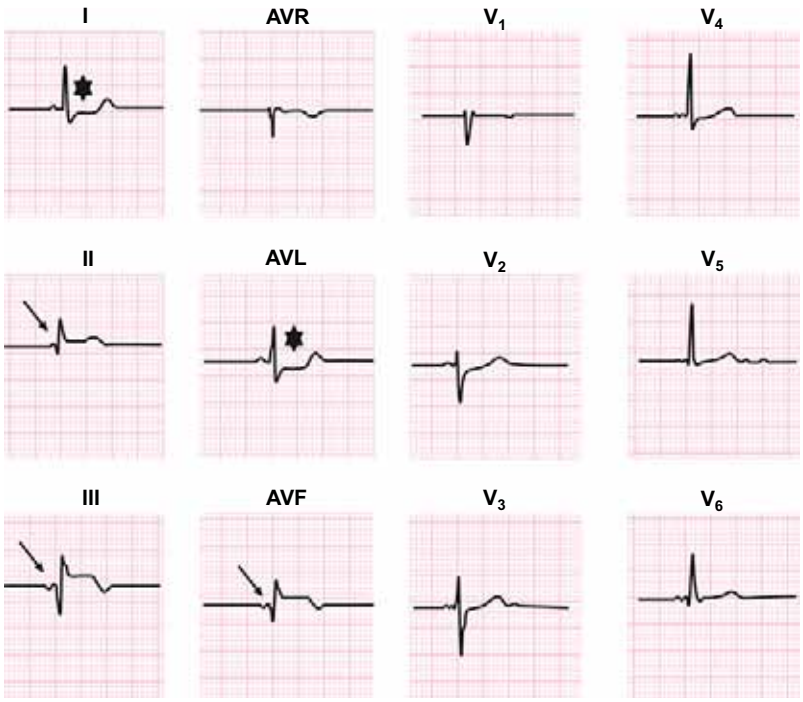


Figure 2.30: ST elevation in II, III, aVF as seen in inferior MI. Arrows indicate Q waves. Note ST elevation is more in III than II indicate probability of right ventricular MI. Asterisk shows reciprocal ST depression in I, aVL

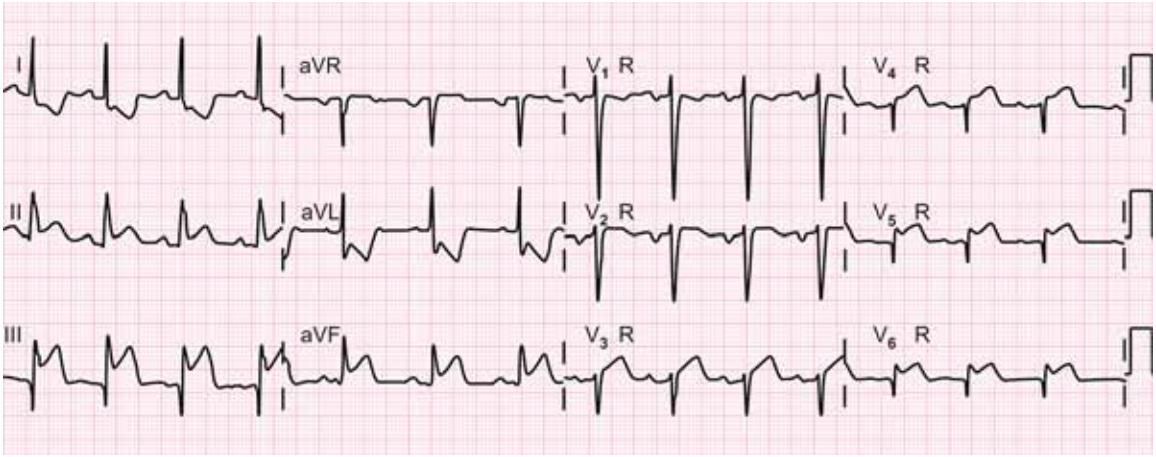


Figure 2.31: This ECG is taken with precordial leads placed right side only (limb leads are same). Note ST elevation in leads II, III, aVF and reciprocal ST depression in leads I, aVL. Also note ST elevation in lead III is greater than lead II. These are clues (ST depression in lead I and ST elevation more in lead III than II) to right ventricular MI in normal 12-lead ECG. This diagnosis is confirmed by right sided precordial leads showing ST elevation in V_2 R–V_6 R. The cause is RCA occlusion

Ischemia

Diagnostic Points (Fig. 2.32)

ST segment depression:

- Having properties described earlier in the text. (Flat, down sloping > 1 mm deep, etc.)
- Symmetrical T wave inversion.
- The areas are same as described in MI, i.e. ST depression in II, III, VF (–T wave inversion) shows inferior ischemia and vice-versa.

Note: Patient's cardiac enzyme levels and serial ECGs help us to differentiate between ischemia and infarction.

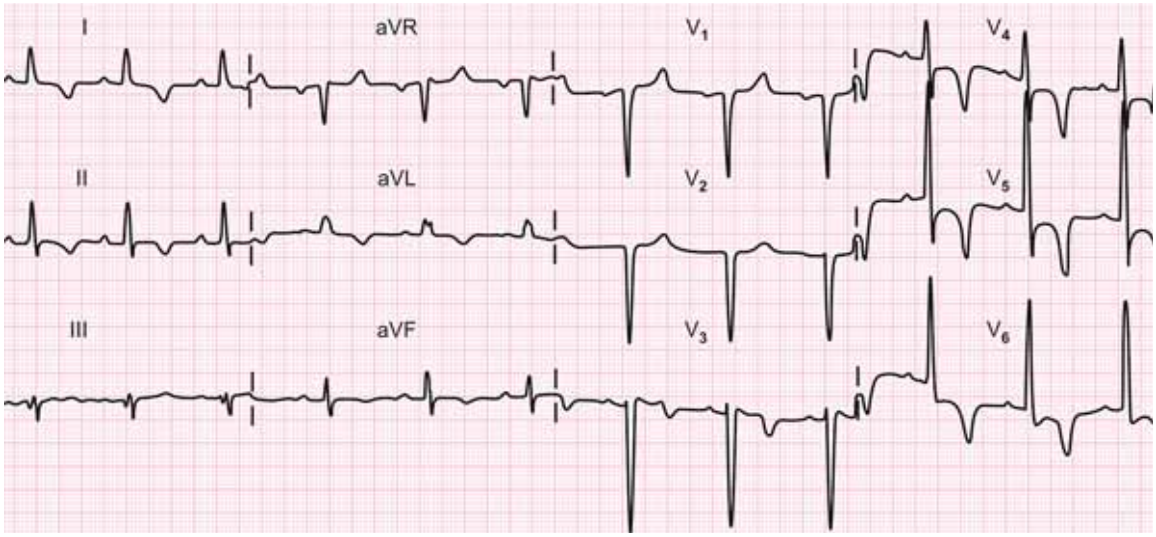


Figure 2.32: Note cardiac ischemia. There are findings indicative of LVH by voltage criteria. Note ST depression in lead V_4–V_6 and T wave inversion in leads I, II, aVL, aVF, V_3–V_6. These changes are associated with myocardial ischemia or LVH or both

RIGHT BUNDLE BRANCH BLOCK (RBBB)

Mechanism of RBBB

Recall again that current flowing towards the lead records positive deflection and flowing away records negative deflection. In RBBB, the blockade is in the flow of current down to right ventricle through right bundle branch, therefore, wave of excitation reaches right ventricle a bit late and it depolarizes after left ventricle.

The current flow in septum from left to right, this causes initial R in V_1 and Q in V_6, then current flows only to LV (because RV blockade).

So being away from the RV lead V_1 records S wave and lead V_6 records R wave.

After that this current finds chance to enter in right ventricle (towards V_1) to depolarize it so we can see R wave again (rR' pattern) in V_1 and S wave in V_6. Since this process is slow and unopposed the S wave in V_6 is wide and slurred (Figs 2.33 to 2.35 and 2.40).

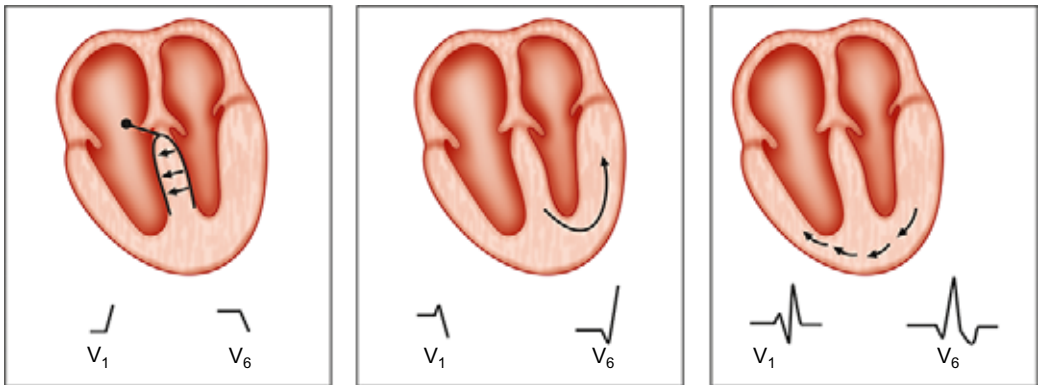


Figure 2.33: Schematic diagram showing RBBB ECG pattern. Note sequence of current flow and its appearance as ECG markings

RBBB (Complete)

Diagnostic Features (Figs 2.34 and 2.35)

- QRS complex ≥ 0.12 sec.
- A secondary R wave (R') in V_1 or V_2 called rSR' (secondary R wave usually taller than primary R wave but not always).
- Wide or slurred S wave in lead V_5, V_6 and I duration >0.04 sec or 40 ms.
- Axis may be normal, right or left deviated if axis is deviated to left in the presence of RBBB it indicated left anterior fascicular block (hemi block) also.
- As described earlier, initial depolarization is as normal (in septum left to right), therefore, initial portion of QRS complex in leads V_1 and V_2 is positive.
- Late onset of intrinsicoid deflection (peak R wave time) in lead V_1 and V_2 (greater than 0.04 sec) indicating delayed onset of right ventricular activation.

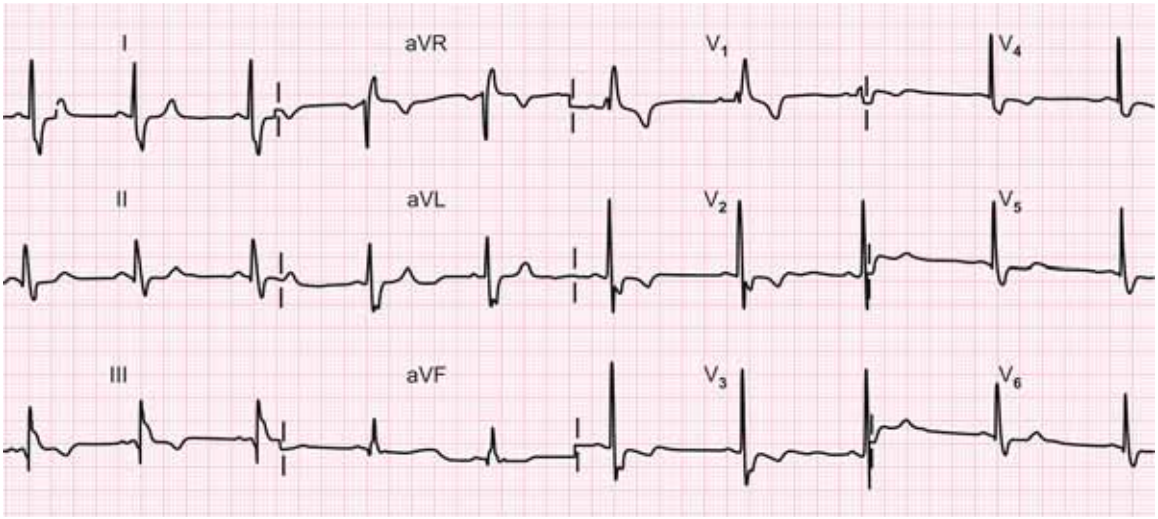


Figure 2.34: Note broad and notched QRS with a rsR' pattern accompanied by T wave of opposite polarity in lead V_1. Also note broad S wave in lead V_6 and I. This is a case of right bundle branch block (RBBB)

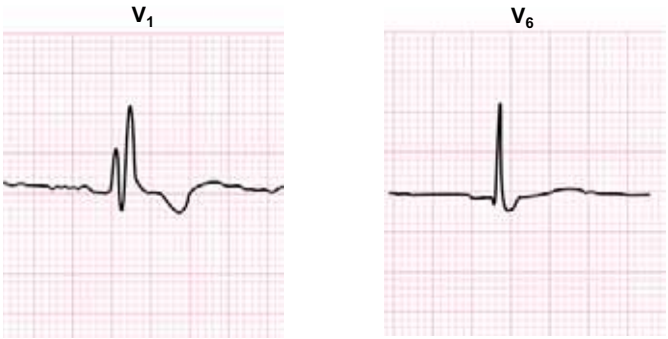


Figure 2.35: Right bundle branch block. Note tall R wave in V_1, opposite T wave to R' and broad slurred S wave in V_6

Incomplete RBBB (Fig. 2.36)

- rSR' pattern in V_1, V_2
- S wave broad in leads I, V_6.
- Duration of QRS may be less than 0.12 (.08–0.12)
- Incomplete RBBB may be found in normal individuals and patients with ASD (atrial septal defect) usually show RBBB.
- Check for WPW syndrome and Brugada syndrome if incomplete RBBB is present.
- T wave usually opposite direction to R' wave called discordant T wave.

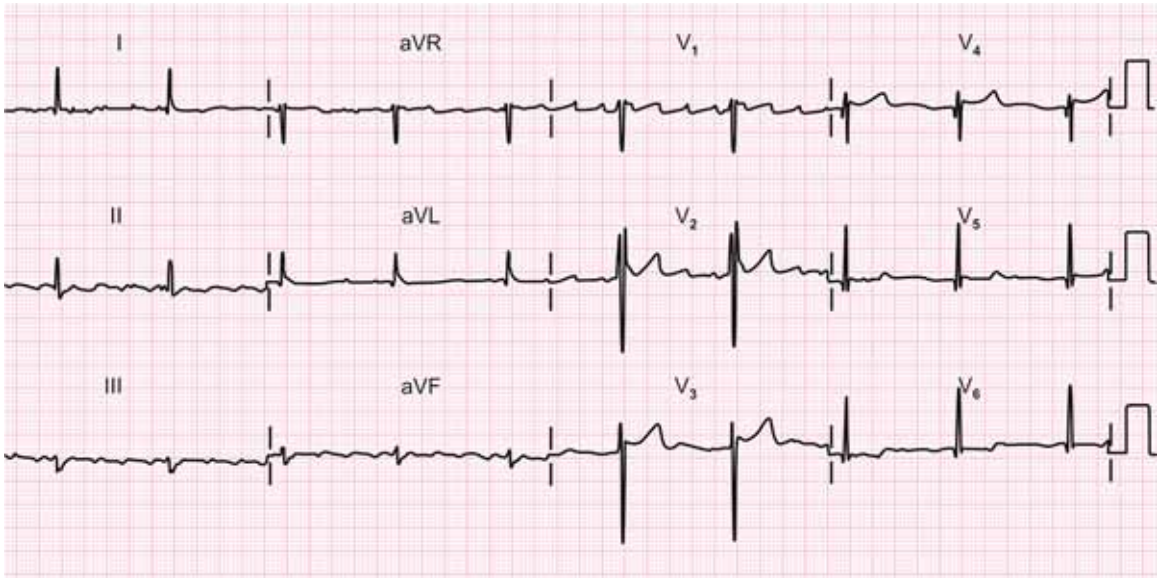


Figure 2.36: A case of atrial flutter (AF). This ECG shows RSR' pattern in lead V_1–V_2 but QRS is not broad and no apparent slurred or broad S wave in lead I and V_6 incomplete RBBB

Left Bundle Branch Block (LBBB)

Mechanism of LBBB (Figs 2.37 and 2.40)

If left bundle branch is blocked, the septum is depolarized from right to left so small Q wave in V_1 and R wave in V_6. Same as RBBB (but reversely) right ventricle is depolarized before LV (causing R in V_1 and S in V_6). Subsequent depolarization of LV causes S wave in V_1 and another R wave in V_6 (like an "M").

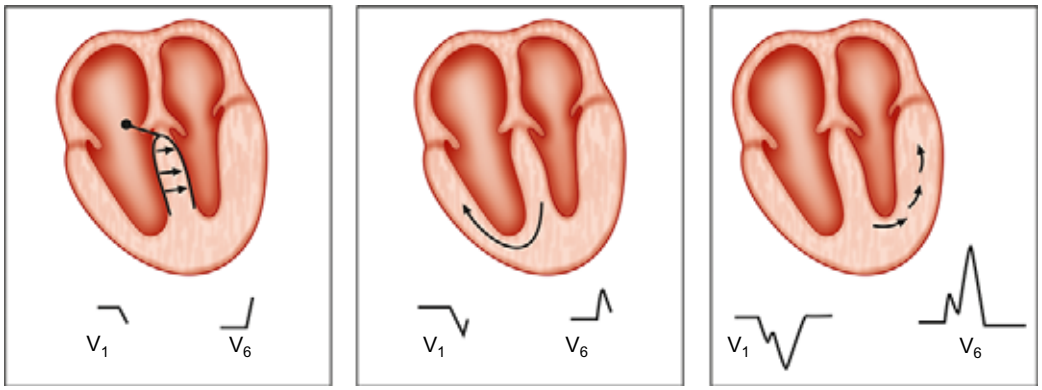


Figure 2.37: Schematic diagram illustrating flow of current and its ECG appearance in LBBB

Left BBB (Complete)

Diagnostic Points (Fig. 2.38)

- QRS > 0.12 sec
- Broad, monophasic R in V_5, V_6 (also in I , AVL) It may show "M" pattern or just show a notched
- Lead V_1, V_2 may reveal bizarre QS or rS pattern or V_1–V_2 may show a "W" like pattern or just notched.
- Note, if it appears with QS pattern, the S wave is wide and slurred usually. If it resents with rS pattern, usually it presents with poor R progression.
- Opposite to RBBB initial portion QRS in left precordial leads is abnormal in V_5, V_6 (because it is LBBB), i.e. absent small septal Q wave in V_5, V_6.
- T wave is opposite the direction of R' in V_5, V_6 (also I, AVL) this is called discordant T wave.
- Late onset of intrinsicoid deflection (peak R wave time) in lead V_5 and V_6 (greater than 0.06 sec) indicating delayed onset of left ventricular activation.

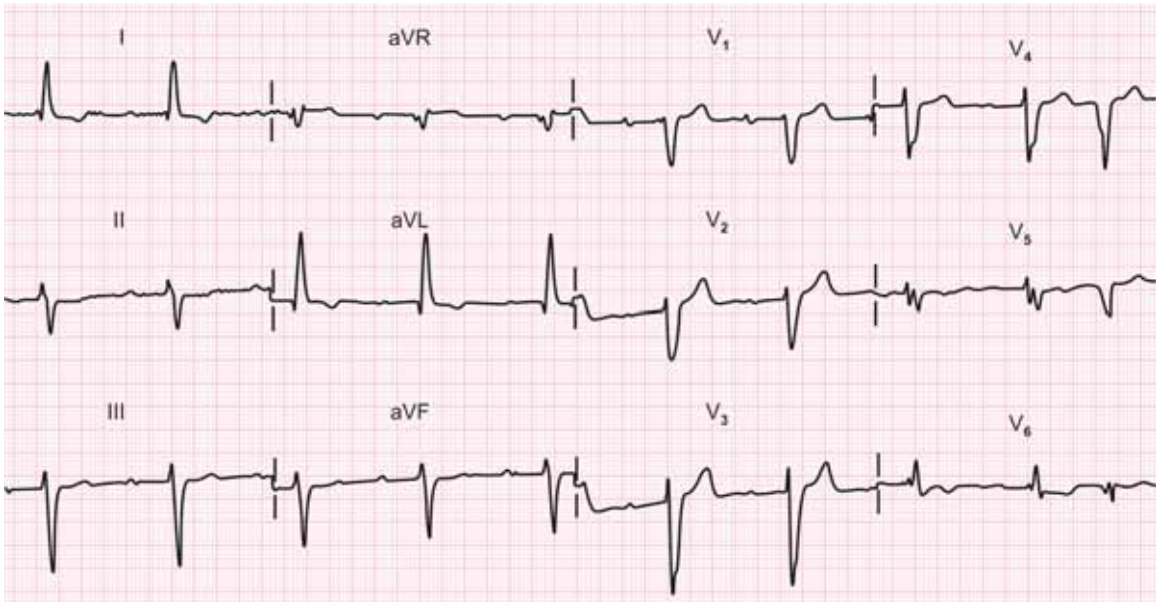


Figure 2.38: Complete left bundle branch block. Note broad monophasic R wave in lead I, aVL, V_6 (V_6 also shows rsR' with discordant T wave to R')/ V_1–V_2 shows rS pattern

Incomplete LBBB (Fig. 2.39)

It may show QRS complex duration < 0.10 to 0.12 sec and V_5, V_6 also show notched/ broad, "M" pattern and V_1, V_2 also show broad S in V_1–V_2 or QS, rS pattern.

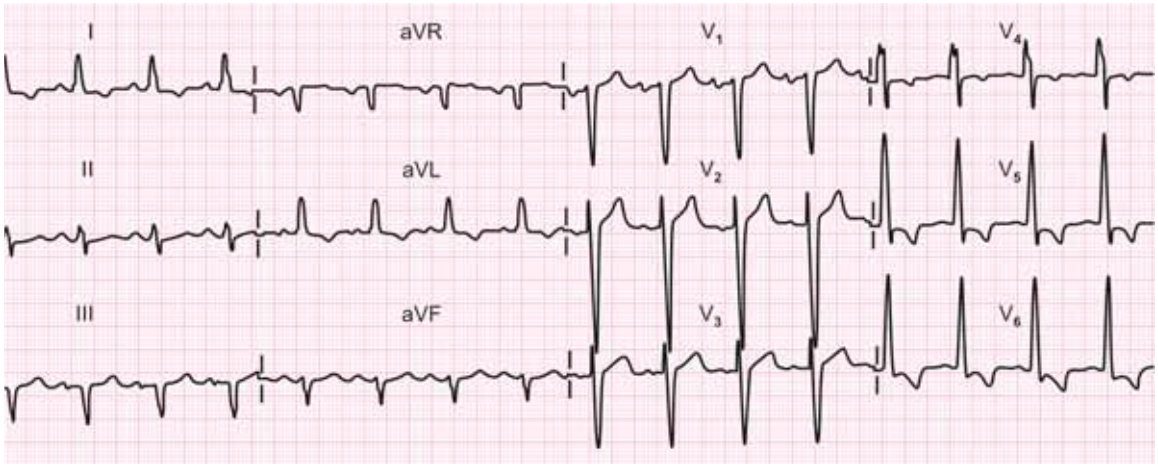


Figure 2.39: Note in this ECG absent septal Q wave in V_6, delayed intrinsicoid deflection (0.06 sec) in V_5–V_6, notching in lead V_4 and T wave inversion in V_5–V_6. There are clues to incomplete LBBB

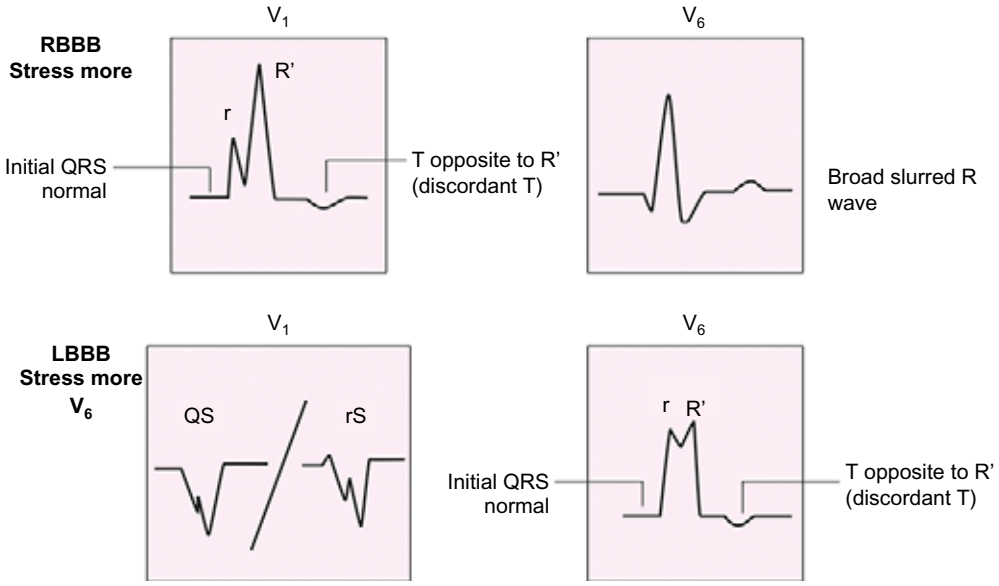


Figure 2.40: Diagram to help memorize main ECG features of RBBB and LBBB

SOME DETAILS OF FASCICULAR BLOCKS

Left bundle branch is divided into two branches:

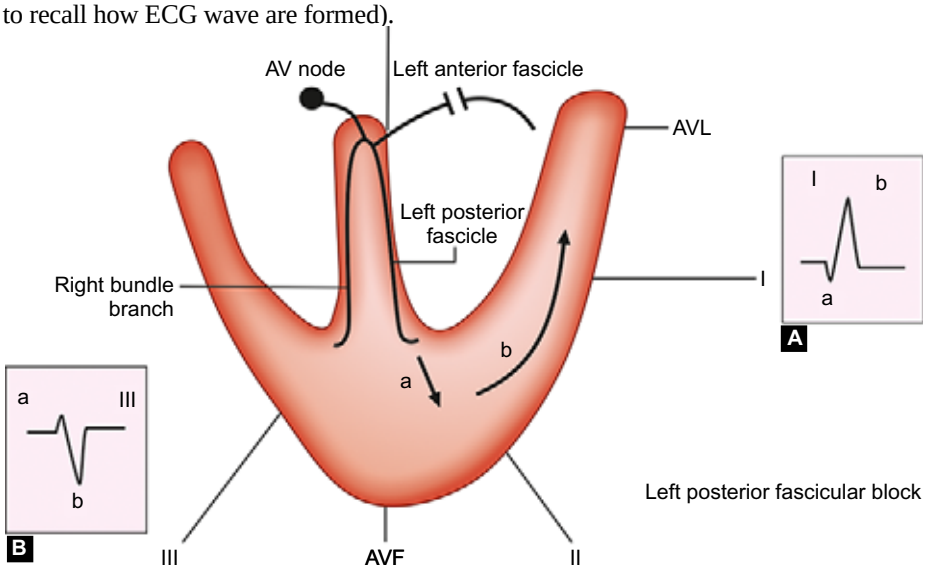
- Anterior fascicle …… A: Supply anterior superior portion of LV
  B: Blood supply from LAD artery and more susceptible to disease
- Posterior fascicle …… A: Supplies posterior inferior portion of LV
  B: Blood supply usually dual from left and right coronary artery.
  Therefore posterior fascicular block is rare.

ECG Pattern in LAF Block

Because LAF block initial wave of current flow is towards posterior fascicle and hence lead I, aVL (anterior superior area) show small Q wave recording (Fig. 2.41A).

And inferior lead II, III, aVF records small R waves (Fig. 2.41B). After that current flow is mainly towards anterior superior portion which remains unopposed causing LAD and large R in I, aVL (Fig. 2.41A) and large S wave in II, III, and aVL (Fig. 2.41B).

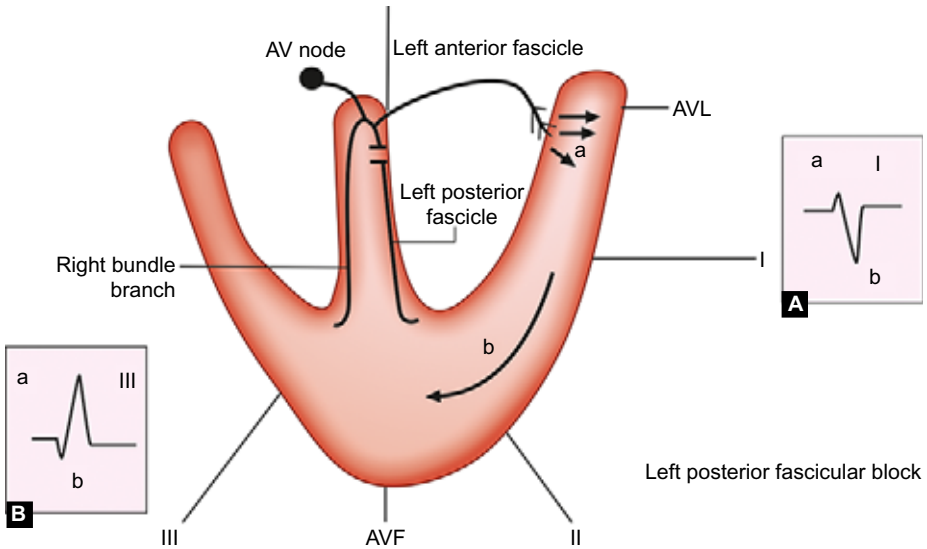
This may also give onset of intrinsicoid delayed deflection in leads aVL, I (Note: It is better to see Figure 1.10 again to recall how ECG wave are formed).



Figures 2.41A and B: Schematic diagram showing formation of ECG pattern of LAFB. Note ECG formation; vector a cause formation of small Q wave in lead I and small R wave in lead III while vector b cause formation of large R wave in lead I and large S wave in lead III

ECG Pattern in Posterior Fascicular Block

The process is same but reverse, therefore, now current flow to the LV free wall is initially through LAF (causing small Q in inferior leads [II, III, aVF (Fig. 2.42B)] and r in I, aVL (Fig. 2.42A) and then it flows unopposed to the posterior inferior part causing deep S in I, aVL (Fig. 2.42A) and large R in II, III, aVF (Fig. 2.42B) and also RAD. This may also give delayed onset of intrinsicoid deflection (R') in II, III, and aVF.



Figures 2.42A and B: Schematic diagram showing posterior fascicular blocks mechanism. Initial flow through LAF (A) cause small r in I, aVL and small Q in II, III, aVF and later unopposed (B) cause deep S in I, aVL, and large R in II, III, aVF

Left Anterior Fascicular Block

Diagnostic Points (Fig. 2.43)

- LAD less than – 45° to – 60°
- Small q wave and large R in I (qR)
- Small r and deep S in lead III, rS in lead III.
- QRS duration is not prolonged (as one part of fascicle overcomes conduction) or slightly prolonged to 0.8–0.11 sec
- There may be delayed onset of intrinsicoid deflection (R') in leads I, aVL.

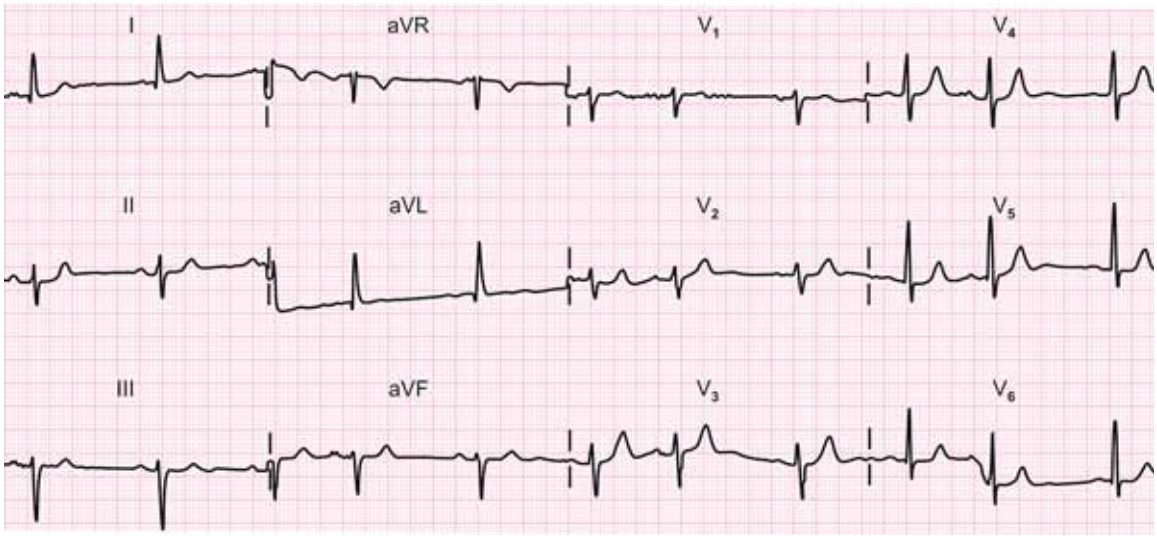


Figure 2.43: Left anterior fascicular block LAFB. Note left axis deviation (–45 degree), small q and large R in lead I, small r and deep S in lead III and delayed onset of intrinsicoid deflection in lead I, aVL

Left Posterior Fascicular Block

Diagnostic Points (Fig. 2.44)

- RAD for more than +110 degree
- Small r and deep S in I, aVL
- Small q and large R in III
- QRS duration may be normal or slightly prolonged (0.08–0.11)
- There may delayed onset of intrinsicoid deflection (R') in III.

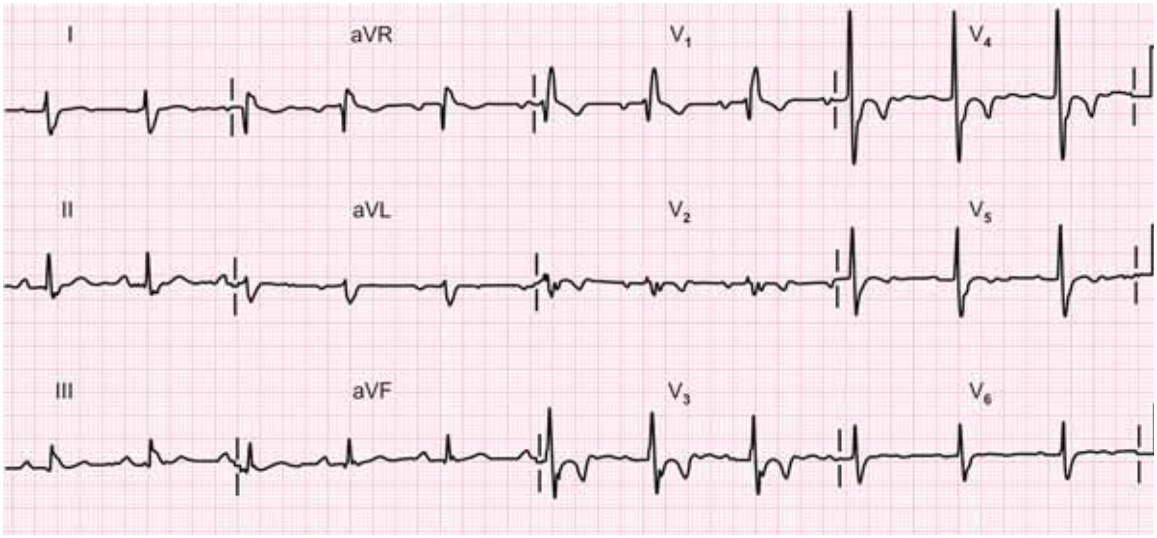


Figure 2.44: Note right axis deviation [RAD] (≈ 110), small r wave, deep S in lead I and aVL, small q and large R wave in lead III indicating LPFB

Bifascicular Block

Diagnostic Points (Figs 2.45 to 2.47)

Bifascicular block is combination of left anterior fascicular block and RBBB. This is common in clinically setting (the combination of left posterior fascicular block and RBBB is rare). So to identify bifascicular block, following should be present:

- Presence of RBBB
- Presence of LAF

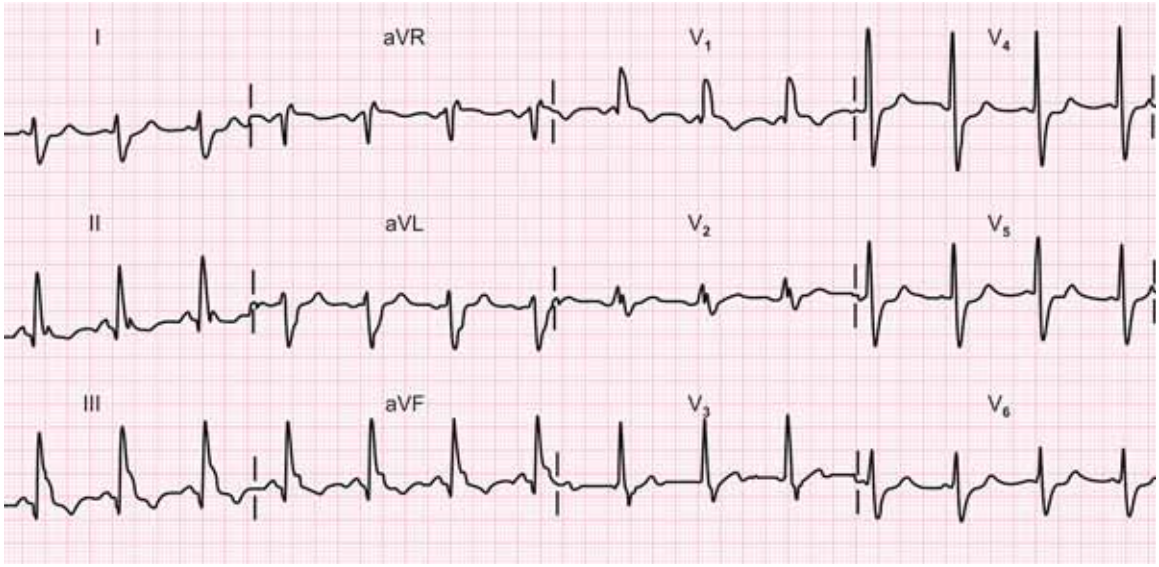


Figure 2.45: Note small r wave and deep S wave in lead I and small q wave and large R wave in lead III. Also note QRS is prolonged. There is right axis deviation (+105 degree) and also RBBB (note rsR' pattern and T wave inversion in V_1 and broad S wave in V_6). Note this type of bifascicular block is rare and usually RBBB with LAFB is seen

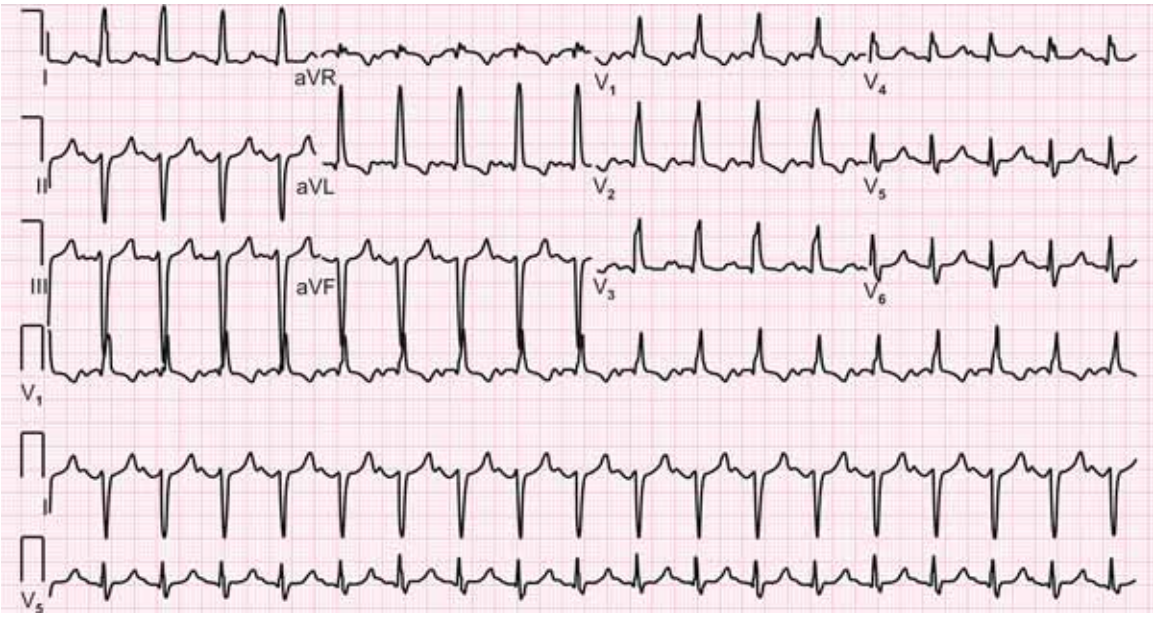


Figure 2.46: Combination of RBBB and LAFB called as bifascicular block and also first degree AV block most obvious in lead V_3 (240 ms). This condition is sometimes called as trifascicular block assuming that the other left posterior fascicle is also partially block but this is not true as it is due to AV delay not due to LPF. Note that the bifascicular block is harbinger of the complete heart block

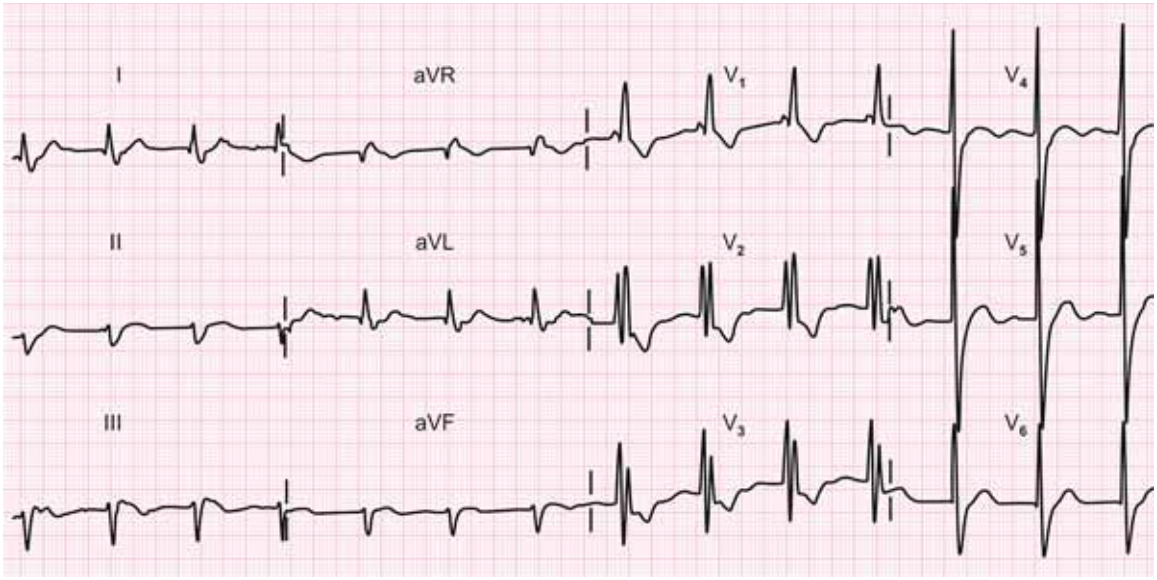


Figure 2.47: Note in this ECG with atrial fibrillation there is small q and large R wave in lead I while small r wave and deep S wave in lead III indicating LAFB while lead V_1 shows broad QRS with rSR' with discordant T wave and broad S wave in lead V_6 indicating bifascicular block

Left Ventricular Hypertrophy (LVH)

ECG criteria for LVH are imprecise and unreliable and there are many criteria for LVH diagnosis. Skipping these details and considering associated changes generally accompanied with LVH (i.e. left atrium hypertrophy, ischemic changes because of increase work load on LV, strain pattern) LVH can be clued by the following:

(*Note:* Since more gradient of current is towards LV, therefore V_1 = increased S and V_6 = increased R) Remember current flow towards a lead = positive deflection, away = negative deflection.

Diagnostic Criteria (Figs 2.48 and 2.49)

- S wave in V_1 or V_2 + R wave in V_5 or V_6 > or equal to 35 mm
- R wave aVL + S wave in V_3 > 28 mm (male) > 20 mm (female)

- Strain pattern (ST depression in leads V_5, V_6) or (ST depression with T wave inversion in which 1st part is sloping and later part is steep)
- Left atrial enlargement (see later).
- Presence of 2 of the above findings indicate probable LVH and more than 2 shows significant LVH
  1. Note also that these criteria are not always applicable in younger than 35 years especially healthy athletes where QRS may be normally increase voltage.
  2. In LAFB these cannot be used. QRS also appears to be increased.

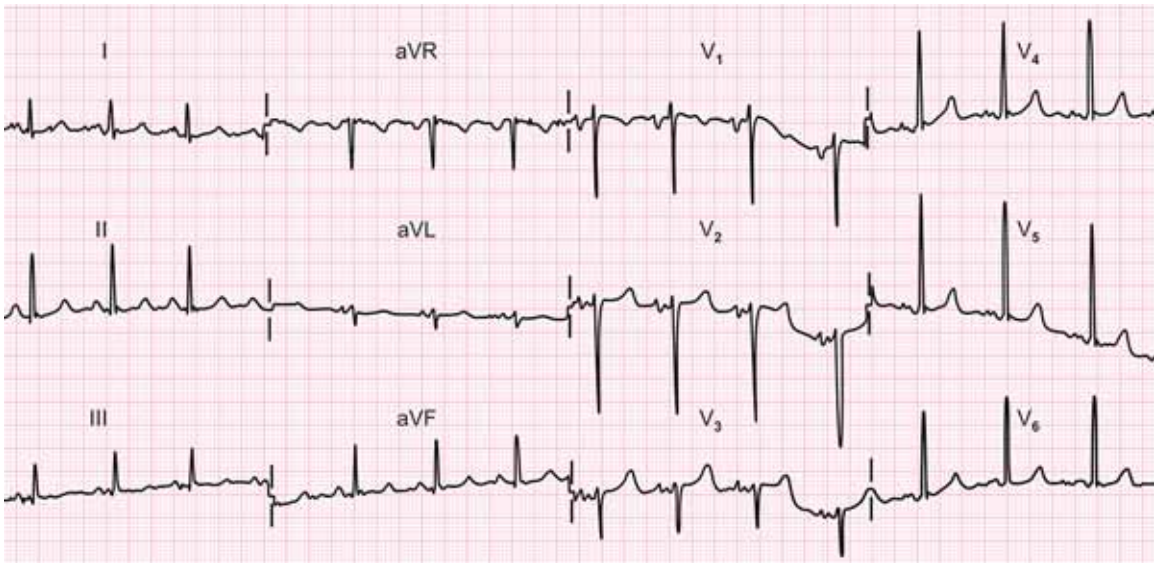


Figure 2.48: LVH. Diagnosis is supported by significant P terminal force in V_1 (also broad P wave in lead II) which shows left atrial abnormality and S wave in lead V_2 + R wave in lead V_5 greater than 35 mm

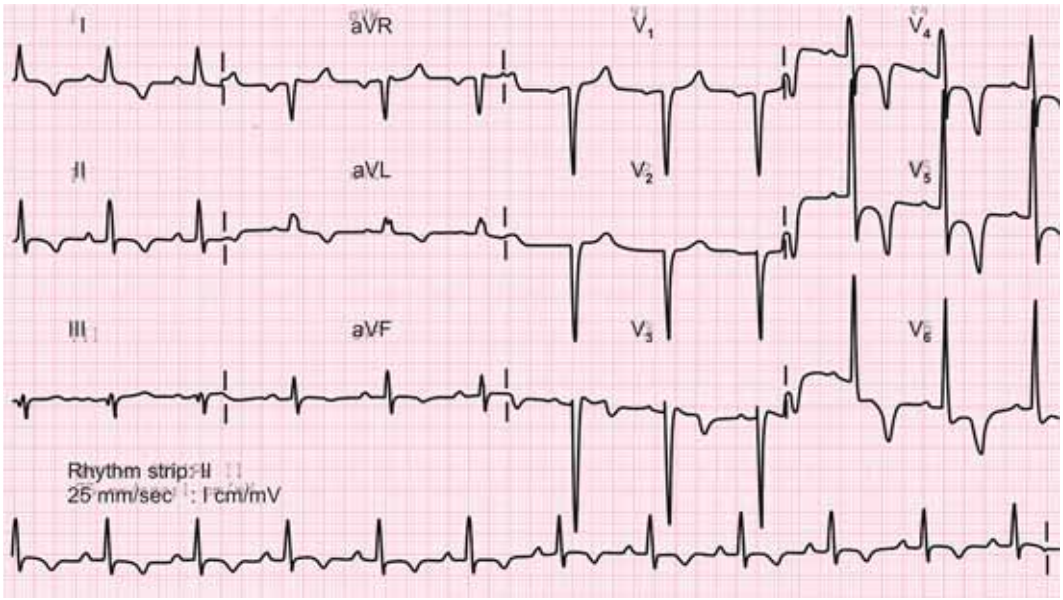


Figure 2.49: LVH by voltage criteria. R wave aVL+S wave V_3 greater than 20 mm in the woman also S wave V_2+R wave V_5 greater than 35 mm. Note flat ST segment depression V_4–V_6 and T wave inversion in I, II, aVL V_3–V_6. These ST-T changes are due to LVH or myocardial ischemia. Note degree of ST segment depression is related to size of R wave therefore it is more obvious in V_4–V_6

Right Ventricular Hypertrophy

Right ventricular hypertrophy is not as common as left ventricular hypertrophy and usually it is accompanied by right atrial hypertrophy.

Diagnostic Points (Figs 2.50 to 2.52)

- Right axis deviation (RAD) greater than +110
- Tall R wave in V_1 > 7 mm (Note: For other causes of tall R wave in V_1 see Table 1.3 and S wave in V_1<2 m)
- S wave in V_5 or V_6 > 2 mm
- R/S ratio in V_1 >1 and R/S ratio in V_6 > 1
- Strain pattern in V_1 (ST depression with T decrease)
- Right atrial enlargement; (see later)
- Delayed intrinsicoid deflection in V_1 (producing R') (>0.04 sec)

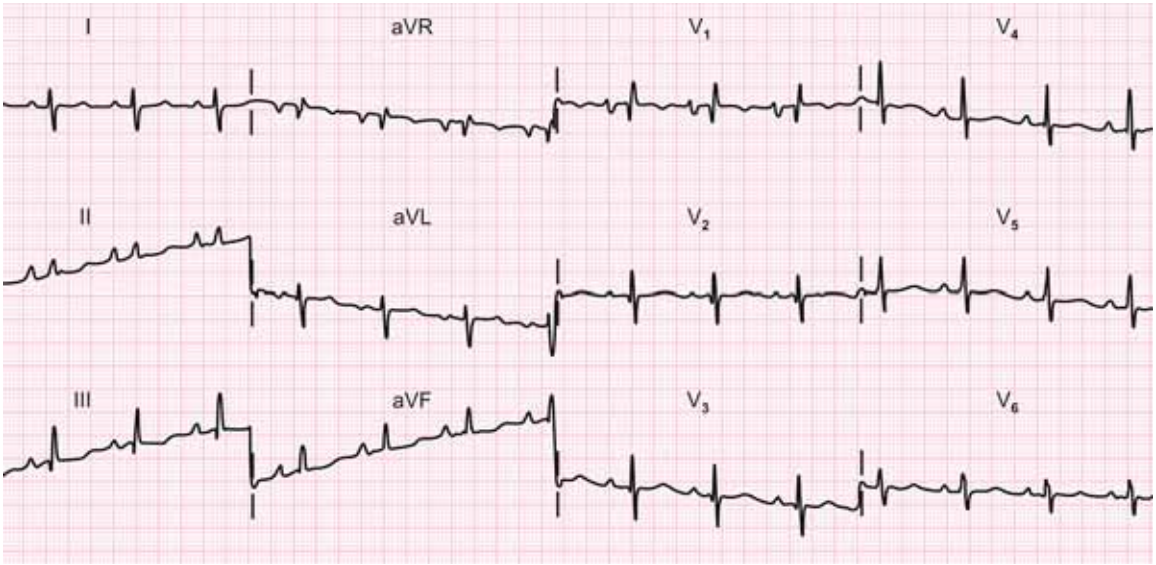


Figure 2.50: Right ventricular hypertrophy RVH with many diagnostic features. Note marked right axis deviation and tall R wave in V_1 with a q wave also, this qR pattern in V_1 is also very specific for RVH. S wave is slightly greater than R wave in lead V_6. Lead II indicates right atrial abnormality (although not meet diagnostic criteria see diagnostic criteria of right atrial abnormality). Lead V_1 shows significant P terminal force (left atrial enlargement also). May be a case of mitral stenosis

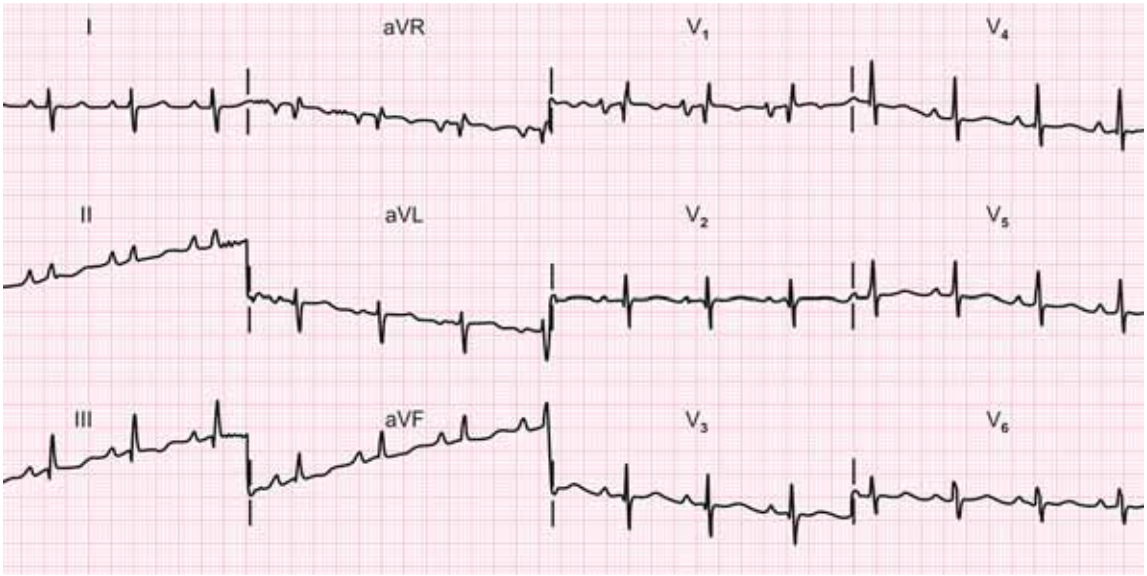


Figure 2.51: Right ventricular hypertrophy RVH with many diagnostic features. Note marked right axis deviation and tall R wave in V_1 with a q wave also, this qR pattern in V_1 is very specific for RVH. S wave is slightly greater than R wave in lead V_6. Lead II indicates right atrial abnormality (although does not meet the diagnostic criteria of right atrial hypertrophy. Lead V_1 shows significant P terminal force (left atrial enlargement). May be a case of mitral stenosis; need further investigations

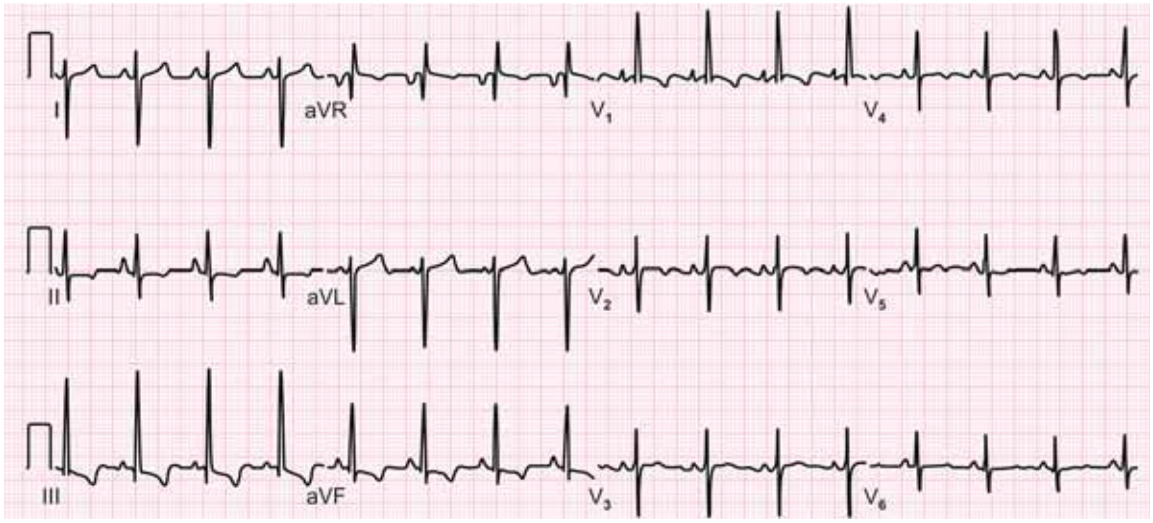


Figure 2.52: Classic presentation of right ventricular hypertrophy (RVH) showing many diagnostic criteria. Note large anterior precordial forces due to RVH indicated by R/S ratio > 1 in lead V_1 and prominent lateral precordial terminal S wave (lead V_6), right axis deviation and right atrial abnormality (peaked P wave in lead II and V_1). This is a case of primary pulmonary hypertension (PPH)

Right and Left Atrial Abnormality

Since the atrial hypertrophy can occur without ECG finding or ECG finding may occur without atrial hypertrophy (due to hypertension, etc.) term atrial abnormality is preferred.

Left Atrial Hypertrophy (Figs 2.53 to 2.56)

Diagnostic Points

- P wave duration ≥ 0.12 sec (3 small squares)
- Lead II, III, aVF and P wave may be notched (Changes more prominent in lead II). Note that lead II is more important because the changes are more prominent in lead II.
- The P wave in lead V_1 is biphasic and terminal negative part having duration ≥ 0.04 sec (one small box) and depth ≥ 1 mm.
- P terminal force in lead V_1 ≥ 0.04 sec (Product of depth and duration of negative portion of P in V_1) (See text earlier).

Right Atrial Hypertrophy (Figs 2.53 and 2.57)

Diagnostic Points

- P wave is tall and peaked with a height ≥ 2.5 mm in lead II, III, aVF and is of normal duration.
- The positive (upward) component of a biphasic P wave in lead V_1 is tall and peaked with a height ≥ 1.5 mm.

Note: Any of the above is enough to make a diagnosis.

Since congenital heart disease in children may cause right atrial hypertrophy. It is better to pay more attention to find atrial hypertrophy if congenital heart disease.

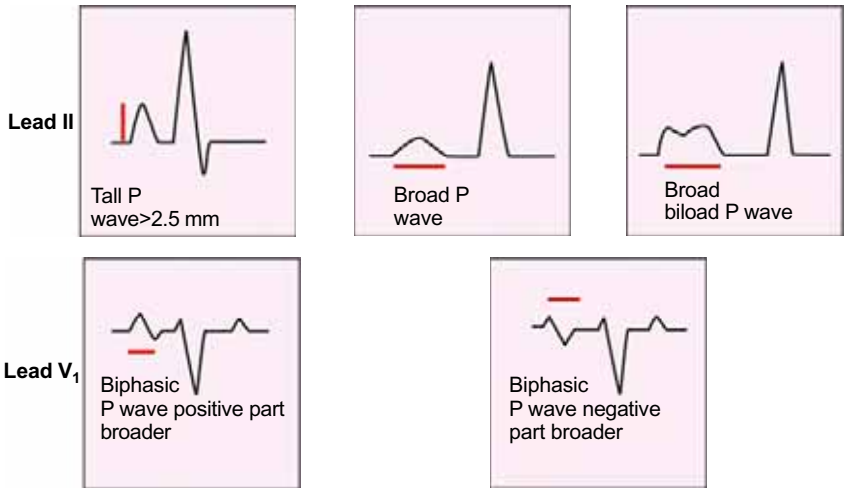


Figure 2.53: Schematic diagram to learn features of right and left atrial abnormalities

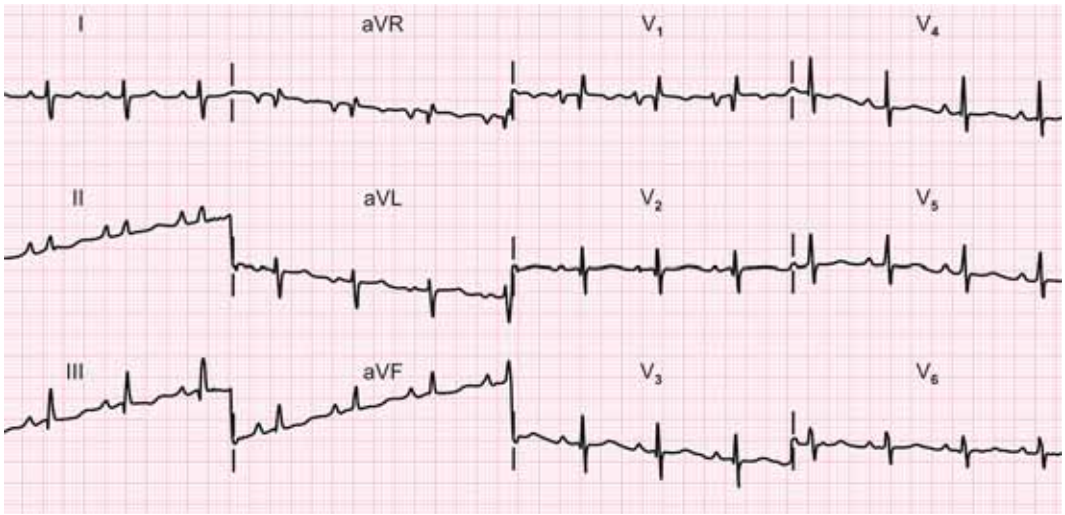


Figure 2.54: Left atrial abnormality identified by broad P wave in lead II and biphasic P wave with significant P terminal force in lead V_1

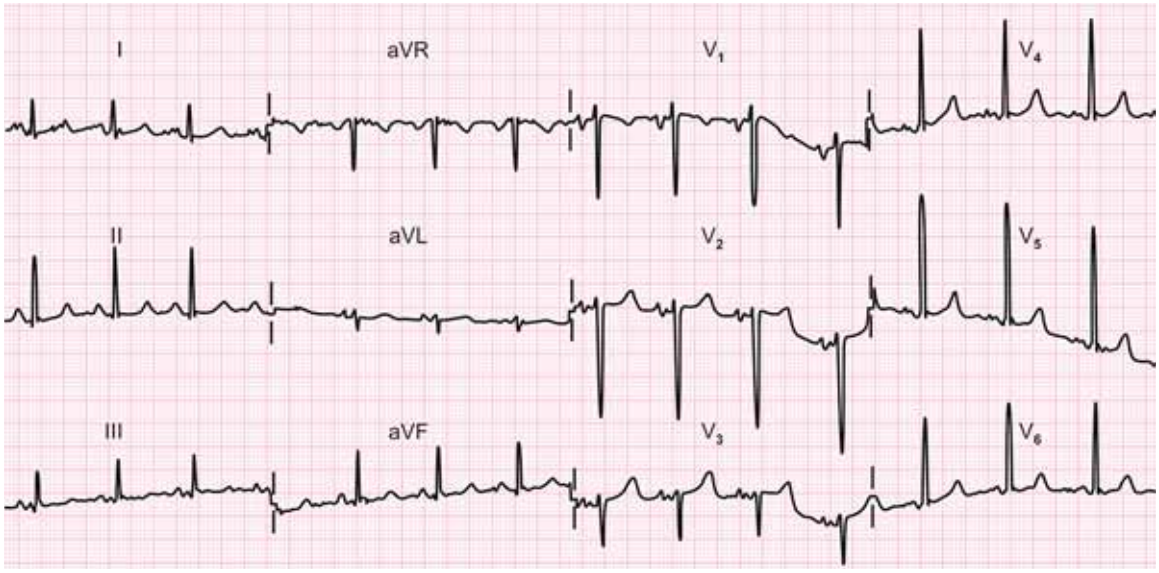


Figure 2.55: Note broad P wave in lead II and biphasic P wave with significant P terminal force in lead V_1 indicates left atrial abnormality. Also note LVH

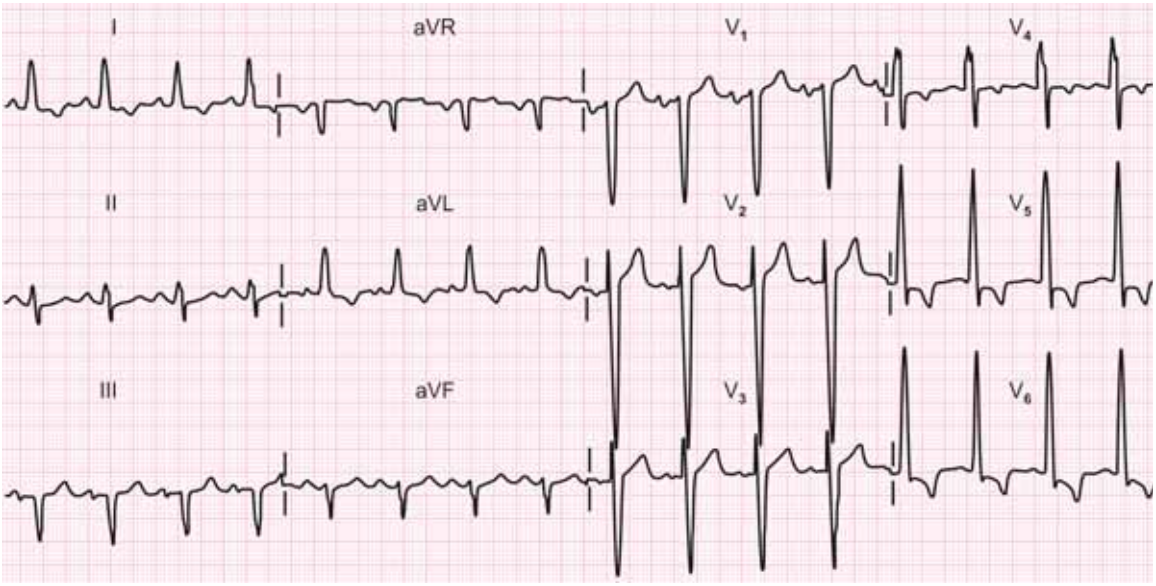


Figure 2.56: Note broad P wave in lead II and biphasic P wave with significant P terminal force in lead V_1 indicates left atrial abnormality. Note also ECG findings of LVH, LAD and incomplete LBBB

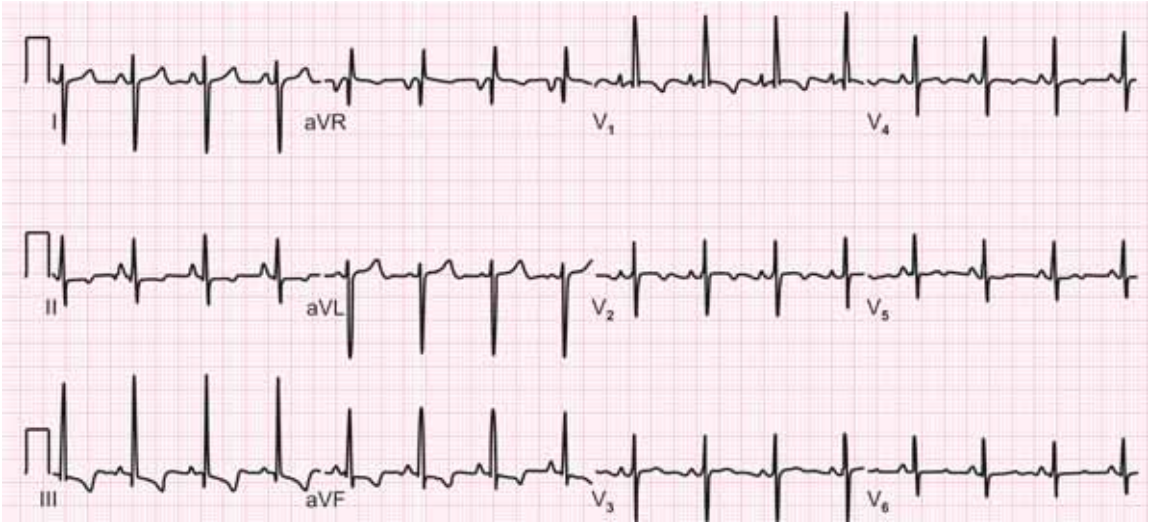


Figure 2.57: Right atrial abnormality. Note the peaked P wave in lead II and V_1. This ECG also shows RVH

Biatrial Hypertrophy (Fig. 2.58)

Simultaneous right and left atrial hypertrophy may be present if:

- Tall peaked P in lead II > 2.5 mm indicates right ventricular hypertrophy.
- Biphasic P in V_1 with P terminal force ≥ 0.04 mm indicates left atrial hypertrophy.

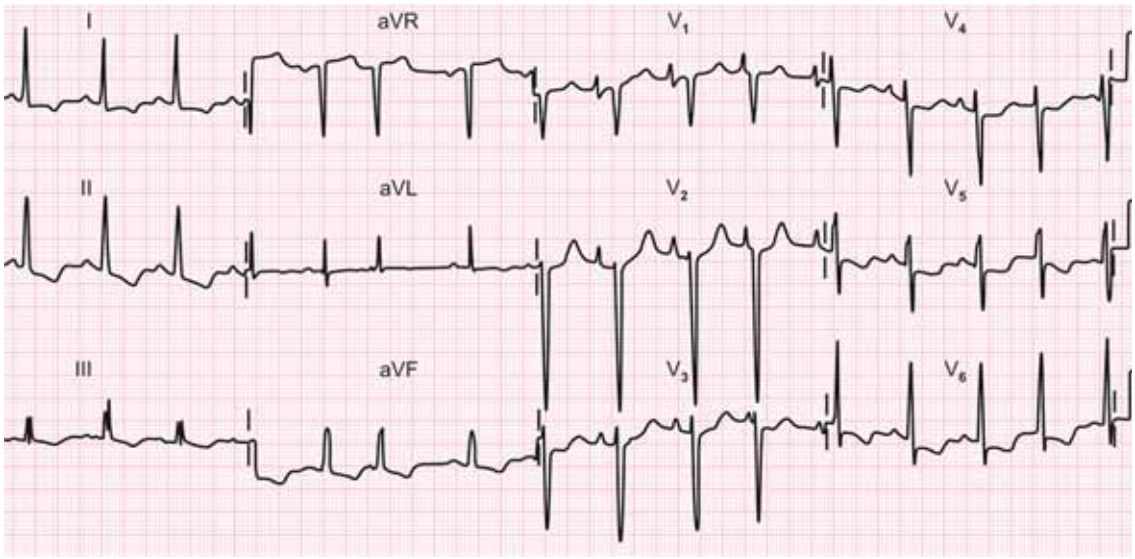


Figure 2.58: This ECG shows biphasic P wave with tall initial component and significant P terminal force in lead V_1 which is indicating biatrial abnormality; S wave in lead V_2 + R wave in lead V_5 > 35 mm and R wave in lead aVL+S wave in lead V_3 > 20 mm in this woman which shows LVH

ARRHYTHMIAS

Atrial Premature Beats or Contractions (APB) or (APC) (Figs 2.59 to 2.62)

QRS complexes occurring earlier than expected time (premature) are called as atrial premature beats (APB) or atrial premature contractions (APC). Since in APCs, the impulse comes from a place other than SA node and does not proceed through a normal atrial conduction pathway that's why P wave in APC is bizarre or inverted. This premature P may fall on T and distort it therefore a notched/distorted T followed by an early QRS is helpful clue for APC.

- Since conduction through ventricle remains unchanged QRS complex following APC looks like the previous QRS complexes. This is called normally conducted APC.
- If APC occurs very early while the ventricles are partially refractory (i.e. yet not fully repolarized from the previous normal sinus beat), APC will cause slightly slurred or widened QRS usually RBBB pattern (rSR') looks like VPC. This is called aberrantly conducted APC.
- If APC occurs too early and ventricle is totally refractory to depolarization the P wave will not be followed by a QRS complex this is called nonconducted APC or blocked APC (Fig. 2.62).
  On ECG, it appears as a pause during a normal sinus rhythm (APC nonconducted is the commonest cause

of a pause in normal sinus rhythm). Therefore, if premature P is not identified rhythm may be diagnosed as sinus pause (See Table 2.2).

- Since ectopic impulse disturbs the normal impulse from SA node, after the ectopic discharge the SA node takes a "compensatory pause" this compensatory pause may be:
  A. Incomplete: P-P cycle including the APC is less than twice the sinus cycle.
  B. Complete: Duration is equal to twice sinus rate.
- PR interval of APC may be shorten, lengthen or unchanged depending on site of ectopic focus.

Aberrantly Conducted APC

Features of Aberrantly Conducted APC

- RR cycle ending in aberrant QRS complex is preceded by a long RR cycle (Ashman's phenomenon) (Fig. 2.63)
- Aberrantly conducting APC can be differentiated from VPC because VPC (ventricular premature beats)
  - Does not has P before QRS (Fig. 2.64)
  - VPC QRS doesn't present with rSR' (triphasic) but usually monophasic or biphasic (qR or QS).
  - Ashman's phenomenon is not present in VPC.
  - If APC occurs after every sinus beat the ECGs condition is called bigeminy (Figs 2.65 and 2.66).
  - If APC is occurring early may initiate atrial tachycardia.

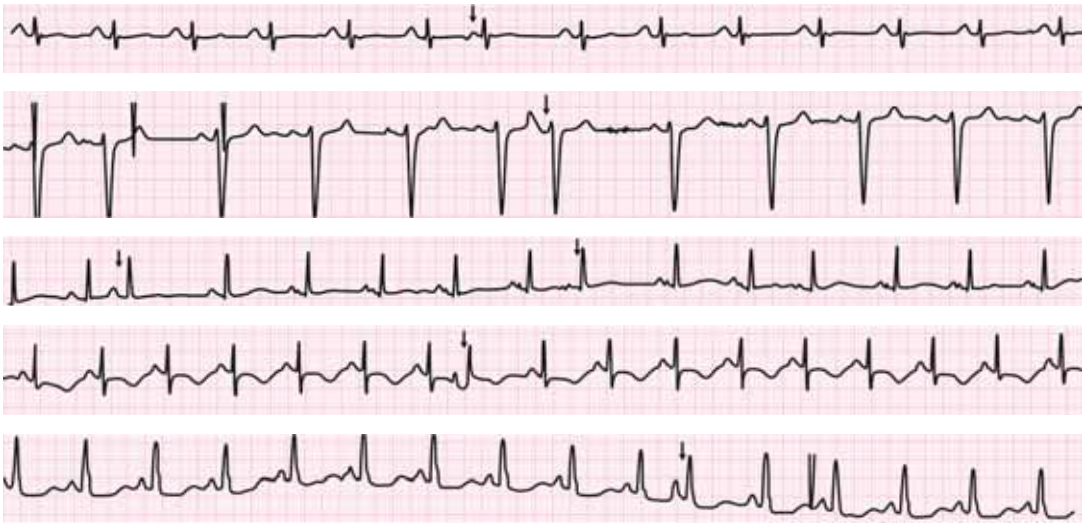


Figure 2.59: Different ECG rhythm strips showing APCs (arrow). Note earlier than expected timing, different looking P waves, normal looking QRS and compensatory pause after APCs

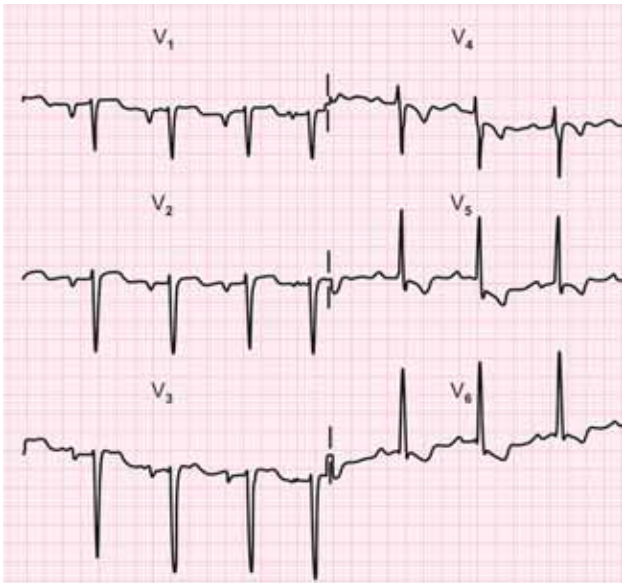


Figure 2.60: Fourth beat is APC (arrow). Note earlier than expected, normal looking QRS but P wave of different morphology

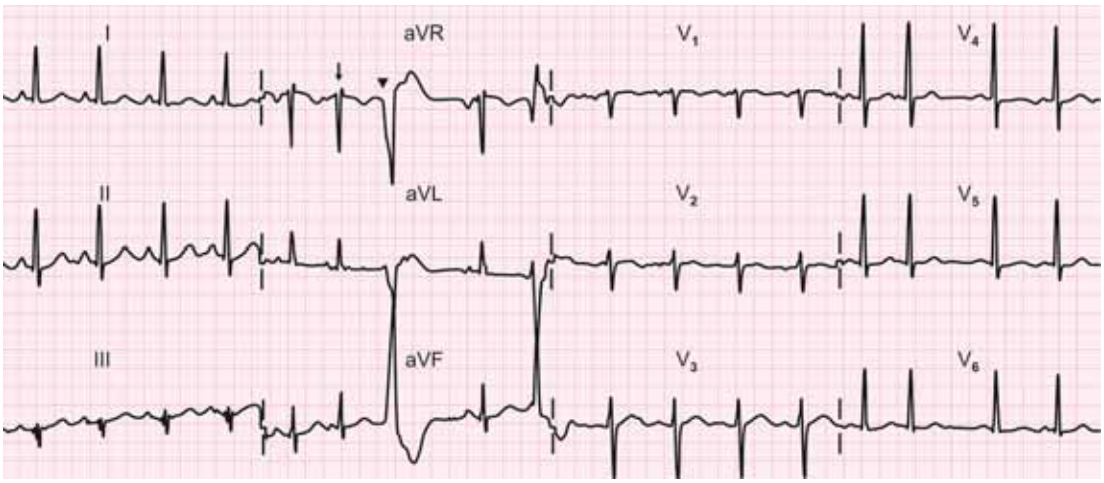


Figure 2.61: Note atrial premature contraction APC in the 6th and 15th beat (arrow). Note P wave preceding normal looking QRS. For comparison there is also ventricular premature contraction VPC (arrowhead)

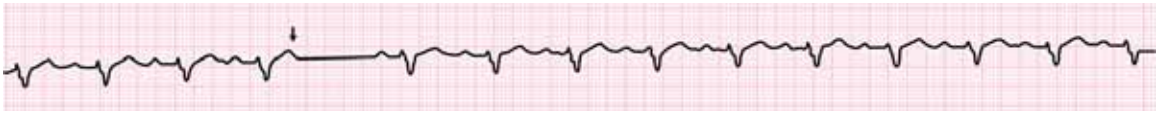


Figure 2.62: Nonconducted APC, it may appear sinus pause but if you look carefully you may find T wave (arrow) before the pause which is slightly more prominent

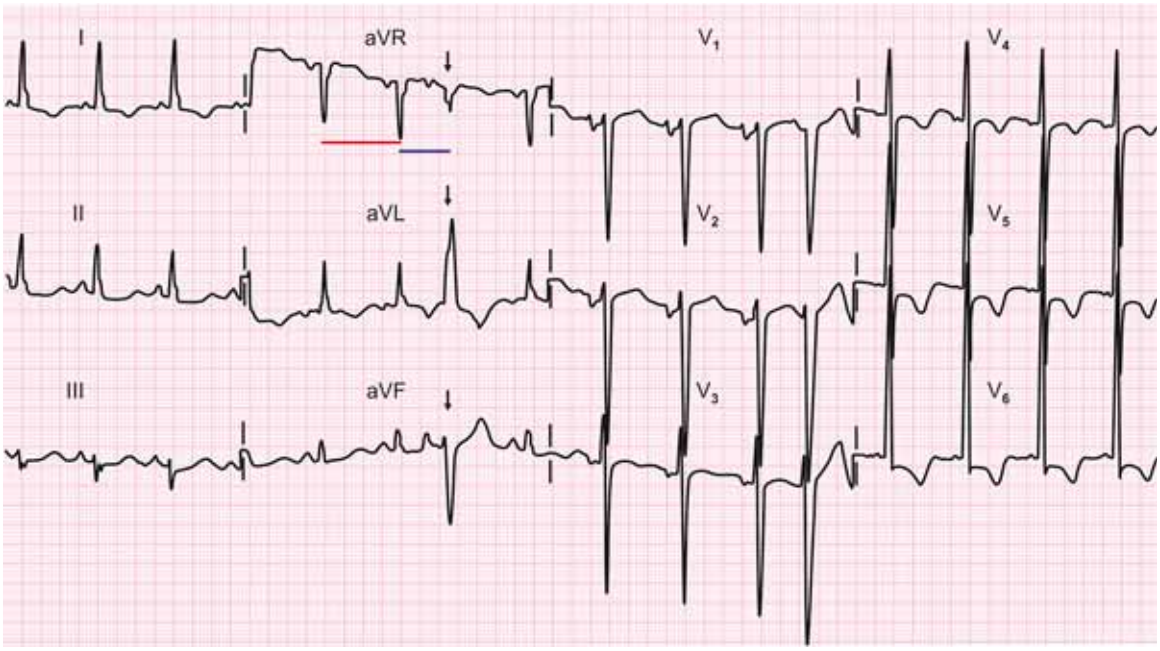


Figure 2.63: Aberrantly conducted APC (6th beat, arrow). Note broad QRS mimicking ventricular in origin but there is P wave distorting the preceding T wave indicating that origin supraventricular. Also note Ashman's phenomenon with long short cycle sequence, i.e. short cycle ending in a broad QRS complex is preceded by a long cycle, slightly triphasic QRS (rsR') complex. All these features differentiate it from VPC

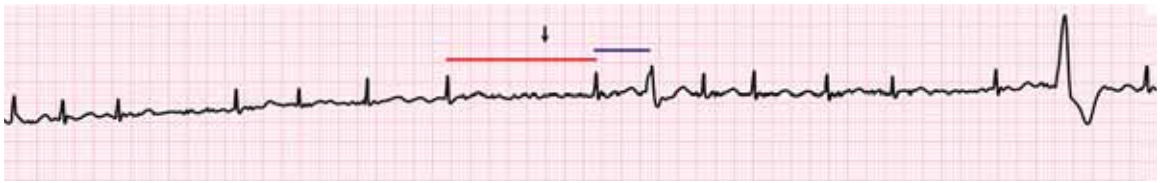


Figure 2.64: A case of atrial fibrillation. Differentiate aberrantly conducted APC and VPC. 9th beat is aberrantly conducted APC while 15th beat is (second last) is VPC. Note in aberrantly conducted APC the long-short cycle sequence is obvious, i.e. short cycle ending in a premature beat (containing broad QRS complex) is preceded by a long cycle (arrow).This is called Ashman's phenomenon. This is not very obvious in VPC

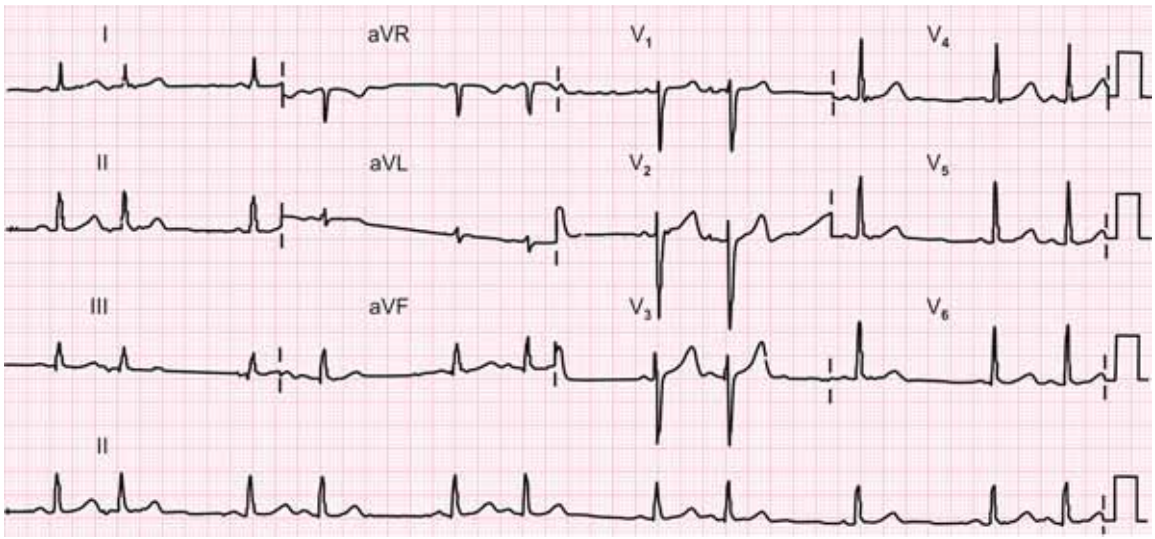


Figure 2.65: Atrial premature beats in forms of pairs bigeminy. Note every sinus beat is followed by APC. Compensatory pause after the APC is also visible

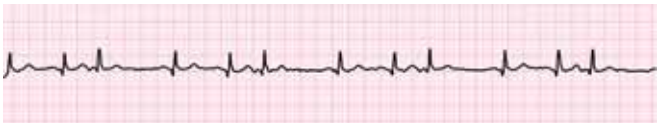


Figure 2.66: APC in trigeminy. Every sinus beat is followed by a pair of normally conducted APCs

Junctional Premature Contractions (JPC)

Diagnostic Points (Figs 2.67 to 2.70)

Here impulse arises from area around AV junction to cause earlier than expected occurring beats among normal occurring sinus beats. Since impulse is at AV junction it may also travel in retrograde direction which causes abnormal shaped P wave usually inverted. Sometimes this P wave buried in QRS and cannot be seen or after QRS (depending on rate of conduction). QRS remain normal looking there for diagnostic clues are:

1. QRS occurring earlier than expected time.
2. QRS is of normal configuration (as previous sinus beats)
3. Retrograde P wave may be visible closely preceding, just after QRS or may not be visible. Best seen in II, III, and aVF.
4. PR interval is usually shortened.

A series of three or more JPCs is called junctional rhythm. The ectopic foci may supersede sinus node and drive heart on its own.

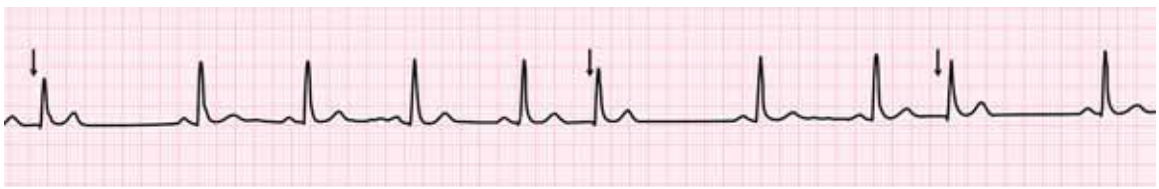


Figure 2.67: Junctional premature beat (arrows), note earlier than expected time, normal looking QRS and absence of P wave

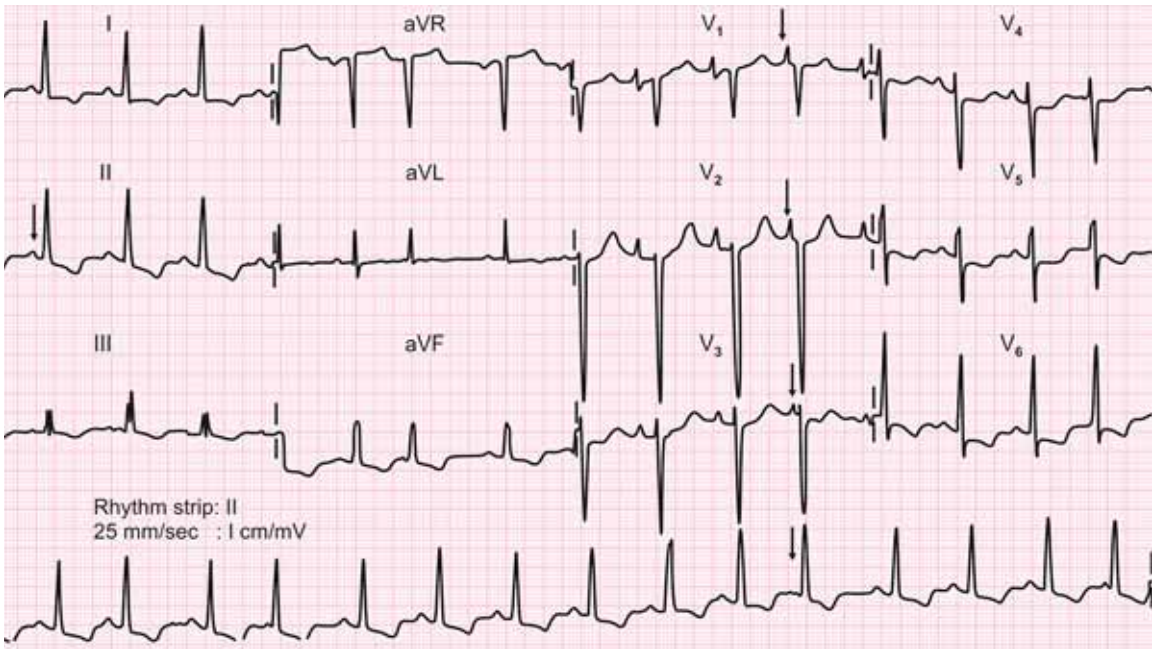


Figure 2.68: JPCs. Note earlier than expected time and different looking or absent P wave and short PR interval

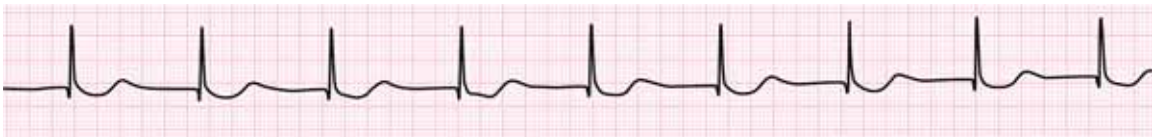


Figure 2.69: ECG of a patient on calcium channel blocker overdose and resulting suppression of sinus rhythm. This is AV junctional (escape) rhythm. Note rate 52 bpm, i.e. slower than sinus rhythm and absent P waves

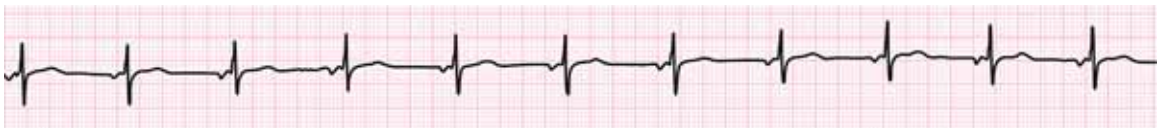


Figure 2.70: AV junctional (escape) rhythm due to verapamil. Note slightly accelerated 67 bpm (accelerated junctional rhythm). Note negative P waves before QRS

Ventricular Premature Contractions (VPC)

Diagnostic Points (Figs 2.71 to 2.75)

(Beats arising from ectopic focus in ventricle)

- QRS occurring earlier than expected time.
- QRS shape is bizarre, wide (Sometimes right [upslope of R] hand bigger than left hand called a rabbit ear) Best seen in V_1.
- Since the focus is in ventricle, no P wave before VPC.
- VPC is followed (usually) by full compensatory pause (which differentiates it from JPC).
- A single VPC between two normal sinus beats is called interpolated VPC (Note PR interval of sinus beat following by VPC is slightly increased than PR of other normal sinus beat).
- If there are many foci generating VPC, these are called "Multifocal VPC" these are more serious than Unifocal and identified by different shapes and different style of coupling, e.g. triplets, couplets

[PVC may occur early and fall on the vulnerable period which is just before the apex of T wave and may result in a sequence of local re-entry that may precipitate ventricular fibrillation or tachycardia. This is called R on T phenomenon (Fig. 2.71).

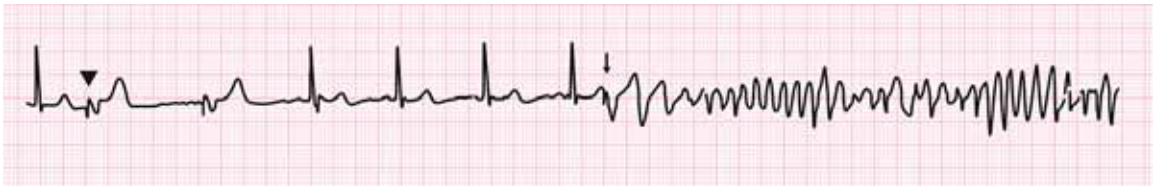


Figure 2.71: This ECG shows R on T phenomenon in 2nd and 3rd beats are normally paced beats and following 4 beats are normal sinus beats but later on the pacemaker fails to sense properly and paced beat interrupts down sloping portion of T wave (R on T) (arrow). The result is initiation of ventricular tachycardia in the form of Torsade de pointes. Arrow head also shows failure to sense

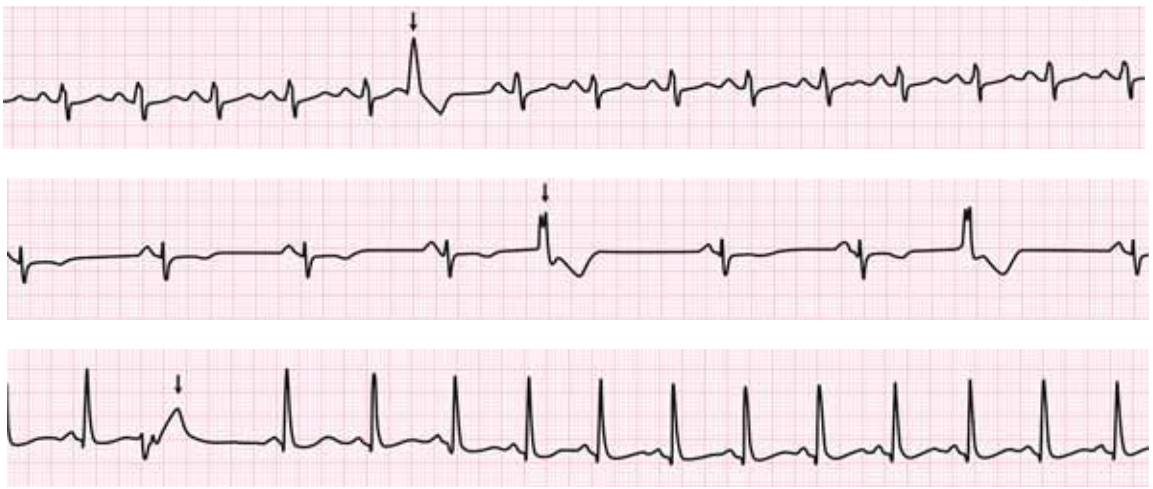


Figure 2.72: Different examples of VPCs. Note broad and bizarre shaped QRS complexes (arrow) and full compensatory pause after VPC

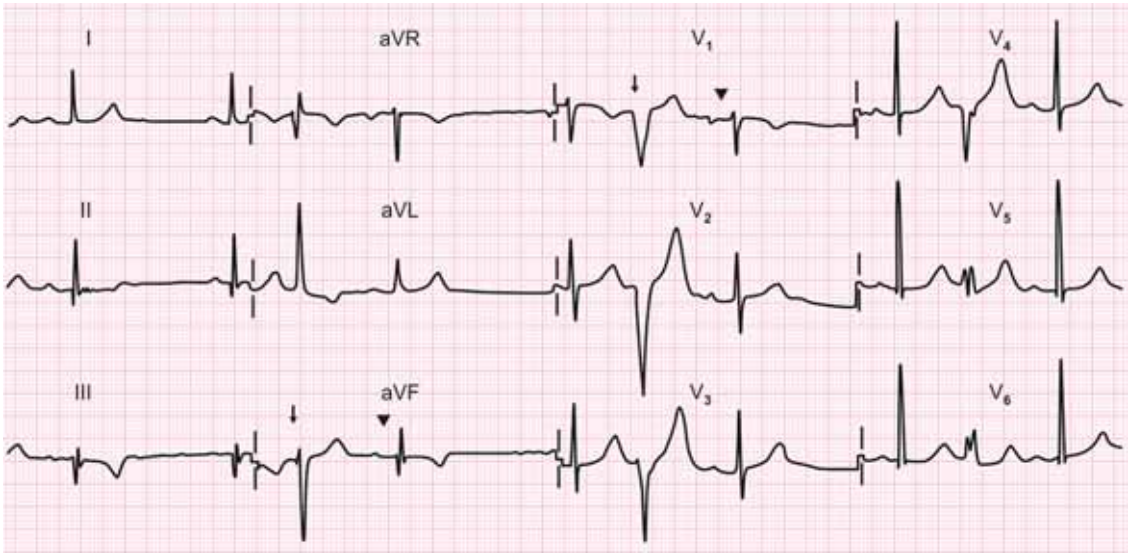


Figure 2.73: Interpolated VPCs (arrows). Note that next beat after interpolated VPCs have prolonged PR interval (arrow head). This is due to retrograde conduction into AV junction and slow down the next beat conduction

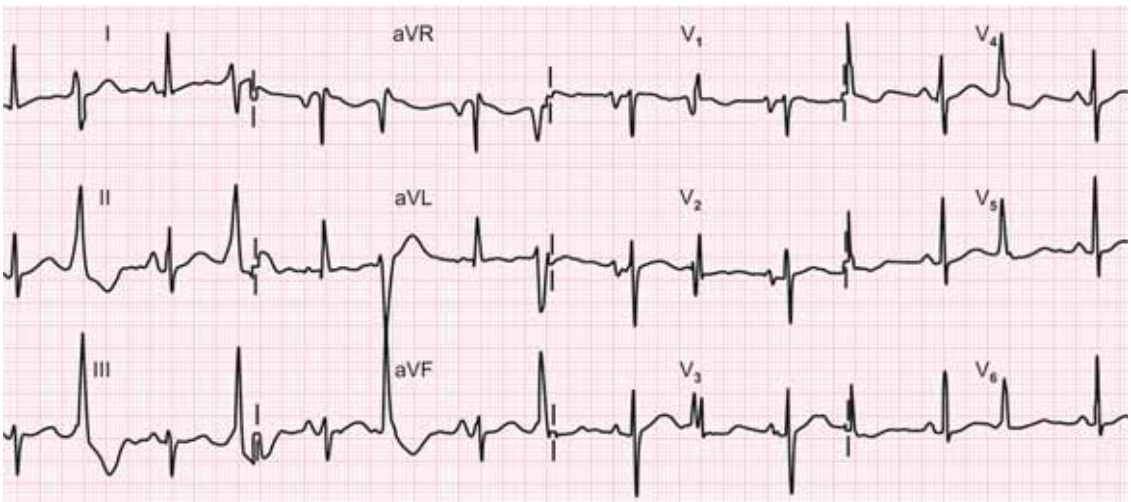


Figure 2.74: Ventricular premature beats. Note bigeminal pattern

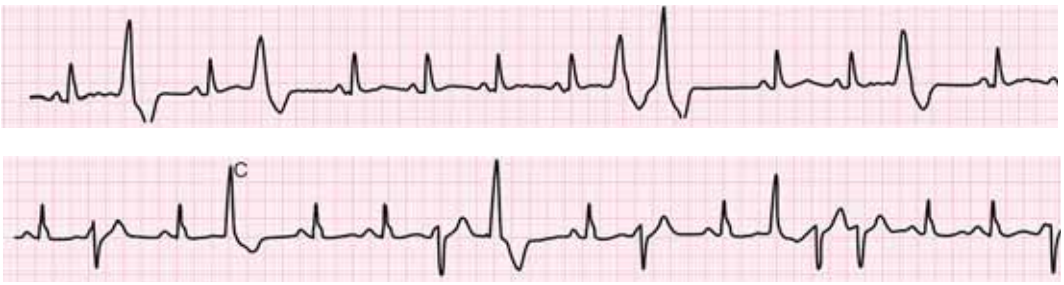


Figure 2.75: Multifocal VPC. Note different morphology of VPCs indicating their origin from different foci in the ventricle

Wandering Atrial Pacemaker (Figs 2.76 and 2.77)

Sometimes multiple ectopic foci in the atrium drive heart together (instead of emitting occasional single scattered beats)

1. Different forms of P wave and PR interval (Because different foci takes different pathways)
2. Average rate is 60–100 bpm.
3. QRS are usually identical to sinus rhythm.

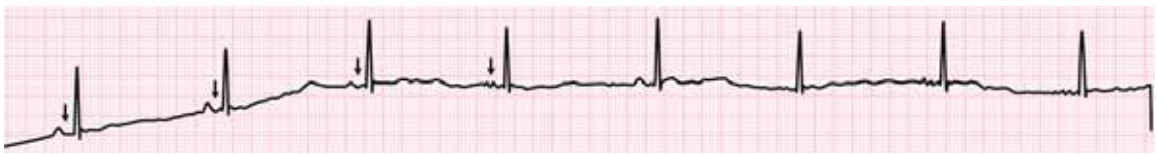


Figure 2.76: Wandering pacemaker. Note the different morphology of P wave (arrows). Here the impulse generating focus moves gradually away from the sinus node therefore the first P wave looks more like sinus P wave and as the focus moves away from the sinus node. It gradually becomes less similar to sinus P wave. This is because the location is within the SA node (or just adjacent to it)

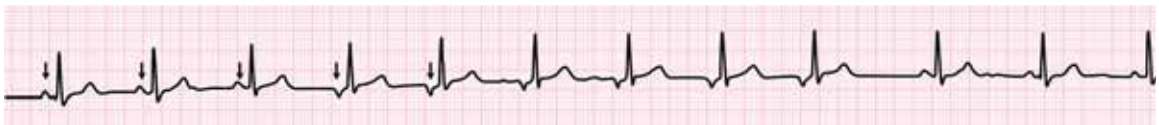


Figure 2.77: Wandering pacemaker. Note different morphology of P wave (arrow) and variable PR interval; the location is near AV node

GET FAMILIAR WITH TACHYCARDIA

The basis of tachycardia may be classified on ECG finding as shown in Table 2.3.

Table 2.3: Common ECG causes of tachycardia and their classification

Classification of tachycardia	
Narrow complex tachycardia	Wide complex tachycardia
Regular	Regular
Sinus tachycardia	Ventricular tachycardia (VT)
Atrioventricular nodal re-entry tachycardia (AVNRT)	Supraventricular tachycardia with bundle brand block (BBB) or pre-exciting syndrome
Atrial flutter (fixed AV conduction)	
Atrial tachycardia (paroxysmal/nonparoxysmal)	
Atrioventricular reciprocating tachycardia (AVRT)	
WPW syndrome (orthdromic circus movement)	
Irregular	Irregular
Atrial fibrillation	Atrial fibrillation with BBB or with WPW antidromic syndrome
Multifocal atrial tachycardia	Torsade de pointes

Narrow Complex Tachycardia

Sinus Tachycardia

1. Most common cause of narrow complex tachycardia (100–130 bpm). See Table 2.4 for range of rates in atrial rhythms.
2. May mimic SVT but carotid sinus massage (which in turn slow down heart rate) can reveal the P wave.
3. See features of sinus tachycardia described before (see page 73).

Atrioventricular Nodal Re-entrant Tachycardia (AVNRT) (Nearly 90% of SVT You See in Emergency Room—ER)

This is the most common of supraventricular narrow complex tachycardia (victims are mainly female). Therefore, it is better to take time to understand how ECG presents its typical feature. AV node has fast tract with long refractory period and slow tract with short refractory period. Normally the impulse passes through fast tract and depolarizes ventricle.

1. In AVNRT (common type > 50%); (Slow-fast type); (Anterograde impulse through slow tract and retrograde impulse through fast tract). The main impulse passes through the slow path. Part of it returns back utilizing fast tract. After reaching in atrium, part of it activates atrium and part of it goes onto stimulate ventricle. Since re-entry current is using the fast pathway, it is almost simultaneous with ventricular depolarization and the resultant P wave is usually not visible and buried in the QRS (Most common presentation 50%).

2. Sometimes (45% of cases) the P wave caused by this re-entrant current finds place in the end of QRS distorting terminal QRS forces. The result is Pseudo S wave in leads II, III, aVF and r' wave in V_1 in RSr wave mimicking RBBB. This is pathognomonic ECG presentation of AVNRT. (The rhythm can be slow even terminated by vagal maneuvers like carotid sinus massage).
3. *Note:* This abrupt termination is characteristic of AVNRT.
4. Even fewer 5 percent cases, this P wave falls in initial QRS can be seen as pseudo Q wave in leads II, III, aVF.

How it Initiates

The culprit is usually atrial premature beat (APB) following in such a phase of time when fast tract is refractory hence does not allow further stimulation therefore it utilizes the slow path. This process may repeat again and again to give common type of AVNRT.

Note: Since it uses slow pathway PR of this APC is prolonged.

AVNRT Common Type (Slow Fast)

ECG Features

Narrow complex tachycardia with:

1. The rate usually 150 to 220 bpm
2. QRS < 0.12
3. In 50 percent of cases, retrograde P wave is usually hidden in the QRS and cannot be seen.
4. In 45 percent of cases, Pseudo S in leads II, III, aVF and Pseudo r' (r'sr) in V_1 (Pathognomonic) .
5. 5 percent cases are pseudo q wave in II, III, and aVF.
6. Often precipitated by an APC whose PR interval is prolonged.
7. Vagal maneuvers usually slow down or terminate this tachycardia (Figs 2.78 to 2.82).

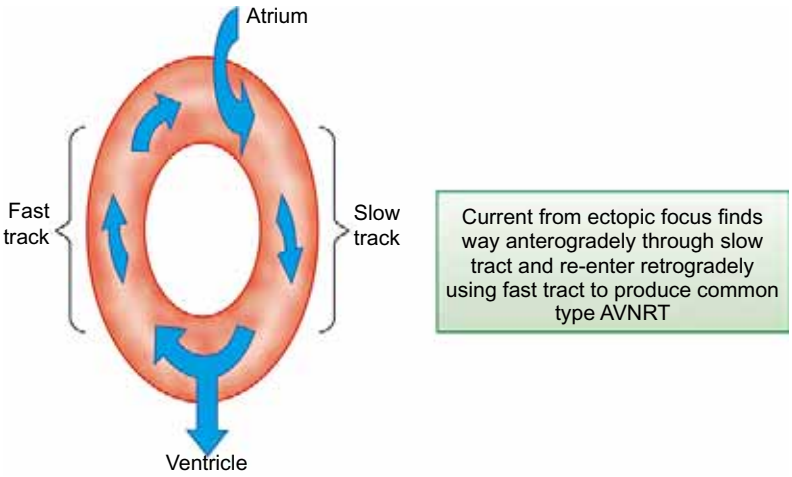
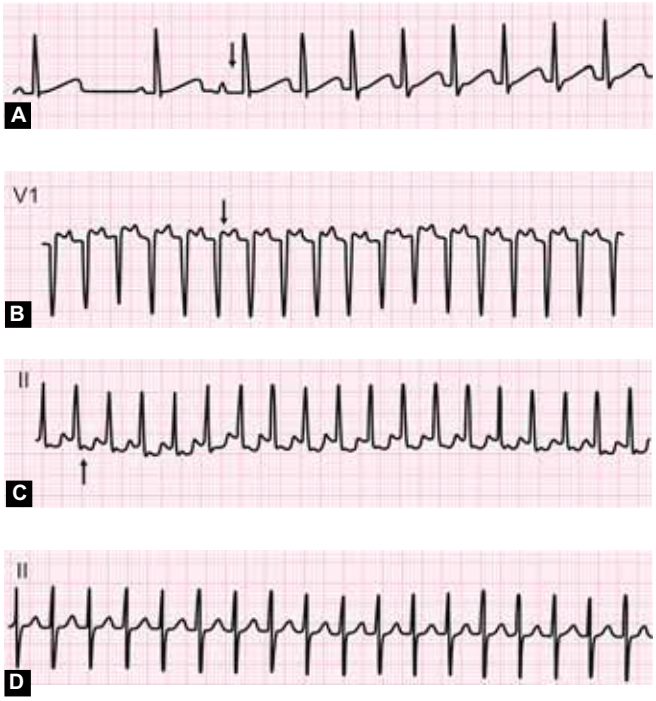


Figure 2.78: AVNRT note pathway of flow of current through slow fast pathway



Figures 2.79A to D: (A) AVNRT, common type (slow-fast). Arrhythmia is started by APC with prolonged PR interval (arrowhead). Pathognomonic findings are rate 150–230 bpm, Pseudo r' wave in lead V_1(B arrow) pseudo s wave in leads II, III and aVF (C arrow) which are actually P wave distorting terminal QRS. P wave may not be visible (D)

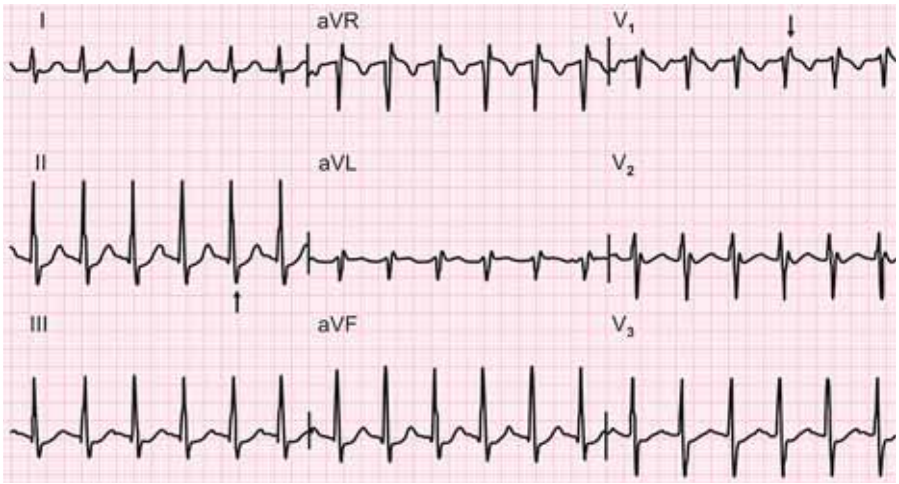


Figure 2.80: AVNRT (after rate slowed down). Note pathognomonic findings. Pseudo r' wave in lead V_1 (arrow) and pseudo S wave (accentuated S wave) in lead II, III and aVF (arrow)

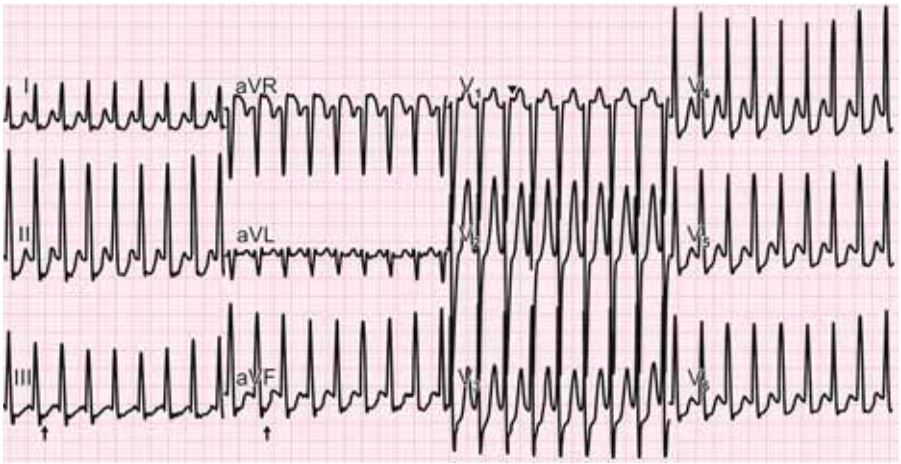


Figure 2.81: AVNRT common type (slow fast) with typical presentation. Note pseudo r' wave in lead V_1 (arrowhead) and pseudo S wave in lead II, III and aVF

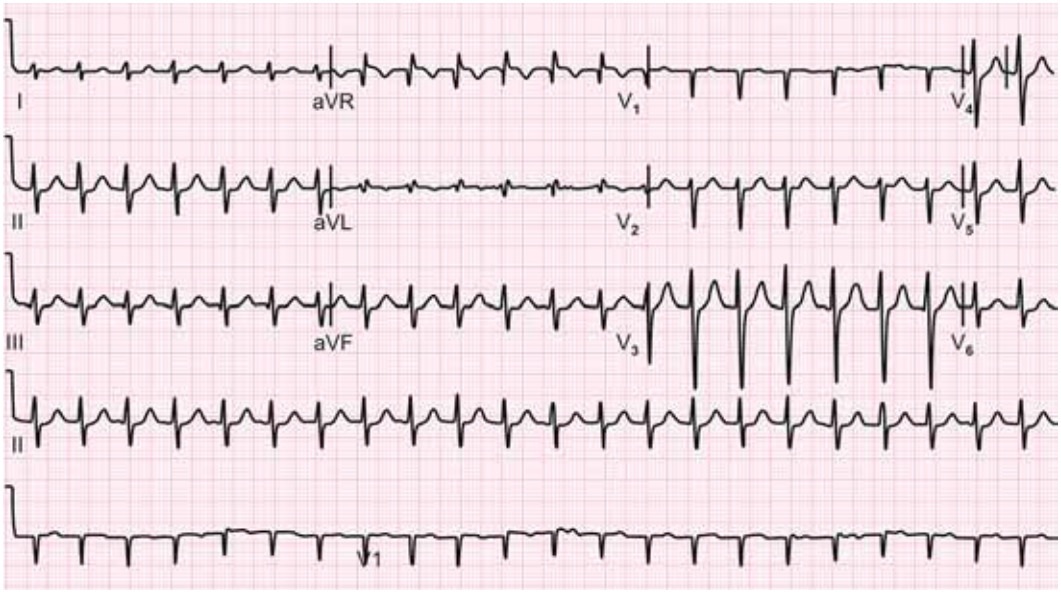


Figure 2.82: AVNRT common type (slow fast) with hidden P wave. Note narrow QRS complex and regular rhythm

AVNRT Uncommon Type (Fast Slow)

Here the anterograde current passes through fast pathway (long refractory pathway) and return via slow pathway. Therefore, RP interval is prolonged because the impulse which causes P wave takes the slow path. It is far from QRS and not distorting it like in common type and P wave negative in leads II, III and aVF. This may initiate by APC or de novo. This cannot be differentiated from rare type of WPW circus movement tachycardia.

Features (Figs 2.83 to 2.85)

1. Narrow complex tachycardia with rate may be slower than common type: AVNRT (150 to 100)
2. QRS (0.12)
3. PR interval of the initiating P wave from APC (example) is not prolonged.
4. Retrograde P wave occurs well after QRS complex and RP interval greater than PR interval.

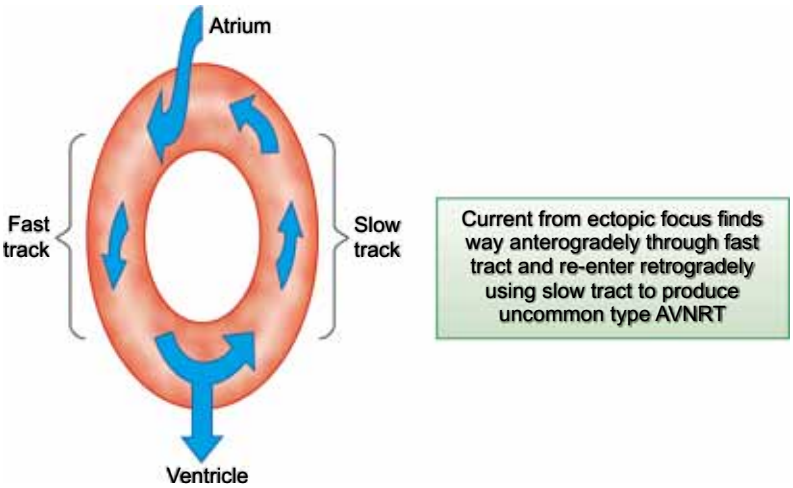
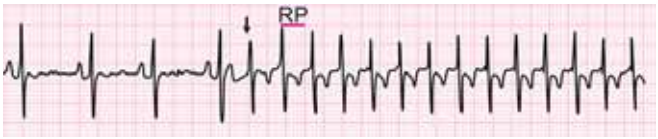


Figure 2.83: AVNRT uncommon type (fast slow). Note pathway of flow of current through fast and slow tracts



AVNRT. Uncommon type (Fast. Slow). The arrhythmia is started by APC (arrow). Note the negative P waves RP interval greather than PR, this is because retrograde impulse is conducted through slow track.

Figure 2.84: AVNRT uncommon type (fast slow). Note main ECG features

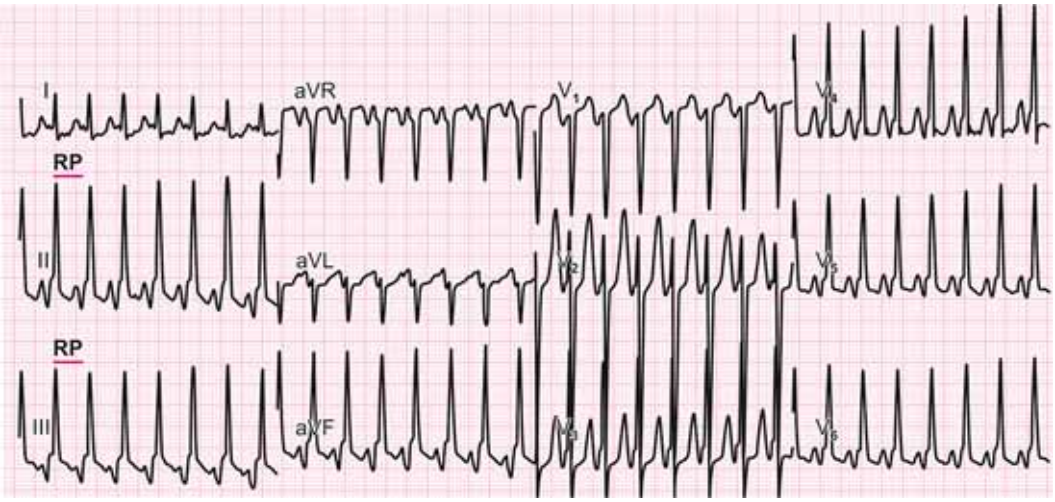


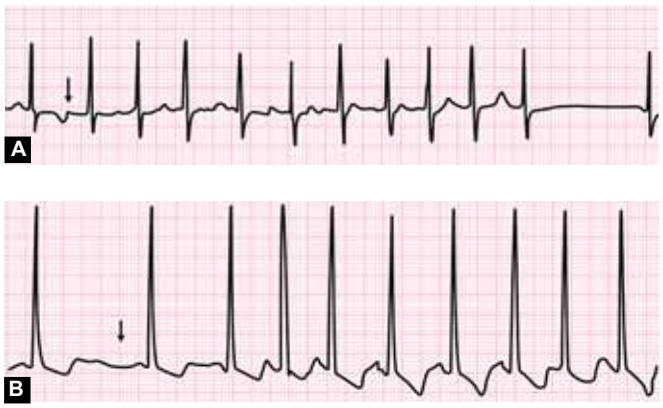
Figure 2.85: AVNRT uncommon type (fast slow). Note the regular rate (around 170 bpm) and RP interval is greater than PR interval. This is because the retrograde impulse passes through the slow tract producing retrograde P wave far from R wave

Paroxysmal Atrial Tachycardia (PAT)

Sometimes AT starts from an APC and stops abruptly, this is called as paroxysmal atrial tachycardia (PAT).

Diagnostic Features (Figs 2.86A and B)

- A run of 6 or more unifocal APC in succession with:
  a. Rate between 140 to 250/min.
  b. Regular P-P cycle.
  c. Ectopic P wave may be of different form (depends on the ectopic location generating APC), therefore, may be inverted, biphasic, isoelectric or difficult to identify. QRS usually resemble that of normal sinus rhythm. There may be normal warm up period at the beginning (i.e. progressive increment in the rate initially)
  d. Morphology of ectopic P waves is different from sinus P waves which differentiate it from sinus tachycardia.



Figures 2.86A and B: Paroxysmal atrial tachycardia (PAT). Note (A) ectopic inverted P wave after which PAT started and stops after some beats (arrow). Also note different voltages of QRS complexes which is a strong clue which shows that the source of these beats are from atrium other than sinus node. B shows another example of isolated PAT arrow shows the starting point

PAT with AV Blocks

PAT with block is usually caused by a combination of digitalis toxicity and low serum potassium (90% of cases) this condition may be dangerous and important to recognize. In this situation AV conduction is delayed and not every APC finds chance to pass through AV node and activate. In this situation the PAT presents with AV nodal blocks (e.g. 2:1, two P waves for each QRS, 3:1, three P waves for each QRS, etc). Also note that type of block (like first degree, second degree or third degree block) is not important for diagnosis and treatment.

Diagnostic Features (Figs 2.87 and 2.88)

PAT may also present with BBB. One should be careful to search for the wave because broaden QRS may mislead to other causes of broad complex tachycardia.

- Regular P wave with same range of rate as in PAT without block (Between 140 to 250/min).
- Some P waves are blocked and not all P wave are conducted.

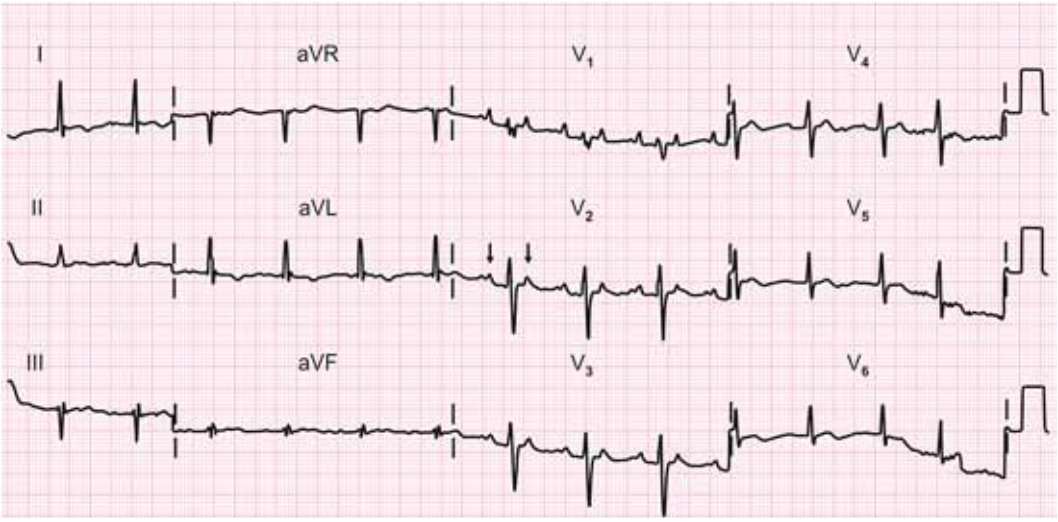
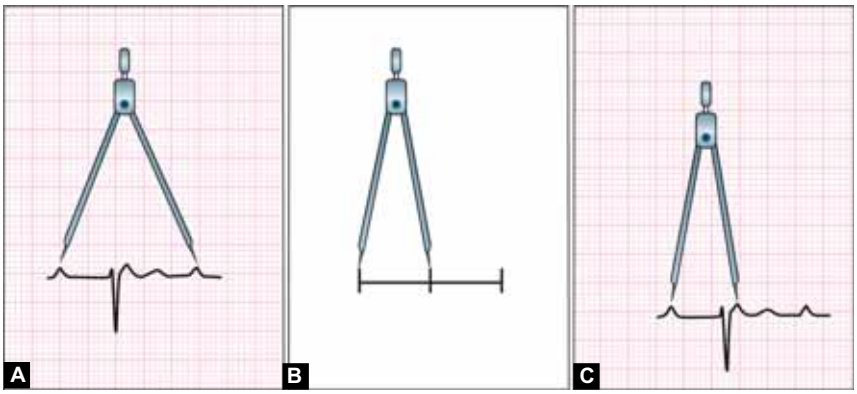


Figure 2.87: Characteristic finding of digitalis toxicity. ECG showing PAT with 2:1 block. Note prominent P waves in lead V_1 and V_2 (arrow).The P wave after the QRS should not be mistaken for T wave which would normally occur further beyond the R wave. You may also check this by using calipers



Figures 2.88A to C: Calipers can be used to calculate the intervals and hidden P waves such as in PAT with block. Use calipers to find apparent PP interval (A) and then calculate half of it by using ECG paper strips (B) and then check whether there is a buried P wave or not (C). Here a wave just after QRS complex with a distant just half of the prior PP interval is a P wave rather than T wave

Multifocal Atrial Tachycardia (MAT)

Diagnostic Points (Figs 2.89 and 2.90)

1. Contrary with PAT, here more than 1 ectopic foci generation APC, therefore, P waves vary in morphology and different PR interval
2. The rate is between 100–200 bpm
3. Rhythm is irregular (PR, RR, PP, etc. all irregular)
4. QRS usually resembles that of normal cardiac rhythm
5. MAT usually found in patient with COPD.

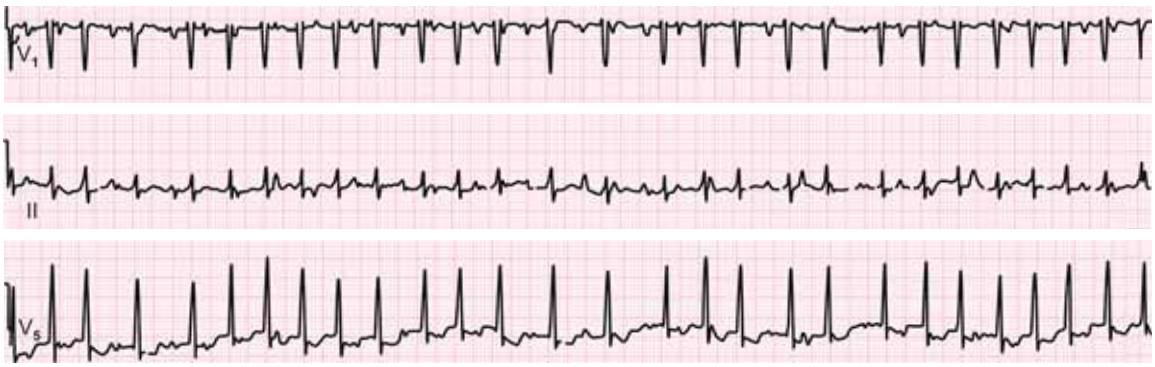
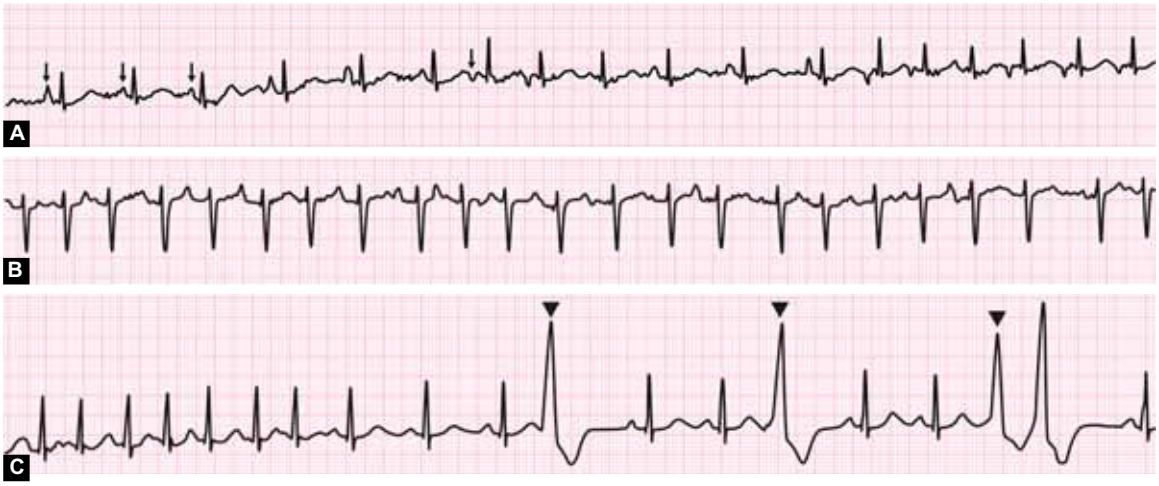


Figure 2.89: Different rhythm strips showing MAT. Note variable P wave morphology and PP interval resulting in irregularly and irregular rate



Figures 2.90A to C: Multifocal atrial tachycardia (MAT). This is suggested by the presence of multiple atrial foci with a rate greater than 100 bpm. Note different morphologies of P waves (arrow) within the same ECG strip different PP and RR intervals (A+B). This rhythm is frequently present in COPD. Also note VPCs (C) (arrowheads)

Atrial Flutter (AF)

Mechanism of atrial flutter is not fully understood, one theory is that it is due to repetitive firing from a focus in atria. The rate of this repetitive firing is high (220 to 350). Thus these waves inverted "flutter waves" replace P waves.

Diagnostic Points

It can be identified by following features:

- Presence of saw tooth like pattern which are actually F waves replacing P waves.
- Atrial rate (F-F cycle usually regular) is 220–350.
- QRS usually resembles that of normal cardiac rhythm.

Supporting Points (Figs 2.91 to 2.95)

- There is usually 2:1 conduction, there for two F for one QRS and thus ventricular rate is half of the atrial rate. (if the rate is too high, it can be slowed down by carotid massage).
- The saw tooth pattern is best seen in leads II, III, aVF and V_1. The downward deflection of the F waves has a gradual slope followed by an abrupt upward deflection (hence the saw tooth)
- Contrary to this, lead I, V_5, V_6. Almost no atrial activity, the F waves are heard to see.
- F waves typically show negative deflection in lead II, III, aVF and in V_1 may be positive.

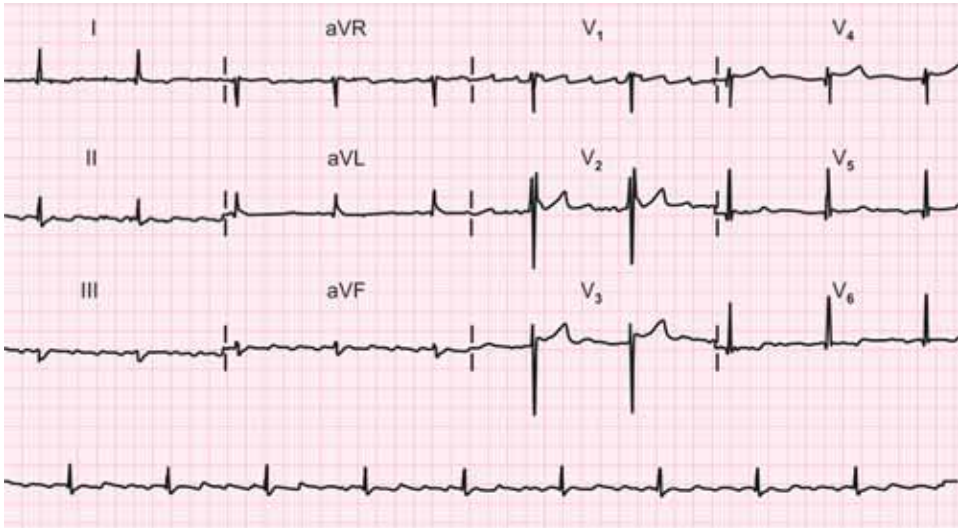


Figure 2.91: Atrial flutter with 4:1 conduction. Note saw tooth pattern of waves. Also note negative flutter waves in leads II, III and aVF

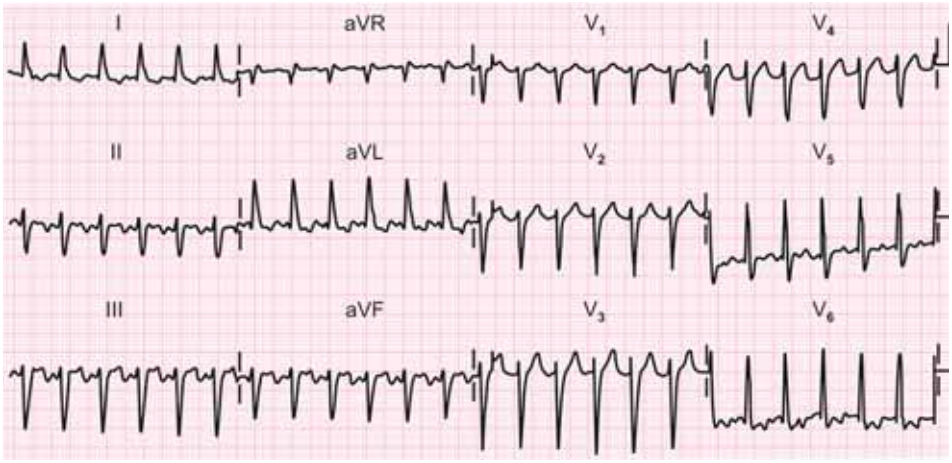


Figure 2.92: Atrial flutter or supraventricular tachycardia (of other types)? The negative P wave in leads II,III and aVF may be mistaken by T waves, but a ventricular rate of 150 bpm alerts us to think of atrial flutter with 2:1 AV conduction. Slowing AV conduction by carotid sinus pressure or pharmacologically can unmask this and make identification easy (see other ECG)

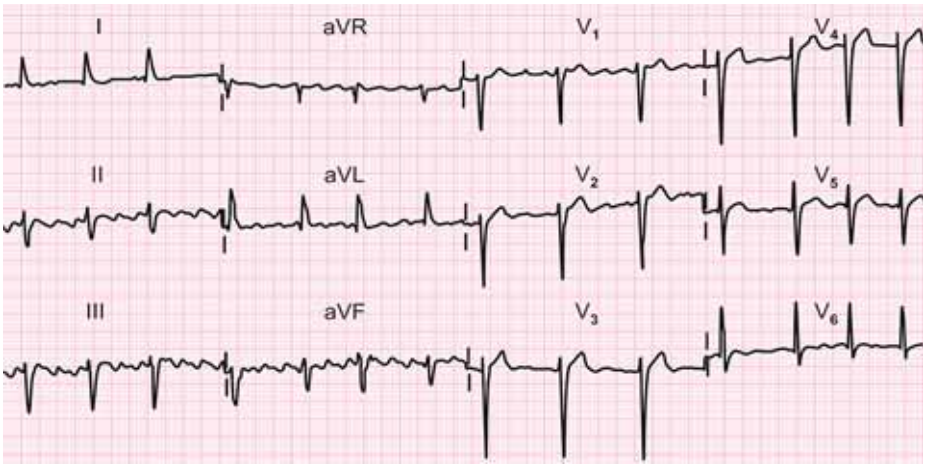


Figure 2.93: Same patient after administration of verapamil (which slows AV conduction). Now flutter waves are obvious

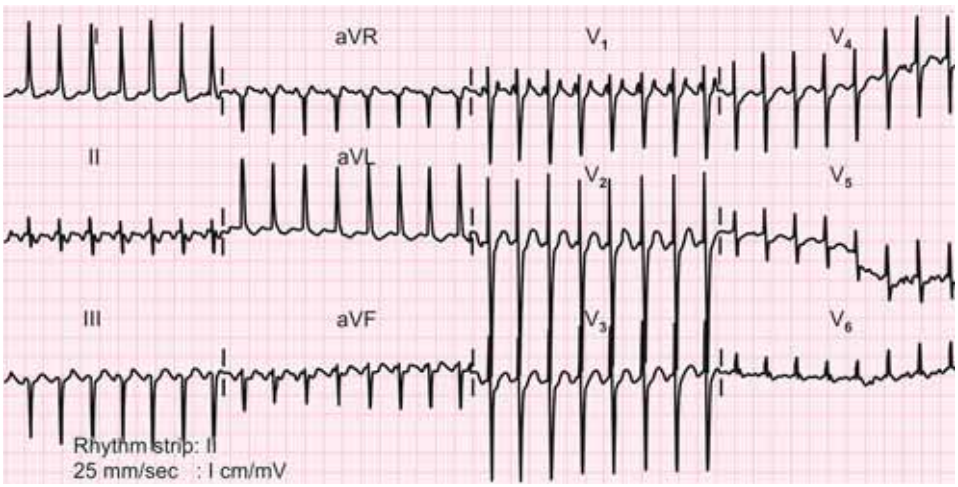


Figure 2.94: Atrial flutter. Atrial rate is rapid 280 bpm with 2:1 conduction. This block is physiologic due to the rate slowing property of AV node and not due to AV disease. Also note F wave negative in lead II, III and aVF positive in lead V_1

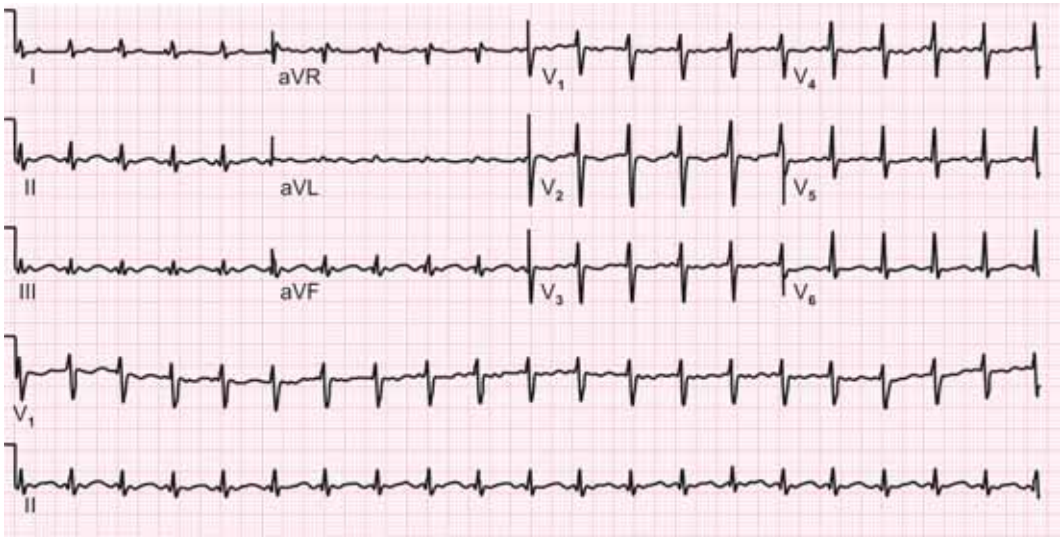
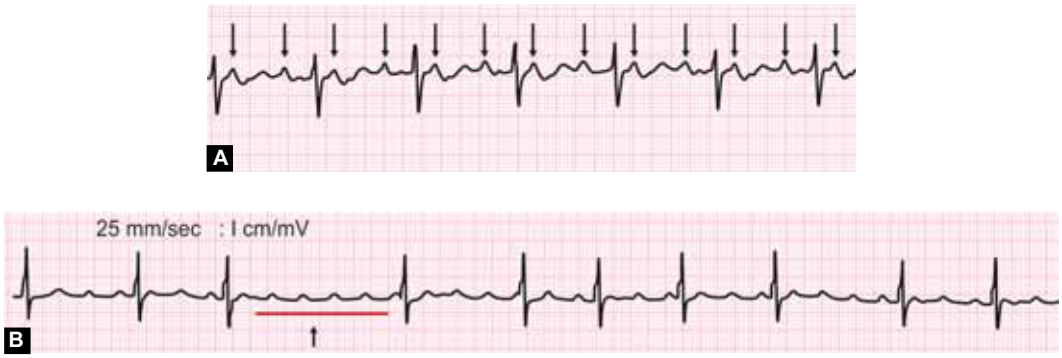


Figure 2.95: Atrial flutter with a 2:1 conduction, rate is 240 bpm which is somewhat slower than usual 250 to 350 bpm, which makes the diagnosis difficult, as here the F waves are superimposed with the QRS and beginning of T wave, also when F wave falls before QRS they simulate Q waves as seen in II

PAT and Atrial Flutter (Figs 2.96A and B)

Atrial flutter may resemble with PAT with blocks which can be differentiated from following features:

- Typical flutter waves as seen in atrial flutter is absent in PAT with block (in PAT there is isoelectric baseline between P waves).
- In PAT, rate rarely exceeds 250 bpm while atrial flutter is usually in the range of 300 bpm (In fact atrial rate 300 bpm and ventricular rate 150 bpm, i.e. 2:1 block should alert us to search for atrial flutter).



Figures 2.96A and B: Atrial tachycardia with a 2:1 block (A) and with variable AV conduction (B). This is characteristic for digitalis toxicity and can be differentiated from the atrial flutter where atrial rate is higher than 250 bpm and characteristic flutter waves that are inverted in leads II, III and aVF while in atrial tachycardia there is isoelectric baseline between the P wave (arrows)

PAT with Bundle Branch Block

PAT may also occur with bundle branch block but it can be identified easily by checking previous features and looking QRS morphology (Figs 2.97 and 2.98).

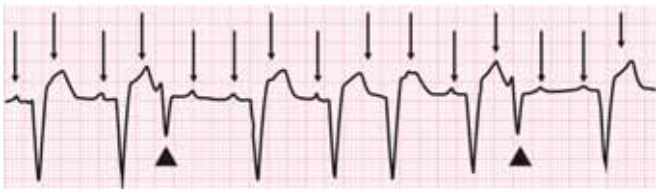


Figure 2.97: PAT with bundle branch block. Note broad QRS, regular rate of 150 bpm and more than one P wave for each QRS. P waves are ectopic (arrow). Note also there are two VPCs (arrowheads) interestingly with narrow QRS. In fact, VPCs in the setting of BBB appear with narrow QRS because of already abnormally widened QRS

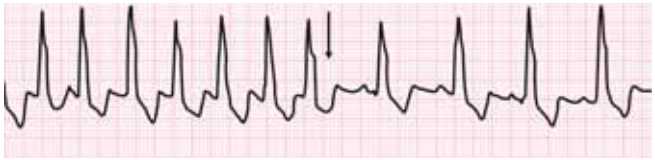


Figure 2.98: A short run of PAT. It may look like a run of VPCs but note that after reverting to sinus rhythm (arrow) the QRS complexes are still widened and shows bundle branch pattern

Atrial Fibrillation

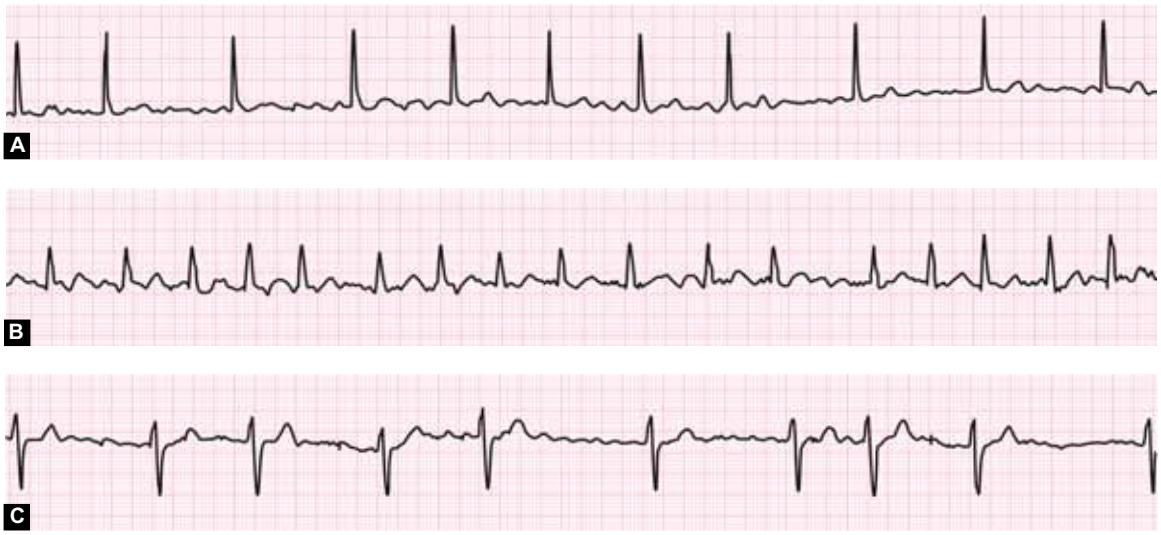
This narrow complex irregular tachycardia is result of impulses originating from multiple ectopic foci in atrium with a rapid rate of 350–650. This rapid rate results in loss of effective atrial motion and on ECG appears as undulations called Fibrillatory waves (f).

Diagnostic Points

1. Multifocal *f* waves, replacing P waves at a rate of 350–650 bpm (*f* waves are fine undulations).
2. Narrow QRS resembles normal cardiac rhythm but with irregular rhythm (said as irregularly irregular).

Supporting Points (Figs 2.99 and 2.100)

- *f* waves are best seen in lead V_1.
- QRS often varies in amplitude.
- Ventricular rate between 120–180 per minute. Atrial fibrillation can present with slow ventricular response and fast ventricular response (Figs 2.99A to C).
  Note: All impulses cannot pass through the AV nodes because AV node cannot conduct the current with high rate; these atrial impulses are called blocked.



Figures 2.99A to C: Atrial fibrillation with variable response. (A) Shows AF with a controlled ventricular response (75 bpm, average) (B) with fast ventricular response (116 bpm, average) and (C) shows AF with a slow ventricular response (60 bpm, average). Note obvious fibrillatory (*f*) waves in each strip

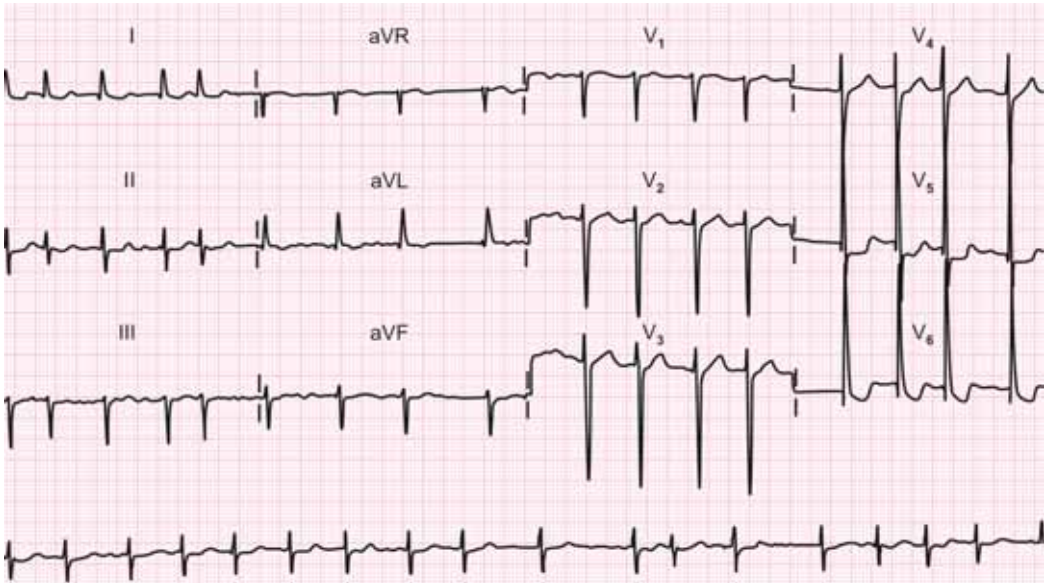


Figure 2.100: Atrial fibrillation. Irregularly irregular rhythm (rate = 105 bpm) and absence of identifiable P waves. Also note LVH by voltage criteria

ATRIOVENTRICULAR RECIPROCATING TACHYCARDIA (AVRT) OR AV PRE-EXCITATION TACHYCARDIA

What is Pre-excitation Syndrome?

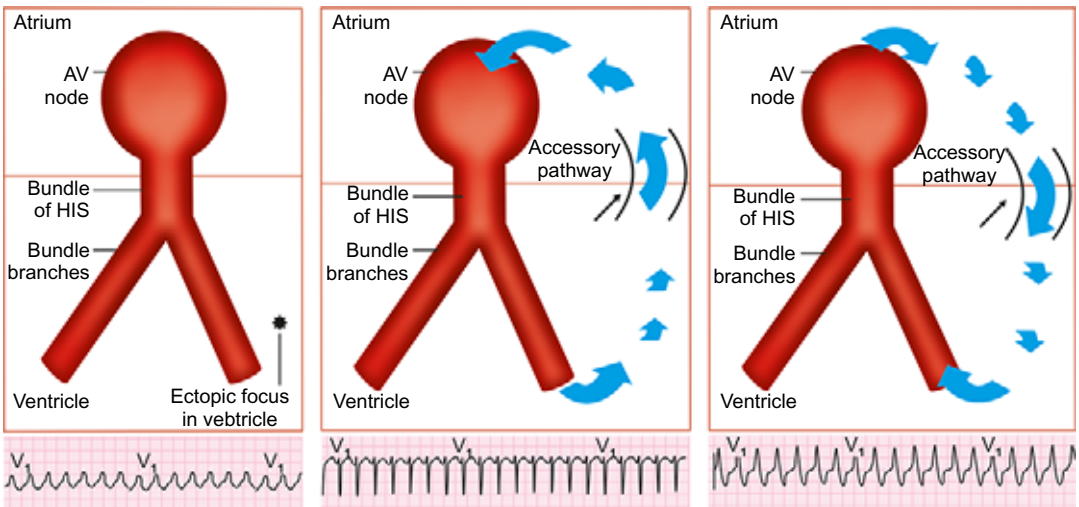
Sometimes besides the AV node there is an accessory pathway (may be more than one pathway), through which the current may bypass the AV node. Recall from physiology of heart the AV node has the following properties;

- It slows down the speed of the impulse traveling through atrium to ventricle (manifest as PR interval or physiological delay).
- If the atrial rate is too high AV node blocks some impulse and allows limited impulses to pass through it so the ventricular rate would not go too high (e.g. in atrial fibrillation the atrial rate is between 350–600 but the ventricular rate rarely exceeds 200 bpm).
- It is slowed by the vagal stimulation (like vagal maneuvers) and drugs like adenosine (all these properties can control the ventricular rate).

Accessory pathways lack these decremental conducting properties. In these condition the impulse may use these pathways to enter in the ventricle through AV node and re-enter in atrium through bypass tract (or

oppositely enter in the ventricle through bypass tract and re-enter in atrium through AV node) forming a self-perpetuating circuit; (Circus movement). Keeping in mind that accessory pathway lacks AV nodal properties, it is easy to understand that this situation may result in tachycardia of very high rates. This tachycardia is variably termed as "Atrioventricular reciprocating tachycardia" AVRT or "Atrioventricular pre-excitation tachycardia" or simply "Pre-excitation tachycardia". Wolff Parkinson syndrome (WPW syndrome) is a good example where accessory pathway can lead to such tachycardia.

When the current passes (anterogradely) through AV node and returns back (retrograde manner) using bypass tract to atrium it is termed as Orthodromic Circus movement or Reciprocating Tachycardia with Orthodromic conduction. When impulse passes (anterogradely) through bypass tract and comes back (retrograde manner) to atrium through AV node this is termed as Antidromic Circus Movement or Reciprocating Tachycardia with Antidromic conduction. This process may go onto produce "circus movements" and results in tachycardia. Since these pathways do not have the "filtering properties" as AV node, the rate of this tachycardia is usually very high. In fact, ventricular rate > 280 bpm almost always (99% certainty) due to pre-excitation syndrome, while rates >250 bpm need to exclude pre-excitation syndrome.

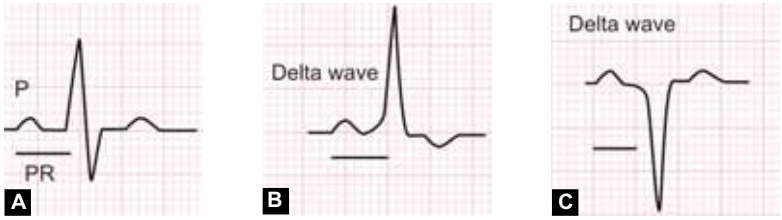


Figures 2.101A to C: Flow of impulse in different tachycardia and resulting ECG pattern. (A) Shows in VT ectopic foci within the ventricle generates impulse which results in VT; (B) Shows bypass tract (arrow) is used for retrograde impulse conduction in orthodromic circus movement; while (C) Shows bypass tract (arrow) is used for retrograde impulse conduction in antidromic circus movement

IMPORTANT PRE-EXCITATION SYNDROMES

Wolff-Parkinson-White Syndrome (WPW)

Here the bypass tract (called Bundle of Kent) is present in the atrioventricular groove which conducts the current directly to ventricle bypassing the AV node. Since this accessory tract lacks the above mentioned properties of AV node, conduction here is rapid resulting in short PR interval (<0.12 sec). Because it directly inserts in right or left ventricle, the normal pattern of depolarization is changed resulting in a slurred initial portion of the QRS complex which is famously known as delta wave. This delta wave may be positive or negative (sometimes this negative delta wave may appear as pseudo Q wave in limb leads mimicking MI) (Figs 2.102A to C). Note WPW syndrome can present with two different types depending upon the anatomical localization of bypass tract (WPW A, WPW B), resulting in differently looking ECG.



Figures 2.102A to C: Delta wave in V_1. Note normal P wave and PR interval (A) and decreased PR interval and delta wave (arrow, B and C). Note C present with negative delta wave

WPW A (Figs 2.103A and B)

Here the accessory tract inserts in the left ventricle (current being bypassed to left ventricle skipping right bundle produces right bundle branch like pattern in V_1 except that QRS is usually narrow than actual BBB). Delta wave seems to lead into a tall and broad R wave especially in lead V_1 and V_2. Note this type is far common (nearly 90%) than type B (Figs 2.103A and B). Here the accessory pathway is supposed to be located in the left posteroseptal area.

WPW B (Figs 2.103A and B)

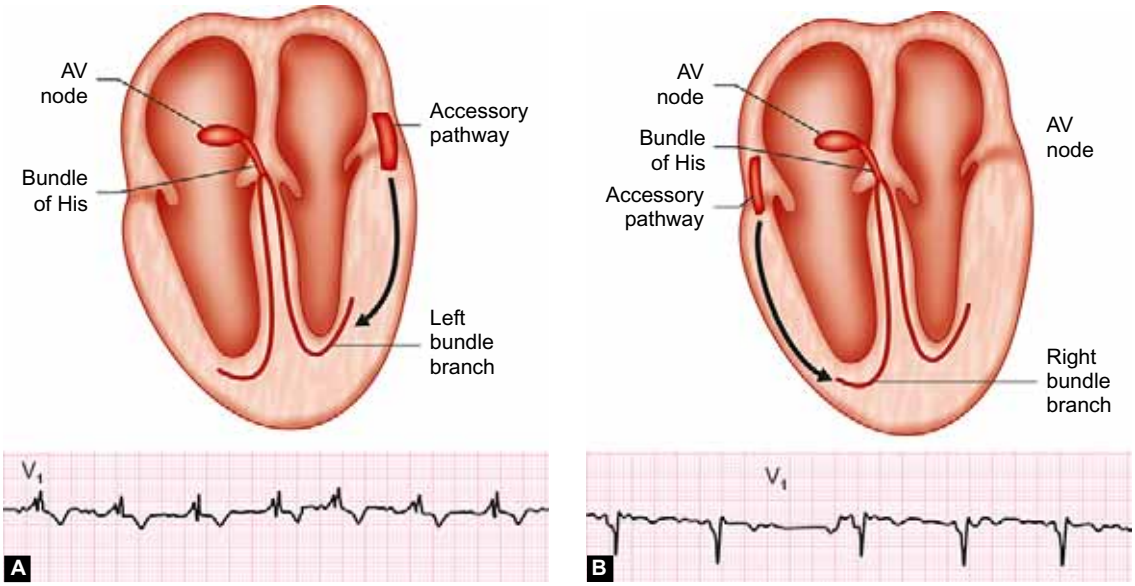
Here the accessory pathway inserts in right ventricle flooding current to right ventricle (while skipping left bundle) will produce left bundle branch pattern in lead V_1 with a negative delta wave. There is usually deep-broad S wave. Here the accessory pathway is supposed to be located in right-anterior area.

- *Note:* In both situations since there is not a true or complete block the QRS is not as wide as complete block usually > 0.11 sec. May be ≤ 0.12 sec especially in tachycardia (Antidromic movements).

How to Identify WPW Syndrome? (Figs 2.104 to 2.108)

When sinus rhythm, WPW syndrome. Is identified by:

1. Short PR interval < 0.12 sec
2. Presence of delta waves
3. In WPW A syndrome. QRS may mimic RBBB with tall R, V_1 to V_2.
4. In WPW B syndrome. QRS mainly negative in V_1–V_3 and resembles LBBB (positive in V_5–V_6)



Figures 2.103A and B: Schematic presentation of WPW bypass tract type A and type B. Note in type A the tract connects directly to left bundle and presents RBBB pattern in lead V_1(A) while in type (B) which is rare tract connect with right bundle branch and present with LBBB pattern in lead V_1 (B)

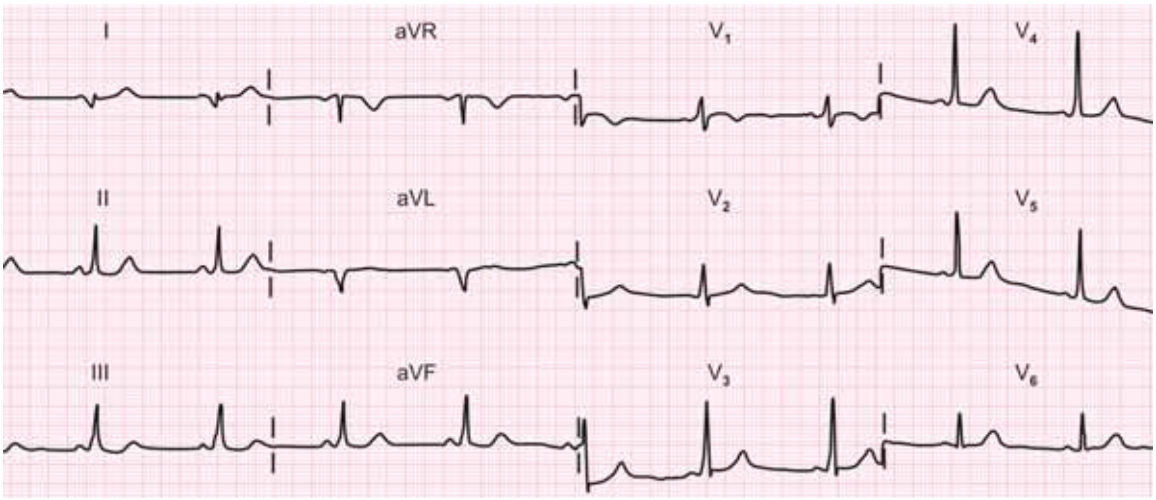


Figure 2.104: WPW syndrome. Note positive delta wave in inferior and precordial leads while negative delta wave in leads I and aVL producing pseudo q wave (pseudoinfarction) pattern. Also note tall R wave in leads V_1 and V_2

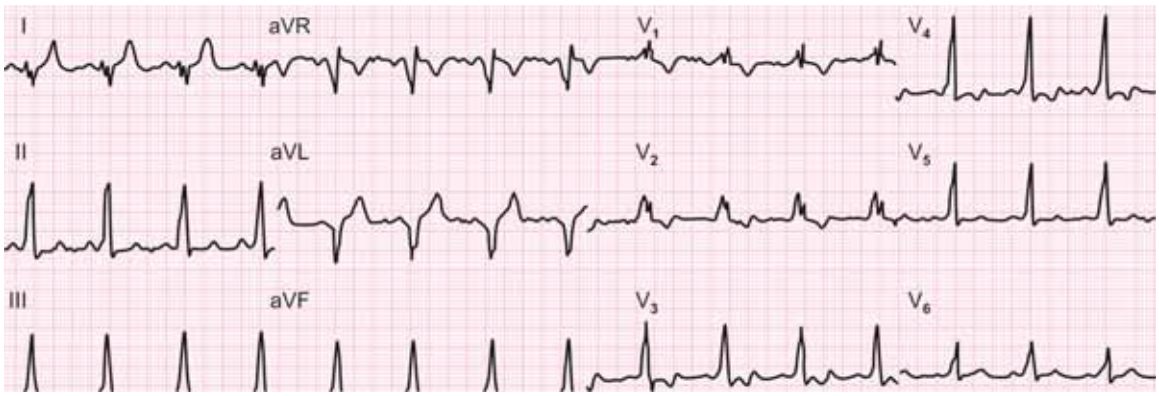


Figure 2.105: WPW syndrome type A syndrome. Note that QRS mimics RBBB with tall R in leads V_1 – V_2

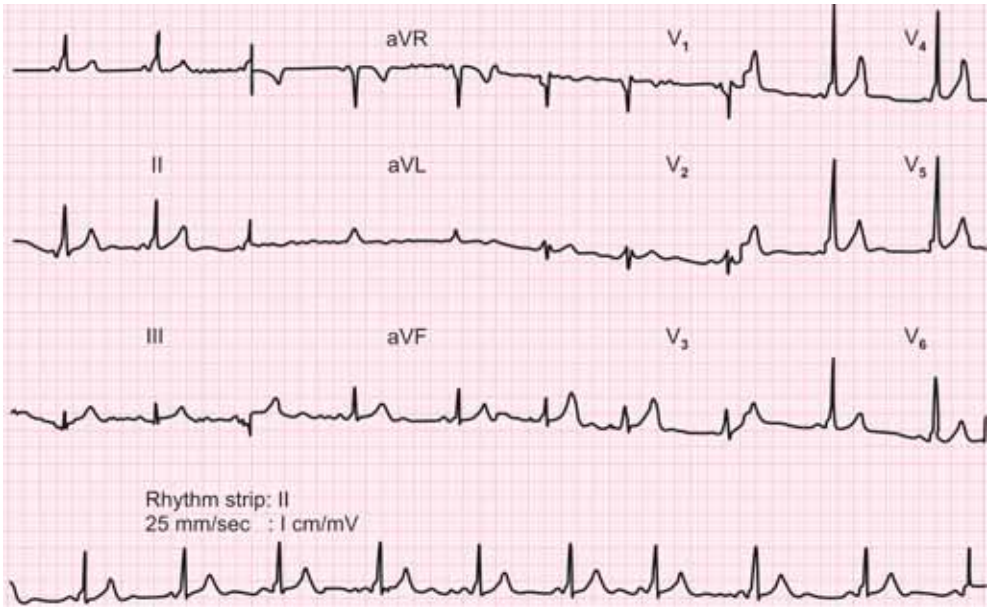


Figure 2.106: WPW syndrome type B syndrome. Note that QRS mainly negative in leads V_1–V_3 and resembles LBBB. Also note that QRS is positive in leads V_5–V_6

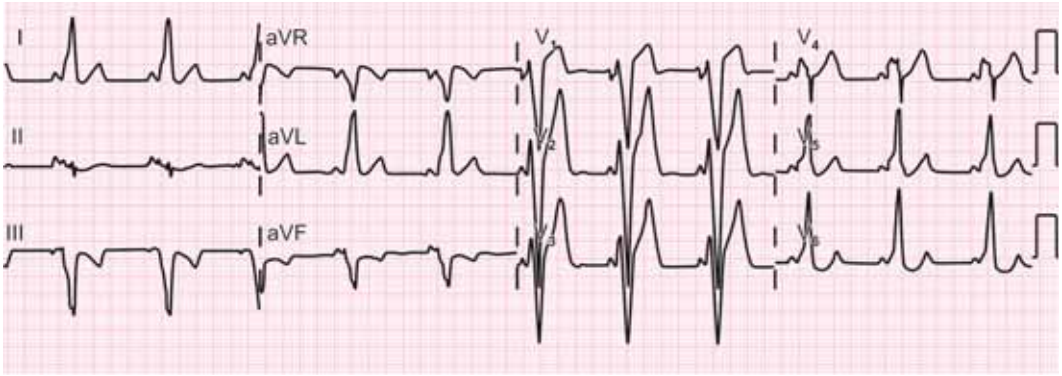


Figure 2.107: WPW syndrome mimicking LBBB (or anterior MI, see ST elevation in precordial leads). Short PR interval and delta wave is obvious in leads I, aVL, V_5 and V_6

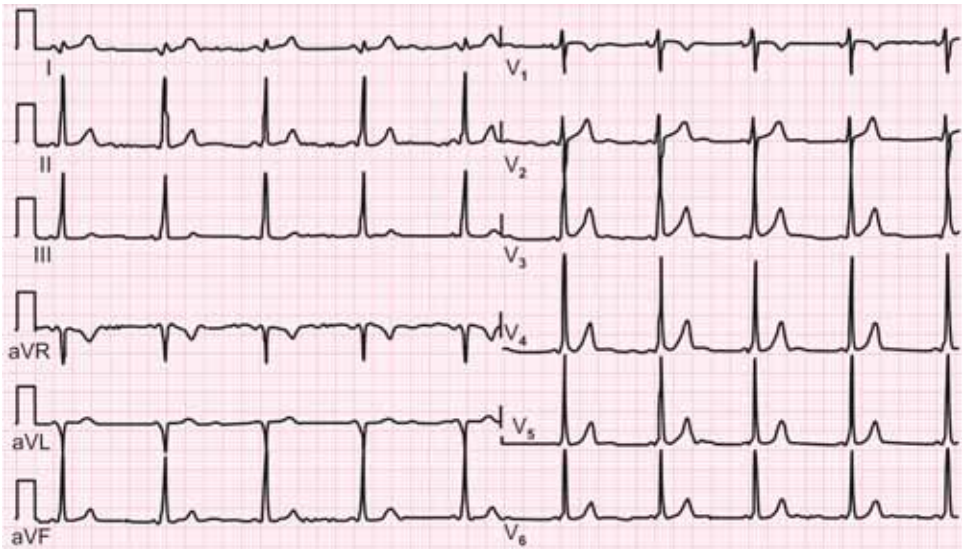


Figure 2.108: WPW syndrome, right lateral pathway. Note short PR interval and delta wave

WPW Syndrome as Pre-excitation Tachycardia

WPW syndrome can lead to tachycardia (pre-excitation tachycardia, or AVRT). This can be orthodromic circus movement tachycardia, atrial fibrillation or antidromic circus movement tachycardia. Orthodromic circus movement is most common, atrial fibrillation is the deadliest while antidromic movement is the least common presentation.

Orthodromic Circus Movement Tachycardia

A critically timed premature atrial beat that occurs during the refractory period of accessory pathway typically initiates orthodromic circus movement tachycardia.

Diagnostic Points (Fig. 2.109)

- Most important clue is the rate. Rate > 250 (according to some authors 230 bpm) is a strong clue for pre-excitation pathway (>280 bpm almost certain clue).
- QRS is narrow
- Usually circus movement is regular.
- Delta wave pattern is not identifiable because the current passes through the AV node anterogradely therefore, no delta wave is visible.
- Retrograde P wave due to retrograde impulse negative in lead II, III, aVF and positive in aVR, aVL (WPW type A).
- Since retrograde impulse is through accessory pathway which is usually fast, the RP interval is shorter than PR interval.
- Electrical alternans (alternating increasing or decreasing voltage of QRS) may be seen, which shows that there are more than pathways for AV conduction.

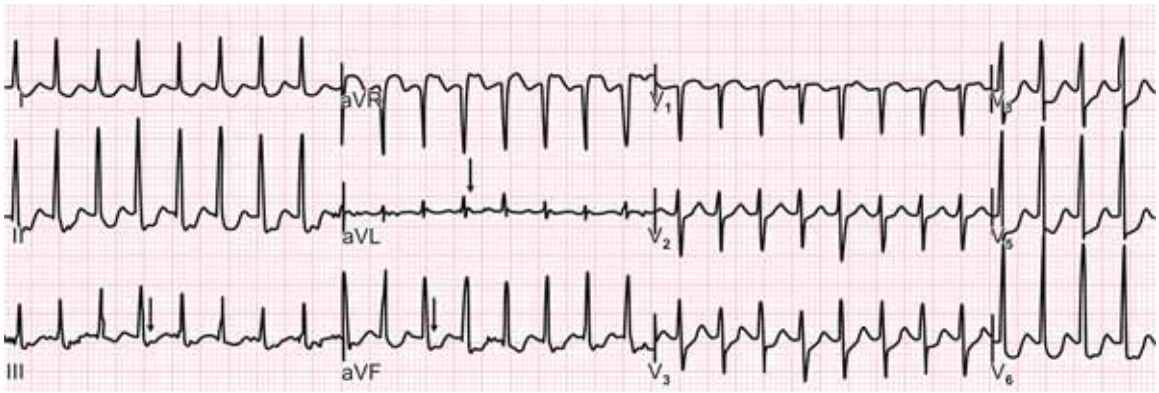


Figure 2.109: Orthodromic AVRT in a patient with WPW syndrome. Note presence of many diagnostic features, viz, rate >200 bpm, narrow QRS complexes, regular rhythm, retrograde P wave (arrows) with negative polarity in leads II, III and aVF(arrow) and with positive polarity in aVL (arrow) and RP interval shorter than PR interval

WPW with Atrial Fibrillations

In atrial fibrillation without preexcitation the passage is through AV node only, which keeps the ventricular rate within limits (usually 150, see Table 2.4 for differential diagnosis of atrial ectopic rhythm rates) and this conduction can be slowed by vagus (by carotid massage) or by drugs. (Like adenosine or digitalis). The case is different where atrial fibrillation is present with pre-excitation syndrome like WPW hence rate can go very

high. Therefore, it is important to differentiate between them. Following features help diagnosis (Figs 2.110 and 2.111).

1. Rate > 250 bpm (most important clue)
2. Bizarre wide complex QRS - irregular

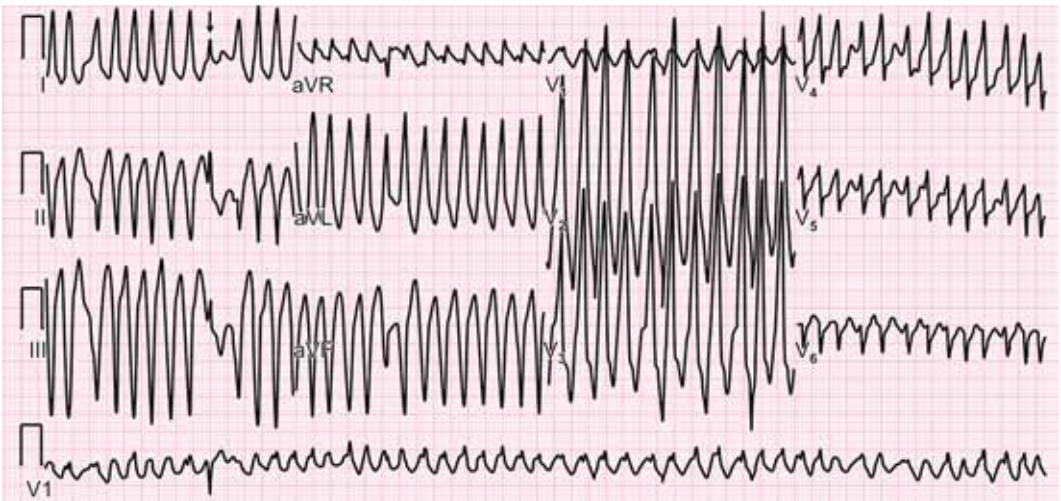


Figure 2.110: Almost diagnostic ECG of atrial fibrillation with WPW syndrome. Characteristic features are irregular ventricular response due to AF, Rate > 250 bpm, bizarre wide QRS complexes (note different width and amplitude = electrical alternans) and irregular response due to variable degree of pre-excitation. Note normal QRS complex (arrow). Differentiation of AF using AV node and using bypass tract is important because of different management

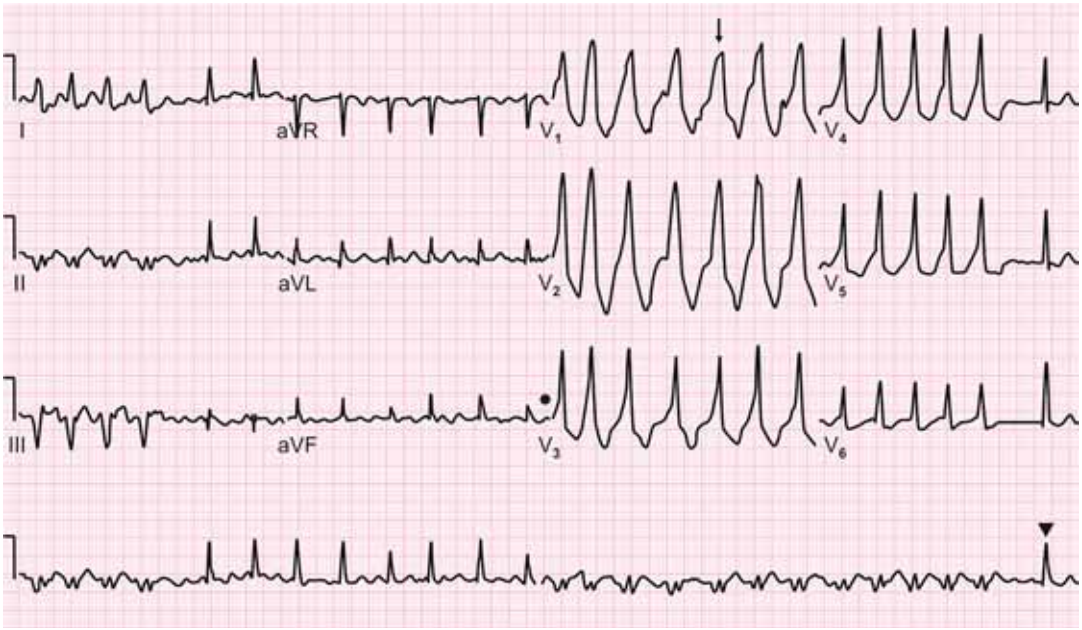


Figure 2.111: WPW syndrome with AF. Note irregularly irregular tachycardia impulse from the atrium are conducted to the ventricle via either both the AV node and accessory pathway producing a broad fusion complex (arrow) or just the AV node producing a narrow complex (without a delta wave, arrow head) or just the accessory pathway producing a very broad "pure" delta wave (asterisk)

Antidromic Circus Movement Tachycardia

Here the anterograde current to ventricle passes through accessory pathway and retrograde to atrium through AV node. Since current passage to ventricle is abnormal it causes broad complex tachycardia. Remember every broad complex regular tachycardia is ventricular tachycardia until proven otherwise (*See* Table 2.4).

Diagnostic Points (Figs 2.112 to 2.114)

- Here the anterograde current passes through the accessory pathway (not by AV node). Therefore, the QRS is wide (mimic VT).
- The rhythm is regular.
- Delta wave may be seen.
- Retrograde P wave (if seen) passes through AV node and RP interval > PR interval.

Table 2.4: Comparison of common causes of wide complex tachycardia and their ECG patterns

Atrial fibrillation with WPW syndrome	*Polymorphic ventricular tachycardia*	*Atrial fibrillation with bundle branch block*
Rate > 200 bpm	Rate > 180–280 bpm	Rate < 180 bpm
Irregular rhythm	Irregular rhythm	Irregular rhythm
Bizarre QRS	Bizarre QRS	Wide QRS showing BBB and minimal if any changes in morphology
Stable axis	Undulating axis	Stable axis

Note: Bizarre means here changing QRS width and amplitude.

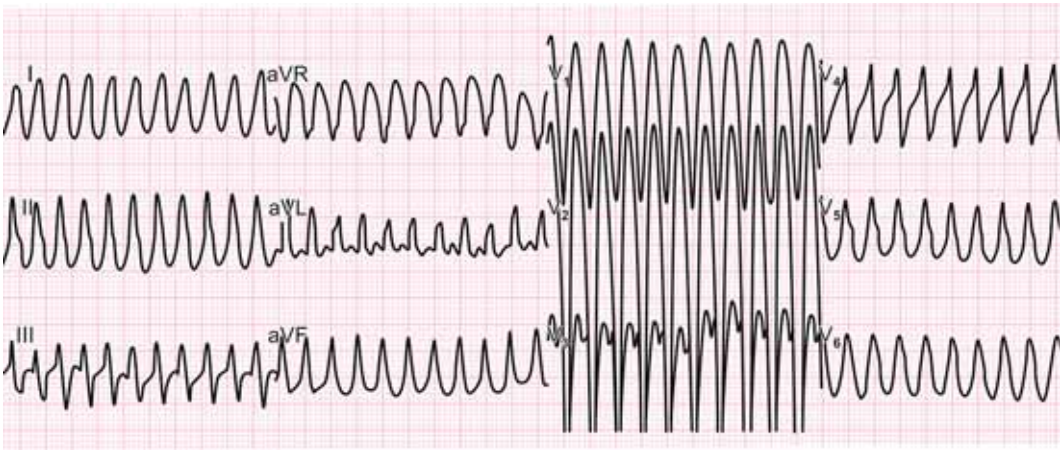


Figure 2.112: Antidromic AVRT (baseline rhythm was WPW syndrome). Although the differentiation from VT is difficult but it can be possible by comparing previous ECG and some identifiable features in ECG (not always present) like rate around 200 bpm, stable axis, delta waves (arrow) (See Table 2.4).

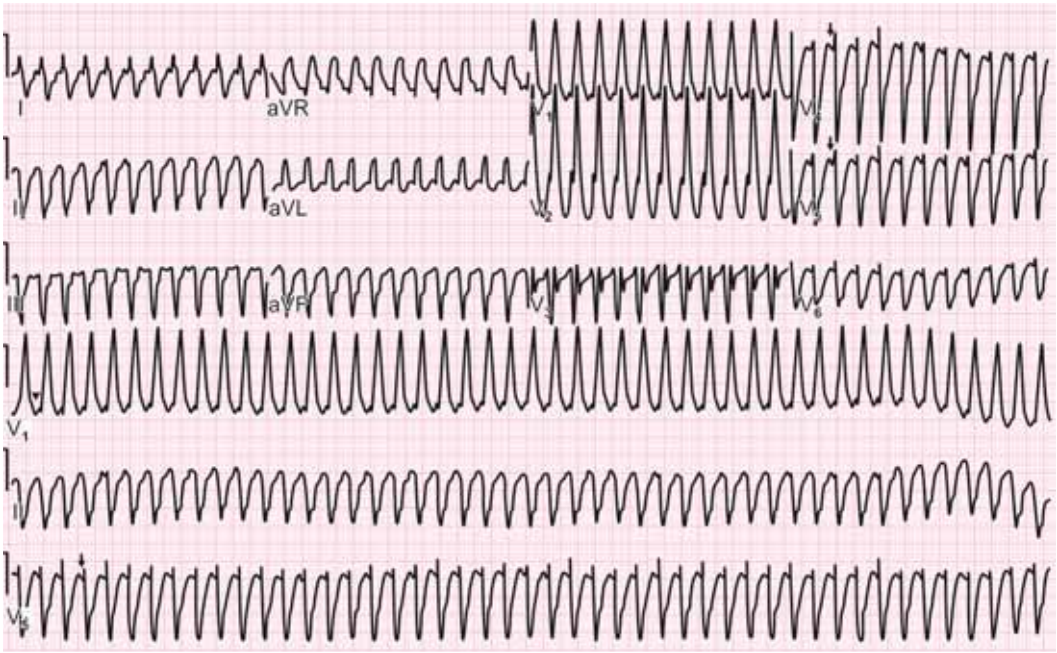
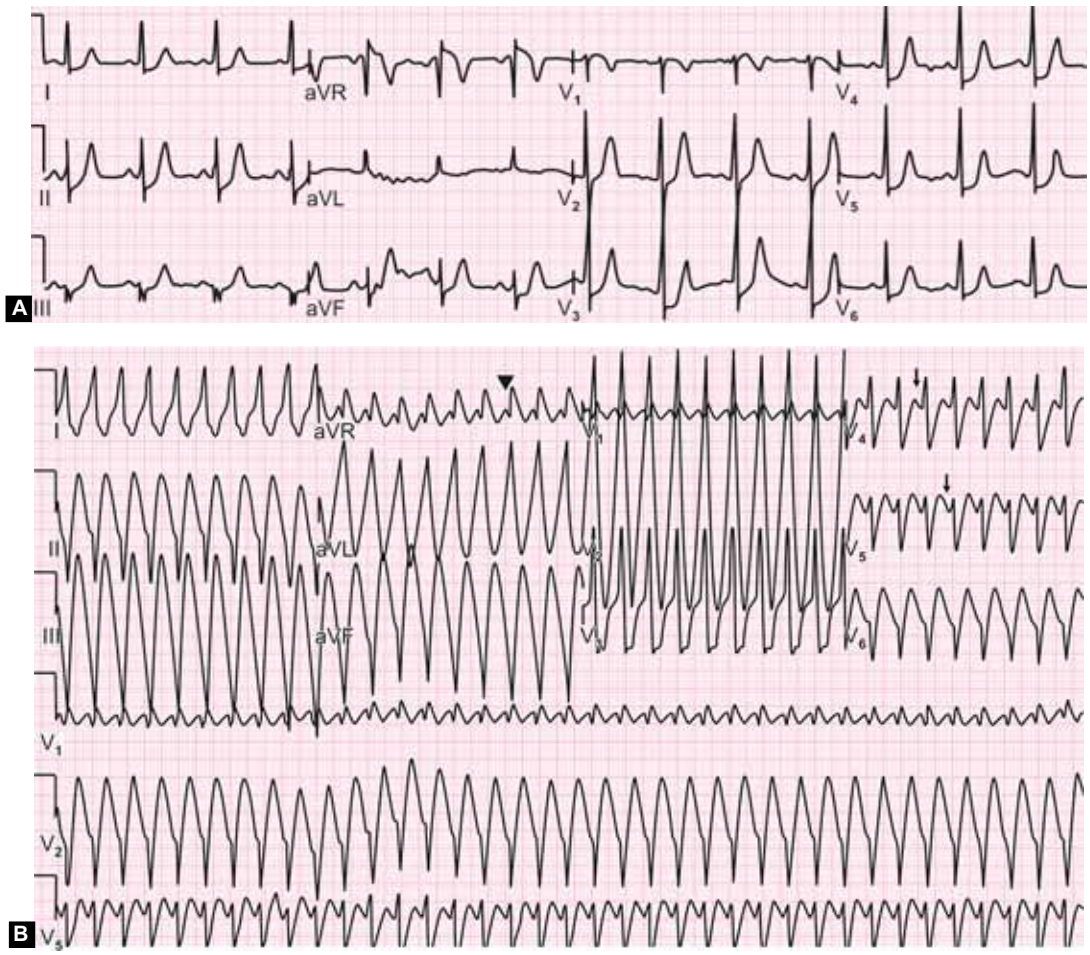


Figure 2.113: Antidromic circus movements. Note wide QRS complex tachycardia, short PR interval (arrowhead V_1 rhythm strip). QRS wider in the initial phase and narrower at terminal phase (arrows V_4, V_5) gives clue to delta wave



Figures 2.114A and B: Antidromic re-entry tachycardia ART. Since it mimics VT a previous ECG (A) is helpful to identify baseline rhythm. Note (B) stable axis, initial wide and narrow terminal QRS (arrow V_4, V_5) is due to delta wave. Also note shorter PR interval than RP (arrowhead aVR)

Lown-Ganong-Levine (LGL) Pre-excitation

Here the pre-excitation (or accessory) tract (James Fiber) connect atrium to bundle of His, therefore, PR is shortened but delta wave is not present. The reason is that the pre-excitation impulse joins high enough before the division of bundle branch.

Diagnostic Points (Figs 2.115 and 2.116)

- PR interval shortens
- No delta wave

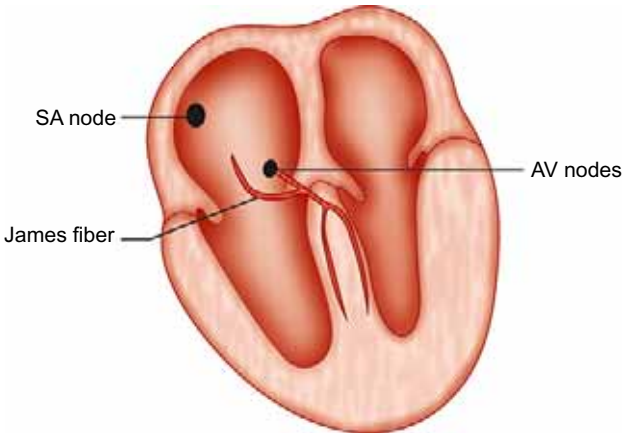


Figure 2.115A: Lown-Ganong-Levine syndrome (LGL) is a syndrome of pre-excitation of the ventricles due to an accessory pathway providing an abnormal electrical communication from the atria to the ventricles. See Jamed fiber, by pass tract which connect atrium to bundle of His

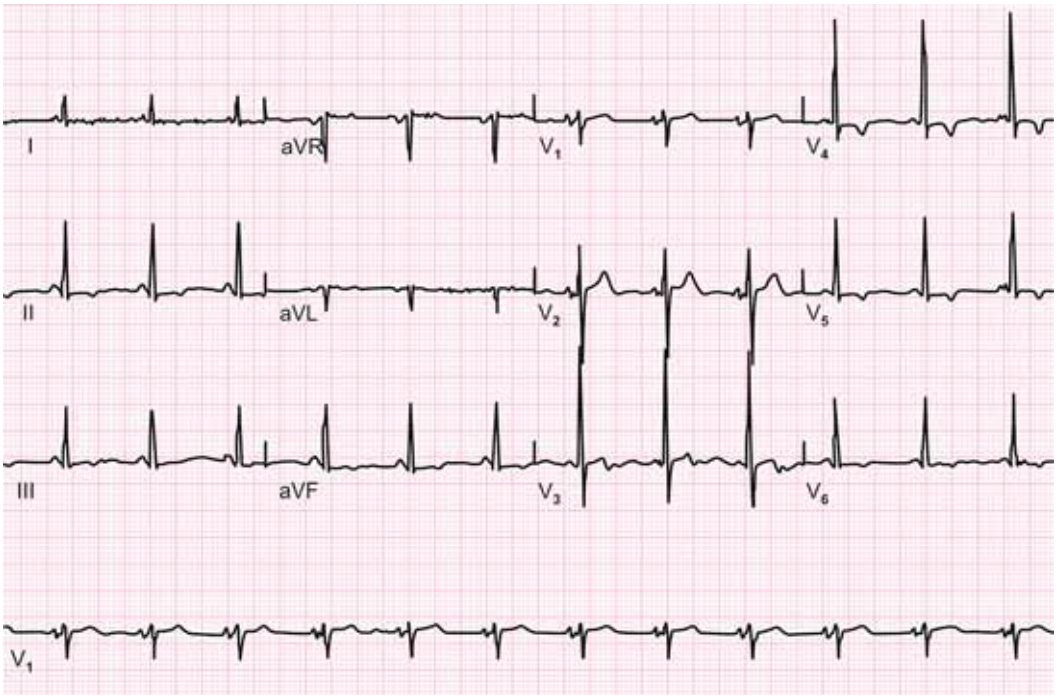


Figure 2.115B: Lown-Ganong-Levine syndrome. Note short PR interval (100 ms) and absence of delta wave

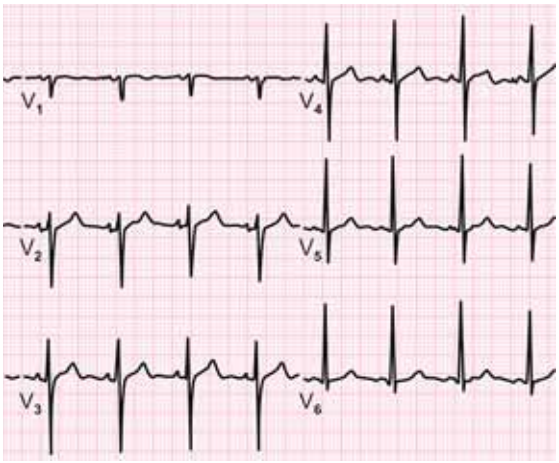


Figure 2.116: Lown-Ganong-Levine syndrome. Note short PR interval (100 ms) and absence of delta wave

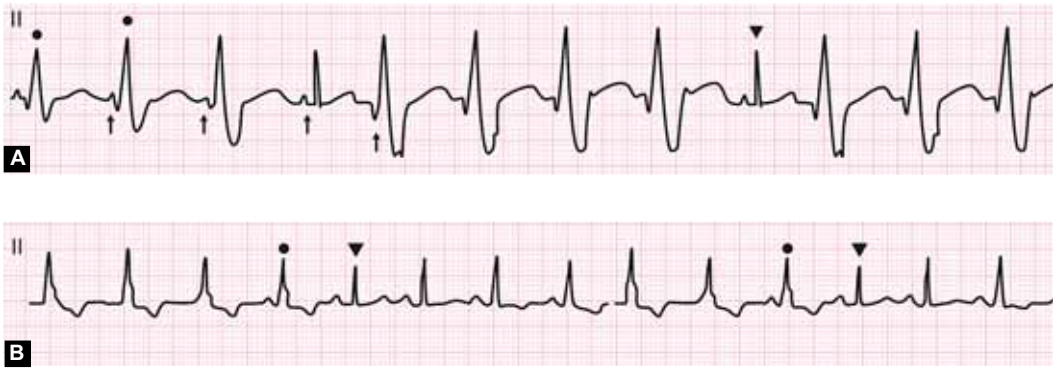
Accelerated Idioventricular Rhythm (AIVR)

Recall that usually the focus with inherently higher rate of discharge suppresses the focus with slower rate. The AIVD occurs when discharging rate of normally suppressed focus increases to above that of higher order foci. The ventricular escape rhythm which is usually 30–40 bpm is increased to 100–120 bpm and now called accelerated idioventricular rhythm (AIVR). The reason is increased automaticity of cardiac muscle that is why this condition is commonly seen in successful reperfusion after acute MI. Therefore, signs of MI are not uncommon in the ECG pattern of AIVR. The situation resembles ventricular tachycardia (VT) but this can be identified by the following features:

Diagnostic Points (Figs 2.117 to 2.119)

- Wide, bizarre QRS complex.
- The most important feature to distinguish from VT is that here the rate usually less than 100 to 120.

- The dominant pacemaker switches back and forth between the 2 competing pacemaker sites (i.e. normal or sinus and idioventricular), which leads to fusion beats (produced by simultaneous discharge from two foci and they fused to produce the hybrid deformed wide QRS complex) at the onset and termination of the arrhythmia. Because of the slow rate sometimes sinus impulse succeeds to produce QRS alone called capture beats.
- AV dissociation (no relation between atrial and ventricle).



Figures 2.117A and B: Different rhythm strips showing AIVR. Note AV dissociation (arrow) capture beats (arrowhead) and fusion beats (circle). Also note wide QRS complex

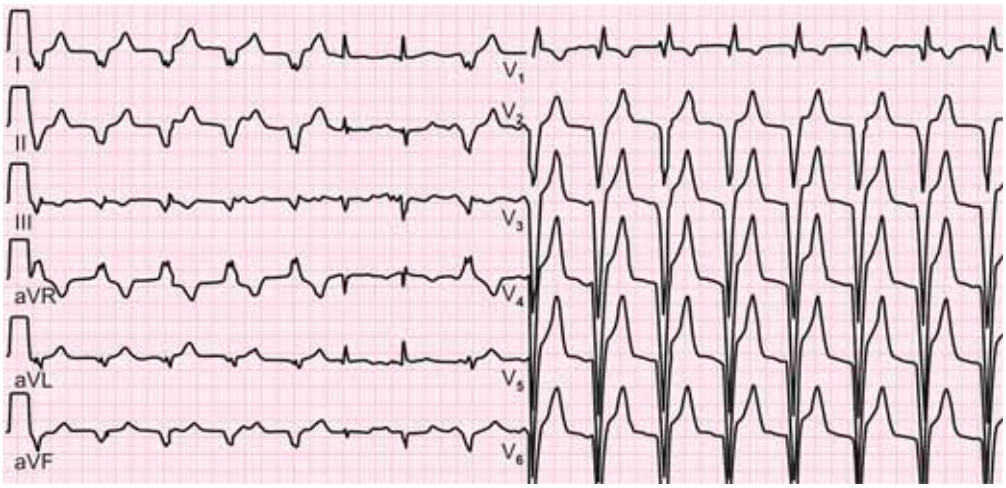


Figure 2.118: Accelerated idioventricular rhythm (AIVR) in a patient with AMI. There is reperfusion after the patient was treated with primary PCI. The first 5 beats and last 9 beats are AIVR. In between two narrow beats are seen of which second beat is probably a normal sinus beat .Note obvious difference between sinus beats and beats from ectopic idioventricular foci

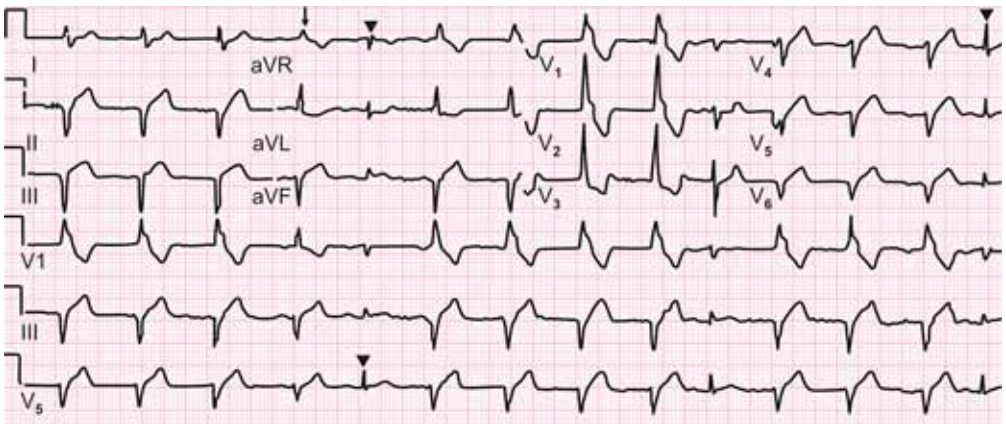


Figure 2.119: Accelerated idioventricular rhythm. Note many diagnostic findings such as ventricular rate around 80 bpm, wide QRS complexes, presence of fusion beats (arrow) and capture beats (arrowhead). Since this type of rhythm is frequently seen following reperfusion after acute myocardial infarction ST elevation also seen frequently (see lead II and aVF)

Ventricular Tachycardia (VT)

All wide QRS tachycardia should be regarded as ventricular tachycardia until proven otherwise. Reason may be due to increased automaticity of a single point in either right or left ventricle or due to re-entry circuit within any point in ventricular conduction system (from bundle branch to Purkinje system). Ventricular tachycardia usually manifests as monomorphic VT and if it is due to abnormalities in the ventricular muscle repolarization it manifests as polymorphic VT.

Diagnostic Points (Table 2.5 and Figs 2.120 to 2.125)

- Wide QRS: Strongest clue as a rule, the wider the QRS, the stronger the clue towards VT, especially QRS > than 0.16 sec, almost certain.
- Rhythm:
  - Rate is usually between 120 to 300 bpm and is regular.
  - Irregular rate usually are due to atrial fibrillation or aberrant conduction.

  See Table 2.5 for difference between ventricular tachycardia and aberrant conduction.

Table 2.5: Diagnostic points of ventricular tachycardia

• Wide QRS • Regular rhythm • AV dissociation: – Scattered P waves – Capture beats – Fusion beats • Extreme axis deviation • Concordance positive/negative • QS or rS in lead V_6 • Monophasic R with notch/slur in down-stroke of R • RS interval from beginning of R to nadir of S > 0.1 sex in any precordial lead • Notching near nadir of a wide S in V_1

- Atrioventricular (AV) dissociation: That is, no relationship between atrial and ventricular activity. This can be identified from following features:
  - Independent P waves (dissociated P waves) marching through the tachycardia.
  - Capture beats: If P waves successfully pass through AV-His-Purkinje system and produce QRS, which is earlier than the expected and is narrow.

  - Fusion beats: Likewise capture beats impulse from AV node passes through AV-His-Purkinje system but fuses with the impulse produced in ventricles. Hence the resultant QRS is the hybrid of sinus beat and ectopic focus beat. Fusion beat is differentiated from sinus beat by a short PR interval. All these are very strong clue which can confirm the diagnosis of VT, but their absences don't exclude the diagnosis.
- Extreme Axis Deviation:
  Axis from +150 to –90 (Nomann's land) suggest VT.
- Concordance: All or majority of QRS complexes may be positive (indicate origin from posterior ventricular wall) or may be negative (indicate origin from anterior ventricular wall).
- Clues from QRS morphology:
  - Monophasic R wave with a notch or slur or the down stroke of R wave suggests VT (90% of cases)
  - monophasic R wave with a notch or slur on the up stroke of R wave (50% possibility)
  - QS or rS (net negative QRS) in V_6.
  - Notching near the Nadir of S wave in V_1 when V_1 consists of a wide S.
  - RS interval > 0.1 sec, i.e. from R to the nadir (deepest part) of S wave in any precordial lead.
  - An rSR' or rsR' pattern in lead V_1 > 90 percent chance of aberrancy (RBBB) or if QRS in V_1 is negative with rapid down stroke of S wave with or without of a proceeding thin r wave. It also suggests (LBBB aberrancy) (Table 2.5).

How to Differentiate from Aberrancy?

We have learnt that if APC falls in a critical time where the ventricles have partly recovered from the previous depolarization, it can generate a new depolarization (aberrant conduction) whose shape is wide (often rSR' or rsR'). A run of such aberrantly conducted beats may result in a wide QRS complex tachycardia which is SVT in fact. How to differentiate this type of SVT from VT? The answer is simple, absence of above features and presence of three components rsR', rSR' pattern is a fair clue towards aberrancy or follow this questionnaire suggested by Brugada et al.

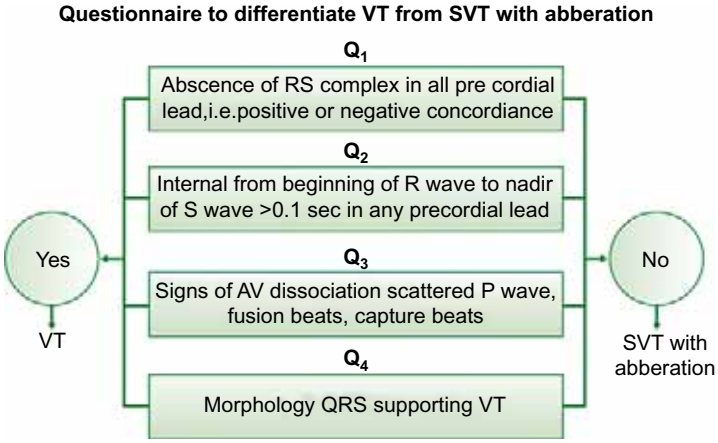


Figure 2.120: Sequences in form of a questionnaire to differentiate SVT with aberration with VT

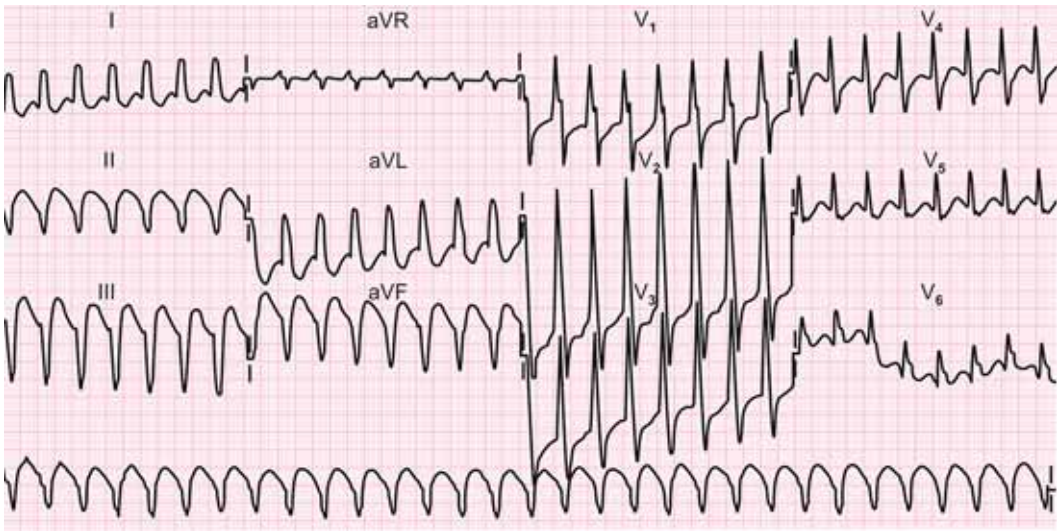


Figure 2.121: VT. Note presence of many diagnostic points described in the text including wide complex QRS, monophasic QRS with a notch or slur on the down stroke of R wave (V_1 and V_6), concordance and left axis deviation

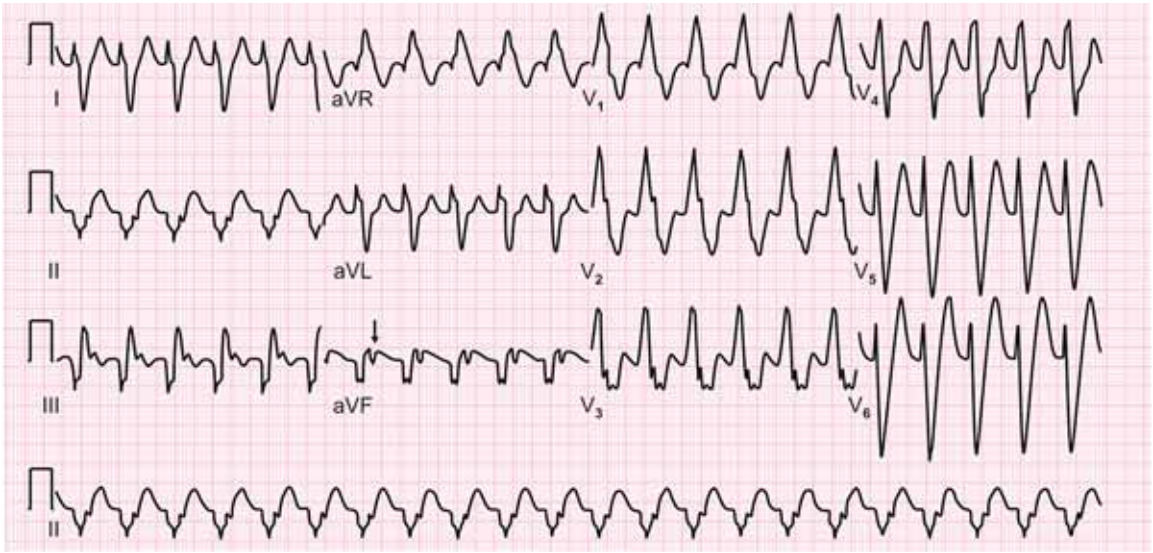
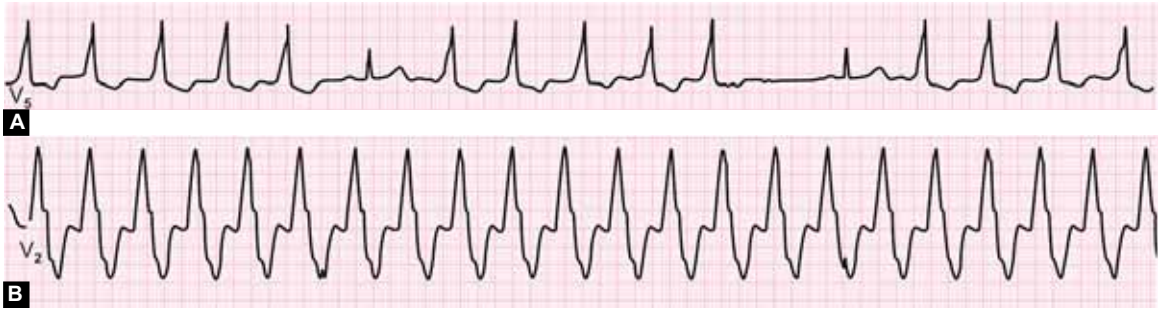


Figure 2.122: Ventricular tachycardia with many diagnostic features. Note regular rhythm, long QRS duration (160 ms or more), extreme axis deviation, RBBB pattern with decreasing R wave amplitude and growing S wave predominance from V_1 to V_6, QRS with a notch or slur on the down stroke of R wave (V_2 and V_3), biphasic (rather than triphasic; cf. SVT with aberrancy). Note that interval from the beginning of R wave to the nadir of S wave is greater than 100 ms in precordial leads. Also note in lead aVF, QRS seems to be exceptionally wide. In fact, there is retrograde P wave (arrow) thus making QRS duration of 160 ms



Figures 2.123A and B: ECG showing VT with LBBB pattern (A), with RBBB pattern (B). Generally tachycardia originating from left ventricle produces RBBB pattern and tachycardia originates from right ventricle (and septum also) produces LBBB pattern

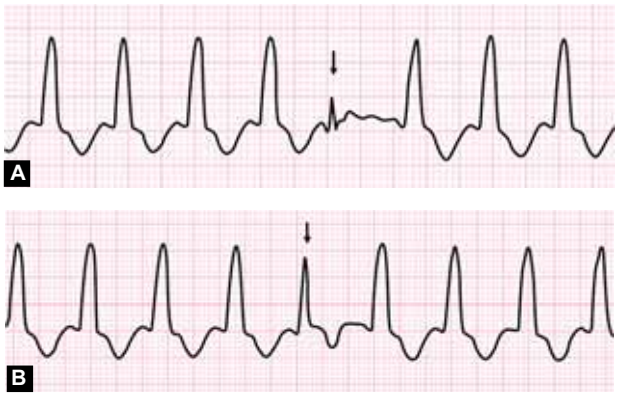


Figure 2.124A and B: Example (arrows) of capture beat (A) and fusion beat (B). Capture beats arise when the P wave activates the entire ventricle before the VT cycle, resulting in a narrow premature beat. A fusion beat is a QRS complex that has two sources of activation; from a P wave that has activated a part of the ventricle over the recovered AV node and from the next VT complex, resulting in a complex of intermediate width

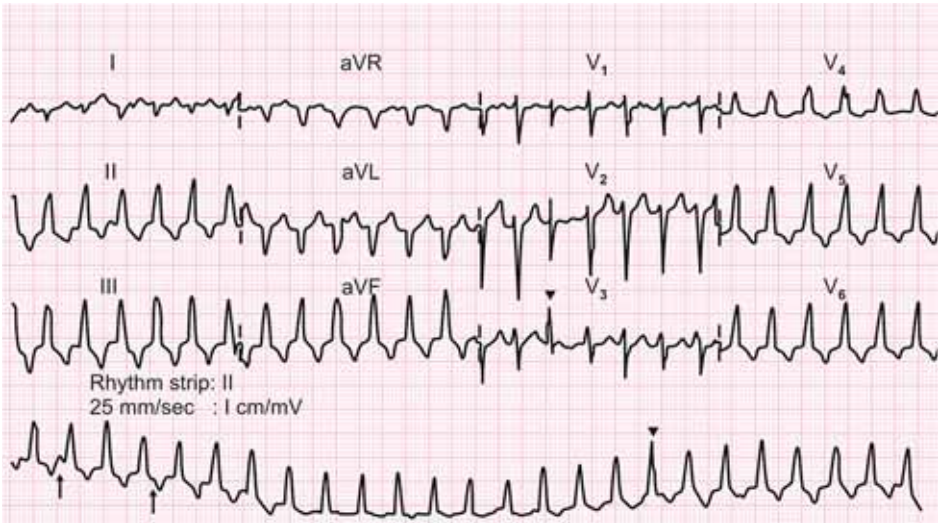


Figure 2.125: VT showing broad complex regular tachycardia, AV dissociation (note independently conducting P waves) (arrows); capture beats (arrowhead V_3) and fusion beat (arrowhead rhythm strip)

Torsade de Pointes

"Torsade de points" (twisting of points) is a polymorphic ventricular tachycardia. Mostly long QT interval results in (torsade de points. It may be brief episode or lead to ventricular fibrillation and may be fatal. Causes of long QT are mostly acquired drug (class I antiarrhythmic: Quinidine and procainamide, etc. tricyclic antidepressant, phenothiazine) or electrolyte imbalance (hypomagnesemia, hypokalemia, hypocalcemia) or rarely congenital long QR syndrome (Romano-Ward-Syndrome) or if congenital long QT associated with congenital deafness called Jervell and Lange-Nielsen syndrome.

Diagnostic Points (Figs 2.126 and 2.127)

- QRS complexes change rapidly and progressively from upright (positive) to isoelectric to downward (negative) and back to upright (Shifting electric axis).
- Rates vary from 200–300 bpm or may be higher but usually not sustained. But if sustained can change into ventricular fibrillation (VT).
- Sinus rhythm prior to onset demonstrates prolonged QR interval.

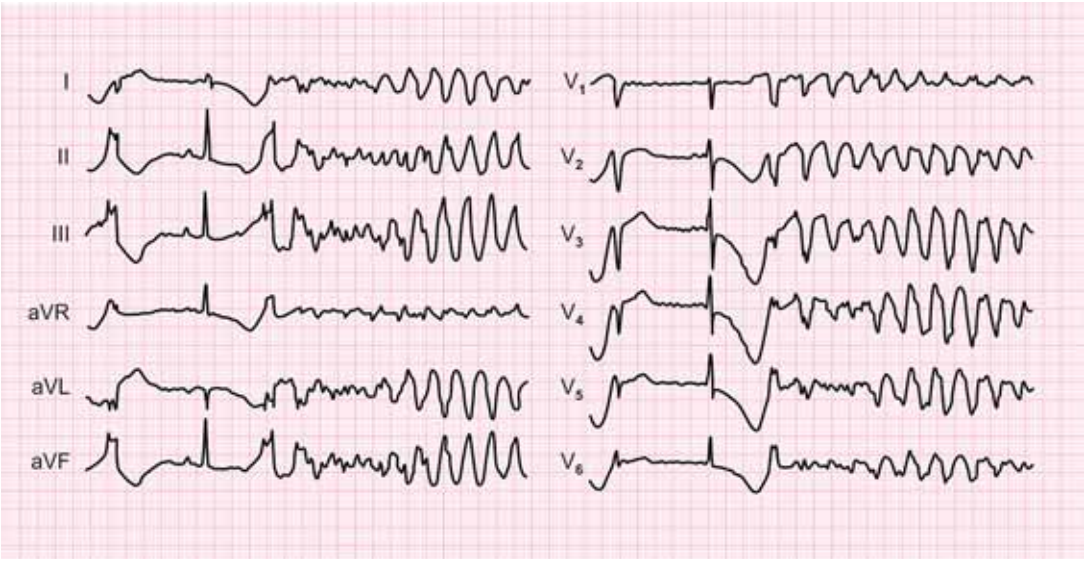


Figure 2.126: Classic example of Torsade de pointes (a polymorphic VT). Note before initiation of Torsade classic features of sinus bradycardia, prolongation of T wave and QT interval and bizarre T wave morphology

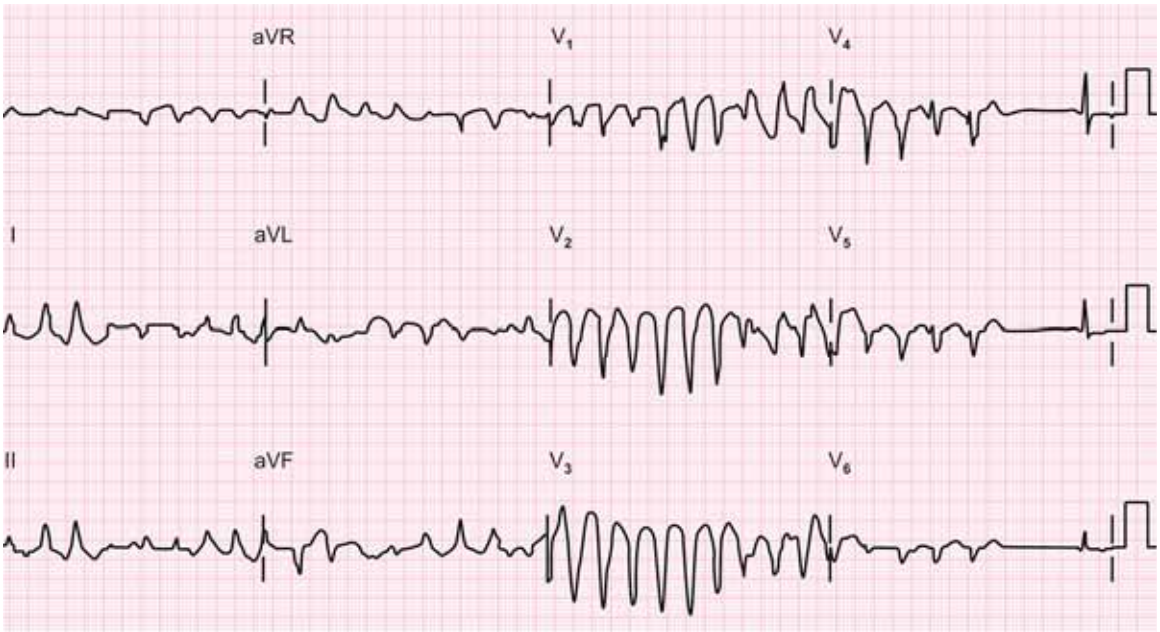


Figure 2.127: This ECG is of a patient with long QT. *Note:* Polymorphic ventricular tachycardia (PVT). Note also fast rate (200–300 bpm) and varying QRS morphology (amplitude and contour) and shifting electrical axis. These presentations constitute Torsade de pointes

Ventricular Fibrillation (VF) (Fig. 2.128)

The condition starts when multiple re-entrant foci in ventricle result is an inefficient contracting heart which in fact just quivering with no pumping activity at all.

Diagnostic Points

- Large or small fine or coarse disorganized set of deflections with not identifiable QRS complexes.
- Rapid rate may be >350 /minute.
- Of course, no P wave.

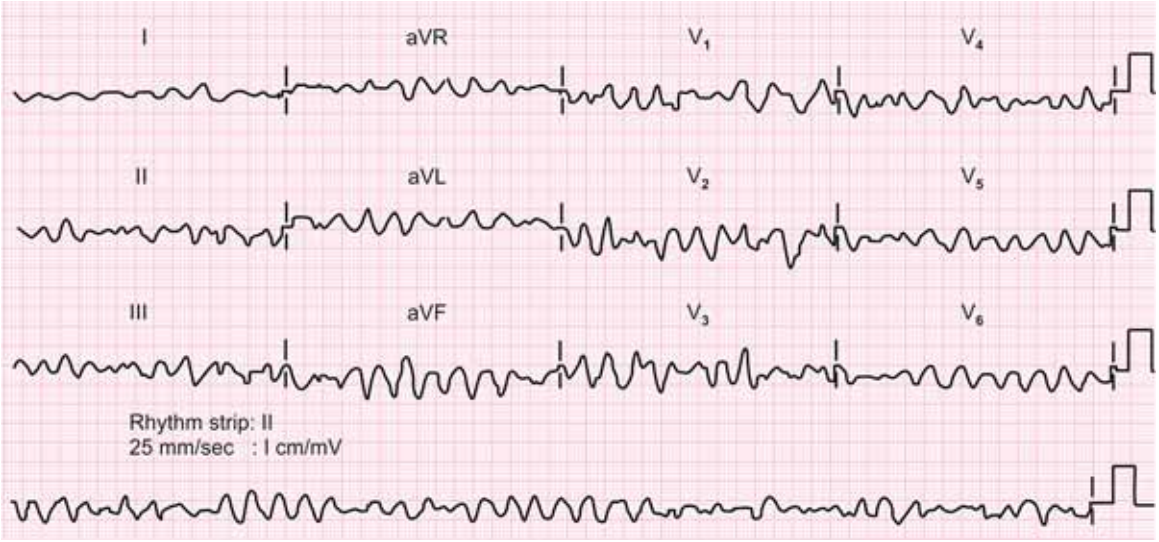


Figure 2.128: Ventricular fibrillation VF. Note rapid disorganized deflection and no identifiable QRS or P waves

MISCELLANEOUS CONDITIONS

Different isolated pathologic conditions can be identified from ECG tracing by their specific presentation. These conditions can be grouped as follows:

Cardiac Pathologies

- Atrial septal defect
- Acute pericarditis
- Pulmonary embolism
- Electrical alternans
- Dextrocardia

Drugs Electrolytes

- Digitalis
- Type I antiarrhythmic
- Hypokalemia

- Hyperkalemia
- Hypo or hypercalcemia

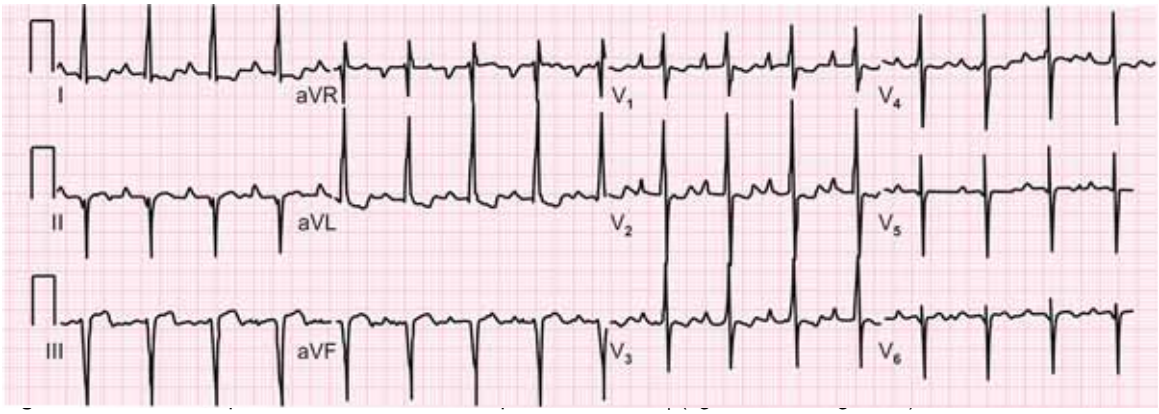
Physical

- Hypothermia
- Electric pacing
- Artifacts

Atrial Septal Defects (ASD) (Fig. 2.129)

Although no tight diagnostic criterion but following are strong diagnostic clues.

- Incomplete RBBB
- A characteristic "Crochetage" on R wave of lead II, III, and aVF. ST and /or T wave changes in right precordial leads showing strain (as in LVH).



ratio in lead V_1 > 1 while in lead V_6 < 1which shows RVH. Again recall presence of LAD in the presence of RVH is important clue and indicator atrial septal defect (ASD) as usually RAD is present with RVH. Note Crochetage sign is absent here

Pericarditis (Figs 2.130 to 2.133)

"Pericarditis" (inflammation of pericardium) can be identified by following diagnostic points:

Early Stage

- Diffuse ST segment elevation with concavity upward in most leads except in aVR and V_1, where there is reciprocal depression generally. Note this ST elevation can be differentiated from ST elevation seen MI by upward concavity (as described in text earlier) and the pattern of ST elevation in leads. For example, in acute pericarditis the ST elevation in lead II > in lead III (in inferior MI ST elevation in lead III>II) while in precordial leads are in the order of $V_6>V_5>V_4>V_3>V_2$ (which is reverse in anterior MI).
- PR segment depression in most leads except in aVR (occasionally in lead V_1 also) where PR segment is elevated.
- T wave remains upward during acute stage.

Later Stage

- ST and PR segment return to normal (isoelectric position)
- T wave becomes flattened or inverted.
- If pericardial effusion has resulted due to inflammation there is also:
  a. Low voltage QRS
  b. Electrical alternans (in case of large effusion), see later.

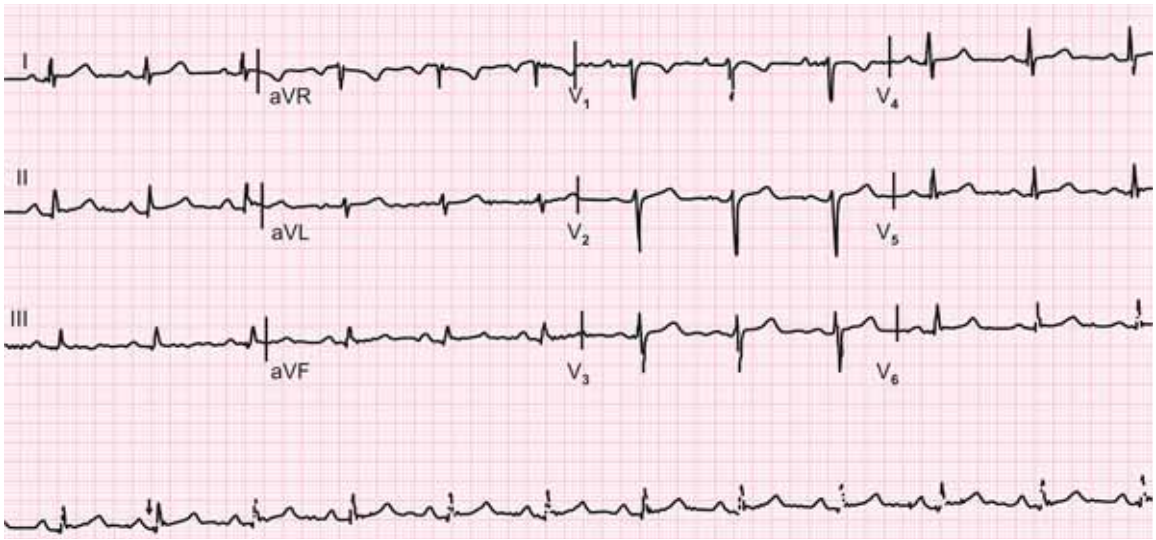


Figure 2.130: Acute pericarditis. Note ST elevation with upward concavity in leads I, II, aVF and V_2–V_6. Also note PR depression in leads II and aVF (more obvious in rhythm strip, arrow)

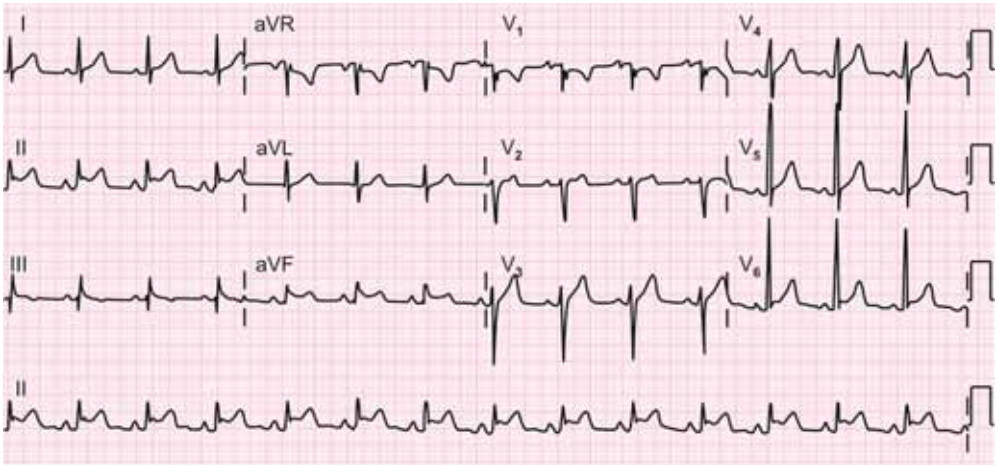


Figure 2.131: Acute pericarditis. This is classic diffuse pericarditis, with diffuse ST elevation in limb leads I, II, III and aVF and in precordial leadsV_2–V_6. Note that ST elevation (STE) in lead II >lead III (differentiate it from inferior MI where STE lead III> lead II) and ST elevation in leads V_6 and V_5 > V_4>V_3>V_2 (in anterolateral MI sequence in reverse usually). There is significant PR depression in leads II and leads V_3–V_6. The STE is upwardly concave, which also supports the diagnose pericarditis

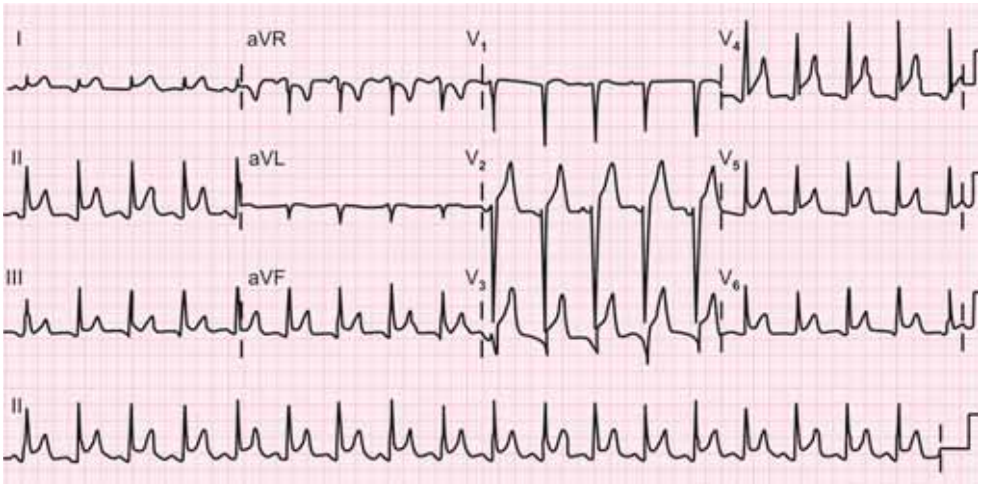


Figure 2.132: Note the widespread ST segment elevation in the anterior, inferior and lateral leads. Also note the PR segment depression in the inferior leads and, importantly, the “reciprocal” PR segment elevation in lead aVR. This ECG demonstrates findings suggestive of myopericarditis

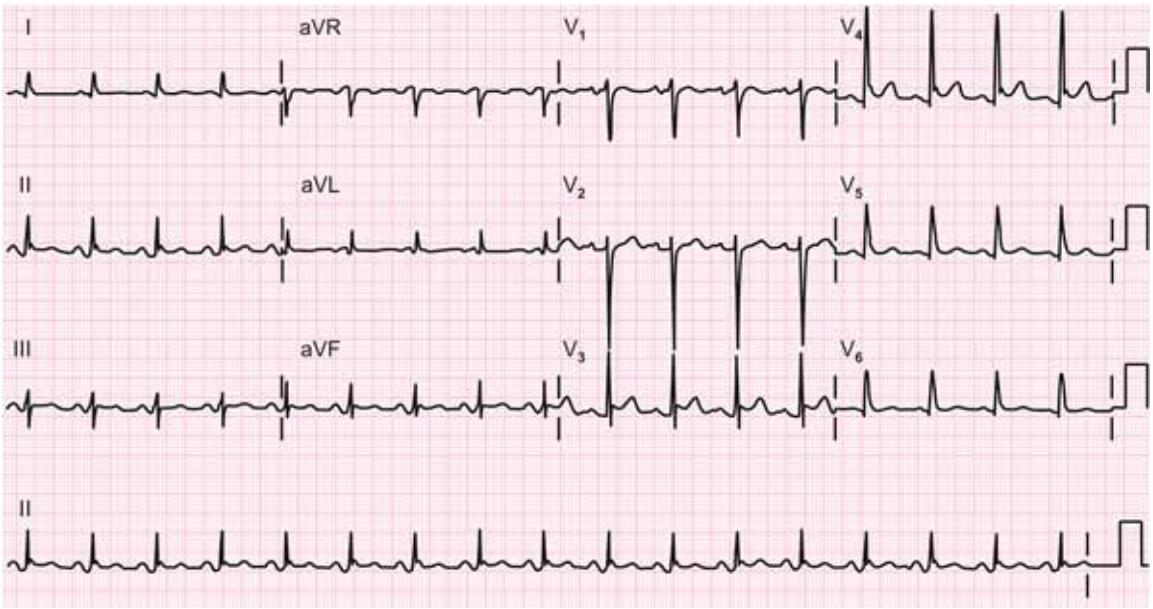


Figure 2.133: Acute pericarditis. PR depression in lead II, III, aVF and also V_3, V_4, PR elevation in aVR, slight ST elevation in II, III, aVF V_3, V_4, note ST elevation may be slightly elevated in many leads and difficult to identified but PR depression when present in limb leads along with precordial leads points to pericarditis. Note J point in lead V_6 is almost at the level of T wave

Pulmonary Embolism (PE)

Since the pulmonary embolism, the clot lodged in the pulmonary circulation resulting in Right atrium and right ventricular strain.

Diagnostic Points (Figs 2.134 and 2.135)

- Fast cardiac rhythm which may be sinus tachycardia or transient supraventricular arrhythmias like atrial fibrillation, atrial flutter or PAT.
- Symmetrical T wave inversion, strain pattern in V_1–V_3 (right ventricle strain). There may be appearance of ST-T changes (especially in right precordial leads) and Q waves mimicking infarction in inferior leads/anterior leads. This needs to be differentiated it is better to obtain serial ECG tracing and look for other changes of PE in ECG (e.g. there may be q waves in leads V1, III, aVF but not in II so can be differentiated from inferior wall MI).

- Incomplete or complete RBBB.
- May be classic PE presentation (not always present) $S_1Q_3T_3$ or $S_1Q_3T_3$, S wave in lead I, Q wave in lead III and inverted T wave in lead III.
- Chronic RV strain may cause right atrial enlargement (see right atrial abnormality)
- RAD.

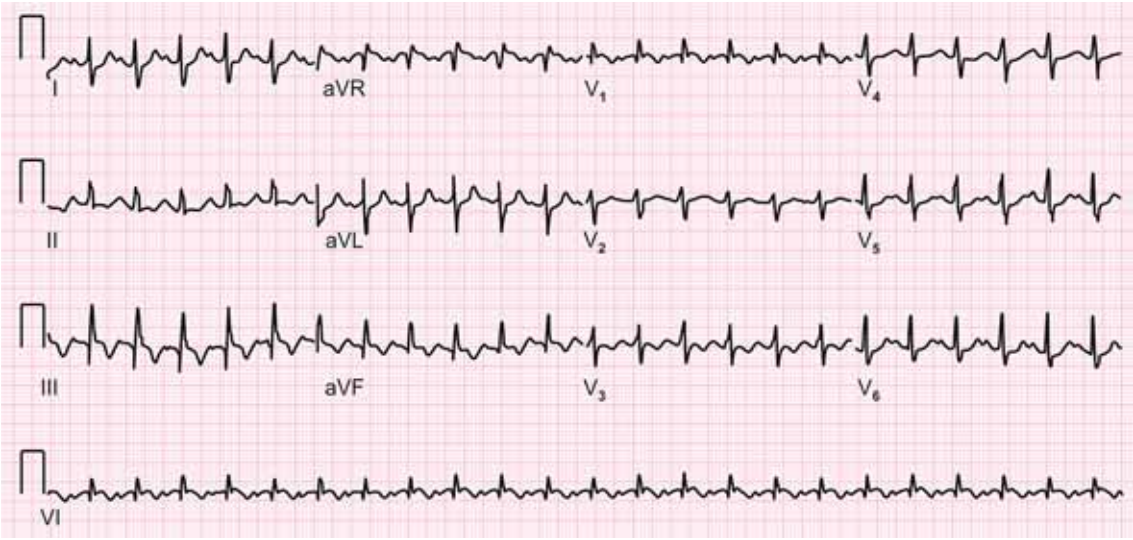


Figure 2.134: ECG showing classical features of pulmonary embolism (PE). Note sinus tachycardia, incomplete RBBB, S wave in lead I and Q wave in lead III, T wave in version in lead III ($S_1Q_3T_3$), slight ST elevation in lead V_1. T wave is also inverted in lead aVF and V_3

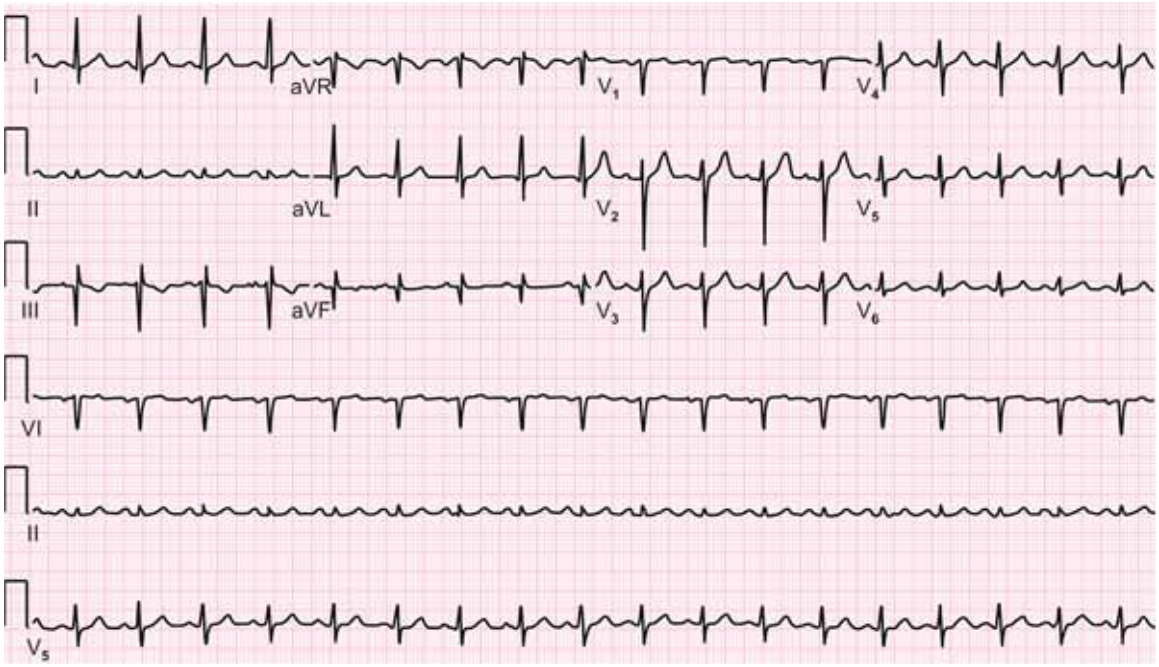


Figure 2.135: Another ECG showing features of PE. Presence of sinus tachycardia is the most constant feature. S wave in lead I, Q wave in lead III and inverted of flat T wave in lead III ($S_1Q_3T_3$) although not always present; can be seen here. There is also slight ST elevation in V_1 indicating right ventricular strain

Electrical Alternans (Figs 2.136 to 2.138)

This condition refers to alternate beat to beat variation in the amplitude, direction (polarity), duration (intervals) and morphology of QRS complex. Most often the cause is a large pericardial effusion when floating heart continuously changes its position in relation with ECG leads (swing back and forth like a pendulum). This type is called as motion alternans. That is why electrical alternans is diagnostic of cardiac temponade.

On the other hand presence of electrical alternans may also indicate presence of a bypass tract or myocardial ischemia. In this case the alternation is in the form of conduction which may also result in alternative variation in PR ,QRS or RR intervals or amplitude and is called as conduction alternans. Presence of conduction alternans helps to differentiate sinus tachycardia from pre-excitation SVT which uses bypass tracts.

Identification Points

- Cyclic alteration in the amplitude and/or polarity of R wave from beat to beat.
- May involve P or T wave or QRS complex all three (total electrical alternans).
- If conduction alternans alternating variation may be seen in PR, QRS and RR intervals.

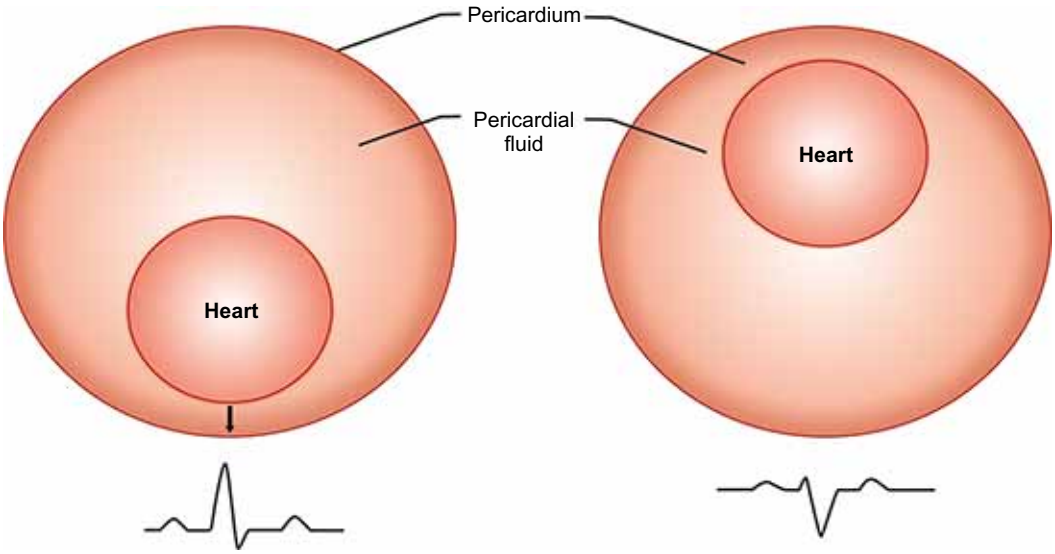


Figure 2.136: Pericardial fluid and floating heart. Note heart direction toward or away the leads change polarity and amplitude of resulting QRS

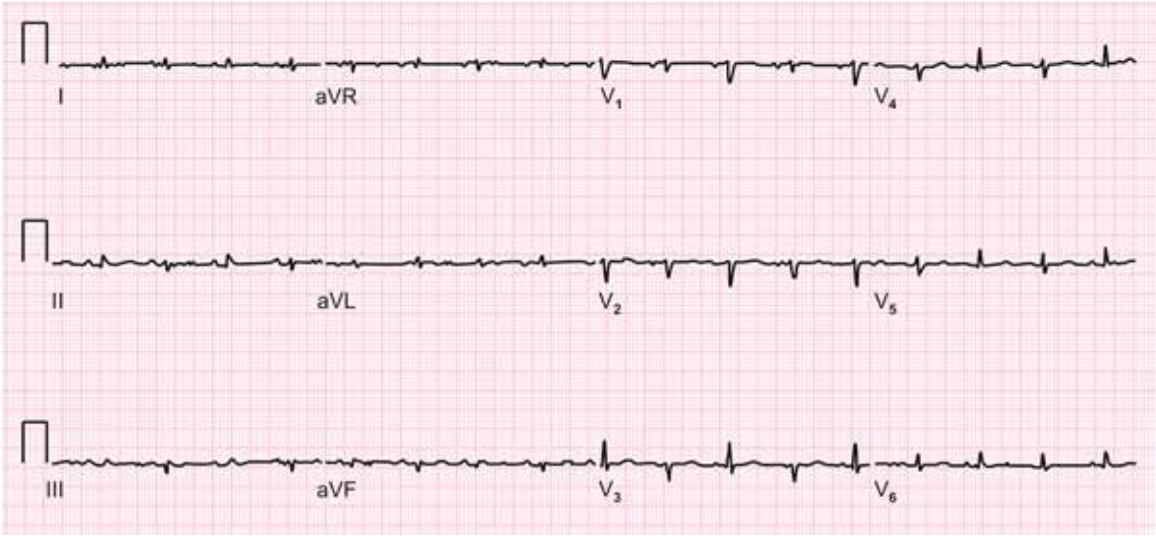


Figure 2.137: Low voltage beat-to-beat variation of the amplitude and direction of QRS which is characteristic of electrical alternans

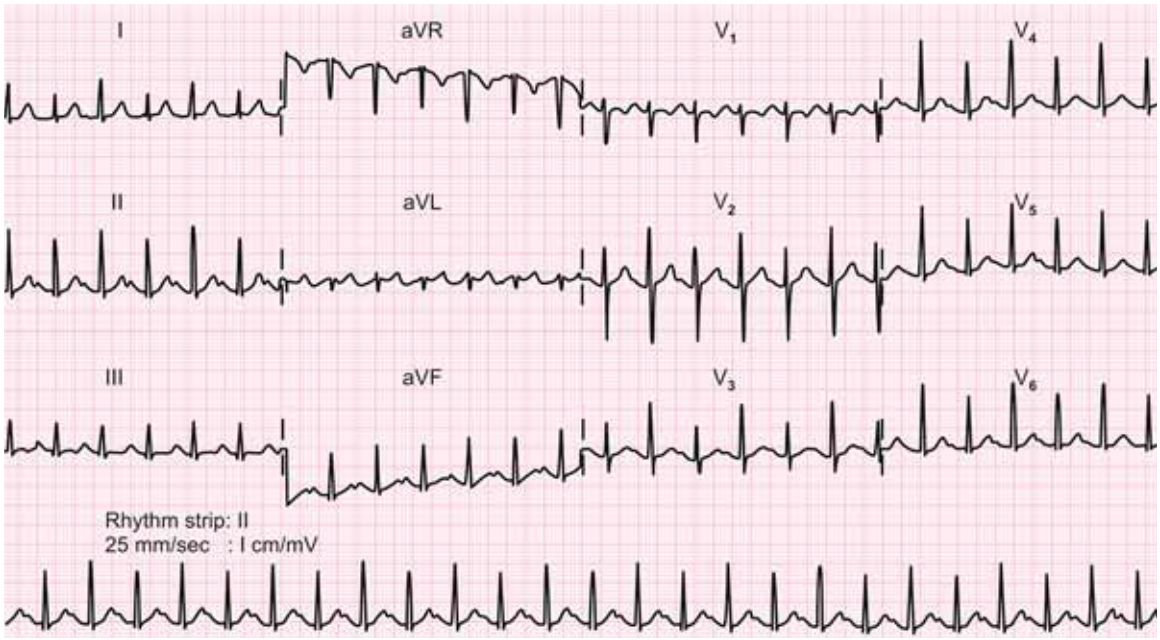


Figure 2.138: Electrical alternans. Note alternating heights of QRS complexes which indicate that current also utilizes some other pathway to reach from atrium to ventricle as in this case of atrial tachycardia. Hence the presence of electrical alternans here excludes sinus tachycardia. This type of electrical alternans sometimes called conduction alternans. Compare with other ECG showing electrical alternans due to pericardial effusion which is due to cardiac floatation hence sometimes called as motion alternans

Digitalis and ECG

Digitalis inhibits sodium potassium transport mechanism resulting increased intracellular Na^+ and net loss of K^+ from the cell. That is why digitalis use in normal dose limits is mainly seen in ST segment and T wave (repolarization). This is called as digi-effect (i.e. ECG changes in the absence of digitalis toxicity).

Identification Points of Digieffect (Figs 2.139 and 2.140)

- Decreased amplitude/flattening/inversion/biphasic T wave.
- ST segment depression with upward concavity shape may be identified as sagging/scooping/reversed check mark.
- Modest P-R interval prolongation; First degree AV block.
- Shortening QT interval.
- Prominent U waves.

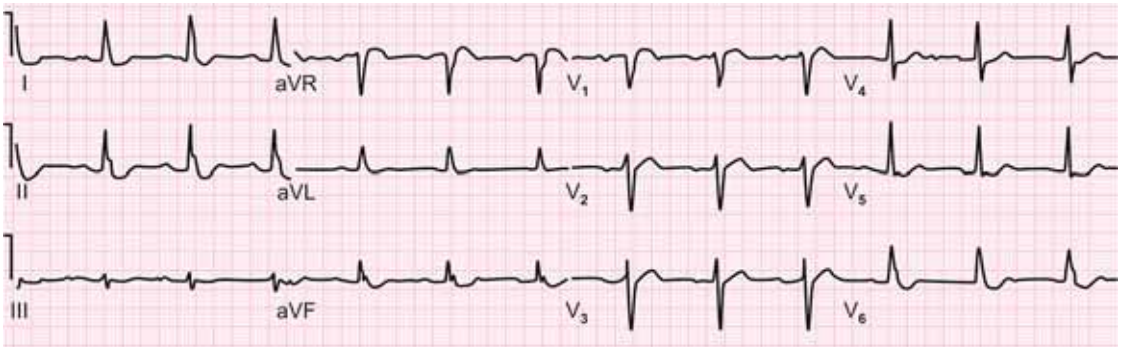


Figure 2.139: Digitalis effect, i.e. pharmacological effect rather than toxic effect of digitalis. Note typical concave scooped ST segment depression most prominent in leads with large R wave, e.g. inferiorly leads II and aVF (lead III is typically spared) and laterally (lead I, aVL, V_5 and V_6). Also note diffuse flattening of T wave

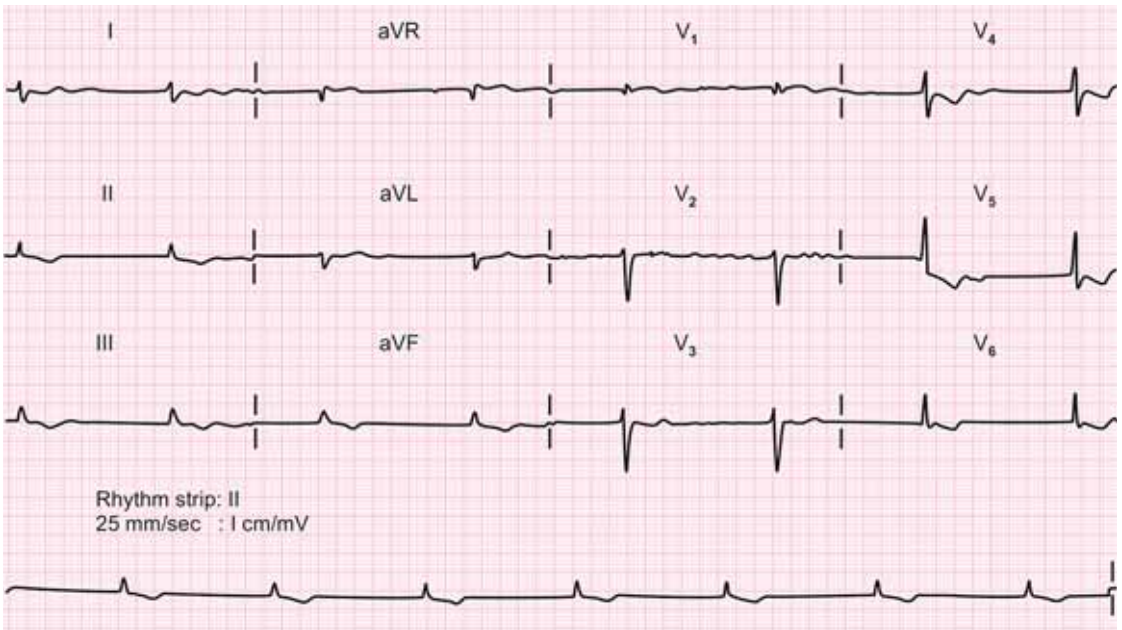


Figure 2.140: Digitalis effect. Here the ST segment depression is down sloping or "reversed check mark" like shape (lead II, III, aVF, V_4–V_6)

Digitalis Toxicity (Figs 2.141 to 2.143)

Digitalis toxicity is dangerous and results in almost any type of arrhythmia. Can be classified as:

1. Atrial Arrhythmia: Sinus Bradycardia, APCs, Atrial tachycardia with AV block (a well-known digitalis toxicity arrhythmia and of diagnostic value as seen in figure 2.141).
2. AV junctional arrhythmia: 1st degree AV block, 2nd degree AV block usually Mobitz type I, 3rd degree complete AV block, JPCs
3. Ventricular arrhythmias: VPC, Bigeminy, VT (e.g. bidirectional tachycardia) and Ventricular fibrillation.

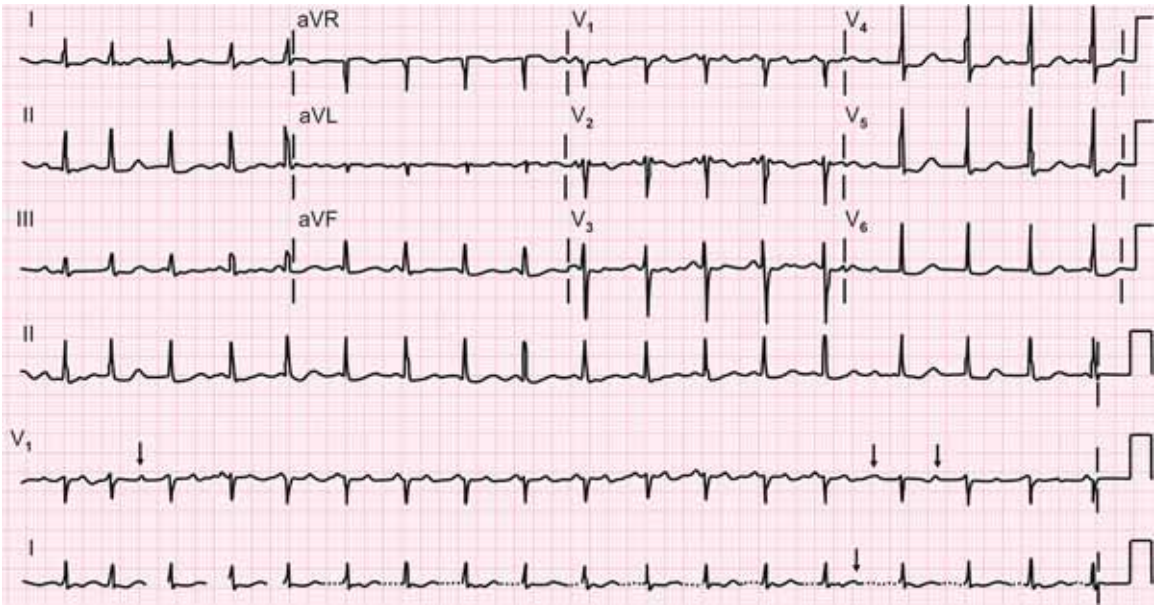
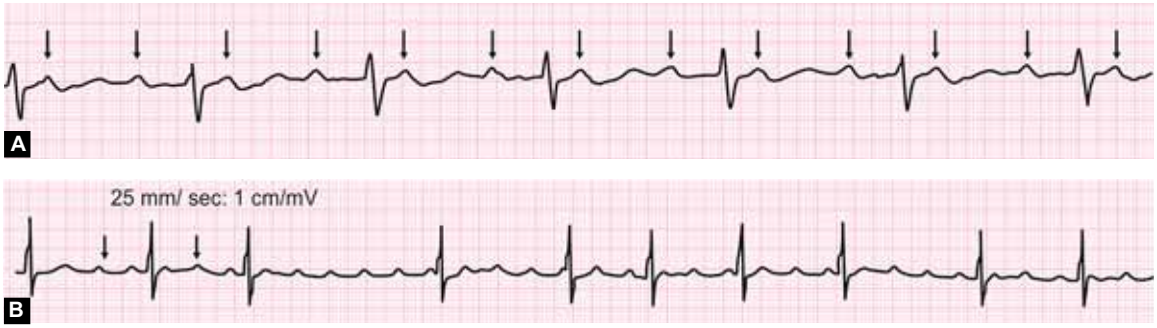


Figure 2.141: This ECG demonstrates paroxysmal atrial tachycardia (PAT) with AV block. The hallmark of digitalis toxicity is conduction block at AV node and increased automaticity of atrial, junction or ventricular tissue. Note blocked P waves (arrow)



Figures 2.142A and B: Paroxysmal atrial tachycardia (PAT) with 2:1 block (A) and with variable AV conduction (B). This is characteristic of digitalis toxicity. This can be differentiated from atrial flutter. In atrial flutter, the atrial rate is greater than 250 bpm and characteristic flutter waves that are inverted in lead II, III, aVF while in atrial tachycardia there is isoelectric baseline between the P waves (arrows)

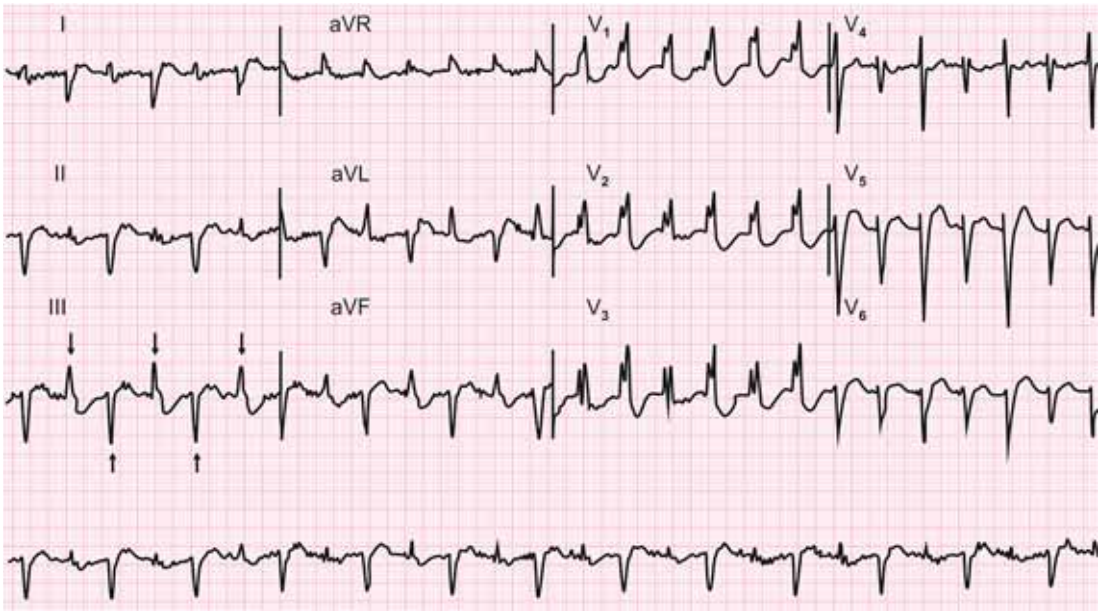


Figure 2.143: Bidirectional tachycardia is an unusual tachyarrhythmia characterized by beat-to-beat alternation of the morphology and the axis of the QRS complexes as seen in this ECG (arrows). The usual ventricular rate in bidirectional tachycardia ranges from 140 to 180 bpm and can be regular or irregular. Bidirectional tachycardia is usually associated with digitalis toxicity

Type I Antiarrhythmic Drugs, Quinidine, etc.

These drugs directly affect ion transport across sarcolemmal membrane resulting in an increase in the duration and decrease in the amplitude and rate of rise of the action potential (Phase 0) (also vagolytic effects that are why ECG shows tachycardia). Quinidine is the prototype (also procainamide and disopyramide).

Diagnostic Points; Therapeutic Effects (Fig. 2.144)

- Sinus tachycardia
- Prolongation of QT interval. Class I antiarrhythmic drugs are notorious for causing prolonged QT.
- Widening of P wave and QRS complex
- Prolonged T wave duration with flattening or inversion
- Repolarization changes ST segment depression
- Prominent U wave.

Diagnostic Points; Toxic Effects

- QT prolongation
- Markedly increase QRS duration
- 1º, 2º, 3º (degree) AV block
- VPC
- VT
- Torsade de pointes
- VF

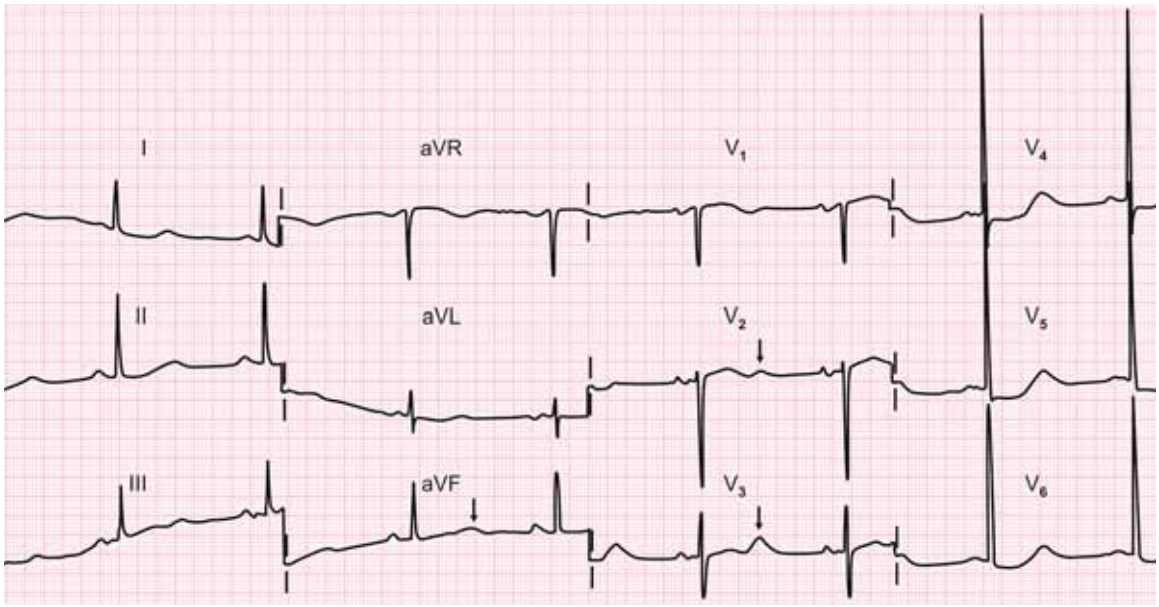
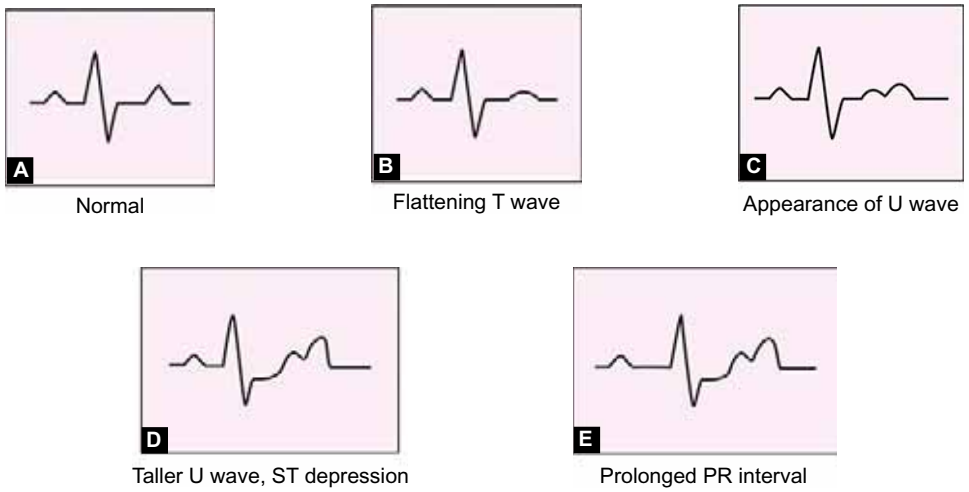


Figure 2.144: Classic features of quinidine excess. Note there is ST depression in leads I, II, aVL, aVF and V_4–V_6. U waves can be seen (arrow) merged with T wave. QT or QU interval is markedly prolonged. T waves show flattening. Other findings are LVH and left atrial abnormality

Hypokalemia (Figs 2.145 to 2.147)

Hypokalemia has characteristic ECG finding and roughly correlate with the levels of serum potassium. For learning ease following sequence can be used:

- Normal QRS
- Gradual flattening/inversion of T wave (<3.5 mEq/L)
- Prominent U waves (< 3.5 mEq/L) (U wave may become taller than T wave)
- T wave may become fused with U waves (<1.5 mEq/L); best seen in leads V_2–V_5. (*Note:* Presence of U waves may give a false impression of prolonged QT (QU) interval)
- ST segment depression.
- Slight prolongation of PR interval.



Figures 2.145A to E: Schematic sequence of ECG finding in hypokalemia

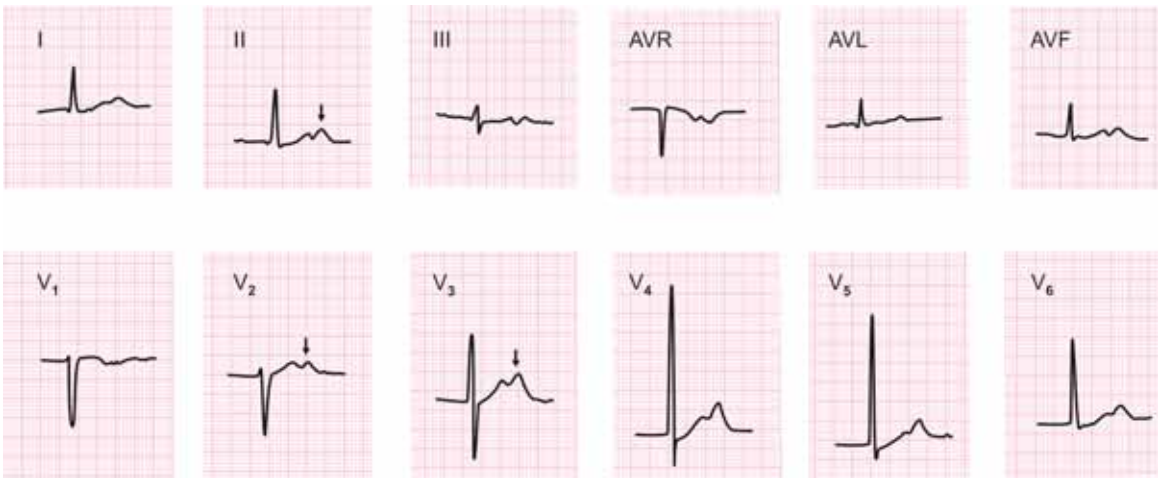


Figure 2.146: Obvious features of hypokalemia. Note flattening of T waves, prominent U waves (arrows). Also note ST depression (leads II, III, aVF and V_3–V_6)

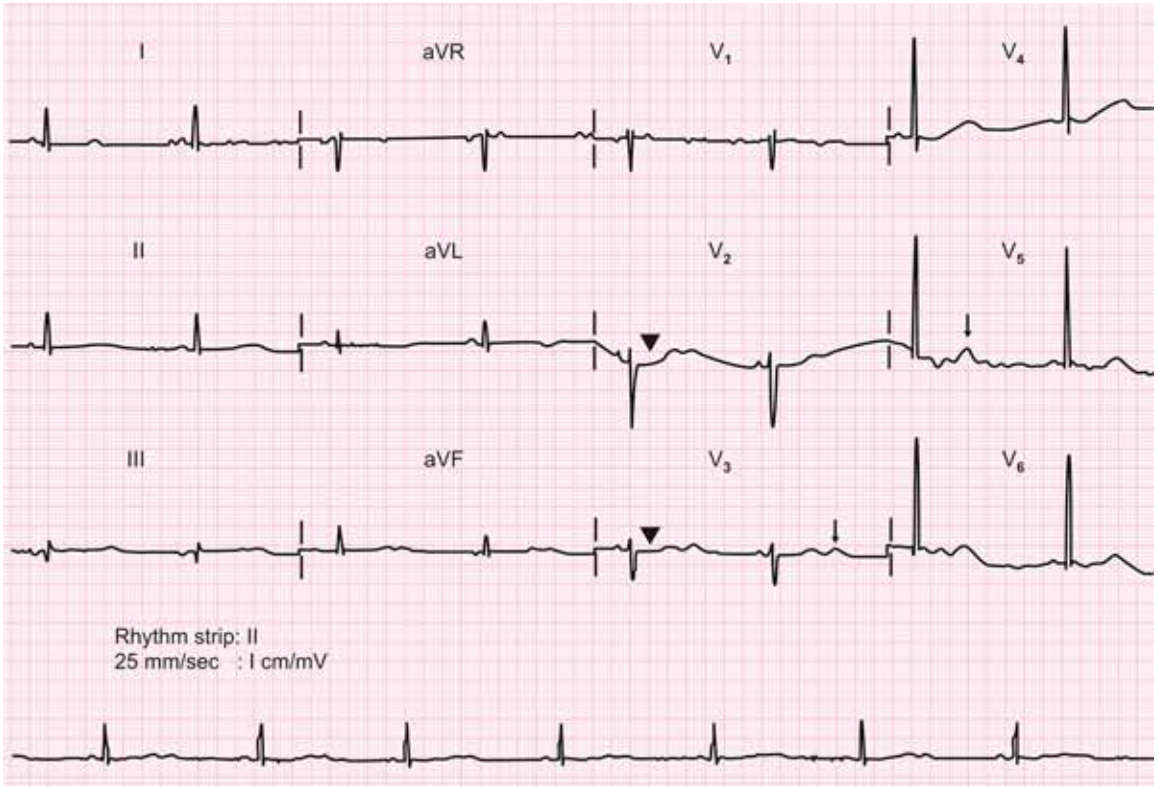


Figure 2.147: ECG showing features of hypokalemia. Note flattening of T waves (see rhythm strip), U waves (arrow) and slight ST depression (arrowhead)

Hyperkalemia (Figs 2.148 to 2.150)

- Increasing serum levels of K^+ produce different patterns of ECG.
- Mild to moderate hyperkalemia (>6.5 mEq/L)
  - Tall peaked narrow based (tented) T wave in most of the leads.
  - T wave becomes flattened and widened.
  - PR interval increased.
- Higher levels depress (7–8 mEq/L) SA node and AV conduction.

- Severe elevation (8–9 mEq/L) causes depression of intraventricular conduction (wide QRS) also and may completely suppress SA node (sinus arrest). So heart may actively be maintained by junctional/ventricular escape rhythm. Therefore:
  - Wide QRS, later portion of QRS may fuse with tall T wave.
  - Disappearance of P wave.
  - Junctional or ventricular escape rhythm.
- Lethal levels around 12 mEq/L is likely to produce:
- Ventricular fibrillation
- Heart block.

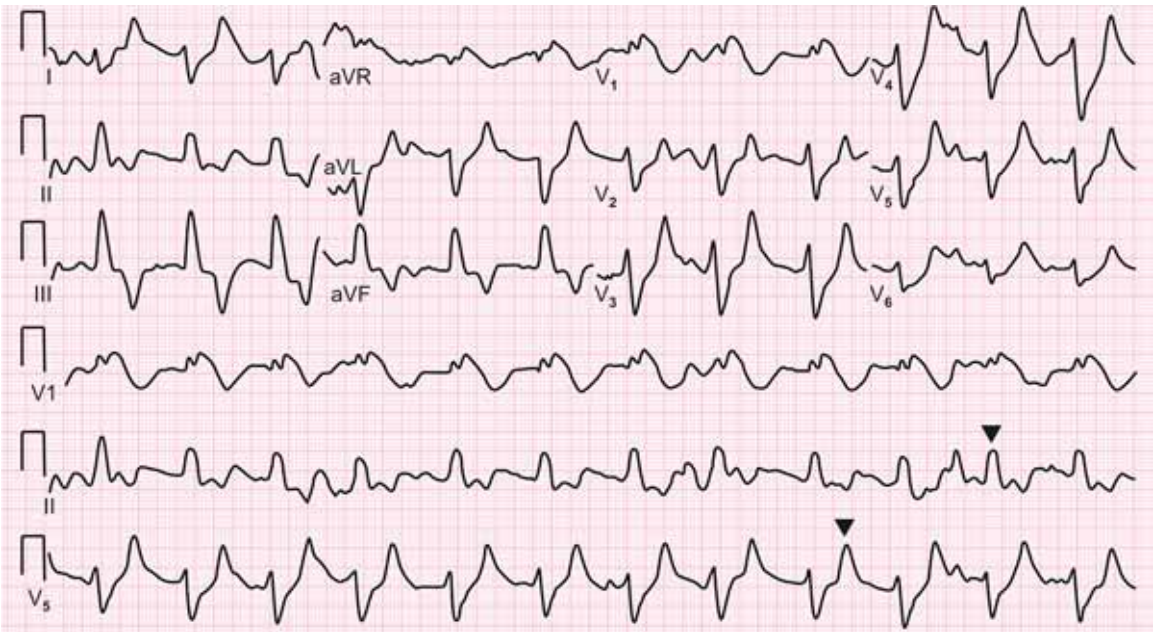


Figure 2.148: Note in this broad QRS complex with RBBB morphology and a rhythm of 75 bpm tall T waves are due to hyperkalemia (arrowhead)

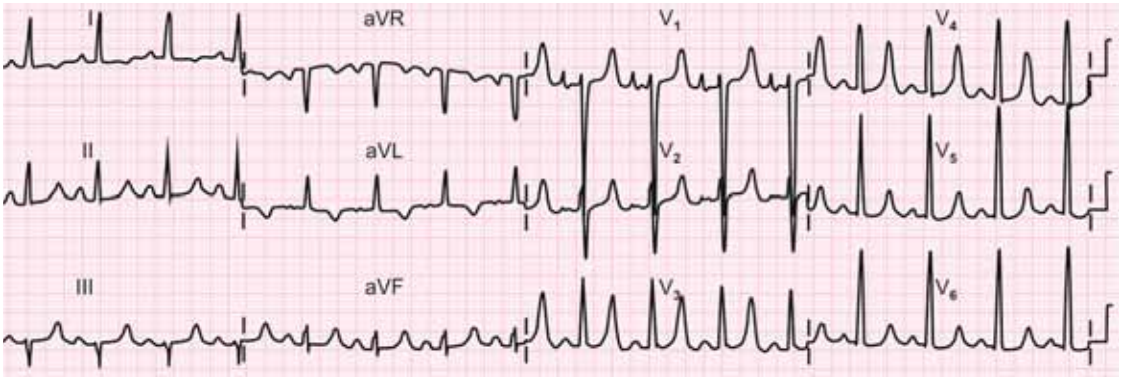


Figure 2.149: This ECG shows right atrial abnormality indicated by peaked P wave in leads II and V_1. Also note LVH (S wave in lead V_1+R in lead V_5 greater than 60 mm) with ST depression in V_4–V_6. Tall tented T waves in many leads especially precordial leads are due to hyperkalemia

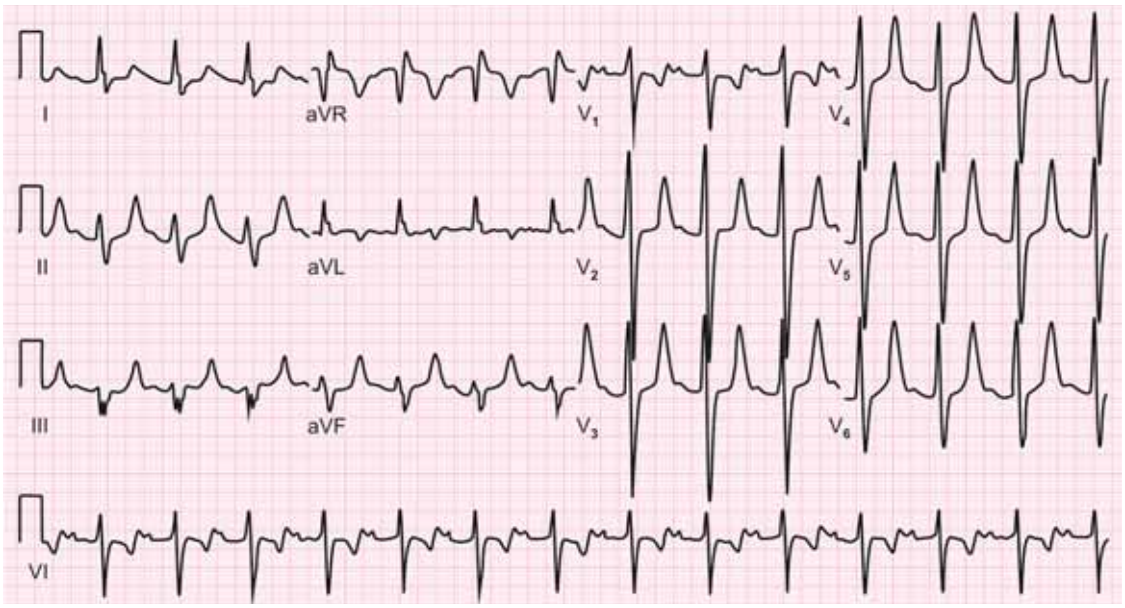


Figure 2.150: A case of chronic renal disease. Severe hyperkalemia (8.2 mEq/L), note prolonged QRS interval, peaked T wave, QTc is prolonged (almost one second). This patient also has hypocalcemia (6.8 mg/dL). Note that long QT interval is mainly due to prolonged ST segment (compare QT prolongation in quinidine, etc.) which is specific to hypocalcemia

Hypo and Hypercalcemia

Since calcium has direct favorable effect on cardiac contraction in hypercalcemia the heart contraction is faster and ECG manifestation is shortened QT interval.

Reverse happens in hypocalcemia and QT interval is prolonged but this QT prolongation is mainly because the prolongation of ST segment and not by T wave (differentiate from Quinidine) (*See* Fig. 2.150).

Physical Changes

Hypothermia (Fig. 2.151)

- Sinus bradycardia
- Prolongation of all intervals (PR, QRS, ST, QT).
- Osborn wave (also called O wave or J wave). This is an elevated hitched-up wave at the end of QRS; best seen in leads V_3–V_4 and represent abnormal repolarization.
- Below 32°C or rapid re-warming often results in atrial fibrillation.

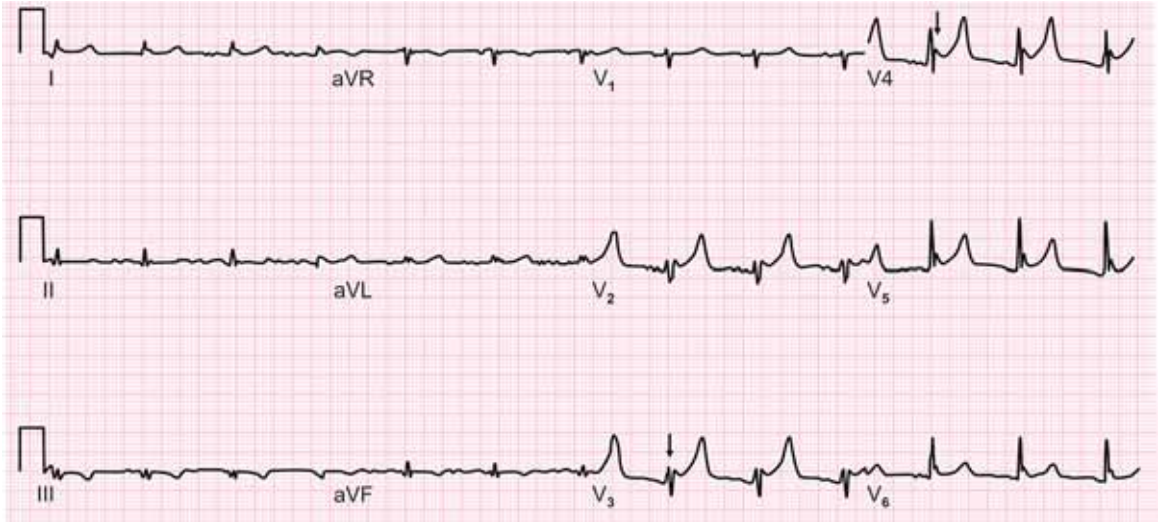


Figure 2.151: Hypothermia. Osborn or J wave best seen in precordial leads (arrows). Also note prolongation of all intervals including QT interval, PR interval QRS interval

Electric Pacing

- Pacemaker impulse results in ECG patterns which are taken into account for its well functioning or malfunctioning. Some basic information is necessary to indentify pacemaker rhythm. For proper understanding of ECG pattern produced by pacemaker, it is good to have some understanding of pacemakers. Usually pacemaker's nomenclature (which can contain up to five letters or positions like DDI, DDD, and VDD, etc.) tells important features of pacemaker function. Currently NGB code is being used (Table 2.6).

There are four types of pacemakers: asynchronous, single-chamber synchronous, double-chamber AV sequential, and programmable.

- Asynchronous or fixed-rate (AOO, VOO, DOO)—discharge at a preset rate that is independent of the inherent heart rate.
- Single-chamber synchronous or demand (AAI, VVI)—discharge at a preset rate only when the spontaneous heart rate drops below the preset rate.
- Dual-chamber AV sequential pacing (VDD, DVI, DDD)—usually uses two electrodes, one in the atrial appendage and one in the right ventricular apex. The atrium is stimulated to contract first, then after an adjustable PR interval, the ventricle is stimulated.
- Programmable pacemakers—pacing rate, pulse duration, voltage output, and R-wave sensitivity are the most common programmable functions.

  The most frequently used are the DDD (dual chamber pacing and sensing, both triggered and inhibited mode), VVI (for single chamber, ventricular pacing in the inhibited mode), VDD (ventricular pacing with atrial tracking), and DDI (dual chamber pacing and sensing, but inhibited mode only). The first two positions of this code (Chamber Paced and Sensed) are relatively straightforward. The third position is described as follows:

  D - (dual): In DDD pacemakers, atrial pacing is in the inhibited mode (the pacing device will emit an atrial pulse if the atrium does not contract).

  In DDD and VDD pacemakers, once an atrial event has occurred (whether paced or native) the device will ensure that an atrial event follows.

  I - (inhibited): The device will pulse to the appropriate chamber unless it detects intrinsic electrical activity. In the DDI program, AV synchrony is provided only when the atrial chamber is paced. If on the other hand if intrinsic atrial activity is present, then no AV synchrony is provided by the pacemaker.

  T - (triggered): Triggered mode is only used when the device is being tested. The pacing device will emit a pulse only in response to a sensed event.

  The VDD pacemaker is used for AV nodal dysfunction but intact and appropriate sinus node behavior. DDI is rarely used as the primary mode of pacing. The DDI pacer is used for a patient with a dual-chamber pacemaker that has episodes of paroxysmal atrial fibrillation. DDI prevents high ventricular rates. Some DDD pacemakers are programmed to enter the DDI mode when high atrial rates occur.

  The fourth position, rate modulation, increases the patient's heart rate in response to "patient exercise". A

number of mechanisms (vibration, respiration, and pressure) are used to detect "patient exercise". As the exercise wanes, the sensor indicated rate returns to the programmed mode.

The fifth position describes multisite pacing functionality. Atrial multisite pacing is being investigated as way to prevent atrial fibrillation. Ventricular multisite pacing is a treatment for pacing a patient with dilated cardiomyopathy.

- Different types and their site produce different types of ECG pattern. The first letter denotes the chamber which is paced (i.e. stimulated). Whereas second letter denotes the chamber that is sensed. This third letter refers to the pacemakers response to what it sensed which may be I (inhibition) or T (trigger) of output circuit, D (dual function/dual chamber), O (no response to sensing-fixed rate). Fourth letter denoted programmable functions or rate of modulation. Fifth letter denotes specific antiarrhythmic functions (for details consult cardiology texts).
- Pacemaker impulse is identified by narrow straight vertical spike called pacemaker spike followed by P wave and narrow QRS (if pacemaker is placed in right atrium). If pacemaker is placed in right ventricle, pacemaker spike is followed directly by wide QRS (different from normal intrinsic QRS). Similar to LBBB (because right ventricle activated first).
- Dual chamber pacemaker activates atrium first and then ventricle, thus two spikes one before P and one before QRS.
- Atrial and ventricular pacemaker spikes may not be visible in every lead and may vary in height and limb leads and chest leads (*See* Figs 2.152 to 2.155).

Table 2.6: NBG code for pacemaker classification. (NBG; N = NASPE is the North American Society of Pacing and Electrophysiology; B = BPEG is the British Pacing and Electrophysiology; Group)

NBG code				
I: Chamber paced	II: Chamber sensed	III: Response to sensing	IV: Programmable function/rate modulation	V: Antitachycardiac functions
V: Ventricle	V: Ventricle	T: Triggered	P: Simple Programmable	P: Pace
A: Atrium	A: Atrium	I: Inhibited	C: Communicating	S: Shock
D: Dual (A + V)	D: Dual (A + V)	D: Dual (T + I)	R: Rate modulating	D: Dual (P + S)
O: None	O: None	O: None	O: None	O: None
S: Single (A or V)	S: Single (A or V)			

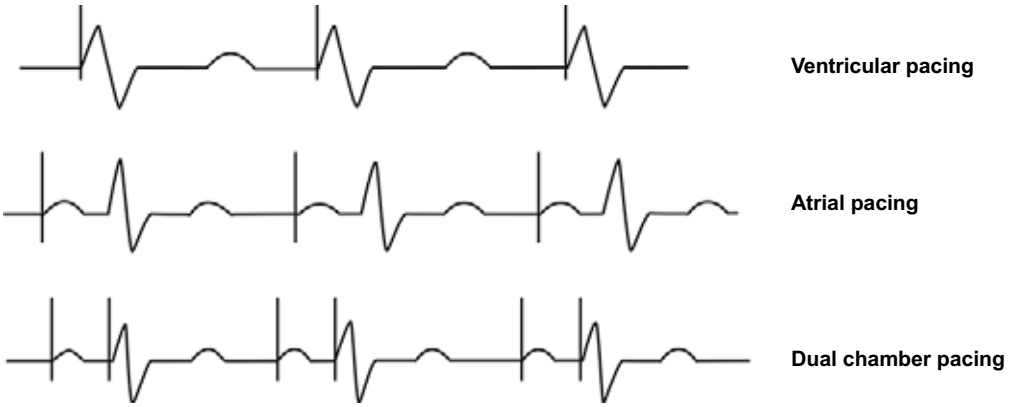


Figure 2.152: Note pacing location and their resultant ECG pattern. Note atrial and dual chamber pacing yield p wave after pacemaker spike

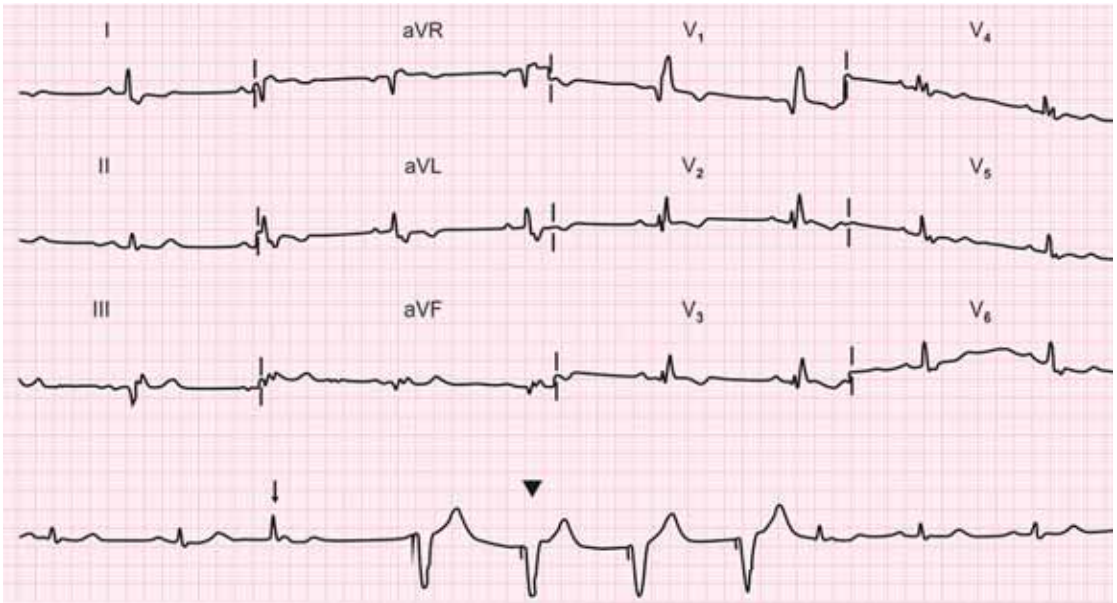


Figure 2.153: Ventricular pacemaker functioning on demand. Note (below; rhythm strip) a VPC (arrow) when interrupts the sinus rhythm and produces a pause (to nearly a rate of 50 bpm), the pacemaker starts firing (arrowhead) with a rate of 72 bpm until normal sinus rhythm again takes over

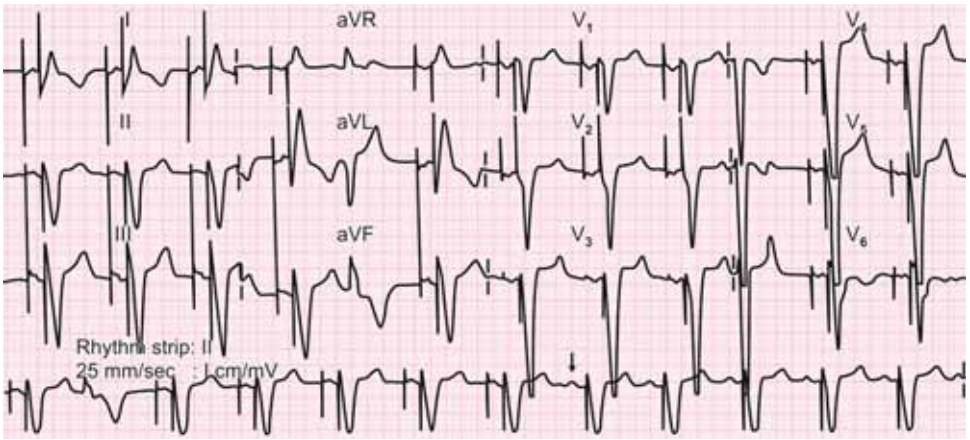


Figure 2.154: ECG showing a dual chamber pacemaker with appropriate atrial and ventricular sensing and capture. Note after a pacemaker spike, atrium is paced producing P wave then another pacemaker spike pacing ventricle and producing QRS complex showing ventricle is paced. Note after arrow sinus P wave starts originating (i.e. from sinus node) thus pacemaker stops pacing atrium (which shows pacemaker is working properly)

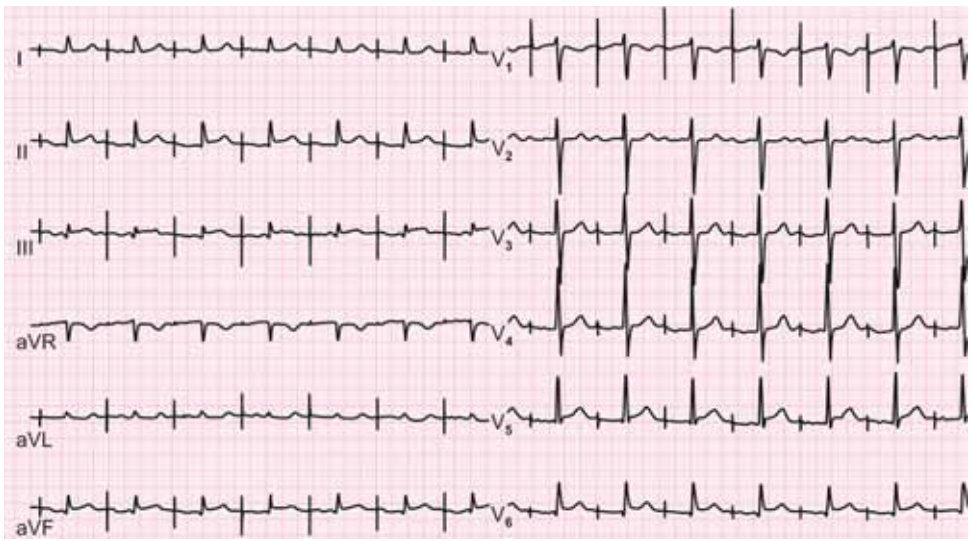


Figure 2.155: The ECG shows atrial pacemaker. Pacemaker spikes are seen before each QRS complex and initiates a tiny P wave. The PR interval is prolonged (around 250 ms)

Pacemaker Abnormality

Loss of Capture

Pacemaker senses and delivers the stimulus into the atrium and ventricle (depending on the type) but fails to produce P wave or Q wave (although stimulus apparently falls outside the refractory period). This can be identified by the pacemaker spike followed by no P wave or no QRS complex.

Loss of Sensing

Normally pacemaker senses the intrinsic atrial or ventricular activity and the responses (inhibits or triggers). Here pacemaker stimuli are delivered at the programmed rate without considering atrial or ventricular activity (thus works like fixed rates pacemaker). Note capture occurs normally whenever pacing stimuli occur outside the atrial/ventricular refractory period.

Pacemaker Failure

Pacemaker may stop firing (may be due to inadequate output from pulse generator, broken lead wire or electrode displacement). The result is failure of appropriate pacemaker output.

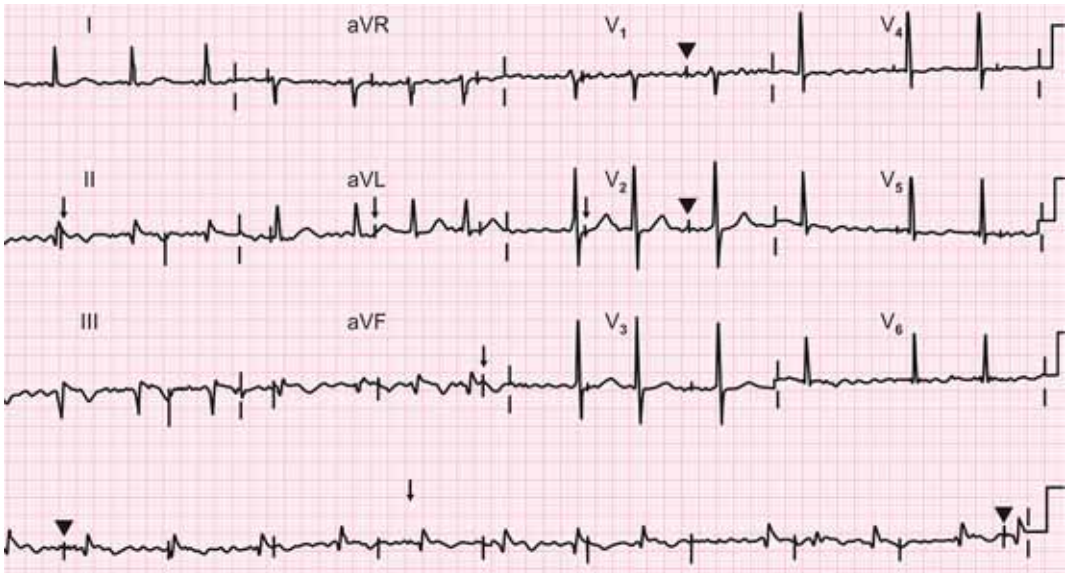


Figure 2.156: Pacemaker failure to sense and capture. Note the pacemaker is firing at a set rate 60 bpm instead of firing when it is needed (failure to sense). Therefore, some pacemaker spikes fall in the refractory period and unable to produce ventricular capture (arrows). But still the pacemaker spikes which are outside the refractory period are unable to produce capture (failure of capture) (arrowheads)

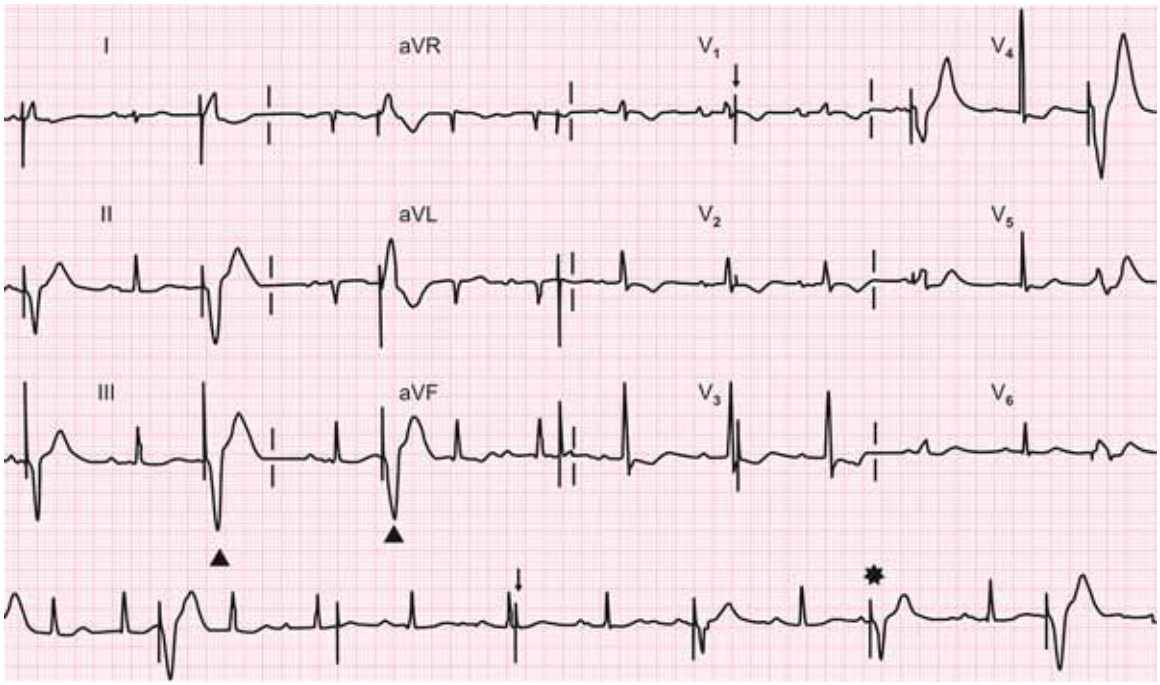


Figure 2.157: Pacemaker failure to sense. Note instead of sensing the intrinsic ventricular rate and then response the pacemaker continues to pace at constant intervals (like fixed rate pacemaker). The result is many pacemaker spikes fall within the ventricular refractory period and fail to capture (arrows), while some do not fall within the ventricular refractory period and result in capture beats seen interpolated between two sinus complexes (arrowhead). Interestingly, some spikes are outside refractory period but still not functioning on demand as impulse from sinus node is also delivered simultaneously so the result is ventricular fusion beats from both sources, i.e. hybrid of sinus node and pacemaker impulses (asterisk, note P wave before the spike)

ECG Changes during Exercise (Stress Test)

During exercise testing (as in tread mill test), ECG changes provide clues to risk of coronary heart diseases and these are considered in the light of Bruce protocol.

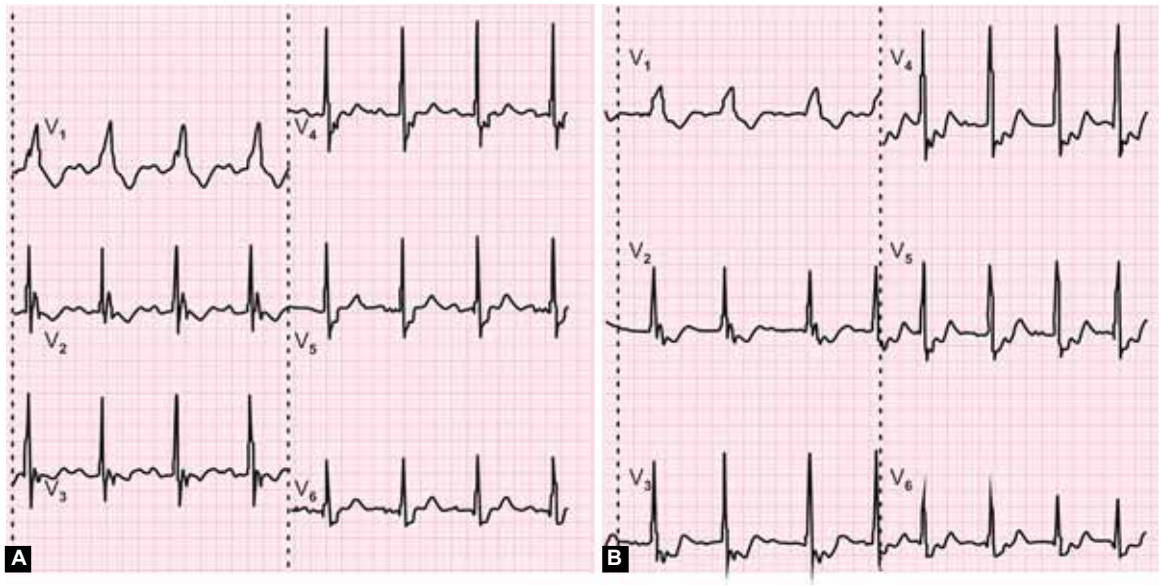
Normal ECG Responses

- Increased heart rate.
- Increased P wave amplitude.
- Shortening of PR and QT interval.
- Decrease in R wave height.
- Shift of QRS to right.
- Depressed J point with up sloping ST segment.
- Variable T wave changes (usually decreased height).

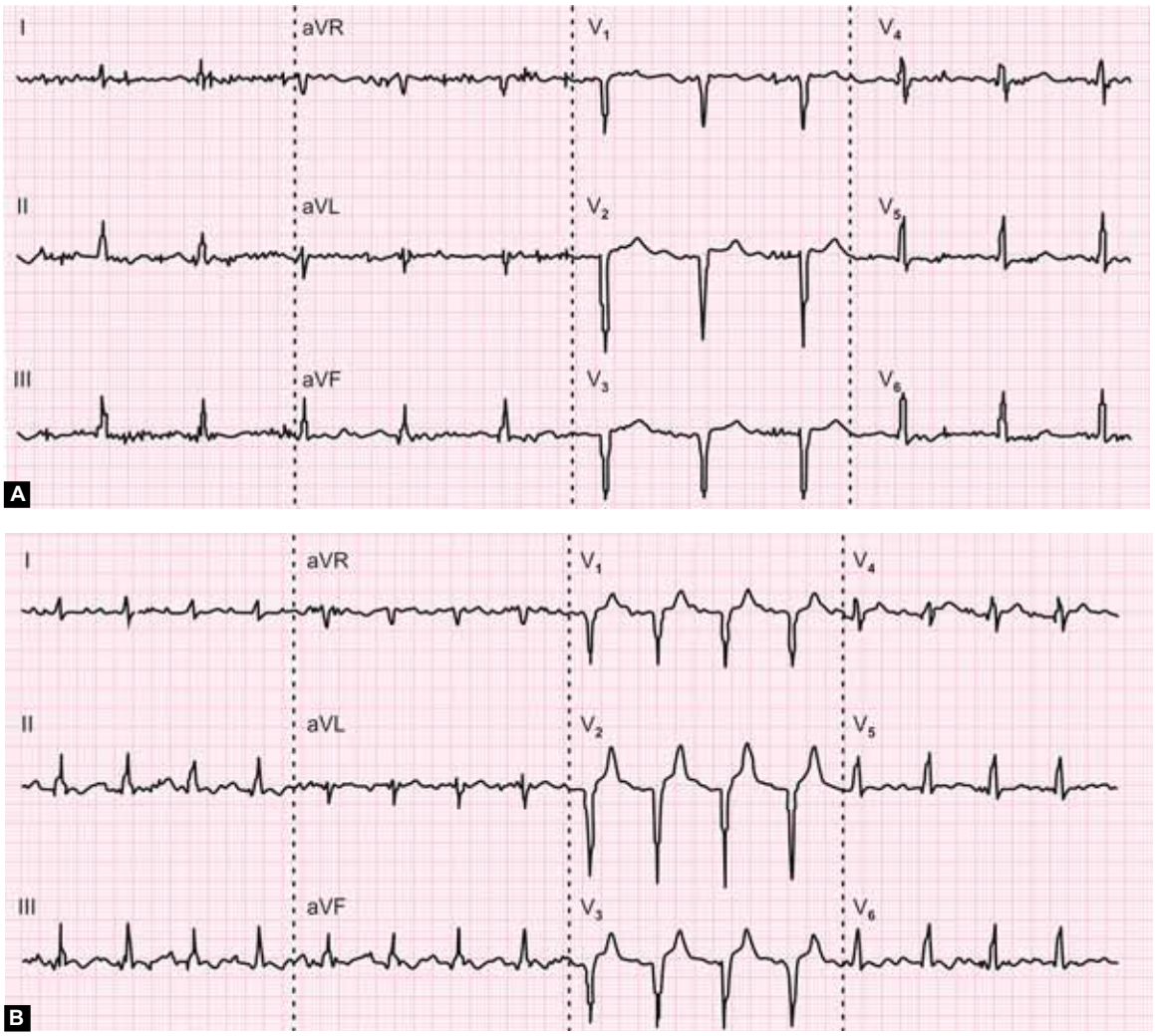
Abnormal Responses Indicating CHD (Coronary Heart Diseases)

Horizontal Down Sloping

- Horizontal or down sloping ST segment depression > 1 mm (more than 3 consecutive beats) is an important diagnostic criteria. (Upsloping ST segment depression; if rapid is not diagnostic for ischemia but slows upsloping is often indicative of ischemia.)
- Early positive response within 6 minutes.
- Persistence of ST depression for more than 6 minutes in recovery phase.
- ST elevation >1 mm in a non-Q wave lead more than three consecutive beats is a strong clue in a Q wave leads (i.e. already infracted).
- "U" wave inversion occasionally seen is precordial leads at heart rates =120 beats/min
- In population with low CAD prevalence pseudo-normalization of T wave (inverted at rest and becoming upright with exercise) is a nondiagnostic feature (Braun Wald).
- Changes in R wave amplitude are not specific and related with level of exercise—also if R wave height is so increased that it meets the criteria of LVH, ST segment cannot be used to diagnose CAD.
- Note these changes may also be due to other causes like (digitalis, hypokalemia, ventricular hypertrophy, BBB, WPW, etc.) so exclude these "false positive results". These false positive results are also common in females.



Figures 2.158A and B: Horizontal or downsloping ST segment depression. (A) Shows ECG before and (B) shows stress ECG. Note ST segment depression more than 1 mm in precordial leads



Figures 2.159A and B: (A) Shows ECG before the stress test having q waves. (B) Shows exercise testing with ST elevation. ST elevation >1 mm in a non-Q wave lead more than 3 consecutive beats is a strong clue in a Q-wave lead (i.e. already infracted). ST elevation usually indicates free wall motion not ischemia

Artifacts

Any outside interference that causes marks on ECG strip other than electrical activity of heart is called "artifact". This makes quick and accurate arrhythmia identification difficult or impossible. Some causes are as follows:

AC-Interference

Inadequate grounding of the ECG machine, excessive leakage of current by an ECG machine located too close to other electronic equipment or an unstable or dry electrode.

Feature

Initial portion of the ECG strip shows no artifact, but the latter portion demonstrates the thickened and darkened tracing.

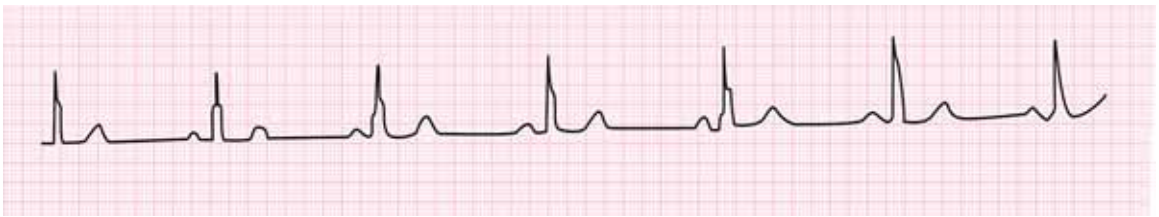


Figure 2.160: Thickened base line in AC interface, note initial portion of ECG is normal but later demonstrates a thickened tracing

Muscle Tremor

Patient is shivering due to cold, uncooperative or not fully relaxed. If patient is immobile this can be minimized by placing electrode on bones on the torso or limbs.

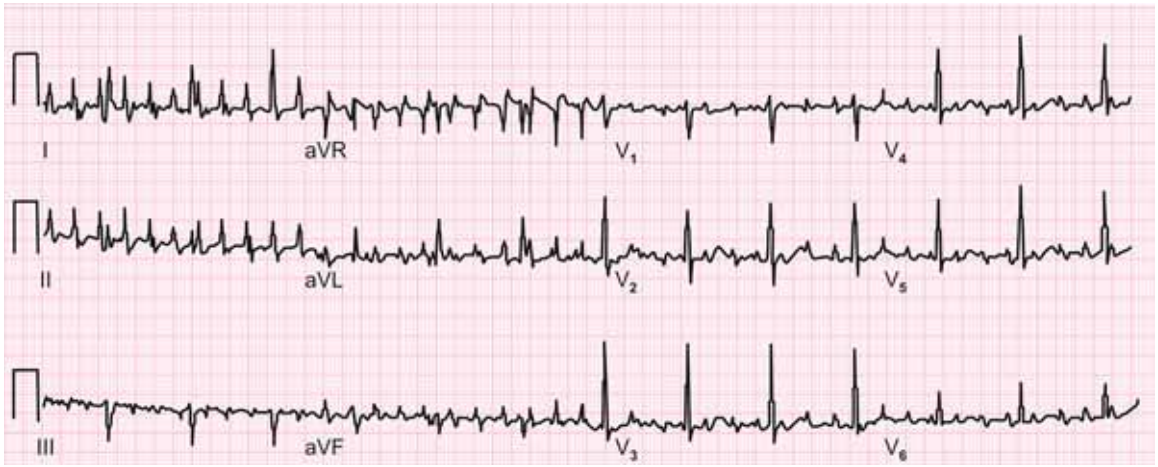


Figure 2.161: The ECG has sinus rhythm but the patient has Parkinsonism with muscle tremors which have obscured the P waves

Wandering Baseline

Excessive patient movement, heavy respiration or unstable electrode (poor contact) which is often due to heavy diaphoresis when patient suffers from acute MI.

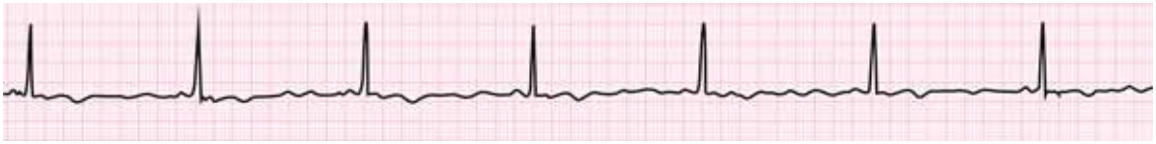
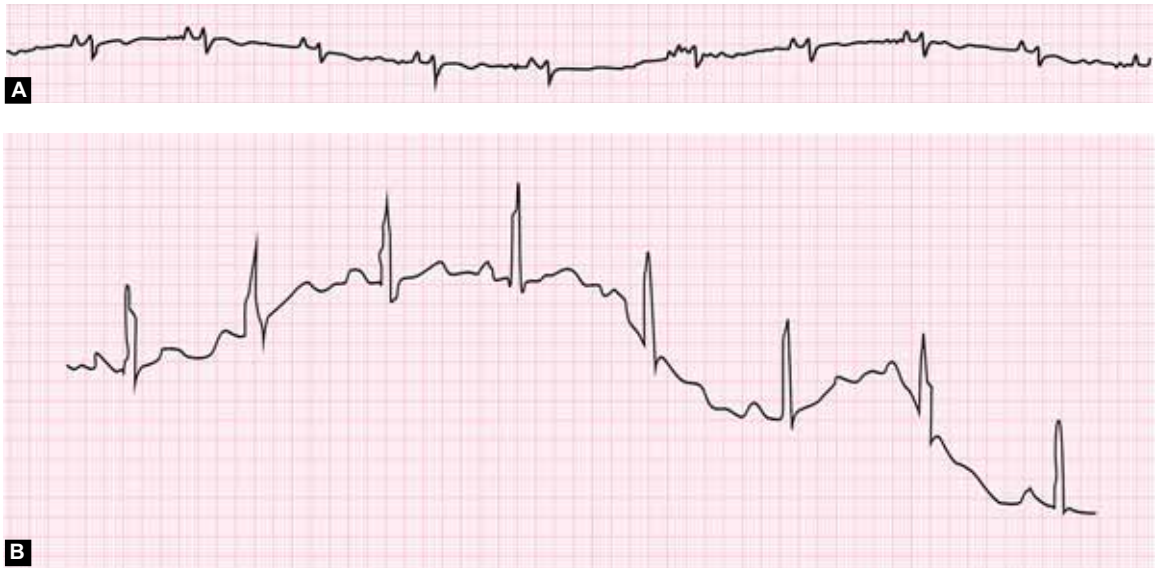


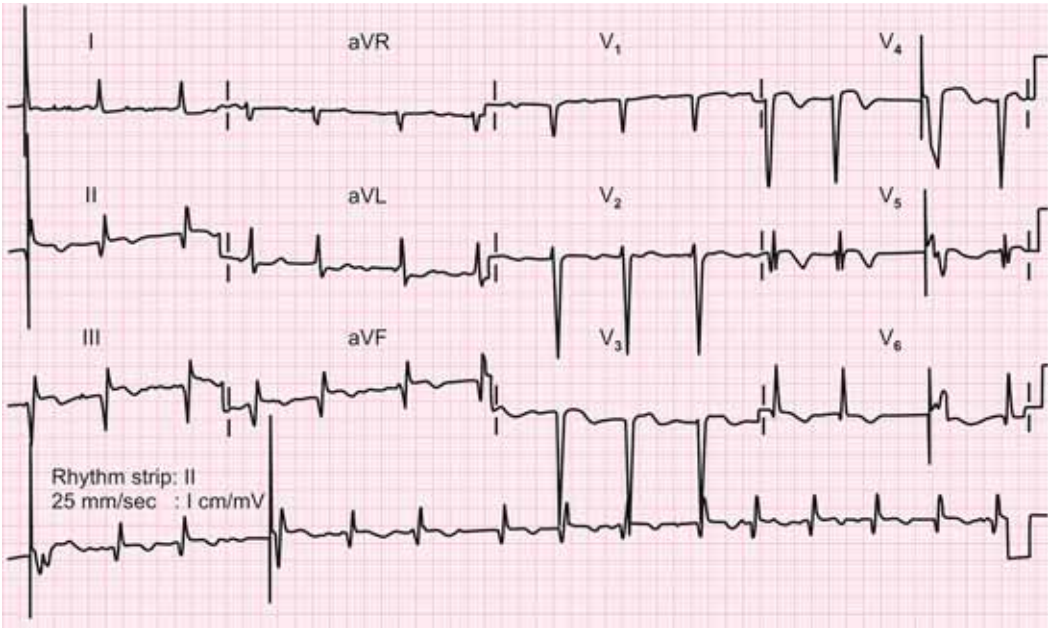
Figure 2.162: This ECG shows irregular base line which may falsely interpreted as atrial fibrillation but here the cause is body tremor



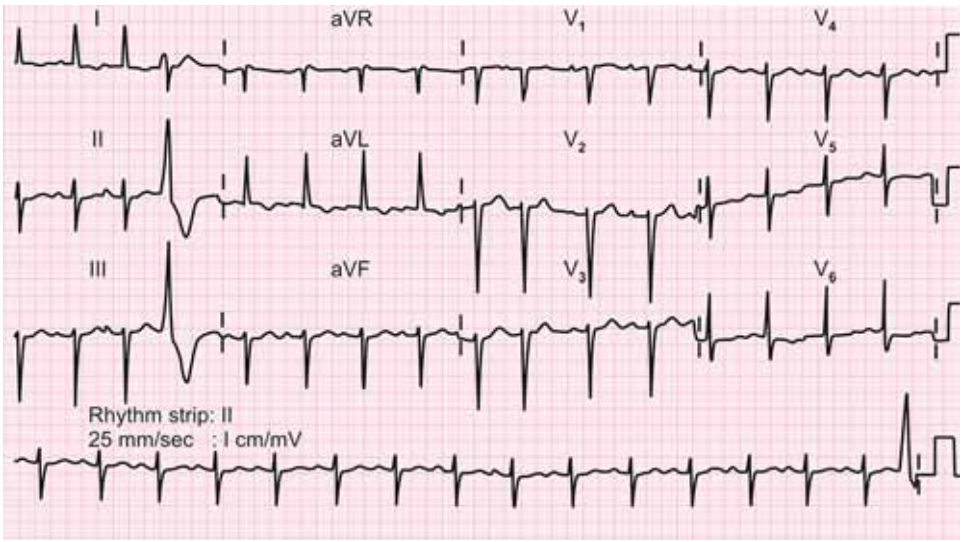
Figures 2.163A and B: Wandering baseline. Note baseline is not at same level. The cause may be excessive movement, poor contact or heavy diaphoresis when patient suffers AMI

SELF-ASSESSMENT

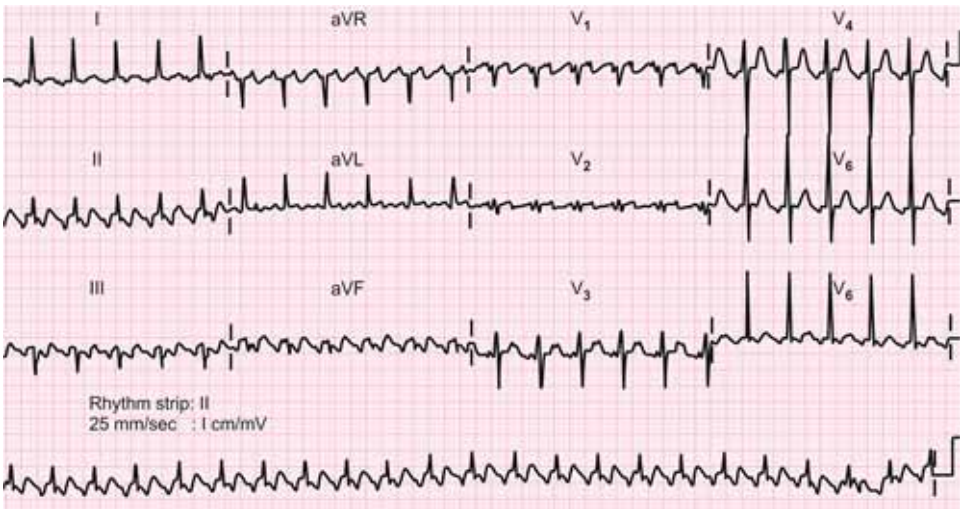
Below are some ECG tracings look and diagnose using our previous discussions and five-finger method. Each ECG contains more than abnormality (See answers on back).



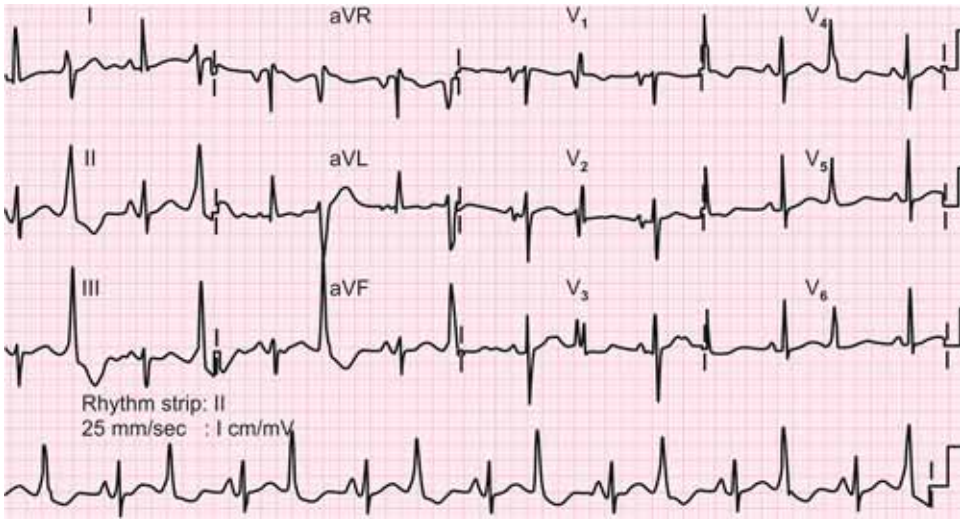
Test 1


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

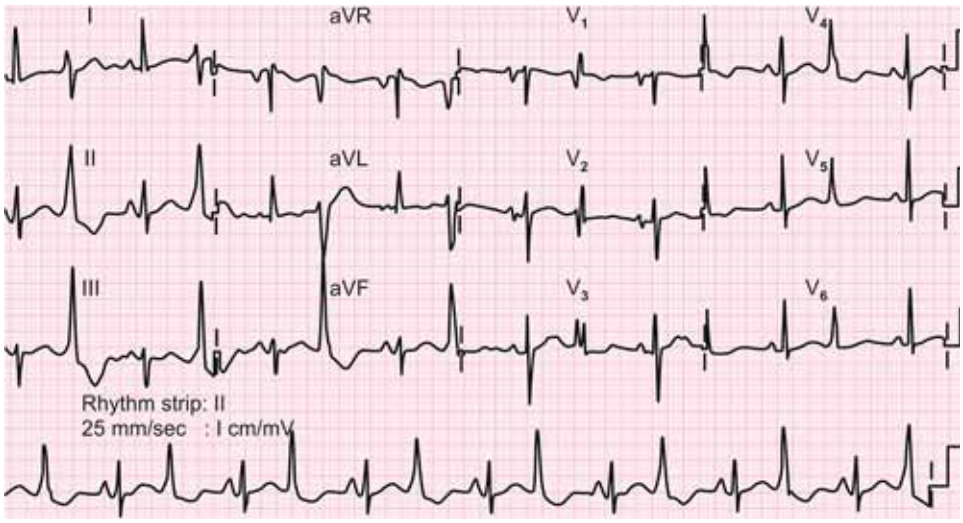
Test 2


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

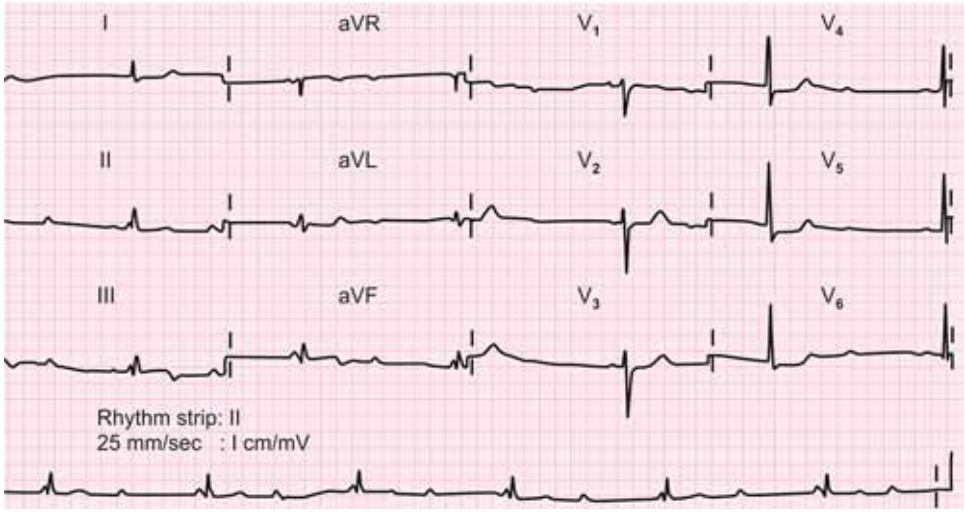
Test 3


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

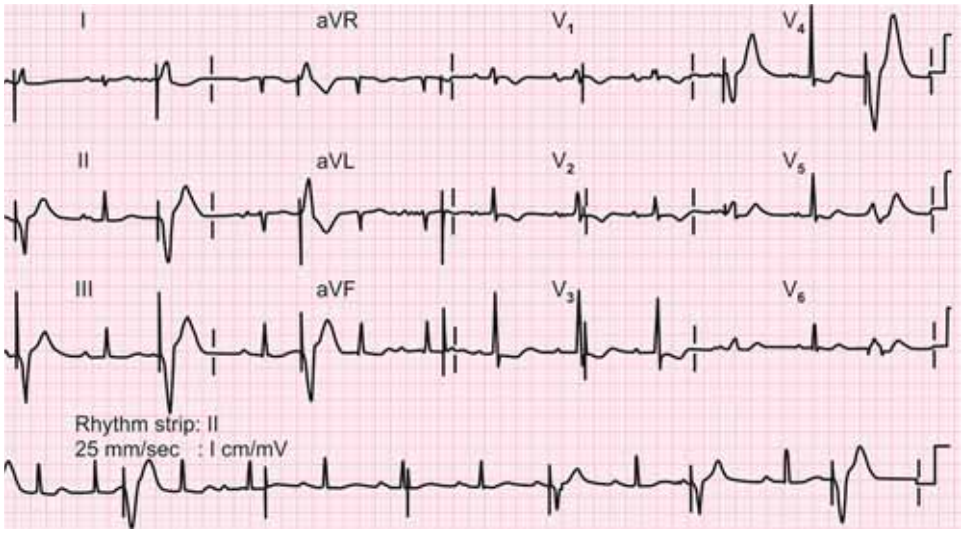
Test 4


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

Test 5


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

Test 6


I
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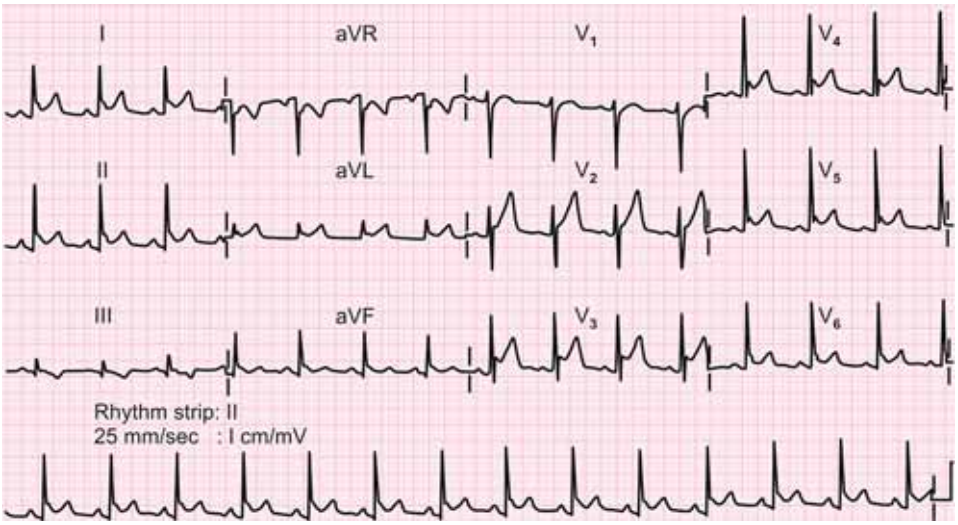
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

Test 7


I
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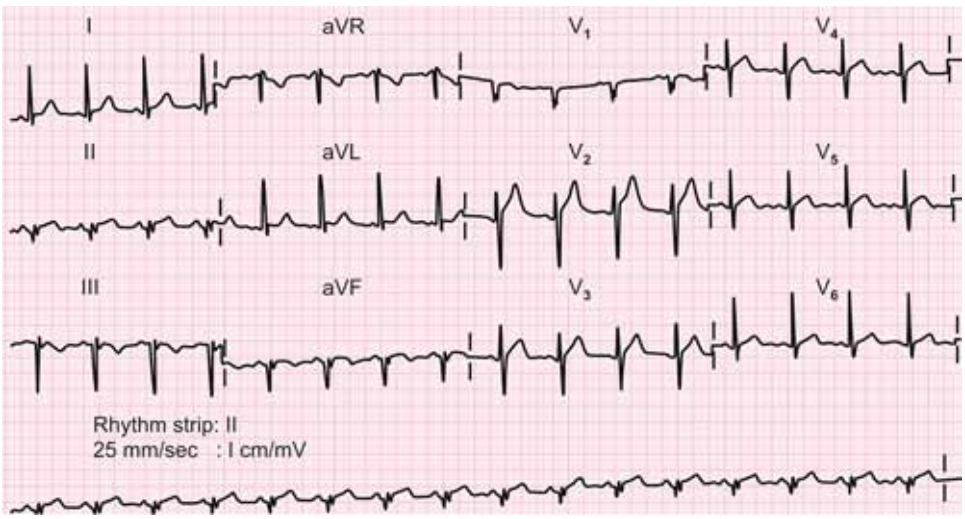
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

Test 8


I
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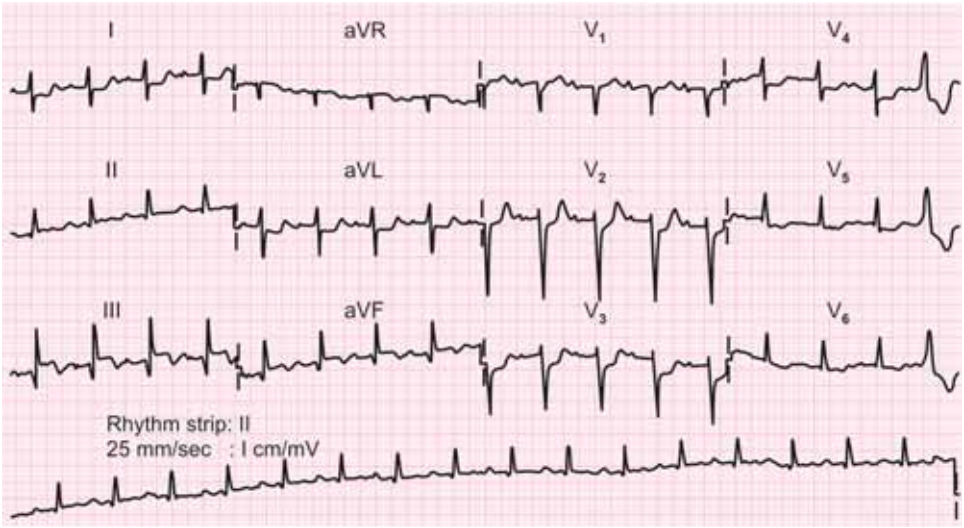
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm strip: II
25 mm/sec : I cm/mV

Test 9



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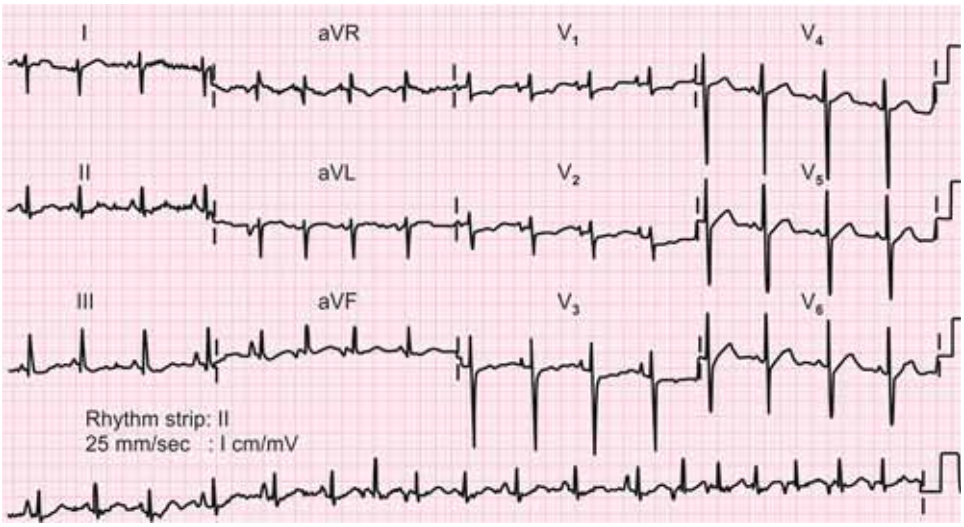

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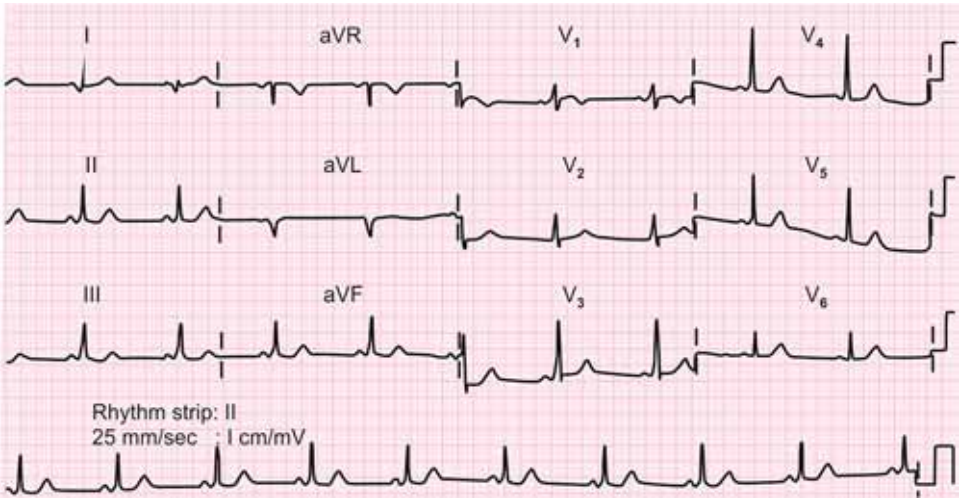

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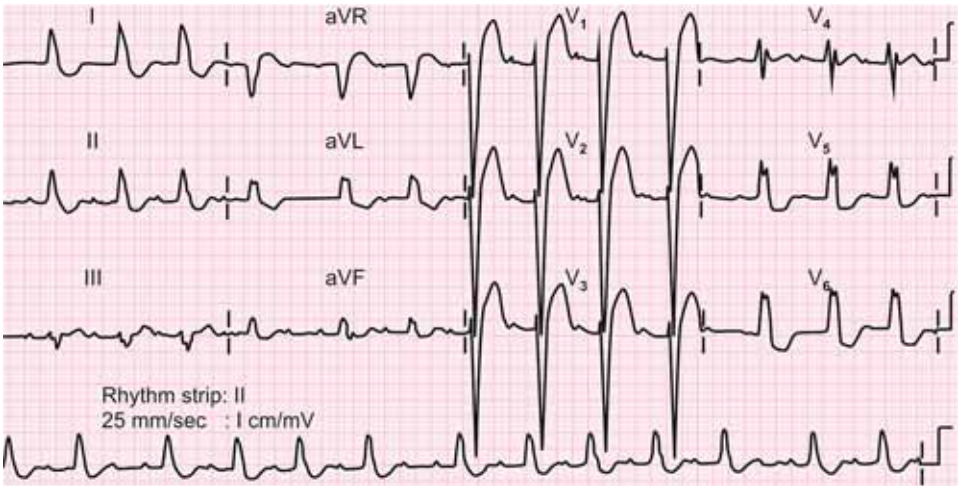

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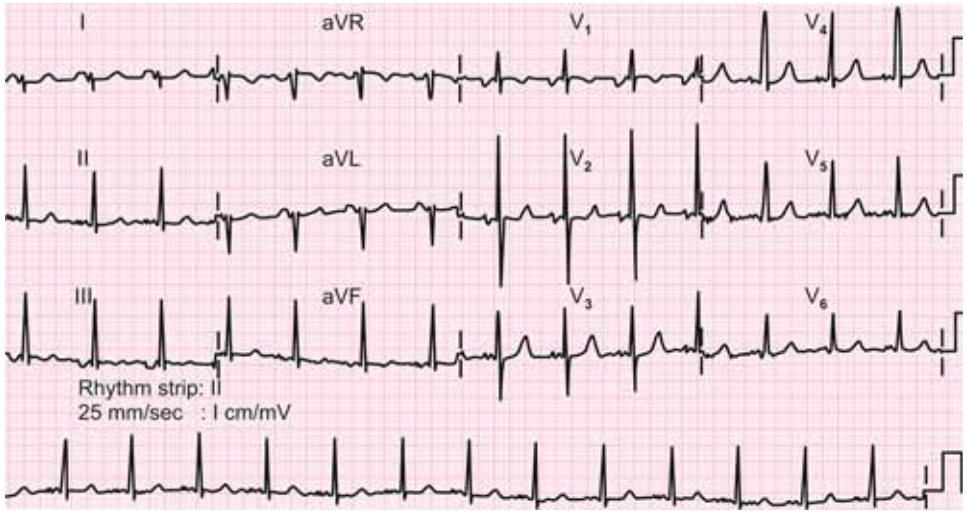

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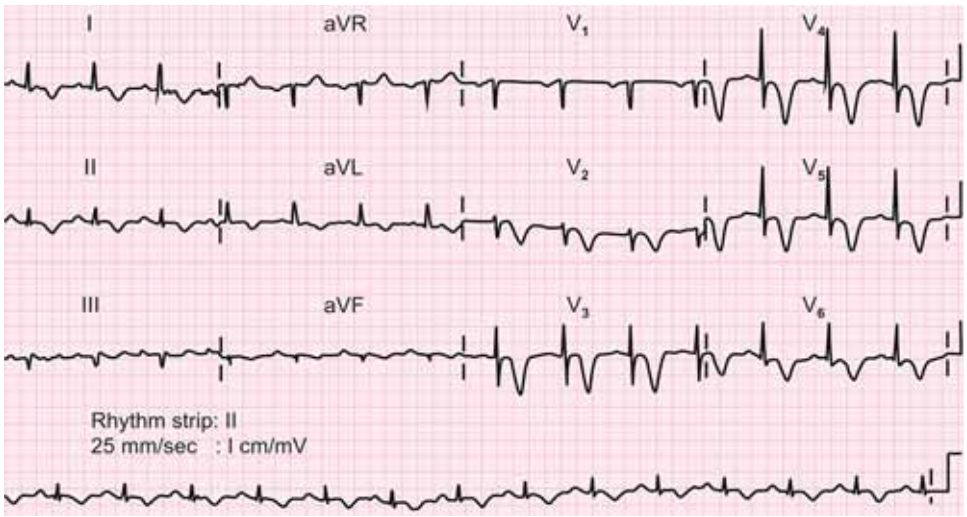

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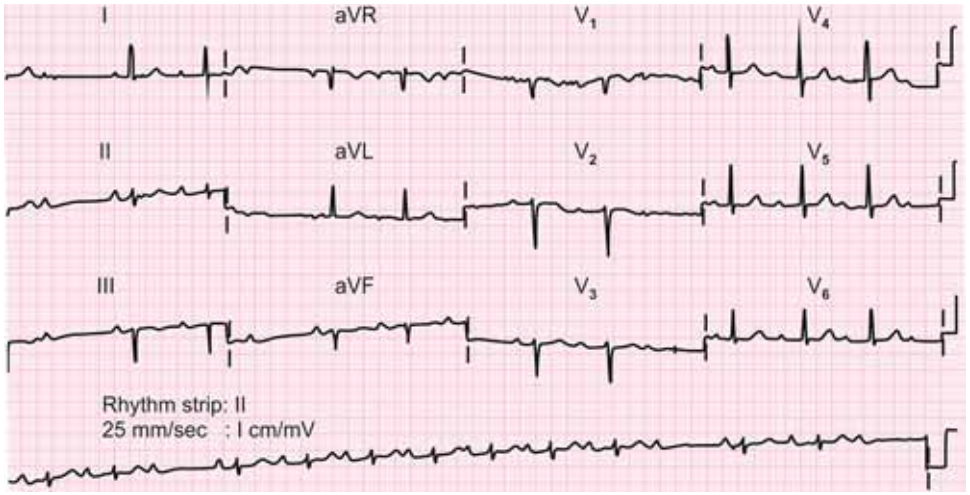

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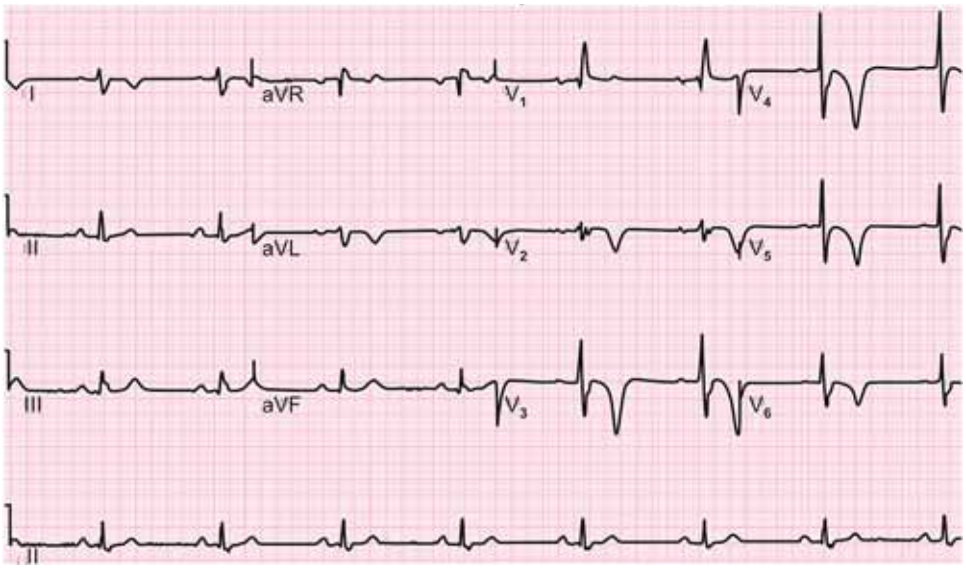

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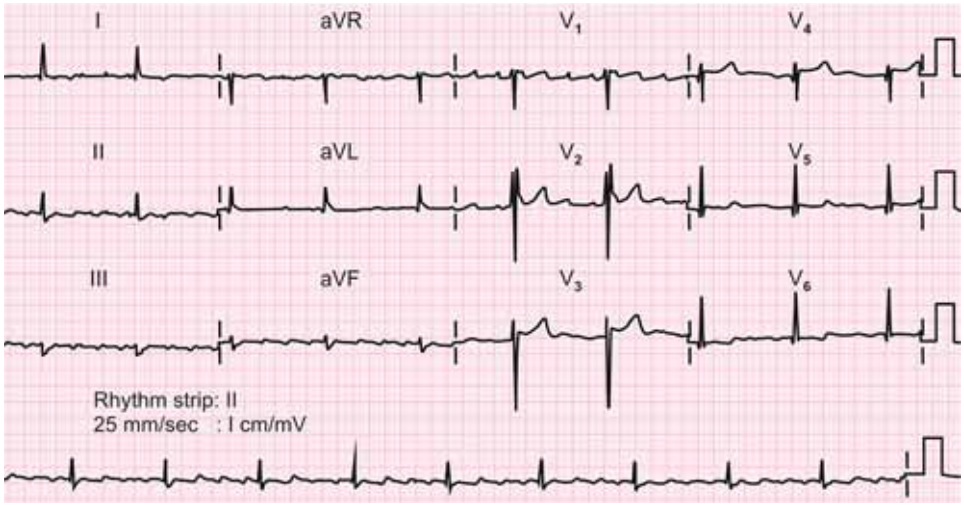

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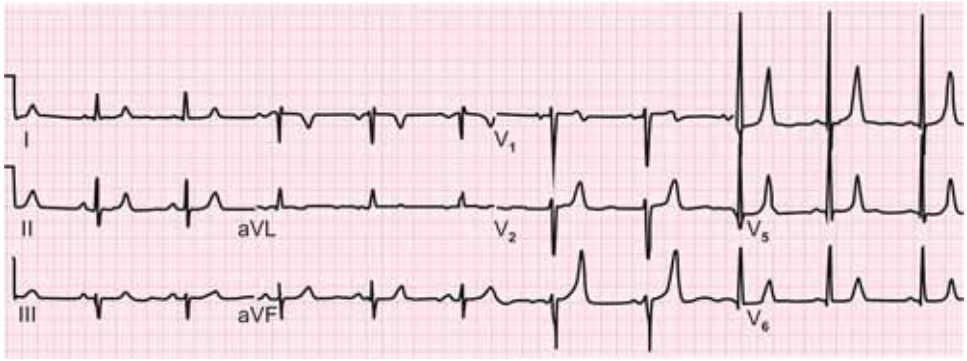

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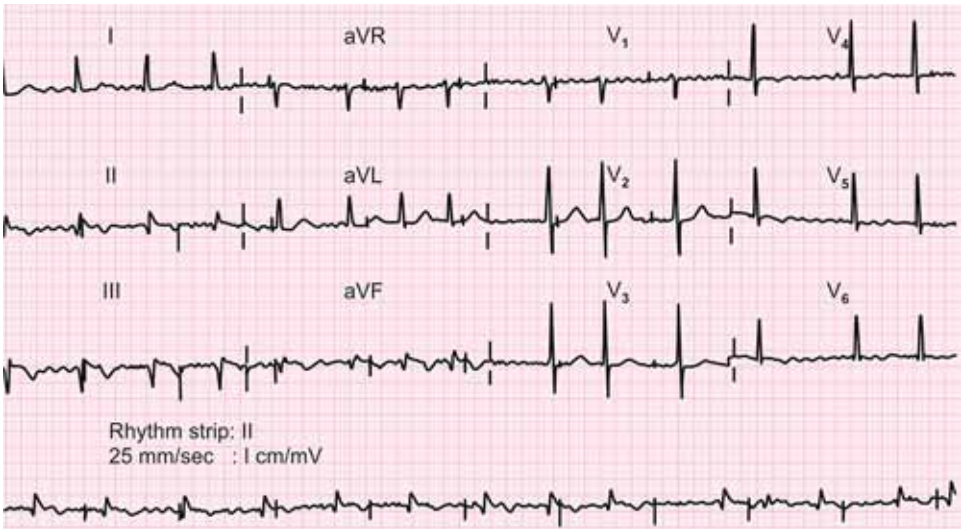

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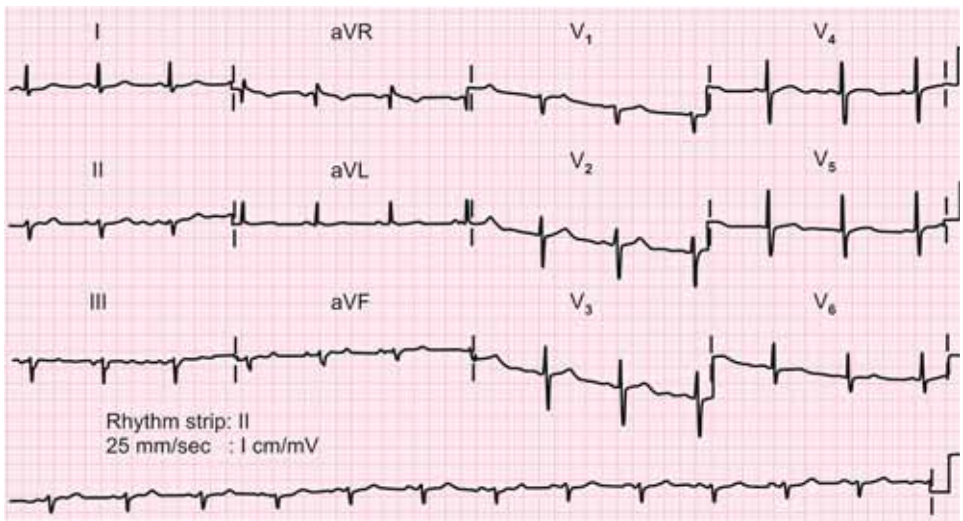

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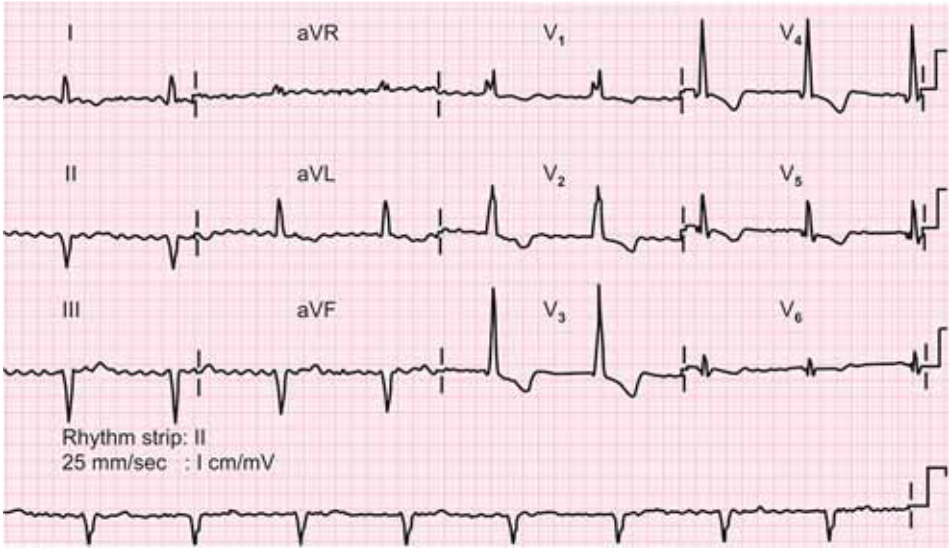

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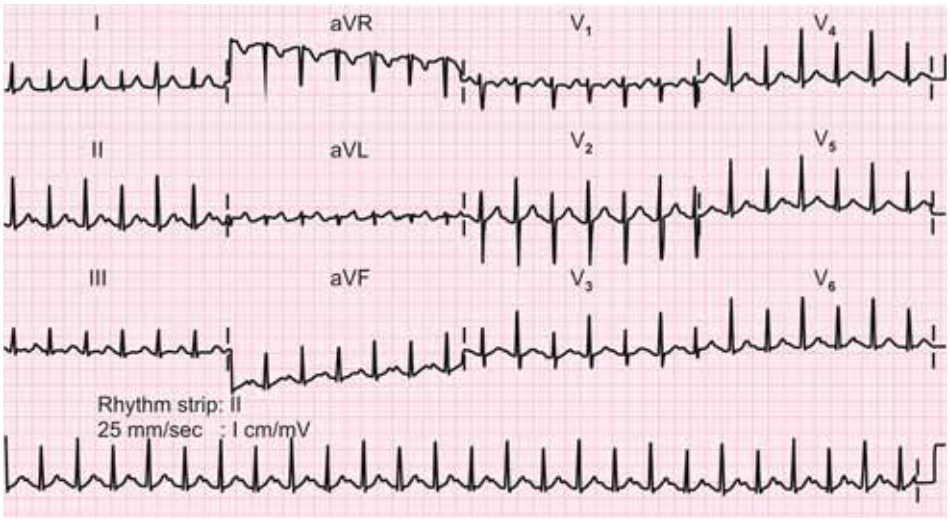

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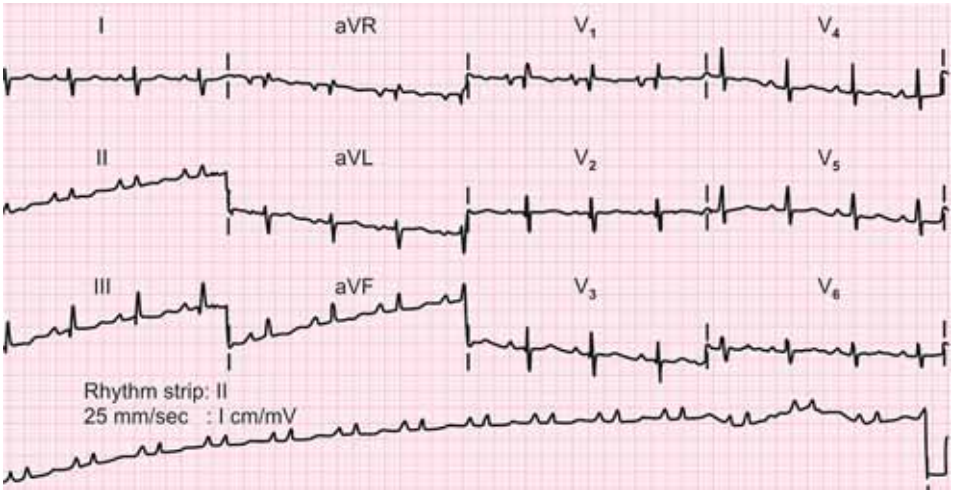

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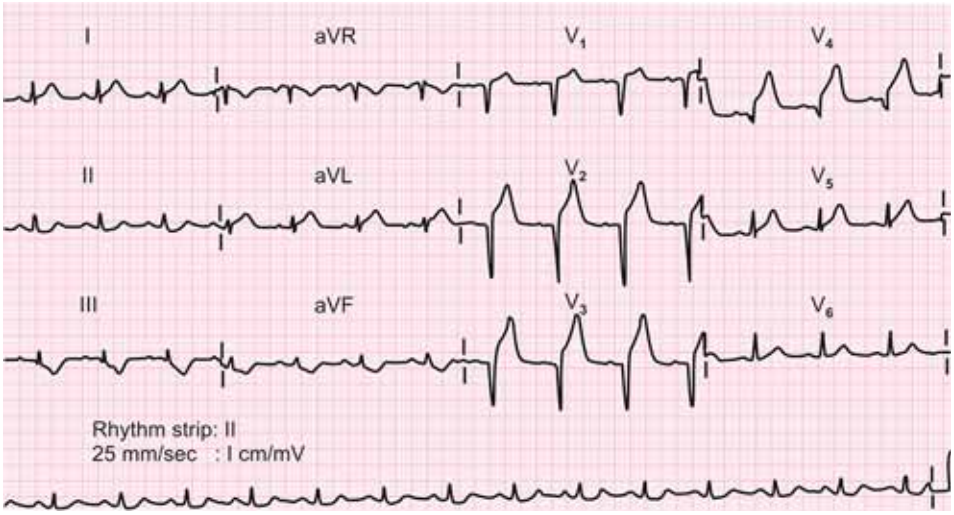

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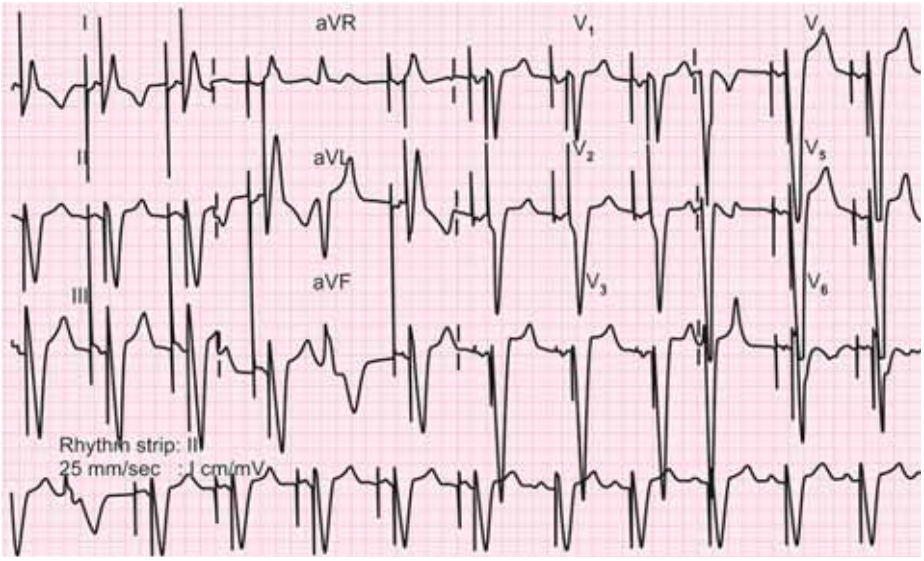
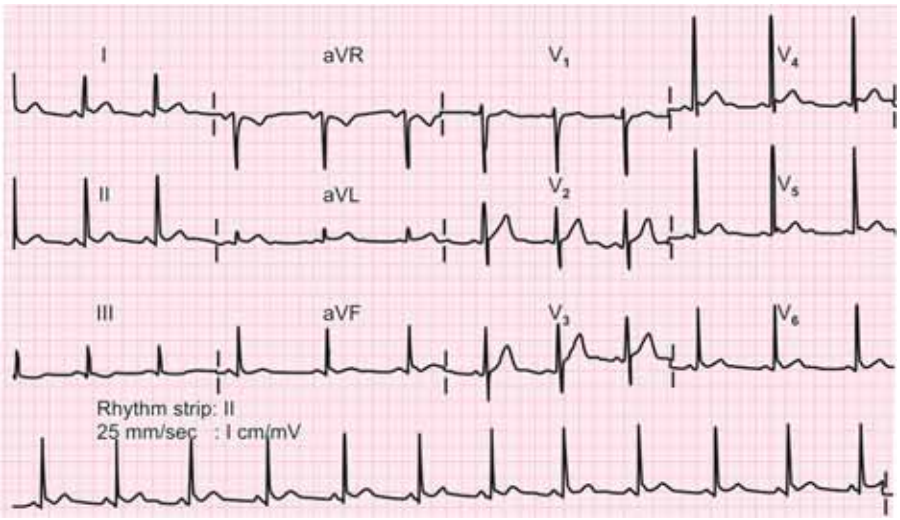
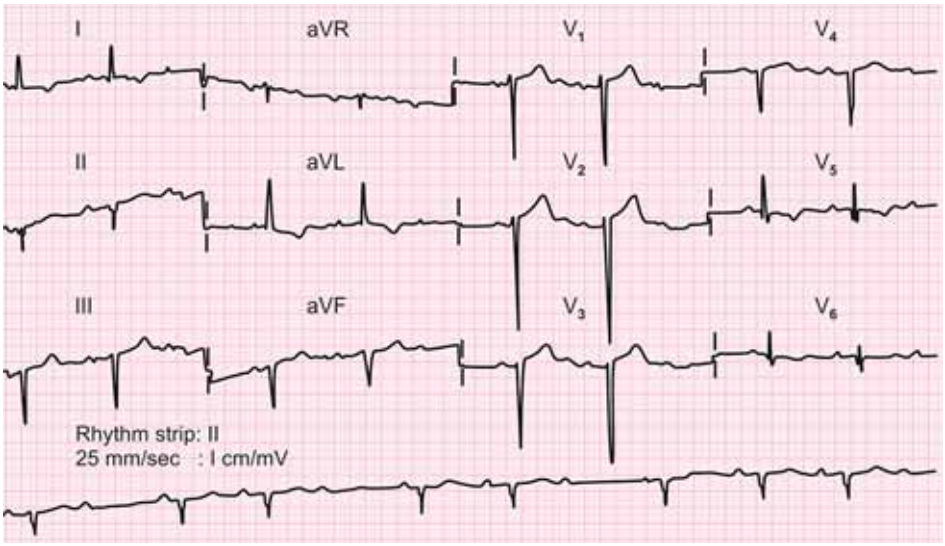


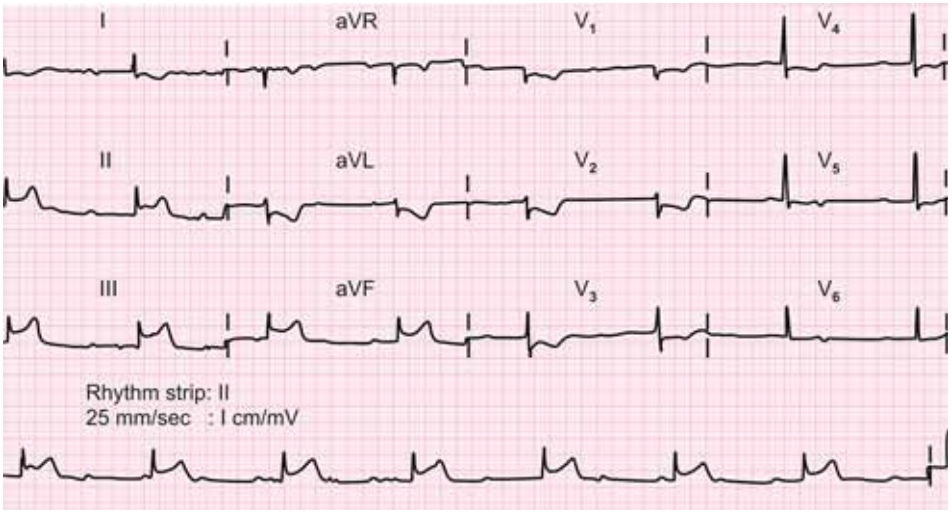
Test 26



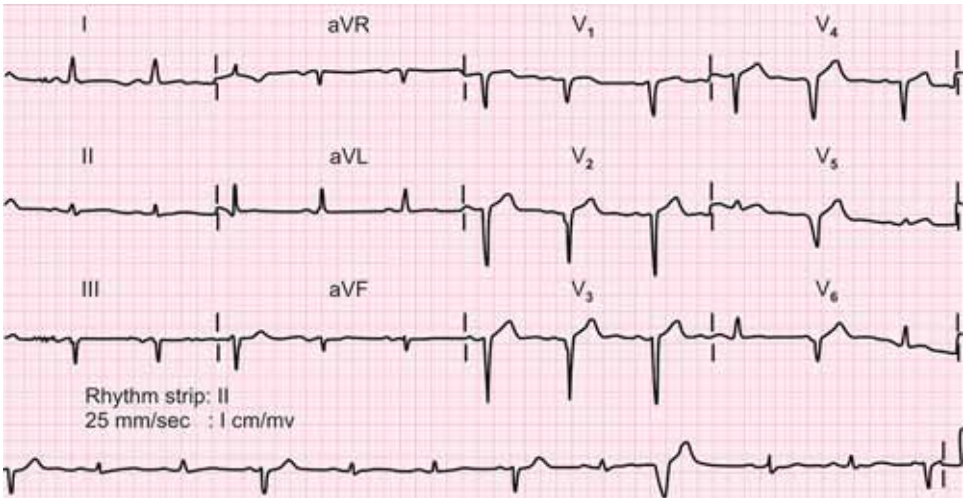
Test 27



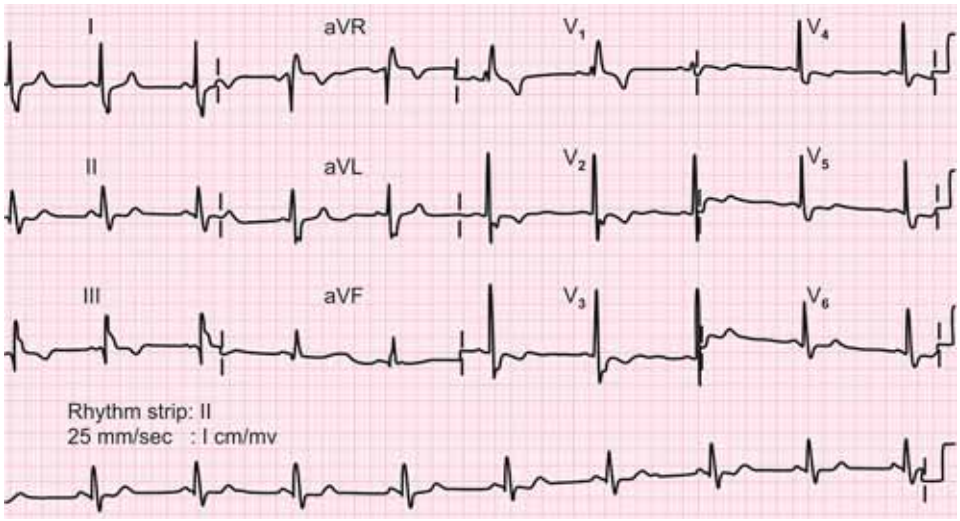
Test 28



Test 29



Test 30



Test 31

Answers

Note answers contain main findings present in the given ECGs with some details; readers are advised to consult the text again and again to have a better grip on diagnostic criteria.

Test 1: Pacemaker spikes can be seen. (sense on demand). Spikes are rightly timed (pacing is appropriate). Atrial fibrillation (note absence of P waves and irregular ventricular response). Inferior wall infarctions (note q waves +ST elevations leads II, III and aVF), anteroseptal MI (note R wave less than three mm in leads V_1–V_4) and Left ventricular hypertrophy (note R wave in lead aVL+ S wave in lead V_3>28 mm).

Test 2: Left axis deviation (note QRS direction in lead I and in lead aVF), left anterior fascicular block LAFB (note small r wave in lead III along with left axis deviation [LAD], VPC, APC 10th beat), LVH (R wave in lead aVL+ S wave in lead III >25 mm), poor R wave progression from lead V_1–V_4 (note R wave lead V_1–V_3 is < 3 mm), ST -T wave abnormalities are due to ventricular hypertrophy (lead V_6).

Test 3: Atrial flutter with 2:1 AV conduction (note "saw tooth" shaped flutter waves [F waves] also note that F waves typically show negative deflection in leads II, II, aVF and in V_1 is positive. While positive in lead V_1), the 2:1 ratio is also a strong clue to atrial flutter, i.e. 2 P waves for 1 QRS.

Test 4: Left atrial abnormality (peaked P wave in lead II, significant P terminal force in lead V_1), LVH (R wave in lead aVL+S wave in lead V_3 >20 mm in this female), Ventricular Bigeminy, i.e. VPCs in bigeminal pattern.

Test 5: Supraventricular tachycardia; SVT (rate 155 bpm). Note the presence of retrograde P waves (more prominent in lead II, II, aVF) support the diagnosis.

Test 6: Complete (3rd degree) AV block. Again AV dissociation (note wandering P waves). There is AV junctional escape rhythm (the QRS complex which is not broad as seen in ventricular rhythm, is a clue toward junctional rhythm) Inferior wall MI (ST elevation in lead III, aVF while ST segment is almost isoelectric in lead II but simultaneous reciprocal ST depression in lead I, aVL and V_2–V_6 support the diagnosis of inferior wall MI.

Test 7: Sinus rhythm. First degree AV block (PR interval is increased 0.26 sec with same duration in all leads), Right Axis Deviation [RAD] (note QRS in lead I and aVF), Right Ventricular Hypertrophy [RVH] (supporting points are RAD and tall R wave in right precordial leads along with ST depression and T wave inversion which indicates strain on the right ventricle), Ventricular pacemaker malfunctioning with complete sensing failure. Note pacemaker spikes are not on demand and they are functioning independently that is why many of them fall within the refractory period; (look rhythm strip), hence failure to capture.

Test 8: Acute pericarditis. Note diffuse ST elevation with upward concavity in most of the leads except lead aVR where there is ST depression. Also note PR depression most pronounced in lead II and aVF which is very specific with acute pericarditis and differentiates it from "early repolarization".

Test 9: Inferior wall MI (note ST elevation in lead II, III, aVF). Anterolateral wall MI (note ST elevation in leads V_2–V_6 and leads I, aVL. Left Axis Deviation [LAD] (note QRS direction in lead I, aVF).

Test 10: First degree AV block (note prolonged PR interval). Inferior wall MI (note ST elevation in lead II, III, aVF, Q wave in lead II, III, aVF which indicate the age is intermediate). Anterolateral wall ischemia (note ST depression in leads I, aVL, V_2–V_6){these maybe due to reciprocal changes}.

Test 11: Periods of Multifocal Atrial Tachycardia [MAT], see rhythm strip (Note the different morphologies of P waves indicating their origin from different foci). Right Axis Deviation [RAD] (note QRS complex in lead I, aVF. There is slight ST depression in beat II, III, aVF and in leads V_1–V_3. The RAD along with ST depression in right precordial leads indicates the possibility of RVH. In fact, multifocal atrial tachycardia is frequently seen with COPD and cor pulmonale in which RVH is usually found.

Test 12: Wolff-Parkinson-White [WPW] syndrome, in the setting of sinus bradycardia (HR around 57 bpm). Note short PR interval, and QRS prolongation (0.10 sec) and the presence of delta waves positive in leads II, III, aVF and in precordial leads while negative in lead I, aVL. Recall negative delta waves can mimic Q waves and can be mistaken as a case of MI.

Test 13: Atrial tachycardia with AV block showing progressive prolongation of PR interval until loss of conduction resulting in a blocked P wave (Wenckebach phenomenon). LBBB (prolonged QRS + wide S wave in lead I and V_6 + "M" shaped QRS in lead V_5, V_6). Also note there is ST elevation in right precordial leads which is usually seen in cases of LBBB so differentiation of this type of elevation of MI is difficult. Importantly, the combination of atrial tachycardia with AV conduction block (atrial tachycardia with block) is very specific of digitalis toxicity.

Test 14: Left atrial abnormality (significant P terminal force in V_1 with RAD). Tall R wave in lead V_1, V_2 indicates RVH. The combination of Left atrial abnormality RAD and RVH is characteristic for Mitral Stenosis.

Test 15: Sinus rhythm (84 bpm). Myocardial ischemia. Diffuse T wave inversion in almost all leads (leads I, II, III, aVL, aVF, V_2–V_6). Note the typical arrowhead T wave inversion (V_3–V_6) which is usually seen in patient with unstable angina. Also note ST segment depression V_3–V_6.

Test 16: Second degree AV block Mobitz type I (Progressive increase in PR interval until failure to conduct P wave resulting in a blocked P wave. Also note poor R wave progression (V_1–V_3).

Test 17: First degree AV block (note prolonged PR interval) RBBB (note wide QRS complex {0.14 sec} + rSR' pattern in lead V_1 and V_2 + wide and deep S wave in lead I and V_6). RAD (See direction of QRS complex in lead I and aVF). Left posterior fascicular blocks (small r wave in lead I + small q wave in lead III in the presence of RAD). Deep T wave inversion leads I, aVL, V_2–V_6 indicating myocardial ischemia.

Test 18: Atrial flutter with 4:1 AV conduction (note "saw tooth" shape flutter waves). Incomplete RBBB (RSR' pattern in lead V_1, V_2 with QRS duration around 0.10 sec with absence of wide and deep S waves in lead I and V_6, hence incomplete RBBB.

Test 19: Hyperkalemia. Note tall, peaked and narrow based (tented) T waves in most of the leads.

Test 20: Acute inferior wall (Q waves in lead II, III, aVF + ST elevation in leads II, III, aVF) and posterior wall MI (tall R wave with R >S and upright T wave in lead V_2), (Remember that posterior wall MI usually seen with inferior wall MI simultaneously). Atrial fibrillation; (unidentifiable P waves with irregular ventricular response). Pacemaker rhythm with pacemaker malfunction (note failure of capture, i.e. you can see the pacemaker spikes without resulting in a QRS complex (rhythm strip).

Test 21: Left anterior fascicular block (LAFB) (Small r wave in lead III + LAD).

Test 22: Atrial fibrillation. Complete AV block (AV dissociation with junctional escape rhythm). RBBB(Wide QRS + rSR' pattern in lead V_1 + wide S wave in lead I and V_5). Left anterior Fascicular block [LAFB] (small q wave in lead I and small r wave in lead III in the presence of LAD).

Test 23: Atrial tachycardia with rate of around 157 bpm (note the presence of P waves buried in T waves) Electrical Alternans (note the alternating heights of QRS complexes). Here the presence of electrical alternans in the settings of atrial tachycardia suggests the presence of atrial-ventricular bypass tract.

Test 24: RVH. Supporting points are:
1. RAD
2. qR pattern in lead V_1 which is very specific for RVH
3. S wave which is slightly > R wave in lead V_6.
   Left atrial abnormality (note biphasic P wave in lead V_1 with significant P terminal force).

Test 25: Extensive anterior and lateral wall MI (ST elevation in ≥ 10 leads), here ST elevation is in lead I, aVL, V_1–V_5. Note that ST depression in leads II, III, aVF is due to reciprocal depression.

Test 26: Dual chamber pacemaker (DDD mode). Pacemaker sense and capture appropriately. Note 2 pacemaker spikes, the first gives P waves and the second give QRS complex.

Test 27: Normal ECG with sinus rhythm showing isolated "J" point elevation seen in early repolarization. This condition is normally seen in some healthy individuals especially the African-Americans.

Test 28: Anterolateral wall MI (poor R wave progression with R wave <3 mm, lead V_1–V_3, loss of R wave in lead V_4, Q waves in lead V_5 and V_6). Second degree AV blocks Mobitz type I (progressive prolongation

of PR interval until P wave fails to conduct. LVH (R wave in lead aVL + S wave in lead V_3 > 28 mm in this man, S wave in lead V_2 + R wave in lead V_5 > 35 mm), LAD.

Test 29: Acute inferior wall MI (ST elevation in lead II, III, aVF; with reciprocal ST depression in leads I, aVL and precordial leads along with T wave inversions). 3rd degree AV block (complete heart block with AV dissociation and junctional escape rhythm.

Test 30: Anterior wall MI (Intermediate age). Note QS waves in lead V_1 and V_2 with loss of R wave in lead V_3 and V_4. VPCs (1st, 4th, 11th beats). Fusion beats are clearer in rhythm strip (1st, 4th, 7th, 12th beats, note variable morphology of these beats and presence of P waves before these beats indicate that these beats are hybrid of impulses from sinus node and ectopic foci from the ventricle.

Test 31: RBBB (Note wide QRS complex 0.12 sec + deep and wide S wave in lead I and V_6 + rSR' pattern in lead V_1). Also note sinus bradycardia (HR around 58 bpm).

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