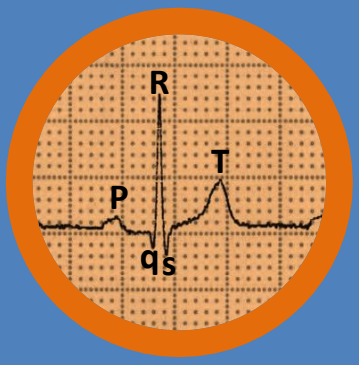

ECG Review : Cardiac Electrophysiology and Some momentous articles

Series 5

(eBook for Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

MD (MEDICINE), FCGP (IND), FIAMS (MEDICINE), FICP , FICCMD

O Heart
Being Conscious of Thou 'Electrophysiology'
Opens the Gateway to ECG 'Phraseology' ...

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Knowledge and skill in the field of electrocardiography are constantly changing with the new researches and understanding.

With humble words I wish to say that everything of 'Cardiac Electrophysiology' is not possible to be covered within this book. Herein I am putting only some important points related to the subject. It is only a step toward the vast ocean of knowledge. Some momentous articles are also enclosed in this book. I may be excused for any error or omission.

With thanks and regards


DEDICATED
TO ALL THE
FELLOW COLLEAGUES

Index

CARDIAC ELECTROPHYSIOLOGY

ECG

CARDIAC ELECTROPHYSIOLOGY

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OUTLINE

Introduction

- ☐ **Pacemaker cells** – SA node (other subsidiary pacemakers AV node and purkinjee fibers but at a slower rate).
- ☐ **Non-Pacemaker cells** – contractile unit (atrial and ventricular myocytes)

The Genesis of Action Potential from SA Node and its Conduction to the Ventricles

SA Node and its Action Potential

Spread of Impulse to Adjacent Atrial Myocytes through 'Transitional Zone'

AV Nodal delay (0.09 second)

Rapid Transmission Through the Ventricular Purkinje System
(At velocity of 1.5-4 meters /second)

Ventricular Transmission

Phase Wise Classification of Ventricular Action Potential

- Depolarization INa (Phase 0)
- Repolarization Ito Ica Ikr Iks (Phase 1, 2 and 3 respectively)
- Restoration to the resting state by Na^+-K^+ ATPase Pump and IKi (Phase 4)

Cardiac Action Potential occurring at Different Level (In brief)

The Mechanism of Excitation-Contraction Coupling of the Cardiac Muscle

Concluding remark

References

CARDIAC ELECTROPHYSIOLOGY

A Narrative Review

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The secrecy of cardiac events gets revealed with the understanding of its electrophysiology - a magnificent planning to uncover the truth is needed. The fundamental unit of this play-route smiles through what is known as 'Action Potential'. The Cardiac action potential initiates brief changes in voltage across cardiac cell membrane – it is brought about by the fluxes of ions through ion channels. All these electrocardiac events are well organised sailing the boat of cardiac life smoothly, without which the life would not be possible.

1. Introduction

The Cardiac Action Potential is a series of brief changes in voltage across the cardiac cell membrane, brought about by fluxes of ions through ion channels.

There are two groups of cells contributing to the genesis of action potential :

- **Pacemaker cells** – SA node (other subsidiary pacemakers AV node and purkinjee fibers but at a slower rate)
- **Non-Pacemaker cells** – contractile unit (atrial and ventricular myocytes)

Accordingly , there are two sets of action potentials for the purpose :

(1) **Pacemaker Potential** (Slow Action Potential) : There is an automatic slow depolarization of the pacemaker cells , which is capable of reaching to its threshold level - so as to generate slow upstroke. This occurs due to the influx of **Ca^{++}** ions inside.

(2) **Non-Pacemaker Potential** (Fast Action Potential) : This potential seen in contractile cells (atria and ventricles) that are backed up by rapid depolarization due to the opening of fast **Na^{+}**channels.

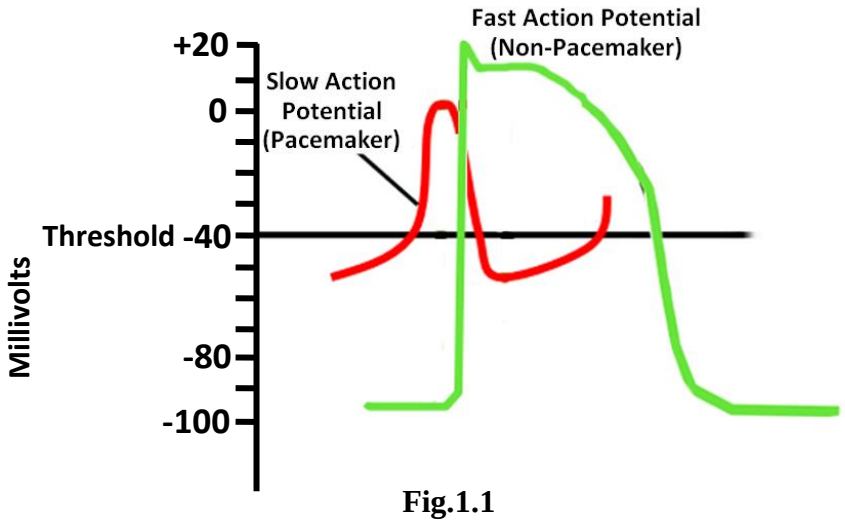


Fig.1.1

2. The genesis of Action Potential from SA node and its conduction to the ventricles

There is well organised cardiac system that causes the automatic generation of action potential from the SA node, which is located in the wall of the right atrium, laterally to the entrance of the superior vena cava, in a region called the sinus venarum (hence termed as sinoatrial node)

Further steps: All cardiac cells are electrically linked to each other through gap junctions, allowing the action potential to pass from one cell to the next.

- The initial action potential generated from the SA node passes to the atria evoking its action potential resulting in its contraction.
- Through the conducting pathway (AV node – HIS-Purkinje system) the impulse passes onwards to the ventricles.
- The ventricular myocytes evoke its action potential causing its contraction.

3. SA Node and its Action Potential

The SA node exhibits **automaticity** – these tissues don't need an external trigger to undergo depolarization. This can be explained by the fact that **the initial potential of this nodal tissues is more negative compared to the RMP of the contractile units of a myocytes**. Thus, this allows Na^+ channels to be automatically activated in a slowly rising semi-curve manner.

Its electrophysiology is summarised as below:

- The spontaneous depolarization of the pacemaker potential during **phase 4** imparts the SA node its auto-rhythmicty. The presence of the so called pacemaker current generated by HCN (Hyperpolarization – activated cyclic Nucleotide gated) channels which are abundant over SA Node and are responsible for the flow of cation – Na^+ + K^+ (mainly Na^+) into the cell. Due to the unusual property of being activated by low negative membrane potential , the movement of ions through these channels is known as **funny current** (If).
- The upstroke of action potential **phase 0** is created by a slowly increase in Ca^{++} conductance through L-type Calcium channels instead of by fast Na^+ currents.
- **Phase 1 and 2 are absent** in the SA node action potential (here there is no question of myocontraction).
- **Phase 3** of repolarization is caused by increase in K^+ conductance, causing an outward flow of K^+ ions. Once the cell is completely repolarized at about -60 mV , the cycle gets spontaneously repeated.

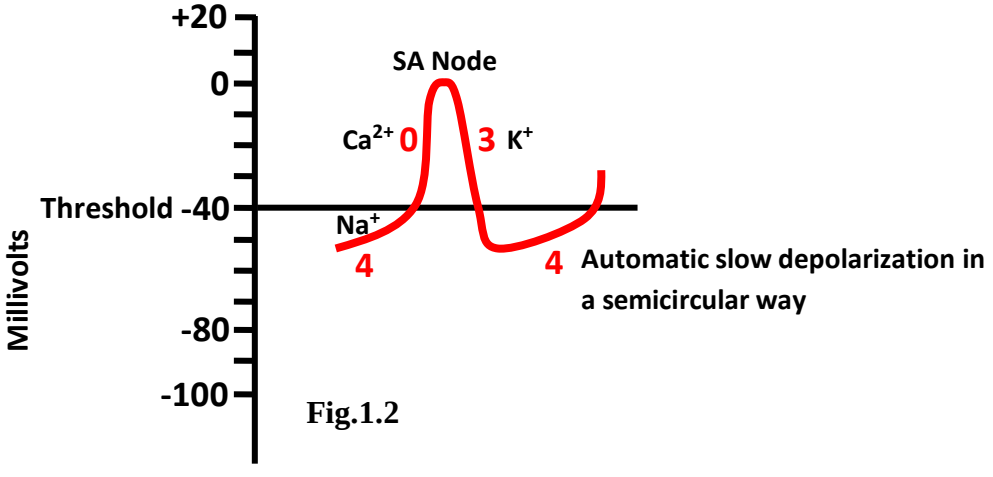


Fig.1.2

4. Spread of impulse to adjacent atrial myocytes to transitional Zone

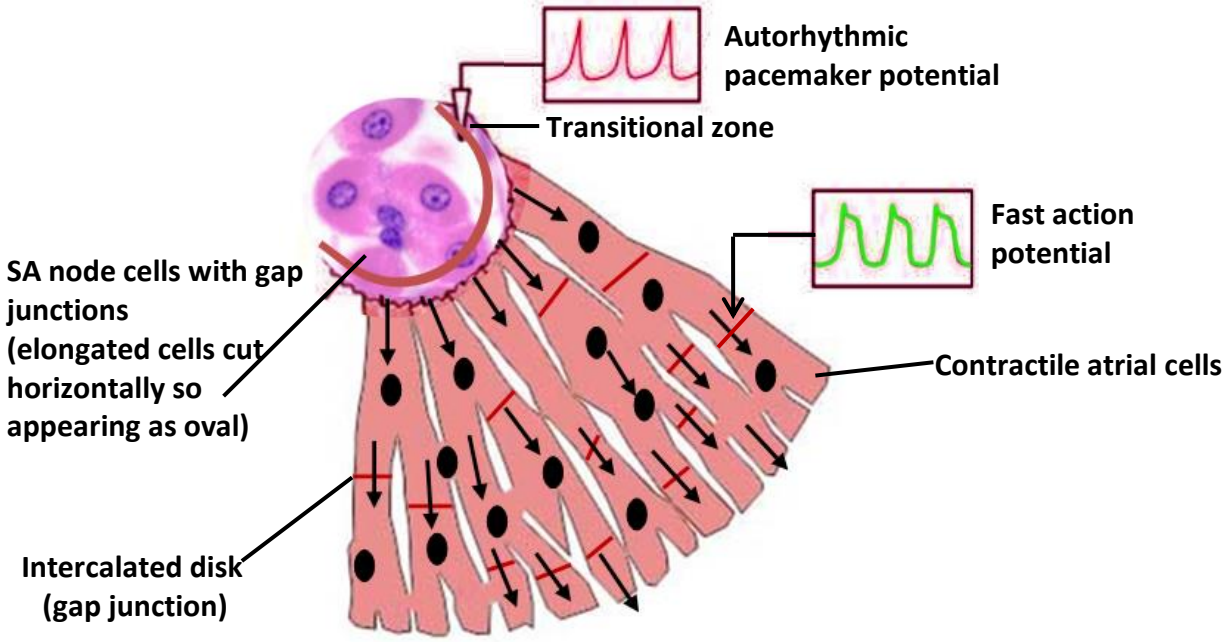


Fig.1.3

The cellular architecture of the SA node:
The main cellular cluster is consisting of elongated cells densely packed within, having its contact with each other , which are also making its connection with the nearby atrial myocardium through the interconnecting collar of **'transitional' cells** (as illustrated in the above sketch). Numerous autonomic ganglia and nerves are found enclosed inside. The SA node is mainly under the influence of vagus nerve and increase in vagal tone slows the heart rate; a decrease in vagal tone accelerates the heart rate. The pacemaker cells are sensitive to stretch by pressure volume, as well
The cytoarchitecture of this transitional zone makes contact with atrial myocytes and this is essential for smooth propagation of SA node action potential through its geometrical arrangements , with sub-bifurcation channels to maintain the dynamic flow of the impulse onwards to the atria.

5. AV Nodal delay

AV node is situated over the posterior wall of the right atrium, just behind tricuspid valve. There is a momentary delay of the impulse in the AV nodal system, **due to the diminished number of gap junctions between successive cells and thus, there is a comparative resistance to the ongoing conduction**. There is a delay of almost 0.09 second in the AV node.

Gap Junctions : These are constituted with the connexin proteins that form gaps through which the fluxes of ions may pass across the cardiac cell membrane. Since potassium is highest intracellularly , these ions may pass through these gaps. The increased potassium in the nearby cells causes the membrane potential to be increased slightly towards negativity , and thus there is an activation of fast sodium channels of contractile cellular units.

6. Rapid transmission through the ventricular Purkinje system

Purkinje fibers are very large fibers, even longer than the ventricular muscle fibers. There is a speedy conduction of the action potential at velocity of 1.5 to 4 m/s. This rapid propagation transmits the electrical potential almost instantaneously to the entire ventricular muscular system. This rapid transmission is offered to the system by a very high level of permeability of the intercalated disc gaps.

7. Ventricular transmission

The standard model used to demonstrate cardiac action potential is that of the ventricular myocytes.

The contractile unit of ventricular myocytes lies in between endocardium and epicardium. The current of **depolarization** is spread through endocardial cells with an inner rich network of Purkinje fibers adjoining with myocytes forming **Purkinje-myoctes unit**. This allows the propagation of impulses from endocardium to epicardium and from apex to the base (due to the spiral arrangements of the ventricular muscle the impulse from the Purkinje fibers takes the spiral conduction from apex to the base). **This is to mention here that the depolarization makes the entire myocardium somewhat isoelectric but with reverse polarity on either side so that a harmonized myocardial contraction may set in without its trembling**. The **repolarization** journey starts towards the epicardium firstly by a transient opening of a specialized channel known as calcium-insensitive **transient outward** current (Ito) . This Ito current opens L-type current ('L' stands for long lasting referring to the length of activation) and this further opening of Ca^{2+} channel is responsible for the exchange in between Ca^{2+} and K^{+} to bring 'excitation-contraction coupling' operation resulting in harmonized myocardial contraction. Once the myocardial contraction gets completed , there is a further sequential opening of different K^{+} channels in succession and ultimately restoration of the myocytes to the previous polarized state by Na-K ATPase pump and inward rectifier channels (IKi).

The concept of ionic channels is summarized as below :

- **Fast Na^{+} Channel** brings positive charge inside the cell and depolarizes the cardiac membrane , the threshold potential for this purpose is at -70 mV.
- **Ito channel** (transient outward current) The opening and closing of the channel rapidly allows a transient flow of potassium ions out of the cell , making the membrane potential slightly more negative .
- **Ica^{2+} Channel** Ito channels open the Ica^{2+} channels to facilitate the exchange in between calcium ions and potassium ions.
- The main potassium channels involved during repolarization are the rapid rectifiers **(IKr)** , slow rectifiers **(IKs)** and the inward rectifiers **(IKi)**.
  IKr and IKs channels are activated from the beginning of repolarization – IKs act upto the phase 3 of repolarization and IKi remain active upto the last phase of repolarization to push the remaining potassium ions inside the cardiac cells.

This is to be mentioned here that depolarization current is only concerned with the reversal of the polarized state resulting in negatively charged surface outside the cardiac membrane and positively charged inside. This is possible by the inward current facilitated through Na^+ conductance pushing the sodium ions inside the membrane. That's why , the endocardial cells are rich in INa showing 'dome pattern'. **This whole happens due to the transmission through the syncytial ventricular mass and only INa channels are opened during depolarization**.

The repolarization is a journey proceeding towards normal polarity. That's why , **it is accordingly equipped with genetically determined ion channels – Ito , Ica, Ikr , Iks , Iki which are opened in succession during repolarization travelling through the syncytial ventricular mass.** These epicardial cells exhibits spike-and-dome pattern brought about by the Ito ion channels to initiate the subsequent phases of repolarization.

In Nutshell, 'Cardiac Action Potential' results with the sequential opening and closing of genetically determined ion channels (INa, Ito , Ica, Ikr, Iks ,Iki, etc)
The 'Endocardial cells' and 'Epicardial cells' do not show the ionic mirror image. The following differences in between two are observed:

- **'Endocardial cells' without notch pattern (dome pattern)**, having little or no expression of Ito channels. This is to be mentioned here that 'Endocardial cells' is concerned mainly with the depolarization of the ventricles.
- **The 'Epicardial cells' exhibits spike-and-dome pattern** brought about by the pool of Ito ion channels to initiate the subsequent phases of repolarization.
- In the experiment in canine myocytes no sustained component of L-type Ca^{2+} channel was observed in ENDO cells, while with EPI cells a somewhat deeper and longer notch was associated with a larger population of Ca^{2+} channels. The notch is practically absent in ENDO cells, i.e. the major difference between the EPI and ENDO cells is that **EPI cells do have a remarkable functional pool of Ca^{2+} channel to be activated while ENDO cells do not**.
  (**Reference** :Endocardial versus epicardial differences in L-type calcium current in canine ventricular myocytes studied by action potential voltage clamp by Tamas Banyasz et al, https://pubmed.ncbi.nlm.nih.gov/12667947/

The events discussed upto this stage may be summarized with the following formulas, as illustrated below phasewise :

- **Depolarization INa (Phase 0)**
- **Repolarization Ito Ica Ikr Iks (Phase 1, 2 and 3 respectively)**
- **Restoration to the resting state by Na^+-K^+ ATPase Pump and IKi (Phase 4)**

8. Phase wise classification of Ventricular Action Potential

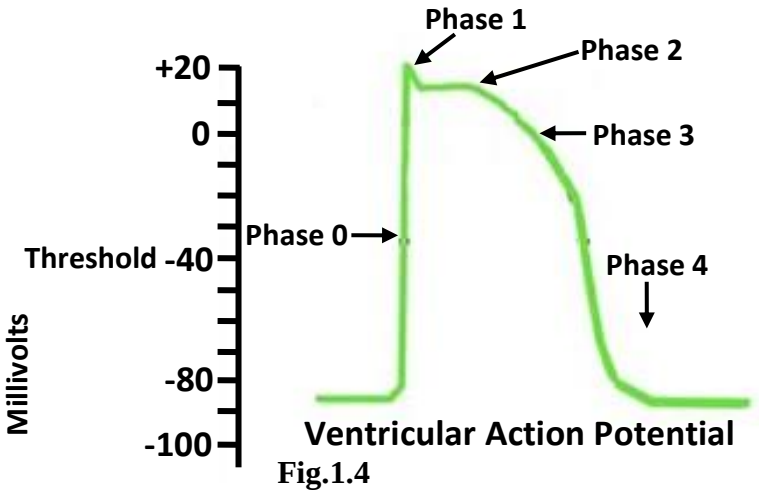


Fig.1.4

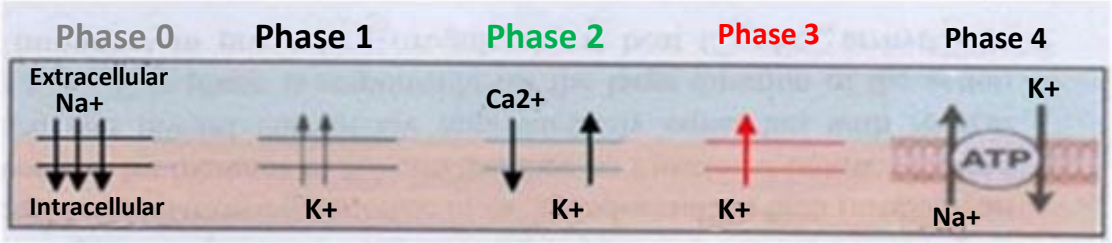


Fig.1.5

- ☐ The resting membrane potential of ventricular myocytes is about -90 mV indicating that inside of the membrane is more negative than the outside. The main ion outside the cell is addressed as Sodium (Na^+) , whereas inside the cell is mainly Potassium (K^+)

- ☐ Inward current facilitated through fast Na^+ channel brings positive charge inside the cell and depolarizes the cardiac membrane , the threshold potential for the purpose is at -70 mV. Due to the rapid influx of sodium ions , the phase 0 is having steep configuration – activation and inactivation of these channels almost occur at exactly the same time (**Phase 0 rapid depolarization**)

- ☐ A new turning point before repolarization sets in is the rapid inactivation of the Na^+ channels – K^+ channels (Ito) open and close rapidly, allowing for a brief flow of potassium ions out of the cell, making the membrane potential slightly more negative. This is referred to as a 'notch' on the action potential wave form. (**Phase 1 transient repolarization**)

- ☐ The next phase is the **phase of plateau** – stage of an alliance for cardiac contractility to take place. This is caused by increase in Ca^{2+} conductance through L-type calcium channels which cause an inward Ca^{2+} current and there is also increase in K^+ conductance resulting in outward flow of K+ current . During this phase outward and inward currents are approximately equal but with reverse polarity on either side. This phase is responsible for Excitation-Contraction Coupling of cardiac myocytes (**Phase 2 Plateau**)

- ☐ During this phase Ca^{2+} channels are closed and K^+ conductance is continued resulting in an outward K^+ current (**Phase 3 rapid repolarization**)

- ☐ This last phase is mainly due to the resetting of the cardiac potential brought about by Na^+- K^+ adenosine triphosphate pump , assisted by inwardly rectifying potassium channels (IKi) to have the fine tuning of ionic balance. IKi extends upto this phase 4 for the purpose (**Phase 4 resting potential**)

9. Cardiac action potential occurring at different levels (in brief) are illustrated below in a sequential manner

SA node is having a considerable higher frequency of depolarization (60-100/min) and overrides the pacemaker activity of AV node. AV node serves as an electrical relay station delaying the electrical current sent by the sinoatrial node before the signal is allowed to pass down to the ventricles. This delay allows atria enough time to empty its blood contents into the ventricles before ventricular contraction takes place.

SA Node Potential

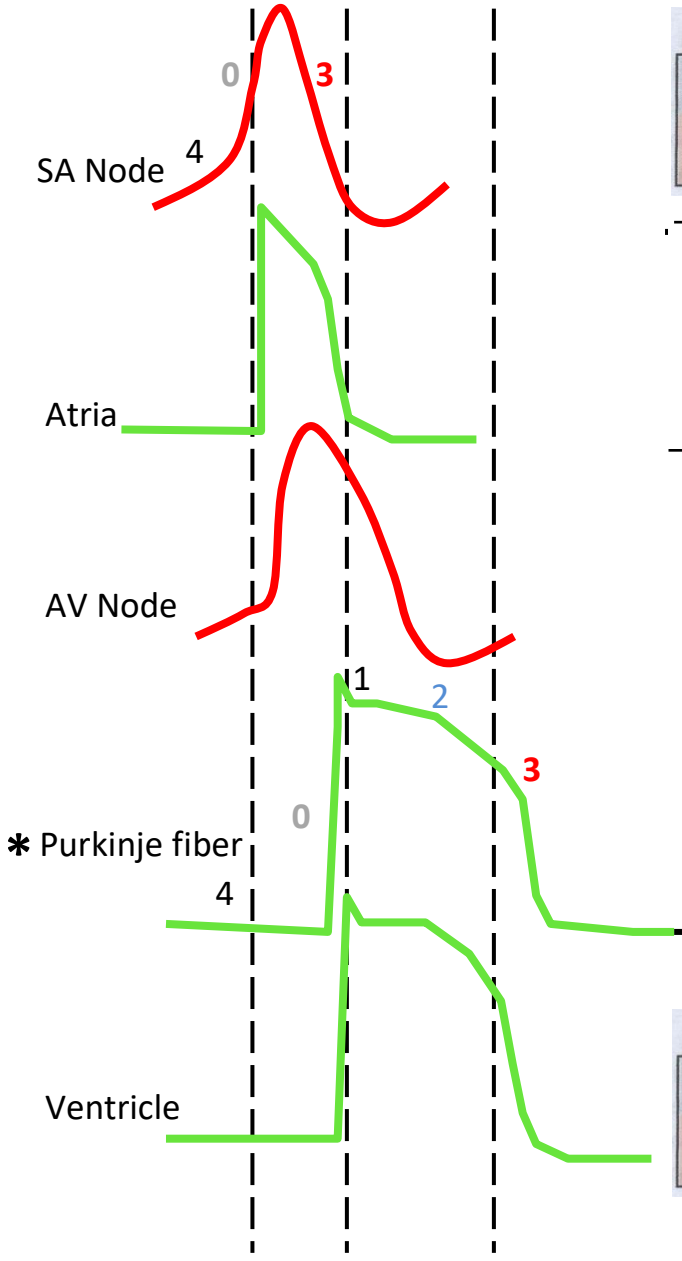
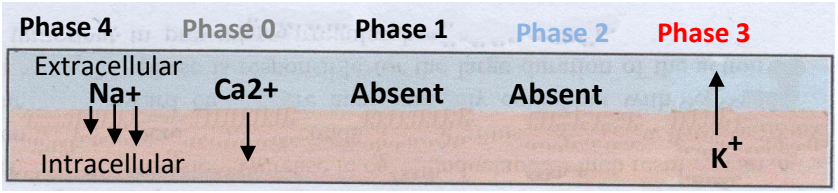


Fig.1.6

SA Node Action Potential



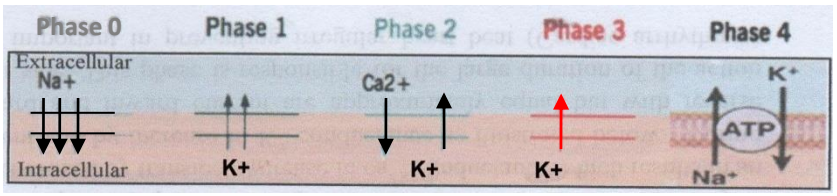
Atrial Action Potential – typically exhibits a triangular morphology

A more narrow phase 2 (plateau phase) due to a smaller calcium influx (Less muscular atria)

Relay through AV Node with further propagation.

Action potential in the AV node , like the SA node is governed primarily by changes with inward Ca^{2+} and outward K^{+} currents , and does not involve fast Na^{+} current. AV nodal tissue like SA nodal cells have also pacemaker activity but with a slower rate (40-60 per minute) due to diminished number of gap junctions between successive cells , with a somewhat resistant to the conduction.

Ventricular Action Potential



∗ Purkinje fibers are having the longest duration of action potential compared to the rest. This helps in preventing the backward impulse propagation from ventricles to upward direction , thus eliminating the chance of reentry mechanism.

10. The mechanism of Excitation Contraction Coupling of the cardiac muscle

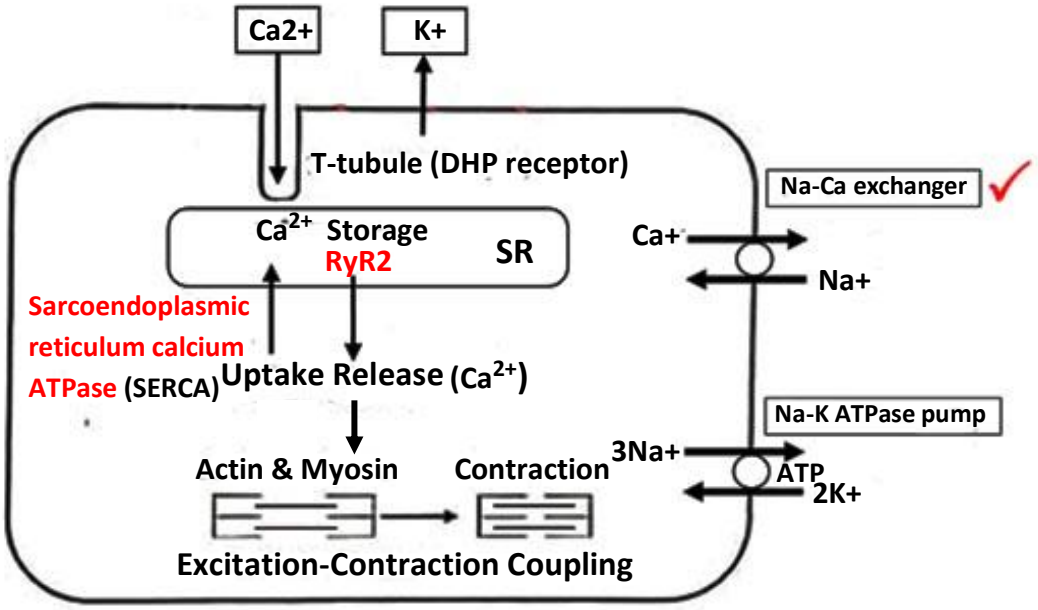


Fig.1.7

Steps in 'Excitation Contraction Coupling' are as follows:

- Action potential causes the impulse to travel along the Sarcolemma down to the transverse tubule (T-tubule) system through the depolarized cell membrane.
- Voltage sensitive DHP (Dihydropyridine) receptors open the gate to allow more calcium entry inside the cells during the Phase 2 of the action potential.
- During this plateau phase of the action potential Ca^{++} ions enter the inward of the cells through L- type calcium channel.
- This entry of Ca^{++} triggers more Ca^{++} release from sarcoplasmic reticulum through special channel – (ryanodine receptors) – RyR.
- With this further release of Ca^{++} ions there is increase in intracellular concentration of Ca^{2+}.
- Actin and myosin filaments are bound together , which allows sliding of these filaments over each other with the help of Ca^{2+} resulting in the contraction of myocardial cell (**Excitation-contraction coupling**)
- Ultimately the cardiac relaxation occurs when Ca^{++} is recycled back to the interior of sarcoplasmic reticulum by **'sarcoplasmic reticulum calcium ATPase' (SERCA)**. The extra calcium ions might also be pushed outside the cardiac myocytes through Na^{+}-Ca^{2+} exchanger mechanism whenever such ionic operation is needed.

11. Concluding remark

The cardiac action potential can be called as the '**Integrative Biochemical Laboratory of the Heart'**. Without its proper concept it would be rather impossible to understand the basic physiology of the heart and simultaneously it would also be impossible to have the concept of ECG interpretation. We have discussed a lot on cardiac action potential , this becomes essential to organize this entire concept in mind as a study pool .

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THE CARDIAC ELECTRICAL FIELD AND ITS ANALYSIS

ECG

THE CARDIAC ELECTRICAL FIELD AND ITS ANALYSIS

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OUTLINE

Introduction

The production of electrical current creating the electrical field is associated with all the cellular membranes dealing with the electrolytes ——

so it is also true with the heart.

The concept of cardiac electrical field

The heart produces a significant electrical field with each systole due to the sequential and coordinated depolarization of cardiomyocytes.

The area that surrounds the electrically charged particles is referred to as electrical field.

Historical steps in the evolution of cardiac electrical field

- The concept of electrical Einthoven' triangle and hexaxial lead system
- The birth of the chest leads : American Heart Association and the Heart Society of Britain and Ireland.

The cardiac electrical field and its clinical application in ECG interpretation

- **Cardiac vector hypothesis related to hexaxial lead system**

  As per cardiac vector hypothesis , the cardiac electrical field as a whole is having its own separate vector known as electrical axis and the voltage recorded in a particular lead is the result of scalar product between cardiac and the lead vector.

- **ECG recording over the precordial lead system (V1-V6)**

  This is based on the fact that the left ventricle is thicker than the right one and the orientation of leads over the chest is made to pick up the electrical current from the anatomical sites of the respective ventricles.

  V1/V2 faces right ventricular surface , V3/V4 points toward transitional zone and V5/V6 faces left ventricular surface.

Concluding remark

References

THE CARDIAC ELECTRICAL FIELD AND ITS ANALYSIS

A Narrative Review

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The electricity is a gleaming companionship – an arc of dynamic flow.
The heart also glimmers with such a spark within the realm of its electrical field. This fact aspired a few electrophysiologists to hold a separate vision to interpret the ECG.

First of all Augustus D Waller captured this concept of cardiac electricity and Einthoven converted this idea into a science of 'Artificial Intelligence' by formulating an equilateral triangle formed by the standard limb leads I , II and III – popularly known as 'Einthoven Triangle'. The science has made and still making all the possible efforts to interpret ECG in the light of this concept.

Two methodology exist today in this regard :

- ☐ **Hexaxial lead system records the flow of electrical current across the vertical plane of the body with the help of limb electrodes (bipolar leads I , II , III and unipolar leads aVR, aVL and aVF).**
- ☐ **Precordial lead system records the flow of electrical current across V1 to V6 leads , as placed over the horizontal plane across the chest.**

There is a dire need of understanding the cardiac electrical field for the purpose.

1. Introduction (Keypoints)

- The Heart produces electrical current which is spread over the body surface. This cardiac electrical field is about 60 times greater in its expression than the electrical activity expressed by the brain.
- The production of electrical current creating the electrical field is associated with all the cellular membranes dealing with the electrolytes ——
  So it is also true with the heart.
  The electrical current may be attributed to the selective ionic transport across the cellular membrane from negative pole towards the positive pole. This ionic environment is due to the presence of different electrolytes in the surrounding vicinity.

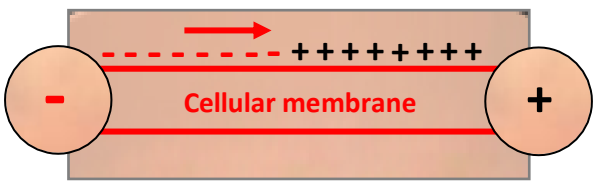


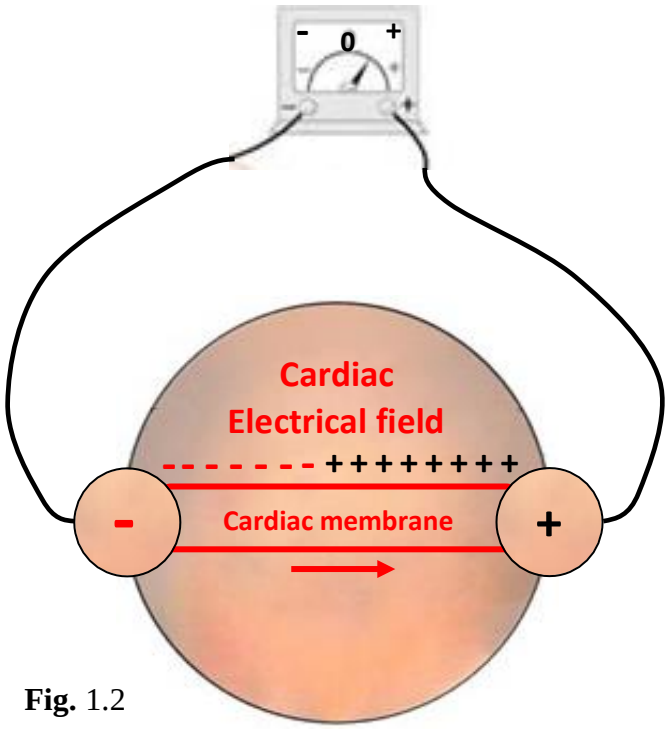
Fig. 1.1

It is well known to the science that this flow of ions (positively and negatively charged) contributes to the genesis of a field of cardiac electrical current.

- The sequential journey of cardiac waves P QRS T U pace their way through the cardiac electrical field. This had opened a separate research gate for ECG analysis. And this was made possible by creating two artificial lead system.
  - **Hexaxial lead system** : The heart is referred to be at the centre of cardiac electrical field (with zero potential) , this field is not confined only to the heart but also extends to a considerable distance over the body surface , including the limbs. The exploring electrodes are applied over the different limbs to capture the sequential flow of P QRS T U. This was made possible by rearranging the augmented unipolar leads (aVR , aVL , aVF) with that of limb leads of Einthoven (I , II , III).
  - **Precordial lead system** records the flow of electrical current by placing the specific chest leads over different specified points over the chest across its horizontal plane , designated as V1-V6 for the purpose. Any precordial lead so placed over the chest records the electrical potential through the small area of the underlying myocardium.

2. The concept of cardiac electrical field

The heart produces a significant electrical field with each systole due to the sequential and coordinated depolarization of cardiomyocytes. **The area that surrounds the electrically charged particles is referred to as electrical field** , as illustrated by the following sketch :



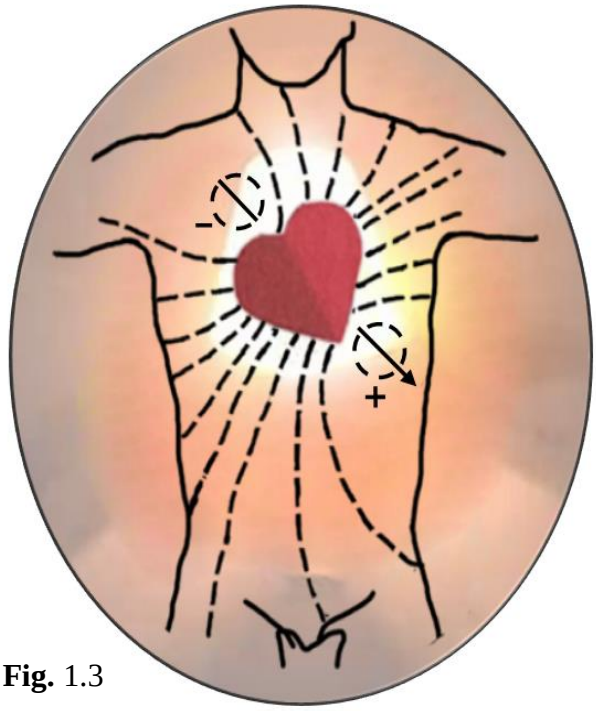
A simplified model of cardiac electrical field

Fig. 1.2

In order to understand the whole realm of cardiac activities over ECG , it is essential to know the concept of cardiac electrical field; the detailings have been put over the next page.

The cardiac electrical field is having dipolar source with positively and negatively charged poles and the flow of current is from the negative towards the positive pole. This movement produces an electrical force. This is to mention here that this electric field is created by all the existing charged ionic particles – this causes the cardiac membrane to be depolarized from negative pole towards the positive pole. **This cardiac electrical field carries electrical energy** ; this can be demonstrated with the help of a galvanometer by connecting positive and negative terminals through the conducting wires. This force of electrical energy can be well appreciated by charging an inflatted balloon by rubbing over its surface and one can see that it is attracted towards his shirt if it is brought nearby.

This is the story of cardiac electrical field in brief for the purpose.



The dotted small lines represents individual dipole.

Fig. 1.3

The cardiac electric field is illustrated on the surface of the thorax as viewed by Augustus D Waller (1887) based on the concept of dipolar source. The thoracic torso acts as a good conducting medium for the flow of underneath cardiac current.

A concept of dipolar source – A positive and negative charge separated by a small distance is known as a dipole. Such a single dipole is having very small electrical field which cannot be measured but the sum up of all the individual dipoles creates a larger field with more potential difference which may be large enough to be recorded on ECG. The flow of current across these two summed up dipoles is from negative to the positive pole having a definite magnitude and direction (known as vector) , as illustrated with the above sketch.

3. Historical steps in the evolution of cardiac electrical field

DR. WILLIAM EINTHOVEN surprised the world by delivering a lecture in 1912 at the Chesla Clinical Society of London . He inscribed a tri-axial bipolar system with three leads known as standard leads I , II and III. The triangle formed by these leads was later called **Einthoven's Triangle.**

Einthoven's Triangle is an imaginary three limb leads based triangle , useful in electrocardiograpy formed by the two shoulders (left and right) and the pubis (later pubis centre was replaced by left foot). **This forms an inverted electrical equilateral triangle with the heart lying at the centre**. It is so named after William Einthoven who first theorized its existence.

Einthoven used these measuring points by immersing the hands and L.foot in saline water , keeping this system in contact with his string galvanometer.

The concept of electrical Einthoven's triangle and hexaxial lead system

'With distances greater than 15 cm from the heart, the decrement in the intensity of the electrical field is hardly noticeable. Consequently, all electrodes placed at a distance greater than 15 cm from the heart may, in an electrical sense, be considered to be equidistant from the heart.' **(Reference** -LeoSchamroth – AN INTRODUCTION TO ELECTROCARDIOGRAPHY - EIGHTH ADAPTED EDITION – Basic Principles P – 6).

The three points used by Einthoven are to be considered having somewhat larger distance more than 15 cm. The triangle may be .'assumed to be equilateral in nature , as illustrated below.

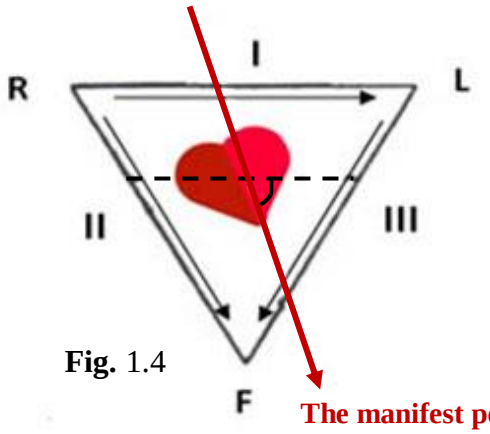


Fig. 1.4 **The manifest potential difference in the heart**

Bipolar Limb Electrodes
R – L = Lead I
R – F = Lead II
L – F = Lead III
R = Right arm , L = Left arm ,
F = Left foot

- **The manifest potential difference in the heart**– His remarkable concept of biophysics set forth a new concept first time in the history of ECG. Einthoven inscribed the famous equilateral triangle framed by lead I, II and III at its sides and derived a very important calculation what he called **'the manifest potential difference in the heart' - now known as Electrical axis.**

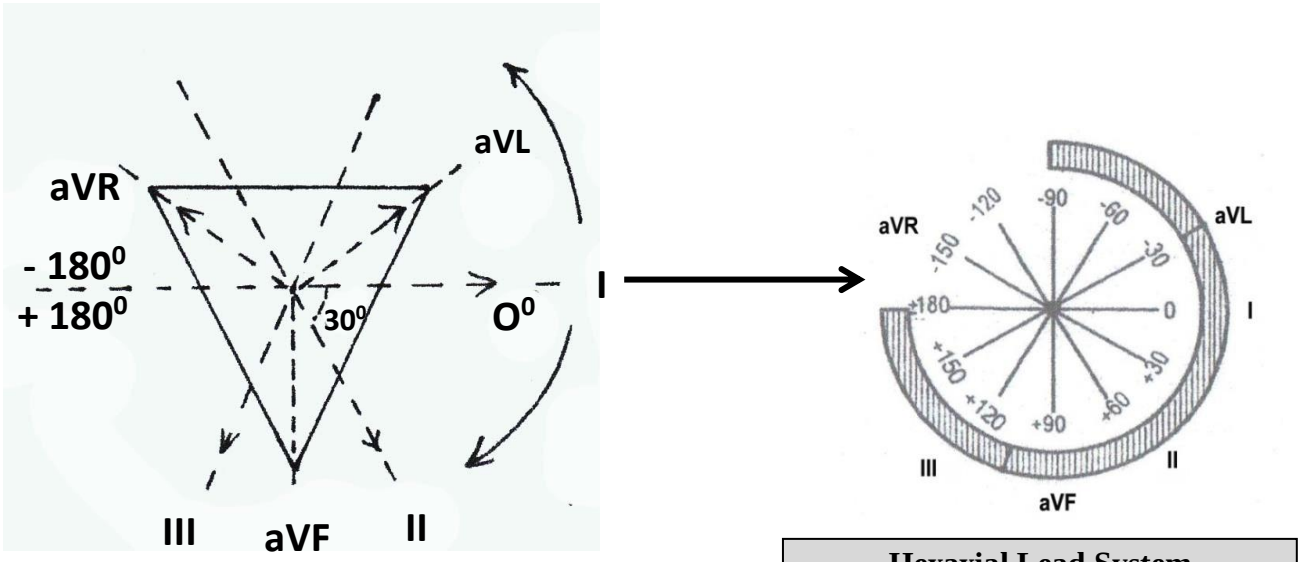
➢ **A new concept of an electrical vector came into existence :**

To his statement "**The curve must represent under all circumstances and in every moment, the algebraic sum of all the potential differences which at that moment are developed in the heart**. ." His this thinking might have hinted the concept of the manifest potential difference having a definite magnitude and direction

A Geometrical model of Hexaxial lead system

Einthoven's triangle can be made into a triaxial lead system by sliding the axes of leads I , II and III , allowing them to intersect at a central point. The augmented leads of Emanuel Goldberger aVR, aVL ,aVF also create a triaxial lead system and by combining the system of Dr. Einthoven and Dr. Emanuel Goldberger together accorded birth to hexaxial lead system.

By rearranging the augmented unipolar leads (aVR, aVL, aVF) and by combining with limb leads of Einthovens (I, II ,III) - the ' Hexaxial Limb Lead System' came into existence.



Hexaxial Lead System

Fig. 1.5

By convention the leads are arranged degreewise around a circle shown as above. Lead I is located at 0^o being used as the reference lead. Positive designation gets increased by 30^0 increments in a clockwise direction to $+180^0$ and negative designation gets increased by the same increment (30^0) in a counter clockwise direction to -180^0(please see the above diagram). Thus, so oriented negative and positive nomenclature of the various leads are used by convention for the mathematical determination of the electrical axis. The positive and negative degrees are nothing to do with the positive or negative pole of the leads. This is to be mentioned here that this system came into existence because of its conventional and

constant usage in clinical practice. This inscription of 'Hexaxial lead system' is very much helpful in the determination of the electrical axis.

The birth of the chest leads : American Heart Association and the Heart Society of Britain and Ireland.

The credit goes to the American Heart Association and the Heart Society of Britain and Ireland which in 1938 published their recommendation for recording the current flow from inside to outside over the horizontal plane by **placing the exploring leads opposite the six sites –namely V1 through V6 across the chest**. The 'V' stands for 'Voltage'. A conjoint adventure led the foundation of the Chest leads – the birth of recording the electrical current of the heart over the horizontal plane. A mystery of knowledge came into existence.

The anatomical concept reveals the fact that this fleshy heart is having a wonderful anatomical orientation within the thorax – the right-sided chambers of the heart are located somewhat anteromedially and the left-sided chambers are located somewhat posterolaterally

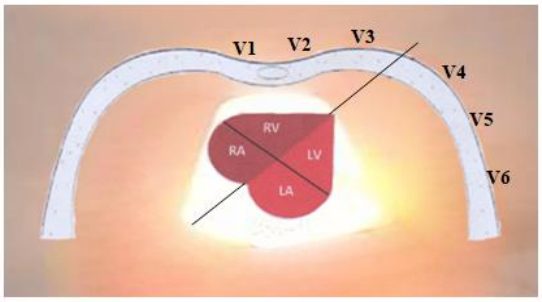


Fig. 1.6

V1/V2 faces right ventricular surface , V3/V4 points toward transitional zone and V5/V6 faces left ventricular surface.

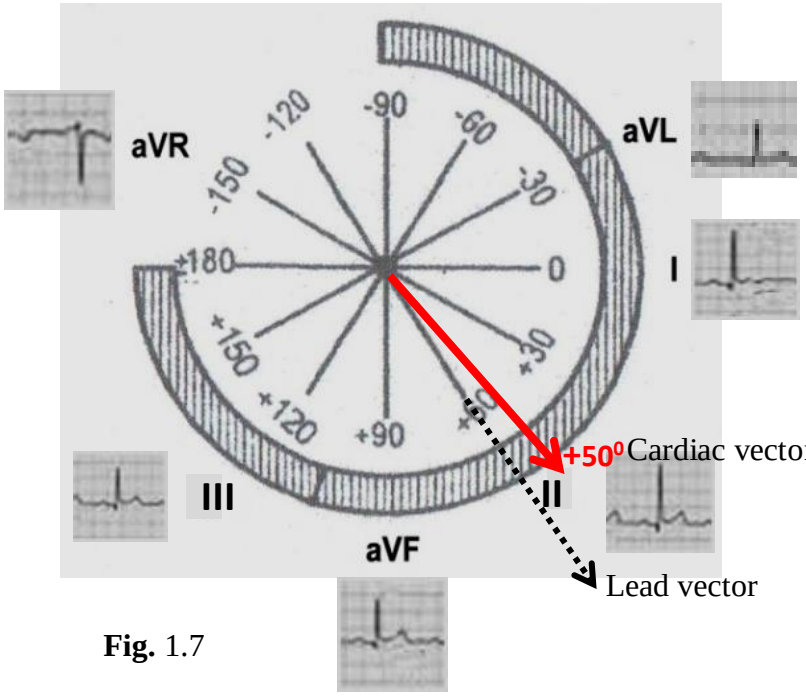
4. The cardiac electrical field and its clinical application in ECG interpretation

The two systems – Hexaxial and Precordial lead system need to be discussed separately in relation with the cardiac electrical field

Cardiac vector hypothesis related to hexaxial lead system

As per cardiac vector hypothesis , the cardiac electrical field as a whole is having its own separate vector known as electrical axis and the voltage recorded in a particular lead is the result of scalar product between cardiac and the lead vector.

The lead that is nearest to the cardiac electrical axis shows the maximum voltage inscribing the maximum amplitude on ECG. In other words, the voltage recorded in a particular lead is dependent on the distance quantum of main cardiac electrical vector (electrical axis).



The cardiac electrical axis lies at $+50^0$ and since the lead II (lead vector) is nearest to this electrical axis , it shows the maximum deflection by its amplitude.

Fig. 1.7

Accordingly a rule has been framed stating that

Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive deflection , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection (with reference to hexaxial lead system)

ECG recording over the precordial lead system (V1-V6)

This is based on the fact that the left ventricle is thicker than the right one and the orientation of leads over the chest is made to pick up the electrical current from the anatomical sites of the respective ventricles.
V1/V2 faces right ventricular surface , V3/V4 points toward transitional zone and V5/V6 faces left ventricular surface.

And the following rule may be framed to interpret ECG over precordial lead system recording.

Wave morphology is as per sequential activation from the thinner right ventricle towards the thicker left ventricle. The transitional zone lies at V3/V4 inscribing the equiphasic deflection on either side.

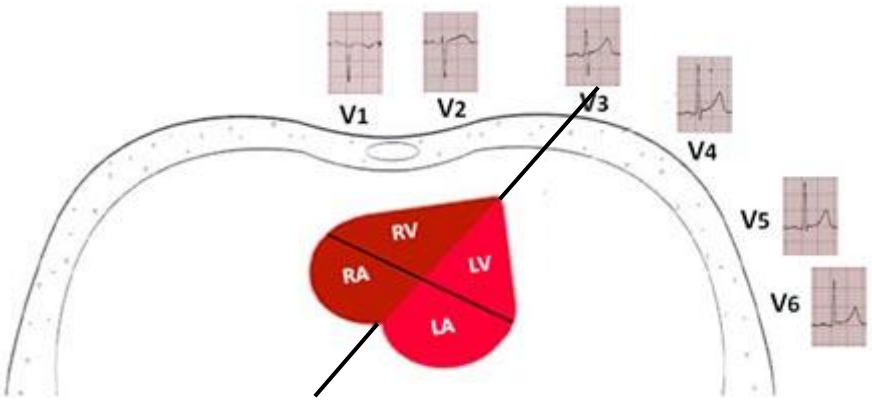


Fig. 1.8

Right ventricular epicardial complex - V1/V2 (rS)

Transistional zone - V3 / V4 (RS)

Left ventricular epicardial complex - V5, V6, aVL, I (qR/qRs)

In keeping view of the above mentioned facts it can be stated that there is a mirror reflection of the waves recorded on ECG over right and left ventricle because they are normally situated in the opposite direction to each other (rS over the V1/V2 represents the mirror effect of left ventricular voltage qR while r/q represents septal activation).

5. Concluding remark

The concept of cardiac electrical field describing its electrical activities over both the planes – vertical (hexaxial lead system) and horizontal plane (precordial lead system) is very essential to be understood for the interpretation of ECG tracings. And for the purpose a detailed description regarding both the planes has been forwarded in the preceding pages of this article.

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ACTION POTENITAL AND ECG – A NEW APPROACH

ECG

ACTION POTENTIAL AND ECG- A NEW APPROACH

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OUTLINE

Introduction

The Electrical Induction by Electrolytes

With reference to the 'Action potential' of the heart, the electrical current is attributed to the selective ionic transport across the cellular membrane. The ionic influx and efflux through the intra and extracellular environments are responsible for the genesis of electrical current. This ionic environment is due to the presence of different electrolytes in the surrounding vicinity.

The Cardiac Electrical Field

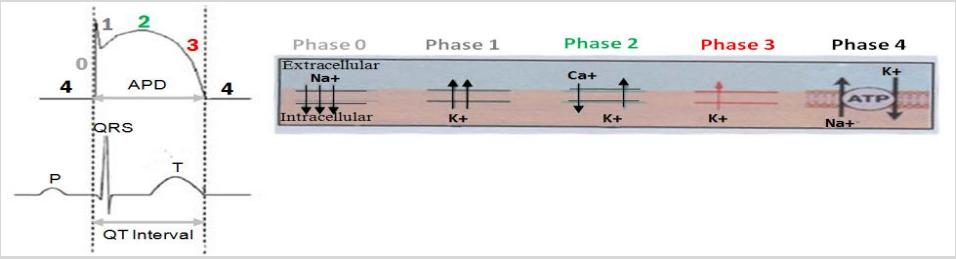
The heart's electrical field is about 60 times greater in expression than the electrical activity being expressed by the brain.

Basic Concept of Such Electrical Current

The production of electrical current creating the electrical field is associated with all the cellular membrane-functioning that deals with electrolyte's movement. So it is also true with the heart.

Action Potential (in brief) ↓

The Events of Cardiac Action Potential And The Genesis of ECG Changes



Basic ECG Waves : P QRS T U

Home take message

References

Action Potential and ECG – A new approach

© DR. D.P. KHAITAN
MD (MEDICINE), FCGP(IND), FIAMS(MEDICINE), FICP FICCMD

With reference to the 'Action potential' of the heart, the electrical current is attributed to the selective ionic transport across the cellular membrane.

- ☐ **The ionic influx and efflux through the intra and extracellular environments are responsible for the genesis of electrical current.**
- ☐ **This ionic movement is made possible due to the presence of electrochemical gradient across the cardiac membrane.**

Such electrical activities are picked by the ECG inscribing P QRS T U waves with intervening PR interval and ST segment.
I would like to quote here the prediction of Sir Thomas Lewis (British cardiologist - the father of clinical electrophysiology) who stated in the 1910s in connection with ECG that **" The Time is at hand , if it has not already come, when examination of the heart is incomplete if this new method is neglected ". And this is known to all that his prediction quickly became a reality**.

1. Introduction

The ECG is the graphic recording of electrical events during each cardiac cycle. There is an ionic flow across the cardiac cellular membrane - this flow contributes towards the genesis of electrical current. The word **electricity** had been coined denoting the flow of electrical current **by the movement of electrons** from the outer orbit of an atom to the atoms in succession and so on. In other words, the electrical current is a property induced by the flow of electrons. It has also been shown in science that the **flow of ions** (positively and negatively charged) also contributes to the genesis of a field of electrical current , as seen during the electrolytes influx and efflux across the cardiac membrane. And this ionic movement against the electrochemical gradient is responsible for the inception of cardiac electricity which is being picked up by ECG.

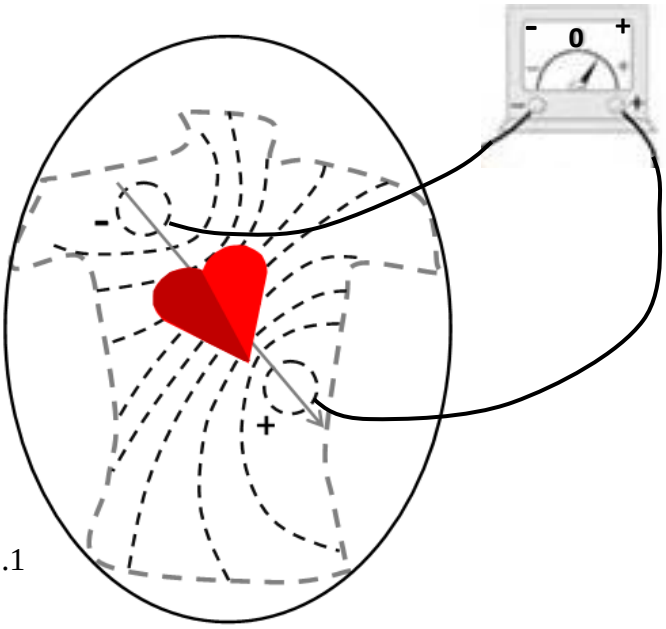


Fig. 1.1

2. Electrical induction by electrolytes

In chemistry , an electrolyte is electrically charged anion (-ve) or cation (+ve) which is present in such dissociated form in an aqueous solvent. The resulting solution with its dissociated anions and cations can conduct electricity under circumstances , as explained under para 2 of this page. The word electrolyte was coined in the 1800s by the combination of two Greek words - '**elektro**' (electrical) , and '**lytos**' (able to be lysed) , such lysed particle behaves as electrically charged unit - **'Electrolytic dissociation phenomenon'**

This can be stated that these dissolved electrolytes get separated into cations (+ve) and anions (-ve) with an uniform distribution in the environment of the solvent. The solution containing the lysed electrolytes is neutral in nature. If two electrodes of different nature are applied to such a solution, the cations (+ve) are attracted towards negative electrode in vicinity and on the other hand , anions (-ve) are deviated towards the positive electrode. The potential difference in between these two electrodes connected with each other causes the current to flow across.

This concept is very essential to understand the fundamental of ECG :

Fig. 1.2

An Electrolyte is electrically charged anion (-ve) or cation (+ve) , being present in such dissociated form in an aqueous solvent and is capable of producing electricity under circumstances as explained above.

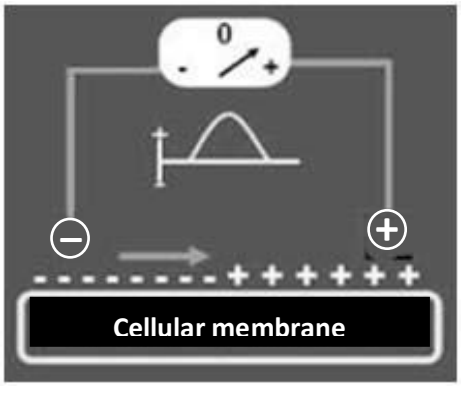


Fig. 1.3

With reference to the 'Action potential' of the heart, the electrical current may be attributed to the selective ionic transport across the cellular membrane from negative pole towards positive pole. **This ionic environment is due to the presence of different electrolytes in the surrounding vicinity – <u>A concept</u>.**

Fundamental
The current flows from negative pole towards the positive pole

3. The Cardiac Electrical Field

The ionic current is produced and its glow through the electrical field is detectable at a considerable distance. The summed up quanta of the electical current gets spread over the body surface creating the heart's electrical field which is about 60 times greater in expression than the electrical activity being expressed by the brain. Thus, the current produced by the heart is spread over the body surface tissues and this electircal field represents bio-potential electricity generated by the heart. This field is being picked up through ECG by placing different electrodes across the limbs and chest.

In nutshell ,it can be said that the cardiac action potential generated by the charged ionic movement across the cell membrane is being reflected upon the ECG in different wave forms.

The credit goes to **Augustus D waller** (British Physiologist of St. Mary's Medical School of London) who registered first time in the history , the electrical field on the surface of the thorax, generated by the electrical activity of the human heart in 1887. He was able to record the isopotential lines across the thoracic wall , known as dipolar units. These electrical lines are considered to be generated by the ionic movement during the depolarization and repolarization of the cardiac membrane , paving the way through the tissue of the throax **in the form of a dipolar flux (as illustrated in Fig. 1.4 on the next page).** Here to mention that the body surface acts as a good conductor for this dipolar flux , so as to produce the electrical field across the heart. Augustus D waller was the man behind the concept of this dipolar source of electrical field. The discovery of Human ECG using a Capillary Electrometer had also been credited to the his name.

4. Basic concept of such electrical current

There are certain important and interesting points to be considered here :

- The production of electrical current creating the electrical field is associated with all the cellular membrane-functioning that deals with electrolytes movement.
  So it is also true with the heart.
- There is a creation of extracellular cardiac electrical field due to ion fluxes across the cellular membranes and between adjacent cells of the cardiac syncytium. This ionic current is being expressed during depolarization and repolarization phases of the cardiac cycle. The polarity of such transmembrane potential creates a net dipole unit oriented in the direction of its propagation. On the negative pole there lies a summed up of all the negative dipoles into a single negative unit of dipole and on the other side there is a creation of single unit of positive dipole .The connecting link in between these two poles is the pathway through which an activation propagates ahead due to its potential difference , as depicted by the vector (illustrated by a sketch on the next page)

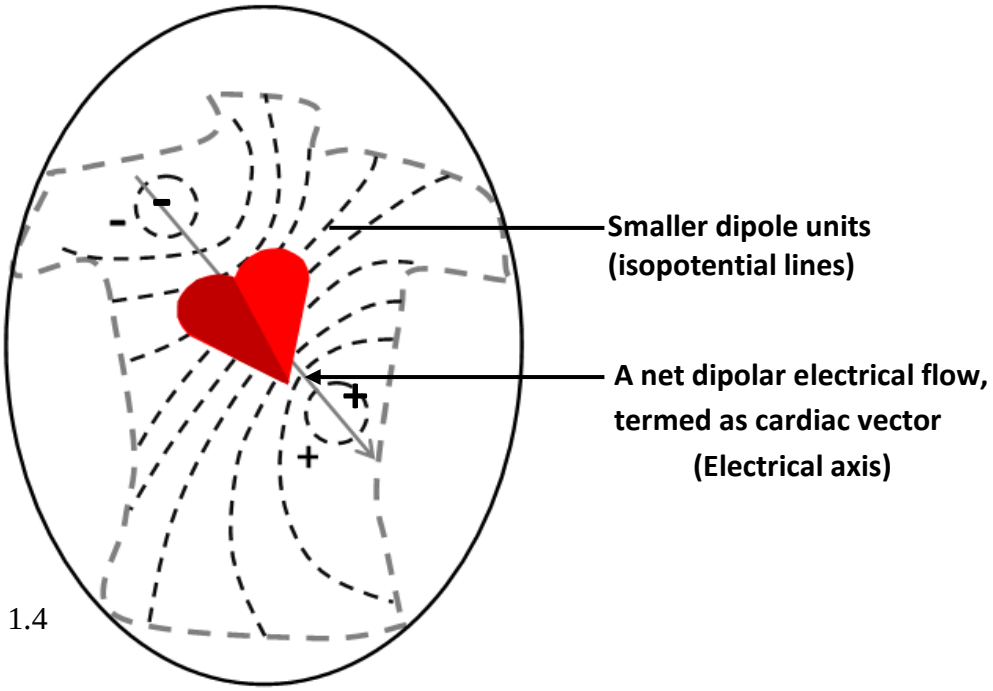


Fig. 1.4

Dipole – The positive and negative charged isopotential lines separated by a shorter distance create so many dipole units. Such every dipole is having very small electrical field which cannot be measured but the summed up of all these individual dipoles creates a larger field to be recorded.
The flow of current is from negative pole towards the postitive pole , as illustrated above .

5. Action Potential (in brief)

Action potential activities within the heart are recorded on electrocardiogram (ECG) in the form of a series of upward and downward waves – P QRS T U with intervening PR interval and ST segment during the depolarization and repolarization events of each cardiac cycle.

During the polarized state the cell membrane of the contractile unit (atria and ventricles) remains in resting phase by having positively charged outer surface and negatively charged inner surface , mainly with the help of Na-K ATPase pump there is an orientation of 3 Na^+ outside the cells and 2 K^+ inside the cells (in term of ratio). Further to mention that there are genetic channels over the membranous phospholipid bilayer which are very selective in respect to these ionic flow across.

The cardiac action potential is generated from the pacemaker SA node and its impulse is transmitted to the atria with the subsequent passing through in continuity from AV-His Purkinje system to the

ventricles to be activated. Then the next cycle of repolarization sets in. The phases of depolarization and repolarization make a single fuctional unit to be expressed as cardiac action potential.

6. The events of Cardiac Action Potential and the genesis of ECG changes

A relation of the 'Action Potential ' to the ECG is illustrated below by the following sketch :
(The standard model used to demonstrate the cardiac action potential here is that of the ventricular myocardium)

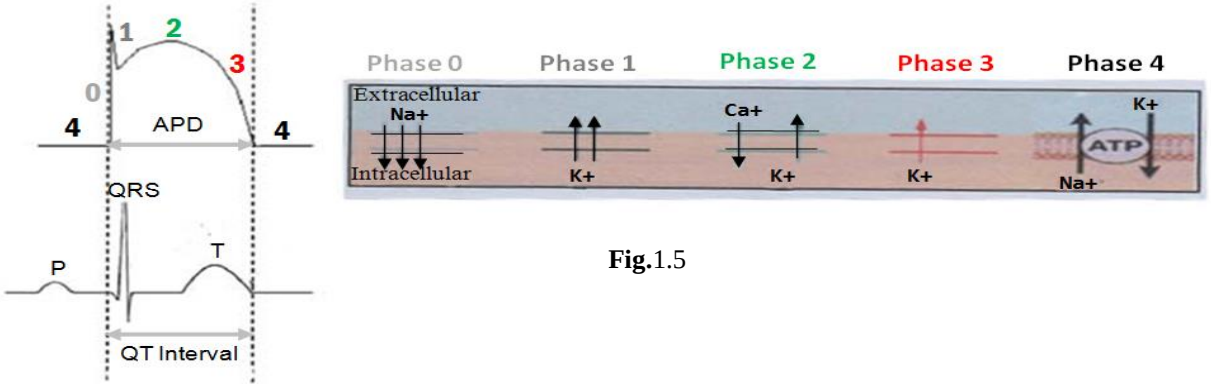
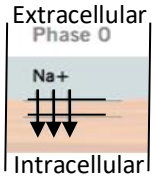


Fig.1.5

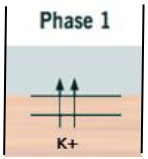
There are two group of cells in the heart , namely the main pacemaker cells (the SA node) and the contractile units (atria and the ventricles) , both of which are normally operative during each cardiac cycle. The pacemaker cells do not possess phases 1 and 2 of the cardiac action potential - lacking the contractile activity.

The inside of the cardiac membrane is more negative than the outside. The main ions responsible for this status are 3Na+ outside the cells and 2K+ inside the cells (in term of ratio).

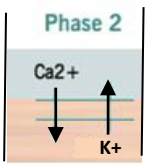
The events of cardiac action potential and the genesis of ECG changes are outlined as below in brief :



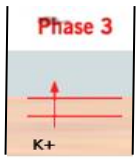
☐ The Rapid Inward cellular current facilitated through Na^+ conductance brings positive charge inside the cell - this depolarizes the cardiac membrane of the atria and ventricles separately inscribing the P-wave for atria and QRS complex for ventricles **(Phase 0)**



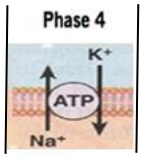
☐ During **phase 1**, there is some outward movement of K^+ for a transient period making the membrane potential slightly more negative. This is referred as a 'notch' over the action potential. The same is being reflected on ECG as a notch known as j (junctional) point being inscribed at the end of the QRS complex.



☐ The **plateau in phase 2** is due to the balance in between inward movement of Ca^{2+} and outward movement of K^+. This phase actually corresponds to the ventricular contraction and is being reflected on ECG as isoelectric ST segment.



☐ Replolarization occurs during **phase 3** due to the further outward movement of K^+ ions outside with the closing of Ca^{++} channels and this phase is reflected on ECG in the form of T wave.

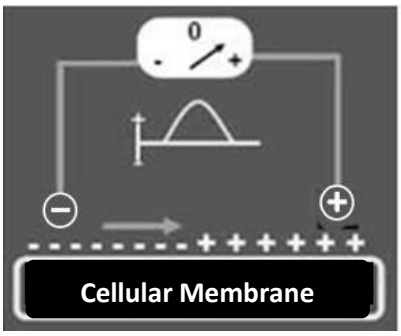


☐ The phase 3 ends in **phase 4** where the cardiac membrane potential is brought to the original polarized state mainly by the Na^+-K^+-ATPase pump , other helper being inward rectifier IKi current. This resting phase on ECG is reflected by TP segment - an isoelectric line.

The PR segment starts from the point of atrial depolarization and this represents the time taken for a subsequent journey through the AV node (with a transient stay of 0.09 sec) to the Purkinje system.

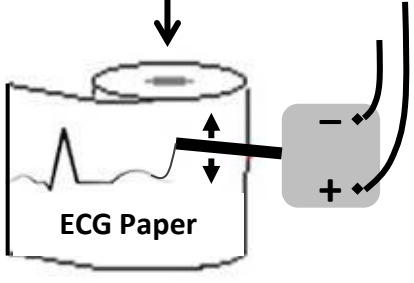
The wave representing atrial repolarization (Ta) is not usually visible on ECG because it is having low amplitude (100-200 microvolts) and is hidden inside the ensuing QRS complex.

The registration of these entire electrical events on ECG are summarised as below:



Positive deflection (on ECG) towards the flow of current

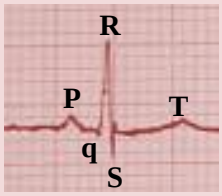
Through the electrode



Heated stylus moves with voltage change as paper moves

(The electrical energy produced during action potential is converted into mechanical-cum-heat energy to move the stylus and record the tracings)

Fig. 1.6



P wave= Atrial depolarization .

PR interval = The interval in between the beginning of P wave to the beginning of QRS complex.

QRS Complex = Ventricular depolarization (both the ventricles depolarise synchronously).

ST segment= The interval in between the end of ventricular depolarization and the start of repolarization phase (the end of S wave / J point and the beginning of the T wave).

T = Ventricular repolarization.

TP interval = The period of cardiac rest.

QT interval= From the beginning of the QRS complex to the end of the T wave (depolarization **+** repolarization).

7. Basics of ECG waves

- ☐ When the flow of current is towards the positive electrode of a lead , the deflection on ECG is having an upward positive wave.
- ☐ The reverse is true when the electrical current flows to the opposite direction , it causes a negative deflection on ECG.

GENESIS OF P WAVE

(longitudional transmission)

The P-wave is due to atrial depolarization

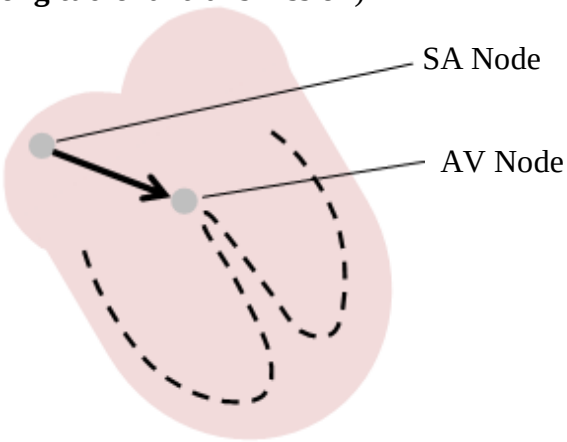


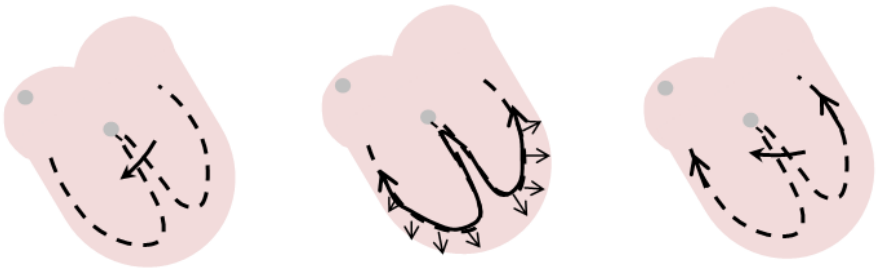
Fig. 1.7

Atrial depolarization

The initial wave on ECG is the P wave which reflects atrial depolarization. The PR interval is the distance measured between the onset of P wave to the onset of the QRS complex.

GENESIS OF QRS

(transverse transmission : from endocardium to epicardium)



Septal activation from left to right

r/q

(Recorded as **r** over the right precordial leads, **q** over the left precordial leads)

Activation of major portion of ventricular myocardium from endocardium to epicardium surface

R

Late activation of posterobasal portion of the left ventricle, the pulmonary conus, and the uppermost portion of the interventricular septum

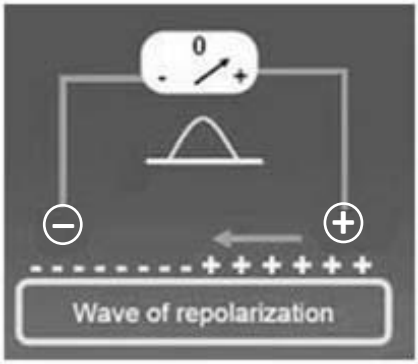
S

Fig. 1.8

GENESIS OF T WAVE

The repolarization wave is known as T wave. It is normally upright because of the following facts :

- The process of repolarization starts from the point where the depolarization wave QRS gets ended . This is to mention here that the last portion of ventricular depolarization is mainly the posterobasal portion of the left ventricle and that's why , the wave of repolarization starts from this point and runs towards the epicardium to start the journey of repolarization. The wave of repolarization propagates ahead leftward and downward in a similar positive direction as that of QRS complex , thus inscribing upward T wave.
- Epicardial cells are normally having a shorter duration action potential than endocardial cells. This shorter duration action potential causes them to repolarize earlier compared to those of endocardial cells having longer action potential duration.



(Positive T-wave in the same direction as that of QRS complex)

Fig.1.9

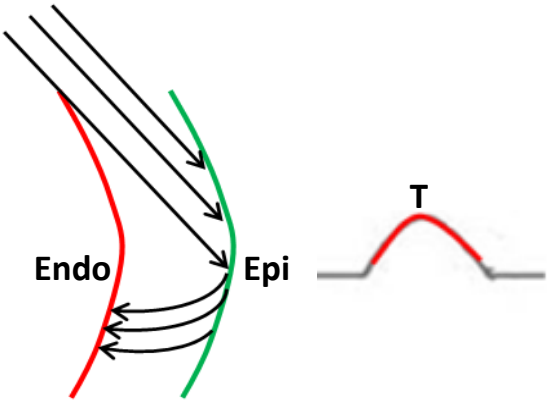


Fig.2.0

GENESIS OF U WAVE

The T wave is followed by another tiny wave known as U wave (<25% of the corresponding T-wave). The origin of the U wave is still in question, although most authorities correlate the U wave with after potential resulting from mechanical force in the ventricular wall.

other reasons for U wave -

- Delayed repolarization of Subendothelial Purkinje fibres
- Prolonged repolarization of Mid-myocardial "M-cells"

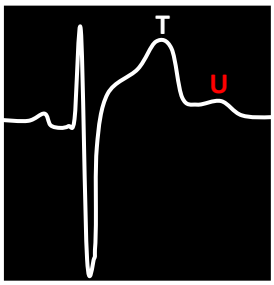


Fig. 2.1

New hypothesis

Recently a new hypothesis (**put by Kenneth Tsan He and Helena Ai He , 2020**) regarding the U-wave genesis has been proposed , whereby collision of the heart apex with its vicinity against the corresponding chest wall causes a somewhat delayed repolarization of some myocardial cells. Therefore , two waves are generated in succession – first due to initial ventricular repolarization , T-wave and second tiny U-wave due to the repolarization of the remaining myocardial cells. Thus , the left ventricular cells are dichotomized into two parts : the former with a normal repolarization process inscribed the T-wave , while the latter with a delayed repolarization forms the U-wave.

8. Home take message

- ☐ The different waves on ECG are inscribed due to the successive influx and efflux of different ions during the cardiac action potential.
- ☐ This flux of ionic exchange across the cardiac membrane produces many dipole units. Such individual dipole is having very small electrical field which cannot be measured but the summed up of these individual dipoles creates a larger field to be recorded, as already discussed previously.
  Thus , the vector is the resultant summed up of all these smaller dipole currents .

- ☐ The current flows from negative pole to the positive pole. This is detected by applying the exploring electrodes over the concerned areas.
- ☐ When the flow of current is towards the positive electrode of a lead , the deflection on ECG is having an upward positive wave.
  The reverse is true when the electrical current flows to the opposite direction , it causes a negative deflection on ECG.

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SINE WAVE ON ECG IN HYPERKALEMIA : A CONSIDERATION

ECG

SINE WAVE ON ECG IN HYPERKALEMIA : A CONSIDERATION

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OUTLINE

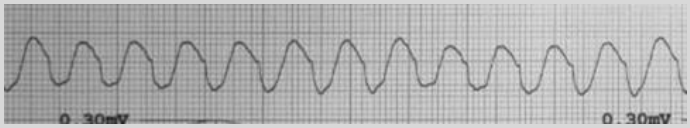
Introduction

Sine wave pattern on ECG in hyperkalemia though rarely encountered in practice , is a clinically significant and dreadful condition – it should be diagnosed promptly with its emergent treatment to prevent the lethal consequences

Electrophysiology

A worsening of cardiac conduction system leading to somewhat exaggerated widening of the QRS complex which ends up in its fusion with the ensuing widened T-wave (**sine wave pattern**).

The classical sine wave is without the co-existing P-wave.



Classical sine wave as up (**A** – widened ventricular depolarization phase) and down (**B** – widened ventricular repolarization phase) oscillation

Concluding remark

References

SINE WAVE ON ECG IN HYPERKALEMIA : A CONSIDERATION

A Narrative Review

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The Sine Wave in mathematics represents a repetitive up and down oscillations with the same shape , frequency , and amplitude on either side – repeating itself over time as if a continuous graph. In other words the sine wave is a graph having a smooth curve oscillation between positive and negative values.
Sine wave on ECG in hyperkalemia is having a sinusoidal pattern , very much mimicking as that of sine wave in mathematics , and when seen , it points towards the warning of imminent death.

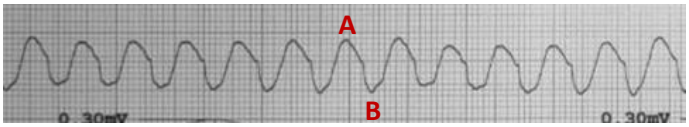
- ☐ **As the serum potassium level increases to a critical level, there is worsening of cardiac conduction system causing a merger of the resultant widened QRS complex with that of widened T-wave – an unified run with equally expressed up and down troughs of QRS and T waves respectively**
- ☐ **With downgoing cardiac conduction system there is every possibility of collapsing of such sine wave to a straight line of cardiac asystole.**

A sine-wave pattern on ECG is having a high specificity for detecting hyperkalemia. By its timely diagnosis and prompt management the further steps of this gloomy journey may be reverted back to normal.

1. Introduction (Keypoints)

- Sine wave pattern on ECG in hyperkalemia though rarely encountered in practice , is a clinically significant and dreadful condition – it should be diagnosed promptly to prevent its lethal consequences.
- ECG is a simple tool to diagnose the hyperkalemia even before the potassium estimation by the laboratory is available. There are some pointers of hyperkalemia on ECG , such as symmetrical T-wave tenting , PR interval prolongation with reduced P-wave amplitude or even its absence , prolongation of the QRS ——

  The dreaded scenario of hyperkalemia on ECG is the appearance of sine wave.
- A classical sine wave in hyperkalemia denotes an unified run of up and down oscillations with equal spacing and amplitude ; this oscillation is considered due to the worsening of cardiac conduction during ventricular depolarization and ventricular repolarization , as illustrated by the following ECG tracing.

Fig. 1.1 

Classical sine wave is displayed here as up (**A** – widened ventricular depolarization phase) and down (**B** – widened ventricular repolarization phase) oscillation

The sine wave of hyperkalemia can be compared to that of sine wave in mathematics :

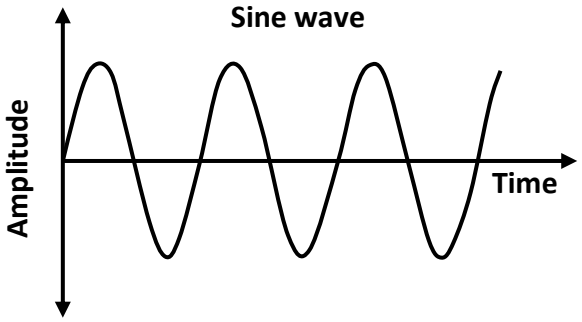


Fig. 1.2

The sine wave in mathematics is up and down curve that oscillates up and down with equal spacing and amplitude – a graph of repetitive oscillations.

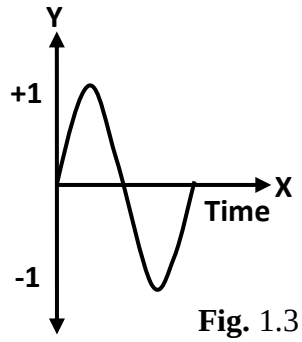


Fig. 1.3

A concept of Sine

Why is it called sine wave ?

The sine wave is a mathematical sinusoid curve that describes a smooth repetitive oscillation graph. Here the up turn is the same as that of down turn in its spacing and amplitude. This visual representation is known as sine graph , as illustrated with the side sketch.

- **If the sine wave pattern is detected on ECG , it is having a high specificity for the diagnosis of severe hyperkalemia**. Under such circumstances even with non-availability of serum potassium estimation in the emergency department , initiation of emergent treatment should be instituted.
  (It would be worthwhile to mention here that **Venous blood gas analysis** provides an immediate result of the patient's potassium level).
- The status of hyperkalemia is further strengthened in the presence of symptoms such as nausea , vomiting , paraesthesia , muscle weakness , palpitation , unability to standing up ± drowsy status with any of the followings :
  (commonly encountered conditions are innumerated here)
  - Skipped haemodialysis with pre-existing renal disease
  - Acute kidney injury
  - History of diabetes mellitus / hypertension as a predisposing cause of renal insufficiency
  - Drugs
    Potassium-Sparing Diuretics, including Spironolactone
    ACE inhibitors (or ARBs)
  - Excessive consumptions of potassium rich diet (fruits , leafy vegetables , potatoes , etc.) by a patient with predisposed conditions.
  - History of trauma resulting in 'Rhabdomyolysis'

For details of causative factors in hyperkalemia
Ref : Davidson's Principles and Practice of Medicine (International 24th Edition)
P 629 , Table 19.16 Causes of hyperkalemia

- This is to be mentioned here that the sine waves are more dramatic in its appearance on ECG with faster evolution towards the lethal journey (cardiac asystole / ventricular fibrillation). There should be no delay in the institution of its treatment , as needed.
  One suggested regimen :
  - 10 ml of 10% of calcium gluconate mixed with 100 ml D5W or NS to be infused over 5-10 minutes (it may be repeated as per need to achieve QRS < 100 ms or till the appearance of P-wave on ECG)
  - 2 Amps of D50W followed by 10 units rapid acting insulin IV
  - Salbutamol 8 puffs by aerochamber or 20 mg nebulized , but after IV insulin

  Immediately refer the case to the expertise centre for further treatment and haemodialysis , as per need.

2. Electrophysiology

In hyperkalemia there is usually a stepwise progression of changes on ECG.

As the serum potassium level increases to a critical level , there is a worsening of cardiac conduction system leading to somewhat exaggerated widening of the QRS complex which ends up in its fusion with the ensuing widened and inverted T-wave. Such fusion of the QRS complex with the corresponding T-wave is referred to as **sine wave pattern**.

For proper understanding let us review stepwise progression of ECG changes in hyperkalemia :

Accelerating response

The potassium current responsible for potassium efflux during repolarization is much sensitive to increased extracellular potassium level. Function of I_{Ki} (rapid evolving potassium current) increases with hyperkalemia causing enhanced potassium conduction during phase 2 and 3 of the action potential. This leads to faster and shorter repolarization , being revealed on surface ECG as the symmetrical tented T wave with shortening of ST segment , peaked up as shortened QT interval.

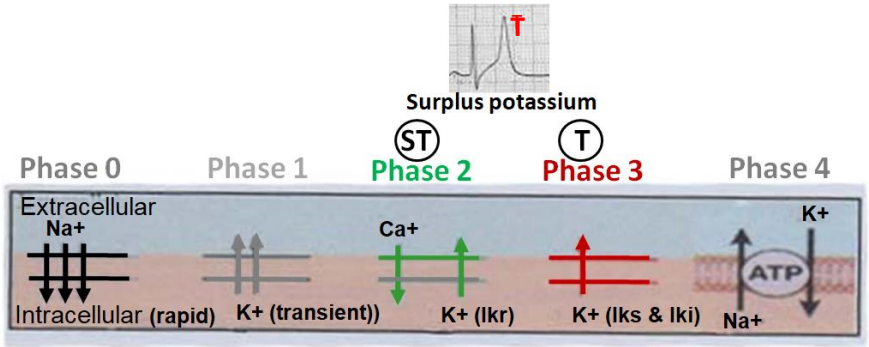


Fig. 1.4

Deaccelerating response

Hyperkalemia leads to conduction abnormality with impaired myocardial conduction :

A stepwise journey :

- **Atrial paralysis :** Atrial myocytes are more sensitive to hyperkalemia than the SA node , ventricular tissues or the Bundle of His. Even with further increment of serum potassium level , **sinus node continues to propagate its electrical activity to the ventricles even in the absence of atrial depolarization , such conduction is called as sino-ventricular rhythm.**

  Theoretically the existence of such sinus node activity passing to the ventricular system in the absence of P wave can be confirmed from the induced respiratory sinus arrhythmia – inspiration increases and expiration decreases the ventricular rate.

- **QRS widening**

  The conduction velocity through the ventricle is determined by upstroke phase 0 of the ventricular action potential that is governed by rapid influx of sodium ion. The influx rate is related to the relationship between the resting membrane potential = - 90 mV and threshold potential (-60 mV) after which a spontaneous action potential occurs.

  With ongoing hyperkalemia , the resting membrane potential is less negative with its closer relationship to the normal threshold potential. That's why , the influx of sodium ions is retarded with the subsequent decrement in the conduction velocity – causing a widening of the QRS complex.

  Closer is the value of resting membrane potential to the threshold potential , the less rapid is the influx of sodium ions with the slower conduction velocity , as illustrated below as red dash line (in hyperkalemia).

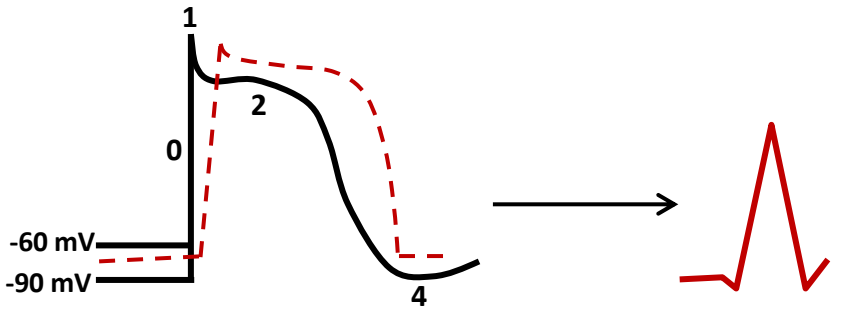


Fig. 1.5

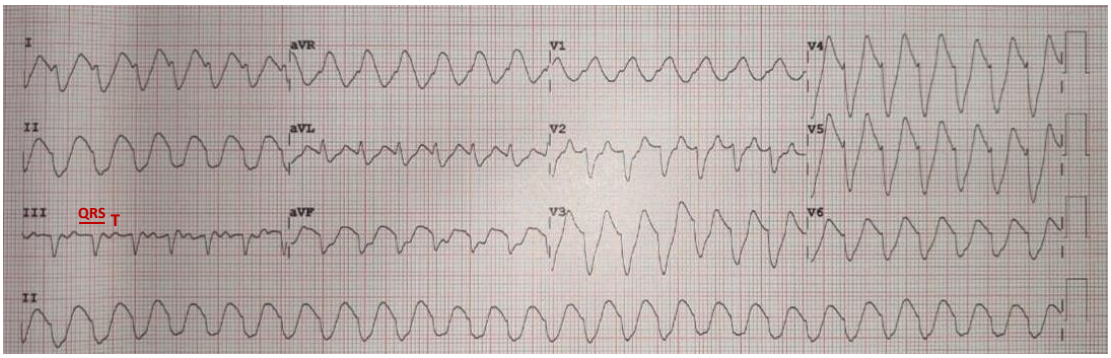
-90 mV = more negative resting membrane potential

Somewhat less negative resting membrane potential (with ongoing hyperkalemia)

-60 mV = Normal threshold membrane potential

Shorter is the distance in between resting membrane potential (induced by hyperkalemia) and normal threshold membrane potential , the less rapid is the influx of sodium ion with the slower conduction velocity resulting in QRS widening.

- **Merging together of widened QRS complex with that of the widened T-wave = Classical sine wave (with the absence of P-wave)**
  This phenomenon of sine wave is well illustrated by the ECG , as put below :
  (History : Middle aged Diabetic female presenting with weakness , giddiness , nausea and vomiting)



Source : CME INDIA on 12.08.2023 by Prof. Dr. A.N. Rai , Gaya (Bihar)

Findings on ECG :

- Ventricular rate 186 bpm
- Rhythm lead II shows a run of up and down oscillations , almost with equal pacing and amplitude **— sine wave**
- Lead V1 is showing much widening of the QRS complex almost equivalent to 0.28 sec simulating with right bundle branch block pattern.
  (See carefully lead III and aVL there is also fusion of widened QRS with widened T)

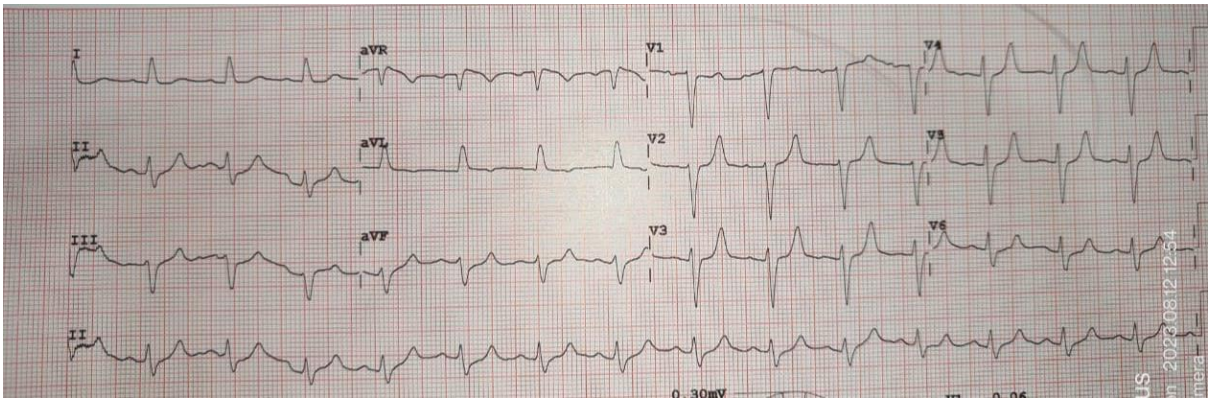
Comments :

- In this case the presence of sine waves on ECG is highly suggestive of severe hyperkalemia , consistent with the laboratory findings : serum potassium 7.6 mmol/L (and serum creatinine 4.80 mg/dL).
- With ongoing hyperkalemia , the resting membrane potential is less negative with its closer relationship to the normal threshold potential. That's why , the influx of sodium ions is retarded with the subsequent decrement in the conduction velocity – causing a widening of the QRS complex.
- Widened T-wave is also due to the worsening of cardiac conduction system.

D/D :

The presence of sine wave with so extreme ventricular rate (186 bpm) might impart a pseudo impression of ventricular tachycardia but it should be kept in mind that ventricular tachycardia is not having so super wide QRS as in this case. The so fast ventricular rate encountered in this case is most possibly due to the much closer merging of QRS complex wtih T wave (If anyone administer Amiodarone or apply direct current cardioversion to the patient by thinking this to be VT , it would bring further deterioration in cardiac conduction with immediate cardiac asystole).

See the ECG on the next page after correcting hyperkalemia – this returned back to normal



3. Concluding remark

- ☐ The presence of sine wave on ECG is having a high specificity of severe hyperkalemia
- ☐ Sine wave in hyperkalemia denotes an unified run of up and down oscillations with equal spacing and amplitude ; this oscillation is considered due to the worsening cardiac conduction during ventricular depolarization and ventricular repolarization.
- ☐ Even with non-availability of serum potassium estimation in the emergency department , initiation of emergent treatment should be instituted to save the life of the patient.

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P 629 , Table 19.16 Causes of hyperkalemia

PARASYSTOLE : A COMPETITIVE CARDIAC ARRHYTHMIA

ECG

PARASYSTOLE : A COMPETITIVE CARIAC ARRHYTHMIA

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OUTLINE

Introduction – Salient features

Parasystole is a very rare arrhythmia which is considered as a specific competitive run of a dual rhythm, framed by two separate autonomous foci with different inherent rates – one focus is SA NODE, the normal dominant pacemaker and other is the non-dominant subsidiary focus usually occupying the VENTRICLE but can also be situated in AV node or the atrium.

Epidemiology

Incidence , Age / sex , Race , Risk factors

Natural history

Mechanism – its electrophysiology

Ventricular Parasystole is due to the interaction between two fixed rate pacemakers having different inherent rates – first is the fast beating SA node and the second one is the slow beating ventricular parasystolic pacemaker.

When a sinus pacemaker and a protected subsidiary pacemaker coexist , a characteristic arrhythmia appears - known as 'Parasystole'

Typical ECG findings in parasystole

(i) Variable 'coupling intervals' between sinus beats and parasystolic ventricular beats
(ii) The longest interectopic interval is almost exact multiple of the shortest basal interectopic interval
(iii) ± The presence of fusion beats

Concluding remark

References

PARASYSTOLE : A COMPETITIVE CARDIAC ARRHYTHMIA

A Narrative Review

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A good competition always forces one to do the best , where two or more strive for a common goal.
This is an old proverb that the weak competes and the stronger dominates. The framing of some intelligent step remains only the answer for a weaker one to succeed in this competitive race.
The same holds true with the parasystolic centre , standing as slower subsidiary pacemaker which has acquired the property of protection against the existing dominancy of SA node.

- ☐ **Here the competition is usually in between the dominant pacemaker SA node and the parasystolic one , commonly situated in the ventricle ——— the resultant arrhythmia is the 'Parasystole'.**
- ☐ **The competitive parasystolic pacemaker protects itself from the jolt of dominant one by creating an entrance block around itself.**

This entrance block created by the parasystolic focus is a defensive step to prevent the interference by the foregoing run of the dominant one.

1. Introduction – salient features

- Parasystole is a very rare arrhythmia which is considered as a specific competitive run of a dual rhythm, framed by two separate autonomous foci with different inherent rates – one focus is SA NODE, the normal dominant pacemaker and other is the non-dominant subsidiary focus usually occupying the VENTRICLE but can also be situated in AV node or the atrium.
- NORMALLY there is an autonomous impulse initiation at the SA node which dominates over all other subsidiary potential pacemakers due to its relatively faster rate of discharge. However, occasionally a subsidiary pacemaker centre acquires the property of protection against the existing dominancy of the SA node.
- **When a sinus pacemaker and a protected subsidiary pacemaker coexist , a characteristic arrhythmia appears - known as 'Parasystole'.**
- Parasystole was first observed and reported by **Schamroth in 1967**. He described parasystole as a dual rhythm entity in which the slow running subsidiary pacemaker is protected from the effect of the dominant pacemaker SA node with faster rate of discharge : this protection is the essential requisite of this arrhythmia. This protection is considered to lie within the immediate territory of the parasystolic focus.

2. Epidemiology

- **Incidence** : This is a very rare arrhythmia with the incidence of 0.13 percent of all the electrocardiograms recorded in a general hospital.
- **Age / sex** : Patients of all age groups may have this arrhythmia but it is commonly encountered in older patients , usually more than 60 years of age.
  Males are more commonly affected , compared to females with an approximate ratio of 2:1.
- **Race** : No race is immune to parasystole.
- **Risk Factors**
  - The most common association with this arrhythmia had been found with atherosclerotic heart disease and / or hypertensive cardiovascular disease (60 percent) and surprisingly half of these patients were found to have congestive heart failure in addition. Other acquired and congenital heart diseases were also observed to be associated with this arrhythmia.
    (The association of parasystole with heart diseases is not definitely proven and such arrhythmia is mostly benign and self limited)
  - Some cases (14 percent) were found to be idiopathic in nature without any evidence of heart disease.
  - Since the main requisite of this arrhythmia is the presence of a secondary pacemaker in heart , any cardiac arrhythmia might work as a rich factor.
  - Sometimes its occurrence is seen with digitalis toxicity.

3. Natural history

- **Symptoms**
  - Parasystole may be felt as palpitation or at times as slowness of the heart beatings.
  - Shortness of breath due to impaired pumping of the heart either due to fast / slow heartbeats or its association with congestive heart failure.
  - Symptoms may be attributed to the concomitant heart diseases , such as chest pain (coronary heart disease)
- There are some rare incidence of ventricular tachycardia (VT) and ventricular fibrillation (VF) , reported with parasystolic arrhythmia , even without structural heart disease.

4. Mechanism – its electrophysiology

In parasystole the ventricular site is common as compared to the other sites such as the AV node or the atrium. The following points should be kept in mind while understanding the concept of ventricular parasystole :

(i) **A concept** : Ventricular Parasystole is due to the interaction between two fixed rate pacemakers having different inherent rates – first is the fast beating SA node and the second one is the slow beating ventricular parasystolic pacemaker.

(ii) **Entrance block : ** The parasystolic ventricular pacemaker is protected from the overdriven jolt of the dominant SA node and this happens by its intermittent or constant entrance block – the dominant impulses of the sinus origin fail to discharge the parasystolic pacemaker.
Such entrance block around the ventricular pacemaker is unidirectional – its exit allows the activity from this secondary pacemaker to proceed onwards. And with such onward journey there is a rim of depolarization surrounding the secondary one which works as an entrance block for the incoming impulse from the SA node.

(iii) **The dual pacemakers rhythm :** As the dual pacemakers operate asynchronously, the myocardium is activated predominantly by the faster running sinus pacemaker. This would be worthwhile to mention here that the slower pacemaker activates the myocardium whenever it finds this non-refractory.

(iv) **Variable coupling interval :** Both the pacemakers work asynchronously with its different rates and do not have any relation with each other , it explains variable coupling intervals in between the run of sinus and ectopic complexes – **noticeable variation in coupling intervals is a cardinal sign of parasystole.**

(v) **A simple mathematical relationship in between the interectopic intervals :** The longest interectopic interval is always the multiple of the shortest interectopic interval. Just to explain – the ectopic focus is discharging its impulses with its inherent rate , whether they manifest or not , and so the longer interectopic interval appears as the multiple of the shortest interval. This is also a very characteristic sign of parasystole.

(vi) **Fusion beat :** Occasional discharge from both the pacemakers (sinus and parasystole) do occur concurrently resulting in the fusion of both the impulses , and hence having a configuration in between these two.

(vii) **Exit block :** The parasystolic focus may also have exit block , during which it may fail to depolarize the myocardium.

(viii) **Modulated parasystole** : There may be an electronically active zone surrounding the ectopic parasystolic site , which can allow the rhythmic activity of the surrounding tissue to have its impact over the periodicity of the parasystolic discharge rate. In other words , the barrier of the entrance block may not be complete. Subthreshold inputs from this surrounding tissue can cross the entrance barrier to depolarize the myocardium. This modulation phenomenon may alter the timing of parasystolic stimulation – elctrotonic impact arriving at the early stage of diastolic depolarization results in a delay in the firing of the parasystolic focus , while arriving late might accelerate the discharge of the parasystolic focus.

(ix) **Parasystole of all types begins and ends spontaneously**.

NB :
Parasystolic ventricular beat differs from the coupled ventricular premature beat in the sense that the discharge from the parasystolic focus does not require a triggering impulse – it is an autonomous phenomenon.

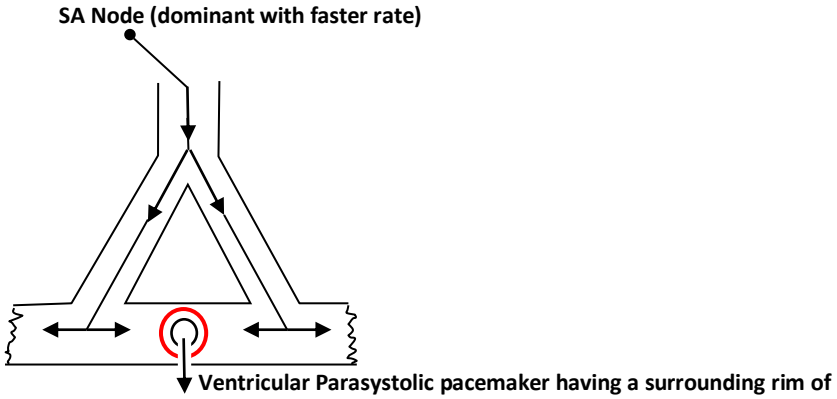

Fig. 1.1

Fig. 1.2 **A strip of standard lead II**

A = Dominant sinus beat coming from the SA node

B = Ventricular beat coming from the parasystolic focus

The distance in between **A** and **B** is the coupling interval (Variable in nature)

C = Fusion beat

5. Typical ECG findings in parasystole

(i) Variable 'coupling intervals' between sinus beats and parasystolic ventricular beats

(ii) The longest interectopic interval is almost exact multiple of the shortest basal interectopic interval

(iii) ± The presence of fusion beats

It would be a very essential step to recognize the run of sinus beats and parasystolic ventricular beats with the variable coupling interval in between these two. A caliper is needed to measure the coupling intervals variability and its associated interectopic intervals.

6. Concluding remark

- ☐ Parasystole is a dual rhythm wherein the parasytolic pacemaker is being protected from the impact of the dominant pacemaker SA node by creating an entrance block around itself. The protective entrance block is found usually situated within the immediate vicinity of the parasystolic focus.
- ☐ When the sinus and parasystolic beats co-exist , the resultant is a specific arrhythmia – known as 'Parasystole'.
- ☐ The parasystolic ventricular beats bear no constant relation to the preceding sinus beats – the coupling intervals are variable.
- ☐ The longest interectopic interval is always the multiple of the shortest basic interectopic interval.
- ☐ Fusion complex : Occasionally a concurrent discharge of both pacemakers (sinus and parasystole) do occur resulting in the fusion of both the impulses , and hence having an intermediate configuration in between these two
- ☐ The parasystolic pacemaker may be located anywhere in the heart but is commonly situated in the ventricles , less commonly in the AV node and rarely in the atria.

Parasystoles are quite benign and require no treatment. The resultant pattern on ECG at times might be puzzling which may cause a significant concern to the attending physician.

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