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CME MODULE

ECG AND PRIMARY CARDIAC CARE MANAGEMENT

STATE INSTITUTE OF HEALTH AND FAMILY WELFARE, UTTAR PRADESH

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MESSAGE

Shri Brajesh Pathak

Hon'ble Deputy Chief Minister
Hon'ble Minister of
Medical Health and Family Welfare
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Government of Uttar Pradesh

Continuing medical education (CME) allows medical professionals to update their knowledge base and opportunity for knowledge creators to share their learning with breadth of the medical community. CMEs provide an opportunity for all medical professionals to come together and get to know each other, thereby leading to excellent networking opportunities.

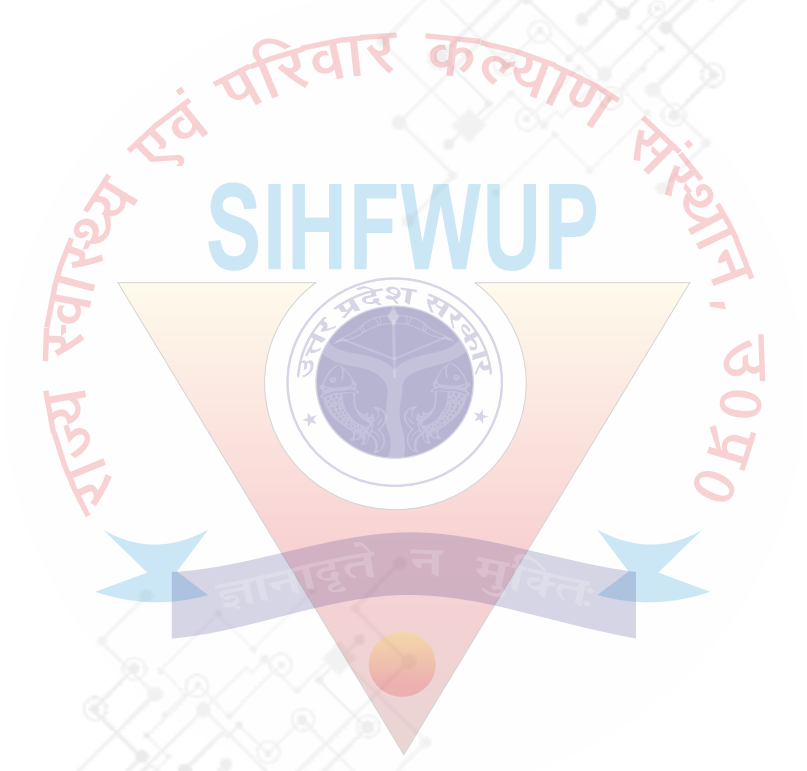
To achieve sustainable development goals and reduce global burden of non- communicable disease, the Government of Uttar Pradesh in its efforts to improve its Healthcare Ecosystem, intends to make giant strides through CME by incorporating technological and medical advances.

PHCs/CHCs serves as a first port of call to a qualified doctor in the public health sector and though these CME programmes recent knowledge and skills will be imparted to Medical Officers in systematic manner to update the existing proficiency of Medical Officers. This will definitely improve the patient care, patient confidence and patient satisfaction.

In lieu of the above the State Institute of Health & Family Welfare, Uttar Pradesh (SIHFW), has started developing Modules for CME which are need of the hour required for our health personnel. I hope that this module on CME on ECG and primary cardiac care management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, will aid our Medical Officers in knowledge up gradation on concurrent intervention practices.

I wish the team SIHFW that they should continue developing such module on CME for the benefit of Medical Officers in Provincial Health & Medical Services in Uttar Pradesh that will ultimately benefit their patients too.

(Brijesh Pathak)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MESSAGE

Shri Mayankeshwar Sharan Singh

Hon'ble State Minister
Medical Health and Family Welfare Department
Government of Uttar Pradesh

I am proud of the fact that the State Institute of Health & Family Welfare, Uttar Pradesh (SIHFW) through this module on Continuing Medical Education (CME) on Electrocardiographic (ECG) and primary cardiac care management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, is addressing the need of knowledge upgradation in Electrocardiographic and its primary management.

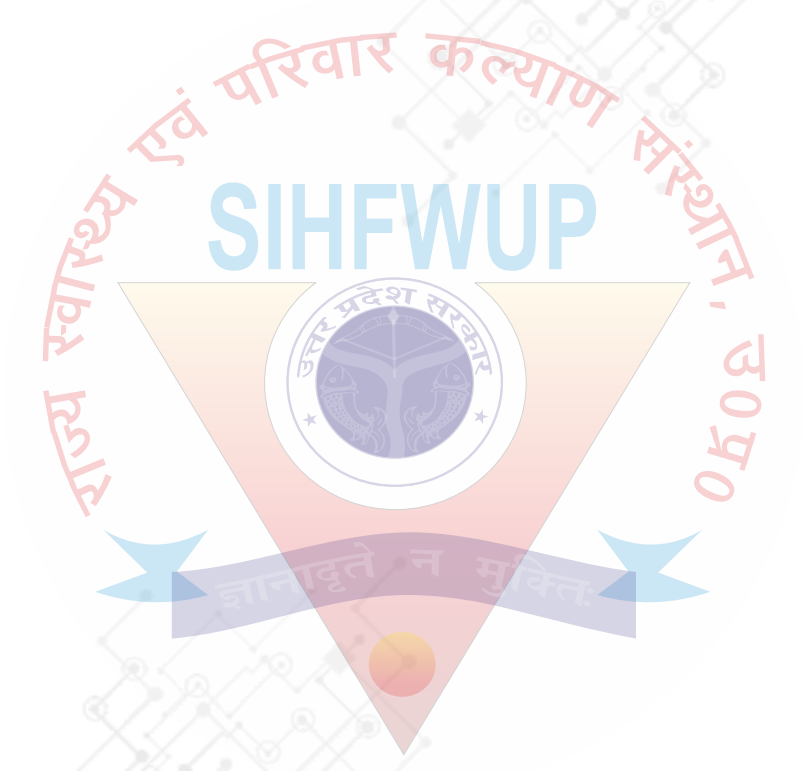
Chronic and catastrophic disease remains one of the main factors that push households from poverty into deprivation. Cardiovascular disease is the most common non-communicable disease imposing burden on human health worldwide as well as in India.

This module on CME on ECG and primary cardiac care management intends to facilitate our Medical Officers to address the better understandingabout primary cardiovascular care management and in turn improve the healthofthe served population.

It is important to note that to achieve the desired goals and objectives of the Department of Health and Family Welfare, we must enhance the skills of our Medical Officers in order to cater to the demands of public health services with best of their capabilities. This CME module on ECG and primary cardiac care management will definitely serve as a tool to achieve above mentioned goal.

I wish team at SIHFW success in their endeavors of aiding an improved health service delivery system through such CME on ECG and its care management.

(Mayankeshwar Sharan Singh)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FOREWARD

Shri Partha Sarthi Sen Sharma

Principal Secretary
Department of
Medical Health and Family Welfare
Government of Uttar Pradesh

Continuing Medical Education (CME) module is a mechanism for medical professionals to stay abreast of the rapidly evolving practices in medical and medicine. In the COVID era, it has become more essential for medical officers to keep up with the mode of treatment and management developing in conjunction with the feedback from the medical community.

The availability of both ECG equipment and interpretational skills are not universal at the primary care level. Obtaining rapid, accurate ECG reports with specialist input remains a challenge. Observations from the field have indicated significant heterogeneity in the level of interpretational skill among Medical Officers.

To assuage and remedy this situation, this CME module on ECG and primary cardiac care management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh has been developed by State Institute of Health & Family Welfare (SIHFW), Uttar Pradesh with the help of subject matter experts .

The module is a composite interpretation of recent development in screening, prevention and primary management of cardiovascular disease with inputs required for Medical Officers to enhance their skills and knowledge, ultimately leading to improved health care services to the masses.

I would like to take this opportunity to congratulate SIHFW and other subject matters experts in developing such a comprehensive module. I hope this CME module will provide scope to revisit ECG and its primary cardiac care management.

(Partha Sarthi Sen Sharma


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MESSAGE

Dr. Renu Srivastava Varma
Director General
Department of
Medical Health and Family Welfare
Government of Uttar Pradesh

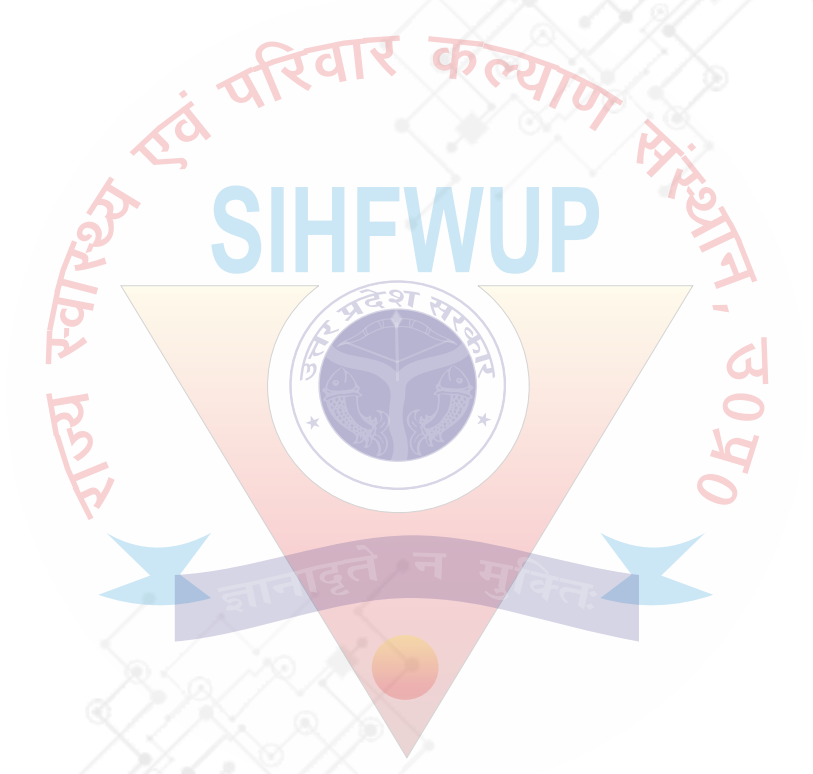
Electrocardiogram (ECG) is a useful diagnostic tool in the diagnosis and management of ischaemic heart disease and cardiac arrhythmia, and its availability in the primary care setting is now common. Routine availability of ECG at the primary care level can facilitate early referrals to secondary care, while reducing unnecessary referrals where appropriate.

Through this module on Continuing Medical Education (CME) on ECG and Primary Cardiac Care Management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, State Institute of Health & Family Welfare (SIHFW), Uttar Pradesh has developed a comprehensive CMEs detailing out steps in essential cardiac anatomy and physiology, the ECG grid, identifying tachyarrhythmia, bradyarrhythmia, mayocardial ischemia and infraction, ECG changes with medications and electrolyte disturbance & miscellaneous conditions and primary cardiac care at PHC level, which will help in early screening, detection, referrals and treatment of patients.

I hope that after this CME,Medical Officers in Uttar Pradesh will be able to scale up the service delivery in provide screening, management, referral and treatment in their health facilities, thus benefitting communities. In addition to improving clinical and technical area of expertise, this CME will lead to providing improved access to health services and enhancing patient satisfaction and population health.

With the development of this module on CME on ECG and Primary Cardiac Care Management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, SIHFW has established genuine links between the theory and practice of healthcare management. I wish team SIHFW the best and hope that many such customized CME modules will be published in the near future.

(Dr. Renu Srivastava Varma)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MESSAGE

Dr. Anita Joshi
Director General Family Welfare
Directorate of Family Welfare
Government of Uttar Pradesh

This module on Continuing Medical Education (CME) on ECG and primary cardiac care management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh is intended to target even those Medical Officers who are posted at CHCs/PHCs. This module will heighten the importance on the role of the Medical Officers at CHCs/PHCs in screening, prevention and primary management of cardiovascular disease, and review public health services in the districts, accordingly.

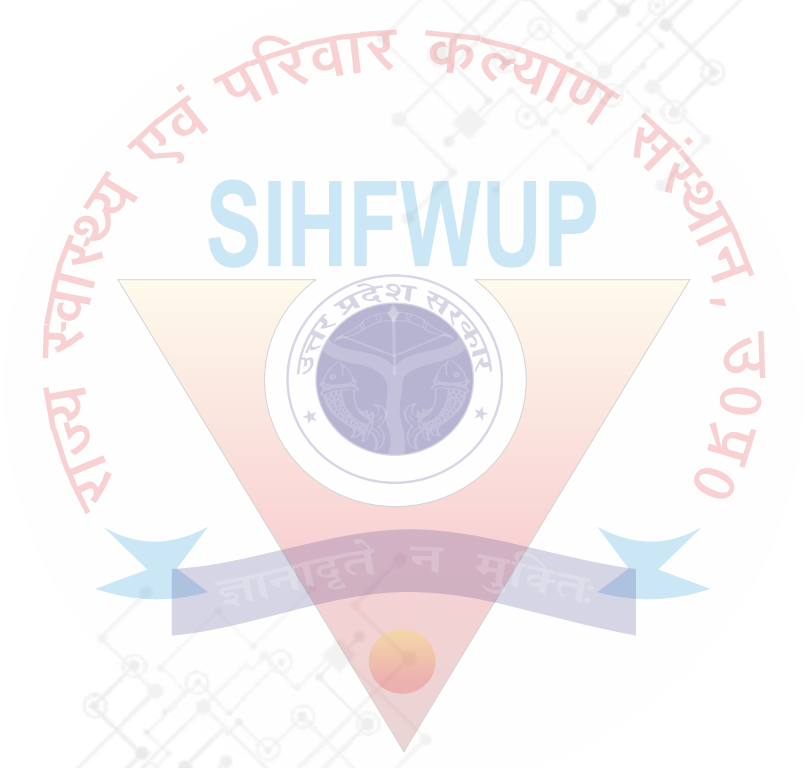
By collating the relevant information in the field of electrocardiogram, covering all domains such as essential cardiac anatomy and physiology, the ECG grid, identifying tachyarrhythmia, bradyarrhythmia, mayocardial ischemia and infraction, ECG changes with medications and electrolyte disturbance & miscellaneous conditions and providing case studies to illustrate best practice, the module seeks to be a working document which can also be reviewed and updated periodically based on the experience of the implementation of the public health services.

I am especially pleased to note that although CMEs in Uttar Pradesh healthcare ecosystem are still in its nascent stage but such developments will lead the way forward in creating tailored CMEs for diagnosis, referral services, patient safety, quality care management and authentic knowledge delivery.

It is vital to introduce CMEs combining video and live demonstrations along with simulation-based skill modules and rigorous assessments led by experts in order to measure further impact.

I congratulate the faculties at State Institute of Health & Family Welfare, Uttar Pradesh in developing this convergent module along with experts from the field of Medicine. This module addresses the need to have a holistic view on public health by also discussing other relevant guidelines and policies that seek to public health service delivery system.

(Dr. Anita Joshi)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MESSAGE

Dr. Deepa Tyagi

Director General (Training)
Medical, Health & Family Welfare
Government of Uttar Pradesh

This module on Continuing Medical Education (CME) on ECG and Primary Cardiac Care Management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh provides a comprehensive, coherent and research-based insight to ECG and Primary cardiac care management. It has been designed and written for Medical Officers and healthcare professionals and takes government perspective in consideration, drawing upon and comparing ideas and developments from national and international health care practices.

The module is structured in such a manner that it covers in the field of cardiac care management, covering all domains such as anatomy and physiology of heart, ECG grid, identifying tachyarrhythmia andbradyarrhythmia, mayocardial ischemia and infraction, ECG changes with medications and electrolyte disturbance & miscellaneous conditions in the context of public health and providing an overview of about future development andtrends in ECG and cardiac care management.

The faculties at State Institute of Health & Family Welfare, Uttar Pradesh has done a commendable job by publishing this module on CME on ECG and Primary cardiac care management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh. I hope the participants coming to attend their upcoming CME will take advantage of this initiative and make the most in their field with this handy module.

(Dr. Deepa Tyagi)


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ACKNOWLEDGEMENT

Dr. Rajaganapathy R.
Director
State institute of
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The purpose of Continuing Medical Education (CME) is to facilitate life-long learning among Medical Officers so that their practices may reflect the best medical care for their patients. The goal of CME is to help Medical officers enhance their performance in practice, in turn enhancing patient care and satisfaction.

In the health care practices significant effort has been undertaken to establish the role of routine ECGs as part of cardiovascular risk assessment in asymptomatic adults in the general population. It has been observed that abnormalities on resting ECG were associated with an increased risk for subsequent cardiovascular events after adjustment for traditional risk factors.

However, a customized CME is the need of the hour is to provide exposure to Medical Officers in in Provincial Health & Medical Services in Uttar Pradesh in recent development in ECGs, cardiovascular risk assessment and management.

To meet this goal and knowledge upgradation, the faculties of State Institute of Health and Family Welfare (SIHFW), Uttar Pradesh with the help of faculties from Department of Community Medicine, Dr. Ram Manohar Lohia Institute Of Medical Science (RMLIMS), Lucknow has conceived and giving action to this CME module on ECG and Primary Cardiac Care Management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh .

I congratulate the faculties at SIHFW & RMLIMS for coming up with the CME module. I am looking forward to a wider dissemination of this module and feedback on its efficacy in the coming months.

(Dr. Rajaganapathy. R)

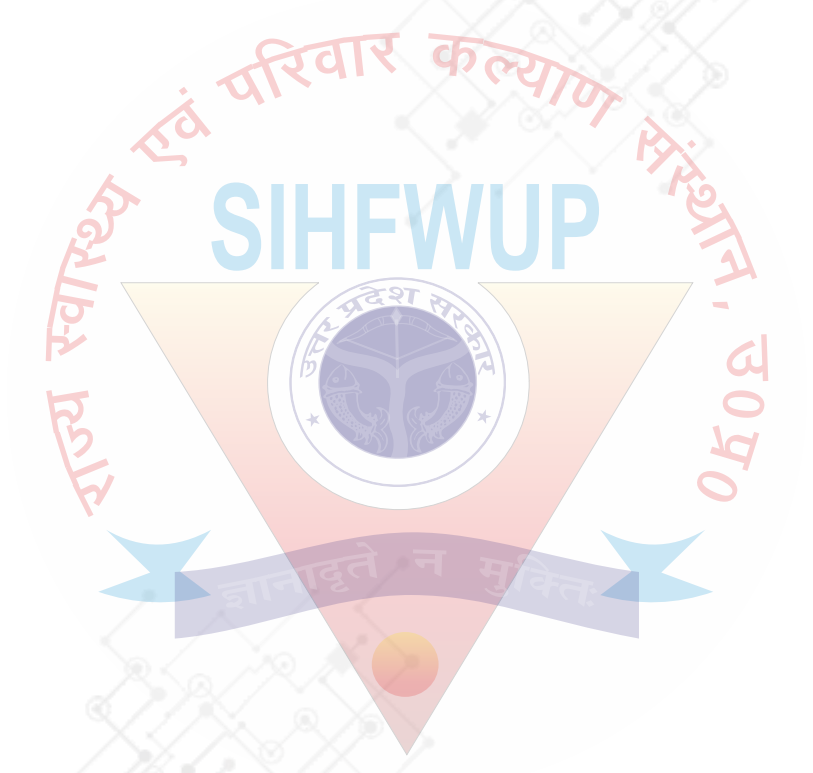
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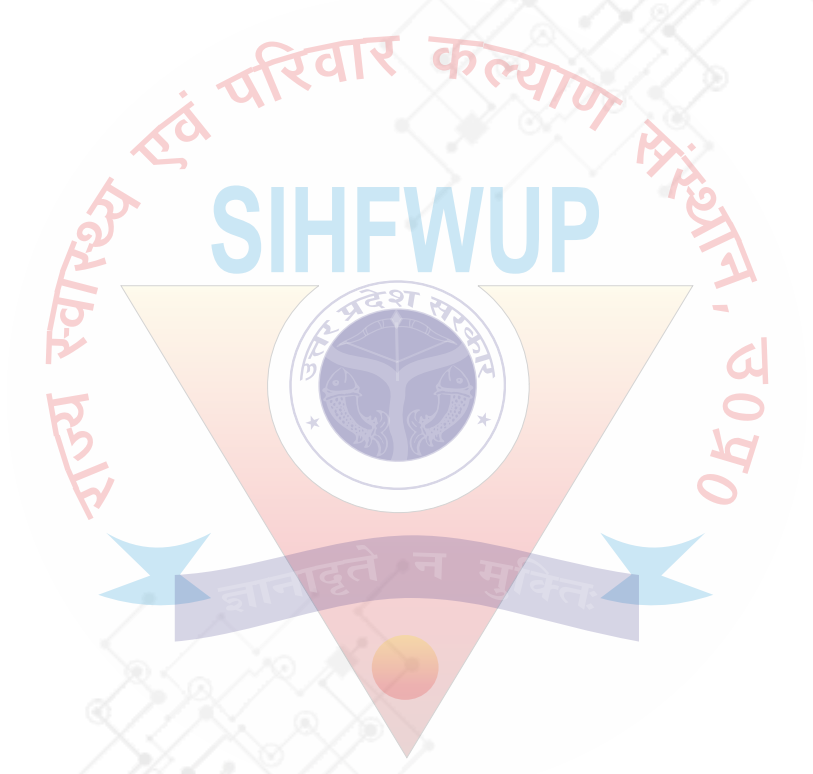
Table of the Content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ESSENTIAL CARDIAC ANATOMY AND PHYSIOLOGY

Dr. Manish Kumar Singh

To understand the 12 lead ECG , It is important to understand the anatomy and physiology of heart. During sinus rhythm, impulse is initiated in the sinoatrial (SA) node of heart and it passes from atria to the ventricles only via the sole pathway the AV node. (except when it is bypassed by abnormal congenital pathways). AV node slows the rate of impulse conduction and gives the atria time to finish emptying of blood and the ventricles time to finish filling with blood.

The common bundle of his next conducts the impulse through the superior ventricular septum and then quickly divides into the right and left bundle branch.

Left ventricle is bigger than the right ventricle, hence the left bundle splits into two hemibundles, one running anteriorly and superiorly out of the page toward the reader, and the other running posteriorly and inferiorly into the page. Finally, the bundle branches divide numerous times into Purkinje fibers, which are the final pathway for conduction of the impulse to ventricular muscle. Once ventricular muscle is stimulated by the impulse traveling down the Purkinje fibers, it depolarizes outwardly from endocardium to epicardium.

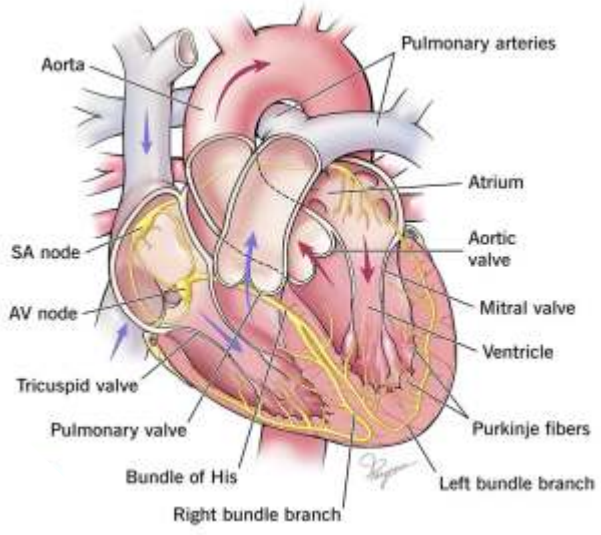


Fig .1.1 Specialised Cardiac Conduction System

The atria pump blood over only very short distances (across the AV valves into the ventricles); therefore, they are very thin-walled structures with very little muscle mass. The right ventricle must pump blood all the way through the lungs, and therefore has a thicker wall and more muscle mass, whereas the left ventricle pumps blood out to the entire body, and thus has the thickest wall of all the chambers, three to four times thicker than the wall of the right ventricle (Figure 1.3).

Size of an electrical complex on the electro

cardiogram is determined by how much voltage is generated by depolarization of a given portion of the heart. More the muscle mass more the voltage generated during depolarisation.

Thus, the QRS complex is normally larger than the P wave because depolarization of the greater muscle mass of the ventricles generates more voltage than does depolarization of the thinner walls of the atria.

A wave of depolarization spreading across a strip of muscle can be recorded by a galvanometer, which is an instrument that measures voltage. Its needle swings up or down as the electrical wave passes through the muscle and the same can be recorded by attaching a pen to the end of the needle and passing a paper strip at a constant speed beneath the pen. The result is called a strip chart recording.

The wave of depolarisation 'axis' travels from the upper right to the lower left of the heart. The needle swings up (initial positive deflection) when an impulse is coming toward a measuring electrode and down when an impulse is going away from an electrode. As the wave passes directly beneath each electrode the needle rapidly swings down toward the neutral position.

As the wave moves away from it, it causes the needle

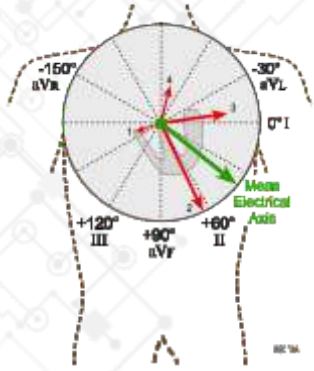
to continue downward past the neutral position to record a negative deflection. When the wave finally reaches the end of the muscle strip and depolarization is over, the needle again swings up to the neutral position and comes to rest on what is called the isoelectric line.

A standard 12-lead ECG records these PQRST complexes in real time from different locations around the heart. Hence, every lead appears slightly different although they all contain a PQRST complex.

Of the 12 leads, six are referred to as 'limb leads'. The limb leads are leads I, II, III (Standard),aVR, aVL and aVF (augmented) and they record the electrical activity in the heart in the vertical plane.

The aVR "looks down" on the heart from above and to the right, at a position of −150 degrees. Lead aVL looks down on the heart from above and to the left, at −30 degrees. Lead I looks at the heart on the horizontal, directly from the left side, at 0 degrees.

Finally, leads II, aVF, and III all look "up" at the heart from below and, together, are called the inferior leads because they look at the inferior wall of the heart from the angles.



The other six are referred to as 'chest' or 'precordial' leads, “look” at the heart in the transverse plane. These leads are V1, V2, V3, V4, V5 and V6 and they record the electrical activity of the heart in the horizontal plane. Thus, different leads can be grouped together when looking for consistency of ECG appearances (normal or abnormal) for different parts of the heart.

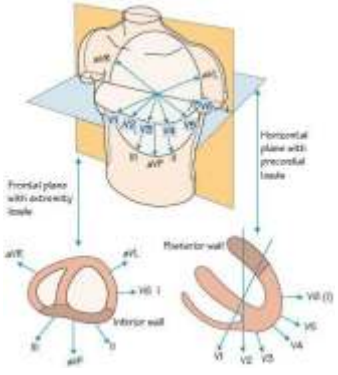
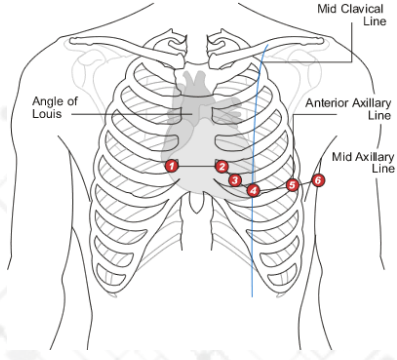


Fig1.3 a) 12 lead ECG



b). Placement of chest leads

In most leads, the P waves and T waves are positive deflections (apart from aVR), and the QRS complexes are predominantly positive (apart from aVR, and sometimes V1, V2 and lead III).

Position of chest leads - V1—right sternal border, 4th interspace V2—left sternal border, 4th interspace V3—midway between V2 and V4 V4—midclavicular line, 5th interspace V5—anterior axillary line, 5th interspace V6—midaxillary line, 5th interspace.

THE ECG GRID

Dr. Manish Kumar Singh

The ECG grid consists of 1-mm squares. Time is measured on the horizontal axis of the grid. Each small box, which measures 1 mm horizontally, equals 0.04 s in time. The width of ECG complexes is commonly referred to as the duration.

The vertical axis is a relative measure of voltage, but is usually expressed in millimeters of positive or negative deflection, rather than in volts. The height or depth of deflection is commonly referred to as the amplitude.

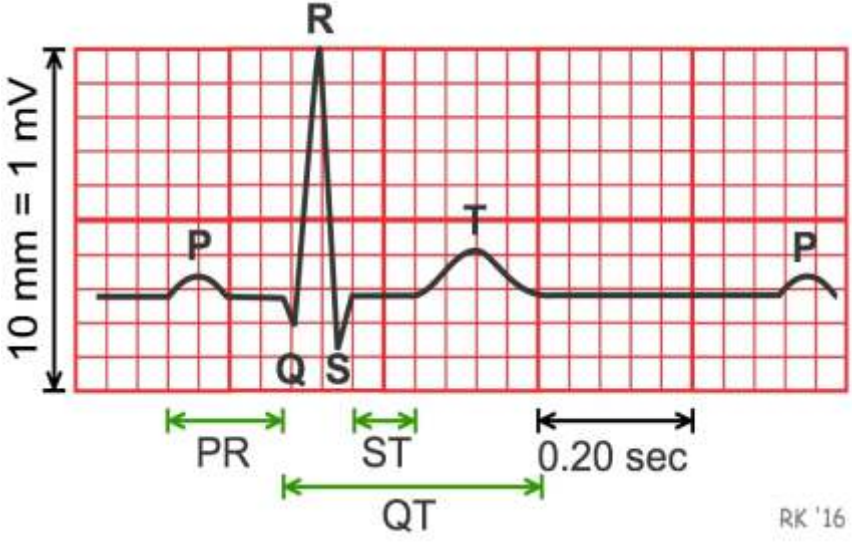


Fig 2.1 – The ECG grid with wave forms and intervals.

The P Wave

The P wave, corresponds to the depolarization of atrial muscle. Because there is relatively little atrial muscle mass, only low voltages are normally produced. The amplitude of the P wave should normally not exceed 2 or 3 mm, and its duration should not be greater than 0.11 s.

Greater amplitude or duration may often indicate enlargement of the atria, with more than the usual amount of muscle mass.

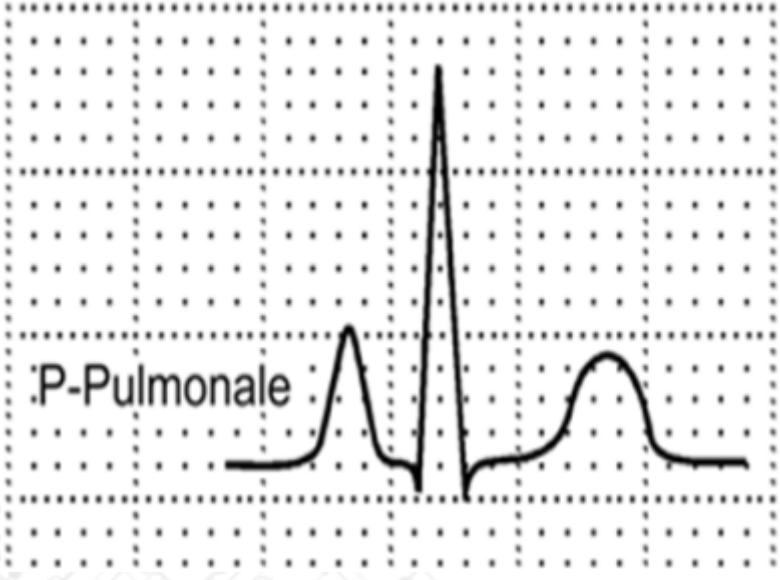


Fig 2.2-P wave abnormalities seen in lead II- P-pulmonale

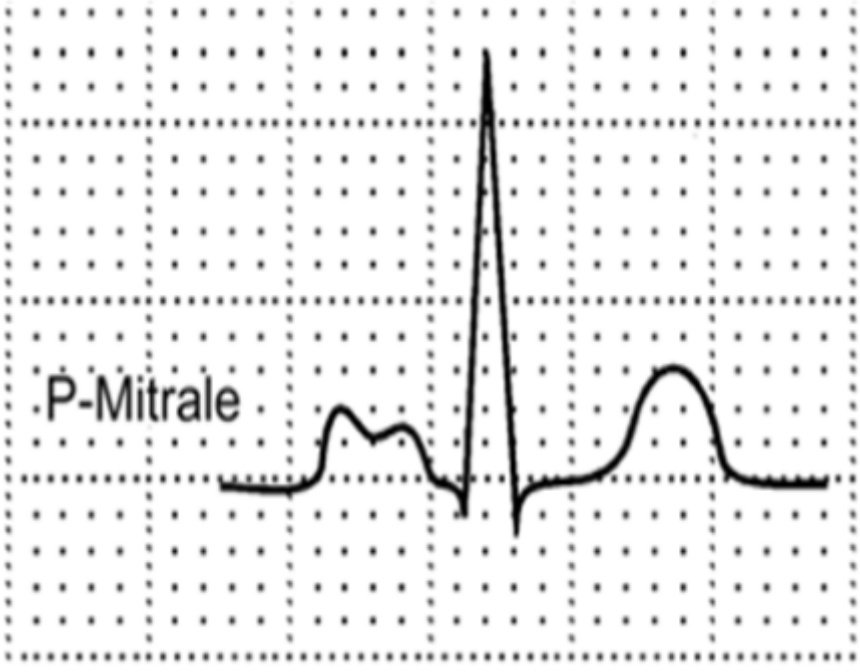


Fig 2.3-P wave abnormalities seen in lead II- P-mitrale

P-pulmonale- The ECG shows tall peaked P waves (amplitude greater than usual 2-3 mm) commonly in patients with right atrial enlargement secondary to pulmonary hypertension.

P-mitrale – The ECG is characterised by broad and notched P waves. It is commonly seen in left atrial enlargement secondary to mitral valve disease.

The PR Interval

The PR interval corresponds to the time it takes an impulse to travel from the SA node all the way down through the conduction system comprising AV node, bundle of His to the first muscle fibres stimulated in the ventricles. It is measured from the beginning of the P wave to the beginning of the QRS. Normal PR interval runs from 0.12 to 0.20s.

PR Interval abnormalities

a) Shorter Interval - It indicates accelerated conduction from the atria to the ventricles.

It is seen in Wolf–Parkinson–White (WPW) syndrome, where there are congenitally aberrant pathways "short circuit" outside of the normal conduction system that bypass the slowing effect of the AV node and rapidly conduct impulses from the atria directly to the ventricles—manifested as the classic delta wave- with early depolarization, or preexcitation.

Fig-2.4 a). Schematic diagram of congenital accessory conduction pathway from atria to ventricles

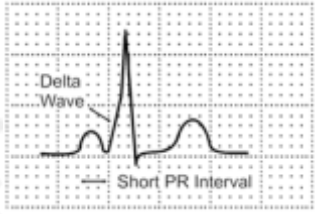


Fig-2.4 b). Delta Wave

b) Longer Interval - PR intervals longer than 0.20s indicate a delay in normal conduction system somewhere between the AV node and the bifurcation of the bundle of His—the familiar first-degree AV block.

The QRS complex

The QRS complex on the ECG grid corresponds to depolarization of the ventricles. Ventricles have a larger muscle mass, hence the amplitude of QRS complex may normally reach as high as 25mm or more (five big boxes) in large individuals, or in those with thin chest walls that actually allow the precordial electrodes to be closer to the heart. If the conduction system is working properly, the duration of the QRS should be <0.10 s.

Understanding the QRS Complex

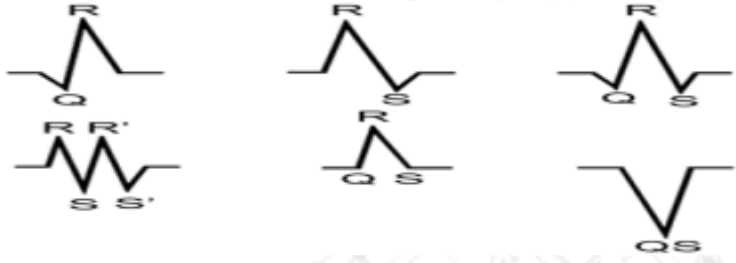


Figure 2.5 Various possible QRS inscription combinations.

The first deflection of the QRS complex is called a Q wave if it is negative and the first positive deflection is called an R wave. The negative deflection coming after an R wave is called an S wave. R′ (R prime) is positive deflections coming after the first R wave and S′ (S prime) is the negative deflections coming after the first S wave. When the complex has no Q or S wave, it is still permissible to call it a "QRS" Complex.

QRS complex abnormalities

a) Amplitudes >25 mm It is associated with ventricular hypertrophy

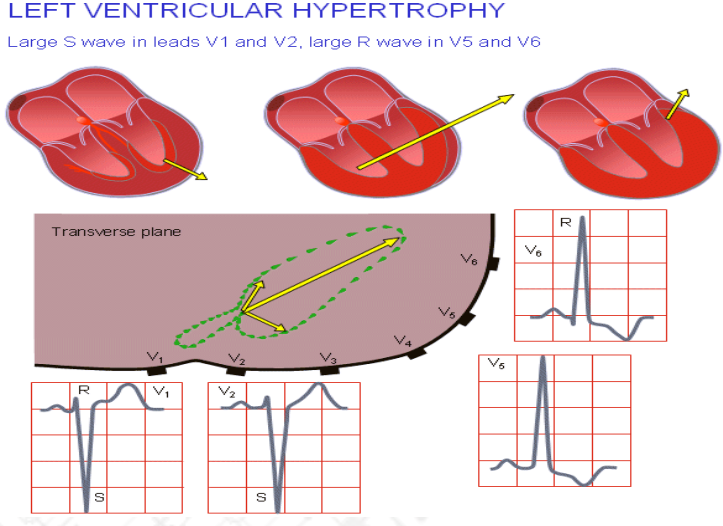


Fig- 2.6 Left ventricular hypertrophy as seen in lead V_6.

b) Very low QRS amplitudes It is seen with diffuse, severe cardiac disease or illnesses such as pericardial effusion and hypothyroidism.

c).Longer duration of QRS complex - Durations of QRS complex of 0.10 s or greater indicate a delay in the spread of depolarization through the ventricles or intraventricular conduction delay. It is seen in Bundle Branch Blocks. There occurs deformity of the ST and T waves, with T waves usually inscribed in the opposite direction from the QRS

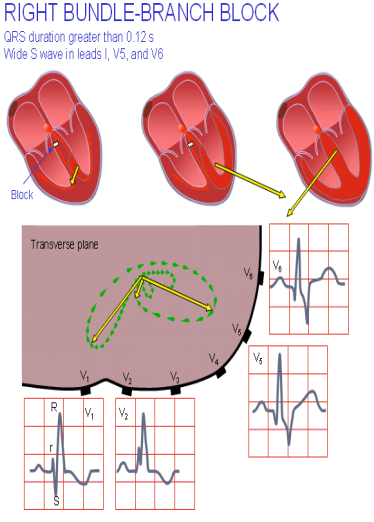


Fig 2.7 a) RBBB.

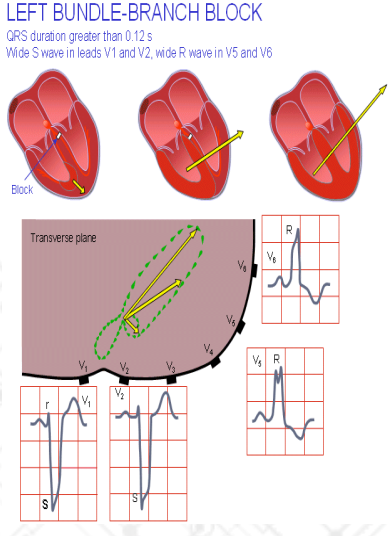


Fig 2.7 b). LBBB

ST Segment

The ST segment represents the time between the completion of depolarization of the ventricles and the onset of repolarization of the ventricles. It is normally isoelectric (neither positive nor negative) and gently blends into the upslope of the subsequent T wave. The point at which the ST segment takes off from the QRS is called the J point.

Note :-The ST segment plays a very important role in the diagnosis of ischemic heart disease. ST segment elevation is one of the hallmarks of Acute Myocardial Infarction. ST segment can also be depressed below the base line in conditions, such as ischemia and ventricular hypertrophy.

The T Wave

The T wave corresponds to repolarization of the ventricles. It is normally inscribed in the same direction as the predominant deflection of the QRS, and has less amplitude than the QRS.

- Abnormalities of the T wave
- Inversion (in the opposite direction of the QRS) seen in BBB, left ventricular hypertrophy (LVH) and AMI.

- Tall (large amplitude) such as in hyperkalemia (Fig 2.8)
- Small (low amplitude) seen in hypokalemia

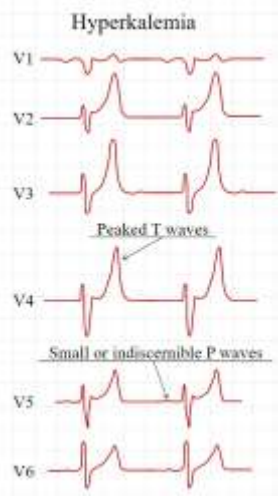


Figure 2.8. Extremely tall, pointed T wave

The QT Interval

Itis measured from the beginning of the QRS to the end of the T wave, and normal intervals vary with heart rate and the person's sex.

Abnormality -

QT interval is prolongation reflects delays in ventricular repolarization. This is commonly the result of the administration of drugs, such as procainamide or quinidine, or of electrolyte imbalance, particularly as in hypocalcemia. When the QT interval is prolonged, there is a higher incidence of ventricular reentry dysrhythmias and sudden death.

IDENTIFYING TACHYARRHYTHMIA

Dr. Shruti Singh

An arrhythmia is an abnormal heart rhythm. Tachyarrhythmia or tachycardia means a fast heart rate. The cut-off may vary with age and various physical conditions.

In adults, Tachyarrhythmia or tachycardia is referred to as a heart rate of more than 100 beats per minute. Rapid heart rate is often an appropriate response to physiologic stress such as fever, dehydration, or anaemia, such factors should be identified early to determine whether the rate is a Primary dysrhythmia or Secondary to an underlying condition and treatment should be aimed at the underlying causes if present.

Classification of Tachyarrhythmias:

1. Based on site of origin of depolarization: When heart is in sinus rhythm, the depolarisation originates from SA node. If the cardiac depolarization begins from somewhere other than SA node, the rhythm is named after the part of the heart where the sequence originates.

a. Supraventricular tachyarrhythmias: Sinus tachycardia, atrial tachycardia, paroxysmal supraventricular tachycardia (AV nodal re-entrant

tachycardia and AV bypass pathway with Wolf-Parkinson-White syndrome), junctional tachycardia.

b. Ventricular tachyarrhythmias

2. Based on the duration of the QRS complex:

Narrow Complex:

- Those with a QRS duration of lesser than 120 milliseconds.
- Mostly all SVTs have narrow QRS complexes. (Except in the settings of pre-existing bundle branch block, AVRT with antidromic conduction, rate dependent bundle branch blocks and SVT with aberrancy)
- The depolarization wave spreads to the ventricles in the normal way via the His bundle and its branches. Hence the QRS complex is normal.

Wide Complex:

- The duration of the QRS complex is greater than 120 milliseconds.
- All ventricular tachyarrhythmias and supraventricular rhythm with an abnormal conduction path have broad QRS complexes.
- The depolarization wave spreads through the ventricles by an abnormal and slower pathway, via

the Purkinje fibres.

- The QRS complex is therefore wide and is abnormally shaped. Repolarization is also abnormal, so the T wave is also of abnormal shape.

Conceptual approach to tachycardias:

Narrow Complex	
Regular	Irregular
1. Sinus tachycardia	1. Atrial Fibrillation
2. Atrial Flutter	2.Atrial tachycardia-variable conduction
3. Atrial tachycardia	3. Atrial Flutter-variable conduction
4. AV nodal Reentrant tachycardia	4. Multifocal Atrial Tachycardia
5. AV Reentrant tachycardia	
6. Junctional tachycardia	

Broad Complex	
Regular	Irregular
1. Ventricular tachycardia	1. Above rhythms with either a. Aberrancy b. Accessory pathway
2. Supraventricular tachycardia with aberrancy	2. Polymorphic ventricular tachycardia

ECG Changes:

Supraventricular Tachycardia:

1. Atrial Tachycardia:

- HR faster than 150 bpm.
- p-waves are superimposed on the t waves of preceding beats.
- QRS complexes are like those of sinus beats.

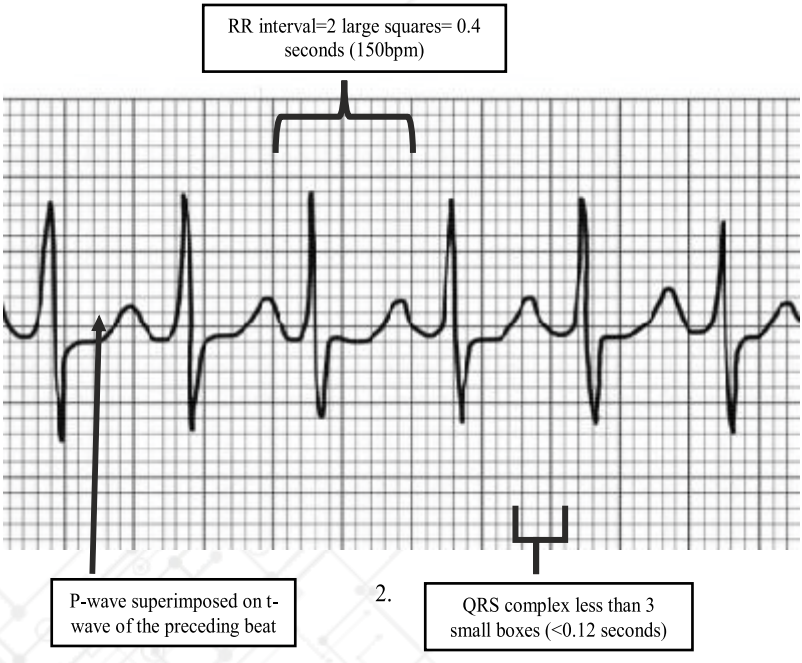


Fig.3.1 - ECG changes in Atrial Tachycardia

2. Atrial Flutter:

- Atrial rate 250-300 bpm. (two P waves are about 6 small squares apart): giving a 'sawtooth' appearance
- Ventricular rate is a fraction of the atrial rate (The AV node cannot conduct atrial rates of discharge greater than about 200 bpm. If the atrial rate is faster than this, an 'atrioventricular block' occurs, with some P waves not followed by QRS complexes.)
- Ventricular rate is in multiple of atrial rate. For example, if the atrial rate is 300 bpm, the Ventricular rate will be as follow
- 2:1 block = 150 bpm (a QRS complex is preceded by 2 p-waves)
- 3:1 block = 100 bpm
- 4:1 block = 75 bpm
- RR interval=12 small squares= 0.48 seconds (125bpm)
- QRS complexes are like those of sinus beats.

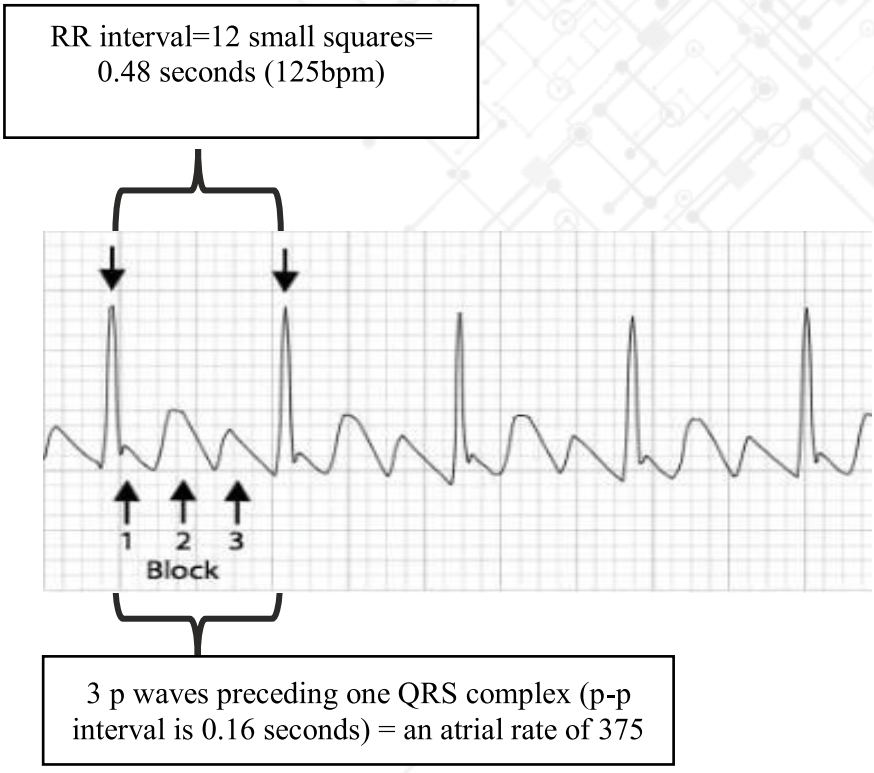


Fig 3.2 -ECG depicting Atrial flutter with 3:1 AV block

1. Junctional Tachycardia: is further classified as:
    - Accelerated Junctional Rhythm
    - AVRT (Atrioventricular Re-entrant Tachycardia)
    - AVNRT (Atrioventricular Nodal Re-entrant Tachycardia)

I. Accelerated Junctional Rhythm: - It occurs when the rate of an AV junctional pacemaker exceeds that of the sinus node. This situation arises when there is increased automaticity in the AV node coupled with decreased automaticity in the sinus node.

ECG Features:

- Narrow complex rhythm; QRS duration < 120ms
- The ventricular rate is usually 60 – 100 bpm.
- Retrograde P waves may be present and can appear before, during or after the QRS complex. They are usually inverted in inferior leads (II, III, aVF), upright in aVR + V1
- AV dissociation may be present with the ventricular rate usually greater than the atrial rate.

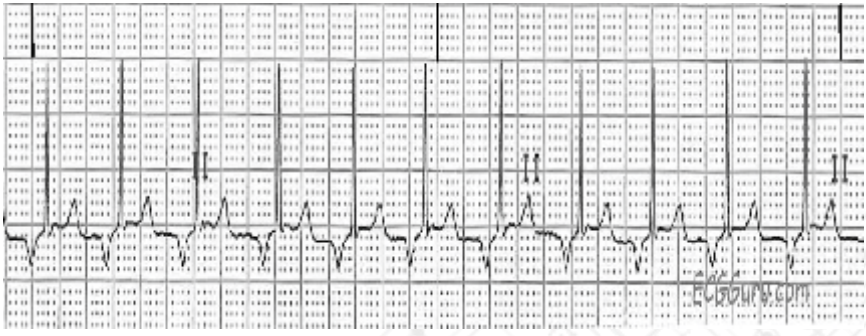


Fig 3.3 -Rhythm strip in AJR

II. AVNRT: It is popularly known as Paroxysmal SVT (PSVT) due to its sudden onset and is the most common cause of palpitations in the otherwise normal heart. Tachycardia is in the range of 140-280 bpm.

ECG Features:

- Regular tachycardia ~140-280 bpm
- Narrow QRS complexes (< 120ms)
- P waves is absent, if visible exhibit retrograde conduction with P-wave inversion in leads II, III, and aVF (pseudo S in inferior leads and pseudo R in V1).

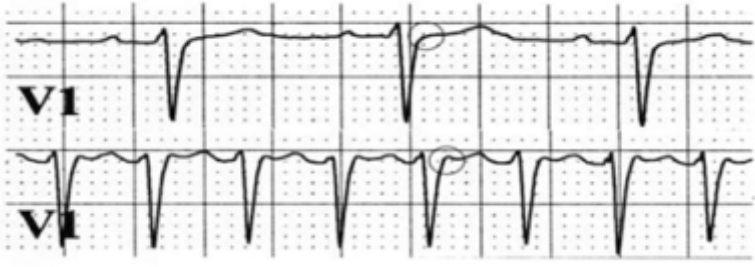


Fig. 3.4 Top strip: Normal sinus rhythm. Absence of pseudo-R waves & Bottom strip**:** Paroxysmal SVT. The P wave is seen as a pseudo-R wave (circled) in lead V1 during tachycardia.

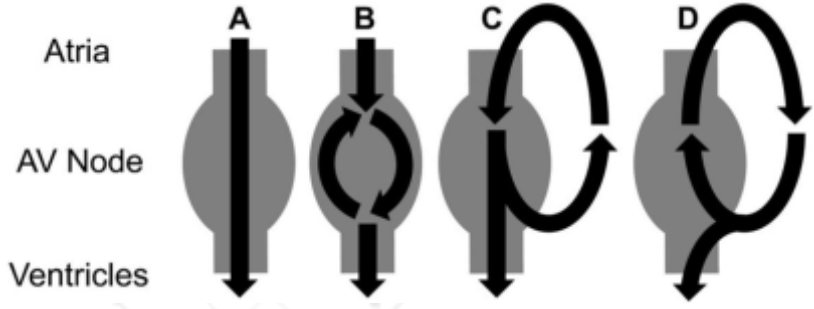


Fig. 3.5 Conduction through the atrioverntricular (AV) nnode in sinus rhythm and paroxysmal supraventricular tachycardia. (A) Sinus rhythm: (B) atrioventricular nodal re-entry tachcardia. (C) orthodramic atrioventricular re-entry tachycardia: (D) antidromic atrioventricular re-entry tachycardia.

III. AVRT:

Is a form of paroxysmal supraventricular tachycardia that occurs in patients with accessory pathways, usually due to the formation of a re-entry circuit between the AV node and accessory pathway. ECG features depend on the direction of conduction, which can be orthodromic or antidromic.

In orthodromic AVRT (more common), anterograde

conduction is via the AV node, producing a regular narrow complex rhythm (in the absence of pre-existing bundle branch block).

In antidromic AVRT (less common), anterograde conduction is via the accessory pathway (AP), producing a regular wide complex rhythm. It is rare and make up only 5% of tachyarrhythmias in patients with WPW. This can be difficult to distinguish from ventricular tachycardia (VT)

ECG Features:

Orthodromic AVRT: Appear very similar to AVNRT, but In AVRT, retrograde P waves occur later, with a long RP interval > 70 msec. Fortunately, treatment too is fairly similar for both.

Antidromic AVRT:

- Rate usually 200-300 bpm
- Wide QRS complexes due to abnormal ventricular depolarisation via AP
- When in doubt, diagnosis of VT should be presumed and treated accordingly.

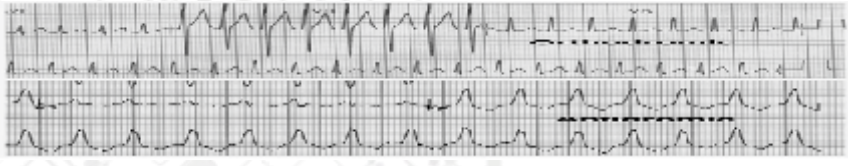


Fig. 3.6 Ventricular Tachycardias:

1. Monomorphic VT:

- Regular, broad complex tachycardia
- Uniform QRS complexes within each lead — each QRS is identical

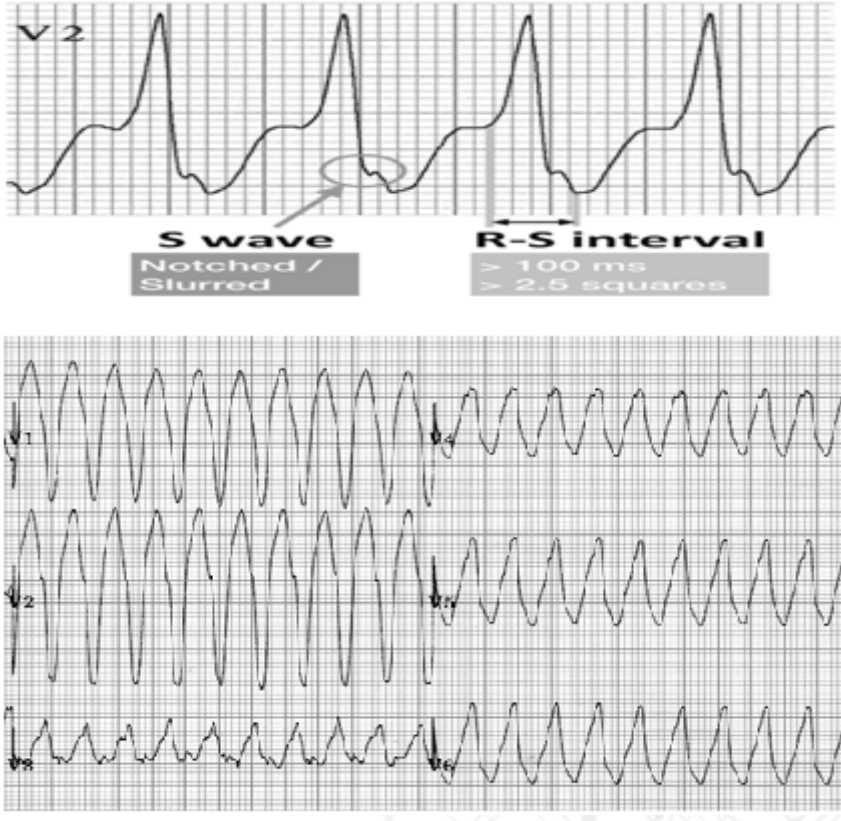


Fig 3.6 -Monomorphic VT

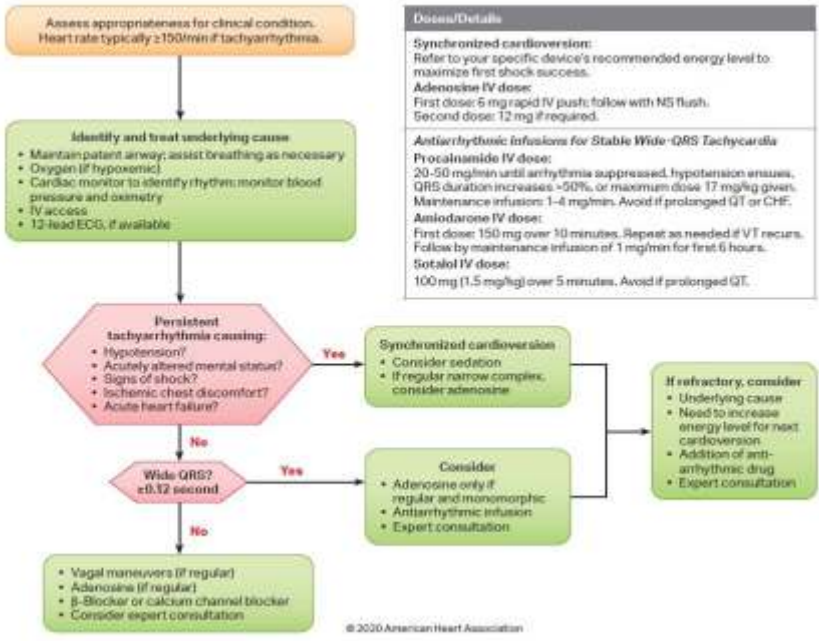
How to differentiate monomorphic VT from other causes of broad complex tachycardia?

ECG features suggestive of VT include:

- Very broad complexes (>160ms)
- Absence of typical RBBB or LBBB morphology
- Extreme axis deviation ("northwest axis")
- AV dissociation (P and QRS complexes at different rates and independently)
- Capture beats — occur when the sinoatrial node transiently 'captures' the ventricles, in the midst of AV dissociation, to produce a QRS complex of normal duration
- Fusion beats — occur when a sinus and ventricular beat coincide to produce a hybrid complex of intermediate morphology
- Positive or negative concordance throughout the chest leads, i.e. leads V1-6 show entirely positive (R) or entirely negative (QS) complexes, with no RS complexes seen
- Josephson's sign – Notching near the nadir of the S-wave
- Brugada's sign – The distance from the onset of the QRS complex to the nadir of the S-wave is> 100ms

- RSR' complexes with a taller “left rabbit ear”. This is the most specific finding in favour of VT. This is in contrast to RBBB, where the right rabbit ear is taller
- Any broad QRS tachycardia in a patient with coronary artery disease with ACS, Severe LV systolic dysfunction (LVEF< 30%), QRS morphology different from baseline, should be presumed to be a VT, unless proven otherwise.

Adult Tachycardia With a Pulse Algorithm



BRADYARRHYTHMIA

Dr. Huda Siddiqui

Bradyarrhythmia is defined as heart rate < 60/min. It is caused by dysfunction in conduction at the level of the sinus node, atrioventricular (AV) node, or HIS/Purkinje system.

Innervation:

- Parasympathetic: reduces sinus node automaticity (↓ heart rate)
- Sympathetic: increases sinus node automaticity (↑ heart rate)

Extrinsic causes:

- Medications:Most common extrinsic cause - Beta blockers, calcium channel blockers (CCBs), digoxin, anti-arrhythmics, Acetylcholinesterase inhibitors (AchEIs), Lithium, Sympatholytics, Ivabradine
- Hypothyroidism
- Hyperkalemia, hypokalemia
- Hypothermia
- Neonatal lupus: Congenital heart block due to transplacental transfer of antibodies (anti-Ro, anti-La)
- Surgery (transcatheter aortic valve implantation,

radiofrequency ablation, trans coronary ablation of septal hypertrophy)

- Obstructive sleep apnea
- Central Nervous System (CNS) conditions (intracranial hypertension)
- Increased vagal activity (e.g., carotid sinus stimulation, vomiting, coughing, Valsalva maneuver)

Bradyarrhythmia Due to Sinus Node Dysfunction

Sinus (Sino Atrial) Node Dysfunction (SND)

- Also called sick sinus syndrome
- Non-physiologic sinus rate of < 50/min and/or sinus pauses > 3 sec
- ECG changes and clinical symptoms are both present.
- ECG findings alone do not indicate SND (e.g., highly conditioned athletes).

Pathophysiology

- Defect in cardiac impulse formation (automaticity) and/or conduction from the SA node

- Often secondary to senescence of SA node (sinus node fibrosis)
- SA node is unable to generate a heart rate sufficient for the physiologic needs of the individual.

Types

Sinus bradycardia: Sinus rhythm with a heart rate < 60/min

SA node sends out electrical impulse at a regular interval.

ECG:

- P wave is followed by a QRS complex.
- Normal PR interval

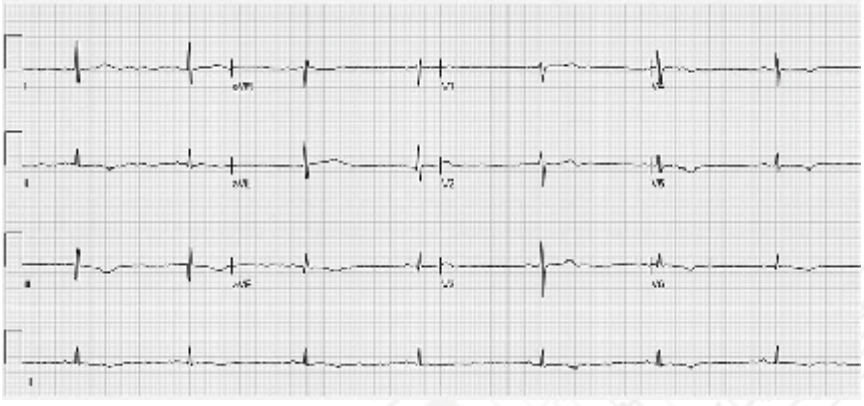


Fig 4.1 -Sinus bradycardia, normal PR interved

Sinus arrest or pause:

- Disorder of automaticity
- Sinus node fails to generate an impulse.

ECG:

- Missing P wave, QRS complex, and T wave → pause generated
- Length of pause has no relation to length of sinus cycle length.
- AV node and lower conduction system become the pacemaker and an escape rhythm is produced.

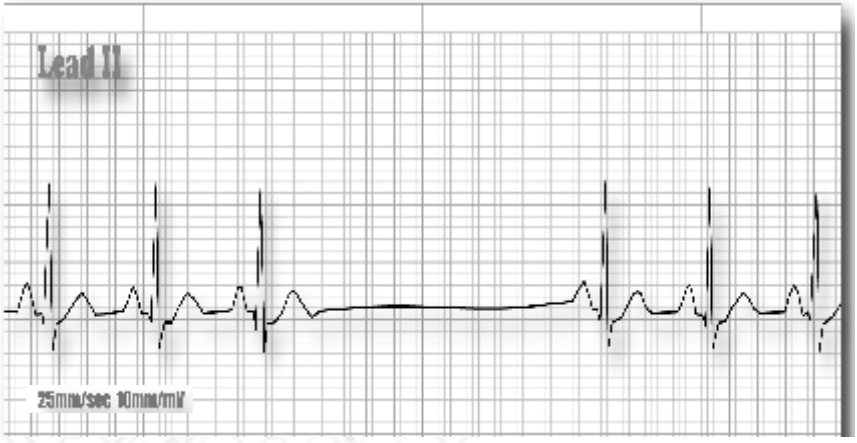


Fig 4.2 -Sinus Arrest

Sinoatrial exit block (SA block):

- Disorder of conduction SA node depolarizes, but there is a failure in the signal transmission to the rest of the atria.

ECG:

- Absence of P wave on the ECG
- Distinguishing feature from sinus arrest: Length of the SA block is a multiple of the P-P interval before the pause.

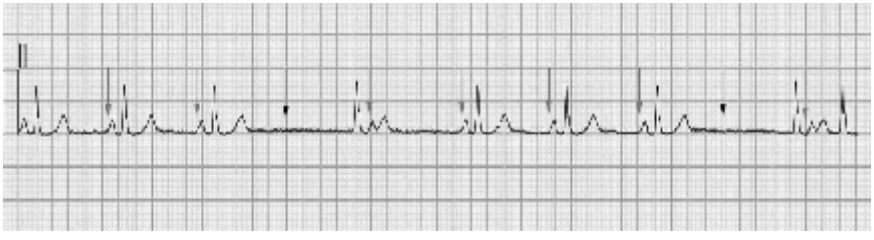


Fig 4.3 -Sinoatrial block

Note : Arrows inducate presumed timing of each sinus impulse, Blue arrows represent normally transmitted impulses resulting in P waves & Black arrows represents blocked sinus impulses (dropped P waves).

The pauses around dropped P waves (2.1 seconds) are exactly the double of the proceeding P-P interval (1.05 seconds)

Bradyarrhythmia Due to Atrioventricular Block

Atrioventricular block

- Conduction defect (from atrium to ventricle)
- Can be transient or permanent
- Physiologic AV block: from enhanced vagal tone (e.g.,athletes, sleep, carotid sinus massage, carotid sinus hypersensitivity syndrome)

Pathophysiology

Delay or interruption in the electrical conduction between the atria and the ventricles due to either anatomical or functional impairment

Types

1st-degree AV block:

- Delayed conduction from atrium to ventricle, without interruption
- Regular QRS complexes
- Can be physiologic
- Often asymptomatic and diagnosed by ECG

ECG:

- PR interval > 0.2 sec or > 200 msec (normal: 0.12–0.20 sec)

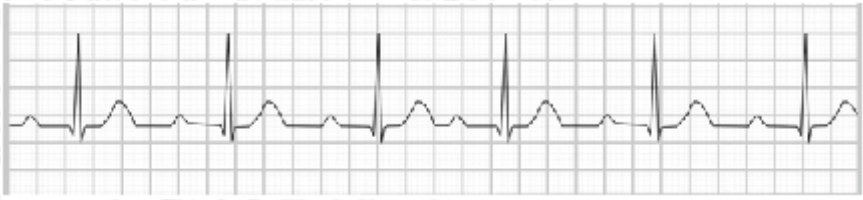


Fig 4.4 -1st degree AV block

2nd-degree AV block:

- Incomplete AV block: There is intermittent atria-to-ventricle conduction.

Mobitz type 1 (Wenckebach):

- Some atrial impulses do not reach the ventricles.
- Impairment usually within the AV node
- Can be physiologic in young and highly conditioned athletes
- Often asymptomatic

ECG:

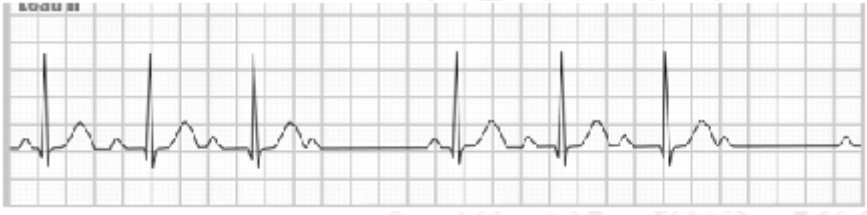


Fig 4.5 -Mobitz Type 1

PR interval progressively and predictably lengthens.
Non-conducted P wave → "dropped" QRS complex

Mobitz type 2:

- Impairment distal to the AV node (His bundle and bundle branches)
- Higher risk to progress to 3rd-degree AV block
- Can be associated with hemodynamic instability

ECG:

- Consistent unchanging PR intervals
- Abrupt failure of P wave conduction: "dropped" QRS complex
- QRS complex is "dropped" unpredictably without PR interval lengthening.

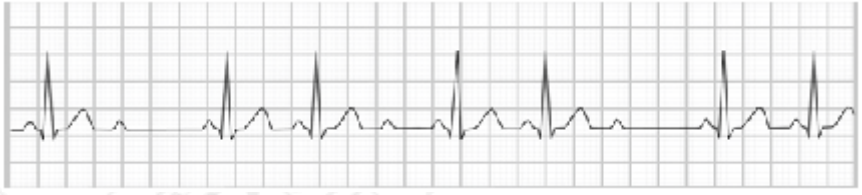


Fig 4.6 - Mobitz Type 2

2nd-degree AV block, high grade: Also called as 2:1 Block

2 or more consecutively blocked P waves

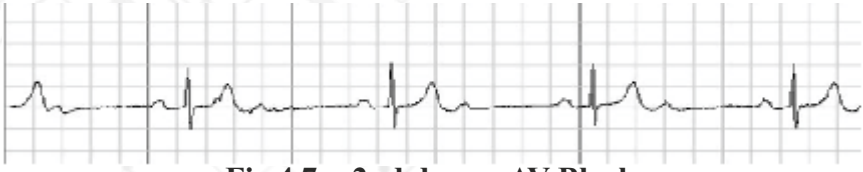


Fig 4.7 - 2nd degree AV Block

3rd-degree (complete) AV block:

- Complete disconnection of electrical pathways between the atria and the ventricular system
- May be associated with hemodynamic instability

ECG:

- No relationship between the P waves and QRS complexes
- Variable PR interval
- QRS, P-P, and R-R intervals are constant.

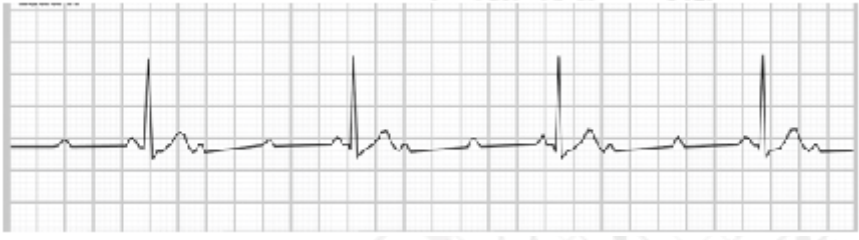


Fig 4.8 - 3rd degree AV Block

Dr. Chhaya Singh

Acute myocardial infarction **(AMI)**occurs any time when a coronary artery becomes essentially completely obstructed and the segment of myocardium served by that artery loses perfusion and begins to die.

Complete obstruction usually occurs in the setting of fixed obstructive coronary lesions that are the result of coronary atherosclerosis.. The acute nature of AMI is usually the result of a clot or thrombus forming in the immediate vicinity of an incomplete fixed obstructive lesion.

Electrocardiographic Categories of AMI

There are two electrocardiographic categories of AMI. The treatment for the two categories is different.

- ST-segment elevation myocardial infarction (STEMI)
- Non-STEMI (NSTEMI).

ECG hallmarks of a classic STEMI: Following three changes in ECG appear sequentially over a period of minutes to hours. Subsequently, the changes may show slow resolution, usually over a period ranging from days to

months. Q waves, however, may persist indefinitely, producing ECG evidence of a scar

- ST-segment elevation
- T wave inversion.
- Q wave formation

There are over 100 identified causes of ST segment and T wave changes, so the diagnosis of ischemia and infarction frequently requires comparison with previous ECGs and correlation with the clinical presentation and laboratory data.

Myocardial ischemia produces a range of changes in the ST segment and T wave, depending on the severity of ischemia and the timing of the ECG.

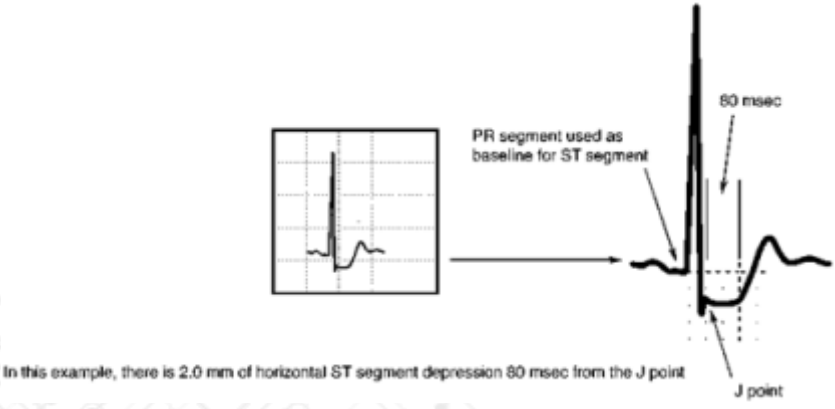


Fig 5.1 - Morphology of ST segment in Ischemia

In exercise stress testing, 1mmormore of horizontal or down-sloping ST segment depression 80 msec from the J point is considered an ischemic response.

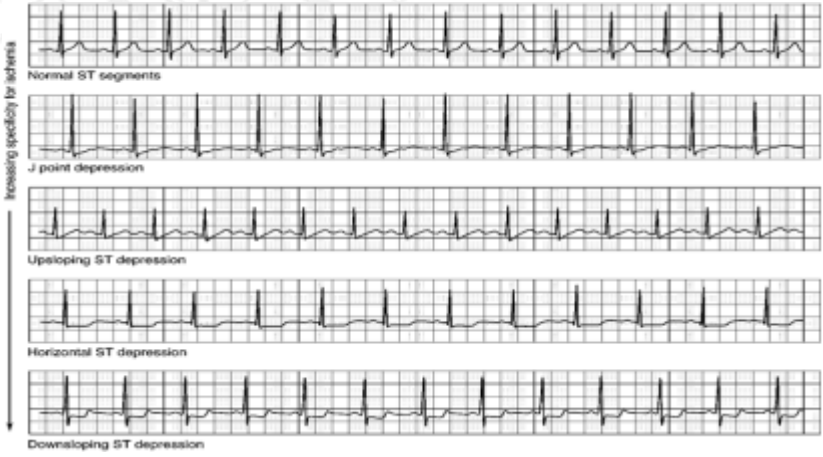


Fig 5.2 - Morphology of ST segment in Ischemia

II. Myocardial infarction(MI)

A. ECG patterns in infarction

1. Azone ofischemia typically produces ST segment depression.
2. Azone ofinjury produces ST segment elevation.
3. Azone ofinfarction produce salarge Q wave in the QRS complex.

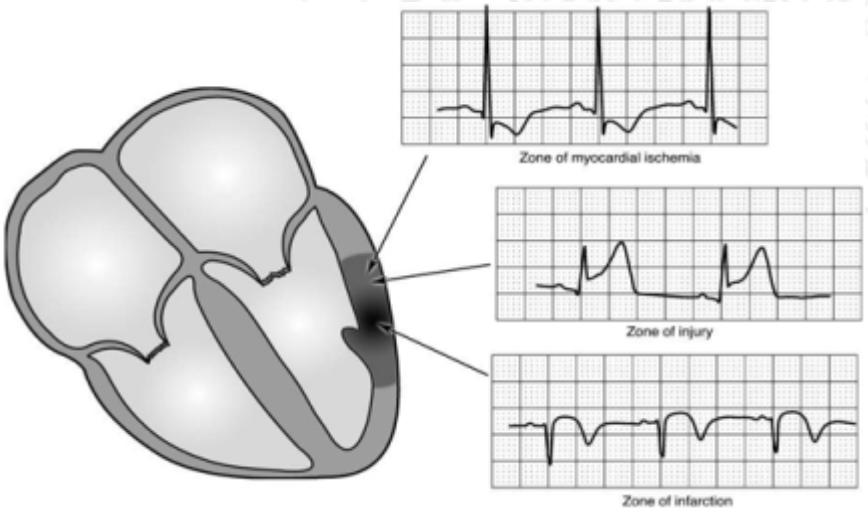


Fig 5.3 - ECG Patterns in infarction

B. Genesis of the Q wave in infarction

The normal situation

a. For example, in Lead I, the QRS complex begins with a small Q wave because left ventricular depolarization begins in the septum and the electrical forces are directed away from Lead I.

b. The small Q wave is rapidly succeeded by forces directed inferiorly and laterally, resulting in a large R wave in Lead I.

The infarct situation

a. If there is a lateral myocardial infarction, however, the electricalvectors in the lateral direction are lost; the forces directed medially are unbalanced.

b. A large Q wave results in Lead I.

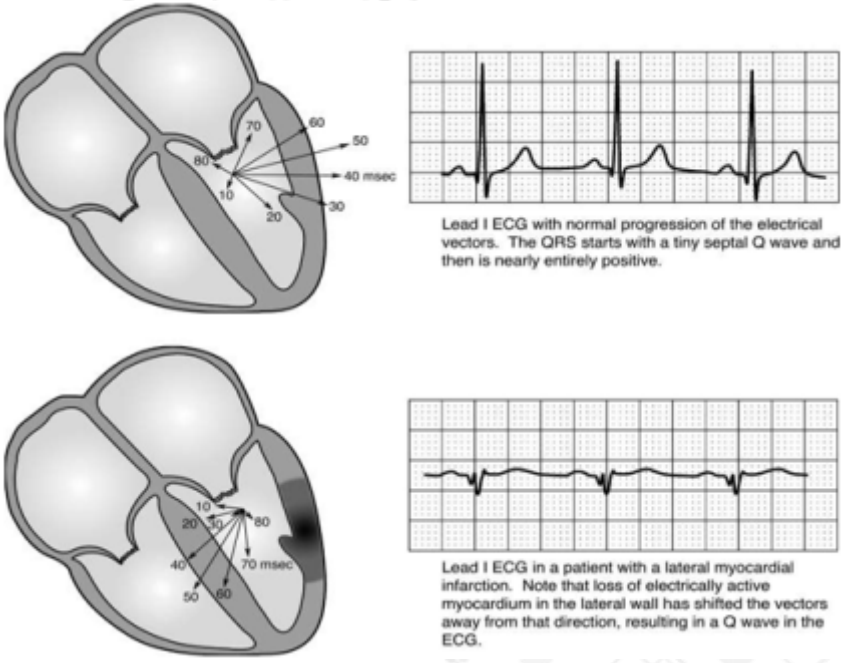


Fig 5.4 - Genesin of Q wave in infarction

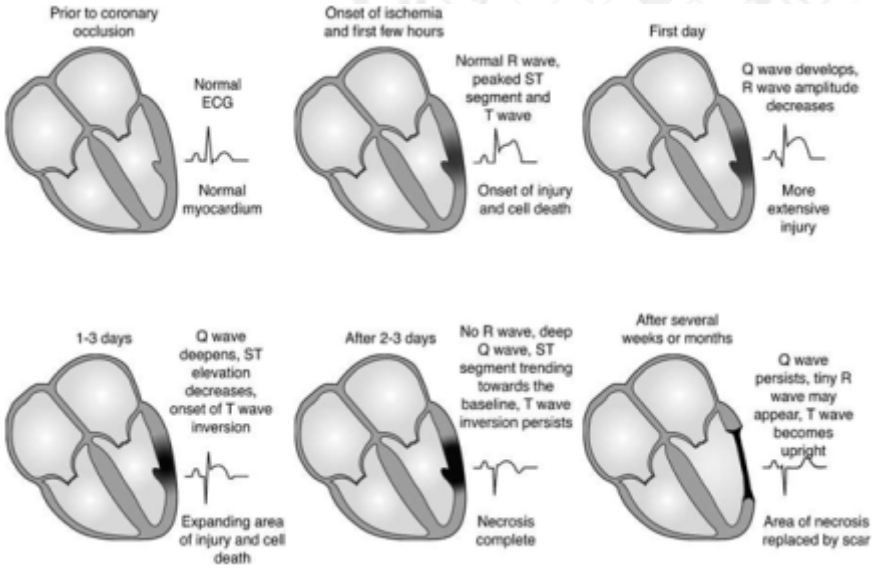


Fig 5.5 - The time course of myocardial and ECG changes during infarction

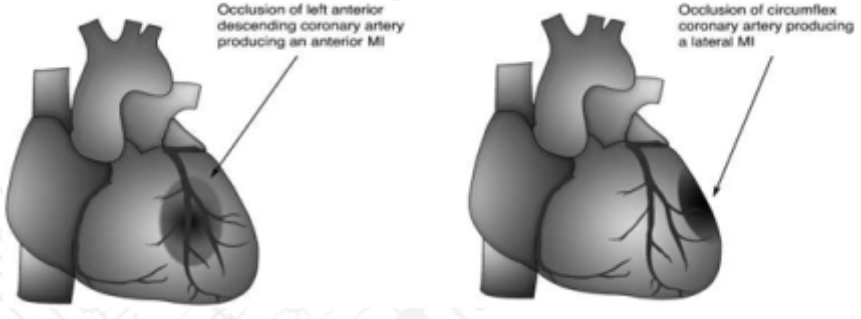
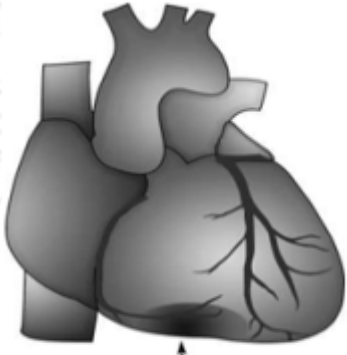
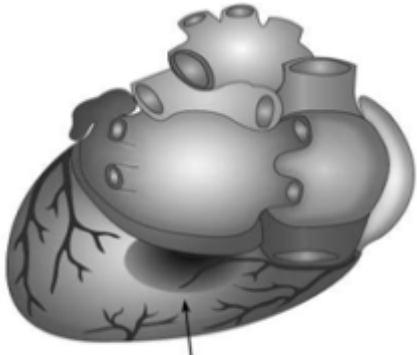


Fig 5.6 - Anatomical & ECG infration of MI

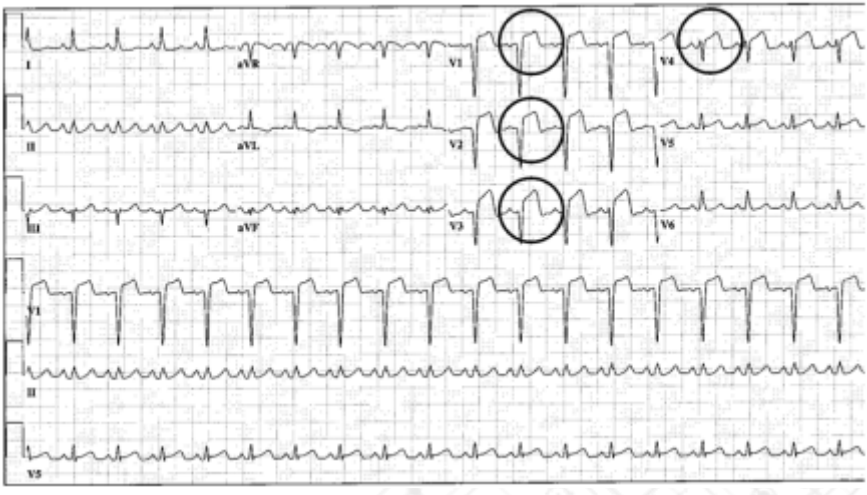


Occlusion of the right coronary artery producing an inferior MI

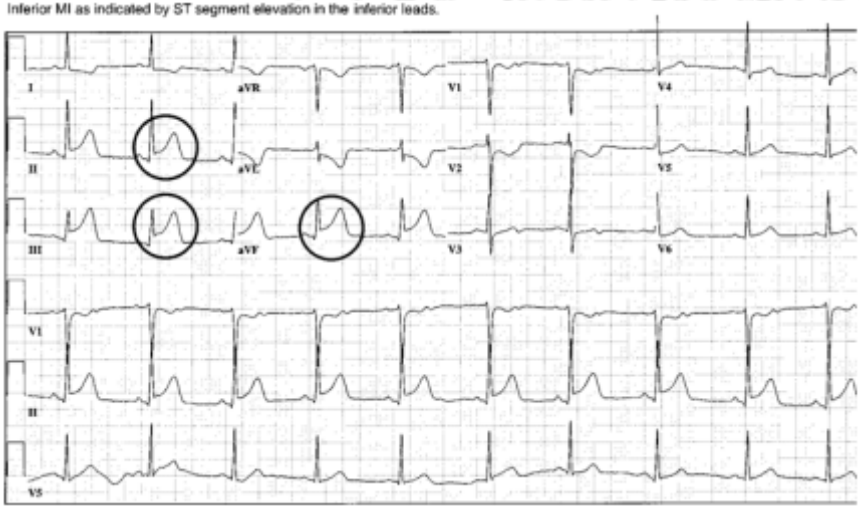


Occlusion of the distal right coronary artery producing a posterior MI

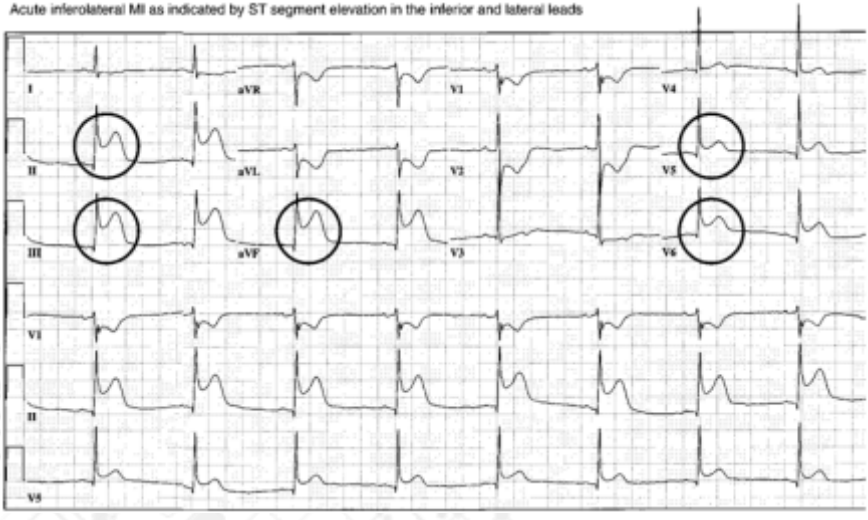
Acute anterior MI as indicated by ST segment elevation in the anterior leads



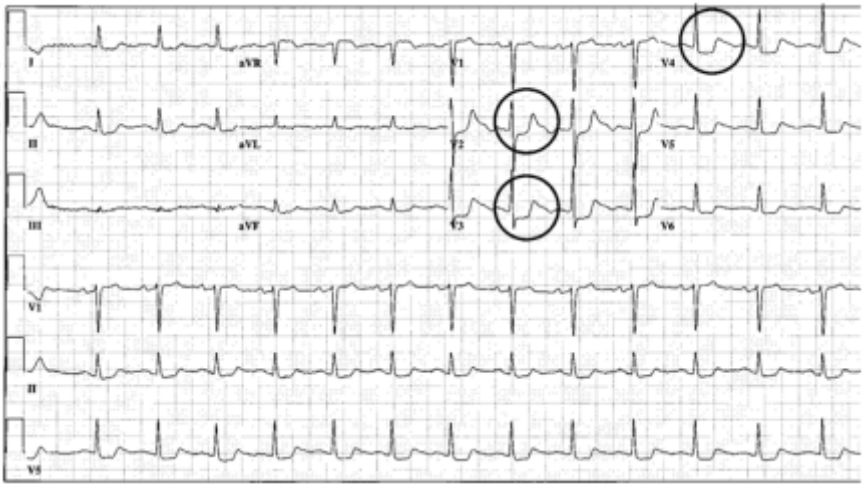
Inferior MI as indicated by ST segment elevation in the inferior leads.



Acute inferolateral MI as indicated by ST segment elevation in the inferior and lateral leads



Posterior MI as indicated by ST segment depression and tall R waves in the anterior leads

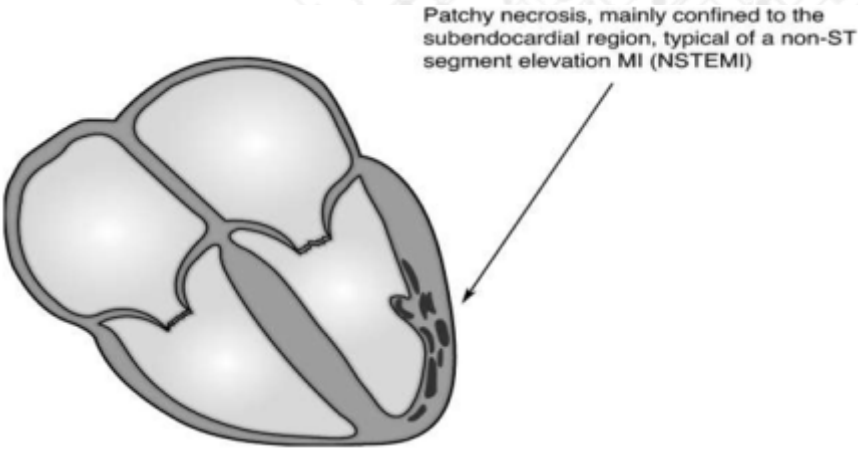


V. Non-ST-segmentelevation MI (NSTEMI) (08)

A. About half of the 750,000 MIs that occur annually in the U.S. do not develop new Q waves.

B. Usually there are ST segment and T wave changes, but about 20% of NSTEMIs have no obvious ECG abnormalities and the diagnosis of MI is base dontheclinical presentation and elevated cardiac markers.

C. Anatomically, NSTEMIs are frequently associated with patchy sub endocardialne crosis.



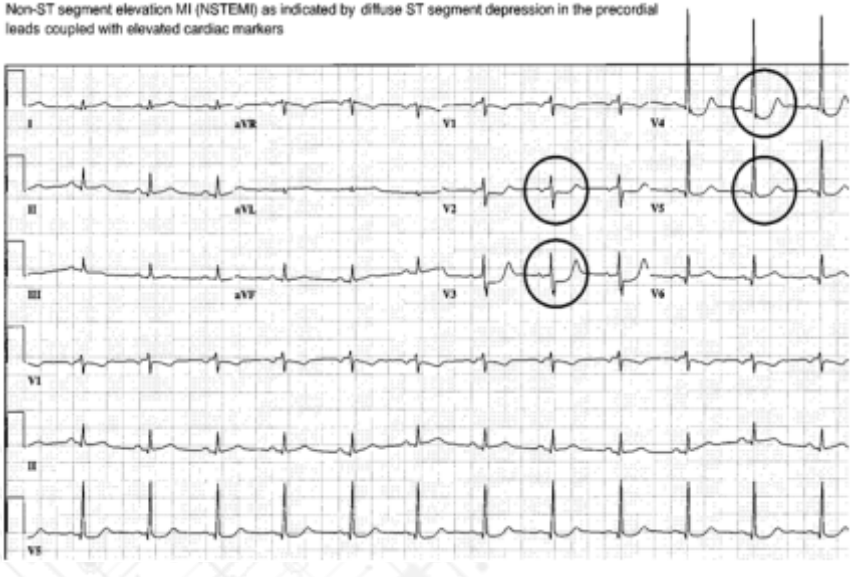


Fig. 5.7 - Non ST segment clevation MI (STEMI)

Leads with ST segment elevation	Affected myocardial area	Occluded coronary artery (Culprit)
V1-V2	Septal	Proximal LAD
V3, V4	Anterior	LAD
V5, V6	Apical	Distal LAD
I, aVL	Lateral	LCx
II, aVL, III	Inferior	90% RCA, 10% LCx
V7, V8, V9 (reciprocal ST depressions are frequently evident in V1 to V3)	Posterolateral (also referred to as inferobasal or posterior)	RCA or LCx

Table: ECG localization of STEMI (ST segment elevation is considered significant if it is ≥ 2.5 mm in males <40 years, ≥ 2 mm in males >40 years and ≥ 1.5 mm in females; in leads v2-v3. For other ECG leads, any ST elevation in 2 or more contiguous chest leads or limb leads of ≥ 1mm is considered significant)

STE DDx	EKG Findings
Acute MI	Upward convexity STE +/- TWI or prior MI with persistent STE
Coronary spasm (Prinzmetal's angina)	Transient STE in a coronary distribution
Pericarditis	Diffuse, upward concavity STE; PR depression, upright Tw
Pulmonary embolism	Occasionally STE in V_1-V_3, TWI (V_1-V_4), RAD, RBBB, $S_1Q_3T_3$
Left main disease	STE in aVR > V_1
Increased mortality	STE >1 mm in aVR
Repolarization abnormalities:	
- LBBB	QRS ≥120 ms, STE discordant from QRS
- LVH	Increased QRS amplitude
- Brugada syndrome	rSR', downsloping STE in V_1-V_2, Na channelopathy a/w SCD
- Hypothermia	Osborn (J) waves (positive deflection at J point, often right precordial leads; proportional to degree of hypothermia)
- Hyperkalemia	STE often V_1-V_2, tented Tw, shortened QT, small Pw, increased PR, AV block, wide QRS, sinusoidal pattern
Early repolarization	Often V_2-V_5 in young adults; 1-4 mm elevation of peak notch or start of slurred downstroke of Rw (i.e., J point) ± up concavity of ST, large Tw; ratio of STE:Tw <25%; may disappear w/ exercise; possible increased VF risk in inferior leads
HCM, CM, Takotsubo, ventricular aneurysm, cardiac contusion	
STD DDx	**EKG Findings**
Myocardial ischemia	STD ± Tw abnormality
Acute true posterior MI	STD in V_1-V_3
Digitalis effect	Downsloping STD,± Tw abnormality; not correlated with digoxin levels
Hypokalemia	STD ± Uw
Repolarization abnormalities:	
- LBBB or LVH	STD often in I, aVL, V_5, V_6

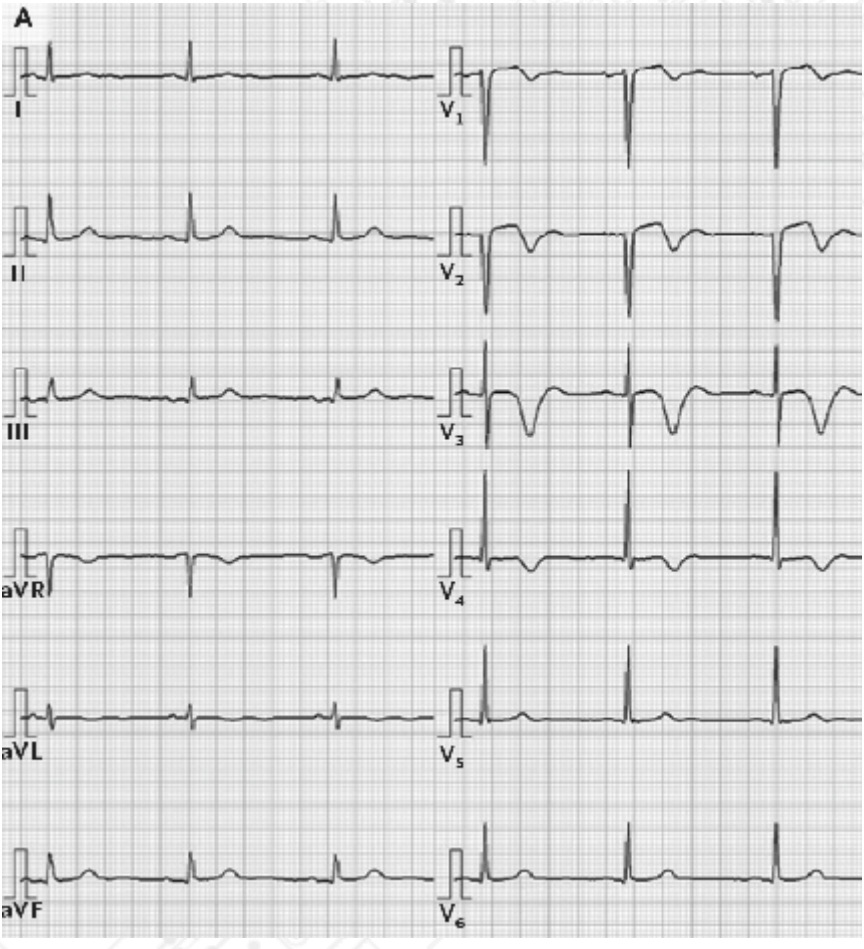


Fig. 5.8-Wellens' Syndrome (biphasic or deep precordial T wave inversions, particularly in V2-V3, seen during pain free period. Often indicates a critical or impending total occlusion of LAD coronary artery)

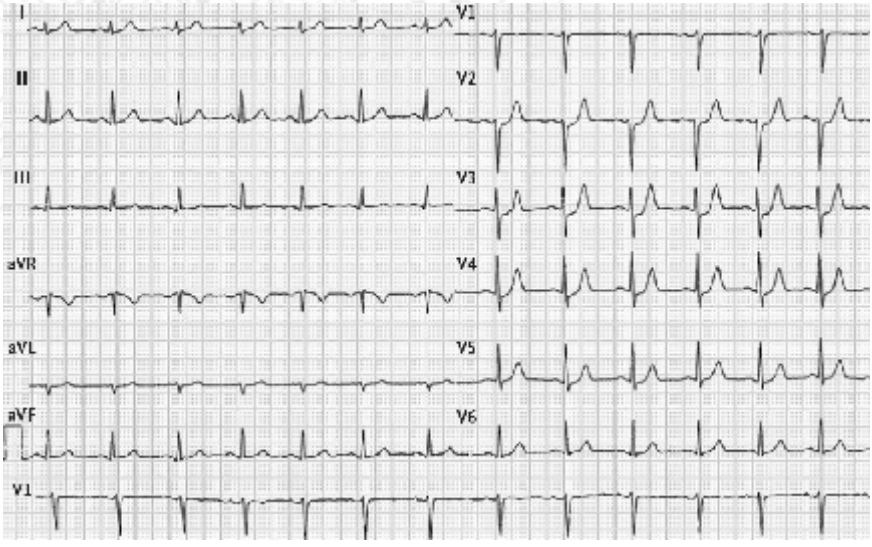


Fig. 5.9- de Winter pattern (up-sloping ST segment depression, that continues tall symmetric T waves in precordial leads; which is often seen in critical occlusion of proximal LAD coronary artery)

ECG CHANGES WITH MEDICATIONS AND ELECTROLYTE DISTURBANCE & MISCELLANEOUS CONDITIONS

Dr. Kunwar Abhimanyu Singh

The use of various medications is associated with characteristic changes on ECG. These changes may occur with either therapeutic or toxic blood levels. Medications that cause changes in electrolyte levels, such as those that result in electrolyte loss, may also cause changes on ECG

Commonly used drugs that result in ECG changes comprise :-

Digoxin – It has a narrow Therapeutic to Toxic ratio, and is a potent stimulator of arrhythmias.

- At therapeutic levels, digoxin frequently causes nonspecific ST changes with "scooping" of the ST segment and shortening of the QT interval.
- Digoxin causes SA nodal suppression and AV block.
- Digoxin can cause virtually any arrhythmia, but, because of its ability to enhance automaticity, ectopic arrhythmias are commonly encountered in digoxin toxicity.
- The commonest arrhythmia manifested by digoxin toxicity is multiform PVCs.
- The two most specific arrhythmias are accelerated

junctional rhythm and atrial tachycardia with AV block.

Sotalol and Amiodarone

- These agents slow conduction in general and result in bradycardia and prolongation of the PR, QRS, and QT intervals.
- Sotalol also has significant beta blocking properties, which exacerbates the bradyar rhythmic effects.
- Sotalol can also prolong the QT interval and cause torsades de pointe.

Quinidine and other Class IA agents

- These agents are less frequently used than previously because of side effects, pro-arrhythmic potential, and possibly increased mortality.
- Quinidine prolongs the QRS duration and QT interval, and may cause Torsades de pointe.
- Verapamil and Diltiazem
- These agents can cause sinus bradycardia, vary in amounts of AV block, and, in toxic doses, intra ventricular conduction defects.
- Their effects are additive with beta blockers.

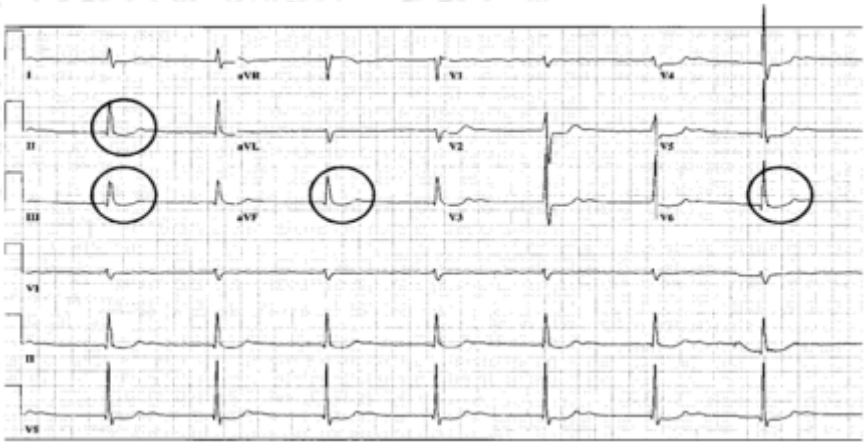


Fig 6.1 Junctional rhythm with "scooping" of the ST segment consistent with digoxin therapy

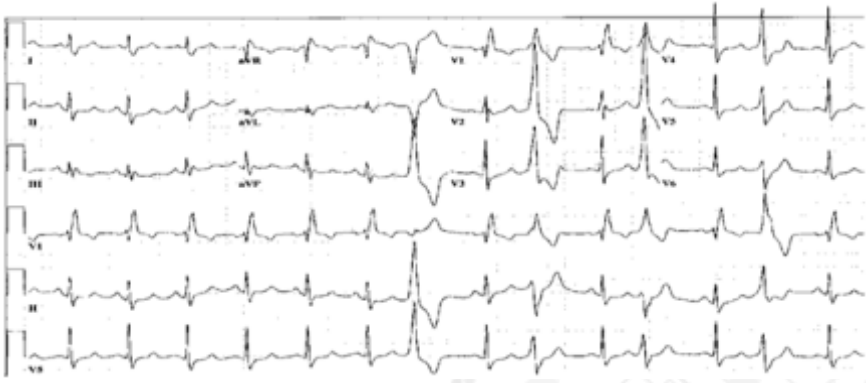


Fig 6.2 Sinus rhythm with frequent PVCs of varying morphology. This is the most common manifestation of digoxin toxicity, although it lacks specificity.

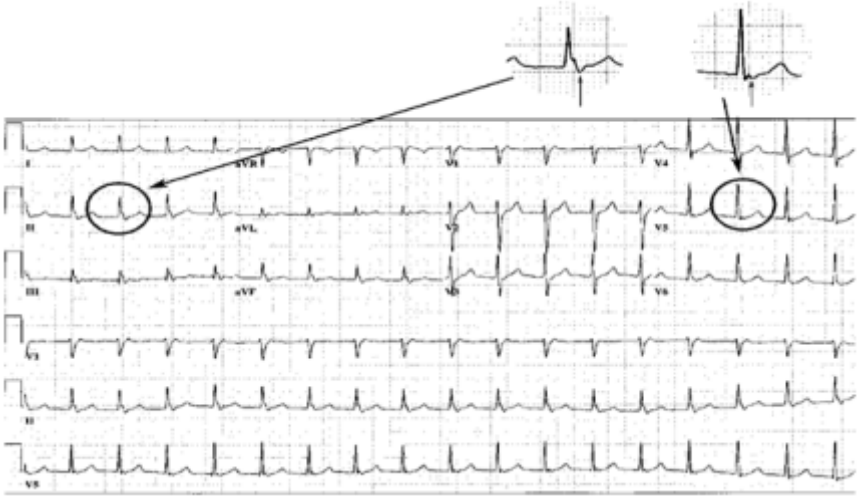


Fig. 6.3 Accelerated junctional rhythm with retrograde P waves following the QRS complexes, suggesting digoxin toxicity

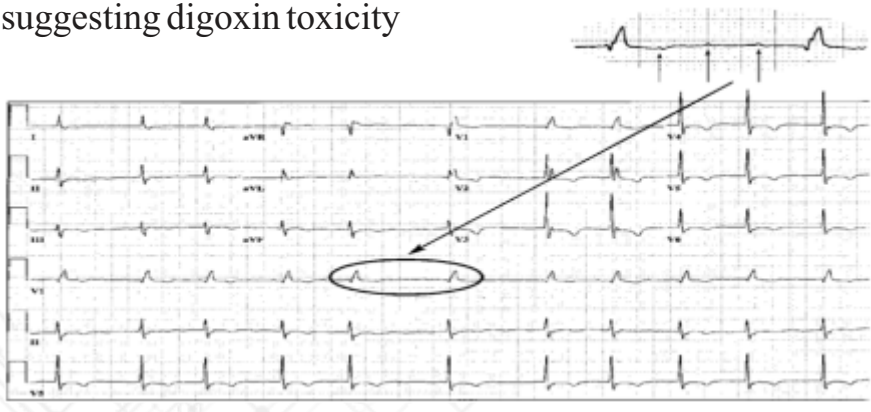


Fig. 6.4 Atrial tachycardia with variable AV block consistent with digoxin toxicity

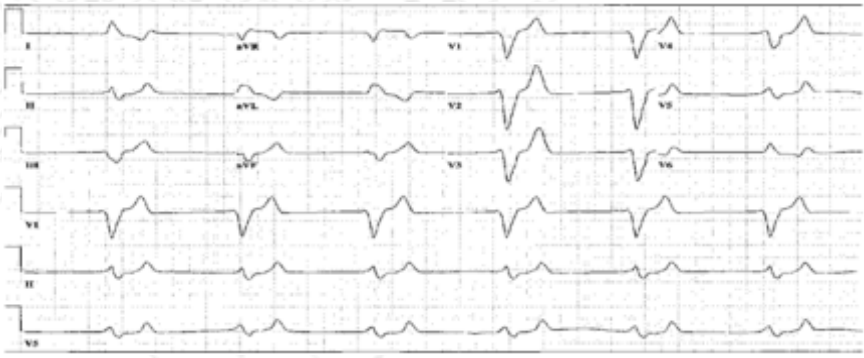


Fig 6.5 Junctional (or possibly idio-ventricular) rhythm with an extremely wide QRScomplex in this patient with sotalol toxicity

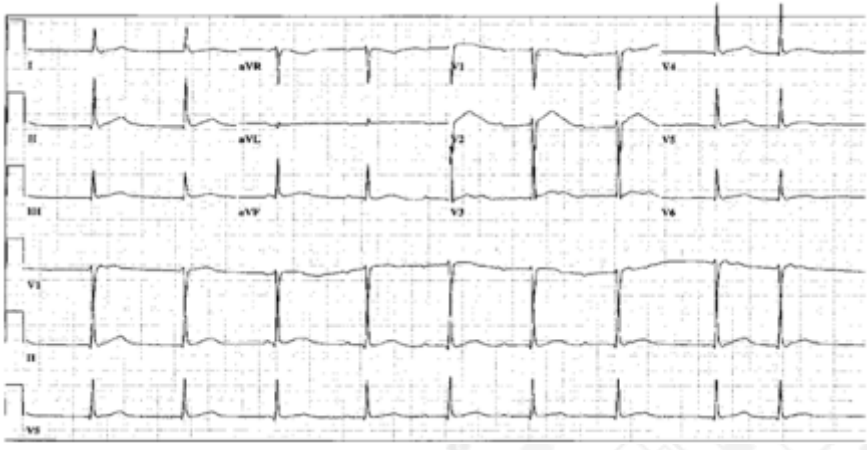


Fig 6.6 Sinus rhythm with varying amounts of AV block, a competing junctional pacemaker, and diffuse ST-T wave abnormalities in a patient with verapamil toxicity

Electrolyte abnormalities

Hypokalemia

- Hypokalemia potentiates a variety of arrhythmias, including VT and torsade de pointes.
- Hypokalemia is associated with ST segment depression, a prolonged QT interval, and a prominent U wave.

Hyperkalemia

- Hyperkalemia is manifested by peaked T waves, loss of obvious P waves or prolongation of the PR segment, and prolongation of the QRS complex.
- When potassium levels reach 8–9 mmol/l, the ECG may resemble a sine-wave; further elevation may cause asystole.

Hypocalcemia

- It is manifested by prolongation of the QT interval; the ST segment is usually flat and the T wave is not distorted.

Hypercalcemia is associated with a short QT interval.

Miscellaneous presentations on ECG

QT prolongation and U wave abnormalities

- QT interval should
- one half of the surrounding R-R interval.

Congenital long QT syndromes

- Jervell and not exceed Lange-Nielsen syndrome is an autosomal recessive disorder associated with deafness.
- Romano-Ward is an autosomal dominant disorder.

Acquired long QT syndromes

- Non-drug causes of long QT interval include ischemia, central nervoussystem (CNS) lesions, and significant bradyarrhythmias.
- Many drugs can prolong the QT interval, including the Class IA, IC, and III anti-arrhythmic agents, erythromycin, some antihistamines, and some psychiatric drugs.

U wave abnormalities

- Prominent U waves are seen with hypokalemia, digoxin, LVH, and Amiodarone (see previous page)
- Negative U waves are encountered in hypertension (HTN), aortic and mitral disease, and ischemia.

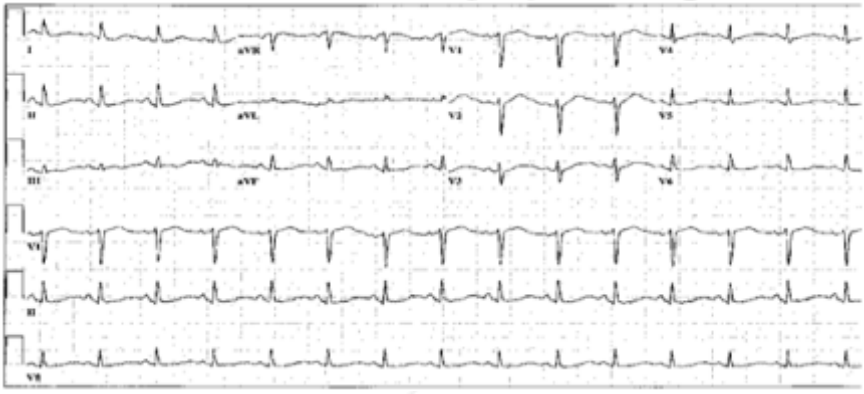


Fig. 6.7 Sinus rhythm with an extremely long QT interval in a patient with severe hypokalemia (k= 1.8 mml/l)

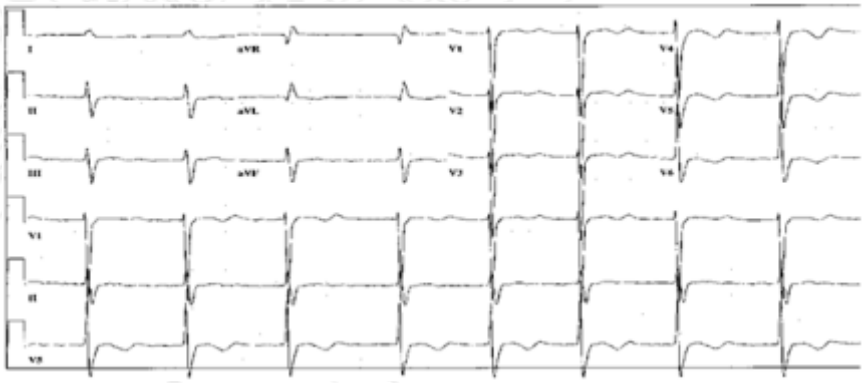


Fig. 6.8 Junctional rhythm with diffuse nonspecific ST segment and T wave changes and prominent U waves in a patient with severe hypokalemia (K = 2.1 mmol/l)

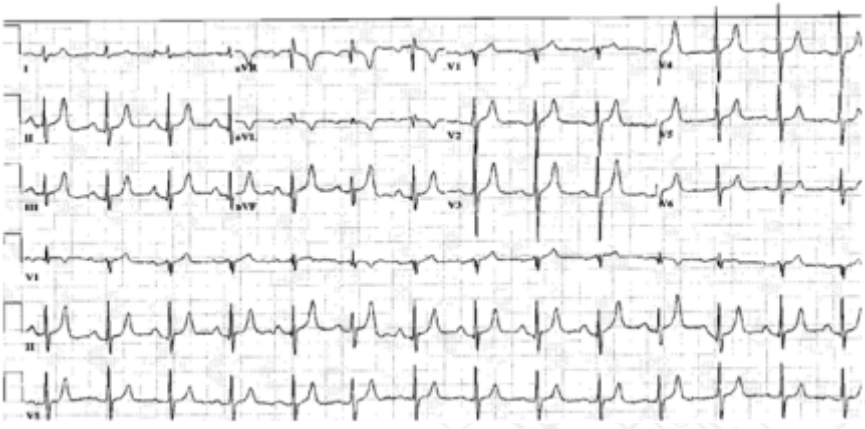


Fig. 6.9 Sinus rhythm with peaked T waves in early hyperkalemia (K = 6.8 mmol/l)

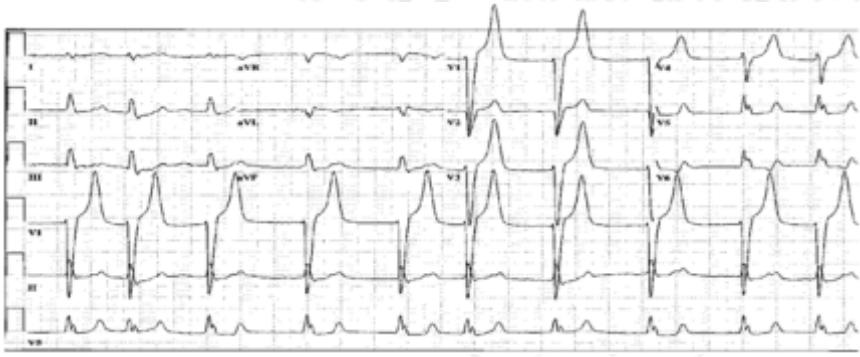


Fig. 6.10 Junctional rhythm with occasional retrograde P waves, increased QRS duration, and extremely tall and pointed T waves in this patient with hyperkalemia (K = 8.2 mmol/l)

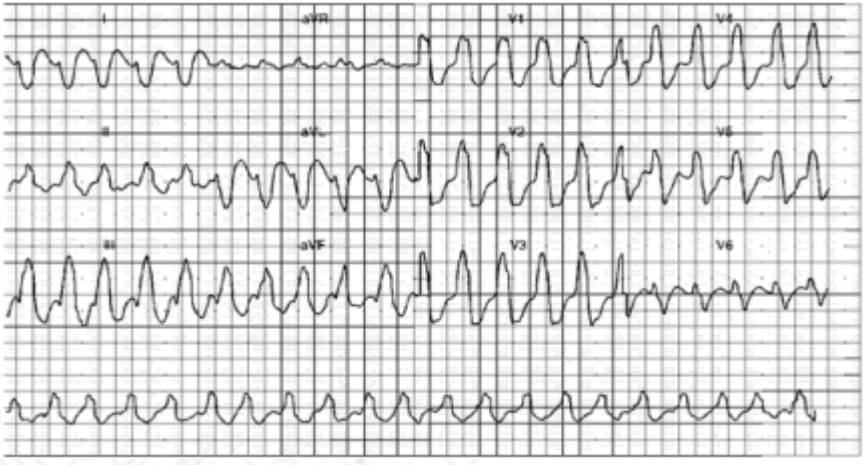


Fig. 6.11 Wide complex tachycardia in a patient with severe hyperkalemia (K =8.4 mmol/l)

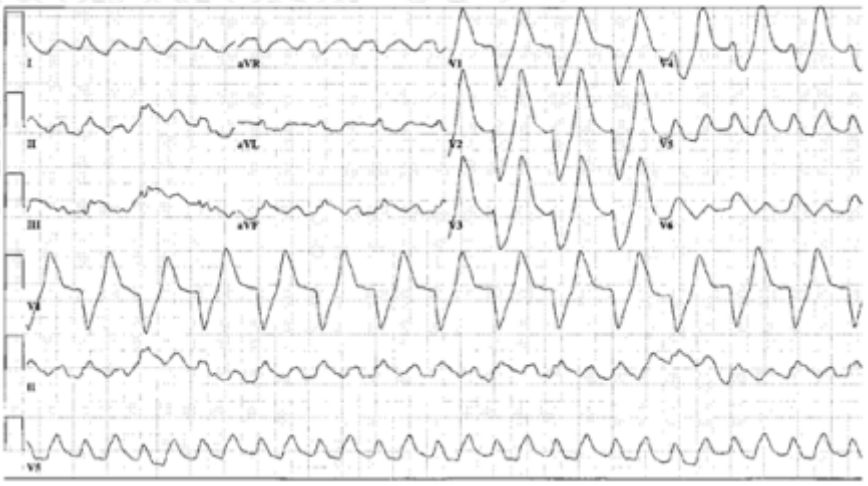


Fig. 6.12 A sinusoidal rhythm in a patient with severe hyperkalemia and acidosis

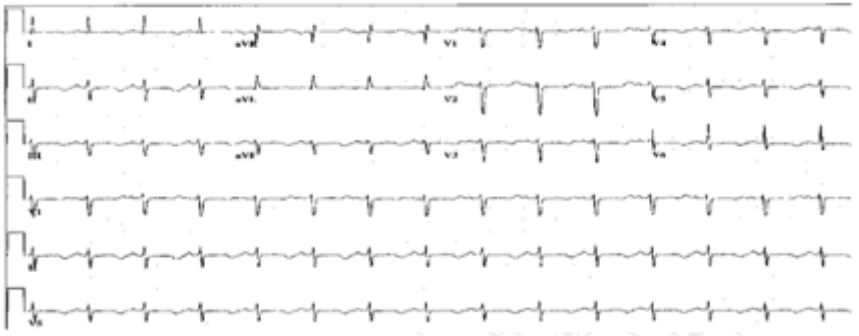


Fig. 6.13 Sinus rhythm with a very long QT interval. The ST segment is flat and the T wave is fairly narrow in this patient with pancreatitis and severe hypocalcaemia.

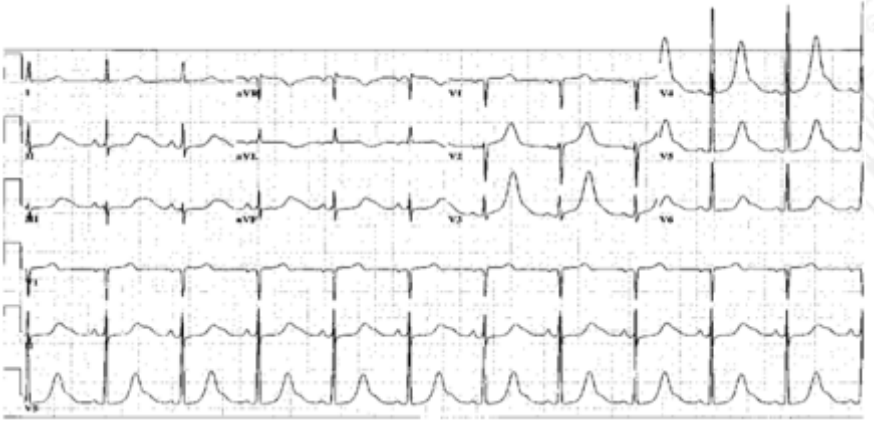


Fig. 6.14 Sinus rhythm with prominent T waves and a very long QT interval, in this case dueto acute ischemia

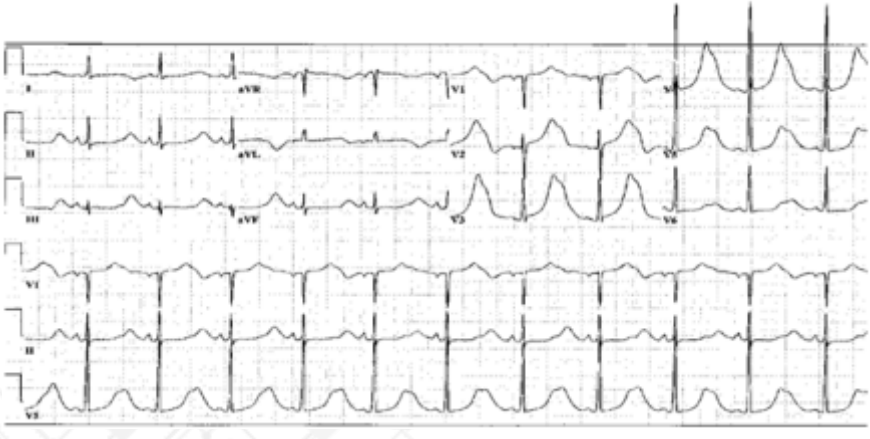


Fig. 6.15 Sinus rhythm with extremely prominent T waves and a prolonged QT interval in a patient with acute ischemia

Causes of tall R waves in V_1

- Right ventricular hypertrophy (RVH) o Posterior MI
- RBBB
- Wolff-Parkinson-White (WPW)
- Hypertrophic obstructive cardiomyopathy (HOCM) with asymmetric septal hypertrophy (ASH)
- Congenital dextrocardia
- Duchenne's muscular dystrophy

Causes of ST segment elevation:

- Acute myocardial injury o Left ventricular aneurysm
- Early repolarization
- Acute pericarditis
- LVH
- LBBB
- Hyperkalemia
- Hypothermiao Scorpion sting

CNS injury and the ECG

- Severe acute CNS lesions, typically subarachnoid hemorrhage are occasionally associated with ST segment and T wave changes
- The most likely explanation for these changes is unilateral perturbation of the sympathetic ganglia at the base of the brain.

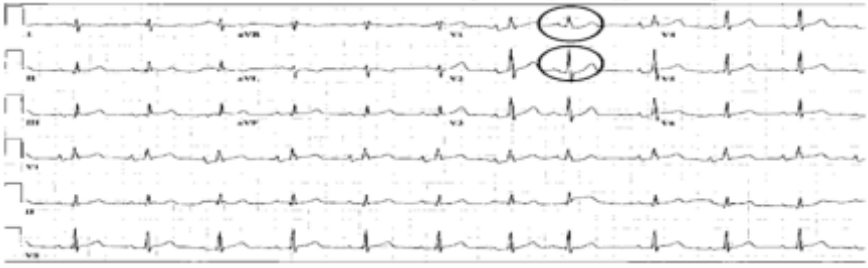


Fig 6.16 Sinus rhythm, right axis deviation, and tall R waves in the early precordial leads in a patient with RVH

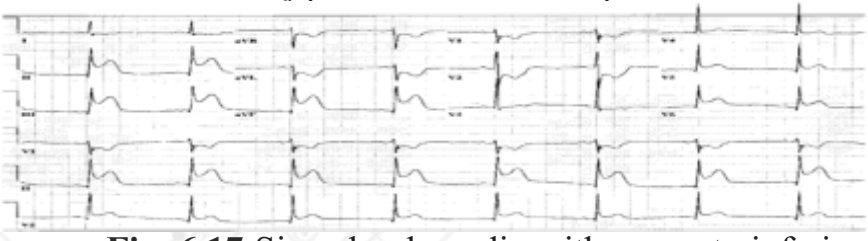


Fig. 6.17 Sinus bradycardia with an acute inferior-posterior MI. Note the ST segment elevation in the inferior leads, the ST segment depression in V_1 and V_2 and the tall R wave in V_2.

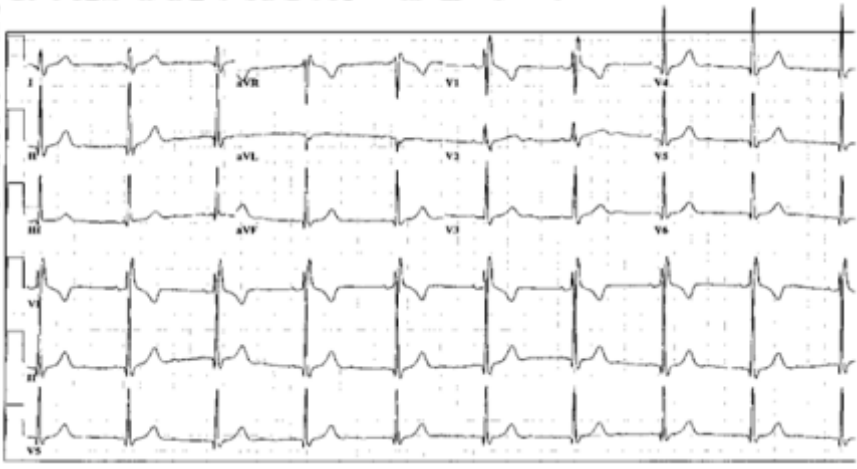


Fig. 6.18 Sinus rhythm and RBBB

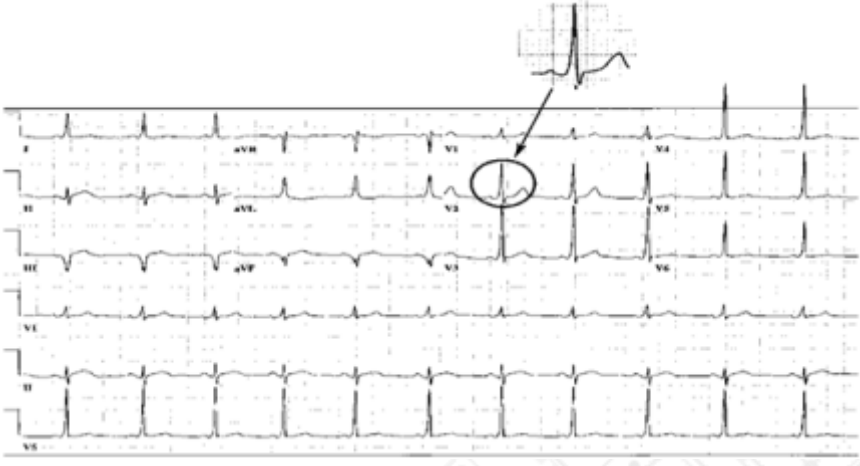


Fig. 6.19 Sinus rhythm with a short PR interval, delta waves and prominent R waves in V_1 and V_2 consistent with WPW

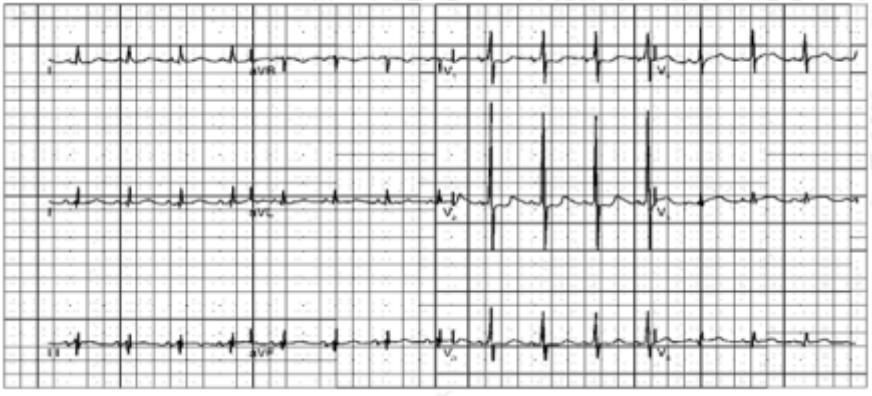


Fig. 6.20 Sinus rhythm with very prominent R waves in V_1 and particularlyV_2 in a patient with hypertrophic cardiomyopathy and an extremely thickened septum

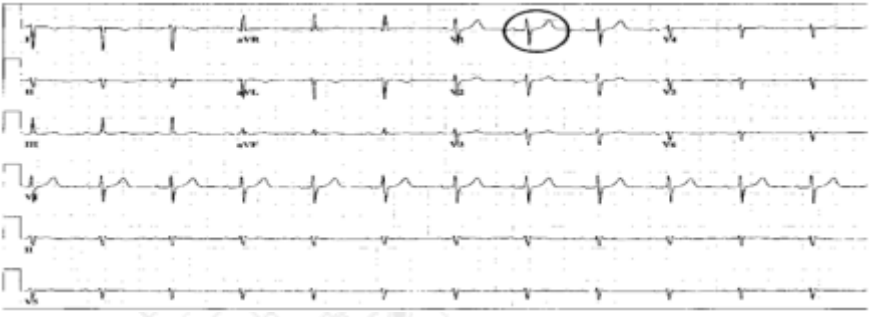


Fig. 6.21 An abnormal P wave and QRS axis, a tall R wave in V_1, and decreasing R waves across the remaining precordial leads in this patient with situs inversus with dextrocardia

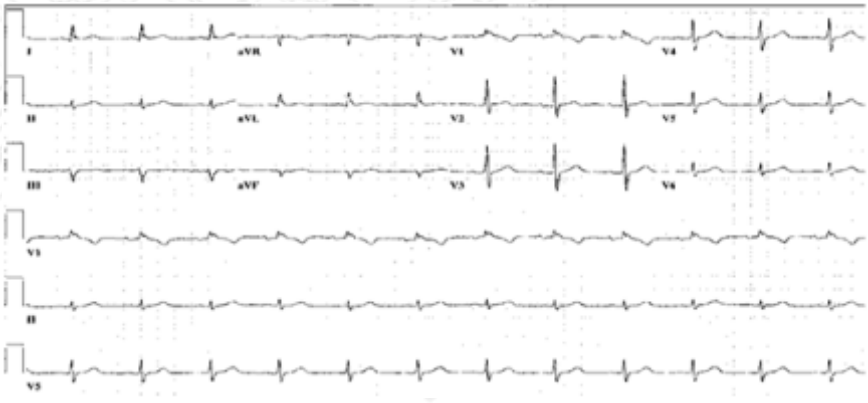


Fig. 6. 22 Sinus rhythm with a prominent R wave in V_1 and V_2 in a boy with Duchenne's muscular dystrophy

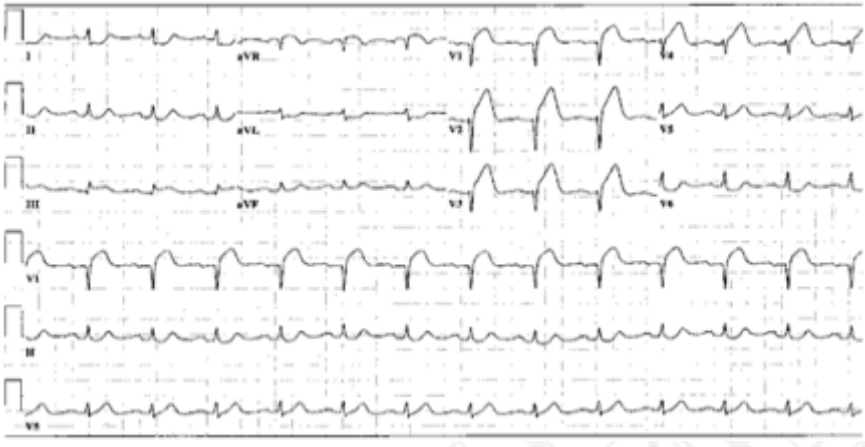


Fig. 6.23 Sinus rhythm with an acute anteroseptal MI

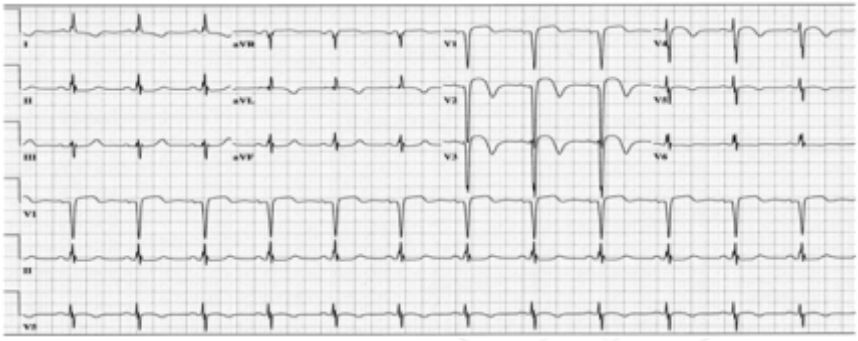


Fig. 6.24 Sinus rhythm with Q waves, ST segment elevation, and T wave inversionin V_1-V_3, all consistent with an anteroseptal MI. However, these findings were present on a previous ECG from 6 months previously, strongly suggesting that the residual ST segment elevation represents a left ventricular aneurysm.

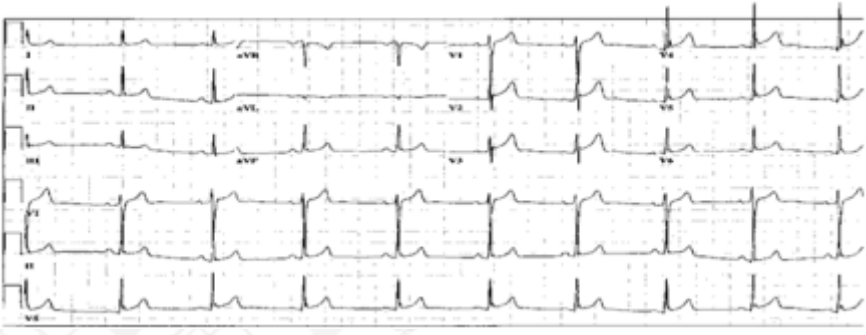


Fig. 6. 25 Sinus rhythm with diffuse ST segment elevation representing early repolarization in this healthy 22-year-old man

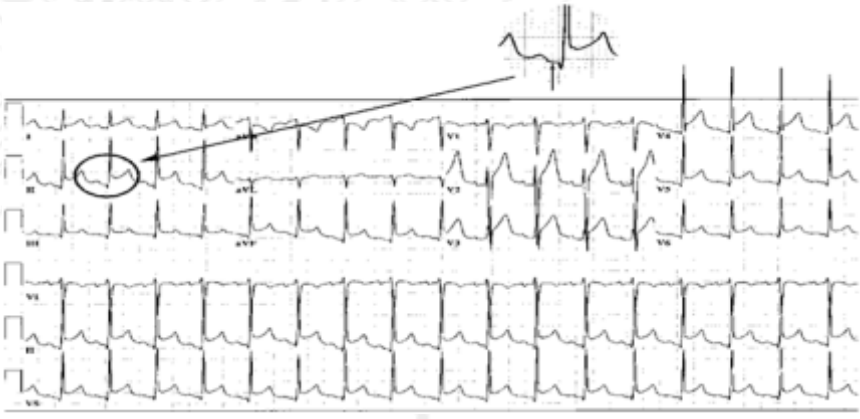


Fig. 6.26 Sinus tachycardia with diffuse ST segment elevation and PR segment depression in Lead II consistent with acute pericarditis

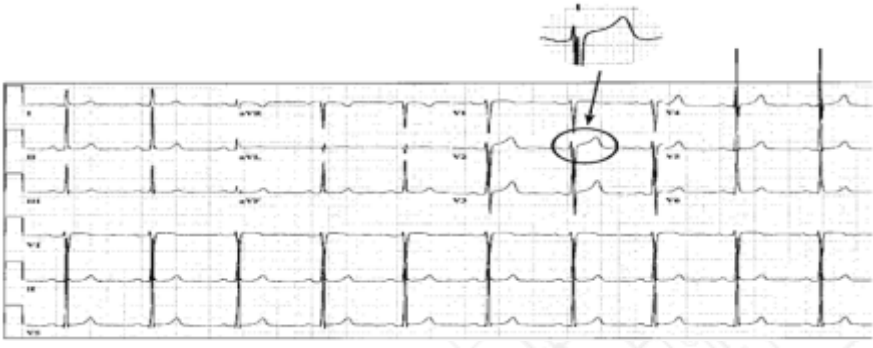


Fig. 6.27 Sinus rhythm withvoltage criteria for LVH and STsegment elevation in V_1-V_4

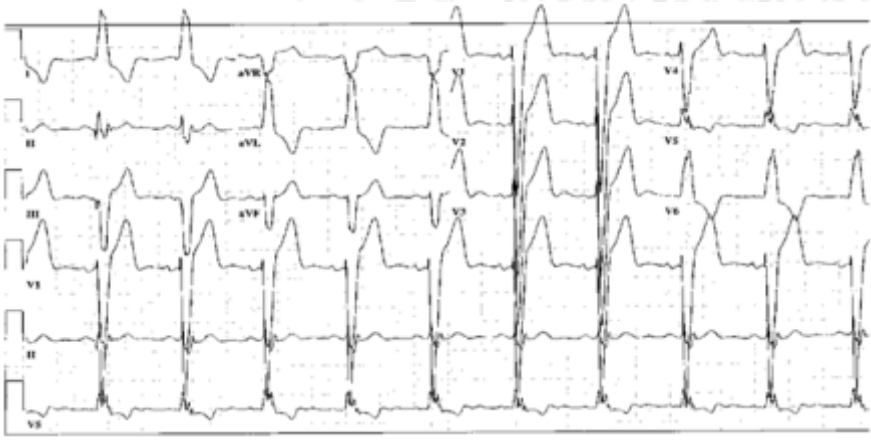


Fig. 6.28 Sinus rhythm with LBBB and ST segment elevation in V_1-V_4

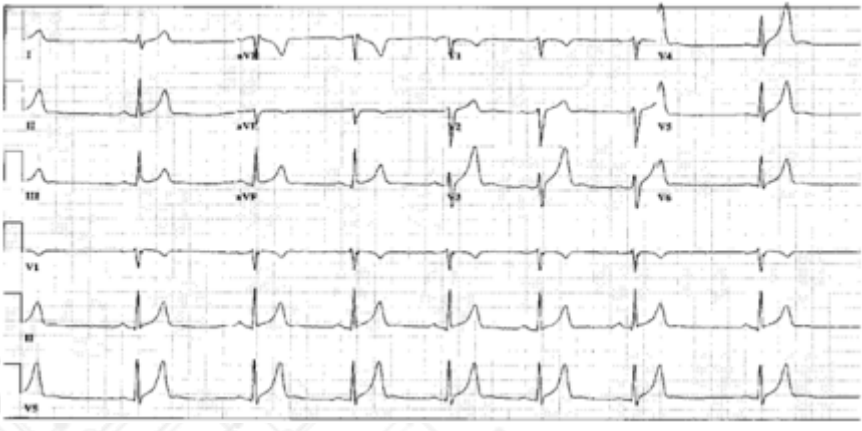


Fig. 6.29 Sinus bradycardia with ST segment elevation and peaked T waves in a patient with early hyperkalemia (K = 6.3 mmol/l)

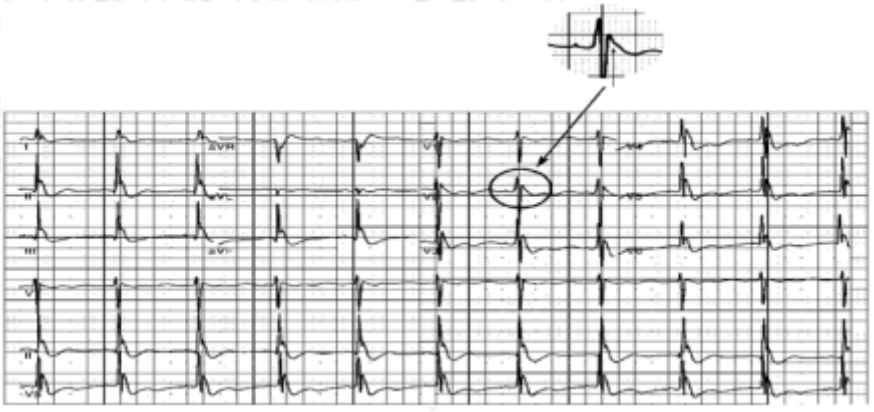


Fig. 6.30 Sinus rhythm with J point elevation (so-called "Osborne waves") in a patient with severe hypothermia

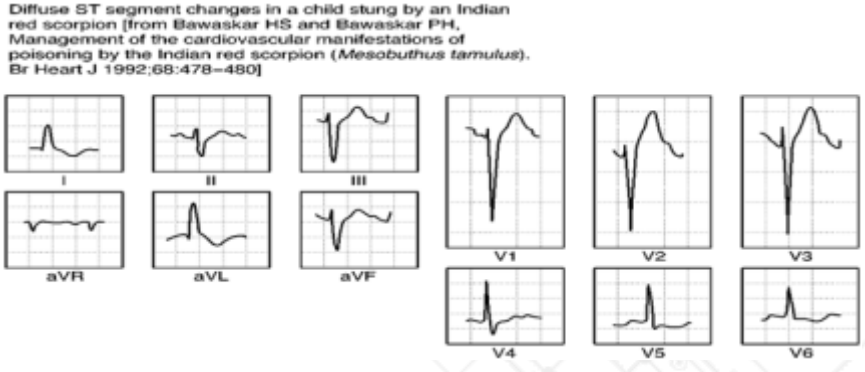


Fig. 6.31 Diffuse ST Segment changes in a child stung by an indian red scorpion

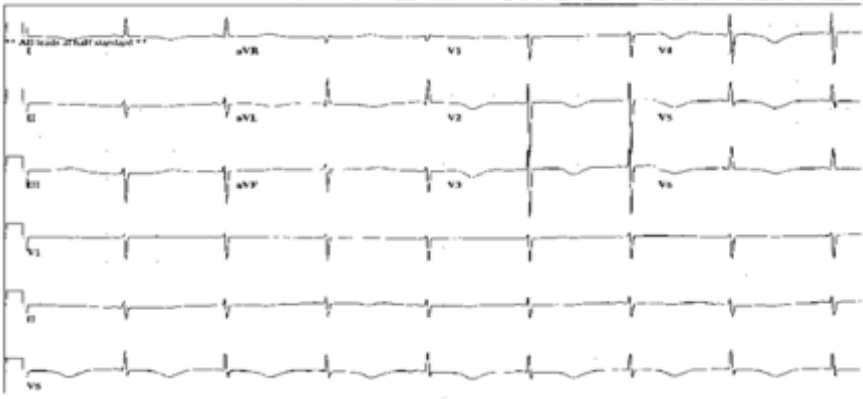


Fig. 6.32 Sinus bradycardia, inverted T waves, and QT prolongation in a patient with meningitis and encephalitis.

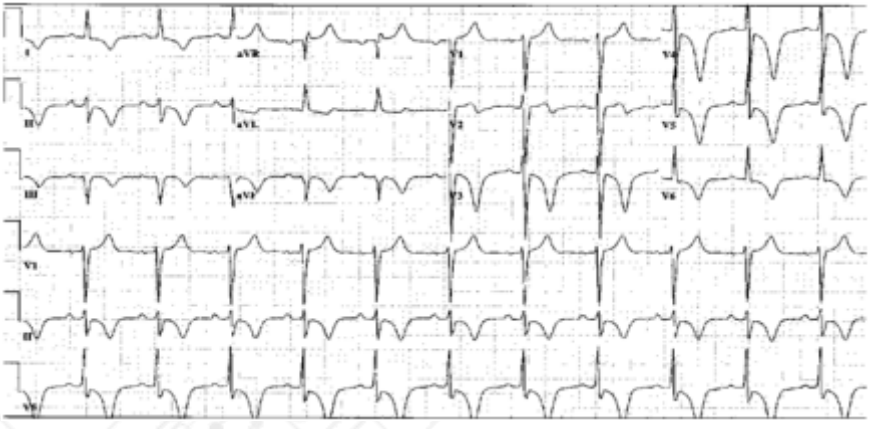


Fig. 6.33 Sinus rhythm with LVH and deeply inverted T waves. An echocardiogram showed LVH and normal LV systolic function. ACT scan of the head showed

PRIMARY CARDIAC CARE AT PHC LEVEL

Dr. Nikhil Gupta

It is important to recognize cardiac emergencies immediately and treat them accordingly. Cardiac emergencies are notorious in a sense that the signs and symptoms are seen in a huge spectrum of diseases, where some differentials are so benign that they do not require any further investigation.

On the other hand, they may present with such vague symptoms that physicians may miss the diagnosis if they do not have a high index of suspicion

Common Signs and Symptoms in Cardiovascular Emergencies : Patients can present with typical and atypical signs and symptoms.

Typical cardiovascular symptomatology

- Chest symptoms (chest pain, chest tightness or pressure-like symptoms, discomfort)
- Breathlessness (Orthopnea, paroxysmal nocturnal dyspnea, exertional breathlessness)
- Palpitation
- Sweating
- Pedal edema or generalized edema

Atypical symptomatology:

- Pain in the neck, jaw, throat or arm (ischemic pain)
- Pain in abdomen (related to heart failure/liver congestion or ischemia)
- Pain or numbness in the leg or cold extremities (related to poor circulation associated with vascular problems or poor cardiac output)
- Dyspepsia
- Light headedness or dizziness
- Pain at the back (ischemia)
- Fainting (Syncope) or near fainting
- Fatigability and tiredness
- Cardiac asthma (cough)

Table 7.1 Major Causes of Acute Chest pain

CONDITION	ONSET/DURATION	QUALITY	LOCATION
Myocardial ischemia	Stable angina: Precipitated by exertion, cold, or stress; 2–10 min Unstable angina: Increasing pattern or at rest Myocardial infarction: Usually >30 min	Pressure, tightness, squeezing, heaviness, burning	Retrosternal; often radiation to neck, jaw, shoulders, or arms; sometimes epigastric
Pericarditis	Variable; hours to days; may be episodic	Pleuritic, sharp	Retrosternal or toward cardiac apex; may radiate to left shoulder
Acute aortic syndrome	Sudden onset of unrelenting pain	Tearing or ripping; knifelike	Anterior chest, often radiating to back, between shoulder blades
Pulmonary embolism	Sudden onset	Pleuritic; may manifest as heaviness with massive pulmonary embolism	Often lateral, on the side of the embolism
Pneumonia or pleuritis	Variable	Pleuritic	Unilateral, often localized
Spontaneous pneumothorax	Sudden onset	Pleuritic	Lateral to side of pneumothorax

Acute coronary syndrome

Acute coronary syndrome includes a spectrum of clinical presentations which range from unstable angina to non-ST elevation MI and ST elevation MI. They can be differentiated on the basis of history, ECG changes and blood investigations. The management started should be according to the diagnosis.

Spectrum of ACS

Unstable angina: It is referred to as pre-infarction angina or pre-occlusive syndrome. It is a warning sign of infarction. It is characterized by angina at rest or on minimal exertion or crescendo angina with rapid worsening in severity of angina.

Myocardial infarction: It is defined as cell death and necrosis. Below mentioned are the criteria satisfying the diagnosis of acute, evolving or recent MI.

Diagnosis

Diagnosis is done based on the history and physical examination and finally after diagnostic testing. The diagnostic testing includes ECG, chest X-Ray, serum cardiac markers, echocardiography, scintigraphy, CT coronary angiography and invasive coronary angiography, depending on the requirement and the availability.

- Typical rise and fall of cardiac markers (Troponin T and I) , along with Symptoms of ischemia
- ECG changes (either Q waves or changes consistent with ischemia like ST or T wave changes)

Management

The management goal is early revascularization (within 12 hours of chest pain onset) and reperfusion using either fibrinolysis or primary angioplasty in ST elevation MI and early coronary revascularization (within 48-72 hours of chest pain onset) for high risk non-ST elevation ACS patients. Medical management is divided into two categories:

Pharmacologic intervention

- Oxygen should be administered when blood oxygen saturation is 90% or if the patient is in respiratory distress. In patients whose ischemic symptoms are not relieved by nitrates and beta-blockers, opiate administration is reasonable while waiting for immediate coronary angiography, with the caveat that morphine may slow down the intestinal absorption of oral platelet inhibitors.
- **Nitroglycerine:** It is a coronary vasodilator and

reduces myocardial pre-load and after load.

- **Pain management:** If the patient is in severe pain and not responding to NTG and beta-blockers.
- **Beta-blockers:** Early administration of beta-blockers should be avoided in these patients if the ventricular function is unknown and should not be administered in patients with symptoms possibly related to coronary vasospasm or cocaine use, as they might worsen coronary artery spasm by leaving alpha-mediated vasoconstriction unopposed by beta-mediated vasodilation.
- **Calcium channel blockers (CCBs):** CCBs are recommended for ischemic symptoms when beta-blockers are not successful, are contraindicated or cause unacceptable side effects. Long-acting CCBs and nitrates are recommended for patients with coronary artery spasm. They can be used for rate control in patients with SVT when beta blockers are not tolerable.
- **ACE inhibitors:** ACE inhibitors should be started and continued indefinitely in all patients with a left ventricular ejection fraction (LVEF) below 40% and in those with hypertension, diabetes mellitus

or stable chronic kidney disease (CKD), unless contraindicated

- **Antiplatelet**: Antiplatelet therapy reduces progression to acute infarction in patients with non-AMI ACS patients. Aspirin or P2Y12 inhibitors (Clopidogrel, Ticagrelor or Prasugrel) may be used. Antiplatelet treatment reduces mortality.
- **Statins**: All patient with ACS should be immediately started on high intensity statin therapy (Atorvastatin 40 or 80 mg or Rosuvastatin 20 or 40 mg), which should then be continued for lifelong.
- **Anticoagulation:** Administer anticoagulation, in addition to antiplatelet therapy, for all patients, irrespective of the initial treatment strategy. **Subcutaneous(SC)** enoxaparin for the duration of hospitalization or until PCI is performed

IV unfractionated heparin (UFH) for 48 h or until PCI is performed (level of evidence: B)

- **Reperfusion therapy -** Either by using thrombolytics or primary PCI, increases the

opportunity to salvage ischemic myocardium. Fibrinolytic therapy improves coronary flow, limits infarct size and improves survival.

Heart failure

It is defined as the pathophysiologic state in which the heart is not capable of pumping sufficient supply of blood to meet the body requirements or else requires elevated ventricular filling pressures to accomplish this goal.

Pathophysiology of acute pulmonary edema

Cardiogenic pulmonary edema is due to increased capillary hydrostatic pressure secondary to acute ischemia or infarction, cardiomyopathy, valvular heart disease or hypertensive emergencies. Non-cardiogenic pulmonary edema occurs due to alteration in the permeability of pulmonary capillary membrane.

Compensatory mechanisms

The compensatory mechanisms secondary to heart failure include increase in stroke volume in response to increased pre-load, increased systemic vascular resistance

and cardiac hypertrophy.

Treatment of heart failure

- Stabilization of patient and resuscitation
- Identify the underlying and precipitating cause and treat it
- Control the symptoms and acute congestive state by reducing the cardiac work load (reducing pre- and after load), controlling excessive salt and water retention and improving cardiac contractility.
- Acute pulmonary edema with adequate perfusion:
  - Nitrates
  - Morphine
  - Loop diuretics
  - Nitroprusside
  - Non-invasive or invasive ventilation
- Acute pulmonary edema in hypotensive patients
  - Vasopressors and inotropes to maintain coronary perfusion
  - Manage hypotension due to cardiogenic shock or due to volume depletion (identified by cardiac index and pulmonary artery

outflow pressure)

- Judicious fluid challenge if low PCWP <15 mm Hg)
- IABP/ mechanical circulatory support
- Emergency revascularization if ischemic cardiogenic shock

Treatment of chronic heart failure:

- Manage hypertension
- Reverse remodelling by beta-blockers, ACE-I, aldosterone antagonists, ARB
- Vasodilator therapy (ACE-I, ARB, Nitrates)
- Diuretics
- SGLT-2 Inhibitors
- Cautious use of calcium blockers for hypertension, angina and dysrhythmia management
- Beta-blocker therapy: Carvedilol may be effective agent in chronic HF
- Digoxin

Hypertensive emergencies

In the Emergency Department hypertension presents as one of the four varieties:

1. Hypertensive emergency or crisis with acute end

organ ischemia

2. Hypertensive urgency: Patients with poorly controlled hypertension
3. Mild hypertension
4. Transient hypertension which is related to anxiety or complaint

Only hypertension crisis requires treatment in the emergency department within 90 min of their presentation. Patients presenting with hypertensive emergencies will have markedly elevated BP and evidence of acute dysfunction in the cardiovascular, neurologic or renal system. Following are the conditions defined as hypertensive crisis:

1. Accelerated or malignant hypertension:

- o Hypertensive encephalopathy
- o Microangiopathic haemolytic anemia
- o Acute renal failure

2. Aortic dissection
3. Eclampsia/pre-eclampsia
4. Severe hypertension in the setting of:

- o Myocardial ischemia
- o LVF

- Uncontrolled hemorrhage
- Systemic reperfusion therapy for stroke or MI
- Postoperative state

Table 7.2 Drugs of choice in treatment of hypertensive emergencies:

Emergencies	Drug of choice	Alternatives
Hypertensive encephalopathy	Nicardipine Labetalol	Esmolol Enalaprilat
Intracranial Hemorrhage	Nicardipine Labetalol	Esmolol
Acute Pulmonary Edema	Nitroglycerine Furosemide Enalaprilat	Nicardipine Sodium Nitroprusside
Aortic Dissection	Esmolol and Sodium Nitroprusside Labetalol	Esmolol and Nicardipine Diltiazem, Verapamil
Ischemic Stroke	Nicardipine Labetalol	Esmolol Enalaprilat
Acute Kidney Injury	Fenoldapam Nicardipine, Clevidipine	Labetalol Sodium Nitroprusside
Preeclampsia/eclampsia	Hydralazine Labetalol	Nicardipine
Sympathetic Crisis	Phentolamine Nitroglycerine	Fenoldapam Clevidipine Nicardipine Sodium nitroprusside

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