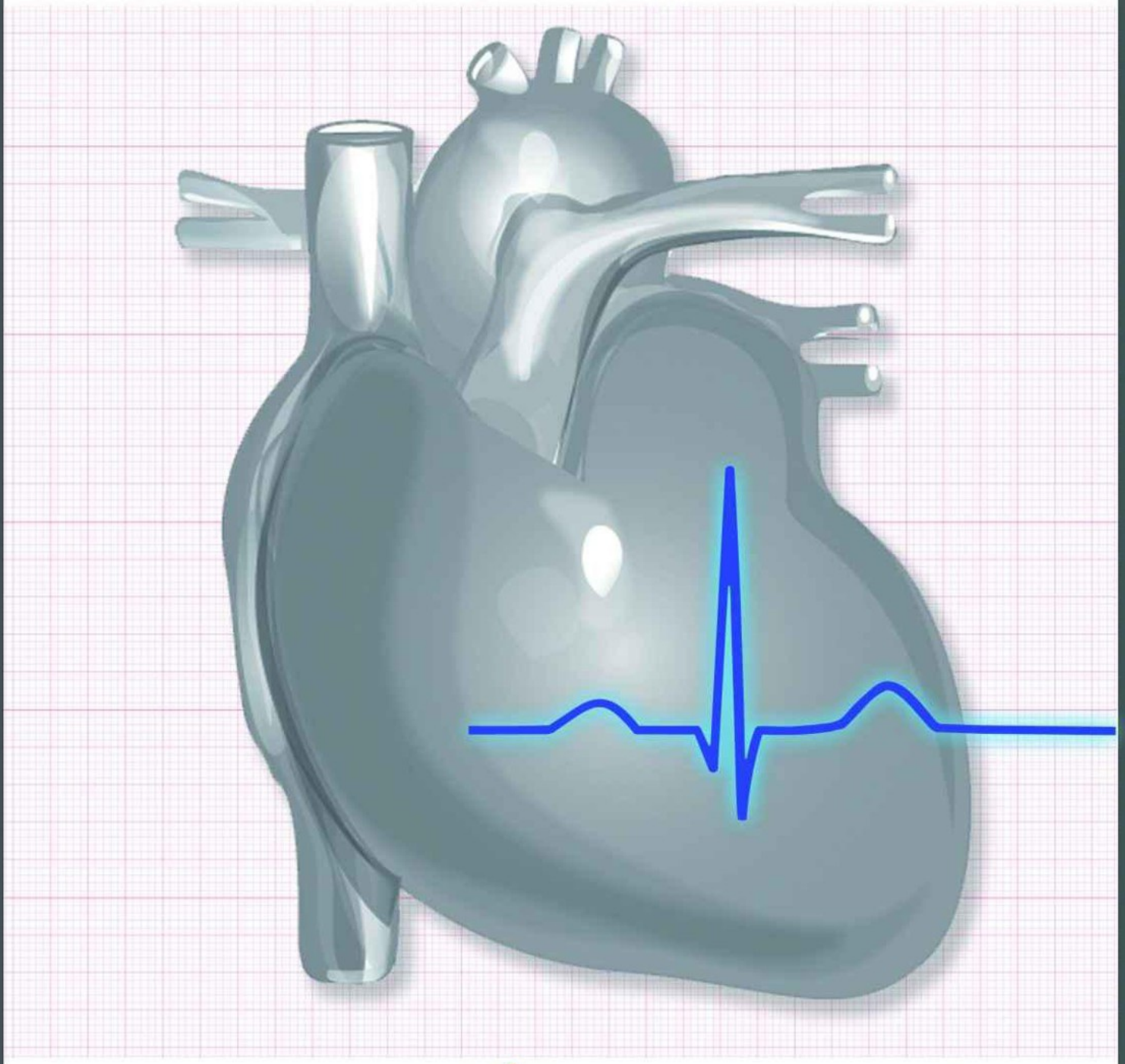
Basic Concepts of EKG

A simplified approach

Harilal K Nair MSN ANP-C CCRN CMC



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USA

Disclaimer

This book is intended for beginners and professionals who are trying to develop better understanding of basic concept of EKG and its analysis. Therefore, this book is in no way a

substitution to clinical judgment as well as established medical guidelines for actual patient care in the field. Since the modern medicine is a dynamic arena with constant addition of newer knowledge from clinical research, the author and the reviewers made genuine efforts to include most accurate and up to date information in all parts of this book. However, because of the ever-changing nature of technology and science, the author or publisher is not responsible for any inaccuracies of information especially related to drugs and devises described in this book. Since this book is not meant as a resource for active patient care, the individual learners are expected to take best efforts to identify most updated information in this regard from other available sources.

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Preface

Basic concepts of EKG- A simplified approach is an attempt to make the complex nature of EKG analysis to fairly simple and logical endeavor. A 'rationalized approach with simplified analogy' method is used throughout this book. I believe in the simple to complex and concrete to abstract nature of student learning and therefore, a substantial effort has been made to make each and every concepts of EKG in to simple building blocks for a complex learning process. With conscious awareness, I haven't gone much in to the complexities of electrophysiology as they may be bit advanced for targeted learners without more practical experience with EKG analysis. I would like my readers to consider this book as one of the basic textbooks they use to build a strong foundation for their advanced learning.

I would like to reiterate the basic concept for effective use of this book that every part is important in building a stable foundation for further advancement in EKG analysis. Being a

constant leaner by myself, I don't appreciate books with complex concepts without sufficient explanations. Therefore, being very critical to my work, I did my best effort to remove any clutter and made it simple and up to the point with adequate rational explanations of events in all possible ways.

In order to facilitate multisensory learning of core ideas, appropriate drawings and highlights of important points are included throughout this book. I hope this will enable the students to easily skim through the chapters after their initial thorough reading. All of the important points are color coded and highlighted so that they are more visible than simple detailed mono color paragraphs of the traditional books.

I understand the fact that there are errors in every work no matter how many reviewers sweep through it. Therefore, I would like my readers to send us feedback so that we can improve ourselves for our future editions. I highly appreciate your comments and looking forward for every opportunity to improve and help someone to understand the complexities of modern medicine.

I would like to thank my mentors and support team for constant encouragement, support and confidence that they impart on me to make this book a reality in considerably short period of time. Without them and above all, the help of god almighty, I wouldn't be able to make this project a success.

Sincerely

Harilal K Nair MSN ANP-C CCRN- CMC

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Design and Art

Sincere thanks to

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The God Almighty

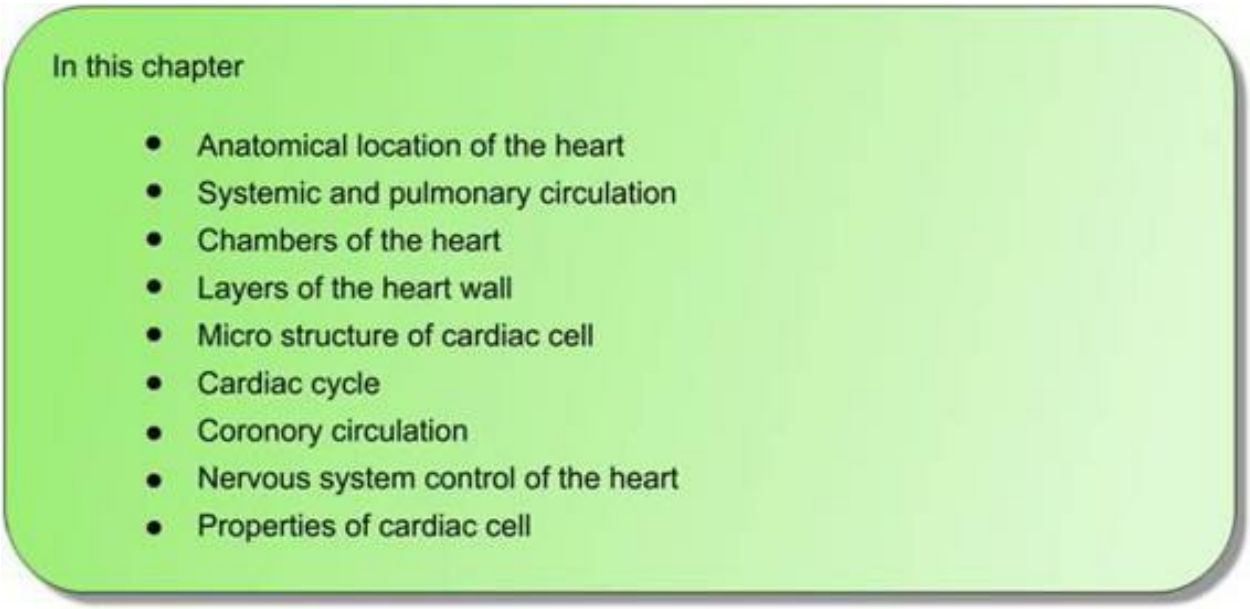
to make this book a reality.

Part I

Basic Cardiac Physiology

Chapter 1

Fundamentals of Cardiac Function



The heart is a delicate muscular structure lies in the mediastinum and is protected by the rib cage. It is usually situated left of the midline and above the diaphragm. The part of the chest wall overlying mediastinum is called precordium. The apex of the heart is the conical part lies at the bottom and is more towards anterior wall of the chest. Base of the heart is the wider surface along with greater vessels situated on the top. Anatomical variations in position of the heart and greater vessels can be found in cases like *dextrocardia,* where the apex of the heart is rotated towards right side.

Circulatory System

The human heart has four pumping chambers known as **right atrium**, **right ventricle**, **left atrium** and **left ventricle**. Along with pumping blood to the systemic and pulmonary circulations, these chambers have unique role in maintaining various hemodynamic parameters. Therefore, assessment of blood volume, pressure, temperature etc. at various parts of the circulatory system can help in identifying pathophysiologic changes of the heart and other associated structures

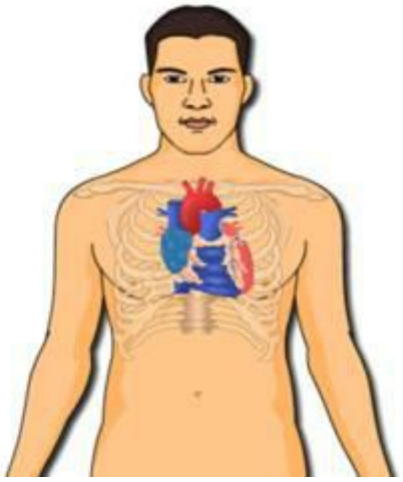


Fig 1.1 Anatomical location of the heart

The heart is a combination of two distinct pumping systems: right pump and left pump. *Right atria and ventricle act as the pump for pulmonary circulation* whereas; *left atria and ventricle constitute the one for systemic circulation*. These two systems are seamlessly connected through the lungs. Both functional units have their own reservoirs (right and left atrium), individual pumping chambers (right and left ventricle) and distinct valve structures that ensure unidirectional flow of blood in the system.

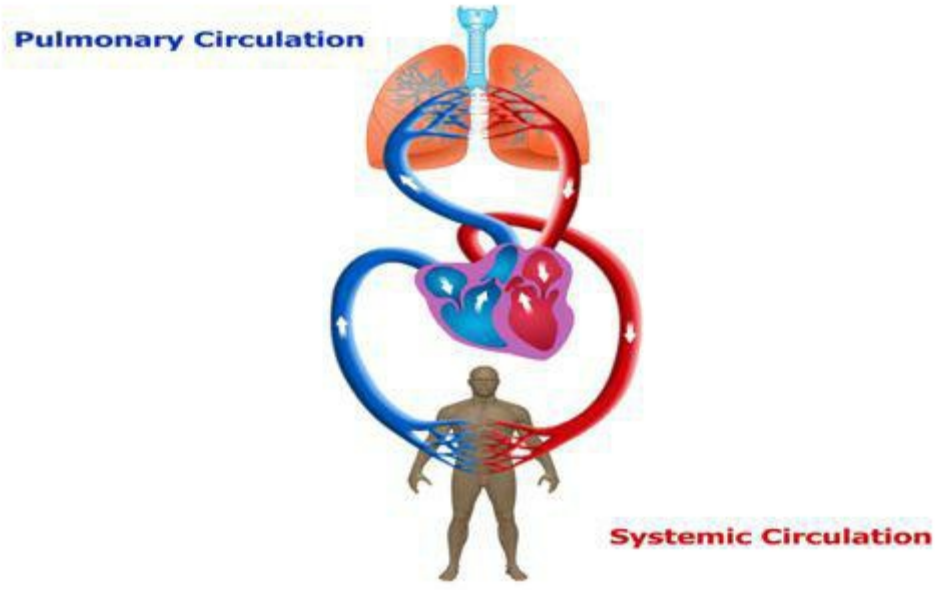


Fig 1.2 Systemic and venous circulation

Absence of flow regulators in the form of valves within the pulmonary system helps in balancing pressure difference between right and left pumping systems. Left side of the heart has much higher pressure than its counterpart.

Chambers of the Heart

Right atrium This is the *right uppermost chamber* of the heart. The venous circulation from rest of the body empties into the right atrium through *superior and inferior vena cava*. Venous blood from coronary circulation also empties into the right atrium through *coronary sinus*. This chamber also has embedded natural pacemaker called "*Sino Atrial node" (SA node)* situated on the right upper corner near the anastamosis of superior and inferior vena cava. Superior and inferior vena cava are the inlet of this chamber whereas, the outlet is one of the atrioventricular valve called "*tricuspid valve*". It opens towards right ventricle and allows blood flow only in downward direction. The right and left atria are divided by intra-atrial septum.

Right ventricle *Origin of pulmonary circulation* is the significant aspect of function of the right ventricle. Blood enters the right ventricle from the right atria through tricuspid valve and exit to pulmonary circulation through *main pulmonary artery*; which later divides into right and left pulmonary trunk. Failure of pumping from the right ventricle results in backing up of venous circulation and clinically manifested by *jugular vein distension* and signs of portal hypertension including peripheral edema. Back flow of blood in to the right ventricle from pulmonary artery is controlled by semilunar valve called *pulmonic valve*.

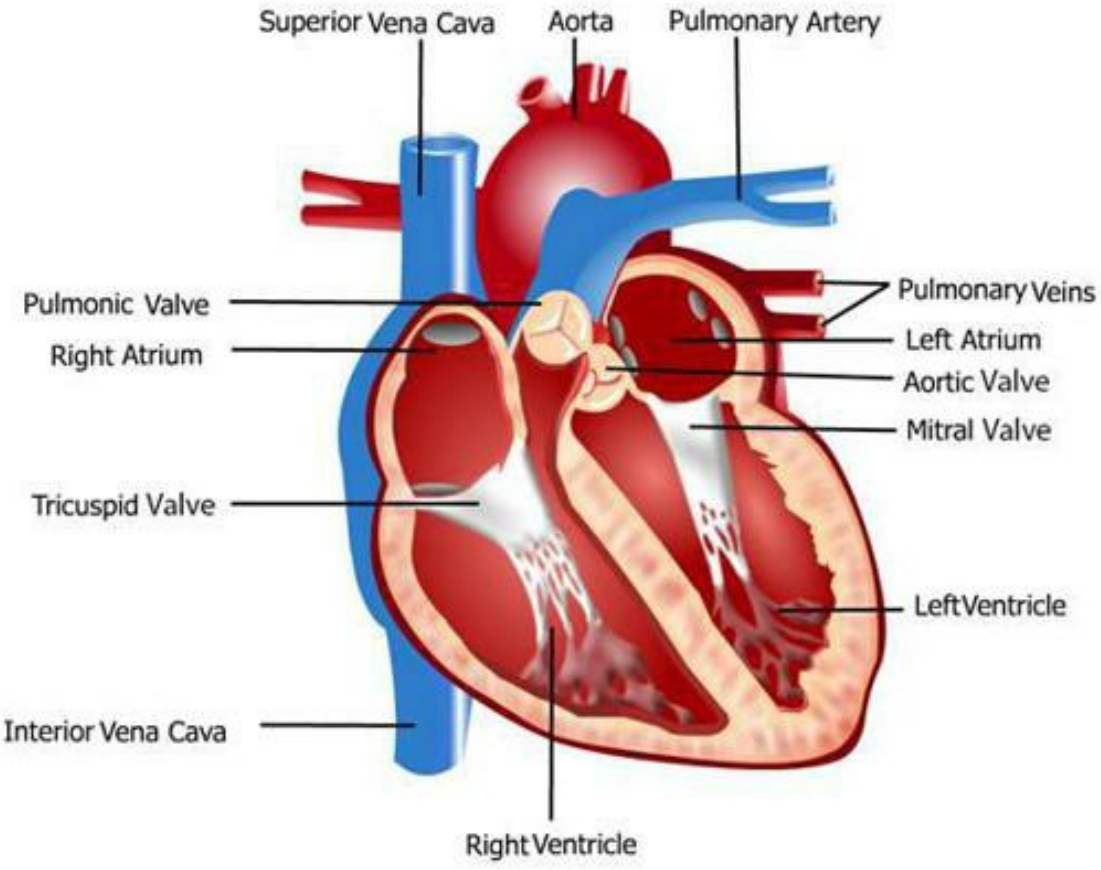


Fig 1.3 Chambers of the heart

Left atrium The left atrium receives oxygenated blood from the pulmonary circulation through two pairs of *pulmonary veins*. This oxygenated blood will get pumped into the left ventricle through bicuspid valve called "*mitral valve*". Increased blood volume and pressure within this chamber leads to stagnation of pulmonary circulation, which in turn develop pulmonary congestion.

Left ventricle It is the *most important pumping chamber* of the heart. Majority of cardiac musculature is concentrated here in order to assist in pumping blood against the high-pressure of systemic circulation. Blood from the left atrium comes to the left ventricle through *mitral valve* and exit through *aorta*. The *Aortic valve* situated in between aorta and left ventricle regulates possible back flow of blood during ventricular diastole (relaxation).

Layers of the Heart Wall

Apart from the three distinct layers of cardiac muscle, the heart is covered in a fibrous double walled sac known as *pericardium*. The surface of the pericardium attached to the chest wall is called *parietal pericardium* and the layer covering the heart is known as *visceral pericardium*. There is a small amount of pericardial fluid between these two layers, which act as a lubricant during the constant movement of heart within the sac. Any excess amount of fluid within this constricted space, either in the form of serous fluid or blood effectively obstructs

normal pumping function of ventricle and known as *pericardial effusion* and *cardiac tamponade*.

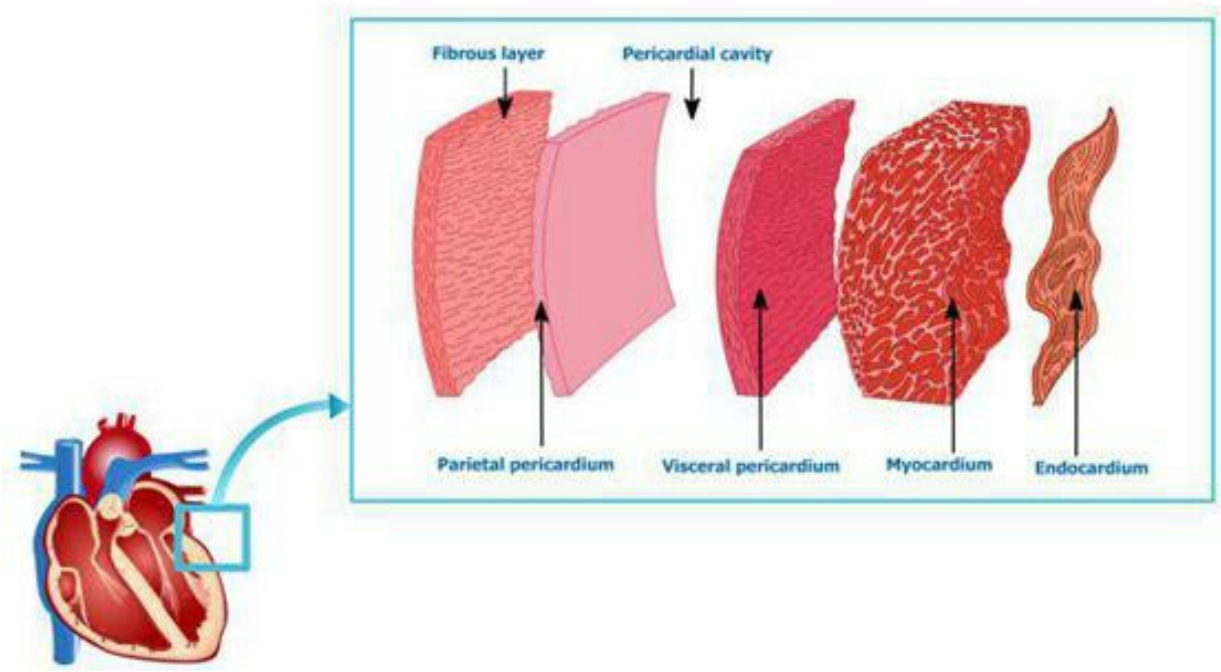


Fig 1.4 Layers of heart wall

The outermost layer of cardiac muscle is called *epicardium*. The *myocardium* is the middle muscular layer that acts as a mechanism behind pumping function of the heart. Myocardium receives blood supply from the *epicardial coronary arteries* with embedded microvasculature throughout myocardium. The innermost layer called *endocardium* and it covers the inner aspect of all the chambers and other mechanical structures within the heart like heart valves and papillary muscle.

Up to 1 lit of fluid will not produce any significant symptoms in Pericardial effusion due to the slow rate of accumulation; where as in cardiac tamponade, 100ml of fluid can compromise hemodynamics because of the fast filling within the pericardium.

Cardiac Muscle Cell

Cardiac myocytes are the building blocks of myocardium, which are striated muscles seen only in the heart. The cross-section of cardiac muscle shows thin and thick protein filaments called *actin and myosin* filaments. These proteins filaments are also known as *myofibrils*. The contracting unit of the myocardium is known as *sarcomere*, which lies

between two adjacent dark lines called *Z line* represented by the disk shaped structure at the end of actin filaments. These fibers are arranged in certain parallel patterns so that, all the fibers will depolarize when any one of them get depolarized.

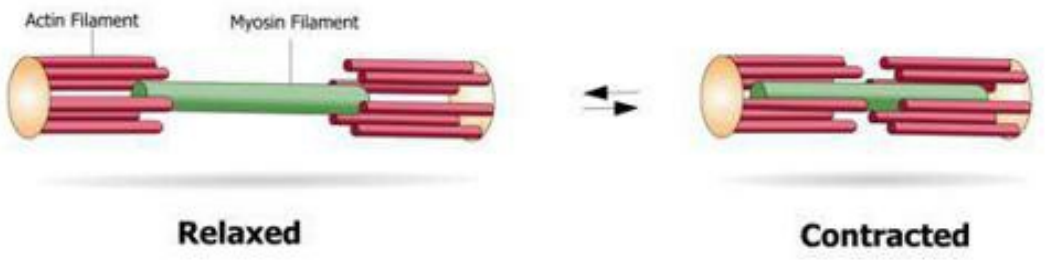


Fig 1.5 Cross-link mechanism

In the microstructure of actin filament, there are two chains of actin molecules wound about each other on a larger molecule called tropomyosin. A group of regulatory proteins called *troponin C*, *I* and *T* are spaced in regular intervals on this filament. Actin does not have intrinsic enzymatic activity like myosin; however, *in presence of calcium and ATP*, *actin can reversibly bind with myosin*. In the absence of cellular excitation, tropomyosin prevents cross-linking of actin and myosin filaments. When calcium is present, it combines with the troponin C and forms a confirmatory change for *actin myosin cross-linking*; which in turn causes contraction.

The rate and extent of actin myosin cross bridging continues until the amount of calcium ions fall below a critical level. Therefore, intra cytoplasmic calcium is a principal determinant of myocardial contractility (*ionotropic stimuli*) and is the basis of using various pharmacologic therapies such as cardiac glycosides (e.g. Digoxin), calcium gluconate, calcium channel blockers etc.

Calcium is the major electrolyte determines cardiac contractility. In the absence of calcium, myosin and actin cannot form bonds between them and therefore diminishes contraction.

Mechanism of Muscle Contraction

The contracting process of myocardial muscle fibers involve "*sliding filament model*", where the *thinner actin fibers slide into the thick myosin filaments* during cellular activation. However, because of the *tropomyosin*, which is the rod shaped protein covering all actin myosin binding sites; these filaments are not able to interact each other under normal circumstances.

During cellular activation in response to electrical impulses, *calcium ions* are released into the sarcomere. In the presence of calcium ions, the *troponin*, which is another structural protein that binds tropomyosin to myosin combines with calcium and release tropomyosin bond with myosin receptor sites. This exposes actin and myosin binding sites and promotes both fibers to crawl along each other and facilitate contraction as shown in the picture.

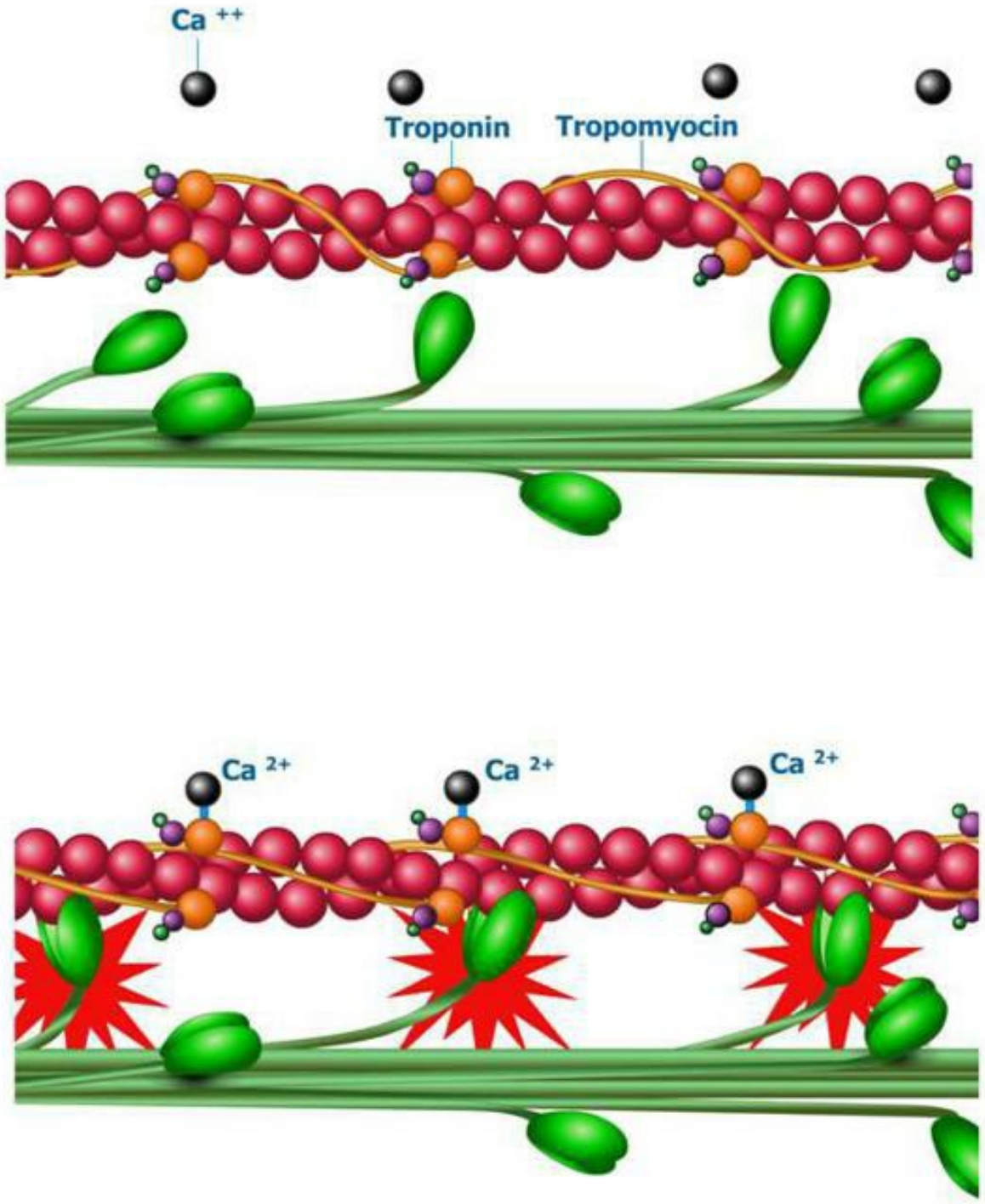


Fig 1.5 (a) Actin and myosin sliding filament action

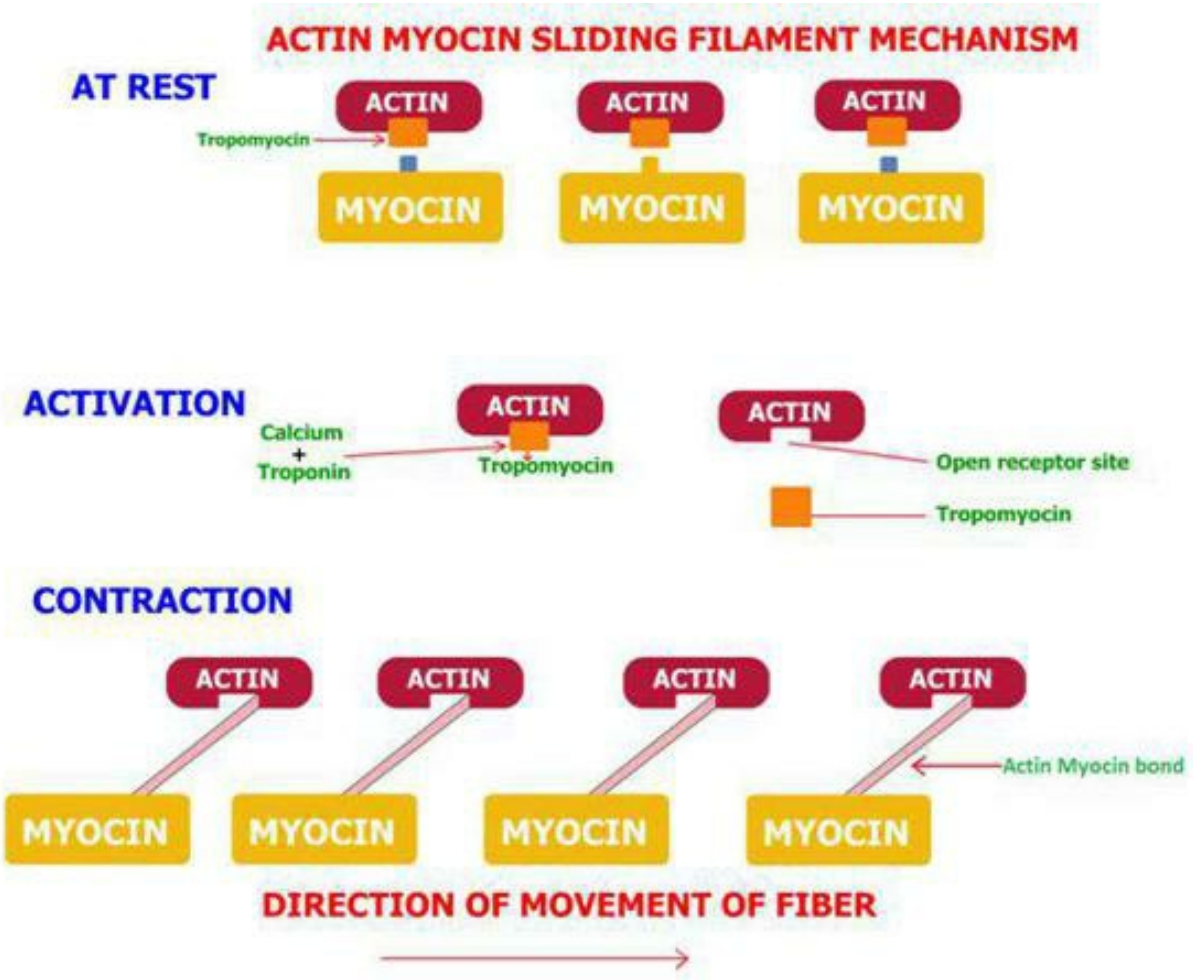


Fig 1.5 (b) Diagrammatic representation of sliding filament theory

In simple terms, consider actin filaments as a spoon and myosin filaments as a can of food. The lid of the can represent tropomyosin, which under normal circumstances prevent the consumer from using the spoon to eat from the can. We have an electric can opener where Troponin is the can opener and calcium ions represent electricity; both together allow us to open the can and access the product. The food will give the consumer energy to work that is essentially the muscle contraction.

Cardiac Cycle

Cardiac cycle represent the series of events happen with each heartbeat. This process involves systematic filling, open and closure of valves, contraction and expulsion of blood from various chambers etc. Synchronous contraction between heart chambers ensures smooth flow of blood through systemic circulation. Events during cardiac cycle can be broadly divided into *ventricular systole* (contraction), *ventricular diastole* (relaxation), and *atrial systole* (contraction). Fig 1.7 describes various events happen during cardiac cycle.

Determinants of Cardiac Performance

The performance of cardiac muscle depends on three main factors such as the amount of muscle stretch at the beginning of contraction (*preload*), the pressure against which it has to pump (*afterload*) and the force of contraction (*contractility*).

Preload Consider each cardiac muscle as an elastic band. The force of contraction of an elastic band after stretching directly relates to the length of band while it is stretched. The same is true with cardiac muscle. The stretching force of cardiac muscle before contraction corresponds to the volume of blood within the ventricle right before systole (*end diastolic volume/pressure* (*EDP/EDV*)). *The higher the volume*, *stronger the contraction will be*. This relationship is called **Frank Starling's law**. In short, right ventricular preload has a direct relation to the amount of blood coming to the right side of the heart and that is *venous return*.

Afterload: Afterload is the amount of *resistance against which the left ventricle has to pump*. Since left ventricle is pumping blood in to the aorta and systemic circulation, afterload is greatly influenced by systemic vascular resistance (SVR) and BP.

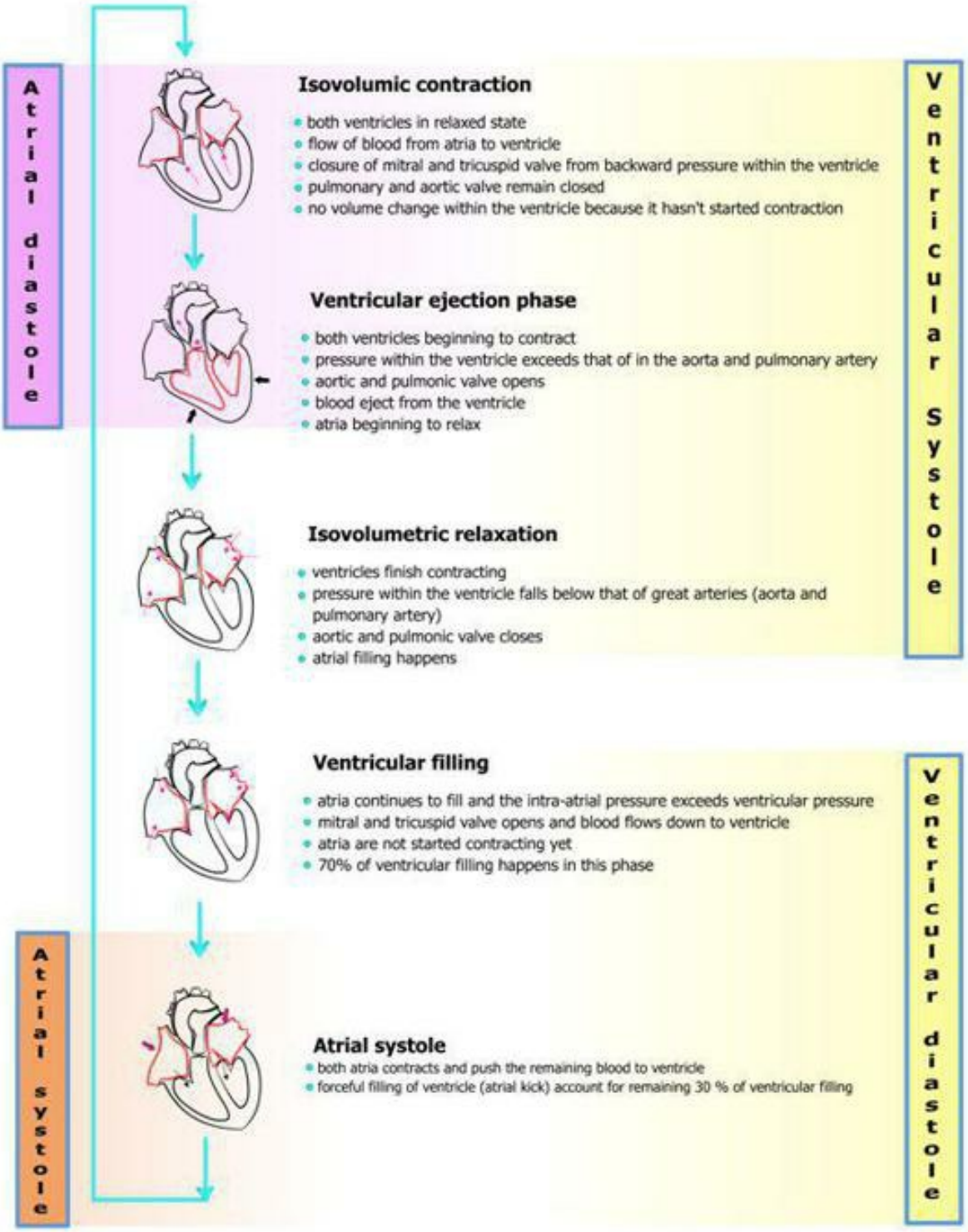


Fig 1.7 Events during cardiac cycle

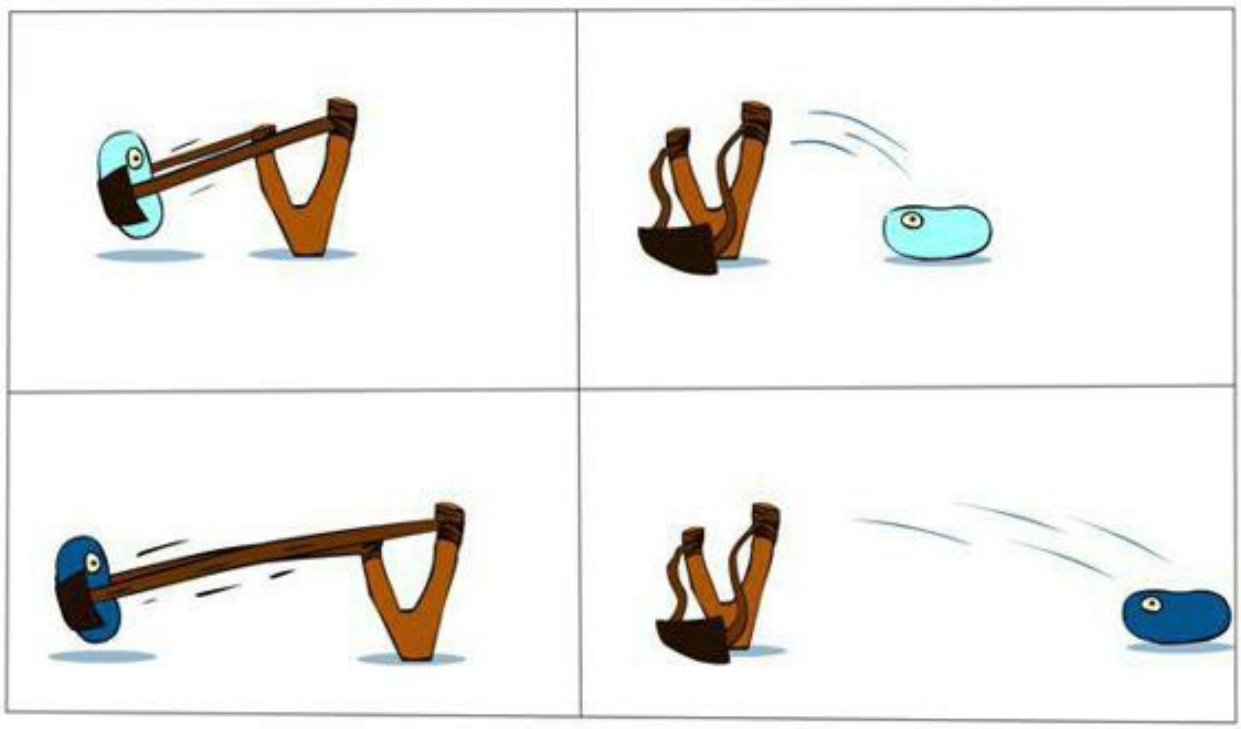


Fig 1.8 A Preload (Frank Starling's Law; *'The greater the stretch of cardiac muscles, the stronger the contraction*' and resultant force of pumping)

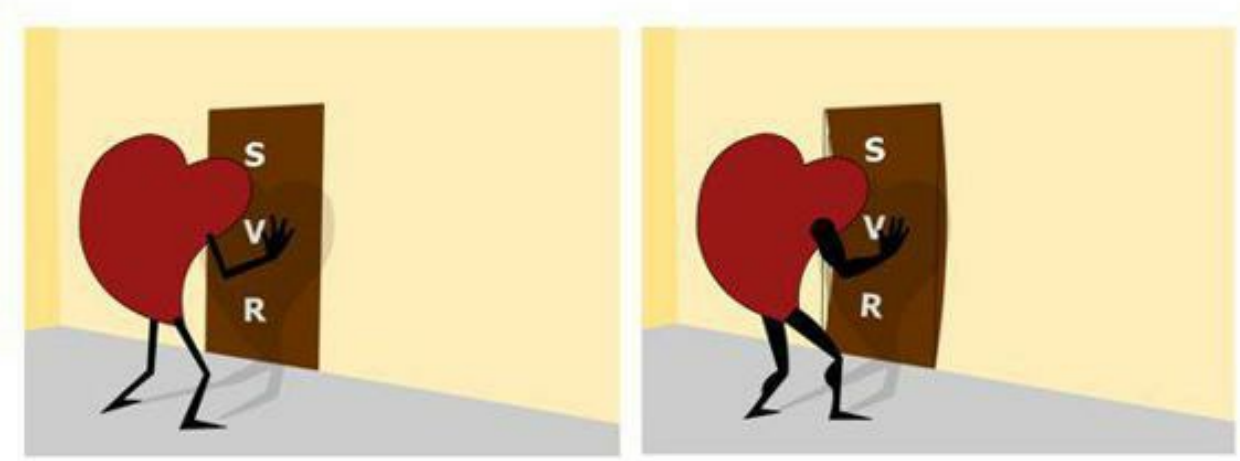


Fig 1.8 B Afterload (Shown as the Systemic Vascular Resistance (SVR) being the force against which the heart pumps blood)

- *Preload reducing pharmacologic agents control venous return to the heart mostly by peripheral venous dilatation.*
- *Afterload reducing agents generally cause peripheral arterial dilation and reduce systemic blood pressure.*
- *Right ventricle also has afterload, which is determined by pulmonary artery*

Coronary Circulation

The heart, being the major pumping organ of the body supplying blood to all of the organs, it itself gets blood supply through a system of circulation called ***coronary circulation***. The coronary arteries supply blood to the myocardium during *diastole*. There are two main coronary artery systems such as ***Right coronary artery*** (**RCA**) and ***Left main coronary artery*** (**LMCA**). The left main coronary artery supplies two third of the cardiac blood supply. A short while after its origin from the aorta near aortic cusp, left main coronary artery divides into the ***Left anterior descending artery*** (**LAD**) and ***Left circumflex artery*** (**LCx**).

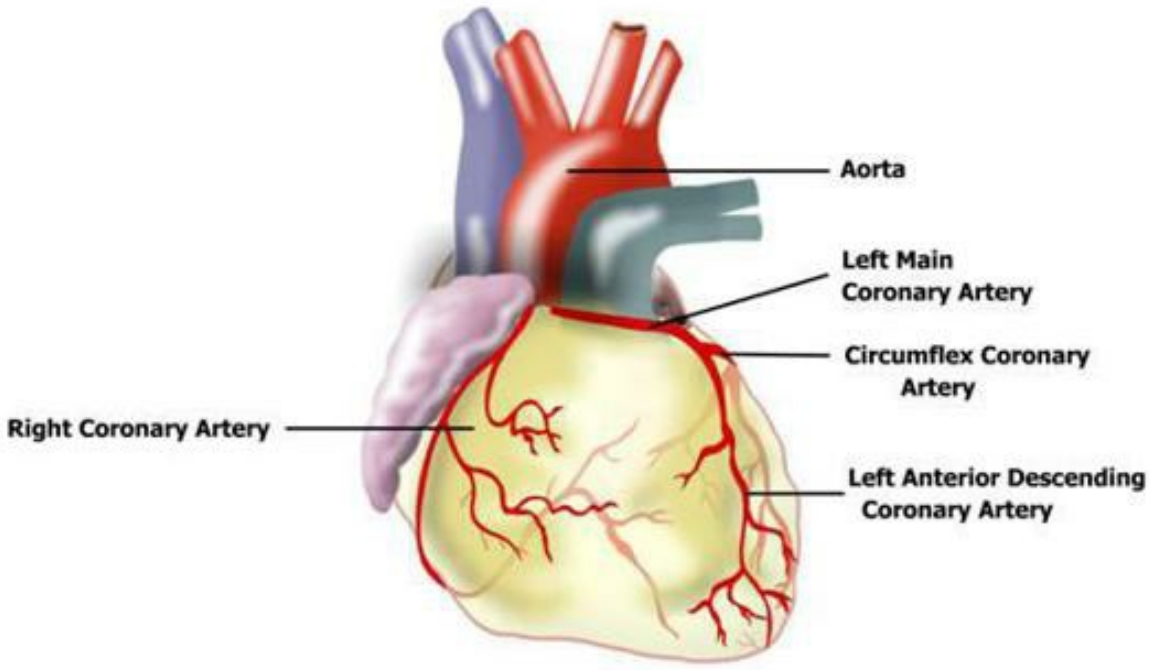


Fig 1.9 Coronary arteries

Left anterior descending artery (LAD) LAD mainly supplies the *anterior wall of left ventricular myocardium*. It has smaller branches called *Septal perforators* and main branches known as *Diagonal arteries*. Being the vital part of left ventricle's blood supply, any occlusion of left anterior descending artery may directly affect the pumping function of left ventricle.

Left circumflex artery (LCx) LCx supplies the *lateral and sometimes parts of posterior wall* of the heart. Left circumflex artery originates *obtuse marginal branches*.

Right coronary artery (RCA) this coronary artery feeds the *right ventricle and inferior wall of left ventricle*. Tiny branches of right coronary artery also supply *SA node and AV node*.

Right coronary artery has a direct effect on heart rate in terms of natural pace making function. This is the reason why patients with the right coronary artery occlusion and resulting inferior wall myocardial infarction may have bradycardia or various degree of heart block.

Nervous System Control of the Heart Rate

The heart is mainly controlled by *autonomic nervous system*. Both divisions of autonomic nervous system such as sympathetic and parasympathetic have effect on heart rate; however, *parasympathetic tone dominates* in healthy young individuals.

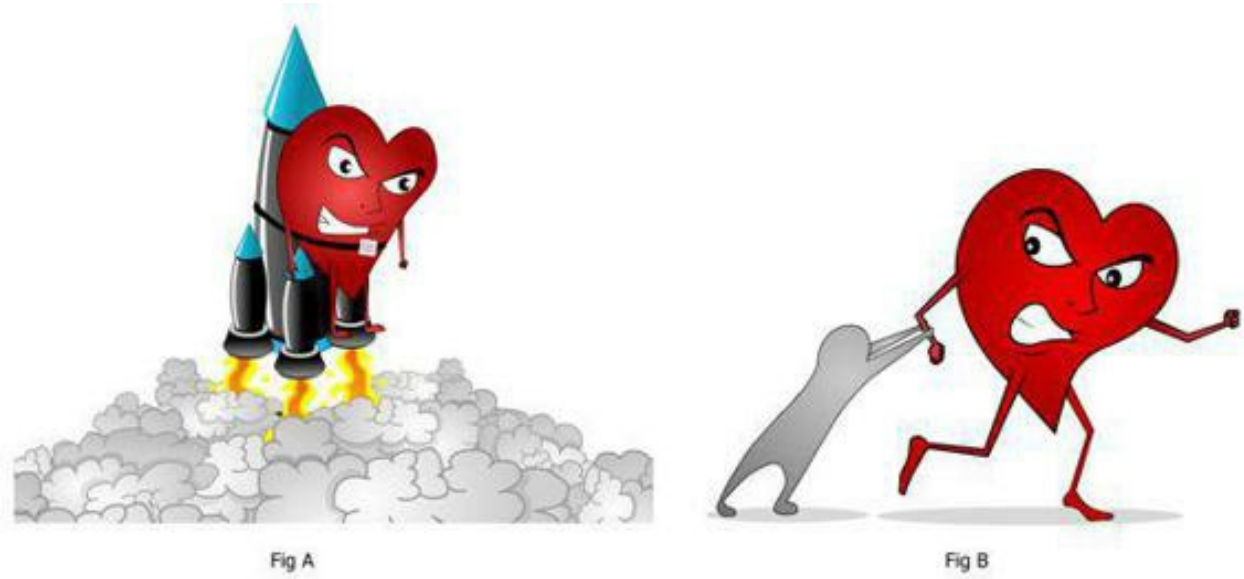


Fig 1.10 Sympathetic (Fig A) and parasympathetic (Fig B) actions on the heart

*Sympathetic system **increases** automaticity whereas parasympathetic system **inhibits**!!!*

In humans, parasympathetic nervous system control is mainly through **vagus nerve** that originates from the Medulla Oblongata and travelled down through the neck near carotid sinus

and end at vagal ganglionic cells located around SA and AV nodes. Therefore, stimulations of vagal nerve either by *carotid sinus massage* or *Valsalva maneuver* increase parasympathetic influence on SA node and thereby *reduce heart rate*. **Acetylcholine** is the major parasympathetic neurotransmitter for the heart. Therefore, SA and AV nodes are rich in an enzyme called **cholinesterase** that aid in rapid degradation of acetylcholine. This natural mechanism ensures a rapid decay of any vagal stimulation in the heart.

Rapid beat-to-beat control of heart by vagal response is clinically manifested in patients with sinus arrhythmia. In these patients, even though they remain in sinus rhythm during entire sleep, heart rate varies with inspiration and expiration. It is thought that during expiration; because of the natural vagal stimulation, the heart rate slows down. Even though there is norepinephrine release as part of sympathetic stimulations during inspiration, it does not provide immediate effect on heart rate as the acetylcholine.

Sympathetic innervations of heart originate from lower cervical and higher thoracic part of spinal column. The chemical mediator for sympathetic response is **Norepinephrine**, which has a *slower onset and relatively slow degradation* compared to that of acetylcholine. Therefore, sympathetic stimulation of the heart does not provide a beat-to-beat control as in parasympathetic stimulation; however, the effect lasts longer since Norepinephrine has to be washed away by bloodstream.

Other forms of nervous system control on the heart come from various chemical and pressure receptors. For example, *baroreceptors* located in the aortic arch and carotid sinus mediates a reduction in heart rate when activated. Peripheral chemoreceptors when activated can have vagal response on cardiac activity. A distinct set of sensory receptors located on the endocardial surface of the ventricles is thought to be involved in vasovagal syncope. These receptors are stimulated by reduced ventricular filling volume coupled with vigorous ventricular contraction.

For example, in a person who is standing from a sitting position will have a sudden change in blood volume coming to the ventricle because of the gravitational pooling in abdomen and lower extremities; leads to a reduction in cardiac output and enhanced sympathetic response. This sympathetic response in turn increase ventricular contraction, which stimulate ventricular receptors leading to profound vagal mediated bradycardia and peripheral arterial vasodilation.

Points to Remember!!!

· Four pumping chambers of the heart along with valves perform systemic and pulmonary circulation.
· Systematic and rhythmic contraction and relaxation of these chambers maintain hemodynamics.
· Epicardium, myocardium and endocardium are the three layers of heart wall.
· Within mediastinum, heart is covered with fibrous layer called pericardium with visceral layer attached to heart wall and parietal layer to the inner aspect of chest wall.
· Cardiac contraction is materialized by cardiac myofibrils known as Actin and Myosin filaments.
· Calcium is the major electrolyte determinant of cardiac contraction.
· Cardiac performance is determined by preload, afterload and cardiac contractility.
· Preload is the volume of blood within ventricle right before systole (contraction) and is also known as End diastolic volume (EDV).
· Frank Sterling's law states 'greater the stretch of cardiac muscles before contraction, stronger the contraction will be'.
· Afterload is the amount of resistance against which the ventricle has to pump.
· Three main coronary arteries are Right coronary artery (RCA), Left anterior descending artery (LAD) and left circumflex artery (LCx).
· LAD supplies anterior wall of left ventricle and part of inter ventricular septum through diagonal branches and septal perforators.
· LCx artery supplies lateral and sometimes part of posterior wall of the heart.
· Right coronary artery supplies inferior wall of heart and mainly structures like SA node and right ventricle.
· Parasympathetic nervous system has more effect on heart rate.
· Parasympathetic system through neurotransmitter called acetylcholine reduces heart rate.
· Vagal nerve is the major parasympathetic control system of the heart.
· Sympathetic nervous system has relatively slow effect through Norepinephrine to the heart compared to that of acetylcholine and is the reason behind sinus arrhythmia.

Test Your Understanding

1. Which of the following represent the inflow valve between right atria atrium and ventricle?

A Semilunar valves

B Tricuspid valve

C Mitral valve

D Aortic valve

2. Which of the following structure is not part of pulmonary circulation?

A Inferior vena cava

B Pulmonary Artery

C Right pulmonary vein

D Left lung

3.Venous return of myocardial tissue involve_____?

A Superior vena cava

B Pulmonary artery

C Coronary sinus

D Pulmonary vein

4. Which of the following clinical symptom is the hallmark of right ventricular failure?

A Palpitation

B Jugular vein distention

C Dizziness

D Right-sided chest pain

5. The layer of pericardium attached to the chest wall is called_____?

A Visceral pericardium

B Parietal pericardium

C Endocardium

D Myofibrils

6. Which of the following represent the most critical electrolyte in contractility of myocardial cells?

A Sodium

B Magnesium.

C Calcium

D Phosphorous

7. Majority of ventricular filling happens during _____?

A Isovolumetric contraction phase

B Ventricular filling phase

C Ventricular ejection phase

D Atrial systole

8. Which of the following event during cardiac cycle corresponds to 'atrial kick'?

A Atrial systole

B Isovolumic relaxation phase

C Ventricular ejection phase

D Ventricular filling phase

9.Which of the following is true regarding Franks Sterling's law?

A The longer the stretch of cardiac muscle before contraction, the stronger the contraction will be

B The longer the stretch of muscles before contraction, the weaker the contraction will be

C The shorter the length of muscles before contraction, the stronger the contraction

D Contractions are not at all related to the length of muscle stretch

10. Which of the following coronary artery supplies lateral wall of the heart?

A Posterior descending artery

B Left circumflex artery

C Septal perforators

D Right coronary artery

11. The neurotransmitter believed to have the highest effect on the heart is ___?

A Norepinephrine

B Acetylcholine

C GABA

D Dopamine

12. Which of the following neurologic system has most influence on the heart?

A Sympathetic nervous system

B Parasympathetic nervous system

C Autonomic nervous system

D Central nervous system

13. Which of the following accurately represent function of sympathetic nervous system?

A Slows things down

B Flight fight response

C Vagal response on the heart

D Cause peripheral vasodilation

14. Sinus arrhythmia originates from_____?

A Parasympathetic nervous system

B Renin Angiotensin system

C Tropomyosin Calcium complex

D Beta 1 adrenergic receptors

15. Which of the following represent the definition of ' afterload'?

A It corresponds to the stretch of cardiac muscle at the end of diastole

B It represents the pressure within the ventricle at the beginning of systole

C It is the pressure against which heart has to pump

D It has no relation with peripheral vascular resistance

Answers

1. B Tricuspid valve
2. A Inferior vena cava
3. C Coronary sinus
4. B Jugular vein distention
5. B Parietal pericardium

6. C Calcium

7. B Ventricular filling phase

8. A Atrial systole

9. A The longer the stretch of cardiac muscle before contraction, the stronger the contraction will be.

10. B Left circumflex artery

11. B Acetylcholine

12. B Parasympathetic nervous system

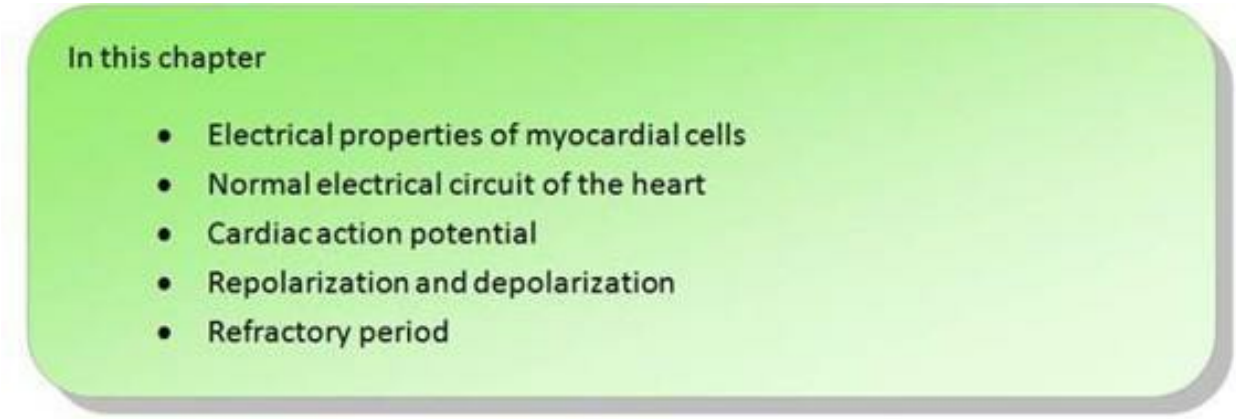
13. B Flight fight response

14. A Parasympathetic nervous system

15. C it is the pressure against which heart has to pump

Chapter 2

Physiology of Cardiac Contraction



The human heart is designed to perform its mechanical pumping function relentlessly through a combination of complex chemical and electrical pathways. In order to provide an appropriate response to electrical stimuli, myocardial cells process charged particles called *ions*. The movements of ions in and out of the cells essentially produce a difference in electrical potential. Specific channels that respond to various stimuli through active and passive mechanisms control this ionic movement.

Properties of Cardiac Cells

Even though autonomic nervous system has effect on cardiac function, the heart doesn't

require nervous control for its basic functions. The major cellular properties that distinguish cardiac cells from other tissues are their *contractility*, *conductivity*, *automaticity* and *rhythmicity*.

Contractility is the property by which *cardiac tissue can contract in response to electrical stimuli*. ***Conductivity*** is the ability of myocardial cells to *conduct electricity after stimulation*. Automaticity and rhythmicity are the major properties that allow perfused heart to beat even when it is completely removed from the body. ***Automaticity*** is the *ability to initiate its own beats* and ***rhythmicity*** is the *capability of maintaining regularity of such pace making activity*.

The capacity of individual cardiac cells to generate, maintain and respond to electrical stimuli serves as a great backup plan in the case of extreme emergencies such as loss of natural pacemaker function. However, the same applies to inappropriate generation, conduction and response of impulses leading to various types of arrhythmias.

By nature, myocardial cells are biologically programmed to respond to the fastest and strongest stimuli available to them. This explains the response of heart towards faster ectopic beats even though the SA node is producing impulses at lower rates.

Conduction System of the Heart

Natural electrical circuit of the heart originates from **SA node** and spreads impulse through the surface of atria and down through pathways to the atrioventricular node. In response to an atrial impulse, the chamber contracts and pumps blood through the atrioventricular valves to the lower ventricles. The impulse travels between right and left atria through *Inter atrial pathway* called **Backmann 's bundle**. From the SA node, there are three internodal pathways called anterior, middle (Wenckebach) and posterior (Thorel's) tracts exist between SA and AV nodes.

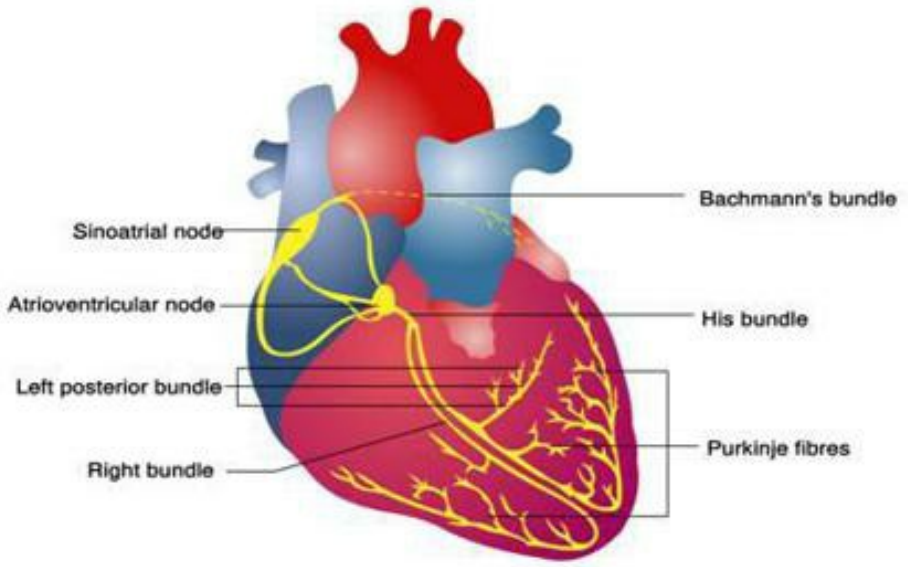


Fig 2.1 Conduction system of the heart

The impulse that reaches atrioventricular node slows down for a time period before spreading downstream. This **AV nodal delay** ensures a *synchronized pumping between upper and lower chambers* of the heart. From the AV node, impulse travels down through pathway known as **Bundle of his**, that further subdivides to right and left nerve bundles. The left bundle branch again subdivides into *anterior* and a *thick posterior division* called **fascicles**. The *right and two subdivisions of left bundle* give rise to complex network of **Purkinje fibers**.

Being the broadest cells within the heart, Purkinje fibers ensures impulse transfer at a higher velocity; which is key in immediate activation of entire ventricular myocardium in response to electrical stimuli. These fibers have a long refractory period that prevents excitation of ventricular tissue in response to inappropriate AV nodal conduction like in the case of atrial fibrillation.

Inherent rate of pacemaker cells

SA node	60 to 100 bpm
AV node	40 to 60 bpm
Ventricle	20 to 40 bpm

The SA node is the natural pacemaker of the heart. In the event of its failure to initiate an impulse, downstream cells start generating electrical impulses in order to maintain cardiac contraction. For example, Junctional rhythm that originates from AV node usually runs at 40 to 60 bpm. If the rate of junctional rhythm is more than 60 bpm, it is called Accelerated junctional rhythm. Similarly, during third-degree heart block or AV dissociation, atrial rate will be somewhere in the order of 60 to 100 beats per minute; however, ventricular rate usually is in 30 to 40 range.

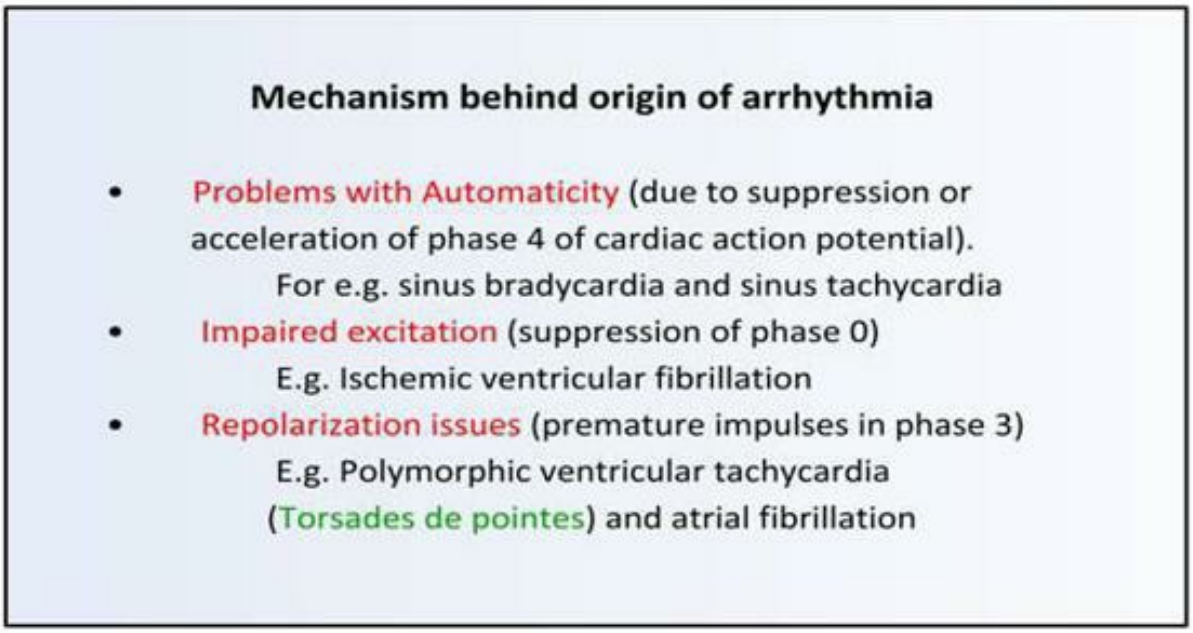
Box 2.1 Inherent pacemaker rates at various parts of the heart

The cells in all areas except SA node and AV node polarizes through **fast pathway** and that ensures rapid conduction of impulse and subsequent repolarization of these cells. However, the cells in SA node take **calcium mediated slow pathway** and are responsible for slow and rhythmic activation of impulses from SA node. These pathways are explained later in this chapter. Various zones within AV node are having *variable conduction velocity* and they help in slowing down impulses (*AV nodal delay*). This process is essential for synchronized electromechanical function of the heart.

In reality, electrical impulse spread through left bundle faster than right bundle. This ensures timely spread of electrical impulses through thick cardiac musculature of left ventricle for synchronized ventricular contraction.

Physiology of Cardiac Contraction

As impulses transmitted down through the myocardial cells, they undergo cycles of **depolarization** and **repolarization**. Even though the amount of ionized calcium is the main determinant of cardiac contractility, movement of other ions such as sodium and potassium in and out of the cells are needed to initiate an impulse. The movement of these charged particles creates a difference in electrical charge or potential known as *electrical gradient* across the semi permeable cell wall. The *transport of charged particles through active or passive techniques* determines the *nature*, *regularity* and other aspects of myocardial contraction.



Box 2.2 Mechanism behind origin of arrhythmias

There are mainly **five phases** of ionic moments during one contraction process of cardiac cell cycle and is represented by so-called **cardiac action potential curve** as shown in the picture (Fig 2.2). Various phases of cardiac action potential are associated with *changes in permeability of cell membrane to sodium*, *potassium* and *calcium ions*. There are specific channels for individual ions to go in and out of the cells. The movement of these ions across cell membrane is controlled by factors such as *electrical* and *concentration gradient*.

In the resting state, the cell membrane is relatively permeable to potassium ions compared to sodium and calcium. Therefore, there are *more potassium ions within the cell* compared to surroundings during resting state. However, because of the sheer number of sodium ions present outside the cell compared to the number of potassium ions within, *the interior of the cell has a net negative charge* even though both of these particles are positively charged ions.

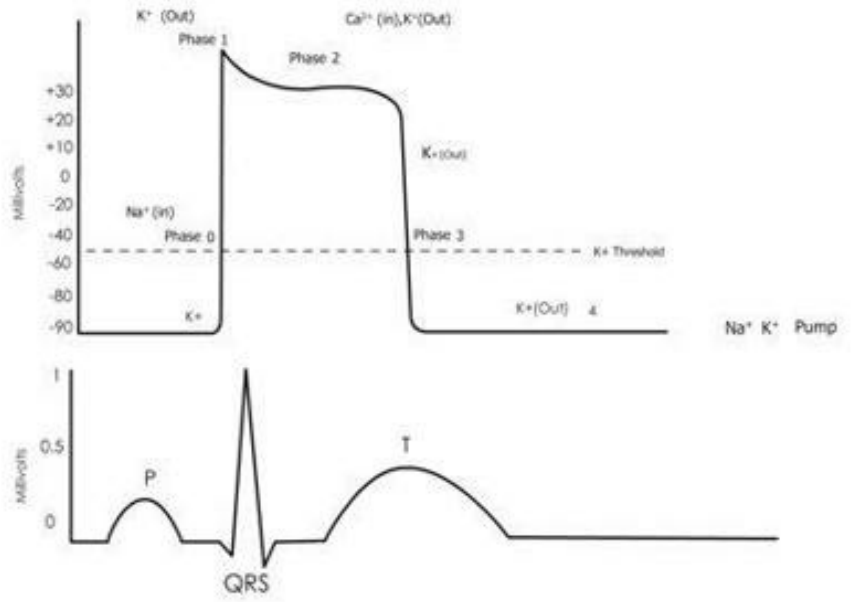


Fig 2.2 Action potential of ventricular myocytes

Interestingly, the speed of cardiac action potential varies from some areas of the heart to other. There are two types of cardiac action potential known as *fast response* and *slow response* channels. For example, myocytes in *atrial*, ventricular and lower conduction pathways such as *Purkinje fibers* are *fast response type* and therefore can propagate the impulse at *higher speeds* and respond accordingly. However, the cells in *Sino atrial node* (*S A node*) *and atrioventricular node* (*AV node*) *are slow response* type and therefore can effectively *control the rate of cardiac activation* at physiologically appropriate levels.

The innate property of *response time difference* between these two distinct areas ensures function of SA node as a natural pacemaker producing rhythmic impulses; which then spread across myocardium at a faster pace. At the AV node, the impulses again slows down and then propagate downstream and thereby provide enough time delay for a sequential contraction of atria followed by ventricles.

Cardiac Action Potential

Cardiac action potential represents the series of ionic moments within myocardial cells and resulting electrical current that trigger muscle contraction. Various myocardial cells have slightly different action potential in the form of slow and fast response pathways as mentioned earlier. For the sake of simplicity, we will analyze action potential of a ventricular cardiomyocyte, which is the common myocardial cell.

The basic principle of action potential is that the cell always tries to come back to its resting electrical state even after disturbance in electrical charge.

Phase 0

Any electrical stimulus that abruptly changes the resting membrane potential to the threshold level generates an *action potential*. At this phase, there is a sudden *influx of sodium ions* into the cell from outside through specific channels called **fast sodium channels**. This accounts for the steep *upslope of phase 0* in the action potential representation curve. Now, because of the large influx of sodium ions that are positively charged, it effectively neutralizes existing negative charge within the cell. Consequently, the interior of the cell becomes more and more positively charged.

Phase 1

This is represented by the area between the *end of upstroke* (*Phase 0*) *and the beginning of plateau* in the action potential representation. During this phase, activation of potassium channels lead to *outward flow of potassium* ions from the cells. This process results in a partial and brief period of repolarization of the cell.

Phase 2

Calcium ions are the main players in this phase of cardiac action potential. In this phase the *calcium ions enters through calcium channels*, which are much *slower than the fast sodium channels*. Simultaneous *outflow of potassium ions* continue from phase 1. This inward and outward movement of positively charged ions effectively counterbalance electrical gradient during this phase.

Slow response cells in the heart such as SA node and AV junction are depolarized not through the fast sodium channels, but through slow calcium channels. The repolarization is achieved by inactivation of calcium channels and increased outflow of potassium during phase 3.

Phase 3

The process of final repolarization starts when the amount of *potassium outflow exceeds influx of calcium ions*. Outward movement of potassium is highest during this phase and it results in bringing electro negativity within the cell. Towards the end of phase 3, sodium and calcium channels are completely closed.

Phase 4

During this phase, the excess of sodium that got into the cell during initial part of cardiac action potential is eliminated by **active sodium potassium ATPase pump**. This leads to increasing negativity within the cell. During this active sodium elimination process, three *sodium ions are exchanged for two potassium ions* within the cell and hence, more intracellular potassium than sodium as in the resting state. Excess of calcium also expelled through **active calcium pump** during this phase. Altogether, the cell repolarizes back to

baseline and another action potential cycle begins.

Various ionic movements during cardiac action potential are summarized in the table below.

Intracellular space					
	Phase 0	Phase 1	Phase 2	Phase 3	Phase 4
Sodium	Large amount goes in				3 sodium ions goes out*
Potassium		Going out	Going out	Going out	2 potassium ions goes in*
Calcium			Going in	Going in	Going out

*sodium potassium pumb

Table 2.1 Ionic movements in cardiac action potential

Various drugs and sympathetic neurotransmitters such as beta-receptor agonist (Isoproterenol) and norepinephrine enhance calcium in flow; however, parasympathetic neurotransmitter such as acetylcholine may reduce calcium conductance. These properties are put to use in calcium channel blockers like Verapamil and Diltiazem.

Due to the blockade of calcium channels, these agents effectively reduce the duration of phase 2 of cardiac action potential and diminish the strength of cardiac contraction. This in turn depresses vascular muscle contraction and thereby produces generalized vasodilatation. Therefore, these agents are called afterload reductors.

Refractory Period

During each cycle of cardiac action potential, **Refractory period** is a certain timeframe in which the cells are *not capable of* responding to another electrical stimuli. There are two types of refractory period known as **Absolute** refractory period and **Relative** refractory period.

Absolute refractory period is the interval from the *beginning of phase 0 of action potential to a point in phase 3* at which the depolarization has reached about -50 mV. During the rest of phase 3, a subsequent cardiac action potential *may be provoked if the impulse is strong enough*. Patients with premature electrical beats may have clinical consequences if the impulse originates early in phase 3. During this moment, the conduction of premature impulse from the *site of origin will be slow* and therefore *higher chance of re-entry* that leads to dangerous arrhythmias like **ventricular fibrillation**.

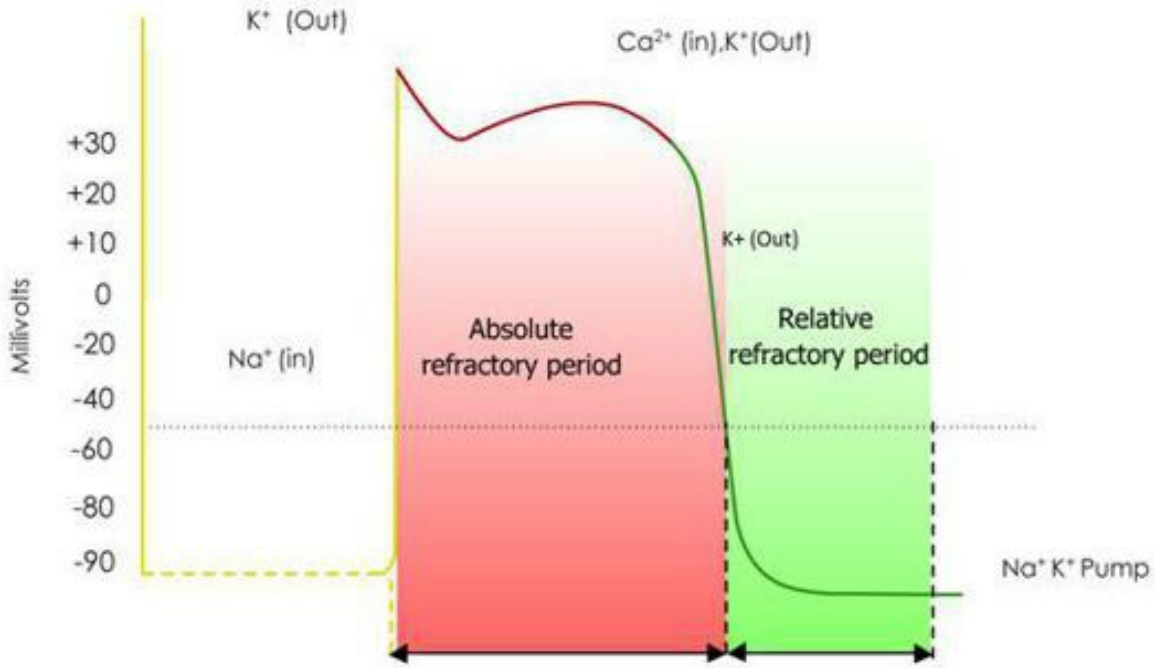


Fig 2.3 Absolute refractory period and relative refractory period

Points to Remember!!!

· Major properties of cardiac cells are contractility, conductivity, automaticity and rhythmicity.

· Automaticity is the ability to generate its own impulse and conductivity is the ability to conduct those impulses. Contractility is the property of contraction in response to those impulses and rhythmicity is the ability to maintain rhythmic contractions.

· Myocardial cells have the inherent property to respond to the fastest stimuli available to them.

· SA node, AV node, Bundle of his and Purkinjee fibers grossly constitute the conduction system of the heart.

· Inherent rate of SA node is 60-100 bpm and the rate of impulse production progressively slows down in cells down the line within conduction system.

· Inherent pacemaker rate of junctional tissue is 40-60 bpm and that of ventricle is 20-40 bpm.

· When a rhythm exceeds its inherent impulse production rate, it is called ‘accelerated rhythm'.

· Cardiac action potential curve represents the electrochemical events during myocardial activity.

· Movement of sodium in to the cells represents upstroke or phase 0 of cardiac action potential.

· Outward flow of potassium ions constitutes Phase 1.

· Inflow of calcium ions through slow sodium channels corresponds to Phase 2 of

action potential curve.

· In phase 3, continuing outward flow of potassium exceeds inflow of calcium.

· In phase 4, active sodium potassium pumps and calcium pumps move ions around, resulting higher concentration of potassium within the cell as in the beginning of phase 0.

· Refractory period is the timeframe within action potential curve where the cell is not capable of responding to another electrical stimuli.

· Absolute refractory period is the time between Phase 0 to a point in Phase 3 where the tissue is incapable of depolarization no matter how strong the impulse is.

· Relative refractory period is the time in which a sufficiently stronger stimulus can produce a premature impulse.

Test Your Understanding

· 1. Which of the following represent the natural properties of myocardial cell that aids in generation of electrical impulses?

A Conductivity
B Contractility
C Automaticity
D Refractory period

· 2. Which of the following statement is true regarding the nature of cardiac cells?

A Myocardial cells respond to the strongest and fastest stimuli available to them
B Myocardial cells respond to the weakest and slowest stimuli available to them
C Myocardial cells do not respond to electrical stimuli other than from SA node
D Myocardial cells only respond to electrical stimuli from SA node no matter how fast or slow they are

· 3. Which of the following structure is part of conduction system of the heart?

A Aortic annulus
B Atrioventricular node
C Mitral valve
D Left atrial appendage

· 4. Inherent pacemaker rate of ventricular tissue under normal circumstances is _____?

A 150-150 bpm
B 45-60 bpm
C 20-40 bpm
D P0-100 bpm

· 5. Which one of the following physiologic mechanism ensures a synchronized atrio ventricular contraction?

A Phase 0 of action potential
B AV nodal delay
C Presence of calcium
D Slow response channels within SA node

· 6. Which of the following phase of cardiac action potential corresponds with inward movement of sodium to the cell?

A Phase 3
B Phase 1
C Phase 4
D Phase 0

· 7. Which of the following statement is true regarding electrical charge at resting cardiac cell?

A There are more negatively charged particles outside the cell compared to intracellular fluid
B There are more positively charged particles inside the cell compared to extracellular fluid
C There are more potassium ions outside the cell compared to sodium
D There are more sodium ions outside compared to intracellular potassium

· 8. The net electrical charge of intracellular fluid is _____?

A Positive
B Negative
C Neutral
D Unable to determine

· 9. Which of the following phase of cardiac cycle involves major action of sodium-potassium pump?

A Phase 1
B Phase 2
C Phase 0
D Phase 4

· 10. Which of the following statement is true regarding absolute refractory period?

A Absolute refractory period represent the peak point of QRS complex
B This is the time when an external impulse can create cardiac action potential
C This is a time when the cell is incapable of responding to any external stimuli
D This is the time frame in which heart rest between contractions

· **Answers**

1. C Automaticity
2. A Myocardial cells respond to the strongest and fastest stimuli

available to them

3. B Atrioventricular node
4. C 20-40 bpm
5. B AV nodal delay
6. D Phase 0
7. D There are more sodium ions outside compared to intracellular potassium
8. B Negative
9. D Phase 4
10. C This is a time when the cell is incapable of responding to any external stimuli

Chapter 3

Basics of EKG

The **e**lectrocardiogram is a graphical representation of electrical activity of the heart. Devised in the early part of 20th-century, EKG became an integral part of screening, diagnosis and management of cardiac diseases in medicine. Each **individual leads** in the EKG shows electrical activity between two different parts of the body. This minute electrical activity is measured, magnified and graphically represented in various forms in electrocardiogram. The heart being a three-dimensional structure involving three planes, it needs more than one view to assess activity along all surfaces. This is how EKG with more than one lead has evolved.

Most commonly EKG can be single lead, two leads, three leads, six leads and 12 leads depending on the number of leads in use. In a *12 lead EKG* we're looking at the heart from *12 different views* such as six in vertical and other six in horizontal direction. These views provide most comprehensive outlook of electrical activity and is especially helpful in

diagnosing events involving a localized area of the heart. A 12 lead EKG provides valuable information regarding anatomical orientation of the heart, chamber size, influence of drugs, some electrolyte imbalance, presence and extent of ischemic changes, abnormal conduction pathways etc. Considering all these benefits, it is impressive to see how much vital health information can be provided through this simple, least expensive and probably most accessible diagnostic exam which hasn't changed much over last 100 years.

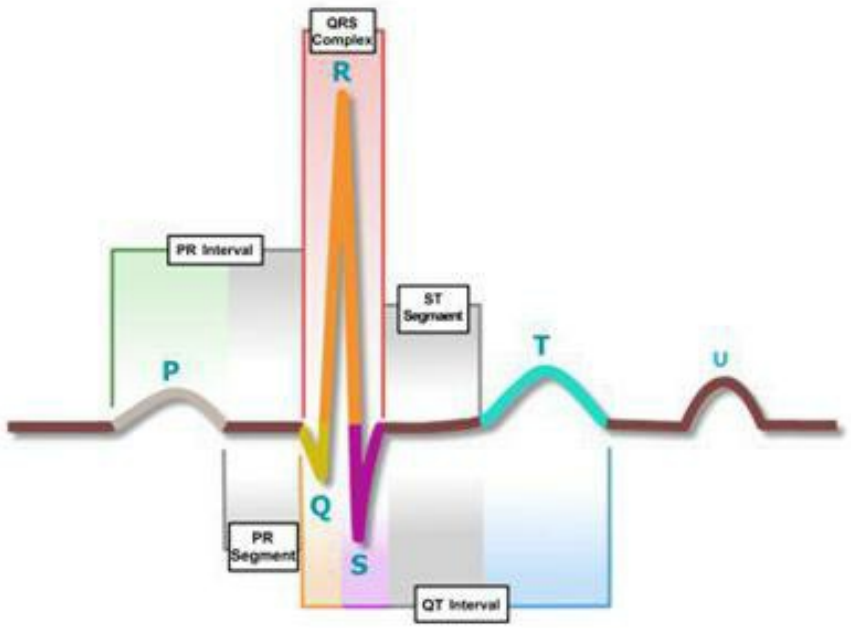


Fig 3.1 PQRST complex and Intervals

In general, the EKG tracing consists of waveforms named **P**, **Q**, **R**, **S**, **T** and occasionally **U** waves. Individual waves represent electrical activity happens in various parts of the heart. *Since the electrical activity is synchronized with mechanical function*, *EKG tracing indirectly represent contraction and relaxation of various chambers* . Considering the electrical excitation and ion transfer at molecular level, these waves also helps in identifying physical, physiologic and pathologic changes in the cardiac cells.

Components of EKG complex	
P wave	Atrial contraction or depolarization
QRS complex	ventricular contraction or depolarization
T and ST segment	ventricular relaxation or repolarization

Box 3.1 Components of EKG complex

Wave Morphology

The direction of complexes in an EKG is determined by the direction towards the current is flowing. Whenever the *current flows towards the positive electrode* , we will see an *up stroke* or *positive spike* in the EKG. The reverse happens when current flows toward *negative* electrode. A *biphasic* or *isoelectric* complex generates when the direction of flow is *perpendicular* to the positive electrode.

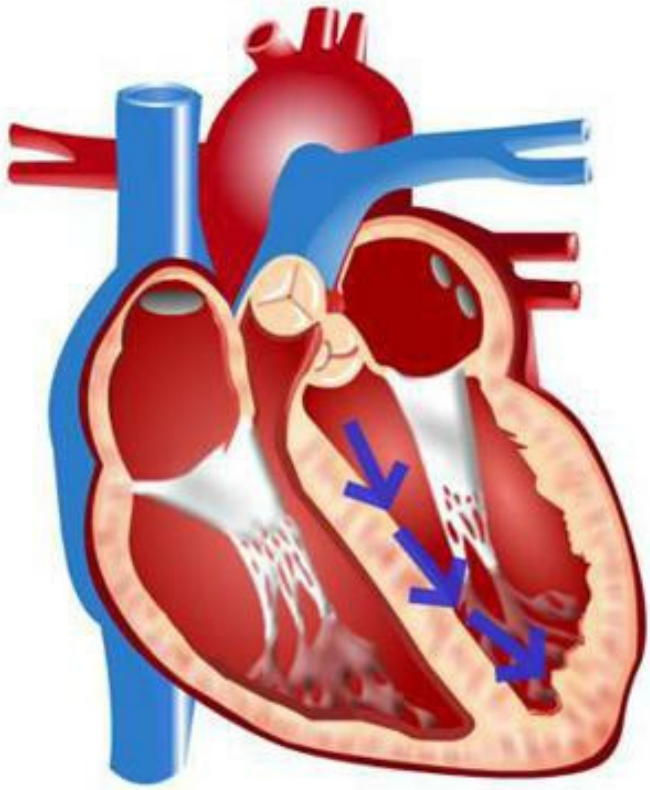


Fig 3.2 Net direction of electrical current in the heart.

This is of great help in understanding morphology of various EKG leads. Because of the large muscle mass concentration in the left ventricle, *the net vector* (*direction*) of electricity in the heart is generally directed towards *left and downward direction*. Therefore, any lead that is closer to the left ventricle such as precordial leads V4, V5 and V6 may see large electrical activity in the form of **tall R waves**. Similarly, in V1 which is situated on the right sternal border; the direction of flow of current is away from it and therefore predominantly negative in nature. This basic idea applies to all the limbs leads and augmented leads in an EKG.

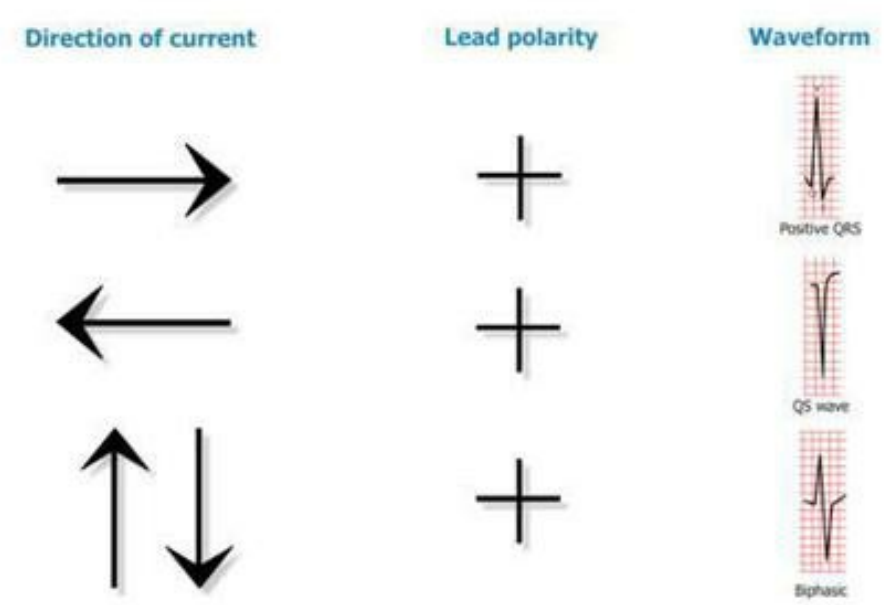


Fig 3.3 Wave morphology and direction of current flow

Current flowing towards positive electrode generate positive waveform and away from it generate negative waveform.

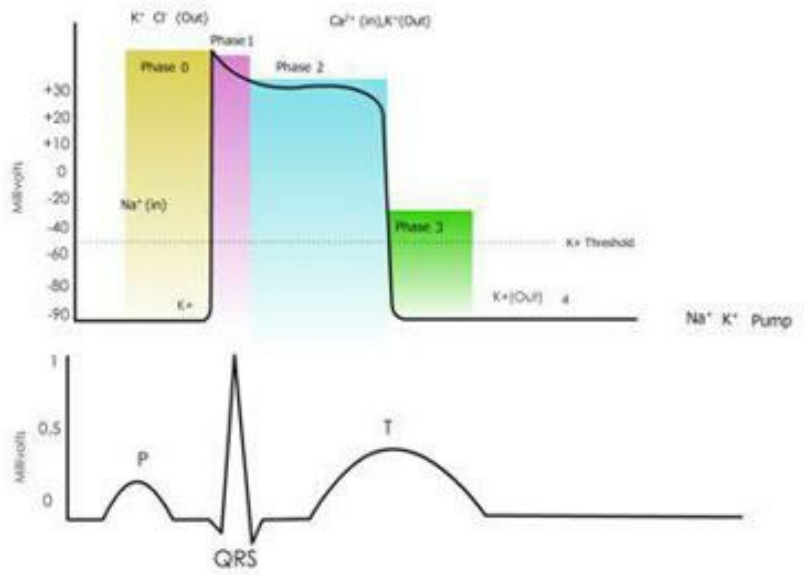


Fig 3.4 EKG and action potential

Lead Placements in a 12 lead EKG

In order to place precordial leads accurately on the chest, we have to follow some anatomical landmarks and imaginary lines on the chest wall. Since the intensity of electrical activity from the heart diminishes as the electrode placement moves away, *inaccurate lead placement results in poor and low amplitude EKG tracing*. The locations of individual leads are carefully determined in relation to the area of the heart under scrutiny.

Lead V1 sits close to right ventricle and should be placed on the space between fourth and fifth ribs along the right border of the sternum. Similarly, lead V2 is placed on the same location but on the left sternal border. Usually lead V3 is placed after V4 because the location of V3 is defined as 'in between V2 and V4'.

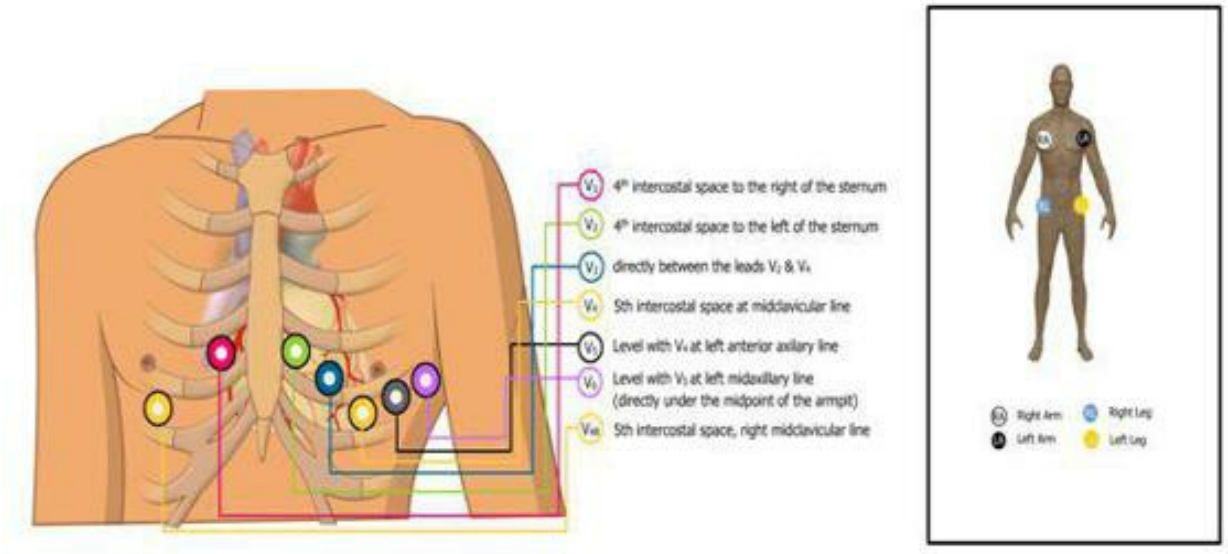


Fig 3.5 Lead placement in EKG

Lead V4 follows an imaginary line drawn from middle of the left clavicle known as midclavicular line (MCL) and usually falls below the nipple line. V5 follow the imaginary line coming down from the beginning of patient's left armpit known as anterior axillary line (AAL). Lead V6 goes under the left armpit on the imaginary line dividing the axilla known as mid axillary line (MAL).

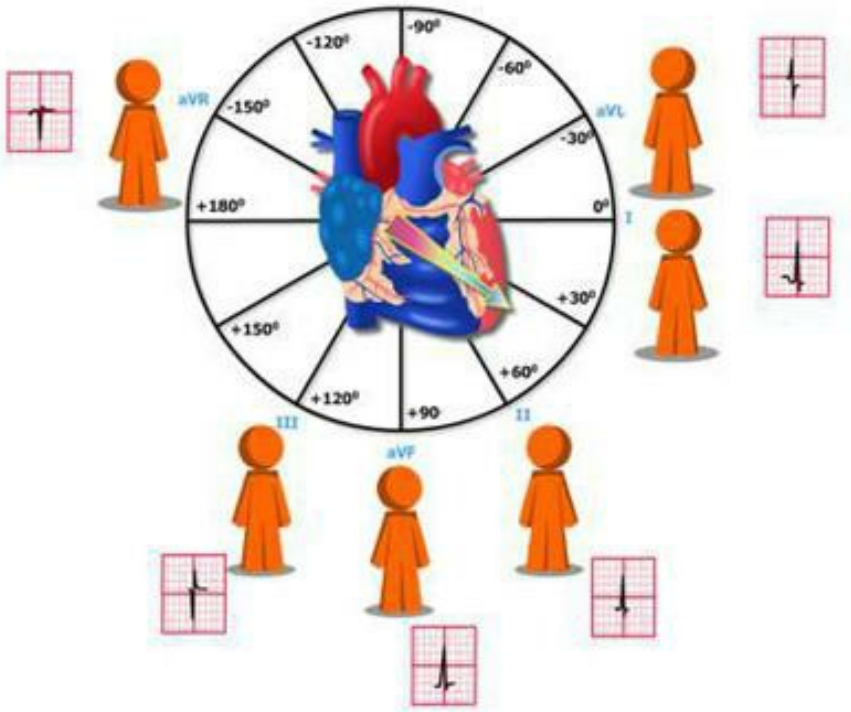


Fig 3.6 View of the heart from various lead positions

Posterior structures of the heart such as right atrium, posterior wall etc. are not well visualized in the regular 12 lead EKG setup. Therefore, we can add specific leads looking at the structures either on the right side of the heart or towards the posterior chest wall. Most common leads used for these purposes are lead V3R and V4R. They assume similar positions as its counterparts on the left chest wall. We also can have lead V7, V8 and V9, which are placed on the posterior chest wall similar to that of lead placement on the anterior wall, but as in mirror image.

Location of chest leads	
V1	fourth intercostal space on the right sternal border
V2	fourth intercostal space on the left sternal border
V3	between V2 and V4
V4	fifth intercostal space in the left midclavicular line
V5	fifth intercostal space in the left anterior axillary line
V6	fifth intercostal space in the left mid-axillary line
V3 R	between V1 and V4R
V4R	fifth intercostal space on the *right midclavicular* line

BOX 3.2 Location of chest lead placement

Electrocardiography Paper

Traditionally EKG is recorded in a graph paper that is divided into small and large columns. In a running strip, **X axis** (from left-to-right) indicates the *time* and **Y axis** (bottom to top) represents *voltage*. Typically the speed at which EKG recording happens is 25 mm per second. If the complexes are too narrow and clustered together as in tachyarrhythmia, the speed of EKG recording can be doubled, which helps in widening the waveforms and possibly better identification of the rhythm.

As shown in fig 3.7, each *small box* from *left-to-right* indicates ***0.04 seconds***. Since one large box includes five small boxes, *each large box* represent ***0.2 seconds***. There are five large boxes in one second. Usually there are three second markings on the top or bottom of the EKG paper and it helps in calculating rate of contraction as explained later in this chapter. In the same manner, each *small box* from *bottom to top* indicates ***0.1 mV*** and therefore, a stack of *10 boxes* in *Y-axis* represents ***1 mV***. These measurements are critical especially in determining various intervals and pathologic conditions from an EKG strip.

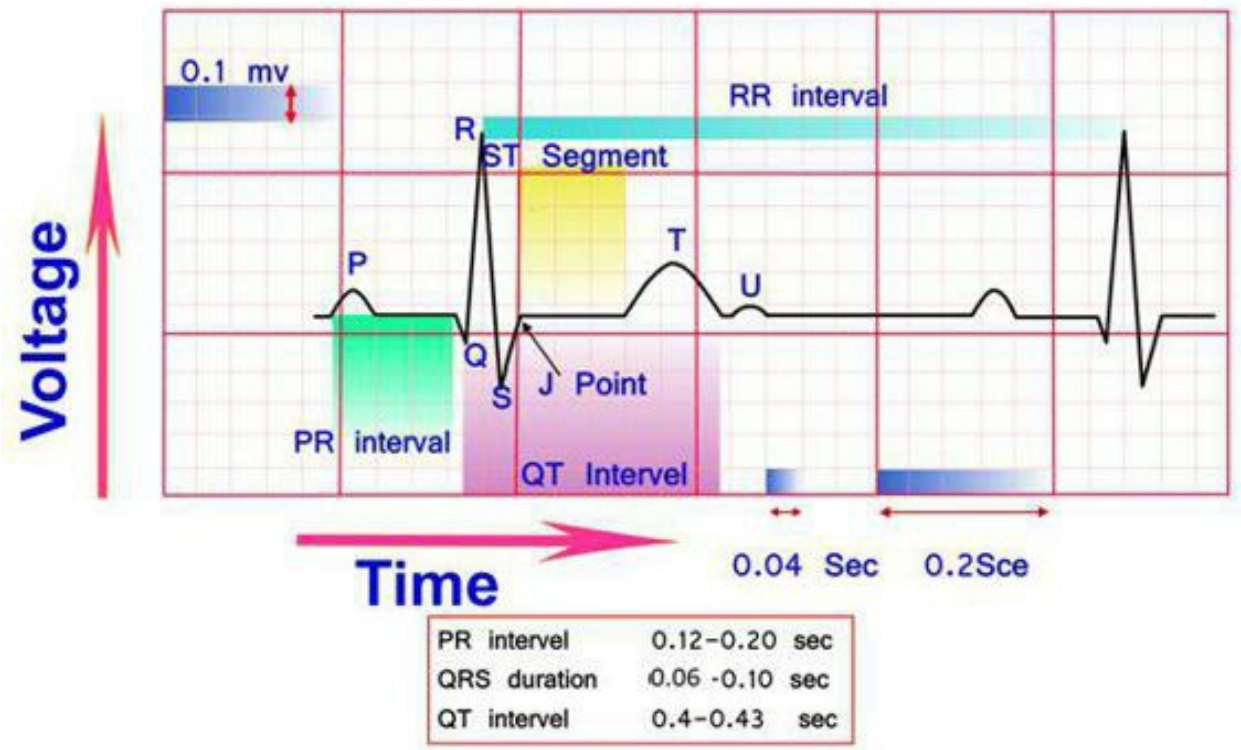


Fig 3.7 EKG with various intervals

Intervals and measurement in EKG

PR interval *Time delay in conduction of impulse in the AV node.*
It is prolonged in some types of heart block.
Normal 0.12 to 0.20 seconds

QRS duration *Time of ventricular repolarization (contraction).*
This value is increased in bundle branch block and premature ventricular contraction.
Normal 0.06 to 0.10 seconds.

QT interval *Total time of cardiac contraction and relaxation.*
Drugs like Amiodarone elongates QT interval.
Normal 0.40 to 0.43 seconds

Box 3.3 Various intervals in EKG

Identification of waves in EKG

P wave	*First positive deflection* in the EKG complex. It is inverted in the junctional rhythm or in aVR, V1 and in some pathologic waves. It determines whether the impulses are originating from the atria or somewhere else.
Q wave	*First negative deflection* in the QRS complex. A large Q wave represents myocardial death. (Greater than 0.04 ms and greater than one third of R wave).
R wave	*Second positive deflection* in PQRST complex. It is usually tall in EKG leads with a positive electrode on the left side of the chest wall such as lead I, II, III, aVL, aVF, V5 and V6.
S wave	*Second negative deflection* in the PQRST complex.
T wave	*Third positive deflection* in the EKG complex. In ischemia or infarction, T waves may be inverted.
U wave	*4th positive deflection* immediately following the T wave.
J point	*The junction between end of QRS and the beginning of ST segment*. J point is elevated in ST elevation.

Box 3.4 Waveforms in EKG complex

Corrected QT interval (QTc)

QT interval in an EKG is depending on the heart rate. With faster heart rates, QT interval will be shorter as there is less time between repolarization and depolarization cycles and conversely long QT interval with slower heart rate. If the given EKG has tachycardia or bradycardia, it can mask an underlying abnormal QT interval. Since the QT interval determination is extremely important in recognizing potential for lethal arrhythmia, a corrected QT interval (QTc) for a given heart rate should be calculated in every rhythm strip. One of the common QTc calculation formula is called **Bazett's Formula** and is shown below.

$$QT_C = \frac{QT}{\sqrt{RR}}$$

where

QTc is the corrected QT interval for the given heart rate

QT is the measured QT interval from the EKG strip

RR is the distance between two consecutive R waves (RR interval)

It is important to remember that corrected QT interval is closely connected to heart rate and therefore calculating QT_C on irregular rhythms such as Atrial fibrillation is very challenging. One of the common method used for measuring QT_C in Atrial fibrillation is that the final corrected QT interval is the average of the QTc of two subsequent long and short QRS complexes as shown below.

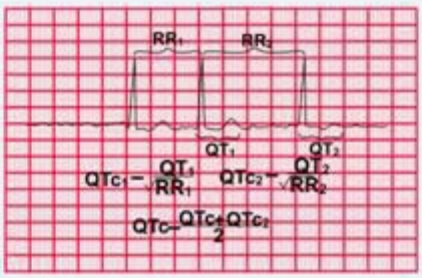


Fig 3.7B Calculating QT_C in Afib

Box 3.5 Corrected QT interval

Various Leads in Electrocardiography

Even though the EKG represents electrical activity of a live organ, for an electrocardiogram machine it is simply the difference in electrical potential between two given points called *leads*. Depending on the number of physical leads needed, it can be classified into **bipolar** (lead I, lead II and lead III) and **unipolar leads** (aVR, aVL, aVF and all V leads).

Bipolar Leads

These are the basic leads proposed by William Einthoven, who was the Nobel Prize winner in 1924 for invention of practical electrocardiogram. According to him, the ***'sum of***

the heights' of QRS in leads I and III equals the height of QRS in lead II. This equation is called **Einthoven's law**.

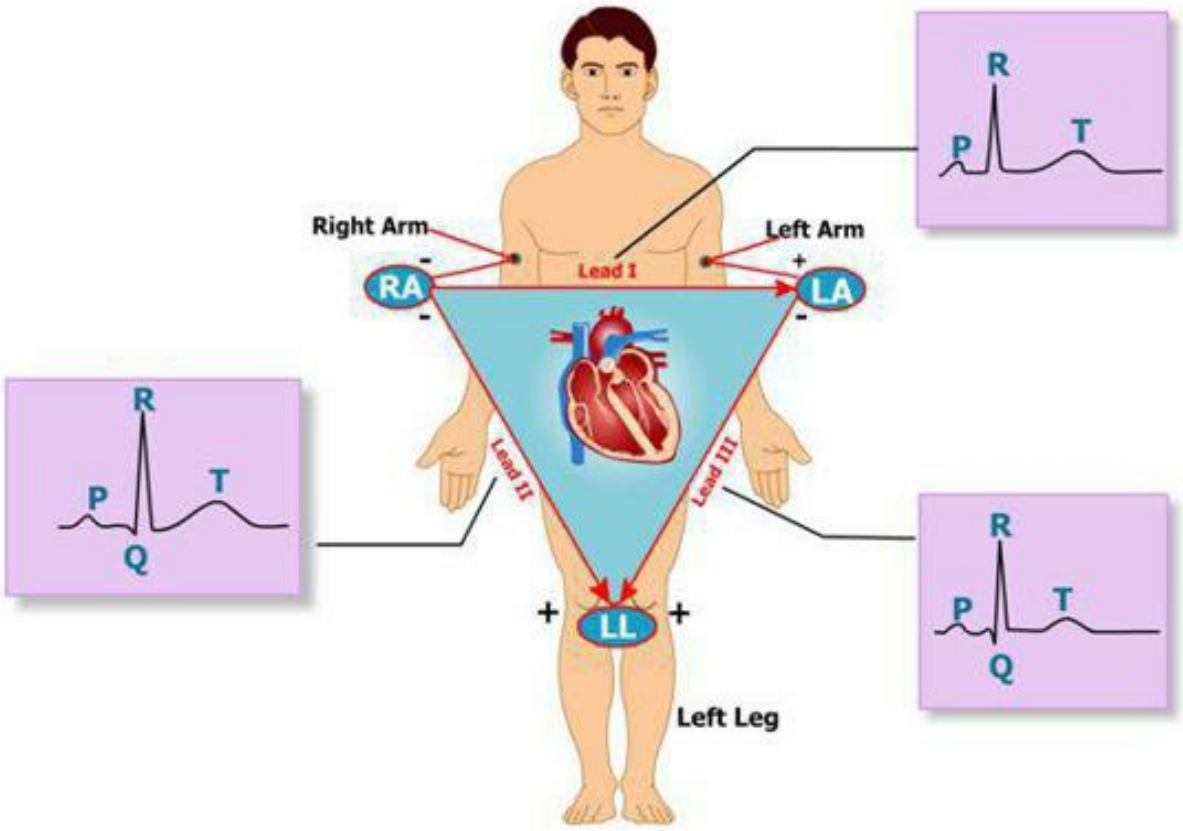


Fig 3.8 Einthoven triangle and limb leads

As shown in the picture, lead I has negative electrode on the right arm and positive on the left arm. Since the direction of current in the heart is towards left side and the positive electrode is sitting on the left arm in this case (flow of current towards positive electrode), *lead I* will have a *positive QRS* under normal circumstances. In *lead II*, right arm is negative and left leg is positive and direction of current is again towards positive electrode; resulting EKG will be predominantly *positive*. In lead III, potential difference is measured between the left arm and left leg with a positive electrode as shown in the picture, resulting EKG will be positive. Among these three leads, *lead II* will have *tallest R* waves since lead II closely represent the *true direction of the net cardiac vector*.

Unipolar Leads

Unlike bipolar leads, unipolar leads have a single physical electrode attached to the body. The machine calculates *electrical difference between the single available lead and an imaginary point at the centre of the heart with Zero electrical potential*. Among these, lead **aVR**, **aVL** and **aVF** are known as **Augmented leads** because the machine has to increase or augment EKG tracing by 50% for the purpose of representation.

All precordial leads like V1 to V6, right-sided precordial leads like V3R, V4R and posterior precordial leads such as V7, V8 etc. are unipolar as augmented limb leads. Posterior leads such as V7, V8 and right-sided precordial leads such as V4R, V5R may be taken under special circumstances like posterior wall myocardial infarction; which results from occlusion of dominant circumflex coronary artery or dominant right coronary with large posterolateral branch. In these conditions, anterior precordial leads may only show '*reciprocal changes'* due to a ST elevation MI. These lead placements are the mirror image of left-sided anterior precordial leads, but in their corresponding body surface.

Remember, if there is no QRS complex, there is no cardiac output!!!

Points to Remember!!!

- EKG is the graphical representation of the electrical activity of the heart.
- Each individual EKG leads show electrical activity between two different parts of the body.
- In 12 lead EKG, we are looking at six horizontal and six vertical angles at the heart.
- Current flowing towards the positive electrode shows a positive waveform in EKG and that flows away from positive electrode give negative waveform.
- The net vector (direction) of electrical activity of the heart is directed towards left and downward direction (towards left ventricle).
- Leads closer to the area of the heart with the net direction of current show tall positive waveforms (Lead V5, V6).
- Since EKG waves indirectly represent cardiac contraction, 'absence of QRS' means no ventricular contraction and therefore, no cardiac output'.
- Right sided and posterior leads are utilized in assessment of posterior walls of the heart.
- In EKG graph, from left to right (X axis) is the 'time' and from bottom to top (Y axis) is the voltage.
- Each small box in an EKG grid represents 0.04 sec in X-axis and 0.1 mV in Y-axis.
- PR interval is the delay in conduction of impulse from the SA node to AV node

(0.12-0.20).

· QRS complex represents time of ventricular contraction (0.06-0.10 sec).

· QT interval is the total duration of ventricular contraction and relaxation (0.40 - 0.43 Sec).

· Lead I, II, III are called bipolar leads and Lead aVR, aVL, aVF and V1-V6 are unipolar leads.

Test Your Understanding

1. Which of the following represent QRS complex in an EKG?

A Ventricular contraction

B Ventricular diastole

C Atrial systole

D Atrial contraction

2. Which of the following statement is true regarding direction of current and EKG waveform?

A Current flowing towards negative electrode produces a positive waveform

B Current flowing away from negative electrode produces a negative waveform

C Current flowing towards the positive electrode produces a biphasic waveform

D Current flowing away from positive electrode produces a negative waveform

3. The net direction of electrical activity of the heart is directed towards_____?

A Right atrial appendage

B Right ventricular outflow tract

C Left ventricle

D Sino atrial node

4. In a normal heart, which of the following represents the direction of EKG complex in lead V6?

A Isoelectric

B Positive

C Negative

D Varying from person to person

5. Which of the following precordial lead is placed in the fifth intercostal space at anterior axillary line?

A Lead V2

B Lead V6

C Lead V5

D Lead V3

6. Which of the following parameter is displayed on X axis of EKG?

A Time

B Voltage

C Heart rate

D Force of contraction

7. Normal QRS complex duration is _____?

A 0.12-0.2 second

B 0.40-0.43 second

C 0.06-0.10 seconds

D 0.24- 0.30 seconds

8. Which one of the following is an example for bipolar leads?

A Lead I

B Lead V4

C Lead aVL

D Lead V4R

Answers

1. A Ventricular contraction
2. D Current flowing away from positive electrode produces a negative waveform
3. C Left ventricle
4. B Positive
5. C Lead V5

6. A Time

7. C 0.06-0.10 seconds

8. A Lead I

Chapter 4

Systematic Interpretation of EKG

For untrained eyes, an EKG strip is a series of squiggly lines that doesn't tell anything by itself. However for the trained mind of medical personnel, it is the holy grail of information about someone's heart. Since there is so much information hidden among these wavy lines; which at times matters life and death; it is imperative that the person who is reading EKG should do it in a systematic and orderly fashion in order to avoid any possibility of overlooking vital information. For individuals who are beginning to practice the techniques of EKG interpretation, the systematic assessment may appear cumbersome and tedious; however, by constant practice and experience this technique becomes a second nature.

Steps in EKG Interpretation

1. Determine Rhythm and Regularity

The first and foremost step in evaluating an EKG strip is identifying components of the rhythm. As we mentioned in earlier chapters, normal EKG is a true representation of electrical functions of the heart. Therefore, we should have a **P wave** that represents channelization of electrical impulse and contraction response of atria in the beginning of each complex. *Presence of P wave* thereby ensures the fact that this *electrical impulse is coming off of atrium*. We should also see a sequential downward movement of electrical impulse to the next

destination, the AV node. As we know, there's going to be a delay in AV node that corresponds an isoelectric line between P wave and QRS complex called **PR interval**. Then the impulse travels through both ventricles results in depolarization (contraction) and produce a tall **QRS complex**. After contraction of chambers in response to this electrical impulse, ventricles relax (repolarize) back to their original state and are represented in the EKG as a **T wave**. There may also be a small **U wave** that represents late ventricular repolarization and it follows T wave. In order to assess the rhythm, we have to make sure all these waves are present and they appear in regular intervals and sequence.

Depending on the presence or absence of P wave, rhythm can be classified into atrial and ventricular rhythm. *If a P wave is present before every QRS complex*, *it represents a rhythm originating above the ventricle*. If there is presence of P wave and it has the same deflection as the QRS complex, mostly it is **sinus rhythm**. If the P wave is absent, inverted or show up after QRS complex; representing rhythm is originating from atrio ventricular junction and is called **junctional rhythm**. If there is no P wave and only QRS complex, then the rhythm is **ventricular** in origin. During this step, also look for regularity of the rhythm by analyzing P-P and R-R interval. In a *regular rhythm*, these measurements are going to be *constant from beat to beat*.

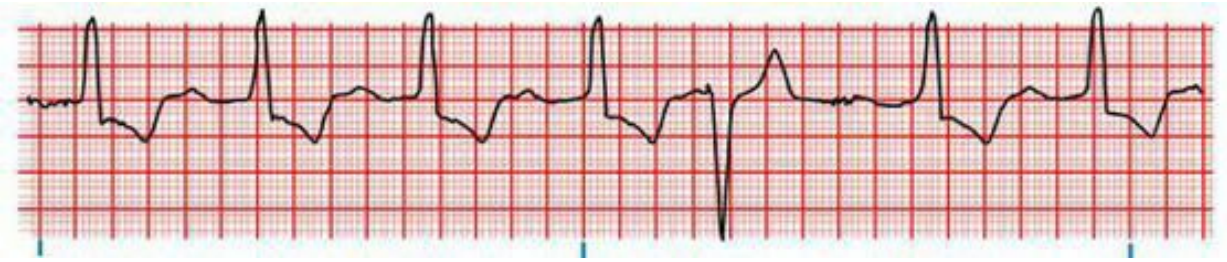


Fig 4.1 Regular rhythm with one irregular beat

In some instances, the rhythm can be regular in most of the part; however, one extra beat can make the entire rhythm look irregular as shown in Fig 4.1. Therefore, while assessing the regularity of rhythm, we need to evaluate the entire length of available EKG tracing. If we find a premature beat in one section, exclude that area from general assessment so that, an accurate interpretation can be made. Most of the time, these isolated premature beats are benign. However, in some situations these premature beats can present at regular intervals such as every other beat or every third beat (bigeminy and trigeminy) and these rhythms should be classified as *regularly irregular*.

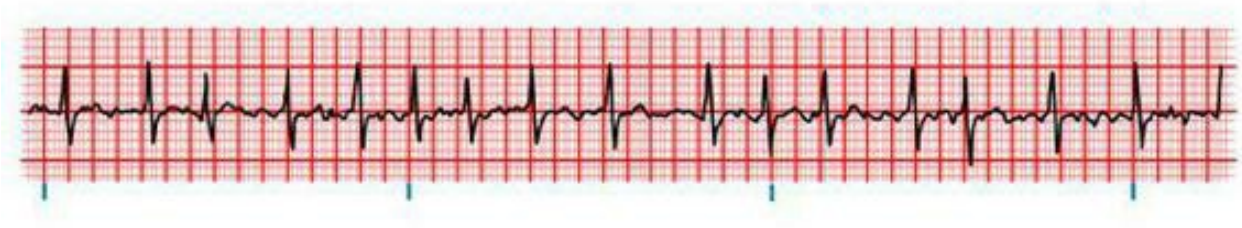


Fig 4.2 Atrial fibrillation (Please note the varying P-P and R-R interval)

In *completely irregular rhythms*, *P-P and R-R measurements will be different from beat to beat* as in Fig 4.2. This chaotic rhythm may have *multiple P waves* between each R wave as in the case of Atrial fibrillation and origin of R waves will be in random. In EKG leads with no R wave and a predominant QS pattern such as Lead V1, S-S interval may be used in place of R-R (Fig 4.3).

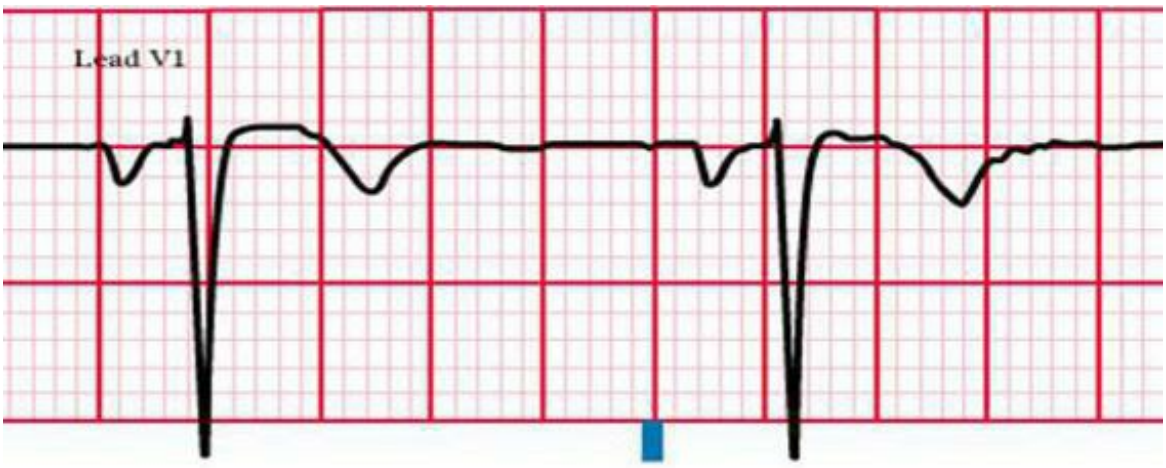


Fig 4.3 Lead V1 with QS pattern instead of usual QRS

2. **Calculate Rate**

While assessing the heart rate, we are more interested in the rate of ventricular contraction than that of atria, since ventricles are the main pumping chambers of the heart. Therefore, heart rate is calculated by assessing the *number of QRS complex in 1 min*. The rate of atrial and ventricular contraction is identical in a regular rhythm, since there is only one P wave preceding every QRS complex. However in irregular rhythms, the rate of atrial contraction is the number of P waves present in 1 min and that of ventricle is the number of QRS complexes in the same timeframe. There are various methods available for calculation of heart rate from EKG.

1. **The Six Second Method**

This is the fastest but least reliable method for assessing heart rate. Here, *count the number of QRS complex in a six second EKG strip and multiply by 10* and that will give you heart rate in 60 seconds or 1 min. For example, in a given EKG if there are six QRS complexes within six seconds, according to this method the heart rate will be 6 X 10 = 60 bpm. This method is particularly used for calculating regular rhythms.

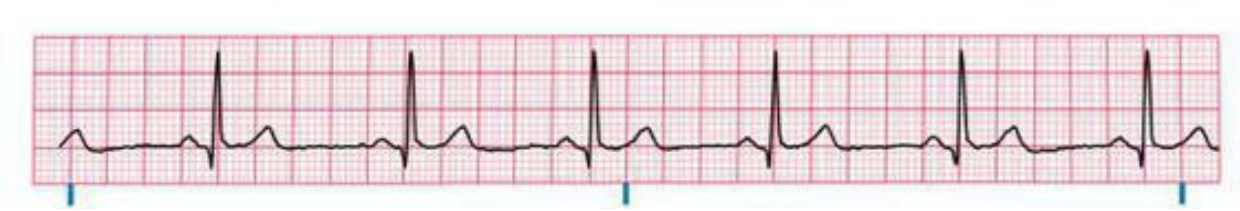


Fig 4.4 Six-second method of calculating rate

Steps in EKG interpretation

1. Determine the rhythm and regularity
2. Calculate the rate
3. Evaluate P wave
4. Calculate PR interval
5. Analyze QRS complex
6. Examine T wave
7. Calculate QT interval
8. Look for other characteristics

Box 4.1 Steps in EKG interpretation

2. **Counting Large Box Method**

This method involves memorizing a sequence of numbers and applying it on a given

strip. This method relies on the fact that there are **300 large boxes within 1 min strip**. Therefore, *counting the number of big boxes between two consecutive R waves and dividing it into 300* will give you the heart rate. You may use the same method on P waves to find atrial rate.

In order to perform this method, we have to find one R wave that is perfectly aligned with the thick line of a large box in the EKG grid and start counting number of large boxes until the next R wave. For example, if there are five large boxes between two R waves, the heart rate will be 300/5 = 60 bpm. For easier calculation, you may memorize the sequence of numbers like **300- 150- 100- 75 - 60- 50** and so on. That means if there are two large blocks between consecutive R waves, heart rate will be 300/2 = 150. If there are three large boxes, heart rate is 300/3 = 100 bpm. So if you have 10 large boxes between two consecutive R waves, rate is 300/10 = 30. In the given example, there are four large boxes between two consecutive R waves and therefore the rate of contraction is 300/4 = 75 beats per minute.

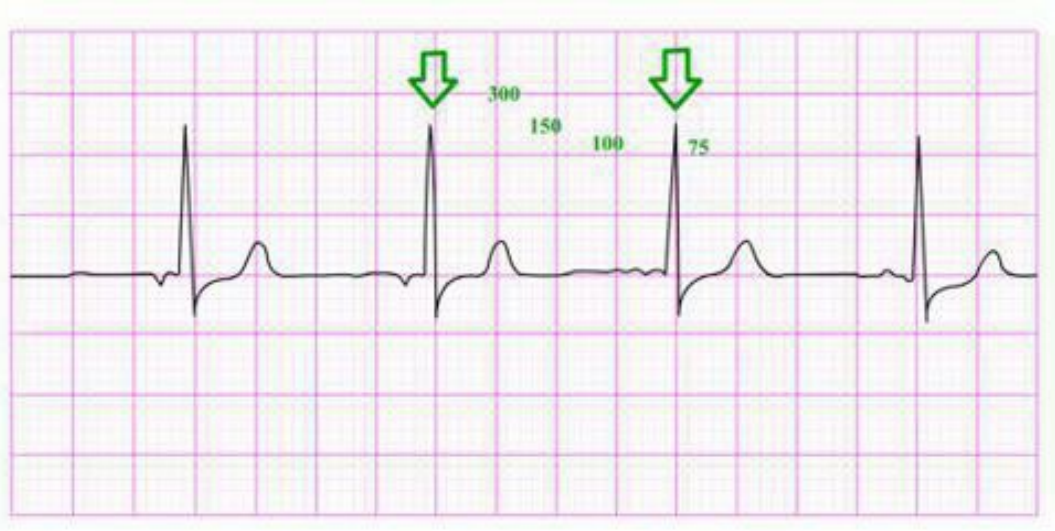


Fig 4.5 Counting large box method

3. **Counting Small Box Method**

Similar to the earlier method, it is based on the fact that there are **1500 small boxes in 1 min strip**. Therefore, in order to calculate ventricular rate, you may count the number of small boxes between two consecutive R waves and divided into 1500. This method is particularly *useful for fast rhythms*, where it may be hard to align one QRS with thick line of large box. For example, if there are 9 small boxes between two R waves as shown in the picture, heart rate will be 1500/9 = 166 bpm.

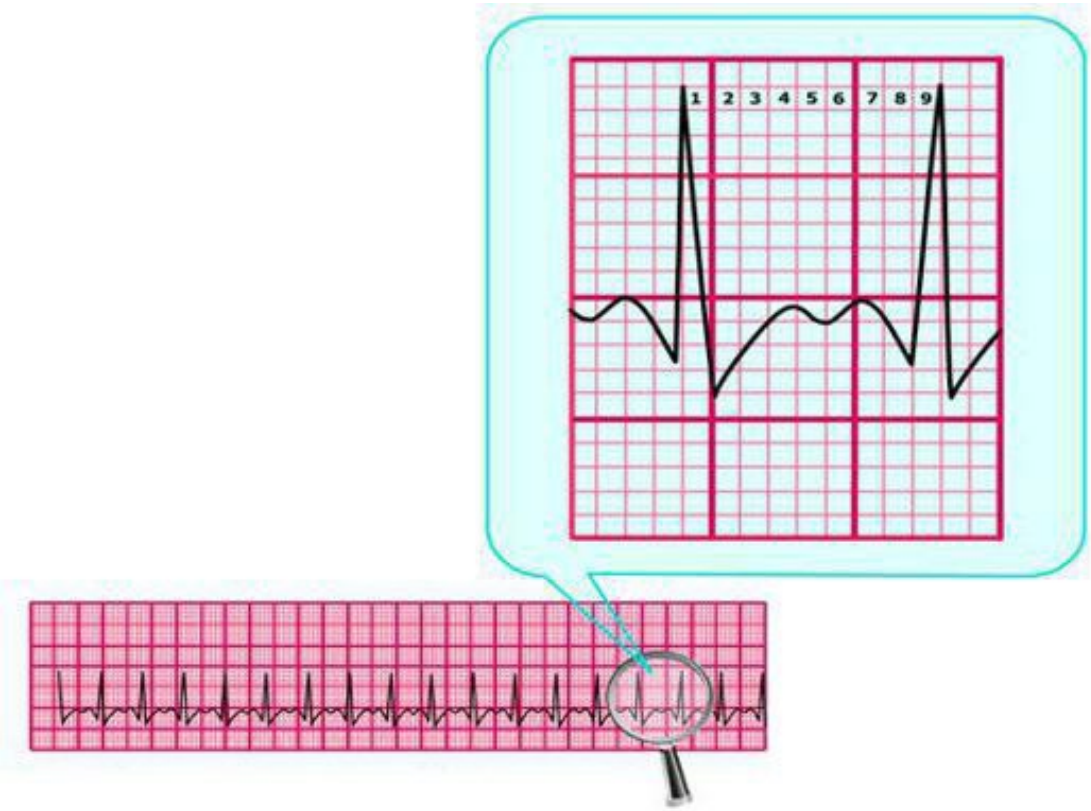


Fig 4.6 Small box counting method

3. **Evaluate P Wave**

Check for presence of P wave, their size and shape, variability and presence of one P wave for each QRS complex. Presence of P wave indicates that the rhythm is originating from above the ventricle. In *normal sinus rhythm*, *there will be one identical P wave before each QRS complex*. In situations where other ectopic foci within the atria produce impulses, the shape of P wave can be varying (wandering **pacemaker**). If the P wave appears to be inverted, biphasic or following QRS, then the rhythm is **junctional**. *P waves are absent in pure ventricular rhythms*. It is worth mentioning that some of the fast rhythms like supraventricular tachycardia, *P waves may not be visible* because of the close proximity of two consecutive QRS complexes.

4. **Calculate PR Interval**

PR interval *is the time taken for the impulse to travel from atria to the ventricle*. Therefore, elongation of PR interval shows additional delay for impulse to travel between the two chambers as seen in various types of heart block. In order to assess PR interval, *count the number of small boxes between the beginning of P wave to the beginning of QRS complex and multiply by 0.4*. Resulting value is the PR interval in seconds. For example, normal PR interval is 3 to 5 small boxes i.e. **0.12-0.20 seconds**. You need to *assess PR interval in*

multiple areas of the same strip so that any irregularity in PR interval, which is the hallmark of various types of heart block can be found.

5. **Analyze QRS Complex**

Count the *number of small boxes starting from the beginning of QRS complex* (i.e. from the end of PR interval) *to the end of S wave and multiply by 0.04*. Normal QRS complex duration is **0.06 - 0.10 seconds**. Look for other characteristics such as the size, symmetry, shape and presence of QRS after each P wave.

6. **Evaluate T Wave**

Examine the EKG for presence of T waves after each QRS, its shape (upright or inverted), amplitude (normally *not more than 1/2 the size of R wave*), any abnormal appearance (presence of hidden P wave within T wave) and presence of U wave after T waves.

7. **Calculate QT Interval**

QT interval represents *the time duration of depolarization and repolarization of ventricles*. Again, *count the number of small boxes between beginning of Q wave and end of T wave and multiply by 0.4*. Normal QT interval is between **0.36- 0.44 seconds**.

8. **Other Characteristics**

Look for other characteristics such as ectopic beats, pauses and any regularity of these events, ST segment changes, dropped beats, presence of multiple P waves for each QRS etc. This time, you essentially put together all the information gathered in previous steps.

Points to Remember!!!

- Presence of P wave in the beginning of an EKG complex represents an atrial rhythm.
- Junctional rhythms may have inverted, absent or trailing P waves.
- In regular rhythms, P-P and R-R interval are constant from beat to beat.
- In regularly irregular rhythm such as bigeminy or trigeminy, measurements of intervals should be done on regular waveforms.
- In completely irregular rhythms as in atrial fibrillation, P-P and R-R interval varies from beat to beat.
- In regular rhythms, heart rate can be calculated by multiplying the number of QRS complexes within six seconds strip in to 10.
- In order to calculate rate by counting large box method, count the number of large boxes between two constitutive R waves and dividing it into 300.

- For rapid rhythms, count number of small boxes between two adjacent R waves and divide into 1500 gives the heart rate.
- All measurements needs to be done in multiple areas of the same strip in order to avoid any possible beat to beat variations affecting the values.
- Presence of P waves with more than one morphology represent origin of impulses from multiple pacemaker sites as in the case of wandering pacemaker.
- In junctional rhythms, there may be inverted, absent or trailing P waves for each QRS complex.
- Normal PR interval is 0.12 to 0.20 seconds and QRS duration is 0.06 to 0.10 seconds.
- Normal QT interval is between 0.36 to 0.44 seconds

Test Your Understanding

1. Which of the following statement regarding an irregular rhythm is true?

A P-P and R-R intervals are constant from beat to beat
B P-P and R-R intervals does not match from beat to beat
C P-P interval is always constant; however, R-R interval varies
D P-P interval varies with a constant R-R interval

2. The hallmark characteristic of atrial rhythm is _____?

A Presence of U wave
B Presence of inverted P wave
C Presence of biphasic T wave
D Presence of P wave

3. Which of the following statement is true in measuring intervals on any rhythm strip?

A All measurements are done on the first beat of the strip
B All measurements are done on the last beat of the strip
C All measurements need to be repeated at various beats within the same strip
D All measurements to be repeated on first and last beat of the strip

4. Which of the following accurately represent calculation of heart rate by counting large box method?

A Count the number of large boxes between two adjacent R waves and divided into 1500
B Count the number of small boxes between adjacent R waves and divided in to 300
C Count the number of large boxes between adjacent R waves and multiply by 10
D Count the number of large box is between adjacent R waves and divided into 300

5. The best method for calculating heart rate in supraventricular tachycardia is _____?

A Six seconds strip method
B Counting small boxes
C Counting large boxes
D Counting number of QRS complex in the whole strip

Answers

1. B P-P and R-R intervals does not match from beat to beat
2. D Presence of P wave
3. C All measurements need to be repeated at various beats within the same strip
4. D Count the number of large boxes between adjacent R waves and divided into 300
5. B Counting small boxes

Part II

Understanding Arrhythmias

Chapter 5

SA Nodal Rhythms

In this chapter

- Normal sinus rhythm
- Sinus arrhythmia
- Sinus bradycardia
- Sinus tachycardia
- Sinus arrest
- Sino atrial exit block
- Inappropriate sinus tachycardia
- Sick sinus syndrome

These are cardiac rhythms originating from the SA node, which is the natural pacemaker of the heart. All of these rhythms will have the characteristic *uniform P waves before each QRS complexes*. These rhythms can be normal or abnormal such as normal sinus rhythm, sinus tachycardia, sinus bradycardia, sinus arrest etc.

Normal Sinus Rhythm

Normal sinus rhythm occurs when an impulse originate in SA node and then proceed to AV node, which then progress down to the ventricles through normal pathways resulting in a normal P and QRS complex.

Basic characteristics are

Rhythm: Atrial: regular, Ventricular: regular

Rate: 60 to 100 bpm

P wave: normal and uniform

PR interval: 0.12 - 0.20 sec (within normal limits)

QRS complex: 0.06- 0.10 Sec (within normal limits)

T wave: normal and uniform

QT interval: 0.36 - 0.44 Sec (within normal limits)

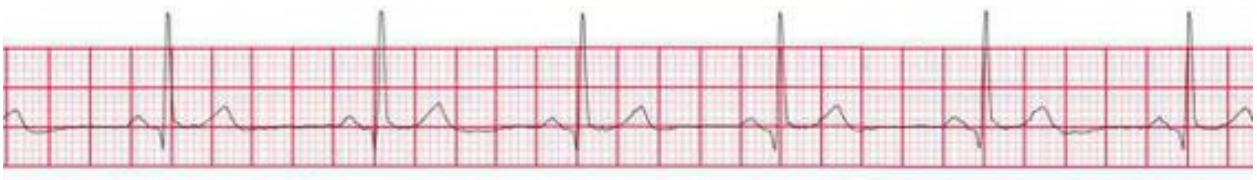


Fig 5.1 Normal sinus rhythm

Sinus Arrhythmia

In sinus arrhythmia, the heart rate stays within normal limits; however, the rhythm will be irregular. There will be *waxing and waning of heart rate in response to respiration*. As mentioned in chapter 1, sinus arrhythmia is the result of **vagal control** over the heart. It is assumed that during expiration there is a natural vagal stimulation and is responsible for slowing down of the heart rate. This is commonly seen in athletes, children and patients with sleep apnea.

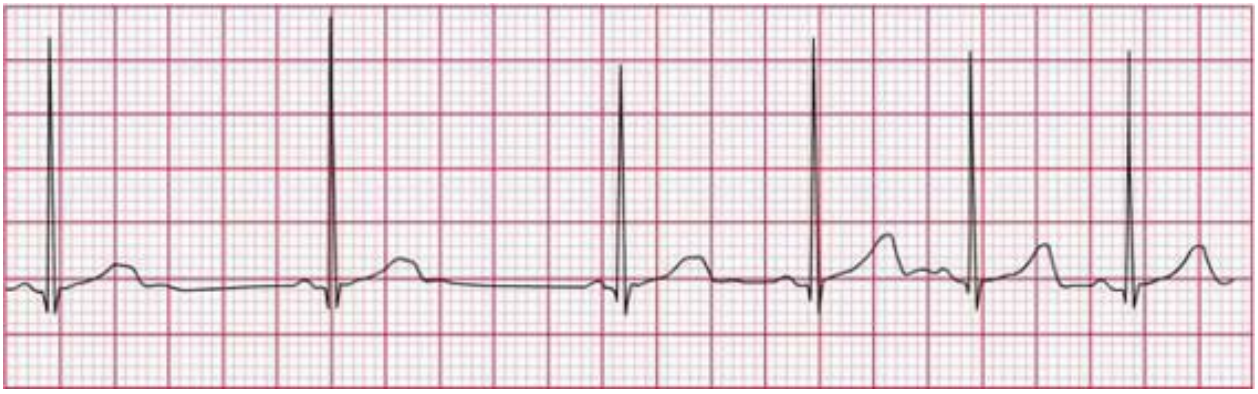


Fig 5.2 Sinus arrhythmia

Basic characteristics are

Rhythm: irregular and corresponds with respiratory cycle

Rate: *rate increases on inspiration and decreases with expiration*

P wave: uniform size and configuration

PR interval: may vary slightly, but remain within normal limits

QRS complex: normal configuration

T wave: within normal limits

QT interval: may vary slightly between beats, but stays within normal

Clinical significance: there is *no pathophysiologic significant and no treatment* is needed.

Sinus Bradycardia

This rhythm is characterized by sinus rhythm with *heart rate below 60*.

Basic characteristics are

Rhythm: regular

Rate: *less than 60 bpm*

P wave: uniform size and configuration

PR interval: within normal limits and constant

QRS complex: normal

T wave: uniform shape

QT interval: normal

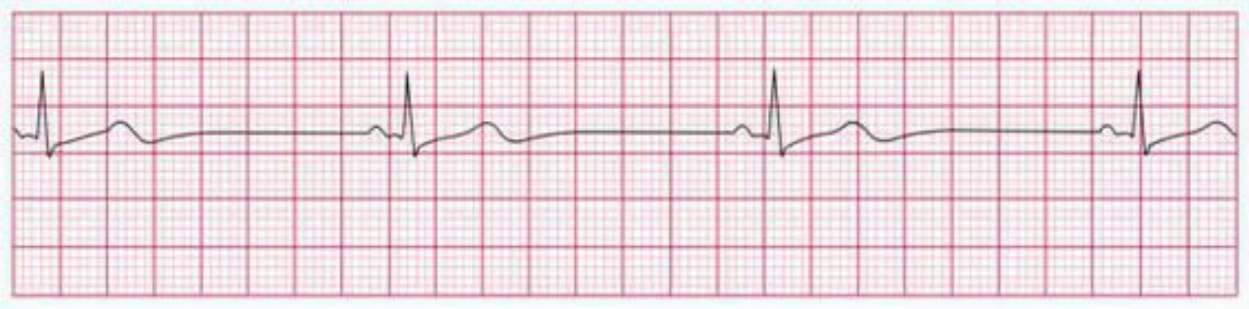


Fig 5.3 Sinus bradycardia

Clinical significance: Brady arrhythmia can result in hemodynamic compromise and symptoms such as *syncope*, *chest pain*, *premature beats*, *ventricular tachycardia* etc. Mostly, sinus bradycardia is benign; however, if the rate goes below 40 bpm, patient may become symptomatic. Sinus bradycardia is normal in athletes because of their conditioning of the heart.

Etiology: Etiology of SA node dysfunction causing brady arrhythmia can be (1) **Extrinsic factors** such as influence of drugs (e.g. digitalis), autonomic nervous system response (vagal stimulations from vomiting or straining for bowel movement), hypothyroidism, sleep apnea, hypothermia, hypoxia, increased intracranial pressure (Cushing's response) or endotracheal suctioning etc. (2) **Intrinsic factors** are degeneration and fibrosis of SA nodal areas from coronary artery disease, pericarditis, myocarditis, rheumatic heart disease, systemic lupus erythematosis (SLE), genetic disorders etc.

Management: *No treatment is necessary if the patient is asymptomatic and the rate stays close to 60 bpm*. If the patient becomes symptomatic, emergency treatment involves

administration of **Atropine**. Once hemodynamically stable, identify the underlying reason for sinus bradycardia. In emergent situations, a **temporary pacemaker** may be helpful. Once sinus node dysfunction is confirmed, patient may benefit from **permanent pacemaker implantation**.

Sinus Tachycardia

Sinus tachycardia involves accelerated firing of SA node with a rate greater than 100 beats per minute.

Basic characteristics are

Rhythm: regular

Rate: *greater than 100 beats per minute* (rarely above 160)

P wave: normal size and configuration. However, as the rate increases it may be hidden in T waves

PR interval: shorter because of the fast heart rate

QRS complex: constant and within normal limits

T wave: constant and within normal limits

QT interval: mostly shorter

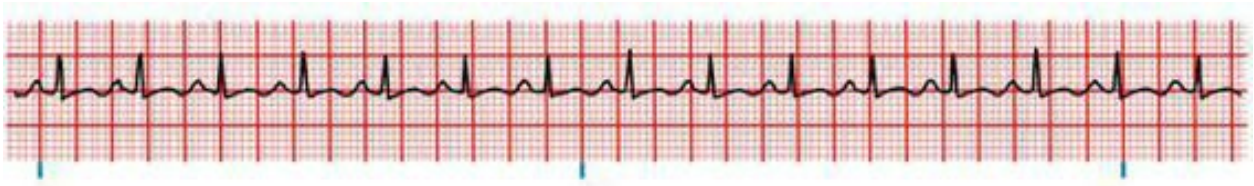
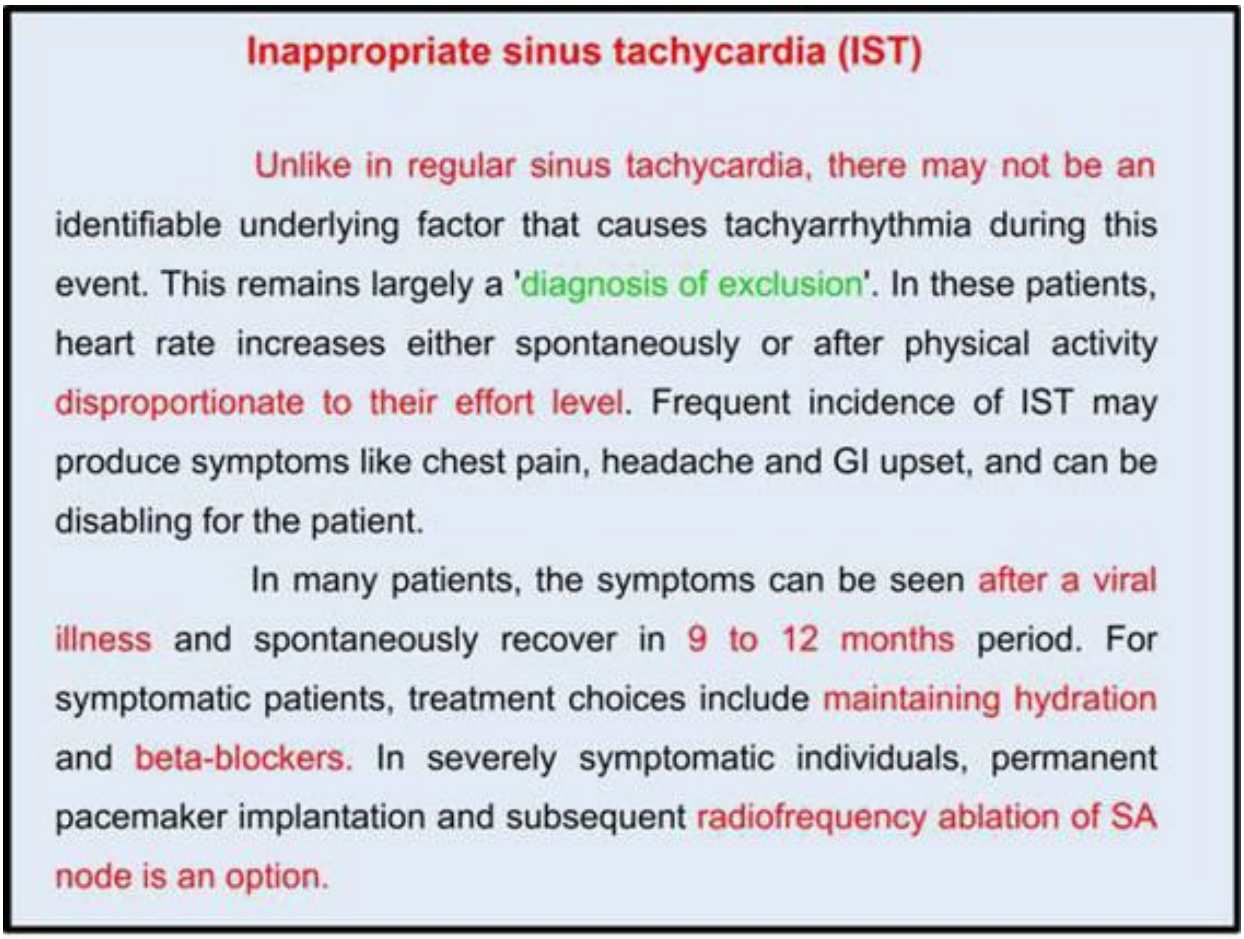


Fig 5.4 Sinus tachycardia

Clinical significance: Commonly, sinus tachycardia is considered as a *symptom of an underlying pathophysiologic process* and therefore, attention should be directed for finding primary cause rather than treating it right away. Rarely, patients can have so-called **'Inappropriate sinus tachycardia' (IST)**. Sinus tachycardia is a *sign of cardiac compensation* to maintain tissue perfusion in the event of hypovolemia, sepsis, hyperthyroidism, heart failure or sympathetic stimulation. Because of the increased heart rate, this rhythm may considerably increases myocardial oxygen demand and can precipitate heart failure and anginal symptoms.



Box 5.1 Inappropriate Sinus Tachycardia

Management: As mentioned earlier, investigation should be directed towards *identifying and treating underlying disease process* that is causing sinus tachycardia. If sinus tachycardia itself is causing hemodynamic compromise, pharmacologic agents like **beta-blockers, calcium channel blockers**, **adenosine** or **digoxin** may be helpful. Patient should be monitored for signs of decreased cardiac output and impaired left ventricular function in the event of profound sinus tachycardia. Supportive measures with administration of oxygen may be helpful in preventing myocardial oxygen depletion.

Sinus Arrest

In this type of rhythm, a normal sinus rhythm is interrupted by *prolonged failure of SA node to initiate an impulse* resulting in *complete missing of PQRST* complex.

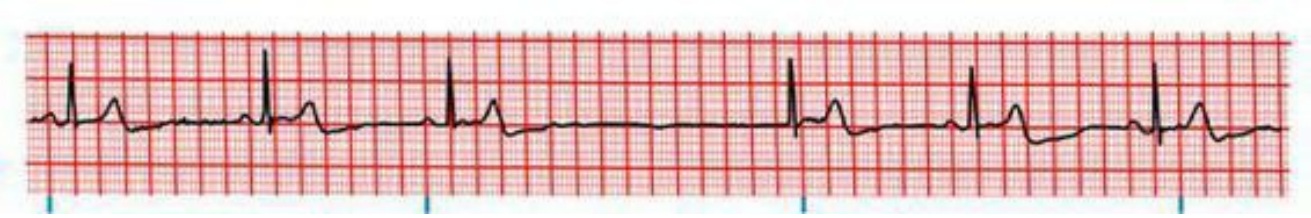


Fig 5.5 Sinus arrest

Basic characteristics are

Rhythm: regular except missing complex (pause)

Rate: usually within normal limits; however, length and frequency of pauses may lead to bradycardia

P wave: *normal except during pause with missing PQRST complex*. The P wave may retain its uniform shape as before depending on whether SA node or other ectopic pacemakers regain function after the pause.

PR interval: within normal limits; however, may be different in following EKG complex depends on the origin of impulse.

QRS complex: one complete QRS complex will be absent during the pause. Following QRS may assume its shape depending on whether it is SA nodal rhythm or escape beat.

T wave: normal except during arrest where there is no T wave. Again depending on the site of origin of impulse, it may be normal or abnormal in the following beats.

QT interval: same as in T wave; absent during pause and normal or abnormal in the following beats

Clinical significance: patients are asymptomatic when the length of pause is shorter; however, longer pause can create hemodynamic compromise and may need intervention.

Etiology: Most common causes for sinus arrest are *ischemia affecting SA node*, *effect of SA nodal blocking drugs* (e.g. beta blockers) and *excessive vagal tone*.

Management: depends on the underlying reasons and presenting symptoms. Treatment options are same as that of sinus bradycardia.

Sinus Exit Block

In this situation, sinus node fires impulse; however, it doesn't generate a subsequent QRST complex because of the lack of conduction down the pathway. Unlike in sinus arrest, this is a *conduction disturbance rather than problem with cell automaticity*. There may be one or more P waves get blocked, leading to a pause in rhythm. Interestingly, the rhythm regains to normal sinus rhythm as before. The most distinguishable character between sinus exit block and sinus arrest is that *the pause will be a multiplier of R-R interval*. For example, if there are two nonconducted P waves, the length of pause will be two times R-R interval. This is because, unlike in sinus arrest where an escape pacemaker takes over impulse formation immediately after pause due to the lack of SA node activity; here, the normal SA node impulse start propagating leading to normal sinus rhythm.

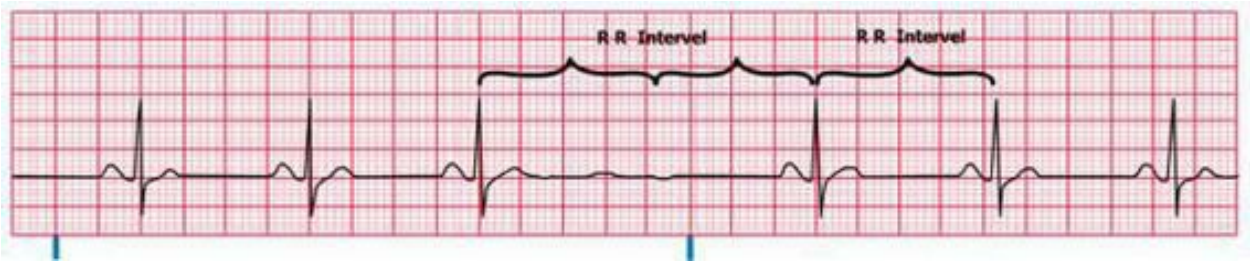


Fig 5.6 Sinus exit block (Note that the length of pause is two times the R-R interval)

Basic characteristics are

Rhythm: regular except during pause

Rate: usually within normal limits; however, bradycardia if length of pause is long

P wave: periodically absent during pause, otherwise normal

PR interval: normal except during the pause

QRS complex: within normal limits; missing during pause

T wave: absent during the pause

Other characteristics: *length of pause is a multiplier of R-R interval*

Clinical significance: only if the length of pause is longer, it may cause hemodynamic compromise or subjective symptoms. Treat as if sinus bradycardia.

Sick Sinus Syndrome (SSS)

Just as the name implies, sick sinus syndrome (SSS) is a clinical condition with

sickness of the natural pacemaker of the heart. This SA node dysfunction can be caused by degeneration and fibrous replacement of SA node tissue associated with inferior wall MI, pericarditis, myocarditis, rheumatic heart disease, SLE and some genetic mutations. Failure of SA node to generate impulses leads to activation of ectopic pacemakers within the atria and resultant tachyarrhythmia mostly in the form of **atrial fibrillation** or **flutter**.

Because of the **override suppression phenomena** of cardiac cells, where the automaticity of pacemaker cells diminishes after they have been excited with an impulse at a higher frequency such as in atrial fibrillation; even after the ectopic foci stop firing, SA node cells remain dormant for little longer. This leads to creation of a long pause in the EKG. Absence of cardiac output during this time can cause clinical symptoms. *The time taken for SA node to reclaim its pacemaker function* after the end of tachyarrhythmia is called **sinus node recovery time**. In patients with sick sinus syndrome, this recovery time maybe longer than usual. If the period of asystole is significantly long, patients can have loss of consciousness. Because of the fact that heart rhythm varies between tachycardia and bradycardia, it is also called '**tachy-brady syndrome**'.

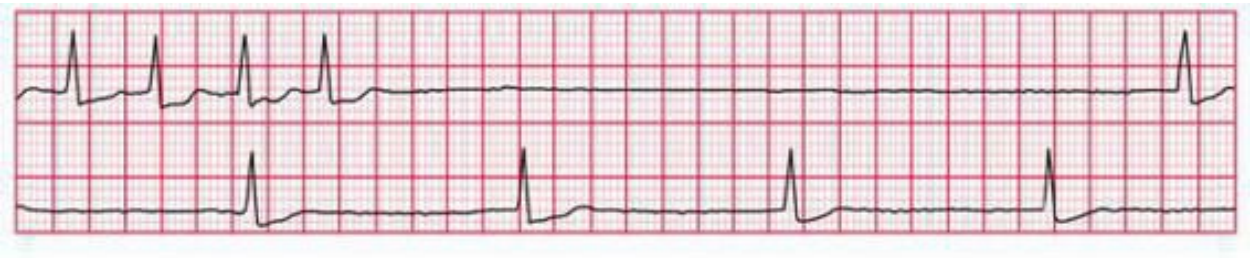


Fig 5.7 Sick sinus syndrome with pause (note the sinus node recovery time after tachycardia leading to a long pause)

Basic characteristics are

Rhythm: irregular with sinus pauses. May have sinus arrest, sinus exit block or long pause following termination of tachycardia.

Rate: alternate between slow and fast pace

Management: depending on the presentation of cardiac rhythm, management may differ. In general, for bradycardia causing profound symptoms **permanent pacemaker implantation** is warranted. As far as the tachycardia component is concerned, management varies depending on the type of rhythm. If the patient has underlying atrial fibrillation or flutter, various drugs or radiofrequency catheter ablation may be helpful. In the event the patient has chronic atrial fibrillation, he or she may need thromboembolic prevention by use of oral anticoagulants like Coumadin. In many instances, patient may require permanent pacemaker to treat bradycardia and rate control agents like beta-blockers and calcium channel blocker for tachycardia.

Points to Remember!!!

- All SA nodal rhythms have a uniform P wave before each QRS complex.
- In sinus arrhythmia, there is waxing and waning of heart rate in response to respiration.
- Sinus arrhythmia is an example of vagal control over the heart.
- Sinus bradycardia can be caused by extrinsic factors (drugs, vagal stimulation, hypothermia, hypoxia) or intrinsic factors (degradation of SA node, pericarditis, myocarditis, rheumatic heart disease).
- Administration of atropine and temporary or permanent pacemaker implantation are the treatment for sinus bradycardia.
- Mostly, sinus tachycardia is a symptom of an underlying pathology than a disease by itself.
- Tachycardia disproportionate to the effort level is called inappropriate sinus tachycardia.
- Beta-blockers, calcium channel blockers, adenosine and digoxin are the possible treatment options for sinus tachycardia.
- Sinus arrest is caused by prolonged delay in initiation of impulse from SA node.
- Excessive vagal tone, ischemia or fibrosis of SA node and effect of nodal blocking drugs are the usual causes for sinus arrest.
- Unlike in sinus arrest, during sinus exit block there is impulse production from the SA node. However, there is failure of impulse conduction down the system.
- Major differentiation of the pause caused by sinus exit block is that the pause is a multiplier of R-R interval.
- In sick sinus syndrome, there is prolonged sinus node recovery time after tachycardia event; leading to a pause.
- Because of override suppression phenomena of cardiac cells, they respond only to the fastest stimuli available to them irrespective of its origin. This is the reason for excitation of heart by fast ectopic pacemakers.
- Permanent pacemaker implantation for management of bradycardia along with nodal blocking agents for treatment of tachycardia is the mode of therapy for tachy-brady syndrome.

Test Your Understanding

1. Which of the following represent the most distinguishable character of sinus arrhythmia?

A Heart rate below 50 bpm
B Heart rate above 150 bpm
C Increasing and decreasing heart rate with respiration
D Presence of intermittent pause

2. Treatment for sinus bradycardia includes _____?

A Beta-blockers
B Digoxin
C Atropine
D Verapamil

3. Which of the following is not a treatment option for inappropriate sinus tachycardia?
A Beta-blockers
B Radiofrequency ablation
C Permanent pacemaker
D Atropine

4. Which of the following statement is true for the pause during sinus exit block?
A Length of the pause is half of RR interval
B Length of the pause is a multiplier of PR interval
C Length of pause is a multiplier of R-R interval
D Length of pause does not correlate with R-R interval

5. Which of the following is a true definition of override protection phenomena of cardiac cells?
A Cardiac cells respond to the fastest stimuli available to them irrespective of its source
B Cardiac cells respond to the fastest stimuli only if it is from SA node
C Cardiac cells respond to the slowest available stimuli irrespective of its strength
D Cardiac cells respond to the slowest available stimuli only if it is strong enough

Answers

1. C Increasing and decreasing heart rate with respiration
2. C Atropine
3. D Atropine
4. C Length of pause is a multiplier of R-R interval
5. A Cardiac cells respond to the fastest stimuli available to them irrespective of its source

Chapter 6

Atrial Dysrhythmia

In this chapter

- Premature atrial complex
- Atrial tachycardia
- Paroxysmal atrial tachycardia
- Multifocal atrial tachycardia
- Atrial flutter
- Atrial fibrillation

These rhythm disturbances are originating from *ectopic foci within atria other than SA node*. They can be either isolated or benign in nature such as **premature atrial complex** (PAC) or sustained and irregular that causes hemodynamic compromise like **atrial fibrillation**. Either way, these rhythms carry a *P wave*, which may get buried in the neighboring QRS if rate is high and *a narrow QRS complex* except in very selected situations with presence of aberrant conduction systems.

Premature Atrial Complexes (PAC)

PAC's are formed when an ectopic focus within the atria generate a premature impulse, which either conducted down through the AV node or die down within atria itself. Depending on the timing of this impulse, it may or may not show in the EKG as a distinct wave. For example, if the atrial premature beat originates way early in the cardiac cycle, it may get buried in the QRS complex. If the impulse happens later in the cardiac cycle, PAC can generate a *ventricular contraction that is out of sync* with the rest of the rhythm. Then it will be characterized by a *different shaped P wave*. Sometimes this P wave can be hidden within the T wave of previous QRS complex and thereby causing a *distorted T wave*. Therefore, whenever there is a T wave that is deformed with subsequent normal QRS complex and normal T wave, it raises the suspicion of a PAC.

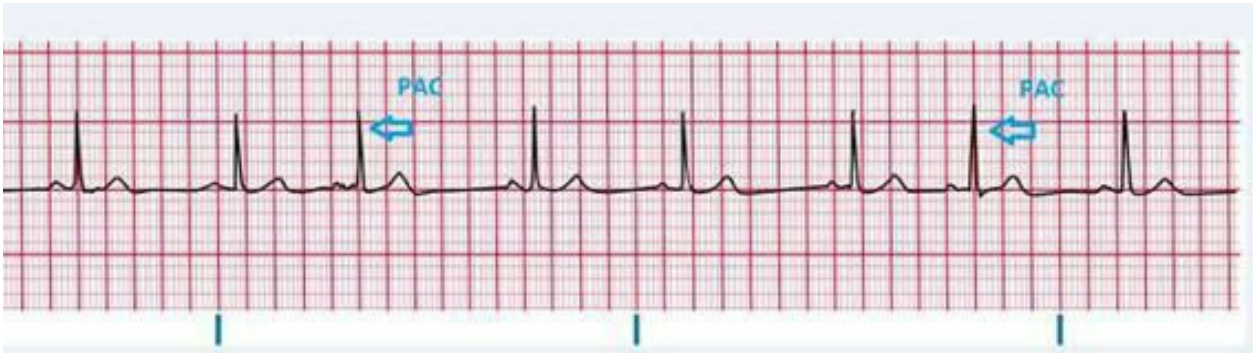


Fig 6.1 Premature atrial contraction

Basic characteristics are

Rhythm: normal except during PAC

Rate: usually within normal limits

P wave: deformed P during PAC

QRS complex: normal

T wave: usually normal unless a P wave hides within it.

QT interval: normal during underlying rhythm; varies if T wave is distorted

Clinical significance: Occasional PACs are completely benign. Once they become more frequent, may cause subjective symptoms such as **palpitations**. However, frequent PACs are sometimes prelude to atrial tachycardia or fibrillation. Recurrent PACs can be treated with *Calcium channel blockers*, *digitalis* or *beta-blockers*.

Atrial Tachycardia

These rhythms can be generally named as **Supraventricular tachycardia** (SVT) because of the fact that all of them *originate above the level of ventricle*. Common forms of atrial tachycardia are **Paroxysmal atrial tachycardia** (PAT) and **Multifocal atrial tachycardia** (MAT) or so-called '**wandering pacemaker**'. These rhythms are characterized by an *atrial rate of 150 to 250 bpm*. Physiologically these accelerated atrial rhythms diminishes '*atrial kick*', which is responsible for approximately 30% of ventricular filling. This may eventually affect cardiac output.

Paroxysmal Atrial Tachycardia (PAT)

Characterized by *sudden onset of three or more beats* o f *narrow complex tachycardia*; usually originate with a premature atrial contraction. These rhythms last only for a short duration and is called '**paroxysmal**'. It resembles sinus tachycardia; however, the characteristic *initiating PAC* is visible here.

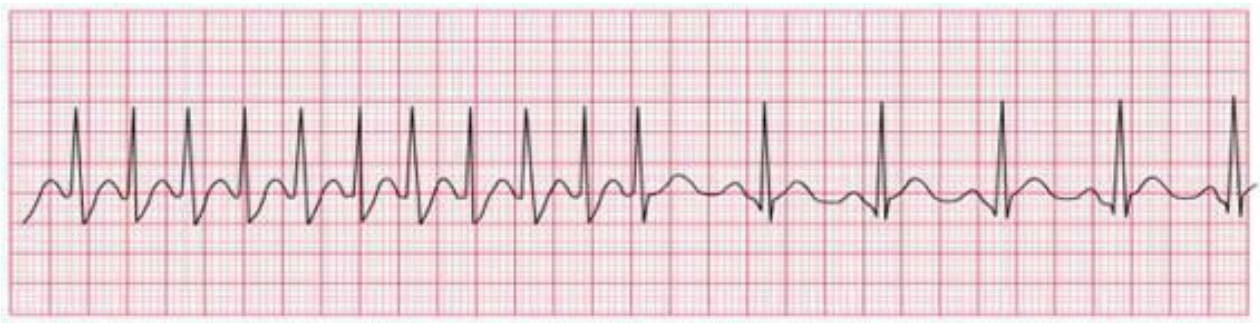


Fig 6.2 Paroxysmal atrial tachycardia converting to sinus rhythm

Basic characteristics are

Rhythm: underlying rhythm is regular; interrupted by tachycardia which itself is regular.

Rate: 150 to 250 bpm

P wave: uniform shape in underlying rhythm; however, different shaped or absent in tachycardia because it buried under neighboring T wave.

PR interval: within normal limits and constant in underlying rhythm; unidentifiable or shorter during tachycardia

QRS complex: narrow throughout the rhythm

T wave: normal during underlying rhythm; however, distorted in tachycardia.

QT interval: normal during underlying rhythm, shorter in tachycardia.

Clinical significance: Depending on the length of atrial tachycardia, patients may have symptoms ranging from mere palpitation to serious hemodynamic compromise. *Isolated PACs are perfectly normal*.

Etiology: Origin of atrial tachycardia may trace back to problems with *automaticity* o r *re-entry* of atrial impulses like in the case of other atrial arrhythmias. Hypoxia, hypocalcaemia, Digoxin toxicity, increased vagal tone, cardiomyopathy etc. are the cause of atrial arrhythmias.

Management: Strategy for management of PAC is based on *the length of tachyarrhythmia* and the *extent of symptoms*. Isolated PACs doesn't need any treatment and are benign. However, symptomatic tachycardia needs medications like *beta-blockers*, *digoxin* or *calcium channel blockers*. Avoidance of stimuli such as *caffeine* or *cigarette* smoking is also important. Rate control in acute situations can be achieved by administration of adenosine or application of vagal maneuvers such as carotid sinus massage or coughing. Depends on the availability, seriously ill patients can have direct current cardioversion for converting them

back to sinus rhythm.

Multifocal Atrial Tachycardia (MAT)

This rhythm is also known as **Wandering pacemaker,** characterized by *more than one shaped P wave*. During this event, impulses are originating from **multiple atrial ectopic foci** and hence, different morphology of P wave. Usually *more than three identifiable types of P waves* can be seen.

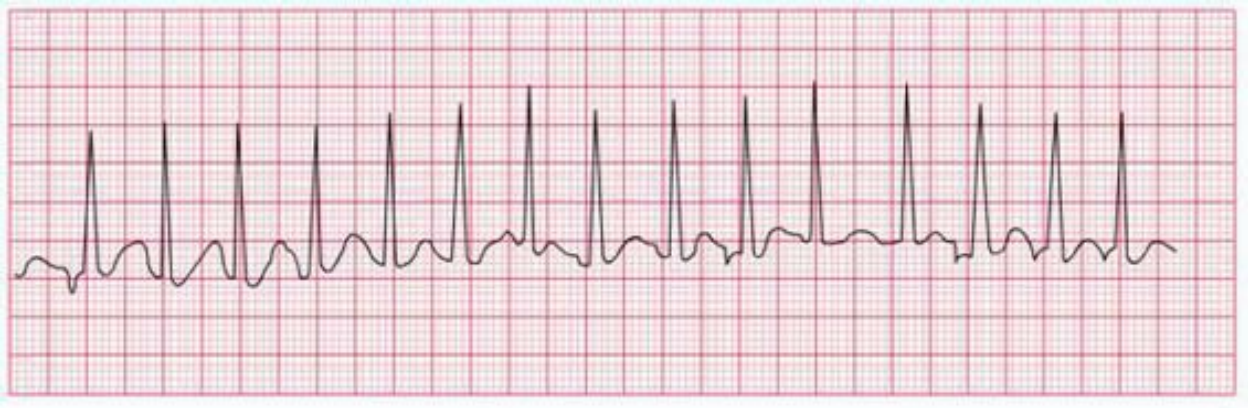


Fig 6.3 Multifocal atrial tachycardia

Basic characteristics are

Rhythm: irregular

Rate: usually between 100 to 150 bpm

P wave: *at least three different morphology*

PR interval: slightly varies from beat to beat

QRS complex: within normal limits

T wave: not uniform

QT interval: may or may not be measurable

Clinical significance: multifocal atrial tachycardia is mostly seen in patients with significant lung disease such as **chronic obstructive pulmonary disease (COPD)**.

Management: Treatment should be directed towards improving underlying disease for long-term management. For rate control, *calcium channel blockers* like **Verapamil** may be helpful. In patients with *no left ventricular dysfunction* or *coronary artery disease, agents such as* **Flecainide** or **Propafenone** may be useful.

Atrial Flutter

It is a form of supraventricular tachycardia with an atrial rate of *250 to 400 bpm*. An aberrant pathway causing re-entry of the impulse usually generates this type of rhythm, leading to recurrent atrial depolarization. The most characteristic pattern of atrial flutter is '**saw tooth**' shaped P waves called **flutter waves**. Because of the inherent safety mechanism within AV node, many of these impulses terminated at the level of AV node leading to 2:1 or 4:1 conduction ratio in the ventricle. Even if there is 2:1 block exists, ventricles may be firing up to 150 bpm and can create serious hemodynamic compromise.

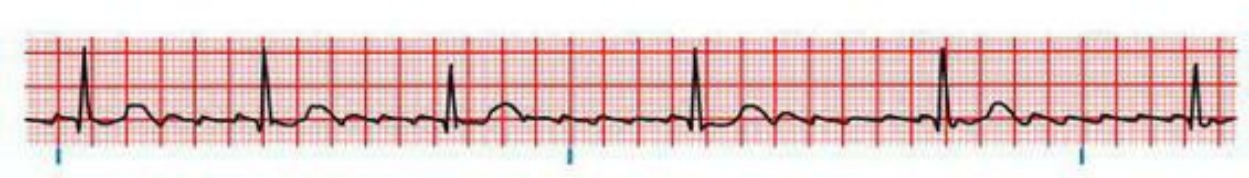


Fig 6.4 Atrial flutter with characteristic saw tooth flutter waves

Basic characteristics are

Rhythm: regular or irregular depending on AV nodal conduction pattern.

Rate: atrial rate of 250 to 400 bpm; ventricular rate usually 1/2 or 1/4 of atrial rate depend on the conduction ratio.

P wave: characteristic P wave in atrial flutter is called *flutter waves*. These flutter waves have unique '*saw tooth*' appearance and are very regular. There may be two or more flutter waves between two QRS complex.

PR interval: not measurable because of multiple flutter waves between QRS.

QRS complex: usually narrow and within normal limits.

T wave: mostly unidentifiable because of the flutter waves.

QT interval: indiscernible

Clinical significance: Atrial flutter is common in patients with *congenital heart disease, coronary atherosclerosis*, *valvular heart disease* etc. If untreated, this tachyarrhythmia can *increase myocardial workload* significantly, leading to *left ventricular dysfunction* and possible ischemia in presence of co-morbid conditions. Because of the pooling of blood within the atria secondary to tachycardia, *risk of thromboembolic events are*

high.

Management: Pharmacologic agents like *calcium channel blockers* (**Diltiazem**, **Verapamil**), *Digoxin* and *beta-blockers* may be helpful in controlling ventricular rate by slowing AV nodal conduction. However, electrical cardioversion after adequate anticoagulation may provide long-term solution.

Most common atrial flutter circuit is located in the right atrium around tricuspid valve annulus, which facilitates re-entry of impulses back to right atrium causing repeated depolarization. Knowledge of this anatomic location of re-entry circuit is extremely important in the treatment of atrial flutter with radiofrequency ablation, where an electrical roadblock is created in the circuit leading to termination of its re-entry.

Atrial Fibrillation

It is the most common sustained atrial arrhythmia characterized by *rapid*, *disorganized* and *irregular atrial activation,* most likely from *multiple ectopic foci*. This multi center stimulation of atria results in disorganized depolarization and practically a '*trembling*' or '*vibrating*' movement of the chamber. During this event, AV node acts as a natural protective mechanism for the ventricles. It blocks most of these erratic impulses and only allows smaller number of them to conduct down to the ventricles, leading to **controlled ventricular response** (**CVR**). However in some instances, AV node allows most of these fibrillatory waves to pass down to ventricles leading to **rapid ventricular response** (**RVR**). There are uneven baseline fibrillatory waves and irregular QRS complexes seen in EKG.

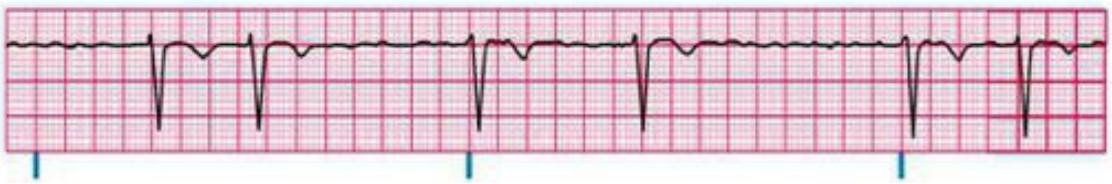


Fig 6.5 Atrial fibrillation

Basic characteristics are

Rhythm: irregularly irregular (both atria and ventricle)

Rate: *Atrial - above 400 bpm*, no distinguishable uniform P waves seen. Ventricle-

depending on the conduction block at AV node, it may be fast (greater than 100 in RVR) or slow (less than 100 in CVR)

P wave: *no distinct P waves*, only fibrillatory waves

PR interval: not measurable

QRS complex: narrow

T wave: mostly indiscernible

QT interval: not measurable

Clinical significance: most important clinical significance of atrial fibrillation are (1) **loss of atrial contractility** (2) **inappropriately fast ventricular response** (3) **loss of atrial appendage contractility** leading *to risk of clots formation* and subsequent *thromboembolic event*. Many patients are asymptomatic; however, palpitation, irregular pulse, hypertension, exercise intolerance, fatigue and pulmonary congestion are seen at times. In some occasions, patients will present with severe dizziness or syncope. This commonly happens in **paroxysmal** (short lasting) **atrial fibrillation,** where sinus node recovery time is unusually longer after termination of atrial fibrillation leading to short period of practical **asystole**.

Interestingly, in some patients with atrial fibrillation lead V1 shows flutter wave pattern rather than chaotic fibrillatory waves because the crista terminalis; which is a thick smooth surfaced portion of heart muscle at the opening of right atrial appendage that blocks fibrillatory waves from conducting to lead V1. Therefore, what lead V1 sees is only atrial activity on lateral aspect of right ventricle.

Classification of atrial fibrillation

Paroxysmal	Recurrent episodes that self terminate in *less than seven days*
Persistent	Recurrent episodes that last *more than seven days*
Permanent	Ongoing *long-term episode*

Box 6.1 Classification of Atrial fibrillation

Management: Aggressive management of atrial fibrillation is done by the use of **direct current** (DC) **cardioversion** if hemodynamic compromise is present. Otherwise treatment goals are (1) **rate control** (2) **prevention of thromboembolic event** (3) **rhythm control**.

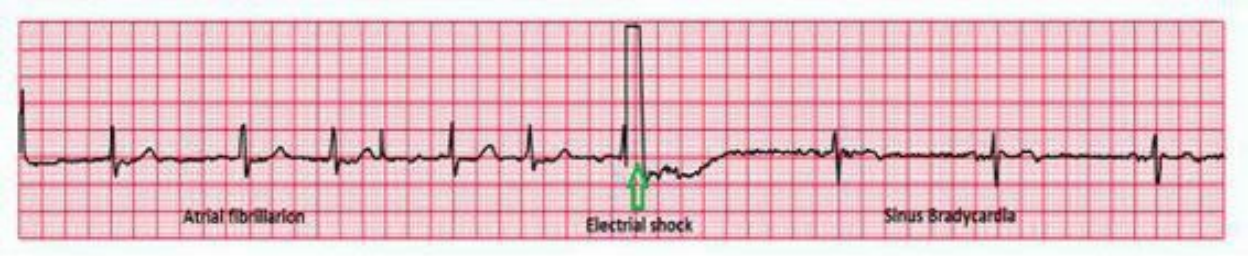


Fig 6.6 Cardioversion of Atrial fibrillation to Sinus rhythm

1 . **Rate Control**: In order to control ventricular response; *beta-blockers*, *calcium channel blockers* (non-dihydropyridines e.g. Diltiazem, Verapamil) and *digoxin* are useful.

2. **Prevention of Thromboembolic Event**: Immediate and long-term anticoagulation is extremely important in preventing thrombotic events like **pulmonary embolism** or **cerebrovascular accident**. Intravenous *heparin* or *low molecular weight heparin* (Enoxaparin) can be used in acute phase. **Coumadin** and **Dabigatran** (Pradaxa) are agents of choice for long-term anticoagulation. Risk of thromboembolism in the event of atrial fibrillation is increased by coexisting factors such as *congestive heart failure, hypertension, advanced age, diabetes mellitus and history of stroke* (known as **CHADS2** Score). **Aspirin** can be useful in patients with a low risk for clot formation.

3. **Rhythm Control**: Atrial fibrillation in and by itself is not dangerous as many other ventricular tachyarrhythmia. As long as the rhythm is not affecting hemodynamics, immediate management is directed towards *rate control and prevention of thromboembolism* . However in long run, *rhythm control* with conversion back to sinus rhythm with the use of chemical or electrical cardioversion is indicated. Most common anti-arrhythmic agents are **Amiodarone**, **Dronedarone** (Multaq), **Flecainide** and **Sotalol**. For further details of these drugs, please refer to chapter 14

Points to Remember!!!

- Premature atrial contractions are originated and sustained in ectopic pacemakers and abnormal re-entry circuits.
- Premature atrial contraction has a different shaped P wave because of its ectopic origin.

- Occasional PACs are benign; frequent ones are treated with beta-blockers, calcium channel blockers or digoxin.
- Paroxysmal atrial tachycardia by nature has short duration.
- Multifocal atrial tachycardia has different shaped P waves and is also called wandering pacemaker.
- Accelerated atrial rhythms diminish atrial kick.
- Along with nodal blockers, avoidance of stimuli and administration of adenosine or vagal maneuvers are other forms of treatment for paroxysmal atrial tachycardia.
- Multifocal atrial tachycardia is predominantly seen in patients with COPD.
- Antiarrhythmic agents such as flecainide and propafenone are contraindicated in patients with coronary artery disease and left ventricular dysfunction.
- Atrial flutter is produced by re-entry of impulses to the re-entry circuit mostly located on the right atrium.
- Saw tooth waves in atrial flutter are called flutter waves.
- Atrial flutter and fibrillation carry the same risk of having a thromboembolic event and therefore need anticoagulation.
- Rate controlling calcium channel blockers, beta-blockers and digoxin may be helpful in controlling ventricular response in flutter or fibrillation.
- Instead of uniform shape flutter waves, chaotic fibrillatory waves are seen in atrial fibrillation.
- Atrial fibrillation causes loss of atrial contractility, inappropriately fast ventricular response and loss of atrial appendage contractility.
- Risk of having a thromboembolic event in patients with atrial fibrillation can be accessed with CHADS2 scoring system.
- Rate control, prevention of thromboembolic event and rhythm control are basic goals of atrial fibrillation treatment.

Test Your Understanding

1. Which of the following mechanism is responsible for origin of atrial arrhythmia?

A Impaired contractility

B Problems with automaticity

C Impaired conductivity

D AV node dysfunction

2. The hallmark finding in premature atrial contraction is _____?

A Presence of multiple P waves between two adjacent QRS

B Wide and distorted QRS complex

C Premature PQRST complex

D Multiple R waves between two P waves

3. Which of the following physiologic mechanism is affected by atrial tachycardia?

A Loss of contraction of right ventricle

B Loss of contraction of left ventricular appendage

C Loss of atrial kick

D Impaired atrial filling during cardiac cycle

4. Treatment option for paroxysmal atrial tachycardia does not include_____?

A Adenosine

B Vagal maneuvers

C Caffeine ingestion

D Beta blockers

5. Which of the following statement is not true regarding multifocal atrial tachycardia?

A It has uniform shaped QRS complex

B It has uniform shaped P waves

C There is P waves with different morphology

D It is commonly seen in COPD

6. Which of the following represent the hallmark symptom of atrial flutter?

A Bizarre and undulating baseline

B Chaotic and irregular P waves

C Presence of uniform shaped flutter waves

D Atrial contraction above 400 bpm

7. Which of the following statement accurately identifies persistent atrial fibrillation?

A Episodes that last less than a day

B Episodes last more than a week

C Atrial fibrillation lasting less than an hour

D Episodes lasting a few years

8. Which of the following is not considered as a major risk factor for thromboembolic event in atrial fibrillation?

A Age greater than 75 years

B Congestive heart failure

C Presence of osteoarthritis

D History of stroke

9. Which of the following is not a treatment option for Afib patients with higher risk of stroke?

A Use of Coumadin

B Use of Dabigatran

C Use of Ibuprofen

D Use of heparin

10. Which of the following is not a goal of treatment in atrial fibrillation?

A Control ventricular response

B Acceleration of atrial response

C Prevention of blood clots

D Converting back to sinus rhythm

Answers

1. B Problems with automaticity
2. C Premature PQRST complex
3. C Loss of atrial kick
4. C Caffeine ingestion
5. B It has uniform shaped P waves
6. C Presence of uniform shaped flutter waves
7. B Episodes last more than a week

8. C Presence of osteoarthritis

9. C Use of Ibuprofen

10. B Acceleration of atrial response

Chapter 7

AV Junctional Rhythms

The junctional rhythm or junctional escape beat is a *pacemaker impulse originating within the atrioventricular node*. As mentioned in the initial chapters, every cardiac cell has the capacity of automaticity, which is the ability to generate its own electrical impulse and the AV junction is of no exception. The rate at which the AV node produces impulse is lower than that of the SA node and is about **40 to 60 beats per minute**. Junctional rhythms are usually seen when there are *no electrical impulses coming down from the SA node or anywhere in the atria*. Since the AV node is sitting in between the atria and the ventricle, electrical impulses originating at the AV node can travel *forward to the ventricles (ante grade conduction) or backward up to the atria (retrograde conduction)*.

There are three distinct regions within the AV node that produce electric impulses and are **high**, **mid and low** regions. An Impulse producing at the **high AV node** will travel to the atria first and creates atrial contraction before it produces contraction of the ventricles. The resulting EKG complex will have an **inverted P wave** because of the *backward flow of the impulse*. Since the atria are contracting prior to the ventricle, this inverted P wave will be positioned before QRS complex.

In the case of an impulse originating from the mid region of AV node, *P wave is*

usually absent because it is buried within the QRS complex. In a low AV node impulse, electricity spreads to the ventricle before the atria resulting in an inverted *P wave after QRS complex*.

There are three distinct presentations of P waves in junctional rhythms such as inverted, absent or trailing. A short PR interval with any one of the characteristics P wave pattern is the hallmark of the junctional rhythm.

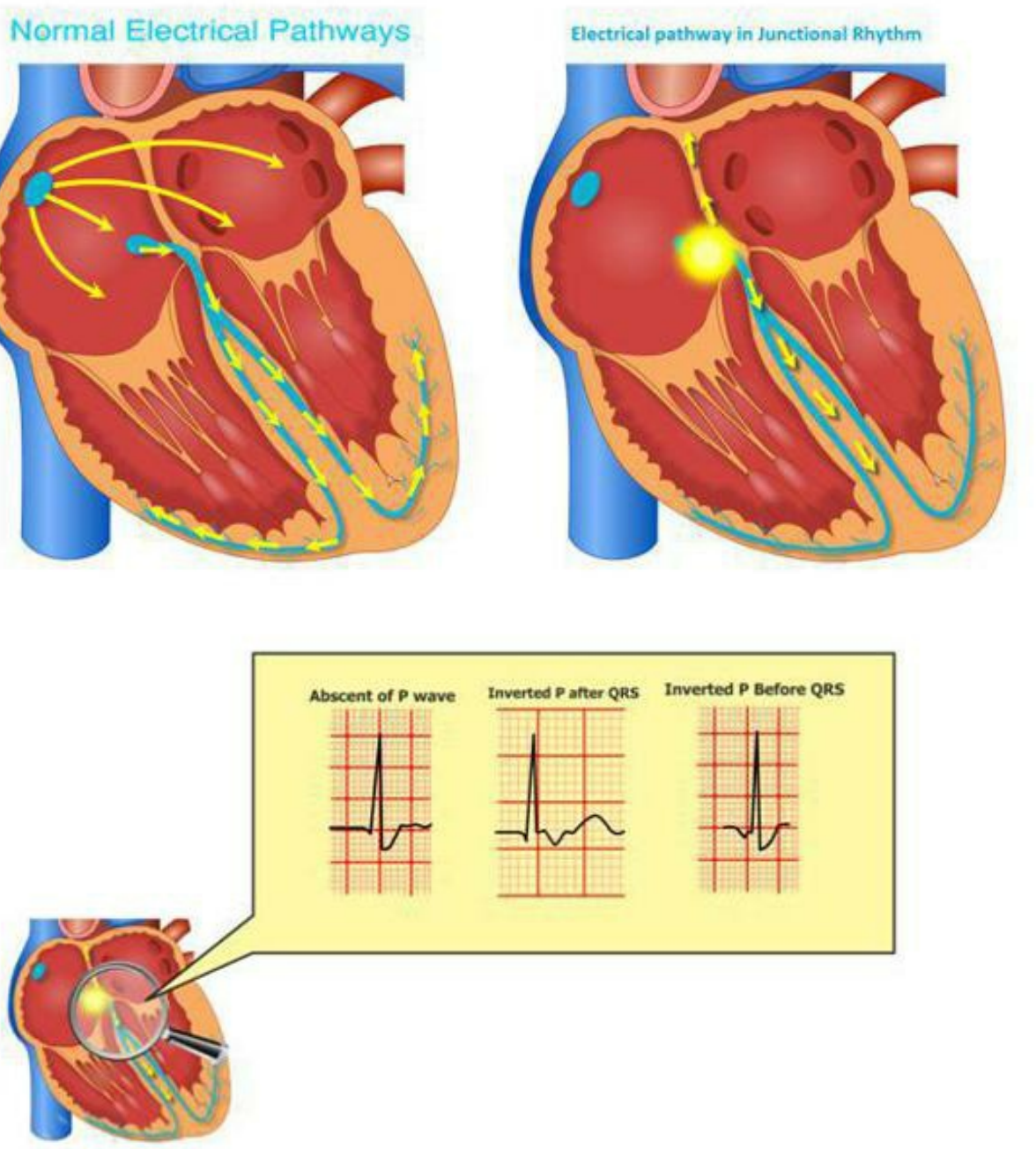


Fig 7.1 Junctional rhythm with three different morphology of junctional P waves

Since the AV node is sitting midway between the atria and the ventricle, *PR interval* in these rhythms are *lower than normal* (less than 0.12 seconds). This is because the impulse doesn't take much longer to get from its origin to the AV node, which is represented by PR interval. In nutshell, *a short PR interval with the characteristic inverted, trailing or absent P wave* is the hallmark of junctional rhythm.

Premature Junctional Contraction (PJC)

These rhythms are originating from an *ectopic focus within the AV junction*. Mostly, PJC's are caused by *coronary artery disease*, *acute myocardial infarction*, *digitalis toxicity*, *hypokalemia* or *chronic lung disease*. This beat occurs before a normal sinus rhythm and it depolarizes the *atria retrograde* (backwards) and the *ventricle antegrade* (forward), producing an inverted P wave and a normal QRS complex. As mentioned earlier, depending on the location of impulse origin within the AV node, the P wave can be inverted, absent or trailing.

Basic characteristics are

Rhythm: underlying regular rhythm, interrupted with the PJC.

Rate: usually normal.

P wave: normal except during PJC; inverted, absent or trailing P wave during ectopic beat.

PR interval: normal except during PJC where it is *shorter* than 0.12 sec if P wave is present.

QRS complex: normal.

T wave: normal, unless P wave superimposes T wave.

QT interval within normal limits, since ventricular contraction is unaffected.

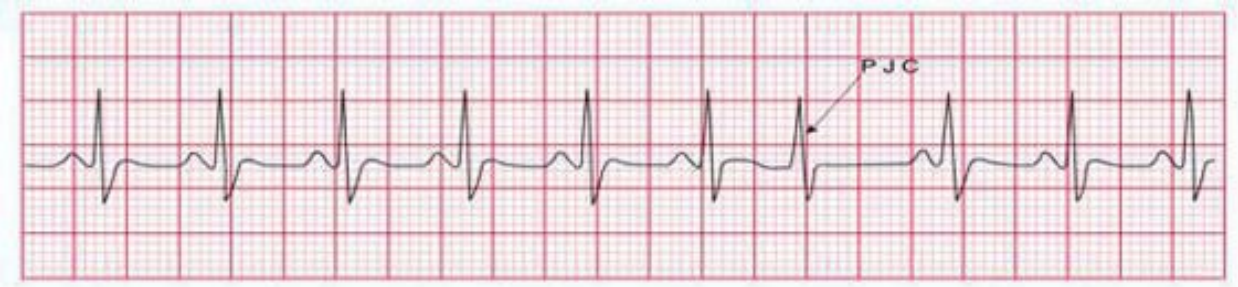


Fig 7.2 Premature junctional contraction

Clinical significance: most of the time, PJCs are benign and unnoticed. However, in the event either a premature beat produces under filling of the ventricle or it doesn't conduct down the ventricle, patient may feel skipped beat or lightheadedness. *No treatment is needed for asymptomatic PJC*. Whenever encounter hemodynamically significant PJCs, underlying reasons need to be investigated.

Junctional Rhythm

It is also known as the **Junctional escape rhythm** with a heart rate of **40** to **60 bpm** with all the characteristics of junctional type P waves throughout the rhythm. Common causes for junctional rhythms are *increased vagal tone*, *toxicity with beta and calcium blockers*, *coronary artery disease*, *degenerative changes in SA node* etc.

Junctional Bradycardia

The major difference of junctional bradycardia from other junctional rhythms is that, the rate of cardiac contraction will be *less than 40 bpm*. Most of the time, this slow heart rate may result in hemodynamic compromise. Therefore, aggressive management with the use of *transcutaneous* or *intravenous pacemaker* is the treatment of choice for hemodynamically significant junctional bradycardia. *Atropine* may also be useful in this setting.

Accelerated Junctional Rhythm

This rhythm is known as 'accelerated' because it fires impulses *above the inherent rate of junctional tissue*, which is 40 to 60 bpm. Similar to other junctional impulses, they are produced as a **coping mechanism of heart** when the atrial impulses terminate. Along with other characteristics of the junctional rhythm, the rate will be between **60 to 100 bpm**.

Junctional Tachycardia

This type of rhythm happens when an *ectopic junctional focus starts firing at rate above 100 bpm*. This is a form of supraventricular tachycardia in which *enhanced automaticity of the AV node* generates fast impulses that essentially suppress the SA node.

Basic characteristics are

Rhythm: regular

Rate: *greater than 100*

P wave: characteristic *inverted, absent or trailing P wave*.

PR interval: *less than 0.12 second*

QRS complex: within normal limits

T wave: normal unless a P wave hides within it.

Clinical significance: retrograde conduction of P wave along with tachycardia can diminish cardiac output. If so, *beta-blockers*, *calcium channel blockers* or *adenosine* maybe the treatment of choice.

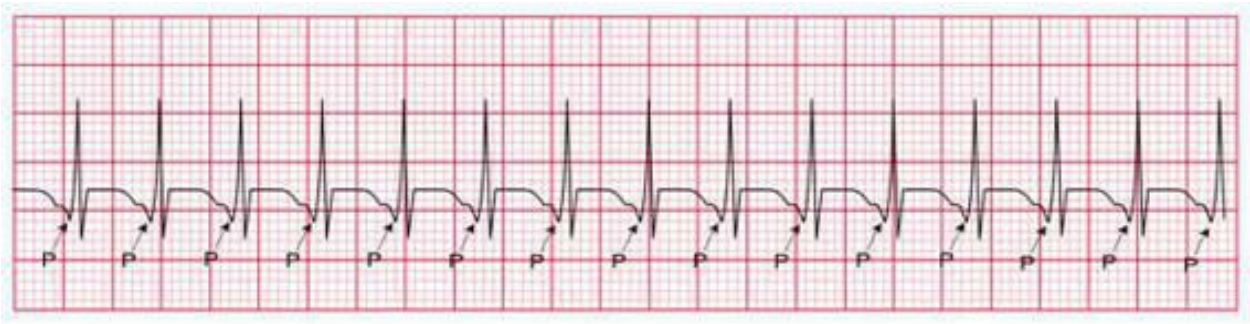


Fig 7.3 Junctional tachycardia

AV Nodal Re-entry Tachycardia (AVNRT)

This is the most common type of paroxysmal supraventricular tachycardia. This rhythm originates because of a peculiar anatomic structure within the AV node. The AV node comprised of myocardial fibers with two distinct depolarization properties. One group has **fast pathway** and *longer refractory time* and the other one has **slow pathway** with *shorter refractory period*. During *normal sinus rhythm*, *only fast pathway* is manifested in an EKG even though impulses pass through both fast and slow pathways.

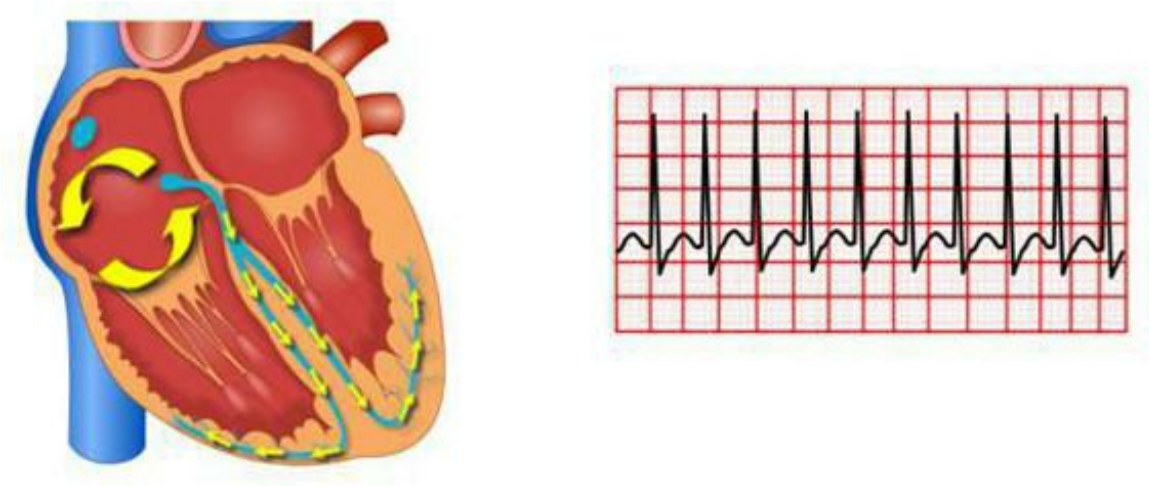


Fig 7.4 AVNRT

A premature atrial contraction (PAC) originating at a critical moment during cardiac cycle may be blocked in the fast pathway because of their longer refractory period. However, a relatively shorter refractory period of slow pathway may allow this impulse to conduct through slow pathway alone. During this slow conduction phase, the fast pathways comes out of their refractory period and therefore available for impulse conduction. The impulse that is conducting through the slow pathway may then get in to the fast pathway circuit and start retrograde conduction, leading to a vicious *re-entry pathway within the AV node*.

The characteristic EKG finding in AVNRT is the *long PR interval of PAC initiating this rhythm*, suggesting *impulse conduction through slow pathway*. Similar to any other junctional rhythm, P waves may either be inverted (retrograde conduction) or absent (buried in T wave).

Basic characteristic are

Rhythm: regular

Rate: *120-250 beats per minute*

P wave: inverted, absent or trailing

PR interval: *longer in the initiating impulse*. Unable to measure if P wave is absent.

QRS complex: narrow and regular

T wave: regular or distorted by buried P wave.

QT interval: normal or varying depending on the T wave.

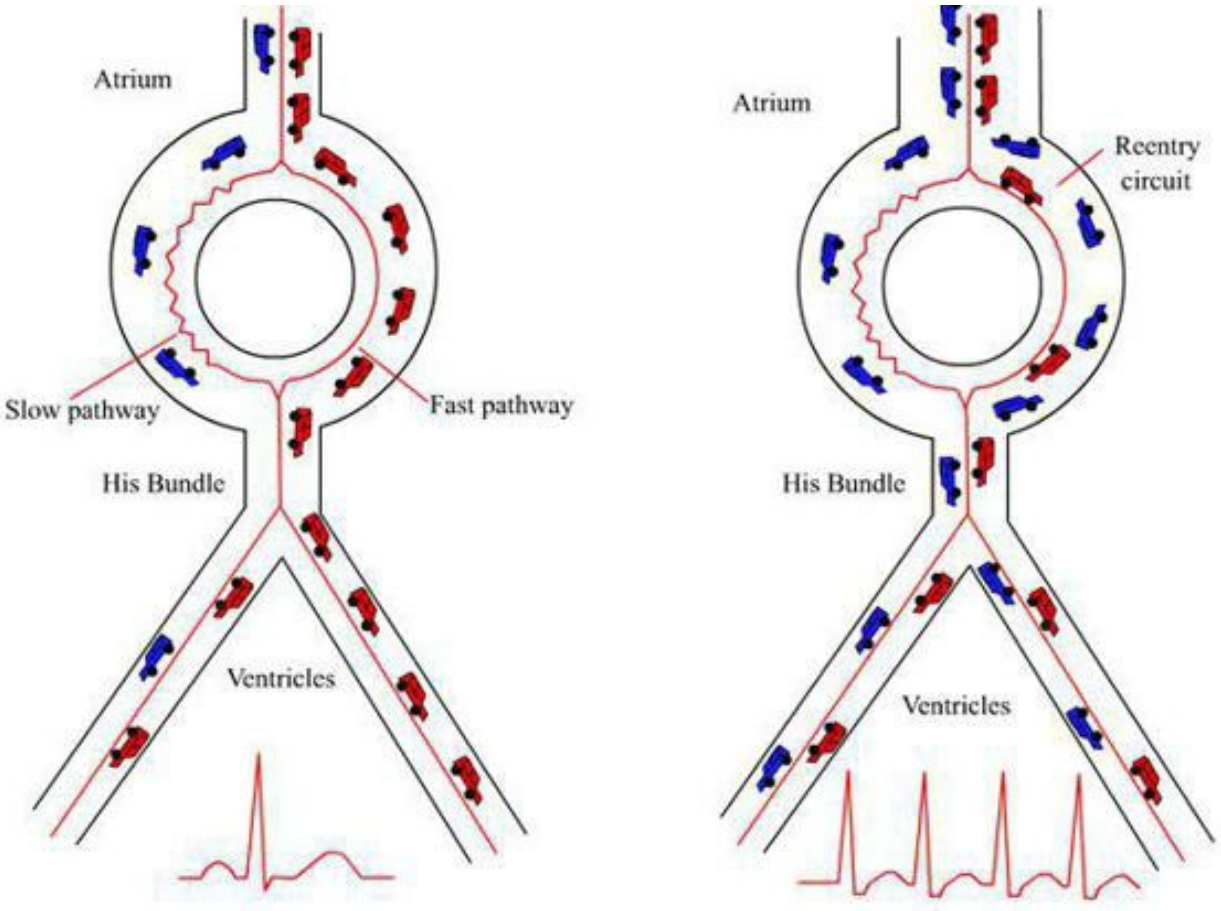


Fig 7.5 Slow and fast pathway for AVNRT

Clinical significance: In the absence of structural heart disease, AVNRT usually is asymptomatic. However, in presence of coexisting structural heart disease this rhythm may produce *hypotension* or *syncope*.

Management: Treatment is directed towards altering conduction within AV node by employing *vagal maneuvers*, *adenosine*, *beta-blockers* or *calcium channel blockers*. If the rhythm is refractory to treatment, **synchronous direct current cardioversion** is the plan of choice. For patients with recurrent AVNRT refractory to medication and cardioversion, **radiofrequency ablation** of slow pathway is an option with high success rate.

Tachycardia Associated with Accessory Pathways

Wolff- Parkinson- White Syndrome (WPW)

Under normal circumstances, an impulse originating from atria travels down to the ventricle only through the single AV nodal gateway. However, in some situations there is existence of abnormal connection between the atria and the ventricle called **accessory pathway.** This short circuit complicate the matter by either *conducting some impulses to the*

ventricle before AV node does it or *allowing impulses to enter back to the atria from the ventricle*. In WPW syndrome, this pathway is called **Bundle of Kent**. WPW is also known as **pre-excitation syndrome**. This re-entry circuit can create **re-entry tachycardia**.

When an antegrade conducting accessory pathway exists between the atria and the ventricle, some impulses bypass the AV node through this fast pathway and therefore result in a short PR interval. Since this accessory pathway does not extend throughout the ventricle like the **His-Purkinje system**, impulse travelling through accessory pathway *cannot initiate a complete ventricular contraction.* So in the resulting EKG, a characteristic *slurring of initial portion of QRS* known as **Delta wave** will be present in some patients.

In WPW syndrome, the *accessory pathway can acts as a re-entry circuit* for the impulse and allows it to return to the atria and can create tachyarrhythmia. The retrograde activation of the atria through accessory pathway is known as **echo beat**. Compared to the AV nodal pathway, this *accessory bundle serves as a low resistance pathway* for impulse conduction. In patients with underlying atrial fibrillation, because of the low resistant conduction in this accessory channel fibrillatory waves can travel down to the ventricles bypassing the AV node. This leads to formation of lethal ventricular fibrillation.

Atrioventricular re-entrant tachycardia (AVRT) can have either narrow or wide complex QRS. AVRT with narrow complex tachycardia is called **orthodromic AVRT** and the one with wide QRS complex as **antidromic AVRT**. In narrow complex AVRT (Orthodromic), *antegrade conduction is through AV node* and therefore there is *no Delta wave during sinus rhythm*. However in wide complex AVRT, forward conduction to the ventricle is through accessory pathway leading to slurred QRS (Delta wave).

Common drugs used for treatment of tachycardia such as beta-blockers, calcium channel blockers or digoxin impart resistance to the AV nodal conduction. If they are used in presence of some AVRT, it may lead to downward conduction of majority of impulses through *path of least resistance*, which is the accessory pathway in this situation. This may produce ventricular tachycardia (V-tach) or fibrillation (V -Fib). Therefore, *it is very important to identify presence of Delta wave before treating tachycardia*.

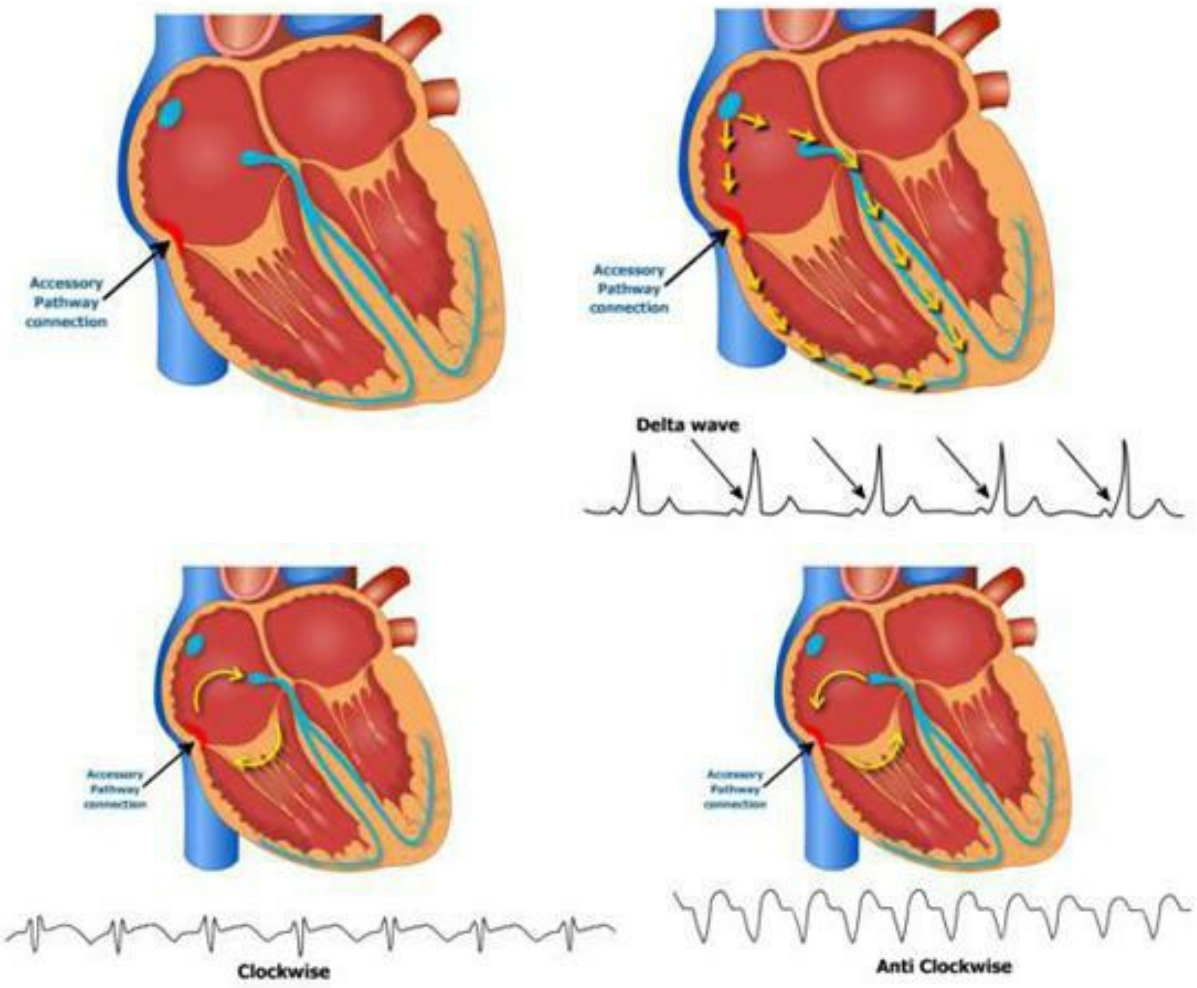


Fig 7.6 WPW Syndrome (Accessory pathways and possible mechanism of tachycardia)

Atrial fibrillation with WPW can create a wide QRS complex tachycardia resembling V-tach. The major differentiation between ventricular tachycardia and atrial fibrillation with WPW is that *ventricular tachycardia is usually regular*; *however*, *latter is irregular*. WPW syndrome is associated with congenital anomalies such as **Ebstein anomaly**, **mitral valve prolapse** and **hypertrophic cardiomyopathy** (**HCM**). These patients generally have AV nodal re-entry tachycardia, atrial fibrillation or flutter.

Asymptomatic patients with WPW do not need any treatment. Definite management of WPW is a **radiofrequency catheter ablation** of the re-entry pathway and is the primary treatment of choice if possible. Depending on the type of AVRT, *intravenous beta-blockers* or *Procanamide* can be useful for suppression of tachycardia in acute phase. Procanamide is of special importance because of its ability to depress conduction and prolong refractoriness except that of the AV node. For patients with infrequent episodes of AVRT, *Propafenone* and *Flecainide* can be used in long-term.

Ashman's Phenomenon

This is a form of aberrant conduction commonly associated with Atrial fibrillation . A premature atrial impulse forms after a long RR interval will be conducted aberrantly (through an alternate pathway within the ventricle) because one bundle branch is not completely recovered from the previous impulse. In general, Right bundle has a slower refractory period than that of Left bundle and therefore the *resulting impulse has a Right bundle branch block configuration* of **rsR'** pattern. It usually results in a small number of wide complex beats on an otherwise narrow complex rhythm.

There is so called Long Short rule for Ashman Phenomenon as follows. *The earlier in the cycle the PAC occurs and the longer the previous cycle, more likely the PAC will be conducted through aberrant pathway within the ventricle*. Clinical significance of Ashman' phenomena is that it should be differentiated from serious preexcitation conditions such as WPW syndrome.

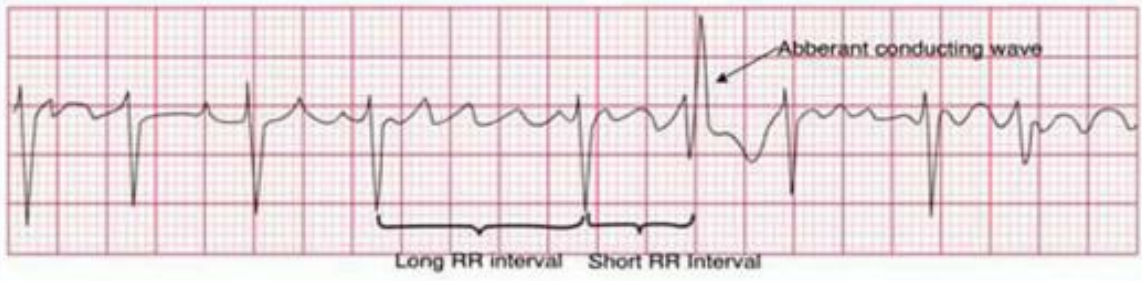


Fig 7.7 Aberrant conduction with Ashman's phenomenon

Box 7.1 Ashman's phenomenon

Points to Remember!!!

· Junctional rhythms originate within the atrioventricular node and are characterized by inverted or absent or trailing P waves and a short PR interval.
· Rate of junctional impulse is between 40 to 60 beats per minute.
· Common causes of junctional rhythms are increased vagal tone, drug toxicity, degeneration of SA node and coronary artery disease.

· Accelerated junctional rhythm is a junctional rhythm with heart rate greater than 60 beats per minute.
· Junctional tachycardia has rate of greater than 100bpm.
· AV nodal re-entry tachycardia (AVNRT) is the most common type of paroxysmal supraventricular tachycardia.
· Characteristic EKG finding in AVNRT is the long PR interval in PAC, which initiate this arrhythmia because of involvement of the slow conduction pathway in the AV node.
· AV nodal blocking agents, vagal maneuvers, DC cardioversion or radiofrequency ablation of the slow pathway are the possible treatment options for AVNRT.
· WPW syndrome is caused by abnormal conduction through accessory pathway known as bundle of Kent.
· WPW is also called pre-excitation syndrome because of the activation of ventricle through accessory pathway before the AV node.
· Premature activation of ventricles through accessory pathway cause formation of slurred QRS complexes called Delta waves.
· In certain situations like in atrial fibrillation, the accessory pathway acts as a low resistance pathway for downgrade moment of impulses and can cause ventricular fibrillation.
· Narrow complex QRS tachycardia in atrio ventricular re-entrant tachycardia (AVRT) is called orthodromic AVRT and one with wide complex called antidromic AVRT.
· It is important to identify presence of accessory pathways before treating re-entry tachycardia, because of the possibility of causing ventricular fibrillation by blocking AV node and facilitate conduction through accessory circuit.
· Radiofrequency ablation is a definite and most desired treatment option for atrio ventricular re-entry tachycardia.
· Procanamide and intravenous beta-blockers are preferred treatment for acute management of re-entrant tachycardia.
· Propafenone and flecainide can be used for prevention of infrequent AVRT.

Test Your Understanding

1. Which of the following is not a characteristic of junctional rhythm?

A Heart rate in 60 to 80bpm
B Inverted P wave
C Short PR interval
D Heart rate in 40 to 60 bpm

2. Junctional rhythm may be caused by _____?

A Decreased vagal activity

B Increased vagal tone
C Use of atropine
D Discontinuation of beta-blockers

3. Which of the following represent the heart rate of accelerated junctional rhythm?
A 20 to 40 bpm
B 40 to 60 bpm
C 60 to100 bpm
D Above 100 bpm

4. Which of the following pathway is actively involved during normal AV nodal conduction?
A Slow pathway with long refractory period
B Fast pathway with shorter refractory period
C Fast pathway with long refractory period
D Short pathway with shorter refractory period

5. Treatment for AVNRT does not include_____?
A Vagal maneuvers
B Adenosine
C Atropine
D Beta-blockers

6. Which of the following represents the accessory pathway of WPW syndrome?
A Bundle of His
B Thorel's tract
C Bundle of Kent
D Buckman's pathway

7. WPW syndrome EKG is characterized by_____?
A J point elevation
B Delta wave
C Multiple P waves
D Inverted T waves

8. Which of the following is the most desirable treatment option for WPW syndrome?
A Amiodarone
B Oral beta-blockers
C Radiofrequency ablation
D Use of Flecainide

Answers

1. A Heart rate in 60 to 80bpm

2. B Increased vagal tone

3. C 60 to 100 bpm

4. C Fast pathway with long refractory period

5. C Atropine

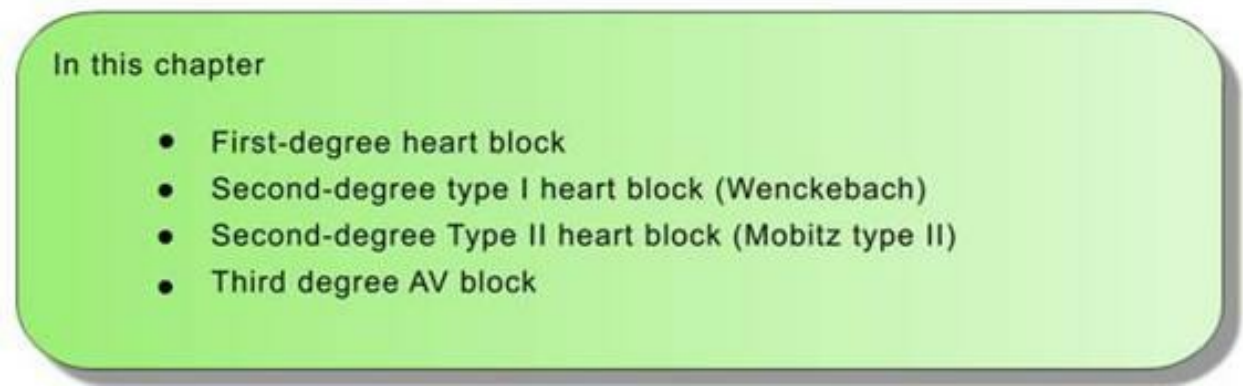
6. C Bundle of Kent

7. B Delta wave

8. C Radiofrequency ablation

Chapter 8

Atrio Ventricular Block



In normal conduction system of the heart, impulses originating from the SA node spread across the atria and channels down to the AV node. At the AV node, these impulses are delayed due to so-called **decremental conduction** and thereby allowing atrial depolarization. Hence, the AV junction serves as the *natural regulator* or *signal point* of ventricular response with its inherent delaying mechanism. However during atrioventricular block, this *delaying mechanism becomes so profound* that either the *delay at the AV junction increases* or some of the atrial beats *does not conduct* down to the ventricle.

There are varying degrees of heart blocks identified. The site of block can be either in the AV node itself or sometimes at the bundle branches. In this chapter, we will discuss about the heart block involving AV node. Varying degrees of bundle branch blocks are discussed later in 12 lead EKG interpretation session. Depending on the severity of the AV block, it can be **first-degree heart block**, **second-degree type I** (Wenckebach), **second-degree Type II** (Mobitz type II) and **third-degree heart block** (AV dissociation).

First-Degree Heart Block

More than usual delay in conduction through the AV node creates the hallmark EKG

change in first-degree heart block as *elongated PR interval*. Even though an increased delay exists, all the impulses get through AV node and complete its conduction.

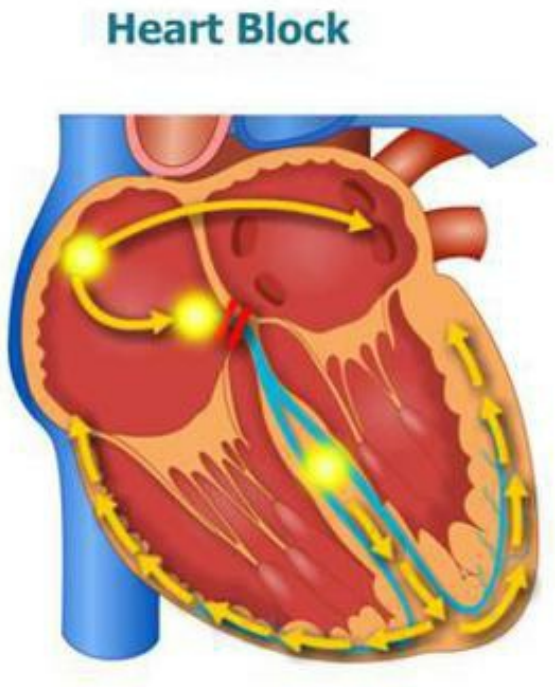


Fig 8.1 Heart block

Basic characteristic are

Rhythm: regular

Rate: both atrial and ventricular rates within normal limits.

P wave: normal and uniform. One P wave for each QRS complex.

PR interval: *greater than 0.2 second*.

QRS complex: normal and uniform.

T wave: within normal limits.

QT interval: unaffected.

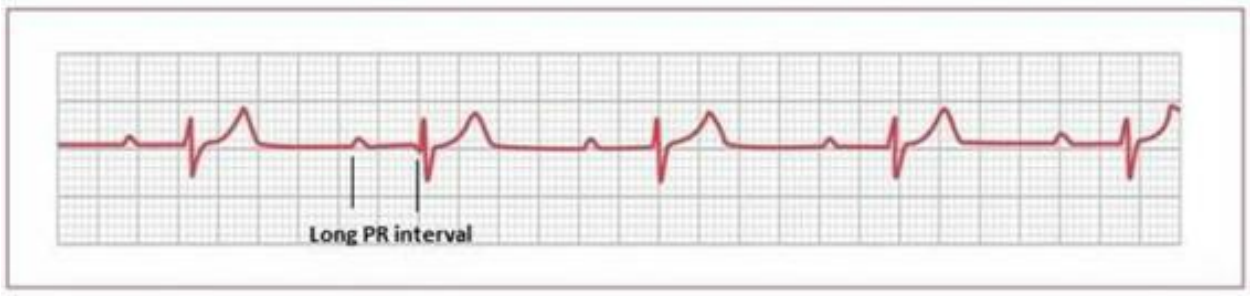


Fig 8.2 First-degree heart block EKG (Note the presence of long PR interval)

Clinical significance: First-degree heart block *by itself is benign*. It is usually caused by *coronary artery disease* causing *AV nodal ischemia*, medications like *beta-blockers*, *calcium channel blockers* and *digoxin* etc. Treatment of underlying causative factor is the management strategy.

Second-Degree Type I Heart Block (Wenckebach)

In this type of heart block, there is a *periodic conduction failure* within the AV node causing *PR interval to progressively lengthen until a drop in QRS complex*. After this dropped beat, the cycle repeats forming groups of beats called **footprint of Wenckebach**. Unlike first-degree heart block, *all the atrial beats does not conduct to ventricle during* Wenckebach phenomena.

Basic characteristics are

Rhythm: regularly irregular, especially at dropped QRS.

Rate: atrial rate 60 to 100; ventricular rate varies with degree of block.

P wave: normal and uniform.

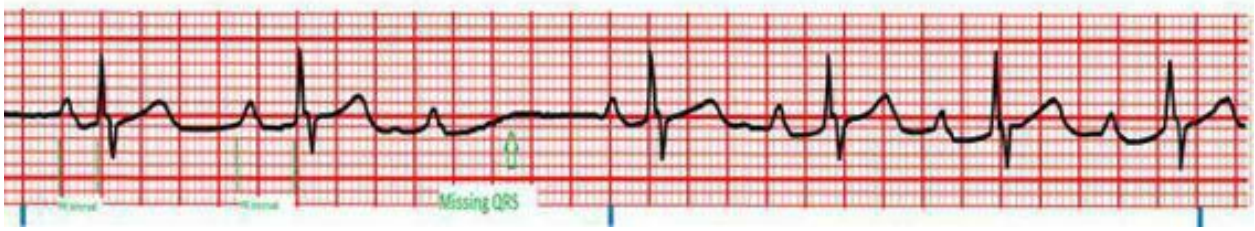


Fig 8.3 Second degree type I Heart block

PR interval: *varies from beat to beat with a progressive lengthening*, *until a complete drop of QRS*.

QRS complex: normal and uniform except during missed beats.

T wave: normal and is absent in missed QRS.

QT interval: not measurable during missed beat.

Clinical significance: second-degree type I heart block has the *same causative factors as first-degree heart block*. Mostly, patients are asymptomatic; however for symptomatic patients, they may need *transcutaneous* or *transvenous pacing*. This rhythm has a small chance of progressing into more serious type of heart blocks and therefore requires close observation.

Second-Degree Type II Heart Block (Mobitz Type II)

This type of heart block is more serious than Wenckebach. In these patients, the location of block can be either at the AV node or at the bundle branch level. Characteristic EKG shows *a regular rhythm with constant PR interval from beat to beat until a* **sudden drop of complete QRS complex**. There is a high likeliness that this *rhythm can progress to the third-degree heart block* and therefore needs immediate attention. Most of the patients have coexisting bundle branch blocks with a wide QRS complex. Occasionally, the block can be seen in regular pattern such as 2:1 or 3:1 conduction; where the QRS drops after every two or after every three P waves respectively.

Basic characteristics are

Rhythm: regular atrial rhythm and irregular ventricular rhythm.

Rate: atrial rate usually within normal limits; however, ventricular rate is lower than the atria.

P wave: normal and uniform.

PR interval: unable to determine at missed beats.

QRS complex: normal and uniform except in missed beats and wide if a bundle branch block present.

T wave: normal size and configuration except in missed beats.

QT interval: not measurable during missed beats.

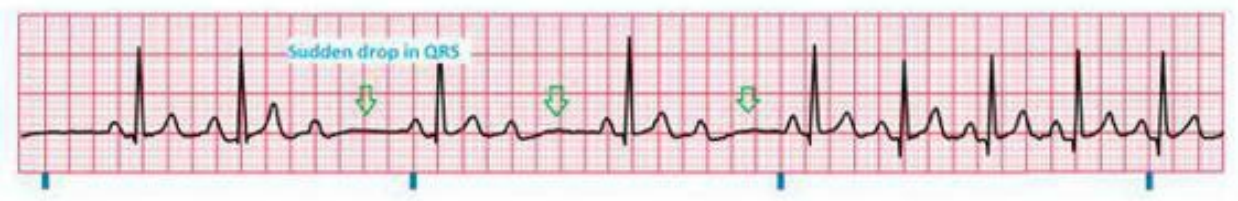


Fig 8.4 Mobitz type II

Clinical significance: If left untreated, this rhythm can progress into *serious AV dissociation*. Mobitz type II heart block is usually caused by *hypoxia*, *myocardial infarction*, *conduction system disturbance* etc. Depending on the extent of block in these patients, it may cause significant reduction in cardiac output. Symptomatic bradycardia with concomitant conduction block may respond better with *epinephrine than atropine*. Transvenous or transcutaneous pacing is the other available option for immediate treatment with transition to permanent pacemaker implantation depending on the reversibility of underlying causes.

Third Degree AV Block (AV Dissociation)

This is the most serious type of heart block. In natural conduction system of the heart, the atria contract first and send blood down to the ventricles and then the ventricles contract. This ensures optimal cardiac output. During third-degree heart block, there is no electrical connection between upper and lower chambers and therefore there is **no synchronized contraction**. Here, atrium fires impulses from the SA node at its own rate and contract accordingly. At the same time, since there is no electrical impulse from upper part of the conduction system, an ectopic focus within the ventricle start generating escape rhythm. This eventually leads to *atrial contraction at one rate* and *ventricular contraction at a different rate*. Since there is no synchronization between these chamber contractions, serious hemodynamic compromise can occur immediately.

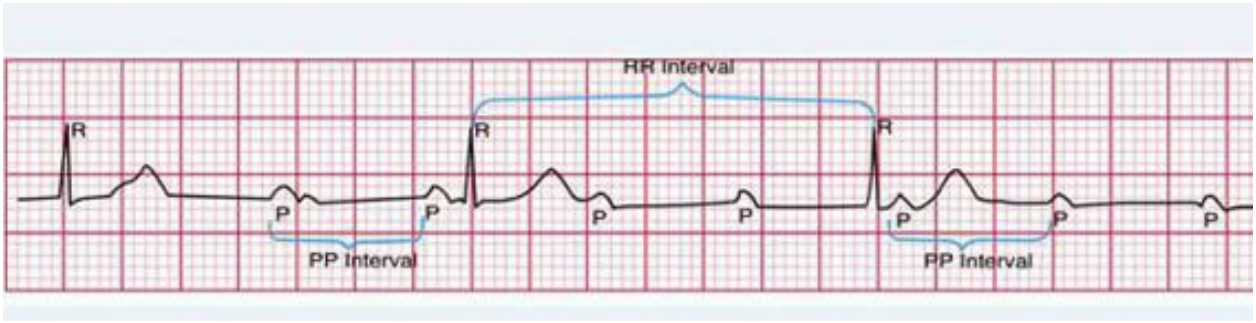


Fig 8.5 Third degree heart block

Basic characteristics are

Rhythm: grossly irregular; however, regular atrial and ventricular rhythms.

P wave: regular and uniform; however, not associated with QRS complexes. *P-P interval is constant*. Sometimes it gets buried in to QRS or T wave.

PR interval: not measurable and there *is no corresponding QRS for each P wave.*

QRS complex: regular and uniform. Narrow or wide morphology depends on presence of bundle branch block. *R-R interval constant.*

T wave: normal and uniform.

Clinical significance: AV dissociation cause significant hemodynamic compromise and therefore patients are usually symptomatic. Third degree heart block is usually caused by the same causative factors as in Mobitz type II heart block. *Transcutaneous* or *transvenous pacing* is a definite choice for immediate treatment. These patients may need permanent pacemaker implantation for long term, unless the causes are reversible.

Points to Remember!!!

- First-degree heart block is characterized by an elongated PR interval than normal because of a delay in conduction at the AV node.
- Causative factors for the first-degree heart block are AV nodal ischemia and toxicity from AV nodal blocking agents.
- In second-degree type I (Wenckebach) heart block, there is progressive prolongation of PR interval until a drop in QRS complex.
- In second-degree Type II (Mobitz type II) there is a sudden drop in QRS complex with no warning signs.
- Second-degree Type II heart block needs urgent attention because of its tendency to progress into third degree heart block.
- In third degree heart block, atrio ventricular contraction is asynchronous.
- In third degree heart block, P-P and R-R interval are constant; however, there is no association between occurrence of P and R waves.
- Immediate transcutaneous or transvenous pacing with advancement to permanent pacemaker implantation is the mode of treatment for third degree heart block.

Test Your Understanding

1. Which of the following statement is true for second-degree type I heart block?

A Constant PR interval throughout the rhythm
B Constant PR interval until a drop in QRS complex
C Progressive lengthening of PR interval until a complete drop in QRS
D Elongated QT interval throughout the rhythm

2. Which of the following represents the value of PR interval in first-degree heart block?

A 0.15 second
B 0.12 second
C 0.19 seconds
D 0.24 seconds

3. Which of the following is not accurate for second-degree Type II heart block?

A QT interval may be constant from beat to beat
B There is one P wave for every QRS complex
C There is complete PQRST complex missing intermittently
D QRS complex are missed intermittently

4. Which of the following is the most detrimental effect of third degree heart block?
A Absence of atrial contraction
B Absence of ventricular contraction
C Formation of blood clots
D Atrioventricular dissociation

5. Treatment options for third degree heart block include_____?
A Digoxin
B Amiodarone
C Transcutaneous pacing
D Defibrillation

Answers

1. C Progressive lengthening of PR interval until a complete drop in QRS
2. D 0.24 seconds
3. C There is complete PQRST complex missing intermittently
4. D Atrioventricular dissociation
5. C Transcutaneous pacing

Chapter 9

Ventricular Rhythms

In this chapter

- Premature ventricular contraction (PVC)
- Idioventricular rhythm
- Agonal rhythm
- Accelerated Idioventricular rhythm
- Ventricular tachycardia
- Torsades de pointes
- Long/short QT syndrome
- Ventricular fibrillation
- Asystole

Ventricular rhythms are originated from an ectopic focus within the ventricle. Since the propagation of this impulse to the myocardium and resulting ventricular depolarization are sluggish compared to its normal counterpart, these rhythms generate *wide and bizarre QRS complexes* with duration **more than 0.12 seconds**. Because of the fact that it originates in the ventricle, most of these impulses doesn't have an atrial component and some of them have retrograde conduction to atria. Some of this retrograde conduction from ventricular beats combined with antegrade SA node conduction creates **fusion beats**. Fusion beats can be distinguished from other PVCs because, *it happens at the exact timing as a normal SA nodal beat* supposed to occur and has *different shape* compared to other PVCs.

Because of the disorganized ventricular depolarization, the *T wave usually assumes opposite direction of the QRS complex* during ventricular rhythms. Even if the atrial component is present, resulting P wave will be buried within the wide QRS complex. Because of the lack of effective atrial contraction and disorganized ventricular depolarization, these rhythms may not provide adequate cardiac output.

Ventricular pacemaker cells have an inherent pacing rate of **20 to 40 bpm**. However during certain situations, they can produce ventricular rate up to 200 bpm because of the presence of re-entry circuits. These tachyarrhythmias produce *no effective cardiac output* and therefore are detrimental to the person's life

Premature Ventricular Contraction (PVC)

Premature ventricular contractions are ectopic beats originate within the ventricle and are mostly benign in nature. They may originate from a single ectopic focus in the ventricle or from multiple foci. If they originate from a single focus, they are uniform in appearance

(**unifocal**). Waves with varying morphology (Multifocal) are seen in rhythms originating from multiple foci. Sometimes, they are isolated and at times occur at regular intervals such as every other beat (**bigeminy**) or every third beat (**trigeminy**). At times, they come in pairs known as **couplets**. If there are *more than three beats in a row*, it is termed as a **run of VT**. Since the retrograde conduction of ventricular impulse can cancel out atrial stimuli coming down through the AV node, mostly there is complete or incomplete compensatory pause following PVCs. This represents the time taken by the SA node to regain its function.

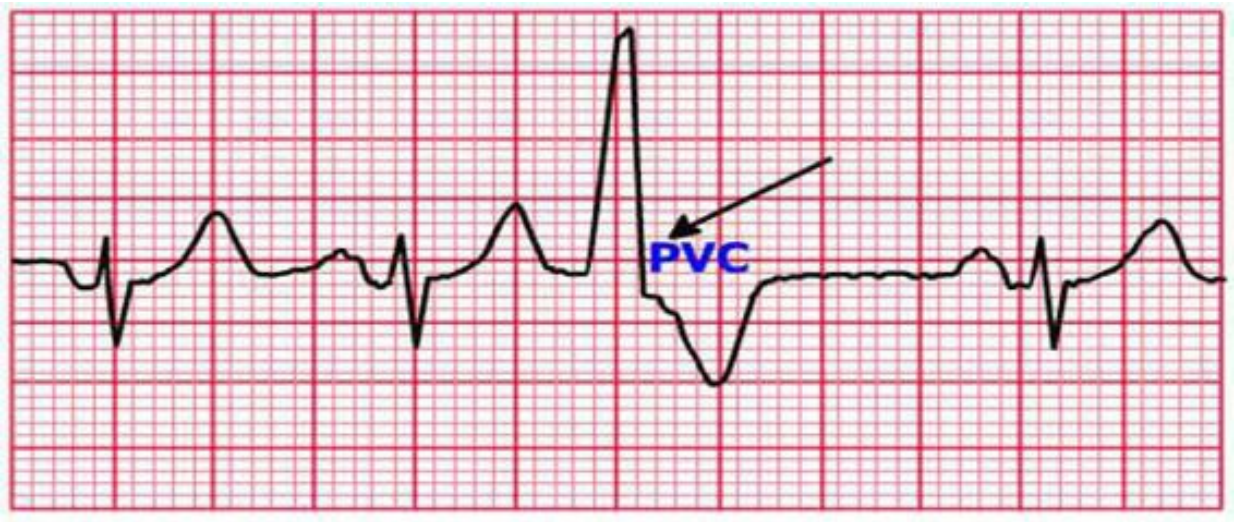


Fig 9.1 Premature ventricular contraction

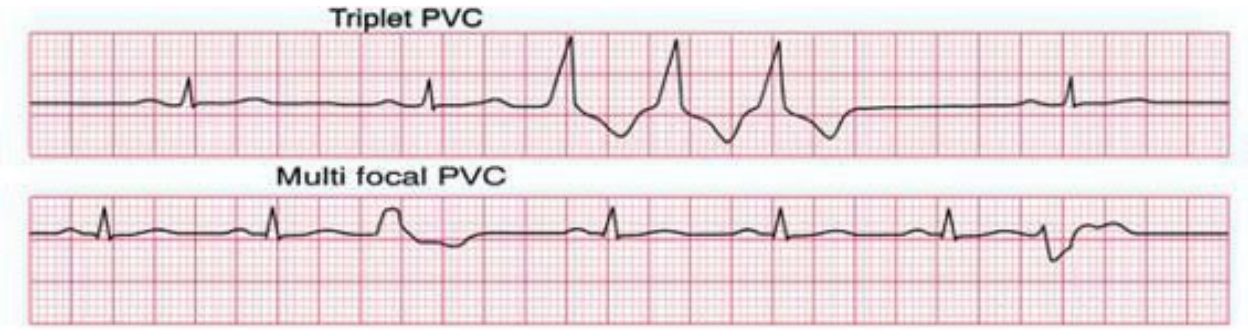


Fig 9.2 Various forms of Premature Ventricular Contractions (PVC)

Premature atrial contraction with aberrant conduction may also produce wide and bizarre QRS complex that needs to be distinguished from that of ventricular tachycardia. *Presence of a premature P wave at the beginning of tachyarrhythmia* or *typical right or left bundle branch block pattern of QRS complex* are the tell-tale signs of differentiating **PAC with aberrant conduction** from that of **Ventricular tachycardia** (**V-tach**). Therefore, it is

imperative to see the origin and termination of tachyarrhythmia in most of the situations in order to differentiate true versus aberrant rhythms.

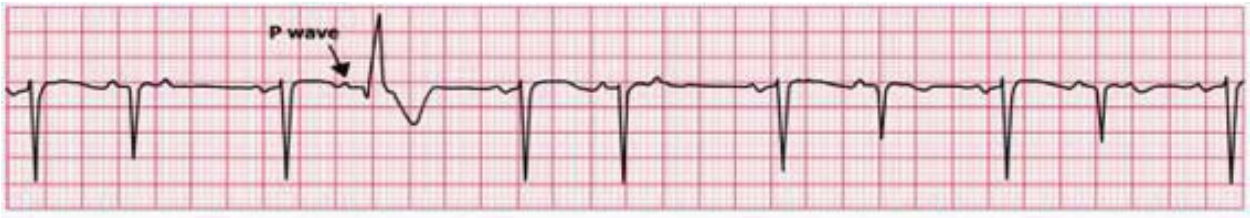


Fig 9.3 PAC with aberrant conduction (Note the p wave before distorted QRS).

Basic characteristics are

Rhythm: underlying regular rhythm with irregularity during PVC.

Rate: usually within normal limits.

P wave: normal except during PVC, where it is absent.

PR interval: normal except during PVC, where it is immeasurable.

QRS complex: *wide and bizarre duration >0.12 second during PVC.*

T wave: *points in opposite direction of QRS deflection* (if QRS negative, T wave positive).

QT interval: not measurable during PVC.

Clinical significance: Premature ventricular contractions are common among *old age* and patients with *structural heart disease*. They are also seen in *electrolyte imbalance*, *acidosis*, *congestive heart failure*, *acute myocardial infarction*, *drug toxicity* etc. Occasional PVCs are benign. However, if they occur more frequent especially in the setting of underlying structural heart disease, it can progress to lethal ventricular tachycardia or fibrillation.

If the PVC happens to originate in the **down slope of T wave** of the previous repolarization wave, it can produce **polymorphic ventricular tachycardia** (**R on T phenomena** or **Torsades de Pointes**). *Magnesium deficiency* is one of the common condition precipitate this arrhythmia.

Management: Treatment of underlying problem such as *correction of electrolytes (potassium, magnesium)* is of supreme priority during treatment of symptomatic PVCs. Anti arrhythmic agents like *Amiodarone* and *beta-blockers* may be helpful in frequent PVCs. Recurrence of multiple PVCs in high frequency can create a **reversible cardiomyopathy** that can further impair ventricular function. It is important to remember when using anti arrhythmic drugs that many of these drugs themselves can create ventricular arrhythmias (**pro arrhythmic**

effect) by slowing down conduction in the ventricles (**Q-T prolongation**). Therefore, judicious use and adequate monitoring is needed while treating patients with these medications to prevent sudden cardiac death (SCD) from lethal arrhythmia.

Osborn wave

These waves are otherwise known as **hypothermia waves** where there is a *positive deflection seen at the J point* (Junction between QRS complex and ST segment). They are more prominent in the precordial leads. This is a characteristic EKG finding in patients with either accidental or therapeutic hypothermia.

These waves usually *resolve with rewarming of the patient*. These waveforms should be differentiated from that of Brugada syndrome or early repolarization. Even though controversy still exists, Osborn waves are considered as a *warning sign of ventricular fibrillation* in hypothermic patients.

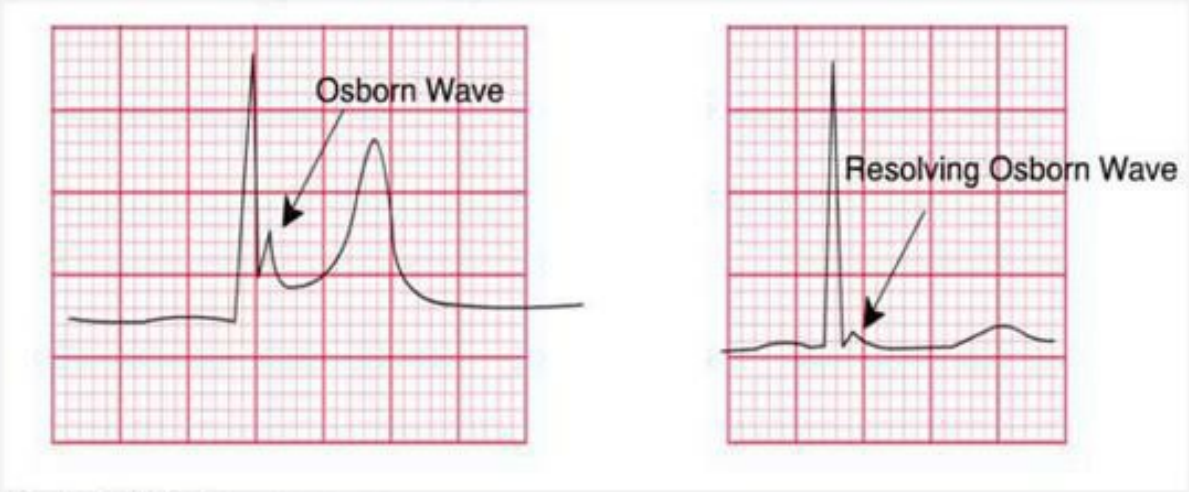


Fig 9.3 C Osborn waves

Box 9.1 Osborn waves of hypothermia

Idioventricular Rhythm

This is a true ventricular rhythm with the firing at a rate of **20 to 40 bpm** and is a telltale sign of *imminent life-threatening events* like *agonal rhythm* or *asystole*. This is also known as **ventricular escape rhythm**. It usually happens when all the higher-level pacemakers

have failed. Mostly, this rhythm does not provide sufficient left ventricular contraction and leads to serious hemodynamic compromise. At times, this rhythm accompanies *complete AV dissociation* (third degree heart block).

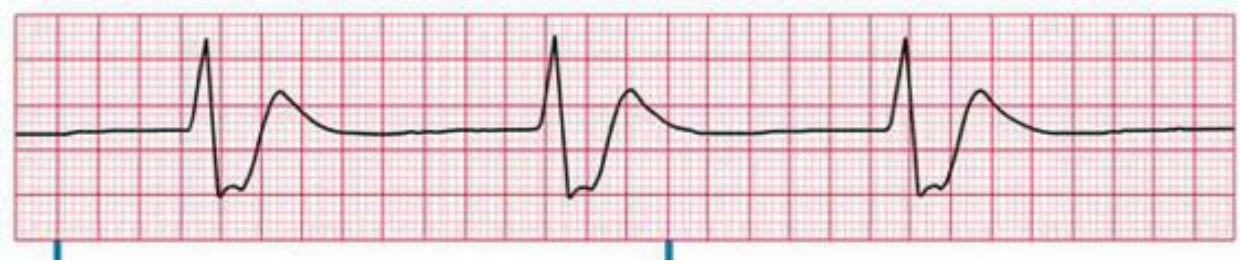


Fig 9.4 Idioventricular rhythm

Basic characteristics are

Rhythm: regular.

Rate: *20 to 40 bpm*.

P wave: none except in third degree heart block.

PR interval: immeasurable.

QRS complex: wide and bizarre with greater than 0.12 seconds duration.

T wave: like PVC, deflects in opposite direction of QRS.

QT interval: prolonged.

Clinical significance: This rhythm *may or may not produce a pulse* and therefore, aggressive management with *basic and advanced cardiac life support* measures are warranted.

Agonal Rhythm

This is the *worst possible cardiac rhythm second to asystole*. It denotes severely impaired cardiac function with impending death. Even the ventricular escape beats are not produced enough to maintain an Idioventricular rhythm. Characterized by irregular occasional wide complex beats.

Basic characteristics are

Rhythm: irregular.

Rate: *less than 20 beats per minute*.

P wave: none.

PR interval: not applicable.

QRS complex: wide and bizarre with greater than 0.12 seconds duration.

T wave: swing in opposite direction of QRS.

QT interval: prolonged.

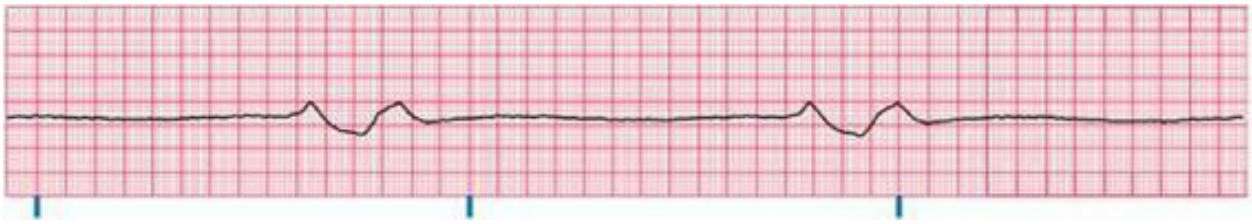


Fig 9.5 Agonal rhythm

Clinical significance: As mentioned earlier, agonal rhythm is the last possible electrical impulses before asystole. *Severe and massive cardiac compromise* has already happened. The patient may essentially be in shock state. Management of underlying reversible causes or aggressive life support measures with *basic and advanced cardiac life support* is the treatment of choice.

Accelerated Idioventricular Rhythm (AIVR)

Accelerated Idioventricular rhythm originates due to *abnormal automaticity* within the ventricle and produce impulse at **40 to 120 bpm**. It can be seen in the absence of any structural heart disease or in the event of *acute myocardial infarction*, *cocaine toxicity*, *digoxin intoxication*, *postoperative cardiac surgery* or after *chemical or mechanical revascularization of coronary arteries* as in **tPA administration** and **coronary angioplasty**. In these settings, it is also known as **reperfusion arrhythmia**.

The basic characteristics that differentiate **AIVR** from that of **slow V-tach** are its *gradual onset and termination with a brief self-limiting pattern*. In order to differentiate **Idioventricular rhythm (IVR)** from **accelerated Idioventricular rhythm (AIVR)**, *look for presence of P waves in AIVR that is absent in Idioventricular escape rhythm*. Remember *in AIVR, SA node is still firing unlike in IVR* where there is no higher order pacemaker functioning.

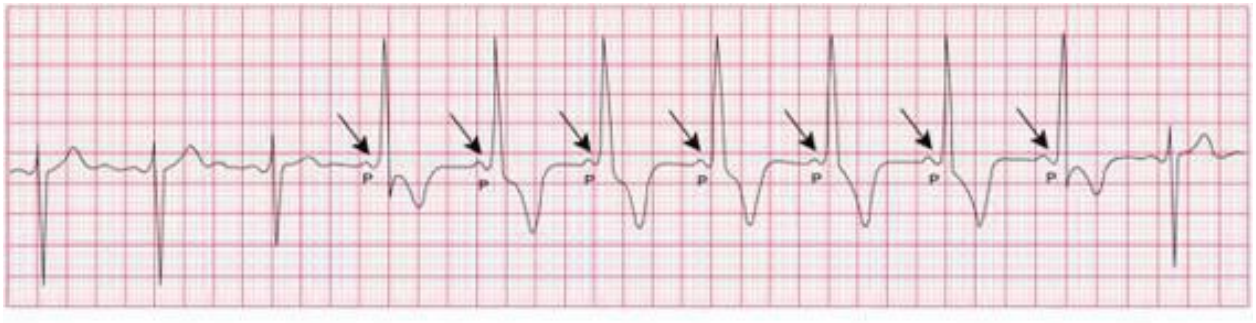


Fig 9.6 AIVR (Note the presence of P waves and brief self limiting pattern of arrhythmia)

Because of the rate at which the ventricles contract, mostly *AIVR does not produce significant compromise in cardiac output*. However in the event of sustained AIVR, particularly in the setting of acute myocardial infarction and postoperative status, lack of AV synchronization can create hemodynamic instability.

Basic characteristics are

Rhythm: mostly regular.

Rate: *40 to 120 bpm*.

P wave: may be seen *before, during* or *after QRS*. At times, *inverted* or *absent*.

PR interval: immeasurable if P waves are absent.

QRS complex: *wide and bizarre*, duration >.12 seconds.

QT interval: prolonged.

Clinical significance: As mentioned earlier, hemodynamic compromise rarely occurs with AIVR. Mostly it is a self-limiting arrhythmia.

Ventricular Tachycardia

Ventricular tachycardia (V-tach/ VT) forms when there are *three or more PVCs in a row* and *ventricular rate exceeds 100 bpm*. If the duration of ventricular tachycardia is less than 30 seconds, it is called **non-sustained ventricular tachycardia (NSVT)**. If the rhythm *persists for more than 30 seconds* or terminated within 30 seconds by either An Implantable Defibrillator (**ICD** or **AICD**) or external defibrillation, it is known as **sustained VT**. Depending on the morphologic characteristics, VT can be classified into **monomorphic VT** and **polymorphic VT**.

Basic characteristics are

Rhythm: usually regular.

Rate: *ventricular rate between 100 to 250 bpm*; atrial rhythm is indiscernible.

P wave: usually absent or indistinguishable.

PR interval: not applicable.

QRS complex: *wide and bizarre* with the duration >0.12 seconds.

T wave: in the opposite direction of QRS deflection.

QT interval: prolonged.

Clinical significance: Depending on the duration and frequency of ventricular tachycardia, it may be well tolerable or having serious hemodynamic effects. Since the contraction of ventricles at 150 to 250 bpm *does not provide time for effective ventricular contraction*; *cardiac output is considerably reduced*.

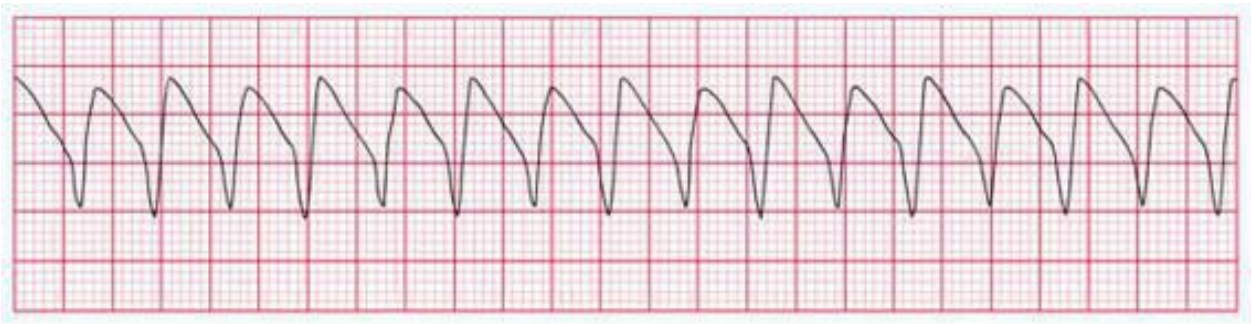


Fig 9.7 Monomorphic VT

Management: Any VT or V-Fib that is compromising hemodynamic function should be treated with immediate **asynchronous DC cardioversion** if available. Intravenous **Lidocaine** or **Amiodarone** are other mainstay treatment choices. *Intravenous antiarrhythmics should be continued even after electrical cardioversion* in order to prevent recurrence of these rhythms. *Basic and advanced cardiac life support* is indicated if the patient doesn't have a perfusing rhythm.

Evaluation of wide complex tachycardia is extremely important as there are some entities that resemble ventricular tachycardia, but not in as lethal as VT. Some of these rhythms are **pacemakers mediated wide complex tachycardia** and **SVT with aberrancy.** Most important characteristics that differentiate ventricular tachycardia from other nonlethal wide complex tachycardia are

(1). A wide complex QRS that *does not match previous* wide complex tachycardia.

(2). Absence of any pre-excited QRS pattern in sinus rhythm *at the beginning* of wide complex tachycardia.

(3). The *bizarre QRS pattern that does not mimic right or left bundle branch block* in a 12 lead EKG.

(4). *Slurring of initial part of QRS*.

(5). *Signs of AV dissociation in the EKG like fusion beats*.

Unlike wide complex supraventricular tachycardia, *VT does not respond to vagal measures* or other standard treatment for SVT. In majority of situations, ventricular tachycardia is seen in patients with prior cardiac structural damage that is evident in EKG by presence of Q waves.

Torsades de Pointes (TDP)

This is the form of **polymorphic ventricular tachycardia** with a *varying QRS morphology*. It has an *undulating QRS complex* with reference to the isoelectric line. This can degenerate into **ventricular fibrillation** with serious hemodynamic compromise. The French term ***Torsades de Pointes*** means *'twisting of the points'*. It may be produced by *acute ischemia*, *myocarditis* etc. and is generally not a reproducible rhythm like monomorphic VT during an electrophysiologic evaluation. Since TDP refers to a more serious unstable situation, urgent treatment is warranted.

Basic characteristics are

Rhythm: may be regular or irregular with varying QRS size.

Rate: atrial rate immeasurable, *ventricular rate 150 to 300 bpm*.

P wave: not seen.

PR interval: unable to calculate.

QRS complex: *wide and bizarre* with the duration greater than *0.12 seconds*. Complexes with *positive and negative deflection* are present compared to isoelectric line.

T wave: difficult to identify.

QT interval: prolonged in beats prior to torsades.

Clinical significance: Torsades is considered as one of the lethal arrhythmias since it can degrade into ventricular fibrillation with serious hemodynamic compromise. *Short runs of torsades are generally well tolerated* in the absence of critical hemodynamic issues. However if longer, it is *treated with the same measures* as of *sustained ventricular tachycardia*.

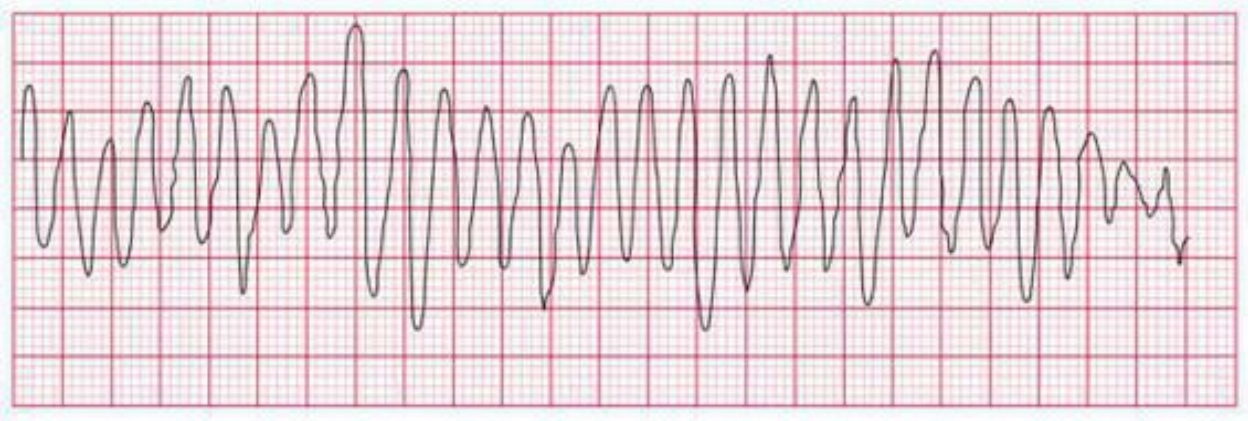


Fig 9.8 Torsades (Polymorphic VT)

Management: Hallmark treatment for torsades is intravenous administration of **magnesium** since *hypomagnesemia at myocardial level* can precipitate this rhythm. Same as that of VT, *torsades can be produced by pharmacologic agents* like *Procainamide* and *Amiodarone*, which are known for their Q-T prolongation property (**pro-arrhythmic effect**). Therefore, frequent monitoring of QT interval is warranted especially in the beginning of treatment with these medications.

VT Storm

Repeated ventricular tachycardia episodes requiring electrical cardioversion or defibrillation with *more than two incidents within 24 hours* are defined as **VT storm**. In reality, these patients experience much more episodes than stated in the definition. In the absence of long QT interval prior to the VT episode, active **myocardial ischemia** or fulminant **myocarditis** should be suspected.

Management strategy differs depending on whether the VT is polymorphic or monomorphic in nature. In patients with recurrent monomorphic VT, intravenous administration of *Amiodarone*, *Lidocaine* and *Procanamide* may prevent recurrence. However, these QT prolonging drugs can make the patient arrythmogenic because of their proarrythmic property. **Radiofrequency ablation** of foci within the ventricle is of great use in *refractory VT*. In the case of polymorphic VT, possible management options include correction of electrolyte imbalance like magnesium and potassium, stopping any Q-T

prolongation drugs, treatment of underlying myocardial ischemia etc.

Other Forms of Ventricular Tachycardia

Long QT Syndrome (LQTS)

This is a *congenital defect in cardiac ion channels* responsible for repolarization of myocardium. In the action potential curve of myocardial tissue, this defect enhances *sodium and calcium inward movement* and *inhibits outgoing potassium ions* during **phase 1** (plateau phase). It essentially prolongs action potential duration *and therefore QT interval*. When the QT is abnormally prolonged, there is more chance for possible ectopic beats to produce polymorphic VT events (**R on T phenomena** or **Torsades**) especially during exercise or activity where QT interval should be shortened in proportion with increase in heart rate.

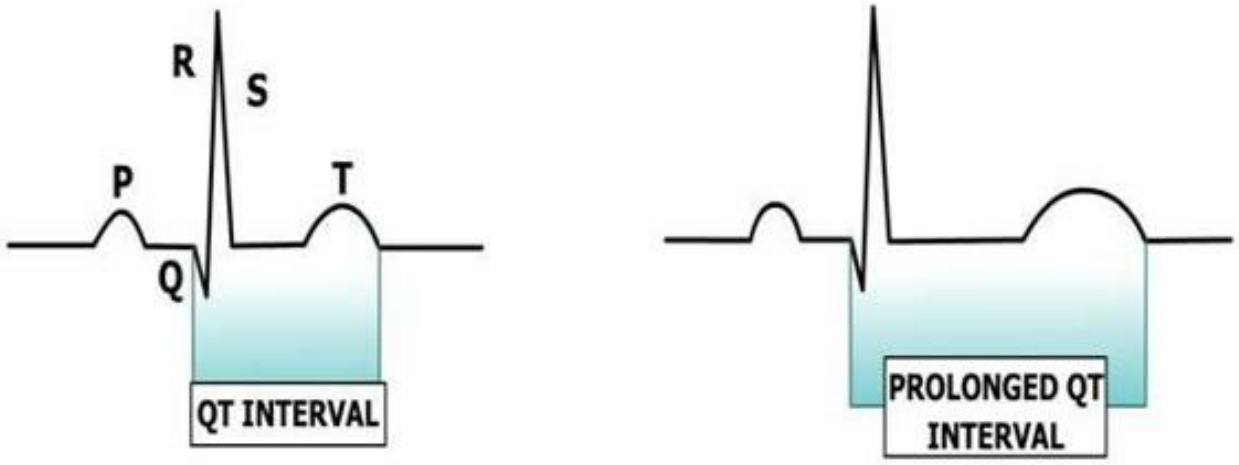


Fig 9.9 Long QT syndrome

In most patients with long QT syndrome, *corrected QT interval* is up to **460 ms** *in men and* **480 ms** *in women*. Q-T prolongation beyond **500 ms** suggests higher risk for arrhythmia. The syndrome can also be precipitated by QT prolonging drugs like **Sotalol** in individuals who are otherwise asymptomatic and therefore close monitoring is warranted during initiation of these drugs. In patients with prolonged QT interval greater than 500 milliseconds and those with strong family history of **sudden cardiac death** (**SCD**), implantation of an **Implantable Cardioverter Defibrillator** (**ICD**) is the treatment of choice for primary prevention of SCD.

Short QT Syndrome (SQTS)

This is one of the relatively uncommon syndromes with effective QT interval of **less than 320 ms** with EKG finding of **tall T waves**. These patients are more prone to have *fibrillation of atria or ventricle*. Implantable cardioverter defibrillators (**ICD**) implantation is needed to prevent sudden cardiac death from V-Fib. However in certain situations, because of the presence of tall T waves, ICD may count them as QRS complex (effectively doubling the number of calculated QRS complexes) and inappropriately Institute shock therapy for tachyarrhythmia.

Brugada Syndrome

Named after Spanish cardiologists, who identified this genetic disorder as the reason for many of unexplained sudden cardiac death in Southeast Asian males. This involves mutation of **SCN5A gene**, which essentially *reduces inward sodium movement* during ***phase 0*** of action potential in myocardial cells located at the epicardium of the right ventricular outflow tract. This leads to dramatic shortening of cardiac action potential of these cells compared to the rest of the ventricle. Therefore, these cells may involve with abnormal repolarization and depolarization circuits compared to the rest of the myocardium leading to lethal ventricular arrhythmia. Patients with Brugada syndrome has EKG showing complete or incomplete **right bundle branch block pattern** with characteristic **coved-type** (In lead V2 as shown in Fig 9.10) or **saddle back** (as in lead V3 in Fig 9.10) **ST elevation** in **V1** to **V3** in sinus rhythm.

Since these individuals are at high risk of having lethal ventricular arrhythmia leading to sudden cardiac death, *ICD implantation* is indicated.

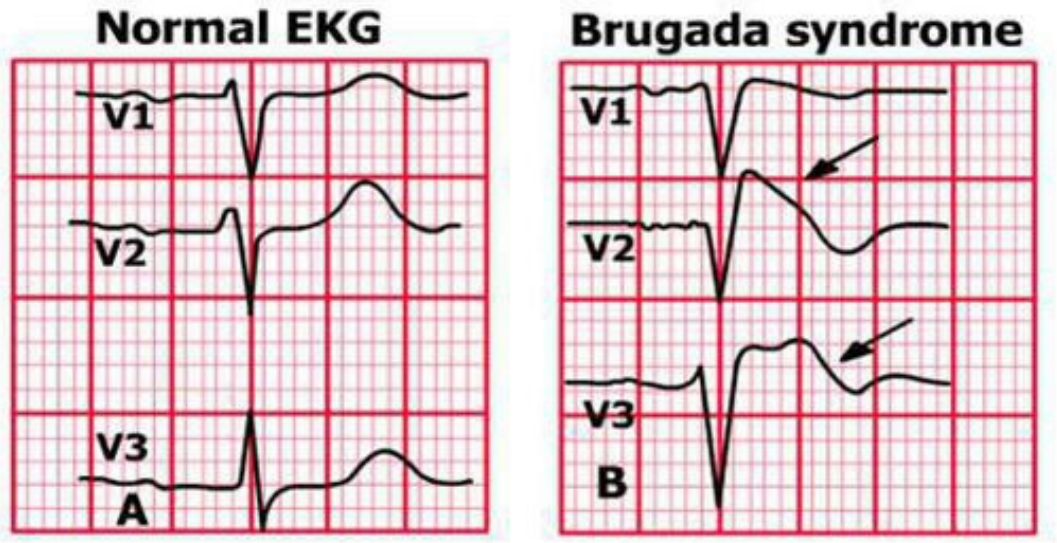


Fig 9.10 Brugada pattern (Note the characteristic ST segment elevation in V1-V3)

The *family members should be screened* for presence of this disorder. Misdiagnosis of ventricular tachycardia resulting from Brugada syndrome and subsequent treatment with class I antiarrhythmic drugs (sodium channel blocking agents like Procanamide and flecainide) may exacerbate chances of lethal arrhythmia.

Major drugs causing QT prolongation

Drug	Use
Amiodarone	Antiarrhythmic
Chloroquine	Antimalarial
Chlorpromazine (Thorazine)	Antipsychotic
Clarithromycin	Antibiotic
Erythromycin	Antibiotic
Disopyramidine (Norpace)	Antiarrhythmic
Dofetilide (Tykosin)	Antiarrhythmic
Droperidol	Anti nausea
Methadone	Opiate agonist
Procainamide	Antiarrhythmic
Sotalol (Betapace)	Antiarrhythmic
Quinidine	Antiarrhythmic
Thioridazine	Antipsychotic

Box 9.1 Major QT prolonging drugs

Ventricular Fibrillation (V Fib)

Similar to atrial fibrillation, V fib is a *completely disorganized and chaotic ventricular rhythm*, which has serious hemodynamic implications. Multiple ectopic foci within the ventricle start firing impulses; leading to unsystematic depolarization of the ventricles. Resultant 'quivering' motion of ventricles essentially produce **ventricular standstill**. V fib can be with coarse or *fine fibrillatory waves*.

Basic characteristics are

Rhythm: *completely irregular, with fibrillatory waves*.

Rate: cannot be determined.

P wave: not seen.

PR interval: cannot calculate.

QRS complex: no definite QRS; *just undulating waves*.

T wave: not seen.

QT interval: cannot calculate.

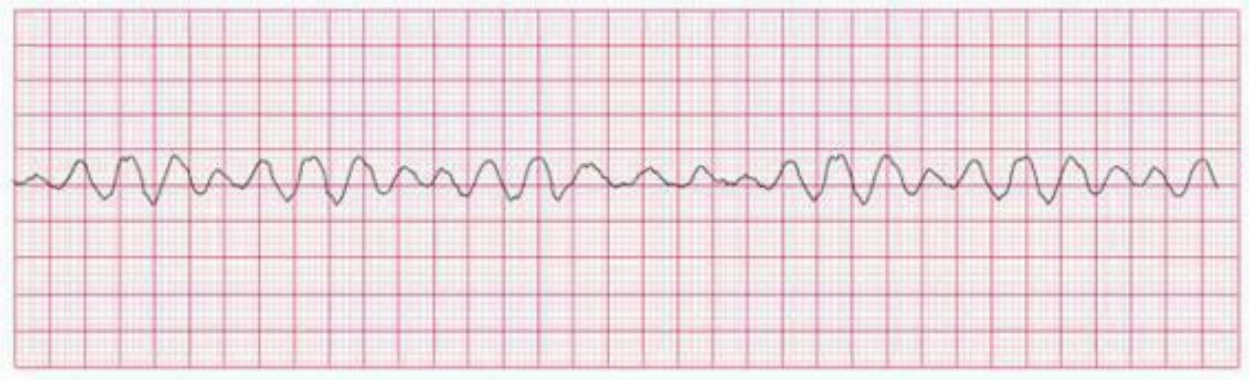


Fig 9.11 Corse V Fib

Clinical significance: Causative factors are the same as that of ventricular tachycardia; however, indicate more serious underlying events. This rhythm does not generate any cardiac output whatsoever and therefore, immediate basic and advanced life support measures including **emergent defibrillation** are of at most necessity. If left untreated, coarse V fib may progress to fine V fib. Pharmacologic agents such as *Amiodarone* and *Lidocaine* are used after electrical defibrillation to prevent recurrence of VT/V fib.

Asystole

This denotes a *complete ventricular standstill* with no cardiac output. In the EKG, a **flat line** appears and it essentially means there is absolutely no pacemaker activity happening anywhere in the heart. In some instances, we may see some atrial activity with no corresponding ventricular response. If the patient has a pacemaker either permanent or temporary, we may see pacing spikes at regular intervals without corresponding P or QRS complex.

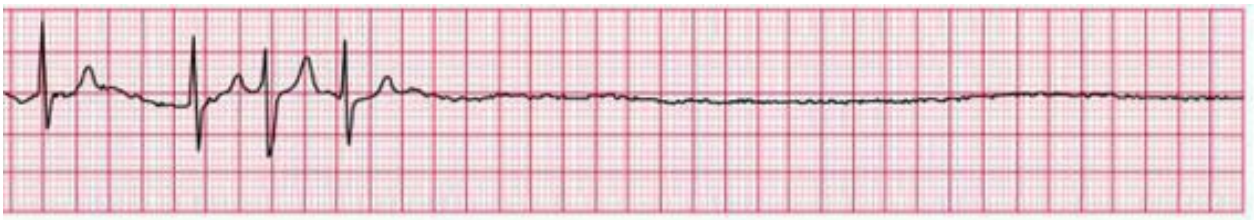


Fig 9.12 Asystole

Basic characteristics are

Rhythm: atrial-irregular if present, ventricular-none.

Rate: indiscernible.

P wave: may or may not be present.

PR interval: not measurable.

QRS complex: not present.

T wave: not seen.

QT interval: not applicable.

Clinical significance: This is the **terminal rhythm** of the heart. Usually seen after *profound cardiac damage* in severe *hypoxia*, *multisystem failure* etc. Aggressive **resuscitative efforts** with *epinephrine*, *atropine* and *CPR* are the way to go. However if this rhythm persists even after reasonable efforts, it is time to declare demise of the patient.

Points to Remember!!!

- Premature ventricular contractions produce rhythms with wide and bizarre QRS complexes having duration more than 0.12 sec.
- In PVC's, T wave follows the opposite direction of that of QRS complex because of the disorganized ventricular repolarization.
- Ventricular pacemaker cells have an inherent pacing rate of 20 to 40 bpm.

· PVC is can be unifocal or multifocal depending on the ectopic focus and its morphology.
· In terms of regularity, PVCs can be every other beat (bigeminy), every third beat (trigeminy) or more than three in a row called 'run of VT'.
· PACs with aberrancy can be distinguished from PVCs by the presence of premature P wave at the beginning of tachyarrhythmia.
· PACs are common in old age, patients with structural heart disease or in electrolyte imbalance.
· Antiarrhythmic agents like Amiodarone, beta-blockers and correction of underlying causative factors are the modes of treatment for PVCs.
· Idioventricular rhythm or ventricular escape beat with a rate of 20 to 40 bpm is a sign of life-threatening events of the heart.
· Agonal rhythm with a rate less than 20 beats per minute shows massive impairment of cardiac function.
· Accelerated Idioventricular rhythm with the heart rate of 40-120 beats per minute is caused by abnormal automaticity within the ventricle.
· AIVR is considered as a reperfusion arrhythmia in the event of revascularization procedures.
· The main differentiating factor between Idioventricular rhythm (IVR) and accelerated Idioventricular rhythm (AIVR) is the presence of P waves in AIVR.
· If the duration of ventricular tachycardia is less than 30 seconds and terminated spontaneously, it is called non-sustained ventricular tachycardia.
· If the VT episode lasts more than 30 seconds or is terminated within 30 seconds by defibrillation, it is called sustained V-tach.
· VT can be monomorphic and polymorphic depending on the morphology of QRS complex.
· Ventricular tachycardia does not provide any significant cardiac output and therefore immediate intervention is demanded for sustained ventricular tachycardia.
· Asynchronous DC cardioversion for rhythm conversion and use of Amiodarone and Lidocaine for prevention of recurrent is the mode of treatment for ventricular tachycardia.
· Torsades the Pointes can produce ventricular fibrillation and serious hemodynamic dysfunction.
· Hypomagnesaemia is the most common causative factor for TDP.
· Incidence of more than two ventricular tachycardia episodes within 24 hours requiring electrical cardioversion or defibrillation is known as VT storm.
· Radiofrequency ablation of the focus within the ventricle is the treatment for VT storm.
· Long QT syndrome is caused by congenital defect in cardiac ion channels causing prolongation of phase 1 in action potential curve; leading to prolonged QT.
· Corrected QT interval above 500 ms suggests higher risk of arrhythmia in patients with long QT syndrome.

· Implantation of ICD for prevention of sudden cardiac death is the treatment option for long QT syndrome.
· Short QT syndrome with QT interval less than 320 ms can cause atrial or ventricular fibrillation.
· Brugada syndrome is a genetic disorder, common in Southeast Asian males causing sudden cardiac death.
· Characteristic EKG finding in Brugada syndrome is the right bundle branch block pattern with ST elevation in V1 to V3.
· The family members should be screened for presence of gene mutation that can cause Brugada syndrome in patients with this disorder.
· In ventricular fibrillation, disorganized and chaotic ventricular rhythm leads to ventricular standstill.
· Emergent defibrillation is the treatment options for ventricular fibrillation.
· Asystole is the terminal rhythm of the heart that requires aggressive resuscitative effort with CPR and advanced cardiac life support.

Test Your Understanding

1. Which of the following QRS complex duration represent possible ventricular rhythm?

A 0. 0.8 seconds
B 0.04 second
C 0.14 second
D 0.7 seconds

2. Treatment option for frequent PVCs include_____?

A Atropine
B Lisinopril
C Amiodarone
D Epinephrine

3. Which of the following is true statement regarding Idioventricular rhythm?

A It is caused by abnormal automaticity within the ventricle
B It is of no significant clinical value.
C It shows imminent life-threatening events
D It has distinct P wave in front of every QRS complex

4. Which of the following possible rhythm can be seen after administration of thrombolytic medications in an acute myocardial infarction patient?

A Sinus arrhythmia
B Atrial fibrillation
C Accelerated Idioventricular rhythm
D Idioventricular rhythm

5. Sustained ventricular tachycardia is defined as _____?

A Self terminated within 30 seconds
B Require defibrillation or last more than 30 seconds
C Last less than 30 seconds but doesn't require defibrillation
D Last less than 30 seconds and rate stays within 100bpm

6. What is the most important differentiating factor between Torsades de Pointes and other forms of monomorphic ventricular tachycardia?

A Presence of wood wide and bizarre QRS complex

B Ventricular rate 100 to 250 bpm

C Long QT interval

D QRS complex with positive and negative deflection

7. Identify the most important electrolyte under consideration in a patient with polymorphic ventricular tachycardia?

A Calcium

B Sodium

C Potassium

D Magnesium

8. Which of the following is the most desirable treatment for VT storm?

A Beta-blockers

B Calcium channel blockers

C Radiofrequency ablation

D Digoxin

9. Untreated long QT syndrome may lead to _____?

A Sinus tachycardia

B Atrial fibrillation

C Polymorphic VT

D Asystole

10. Which of the following statement is true regarding Brugada syndrome?

A It is caused by intake of large amounts of mercury

B It involves genetic mutations that alter ionic movement in cardiac cells

C It involves genetic mutations that change position of the heart

D There is no evidence for Brugada syndrome to run in the family

11. What is the most common characteristic of Brugada syndrome pattern?

A Left bundle branch block pattern

B ST elevation in V5 and V6

C ST depression in V1 to V3

D Right bundle branch block pattern with ST elevation in V1 to V3

12. Which of the following treatment option is ideal for the patient in ventricular fibrillation?

A Cardiopulmonary resuscitation

B Synchronized cardioversion

C Emergency defibrillation

D IV calcium administration

13. Which of the following statement is true regarding hemodynamics during coarse ventricular fibrillation?

A Coarse V-Fib does not require defibrillation as in the case of fine V-Fib

B Coarse V-Fib does not cause complete ventricular standstill

C Courses V-Fib cause physiologic standstill for cardiac output

D In coarse V-Fib, distinct P waves are easily identified

14. Which of the following EKG parameter has to be monitored while starting a patient on Sotalol?

A Shortened QT interval

B Atrial flutter

C Prolonged QT

D Short runs of SVT

Answers

1. C 0.14 second

2.	C	Amiodarone
3.	C	It shows imminent life-threatening events
4.	C	Accelerated Idioventricular rhythm
5.	B	Require defibrillation or last more than 30 seconds
6.	D	QRS complex with positive and negative deflection
7.	D	Magnesium
8.	C	Radiofrequency ablation
9.	C	Polymorphic VT
10.	B	It involves genetic mutations that alters ionic movement in cardiac cells
11.	D	Right bundle branch block pattern with ST elevation in V1 to V3
12.	C	Emergency defibrillation
13.	C	Courses V-Fib cause physiologic standstill for cardiac output
14.	C	Prolonged QT

Chapter 10

Cardiac Electrical Assistive Devices

Modern pacemakers are technological marvels that provide electrical impulses when the natural pacing or conducting properties within the heart fails. They are indicated for patients with various disorders of automaticity and conductivity. There are various classifications of pacemakers based on their function, duration of use and number of chamber

paced.

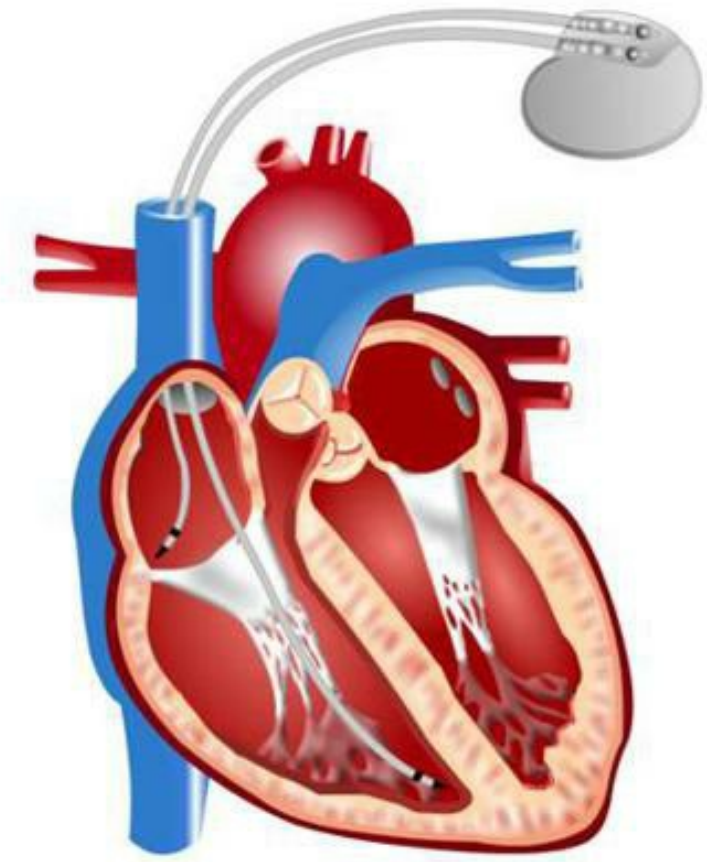


Fig 10.1 Dual chamber Pacemaker

Classification of Pacemakers

Based on the functional significance, pacemakers can be divided into **Fixed** pacemakers and **Demand** pacemakers.

Fixed pacemakers are the old generation of pacemakers with minimal programming options. Here, the pacemaker fires impulses to the designated chamber at a *fixed rate irrespective of the patient's innate cardiac activity* . In normal human beings, this mod of pacing is dangerous because of the fact that either the pacemaker can pace at faster rates or can initiate impulses at inappropriate timing within the cardiac cycle (e.g. on the downslope of T wave) and create dangerous arrhythmia. This limited functionality provide limitted use for this type of pacemakers in the real world.

On the other hand, **Demand pacemakers** are devices that generate impulses *based on the person's innate cardiac impulse*. These types of devices have a preset range of

programming (usually 60-120 bpm) and the device *will pace only when the person's heart rate drops below or above the set rate*. These devices make more sense in the real world since they can support the heart when it is in **demand**.

Another classification is based on the number of chambers being paced. It can be (1) **single chamber** pacemaker, which is *either sensing or pacing the atrium or the ventricle*, (2) **dual chamber** with *pacing and sensing function in the atria and the ventricle*. In patients with SA node dysfunction where there is deficiency in impulse production, an **atrial pacemaker** is helpful by providing much-needed electrical stimuli in the atria. This impulse will then carried through normal conduction pathways down to the ventricle and generate cardiac contraction. However in patients with conduction defect such as *complete heart block*, *single chamber pacemaker does not work* because of the *lack of atrio-ventricular connection*. In this instance, pacing both atria and ventricle in synchronized fashion will improve hemodynamics. Essentially, one of the basic concepts of dual chamber pacemaker is to provide **atrio-ventricular synchronization**.

There is a new generation of devices called **biventricular pacemakers,** which are particularly useful in patients with *depressed ventricular ejection fraction* with *coexisting bundle branch block*. Instead of *contracting together*, both ventricles *depolarize in sequence* due to the delay in impulse conduction from bundle branch block. Because of this, whichever ventricle contracts first may partially push the inter-ventricular septum into the adjacent ventricle and therefore losing part of its contractile effort to pump blood out through aorta and pulmonary artery.

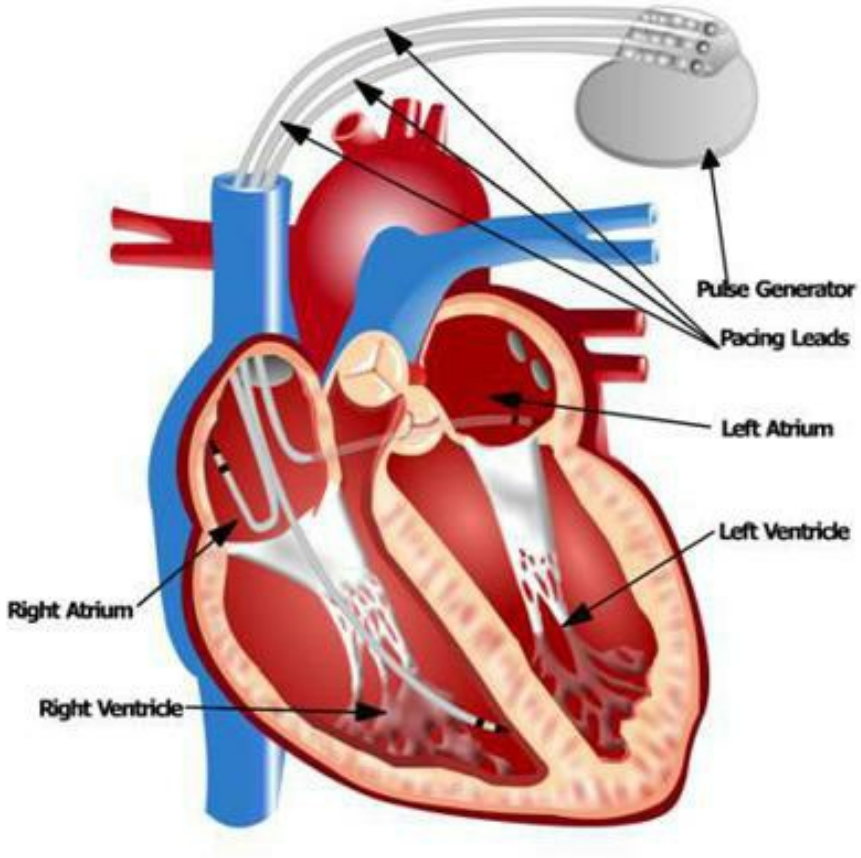


Fig 10.2 Biventricular pacemaker

In **Cardiac Resynchronization Therapy** (CRT), using biventricular pacemakers we ensure *simultaneous contraction of both ventricles*. In most of the cases, these patients have underlying structural heart disease such as severely reduced ventricular ejection fraction that require them to have Implantable cardioverter defibrillator (ICD) implantation for prevention of sudden cardiac death (SCD). Therefore, a combined **biventricular ICD** (**BiV ICD**) is of common use. This device has both *pacemaker function which is continuous and defibrillator function for possible ventricular tachyarrhythmia emergencies*. Since the pacemaker leads are not generally placed on the left ventricle; in a BiV ICD, left ventricular lead is placed in the **coronary sinus**. This structure is situated on the roof of the left ventricle and therefore provides an indirect access to left ventricular wall.

Depending on the duration of use, pacemakers can be **permanent pacemakers** or **temporary** pacemakers.

Temporary pacemakers are usually inserted through femoral or jugular veins and are intended for short-term use while the patients are being stabilized. Most common reasons for

temporary pacemaker insertion are *fulminant inferior wall myocardial infarction*, *post cardiac surgery*, *third degree AV block* etc. There is more chance for complications like *infection*, *bleeding* and *lead displacement* with this type of pacemakers. As soon as the patient is stabilized, these devices are either removed or changed for a permanent pacemaker. Alternate access sites for temporary pacing are **transcutaneous** (with large pads attached to skin), **epicardial** (during and immediately post cardiac surgery) and **transthoracic** (by insertion of a needle into the right ventricle and threading pacemaker wire in to the heart) route.

Permanent pacemakers are indicated for long-term use with extremely long battery life (average **5-8 years**). They consist of pacemaker leads that are implanted commonly through right or left **subclavian vein** into the myocardium. There can be up to 3 leads depending on the type of pacemaker. The other ends of these leads are connected to the pacemaker generator unit that is usually implanted in the **infraclavicular fossa** (underneath the clavicle). These devices can be programmed externally with the use of specialized magnetic probes and therefore are more convenient and user friendly in the real world.

Indications for pacemakers

- Sinus bradycardia (rate less than 40 bhm with pauses)
- Complete atrio-ventricular block (third degree heart block)
- Symptomatic second –degree AV block
- Exercise induced second- degree or third-degree block
- Significant vasovagal symptoms
- Second-degree Type ll AV block with wide QRS
- Idioventricular rhythm

Box 10.1 Indications for pacemakers

Modes of Pacing

Modern pacemakers can be programmed in such a way that it can provide most individualistic treatment protocol for the given patient depending on underlying disease process. There are mainly five letters used to denote specific programming of the pacemaker. They are mentioned in Table 10.2 with details. Sometimes, only first three letters are used since these represent majority of the pacemaker's capabilities.

Complications of pacemaker implantation

- Infection
- Hematoma
- Pneumothorax
- Cardiac perforation
- Diaphragmatic or phrenic nerve stimulation
- Lead dislodgement
- Skin erosion at implantation site

Box 10.2 Complications of pacemaker implantation

Modes of pacing

- First letter — chamber paced
  - V — ventricle
  - A — atria
  - D — dual or both
  - O — none
- Second letter — chamber sensed
  - V — ventricle
  - A — atria
  - D — dual (atria and ventricle)
  - O — none
- Third letter — pacemaker response to intrinsic rhythm
  - T — triggered (trigger pacing in response to sensed event)
  - I — inhibit (inhibits pacing in response to sensed event)
  - D — dual (It can inhibit and trigger impulses in various chambers depending on the event)
  - O — none (It does not respond to the sensed event)
- Forth letter — rate response
  - R — rate responsive (pacemaker provides paced impulse for a pre-determined range of heart rate)
  - O — no rate response
- Fifth letter — pacemaker's response to tachyarrhythmia
  - P — override pacing for tachycardia is available
  - S — shock therapy (available in high – energy devices e.g. AICD)
  - D — dual- ability to pace and shock
  - O — none

Box 10.3 Modes of pacing

Let's look through some examples of pacemaker programming. For the sake of explanation, we are considering only first four letters since the fifth letter is exclusively for **high-energy devices** like ICD.

For **VVIR** pacemaker,

The first letter **V** stands for chamber paced i.e. *ventricle*. Second letter **V** denote the chamber sensed and in this example it is *ventricle*. Third letter **I** shows the response of

pacemaker to the patient's own rhythm and here it is *inhibition*. That means, if the pacemaker sees patient's own ventricular beat; it doesn't produce pacemaker impulse. The last letter **R** corresponds to *rate responsiveness* of this pacemaker, which is explained later in this chapter.

So in nutshell, a **VVIR** pacemaker is functioning as a *single chamber pacemaker with pacing and sensing of ventricle. It does not generate an impulse if the patient has his own ventricular beat*.

Similarly in **DDDR** pacemaker programming,

The pacemaker has ability to pace *both chambers* (first letter **D**- dual), ability to sense from *both chambers* (second letter **D**- dual), ability to inhibit and trigger in *different chambers* (third letter **D**- dual) and has *rate responsiveness* (fourth letter **R**- rate response).

In a patient with **DDDR** pacer settings, the device will *look for intrinsic impulse in both chambers*. If there is no atrial impulse, pacemaker will fire at the atria and thereby generate atrial contraction. If the patient has impulse from his own SA node, the pacemaker inhibit its firing since there is no need for an additional impulse.

Coming to the ventricles, the same response happens with pacing from device if there is no ventricular impulse and inhibition of pacing if the patient has his own ventricular beat. The device will wait for a pre-determined timeframe (in milliseconds) to see whether the atrial impulse is creating a ventricular response (remember, ***AV nodal delay***) before making decisions of how to respond.

Even though this explanation can create some confusion regarding the sequence of pacemaker activity in relation to the letters we mentioned earlier, it is important to remember that the device is constantly sensing the cardiac activity and taking decisions within microseconds to come up with appropriate response for individual beats of the heart. The pacemaker has inbuilt **programmed timeframe** in milliseconds for which the device will wait before initiating either pacing or inhibition.

Rate Responsiveness

In order to better understand the significance of **rate responsiveness** along with other programming options, let's take a patient with atrial fibrillation who has a DDDR **pacemaker** implanted as an example. As mentioned earlier, one of the basic ideas of a dual chamber pacemaker is to *provide AV synchronization* . In this situation, the atrial lead sense *multiple P waves* usually in the order of 350 to 700 beats per minute because of atrial fibrillation. Due to the innate nature of AV node, most of these impulses get blocked at the AV junction and therefore patient will have a controlled ventricular response, which produce decent hemodynamics.

However in presence of a pacemaker, if the pacemaker was trying to track each P wave in the atria and to generate corresponding QRS complex within the ventricle, this patient

will have extremely fast and dangerous ventricular response. In order to prevent this from happening, the **rate response mechanism** is activated. This provides a *range of atrial beats* (60-100 in this situation) *under which the pacemaker will try to match one QRS complex for each P wave*. If the atrial rate falls below 60 or it goes above 100, the pacemaker will provide a **pre-determined rate** of ventricular impulses to maintain circulation. This mechanism is particularly useful in patients with uncontrolled atrial rates; however, need to increase their heart rate during physical activity and exertion.

Identifying paced EKG rhythm

Pacemaker provides characteristic electronic artifact called pacer spikes and is seen in an EKG depending on the chamber paced. For example, if the pacemaker is giving impulse to atria, EKG will have pacer spike which is a straight vertical line right before the origin of P wave. Similarly in ventricular pacing rhythm, location of pacer spike is right before the QRS complex. In dual chamber pacemaker, there are two spikes positioned one in front of P wave and one for QRS.

It is important to remember that in routine telemetry monitoring, the spikes may not appear if the electronic filter setting to eliminate artifacts is enabled within the computer software. Again, in the new generation bipolar pacemakers, this electrical artifact is less obvious because of the close proximity of both positive and negative poles compared to old generation pacemakers where the lead was considered positive pole and the generator unit was negative.

Complications During Pacemaker Therapy

Capture Failure

In ideal setting, each pacemaker impulse should produce corresponding P or QRS complex depending on the chamber being paced. However in some instances, the EKG will show *pacer spikes at regular intervals* denoting normal functioning pacemaker with *no trailing P or QRS complexes*. Here, the myocardium did not capture the impulse given by the pacemaker.

Common causes for capture failure are (1) *lead malfunction* such as broken or dislodged leads either at myocardium or pacemaker generator level, (2) increased energy requirement for myocardial stimulation (*increased pacer threshold*) secondary to *metabolic or electrolyte imbalance*, *fibrosis of myocardium* at the site of lead insertion and use of *antiarrhythmic drugs* (increase pacer threshold).

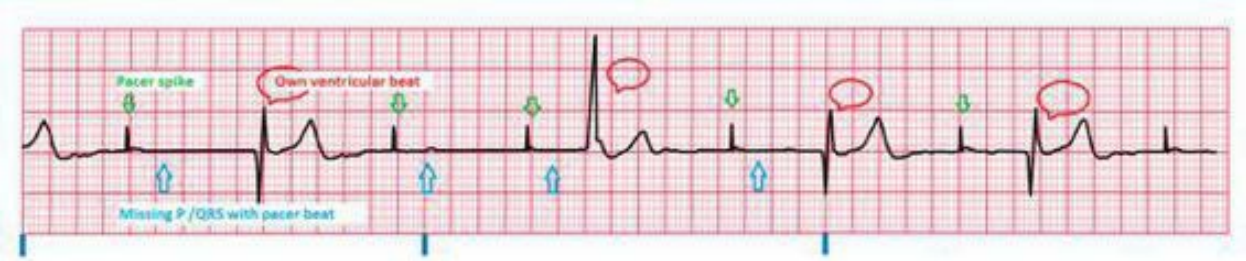


Fig 10.5 Failure to capture

Failure to Pace

This situation is evident in the EKG as *missed beats when pacemaker was supposed to initiate an impulse*. If left untreated, this rhythm is dangerous as it essentially jeopardizes the reason for having a pacemaker at the first place. Probable causes for failure to pace include *weak battery*, *lead failure*, programming issues such as *poor sensing* and *electromagnetic interference (EMI)*.

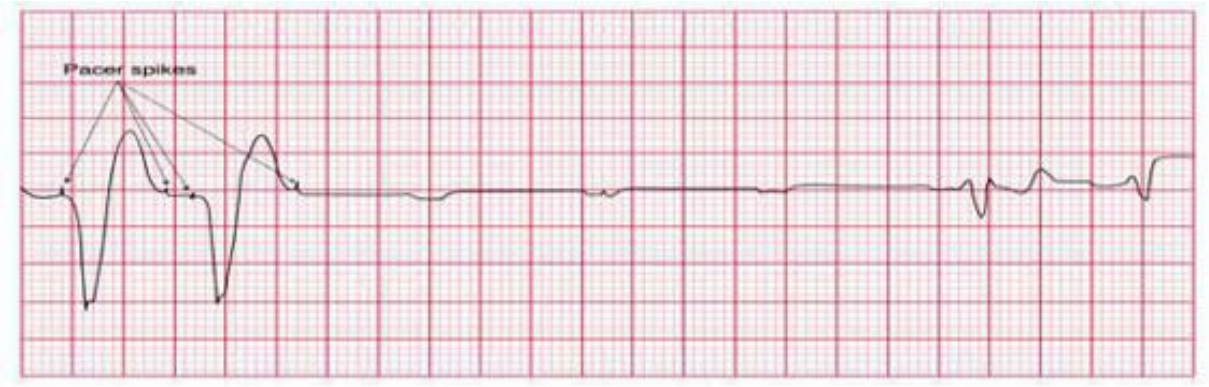


Fig 10.6 Failure to pace

Failure to Sense (Under Sensing)

Here, the pacemaker *does not sense the intrinsic impulse for which it was supposed to inhibit pacing function*. In EKG, there are *multiple pacer spikes irrespective of the patient's own impulses*. Interestingly, all of these pacer impulses do not generate

corresponding cardiac activity because of the **absolute refractory period** within the cardiac cycle. However, if any of these pacer beats happen to strike on the **downward slope of T wave**, where ventricles are most vulnerable for lethal arrhythmias; **ventricular tachycardia** may result. This is mostly caused by *under sensing*, *lead malfunction*, *electromagnetic interference* etc.

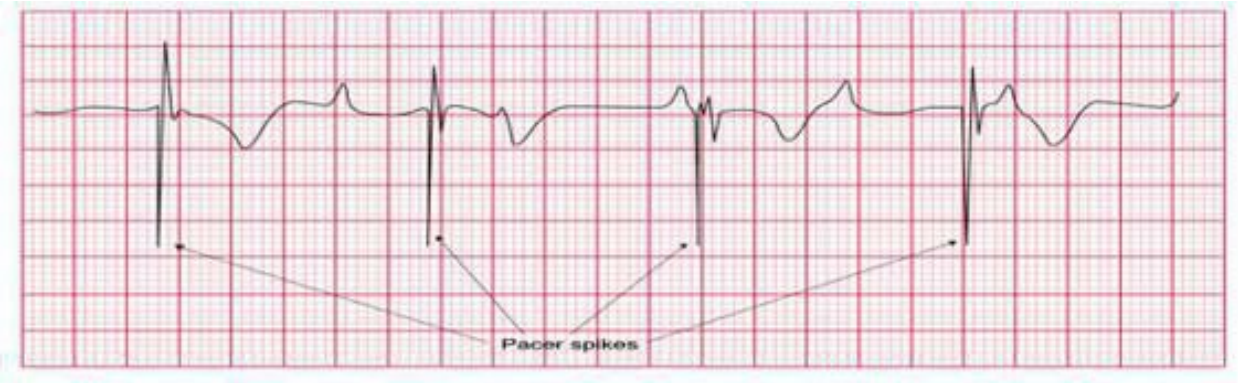


Fig 10.7 Failure to sense

Over Sensing

In this situation, the pacemaker can *misinterpret* muscle movements and normal cardiac waveforms like T wave as innate electrical impulses and can act inappropriately. Misinterpreting T waves as QRS complex and interpreting entire rhythm as tachycardia when the heart rate is within normal limits; is commonly seen in high-energy devices like ICD. This is dangerous because these devices are programmed to terminate ventricular tachycardia by override pacing or electrical cardioversion. Therefore, the patient can get **inappropriate shock therapy** from these devices in these situations. *An appropriate programming change in the devise is* the treatment of choice.

Pacemaker Safety-Patient Education

There are many misconceptions regarding activities and appliances a patient with pacemaker can engage in daily life. It is very important to adequately reassure and inform the patient regarding ***do's and don'ts*** after having a pacemaker. The most important and serious concern is the possibility of **electromagnetic interference** (**EMI**) from adjacent devices.

As a matter of fact, pacemakers have come a long way from their predecessors so that newer generation devices are very much sealed from outside electromagnetic interference generated by day-to-day appliances including cell phones and microwave ovens. Different manufacturers give specifications about their devices; however, in general these devices are safe as long as the person who has the pacemaker stays at a reasonable distance from EMI generating devices. Most common *unsafe devises for pacemakers are high-energy electromagnetic devices such as MRI scan, electrical generators, welding equipments* etc.

Points to Remember!!!

- Fixed rate pacemakers provide impulses at a fixed rate irrespective of person's own cardiac activity; however, demand pacemakers generate impulses within a programmed range based on innate cardiac function.
- Depending on the number of chambers sensed or paced, pacemakers can be single or dual chamber.
- In complete heart block dual chamber pacemaker is of great use, since there is no connection between the atria and the ventricle.
- Biventricular pacemakers are used to re-establish synchronization of ventricular contraction.
- Depending on the duration of use, pacemakers can be temporary or permanent.
- Most common complications of temporary pacemaker are infection and lead dislodgement.
- There are different modes of pacing exists based on the chamber's being paced or sensed and the mode of response of pacemaker based on the intrinsic cardiac activity.
- Rate responsiveness allows the pacemakers to respond appropriately in the event of erratic impulse production such as atrial fibrillation.
- Paced EKG rhythm shows the pacemaker spike before P or QRS or both waveforms depending on the chamber being paced.
- Pacer spikes are not visualized in the EKG if the electronic filter setting to eliminate

artifacts is enabled.

- Compared to older generation unipolar pacemakers, the spikes produced in newer bipolar pacemakers are relatively small and sometimes not seen in the EKG.
- In 'failure to capture', there will be pacer spikes in EKG with no corresponding QRS complexes.
- Lead malfunction, increased pacer threshold, myocardial fibrosis, use of antiarrhythmic drugs etc. are common causes of capture failure.
- 'Failure to pace' exists when the pacemaker failed to initiate an impulse when it is supposed to do so.
- Lead failure, weak battery, poor sensing and electromagnetic interference are the possible causes of pacing failure.
- When the pacemaker does not recognize innate cardiac activity and provide paced impulses inappropriately, it is called failure to sense or under sensing.
- Pacing in the down slope of T wave can produce polymorphic ventricular tachycardia.
- During over sensing, pacemaker misinterprets tall T waves as QRS complexes and respond as if there is tachycardia exist (essentially double counting QRS complexes and so as the ventricular rate).
- Pacemakers are not safe with high-energy electromagnetic devices such as MRI scan, electrical generators, welding equipments etc. However, are pretty safe with the day-to-day household appliance.

Test Your Understanding

1. Which of the following is true regarding fixed rate pacemakers?

A They provide impulse depending on person's heart rate
B They provide impulse irrespective of person's heart rate
C They provide impulse to both atria and ventricle
D They only have left ventricular lead

2. Which of the following option of pacemaker is ideal for sick sinus syndrome?

A Fixed the rate of pacemaker
B Demand pacemakers
C Biventricular pacemaker
D Implantable defibrillator

3. Cardiac resynchronization therapy (CRT) is achieved by_____

A Atrial pacemaker
B Single chamber ventricular pacemaker
C Dual chamber demand pacemaker
D Biventricular pacemaker

4. Which of the following is the most important goal achieved by cardiac resynchronization therapy (CRT)?

A Re-establishing atrio ventricular synchrony

B Preserving simultaneous ventricular depolarization
C Preventing atrio ventricular synchrony
D Suppressing SA node

5. Which of the following is not a complication of temporary pacemaker implantation?
A Infection
B Long-term skin erosion
C Lead dislodgement
D Bleeding

6. A permanent pacemaker is not indicated in _____
A Complete heart block
B Idioventricular rhythm
C Symptomatic bradycardia
D Sinus tachycardia

7. The third letter in pacemaker mode represent_____
A Chamber paced
B Chamber sensed
C Rate response
D Response to intrinsic rhythm

8. In a DDIR pacemaker mode, second letter D stands for_____
A Single chamber pacing
B Single chamber sensing
C Dual chamber sensing
D Dual chamber pacing

9. Which of the following situation benefit from rate responsiveness of a pacemaker?
A Normal sinus rhythm
B Sinus bradycardia
C Atrial fibrillation with rapid ventricular response
D Atrial fibrillation with a controlled ventricular response

10. Which of the following represent 'failure to pace'?
A Presence of extra beats
B Missing beats when pacemaker was supposed to fire
C Presence of pacemaker spike without corresponding rhythm
D Presence of spike within an intrinsic QRS complex

Answers

1. B They provide impulse irrespective of person's heart rate
2. B Demand pacemakers
3. D Biventricular pacemaker
4. B Preserving simultaneous ventricular depolarization
5. B Long-term skin erosion
6. D Sinus tachycardia

7. D Response to intrinsic rhythm
8. C Dual chamber sensing
9. C Atrial fibrillation with rapid ventricular response
10. C Presence of pacemaker spike without corresponding rhythm

Part III

Comprehensive

12 Lead EKG

Analysis

Chapter 11

12 Lead EKG

In this chapter

- Steps in12 Lead EKG interpretation
- Electrical axis of the heart
- Axis deviation
- Right bundle branch block
- Left anterior and posterior hemiblock
- Left bundle branch block

A 12 lead electrocardiogram is essentially the most affordable, accessible and basic diagnostic evaluation that provides a comprehensive view of the electrical activity in the heart. As we discussed in earlier chapters, individual leads provide only a single view of the three-dimensional heart. Since we are trying to recreate a two-dimensional image of the three-dimensional structure of the heart, we have to put together all the available views that provide

maximum amount of information. In a 12 lead EKG; the leads are generally grouped together based on their representation of different walls of the heart. Combination of **limb leads** (lead **I**, **II** and **III**), **augmented leads** (**aVR**, **aVL**, **aVF**) and **precordial leads** (**V1-V6**) are generally used collectively in identification of underlying physiologic processes.

Steps in 12 Lead EKG Interpretation

A systematic approach should be used similar to that of a single lead interpretation while accessing a 12 lead EKG. This method will prevent overlooking any information that could be vital in diagnosis and treatment of a given patient. Important steps are as follows

1 . **Determine the rhythm**: Just like assessing a single lead EKG, *look for the presence of* **P** wave, **QRS** complex, **T** wave and *their characteristics* and *morphology*. *Heart rate*, *various intervals* and *presence of any ectopic beats* should be noted. Some 12 lead EKG machines provide two or more running leads along with regular 12 leads, which help in identifying basic characteristics.

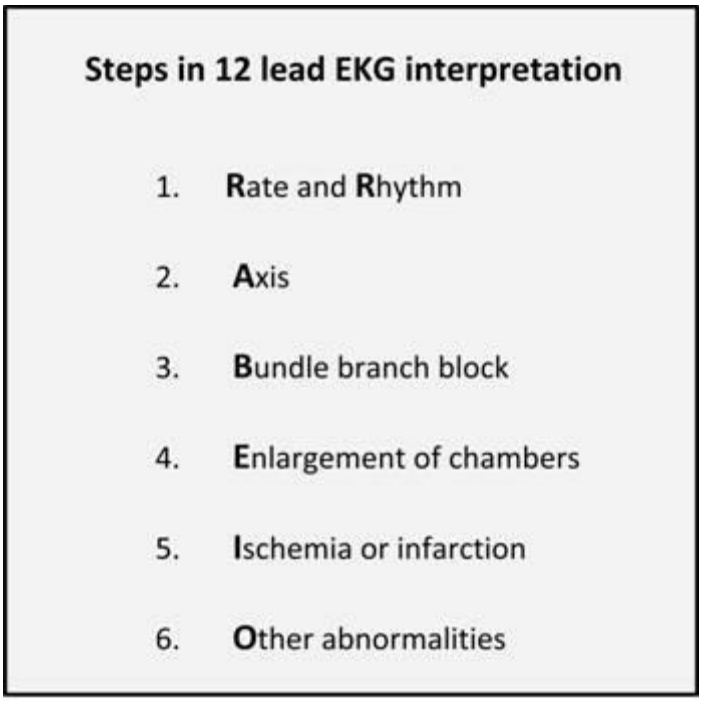
2. **Determine overall axis**: Electrical axis of the EKG simply shows the *direction of electricity flow* within the heart. Normally it is directed towards **left and inferior** aspect of the heart (towards left ventricle). Any defects in normal conduction pattern such as bundle branch block and chamber thickness (hypertrophy) changes overall axis of the EKG.

3. **Presence of block**: Assess for **bundle branch block** or **hemiblock.**

4. Check for **chamber enlargement** or thickness (Hypertrophy).

5. Check for **ischemia** or **infarction**

6. Look for **other abnormalities** like hyperkalemia, A-V dissociation etc.



Box 11.1 Steps in EKG interpretation

Electrical Axis of the Heart

Electrical axis simply means, *overall direction of the electrical flow within the heart*. As we discussed earlier, in a normal heart the electrical impulse starts from the **SA node** and then it travel down to the **AV node** and then spreads across the ventricles through **Purkinje fibers**. The *left ventricle being the major pumping chamber and having the greatest muscle thickness*, *much of this current is flowing towards left ventricle* and essentially **leftward and downward**. This direction is also known as **leftward and inferior** and is **the normal axis** of the EKG. Any defects in electrical circuit changes the direction of electric flow and thereby the axis. Therefore, axis determination helps in identifying *structural as well as electrophysiologic defects* in the heart.

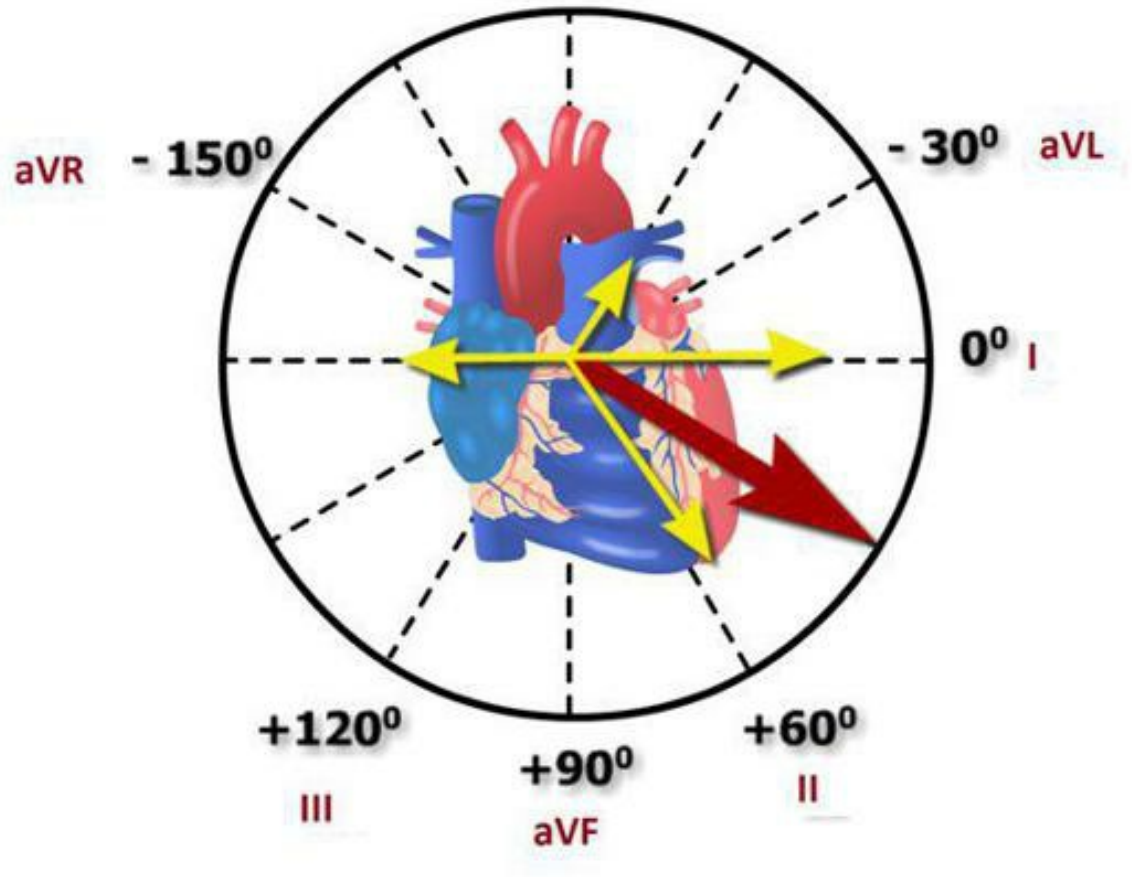


Fig 11.1 Mean electrical axis of the heart

Some of the basic concepts of EKG are very valuable not only in identifying the axis, but also understanding other major concepts in 12 lead interpretation process. Two most important basic concepts are

1. Electricity flows ***towards positive electrode*** generate a ***positive*** *(upright)* deflection in the resulting EKG

2. Individual leads in an EKG provide **single linear view** of electrical activity of the heart.

Applying these basic principles to the conceptual diagram developed by William Einthoven in the turn of 20th-century for which he won the Nobel Prize, it is easier to identify and decipher the conglomeration of various leads in a 12 lead EKG into more manageable and clinically relevant information. Therefore, it is imperative to adequately understand and memorize this session in order to proceed through other areas of EKG interpretation.

Causes of Axis Deviation

Right axis deviation

- Normal variation in children and thin adults
- Chronic lung disease
- WPW syndrome
- Left posterior hemiblock
- Antero lateral MI
- Right ventricular hypertrophy

Left axis deviation

- Normal variation in obesity, pregnancy and ascites
- Left anterior hemiblock
- Inferior wall MI
- Left ventricular hypertrophy
- Premature ventricular contraction

Indeterminant axis deviation

- Premature ventricular contraction

Box 11.2 Causes of axis deviation

Visualizing Axis in the Frontal Plane

Remember, lead **I**, **II**, **III**, **aVR**, **aVL** and **aVF** constitute the **frontal plane leads** in a 12 lead EKG. **Precordial leads V1** to **V6** provide the view of the heart in *horizontal plane*. Among the frontal leads, lead **I** has a positive electrode on the left shoulder and negative electrode on the right. Therefore in lead **I,** an electrical activity travelling *towards left* will generate a *positive deflection* in the resulting EKG. Hence, lead **I** can be used in *differentiating impulses going towards* ***left*** (EKG with a positive wave) *from that going towards* ***right*** (a negative wave).

Similarly, lead **aVF** that is situated with a positive electrode on the left lower extremity; differentiate electricity going towards ***up*** or ***downward direction***. Even though a bit

confusing, it is important to remember this principle that when electricity flows **downward** (towards positive electrode), it produces a **positive waveform** in lead **aVF**. This is because the lead **aVF**, as mentioned before, has a positive electrode situated towards the lower end of the body and the direction of current is towards this positive electrode. Conversely when **aVF** is **negative**, it denotes electricity flowing in **upward direction**.

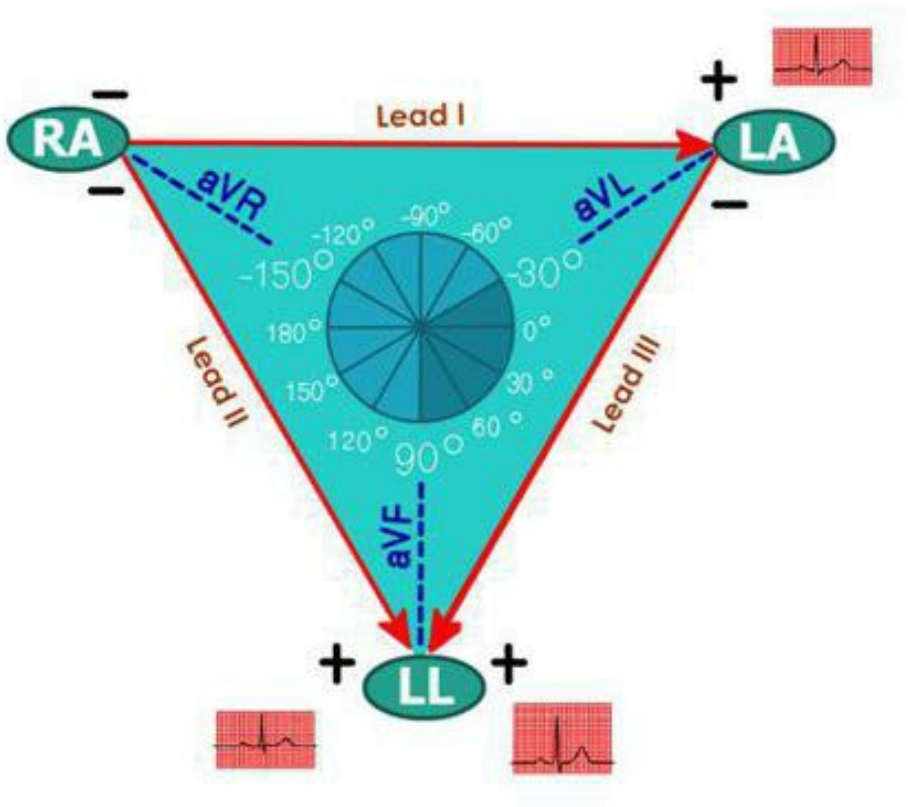


Fig 11.2 Frontal leads with their positive electrodes

Similarly, the rest of the limbs leads are also positioned themselves within this diagram and shows direction of QRS complex depending on whether the impulses travel towards or away from them. Even though lead **I** and **aVF** are the initial leads we assess in order to get a general orientation of the axis, other augmented and limb leads help to pinpoint the axis within this diagram.

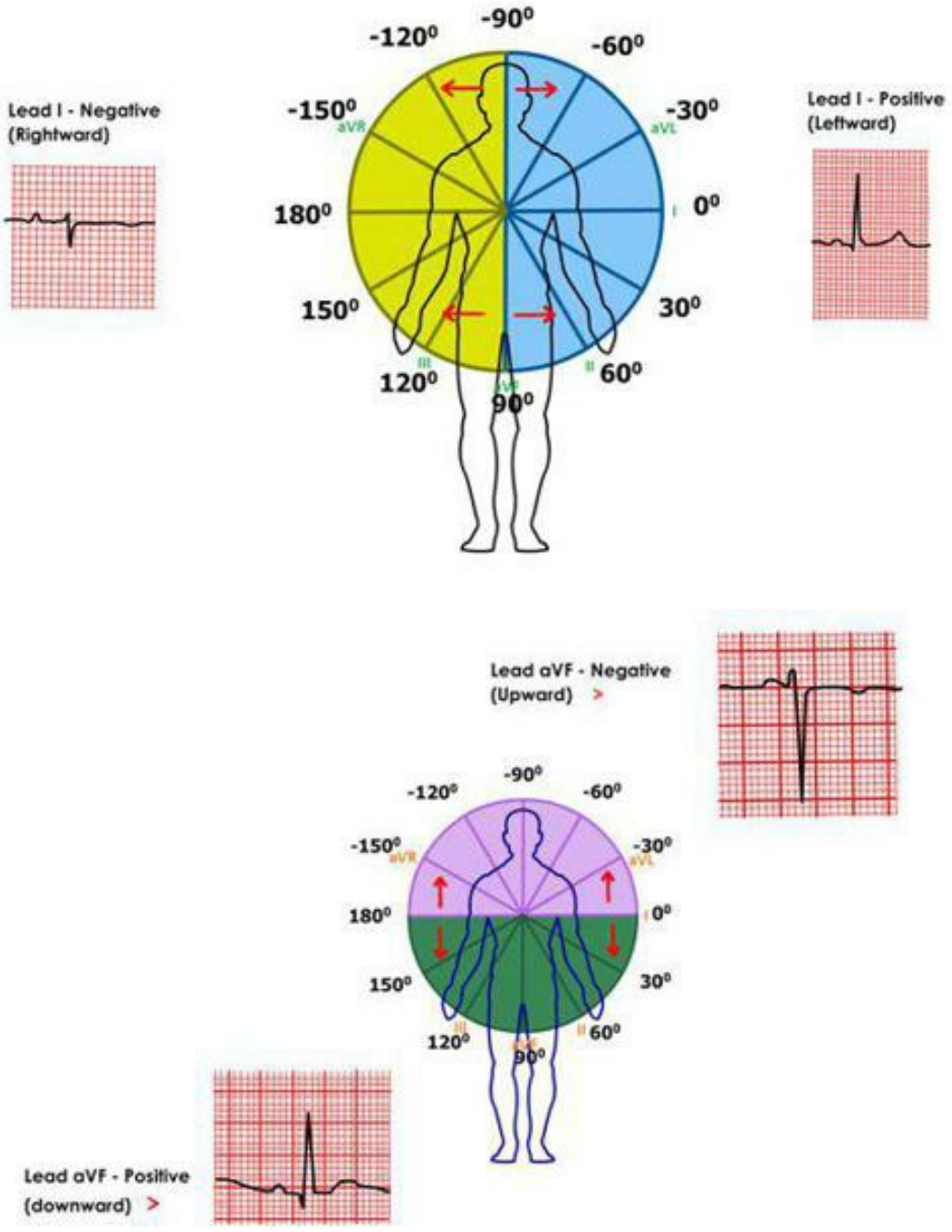


Fig 11.3 Lead I and aVF with their waveform and corresponding direction

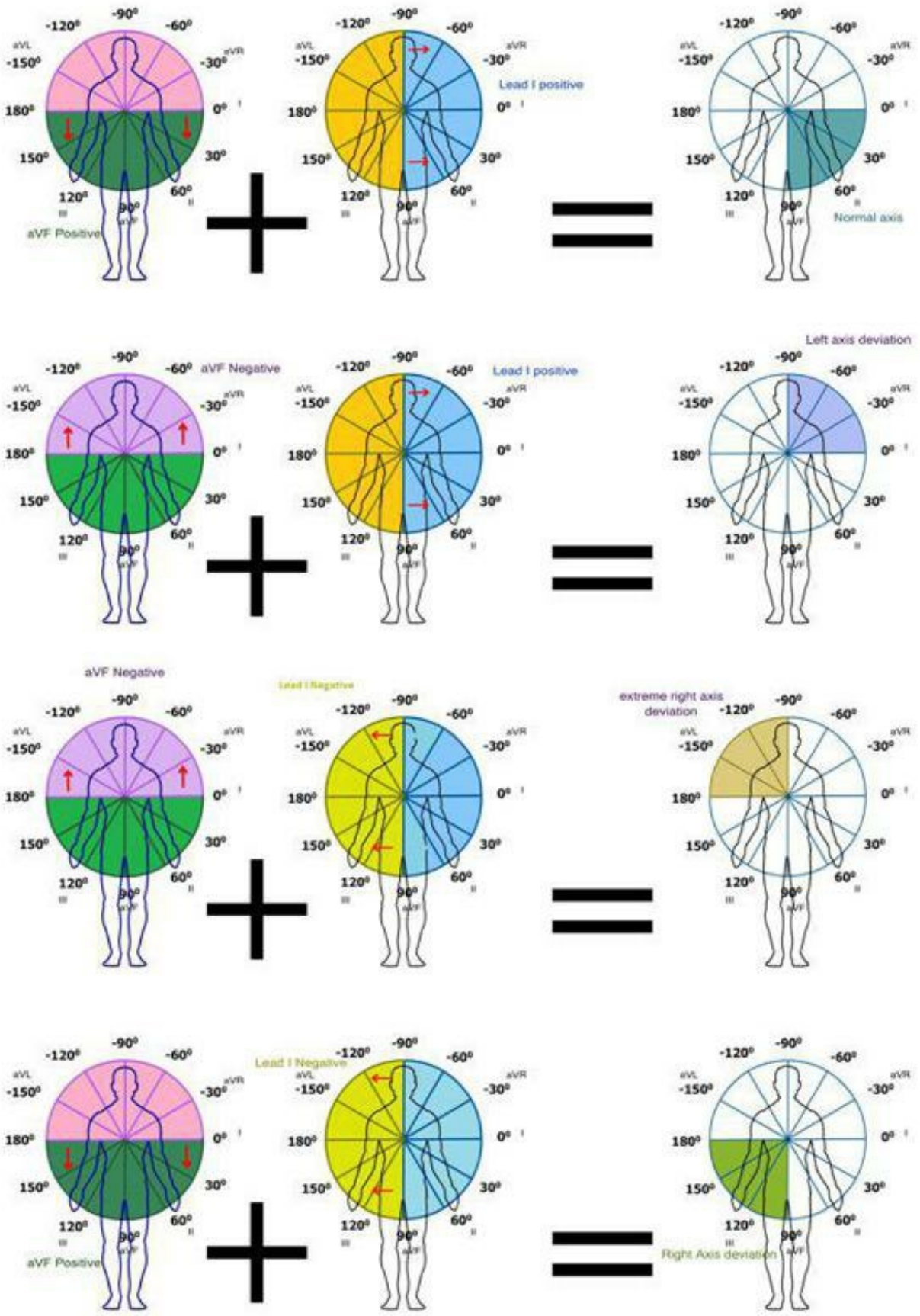


Fig 11.3 B Simple method for axis determination with Lead I and aVF

Simple Method for Right or LeftAxis Determination			
Lead	**Normal Axis deviation**	**Right axis deviation**	**Left axis**
I	Positive	Negative	Positive
II	Positive	Positive or negative	Negative
III	Positive or negative	Positive	Negative
aVF	Positive	Positive	Negative

Box 11.3 Right and left axis determination

We are emphasizing heavily on determining the axis and direction of electrical forces in this session because they serve as the foundation for the rest of EKG analysis including identification of bundle branch block.

Once the vertical axis is determined, it is important to see the direction of electricity in the horizontal plane because of the three-dimensional structure of the heart. Normally the direction of flow of current is **towards the left ventricle** and that is towards **posterior** (Remember, anatomically right ventricle is more towards the anterior chest wall. Compared to the right ventricle, the left ventricle is situated more posteriorly). Flow of current in posterior direction is shown by a **positive wave** in lead **V6**, which is closer to the left ventricle (Remember the position of **V6** electrode). Since lead **V1** and **V2** are situated in the anterior aspect of the chest wall, any flow of current in the anterior direction results in a positive waveform in these leads. The precordial leads are arranged in an *anterior to posterior* fashion so that they help to pinpoint the direction of electrical flow in the horizontal plane.

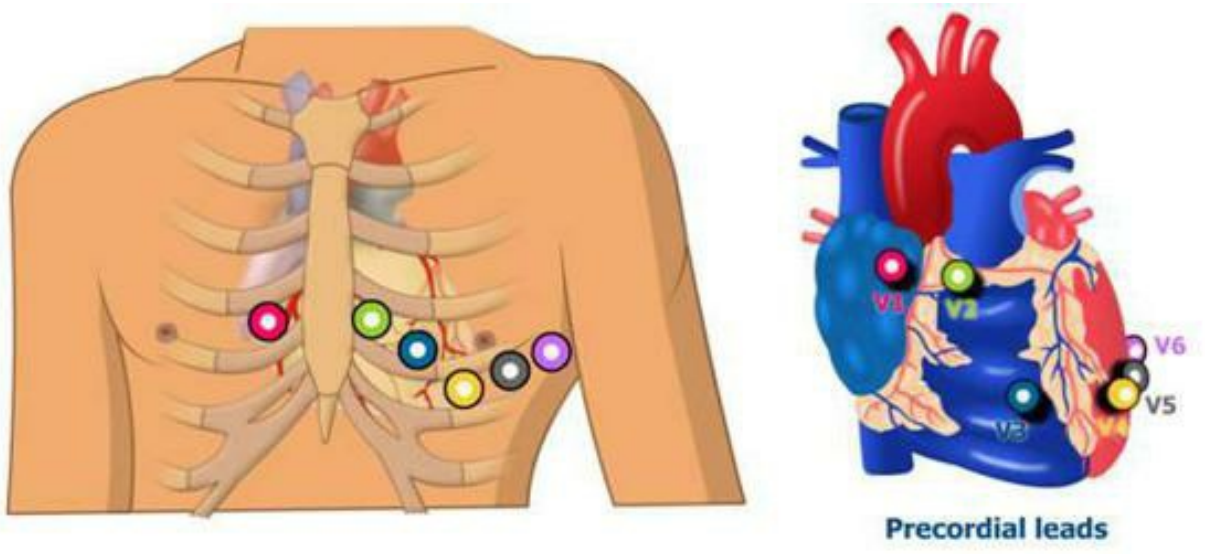


Fig 11.4 Precordial leads in relation to heart

Coronary Circulation to the Conduction System

Conduction system tissues receive blood through various branches of right and left coronary arteries. In general, right coronary artery (RCA) supplies blood to the proximal part of the conduction system including SA node and AV node and left anterior descending artery (LAD) provides supply to middle and distal aspects of the conduction system. As a failsafe mechanism, some of the key elements of conduction system receive *dual blood supply* from two different arteries and that ensures adequate function of these areas in the event of the compromise of any one of the arteries.

The Sino atrial node (SA node) is predominantly supplied by branch of right coronary artery called **SA nodal branch**. The *AV node and bundle of his receive dual blood supply from both the right coronary artery (RCA) and branch of left anterior descending artery (LAD). Left circumflex artery provides blood supply to the posterior fascicle of left bundle branch*. The distal aspect of the bundle branch as such as *right bundle branch and anterior fascicle of left bundle branch receive blood supply from the left anterior descending artery*.

Adequate understanding of the relation between coronary blood supply and conduction system is very important because in many clinical situations involving occlusion of any of these arteries or branches cause corresponding electrical abnormality in the EKG. It also explains the importance of looking for specific types of arrhythmias including bundle branch block, fascicular block or even complete heart block in the event of occlusion of different coronary arteries.

Coronary Circulation to the conduction system	
RCA	SA node, AV node, Bundle of HIS, Posterior fascicle of Left bundle branch
LAD	AV node, Bundle of HIS, Right bundle branch Anterior fascicle of Left bundle branch
LCx	SA node, Posterior fascicle of Left bundle branch

Box 11.4 Coronary circulation to the conduction system

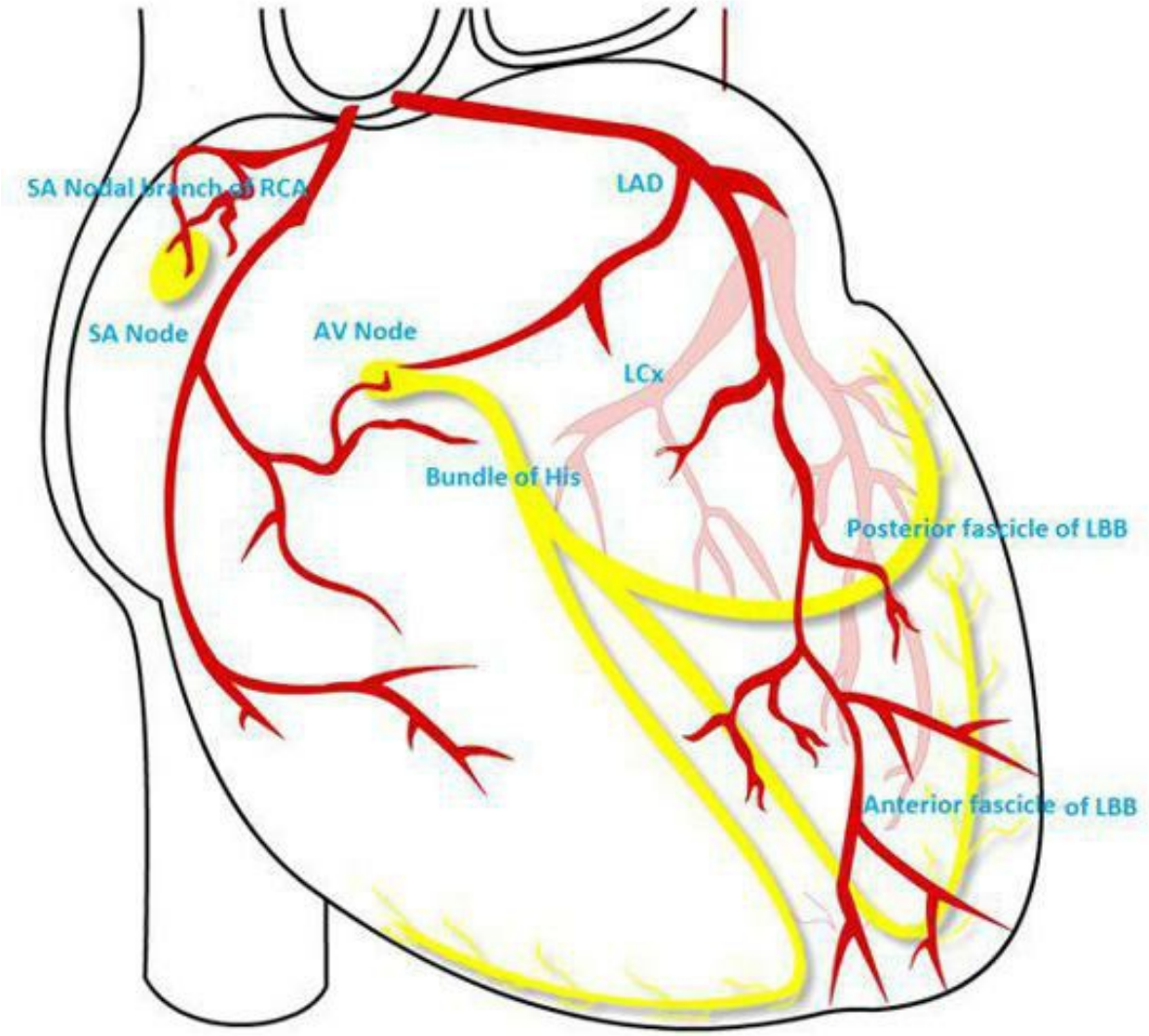


Fig 11.4 B Coronary blood supply to the conduction system

Right Bundle Branch Block

In normal electrical conduction system, impulses traveling down from the AV node take the path of least resistance, which is the **bundle of his**, and subsequent **left and right bundle branches** to spread throughout the ventricle. *Right ventricle is supplied by the right bundle branch* and *left ventricle by anterior and posterior fascicle of the left bundle* branch.

When the right bundle is blocked, the only way right ventricle can get an impulse is from the left ventricle. However, this delay in conduction of impulse from left ventricle to the right ventricle causes the ventricles to contract *sequentially* (back to back) *rather than simultaneously* (together). This process may affect the hemodynamics because of the impaired movement of Inter ventricular septum. In this situation, *rather than flowing towards the left ventricle that is leftward and posterior, the* **last part of QRS complex** *will be directing towards right ventricle, which is* ***anterior*** **and rightward**.

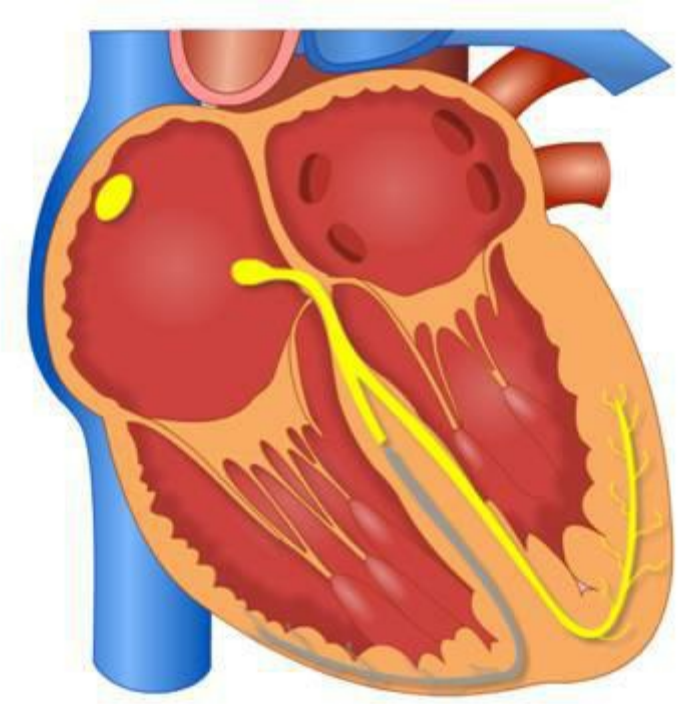


Fig 11.5 Conduction system with RBBB

Mainly lead I, V1 **and** V6 **are considered in identifying** right bundle branch block (RBBB). ***The hallmark of any bundle branch block is a wide QRS complex with duration greater than 0.12 seconds*****. If the duration is between 0.10 seconds to the 0.11, it is called** Inter ventricular conduction delay (IVCD)**.**

Unlike in axis deviation, where the direction of entire QRS complex is under scrutiny, the only area of EKG under consideration in bundle branch block is the ***last half of QRS*** *complex.*

In right bundle branch block, the net direction of flow of current is towards right

ventricle, which is *anterior and rightward*. Therefore, along with a wide QRS complex of duration greater than 0.12 seconds, *lead **I**, **V6** will be **negative*** and ***lead V1** will be **positive*** for right bundle branch block as shown in Fig 11.6. Because of the characteristic electricity flow of bundle branch block in general, **T waves are in opposite direction of QRS complex**. In an EKG with the right bundle branch block, *it is important to evaluate* **overall axis**, **ST segment elevation**, **myocardial infarction** and presence of **hemiblock**. Since the contraction of right bundle is sequential, this EKG is *not reliable for identifying right ventricle hypertrophy*.

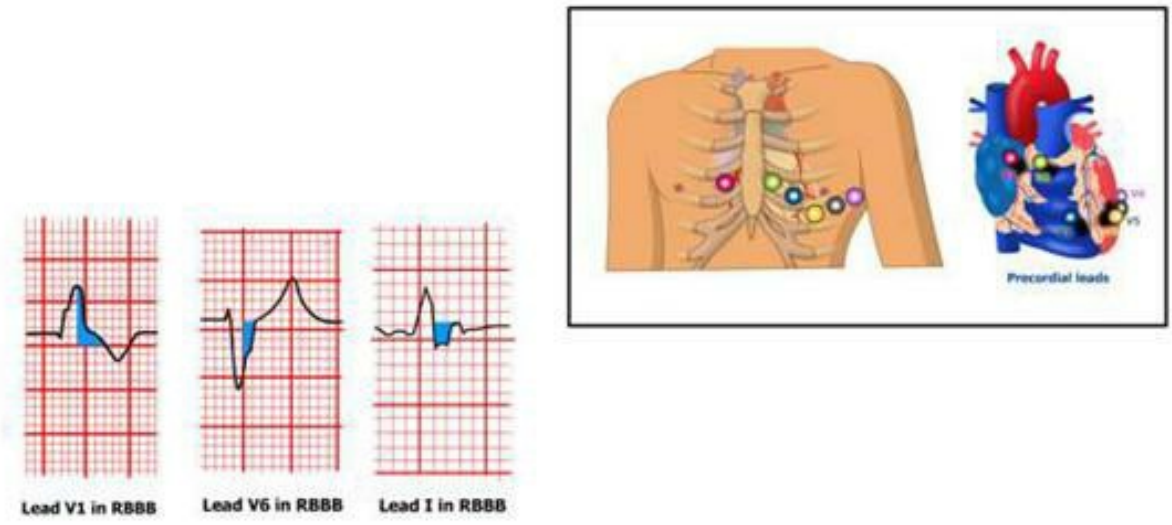


Fig 11.6 Frontal and precordial plane EKG in RBBB (Note the shaded areas under consideration for evaluation of bundle branch block)

*Along with wide QRS complex having duration **greater than** 0.12 seconds, **negative** lead I, V6 and **positive** lead V1 are the criteria for right bundle branch block.*

Left Hemiblock (Left Anterior Superior Hemiblock)

Left bundle has two distinct bundle segments called left **anterior** or **superior fascicle** and left **posterior fascicle**. During *left anterior hemiblock*, *left anterior fascicle is blocked*. In order to depolarize areas of myocardium covered by the anterior fascicle, the impulses spreading through posterior fascicle travel upward (towards anterior).

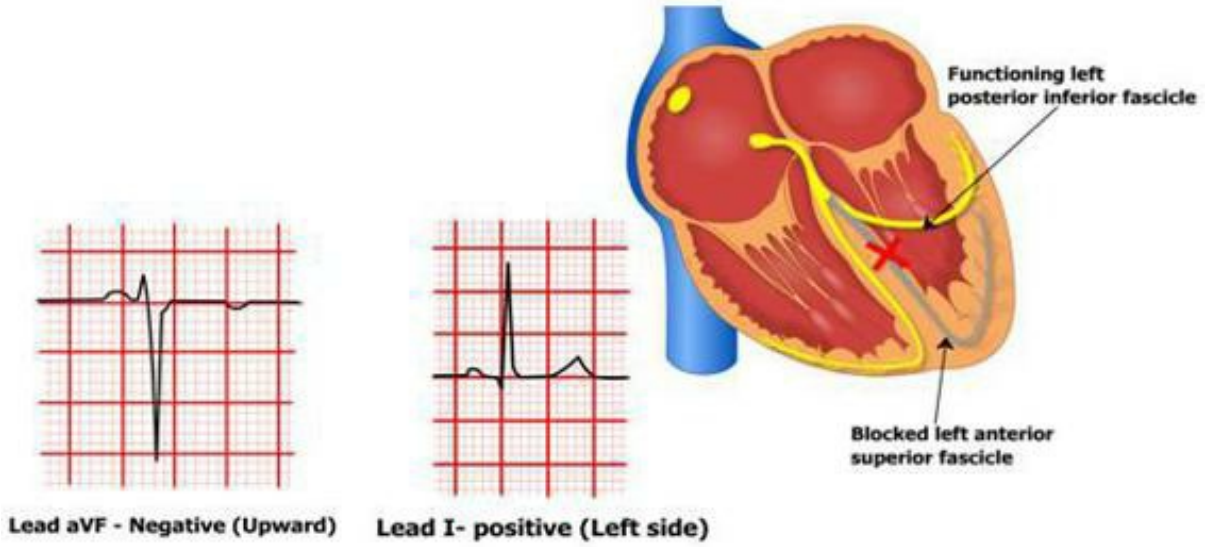


Fig 11.7 left anterior hemiblock direction

This upward movement of current changes the axis of EKG in the frontal plane as evidenced by a **negative aVF** (remember, negative aVF means upward direction of current since aVF has positive electrode towards left leg). The mean direction of QRS in LAHB is therefore **leftward and upward**.

EKG criteria for left anterior hemiblock are

- *positive lead I (left ward) and negative aVF (upward) (Left axis deviation)*
- *qR complex in lateral limb leads (Lead I and aVL)*
- *qS complex in inferior leads (Lead II, III and aVF)*
- *Delayed intrinsicoid deflection*

Since part of the left bundle is still functioning, there *will not be any widening of QRS complex*. However this EKG may have a slight slurring of the QRS complex called **delayed intrinsicoid deflection** where the time taken for the R wave to peak from the beginning of the QRS will be longer than usual (>0.45 Sec or greater than 1 small box). Left anterior hemiblock is also known as **left anterior superior hemiblock**. Left anterior hemiblock has left axis

deviation since **aVL** is positive and lead **II**, **III** and **aVF** are negative.

In **left inferior or posterior hemiblock**, the direction of current will be from the anterior fascicle to the posterior fascicle and essentially *rightward and inferior*. So lead **I** and **aVL** are negative (rightward), lead **II**, **III** and **aVF** are positive (Inferior or downward). Lead **aVR** is mostly isoelectric, showing its perpendicular direction with the axis of EKG.

EKG criteria for left posterior hemiblock are

- *Negative Lead I and positive aVF (Right axis deviation)*
- *rS pattern in lateral limb leads (Lead I and aVL)*
- *Tall R waves in inferior leads (Lead II, III and aVF- goes with right axis)*
- *Looks similar to S1Q3T3 pattern of pulmonary embolus EKG*

Left Bundle Branch Block

As mentioned earlier, the right ventricle is supplied by the right bundle branch and the left ventricle by anterior and posterior fascicles of the left bundle branch. In **left bundle branch block**, *the main left bundle before it's bifurcation in to anterior and posterior fascicle is blocked*.

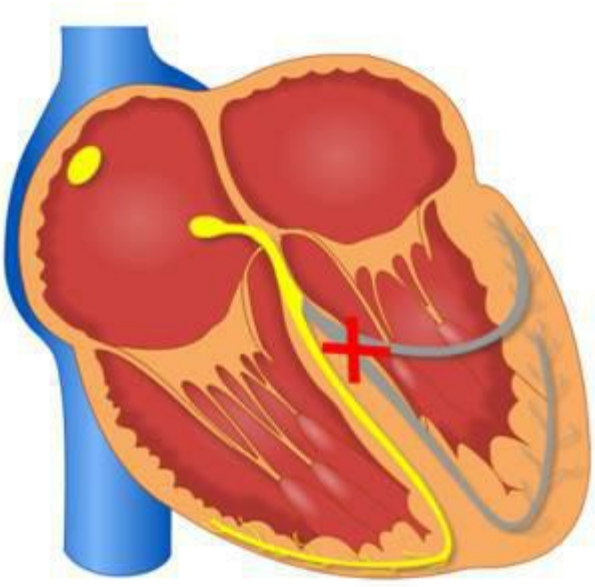


Fig 11.8-Left bundle branch block (Involving both fascicles of Left bundle branch)

This prevents large area of myocardium in the left ventricle to receive electrical impulse through the conventional route. Here, right ventricle contracts first because of the electric impulse through intact right bundle. Then the same impulse travels to the left ventricular myocardium, causing it to contract. Therefore during left bundle branch block, the right and left ventricles contract *sequentially rather than in tandem*.

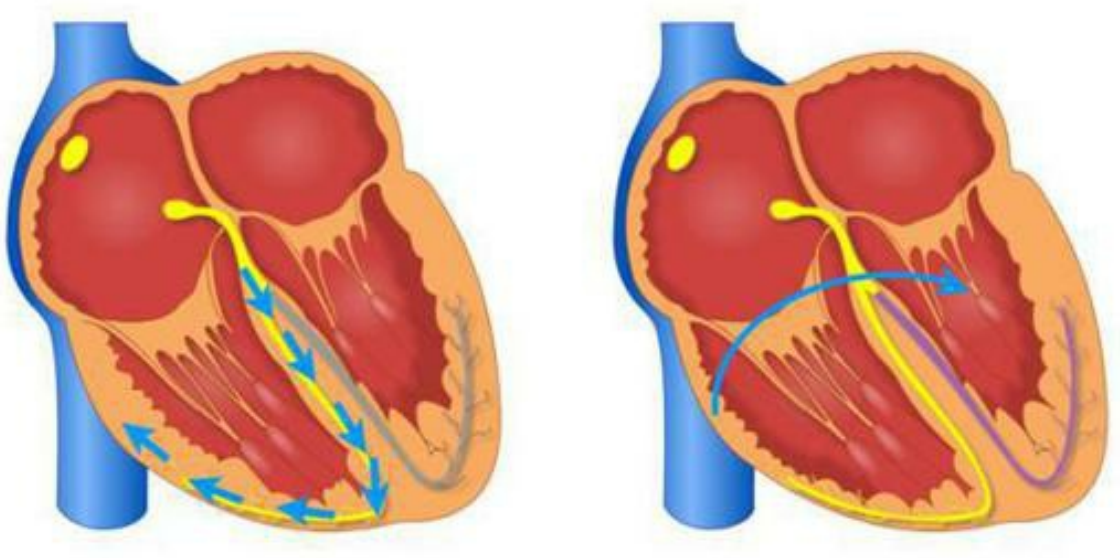


Fig 11.9 LBBB ventricular contraction

Because of the depolarization of right bundle branch before that of the left bundle, areas of the heart including Inter-ventricular septum, which is supplied by the right bundle contracts inward into the right ventricle during right bundle depolarization. Subsequently it remains diskinetic during the sequential left ventricular depolarization. This essentially reduces capacity of left ventricle to pump blood out in to the aorta during systole and cause hemodynamic instability in certain patients. In this situation, the direction of current is from *the right ventricle to the left* and therefore the *last half of QRS direction is towards* patient's **left side and posterior**. (Remember, during assessment of bundle branch block we only look at the *second half of QRS complex and its direction*).

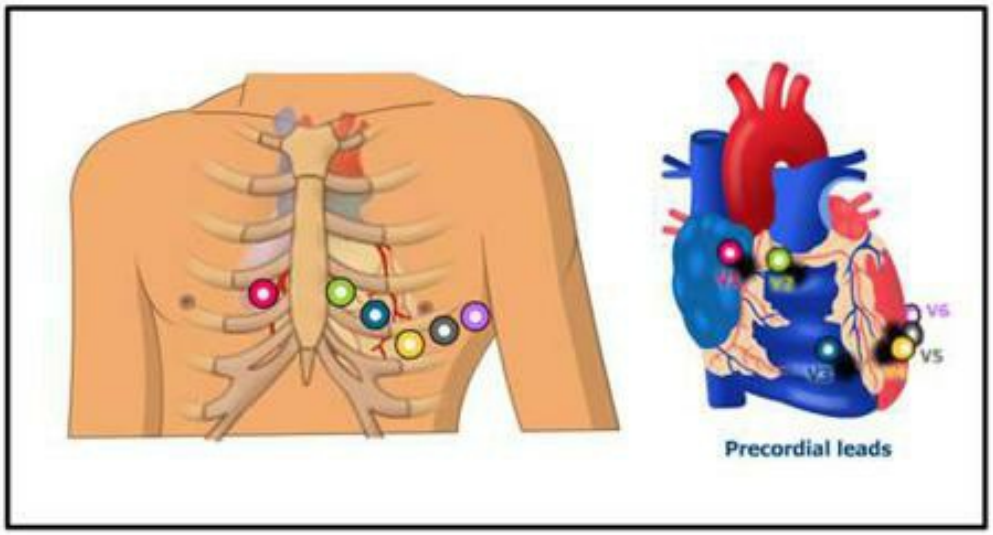


Fig 11.10 LBBB Criteria

*Along with wide QRS having duration greater than 0.12 seconds, EKG criteria for **LBBB** include **positive ending** of lead I and V6 (R or R') and **QS wave** in lead V1.*

Similar to the right bundle branch block, the direction of T wave is opposite to that of QRS complex in left bundle block. Because of the abnormal formation of QRS, LBBB EKG is *not reliable to measure ischemia*, *infarction*, *hemiblock* or *hypertrophy*.

For summery of 12 lead interpretation, please refer to the Appendix A

Points to Remember!!!

- A 12 lead EKG provides three-dimensional view of electrical activity of the heart.
- For accurate interpretation of 12 lead EKG, a stepwise process with identification of rhythm, axis, presence of bundle branch block, chamber enlargement, presence of ischemic changes and other abnormalities needs to be followed.
- Electrical axis represents overall direction of electrical flow within the heart. Normally, it is directed towards left and inferior, i.e. towards left ventricle.
- Presence of structural or electrophysiologic abnormalities may change normal electrical axis.
- Electricity flows towards positive electrode generate positive deflection in the resulting EKG.
- Chronic lung disease, right ventricular hypertrophy, WPW syndrome, anterolateral myocardial infarction etc. are the common reasons for right axis deviation.
- Left axis deviation is seen in left anterior hemiblock, PVC, inferior wall MI etc.
- Left axis deviation is a normal variant in obese or pregnant individuals.
- Premature ventricular contraction may produce in determinant axis.
- A positive waveform in lead **I** shows the leftward direction of current.
- Positive waveform in lead **aVF** represents downward direction of current.
- Lead **I** and **aVF** are used primarily for gross determination of axis.
- In normal axis, lead **I** and **aVF** will be positive.
- In right bundle branch block, the contractions of ventricles are sequential rather than simultaneous.
- The direction of current in right bundle branch block is anterior and rightward.
- For assessing bundle branch block, only the last half of QRS complex needs to be analyzed.
- In right bundle branch block (RBBB), Lead **V1** has a positive ending QRS and lead **V6** and **I** have negative waves as the last half of QRS.
- Wide QRS complex with duration from 0.10 to 0.11 is called inter ventricular conduction delay (IVCD).
- In right bundle branch block EKG; the rhythm can be further analyzed for ischemia, infarction or presence of hemiblock.
- Right bundle branch block pattern is not reliable for assessing right ventricular hypertrophy.
- In hemiblock pattern, there is no widening of QRS complex.
- Left anterior hemiblock changes overall axis of QRS to left and upward direction, creating positive lead **I** and negative **aVF**.
- In left posterior or inferior hemiblock, the direction of current is inferior and rightward resulting in negative lead **I** and **aVL** with positive lead **II**, **III** and **aVF**.
- In left bundle branch block, overall direction of current is towards left and posterior, resulting in an EKG with positive ending lead **I** and **V6** and negative **V1**.

· In bundle branch block pattern, the direction of T wave is opposite to that of QRS complex because of the disorganized repolarization.
· Left bundle branch block pattern EKG is not reliable for assessing ischemic changes.

Test Your Understanding

1. Which of the following is an example for the augmented lead?

A Lead III

B Lead V6

C Lead aVR

D Lead V3

2. 12 lead EKG interpretation can provide information except_____?

A Thickness of ventricular wall

B Presence of ischemia

C Rate of flow through mitral valve

D Atrioventricular synchronization

3. Based on the principle of direction of current and morphology of waveform, which of the following represent direction of current towards left side of the heart?

A Positive lead aVL

B Negative lead V1

C Positive lead I

D Negative lead I

4. Which of the following lead is useful in identifying direction of electricity upward or downward?

A Lead aVR

B Lead aVL

C Lead I

D Lead aVF

5. Which of the following precordial lead has a characteristic tall R wave when electricity is flowing towards left and posterior direction?

A Lead V1

B Lead V3

C Lead V2

D Lead V6

6. Which of the following is not a characteristic of right bundle branch block?

A QRS duration 0.18 second

B QRS duration 0.14 seconds

C Positive ending lead V6

D Positive ending of lead V1

7. Which of the following pathology cannot be assessed from right bundle branch block EKG?

A ST segment elevation

B Left posterior hemiblock

C Left ventricular hypertrophy

D Right ventricular hypertrophy

8. Which of the following statement is true regarding left bundle branch block?

A Even though hemiblock cannot be assessed, ischemic changes are evident in LBBB

B Presence of R or R' in lead I and V6 are characteristic of LBBB

C QRS duration is always greater than 0.18 second in LBBB

D Direction of current flow in LBBB is towards rightward and anterior

9. What is the main difference in method of identifying the axis deviation and bundle branch block from an EKG?

A For axis deviation, consider only first-half of QRS complex whereas in BBB, the entire complex

B In axis deviation, the entire wave to be considered whereas in BBB, only lasts half of QRS

C In BBB, first-half of QRS is under consideration whereas in axis deviation, entire PQRST complex to be considered

D In BBB, QRS complex in the beginning and end of the rhythm is considered

10. Which of the following is not a characteristic of left anterior superior hemiblock?

A Direction of current towards leftward and upward

B QRS duration greater than 0.12 second

C It has positive lead I

D There is negative aVF

Answers

1. C Lead aVR

2. C Rate of flow through mitral valve

3. C Positive lead I

4. D Lead aVF

5. D Lead V6

6. C Positive ending lead V6

7. D Right ventricular hypertrophy

8. B Presence of R or R' in lead I and V6 are characteristic of LBBB

9. B In axis deviation, the entire wave to be considered whereas in BBB, only lasts half of QRS

10. B. QRS duration greater than 0.12 seconds

Chapter 12

Myocardial Ischemia and Infarction

In this chapter

- Pathophysiology of Myocardial infarction
- 12 lead EKG and coronary distribution
- Diagnostic value of 12 lead EKG
- Types of myocardial events
- EKG criteria for various events

In order to diagnose and treat patients with myocardial ischemia and infarction in a timely fashion, the 12 lead EKG has been a cornerstone diagnostic test for last few decades. Because of the widespread availability, minimal expertise for interpretation and instantaneous result have made the 12 lead EKG one of the most widely used clinical diagnostic tool; not only in cardiology, but also in entire modern medicine.

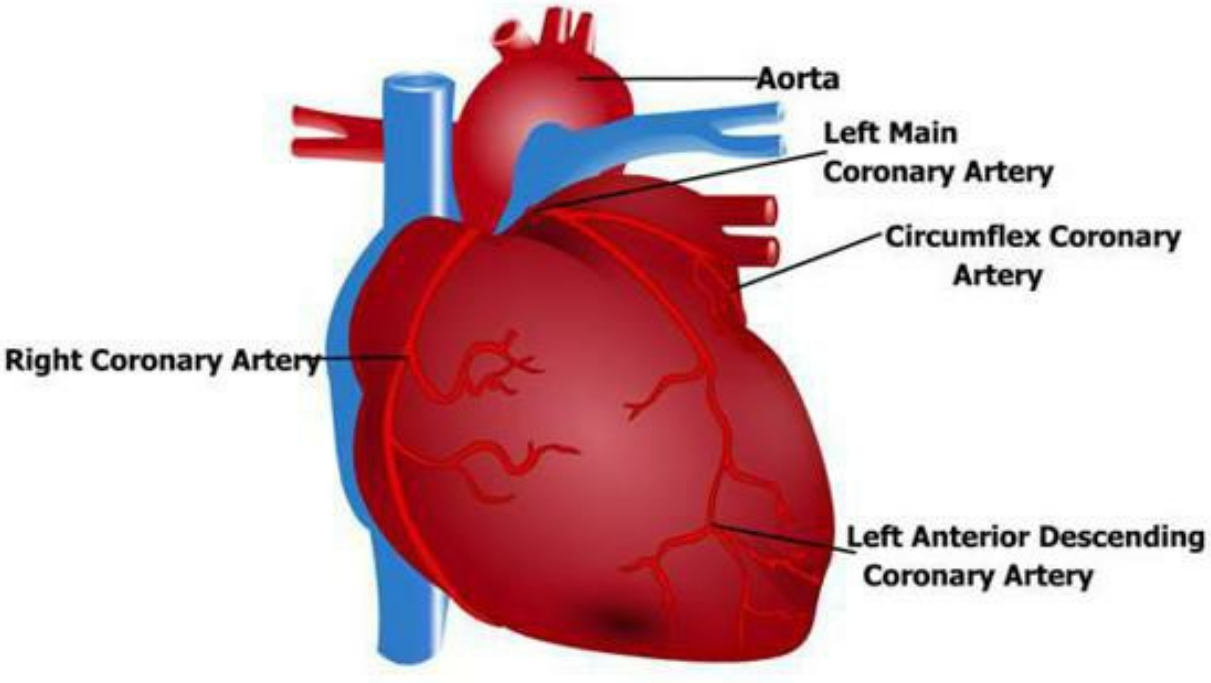


Fig 12.1 Coronary anatomy

Atherosclerotic heart disease (ASHD) accounts for majority of myocardial ischemia and infarction in adult population. Commonly, advanced atherosclerotic heart disease produces a constellation of symptoms known as **Acute Coronary Syndrome** (**ACS**) and **ST elevation myocardial infarction** (**STEMI**).

However, the most common myocardial ischemic event that come across in real world is **Stable Angina Pectoris**, characterized by *chest pain* typically *initiated by exertion* either physical or emotional; which is *relieved with either rest or use of nitroglycerin*. The characteristics of pain include *squeezing, central* or *sub sternal discomfort* (*Levine's sign*) with the *crescendo decrescendo type* (waxing and waning). The pain usually lasts for *2 to 5 min* with *radiation* to *shoulder*, *arm*, *jaw* or *back*. Patient may also has nocturnal angina; however, usually demonstrate considerable difference in symptoms through the course of the day because of the variations in coronary vascular tone.

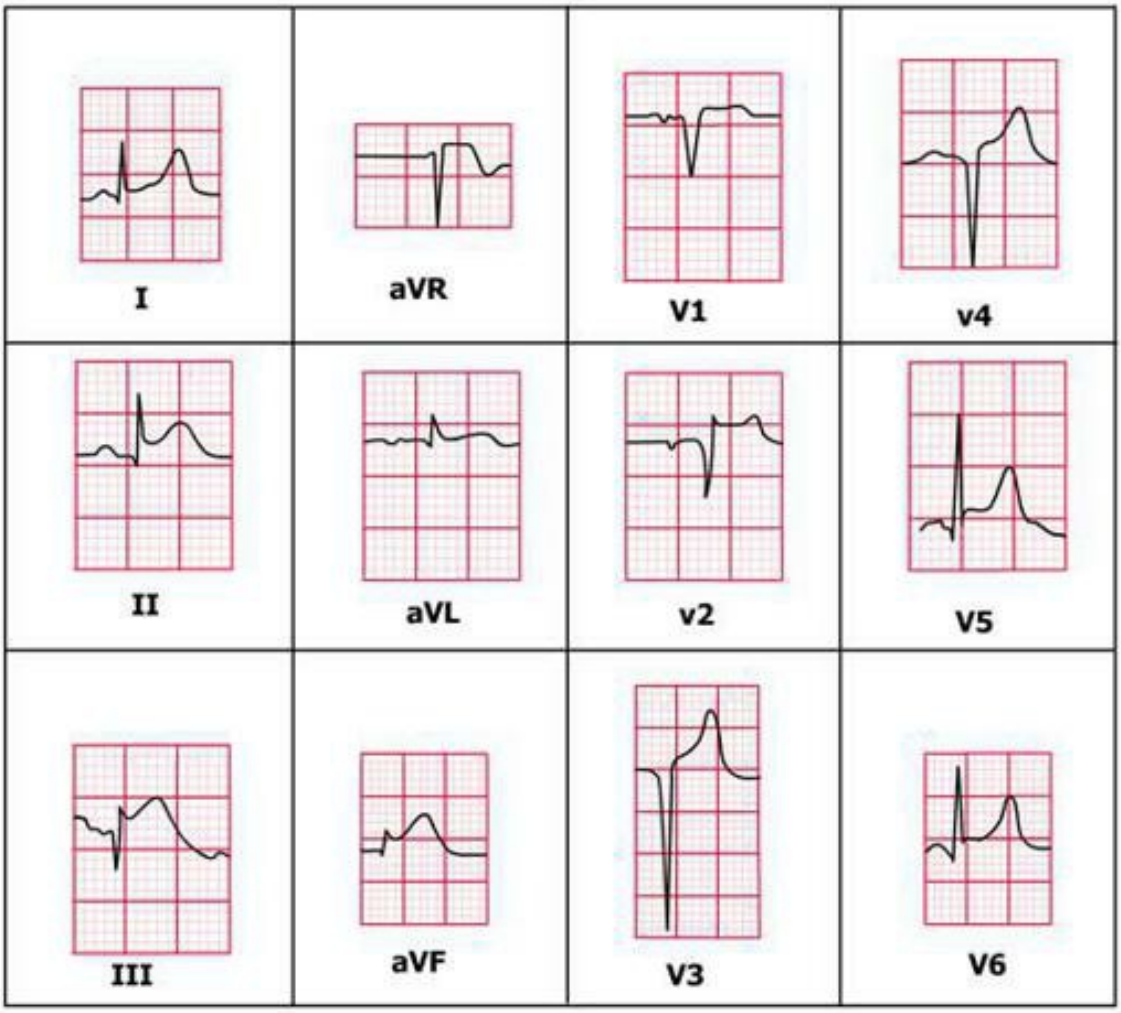


Fig 12.2 ST segment elevation in various leads

Acute coronary syndrome includes two distinct categories of disease process classified based on their presentation, known as **Unstable Angina** (UA) and **Non-ST segment elevation myocardial infarction** (NSTEMI).

Unstable angina (**UA**): Here, the patient presents with *typical cardiac chest pain* with or without radiation to adjacent areas, may or may not *relieved by rest or sublingual nitroglycerin* treatment. These patients may not have any EKG changes or elevation in their target cardiac enzymes such as **Troponin I**, **Creatine phosphokinase** (CK) or **Creatine phosphokinase-cardiac fraction** (CKMB).

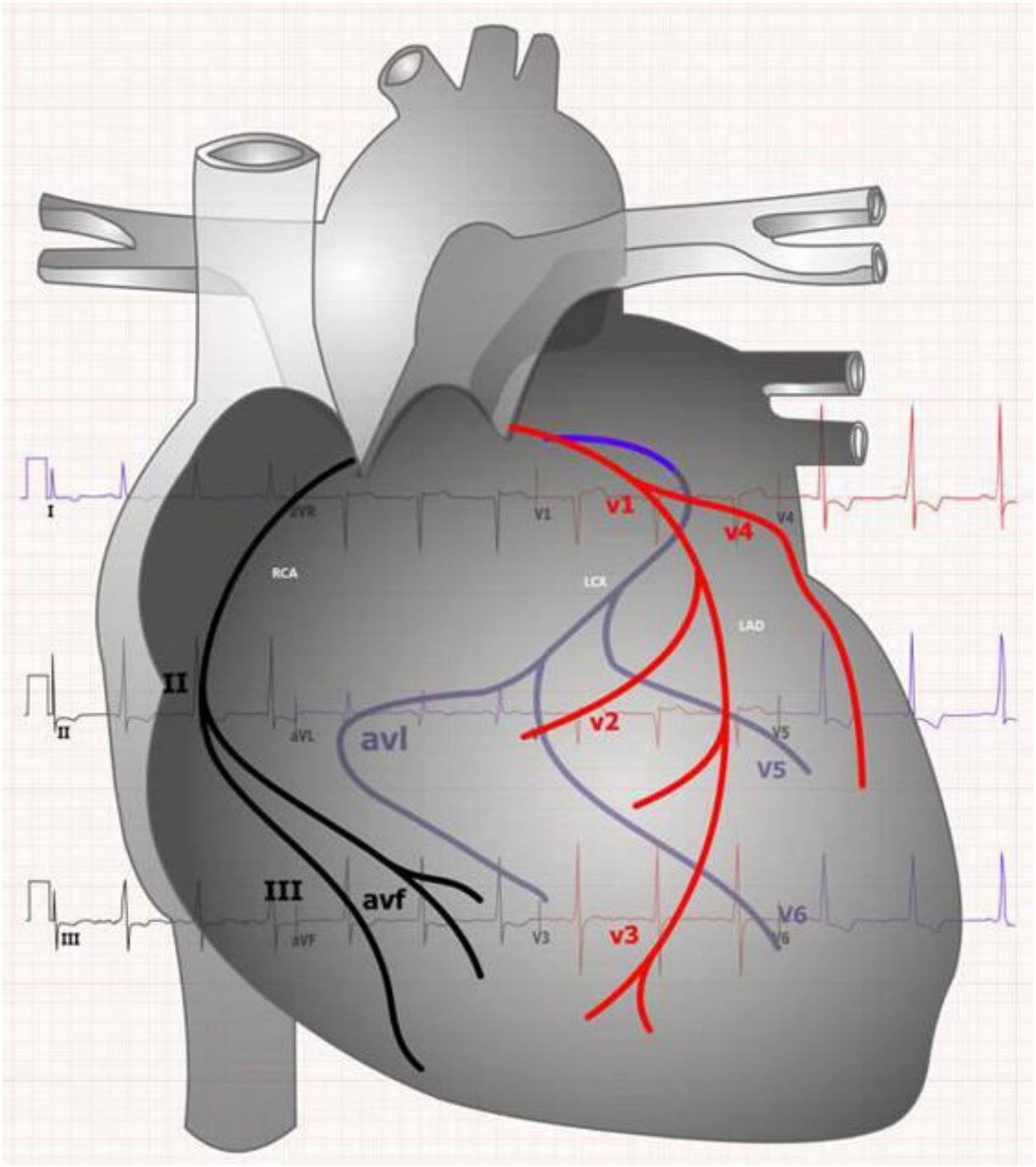


Fig 12.2-B Coronary anatomy and distribution of EKG leads

Non-ST segment elevation myocardial infarction (**NSTEMI**): In these patients, along with *anginal symptoms* there may be *ST segment depression or T wave inversion* and *has positive cardiac biomarkers* such as troponin I and CKMB. Sometimes ST segment changes are transient (short lasting).

ST segment elevation myocardial infarction (**STEMI**): This acute form of myocardial infarction results from *complete and sudden occlusion of a coronary artery*

leading to *acute myocardial injury*. This scenario is characterized by consistent *ST segment elevation greater than 0.1 mV* (larger than one small vertical box in an EKG) in two or more contiguous leads in an EKG or *new onset of left bundle branch block* (LBBB) pattern along with angina. There may be coexisting **mirror image changes** in the form of *ST segment depression and T wave inversion* in the areas of EKG opposite to that of ischemia.

In a 12 lead EKG; ST segment changes are consistent with *area of myocardium supplied by the blocked coronary artery*. For example, in **anterior wall STEMI** the affected vessel is most likely **left anterior descending artery** (LAD) and is evidenced in an EKG by consistent ST segment elevation in lead **V2**, **V3** and **V4** which are looking at electrical activity on the anterior chest wall. There may also be ST segment depression and T wave inversion in inferior leads **II**, **III** and **aVF**, which are on the opposite side of the anterior wall. ST segment changes happen because of the *abnormal electrical transmission and subsequent repolarization in chemically unstable environment that exists in ischemic and infarcted myocardium*.

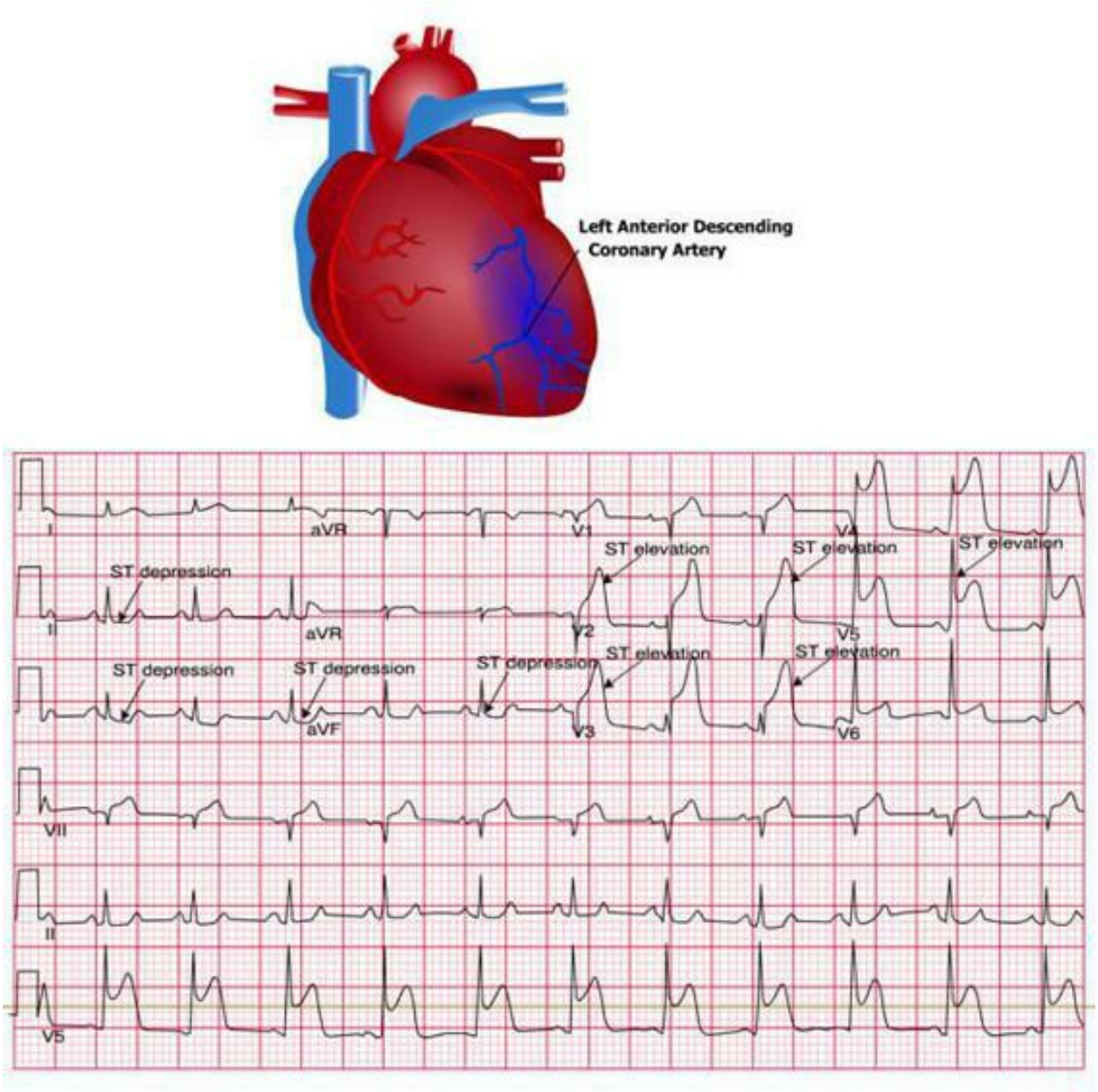


Fig 12.3 Anterior STEMI showing LAD occlusion with resulting EKG changes. (Note the presence of anterior ST elevation and inferior St segment depression changes (Mirror image).

An important principle in diagnosing myocardial events from a 12 lead EKG is that presence of characteristic ST elevation, ST depression, T wave inversion or Q wave in isolated leads are of least diagnostic significance. In order to be clinically relevant, these changes has to be seen in contiguous (neighboring) EKG leads.

For example, presence of Q wave in lead III alone does not indicate inferior wall ischemia without corresponding Q wave in lead II and aVF. This is of extremely important while reporting EKG finding in clinical situations.

Pathophysiology of ASHD

The core of atherosclerotic heart disease pathophysiology is a *disparity in supply and demand of myocardial oxygen*. In normal circumstances, epicardial coronary arteries supply enough blood to support oxygen and nutrient demands of myocardial cells depending on the cardiac workload. In ischemic heart disease, because of the narrowing of coronary arteries some areas of myocardium will deprive of adequate blood flow leading to ischemia and infarction.

Major determinants of **myocardial oxygen demand** (MVO2) are *heart rate*, *myocardial contractility* and *myocardial wall tension*. In normal disease-free heart, the coronary circulation including micro vascular structures are very effective in adapting to the changing needs of heart to ensure adequate myocardial nutrient supply. However, in coronary artery disease the system lacks flexibility and adaptability for changing needs of the heart. Other factors that can adversely affect coronary blood supply are *arterial thrombi*, *coronary vessel spasm*, *emboli* and *severe anemia*.

Coronary atherosclerosis may be produced by factors such as *increased LDL* (Low density lipoprotein), *low HDL* (High density lipoprotein), *smoking*, *hypertension*, *diabetes mellitus* etc. These oxidative events disturb normal function of coronary endothelium, leading to local inflammatory changes along with abnormal cell adhesion and plaque buildup. These chronic changes ultimately produce widespread coronary atherosclerosis.

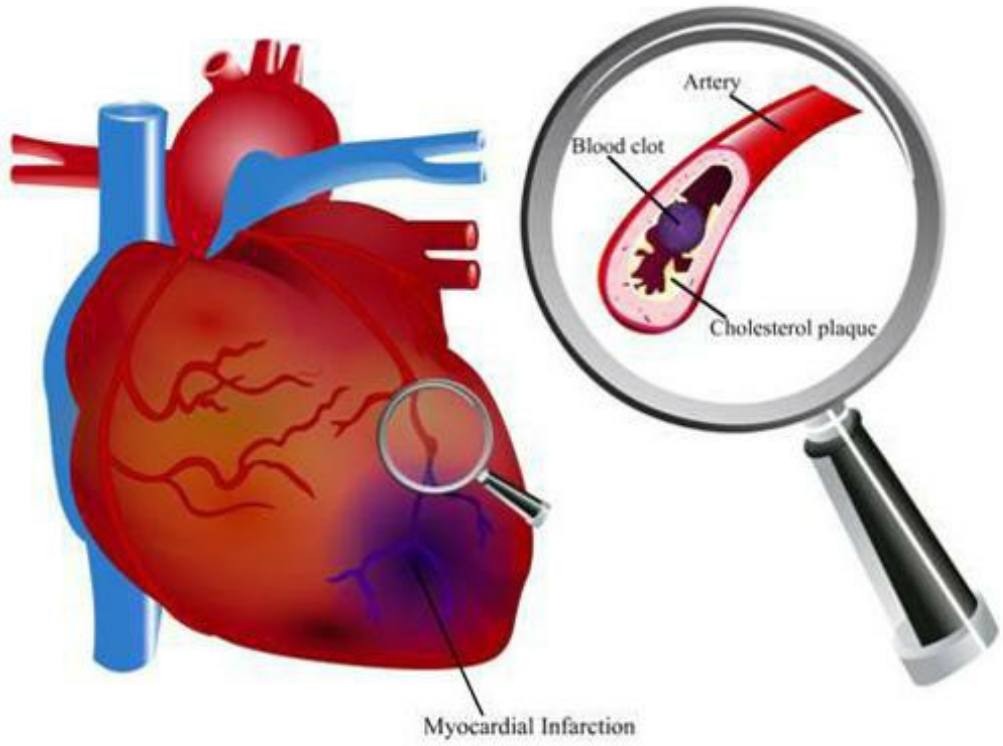


Fig 12.4 Pathophysiology of STEMI

Generally, **50%** reduction in lumen size produces **exercise-induced symptoms** and when it reaches greater than **80%**, patient will have **non-exertional symptoms**. Severity of symptoms, extent of myocardial damage and possible treatment options are largely depending on factors like distribution of atherosclerosis in the coronary system, location and extent of disease, presence of alternate pathways or collateral circulation and the timeframe for development of occlusion (acute versus chronic).

Myocardial oxygen deprivation will produce decreased pH, impaired cell membrane function etc. at the cellular level. If the *ischemia continues for more than 20 min*, *cell death and subsequent scarring of cells (infarction) happens* in the absence of collateral circulation. Ischemia may cause repolarization abnormalities leading to elevation of ST segment, premature beats including ventricular tachycardia and fibrillation.

It has been noted that atherosclerotic changes begin even before the age of 20 and the patients are asymptomatic for a long duration in the life. Ischemic changes also can damage left ventricular myocardium, resulting in heart failure due to **ischemic cardiomyopathy**.

Coronary circulation and 12 lead EKG		
I, aVL V5, V6	lateral wall	Circumflex artery
II, III, aVF	inferior wall	Right coronary artery
V1, V2	septum	Left anterior descending
V3,V4	anterior wall	Left anterior descending

Box 12.1 EKG leads corresponding to anatomical areas of heart

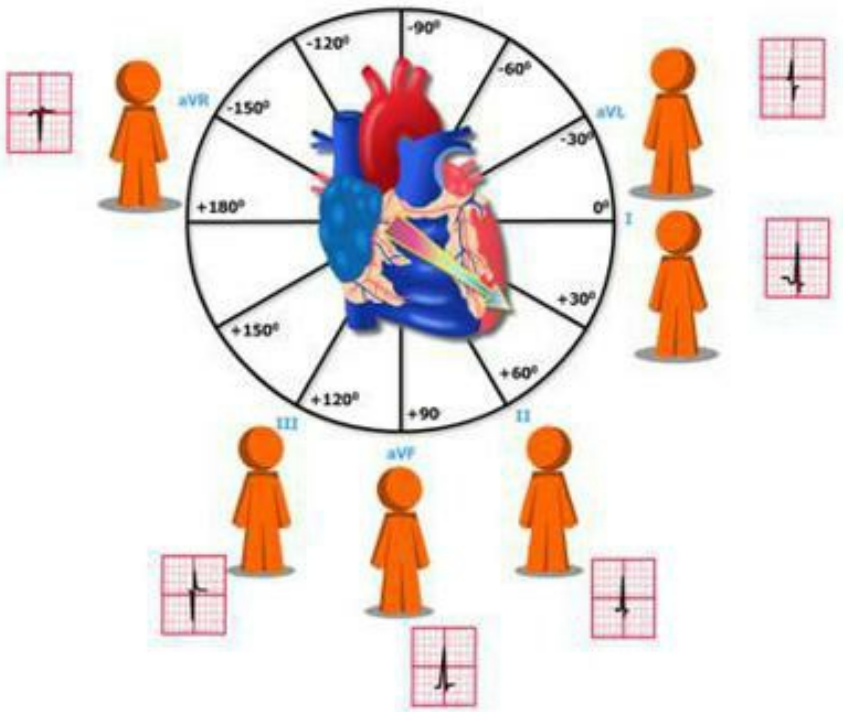
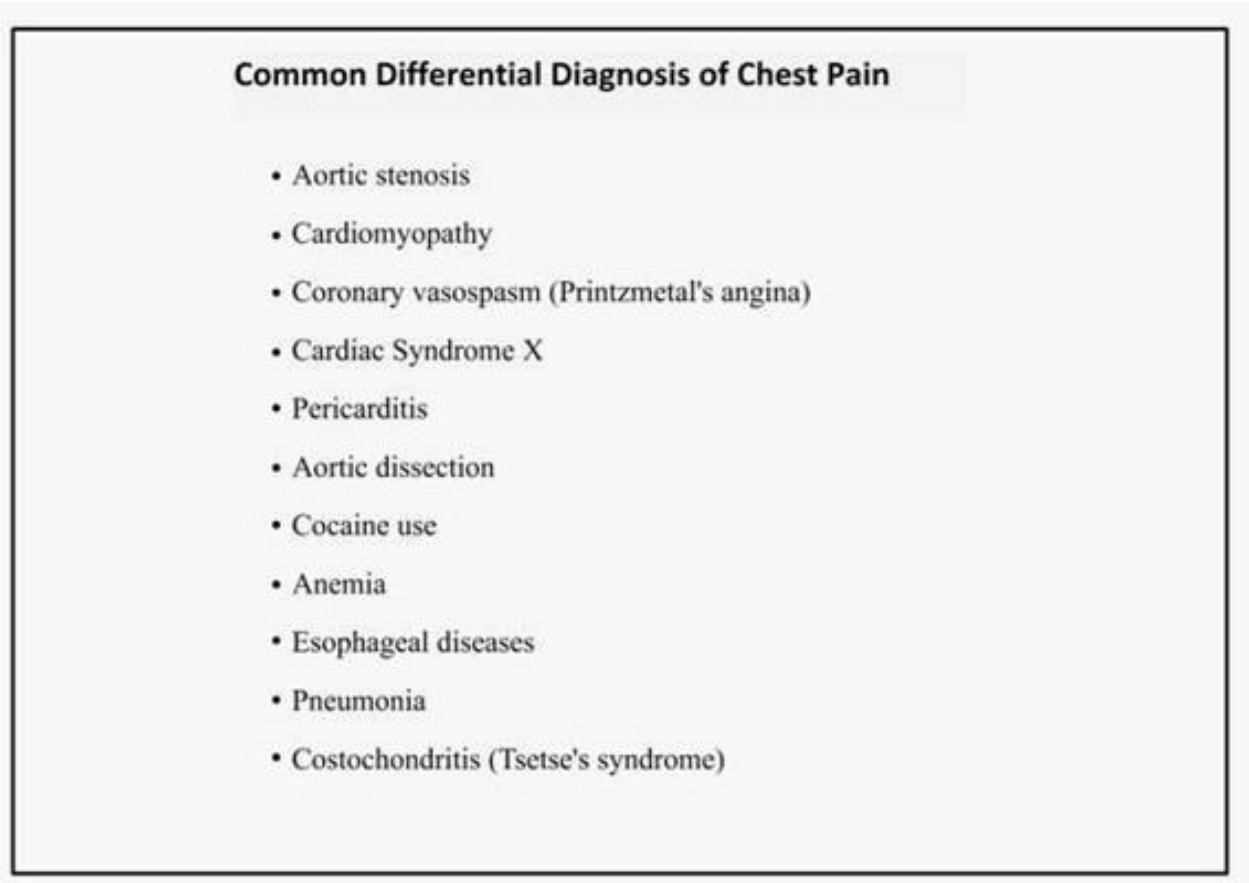


Fig 12.5 12 lead EKG in relation to views of the heart



Box 12.2 Major differentials for chest pain

Diagnostic Testing for Chest Pain

EKG: 12 lead EKG may be normal or with presence of conduction deficits, ST depression and T wave inversions, left ventricular hypertrophy changes etc., unless patient is having a true STEMI.

Exercise stress test: Either physical or chemical stress testing brings out **stress induced ischemic changes** that are usually *reversible in nature*. Typical *positive ST segment response* is indicated by a *flat or down sloping depression of ST segment* for more than **0.1 mV** below baseline. The study also considered positive in events like hypotensive response to exercise and sustained ventricular arrhythmia induced by stress.

Fig 12.6 Exercise treadmill testing

Chemical stress testing with the use of **Dipyridamole**, **Adenosine** and **Regadenosine** (**Lexiscan**) that are vasodilators, combined with nuclear imaging is particularly useful in patients with the left bundle branch block and pacemaker rhythms where EKG is not a reliable indicator of ischemia.

EKG Changes in Myocardial Infarction

EKG changes in myocardial ischemia

- **MI in progress:** Elevation of ST segment in leads corresponding to respective coronary artery territory. For example, during left anterior descending artery (LAD) occlusion, ST elevation is more pronounced in septal anterior leads, i.e. **V1- V4.**
- **Myocardial ischemia:** Depression of ST segment with T wave inversion in the ischemic territory.
- **Reciprocal changes:** In an acute myocardial infarction, the area opposite of infarction shows reciprocal changes in the ST segment. For example, in anterior wall myocardial infarction ST segment elevation is seen in lead **V1- V4**; however, inferior leads (lead **II, III** and a**VF**) show ST segment depression.

Box 12.3 Characteristics of EKG in ischemia

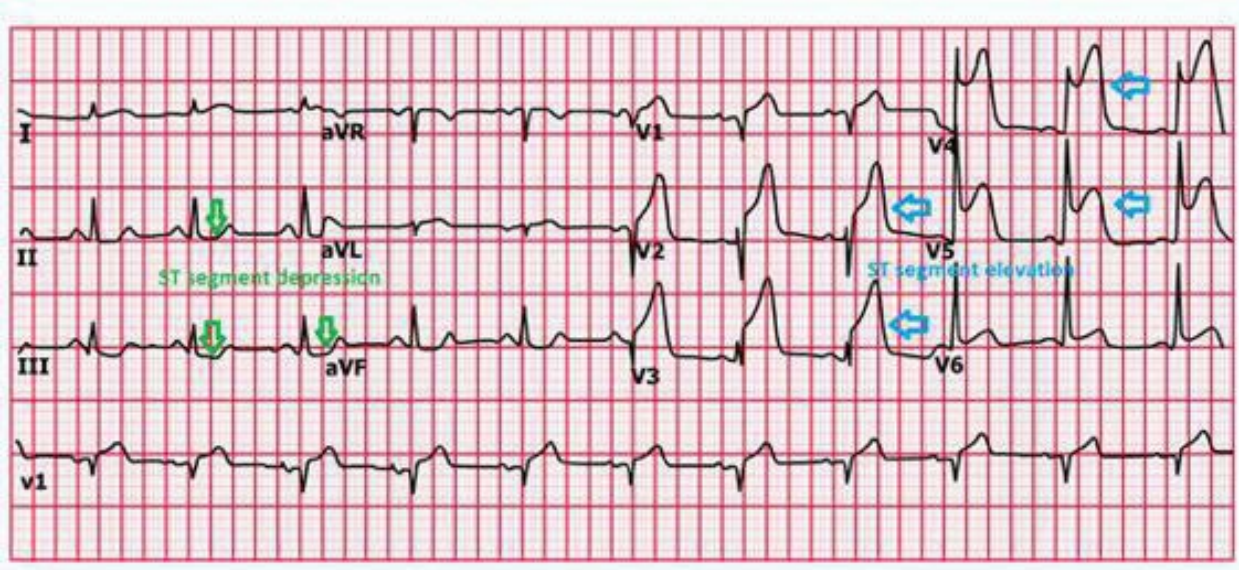


Fig 12.7 Anterior wall MI with its characteristic EKG changes (Note the presence of ST segment elevation in the anterior leads and ST segment depression in the inferior leads).

Prominent Q wave formation is an indication of old myocardial infarction. They are considered significant only when they are wide (0.04 sec) and larger (greater than 1/3 of R wave). Formation of Q wave takes more than 24 hours of myocardial ischemia. Therefore, presence of Q wave in an EKG indicates that MI has happened more than 24 hours before. This is a major determinant in selection of treatment options for individual patients.

Events associated with myocardial infarction

Anterior MI: This is the most lethal type of myocardial infarction due to the involvement of left ventricle and septum. If not intervene in a timely fashion this pathologic process can markedly reduce cardiac output leading to dreadful consequences.

Inferior MI: This produces brady arrhythmias and possible heart block. Since the right coronary artery is the culprit vessel (artery that is occluded) and it supplies the right ventricle, patient may present with symptoms of fluid overload.

Posterior MI: This is the most difficult type of myocardial infarction in terms of diagnosis. A regular surface EKG with precordial leads doesn't always show presence of a posterior MI. Instead of normal QS wave pattern, presence of large R waves in V1 and V2 along with ST depression in antero-septal leads (V1- V4) represent posterior MI (mirror image). It usually originates from a dominant posterior descending artery (PDA) or Circumflex coronary artery (LCx) that supplies posterior wall. If taken Lead V7-V9 will show ST elevation.

Box 12.4 Clinical consequences of MI

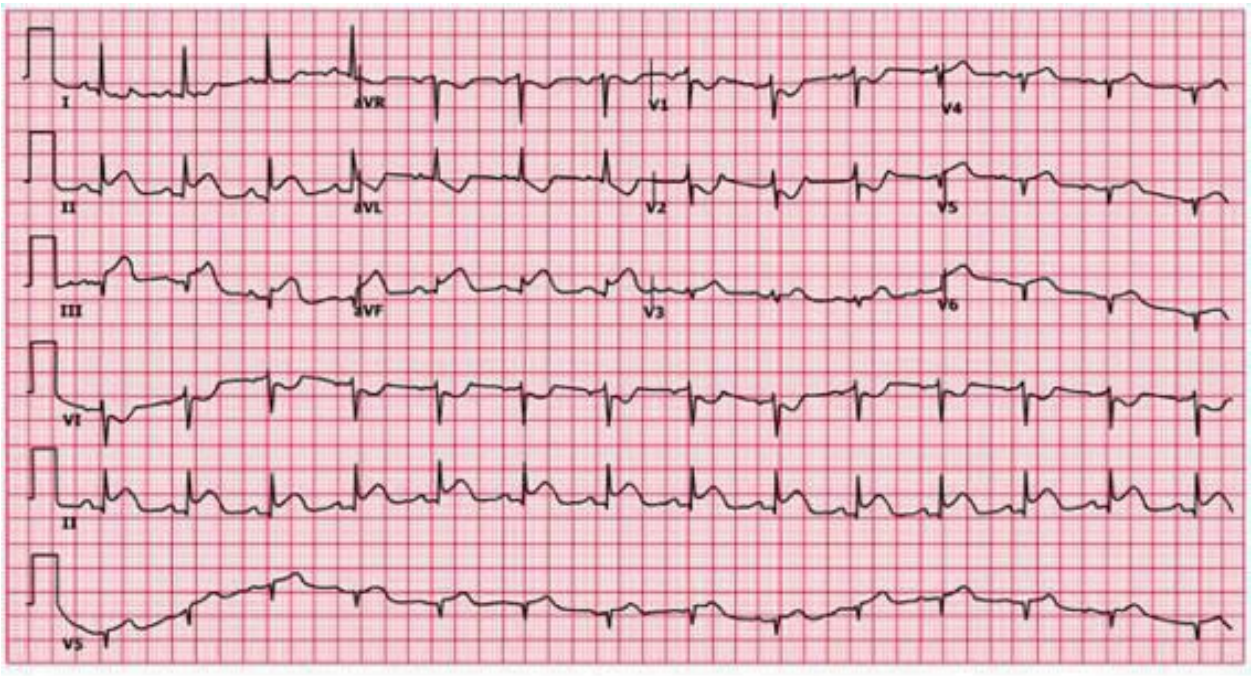


Fig 12.8 EKG findings in Inferior wall myocardial infarction. (Note the presence of ST segment elevation in the inferior leads (II, II and aVF) and mirror image changes in anterior leads (V1, V2)

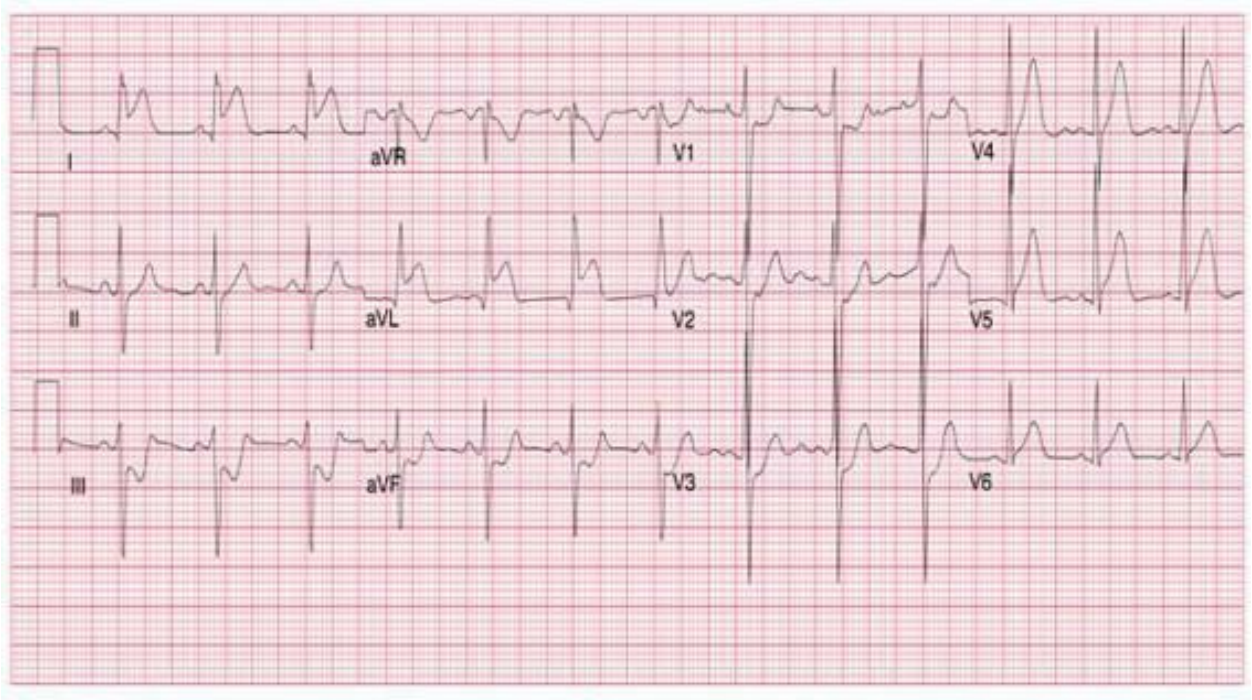


Fig 12.9 EKG in Lateral wall myocardial infarction. (Note the characteristic ST segment elevation in lateral leads (I, aVL) and mirror changes in the inferior leads (III and aVF).

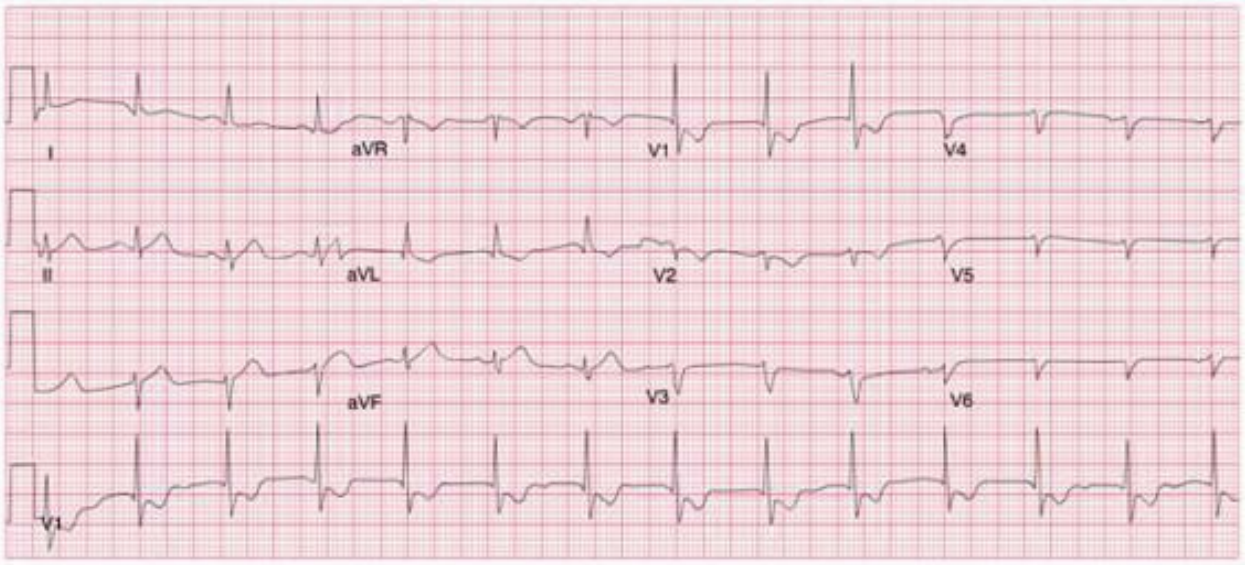


Fig 12.10 EKG tracing from Posterior wall myocardial infarction. (Note the presence of large R waves in Lead V1with ST segment depression in V1 and V2).

Treatment in Myocardial Infarction

Major goals of treatment in myocardial infarction are

1. Management of acute phase

   Anti-ischemic treatment with oxygen, nitroglycerin, morphine, aspirin, beta-blockers etc. and mechanical revascularization using Percutaneous transluminal coronary angioplasty and stenting or chemical revascularization with tPA.

2. Preventing progression of acute phase

   Anti-thrombolytic treatment with antiplatelets like Clopidogrel, Prasugrel, Ticlopidine, Eptifibatide, Tirofiban, Abciximab and Heparin.

3. Preventing of progression of risk factors

   Risk factor reduction with statins, beta-blockers and ACE inhibitors

Box 12.5 Treatment of myocardial infarction

Neurologic T wave

Acute ischemic stroke has sometimes a characteristic EKG finding with deep inverted T waves in multiple vascular distribution areas as follows. So evaluation of patient for not only cardiac but other disorders is very important in accurate diagnosis of clinical situation.

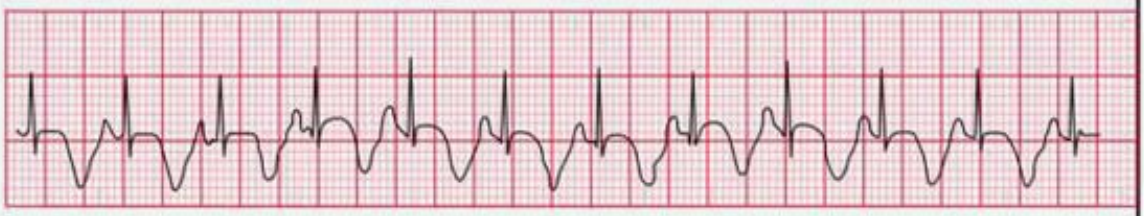


Fig 12.11 Classic deep T wave pattern in Acute stroke

Box 12.6 Neurologic T wave

Points to Remember!!!

- Atherosclerotic heart disease accounts for majority of myocardial ischemia and infarction.
- Stable angina pectoris is characterized by typical chest pain induced by physical or emotional exertion with characteristic nature and radiation, relieved by rest or sublingual nitroglycerin.
- Unstable angina has typical to atypical chest pain that may or may not respond to rest or sublingual nitroglycerin.
- Non-ST segment elevation myocardial infarction (NSTEMI) has characteristics of stable angina with elevated cardiac biomarkers and EKG changes.
- ST elevation myocardial infarction (STEMI) is caused by acute closure of a coronary artery leading to imminent myocardial injury.
- ST elevation MI has characteristic ST segment elevation in target leads depending on the artery affected.
- In anterior wall STEMI, lead V1 to V4 has ST elevations and inferior leads (lead II, III and aVF) have reciprocal changes in the form of ST segment depression.
- ST changes in isolated leads are not much of a diagnostic value.
- Major determinants of myocardial oxygen demand are heart rate, myocardial contractility and wall tension.
- Abnormal lipids, smoking, hypertension, diabetes mellitus etc. are common risk factors for development of coronary artery disease.
- In the event of myocardial ischemia lasts more than 20 min, permanent damage of

myocardium called infarction may occur.

- Profound infarction of myocardial area especially that of left ventricle causes ischemic cardiomyopathy.
- Instead of ST segment elevation, myocardial ischemia causes depression of ST segment with T wave inversion in target leads.
- Presence of Q wave indicates myocardial infarction happened more than 24 hours prior to the presentation.
- Posterior wall myocardial infarction is one of the difficult diagnosis to be made from usual anterior chest wall EKG and is characterized by presence of large R waves in V1 and V2 in a regular EKG.
- Acute management of myocardial infarction depends on the type of MI.
- For STEMI, immediate and timely revascularization, either mechanical (PTCA with stents) or chemical (tPA or other thrombolytics) is of supreme priority.
- For NSTEMI or unstable angina, non-emergent revascularization along with risk factor modification is the treatment approach.

Test Your Understanding

1. Which of the following is not a characteristic of stable angina pectoris?

A Chest pain induced by exertion
B Chest pain relieved by nitroglycerin
C Chest pain initiated without exertion
D Squeezing substernal chest pain

2. Which of the following is a differentiating factor between stable angina and non-ST segment elevation myocardial infarction?

A Typical chest pain with radiation

B Relieved by rest or sublingual nitroglycerin

C Absence of cardiac enzymes elevation

D Presence of elevated troponin or CKMB

3. Which of the following EKG finding is consistent with inferior wall myocardial infarction?

A ST depression in lead II and III

B Elevation of ST segment in lead aVL, V5 and V6

C ST segment elevation in lead II, III and aVF

D Presence of large T wave in V6

4. Which of the following is not a finding in anterior wall ST elevation MI?

A ST segment elevation in lead V3 and V4

B ST segment depression in lead II and aVF

C ST segment elevation in lead II and III

D T wave inversion in lead III and aVF

5. Coronary atherosclerosis risk factors include all of the following except_____?

A Increased HDL cholesterol

B Increased LDL cholesterol

C Smoking

D Diabetes mellitus

6. Which of the following finding is not seen in lateral wall myocardial infarction?

A ST elevation in III and aVF

B ST elevation in aVL and V5

C ST elevation in V5 and V6

D ST elevation in lead I, V5 and V6

7. One of the serious complications of anterior wall MI is _____?

A Right ventricular hypertrophy

B Left ventricular dysfunction

C Right atrial dysfunction

D Bradycardia

8. Which of the following is not a treatment option for non-ST segment elevation myocardial infarction?

A PTCA and stent

B Use of tPA

C Use of aspirin and nitroglycerin

D Use of beta-blockers

Answers

1. C Chest pain initiated without exertion
2. D Presence of elevated troponin or CKMB
3. C ST segment elevation in lead II, III and aVF
4. C ST segment elevation in lead II and III
5. A Increased HDL cholesterol
6. A ST elevation in III and aVF
7. B Left ventricular dysfunction
8. B Use of tPA

Chapter 13

General Diagnostic Value of EKG

In this chapter

- Right atrial overload
- Left atrial overload
- Right ventricular hypertrophy
- Cor-pulmonale due to COPD
- Left ventricular hypertrophy
- Pulmonary embolus

Structural changes in chambers of the heart can create substantial electrocardiographic evidence because it changes the duration and force of electrical activity within the myocardium. Looking at specific leads that are designated for monitoring individual areas of the heart, a 12 lead EKG may provide initial clues for underlying pathophysiologic process. Mostly, enlargement or thickening of chambers produce *wide or tall waveforms* in corresponding leads.

Left Atrial Enlargement

In a normal heart, the sequence of atrial activation starts from right atrium (since SA node is situated here), which then followed by the left atrium because of the minute travelling delay for impulse from right to left chamber. This delayed activation of the left atrium leads to a *slight notching of P wave*. Hypertrophy or scarring of the atrial wall resulting from left atrial enlargement increases this **Inter atrial delay**. Similar to any other conduction delay, the impulses from right atrium takes longer to depolarize the enlarged left atrium, leading to **wide P wave** (>0.12 sec) with a **pronounced notching**. This increase in duration of P wave is known as classic ***'P mitrale'*** sign. These changes are evident in the inferior leads such as lead **III** and **aVF**. In the anterior precordial lead **V1**; left atrial enlargement may produce a **deeply inverted P wave**.

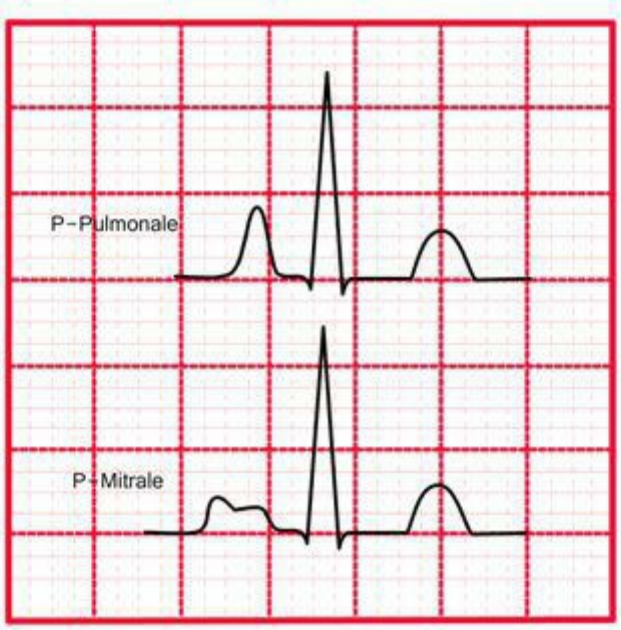


Fig 13.1- Wide and notched P wave in LA enlargement

Right Atrial Hypertrophy (RAH)

Similar to the mechanism seen in the left atrial enlargement, scarring and hypertrophy of right atrium causes *delay in depolarization of right atrium*. Therefore, both atria will depolarize *simultaneously rather than sequentially* creating a *narrow and tall P wave* called '***P Pulmonale***'. This change is pronounced in lead **II** and **V1**. In severe right atrial enlargement, the right atrium may become so large that it extent towards the left atrium creating an inverted P wave in lead **V1** mimicking EKG change of left atrial enlargement.

EKG criteria for atrial enlargement

Right atrial hypertrophy

- Narrow and tall P wave (***P Pulmonale***) in Lead **II** and **V1**

Left atrial hypertrophy

- Wide and notched **P** wave (>0.12 Sec) in lead **III** and **aVF** (***P Mitrale***)
- Deep inverted **P** wave in **V1**

Box 13.1 EKG criteria for atrial hypertrophy

Right Ventricular Hypertrophy (RVH)

Hypertrophy of right ventricle is seen in clinical conditions such as *advanced COPD*, *cor pulmonale*, *tricuspid* or *pulmonic stenosis* and as a sequel of *mitral stenosis (due to back flow of blood from left atrium through pulmonary circulation)* . Thickening of the ventricular wall occurs in order to compensate for elevated right ventricular pressure caused by any of these underlying disease conditions. This increased muscle mass causes *tall R waves* in *anterior precordial leads* (*lead **V1** and **V2***) and *deep S waves* in *lead **V5** and **V6***.

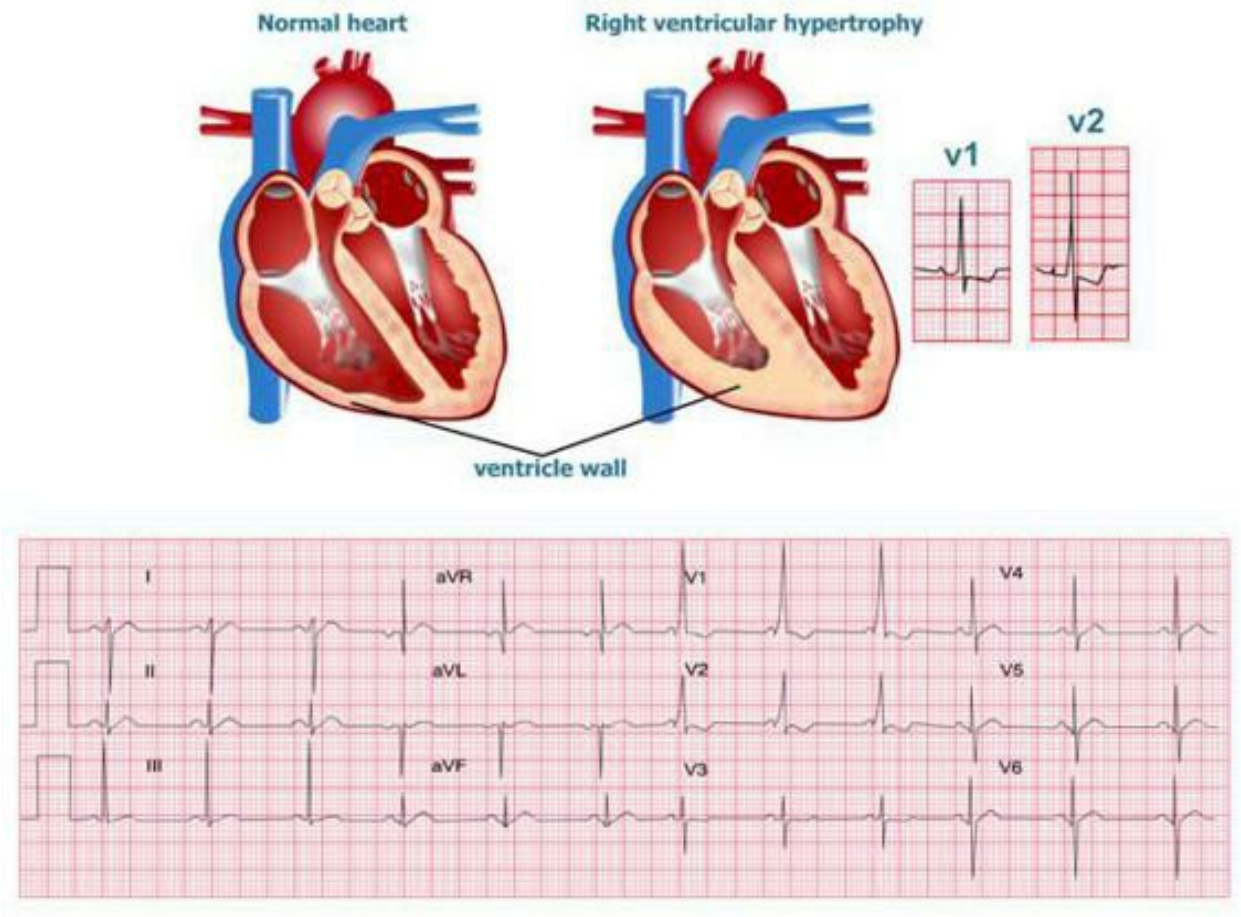
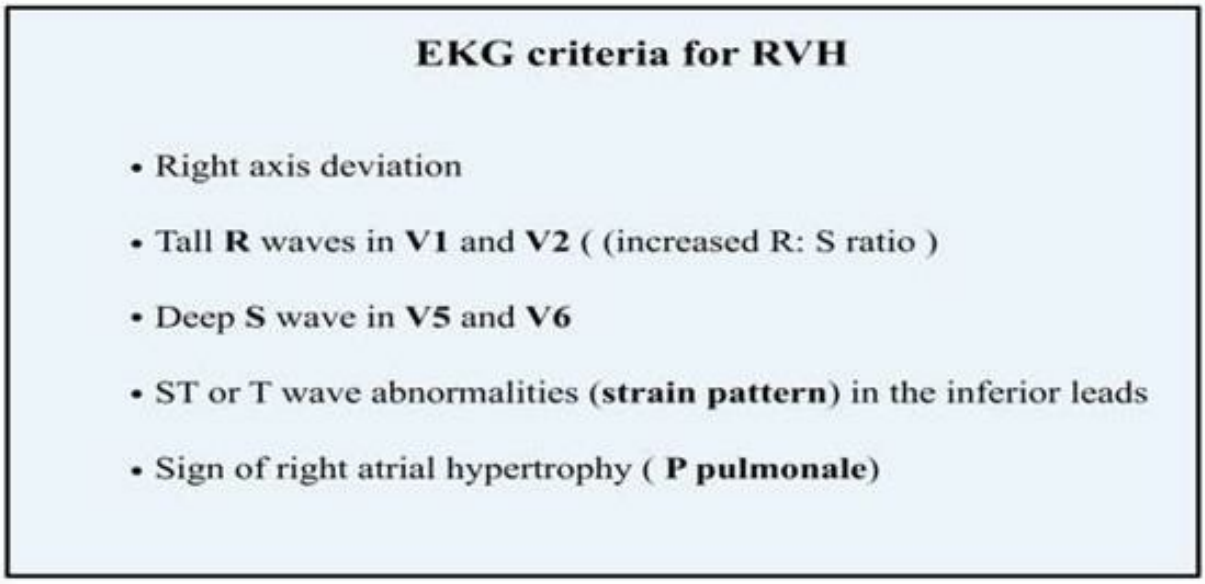


Fig 13.2 Tall R wave in VI and V2- a sign of right ventricular hypertrophy

Unlike normal 12 lead EKG, RVH causes *tall R waves and small S waves* in *lead V1* and *V2* creating an **R: S ratio >1**. However, before jumping into the conclusion of relatively less acute right ventricular hypertrophy, we have to exclude other more serious causes of increased R: S ratio such as *posterior wall myocardial infarction*, *WPW syndrome*, *hypertrophic cardiomyopathy* etc. In some patients, this reversal of R: S ratio can be a normal variant without any underlying pathology. Excessive thickening of ventricular wall may cause **sub endocardial ischemia** in the right ventricular wall, leading to ST segment and T wave changes in the right-sided precordial leads. Right ventricular hypertrophy is also associated

with *right axis deviation*.



Box 13.2 EKG criteria for Right Ventricular hypertrophy (RVH)

Left Ventricular Hypertrophy (LVH)

Being the main pumping chamber of the heart, left ventricle has to increase its effort in any event that causes obstruction of blood flow into the systemic circulation. Common causes that increase workload of the left ventricle are *aortic stenosis*, *hypertrophic cardiomyopathy*, *long-standing elevated blood pressure* etc. The constant strain imparted on the left ventricle results in *increasing muscle mass* and *subsequent thickening* of the ventricular wall in order to assist in effective contraction. The thickened ventricular wall creates more resistance for electrical impulse to travel leading to *slightly wide QRS complexes*.

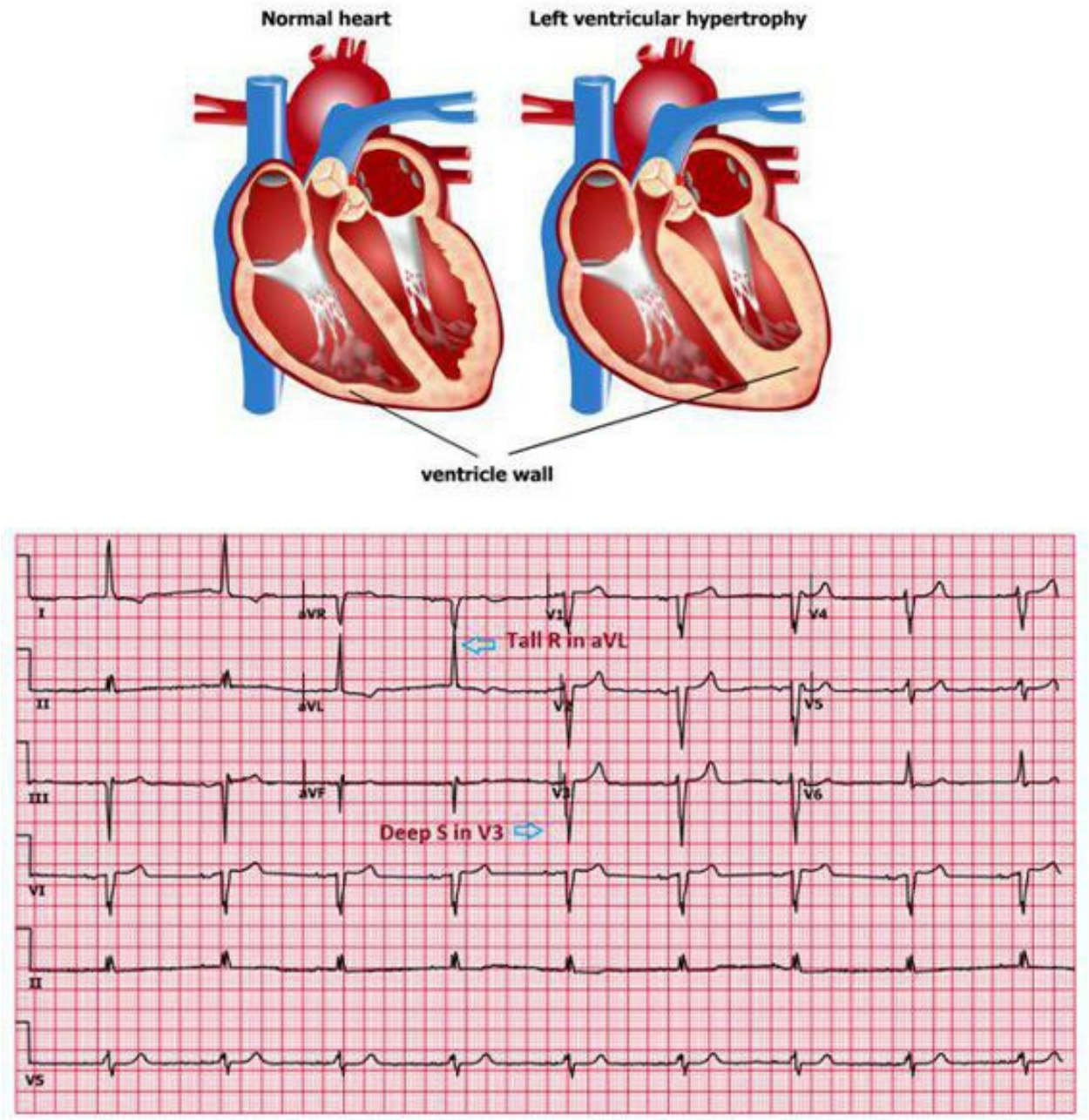


Fig 13.3 left ventricular hypertrophy (Note the presence of tall R waves in Lead III and deep S waves in V3 suggesting LVH)

At the same time, increased muscle mass causes *elevated amplitude* of resulting waveform. There may be *left axis deviation* because of the increased muscle mass in the left ventricle. Similar to the right ventricular hypertrophy, sub endocardial ischemic changes create **down sloping ST segment** and **inverted T wave** (***strain pattern***) in left lateral leads (lead **I**, **aVL**, **V5** and **V6**).

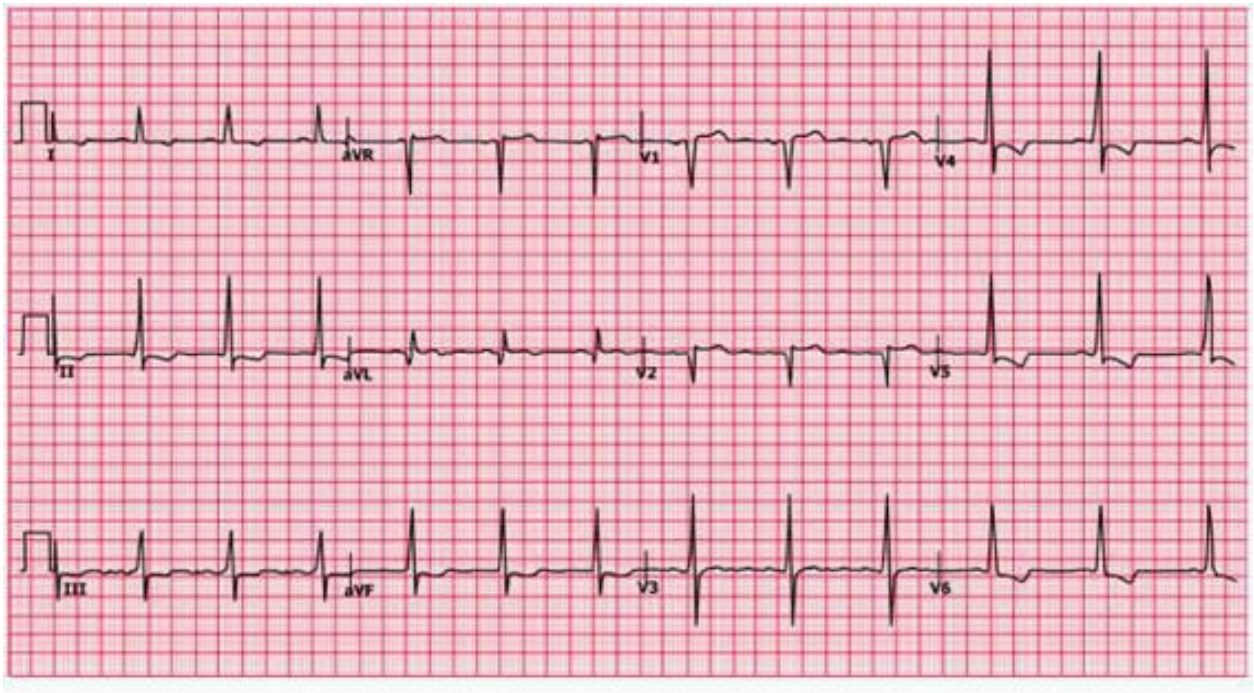


Fig 13.4 LVH (Note the presence of tall precordial R waves with strain pattern. Even though this EKG doesn't meet above-mentioned criteria for LVH, this patient had echocardiogram findings confirming left ventricular hypertrophy).

There are multiple EKG criteria used to diagnose left ventricular hypertrophy from a 12 lead EKG. Most common and reliable criteria are shown in the table.

EKG Criteria for LVH

Sokolow and Lyon Index

Amplitudes of S wave in lead **V1** and **R** wave in lead **V5** or V6 greater than or equal to 35 mm.

SV1 + R V5/ V6 > = 35 mm
R in aVL > = 11 mmv

Cornell criteria

Amplitude of **R** wave in **aVL** and **S** wave in **V3** greater than 28 mm in men or greater than 20 mm in women.

R aVL + S V3 > 28 - men
> 20 - women

Box 13.3 EKG criteria for LVH

Bi-ventricular Hypertrophy

In patients with advanced stages of underlying diseases, coexistence of both right and left ventricular hypertrophy is not uncommon. These patients may have EKG satisfying voltage criteria for *LVH in the precordial leads* along with *right axis deviation in limb leads* or *tall R waves in precordial V1 and V2*. Some of them also have coexisting left atrial enlargement with the characteristic wide P wave as discussed earlier. Biventricular hypertrophy is also seen in children with ventricular septal defect. These children have EKG showing tall biphasic R waves in precordial leads known as **Katz- Wachtel phenomena** as shown in the picture.

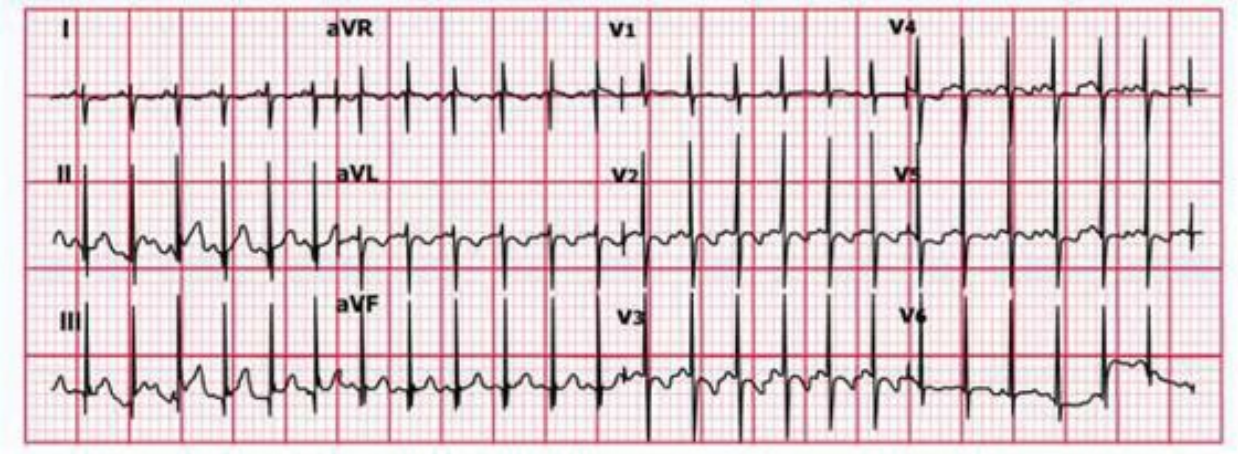


Fig 13.5 Biventricular hypertrophy (Note the presence of tall precordial biphasic R waves with right axis deviation (Negative Lead I and positive aVF) and prominent R waves in V1).

Pulmonary Embolism

Even though not universally present, many patients with massive acute pulmonary embolism shows characteristic EKG changes such as a *prominent **S** wave in lead **I***, *presence of **Q** wave* and *inverted **T** wave in lead **III*** (**S1Q3T3 pattern**), *ST segment and T wave changes in anterior precordial leads* (right ventricular strain pattern), new *incomplete right bundle branch block* and *sinus tachycardia*.

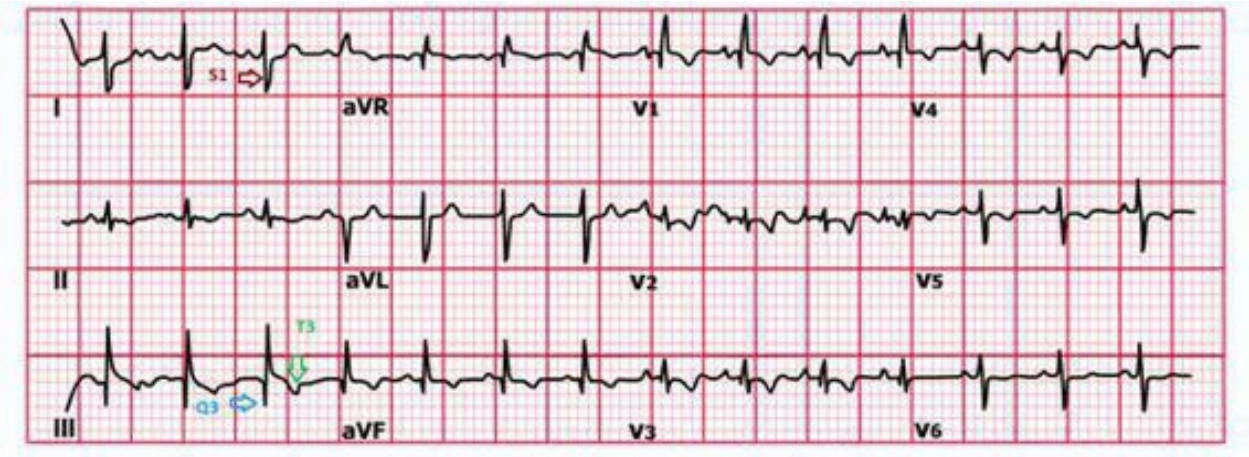


Fig 13.6 Pulmonary embolism (Even though the patient doesn't have tachycardia, this EKG has prominent S in lead I, Q and inverted T waves in lead III along with incomplete RBBB and inverted T waves in anterior precordial leads (V1-V3) suggesting right ventricular strain pattern.

Pericarditis

Pericarditis and acute pericardial effusion can create characteristic EKG changes that mimic signs of acute STEMI due to the inflammation of epicardium. There may also be typical *PR segment depression*. Here, both ST and PR segments deviate in opposite direction. In pericarditis, due to more generalized inflammation and resulting tissue injury compared to focused STEMI, *ST segment elevation may be wide spread in varying degrees in most of the leads* than focused leads.

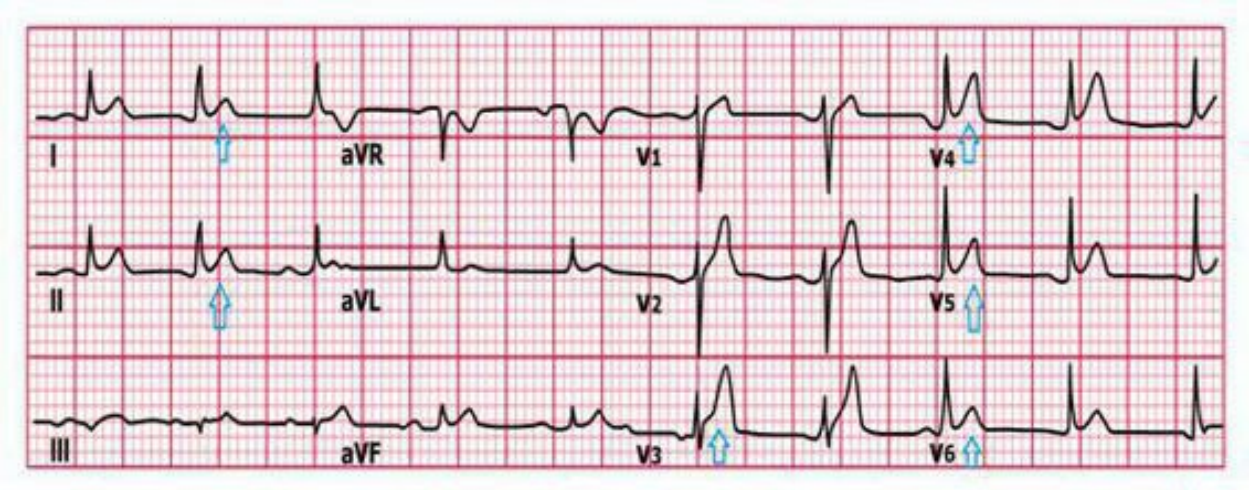


Fig 13.7 Acute pericarditis (Note the presence of diffuse ST segment elevation in inferior, lateral and anterior leads denoting possible wide spread inflammation).

Pericardial Effusion or Cardiac Tamponade

Pericardial effusion or cardiac tamponade can occur resulting from acute or chronic collection of fluid within the pericardium. Acute pericarditis can be a reason for pericardial effusion or cardiac tamponade. In this situation, because of the presence of fluid outside the heart, surface EKG will show *low amplitude or low voltage complexes*. These could also be presence of *alteration in amplitude of QRS in adjacent beats* called **electrical alternance** as shown in Fig 13.8.

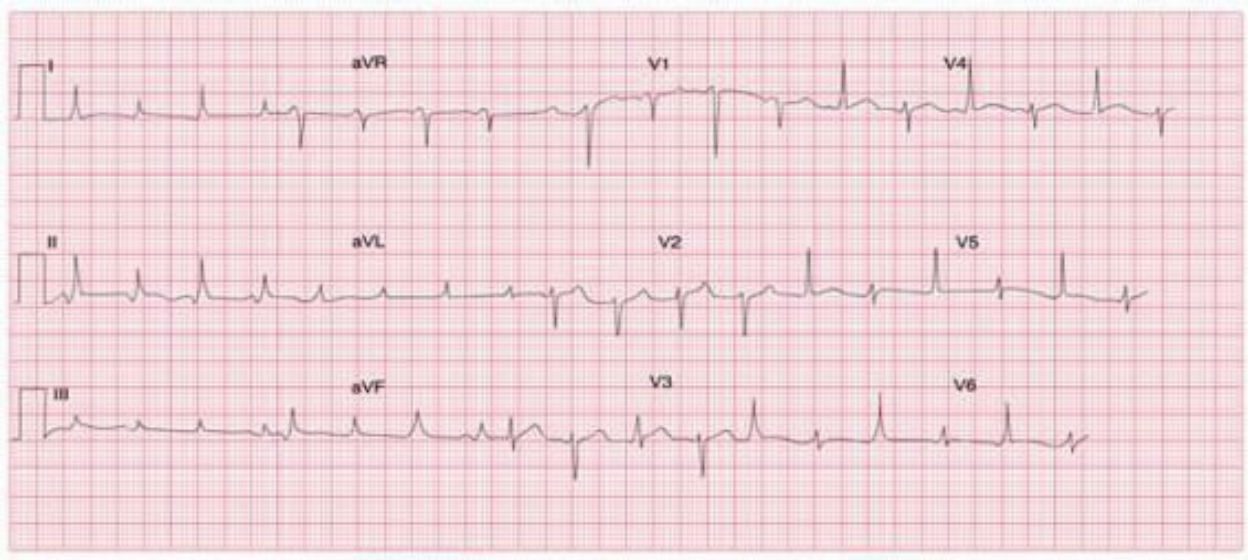


Fig 13.8 Electrical alternance (Note the presence of varying amplitude of QRS from beat to beat).

EKG Findings in Electrolyte Imbalance

Since electrolytes such as Potassium, Calcium and Magnesium play a vital role in various electrical and contractile functions of the heart, any change in the availability of these elements can produce characteristic changes in the resulting EKG as shown in the Box 13.3.

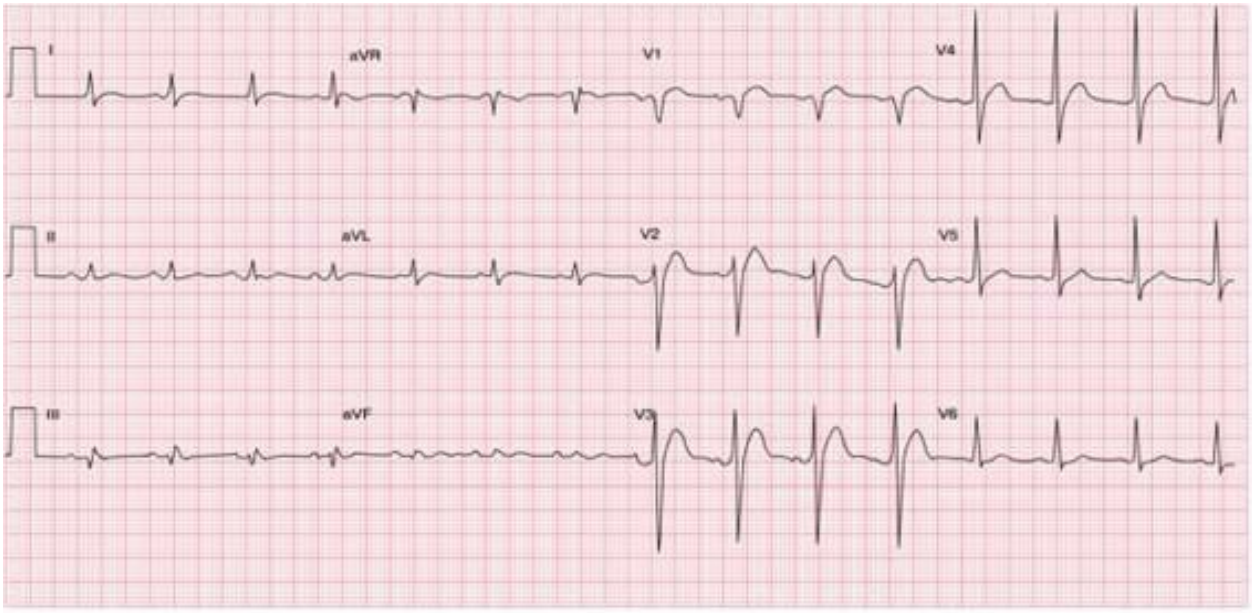


Fig 13.9 EKG changes in hypercalcemia

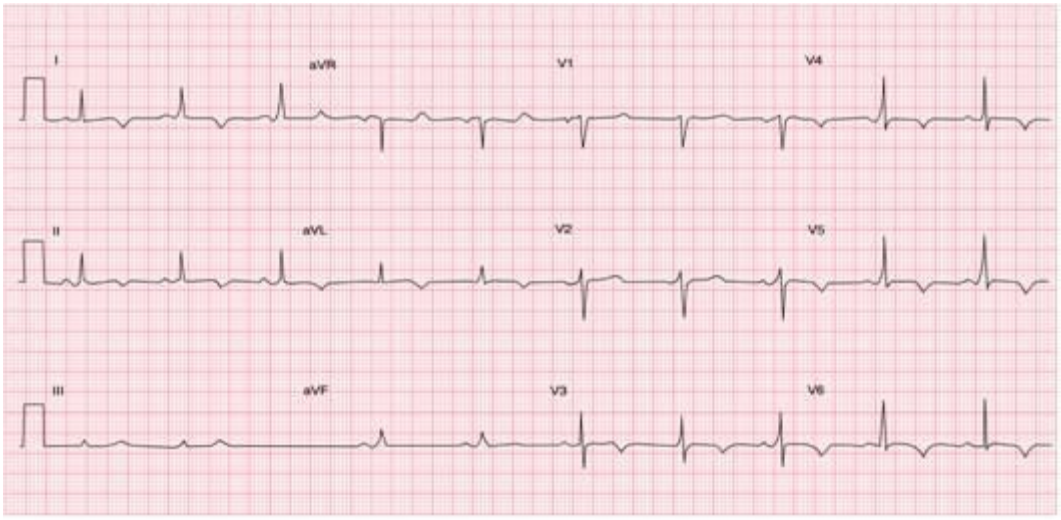


Fig 13.10 Hypocalcemia (Note the presence of long ST segment without increase in duration of T wave).

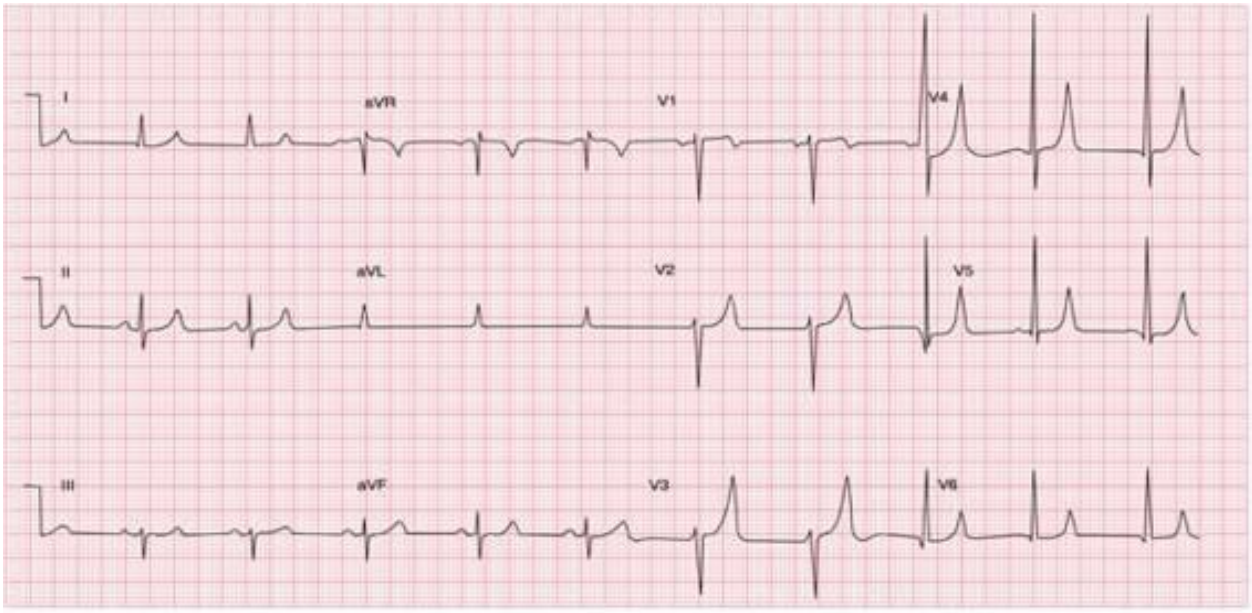


Fig 13.11 Hyperkalemia (Note the presence of tall peaked T waves with narrow base in the precordial leads).

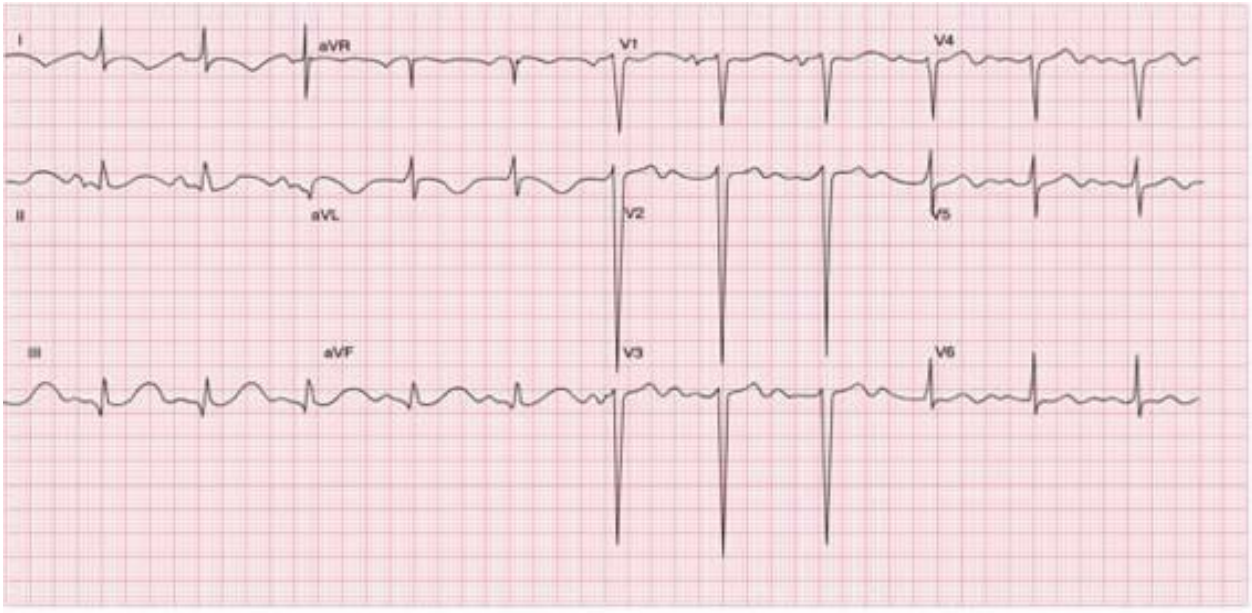


Fig 13.12 Hypokalemia (Note the presence of dominant U waves in precordial leads).

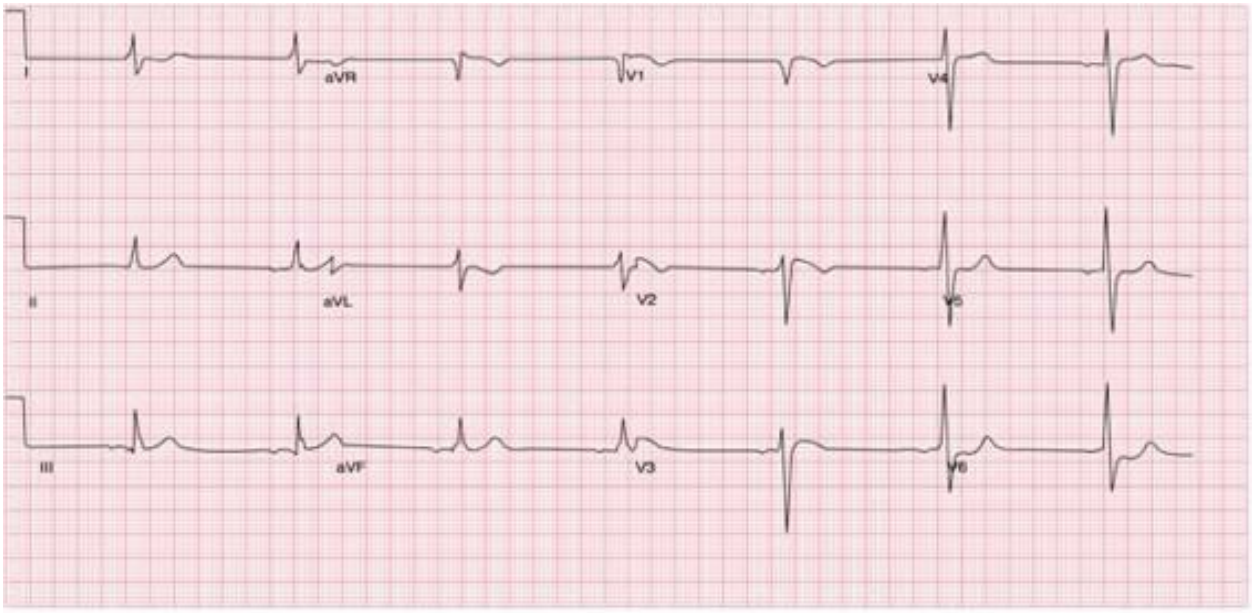


Fig 13.13 Hypernatremia (Note the presence of 'Brugada like' pattern).

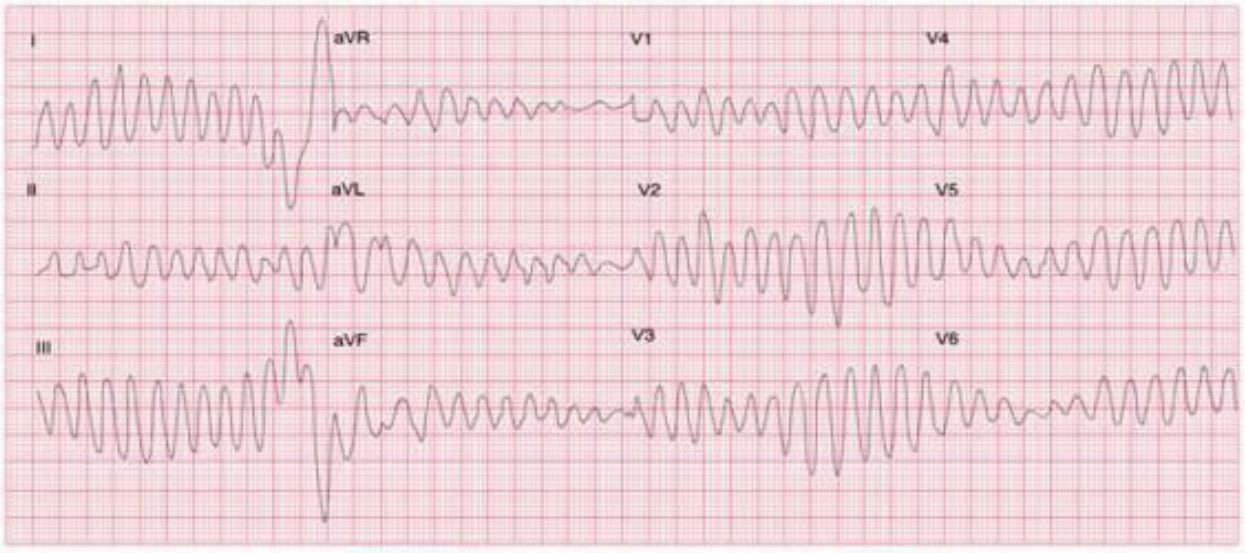


Fig 13.14 Hypomagnesemia showing as polymorphic VT (Torsades).

	Hypo-	Hyper-
Calcium	• Prolonged QTc • Flat or inverted T wave • Prolonged ST Segment without increased duration of T wave	• Short QTc, PR segment prolongation
Potasium	• Tall U waves • Small T waves • Large P waves • ST depression	**5.5- 7.5 mEq/L** • Tall peaked T waves • reversible LAFB or LPFB **7.5 - 10.0 mEq/L** • First degree AV block • Flat/ Wide/Absent P wave • ST segment depression **>10.0 mEq/L** • LBBB, RBBB or IVCD • V tach / fib , Idioventricular rhythm
Sodium	• 'Brugada' like appearance (ST elevation in V1-V3 with RBBB)	
Magnesium	• Peak T waves • Prominent U waves • Prolong QRS • ST depression • Ventricular arrhythmia including Torsades	• Prolonged PR interval • Increase QRS duration • Increase in QT interval • complete heart block/ cardiac arrest if Mg >15 mEq/L

Box 13.4 Effect of electrolyte imbalance in EKG

Points to Remember!!!

· Left atrial enlargement produces slightly notched P wave that is more pronounced in lead III and aVF, called p mitrale sign.
· Instead of the notched P wave as in the left atrial enlargement, right atrial hypertrophy EKG has a narrow and tall P wave called P Pulmonale.
· Because of the increased muscle mass in the right ventricle during right ventricular hypertrophy, there are tall R waves in lead V1 and V2 and deep S waves in V5 and V6.
· Right ventricular hypertrophy should be differentiated from that of posterior wall of MI, WPW syndrome, hypertrophic cardiomyopathy etc.
· Right ventricular hypertrophy also causes right axis deviation.
· Left ventricular hypertrophy is caused by aortic stenosis, hypertrophic cardiomyopathy, long-standing elevated blood pressure etc.
· Because of the increased muscle mass, tall R waves are seen in the lateral and precordial leads.
· Along with tall R waves, down sloping ST segment and inverted T waves (strain pattern)

are also seen in LVH.

- Most reliable and widely accepted criteria for left ventricular hypertrophy is Cornell criteria where R aVL + S V3 > 28 mm in men/> 20 mm in women.
- Pulmonary embolism generates prominent S wave in lead I, presence of Q and inverted T wave in lead III (S1 Q3 T3).
- Pericarditis produces EKG similar to that of a STEMI; however, with varying degrees of generalized ST elevation.

Test Your Understanding

1. 12 lead EKG of a patient with left atrial enlargement involve_____?

A Tall and narrow R waves

B Tall and wide R waves

C Tall and narrow P wave

D Notched P wave

2. Which of the following is a characteristic EKG finding in right ventricular hypertrophy?

A Deep S waves in lead V1 and V2

B Deep S waves in V5 and V6

C Tall R wave in V5 and V6

D Inverted P wave in lead I

3. Which of the following is one of the possible differential diagnoses for an EKG with R waves in lead V1 and V2?

A Right atrial enlargement

B Left atrial enlargement

C Posterior wall MI

D Left ventricular hypertrophy

4. Which of the following is a possible causative factor for left ventricular hypertrophy?

A Dilated cardiomyopathy

B Long-standing hypotension

C Tricuspid regurgitation

D Aortic stenosis

5. Which of the following is a characteristic EKG finding in acute pulmonary embolism?

A Sinus bradycardia

B Ventricular fibrillation

C Prominent S in lead I with presence of Q and T inversion in lead III

D Left bundle branch block

Answers

1. D Notched P wave
2. B Deep S waves in V5 and V6
3. C Posterior wall MI
4. D Aortic stenosis
5. C Prominent S in lead I with presence of Q and T inversion in lead III

Chapter 14

Cardiac Pharmacology

From the information what we discussed in earlier chapters, it is evident that contractile function of myocardium is a complex process involving electrical, chemical and mechanical components at various levels. To the very basis of this process resides movement of ions such as calcium, sodium and potassium. Even though there are other trace elements

involved in the overall process, these ions are of particular importance because of the way many of our cardiac medications work in the system.

Antiarrhythmic Drugs

These medications are used for management of heart rhythm disorders and are classified into four different groups based on their pharmacodynamics. The most common and widely accepted classification is known as **Vaughan Williams classification**. Here, antiarrhythmic agents are grouped based on their action at various levels of cardiac action potential curve. All of these agents essentially regulate flow of ions to and from the myocytes and thereby manipulate either normal or abnormal conduction pathways.

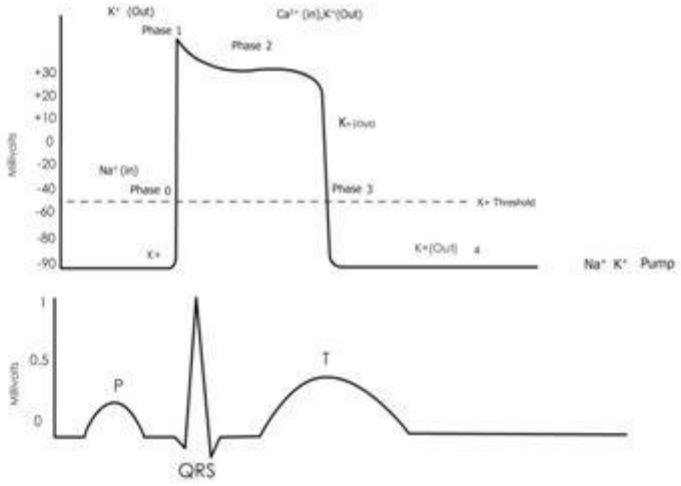


Fig 14.1 Cardiac action potential curve

Based on the electrical ions they regulate, antiarrhythmic drugs are classified largely into four groups. Class **I** agents are **sodium channel blockers**, **beta-blockers** in class **II**, **potassium channel blockers** in class **III** and **calcium channel blockers** in class IV. Adenosine and digoxin are classified as class **V** in the system. Even though there are other classifications available that are more complex, Vaughan Williams classification provides a bird's eye view on antiarrhythmic drugs and provide easier understanding based on its relation with *cardiac action potential curve*. Even though one particular drug is categorized in a category, their effects may overlap each other. In general, *sodium channel blockers slows down the conduction velocity whereas, potassium channel blockers decrease excitability*.

Class I Antiarrhythmics

These agents are otherwise called **sodium channel blockers** that control *movement of*

sodium ions in phase 0 of cardiac action potential curve. By modulating sodium ions at this stage, these drugs can essentially influence the onset of cardiac action potential. Depending on the ease of binding and dissociation with the receptor sites, they are further classified into class **1C** (having *slowest rate of binding and dissociation*), class **1A** (with *intermediate binding and dissociation*) and class **1B** (has *rapid binding and release*).

Because of the fact that these agents vary in their rate of receptor attachment, it is of great use in managing both faster and slower rhythms. For example, class 1C drugs (**Flecainide** and **Propafenone**) when used during faster heart rate takes *more time to dissociate from the receptor sites and make less number of receptors available for active contractile function*. This effect essentially lowers the heart rate because of the lack of resources to continue rapid action potential cycles. This property is called **use dependent channel block**. They primarily block sodium channels open for business and thereby slow down conduction. However, these agents may produce pro arrhythmic activity in the myocardium with underlying injury. Therefore, these agents are *not indicated* in patients with *structural heart disease*. The efficacy of sodium channel blocking property is *highest for class 1C drugs, followed by class 1A and class 1B*.

Classification of Antiarrhythmic Drugs

Class I	Sodium channel blockers	
	Class 1A	Procainamide, Quinidine and Disopyramide
	Class 1B	Lidocaine and Mexiletine
	Class 1C	Flecainide and Propafenone
Class II	Beta-blockers	Carvedilol, Metoprolol
Class III	Potassium channel blockers	Amiodarone, Sotalol, Dronedarone, Ibutilide, Dofetilide
Class IV	Calcium channel blockers	Verapamil, Diltiazem
Class V	Digoxin, Adenosine, and Magnesium sulphate	

Box 14.1 Classification of antiarrhythmic drugs

Class 1A

Sodium channel blocking properties of these agents are *intermediate* to that of other classes of class I antiarrhythmic drugs. **Quinidine**, **Procainamide** and **Disopyramide** are prime examples of this group. Among these, Quinidine has many drug-to-drug interactions especially when using with Verapamil, Phenytoin, digoxin etc. Pro-arrhythmic effect of these medications can cause formation of **Torsades De Pointes** especially in patients with prolonged base line QT interval and electrolyte abnormalities. If the baseline *QTC is greater than 500 ms*, use of antiarrhythmics should be *cautioned*. Therefore in susceptible patients, initiation of these drugs requires *in hospital monitoring*. Quinidine is used for treatment of ventricular arrhythmias and Procainamide for atrial fibrillation.

Class 1B

Lidocaine and **Mexiletine** are classified into class **1 B** antiarrhythmics. They are particularly *useful in treating ventricular arrhythmias* in the setting of myocardial infarction because of their efficacy in *fast heart rate and in abnormal myocardium*. Lidocaine has extensive **first pass metabolism** in the liver that essentially reduces availability of drug when taken orally and therefore needs to be administered intravenously. Mexiletine is used as an *oral equivalent* of Lidocaine in many situations. Compared to that of Lidocaine, Mexiletine has minimal hemodynamic side effects. *Nystagmus* is a common early indication of Lidocaine toxicity.

Class 1C

Flecainide and **Propafenone** are used for treatment of *atrial fibrillation*. In patients *with structural heart disease* such as prior history of myocardium ischemia and infarction, Flecainide and Propafenone can cause harmful effects and therefore are *contraindicated*.

Class II Antiarrhythmics

These agents are otherwise known as **beta-blockers,** with their characteristic beta-adrenergic receptor blockade property. Depending on the lipid solubility, their plasma half-life differs. For example, lipid soluble beta-blockers such as Metoprolol and Propranolol metabolize through liver and have shorter half-life; however, Atenolol that has prominent renal clearance stays in the blood longer. Beta-blockers can be broadly classified in to **selective beta I receptor blockers** (cardio selective) or **non-selective beta blockers** (non-cardio selective). Since beta-2 receptors are seen in vascular, smooth muscle and myocardial cells, *non-selective beta-blockers have global effect* on these target areas. Since *beta-1 receptor blockers selectively influence the heart*, they are of great use in clinical situations such as

bronchial asthma where beta-2 receptors can cause adverse effects.

In terms of pharmacodynamics of beta-blockers, they counteract the effect of catecholamine at beta-adrenergic receptor sites. This results in *decreasing contractility* (*negative inotropic effect*) and slowing of the heart rate by *reducing automaticity* (*negative chronotropic effect*). These agents are useful in treating both ventricular and atrial arrhythmia especially during high catecholamine states like myocardial ischemia and perioperative stage. **Sotalol,** even though a beta-blocker, has more class **III** antiarrhythmic properties. Examples of beta-blockers are shown in the box.

Classification of Beta-blockers

Non-selective

Propranolol, Timolol, Labetalol, Carvedilol,

Sotalol*, Pindolol, Bucindolol

Selective

Metoprolol, Atenolol, Bisoprolol,

Esmolol, Betaxolol

Box 14.2 Classification of beta-blockers

Beta-blockers have a wide range of therapeutic usage extending from treatment of stage fright to effective management of supraventricular and ventricular arrhythmias. They are also useful in management of hypertension, congestive heart failure, angina pectoris, hypertrophic obstructive cardiomyopathy, mitral valve prolapse etc.

Class III Antiarrhythmics

This group of medications have characteristic *potassium channel blocking* properties. Exceptions are **Sotalol**, which has *both beta blocking and potassium channel blocking* properties and **Amiodarone** that has a wide range of properties extending from *class 1 to 4 effects*. These medications are particularly useful in atrial fibrillation, flutter and ventricular tachyarrhythmia. They cause *increase in duration of cardiac action potential* and therefore *prolong QT interval* in EKG.

Amiodarone is a major player among class **III** antiarrhythmic drugs and has a wide range of use in both emergent and chronic situations. When given intravenously during an *acute myocardial event*, *Amiodarone* shows more of *class I* and *IV properties* (*sodium* and *calcium channel blocking*) and thereby effectively *treats faster ventricular arrhythmias*. It is highly fat-soluble and takes many weeks before reaching steady state in the body. Because of this extensive fat solubility and distribution, Amiodarone is *not dialyzable* and it stays in body for longer duration. Amiodarone is predominantly *metabolized through liver*. Unlike many other antiarrhythmic drugs such as Sotalol, Procainamide etc., Amiodarone *does not require hospitalization* for initiation because of its low propensity for producing **polymorphic VT** compared to other agents.

There are many side effects of Amiodarone mostly from chronic use including **chronic interstitial pneumonitis**, **thyroid dysfunction**, **photosensitivity**, **peripheral neuropathy** etc. are reported. Like many other antiarrhythmics, Amiodarone also has pro-arrhythmic effect due to *Q-T prolongation*. It can also cause bradycardia especially with concomitant use of beta-blockers. Amiodarone has also shown to increase **defibrillation threshold** (energy required for successful conversion of ventricular tachyarrhythmia) in chronic use. Amiodarone has interaction with **Digoxin** and **Coumadin** and therefore requires careful *monitoring and dose adjustments*.

Sotalol, having both beta blocking and potassium channel blocking properties is widely used for management of *atrial fibrillation* especially in patients *with implantable defibrillator*. Unlike Amiodarone, *Sotalol reduces the fibrillation threshold*. Side effects of Sotalol include *bradycardia* that is due to beta blocking property and *Q-T prolongation* and *risk of polymorphic VT* (Torsades) as part of potassium channel blocking. Because of predominant renal clearance, Sotalol is *not ideal in renal dysfunction*. In patients with risk factors for polymorphic VT (Torsades), initiation of Sotalol should only be done under monitoring in the hospital.

Class IV Antiarrhythmics

These agents are otherwise known as **Calcium channel blockers**. Depending on the chemical properties and resulting pharmacodynamic effect, calcium channel blockers are classified into **Dihydropyridine** (**Nifedi*pine***, **Amlodi*pine***, **Felodi*pine***, **Nicardi*pine*** etc.) and **non-dihydropyridine** (**Verapamil** and **Diltiazem**). Because of the predominant inhibitory effect on the SA and the AV node, *non-dihydropyridine calcium channel blockers have pertinent electrophysiologic properties* compared to that of dihydropyridines. Therefore, Verapamil and Diltiazem are commonly used for *rate control* especially in atrial arrhythmias. These agents *prolong AV node conduction* and *refractoriness* and thereby reduce ventricular rate in AV nodal re-entry tachycardia. They do not have much effect on ventricular arrhythmias. Non-dihydropyridine calcium channel blockers are used for various other purposes in cardiovascular medicine including management of hypertension, vasospastic angina, hypertrophic cardiomyopathy etc.

Calcium channel blockers can cause *bradycardia* and especially orthostatic *hypotension*. These agents are *not indicated in pregnancy*, *post myocardial infarction*, *severe sinus node dysfunction* or *conduction disturbance*, *WPW syndrome*, *severe aortic stenosis* etc. In the event of digoxin toxicity, concomitant use of Verapamil can cause complete heart block. Overdose with calcium channel blockers can be treated with administration of **calcium gluconate**.

Class V Antiarrhythmics

Digoxin and **Adenosine** are categorized in this group. Among these, Digoxin implies it's antiarrhythmic properties due to *increased vagal tone*. It produces *increasing contractility* (*positive inotropic effect*) in myocardium by *increasing intracellular calcium* concentration. Because of the smaller therapeutic window (**0.8 - 1.2** ng/ml), Digoxin needs *frequent monitoring of blood levels*. It also has interaction with many other drugs including Amiodarone, warfarin etc. In toxic dosage, digoxin causes *high-grade atrioventricular block* or *accelerated junctional rhythm* and at times *ventricular tachycardia*.

Effect of digoxin in EKG

Digitalis effect

- Short QT interval
- Down sloping ST depression (Reverse tick mark appearance as shown in leads V5,V6 in Fig 14.2)
- Decreased T wave amplitude
- Long PR interval

Digoxin Toxicity

- Any type of arrhythmia resulting from either a disturbance in impulse formation or conduction except Bundle branch block like
  - Paroxysmal atrial tachycardia ,
  - Atrial fibrillation with heart block
  - Second of third degree heart block
  - Accelerated junctional or Idioventricular rhythm

Box 14.3 Effect of Digitalis in EKG

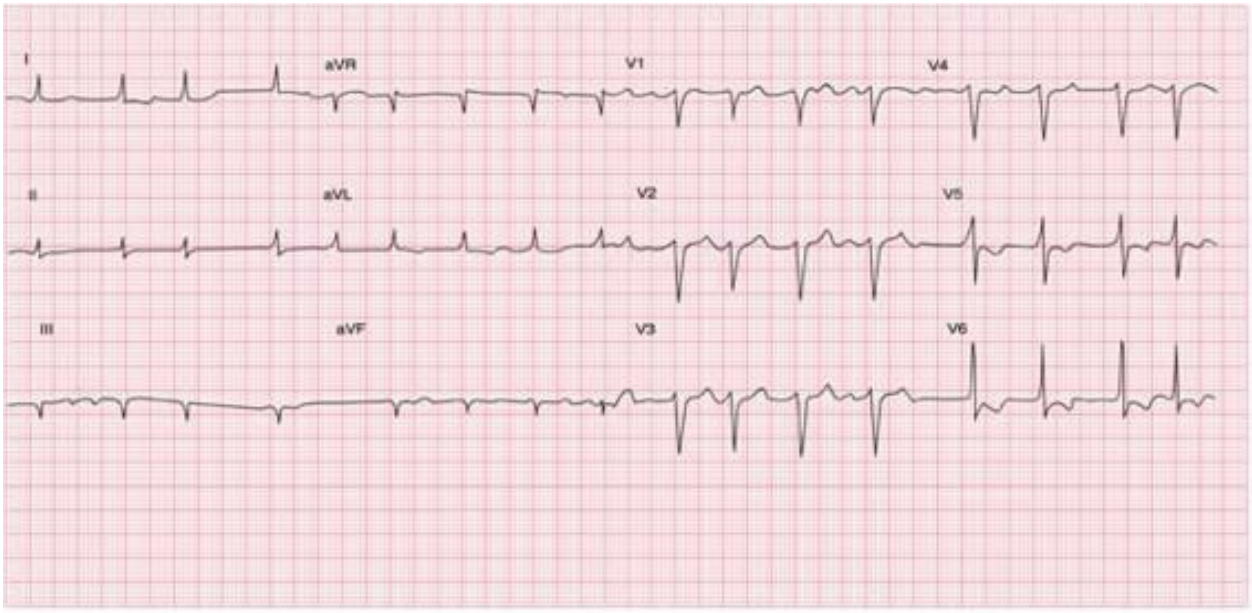


Fig 14.2 Digitalis effect (Note the presence of 'reverse tick mark' appearance of St segment in Lead V5 and V6).

In patients with **WPW pattern** of EKG, digoxin administration should be avoided because of the risk of *delaying AV nodal conduction and promoting accessory pathway*; leading to *ventricular tachyarrhythmia*. **Digoxin immune FAB** antibody therapy can be used in severe digoxin toxicity. Tachyarrhythmia secondary to digoxin toxicity should not be treated with DC cardioversion because of the risk of ventricular tachycardia.

Adenosine acts through specialized *potassium channels* within the atrium, SA and AV node. Because of the lack of these channels in ventricular myocardium, Adenosine *doesn't have any direct effect on ventricles*. Having a very short half-life, rapid intravenous injection of adenosine produces profound and transient AV nodal conduction block and therefore useful in termination of **paroxysmal SVT**. If the patient has concomitant use of **Theophylline**, *Adenosine does not work* because of the adenosine receptor blockade from Theophylline. Because of the possible micro re-entry circuit within atria, Adenosine can produce *atrial fibrillation in 10-15% of patients*. Use of **Dipyridamole** can prolong the effect of adenosine and therefore should be use with caution.

Points to Remember!!!

- Antiarrhythmic drugs are classified based on their mechanism of action into class I (sodium channel blockers), class II (beta-blockers), class III (potassium

channel blockers) and class IV (calcium channel blockers).

· Class I antiarrhythmic are further classified into class 1A (Quinidine, Procainamide and Disopyramide), class 1B (Lidocaine and Mexiletine) and class 1C (Flecainide and Propafenone).

· Class I antiarrhythmic drugs work on phase 0 of myocardial action potential curve.

· Among class I antiarrhythmics, class 1C drugs have maximum sodium channel blocking property.

· Class I drugs can prolong QT interval and therefore needs to be monitored in the hospital on initiation.

· Lidocaine and Mexiletine are classified into class 1B antiarrhythmics, particularly useful in treating ventricular arrhythmias in the event of myocardial infarction.

· Flecainide and Propafenone are major class 1C antiarrhythmics, used for treatment of atrial fibrillation and are contraindicated in structural heart disease.

· Beta-blockers constitute class II antiarrhythmics.

· Beta-blockers can be cardio selective and non-selective depending on specificity to beta-1 or beta-2 receptors.

· Beta-blockers cause negative inotropic effect (reduce contractility) and negative chronotropic effect (reduce automaticity).

· Class III antiarrhythmic drugs are otherwise known as potassium channel blockers.

· Sotalol has both beta blocking and potassium channel blocking activity.

· Amiodarone has predominant class III antiarrhythmic property; however, has class 1 to 4 effect in various conditions.

· Amiodarone does not need hospitalization for initiation.

· Long-term use of Amiodarone can cause pulmonary disease, hyperthyroidism, peripheral neuropathy etc.

· Long-term Amiodarone use may increase defibrillation threshold (energy needed for successful conversion of ventricular tachycardia).

· Sotalol reduces defibrillation threshold and thereby reducing the amount of energy needed for successful defibrillation.

· Because of the major renal excretory pathway, Sotalol is contraindicated for renal dysfunction.

· Calcium channel blockers are classified as class IV antiarrhythmics. They are divided into dihydropyridines (Nifedipine, Felodipine, Amlodipine) and non-dihydropyridines (Verapamil and Diltiazem).

· Non-dihydropyridine calcium channel blockers have electrophysiologic properties of prolonging AV node conduction and refractoriness.

· Calcium gluconate is the antidote for calcium channel blocker toxicity.

· Digoxin has a narrow therapeutic window of blood level (0.8 to 1.2 ng/ml).

· Digoxin should be avoided in patients with WPW pre-excitation because of the

possible facilitation of impulse conduction through accessory pathway and resultant ventricular arrhythmia.

- Adenosine has no direct effect in the ventricle. Theophylline, which is an adenosine receptor blocking agents may prevent adenosine from working when used together.
- Adenosine has the potential for triggering atrial fibrillation.

Test Your Understanding

1. Which of the following represents a member of class III antiarrhythmics?

A Flecainide
B Carvedilol
C Amiodarone
D Procainamide

2. Which of the following best describes mechanism of action of Propafenone?

A Potassium channel blocking
B Calcium channel blocking
C Beta blocking
D Sodium channel blocking

3. Which of the following is the most potent sodium channel blocker?

A Quinidine
B Flecainide
C Lidocaine
D Mexiletine

4. Which of the following sodium channel blocking agents is useful in acute phase of ventricular arrhythmia secondary to myocardial infarction?

A Procainamide
B Amiodarone
C Lidocaine
D Flecainide

5. Which of the following is not an antiarrhythmic agent requiring in-hospital monitoring for initiation?

A Sotalol
B Procainamide
C Amiodarone
D Quinidine

6. Which of the following clinical condition is contraindicated for the use of Flecainide?

A Non-critical atherosclerosis
B Recent myocardial infarction
C Peripheral vascular disease

D Chronic hepatitis

7. Which of the following is an example for nonselective beta-blocker?

A Carvedilol
B Atenolol
C Metoprolol
D Esmolol

8. Which of the following statement is true regarding Sotalol?

A It has only Class III properties
B It increases defibrillation threshold
C It has sodium and potassium blocking effect
D Chronic use cause pulmonary fibrosis

9. Which of the following is an example for non-dihydropyridine calcium channel blocker?

A Amlodipine
B Nicardipine
C Verapamil
D Nifedipine

10. Digoxin is contraindicated in _____?

A Atrial fibrillation
B Atrial flutter
C Frequent PVCs
D WPW syndrome

Answers

1. C Amiodarone
2. D Sodium channel blocking
3. B Flecainide
4. C Lidocaine
5. C Amiodarone
6. B Recent myocardial infarction
7. A Carvedilol
8. C It has sodium and potassium blocking effect
9. C Verapamil
10. D WPW syndrome

Chapter 15

Rhythm Practice

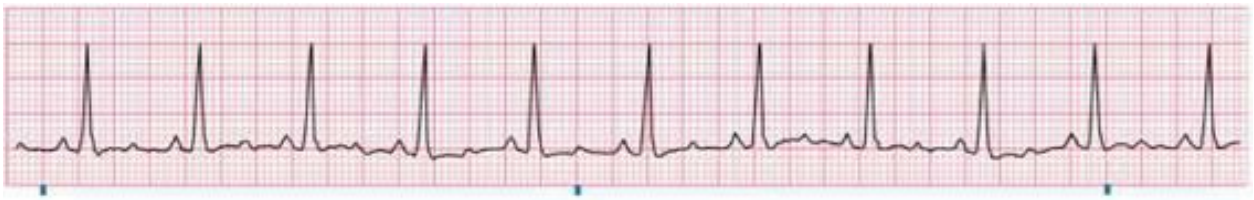
In this chapter

- Single rhythm practice
- 12 lead EKG practice

Most of these rhythm strips and EKG tracings are taken from real patients in various cardiac monitoring settings. Therefore, some of the strips have motion artifact along with underlying rhythm, which can be a bit confusing unless the interpreter is careful. We have done it on purpose to provide a realistic view of EKG interpretation in the field.

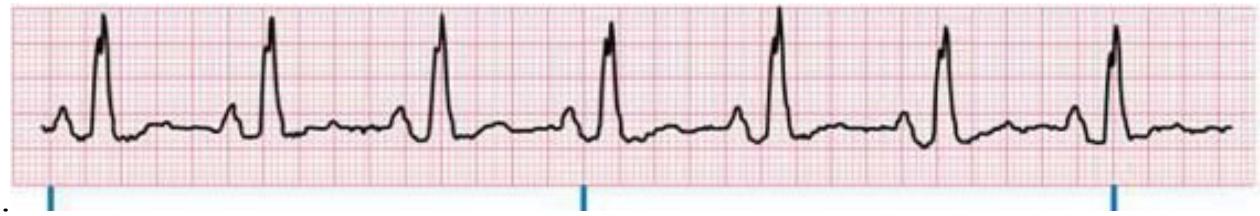
Individual Rhythm Strips

1.

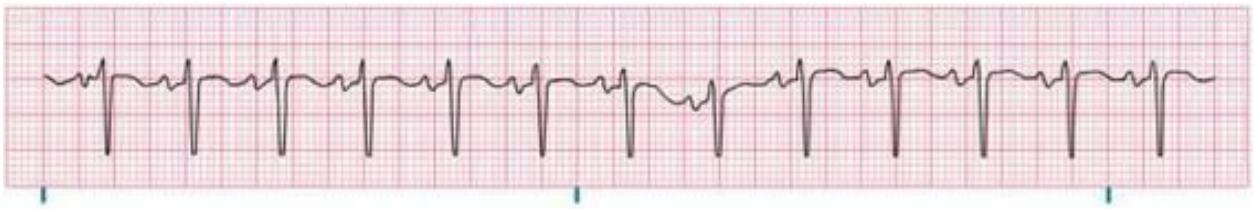


Rate_____ Rhythm _____ P wave _____PR Interval _____
QRS complex_____ QT Interval_____ T wave _____ Other _____
Interpretation _____

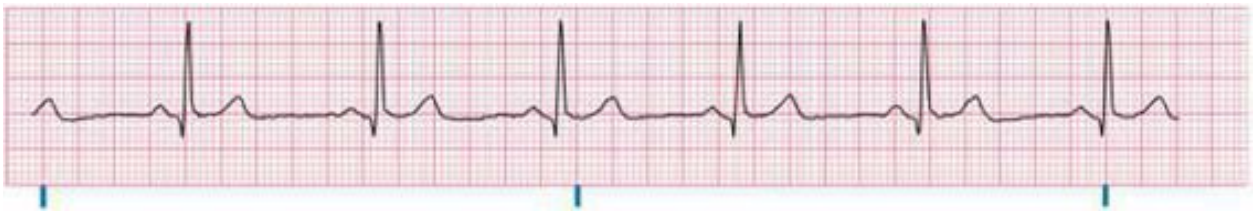
2.



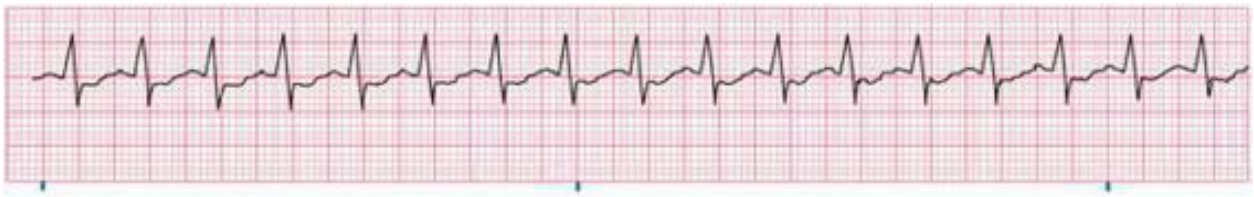
Rate_____ Rhythm _____ P wave _____PR Interval _____
QRS complex_____ QT Interval_____ T wave _____ Other _____
Interpretation _____

3. 

Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

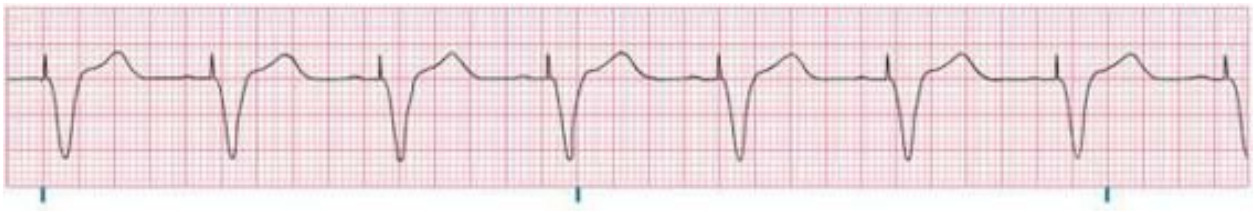
4. 

Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

5. 

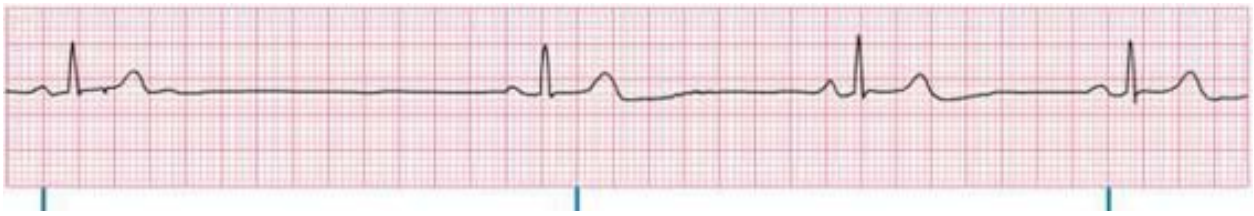
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

6.



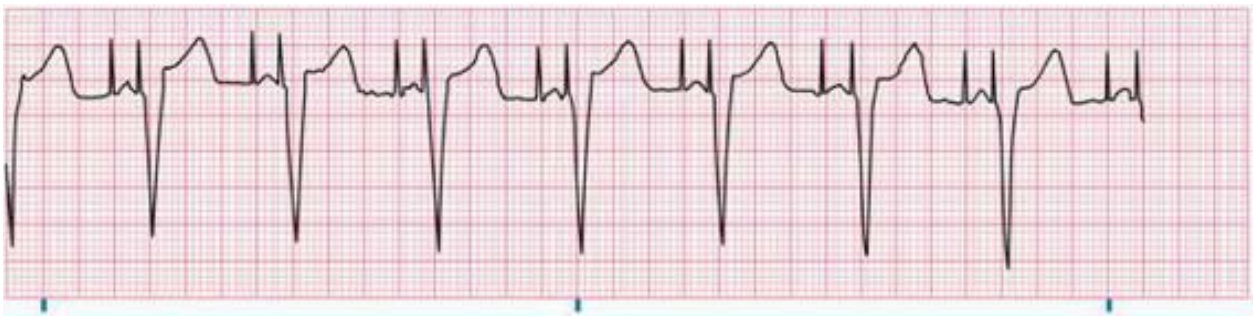
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

7.



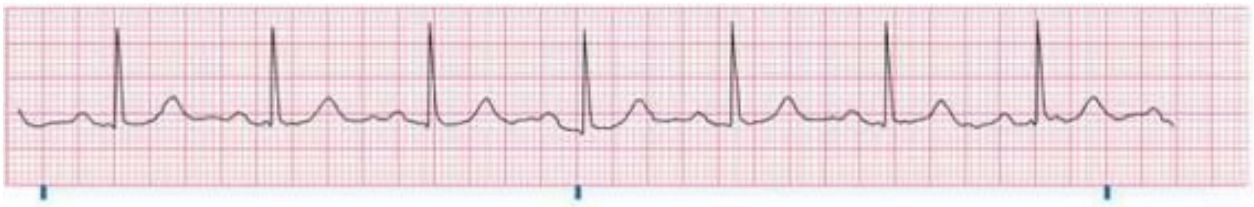
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

8.



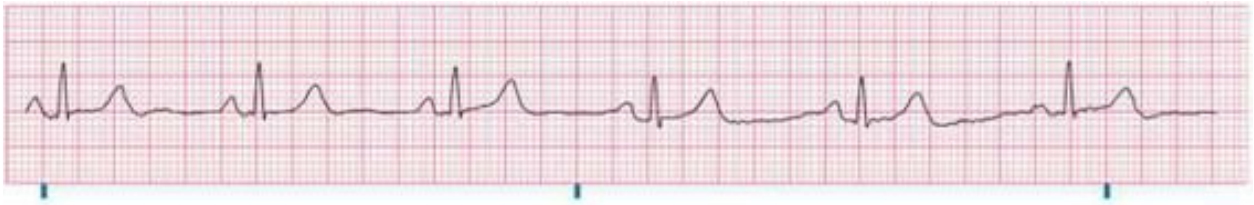
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

9.



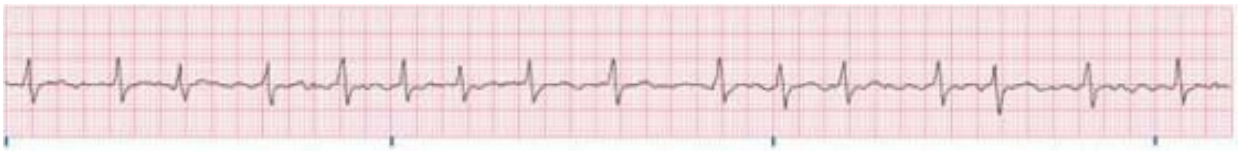
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

10.



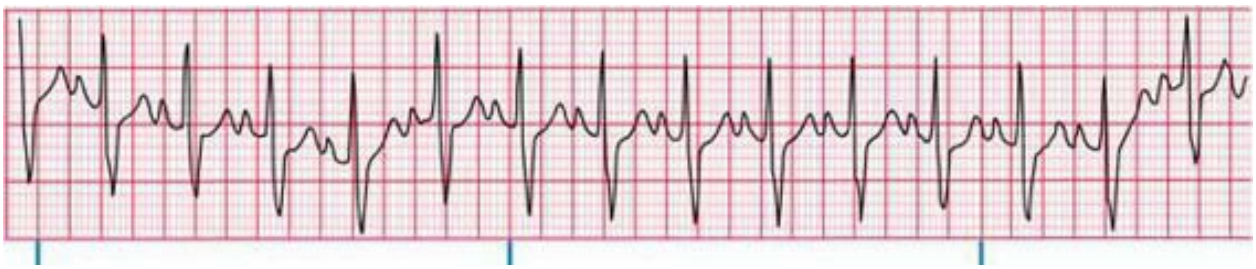
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

11.



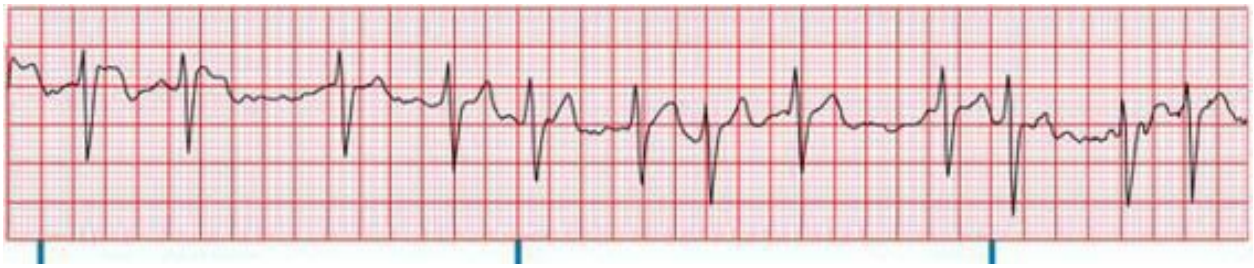
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

12.



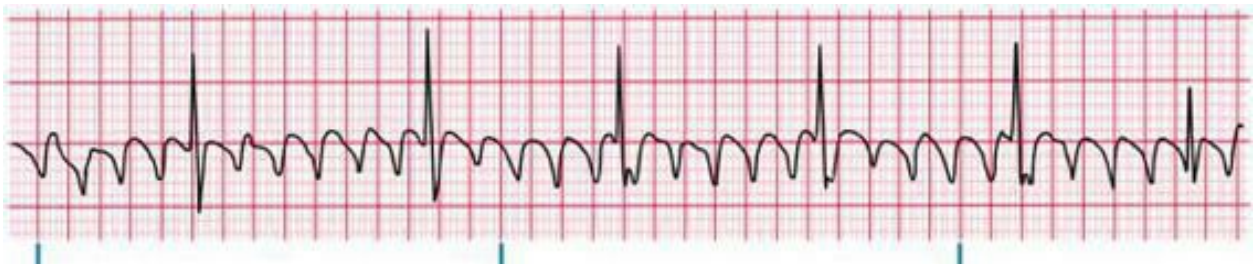
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

13.



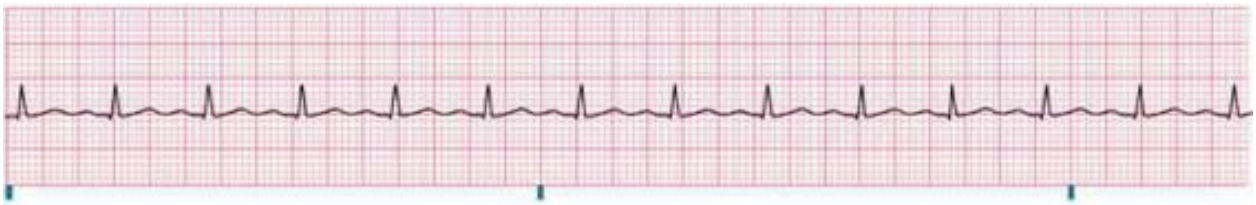
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

14.



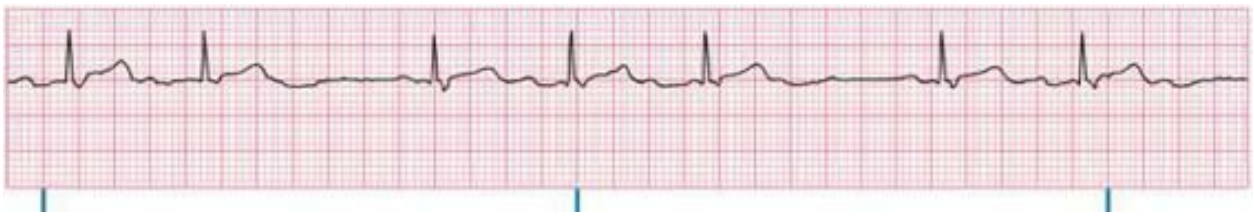
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

15.



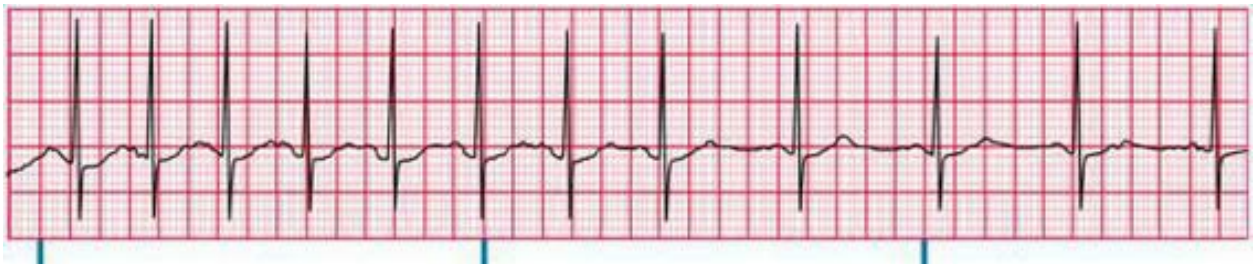
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

16.



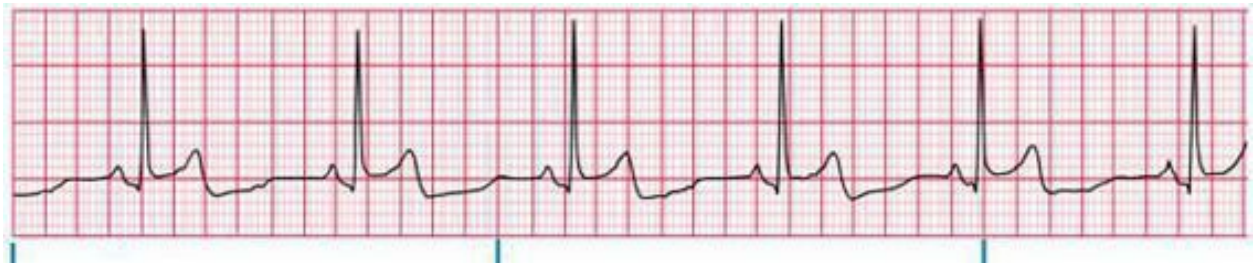
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

17.



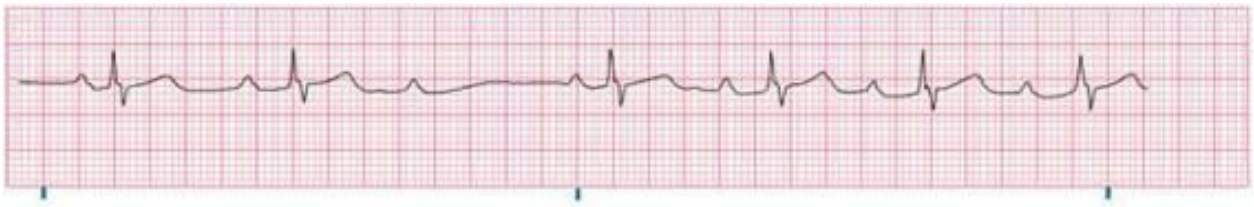
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

18.



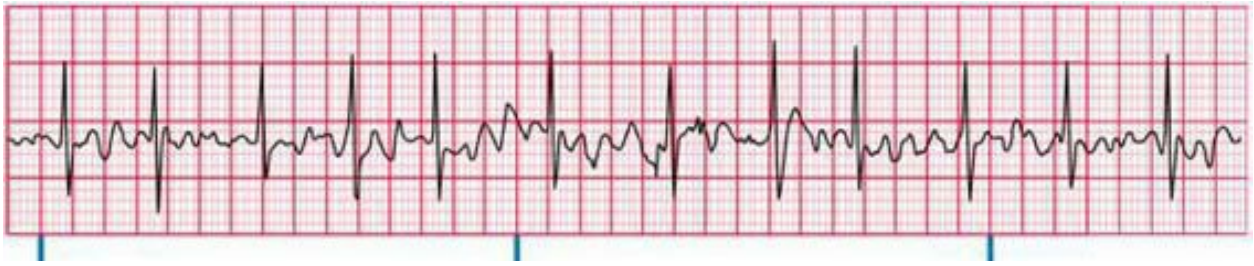
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

19.



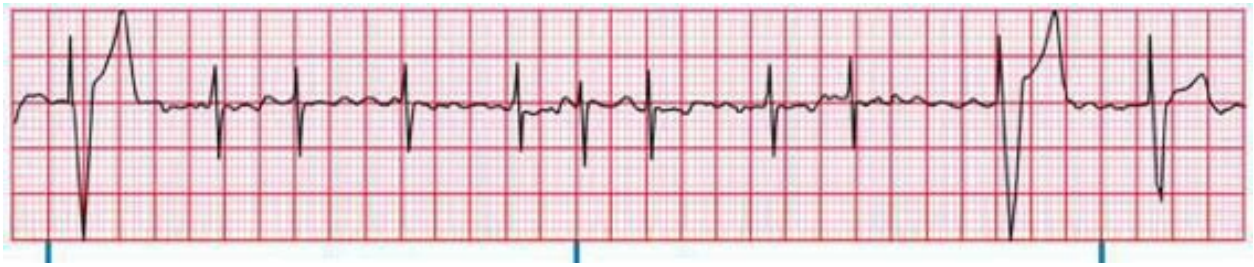
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

20.



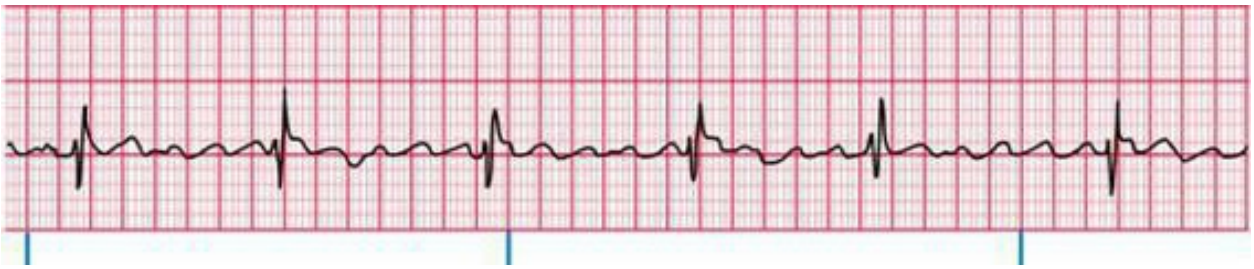
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

21.



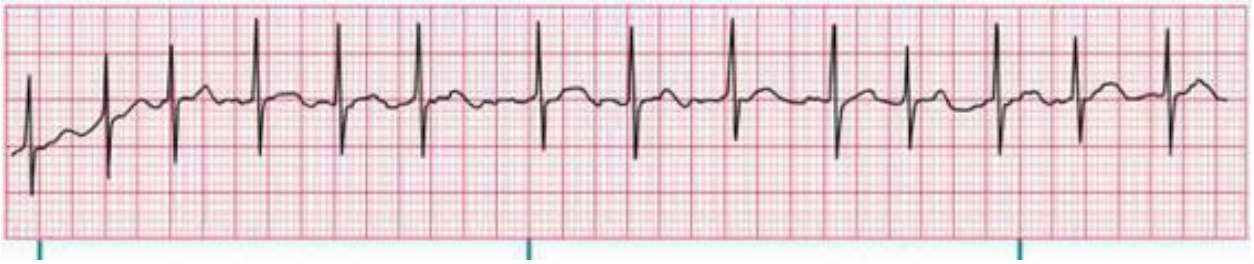
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

22.



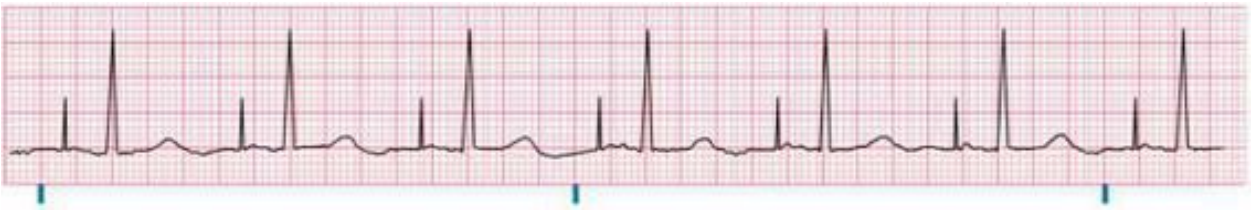
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

23.



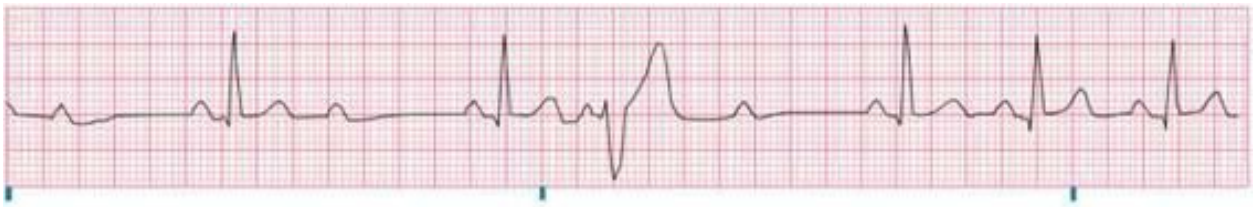
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

24.



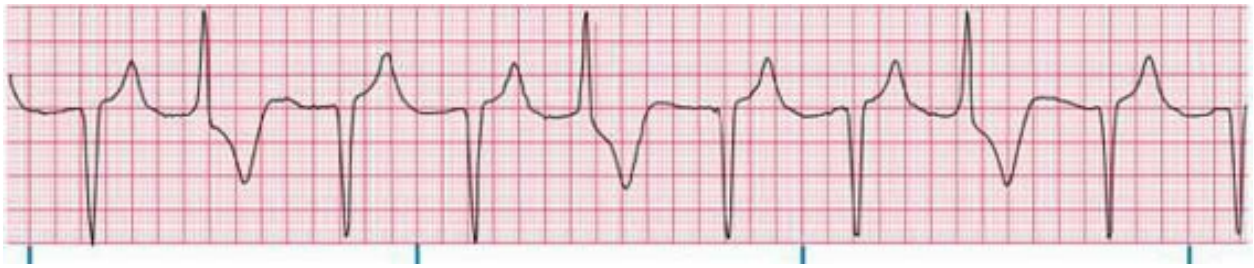
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

25.



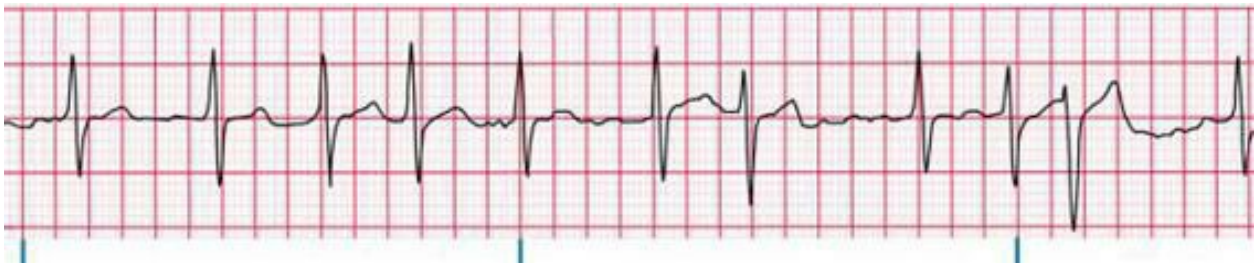
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

26.



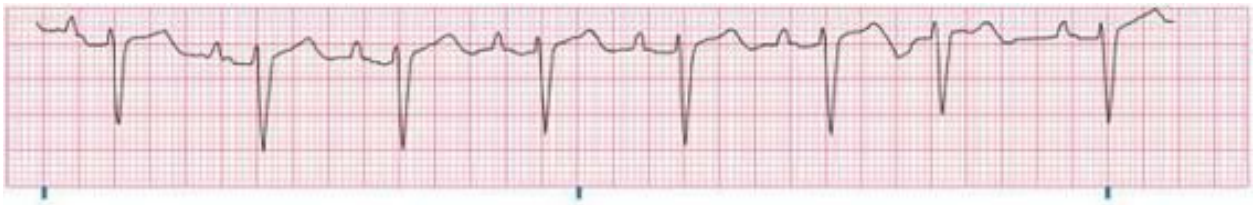
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

27.



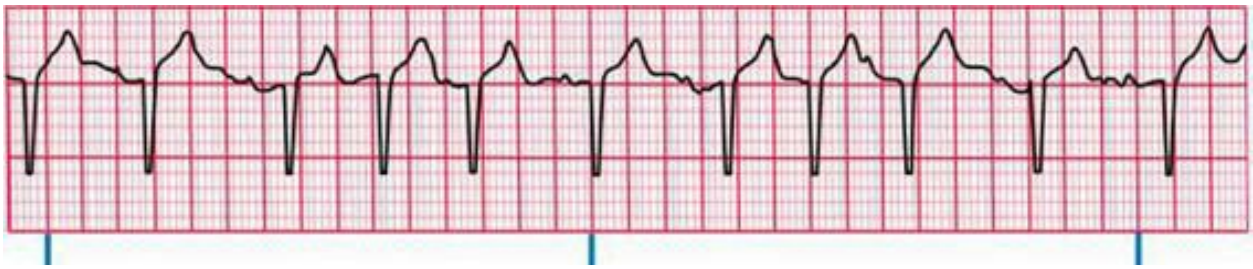
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

28.



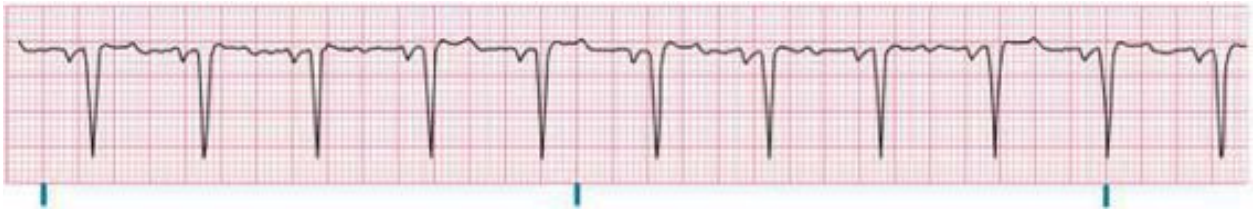
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

29.



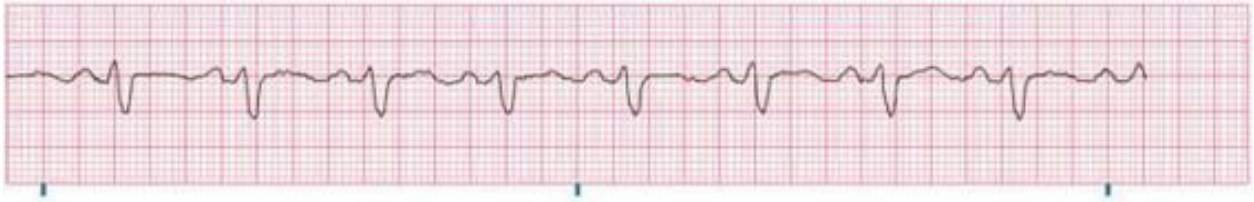
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

30.



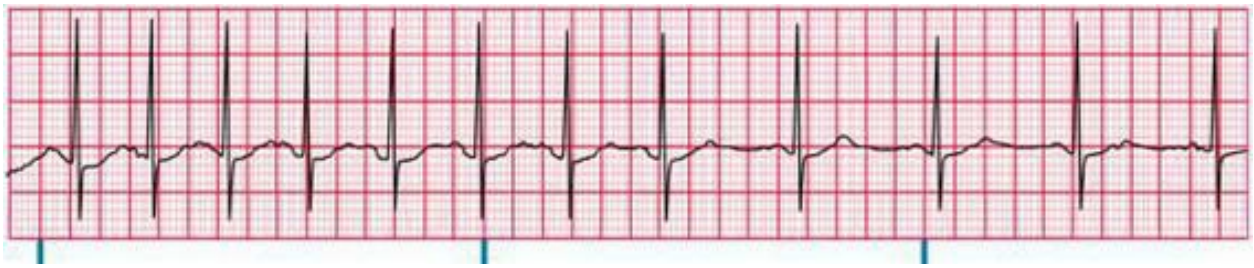
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

31.



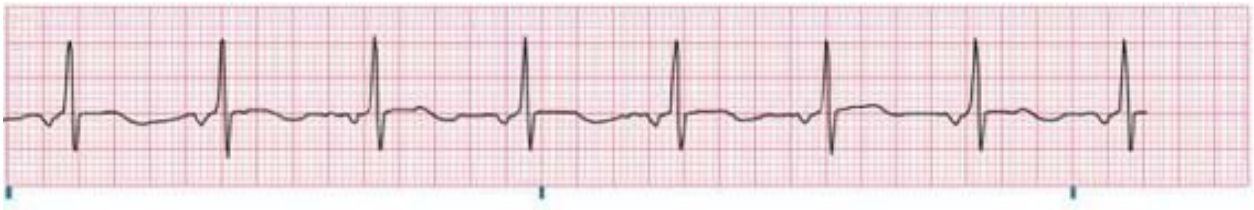
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

32.



Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

33.

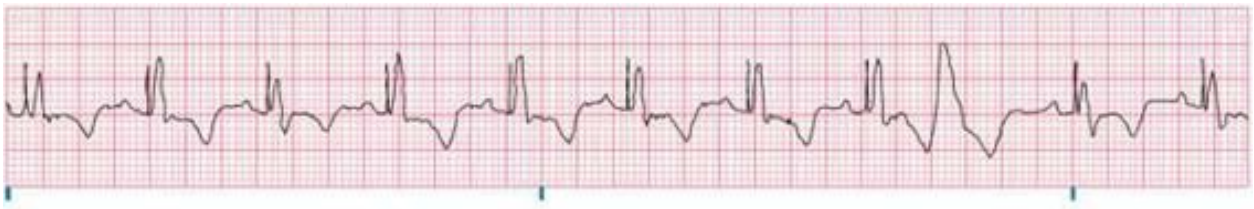


Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

34.

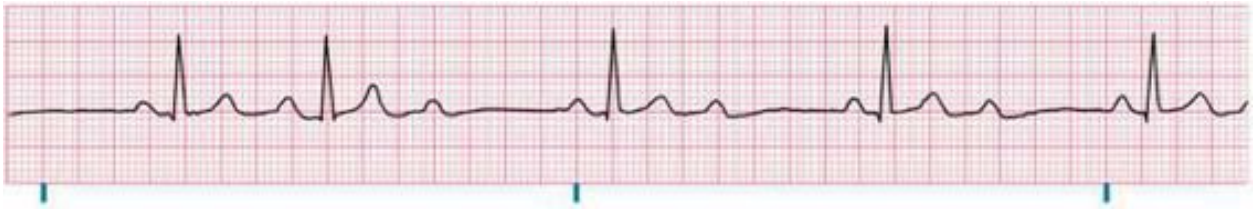
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

35.



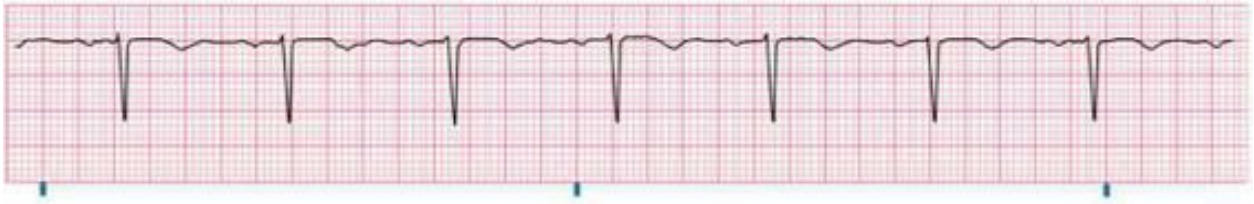
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

36.



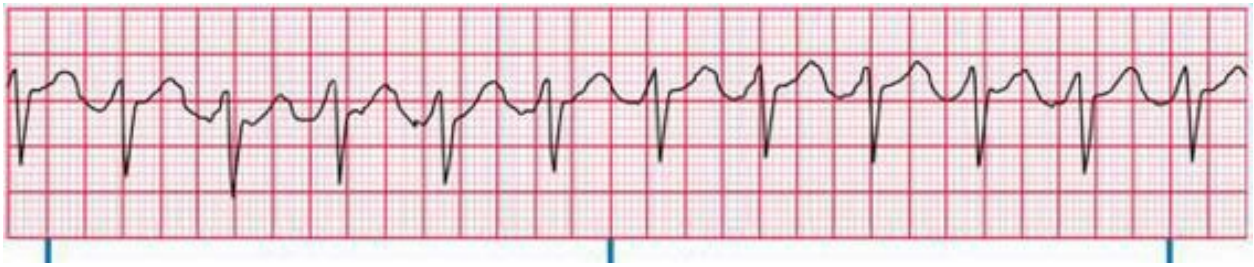
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

37.



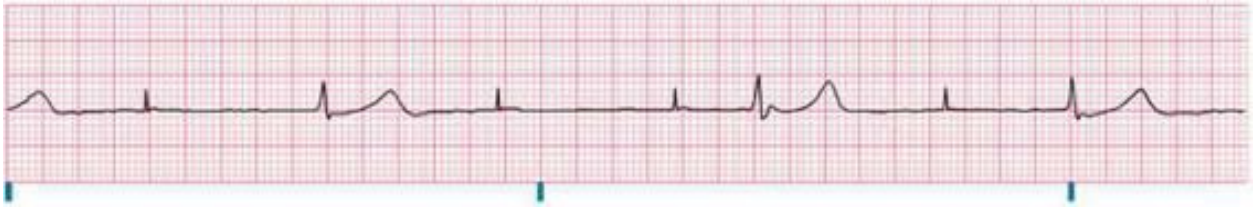
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

38.



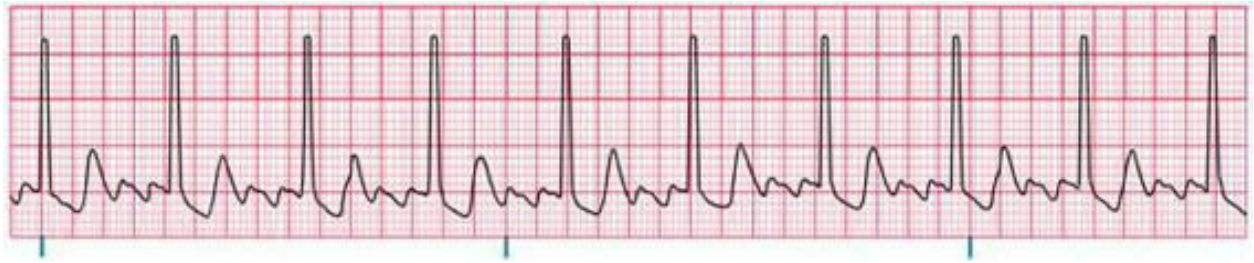
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

39.



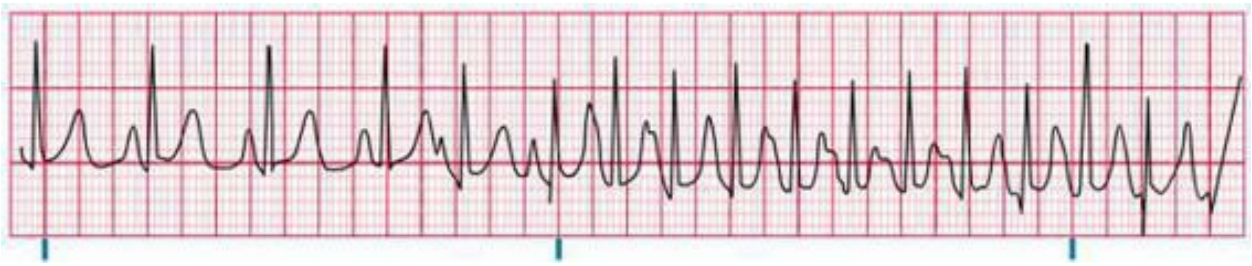
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

40.



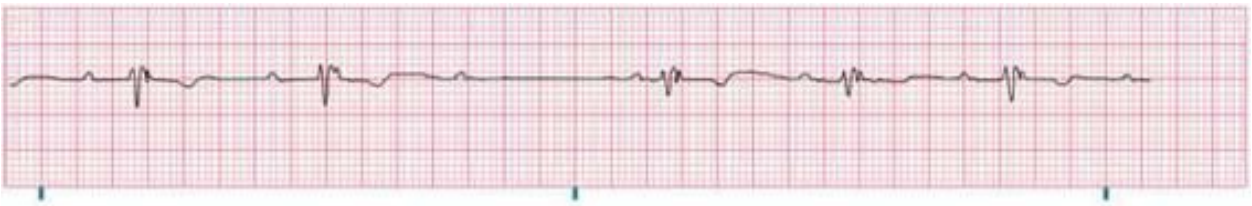
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

41.



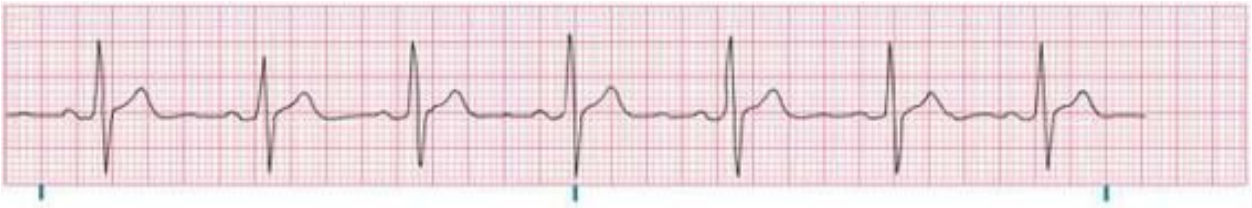
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

42.



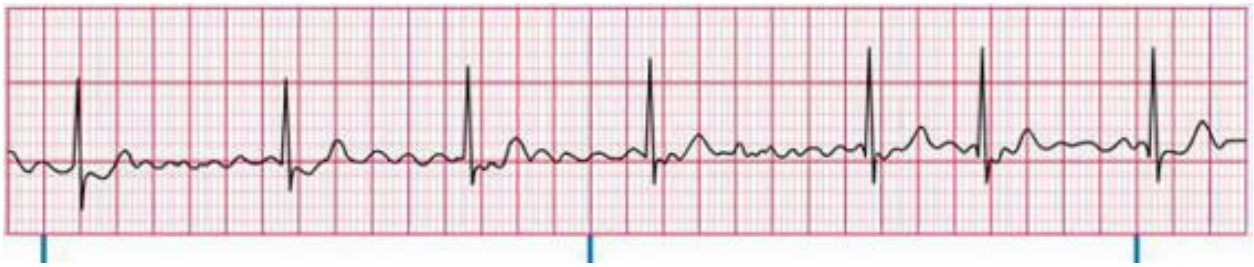
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

43.



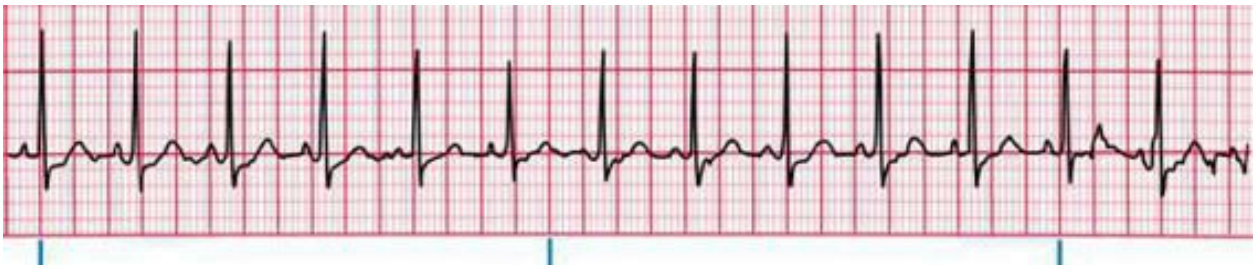
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

44.



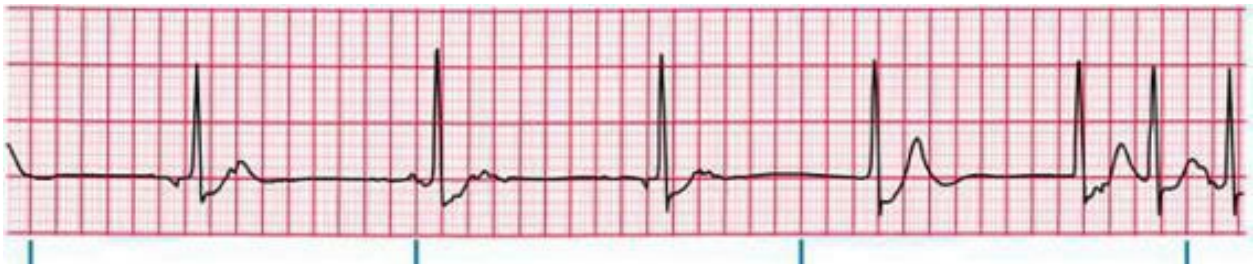
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

45.



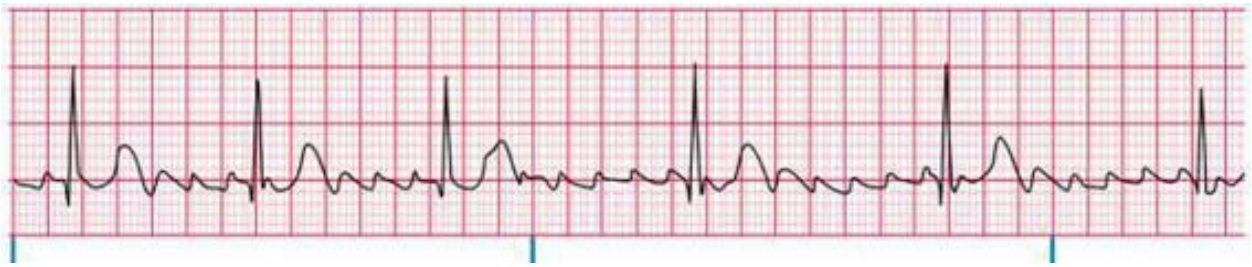
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

46.



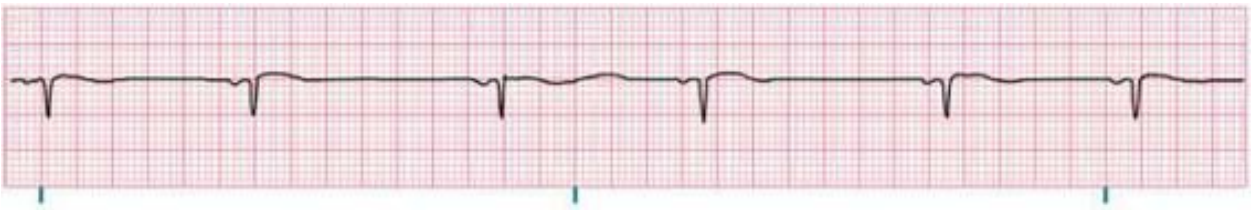
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

47.



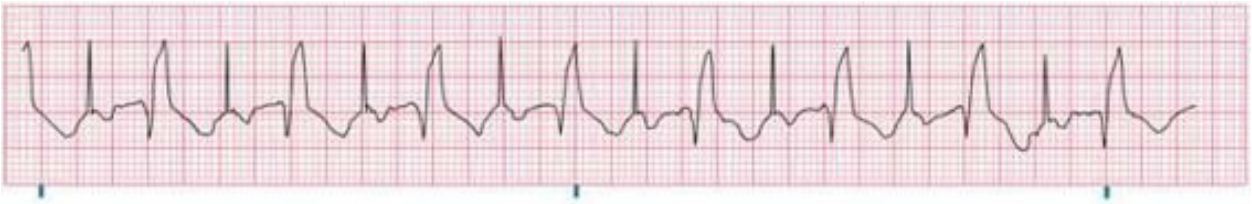
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

48.



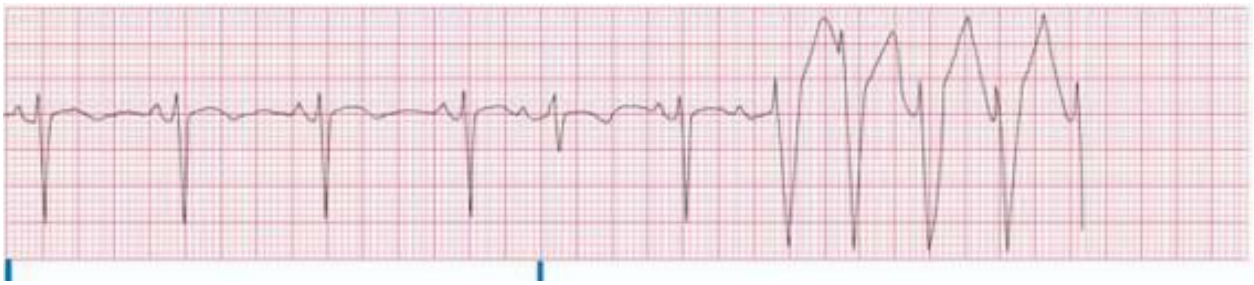
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

49.



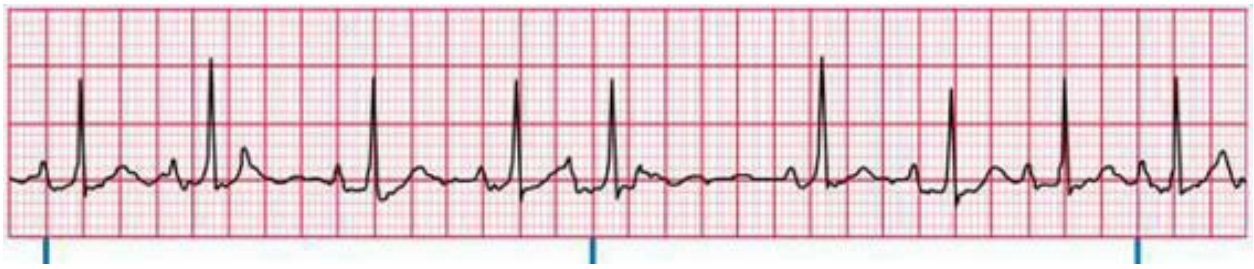
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

50.



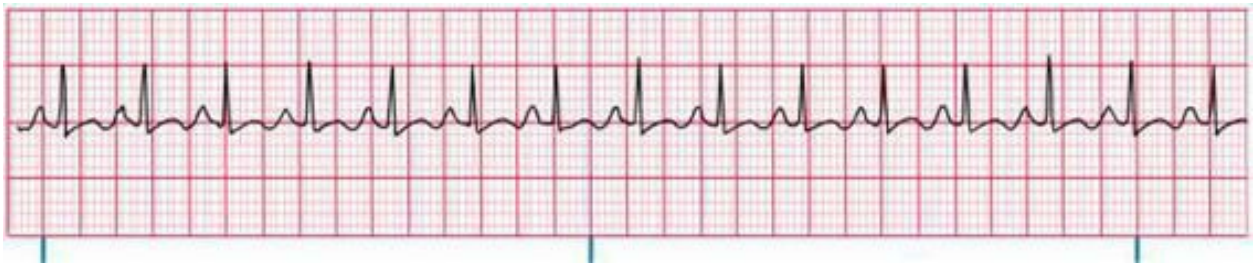
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

51.



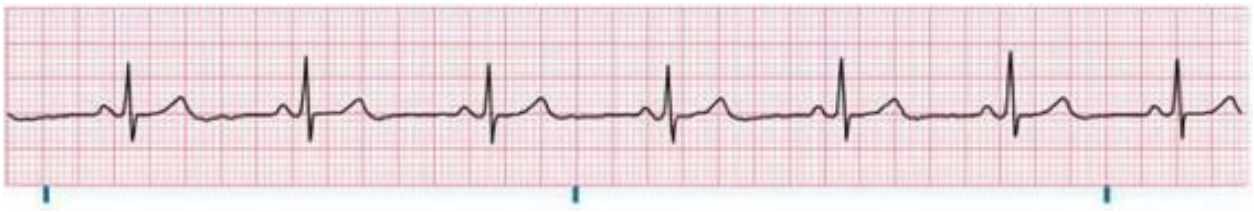
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

52.



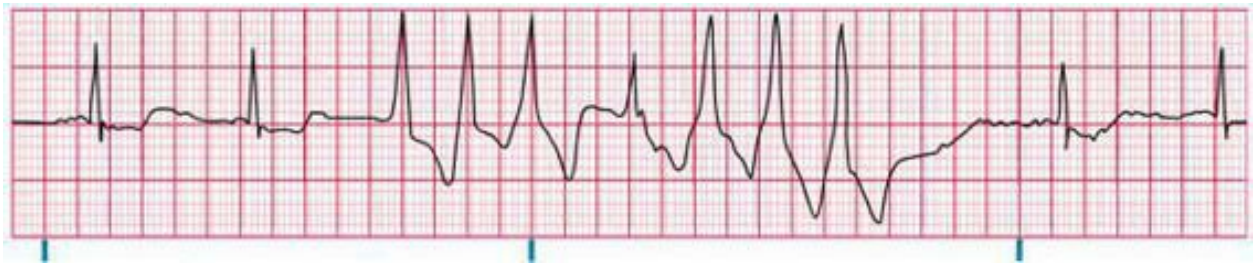
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

53.



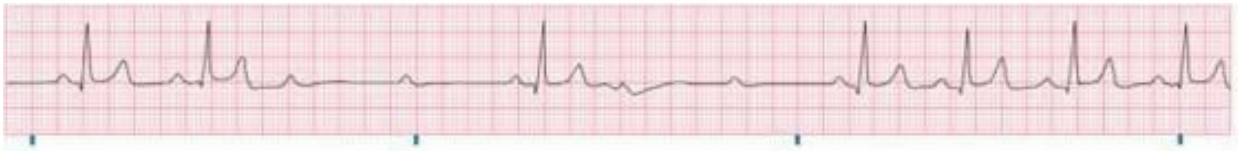
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

54.



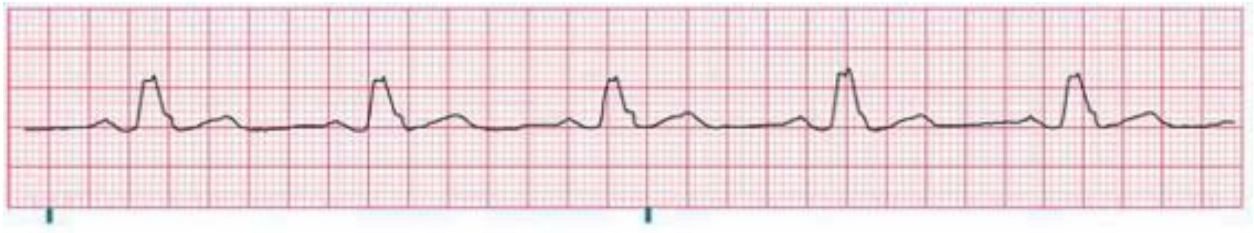
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

55.



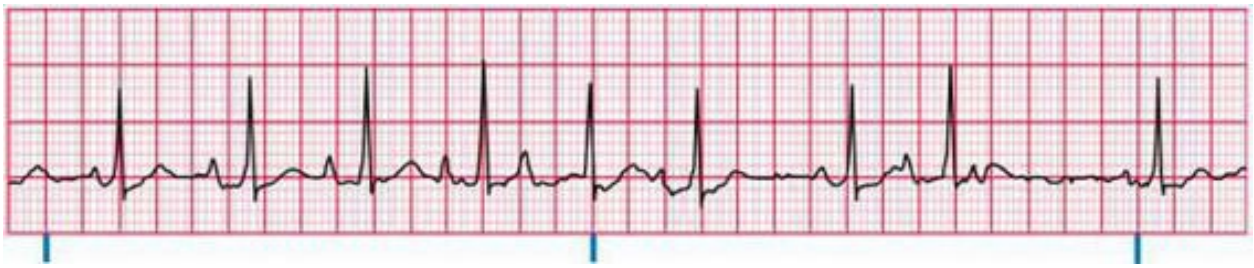
Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

56.



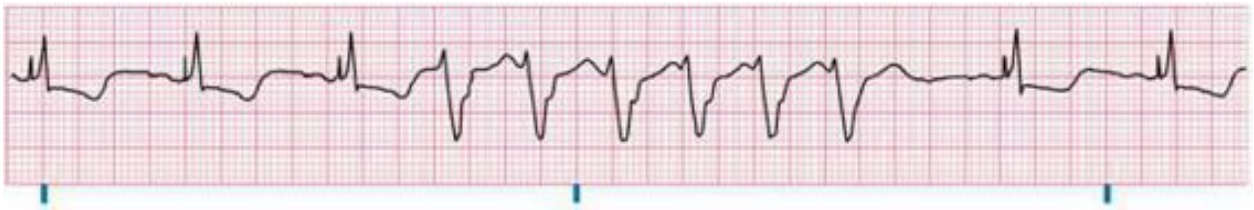
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

57.



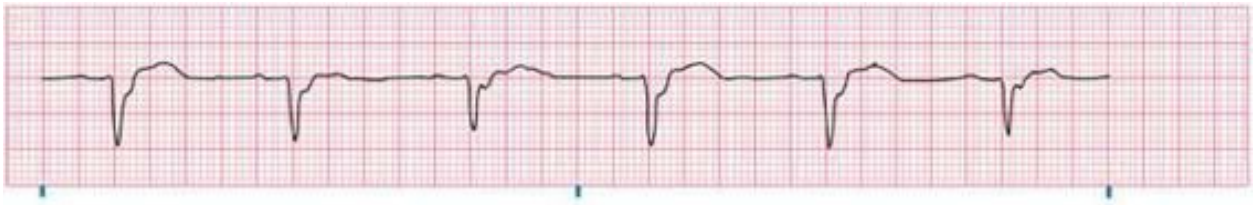
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

58.



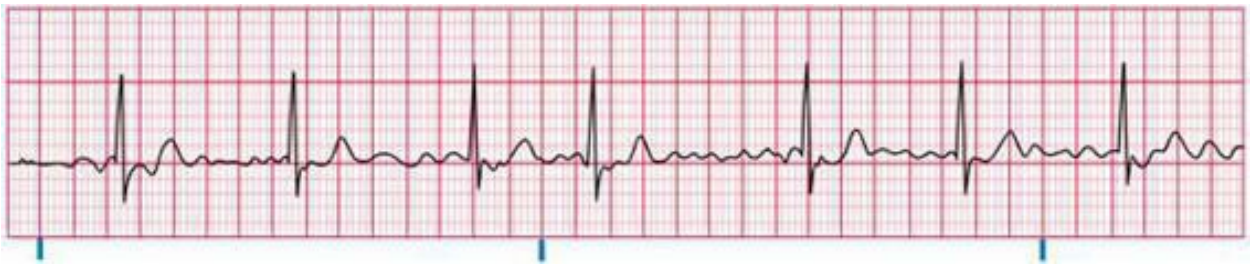
Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

59.



Rate _____ Rhythm _____ P Wave _____ PR Interval _____
QRS Complex _____ QT Interval _____ QTc _____ T Wave _____
Other _____ Interpretation _____

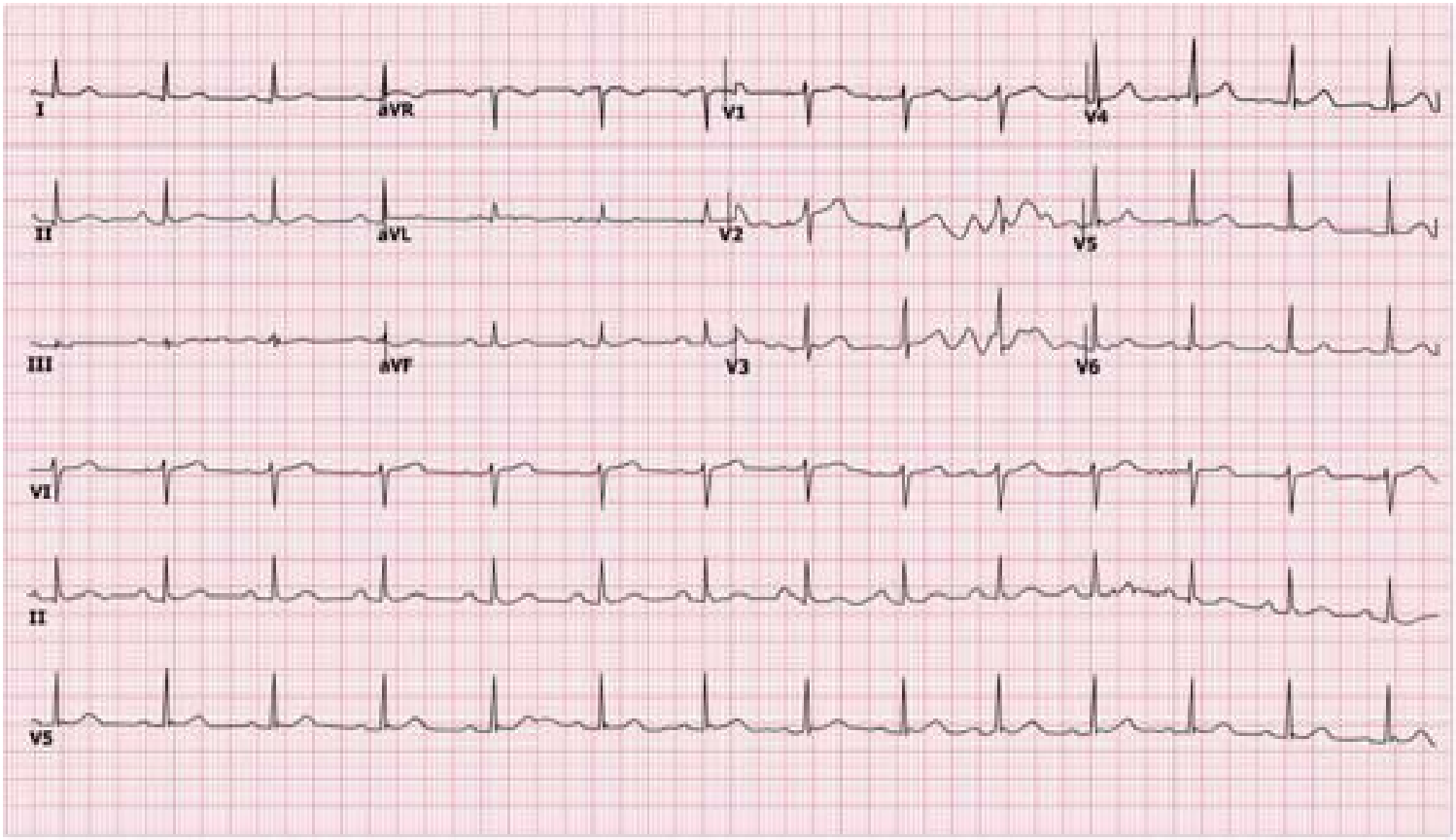
60.



Rate _____ Rhythm_____ P Wave _____PR Interval _____
QRS Complex_____ QT Interval_____ QTc_____ T Wave _____
Other_____ Interpretation_____

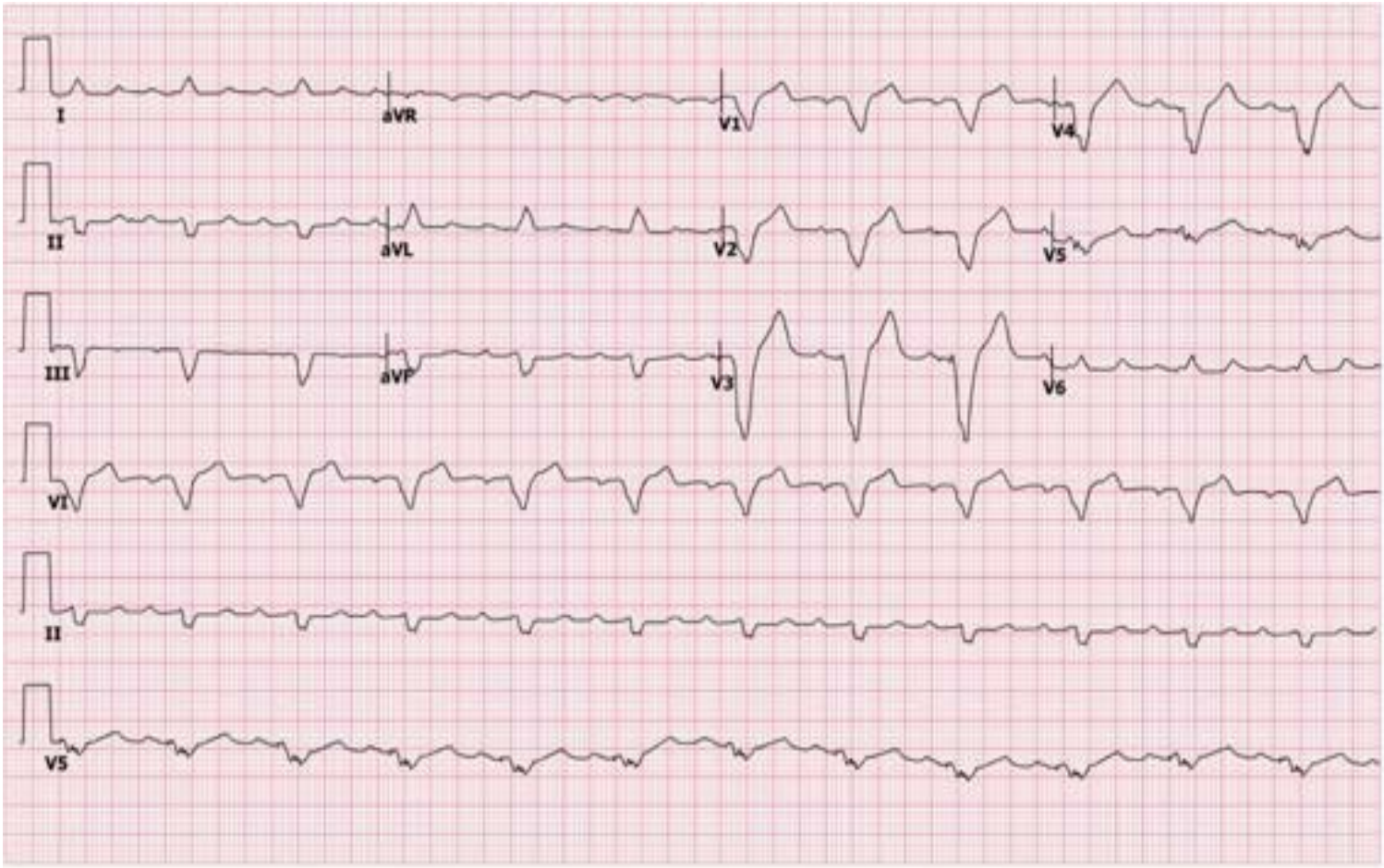
12 Lead EKG Practice

1.



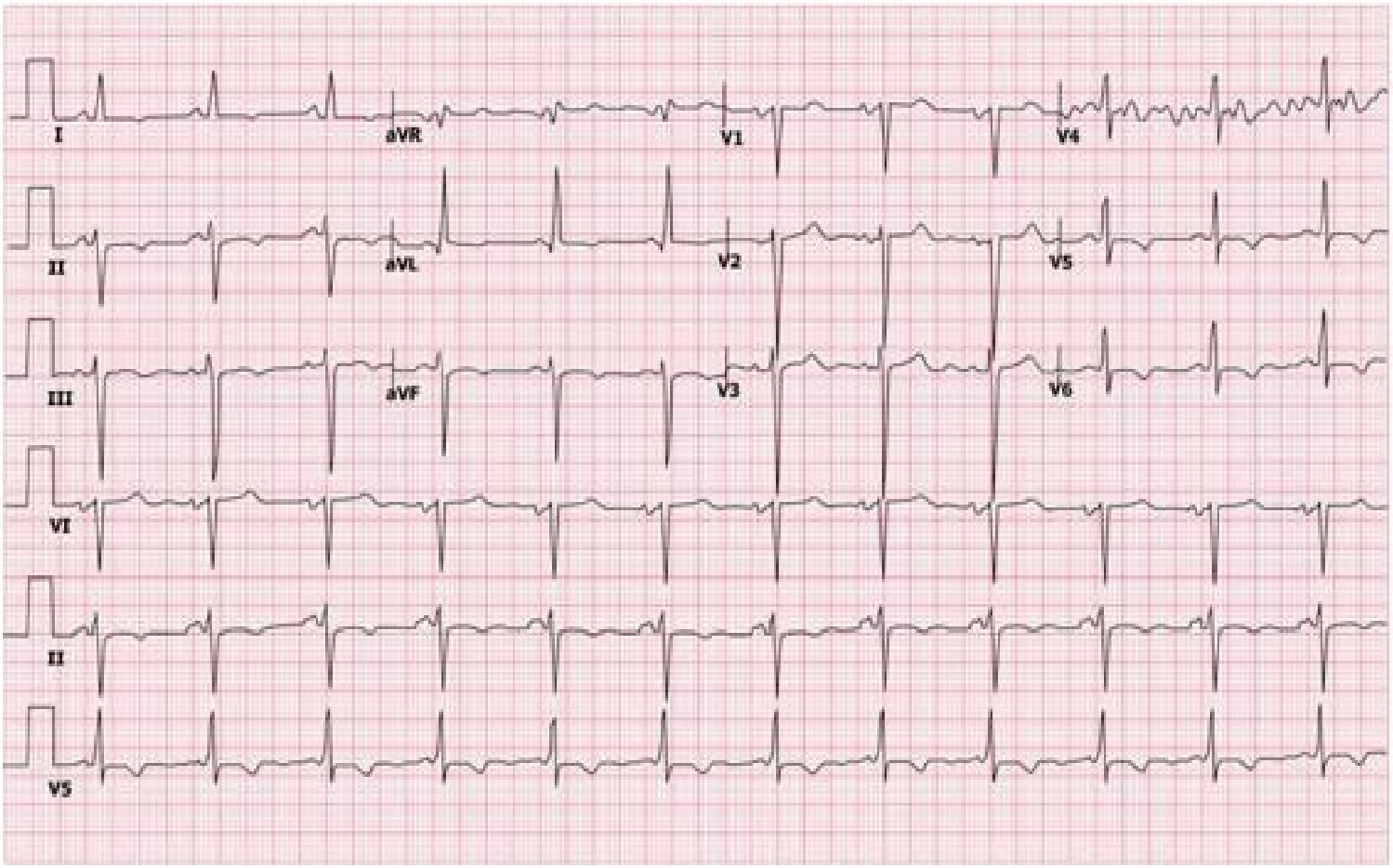
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

2.



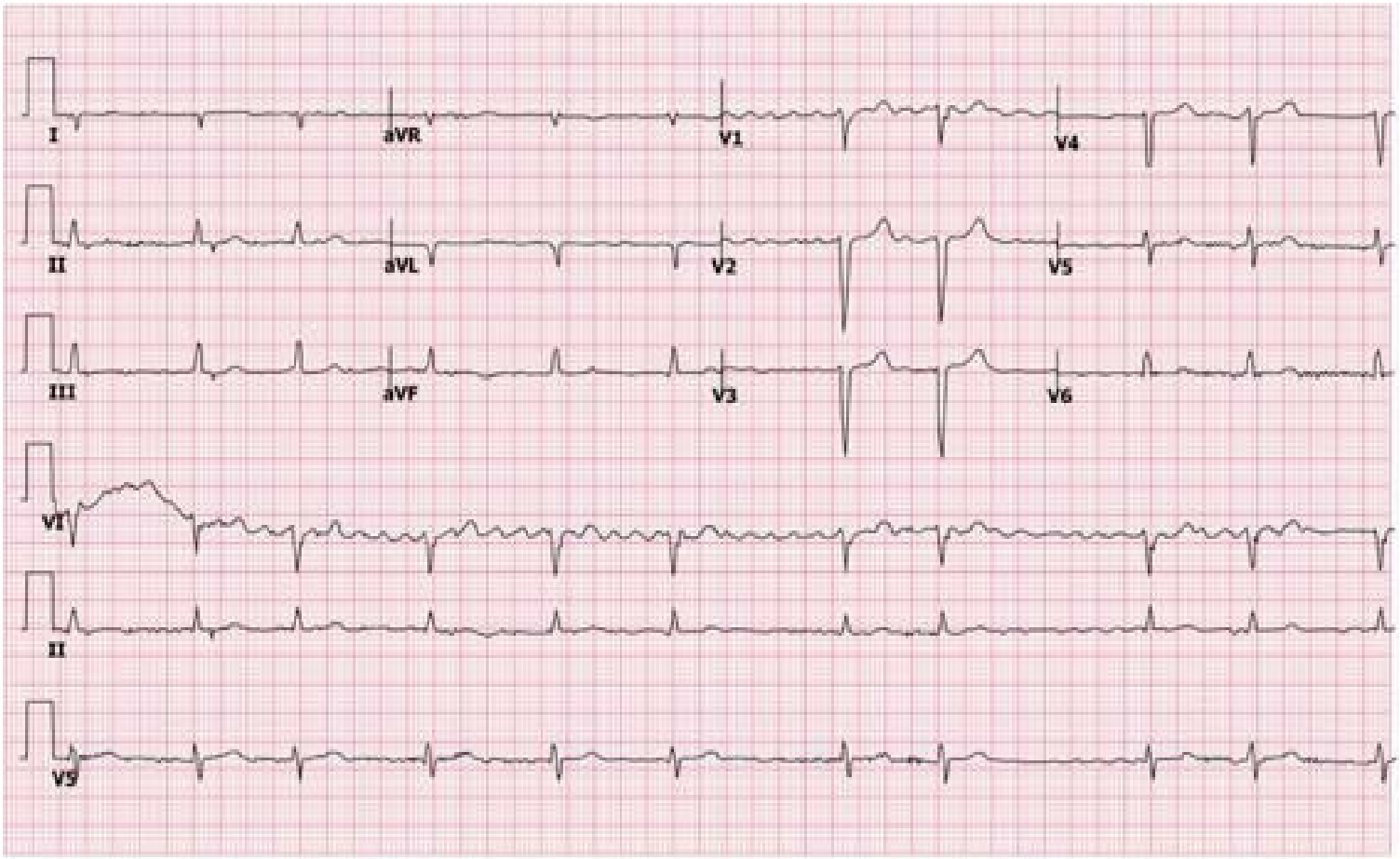
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

3.



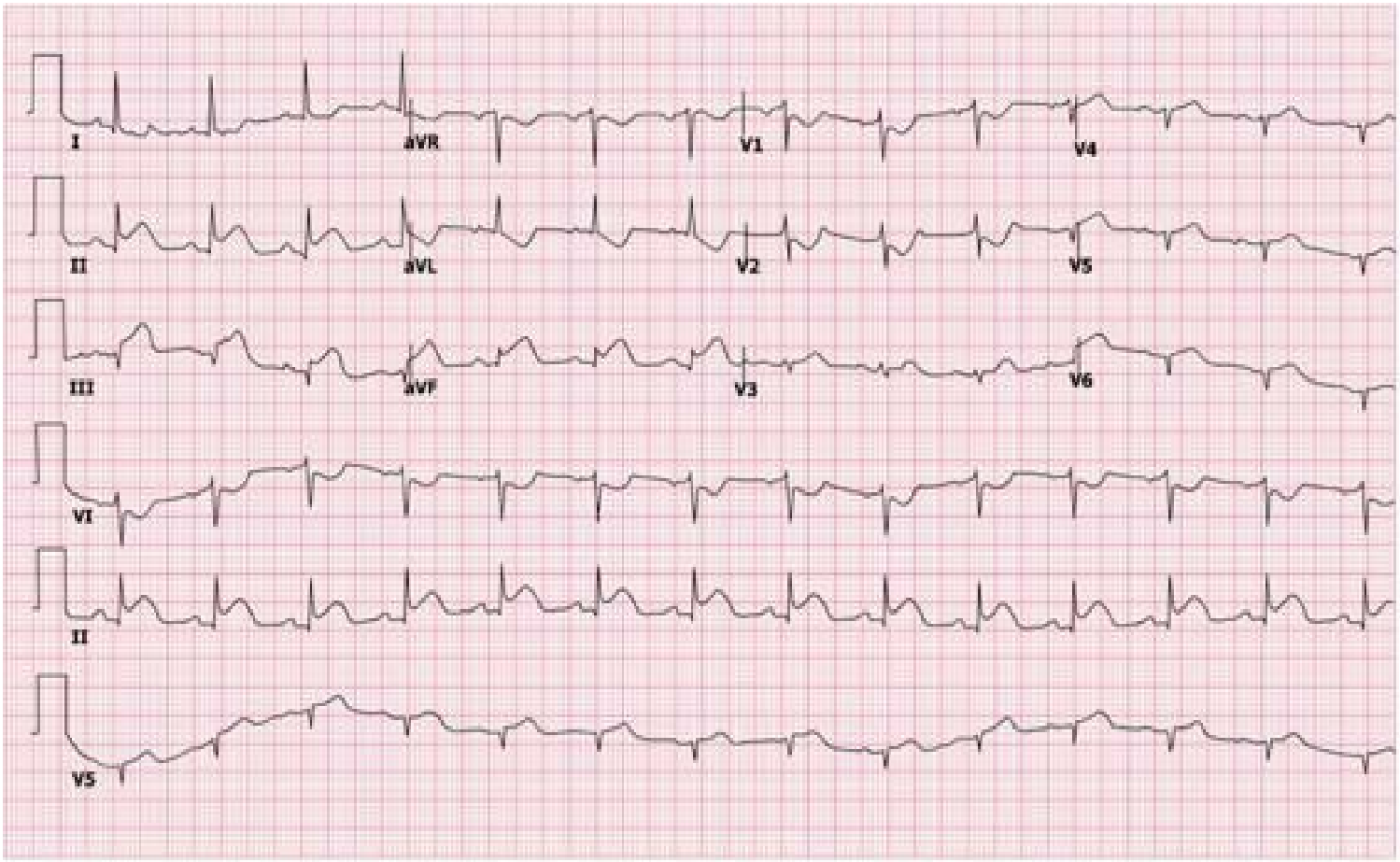
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

4.



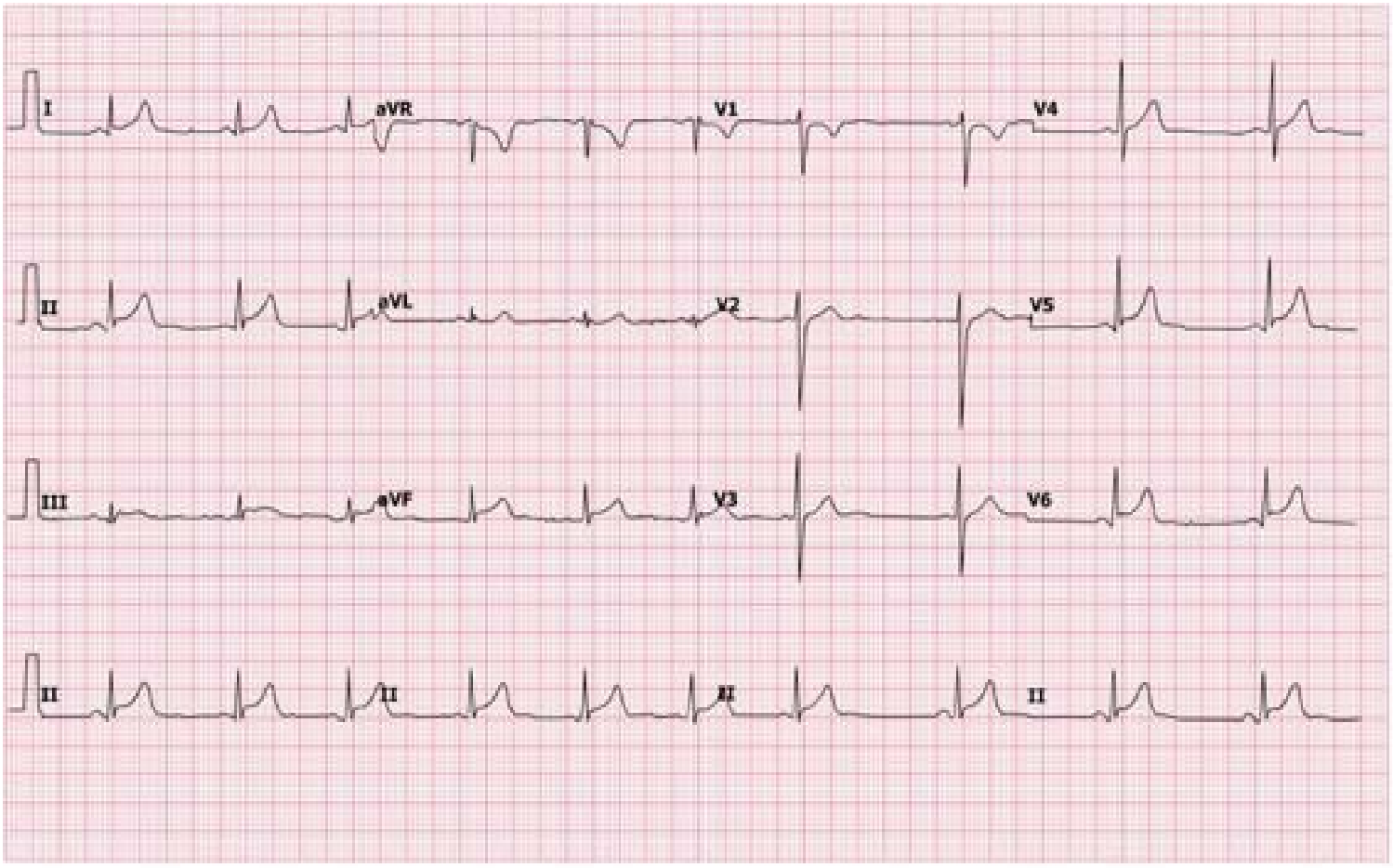
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

5.



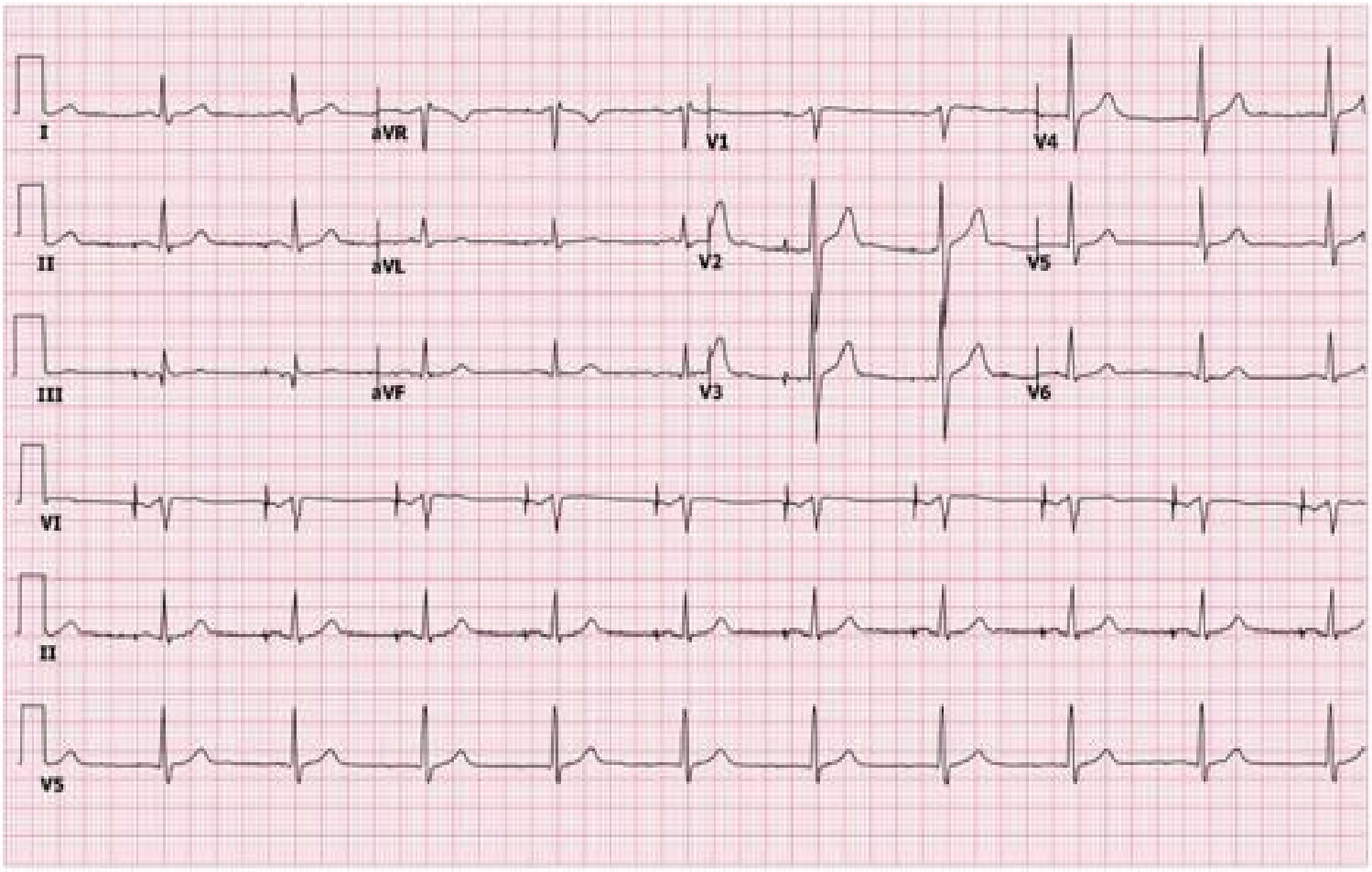
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

6.



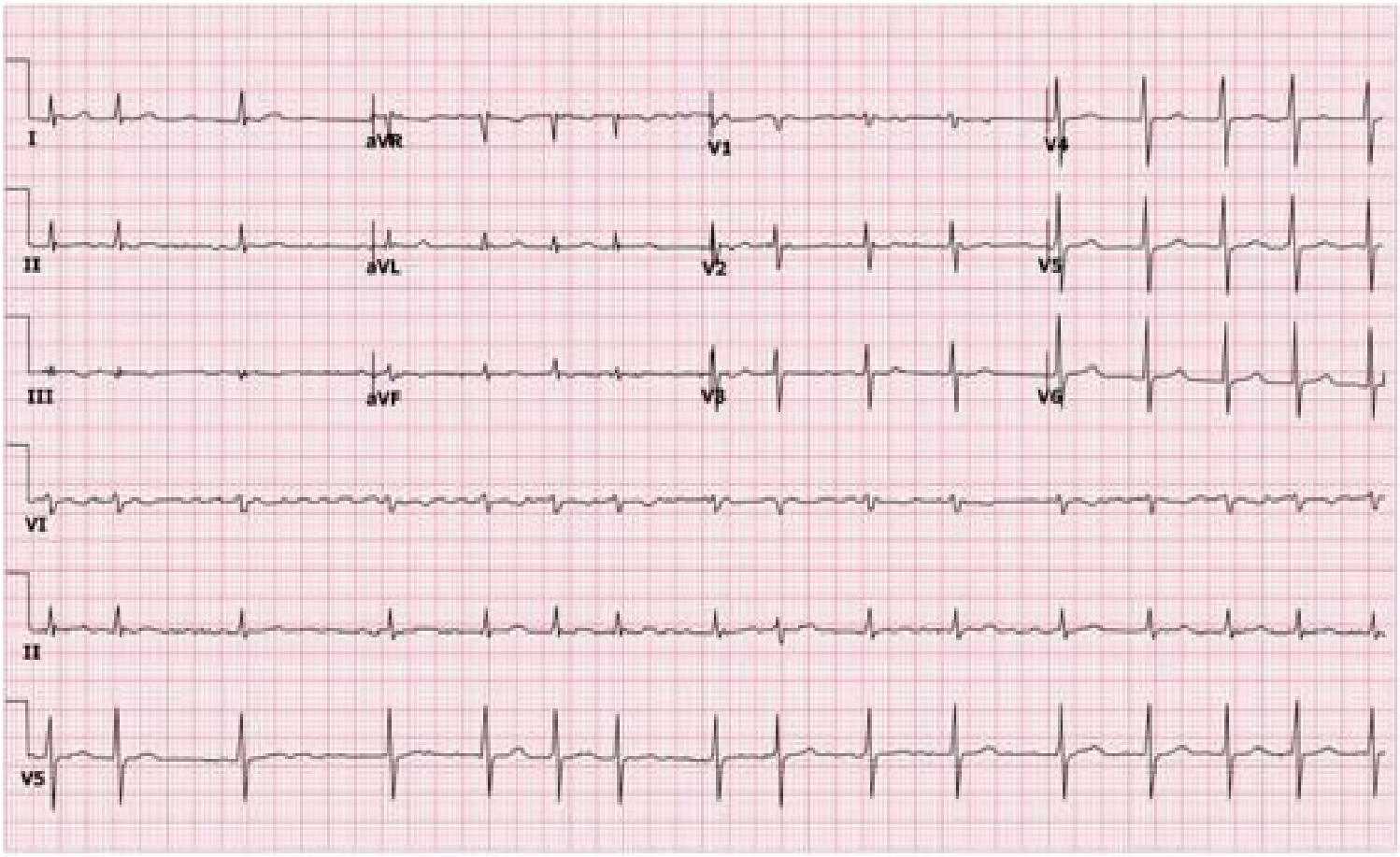
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

7.



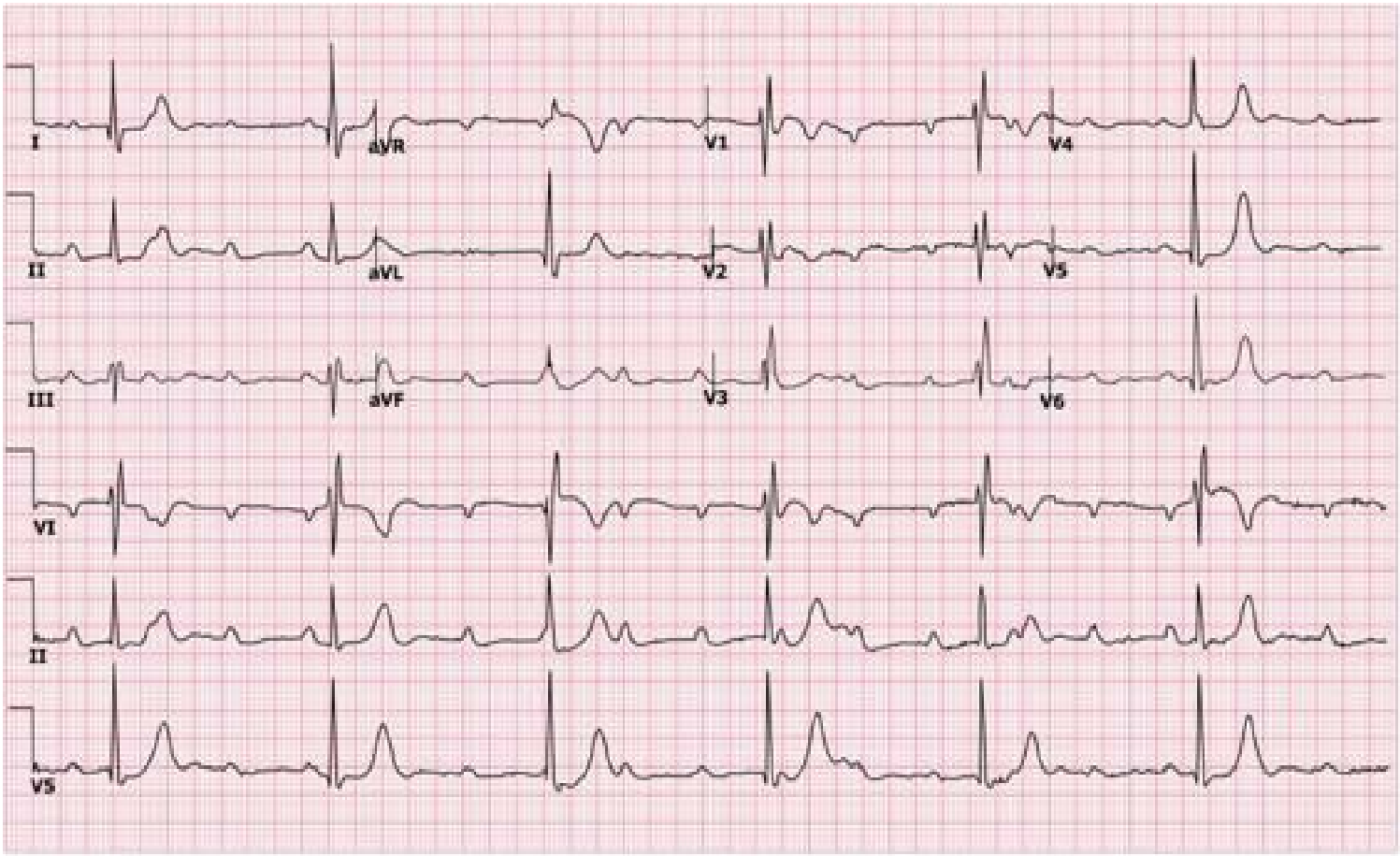
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

8.



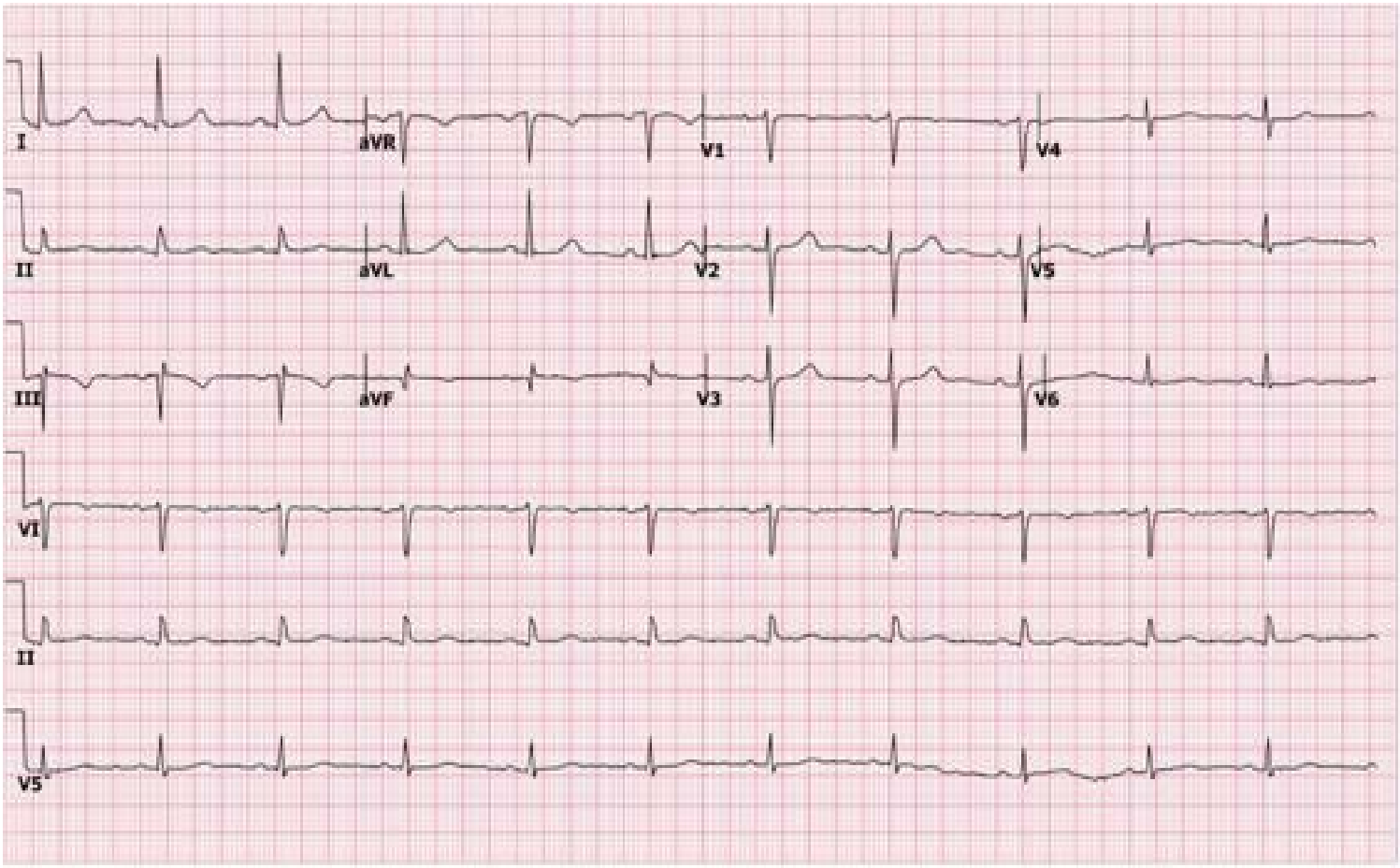
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

9.



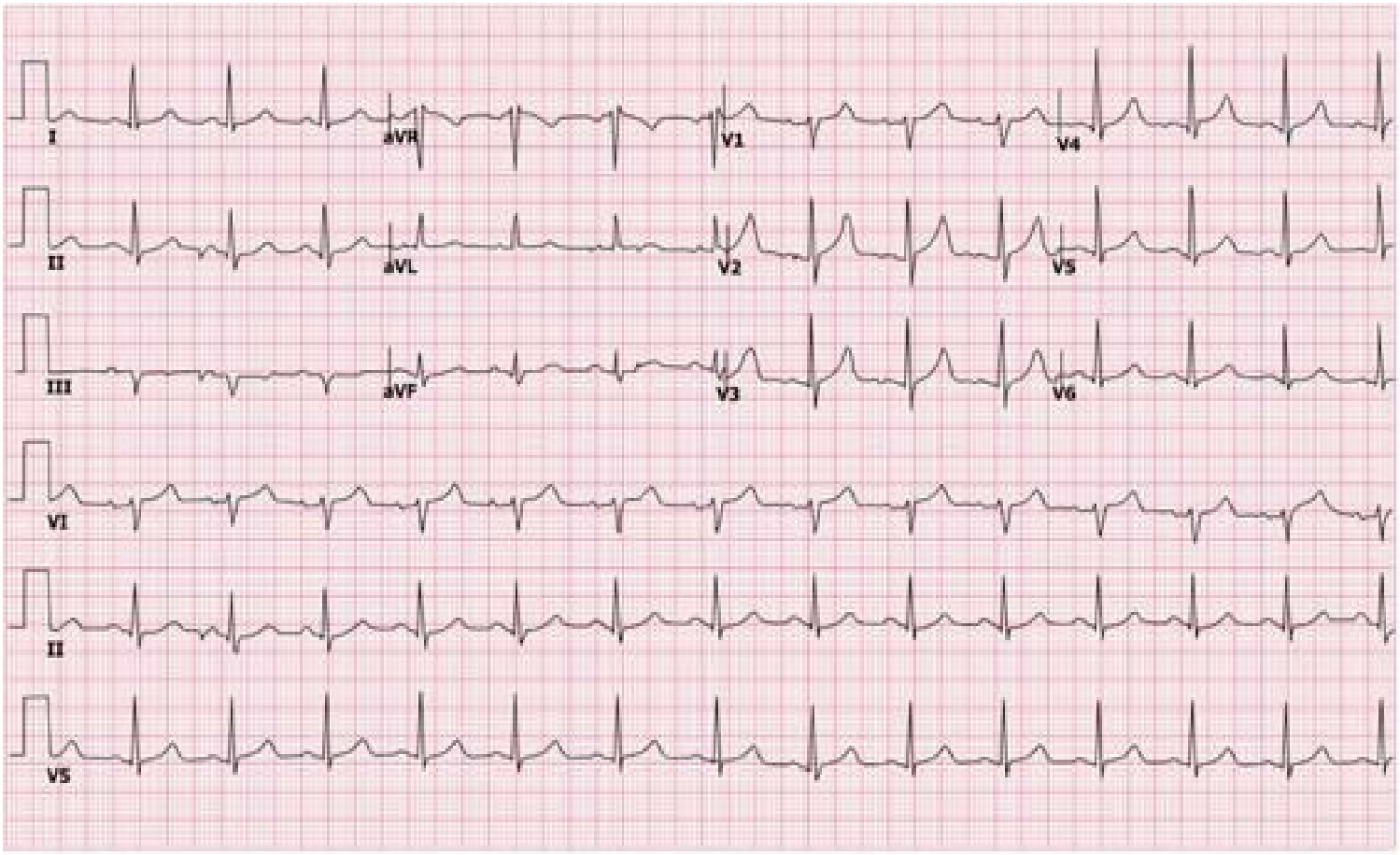
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

10.



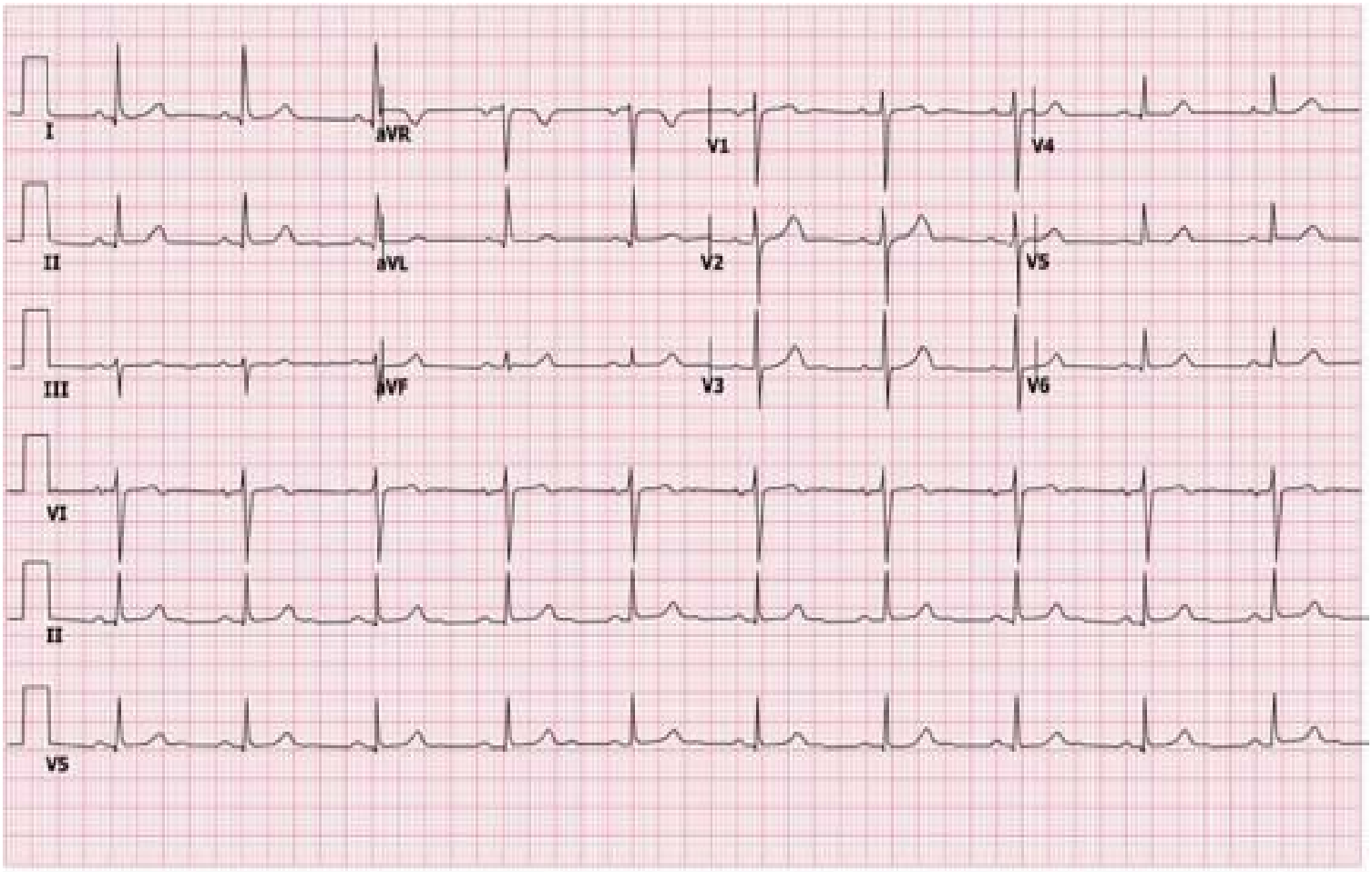
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

11.



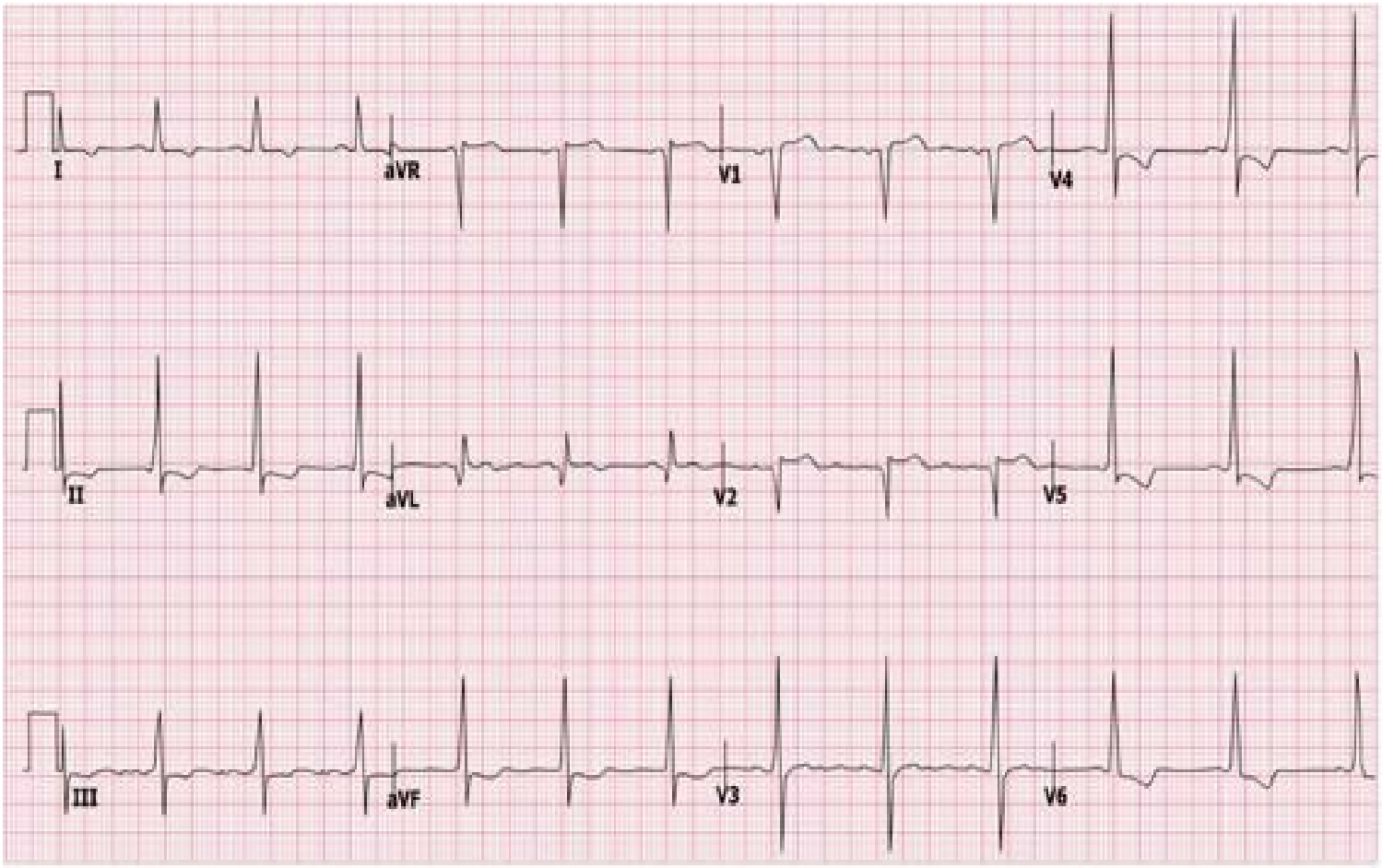
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

12.



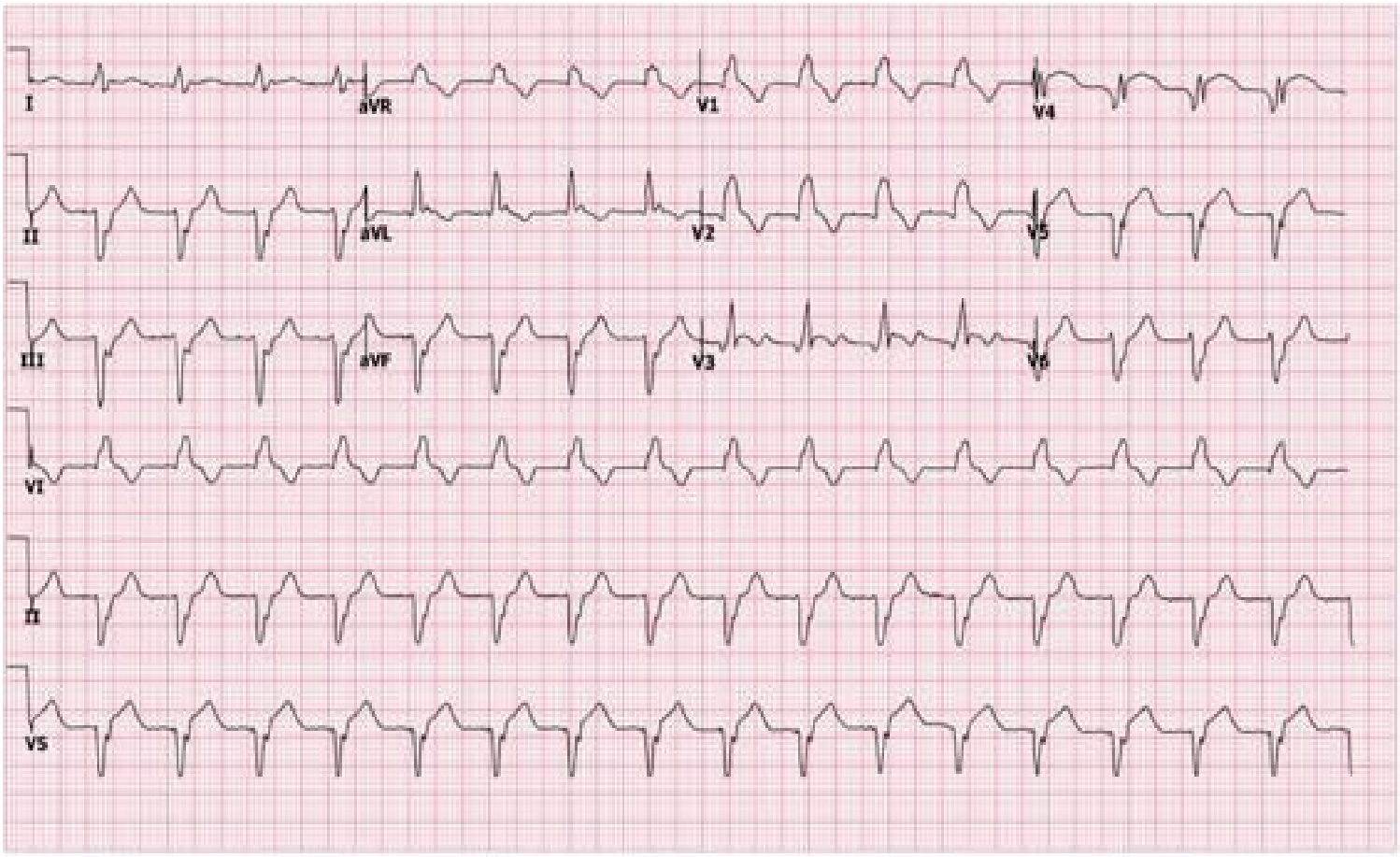
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

13.



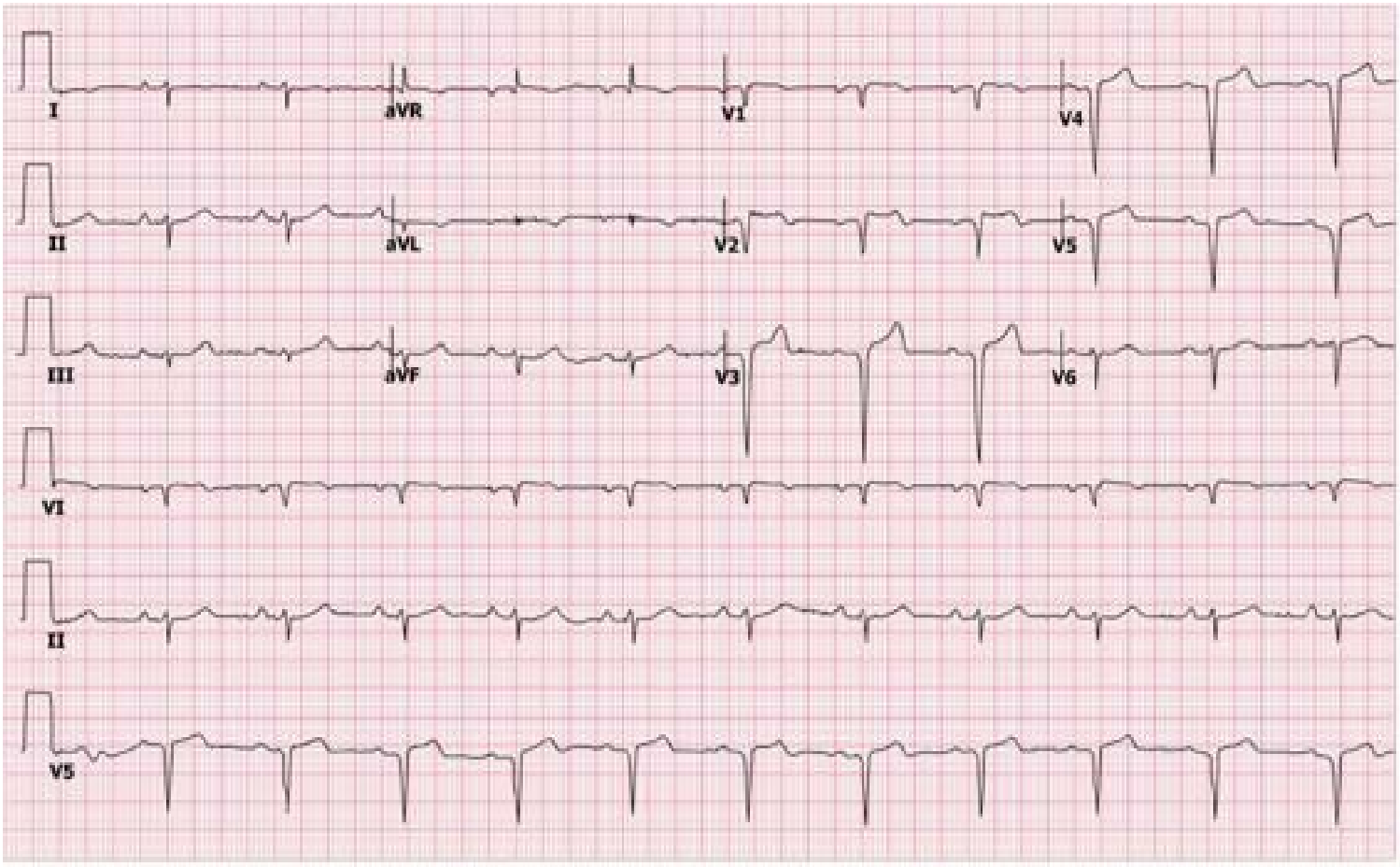
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

14.



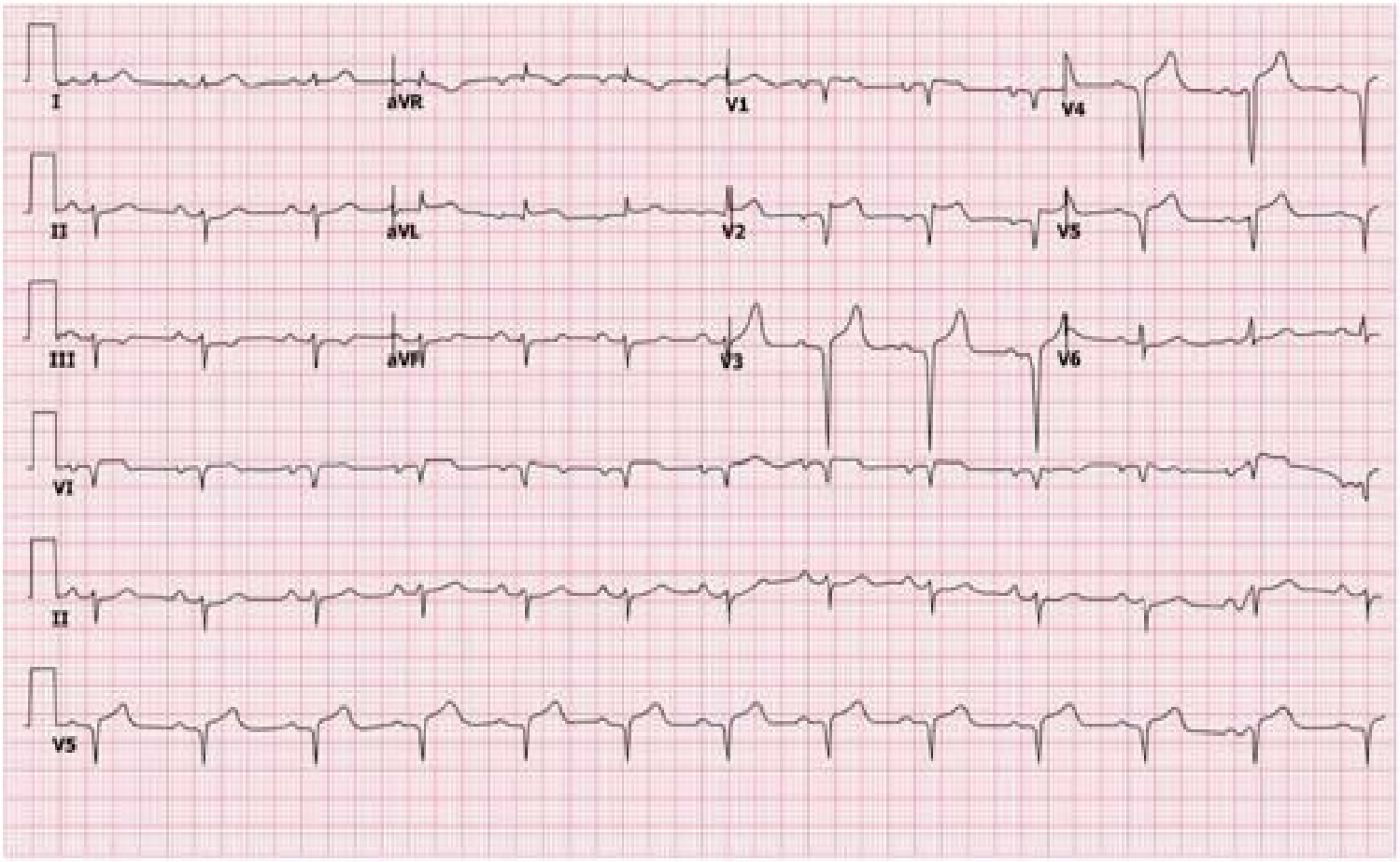
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

15.



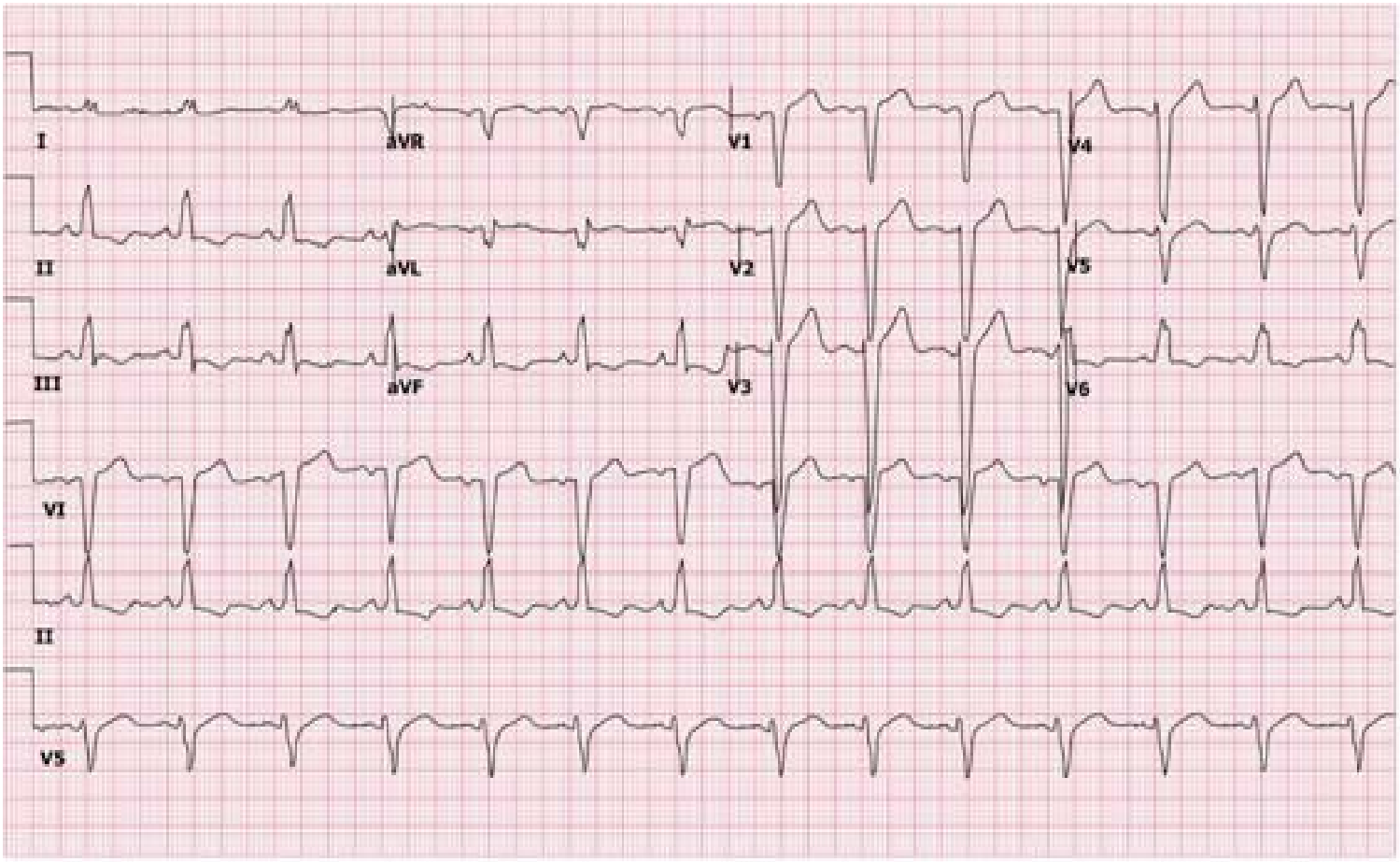
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

16.



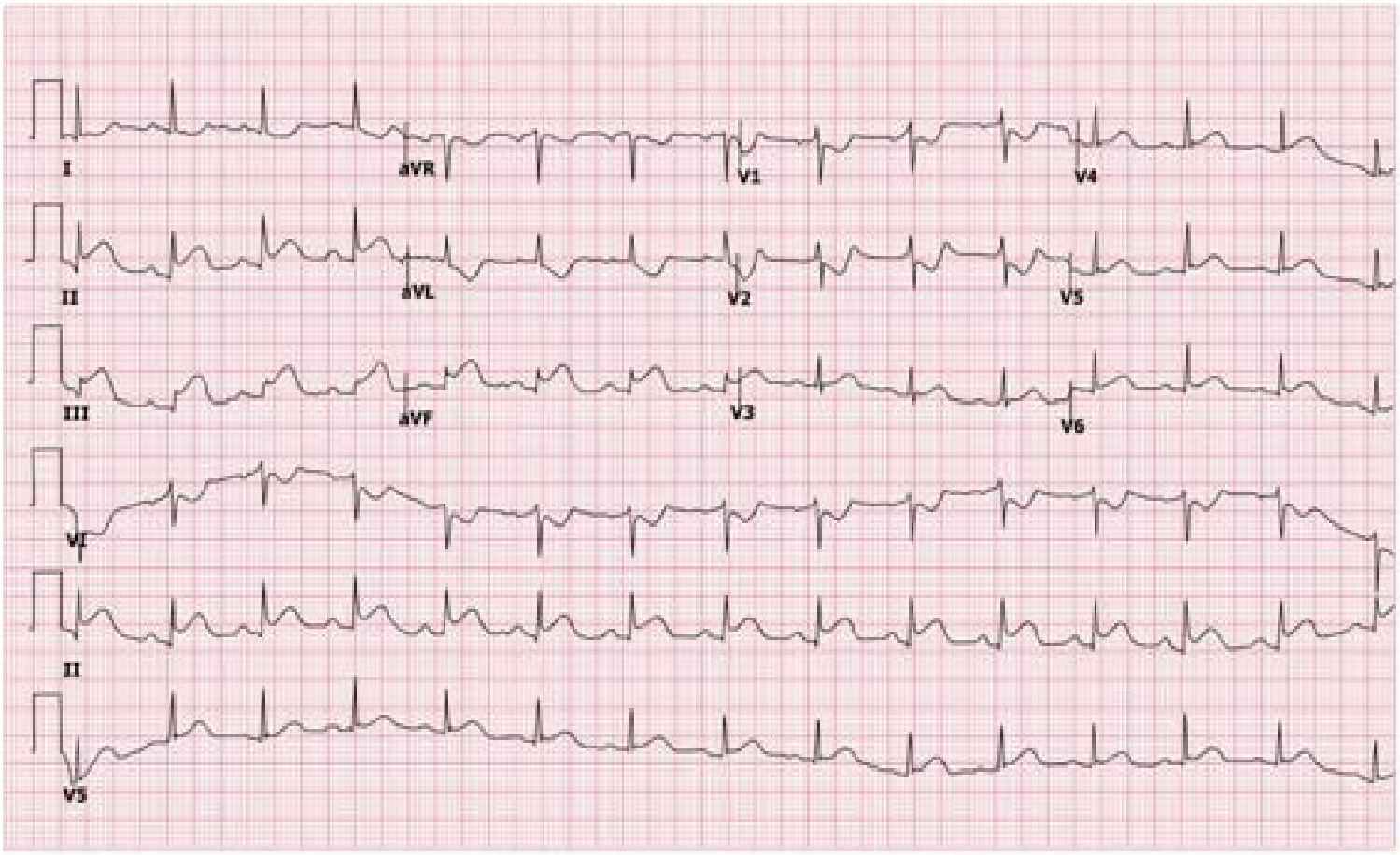
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

17.



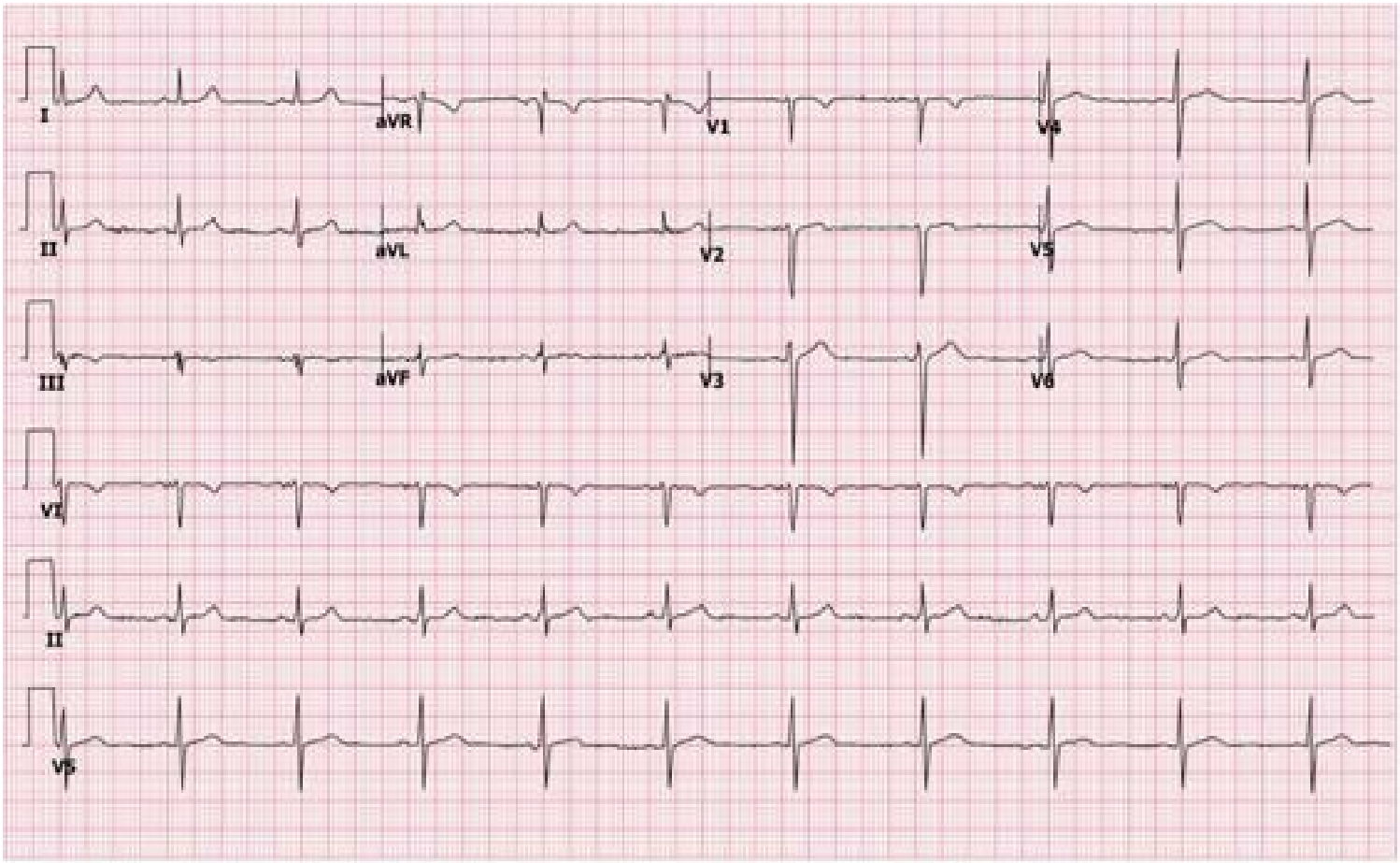
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

18.



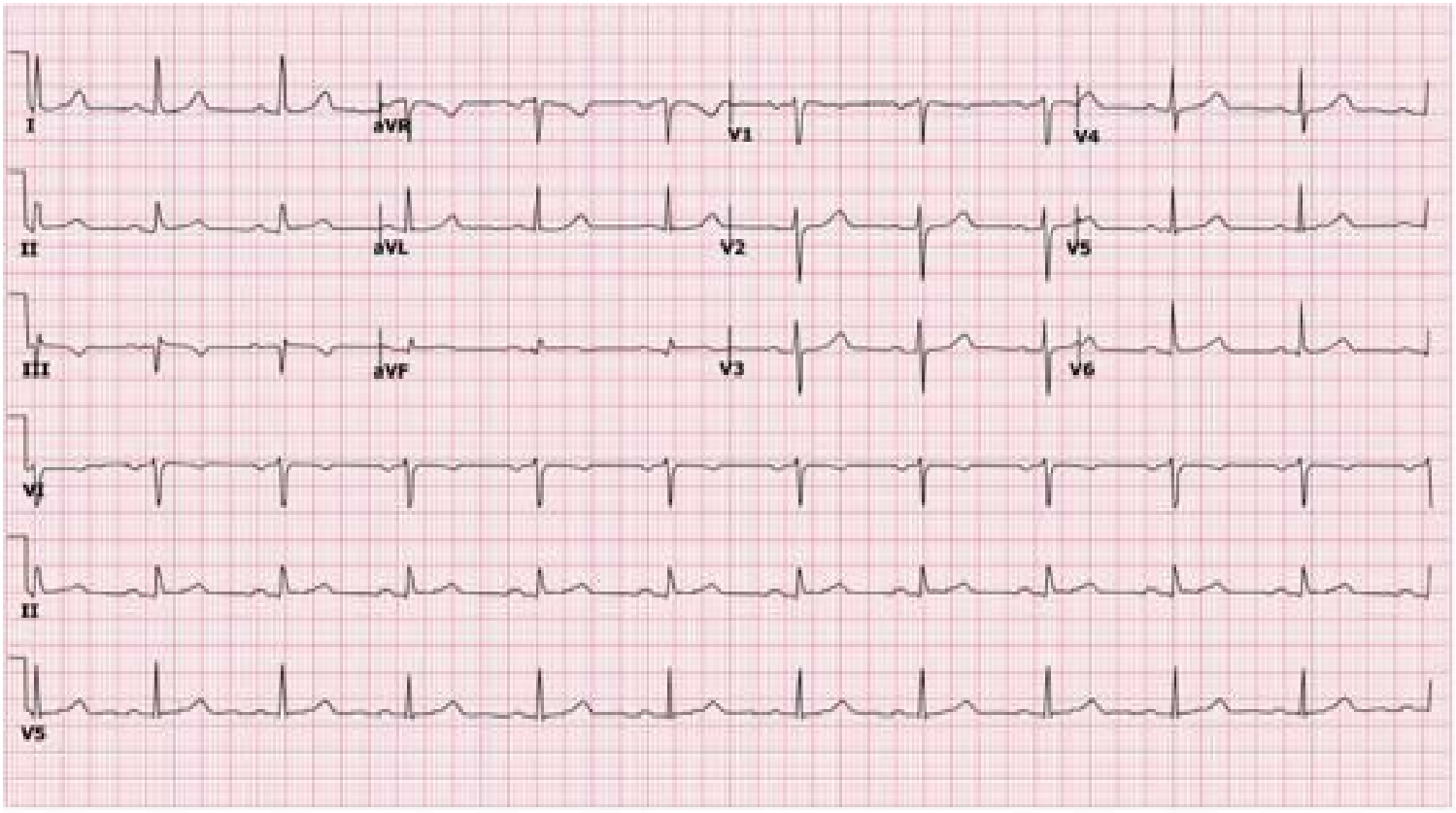
Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

19.



Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

20.



Rate and Rhythm		Enlargement of Atria/ Ventricle	
Axis		Ischemia/ Infarction	
Bundle block/ Hemiblock		Other abnormalities	
Interpretation			

Answer Key
Individual lead interpretation

1. **Rate** __93 BPM ___ **Rhythm** __regular rhythm ___ **P Wave** _present ___**PR Interval** __0.14 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.34 seconds ___ **QTc**__0.439 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __normal sinus rhythm _____

2. **Rate** __63 BPM ___ **Rhythm** __regular rhythm ___ **P Wave** _present ___**PR Interval** __0.22 seconds ___ **QRS Complex** _wide- 0.14 second__ **QT Interval**__0.48 seconds___ **QTc**__0.49 seconds___ **T Wave** _present _ **Other** __possible bundle branch__ **Interpretation** __normal sinus rhythm with bundle branch block _____

3. **Rate** __125 BPM ___ **Rhythm** __regular rhythm ___ **P Wave** _present ___**PR Interval** __0. 16 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0. 26 seconds ___ **QTc**__0.368 seconds ___ **T Wave** _present _ **Other** __tachycardia__ **Interpretation** __sinus

tachycardia_____

4. **Rate** __58 BPM ___ **Rhythm** __regular rhythm ___ **P Wave** _present ___**PR Interval** __0. 20 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0. 42 seconds ___ **QTc**__0.412 seconds ___ **T Wave** _present _ **Other** __presence of Q wave__ **Interpretation** __sinus bradycardia with prominent Q wave_____

5. **Rate** __166 BPM ___ **Rhythm** __regular___ **P Wave** _present but sometimes buried ___**PR Interval** __ 0.14 when present___ **QRS Complex** _narrow __ **QT Interval**__0.22 seconds ___ **QTc** __0.367 seconds ___ **T Wave** _present but distorted with P wave _ **Other** __tachycardia __ **Interpretation** __supraventricular tachycardia _____

6. . **Rate** __65 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _0.16 seconds __ **QT Interval**__0.52 seconds ___ **QTc** __0.536 seconds ___ **T Wave** _present _ **Other** __pacer spikes __ **Interpretation** __ventricular paced rhythm _____

7. . **Rate** __30's ___ **Rhythm** __irregular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _0.06 seconds__ **QT Interval**__0.42 seconds ___ **QTc** __not measured___ **T Wave** _present _ **Other** __present of 2.68 seconds pause __ **Interpretation** __sinus bradycardia with 2.68 second pause_____

8. . **Rate** __75 BPM ___ **Rhythm** __regular___ **P Wave** _present ___**PR Interval** __0.18 seconds ___ **QRS Complex** _0.1 server seconds __ **QT Interval**__0.42 seconds___ **QTc** __0.424 seconds___ **T Wave** _present _ **Other** __presence of pacer spikes __ **Interpretation** __AV paced rhythm _____

9. . **Rate** __68 BPM ___ **Rhythm** __regular___ **P Wave** _present ___**PR Interval** __0.24 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.44 seconds ___ **QTc** __0.469 seconds ___ **T Wave** _present _ **Other** __long PR interval __ **Interpretation** __sinus rhythm with a first degree heart block _____

10. . **Rate** __55 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___**QRS Complex** _narrow __ **QT Interval**__0.44 seconds ___ **QTc** __0.416 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __sinus bradycardia _____

11. . **Rate** __100's BPM ___ **Rhythm** __irregular___ **P Wave** _multiple undulating P waves ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ not measurable ___ **QTc** __not measurable___ **T Wave** _ present but distorted with P waves_ **Other** __none __ **Interpretation** __atrial fibrillation with rapid ventricular response_____

12. **Rate** __115 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.18 seconds ___ **QRS Complex** _0.12seconds __ **QT Interval**__0.38 seconds ___ **QTc** __0.527seconds ___ **T Wave** _present _ **Other** __ __ **Interpretation** __sinus tachycardia with possible bundle branch block _____

13. . **Rate** __90's BPM ___ **Rhythm** __irregular___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _0.12 seconds __ **QT Interval**__0.36 seconds ___ **QTc** __0.398 seconds ___ **T Wave** _present but distorted with P waves_ **Other** __none _ _ **Interpretation** __atrial fibrillation with controlled ventricular response and possible bundle branch block _____

14. . **Rate** __40's BPM ___ **Rhythm** __irregular___ **P Wave** _multiple saw tooth shaped P waves ___**PR Interval** __not measurable___ **QRS Complex** _narrow__ **QT Interval**__ unable to calculate___ **QTc** __not measurable___ **T Wave** _present but deformed by P waves_ **Other** __none __ **Interpretation** __atrial flutter with variable conduction block _____

15. . **Rate** __115 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.28 seconds ___ **QTc** __0.388 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __sinus tachycardia _____

16. . **Rate** __70's BPM ___ **Rhythm** __irregular ___ **P Wave** _present ___**PR Interval** __variable ___ **QRS Complex** _ narrow__ **QT Interval**__0.38 seconds ___ **QTc** __not measured___ **T Wave** _absent in missing waves _ **Other** __missing QRS complex __ **Interpretation** __sinus rhythm with second-degree type I (Mobitz type I) heart block_____

17. . **Rate** __115 BPM in tachycardia and 65BPM in regular rhythm ___ **Rhythm** __regular with tachycardia ___ **P Wave** _present but not always seen in tachycardia ___**PR Interval** __0.20 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.44 seconds in regular rhythm and not measurable during tachycardia ___ **QTc** __0.464 seconds in regular rhythm___ **T Wave** _present _ **Other** __tachycardia turns to regular rhythm __ **Interpretation** __paroxysmal supraventricular tachycardia changing to sinus rhythm_____

18. . **Rate** __45 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.44 seconds ___ **QTc** __0.377 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __sinus bradycardia _____

19. . **Rate** __60's BPM ___ **Rhythm** __irregular ___ **P Wave** _present ___**PR Interval** __variable ___ **QRS Complex** _missing in between__ **QT Interval**__.44 seconds___ **QTc** __not measured___ **T Wave** _missing in between _ **Other** __progressive drop in QRS __ **Interpretation** __sinus rhythm with second-degree type I (Mobitz type I) heart block _____

20. . **Rate** __ 100's BPM ___ **Rhythm** __ irregular___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ not measurable___ **QTc** __not measurable___ **T Wave** _ distorted by P wave_ **Other** __none__ **Interpretation** __atrial fibrillation with rapid ventricular response _____

21. . **Rate** __100's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow except during PVC __ **QT**

Interval __not measurable___ **QTc** __not measurable___ **T Wave** _present but distorted by P wave _ **Other** __none __ **Interpretation** __atrial fibrillation with PVCs_____

22. . **Rate** __50's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple saw toothed P waves ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval** __not measurable___ **QTc** __not measurable___ **T Wave** _ present but distorted by P waves_ **Other** __none __ **Interpretation** __atrial flutter with variable conduction block _____

23. . **Rate** __110 BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__0.36 seconds ___ **QTc** __ 0.445 seconds ___ **T Wave** _present with some distortions because of P waves _ **Other** __none__ **Interpretation** __atrial fibrillation with rapid ventricular response _____ (in order to calculate corrected QT interval, the method of averaging QTc1 and QTc2 was done. RR interval between sixth and seventh ventricular beat from the left was considered for QTc1 and the RR interval between seventh and eighth beat was taken for QTc2.

24. . **Rate** __63 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.24 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.44 seconds ___ **QTc** __0.449 seconds ___ **T Wave** _present _ **Other** __pacer spikes __ **Interpretation** __atrial paced rhythm _____

25. . **Rate** __40's in irregular rhythms and 79 BPM during regular rhythm ___ **Rhythm** __irregular turns to regular ___ **P Wave** _multiple P waves in irregular rhythms and single preceding P wave during regular rhythm ___**PR Interval** __0.20 seconds in regular rhythm without any prolongation ___ **QRS Complex** _narrow except in one QRS complex __ **QT Interval**__ 0.36 seconds ___ **QTc** __not measured___ **T Wave** _missing during some complexes _ **Other** __none __ **Interpretation** __second-degree Type II (Mobitz type II) heart block with a PVC turning to regular sinus rhythm_____

26. . **Rate** __60 BPM ___ **Rhythm** __irregular ___ **P Wave** _absent or inverted ___**PR Interval** __varying ___ **QRS Complex** _0.14 seconds __ **QT Interval**__ 0.54 seconds ___ **QTc** __ 0.54 seconds ___ **T Wave** _in the opposite direction of QRS complex _ **Other** __regular PVCs__ **Interpretation** __junctional rhythm with bundle branch block and bigeminal PVC _____

27. . **Rate** __80's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ 0.44 seconds ___ **QTc** __0.509 seconds ___ **T Wave** _present but sometimes distorted with P waves _ **Other** __one PVC__ **Interpretation** __atrial fibrillation with controlled ventricular response and PVC_____

28. . **Rate** __75 BPM ___ **Rhythm** __irregular at one point___ **P Wave** _present ___**PR Interval** __0.24 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.42 seconds ___ **QTc** __0.47 seconds ___ **T Wave** _present _ **Other** __one PAC __ **Interpretation**

__sinus rhythm with first-degree heart block and one PAC (second beat from the right)_____

29. . **Rate** __90's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple ___**PR Interval** __not measurable ___ **QRS Complex** _narrow__ **QT Interval**__ 0.36 seconds ___ **QTc** __0.466 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __atrial fibrillation with a controlled ventricular response _____

30. . **Rate** __93 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.12 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.32 seconds ___ **QTc** __ 0.413 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __normal sinus rhythm _____

31. . **Rate** __83 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _0.16 seconds __ **QT Interval**__0.44 seconds ___ **QTc** __0.519 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __sinus rhythm with bundle branch block _____

32. . **Rate** __ 115 BPM during tachycardia and 65 BPM during regular rhythm___ **Rhythm** __regular with variable rate ___ **P Wave** _present ___**PR Interval** __0.20 in regular rhythm ___ **QRS Complex** _narrow __ **QT Interval**__ 0.44 seconds in regular rhythm and not measurable in tachycardia ___ **QTc** __0.464 seconds in regular rhythm ___ **T Wave** _present _ **Other** __none__ **Interpretation** __paroxysmal supraventricular tachycardia (PS VT) turns to regular sinus rhythm _____

33. . **Rate** __71 BPM ___ **Rhythm** __regular ___ **P Wave** _present but inverted ___**PR Interval** __0.12 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.40 seconds ___ **QTc** __0.436 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __accelerated junctional rhythm _____

34. . **Rate** __30's BPM ___ **Rhythm** __irregular ___ **P Wave** _absent in one beat ___**PR Interval** __ 0.20 seconds___ **QRS Complex** _narrow __ **QT Interval**__0.44 seconds ___ **QTc** __ 0.367 seconds ___ **T Wave** _present _ **Other** __present of 2.68 seconds pause __ **Interpretation** __sinus bradycardia with 2.68 seconds pause and junctional beat_____

35. . **Rate** __88 BPM ___ **Rhythm** __regular with the one irregular beat ___ **P Wave** _present except in one beat ___**PR Interval** __0.18 seconds ___ **QRS Complex** _narrow (0.08 seconds)__ **QT Interval**__0.40 seconds ___ **QTc** __ 0.485 seconds ___ **T Wave** _present _ **Other** __pacer spike __ **Interpretation** __ventricular paced rhythm _____

36. . **Rate** __40's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple ___**PR Interval** __variable ___ **QRS Complex** _missing in some beats__ **QT Interval**__0.40 seconds ___ **QTc** __not measured ___ **T Wave** _missing in some beats _ **Other** __none __ **Interpretation** __second-degree Type I (Mobitz type I) heart block _____

37. . **Rate** __68 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.24

seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.44 seconds ___ **QTc** __0.469 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __ sinus rhythm with first-degree heart block _____

38. . **Rate** __107 BPM ___ **Rhythm** __regular ___ **P Wave** _present but buried in T wave ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ 0.44 seconds ___ **QTc** __ 0.588 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __sinus tachycardia _____

39. . **Rate** __30's BPM ___ **Rhythm** __irregular ___ **P Wave** _not seen ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__0.54 seconds ___ **QTc** __not measured___ **T Wave** _present _ **Other** __pacer spikes__ **Interpretation** __pacemaker failure to capture _____

40. . **Rate** __75 BPM ___ **Rhythm** __regular ___ **P Wave** _multiple saw toothed ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ not measurable ___ **QTc** __not measurable___ **T Wave** _present but distorted by P waves _ **Other** __none __ **Interpretation** __atrial flutter with a 3 :1 conduction block_____

41. . **Rate** __93 BPM during regular rhythm and 166 BPM during tachycardia ___ **Rhythm** __irregular ___ **P Wave** _present during regular rhythm and not seen during tachycardia ___**PR Interval** __0.18 seconds during regular rhythm ___ **QRS Complex** _narrow __ **QT Interval**__0.36 seconds during regular rhythm___ **QTc** __ 0.443 seconds in regular rhythm___ **T Wave** _present but distorted during tachycardia _ **Other** __one PAC (fifth beat from the left) __ **Interpretation** __sinus rhythm with PAC initiating supraventricular tachycardia _____

42. . **Rate** __50's BPM ___ **Rhythm** __irregular ___ **P Wave** _present ___**PR Interval** __varying ___ **QRS Complex** _ 0.12 seconds__ **QT Interval**__ 0.40 seconds ___ **QTc** __not measured ___ **T Wave** _present _ **Other** __missing QRS complex __ **Interpretation** __second-degree type I (Mobitz type I) heart block_____

43. . **Rate** __68 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.36 seconds ___ **QTc** __ 0.384 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __normal sinus rhythm _____

44. . **Rate** __60's BPM ___ **Rhythm** __irregular___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow__ **QT Interval**__ 0.38 seconds ___ **QTc** __ 0.411 seconds ___ **T Wave** _present but distorted _ **Other** __none __ **Interpretation** __atrial fibrillation _____(even though some saw toothed P waves are seen, it is not considered as atrial flutter because in atrial flutter P waves are mostly of uniform shape).

45. . **Rate** __115 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.14 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.34 seconds ___ **QTc** __0.463

seconds ___ **T Wave** _ present _ **Other** __none __ **Interpretation** __sinus tachycardia _____

46. . **Rate** __34 BPM during bradycardia and 107 BPM during tachycardia ___ **Rhythm** __irregular ___ **P Wave** _inverted and missing in some beats ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__0.52 seconds ___ **QTc** __not measured___ **T Wave** _present _ **Other** __none __ **Interpretation** __junctional rhythm turns to tachycardia (possible tachy-brady syndrome)_____

47. . **Rate** __50's BPM ___ **Rhythm** __irregular ___ **P Wave** _saw toothed ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ not measured___ **QTc** __not measured___ **T Wave** _distorted by P waves _ **Other** __none __ **Interpretation** __atrial flutter with variable conduction block _____

48. **Rate** __50's BPM ___ **Rhythm** __irregular___ **P Wave** _present ___**PR Interval** __0.14 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.32 seconds ___ **QTc** __not measured___ **T Wave** _present_ **Other** __presence of early beats at regular interval__ **Interpretation** __sinus bradycardia with a bigeminal PAC _____

49. . **Rate** __75 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.30 seconds ___ **QRS Complex** _wide (0.16 seconds) __ **QT Interval**__ 0.46 seconds ___ **QTc** __0.514 seconds ___ **T Wave** _present _ **Other** __pacer spikes __ **Interpretation** __atrial paced rhythm with bundle branch block_____

50. . **Rate** __regular rhythm at 79 BPM and tachycardia at 150 BPM___ **Rhythm** __irregular ___ **P Wave** _present except in tachycardia ___**PR Interval** __0.16 seconds during regular rhythm ___ **QRS Complex** _narrow during regular rhythm and wide in tachycardia __ **QT Interval**__ 0.42 seconds in regular rhythm ___ **QTc** __ 0.47 seconds in regular rhythm ___ **T Wave** _ present in regular rhythm_ **Other** __one early beat (fifth beat from the left) and presence of P wave prior to tachycardia __ **Interpretation** __ underlying sinus rhythm with a PAC having supraventricular tachycardia with aberrant conduction (not ventricular tachycardia because of the presence of initiating P wave)_____

51. . **Rate** __80's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__ not measured___ **QTc** __not measured___ **T Wave** _distorted by P waves _ **Other** __none __ **Interpretation** __atrial fibrillation with a controlled ventricular response_____

52. . **Rate** __ 136 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.18 seconds ___ **QRS Complex** _narrow __ **QT Interval**__0.26 seconds ___ **QTc** __ 0.383 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __sinus tachycardia _____

53. . **Rate** __60 BPM ___ **Rhythm** __ regular___ **P Wave** _present ___**PR Interval** __0.16 seconds ___ **QRS Complex** _narrow __ **QT Interval**__ 0.40 seconds ___ **QTc** __ 0.404 seconds ___ **T Wave** _present _ **Other** __none __ **Interpretation** __normal sinus rhythm

_____

54. . **Rate** __62 BPM in regular and 150 BPM in tachycardia___ **Rhythm** __irregular ___ **P Wave** _ present during regular rhythm ___**PR Interval** __0.12 seconds ___ **QRS Complex** _narrow in regular rhythm and wide during tachycardia __ **QT Interval**__ 0.38 seconds during regular rhythm___ **QTc** __0.384 seconds during regular rhythm ___ **T Wave** _present during regular rhythm_ **Other** __presence of fusion beats (4th beat during tachycardia)__ **Interpretation** __sinus rhythm with the short run of VT with a fusion beats in between _____

55. . **Rate** __30's BPM during bradycardia and 68 BPM during regular rhythm___ **Rhythm** __irregular ___ **P Wave** _multiple ___**PR Interval** __variable ___ **QRS Complex** _ narrow _ _ **QT Interval**__0.40 seconds ___ **Q Tc** __not measured___ **T Wave** _present_ **Other** __missing QRS complex __ **Interpretation** __second-degree Type I (Mobitz type I) heart block _____

56. . **Rate** __51 BPM ___ **Rhythm** __regular ___ **P Wave** _present ___**PR Interval** __0.20 seconds ___ **QRS Complex** _wide (0.20 seconds) __ **QT Interval**__0.52 seconds ___ **QTc** __ 0.487 seconds ___ **T Wave** _present_ **Other** __none __ **Interpretation** __sinus bradycardia with a bundle branch block _____

57. . **Rate** __80's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__0.34 seconds ___ **QTc** __ 0.412 seconds ___ **T Wave** _present_ **Other** __none __ **Interpretation** __ atrial fibrillation with controlled ventricular response_____

58. . **Rate** __Paced rhythm at the 71 BPM and tachycardia at 125 BPM___ **Rhythm** __irregular___ **P Wave** _& except tachycardia ___**PR Interval** __constant ___ **QRS Complex** _narrow except tachycardia__ **QT Interval** __not measurable___ **Q Tc** __not measurable___ **T Wave** _seen except in tachycardia _ **Other** __pacer spikes__ **Interpretation** __ventricular pacing rhythm with short run of ventricular tachycardia _____

59. . **Rate** __60 BPM ___ **Rhythm** __regular ___ **P Wave** _present___ **PR Interval** __0.20 seconds ___ **QRS Complex** _0.12 seconds __ **QT Interval**__0.4 server seconds ___ **QTc** __ 0.4 seconds ___ **T Wave** _present_ **Other** __none __ **Interpretation** __sinus rhythm with bundle branch block____

60. . **Rate** __60's BPM ___ **Rhythm** __irregular ___ **P Wave** _multiple and undulating ___**PR Interval** __not measurable ___ **QRS Complex** _narrow __ **QT Interval**__0.40 seconds ___ **QTc** __ 0.431 seconds ___ **T Wave** _present with distortions from P wave _ **Other** __none__ **Interpretation** __atrial fibrillation with controlled ventricular response _____

12 Lead EKG Interpretation

Answer key

1. Rate and Rhythm __sinus rhythm at 75 BPM __ Axis __normal axis__ BBB/ Hemiblock __none __ Enlargement - Atria/ Ventricle ___ not seen ____ Ischemia/ Infarction __none __ Other ___motion artifact in lead V2 and V3__ Interpretation __normal sinus rhythm at 75 beats per minute with some motion artifact ____

2. Rate and Rhythm __ sinus rhythm with a first degree AV block (PR-0.28 seconds) __ Axis __left axis deviation (negative lead aVF and positive lead I) __ BBB/ Hemiblock __ left bundle branch block (negative V1, positive lead I and V6 with a wide QRS complex) __Enlargement - Atria/ Ventricle___ left atrial enlargement (notched and wide P in lead III, aVF and V1) ____ Ischemia/ Infarction __ none __ Other ___none __ Interpretation __left bundle branch block with a first degree AV block and left axis deviation ____

3. Rate and Rhythm __ sinus rhythm at 68 BPM __ Axis __left axis deviation __ BBB/ Hemiblock __ left anterior fascicle block (left axis, qR pattern in lead I and aVL, qS pattern in inferior leads) __Enlargement - Atria/ Ventricle ___ left ventricular hypertrophy (R aVL+ S V3 = 36)____ Ischemia/ Infarction __ infero-lateral T wave inversion __ Other ___none __ Interpretation __sinus rhythm with left anterior fascicular block and LVH with Infero lateral ischemia ____

4. Rate and Rhythm __ atrial fibrillation at 60's BPM __ Axis __right axis deviation (negative lead I and positive aVF) __ BBB/ Hemiblock __ none __Enlargement - Atria/ Ventricle ___ none ____ Ischemia/ Infarction __ anterior infarct (presence of deep S waves in anterior leads) __ Other ___none __ Interpretation __atrial fibrillation with right axis deviation and old anterior infarct ____

5. Rate and Rhythm __ sinus rhythm at 83 BPM __ Axis __normal axis __ BBB/ Hemiblock__ none __Enlargement - Atria/ Ventricle ___ none ____ Ischemia/ Infarction __ inferior ST segment elevation with possible old anterior infarct (prominent S waves in precordial leads)__ Other ___ST segment depression in V1 and V2 (mirror image of inferior infarction)__ Interpretation __inferior STEMI with the possible old antero-septal infarct ____

6. Rate and Rhythm__sinus rhythm at 75 BPM__ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___none ____ Ischemia/ Infarction__up to 1 mm ST segment elevation in lateral leads __ Other ___none __ Interpretation __lateral STEMI ____

7. Rate and Rhythm __atrial pacing at 63 BPM __ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___none ____ Ischemia/ Infarction __not seen__ Other ___none __ Interpretation __atrial paced rhythm (Note that the pacer spike is not visible in every leads and this should not make the interpreter think that pacemaker is not

working. Since all the leads are capturing at the same time, if you see a pacer spike in one lead which means it is a pacer rhythm.)

8. Rate and Rhythm__atrial fibrillation at 90's BPM __ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___not seen ____ Ischemia/ Infarction__not seen __ Other ___saw toothed P waves in lead V1 (Crista Terminalis effect as mentioned in chapter 6)__ Interpretation __atrial fibrillation with a controlled ventricular response____

9.Rate and Rhythm__atrial 100 BPM and ventricular 36 BPM __ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___none ____ Ischemia/ Infarction__not seen __ Other ___atrioventricular dissociation __ Interpretation __third degree heart block ____

10. Rate and Rhythm__sinus rhythm with 65 BPM __ Axis __left axis deviation__ BBB/ Hemiblock__ none __Enlargement - Atria/ Ventricle___not seen ____ Ischemia/ Infarction__inferior Q waves and T wave inversion in lead III and aVF __ Other ___none __ Interpretation __sinus rhythm with possible old inferior infarct and possible inferior ischemia ____

11. Rate and Rhythm__ sinus rhythm at 79 BPM__ Axis __normal axis __ BBB/ Hemiblock__not seen __Enlargement - Atria/ Ventricle___none ____ Ischemia/ Infarction__isolated deep S wave in lead III __ Other ___ QTc 0.485 seconds __ Interpretation __sinus rhythm with the long QT interval ____

12. Rate and Rhythm__ sinus rhythm at 63 bpm __ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___not seen ____ Ischemia/ Infarction__ up to 1 mm Q waves in lateral leads __ Other ___none __ Interpretation __sinus rhythm with possible old lateral infarct ____

1 3 . Rate and Rhythm__sinus rhythm at 75 BPM__ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___left ventricular hypertrophy with strain pattern (tall R waves with characteristic ST depression in precordial leads) ____ Ischemia/ Infarction__inferolateral T wave inversion (subendocardial ischemia from LVH)__ Other ___ none__ Interpretation __sinus rhythm with the left ventricular hypertrophy ____

14. Rate and Rhythm__sinus rhythm at 100 BPM__ Axis __left axis deviation __ BBB/ Hemiblock__right bundle branch block and left anterior fascicular block (bifascicular block) __Enlargement - Atria/ Ventricle___none ____ Ischemia/ Infarction__not seen __ Other ___ none __ Interpretation __sinus rhythm with bifascicular block ____

15. Rate and Rhythm__sinus rhythm at 68 BPM __ Axis __extreme right axis deviation __ BBB/ Hemiblock__ none __Enlargement - Atria/ Ventricle___not seen ____ Ischemia/ Infarction__ anterior ST segment elevation in lead V2 in V3, old septal and anterolateral infarct (deep S waves) __ Other ___none __ Interpretation __sinus rhythm with the extreme

right axis deviation and anterior STEMI with the old septal anterolateral infarct ____

16. Rate and Rhythm__sinus rhythm at 75 BPM __ Axis __left axis deviation __ BBB/ Hemiblock__left anterior fascicular block __Enlargement - Atria/ Ventricle___left ventricular hypertrophy ____ Ischemia/ Infarction__anterior ST segment elevation __ Other ___none __ Interpretation __sinus rhythm with the left anterior fascicle block and anterior STEMI ____

17. Rate and Rhythm__sinus rhythm at the 75 BPM__ Axis __normal axis __ BBB/ Hemiblock__left branch block __Enlargement - Atria/ Ventricle___left ventricular hypertrophy ____ Ischemia/ Infarction__not assessed__ Other ___ none __ Interpretation __sinus rhythm with left bundle branch block and left ventricular hypertrophy ____

18. Rate and Rhythm__sinus rhythm at 83 BPM __ Axis __normal axis __ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___none ____ Ischemia/ Infarction__ inferior STEMI __ Other ___ none __ Interpretation __ sinus rhythm with the inferior STEMI ____

19. Rate and Rhythm__sinus rhythm at 68 BPM __ Axis __left axis deviation__ BBB/ Hemiblock__none __Enlargement - Atria/ Ventricle___ left ventricular hypertrophy ____ Ischemia/ Infarction__ old anterior infarct __ Other ___none __ Interpretation __sinus rhythm with left axis deviation and possible old anterior infarct ____

20. Rate and Rhythm__sinus rhythm at the 65 BPM __ Axis __normal axis __ BBB/ Hemiblock__ none__Enlargement - Atria/ Ventricle ___ none ____ Ischemia/ Infarction __ inferior Q waves__ Other ___ none __ Interpretation __normal sinus rhythm with the possible old inferior infarct ____

Appendix A

Summary of 12 lead EKG Interpretation

Rate and rhythm

- Six second method/ counting small box method
- Presence of PQRST and its characteristics
- Look for intervals

Axis

- Look at Lead I and aVF (consider entire QRS complex) for axis in vertical plane
  - I and aVF positive – **Normal axis**
  - I Negative and aVF positive- **Right axis**
  - I positive and aVF Negative- **Left axis**
- Look at V1, V2 and V5, V6 for axis in horizontal plane
  - Positive V1 and V2- **Anterior axis**
  - Positive V5 and V6- **Posterior axis**

Bundle Branch Block

- For BBB look at Lead I, V1 and V6 (Only last half of QRS complex)
  - Negative Lead I and V6 and Positive V1- RBBB
  - Negative Lead V1 and Positive Lead I and V6(R or R')- LBBB
  - BBB require QRS duration greater than 0.12 sec
- For Hemiblock, look at lead I and aVF
  - LAHB
    - Left axis deviation (Lead I positive and aVF negative)
    - qR complex in the lateral limb leads (I and aVL)
    - rS complex in the inferior leads (II, III and aVF)
    - Delayed intrinsicoid deflection (time for R wave peak) in aVL>0.045 sec
    - Do not diagnose LAHB in presence of Inferior infarct (Prominent Q in II, III and aVF)
  - LPHB
    - Lead I Negative and aVF positive (Right axis deviation)
    - rS pattern in I and aVL
    - Tall R waves in II, III and aVF (goes with right axis)
    - Looks similar to S1Q3T3 pattern in pulmonary embolus

Summary of 12 lead EKG Interpretation- Cont..

Chamber enlargement

- For Right atrial enlargement
  - Narrow and tall P wave in Lead II and V1 (P Pulmonale)
- For left atrial enlargement
  - Wide P wave with notching in Lead III, aVF and V1
- For right ventricular hypertrophy
  - Tall R waves in V1, V2 and deep S waves in V5, V6
  - Right axis deviation (Negative lead I and positive aVF)
- For Left ventricular hypertrophy
  - Left axis deviation (Positive Lead I and negative aVF)
  - Down sloping ST and Inverted T (strain pattern) in lateral leads
  - R in aVL + S in V3 > 28 mm in men and > 20mm in women

Ischemia or infarction

- For ischemia -ST segment depression and T wave inversion in
  - Lead I, aVL, V5 and V6 – Lateral wall (Left circumflex territory)
  - Lead II, III and aVF – Inferior leads (RCA territory)
  - Lead V1, V2, V3 and V4 – Anterior wall (LAD territory)
- For acute Myocardial infarction
  - St segment elevation in the target area with ST segment depression and T inversion in the opposite area
- For old myocardial infarction
  - Presence of large Q waves (at least >1mV) in target areas

Other abnormalities

- For Pulmonary embolism
  - Prominent S in Lead I, Q and inverted T in III (S1Q3T3)
  - Right ventricular strain pattern (ST depression in V1- V3)
  - Sinus tachycardia
  - New incomplete RBBB

Summary of 12 lead EKG Interpretation- Cont..

- For Hyperkalemia (depending on serum level)
  - Tall peak T waves
  - ST segment depression
  - Various bundle branch block
  - Severe bradycardia with AV block
  - V tach/ fib
- Pericarditis
  - PR segment depression
  - Generalized ST segment elevation
- For Hypocalcemia
  - Prolonged QTc
  - Flat or inverted T wave
  - Prolonged ST segment without increase in T wave duration

For hypercalcemia

  - Short QTc
  - PR segment prolongation

For hypomagnesemia

  - Peak T wave
  - Prominent T wave
  - Prolonged QRS
  - ST depression
  - Polymorphic Ventricular arrhythmia
- For Pericardial effusion or cardiac tamponade
  - Low voltage EKG
  - Electrical alternance (beat to beat change in amplitude)

Table of Contents