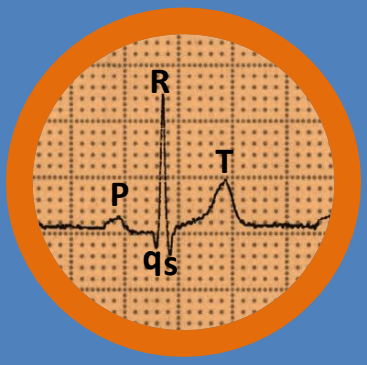

ECG Review : Some momentous articles

Series 8

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

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

On canvas of ECG
each wave whispers meaningful expression .....

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On canvas of ECG
each wave whispers meaningful expression ...

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 some momentous articles of my write-up are being covered within this book.. 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
FELLOW COLLEAGUES

Whose companionship gave knowledge a new direction
And whose inspiration turned every ECG into a story of life

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PEA (PULSELESS ELECTRICAL ACTIVITY) : THE HEART’S LAST CALL BEFORE SILENCE

PEA (PULSELESS ELECTRICAL ACTIVITY) : THE HEART'S LAST CALL BEFORE SILENCE

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OUTLINE

Introduction
In Pulseless Electrical Activity (PEA) , the concerned person is unconscious without a palpable pulse and blood pressure , despite the heart's electrical system being intact.

Electrophysiology in PEA
- ☐ The mechanism of normal myocardial contraction
- ☐ Some metabolic factors responsible for Myocardial Non-contractibility in PEA

Etiology
Assess PEA on the basis of mnemonic "5Hs and 5Ts"

Evaluation

Managing PEA through ACLS (Advanced cardiac life support) Protocol

Prognosis often poor , even with the best emergency resuscitation efforts.

Illustration by ECG

Take Home Message

Reference

PEA (Pulseless Electrical Activity) : The Heart's Last Call Before Silence

A Narrative Review

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I consider "Pulseless Electrical Activity" as the cry of a drowning man with his limbs paralysed, although he is acquainted with the art of swimming ..... His Last Call Before Silence .

PEA represents the paradox where electrical activity persists on ECG but without mechanical support to the pulse to serve.....electromechanical dissociation (EMD)

- ☐ **In PEA the ECG may show life , but the absence of pulse in an unconscious patient tells the story of death unless reversed quickly.**
- ☐ **This event reminds us that electricity alone does not define the life – mechanical and cellular integrity must unite to sustain it.**

A clinician must recognise this weeping cry of the dying heart by the presence of coordinated electrical activity on ECG , otherwise the heart is bound to die if the emergent action is not taken so quickly . It is just to remind that PEA is an unshockable rhythm where cardiac defibrillator does not work at all.

1. Introduction (Keypoints)

- **In Pulseless Electrical Activity (PEA) , the concerned person is unconscious without a palpable pulse and blood pressure , despite the heart's electrical system being intact.** In other words , the patient has cardiac electrical activity but without its mechanical translation to action , that's why this is also known as electromechanical dissociation (EMD). It is just to remind here that PEA is unshockable rhythm where cardiac defibrillator does not work at all.

- How to recognise PEA on the spot , the primary pointers include :
  - The presence of coordinated electrical activity on ECG
  - Assess responsiveness : Call the patient's name loudly or ask , 'Are you okay ?' and provide a painful stimulus if there is no response to verbal stimuli (e.g. , an sternal rub or pressing of the nail-bed) and / the responsiveness may also be judged by the clinician's acumen) → unconsciousness , suggestive of no effective perfusion to the brain.
  - Unrecordable pulse and blood pressure (feel the pulse at the carotid artery for a period of 10 seconds) – it confirms that there is no mechanical cardiac output despite electrical activity on ECG.
  - No heart beat is audible on the stethoscope. No respiratory movement is present.

All these steps must be completed very quickly. It is a red signal alarming situation.

- The concept of coordinated electrical activity on ECG .
    - PEA produces visible electrical activity on ECG but without mechanical support to cardiac contraction.
    - The cardiac electrical activity must have a logical pattern in an organized or semi-organized manner. This may include sinus rhythm , atrial fibrillation or even paced rhythms.
      The goal of identifying organized electrical activity in PEA is to determine whether the heart is generating electrical signals that would lead to coordinated contraction , the QRS complex whether normal or abnormal is the most reliable indicator for this. The P wave may precede QRS complex or may not be present there. T-waves might be abnormally widened in severe hyperkalemia.
    - Exclusion of Terminal Rhythms : PEA excludes conditions like ventricular fibrillation (VF) , Ventricular tachycardia (VT) or ventricular asystole. These are non-perfusing rhythms as de novo.
- Risk Factors and Frequency of Pulseless Electrical Activity
    - PEA is noticed in 30-38% of adults patients who suffer from cardiac arrest within their hospital-stay (such hospitalized patients are more at risk of experiencing the episodes of pulmonary embolism).
    - The administration of drugs like Beta-blockers and calcium channel blockers may make the concerned person more vulnerable to PEA.
    - Females are at higher risk of developing pulseless electrical activity compared to males.
    - The risk of having this condition increases more in persons over 70 years of age , particularly in females.

NB : **There exists a concept of pseudo-PEA , but a clinician should not struggle to establish this , otherwise the valuable time to save the life would be lost.**

"Pseudo-PEA describes electrical activity that can still cause the heart to contract slightly and pump some blood. However, heart function is still too weak to pump enough blood to meet the body's demands."

A 2020 study Trusted Source suggests that the incidence of pseudo-PEA is increasing and that it may be more common than previously estimated. This may be due, in part, to significantly less research on pseudo-PEA than true PEA.

Ref :

Pulseless Electrical Activity : Causes and treatment
https://www.healthline.com/health/heart-failure/pulseless-electrical-activity

---

2. Electrophysiology in PEA

Now it is obvious that in case of PEA the myocardium may fail to respond to mechanical contraction despite organized electrical activity on ECG. Therefore , it becomes essential to understand how systolic events on ECG are being inscribed :

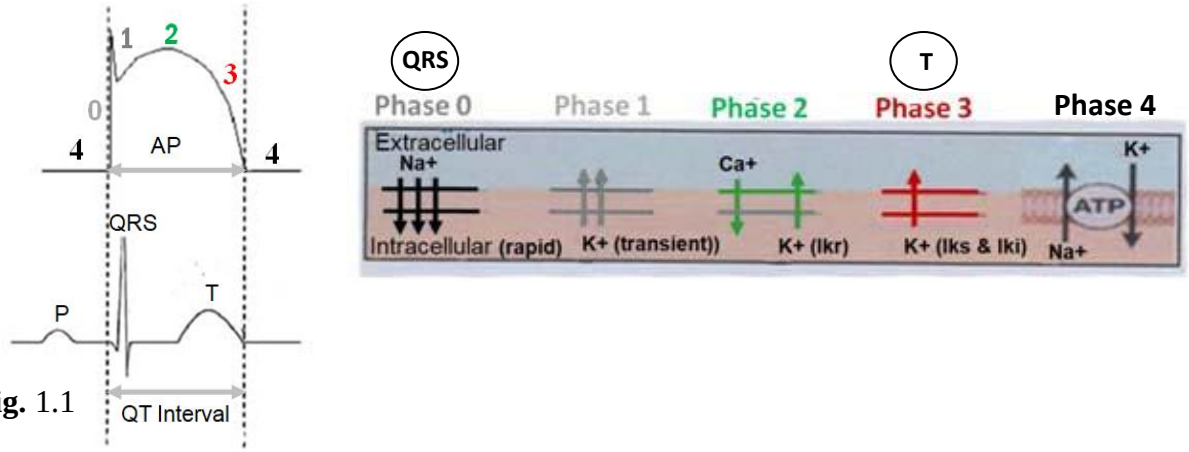


Fig. 1.1

The mechanism of normal myocardial contraction :

- QRS , the wave of depolarization precedes the onset of ventricular myocardium contraction , initiated through rapidly acting Na+ conductance channels (**Phase 0**).
- Opening of the L-type Ca2+ channels for the homogenous exchange in between Ca2+–K+ → the initiation of the excitation-contraction coupling leading to ventricular systole. This coupling occurs in cardiac myocytes lying underneath the isoelectric ST segment (**Phase 2**)
  (Phase 1 is not discussed here , which denotes a transient outward potassium current with a transient potassium efflux out of the cells giving rise to J-point on ECG)
- The closing of the L-type Ca2+ channels , followed by efflux of only potassium through K+ channels → T-wave (**Phase 3**)
- Restoration to the previous polarized state mainly by Na+ – K+ ATPase pump (**Phase 4**)

Some metabolic factors responsible for Myocardial Non-contractility in PEA

- Surplus potassium in extracellular compartment impairs rapidly acting sodium conductance channels and the process of depolarization thereby as well → this reduces myocardial excitability and contractibility both.
- Surplus calcium in extracellular compartment disrupts the exchange in between Ca2+ ions and K+ ions → impairing the excitation- contraction coupling, essential for ventricular contraction.
- Low potassium and calcium ions also reduce the cardiac contractibility by impairing the exchange in between Ca2+ and K+ ions

- Severe metabolic acidosis : High concentration of hydrogen ions reduce calcium sensitivity in cardiac myocytes impairing contractility.
- Hypoxia → less production of ATP molecules → impairment of Na+ - K+ ATPase pump → impaired regaining of polarized state. Cardiac myocytes cannot generate sufficient mechanical force for contraction
- Miscellaneous factors :
  - Mechanical constraints : Insufficient preload or excess pericardial fluid with precardial temponade by compressing the heart may prevent effective contraction despite the presence of electrical activity .
  - Tension pneumothorax , pulmonary embolism and even myocardial infarction may impair the cardiac contractility by inducing hypoxia and less volume return to the heart. MI may also cause the pump failure.

3. Etiology

The etiology of pulseless electrical activity is classified into primary i.e., cardiac, and secondary i.e., noncardiac causes.

Primary pulseless electrical activity is often caused by the depletion of myocardial energy reserves. It responds poorly to therapy

Its seems rather logical and essential to discuss the secondary causes of PEA first because the recognition of which is the main key to successful treatment.

Causes of secondary pulseless electrical activity are accessed on the basis of mneumonic "5 Hs and 5 Ts." These are as follows:

"Hs and Ts" of Pulseless Electrical Activity"

5 Hs	5 Ts
1. Hypovolemia 2. Hypoxia 3. Hydrogen ion (acidosis) 4. Hypo/hyperkalemia 5. Hypothermia	1. Thrombosis , coronary (myocardial infarction) 2. Thromboembolism (pulmonary embolism) 3. Temponade (pericardial) 4. Tension pneumothorax 5. Tablets (drugs : calcium channel blockers , beta-blockers, Tricyclic antidepressant , certain antipsychotic drugs , digoxin in high doses , cocaine , etc.) / Toxins

4. Evaluation

- Investigations should include an electrocardiogram (ECG), arterial blood gas analysis, serum electrolytes, chest radiology, and echocardiogram
  (Venous blood gas analysis provides earlier results of pH bicarbonate , CO2 and K)

- Core body temperature should also be assessed (Body temperature drops below 95^0 F/35^0C – hypothermia)

5. Managing PEA through ACLS (Advanced cardiac life support) protocol

- Start CPR : Perform high-quality chest compressions with an adequate rate and depth. Allow the chest to fully recoil between compressions.
  (100 Compressions/minute , 10 breathes/minute , 30 : 2 ratio if unintubated)
- Establish IV or IO access : gain vascular access as possible to administer medications.
- Administer epinephrine : Give 1 mg of epinephrine intravenously (IV) or intraosseously (IO) every 3–5 minutes.
- Looking for reversible causes and treat them accordingly
  (Essentially focusing on the "5Hs and 5Ts" mnemonic to address potential underlying causes)
- Advanced airway management to ensure adequate ventilation 100% oxygen
- Monitor rhythm and pulse :
  Regularly check for a pulse and rhythm to access the effectiveness of intervention

  ✓**NB** : **Intraosseous (IO) access through bone marrow to administer drugs , fluid and blood products when IV route is not available.**

6. Prognosis

- The prognosis for Pulseless Electrical Activity (PEA) is often poor, even with the best emergency resuscitation efforts.
- In one study, only 23% of patients with PEA were able to be revived and lived until they reached the hospital , and even fewer (11%) were still alive when discharged from the hospital. PEA is often a fatal condition.

Ref :
Pulseless Electrical Activity
Tony Oliver MD, FRCP (Glas, Edin), FACP, Shamai Grossman MD, MS, Usama Sadiq MD. Pulseless Electrical Activity StatPearls [April 7, 2023]
https://mdsearchlight.com/heart-health/pulseless-electrical-activity/?utm_source=pubmedlink&utm_campaign=MDS&utm_content=28073

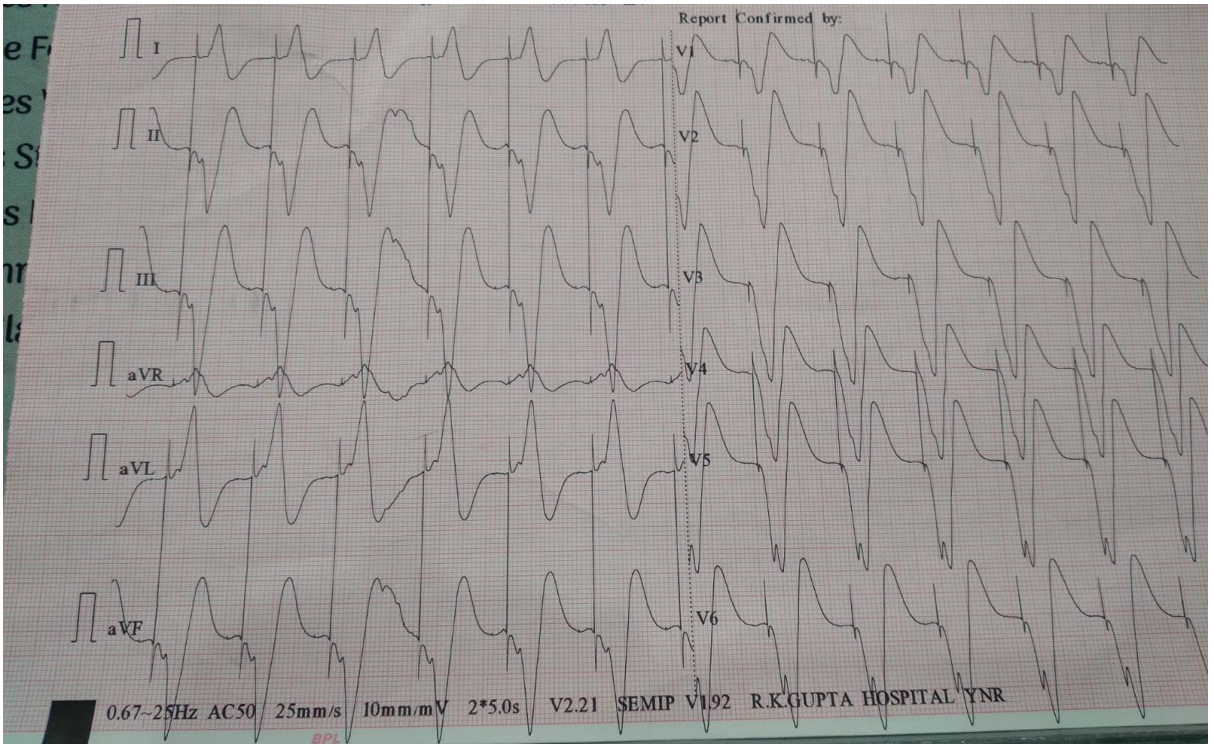
- Overall, prognosis of PEA is far less favourable than VF/VT, prognosis for out-of-hospital cardiac arrests (OOHCA) with initial asystole or pulseless electrical activity is <3% survival… though excpetions may include PEA with heart rate >60/min and 'pseudo-PEA''

In nutshell , PEA typically has a poor prognosis , with healthy survival is dependent on rapid intervention and the reversibility of the underlying cause , identifying the cause. Identifying the cause and providing effective CPR are critical for improving outcomes.

7. Illustration by ECG

History : 60 yrs male who had PPI implanted , was brought unconscious. On exam BP, PULSE unrecordable. No Respiration. No heart beats audible. Pupil B/L Dilated (ultimately the patient succumbed to death soon after the recording of ECG , he did not give time to act further). However no investigation other than ECG could be done.

ECG recorded at the presentation:



Source : Global Heart Rhythm Forum (14.01.2025) by Dr. R.K. Gupta ,
Senior consultant physician , Yamunanagar , Haryana

Findings on ECG

- PPI implanted (RV apical pacing , evident by unipolar spikes with LBBB morphology and negative complexes in inferior leads)
  Presence of pacemaker spikes indicate that the pacemaker is functioning (pacemaker spikes are appropriately followed by electrical activity)
- Electrical activity seen as the widened QRS complexes merging with T-waves. There is somewhat prolongation of second half of T (Peak to end T – proarrhythmic T).

Comments

- This is the case of pulseless electrical activity (PEA) , the concerned person came in in unconsious state without a palpiable pulse and BP but his ECG was showing the display of electrical activity , as evident by the presence of spikes induced wide QRS/T complexes.
- However , the non-contractile state of the myocardium as reflected by his unconscious state with the absence of pulse and blood pressure might be attibuted to hypoxia , metabolic dearrangements (acidosis , hyperkalemia $\pm$) , etc.

- The patient came very late with dilated pupils , suggestive of prolonged cerebral perfusion. There was no time to act further and the patient succumbed to death.

8. Take Home Message

- ☐ In "Pulseless Electrical Activity (PEA)" , the concerned person is unconscious without a palpable pulse and blood pressure , despite the heart's electrical system being intact.
- ☐ Have ECG to show the presence of coordinated electrical activity
  Assess his orientation to consciousness , see pulse and record blood pressure
  No heart beat is audible on the stethoscope. No respiratory movement is present
- ☐ Risk factors :
  - The administration of drugs like Beta-blockers and calcium channel blockers may make the concerned person more vulnerable to PEA.
  - Females are at higher risk of developing pulseless electrical activity compared to males.
  - The risk of having this condition increases more in persons over 70 years of age , particularly in females.
- ☐ Assess the etiology as per mneumonic – "Hs and Ts" of , as discussed on page 5
- ☐ It is a red-signal alarming situation- adopt the ACLS protocol to save the life.
- ☐ The prognosis for Pulseless Electrical Activity (PEA) is often poor, even with the best emergency resuscitation efforts.

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THE TRIO-LEAD SYSTEM IN STEMI : THE COMMON ASSOCIATION OF LEADS I , aVL, AND V2

THE TRIO-LEAD SYSTEM IN STEMI :
THE COMMON ASSOCIATION OF LEADS I , aVL , and V2

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OUTLINE

Introduction

Understanding the common association of trio of leads I , aVL , and V2 in STEMI may prove itself a mind boggling problem but a careful searchout through different leads makes the roadmap clear to reach to the site of coronary occlusion.

The concerned Electrophysiology

- ☐ ST vector is passing through lead I , aVL , lead ±V2 as a continuous alignment with coronary occlusion at D1 level.
- ☐ A reasoning to understand the ECG changes with occlusion of proximal LAD / proximal LCX : Here , ST elevation is due to the flow of current from endocardium towards the epicardium in the same direction as that of the placement of exploring leads.

Illustration by ECGs

- ☐ 1st Case : D1 occlusion with 'South African Flag sign'
- ☐ 2nd Case : Proximal LAD occlusion (acute anterior – lateral wall STEMI)
- ☐ 3rd Case : Left proximal LCX occlusion with h/o PTCA to LAD 12 yrs back (atypical case with multivascular involvement)

Take Home Message

References

The Trio-Lead System in STEMI : The Common Association of Leads I , aVL , and V2

A Narrative Review

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The ECG is a window into the city of the heart , through which the different patterns of ST elevation MI may peep through. Truly speaking , it is the ST elevation , mapping through a specific lead system , that reveals the story of which coronary artery is involved in the current scenario.

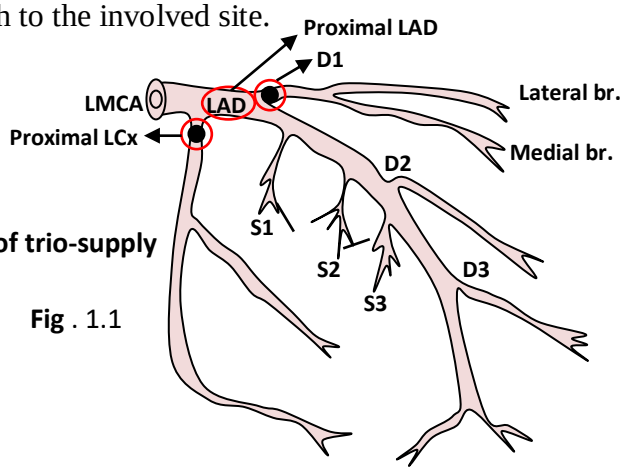
Just like the first stride of a storm on the horizon , ST elevation in I , aVL , and V2 signals an impending typhoon within the vista of the coronary arteries. It is an intriguing task to decipher these changes within the network of coronary supply , tracing how they sprout and unfold. .

- ☐ **The trio of leads I , aVL , and V2 primarily represents the high lateral and anteroseptal regions of the left ventricle.**
- ☐ **There exists a critical ischemic territory – confluence of trio vascular supplies : first diagonal branch (D1) of the LAD , proximal LAD and the left circumflex (LCX).**

The symphony of this trio-lead system marked by ST elevation whispers the secrets of an evolving infarct , unveiling the territory of a specific coronary supply. Clinicians trace this roadmap by carefully analysing different leads to pinpoint the site of the brewing acute infarct.

1. Introduction (Keypoints)

- Understanding the common association of trio of leads I , aVL , and V2 in STEMI may prove itself a mind boggling problem but a careful searchout through different leads makes the roadmap clear to reach to the site of coronary occlusion.
- This becomes essential to understand the anatomical supply of this critical ischemic territory to reach to the involved site.



Anatomy of trio-supply

Fig . 1.1

The details are discussed on the next page --

- Diagonal branches arising from proximal LAD are usually denoted by D1 , D2 and D3 , etc. and variable in number (often 2 to 9). **The first diagonal branch (D1)** tends to be more prominent and supplies the high lateral area of left ventricle , demarcated by leads I , aVL , and ±V2 . It is liable to be obstructed just close to the point of its origin from LAD.
- The occlusion of **proximal LAD prior** to D1 usually involves primarily the anterior-lateral , including apical region of the left ventricle. The whole spectrum is being reflected on trio-lead system I , aVL , and V2 , including the chest leads from V2 onwards to V6 in addition.
- **The proximal part of LCX** supplies the left ventricular area , being mapped out through leads I , aVL and V5-V6 in the place of V2

- The involvement of this trio-lead system may present on ECG as 'South African Flag Sign' , usually with D1 occlusion , but at times V2 may be replaced by other chest leads , as per occlusive lesion of the concerned vascular territory – the pertinent facts are well illustrated by the following sketch :

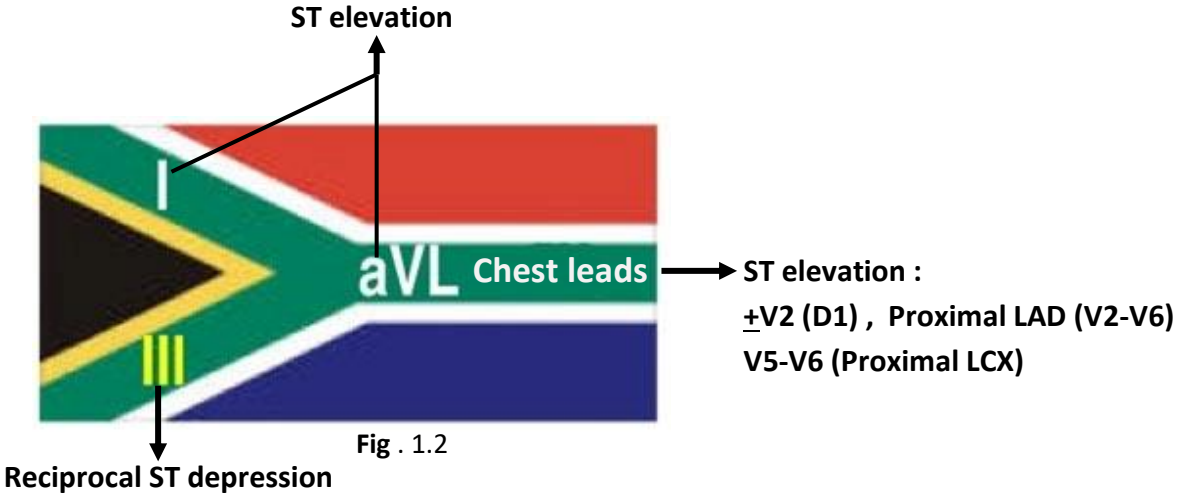


Fig . 1.2

- The ECG is a decisive tool in the early identification of the culprit vessel in STEMI and it can guide key management strategies by revascularisation. The clinician should be aware about the fact that the 'South African flag sign' (SAFS) is not only reflective of 'high lateral MI' (D1 occlusion) strictly but may also denote other coronary territory involvement as per se the association with other chest leads (see figure 1.2).
- In 2015 , Durant et.al. reported an ECG pattern of STEMI in leads I , aVL and V2 with ST depression in the inferior leads (maximally over lead III) , being associated with occlusion of the first diagonal branch (D1) of LAD. Subsequently , Littman coined the term 'South African flag sign' (SAFS) of such ECG scenario – the purpose behind this nomenclature was to facilitate earlier detection of 'high lateral MI' due to D1 occlusion. **This term was proposed as the South African flag sign due to placing of ST segment changes over its horizontal "Y limb" , as illustrated in the above sketch.**

2. The concerned Electrophysiology

☐ A model to understand ST vector in high lateral MI due to occlusion in the first diagonal branch (D1)

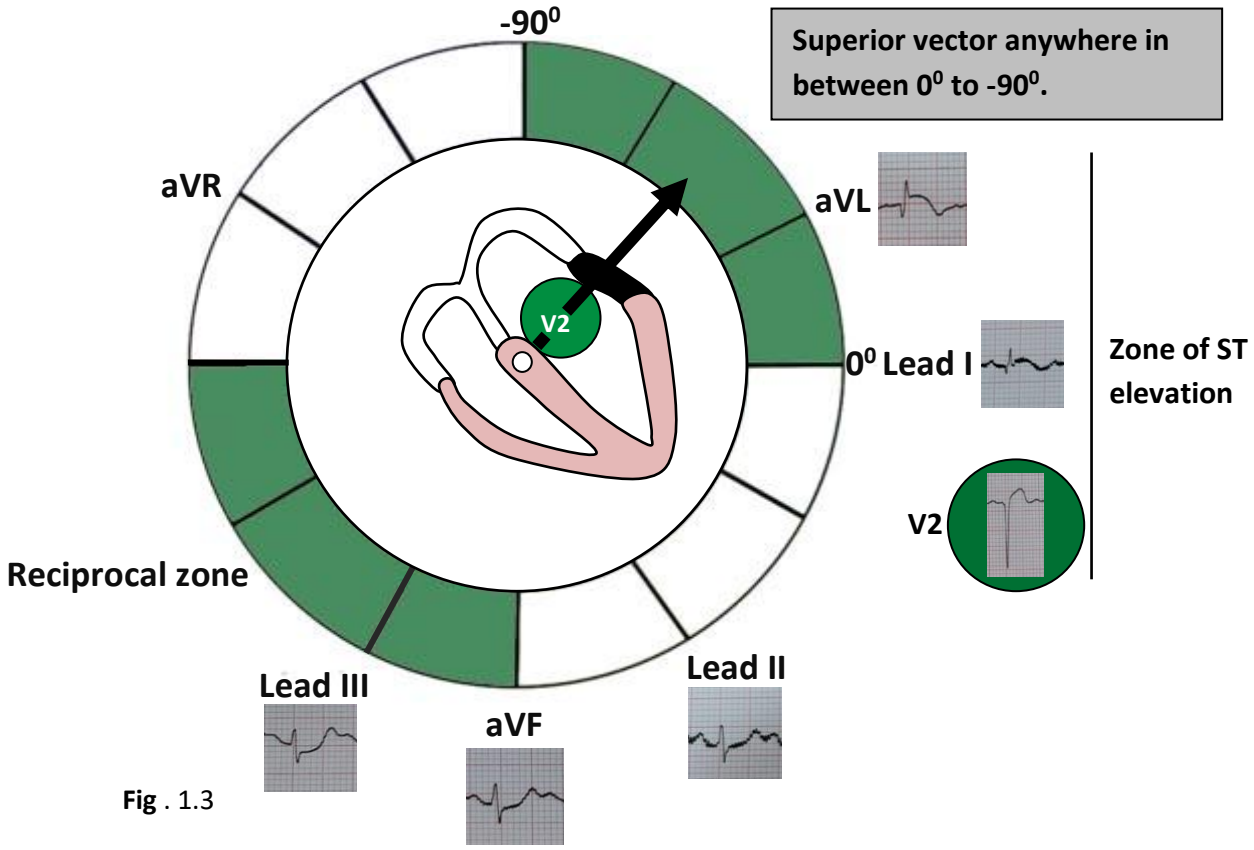


Fig . 1.3

A model to understand ST vector in high lateral MI with its changes on ECG

In high lateral MI , the ST vector points towards anywhere in between 0^0 to -90^0 on the frontal plane. Running through the left and higher than the centre of the heart , the frontal plane projection of lead V2 is also pointing towards the same vector direction as that of leads I and aVL – so having the ST elevation in this V2 lead also.

✓ Now it becomes easier to understand the concept of high lateral MI on ECG – characterized by ST elevation in leads I , aVL and V2, accompanied by reciprocal ST depression in inferior leads , most prominent in lead III.

With this common upward direction of ST vector – the leads I , aVL , and V2 are considered as lying in a contiguous alignment in respect to the vector. Therefore , these leads in reality can be addressed as the 'contiguous leads' in respect to high lateral STEMI – a very specific pattern on ECG. **Sometimes ST elevation in lead V2 might be absent due to its non-inclusion in the genesis of superior vector in continuity.**

☐ A reasoning to understand the ECG changes with occlusion of proximal LAD / proximal LCX.

The ST elevation in acute MI is attributed to the current of injury present over the subepicardial region , the reasoning for such ST elevation is due to the flow of current from endocardium towards the epicardium in the same direction as that of the placement of exploring leads.

- With occlusion of proximal LAD : the proximal LAD supplies the anterior-lateral , including apical region of the left ventricle. The whole scenario is being reflected as ST elevation in leads I , aVL , and V2 including the chest leads from V2 onwards to V6 in addition as well.
- The proximal part of LCX supplies the left ventricular area , being mapped out as ST elevation in leads I , aVL and V5-V6.

3. Illustration by ECGs

A basic concept how to localize the culprit vessel in STEMI in the presence of ST↑ with trio-lead system : I , aVL , and V2 , where V2 may be replaced by other chest lead denominators as per the site of occlusive coronary artery –

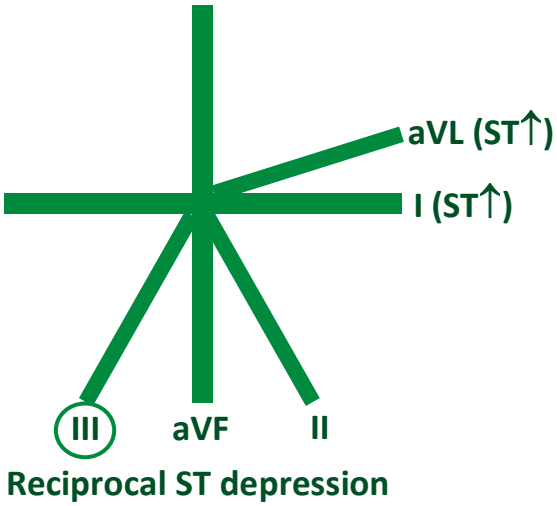


Fig . 1.4

To search out chest leads with ST elevation

ST↑ in chest leads as per site of occlusive coronary artery

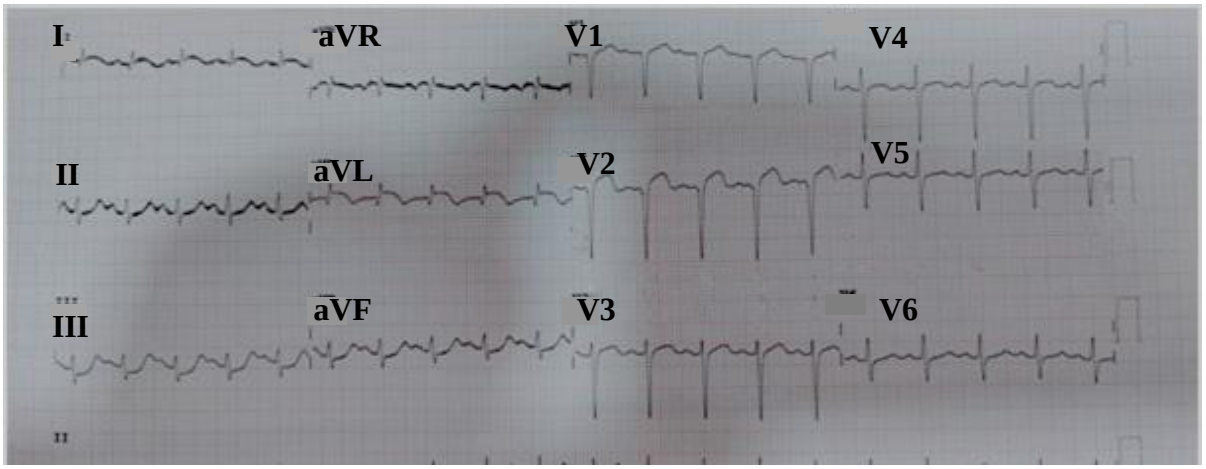
Common denominator on the frontal plane : ST↑ in aVL and I with reciprocal ST changes in inferior leads

☐ D1 occlusion evident by ST elevation in leads I , aVL and ± V2 , associated with reciprocal depression in inferior leads , most marked in lead III.

☐ Proximal LAD occlusion is reflected on trio-lead system as ST elevation in lead I , aVL with reciprocal depression in inferior leads , and associated ST elevation from V2 onwards to V6 in addition .

☐ The proximal occlusion of LCX is mapped out through ST↑ in leads I , aVL and V5-V6 , associated with reciprocal ST depression in inferior leads.

☐ 1st CASE : D1 OCCLUSION WITH 'SOUTH AFRICAL FLAG SIGN'



Source : **CME INDIA** , Feb 13 2023 by **Dr. Satish kumar** , Senior Consultant Cardiologist , Bokaro

History : 56 years male admitted with acute chest pain and sweating.
Mild lateral hypokinesia with LVEF 50% on cardiac echo

Interpretation : The following ECG changes are suggestive of high lateral myocardial infarction

- ST elevation primarily localised to leads I, aVL and V2
- Reciprocal ST depression in inferior leads , most prominent in lead III

This should be easier to memorize the ECG changes of high lateral MI by putting all these over the South African flag. It also imparts a concept that ST elevation seen in V2 is running in the same direction as that of common ST vector (leads I and aVL).

The findings on this ECG are plotted over South African flag as a very specific sign.

SOUTH AFRICAN FLAG SIGN

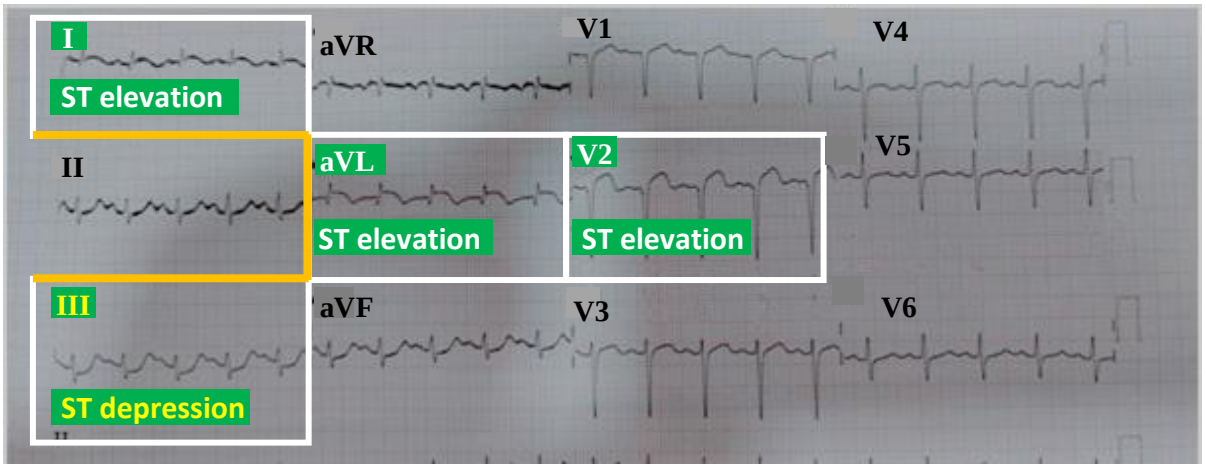
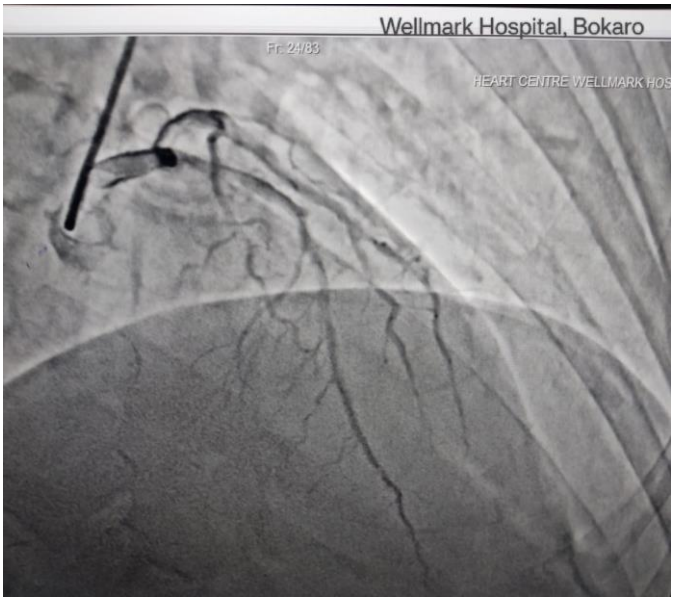


Fig . 1.5

Further to say

Coronary angiography (CAG) of the same patient shows the site of occlusion at D1 (thrombotic)



D1 occlusion is an obstruction distal to the point of its origin from LAD (Please see also figure 1.1 in this context)

Fig . 1.6

The same patient post PCI at D1

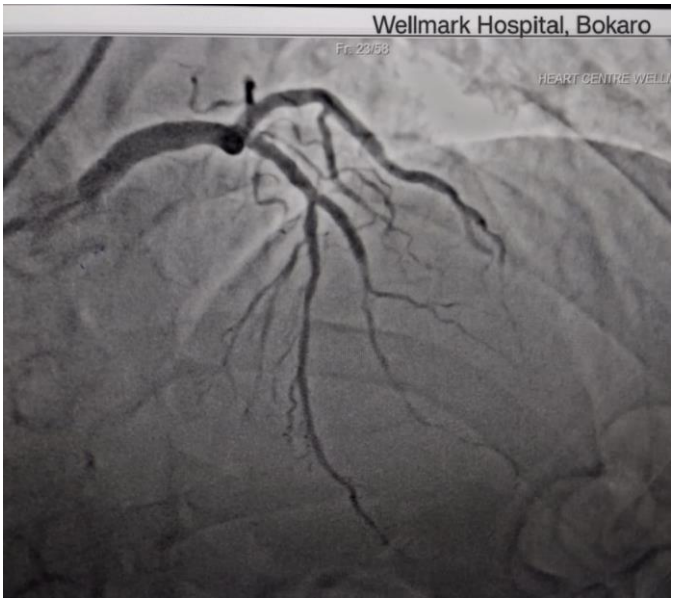
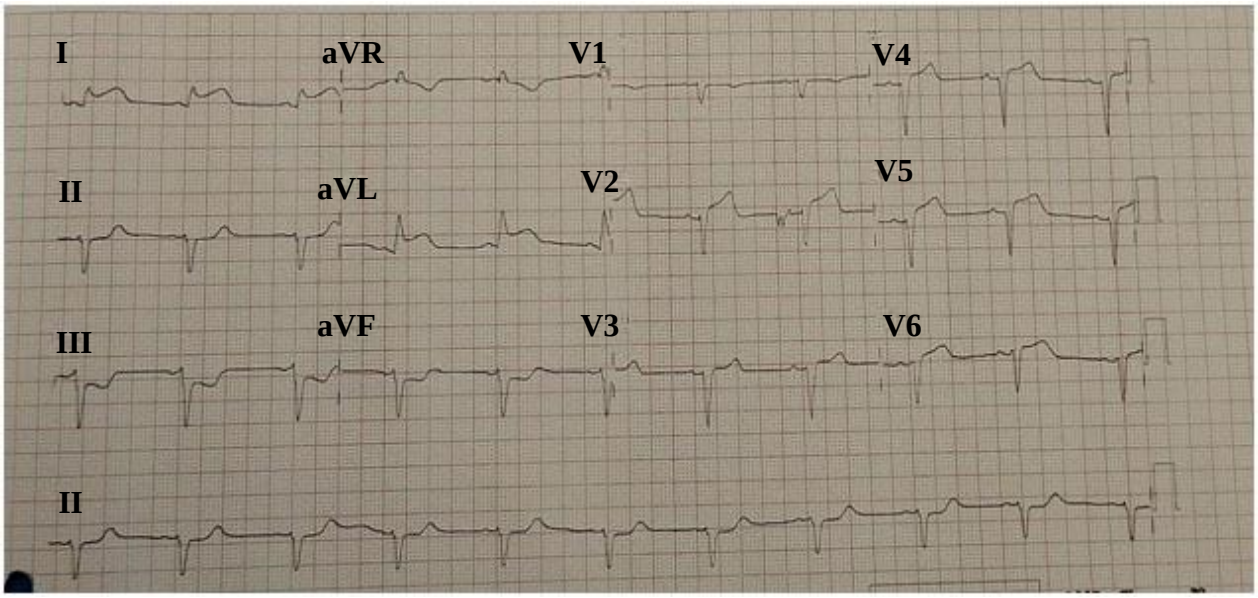


Fig . 1.7

Source : **CME INDIA** by **Dr. Satish kumar** , Senior Consultant Cardiologist , Bokaro

☐ 2nd CASE : PROXIMAL LAD OCCLUSION (acute anterior-lateral wall STEMI)

History : 60 years M, No past h/o DM, HTN or CAD presented with acute chest pain for 5 hrs. Troponin was High, Echo suggested Mid and Apical IVS, Anterior - Lateral Wall Akinesia, LVEF - 30-35%



Source : Dr. Satish kumar , Senior Consultant Cardiologist , Bokaro

Findings : The following ECG findings are suggestive of proximal LAD occlusion before D1

Over Frontal plane

- ST elevation in leads I and aVL with reciprocal ST depression in inferior leads with maximum over lead III.
- High left anterior fascicular block (frontal QRS axis -70^0)

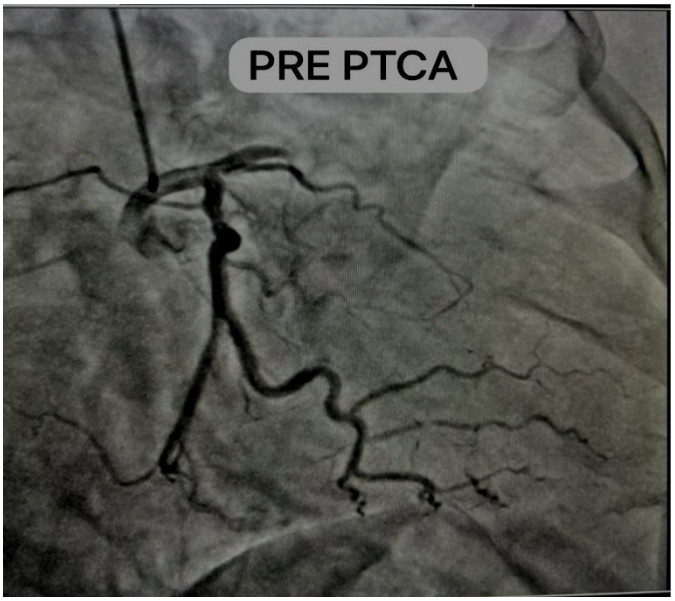
Over Horizontal plane

- ST elevation – from V2 to V6
- Poor R-wave progression (please see lead V3) , associated with very tiny r throughout precordial leads with extreme clockwise rotation

Discussion :

- ST elevation in leads I , aVL and over chest leads extending from V2 to V6.
- There is associated poor R-wave progression , associated with extreme clockwise rotation with very tiny r
- The presence of high left anterior fascicular block without accompanying RBBB (if the occlusion is proximal it may spares some twigs to septal perforators or the RBB may still receive blood from collaterals – via RCA or other branches) but the left anterior fascicle has fewer collateral pathways , making it more susceptible to ischemic insult and it is also having the single blood supply from LAD .

Coronary angiography (CAG) of the same patient shows the site of occlusion at proximal LAD , prior to D1



Proximal LAD occlusion prior to D1

Fig . 1.8

The same patient Post PTCA

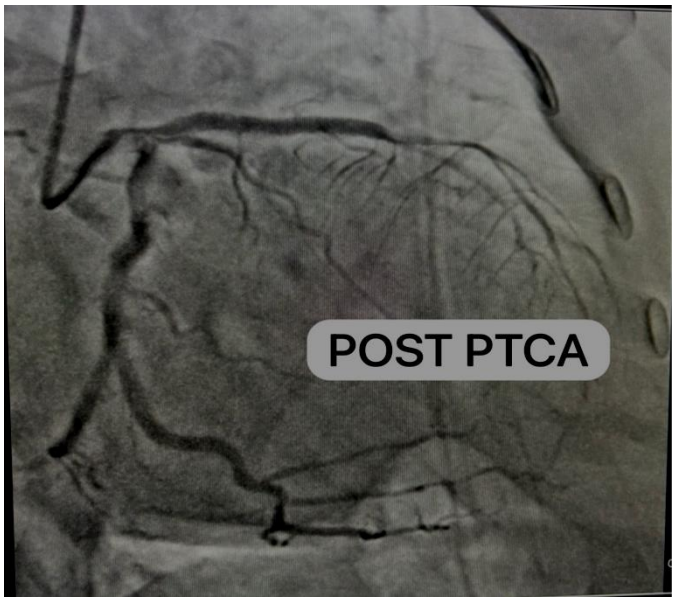


Fig . 1.9

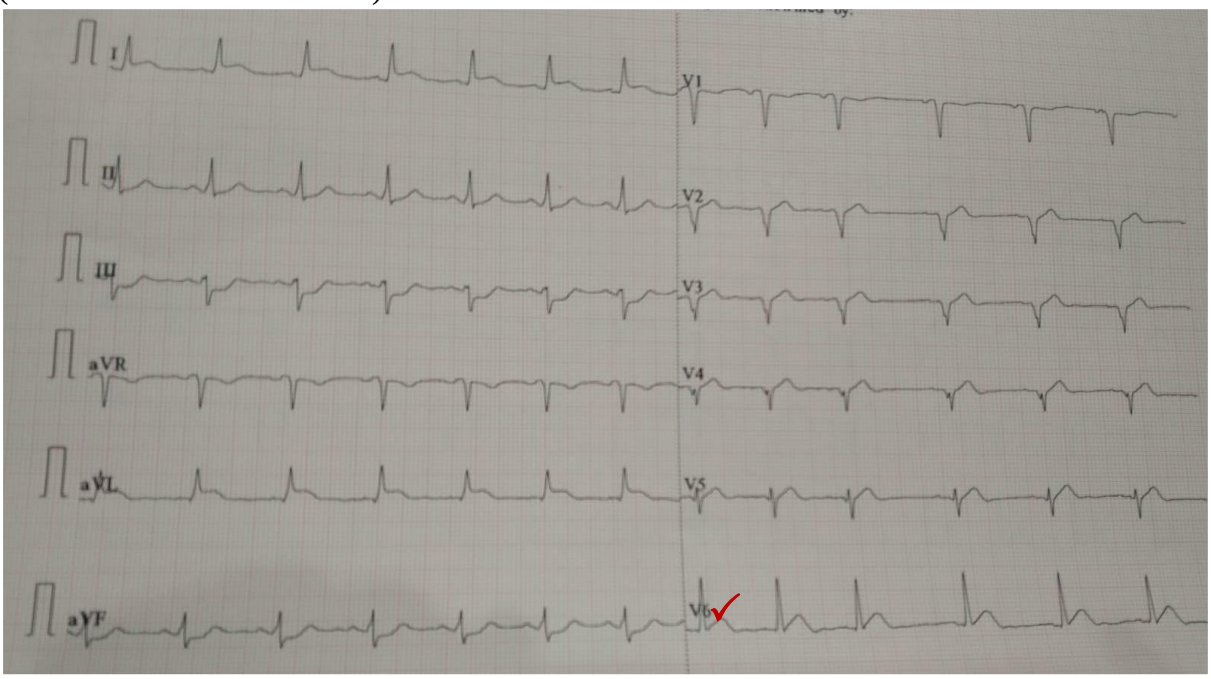
Source : **Dr. Satish kumar ,** Senior Consultant Cardiologist , Bokaro

☐ 3nd CASE : LEFT PROXIMAL LCX OCCLUSION WITH H/O PTCA TO LAD 12 YRS BACK (Atypical case with multivascular involvement)

History

This patient had h/o PTCA to LAD 12 years back. This ECG is with High chest leads in view of SAF sign. He was not taking any medication now. His Echo showed severe LV DYSFUNCTION 30 %. LV Dilated. Angiography showed 70 % LCX, partially patent LAD stent , Culprit PTCA to LCX done. RCA also has 80% Stage PTCA

(ECG Prior to PTCA to LCX)



Source : Global Heart Rhythm Forum , 14th February , 2025 by
Dr. R.K. Gupta , Senior Consultant Physician , Yamunanagar , Haryana

Findings :

- ST elevation in lead I , aVL and V6 with reciprocal changes in inferior leads , most prominent over lead III
- Old anterior transmural Q wave infarction (obvious over V1-V4)

Discussion :

- ST elevation in leads I , aVL along with lead V6 points towards occlusion of the left circumflex artery (plus old anterior transmural Q wave infraction in addition , due to the previous LAD occlusion - h/o PTCA to LAD 12 years back).
  - ∗ If ST elevation is missed to be observed in lead V6 , it would lead to the false impression of occlusion at first diagonal branch (D1) , wherein ST elevation in V2 might be absent.
- By the report of cardiac echo , it is multivascular involvement (70% occlusion in LCX , partially patient LAD stent , 80% occlusion with RCA)

Ref :

The South African Flag Sign: An Electrocardiographic Flag for All Coronary Territories?
Steven Swarath [1], Nicole Maharaj [1], Andrew Hall [2], Jean Marie Frederick [1], Rajeev Seecheran [3], Valmiki Seecheran [1], Naveen Anand Seecheran
https://pmc.ncbi.nlm.nih.gov/articles/PMC10413884/

4. Take Home Message

☐ The trio of leads I , aVL , and V2 primarily represents the high lateral and anteroseptal regions of the left ventricle.

☐ **There exists a critical ischemic territory :**

- **The first diagonal branch (D1)** tends to be more prominent and supplies the high lateral area of LV , demarcated by leads I , aVL , and ±V2 . It is liable to be obstructed just close to the point of its origin from LAD.
- The occlusion of **proximal LAD prior** to D1 usually involves primarily the anterior-lateral, including apical regions of the left ventricle. The whole spectrum is being reflected on trio-lead system by ST elevation in I , aVL , and V2 , including the chest leads from V2 onwards to V6 in addition.
- **The proximal part of LCX** supplies the left ventricular area , being mapped out through leads I , aVL and V5-V6.

One Should also look for reciprocal ST depression in inferior leads.

☐ The involvement of this trio-lead system may present on ECG as South African Flag Sign , usually seen with D1 occlusion , but at times V2 may be replaced by other chest leads , as per occlusive lesion of the concerned vascular territory. Therefore , the South African flag ECG sign (SAFS) is not only reflective of 'high lateral MI' (D1 occlusion) strictly but may also denote other coronary territory involvement as per se the association with other chest leads.

☐ In nutshell , a basic concern is how to localize the culprit vessel in STEMI in the presence of ST↑ with trio-lead system : I , aVL , and V2 (V2 may be replaced by other chest lead denominators as per the site of occlusive coronary artery)

NB : It would be worthwhile to mention here why the proximal sites of coronary arteries **(here in context with critical ischemic territory)** are more vulnerable to occlusion MI. There are several reasonings for the same :

- More shear stress and turbulent blood flow at the site of proximal coronary arteries , predisposing them to endothelial injury.
- Greater pressure fluctuations , causing more mechanical stress on the vessels wall at the proximal sites
- More atherosclerotic plaques in areas of more shear stress and turbulent flow.
- The proximal areas are more predominantly exposed to circulating lipids with subsequent inflammatory cells aggregation , leading to plaque formation.
- Proximal plaques often have a thin fibrous cap with a large lipid core , making them more susceptible to rupture.
- The proximal coronary arteries have a fewer collateral vessels , compared to distal branches.

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Steven Swarath [1], Nicole Maharaj [1], Andrew Hall [2], Jean Marie Frederick [1], Rajeev Seecheran [3], Valmiki Seecheran [1], Naveen Anand Seecheran
https://pmc.ncbi.nlm.nih.gov/articles/PMC10413884/

NORMAL T-WAVE :
A WAY TO ITS UNDERSTANDING

NORMAL T-WAVE – A WAY TO ITS UNDERSTANDING

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OUTLINE

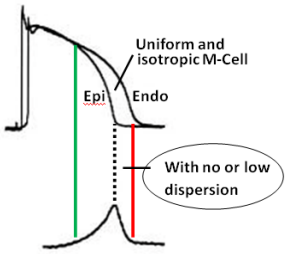
Introduction

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

Electrophysiology – Genesis of T-wave

A journey of repolarization as compared to that of the depolarization

Exploring the facts leading to T-wave morphogenesis



Epicardium with a shorter action potential than endocardium

- ☐ Normally the myocardial cells are having uniform and isotropic conductivity, practically with no or low dispersion , hence having no impact on the shape of the T-wave
- ☐ It is the rate of repolarizatiion over the epicardium and endocardium which determines the asymmetrical nature of the T-wave.
- ☐ The descending limb of the T-wave is steeper because the rate of repolarization accelerates as the endocardium completes it recovery.

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 Message

References

Normal T-wave – a way to its understanding

A Narrative Review

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

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 it 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 .**
- ☐ **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 again restart the journey (the polarized state is ultimately due to phase 4).

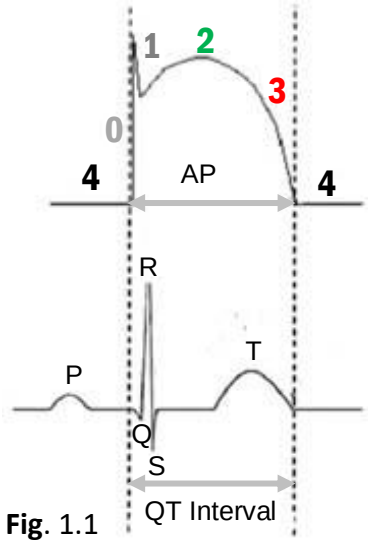
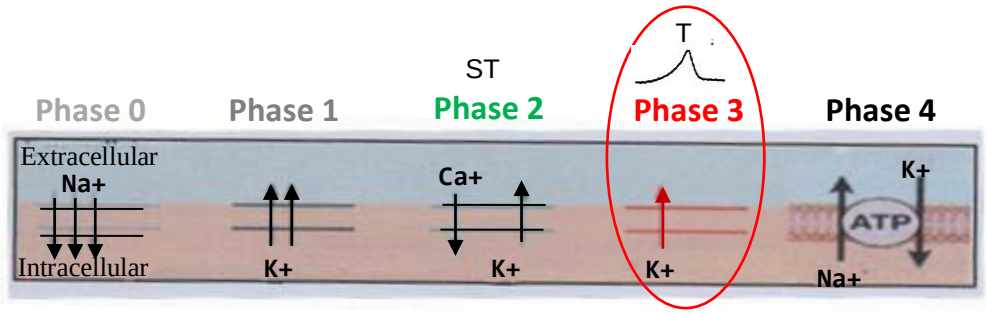


Fig. 1.1



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 : 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 and by propagating ahead leftward and downwards , it causes 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 :

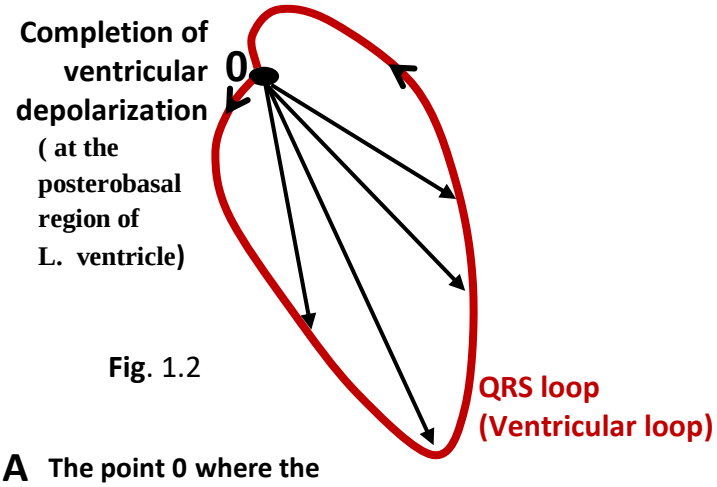


Fig. 1.2

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.

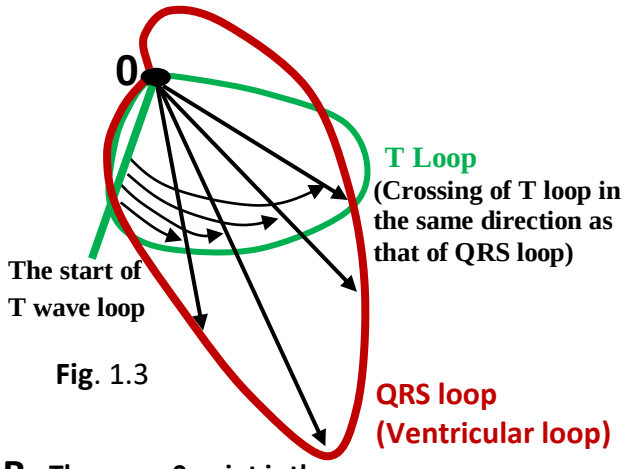


Fig. 1.3

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.

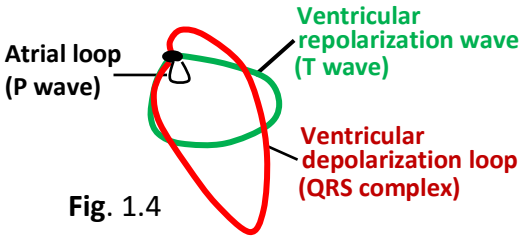


Fig. 1.4

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

Understanding : Step 2

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

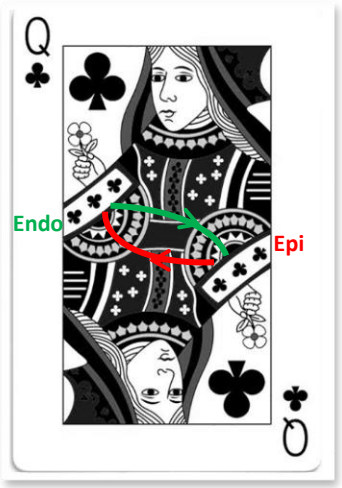
- ☐ 

Fig. 1.5

A journey of repolarization compared to that 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 , as illustrated in Fig. 1.5/1.6.

Genesis of T-wave

- The ascending limb of the T-wave reflects the gradual buildup of repolarization current along the epicardium resulting in a smooth , gradual rise on the ECG.
- The descending limb of the T-wave is steeper because the rate of repolarization accelerates as the endocardium completes its recovery.
- The T-wave is normally asymmetrical, with a gradual ascending limb and a steeper descending limb.

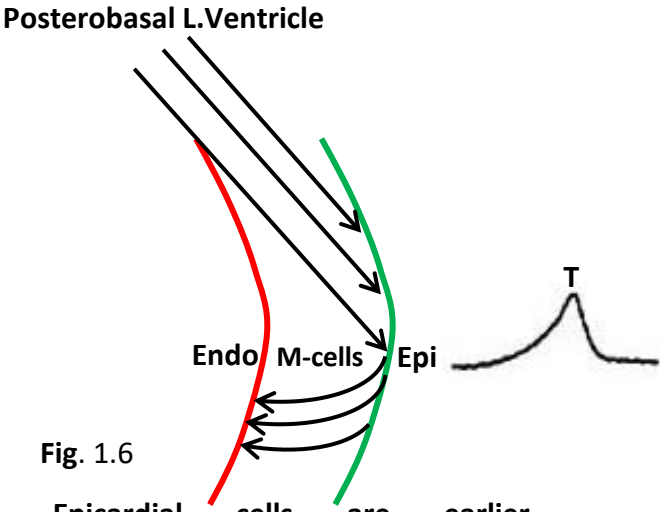


Fig. 1.6

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

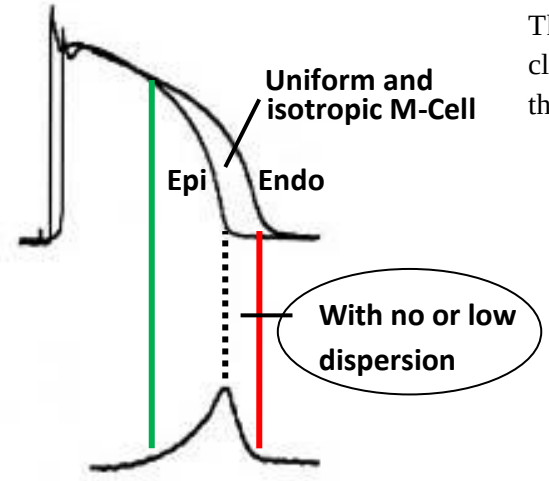


Fig. 1.7 Epicardium with a shorter action potential than endocardium

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

- ☐ Normally the myocardial cells are having uniform and isotropic conductivity, practically with no or low dispersion , hence having no impact on the shape of the T-wave
- ☐ It is the rate of repolarizatiion over the epicardium and endocardium which determines the asymmetrical nature of the T-wave.
- ☐ The descending limb of the T-wave is steeper because the rate of repolarization accelerates as the endocardium completes it recovery.

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 :

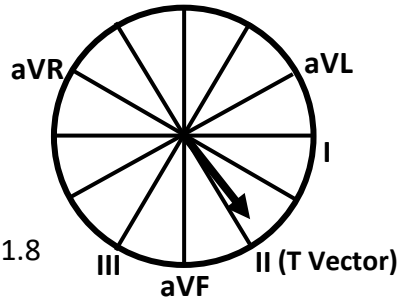


Fig. 1.8

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 is 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.

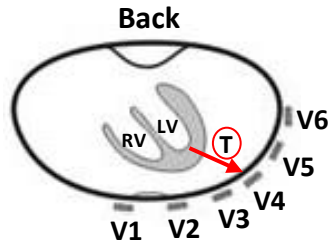


Fig. 1.9

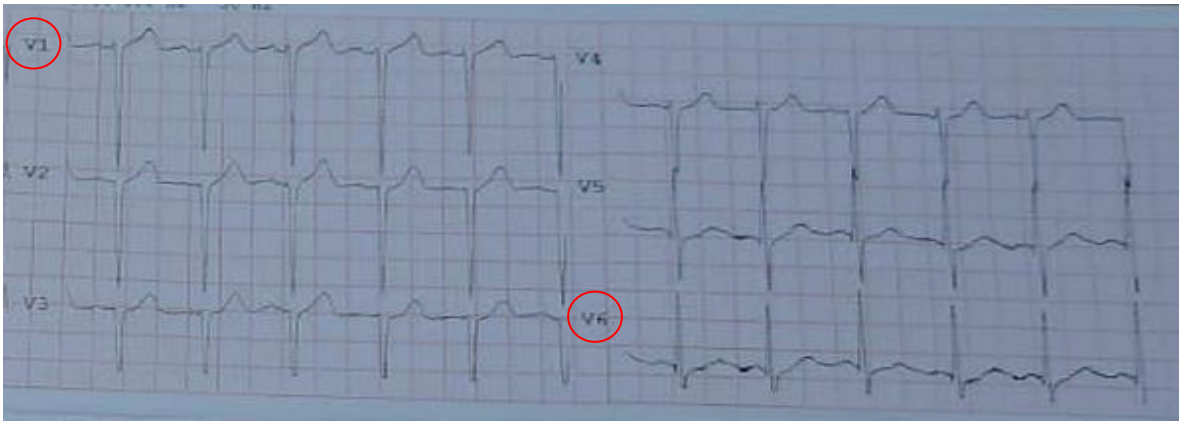
- The T-wave in V1 is usually negative , it may be flat or biphasic (with an upright first portion and inverted second portion)
- The amplitude of T in V6 is always more than in V1
- As one proceeds towards the intermediate zone V3/V4 in alignment with T vector , usually lead V4 is having the largest 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 , and especially if it is new
- This finding indicates a high likelihood of coronary artery disease, and when new it 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 Message

- **A reversed sequence of repolarization compared to that of the 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.
- Normally the myocardial cells are having uniform and isotropic conductivity, practically with no or low dispersion , hence having no impact on the shape of the T-wave
- It is the rate of repolarizatiion over the epicardium and endocardium which determines the asymmetrical nature of the T-wave.
- The descending limb of the T-wave is steeper because the rate of repolarization accelerates as the endocardium completes it recovery.
- Normal T-wave is slightly asymmetrical in shape, with a rounded apex that occurs closer to its end than its beginning.
- T-wave 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 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 T vector** is oriented leftward , inferiorly and anteriorly **over the frontal plane**
- 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.

(**Red signal** – an applied concept (for details please see page 28)

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THE CEREBRAL T-WAVE :
WHISPER OF BRAINSTORMING ON ECG

THE CEREBRAL T-WAVE : WHISPER OF BRAINSTORMING ON ECG

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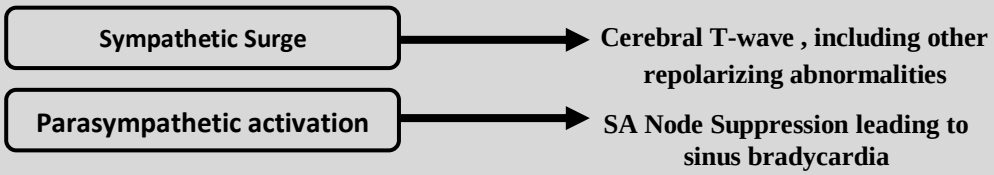
OUTLINE

Introduction

Cerebral T-waves are considered to be the echoes of acute intracranial catastrophe on ECG – particularly with intracerebral / subarachnoid haemorrhage , massive stroke , acute traumatic brain injury or rarely cerebral metastasis. Here ECG may display profound repolarizing abnormalities , most notably are widely deep cerebral T-wave inversions.

Understanding pathophysiological impact of acute intracranial catastrophe in the light of 'Cushing triad' : is a 'must' to understand cerebral T

Concerned ECG changes



This "dual run" is the main basis , where one can see the footprints of both autonomic nervous systems.

Illustration by ECG

Take Home Message

References

The Cerebral T-Wave : Whisper of Brainstorming on ECG

A Narrative Review

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Getting timely information of a disaster often saves our lives. At times neurologically-driven disaster may come in the form of acute brain insult – particularly intracerebral / subarachnoid haemorrhage , massive stroke , or acute traumatic brain injury. The heart speaks brain language through ECG as a messenger. Here the ECG may be the warning signal – not of broken heart but of a catastrophe in the brain. The brain suffers but the heart weeps.

- ☐ **"Cerebral T-wave" is the tale of such an acute neurological catastrophe , being expressed through ECG – often wide and deeply symmetrically inverted , best seen in precordial leads , usually associated with other repolarizing abnormalities like ST segment depression , QT prolongation , prominent U-wave , etc.**

- ☐ **The electrophysiology of this cerebral T-wave operates through sympathetic surge having its impact on the heart's physiology as well . This fact points towards the complex heart-brain interaction.**

Clinicians view this scenario through the lens of neurocardiology – its early recognition may enhance early diagnosis and management of this neurologically – driven catastrophe.

1. Introduction (Keypoints)

- Cerebral T-waves are considered to be the echoes of acute intracranial catastrophe on ECG – particularly with intracerebral / subarachnoid haemorrhage , massive stroke , acute traumatic brain injury or rarely cerebral metastasis. Here ECG may display profound repolarizing abnormalities , most notably are widely deep T-wave inversions , as illustrated below:

Cerebral T-wave

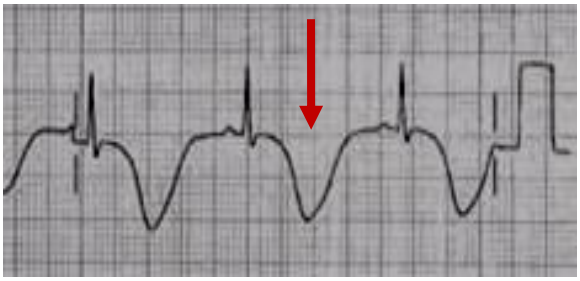


Fig. 1.1

- Characteristics of cerebral T-waves :
  - These T-waves are widely slayed and deeply inverted (usually ≥ 5 mm depth with ≥ 4 contiguous precordial leads) ,but truly they are diffuse in location.

- Other associated repolarizing abnormalities include ST depression , QT prolongation , prominent U-wave , etc. Sinus bradycardia may stand there as an accompanion.

  In one case series, the ECG pattern of cerebral T-waves with prolonged QT interval was seen in 72% of patients with SAH and 57% of patients with intraparenchymal haemorrhage.

Ref :
Raised Intracranial Pressure
Mike Cadogan and Robert Buttner
Jan 14, 2025
https://litfl.com/raised-intracranial-pressure-ecg-library/

- These ECG changes can very much simulate with those of acute coronary syndrome and can fire misdiagnosis with unnecessary cardiac interventions.
- This would be worth to mention here that the presence of cerebral T-waves on 12-lead ECG is not diagnostic of intracranial neurological catastrophe but can be suspected strongly in the appropriate clinical setting , and further diagnostic imaging (CT/ MRI of the brain) is required for its confimation. This is a life-threatening condition , the missing of which may lead to fatal catastrophe.
- But it is also true to realize that patients in ischemic stroke with cerebral T-waves should be assessed for cardiac dysfunction as well to exclude thromboembolic phenomenon as a causative factor.
- The mechanism responsible for such ECG changes associated with acute neurological insult is not well understood but likely the result of a sympathetic surge resulting in repolarizing abnormalities.
- Cerebral T-waves generally resolve spontaneously , but can persist upto 6 weeks.
- **Clinical Relevance of Cerebral T-wave** : importance of recognizing this pattern
  In medicine , we listen to organs. But sometimes , the heart does not speak for itself , rather it speaks for the brain.

✓**Broadly to say - Lives may depend on its recognition.**

Avoiding misdiagnosis as acute coronary syndrome
and unnecessary angiography or thrombolysis

Role of ECG as a red flag for brain imaging

ECG normalization after treatment of the cerebral cause

2. Understanding pathophysiological impact of acute intracranial catastrophe in the light of 'Cushing triad' : is a 'must' to understand cerebral T

- High intracranial pressure (ICP) is life-deadly. It sparks vasoconstriction resulting in a **decrease cerebral blood flow (CBF)** leading to an increase in carbon dioxide (CO2). This vasoconstriction response makes the body to believe that the brain is not getting enough blood. The resultant trend sends signal to the rest of the body to slow down (vasoconstriction as sympathetic surge) to supply more blood to the brain. This is translated into an **enhanced mean arterial pressure (MAP) in an attempt to increase the cerebral perfusion pressure (CPP)**.

- The baroreceptors in the aortic arch and carotid arteries perceive this increased blood pressure and whisper to the heart to be in relaxing mood (through parasympathetic activation) leading to bradycardia, but the brain is still under strain. This becomes a vicious cycle of the brain trying to get more blood and the baroreceptors trying to equilibrate, so the result is hypertension with bradycardia.

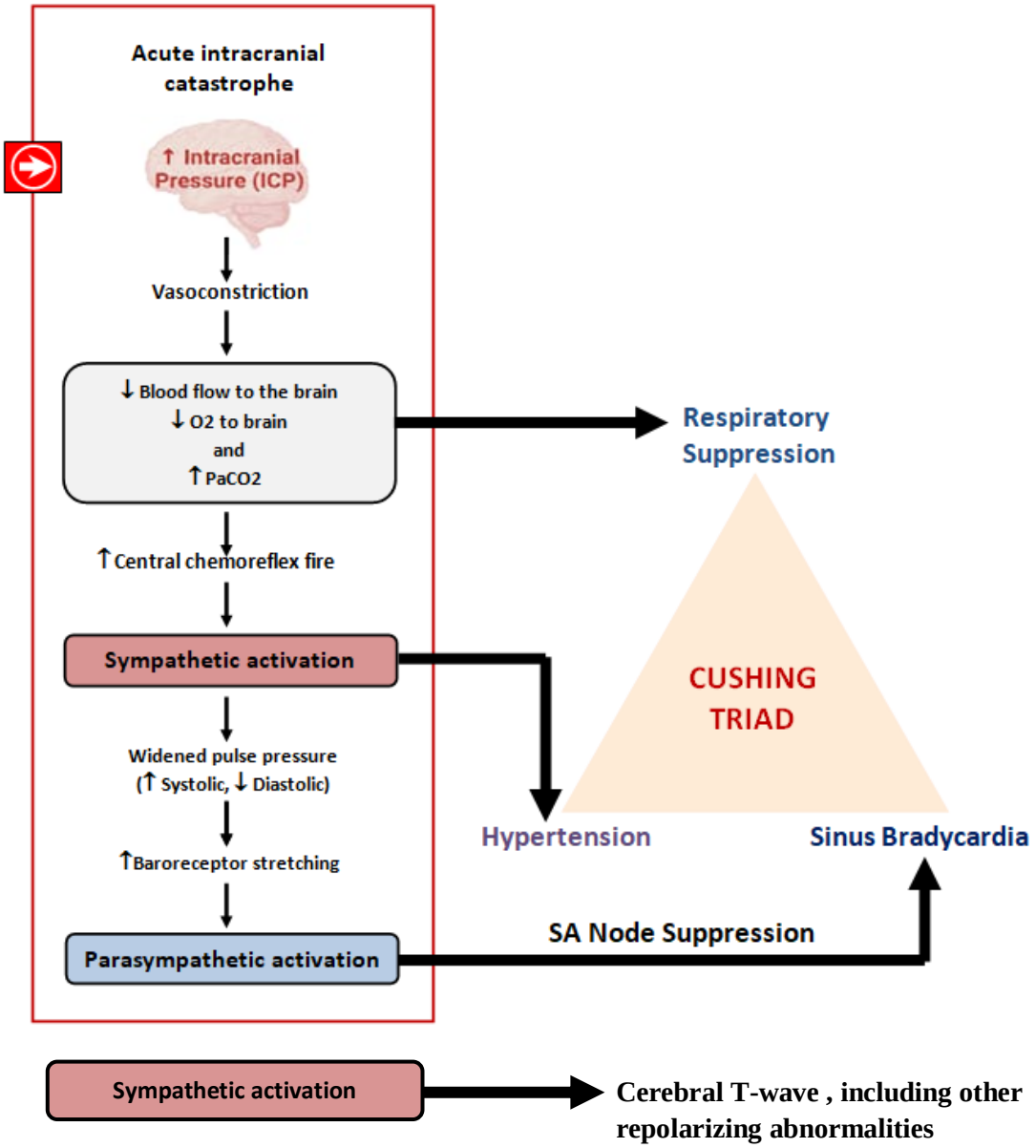


Fig. 1.2

(See 'explain' on the next page)

3. Concerned ECG changes

Basis

The electrophysiological basis of concerned ECG changes is attributed to the simultanenous activation of both sympathetic and parasympathetic nervous symptoms through the 'Cushings reflex' , as illustrated on the previous page. This "dual run" is the main basis , where one can see the footprints of both autonomic nervous systems.
Let us capture this essence through the following steps :

Sympathetic activation → **Cerebral T-wave , including other repolarizing abnormalities**

Parasympathetic activation → **SA Node Suppression leading to sinus bradycardia**

This autonomic tug of war in between both autonomic nervous systems is not mere opposition ; this is internal complementary—a paradoxical synergy. The heart becomes the foothold but not for dominance. Its physiological alteration is to protect the brain, to sustain the body in this storm, cerebral T waves are born—not from chaos, but from this orchestrated impact occurring in this context.

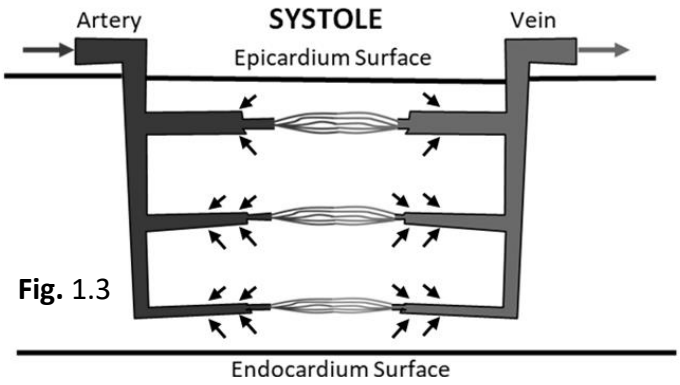


Fig. 1.3

The subenedocardial territory is richly innervated by sympathetic fibers , responsible for **"subendocardial sympathetic storm".**

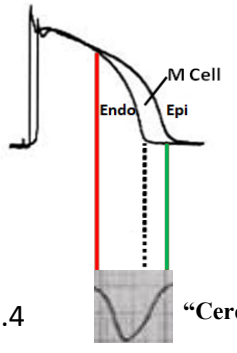


Fig. 1.4 **"Cerebral T"**

✓Reversed repolarization (endocardium to epicardium)
(Normal repolarization – epicardium to endocardium)

Sympathetic activation (surge)

The subendocardial territory of myocardium is richly innervated by sympathetic fibers – such a widespread and synchronized sympathetic surge brings a series of ECG changes :

- Accelerated AV conduction → shortened PR interval
- Myocardial stunning → reduced R wave
- Reversed repolarization (endocardium to epicardium)
  This reversal is mainly attributed to an abrupt and intense sympathetic dive – spreading diffusely into the subendocardial territory , most richly innervated by sympathetic fibres → the repolarization vector is reversed en masse → deep symmetric T-wav inversion **("Cerebral T").**
- Moreover, the intense adrenergic drive prolongs repolarization across myocardial layers, thereby lengthening the QT interval.

The heart , under this sympathetic storm , does not simply react – it echoes the chaos of the brain.

- **U-wave :** Its amplitude is increased during bradycardia. It may also fuse with terminal T-wave.

Heterogeneous myocardial repolarization (Variable regional sympathetic surge)**:**
The heart doesn't respond uniformly to the sympathetic surge. There can be regional differences in sympathetic innervation with variable coronary microvascular network. There may be also individual variation in autonomic tone and heart sensitivity. The net response also depends upon the location of hemorrhage , the maximum sympathetic impact being at site of insular cortex (temporal lobe interoceptive centre)involvement.
The overall footprint of heterogeneous myocardial repolarization can result in :

- ☐ Areas having more myocardial stunning or with more ischemia →sympathetic surge→cerebral T-waves
- ☐ Differently other areas (having big transmural repolarization gradient) may experience early repolarization–like pattern, producing giant upright T-waves
  [J-point/ST elevation – Osborn like pattern with giant T-waves]

NB :
In some cases, these ECG abnormalities may be associated with echocardiographic evidence of regional ventricular wall motion abnormality (so-called "neurogenic stunned myocardium").

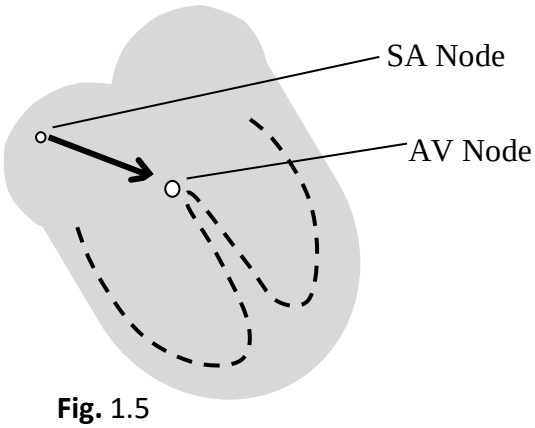


Fig. 1.5

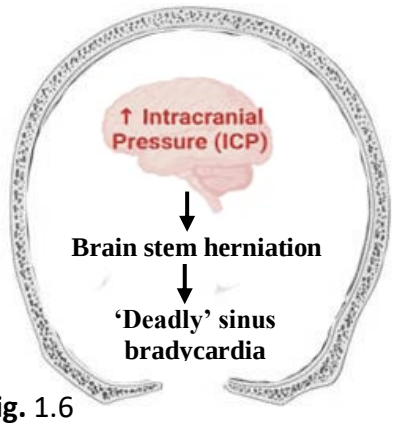


Fig. 1.6

Parasympathetic activation

Impact on SA node : Sinus bradycardia ; advancing bradycardia indicates imminent brain stem herniation

↓

Brain stem herniation : Increase intracranial tension resulting from acute neurological catastrophe can cause further slowing of the heart rate with impending danger to life.

In context with a case of brain stem herniation , see the following :

"You review his vitals over the past 2 hours and see that his heart rate on admission was in the 80s and has now dropped to the 30s, while his blood pressure was initially 110/54 and has now skyrocketed to 187/125! What could be going on?"

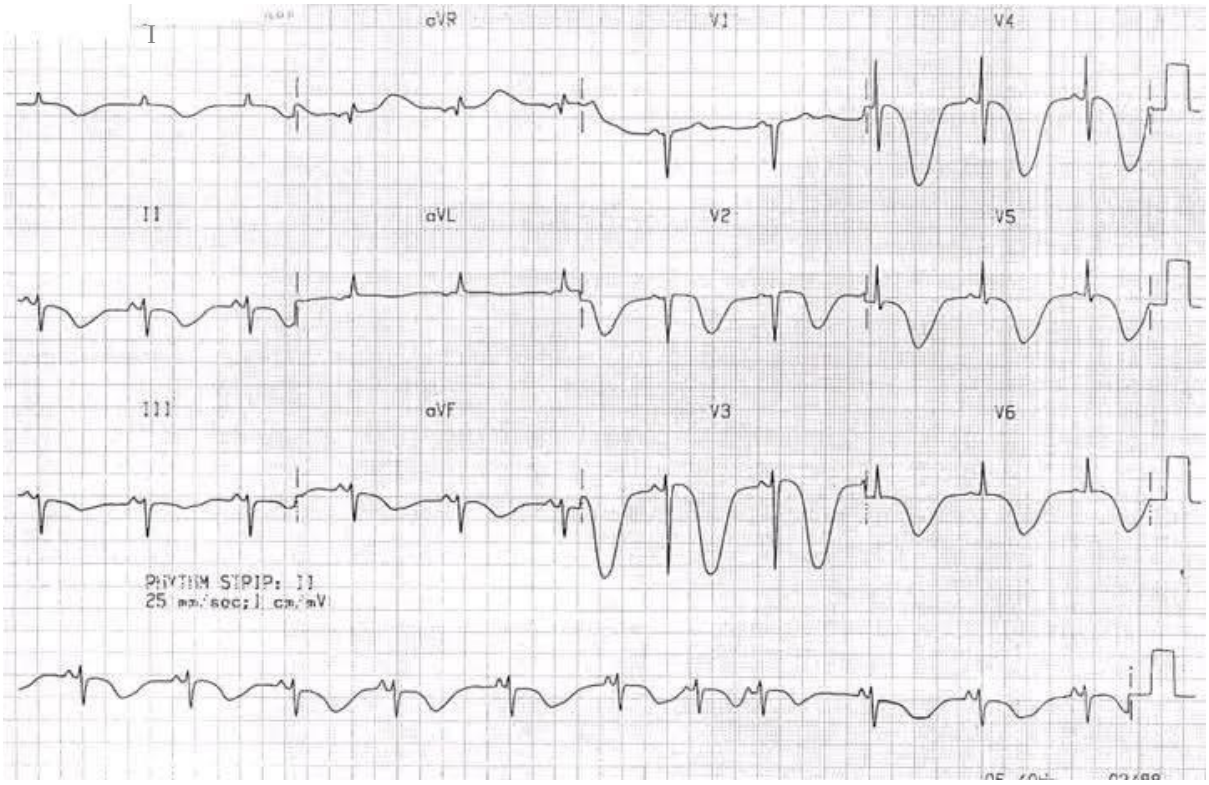
Ref : (See the details here)
ECG Pointers: Intracranial Hemorrhage
November 14, 2018
Lloyd Tannenbaum
https://www.emdocs.net/ecg-pointers-intracranial-hemorrhage/

This 'dual run of sympathetic and parasympathetic activity' results in an ECG that can easily mislead , unless the brain is suspected as the source (e.g. , sudden headache + vomiting + altered sensorium with neurologically unexplained bradycardia). CT/MRI brain should be urgently pursued in.

4. Illustration by ECG

History : 68-year-old man with history of prostatic neoplasia, consulted for the appearance of headache waking him up in the 2nd half of the night, accompanied by jet projectile vomiting, slightly obnubilated.

The ECG of the corresponding case is as follows :



Source : CME INDIA , Dated 8th April , 2025 by Dr. C.B. Prasad , Senior Consultant Physician Sitamarhi Bihar

ECG findings	Comments
1. Heart rate 62 bpm 2. Shortened PR interval (0.10 sec)	Heart rate towards the upper limit of sinus bradycardia with accelerated AV conduction
3. Deep symmetrical T-wave inversion (>5 mm depth) in precordial lead V2-V6 in a contiguous manner. Leads I , II and aVF also show somewhat deep T-wave inversion , when compared to the corresponding R wave	Strongly suggestive of cerebral T-wave in the presence neurological symptoms , as enumerated under the history.
4. Much reduced R-wave amplitude in comparison to deep symmetrical inverted T-wave	Myocardial stunning
5. Prolonged QT interval (>1/2 of the corresponding RR interval)	Prolongation of the repolarization phase across myocardial layers , thereby lengthening the QT interval
6. Left anterior fascicular block; Occasional atrial extrasystole as seen in rhythm strip II	----

These findings are suggestive of acute neurological catastrophe.
(CT/MRI of the brain not available)

5. Take Home Message

- "Cerebral T-wave" is the tale of an acute neurological catastrophe , being expressed through ECG – often wide and deeply symmetrically inverted , best seen in precordial leads , usually associated with other repolarizing abnormalities like ST segment depression , QT prolongation , prominent U-wave , etc.

- These T-waves are widely slayed and deeply inverted (usually $\geq$ 5 mm depth with $\geq$ 4 contiguous precordial leads) , but truly they are diffuse in location.

- This would be worth to mention here that the presence of cerebral T-waves on 12-lead ECG is not diagnostic of intracranial neurological catastrophe but can be suspected strongly in the appropriate clinical setting , and further diagnostic imaging (CT/ MRI of the brain) is required for its confirmation. This is a life-threatening condition , the missing of which may lead to fatal catastrophe.

- Understanding pathophysiological impact of acute intracranial catastrophe in the light of 'Cushing triad' : is a 'must' to understand cerebral T.
  Cushing's triad is a set of three signs of increased intracranial pressure (ICP), including hypertension with a widening pulse pressure, bradycardia, and irregular respirations. It points towards a severe neurological problem and signals potential brain herniation or brainstem compression.
  Sympathetic surge occurs as a part of Cushing reflex leading to cerebral T-wave genesis , including other repolarizing abnormalities.

- **Sympathetic activation (surge)** : The subendocardial territory of myocardium is richly innervated by sympathetic fibers – such a widespread and synchronized sympathetic surge brings a series of ECG changes :
  - Accelerated AV conduction → shortened PR interval
  - Myocardial stunning → reduced R wave amplitude
  - Reversed repolarization (endocardium to epicardium) → "Cerebral T"
  - Moreover, the intense adrenergic drive prolongs repolarization across myocardial layers, thereby lengthening the QT interval.

  Parasympathetic activation : Impact on SA node : Sinus bradycardia ; advancing bradycardia indicates imminent brain stem herniation

- This 'dual run of sympathetic and parasympathetic activity' results in an ECG that can easily mislead , unless the brain is suspected as the source (e.g. , sudden headache + vomiting + altered sensorium with neurologically unexplained bradycardia).
  CT/MRI brain should be urgently pursued in.

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15. Referred to ChatGPT whenever needed

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

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

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OUTLINE

Introduction : Keypoints
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.

The electrophysiological mechanism
When the sequence of repolarization is reversed i.e. epicardial repolarization is delayed , the subsequent takeover of repolarization by the endocardial territory. Thus , the resultant more conduction delay across the transmural myocardium means the reversal of the image of T-wave so produced as deep T-wave inversion

Mechanism of deep T-wave inversion with some important clinical electrocardiographic scenarios , as examples
- Enhanced ventricular diastolic filling with ventricular distension
- Transmural ventricular wall stress
- Secondary T wave alternations
- Idiopathic global T wave inversion syndrome

An approach to its diagnosis

Take Home Message

References

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

A Narrative Review

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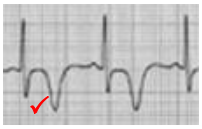
There are so many happenings in nature , which make a man surprised. A small child may cry by seeing an inverted image in water. It is a well-known phenomenon that the image of an object in water looks to be inverted i.e. the object's top side image forms on the bottom side and the bottom side on the 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. epicardial repolarization is delayed , the subsequent takeover of repolarization by the endocardial territory. Thus , the resultant more conduction delay across the transmural myocardium means the reversal of the image of T-wave so produced as 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)

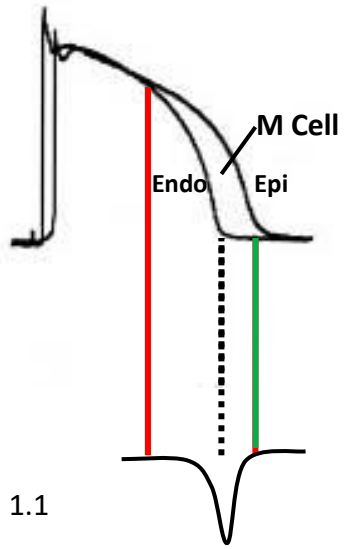


Fig. 1.1

The reasoning for deep T-wave inversion

When the sequence of repolarization is reversed i.e. epicardial repolarization is delayed , the subsequent takeover of repolarization by the endocardial territory. Thus , the resultant more conduction delay across the transmural myocardium means the reversal of the image of T-wave so produced as deep T-wave inversion. In other words the net result is the reversed T-wave polarity with more deepness.

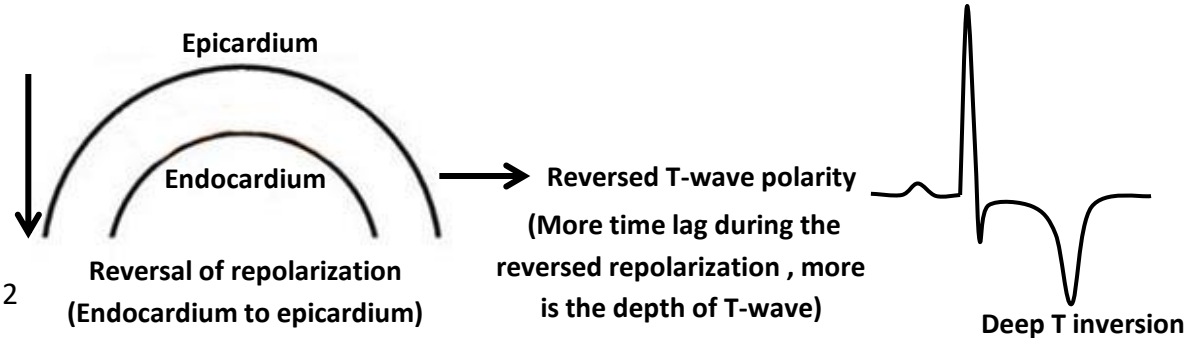


Fig. 1.2

3. Mechanism of deep T-wave inversion with some important clinical electrocardiographic scenarios , as examples :

Basis : Reversal of repolarization (endocardium to epicardium)

- **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. Ventricular overloading
  - 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.
  (The diagnosis idiopathic global T wave inversion syndrome depends upon the exclusion of the clinical entities , as mentioned under this head).

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 ± 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 with excessive sympathetic surge	History of cerebral causes with diffuse / giant T-wave inversions (See page 31-40 for the details)
☐ **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. Take Home Message

- **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.
- 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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T-WAVE ALTERNANS :
ITS ELECTROPHYSIOLOGICAL INSIGHT

T-WAVE ALTERNANS : ITS ELECTROPHYSIOLOGICAL INSIGHT

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OUTLINE

Introduction : Keypoints

- T-wave alternans is a periodic beat-to-beat alteration of the amplitude and /or shape of the T-wave on ECG.
- It is a rate dependent primary phenomenon
- The endocardial myocytes are more prone to repolarization alternans
- T discordant alternans creates a myocardial substrate that is vulnerable to cause unilateral conduction block with the setting of reentrant ventricular arrhythmias.

Classification of TWA

T-wave alternans can be divided into two subtypes :

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

Concerned electrophysiology

- ☐ 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

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

T-wave alternans analysis techniques

Prognostic significance of T-wave alternans

Take Home Message

References

T-wave alternans : its electrophysiological insight

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)

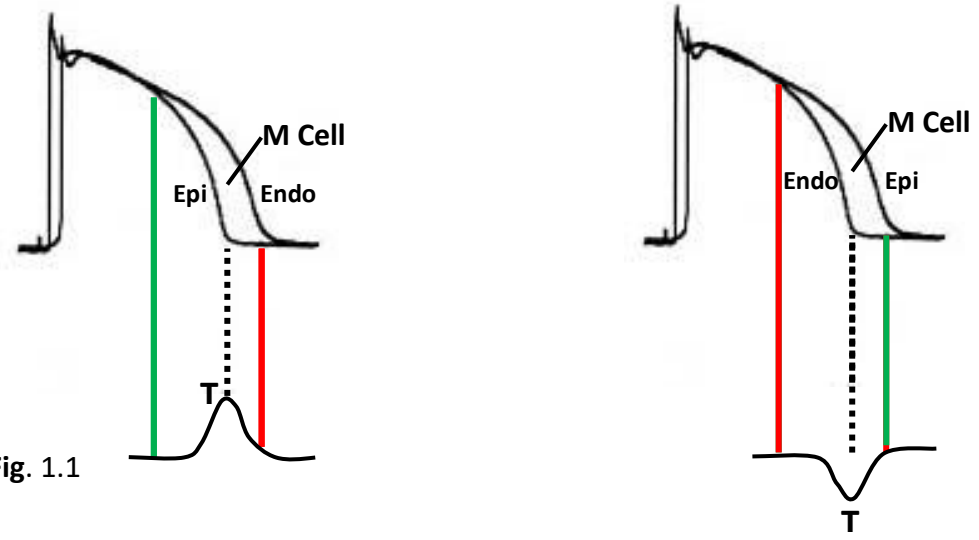


Fig. 1.1

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)

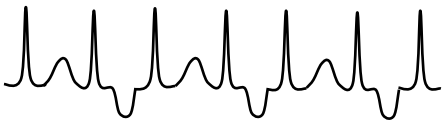


Fig. 1.2

- 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 <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 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 :

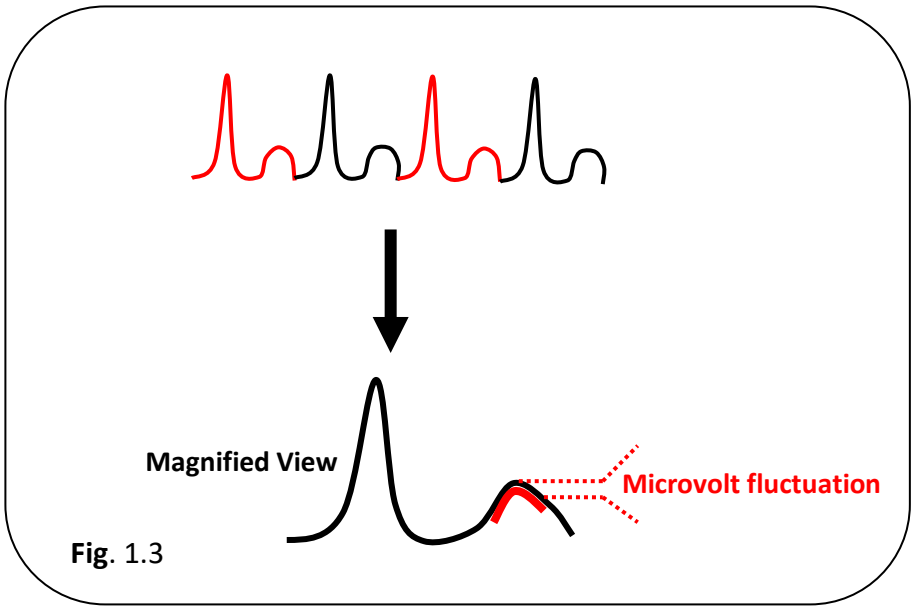


Fig. 1.3

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 , 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.

The 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

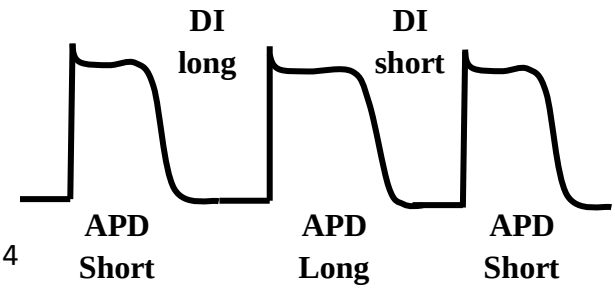


Fig. 1.4

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 filling 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

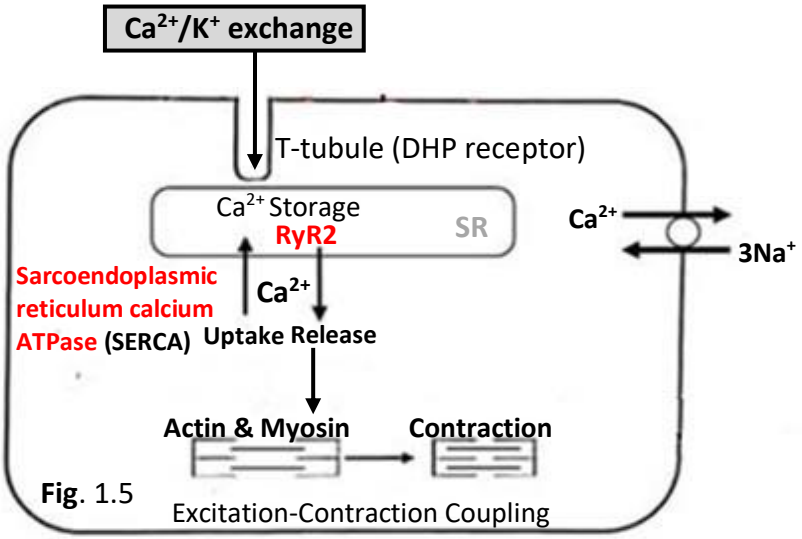


Fig. 1.5

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) :

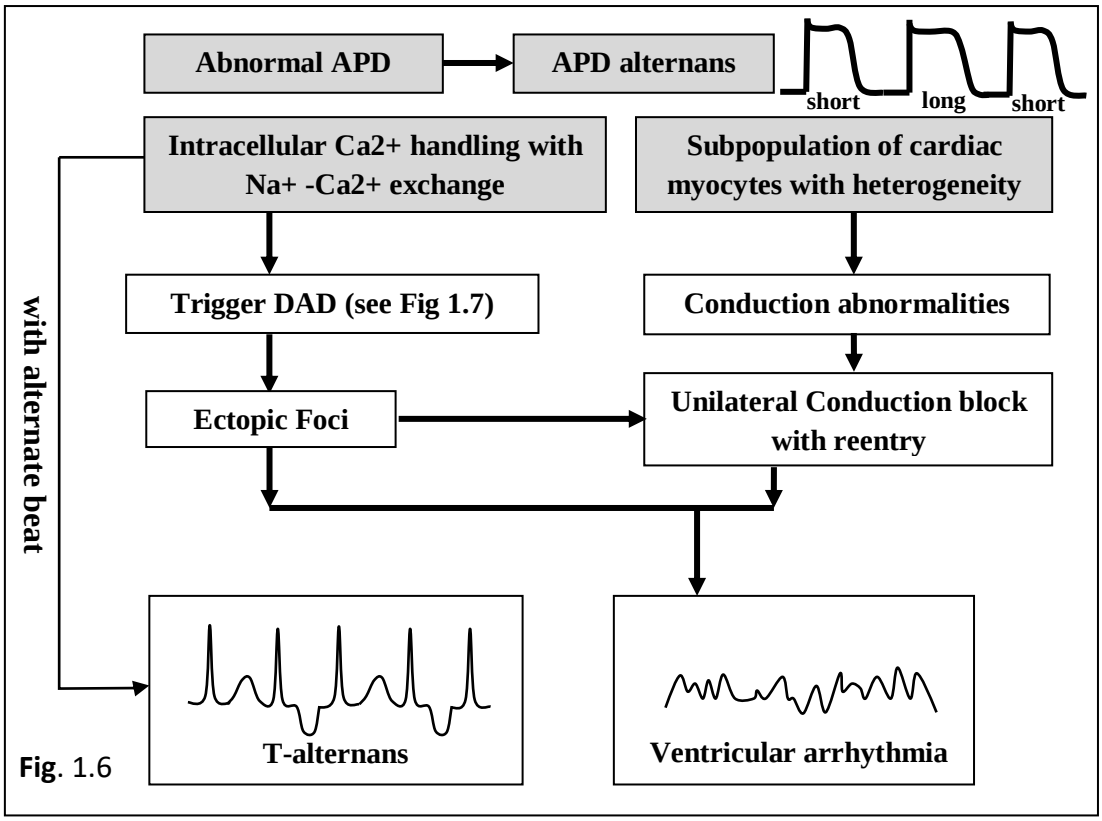


Fig. 1.6

Tachycardia → changes in calcium handling mechanism , as discussed in page 53 (Fig 1.5)

→ To exit out more accumulated Ca2+ → Na2+ ions entrance inside

Ca^{2+} ⇄ Na^{+}

→ Triggering delayed afterdepolarization

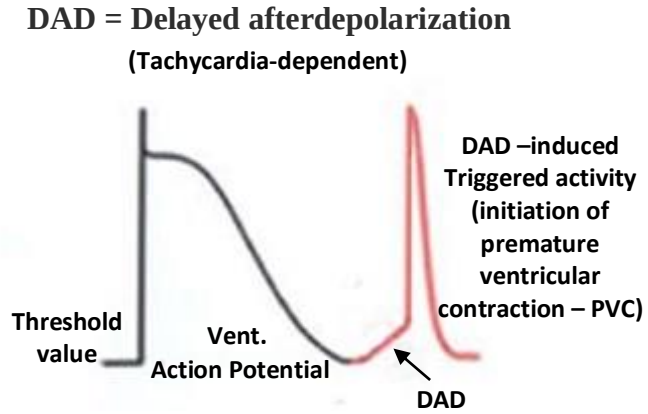
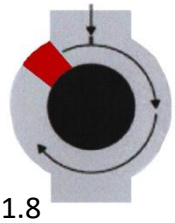


Fig. 1.7



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.

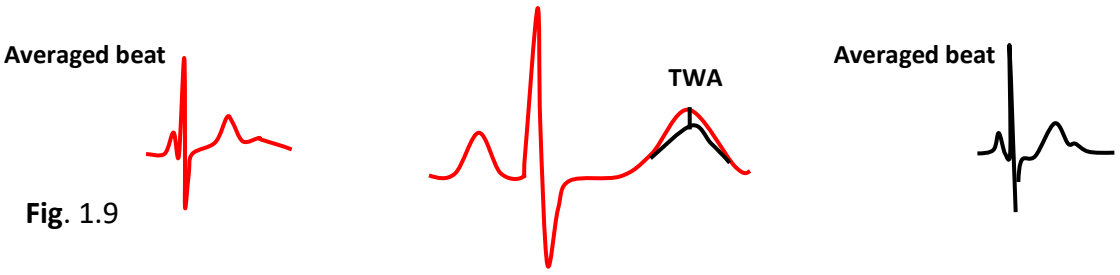


Fig. 1.9

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. Take Home Message

- T-wave alternans is a periodic rate dependent beat-to-beat alteration of the amplitude and /or shape of the T-wave on ECG.
- **TWA is a rate dependent primary phenomenon** induced with higher heart rate threshold and once perceived it remains remarkably stable and persistent provided the heart rate remains the same.
- **The endocardial myocytes are more prone to repolarization alternans** compared to epicardial myocytes. Therefore , discordant TWA is more observed compared to concordant one.

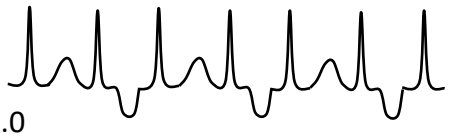


Fig. 2.0

- T-wave alternans can be divided into two subtypes :
  - Visually apparent – macro-TWA
  - Visually inapparent – micro-TWA
- Main mechanism dealing with intracellular calcium handling inside the cardiac myocyte , as stated below :

Tachycardia → changes in calcium handling mechanism , as discussed in page 53 (Fig 1.5)
→ To exit out more accumulated Ca2+ → Na2+ ions entrance inside

→ Triggering delayed afterdepolarization

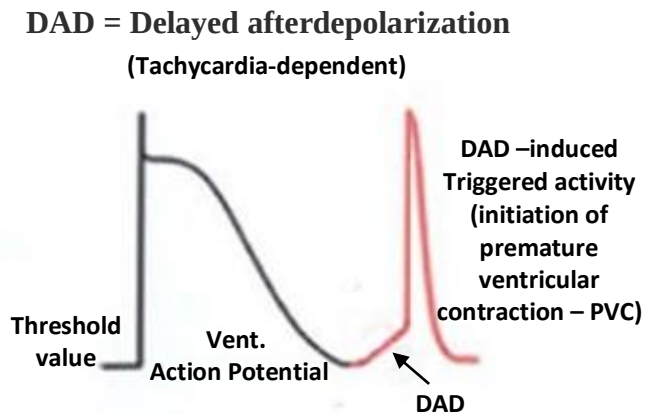


Fig. 2.1

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).

- 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 <40% , and is a good predictor of risk for SCD.

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PSEUDONORMALIZATION OF T-WAVE : A DECEPTIVE ECG PHENOMENON

PSEUDONORMALIZATION OF T-WAVE : A DECEPTIVE ECG PHENOMENON

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OUTLINE

Introduction

'Pseudonormalization of T-wave' is a deceptive ECG phenomenom which causes pre-existing abnormal negative T-wave to its complete reversal to positive T-wave during a fresh acute anginal episode.

Electrophysiological mechanism of T-wave pseudonormalization

supposed to be the result of superimposition of a fresh acute myocardial ischemia over chronic ischemic injury. .

A. Electrophysiological changes with pre-existing chronic myocardial ischemia
B. Electrophysiological changes during superimposing of a fresh acute anginal episode (Pseudonormalization of T-wave)

Discussion

Take Home Message

References

Pseudonormalization of T-wave : A deceptive ECG phenomenon

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. This pseudonormalization may impart a temporary feeling of its being normal - if such a situation remains without being noticed , some sinister happening might take place.

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

- ☐ **Occasionally , a negative T-wave on ECG in chronic myocardial ischemia may completely revert to an upright T-wave during a fresh acute anginal episode - this phenomenon is known as 'Pseudonormalization of T-wave'.**
- ☐ **This reversal is commonly indicative of a severally narrowed or near-total coronary occlusion.**

T-wave pseudonormalization is not a return to health but rather a signal of evolving electrical instability in the myocardium.
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 phenomenon which causes pre-existing abnormal negative T-wave reversal to positive T-wave during a fresh acute anginal episode.

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

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

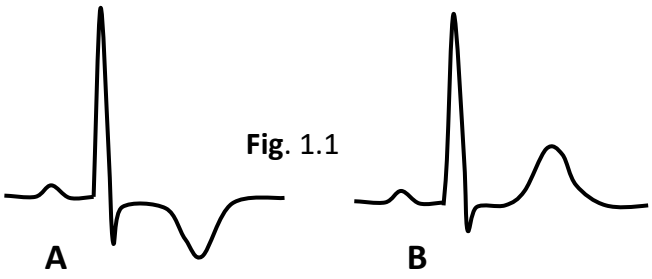

Fig. 1.1

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

- Spontaneous pseudonormalization (PN) is an unique 12-lead electrocardiographic (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 term pseudonormalization is most commonly used in the context of chronic myocardial ischemia , as discussed above. However , pseudonormalization can also occur in other conditions with pre-existing T-wave inversion on ECG.
- If such a 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 a fresh myocardial ischemia over chronic ischemic injury. This impact affects the myocardial action potential steps accordingly during the repolarization phase of T-wave genesis.

Normal T wave: In healthy myocardium, the epicardium repolarizes earlier than the endocardium due to shorter action potential duration in the epicardial cells. This creates a current flow from the endocardium to the epicardium, producing an upright T wave on the ECG.

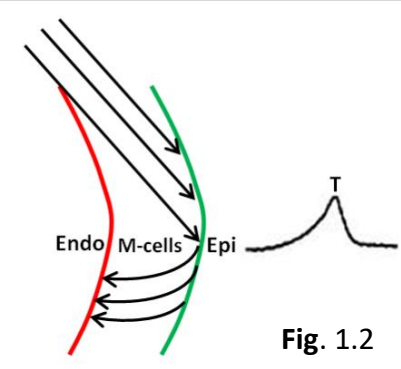


Fig. 1.2

A. Electrophysiological changes with pre-existing chronic myocardial ischemia

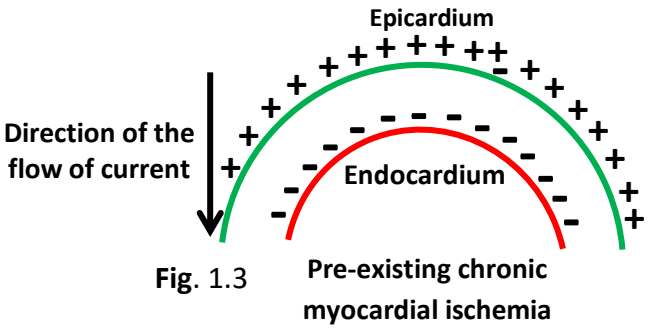
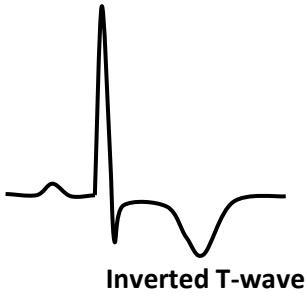


Fig. 1.3 Pre-existing chronic myocardial ischemia


Inverted T-wave

- The endocardium , being closure to the oxygen – demanding myocardium and further away from the surface collateral vessels, is more severely affected by the ischemia with ATP depletion and cellular damage resulting in potential negativity on its surface (see fig.1.3)

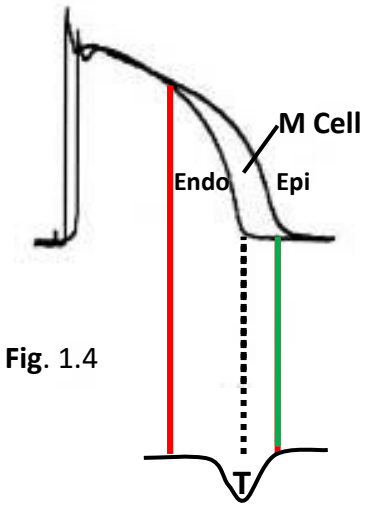


Fig. 1.4

- The epicardium might retain relatively better perfusion due to proximity to surface collateral circulation. This allows preservation of positive charge over the epicardium.
- Positive charge over epicardium and negative charge over endocardium → the current of flow is from epicardial region towards endocardial region → **T-wave inversion** (if the flow of current is away from the exploring electrode , the wave recorded is negative in nature by the law of electrocardiology).

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

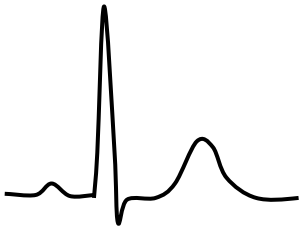


Fig. 1.5 **Upright T-wave**

The mechanism of pseudonormalization of T-wave with superimposed acute myocardial ischemic insult involves dynamic changes in myocardial repolarization. The underlying mechanism is explained as below :

- New ischemic insult disrupts repolarization ionic currents (e.g. , increased extracellular K+ concentration) → Shortened action potentials and vector shift towards the epicardium → previously inverted T-wave becomes upright.

  The epicardium may once again repolarize earlier than the endocardium due to transient changes in its action potential duration, making the current flow towards the epicardium as with the normal state. However, this T wave appearance is misleading and does not reflect true recovery

- This is worthwhile to mention here that this reversal is purely pathological and not indicative of acute myocardial recovery. This gives the false impression of "normalization" , hence the term "pseudonormalization".
- Psudonormalization is often a transient event. If ischemia worsens , the T-wave may revert again to inversion. Serial ECGs are essential to capture this dynamic progression.

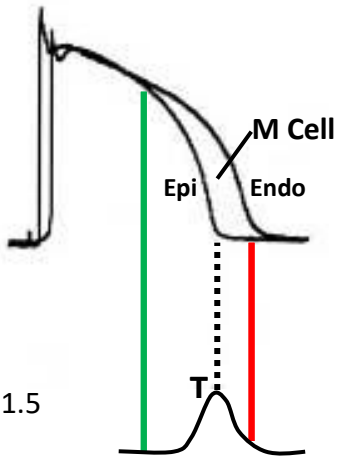


Fig. 1.5

In nutshell , the main concern here is the shortening of action potential duration in context with the epicardium

- ☐ The opening of K(ATP) channels allows potassium efflux, leading to the shortening of the action potential
- ☐ This ionic change reverts the direction of the flow of current towards epicardium , thus again modifying the repolarization sequence from endocardium to epicardium which may result in T-wave pseudonormalization

3. Discussion

This 'pseudonormalization of T-wave' is a definite entity , a paucity of data exists 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 pre-existing chronic ischemic injury which converts the entire zone into near - transmural myocardial ischemia.

This spontaneous pseudonormalization is found to be associated with a severe narrowed or near- total coronary occlusion , which usually results 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-wave occurs in patient with clearcut history of chronic anginal background and is a sign of superadded acute ischemic insult. 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.

This would be worthwhile to mention here that after thrombolysis or percutaneous coronary intervention (PCI) , previously inverted T-waves may become upright , sometimes due to successful reperfusion or reperfusion injury.

4. Take Home Message

- 'Pseudonormalization of T-wave' is a deceptive ECG phenomenon 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 well-known unique 12-lead electrocardiographic (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.
- Pseudonormalization is not exclusive to chronic myocardial ischemia but can occur even in any scenario where in the baseline T-wave inversion is pre-existing due any other pathological entity. It is crucial to correlate this finding clinically and with other diagnostic data to determine its significance.
- Pseudonormalization of the T wave during an active anginal episode should be considered a red flag for severe coronary artery disease , often indicating critical stenosis or near-total occlusion. Immediate assessment with serial ECGs , cardiac biomarkers , and urgent coronary angiography is essential to prevent complete infarction.

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