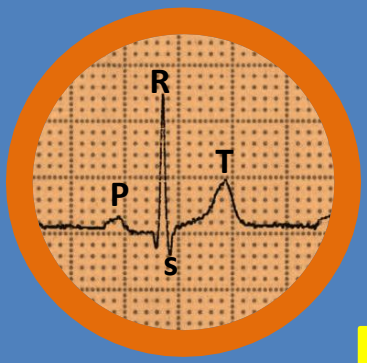


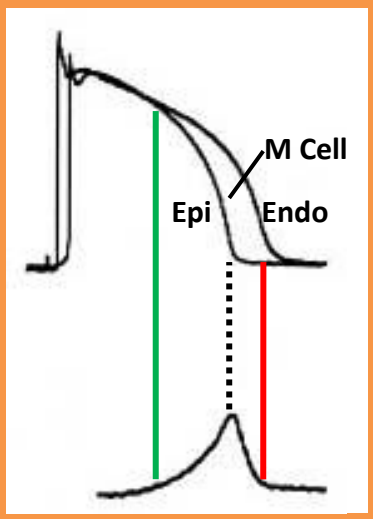
ECG Review : T-wave changes

(For Academic Purpose Only)

Dr. DURGA PRASAD KHAITAN

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

T-wave
Thou cast so many images on ECG
Uncoils so many threads of its secrecy



ECG Review : T-wave changes

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T-wave
Thou cast so many images on ECG
Uncoils so many threads of its secrecy

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 'T-wave changes' are not covered with this book. Here I am putting some of my articles related to T-wave changes. It is only a step towards the vast ocean of knowledge. I may be excused for any error or omission.

With thanks and regards

DEDICATED
TO ALL THE
FELLOW COLLEAGUES

Index

NORMAL T-WAVE – A WAY TO ITS UNDERSTANDING

ECG

NORMAL T-WAVE – A WAY TO ITS UNDERSTANDING

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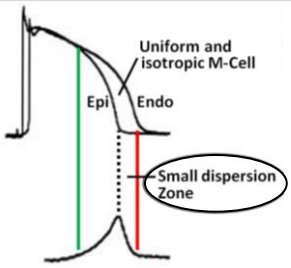
OUTLINE

Introduction
The T-wave is the repolarizing event on ECG , formed at the end of ST-segment

Electrophysiology – Genesis of T-wave
A reversed sequence of repolarization versus the sequence of depolarization

Exploring the fact leading to T-wave morphogenesis



- ☐ The myocardial cells are having uniform and isotropic conductivity, practically with no or low dispersion.
- ☐ When dispersion , or time displacement , is small , the difference will result in asymmetrical waveform (The asymmetry is due to a steeper downslope than the upslope of the wave).

Normal T-wave characteristics
Normal T-wave is slightly asymmetrical in shape, with a rounded apex that occurs closer to its end than its beginning

- ➢ Amplitude
- ➢ The T vector
- ➢ Precordial T-wave balance

Take Home Points

References

Normal T-wave – a way to its understanding

A Narrative Review

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It is a wonderful metaphor , catching a wave and analysing it with mindfulness. Waves are the voices of tides – rising and falling , it needs a sincere effort to analyse them. Understanding the nature of waves with their characteristics makes one more understandable. Every wave is a challenge unless and until we don't know about it fully.
The waves on ECG are a continuous disciplined series of depolarizing and replolarizing events. T-wave – the repolarizing wave needs a special reference.

- ☐ **The event of repolarization starts from the point where the depolarizing wave QRS gets ended – a reversed sequence.**
- ☐ **Epicardial cells repolarize earlier than endocardial cells , with its initial and rapid spread in the similar direction as that of the QRS complex – so resulting in a positive T-wave.**

There is a dire need of being acquainted with the morphology of normal T-wave , then it would be possible to differentiate it from abnormal T-waves whenever they are met with.

1. Introduction

The T-wave is the repolarizing event on ECG , formed at the end of ST-segment. When the phase of ventricular systole (coinciding with ST segment of phase 2 , as illustrated below) gets ended , there is a need of returning back to the initial polarized state of the cardiac membrane. In other words , it can be stated that this part of ventricular repolarization is the process by which the ventricular myocytes are returned to their positive resting potential , so that they can repolarize ('recharge') again.

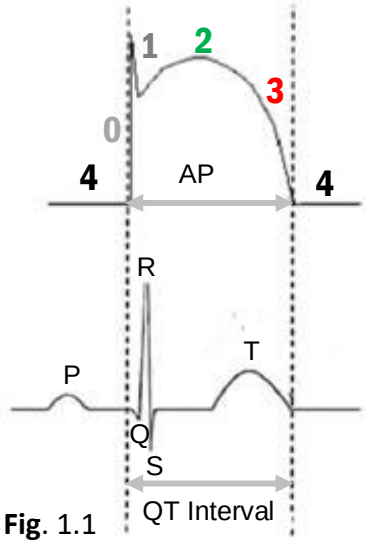
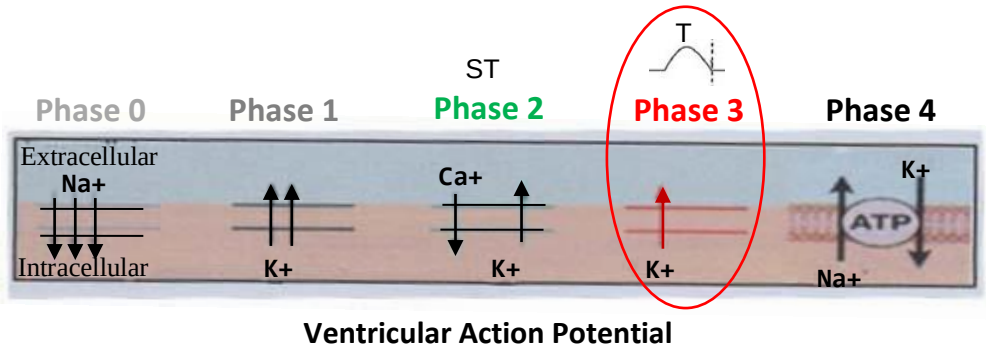


Fig. 1.1



Ventricular Action Potential

Isoelectric ST segment (Phase 2) ⟶ T-wave (Phase 3)
(The T-wave is having a somewhat shallow amplitude with wider base , compared to the corresponding QRS complex – the K+ ions outflow is slower with low potential difference in between intracellular and extra cellular compartment **during phase 3**)

Normally , there is a cessation of Ca^{2+} inflow but with the continuation of K+ ions outflow from intracellular compartment to extracellular compartment during the phase 3 , interrupting

the isoelectric ST segment due to the newly created potential difference in between intracellular and extracellular compartments – the resultant situation causes upright T wave.

2. Electrophysiology – Genesis of T-wave

Understanding by Step 1

- **The process of repolarization starts from the point where the depolarization wave QRS gets ended** . This is worthwhile to mention here that the last portion of ventricular depolarization is the posterobasal portion of the left ventricle (including the pulmonary conus and the uppermost part of the interventricular septum). That's why , the wave of repolarization starts from this posterobasal portion of the left ventricle toward the epicardial surface by propagating ahead leftward and downwards causing upward T wave in a similar positive direction as that of QRS complex.
- **Epicardial cells are normally having a shorter duration action potential than endocardial cells**. This shorter duration action potential causes epicardial cells to repolarize earlier.

This can be better understood in the light of QRS and T loop on the cardiac vectogram :

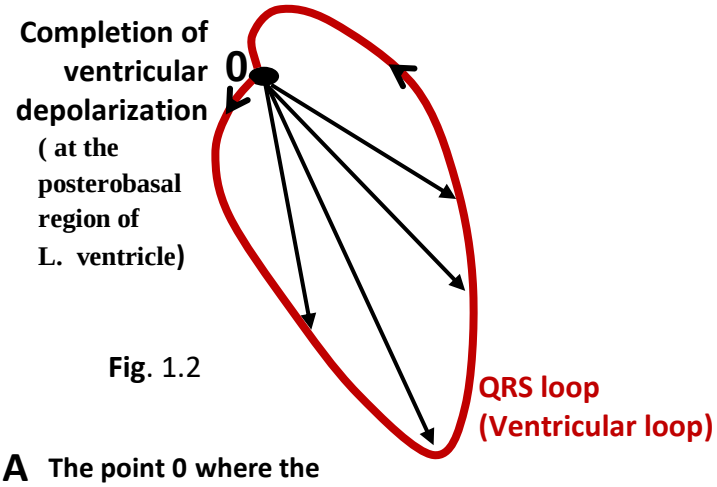


Fig. 1.2

A **The point 0 where the journey of depolarization wave QRS gets ended**

The loop of ventricular depolarization so formed is having a semicircular circumscribed shape of the electrical pathway, usually its vectorial axis pointing towards downwards and left.

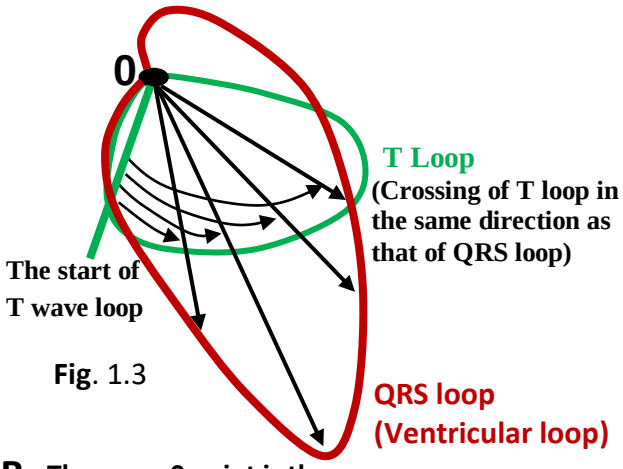


Fig. 1.3

B **The same 0 point is the start of the journey of repolarization T-wave.**

The shorter epicardial action potential causes this zone to repolarize earlier than the longer action potential in the endcoardial cells – this causes the ascending limb of the positive T-wave.

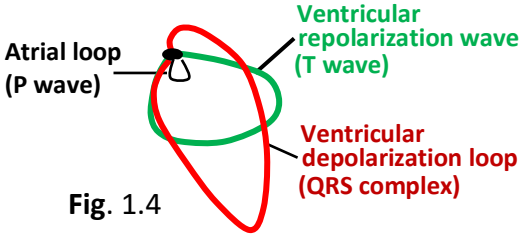
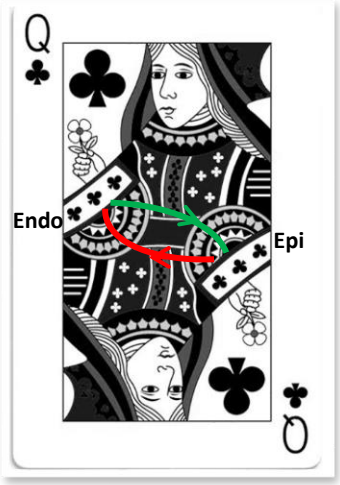


Fig. 1.4

These so formed loops are projection onto the three dimensional space of thoracic torso in real time.

Understanding by Step 2

- ☐ **The myocardium has uniform and isotropic conductivity** – so the journey of repolarization occurs smoothly and systemically.

- ☐ 

A reversed sequence of repolarization versus the sequence of depolarization

To have the concept of normal polarity of T-wave , it becomes essential to be understood the fact that the fibers that are depolarized last would be repolarized first and the fibers depolarized first would be repolarized last.

Fig. 1.5

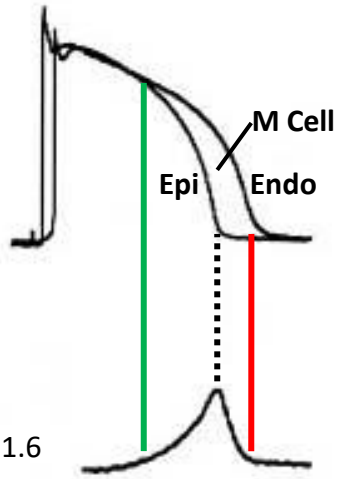


Fig. 1.6

Magnified view of Epi-Endo – action potential with myocardial cells in between

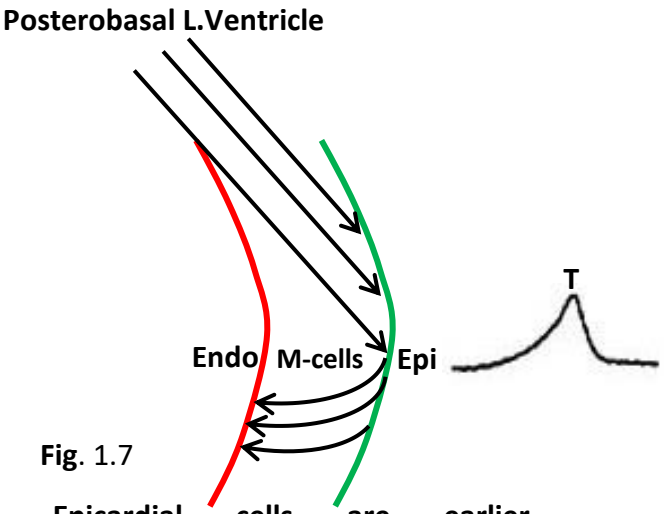


Fig. 1.7

Epicardial cells are earlier repolarized → inscribing ascending limb of T-wave → then , passing across the myocardium the endocardial cells are repolarized last → inscribing descending limb of T-wave

Any exploring lead placed towards the current flow records a positive reflection and away from the current of flow it records negative deflection. Here , the epicardial current of flow is towards the exploring electrode , it records ascending limb of the T-wave as a positive deflection and the flow of current in opposite direction (over the endocardium) records the negative deflection as the descending limb of the T-wave.

3. Exploring the facts leading to T-wave morphogenesis

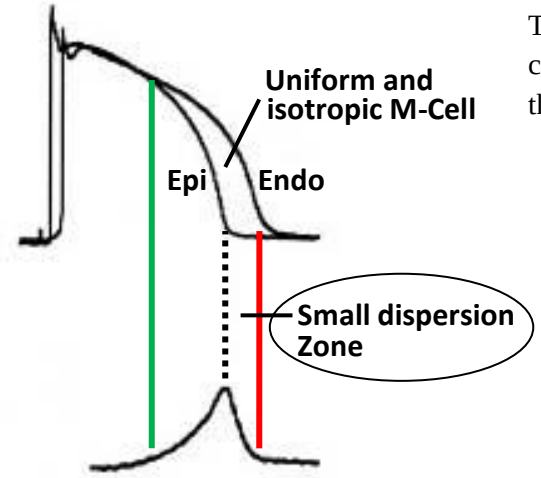


Fig. 1.8

The reason for the T-wave morphology is not so clear. The following facts are to be considered in this context.

- ☐ The myocardial cells are having uniform and isotropic conductivity, practically with no or low dispersion.
- ☐ When dispersion , or time displacement , is small , the difference will result in asymmetrical waveform, as illustrated in the preceding sketch (The asymmetry is due to a steeper downslope than the upslope of the wave).
- ☐ Normal T-wave is slightly asymmetrical in shape, with a rounded apex that occurs closer to its end than its beginning.

Ref :

Explaining the T-wave shape in the ECG

Diego di Bernardo & Alan Murray

Published: 06 January 2000

https://www.nature.com/articles/47409

4. Normal T-wave characteristics

- **Amplitude**
  - Normally , T-wave is having lesser amplitude but with a wider base compared to the corresponding QRS complex – this happens so due to the slower outflow of K+ ions with lesser potential difference across the cardiac membrane during its formation.
  - Amplitude < 5 mm in limb leads , <10 mm in precordial leads (10 mm males , 8 mm females) – usually one-tenth of the preceding R-wave amplitude.
  - The amplitude tends to diminish with advancing age , and is larger in males than in females.
  - The width of T-wave is not measured by its width at the base , QTc measurement serves the purpose.
    (QTc is directly proportional to the base-width of the corresponding T-wave)
  - The amplitude of T-wave is seen to be directly proportional to the amplitude of QRS complex in the same lead.
- **The T vector** is oriented leftward , inferiorly and anteriorly **over the frontal plane** , as illustrated by the following sketch :

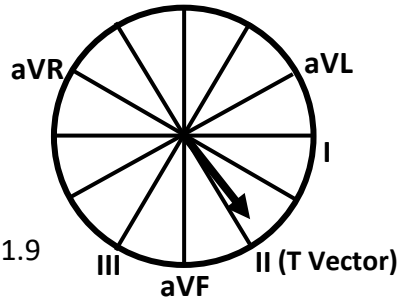


Fig. 1.9

T-wave polarity depends upon T vector whether it is more vertical or more horizontal. T-wave vector runs closer to lead II in most of the cases , therefore , T-waves are always upright in lead I and II but always inverted in lead aVR. There may be an upright or inverted T in lead III (if inverted the T-wave polarity should be tested on deep inspiration which makes it upright). In lead aVF , the T-wave is usually upright but occasionally is flat or slightly inverted.

In general , T-wave are tallest in lead II.

At times T-waves in right precordial leads V1-V3 may be inverted in some normal adults , this ECG is considered to resemble with that of normal children. This phenomena is known as **Persistant Juvenile T pattern**.

➢ **Precordial T-wave balance**

Since the repolarization starts first at the posterobasal region of the left ventricle , the T-wave polarity is tilted towards the left precordial leads i.e. T-wave polarity in lead V6 is larger than in V1. This entire concept is known as precordial T-wave balance. The following points are to be considered in this context.

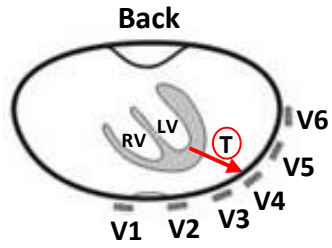


Fig. 2.0

- The T-wave in V1 is usually negative , it may be flat or biphasic (with an upright first portion and inverted second portion)
- The amplitude of T in V6 is always more than in V1
- As one proceeds towards the intermediate zone V3/V4 in alignment with T vector , usually lead V4 is having the largest amplitude compared to the other precordial leads .

Thus , the T-wave progresses normally across the precordial leads , as mentioned.

NB : Red signal – an applied concept.

Loss of precordial T-wave balance occurs when the upright T-wave in V1 is larger than that in V6. This is a type of **hyperacute T-wave**.

- The normal T-wave in V1 is inverted. An upright T-wave in V1 is considered abnormal — especially if it is tall (TTV1), and especially if it is new (NTTV1).
- This finding indicates a high likelihood of coronary artery disease, and when new implies acute ischemia

Source : CME INDIA dated 24.04.2023 by Dr. N.K. Singh , Director Diabetes and Heart research centre, Dhanbad , Editor : www.cmeindia.in

Interpretation : Positive T in V1 with more amplitude compared to that of V6. This indicates a high likelihood of coronary artery disease.

5. Take Home Points

- **A reversed sequence of repolarization versus the sequence of depolarization**
  To have the concept of normal polarity of T-wave , it becomes essential to be understood the fact that the fibers that are depolarized last would be repolarized first and the fibers depolarized first would be repolarized last.
- The myocardial cells are having uniform and isotropic conductivity, practically with no or low dispersion.
- When dispersion , or time displacement , is small , the difference will result in asymmetrical waveform (The asymmetry is due to a steeper downslope than the upslope of the wave)
- Normal T-wave is slightly asymmetrical in shape, with a rounded apex that occurs closer to its end than its beginning.

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WHAT DOES TALL T-WAVE REPRESENT ON ECG

ECG

WHAT DOES TALL T-WAVE REPRESENT ON ECG

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OUTLINE

Introduction

- Normal T-wave is usually having amplitude < 5 mm in limb leads , <10 mm in precordial leads (10 mm males , 8 mm females) – exceeding this parameter it is known as 'Tall T'.
- Upright T-wave irrespective of its amplitude in lead V1 is considered equivalent to tall T.

Causes of tall T-waves

- ☐ Normal variant , including early repolarization syndrome
- ☐ Primary repolarization abnormality
  - Hyperkalemia
  - STEMI (Hyperacute T wave)
- ☐ Secondary abnormal depolarization : LBBB , LVH
- ☐ Miscellaneous

Electrophysiology related to Tall T-wave with primary repolarizing abnormality

Enchanced electrochemical gradient by surplus K+ ions in vicinity ± an increase in regional dispersion during repolarization

Electrophysiology related to tall T-wave with secondary abnormal depolarization

Home take points

References

What does Tall T-wave represent on ECG

A Narrative Review

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A researcher's mind sees a problem through the overwhelming intricacy to find out its solution by rallying and organizing a principle – he touches the innerdepth of the concerned issue. There are many issues which need a thorough peeping through to seek out its proper explanation.

The tall T-wave on ECG has been a burning issue – it is not a ball dropped from the height but rather a big bounce from below at the level of cardiac membrane during the last phase of repolarization. There is a need to search out what determines the tall T on ECG. Let us explore determining forces working through :

- ☐ **The electrochemical gradient across the cardiac membrane created by surplus K+ ions in vicinity ± increase in the dispersion of regional repolarization.**
- ☐ **Tall T is the earliest red signal to point towards some life threatening conditions like hyperkalemia , STEMI , vasospastic angina , preinfarction state (de Winter T-wave) , etc.**

Tall T-waves may be a normal variant or secondary to abnormal depolarization (LBBB , LVH) – these issues should be kept in mind while dealing with tall T-wave.

1. Introduction

ECG is a simple and cheap modality that can impart a prewarning information towards the diagnosis of some life-threatening disorders by displaying tall T on its graph. There is no one point definition of tall T but the following facts should always be considered while dealing with tall T on ECG.

- Normal T-wave is usually having amplitude < 5 mm in limb leads , <10 mm in precordial leads (10 mm males , 8 mm females) – exceeding this parameter it is known as 'Tall T'.
- A normal isolated tall T may be present in lead II and in lead V4 by keeping its alignment with the corresponding T-vector.
  T-waves may be tall as a normal variant specially in young individuals and athletes typically in the precordial leads V2-V4.
- The amplitude of T-wave is relative to QRS : T-wave may be only relatively and not absolutely increase in amplitude
  Upright T-wave irrespective of its amplitude in lead V1 is considered equivalent to tall T .... specially if amplitude of upright T-wave in V1 is taller than V6 , or a new upright T-wave in V1 (would be wiser to compare this with old ECG if available).
- Since the T-waves are normally well recorded over the precordial leads , tall T-waves are more visible therein.

- There are many clinical entities which are associated with tall T on ECG.

History taking is a 'must' while dealing with such a situation.

- The presence of anginal pain , breathlessness , nausea , ± excessive sweating or other symptoms might be suggestive of acute coronary syndrome (STEMI) as a pointer towards this diagnosis with tall T-wave.
- The presence of known or suspected renal failure , whether on dialysis or not and review of the drugs intake (e.g. ACE inhibitors , angiotensin receptor blockers, Aldosterone antagonists , potassium retaining diuretics , etc.) may serve as important clues to the diagnosis of tall T-waves associated with hyperkalemia.

2. Causes of tall T-waves

There are many causes of tall T-waves :

☐ **Normal variant**, including early repolarization syndrome

☐ **Primary repolarization abnormality**
- **Hyperkalemia:** "peaked T waves" are diffuse, look pinched (**narrow base and sharp symmetric peak**), and are associated with other signs of hyperkalemia (eg decreased P-wave amplitude with long PR , widening of the QRS complex with finally evolution to sine wave and other conduction pathway abnormalities like suppression of SA node , different degree of AV block , bundle branch block , etc.)
- **STEMI :** "Hyperacute T waves" are regional, look to be inflated (**broad base,rounded peak, and large relative to QRS**), and are associated with other signs of STEMI (loss of R-wave , ST elevation , Q wave – as evolutionary changes)
- **Acute transient transmural ischemia** (Vasospastic angina , also known as Prinzmetal's angina)

☐ **Secondary abnormal depolarization**
- LBBB
- LVH

See Right precordial leads associated with deep / wide anterior S wave followed by ST elevation and tall T-waves , usually with left precordial ST segment depression and T-wave inversion indicating strain pattern therein.

☐ Miscellaneous (occasionally)
Acute pericarditis
Cerebrovascular hemorrhage (more commonly – T-wave inversions)

NB :

- Tall T in hyperkalemia is usually seen when the serum potassium exceeds 5.5 mEq/L
- In STEMI , hyperacute T-wave appears within 0-30 minutes of its onset.

3. Electrophysiology related to Tall T-wave with primary repolarizing abnormality

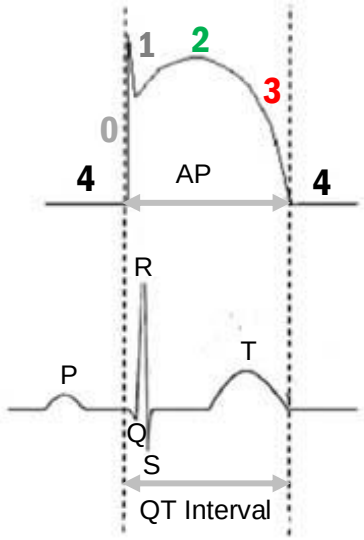
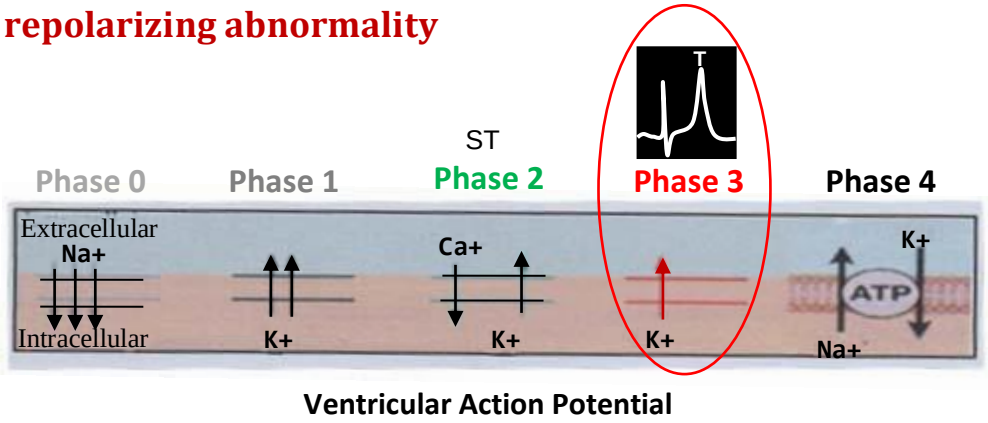


Fig. 1.1



Ventricular Action Potential

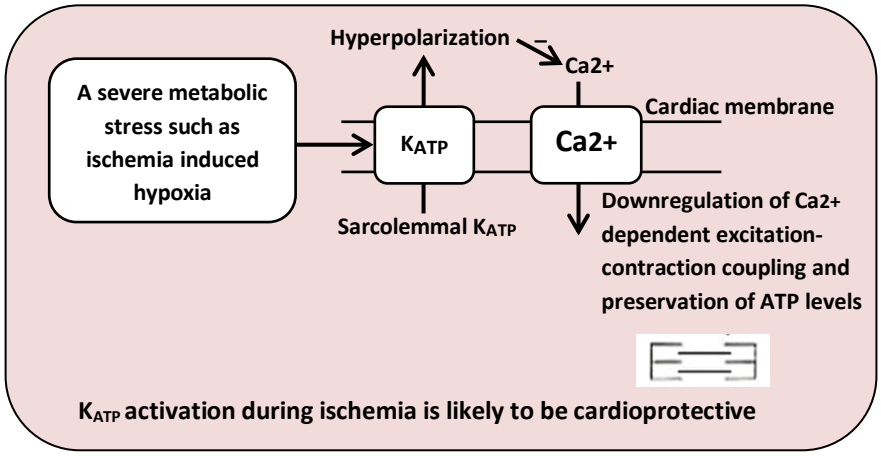
Basis :

- ☐ The cessation of Ca2+ inflow and the continuation of K+ outflow during phase 3 cause the formation of T-wave. The tallness of T-wave depends upon the **electrochemical gradient** across the cardiac membrane caused **by surplus K+ ions in vicinity** of extracellular compartment.
- ☐ T-waves become more symmetrical as a result of an **increase in regional dispersion during repolarization** of cardiac muscle.

☐ **Causes of surplus K+ ions in vicinity**

Conditions	**Factors leading to surplus K+ ion**
1. Hyperkalemia	Surplus K+ in hyperkalemia and Tall T is usually seen when the serum potassium exceeds 5.5 mEq/L
2. ST elevation MI (Hyperacute T-wave)	The resultant ischemic insult causes : • Extracellular leakage of K+ ions due to enhanced permeability of cardiac membrane. • Impairment of Na/K-ATPase pump-functioning leading to decreased transport of K+ ions from extracellular to intracellular compartment. Both these conditions lead to somewhat surplus K+ ions in vicinity.
3 de Winter T-wave Acute occlusion in the territory of proximal left anterior descending artery (LAD) with the preservation of a small rim of subepicardial tissue.	Subepicardial tissue is prevented from dying out due to the liberation of KATP (hypoxia induced) → a transient cessation of excitation-contraction coupling , lessening the O_2 demand , as reflected by rapid ST-depression (hyperpolarization) on ECG → **sudden return from hyperpolarized state to the previous repolarized state with more rapid outpouring of K+ ions** in the extracellular compartment → Surplus K+ ions in vicinity. The entire phenomenon is illustrated in the next page.

Fig. 1.2
de Winter T-wave



K_{ATP} belongs to inward rectifier channel pushing the K^+ inside the cardiac cells resulting in hyperpolarization of the cardiac membrane towards negativity, action potential ceases transiently during this period , inscribing rapid ST depression (hyperpolarization) – then , a sudden return to the previous state of repolarization with more outpouring of K+ ion in vicinity with the formation of tall T.

4. Early repolarization syndrome (ERS)

In ERS all is due to the diffuse ion channel alterations, markedly over epicardium with reorientation of cardiac channels, as below.

↓ INa ,↑Ito ,↓ICa

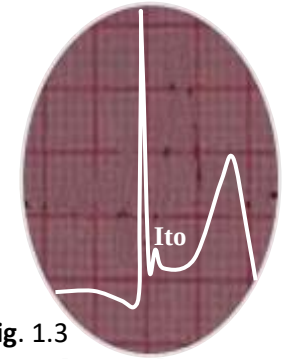


Fig. 1.3
Early repolarization syndrome

Here to mention – activation of Ito current (transient outward current) → comparatively less activation of ICa2+ (here inverse relationship with Ito and Ica current – ↑Ito - ↓ICa), with a relative decrease in the intracellular movement of Ca2+ inside, and more activation of repolarizing K+ currents with outpouring of surplus K+ ions in vicinity.

Earlier activation of Ito current with transient K+ loss during phase 1 leading to accenutation of epicardial potential notch, appearing on ECG as an accenutated J-point with concave ST-segment elevation. Here , T-wave usually 4 times the amplitude of the ST elevation.

☐ Increase in the regional dispersion during repolarization

T-wave is asymmetrical in healthy subjects , but tends to become symmetrical with diseased heart. The accurate reason for this T-wave shape alteration is not so clear but it has been observed that T-waves become more symmetrical as a result of an increase in regional dispersion during repolarization of diseased cardiac muscle.

Ref :

Explaining the T-wave shape in the ECG

Diego di Bernardo & Alan Murray

Published: 06 January 2000

https://www.nature.com/articles/47409

Combined impact of surplus K+ ions in vicinity ± increase in regional dispersion during repolarization of cardiac muscle

A. HYPERKALEMIA

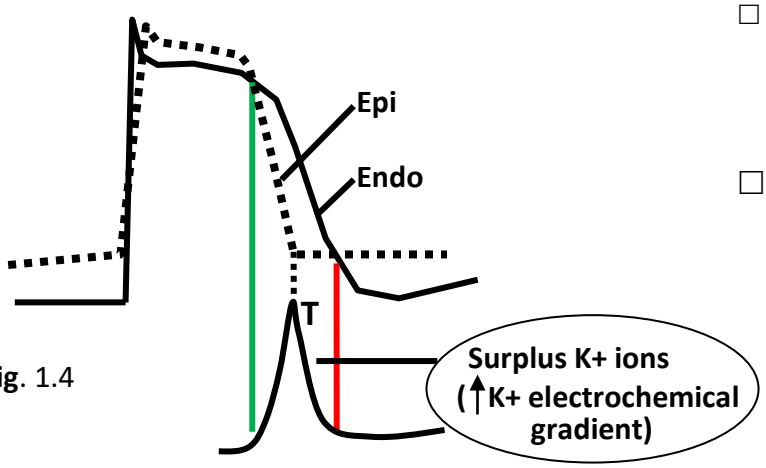


Fig. 1.4

- ☐ **Shortening of action potential duration** (↓QTc interval) → narrow base and sharp symmetric peak : such changes are diffuse on ECG.
- ☐ The **proximal limb** of T-wave is having a rapid , somewhat vertical ascent due to more K+ electrochemical gradient. The **distal limb** , assumes a somewhat more gradual descend and slope. The proximal limb of the T-wave is accordingly steeper than the distal limb , as illustrated in the preceding sketch.
- ☐ Other signs of hyperkalemia are present

B. STEMI : Hyperacute T-wave

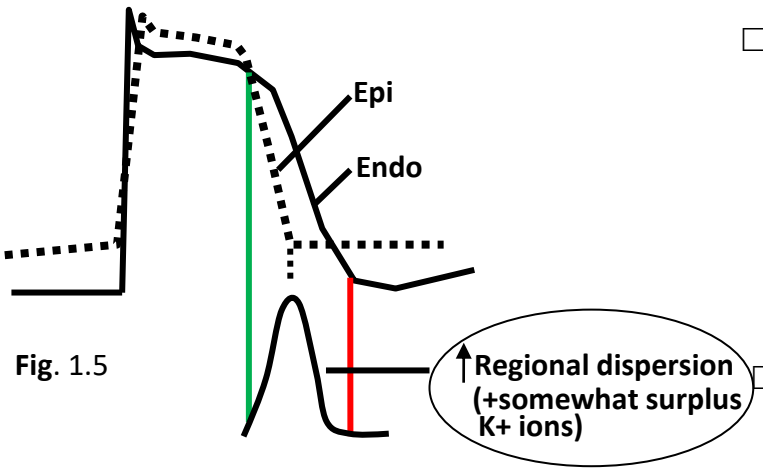


Fig. 1.5

- ☐ **Hyperacute T-wave** within 0-30 minutes of STEMI onset
- ☐ **Coronary insufficiency** with increased regional dispersion (↑QTc interval) plus somewhat surplus K+ ions in vicinity
  → tall T-wave with **broad base, rounded peak , and large relative to QRS.**
  (such changes are localized in the territory of occluded coronary artery)
- ☐ Hyperacute T-wave may be associated with ST elevation or a rapidly upsloping ST segment **(Pardee's sign)**
- ☐ **Evolving changes** on ECG such as ST elevation , Q wave are seen in successive recorded ECGs .

4. Electrophysiology related to tall T-wave
With secondary abnormal depolarization

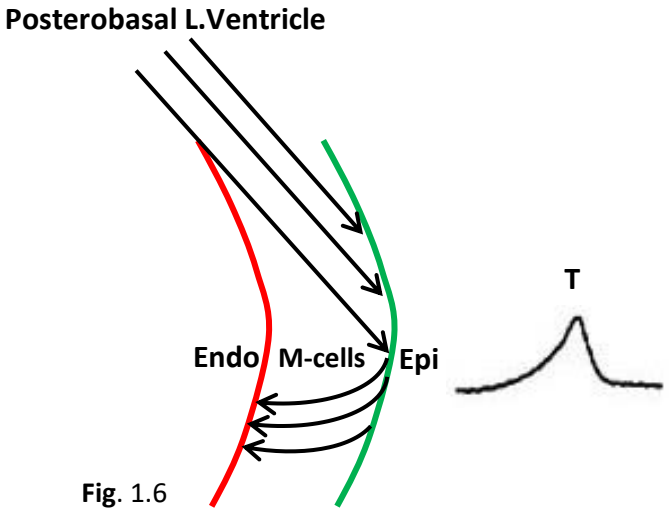


Fig. 1.6

Normally Epicardial cells are earlier repolarized ⟶ inscribing ascending limb of T-wave ⟶ then , passing across the myocardium the endocardial cells are repolarized last ⟶ inscribing descending limb of T-wave

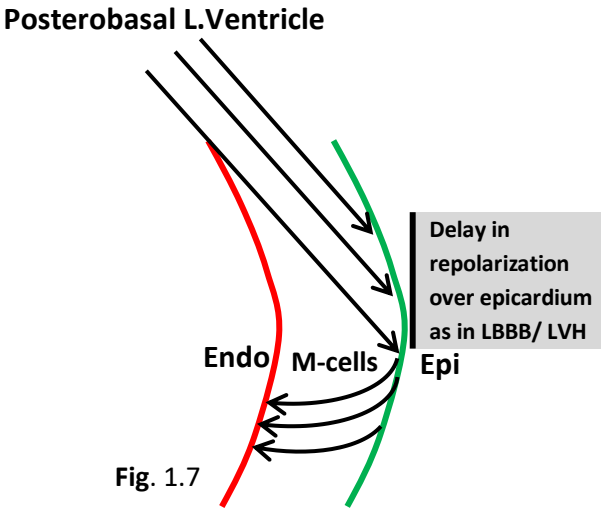
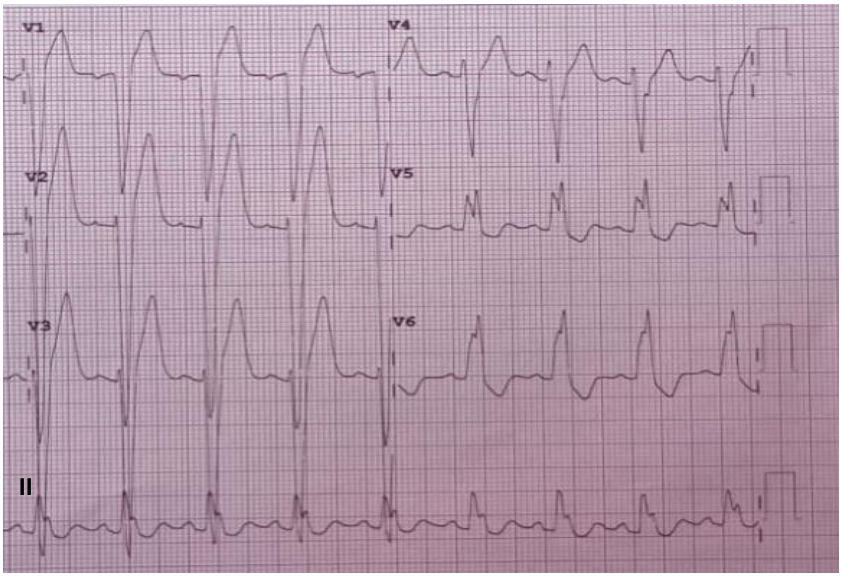


Fig. 1.7

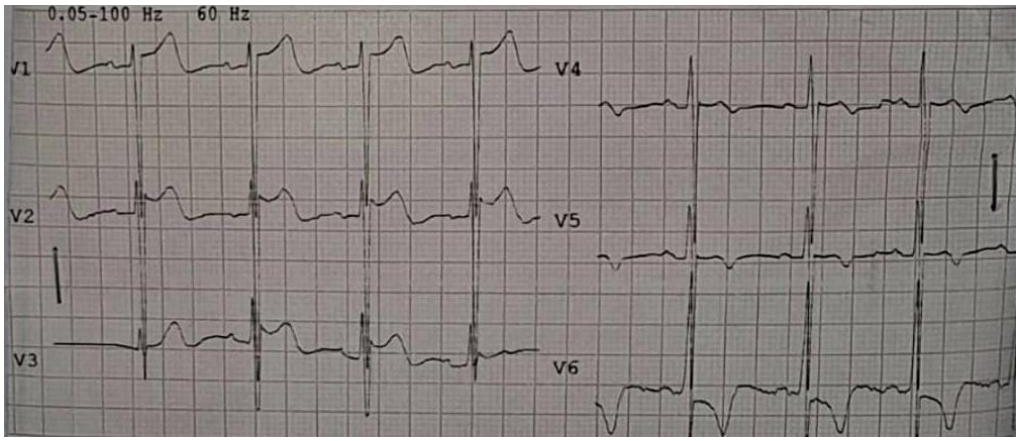
Basis :

- ☐ In LBBB/LVH , there is a delay in repolarization over epicardium
- ☐ As a consequence the repolarizing waves turn towards the endocardium instead of repolarizing epicardium first.
- ☐ Since the endocardial repolarization sequence is in the opposite direction compared to that of epicardial repolarization :
  right precordial leads are associated with deep / wide anterior S wave followed by ST elevation and tall T-waves , usually with left precordial ST segment depression and T-wave inversion indicating strain pattern therein.



Source : Dr. Bhanu Pratap Singh , Senior consultant Physician , Siwan (Bihar)

Left bundle branch block pattern is characterized by the wide notched QRS complexes in lead V5-V6 with secondary repolarization abnormality in the form of ST segment depression and T wave inversion. **The opposite pattern of a wide S wave with upsloping ST and upright tall T is seen in right precordial leads.**
(Tracings of precordial leads are only illustrated here)



Source : CME INDIA, Dated 22.12.22 by Dr. N.K. Singh , Director Diabetes and Heart research centre, Dhanbad , Editor : www.cmeindia.in

67 years old with SOB , hypertensive

This is ECG dictum that discordant ST elevation over right precordial leads V1-3 , specially with up concavity in the presence of ECG evidences in favour of LVH should be treated as a part of LVH itself unless and until disapproved **(the upright T-wave irrespective of its amplitude in lead V1 is considered equivalent to tall T)** (Tracings of precordial leads are only illustrated here)

5. Take home points

- Tall T-wave can be a normal variant (including early repolarization) , secondary to abnormal depolarization (LBBB, LVH) , or as a consequence of primary repolarizing abnormality (hyperkalemia, STEMI with hyperacute T-wave).
- Secondary abnormal depolarization if any such as LBBB or LVH should be excluded first – see below.
  Right precordial leads associated with deep / wide anterior S wave followed by ST elevation and tall T-waves , usually with left precordial ST segment depression and T-wave inversion indicative of strain pattern.
- In hyperkalemia , there are “peaked T waves” which are diffuse, look pinched (narrow base and sharp symmetric peak), and are associated with other signs of hyperkalemia.
- ‘Hyperacute T-waves’ in STEMI are regional , look to be inflated (broad base, rounded peak, and large relative to QRS), and are associated with other signs of STEMI (loss of R-wave , ST elevation , Q wave , etc).
  Hyperacute T-waves are not always tall and small T-waves can still be hyperacute when paired with the corresponding low amplitude QRS complex.

Except in Hyperkalemia , abnormality in tall T-wave alone is not considered a justified diagnostic clue of any particular condition. The associated T-wave abnormalities can provide added assistance to support the clinical diagnosis and the vice-versa is also true.

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THE INVERTED T-WAVE : PRIMARY OR SECONDARY ?

ECG

THE INVERTED T-WAVE : PRIMARY OR SECONDARY ?

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OUTLINE

Introduction : Keypoints

Primary T-wave abnormalities are the direct change in myocardial cellular electrophysiological pattern.

In **secondary repolarization abnormalities** the basic defect is the abnormal depolarization of the ventricle , which causes the ventricular repolarization to be abnormal as well.

Condition with secondary repolarization abnormalities

- ☐ Conduction pathway abnormalites
  - Bundle branch block (RBBB , LBBB)
- ☐ Ventricular hypertrophy (RVH , LVH)
- ☐ WPW syndrome
- ☐ Beats as ventricular extrasystole
- ☐ Ventricular pacing

Concerned Electrophysiology

Basic cardiac electrophysiology in a normal person

Primary T-wave abnormalities (ischemia or injury) are due to a direct change in myocardial cellular electrophysiological pattern.

Secondary T-wave abnormalities (as in case of bundle branch block or ventricular hypertrophy) are due to alteration of sequence of ventricular activation pattern

How to differentiate between Primary and Secondary T inversion

Concluding remark

References

The inverted T-wave : Primary or Secondary ?

A Narrative Review

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Any group of 'BioTissue' is having a basic signal of its malfunctioning - telling the story of its causation which may be directly and closely related to its site. This might be termed as primary phenomenon. And whenever the signal of such malfunctioning is noticed coming from elsewhere not indicative of direct involvement of such tissue – might be termed as secondary phenomenon.

T-waves represent the last phase of ventricular repolarization , being graphed on ECG as a positive deflections. At times these waves are inverted – it remains one of the major diagnostic challenges to reveal its site of causation. Such T inversions are not always a potential red flag on the ECG.

- ☐ **Primary T-wave abnormalities (ischemia or injury) are due to a direct change in myocardial cellular electrophysiological pattern.**
- ☐ **Secondary T-wave abnormalities (bundle branch block or ventricular hypertrophy) are due to alteration of sequence of ventricular activation pattern.**

Both the phenomena are having the inverted T-waves on ECG – the primary ones are usually having sinister prognosis. It become essential to differentiate in between these two – remembering the fact that double may be the way of such ventricular repolarization malfunctioning depending upon the causative site.

1. Introduction : Keypoints

- T-waves represent the last phase of ventricular repolarization as positive deflections, and are normally inverted in leads aVR , V1 and at times in lead III.
- T-wave inversions (TWI) have a wide spectrum of its causation – some of them may be life-threatening such as acute coronary ischemia.
- The presence of T-wave inversion (TWI) on 12 lead ECG is one of the major diagnostic challenges to the clinicians.
  T-wave inversions are not always harbinger of potential alarming conditions. It may need a proper evaluation , indicative whether they are primary or secondary events.
  Primary T-wave abnormalities are the direct change in myocardial cellular electrophysiological pattern.
  In **secondary repolarization abnormalities** the basic defect is the abnormal depolarization of the ventricle , which causes the ventricular repolarization to be abnormal as well.
- Taking history is always rewarded in such a situation , as outlined below.
  - Case / gender / age
  - Clinical presentation

- The pattern of T-wave inversions on ECG with noting any extra ECG pointers if any.
- Investigation modalities like enzymes study , cardiac echo , even at times coronary angiograms, etc.

2. Conditions with secondary repolarization abnormalities:

Needless to say that there are so many causes of primary T-wave abnormalities (not mentioned here) but there is necessity of enumerating the secondary causes of T-wave inversions , which need to be evaluated properly. The recognition of this phenomenon helps a lot to the clinicians to minimize unnecessary hospital admission , cardiac testing and cardiac catheterization.

- ☐ Conduction pathway abnormalites
  - Bundle branch block (RBBB , LBBB)
- ☐ Ventricular hypertrophy (RVH , LVH)
- ☐ WPW syndrome
- ☐ Beats as ventricular extrasystole
- ☐ Ventricular pacing

Words of caution

This should always be kept in mind that TWI might be a potential red flag particularly on the ECG of younger healthy athletes. This imparts a prehand warning to have investigations to exclude inapparent cardiomyopathies with this group of persons. It may be the possibility that later on with coming years the person concerned may have evident picture of cardiomyopathy with adverse outcome.

3. Concerned Electrophysiology

Basic cardiac electrophysiology in a normal person

There is a continuous running of the flow of current as depolarization and repolarization phenomena , recorded on ECG as P-QRS-T waves in a sequential pattern.

This would be worthwhile to mention the following facts here in relation with the last phase of repolarization (the basis of T-wave genesis) :

- ☐ **The process of repolarization starts from the point where the depolarization wave QRS gets ended**. The last portion of ventricular depolarization is the posterobasal portion of the left ventricle (including the pulmonary conus and the uppermost part of the interventricular septum). That's why , the wave of repolarization starts from this posterobasal portion of the left ventricle toward the epicardial surface by propagating ahead leftward and downwards causing upward T wave in a similar positive direction as that of QRS complex.
- ☐ **Epicardial cells are normally having a shorter duration action potential than endocardial cells**. This shorter duration action potential causes epicardial cells to repolarize earlier.

The entire concept has been illustrated with sketches , as below :

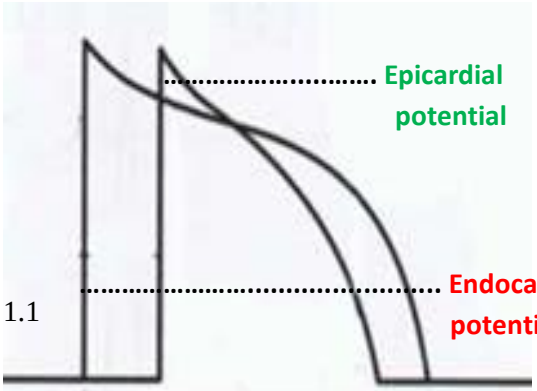


Fig 1.1

Epicardial cells are normally having a shorter duration action potential compared to endothelial cells → early repolarization of epicardial cells

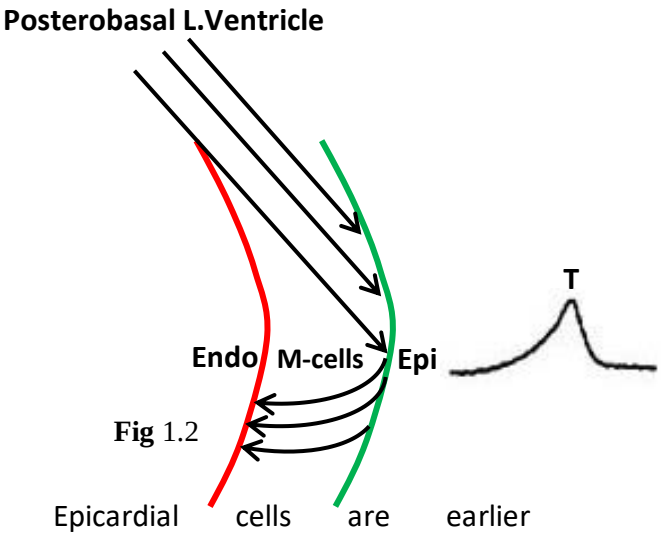


Fig 1.2

Epicardial cells are earlier repolarized →inscribing ascending limb of T-wave → then , passing across the myocardium the endocardial cells are repolarized last →inscribing descending limb of T-wave

Any exploring lead placed towards the current flow records a positive reflection and away from the current of flow it records negative deflection. Here , the epicardial current of flow is towards the exploring electrode , it records ascending limb of the T-wave as a positive deflection and the flow of current in opposite direction (over the endocardium) records the negative deflection as the descending limb of the T-wave.

Primary T-wave abnormalities (ischemia or injury) are due to a direct change in myocardial cellular electrophysiological pattern.

☐ **Coronary artery is the main distributor of oxygen and nutrients to the heart , its subtotal occlusion may lead to the myocardial ischemia** : subendocardium is more vulnerable to ischemia due to the systolic compressive effect on coronary circulation.

- Subendocardium is having its proximity to the high pressure LV chamber.
- The distance from the epicardial coronary arteries that penetrate to the endocardium have to travel a longer distance to pass through the high pressure left LV chamber –compressing these blood vessels.

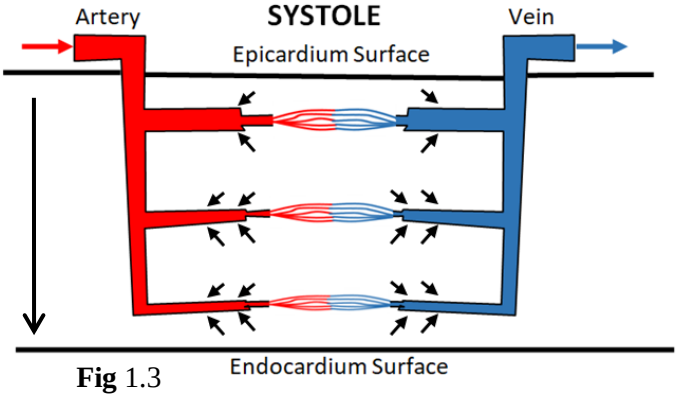


Fig 1.3

☐ **The resultant subendocardial hypoxia** causes the current of injury over the subendocardial zone (negatively charged) → the current of flow is from subepicardial region towards subendocardial region →T-wave inversion with ST segment depression.

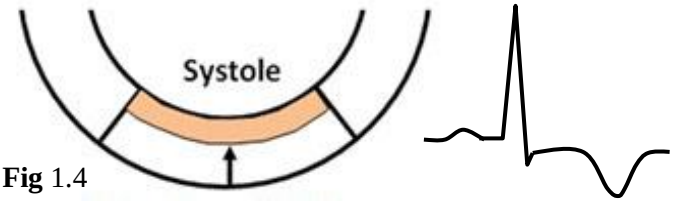


Fig 1.4

Subendocardial ischemia

Secondary T-wave abnormalities (as in case of bundle branch block or ventricular hypertrophy) are due to alteration of sequence of ventricular activation pattern.

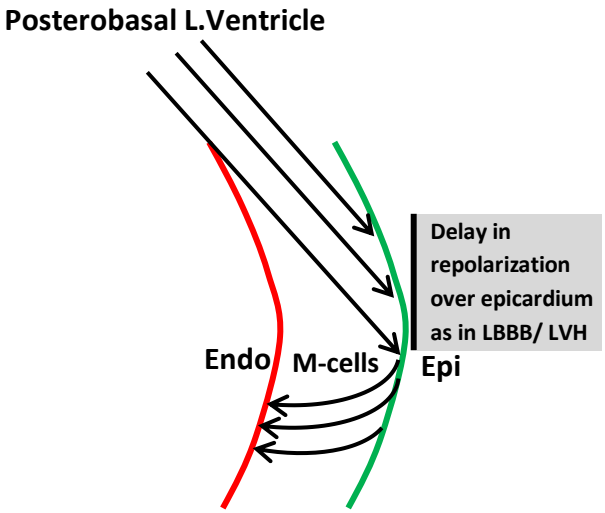
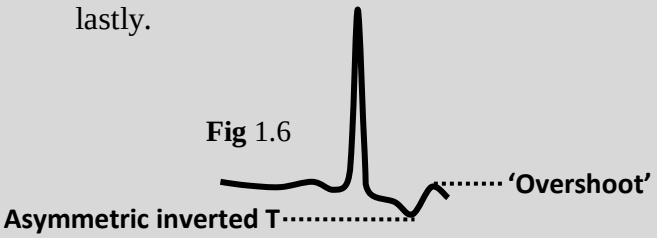


Fig 1.5

Basis :

- ☐ In LBBB/LVH , there is a delay in repolarization over epicardium
- ☐ As a consequence the repolarizing waves turn towards the endocardium instead of repolarizing epicardium first.
- ☐ Since the endocardial repolarization sequence is in the opposite direction , compared to that of epicardial repolarization :
  Left sided ST segment depression and T-wave inversion indicating strain pattern therein.
- ☐ The T-waves are inverted with an asymmetric morphology with a gradual initial downslope , a steep abrupt return to the baseline.
  With terminal 'overshoot' , possibly due to the repolarization of the remaining epicardium lastly.

Fig 1.6



NB :

- QRS morphology would be abnormal with secondary T-wave inversions. The morphology of QRS abnormality would depend upon the nature of involvement – conduction pathway abnormalties , ventricular hypertrophy , WPW syndrome , spells of ventricular extrasystoles , ventricular pacing , etc. These morphologies are well known to the clinicians and need not to be mentioned here.
- The consideration whether the TWI is secondary to abnormal depolarization.
  LBBB and LVH result in discordant ST depression and TWI in the left sided leads , while RBBB and RVH results in discordant ST depression and TWI in right sided leads.
- This is worthwhile to mention here that a patient's risk of sinister event is low in secondary T-wave inversion.
- Whenever there is a T-wave abnormality on ECG with a low probability of cardiac ischemia , the ECG should be repeated with breath held at the end of inspiration and expiration both. This is to be noted that change in the polarity of T-wave with respiration is a physiological response , which indicates towards a non-cardiac cause.

4. How to differentiate between primary and secondary T inversion

Primary T-wave inversion	Secondary T-wave inversion
ECG changes  **Fig** 1.7 • Normal QRS morphology • ST / T changes represent primary ischemia ST depression : horizontal or downsloping (ongoing angina) T changes : symmetrical inversions with somewhat arrow tip. (symmetrical T-wave inversion is due to localized involvement of coronary artery – not travelling the longer distance) , its evolution is somewhat rapid.	**ECG changes**  **Fig** 1.8 • Altered QRS morphology , depending upon the nature of secondary involvement. • ST / T changes represent secondary strain pattern. ST segment usually associated with QRS abnormality. The T-waves are inverted with an asymmetric morphology with a gradual initial downslope , a steep abrupt return to the baseline with a rounded tip. With terminal 'overshoot' , possibly due to the repolarization of the remaining epicardium.

These ST and T changes should always be analyzed in the light of clinical history.

NB :
When LBBB / RBBB is associated with upright T-waves or disproportionate ST depression and deeper pointed TWI in the concerned leads , these indicate superimposed ischemia.

5. Concluding remark

- Several clinical entities might be associated with inverted T-wave.
- This would be better to classify these T-wave inversions whether they are primary or secondary.
  Broadly to say , primary repolarizing abnormality is said to exist if the preceding depolarization (QRS complex) is normal in shape.
  Secondary if the QRS complex is abnormal.
  Horizontal or downsloping ST depression with symmetrical T-wave inversions having somewhat arrow tip represents primary ischemia or injury.
  Asymmetrical ST segment depression with inverted asymmetrical T-wave inversion having a gradual initial downslope and steep abrupt return to the base line ; which may be associated with 'overshoot' possibly due to repolarization of the remaining epicardium lastly.

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DEEP T-WAVE INVERSIONS ON ECG : A CONSIDERATION IN BRIEF

ECG

DEEP T-WAVE INVERSIONS ON ECG : A CONSIDERATION IN BRIEF

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OUTLINE

Deep T-wave inversions on ECG : A consideration in brief

A Narrative Review

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There are so many happenings in nature , which make a man surprised. A small child may cry by seeing an inverted image in water. It is a well-known phenomenon that the image of an object in water looks to be inverted i.e. the object's top side image forms on the bottom side and the bottom side on the top side. It appears so due to somewhat bending and conversing of light rays as passing through the object into the depth of water , a denser medium than air.

T-wave inversion though surprising but is based on a disciplined electrophysiological principle – here the platform for passing the cardiac impulses is transmural thickness of ventricular wall , which is having a layering of myocytes in between epicardium and endocardium.

- ☐ **Epicardial cells repolarize earlier than endocardial cells , with its initial and rapid spread in the similar direction as that of the QRS complex – so resulting in a positive T-wave.**
- ☐ **When the sequence of repolarization is reversed i.e. endocarium repolarizes first through the heightened electrogradient across the ventricular wall with surplus negative potential towards the endocardium and / or with conduction delay , it results in deep T-wave inversion.**

Clinicians seek through the deep inverted T-waves on ECG – a search of its causation so that the situation may be handled properly.

1. Introduction : Keypoints

- T-waves represent the last phase of ventricular repolarization as positive deflections, and are normally inverted in leads aVR , V1 and at times in lead III.
-  **Deep T-wave inversion is defined when the voltage of T-wave inversion is ≥ 5 mm**. The amplitude of T-wave may vary , sometimes ≥ 10 mm , then it is known as giant T-wave inversion.
- Deep T-wave inversions have a wide spectrum of its causation – some of them may be with immediate life threatening impact such as acute coronary ischemia, pulmonary embolism , with CNS injury , etc.
- The presence of deep T-wave inversion on 12 lead ECG is one of the major diagnostic challenges to the clinicians and it may harbinger potential alarming conditions , which need a proper evaluation.
- Exploring the history in details , including the family history is very much of significance in this context.

- The presence of extra ECG findings such as evidence of ventricular hypertrophy , bundle branch block , $S_1Q_3T_3$, etc. may help in making its electrocardiographic diagnosis.
- Apart from 12 lead ECG some other investigation modalities such as enzymes study , cardiac echo , even at times coronary angiography , sometimes higher sophisticated tests might be needed in establishing the diagnosis.

2. The electrophysiological mechanism

Points to be considered :

☐ Epicardial cells repolarize earlier than endocardial cells , with its initial and rapid spread in the similar direction as that of the QRS complex – so resulting in a positive T-wave.

☐ **When Endocardium repolarizes first**

↓

Deep T-wave inversion
(the reasoning below)

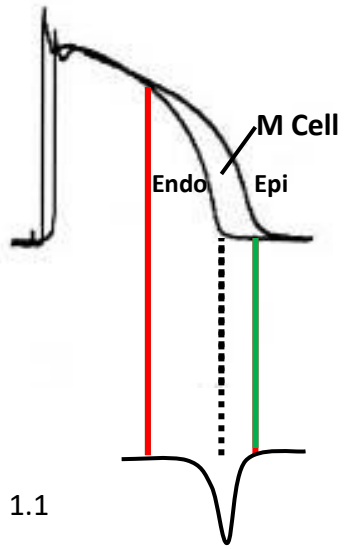

Fig. 1.1

The reasoning for deep T-wave inversion

Unusual transmural stress over the ventricular wall either from the causes inside the ventricles or over the transmural myocytes itself is the basic factor for causing reversal of the phenomenon of repolarization. The burnt of this unusual stress is maximum over the subendocardium creating more surplus negative potential over this zone resulting in enhanced electrogradient across the ventricular wall , which causes the flow of current from the epicardium towards the endocardium with deep T-wave inversion.

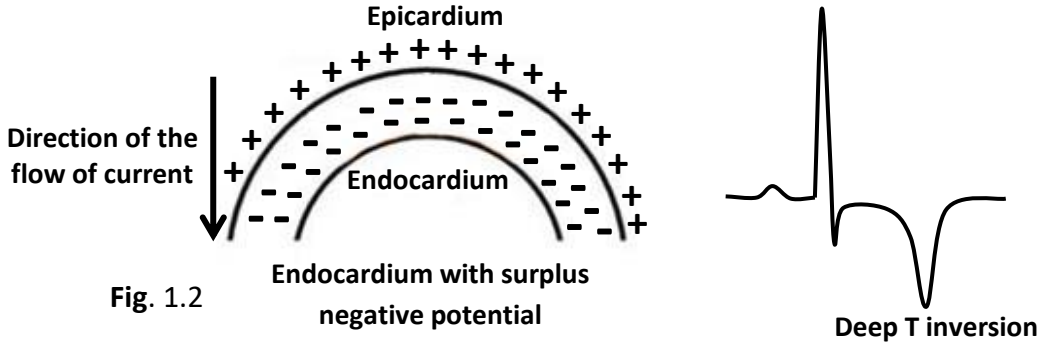

Fig. 1.2

3. Mechanism of deep T-wave inversion with some of the important electrocardiographic entities as examples :

- **Enhanced ventricular diastolic filling with ventricular distension** , as with **Takotsubo cardiomyopathy with apical ballooning** (hypokinetic apex ± hyperkinetic base) , **intermittent complete heart block with Stokes-Adams syndrome** (a slow heart rate with prolongation of ventriclular diastolic filling time with subsequent ventricular distension) , **Athletes heart** (usually associated with sinus bradycardia causing the same enhancement in diastolic filling time , associated with others findings such as LVH)

- **Transmural ventricular wall stress due to the followings :**

  A. Left or right ventricular overload
  - Typical patterns (formerly referred to as "strain" patterns)
  - Apical hypertrophic cardiomyopathy (Yamaguchi syndrome)
  - Chronic or acute pulmonary thromboembolism

  B. Hyperadrenergic drive
  As observed in the following conditions :
  - Cerebral causes (head injury with subarachnoid or intracerebral haemorrhage , post-epileptic status or even coma)
  - Takotsubo cardiomyopathy

  C. Acute myocardial ischemia may slow the propagation of cardiac impulse with takeover by endocardium.

  D. Reperfusion of STEMI reperfusion injury resulting in inversion of T-wave with progressive deepening as evolution progresses over time. This T-wave inversion is not indicative of new ischemia.
  Wellens' Syndrome is also one of the examples of reperfusion injury.

  E. Cardiac remodelling as an early sign of an underlying concealed structural heart disease or life threatening arrythmogenic cardiomyopathies.

- **Secondary T wave alternations** : bundle branch blocks, Wolff-Parkinson-White patterns , Ventricular pacing.

- **Idiopathic global T wave inversion syndrome**
  Deep T inversion in most leads except aVR. In some cases it is really idiopathic in nature but it may also be seen with acute myocardial ischemia, cerebral-T, hypertrophic cardiomyopathy, cocaine use, myocarditis, Takotsubo cardiomyopathy , pulmonary embolism, advanced atrioventricular block , etc.

4. An approach to its diagnosis

It would be worthwhile to mention here that only the presence of deep T-wave inversions on ECG is not suffice to diagnose the very cause of its associated electrocardiographic entities – it requires the supplementation by the proper history and in addition witnessing the extra ECG pointers to diagnose this condition. It is equally important to see which leads are showing deep T-wave inversions – usually anterior leads (right-sided V1-3 or more anterolateral leads V4-6) and the changes may be extended to inferior leads II , III and aVF as well.

The next consideration should be whether TWI is secondary to abnormal depolarization as with cases of bundle branch block or ventricular hypertrophy – left or right.

In brief , the diagnostic modality in such cases should include.

- Clinical history in details with stepwise clinical presentation.
- Age / Gender
- Deep TWI in what leads on ECG
- Whether TWI is secondary to abnormal depolarization
- Extra ECG pointers suggestive of any particular pathology
- Diagnostic modalities , as per need of the case.

It would be better here to discuss some important electrocardiographic entities associated with deep TWI and their associated highlighted points

Electrocardiographic entity	Highlights
☐ **Coronary artery disease**	History of anginal pain but sometimes absent in diabetics and adult population.
• Acute coronary insufficiency	Sometimes presented as deep TWI over the involved anatomical territory of the coronary artery.
• Wellens' syndrome (preinfarction state with a critical occlusion of proximal LAD artery) – reperfusion injury	Biphasic T-wave or deeply inverted T-waves commonly seen on V2 and V3 without the evidence of acute anterior myocardial infarction (without Q wave with isoelectric or minimally elevated ST segment <1 mm with the absence of precordial poor R wave progression) . Normal or slightly elevated cardiac enzyme.
• Reperfusion of STEMI	T wave inversion with its progressive deepening as evolution progresses over time.

☐ **Apical hypertrophic cardiomyopathy (Yamaguchi syndrome)** Mainly observed in younger generation with positive family history.	Left ventricular hypertrophy – not solely explained by abnormal ventricular loading , symmetrical deep TWI (apicolateral leads) , sometimes even with giant T inversion. Echocardiography plays a very important role in its diagnosis
☐ **Pulmonary thromboembolism**	Acute onset of breathlessness and chest pain. On ECG the presence of sinus tachycardia in majority of the cases with or without the followings – singly or in combination. RBBB , right ventricular strain pattern , right axis deviation , prominent R wave in V1 , right atrial enlargement (P pulmonale) , $S_1Q_3T_3$ pattern , atrial tachyarrhythmias **All the findings are not present on ECG.**
☐ **Takotsubo cardiomyopathy** (Stress cardiomyopathy) Majority of the cases in post menopausal women , usually over 60 years. Hypokinesia at the apex with ballooning out and $\pm$ hyperkinesia at the base.	History of emotional stress or physical stress ST elevation during the initial stage but dynamic and diffuse T-wave inversions lasting for a few days are the most consistent ECG finding. Q waves might be there due to associated apical left ventricular dyskinesia (D/D STEMI)
☐ **Cerebral T** (causes as discussed before) Elevated levels of circulating catecholamines and excessive sympathetic stimulation leading to cardiomyocytolysis.	History of cerebral causes with diffuse / giant T-wave inversions
☐ **Secondary T-wave alterations**	Presence of voltage criteria in case of ventricular hypertrophy ECG evidence of LBBB or RBBB. Wolff-Parkinson-White syndrome with short PR interval , delta wave, secondary ST and T changes.

5. Concluding remark

- **Deep T-wave inversion is defined when the voltage of T-wave inversion is $\geq$ 5 mm**. The amplitude of T-wave may vary , sometimes $\geq$ 10 mm , then it is known as giant T-wave inversion.
- When the sequence of repolarization is reversed i.e. endocarium repolarizes first through the heightened electrogradient across the ventricular wall with surplus

negative potential towards the endocardium and / or with conduction delay , it results in deep T-wave inversion.

- Only the presence of deep T-wave inversions on ECG is not suffice to diagnose the very cause of its associated electrocardiographic entities – it requires the supplementation by the proper history and in addition witnessing the extra ECG pointers to diagnose this condition.
- It is equally important to see which leads are showing deep T-wave inversions – usually anterior leads (right-sided V1-3 or more anterolateral leads V4-6) and the changes may be extended to inferior leads II , III and aVF as well.

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PSEUDONORMALIZATION OF T-WAVE : AN INNER PERSPECTIVE INSIGHT

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PSEUDONORMALIZATION OF T-WAVE : AN INNER PERSPECTIVE INSIGHT

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OUTLINE

Pseudonormalization of T-wave : an inner perspective insight

A Narrative Review

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The outer smile may hide one's inner grief. A voice murmuring from the inner truth might be overlooked ..... a pseudoface runs with pseudomimicking as if true is the smile. Any expression that does not correlate fully with the existing situation , is not a real but a pseudo projection. It needs a proper consideration towards its analysis. The pseudonormalcy may impart a temporary feeling of its being normal - if such a situation remains careless , some sinister happening might take place.

Pseudonormalization of T-wave during anginal symptoms may befool the clinicians.

- ☐ **Occasionally negative T-waves on ECG with Chronic myocardial ischemia may have a complete reversal to positive T-waves during acute anginal episode – this phenomenon is known as 'Pseudonormalization of T-wave'.**
- ☐ **This reversal is typically associated with severally narrowed / totally occluded coronary arteries.**

These patients need to be treated on urgent basis with aggressive medical treatment and percutaneous coronary intervention.
A way of its better understanding would create a new horizon for its early recognizition and management accordingly.

1. Introduction : Keypoints

- **'Pseudonormalization of T-wave'** is a rare entity which causes pre-existing abnormal negative T-waves to its complete reversal to positive T-waves during acute anginal episode.

A Pre-existing inverted T-wave with chronic myocardial ischemia

B Reversal to upright T-wave due to superimposition of acute ischemia upon the previous

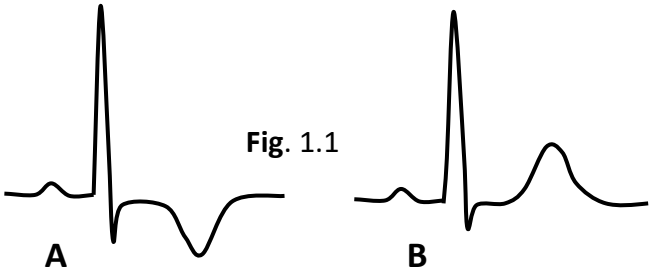

Fig. 1.1

T-wave is delusively upright – appearing to be normal , but it is 'pseudo' normal.

- 'Spontaneous pseudonormalization (PN) is a unique 12-lead electrocardiography (ECG) finding which has been reported to be associated with severe, transmural myocardial ischemia'.

Ref : Pseudonormalization: clinical, electrocardiographic, echocardiographic, and angiographic characteristics
https://jag.journalagent.com/anatoljcardiol/pdfs/AnatolJCardiol_7_1_175_177.pdf

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- This phenomenon was first detected and defined in the year 1970s , when the facility of continuous electrographic recording became available.
- The clinical history is a 'must' , which should be suggestive of true anginal symptoms on the background of pre-existing abnormal inverted T-wave.
- The significance of T-wave pseudonormalization is still not so clear. There are so many clinical studies showing its implication in the diagnosis of ischemic heart disease To date , some data are available to support this concept.
- If such situation is not recognized in time , it would quickly lead to irreversible myocardial loss.

2. Electrophysiological mechanism of T-wave pseudonormalization

The exact mechanism of T-wave pseudonormalization is not yet fully clear. This is supposed to be the result of superimposition of acute myocardial ischemia over chronic ischemic injury. This impact affects the myocardial action potential steps accordingly during the repolarization phase of T-wave genesis.

Normally epicardial cells are having a shorter duration action potential , compared to endocardial cells and so these epicardial cells are earlier repolarized resulting in upright T-wave.
But the mechanism of repolarization differs in case of pre-existing inverted T-wave and also with upright T-wave during pseudonormalization phenomenon.
Whichever endocardial or epicardial cells are having the shorter duration of action potential would be the site to be repolarized first.
Both the events are illustrated below :

A. Electrophysiological changes with pre-existing chronic myocardial ischemia

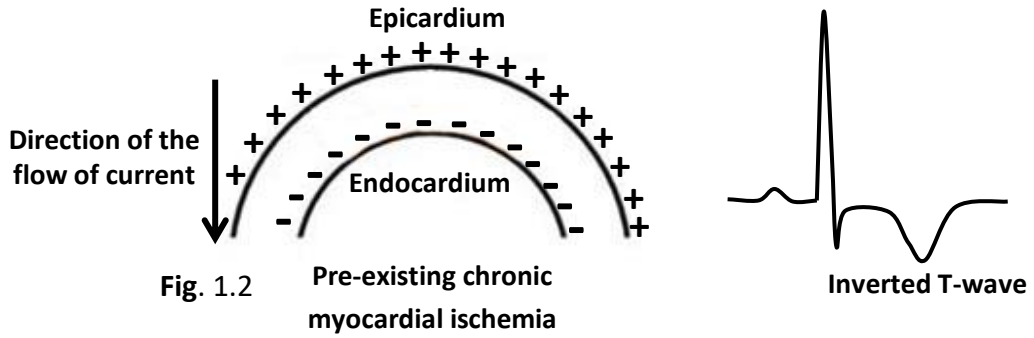

Fig. 1.2 Pre-existing chronic myocardial ischemia

- With such existing chronic myocardial ischemia (malfunctioning zone) there is a somewhat delayed repolarization over epicardium – positive charge over epicardium and negative charge over endocardium resulting in inverted T-wave.
  (The details are on the next page)

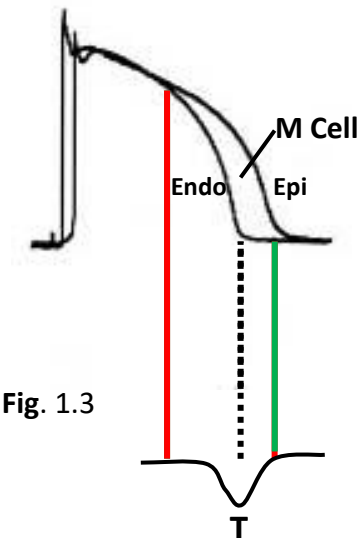

Fig. 1.3

The following points are to be considered here :

- ☐ In a patient with pre-existing chronic myocardial ischemia – the action potential duration in the epicardial cells is somewhat delayed and prolonged , compared to the endocardium . In other words , endocardial cells becomes the first site of repolarization
- ☐ Positive charge over epicardium and negative charge over endocardium → the current of flow is from epicardial region towards endocardial region →T-wave inversion.

Endocardium is the first site of repolarization

B Electrophysiological changes during acute anginal episode (Pseudonormalization of T-wave)

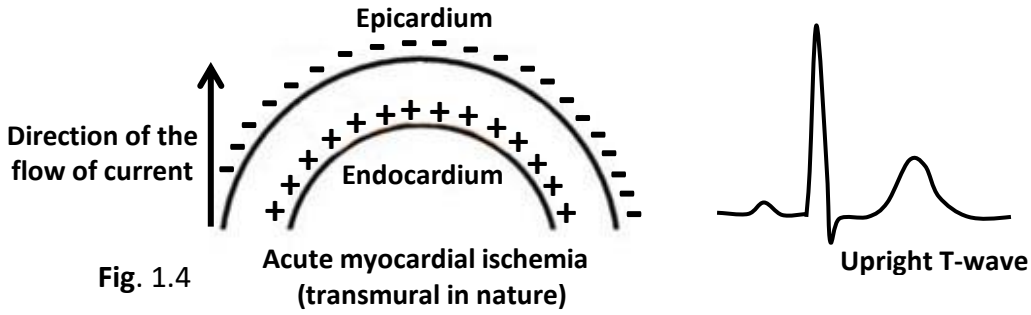

Fig. 1.4

- Acute ischemia may superimpose over chronically ischemic myocardial cells , which may convert the involved myocardium into transmural myocardial ischemic zone.
- The resultant transmural myocardial ischemia with hypoxia opens the KATP channels → earlier entry of K+ ions inside the myocardial cells →shortening the epicardial action potential duration.

Ref :

Pharmacology of cardiac potassium channels

Juan Tamargo, Ricardo Caballero, Ricardo Gómez, Carmen Valenzuela, Eva Delpón

Volume 62, Issue 1, April 2004, Pages 9–33,

https://academic.oup.com/cardiovascres/article/62/1/9/373105

✓(Read inside as per 4.3.2 Role of KATP channels)

- These changes result in upright T-wave. (The details are on the next page)

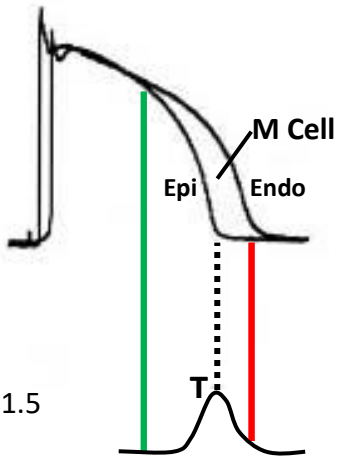

Fig. 1.5

The following points are to be considered here :

- ☐ Acute ischemia is superimposed over chronically ischemic myocardial cells , which may convert the involved myocardium into transmural myocardial ischemic zone.
- ☐ Due to the opening of KATP channels (hypoxia induced) causes the earlier entry of K+ ions inside the cardiomyocytes resulting in the shortening of epicardial action potenital (as explained on the preceding page)
- ☐ This causes upright T-wave on 12 lead ECG

Epicardium is the first site of repolarization

3. Discussion

Since this entity 'pseudonormalization of T-wave' is a rare entity , a paucity of data exist for its clinical characteristics and significance. But the recent data are available to support the evidence that this is due to the superimposition of acute myocardial ischemia over chronic ischemic injury to convert the entire zone as transmural myocardial ischemia.
This spontaneous pseudonormalization is found to be associated with severe narrowed or totally occluded coronary arteries , which usually result in transmural myocardial ischemia. This has been elucidated by the work of Cem Ulucan et.al in their article "Pseudonormalization: clinical, electrocardiographic, echocardiographic, and angiographic characteristics". This has been further strengthened by Carl J. Lavie et.al in their article "Significance of T-Wave Pseudonormalization during Exercise: A Radionuclide Angiographic Study". Most of the researchers are in favour that pseudonormalization of the T-waves occurs in patient with clearcut history of anginal background and is a sign of ischemia. Even immediately after angioplasty the so observed phenomenon of pseudonormalization indicates its occurence due to acute vascular occlusion after the procedure. Its occurrence after angioplasty indicates acute transmural ischemia with the urgent need of reconsideration of coronary intervention.

4. Concluding remark

- 'Pseudonormalization of T-wave' is a rare entity which causes pre-existing abnormal negative T-waves to its complete reversal to positive T-waves during acute anginal episode.
- Acute anginal episode may superimpose over chronic ischemic myocardial cells , which may convert the involved myocardium into transmural myocardial ischemia.
- Spontaneous pseudonormalization (PN) is a unique 12-lead electrocardiography (ECG) finding which has been reported to be associated with severe transmural myocardial ischemia.

- There is a need to pay a close consideration to the history and the symptoms of the patients while analyzing the phenomenon of pseudonormalization of T-wave.

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T-WAVE ALTERNANS : ITS ELECTROPHYSIOLOGICAL CONSIDERATION

ECG

T-WAVE ALTERNANS : ITS ELECTROPHYSIOLOGICAL CONSIDERATION

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OUTLINE

T-wave alternans : its electrophysiological consideration

A Narrative Review

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Everything is having a disciplined designing in nature. A few are not designed well – they may breakdown into disharmony. This disharmony might result in chaotic seedlings , bringing some dreadful disaster onto the surface. The ultimate result is nothing but a ruined battleground. Harmony kisses the life while disharmony misses the life. At times disharmony is bound to happen.

T-wave alternans is one of the examples of such disharmony on ECG. It is a very sensitive index of its susceptibility to sudden cardiac arrest (SCA).

- ☐ **T-wave alternans is a periodic rate dependent beat-to-beat alteration of the amplitude and /or shape of the T-wave on ECG.**
- ☐ **This reflects heterogeneity in ventricular myocardium during repolarization , which may break beat-to-beat harmony , appearing as T-wave alternans – it may clasp the life and life may be missed through its susceptibility to ventricular tachycardia (VT) or ventricular fibrillation (VF).**

Clinicians peep through the ECG tracings to diagnose such electrophysiological entity which helps in accessing the risk of life threatening ventricular arrhythmias associated with this.

1. Introduction : Keypoints

The T-wave corresponds to the repolarization phase of ventricular myocardium , the analysis of its morphology is commonly used to diagnose some cardiac pathology and accordingly to access the risk of life threatening situation like ventricular arrhythmias.
T-wave alternans is one of such T-wave electophysiological lacunae , rarely encountered in clinical practice.

- **T-wave alternans (TWA)** is a periodic beat-to-beat alterations of the amplitude and / or shape of the T-wave on 12 lead ECG.
- This repolarizing T-wave fluctuation is a **primary phenomenon** , not associated with other phenomenon like QRS alternans simultaneously. This was first reported in the early 1900s during the phase of tachycardia and ischemia in an observation made by Hering H and Thomas Lewis.
- **TWA is a rate dependent phenomenon** induced with higher heart rate threshold and once perceived it remains remarkably stable and persistent provided the heart rate remains the same.
  And its analysis can be done as a part of an exercise (graded elevation of heart rate) stress test or during a long term (Holter ECG recording).

- **The endocardial myocytes are more prone to repolarization alternans** compared to epicardial myocytes. Therefore , discordant TWA is more observed compared to concordant one.

A = Normal Paced heart beat with upright T (arising from epicardium)

B = T-wave alternans beat with inverted discordant T (arising from endocardium)

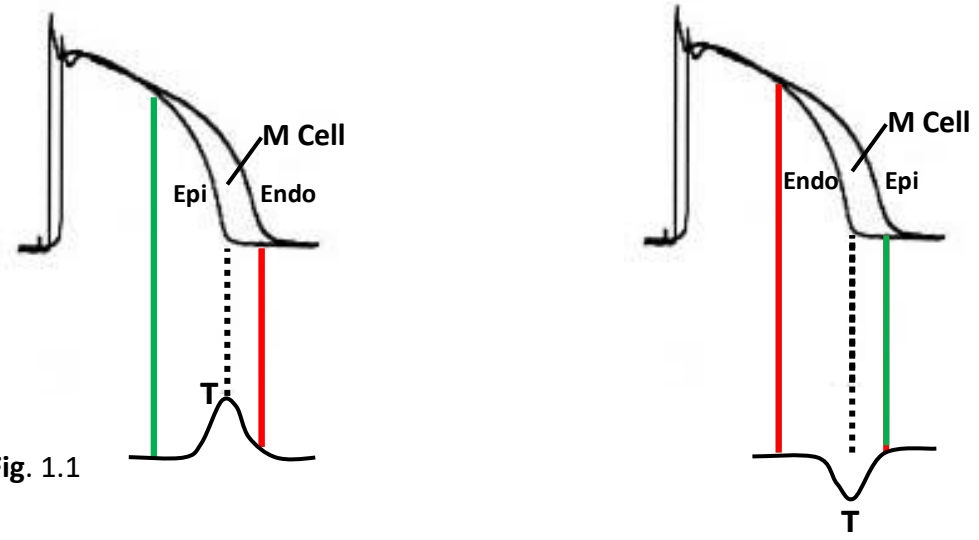


Fig. 1.1

Discordant T-wave alternans can be represented with a formula given below :

Normal paced beat with upright T-wave .....T-wave alternans beat with inverted T-wave (each on alternate basis)

Fig. 1.2

- Recent studies have clearly demonstrated that T discordant alternans creates a myocardial substrate that **is vulnerable to cause conduction block with the setting of reentrant ventricular arrhythmias.** This phenomenon occurs due to the associated dispersion of repolarization (heterogeneity) and can be used as a ECG marker to predict these malignant arrhythmias and sudden cardiac death (SCD).

2. Classification of TWA

T-wave alternans can be divided into two subtypes :

- Visually apparent – macro-TWA
- Visually inapparent – micro-TWA

Electrophysiologists as per recent observation refer microvolt T-wave alternans as a more significant phenomenon in this context. This microvolt level fluctuation is relatively common as detected in approximately 50% of patients having LVEF <0.40 , and is a good predictor of risk for SCD.

This would be worthwhile to mention here that the absence of TWA with ischemic or non-ischemic cardiomyopathy with heart failure even with reduced ejection fraction is associated with low risk of sudden cardiac death (SCD).

Micro T-wave alternans is visually inapparent specially if it is associated with such phenomenon at the same level i.e. with concordant T-wave alternans , as illustrated below :

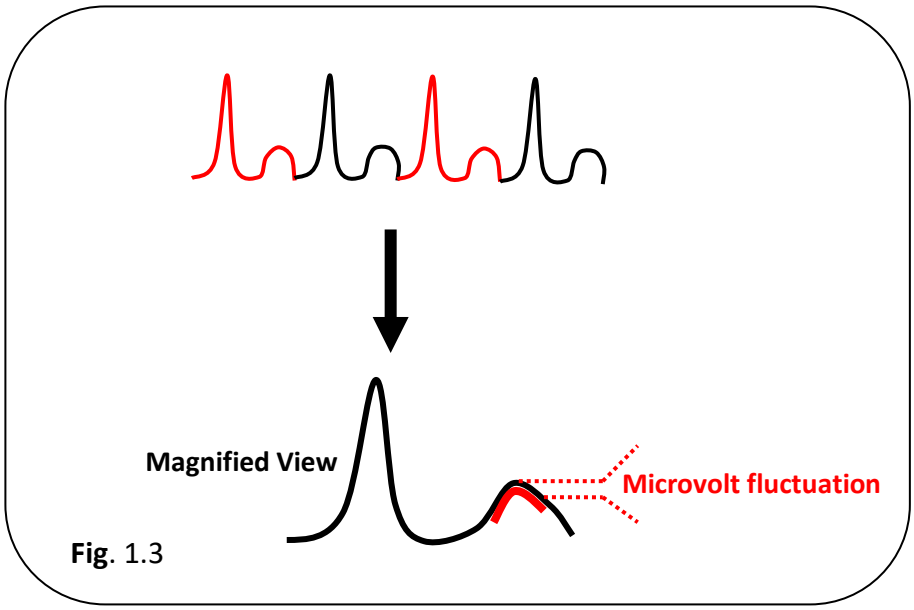


Fig. 1.3

This sketch explains how the visually inapparent micro-TWA becomes visible by placing the corresponding T-waves over each other. Microvolt TWA reflects spatiotemporal heterogeneity that is visioned in beat-to-beat alterations.

3. Concerned electrophysiology

The electrophysiology concerned with T-wave alternans is still not so clear – still an issue of debate. Broadly to say that such entity is said to involve two mechanisms , as elaborated below :

- ☐ Mechano-dispersion (spatiotemporal dispersion) causing beat-to-beat alterations in T-wave morphology by the alternative change in action potential duration of ventricular myocytes.
- ☐ Biochemical induced dispersion causing beat-to-beat alteration in T-wave morphology by alternative change in intracellular Ca^{2+} handling.

It is still currently unclear which one – mechano-dispersion or biochemical Ca^{2+} induced dispersion is the primary event in the causation of TWA.

Mechano-dispersion (spatiotemporal dispersion) mechanism involves two types of mechanism – one is concerned with spatial dispersion and the other one with temporal dispersion , discussed as below :

Spatial dispersion : There are certain subpopulation of myocytes at different ventricular locations. T-wave alternans comes in view when the heterogeneity of such

subpopulation of cardiac myocytes exhibits a longer duration action potenital compared to the preceding one.
In other words , such subpopulations of myocytes are depolarized on every other beat but with differing T-wave contour as noticed with the previous beat.

Temporal dispersion

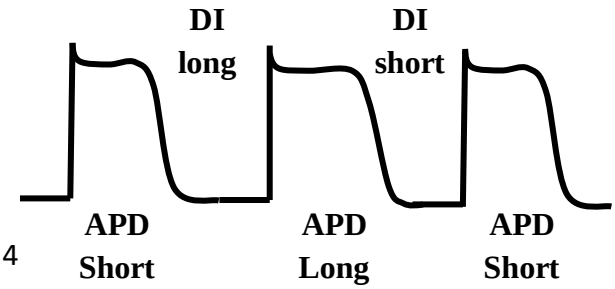


Fig. 1.4

DI = Diastolic interval APD = Action potential duration

Temporal dispersion is also known as **'Restitution phenomenon'**. This phenomenon is a sort of adaptive mechanism to preserve diastolic feeling at faster heart rate. In other words , it is the self-perpuating mechanism induced by changes in action potential duration and accordingly T-wave alternans . APD shortening with fast heart rate is followed by DI long (diastolic interval) , which in turn will increase action potential duration as a subsequent event. Each action potential is having a separate contour of T-wave resulting in T-wave alternans.
As per recent observation this temporal dispersion phenomenon is still treated as a hypothesis , there are certain clinical evidences not to support this concept.

Calcium handling mechanism

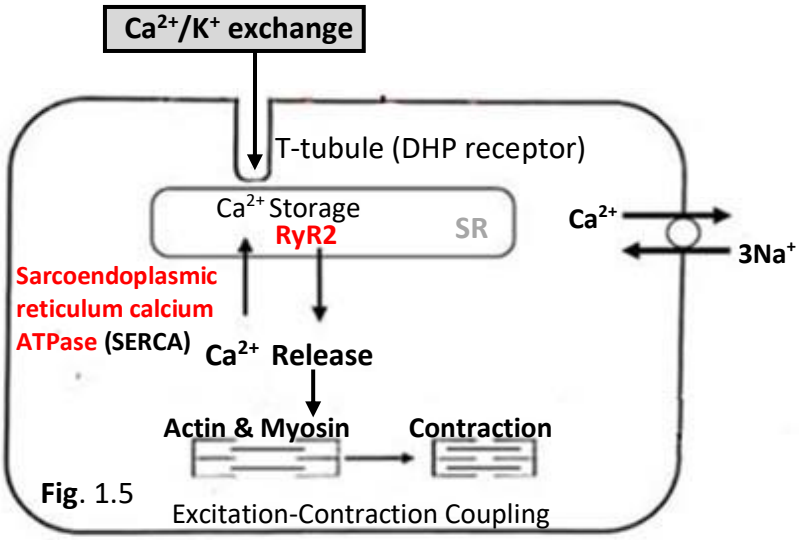


Fig. 1.5

Cycling of Ca2+ inside ventricular myocytes

Entrance of Ca2+ inside through T-tubule → triggering Ca2+ release from sarcoplasmic reticulum through special channel (RyR2) → Ca2+ release leading to excitation-contraction coupling → Ca2+ recycling back to the sarcoplasmic reticulum through SERCA.

- With a normal steady-state at resting heart rate – Ca2+ transient alternans will not develop because SR Ca2+ release equals SR Ca2+ reuptake.

- With increasing HR the capacity of SERCA to pump Ca2+ into the SR becomes overwhelmed, creating a state in which subpopulations of SERCA only respond on alternating beats for such reuptake leading to Ca2+ alternans and accordingly T-wave alternans.

4. A bird's eyeview over electrophysiological events (T-wave alternans)

The entire events of T-wave alternans are illustrated with the following sketch (unilateral conduction block with reentry is also included here) :

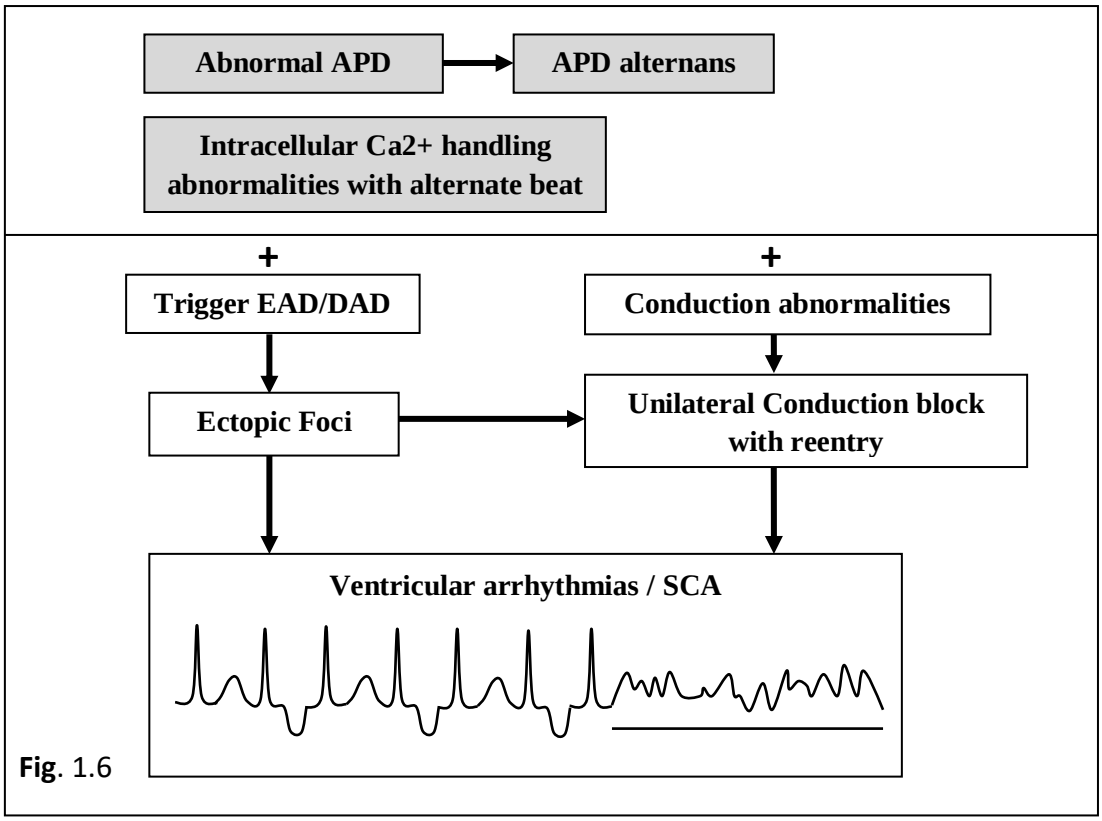


Fig. 1.6

EAD = Early afterdepolarization **DAD = Delayed afterdepolarization**

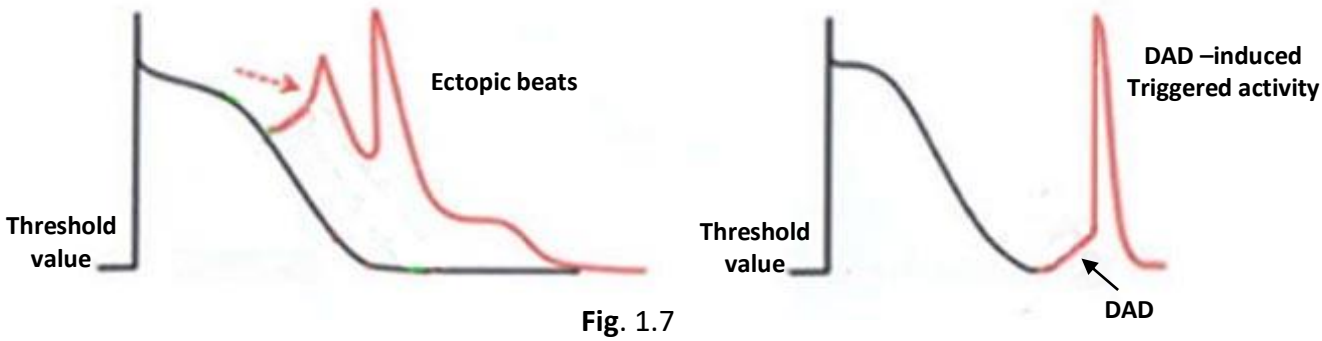
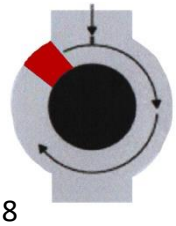


Fig. 1.7



Unilateral conduction block (as indicated by red zone) with reentry. (central black circle indicates ventricular heterogeneity).

Fig. 1.8

5. T-wave alternans analysis techniques

There are two methods :

- Spectral method
- Modified moving average (MMA) method

An exercise (graded elevation of heart rate) is the initial step.

Spectral method : It requires exercise stress test to achieve a target heart rate of 105-110 beats / min , to maintain an increased HR for a suitable period of time (usually 1-3 minutes) to make a justified measurement of TWA.

Modified moving average (MMA) method : The MMA method considers both beats of T-wave alternans by averaging – then finally the average of both the beats are superimposed upon each other to visualize TWA.

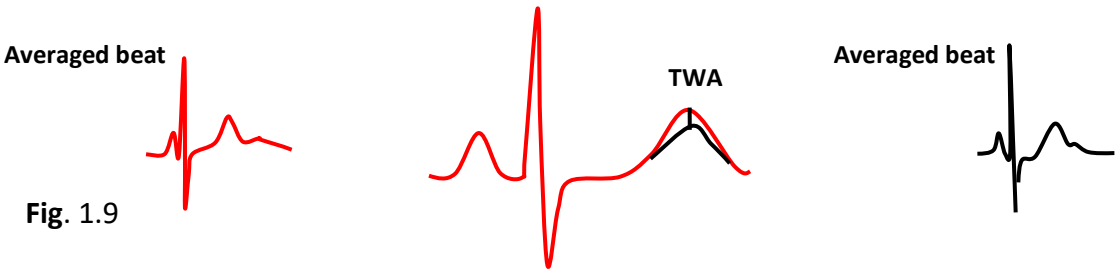


Fig. 1.9

6. Prognostic significance of T-wave alternans

T-wave alternans is observed in individuals with both ischemic and non-ischemic cardiac pathology – with reduced as well as preserved left ventricular function.
Recent studies have clearly demonstrated that T discordant alternans creates a myocardial substrate that **is vulnerable to cause conduction block with the setting of reentrant ventricular arrhythmias.** This phenomenon occurs due to the associated dispersion of repolarization (heterogeneity) and can be used as a ECG marker to predict these malignant arrhythmias and sudden cardiac death (SCD).
In most of the studies , the risk associated with TWA has been at least two-to-three fold , but much higher risk ratios have been also documented in some patients.
Such patients with TWA may need ICD support. However, at present there is no enough evidence to support ICD device in such situation.

7. Concluding remark

The recent observations support the view that ventricular heterogeneity with the mechanisms as discussed in preceding pages is reflected in TWA associated with an increment in the heart rate.

8. Acknowledgement

I express my sincere gratitude and regards to Dr. Satish Sir , a Senior Consultant in Cardiology , Wellmark hospital , Bokaro for his timely help for procuring some important references concerned with this article.

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CARDIAC MEMORY :
A RARE CAUSE OF T-WAVE INVERSION
(ELECTROCARDIOGRAPHIC DILEMMA)

ECG

CARDIAC MEMORY : A RARE CAUSE OF T WAVE INVERSION (ELECTROCARIDOGRAPHIC DILEMMA)

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OUTLINE

Introduction
Abnormal Ventricular activation (eg. episode of tachyarrhythmia), even occurring transiently , may cause electrical remodelling of the heart. This remodelling phenomenon is revealed on the next following ECG which has resumed sinus rhythm - as inverted T-wave , known as **'Cardiac memory'**.

What Cardiac memory denotes?
With resumed intrinsic sinus rhythm – T wave inversion tracks the QRS vector of the preceding abnormal activation.

Abnormal ventricular activation – background setting to initiate the phenomenon of Cardiac memory is a 'must'

Electrophysiological basis

- ☐ First to consider 'Homogeneous repolarization' with a normal person
- ☐ Then to consider , 'Heterogeneous repolarization' occurring during cardiac memory (on the ECG with resumed sinus rhythm)

Two types of cardiac memory exist :
short-term memory and long-term memory

Tips and tricks to recognize T-wave memory

Prognostic significance

Illustrating the phenomenon of cardiac memory by ECGs

Concluding remark

References

Cardiac memory : a rare cause of T-wave inversion (Electrocardiographic dilemma)

A Narrative Review

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O Heart , thou adopt a disciplined path of electrophysiology - dictated by 'P-QRS-T' on 12 surface ECG – a run of 'action with recovery' in sequence.

- ☐ **The internal intelligence running with thy steps at times may have the imprint of 'abnormal ventricular activation' in memory as a blackened trail episode.**
- ☐ **An altered pattern of ventricular activation , even occurring transiently , may cause electrical remodelling of the heart which is revealed on the subsequent surface ECG which has resumed sinus rhythm - as a significant change in the polarity of T-wave - Known as 'Cardiac memory'.**

1. Introduction

The cardiac activation starts from the SA Node and spreads through the conduction pathways to the ventricles to have its synchronised contraction in an orderly manner. At times , this pattern of activation might be altered even with the transient episode of tachyarrhythmia , right ventricular pacing , intermittent Left bundle branch block , intermittent WPW syndrome , etc. **Any altered pattern of ventricular activation, even occurring transiently , may cause electrical remodelling of the heart which is revealed on the subsequent surface ECG which has resumed its sinus rhythm - as a significant change in the polarity of T-wave – known as 'Cardiac memory'**. The entire concept is illustrated by the following ECG strips with inferior leads.

A. Abnormal activation (episode of tachyarrhythmia as shown with inferior leads)

✓B. The next ECG with resumed sinus rhythm but with inverted T-waves following the direction of QRS complexes of preceding episode of Tachyarrhythmia (**T-wave memory**)

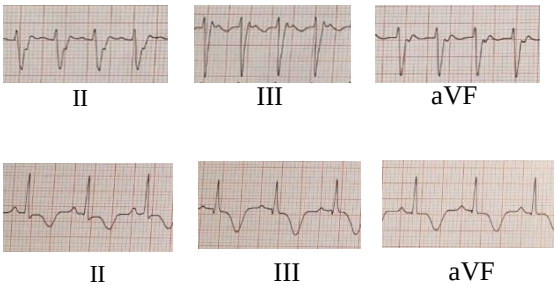


Fig 1.1

Rosenbaum et al , in 1982 were the first group of researchers to coin the term Cardiac memory for such changes. **First time they formulated the following basic principles revealing the secrecy of the phenomenon of cardiac memory**.

- The direction of T-wave with resumed sinus rhythm follows the direction of QRS complex of the past episodes of abnormal activation.
- The duration of T-wave memory induced as T inversion is directly proportional to the duration time of the preceding abnormal ventricular activation.
- The subsequent repeat episodes of abnormal activation after complete normalization of T-waves result in more rapid and prominent T wave changes.

The geometry of cardiac memory might open a new perspective concept to think over.

2. What Cardiac memory denotes ?

Cardiac memory is a specialised concept of electrophysiological remodelling , denoted by the alteration in T-wave polarity , manifested as T-wave inversion on the subsequent ECG with resumed sinus rhythm. Again , in due course the ECG gets recorded as a normal. This entire phenomenon can be summed up with the following ongoing tract- events :

Abnormal ventricular activation
↓
Altered T wave polarity (T-wave inversion) on subsequent ECG with resumed sinus rhythm
↓ Again , in due course the ECG gets recorded as a normal.

As per concept , it can be stated that cardiac memory is the consequence of adaptive property of the heart , being reflected by the adjustment of repolarization to a new activation sequence , as manifested by the change in T polarity. This would provide a new insight into the electrophysiological functioning of the heart in relation to cardiac memory.

Again to say , even after the resumption to normal ventricular conduction , T wave remembers as mirror-image the direction of the QRS complex of the previous abnormal ventricular activation i.e. even with resumed intrinsic sinus rhythm T-wave tracks the direction of QRS vector of the preceding abnormal activation – seems to maintain the pacing steps of each other.

3. Abnormal ventricular activation – background setting to initiate the phenomenon of Cardiac memory

(1) 'Post-tachycardia change in T-wave polarity' as imprint of cardiac memory.
e.g. following the transient episode of tachyarrhythmia

(2) Wide QRS complex pattern
e.g. Right ventricular pacing , intermittent left bundle branch block , intermittent WPW syndrome , sodium channel blocker toxicity , even after a single ventricular premature beat , etc.

Rosenbaum first noticed that activation pattern of the ventricles was a more concerned trigger factor than change in heart rate in inducing cardiac memory oriented T-wave changes.

4. Electrophysiological basis

- **First to consider 'Homogeneous repolarization' with a normal person:**
  The phenomenon of ventricular repolarization follows the path of homogeneity. The journey of ventricular repolarization starts from the region where the ventricular depolarization ends. This is to mention here that the last portion of ventricular depolarization is the posterobasal portion of the left ventricle , from where the wave of repolarization starts - epicardial cells having shorter duration potential cause the wave of repolarization to be spread longitudinally in the same direction as that of QRS complex. This causes upright T-wave.

This entire concept is illustrated with T-loop in relation with QRS loop vectocardiogram , as illustrated below :

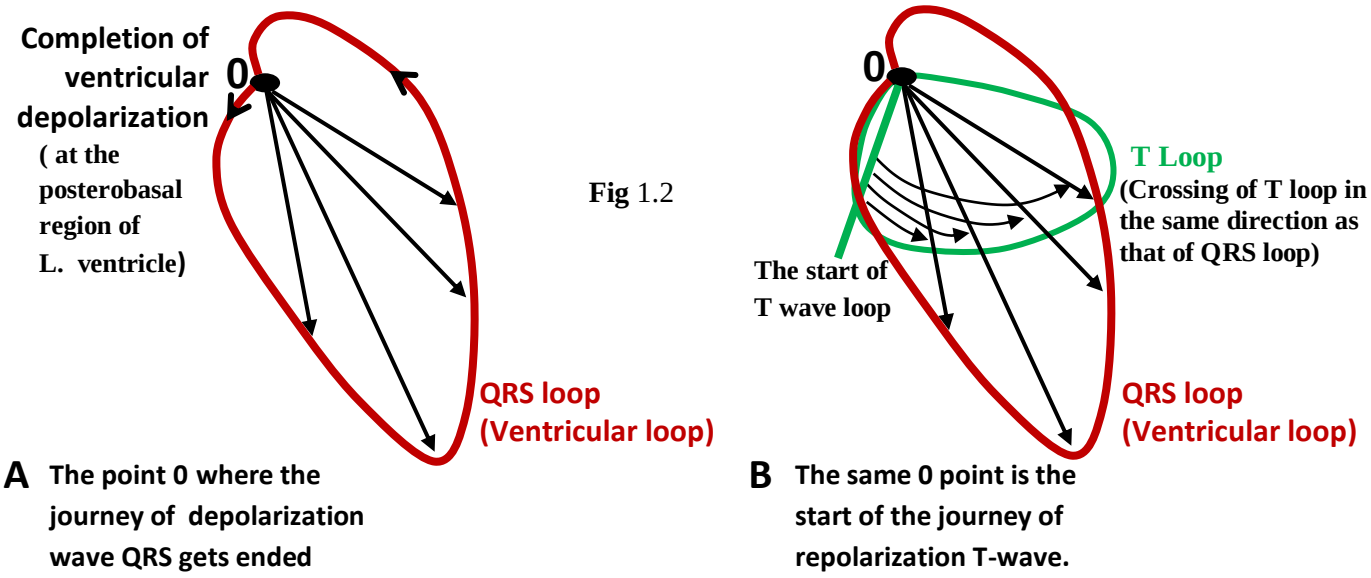


Fig 1.2

In nutshell , the concordance of QRS with T wave under normal condition of activation is explained by the presence of a homogeneous transmural repolarization across the ventricular wall with a shorter epicardial action potential – the epicardial cells are being repolarized earlier in this homogenous ventricular environment.

□ **Then to consider , 'Heterogeneous repolarization' occurring during cardiac memory (on the ECG with resumed sinus rhythm):**
With regional abnormal ventricular activation the epicardial cells in its neighbourhood have somewhat prolongation of epicardial potential duration (APD). Due to this epicardial APD prolongation there is a change in the direction of regional repolarization i.e. takingover of repolarization by the endocardial cell with reversal of the polarity of T wave , **manifested as T-wave inversion over the involved area**

Therefore , regional repolarization potential difference between different regions of LV as created by abnormal ventricular activation is said to be responsible for T-wave inversion accordingly. Here , the action potential duration is heterogeneous in nature across the involved region of the left ventricle – the causation for T-wave inversion associated with cardiac memory.

5. Two types of cardiac memory exist : Short-term memory and long-term memory

	Short-term memory	Long-term memory
Duration	Minutes to hours	Hours to days
Preceding abnormal ventricular activation	Brief episode of tachyarrhythmia	Wide QRS complex pattern
Region involved (on ECG with resumed sinus rhytnm)	The site of altered activation – the inferior septal wall	Lateral wall of the left ventricle
Mechanism (Ionic remodelling)	Concomitant ↑ angiotensin II leads to the internalization of a subunit Kv4 of transient outward potassium channel Ito ⟶ reduced Ica /Ikr channel with reduced Connexin 43 expression ⟶ Heterogeneous transmural myocardium	○ Altered gene expression with reduced protein synthesis of ion channels and connexion 43. ○ Heterogeneous transmural myocardium.

The mechanism , as explained under short-term and long-term memory is responsible for heterogeneous myocardial strain with slow cell-to-cell conduction. Further to say , the altered pattern of ion channels with reduced connexin is seen in the involved anatomical location over the myocardium , as mentioned.

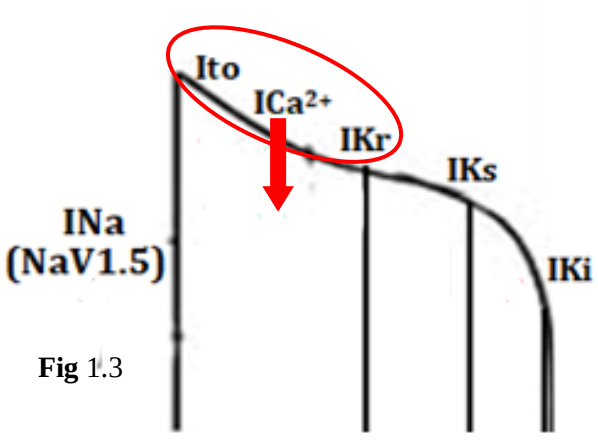


Fig 1.3

APD with reduced Ito and reduced Ica/Ikr channel expression
(As seen with 'Short-term memory')

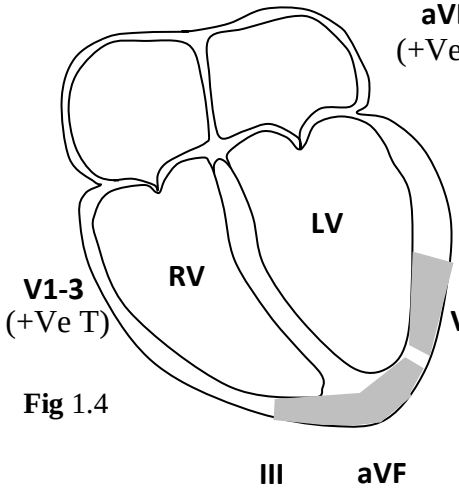


Fig 1.4

Heterogenous repolarization (reversal of T polarity)

(Heterogenous repolarization over inferior surface / lateral wall of the left ventricle : inverted T in inferior leads / V4-6 : with positive T in lead aVL and right sided precordial leads)

6. Tips and tricks to recognise T-wave memory

Cardiac memory as a diagnosis should not be accepted as the basic cause of TWI until other causes for such changes are ruled out. The recognition of this phenomenon helps a lot to minimize unnecessary hospital admission , cardiac testing and cardiac catheterisation.
It should be kept in mind that myocardial ischemia (coronary insufficiency) stands as a very important cause for TWI. Let us have a cursory glance at the important diagnostic pointers , as mentioned below :

- One should always try to correlate the electrocardiographic events with the clinical history of the case. Age-consideration imparts an important clue to the diagnosis.
- Shvilkin et al found 92% sensitivity and 100% specificity for diagnosing cardiac memory when the following criteria are met with :
    1) A Positive T wave in aVL combined with a positive or isoelectric T wave in lead I.
    2) Precordial TWI with larger magnitude than any TWI in lead III
- Both depolarization and repolarization events of the heart occur in a three dimensional space. Cardiac memory always produces a mixture of positive and negative T wave changes . Although positive T wave changes are often missed, they are as significant as TWIs as diagnostic tool. **Positive memory T waves over the right precordial leads might be missed.**
- Based on the presence of the inferior TWI and positive T waves in the right precordial leads in the absence of ischemia , one can suspect the presence of T-wave inversions originating in the inferior septal aspect of the left ventricle.

In nutshell , one should have analytic approach to the electrocardiographic events pointing towards the cardiac memory. In this context it is very important to keep the basic concept of Rosenbaum et al – the direction of T-wave in sinus rhythm follows the direction of QRS complex during the past episodes of abnormal activation.

7. Prognostic significance

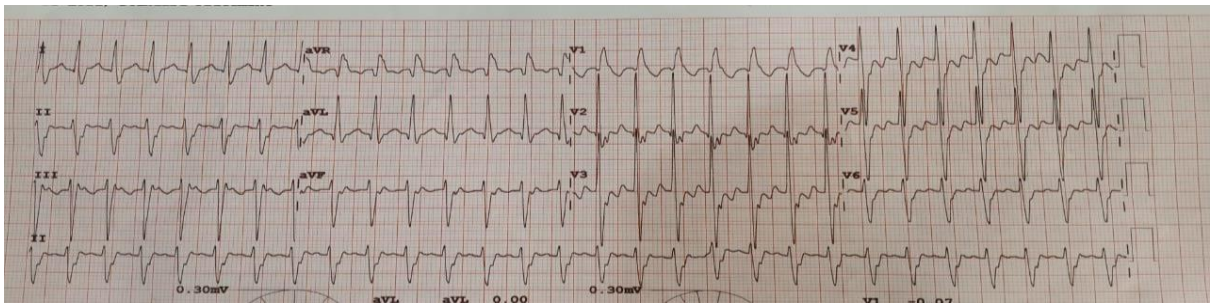
Nowadays , this condition is not thought to be benign in nature. The harmful consequences of altered activation are related to the adverse electro-mechanical remodelling of the ventricle. The electrical remodelling was first to be witnessed in patients who had increased susceptibility to ventricular arrhythmias following AV node ablation in a case of atrial fibrillation. The following considerations should be kept in mind for viewing the prognostic significance :

- ☐ Multiple case reports of TdP in patients who experienced cardiac memory are available to our study (changes from a pattern of abnormal ventricular activation to sinus rhythm can prolong the QT interval).
- ☐ A marked regional action potential gradient across the ventricular wall so created during this phenomenon of cardiac memory also acts as a nidus for ventricular arrhythmias.

8. Illustrating the phenomenon of cardiac memory by ECGs

ECGs A , B & C are kept here to illustrate the phenomenon of cardiac memory (see below)

A. ECG in a young boy with a history of Recurrent Palpitation



Source : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

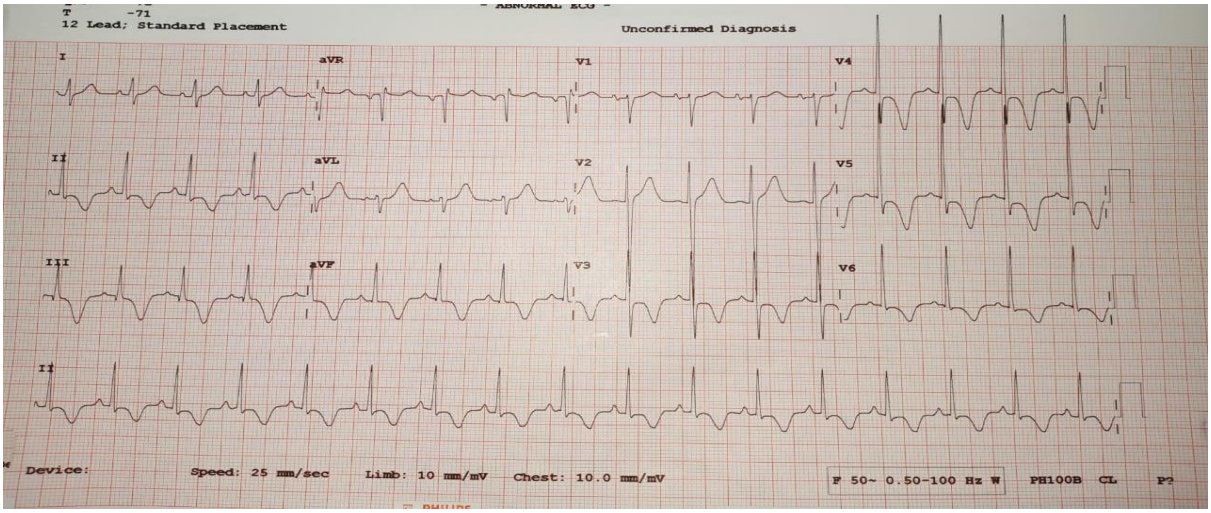
Findings on ECG

- Tachyarrhythmia with heart rate 200 bpm
- Careful inspection of lead II shows the presence of atrial flutter waves with 2:1 AV conduction.
- Associated RBBB and left anterior fascicular block

Discussion

Atrial flutter with 2:1 AV conduction associated with ventricular aberrancy.

B. Post DC Shock ECG



Source : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Prinicipal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

Findings on ECG

- Resumption of normal sinus rhythm
- T inversion in inferior leads II, III, aVF and precordial leads V3 through V6.
  T inversions more prominent over precordial leads compared to T inversion in lead III
- T is upright in I and aVL

  Discussion on the next page.

Discussion

The initial diagnosis was Apical HCM but Echo was normal. Keeping this in mind the final diagnosis of cardiac memory as a cause of T-wave inversion (TWI) was thought of. Post Tachycardia. T-wave inversion may occur in some patients with normal underlying heart , it is called cardiac memory. In support of this diagnosis the following points were kept into consideration.

- ☐ The direction of inverted T-waves as mentioned in this Post DC shock ECG with sinus rhythm follows the direction of QRS complex recorded during the preceding episodes of tachyarrhythmia. This favours cardiac memory as a cause of these T-wave inversion.

- ☐ Shvilkin et al criteria also supports the diagnosis of cardiac memory. They found 92% sensitivity and 100% specificity for diagnosing cardiac memory when the following criteria are met , as in this case.
  - A Positive T wave in aVL combined with a positive or isoelectric T wave in lead I.
  - Precordial TWI with larger magnitude than any TWI in lead III

'An electrocardiographic syndrome consisting of transient inversion of T-waves after paroxysms of tachycardia in normal hearts has been recognized for sometime. The resemblance of these changes to those of serious organic heart disease makes obvious the importance of their recognition and understanding.'

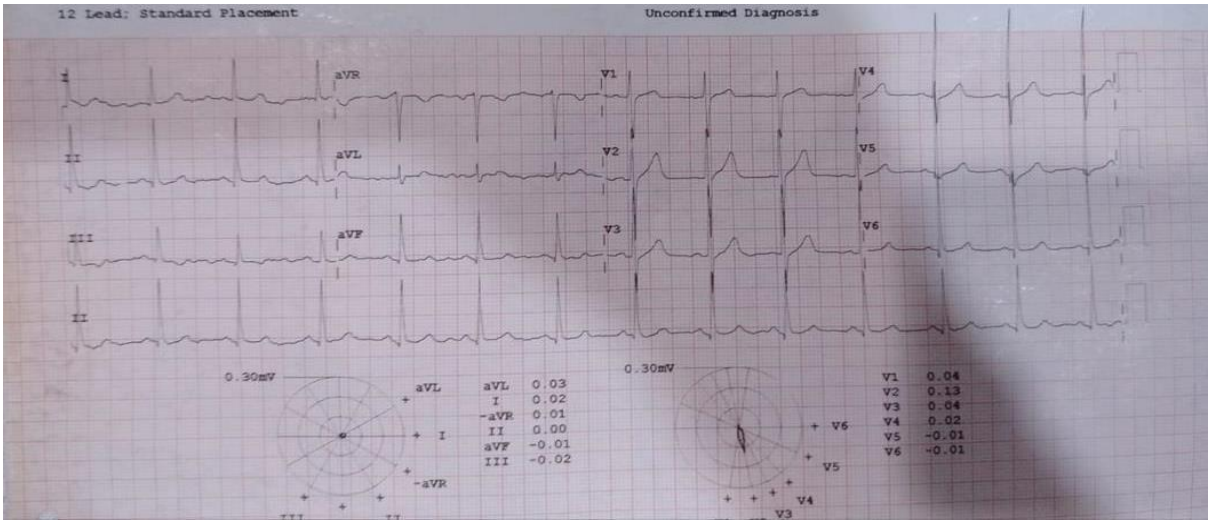
Ref : https://jamanetwork.com/journals/jama/article-abstract/313520

Darwin Jeyarai et al also narrated this phenomenon in their article entitled **'Pathophysiology and clinical implication of cardiac memory'.**

'There are several case reports since the 1940's of marked T-wave polarity changes in patients who present after transient episodes of tachyarrhytmias'. Since these changes commonly occurred following episodes of tachyarrhytmias they were termed **"Post-tachycardia T-wave changes"**

Ref : https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2865579/

C. ECG after one week



Source : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Principal ANMMCH , Gaya, Bihar ; Chairman AIMS, Gaya

The ECG became normal

9. Concluding remark

Cardiac memory (CM) is an unique adaptive property of the heart expressing the story of the adjustment of repolarization to a new activation sequence , which is manifested by T wave inversion. CM can be detected during both narrow and wide QRS rhythm and its knowledge improves ECG diagnosis. Further research in CM may provide more insight into the mechano-electrical functioning of the heart and potentially it may be used in risk assessment of the cases with cardiac memory.

Cardiac memory is still a diagnosis of exclusion and the electrocardiographic curiosity with such changes would further continue to bring more insight in understanding the mechanism regarding its pathophysiology.

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